IDEF/CFIDS

Dr. Cecile Jadin's Papers are now available in full Click Here

Updated on: 02/25/2001

[French] [German] [Italian] [Portuguese] [Spanish]

Welcome! I am a Chronic Fatigue Syndrome sufferer and started this site to try bringing together all of the research in a manner that may make sense to a 'brain-fogged' CFSer or PWC (Person With Chronic Fatigue), and help me better understand this illness.

I am currently in full remission, in fact, 102% recovered - I am better now than *before* my sudden onset. For a presentation on my current understanding of this illness, click here. My wife and two of my children also have CFIDS diagnosis, under treatment and *improving*.

This illness has been documented for about 250 years[BMJ] but due to its mechanism (an infrastructure illness), a partial medical understanding has only recently arose. There are over fifty(50) different *theories* of the IDEF / CFIDS mechanism [1].

I have evolved to a model which appears to simply explain both the symptoms and the successful treatments reported of this illness. With this model, I have gone into almost complete symptom remission (and what's left is fading week by week)...

The illness is caused by commonly by one of three things:

- A bug similar to 'walking pneumonia' except that it affects the blood system and not the lungs.
- Coagulation (thickening of the blood)
- Alkaline blood

Once it is started, all three of the above may quickly occur (they assist each other!) The body may tricked by this bug to produce a variety of blood thickening substances that 'chokes' the body AND/OR modifies the pH (acid level) of the blood to one that inhibits the release of oxygen from red blood cells. Or the alkaline blood allows bugs to become established. This choking results in a drop of oxygen level in the blood causing symptoms that matches exactly Acute Mountain (Altitude) Sickness [Hypoxia]. This bugs hates oxygen, so this trick allows it to create a new home! The bug then finds several 'homes' in the body, including the lower gut... resulting in sensitivities to food and chemicals ("leaky gut"), poor vitamin absorption (by reducing the acid content of the stomach) etc. It also, cause the number of red blood cells to drop (and many are deformed by the thickening of the blood). It also produces a ton of toxins (poisons) which further affects the body - ugly!

Other cases may be caused by chemical, genetic or other damage to the body's ability to transfer oxygen in the blood system. This also produces Hypoxia symptoms. This appears to be rarer but may be as high as 10%.

With an understanding of what is going on, several treatments are possible - and they work for most (but not all) CFS patients :-(. I have no interests in the treatments - except that they have worked for me and others... and you can try them yourself... The treatment goal is to raise O2 level in the blood resulting in two benefits:

- Symptom relief
- An anti-bacteria *and* anti-viral effect on the body (O2 does both), leading to eventual 'cure' or control.

The first case of this illness can be described as a *mycoplasma-coagulation induced Hypoxia* (acute mountain sickness), the second case can be described as blood-alkalinity induced Hypoxia.

Mycoplasma (or similar) infection is probably the most *common* maintainer of this state -- but other causes are likely (chemical exposures, genetics, etc). Any sickness that could result in a long term decrease of oxygen transportation to the body may create CFS symptoms (and thus be called CFS).

"Although virtually *all microorganisms* can cause DIC, bacterial infection is most frequently related to the development of the syndrome...Disseminated intravascular coagulation (DIC) is

characterized by the *widespread activation of coagulation, which results in the intravascular formation of fibrin and ultimately thrombotic occlusion of small and midsize vessels*." [*]

This ***family*** of *infectious* illnesses bears many (mis-)names (and the U.S. Congress has a committee to decide an official term for the illness). Other common names are:

- ISAC - Immune System Activation of COAGULATION
- REDD - Rnase-L Enzyme Dysfunction Disease ,
- World Health Organisation (International Classification of Diseases, 10, G93.),
- Gulf War Syndrome - 50+% of GWS matches the CDC definition of CFIDS
- Akureyri Fever (epidemic neuromyasthenia) or Icelandic disease - after a 1948 outbreak - there are still 77 patients of this outbreak alive that has never recovered ...
- CFS - Chronic Fatigue Syndrome
- CFIDS - Chronic Fatigue and Immune Dysfunction Syndrome (older form is Chronic Fatigue and Immune Deficiency Syndrome)
- ME - Myalgic Encephalomyelopathy
- CBEV - Chronic Epstein-Barr Virus
- Exertion Fatigue, Post-Viral Fatigue Syndrome,
- Acquired Neurasthenia
- Fibromyalgia - *may be a "first cousin"*
- Post Polio Syndrome [1, 2] - *another possible "first cousin"*
- *For addition names see "The Disease of a thousand names"*

There is evidence suggesting that Florence Nightingale(1820-1910) suffered from IDEF/CFIDS, a Canadian CFIDS organization is named after her. The disease was first described in 1750 by Sir Richard Manningham, "***febricula***" or little fever

It is recognized by the Centers for Disease Control (CDC) and there are approximately 36 web pages describing research at their site.

You may also wish to visit the National CFIDS Foundation.

and Jen's site: http://www.munn.com/~jmunn/cfids/cfids.html

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

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Updated on:
10/15/2000

(Her presentation is available at:
http://www.geocities.com/HotSprings/Spa/9168/rickettsia01.html
)

Notes from her talk at:
http://www.cfs.inform.dk/Nyheder.udland/cfsnews19aug.txt

The ***papers on the left*** are published here with the permission of Dr. Jadin. The PDF files are in the same sequence as the HTML form.

NOTE: I will be improving the formatting in the next few days.

NOTE: MDs are advised to contact Dr Jadin (email:gerinjadin@icon.co.za) for clarifications or changes of protocol. This is an *informational* page only.

From ©1999 Clinical and Scientific Basis of Chronic Fatigue Syndrome - From Myth towards Management Sydney'98 International Conference

Treatment of Rickettsial Infection

Dr. Cecile Jadin, Randburg, South Africa.

NOTE: MDs are advised to contact Dr Jadin (email:gerinjadin@icon.co.za) for clarifications or changes of protocol. This is an *informational* page only.

Following regimes should be applied consecutively 7 days/month for a period of 3 months to 2 years.

1. Vibramycin (doxycycline) {100 - 200 mg BD}: According to weight and tolerance
2. Riostatine-F(oxytetracycline) {250 - 500 mg QID, 500 TDS}: According to weight and tolerance
3. Minomycin (Minocycline) {50 mg + 100mp/day, 100 mg BD}: According to weight and tolerance
   plus Rulide (Macrolide) 150 BD

4. Tetralisal (lymecycline) 300mg BP x 7 days
   plus Flagyl/Metronidazole {200 - 400 mg BD}: According to weight and tolerance
5. Dumoxin (doxycycline) (100 mg + 50mg dly, 100mg BD)
   plus Quinolone = Ciprobay(ciprofloxacin) {250 BD} or
   Maxaquin T nocte (Lomefloxacin)

Adjuvants: B12 injections (Cobalamin), Acidobacillus, proton pump inhibitor daily or BD
Nivaquine(Chloroquine sulphate) when RF, CRP, AND(+), used in between treatments

Editor Note: This treatment protocol is also effective against mycoplasma infections.

Rickettsiae

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Jadin Protocol
Description of Rickettsia

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Updated on:
02/07/2000

A group of gram-negative organisms that are half way between bacteria and virus (like Mycoplasma). which includes:

- Rickettsia [Known Species]
- Coxiella [Known Species]
- Rochalimaea [Known Species]

Some CFIDS is believed to be caused by chronic rickettsiae infections. See Presentation at the 1999 Sydney CFIDS/ME Conference by Dr. CL Jadin for more explanation.

Britannica Description of Acute Rickettsiae C.J.H. Nicolle, Noble Prize 1928

English Introduction to Literature of Rickettsiae and CFIDS

Jadin's Treatment Protocol

Disease Vector

- transmitted through the air
- transmitted by the bite of infected ticks, lice,and fleas

Symptoms

See
http://www.mitretek.com/mission/envene/biological/agents/rickettsia.html

Leading Researchers

- Giroud P
- Jadin JB
- Dr Philippe Bottero

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CWD

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Rickettsiae Mycoplasma Testing

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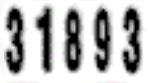

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02/08/2000

On this website the term "CWD Bacteria" or Cell-Wall Deficient Bacteria is used to describe a ***variety*** of intracellular organism which may be cofactors in CFIDS. These include:

- Known (and unknown) Cell Wall Deficient bacteria (Mycoplasma is the best known of these)
- Mycoplasma: Mycoplasmas are among the smallest of bacterial organisms.
  See:
  - Mycoplasmas: Sophisticated, Reemerging, and Burdened by Their Notoriety
- Rickettsiae:
- Chlamydiae: *C. pneumoniae* was identified as a separate *Chlamydia* species in the 1980s. It causes various respiratory-tract infections, most commonly a mild, atypical pneumonia with symptoms of fever, cough, and sore throat.
  See:
  - Chlamydiae as Pathogens: New Species and New Issues, Note: Chlamydiae *pneumoniae versus Mycoplasma pneumoniae*

All of these organisms are typically treated with (the same) antibiotics.

Introduction to Cell Wall Deficient Bacteria

http://garynull.com/Documents/Arthritis/free-living_amoeba.htm

"many if not most CFS patients have both Chlamydia and Mycoplasma infections"

- Prof Nicolson

"it has been demonstrated that that mycoplasma can induce increased cytokine levels when infected in white blood cells, and ***can allow dormant viral infections to activate and replicate at abnormal levels***." **Bill Paspaliaris [*]**

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Antibiotics

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International CFS Information

Updated on:
02/20/2000

The most common prescribed antibiotic for Cell Wall Deficient infection is doxycycline. Doxycycline is one of the 20 most prescribed drugs in the US, with extremely few side-effects, well understood, *cheap* and in use for generations. Today, it is often prescribed for Acne [illustrating the very low risk factor]. See side bar for what is meant by 'Mycoplasma' on this web site.

- http://members.aol.com/ghylak/supplement.htm

Father of all Antibiotic Mycoplasma Treatment

RHEUMATOID ARTHRITIS appears to be caused/assisted by mycoplasma infection and successful antibiotic treatment (80%) has been reported for 20 years. I believe that their experience may significantly benefit CFS patients. The following sites are particularly informative:

- http://www.rheumatic.org/
- NIH information [*]
- Radio Show Transcript:
  - http://www.power-surge.com/transcripts/scammell.htm

"*The most frequent reason for failure to respond to the protocol is lack of adherence to the* ***dietary guidelines***". Thus antibiotics are NOT a cure by itself, just a helper!

Editor note: A lot of Rheumatoid Arthritis medical mystery reads very much like CFS medical mystery...

Doxycycline Resistant Mycoplasma

Yes, there are some mycoplasma/infections that doxycycline does not have any effect on. Minocin (*Minocycline hydrochloride*) has been reported to been successful in these cases. (Note that unlike the other tetracyclines antibiotics, it tends not to cause yeast infections). Azithromycin is also suggested [*]

IV Clindamycin is also used - but it is expensive and must be given by IV.

Jarisch Herxheimer Reaction

Also know as "die-off", is the effect that many dead mycoplasma have on the body.... in short, the dead bodies causes additional thickening of the blood resulting in an "***apparent relapse***".

For additional resources on this reaction to antibiotics, see the following sites:

Asthma - another Mycoplasma Infection

Asthma has been reported to be cured by the use of doxycycline. As with rheumatoid arthritis and CFS, the mycoplasma may be either the maintainer of the illness or an immune-system disruptor (prevent a natural cure from occurring).

Other antibiotics used in place of doxycycline are:

- Ampicillin,
- Amoxicillin [*]
- azithromycin
- erythromycin [*]

'Walking Pneumonia'

Also know as Mycoplasma Pneumonia ..is the only illness that is completely accepted as being caused by a mycoplasma.

Simple description:

- http://www.drgreene.com/960205.html (note the section on contagious)
- http://www.foodsafety.org/ny/ny040.htm (note: blood tests often report false negatives - no mycoplasma found)
- Adam's
  - http://www.adam.com/ency/article/000082.htm
  - http://www.adam.com/ency/article/000082trt.htm

Treatment:

- erythromycin,
- clarithromycin (Biaxin),
- azithromycin (Zithromax), and
- tetracyclines (includes doxycycline)

Testing for Chronic Mycoplasma & Rickettsia

My own opinion (see *walking pneumonia* above) is that lab tests have too high an incident of false negatives. In addition to this, the actual bacteria could be in another family (CFS is a list of Symptoms -- many different types of bacteria could produce these symptoms). I would advocate the MDs consider 'testing by antibiotics' if the lab test reports negative --

- 30 days with doxycycline with 2400 GDU/day of bromelain: if no effect, then
- 30 days with another antibiotic listed above with 2400 GDU/day of bromelain...

At least until they have developed test that are 100% accurate for *walking pneumonia*.

Supplements to take with Antibiotics

Bromelain (pineapple extract) at 1000mg per day is suggested. It has known Antibiotic potentiation (makes them work better).

http://www.thorne.com/altmedrev/fulltext/bromelain1-4.html

Non-Antibiotic ways of fighting Mycoplasma

- Antioxidants as Vit. A,C,E (See Cheney O2 also), Mycoplasmas can not grow in oxygen rich blood
- Salmon oil & flax seed oil- prevent mycoplasmas from attaching to cell walls
- Colloidal silver kills mycoplasmas on contact in dishes. We personally find it works best for us as a nasal spray or in a nebulizer.

From Mycoplasma Registry for gulf war illness & chronic fatigue syndrome

Treatment

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Blood pH
Diet
Antibiotics
Blood Thinning
Supplements
Stress
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Ampligen

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International CFS Information

Updated on:
01/10/2000

The following is NOT medical advise, any treatment must be done in consultation with a qualified MD. This page attempts to simplify the issues for CFS patients suffering from "brain fog" (caused by Hypoxia?).

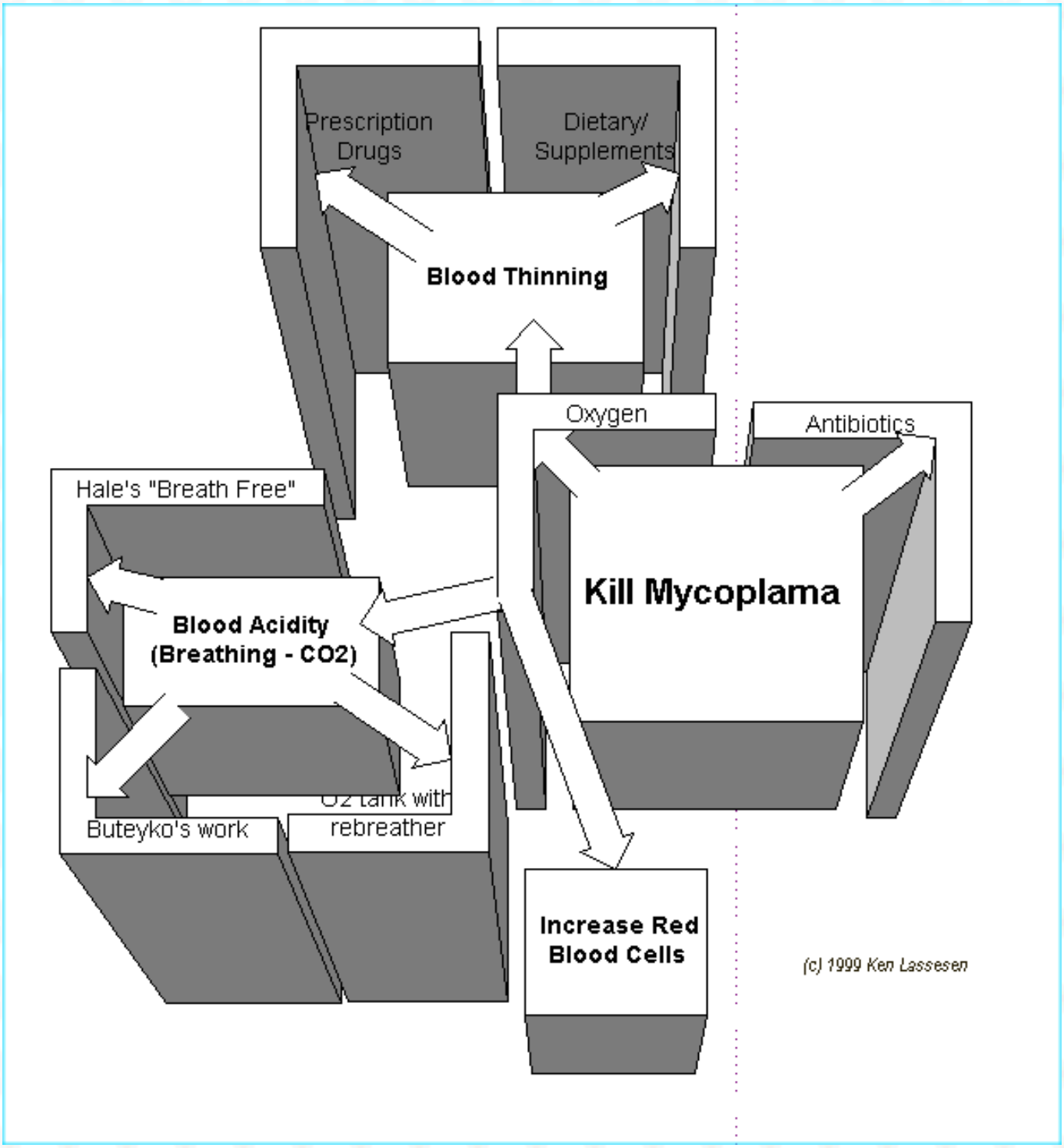
Treatment of IDEF/CFIDS may be done by prescription medicine or by supplements (non-prescription) and other 'alternative' techniques. If the supplements route is taken, the goal is to allow the body to heal itself [assuming that collateral damage from CFS is not severe enough to prevent this]. The prescription medicine either treats the symptoms or the underlying cause. The supplement route *may be required* to correct problems before a prescription medicine may be given effectively.

This page is derived from a specific model for the development of CFS. Treatments for 2nd and 3rd stage symptoms are excluded. The model and reported successful clinical treatments (defined as 50+% recovery without relapse) are in agreement. It may take *several* of the approaches to effect a full cure. Some CFS cases may not be caused by Mycoplasma infection -- the oxygen branch should still apply to those cases.

Full List of Treatments

For a more complete compendium of treatments see
http://www.cfs.inform.dk/Behandling/eng.beh.html

Basic Approach



Kill Mycoplasma

The *sustaining* cause of most CFS cases are mycoplasma infections. These infection cause a drop in blood oxygen saturation level (producing the initial symptoms) and causing slow long term damage to the entire body.

Mycoplasma hate oxygen and some antibiotics are effective against them - producing two methods of attack.

Antibiotics [90+% recovery]

Attacking the probable root cause: mycoplasma class of infections

For study see: http://www.haciendapub.com/article24.html,
~79% with *full recovery. Developed by Prof. Nicolson, a nobel prize nominee, a short bio, his website http://www.immed.org/*

AFTER CORRECTING vitamins and minerals problems (see below)

Start with doxycycline (200-300 mg/day) until there is no further improvement (may be 6+ months) then followed by 36 weeks of alternating the following antibiotics in 6 week cycles:

- doxycycline (200-300 mg/day),
- ciprofloxacin (1,500 mg/day),
- azithromycin (500 mg/day) or clarithromycin (750-1,000 mg/day)

Australian PWCs are using this treatment [more links]. The "die-off" effect of the antibiotics killing the mycoplasma will generally makes patients feel much worst... major discomfort for up to 12 weeks in some cases.

Oxygen

The primary cause of a drop in oxygen level is the thickening of the blood. This thickening may include 'filaments' which slices up red blood cells. The thick blood flows slower.

Blood Thinning [80+% recovery]

There are two forms of blood thinning:

Hemex ISAC Panel and Protocol

Hemex ISAC test and protocol (which address ALL forms of blood thickening) allows the blood to flow freely again resulting in the oxygen killing off the mycoplasma in mass and not allowing them to reproduce. We are taking serious drugs that require ongoing monitoring during treatment.

For study see: http://www.hemex.com/cfs/cfs_model.html
~80% positive for correctable ISAC condition.

- Blood thinners - after a positive ISAC Panel of tests, *low dosages of* heparin followed by warfarin.
  - (at least one study shows warfarin and aspirin may have equivalent therapeutic value [*])
  - CoQ10 should not be taken at the same time[*].

Aspirin and Dietary Protocols

This is based on the same theory as above, with significant results ONLY for those having major palette activation (< 30%). It is simple: take food/supplements that thin blood or improve circulation by widening blood vessels.

- Concord Grape Juice / Cranberry juice: at least 16 oz/day. (decreases blood stickiness etc, [latest research])
- Vitamin E - at least 1000 iu
- Vitamin C - typically orange juice, also supplies potassium, malic acid. At least 16oz/day

- ASA (Aspirin), one 325 mg tablet per meal, and at bed time (used as a non prescription *blood thinner*). [My history and why this is here] Other blood thinners are:
  - Garlic,
  - Ginkgo Biloba,
  - Cayenne, and
  - Bilberry (European Blueberry).

  WARNING: ASA should not be used for more than 1 week unless under a physician's direction. If there is not a dramatic improvement within 72 hrs discontinue.

Breathing Techniques

The body ability to release oxygen into the body is governed by its acid/alkaline balance. Carbon Dioxide (CO2) is an essential part of this process with too low a level of CO2 resulting in less oxygen being released. These techniques seek to increase the CO2 levels by breathing techniques or equipment so more oxygen is released. Some successes have been reported but no published studies for CFS patients.

NOTE: Deep breathing reduces the CO2 content and reduces the amount of oxygen that may be released.

Breath Free

Buteyko Techniques

O2 tank with rebreather

Production of Red Blood Cells

Low red blood cell count and deformed red blood cells have been reported as being common with CFS patients. The deformities may be caused by the filaments in the thicken blood. Since the red blood cell is what carries oxygen, then a diet rich in nutrients needed for red blood cells is essential.

The key component appears to be B-12 which is used for both producing red blood cells AND for removing toxins from the body. If the mycoplasma are producing too much toxins, there is little left for new blood cells.

Treatment of Deficencies

Note: patients with these chronic illnesses often have poor absorption [1]. RDA of vitamins may be insufficient.

Environmental Insults

- Treatment of symptoms and removal of possible catalysts that helps the illness
  - Food Allergy -- go on anti-allergy diet
  - Chemical Allergy and Allergies -- clean house, no perfumes, chemical air cleaners, etc (environmental insults)

Vitamin Deficiencies (or poor absorbtion)

- Vitamin B-12, essential for the development of red blood cell (help improve blood volume) - at least 750mg

- research])
- Vitamin E - at least 1000 iu
- Vitamin C - typically orange juice, also supplies potassium, malic acid. At least 16oz/day
- Minerals: zinc, magnesium, chromium and selenium are often depleted
  - Magnesium Malate Forte supplies magnesium and malic acid
  - Iron and Copper are often in SURPLUS..

Supplements that are used in the body's Energy Systems

- Use of NADH (a non-prescription co-enzyme) to reduce the fatigue symptoms (FDA confirms effectiveness for CFS)
- Enzyme CoQ-10, 100-300 mg/day (see summary), some believe it interacts with NADH to further improve symptoms. It is also reported as being depleted in CFS patients (compared to normal).
- Malic Acid (typically from Magnesium Malate Forte), also supplies magnesium (often low in CFS patients)

Other Prescription Treatments

AMPLIGEN

This drug once held great promise, but information leaking suggests that it is less effective *in general* than the above. It may be helpful when the above techniques do not work AND there is confirmation of a defective 37kDa RnaseL protein. For more information see:
http://www.cfids-me.org/redd/
for links, and
http://www.cfids-me.org/marys/redd.html

Affecting the immune system (symptom relief / improved self healing)

For information see: http://www.cais.net/cfs-news/ampligen.htm
< 5% with full recovery (?), 85% relapse rate after treatment reported.

- Use of Ampligen® (an expensive experimental prescription drug that is also effective for AIDS)
- Severe negative side effects have been reported for some PWCs. The FIRST patient has written a public letter on this drug, click here.
- Patients experiences (email if you know of more): http://www.geocities.com/HotSprings/Spa/4225/amp.html
- A current patient reports very good results and no side-effects http://www.cfids-me.org/marys/ampdiaries.html (reconfirmed in Jan, 2000)

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Characteristics

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Hypoxia
Physical Changes
Symptoms
Sleep
Outbreaks
Incidence
Testing Labs
Anesthesia

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National CFIDS Foundation

International

There are many characteristics or symptoms for IDEF/CFIDS. The short description is 'any symptom that may occur with a viral infection may occur with CFIDS - but instead of lasting one or two days, they may last for weeks, months or years'.

Note: Click on the [*] for information source/study.

Unusual findings with CFIDS

The following characteristics are reported to be observed in 50% or more of CFS patients:

- 70% test positive for rickettsiae [*]
  - 84% - to 96% recovery rate on antibiotics.[* C.Jadin]
- 93% has low Red Blood Cell Count [*]
- 53% has low Plasma Volume [*]
- 62% has low Blood Volume [*]
- 92% has a demonstrable hypercoagulable state [*]
- 96% has neurally mediated hypertension [*]
- 60% has a mycoplasma infection [* G.Nicolson]
  - 98% recovery rate on antibiotics

- up to 84% of cases: important improvement resulted after a minimum of six months of antibiotics [* P.Bottelo]
- maximal work capacity is 50% below normal[*. P. De Becker et al]
- 88% has a 37 kDa 2-5A binding polypeptide (compare to 28% of control population). The American Journal of Medicine: Volume 108 Issue 2 (February 2000) Pages 99-105
- An increase in IgG and IgM titer in the sera [*] 77% (compare to 12% of control population) **Human Herpesvirus 6: An Emerging Pathogen**

Some additional characteristics include:

- delayed recovery after exercise [1] , typically moderate 'over-doing it' will typically take days to recover
- development or worsening of chemical sensitivity, allergies, food sensitivity
- allergies/asthma worsen [3]
- drug sensitivity (often only 1/4 of the normal dosage is required)
- Occurrence: Adult 70%, children 30%
- A viral infection occurred prior to onset: 60%-80%
- Stress preceding the illness is evident in 67% of patients [Nancy Klimas at Brussel Conference, 1999]
- lost of up to 20% of IQ during the illness
- sleep disorders:[2]
  1. 90% CFS patients complain of unrefreshed sleep;
  2. 50% CFS patients take more than one hour to fall asleep;
  3. 60% CFS patients wake in the night for more than one hour.
- 25% patients with acute onset tested positive by PCR for enteroviruses, , while none did who had had gradual onset.[*]

For other characteristics see:

- Lyndonville Newsletter (site of 1985 Outbreak)
- Blood Volume Symptoms

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Personal Experience

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A Similar Prior Illness
Sudden Onset
A Relapse
Recovery

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International CFS Information

Updated on:
10/10/1999

I have made a very strong recovery since the onset of IDEF / CFS / CFIDS. Some factors that may have influenced recovery:

- from a prior illness (may have been mild version), I knew that I had to do a ***complete shutdown*** of all stress and pushing myself ***immediately***
  - the recovery behaviors that I had told to do for the prior illness (total de-stress, plenty of rest, do as much exercise as you feel comfortable with) appear to have been appropriate here
- my family physician was familiar with this illness and came to the diagnosis within 3 months, she has been a great support (also my wife, and family!)
- financial situation (Short and Long Term Disability pay, savings) removed financial stress
- belief from the prior illness that I will recover fully
- listened to what my body was telling me (and not what everyone around me was telling me!)
- read up heavily in the literature with a critical eye
  - NADH started, then dosage adjusted to 15 mg/day - taken with a walk
  - B-12 started (after NADH dosage established for 30+ days)

- realized that the best chance of recovery was in the first year (more chance of recovery at this time then there is for the rest of my life!)

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Model

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Stages
Notes
AutoImmune

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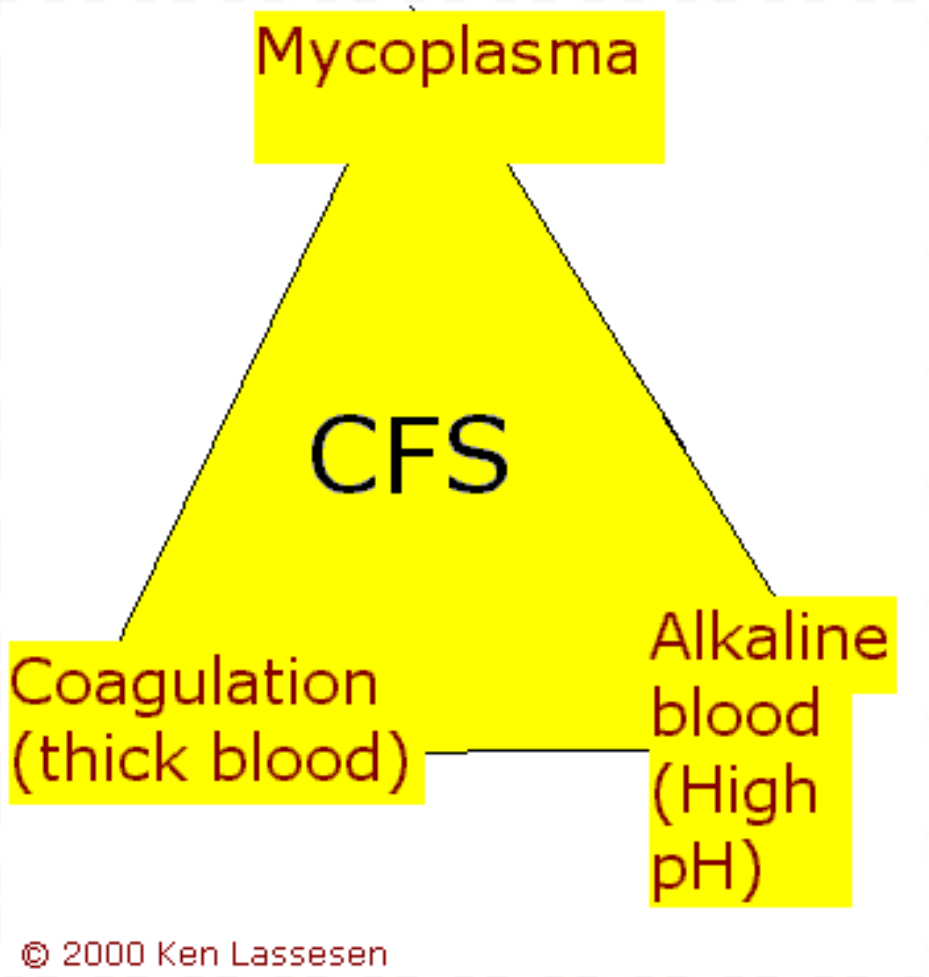
International CFS Information

Updated on:

A model is a description that appears to explain research and *which has predictive properties*. The following is my personal model - almost everything is based on findings that occurred in 1998 and later [hence it is likely unknown to most physicians who do not specialized in CFS].

The Trinity of Common CFS



Common means that which happens most often -- there are exceptions! The original starting point could be any of the three corners (or other causes) - but once CFS gets established, all three corners are likely to develop quickly! The best strategy appears to be to concurrently attacks all three corners at the same time since each corner helps the next corner!

Alkaline Blood

Alkaline blood occurs is less than 4% of the *general* sick population but in 80+% of CFSers [Acidic blood occurs over 70+% in the general sick population and some alt-medicine types have suggested that a universal cure is to make blood more alkaline].

Alkaline blood releases less oxygen creating an environment ideal for mycoplasma to thrive (they like low oxygen). Mycoplasma is around us, so it is an opportunistic infection. Alkaline blood can result in leaky gut and multiple chemical sensitivities. It reduces the body's ability to absorb food - resulting in deficiencies (especially nutrients needed for red blood cells).

Prior Symptoms suggesting Alkaline Blood

- Asthma
- Walking pneumonia (technically: Mycoplasma Pneumonia)
- Needing enzymes to digest food (especially meat)
- Multiple Chemical/Food Sensitivities
- Vitamin or mineral deficiencies that do not correct with supplements

Mycoplasma Infection

This is a bacteria that is half-way between a bacteria and a virus. It does not have cell walls so it cohabits with normal cells (without killing them). Mycoplasma infection appear to cause alkaline blood (? by releasing ammonia). "Thus, in the case of *M pneumoniae*, the host may be largely responsible for the pneumonia by ***mounting a local immune response*** to the parasite."[*]

Prior Symptoms suggesting Mycoplasma Infection

- Asthma
- Walking pneumonia (technically: Mycoplasma Pneumonia)
- Rheumatoid Arthritis

Coagulation

The body (probably as an ***Immune System response*** but may be other causes) starts to produce coagulation (thickens the blood). This thickening include fibers which slow down blood and also slices apart cells into odd shapes. With less blood flowing and *damaged or destroyed* cells, there is less oxygen available allowing mycoplasma to thrive.

Prior Symptoms suggesting Coagulation

- Poor circulation (cold feet or hands)
- Sensitivity to heat or cold

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Causes

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Lyme and CFS

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International CFS Information

Updated on: 01/08/2000

The cause of CFS is unknown -- the pathology of what causes the hyproxia-like symptoms may be one or more of several routes, a few of them are:

- low red blood cell count
  - because they are dissected/destroyed by coagulation elements
  - OR a genetic problem)
- slow moving blood (coagulation)
- malformed red blood cell cells (Dr. Les Simpson)
- alkaline blood (does not need coagulation)
- mycoplasma infection releasing an unknown into the blood system
- damaged ability to transport oxygen due to:
  - fluorine
  - mercury (from tooth amalgams)
  - nitrous oxygen imbalance
  - carbon monoxide poisoning

The agents that causes each of these pathology is unknown. Some cases may evolve into pathologies allowing other pathologies to occur. Some "causes" are suspected to be:

- RNA / DNA damage (found by Hemex labs, http://www.hemex.com/cfs/ and also Dr. Urnovitz)

- genetic predisposition
- bacterial or viral infection in the bone marrow
- opportunistic infection following a flu, viral or environmental insult to the body
- virus (see http://www.cfs.inform.dk/Virus/eng.virus.html for links)
  - a stealth virus (Jay A. Goldstein, MD) [*]
  - Re-activated Herpes virus HHV6
  - Borna Disease virus
  - Epstein Barr virus
  - cytomegalovirus
  - SV40 from Polio Vaccines
  - enterovirus
  - retrovirus
  - Coxsackie B virus

Note: The 'common cold' has over 100 known distinct virus that may cause it.

Many of the above pathologies may be self-maintaining (i.e. the original agent may no longer be a factor - thus a "cure" may mean correcting the pathology only).

Specialists

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CFS Friendly MD's

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Updated on: 12/17/1999

The following are specialists that some CFSers have given good reports about. This is informational and not endorsements - information about their *approach* is based on heresay (often patients). New patients should always discuss the MD's approach at the first visit.

Dr. P. Cheney

- Website: http://fnmedcenter.com/
- 1999 Transcript on Treatment
- Oxygen Therapy
- On exercise

One of the original physicians at the Incline Village Outbreak.

Dr. Patricia Salvato

- Houston, Tx, 713-961-7100
- Focused on glutathione production [ATP Injections /gth(gluathione)]
- Patients Improved with Glutathione, CFIDS Chronicle of Jan/Feb, 1998

- Klimas N; Salvato P; Morgan R; Fletcher M; "Immunologic abnormalities in chronic fatigue syndrome". J of Clinical Microbiology 28:1403-1410 (June 90) [Study showing that NK cells (a kind of immune cell) malfunction in CFS patients; other abnormalities]
- Strayer, D.R., Carter. W.A., Brodsky, I., Cheney, P., Peterson, D., Salvato, P., Thompson, C., Loveless, M., Shapiro, D.E., Elsasser, W., & Gillespie, D.H. (1994). A controlled clinical trial with a specifically configured RNA drug, poly(I)poly (C12U), in chronic fatigue syndrome. Clinical Infectious Diseases, 18(Suppl 1), S88-S95.

Diagnosis

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(Mis-)Diagnosis
RNA Changes
Oxygen
Urine pH
VO2-Max

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International CFS

Official Diagnosis

Symptomic analysis depends on:

- elimination of other possible causes,
- waiting six months, and
- having enough of the possible symptomns deemed significant.

There is much heated discussion on what the exact criteria should be as is shown by the following links.

- Meetings Held at CDC to Review CFS Case Definition

Research Diagnosis

There are several tests that have over a 80% chance of detecting a CFS patient, with < 2% chance of deeming a normal patient to be CFS. There seem to be significant resistance from the CDC in accepting these tests for diagnosis of CFS (***rumor*** *has it that Long Term Disability companies may be a factor here, and with the admitted (and unexplained) misdirection of CDC research funds from CFS research -- their liability exposure would be extreme if a test is accepted by the CDC [the*

Information

Updated on:
06/06/2000

numbers]).

- a very specialized blood test that measures the molecular weight of the RNase-L enzyme. For a description before the US Congress by the discoverer click here. For additional information click here. Results have been replicated by several labs.
- Analyses of the excretion of metabolites in the urine (pioneered in Australia) testing for CFSUM1 (more info, publication)
- The Immune System Activation of Coagulation (ISAC) panel for CFS from Hemex. (more info),
- Tests for low blood volume and low red blood cell counts (more info)
- Mycoplasma tests for *Mycoplasma spp*, this recent test was developed by Federal funds and with some refinement should evolve above the 80% cited above. (more info)
- RNA Changes on chromosome 22

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Recovery

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International CFS Information

Updated on: 12/12/1999

I've been asked to write up my recovery from CFS (my second), and after lots of thoughts of what to write, I came up with the following...

First, I was very fortunate with many of the early events centering around the onset of CFS. I knew what was the "insult" that allowed CFS to become established - ***stress***, I had a supporting wife and knowledgeable MD, I had excellent short term disability insurance, and I knew what was going on.

The early days

I knew that I was about to get sick 30 days before onset. I developed a dry-cough that had preceded my prior PVCF experience from 26 years earlier. I knew that I needed to de-stress, slow down etc... but failed to receive support from my manager. After reading an email from the boss denying me permission to look for another position in the company, I took sick that evening. The boss changed his mind the next morning -- unfortunately, CFS onset also arrived. I fell asleep at a stop light at 11am in the morning, headaches, nausea, dizziness plus night sweats, chills, etc. I was running at 1-2% of normal.

The first steps on recovery

There was a violent change in eating habits... from 10+ cups of coffee to zero cups (just thinking about a cup of coffee made me very sick), no donuts or anything sweets, no fatty food -- all were sudden repulsions... and there was this desire for peanut butter (without any craving for more when I had some). Unknown to me at the time, this appear to have been the right change of food! I kept to these habits even after the strong repulsion faded...

Because of my earlier experience with PVCF, I knew that I must immediately and *totally* de-stress. I used up all of my banked sick time (dropping off an important project that I was both the development team manager and main technical resource -- probably blowing away 4-6 man-years of work). It was not enough to undo CFS. Progressed on to short-term disability (hoping to recover enough in 6 months) -- believing it to be like my prior PVCF, just more severe. I maintained the belief that I would recover.

The search for knowledge

At 3 months, the family MD came to the diagnosis of CFS. Fortunately she has seen CFS patients for 17+ years and accepts it as an actual disease! In researching my symptoms, I also came to the CFS conclusion at the same time.

My research found that the odds of recovery decreased exponentially for each year that you are sick... so recovery is possible, but ***it must happen NOW***... every hour, day or week of delay makes recovery less likely.

The only treatment that had a 'government stamp' of approval was the use of NADH (verified by FDA). Ordered it and started at 10 mg, the same level as in the study. It worked: fatigue was less deep and had slightly more energy.

When I found myself bordering on depression, I re-read the study and found that dosage was still an unknown. Since I was 240 lbs, I suspected that a larger dosage would be appropriate and increased it to 15 mg. To my great relief, the depression disappeared within 48 hours.

When NADH no longer had significant improvement, I returned to the web and found that B12 was the most cited beneficial supplement. I started with 1000 mg/day of B12 tablets and 48 hours later there was significant improvement with some symptoms.

Two experimental protocols with good odds!

In reading everything I could on the web, I finally came across Prof. Nicolson and Dr. Berg's works/papers/institutes. Dr. Berg's work impressed me greatly and because there appear to be a genetic history of this illness in my family, I was going to press my MD to have the Hemex's ISAC Panel done at my next visit.

Fortunately or unfortunately, I had ongoing hassles with my LTD company and had an IME scheduled... which caused a relapse. My reading found that for some purposes, one of the Hemex drugs and aspirin were equivalent, and that aspirin flushes out of your system in 6 hours. So because of the relapse, and impatience waiting for the next MD appointment, I started taking one aspirin every six hours for 1 week (*which was ok to do according to the label on the aspirin box*). I was hoping that it would stop the decline that the LTD hassles had started... to my surprise, within 48 hours, I was bouncing off the walls... I was at 80% of normal compared to my prior *best* of 30% of normal... blood thinning worked! I know now that I was extremely lucky -- platelets were very significant in my case at that point of time.

Improved Understanding and more treatment

Now that I knew that blood thinning worked, I naturally went out to find other dietary blood thinners and add them to my diet. This included things to make blood vessels larger (thus healthy heart diet, concord grape juice)... "the more blood that flows and the faster that it flows, the better I will become..."

Reading about the low red blood cell count, I researched what was needed for red-blood cells and discovered that peanut butter had everything EXCEPT B12. Now I knew why I wanted peanut butter and why B12 works.

At my next MD appointment we discussed Dr. Berg's work and my miracle recovery using aspirin - and also Prof. Nicolson work on mycoplasma. I left with a prescription for 30 days of doxycycline (300 mg/day) because it was the least risk and simplest for the MD -- besides, I had recovered so much already!

Die-off and more ammo

I had a very easy time with die-off from the antibiotics [I suspect that blood thinning was a factor]. Only two weeks of it lasting all day, and then the length became shorter and shorter. 8 weeks

later, I would only get 'die-off' if I became physically active 30 minutes after taking antibiotics (nice way to remind yourself that the antibiotics are still needed!).

I am still on antibiotics (planning to do the full 12 months as recommended by Prof. Nicolson)

Additional ammo that I have added (which did *produced* a change) were:

- Hale's breathing
- Diet
- ... other items are in progress ...

Where Am I now?

At this point of time, I have no CDC symptoms, thus I 'am cure'. I do have symptoms that started with the sudden onset and are still with me. They are:

- ringing in the ears (no longer constant, slowly lessening)
- frequent urination (at least one each night that wakes me)
- skin rashes on foot (improved greatly with antibiotics)
- needing 8 hrs of sleep (instead of my prior 4 hrs)

Ongoing prevention

Since I do not desire to have a third round of this illness, the following are now permanent part of my life -- as prevention.

- Hale breathing exercise
- One baby-aspirin a day
- Avoid heavy stress (or medium for any length of time)
- Diet changes -- this will be a battle! I like my beef!

Insurance Issues

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Independent Medical Evaluation
Employment
Disabilities
Fraud?

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International CFS Information

Updated on:
12/26/1999

An illness that has no standard lab test to confirm and has over fifty theories can be a challenge to suffers because the theory that allows the claim to be disqualified will often be selected by Insurance companies to deny any claims (For an example, also see "Some Disability Insurance Companies May Discriminate Against CFIDS" by Mark Oring, Professor of Law, UWLA)

Need help?

The following links should be checked out:

- http://www.cfids.org/disability.html
- An excellent private list (disinissues) of people experienced with insurance issues. http://cfids-me.org/disinissues/list.html
- LTD: http://personal.riverusers.com/~searcher/help/sue.html

Long Term Disability (LTD) Insurance

The Society of Acturaries have made some numbers of Long Term Disability claims available on the net [1, Mar 19, 1999]. CFS is often classified as "Mental/Nervous" or M&N and not as "Other", as suggested in M&N rates for females being 50% more than that of males. Very dry and technical reading.

UNUM

UNUM and Duncanson & Holt are all part of the same

corporate entity. With D&H now handling LTD claims. [UNUM procedures for CFS Claims by Carolyn L. Jackson M.D; another possible UNUM document]. Duncanson & Holt manages LTD for by exclusively by Humuna . UNUM is attempting to sell off D&H.

Humuna had a class action lawsuit filed October 5th, 1999 against it for Medical Insurance that alleges "bonuses were based principally on the number of claims that were denied" and "paid direct financial incentives to physicians to deny coverage". It is not unreasonable to suspect the same practice may be occurring with LTD coverage handled by the same corporation.

The following links show that this is changing due to various court decisions and law changes. This process is being helped by recent medical evidence supporting that it is a distinct physical disease. The following is not legal advise.

Canada

"The March decision in Edmonton Court of Queen's Bench is the first case in Canada to recognize chronic fatigue as an organic illness and not the product of a psychiatric disorder." April 22, 1998 [1] (see below for US equivalent). [Full Text]

United States

A list of decisions from Circuit Courts is available online. These are technical documents that require experience in reading law documents.

SSA guidelines for Chronic Fatigue Syndrome.

'Mongeluzo suffers from a disability [CFS] not "caused by mental illness or functional nervous disorder," as we have construed those terms.' U.S. 9th Circuit Court of Appeals [5]

'The district court also determined that the accommodation Kennedy requested, a "work-when-able" work schedule, was unreasonable as a matter of law.' [2] Echoed in REDDICK v CHATER, U.S. 9th Circuit Court of Appeals, reasons for judgement (at end):

> "ALJ: If I were to credit the claimant's testimony concerning her fatigue and pain, would the claimant be able to perform her past relevant work?
>
> Vocational Expert: No, she would not.
>
> ALJ: Why is that?
>
> Vocational Expert: Because of the need to take frequent naps during the day. Some days unable to get out of bed to get to work. Performing repetitive

tasks, such as using a computer, would probably increase the fatigue that is being described.

ALJ: Okay [Claimant's attorney], any questions of the Vocational Expert?

Claimant's Attorney: Just one. If she was limited to her bed even one day a week, only one day a week, would your answer still be the same about her inability to perform her past relevant work or any other work?

Vocational Expert: Yes." [4]

"It is now widely-recognized in the medical and legal communities that there is no `dipstick' laboratory test for chronic fatigue syndrome." Sisco v. United States Dep't of Health & Human Services, 10 F.3d 739, 744 (10th Cir. 1993). " cited in May 8, 1997 UNITED STATES COURT OF APPEALS FOR THE THIRD CIRCUIT, Reasons for Judgement [3] which ends with

"it would defeat the legitimate expectations of participants in the Kodak Plan to require those with CFS to make a showing of clinical evidence of such etiology as a condition of eligibility for LTD benefits. Thus, it was arbitrary and capricious for the Administrator to deny Mitchell benefits because of a lack of such clinical evidence of the etiology of his CFS."

United Kingdom

'The Industrial Tribunal found that ME constituted a "disability" for the purposes of the DDA' [4]

Resources

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News Groups
Advocacy
Epidemiology
Schools and CFIDS/ME
Town Hall
Sydney

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National CFIDS Foundation

International CFS Information

Updated on:
03/01/2000

- An excellent collection of links to other sites: http://home.netinc.ca/~kcoleman/mess/
- David Axford's Myalgic Encephalomyelitis / Chronic Fatigue Syndrome **** An excellent resource of current literature maintained by Medical Professionals!
- LATEST NEWS about chronic fatigue syndrome
- Dr David Bells' Lecture - Feb 98
- Chronic Fatigue Syndrome
- WI CFS Articles Page
- Jen's CFIDS/ME Research
- Chronic Illnet Report 6
- Correlations Between CFS and AIDS II
- Chronic Fatigue Syndrome
- CFS Radio Show Index - interviews with leading researchers

New or Novel Treatments

- http://www.shasta.com/cybermom/novel%20tx.htm

Other Recommended Links

Ask NOAH About: CFS

Comprehensive guide to all aspects of CFS; a good place to start learning about the illness.

BBC Medical Notes on ME

From the UK's BBCNews site, a good overview of myalgic encephalomyelitis (or CFS).

CFIDS Acronyms

Use Robyn Pollman's handy list to make sense of all the CFS-related "ABCs."

CFIDS Symptoms

Easy-to-read summary of symptoms, from Katrina Berne's book, *Running on Empty*.

"CFIDS vs. Chronic Fatigue"

A brief, but worthwhile distinction between CFIDS illness and the symptom of "chronic fatigue" associated with many conditions.

CFS Big Picture

From *Prevention Magazine* online, some general information about how many people may have CFS.

CFS Case Definition

Abridged version of 1994 Center for Disease Control's definition of Chronic Fatigue Syndrome.

CFS Demographics

A look at the population and prevalence of CFS sufferers, plus speculation on whether it is contagious and how many people recover.

CFS Fact Sheet

This government publication provides an excellent overview of CFS.

CFS/ME Fact Sheet

Mary Schweitzer's overview includes information about the name, demographics, duration, and possible etiology.

Chronic Fatigue Syndrome

A worthwhile summary of CFIDS from the Environmental & Preventive Health Center.

Chronic Fatigue Syndrome In Men

Good overview of CFS and its unique impact on male sufferers.

Disease of a Thousand Names

Compiled from the work of several doctors, these are just a few of the names that have been given to this dreaded disease.

Facts About Chronic Fatigue Syndrome

US Centers for Disease Control and Prevention (CDC) wrote the text of this comprehensive booklet, including common treatments.

Facts About CFS From CDC

Center for Disease Control Media Relations provides this concise statement; a good brief summary.

Fatigue Severity Scale

A useful tool for differentiating severe fatigue from depression; CFS sufferers score much higher than depressed patients.

Fibromyalgia: CFS by Another Name?

An informative look at the question of how these illnesses might be related.

FM/Related Illness Pamphlet

This overview notes the relationship between Fibromyalgia/CFS and other similar disorders; also available in French and Danish.

Glossary of Terms

A helpful listing of medical and scientific terms often encountered in relation to CFS.

"Have I Got Chronic Fatigue Syndrome?"

An informative patient leaflet from a family practice center in the UK, intended for British patients, but helpful to all.

Information in Other Languages

From Moira Smith's excellent Web site, a list of CFS links in many languages.

Japanese CFS Page

From Kobe City, Japan, this website provides CFS research and information in Japanese.

M.E./CFS vs. Depression

A chart summarizing written psychological tests that show the differences between M.E./CFS and depression.

"Medicine For the Public"

This publication from the National Institutes of Health has a good discussion of CFS, including it's history and the role of medicine.

Myalgic Encephalomyelitis/CFS

From Amsterdam Kliniek, an overview of ME/CFS, including cause, diagnosis, and treatment.

"The Mysteries of CFS"

From *WebMD*, an excellent article on the complex disorders that may be involved in Chronic Fatigue Syndrome.

Overlapping Disorders

Discusses the similarities *and* differences between CFS, FMS, MCS, and GWS; well-written and to-the point.

Prevention on CFS

From Prevention Online's easy-to-understand site, a brief but telling description of Chronic Fatigue Syndrome.

Questions & Answers about CFIDS

Mediconsult.com provides information on a wide range of medical topics; their discussion of CFIDS is excellent.

"Understanding CFIDS"

A thorough and well-written information pamphlet from The CFIDS Association.

"What is CFIDS/ME?"

Myalgic Encephalomyelitis (M.E.) is the name used for CFIDS in the UK; this is a good summary for newly-diagnosed.

What Is CFS?

A good, concise overview; especially helpful to those who may not be familiar with CFS.

What Is CFS/ME?

From an excellent website available in both English and Danish, a well-written overview of how this illness is defined.

"What is Chronic Fatigue Syndrome?"

Chapter 1 from Jacob Teitelbaum's book, *From Fatigued to Fantastic*, provides an excellent explanation of CFIDS.

What Is M.E.?

Myalgic encephalomyelitis (M.E.) and CFS are not the same entity, according to these articles defining M.E.

"What is M.E. Like?

Young ME/CFS sufferers explain what it's like to have this illness, in their own words.

Find more information on CFS Diagnosis...

For MDs and RNs

Dr. Cecile Jadin's Papers are now available in full Click Here

First Tests

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National CFIDS Foundation

International CFS Information

Updated on: 06/18/2000

This page is for MDs and RNs that are 'new' or 'out-of-date' to handling CFS patients. There are many constraints on you: What insurance will coverage, following conventional medical practice ("no quackery please"), risk to patient and personal belief systems. We assume that all of the conventional tests have been done....

Level One Follow up

- Bacteria Causes?

  Warning: multiple infections from multiple families is not uncommon. The first infection usually makes the body friendly for others of these groups.

  - Check for titers for following bacteria: (*less reliable but easier to get)*
    - Mycoplasma
    - Rickettsia [or Weil-Felix test]
    - Spirochetia
    - Chlamydiae
  - [if no positivies] Have PCR testing for following bacteria (if available):
    - Mycoplasma
    - Rickettsia [or use Giroud's Micro-Agglutination testing]
    - Spirochetia
    - Chlamydiae

- [if no positivies above] Prescribe Doxycycline at 300mg/day for 30 days, remember to warn patient of possible Herxheimer effect.
  - If patient feels better OR has a herxheimer effect, see literature on mycoplasma infections and review Prof. Nicolson Research AND also see Rickettsiae infections and review Dr. Jadin's Protocol [probably the better one].
  - Have a LUAT test for Lyme Disease (see Florida CFS results). The antibiotics must be prescribed to test using the LUAT test.
  - If no effect, add bromelain (2000GDU+) for one more week
  - If still no effect, discontinue
  - If you continue with antibiotics, then when the patient stops having herxheimer effects, consider adding Bromelain supplements (it is used in Europe as an antibiotic potenator) - patient may experience additional herxheimer effects when they start it so start with low dosage and increase over time.
  -
- If patient has a low (0-5) SED.
  - Immune System Activation of Coagulation is likely,
    - Do the ISAC Panel on the patient (See http://www.hemex.com/cfs/)
    - If not possible ($?), suggest Bromelain (a Pineapple extract available at health food stores) at 1000-2500 GDU/day for a month. If no improvement (SED rate OR personal report) - discontinue. See http://www.thorne.com/altmedrev/fulltext/bromelain1-4.html

Specific conditions to check for

- Does the patient takes enzymes or other digestive aides? Yes
- Does patient take ibuprofen regularly? Have multiple chemistry sensitivity? multiple food allergies? Yes
- Check the level of all of the patient's minerals, prescribe supplements as needed.

Level TWO Follow up

The items below are intended to eliminate rarer diseases that could be misdiagnosis as CFS (matching symptoms); or forms of CFS that are more *experimental* or expensive to test and treat.

- RnaseL Enzyme Dysfunction Disease (REDD form of CFS): Test for low molecular weight (37kDa) RNase-L (this must be done by European labs), if positive than Ampligen is a possible treatment. Suspected to be more common in long term CFS patients.
- Hemochromatosis (iron overload) (See http://www.cfs.inform.dk/Borreliosis/hemochrom.html)

- Chiari (spinal cord compression) - following symptoms may be indicators:
  - Headache in the back of the head that may radiate behind the eyes and into the neck and shoulders
  - Lack of gag reflex and difficulty swallowing
  - Symptoms are exacerbated by exertion, and especially by leaning the head backward or coughing
  - Chiari patients reported 60% to have had a prior diagnosis of fibromyalgia and 12% of chronic fatigue syndrome.
  - 20% of the fibromyalgia patients had cervical *compromise*.

Additional Information

Use the search engine on the National Library of Medicine:

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?CMD=search&DB=PubMed

PS: Although I am not a professional medical person, I do have a Master of Science, taught science at High School and College, and have worked as a medical statistician.

Genetics

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International CFS Information

Updated on: 02/03/2001

At the 2001 AACFS meeting in Seattle, there were several posters that appear to confirm that pre-desposition to CFIDS is genetic.

Poster: #121 Exercise Capacity in Monozygotic Twins Discordant for Chronic Fatigue SyndromeRobert Schoene, MD, Debra Buchwald etc

Study:

- 22 IDENTICAL TWINS, one with CFS and one without CFS were examined.
- Only significant difference between them was a slightly different VO2-max level
  - Twin with CFS: 17.8 cc/kg/min SD: 3.1
  - Twin without CFS: 19.8 cc/kg/min SD: 3.7
  - Probability of significant 0.021
- Very STRICT selection criteria for CFS in one and no sign in the other.

"***The most remarkable finding was the abnormally low VO2-max in both twins that was only slightly higher in the unaffected twin.***" from Summary on Poster

The VO2-max for the equivalent normal population is 34-38 cc/kg/min.

In correspondence with one of these twins: "I haven't yet gotten the money together to get ISAC panel tests but sed rate for the last 15 years has been 0-1 and one of my brothers (who doesn't have CFS) appears to have a *coagulation problem*."

Genetic Predisposition to Chronic Fatigue Syndrome and Fibromyalgia?, Results from UofW, http://www.cfs.inform.dk/Hypoteser/geneticpredisp.aug00.html

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Updated on:
06/08/2000

Text Search

If you are having difficulty re-locating material, please try searching for it here.

Use the form below to search for documents in this web containing specific words or combinations of words. The text search engine will display a weighted list of matching documents, with better matches shown first. Each list item is a link to a matching document; if the document has a title it will be shown, otherwise only the document's file name is displayed. A brief explanation of the query language is available, along with examples.

Search for:

Start Search Clear

Query Language

The text search engine allows queries to be formed from arbitrary Boolean expressions containing the keywords AND, OR, and NOT, and grouped with parentheses. For example:

information retrieval

finds documents containing 'information' or 'retrieval'

information or retrieval

same as above

information and retrieval

finds documents containing both 'information' and 'retrieval'

information not retrieval

finds documents containing 'information' but not 'retrieval'

(information not retrieval) and WAIS

finds documents containing 'WAIS', plus 'information' but not 'retrieval'

web*

finds documents containing words starting with 'web'

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First Tests

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Updated on: 02/14/2000

CFIDS is a diagnosis of elimination by the usual medical tests. Once these tests are done, we enter the world of CFIDS specific testing:

There are a tons of things that can be tested for etc.. some results in information / confirmation of a problem. Others tests lead to direct treatment of factors that could be a significant part of your illness. The latter (tests that results in treatment that are known to correct specific problems) are listed below:

- Test: Immune System Activation of Coagulation Panel: Treatment: Prescription blood thinners Info: http://www.hemex.com/cfs/
- Test: Mycoplasma PCR Testing Treatment: Long term antibiotics Info: http://www.immed.org/
- Test: Rickettsia PCR Testing Treatment: Long term antibiotics Info: http://www.folkarts.com/idef/rickettsia.htm
- Test: Lyme Disease Testing (LUAT etc) Treatment: Long term antibiotics Info: http://www.igenex.com/lymeset7.htm
- Test: Chlamydia PCR Testing Treatment: Long term antibiotics Info: http://www.immed.org/

All of the above appear to have a 80+% success rate for very significant improvement or full recovery, if any test results are positive. Four out of five treatments above use the same antibiotics - and in some cases (my own for example), some MDs are willing to give the antibiotics a short term trial (30-60 days) without any testing to identify the exact infection [many MD's gives the same prescription for Acne, and CFIDS is far more serious than acne]. If the patient improves significantly OR experiences herxing (see http://www.folkarts.com/idef/herxheimer.htm for what this means), then one of them is assumed present and one of the long term antibiotic protocols are started (Nicolson's or Jadin's). If you

test by using antibiotics, the use of bromelain (a digestive aid) with the antibiotics is suggested to encourage herxing to show itself... The base antibiotic is one of the twenty most prescribed drugs in the US and is often prescribed for Acne... so it's very low risk and not an experimental drug etc.

Then there are a few supplements etc that are generally accepted as effective helping most CFIDS: B12 (large dosages), B-100's, NADH and whey (see http://www.folkarts.com/idef/cheney_whey.htm for an introduction).

After that, you step into the world of experimental drugs, testing to confirm problems, higher risk drugs, specific symptom treatments etc...

Herxheimer

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International CFS Information

Updated on: 06/11/2000

The Jarisch-Herxheimer reaction (referred to as "Herx" often) is believed to be a reaction caused by organizisms (bacteria) dying off and releasing toxins into the body faster than the body may comfortably handle it. It was originally observed in patients with syphilis who received mercury treatment [*].

Most CFSers will have moderate to severe herx effects from antibiotics that will usually have no effects on normal healthy individuals[83% in one R.A. study, 51.4% in Lyme Disease, 89% with B. recurrentis infection]. The reason for this may be the diminished blood flow system caused by the thickening of the blood commonly found with CFS patients. Alternatively, "it seemed to occur when injured or dead bacteria released their products into blood and tissues, provoking a sudden and exaggerated inflammatory response"[*] - thus the response may be connected to the *specific organism* being killed off. Many non-antibiotic treatments (Hale's breathing, glutathione - whey products) are reported to also produce a herx effect -- whether it is bacteria die off or simply toxin release, the effect is the same: misery!

Readings:

- ***What is a Herxheimer Reaction?*** http://www.x-l.net/Lyme/HERX.html
- The Herxheimer Reaction History [History and current theories (technical)] http://garynull.com/Documents/Arthritis/Herxheimer_Effect.htm
- The New England Journal of Medicine -- August 1, 1996 -- Vol. 335, No. 5 http://www.nejm.org/content/1996/0335/0005/0347.asp
- http://www.cmc.net/~jadevoll/herxheimer.htm

Onset

- Depending on illness and antibiotic: from 1-2 hrs to 10 days after antibiotics started

Symptoms

The most common ones include:

- increased joint or muscle pain
- headaches

- chills
- Heavy perspiration and night sweats
- Nausea
- Burning micturition
- Bone pain
- swollen glands
- bloating
- constipation or diarrhea
- fever (usually low grade)
- hypotension (low blood pressure)
- Itching, hives and rash (sometimes mistaken for an allergic reaction - this must be an MD's call)
- heart palpitations, elevated heart rate, orthostatic Tachycardia are reported on http://www.onelist.com/community/cfs_Mycoplasma/

Treatment

- Probenecid (Dr. Jadin [*])
- Benadryl (antihistamine)
  - Therma-Flu or equivalent
- Aspirin (Bromelain may increase it!)
- Meptazinol [*, *]
- Increase in blood thinning supplements
- Lemon/Olive Oil drink (may be done with water or Grape Juice)
  - 2 Tbsp Lemon Juice (Organic) or 1/2 lemon rind)
  - 1 Tbsp cold pressed Extra Virgin Olive Oil
  - Water or juice ... stir
- Concord Grape Juice with flavoids (widens blood vessels)
- NSAIDs (non-steroidal anti-inflammatory drugs),
- pain medication (see ibuprofen risks..)
- muscle relaxers,
- hot baths / hot tub
- steam (dry or hot) sauna
- a saltz bath: add 1 cup salt, 1 cup soda, 1 cup epsom salts, 1 cup aloe vera, to a hot bath, remain in and keep hot for about 1-1/2 hours all the while consuming about 2 quarts of warm water.
- Hale's breathing
- Control panic attacks, anxiety and worry (they constrict the blood vessels, worsening the effect).

Differentiating between a Herxheimer, a flare and an allergic reaction to the drug

"Laboratory tests can help differentiate between a worsening of disease (RA flare), a Herxheimer reaction to microbial toxins, and an allergic reaction to medication.

1) WBC will **elevate** in a Herxheimer and **lower** in a flare.

2) A **Herxheimer** will also exhibit a coincidental **elevation of SED rate, gamma globulin and total globulin,**

and a fall in serum albumin and hematocrit. Patients who exhibit this flare reaction accompanied by **anemia, depression of serum albumin, elevated total globulin and gamma globulin** are probably reflecting a **more intense reaction pattern to anti-L substances** than in hematologically mild cases.

3) **A marked increase in eosinophils** (for instance about 30%) is an indication of an **allergic reaction to the drug."**

From
http://home2.freegates.be/nvdeynde/mycoplasma/publications/mycoplasma/treatment/8_bestanden/8.pdf

Blood pH

Dr. Cecile Jadin's Papers are now available in full Click Here

Cheney O2 Hypochlorhydria

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Updated on: 02/25/2000

The body ability to release oxygen into the body is governed by its acid/alkaline balance. Carbon Dioxide (CO2) is an essential part of this process with too low a level of CO2 resulting in less oxygen being released. These techniques seek to increase the CO2 levels by breathing techniques or equipment so more oxygen is released. Some successes have been reported but no published studies for CFS patients.

Dr. Cheney sample report on oxygen consumption of CFS patient illustrates this low oxygen consumption (see oxygen.htm)

NOTE: Deep breathing reduces the CO2 content and reduces the amount of oxygen that may be released.

Blood pH and Breathing

A blood pH between 7.35 and 7.45 is normal

- http://www.acp.edu/web/genchem/thedisk/bloodbuf/zback.htm]
- http://www.acp.edu/web/genchem/thedisk/bloodbuf/zback2.htm
- http://www.nursing99.com/ce/p812a.htm

IMPORTANT NOTE: (most) Asthma and (most) CFS cases appear to be caused by Mycoplasma infections. This technique affects the mycoplasma *by killing them with higher oxygen levels*.

Poor Man's Rebreather

Wearing a {painter's, organic vapor or dust mask} for several hours a day seem to be effective in raising CO2 level. Some patients have reported "herxing" if they wear it a long time each day (in theory, the CO2 increase causes more oxygen to be released and thus killing mycoplasma-like bugs [oxygen becomes an antibiotic]). It's cheap,

does not require a prescription and simple to use. :-)

Buteyko / Hale Breathing

The purpose of these exercises is to correct long term "hidden hyperventilation". A 'point form' description is available at "Anne and Janet's introduction to The Buteyko Method", http://www.wt.com.au/~pkolb/a&j.htm.

A "popular magazine report", AUSTRALIAN WELLBEING, 1997, 68:86-93
http://www.buteyko.com/media/healthy_breathing.html

For a good scientific study (1981) see http://www.buteyko.co.nz/buteyko/trials/frames/russia.htm , this is linked to a lot of New Zealand and Australian experiences. Another site is http://www.buteyko.com/trials.html

The Control Pause (CP)

This is the key monitoring component for retraining yourself to breath correctly. As a first step, you should read several description of this measurement and derive your own version. For example, if you cannot sit in a chair comfortably, use a recliner etc..

This CP is described by Teresa Hale as follows:"

1. Sit comfortably in an upright chair close to a clock with a second hand, or hold a stopwatch.
2. Relax and breathe in and out gently, mouth closed.
3. Pinch your nose with your fingers, after the exhalation.
4. Keeping your mouth closed, count how many seconds you can comfortably last before you need to inhale again.
5. Don't push yourself too hard. The accuracy of the test depends on your stopping before you reach the threshold of discomfort.
6. Remember: you are not holding your breath in, you are emptying your lungs and then counting.
7. When you breathe in again, try not to take in large gulps of air, control your breathing, keep your mouth closed.
8. Do *not* push your Control Pause above 60. This can only be done under the guidance of a Breath Connection Practitioner. These breathing exercises, like medication, must be administered correctly" p. 48, ***Breathing Free***

Alternative versions are available online at:

- Instep Asthma Free, http://www.nqnet.com/buteyko/buttest.html

- The Buteyko Breathing Centre
  http://www.buteyko.co.uk/buteyko/theory.htm
- Chicago Yoga News, March 1999
  http://www.yogachicago.com/march99/Yoganews.html

Re-Training your breathing

All of the techniques are modification of the following pattern:

1. Check pause for 1 minute (helps to settle you down)
2. Do a Control Pause (CP)
3. Do shallow breathing for 5 (or more) minutes.. REPEAT 2,3
4. take a slightly bigger breath, exhale. Try to hold extend your CP for 5 additional seconds. REPEAT 3,4
5. Increase to 10 additional seconds (but never over 60 seconds).

This is done before each meal and at bed time (or more often if the situation permits it).

A modification that Hales teaches for children is to walk while holding the exhausted breath and count the number of steps taken (ideally 120 steps). Then continue walking in a normal manner (with shallow breath) and repeat in 5-10 minutes. For people who do walk regularly, this method may be helpful.

Reference: Breathing Free, Teresa Hale, *Harmony Books*, 1999

Oxygen tanks with rebreather etc.

CFSFMExperimental reports that breathing oxygen did not improve CFS, and may actually cause relapse. Oxygen using a rebreather(to increase CO2) has been prescribed by Dr.Cheney and several people are trying this.

Hyper baric Breathing

CFSFMExperiment have several people waiting to spend some time in hyperbaric chambers (decompression chambers) where the atmospheric pressure will be increased several fold. The theory is that this will allow more oxygen into the blood (note that this is normal air that is pressurized).

Other Oxygen Therapy...

This is informational only -- no reports of western physicians trying or suggesting this (one report of Eastern European physician).

http://www.oxytherapy.com

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

Diet

Dr. Cecile Jadin's Papers are now available in full Click Here

Frugal CFSer
Red Blood Cells
Blood Thinning
Catalysts
Supplement Schedule
Diet pH
Peanut Butter
Anticoagulants
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National CFIDS Foundation

International CFS Information

Updated on:
02/06/2000

New and Revised!

I am NOT a "diet person", but have accepted that diet may play a very significant role in recovery... the basis of the diet shown below are from the following sources:

- recommendations for treating Acute Mountain Sickness [AMS]
- recommendations from Teresa Hale's book "Breathing Free", Harmony Books, 1999 [TH]
- recommendations from antibiotic treatment for Rheumatoid Arthritis (http://www.rheumatic.org/) [RA]
- reported food aversions from CFSers early in the disease [FA]

In going thru these alternative diets, I made *personal judgment calls*. I've attempted to identify them below.

General Information

- Breakfast: Porridge (Barley flakes, oats etc - no wheat), (no added sugar) cereals with rice milk [TH], (no corn or wheat cereals)
- Lunch: should include small amount of protein (lamb, chicken, fish) and salad [TH]
- Supper should be low or no protein (no meat, cheese etc) [TH]. Example: Rice with stir-fried/steamed mixed greens (we walk thru the leafy dark green section and just chop up everything to create a mix)

Do not use microwave on food.

Foods

+ - should have, - should not, blank means no information

Food	AMS	TH	RA	FA
- Alcohol	-	-		-
- Tobacco	-			-
- Coffee	-	-		-
- Soda Pop	-	-		
- Fatty food (No margarine or trans fat)	-	-	-	-
- High protein intake (Meats, fish)	-	- [a little lamb, chicken, fish is ok]		
- pork			-	
+ cold water fish		+	+	
- shell fish			-	
- Dairy		-	-	
- Soy based		-	-	
+ dark green vegetables (avoid lettuce)		+	+	
+ rice		+	+	
- potatoes		moderate	-	
- refined sugar, foods with sugar added		-	-	-
+ buy organic		+	+	
- nutrasweet, aspartame and other artificial sweeteners				-
+ oats, millet, barley			+	
- wheat, corn			-	
- carrots (sugar content)		-		
- pork			-	
+ beans and legumes (includes peanut butter)			+	+
- nuts				-
- black, white and green pepper / peppercorn				-
+ Salt (for NMH)				

Positive Foods

Juices

Warning: Anyone with a Candida infection should not drink any fruit juice.

Juices are a 'dilemma' - they are high in (unrefined) sugars but also significant positive effects. The following four juices should be considered.

The ideal juice will be unsweetened, not from concentrate and organic. Juices supply potassium (CFS are often low) and water volume (that helps blood volume).

Min	Food	Medical Condition	Explanation
8 oz	Orange Juice	Low energy / Chemical deficiencies	• High in Malic Acid (Energy chemical) • High in Vitamin C
8 oz	Apple Juice	Low energy / Chemical deficiencies	• High in Malic Acid (Energy chemical)
16 oz	Grape Juice Cranberry Juice	Thick Blood	• Increases blood vessel size • thins the blood

Foods

As with juices, organic (so chemical sensitivities issues are not triggered) and with the least (or no) additives. I use Adams 100% Natural Peanut Butter.
Warnings:

- Many peanut butters are flavored margarine... check the ingredient list always!
- Some people are *allergic* to peanuts (allergy testing should be considered if you are in doubt). Peanuts are not nuts.

Min	Food	Medical Condition	Explanation
2 TBSP	Peanut Butter	Low Red Blood Cell count	• rich in material needed to produce red blood cells

Negative Foods

A low cholesterol diet is suggested, since CFS reduces the capacity of the blood system you wish to improve the blood flow by eliminating other problems.

Food	Medical Condition	Explanation
Coffee	Low Blood Volume	hydrophilic (absorbs water) - reduces blood volume further caffeine may adversely affect blood chemistry [Conj]
Fatty foods	Thick blood Low Blood Volume	fat requires a lot of fluid to process -- drawing it from the already low blood volume fat can thicken blood vessels reducing blood flow further.
Smoking	Thick blood Low Blood Volume	reduces capacity of blood system further

Blood Thinning

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Hemex Protocol

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National CFIDS Foundation

International CFS Information

Updated on: 08/05/2000

The majority of CFS patients appear to suffer from thick blood. This is a treatable condition which seems to cause near-complete recovery in most patients. This is not a 'one-researcher' treatment as seen by the following quote...

Quick Indicator: LOW SED

Sed rate or ESR means erythrocyte sedimentation rate. It is a laboratory test calculating how far red blood cells settle in a test tube in one hour. (Elevation of the erythrocyte sedimentation rate can be caused by any type of inflammation from arthritis to burns, infections, myocardial infarctions, malignancies, fractures and tissue damages). A LOW SED is often ignored by MDs. In "Osler's Web", p.214, Dr. Cheney reports that his patients were always in the 1-3 rate (normally only HIGH SED rates are *clinically important)*

A sed rate between 0-5 has been hypercoagulation.

UK Research

"...as we reported in our fall edition [1999] of The National Forum, when David Berg spoke at a world conference on thrombology, another much larger and world famous group of hemotologists from London had also announced on the same work they had done in England with 200 ME patients. Without knowing what the other was doing, they had, in effect,

replicated each other's work! But Hemex has gone much further and has tests that are for genetic markers. He can tell, many times, who will repond quickly and who will not from these tests. Of course this work is experimental. It needs a lot more testing. David Berg has spent more than $100,000 out of his own pocket. There aren't many researchers who would even think of doing this without some personal connection to CFS and he has none. He didn't even know about it until he tested his first patient! Heparin is not only a vasodilator, but also acts as a microbial as well as an antiviral by stopping the pathogen's ability to make fibrin. Those that the program does not work in are found to be extremely severe and have a genetically found hypofibrinolysis. He's now concentrating his research on these very severe patients. He has had samples sent to him by some of the most famous CFIDS experts in the country. Research takes a long time to arrive at all answers, but the information you wrote upon was outdated already!"

"..the study that you refer to was the original one that was presented at the 1998 AACFS conference in Cambridge, MA. The National CFIDS Foundation was not very impressed with it, either. But we followed it up three months later and found that 90% had responded. We were impressed then. It was then that we funded a small pilot trial with David Bell supplying the blood samples. During the initial findings, which you refer to, all of the patients were taken from one rheumatologist's office after one patient was tested and ***the results of her treatment were fairly miraculous***. All those tested had been diagnosed with FM but the ones not reported on that took longer to for it to take effect actually had CFS/FMS. The rheumatologist had only given them the DX of FMS but did say they had both when he referred them. Since then, many, many more patients have tried the therapy and a double-blind trial is being worked out...."

- Gail Kansky
President, National CFIDS Foundation, Inc.
posted on CFSFMExperimental

Actual Experiences

Click on the 'Hemex protocol' at the left for accounts.

There is a news list for people following the Hemex protocol. If you are interested in their experiences and concerns: then go to http://www.egroups.com/group/atsg/info.html

Additional Information

See Blood Thinning Foods and

".. At least 6% of Western populations have an hypercoagulability state, "
http://www.maxillofacialcenter.com/NICOcoag.html

and Hemex Labs

Supplements

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National CFIDS Foundation

International CFS Information

Updated on: 12/30/1999

The following is a list of supplements that have generally been reported as helpful for CFS. The dosage range are based on reports from CFS patients -- ***please discuss with your physician always***. For a list of links dealing with these supplements see: http://www.immunesupport.com/news/. Other list of supplements is available at:

- http://www.seaquake.com/cfs-fm-recovery/meds1.html

Key

How each supplement appear to help is identified by the following keys:

- RBC - Red Blood Cells: provides nutrients needed for the formation of red blood cells (thus increasing the number of cells moving oxygen)
- BT - Blood thinning: contains at least one substance known to either thin blood OR widen blood vessels. For all blood thinning items -- please keep your MD informed.
- ATN - antioxidant - help removes toxins (from mycoplasma or other sources [chemical?]) from the body. Dr. Ray Strand has reported significant improvement with CFS patients using only antioxidant treatments.
- ATNC - antioxidant co-factors (supports ATN)
- EP - Energy Production

Supplement	Suggested Dosage	How it helps	Notes / links
ASA / Aspirin	80 mg every 6 hrs	BT	Platelets only. MD approval advised.
Concord Grape Juice (**Bioflavonoids)**	16+ oz	BT, ATN, ATNC	widen blood vessels, (Vitamin C also)
Citric Acid		BT	Anticoagulation
Copper		RBC	
CoQ10	100 - 400mg	BT, EP, ATN	
Folic Acid (Folacin)		RBC, ATNC	
Ginkgo Biloba		BT	
Glutathione		ATN	
Lipoic Acid (thioctic acid)		ATN	*,*
Malic Acid		EP	
NADH	10 - 15 mg	EP	precursor to Niacin (B3)
Oil of Evening Primrose		BT	
Omega-3		BT	
Piracetam (nootropil) [obtaining]		BT	enhances cognition under conditions of ***hypoxia*** inhibits the production of thromboxane B_2
Vitamin B3 (Niacin)	500 mg	BT	Affects urination. Do NOT Use No Flush version. Reduces circulating blood lipids[*].
Vitamin B6 (Pyridoxine)		RBC, ATNC	
Vitamin B12 (Cyanocobalamin)	1000 mg -	RBC, ATNC	
Vitamin B12 Injected	1000 mg -	RBC, ATNC	Recommended (but expensive)

Vitamin C (ascorbic acid)		ATN	
Vitamin E	1000-4000 iu	RBC, ATN	

Deficiencies

The following are reported as being deficient in many CFS patients. Supplements may be needed (a blood test is *recommended*)

Supplement	How it helps	Notes / links
Selenium	Helps with blood pH	
Chromium		
Magnesium		

Stress

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National CFIDS Foundation

International CFS Information

Updated on: 12/18/1999

Stress reduction is ***essential*** for treating this illness.

Blood Flow and Stress

Immune System and Stress

- Stress-Induced Immunomodulation, Implications for Infectious Diseases? JAMA, **Vol. 281 No. 24**

Duration

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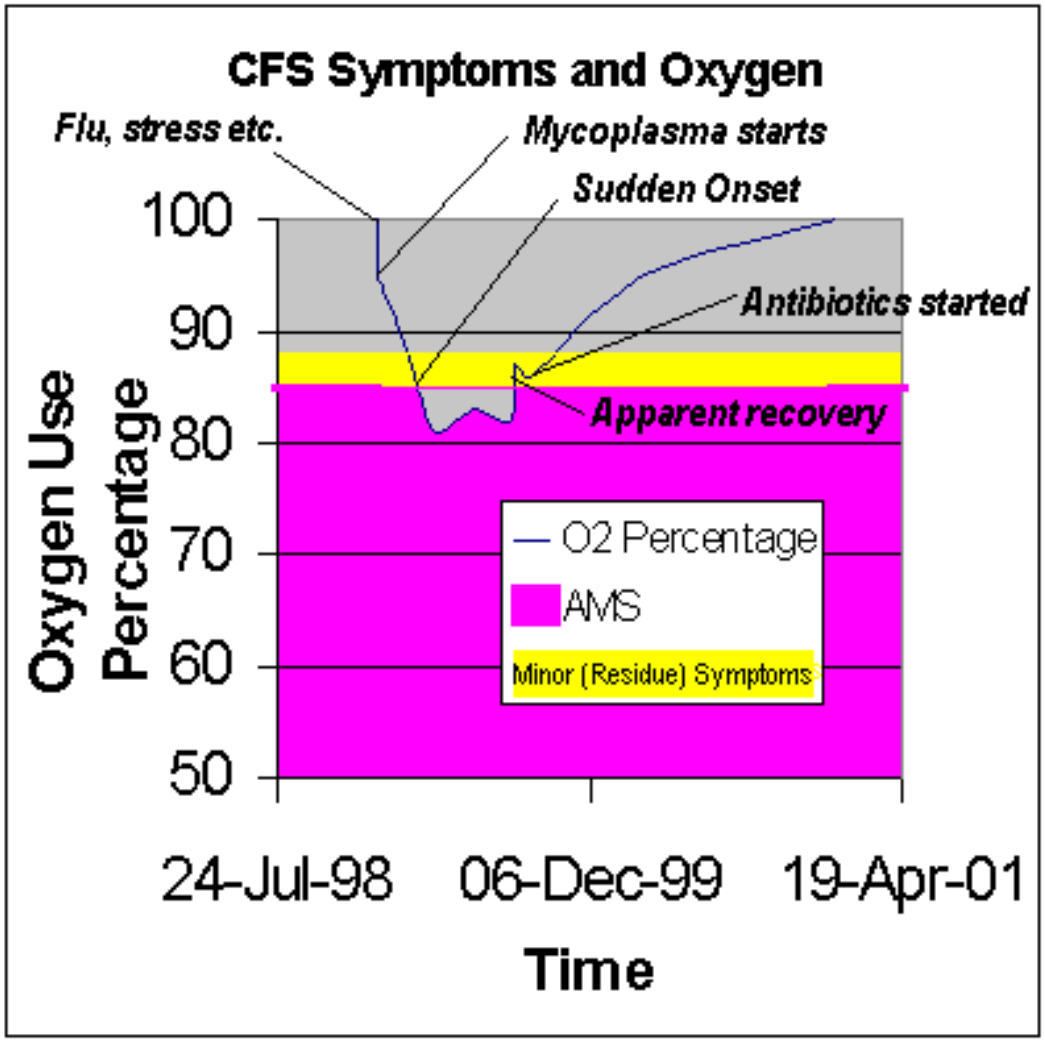
National CFIDS Foundation

International CFS Information

Updated on:
12/23/1999

The greatest problem with CFS is determining if someone is "cured" or if they have just had symptom relief. The chart below illustrates the simplest case of CFS (using the Acute Mountain Sickness Symptom model).

Illustrated Example



Some "insult" (flu, stess) caused the oxygen percentage to drop enough to get Mycoplasma (or similar) established. The mycoplasma continues to grow until the oxygen use reaches 85% of normal and the symptoms of acute mountain sickness (AMS) appears (Sudden Onset). This patient was blessed with sudden remission (for example, due to dietary changes, blood thinning, IMUPLUS) because they raised the oxygen use by 4% (just enough to get over 85%!). In this case, the patient started long-term antibiotics and eventually returned to 100% (or normal). At 100% -- the high oxygen content prevents mycoplasma from regrowing.

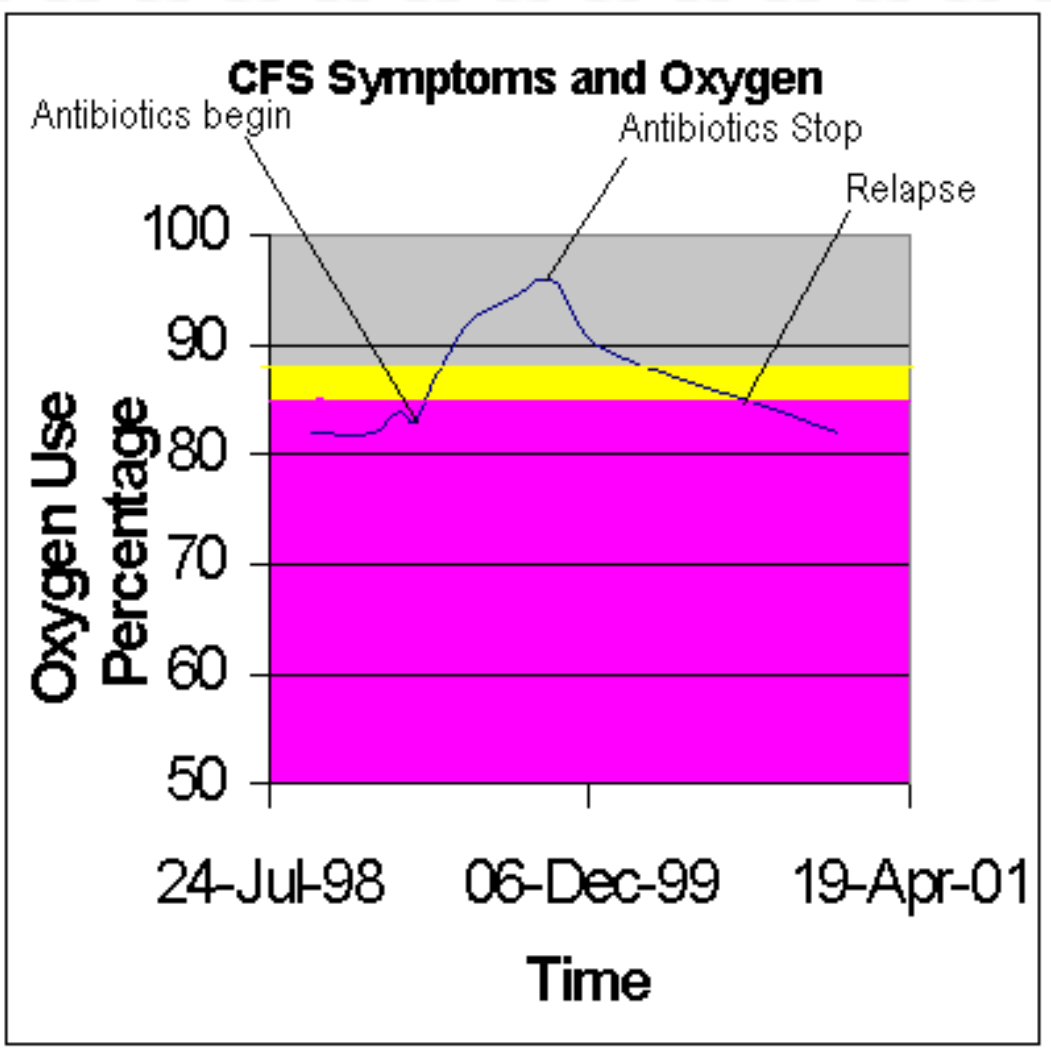
Criteria for Declaring "Cure"

I propose that ALL of the following criteria must be passed before CFS in a specific patient may be deemed 'cure' (versus symptom relief).

- Via Bicycle Ergometry with Gas Analysis, the a maximum heart rate of patient's age-dependent maximum *percentage* must be less than or equal to patient maximum oxygen consumption *percentage* (indicating normal oxygen use) (Info)
- Venus blood pH values under 7.4 and urine pH values over 6.0 (info)
- SED rate > 6 (Info)
- Normal cytokine levels (indicating ability to control viral infections)

Short Term Antibiotic Example

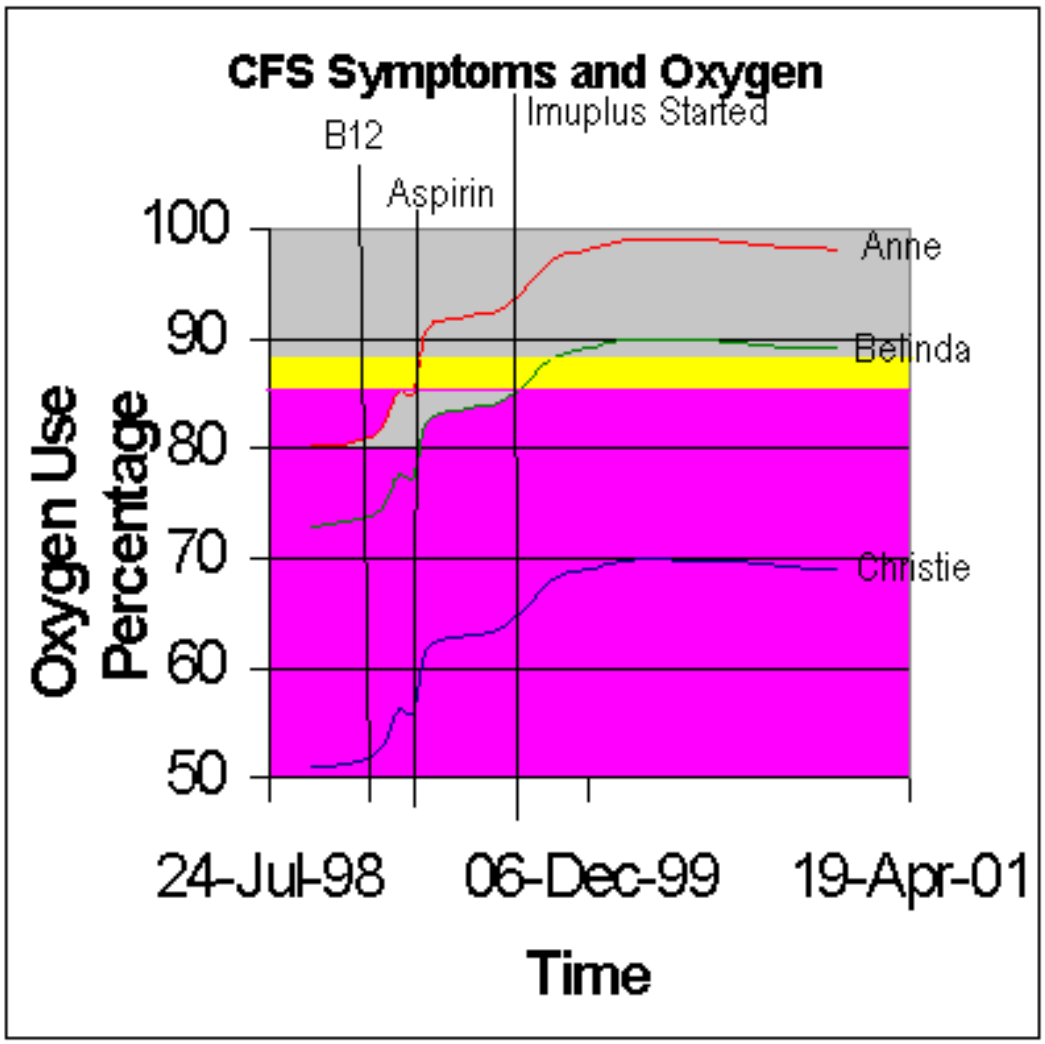
The example below illustrates the problem of stopping treatment when symptom relief occurs. Prof. Nicolson see this type of pattern with those who stop antibiotics 'prematurely'



Why X cures some and not others...

The chart below illustrates why some supplements and other

changes may improve some only and appear to "cure" others...



Anne, Belinda and Christie are three CFS patients that all started a series of supplements together. Anne after B12, felt almost normal, the rest had minor improvements. When they all started aspirin, Anne went into sudden remission and was jumping for joy until she saw Belinda and Christie crying. When they started IMUPLUS, Belinda started to feel better and better and appear to slowly go into remission while Christie felt only a little better.

The degree of improvement from any supplement depends on what your starting point was.

Ampligen

Dr. Cecile Jadin's Papers are now available in full Click Here

Ampligen Worked
Ampligen Dangers

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National CFIDS Foundation

International CFS Information

Updated on:
01/13/2000

Ampligen is a very controversial drug in the CFS community. The current thinking is that it should only be given to those who have been tested *positive* for light weight (37kDa) RNase-L.

This test was not available for the first trials -- with some unfortunate consequences. I choose *not* to get involved in arguments that have arisen (although I am the moderator of a Ampligen group: http://www.onelist.com/community/CFS-Ampligen).

I will post first hand accounts of any person who has been on Ampligen as an informational service at this site (see left column) - or provide links to them below. The decision on this drug must be reached by the people whose life will be affected by it.

http://www.cfids-me.org/marys/ampdiaries.html
(confirmed to still be very positive on the drug in January 2000).

Ampligen Worked

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National CFIDS Foundation

International CFS Information

Updated on: 01/08/2000

Posted with permission

"Someone inquired last week about my current health status. Before I proceed, however, I must emphasize that I am speaking first hand, and only for myself. I am extremely concerned about the filtering that naturally occurs when a person's experience with this or any other therapy is related by someone else. What happens when one person speaks for another, no matter how well-intentioned that person is, is that the other person's experiences are no longer clearly their own, but are subtly changed by the speaker's personal experiences and perceptions. Several people on this list have generously offered to post a message from me updating my health status for those who wish to know. It would have been easier for me to let them do it, and I appreciate their offers, but we need first hand information, not someone else's opinion. There is so much rumor and innuendo surrounding Ampligen, I certainly don't want to add any more!

I started taking Ampligen in December of 1997. With a few missed doses here and there, I am still taking the drug twice a week. I get 200 mg in around 40 minutes. I am currently working 20 hours a week at our local ski resort, part in Guest Relations, part as a ticket cashier, and part as a ski instructor with the adaptive (disabled) ski program. In the months before starting on Ampligen I was not capable of planning and preparing a meal more complicated than microwaving a box out of the freezer, and even then I didn't always grab the right box. I accurately balance my own cash drawer (thousands of dollars daily) these days, and also do the verification count for other cashiers at the end of the day. I'd kept the books for our family farming operation prior to getting sick with CFS in 1989 but I lost my ability to do math calculations and accurately handle money early in the disease. This cognitive skill has improved tremendously over the past 2 years.

I credit both Ampligen AND the energy conservation techniques I've learned from exercise physiologist Staci Stevens with my improved quality of life. For me, this has been a winning combination. If I were measured directly against a healthy person, I would still be considered at least partly disabled, but measured against where I was a few years ago, my improvement is remarkable.

My health improvements have not been steady over the past 2 years. Unfortunately, the fluctuations I've experienced have had more to do with the backlash from political infighting than from the drug itself. Last May, as most of you know, a group of CFS activists met in Philadelphia to discuss forming a community advisory board for Ampligen. I was elected by those in attendance to chair the steering committee whose task it was to organize a meeting to explore the potential for forming a CAB. Within weeks, this ad hoc committee fell victim to the kind of political infighting and guilt by innuendo that seem to characterize many efforts by our patient community to get things done.

Territorialism, possessiveness, and accusations of political maneuverings simmered for a short while then came to the surface in an ugly boil. I was accused of not caring about patient safety, of being a puppet manipulated by the drug company, and of being driven by my own selfish financial needs. When I pressed my accusers for specifics that would substantiate their accusations, I was ultimately told it was the PERCEPTION that I had a secondary agenda that had stirred up their anger.

Perception, innuendo, rumor, conjecture -- these are the elements that legends and fairy tales are made of. No other patient community attacks its own and cannibalizes its own in the way the CFS patient community does. I fear that we will never succeed in gaining true legitimacy for our disease, or truly substantial funding for research to develop effective treatments (even the cure!) until we are able to stop attacking and destroying one another, and come together to destroy the ignorance and the dismissive attitude that our political and social culture holds against us.

Ampligen patients must have a board that is specific to THEIR needs for safety, information and the provision of the highest quality of study design and investigator implementation. Their safety is first and foremost. When patients encounter problems in trials with Ampligen, as in trials with any other experimental drug, they must have somewhere they can turn for information and recourse. The board that is created, whatever it is named, must focus entirely on the needs of the patients who are enrolled in the clinical trials. Other CFS political activist agendas must find other forums for their expression.

Karen"
Aglady1@aol.com Saturday, January 08, 2000

Ampligen Dangers

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National CFIDS Foundation

International CFS Information

Updated on: 01/13/2000

Dear_____,
The incorrect folklore around my experience with Ampligen is frustrating. I have tried to keep a low profile but I will explain to you why I am now speaking out. I am trying to clear things up--and hopefully still keep that low profile. I have been told numerous times about the exaggerations by Hemispherx and some of the P.I.'s (principal investigators) regarding my "successful recovery" and I chose to ignore them and spend my time getting well--not getting even. However, if someone calls me and asks me a direct question concerning the drug-- they will get an honest and direct answer. That is my style. I am really concern about the false impression they are giving about my five years on Ampligen. It incorrectly influences people who are considering hocking their life's savings and their health by what they are told about me and what Ampligen is capable of accomplishing. After 5 years on the drug (400 mgs 3xs a week) and a lot of side effects--plus not getting well--no one should know better than I do--what this drug can and can't do.

The last straw was when a friend of mine sent me a copy of the U.S. patent (number 5,958,718) which was filed by Hemispherx and granted by the U.S. Patent Office. In their application Hemispherx claimed patient 00 (me) was a professional golfer and was now "leading a normal life fully self-sufficient" Seeing as I had an 18 handicap back many years ago when I was able to play golf (not bad for a sweet little old lady) I would hardly qualify for professional golfer status. Professional golfer's handicaps are 0--thus no handicap is used when they play in tournaments. Not to mention
they play for money. As far as "leading a normal life and am self-sufficient"--I should be so lucky. Ask my husband who cares for me (even though he has Parkinson's Disease) how correct that statement is. Those are bold face lies and it bothers me. We are seriously considering going to our lawyer (who is a very good one, I may add) and ask him to write a letter to the FDA and the US Patent office telling them the truth. I doubt if we would sue at this point--just inform both agencies...but if I hear any more of that

sort of thing-- they had best watch out !! Lying to the government is not one of my strong points. U.S. Senator Pete Domenici (R-N.M.) was very instrumental originally--in 1988--helpimg me to obtain Ampligen on a compassionate care basis. He is a very powerful senator and one of those rare politicians who is extremely honest and keeps a very personal relationship with his constituents. In fact, I may go to him with this issue. I respect his honesty and excellent reputation so he deserves the truth and so does the FDA and Patent office.

Now--let's see if I can answer your questions. Does Hemispherex, FDA, Dr Peterson, or Dr Levine, etc ---have my correct address---- yes, absolutely--and the same for most of the others. In fact Dr Peterson wrote to me several years ago and offered me the opportunity to go back on the drug. My answer was a resounding and emphatic-----NO !! To be honest with you --if I had it to do over again I would not go on it the first time around knowing what I do today. I was astounded when I reported the heart problems to the doctor who was my principal investigator-- and his answer was "Ampligen couldn't have caused that" and that was it. How would he know and shouldn't it have been reported anyway ? At this point NO ONE in the original study with whom I am acquainted -- or those in the following double blind placebo study -- is well. (The double blind placebo study is jokingly called the" double cross " placebo study as Hem did not keep their word.) I was on the drug the longest. NOT ONE, including myself, of the participants I know (and I exchange info on a regular basis with MANY OF THE ORIGINAL RECIPIENTS) has been contacted in ANY way by Hem or the FDA, etc. Scarey isn't it. I know of one of the first 10 Ampligen patients has Mantle Cell Lymphoma and a second person who was one of the original 15 has stage 4 cancer. Another of the first 10 has found a lump in her breast and is seeing an oncologist next week. Two of the first 10 have committed suicide. I had a hemangioma tumor removed from my liver in November plus all the heart complications. Nothing has been documented. Seeing as many of us were on the drug 4 or 5 years --I find this disinterest amazingly careless or maybe they don't want to know or admit to developing problems. Who knows--but is this how all drugs are approved??? I hope not.

You would think that would be of interest to the FDA seeing as we were all on Ampligen for years. I seems logical we would be of great interest to those people responsible for both the safety and efficacy of this drug such as Dr Levine. As far as the arrhythmias and mitral valve prolapse---no, I
did not have either before I took Ampligen. They were diagnosed by the Johns Hopkins cardiologists, after i was off Ampligen, who

found I also had NMH. I was treated by a cardiologist (electro physiologist) in Albuquerque for all those complications. We have the arrhythmias calmed down now but it took several years. You asked about intolerance for alcohol--yes -- I have always been intolerant. My husband kiddingly use to called me a "cheap date" because I never ordered an alcoholic drink while we were dating. With all my medical bills now, I don't fit in the category of a cheap date anymore.

You are welcome to share this information with anyone. It is the simple truth and I think that truth is needed in this vast web of inappropriate information regarding Ampligen.

Nancy Kaiser

(Reprinted from CFSFMExperimental at www.onelist.com)

Hypoxia

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National CFIDS Foundation

International CFS Information

Updated on: 12/21/1999

CFS and Hypoxia

The initial symptoms of CFS are identical to Hypoxia, and the recent findings of severe loss of oxygen capacity in CFS patients leads to the conclusion that they are hypoxia (at sea level) symptoms caused by a *change of pH in the blood*, *coagulation (slowing down the blood flow)* and *destruction of red blood cells*.

The term hypoxia includes the following:

- Acute Mountain Sickness (AMS),
- High Altitude Pulmonary Edema (HAPE) and
- Cerebral Edema (HACE).

There are two processes occurring with the above:

- ***decrease of oxygen*** to the body
- loss of air pressure to the lungs.

The latter does not occur with CFIDS, the former does (because of thick blood)- thus all lung/breathing symptoms are omitted from the discussion below. Most of the text below are direct quotes. There are no laboratory tests to identify AMS [2]

Altitude Sickness Symptoms

The typical symptoms [1, 2] are (% for 8000'):

- mild headache (70%)

- a severe headache which fails to respond to any normal treatment (7%)
- sleep disturbance, insomnia (30%)
- weakness, fatigue (30%)
- increased thirst
- nausea (5%)
- loss of appetite, anorexia (5%)
- dizziness (20%),
- apathy
- flu like symptoms in the absence of a fever or only a very low grade fever
- noticeable symptoms of slurred speech,
- loss of decision making skills,
- loss of coordination and vision

Clinical Determination

At altitudes over 2400m / 8000 ft, the diagnosis of **AMS** is based on a headache plus at least **one** of the following symptoms[3]:

- GI upset (loss of appetite, nausea, vomiting)
- fatigue/weakness
- dizziness/light-headedness
- insomnia (more than just the usual frequent waking)

HACE will have symptoms of AMS plus either gait ataxia or mental status changes, or will have both gait ataxia and mental status changes regardless of AMS symptoms. ... use a simple tandem-gait test: asking the patient to walk heel-toe along a straight line..., they should be able to perform this test without difficulty. If they struggle to stay on the line, fall off it, or are unable to walk without assistance, they fail and are presumed to have HACE. [4]

Side effects are reduced by drugs that increase the rate of respiration (thus not likely to be effective for CFS):

- Diamox
- Dexamethasone

The direct use of oxygen may be helpful.

At 9000 feet O_2 saturation measurements between 85-95% of normal are common (minor drop only). The illness usually begins

within 4 hours of arrival in 60 percent of those affected. On occasion the onset of symptoms may be delayed for two to three days.

Dietary Recommendations

The following is recommended for AMS (and thus, may apply to CFS)[*]

- no alcohol,
- no tobacco,
- no coffee (because it dehydrates)
- no soda (pop)
- lots of liquids..
- a general carbohydrate diet of at least 60% of the total calories
- reduce proteins and fat, these put more stress on the body.

Physical Changes

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National CFIDS Foundation

International CFS Information

Updated on: 02/10/2000

Physical Characteristics

The following physical changes are very frequently found in CFS patients (> 50%). Surgeons operating on CFS patients needs to be aware of these characteristics:

- neurally mediated hypotension (NMH)
- low red blood cell count
- low blood plasma volume
- hyper coagulation (thick blood)
  - A LOW SED (0-5) is often seen
- alkalotic (urine pH < 6, venus blood ph > 7.4)
- leaky gut (creating drug, food sensitivities, chemical sensitivities)
- low rate of oxygen release from red blood cells (up to 50% below normal)
- 80+% chance of severe herxheimer effect from some antibiotics

Also, many of the supplements that CFS patients take are also *blood thinners*.

***Note**: There is significant evidence suggesting that CFS may be transferred by blood transfusion - however, there is evidence that CFS uses coagulation as a significant part of its infection mechanism. If there is concerned about infection of medical staff, a "baby" aspirin (82g) a day for a month should prevent the infection from becoming established*

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

Symptoms

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International CFS Information

Updated on: 12/23/1999

The following tables lists common CFS symptoms with the *first probable* cause according to the mycroplasma-> coagulation -> hypoxia -> systemic starvation -> viral reactivation model that I am favoring.

Symptom	Mycroplasma	Coagulation	Hypoxia	Sys. Starv.	Viral React.
Natural Killer Cell decrease				missing nutrients	
Crimson crescents	chronic red discoloration of the anterior pharyngeal pillars				
Brittle, thinning hair with reddish tint				missing nutrients	
Headache			AMS		
Sleep Disturbance			AMS		
Weakness, fatigue			AMS		
Loss of appetite			AMS		

Flu like symptoms			AMS		
Brain fog			AMS		
Low grade fever			AMS		
low RNase-L				missing nutrients	
Low SED (0-5)		Blood cells do not deposit			
Poor Absorption of vitamins	?	?			
Delayed crash after mild exertion	lowered O2 allows new growth spurt of M.				
Weight loss			AMS		
Weight gain				fat used to store toxins	
Sallow (or gray) skin tone					
Low red blood cell count		blood cells sliced up			

Sleep

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National CFIDS Foundation

International CFS Information

Updated on: 01/01/2000

Sleep disturbances are associated with both CFS and Hypoxia. The apparent mechanism appears to be an ***avoidance of the blood pH becoming alkaline***. Alkaline blood has a reduced capacity to *release* oxygen. If the body is short of oxygen, it will attempt to prevent the deeper loss produced by sleep.

The first urination of the day has the lowest (most acid) pH because the blood has become alkaline during the night. The last urination is a significantly higher pH, and may be alkaline in a normal person.

Treatment

Traditional CFS patients have been treated with a variety of sleep medication. Often several must be tried before an effective one is found.

An alternative that has worked *for some* people is make the blood more acid just before bed time:

- An electrolyte low PH water. for example: Green Label Electrolytes (available from www.lifebalances.com).
- Wearing a "Painter's mask" for a few hours (more CO2 - carbonic acid - in the blood)
- Hale's Breathing before bed time
- Take Imuplus or Immunocal as a bed time drink

See Diet pH for more suggestions on food to modify blood pH.

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Outbreaks

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Blood Transfusions
Osler's Web Survey

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National CFIDS Foundation

International CFS Information

Updated on:
01/08/2000

IDEF / CFIDS is an infectious disease that may become contagious in some circumstances. There has been a history of recorded outbreaks for the last 70 years.

Not Very Contagious, usually

Mycoplasma appears to match the infection vector well, with *walking pneumonia* (***mycoplasma pneumonia***) being the best analogy. It appears to take prolonged and repeated contact to infect in most cases (like that in a family). Infection likely happen when the uninfected person is suffering 'insults' to their defense mechanisms (have another illness (particularly flu), severe stress). CFSers should practice good 'cold hygiene'

- wash hands often
- keep a little further away from people
- close care givers should take a baby aspirin each day

All of the outbreaks are suggestive of infection by contact or breath.

Contagious Varieties - Outbreaks

Current mycoplasma testing for CFS targets six different mycoplasma. There may be many more, and some of them appear to be highly contagious *for a short period* of time (-5 - 30 days after sudden onset).

"Dr Dowsett outlined the prevalence and geographical pattern of ME. noting
that it affects cool and affluent areas, often follows epidemics of poliomyelitis, and is seasonal. She stated that there has been an enormous
pandemic from 1980s onwards, and emphasized that we are likely to see
pandemics every 20 years and epidemics every 10 years. She made clear that enteroviral mediated illnesses in general follow this pattern. "

> - from: UK All Party Parliamentary Group Meeting Minutes 23.11.99
> http://members.aol.com/MEwebsite/APPG1.html

A plausible spread pattern from a "patient Zero" for many of the 1980's outbreaks is documented in the "Osler's Web" link on the left.

The infection vector is unknown but would appear to similar to that of the flu virus. The Attack Rate for outbreaks range form *19%* to *55%* of the population [Henderson/Shelokov [CDC], Epidemic Neuromyasthenia-Clinical Syndrome?, NEJM, 1959]

A recent study found that "5% of this group the syndrome was precipitated by a blood transfusion a few weeks prior to a flu-like syndrome that later proved to be the acute onset of their CFS"

Model: The illness develop as a minor mutation of an existing active virus (borrowing DNA from bacteria), if this mutation happens before the general population is exposed to the original active virus then an outbreak may occur. If the virus mutates after the general population is exposed, then the antibodies developed for the original virus is also effective for this minor mutation, an outbreak is unlikely.

"The first reported US outbreak was in Los Angeles in 1934 (Gilliam, 1938), and in the UK in London (Acheson, 1952) and Coventry (1953), though some 60 epidemics, often in residential hostels and mainly affecting younger people, have since occurred in parts of the world as diverse as Iceland and Melbourne (Behan & Behan, 1980). Three major London outbreaks (Royal Free Hospital, 1955; Middlesex Hospital, 1952; and Gt. Ormond St. Hospital, 1970) .... These outbreaks were reported by Ramsay, 1989, Acheson 1954,

and Dillon, Marshall et al., 1974 respectively.

In Ayrshire an outbreak in West Kilbride was described (Fegan, Behan et al., 1983) affecting 22 patients between January 1980 and June 1983, mainly female. The most characteristic symptom was extreme exhaustion, particularly after exercise. No clear aetiology or diagnostic marker for ME has yet been isolated. Though according to Ramsay (1989) the disorder is often associated with Coxsackie B viral infection (which was found in 18 of the 22 West Kilbride patients), it is not clear whether the viral insult is post-opportunistic, since no clear infectious pathway has been isolated. "

from http://www.cogreslab.demon.co.uk/MEstudy.htm

Other outbreaks:

- 1975 Health care workers at the Mercy San Juan Hospital in Carmichael, CA (Dr. Erich Ryll), for his comments see:
  - First hand account of Mercy San Juan Hospital, 1975 Outbreak
  - http://www.med-help.com/Page11.html

1984 onwards: See Osler's Web on left

Incidence

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International CFS Information

Updated on: 10/30/1999

This illness is more common among woman than with men. I suspect that this is caused by the stress of the female reproductive system, including birthing. The CDC reports

Population	Per 100,000
Caucasian women	340
All Men	53
Men and Women	183

Latest estimate is 422 per 100000 people [AMA Oct 11,1999]

The incident rates for other diseases are:

- 125/100,000 for HIV in women
- 12/100,000 for AIDS in women
- 63/100,000 for Lung Cancer in women

The highest incidents of IDEF/CFIDS is reported to be in the following professions:

1. Medical Professionals
2. Teachers

Also of interest, "CFS patients (60) and polio survivors (2,25,64) have been shown to be within the high normal or superior range on measures of higher-level cognitive processes and I.Q. and have ***higher than average levels of educational and professional achievement***."

Both of these professions have the nurturing environment for

IDEF/CFIDS:

- high rate of exposure to viruses
- high amount of stress

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Testing Labs

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National CFIDS Foundation

International CFS Information

Updated on: 02/16/2000

HHV6:

- http://www.hhv6.com Wisconsin Viral Research Group, no Medicare

Natural Killer (NK) Function

- Lane Labs - 1-800-LANE-005

RNase-L Testing

- US Labs Experienced/Recommended in drawing samples
  - Bioreference Labs in Elmwood Park, New Jersey. 201-791-3600

Antigen Leukocyte Cellular Antibody Test (ALCAT)

- American Medical Testing Laboratories, Hollywood, FL, 954-923-2990.

Organic Acid Test (used by Cheney)

- Great Plains Laboratory in Overland Park, KS, 1-913-341-8949.

Mycoplasma Testing

- Medical Diagnostic Laboratory
  133 Gaither Drive, STE C
  Mt. Laurel, NJ 08054
  609-608-1696
  - As of 9-98 their mycoplasma general test is $120. If it is positive they do 8 subspecies for an additional

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Anesthesia

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National CFIDS Foundation

International CFS Information

Updated on: 02/10/2000

Information Regarding Anesthesia

National CFIDS Foundation, Inc.
103 Aletha Rd., Needham, MA 02192-3931

- http://www.cfs.inform.dk/Behandling/anesthesia.html
- http://wwcoco.com/cfids/anesthesia.html

"I would recommend that potentially hepatoxic anesthetic gases not be used including Halothane. Patients with Chronic Fatigue Syndrome are known to have reactivated herpes group viruses which can produce mild and usually subclinical hepatitis. Hepatotoxic anesthetic gases may then provoke fulminate hepatitis. Finally, patients with this syndrome are known to have intracellular magnesium and potassium depletion by electron beam x-ray spectroscopy techniques. For this reason I would recommend the patient be given Micro-K using 10mEq tablets, 1 table BID and magnesium sulfate 50% solution, 2cc IM 24 hours to surgery. The intracellular magnesium and potassium depletion can result in untoward cardiac arrhythmias during anesthesia. For local anesthesias, I would recommend using Lidocaine sparingly and without epinephrine."

- Paul R. Cheney, MD, PhD, 1992

"Suggestions on anesthesia include using Diprivan (propofol) as the induction agent along with nitrous oxide and isoflurane (Forane) as the maintenance agent. The ones to avoid are

histamine releasers that include sodium pentothol as well as a broad group of muscle relaxants in the Curare family, including Tracrium and Mevacurium."

- Patrick. L. Class, MD, 1996

CFS ANESTHESIA PROTOCOL

From http://www.cfs.inform.dk/Behandling/anestesi.txt

I have used the following anesthesia protocol with success during surgery on CFS patients. First, I perform skin tests for all the agents I am considering using with the patient. With CFS patients, I recommend Diprivan (propofol) as the induction agent; Versed (midazolam), fentanyl (a short-acting narcotic) and droperidol (an anti-nausea agent) during the anesthetic; and a combination of nitrous oxide, oxygen and iso-flurane (commonly called Forane) as the maintenance agent.

In contrast to the above agents, there is a group of commonly used anesthetic agents which are known histamine-releasers and are probably best to be avoided by CFS patients. This group includes the thiobarbiturates such as sodium pentothal, which is probably the most common induction agent, but is a known histamine-releaser. In addition, there are a broad group of muscle relaxants in the Curare family, name Curare, Tracrium and Mevacurium, which are also potent histamine releasers and should be avoided by CFS patients. Since so many of these histamine-releasing agents are commonly used during emergency surgery, it would be advisable for you to wear a medical alert bracelet in the event you are unconscious and would have to have an anesthetic. I would mention on the bracelet that you cannot receive any histamine-releasing drugs.

Blood Transfusions

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National CFIDS Foundation

International CFS Information

Updated on: 10/05/1999

The Center for Disease Control and Prevention (CDC) has cautioned CFS patients not to give blood... however:

- the FDA does not specifically prohibit blood donation by CFIDS patients [1]
- a British study had 5 % of new CFIDS patients had blood transfusion immediately prior to a 'flu' that preceded the sudden onset of CFIDS, it is hard not to conclude that CFIDS may be transmitted by blood.

Osler's Web

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National CFIDS Foundation

International CFS Information

Updated on: 12/30/1999

The following information is extracted from "Osler's Web" by Hillary Johnson. The conclusions /presentation are the website author (not Johnson's).

It illustrates that a major outbreak (pandemic?) of CFS in the US may have started with one person.

p. 88-9: in summer 1982, 63 yr old South African ("patient zero") (of Danish descent) visited relatives in the US on a 6 week visit (California, Georgia, Washington State)... just before the trip she had "mono-like symptoms"...not in good health during the visit...

- All 4 relatives developed non-Hodgkin's lymphoma(very rare) within 200 days of the visit... One of them had moved to Truckee in mid-1984, and visited Incline Village often.....
  - 1983-4 San Francisco, (Carol Jessop, MD): cascade of patients developed CFS
    - 1986 Los Angeles
  - 1984-5 Incline Village (Cheney & Pedersen, MDs): major outbreak

    p. 207 .."A suspiciously high number of patients develop a rare cancer of the immune system called non-Hodgkin's lymphoma"

    (in reference to Tahoe epidemeic)
    - 1985 Truckee, California (1 hr drive from Incline Village)
    - 1985 Yerington, Nevada (> 105 people)

- 1983 Sweden (? did "patient zero" visit Denmark and Sweden earlier?)
  - 1984 Key West, Florida: 37 "MS" cases
  - Same antibodies as in Sweden, was it carried by tourists from Sweden?

- 1985 Raleigh, NC: North Carolina Symphony
- 1985 Lyndonville, NY

Survey

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Updated on: 01/03/2000

An Internet-based survey of CFSers with onset prior to 1984 (the Incline Village pandemic) is currently in progress. If you had CFS prior to 1984, please email CFS@Folkarts.com with the month/year of onset, where you were living at onset (and the 6-8 months prior), put "survey" on the subject line. Thanks.

Other CFS Surveys: CAMEO (http://www.tertius.net.au/foothold/cameo/cameo.html)

raw data: (each bullet is one report, [prior 8 months])

California

- 1971 Long Beach
- 1980 January, Hayward

Colorado

- 1973 Denver

Maine

- 1951 Bangor

Michigan

- 1955 Detroit

Missouri

- 1968 November, Fort Leonard Wood [Washington NJ, and Fort Dix NJ]
- 1980 September, Kansas City

New York

- 1978 October, Buffalo

Oregon

- 1979 July, Portland

Utah

- 1981 Snowbird and Salt Lake City
- 1981 November, Provo [New London, CT]

Statistical Notes

Using the internet to gather this information should allow a relatively unbiased sampling of information.

Planned tests are:

- Incidence by Year [*this will likely be biased*]
- Incidence by Altitude of City (0 - 2000 ft, 2000-4000 ft, 4000+ ft)
- Incidence by Coast / Non Coastal
- Incidence by Western, Central and Eastern US
- Incidence by Northern, Middle and Southern US
- Incidence by USDA Climate Zones
- Incidence by Season
- Incidence by Month

Urine pH

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Updated on: 01/27/2000

Dr. Cheney reports that CFSer Urine pH is acid typically 6.0 or lower (with the blood alkaline). Urine pH is simple to self-test and the testing may be done with 'litmus strips' (for example, those used for measuring pH in hot tubs) or with electronic meters (for example, those used for measuring garden soil pH).

- Only in severe diseases does the pH get slightly below 7.0 or close to 8.0. [Adams]
- A range of 6.50 - 6.80 for the first urination of the day is viewed as "optimum" - Biological Terrain test[*] (used by Cheney)
- Ideal pH by the time of day:[*]
  - in the morning, (pH = 6.5 - 7.0)
  - in the evening (pH = 7.5 - 8.0)

7.0 is Neutral pH. below 7.0 is an acid, above 7.0 is a base (alkaline)

Blood pH

See breathing.htm

Literature on Urine pH

- http://www.rnceus.com/ua/uaph.html
- Adam's http://www.adam.com/ency/article/003583.htm
- What is pH? http://www.britannica.com/bcom/eb/article/7/0,5716,61047+1,00.html

Equipment

A cheap garden pH meter gives more accurate readings than litmus strips (and over a long run, are cheaper!). They should be viewed as RELATIVE pH meters. The following are a few sources:

- $19.99 http://www.hirts.com/p3211.htm, #8203
- $17.50 http://www.frostproof.com/catalog/t511.html
- $19.75 http://charleysgreenhouse.com/inst4_PH.htm

Litmus papers must be checked to verify that they will display a 6.0 or lower

pH (many do not). Example of Litmus paper cost: http://www.dharmatrading.com/php.html

Semi-Professional ph Meters

The following 2 meters give accurate / true pH results (and are reasonably priced):

- pH meter PC interface (0 to 14 / 0.1 pH resolution) $67 (Does temperature compensation). Connects to PC (includes software)
- Low cost pH stick (pH Range: 0 to 14) $49 (Does NOT do temperature compensation)

Process

Create a log book of pH measurements, and measure at least three times per day

- Waking (pH is usually highly acidic)
- After a meal
- Before a meal

(Mis-)Diagnosis

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Updated on: 10/05/1999

Prior to AIDS research revealing the mechanics of IDEF/CFIDS, the difficulty of understanding the infrastructure nature of the disease resulted in various symptoms being deemed to be the cause!

The sensitivity of the illness to stress in both onset and during recovery, suggested to many that it was a 'slackers' illness or a mental condition. Depression was a common symptoms, and treatment for depression often resulted in some improvement of the condition. Any symptoms that produces stress (mental or physical) needs to be treated promptly - but not as the *cure*, but to remove a catalyst that assists the illness. [1]

Some suffers from IDEF/CFIDS include:

- Welterweight champion Grahame Cheney of Australia, who announced on March 26, 1996 that he is retiring due to CFIDS

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National CFIDS Foundation

International CFS Information

Updated on: 12/08/1999

A DIAGNOSTIC MARKER FOR CFS?

THE POKER GAME GOING ON IN OUR GENES

This article, written by Carol Sieverling, is based on a conversation with Dr. Cheney taped in October 1999. Dr. Cheney has reviewed this article and it is shared with his permission. It will appear in the Winter 2000 issue of the *DFW Lighthouse*, (which can be found at www.virtualhometown.com/dfwcfids), the newsletter of the CFS/FM Support Group of DFW. Feel free to repost and reprint.

Dr. Cheney read a newly published study this summer and glimpsed a possible diagnostic marker for CFS. Test subjects included three Gulf War Veterans, seven healthy control subjects, and two people with active polio-virus. Researchers probed their blood for both RNA and DNA. They found thousands of different sized RNA segments floating around in the serum of veterans, a small amount in the polio subjects, and none in the healthy controls. The researchers called this voyager RNA since it travels around in the blood outside of the cells.

This finding in the veterans is highly unusual on four counts. They had RNA segments in their blood, they had a lot of RNA segments in their blood, they had a lot of aberrant RNA segments in their blood, and they all the same aberrant RNA segments in their blood. (The segments from the three veterans varied by less than 1%.)

The researchers isolated the aberrant sequence and examined it in

detail. They began recognizing certain pieces, which they realized all came from part of chromosome 22. It appeared that a section of chromosome 22 had been sliced up, rearranged, pieces from somewhere else inserted, and the whole thing reconnected. Amazingly, a section of chromosome 22 appears to be altered, and it's altered the same way in all three veterans!

Since these veterans had symptoms identical to CFS, Dr. Cheney began testing CFS patients and almost all had this same aberrant RNA segment. This strongly suggests that the veterans and the CFS patients have the same illness, and that the aberrant segment of RNA is very likely a diagnostic marker. Dr. Cheney suspects that this marker only appears well into the illness, and will not be found close to onset. He also believes that the amount of aberrant RNA in the blood serum may correlate with illness severity.

Why would patients with CFS and GWS have an aberrant piece of RNA, and why would they all happen to have the same one? Dr. Cheney uses a wonderful analogy to explain it - a poker game. When we're faced with an extreme threat to our health, our body plays poker with its DNA in order to find something that will help. Our body is breaking some of our DNA up into cards and shuffling them to see if it can deal a winning hand.

There are three possibilities when dealing out poker hands. You can deal a winning hand. That's possibly what this RNA segment found in both CFS and GWS is – a winning hand. The body shuffles its way to something that it senses might help, so it remembers it and makes a lot of copies. These segments float around in the blood on their way to other cells to make more copies, and they show up on the test more easily because there are so many of them. The potential diagnostic marker is actually a winning hand, or as close as the body can come to one. And it's a marker because everyone with the same illness will eventually shuffle to the same solution. Same problem – same helpful answer.

A second possibility is a bust hand. You don't win or lose - the new segment doesn't help, but it doesn't hurt either. The third possibility is bad news. Every so often you deal a hand with the joker in it. The body shuffles and deals out a segment that is a metabolic toxin. If it is extremely poisonous it will destroy the cell in which it was created, thus destroying itself. The real problem is the minor toxins, the ones that make you sick but won't kill you. You shuffle out enough of these bad hands and it can keep you from getting well. The hope is that the new treatment Dr. Cheney is testing, fetal bovine growth factor, will be able to destroy some

or all of these aberrant segments of RNA.

More research is needed to confirm that this segment of RNA is a diagnostic marker, but Dr. Cheney believes this is by far the best candidate yet. He also notes that while it would be a genetic marker, it is not one we are born with. It is one our body creates in response to this illness.

The study mentioned is entitled "RNAs in the Sera of Persian Gulf War Veterans Have Segments Homologous to Chromosome 22q11.2", was written by Urnovitz, Tuite, Higashida, and Murphy, and was published in "Clinical and Diagnostic Laboratory Immunology" in May 1999, p. 330-335. A transcribed interview with Urnovitz can be found at www.co-cure.org, archives, Dec 1999 Week 1, "MED,RES Major Breakthrough in Chronic Illness Research"

Oxygen

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Self Carbon Monoxide Poisoning

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International CFS Information

Updated on:
02/01/2001

The following report describes one method of testing a patient for disability. Of special interest is the very low oxygen consumption reported of 43.4% (normal is 85+%) which is typical of many CFS patients and suggests low oxygen levels or transfer rates in the body.

It may be helpful for many MDs in understanding CFS and knowing what to test for... See VO2-Max for recent studies as well as genetic component.

TEST: BICYCLE ERGOMETRY WITH GAS ANALYSIS

To whom it may concern:

XXXX is a ** year old female who was evaluated for complications related to chronic fatigue syndrome on **/*/**. The following is a report of performance on a cardiopulmonary exercise stress test done at the Cheney Clinic. Our clinic performs cardiopulmonary exercise testing by using a ramp protocol designed to reach max test criteria over a ten minute exercise period using bicycle ergometry. By measuring expired gases at rest, anaerobic threshold and peak exercise, this test procedure can provide objective evidence of metabolic defects which limit the functional capacity of persons suffering from CFS. (1)

On **/*/**, XXXX tolerated 10 minutes and 16 seconds of a ramped bicycle ergometry protocol. She achieved a maximum heart rate of 160 or 89.4% of her age-dependent maximum. Her baseline EKG demonstrated no significant ST changes nor ectopy before, during, or after the exercise. Her respiratory quotient (RER) - the ratio of carbon dioxide produced to oxygen consumed - reached a maximum of 1.18 indicating excellent effort.

Her maximum oxygen consumption at peak exercise was 714 ml/min, or a normalized result of 11.7 ml/kg/min (normal subjects typically achieve no less that 25 ml/kg/min). This is 43.4% of her predicted maximum for her age, sex, height and weight using the Jones' criteria (normal is greater than 85%). She achieved a maximum work rate of 64.0 watts which is 56.5% of her predicted maximum work rate. A low maximum oxygen oxygen consumption at peak exercise is likely due to a defect in aerobic energy production in CFS patients as described by a number of investigators. (2,3,4)

She reached her anaerobic threshold (AT) at 4 minutes and 55 seconds into the test protocol. Her oxygen consumption at anaerobic threshold was 314 ml/min which is 44% of her maximum predicted oxygen consumption. 73% of the work she performed was accomplished above her anaerobic threshold (normal subjects perform less than 50% of their work above AT). This high percentage suggests unusual tolerance for a deconditioned individual to anaerobic metabolism characteristic of many patients with CFS. Her ability to continue exercising 5 minutes and 11 seconds past her anaerobic threshold suggests that her defect in oxygen consumption cannot be explained solely on the basis of deconditioning.

She had a low PET CO@ during exercise, with a value of 26.0 mmHg during the thirty seconds leading up to her peak oxygen consumption (normal subjects range between 40-60 mmHg). This demonstrates evidence of

chronic, compensatory hyperventilation seen in most patients with CFS and is most likely die to a chronic intracellular metabolic acidosis (4) demonstrable by organic acid analysis (5) and low urinary pH. In addition, a high degree of tidal volume variation was observed at peak exercise with a variance of 0.17 (normal subjects range from 0.05 to 0.15).

Many values fall outside of predicted ranges. Taken together these abnormalities suggest a significant impairment of work capacity which cannot be explained on the basis of deconditioning. Also, the defect seen in central respiratory control points to significant functional problems originating within the central nervous system. These findings are comparable with the observations of other investigators and are likely to cause severe functional capacity difficulties for Ms. Sieverling.

I hope that you find this information helpful. If there are any additional questions, please feel free to call me.

Sincerely,

Paul Cheney, M.D., Ph.D.

_____

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VO2-Max

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Following were presented at the AACFS Conference in Seattle, 2001. VO2-Max and CFIDS appear to have a strong correlation, and VO2-Max appears to be abnormally low in individuals susceptible to CFIDS (the Genetic Factor). The VO2-max for the normal population is 34-38 cc/kg/min.

#26 Snell, etc "Comparison of Maximal Oxygen Consumption and Rnase-L enzyme in patients with chronic fatigue syndrome"

- RNase L ratio < 0.6 had VO2-max at 23.1+/_ 0.7 (n=27)
- RNase L ratio > 0.6 had VO2-max < 20 (n=51)

#24 VanNess etc "Assessement of the Functional Impairment by Cardiopulmonary Exercise Testing in Patients with Chronic Fatigue Syndrome"

Impairment	VO2-Max
none-Mild	23.4
Mild-Mod	18.4
Mod-Severe	13.6
Severe	8.0

Self Carbon Monoxide Poisoning

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Updated on: 02/01/2001

The deficient oxygen delivery mechanism (due to Coagulation, vascular constriction, blood pH or other causes) results in not sufficient oxygen to 'burn to CO2', instead CO is produced. CO inhibits the delivery of additional oxygen (Catch 22).

Seattle AACFS Conference, 2001. Poster #30. The role of exogenous and endrogenous carbon monoxide poisoning in the etiology of Chronic Fatigue Syndrome, Fibromyalgia and Multiple Chemical Sensistivity.

- CFS/FM/MCS has > 4 ppm of CO
- Normal had < 2 ppm of CO

"The elevated carbon monoxide levels that Dr. Mazlen and I have found in the end-tidal breath of CFS, FMS and MCS patients may explain the reduced VO2 max in CFS patients that several reported at the AACFS conference. (The abstract of our poster should be in the conference proceedings, although we weren't able to be there to present it).

Elevated CO, whether due to endogenous and/or exogenous sources, directly impairs VO2 max and may cause all the other symptoms of CFS. I recommend that all cases of CFS/FMS/MCS be screened for the level of CO in their breath, which if persistently high may be easily, inexpensively and safely treated with supplemental oxygen (2 hours per day at home). If breath CO is elevated (>=5ppm, v. 1-2normal and 3-4 borderline), I recommend ABG and VBG testing(or as some labs cutely but idiotically call it, "ABG venous") to calculate the a-v gap in PO2 and the v-a gap in PCO2. If low, these confirm reduced O2 uptake and reduced O2 metabolism, both consistent with reduced VO2."

Albert Donnay, MHS President, MCS Referral & Resources, Inc.
adonnay@mcsrr.org , www.mcsrr.org via Co-Cure

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

Glutathione
Exercise
Whey
Rebreather

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International CFS Information

Updated on: 02/22/2000

DR. CHENEY'S NEW OXYGEN TREATMENT

(This article, written by Carol Sieverling, is intentionally technical so that those who wish to try this treatment can share it with their doctor. It is based on a conversation with Dr. Cheney taped in October 1999. Dr. Cheney has reviewed this article and it is shared with his permission. It will appear in the Winter 2000 issue of the *DFW Lighthouse*, the newsletter of the CFS/FM Support Group of DFW, (which can be found at www.virtualhometown.com/dfwcfids). Feel free to repost and reprint.

Dr. Cheney recently began prescribing oxygen for patients with alkaline venus blood. Up to an hour of oxygen in the morning can provide half a day of significant improvement and numerous benefits. He has been seeing alkaline blood results in patients for years, but dismissed it as insignificant, based on what he was taught in medical school. His growing suspicion that it was actually very significant was confirmed when another speaker at an international conference on fatigue in London began a presentation by announcing "Ladies and gentlemen, I'm here to tell you that CFS patients are alkalotic."

Blood alkalosis inhibits the transport of oxygen to tissues and organs, constricts the blood vessels, and lowers overall circulating blood volume. The putative cause of the alkalosis is the glutathione deficiency that is pervasive in CFIDS. Low glutathione causes an elevation in citrate, which in turn lowers a substance (2,3 DPG) that controls the release of oxygen from the hemoglobin. Our blood could be full of oxygen, but without enough of this substance it cannot break free of the hemoglobin and get into the cells. This causes oxygen deprivation in the tissues (hypoxia), which makes the body switch over to anaerobic metabolism, and that produces tissue acidosis, which can be painful. (The acidosis here is unusual because instead of generating a lot of carbon dioxide, it generates a lot of organic acids that stay inside the cell.) The body compensates for tissue acidosis, in part, by increasing renal bicarbonate reabsorption, and hence developing blood alkalosis.

This blood alkalosis is unusual in that Cheney usually sees venus blood pH values over 7.4 and urine pH values under 6.0. When both blood alkalosis and urine acidosis are seen, it's a metabolic problem – not a psychogenic reaction to a needle stick. A blood pH above 7.4 shows impairment, and above 7.5 there is significant impairment – almost no oxygen transport at all. A urine organic acid test will also reveal this problem: elevated citrate and/or low 2-oxo-glutaric are markers.

The really terrible thing is the presence of a vicious cycle: the blood alkalosis further lowers the levels of 2,3 DPG (inhibiting the release of oxygen), causing tissue hypoxia, which causes tissue acidosis and pain, which then causes blood alkalosis, which lowers 2,3 DPG even further. And around and around we go.

The ultimate treatment for this situation is Immunocal or IMUPlus, the undenatured whey protein supplements that helps restore glutathione. But some patients cannot afford them, and they do not work on all patients. An immediate solution to the oxygen transport problem is to use a partial rebreather mask set at 35 to 40% FIO2 (Fraction of Inspired Oxygen), which requires a flow rate of about 10 liters per minute. Try to do an hour a day, broken into one, two, or three sessions. You can do more than one hour a day, but do not do more than one hour at a time. Do not breathe heavily – breathe normally. Most CFS patients have headaches, and this can help those headaches. If the prescription is written for headaches, insurance may cover it. One hour of oxygen a day can run $75 to $100 a month.

Oxygen through nasal prongs will not work. Oxygen alone in a mask will not work. It has to be a partial rebreather mask, which has a bag attached. This allows you to rebreathe your expired carbon dioxide along with the oxygen that is flowing into the mask. It is important to the function of the rebreather that the bag contract and expand with the breathing cycle. It's not working properly otherwise. Breathing increased levels of both CO2 and O2 at the same time is essential. The CO2 breaks the cycle. It corrects the alkalosis and frees the O2 in your blood to move into your cells. With proper functioning, vessels dilate and you start perfusing your brain and tissues, bringing out the toxins and bringing in the nutrients. Raising oxygen levels will also help kill off yeast and other pathogens. Lack of oxygen allows them to multiply.

The speaker at the London fatigue conference sends his patients to breathing experts like Teresa Hale, who wrote ***Breathing Free***. Most patients are walking around over breathing and thus

becoming more alkaline. Learning to under breathe properly can help increase oxygen perfusion and transport.

Two problems can be seen in some patients on a rebreather mask.

1. Rapidly correcting blood alkalosis or overcorrecting (ie. acidosis) can provoke vasodilation. If this results in significant blood volume contraction some patients will become hypotensive and feel dizzy or faint. This problem can be prevented by taking oxygen lying down and by expanding blood volume with an isotonic electrolyte drink such as Gookinaid ERG (Electrolyte Replacement with Glucose) (http://members.aol.com/Gookinaid) (1-800-283-6505). You can also address this problem by reducing the time spent on the mask rebreather.
2. Patients with a history of migraine may provoke a migraine in the moments just after going after going off the rebreather. Again, expanding blood volume and reducing the time on the rebreather can help this side effect.

Rebreather

It is important to the function of the rebreather that the bag contract and
expand with the breathing cycle. It can fully expand when you exhale, but it
must collapse when you inhale, though no more than two-thirds. It's not
working properly otherwise. If the flow rate is too high (usually above 10
Lpm) the reservoir bag will remain expanded during the entire breathing cycle
and there will be insufficient rebreathing of CO2. If the flow rate is too
low, the reservoir bag will collapse fully when breathing in. It must not
collapse more than two-thirds. If the bag will not collapse well, check for
leakage around a poor fitting face mask. The openings on the mask near the
nose can be open or fitted with the rubber disks that turn the openings into
one-way valves. An open mask has less rebreathing potential. With one-way
valves, the CO2 rebreathing potential is increased.

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

Hypochlorhydria

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Updated on: 01/13/2000

Hypochlorhydria (or atrophic gastritis) means that the stomach produces too little HCl (Stomach acid) to properly digest food. The reason of this happening is Alkaline blood (high pH) [*]. Normally stomach acidity is around a pH of 1 (water is 7.0)

Medical diagnosis of this state is difficult [ref] however the ***typical suffer need to take 'Enzymes' or 'Digestive Aids'*** which usually contain some form of "HCl" in the ingredients.

Hypochlorhydria leads to Leaky Gut [ref] which leads to Multiple Chemical Sensitivity and food allergies. Furthermore, "*Most autoimmune diseases* are associated with a lack or insufficiency of hydrochloric acid production by the stomach(11). " from http://www.selene.com/healthlink/fibromyalgia.html

Hypochlorhydria makes digestion and absorption of meat difficult (which is the usual way of correcting alkaline blood). Meat and other flesh is the primary source of B12 in the diet resulting in B12 deficiency (very common in CFSers). This produces a cycle of keeping the blood alkaline.

Whey is the easiest digested form of protein and is suggested as a method of correcting the blood pH. Niacin (vitamin B3), pantothenic acid (vitamin B5), vitamin C, PABA and pyridoxine hydrochloride (vitamin B6) is reported to help. Reducing alkaline foods is also suggested. Dosages of Whey(up to 60 grams/day for several months) is reported to help. (Start slowly -- some people reacts to it).

Effects

- Increase risk of mycoplasma infections.[*]
- Impair the body's absorption of important minerals (iron [CDC], calcium, zinc, and B-complex vitamins, including folic acid)

See also:

- http://www.drugstore.com/guide/Concern/Indigestion.asp
- http://oak.conncoll.edu/%7Erjcha/wells/articles/hypoclor.htm

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Leaky Gut

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Glutamine

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Updated on: 01/07/2000

"Leaky Gut" is the common term for intestinal hyperpermeability which frequently develop in CFS patients. It may produce food sensitivities and multiple chemical sensitivities. The cause of this hyperpermeability is believed by some to by hypochlorhydria.

> "... I'd want to test her gut permeability. Increasingly it's thought that all arthralgias or arthritises are due to leaky guts. Which run in families. Gut permeability doesn't mean you necessarily get CFIDS per se, but if you get food protein leaks, which generate super antigen formations, which can evoke joint arthritis, and, and what it really is, is a complication of food-related reactions that are related to leaky gut. Even more pressing is the Motrin [Editor: ibuprofen]. Because Motrin increases leaky gut. It can help the joint pain immediately, but it can predispose to further exacerbating the underlying cause, resulting in more pain and continued need for Motrin. It becomes a vicious cycle." - Dr. Cheney (From CFSFMExpermental transcript)

- Other resources:
  http://www.greatsmokieslab.com/news/leakygut.html

Treatment

L-Glutamine has been reported to be helpful to many. (Information http://www.integratedhealth.com/infoabstract/glutamineab.html) Whey is rich in the components to build glutamine and also helps hypochlorhydria

A more general treatment is described at:

- http://www.moreton.com.au/ana/handbook/leakygut.htm
- http://www.mdheal.org/leakygut.htm

Head Aches

The use of Oxygen tank with rebreather appears recommended by

Dr. Cheney for headaches (especially if paid for by insurance). This allows the ibuprofen cycle to be broken, general improvement for CFS and headache relief.

Aspirin should also be consider if the CFS patient is not a child (Reye's syndrome is a childhood only illness, see http://www.huttonlaw.com/reyes.htm). Aspirin also helps with blood thinning.

Catalysts

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Leaky Gut

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Updated on: 08/30/1999

A catalyst is something that ***may*** assists a disease - for example overweight is a catalyst for diabetes. IDEF / CFIDS is an infrastructure problem, the body immune system is not functioning correctly! We know that a fit, unstressed individual recovers from illness better than an unfit stressed individual so the prescription is simple: ***get as fit as you can***!

Physical Stress

What does this mean? First, recognize that food and chemical sensitivity is common once the illness occurs... you may wish to go on a strict anti-food allergy/sensistivity diet and bring in new (actually your former) food once a week to see if it has any effect. Keep to a very simple, regular and consistant diet - variety may be the spice of life, but variety in how you are feeling is not a desirable life.

Shift to an anti-allergy environment, use hema filters, etc. Read up on allergies and sensistivities and be aggresive in reducing the risk of *sensistivites* (a person allergic to bee stings may die, a person who is sensistive may have different symptomns).

Reduce weight and keep it off! Lost of appetite and weight loss is characteristic at the start of CFIDS, unfortunately weight gain often occur later when the person's metabolism slows down and they often start eating 'for energy'. Take a nap for energy and not a donut!

Have your iron level checked! 10% of CFIDS suffers have an iron surplus - this is a clinical illness that have some of the

characteristics of CFIDS.

Do not overdose on minerals or vitamins! You have an immune disfunction or dis-regulation. Large dosages will not be used and may help to further confuse the immune system. Keep to the FDA amounts! Be very careful of the B-vitamins, large dosages of B-vitamins are common before the onset of CFIDS, they may help deal with "stress" but they may also assist the illness.

Maintain the highest level of activity that does not result in the next day being a recovery day. You may wake up sore and tired, but each day you will find that you can do more!

Psychological Stress

Psychological stress may be the worst catalyst! Do not feel guilty about not looking sick, I have used the line

> "I have an immune disfunction, the only disease that I know that is similar is AIDS. With AIDS it is an immune DOWN regulation so people catches disease easily, I have an UP regulation which means that I get a constant replay of any virus that I have had before - not fun"

Enjoy life! Get off agendas and schedules (I'm an over-achiever because of a handicap, so it meant letting things go)! Be GOOD to yourself!

Frugal CFSer

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Updated on: 01/21/2000

IN PROGRESS

CFSers' are often reduced to very low financial resources, heavy brain fog and low physical capacities... this combination often result in a diet that hurts and not helps the CFS patient.

The $20 or $40/month CFSer

Below are suggestions from CFSers on what they would pick in supplements if they were reduced to only $20/month for ongoing supplements PLUS a set of single bottles of one time supplements. After this, they cite what they would do if they could add another $20. If you wish to contribute, please email your choices to CFS@Folkarts.com and include what % (from your own experience) that you believe that your energy level improved from this combination (with 0% being worst CFS and 100% being completely normal).

KenL@Folkarts.com

- 1500 mg of B12 : 10 cents per day
- 500 mg of Niacin: 3 cents per day
- 2400 GDU of Bromelain: 50 cents per day

for $0.63 per day or $19.00 per month [Improvement 50%].

One time bottles of:

- Zinc
- Chromium
- Selenium
- Magnesium Fortate
- B-100

taken at one tablet per day until exhausted.

With an addition $20, Yarrows Whey - 2 lbs (32 ounces) taken at 1/3 ounce taken three times per day (see http://store.yahoo.com/iherb/whey.html) [Improvement 10%]

Breakfast

Porridge with skim milk	

Lunch

Peanut butter and jam sandwiches on Rye Crisp Bread	

Snack

Peanut butter and jam sandwiches on Rye Crisp Bread	

Dinner

Rice	

Snack

Peanut butter and jam sandwiches on Rye Crisp Bread	

Red Blood Cells

Dr. Cecile Jadin's Papers are now available in full Click Here

NMH

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Updated on: 12/07/1999

Low blood plasma volume and low red blood cell count was been reported as typical for CFS patients (Bell etc: up to 50% below normal). Since oxygen is carried by the red blood cell, this results in low oxygen rates in the blood (found to be up to 50% below normal in some patients [*]). Low oxygen level produces many symptoms - typically "***brain fog***" (for other symptoms)

Dietary Assist

If vitamins deficiencies are corrected, and the diet is modified to include food ideal for the creation of red blood cells, some improvement would be expected.

The following are needed for forming red blood cells (reference)

- vitamin E
- Vitamin B6 Pyridoxine
- Vitamin B12 Cyanocobalamin
- Folic Acid (Folacin)
- Copper

Peanut Butter

1 oz of peanuts OR 2 Tbsp of peanut butter contains the following percentage of RDA allowance

Vitamin E	25%	Niacin	19%
Folate	10%	Thiamin (B1)	8%

B6	4%	Riboflavin (B2)	2%
Magnesium	12%	Copper	10%
Phosphorous	10%	Potassium	10%
Zinc	6%	Iron	4%
Calcium	2%		

Prescription Drugs

The following drugs may have some effect. There are some experimental trials (no reports). These drugs do not have 'generics'.

Colony-Stimulating Factors (CSF)

Proteins that stimulate the development of blood cells are called colony-stimulating factors (CSFs) or growth factors. They belong to a class of proteins called "cytokines". (For more background see http://www.bmtnews.org/newsletters/issue14/colony.html)

Common Side Effects of Colony-Stimulating Factors
Rash, Bone Pain, Muscle Pain, Weakness, Fever, Headache, Chills

Erythropoeitin

Epogen

A recombinant human erythropoeitin used to treat anemia associated with chronic renal failure for patients on dialysis. (see http://www-ext.amgen.com/news/EpogenNews.html for Epogen news)

It is reported Dr.Nancy Klimas (the University of Miami) is *proposing* a study this drug with CFS patients. Dr Streeten has a published paper on epogen in Neurally Mediated Hypotension (NMH) patients with low blood volume.

Procrit

PROCRIT helps increase the body's natural supply of **erythropoietin**, a protein made in the kidneys that is critical to red blood cell production.

See http://www.procrit.com/procrit/index.html

rHuEPO

See http://www.va.gov/publ/direc/health/infolet/119702.htm

GM-CSF and Interleukin-3

Foster growth of both red blood cells and platelets. The growth of platelets may thicken blood - and thus caution/research is definitely needed.

Blood Thinning

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STEP ONE: Go on a HEART WISE diet... there are many foods that will thicken blood or narrow blood vessels -- margarine etc, these must be eliminated first!

There are different types of "blood thinning" that may help CFS patients. The goal is to counter the following "thickening" or blood clotting activities...

- platelet activation
- fibrinogen
- thrombin generation

[info, more info on Coagulation - *thick blood*].

Note: This is NOT a recommendation to take any of the following - it is simply a listing of items that you may wish to research.

unknown mechanism

- DMAE: "helps prevent sludging or clumping of red blood cells and makes more of them available for carrying oxygen to the tissues. DMAE also has several positive influences on red blood cells." http://healthkraze.com/glossary.htm

platelet activation suppressors

The following suppresses platelet activation (one form of thickening)

- Bromelain (****) recommended
- Aspirin (ASA - acetylsalicylic acid) [1], salicylic ester of acetic acid, white willow bark caps,
  (82 mg works better than a normal 325 mg aspirin: a larger dosage works less well! [study])

- Concord Grape Juice [7]
- Oil of Evening Primrose [6]
- Omega-3 [10]:
  - From Flaxseed Oil or Fish
  - Personal note: we use Udo's Oil on salads (also contains Primrose Oil)
- Ginger [2, 4], disputed [3]
- Garlic [5]
- Ginkgo Biloba [8]
- CoQ 10 [9]
- Taurine

thrombin generation

- Bromelain proteases is suspected to reduce human platelet aggregation, and thrombus formation.[dosage: 920mg/day , 750mg to 2250 mg/day]. Review of its history and literature see:
  - http://www.thorne.com/altmedrev/fulltext/bromelain1-4.html
  - http://www.thorne.com/altmedrev/brom3-4.html
- PIRACETAM, also called nootropil (for studies: http://www.ds-health.com/piracet.htm) Piracetam inhibits the production of thromboxane B_2
  - NOT available in the US. [To locate a Source]

fibrinogen generation

- Niacin (B3) decreases plasma fibrinogen. http://www.lef.org/protocols/abstracts/abstr-049.html#2

Food with Coagulant Activity

Note: Some both contains both thickening and thinning (carrots, pineapple)

Source: Dr. Duke's Phytochemical and Ethnobotanical Databases

Thickening Food (AVOID!)

Coagulation chemical	Food
ARACHIDONIC-ACID	Brussels Sprouts, Carrot, Date Palm Fruit, Dong Quai Root, Garlic, Scotch Kale, Soybean seed
VERBENALIN	American Dogwood Bark

SEROTONIN	Avocado, English Walnut, Pineapple Fruit, Plantain Fruit, Plum, Stinging Nettle,

Thinning Food (Take!)

Anticoagulation chemical	Food
CITRIC-ACID	Anabasis, Black Currant, Carrot, Celery, Chives, Giant Taro, Horse Chestnut Bark, Monk's Hood, Gooseberry, Lingonberry, Onion, Papaya, Pineapple, Soursop, Spring Adonis, Sweetsop, Wine Grape
CATECHIN, D-CATECHIN	Babul, Black Currant, Green Wattle, Gooseberry, Lingonberry, Wine Grape
SALVIANOLIC-ACID-A	Tan-Shen Root
TANSHINONE-II-A	Tan-Shen Root
PAPAIN	Papaya
HERACLENIN	Carrot

fibrinogen / thrombin generation

Thickening Food (AVOID!)

Thrombogenic chemical	Food
VINBLASTINE	Rosy Periwinkle

Antifibrinolytic chemical	Food
GALLIC-ACID	Strawberries, Tarragon, Carob, Tea, Cashews, Teaberry, Wintergreen, WitchHazel, Mango, Bayberry, Evening Primrose, Guava, Pomegranate, Rhubarb, Raspberry leaf, Sage, Thyme, Bilberry, Blueberry, Lingonberry, Wine Grape
HEDERAGENIN	Black Caraway, Black Cumin, Fennel-Flower, Nutmeg-Flower, Roman Coriander

LINOLEIC-ACID	Garlic, Onion, Chives, Cashew, Dill, Celery, Peanut, Beet,
PALMITIC-ACID	Garlic, Onion, Chives, Cashew, Dill, Pineapple, Peanut, Cauliflower, Beet,....
PENTAGALLOYL-GLUCOSE	Chih-Shao Root

Thinning Food (Take!)

Antithrombogenic chemical	Food
QUERCITRIN	Coca Leaf, Cauliflower, Catechu, Fishwort, Hops Fruit, Japanese Knotweed, Neem, Oleander, Prostrate Knotweed, St. John's-wort, Tea Leaf, Tomato, Woodland Hawthorn, White Mulberry Leaf, Yarrow
RUTIN	Coca Leaf, Cauliflower, Catechu, Fishwort, Hops Fruit, Japanese Knotweed, Neem, Oleander, Prostrate Knotweed, St. John's-wort, Tea Leaf, Tomato, Woodland Hawthorn, White Mulberry Leaf, Yarrow

Fibrinolytic chemical Breakdowns of fibrin, usually by the enzymatic action of plasmin.	Food
GINSENOSIDE-R-O	Ginseng Root
PANAXYNOL	Ginseng Root
CYCLOALLIIN	Garlic, Kurrat, Onion
BROMELAIN	Pineapple
Curcumin	Indian Saffron, Turmeric, Ginger

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Supplement Schedule

Dr. Cecile Jadin's Papers are now available in full Click Here

NADH
CoQ10
Vitamin B-12
Malic Acid
Vitamin E
Iron
Zinc
Glycyrrhizic Acid

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The following is my personal supplement schedule. Some important issues are described at the bottom.

- ASA - Aspirin or ASA tablet (if approved by MD)
  - ideal dosage unknown: I'm 83gm (baby) tablets now
- AXB - Antibiotic (if prescribed by MD)
  - I'm doing 300 mg of doxycycline (150mg every 12 hrs)

Upon waking (usually I do a pre-breakfast walk):

- 15 mg NADH
- 100 mg CoQ 10
- 1000 IU Vitamin E
- 500 mg No-Flush Niacin
- 1 tablet Magnesium Malate Forte (500 mg Malic acid)
- with Orange Juice (not from Concentrate or Organic)

Hour before breakfast

- IMUPLUS or Immunocal package [Why?]

Breakfast

- baby ASA (82mg)
- AXB (delay 1 hr if not to be taken with food)
- 500 mcg B-12
- Multi-B vitamin (for example "Balanced B-100")

- with 8oz of Concord Grape Juice

Lunch

- Minerals (not to be taken with AXB)
  - Selenium: 400mcg
  - Zinc: 60 mg
  - Chromium: 400 mcg
  - (Magnesium: 250+mg from Malate, etc)
  - (Potassium from juices)
- 100 mg CoQ 10
- 1000 IU Vitamin E
- 1 tablet Magnesium Malate Forte (500 mg Malic acid)
- with 8 oz of Orange juice
- Yogurt with active culture (for AXB)
- Peanut Butter (red blood cell ingredients)

Hour before Supper (3-4 pm)

- IMUPLUS or Immunocal package

Supper

- baby ASA (82 mg)
- AXB (delay 1 hr if not to be taken with food)
- 500 mcg B-12
- 1000 IU Vitamin E
- with 8oz of Concord Grape Juice

Evening Snack

- 1000 mcg B-12
- 500 mg No-Flush Niacin
- 100 mg CoQ 10
- 1000 IU Vitamin E
- with 8oz of Concord Grape Juice
- Yogurt with active culture (for AXB)
- Peanut Butter (red blood cell ingredients)

Issues:

- Many CFS patients do not absorb vitamins/minerals well, and some vitamins may need to be given as shots etc... Problems with poor B-12 absorption is common.

- The quantity above are based upon my ***own experience***. They are dosages that produced a distinct beneficial change ***with me*** within 4 days of an increase.

This is not medical advise. Simply a statement of what worked for me... Always keep your MD inform of your schedule and supplements. Always observe the warnings on all supplements.

Diet pH

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Food pH

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Updated on: 01/01/2000

Diet may be taken another step due to CFSer's having alkaline blood (and thus acid urine). The foods listed below are believed to modify blood pH.

To help combine this list with the other food lists on this site, the foods are color coded:

blood thickener (bad), blood thinner (good), unknown on thickening

digest easily (good), digest poorly (bad)

Amount	More Acid Blood (GOOD) ***Meats, Fish, Grains, Nuts***	More Alkaline Blood(BAD) ***Fruits, Vegetables, Millets, Almonds, Dried Beans***
High	Pudding, jam, jelly, table salt (NaCl), beer, yeast, hops, malt, sugar, cocoa, white (acetic acid) vinegar, processed cheese, ice cream, beef, lobster, pheasant, barley, cottonseed oil, hazelnuts, walnuts, brazil nuts, fried foods, soybean, and carob	Baking soda, sea salt, mineral water, pumpkin seed, lentils, seaweed, onion, taro root, sea vegetables, lotus root, sweet potato, lime, nectarine, persimmon, raspberry, watermelon, tangerine, and pineapple.

Moderate	Nutmeg, coffee, casein, milk protein, cottage cheese, soy milk, pork, veal, bear, mussels, squid, chicken, maize, barley groats, corn, rye, oat bran, pistachio seeds, chestnut oil, lard, pecans, palm kernel oil, green peas, peanuts, snow peas, other legumes, carrots, garbanzo beans, cranberry, and pomegranate.	Spices, kambucha, unsulfured molasses, soy sauce, cashews, chestnuts, pepper, kohlrabi, parsnip, garlic, asparagus, kale, parsley, endive, arugula, mustard green, ginger root, broccoli, grapefruit, cantaloupe, honeydew, citrus, olive, dewberry, loganberry, and mango.
Low	Vanilla, alcohol, black tea, balsamic vinegar, cow milk, aged cheese, soy cheese, goat milk, game meat, lamb, mutton, boar, elk, shell fish, mollusks, goose, turkey, buckwheat, wheat, spelt, teff, kamut, farina, semolina, white rice, almond oil, sesame oil, safflower oil, tapioca, seitan, tofu, pinto beans, white beans, navy beans, red beans, aduki beans, lima beans, chard, plum, prune and tomatoes.	Most herbs, green tea, mu tea, rice syrup, apple cider vinegar, sake, quail eggs, primrose oil, sesame seed, cod liver oil, almonds, sprouts, potato, bell pepper, mushrooms, cauliflower, cabbage, rutabaga, ginseng, eggplant, pumpkin, collard green, lemon, pear, avocado, apple, blackberry, cherry, peach, and papaya.
Very Low	Curry, Koma coffee, honey, maple syrup, vinegar, cream, butter, goat/sheep cheese, chicken, gelatin, organs, venison, fish, wild duck, triticale, millet, kasha, amaranth, brown rice, pumpkin seed oil, grape seed oil, sunflower oil, pine nuts, canola oil, spinach, fava beans, black-eyed peas, string beans, wax beans, zucchini, chutney, rhubarb, coconut, guava, dry fruit, figs, and dates.	Ginger tea, umeboshi vinegar, ghee, duck eggs, oats, grain coffee, quinoa, japonica rice, wild rice, avocado oil, most seeds, coconut oil, olive oil, flax oil, brussel sprout, beet, chive, cilantro, celery, okra, cucumber, turnip greens, squashes, lettuces, orange, apricot, banana, blueberry, raisin, currant, grape, and strawberry.

Sources

- http://allocca.com/detox.htm

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Peanut Butter

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Updated on: 01/18/2000

Several CFSers report a strong desire to each peanut butter (myself included). Peanuts are not nuts but legumes (root crops). Many believe that the body is asking for it because it needs the benefits found in peanut butter. These benefits appear to include the following:

- makes blood more acid [CFSers have too alkaline blood]
- high in resveratrol (1.7 to 3.7 micrograms per gram of peanut *)
  - "now known as antioxidants, cancer chemopreventive agents, and also known to reduce mortality from coronary heart disease by increasing high density lipoproteins like cholesterol and inhibiting platelet aggregation"[*]
  - adversely affects some phrases of viral reproduction [*]
- contains all of the ingredients (except B12) for red blood cell production [CFSers have low red blood cell counts]
  - folic acid, vitamin E, fiber, and zinc [*] For details: http://www.peanut-institute.org/NutritionBasics.html

More readings:

http://www.peanut-institute.org/ScientificResearch.html

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Anticoagulants

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International CFS Information

Updated on:
06/04/2000

On the left are various non-prescription anti-coagulants that significant *positive results* have been reported from. *For other herbal anti-coagulant click here*.

Ideally, the Hemex's ISAC Panel should be done and appropriate drugs/supplements should be done. The reality is that the state of anti-coagulation treatment medical knowledge is very limited and prescription Heparin is the probable outcome of the test. There is no definitive knowledge on the effectiveness of non-prescription anti-coagulants.

Heparin is administered by injection, requires regular monitoring and may result in significant bone loss (a significant issue for many females). Some people elect this to be a 'fall-back' protocol if other items are ineffectual.

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Antivirials

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Black Elderberry

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Virus reactiviation and unusual growth is commonly reported with CFIDS. This may be a by-product of a Rickettsia/mycoplasma infection.

"it has been demonstrated that that mycoplasma can induce increased cytokine levels when infected in white blood cells, and ***can allow dormant viral infections to activate and replicate at abnormal levels***." **Bill Paspaliaris [*]**

Regardless of whether the viral load is primary or secondary with CFIDS, attacking the viral load with anti-vir1als assists the body in attacking all of the infections present. The items on the left are possible anti-virials, some are non-prescriptions, some are prescription. Other anti-virials are:

- Whey (immunepro is recommended as the most effective)
- Olive Leaf Extract (EastPark is recommended)

HHV6 antivirials

The following indicates some success with treatment of HHV6 (or HHV6A) with anti-herpesvirals.

- http://www.ncf-net.org/library/jcfsvirus95.htm
- http://pharminfo.com/pubs/msb/valacyc.html

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Black Elderberry

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Updated on: 06/04/2000

Sambucol Black Elderberry has been demonstrated to be an effective antiviral, see:

- http://www.tiud.com/ssb/razeibar/research/research.htm .
- http://www.execpc.com/~keephope/report17.html

It is non-prescription and may be purchased easily in the US (Imported by Nature's Way). At present, the cheapest source appears to be http://www.iherb.com/iherb/sambucol.html

Home Made Option

(for other variations, see: http://www.execpc.com/~keephope/report17.html)

"The basic recipe is one pound dried elderberries, one fifth vodka, one gallon of water. Let soak three days, then put on a very low heat for about four hours to steam off the alcohol.

This makes a gallon of high quality extract for about $20, depending on what you pay for the elderberries. No sweetners or additives,though you can use other herbal antivirals in the mix if you like. I get elderberries from SF natural herb Co at 510 601-0700. They charge $14/lb which is about normal retail." - From CFSFMExperimental

ImmunePro

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Updated on: 07/17/2000

The following information is "as received" from the US distributor. For the opinion of Dr. Cheney on this product, click here. The distributor may be contacted at: contact@immunepro.com. Website: www.immunepro.com

This is not an endorsement nor are these claims verified.

Release Date: 3-22-2000

The Finest Milk Whey Protein Naturally Contains Exceptional Amounts of Undenatured Biologically Active Protein, Lactoferrin, Immunoglobulins and Serum Albumin. ImmunePro™ is the result of years of work to perfect a system that produces only the highest quality milk whey protein. The process uses no chemical modification or pH regulation in the whey production. There is no cheese produced in any part of our manufacturing process. The result: a superior product.

ImmunePro™ contains:

- Lactoferrin, an iron-binding, iron-modulating protein that enhances iron absorption when needed, also anti-viral, anti-bacterial and anti-inflamitory properties.
- Immunoglobulins (IgG), with numerous immune system benefits.
- Bovine Serum Albumin, along with Lactoferrin and IgGs contain generous amounts of Cysteine and Glutamine, precursors in Glutathione (GSH) production.

Undenatured Protein is Essential for a Vital Immune System
ImmunePro™ maintains exceptional amounts of the biologically active proteins including the vital protein bound fats that whey protein isolates remove.

Amounts per 10 grams

Biologically Active Proteins*	%	Amounts per 10 grams
Lactoferrin	3.4%	285 milligrams
Immunoglobulin (IgG)	16.1%	1.4 grams
Bovine Serum Albumin	2.5%	210 milligrams
beta-lactoglobulin	48.0%	
alpha-lactalbumin	13.7%	
Total:	**83.7%**	

Compare, so you will know you are receiving the highest biological value of the most beneficial proteins.

Distributed by: Wellsprings contact@immunepro.com El Cajon, CA 92020

*This analysis of ImmunePro™ was performed by an independent laboratory using the standard SDS PAGE Gel method for protein fraction analysis of the protein content. These statements have not been evaluated by the FDA. This product is not intended to diagnose, treat, cure, or prevent disease.

Traditionally, even the most expensive whey products have been the byproducts of cheese production with definite limitations in preserving the complete biological activity of the whey proteins. Normally they go through a heating process, chemical modification and pH regulation to produce cheese. This denatures the whey proteins.

It may be necessary to introduce ImmunePro™ into the diet gradually, at a
reduced intake, if you are unaccustomed to this undenatured whey protein.

Greetings,

Wellsprings is happy to announce that ImmunePro™, the new name for ImmunoPro, now has a web site available on the internet. www.immunepro.com

Its focus is as an information and educational aid and we welcome you to
utilize it for yourself and others. There will be no product sales from our
web site. If you have a web site or know of an appropriate one and wish to link to us we are fine with that.

Please use the Email in our web site for all correspondence. If you have our previous Email address in your file please correct it, as it will be
discontinued. Thank you.

Michael Keenan
Managing Director
Wellsprings

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Whey

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The watery part that forms along with curd when milk thickens. It contains the water-soluble constituents of milk and is essentially a 5 percent solution of lactose in water, with some minerals and lactalbumin. Whey is removed from the curd during the process of making cheese. Whey Protein comprising major isolate fractions: 51% Beta Lactoglogulin, 20% Alphalactalbumin, 10% Immunoglobulin, 10% Bovine serum albumin and 7% minor fractions: betamicroglobulin, lactoferrin, lactoperoxidase, lysozyme, lactollin and relaxin

Non-Denatured Whey

Many whey products are by-products of cheese production (with little care taken over maintaining the biological activity of its components). These produces go through a heating, chemical modification and pH regulation in the process of producing cheese. This process "denatures" the whey proteins of the milk (breaks down or destroys the original components). This is why non-denatured whey is preferred.

Side Effects

- start out at one teaspoon daily, and slowly increase it

"You may experience nausea, headache or gastrointestinal symptoms. Bloating at the start of therapy is common and can be reduced by slowly ramping the dose." - From Consent form for those using whey with Dr. Cheney

Article reviewing whey: http://www.optimune.com/goodopt2/omunefinal2/WhiteSheet.pdf

Little Miss Muffet,
sat on a tuffet,
eating her curds and whey,

... and since the heroine of the nursery rhyme is assumed to be

Patience
Moffat, the daughter of Thomas Moffat (d. 1604), an Elizabethan physician
-- we must assume it is good medical advice...

The contents of some common Whey products are shown below:

Amino Acid	Immunocal	Jarrows Whey *	ImuPlus	ImmunoPro
Alanine	4.37	2.84	4.55	4.2
Arginine	1.73	1.72	1.81	1.9
Aspartic Acid	10.00	7.28	10.20	9.6
Glutamic Acid	16.00	11.40	16.50	15.0
Glycine	1.47	1.40	1.46	1.4
Histidine	1.51	1.48	1.52	1.3
Isoleucine	6.11	5.96	6.20	4.6
Leucine	9.46	7.36	9.81	6.9
Lysine	7.79	6.64	8.08	8.1
Methionine	1.64	1.24	1.77	1.9
Phenylalanine	2.61	2.28	2.68	2.4
Proline	6.11	4.24	6.20	5.2
Serine	3.95	3.68	4.01	4.2
Threonine	6.44	4.60	6.49	6.3
Tyrosine	2.66	2.04	2.72	2.7
Valine	5.65	3.92	5.71	3.8
Total	87.60	70.48	90.20	79.5
Solubility	87.7 mg/ml	n/a	84.8 mg/ml	
ph (%wt/wt)	6.3	n/a	6.5	
Protein (%wt/wt)	85.20	n/a	83.40	
Biologically Active Proteins	%	%	%	%
Lactoferrin				3.4
Immunoglobulins (IgG)				16.1
Bovine Serum Albumin				2.5%
beta-lactoglobulin				48.0%
alpha-lactalbumin				13.7%

Some data From CFSFMExperimental at http://www.onelist.com/

Jarrows Whey numbers are typical for 'muscle building whey products'.

For notes on other Wheys click here.

Glutathione

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International CFS Information

Updated on: 07/17/2000

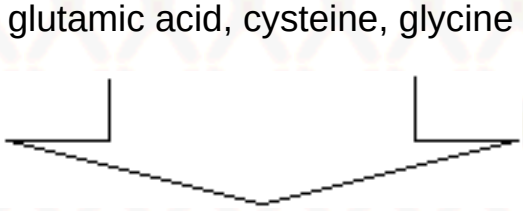
Glutathione is a short protein which consists of glutamic acid, cysteine and glycine. It is an antioxidant. Immunocal and IMUPLUS are milk whey protein **isolates** containing Glutathione (GSH) precursors. [Cheney on Glutathione], where as Immunepro is a **concentrate** (hence Cheney's preference for it)

Studies:

- Patients Improved with Glutathione, CFIDS Chronicle of Jan/Feb, 1998
- CFS patients have glutathione precursors consumed in abnormal amounts [*].

Information about Whey: http://members.toast.net/cec/nutrition/whey.html

Chemical progression



Products/protocols that increase Glutathione levels

Source: http://www.execpc.com/~keephope/report16.html

1. Injectable glutathione/ATP (100 mg glutathione +1 mg ATP one to three times weekly)
2. Reduced Glutathione - 500 mg 1 or 2X daily (Jarrow Formulas) - use with alpha lipoic acid & other ATP stimulators.

3. Cold processed whey proteins - Immunocal (most effective choice), IMUPLUS, Optimune.
4. **Selenium** (use 200 to 300 mcg daily). Do not exceed that amount for adults.
5. L-Cysteine 500 mg daily plus N-Acetyl Cysteine (NAC) 500 to 600 mg daily.

Note: when glutathione levels have bottomed out, try using all the above protocols simultaneously

Other Wheys:

- ImmunoPro:
  - Wellsprings (Michael Keenan) San Diego, Ca. Tel: 619-469-8196, is reputed to be 'stronger' than Immunocal or Imuplus. contact@immunepro.com
  - NEEDS: (I'm told the # will work internationally, though it will not be free). 1-800-634-1380.
- BioPure Protein:
  - New Zealand, 300 gram can costs $32 from Center for Natural Medicine Dispensary: 888-305-4288.
- Optimune is being reformulated
- Designer Protein is reported not to be cold process (thus *denatured*)
- LEF 379
  - November 1999: was reported to now contain carrageenan (decreasing effectiveness/other risks)
  - Janurary 2000: carrageenan was discontinued and replaced by guar gum :-)
- Jarrow Whey (~ $10/lb) - produced in US (thus Bovine Growth Hormone issues)

Exercise

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International CFS Information

Updated on: 12/17/1999

DR. CHENEY'S COMMENTS ON EXERCISE

(from CFSFMExperimental at http://www.onelist.com)

Golden Rule: find the boundaries of what you can do and then stay within them. Both trying to do too much, or pulling back and doing too little are counter productive.

Aerobic Training beyond certain limits cannot be attempted until you are much improved. Be very cautious about any aerobic exercise (any sustained activity, such as running, walking, or swimming, designed to raise the heart rate and increase oxygen flow throughout the body). The aerobic system is injured and reactive oxygen species (free radicals) generated in the mitochondria by excessive training may not be detoxified with resulting injury which can potentially be permanent (DNA damage). Walk, cycle or swim only as much as your body will allow, certainly no more than 20 minutes, three times per week. Aerobic exercise past a certain point can dramatically worsen this disorder.

Anaerobic Training - The anaerobic pathway is largely intact in CFIDS. Weightlifting, isometrics, and stretching can maintain muscle tone and strength and improve the elimination of toxins formed by the pathway itself. Do low level weight lifting with 1 to 20 pounds, using all muscle groups. Lift for 10 seconds then rest for 60 seconds - repeat for each muscle group. Do lift/rest cycles no more than 20 minutes three times per week. Sequential isometric contractions can be substituted for weight lifting. (This can be done while lying down.) Still use the 10 seconds on and 60 seconds off rule.

Rebound Exercise - with the bounce-back chair (a tall bungee cord-like contraption is probably the best form of exercise for CFIDS. Low level, non-vigorous bouncing for ten to fifteen minutes every other day is best. Less ill patients can add aerobic exercises between five minute periods of bouncing per the video-tape instructions. Its advantages include correcting dysautonmia, the dysfunction of the autonomic nervous system which underlies many of the symptoms in CFIDS. The Bounce Back Chair was studied by NASA to treat astronauts returning from orbit who fainted upon standing. It also improves immune regulation by pumping lymphatic fluid back into the blood. Lymph acts just like gamma gobulin. Finally, this exercise was shown by NASA to be 68% more efficient as an exercise routine than running. ("Efficient" means maximum gain for minimum effort.) It is therefore ideal for

people with little energy to spare. The Bounce Back Chair is available from Sun-Ray Supply at 1-800-437-1765. As of July 1998 is cost $395, plus $20 for shipping and handling.

Rebreather

Dr. Cecile Jadin's Papers are now available in full Click Here

Updated on: 02/25/2000

Comments and explanation from a CFIDS patients of Dr. Cheney (posted here with permission from original post on CFSFMExperimental)

.. first let me say that the oxygen on a partial rebreather is wonderful. It has completely stopped my headaches. It significantly reduces my muscle aches, and gives me more energy and mental clarity. I no longer worry about my weight being too low (120 lbs at 5'10") For the first time in three years I'm eating less and putting on weight. But not too much. :-) When I reached my normal 132 it stopped. I can't recommend the oxygen/carbon dioxide combo of this particular treatment too highly. And oxygen alone won't do what this treatment does. That's why you really need the partial rebreather - it gives you the carbon dioxide component that lets the oxygen get off the hemoglobin where it's stuck and into the cells of your body.

The prescription that Cheney wrote for me specified a partial rebreather mask. It took some calling to find a company that provided one, even here in the Dallas Ft. Worth area. If anyone wants to refer their medical equipment company to one that can tell them where to get a partial rebreather, they can call Rhema in Irving, TX. (972-445-0007) The masks can't be that unusual - I see them on ER all the time!! :-)

I just checked the extra mask I have that still has the paper inside the package. It's called "Airlife" and is made by Allegiance Healthcare Corporation in McGraw Park, IL 60085. Catalogue

#001203. Because it can be converted to a high concentration nonrebreather or a medium concentration nonrebreather as well as a rebreather, it's called a Three-In-One mask.

It consists of the mask that covers your nose, mouth and chin, a bag attached under the nose, two perforated circular areas on each side of the nose, and two rubber disks with little holes in the middle. The disks can fit over protruding knobs in the perforated areas.

One disk is designed to fit over the inner opening to the bag - making it a one way valve. (Exhaling slams the disk against the opening, closing it and preventing CO2 from entering the bag, which is filled with O2 when it functions as a nonrebreather. Inhaling pulls it part way up and allows you to breath air from both the O2 flowing in through the tube and from the bag.)

When the mask is used as a partial rebreather, the disk is not placed over the inner opening to the bag. When you exhale the bag fills with carbon dioxide. The bag should fully inflate when you exhale.

The key to this treatment is what happens when you inhale. The prescription should state "FIO2 of 35 to 40%". This means the fraction of inspired (or inhaled) oxygen should be 35 to 40%. The bag has to collapse between half and 2/3 when you inhale in order for this to happen. Cheney emphasized that the bag should never, never collapse more than 2/3.

I finally figured out that if the bag collapses halfway, then you've just inhaled 50% oxygen (from the tube connected to the mask from the tank/canister) and 50% carbon dioxide (from the bag). If the bag collapses 2/3, you've inhaled about 33% oxygen and about 67% carbon dioxide.

Two factors control how much the bag collapses and therefore how much oxygen and carbon dioxide you breath in: the flow rate of the oxygen, and the placement of the rubber disks on the side of the nose over the holes.

The flow rate should be between 8 and 12 liters per minute. That's a lot of oxygen, and it's why the medical company switched me to a canister of liquid oxygen. They were bringing these huge tanks over four feet tall out to the house, and they were only lasting about a week and a half. And I was only doing an hour of O2 a day. The liquid canister lasts about six weeks. If I haven't been moving around, and I take 30 minutes of O2 lying down, a flow rate of 8 is great. If I get up and move around then I move it up to 10. The liquid canister is $150 dollars, but insurance pays the full 80%. At $30 bucks for six weeks of O2, that's $5 a week and a great return on my money in terms of improved comfort and functioning.

The placement of the disks took some experimenting. The instructions said not to use them at all. I had to in order to get the bag to collapse, and Cheney said most patients had to use them also. In fact, I have one disk taped over the holes, and the other disk loose so it's a two-way value. (Opens when I exhale, closes when I inhale.)

Well, I think I've given you every possible detail that I know about this. Didn't mean to write a book here, but hope some of it helps.

Immunocal

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Updated on: 03/18/2000

The following has been volunteered to this site. Provided for information only (if you have additional sources, please email!)

Websites:

- http://immunocal.org/
- www.immune-response.net/go/deb
- www.immunocal.com

From patent holder/manufacturer:

Immunocal®	$ 99.00

Reduced to $ 74.25 for internet orders .. and to $59.00 on your first order.. (see http://www.glutathione.com/option1.htm).

Imuplus

Dr. Cecile Jadin's Papers are now available in full Click Here

Updated on: 12/12/1999

The following has been volunteered to this site. Provided for information only (if you have additional sources, please email!)

IMUplus is available from N.E.E.D.S. for $82 or $88 for 60 pkts. their # is 1-800-634-1380.

http://www.biogene.net/ (Manufacturer/patent owner) has specials on occasionally, for example:

- 2 Cartons Of IMUPlus & 1 Portable Mixer, $160.
- by the single case: $89
- 6 case lots: $80/case

Mixing Whey

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International CFS Information

Updated on:
01/05/2000

Other suggestions from CFSFMExperimental:

- Imuplus sells a mixer with a plastic paddle that works well.
- Questions have been asked whether a high speed steel mixer may damage the whey.
- "The easiest way to mix whey is to put it in a small jar with lid, shake it and let it sit for half and hour, shake it again if you think of it. It will totally dissolve into almost a clear juice. Just have to think ahead."
- Should NOT be mixed with juice (acid in juice will damage), may be mixed with milk products or water.

Quote from Cheney presentation

February 1999: (Transcription)

. . . immune-activation states can also induce the activation of endogenous microbes in the presence of glutathione deficiency. And that might explain why in this immune-activation state that we call Chronic Fatigue Syndrome you see a lot of endogenous viral activation such as EBV, CMV, HHV6, mycoplasma incognitus, chlamydia pneumonia, candida, and on and on and on. You see the activation of this microbial ecology, and why is this happening? It could be that it happens because cytokines in excess stimulate these organisms, especially in the presence of glutathione deficiency. The converse is true, however. In the presence of good glutathione levels, it's very difficult for that to happen.

. . . Conclusions from all of this are: Glutathione has potent anti-viral properties--if you raise the glutathione level you can stop the replication of most any, at least, intracellular pathogen. Chronic fatigue syndrome patients are glutathione deficient. Glutathione deficiency itself has a potent pro-viral effect. That is, not only does (high?) glutathione levels tend to act as an anti-viral, but glutathione deficiency produces a pro-viral effect. It can actually augment viral replication. Augment it from the case of toxins, toxins could augment viral replication and also cytokines themselves. So immune-activation states would itself augment these things.

. . . I'm trying to set the stage for how important it is to address this glutathione defect. It could be THE major issue in this illness. Maybe not so much in the beginning, but over time become the major issue. Because we're dealing with a sub-group of people who have cellular detox failure and all that that causes. Because if you

have cell detox failure, you become a canary to your environment. . . . If you get a glutathione defect, then you become vulnerable to your own cell toxicity, specifically the portal circulation.

We found out that when you give oral reduced glutathione, it helps a little bit in some people, especially these pressure toxic headaches they get. But when you keep raising the dose, they actually get sick again, and it was never a very impressive response. When we tried NAC we saw some evidence of toxcicity. In the use of NAC--I'm concerned about high-dose NAC in this disease. I think it may be toxic. We tried other methods to affect glutathione. Nothing seemed to be working.

Then we got wind of . . . undenatured whey protein, lightly denatured to preserve the peptide action of this milk protein. It's concentrated to about 90 percent protein and it's very, very lightly denatured. In fact, the more lightly they denature it, the better the action appears to be. And the more they denature it, the less active it appears to be. In fact, if you denature it completely, down to its constituent amino acids, it really doesn't work well at all.

People who normally have milk protein allergy seem to tolerate this, by and large. Not 100 percent, but by and large.

This is the data from a six month study. There were eight people entered into the study, seven of them completed the study. We got data on seven of them. One dropped out at three months for a reason involved with the design of the study. (Note from Carol: the patient dropped out when the study protocol randomly required half of the participants to drop to one packet a day at the half-way point. He was improving so much on two packets a day that he refused to drop back, so he quit the study.) The first three months of the study we treated with two packets a day, and then the second three months, half were randomized to two packets a day and half were randomized to one packet a day. We wanted to see if you could tell a difference clinically or by other means between one packet a day versus two packets a day.

We did this because there was some indication that the more you treat with this, the higher the dose, the better the effect. When you look at the group that goes from two packs a day to one pack a day, you can see this nice dip where they started going back up (in their urine lipid peroxides). Suggesting that one pack a day doesn't work very well. (He's referring to a slide of a chart here, I think.)

By the way, you can extend this--there are people, I've discovered since the study was done, that do really well on three packs a day and not very well at all on two. (Note from Carol: Cheney told me in October that he has patients on 4, 5, and even 6 packets a day!!!) So clearly there is a dose response issue. Two packs a day would probably be my recommended starting dose, but I wouldn't hesitate to go up if it seemed like it wasn't working.

This is the exciting stuff. We wanted to see not only if this product improved glutathione functionality, which it did, but we also wanted to see if it knocked out micro-organisms, like the PNS article said it would. Chlamydia pneumoniae is an intracellular pathogen. It's a common cause of hospital-acquired pneumonia. It ubiquitously infects the population, but seems to activate under certain conditions. And if it activates, some of the clinical conditions of this organism are chronic sinusitis, pharyngitis, and laryngitis. But it also gets into the central nervous system.

In a study published by a neurologist out of Vanderbilt showed that chlyamdia pneumoniae may be a very important pathogen in multiple sclerosis. Indeed, data they shared with me recently (and this is coming to publication soon) showed that 80 percent of the cerebral spinal fluid of MS patients is actively infected with this organism. Versus 15 percent of other neurological diseases that are not MS. In a journal-published article on neurology, aggressive treatment for chlyamdia pneumoniae rapidly reversed an acute exacerbation of multiple sclerosis.

So we measured IgM levels for this pathogen at Vanderbilt. Most laboratory measurements of this organism are not very good, so this is a research grade assessment, and probably may not generalize to the run-of-the-mill types of tests that you might get in your local labs. But IgM elevations of 1 to 1600 (?) dilutions is evident of significant active infection with this organism. Six months later, it just wiped it out. IgM just fell to normal levels. It didn't really matter whether you were taking one pack a day or two packs a day. Just wiped it out. Makes you wonder what this might do for MS. Think about that.

We also looked at mycoplasma fermentans and mycoplasma penetrans. Both of these pathogens have been linked to Gulf War Syndrome. They've been linked to chronic fatigue syndrome. Again, they may be a relatively ubiquitous mycoplasma species, intracellular, and can cause a variety of problems when active. Again, by PCR done in Irvine, California. We were able to show that this product also wiped out mycoplasma incognitus and penetrans.

Then we looked at HHV6. It was a little mixed here. We tested three people.

By the way, this study was designed to do some microbial testing on everybody, but not everything on everybody. The patients were allowed to pick and choose depending on what we had in their chart before. We weren't able to do everything on everybody because they were paying for this.

We did HHV6 rapid culture testing, which is a technique developed by a company in Wisconsin. This particular culture technique uses an intermediate (captures fiberglass?) cell line, so that you are positive only if you are really infected, so it reduces false positives to zero. That is, under these conditions, all normal people are negative. You have to do that because HHV, both A and B strains, are relatively ubiquitous. Under these conditions, we had two positives and one negative at beginning of the study. The person on two packs a day went to zero culture (negative); the person on one pack a day stayed positive. The person that was negative stayed negative. Suggesting that maybe this isn't as good against viruses as it is against bacteria, but at two packs a day it might be good against viruses. Again, the numbers (of participants) are small.

But to me, the satisfaction of this is tremendous because I'm always faced in this disease population--well, are they sick from EBV? or are they sick from HHV6? or are they sick from mycoplasma incognitus? or are they sick from c pneumoniae? And the [traditional] treatment for mycoplasma and c pneumoniae is 18 months of triple drug antibiotic therapy. And if we're wrong on this issue, we've wiped out their gut flora and leave them a gut ecology cripple for the rest of their lives. So now what we have is a nice way to address almost any micro-organism that happens to be there. Just as the PNS article suggested.

UNDENATURED WHEY

Detoxification and Antimicrobial Benefits

(written by Carol Sieverling, based on transcripts of a lecture by Dr. Cheney and recent letters from his Clinic)

In January Dr. Paul Cheney informed those scheduled to participate in his study of un-denatured whey that he was switching to a new, more powerful product: ImmunoPro. Based on sophisticated biochemical analysis, it is believed to be two to

three times more powerful than the other two whey products currently used by his patients, Immunocal and ImuPlus. It also happens to be cheaper!

Why all the excitement about un-denatured whey? Dr. Cheney's original study with Immunocal showed that it had the ability to restore intracellular glutathione levels, something that neither glutathione supplementation nor injections have been able to accomplish to any significant degree. Virtually all CFS patients have low levels of glutathione. Even if glutathione levels are at the low end of the normal range, other markers (elevated lipid peroxides, elevated citrate, depressed alpha ketogluterate) usually indicate problems with its functionality. Dr. Cheney believes that this deficiency is the key problem in CFS patients, especially over time.

Glutathione has many important functions, two of them critical for CFS patients. First of all, it is a major player in our detoxification pathways. Glutathione deficiency impairs the body's ability to get rid of toxins, whether they are environmental or the by-products of cellular metabolism. We slowly become toxic, storing away poisons in our fatty tissues, muscles, organs, and brain. This cellular detox failure can make us canaries to our environment. Good detox programs that have worked well on people with other illnesses can actually put some CFS patients in the hospital. Therefore the glutathione deficiency needs to be addressed before any serious attempt is made at detoxification. Immunocal, ImuPlus and now ImmunoPro appear to restore normal levels of intracellular glutathione in most patients, allowing the body to detoxify. (If the dose is too high it is possible to mobilize more toxins than the body can handle, resulting in a "crash". Reducing the dose eliminates this problem.)

Secondly, glutathione is a powerful antiviral/antimicrobial mechanism. A glutathione deficiency compromises our ability to keep old viruses dormant and fight off bacteria. This is why so many of us test positive for EBV, CMV, HHV6, Mycoplasma, and Chlamydia Pneumoniae, etc. These pathogens are likely not the cause of our illness, but simply opportunistic infections. Indications are that undenatured whey can stop the replication of any intracellular microbe, including HHV6, Chlamydia Pneumoniae, and Mycoplasma. Treating them can greatly reduce symptoms and improve well being, and in some cases lead to recovery.

Patients who tested positive for Chlamydia Pneumoniae and Mycoplasma before the original study tested negative at its conclusion six months later. One packet a of Immunocal, the original patented undenatured whey, was sufficient for the C. Pneunoniae. Regarding HHV6, only those on two packets a day of Immunocal tested negative after treatment.

The traditional treatment for Mycoplasma and Chlamydia Pneumoniae is 18 months of triple antibiotics, which can wipe out a patient's gut flora and leave them a gut ecology cripple for the rest of their lives. This treatment approach is much gentler and appears to be just as effective.

ImmunoPro is available from NEEDS, 1.800.634.1380. A 300 gm canister is $39.95. Six canisters are $36 each, 12 are $32 each. Dr. Cheney recommends starting with one teaspoon twice a day, increasing to two teaspoons the second day, etc. In the current study half the patients are taking two scoops a day (a total of 20 grams, or 4 tablespoons), and the other half are taking four scoops a day. Some patients may need to start with much smaller doses and slowly work up. Dr. Cheney recommends split doses in distilled water on an empty stomach. For those familiar with Immunocal or ImuPlus, one scoop of ImmunoPro equals one packet, though ImmunoPro has two to three times the bioactivity. A three-month trial is recommended to determine if this product will be effective. As with all treatments, what works for some does not work for others.

Glutamine

Dr. Cecile Jadin's Papers are now available in full Click Here

Updated on: 12/12/1999

The following has been volunteered to this site. Provided for information only (if you have additional sources, please email!)

You can buy 250 grams of certified 99.6% pure pharmaceutical grade L-Glutamine from The Moby Dick society http://www.seaquake.com/bulk.html for $18.

And http://www.beyond-a-century.com/ has powder

- 100 grams for $7.50. Code 016.0
- 250 grams, $14.50. Code 016.5
- 1000 grams, $47.50. Code 016KG

At http://www.webvitamins.com/ has

- CL L-Glutamine 1000mg RR 60 Tablets, 9.50 (60 g total)

Olive Leaf Extract

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National CFIDS Foundation
International CFS Information

Updated on: 03/05/2000

Olive Leaf Extract is:

- a natural (non-prescription) antibiotic [NLM: *,],
- results in higher NO production, currently believed to be beneficial for cellular and organism protection [*] and
- appears to be an antiviral agent[*]

It has been reported effective with CWD infection - producing herxing.

Main Ingredients: Oleuropein (pronounced oh-lee-or-oh-pin) and Calcium Elenolate. Each brand has different concentrations, for example some run at 6% and others at 20%

Reference Material

- http://www.chiroweb.com/archives/15/15/20.html

Dosage

200 mg of Oleuropein (with bromelain suggested for better penetration) per meal will cause herxing in some people. Adjust dosage up or down until an acceptable degree of herxing occurs.

- 6% at 500 mg/capsule = 30mg/capsule or 6 capsules
- 20% at 500 mg/capsule = 100mg/capsule or 2 capsules.

NOTE: Olive Leaf Extract will kill L.Plantarum :-(If taking both, take them 12 hrs apart.

Penicillin / Sulfonamides - Avoid

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Updated on: 06/23/2000

"Cortisone is avoided as it reactivates the germ"
http://www.cfs.inform.dk/Nyheder.udland/cfsnews19aug.txt

"The sulfonamides stimulate rickettsial growth and thus are contraindicated in the treatment of these diseases." (Rickettsia is found in CFIDS often)
http://www.kcom.edu/faculty/chamberlain/Website/Tritzmed/LECTS/RICKETT.HTM
The following was received from one of my correspondents...

The penicillin family of antibiotics (which include penicillin derivatives) & also the cephalosporin family (including cephalosporin derivatives) are known as "cell wall inhibitors" Obviously, it would not make good sense to use a "cell wall inhibiting" antibiotic for a cell wall-less mycoplasmal infection. Taking penicillin can be like throwing kerosene on a fire, if it is taken, unknowingly, for an unsuspected mycoplasmal infection. Treatment with the wrong antibiotic can make a mycoplasma infection much worse. Penicillin is to have a stimulatory effect on the growth of cell wall deficient (CWD) microorganisms. In fact, it is used for this purpose in laboratory cultures, in order to induce and promote their growth. Penicillin itself is the worst of all (or best, depending upon one's purpose) Because it exerts its effect over a wide range of concentrations. ("Cell Wall Deficient Forms: Stealth Pathgogens", by Dr. Lida Mattman, 2nd edition, CRC Press, Baca Raton, Fl) Penicillin is one of the most popular & frequently prescribed antibiotics because it is one of the cheapest.....

Another important caution to beware of, when taking any of the tetracyline family of antibiotics (such as doxycline or Minocin) it is important to swallow the pill while standing or sitting upright, not lying down, and to drink lots of water with it in order to prevent an esophageal burn and possible painful scarring. This has to be kept in mind for anyone who is also treating pets.....

Also, Tetracylines, including minocyline and doxycyline, that are out of date are known to be TOXIC, reference "Nursing Drug Handbook". The self life for all drugs in the teracycline family is relatively short.
See http://garynull.com/Documents/Arthritis/free-living_amoeba.htm for additional information "The study of Cell Wall Deficient organisms is not an unknown field, but simply an esoteric specialty that has yet to be integrated into the routine of physician knowledge and practice." Mycoplasma is a Cell Wall Deficient organism.

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

Guide to Antibiotics

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Tetracyclines
Macrolides
Metronidazole

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Updated on: 06/11/2000

See http://becker.wustl.edu/lexicomp/patch_f/html/index/idxid1.htm for a good set of drug-fact sheets.

For effectiveness against certain bacteria see: http://www.mcmahonmed.com/wworks/CHARTS/antimicrobi/microbes.htm

Name	Notes	Protocols [Family]
doxycycline		N,M,R [T]
ciprofloxacin(Cipro)		N,R
azithromycin (Zithromax)		N,M, R [M]
clarithromycin (Biaxin)	Some strains resistant[*]	N,P, R
Trovafloxacin		A,M,R [Q]
BAY 12-8039		A,R [F]
Ceftriaxone		P
Chloramphenicol		M
Minocycline	1. can cause lupus like symptoms [*] 2. penetrates tissues better	M,R [T]
Sparfloxacin		M
Rifampin		M
Gentamicin		M
Lincosamides		M
Clindamycin		R

Erythromycin	M. hominis and otheres are resistant [*]	R
cetracycline	Some strains resistant[*]	R [T]
temafloxacin		R
difloxacin		R
Tetracycline	Some strains resistant[*]	R [T]
oxytetracycline	resistance seen by vets [*]	J [T]
Tiamulin		M
Enrofloxacin		M
Danofloxacin		M
Valnemulin	Very promising [*]	A,M
tylosin	resistance seen by vets [*]	M
Lymecycline		J, R,

Source of information: Links from:
http://home2.freegates.be/nvdeynde/mycoplasma/publications

Protocol Legends:

- N - Prof. Garth Nicholson
- J - Jadin
- R - Rheumatoid Disease
- A - New Drug
- P - (atypical/ Mycoplasma) Pneumonia
- M - Mycoplasma General [*,*]

Family

- F - Fluoroquinolone
- Q - Quinolone
- T - Tetracycline
- M - Macrolides (complete resistance exhibited by *M. hominis* [*])

Lactobacillus

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National CFIDS Foundation
International CFS Information

Updated on: 03/05/2000

There are several types of lactobacillus available, these include:

- Lactobacillus Acidophilus
- Lactobacillus Bifidobacterium
- Lactobacillus Casei (often in Cheese)
- Lactobacillus Plantarum (often in sourdough)
- Lactobacillus Salivarius
- For a full list of Lactobacilli see: http://www.dsmz.de/bactnom/nam1537.htm

These Lactobacillus have different characteristics and benefits. For the purpose of antibiotics with CFIDS, it appears that ***Lactobacillus Plantarum*** is the best one (superior to L. Acidophilus).

Lactobacillus Plantarum

- "A marked increase was found in the content of leucine, alanine, valine, isoleucine, glutamic acid, glutamine, arginine, lysine, methionine, phenylalanine, tyrosine and serine. Lactobacillus plantarum showed a higher proteolytic activity" [*]
- "promote the production of B vitamins in intestines" [*]
- "helps maintain the proper balance between Lysine and Argenine" [*]
- "It seems unique among the lactobacilli for *L. plantarum* to use mannose-specific adhesins, uncommon among gram-positive, but common among gram-negative bacteria, which makes it possible that *L. plantarum* competes with gram-negative other potential pathogens for receptor sites at the mucosal cell surfaces." [*] Editor Note: Rickettsia and Mycoplasma are gram-negative
- *"L. plantarum* is the only *Lactobacillus* not harmed by antibiotics and can be taken simultaneously with them."[*]
- One possible source for a suitable mixture:
- http://www.mailordercentral.com/webvitamins/prodinfo.asp?number=JA-JARRODOPH-200C&variation=&aitem=4&mitem=5

NOTE: Olive Leaf Extract will kill L.Plantarum :-(If taking both, take them 12 hrs apart.

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Terminating Antibiotics

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International CFS Information

Updated on: 02/16/2000

Question: How will one know when the antibiotic treatment is complete?

Answer: from Ken and Laurie

The following is a summary of our (Laurie and I) agreement with our MD on when we stop antibiotics:

- Duration will be at least the minimum recommended by Prof. Nicolson for each antibiotic (we will check with him for current protocol at the end of each antibiotic cycle)
- For each cycle/antibiotic change: the antibiotic will continue until we have no herxing effect from taking in a single dosage: all of the antibiotic for a day, and a high dosage of bromelain (4800 GDU+). This is done after herxing has completely stopped for the antibiotic and with 4800+ GDU of bromelain distributed throughout the day.

Whys?

- Testing means being off Antibiotics for at least 4 weeks, and there is concern that 'below detectable' levels may occur, as well as reserves in various parts of the body, which may mean a false negative.
- We hope that above "exit antibiotic shock dosage" will penetrate far enough to indicate if there are any other reserves remaining...

This page is strictly informational and not advise - if you and your MD's have other exit conditions that you are willing to share, please send them to cfs@Folkarts.com

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Bromelain

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International CFS Information

Updated on: 07/16/2000

For a review of this ENZYME extracted from Pineapples (Discovered in 1957 by Napier [*], originally called "ANAVIT F-3") see:

http://www.thorne.com/altmedrev/fulltext/bromelain1-4.html

- Reduces platelet aggregation (like aspirin does)
- Antithrombotic and anticoagulant activities [ref]
- Can be used to produce hypoallergenic bread from wheat [ref]
- Not known to cause Reye's syndrome (may be taken by all ages)
- Helps digest meat (a common problem for CFS patients)
- anti-inflammatory and pain relieving [*]
- May be taken long term without problems
- Antibiotic potentiation [ref]
  - Demonstrated with amoxycillin and tetracycline - *50-100%* [*]
  - (Warning: If you are herxing, it may make it a lot worst - wait until herxing stops - bromelain may cause it to resume :-) as the deeper infections are attacked)
- A report (one person) on CFSFMExperimental that it was prescribed by a Holistic MD for leaky gut, and healed it.
- If you are allergic to *bee stings*, *olive oil* or *pineapples* see above review *first*.

An excellent summary was prepared for CFSFM_Antibiotics,
http://www.egroups.com/message/CFSFM_Antibiotics/141?&start=135

Bromelain Protocol

The following appears to a more effective version of the 'aspirin

protocol'. As always, if successful (for energy level or brain fog), review hemex's research with your MD (http://www.hemex.com/cfs)

- 400-600 GDU of Bromelain taken 4 times per day
- May be taken with or without meals (some suggests between meals is better - but no research on this issue)
- If no effects at all then after 2 weeks discontinue.

NOTE: Bromelain and Piracetam appear to enhance each other for combating 'brain-fog', for CFIDS it is suggested that both be taken concurrently. There are no studies -- this is from experiences reported on CFSFMExperimental. Current *suggestion:*

- Bromelain: 2400 GDU/Day
- Piracetam: 1600+ mg/Day

GDU

"Today, bromelain is measured in MCUs (milk clotting units) or GDUs (gelatin
dissolving units).

One GDU equals approximately 1.5 MCU. Strong products contain at least 2,000 MCU (1,200-1,333 GDU) per gram (1,000 mg).

A supplement containing 500 mg labeled "2,000 MCU per gram" would have 1,000 MCU of activity.

Some doctors of natural medicine recommend as much as 3,000 MCU taken three times per day for several days, followed by 2,000 MCU three times per day. Much of the research, however, uses smaller amounts, more like the
equivalent of approximately 2,000 MCU in divided amounts in the course of a
day (500 MCU taken four times per day). "

From: http://www.healthzone.com/healthnotes/Supp/Bromelain.htm

Online Ordering:

http://www.webvitamins.com/ or any other eVitamin shop

Cost per day: ~75 cents per day [*].

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Aspirin Protocol

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National CFIDS Foundation

International CFS Information

Updated on: 03/04/2000

Aspirin Protocol

NOTICE: A better test for Coagulation is using Bromelain *[less risks, more types of anticoagulation]*, and it is suggested instead of the item below (click here for details):

The following appears to be effective in reducing symptoms for *some CFSer (approximately 30%)*. If it is successful, see http://www.hemex.com/cfs/ for a more effective protocol. Or, look at changing your foods

See http://www.seaquake.com/cfs-fm-recovery/meds1.html#18 for more information about what aspirin may be doing.

For 4 days:

- Take 82 mg (baby aspirin) or 1/4 of a 325 mg aspirin with breakfast and supper (higher dosages increase ringing in the ear for many - also a *larger* dosage thins ***less***! [see recent medical study on blood thinning effectiveness of aspirin]. More is NOT better!
- If no improvement - stop
- If improvement - continue taking for up to 10 days at most [during this period, ask for MD permission to continue]. Most MDs will ok a continuation unless there is another health issue.

Note: Some people have found that 82 mg had little effect, but 325 mg had significant effect.

My experience with Aspirin

My history is simple (fuller story):

- Found the hemex site and got interested, drop material off at my MD's office (so she could read before next appointment)
  - Researched the drugs used and found some research articles that found aspirin was as effective as warfarin ..
  - Researched aspirin - found that it stayed in the blood for only 6 hours....(thus warfarin is more convient!)
- Started to deteriorate (stress from IME exam), so I decided to try aspirin "just to see what would happen -- since there was a chance, have not had any problems with aspirin in the past..."
  - Checking the warnings on the aspirin container(325 gm) and saw no problem trying aspirin for 1 week at 4 tablets/day taken with food always... that course was "safe" according to the label and web information...
  - Went into MAJOR recovery within 72 hrs... and stayed there (a fragile recovery -- had minor signs of a slow decline) until I my MD add antibiotics
  - With antibiotics, and the recovery started to improve more (slowly, but constantly!)

Convinced me of the blood-thinning theory.... and then started to research natural and dietary/supplement blood thinner -- and behold:

- the list was full of the items that reported improvement for SOME CFSers.... (Vitamin E, Oil of Evening Primrose, etc....) ....
- I was sold that my diet needs to be rich in blood thinning foods (and omit blood thickening!)....

Since then have read that there are 3 different blood thickening mechanism..... and most of the dietary stuff is effective against one only.... the Hemex's recommended COMMON drugs (they do NOT produce the drugs!) does the other mechanism....

Because of the improvement, MD decided not to do Hemex but did approved the continued use of aspirin. The "die-off" effect from the antibiotics killing mycoplasma (what is the probable source of the thickening) was very mild -- I suspect the blood thinning is responsible for it.

If I should go into relapse, then the Hemex ISAC panel is #1 item

for the MD, followed by the prescription blood thinner. It produces dramatic and quick results in all except the most severe 5% (which Dr.Berg is concentrating his research on)...

I've been advertising my success because a few CFSers will be as fortunate as me (generally the new victims - but not always), and if there is any improvement.... then I hope this will convince them to seriously look at the Hemex test and protocol.... (there is resistance reported from some MD's over the use of 'industrial strength' blood thinner for CFS...)

Not only do CFSer have to fight this illness, many have to fight their MDs, their HMO (who will pay for warfarin tablets but not heparin injections), and a history of ineffectual treatments...

I hope this helps and explain things...

Niacin

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International CFS Information

Updated on: 06/13/2000

This term includes the following:

- Nicotinic acid
- Nicotinamide (non-flush Niacin):
  - niacinamide has **no vasodilatation activity** in 99 percent of people who take it.[*]
- Niaspan [new version:Warnings: *,*]

Beneficial effects on low-density lipoprotein cholesterol; high-density lipoprotein cholesterol; the apolipoproteins B and A-I constituting these fractions; triglyceride; and lipoprotein(a). Together, these benefits lead to a *diminished incidence of coronary artery disease* among niacin users.[*]

- enhancement of zinc absorption[*]
- enhancement of hemoglobin and liver iron [*]
- Excessive dosages may result in niacin-induced hepatitis [*]
- enhances insulin secretion and increases insulin sensitivity [*]
- Treats dizziness and ringing in ears.[*]
- It is also used for Raynaud's syndrome [*]
- Note: Rickettsia secrete vasoconstrictive toxins[*]

NADH is the precursor to Niacin. Niacin decreases plasma fibrinogen (thins thick blood). Repair genetic damage from virii.

Background Literature

- http://www.medsafe.govt.nz/Profs/datasheet/Aponicotinicacidtab.htm
- http://www.grandmed.com/anti-aging/niacin_story.htm
- http://www.thorne.com/altmedrev/fulltext/inositol1-3.html
- http://www.bookman.com.au/vitamins/niacin.html
- http://www.vitaminsplus.com/vplus/vitb3.htm

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Piracetam

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Smart Drugs
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National CFIDS Foundation
International CFS Information

Updated on: 06/21/2000

Piracetam is a non FDA-approved drug that is available over the counter in Europe. It is available from a few specialized pharmacies in the US with a MD's prescription. For Internet ordering

NOTE: Bromelain and Piracetam appear to enhance each other, for CFIDS it is suggested that both be taken concurrently. Current *suggestion:*

- Bromelain: 2400 GDU/Day
- Piracetam: 1600+ mg/Day

Benefits:

- potent inhibitor of plasma thromboxane B2 [*]
- anti-platelet effect [*]
- improves brain function in low oxygen situations
- half life: 4.5 hrs [*]

Stoke

Piracetam is used for acute ischaemic stroke in Europe (very common to CFIDSers - usually not diagnosis due to brain fog) see
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=10338105&dopt=Abstract (Poland)
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=9591298&dopt=Abstract (France)
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=9412612&dopt=Abstract (Belgium)
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=9316679&dopt=Abstract (England)

Drug Fact Sheets

- http://www.home.intekom.com/pharm/ucb/nootropl.html
- http://www.home.intekom.com/pharm/ucb/notrop_t.html
- http://www.edoc.co.za/medilink/actives/824.html

Summaries:

- http://www.ds-health.com/piracet.htm
- http://erowid.org/smarts/piracetam/piracetam_faq.shtml

Serrapeptase

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International CFS Information

Updated on: 02/09/2000

Serrapeptase is an enzyme derived from silk worms. It is marketed in Asia under the trade name Danzen and in Europe as Anaflazyme. It is similar to Bromelain (antibiotic potenator, anticoagulant) except there is far less research on it. A search on the National Medical Library for "Serrapeptase" is recommended (29 or more articles). Some interesting ones are:

- A case of pneumonitis due to serrapeptase [*]
- Studies on the distributions of antibiotics in the oral tissues: Experimental staphylococcal infection in rats, and effect of serratiopeptidase on the distributions of antibiotics [*]

Sources: http://www.smart-drugs.com/oly-order.htm

Other Reference URLs

- http://www.internetwks.com/saynotodrugs/cardio.html

Wobenzym N

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Updated on: 02/09/2000

A commercial combination of enzymes designed for athletes. Other combo's are: Phlogenzym

Ingredients

From fact table at: http://www.mucos.com/uk/prep/wobenzym_n_dragees_eng.htm

- 100 mg of pancreatin (equivalent to 300 proteases Ph. Eur. units)
  - derived from pigs, for digestion
- 1 mg of chymotrypsin (equivalent to 5 µkat)
  - A pancreas extract, affects digestion
- 45 mg of bromelain (equivalent to 225 FIP units)
- 60 mg of papain (equivalent to 164 FIP units)
  - papaya fruit extract, affects digestion
- 50 mg of rutoside x 3 H_2O
  - A Bioflavonoids

Studies

- http://www.gth-online.de/thrombo/Abstract/p245.htm
- http://www.mucos.cz/eng/far_mech/peowotb.htm
- http://www.mucos.com/uk/wiss/sichtrhe.htm

Available From...

Cheapest source so far:
http://www.mistergreengenes.com/wobenzym2.html

Size	Sale
100	$21.95
200	$28.95
800	$109.95
2-800	$199.90

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Rosavin

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Adrenal Glands

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International CFS Information

Updated on: 07/08/2000

- Also known as Rhodiola Rosea, Rose root, Golden Root and Arctic Root.
- 29 (almost all Russian) articles on Nat.Med.Lib
- An Adaptogenic plant, Others are:
  - Acanthaceae , Trichopus zeylanicus, Withanina somnifera, Cicer arietinum, Codonopis pilosula, Panax ginseng, Gentianacease, Leuzea carthamoides, Rhaponticum carthamoides, Ganoderma lucidum, Eleutherococcus senticosus, Ocimum sanctum)
- enhance transport of serotonin precursor
- immunostimulation.[*]
- anti-arrhythmia
- "Adaptation is characterized by a decrease or total prevention of hormonal changes peculiar to stress."[*]
- helps functioning in hypoxia (low oxygen)

Sources:

In capsules;

- http://www.ameriden.com or
- http://www.hpfonline.com/prhoros.htm or
- http://www.iherb.com/iherb/rhodiola.html (Warning: with St. John's Wort)or
- http://mineralconnection.com/rosavin.htm or
- http://www.naturalhealthconsult.com/_vti_bin/shtml.exe/Monographs/rosavin.html
- http://www.naturalhealthconsult.com/_vti_bin/shtml.exe/Monographs/rosavin.html/map3

Bulk:

- http://www.pharmlineinc.com/
- http://www.beyond-a-century.com/

Adaptogen Bibliography

- http://www.juststrategies.com/prod_horses_eg5.html
- http://www.healthy.net/hwlibraryarticles/hobbs/zadapt5.htm

Bibliography:

- Carl Germano, Zakir Ramazanov, Arctic Root (Rhodiola Rosea), Kensington books, 1999

- http://www.oracleweb.net/pagansonline/sedumrhodiola.htm
- http://www.nutrimart.com/Bulk/Description/rhodiola_rosea.htm

Coagulation Factors

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International CFS Information

Updated on:
06/21/2000

Adrenal Glands

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National CFIDS Foundation

International CFS Information

Updated on: 05/27/2000

CFIDS is very stress sensitive, and the physical connection appears to be the Adrenal Glands, or in a general sense: hypothalamic-pituitary-adrenal axis dysfunction (HPA). Dr. See states this is a secondary phenomenon and not a cause.

Treatment:

- Replacement (Darryl See, M.D.,[1997]): Supplying externally
  a. DHEA, 10-200 mg daily
  b. Testosterone - Androderm patches 2.5 - 7.5 mg daily
  c. Florinef - 0.1 mg tiw to qd for orthostasis
  d. Prednisone - only for blunted response to ACTH
- Support (see *): Supplying components
  a. Pantothenic acid (B-vitamin)
  b. Vitamin B complex, Vitamin C plus bioflavonoids
  c. L-Tyrosine
- Adaptogenic: moderate and balance the body's response (hence less use). For more information see http://herb.com/adapt-2.html : Reducing needs
  a. Chinese Ginseng (*Panax ginseng*)
  b. Siberian Ginseng (*Eleutherococcus senticosus*)
  c. Rosavin (Rhodelio Rosea)
  d. Indian Ginseng (Ashwagandha root)

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Smart Drugs

Dr. Cecile Jadin's Papers are now available in full Click Here

Aniracetam

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31893

National CFIDS Foundation

International CFS Information

Updated on: 03/14/2000

The drugs are ***not*** a cure, they only grant symptom relief ("buys some brain time").
Do not view any recovery using these drugs as a cure or remission of CFIDS.

"Smart Drugs" is a class of compounds developed to improve brain function. Since most CFIDSers suffer from significant loss of brain function ("brain fog"), they are a logical symptomatic supplement. Many of these are also anticoagulants, and thus have significant benefits for many. [General introduction, Summary]

The FDA has not approved the use of most of these drugs *["Smart Drugs" and hippies's "mind expanding drugs" are associated in some bureaucrat's minds]*. They are not available from most US/Canadian/British pharmacies even with a prescription (they may be available *with prescriptions* from some specialized pharmacies "compounding pharmacies"). On the other hand, these drugs are available "over-the-counter" in much of Europe and may be ordered on the internet.

"A little known FDA ruling now allows the importation of a three-month personal supply of drugs as long as they are *regarded as safe in other countries*. Ordering safe but unapproved drugs is now legal under the new FDA pilot guidelines, Chapter 971."[*]

							same base molecule				
Drug	Pyritinol, Pyrithioxine [Encephabol]	D	H	Centrophenoxine, [Lucidril]	DMAE Dimethylaminoethanol	Vinpocetine [Cavinton, Ceractin]	Aniracetam	Oxiracetam	Pramiracetam [NeuPramir]	Pyroglutamic acid, Oxoproline	Piracetam [Nootropil®]
Drug Fact Sheet	[*],[*]	[*][*]	[*], [*], [*], [*]							[*]	[*],[*]
Sleep Disorders	Yes	Yes[*]		No, Can cause Insomnia		Yes [*]	Yes [*]				No, can cause insomnia
Emotional liability / depression	Yes	antidepressant [*]		No, Can cause excitability, increase depression			Yes [*]				
Memory	Yes			Yes [*]	Yes	Yes [*]	Yes[*]	Yes[*]		Yes	Yes
Concentration	Yes							Yes[*]			
Reduces Hypoxia effects	Yes [*]		Yes			Yes [*]	Yes [*]	Yes[*]			
Antioxidant	Yes [*]			Yes [*]		Yes [*]		No [*]			No [*]
Eye Problems						Yes [*]					
Libido		Yes									Yes
Immune Enhancer	Yes [*]	Yes [*]									
Comments	Pyritinol molecule is structurally similar to Vitamin B6 (Pyridoxine)		Anticoagulant			anti-platelet aggregation, Available in US[*] inhibition of aggregation of thrombocytes[*]			More effective form of Piracetam		anticoagulant
Warnings	[A]	[E]	[C]				[B]				

[*] Click to see source document.

General Note: Usually take them early in the day and not in the evening. Many have a short term stimulant effect.

[A] Significant side effects are reported with rheumatoid arthritis [which may be mycoplasma based - like some CFIDS cases] [*]

[B] Not approved in any country [*]

[C] OVERDOSE DANGER: The symptoms of overdosage with Hydergine are nasal stuffiness, flushing of the face, headache, nausea and vomiting, tremulousness, spasticity, hypotension, circulatory collapse and coma. [*]

[D] To date Selegiline has been medically approved by regulatory agencies for use only in treatment of Parkinson's disease [*], approved by the Food and Drug Administration for the control of the clinical signs associated with canine Cognitive Dysfunction Syndrome[*]

[E] risk of adverse effects when it is used in combination with selective serotonin reuptake inhibitors and tricyclic antidepressants [*], many other risks [*]

Aniracetam

Updated on:
09/01/2000

* Improves sleep ability (counters insomnia) [*]

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International CFS Information

Updated on:
12/03/1999

Off Shore Pharmacies

The following site sell supplements that are not approved by the FDA and not available for sale in the US. Buyer Beware on the use of any non-FDA approved supplements -- never use them if you are on any other medication or pregnant without good research. Always consult with your MD.

http://www.nubrain-store.com/

Libertarian Site:
http://www.libsol.net/frames.htm

International Antiaging Systems
http://www.smart-drugs.net/price-ord.html

Quality Health
http://www.qhi.co.uk/index-pn.htm

Antiaging-Systems
http://www.antiaging-systems.com

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October, 1999

I've been asked to write up my recovery from CFS (my second), and after lots of thoughts of what to write, I came up with the following...

First, I was very fortunate with many of the early events centering around the onset of CFS. I knew what was the "insult" that allowed CFS to become established - ***stress***, I had a supporting wife and knowledgeable MD, I had excellent short term disability insurance, and I knew what was going on.

The early days

I knew that I was about to get sick 30 days before onset. I developed a dry-cough that had preceded my prior PVCF experience from 26 years earlier. I knew that I needed to de-stress, slow down etc... but failed to receive support from my manager. After reading an email from the boss denying me permission to look for another position in the company, I took sick that evening. The boss changed his mind the next morning -- unfortunately, CFS onset also arrived. I fell asleep at a stop light at 11am in the morning, headaches, nausea, dizziness plus night sweats, chills, etc. I was running at 1-2% of normal.

The first steps on recovery

There was a violent change in eating habits... from 10+ cups of coffee to zero cups (just thinking about a cup of coffee made me very sick), no donuts or anything sweets, no fatty food -- all were sudden repulsions... and there was this desire for peanut

butter (without any craving for more when I had some). Unknown to me at the time, this appear to have been the right change of food! I kept to these habits even after the strong repulsion faded...

Because of my earlier experience with PVCF, I knew that I must immediately and *totally* de-stress. I used up all of my banked sick time (dropping off an important project that I was both the development team manager and main technical resource -- probably blowing away 4-6 man-years of work). It was not enough to undo CFS. Progressed on to short-term disability (hoping to recover enough in 6 months) -- believing it to be like my prior PVCF, just more severe. I maintained the belief that I would recover.

The search for knowledge

At 3 months, the family MD came to the diagnosis of CFS. Fortunately she has seen CFS patients for 17+ years and accepts it as an actual disease! In researching my symptoms, I also came to the CFS conclusion at the same time.

My research found that the odds of recovery decreased exponentially for each year that you are sick... so recovery is possible, but ***it must happen NOW***... every hour, day or week of delay makes recovery less likely.

The only treatment that had a 'government stamp' of approval was the use of NADH (verified by FDA). Ordered it and started at 10 mg, the same level as in the study. It worked: fatigue was less deep and had slightly more energy.

When I found myself bordering on depression, I re-read the study and found that dosage was still an unknown. Since I was 240 lbs, I suspected that a larger dosage would be appropriate and increased it to 15 mg. To my great relief, the depression disappeared within 48 hours.

When NADH no longer had significant improvement, I returned to the web and found that B12 was the most cited beneficial supplement. I started with 1000 mg/day of B12 tablets and 48 hours later there was significant improvement with some symptoms.

Two experimental protocols with good odds!

In reading everything I could on the web, I finally came across Prof. Nicolson and Dr. Berg's works/papers/institutes. Dr.

Berg's work impressed me greatly and because there appear to be a genetic history of this illness in my family, I was going to press my MD to have the Hemex's ISAC Panel done at my next visit.

Fortunately or unfortunately, I had ongoing hassles with my LTD company and had an IME scheduled... which caused a relapse. My reading found that for some purposes, one of the Hemex drugs and aspirin were equivalent, and that aspirin flushes out of your system in 6 hours. So because of the relapse, and impatience waiting for the next MD appointment, I started taking one aspirin every six hours for 1 week (*which was ok to do according to the label on the aspirin box*). I was hoping that it would stop the decline that the LTD hassles had started... to my surprise, within 48 hours, I was bouncing off the walls... I was at 80% of normal compared to my prior *best* of 30% of normal... blood thinning worked! I know now that I was extremely lucky -- platelets were very significant in my case at that point of time.

Improved Understanding and more treatment

Now that I knew that blood thinning worked, I naturally went out to find other dietary blood thinners and add them to my diet. This included things to make blood vessels larger (thus healthy heart diet, concord grape juice)... "the more blood that flows and the faster that it flows, the better I will become..."

Reading about the low red blood cell count, I researched what was needed for red-blood cells and discovered that peanut butter had everything EXCEPT B12. Now I knew why I wanted peanut butter and why B12 works.

At my next MD appointment we discussed Dr. Berg's work and my miracle recovery using aspirin - and also Prof. Nicolson work on mycoplasma. I left with a prescription for 30 days of doxycycline (300 mg/day) because it was the least risk and simplest for the MD -- besides, I had recovered so much already!

Die-off and more ammo

I had a very easy time with die-off from the antibiotics. Only two weeks of it lasting all day, and then the length became shorter and shorter. 8 weeks later, I would only get 'die-off' if I became physically active 30 minutes after taking antibiotics (nice way to remind yourself that the antibiotics are still

needed!).

I am still on antibiotics (planning to do the full 12 months as recommended by Prof. Nicolson)

Additional ammo that I have added (which did produced a change) were:

- Hale's breathing
- ... rest in progress,,,

A Similar Prior Illness

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International CFS Information

Updated on: 01/07/2000

In 1973, I may have had my first round with IDEF/CFIDS although it was not named as such, instead it was described as a "persistent viral infection, aided by being run down". The ***deep*** exhaustion characteristic of severe cases did not occur but many other characteristic were present:

- Felt close to vomiting, sweats, dizziness - ascribed to the virus
- Bowel movements (2 - 3x more movements/day than normal) - ascribed to the prescriptions given to fight secondary infections
- Cognitive loss - dropped from doing double honors to passing only 60% of normal courses the following year. Marginal improvement in subsequent year [did not return to same academic performance for almost 4 years]
  - Ability to do math (my strength) greatly reduced
  - Ability to do Arts courses (History of Western Religion was a minor) was only slightly reduced
- mild photophobia, some weight loss followed by weight gain, vision: floaters (round black dark areas)

Onset Situation

Environment: Undergraduate Student doing honors (originally: Mathematics, Physics, Chemistry), working 36 hours per week at a part time job, active in student activities (Newspaper, Alma Society Representative, various clubs - including being on national boards), father became disabled with a back problem and had great trouble dealing with the restrictions.

Viral Infection: Came down with *flu* towards end of the flu season. Saw physician when tiredness did not leave for eight weeks after the flu.

Prescription / Treatment: Rest, reduction of stress. Part-time job involved light physical activity (Liquor Store Clerk / Cashier). Tiredness ascribed to being over-stressed and extended de-stressing required, lightened my academic load greatly (failed some courses too!).

Also, was put on a high protein, low carbohydrate diet for weight loss. This may have been what helped my recovery greatly (acidifying my blood...) see hypochlorhydia

Recovery: Approximately 4-5 years before energy levels and mental performance returned to prior level or equivalent.

Sudden Onset

Blood Volume Diet

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International CFS Information

Updated on:
10/10/1999

The second occurrence of IDEF / CFIDS occurred 26 years after the first. In the years between CFIDS was in complete remission (at age 35, running 10 km in 100 degree heat with no difficulty, working for Microsoft and putting in 18 hrs days x 7 days/week for months on end, getting exceeding reviews....).

The environment of the second occurrence had strong similarity to the first.

Environment: Working for the '*evil empire' (Microsoft) pulling 16 hrs/days by 7 days/week*

After the onset, I found the following occurred:

- strong repulsion of coffee (from 8+cups per day to 0 cups - almost overnight!)
- strong urge to drink lots of orange juice
- loss of appetite
- very frequent urination ("pee on the hour")
- strong distaste for meat and other fats heavy food (i.e. cheese, dairy)

Analysis

In keeping with the reduced blood volume theory[1], the repulsion of coffee is due to coffee's tendency to absorb water (further reducing blood volume). Other food that absorb water (and thus reduces blood volume - these should be avoided) are:

- coffee
- alcohol

The lost of appetite is caused by the body not wishing to share its reduced blood supply with the stomach. According to the SAS Survival Manual, digestion of fats (even if suspended in water) requires more water and should be avoided if there is a shortage of water...

Vitamin Tablets

One of the additional changes that I noticed was 'hesitancy' in taking vitamin tablets (I had been taking them prior to onset), yet I had a strong urge for vitamin C in fresh orange juice (2+ quarts per day!). This appears to make sense now! The tablets required water / blood to dissolve and further saturates the overloaded/super-saturated blood. Vitamin C in orange juice is easily passed thru in the water that it contains.

A Relapse

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International CFS Information

Updated on: 10/10/1999

IDEF / CFS / CFIDS is characterized by relapses if you go outside of a small envelope of acceptable activities. The following describes one such relapse (Onset + 7 months) and the recovery I had:

6-Oct	Quite a day: discovered my LTD claim is being processed by the insurance company that has the worst reputation (on the web) for CFS claims. Later that same day, I found out that the LTD Policy was other than advertised in my employer's literature (the policy is for 24 months and not the published 60 months). Suspected that tomorrow will be a bad day since CFS is stress-sensitive....		
7-Oct	RELAPSE:	sore wrists	very loud tinneratis while going to sleep last night
		bad headache	woke up at 4am and pee-on-the-hour afterwards
		dizziness	both wrists aching
		sore throat	unable to read more than a page of text with focus
		sweating after short (< 1/2 normal) walk, exhausted	
		reduction of appetite	stiff sore muscles
		some light sensitivity	eyes sore
	REMEDY:	Bed rest (in bed except for 2 short walks) Not hungry and intentionally ate little	

		NADH (15mg+walk) & B12 as usual (250mg with each meal)
		Lots (2 quarts/day) of Orange Juice (Not from concentrate)
8-Oct	Recovering	sweating after short (~ 3/4 normal) walk, tired
		Wife reports that I was clammy all night
		light headache
		slept until 6am before waking to go pee
		mildly sore throat
		loud tinneratis while going to sleep last night
9-Oct	Recovering	Slight sweat after normal walk, slightly tired
		Slept until 8am before going pee
		tinneratis while going to sleep last night
10-Oct	Recovering	Slept until 6am before going pee, easy back to sleep afterwards
		Fine after normal walk
		noticed some tinneratis in the morning, rest of relapse symptoms are very mild. Prior energy level is coming back (likely ~2-3 more days)

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International CFS Information

Updated on:
03/04/2000

November 5th, 1999

- Day 4 of antibiotics (300mg/day of doxcycline)
- Day 16 of anticoagulant (aspirin 325 mg : 4 times a day)

This is my 2nd CFS session (26 years between) - this is far more severe than the first, but recovery is proceeding far faster because of the two treatment protocols. Stress was the cofactor in both cases, with a warning symptom (distinctive dry cough - matches the typical symptom for mycoplasma pneumonia (Walking Pneumonia) -- Assuming a full recovery.... the next time that I hear that cough it is 'bye-bye source of stress' -- be it quitting on the spot and taking a vacation (after a month of preventative aspirin and antibiotics) or taking all of my vacation and sick leave on the spot, or disappearing to the Falkland Islands...

I learn that lesson on day 6 of the aspirin when I bounced into a 'slightly less good feeling' (I over did it!), and now have added a few quiet prone sessions into each day -- regardless of need....

, I've been definitely experiencing the "die-off" or Herxheimer effect... (This is when the bugs bodies pile up faster than the body can remove them.....)

http://immunehealthsystems.com/herxheimer.htm

http://garynull.com/Documents/Arthritis/Herxheimer_Effect.htm

It did not start until 48 hrs after starting the antibiotics (which surprised me - usually I get it sooner...). Not severe at all (like a mild bad day -- remember the ASA put me into major remission first). Probably running close to 40 oz of orange juice and 24 oz of grape juice to help flush stuff out...

I'm doing 2 dosages @ 150mg apart 12 hrs apart... the "die-off" effect usually starts about 1 hr after the pills and end about 8 hrs later.... [thus I do morning walk, etc just before I take the pills) -- if you are not expecting or familiar with it, it could get a little worrisome -- the first night that it started, I was real clammy and warm in bed -- wife was a little concerned until I reminded her that this is normal for antibiotics (I think I've never taken any antibiotics during our 12 years of our marriage -- usually avoid prescriptions like the plague...)

What is also unusual is that usually "die-off" effect lasted 48 hours when I've taken antibiotics before.... well, there must be masses of mycoplasma... it is still ongoing...

My detail schedule of vitamins etc is below -- in case any one is interested... (I would never have dreamt that I would become a pill-popper!)

Life is feeling very normal -- apart from sore muscles (exercise tired) that
feel far looser than they have for months (probably detoxing), running nose (first one in 8 months). Marked improvement with nightly urination starting on day 2 of doxcycline. Active from 7am to 10 pm - no fatigue crashes. Feeling better day by day....

Also got my LTD approval (on first go - the experience reminded me of a
divorce; fortunately I learn a lot about strategy and law from my ex-wife:
happy married now for 12+ year...)

Before Morning Walk

- 15 mg NADH
- 8 oz Orange juice
- 1000 ui Vit E

With Breakfast

- 325 mg ASA
- 8 oz Grape juice
- 500 mcg B12
- B-100, 1 tablet
- CoQ10, 100 mg
- Magnesium Malate, 1 tablet
- 150 mg of doxcycline (Antibiotic for Mycoplasma)

Lunch

- Peanut butter Sandwich
- 8 oz Orange juice
- 325 mg ASA
- 500 mcg B12
- 1000 ui Vit E
- 200 mcg Chromium
- 30 mg Zinc
- 200 mcg Selenium

Supper

- 325 mg ASA
- 8 oz Grape juice
- 500 mcg B12
- 1000 ui Vit E
- CoQ10 - 100 mg
- Magnesium Malate, 1 tablet
- 150 mg of doxycycline (Antibiotic for Micoplasma)

Bedtime

- 325 mg ASA
- 8 oz Grape juice
- 1000 ui Vit E

Dec 1999

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National CFIDS Foundation

International CFS Information

Updated on: 03/04/2000

December 4th, 1999

My wife appears to have gradual onset CFS (started after a C-section, some 10 years ago) which has been progressively limiting her life. She appears to have developed "leaky-gut" with the classic problems of food sensitivity and multiple chemical sensitivities. On Monday, she is to the MD -- and we expect a CFS diagnosis -- she is both happy and frightened about this. "It is not all in her mind/will" -- which is what she has been assuming/told by too many people. She is happy because of this, but also frightened because of what this means. She cannot take aspirin and has had only limited results from other blood thinning..., we are starting on other protocols --- and I know that it will be an uphill fight (she does not have the strict discipline that I have and have been very conditioned to "will/ignore" things and not listen to her body).

We are hopeful, (and in one real sense thankful that I got CFS -- because it may change around her life direction/resignation)...

December 16th, 1999

Well folks, Laurie is technically on antibiotics for a stomach ulcer -- but with our cooperative rural MD, we got to pick from any of the FDA approved sets of antibiotics... and opted for the one that had

a mycoplasma effective antibiotics (two bacterias with one pill).

We changed our diet to the one that I created by merging antibiotic, acute mountain sickness and Hale's breathing diet - plus CFS adversion foods.. before she started...

By day 3, she was in a real painful herx effect (a lot of pain that had been ascribed to structural problems may have been actually FM pain)...dizziness, pain, stiffness ... she had to crawl to the bathroom - incapable of walking. Last night, IMUPLUS arrived in the mail and we both took our first package. She had a much more restful night - actually slept for 5 hours. Still pain but duller. She has not taken any painkillers... how that she understands the relationship between them and MCS -- she is suffering the pain.

This morning, she took her second package... and just walked in (3 hrs later).... 'she feels like she is bouncing off the walls' -- I know EXACTLY how that feels -- this was what happened when I had the miracle aspirin recovery.... Oxygen is getting thru the system in huge quantities (compared to before) ... I've cautioned her about needing to rest and fortify the effect (instead of starting a massive housecleaning and catch up). She had been doing the Hale's breathing thing faithfully (with improvement of her CP) and I believe the two are acting like catalysts with each other.

NOTE: This appears to be significantly reducing the Herx effect!!!!

For myself, I've noticed no significant effect - but I was not expecting any since the remaining symptoms are co-lateral. I' taking IMUPLUS as a preventative 'tonic' and an additional 'deep cleaner' to assist the antibiotics.

More updates as things progress...

Ken

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International CFS Information

Updated on:
03/04/2000

January 15th, 2000

This is an update to my earlier report,

History: On antibiotics (week 11 approximately), Herxing stopped at week 3

Started adding Bromelain on January 11th - four days ago.. Same day reaction: Arms and Legs became very still and sore -- herxing in the joints and muscles - a pain

Accidentally observed that my memory had improved -- better recall than in the 6 months prior to CFS onset! Had started Piracetam at the end of December and found that it had no apparent effect. The mixture or Piracetam and Bromelain has a dramatic effect.

- Today's status:
- Arms and legs continue to be sore -- less so, other minor herxing effects

- Mental alertness and mental energy to do things have improved (in fact, have started to overrun my physical capacity)
  - [brain fog disappeared back in August - from aspirin, this is an additional upgrade]

Info on bromelain: http://www.folkarts.com/idef/bromelain.htm (including a
link to a study showing that it improves the >>>penetration<<< of the family
of antibiotics used for CFS by 50-100%)

Laurie started antibiotics at the beginning of December for a stomach ulcer. Our MD allowed us to pick which FDA protocol, we picked one that also hits mycoplasma. Laurie had two heavy weeks of herxheimer (it stopped when the 2 week supply for the stomach ulcer ended). It sure looks like she has a mycoplasma infection and CFIDS -- so she will likely be starting antibiotics in a week or two. I suspect that I will be chief cook and bottle washer for a few weeks at least.

Feb 2000

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Supplement Feb

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National CFIDS Foundation

International CFS Information

Updated on: 03/04/2000

February 10, 2000

Ken:

Sudden Onset: March 1999,

Treatment: On month 4 of antibiotics with non-prescription anticoagulants (Bromelain & Piracetam), plus 2-4 packets of Imuplus (just 8-12 months to go)

Current state: I am up to (prior) "normal" performance for ~ 5 continuous hours/day doing 'guru-grade programming' in a normal work environment (I'm/was a very high performer at Microsoft), after 2-3 days in a row, the duration drops to ~ 4 hrs until I take a day off (or a weekend). Memory has returned with great clarity, ability to read and absorbed quickly has returned. Doing a crisp 2 mile walk rising 600'/mile daily. Sleeping soundly - typically 8-9 hrs. Have survived on 6 hrs, but I feel it the next day. Wake up fresh in the morning at 7am -- but often ready to crawl to bed by 8pm...

Herxing: Amount is controlled by bromelain dosage, mainly aches, stiffness and pain in arms and legs -- I suspect this is where the reserves of the infection are located. Very low grade fever often. A zero-bromelain day is a very high energy day

with no 'morning after' effect.

Laurie:

Gradual Onset: Starting ~ April 1988

Treatment: On month 2 of antibiotics with non-prescription anticoagulants (Bromelain & Piracetam), plus 4-6 packets of Imuplus

Current state: Appears to becoming out of the worst of herxing: Has started to drive again but lots of herxing induced symptoms remain (but slowly reducing). Herxing caused her to become far worst than she ever was before - including urinating every hour on the hour for a week (I did the same for many months before the recovery started to progress significantly), great increase in NMH (dizziness), 16-18 hrs of sleep/day (now down to ~ 11 hrs /day), very easily overload by stimula (drives the kids crazy - because I'm all over them when Laurie gets overloaded), she is starting to walk 1 mile/day (taking NADH just before starting has helped her). On the one day that she cut Bromelain to zero, she was climbing the walls with energy -

She is slowly finding her level with bromelain to keep enough herxing to keep her on treatment but not too much so she will herx her way thru the entire day confined to bed. Boy, does she look younger in her face :-) , very positive attitude to the whole process. There is a lot of laughter around the house ...

This week: very marked increase in mental abilities, less sleep, more activities..

16 year old daughter

- some signs (multiple food sensitivities, tiredness, emotional flare-ups/ depression - including seeing psychiatrists, rashes and symptoms that are unusual for our MD), but no diagnosis.

Treatment: non-prescription anticoagulants (Bromelain & Piracetam), 1 packet of Imuplus

Current State: She LOVES the anticoagulants, she calls them

her 'brain food', does not like the Imuplus - it makes an outstanding difference in her ability to concentrate and be alert at school, emotional state is very positive, no signs of any depression since she started taking both.

NOTE: Our MD is completely informed of what everyone is taking, and advises us of any possible risks...she knows our logic and model - and sees the results...

Mar 2000

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Supplements March

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National CFIDS Foundation

International CFS Information

Updated on: 03/04/2000

Ken:

Sudden Onset: March 1999,

Treatment: On month 5 of antibiotics with non-prescription anticoagulants (Bromelain & Piracetam), plus 1-2 packets of Imuplus. After research by Laurie (her brain fog has cleared very nicely) Olive Leaf Extract (300 mg of Oleupeinper day) and L. Plantarum (produces B vitamins and is not killed by antibiotics) have been added.

Current state: Received a medical release to work 4hrs/day as of March 1st (with Herxheimer Effect being explicitely cited as preventing regular full time work for many months - especially when antibiotics change). Doing a change of employer to one .5 miles away instead of 1+ hr away. Using an organic vapor mask full time at work for two reasons: it acts like a rebreather (keeping me more alert) and also reduces the possibility of my fellow employees contracting the illness from me. Bosses (other ex-Microsoftees) are fine with it, and with my 'work as I can' restrictions [having a glorious CV http://www.folkarts.com/kenl/ helps to good accomondation]. Wake up fresh in the morning at 6am --

but ready to crawl to bed by 9pm... 2hrs more of the day is available.

Herxing: Amount is controlled by bromelain dosage, mainly aches, stiffness and pain in arms and legs -- I suspect this is where the reserves of the infection are located. Very low grade fever often. A zero-bromelain day is a very high energy day with no 'morning after' effect. Had two weeks of heavy herxing when I started the Olive Leaf Extract. Once it cleared, found that my active period of the day was stronger.

Laurie:

Gradual Onset: Starting ~ April 1988

Treatment: On month 3 of antibiotics with non-prescription anticoagulants (Bromelain & Piracetam), plus 4-6 packets of Imuplus. Also Olive Leaf Extract and L.Plantarum.

Current state: Coming more out of the worst of herxing: Has started to drive again but a 30 minute round trip into town does wipe her out for the rest of the day (so it's a once a week event at most). Herxing induced symptoms remain (but slowly reducing). She is approaching pre-antibiotics normal for physical activities. Mentally -- she has not been this good for 3-5 years. She has taken on editing a book, and may have to also do some major revisions of the text (almost co-authoring) - she is very excited about it and spending several hours per day doing it. Sleep/day down to ~ 10 hrs /day, less easily overload by stimuli than last month. She is walking 1-2 mile/day (taking NADH just before starting has helped her). Some structure problems (horse riding accident while schooling in Australia) are re-surfacing -- combination of a lot of bed rest for the last month and likely Bromelain dissolving fibrin around old injuries - so she is having structural pain :-(.

The first 2 weeks after starting Olive Leaf Extract had her going to the washroom almost on the hour all through the night (reminded me of my early experiences) and then it eased so she is sleeping through the night again.

16 year old daughter

- some signs (multiple food sensitivities, tiredness, emotional flare-ups/depression - including seeing psychiatrists, rashes and symptoms that are unusual for our MD), but no diagnosis.

Treatment: non-prescription anticoagulants (Bromelain & Piracetam), 1 packet of Imuplus, Kelp and multivitamins added this month. Also starting Olive Leaf Extract.

Current State: She was real ticked when we ran out of Piracetam for 4 days (shipment from Switzerland was delayed) because she found her concentration at school dropped quickly. She also mentions that her nails are now healthier than she can ever remember - Laurie and I attribute that to Whey.

She is NOW *actively interested* in taking vitamins etc to keep healthy -- we are real pleased.

10 year old daughter

- only one possible sign - "isolation" mental state that almost have us dragging her to a psychiatrist. Often states "I'm depressed" etc...

Treatment: a glass of chocolate ion-exchange whey (laced with 1200 GDU of bromelain) (Prozone is the specific brand). Refuses to take supplements etc. A compromise.

Current State: She is a lot easier to live with, general mood has improved considerably.

As an FYI: Laurie found depression arising when we ran out of Piracetam -- which quickly disappear when it arrived and we were able to resume our 1600 mg/day.

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

Apr 2000

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Supplements_April

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National CFIDS Foundation

International CFS Information

Updated on: 05/09/2000

Flash:

- We changed from ImuPlus to ImmunoPro this month (awesome).
- We started using EastPark Olive Leaf Extract (38%)
- Doing daily hot tub sessions in the evening.

Ken:

Sudden Onset: March 1999,

Treatment: On month 6 of antibiotics with non-prescription anticoagulants (Bromelain & Piracetam). Whey: 20 mg of Immunpro. Large dosages of Olive Leaf Extracts. The new whey had a dramatic effect on endurance during the day, higher energy levels and longer durations. The initial effect is very close to the effect I experienced originally from aspirin: "Hyper, running up the walls".

Herxheimer reaction decreased so increased bromelain and olive leaf extract - they got going again.

Current state: [Pre-Immunopro] Finding that there was a slow constant increase of ***active*** day (~ 30 minutes/month).

Dramatic jump from Immunpro *above this* - but I want to go a few more weeks before giving an estimate. Find myself waking up with the sun automatically now.

Herxing: Still here, but I've increased dosage of bromelain and olive leaf extract as it ebbed. Herxing is mainly after 4pm

- 6AM 10mg of Immunpro in water
- 7AM Antibiotic, piracetam(800mg)
- 2PM 10mg of Immunpro in water
- 3PM Olive Leaf Extract (Oleuropein: 380 mg), piracetam(800mg)
- 6PM Antibiotic
- 10 PM Olive Leaf Extract (570 mg)

Current State: I'm 47 and for 8+ hr/day, I seem to be able to match many 30 yrs olds. Duration with moderate-heavy labor is about 90 minutes now (hauling sod, 2 cu ft bags of top soil) before I need a rest. No need for afternoon naps unless doing moderate-heavy labor. Down side: In a general stress situation (like city traffic), my capacity drops to ~ 4 hrs. *"Green acres that the place to be, farm living that's the life for me, ..."*

Laurie:

Gradual Onset: Starting ~ April 1988

Treatment: On month 4 of antibiotics with non-prescription anticoagulants (Bromelain & Piracetam), Whey: 20 mg of Immunpro. Olive Leaf Extract and L.Plantarum.

Current state: Taking 950 mg of Oleuropein (Olive Leaf Extract's active ingrediant) at bed time -- she herx's herself to sleep and sleep through the night nicely. Laurie is also bouncing with energy from Immunopro -- we have three shocked daughters around the house due to the change of her energy level. She is now able to drive to town every day (Dad's taxi service is semi-retired). Increase of tinnitus (ringing in the ear) - not concerned (herx effect). Her structural problems are now under control

Need about two hours less sleep a night, and not taking naps anymore – although resting occasionally.

- with new whey, my mood is significantly lighter for the first hour or so after drinking it.
- "Relapsed for a couple of weeks, due to family politics with an in-law. Couldn't tolerate 101 degrees of hot tub, where I could before that. Improved, but still not up to where I was before the event."
- One day she pushed herself too far: "I experienced shakes, difficulty with manual dexterity and brain fog. No apparent relapse during the following 48 hours." - the no relapse is very good news!

16 year old daughter

- some signs (multiple food sensitivities, tiredness, emotional flare-ups/depression - including seeing psychiatrists, rashes and symptoms that are unusual for our MD), but no diagnosis.

Treatment: non-prescription anticoagulants (Bromelain & Piracetam), 1 packet of Imuplus, Kelp and multivitamins added this month.

Current State: Her response from ImmunoPro (10mg) was that she crashed at school later that day (fell asleep). We are back to ImuPlus on the school day mornings, and will try getting her over to ImmunoPro on weekends and possibly Olive Leaf Extract too. We are planning to ask our MD to have the ISAC panel on her (because of her unusual allergies and Dave Berg comments about coagulation and allergies).

10 year old daughter

- only one possible sign - "isolation" mental state that almost have us dragging her to a psychiatrist. Often states "I'm depressed" etc...

Treatment: a glass of chocolate ion-exchange whey (laced with 1200 GDU of bromelain) (Prozone is the specific brand). Refuses to take supplements etc. A compromise.

Current State: She is a lot easier to live with, general mood has continued to improve from last month.

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May 2000

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International CFS Information

Updated on: 05/09/2000

Extremely High Stress Month:

- Ugly family politics (including "It's all in your head") resulted in our obtaining a protection order, implementing call blocking and email blocking.
- Both Laurie and I stopped herxing with the stress and energy levels dropped significantly.
- After restraining order issue and counselling to deal with stress, herxing resumed.
- Laurie displayed many stroke symptoms (see Berg's Townhall) from the traumatic stress

Ken:

Sudden Onset: March 1999,

Treatment: Due to stress and stroke risks, increased amount and different types of anticoagulants (Bromelain,Piracetam, Rosavin, Wobenzym N). Whey increased: 30 mg of Immunpro. Large dosages of Olive Leaf Extracts. The new whey had a dramatic effect on endurance during the day, higher energy levels and longer durations until the stress hit. We believe that it has helped us handle the stress better

Rheumatoid Arthritis symptoms started to appear over the last 6 weeks, if I sat for a short while, I would get up like an old man. MD switched me to minocycline which is suppose to be more effective for RA. New type of herxing started immediately (Running nose for 7 days, need 4 hr more sleep, light dizziness/NMH). RA symptoms appear to be lessening.

Current state: Improving again as the stress decreases, waiting for the herxing to lessen from minocycline. I'm needing midday naps again -- the stress did a major number on my system

Weight: dropped 10 lbs since starting Immunopro (had gained weight with antibiotics)

Laurie:

Gradual Onset: Starting ~ April 1988

Treatment: On month 5 of antibiotics with non-prescription anticoagulants (Bromelain & Piracetam), Whey: 20 mg of Immunpro. Olive Leaf Extract and L.Plantarum.

Current state: Taking 950 mg of Oleuropein (Olive Leaf Extract's active ingrediant) at bed time -- she herx's herself to sleep and sleep through the night nicely. With the stress there is some insomnia if she awakes, but then she takes more Olive Leaf Extract and herxs back to sleep.

Laurie energy increased from Immunopro -- the stress reduced the energy slightly, but the stress mainly resulted in major loss of brain function (large dosages of anticoagulants helped). Laurie is handling this stress far better than any stress over the last 12 years. Rosavin and the anticoagulants are getting the credit.

Weight: dropped 11 lbs since starting Immunopro

16 year old daughter

- some signs (multiple food sensitivities, tiredness, emotional flare-ups/depression - including seeing psychiatrists, rashes and symptoms that are unusual for our MD), but no diagnosis.

Treatment: non-prescription anticoagulants (Bromelain & Piracetam), 1 packet of Imuplus, Kelp and multivitamins added this month.

Current State: Same as last month - active. Just completed certification for Search and Rescue for Washington State and had 2 days of snow rescue training that she handled with no difficulty.

10 year old daughter

- only one possible sign - "isolation" mental state that almost have us dragging her to a psychiatrist. Often states "I'm depressed" etc...

Treatment: a glass of chocolate ion-exchange whey (laced with 1200 GDU of bromelain) (Prozone is the specific brand). Refuses to take supplements etc. A compromise.

Current State: She is a lot easier to live with, general mood has continued to improve from last month.

5 year old Bed Slug Dog

We have a PWC as a pet (Pembroke Welsh Corgi) who has been a bed slug. We gave him 1 oz of our mixed whey one evening... and the next day we had a puppy. In fact, we discovered that he had NO MANNERS at all -- what we mistook for manners was him being too tired! He is now on 1 oz of Immunopro (mixed) per day, and is loosing weight and keeping up the active puppy thing!!!!

June 2000

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International CFS Information

Updated on: 05/26/2000

Month 1 of Minocycline (changed from doxcycline):

Remission is proceeding well, we have started the rotating antibiotics phrase, with the rotation being every 2 months (versus Nicolson's 6 weeks).. the first change put us into major herxing for a month before returning to prior level (and expect further improvement this month prior to the next antibiotic) - again, the switch criteria is no significant herxing with high bromelain dosages.

Ken:

Sudden Onset: March 1999,

Treatment: 300 mg of Minocycline per day, ~ 1000 mg of Oleuropin (Olive Leaf Extract) and 30 grams of Immunepro per day. Rheumatoid Arthritis symptoms greatly reduced with the change to minocycline. Heavy herxing lasted ~ 3 weeks and then started to lessen (Running nose for 7 days, need 4 hr more sleep, light dizziness/NMH). Returning to state prior to starting minocycline. Will be on minocycline for one more month then we'll be changing antibiotics again [oh joy, more heavy herxing!].

Anticoagulants: Niacin, Piracetam, Bromelain, Rosavin, Aspirin,

Grape Seed Extract

Weight: no weight change (expected from the amount of herxing), blood pressure improved more (112/74 versus 128/90 at start of treatment)

Physical State: Just spent 4 hours clearing/redigging ditches with no hassle - much higher sustained energy level but with a feeling of a low 'maximum exertion ceiling'. When I strongly de-stressed my environment, found that energy level returned quickly afterwards. Adrenalin glands have little capacity - clearly see the need to keep very low stress environment (not an option, but a requirement).

Laurie:

Gradual Onset: Starting ~ April 1988

Treatment: Same as above, also switched over to minocycline (one week after me) -- heavy heavy herxing for 3 weeks, herxing is lessening now, energy level are heading back to pre-minocycline levels. At start of minocycline, she could not tolerate hot tub at all (even at 98F), but now we are enjoying 103F evening quiet times again.

Anticoagulants: As above

Weight: small additional drop of weight (unexpected because of heavy herxing). Major improvement in color (no longer a "gray"), seen very strongly in comparing her new driver license photo with her old (5 years ago) -- she looks healthier and younger with the new one!

Rest of family pretty much as prior month

July 2000

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International CFS Information

Updated on: 06/30/2000

Well, this is the month of Independence Day with it various meanings.

Ken Status

For myself, I have started to use the expression "symptomless in Seattle" - because that is the current reality. I just started my third antibiotic: Zithromax (Azithromycin) via Z-Pak (250 mg/day). I stopped bromelain (antibiotic potentator) before the switch and found that I was feeling real real good with the change. Today (day 4), I took some bromelain and felt bad about an hour later for about 4 hrs (Azithromycin has a very long half life, so the duration is governed by bromelain's half life).

At present, without bromelain - I have no classic CFIDS symptoms (i.e. I have NONE of the CDC symptoms), no fatigue or tiredness. Can spend an active day doing stuff - working up a sweat with no penalty to pay the next day.

With bromelain, I do have mild herxing which means that I am ***not free*** of the infection, but the infection has been eliminated far enough to remove all of the *hypoxia* symptoms and viral activation has been reduced to non-apparent levels.

Laurie Status

She continues to be on minocycline, there is still significant improvement from month to month with it. Next week, she will be having the ISAC panel - because of the recent mild transient ischaemic strokes - we wish to know exactly the coagulation state. Heparin may be needed.

Laurie's symptoms are: slightly nausea and unstableness in the morning, needing a slow wakeup (I snap awake, refresh each morning between 6am and 7am), needing some bed rest after some types of physical activities, significant heat sensitivity (I'm free of it, last year was real bad for me). On the plus side, doing one or two 5-6 miles walk per day present no problem, she is driving into town again with no problems, MCS is less. She has found that Benadryl tablets helps her MCS quickly and effectively (trying it came as a result of reading about how mycoplasma is known to cause hyper-reactivity to histamines).

17 year old

We are going to have her tested for helicobacter pylori infection because of her unusual food allergies and a recent article finding a correlation between this bacteria and food allergies (http://www.msi.com.pl/pdf/vol_6/no_03/530.pdf). She continues to do real fine with 10g of immunepro/day with rosavin and piracetam as needed (i.e. when she becomes unexpectedly tired OR emotionally volatile).

"Flying Sausage"

Our corgi continues being playful -- in fact, has successfully regain some 'dominance turf' from the other dogs.... we having adjusting dog politics as his energy level and mental state improve... He is on 1 oz of mixed Immunepro per day.

Other daughters

This month is getting all of the kids on 10g/day of Immunepro over the summer - as both preventative and addressing any sub-symptomatic infection that they may have acquired from the others... They know that they could get 'mild flu' from it when they start - hence the wait until summer so it does interfere with their schooling.

Supplements are as with prior months....

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August 2000

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International CFS Information

Updated on: 08/23/2000

Ken:

The last month had me crawling the walls looking for things to do from having energy etc... The cycling to zithromax triggered this feeling -- but no herxing at all (despite large dosages of bromelain). Just before our last MD appointment I drop my resume into various job sites and got a strong response. Our MD okayed a search for full time work ("reasonable" - of course) and we decided to try a month without any prescription antibiotics (giving the body a break per Jadin) - but keep taking Olive Leaf Extract which I still herx from [when I stop herxing from a large dose - I had drop the dosage down - and infact stopped it for the first two weeks of zithromax to better judge my reaction to zithromax, when I restarted, there was herxing]. -

To date: 10 months of antibiotics with heavy potentation with bromelain, 3 different prescriptions (actually 5 different brands - which is a good thing) and two non-prescription antibiotics. Non-'prescription' blood thinning for 11 months.

Wednesday and Thursday of this week was interviewing in person -- two full 8 hr days of back to back interviews, with an additional 3-4 hrs of commuting .... came thru it with flying colors - had excellent mental clarity for the
last interview (but Laurie did notice blood shot eyes when I came

home on the second day) and there was no tiredness on following days. So it looks like I will likely be back to working in the fall (September or later).

Laurie:

The real major improvement over the last month has been her back problems. With the minocycline she have noticed a band of "herxing" in her back starting where she had damage from a horse riding accident some 20 years ago, and this band has been moving up her back gradually. In May, her back problems would force her to do 1-2 hrs of stretching exercises in bed before she could get up (or other wifey things) - usually it was 11 am before she was up. Today, she often does not need stretching exercises at all and will often be up by 8 am. She no longer (after 8 months) has doubts over whether the treatment is working ... and all of the herxing has been worth it. On Monday she is getting the blood draw for ISAC and is looking forward to getting the results and possibly on heparin (if needed). She is having the common struggle of feeling better, doing more and not keeping to the protocol as rigorously as when she was sick -- she doesn't miss taking her antibiotics, but on some days, no immunepro happens at all. She continues being on minocycline.

Supplements are like prior months see
http://www.folkarts.com/idef/recovery1.htm

ISAC PANELS

We are finally doing the ISAC Panels - for Laurie, because she is willing to do injections now (thus the panel's benefit of treatment may be realized), for Ken, to verify the state of recovery and to identify any remaining issues / problems.

ISAC Panels

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Fibrinogen
Soluble Fibrin Monomer

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International CFS Information

Updated on:
02/04/2001

For our experiences on LMW Heparin click here

See Berg's Town hall meeting for interpretations:

- Meeting 2
- Meeting 1

Yellow: ISAC Panel,
Light Green: Hereditary Thrombosis Risk Panel

Measurement	***Normal Range***	***Laurie [1]***	***Ken [2]***	***17 yr old daughter [3]***	***11 yr old daughter [4]***
SED	4	34	2	5	2
Fibrinogen	180-310	459 (H)	281	308 {B}	259
Thrombin / Antithrombin Complexes	1.0-4.1	1.6	2.1	1.6	1.6
Soluble Fibrin Monomer	0-17	30 (H)	46 (H)	10	16{B}
CD62	0-27	30 (H)	20	34 (H)	27{B}
CD62P + ADP	39-80	57	53	36 (L)	61
Plt Activation Index	Normal	Normal	Normal	1+ (H)	Normal
Prothrombin Fragment 1+2	0.4-1.1	0.8	1.2 (H)	0.9	1.2 (H)
Antithrombin Activity	75-125		123{B}	136 (H)	123 {B}

Protein C Activity	60-140	129	129	122	130
Protein S Activity	65-150	55(L)	92	112	82
APC Resistance		NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
Factor II Activity	60-120	114	152 (H)	138 (H)	127 (H)
Lp(a)	0-30	10	17	6	6
PAI-1 Activity	0-15.5	0.8	normal	23.2 (H)	3.3
Homocysteine	0-9.0	6.9	normal	4.6	4.9

[1] August 2000. After 8 months of heavy non-prescription anticoagulation supplements

[2] August 2000. After becoming free of CDC defined symptoms for CFIDS. There was very significant Platelet activation early in the illness (see Aspirin)

[3] Daughter of Ken and 1st wife [who now has FM Diagnosis]

[4] Daughter of Ken and Laurie

Sepember 2000

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International CFS Information

Updated on: 09/02/2000

Ken:

Continue to do well (full recovery) -- no longer taking dogs for walk in the morning, instead I take them for a short jog instead. Off antibiotics, still doing Whey and Olive Leaf Extract.

Accepted a job (Starting September 11th) because of the firmness of the recovery (no financial need - still had a year of same-job 70% disability pay left). Got the ISAC partial results, only Soluble Fibrin Monomer (SFM) remains high for me. This agrees with the absence of herxheimer reaction, there was no high palette results (thus no apparent latent or active infection remains)

As an experiment, we will running me on high levels of bromelain, wobenzym and various nootropics (Piracetam: 6g day, Aniracetam 800 mg/d, Oxiracetam 800 mg/d, Pramiracetam 800 mg/d, Oxoproline 800 mg/d) and rosavin to see if these OR nature reduces the SFM level significantly. If it does not, then heparin is probable.

MD decided to give me a week of doxcycline to take immediately prior to the next appointment to see if any herxheimer reaction happens (because of regrowth of bacteria).

Laurie:

The summer heat brought on a relapse. The relapse was caused by the heat and compounded by not keeping up with walks (due to the heat). As often happens with CFIDS, a cascade of events developed. A switch from minocycline back to doxcycline started a recovery - she is recovering well but is about 2 months back from where she was.

Her ISAC panels indicated major coagulation and we are hoping that the heparin will be prescribed soon (this is our MD's first use of heparin for CFIDS so there is some caution (aka dragging feet a little)).

Kids

Our 17 yr old appears to have Salicylate sensistivities (she been telling us that she is *allergic to most greens and fruits* - she was telling the truth!) and is scheduled for an ISAC panel in 2 weeks, our youngest will also be having the ISAC panel since we are of the opinion that it is the most likely test to detect early CFIDS.

October 2000

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International CFS Information

Updated on: 10/02/2000

Ken:

On September 11th, I started full time work as Technical Product Architect for VisionCompass, a one year old startup. My typical day is up at 6am, drop one of daughters off at school at 7am, drive to Bainbridge Island and then walk aboard the 7:50 am sailing and then walk about a mile to work in downtown Seattle. I usually leave work at 5:10 pm and arrive home about 6:50 pm - a total of 12 hours away from home.

In general I am normal tired when I come home (likely partially cause by just having whey for breakfast and a bowl of soup for lunch). After supper, I often tutor my 17 year old daughter in pre-calculus for up to two more hours. Both of us often take 1-2 piracetam before we start so that our concentrations would be better -- when we do, the tutoring goes very well.

On weekends, I load up on heavy dosages of olive leaf extract and do notice a mild herxing reaction around by eyes and a light headache. There are several ways of interpreting this -- <my preferred> as I am still mildly coagulated, I suspect the fibrin dissolver (bromelain) and Nootropics are *exposing prior existing infection reserves* in my brain and nerves. My body could be handling them as they are exposed -- and the OLE may not be needed -- but my maxim is "***It is better to over-cure CFIDS***

than under-cure it, get as much insurance as you can!". I noticed a similar effect around the eyes when I took a week of doxcycline - which disappeared after 36 hrs after starting doxcycline.

My work involves long period of concentration (like producing a detailed written code review of 400 procedures citing problems in each one in 2 working days), and I have not noticed any problems. On occasion, I may find myself not being as sharp as I would like in the afternoon -- which I correct by taking 2-4 piracetams.

Last weekend, I was cutting firewood and hauling it up the house from the back 20 acres -- after a week sitting, it felt very good to have that extra cardiovascular activity -- if there was vascular constriction as part of the CFIDS, such activity is essential for a full recovery.. This coming month I will be taking 5 days of zithromax as additional insurance against the infection.

With work taking such a chunk of the day, and tutoring my daughter even more, I can barely scan my email and answer email directly written to me... before I need to hear off to bed to get 8 hrs a sleep.

Laurie:

The change to Zithromax and the increase (and variety) of nootropics have resulted in Laurie becoming more active and much more motivated. She is waiting for the start of heparin and well as testing for the inherited factor (there is a possible pattern with her mother("becoming like lead") and one sibling (Raynaud's syndrome). Her style of herxing has changed very much from the earlier tetracyclines...

Kids

Our 17 yr old ISAC panel confirmed that she has hypercoagulation. Our 10 yr old has two borderline measurements - if they were 1% higher than she would also have hypercoagulation. MD tentative plan is heparin for both of them, followed by antibiotics. See ISAC Panel Test Results for the specifics.

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February 2001

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International CFS Information

Updated on: 02/07/2001

Feb 1: Today, the family(11 yr old is being delayed for 1 day because of panic attack over needles) started on Heparin (Lovenox): 30 mg/person. About 90 minutes after the first injection: Laurie and I are feeling alert, mentally awake -- this is a new feeling with her and she is a little disorientated by it. I am feeling alert, almost hyper: very similar to how I felt when I tried Aspirin the first time. My 17yr old is asleep on the sofa (she has not been on any antibiotics yet but does have an active infection (High Platelet Activation)). After 3 hrs, Laurie reports that this new alertness was starting to fade.

First Injection Experiences:

- 17yr old decided to go tough: no ice or spray. Nurse injected her. She reports that it felt like a bee sting except it was not itching.
- Laurie: Iced the site. Nurse injected her. Felt nothing
- Ken: Iced the site, injected self, felt nothing

Diet Changes

Removing Wheat, Alcohol and Sugar was easy for Laurie and I (we picked up a bread machine and now making spelt-rye bread) but for the kids it is a lot harder for them to accept it.

Feb/Day 2: Well, Laurie has been playing Alchemy (http://zone.msn.com/alchemy/) for several weeks and yesterday, about three hours after the first heparin injection she reached Alchemist 1st class for the very first time. She been repeating that performance today....Ken: I've noticed that she is speaking much faster (as I am myself). Significant increase of mental speed also. I have burning / watering eyes -- this is consistent with current herxing areas / niacin flush area. We are off antibiotics, but doing Whey and olive leaf.

Laurie: "More mental speed is right, it is a very weird feeling :-O I have been on the go for more of the day than usual and feeling more alert. Also, my back pain has lessened since yesterday's Lovenox injection. I likely have an infection in injured tissues in my lower back from a horse fall in 1970, so this is very promising. All this, and I thought we wouldn't notice much until 72 hours into it! On the down side, I may be herxing. Sure feels like it... and today the injection site is sore, but I didn't ice it as long before hand." The Herxing increased over the night.

17 year old: Appears more alert and sharper.

Feb 3rd Laurie score went up to a Master Alchemist playing Alchemy (http://zone.msn.com/alchemy/), so her cognitive functions has improved further. She is herxing, especially about 30 minutes after each injection. On our daily walk, the pace was much faster and she was not winded at all from it. Suddenly she is talking so fast that I have difficulty getting in a word edge wise!

Ken: I continue to have only light stinging in the eyes (herxing), been very motivated all day (in fact found that it was hard to sit at the computer -- I wanted to be up and doing stuff.

17 Year old: Just arrived home - having to leave her Search and Rescue training weekend due to sickness. It looks like herxing has started with her (she is one miserable kid): Headaches, stomach upset... I am glad she is starting Lovenox. She just related how perfume smells are starting to trigger headaches: i.e. she is approaching multiple chemical sensitivities in addition to salycilate sensitivities. There is a chance that we may be able to back her out of these sensitivities by eliminating the chronic infection which is likely producing these sensitivities.

Feb 4th: Ken: Main item that I noticed today was that my vision

appears to be shifting (from near sighted towards normal) - having to hold books further away from me than normal.

Laurie: Herxing is less (and also different than prior herxing), still far more active than usual.

17 Year old: She had some Theraflu and felt better as a result. She does not like the shots, but is continuing along with it. She finds that if she ices the injection site, she both feels it more and also bruises.

Only significant/interesting items from now on

Feb 5th: Ken: Noticing a headache about 2-3 hrs after the Lovenox. Strongly suspect it is a herxheimer reaction. History/Evidence infers that the remaining reserves of the infection is likely in brain tissue (and the infection was probably what gave me a speech defect as a child).

11 Year old: After many pleads and tears, she accepted her first injection. Her night time insomnia disappear and she was asleep by midnight. Up in the morning... and we mean UP - far more energy than we have seen in months/years. She is almost persuaded to take another injection (but the terror of the needle is still very strong!)

Feb 6th: Laurie and the girls are increasingly herx-sleeping. 11 yr old is having no trouble sleeping at night (or in the day) - it is still a lengthy process each night to get the injection into her. 17 year old takes a 3-4 hr nap when she gets home from school. Ken seems to escape this reaction - probably eliminated the infection by antibiotics prior to heparin.

Laurie: Her lower back pain has improve greatly. It takes a lot less hot and cold time to get comfortable. Although 'tired' (in a herxing sense, - not a CFIDS sense), she has no trouble keeping up a good pace on our daily walk.

Fibrinogen

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Updated on: 08/05/2000

aka

factor I; hypofibrinogenemia test; plasma fibrinogen; serum fibrinogen

Normal Range

- 80-310 mg /dl [Berg, Hemex Labs]
- 150-350 mg/dL [ARUP Labs *]
- 160-340 mg/dl [University of Iowa *]
- 207 and 436 mg/dl [Kamiya Labs *]
- 200 to 400 mg/dl. [Adam's *]

Explanation:

"Fibrinogen is a soluble precursor of the insoluble fibrin, the major component of a blood clot. It is a long, 34,000 dalton glycoprotein composed of six subunits. When fibrinogen is activated by the hydrolytic enzyme thrombin, four subunits are removed. *The remaining units polymerize into fibrin strands which form the basic structure of a blood clot*. Most fibrinogen is intravascular. It is synthesized in the liver, approximately 2-5 grams per day.[1]

Elevated levels of fibrinogen are associated with inflammation, trauma, surgery, and malignancy.[2] Decreased levels are associated with congenital deficiencies or an increased use due to thrombosis or disseminated intravenous coagulation. The most common cause of low plasma fibrinogen is *disseminated intravascular coagulation (DIC), a condition in which blood clots form throughout the microvascular system*. DIC can be associated with some of the serious complications of childbirth. When fibrinogen levels fall to the point where blood is unable to clot, dangerous bleeding can occur. Fibrinogen levels below 100 mg/dl are associated with an increased risk of bleeding." [*]

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Soluble Fibrin Monomer

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International CFS Information

Updated on:
08/24/2000

aka

see http://www.hemex.com/sfmdiagram.html for the relation of SFM to other coagulation factors.

Normal Range

- 0 - 17 mg/L [Berg / Hemex]
- 0 - 15 mg/L [Emory University *]

Explanation:

Increased blood viscosity due to the generation of Soluble Fibrin Monomer (SFM). [*]

Transcripts

Dave Berg 913

July 16th, 2000

OPENING COMMENTS:

[BERG] Greetings to all and thanks to KEN for asking me to return to the TOWNHALL. Before we get to specific questions, I want to update everyone with new info about coagulation that I garnered at the Scientific Sub Committees (SSC) Meeting (International Society Thrombosis & Hemostasis-ISTH) in Maastricht, Netherlands last month. The more we learn about coag, the more our model stands up and appears to be correct. I even spoke to Prof Vermylen (editor of the ISTH journal 1993-1999) about his model of "Antibody Mediated Thrombosis" (published in 1997) and ask whether his model could apply to Fibrin Deposition as well as a blood clot. After several minutes of silence, he said that it could-yes indeed. He hadn't thought about that concept before, only that of a full blown clot. I thanked him for his validation.

[BERG] A question that I have always wondered about was "Why don't CFIDS patients get blood clots routinely?" The answer came from a lecture about new findings on coagulation Factor XIII (thirteen). XIII cross links Soluble Fibrin into INSOLUBLE FIBRIN STRANDS. [The fibrin strands are what hold a scab together after you injure yourself. The red color of the scab is due to the trapped RED CELLS in the scab.] A high level of SFM in the blood is only one SMALL step away from a blood clot. The answer lies in the amount of thrombin needed to activate XIII (one chain) to its active two chain form, XIIIa, which is the active enzyme that forms insoluble fibrin. You need a "THROMBIN BURST" to activate XIII to XIIIa. When there is continuous ongoing LOW LEVEL THROMBIN GENERATION, you generate a lot of SFM, which becomes "Fibrin Deposition", not a blood clot. Thus, the basis of our model is further substantiated.

[BERG] POP QUIZ TIME ! Did you understand all that technical stuff????? This answer makes it even MORE IMPERATIVE that CFIDS patients learn what their underlying coag protein defect IS. Eventually, all of us will have an accident, trauma or elective surgery. When you have a major wound to the body, you WILL have a THROMBIN BURST. At that time, it will be very important that your physician give you something to prevent a blood clot !!! (low dose heparin or Coumadin). This is a statement to CFIDS patients to be cautious when you need to be.

---

*[Fluffy]**What is the significance of CD62P + ADP ?***
My test results are as follows for Platelet Activation by Flow: CD62P is 61 which you says indicates an underlying infection along with Platelet Activation Index which is 3+. But CD62P+ADP is 6 which is low. DOes this mean an inactive infection or what ? Could you clarify what this means ?

CD62P:

[BERG] CD62P is a platelet surface marker for a glycoprotein that is normally inside the platelet. When is it expressed on the surface, it means that the platelet is activated. Normal people have values 8, 12, 15%. Because we use 2 Standard Deviation, the Normal Range goes to 27%. If a platelet is already activated (61%), there is LESS to activate: CD62P + ADP = 6 (low). Thus, the

3+ Index. My values are 14% & 62%, ie, my platelets have little activation initially, and when you add ADP, they activate a lot (62%), which = NORMAL.

[BERG] Now, there is more to the story than this, HHV6, according to the patent held by the US government, states that HHV6 infects endothelial cells (EC), megakareocytes (platelet producing cells) and neurons (brain). The Platelet Activation Index (PA) test gives us much information. CD62P values that are 25-40%, with normal activation, indicates that the platelets are coming out of the bone marrow activated from the infection. This is a latent infection. When both the CD62P and the PA Index are Positive, this indicates an active infection. I have missed interpreting values at times because both values are normal in patients. I believe that this is because the bone marrow is NOT infected at that time, so around 20% of CFIDS patients may have normal PA values. When patients who have normal or only elevated CD62P values are treated, usually the PA index becomes very positive, which is like the Herxheimer reaction that Ken talks about. It shows activation of the PATHOGEN in reaction to the therapy to kill the pathogen. I hope this answers the question.

---

*[bink]**Can diet have a postitive effect on clearing coagulation abnormalities?***
The "alternative" specialist believes one can thin their blood by eating a particular diet. I believe one based on veganism. Can diet have a postitive effect on coagulation?

DIET:

[BERG] YES, BUT.....Diet is a very slow process to anticoagulate a patient. Instead of relief in a matter of a few days, dietary changes may take MONTHS to achieve the same effect. If a patient has IBD (irritable bowel disease) from the illness, diet may not do anything, since the IBD may not allow for proper adsorption of nutrients from the gut. The IBD is primarily caused by thick blood (increased SFM), with the decreased blood flow not providing the proper nourishment to the gut itself. ISCHEMIA is defined as lack of oxygen and nutrients to a given area. If the bowel itself becomes ischemic, it will sluff it contents. So, you end up with IBD from increased SFM. In this case, only exogenous anticoagulation will help in a timely manner. A healthy diet is always something desirable for all. Just think when you have had a delicious heavy meal with lots of fats and you can feel those arteries clogging up from the chylomicra floating in the plasma. AH, those good old days !

[BERG] Antibiotics kill the good bacteria in the gut that make Vit K for us. Therefore, antibiotic therapy can produce a mild anticoagulated state, similar to using low dose Coumadin. But many patients have reported that Coumadin does not work very well on them, so they go back to low dose heparin.

---

*[James Roberton]**Chiari, cervical spinal stenosis and thick blood***
Do you believe that some people are unecessarily having surgery to relieve symptoms that are actually the result of thick blood and swelling of the CNS? Is there any evidence to support this idea?

CHIARI:

[BERG] This question is a difficult question to answer: the chicken and the egg question. If the blood viscosity goes up from the increased SFM, it is possible to cause brain swelling. The only way to answer this question would be to take a study group and do surgery on half and give heparin to the other half and compare the results. My personal thinking is I would rather try the heparin approach first, and if it did not solve the problem, then consider the surgical intervention.

---

*[Cindi Anderson]**SED Rate Definition***
When you talk about low SED rates? Are you speaking of them measured as ESR?

SED RATE:

[BERG] The ESR (erythrocyte sedimentation rate - red blood cell sed rate) is called SED RATE for short. We are close to having enough data to publish that the normal range for SED RATES should start above 3 or 4. Values below this are correlated with high SFM values. As the Soluble Fibrin Monomer (SFM) goes up in the plasma, these molecules form dimers (2 stuck together). This physically blocks the RBCs from settling out of the plasma, thus a low sed rate. The only other clinical condition to demonstrate low values involves paraproteins in the plasma such as in a cancer patient.

[Cindi Anderson]Influences on testing for Coagulation

I have been trying to get an answer to this question from your clinic. What medications would make the results from your tests not valid? For example, I was taking Niacin to reduce Lp(a). Would this affect the hypercoagulation tests and if so how long should I not take the Niacin before taking the tests? What about antibiotics, gamma globulin, Catapres. These are things I take; I think a general answer covering all types of medications would be useful to people.

TESTING:

[BERG] The ISAC panel answers the question "what is my current state of activation of the coagulation system". There is really only one therapy that can give erroneous answers, and that is an IV of 500 mls or more prior to having the blood drawn for the testing. If a patient has any type of IV within 5 days of the blood draw, this can result in negative or normal answers, which may not be representative of the patient. Aspirin, in high doses, might lessen the platelet activation, but if there is an HHV6 infection, the CD62P should still be positive. My suggestion is to continue with what you are taking now, other than an IV or aspirin, because we want to know how well those current medications are working or not working. We also use the ISAC panel as repeat testing one month or so on heparin to see the beneficial changes it has. This also answers the question "is this enough heparin or not?". We use the one month testing to adjust therapy in most patients, as well as looking for the reactivation of the pathogen(s) [herxheimer reaction] in the patient. Occasionally, the physician will order the ISAC late in therapy to look for complete normalization of the test values.

[BERG] As for the HTRP (Hereditary Thrombosis Risk Panel - risk factors), most of these protein levels do NOT change much during life. They are genetically controlled. So as long as your physician knows what you are taking at the time of the blood draw, these values can easily be interpreted. If you know you have a high Lp(a), for example, stay on the niacin and see what the value really is (is the therapy really working?). As for Bromelain, this aids in the process of cleaning up the blood vessels, just as heparin aids in shutting down the SFM generation. Neither effect the protein levels. They are exogenous components.

[BERG] As for the positivity we see in the ISAC panel, we just submitted an abstract for the AACFS meeting next January, in Seattle, where we looked at 400+ patient results. 79% of the patients had positive (2 or more tests) ISAC panel results. Another 19% had one test positive. This equals 98% positivity in patients. When analyzing the data further, I have come to the conclusion that as the pathogen(s) gets a large, comfortable area which is infected with lots of fibrin deposition, the less active the pathogen needs to be. Thus, in patients who have been ill for more than 10 years or so, the less likely they will test positive for 2 or more tests. HOWEVER, when given heparin and/or Transfer Factor and/or antibiotics, as the fibrin layer is cleaned up, these pathogen(s) reactivate the coag system in an effort to make fibrin again. This is why on repeat ISAC testing at one month, we see MORE positivity than we may have seen on baseline testing. In regards to the HTRP panel, around 75% of all patients have a demonstrable defect of risk factor in this panel of 8 proteins. I think it highly likely that if we tested for 4-8 minor regulatory proteins, we would find a defect in over 90% of all patients. Part of this conclusion is

reached because in patients with no defect found, there is still an increase in Protein C, Protein S &/or AntiThrombin in an effort to shut down the hypercoagulable state ! This panel also gives us much more information than just protein defects.

[JJWalker]Hormones and Coagulation

How would the lack of particular homones that patients with M.E. are known to be absent, such as ADH and or Oxytocin, effect blood coagulation.

HORMONES:

[BERG] I can only answer this question as a general statement. If the blood viscosity goes way up (very low sed rate), then the blood flow throughout the body is greatly decreased. This includes blood flow through the endocrine glands from which the hormones are produced, such as all the publications on the HPA axis and decreased hormone levels. If you were to study a group of patients with low HPA hormone levels baseline, then give them 30 days of heparin and retest those patients, I bet that the hormone levels would normalize. The exception to this would be that, if there is reduced blood flow for a very long time to an endocrine gland, the gland itself might be in failure. This would cause continued low hormone levels for a long time.

[BERG] Speaking of thick blood, there are many studies about reduced BLOOD VOLUME. I think we finally have an answer to this also. In looking at microscopic blood samples, we have now seen actual SFM fibers precipitate out on the slides. These are single strands, not cross linked strands as in a clot. This is also what the model predicts. Since this is correlated with high SFM levels in the plasma on these patients, we can say that the levels on the slide are high also. The additional effect seen on the slide is the remarkedly high ROULEUX FORMATION of the RBCs (red blood cells). This looks like a stack of coins, if you can imagine such. Since rouleux formation is NOT normal, it is possible to conclude that the increased SFM is responsible for holding the RBCs together in chains of 3-10 cells long. Since this is what is observed at 1000 power and 8000 power under the microscope, then it is possible that this is happening in the body. If so, then the oxygen delivery will be greatly reduced because the cells stick together and oxygen cannot escape, ie, hypotention. If you are looking at blood volume based on RBCs, then a group of RBCs stuck together would act as one cell, not 3, 8 or 10, etc. Thus you could conclude that there is a low blood volume. This will be interesting experiment to repeat a patient with “low blood volume” after a short period of time on heparin. The blood volume would probably correct, since the cells would not be stuck together by the SFM.

[BERG] I don’t want to appear as a know-it-all, but there are logical answers for most of the problems seen in CFIDS symptoms. We just have to look for answers to these complex questions, and most answers come from RHEOLOGY (Blood Flow) and are base on the laws of physics. So if you increase the viscosity (SFM) you create different results in CFIDS patients than seen in people with normal blood viscosity. This may also be involved in the question of alkaline blood pH in patients verses and normal acidic values found in healthy people.

[BERG] I have to go back to the statement I made 2 years ago. 30% of the proteins in the blood plasma (liquid portion) are involved in the regulation and maintenance of the coagulation system. Understanding this, it is no wonder that so many defects can appear if there is a coag problem. And we are all born with about 40 defects in our human genome (which causes us to be different from each other). Thus, around 5% of the population have bleeding defects (hemophilias, von Willebrand disease, XI defects, etc) and 5% have clotting defects, which can lead to chronic illnesses when the coag system is not controlled properly.

[JJWalker]Athletes and overtraining

I read that a doctor at UGA found that many ME patients who were overtraining had coagulation problems. How does overtraining contribute to hypercoagulation and is this a different subset than the rest?

ATHLETES

[BERG] I'm sorry, but I cannot answer this question. Overtraining causes what TYPE of coag problems? I don't have enough information on this question to answer it.

[Fluffy]How would Bromelaine effect the ISAC test ?
I was taking that and wonder whether any of the test results would be invalid. I did stop taking it about 36 hours before taking the heriditary part of the test.

BROMELAIN

[BERG] I do not have much knowledge on Bromelain. What I have read in the scientific literature is that it helps with the activation of fibrinolysis, like tPA does. The literature does NOT state how it might activate fibrinolysis, but if there is a chance that it can, then it should be used by patients who have either a high Lp(a) or PAI-1 protein. As to standardization of the various manufactures, this is information I do not have either. I was told by a knowledgable person that one should take 400 mg, 3 times a day, ie, between 1000 and 1500mg a day. If someone has all the conversion factors and knows what is the best dosing, please email me with the information. Thanks.

[BERG] I am pleased to announce that we have had the first 2 patient trails of using tPA in an outpatient IV setting on patients with very high Lp(a) values. The first patient's comments were that he felt better, the tingling in his hands and legs disappeared and the mental fog dissipated. Not too shabby ! Similar results were seen in the second patient. This is a VERY EXPENSIVE therapy, $1500 for the drug alone. But in patients with high Lp(a) or PAI-1, this may be "just what the doctor ordered". As we get more experience with this, we will let patients know.

[] Are there any special considerations for patients who test positive for >parasites, yeast and/or Mycoplasma??

(For example, should any of these be treated before treating coagulation problems)

[BERG] This is a good question to drive home the idea that the PATHOGEN(S) are half of the problem with coag defects being the other half. Whether the underlying problem is HHV6, CMV, EBV, Chlamydia Pneumonia, Mycoplasma, Rikettsia, Lymes Disease, Candida, etc., the pathogens MUST be treated. ALL of these activate the coag mechanism, because it is part of the host defense mechanism. It is when a patient has a regulatory defect that the disease turns into chronic illness. Anticoagulants (heparin & Coumadin) shut down the ability of the pathogen to generate fibrin. So the pathogen is left in a vulnerable state. Thus combined therapies, anticoagulants, antibiotics, antifungals, transfer factor, Ultraviolet blood irradiation, IV peroxide, and perhaps even IV high dose vitamin C therapy, may all have there place. It is up to the clinician to decide the best therapy. My personal experience of seeing positive ISAC values turn negative with TF is the first time I have ever seen anything touch HHV6. Treatment of the pathogens can be concurrent therapy, or anticoagulants can be started first. Using heparin to help clean up the capillaries before therapy makes many of these therapies more effective. I also advocate staying on heparin for 1-2 months after the pathogen therapies is completed because if there are any pathogens left, they cannot generate fibrin with the heparin present. This also gives the body the extra time to wipe out the remaining pathogens naturally.

[SUSAN] ISAC Panel

My 21yr old daughter has had SED rates of 2 and 5 over recent years. Her ISAC panel was all within range, however. The Hereditarty Panel showed a borderline 66 on Protein S activity and a 10.8 for Homocysteine. Is this enough evidence of a hypercoaglable state and need to proceed with your protocol?

[BERG] As stated before, low sed rates indicate a hypercoagulable state at that point in time. Borderline Protein S deficiency is a very real problem. When the body is stressed beyond normal, the Protein S dropps even lower (C4b Binding Protein is the carrier protein and when it increases, it binds up the free Protein S). Homocysteine usually reflects dietary problems. If B12, Folate & B6 doesn't correct the value, then there are 2 genetic enzyme deficiencies that should be tested.

This is a great question in that some patients are borderline on their testing. Does this prove anything??? I suggest that because the low dose heparin therapy is NOT dangerous, give it a try for 4 weeks. If the patient feels better, GREAT. If the patient gets worse, check the ISAC again to see if you have activated the coag system from a pathogen (herxheimer reaction). This is the case in over 90% of the patients. Patients DO NOT react to heparin (less than 1%). Heparin is in all of us and as we age, the loss of the ability to make heparin may be the biggest part of the aging process. Patients who have reactions are usually reactions from the pathogens !

[RUTH]Platelet activation by Flow and chronic, active infection and Lovenox usage

My last CD62 P was 10
CD62 P + ADP 36
and Plt Act Index 1+

Is this an active infection state? My need for Lovenox is continuing to diminish and is now down to 15mg a day. Would that indicate active infection to you or would you say that I am better maintaining the chronic DIC?
Thank you David for all your work
Ruth

[BERG] You have pointed out the third possibility with the PA Index, that of a normal CD62P and a Positive Index. This is easy to answer in that we see this type of activation in PEOPLE who have bad coronary or carotid arteries or veins (vericose veins). It is due to a restriction in the size of the vessel which speed up the blood flow through the constriction, just as water speeds up if you put some bolders in the stream and narrow the channel. This is MECHANICAL activation of platelets due to a stenosis. These patients need 81mg of Aspirin daily to minimize this effect.

[BERG] I don't know if you have had a repeat ISAC or not. If you have children, then vericose veins are a real probability. If you are still concerned about this, call me. Since you are doing much better and the Lovenox doseage is less, that's GREAT !

[ ]Any further ideas for non-responders

For people who do not respond to well heparin alone and who do not have hypofibrinolysis, do you have any new suggestions beyond adding Tranfer factor, and antibiotics?

[BERG] As I have said in other answers, patients should not have reactions to heparin. It is the pathogens that are causing the reactions. This question sounds like the pathogens are NOT yet

identified in the patient. What about parasites or fungal infections. You must get rid of the pathogen to get well.

[BERG] We ARE seeing success in many patients now. Our long term patient, Beth, is probably back to 90% of her original life style. That's GREAT ! She is an example of hypofibrinolysis and HHV6 and it has taken over 4 months of TF therapy to get rid of the HHV6.

[BERG] In closing, there is some information that I want to share with you about one other protein that fits into this puzzle. Dr Urnovitz (Berkeley, CA) has discovered chromosome rearrangement on chromosome 22q11 [*], especially in Gulf War illness. This may also be the basis of chemical sensitivity patient problems. This is the location for the production of Heparin Cofactor II. This is one of 4-8 minor regulatory coagultion proteins involved with control of thrombin generation. Several of the patients tested have had low levels of this protein.

[BERG] When thrombin is generated, AntiThrombin (AT) grabs the excess thrombin in the plasma. There are 2 binding sites on the thrombin molecule, exocite I & II. AntiThrombin binds to exocite I on the thrombin molecule forming T/AT complexes. When there is fibrin (SFM) present, thrombin binds to SFM via exocite I and AntiThrombin cannot bind and down regulate the thrombin. Thrombin bound to fibrin remains active for a short period of time, causing more thrombin generation. This is how a blood clot forms quickly (within a few minutes). Heparin Cofactor II binds to exocite II, and thus, it can down regulate thrombin even when it is bound to fibrin (SFM). This is the backup mechanism for AT. So, if there is a defect in chromosome 22q11, this could explain some of the lack of control and excess SFM generation in patients who do not have a demonstrable risk factor in the HTRP.

[BERG] Thanks again to Ken for inviting me back to the TOWNHALL. I continue to give thanks to the good Lord for giving us the ability to solve these “Blood Curdling Mysteries”. Additionally, thanks to all who have sent me literature that you have found pertinent to coag and these disease processes. Your tireless reviews of web sites, literature banks and news is very appreciated. One person just does NOT have the time to gather all this information. At least with your help, I am able to add important findings to the disease model and processes, which gives benefit to all patients. KEEP THOSE CARDS AND LETTERS COMING, FOLKS !!! Email: dberg@hemex.com. I am still way behind in answering some email and phone calls. Please be patient or email me again.

[BERG] There are several conferences, presentations and meetings coming up which require a lot of travel for me.

- San Francisco, July 25 & 26th, for the AACC meeting and exhibits,
- Washington, DC, Aug 14th, for the Institute of Medicine hearings on Gulf War Illness,
- Rochester, NY, Aug 18 - 20th, for the CFIDS meeting
- Honolulu, HA, Sept 1th, for Grand Rounds and teaching conference for Kaiser Permenete Hosps,
- New Orleans, Sept 7-9th for the Infectious Disease meeting, And finally,
- Tours, France, for the International AntiPhospholipid antibody Syndrome meeting where we are presenting two papers on CFIDS & APS.

If you are interested in some of these abstracts, they are available on our web site: www.hemex.com.

TO BETTER HEALTH FOR ALL.

David Berg , MS, Director, HEMEX Laboratories , Inc.

Transcripts

Transcripts
Guests
Speaker Info

CFIDS Town Hall

It has moved to:http://64.45.50.181/townhall/index.asp

Transcripts

Transcripts

Transcripts of prior town hall meetings are available on click on the speaker's last name on the left.

Any member of the CFIDS/ME/FM community may invite speakers. The process is simple:

- Email them, asking them to attend (you may cc: CFS@Folkarts.com so we are aware of it)
- If they accept, then they have the next available date that works for their schedule (Sundays preferred but not required)

Speaker Instructions

Process

- Audience post questions, both during the meeting and days before (pre-submitted) questions
- Audience may vote for questions - allowing a ranking of the questions
- Speaker select the question(s) they choose to answer

Posting Answers

- The speaker page displays questions on the right and a page to provide answers on the left.
  - Clicking on a question moves the question text to the answer page
  - Type in an answer and then click submit to send it to the website
- Backups: technical snafus do happen, the following are 'fall-back' procedures
  - Backdoor.asp is a page that allows you to 'cut and paste' questions and answers. This sends the contents directly to a text file on the server where **support** will retrieve them and then manually post them onto the web site.
  - Email answers: Cut and paste questions and answers into an email and email it to CFS@Folkarts.com. Usually messages will arrive within a minute, then support will manually post them to the web site
  - Telephone 1-360-297-4717 (Ken Lassesen): You dictate, he will type (using hands-free headset)
- Pre-check: Speakers are encouraged to go to the Checkup page (using the PC and Internet connection they intend to use) a few days before the talk to verify that the technical side will work correctly.
- Answering pre-submitted questions is *encouraged*. The answers will not be available to the audience until 30 minutes *before the Town Hall meeting starts*.

Distribution of Transcript

- After the chat is finished, the transcript may be corrected or added to before being distributed via email to various groups that have expressed interest.
- The purpose of the Town Hall is to provide information to CFIDS/ME sufferers around the world, thus we encourage republication of the transcript by email, mail lists, websites and newsletters *without any restrictions. Feel free to use on your own website as you wish.*
- *If a speaker wishes to restrict distribution - this is acceptable.* Please insure that you enter a **Copyright Notice** when you logon, as well as emailing cfs@folkarts.com with the restrictions notice that you wish placed on the transcript.

Transcripts

Speaker's Browser Checkup

There are several technical snafus that can occur with a town hall meeting. To insured against it the following is recommended:

- 1 or 2 days before the town hall, read a few of the pre-submitted questions. Select a few questions, answer them in an email and send it to cfs@folkarts.com . This allows some answers to be posted by support if technical problem arise. (It is perfectly ok to answer all of the pre-submitted questions before the town hall) .
- Fill in the box below and then click submit. This test that what you type can be transmitted to the Server successfully.

Type something below

Submit Reset

Transcript 3 2 5 9

- Speaker: David E Berg
- Latest work/publication: SUGGESTED TREATMENT PROTOCOLS FOR CFS / FM PATIENTS (http://www.hemex.com/cfs/cfs_tx-2.html)
- Website: http://www.hemex.com/cfs/
- March 26th, 2000 12PM (Noon) PST, 3PM EST

Held at the CFIDS Town Hall (http://www.folkarts.com/townhall/)

[David] Greetings to all. It's an honor and pleasure to be on line today and especially to follow Mr. Regush. His book is excellent and RIGHT ON !. (It's funny not being able to see faces that are included in the dialog.) But such is the NET. For some time now, I have been frustrated knowing that the coagulation part is only half of the problem and that one or more pathogens are the other. And that HHV6 has had no know treatment until now. So let's begin where Mr. Regush's book ends in October, 1999.

In November, 1999, at the Infectious Disease Annual Meeting in Philadelphia, I saw a poster on HHV6 and spoke with the author, Dr. Joe Brewer of Kansas City. Over a four hour plus dinner meeting, we worked out the model that is being presented now about a basic coagulation or fibrinolysis regulatory protein defect in CFIDS patients as the genetic culprit. Then you add in a pathogen (HHV6, CMV, Mycoplasma, Chlamydia pneumonia, etc, or a combination of several of these pathogens) and the patient goes down hill rapidly into chronic illness due to the pathogen activating the coagulation mechanism. This is due to an immune response as well as inflammatory responses to the pathogen and probably the pathogen itself activating the coagulation system. Anticoagulants (primarily heparin) shut down the Soluble Fibrin generation and fibrin deposition on the Endothelial Cell (EC) surfaces. But unless the patient can get treatment for the pathogen, the healing response can only reach 50% or so.

My frustration has been HHV6. Dr. Brewer told me about a new colostrum derived, highly purified Transfer Factor (TF) that would contain only specific IgG and IgM antibodies against CMV and HHV6 (see www.immunitytoday.com). He started testing many of his patients for their coag defects and we found such in every patient. Each patient also had documented HHV6 infection. Beginning in December, Dr Brewer began treating his patients with this new TF. Patient stories are dramatic. We will discuss some of them.

In early December, 1999, at the American Society of Hematology, we met Dr. Konnie Knox. After spending two plus hours discussing theories and therapies, we were all singing the same hymn. So the circle from last week to now is complete. The Good Lord has put Lois & I here at the right time, in the right place with the right knowledge and the right people to be able to solve these "Blood Curdling Mysteries" of chronic illnesses, and they extend beyond just CFIDS patients.

WHY is it important to be tested for the coagulation defects? It is VERY important, because at some point in time, all CFIDS patients will need surgery, be in an accident or traumatic situation and NEED to have PROPHYLAXIS to prevent a blood clot, stroke, heart attack or Pulmonary Embolism from happening. If you know your protein defect, then proper anticoagulant therapy will prevent catastrophic events. I feel very strongly about this.

If you look at the population of America and the patient race distribution of CFIDS patients, there are about 5% of bleeders (hemophiliacs or von Willebrand Factor deficient patients) and about 5% clotters. Using a bell shaped curve, 260 million USA population yields 13 million clotters. 1 million CFS, 8 million Fibromyalgia, ? Million Multiple Sclerosis, ? Recurrent Spontaneous Abortors, etc. Are we close? The protein defects have mostly risen from European decent and are mostly white people. Hundreds of years ago, when someone cut themselves hunting or preparing food, it was advantageous to clot fast (not bleed to death). Life spans were shorter then also, so these coagulation or fibrinolysis regulatory protein defects were beneficial. Today, with much longer life spans, these defects cause chronic illnesses by not controlling the coag response properly. So much for my PhD thesis.

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1. **[Fluffy]Are there any non prescription treatments you could recommend and what about future treatments ?**

   Aspirin supposedly attacks one of the factors involved in coagulation. Bromelaine supposedly attacks all 3 of the factors. There are research indicating that bromelain increases antibiotic absorbtion. I currently take 2500 mcu per day with minocin and it seems to help. I did experience a very slight headache initially and it seems to help with the brain fog. Any comments on this and other possible supplements. Also what is the timeline on future treatments ? Please also include anecdotal and personal opinions in your comments.

   [David] The ISAC Panel contains a test called the Platelet Activation Index. What we have learned is that this is really showing us whether or not there is an infection in the patient. If the CD62P alone is elevated, then this indicates an UNDERLYING INFECTION. When both are elevated and the PA Index is 1-3+, then this indicates an ACTIVE INFECTION. My guess is that HHV6 has infected the bone marrow (as it can!) and is inside the platelets when they leave the bone marrow for the blood stream. Because the immune system “sees” infected cells, Immunoglobulins (IgG or IgM) attach to the platelets, causing the alpha granules to partially release and CD62P gets transferred from the inside of the platelet to the outer membrane. This accounts for the elevated CD62P value in the assay. The higher the CD62P value, the greater the infection. Aspirin is NOT going to be effective against infected cells, and this is what we have seen in general, that ASA does not make the patient feel much better.

   [David] I am in the process of forming my opinion on Bromelain. Elly (in Wash DC) told me about this last fall, but I did not understand her. Several months ago I did a literature review on Bromelain and was amazed at the scientific articles related to Bromelain. Bromelain, from pineapples and totally natural, seems to help FIBRINOLYSIS. There are no studies to actually prove this, but it is STRONGLY suggested in literature that it does activate fibrinolysis. Since no docs or researchers will touch using tPA or Urokinase (drugs that activate fibrinolysis in vivo) in CFIDS patients, Bromelain seems to be just the ticket. And it is all natural. Many anecdotal responses that I have received, confirm that it helps in patients that have elevated inhibitors of fibrinolysis - Lp(a) or PAI-1 - as their underlying genetic defect. So, Bromelain helps increase fibrinolysis. As for it inhibiting platelets or the coagulation cascade, nothing in literature suggests that it does such. I may have overlooked something, so if anyone has a reference to the contrary, please send it to me. Thanks.

   [David] As for minocin, I have no knowledge of its properties or use, except that others report good results using it.

   [David] As for Bromelain enhancing antibiotic adsorption, I believe it would work like this. By increasing fibrinolysis, any fibrin on the endothelial cell (EC) surfaces (the cells that line the capillaries or very small blood vessels in the body) would be mostly removed, and that would make the antibiotics more effective at getting into the infected EC. Since Bromelain is a digestive aid, then more might be absorbed through the GI track as another possibility.

[David] As for a time line for therapeutic agents, we just posted one on our web site Friday. It uses heparin for 6 months, adds Bromelain at the beginning for 4-6 months for patients that have a increased Lp(a) or PAI-1. The time line starts with Transfer Factor after 30 days of heparin for 2-3 months. Also, antibiotics are started after 30 days of heparin. Using heparin for 30 days first (plus bromelain if indicated), gives the body time to shut down the coagulation mechanism during the first 30 days and allow the fibrinolytic system to clean up part of the fibrin deposition on the EC surfaces. This makes the Transfer Factor (TF) and antibiotic use MUCH more effective. The patient continues to use heparin for another 2 months, just in case. If there are still a few pathogens left after these therapies, they will attempt to reactivate the coagulation cascade again, to generate Soluble Fibrin &/or fibrin deposition. So by continuing heparin, this will prevent cascade reactivation and the immune system will be able to clean up the remaining pathogens. From information given to me by patients on this new TF, I think we NOW have a treatment protocol that will get patients ALL the way back to good health. This was a long winded answer, but the question was a good one to answer and gives much of the information about these processes.

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2. **[Bob R.]Time Frame of Treatment**

   David, I have been on Lovenox 30 mg for almost 4 months. I received an dramatic improvement in IBS symptoms, brain fog improved, fatigue improved somewhat however nothing dramatic. Two weeks ago I switched over to standard heparin and have started to feel a little better. In short , if possible at this point, have you had any experience with patients recovering very slowly for lets say a year time period. Or do you notice immediate improvement with your patients over a very short time frame?

   [David] I BELIEVE that most (>80%, if not ALL) CFIDS patients have an underlying infective pathogen (HHV6, CMV, Mycoplasma, Chlamydia pneumonia, etc, or a combination of several of these pathogens). Anticoagulants stops the coagulation component but does nothing against the underlying pathogen. Thus the need for antibiotics, antivirals, Transfer Factor, etc. You need BOTH heparin and some treatment against the pathogens. That's why patients on heparin ONLY get about 50-70% well and not 100%.< /P>

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3. **[Fluffy]Could food sensitivity of ME/CFIDS people be related to coagulated blood ?**

   ME/CFIDS are prone to food sensitivity. Calcium is suppose to promote blood coagulation so foods like milk, cheese may seem like they could promote blood coagulation and have negative consequences for people with coagulation problems. Are there any foods which seem to promote blood coagulation ? Please also include anecdotal and personal opinions.

   [David] Most of the peripheral problems of the CFID patients (HPA axis, headaches, brain fog, IBS, and allergies) are caused by poor blood flow due to thick blood (hyper viscous blood). When heparin is used to "thin out the blood", this decreases the high blood viscosity by shutting down Soluble Fibrin Monomer generation. When viscosity returns to normal, these peripheral problems lessen or go away completely. We have seen these allergy problems (complete with increased eosinophils on blood smears) from our early days of infertility testing 7 years ago. The allergies decreased significantly in these patients as they use heparin throughout their successful pregnancies. (To date, we have already had over 400 successful first time deliveries of normal healthy children in previously infertile women.)

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4. **[Kru Heller]**What other non prescription treatments can be used for treating thick blood.

   Aspirin and Bromelain were mentioned above. I have also heard of the use of Vit. E, Garlic, Pycnogenol and Ginko. What amounts should be used? and how often?

[David] Remember the ACE of Hearts! Use Beta Carotine (15mg or 25000 IU) at NIGHT time,1gm Vit C am & pm, and 400IU Vit E pm (A,C,E for a healthy heart) . 60 mg Ginko am & pm, and Glucosamine (500mg)/Chondroitin (400mg) am & pm and 81mg ASA at night. The Ginko & Glucosamine/Chondroitin have very mild anticoagulant effects as well as aspirin as an antiplatelet. Since these are VERY mild in their anticoagulant effect, it would take many months to notice any improvement in CFIDS as an anticoagulant using these. That is why I strongly recommend the heparin protocol for immediate therapy. The B-Carotine increases tPA release from ECs over a 12 hour period, so take at night when PAI-1 routinely goes up at night. Everyone has an opinion on supplements. All I can say is to find the right combination for you.

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5. **[Sean L]Different blood thinners**

   Dear Mr. Berg, When you use heparin to treat CFIDS, do you think it is purely its blood thinning properties that help, or are it's other properties (such as it's antiviral properties) part of the picture. I ask because when I talk to people who have tried different blood thinners they seem get quite different reactions to each. Heparin seems to get the best response, Coumadin the weakest and Lovenox somewhere in between. Thank you for all your hard work in this area. Best regards, Sean (Lovenox 30mg/day for 3 months, slight +'ve response, soon to switch to heparin to see if there is a difference in response).

   [David] Coumadin is only an anticoagulant. It works by decreasing Factors II, VII, IX & X and Protein C and Protein S in the blood. The negative about coumadin is that any green foods that contain Vit K counteracts the coumadin effect, so you have to be very careful about diet, even if you are on low dose coumadin(<2.5mg/day). Heparin is an anticoagulant (anti Factor X and II), anti-inflammatory, antiplatelet, vasodilator, increases NO production and other beneficial side effects. It is normally occurring on the surface of ECs as heparans or heparan sulfate. It is a large molecule and the heparin solutions contain many different sizes, from low molecular weights of 2000-10,000 to high molecular weights up to 25,000. There are two sources for heparin: bovine and porcine. Porcine is less allergenic and the recommended type. Low molecular weight heparins (LMWH), such as Lovenox, is made up of heparins form 2000-9000 size (frequently around 4-6000 size). I like the regular heparin because it is inexpensive compared to Lovenox and seems to work the best.

   [David] There is hope for 2001 to get rid of the needle when an oral heparin from Emisphere Technologies will be available. I've asked about compassionate use for 2000, but Emisphere will not release any until the current Phase III trials are finished and the product is approved by the FDA. Our work on this technology over the last 2 years indicates that the product really does work!!!

   [David] Anticoagulants still do not address the problem of THE UNDERLYING PATHOGENS (HHV6) !!!.

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6. **[Sean L]Plavix**

   Recently Prof. Al Cocchetto told me that some GWS sufferers where doing well on a potent new platelet activation inhibtor called Plavix. Do you have any opinions on the use of this drug for CFIDS/FMS/GWS? Best regards, Sean.

   [David] YES. Most of the GWI patients have platelet activation from sources other than infection. So these patients react well to Plavix. CFS patients have infected bone marrows, so ASA or Plavix doesn't solve this type of activation. (See Fluffy's question for an extended answer)

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7. **[Kuby]Sed Rates**

   How often do you find an Myalgic Encephalopathy patient with a sed rate below 3 who does not have a coagulaton problem and how often does a patient with a sed rate of above 5 encounter

coagulation?

[David] We are writing a new journal article addressing the Normal Range of Sed Rates (ESRs). <5 test values are indicative of a hypercoagulable state. The only time this is not true is a cancer called Multiple Myeloma where there is a lot of extra protein produced by the cancer cells. In either case, because of the Soluble Fibrin or extra proteins, the RBCs cannot settle out of the plasma and thus you have rates of 0-4. The lower the Sed Rate, the more SFM and the more hypercoagulable the patient is !.

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8. **[KenL] Whey - an Alternative to Transfer Factor?**

   Non-denatured wheys, like Immunopro, appear to function in a manner similar to Transfer factor - but is significantly cheaper. Do you have any comments or have you investigated this type of product?

   [David] Both are from Cow's Milk. Both are extracts from the milk. Both have "flu like symptoms" / Herxheimer reactions reported at the start of use. It is an interesting concept and worthy of study. But I do not have knowledge to clearly answer this question at this time.

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9. **[DebbieSinKC] new protocol time lines**

   i don't understand the time lines - is it saying transfer factor for only 3 mos.?!?!

   [David] We will change our chart to DAYS on the time line instead of MONTHS to make it clearer. Thanks for the comment. {Editor note: Already done}

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10. **[karen]:If blood work results from a "standard work-up " are normal, can?you still justify ordering the ISAC panel?I.**
    I am very interested in getting the Isac panel but my doctors hesitate because they say there is no indication of blood abnormality in standard lab work that would justify persuing this avenue. Could standard work up be normal and ISAC panel still be positive. If so, could you explain this so that I could refer my doctor to your explanation? Are there patterns in normal blood work that correlate with positive ISAC oanel/ If so, what are they?
11. **[karen]:If my doctor ordered a hyperocagulability panel from another lab, would this have to be duplicated at Hemex to get the**
    My physcian was somewhat interested in the earlier information I brought to him on your work and wrote out a script for a hypercoagulability profile but did not specify Hemex or Isac panel. I did not get the test done because I suspect I need the specific Hemex tests but I have not yet discussed this with him. Can you comment on these issues in a way that will help me communicate with and educate my doctor to be sure Im getting a good evaluation regarding usefulness and specificity of tests? Im sick and ndot much of a biologist so this would be very helpful to me and probably to others.

    [David] Good questions and ones that I have not answered before. "Standard coagulation workup" would NOT show any abnormalities unless the aPTT was BELOW the normal range, which indicates a hypercoag condition, but docs are not taught this information. The ISAC panel is like 10 - 20 times as sensitive as the standard screening tests.

    [David] Most laboratories report a normal range for Fibrinogen of 200-400 or higher. The real range should be 200-300. Ours goes up to 315mg/dl. Most labs don't want to deal with minor elevations in results, so they increase the acceptable range a little. That is why patients with activated coag systems have minor fibrinogen elevations which are very significant to us but not to the physicians who routinely see higher normal ranges. The Prothrombin Fragment 1+2 test indicates that thrombin has been generated when this test is increased. This excess thrombin should be removed by AntiThrombin, which will give increased T/AT Complexes. There are

probably 12 labs around the country that can do these two tests, so they would not be included in the standard screening. The Soluble Fibrin Monomer (SFM) test indicates that the thrombin has converted fibrinogen to SFM when this test goes up. SFM is the culprit for FIBRIN DEPOSITION and increasing BLOOD VISCOSITY. There are probably only 5 labs around the country that can do this assay. As for the Platelet Activation test, this is our proprietary assay. We have learned so much from using this assay. If time permits later this year, we will submit our findings and methodology to a peer reviewed lab journal for publication.

[David] As to the hypercoag panel or Hereditary Thrombosis Risk Panel (HTRP), there are several labs that offer the routine tests in these panels. Certainly Antithrombin III (AT), Protein C, Protein S, APC Resistance can be done elsewhere. You should always ask for the "ACTIVITY" assay of these proteins. Do not let the lab substitute the "ANTIGEN" assay as it is not as sensitive as the activity assays. Remember that <50% of the patient defects are in this group (HEMEX 1999 stats = 47% in 300+ patients). Homocysteine is run routinely in many labs. The other 3 assays are more specialized. Factor II level or the Prothrombin Gene Mutation is rarely performed but positive in about 20-25% of patients. Lp(a) and PAI-1 defects have been found in 53% of our 1999 patient data base. These tests would be performed in maybe 12 labs around the country. So, all in all, send your blood to laboratories that specialize in this type of testing. Our technologists do these assays daily and are very competent in what they do, instead of a tech that might run these assays once a week or month in other labatories.

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12. **[Patti]:Started heparin 1 1/2 weeks ago.**
So far I haven't noticed any benefit from heparin (except warm toes :). I have had reallyl bad headaches. Could the headaches be related to the heparin? Also - I have really high PAI levels but allergic to bromelain and garlic. Would niacin be an effective way to reduce PAI? Also - do different labs have different norms for fibrinogen levels? I saw a result from a different lab that said a fibrinogen level of 400 was within "normal" range?

    [David] See my previous answer on fibrinogens. High PAI or Lp(a) patients are the hardest to treat. If you can't use bromelain, then niacin may be the next choice. Niacin is hard on the liver. Consult with your physician on this. There is a time release formula that is less toxic and hard on the body. I tried niacin myself, but I couldn't handle the vasodilitation (flushing effect). Give yourself time on the heparin. It takes much longer to see beneficial effects when a patient has a high PAI-1 or Lp(a), sometimes 2-3 months.

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13. **[Patricia]:Blood Tests**
Dr.Berg Thank you for joining us today. Are there any blood tests you would reccommend to our Drs. for us to have in conjunction with Hemex's blood testing?
14. [Patricia]:EBV & or HHV6a,b
Have you noticed patients with high titers or counts with EBV reactivation and or HHV6a or b ?

    [David] With the time line that we have proposed, knowing that one is positive for CMV, EBV, or HHV6 may be academic. It may cost less to go through the therapy of TF and antibiotics than getting these viral test performed. I do not know the cost or time to run these tests. Personally, I would want to know the data, so I would get tested. It is an individual choice.

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15. **[Kru]:I'm interested in sub groups of CFS**
Are you noticing anything about sub groups or sub sets of people that have CFS in relation to when blood thining works and when it doesn't? Or anything else about sub sets for that matter.

    [David] The two subgroups that we see are the genetic defects in Thrombin regulation (THROMBOPHILIA) or Fibrinolysis regulation (HYPOFIBRINOLYSIS). HYPOFIB patients are definitely harder to treat, since the process to clean up the vessels is inhibited by high values of Lp(a) or PAI-1. It may take 2-3 months for these patients compared to 2-3 weeks for

thrombophilia patients to get to equivalent points in relief.

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16. **[Patti]:Injection questions**
I ice my injection sites, but sometimes I get large bruises (2-3 inches in diameter) and other times I get small ones (~1/2 inch). Is there anything to be worried about with the large bruises? The injection sites on my stomach look much worse than the ones on my leg (very red), does this mean anything? How long should I wait until I "revisit" and area for injection? Can the top side of the leg be used for injection? About stomach injections, should you go above the waist AND below? How high above the waist? What can you do to make bruises go away faster?

[David] I don't have any good answer to these questions. Beth, our long term patient, has much experience on this. Contact her at pbdrechsel@msm.com. Beth's husband asked me about UBS! "*What*?" I asked. He responded - "*the Ugly Belly Syndrome*" !!!!I We all laughed. This is a small price for improved health. I am looking forward to 2001 when Emisphere has oral heparin available, because it will allow scientific study whereas today we don't have much. If you give heparin as an injection, it will bruise. What can we use as a PLACEBO for controlled crossover studies? The oral heparin will allow these crossover studies where the patient will not know if it is heparin or placebo. WE NEED THESE SCIENTIFIC STUDIES."

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17. **[Ruth]:dosage requirements**
Why does your suggested protocol have a 30mg Lov. morning shot and a 15mg evening shot. Are you stating that the half-life of Lovenox is up to 24 hrs? Or, is it thought that less dosage is needed during sleep intervals?

[David] LMW Heparin dosing is based on a body height & weight calculation. If the person is average height & weight, then 30 mg/day in the AM is a good prophylaxis level. If the person is over weight moderately or more, then a second dose at 15 mg given at night may be needed for the extra body weight. Heparin is a fat soluble product and a full dose may not make it into the blood stream if there is a lot of adipose tissue. Thus a second reduced dose injection for some patients.

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18. **[James Roberton]**:Are you familiar with the work of Professor Kakkar of the Thrombosis Research Unit in Europe?
Are you aware of any other research correlating with your findings? Professor Kakkar's team research on PWME has found poor blood circulation, reducing oxygen supply to the brain and muscles. The production of the normal blood thinning enzyme TPA is also reduced, as well of that of certain blood clotting proteins. Prof Kakkar advocates thermo regulatory hydrotherapy (TRHT), cold baths to the rest of us, as a treatment in CFS/ME to improve circulation and stimulate the endocrine and immune systems. What is your opinion on this?

[David] Last summer, Prof Kakkar called me from England and we chatted for some time. He told me that we had our manuscript accepted just before they were to submit theirs. Such is science. Anyway, there are only a handful of laboratories around the world that have capabilities to run these assays properly. His is one of them. Regarding his TRHT (cold baths) therapy, I am not familiar with the protocol to improve circulation as you state. It still seems to me that if a patient wants to improve their health quickly, rather than slowly over time, prescribed anticoagulants is the fastest way to improve it, not over the counter items or cold treatment.

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19. **[DebbieSinKC]**:blood thinning and TF
should those of us who started the TF (formula 560) without the blood thinning stop now, and do a month of bromelain? this is all so confusing . . . i have so many questions . . .

[David] NO. Follow your physician's instructions. Our recommended protocols are just that -

recommendations. Your physician has responsibility for you as his patient, not us. In regards to Dr. Brewer's protocol, don't change it! Follow it. If he wants to modify it, it is his prerogative to do such, not yours or mine. We (HEMEX) are consultants to the patient's physician.

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20. **[JamesD.]**:Coagulable State Fluctuations
I notice my blood becomes thicker, I get dizzy and faint more easily, bruise all over, and can't have blood drawn when I seem to be in an activated state of infection or partial, short relapse condition. Do you see the symptoms of coagulable blood fluctuate with infectious activities in many of your patients. What does such a fluctuation imply?

    [David] Active infections activate the coagulation mechanism. These relapses are the pathogen's way of creating an environment that the bug wants, usually an anaerobic environment. So it is natural to have increased SFM which increases blood viscosity and makes it difficult to draw a blood sample. Active infections also cause inflammatory reactions, which again triggers the coagulation mechanism. As the active infection becomes more dormant, there should be less SFM in the plasma. We have seen this repeated cycle in patients many times, from relapses several days apart to several weeks apart. It all depends on what the underlying pathogen is or are if there are multiple infections. I believe that HHV6 is the biggest player and should be treated accordingly.

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21. **[Annie]**:Autonomic Nervous System and hypercoagulable state
Is there any connection between multiple systems dysautonomia and hypercoagulable blood? Thank you.

    [David] In patients where there is no demonstrable pathogen, there is still a trigger to activate the coagulation mechanism, whether it is stress, trauma, accident, surgery, pregnancy, undetected pathogen, etc. The BASIC PREMISE is that the patient has a genetic protein defect !. People develop blood clots for many unknown reasons. We still have much to learn and this is a multi-system interactive process.

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22. **[NancyMcFadden]**:th1 (cell mediated /th2 (humoral) imbalance, relation to coag.
Last year i was tested by immunosciences lab, and my (th2) humoral immunity was definitely dominant over my cell mediated immunity (th1). in addition, my helper/ suppressor ratio (of cd4 cells) was high, which shows an immune activation state. have you found hypercoagulation seems to correspond to a th1/th2 imbalance and to a high helper/suppressor ratio? I know Nancy Klimas spoke about th1/th2 imbalance being common in cfids, last year in Connecticut, I heard it on tape.... if this correlates, then my doctor will be easily convinced to run these tests, so i appreciate your answer. Thanks! Nancy McFadden, Nashville TN

    [David] The problem with HHV6 as pointed out by Mr. Regush is that this virus is capable of altering many systems, including the immune system. I have had many patients reiterate the same comments as your question. If I were the patient, I would ask my physician to test me for both a pathogen and coag screens, including HHV6, Mycoplasma, Chlamydia, ISAC, HTRP and the B2GPI panels. Once I knew what the defects are and the triggering pathogens, treat all of it. The coag problem is only half of the problem.

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23. **[DebbieSinKC]**:treating pets
any ideas on how much bromelain or other blood thinners (NO ASPIRIN - i think it's poison to them) for our cats/dogs/birds. then how much TF? maybe i should just print off the HEMEX human protocol and ask the vet if it's alright to dose them, and how much . . . i don't want to take up limited time with this LOL - but in case many others are interested, thought i'd ask. we have 3 cats, one (maybe two) i'm sure has something similar to cfs/me/fm. i've never mentioned this to vet (think she might think i'm nuts), the cats always pass annual check ups. only way i can think of to get this stuff down a cat is mixing it in baby food.

[David] I know that pets can be effected by these pathogens and need to be treated. Your question is a good question, but I am not a VET, and I have no knowledge of small animal systems as a vet will. Ask your vet to look at these materials and then ask for a recommendation.

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24. **[Nelly]**:Rickettsia and hypercoag, the CFIDS missing link ?
Infections of the Rickettsia type have an affinity for the endothelium. Could these particular organisms (or close relatives like Coxiella Burnetii) CAUSE the hypercoagulation cascade you are describing ? Can the vicious circle hypercoag/infection be broken or do the people affected need to be on blood thinning medication forever ?

    [David] As I have stated in other questions, the underlying infective pathogen(s) must be treated for a good recovery. Any of the organisms can trigger the inflammatory and immune systems, which in turn triggers the coagulation system. Once the pathogen(s) are treated, stay on anticoagulation therapy for another several months to prevent a few organisms from becoming overwhelming again (see question 1 for additional information).

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25. **[wanda]**:Common CFS and atypical CFS--differences in hypercoagulability
Are you aware of the subsets of CFS and, if so, how do they differ with regard to blood disorder conditions?

    [David] Regarding subsets of CFIDS, see question 15 for Thrombophilia or Hypofibrinolysis sub groups. We don't get much feedback from clinicians in terms of patients, other than they are doing better or basically no change. I would like more information, but we don't have staff or time to process such information.

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26. **[Larry]**:Labs, anticoagulation treatment appropriate here?
Homocysteine, serum 9.3 UMOL/L (ref 0.0-9.0) Coagulation: Fibrinogen 260 MG/DL (ref 200-400) Protein S, Antigenic 105% (ref 70 or greater) Plasminogen, Functional 104% (ref 70-143) ERS 9 MM/HR (ref 0-15) Coagulatin INR 0.88 (ref .8-1.10) Prothrombin time 9.1 seconds (ref 8.8-11.8(Cardiolipin AB IGM 36 MPL Units (ref 20-80 medium positive) Anti Thrombin, Functional 132% (ref 80-120) Protein C, Antigenic 88% (ref 70 or greater)

    [David] I don't usually discuss patient information, other than to the patient directly. However, these lab data do have several interesting points. See Q 11 for fibrinogen. This reference range is exactly what is discussed in #11. Again, "Activity" assays are better than the "Antigen" assays because you may have a normal "antigen" level (amount of protein) with a not so functioning protein (decreased "activity"). What is missing in this workup is the Factor II level (screening for the Prothrombin Gene mutation), Lp(a), PAI-1 and Heparin Cofactor II levels. Lp(a) and PAI-1 increases (hypofibrinolysis) account for 53% of the defects we found in patients in our 1999 data base. Another concept that is seen in Larry's labs is that the AntiThrombin is ELEVATED. When there is a genetic defect (none has been demonstrated in Larry at this time), Protein C, Protein S or AntiThrombin will be increased in these patients as the body is trying to shut down the hypercoagulability due to the defective protein. We have seen this in around 30-40% of our patients that have such a defect and we are submitting a manuscript on this in the near future. So, Larry, get the rest of the testing done by a qualified laboratory.

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27. **[Patricia]**:Funding Research
A question from My Husband & Brother, Have you been helped with funding this besides out of your own personal monies and donations from The National CFIDS Foundation. Have you received any financial help or inqueries from the CDC,NIH and or any part of medical gov't branches?

    [David] The only funding that HEMEX has received for our research has been $4000 from Gail

Kansky and her association and $1000 from Dr. David Bell, when we were working on the original prospective studies in early 1999. HEMEX has paid the bills on over $45,000 of work in this research. We have not received any other donations except for less than $ 200.00 from sales of video tapes of previous lectures. There is a VERY REAL NEED for a foundation or such that HEMEX can bill for work done on sick patients that cannot afford to have the lab testing performed. We are not in a financial place that we can do the testing for free. If monies were available, then I know many patients could be tested from the requests that we have received to do free testing. These assays (specific antibodies) are expensive for us to purchase. We have lowered several of the fee schedule prices as we have become more efficient in some assays. But our direct costs are still very high. If anyone knows of such a foundation or grant, please let me know.

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28. [CARLSON] One of our members has a question for Dr. Berg. She is not on-line, so I am asking this for her. She is currently receiving intravenous infusions of gamma globulin about every 3 weeks to one month. I don't know how many grams she is getting, however. Her question is: would it do her any good to get the coag test since the gamma thins the blood? How long would it affect the blood after it is given? Could she get an accurate result with the coag test under these circumstances?

    [David] Gamma Globulin: Patient testing is relevant most of the time, except within 4-5 days of 1 liter of IV saline or solutions which are used at times for therapy. We want to see what the patient is like in a basal state if possible. If the gamma globulin helps in the therapy, then have the coag labs drawn a day or so before the next therapy. This will be the closest to a baseline condition. The ISAC panel is also good for monitoring therapy, especially after 1 month of heparin. We look for the SFM to be in the normal range and the fibrinogen to drop from the first draw. We also look to see if the platelets are showing signs of reactivation. The F1+2 should be going down and the T/ATs should be increasing in the normal range. By looking at 2 data points, we can fine tune therapies if needed.

SUMMARY:

[David] I trust that the combined information from last week and this week makes sense and is logical. The coag Paradigm Shift is that we should now treat patients with fibrin deposition as we treat patients who have had a blood clot. The new protocol on our web site (www.hemex.com/cfs), should help most CFIDS patients get back to almost complete health for under $3000, including lab testing, TF, antibiotics and physician charges. There will always be the coag protein defect in the patients, but once the infection is treated completely, then the protein defect can be monitored over time. When a relapse occurs, use heparin to control the infection quickly before becoming a CFIDS patients again. Remember, the longer one has been ill, the longer it may take to get rid of the HHV6.

My goal in this process of answering these questions was to get information to patients and researchers that is easy to understand. These answers may seem simplistic by design. I do NOT want to understate the COMPLEXITIES of these disease processes. There are numerous interactions that are not understood yet. As Dr. Triplett says: "There are many issues we don't understand. It's like a detective story, where people try to piece together the pieces of the puzzle. We're getting part of the puzzle put together, but we still have a big enough segment that we can't explain about thrombosis. So the story will continue to evolve, and the answers will become more complex." Dr. Triplett proposed the model that we have expanded about Thrombophilia or Hypofibrinolysis defects in 1997. I have attempted to give a model that in its simplicity is understandable and logical. Remember that the elephant can be described by many parts. It is dependent on what part is being examined at the time.

If people want to contact us by email, our address is clients@hemex.com or dberg@hemex.com . I

receive many emails a day and cannot answer all in a timely manner. Please be patient.

TO BETTER HEALTH ! David Berg.

Transcript 1197

- Speaker: Burton Goldberg, Lh.D., Hon. **"The Voice of Alternative Medicine"**
- Spent over 20 years carefully researching every aspect of holistic medicine, from California to Israel, Mexico to Russia.
- Is a leading spokesman for the rapidly growing field of alternative medicine; featured speaker for the Young Presidents' Organization, World Presidents' Organization, Commonwealth Club of California, National Health Federation, Citizens for Health.
- Published Alternative Medicine: The Definitive Guide in 1994. This 1100-page reference book, hailed as "the bible of alternative medicine," has sold over 600,000 copies.
- Published Alternative Medicine Yellow Pages, Natural Medicine Chest video and handbook, and You Don't Have to Die: Unraveling the AIDs Myth.
- Publishes Alternative Medicine, a unique consumer-oriented bimonthly health magazine.
- Featured guest on CNN, PBS, QVC, CBN, CTN, Global TV, CBC-TV, FOX, NET, Channel America, Voice of America, and many local, national and international television programs.
- Featured guest on National Public Radio. Participates in special fund-raising marathons, raising tens of thousands of dollars each pledge drive.
- 1997 - Published the hallmark work on cancer, Alternative Medicine Definitive Guide to Cancer, Alternative Medicine Definitive Guide to Headaches, and Alternative Medicine Guide to Heart Disease, Stroke & High Blood Pressure, the first in a series of soft cover books on various health conditions.
- 1998 - Published Alternative Medicine Guide to Chronic Fatigue, Fibromyalgia and Environmental Illness; Alternative Medicine Guide to Women's Health 1; Alternative Medicine Guide to Women's Health - Two; Alternative Medicine Guide: The Enzyme Cure.
- 1999 - Published Alternative Medicine Guide: The Supplement Shopper and Alternative Medicine Definitive Guide: Arthritis.
- 2000 - Published Weight Loss: Alternative Medicine Definitive Guide.
- Member of Advisory Board of Capital University of Integrative Medicine, which awarded him an Honorary Doctor of Humanities Degree in June 1999.
- Member of Advisory Board of The Sheppard Foundation.
- Member of Advisory Board of The Rolfing Institute. Served on the Board of Southwest Naturopathic College for four years.
- April 2nd, 2000 6 PM PDT, 9 PM EDT

Held at the CFIDS Town Hall (http://www.folkarts.com/townhall/)

Manuscript still to be proofed... answers were transcribed from verbal answers spoken over the phone -- so needs proofing.

[GOLDBERG] Just a reminder before we start, you may sign up on my website for my free newsletter - you will get discounts on books and lots of good new information.

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1. **[Lori] :** are there other supplements like Bromelain for blood thinning? Last week David Berg discussed Bromelain as an alternative to some prescription anticoagulants. Are there other supplements that work against other types of coagulation? Can you identify which Factors each effects?

   [Goldberg] Bromelain cleans the blood rather than thinning it. **WOBENZYME** is a wonderful product full of enzymes and herbals that can help clean out pathogenesis. Some of these are pancreatin, papain, rutin, trypsin, etc.
   [Support] Following are medical reports on wobenzyme: [1], [2],[3]
   Ingredients

Pancreatin	300 mg
Papain	180 mg
Bromelain	135 mg
Trypsin	72 mg
Rutosid	150 mg

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2. **[Lori]:** Cheaper alternatives to Transfer Factor? $275/month for transfer factor is a luxury that many cannot afford. Are there other substances that are rich in these substances?

   [Goldberg] You can purchase Immunity which is a form of transfer factor from Dr. Gary Gordon at (800) 580-PLUS. There are 60 tablets in a bottle and sell for $36.00 retail. This is a very fine product. Some specific transfer factors can be expensive and can have up to 8 transfer factors. Immunity is your best buy, I believe. We did a magazine article recently on the immune support. This is a very important new therapy.

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3. **[Ellie]:** what do you recommend for severe hypothyroidism? I have severe hypothyroidism, but have developed a real problem taking synthetic thyroid supplements. They now cause me to have terrible heart palpitations. Do you have a natural remedy that might help me? I am not able to take the supplements without a calcium channel blocker (Diltiazerm) which exhausts me further. Any suggestions would be welcome.

   [Goldberg] Go immediately to a holistic physician and get a prescription you can use. Go to our web site at alternativemedicine.com , use the search engine and type in thyroid. Read all the information and pay particular attention to Dr. Kellman as to the kind of testing. With a good physician, you probably won't need channel blockers.

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4. **[george]:** Dr. Poesnecker of Quakertown, PA has been treating CFS patients for adrenal exhaustion. Do you know if he is getting any results with his treatment?

   [Goldberg] I don't know him. Adrenal exhaustion is certainly a part of CFS. Read my book entitled **Alternative Medicine Guide to Chronic Fatigue & Fibromyalgia** or go to my web site alternativemedicine.com and use the search engine to look up Chronic Fatigue System. Also read about Lyme disease.

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5. **[Anne]:** What is the best alternative way to treat both mycoplasma and HHV6 infections?

   [Goldberg] The best way is with a serum made from your blood feeding the bad mycoplasmas. This is being done Mexico with great success. The San Diego Clinic should be able to give you some assistance/information.

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6. **[kuba]:** Olive Leaf Extract as a Therapy? I have heard mixed results with using Olive Leaf Extract. Is this a good therapy or just hype? If it makes you feel bad at first should you keep using it?

   [Goldberg] No. What is the problem that causes you to use it? Go to our web site at alternativemedicine.com, use the search engine and type in Olive Leaf. There is a lot of information printed there. If you're not feeling good using Olive Leaf Extract, try changing brands. Consider electrical dermal screening. Consider taking **WOBENZYME**.

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7. **[Odie]:** I'm allergic to Bromelain and have thick blood (coagulation). I will not do injection of blood thinners and I cannot take aspirin. What can I take? What type of blood thickening does it effect?

   [Goldberg] If I were you, I'd run to the nearest holistic physician and find out the cause. Consider taking **WOBENZYME** or Olive Leaf Extract. You drastically need a special diet-one of mostly vegetables for a while with not much protein. Thick blood can cause cancer, heart disease and lots of other things "coming down the pike". In your case, **DO NOT SELF-MEDICATE!** Run, don't wait to the nearest holistic physician and work with him or her to heal yourself.

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8. **[kuba]**: What are the benefits of apple cider vinegar?
   Why do all the 'alternative' specialist suggest I take apple cider vinegar? What are the benefits of apple cider vinegar?

   [Goldberg] Basically it is to balance pH. Apple cider vinegar changes from acid to alkaline in the body. If you are too acid it will alkaline in the body.

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9. **[zoe]:** What would be your top two herbs for hypoadrenal gland support?

   [Goldberg] Adrenal Cortex Extract. B5 - 500-2000 mg (should be muscle tested). 5000 mg of Vitamin C. RNA.

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10. **[John]**: Which type of B12 is best? Is there a risk with cobalamin?
    There has been debates on some lists about the US version of B12 being cyanide based and thus unhealthy in the large dosages that are often suggested. Is there a US source for B12 tablets that is not cyanide based.

    [Goldberg] There is no risk. At 73 years of age, I personally take 1 cc of cobalamin a week. You are better with the injected B12 - it is far more absorbable.

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11. **[BEA]**: Your experience with treatments for active Epstein Barr Virus
    Having this specific type of CFC, I have been taking vitamin suppliments, Co-Q10, collostrum, glucosamine (already had arthritis) and methylcobalimine (B12) shots. Several symptoms have improved. Memory, vertigo, nausia and arthritis are better now but the fatigue, and that as a reaction to any small stressor, is still severe. Wondering what you recommend as well as what

you have seen as an average recovery time line.

[Goldberg] Check for Lyme disease - it is almost always a component of this disorder. Although you may not test positive for Lyme disease, it is still a component. You need to find a holistic physician. Check the list of them on my website. I did a book entitled Alternative Medicine Guide to Chronic Fatigue & Fibromyalgia or go to my web site alternativemedicine.com use the search engines for Chronic Fatigue & Fibromyalgia. You will read about it there. Not only is EBV causing these illnesses but is a major cause of heart disease.

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12. **[babawawa]**:Toxic Mold Exposure
After exposure to stachybotrys and other toxins at my office, I began to have difficulty clotting, while CFS victims seem to have the opposite. Should we be following the same treatment protocols? If not, what differences?

[Goldberg] .. First get out of the mold. There are ways of handling the mold. You must have your office completely clean of the mold. Filtration systems like negative ion generation. Go to my search engine and look up: NAET - Nambudripad Allergy Elimination Treatment. It's a non-evasive technique, the patient holds the substance and the doctor applies acupressure. Until you get out of the mold, the mold can cause horrible depression and mental aberrations.

13.] **[Fluffy]**:I hate swallowing pills. Is there an alternative especially for mag/mal ?
I need to take magnesium glycinate and malic acid combination. What I take also includes b1, b6 and tyrosine. It seems my body does not absorb it well or my body utilizes a lot. Maybe this could also be due to the blood coagulation factors since I have at least 4 abnormal coagulation results in my tests from Dr. Berg. This helps greatly with sleep and with many other things. Cheney recommended the glycinate form of magnesium since the regular magnesiums will negatively affect the brain by pushing it more toward micro seizure activity. I need to swallow about 15-20 capsules every day or so. Injection is literally a real pain and inconvenience but not sure about it's effectiveness. 18 capsules equal about 1300 mg of magnesium glycinate and 3600 mg of malic acid I have been searching around for an alternative for a while but have not found any yet. I do take antibiotics and they usually recommend 1 hour before or 2 hour after. I try to take it at least 6 hours after the antibiotic to avoid interference with the antibiotic since I don't think 2 hours is enough and this is also a real inconvenience also. Also I don't mind swallowing some pills so the fewer the better. Also, in any alternative, include the cost and efficacy factors. Also, is there anything else that would be more effective. Could you discuss all these questions and concerns that I posed.

[Goldberg]: If you are severely deficit in Magnesium it is essential for injections. It is easy to learn these injections without hurting yourself. Understand that Electrodermal screening or muscle testing to determine exactly what your body is screaming for. See "Fatigued from an Underactive Thyroid" on my site.

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14. **[Patti]**:compromised immune system
Many people with CFS have extremely low cell-mediated immunity (CMI). I also know that low CMI is highly correlated with cancer and I'm concerned about that since my Natural Killer cell function is still very low in spite of taking b 1,3 glucan, IP6 and various other immune system builders. I read about a test called the AMAS which is supposed to detect cancer (from anti-malignin antibody) anywhere in the body. Are you familiar with this test? Do you think this is a cost effective

[Goldberg] I do not trust the AMAS test. If you have advanced cancer it is worthless. It may be helpful with early cancers. I've been disappointed with it. The best test to detect cancer is the

Biological Terrain Assessment (BTA) - it tests salvia, etc. The DARK FIELD microscopy test - you can look at a drop of blood with a microscope - with a competent person you can see disease coming 2-5 years a head.

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15. **[Patti]**:Colloidal silver
In your experience, how effective is colloidal silver in fighting intracellular infection? Are there any types you recommend over others?

[Goldberg] There are different forms and different intensities. Use muscle testing to find out which one is best for you.

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16. **[Patti]**:Oxygen
In your experience, how effective are oxygen therapies? There are portable mild hyperbaric chambers that are currently used for high altitude sickness (like the Gamow bag). Are you aware of anyone using these for conditions like CFS?

[Goldberg] The hyperbaric portable unit does not give a deep enough dive. Our doctors recommend the use of hydrogen peroxide, vitamin C drips and ozone ... CFS is reversable -- again look at Lyme disease.

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17. **[Judy in MI]**:Do you also recommend treatments such as stretching, massage, accupressure, etc. or just supplements, herbs, etc.?
I also have Crohn's disease (symptoms begain 1975, Dx'd 1976, resection 1978). I find I am very sensitive to most meds, and there is no reason to exclude supplements and herbs with these. Anything I put in my mouth and thru my GI tract is a potential aggravation to the Crohn's. I've had my most success getting relief from FM and CFS w/gentle stretches for myofascial pain syndrome learned in physical therapy, massage, percussion massage and some gentle osteopathic/chiropractic manipulation. Hypnosis for relaxation also helped. Though I do wish I was more receptive to hypnosis. In the past I've tended to be an "adrenalin junkie" which is something I've had to 'unlearn' and it hasn't been easy! Learning to pace myself was one of the hardest lessons to learn! Its been said symptoms of stress don`t just stop when stress stops either. Giving up a small daily dose of adrenaline is like coming off medication, it has to get out of your system and can take months to do so.

[Goldberg] Get my book, go to Gastrol Intestinal chapter and read it. L- glutamine with a low carbohydrate diet. It is important to take easily absorbed nutrients. Take Olive Leaf Extract and **WOBENZYME** it.. It is fine to do transfer factors and colostrum to build your immune system. You must nutritiously build up your body. Breathing exercises, chiropractor are essential in this process. The adrenals become depleted from stress.

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18. **[Carol Mahoney]**:Mercury and CFS
I've done lots of research on amalgam fillings and their relationship to CFS. [I've also read the section in your book on this topic.] Has any new research come out on the mercury/CFS link? Also, different practitioners have different theories on removal procedures, detoxing, etc. What's your own personal view? And what's your experience with outcome--how many CFS people have improved after amalgam (and to what degree), and how many saw no improvement? Thanks!

[GOLDBERG]: It is inconceivable that the dentists of America are stupid enough to use silver filling to this day that are 50% mercury. If you child broke a thermometer in his mouth , you would have him in the hospital immediately. Dr. Hal Huggins tells his view on protecting the patients and the doctor, once the filling is out of the mouth. You must then remove it from

the body of the patient. One of the most effective is Eccomer - a sharp liver oil processed in Norway. The Oxiglycerial coats the and takes it out of the body without disturbing the kidney and liver. After the removal, most patients feel better. Other problems is electrolytic action between other metals (gold, silver, mercury). Another problem is prior teeth removed or root canals where there can be other infections.

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19. **[Judy in MI]**:Could you tell us more about serum from our own blood to fight infection such as HHV6, etc.?
I assume you are talking something like an autogneous bacterim?

    [Goldberg] Yes, Autogneous blood can very well boost your immune system in this country as well as Germany. What you want to look for in all of these diseases is the mycoplasma involvement. The results of the mycoplasma infection can be seen in the Dark Field Microscope. See San Diego Clinic or Biopulse which should be able to give you some assistance/information.

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20. **[Patricia]**:Water
Hello Dr.Goldberg, Thank you for joining us tonight. Is it better too snip water during the day or drink several glasses at different times of the day? At meals do you advise to drink during the meal or before and after meals?

    [Goldberg] You must drink a lot of water during the day - without ice. Sipped. The water must be filtered, filtered, filtered! The quality of water in the US is a major source of disease

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21. **[Mike]**:Effects of abx on natural immunity
Are there any dangerous side-effects, in your opinion, to the use of long term tetracycline antibiotics? What would you suggest to counteract any problems from anti-biotic use, should abx. be needed. Which types of patients do you feel should avoid antibiotics if any?

    [Goldberg] In certain life threatening conditions, one may need to take antibiotics. With the antibiotics you also must take probiotics this will help neutralize the negative aspects. A product called "Oleuropein" (Olive leaf extract) and IV Vitamin C. Transfer Factors, thyrmic extracts, kyolic garlic, colloid silver (used prior to the discover of antibiotics in 1935). See my website for other powerful natural antibiotics.
    One of the problems with beef is that the fats are loaded with antibiotics used to fatten them up. We end up with all of these poisons in our bodies. This is one of the reasons for the epidemic of chronic illness.

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22. **[Patricia]**:Blood Volume
With the info on blood volume, what would be the best way to increase volume.

    [Goldberg]: Our doctors advise you to go heavy on vitamin E, A, C folic acid and heavy on B-vitamins, WOBENZYME.

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23. **[Judy in MI]**:How is one tested for adrenal gland function?

    [Goldberg]: Muscle testing.

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24. **[zoe]**: Should patients try to get sun exposure for Vitamin D? Cost to benefit?

[Goldberg]: I get some sun every day on the forehead primeval gland. It has many other benefits.

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25. **[Patti]**:Biomeridian
My doctor started using the Biomeridian machine. It measures the state of various acupuncture pathways and then comparing the effect that various supplements and drugs on your current state. Are you familiar with this testing and how effective do you think it is?

[Goldberg]: Any doctor who does not use this type of equipment is ignorant. The machine uses the meridian system known to the ancient Chinese. Can you imagine an energy system in the body that has been charted by French scientists using isotopes. It is no different than using a stethoscope. Read about it on my website. It is one of the major tools of holistic medicine.

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26. **[David Newton]:**Muscle testing
Being a Chiropractor, I am familiar with muscle testing but find it too subjective. Is there a particular method you use that has a more objective outcome. For example the varying resistance of the patient, the pressure of the practitioner. The expected outcome of the practitioner. Also, due you know a clean source for obtaining ACE.

[Goldberg]: David, they have been using muscle testing since ancient Egypt. Hieroglyphs showed this technique. Make sure that the patient being tested is not on a geopathic zone (see my site). Send me an email on ACE (my email is burton@alternativemedicine.com). You are my type of doctor! :-)

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27. **[Nancy Bzzz...]**: AIDS/CFS/MS etc. and contaminated polio vaccines
Are you familiar with Dr. Howard Urnovitz' findings suggesting that the presence of antibodies to retrotransponsons may provide a window into the advancement of chronic diseases? Also about his findings that polio vaccines have been contaminated by simean viruses resulting in "new syndromes"? If so, what are your thoughts on his theory? What are your thoughts on the origins of CFS?

[Goldberg]: It is true - the main cause of CFIDS is the overuse of antibiotics that brings about estrogen dominance caused by the pesticides in our animals and vegetables. This is also responsible for earlier puberty in girls. This estrogen dominance is why breast cancer is now 1in 8 back in 1915 it was 1 in 20.

In 1900 1 in 33 had cancer of any form, it is a lot higher today. The denuding of the crops by farmers tossing chemical at them for the sake of the quick buck has resulted in essential minerals not being present. Keeping animals alive in filthy conditions and then using antibiotics further compound matters. Read my website, the answers are there.

AIDS is not HIV - it is an impugned immune system. see my book on AIDS, **You Don't Have to Die: Unraveling the AIDS Myth**

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28. **[]What is the significance of extremely high titers to CMV, very high titers to EBV & equivocal titers to HHV6 if the IgG and IgM are within normal ranges??**

[Goldberg]: Your immune system is functioning well, these virus can be eliminated with Transfer Factor and NGN3 - a mushroom that produces natural killer cells as well a Bioprobe thymic A (pricey but devastating against pathogens, I always carry some with me).

29. **[]**:I recently read THE RIVER by Hooper and he certainly presents a strong case for the polio vaccine contamination.
Given Hilary Johnson's reporting in OSLER'S WEB and the current NIH/CDC scandal of diverted, misallocated and missing CFS and Hanta virus funds one really has to wonder about governement research, government sponsored research and just how much we can believe our government health agencies.

[Goldberg] Go to my home page and read my controversial essay on Gulf War Illness.

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30. **[Paul]**:Could you explain what the Mycoplasma Vaccine is exactly?

[Goldberg] Mycoplasma are cell wall deficient, simply put, this means that it changes its form. There are over 3000 types of mycoplasma - 50% good and 50% bad. The mycoplasma vaccine is a vaccine made from the patient's blood, producing polysaccharide (like feeding a bad dog a bone so your immune system can wipe them out). It is one of the marks of a master holistic physician.
It is in my cancer book.... and I'm doing a story on it in the next issue of my magazine (If you subscribe to my magazine for $20 you can get one of my paper back books (<$20) for free).

[Support] Some additional reading for some [1]

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31. **[Nancy Bzzz...]:**Alternatives for Neurally Mediated Hypotension
Any suggestions on alternative treatments for NMH symptoms?

[Goldberg]: Breathing (see [1],[2]). Garlic, Hawthorn berry, B-Complex, C, E, A and CoQ 10

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32. **[Mike]:**Why take TF and whey
What different effects does TF have in the body from whey powder

[Goldberg] : Transfer Factor (TF) kick starts the immune system. Whey repairs the gut. Speaking of whey and its effects - you need 2-3 bowel movements a day. This is essential for maintaining good health, if you are not having this many bowel movements, see some of the articles on my site [1],[2],[3]

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33. **[Larry]:**Wobenzyme
Which Wobenzyme do you recommend, the Wobenzyme med or Wobenzyme N?
34. **[Goldberg]** They are the same thing - one is labeled for physicians and the other for retail. God bless.

END OF TOWN HALL

Regush #1

Transcripts

Town hall meeting

[Return to Town Hall Logon]

Nicholas Regush

Virus Within : The Coming Epidemic

- http://www.amazon.com/exec/obidos/ASIN/0525945342/o/qid=953597284/sr=8-1/102-3367457-6160035
- http://shop.barnesandnoble.com/booksearch/isbnInquiry.asp?userid=4MZVC4VJVH&mscssid=AVUE5WSPXWSH2JKJ001PQUW8MEGFAPS7&srefer=&isbn=0525945342

Bookcover

March 19th, 2000, 7pm PST, Attendance: 430+
Support:*Welcome Nicholas Regush, there are many here who have read your book and have many questions.*

[Early Birds]:Brain lesions. If a pt has viral CFS symptoms and lesions, what can the pt do [pat fero]:Brain lesions. If a pt has viral CFS symptoms and lesions, what can the pt do Would you comment further about the need for collaborative studies on MRI and HHV6?
Nicholas Regush:*The possibility of studying hhv-6 and MRI is something that must be done - but lack of funding prevents the proper type of collaboration.*

[Early Birds]:Did you research Sidney Grossberg's work(at the University of Wisconsin?)? If so, what do you think? I have just finished his book--I liked it a lot--and it covers most of the HHV-6 research that many of us already knew quite a bit about. Your reporting on Knox and Carrigan was particularly thorough and interesting. From rhbailey@catskill.net (CFSFMExperimental)
Nicholas Regush:*I'm not impressed with S. Grossberg's claims for a virus. The work needs to be published and if there is an actual virus, then it must stand up to proper scrutiny. I think this entire field of research (CFS research) needs a major credibility boost - and that is why I focused on Carrigan and Knox. Too much money from CFS patients has gone to research that has lacked appropriate scientific controls and conceptual development.*

[Mike C]:Do you have any standard treatments for people with CFS?
Nicholas Regush:*There are unfortunately no standard treatments for CFS. Mostly, there are interesting leads, including the use of certain anti-herpes virus drugs and immune boosters but for the most part physicians must make careful and informed decisions about what to try on patients - with full informed consent for those patients. This again is why I think the HHV-6 research is promising because the next phase will probably bring some important clinical trials into play.*

[April]:Orthostatic intolerance You noted that Carrigan's work with HHV6 showed that the virus interfered with the bone marrow's ability to produce new blood cells. Do you know if the researchers have considered HHV6 as a cause of low blood volume and orthostatic intolerances in PWCs? What are your own thoughts on the possibility?
Nicholas Regush:*Carrigan and Knox certainly show that HHV-6 can affect propduction of new blood cells but their work has centered on primarily on identifying factors in the breakdown of production and not their relationship to clinical problems; so I'll also have to pass on this one because there is no data available that satisfies me.*

[April]:HHV6-A vs. HHV6-B Please explain the difference between HHV6-A and HHV6-B. Are both strains common in the general population? I've read that AIDS and CFS patients have the A strain in common, and that CFS could be considered non-HIV AIDS. Comments?
Nicholas Regush:*Variant A and B are suffiently different from one another to almost be seen as different viruses - not quite, but you should get the picture of enough variance to probably make a difference, perhaps even in the way that they are transmitted. We know that B is extremely common because the serology has been done; A is up for grabs in terms of just how many people contract it; there is confusion in the literature about who gets it. Regarding the AIDS question, I personally look at illnesses as interlinked. Some have common features as does CFS Sand what is commonly termed as AIDS, with obvious differences in terms of extreme illness. But I have long argued that if we are ever to understand CFS better, we must get rid of the idea that this construct - i.e. CFS - is something unto itself. The body is not static. It is a dynamic entity, interacting internally to the outside world - the dynamics are poorly understood, and that includes how the immune system is affected. As time goes on, we shall probably see more common pathways being exposed in a number of*

diseases, with viruses like V-6, (one or both variants) playing a trigger-like role in setting up a series of bodily events. Chronic illness will be more complex than many researchers like to suggest it is. We try too hard to find single evil events and sin

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[April]:Blood donations There's a growing movement among PWCs to give their intent to give blood (see www.geocities.com/pledge_now/) as a way of protesting the NIH/CDC not taking the infectious threat to the public seriously. Many have even claimed that during recovery periods they have donated blood with the full knowledge/approval of the Red Cross. Do you think blood donation by persons with active HHV6 infection is more dangerous to the blood supply than healthy people with a latent infection? With the virus latent in most everyone, would it even be practical to screen for it in the blood supply?
Nicholas Regush:*In short, yes it would be practical to screen for HHV-6 in the blood supply.*

[Marisa, San Francisco]:recent blood work shows transit from bac infection to viral infection (I have activehhv-6/ c.pneumoniae/parvo b19/EBV My immunolgist was obviously thrown by this, and could only hazard a guess that two of the viruses I have been diagnosed with have "joined up" (his owrds) I wonder: do KNox and Corrigan have any thoughts on HHV-6 in conjuction with other active infections such as these... I have received your book but have been too sick to finish more than a few early pages, my apologies if this question is answered int eh book....
Nicholas Regush:*Knox and Carrigan believe on the basis of their unfolding CFS research that HHV-6 and EBV may work together, and that active V-6 reawakens EBV. Also, there is no reason to believe that other viruses may also become involved at some point in the multi-phase process that leads to chronic illness. WE are only at the very beginning of our understanding of how this all might work. WE also need to learn more about how viral triggers can affect our host genetics. Research in this vein is beginning to show some idea that our genes can even get reshuffled as a result of viral hits.*

[Marisa, San Francisco]:Your thoughts on what the cfs/gws/etc patient pop can do to change the ignorant course at NIH/CDC The rectn years have shown the course to be of littlehope for the patient population and I fear that due to enormous discord within the population that internal fractures lead the NIH CDC tothink we cannot accomplish much, do you have thoughts on what course of action the patient population might take to raise conciousness there and with Congress/Media?
Nicholas Regush:*Unfortunately, discord in any "illness" grouping becomes common, particularly as research and promises fail. People must come to understand who the real enemy is - and it's not people who are ill. The enemy as I see it is bureaucratic stupidity on a massive level; one way to fight it is through strong coordination of efforts. Congressional committees are beginning to show some real interest in coming to terms with issues about chronic illnesses. CFS could be very much in the spotlight. I am doing my best to try to make this happen.*

[Fluffy]:Do you really believe the government is seriously interested in finding treatments for these diseases AIDS,GWS,ME/CFIDS,FMS,MS,LYME,MCS,PPS,Agent Orange If you think so, please state evidence
Nicholas Regush:*It doesn't work that way. Government officials listen to scientists who traditionalloy have maintained old boys networks of funding and advice-giving. These networks become entrenched and try to protect themselves at all costs, often referring to those who question orthodoxy as flat earthers or whatever. I for instance find it appaling that Straus is still emmersed in this idiotic psychobabble theory of cfs. This has been maintained, in part, because so much bad research has flooded CFS. Sorry to say that, but I'm convinced a lot of people that groups such as yours may have funded dhave been led down the dogpath by ineffectual researchers who promised much too much. They then become fodder for networks functioning at the government level. This is why I wrote a book focused on Carrigan and Knox. They do terrific science. It is well-published and involves the appropriate methodology. Too many CFS researchers are using unsuitable research probes, including PCR and serology when they should be trying to isolate virus. In the case of CFS, cultures show evidence of virus before antibodies even appear to HHV-6 - if they in fact ever appear. To continue to do research with antibody testing and pcr solely is bordering on defrauding cfs patients. This is just dumb science.*

[NancyM]:McMinnville commentary- nejm hysteria Off subject, but you wrote a commentary on the bogus nejm article of 1-13-2000, and i wrote you an email never hearing back, where i actually had proof that their article was inaccurate to say the least. I now have the epa documents, showing very poor building maintenance, poor air quality, a host of problems. Do you want me to send them to you at abc, if so, let me know where to send them.... This is relevant to cfids, due to the editorial accompanying the nejm "research" by simon wessely who has disparaged cfids as hysteria in the past... I live in Tennessee, so that explains how i have the data in part. Nancy McFadden
Nicholas Regush:*Anyone who wants to send me materials at ABC News should sent them to me at 47 west 66th street, NY, Ny 10023. Sorry not to answer your e-mail. I get up to three thousand every week in regard to my column. I just can't keep up.*

[Sophie]:HHV-6 - is it best to try to treat active HHV-6 infection? If so, what treatment do you recommend? If HHV-6 is just an opportunistic infection, perhaps it will just reoccur after treatment. Perhaps it is not that "causitive" in CFS. Maybe you DO consider it a causitive factor in CFS. If you feel treatment is useful for long term, what is the best treatment - gancyclovir, etc. (most of these are high-powered anti-virals with some significant side effects)
Nicholas Regush:*Sorry, not sure what just happened but I got kicked off in mid-stream. I was answering Sophie. V-6 may not just be opportunistic. Carrigan and Knox show that it can cause progressive disease - and quickly. There is a lot we don't know about Variant A and how it is transmitted and how it does its dirty work. For a virus to be considered "causative" requires a lot of proof. We need more. But the preliminary evidence is a red flag. It is active in the body when CFS patients have symptoms. There is more viremia when patients, for example, complain of neurological effects. Remember, this virus has a thing for nerve cells as well as immune cells. Gancyclovir used intravenously seems to help some*

patients with MS. Oral gancyclovir combined with an interferon or immune booster might help some; b ut I want to emphasize that we are far from understanding what is going to work because

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[pat fero]:Infant death due to HHV6 transmitted at birth. Health status of mothers? Transmissability through birth, breast milk, blood is an issue I am concerned about.
Nicholas Regush:*There is little or no data on the health status of mothers whose children have a hard time with HHV-6 in early life. In some cases, the virus has run through children very powerfully early in life, but we don't understand why one child and not another.*

[pat fero]:HHV6 kills natural killer cells. Why no collaboration on HHv6 and NK research?
Nicholas Regush:*Good question. When a virus has the potential to destroy so many components of the immune system, clearly the scientific community has its head in the sand.*

[Fluffy]:Aren't you sanitizing ("differences of opinion" and "bias") what is actually an assault against the chronically ill If I kick you in the balls once, I could just say I made a mistake and you may believe me. If I kick you in the balls twice, I can say I made a mistake. You may suspect otherwise. If I kick you in the balls a third time, I can say I made a mistake. You can say I intentionally did it. We now have a "difference of opinion" and some "bias" is'involved where I place my foot. The chronically ill have been having their balls kicked in by the federal government for decades. How many more kicks do we have to take before it dawns on you that there is an actual violent assault taking place You call the problem "differences of opinion" and "bias". I call that view extremely naïve at best.
Nicholas Regush:*Well, don't get pissed off with me. I'm not the enemy. If you read my second opinion column at ABC, you can get the full flavor of what I believe and how I put my career on th*

[pat fero]:Explain the signficance of red cells, bone marrow and HHV6... Could HHV6 infection explain red cell wall abnormalities seen in ME/CFS?
Nicholas Regush:*HHV-6, according to research by Carrigan and Knox has the capacity to disturb the bone marrow's normal production of blood cells. Re: red cell wall abnormalities. Could be. The research needs to be focused on this.*

[pat fero]:Please make suggestions for further research and discuss funding of such efforts. Scientific bias against innovative research is clearly demonstrated by the NIH and CDC meaning that"safe" research is being funded. With this approach, you can kiss goodby the ideas in THE VIRUS WIHIN. What can pts do?
Nicholas Regush:*Well, some people are not going to like my answer to your question. I think that each CFS group around the country has to come to terms with the unfortunate fact that you have been supporting too many researchers that are not delivering. Too many experts out there that are taking you for a ride. I mentioned earlier that this PREVENTS recognition of real scientific work in CFS. I read the overall literature and frankly I get appalled at the slovenly science. This is a major problem that you will have to deal with before much else gets done. Some people in CFS groups obviously can read the science and should help out to point out that some people - particularly Carrigan and Knox - are getting it right because they don't take stupid short-cuts and can defend their science when under attack. Yes, the ideas in my book will be kissed off if the right people don't get the funding. I am going to do a major investigative report in the near future on the state of the science - separating the bs from the good stuff. Too bad for those who have been given an easy ride. I think the CFS community has to get more serious about where it puts its money.*

[pat fero]:Is there information in the book you have since found out to be questionable? One is never "finished." To do over again, would you add or change information? What?
Nicholas Regush:*No, there's nothing in the book that I would necessarily change, but I will continue to track this story and continue to update my writing on it. I choose issues when I feel I can have an impact. I plan to have an impact.*

[Ellen]:What do you think of possibility HHV6 is involved in cases of spinal stenosis and Chiari formation? Is it possible that CFS or what causes it also causes brain swelling in some and that the surgery merely relieves the symptom?
Nicholas Regush:*I'm leery of the surgery being proposed - but that's because I have not seen any data from that surgery center. It bothers me that they haven't published.*

Server crashed - end of townhall

Transcripts

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Nicholas Regush

Virus Within : The Coming Epidemic

- http://www.amazon.com/exec/obidos/ASIN/0525945342/o/qid=953597284/sr=8-1/102-3367457-6160035
- http://shop.barnesandnoble.com/booksearch/isbnInquiry.asp?userid=4MZVC4VJVH&mscssid=AVUE5WSPXWSH2JKJ001PQUW8MEGFAPS7&srefer=&isbn=0525945342

Bookcover

Support:*We have a lot of questions today. Take a few minutes to vote for 2 or 3 questions to identify which questions are most important to you.*

[Laurie Lassesen]:Oleuropein and HHV6A Burton Goldberg suggests that Oleuropein (found in Olive Leaf Extract) is effective against the HHV6A virus. Have any researchers verified or discounted this?
Regush: *Hi. Thanks for having me back. No one that I know of has verified this claim*

[bui]:How do you feel about all the unproven claims that are being presented as fact about HHV6A? Here is one. "The basic thing you should know is that nearly all ME/CFIDS patients have a virus called HHV-6A and inside that is a retrovirus that one researcher has named the JHK virus" The National CFIDS Foundation, National Forum, Winter 2000
Regush: *I've heard a lot of good ones. This is totally nuts.*

[Cfbon]:To me, Urnovitz's "reshuffled gene" theory and Martin's mutated "stealth virus" theory sound similar? Agree? Urnovitz speculates about endogenous retroviruses have genes "reshuffled", while Martin talks about mutated viruses with segments of other viruses, bacteria, and maybe even fungi, inserted in the gene sequence. Other than etiology (coming from inside or outside of the body), aren't these two ideas conceptually similar? How do they relate to HHV-6?
Regush: *Urnovitz is looking at various ways environmental "hits," including viruses can damage cells in a way that cause gene reshuffling. John Martin's views which I detail in my book speaks to possibilities of recombination, based on the notion that there is a simian herpes virus that serves as a platform for this process.*

[Jerry G.]:Knox, Carrigan and JHK Sydney Grossberg's patent on the JHK virus also lists Drs. Knox and Carrigan as co-discoverers. I'm only halfway through "The Virus Within" but have noticed no mention of their work with Grossberg and JHK. Can you share any information of their involvement with Grossberg and JHK and their feelings about JHK's involvement in diseases such as CFS?
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Regush: *Knox and Carrigan never agreed to have their names assigned to any patent - with Grossberg. They are not too happy about this development. My own view of Grossberg's work is not very appreciative - in other words, I don't feel there was any reason in the world to include his "discovery" in my book." JHK is a*

phantom as far as I'm concerned - and a very expensive one at that.

[GK]:Why do you think the media has a hard time reporting on CFS? Is it the Name? When they actually try to do a good report, They seem to focus on just being tired. Plus, they always say "chronic fatigue", in place of CFS or CFIDS. Its insulting to use any of these names, but even worse to use describe the illness as "chronic fatigue". "Chronic fatigue" is a symptoms, not and illness.
Regush: *Medical media in this country is in big trouble. Too little time, too few brain cells ticking and generally speaking - lack of imagination and effort to do homework. CFS got off on the wrong footing with this "yuppie flu" business and unfortunately because of NIH ignorance got locked into that.*

[DebbieSinKC]:testing for active hhv6 any chance of a quicker, cheaper test for the active hhv6 that can be done in dr's office - like the mono "spot" test, or the 10 minute throat swab strep test (even though these may not be entirely accurate).
Regush: *Maybe, but not yet. Such a test won't be around for a while.*

[JT]:National CFIDS Foundation and The CFIDS Association of America Dear Sir, Of the science endorsed by these two organizations, which in your careful estimation is producing more useful information for "CFS" sufferers.
Regush: *I honestly can't answer that question. I do think, however, that any future funding of so-called retrovirus research should be placed at extremely low premium.*

[Renee]:Would irradiation of the blood help with some of the fatigue we suffer with? Thank You
Regush: *I can't see what that would do, frankly. I hope no one is suggesting that without proper research.*

[Renee]:Sorry!! I have been diagnosed with unknown virus in bloodstream and Epstein Barr at very high titers . Plez add to other
Regush: *diagnosis with an unknown virus? how does anyone know it's a virus? is there evidence for this? and what kind? Re: EBV, high titers may suggest there is a problem but lab standards differ here and there are individual differences - so don't jump to anhy conclusions.*

[Bob B]:Possible TV coverage of CFIDS and HHV6A ABC (on 20/20) covered the "brain surgery" for Chronic Fatigue and FM. Is there a chance you can get them to produce a segment on a major show about HHV6 and it's link to CFIDS, AIDS and MS?? We really need the main stream folks to "wake up" and take CFIDS seriously. Thanks
Regush: *20/20 passed up an opportunity recently to do a show on hhv-6. not much I can do about that.*

[Cfbon]:What is the difference between the Carrington/Knox blood test for HHV-6 and the standard antibody /PCR tests? In your book, you criticize the use of PCR results in HHV-6 research and state that Carrigan and Knox use a better technique. What is their test, and how does it differ from PCR? Does their test involve culturing the virus? If so, don't cultures have a very high false negative rate? I know that antibody tests can have fairly high false positive rates, but don't understand what is wrong with using PCR to indicate whether a person has active HHV-6. Please explain.
Regush: *Knox and Carrigan run a culture test. This can spot active virus. PCR is not the proper test to determine if a virus is active. Antibody tests determine whether there has been an immune response to an invader. The longer researchers continue to only use PCR, the longer it will take to show strong evidence for a viral factor in cfids*

[Cfbon]:What would it mean to you if someone had blood work that was negative for HHV6 IGG & IGM but pos. for PCR HHV6?
Regush: *most people will be positive for v-6 igg. igm is not a test that will show the virus is currently active. PCR will not prove productive infectivity. a culture will show that the virus is active at the time the blood was drawn.*

[Cfbon]:Through your reporting, you succeed in bringing needed attention to HHV6. Are you donating book proceeds to research?
Regush: *No, I'm not donating book proceeds to research. I have my private ways of helping people out.*

Ad Hoc Comment
Regush: *I have no idea what John thinks of my book. 20/20 passed on my book for a segment.*

[Jerry G.]:Knox, Carrigan and JHK Sydney Grossberg's patent on the JHK virus also lists Drs. Knox and Carrigan as co-discoverers. I'm only halfway through "The Virus Within" but have noticed no mention of their work with Grossberg and JHK. Can you share any information of their involvement with Grossberg and JHK and

their feelings about JHK's involvement in diseases such as CFS?
Regush: *I've mentioned that Knox and Carrigan are not happy about being included in the patent - they never were given the courtesy of reviewing the patent. nice.*

[Jerry G.]:Lipid Envelope There has been some discussion that the key to solving AIDS and possibly CFS is to dissolve the virus' lipid envelope. This is also something that was supposed to have been buried by the U.S. government. Any thoughts?
Regush: *I have no idea what this is about. sorry.*

[JT]:Most effective procedure to identify HHV-6A Sir, Thank you. I have had HHV-6A for well over a decade beginning with a severe encephalitis. What medical scientific procedure is the most foolproof test to discover active HHV-6 in my body at this late stage. Would a spinal tap be definitive? Or perhaps a SPECT or PET scan to identified damaged areas followed by a brain biopsy to affected brain tissue? Like to know before autopsy. Many thanks again.
Regush: *culturing a virus is the the gold standard - for activity.*

[GK]:HHV6 and illness. Why would HHV6 be highly active in so many diseases, yet have such a profound difference in the symptoms it produces. Ex. in AIDS and CFIDS. The infection is active in both syndromes yet CFIDS patients are more debilated for much longer periods of time, decades even, and they have different symptoms.
Regush: *An excellent question. As I try to point out in my book, chronic illnesses will turn out to be highly complex. They will likely involve a variety of inputs and processes and phases. V-6 may well serve as a trigger in some and only come in for the final hit in others, or work together with other microbes to launch complicated processes, which could involve some gene rearrangements. We are only beginning to touch the complex nature of illnesses such as cfids, aids, etc.*

[DebbieSinKC]:HIV negative AIDS is there any evidence, in HIV neg. AIDS, that another virus, instead of HIV, is working with the hhv6?
Regush: *No. But the sky is probably the limit. Lots of research will need to be done before we can figure out what is going on here.*

[DebbieSinKC]:lyme disease & cfs/me cfs and lyme disease seem to be so intertwined - could lyme be an opportunistic infection?
Regush: *I have no idea. sorry.*

[Fluffy]:Are there many "reporters" who know about the HHV6 problem but prefer to keep silent ? - because they are in fear of the having their careers and jobs destroyed (maybe even risk death) by delving into such sensitive (maybe "national security") matters. Hillary Johnson's career wasn't helped. Gary Webb had his career destroyed for reporting truthfully and delving into sensitive matters. I usually don't distinguish between the government and media in most cases. It seems the only medium left to publish things is in book form but even then one needs to traverse through a web of disinformation. What are some of the fears and considerations that you have had to contend with in treading on HHV6 - a seemingly sensitive issue for the government ? Also discuss any negative experiences that you have had (besides my questions) since publishing the book.
Regush: *I can't speak for other reporters. There is naturally a tendency to try to cut people down who raise q*

[DebbieSinKC]:opportunistic infections, again :-\ i caught a blurb on the local news 2-3 wks ago - about a common childhood virus, called RSV, i think. they were saying it normally caused very few symptoms. but increasingly over the last 20 yrs, has been causing more serious illness and fatalities in infants and young children. i thought the 20yr window was interesting, since this is also when aids, cfs/me, gws, and lyme have been occurring and increasing. how many other viruses/illnesses have also been increasing and/or getting more severe over the last 20-30 yrs or so, and have carrigan & knox, or anybody else, linked them to hhv6?
Regush: *a good question but I don't know the answer to that. I don't think anyone knows.*

[JT]:CFIDS community resistance to viral role in illness Mr. **Regush:** I have noticed many people with "CFIDS" are resistant to the idea that any chronic illness in which viruses play a role are actually considered CFIDS. Have you noticed this also? Any ideas why?
Regush: *yes, I have, and I'm not sure what's behind this. Fear of giving up some control to an invader? I'm not sure.*

[Kuby]:Myalgic Encephalopathy Research In an earlier chat you mentioned that Patient Organizations were responsible for funding poor quality research. Would you continue in more depth on this issue?
Regush: *More on this will be found in the chat transcript.*

[Tina]:Media Exposure Sir: How can we each work individually to get HHV-6A exposure out to the public and light a fire under the government? Where focus? My

second attempt to get this question listed. Thank you
Regush: *sorry if I missed it the first time. Two ways: put pressure on congressional oversight committees, much like organizations with interests in vaccines are now doing and 2) CFIDS groups with money to spend on research must figure out what credibility they can bring to the research arena by focusing on the top stuff out there an d not go on fishing expeditions.*

[dm]:Other research? I read your book and was happy to see a good representation of the facts on HHV6. While you were researching for the book did you come across any other promising research on Myalgic Encephalopathy that you did not include in your book?
Regush: *I think that I might have spent more time focusing on some of their studies conducted by John Martin. He's in early stages too with his notion that there are many brain diseases, some of which may tie into what is called cfids andetc.*

---

undefined
Regush: *well, it seems we've run out of questions. thanks for having me back. and goodnight*

Dr. Cecile Jadin's Papers are now available in full Click Here

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31893

National CFIDS Foundation

International CFS Information

Updated on: 02/09/2000

Dr. Howard Urnovitz, a research microbiologist from Berkeley, California he founded Calypte Biomedical in 1988 (http://www.calypte.com/) and associated with http://www.chronicillnet.org/

Publications: http://www.calypte.com/pages/research.html

Recent Publications:

- Cell and molecular biology in simian virus 40: Implications for human infections and disease / Response Journal of the National Cancer Institute [*] 7/7/1999
- (ADS) Urine Antibody Tests: New Insights Into the Dynamics of HIV-1 Infection

  Clinical Chemistry (09/99) Vol. 45, No. 9, P. 1602

  Urnovitz, Howard B.; Sturge, Jerrilyn C.; Gottfried, Toby D.; et al.

Interview on Dec 5, 1999: http://www.cfs.inform.dk/Nyheder.udland/cfsradio5dec.txt

"there is a cofactor in AIDS and that cofactor is HIV." HIV seems to be the "trigger" for a mild immune suppression.[*]

"he forwarded the theory that early "inactivated" Salk vaccines given to some 98 million Americans were also ***contaminated with monkey viruses*** and may be one reason why there has been an explosion of cancer, new infectious agents and other new immune and neurological disorders among the baby boomers born between 1941 and 1961." [*]

Joint paper with Prof. Nicolson in 1995, http://www.immed.org/free/htmldoc/!IJOMT-N.html

Testimony before House of Representatives Committee, August 3, 1999 http://909shot.com/urnovitz51899.htm

Main CFS Claim to fame: genetic alterations in the 22q11.2 region, possibly induced by exposures to environmental genotoxins may have played a role in the pathogenesis of CFS and GWI. (via Cheney) [*] More detail: http://www.chronicillnet.org/rna/RNAinblood.html

Editorial Comment: RNA changes are found but it may be *the chicken and the egg* question:

- Did the RNA changes happen because the body wanted to mutate *around an illness* or *condition (non-random environment-induced evolution),* ***or***
- Did the illness develop because of a RNA change

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

Supplements April

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National CFIDS Foundation

International CFS Information

Updated on: 03/28/2000

	Ken		Laurie
Prescription			
	300 mg Doxcycyline		300 mg Doxcycyline
			50000U Nystatin (1/day)
Minerals			
Iron			2 capsules
Zinc	50 mg	Complement Whey	50 mg
Iodine	330 mcg	Kelp	
Vitamins:			
A	2 @ 1250 IU	via Cod Liver Oil	2 @ 1250 IU
B12	4 @ 500 mcg		4 @ 500 mcg
B6	2 @ 100 mg	Close to Piracetam	2 @ 100 mg
B3 (niacin)	2 @ 500 mg		2 @ 250 mg
C	2 @ 1000 mg		2 @ 1000 mg
D	2 @ 130 IU	via Cod Liver Oil	2 @ 130 IU
E	1 @ 1000 IU		2 @ 400 IU
Supplements			
Glucosamine Complex			3 @ 500 mg
Whey	20 mg/day	ImmunoPro	20 mg/day
Bromelain	6 @ 600 GDU	Anticoagulation	4-6 @ 600 GDU

Piracetam	2 @ 800 mg	Anticoagulation / Brain Fog	2 @ 800mg
Polyphenols	170 mg	Grape Seed Extract	170 mg
CoQ10	600 mg		600 mg
DHEA	300 mg		300 mg
Oleuropein	950 mg	Olive Leaf Extract	950 mg
Jarro-Dophilus	3 capsule/day		4-5 capsules

Supplements March

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National CFIDS Foundation

International CFS Information

Updated on: 03/11/2000

Notes for the MD

	Ken		Laurie
Prescription	300 mg Doxcycline		300 mg Doxcycline - in morning only 50000U Nystatin (1/day)
Minerals		Stopped	
Iron			2 capsules
Vitamins:			
A	2 @ 1250 IU	via Cod Liver Oil	2 @ 1250 IU
B12	4 @ 500 mcg		4 @ 500 mcg
B6 (niacin)	2 @ 500 mg		1 @ 500 mg
C	2 @ 1000 mg		2 @ 1000 mg
D	2 @ 130 IU	via Cod Liver Oil	2 @ 130 IU
E	1 @ 1000 IU		2 @ 400 IU

Supplements			
Glucosamine Complex			3 @ 500 mg
Whey	1-2 packets/day	Imuplus	3-6 packets/day
Bromelain	4 @ 600 GDU	Anticoagulation	4-6 @ 600 GDU
Piracetam	2 @ 800 mg	Anticoagulation / Brain Fog	2 @ 800mg Nootropil
CoQ10	600 mg		600 mg

DHEA	300 mg		300 mg
Oleuropein	150 mg/day	Olive Leaf Extract	300 mg/day
Jarro-Dophilus	3 capsule/day	(see below)	4-5 capsules
As Needed (bad day recovery)			
NADH	15 mg		15 mg
glycyrrhizic acid	16-32 mg	solid licorice	16-32 mg
vinegar	1/2 oz		1/2 oz
Major Changes			
	Significant herxing for 2 weeks from OLE		Increasing energy in day
	3-4 hrs/day of work simulation went fine		Sleeping 10-12 hrs/day [Herx] (less)
	crisp 2 mile walk to top of Kingston hill daily		Back problems more prominent
	PM has increased herx		*Physical therapy
	* cummulative toxins from day plus herxing		* Chiropractor.
			appetite for sweets came back (back pain medicine)
			No apparent problems with Candida (Olive Leaf Extract)
			Taking longer walks (30-60 minutes) before back pain
Action Plans:			Appear to be passing pin worms in stools since Olive Leaf Extract
	Increasing Bromelain this weekend		Adjust Bromelain to control herxing level

Starting paid employment locally on hrly basis

work only at comfort level, no "PUSH" scenarios.

Severence package from Microsoft

Minimize sitting down time (for back)

Jarro-Dophilus Contents below:

20% L. rhamnosus
20% L. casei
10% L Plantarum
20% L. acidophilus
20% B. Iongum
10% B. Breve

Plantarum: Survives antibiotics
Produces B vitamins

Ken Lassesen

Curriculum Vitae

Email: KenL@exMSFT.com or KenL@Folkarts.com

* Short CV (MS Word) 2 pages
* Full CV (MS Word), 14 pages

Strengths:

982

A Guru of all software trades, Trouble Shooter and Performance Analyst, Web Technologies Developer, NT SQL Developer, VB Developer, C developer, Statistician and Operations Research scientist, Business Analyst, Educator and Technical Writer.

Disciplined mathematical mind that applies discipline and care to software analysis (performance analysis, testing and design benefit greatly). Great love of being on the first wave of new technologies... and doing complete business analysis of requirements. A leader (often by example) of developers

Not currently seeking positions

Other Courses

1981 - University of British Columbia, Faculty of Commerce

9 8 2

A Masters of Business Administration that concentrates on technical aspects of business administration and commerce. Usually a precursor to a Ph.D. Accepted into Ph.D. program at the University of British Columbia, but entered business instead.

Disciplines:

Information Management

- Databases
- Information Management

Operations Research

- Linear Programming
- Game Theory
- Non-Linear Programming
- Energy Policy Modeling (Doctorate Course)

Statistical Analysis

- Statistical Analysis

Master's Topic: Statistical Analysis of Paramedic Data concerning Cardiac Arrest.

Professional Teaching Certification

1975 - University of British Columbia, Faculty of Education. Secondary Certification

Focus:

- Mathematics
- Computer Assisted Instruction / Computers in the Classroom
- Adult Education

Bachelor of Science (Mathematics)

1974 - University of British Columbia, Faculty of Science

Focus:

- Probability and Statistics
- Geometry
- Problem Solving

Related Activities

- Putnam Mathematical Competitions
- Canadian Association of Physicist Competitions
- Alma Mater's Science Undergraduate Representative

Patents

[Leading Edge Projects](#)

The following patent applications are in process:

- Accelerated Data Transfer Protocols (MS 39534.1)
  - may be divided into three separate ones...
- Client Side Localization on the World Wide Web (MS 124469.1)
- Client Side Bulk Updates on the World Wide Web (MS 126571.1)
- Client Side Report Generation on the World Wide Web (MS 124467.1)

9 8 2

Prior History
Teaching

2000 - Present: Vision Compass

- Product Architect reporting to VP for Development

1994 - 2000: Microsoft Corporation

9 8 2

Typical performance review was 4.0 (out of 5) during my entire employment.

- ITG: Senior Applications Developer
- IMG Performance Analyst
- MSN Performance: Software Design Engineer / Test
- MSDN Library: Developer Technology Engineer

Lead Senior Application Developer

Functions included:

- Group Lead (3 SQL staff Developers, varying numbers of Contractors)
  - Regular interviewer of contractors and potential staff
- Preparing and giving "*What did we learn this week*" sessions to bring team up to speed on Web Technologies
- Lead, design/architect and implement a prototype that would allow *real fast* data access on a world-wide basis to all of Microsoft Licensing customers. These customers generate over $10 billion dollars of revenue to Microsoft annually. Criteria included:
  - High latency, low bandwidth
  - "Blow their socks off" performance. Demo trip to major customers in US and Europe was extremely well received (including requests from Digitial/Compaq to gain access to the technologies being used).
  - Multiple language and character set support (potentially the site may need to be available in 100+ languages)

    - Plato(OLAP / Data Warehouse) backend, IIS4 front end, IE5 client side

  Three patent applications resulted from the design.
- Reference: DCozart@Microsoft.com / dcozart@mslicense.com
- Maintenance and updating (better multi-language support and less testing cost) existing application. Reference: sgehres@Microsoft.com

Interact Media Group Performance Analyst

Functions included:

- Performance analysis of MSN web sites (from general design down to Bloodhound analysis).
- Designed and lead development of "packet sniffing web performance tool" in MFC
- Demonstration rewrites of sites (appears identical to the original) which saw decreases of "time to load" from 90+ seconds to < 15 seconds.
- SQL Developer, VB Developer, Web Developer as needed for internal systems (i.e. Dashboard Website Reporting System)
- Architect web-based (ActiveX) newsreader for MSN, lead team of three C-developers to implement it.
- Web technologies lead
- Regular interviewer of contractors and potential staff
- Reference: RobLamb@Microsoft.com

MSN Performance: Software Design Engineer / Test

- Research and educate team on Internet technologies, SQL and Visual Basic.
- Architect and developed first prototypes for “keep-alive” and performance monitoring of MSN website and services (http, nntp, pop, smtp and chat services). Known internally as the W3Monitoring Series.
- Regular interviewer of contractors and potential staff

MSDN Library: Developer Technology Engineer

Research and produce articles on Microsoft products and features that are not adequately documented. Demonstrate and advocate evolving technologies and assist in the rapid acceptance of the technology by customers. For a list of articles produce see Publications. Technical contributor to Visual Basic 4.0 and Microsoft Access 95

Certification Examinations. Presented at Conferences (TechEd) and booth duty at trade shows.

Architect and developed first production prototype of the PANDA system (1994), a converter of Rich Text Format documents into HTML designed to manage large sites and convert the entire the MSDN library into HTML. Oversaw contractors for subsequence versions of Panda.

Publications

Microsoft Developers Network

Many of the following articles are published on MSDN Library CD, http://www.msdn.microsoft.com and individual Microsoft Products.

Available on Line:

- An Extended Map of the **Active Server Page and Scripting Objects**
- An Extended Object Map of **Internet Explorer** 4.01
- Mapping the Data Access Object: DAO 3.0
- Mapping the Remote Data Object: RDO 1.0
- Mapping the Visual Basic for Applications Object Library: VBA 2.2
- Mapping Visual Basic 4.0: The VBIDE Object
- Mapping the Microsoft Graph Object: Graph 5.0
- Mapping the Microsoft Project for Windows 95 Object Library: MSProject 4.1
- Mapping the Data Access Object: DAO 3.1 (for ODBC Direct Data Sources)
- Mapping the Office Binder Object Library: OfficeBinder 1.0
- Mapping the Microsoft Office 95 Object Library: MicrosoftOffice
- Mapping the OLE Messaging Object Library: MAPI 1.0
- Mapping the Schedule+ OLE Automation Server: Programming Model
- Mapping the Schedule+ 7.0 Object Library: SPL 7.0
- Mapping the Standard OLE Types Object Library: StdType 1.0
- Mapping the VoiceCommand 1.0 Type Library: VCmdAuto
- Mapping the VoiceText 1.0 Type Library: VTxtAuto
- Mapping the "Word95 Objects for ACCESS": Word95Access
- Using Microsoft OLE Automation Servers to Develop Solutions

- Creating OLE Servers in Visual Basic to Simplify Windows Function Calls
- Mapping the Schedule+ OLE Automation Server: Internal Objects
- An Extended Introduction to Schedule+ OLE Automation Programming
- Visual Basic Script and the Access Developer
- Mapping the Microsoft Access 95 Object: MSAccess 7.0
- Mapping the Visual Basic 4.0 Object: VB 4.0
- Creating 16-Bit and 32-Bit Screen Savers with Visual Basic
- Introduction to Using the Remote Data Object
- ADODB: ActiveX Data Objects 2.1
- Mapping the SQL Distributed Management Object: SQLOLE 6.0
- A Hot Date: How OLE Automation Boosts Functionality in Schedule+
- Creating Useful Native Visual Basic and Microsoft Access Functions
- Building Add-Ins for Visual Basic 4.0
- Corporate Developer's Guide to Office 95 API Issues
- A Collection of Useful Native Visual Basic and Microsoft Access Functions
- Building Asynchronous Data Access Solutions Using MAPI
- Using Microsoft OLE Automation Servers to Develop Solutions
- Getting the Most from Your Visual Basic 4.0 OLE Automation Server
- Issues to Consider When Porting 16-bit Office Solutions to Windows 95
- Leveraging the Mainframe in Business Solutions with Microsoft Access and Visual Basic
- Microsoft Office for Windows 95 Resource Kit, MS Press, Writing Contributor.
- Porting to 32-bit Office, Visual Basic Programmers Journal, October 1995. With Mike Risse.
- Porting Your 16-Bit Office-Based Solutions to 32-Bit Office
- Visual Basic, Visual Basic for Applications, and Microsoft Access Basic: Optimizing Basic Performance
- Newspaper Article on Schedule+ (March 1996 MSDN Newspaper)
- Newspaper Article on VB4 Add-ins (March 1996 MSDN Newspaper)

Pinnacle Publications

- Schedule+ OLE Automation, Visual Basic Developer, Pinnacle Publication, April 1996.
- VB4: OLE Automation Server Design, Pinnacle Publications - Online
- Visual Basic Script and the Access Developer, Pinnacle Publications, January 1997

Other Publications

- **Web-base Applications: Extend Client/Server Applications to be Web-based**, white paper for Microsoft InterDev Group, 1997

- **Visual Basic 5.0 Internet Component Download**, white paper for Microsoft Visual Basic 5.0 Group, 1996
- A First Primer On OLE Automation, Apr 1995, CLVB Digest.
- A Word for Windows Doc to WinHelp Converter, Jun 1995, CLVB Digest.
- Statistical Validation of Quality of Service Measures.(B.C.Tel, 1983, 4 Volumes for the Canadian Radio and Telecommunications Commission, Ottawa)
- Emergency Response Treatment Factors affecting Patient Survival after Cardiac Arrest (Master's Paper, 1981)

Management Experience

- 1975-1980 Secondary Teacher
- 1983-1987 Manager Computer Applications (BC Tel)
  - Up to 3 staff developers
  - Additional contractors as needed
  - Clerical Support Staff
- 1988-1991 Systems Analyst
  - Small project lead (temporarily assigned staff)
- 1989-1995 Adjunct University Instructor
  - 3rd and 4th year computer science students (up to 30 in class)
- 1996-2000 Lead (rated as "developing manager" on last review)
  - maximum team size: 7 developers (contractors / staff)

9 8 2

Nota Bene

In 1988 (moved to the US), there was an intentional decision to move back into technical and away from management due to the major downsizing of management in the Telephone Industry happening at that time. At Microsoft, management had often stated that I was a too valuable technical resource and hence directed me to lead positions as a compromise (managing and being technical).

Languages / Products

The following are languages acquired through the years...

Language / Product	First Used	Last Used	Current Proficiency 0 - Chilled 10 - Sizzling
APL	1969	1990	2
ALGOL	1971	1972	0
B (father of C)	1984	1986	4
C	1985	1998	7
C++ (MFC / ADL)	1995	1998	4
COBOL	1971	1990	3
BASIC	1974	2000	10
Visual Basic	1990	2000	10
Fortran	1969	1986	3
SQL Server	1992	2000	10
Relational Databases (Misc, DB2 etc)	1980	2000	7+
Oracle	1985	1989	5
Javascript (Livescript)	1995	2000	10
Java	1996	1998	6
ASP Pages	1996	2000	10
Perl	1994	1998	4
LISP	1991	1995	3
REXX	1986	1989	4
Forth	1980	1986	3
SAS	1980	1988	7

Simula (father of C++)	1983	1987	4
Pascal	1977	1994	6
Microsoft Office Products	1988	2000	10
HTML	1993	2000	10
SGML	1995	1997	5
XML	1998	2000	6+
Assembly	1970	1987	5
Level 5 Object	1990	1992	5
Focus	1986	1989	5
C##	2000		*Learning*

Current Proficiency is assuming a 1-2 week of refreshing knowledge.

1991 - 1994: Best Enterprise Systems and Technology

9 8 2

- **Consultant: Provide consulting services to their clients located in the Seattle Area.**

Microsoft - Internal Technology Group

- Revision of Employee Review software system (Visual Basic).
- Wrote a converter of System Architect Data Files into Multimedia Viewer Books, including automatic hot spotting of all graphics.
- Developed SQL Server stressing and benchmarking software.
- Statistical analysis and research on NT SQL performance issues.
- Performance stressing of the CITS NT SQL database (still some of the worlds largest application on NT SQL)
- Created custom DLL's and VBXs in C.
- SQL Developer for International Conversion Costs Tracking Systems

Microsoft - SQL Group

- Developed a document database and UI for marketing material.

PRISM

- Converted DOS C programs for Sales Tax calculations into Windows DLLs.

1988 - 1991: General Telephone and Electric Northwest

• Systems Analyst

- **E911**: Designed and developed the data collection process from municipal governments into the GTE systems (Microsoft QuickBasic, Procomm Scripting)
- **EDI:** Supported the EDI system (written in APL).
- **Level 5 Object Task Force:** Technical Support on Level 5 Object and Windows for Domain Experts. Develop knowledge bases for Help Desk Support and development tools for creating Knowledge Bases (Windows, C++, Visual Basic, Clipper). Research and development on delivery of knowledge bases across multiple platforms. Design and develop Application Generators.
- **Help Desk**: 2nd level support for dBase, Lotus, Microsoft Project, Rbase, communications software and other MS-DOS products. Founded and sysop for DBASE board on GTE Telemail, frequent contributor to and sysop for PC.Board. Supply technical expertise to other IM groups on PCs as needed. Terminate and Stay Resident (TSR) development and various languages (C++ and xbase dominantly).
- **Sell One More**: Produced and support remotely the Sell One More Tracking System for GTCA, GTNW, GTNO, GTSW. [I.B.O.B.] MS-DOS, 3Com Lan, Carbon Copy, dBase.
- **Point of Sale:** Converted from Rbase to dBase, enhanced and supported. MS-DOS, QuickBasic, dBase, Rbase, IRMA, C.
- **PC/Terminal Survey**: Produced system for tracking equipment and facility needs during a move between locations. RBASE, 3Com.

1981 - 1988: British Columbia Telephone Company

1987 - 1988 Budget Systems Coordinator - Comptroller

Modifications and enhancement of the corporate budget and reporting systems written in

XSIM.

1985 1987 Computer Applications Manager - Operation Support Services

The primary job was management of systems for Operator Services and Marketing (including Retail Outlets). Systems included Directory Assistance Software and Point of Sale systems.

1983 - 1985 Computer Applications Supervisor - Operator Services

The primary job function was specification, design and development of software needs for Operator Services. Software development included: statistical reporting and analysis, employee scheduling (ErlangB and Queuing Theory). Supervised two programmer/analysts and clerical positions. Maintance of B-language programs on GTEDS System. Performance monitoring program for Operator Service CAMA positions (Digitial C).

1981 - 1983 Time Share Programmer

The primary job function was statistical analysis of Quality of Service measurements for reporting to the federal regulatory body. Tasks include production of a report on Reliability of Quality of Service measurement. The primary software used was the Statistical Analysis System (SAS) on the TSO operating system. Additional work included creating an automated analysis system of budgets submitted across the company against historical data.

1980 - 1981: Freelance Computer Consultant

Vancouver School Board, Vancouver, B.C.

Designed and developed an Audio Visual Booking system on an IBM 4341 using COBOL, CICS and VSAM. The system was *a relational database system* hand built written using VSAM. No commercial relational databases software existed at this time (IBM announced 'System-R' during this project). **In 1997**, this system is still in full production use with only minor modifications. Contact: Herb Peters

1975 - 1980: Secondary Teacher

- Vancouver School Board

Teaching Experience

Full Time

9 8 2

1980-1981 University of British Columbia, Faculty of Commerce

Instructor

- Computer Applications in Business

Teaching Assistant

- Management Science

1975 - 1980 Vancouver School Board, Vancouver, B.C. Canada

- Junior High School Science Courses
- Computer Science
- Probability and Statistics
- Astronomy
- Mathematics

Part Time

1989 - 1995: Chapman University, Orange Country, California

- NSB Bangor, WA
- Computer Sciences Instructor
  - CS 380 Operating Systems
  - CS 390 Artificial Intelligence
  - CS 402 Operating Systems
  - CS 352 Artificial Intelligence
  - CS 353 Data and Computer Communication
  - CS 380 Compiler Design
  - CS 404 File Processing

    - CS ... Database Design

1989: City University, Bellevue, WA

- Telecommunications Instructor
    - *Switching Techniques*
    - *Basics of Transmission*

1989: University of Washington Extension Department

- Oracle Instructor

1975 - 1977 Vancouver City College, Basic Skills Development

Adult education program

- Chemistry 12
- Physics 12
- Mathematics 12

Microsoft

MSN Performance: Software Design Engineer / Test

- Research and educate team on Internet technologies, SQL and Visual Basic.
- Architect and developed first prototypes for “keep-alive” and performance monitoring of MSN website and services (http, nntp, pop, smtp and chat services). Known internally as the W3Monitoring Series.

MSDN Library: Developer Technology Engineer

Research and produce articles on Microsoft products and features that are not adequately documented. Demonstrate and advocate evolving technologies and assist in the rapid acceptance of the technology by customers. For a list of articles produce see Publications. Technical contributor to Visual Basic 4.0 and Microsoft Access 95 Certification Examinations. Presented at Conferences (TechEd) and booth duty at trade shows.

Architect and developed first production prototype of the PANDA system (1994), a converter of Rich Text Format documents into HTML designed to manage large sites and convert the entire the MSDN library into HTML.

1992 - 1994: Best Enterprise Systems and Technology

Provide consulting services to their clients located in the Seattle Area.

Microsoft - Internal Technology Group

- Revision of Employee Review software system (Visual Basic).
- Wrote a converter of System Architect Data Files into Multimedia Viewer Books, including automatic hotspotting of all graphics.
- Developed SQL Server stressing and benchmarking software.
- Statistical analysis and research on NT SQL performance issues.
- Performance stressing of the CITS NT SQL database (still some of the worlds largest application on NT SQL)
- Created custom DLL's and VBXs in C.
- SQL Developer for International Conversion Costs Tracking Systems

Microsoft - SQL Group

- Developed a document database and UI for marketing material.

PRISM

- Converted DOS C programs for Sales Tax calculations into Windows DLLs.

1988 - 1992: General Telephone and Electric Northwest / GTE Field Services

1988 - 1991: Systems Analyst

- **E911**: Designed and developed the data collection process from municipal governments into the GTE systems (Microsoft QuickBasic, Procomm Scripting)
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Part Time

1987 - 1996 C.Alan Johnson Company

Design and develop production tracking and planning systems in dBase, Clipper, Smart, Javelin, Visual Basic and Microsoft Office products.

1991 A.C. Nelson Company

Develop software to allow clickable WMF maps as part of a user interface.

Leading Edge Projects

Example of especially successful leading edge projects

1979-1981: Hand-build a Relational DBMS using IBM's VSAM and implemented an Audio Visual Booking system using COBOL and CICS for the Vancouver (BC) School Board. This was done *before* the first commercial RDBMS (System-R) was released. System continued in use past 1997.

9 8 2

1994-1995: Build "PANDA" an RTF to HTML system for MSDN that worked with their existing databases and documents (~ 1 Gigabyte of source documents). Original version was created in 14 working days and was database driven. This was done as a result of BillG's turning Microsoft *on a dime* towards the Internet.

1996-1997: Build the W3Monitoring series of application to monitor the continued availability and performance (*from the real world)* of MSN Servers.

1998-1999: Developed a high performance website designed for worldwide use by external language, including support for unlimited languages and character sets. (See Patents)

Other Professional Courses

1995	University of Washington, Faculty of Engineering	• Technical Writing
1991	NCR/GTE	• TCP/IP Workshop
1990	IBI	• Level 5 Object
1988-1991	GTE	• Basic Asi-St • Advance Asi-St • DB 2 Programming • RAMIS II Report Writing • Intro to LAN, • Basics of Data Transmission • Managing Personal Growth
1986-87	SAS	• SAS Conference
1987	IBM	• DAS Conference
1981-87	BC Tel Education Center	• Quality Circle • Quality Circle Leadership Training • Basics of Telephony I,II and III • Traffic Analysis • Thinking on Your Feet • Labour Relations • Giving Effective Presentations • First Aid • Defensive Driving • ACF
1980-81	IBM	• VSAM • CICS • JCL • DOS/VSE
1975-81	BC Teacher Federation	• Classroom Management and Leadership Courses

Supplement Feb

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National CFIDS Foundation

International CFS Information

Updated on: 03/11/2000

Notes for the MD

Prescription	Ken		Laurie
	300 mg Doxcycline		300 mg Doxcycline
			50000U Nystatin (1/day)
Minerals		Stopped	
Iron			2 capsules
Vitamins:			
A	2 @ 1250 IU	via Cod Liver Oil	2 @ 1250 IU
B12	4 @ 500 mcg		4 @ 500 mcg
B6 (niacin)	2 @ 500 mg		1 @ 500 mg
C	2 @ 1000 mg		2 @ 1000 mg
D	2 @ 130 IU	via Cod Liver Oil	2 @ 130 IU
E	1 @ 1000 IU		1 @ 1000 IU
Supplements			
Glucosamine Complex			3 @ 500 mg
Whey	2 packets/day	Imuplus	5-6 packets/day
Bromelain	4 @ 600 GDU	Anticoagulation	4-6 @ 600 GDU
Piracetam	2 @ 800 mg	Anticoagulation / Brain Fog	3 @ 800mg
CoQ10	300 mg		300 mg
DHEA	300 mg		300 mg
As Needed (bad day recovery)			
NADH	15 mg		15 mg

glycyrrhizic acid	16-32 mg	solid licorice	16-32 mg
vinegar	1/2 oz		1/2 oz
Major Changes			BETTER Libido, Vaginal dryness gone
	Temperature Sensitivity gone		Reduced Chemical Sensitivity
	Significant Memory improvement [Bromelain/Piracetam]		Very tired/sleeping 14-16 hrs/day [Herx]
	Herxing re-started [Bromelain] - arms and legs		Increase Sound Sensitivity [Herx]
	With Herx: CP 25-30 sec		Typical: CP 15 sec
	Best CP:45 sec		Increased Anxiety [Herx]
	Rashes: disappears with hot tub (ozonator)		One night of insomnia, more urination at bed time
Action Plans:			Decreased appetite (probably feeling bad & Whey)
	As herxing decreases,		Adjust Bromelain to control herxing level
	increase Bromelain dosage		
			REPLENSE - as needed (just used once)

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

NADH

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National CFIDS Foundation

International CFS Information

Updated on: 01/10/2000

A nutritional supplement called NADH (nicotinamide adenine dinucleotide) - ENADA is the trade name - has been found to be effective in treating the fatigue associated with IDEF / CFIDS.

- ABCNEWS.com : Enzyme Helps Fight Chronic Fatigue
- FDA-Approved Trial Heralds Promising ENADA NADH Supplement for Treating Chronic Fatigue Syndrome (text version)

The theraputic dosage is ***at least*** 10 mg/day for most individuals. The study was published in the February 1999 issue of *Annals of Allergy, Asthma and Immunology.* Click here

The Niacin Link...

NADH is a precursor to Niacin (B3). No studies have been done on B3 and CFS. Niacin may also help or be as effective as NADH.

Sources:

The following has been volunteered to this site. Provided for information only (if you have additional cheaper sources, please email!)

http://www.beyond-a-century.com/

- NADH. 5 mg, 30 tabs, $24.75. Code 751.5

http://www.webvitamins.com

- NA Enada NADH 5mg 30T, $18.80

CoQ10

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Updated on: 07/30/2000

SHOULD NOT BE TAKEN WITH PRESCRIPTION BLOOD THINNERS..

The blood thinner ***Warfarin*** is specifically cited in

http://wwwchem.csustan.edu/chem4400/SJBR/laden98.htm

Also, it increases the uptake rate of vitamin E into the body.

List of research papers on CoQ10 is available at:

http://www.pharmanord.com/pnrm/c3.html

Sources:

The following has been volunteered to this site. Provided for information only (if you have additional cheaper sources, please email!)

www.nutrition-and-health.com

- They sell 30x100mg for 14.95 and they offer free shipping on even one bottle. (~ 0.5 cents / mg)

http://www.beyond-a-century.com/

- 120 caps, 30mg, $21.50. Code 203.3 (1.6 cents/mg)

http://www.webvitamins.com

- NC CO-Q-10 100mg-60S, $51.40 (1.2 cents/mg)

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

Vitamin B-12

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National CFIDS Foundation

International CFS Information

Updated on: 01/07/2000

Vitamin B-12 is essential for biosynthesis of nucleic acids, nucleoproteins, and red blood cells. It is needed to prevent anemia and aids in cell formation and cellular longevity. B12 is also required for proper digestion, absorption of foods, protein synthesis, and metabolism of carbohydrates and fats.

CFIDS

The positive results from taking B12 in some patients appears to be in agreement with low blood volume and low red blood cell count. One explanation for CFSers having low B12 absorption is hypochlorhydria.

"antioxidants including a type of vitamin B-12 are helpful to some patients." - Dr. Cheney

"Some reported having benefited from vitamin B12 and evening primrose oil" NZ Study

Vitamin B12 deficiency

Since B12 improves *some patients* - there is a possibility of B12 deficiency playing a role in some cases. The following applies to B12 deficiency (pathology of the disorder lies in the spinal cord and the sensory nerves [1]). A recent Swedish study suggests that current 'normal' B-12 levels may be too low. [More Research]

- tingling and numbness of the extremities, specially the legs
- imbalance of gait
- stiffness of the legs
- problems with urination [More info]
- problems with memory and thinking

Testing:

- homocysteine and methylmalonic acid levels

Personal Experience

For myself, taking of Vitamin B12 had a dramatic effect on one symptom: the waking up in the middle of the night to go pee, and then pee on the hour afterwards!! In the prior 6 months, I had had about 8 nights in total where that did not happen. After that dramatic improvement, I increased B12 supplements to 250mg/meal on the assumption that CFS was affecting the effectiveness of B12 usage or had caused the body ability to store B12 to be diminished (via hypochlorhydria).

There was a noticeable improvement in memory and thinking as a consequence.

Malic Acid

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International CFS Information

Updated on:
10/12/1999

Since the beginning of my illness, I had a strong need to drink lots of orange juice (2+ quarts per day). Only recently did I discover that the reason was not for Vitamin C but for the Malic Acid content of orange juice [2].

Malic Acid is recommend by several CFS specialists [1]

Vitamin E

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National CFIDS Foundation

International CFS Information

Updated on:
10/30/1999

Iron

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International CFS Information

Updated on: 06/02/2000

Hemochromatosis (iron overload) produces symptoms similar to CFS. This occurs in approximately 0.5% of the population. (See http://www.cfs.inform.dk/Borreliosis/hemochrom.html)

Iron Deficiency

In theory/by definition, no CFSer should have an iron deficiency since this is a recognized illness that should have been diagnosis before the diagnosis of CFIDS was given, however CFIDS is often given and an Iron deficiency is found

However, hypochlorhydria is a cause of iron deficiency [CDC].

"The ability to acquire iron in this way is particularly important to pathogenic bacteria, which must compete with their host for iron. In anaerobic environments, iron can exist in the more soluble ferrous state and is readily used by bacteria." Britannica [*]

Researchers:

"Beware of iron and copper supplements." Dr. Paul Cheney, MD [*]

Rickettsia/Mycoplasma and iron

"Since the average diet in the western world includes 15 mg of iron per day, iron deficiency must have some underlying cause. This could be hereditary hemochromatosis, a deficiency of vitamin B12 or folate, or, in the case of hemolytic anemia, an acquired blood disorder."

The Lancet, 04/2000[*]

"Three Cornerstones of Diagnosing Chronic Rickettsia Infection:

2) ... Iron study - abnormalities sometimes"

Dr Cecile Jadin MD MBBCH.[*]

Zinc

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International CFS Information

Updated on:
01/12/2000

- 85 percent of CFS patients may have a serious zinc deficiency [*]
- deficiency found with mycoplasma-positive CFSers [*]
- may be responsible for alcohol intolerance [*]
- because of risk of upsetting copper/zinc ratio - should be tested for.

Glycyrrhizic Acid

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Updated on:
01/31/2000

Licorice or SPEZZATINA SOLID

See http://www.seaquake.com/bulk.html#42 for source

- Anti-inflammatory
- Increases blood pressure (helps with NMH)
- Helps with adapting to stress better (in rats)
- Helps with intestinal absorption of drugs [*]
- Effective anti-thrombin action [*]
- Reduces mortality rates for flu [*]
- Inhibits some retrovirus [*,*]

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International CFS Information

Updated on: 12/07/1999

Neurally Mediated Hypotension

Patients with CFS have a high prevalence of neurally mediated hypotension, and open treatment of this autonomic dysfunction has been associated with improvements in CFS symptoms.

- NEURALLY MEDIATED HYPOTENSION AND ITS TREATMENT http://ww2.med.jhu.edu/peds/cfs.html
  "individuals with neurally mediated hypotension need to take in ***much higher amounts of salt***"
- Neurally Mediated Hypotension and Chronic Fatigue Syndrome, http://www.cfs.inform.dk/Nyheder.udland/wa5.txt
- Neurally Mediated Hypotension) http://ncchem.com/cfids_new.htm

Blood Volume Diet

Updated on: 09/03/1999

Keyword: Hydroscopic

- Caffeine lowers sodium reabsorption (decreases blood volume)
- Cold dry air absorbs water from the lungs and the body loses fluids with every breath
- A high concentration of salt in the bloodstream causes the blood plasma to absorb water
- Glycerol consumption may be helpful [1]

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National CFIDS Foundation

International CFS Information

Updated on: 01/01/2000

Once IDEF / CFIDS becomes established,patients are suffering from a significantly reduced blood volume (per Dr David Bells, Lyndonville, Upstate, New York [1,2,3]) which consequentially produces many of the common symptoms. Florinef is a medication that may be effective in treating blood volume.

The mechanism is thus:

- *less blood cells* to:
  - carry oxygen to the muscles / brain
  - remove wastes from the muscles after exertion
    - mental impairment results
    - long recovery periods after exertion (the wastes must be removed!)
- less dilution of material in the blood (higher concentrations)
  - drugs administered are more concentrated, resulting in sensitivities
  - increase in allergy / asthma: a normal amount of histamine pouring into a reduced blood volume

See "Case Study - Round Two" for specific observations.

This impaired material-delivery (aka blood) system confuses the body system to massively overproduce Rnase-L, an enzyme used to keep past virus in control (the vireo that the person has become immured to in the past). The body in this overproduction mode proceeds to manufacture a defective (lighter molecular weight) version which is ineffectual for controlling past infections. The person re-experiences the past vireo symptoms.

Since the immunity to the virus exists, the body already has developed the immunity to these vireo, blood chemistry does not show any new viral infections.

Mycoplasma

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Nicolson Protocol

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International CFS Information

Updated on:
03/18/2000

Introduction to Mycoplasma

http://www.md.huji.ac.il/microbiology/book/ch037.htm

Other Mycoplasma Diseases

I believe that others experience with other mycoplasma illnesses can help greatly with CFS. Treatment protocols that attacks mycoplasma should share common characteristics, and diets that increases the chance of success with one illness will likely increase the odds with CFS.

CFS and Mycoplasma Research

Aristo Vojdani, PhD, MT Al Robert Franco, Multiplex PCR for the Detection of Mycoplasma fermentans, M. hominis, and M. penetrans in Patients with Chronic Fatigue Syndrome, Fibromyalgia, Rheumatoid Arthritis, and Gulf War Syndrome [Journal of Chronic Fatigue Syndrome (The Haworth Medical Press, an imprint of The Haworth Press, Inc.) Vol. 5, No. 3/4, 1999]

Nicolson, G.L. Considerations when Undergoing Treatment for GWI / CFS / FMS / Arthritis; Antibiotics Recommended When Indicated for Treatment of GWI / CFS / FMS / Arthritis Intern. J. Medicine 1998; 1: 123-128 http://www.immed.org/prod/research/antibio.html

Mycoplasmal Infections in Chronic Illnesses: Fibromyalgia and Chronic Fatigue Syndromes, Gulf War Illness, HIV-AIDS and Rheumatoid Arthritis, Medical Sentinel 1999

http://www.immed.org/prod/research/ms99.html

For further articles on Mycoplasma see:

- http://home.freegates.be/nvdeynde/mycoplasma/
- http://www.immed.org/prod/chronic/chronic.html
- http://goulburn.net.au/~shack/mycoplasma.htm

Testing

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International CFS Information

Updated on: 02/08/2000

Testing Resources:

http://www.shasta.com/cybermom/myco%20resource.htm

If you have information on other labs that does PCR please email me so I may add it here. These are informational postings only (no endorsement is intended).

INTERNATIONAL MOLECULAR DIAGNOSTICS, INC. (IMD)

MYSTERIOUS MYCOPLASMAS were nearly undetectable until Dr. Nicolson employed a DNA analysis technique called Forensic Polymerase Chain Reaction. Dr. Nicolson and other researchers are finding high percentages in victims of many diseases whose causes are unknown Researchers believe that mycoplasmas may be the cause or cofactors in the following diseases:

- Chronic Fatigue Syndrome
- Gulf War Illness
- Fibromyalgia
- Rheumatoid Arthritis
- AIDS
- Lupus
- Scleroderma
- Auto immune disorders
- Asthma
- Inflammatory Bowel Disease

- Respiratory Distress Syndrome
- Multiple Sclerosis
- Sarcoidosis
- Amyotrophic Lateral Sclerosis (ALS)
- Wegener's Disease
- Kiku-chi's disease

INTERNATIONAL MOLECULAR DIAGNOSTICS, INC. take all insurance:

- Medicare
- Medi-Cal
- CHAMPUS
- & all other insurance

FULL SERVICE LABORATORY - The laboratory has expanded its services to include other blood tests. They will be offering all routine chemistry, endocrinology, HIV testing, etc. but will also offer specialty testing for Mycoplasmas, Chlamydia pneumonia and is now a full service REFERENCE laboratory. They will be adding new and innovative blood tests in the near future.

FOR PRICE, INSURANCE INFORMATION & TYPES OF TESTS AVAILABLE please call:

International Molecular Diagnostics, Telephone: 714-799-7177.

Nicolson Protocol

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Prof. Nicolson

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International CFS Information

Updated on:
02/13/2000

For the latest protocol go to

http://home2.freegates.be/Mycoplasma/nicolson/treatment/index.htm

"Several months (starting with 6 months [no break], then 6-wk on 2-wk off antibiotic cycles) of doxycycline, ciprofloxacin, azithromycin, minocycline, clarithromycin or similar antibiotics work best as capsules without starch fillers. Oral antibiotics must be taken with a full glass of water, crackers or bread to avoid esophageal irritation (do not lie down for at least 1 hr)." [*]

Prof. Nicolson

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International CFS Information

Updated on: 01/01/2000

"Dr. Garth Nicolson, professor and chairman of the Department of Tumor Biology at the University of Texas M.D. Anderson Cancer Center.

Dr. Nicolson's daughter, a Blackhawk helicopter crew chief with the 101st Airborne Division, had herself been infected and had subsequently infected the entire Nicolson family. A Nobel Prize nominee and world-renowned cell biologist, Dr. Nicolson decided to investigate what to him was more than simply the beginnings of a coincidental epidemic. What he soon discovered was a nightmare beyond even his own expectations.

With enough expertise to unravel the molecular structure of whatever it was that had infected his family, as well as the hundreds of veterans whose blood he'd sampled, Dr. Nicolson developed a technique he called "gene tracking." Armed with this new technique, he'd learned not only that the microorganism responsible for many Gulf War Illness symptoms was *Mycoplasma incognitus* (one of the microbes used in developing germ weapons) but also that *this* particular strain of mycoplasma was man-made and had incorporated into it 40 percent of the HIV protein coat, making it extremely pathogenic." (from: http://www.healthnewsnet.com/page41.html)

"Garth Nicolson before setting up the Institute for Molecular Medicine, a 501c3 corporation, in Huntington Beach, Calif. was the David Bruton, Jr., Chair in Cancer Research and professor at the University of Texas M.D. Anderson Cancer Center in Houston, and professor of internal medicine and professor of pathology and laboratory medicine at the University of Texas Medical School at Houston.

He was also adjunct professor of comparative medicine at Texas A&M University. Among the most cited scientists in the world, having published over 480 medical and scientific papers, edited 13 books, served on the editorial boards of 12 medical and scientific journals, and currently serving as editor of two (Clinical &

Experimental Metastasis and the Journal of Cellular Biochemistry), he has been the recipient of numerous research grants from the U.S. Army, the National Cancer Institute, National Institutes of Health, the American Cancer Society, and the National Foundation for Cancer Research. In 1998, he received the Stephen Paget Award from the Cancer Metastasis Research Society and the Albert Schweitzer Award in Lisbon. Nancy Nicolson, a molecular biophysicist, was on the faculty at Baylor College of Medicine's Department of Immunology and Microbiology." (From http://www.abcjb.com/news/gws_feature_article_issue_date_o.htm)

Description of Rickettsia

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Updated on: 09/30/2000

ricket.gif (1604 byteses)

The rickettsies are parasitic endocellulari obbligati.Possono to be cultivated (exception ***Rickettsia conorii***) also on different cellular substrata from the cells fagocitiche.Data the nature intracellulare of these bacterias, they often determine infection cronica.Dipendono from the energy in the form of ATP of the cell ospite.Hanno rather long once of duplication (around 12 hours) .Le rickettises have a form coconut-bastoncellare and their transmission happens through for the arthropods vectors. The kind ***Rickettsia*** consists of two groups antigenicamente, physiologically and ecologically well definiti:gruppo of the fever bottonosa and group of the tifo;le rickettsies of the group of the typhus of the brushwood differ in not to have the lipopolisaccaride instead, the peptidoglicano, misses besides of a layer external type capsule or " slime ", therefore a new kind ***Orientia has*** been created. The cultivation of the rickettsies happens on cells vital eucariotiche of crops cellular, eggs embrionatre and in animal suscettibili.Per the diagnosis sierologica always needs richiedereere two champions of serum one of the acute phase, the other of the phase convalescente.Di entrambi the serums it is found the title anticorpale in the same session .Le rickettsie they are present in the infected blood to a low concentration of around 10-100 vital organisms for ml but forgives vitality after a few times to temperature ambiente.Il blood you/he/she can be insofar used for the isolation of the rickettsies when picked in the acute phase and before the antibiotic therapy.

The various technical sierologiche for the diagnosis of rickettsiosi is exposed in this chart:

Technique	positive least title	Departed time per the survey of the antibodies	Comments

IFA	16-64	2-3 weeks	relatively sensitive and it requires little antigene;riesce to distinguish the isotipis of the immunoglobulines
CF	8 or 16	3-4 weeks	Less sensitive than the method IFA or of the annulled one, but very specific
ANNULLED	The controls' DO>0,25	1 week	the test of capture of the IgMs promises to be reliable for the precocious diagnosis
agglutination to the latex	64	1-2 weeks	it misses of sensibility for the serums of convalescents
IHA	40?	1-2 weeks	greater sensibility of the IFA and the CF
Immunoperossidasi	20	7 days	still from valutare;utile in diagnosis of field
Microagglutinazione	>8	1-2 weeks	it asks for a lot of ag sensitive ;meno of the IFA
IT GETS BACK	10?	7-10 days	the IgMs can be useful in the precocious diagnosis
Weil-Felix	40-320	2-3 weeks	it misses of sensibility and specificity

The Diagnosis of laboratory happens through the following methods: 1) isolation of the microorganismis through inoculation in animals from laboratory, cellular crops, eggs embrionate (but since to treat the rickettsies is difficult and dangerous few they are the laboratories that do him/it); 2) Ehrlichia, another kind of rickettsia, can be shown in coloration Giemsa of the blood in the monocitis, while the other rickettsies are often looked for in sections of biopsies of skin or plotted post colored mortem with marked antibodies with fluoresceina; 3) IFI indirect immunofluorescenza in 2 following champions to 10-15 days of distanza.Il according to champion during the convalescence, this is worth as it regulates general; 4)La fixation of the complement is used in the diagnosis sierologica of fever Q; 5) The antigens of the rickettsies for test Annulled, agglutination to the latex they commercially are not obtainable but through institutes of reference what the CDCs in Atlanta (in Italy to turn to the Superior institute of health); 6) The old Weil-Felix test exploits the cross

reaction sierologica among some kinds of the kind Rickettsia and Proteus OX2, OX19, OXK but it is not specific and it is by now obsolete .Alcune rickettsies have therefore antigens polisaccaridici in common with ***Proteus vulgaris*** log OX19 and OX 2 and with the log OXK of ***Proteus mirabilis.*** But "are in 2000 and only in Italy make ancora the Weil-Felix!" (Donato Fumarola of the university in Bari to a conference on the randagismo 30-10-98 to Montesilvano-PE).

In the reaction of Weil-Felix the serums are contemporarily ventured with the suspensions of the three separate antigens allowing cosi' a differential diagnosis among the numerous illnesses from rickettsia.Si they consider the titles superior agglutinations to 1/100.L'aumento of the title agglutinant between 7 and the 14 day it is what has greater diagnostic meaning.

"The use of the Weil Felix has produced more wrong and misleading results of every other test sierologico used for noticing the antibodies anti rickettsia.Non I wag, if of this is kept in mind, the Weil Felix test is able of to make a first presumptive diagnosis in the illnesses caused by the groups of the typhus and the fever bottonosa.Come is seen by the chart the test it is a lot of aspecifico and little sensibile for the fever bottonosa and therefore it doesn't help in the diagnsoi of the illness or in to differentiate her/it from the typhus ...Gli commercially walls up acceptable antigens of Proteus they are those standardized against the human serums of the phase convalescente.Se these conditions they don't subsist, not to use the test.I resulted you/they would not have meant and they would cause only confusion." (From Manual of Clinical Microbiology 4th editions pag.850."

Illness	Agent	OX 19	OX 2	OX K
Typhus esantematico	R. PROWAZEKII	+++	+	-
Typhus walls up	R. MOOSERI	+++	+	-

Fever maculosa of the rocky mountains	R. RICKETTTSII	+ or +++	+++	- or +
Fever bottonosa of the Mediterranean one	R. CONORII	+ or +++	+++	-
River fever of Japan	O. TSUTSUGAMUSHI	-	-	+++
Fever Q	C. BURNETII	-	-	-

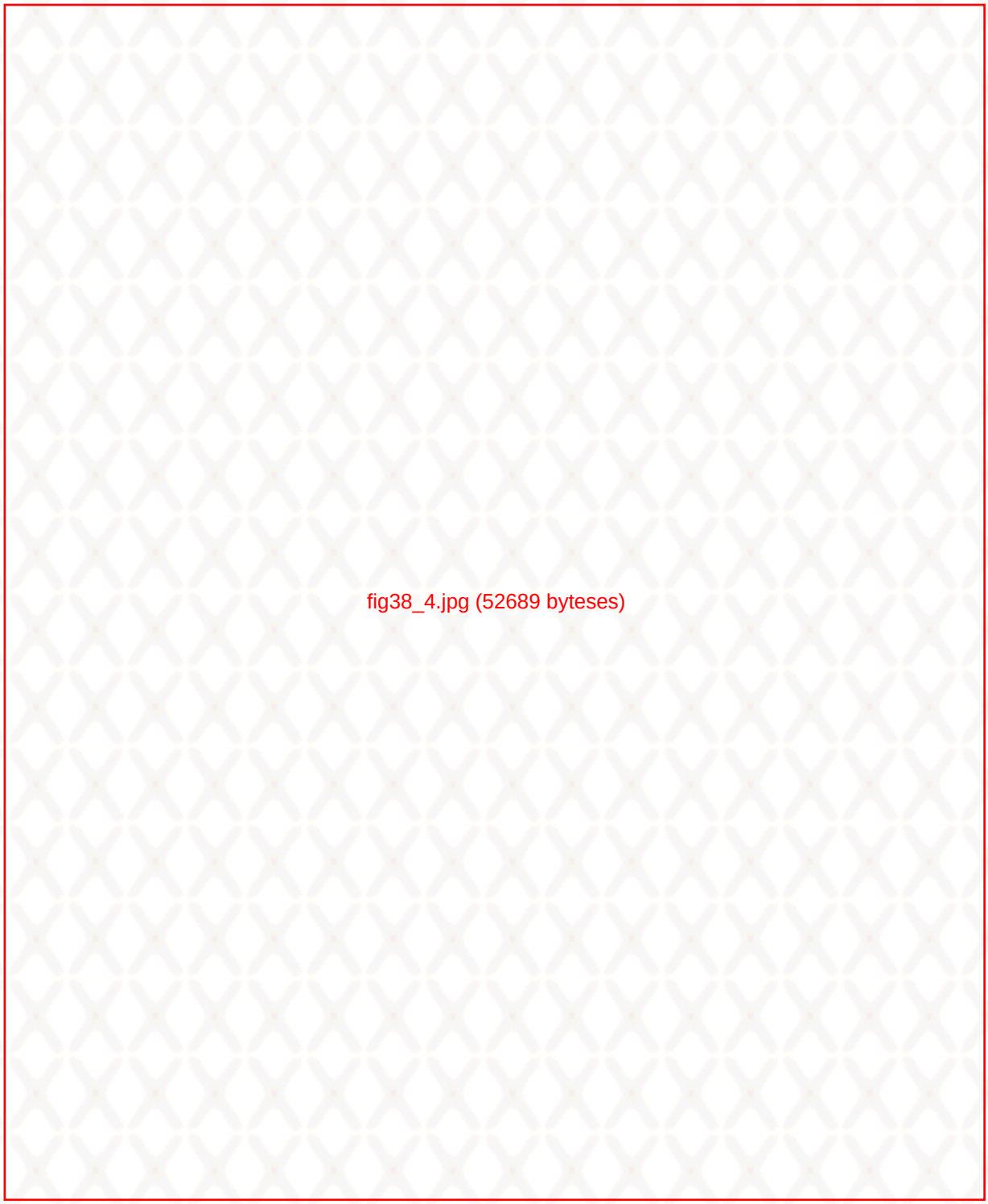
I principal groups of rickettsie illnesses are the group of the typhus, of the fever bottonosa and of the river typhus or of the brushwood .Essis have a picture similar nosologico: 1) a period of incubation of 1, 2 weeks; 2) a feverish period from 1 to 3 weeks; 3) a rash that begins in the first week of illness; 4) a vasculite disseminated of the capillary ones that it results from the infection of the cells endoteliali and that it associates him to an increased capillary permebilità and ipotensione; 5) an answer anticorpale after 1, 2 weeks from the beginning of the illness; 6) rickettsiemia from the beginning at the end of the feverish period, that coexists with an increase of the title anticorpale in the final period of the fever; 7) a persistent very strong specific hypersensitivity.

order	rickettsiales
family	rickettsiaceae
tribu'	rickettsiea
son-in-laws	Rickettsia-Orientia-Coxiella-Erlichia

Il name derives from Howard Taylor Ricketts, that scopri' the agente of the fever bottonosa of the rocky mountains to the beginning of 900.

Her rickettsies are bacterias gram negativi.Hanno dimensions similar to those of the bacterias and form bacterial coconut (0,2 µms x 2 µms) .Nons become flushed well with the Gram but with derived stronger than the Gram what the coloration of Gimenez.I Plasmidi I/you/they have been found only in ***Coxiella burnetii.Ehrlichia canis it*** enters the monocitis of the blood, it is replied in the fagosoma while ***C. burnetii*** enters the cell guest (macrofago) and it is replied in the fagolisosoma.Il type of cells parassitate (cells endoteliali,macrofago) determina the nature of the lesion (endovasculite, granulomi) with presence or absence of rash cutaneo.L'infezione can be persistent, chronic or ricorrente.Le rickettsies grow in macrofagi not activated but they are killed by macrofagi activated by interferon gamma.Sono opsonizzati from antibodies and complement that favor the killing in the fagolisosoma

except the case of ***C.burnetii*** the most resistant rickettsia .Importante it is also the role of the quote-toxic Linfocitis T that lisano the infected cells from rickettsie to the contact.



Il cycle of development of the rickettsie s happens between the mammals and the arthropods vectors what mints, acari, lice, shown pulci.E' the vertical

Ixodes_3.gif (28185 byteses)

transmission or transovarica from a generation to the other in the mints or acari through the uova.Eccettos the fever Q the arthropods are the half principal of transmission to the uomo.Ad exception of the typhus petecchiale transmitted by the pidocchi,cons only reservoir the uomo, a lot of rickettsiosis they are of the real zoonosi;cioès they are infections of animals in which the man is an accident in the normal cycle enzootico.

In the figure the mint of the fallow deer ***Ixodes*** in partnership ***scapularis*** with a form of erlichiosi granulocitica umana(Erlichia chaffeensis) in the north of the United States.

Vettore:per vector him it intends the artropode that spreads the infection propagating the organism that completes a cycle of development to his/her inside without multiplying himself/herself/themselves.

Reservoir or serbatoio:per reservoir him it intends the animal where the organism infettante him multiplication attivamente.Nel case of the arthropods they are generally reservoirs through the transmission transovarica or rather the organism infettante it is actively duplicated in the ovocitis and you/he/she is transmitted to the following generation.

Group of the fever bottonosa

Rickettsia rickettsii	Fever bottonosa of the Rocky Mountains (mints)
Rickettsia conorii	Fever bottonosa of the Mediterranean (Boutonneuse fever)(zecche)
Rickettsia africae	African tick-bite fever
Rickettsia australis	Queensland tick typhus
Rickettsia siberica	Siberian tick typhus
Rickettsia akari	Rickettsialpox

Group of the Typhus

Rickettsia prowazekii	Epidemic typhus esantematico (louse)

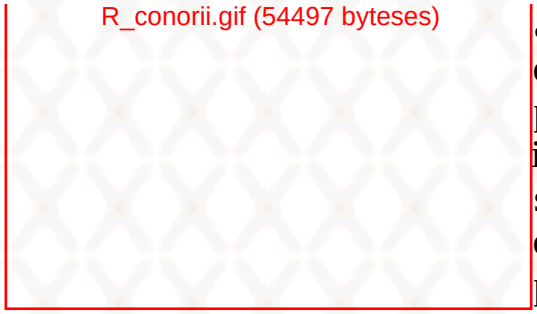
Rickettsia typhi	Typhus walls up (typhus endemico)(pulce)
Orientia tsutsugamushi	Scrub typhus (river fever of Japan etc.) (acaro)

Other illnesses

Coxiella burnetii	Fever Q
Ehrlichia sennetsu	Sennetsu fever
Ehrlichia chaffeenis	Human monocytic ehrlichiosis
Ehrlichia sp.	Human granulocytic

Da the 1991 *Orientia tsutsugamushi is* a germ apart. *R. prowazeki, R. typhi and R. rickettsias* are not common in Italy.The coxiella doesn't provoke esantema, it is tied up to the randagismo, it survives to the environment esterno.Non it asks for vettori.Provoca a pneumonia interstiziale.I stray dogs they are of the diffusers through infected placentas ,ingerite from the cani.Nons it seems however present in Abruzzo unlike *R. conori* that is the rickettsia most common in Abruzzo. *R. conori* provokes esantemi maculo papulosi, hemorrhages on the whole corpo.Si he/she calls fever bottonosa for the presence of the typical " tache " " noire " .E communicated by the *Ripicephalus sanguineus*, mint of the cane.Queste mints stay infected for the whole vita.Le rickettsies locate him in the endotelis of the small vasi.Dopos a certain period of incubation there is the debut with elevated fever, cefalea, injection of the conjunctiva, is tossiemico.Se it is found the " tache " " noire " we can tell us fortunati.L'esantema avviene the 3 day from the beginning of the malattia.Gravi complicanze emorragiche, encephalitises, dark polmoniti.L'escara or " tache " hard " noire " 8-10 giorni.Las therapy is to basic of tetracicline, cloramfenicolo, doxiciclina.

There are cases recrudescenti in Italy of fever bottonosa.Si they in red the rickettsies with fucsina fenicata. ***Rickettsia conorii is*** the most diffused rickettsia in Italia.Si you/he/she can also make now the PCR. The " tache " " noire " is a cutaneous necrosis caused by the infection of the rickettsia in the point of the inoculation for mint puncture, you/he/she is observed in around half the patients with fever boutonneuse.Nelle endemic zones there is



a tall prevalence of antibodies against R. conorii in the population sana.Questa prevalence can be explained by pregresse illnesses not diagnosed, infection subclinica, infection with a rickettsia correlated but less antigenicamente patogena, or for the not specificity of the test of laboratory that is used for titular these antibodies.

L' erlichiosi is a zoonosi emergente.E' transmitted by zecche.Non it provokes damage endoteliale.

" tache " " noire "

Fever Q

INTRODUCTION

La fever Q (from Queensland) it was for the first time described in Australia in 1937,ma it is to tuttoggi considered a diffusion malattia mondiale.La it causes it is the rickettsia ***Coxiella*** bovine ***burnetii.I***, the ovine ones, the goats and other animals I am the reservoir and therefore the fever Q is an industrial disease for those that you/they work in the industry of the meat and the latte.La illness in the man as result of the inhalation of aerosol that the organism contains happens.

L' acute infection him apparent in the man with a feverish state, illness auto limitante that has among the symptoms the fever, cefalea and mialgia. The direct isolation of the microorganismo is dangerous, therefore the diagnosis is indirect through the survey of specific antibodies in the serum, with a method Annulled that the most sensitive method is considered.

L' I increase specific of the Igs M of the phase II of ***C. burnetii is*** commonly used in the diagnosis of the acute illness, with the antibodies that immediately appear after the beginning of the symptoms and rilevabili after 3 months. ***C. burnetii*** has that is a characteristic variation of phase it exists in 2 phases antigeniche The and II, that are analogous to the smooth and wrinkled forms of some bacterias. ***C. burnetii*** exists in the phase antigenica The in nature, but it changes to the fase II after continuous passages in the cellular crops or in the eggs embrionate.Il passage to the phase II depends on the deletion of some component ones of the LPS (lipopolisaccaride) from the part of the half carboidratica. In the acute infection the

antibodies predominate against the antigens of phase II.Nella chronic infection of the fever Q the antibodies of the phase The exceed or eguagliano those of the phase II.Il I compare phase I/fase II it is a discreet indicator to distinguish the acute infection from the cronica.Rapporto <1 in the primary fever, >1 for patient to risk of endocardite.La greater part of the patients with fever Qs have an answer from Ig Á to the antigen of the phase II, therefore the use combined of IgA and Ig M increases the specificity of the diagnosi.I you level anticorpali of IgG they are useful in to determine a precedent exposure for the screening prevaccinale. The phase The it is the form found in nature and in the infections umane.La phase II it contains a truncate lipopolisaccaride, it is avirulenta.Le cells target they are macrofagi of the bellows, liver, bony marrow, spleen, valves of the heart and other organs. ***Coxiella burnetii is*** fagocitata from the cells of the Kupffer and other macrofagi and it divides him inside the fagolisosomis for binary fission. ***Coxiella burnetii has*** been found in the urines, in the milk, in I did her some infected animals.

MORPHOLOGY

- The causal agent is ***Coxiella burnetii***, a rickettsia
- it is a bacterium forced intracellulare
- characterized by 2 phases antigeniche (The & II)
- phase The - it is found in nature, virulent
- phase II - it is found after frequent passages in the laboratory
- it is the most stable rickettsia to the external environment
- it withstands in fact to the disseccamento, to the chemical substances and many disinfectants

PATOGENESI

- vettore is a mint
- animal reservoirs are - cattle, goats, sheep, domestic cats, wild animals
- the organisms are found again in great number in the attached ones of the birth and in the excrements of infected animals
- the infection in the man has a respiratory street through the inhalation of particles of dust that you/they contain dry parts of animal fluids
- period of incubation commonly from 2 to 3 weeks

YOU WAIT CLINICAL

- variety of symptoms that goes from inapparente to mild to severe
- the beginning can be sudden
- shivers
- cefalea
- muscular cramps
- weakness and general discomfort
- abundant perspiration
- hepatitis
- pneumonia and other respiratory symptoms
- you/he/she can conduct to complications in women in pregnancy
- meningitis
- the illness introduces him in acute and chronic form
- the endocardite is the symptom most common of chronic illness
- the illness in the animals is often asintomatica, but you/he/she can cause abortions in the livestock.

ANSWERED ANTICORPALE

- IgM and IgA of the fase IIs immediately appear after the beginning of the sintomi·s
- IgM and IgA of the phase IIs are rilevabili for more than 3 months
- IgAs are able not to be rilevabili all the patients
- The answer IgM can be very prolonged

- IgG antibodies immediately appear after the IgMs, but they are revealable for years in the life.
- The specific antibodies of the phase The are characteristic della chronic illness

DIAGNOSIS

- clinically the fever Q can be confused with the brucellosi and the leptospirosi.
- the method most common of diagnosis is then the survey of specific antibodies in the serum.

CROP

- **very dangerous, therefore it is an examination not performed in routine**

- it asks for an equipped laboratory

ANNULLED

- the method of more sensitive diagnosis is considered (studies on IgG)
- it correlates well with the test of fixation of the complement
- for the diagnosis it serves an alone champion of serum
- you/he/she can measure the answer to different classes of antibodies

TEST OF FIXATION OF THE COMPLEMENT (CFT)

- they serve 2 champions of serum with an increase of 2 times in the concentration
- the antigen is that of the phase II
- the answer anticorpale can be slow over the 21 days in some patient ones
- the answer anticorpale can persist to low levels for mesi/anni
- static titles are however a problem

TEST OF IMMUNOFLUORESCENZA (IFA)

- it is a method recognized of diagnosis
- more sensitive del CFT because the antibodies already appear in 5 days
- you/he/she can measure classes different anticorpali for different phases
- however it is difficult to standardize

PREVENTION

- the employees' vaccination to the butchery and others in dangerous occupations
- the milk's pasteurizzazione from cows, goats and sheep to inactivate the microorganismo

TREATMENT

- Somminstrare Tetracycline after the patient is afebbrile
- to eliminate the source of infection

(To) bacterial Cells of ***Rickettsia conorii*** in cells human endotelialis in crop are found again free in the citoplasma.Si he/she sees well a rickettsia while him you/he/she is dividing for binary fission (arrow). (B) These rickettsies can move inside the cytoplasm of the cell guest because of the strength propulsiva created by the tail of the filaments of actina of the cell (arrow) .Barre=0,5 µm.

What is the Fever Q?

Ti I can say that it is a zoonosi, in the sense that the microorganismo responsible of the infection ***(Coxiella Burnetii) it*** completes his/her vital cycle in the animal (also domestic, but more often caprini, ovine and cattle) in whose defecations you/he/she is sent forth, and then you/he/she can be transmitted to the man.

How does he transmit?

Più often inhaling dusts infected coming from contaminated environments (stalls, slaughter houses, butcheries) or from animal by-products (skins, wool, torn), rarer it is the contagion interumano. N.B. ***C. Burnetii is*** very resistant to the external environment, at

times you/he/she can withstand (es. in the milk) also for 3 years.

How does the infection manifest him?

as a rule the nfezione can elapse asintomatica, or the typical picture sintomatologico of the Fever Q can be introduced, after incubation of 2-4
weeks: abrupt debut with elevated fever (39-40°C), prostration, inappetenza, mialgie and, important for the differential diagnosis:
- BRADICARDIA
Intense -CEFALEA, gravativa, with pain resistant retroorbitario a great deal to the
analgesics
Detached -FOTOFOBIA and injection congiuntivale.
There is at times involvement of the sensory thin to a state confusionale.
Non excessive -EPATO-SPLENOMEGALIA
There is finally the involvement of the respiratory apparatus, with a picture typical of interstitial pneumonia radiologicamente similar to the pneumonia from ***Mycoplasma Pneumoniae*** ("to polished glass"); scarce objectivity semeiologica, eventually dry cough little productive.

How does the infection elapse?

La prognosis is good of rule, with evolution toward the recovery in 7-10 ggs; various but you don't frequent the complicanzes, with the important exception (30% of the cases) of the liver interest, characterized by pain to the right ipocondrio. Often fatal it is the endocardite (2% of the cases), that strikes usually the valve aortica.

Qual'è the therapy?

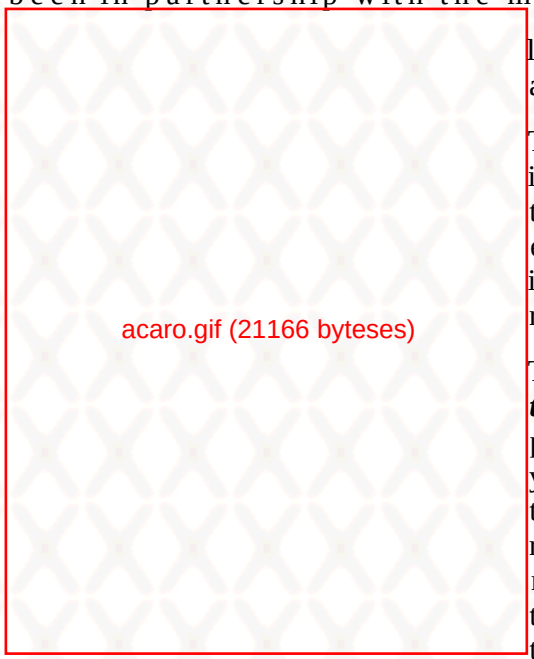
Terapia: the infection him autolimita, but it is opportune to follow a suitable
antibioticoterapia with DOXICICLINA, TETRACICLINE or CLORAMFENICOLO or,
eventually, the MACROLIDIs.

SCRUB TYPHUS

(typhus of the brushwood-fever river tsutsugamushi-fever of Japan)

INTRODUCTION

The fever of the brushwood, note as river fever or fever tsutsu, is transmitted by the puncture of the larva of the acaro trombiculide, a parasite of the roditori.Las illness is in partnership to trips toward the zones infested by the acaro as forests and areas deforestate recentemente.Pertanto the typhus of the brushwood or tsutsugamushi you/he/she can be an occupational risk, and you/he/she has been in partnership with the military service in the endemic zones.



larva esapode of ***Neotrombicula*** great ***autumnalis.E'*** as soon as 2 mms

The typhus of the brushwood is a diffused illness in the world, particularly in the Asian east south, the western Pacific, and in smaller measure The endemic Australia.E' in the Asian east south, and it is the cause of the 20% of the fevers in some regions.

The causal agent is ***Orientia (Rickettsia) tsutsugamushi***, a rickettsia.Il microorganismo persists in the rapes and in other rodents over one year from the infection, and you/he/she is transmitted to the acaris that infest the roditori.L'organismos it persists and him multiplication in the acaris, where the vertical transmission is possibile.La puncture of the acaro trombiculide he/she leaves a characteristic ulcer of the skin, that is the first symptom of the malattia.Altri symptoms they include cefalea, fever and rash.Il hard rash for a period from 9 to 18 days, but if not treated, complication or death can happen. Since ***O. tsutsugamamushi is*** also transmitted for by ovarian trans from a generation of acari to the other, the zones of endemic have the tendency to persist with to persist some ecological condzionis.

MORPHOLOGY

- The causal agent is ***Rickettsia (Orientia) tsutsugamushi*** (***Rickettsia orientalis***)
- ***Or tsutsugamushi*** has manifold, fetterses separate serologicamente what Kato, Gilliam, Karp

PATOGENESI

- The infection results from the puncture of the larva of the acaro trombiculide (***Leptotrombidium***)
- Direct transmission doesn't exist
- Period of incubation from 7 to 21 days

YOU WAIT CLINICAL

- Sudden debut
- Characteristic is an ulcer of the skin (escara) to the site of the attack of the acaro
- Linfoadenopatia
- The rash maculopapulare starts from the trunk and migrates toward the extremities

- Cefalea
- Shivers
- Pain to the back
- Profuse perspiration
- The pneumonia is common
- If not treated you/he/she can evolve in miocardite and delirium
- Death's danger if not trattata.La recovery happens when the treatment is sudden.
- The infection can be nevertheless inapparente
- More than an infection you/he/she can concern the population that he/she lives in the endemic zones

ANSWERED ANTICORPALE

- The levels anticorpali can be low or absent during the first phases of the infection.
- The levels anticorpali can delay or to annihilate himself/herself/themselves in patient essays with antibiotic
- Elevated levels or increased di IgM e/o IgG they point out recent or active infection
- the infection gives one prolonged immunity for homologous fetterses
- The infection with fetterses erterologhi gives an illness more attenuated

DIAGNOSIS

The precocious diagnosis is very important, in how much you/he/she immediately can be begun a correct trattamento.Un effective treatment it reduces the time of recupero.Però the diagnosis it is almost always difficult for the difference antigenica of the various fetterses.

TEST OF WEIL-FELIX

- It misses both of sensibility and of specificity
- In fact is the Leptospirosi that other feverish illnesses can cause a reaction to the positive Weil-Felix
- The king infection doesn't always conduct to an increase of the agglutines of the Weil-Felix

Dot Elisa

- E' a rapid method of diagnosis
- Some however they miss of sensibility and specificity

IFA

- It has a good sensibility and specificity
- It asks for a firm that furnishes the antigen
- It asks for a very experienced technical personnel for the interpretation of the results

PREVENTION

- Use of repellent against the acaris in the exposed superficial parts
- Elimination of the acaris from the populous zones

- Doxycycline is a good garrison preventivo
- An effective vaccine is still in phase of development

TREATMENT

- antibiotic to wide ghost (for es. tetracycline, chloramphenicol) I am an effective care
- A second cycle of antibiotic after an interval of 6 days (doxycyline) you/he/she can be effective in the prevention

RICKETTSIALPOX

What is the Rickettsialpox?E' a bacterial illness caused by the rickettsia ***Rickettsia*** rare ***akari.E'*** among the residents of New York, around 30 cases diagnosed in the last 10 years.

Who is to risk of Rickettsialpox?Rickettsialpox is a zoonosi, an illness transmitted by the animali.Ils bacterium has as reservoir the mice, and you/he/she is transmitted to the men by the acaris of the mouse (*Liponyssioides sanguineus*) .Ogni person that is stung by an infected acaro can get sick of Rickettsialpox.

How does the Ricketttsialpox spread?Rickettsialpox is diffused from the mice to the men with the punctures of acari of the mouse. These acaris are very small and colorless, and the puncture doesn't provoke pain.

Which are the symptoms of the Rickettsialpox?
In many cases, the first symptom is a deprived papula of pain (red, in relief lesion) that ulcer and it becomes a scar of around 0,5-3 cms .Da 3 to 7 days later, the illness begins with brividi,febbre and cefalea.Altri symptoms I am muscular pains and a similar vesicular arsh al chickenpox.

How long after the infection the illness appears that is the symptoms?
The symptoms normally appear after 9-14 days after the puncture of an infected acaro.

How diagnosis of Rickettsialpox is it set?
Rickettsialpox with an examination of the blood is diagnosed for noticing the antibodies against *Rickettsia akari.*

Which is the treatment for the Rickettsialpox?
A good atibiotico is la tetracycline recidivous .La it is not frequent.

How is the Rickettsialpox prevented?
through the disinfestazione of the mice

The fever esantematica of the Rocky Mountains (RMSF)

Description

The fever esantematica of the Rocky Mountains (FEMR) it is an acute pathology, feverish, potentially deadly caused by the ***Rickettsia rickettsii*** and transmitted by the puncture of a mint. The pathology of base is a due vasculite to a direct invasion endoteliale of the rickettsies. The characteristic clinical signs of the illness are cefalea, fever and a centripetal esantema that it often assumes an aspect petecchiale

Interested Sistemi/apparati: cardiovascular apparatus, skin and attached, apparatus muscoloscheletrico

Genetics: anybody

Incidenza/prevalenza in USA: you/they are brought around 600 new cases a year. There

is a considerable geographical variety; the most greater part of the cases are signalled in the states of the Atlantic south and in the center-south. The peak of incidence is in the late spring and in the summer

Predominant age: the tallest incidence is between the boys and the young adults, due mainly to the habits of life. All the ages can be stricken.

Predominant sex: males> females (due to the most greater activity to the open one)

Signs and symptoms:

Fever (100%)

Esantema (maculare, maculopapuloso, petecchiale) (90-100%)

Cefalea (65%)

Alone Esantema petecchiale (50%)

Cefalea, fever, esantema (50-60%)

Other sintomatologia neuropsichiatrica (40-50%)

Nausea, vomit (30-50%)

Cefalea, fever, petecchie (33%)

Abdominal pain (30%)

Mialgie (30%)

Epatosplenomegalia (30%)

Linfoadenopatia (25%)

Artralgie (10%)

Cough (15%)

You disturb to load of the central nervous system (is soporoso, is confusionale, coma, focal anomalies) (10-30%)

Causes You FEMR is caused by the *Rickettsia rickettsii* that dalla is transmitted puncture of mint ***(Dermacentor andersoni, Dermacentor variabilis).*** Very rarely from the direct inoculation of the mint in the circle ematico through wounds or through the conjunctiva

Factors of risk: Activity to the open one during the warm months. I contact with dogs

Groups to rischio:anziani, alcoholics, patient with deficit in the glucose 6 phosphate deidrogenasi.

Differential diagnosis

Viral Esantemi (measles, rosolia etc.)

Meningoencefalite (meningitis or viral encephalitis, bacterial meningitis)

Typhus, rickettsia chickenpox

Ehrlichiosi

Illness of Lyme

Meningococcemia

Leptospirosi

Examinations of laboratory:Data of laboratory aspecifici

Piastrinopenia

Normal Leucometria, increased or decreased

Anemia (light)

Iponatriemia (usually light)

Proteins and leucociti moderately increased in the liquid cefalorachidiano, glucose usually normal Increase of the time of protrombina, of the PTT; reduction of the fibrinogeno; increase of the products of degradation of the fibrina (FDP) (rare)

Specific data of laboratory:Antibodies anti-Proteus Ox-19: increased of 4 times (in the acute phase and in the period convalescenziale) or title> 1:320 (relatively specific) .Anticorpi the complement's antifissazione: increased of 4 times or superior title to 1:16 indirect fluorescent .Anticorpis: increase of 4 times or superior title to 1:64 direct fluorescent .Anticorpis on the cutaneous biopsy (currently available in all the laboratories)

Medicines that can alter the results of the examinations of laboratory: anybody

You condition that can alter the results of the examinations of laboratory: a precocious treatment can alter the answer anticorpale

Pathological finds :La principal alteration anatomopatologica is the systemic vasculite her rickettsie you/they can be underlined inside the cells endoteliali through the direct fluorescence or to the electronic microscope her vasculite petecchie you/they can be underlined on the surface of various organs (liver, encephalon, epicardio) .Si can underline secondary thrombosis and necrosis tissutale

Test special Anybody

Diagnostic for images Á. the of out of infiltrators polmonitici aspecifici, that can be noticed during a Rx of the chest of routine, rarely the diagnostic one for images is of help.

Diagnostic procedures You cutaneous biopsy is the priority examination if the immunofluorescenza is available directed or an electronic microscope

Usually The diagnosis is presumptive and founds him on a picture compatible sindromico in a patient with suggestive anamnesis in an endemic geographical zone; the confirmation is gotten with the examinations sierologici

TREATMENT

The patients with a complete clinical picture or that they are moderately in a situation important clinica you/they should be patient ospedalizzati.Il with a light clinical form ambulatorialmente you/he/she can be treated. Important E' a narrow monitoraggio to identify the possible complicanzes

General measures :Ossigenoterapia and support ventilatorio in case of complicanze pulmonary, if necessary

Good oral hygiene

Emotrasfusione in case of anemia

In the allured patients to often change the sheets

Monitoraggio tightened of the patient for possible signs of renal insufficiency

Surgical measures

Anybody

Physical activity

I rest in bed until the symptoms they subsist

Feeding: The particularly serious patients can also need a nutrition parenterale. In the other cases it is enough a checked diet and balanced for maintaining a suitable state nutrizionale

MEDICAL THERAPY

Medicines of choice

In the adults (to choose one of the followings)

Doxiciclina 200 mgs for os initially followed by 100 mgs for os 2 volte/dies for 7-10 days; same dosing ev; 100 mg/dies in case of renal insufficiency Tetraciclina 500 mgs for os every 6 hours for 7-10 days; not to use him in case of renal insufficiency Cloramfenicolo 500-750 mgs for os for 7-10 days; 20 mg/kgs ev every 6 hours (maximum 4 g/dies); same dosing in case of renal insufficiency In his/her/their children (to choose one of the followings)

Cloramfenicolo 20 mg/kgs for os every 6 hours; same dosing ev Doxiciclina 2-2,5 mg/kgs for os every 12 hours for 7-10 days; 4,4 mg/kgs ev initially, then 2,2 mg/kgs ev every 12 hours (you see Side effects) Tetraciclina 10 mg/kgs for os every 6 hours for 7-10 days (you see Side effects)

Side effects In his/her children below the 9 years the doxiciclina and the tetraciclina are not usually used for the correlated problems to the coloration of the teeth In the women in pregnancy the doxiciclina and the tetraciclina they are contraindicated because you/they can cause a serious epatopatia in his/her mother and a delay of bony growth in the fetus

Precauzioni: The patients that assume doxiciclina and tetraciclina should not compromise himself/herself/themselves to the sun to avoid phenomenons of fotosensibilizzazione The newborns or his/her children with epatopatia that assumes cloramfenicolo you/they should have the levels ematici of the medicine monitorati Interactions: the absorption of the tetraciclines can be inhibited if the assumption happens with products what the milk and the latticinis, iron for os or antiacidi containing aluminum and magnesium Medicines Currently alternativi s are not available studies on alternative medicines.

The patient's FOLLOW-UP Monitoraggio If the patients are not hospitalized, you/they should be visited every 2-3 days up to the disappearance of the symptoms Monitorare emocromo, creatinina and electrolytes Prevention Her people that are direct in places infested by mints can operate some measures preventive .Indumenti that covers the more possible the body and repellent substances to apply on the cute . After a possible exposure the whole body should be inspected for the search of mints, especially the legs, the zones inguinali, the genital outside and the life from the moment that the risk of illness aumenta with to increase some contact with the mint. The mints must have removed from the man or from the animals with extreme attention; to wear gloves or to use pliers to reduce to the least one the possible contact. To set a drop of oil, alcohol, gasoline or gas-oil on the mint. To carefully wash the hands after the operation

Complicanze :Encefalopatia, usually transitory (30-40% epileptic) Crisi, focal neurological signs (10%)

Renal insufficiency (10%) Hepatitis (10%) cardiac Scompenso congestizio (5%) respiratory Insufficiency (5% attended)Decorso and prognosis

If quickly taken care of, the prognosis is excellent with complete risoluzionedella sintomatologia in the turn of a few days and without sequences You mortalitàè a rare eventuality if pathology is quickly and appropriately curata.In case of complicanze (you see above), the elapsed one can be more serious and you/they can persist for a long time sequences termine.Sinonimi.Tifo from zecche.Annotazioni: The treatment should have beginning on the base of the clinical diagnosis and the result of the liver biopsy. The care must not be postpone even if the confirmation sierologica is not had:

Note:Following information are been translated by the site of the ARUPs Laboratories

arup.gif (2247 byteses)

Test for You RMSF fever maculosa of the rocky mountains

Teast Mnemonic: RMSF G/M

Methodology: Enzyme-Linked Immunosorbent Assay ANNULLED
Performed: Lun-Friday
Report: same day

In demand champion:

Test-tube: a 6 mL SST. (Min: 4 MLS SST)

Quantity: 1 mLs serum to 2-8°C. (Min: 0.5 mLs)
Notes: To separate the serum from the cells ASAP. To label the acute and convalescent serums; the test in parallel it is preferable therefore the champions of convalescent serum are in demand within 30 days from the arrival of the champions acuti.Per favor to label well the champion as acute or convalescent.
Unacceptable conditions: Plasma, serum lipemico, contaminated or emolizzato.
Stability: Environment: 4 hours; Refrigerated: 5 days; Frozen: 1 year

Reference Range:

COMPONENT	REFERENCE RANGE

Rocky Mountain Spotted Fever antibodies, IgG	<0.9 IVS: negative 0.9-1.1 IVS: doubt > 1.1 IVS: Positive
Rocky Mountain Spotted Fever antibodies, IgM	<0.9 IVS: negative 0.9-1.1 IVS: doubt > 1.1 IVS: Positive

Interpretation of the data:
IV = Value index

These tetses are for the search of the antibodies against *Rickettsia rickettsii.Ogni* reactivity anticorpale to *Rickettsia rickettsii* owes cross-reattiva to be considered toward the group of the fevers maculose (*Rickettsia conorii*, *Rickettsia australis* and *Rickettsia sibirica*) positive weak .Un suggests a trail exposure or infection, while a positive to tall title points out recent or current infection, but it doesn't clarify the diagnosis. The diagnosis of acute infection can be done showing a meaningful rise in the level anticorpale among you sample acute and convalescent allorquando both the tests are made in the same laboratory and at the same time.

The answer anticorpale type IgM immediately appears normally after 7-14 days from the beginning of the malattia.Il test after the exposure it is of any value without a convalescent champion taken after other giorni.Mentre the presence of antibodies IgM suggests a recent infection or

in progress, low levels of antibodies IgM can occasionally persist for more than 12 days after the infezione.Questa answered residual IgM has to be separate from the first answer making a will serums of patients 2-3 weeks later to see if there has been a change in the levels of specific antibodies IgM.

Test for the group of the fever of the Typhus

Mnemonic test: TYPHU G/M

Methodology: Indirect Immunofluorescenza performed: Giovedi', Venerdi'
brought: The same giorno,successiva sat if it needs to make the title

In demand champion:

Test-tube: a 6 mL SST. (Min: 4 MLS SST)
Quantity: 1 mLs serum to 2-8°C. (Min: 0.5 mLs)
Notes: To separate the serum from the cells ASAP. To label the acute and convalescent serums; the parallel test is preferable and the champions of convalescent serum have to be orders within 30 days from the arrival of the champions acuti.Per favor to observe well the champion as acute or convalescent.
Unacceptable conditions: Plasma, serum lipemico, contaminated or emolizzato.
Stability: Environment: 4 hours; Refrigerated: 5 days; Frozen: 1 year

Reference Range:

COMPONENTS	REFERENCE RANGE

Typhus Fever Antibody, IgG	<1:64: negative 1:64-1:128: it suggests an infection but we don't know if actual or pregressa > 1:256: presumptive evidence of a recent or current infection from organisms of the group of the Typhus Fever group
Typhus Fever Antibody, IgM	<1:64: negative > 1:64: presumptive evidence recently or current infection from organisms of the group Typhus Fever group

Interpretation of the data:
These tests are for the antibodies anti ***Rickettsia typhi.*** Whatever reactivity a ***Rickettsia typhi*** has to be considerata group reactive towards the Typhus Fever group (***Rickettsia prowazekii***). Sieroconversione or an increase of the titles of IgG and acute and convalescent IgM than at least 4 times it is considered evidence sufficiente of a current infection or recente.L'apparizione of an answer anticorpale of the type IgM it happens after 7-14 days the beginning of the true illness and propria.Fare this test after the exposure it doesn't have any value without a champion in convalescence taken in the days seguenti.La presence of a tall title of antibodies IgM it suggests an infection in action or recent, but low levels of antibodies of the type IgM they are able nevertheless to also persist for 12

months after the infezione.Questa residual answer IgM owes therefore to be separate from a precocious answer IgM to the infection making a will later the serums of patients 2-3 weeks to underline changes in the levels of specific antibodies IgM.

Therefore the thing most important to do for the diagnosis of rickettsiosi it is the demonstration of an increase in the antibodies when serums of the acute and convalescent phase are compared in the same seduta.Il title absolute anticorpale it is not of great aiuto.Per I increase some title him it intends an increase than at least 4 times of the title between a serum and the other.

time of appearance of the antibodies in the serum of patients

illness	CF fixation of the complement	Weil Felix	Agglutination
Group of the fever bottonosa	8-10 days	5-12 days *	unknown
Fever Q	8-14 days	anybody	5-8 days
Epidemic typhus and walls up	7-9 days	7-14 days	5-7 days
Typhus of the brushwood	unknown	10-14 days	unknown

*in the rickettsialpox the antibodies of the Weil Felix test don't appear.

The rickettsiosi and the Encefalopatia mialgica (ME):co-cause or factor?

To the recent lecture medical estate to Sidney, Australia in February 1999, the dssa Cecile Jadin, of Johannesburg, South Africa, presentava an interesting theory that explored the relationship between the rickettsies and the neurological illnesses what her Me and the sclerosis multipla.La dssa Jadin valuto' the patients with Encefalopatia mialgica for the presence of chronic rickettsiosi, observing certain similarita's among the chronic infections from known Rickettsia and the Encefalopatia mialgica particular .In has been observed the tall percentage of recovery after a specific therapy anti rickettsie.Usando the Micro test of agglutination of Giroud, the doctor mostro' as there pits a tall prevalence of kind of positive rickettsie to the test in the patients with ME.Fra the kinds made a will there were, R.Prowazeki (epidemic typhus), R. Mooseri (typhus walls up), R.Conori (fever bottonosa), Coxielia Burnetti (fever Q) and le Mole - Rickettsia Chlamydiae.Questi bacterias are parasitic that they grow and they reproduce him inside the cells animali.Come the viruses, the rickettsies they are very small, but unlike the viruses they contain the code genetico Dna and RNA, the plasmatic membrane, the ribosomis and the enzymes that participate in the streets metaboliche.Molte rickettsie they have a form of bastoncello or coccoide with the wall

Gram negativa.Questi bacterias they have the caratteristica to elude the mechanisms of defense cellulare.Sfuggendo the fagocitosi they reproduce him in the space cellular called cytoplasm with a trial called fission binaria.Le infected cells can burst and to release multiple copies of the batteri.Les Rickettsie they are very different from the bacterias as it regards physiology and metabolismo.Mancano of the by glicolitica and don't use the glucose as source of energia.Invero they use the ATP of the cells you entertain (energetic molecules), the intermediary ones of the sour tricarbossilicis, and other nutrienti/coenzimi.La seeks on the Rickettsies you/he/she has postulated that the organism is often found in patient not sintomatici.Queste infections asintomatiche can last for years. But the thick organism causes serious pathologies during this period because of a concomitant viral infection, bacterial, stress and pollution that immunodeficienza and the malattia.La determine it seeks French you/he/she has pointed out that the rickettsie infections can produce an immunodeficienza, included the activation of the virus EBV and of the CMV citomegalovirus.Lo been of immunodeficienza changes with the overcoming of the infection from rickettsie.Durante the presentation of the dssa Jadin it gave some suggestion on as to treat the patients with Encefalopatia mialgica infected by the rickettsie.Ils treatment consists of 7-12 days to tall doses of tetracicline to the mese.Le rickettsie infections I am a lot of piu' toxic epato of a long therapy with tetracicline.Pazienti that has an abnormal liver function they often improve with an antibiotic therapy. An addition to these considerations included the use of the probenecid for some severe reactions Herxheimer (the liberation of bacterial toxins from the killing of the bacterias in the first months of therapy ndr), the alternation of various kinds of due tetracicline to the bacterial resistances and multiple fetterses, the addition of other antibiotic (chinoloni, macrolidi or the metronidazolo) and an antibiotic therapy had been prolonging of the duration for 6 months to 3 anni.Las therapy has disclosed two categories of patients, the group of patients that you/they easily recover, that you/they probably have only rickettsia infections and the group that it recovers less quickly that it would have additionally a rickettsia infection to other factors that inhibited the recupero.Il dr Philippe Bottero that altresi' studies the relationships between rickettsie infection and their treatment in the patients with Encefalopatia mialgica it brought the fact that the majority of its patients had for a long time problems vascular plants and periferici.E' known that the rickettsies sopravvivono in the cells of the sistem network endoteliale and in the walls of the vases sanguigni.Altresi' Bottero has found a very satisfactory answer to the therapies with antibiotic. (Drew Martin dmartin@mindspring.com)

Chronic rickettsial and mole-rickettsial infection causes as of chronic illness, 1993

Kind Dr. Bruno
I make her my most sincere compliments for the initiative (notes of microbiology) and for the quality of the contents.
The fever bottonosa of the Mediterranean is endemic in Italy centromeridionale and islander, the most greater number of the cases they are notified in
Sicily, this form of rickettsiosi is presentenei in all the wet countries from the Mediterranean and especially in Spain, in Israel it is present a form slightly different.
Such illness not cronicizza never and it recovers after a suitable treatment antibiotic (but also without) it is an illness that in the elderly subject or with pathology to load of the microcircolo can give serious complicanze especially to load of the kidney and the SNC.

Available molto,sono has not dwelled however for further explanations

Talk to you soon

Prof Anthony Cascio - University in Palermo (cascioa@unipa.it)

Mr Bruno,

Thank you for your message. The believe the rickettsial infections Khan be chronic and to patient Khan positive be dark for than one organism in this family. You Khan also be postive without symptoms. To high test level most likely means high level of antibody and is related to antigen quality.

Myalgic Encephalopathy is an illness of unknow causes, it most notably multiple resembles sclerosis and in burdens cases AIDS. To dark learn visit the NATIONAL CFIDS FOUNDATIONS WEBPAGE AT HTTP://WWW.CFIDSFOUNDATION.ORG AND
http://www.cfidsfoundation.org/encephalopathy.htm

The would like to ask what your interest in this information is intended?

Expensive Take
Drew Martin (dmartin@mindspring.com)

(This document is the first one on the rickettsies in Italian appeared on Internet.Fa it departs of the situated www.geocities.com/hotsprings/spa/5276)

Tetracyclines

Dr. Cecile Jadin's Papers are now available in full Click Here

Updated on:
06/11/2000

Tetracyclines are grouped as follows:

Macrolides

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Updated on: 06/13/2000

Azithromycin [Zithromax]

- Adverse Reactions
  - 1% to 10%: Gastrointestinal: Diarrhea, nausea, abdominal pain, cramping, vomiting (especially with high single-dose regimens)
  - <1%: Ventricular arrhythmias; fever headache, dizziness; rash, angioedema; hypertrophic pyloric stenosis; vaginitis; eosinophilia; elevated LFTs, cholestatic jaundice; thrombophlebitis; ototoxicity; nephritis; allergic reactions
- Pharmacodynamics/Kinetics
  - Half-life, terminal: 68 hours

Clarithromycin [Biaxin]

- Adverse Reactions
  - 1% to 10%: Headache; diarrhea, nausea, abnormal taste, dyspepsia, abdominal pain
  - <1%: Ventricular tachycardia, torsade de pointes; decreased white blood count, elevated prothrombin time; elevated AST, alkaline phosphatase, and bilirubin; elevated BUN/serum creatinine
- Pharmacodynamics/Kinetics
  - Half-life: 5-7 hours

Dirithromycin

- Adverse Reactions
  - 1% to 10%: Central nervous system: Headache, dizziness, vertigo, insomnia, Dermatologic: Rash, pruritus, urticaria,Endocrine & metabolic: Hyperkalemia, Gastrointestinal: Abdominal pain, nausea, diarrhea, vomiting, dyspepsia, flatulence, Hematologic: Thrombocytosis, eosinophilia, segmented neutrophils, Neuromuscular & skeletal: Weakness, pain, increased CPK, Respiratory: Increased cough, dyspnea
  - <1%: Palpitations, vasodilation, syncope, edema, anxiety, depression, somnolence, fever, malaise, dysmenorrhea, hypochloremia, hypophosphatemia, increased uric acid, dehydration, abnormal stools, anorexia, gastritis, constipation, abnormal taste, xerostomia, abdominal pain, mouth ulceration, polyuria, vaginitis, neutropenia, thrombocytopenia, decreased hemoglobin/hematocrit; increased alkaline phosphatase, bands, basophils; leukocytosis, monocytosis, Increased ALT/AST, GGT; hyperbilirubinemia, paresthesia, tremor, myalgia, amblyopia, tinnitus, increased creatinine, phosphorus, epistaxis, hemoptysis, hyperventilation, hypoalbuminemia, flu-like syndrome, diaphoresis, thirst
- Pharmacodynamics/Kinetics
  - Half-life: 8 hours (range: 2-36 hours)

Erythromycin:

- Adverse Reactions
  - >10%: Abdominal pain, cramping, nausea, vomiting
  - 1% to 10%: Oral candidiasis; cholestatic jaundice; phlebitis at the injection site; hypersensitivity reactions
  - <1%: Ventricular arrhythmias; fever; rash; hypertrophic pyloric stenosis, diarrhea, pseudomembranous colitis; eosinophilia; cholestatic jaundice (most common with estolate); thrombophlebitis; allergic reactions

Lincomycin

- Adverse Reactions
  - 1% to 10%: Gastrointestinal: Nausea, vomiting, diarrhea
- Pharmacodynamics/Kinetics
  - Serum half-life, elimination: 2-11.5 hours

Troleandomycin

- Adverse Reactions
  - >10%: Gastrointestinal: Abdominal cramping and discomfort (dose-related)
  - 1% to 10%:Dermatologic: Urticaria, rashes; Gastrointestinal: Nausea, vomiting, diarrhea
  - <1%: Rectal burning, cholestatic jaundice

Roxithromycin (a semi-synthetic)

- Adverse Reactions
  - 4% experienced side-effects
- Pharmacodynamics/Kinetics
  - Serum half-life, elimination: 10.5 hours

Metronidazole

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Updated on: 06/11/2000

Metronidazole *(Flagyl)*

- Adverse Reactions
  - >10%:Central nervous system: Dizziness, headache; Gastrointestinal (12%): Nausea, diarrhea, loss of appetite, vomiting
  - <1%: Ataxia, seizures, disulfiram-type reaction with alcohol, pancreatitis, xerostomia, metallic taste, furry tongue, vaginal candidiasis, leukopenia, thrombophlebitis, neuropathy, hypersensitivity, change in taste sensation, dark urine

Food pH

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Drinks pH

pH	Food
4 -6	Beers
6.0	Mountain Dew
6.5	Coke Classic
6.6	Concord Grape Juice, Orange Juice, Seltzer Water
7.0	Water
9.0	Coffee

Hemex Protocol

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Updated on: 01/14/2000

This page contains links to people's experiences with the Hemex Protocols (both positive and negative). If you have a first hand experience that you wish to share, please email: cfs@folkarts.com

On other sites:

- Saran Lyon,

Lyme and CFS

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Updated on: 01/21/2000

An interesting 1993 letter from an MD about Lyme and CFS:

- http://www.abcjb.com/news/medother/antibiotic_responsive_cfids.htm

This announcement may be copied and distributed only in its entirety.

ANNOUNCEMENT

The Suncoast CFS/FMS/MCS/GWS Support Network has added Lyme Disease to its name and to its focus.

We have made this change because, even in this supposedly low-risk area, ***all of the local patients tested in their Ampligen workup to rule out Lyme Disease were found to have Lyme Disease*** *based on the* ***LUAT test****. Additionally, not all of those positive have migrated from high risk areas.*
(Risk areas information can be found at http://www.geocities.com/HotSprings/Spa/6772/lyme.html)

(The LUAT test is performed by Igenex (http://www.igenex.com. This URL is provided for informational purposes only and should not be considered as an advertisement or endorsement or their products or tests.)

Additionally, this makes the Suncoast Support Network one of the few support groups for Lyme Disease in Florida.

In my recent literature searches, it appears that if a person tests positive, Lyme Disease could be the cause of some or all of the CFS/FMS symptoms or is, at least, a perpetuating factor for those symptoms.

I have started adding Lyme Disease information to my website at

- *http://www.geocities.com/HotSprings/6028/ or its mirror site at*
- *http://www.geocities.com/marilynk.geo*

I hope that CFS/FMS patients consider being tested for Lyme Disease and find the information provided at my website helpful in making such a decision.

Suncoast CFS/FMS/MCS/GWS/Lyme Support Network meeting information:

First Sunday of every month, 2-4PM, 702 S. Kings Ave., Brandon, FL
Third Sunday of every month, 2-4PM, University Community Hospital Auditorium, Fletcher Ave. and Bruce B. Downs Blvd., Tampa, FL

Information on upcoming speakers can be found at the Suncoast Support Network Home Page at http://www.geocities.com/marilynk.geo/suncoast.htm

Marilyn Kerr RN
Pres., Suncoast Support Network

December 1999

From Co-Cure Announcements http://www.co-cure.org

News Groups

Dr. Cecile Jadin's Papers are now available in full Click Here

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Updated on: 01/24/2000

<% PW=trim(lCASE(Request("PW"))) ET=Request("EmailTo") EF="" ES=Request("EmailSubject") EB=Request("EmailBody") If len(PW) > 0 then Select Case PW case "kenl@folkarts.com" EF="AmpligenAnonB" case else Response.Write "

Not registered - email will not be sent

" end select End if if len(EF) > 0 then set objEMail = Server.CreateObject("CDONTS.NewMail") objEMail.To = ET objEMail.From = EF objEMail.subject = ES objEMail.body = EB+ Chr(13)+Chr(10)+"Sent from http://www.folkarts.com/AnonEmail.asp" response.write "

Email was sent

" objEMail.send() ET="":EF="":EB="":ES="" set objEMail = nothing else response.write "

Please complete the form below" end if %>

An excellent source of support are 'Lists' or News Groups. The following lists are active and may assist you with both information and allow you to vent to those who have been there and understand. To *qualify* to be listed below -- a list need at least 100 members...

Treatment Oriented News Groups

The following groups are focused on one specific treatment protocol. It allows experiences and side effects to be shared and discussed.

- ☐ CFS-Ampligen Ampligen - a new experimental drug.
  http://www.onelist.com/community/CFS-Ampligen
- ☐ CFS_Mycoplasma Antibiotic treatment for CFS caused by mycoplasma
  http://www.onelist.com/community/cfs_mycoplasma/
- ☐ **Anticoagulant** Therapy Support: (or Immune System Activation of Coagulation)
  http://www.egroups.com/group/atsg/info.html

http://www.onelist.com

There are 66 lists there... the most important ones are:

- ☐ C-act
  C-ACT members serve as the grassroots link between chronic fatigue and immune dysfunction syndrome* (CFIDS) lobbyists and members of Congress. C-ACT members provide "proof" to members of Congress and others working in the federal government that there a lot of real people who care about CFIDS. By telling of their own experiences with the disease, persons with CFIDS (PWCs) and those who care about them provide compelling evidence that CFIDS warrants a swift federal response. 765 members
- ☐ cfcured
  We discuss about toxins, water, supplements, candida, arthritis, candidasis, fybromyalgia, PMS, diet, parasites, cleansing, massage, acupuncture, homeopati, acupressuer, Yumeiho therapi, constipations, gallstones, amalgam, fluoride, nickel, root canal, cavitations, vegetable juices, flaxseed oil, Dr.Budwig diet, parasite cleanse, bowel cleanse, and many other things 122 members
- ☐ CFSFMExperimental
  An information and discussion list for chronic fatigue syndrome and fibromyalgia patients who are taking experimental medical treatments or who are interested in learning about them. 281
- ☐ ampligenmeeting
  A news/announcement list for information regarding the upcoming Exploratory Meeting for the purpose of establishing a Community Advisory Board for the treatment of Chronic Fatigue Immune Dysfunction Syndrome with Ampligen. 102 members

Research Announcement Lists

Latest publications and news list of CFS and FM

☐ Co-Cure,
http://listserv.nodak.edu/scripts/wa.exe?SUBED1=co-cure&A=1&D=0&H=0&O=T&T=1

General Discussion

☐ alt.med.cfs

This is a very active moderated group that is available via news reader (you can read and browse just the topics that interest you)

☐ alt.med.fibromyalgia

Fibromyalgia is believed to be the same underlying illness as CFS but with a different set of symptoms. The pain of this illness is ascribed to be the cause of insomnia and tiredness.

Quick Sign up [Not working yet]

Your Email: {YourEmail}@{yourDomain}

- Receive ⊙ :
  - A daily summary ⊙
  - Individual messages: ○
- Stop Receiving ○

Sign me up for the lists checked above

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

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Updated on: 02/08/2000

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Activism Mailer

From

To: CDC-Oversight: Subcommittee on Labor, Health and Human Services, and Education

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THE EPIDEMIOLOGY OF MYALGIC ENCEPHALOMYELITIS (ME) IN THE UK – 1919-1999

INTRODUCTION

Although ME is one of the commonest chronic neurological diseases in the UK today no official government sponsored statistical evaluation has yet been made. We therefore have to rely on individual studies which suggest a point prevalence of some 300,000 sufferers. The illness mainly affects young to middle aged adults with a secondary peak at puberty, after which female subjects significantly outnumber males throughout the childbearing years. Chronic disablement affecting the most socially, economically and educationally active sector of the population, many of whom have lost education, training or the ability to work, will pose grave economic problems for the future.

A comprehensive demographic survey (including age, gender, occupation, geographical location, length and severity of illness) is urgently needed.(1). Forward planning based on rational assessments of diagnostic, therapeutic, preventative medical strategies and/or the social, financial and educational support required, is impeded by lack of this vital information. Currently, sufferers from ME require access to NHS and special educational facilities, as for those with any similar chronic illness. Without an epidemiological study no government can hope to stem the true health costs of this disability, to say nothing of delayed diagnosis poor management and the expensive or inappropriate prescription of drugs. Realistic long term planning to provide the care and assistance needed for stabilisation of this illness must surely offer the most cost effective strategy.

WHAT IS ME/CFS?

DESCRIPTION AND CLASSIFICATION

ME/CFS is a syndrome (a linked group of symptoms) which usually follows a common and apparently trivial virus infection (often described as a self limiting respiratory/gastro intestinal upset with headaches, malaise and dizzyness from which the majority of people recover). However, after an interval, a second more serious multi system disease can develop with variable involvement of cardiac or skeletal muscle, liver, pancreas, lymphoid or endocrine organs. Nevertheless, ME is primarily a neurological illness with well documented encephalitic

features and classified as such by the WHO international classification of diseases (ICD 10).

2.

CHARACTERISTIC SYMPTOMS INCLUDE;[2,3,14]

(a) An episodic and unpredictable state of central nervous system exhaustion, triggered by mental or physical over exertion often with delayed onset and prolonged recovery.

(b) A profound neuroendocrine disturbance affecting the hypothalamic/pituitary/adrenal response to stress as well as the normal homoeostatic control of sleep and temperature rhythms, of fluid balance, of growth and reproductive hormones and of emotional stability.

(c) Defective control by the autonomic and sensory nervous system of cardiovascular and gastro-intestinal function and of pain and other tactile sensations.

(d) Brain dysfunction includes cognitive problems such as defective attention span and memory, impaired verbal and mathematical ability as well as difficulties with balance, fine motor control and spatial perception and disturbance of auditory and visual function.

(e) Musculo-skeletal problems affect some 70% of patients, one third of whom have an abnormal metabolic response to muscular exercise.

(f) The disease pursues a chronic, relapsing and unpredictable course.

DIFFERENTIAL DIAGNOSIS FROM "CHRONIC FATIGUE SYNDROMES"

ME has a characteristic clinical presentation, an unique neuroendocrine profile and a distinguishing epidemiological pattern which clearly separate it from some 30 other pathological and physiological fatigue states. Supplementary evidence supporting the clinical examination and symptoms (a-f above) may be obtained from:

(a) Tests of adrenal response to stress.[4.]

(b) Estimation of pituitary hormones.[5.]

(c) Tests of autonomic function including tilt table testing for cardiovascular response.[6,7]

(d) Brain imaging and neuropsychometric testing.[8,9]

(e) Response to subanaerobic exercise tests.[10.]

(f) Extension of clinical records and observation periods from weeks and months to years.[2,11,12,13,14]

3.

HISTORICAL ASPECTS – THE PARADOX AND ENIGMA OF "DISEASES OF AFFLUENCE"[11,12,15,16,17]

ME is not a new disease and, until the late 1950s, it was generally considered to be an atypical, non paralytic or milder form of poliomyelitis. However, a new awareness of its disabling potential was first recognised in the more affluent communities of North America and Europe, then engaged in vast housing and public hygiene improvements accompanied by major redistribution of population from inner cities to suburbs. These laudable plans paradoxically destroyed the age-old circulation of asymptomatic, but naturally immunising childhood infections, while the burden of severe disease (with previously unrecognised complications) fell upon non immune adolescents and adults. These "diseases of affluence", of

which poliomyelitis is the classic example, are largely confined to geographically temperate climates, where bowel and respiratory infections are seasonally interrupted, compared with tropical areas, many of which still maintain solid adult herd immunity by reason of continuous exposure to infection and lesser standards of hygiene. It was these social changes, rather than any sudden viral mutation, that led to another 20th century phenomenon – major epidemics, then pandemics of polio, followed seasonally and sequentially by the milder but more chronic and relapsing form of illness, previously referred to as "atypical" or "non paralytic" polio, but following the major Royal Free Hospital epidemic in 1955, as "Benign" Myalgic Encephalomyelitis. Some 70 outbreaks are clearly recorded in medical literature, 38 before successful mass polio immunisation in selected countries interrupted the natural circulation of 3 polio virus strains in 1965. During the next 30 years, a further 32 epidemics of ME have been recorded along with a rising incidence of the disease. This rise culminated in a 5-8 fold increment world wide, during the period 1980-1989, since when it has remained an endemic disease with periodic epidemic potential. 40 years ago ACHESON(17.), in his seminal review of 14 geographically widespread epidemics, had already suggested that the illness follows infection by a group of related agents.

EPIDEMIOLOGICAL DATA RELATING TO ME IN THE UK

Apart from the USA, no government has yet adequately funded epidemiological studies of ME and, in the UK, we still have to rely upon data based on a relatively small number of subjects collected by individual workers and mainly related to medical referrals. These have the defect of excluding the many patients (especially the severely affected, the house bound and those disillusioned by conventional medicine) who are seldom seen by doctors.

4.

LOCAL PREVALENCE STUDIES 1975-1997

Figures for point prevalence of ME in the general population of the UK are unknown, therefore those derived from local studies (below) can only be speculative:

Location & Number of Subjects	Years Of Study	Local Prevalence (Range)	Projection to Adult General Population	Source Of Data	Notes
A) Mainly Southern England (420)(2)	1975 -1987	272/100,000 -545/100,000	155-300,000	GP referrals to hospital infectious diseases facility	Three times the local prevalence of MS
B) North Scotland Including Highlands And Islands (289)(18)	1990	0.3 – 2.7/1,000 (average 1.3/1,000)	17 – 150,000 74,000	Postal Survey Of GPs using hospital Infectious Diseases facility	Epidemic of BORNHOLM DISEASE in Area of highest Prevalence Within past 5 years

C1) Southern England & Midlands (27,327 School staff) (19)	1991-1995	400-700/ 100,000 (average 500/ 100,000)	230-400,000 285,000	medical certification of long term sickness absence in local education Authority areas	Figures highly variable in different locations with evidence of clustering
C2) Same Location 333,024 (school pupils)(19)	1991-1995	38 – 102 / 100,000 (average 70/ 100,000)	Projection to general population of school children 4,000 – 9,000 6,300	"	"
D) 4 schools In <u>one</u> of The locations Studied in C2 Above (32,924 school pupils (20)	1996/1997	0.05% -0.1% individual schools (combined schools 70/100,000)	Projection as Above 49 – 9,000 6,300	Postal Questionnaire addressed to Local education Authorities, Community Paediatricians & Child psychiatrists and to parents of children on "special needs" register	As above with Possible Evidence of Clustering

5.THE INCIDENCE OF ME IN EPIDEMIC AND PANDEMIC YEARS

Apart from 2 UK studies and one each in Canada and the USA, little information is available about the chronic aspects of ME or the significant numbers of patients involved. Although many incidence studies have been published for individual hospital and other epidemics, there

has been no serious attempt at nationwide or long-term follow up of those affected. Some comparisons are now made between 3 UK studies and one Canadian, during the period of the recent North American pandemic of ME between 1980 and 1989.

Figure 2

STUDY (A) PERIOD OF STUDY: 1954-1992; SOURCE OF INFORMATION: Enteroviral related illness over 4 decades in General Practice; GEOGRAPHICAL AREA: Widespread but mainly Northern England and Scottish Borders; SUBJECTS : 5,348 PATIENTS WITH ENTEROVIRAL INFECTIONS; categories of illness: G1: Acute enteroviral infection, G2: Recurrent enteroviral infection, G3/4: patients with single or multiple end organ pathology including those with ME. G5: Deaths (all within category G3/4)

Figure 1

STUDY (B) PERIOD OF STUDY: 1965-1998; SOURCE OF INFORMATION: Patients with all types of infection referred to a hospital Infectious Diseases facility; GEOGRAPHICAL AREA: Widespread but mainly Southern England and the Midlands. SUBJECTS: Of some 9,000 patients referred for investigation, 3,031 were considered to have ME as the main diagnosis or, in conjunction with other illness.

STUDY A[12] Figure 2 **STUDY B[2] Figure 1**

Patients with ME **Patients with ME**

Decades of Attendance Charted by Year of Onset

		G1	G2	G3/4	G5		
(1)	1954-64	315	0	28	17 (7%)	(1) 133 (5%)	
(2)	1965-74	659	5	63	22 (14%)*	(2) 239 (9%)*	
(3)	1975-84	784	131	263	46 (23%)	(3) 800 (30%)	
(4)	1985-92	189	565	540	19 (56%)**	(4)1480 (55%)**	
TOTAL	(5349)	3560	701	894	104	TOTAL (2712)	

Percentage of Illness (In Epidemic (2)* and Pandemic Years (4)**)

Figure 4

STUDY (C)[21] PERIOD OF STUDY 1935-1992; SOURCE OF INFORMATION: Patients referred to a medical clinic in Ottawa, Canada; GEOGRAPHICAL AREA: Widespread in Canada and

North America; SUBJECTS: a pilot study of 1,826 patients with chronic ME which also provides detailed demographic information.

INCIDENCE OF ME IN CANADA (BY DATE OF ONSET OF ILLNESS) DURING NORTH AMERICAN PANDEMIC.

(1)	PRIOR TO 1977	105	6%
(2)	BETWEEN 1977 and 1979	451	25%
(3)	BETWEEN 1979 and 1992	1,260	69% ** pandemic years
	TOTAL	1,826	

Figure 3

STUDY (D)[14] PERIOD OF STUDY: 1995-1998. SOURCE OF INFORMATION: Participants sought through patient organisations and individual physicians. Data collected by means of self report questionnaires. (Evidence of chronicity required:- illness not less than 2 years duration with specified level of disability eg. inability to leave home without assistance).

GEOGRAPHICAL AREA: Widespread in UK

	YEARS OF ONSET OF ILLNESS	NUMBER OF CASES	
(1)	1943-1980	43 (19%)	
(2)**	1981-1984	28 (12%))	
(3)**	1985-1986	26 (12%))	158 (70%)
(4)**	1987-1988	36 (16%))	** total in
(5)**	1989-1990	40 (17%))	pandemic years
(6)**	1991-1992	28 (12%)	
(7)	1993-1994	24 (11%)	
	TOTAL	**225**	

7.

COMMENT:

Though the information in all Incidence Studies (A-D) was collected independently, they reveal a staggering increase in the incidence of ME between 1965-1990, when between 50% and 70% of the patients recorded in individual studies fell ill or presented to the investigator. Though records in Study A were entered by date of attendance and might reflect medical or lay press publicity, those in studies B-D were recorded by date of onset of illness and were free of this bias. The smaller increases between 1965 and 1975 coincided with widespread epidemics in the UK of BORNHOLM DISEASE[12,18] (epidemic pleurodynia – a well recognised enteroviral infection associated with neurological and cardiac complications) of which some 340 cases were recorded in Study A, representing 18% of patients with ME, their family members or social contacts. The major increases in all studies between 1980 and 1990 coincided exactly with the North American pandemic years, followed by a decline in incidence to that of the late 1970s in 1998 but by no means to the earlier base levels.

LABORATORY EVIDENCE OF ENTEROVIRAL INFECTION BETWEEN 1980 AND 1990[2,12]

	Enterovirus Serology	Immunology	Comments
1. Dowsett/Ramsay: (420 patients) (a) 205 seen before 1985 (b)124 seen when the more accurate	Neutralisation Tests 68/205 (33%) indicative of recent infection EVIgM +ve 38/124 (31%)	Circulating Immune Complexes 128/276 (46%) positive (evidence of antibody/	16 Patients (13%) when retested annually for 3 years, were shown to be persistently or intermittently positive for (EV) IgM * Local Blood Transfusion Service

enteroviral (EV) IgM test became available	evidence of recent/active infection *controls (12%)	complement activation)	
1. Richardson: (898 patients)	EVIgM +ve 295/898 (33%) *controls 4-9%	146/898 (16%) positive	*Local Blood Transfusion Service

8.

COMPLEMENTARY AND SUPPORTIVE EVIDENCE OF NEURO HORMONAL DISTURBANCE AND IMPAIRED CENTRAL NERVOUS SYSTEM METABOLISM IN PATIENTS WITH PERISTENT ENTEROVIRAL INFECTION – 1994-1998[9]

RICHARDSON published evidence of hypothalamic disturbance in 30 patients with ME (95 males and 25 females) who had persistent enteroviral infection, by means of Buspirone stimulation of the pituitary and adrenal hormones, prolactin and cortisol. This study was followed up 3 years later by the correlation of this biochemical disturbance with SPECT Scan evidence of impaired metabolism in the hypothalamic and related areas of the brain in 39 patients. More sophisticated methods of detecting persisting enteroviral infection were available than in earlier studies (eg. PCR)

THE SEASONAL AND GEOGRAPHICAL CHARACTERISTICS OF ME[2]

These rely entirely upon the interaction between climate and hygiene. The majority of patients (between 60% and 80%) report that a “flu-like” illness ushers in the onset of ME, which is more common in Summer and Autumn than in Winter and Spring (63% compared with 37% in a study of 225 patients). The geographical distribution of the disease bears a striking resemblance to that of polio, multiple sclerosis and several other disabling neurological conditions which are uncommon in developed or tropical areas of the world but share the

same incidence as that of the adopted country in ethnic minorities who migrate to developed areas after childhood.

GENDER AND AGE OF ONSET OF ILLNESS[2,14,21] Figures 5-7

The commonest age at onset in both sexes lies between the third and fourth decade (30-45 years). However, there is a secondary peak at puberty, more marked in females than in males. From this point for F:M ratio rises sharply from near unity before puberty to a 3:1 ratio (or 5:1 in the more severe and prolonged cases) throughout childbearing years until the menopause. Clearly, when girls enter the endocrine differentiation of an adult female they also undergo hormonally determined changes in T cell response to infection and may therefore suffer a more severe and chronic illness (with a greater liability to auto immune disease) than males.

After 50 years of age in both sexes, there is a gradual decline of new illness. However recent experience (combined with prolonged follow-up of sufferers who may well have stabilised over 30-40 years) indicates that a proportion may relapse with new muscular weakness and other late effects, indistinguishable from those described in the post-polio syndrome and register as “new” patients in ME charity self help groups.

OCCUPATIONAL RISKS OF ME

Apart from a single epidemiological survey of school staff, 3 hospital based infection studies and innumerable small anecdotal surveys made by patients (e.g. survey No.6, below), no serious investigation of occupational liability to ME has been published in the UK. However, a major epidemiological study of patients presenting to a Canadian Medical clinic is available for comparison;

1. CANADA 1935-1992(1,826 subjects).[21] The seven most commonly reported occupations at onset of illness (with risk factors in parenthesis) were:-

a) nursing (3.59). b) teaching (3.26). c) medical laboratory

technicians (2.86). d) secretaries (0.88). e) bank clerks (0.48).

f) bookkeepers (0.28).

Male employees in teaching had only half the risk of developing ME compared with their female colleagues while the figures for all occupations suggest that contact with infections as in health care and teaching, presents a greater risk than in other stressful jobs such as Banking.

2. UK 1991-1995 (27,327 school staff).[19] The prevalence of ME in this population bears a close relationship to that reported from Canada – that is, between 400 and 700/100,000 (average 500/100,000) or some 2 to 3 times the average in the general population

3. UK – 1990 (289 patients with ME)[18] Postal Survey of Doctors Referring Patients to hospital Infectious Disease facility; teachers or students and hospital staff (29%), skilled or unskilled manual 17%), not employed (16%), homecare (13%), Service industries (11%), Secretarial (9%), Other Professional (5%)

4. UK 1974-1987 (420 patients with ME)[2] referred to a hospital Infectious Diseases facility; Teachers/Healthcare (41%); clerical and administrative (26%); skilled or unskilled manual (21%); not gainfully employed including children and pensioners and homecare (12%). Corresponding figures for 6,000 patients attending with other disorders: 4%, 21%, 12%, 63%.

5. UK Hospital Infection Surveys[2,23] - area and years of study as in (4): Staff locations at highest risk of ME –

Theatre and intensive care; paediatrics, midwifery, dental and eye clinics, physiotherapy; Mental subnormality and chronic care facilities. OTHER OCCUPATIONS OUTSIDE HOSPITAL: Ambulance and paramedical; parents/playgroup attendants; prison and similar institutional staff; sewage and water workers; participants in recreational water sports; visitors to the tropics.

6. UK POSTAL SURVEY OF 1,883 holiday makers (mainly to the Mediterranean region) in 1992.[22.23] 882 of the travellers (49%) developed an initial respiratory/gastrointestinal illness of whom 474 (52%) later suffered from ME – a 25% attack rate

THE PROGNOSIS OF ME[2,11,12,14]

Of 420 patients (41% of whom presented early to a hospital Infectious Diseases facility – between 10 days and 2 years after onset of their illness), 31% were judged to be improving; 20% were still fluctuating between relapse and remission; 25% had achieved a steady level of disability; 24% had experienced no remission or were deteriorating.

ME is not a short term illness and improvement does not proceed in linear progression. After an initiating virus infection, which commonly circulates in the local population (often asymptomatic or so trivial as to be overlooked) the vast majority of those exposed make a complete recovery. Others may enter an indeterminate period of fluctuation before complete resolution. After a variable period, some 6-10% of those exposed may develop neurological (encephalitic) complications, from which recovery is still possible. However, others proceed to a more serious, multisystem disease involving cardiac, endocrine and other end organs which, with careful management, may yet stabilise at a level of disability which permits gradual return to education, training or work. Unfortunately, some 25% of patients enter a slow but inexorable downhill course over many years. The death rate in this group is about 10%, mainly from end organ failure (eg myocarditis or pancreatic disease) which is certified as such, rather than from ME. Others succumb to age related degeneration or malignant growths but some, tragically, to preventable causes such as suicide and inappropriate management of undetected complications (such as myocarditis).[12,24] Unfortunately, there appears to be a lifetime risk of relapse in all patients.

11.

FACTORS AFFECTING PROGRESSION OF THE ILLNESS

(A) PERSONAL: The patients immune status at onset (though prior contact with the initiating infection in earlier years may be protective); The type of infecting agent, depending on its potential for inducing neurological, and other serious complications; the degree of exposure to secondary cases in family and social contacts. Though suspicion has been cast on genetic risk and a significant number of patients (between 31% and 90% in different surveys)[2,21] report 2 or more cases in the family, clustering of cases is equally common in the staff of schools and health care institutions, suggesting cross infection rather than genetic susceptibility to be the underlying cause.

(B) DELITERIOUS FACTORS NOT SHOWN TO BE CAUSAL WHICH MAY NEVERTHELESS TRIGGER ONSET OR RELAPSE OF ME: The commonest triggers of relapse still remain mental or physical over exertion/stress and secondary viral, bacterial or other infection. OTHER RISK FACTORS INCLUDE: (1) IMMUNOSUPRESSION: partial or temporary following immunisation, pregnancy, the use of steroid or cytotoxic drugs, cigarette smoking (which alters mucosal

immunity) (2) HORMONAL IMBALANCE: Following puberty and childbirth the use of certain contraceptive preparations and hormone supplements (eg thyroid) without supervision. (3) PHYSICAL STRESS: Following surgery, head injury, climatic change, malnutrition. (4) EXPOSURE TO : psychoactive and vasoactive drugs or substances (including alcohol) and environmental toxins, especially those which are neurotoxic, cholinergic or muscarinic eg. Organophosphates and similar agents.

CONCLUSION

(1) ME is a physical illness distinguished by its encephalitic features, its unique neuroendocrine profile and close epidemiological similarity to poliomyelitis. It has the potential to cause serious long term disability in the most economically and socially active members of society.

(2) Research into the cause, diagnosis, prevention and management of the illness is seriously under funded and the late chronic phase is almost devoid of attention.

(3) In the absence of an early government sponsored epidemiological survey (including patients in the community who do not present to medical clinics) it will be impossible to plan or cost suitable management strategies.

(4) Redirection of research into institutions, occupations or communities shown to be at high risk of developing ME (eg school populations, teaching and health care) indicates that this is a simple and economical method of identifying the infective agents capable of triggering the onset or relapse of ME and of quantifying long term problems. Rapid diagnostic tests, antiviral drugs and vaccines in the process of development (in response to the rising tide of viral meningitis and myocarditis, worldwide) could well be applied to ME, possibly providing a means of diagnosis, prevention and management in the short term.

(5) The many individual studies recorded in this paper, which have been conscientiously and economically collected, undoubtedly broaden our knowledge of the problems to be tackled. The current implications of these findings in respect of future management of the illness are:-

a. They should be pursued in relation to possible reparative treatment which may involve immunological and other methods, although according to our present state of knowledge, in neurological conditions, healing and repair are notoriously imperfect.

b. That they should engender a more sympathetic attitude to the ME patient.

DATA COMPILED BY E.G. DOWSETT AND J. RICHARDSON

With special thanks to CHRIS RICHARDS and RAY GIBBONS of the CHROME Database.

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Ray Jenkins BSc and Chris Richards PhD inaugurated a database of patients severely disabled by chronic ME in 1995, in order to monitor progress over the next 10 years. They have already disclosed the exceptional severity of the disease in those who develop ME below the age of 16 years and currently have over 300 patients registered. Address: CHROME, 3 Britannia Road, London SW6 2HJ

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FIGURES:-

1. DOWSETT/RAMSAY DATABASE (11.)

2. RICHARDSON DATABASE (12.)

3. CHROME DATABASE (13.)

4. HYDE, CAMERON, DUNCKER (21.)

EPIDEMIOLOGY OF ME

5. DOWSETT/RAMSAY DATABASE (11.)

6. CHROME DATABASE (13.)

ME/CFS IN THE UK SCHOOL POPULATION

1. THE PROBLEM

Do children and adolescents suffer from ME/CFS? Simple common sense tells most parents, teachers and doctors that they do and often more severely than adults. Yet, there remains a sizeable proportion of professionals in Health Care, Educational and Social Services who are still prepared to ascribe the numerous, disabling but seemingly unconnected symptoms of this illness in young people to anorexia, depression, school phobia or a dysfunctional family background. All are, at least, agreed that the illness presents a considerable economic, educational and social problem.

2. MARKET RESEARCH

No hopeful salesman can, nowadays, expect to succeed without prior market research and no-one intending to raise a bank loan for such a purpose will be considered without a well researched business plan. Unfortunately, when we turn our attention to human disease, conclusions are often reached without prior study of the WHO, WHERE and WHEN which can lead to effective consideration of WHY. In medical terms, this is called EPIDEMIOLOGY, which means the study of human conditions in relation to their environment rather than, for example, the behaviour of small animals or tissue cultures in laboratory setting.

3. HISTORICAL BACKGROUND [1.]

Over 60 years ago, epidemiological studies of ME/CFS were commonplace and usually initiated by doctors who, lacking modern technology, simply used their eyes and ears. Of over 70 recorded epidemics of ME/CFS since that time, 13 clearly mention young people while one is entirely devoted to the age group. These old surveys were small and without government funding but they clearly deliniated present day findings such as female predominance of cases, peak incidence at puberty, variable prevalence linked closely to seasonal and geographical features (summer/autumn onset, cool/temperate climates and rural or suburban settings) as well as the key role of schools and similar institutions in the spread of the illness, with clustering of cases within families and schools. The serious disabling potential and chronicity of ME/CFS leading to relapse, is always noted.

4. WHY DID WE EMBARK ON OUR SCHOOL SURVEY?

To our knowledge, no government anywhere in the world has yet funded an epidemiological survey of this type and scale in schools. 10 years ago, a group of sufferers from ME/CFS who were also teaching professionals, parents or carers of young people similarly affected, conducted a postal survey of sufferers under the age of 25 years which stimulated a response from nearly 600 young people in the UK and abroad. The results were disheartening but they supplied the initial impetus to seek further information about the WHO, WHAT, WHERE and WHEN with an unexpected bonus

in relation to WHY.

WHAT DID THIS SURVEY DISCLOSE?

There were 3 major findings:-

a) Only 29% of respondents under the age of 25 were in full time education, training or work, while 34% were totally excluded from all three. The remaining 37% were making various compromises, few of which could be considered satisfactory.

b) An enormous loss of independence and self esteem was reported, with additional grief at the general disbelief expressed not only by the press and by various professionals but at the loss of support even from friends, colleagues, family members, and those in a previously loving relationship.

c) The bonus was a personal invitation to study, at close hand, the consequences (after 2 years) of a seemingly trivial respiratory/gastro-intestinal infection causing symptoms in 1/3 of a village school roll, leaving some 10% of children so affected with chronic relapsing energy problems, musculo-skeletal pain and disabling prolonged cognitive disturbance. Early laboratory investigation was missed but relapses in the following year were significantly associated with the viruses then circulating in the school (Influenza A and parvovirus infection)

5. HOW DID WE SET UP THE 1991-95 SCHOOL SURVEY? [2]

With a clear indication from the young people themselves of what to look for, we set out to find if such a cluster of ME/CFS cases in a single school was a unique event or if (as we already suspected) it would be duplicated elsewhere. We hoped that, if successful, the study might provide clear indication of a cause as well as some helpful pointers to diagnosis, management, treatment and prevention. Since we were not sanguine about the funding of our “business plan” by medical or charitable agencies, we were grateful for the prompt and unreserved backing of the educational profession to whose understanding of the importance of this subject, all students and young sufferers from this illness must pay tribute.

6. WHAT METHODS WERE USED?

A search was made in six English Local Education Authority (LEA) areas, chosen initially for their widely varied geographical, economic and social mix, for all causes of medically certified long term sickness absence in pupils and staff. Confidentiality was assured in the collection of these details which were handled only by senior educational staff and specifically excluded all personal identification. The information requested included geographical location, age, gender and school class location of sufferers, size of pupil and staff roll and education or management provision while sick as well as the outcome.

7. WHAT WERE THE MOST IMPORTANT FINDINGS?

Between 1991 and 1995 we were able to collect details of all types of long term medically certified sickness absence from schools with this problem as well as useful information from those without and from 63 private schools outside the LEA jurisdiction. Excluding the private sector, whose details did not differ significantly from the public one, our survey comprised 1,098 schools 333,024 pupils and 27,327 staff - the largest epidemiological survey of this type made to date.

Significant findings included:-

a) Prevalence of ME/CFS in Schools

Over one third of the schools providing information reported long term sickness absence and of these, 2/3 had cases of ME/CFS (230 pupils and 142 staff) suggesting a prevalence in this population of 70/100,000 in pupils and 500/100,000 in staff - a rate some two or three times that quoted in other adult population surveys.

b) Types of Illness Which Cause Long Term Sickness Absence

Among the 885 individual sickness records received in 6 LEAs, ME/CFS was by far the commonest cause (41% overall, 33% in staff and 51% in pupils), followed by cancer and leukaemia (23%) general medical or surgical conditions (13%) musculo-skeletal problems 12%, psychiatric disturbance and virus infections (5% each).

c) Clustering of Cases

Using a definition of 3 or more cases with the same diagnosis in the same school, we looked at all illness falling within this category and found 54 clusters (36 due to ME/CFS, 7 to virus infections, 4 to psychiatric disturbance, 3 each to cancer/leukaemia and musculo skeletal conditions and one to diabetes). 45 clusters, including all but one of virus infection, occurred in schools with ME/CFS, the exception being in close geographical proximity. 4 small clusters (less than 6 cases) of cancer, musculo-skeletal conditions and psychiatric disturbance were noted in schools with no evidence of ME/CFS. This was a noticeable difference from the large clusters of viral infection (variously described as respiratory/gastro intestinal, “flu” or “glandular fever” numbering up to 16 cases) associated with ME/CFS. Of the 372 ME/CFS cases in pupils and staff 149 cases (40%) were distributed as single cases 78 (21%) as pairs and 145 (39%) as clusters of 3-9 cases, a remarkably high prevalence of coincident ME/CFS and viral infection in selected geographical areas.

d) Geographical Prevalence of ME/CFS

Though single instances were noted in all LEA areas but one, we were surprised to find the majority of ME/CFS case clusters associated with virus infection grouped in a LEA district which was by no means the largest but characterised by its surburban growth after much recent population influx from the building and expansion of “New Towns” in green-field sites. It has always been a characteristic feature of certain epidemic infections (eg. poliomyelitis) and of illnesses now suspected to be triggered by environmental factors (eg. childhood leukaemia) that, when isolated rural or suburban communities which have an established and naturally acquired “Herd immunity” are subjected to an influx of new population, the prevalence of such illnesses increases.

d) The Effect of Age and Gender on the Prevalence of ME/CFS in Schools

Of the 230 pupils certified as suffering from this illness, 157 were female and 73 male, a F/M ratio of 2:1. The average age was 13 with peak prevalence at 15 years. Sex ratios below puberty were more even, indicating a hormonal influence upon the known frequency and chronicity of the illness in females during child-bearing years. The majority of these sufferers were located in senior schools.

Of the 142 staff with ME/CFS, 111 were female and 31 male, a F/M ratio of 4:1, influenced by the fact that most staff employed in schools are female. This is especially noted in the primary and junior sectors where female staff are more common and there is close personal contact with children suffering from various infections and where the majority of staff suffering from ME/CFS are located.

e) Education and Work Modification for ME/CFS

Although varying education and work management patterns were used in sequence or in combination in all LEAs surveyed, we consider the following to be key factors in reducing physical over exertion and mental stress which, together with secondary infection, are the comonest causes of relapse in subjects with ME/CFS in the school environment:-

(i) Pupils:

Home Tuition for those too ill to attend school (but not generally suitable for the very sick who make a better recovery if education is postponed until stabilisation of the illness).

Modified Time Table which permits, for example, continued participation in selective school activities (excluding sport) and the taking of examinations sequentially over longer periods.

School Withdrawal which, in the absence of such concessions may oblige education "otherwise" at home. There is however, no evidence that young people educated this way fare worse than in conventional school classes, while many sufferers achieve better results in the absence of school stress and repeated exposure to infection.

In our survey, provision over the 6 LEAs for HOME TUITION averaged 48% (range 14-79%), MODIFIED TIME TABLE 57% (range 50-86%) and SCHOOL WITH DRAWAL 6% (range 0-17%). Though some other medical conditions enjoyed more generous provision, the majority of pupils with ME/CFS were not removed from the school roll.

(ii) Staff

The average provision for OPTIONAL PART TIME was 50% (range 38-80%) but EARLY RETIREMENT was taken up by 38% (range 27-100%) representing a serious and probably avoidable loss of career potential compared with other illnesses.

SUMMARY

1. Of all the symptoms associated with ME/CFS, disturbance of cognitive function is the most disabling and long lasting in both pupils and staff [3]. It induces prolonged difficulties in maintaining wakefulness and attention, in concentration and memory, in language and mathematical ability and in appreciation of shape and judgment of distance which, combined with motor dysfunction affecting balance and fine motor control interferes with practical tasks and independence. Funding for educational research [4] into the correct management and educational needs of those affected (such as that already received by individuals with other movement, speech or cognitive disorders) would be well repaid by preventing the repaid loss of educational potential in pupils at an age when brain development is at its peak.

2. Our survey into clusteirng of ME/CFS cases in schools and the wide variations in geographical prevalence disclosed, suggests that it would not be difficult to identify specific infections which can trigger the onset or relapse of ME/CFS. Epidemiological research directed to the school population, where good records are kept and prolonged follow up of physical and cognitive problems is possible, would undoubtedly be as economical in terms of diagnosis, management and prevention as it was of the understanding of the true prevalence and mode of transmission of poliomyelitis in the past.

3. The recent issue of CR54 [5] and the guidelines relating to management of young people with ME/CFS (eg lack of provision for Home Tuition, encouragement of early return to school, intervention with anti-depressant therapy and graded exercise) may well leave us with a generation of young people suffering from educational deficit and an inability to assist themselves back into work, to meet government targets.

PS. Since the publication of our work, a community paediatrician, unaware of our work and unknown to us, has, in subsequent years, completed a similar epidemiological survey in just two boroughs of one LEA mentioned in our study, with surprisingly similar results. This work is prepared for press but has not yet been published in the UK.

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Town Hall

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http://www.cfs-news.org/news.htm is recommended reading always

1998 Sydney Conference

Official Site:

- http://www.ahmf.org/Miscell.htm
- http://www.ahmf.org/conf98.htm
- http://www.ahmf.org/Conf98_2.htm

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Reports on the February 1999 Conference

- http://www.cfs.inform.dk/Nyheder.udland/cfsnews16aug.txt
- http://www.cfs.inform.dk/Nyheder.udland/cfsnews17aug.txt
- http://www.cfs.inform.dk/Nyheder.udland/cfsnews18aug.txt
- http://www.cfs.inform.dk/Nyheder.udland/cfsnews19aug.txt
- http://www.cfs.inform.dk/Nyheder.udland/cfsnews20aug.txt

Official Site:

- http://www.ahmf.org/conf99.htm
- http://www.ahmf.org/Conf99_2.htm

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

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Updated on: 03/27/2000

Nicholas Regush

Virus Within : The Coming Epidemic

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- http://shop.barnesandnoble.com/booksearch/isbnInquiry.asp?userid=4MZVC4VJVH&mscssid=AVUE5WSPXWSH2JKJ001PQUW8MEGFAPS7&srefer=&isbn=0525945342

Bookcover

March 19th, 2000, 7pm PST, Attendance: 430+
Support:*Welcome Nicholas Regush, there are many here who have read your book and have many questions.*

[pat fero]:Brain lesions. If a pt has viral CFS symptoms and lesions, what can the pt do Would you comment further about the need for collaborative studies on MRI and HHV6?
Nicholas Regush:*The possibility of studying hhv-6 and MRI is something that must be done - but lack of funding prevents the proper type of collaboration.*

[Early Birds]:Did you research Sidney Grossberg's work(at the University of Wisconsin?)? If so, what do you think? I have just finished his book--I liked it a lot--and it covers most of the HHV-6 research that many of us already knew quite a bit about. Your reporting on Knox and Carrigan was particularly thorough and interesting. From rhbailey@catskill.net (CFSFMExperimental)
Nicholas Regush:*I'm not impressed with S. Grossberg's claims for a virus. The work needs to be published and if there is an actual virus, then it must stand up to proper scrutiny. I think this entire field of research (CFS research) needs a major credibility boost - and that is why I focused on Carrigan and Knox. Too much money from CFS patients has gone to research that has lacked appropriate scientific controls and conceptual development.*

[Mike C]:Do you have any standard treatments for people with CFS?
Nicholas Regush:*There are unfortunately no standard treatments for CFS. Mostly, there are interesting leads, including the use of certain anti-herpes virus drugs and immune boosters but for the most part physicians must make careful and informed decisions about what to try on patients - with full informed consent for those patients. This again is why I think the HHV-6 research is promising because the next phase will probably bring some important clinical trials into play.*

[April]:Orthostatic intolerance You noted that Carrigan's work with HHV6 showed that the virus interfered with the bone marrow's ability to produce new blood cells. Do you know if the researchers have considered HHV6 as a cause of low blood volume and orthostatic intolerances in PWCs? What are your own thoughts on the possibility?
Nicholas Regush:*Carrigan and Knox certainly show that HHV-6 can affect propduction of new blood cells but their work has centered on primarily on identifying factors in the breakdown of production and not their relationship to clinical problems; so I'll also have to pass on this one because there is no data available that satisfies me.*

[April]:HHV6-A vs. HHV6-B Please explain the difference between HHV6-A and HHV6-B. Are both strains common in the general population? I've read that AIDS and CFS patients have the A strain in common, and that CFS could be considered non-HIV AIDS. Comments?
Nicholas Regush:*Variant A and B are suffiently different from one another to almost be seen as different viruses - not quite, but you should get the picture of enough variance to probably make a difference, perhaps even in the way that they are transmitted. We know that B is extremely common because the serology has been done; A is up for grabs in terms of just how many people contract it; there is confusion in the literature about who gets it. Regarding the AIDS question, I personally look at illnesses as interlinked. Some have common features as does CFS Sand what is commonly termed as AIDS, with obvious differences in terms of extreme illness. But I have long argued that if we are ever to understand CFS better, we must get rid of the idea that this construct - i.e. CFS - is something unto itself. The body is not static. It is a dynamic entity, interacting internally to the outside world - the dynamics are poorly understood, and that includes how the immune system is affected. As time goes on, we shall probably see more common pathways being exposed in a number of diseases, with viruses like V-6, (one or both variants) playing a trigger-like role in setting up a series of bodily events. Chronic illness will be more complex than many researchers like to suggest it is. We try too hard to find single evil events and sin*

[April]:Blood donations There's a growing movement among PWCs to give their intent to give blood (see www.geocities.com/pledge_now/) as a way of protesting the NIH/CDC not taking the infectious threat to the public seriously. Many have even claimed that during recovery periods they have donated blood with the full knowledge/approval of the Red Cross. Do you think blood donation by persons with active HHV6 infection is more dangerous to the blood supply than healthy people with a latent infection? With the virus latent in most everyone, would it even be practical to screen for it in the blood supply?
Nicholas Regush:*In short, yes it would be practical to screen for HHV-6 in the blood supply.*

[Marisa, San Francisco]:recent blood work shows transit from bac infection to viral infection (I have activehhv-6/ c.pneumoniae/parvo b19/EBV My immunolgist was obviously thrown by this, and could only hazard a guess that two of the viruses I have been diagnosed with have "joined up" (his owrds) I wonder: do KNox and Corrigan have any thoughts on HHV-6 in conjuction with other active infections such as these... I have received your book but have been too sick to finish more than a few early pages, my apologies if this question is answered int eh book....
Nicholas Regush:*Knox and Carrigan believe on the basis of their unfolding CFS research that HHV-6 and EBV may work together, and that active V-6 reawakens EBV. Also, there is no reason to believe that other viruses may also become involved at some point in the multi-phase process that leads to chronic illness. WE are only at the very beginning of our understanding of how this all might work. WE also need to learn more about how viral triggers can affect our host genetics. Research in this vein is beginning to show some idea that our genes can even get reshuffled as a result of viral hits.*

[Marisa, San Francisco]:Your thoughts on what the cfs/gws/etc patient pop can do to change the ignorant course at NIH/CDC The rectn years have shown the course to be of littlehope for the patient population and I fear that due to enormous discord within the population that internal fractures lead the NIH CDC tothink we cannot accomplish much, do you have thoughts on what course of action the patient population might take to raise conciousness there and with Congress/Media?
Nicholas Regush:*Unfortunately, discord in any "illness" grouping becomes common, particularly as research and promises fail. People must come to understand who the real enemy is - and it's not people who are ill. The enemy as I see it is bureaucratic stupidity on a massive level; one way to fight it is through strong coordination of efforts. Congressional committees are beginning to show some real interest in coming to terms with issues about chronic illnesses. CFS could be very much in the spotlight. I am doing my best to try to make this happen.*

[Fluffy]:Do you really believe the government is seriously interested in finding treatments for these diseases AIDS,GWS,ME/CFIDS,FMS,MS,LYME,MCS,PPS,Agent Orange If you think so, please state evidence
Nicholas Regush:*It doesn't work that way. Government officials listen to scientists who traditionalloy have maintained old boys networks of funding and advice-giving. These networks become entrenched and try to protect themselves at all costs, often referring to those who question orthodoxy as flat earthers or whatever. I for instance find it appaling that Straus is still emmersed in this idiotic psychobabble theory of cfs. This has been maintained, in part, because so much bad research has flooded CFS. Sorry to say that, but I'm convinced a lot of people that groups such as yours may have funded dhave been led down the dogpath by ineffectual researchers who promised much too much. They then become fodder for networks functioning at the government level. This is why I wrote a book focused on Carrigan and Knox. They do terrific science. It is well-published and involves the appropriate methodology. Too many CFS researchers are using unsuitable research probes, including PCR and serology when they should be trying to isolate virus. In the case of CFS, cultures show evidence of virus before antibodies even appear to HHV-6 - if they in fact ever appear. To continue to do research with antibody testing and pcr solely is bordering on defrauding cfs patients. This is just dumb science.*

[NancyM]:McMinnville commentary- nejm hysteria Off subject, but you wrote a commentary on the bogus nejm article of 1-13-2000, and i wrote you an email never hearing back, where i actually had proof that their article was inaccurate to say the least. I now have the epa documents, showing very poor building maintenance, poor air quality, a host of problems. Do you want me to send them to you at abc, if so, let me know where to send them.... This is relevant to cfids, due to the editorial accompanying the nejm "research" by simon wessely who has disparaged cfids as hysteria in the past... I live in Tennessee, so that explains how i have the data in part. Nancy McFadden
Nicholas Regush:*Anyone who wants to send me materials at ABC News should sent them to me at 47 west 66th street, NY, Ny 10023. Sorry not to answer your e-mail. I get up to three thousand every week in regard to my column. I just can't keep up.*

[Sophie]:HHV-6 - is it best to try to treat active HHV-6 infection? If so, what treatment do you recommend? If HHV-6 is just an opportunistic infection, perhaps it will just reoccur after treatment. Perhaps it is not that "causitive" in CFS. Maybe you DO consider it a causitive factor in CFS. If you feel treatment is useful for long term, what is the best treatment - gancyclovir, etc. (most of these are high-powered anti-virals with some significant side effects)
Nicholas Regush:*Sorry, not sure what just happened but I got kicked off in mid-stream. I was answering Sophie. V-6 may not just be opportunistic. Carrigan and Knox show that it can cause progressive disease - and quickly. There is a lot we don't know about Variant A and how it is transmitted and how it does its dirty work. For a virus to be considered "causative" requires a lot of proof. We need more. But the preliminary evidence is a red flag. It is active in the body when CFS patients have symptoms. There is more viremia when patients, for example, complain of neurological effects. Remember, this virus has a thing for nerve cells as well as immune cells. Gancyclovir used intravenously seems to help some patients with MS. Oral gancyclovir combined with an interferon or immune booster might help some; b ut I want to emphasize that we are far from understanding what is going to work because*

[pat fero]:Infant death due to HHV6 transmitted at birth. Health status of mothers? Transmissability through birth, breast milk, blood is an issue I am concerned about.
Nicholas Regush:*There is little or no data on the health status of mothers whose children have a hard time with HHV-6 in early life. In some cases, the virus has run through children very powerfully early in life, but we don't understand why one child and not another.*

[pat fero]:HHV6 kills natural killer cells. Why no collaboration on HHv6 and NK research?
Nicholas Regush:*Good question. When a virus has the potential to destroy so many components of the immune system, clearly the scientific community has its head in the sand.*

[Fluffy]:Aren't you sanitizing ("differences of opinion" and "bias") what is actually an assault against the chronically ill If I kick you in the balls once, I could just say I made a mistake and you may believe me. If I kick you in the balls twice, I can say I made a mistake. You may suspect otherwise. If I kick you in the balls a third time, I can say I made a mistake. You can say I intentionally did it. We now have a "difference of opinion" and some "bias" is'involved where I place my foot. The chronically ill have been

having their balls kicked in by the federal government for decades. How many more kicks do we have to take before it dawns on you that there is an actual violent assault taking place You call the problem "differences of opinion" and "bias". I call that view extremely naïve at best.
Nicholas Regush:*Well, don't get pissed off with me. I'm not the enemy. If you read my second opinion column at ABC, you can get the full flavor of what I believe and how I put my career on th*

[pat fero]:Explain the signficance of red cells, bone marrow and HHV6... Could HHV6 infection explain red cell wall abnormalities seen in ME/CFS?
Nicholas Regush:*HHV-6, according to research by Carrigan and Knox has the capacity to disturb the bone marrow's normal production of blood cells. Re: red cell wall abnormalities. Could be. The research needs to be focused on this.*

[pat fero]:Please make suggestions for further research and discuss funding of such efforts. Scientific bias against innovative research is clearly demonstrated by the NIH and CDC meaning that"safe" research is being funded. With this approach, you can kiss goodby the ideas in THE VIRUS WIHIN. What can pts do?
Nicholas Regush:*Well, some people are not going to like my answer to your question. I think that each CFS group around the country has to come to terms with the unfortunate fact that you have been supporting too many researchers that are not delivering. Too many experts out there that are taking you for a ride. I mentioned earlier that this PREVENTS recognition of real scientific work in CFS. I read the overall literature and frankly I get appalled at the slovenly science. This is a major problem that you will have to deal with before much else gets done. Some people in CFS groups obviously can read the science and should help out to point out that some people - particularly Carrigan and Knox - are getting it right because they don't take stupid short-cuts and can defend their science when under attack. Yes, the ideas in my book will be kissed off if the right people don't get the funding. I am going to do a major investigative report in the near future on the state of the science - separating the bs from the good stuff. Too bad for those who have been given an easy ride. I think the CFS community has to get more serious about where it puts its money.*

[pat fero]:Is there information in the book you have since found out to be questionable? One is never "finished." To do over again, would you add or change information? What?
Nicholas Regush:*No, there's nothing in the book that I would necessarily change, but I will continue to track this story and continue to update my writing on it. I choose issues when I feel I can have an impact. I plan to have an impact.*

[Ellen]:What do you think of possibility HHV6 is involved in cases of spinal stenosis and Chiari formation? Is it possible that CFS or what causes it also causes brain swelling in some and that the surgery merely relieves the symptom?
Nicholas Regush:*I'm leery of the surgery being proposed - but that's because I have not seen any data from that surgery center. It bothers me that they haven't published.*

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Transcript

- Speaker: David E Berg
- Latest work/publication: SUGGESTED TREATMENT PROTOCOLS FOR CFS / FM PATIENTS (http://www.hemex.com/cfs/cfs_tx-2.html)
- Website: http://www.hemex.com/cfs/
- March 26th, 2000 12PM (Noon) PST, 3PM EST

Held at the CFIDS Town Hall (http://www.folkarts.com/townhall/)

support:*Welcome David, you have an international audience today - including people from Norway and France. We are all looking forwards to you views and opinions. Thank you for participating!*

Greetings to all. It's an honor and pleasure to be on line today and especially to follow Mr. Regush. His book is excellent and RIGHT ON !. (It's funny not being able to see faces that are included in the dialog.) But such is the NET. For some time now, I have been frustrated knowing that the coagulation part is only half of the problem and that one or more pathogens are the other. And that HHV6 has had no know treatment until now. So let's begin where Mr. Regush's book ends in October, 1999.

In November, 1999, at the Infectious Disease Annual Meeting in Philadelphia, I saw a poster on HHV6 and spoke with the author, Dr. Joe Brewer of Kansas City. Over a four hour plus dinner meeting, we worked out the model that is being presented now about a basic coagulation or fibrinolysis regulatory protein defect in CFIDS patients as the genetic culprit. Then you add in a pathogen (HHV6, CMV, Mycoplasma, Chlamydia pneumonia, etc, or a combination of several of these pathogens) and the patient goes down hill rapidly into chronic illness due to the pathogen activating the coagulation mechanism. This is due to an immune response as well as inflammatory responses to the pathogen and probably the pathogen itself activating the coagulation system. Anticoagulants (primarily

heparin) shut down the Soluble Fibrin generation and fibrin deposition on the Endothelial Cell (EC) surfaces. But unless the patient can get treatment for the pathogen, the healing response can only reach 50% or so.

My frustration has been HHV6. Dr. Brewer told me about a new colostrum derived, highly purified Transfer Factor (TF) that would contain only specific IgG and IgM antibodies against CMV and HHV6 (see www.immunitytoday.com). He started testing many of his patients for their coag defects and we found such in every patient. Each patient also had documented HHV6 infection. Beginning in December, Dr Brewer began treating his patients with this new TF. Patient stories are dramatic. We will discuss some of them.

In early December, 1999, at the American Society of Hematology, we met Dr. Konnie Knox. After spending two plus hours discussing theories and therapies, we were all singing the same hymn. So the circle from last week to now is complete. The Good Lord has put Lois & I here at the right time, in the right place with the right knowledge and the right people to be able to solve these “Blood Curdling Mysteries” of chronic illnesses, and they extend beyond just CFIDS patients.

WHY is it important to be tested for the coagulation defects? It is VERY important, because at some point in time, all CFIDS patients will need surgery, be in an accident or traumatic situation and NEED to have PROPHYLAXIS to prevent a blood clot, stroke, heart attack or Pulmonary Embolism from happening. If you know your protein defect, then proper anticoagulant therapy will prevent catastrophic events. I feel very strongly about this.

If you look at the population of America and the patient race distribution of CFIDS patients, there are about 5% of bleeders (hemophiliacs or von Willebrand Factor deficient patients) and about 5% clotters. Using a bell shaped curve, 260 million USA population yields 13 million clotters. 1 million CFS, 8 million Fibromyalgia, ? Million Multiple Sclerosis, ? Recurrent Spontaneous Abortors, etc. Are we close? The protein defects have mostly risen from European decent and are mostly white people. Hundreds of years ago, when someone cut themselves hunting or preparing food, it was advantageous to clot fast (not bleed to death). Life spans were shorter then also, so these coagulation or fibrinolysis regulatory protein defects were beneficial. Today, with much longer life spans, these defects cause chronic illnesses by not controlling the coag response properly. So much for my PhD thesis.

1. **[Fluffy]Are there any non prescription treatments you could recommend and what about future treatments ?**

   Aspirin supposedly attacks one of the factors involved in coagulation.

Bromelaine supposedly attacks all 3 of the factors. There are research indicating that bromelain increases antibiotic absorbtion. I currently take 2500 mcu per day with minocin and it seems to help. I did experience a very slight headache initially and it seems to help with the brain fog. Any comments on this and other possible supplements. Also what is the timeline on future treatments ? Please also include anecdotal and personal opinions in your comments.

The ISAC Panel contains a test called the Platelet Activation Index. What we have learned is that this is really showing us whether or not there is an infection in the patient. If the CD62P alone is elevated, then this indicates an UNDERLYING INFECTION. When both are elevated and the PA Index is 1-3+, then this indicates an ACTIVE INFECTION. My guess is that HHV6 has infected the bone marrow (as it can!) and is inside the platelets when they leave the bone marrow for the blood stream. Because the immune system “sees” infected cells, Immunoglobulins (IgG or IgM) attach to the platelets, causing the alpha granules to partially release and CD62P gets transferred from the inside of the platelet to the outer membrane. This accounts for the elevated CD62P value in the assay. The higher the CD62P value, the greater the infection. Aspirin is NOT going to be effective against infected cells, and this is what we have seen in general, that ASA does not make the patient feel much better.

I am in the process of forming my opinion on Bromelain. Elly (in Wash DC) told me about this last fall, but I did not understand her. Several months ago I did a literature review on Bromelain and was amazed at the scientific articles related to Bromelain. Bromelain, from pineapples and totally natural, seems to help FIBRINOLYSIS. There are no studies to actually prove this, but it is STRONGLY suggested in literature that it does activate fibrinolysis. Since no docs or researchers will touch using tPA or Urokinase (drugs that activate fibrinolysis in vivo) in CFIDS patients, Bromelain seems to be just the ticket. And it is all natural. Many anecdotal responses that I have received, confirm that it helps in patients that have elevated inhibitors of fibrinolysis - Lp(a) or PAI-1 - as their underlying genetic defect. So, Bromelain helps increase fibrinolysis. As for it inhibiting platelets or the coagulation cascade, nothing in literature suggests that it does such. I may have overlooked something, so if anyone has a reference to the contrary, please send it to me. Thanks.

As for minocin, I have no knowledge of its properties or use, except that others report good results using it.

As for Bromelain enhancing antibiotic adsorption, I believe it would work like this. By increasing fibrinolysis, any fibrin on the endothelial cell (EC) surfaces (the cells that line the capillaries or very small blood

vessels in the body) would be mostly removed, and that would make the antibiotics more effective at getting into the infected EC. Since Bromelain is a digestive aid, then more might be absorbed through the GI track as another possibility.

As for a time line for therapeutic agents, we just posted one on our web site Friday. It uses heparin for 6 months, adds Bromelain at the beginning for 4-6 months for patients that have a increased Lp(a) or PAI-1. The time line starts with Transfer Factor after 30 days of heparin for 2-3 months. Also, antibiotics are started after 30 days of heparin. Using heparin for 30 days first (plus bromelain if indicated), gives the body time to shut down the coagulation mechanism during the first 30 days and allow the fibrinolytic system to clean up part of the fibrin deposition on the EC surfaces. This makes the Transfer Factor (TF) and antibiotic use MUCH more effective. The patient continues to use heparin for another 2 months, just in case. If there are still a few pathogens left after these therapies, they will attempt to reactivate the coagulation cascade again, to generate Soluble Fibrin &/or fibrin deposition. So by continuing heparin, this will prevent cascade reactivation and the immune system will be able to clean up the remaining pathogens. From information given to me by patients on this new TF, I think we NOW have a treatment protocol that will get patients ALL the way back to good health. This was a long winded answer, but the question was a good one to answer and gives much of the information about these processes.

2. **[Bob R.]Time Frame of Treatment**

David, I have been on Lovenox 30 mg for almost 4 months. I received an dramatic improvement in IBS symptoms, brain fog improved, fatigue improved somewhat however nothing dramatic. Two weeks ago I switched over to standard heparin and have started to feel a little better. In short , if possible at this point, have you had any experience with patients recovering very slowly for lets say a year time period. Or do you notice immediate improvement with your patients over a very short time frame?

I BELIEVE that most (>80%, if not ALL) CFIDS patients have an underlying infective pathogen (HHV6, CMV, Mycoplasma, Chlamydia pneumonia, etc, or a combination of several of these pathogens). Anticoagulants stops the coagulation component but does nothing against the underlying pathogen. Thus the need for antibiotics, antivirals, Transfer Factor, etc. You need BOTH heparin and some treatment against the pathogens. That’s why patients on heparin ONLY get about 50-70% well and not 100%.< /P>

3. **[Fluffy]Could food sensitivity of ME/CFIDS people be related to coagulated blood ?**

   ME/CFIDS are prone to food sensitivity. Calcium is suppose to promote blood coagulation so foods like milk, cheese may seem like they could promote blood coagulation and have negative consequences for people with coagulation problems. Are there any foods which seem to promote blood coagulation ? Please also include anecdotal and personal opinions.

   Most of the peripheral problems of the CFID patients (HPA axis, headaches, brain fog, IBS, and allergies) are caused by poor blood flow due to thick blood (hyper viscous blood). When heparin is used to "thin out the blood", this decreases the high blood viscosity by shutting down Soluble Fibrin Monomer generation. When viscosity returns to normal, these peripheral problems lessen or go away completely. We have seen these allergy problems (complete with increased eosinophils on blood smears) from our early days of infertility testing 7 years ago. The allergies decreased significantly in these patients as they use heparin throughout their successful pregnancies. (To date, we have already had over 400 successful first time deliveries of normal healthy children in previously infertile women.)

4. **[Kru Heller]**What other non prescription treatments can be used for treating thick blood.

   Aspirin and Bromelain were mentioned above. I have also heard of the use of Vit. E, Garlic, Pycnogenol and Ginko. What amounts should be used? and how often?

   Remember the ACE of Hearts! Use Beta Carotine (15mg or 25000 IU) at NIGHT time,1gm Vit C am & pm, and 400IU Vit E pm (A,C,E for a healthy heart) . 60 mg Ginko am & pm, and Glucosamine (500mg)/Chondroitin (400mg) am & pm and 81mg ASA at night. The Ginko & Glucosamine/Chondroitin have very mild anticoagulant effects as well as aspirin as an antiplatelet. Since these are VERY mild in their anticoagulant effect, it would take many months to notice any improvement in CFIDS as an anticoagulant using these. That is why I strongly recommend the heparin protocol for immediate therapy. The B-Carotine increases tPA release from ECs over a 12 hour period, so take at night when PAI-1 goes up routinely goes up at night. Everyone has an opinion on supplements. All I can say is to find the right combination for you.

5. **[Sean L]Different blood thinners**

   Dear Mr. Berg, When you use heparin to treat CFIDS, do you think it is

purely its blood thinning properties that help, or are it's other properties (such as it's antiviral properties) part of the picture. I ask because when I talk to people who have tried different blood thinners they seem get quite different reactions to each. Heparin seems to get the best response, Coumadin the weakest and Lovenox somewhere in between. Thank you for all your hard work in this area. Best regards, Sean (Lovenox 30mg/day for 3 months, slight +'ve response, soon to switch to heparin to see if there is a difference in response).

Coumadin is only an anticoagulant. It works by decreasing Factors II, VII, IX & X and Protein C and Protein S. The negative about coumadin is that any green foods that contain Vit K counteracts the coumadin effect, so you have to be very careful about diet, even if you are on low dose coumadin(<2.5mg/day). Heparin is an anticoagulant (anti Factor X and II), anti-inflammatory, antiplatelet, vasodilator, increases NO production and other beneficial side effects. It is normally occurring on the surface of ECs as heparans or heparan sulfate. It is a large molecule and the heparin solutions contain many different sizes, from low molecular weights of 2000-10,000 to high molecular weights up to 25,000. There are two sources for heparin: bovine and porcine. Porcine is less allergenic and the recommended type. Low molecular weight heparins (LMWH), such as Lovenox, is made up of heparins form 2000-9000 size (frequently around 4-6000 size). I like the regular heparin because it is inexpensive compared to Lovenox and seems to work the best.

There is hope for 2001 to get rid of the needle when an oral heparin from Emisphere Technologies will be available. I've asked about compassionate use for 2000, but Emisphere will not release any until the current Phase III trials are finished and the product is approved by the FDA. Our work on this technology over the last 2 years indicates that the product really does work!!!

Anticoagulants still do not address the problem of THE UNDERLYING PATHOGENS (HHV6) !!!.

6. **[Sean L]Plavix**

Recently Prof. Al Cocchetto told me that some GWS sufferers where doing well on a potent new platelet activation inhibtor called Plavix. Do you have any opinions on the use of this drug for CFIDS/FMS/GWS? Best regards, Sean.

YES. Most of the GWI patients have platelet activation from sources other than infection. So these patients react well to Plavix. CFS patients have infected bone marrows, so ASA or Plavix doesn't solve this type of activation. (See Fluffy's question for an extended answer)

7. **[Kuby]Sed Rates**

   How often do you find an Myalgic Encephalopathy patient with a sed rate below 3 who does not have a coagulaton problem and how often does a patient with a sed rate of above 5 encounter coagulation?

   We are writing a new journal article addressing the Normal Range of Sed Rates (ESRs). <5 test values are indicative of a hypercoagulable state. The only time this is not true is a cancer called Multiple Myeloma where there is a lot of extra protein produced by the cancer cells. In either case, because of the Soluble Fibrin or extra proteins, the RBCs cannot settle out of the plasma and thus you have rates of 0-4. The lower the Sed Rate, the more SFM and the more hypercoagulable the patient is !.< /P >

8. **[KenL]Whey - an Alternative to Transfer Factor?**

   Non-denatured wheys, like Immunopro, appear to function in a manner similar to Transfer factor - but is significantly cheaper. Do you have any comments or have you investigated this type of product?

   I do not have knowledge to answer this question at this time. It is an interesting concept.

9. **[DebbieSinKC]new protocol time lines**

   i don't understand the time lines - is it saying transfer factor for only 3 mos.?!?!

   We will change our chart to DAYS on the time line instead of MONTHS to make it clearer. Thanks for the comment.

10. [karen]:If blood work results from a "standard work-up " are normal, can?you still justify ordering the ISAC panel?l.
    I am very interested in getting the Isac panel but my doctors hesitate because they say there is no indication of blood abnormality in standard lab work that would justify persuing this avenue. Could standard work up be normal and ISAC panel still be positive. If so, could you explain this so that I could refer my doctor to your explanation? Are there patterns in normal blood work that correlate with positive ISAC oanel/ If so, what are they?
11. [karen]:If my doctor ordered a hyperocagulability panel from another lab, would this have to be

duplicated at Hemex to get the
My physcian was somewhat interested in the earlier information I brought to him on your work and wrote out a script for a hypercoagulability profile but did not specify Hemex or Isac panel. I did not get the test done because I suspect I need the specific Hemex tests but I have not yet discussed this with him. Can you comment on these issues in a way that will help me communicate with and educate my doctor to be sure Im getting a good evaluation regarding usefulness and specificity of tests? Im sick and ndot much of a biologist so this would be very helpful to me and probably to others.

Good questions and ones that I have not answered before. "Standard coagulation workup" would NOT show any abnormalities unless the aPTT was BELOW the normal range, which indicates a hypercoag condition, but docs are not taught this information. The ISAC panel is like 10 - 20 times as sensitive as the standard screening tests. Most laboratories report a normal range for Fibrinogen of 200-400 or higher. The real range should be 200-300. Ours goes up to 315mg/dl. Most labs don't want to deal with minor elevations in results, so they increase the acceptable range a little. That is why patients with activated coag systems have minor fibrinogen elevations which are very significant to us but not to the physicians who routinely see higher normal ranges. The Prothrombin Fragment 1+2 test indicates that thrombin has been generated when this test is increased. This excess thrombin should be removed by AntiThrombin, which will give increased T/AT Complexes. There are probably 12 labs around the country that can do these two tests, so they would not be included in the standard screening. The Soluble Fibrin Monomer (SFM) test indicates that the thrombin has converted fibrinogen to SFM when this test goes up. SFM is the culprit for FIBRIN DEPOSITION and increasing BLOOD VISCOSITY. There are probably only 5 labs around the country that can do this assay. As for the Platelet Activation test, this is our proprietary assay. We have learned so much from using this assay. If time permits later this year, we will submit our findings and methodology to a peer reviewed lab journal for publication.

As to the hypercoag panel or Hereditary Thrombosis Risk Panel (HTRP), there are several labs that offer the routine tests in these panels. Certainly Antithrombin III (AT), Protein C, Protein S, APC Resistance can be done elsewhere. You should always ask for the "ACTIVITY" assay of these proteins. Do not let the lab substitute the "ANTIGEN" assay as it is not as sensitive as the activity assays. Remember that <50% of the patient defects are in this group (HEMEX 1999 stats = 47% in 300+ patients). Homocysteine is run routinely in

many labs. The other 3 assays are more specialized. Factor II level or the Prothrombin Gene Mutation is rarely performed but positive in about 20-25% of patients. Lp(a) and PAI-1 defects have been found in 53% of our 1999 patient data base. These tests would be performed in maybe 12 labs around the country. So, all in all, send your blood to laboratories that specialize in this type of testing. Our technologists do these assays daily and are very competent in what they do, instead of a tech that might run these assays once a week or month.

12. [Patti]:Started heparin 1 1/2 weeks ago.
So far I haven't noticed any benefit from heparin (except warm toes :). I have had reallyl bad headaches. Could the headaches be related to the heparin? Also - I have really high PAI levels but allergic to bromelain and garlic. Would niacin be an effective way to reduce PAI? Also - do different labs have different norms for fibrinogen levels? I saw a result from a different lab that said a fibrinogen level of 400 was within "normal" range?

See my previous answer on fibrinogens. High PAI or Lp(a) values are the hardest to treat. If you can't use bromelain, then niacin is the next choice. Niacin is hard on the liver. Consult with your physician on this. There is a time release formula that is less toxic and hard on the body. I tried niacin myself, but I couldn't handle the vasodilitation (flushing effect). Give yourself time on the heparin. It takes much longer to see beneficial effects when a patient has a high PAI-1 or Lp(a), sometimes 2-3 months.

13. [Patricia]:Blood Tests
Dr.Berg Thank you for joining us today. Are there any blood tests you would reccommend to our Drs. for us to have in conjunction with Hemex's blood testing?

14. [Patricia]:EBV & or HHV6a,b
Have you noticed patients with high titers or counts with EBV reactivation and or HHV6a or b ?

With the time line that we have proposed, knowing that one is positive for CMV, EBV, or HHV6 may be academic. It may cost less to go through the therapy of TF and antibiotics than getting these viral test performed. I do not know the cost or time to run these tests. Personally, I would want to know the data, so I would get tested. It is an individual choice.

15. [Kru]:I'm interested in sub groups of CFS
Are you noticing anything about sub groups or sub sets of

people that have CFS in relation to when blood thining works and when it doesn't? Or anything else about sub sets for that matter.

The two subgroups that we see are the genetic defects in Thrombin regulation (THROMBOPHILIA) or Fibrinolysis regulation (HYPOFIBRINOLYSIS). HYPOFIB patients are definitely harder to treat, since the process to clean up the vessels is inhibited by high values of Lp(a) or PAI-1. It may take 2-3 months for these patients compared to 2-3 weeks for thrombophilia patients to get to equivalent points in relief.

---

16. [Patti]:Injection questions
I ice my injection sites, but sometimes I get large bruises (2-3 inches in diameter) and other times I get small ones (~1/2 inch). Is there anything to be worried about with the large bruises? The injection sites on my stomach look much worse than the ones on my leg (very red), does this mean anything? How long should I wait until I "revisit" and area for injection? Can the top side of the leg be used for injection? About stomach injections, should you go above the waist AND below? How high above the waist? What can you do to make bruises go away faster?

I don't have any good answer to these questions. Beth, our long term patient, has much experience on this. Contact her at pbdrechsel@msm.com .

SUMMARY:

I trust that the combined information from last week and this week makes sense and is logical. The coag Paradigm Shift is that we should now treat patients with fibrin deposition as we treat patients who have had a blood clot. The new protocol on our web site (http://www.hemex.com/cfs/), should help most CFIDS patients get back to almost complete health for under $3000, including lab testing, TF, antibiotics and physician charges. There will always be the coag protein defect in the patients, but once the infection is treated completely, then the protein defect can be monitored over time. When a relapse occurs, use heparin to control the infection quickly before becoming a CFIDS patients again. Remember, the longer one has been ill, the longer it may take to get rid of the HHV6. T

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

Independent Medical Examination

for

Chronic Fatigue Syndrome

Dear Physician,

Chronic Fatigue Syndrome (CFS) claims results in a large number of court cases. To reduce costs to both insurers and patients due to a lack of familiarity with current research and laws on CFS, the following survey have been developed.

Please take a moment to review and answer the questions below. Please deliver a completed copy to the patient before the examination and include a copy with your report to the insurer. We hope this sheet will help reduce costs for all.

Definition: *Insurer*: includes Social Security Administration or an Insurance Company

At the end of the document is a list of web resources that may interest you.

Physician

Your Name:	_____
Board Certified in following areas:	
Years in practice:	_____ years

Please circle the appropriate answer below:

Your CFS Experience

Approximately how many cases have you diagnosed as Chronic Fatigue Syndrome during your medical career?	____	
Approximately how many of these patients recovered while under your treatment?	____	
Are you current with the latest findings and recommendations from the Center for Disease Control?	Yes	No
Are you aware of the difference in symptoms between *Sudden Onset* CFS and *Gradual Onset* CFS?	Yes	No
Are you aware of the proposed renaming of CFS to RNase-L Enzyme Dysfunction Disease?	Yes	No

Are you aware of the proposed sub-classification of CFS to Immune System Activation of Coagulation syndrome?	Yes	No

Clinical Tests

Will you be testing for low molecular weight RNase-L Enzyme, the current research marker for CFS and proposed clinical marker?	Yes	No
Will you be testing for low red blood cell counts?	Yes	No
Will you be doing the Immune System Activation of Coagulation (ISAC) panel for CFS?	Yes	No
Will there be testing for low blood plasma volume?	Yes	No
Will there be testing for CFSUM1 and CFSUM2?	Yes	No
• If none of the above tests are done, please explain why?		
Will there be any other clinical tests that are not elimination tests?	Yes	No
• If other tests, which tests?		

Purpose of Independent Medical Examination

Were you informed by the insurer...

The purpose of this examination was an evaluation of the patient resuming the specific duties of their former occupation?	Yes	No
The purpose of this examination was an evaluation of the patient resuming their specific duties of their former occupation?	Yes	No
Were you provided a detailed list of those duties and related work requirements?	Yes	No
Will you be discussing them with the patient?	Yes	No
The purpose of this examination was for the patient resuming **ANY** occupation?	Yes	No
Was there a set of specific occupations suggested by the insurer?	Yes	No
Is the Insurer the Social Service Administration	Yes	No
• If not, please identify the Insurer:_____		

Legal Criteria for Disability

Are you familiar with rulings of Federal Courts on the criteria needed for a diagnosis of CFS?	Yes	No
Are you familiar with rulings of Federal Courts on the "availability to work" criteria that a patient must have in order not to be deemed disabled?	Yes	No

Were you provided with guide lines or training by the Insurance company on criteria to apply?	Yes	No

The above is correct to the best of my knowledge,

_____, Date_____, Place_____
Physician Signature

Web References:

The following may be of interest:

CFIDS SYMPOSIUM, Temple Univ., Nov. 1, 1998 ,
http://www.abcjb.com/fm/events/summary_of_a_cfids_symposium.htm

Circulating Blood Volume in Chronic Fatigue Syndrome, 1998,
http://www.cfidsfoundation.org/Library/Bell.htm

Hemex Labs CFS Pages: http://www.hemex.com/cfs/main.html, ISAC Panel

HOMEOSTATIC HETEROGENEITY IN CFS PATIENTS,
SCL-90-R Psychological Inventory Responses in Patients With Chronic Fatigue Syndrome
at http://www.ahmf.org/conf98.htm

REDDICK v CHATER, U.S. 9th Circuit Court of Appeals, October 6, 1998
http://laws.findlaw.com/9th/9715111.html

GEORGE W. MITCHELL v. EASTMAN KODAK COMPANY, U.S. 3rd Circuit Court of Appeals, May 8, 1997 **http://laws.findlaw.com/3rd/971585p.html**

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Employment

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National CFIDS Foundation

International CFS Information

Updated on: 10/05/1999

Employment for persons with chronic fatigue (PWC) is generally complicated due to the need for rest (typically horizontal bed rest) through out the working day. As a result, employment opportunities are limited as employees. This means that self-employment is often the only possibilities and yet the number of hours of work usually required to start up a business generally prevents this. The other option is jobs that may be done *telecommuting*. The following are some potential self-employment for suitably trained individuals:

- Writing and editing (assuming suitable skills and knowledge)
- Consulting (assuming suitable experience and market)
- Running an eBay based business (assuming access to a suitable product).

Disabilities

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International CFS Information

Updated on: 12/12/1999

The following disabilities are common with CFS. For resuming your present/former employment, these disabilities needs to be assessed. Items that affect driving (a requirement for most people to get to, and back from work) are noted.

It should assist Disability claims to have as many listed in your medical records as you have (ongoing or ***intermittent***)

- Fluid Balance Dysfunction: frequent bathroom breaks
- Temperature Regulation Dysfunction: environmental temperature must be carefully control (air conditioning/heating issues)
- Acquired Cognitive Dysfunction:
  - Loss of Verbal and Performance Intelligence: typically 20%
  - Dysfunction in Simultaneous Processing: lack of concentration, irritability, short term memory recall
  - Receptive and expressive dysphasia: decrease ability to understand verbal speech, use of incorrect speech syntax
  - Reading Comprehension Dysfunction: decreased ability or inability to understand written material
  - Sequencing Dysfunction: difficulties in filing, ordering information
  - Facial Agnosia: failure to recognize faces and names, "jamais vu"
  - Discalculia: difficult in doing simple arithmetic or calculations
  - Memory Dysfunctions:
  - Volition Dysfunction:
  - Proprioceptive Dysfunction: affects driving
  - Short term Amnesia: loss of recall of recent events
  - Tactile Dysfunction: misjudge weight and location

- Photophobia: inability to stand bright lights
- Latency of Accommodation: vision adjusts slowly for a change of distance (affects driving, etc)
- Nystagmus:
- Ophthalmoplegis (Internal and External):
- Tunnel Vision: (affects driving)
- Night Vision Loss: (drive to and from work may need to be during daylight hours)
- Color Vision problems:
- Palpebral Oedema
- Distance and Spacial Dysfunction: (affects driving)
- Depth of Field Dysfunction:

This partial list is from: Clinical Observations of Central Nervous System Dysfunction in Post-Infectious, Acute Onset M.E.CFS, Byron Hyde, MD, Anil Jain, M.D. 1992

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International CFS Information

Updated on: 01/01/2000

Any persons that have experienced a similar plight as mine should E-mail me ASAP at paulinsclu@aol.com. (This includes anyone, not just those that have experienced claim problems.)

AFFIDAVIT of PAUL D. HUGHES

On this day, Paul D. Hughes, appeared before me, the undersigned notary public, and after I administered an oath to him, he declared as follows:

My name is Paul D. Hughes. I am over the age of 18 years and have personal knowledge of the facts contained in this declaration. If called upon as a witness, I could and would testify to these facts.

1. I am a self-employed insurance agent and a Chartered Life Underwriter (CLU). I have been licensed by the state of Texas since 1972.

2. In 1989, I purchased Disability Income insurance from Indianapolis Life Insurance Company. The effective date of both policies is October 15, 1989.

3. After the date of purchase, Indianapolis Life in concert with UNUM Life Insurance Company engaged in a so-called "Joint Marketing Partnership."

4. In May 1993, I file a claim for benefits. Exhibit 1

5. After filing the claim, I was dismayed when I was informed that UNUM Life Insurance Company was responsible for my claim. Exhibit 2

6. In the following eleven months, I experienced: Exhibit 3 & 4
a. Intentional delays.
b. False and misleading statements regarding my medical records.
c. Attempt to change my diagnosis to a "mental disorder."
d. False and misleading statements regarding the coverage provisions.
e. Claim denied with no justifiable reason.
f. Forced to hire an attorney.

7. After eleven months, under the threat of litigation, Indianapolis Life and UNUM rescinded their previous denial and made a payment in the amount that was owed to me through December 1993. Payment included an additional $750 for attorney fees.

8. I am still due benefits from January 1, 1994, through the present and Indianapolis Life and UNUM have denied all these benefits.

9. Personal Statement: I believe that Indianapolis Life and UNUM denied my claim in bad faith. I have since become aware that UNUM has been involved or is a defendant in over 1700 lawsuits related to bad faith claims practices. As previously stated, I have been active in the marketing of insurance products for over 28-years. I have never before witnessed such malfeasance by these two companies. The insurance that I purchased as protection against a very real and dangerous peril has proven itself to be a defective product.

I affirm that the foregoing is true as to statements made upon information and belief, and as to those I believe them to be true.

Witness my hand under penalties of perjury this 6th. Day of December, 1999

_____

Paul D. Hughes
2807 Durant Drive
Midland, TX 79705

State of Texas
County of Midland
On December 6, 1999 before me, Paul D. Hughes, personally

appeared.

_____
Notary Public

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National CFIDS Foundation

International CFS Information

Updated on: 01/01/2000

Simplified Actuary Calculation

The latest rate for Chronic Fatigue Syndrome(CFS) is 422/100,000 [AMA Oct 11,1999]

Thus a long term disability policy that would pay $50000/year would require:

424/100000 * 50000= $212.00 per year to breakeven for CFS cases.

Fibromyalgia may be the same illness (most tests return positive for BOTH illnesses) and has a rate of 9 per 1000 [Adam's].

9/1000 * 50000= $450.00 per year to breakeven for FM cases.

So these two severe disabling diseases would need $662/year in premium to break even.

Well, I currently pay less than $250/year for $45000 coverage...

And what does having no clinical test mean? A severe disabling disease will produce depression in most people, and depression is a "mental illness" and thus limited to 2 years of payments in most Long Term Disability(LTD) policies (Thus $662/year is reduced to between $30 and $60/year - and the LTD make a profit).

The LTD companies are not collecting sufficient premiums for current CFS and FM claims (and have massive potential liabilities for past policies) if a clinical test for FM or CFS is accepted by the CDC.

CFS Friendly MD's

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Updated on: 01/05/2000

Physicians on this page have been deemed by their patients to accept CFS as a real illness and treat CFS patients with respect. These are NOT specialists - simply GP's that are CFS friendly!

A larger list of MDs is available at Co-Cure: http://www.co-cure.org/Good-Doc.htm

Canada

- Toronto, Ontario Dr. J.A.Sherkey, 168 Annette Street, Toronto, Ontario, Canada M6P 1P4
  Tel:(416)767-6383 Fax(416)767-4898
  (Interested in and is taking new patients with CFIDS/FMS. Dr Sherkey has the illness and can relate to the patients' concerns and problems.) More info

Arizona

- Tempe: Dr. Scott Rigden, 2501 E. Southern Ave , 480 820 4297
- Scottsdale: Dr. Gary L. Craine, 3501 N. Scottsdale Road, 602 941 5266

South Dakota

- Rapid City: Dr. Ray Strand

Washington

- Kingston: Dr. Susan Shliffer

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Stages

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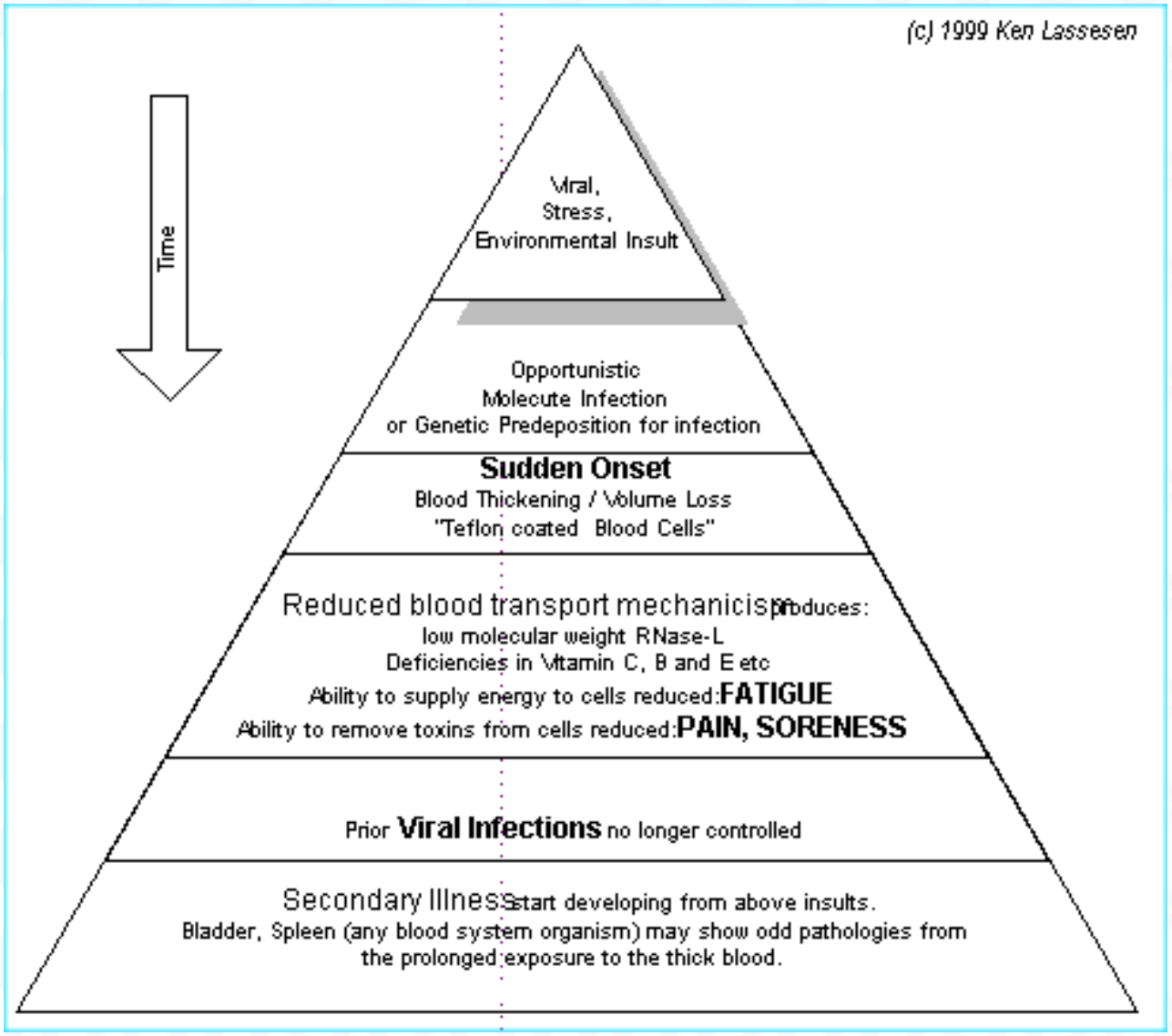
31893

National CFIDS Foundation

International CFS Information

Updated on: 10/30/1999

The diagram below shows a simple model for CFS built from the latest research findings.



The start point is assumed to be a weakened immune system that is attacked by small bacterial mycoplasmas (for example molecute).

They multiply until reaching a sufficient critical mass that the body's immune system is triggered. [1]

The immune system response is the activation of Coagulation, resulting in a thickening of the blood [2]. This thickening also reduces blood volume (while keeping blood pressure *normal*) and may result in a low red blood cell count. Somewhere in this process the body as part of the original activation, or due to lack of needed components, start to produce large quantity of low molecular weight RNase-L.

Once the blood thickens (reduced ability to transport), fatigue, pain and soreness occurs thru the entire body. The cell shapes may be significantly distorted (as reported in a New Zealand study).

Temperature sensitivities and circulation problems also results. Existing cell-stored vitamins are exhausted. The blood does not transport replacement vitamins well. The RDA requirements for some vitamins/minerals for many CFS patients may be 10 fold higher -- while other RDA requirements may be reduced because released cell-stored vitamins are not able to be purged from the body due to the restricted blood transportation mechanism.

The body is partially starving and partially gorging from this insult to the blood transportation system. The change of RNase-L allows past viral infections to become active. "Weak" parts of the body may suffer damage or change -- for example, loss of night vision from lack of nutrients to the eyes, or depression from lack of nutrients to the brain.

Notes

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International CFS Information

Updated on: 01/15/2000

From Jain, "General Information Post-Infectious, Acute Onset ME/CFS", 1992

Incubation period 1

4-10 days: [From outbreak data] From exposure until a mild flu like illness appears.

Incubation period 2

~ 21 days: From getting over a mild illness until sudden onset occurs. Emotional changes may be seen in patients for example: increased irritability.

Initial Stage

3 weeks to 6 months: time to develop the full set of symptoms

Recuperation Stage

2 weeks-24 months: Most likely time for recovery [very rarely a full recovery on close examination -- although patient may claim it is full]

Early Chronic Stage

1- 6 years: unstable level of dysfunction, impossible to separate the physical from the psychosocial aspects for rehabilitation purposes... cascading iteration of illnesses/symptoms.

Late Chronic Stage

6+ years: High rate of retroperitoneal carcinoma, lower rates of breast cancer, irregular cardiac rhythms develop.

The Disease

CFS is a hypercoagulable state induced an immune activation of the coagulation system [1] producing a condition known as "disseminated intra-vascular coagulation" (DIC)[5]. This activation appears to be triggered by microorganisms of the class *Molecutes*, small bacterial mycoplasmas, lacking cell walls, that are capable of invading several types of human cells and are associated with a wide variety of human diseases[17]. The name "Immune System Activation of Coagulation (ISAC) has been suggested[15]. This activation results in the production of low molecular weight RNase-L [2], a proposed clinical marker for CFS [3]. This may also account for the change of shape of the red blood cells reported in CFS patients [8].

This "thick blood" would then account for the significant (up to 47% of normal) reduction of blood volume reported by Streeten and Bell [4]. The mechanism for such a drop in blood volume without a corresponding drop of blood pressure was not understood.

This thickening of the blood may explain the chronic inflammatory changes of uncertain etiology found in a spleen removed from a CFS patient (for other reasons), that proceeded to recover after the removal of her spleen. [6]

Gulf War Syndrome (GWS)

The soviet biological weapon "Yellow rain" produces DIC [11]. The strong similarity between GWS and CFS symptomns have already been noted in the literature. Additional special interest may be a case of DIC in a political prisoner that died unexpectedly in South Africa (a nation how having hearings on biological weapons for apartheid). [12]

CFS Patient Appearance

"If you look around at support meetings you will notice that PWC's have a yellow to ashen cast to their skin."[7] and "I was told by a close friend when taking me to the emergency room in the beginning that I had gone gray in the face."[7], "Dr. Bell [leading researcher] says that pallor is a symptom he notes in almost all people with CFS." [7]

Diagnosis

Of the three best research indicators (RNase-L testing, Low blood volume and fibrinogen levels), fibrinogen levels is the simplest to obtain. "Physicians may call HEMEX to arrange for testing individual patients by calling 1-800-999-CLOT (2568)."[5] (website http://www.hemex.com) or from other labs [9]

Treatment

If fibrinogen levels are abnormal, Heparin appears effective in treatment of symptoms. For a first hand account of the results of this treatment read BD's Diary[16]. [Editor Note: This reads *very much like* my own reaction to a blood thinner]

Isoprinosine(R) has had good results and appears to target the activation. [13] Isoprinosine is also used in AIDS treatment.[14]

Warnings on Heparin:

In general, no problems....

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AutoImmune

AutoImmune

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Updated on: 01/18/2000

Parts of the immune system are:

- antigen-specific - they recognize and act against particular antigens (An antigen is any substance that elicits an immune response, from a virus to a sliver)
- systemic (not confined to the initial infection site, but work throughout the body).

These parts have memory (recognize and mount an even stronger attack to the same antigen the next time) [*].

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Updated on: 10/05/1999

Blood Volume Symptoms

The following symptoms are *predictable* due to low blood volume: Low fluid volume results in higher concentrations of substances in the blood as well as severe reduction of blood to non-essential areas.

Symptoms	Explanation	Actions:
Sensitivity to heat and cold - Chill easily - Sick from heat	Impaired ability to transfer heat and cold since blood is the 'heat transfer' material	- Buy air conditioner - Keep house at moderate temperature always
High Iron Count	Higher Concentrations	- Test for excess Iron (10% chance)
Increased Sensitivity	A normal amount of histamine pouring into a reduced blood volume	- Maintain a low-food sensitivity diet. - Maintain an anti-alergy house
Aching muscles and joints	Reduced ability of toxins and wastes to be removed. Fluid to non-active locations reduced	- *Light* activity will increase blood flow allowing toxins and wastes to be removed
Drug Sensitivity	A normal amount of drug pouring into a reduced blood volume	- Reduced dosages

Research

"Following oxygen inspiration, participants showed significantly improved simple and choice reaction times and a trend towards improved word recall" in *Cognitive defects in chronic fatigue*

syndrome are reversed by oxygen administration

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

CFIDS/ME
Identification and Treatment

Ken Lassesen, MS

- Moderator of Egroups CFIDS Treatment Lists:
  - CFSFMExperimental
  - CFSFM_Antibiotics
  - CFS-Ampligen

From Cellular Anoxemia caused by the presence of Rickettsiae to CFS

Padua, September 1999

CL Jadin MD MBBCh Johannesburg South Africa

Rickettsial infection was discovered in 1909, when Ricketts saw and described the germ that causes R M S F in man. Ricketts, as well as another scientist, Prowazek, contracted Typhus and died.

1. The epidemic forms of Rickettsiae were described by Zinsser in his classic book “Rats, Lice and History”, in which he contends that soldiers have rarely won wars. Typhus and other infectious diseases have decided the outcome of more military campaigns than Caesar, Hannibal, Napoleon (2) and all generals in history. Depending on the outcome for each warring faction, either the epidemics were blamed for defeat, or the generals were credited with victory. It has contaminated an estimated 25 million Russians, causing 3 million deaths during the 1st World War.

2. Nowadays, following on from these historical memories, there are forms less virulent, evolving slowly, but able to induce vascular and neurological pathologies (52).

3. Rickettsiae are found in ticks, lice, fleas, mites, meat, milk, stools and dust. From the entry into the skin, the lungs, conjunctives, and the digestive mucosa, Rickettsiae spread via the bloodstream to infect vascular endothelium. These organisms grow and multiply by binary fission in and only in the cytoplasm of the host cell until the number of Rickettsiae is so great that the cell bursts, releasing hundreds of them. This invasion will impair or paralyse the vascular function, acting like a sponge between blood and organs. They will enlarge the endothelial cells of small vessels with partial or complete occlusion of the vascular lumen. They are known for long survival in various organs and lymphatic tissue. According to which vessel they invade, they might display an amazing constellation of diseases:

- CFS, Fibromyalgia, where they cause a cellular anoxemia (41,47).
- Cardio-vascular diseases (3,4,5,11,12,27,39,42,45).

- Neurological diseases (from acute encephalitis to MS, epilepsy etc) (3,5,9,43).
- Abdominal diseases (appendicitis, cœliac disease and others) (23).
- Ocular diseases (uveitis, retinal angiopathy, optic nevritis sometimes a long time after a general infection) (3,29).
- AutoImmune diseases (41,47).

Rickettsiae release into the bloodstream angiotropic, endotoxins, producing inflammations, allergies and demyelinisation (H Perron).

4. Rickettsiae:

- are resistant to humidity and to dryness
- will stay virulent for;
- 60 days in milk
- 4 months in sand
- 6 months in meat
- 7 - 9 months in cotton (4).

They are spread by rodents and birds. Through the centuries, bird migration has been responsible for changing the geographical distribution of disease (27) - but this is nothing compared to the effect of the explosion of these diseases due to the cocktail effect created by distribution through global air traffic (26).

Equally the transport of insects compared to the import and export of livestock - as in the case of the importation of 10,000 parrots from Paraguay to Belgium when some 2,000 died, leaving the virus well and alive behind them (27), (identified by JB Jadin as Neo-Rickettsia Bedsonia).

This world distribution does not include Antarctica, where they do not survive.

Fish also share this disease, as Erlichioses is, according to breeders, a common problem (31). They have been found in oysters by Deltreil.

5. 3,600 patients presented with CFS, Fibromyalgia, RA, depression and MS have been diagnosed as suffering from Chronic Rickettsial Infection (CRI) after eliminating other diseases as a cause (diabetes, cancer etc.).

The majority of my patients report a flu-like infection, with often an elevated temperature and severe headaches.

This lasts for a few days, disappears or reoccurs, and then leaves them with a chronic condition of CFS, Fibromyalgia etc. as mentioned above.

6. Diagnosis of CRI is established by Giroud's Micro-Agglutination test against five strains of Rickettsiae:

- R. Prowazeki: the epidemic type of Typhus
- R. Mooseri, which is endemic
- R. Conori, which belongs to the spotted fever group
- Coxiella Burnetti, which is well known as Q Fever. It has 2 phases; Phase II is pathogenic
- Neo Rickettsia Chlamydiae which has an affinity for uteral mucosa, and will be the cause of many abortions.

Important Points:

a) A high reading means a high serological level of antibodies - a negative reading in endemic areas reflects the poverty of the immune system (24).

b) Agglutination happens or does not - therefore there is no possibility of personal interpretation. Test quality depends on Antigen quality (3).

c) Positive tests can be found in people who display no symptoms (Giroud, Jadin (18); 26% according to Drancourt (39)).

7. However, the Micro-Agglutination test of Giroud is not our only tool to establish the diagnosis of Rickettsial infections. We find the following blood tests most relevant:

a) LFT: the hepatotoxicity of Rickettsiae has been reported as early as 1937 by Derrick in Q Fever (19, 29), followed by many others - Giroud, Lenette, Legag, Brezina, Perron, Kelly, Raoult, etc. In these cases, Tetracyclines are improving or normalising liver function (6).

b) Iron study (50% of abnormalities corrected with Tetracyclines only and when necessary with a short course of iron supplement).

c) Thyroid AB rather than TFT, although the TFT show abnormalities in 3% of patients, the thyroid AB are elevated in 28% of cases and improve or normalise rapidly with treatment.

d) CRP, RF, ANF, WR was positive in 53% of patients, (39) and also improved with treatment and often normalised.

e) Mycoplasma, first classified as a Rickettsia, is now considered to be an independent entity.

8. Patients' symptoms most commonly exhibited are:

· Tiredness (4,5)
· Headaches, retro-orbital and temporal, worst after prolonged horizontal position or mental effort (4).
· Myalgia (3)

· Arthralgia migrating (2,3,5)
· Loss of balance (29)
· Vision abnormalities (3,29)
· Raynaud syndrome (18)
· Nausea (8,9,18)
· Recurrent sore throat (23)
· Memory and concentration deficit (4).
· Chest pain, palpitations (8,12, 18)
· Sweats, low grade fever (4)
· Bruising (4)
· Psychological and neurological disorders(4,5,18,29,30)

9. We find quite a constant guideline in the physical examination, which often shows

- An inflamed throat
- Multiple adenopathies
- Heart abnormalities (vascular (4,12,30) and valvular impact (2, 39))
- RIF tenderness (chlamydiae 18 in appendix (23))

10. Treatment is administered:

- Guided by our predecessors, (Giroud, Jadin, Legag etc.)
- And by my own daily, private lessons (each patient is one).

The treatment consists of 7 to 12 days per month of a specific **Tetracycline**. The monthly treatment aims to follow the Rickettsial development in the cell.

a) A **high** dosage is required (4,5) with the limitation of:

- Safety (32) Goodman et al (33) highlights irreversible hepatotoxicity in intravenous administration only. Our experience was that when liver functions were normal to start with, they stay normal. If they were abnormal, they will improve during treatment and generally return to normal. Cases of fatty acid depots (as shown by liver scan, before and after 6 months to 1 year of treatment) have disappeared (1 MS, 4 ME). This confirms the fact that Rickettsiae are more hepatotoxic than Tetracyclines.

- Tolerance.

The gastric intolerance will be successfully prevented by using a gastric pump inhibitor during and if necessary

before and after the administration of the Tetracyclines.

The tolerance of the treatment is directly related to the Herxheimer reaction (4, 6, 26, 37), which is a reactivation of old symptoms and/or exacerbation of present symptoms that occurs on antibiotherapy. Its presence has a very important diagnosis and prognosis value (4). They might or might not be parallel to a serological reactivation. It will fade with the number of treatments received. When very severe, the HR is treated with Probenecid.

b) The Tetracyclines are **alternated** because:

- A patient is frequently contaminated by many strains of Rickettsiae (5) and different Rickettsiae have different sensitivity to different Tetracyclines or combinations. (4).

- A patient might build resistance to each Tetracycline (4, 17).

- Patients show individual sensitivity to different Tetracyclines or combinations and there is very often a privileged reaction to a specific treatment (6).

c) The Tetracyclines are **combined** with Quinolones, Macrolides or Metronidazole (7), because Rickettsiae present a wide heterogenicity of susceptibility to different drugs (4).

d) The treatment is often **long** due to:

- The chronicity of the germ (4)

- The multiple foci of Rickettsiae (18)

- The fact that Rickettsiae have a slow evolution and some foci are dormant, encapsulated and therefore protected from antibiotherapy. Only when they become active can they be treated (5).

e) Each treatment will allow the immune system to produce and maintain a proper and efficient level of antibodies. This happens each time the antigen Rickettsiae are released from the cell to the blood stream while on antibiotherapy (Legag) (4).

f) The length of the disease should logically imply a lengthy treatment. In our experience, this point is not always true. Patients, ill for many years, may recover after a few months treatment.

g) **Antimalaria drugs** have been found efficient to improve Rheumatoid symptoms and Rheumatoid biological findings (see patients' files).

h) **Adjuvants** such as Vitamin B complex and acidobacillus are also used.

i) Cortisone is avoided as much as possible as it is known to weaken the Immune System in general (3) and also to reactivate the disease in experiments on guinea-pigs (39). Cortisone has been accused of interfering with the diagnosis of Rickettsia by masking the antibody level (4).

j) **Exercise** is recommended, for the following 3 reasons:

- Rickettsiae is a vascular disease and exercise, properly done, will improve the smooth peri-vascular muscle function, as well as develop our biggest muscle, the heart.
- The fact that strains of Rickettsiae grow better in vitro when maintained in a CO2 enriched atmosphere (34).
- The suggestion that Rickettsiae grow best when the metabolism of the host cell is low (38).

k) **Hot baths** are important to eliminate toxins via the skin, produced by Rickettsiae antigens when liberated in the bloodstream by antibiotherapy.

10. Reinfection may obviously occur. Reactivation (called so rather than relapse) may also happen due to the interaction of bacteria, virus, stress, pollution, etc. causing the Rickettsiae forms' to change from dormant to active (35).

11. Measurement of Progress - Patients are seen monthly to judge progress on:

a) Symptoms

b) Activity increase (From bedridden to back to exercise or back to work)

c) From being treated by painkillers, antidepressants, sedatives, cortisone to none

d) Medical examination

e) Biological investigation: from having:

- ESR
- LFT
- RF raised
- CRP raised - Back to normal, or nearly so
- ANF raised
- KFT raised
- Thyroid antibodies raised

· Iron

12. Based on this assessment, the treatment is prolonged or stopped (3 months to 2 years: 8 months on average). However, as previously mentioned, the length of treatment is not directly correlated to the length of illness:

Therefore patients can be divided into 2 categories:

¨ Fast progress - their illness was mainly Rickettsia

¨ Slow progress - their illness was Rickettsia plus other factors (20).

"La salute e come un pallone d'aria calda; a volte bisogna rilasciare il peso"

Health is like a hot air balloon. You have to get rid of excess burdens to keep it in the air. Rickettsia is the easiest one to lose

Discussion

1) 12 years ago, one of my friends became unable to walk and was diagnosed as having ME. For 4 years I suggested the diagnosis of Rickettsial Infection, and therefore the Weil-Felix test was performed several times in South Africa, but the results were negative. My friend developed an acute appendicitis. After I removed her appendix, her serum was sent to Prof. JB Jadin in Belgium to test for Rickettsiae, and the result was positive. I treated her with Tetracyclines and 3 weeks later, from being in a wheelchair; she was riding her horse again. I was sceptical. But this case brought me 200 patients and the publicity surrounding an investigation of my methodology by the South African Medical Council brought me several thousand more. Thus I started to focus on the Rickettsial approach.

2) There are many reasons suggesting the infectious etiology and, more specifically, Rickettsial-like organisms of CFS. Amongst those reasons:

a) Consider the following :

- CFS was first reported in Incline, Nevada in 1984 (1) and developed into epidemic proportions.
- Rocky Mountain Spotted Fever originated from the same place in 1916 (9,29).
- The spirochete Borrelia Duttoni, first blamed for causing the recurrent Malgache fever described in the journals written by Drury in 1702 (24) in Madagascar, then by Scheltz in the Belgian Congo in 1933, by Palakov in

Cape Town in 1944, by Heisch in Kenya in 1950.

- Lyme Disease appeared (or reappeared?) more recently in Lyme, Connecticut in 1975 (Borrelia Burgdorferi) (25). As Lyme Disease is a new name for Malgache Fever, could CFS be a new name for Rickettsial Disease?

All of the above highlights the life of a germ as an individual emerging and disappearing in a wave pattern epidemically and historically. Like us, germs have to adapt, producing new variations of themselves, (not new species), that may or may not survive on their own, with or without the help of another germ. This is circumstance-dependent, and these particular circumstances will never reoccur. Some of those variations will acquire specific and consistent characteristics.

This is their 'civilisation'. We only see them when they succeed, and only then do new avenues of investigation open up, while others are abandoned.

b) A link has been established between Florence Nightingale disease and CFS (21). The fact that she was working surrounded by lice, fleas and ticks, treating soldiers with wounds and with epidemic typhus during the Crimean war, could be a logical explanation as to why she was terribly tired during the last 2 decades of her life; and possibly has relevance to Gulf War illnesses (13).

c) Lymphocyte studies conducted on sheep with tick-borne diseases (14), CFS patients (15,16) and patients with Q Fever endocarditis (11) are showing amazingly similar results.

d) Coincidentally, the new name suggested in the Lancet for CFS is PQFS (Post Q Fever Syndrome) in April 1996 edition (22).

e) As mentioned above, during the First World War an estimated 25 million Russians contracted Louse-borne epidemic typhus, resulting in 3 million deaths. Why not before or after? It could suggest that the stress factor reactivates the virulence of Typhus Prowazeki (2, 3, 9). In the medical history of CFS patients, stress has often been described as the start of the illness.

f) The symptoms displayed by CFS, Fibromyalgia, RA, and even neurological patients as MS, show the same diversity of symptoms as Rickettsial patients. How many scientists blamed the diversity of symptoms for misleading unprepared practitioners in the diagnosis of chronic Rickettsial infection (30)? That same diversity could have contributed to the delay in recognising CFS. French authors (Giroud, Jadin, Legag) attribute those multiple aspects to a generalised micro-vascular invasion. They widely demonstrated the persistence of Rickettsiae in the vessels (4), (18). The suggestion here is that the well-known, well-documented entity of Rickettsial disease, showing the same symptoms as the newly arrived CFS, might simply, partially or totally be caused by the same agent.

g) The last, but not the least reason, is the success rate of the Rickettsial treatment, Tetracycline, applied on CFS, Fibromyalgia etc. patients. Dr Phillipe Bottero on 100 patients, Dr Peter Tableton on 300 patients (17) and myself on a much larger number of patients, maintain an 84% to 96% recovery rate.

CFS - Rickettsial Infection: Sources of References

-

Presented by Dr. Cécile Jadin, Johannesburg, South Africa 1999

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International CFS Information

Updated on:
01/13/2000

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

The Rickettsial Approach and treatment of patients presenting with CFS, Fibromyalgia, Rheumatoid Arthritis and Neurological Dysfunction.

Manly Conference

February 1999

CL Jadin MD MBBCh

Republic of South Africa

Introduction

The author, Cécile Jadin, is originally from Belgium, but has been practising in South Africa for the last 17 years. She is a surgeon by profession. In South Africa, in addition to practising as a surgeon, she also assisted her husband in his general practice. For the last 7 years, she has focused on the subject of Rickettsia and her approach has naturally been that of a clinician, and it is in this context that the paper is written.

To understand why she took the Rickettsial approach her background needs to be explained. Her father was Professor JB Jadin, who undertook groundbreaking research on tropical diseases, among them Rickettsial infection, with Professor Paul Giroud in Central Africa, South Africa, the Near East, and in Europe, developing the work started in the Pasteur Institute of Tunis, with Professor Charles Nicolle, who was a disciple of Louis Pasteur. Thus she was familiar with those germs from an early age and her work represents the results of teamwork through the last 100 years.

12 years ago, one of her friends became unable to walk and was diagnosed as having ME. For 4 years Dr. Jadin suggested the diagnosis of Rickettsial Infection, and therefore the Weil-Felix test was performed several times in South Africa, but the results were negative. The friend developed an acute appendicitis. After Dr. Jadin removed her appendix, her serum was sent to Prof. JB Jadin in Belgium to test for Rickettsiae, and the result was positive. Dr. Jadin treated her with Tetracyclines and 3 weeks later, she was riding her horse again. Dr. Jadin was sceptical. But this case brought her 200 patients and the publicity surrounding an investigation of her methodology by the South African Medical Council brought her several thousand more. Thus Dr. Jadin started to focus on the Rickettsial approach.

Original Research

Research on Rickettsioses was originally developed by French, Polish and Russian scientists. They followed Charles Nicolle's (Pasteur Institute, winner of the Nobel Prize for medicine in 1933) hypothesis, which is that occult diseases are a reality and their cohabitation in the same host will lead to the bankruptcy of the immune system (8). By occult disease Charles Nicolle implies the asymptomatic stage of the disease, where the agent is present in the host, but dormant (3). The emergence of a virus, bacteria, stress or pollution can activate this agent, which leads to the symptomatic stage.

An example of this cohabitation is the infant mortality rate described by J.B. Jadin in Central Africa. Neonates diagnosed with malaria and Coxiella Burnetti all died as opposed to those with malaria only (20).

The numerous publications of these authors are unfortunately all in French, so their circulation was limited. They also, as academics, excluded the media. Therefore the real importance of their discovery is still to be made widely known.

Rickettsia and CFS

The fairly recently recognised entity of CFS gives us a perfect opportunity to try the etiological route to understand this disease. Along this route we will automatically enter other medical fields, inviting us to consider an infectious etiology in cardiology (4,5,9,11,12), in psychiatry (3,17), in neurology (3,29) and in rheumatology (28), rather than describing the symptoms and gathering them into syndromes (20, 40).

Obviously one germ can cause many diseases depending on a selective topicality for one or more particular tissues as well as one disease can be caused by different germs alone or simultaneously. Therefore we would like to concentrate on the causative agent, rather than on the name of, and the criteria to classify, the diseases.

There are many reasons suggesting the infectious etiology and, more specifically, Rickettsial-like organisms of CFS. Amongst those reasons:

1. Consider the following :

- CFS was first reported in Incline, Nevada in 1984 (1) and developed into epidemic proportions.
- Rocky Mountain Spotted Fever originated from the same place in 1916 (9,29).
- The spirochete Borrelia Duttoni, first blamed for causing the recurrent Malgache fever described in the journals written by Drury in 1702 (24) in Madagascar, then by Scheltz in the Belgian Congo in 1933, by Palakov in Cape Town in 1944, by Heisch in Kenya in 1950.
- Lyme Disease appeared (or reappeared?) more recently in Lyme, Connecticut in 1975 (Borrelia Burgdorferi) (25).

Could these be new names for old diseases?

All of the above highlights the life of a germ as an individual emerging and disappearing in a wave pattern epidemically and historically. Like us, germs have to adapt, producing new variations of themselves, (not new species), that may or may not survive on their own, with or without the help of another germ. This is circumstance-dependent, and these particular circumstances will never reoccur. Some of those variations will acquire specific and consistent characteristics that will allow them to survive. This is their 'civilisation'. We only see them when they succeed, and only then do new avenues of investigation open up, while others are abandoned.

1. A link has been established between Florence Nightingale disease and CFS (21). The fact that she was working surrounded by lice, fleas and ticks, treating soldiers with wounds and with epidemic typhus during the Crimean war, could be a logical explanation as to why she was terribly tired during the last 2 decades of her life; and possibly has relevance to Gulf War illnesses (13). Zinsser has developed the same concept in his classic book "Rats, Lice and History". He contends:

"Soldiers have rarely won wars. Typhus and other infectious diseases have decided the outcome of more military campaigns than Caesar, Hannibal, Napoleon and all generals in history. Depending on the outcome for each warring faction, either the epidemics were blamed for defeat, or the generals were credited with victory." (2). More examples of this phenomenon were reported by JB Jadin (29).

2. Lymphocyte studies conducted on sheep with tick-borne diseases (14), CFS patients (15,16), and patients with Q

Fever endocarditis (11) are showing amazingly similar results.

3. Coincidentally, the new name suggested in the Lancet for CFS is PQFS (Post Q Fever Syndrome) in April 1996 edition (22).

4. During the First World War an estimated 25 million Russians contracted Louse-borne epidemic typhus, resulting in 3 million deaths. Why not before or after? It could suggest that the stress factor reactivates the virulence of Typhus Prowazeki (2, 3, 9). In the medical history of CFS patients, stress has often been described as the start of the illness.

5. The symptoms displayed by CFS, Fibromyalgia, RA, and even neurological patients as MS, show the same diversity of symptoms as Rickettsial patients. How many scientists blamed the diversity of symptoms for misleading unprepared practitioners in the diagnosis of chronic Rickettsial infection (30)? That same diversity could have contributed to the delay in recognising CFS. French authors (Giroud, Jadin, Legag) attribute those multiple aspects to a generalized micro-vascular invasion. They widely demonstrated the persistence of Rickettsiae in the vessels (4), (18). The suggestion here is that the well-known, well-documented entity of Rickettsial disease, showing the same symptoms as the newly arrived CFS, might simply, partially or totally be caused by the same agent.

6. The last, but not the least reason, is the success rate of the Rickettsia treatment, Tetracycline, applied on CFS, Fibromyalgia, Depression and MS etc. patients. Dr Phillipe Bottero on 100 patients since 1981, Dr Peter Tarbleton on 300 patients in 1993 in South Africa(17) and myself on a much larger number of patients, maintain an 84% - to 96% recovery rate.

Transmission of Rickettsiae

Rickettsiae are transmitted by arthropods (36), except for Q Fever, which does not really need vectors;

- they are resistant to humidity and to dryness
- they will stay virulent for 60 days in milk
- 4 months in sand
- 6 months in meat
- 7 - 9 months in cotton (4).

They are spread by rodents and birds. Through the centuries, bird migration has been responsible for changing the geographical distribution of disease (27) - but this is nothing compared to the effect of the explosion of these diseases due to the cocktail effect created by distribution through global air traffic (26).

Equally the transport of insects compared to the import and export of livestock - as in the case of the import of 10,000 parrots from Paraguay to Belgium when some 2,000 died, leaving the virus well and alive behind them (27), (identified by my father as Neo-Rickettsia Bedsonia).

This world distribution does not include Antarctica, where they do not survive.

Fish also share this disease, as Erlichioses is, according to breeders, a common problem (Psichi Rickettsia Salmoni, first

described in Chile) (31).

Patients and Diagnosis

3,400 patients presented with CFS, Fibromyalgia, RA, depression and MS have been diagnosed as suffering from Chronic Rickettsial Infection (CRI) after eliminating other diseases as a cause (diabetes, cancer etc.).

The majority of my patients report a flu-like infection, with often an elevated temperature and severe headaches. This lasts for a few days, disappears or reoccurs, and then leaves them with a chronic condition of CFS, Fibromyalgia etc. as mentioned above.

Diagnosis of CRI is established by Giroud's Micro-Agglutination test against five strains of Rickettsiae:

- R. Prowazeki: the epidemic type of Typhus
- R. Mooseri, which is endemic
- R. Conori, which belongs to the spotted fever group
- Coxiella Burnetti, which is well known as Q Fever. It has 2 phases; Phase II is pathogenic
- Neo Rickettsia Chlamydiae which falls into the Neo-Rickettsia group (18)

Important Points:

a) A high reading means a high serological level of antibodies - a negative reading in endemic areas reflects the poverty of the immune system (24).

b) Agglutination happens or does not - therefore there is no possibility of personal interpretation. Test quality depends on Antigen quality (3).

c) Positive tests can be found in people who display no symptoms (Giroud, Jadin (18); 26% according to Drancourt (39)).

However, the Micro-Agglutination test of Giroud is not our only tool to establish the diagnosis of Rickettsial infections. We find the following blood tests most relevant:

- LFT: the hepatotoxicity of Rickettsiae has been reported as early as 1937 by Derrick in Q Fever (19, 29), followed by many others - Giroud, Lenette, Legag, Brezina, Perron, Kelly, Raoult, etc. In these cases, Tetracyclines are improving or normalizing liver function (6).
- Iron study (50% of abnormalities corrected with Tetracyclines only and when necessary with a short course of iron supplement).
- Thyroid AB rather than TFT, although the TFT show abnormalities in 3% of patients, the thyroid AB are elevated in 28% of cases and improve or normalise rapidly with treatment.

- CRP, RF, ANF, WR was positive in 53% of patients, (39) and also improved with treatment and often normalised.

- Mycoplasma (only researched after the Manly conference, February 1998).

Symptoms

Patients' symptoms most commonly exhibited are:

· Tiredness (4,5)
· Headaches, retroorbital and temporal, worst after prolonged horizontal position or mental effort (4).H
· Myalgia (3)
· Arthralgia migrating (2,3,5)
· Loss of balance (29)
· Vision abnormalities (3,29)
· Raynaud syndrome (18)
· Nausea (8,9,18)
· Recurrent sore throat (23)
· Memory and concentration deficit (4).
· Chest pain, palpitations (8,12, 18)
· Sweats, low grade fever (4)
· Bruising (4)
· Psychological and neurological disorders(4,5,18,29,30)

We find quite a valuable guideline in the physical examination, which often shows

- An inflamed throat and multiple adenopathies, reflecting the selective topicality of Rickettsiae to endothelial tissue

- Heart abnormalities (vascular (4,12,30) and valvular impact (2, 39))

- RIF tenderness (chlamydiae 18 in appendix (23))

Treatment

After establishing these 3 cornerstones Symptoms

Physical examination

Blood tests

treatment is administered:

- Guided by our predecessors, (Giroud, Jadin, Legag etc.)

- Refined by our contemporaries, (Bottero and Raoult)

- And by my own daily, private lessons (each patient is one).

The treatment consists of 7 to 12 days per month of a specific Tetracycline. The monthly treatment aims to follow the Rickettsial development in the cell.

1. A high dosage is required (4,5) with the limitation of:

- **Safety** (32) Goodman et al (33) highlights irreversible hepatotoxicity in intravenous administration only. Our experience was that when liver functions were normal to start with, they stay normal. If they were abnormal, they will improve during treatment and generally return to normal. Cases of fatty acid depots (as shown by liver scan, before and after 6 months to 1 year of treatment) have disappeared (1 MS, 4 ME). This confirms the fact that Rickettsiae are more hepatotoxic than Tetracyclines.

- **Tolerance**.

a) The gastric intolerance will be successfully prevented by using a gastric pump inhibitor during and if necessary before and after the administration of the Tetracyclines.

b) The tolerance of the treatment is directly related to the Herxheimer reaction (4, 6, 26, 37), which is a reactivation of old symptoms and/or exacerbation of present symptoms that occurs on antibiotherapy. Its presence has a very important diagnosis and prognosis value (4). They might or might not be parallel to a serological reactivation. It will fade with the number of treatments received. When very severe, the HR is treated with Probenecid.

1. The Tetracyclines are alternated because:

a) A patient is frequently contaminated by many strains of Rickettsiae (5) and different Rickettsiae have different sensitivity to different Tetracyclines or combinations. (4).

b) A patient might build resistance to each Tetracycline (4, 17).

c) Patients show individual sensitivity to different Tetracyclines or combinations and there is very often a privileged reaction to a specific treatment (6).

3. The Tetracyclines are combined with Quinolones, Macrolides or Metronidazole (7), because Rickettsiae present a wide heterogenicity of susceptibility to different drugs (4).

4. The treatment is often long due to:

a) The chronicity of the germ (4)

b) The multiple foci of Rickettsiae (18)

c) The fact that Rickettsiae have a slow evolution and some foci are dormant, encapsulated and therefore protected from antibiotherapy. Only when they become active can they be treated (5).

d) Each treatment will allow the immune system to produce and maintain a proper and efficient level of antibodies. This happens each time the antigen Rickettsiae are released from the cell to the blood stream while on antibiotherapy (Legag) (4).

e) The length of the disease should logically imply a lengthy treatment. In our experience, this point is not always true. Patients, ill for many years, may recover after a few months treatment.

3. Antimalaria has been found efficient to improve Rheumatoid symptoms and Rheumatoid biological findings (see patients' files). Christopher Columbus knew it in the 15th century, as he gave tree bark containing quinine to his crew to prevent malaria and also mysterious body pains. The Imperial army of Queen Victoria did the same and so was born Indian Tonic water.

4. Adjuvants such as Vitamin B complex and acidobacillus are also used.

5. Cortisone is avoided as much as possible as it is known to weaken the Immune System in general (3) and also to reactivate the disease in experiments on guinea-pigs (39). Cortisone has been accused of interfering with the diagnosis of Rickettsia by masking the antibody level (4).

6. Exercise is recommended, for the following 3 reasons:

- Rickettsiae is a vascular disease and exercise, properly done, will improve the smooth peri-vascular muscle function, as well as develop the most important muscle, the heart.
- The fact that strains of Rickettsiae grow better in vitro when maintained in a CO2 enriched atmosphere (34).
- The suggestion that Rickettsiae grow best when the metabolism of the host cell is low (38).

7. Hot baths are important to eliminate toxins via the skin, produced by Rickettsiae antigens when liberated in the bloodstream by antibiotherapy.

8. Reinfection may obviously occur. Reactivation (called so rather than relapse) may also happen due to the interaction of bacteria, virus, stress, pollution, etc. causing the Rickettsiae forms' to change to active from dormant (35).

Measurement of Progress

Patients are seen monthly to judge progress on:

1. Symptoms
2. Activity increase (From bedridden to back to exercise or back to work)
3. From being treated by painkillers, antidepressants, sedatives, cortisone to none
4. Medical examination
5. Biological investigation: from having:

- LFT
- CRP raised
- KFT raised
- Iron
- RF raised
- ANF raised
- Thyroid antibodies raised

Back to normal, or nearly so

Based on this assessment, the treatment is prolonged or stopped (3 months to 2 years: 8 months on average). However, as previously mentioned, the length of treatment is not directly correlated to the length of illness:

Therefore patients can be divided into 2 categories:

1. Fast progress - their illness was mainly Rickettsia

2. Slow progress - their illness was Rickettsia plus other factors (20).

"La santé est comme une mongolfière: il faut parfois lâcher du lest"

Health is like a hot air balloon. You have to get rid of excess burdens to keep it in the air. Rickettsia is the easiest one to lose

Appendix 1: CFS - Rickettsial Infection: Sources of References

-

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Updated on: 03/14/2000

The drugs are ***not*** a cure, they only grant symptom relief ("buys some brain time").
Do not view any recovery using these drugs as a cure or remission of CFIDS.

"Smart Drugs" is a class of compounds developed to improve brain function. Since most CFIDSers suffer from significant loss of brain function ("brain fog"), they are a logical symptomatic supplement. Many of these are also anticoagulants, and thus have significant benefits for many. [General introduction, Summary]

The FDA has not approved the use of most of these drugs *["Smart Drugs" and hippies's "mind expanding drugs" are associated in some bureaucrat's minds]*. They are not available from most US/Canadian/British pharmacies even with a prescription (they may be available *with prescriptions* from some specialized pharmacies "compounding pharmacies"). On the other hand, these drugs are available "over-the-counter" in much of Europe and may be ordered on the internet.

"A little known FDA ruling now allows the importation of a three-month personal supply of drugs as long as they are *regarded as safe in other countries*. Ordering safe but unapproved drugs is now legal under the new FDA pilot guidelines, Chapter 971."[*]

							same base molecule				
Drug	Pyritinol, Pyrithioxine [Encephabol]	D	H	Centrophenoxine, [Lucidril]	DMAE Dimethylaminoethanol	Vinpocetine [Cavinton, Ceractin]	Aniracetam	Oxiracetam	Pramiracetam [NeuPramir]	Pyroglutamic acid, Oxoproline	Piracetam [Nootropil®]
Drug Fact Sheet	[*],[*]	[*][*]	[*], [*], [*], [*]							[*]	[*],[*]
Sleep Disorders	Yes	Yes[*]		No, Can cause Insomnia		Yes [*]	Yes [*]				No, can cause insomnia
Emotional liability / depression	Yes	antidepressant [*]		No, Can cause excitability, increase depression			Yes [*]				
Memory	Yes			Yes [*]	Yes	Yes [*]	Yes[*]	Yes[*]		Yes	Yes
Concentration	Yes							Yes[*]			
Reduces Hypoxia effects	Yes [*]		Yes			Yes [*]	Yes [*]	Yes[*]			
Antioxidant	Yes [*]			Yes [*]		Yes [*]		No [*]			No [*]
Eye Problems						Yes [*]					
Libido		Yes									Yes
Immune Enhancer	Yes [*]	Yes [*]									
Comments	Pyritinol molecule is structurally similar to Vitamin B6 (Pyridoxine)		Anticoagulant			anti-platelet aggregation, Available in US[*] inhibition of aggregation of thrombocytes[*]			More effective form of Piracetam		anticoagulant
Warnings	[A]	[E]	[C]				[B]				

[*] Click to see source document.

General Note: Usually take them early in the day and not in the evening. Many have a short term stimulant effect.

[A] Significant side effects are reported with rheumatoid arthritis [which may be mycoplasma based - like some CFIDS cases] [*]

[B] Not approved in any country [*]

[C] OVERDOSE DANGER: The symptoms of overdosage with Hydergine are nasal stuffiness, flushing of the face, headache, nausea and vomiting, tremulousness, spasticity, hypotension, circulatory collapse and coma.[*]

[D] To date Selegiline has been medically approved by regulatory agencies for use only in treatment of Parkinson's disease [*], approved by the Food and Drug Administration for the control of the clinical signs associated with canine Cognitive Dysfunction Syndrome[*]

[E] risk of adverse effects when it is used in combination with selective serotonin reuptake inhibitors and tricyclic antidepressants [*], many other risks [*]

Depression, Psychotic Dysfunction, Rickettsial Infection Case Study

by

Dr. Cécile Jadin, Randburg, South Africa

Summary: The possibility of a Rickettsial origin for symptoms of depression and psychotic disfunction has been suggested by French scientists (Ch. Nicolle, Giroud, Legag, Jadin, Bottero) in their published works. Hence 300 patients, diagnosed as suffering from depression, or other neuropsychiatric dysfunction have been treated with antibiotic where a positive indicaton of Rickettsial infection was revealed as follows:

1. Many symptoms of these patients were similar to those exhibited in chronic Rickettsial diseases.

2. The treatment followed the finding that their serum reacted positively to the **Giroud** micro-agglutination test.

Giroud Test - specific for testing antibodies to these 5 antigens (**R36**):

§ Rickettsia Prowazeki
§ R. Mooseri
§ R. Conori
§ Coxiella Burnetti
§ Neo-R. Q18

Done by micro agglutination

Depends on the quality of antigens
Comparative studies with IFA test gave very similar result

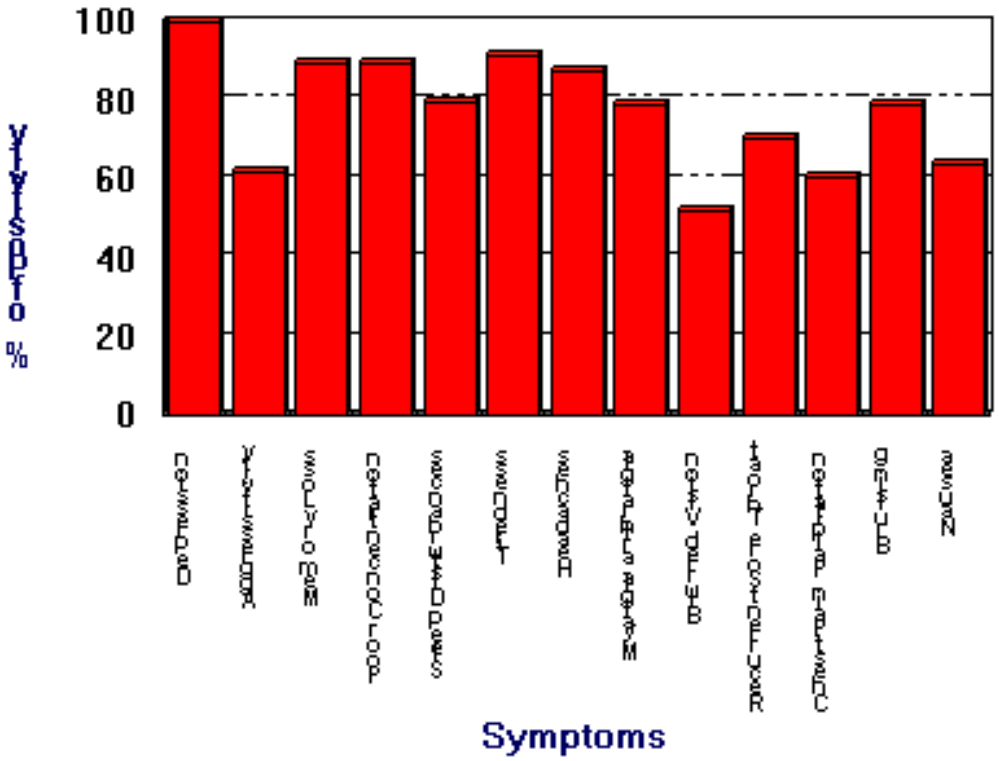
§Positive reaction = presence of antibodies;
(does **not** necessarily mean illness)

§Negative reaction does **not** suppress Rickettsial etiology (**R1,25**)

Patients and Methods: **Statistics of 300 patients (100% Caucasian)**

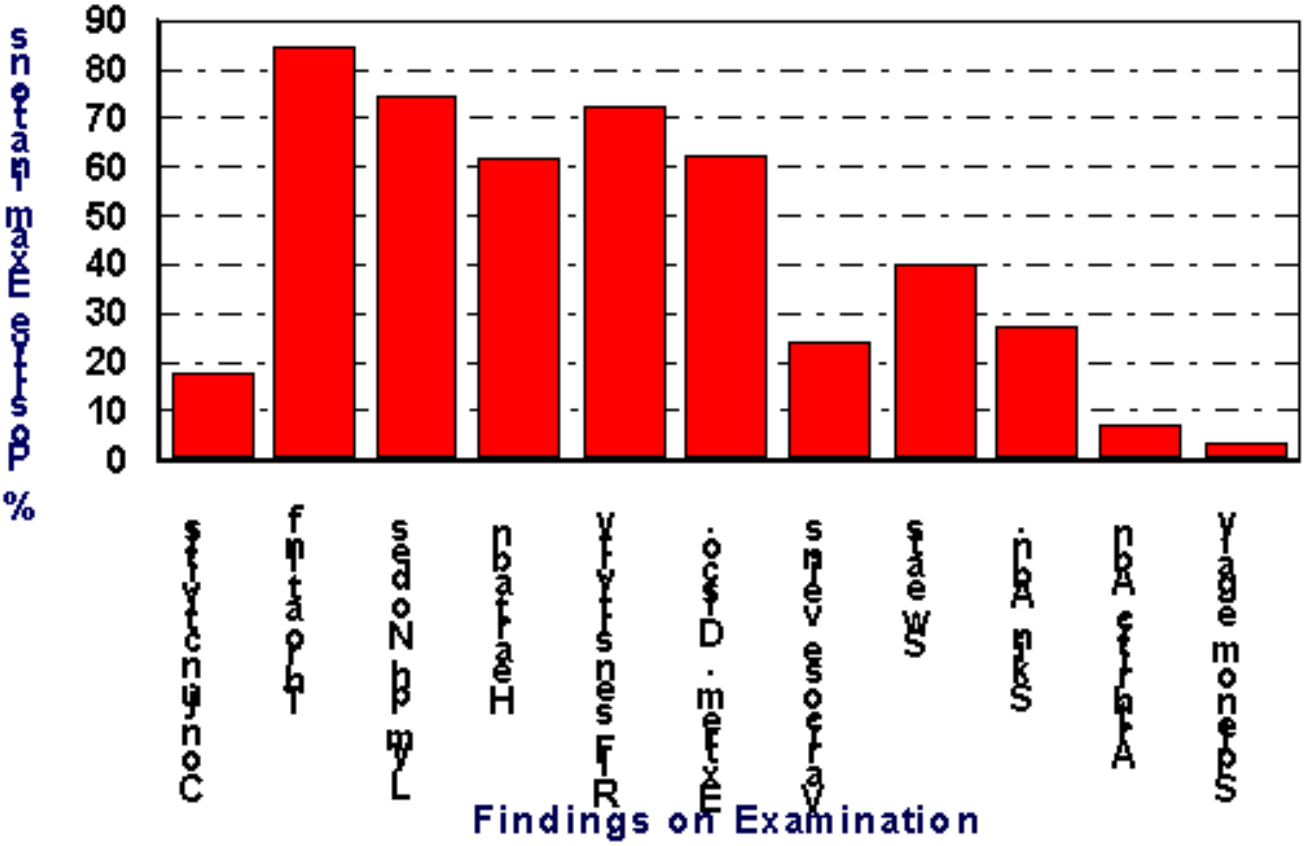
Selection Criteria: first dsed as Depression and Psychotic D

Sex		**Age Group**	**Length of Illness:**	
Male:	**127**	**12 - 69**	**Minimum**	**6 months**
Female:	**173**		**Maximum**	**20 years**
			1 exc . 3 months	



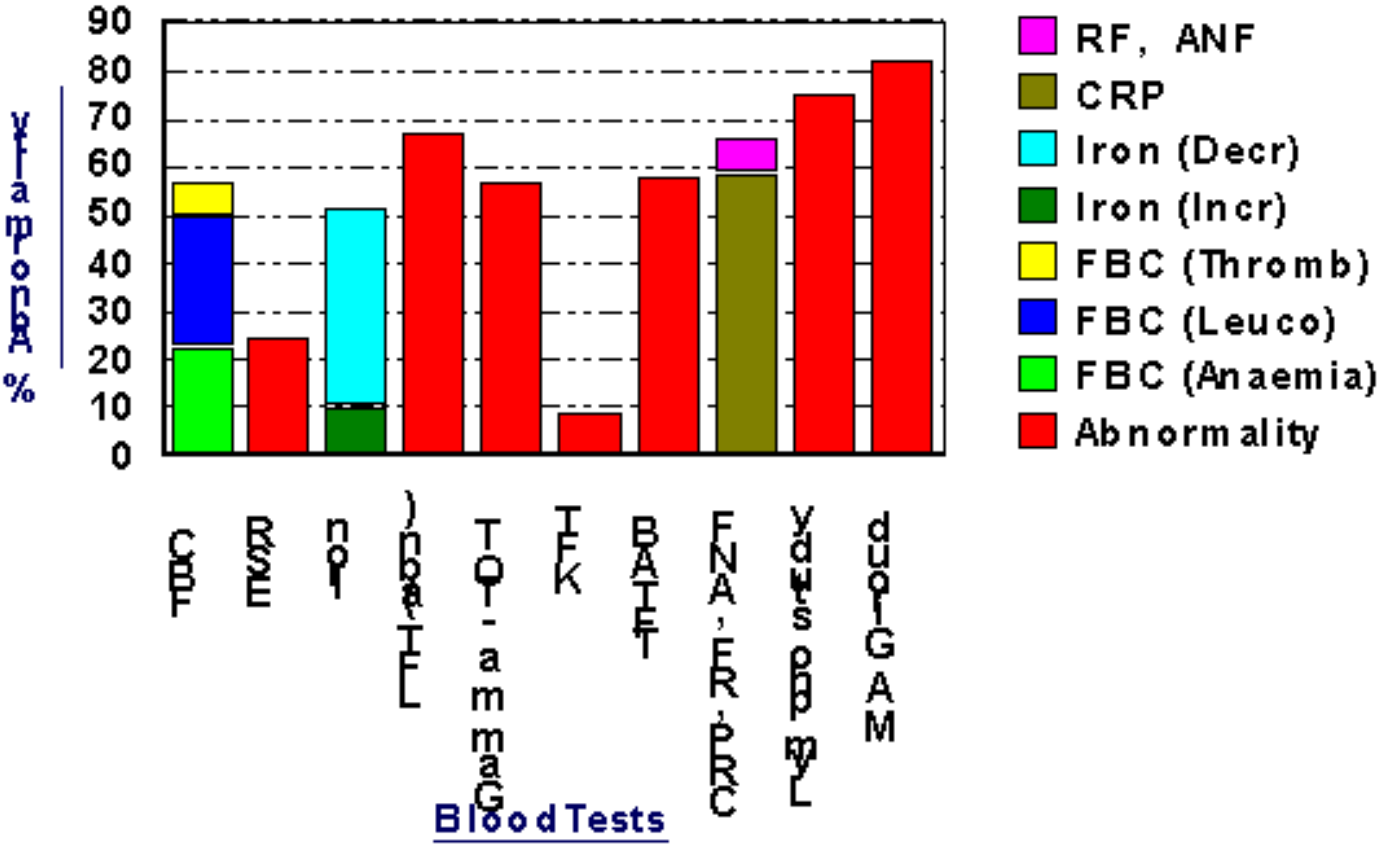
I. **Main Symptoms:**

II.



Clinical Examination

III. Biological Investigations



NB: The difference between Poster 1 and Poster 2 results was so small that I took the liberty of showing the same chart

IV. CXR - MRI (Brain Scan done on 62% of cases = NAD) - Joints X Ray

V. Treatment: average of 7 days/month of Tetracyclines

1.alternated
2. combined with Quinolones, Macrolides, Metronidazole
3. high dosage
4. varying in length (fast response, slow response)
5. Anti malaria

6. Adjuvants
7. Exercise (Rickettsia has vascular impact)

VI. Herxheimer reaction: Prognosis value (**R1**: P734 and P751; **R11**, **R18**:P437; **R47**)

VII. Results: 300 patients found to be suffering from depression and or psychotic dysfunction were treated with antibiotics in a regime designed to control Rickettsial infection. The success rate for the treated individuals was 92%. 10 individual cases are presented below.

VIII. Detailed Review of 10 cases

1. Male - 12 years old; 3 months illness. Returning from camp and developed encephalitis, hospitalised 1 week, came home with severe aggressitivity and depression; 1st diagnosed as psychopath by 3 different psychiatrists. He was treated with Tofranil, Melleril, Aterax. and was about to be admitted to a psychiatric hospital. 2nd diagnosis: CRI. He was given 3 treatments of Tetracyclines. He has stopped all the other medication, he became a school prefect in November 94 and his condition is maintained to present day. Liver function tests previously tested abnormal, now test normal.

2. Female - 15 years old (the sister of the above patient), returning from same camp as her brother with Flu, developed epilepsy., and was treated with Tegretol. 2nd diagnosis CRI. She was treated for 5 months with Tetracycline. She stopped taking Tegretol in November 94 and has had no relapse as of today.

3. Male - 56 years old. A psychiatrist by profession. Illness of 4 years. Diagnosed as endogenic depression. Recovered completely after 8 treatments. Subsequently treated 300 of his own patients with the same antibiotherapy very successfully (**R28)**.

4. Male - 52 years old. Illness since childhood. 1st diagnosed as having endogenic depression. Treated with various antidepressants without satisfactory results. 2nd diagnosis: - LFT D. (CRP increased, MRI normal). Given 15 treatments of Tetracyclines. He reported that tetracycline was his "best antidepressant". (LFT normalised and CRP reduced). Sadly, this patient committed suicide (reasons unknown).

5. Male - 36 year old, farmer. Illness of 3 years. Diagnosed as acute psychosis; treated with antidepressant, and given shock therapy on alternative months. 2nd diagnosis: CRI. (LFT D, iron increased, MRI normal). He was given 8 treatments of Tetracyclines. Good improvement was observed after the 1st treatment; and he was asymptomatic after 8 treatments. No other drugs are now required.

6. Female - 26 years old. Illness of 20 years. Originally diagnosed as endogenic depression. Treated with Aropax twice daily, Prozac 4 times a day, pain killers 8 times a day for +/- 10 years. Had many magnesium drips, sleeping therapy. Was very depressed, suicidal, (She wrote off 5 cars!) had aggressive

behaviour towards her family, exhaustion and headaches. 2nd diagnosis: CRI: - (thyroid ab raised, CRP raised), currently on 5th treatment of Tetracyclines. Vast improvement after 1st treatment; stopped Prozac, Aropax, and painkillers. Coping well.

7. Female - 59 years old. Illness of 20 years. After acute tick bite fever, developed depression and treated with antidepressants, and gamma globulines. 2nd diagnosis: Rheumatoid Arthritis due to CRI. (RF 236, RW +ve. ANF +ve.) She was given 15 treatments of Tetracyclines. (She had a violent Herxheimer reaction after the 1st treatment; requiring hospitalisation of 2 days). She is now well improved, and is no longer taking antidepressants. (RF 194, RW normal ANF normal).

8. Female - a 44 year old. Nursing Sister. Illness of 5 years. 1st diagnosis: Endogenic depression. Treatments given were: various antidepressants, shock therapy, sleeping therapy. 2nd diagnosis: CRI. She was given 10 Tetracyline treatments. She is now very well recovered, she is no longer taking antidepressants, and she is back at work, working night shift!

9. Female - 40 years old. Diagnosed 5 years ago as maniac-depressive, and was treated with Lithium, Zoloft and Ativan for last 2 years. She was also given electroshock therapy 7 times. She has had duodenal ulcers since the age of 12. She had an appendicectomy at age 28. Suicidal. 2nd diagnosis: CRI. Put on course of 8 treatments of Tetracyclines. Well recovered. Stopped Lithium after 4 treatments, Zoloft after 2 treatments, still takes Ativan occasionally. She is now back at work (previously, she was hardly able to walk 20 metres).

10. Male - 49 yearsold. Professor of Botany at SA university. Suicidal, exhaustion, myalgia, arthralgia, hypertensive. Diagnosed as chronic depression for 15 years; refused antidepressants. 2nd diagnosis: CRI, liver abnormality and CRP increased. Put on 7 treatments of Tetracycline for 8 months. (1991 - 1992). Remarkable improvement after the 2nd treatment. 4 treatments in 1993. 3 treatments in 1994. 3 treatments in 1995. 2 in 1996, 3 in 1997. This continuation is due to his work risk factor (field work, in the South African bush).

-

IX. **Conclusion:** When confronted with psychiatric disorders, we should always look for associated pathogens such as:

- Rheumatoid disorders
- Liver disorders
- Peripheral angiopathy
- Iron disorders
- Thyroid antibodies

to help us to orientate a diagnosis and treatment, because as shown by the study there may be a causative

link between depression and an infectious agent.

CFS - Rickettsial Infection - Case Studies by Dr. Cécile Jadin, South Africa

Summary: Since January 1991, over 3.000 patients, previously diagnosed as ME, CFS, psychopathic, fibromyalgia, arthritic diseases, or unknown, were treated with Antibiotherapy. The reason for this treatment was as follows:

q The symptoms of these patients were similar to those exhibited in chronic Rickettsial diseases.

q The treatment followed the finding that their serum reacted positively to the **Giroud** micro- agglutination test.

Giroud Test - specific for testing antibodies
to these 5 antigens (**R36**):

§ Rickettsia Prowazeki
§ R. Mooseri
§ R. Conori
§ Coxiella Burnetti
§ Neo-R. Q18 (**R50**)

Done by micro agglutination

Depends on the quality of antigens
Comparative studies with IFA test gave very similar result

§ Positive reaction = presence of antibodies;
(does **not** necessarily mean illness)

§ Negative reaction does **not** suppress Rickettsial etiology (**R1,25**)

BECAUSE

1. The occult infection is biologically similar to the disease itself (Ch. Nicolle) (**R2, R34**)

2. Walker and Jadin described positivity for Rickettsiae on people without any symptoms (26% according to Walker) (**R3, R9, R25, R45**)

3. If doubtful and if negative, the test should be repeated to follow the antibody curve (**R37**)

4. Same applies for many pathogenic agents e.g. coxsackies (**R38**), chlamydiae pneumoniae (**R39**) etc.

THEREFORE

the diagnosis of Rickettsial disease stands on 3 corner stones:

Symptoms **Clinical findings** **Biological investigations**

Patients and Methods: **Statistics of 500 patients (100% Caucasian)**

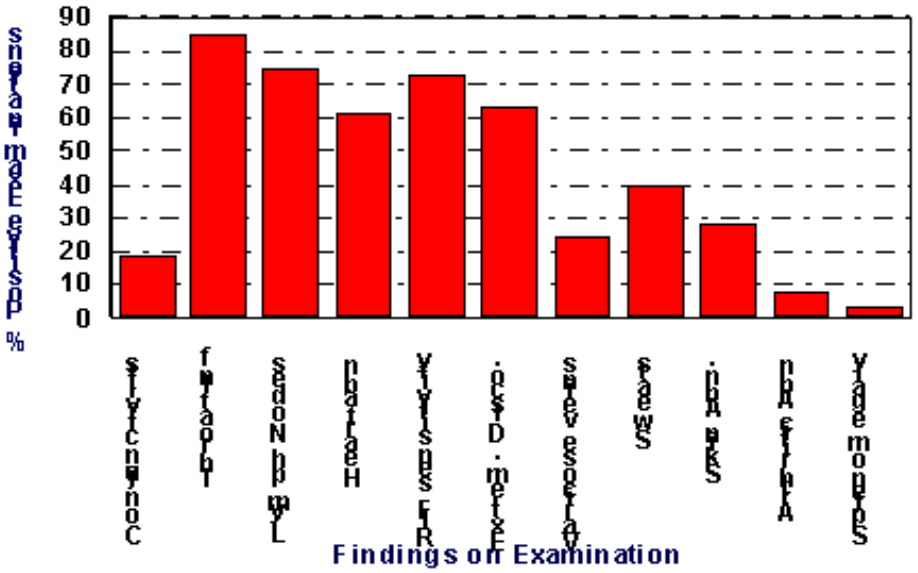
Criteria for selection: first dsed as ME, CFS, or Depression

Sex		**AgeGroup**		**Length of Illness:**	
Male:	**236**	**<10:**	**2.1%**	**< 1 year:**	**12.5%**
Female:	**264**	**10 - 20:**	**16.7%**	**1 - 2 years:**	**20.7%**
		20 - 40:	**43.7%**	**2 - 5 years:**	**29.3%**
		> 40:	**37.5%**	**> 5 years:**	**37.5%**

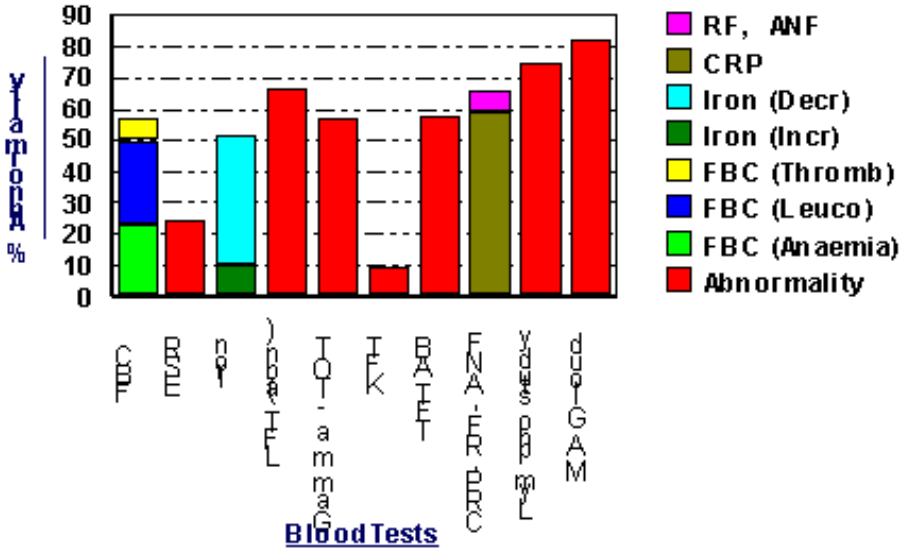
Exposure to Rickettsia:	**Known:**	**95%**
	Unknown:	**5%**

I. Rickettsia infection symptoms and diseases according to tissue type:

Vascular Tissue			**Reticulo Endothial Tissue**	
Tiredness (**R1,4,7,9,13,28**)	92%		Recurrent sore throat (**R1**)	85%
Myalgia Arthralgia (**R5**)	90%		Appendicitis (**R5,8**)	65%
Cardiac. Valves D (**R1,6,7,8,9**)	90%		Lymphadenopathy (**R1**)	73%
Memory. Concentration (**R9**)	89%		Systemic Candidosis	53%
Headaches (**R1,9**)	88%			
Bruising (**R26**)	81%		**Neurological Tissues**	
Vision - Uveitis Conj. (**R1**)	39%		Encephalitis (**R1,11,13,17)**	12%
Dermatological (**R9,10)**	23%		Epilepsy (**R1,13,24)**	69%
Psychotic disorder treated (**R9**)	69%		MS (**R4,9,24,37)**	5%
Atypical Hepatitis (nausea-vom.) (**R1,11,12,27,31**)	64%		Loss of Balance	28%
Raynaud Syndrome (**R13**)	69%			
Pulmonary Disease (**R6,8,14,31**)	21%			
HBP	9%			



II. Clinical Examination



III. Biological Investigations

IV. CXR - MRI (Brain Scan) - Joint X-Ray

V. **Treatment:** average of 7 days/month of Tetracyclines
1.alternated (**R6,9,11,28**)
2. combined with Quinolones, Macrolides, Metronidazoles (**R6,43**)
3. high dosage (**R1,9,11,13,43**)
4. varying in length (fast response, slow response) (**R1,9,13,16,25,43,45,48**)
5. Anti malaria
6. Adjuvants
7. Exercise (Rickettsia has vascular impact) and hot baths (**R9**)

VI. **Herxheimer reaction**: Prognosis value (**R1,11,18,44,47**)

VII. **Results - Statistics:**

Including patients not treated, or treatment not completed

358 patients very well, or cured	**72%**
101 patients stopped consultation after 1 to 3 treatments	**20%**
26 patients never commenced treatment	**5%**
15 patients showed no improvement to treatment	**3%**
Excluding patients not treated or treatment not completed	
:358 patients very well, or cured	**96%**
101 patients stopped consultation after 1 to 3 treatments	**4%**

NB:

% of reactivation or reinfection of patients well improved: 7%
- , but patients recover after further treatment (2 to 3 months average) (R25,26)

VIII. This **Assessment** is performed monthly to judge patients progress towards "**very well"** : symptoms meaurement

increase in activity

symptomatic medication independence (pain killers, anti-depressants, hypnotics, cortisone)

medical examination

biological investigation.

Based on this, the treatment is prolonged or stopped (3 months to 2 years - 8 months average).
However, length of treatment is not directly correlated to the length of illness.

Patients can be divided into 2 categories:
1. Fast progress - their illness was mainly Rickettsia

2. Slow progress - their illness was Rickettsia plus other factors (**R36**)

IX. **Followup** - was done by means of research questionnaire sent to patients on or off treatment every 6 months for 2 years (1992-1993). 78% answered, and of those, 93% were still "very well".

X. **Discussion:**

1. CFS in Nevada (**R40**) - Rocky Mountain fever (**R7,8**)

2. CFS - Nightingale disease (**R33**)

3. Lymphocyte study results similar in sheep with tick-borne diseases and in patients with CFS (**R19,21,22,23**) and

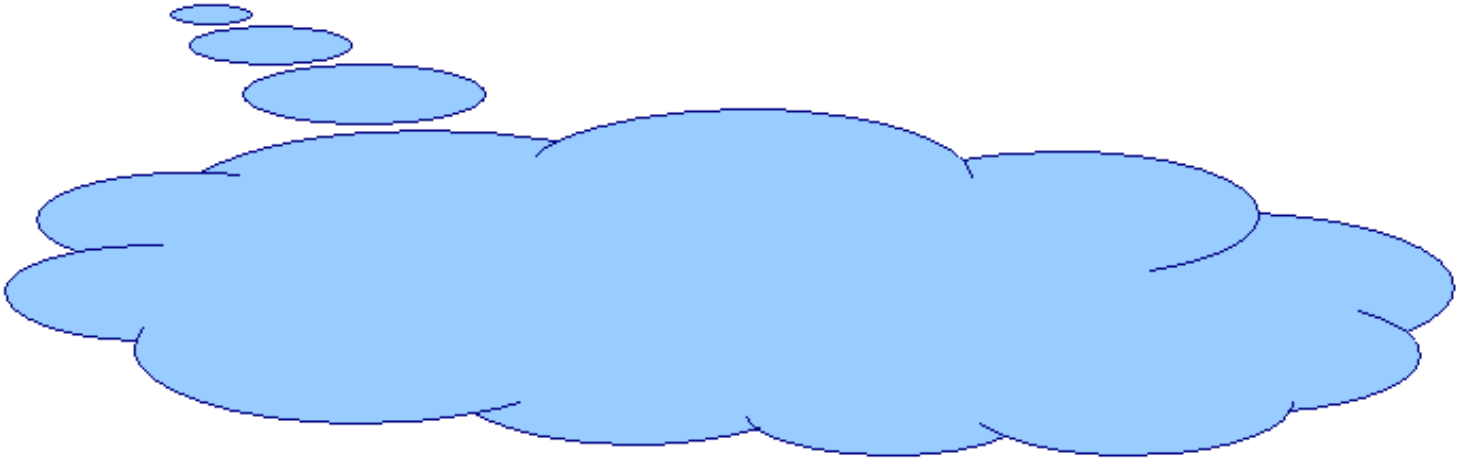
patients with Q Fever endocarditis (**R48**)

4. CFS - PQFS (**R4**)

5. Rickettsial Disease through History (**R7,8**)

6. Treatment rates success (**R28**)

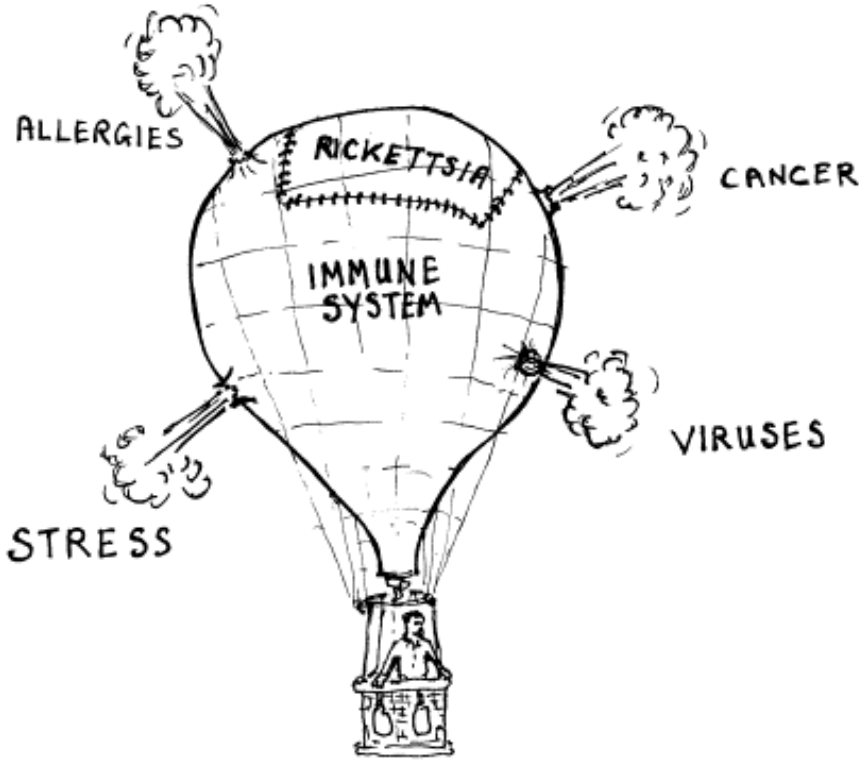
7.



Interaction between protozoa, viruses, bacteria, and stress? (**R9,29,34,36,42**)

8.

Rickettsia



is a suggested way
to repair an Immune System,
quickly or slowly.

"La santé est comme une mongolfière: il faut parfois lâcher du lest"

Dr. Cecile Jadin's Papers are now available in full Click Here

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National CFIDS Foundation

International CFS Information

Updated on:
12/17/1999

Physicians on this page have been deemed by their patients to accept CFS as a real illness and treat CFS patients with respect. These are NOT specialists - simply GP's that are CFS friendly!

WASHINGTON

Kingston - Dr. S. Shliffer

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National CFIDS Foundation

International CFS Information

Updated on: 02/17/2000

Smart Drug

Like Piracetam, Hydergine (Co-dergocrine, Ergoloid Mesylates) is classified as a "smart drug" (and thus may help *some* CFIDSer's brain fog). FDA has approved the use of Hydergine for only dementia and related circulatory problems. A prescription is required.

- appears to be an anticoagulant (5 studies (66-73), no abstracts on NML)
- increases blood supply to the brain
- increases the amount of oxygen delivered to the brain
- enhances metabolism in brain cells
- protects the brain from damage during periods of decreased and/or insufficient oxygen supply

Technical Descriptions

- http://www-nmcp.med.navy.mil/pharmacy/formulary/ergoloidmesylates.htm
- http://www.healthsquare.com/pdrfg/pd/monos/hydergin.htm
- http://www.rxmed.com/monographs/hyderg.html [Warnings!]
- http://www.lef.org/prod_hp/abstracts/hydergine.html

Notes:

Dosage is reported (not published) as:

- Europe the typical dose is 9 milligrams a day
- U.S. is 3 milligrams a day (for approved uses [*])

Dr. Cecile Jadin's Papers are now available in full Click Here

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International CFS Information

Updated on:
01/01/2000

The following are my own observations and memories of my experience with CFIDS (on the 2nd round!)

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International CFS Information

Updated on: 12/01/1999

There is still debate in the medical profession on the relationship between Mycoplasma and asthma. Despite the debate, there have been consistent reports of marked improvement with anti-mycoplasma antibiotics (INFECTIONS, ASTHMA AND COPD).

- Asthma and mycoplasma pneumonia
- *Bacteria's Role in Asthma* .. *""This suggests that in humans, mycoplasma may also interact with allergens to produce asthma,"*

"Short term treatment with antibiotics is ineffective, but taking doxycycline (100 mg BID) or azithromycin (1 gram once a week) for at least 6 months has been shown in at least one paper to cure asthma (31)." http://www.wdn.com/mirkin/7165.html

The high success rate of the Buteyko method (see breathing) for asthma may be ascribed to a raised oxygen level killing off mycoplasma.

Editor Note: A critical point is the debate has been not finding a mycoplasma infection in some patients. This *may* be a false argument if the tests fails to detect all mycoplasma...

Depression, Psychotic Dysfunction, Rickettsial Infection Case Study

by

Dr. Cécile Jadin, Randburg, South Africa

Summary: The possibility of a Rickettsial origin for symptoms of depression and psychotic disfunction has been suggested by French scientists (Ch. Nicolle, Giroud, Legag, Jadin, Bottero) in their published works. Hence 300 patients, diagnosed as suffering from depression, or other neuropsychiatric dysfunction have been treated with antibiotic where a positive indicaton of Rickettsial infection was revealed as follows:

1. Many symptoms of these patients were similar to those exhibited in chronic Rickettsial diseases.

2. The treatment followed the finding that their serum reacted positively to the **Giroud** micro-agglutination test.

Giroud Test - specific for testing antibodies to these 5 antigens (**R36**):

§ Rickettsia Prowazeki
§ R. Mooseri
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Done by micro agglutination

Depends on the quality of antigens
Comparative studies with IFA test gave very similar result

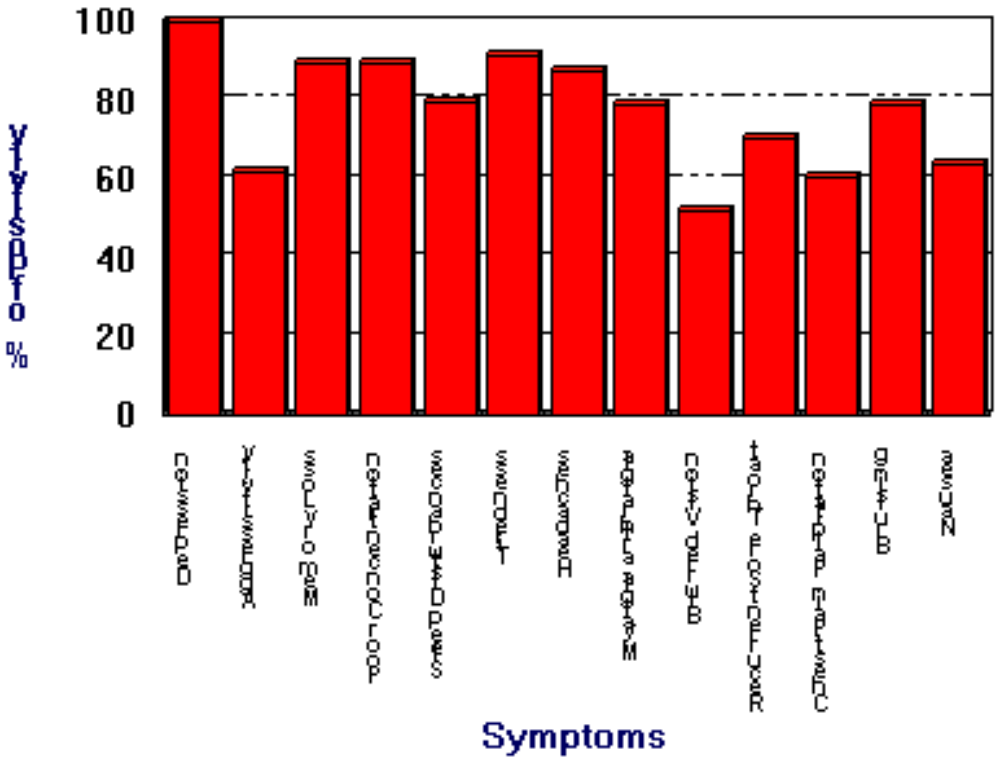
§Positive reaction = presence of antibodies;
(does **not** necessarily mean illness)

§Negative reaction does **not** suppress Rickettsial etiology (**R1,25**)

Patients and Methods: **Statistics of 300 patients (100% Caucasian)**

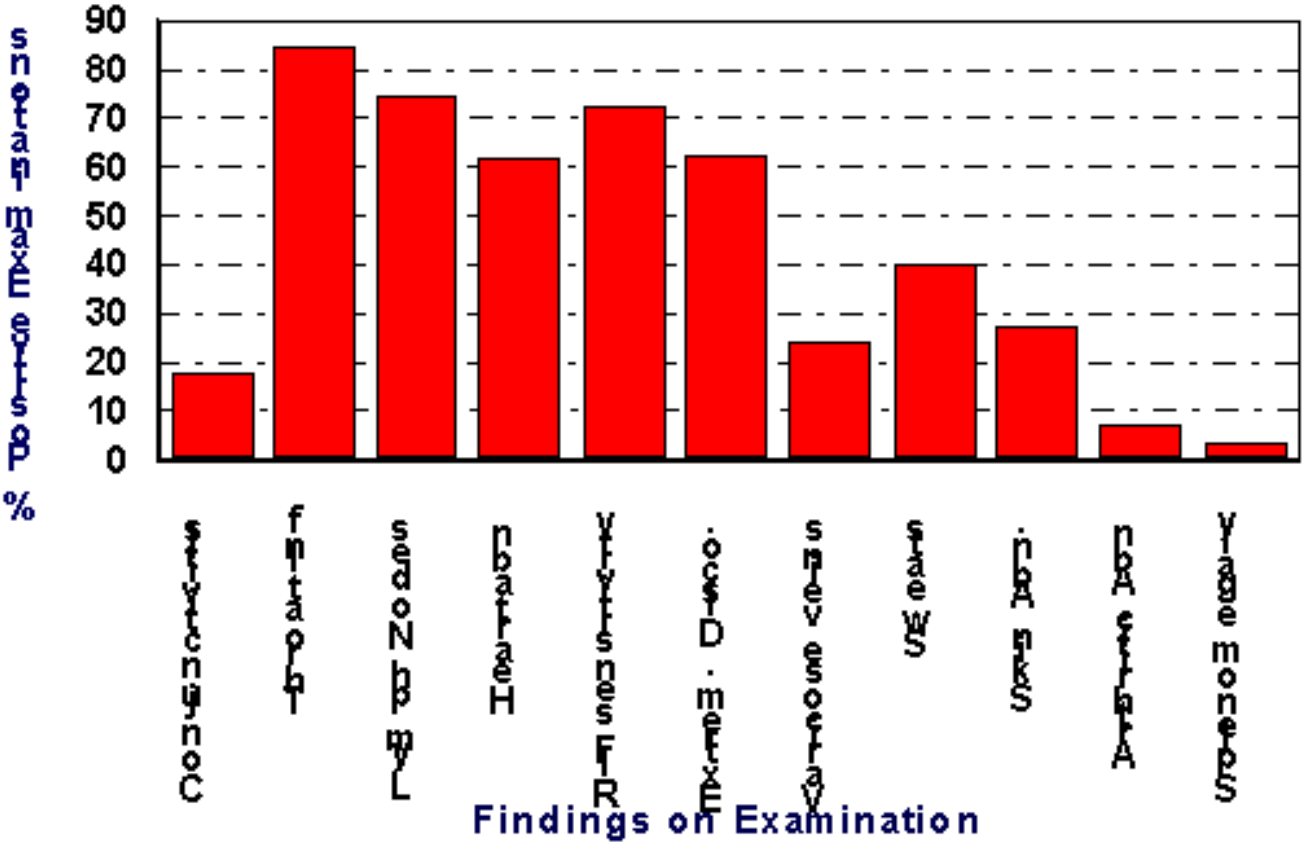
Selection Criteria: first dsed as Depression and Psychotic D

Sex		**Age Group**	**Length of Illness:**	
Male:	**127**	**12 - 69**	**Minimum**	**6 months**
Female:	**173**		**Maximum**	**20 years**
			1 exc . 3 months	



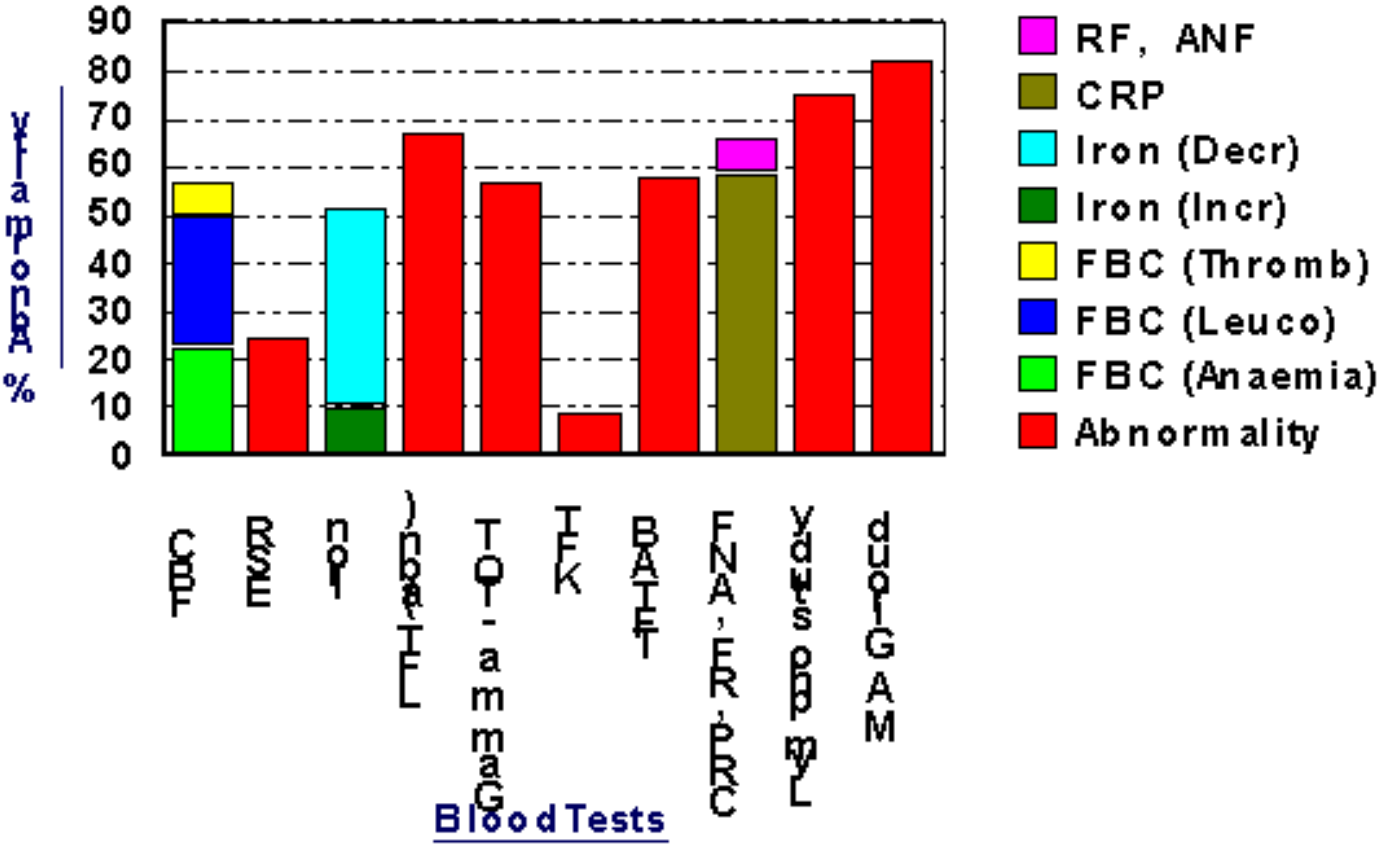
I. **Main Symptoms:**

II.



Clinical Examination

III. Biological Investigations



NB: The difference between Poster 1 and Poster 2 results was so small that I took the liberty of showing the same chart

IV. CXR - MRI (Brain Scan done on 62% of cases = NAD) - Joints X Ray

V. Treatment: average of 7 days/month of Tetracyclines

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3. high dosage
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5. Anti malaria

6. Adjuvants
7. Exercise (Rickettsia has vascular impact)

VI. Herxheimer reaction: Prognosis value (**R1**: P734 and P751; **R11**, **R18**:P437; **R47**)

VII. Results: 300 patients found to be suffering from depression and or psychotic dysfunction were treated with antibiotics in a regime designed to control Rickettsial infection. The success rate for the treated individuals was 92%. 10 individual cases are presented below.

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CFS for Dummies

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National CFIDS Foundation

International CFS Information

Updated on:
11/28/1999

 This website is intended to be a 'Rolodex' of summaries for CFS patients and URLS for their MDs. Documents are for informational purpose only, based on web surfing, and not written by a medical professional. Issues have often been ***simplified*** for easier understanding by CFS patient (commonly having Brain Fog / 40-60 IQ point drop) and may not be 'technically accurate'.

CFS - Rickettsial Infection - Paper presenting the results of 5 years of diagnoses and therapy

Presented by Dr. Cécile Jadin

Johannesburg, South Africa

February 1998

Summary: Since January 1991, over 3.000 patients, previously diagnosed as ME, CFS, psychopathic, fibromyalgia, arthritic diseases, or unknown, were treated with Antibiotherapy. The reason for this treatment was as follows:

q The symptoms of these patients were similar to those exhibited in chronic Rickettsial diseases.

q The treatment followed the finding that their serum reacted positively to the **Giroud** micro- agglutination test.

The **Giroud Test** is specific for testing antibodies to the following 5 antigens (**R36**). According to Giroud, all those different antigens have the same behaviour and he insists that they may change depending on their host. (**R50**)

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§ R. Mooseri
§ R. Conori
§ Coxiella Burnetti
§ Neo-R. Q18 (**R50**)

Done by micro agglutination

Depends on the quality of antigens

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Symptoms **Clinical findings** **Biological investigations**

Patients and Methods: **Statistics of 500 patients The Criteria used for selection was that the patients were first diagnosed as ME, CFS, or Depression.**

Sex		**AgeGroup**		**Length of Illness:**	
Male:	**236**	**<10:**	**2.1%**	**< 1 year:**	**12.5%**
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		20 - 40:	**43.7%**	**2 - 5 years:**	**29.3%**
		> 40:	**37.5%**	**> 5 years:**	**37.5%**

Exposure to Rickettsia:	**Known:**	**95%**
	Unknown:	**5%**

I. The following table displays the percentages of symptoms and diseases caused by Rickettsial infection in the above group of patients. The symptoms and diseases vary according to the main localisation of the infection.This is based on clinical findings and also described in literature.

Vascular Tissue	
Tiredness (**R1,4,7,9,13,28**)	92%
Myalgia Arthralgia (**R5**)	90%
Cardiac. Valves D (**R1,6,7,8,9**)	90%
Memory. Concentration (**R9**)	89%
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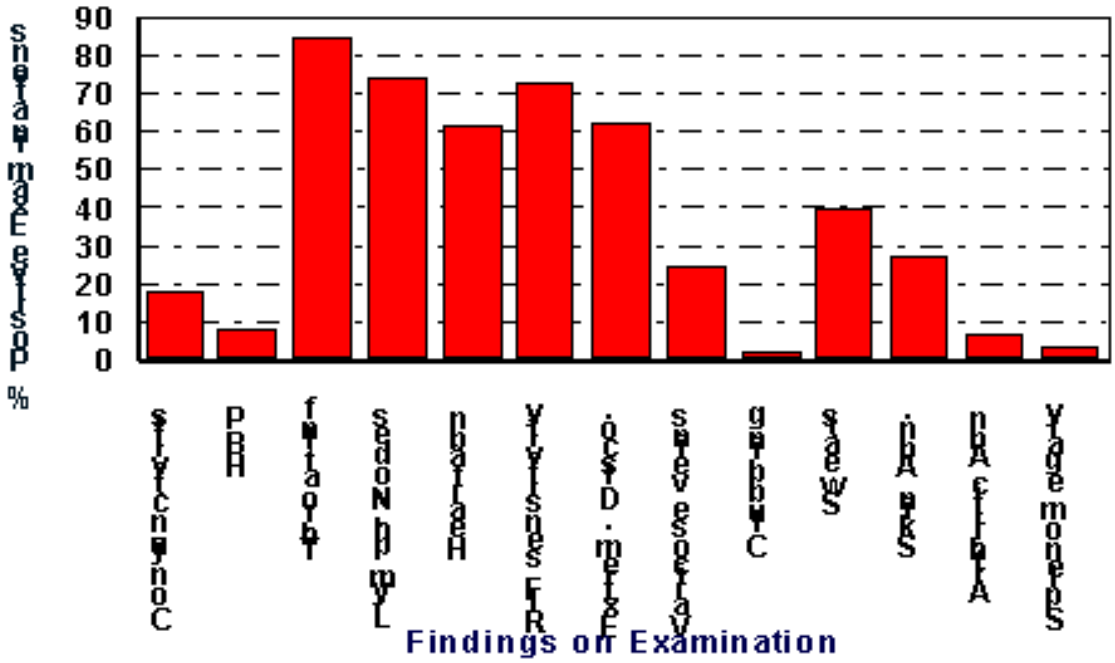
Bruising (**R26**)	81%
Vision - Uveitis Conj. (**R1**)	39%
Dermatological (**R9,10**)	23%
Psychotic disorder treated (**R9**)	69%
Atypical Hepatitis (nausea-vom.) (**R1,11,12,27,31**)	64%
Raynaud Syndrome (**R13**)	69%
Pulmonary Disease (**R6,8,14,31**)	21%
HBP	9%

Reticulo EndothialTissue	
Recurrent sore throat (**R1**)	85%
Appendicitis (**R5,8**)	65%
Lymphadenopathy (**R1**)	73%
Systemic Candidosis	53%

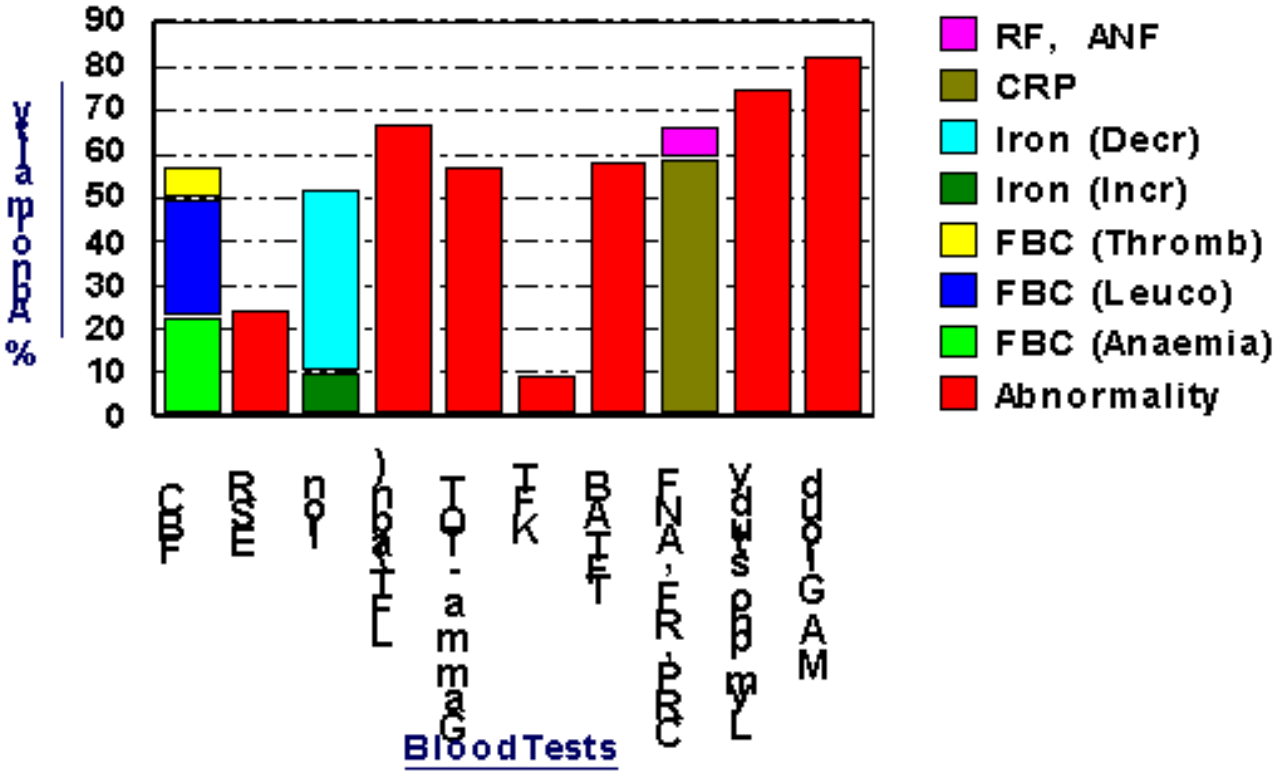
NeurologicalTissues	
Encephalitis (**R1,11,13,17**)	12%
Epilepsy (**R1,13,24**)	69%
MS (**R4,9,24,37**)	5%
Loss of Balance	28%



II. Clinical Examination The following graphic shows that most patients display the same clinical findings.

III. Biological Investigations

This graphic highlights similar blood disorders in the same target group. These are investigated in order to orientate the diagnosis and eliminate other pathologies.



IV. CXR - MRI (Brain Scan) - Joint X-Ray

Chest Xrays are routinely done to eliminate diagnosis of TB, cancers etc. The brain scan is performed to eliminate the possibility of tumours, MS, or other pathology. The joint X-ray is only required if strong clinical findings exist.

V. Treatment: consists of 7 to 12 days /month of of Tetracyclines

1. The Tetracyclines should be **alternated** because

a) A patient is often contaminated by many Rickettsiae (**R9**) and different Rickettsiae have different sensitivity to different Tetracyclines.(**R6**, **R11**)

b) A patient might build resistance to each Tetracycline(J.Jadin) (**R28**)

c) Patients show individual sensitivity to different Tetracyclines and there is very often a better reaction to a particular antiobiotic. (**R1**)

2. Tetracyclines should be **combined** with Quinolones, Macrolides, Metronidazoles because Rickettsiae present a wide heterogeneity of susceptibility to different drugs (**R6, R43**)

3. A **high dosage** of Tetracyclines is required to avoid formation of occult focci, although keeping within the limits of tolerance and safety (**R1, R9, R11, R13, R43**):

· Gastric intolerance will be successfully prevented by using a gastric proton pump inhibitor during the administration of the Tetracyclines

· No liver toxicity was found in any patient. Our experience was that if the liver function tests were normal at the start, then they stayed the same. If the LFT were bad, they generally would improve during treatment. This confirms the fact that Rickettsiae are hepatotoxic (**R1, R5, R7 &8, R11,R12,R46, R50**)

4. The treatment is often **long or prolonged** due to

· The chronicity of the germ

· The multiple focci of Rickettsiae

· The fact that Rickettsiae are slow germs; some focci are dormant and will only be controlled by the treatment when they become active. (**R9**)

· Each treatment will allow the immune system to produce and maintain a proper and efficient level of antibodies; this takes place each time the Rickekettsiae antigens are released from the cell to the blood circulation while on antibiotherapy (**R1, R9**)

· Although this research does not show that the length of illness is directly correlated to the length of treatment, this has often been described to be the case (**R1, R9, R13, R16, R25, R43, R45, R48**):

5. Anti malaria has been found efficient in improving rheumatoid symptoms and rheumatoid biological findings

6. Adjuvants like Vit. B co, acidobacillus, gastric proton pump inhibitor are recommended

7. Exercise is recommended keeping in mind that Rickettsia has a vascular impact. Hot baths are important to eliminate toxins produced by Rickettsiae antigens liberated in blood circulation when patients are on AB

VI. Herxheimer reaction (R1, R11, R18, R47): is a reactivation of old symptoms and/or exacerbation of present symptoms that occurs on antibiotherapy. Its presence has a very important diagnosis and prognosis value. It may or not be parallel to a serological reactivation. It often fades with the number of treatments received. When very severe, the HR is treated with probenecid.

VII. Results Of 500 patient records analysed:

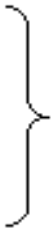
358 patients very well, or cured
101 patients stopped consultation after 1 to 3 treatments
26 patients never commenced treatment
15 patients showed no improvement to treatment

NB:

Reactivation or reinfection of patients: (R1, R11, R18, R44) called so rather than relapse, may occur (2 - 7%). This may be due to an interaction between Rickettsiae and viruses or bacteria or any other parasite: from a dormant (asymptomatic) form, Rickettsia can be reactivated to an acute, subacute or chronic condition with the help of other agents. An example of this change of condition was the infant mortality rate described by J. Jadin in Central Africa. The children identified and having malaria and Coxiella Burnetti all died as opposed to those with only a malarial condition (**R49**). If reaction or reinfection occurs, 1 to 3 treatments on average will be necessary if the condition is recognised quickly.

VIII. This **Assessment** is performed monthly to judge patients progress towards "**very well"** :

1. Symptoms
2. Activities increase eg. From bedridden to back to exercise or back to work
3. From being treated by painkillers, antidepressants, sedatives, cortisone to none
4. Medical examination
5. Biological investigation: from having:



· LFT D

· RF -, CRP -, ANF -

· KFT - To normal or nearly so

· Thyroid antibodies -

· Iron D

Based on the assessment, the treatment is prolonged or stopped (3 months to 2 years - 8 months average). However, as previously mentioned, the length of treatment is not directly correlated to the length of illness:

Patients can be divided into 2 categories:
1. Fast progress - their illness was mainly Rickettsia

2. Slow progress - their illness was Rickettsia plus other factors (**R36**)

IX. Followup - was done by means of research questionnaires sent to patients on or off treatment every 6 months for 2 years (1992-1993). 78% answered, and of those, 93% were still "very well".

X. Discussion:

1. CFS was first reported in the village of Incline in Nevada in 1984 (**R40**) and developed into epidemic proportions. That is also where Rocky Mountain Spotted Fever originated. (**R7**)

2. The proposition to change CFS into Nightingale disease, (because of the tiredness Florence experienced for the last 2 decades of her life, working with soldiers infested with lice and fleas during the Crimean war) could reinforce the epidemiological hypothesis. (**R33**)

3. The T-Lymphocyte study (CD4, CD5, CD8) done in sheep experimentally infected with tick-borne disease described by the University of Liverpool (**R20**) is amazingly similar to the Lymphocyte study done on CFS patients (**R19,21,22,23**) and also on patients with Q Fever endocarditis (**R48**).

4. In April 1996, in a letter published in the Lancet, the suggestion was made to change CFS into PQFS (Post Q Fever Syndrome) (**R4)**

5. Rickettsia has been described throughout history as causing the defeat of Hannibal, Caesar and Napoleon. (**R41**) It also has been accused of causing the death of 3 million people in the 1st World War.and in the Nazi Concentration camps. How, suddenly, is it not around any more?

6. The fact that the treatment with Tetracycline has been successful on an extended number of patients (more than 3000) previously diagnosed as CFS, Fibromyalgia, Depression, should be given attention (**R28**).

7. The CFS - Rickettsial disease could simply be another example of the relationship between protozoae, viruses, bacteria, and stress. Is there a possibility of an immuno deficiency created by viruses or parasites or bacteria that would bring the Rickettsiae from dormant to acute, subacute or chronic?. (**R9,34,36,42**)

8.

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National CFIDS Foundation

International CFS Information

Updated on: 01/18/2000

JHK Retrovirus

- "JHK virions measure 85 nm in ultrathin sections, ***much smaller*** than other Retroviridae." [*]
- "The virus, designated JHKV, is an enveloped, relatively fragile particle containing RNA, reverse transcriptase activity, and prominent, knobbed, peplomers (trans-envelope proteins)".[* has electron microscope photo]
- Resembles C-type retroviruses [*]

C-type retrovirus

- Examples: Leukemia (cancers of the bone marrow: prevents the normal manufacture of red and white blood cells and platelets. Results in anemia, increased susceptibility to infection, and impaired blood clotting): gibbon ape leukemia virus [*]
- Found in HIV blood cells [*]

CFS - Rickettsial Infection - Paper presenting the results of 5 years of diagnoses and therapy

Presented by © Dr. Cécile Jadin

Johannesburg, South Africa

February 1998

Summary: Since January 1991, over 3.000 patients, previously diagnosed as ME, CFS, psychopathic, fibromyalgia, arthritic diseases, or unknown, were treated with Antibiotherapy. The reason for this treatment was as follows:

- The symptoms of these patients were similar to those exhibited in chronic Rickettsial diseases.
- The treatment followed the finding that their serum reacted positively to the **Giroud** micro- agglutination test.

The **Giroud Test** is specific for testing antibodies to the following 5 antigens (**R36**). According to Giroud, all those different antigens have the same behaviour and he insists that they may change depending on their host. (**R50**)

- Rickettsia Prowazeki
- R. Mooseri
- R. Conori
- Coxiella Burnetti
- Neo-R. Q18 (**R50**)

Done by micro agglutination
Depends on the quality of antigens
Comparative studies with IFA test gave very similar result

- Positive reaction = presence of antibodies; (does **not** necessarily mean illness)
- Negative reaction does **not** suppress Rickettsial etiology (**R1,25**)

BECAUSE

1. The occult infection is biologically similar to the disease itself (Ch. Nicolle) (**R2, R34**)
2. Walker and Jadin described positivity for Rickettsiae on people without any symptoms (26% according to Walker) (**R3, R9, R25 R45**)
3. If doubtful and if negative, the test should be repeated to follow the antibody curve (**R37**)
4. Same applies for many pathogenic agents e.g. coxsackies (**R38**), chlamydiae pneumoniae (**R39**) etc.

THEREFORE

the diagnosis of Rickettsial disease stands on 3 corner stones:

Symptoms	Clinical findings	Biological investigations

Patients and Methods: **Statistics of 500 patients** **The Criteria used for selection was that the patients were first diagnosed as ME, CFS, or Depression.**

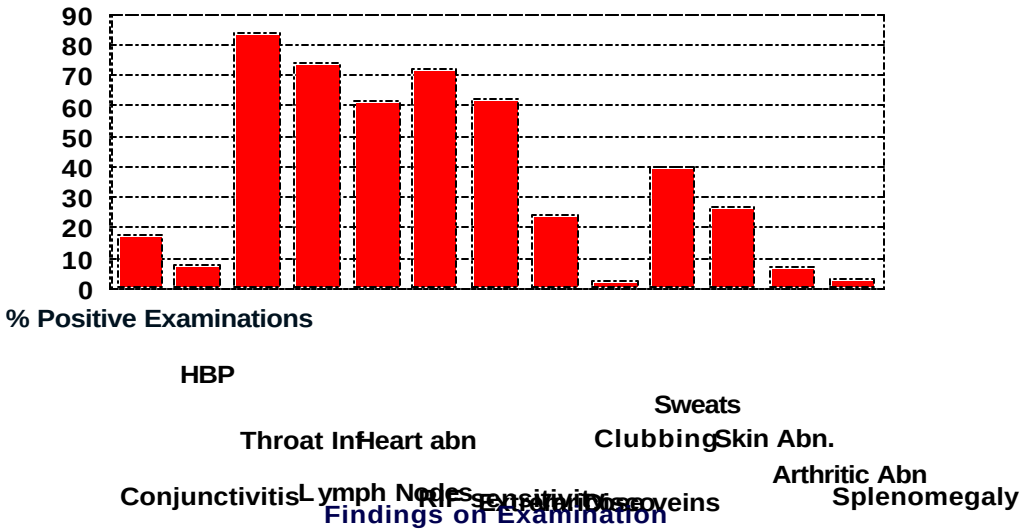
Sex		**Age Group**		**Length of Illness:**	
Male:	**236**	**<10:**	**2.1%**	**< 1 year:**	**12.5%**
Female:	**264**	**10 - 20:**	**16.7%**	**1 - 2 years:**	**20.7%**
		20 - 40:	**43.7%**	**2 - 5 years:**	**29.3%**
		> 40:	**37.5%**	**> 5 years:**	**37.5%**

Exposure to Rickettsia:	**Known:**	**95%**
	Unknown:	**5%**

I. The following table displays the percentages of symptoms and diseases caused by Rickettsial infection in the above group of patients. The symptoms and diseases vary according to the main localisation of the infection.This is based on clinical findings and also described in literature.

Vascular Tissue		Reticulo Endothial Tissue	
Tiredness (**R1,4,7,9,13,28**)	92%	Recurrent sore throat (**R1**)	85%
Myalgia Arthralgia (**R5**)	90%	Appendicitis (**R5,8**)	65%
Cardiac. Valves ??**R1,6,7,8,9**)	90%	Lymphadenopathy (**R1**)	73%
Memory. Concentration (**R9**)	89%	Systemic Candidosis	53%
Headaches (**R1,9**)	88%		
Bruising (**R26**)	81%	**Neurological Tissues**	
Vision - Uveitis Conj. (**R1**)	39%	Encephalitis (**R1,11,13,17)**	12%
Dermatological (**R9,10)**	23%	Epilepsy (**R1,13,24)**	69%
Psychotic disorder treated (**R9**)	69%	MS (**R4,9,24,37)**	5%
Atypical Hepatitis (nausea-vom.)	64%	Loss of Balance	28%
Raynaud Syndrome (**R13**)	69%		
Pulmonary Disease (**R6,8,14,31**)	21%		
HBP	9%		

II. **Clinical Examination**The following graphic shows that most patients display the same clinical findings.



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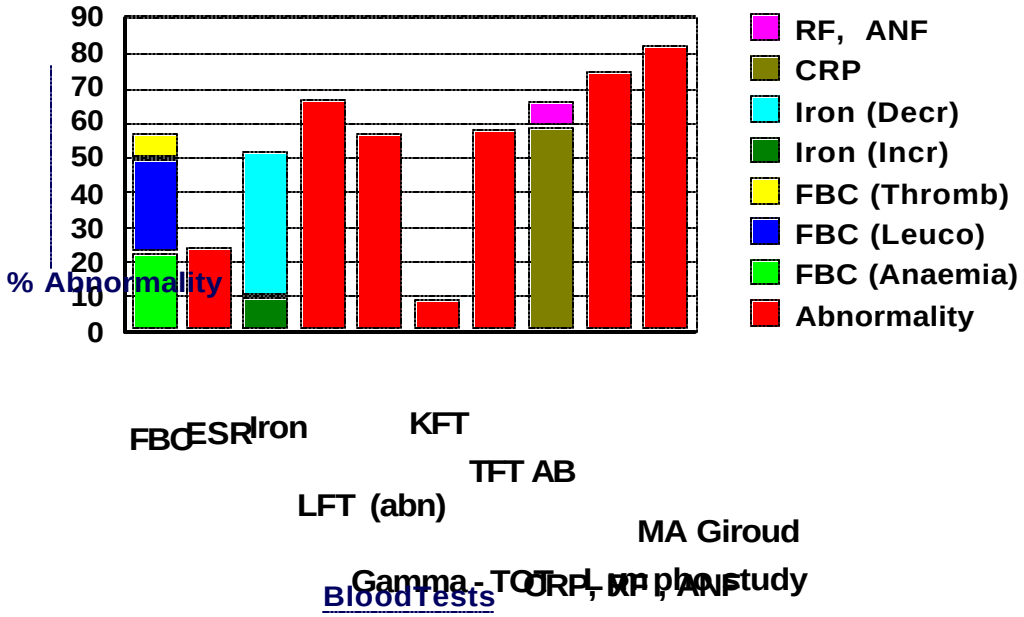

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3. From being treated by painkillers, antidepressants, sedatives, cortisone to none
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5. Biological investigation: from having:
   - LFT Δ
   - RF ↑, CRP ↑, ANF ↑
   - KFT ↑
   - Thyroid antibodies ↑
   - Iron Δ

   } To normal or nearly so

Based on the assessment, the treatment is prolonged or stopped (3 months to 2 years - 8 months average).
However, as previously mentioned, the length of treatment is not directly correlated to the length of illness:

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8.

"La santé est comme une mongolfière: il faut parfois lâcher du lest"

CFS - Rickettsial Infection - Case Studies by Dr. Cécile Jadin, South Africa

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THEREFORE

the diagnosis of Rickettsial disease stands on 3 corner stones:

Symptoms — **Clinical findings** — **Biological investigations**

Patients and Methods: **Statistics of 500 patients (100% Caucasian)**

Criteria for selection: first dsed as ME, CFS, or Depression

Sex		Age Group		Length of Illness:	
Male:	236	<10:	2.1%	< 1 year:	12.5%
Female:	264	10 - 20:	16.7%	1 - 2 years:	20.7%
		20 - 40:	43.7%	2 - 5 years:	29.3%
		> 40:	37.5%	> 5 years:	37.5%

Exposure to Rickettsia:		
	Known:	95%
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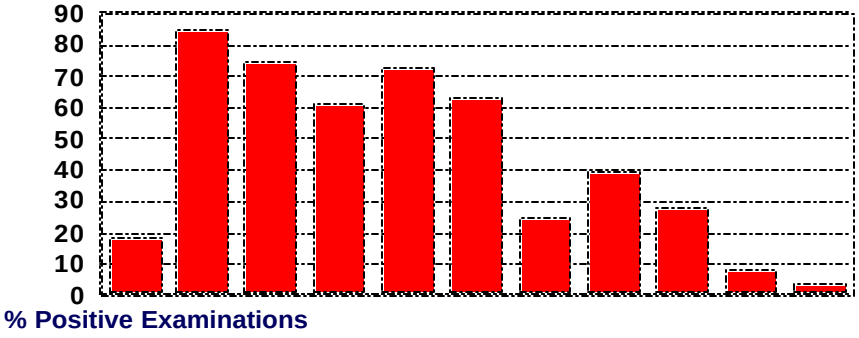

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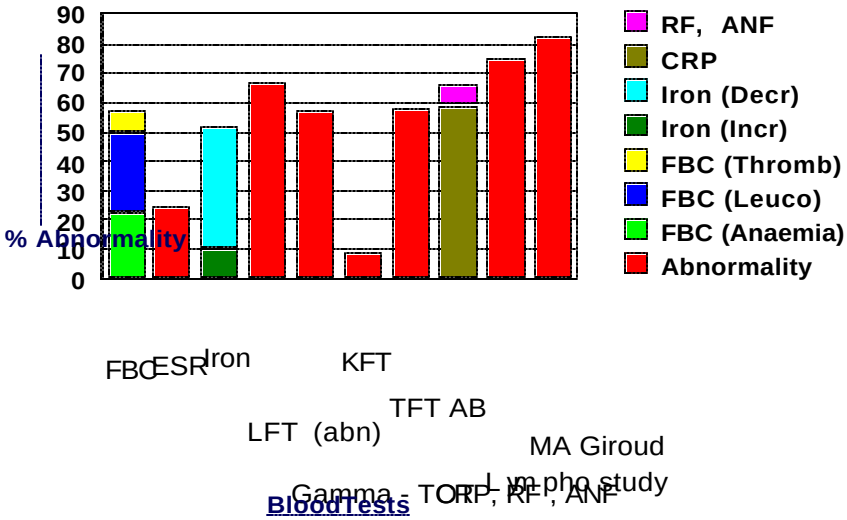

IV. CXR - MRI (Brain Scan) - Joint X-Ray

V. **Treatment:** average of 7 days/month of Tetracyclines

1.alternated (**R6,9,11,28**)
2. combined with Quinolones, Macrolides, Metronidazoles (**R6,43**)
3. high dosage (**R1,9,11,13,43**)
4. varying in length (fast response, slow response) (**R1,9,13,16,25,43,45,48**)
5. Anti malaria
6. Adjuvants
7. Exercise (Rickettsia has vascular impact) and hot baths (**R9**)

VI. **Herxheimer reaction**: Prognosis value (**R1,11,18,44,47**)

VII. Results - Statistics:

Including patients not treated, or treatment not completed

358 patients very well, or cured	72%
101 patients stopped consultation after 1 to 3 treatments	20%
26 patients never commenced treatment	5%
15 patients showed no improvement to treatment	3%

Excluding patients not treated or treatment not completed

358 patients very well, or cured	96%
101 patients stopped consultation after 1 to 3 treatments	4%

NB:

% of reactivation or reinfection of patients well improved: 7%

- , but patients recover after further treatment (2 to 3 months average) (R25,26)

VIII. This **Assessment** is performed monthly to judge patients progress towards "**very well**" : symptoms

meaurement

increase in activity

symptomatic medication independence (pain killers, anti-depressants, hypnotics, cortisone)

medical examination

biological investigation.

Based on this, the treatment is prolonged or stopped (3 months to 2 years - 8 months average).

However, length of treatment is not directly correlated to the length of illness.

Patients can be divided into 2 categories:

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2. Slow progress - their illness was Rickettsia plus other factors (**R36**)

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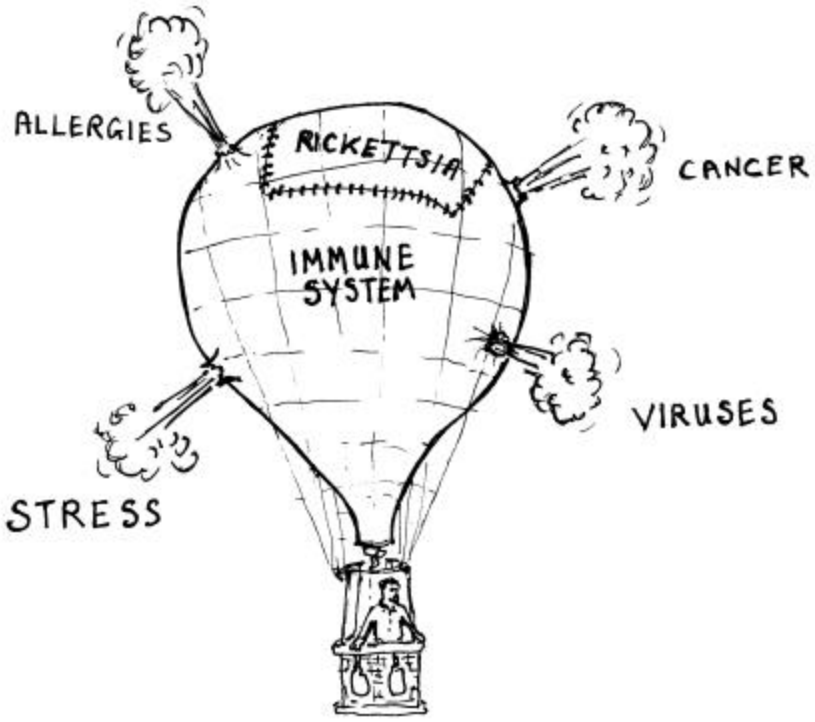
2. CFS - Nightingale disease (**R33**)
3. Lymphocyte study results similar in sheep with tick-borne diseases and in patients with CFS (**R19,21,22,23**) and patients with Q Fever endocarditis (**R48**)
4. CFS - PQFS (**R4**)
5. Rickettsial Disease through History (**R7,8**)
6. Treatment rates success (**R28**)
7. Interaction between protozoa, viruses, bacteria, and stress? (**R9,29,34,36,42**)



8.

Rickettsia

is a suggested way

to repair an Immune System,

quickly or slowly.

"La santé est comme une mongolfière: il faut parfois lâcher du lest"

Depression, Psychotic Dysfunction, Rickettsial Infection Case Study
by
Dr. Cécile Jadin, Randburg, South Africa

Summary: The possibility of a Rickettsial origin for symptoms of depression and psychotic disfunction has been suggested by French scientists (Ch. Nicolle, Giroud, Legag, Jadin, Bottero) in their published works. Hence 300 patients, diagnosed as suffering from depression, or other neuropsychiatric dysfunction have been treated with antibiotic where a positive indicaton of Rickettsial infection was revealed as follows:

1. Many symptoms of these patients were similar to those exhibited in chronic Rickettsial diseases.
2. The treatment followed the finding that their serum reacted positively to the **Giroud** micro-agglutination test.

Giroud Test - specific for testing antibodies to these 5 antigens (**R36**):

- Rickettsia Prowazeki
- R. Mooseri
- R. Conori
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- Neo-R. Q18

Done by micro agglutination
Depends on the quality of antigens
Comparative studies with IFA test gave very similar result

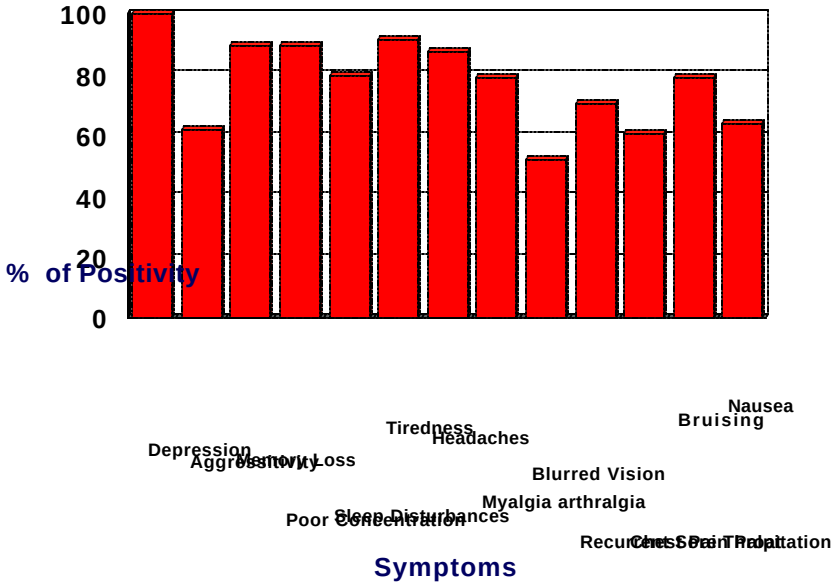
- Positive reaction = presence of antibodies; (does **not** necessarily mean illness)
- Negative reaction does **not** suppress Rickettsial etiology (**R1,25**)

Patients and Methods: Statistics of 300 patients (100% Caucasian)

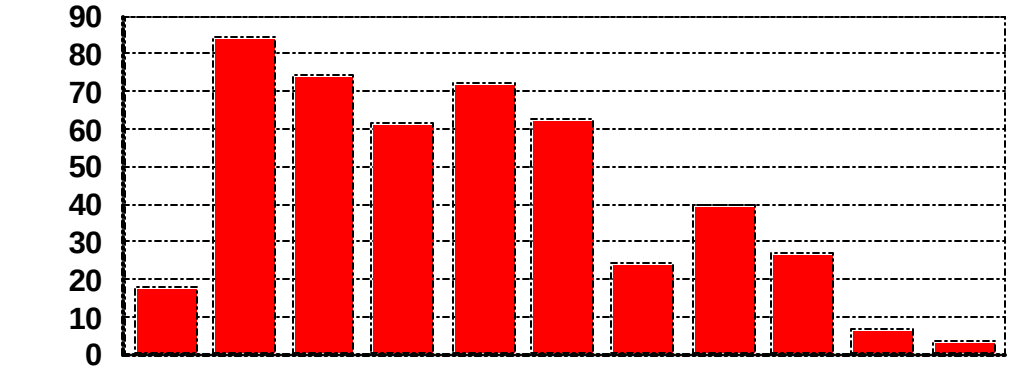
Selection Criteria: first dsed as Depression and Psychotic ?

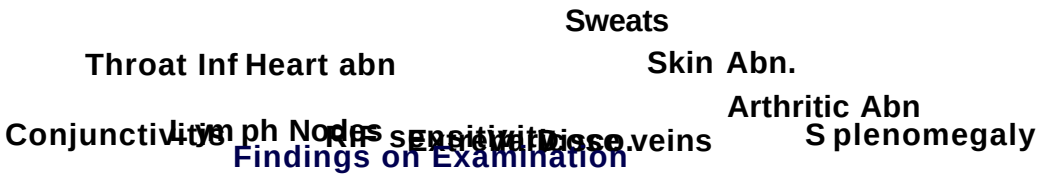
Sex		Age Group	Length of Illness:	
Male:	**127**	**12 - 69**	**Minimum**	**6 months**
Female:	**173**		**Maximum**	**20 years**
			1 exc .	**3 months**

I. Main Symptoms:



II. Clinical Examination





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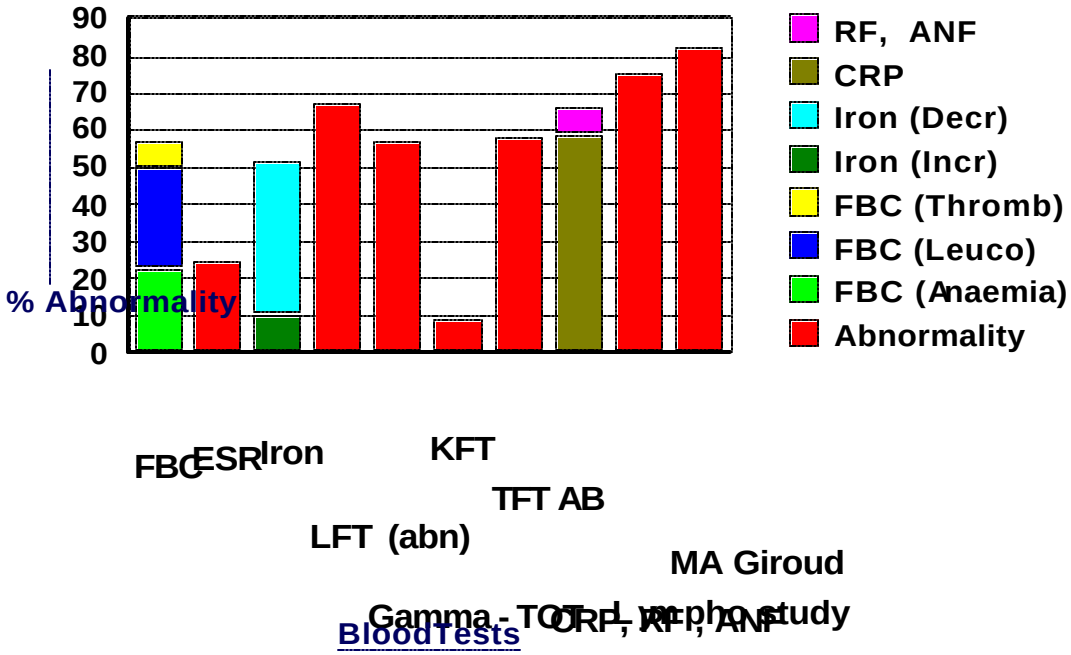

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When confronted with psychiatric disorders, we should always look for associated pathogens such as:

- Rheumatoid disorders
- Liver disorders
- Peripheral angiopathy
- Iron disorders
- Thyroid antibodies

to help us to orientate a diagnosis and treatment, because as shown by the study there may be a causative link between depression and an infectious agent.

Depression, Psychotic Dysfunction, Rickettsial Infection Case Study
by
Dr. Cécile Jadin, Randburg, South Africa

Summary: The possibility of a Rickettsial origin for symptoms of depression and psychotic disfunction has been suggested by French scientists (Ch. Nicolle, Giroud, Legag, Jadin, Bottero) in their published works. Hence 300 patients, diagnosed as suffering from depression, or other neuropsychiatric dysfunction have been treated with antibiotic where a positive indicaton of Rickettsial infection was revealed as follows:

1. Many symptoms of these patients were similar to those exhibited in chronic Rickettsial diseases.
2. The treatment followed the finding that their serum reacted positively to the **Giroud** micro-agglutination test.

Giroud Test - specific for testing antibodies to these 5 antigens (**R36**):

- Rickettsia Prowazeki
- R. Mooseri
- R. Conori
- Coxiella Burnetti
- Neo-R. Q18

Done by micro agglutination
Depends on the quality of antigens
Comparative studies with IFA test gave very similar result
- Positive reaction = presence of antibodies; (does **not** necessarily mean illness)
- Negative reaction does **not** suppress Rickettsial etiology (**R1,25**)

Patients and Methods: Statistics of 300 patients (100% Caucasian)

Selection Criteria: first dsed as Depression and Psychotic ?

Sex

Male:	127
Female:	173

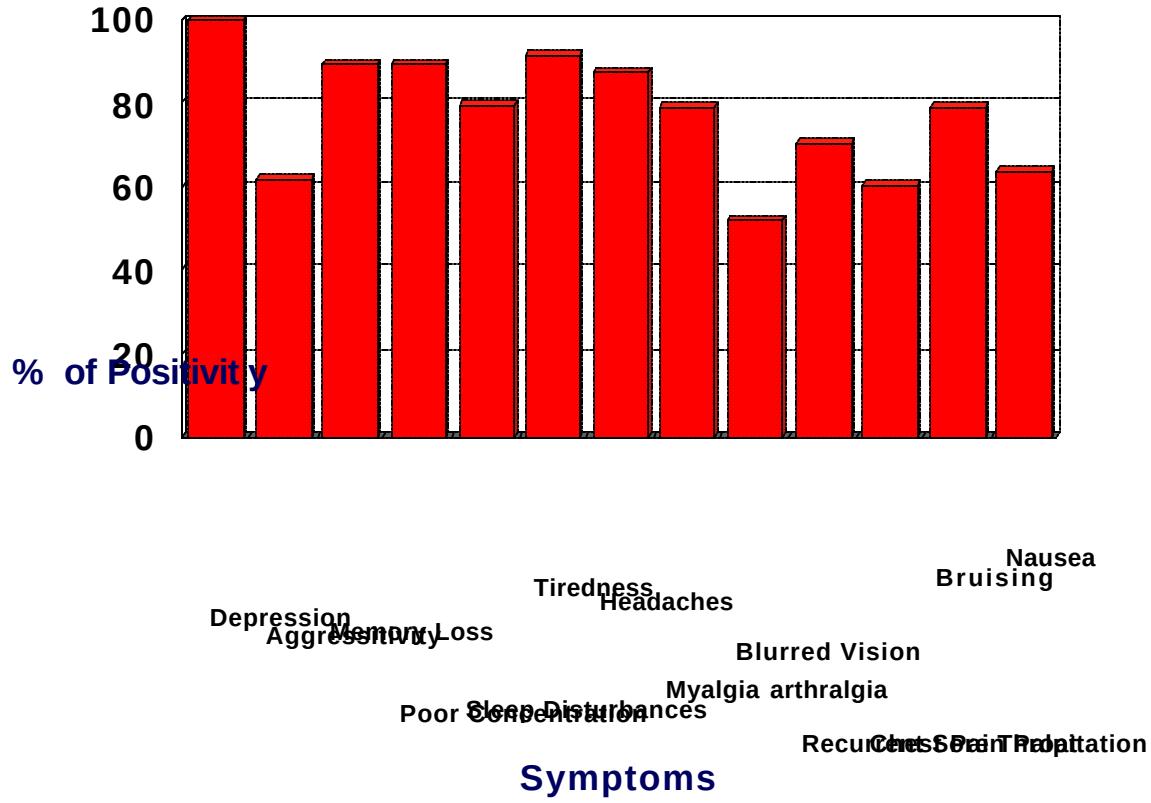
Age Group

12 - 69

Length of Illness:

Minimum	6 months
Maximum	20 years
1 exc .	3 months

I. Main Symptoms:



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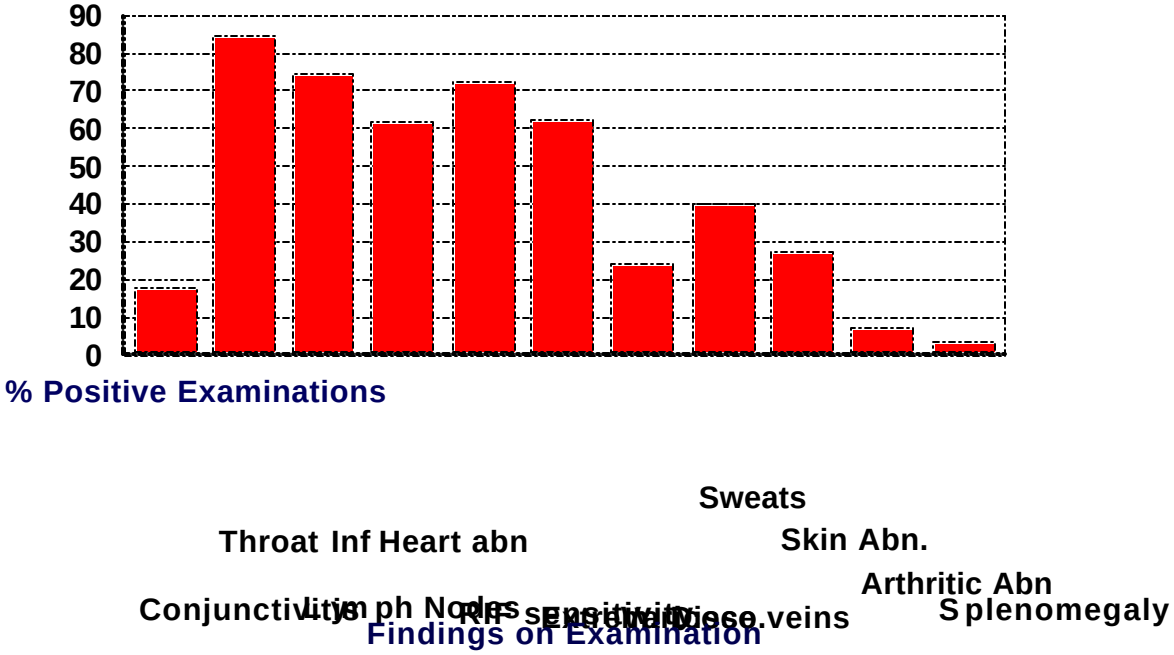

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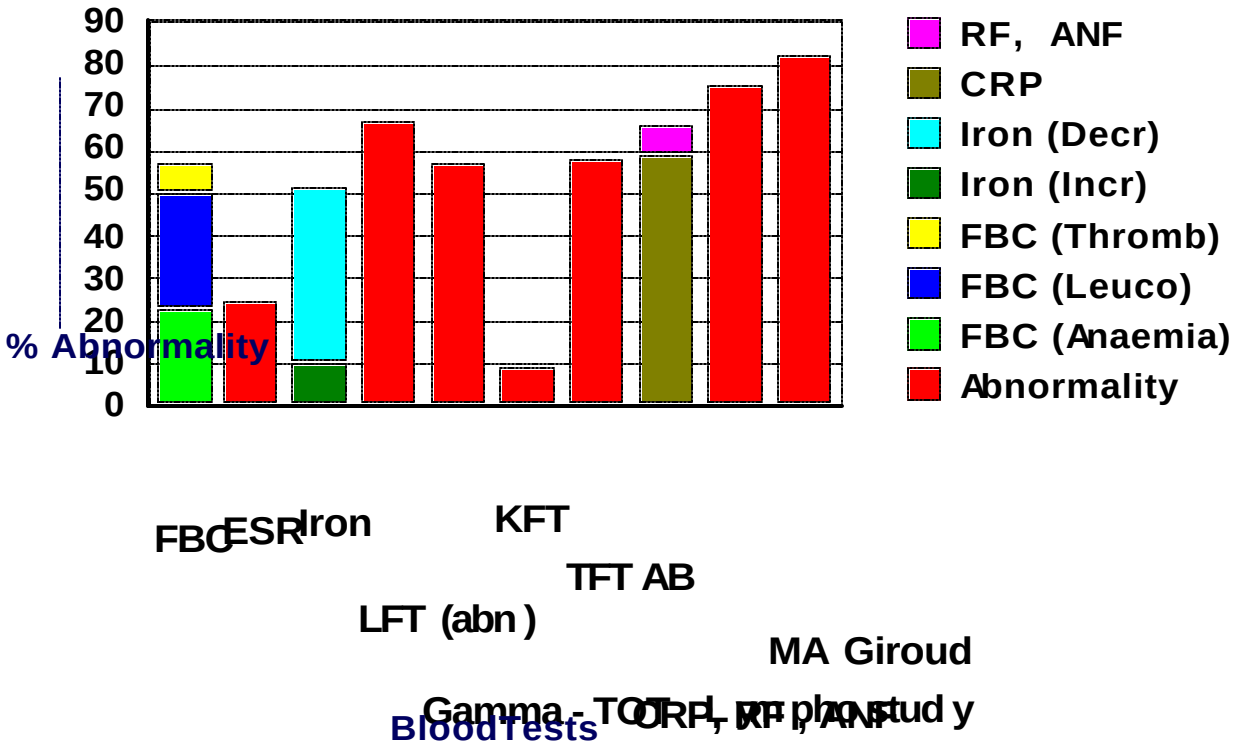

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The Rickettsial Approach and treatment of patients presenting with CFS, Fibromyalgia, Rheumatoid Arthritis and Neurological Dysfunction.

Manly Conference
February 1999

CL Jadin MD MBBCh

Republic of South Africa

Introduction

The author, Cécile Jadin, is originally from Belgium, but has been practising in South Africa for the last 17 years. She is a surgeon by profession. In South Africa, in addition to practising as a surgeon, she also assisted her husband in his general practice. For the last 7 years, she has focused on the subject of Rickettsia and her approach has naturally been that of a clinician, and it is in this context that the paper is written.

To understand why she took the Rickettsial approach her background needs to be explained. Her father was Professor JB Jadin, who undertook groundbreaking research on tropical diseases, among them Rickettsial infection, with Professor Paul Giroud in Central Africa, South Africa, the Near East, and in Europe, developing the work started in the Pasteur Institute of Tunis, with Professor Charles Nicolle, who was a disciple of Louis Pasteur. Thus she was familiar with those germs from an early age and her work represents the results of teamwork through the last 100 years.

12 years ago, one of her friends became unable to walk and was diagnosed as having ME. For 4 years Dr. Jadin suggested the diagnosis of Rickettsial Infection, and therefore the Weil-Felix test was performed several times in South Africa, but the results were negative. The friend developed an acute appendicitis. After Dr. Jadin removed her appendix, her serum was sent to Prof. JB Jadin in Belgium to test for Rickettsiae, and the result was positive. Dr. Jadin treated her with Tetracyclines and 3 weeks later, she was riding her horse again. Dr. Jadin was sceptical. But this case brought her 200 patients and the publicity surrounding an investigation of her methodology by the South African Medical Council brought her several thousand more. Thus Dr. Jadin started to focus on the Rickettsial approach.

Original Research

Research on Rickettsioses was originally developed by French, Polish and Russian scientists. They followed Charles Nicolle's (Pasteur Institute, winner of the Nobel Prize for medicine in 1933) hypothesis, which is that occult diseases are a reality and their cohabitation in the same host will lead to the bankruptcy of the immune system (8). By occult disease Charles Nicolle implies the asymptomatic stage of the disease, where the agent is present in the host, but dormant (3). The emergence of a virus, bacteria, stress or pollution can activate this agent, which leads to the symptomatic stage.

An example of this cohabitation is the infant mortality rate described by J.B. Jadin in Central Africa. Neonates diagnosed with malaria and Coxiella Burnetti all died as opposed to those with malaria only (20).

The numerous publications of these authors are unfortunately all in French, so their circulation was limited. They also, as academics, excluded the media. Therefore the real importance of their discovery is still to be made widely known.

Rickettsia and CFS

The fairly recently recognised entity of CFS gives us a perfect opportunity to try the etiological route to understand this disease. Along this route we will automatically enter other medical fields, inviting us to consider an infectious etiology in cardiology (4,5,9,11,12), in psychiatry (3,17), in neurology (3,29) and in rheumatology (28), rather than describing the symptoms and gathering them into syndromes (20, 40).

Obviously one germ can cause many diseases depending on a selective topicality for one or more particular tissues as well as one disease can be caused by different germs alone or simultaneously. Therefore we would like to concentrate on the causative agent, rather than on the name of, and the criteria to classify, the diseases.

There are many reasons suggesting the infectious etiology and, more specifically, Rickettsial-like organisms of CFS. Amongst those reasons:

1. Consider the following :
   - CFS was first reported in Incline, Nevada in 1984 (1) and developed into epidemic proportions.
   - Rocky Mountain Spotted Fever originated from the same place in 1916 (9,29).
   - The spirochete Borrelia Duttoni, first blamed for causing the recurrent Malgache fever described in the journals written by Drury in 1702 (24) in Madagascar, then by Scheltz in the Belgian Congo in 1933, by Palakov in Cape Town in 1944, by Heisch in Kenya in 1950.
   - Lyme Disease appeared (or reappeared?) more recently in Lyme, Connecticut in 1975 (Borrelia Burgdorferi) (25).

   Could these be new names for old diseases?

   All of the above highlights the life of a germ as an individual emerging and disappearing in a wave pattern epidemically and historically. Like us, germs have to adapt, producing new variations of themselves, (not new species), that may or may not survive on their own, with or without the help of another germ. This is circumstance-dependent, and these particular circumstances will never reoccur. Some of those variations will acquire specific and consistent characteristics that will allow them to survive. This is their 'civilisation'. We only see them when they succeed, and only then do new avenues of investigation open up, while others are abandoned.
1. A link has been established between Florence Nightingale disease and CFS (21). The fact that she was working surrounded by lice, fleas and ticks, treating soldiers with wounds and with epidemic typhus during the Crimean war, could be a logical explanation as to why she was terribly tired during the last 2 decades of her life; and possibly has relevance to Gulf War illnesses (13). Zinsser has developed the same concept in his classic book "Rats, Lice and History". He contends:

   "Soldiers have rarely won wars. Typhus and other infectious diseases have decided the outcome of more military campaigns than Caesar, Hannibal, Napoleon and all generals in history. Depending on the outcome for each warring faction, either the epidemics were blamed

for defeat, or the generals were credited with victory." (2). More examples of this phenomenon were reported by JB Jadin (29).

2. Lymphocyte studies conducted on sheep with tick-borne diseases (14), CFS patients (15,16), and patients with Q Fever endocarditis (11) are showing amazingly similar results.
3. Coincidentally, the new name suggested in the Lancet for CFS is PQFS (Post Q Fever Syndrome) in April 1996 edition (22).
4. During the First World War an estimated 25 million Russians contracted Louse-borne epidemic typhus, resulting in 3 million deaths. Why not before or after? It could suggest that the stress factor reactivates the virulence of Typhus Prowazeki (2, 3, 9). In the medical history of CFS patients, stress has often been described as the start of the illness.
5. The symptoms displayed by CFS, Fibromyalgia, RA, and even neurological patients as MS, show the same diversity of symptoms as Rickettsial patients. How many scientists blamed the diversity of symptoms for misleading unprepared practitioners in the diagnosis of chronic Rickettsial infection (30)? That same diversity could have contributed to the delay in recognising CFS. French authors (Giroud, Jadin, Legag) attribute those multiple aspects to a generalized micro-vascular invasion. They widely demonstrated the persistence of Rickettsiae in the vessels (4), (18). The suggestion here is that the well-known, well-documented entity of Rickettsial disease, showing the same symptoms as the newly arrived CFS, might simply, partially or totally be caused by the same agent.
6. The last, but not the least reason, is the success rate of the Rickettsia treatment, Tetracycline, applied on CFS, Fibromyalgia, Depression and MS etc. patients. Dr Phillipe Bottero on 100 patients since 1981, Dr Peter Tarbleton on 300 patients in 1993 in South Africa(17) and myself on a much larger number of patients, maintain an 84% - to 96% recovery rate.

Transmission of Rickettsiae

Rickettsiae are transmitted by arthropods (36), except for Q Fever, which does not really need vectors;

- they are resistant to humidity and to dryness
- they will stay virulent for 60 days in milk
- 4 months in sand
- 6 months in meat
- 7 - 9 months in cotton (4).

They are spread by rodents and birds. Through the centuries, bird migration has been responsible for changing the geographical distribution of disease (27) - but this is nothing compared to the effect of the explosion of these diseases due to the cocktail effect created by distribution through global air traffic (26).

Equally the transport of insects compared to the import and export of livestock - as in the case of the import of 10,000 parrots from Paraguay to Belgium when some 2,000 died, leaving the virus well and alive behind them (27), (identified by my father as Neo-Rickettsia Bedsonia).

This world distribution does not include Antarctica, where they do not survive.

Fish also share this disease, as Erlichioses is, according to breeders, a common problem (Psichi Rickettsia Salmoni, first described in Chile) (31).

Patients and Diagnosis

3,400 patients presented with CFS, Fibromyalgia, RA, depression and MS have been diagnosed as suffering from Chronic Rickettsial Infection (CRI) after eliminating other diseases as a cause (diabetes, cancer etc.).

The majority of my patients report a flu-like infection, with often an elevated temperature and severe headaches. This lasts for a few days, disappears or reoccurs, and then leaves them with a chronic condition of CFS, Fibromyalgia etc. as mentioned above.

Diagnosis of CRI is established by Giroud's Micro-Agglutination test against five strains of Rickettsiae:

- R. Prowazeki: the epidemic type of Typhus
- R. Mooseri, which is endemic
- R. Conori, which belongs to the spotted fever group
- Coxiella Burnetti, which is well known as Q Fever. It has 2 phases; Phase II is pathogenic
- Neo Rickettsia Chlamydiae which falls into the Neo-Rickettsia group (18)

Important Points:

a) A high reading means a high serological level of antibodies - a negative reading in endemic areas reflects the poverty of the immune system (24).
b) Agglutination happens or does not - therefore there is no possibility of personal interpretation. Test quality depends on Antigen quality (3).
c) Positive tests can be found in people who display no symptoms (Giroud, Jadin (18); 26% according to Drancourt (39)).

However, the Micro-Agglutination test of Giroud is not our only tool to establish the diagnosis of Rickettsial infections. We find the following blood tests most relevant:

- LFT: the hepatotoxicity of Rickettsiae has been reported as early as 1937 by Derrick in Q Fever (19, 29), followed by many others - Giroud, Lenette, Legag, Brezina, Perron, Kelly, Raoult, etc. In these cases, Tetracyclines are improving or normalizing liver function (6).
- Iron study (50% of abnormalities corrected with Tetracyclines only and when necessary with a short course of iron supplement).
- Thyroid AB rather than TFT, although the TFT show abnormalities in 3% of patients, the thyroid AB are elevated in 28% of cases and improve or normalise rapidly with treatment.
- CRP, RF, ANF, WR was positive in 53% of patients, (39) and also improved with treatment and often normalised.
- Mycoplasma (only researched after the Manly conference, February 1998).

Symptoms

Patients' symptoms most commonly exhibited are:

- Tiredness (4,5)
- Headaches, retroorbital and temporal, worst after prolonged horizontal position or mental effort (4).H
- Myalgia (3)
- Arthralgia migrating (2,3,5)
- Loss of balance (29)
- Vision abnormalities (3,29)
- Raynaud syndrome (18)
- Nausea (8,9,18)
- Recurrent sore throat (23)
- Memory and concentration deficit (4).
- Chest pain, palpitations (8,12, 18)
- Sweats, low grade fever (4)
- Bruising (4)
- Psychological and neurological disorders(4,5,18,29,30)

We find quite a valuable guideline in the physical examination, which often shows

- An inflamed throat and multiple adenopathies, reflecting the selective topicality of Rickettsiae to endothelial tissue
- Heart abnormalities (vascular (4,12,30) and valvular impact (2, 39))
- RIF tenderness (chlamydiae 18 in appendix (23))

Treatment

After establishing these 3 cornerstones Symptoms
Physical examination
Blood tests

treatment is administered:

- Guided by our predecessors, (Giroud, Jadin, Legag etc.)
- Refined by our contemporaries, (Bottero and Raoult)
- And by my own daily, private lessons (each patient is one).

The treatment consists of 7 to 12 days per month of a specific Tetracycline. The monthly treatment aims to follow the Rickettsial development in the cell.

1. A high dosage is required (4,5) with the limitation of:
   - **Safety** (32) Goodman et al (33) highlights irreversible hepatotoxicity in intravenous administration only. Our experience was that when liver functions were normal to start with, they stay normal. If they were abnormal, they will improve during treatment and generally return to normal. Cases of fatty acid depots (as shown by liver scan, before and after 6 months to 1 year of treatment) have disappeared (1 MS, 4 ME). This confirms the fact that Rickettsiae are more hepatotoxic than Tetracyclines.
   - **Tolerance**.
     a) The gastric intolerance will be successfully prevented by using a gastric pump inhibitor during and if necessary before and after the administration of the Tetracyclines.
     b) The tolerance of the treatment is directly related to the Herxheimer reaction (4, 6, 26, 37), which is a reactivation of old symptoms and/or exacerbation of present symptoms that occurs on antibiotherapy. Its presence has a very important diagnosis and prognosis value (4). They might or might not be parallel to a serological reactivation. It will fade with the number of treatments received. When very severe, the HR is treated with Probenecid.
1. The Tetracyclines are alternated because:

a) A patient is frequently contaminated by many strains of Rickettsiae (5) and different Rickettsiae have different sensitivity to different Tetracyclines or combinations. (4).
b) A patient might build resistance to each Tetracycline (4, 17).
c) Patients show individual sensitivity to different Tetracyclines or combinations and there is very often a privileged reaction to a specific treatment (6).

3. The Tetracyclines are combined with Quinolones, Macrolides or Metronidazole (7), because Rickettsiae present a wide heterogenicity of susceptibility to different drugs (4).
4. The treatment is often long due to:
   a) The chronicity of the germ (4)
   b) The multiple foci of Rickettsiae (18)
   c) The fact that Rickettsiae have a slow evolution and some foci are dormant, encapsulated and therefore protected from antibiotherapy. Only when they become active can they be treated (5).
   d) Each treatment will allow the immune system to produce and maintain a proper and efficient level of antibodies. This happens each time the antigen Rickettsiae are released from the cell to the blood stream while on antibiotherapy (Legag) (4).
   e) The length of the disease should logically imply a lengthy treatment. In our experience, this point is not always true. Patients, ill for many years, may recover after a few months treatment.
3. Antimalaria has been found efficient to improve Rheumatoid symptoms and Rheumatoid biological findings (see patients' files). Christopher Columbus knew it in the 15th century, as he gave tree bark containing quinine to his crew to prevent malaria and also mysterious body pains. The Imperial army of Queen Victoria did the same and so was born Indian Tonic water.
4. Adjuvants such as Vitamin B complex and acidobacillus are also used.
5. Cortisone is avoided as much as possible as it is known to weaken the Immune System in general (3) and also to reactivate the disease in experiments on guinea-pigs (39). Cortisone has been accused of interfering with the diagnosis of Rickettsia by masking the antibody level (4).
6. Exercise is recommended, for the following 3 reasons:
   - Rickettsiae is a vascular disease and exercise, properly done, will improve the smooth peri-vascular muscle function, as well as develop the most important muscle, the heart.
   - The fact that strains of Rickettsiae grow better in vitro when maintained in a CO2 enriched atmosphere (34).
   - The suggestion that Rickettsiae grow best when the metabolism of the host cell is low (38).
7. Hot baths are important to eliminate toxins via the skin, produced by Rickettsiae antigens when liberated in the bloodstream by antibiotherapy.
8. Reinfection may obviously occur. Reactivation (called so rather than relapse) may also happen due to the interaction of bacteria, virus, stress, pollution, etc. causing the Rickettsiae forms' to change to active from dormant (35).

Measurement of Progress

Patients are seen monthly to judge progress on:

1. Symptoms
2. Activity increase (From bedridden to back to exercise or back to work)
3. From being treated by painkillers, antidepressants, sedatives, cortisone to none
4. Medical examination
5. Biological investigation: from having:

- LFT
- CRP raised
- KFT raised
- Iron
- RF raised
- ANF raised
- Thyroid antibodies raised

Back to normal, or nearly so

Based on this assessment, the treatment is prolonged or stopped (3 months to 2 years: 8 months on average). However, as previously mentioned, the length of treatment is not directly correlated to the length of illness:

Therefore patients can be divided into 2 categories:

1. Fast progress - their illness was mainly Rickettsia

2. Slow progress - their illness was Rickettsia plus other factors (20).

"La santé est comme une mongolfière: il faut parfois lâcher du lest"

Health is like a hot air balloon. You have to get rid of excess burdens to keep it in the air. Rickettsia is the easiest one to lose

THE IMMUNE BALLOON

Appendix 1: CFS - Rickettsial Infection: Sources of References

	References		Authors	
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2	Annals New York Academy of Sciences	1990		Preface
3	Acta Mediterranea di Patologia. Infectiva e Tropicale.	1984 Vol 3 1986 Vol 5 1987 Vol 6 no 3	JB Jadin, Ph. Bottero	P213
4	Bulletin Societé de Pathologie Exotique	1963	J. Gear Monteiro S. Nicolau J.G.Bernard N.R.Grist A. MasBernard Roche	P588 P680 P691-714 P758-793 P684-687 P714-724 P724-740
5	Clinique de la Résidence du Parc	1986		
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	Dermatology		Aymard	
6	Extrait Bull. Acad. Nat. Médecine	Vol 158 № 1	P. Giroud & J. Jadin	
7	European Journal of Epidemiology	1991	D. Raoult	P276
8	Academie Royale des Sciences d'Outre Mer	1963 № 6	J.B. Jadin	P1128-1129
9	Ann. Soc. Belge Med. Tropic. : Au Sujet des Maladies Rickettsiennes	1962 Vol 3	J. Jadin	P321
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14	Res. Vet. Scient.: Lympho. Subpopulations in Peripheral Blood of Sheep Experimentally infected with Tick-Borne Disease	July 1991	Woldehiwet	
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19	Maladies Infectieuses	1976 Ch 41	J. Orfila	
20	Arch. Inst. Pasteur Tunis: Les Infections Superposées Sont à la Base des Faillites de L'Humanité	1986 Vol 63	P. Giroud J.B. Jadin	P 97-99
21	Study Annales Internat. Med.: CFS: May 12: F. Nightingale's Birthday Comprehensive Approach	1994 - 121	T. Hennessy	P953-959
22	Lancet	Vol 347, April 1996		P977,978
23	Bulletin Acadamie Nationale Medicine	Vol 163 No 6 1979		
24	Bulletin Societé Pathologie Exotique	January 1952		
25	Lyme Disease CDC			
26	Rickettsiae and Rickettsial Diseases	1973	Brezinia	
27	Bulletin Societé Pathologie Exotique	1969	JB Jadin	
28	Annales of Internal Medicine	January 1995		
29	ASBMT	Vol 3 1952	JB Jadin	
30	EEG report of 18 year old Epilectic	February 1998	CL Jadin	
31	An Emerging Group of Pathogens in Fish	1997 Oregon S U Synopsis	JL Fryer M. Mauel	
32	SAMJ Tetracycline in ME - fad or fact?	Vol 82 1992	Bettina Schön	
33	Pharmalogical Basis of Therapeutics	1991	Goodman et al	

34	In vitro susceptibilities of Rickettsiae	1997	University of NC	
35	Relation entre Protozoaires, Virus et Bacteries	1984	JB Jadin	
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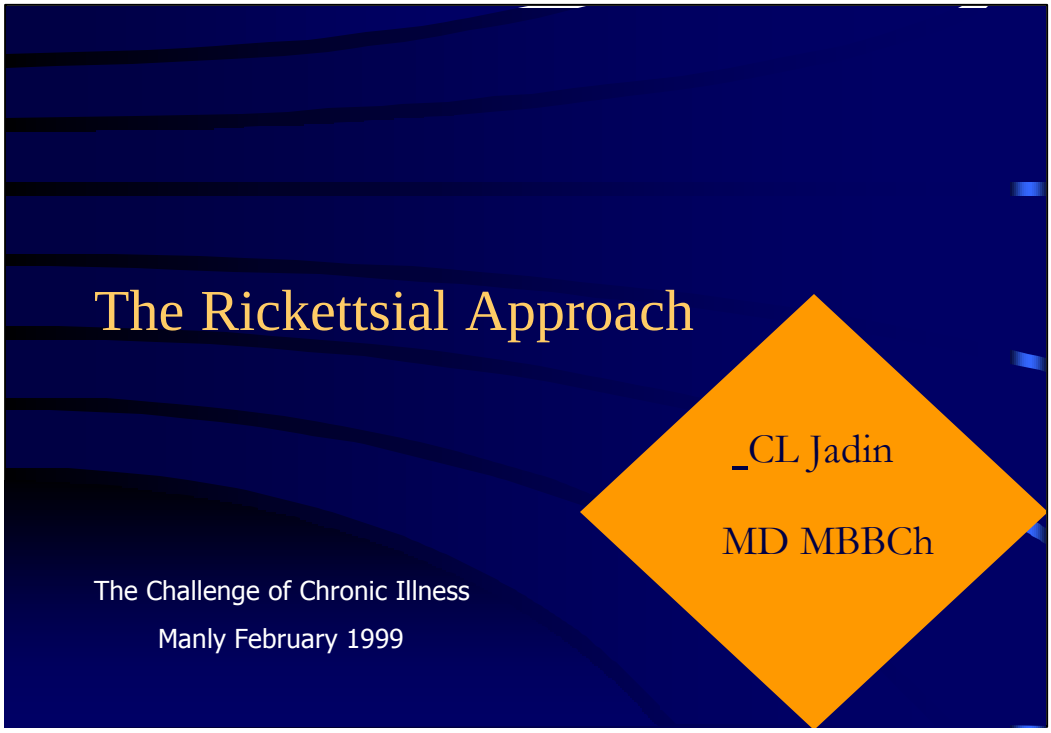

Good Morning/Afternoon, Ladies and Gentlemen

Thank you for this opportunity to be in your beautiful country and to tell you about my experiences in South Africa.

The subject of my address to you is: **The Rickettsial approach to the treatment of diseases grouped today as CFS**

PRESS FOR NEXT SLIDE ¿

PRESS ENTER ¿

This is what I will be covering today ----------,

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and this paper will be available as a handout. In my handout, I have noted the references which I used in drawing my conclusions. I have also brought all my reference material with me, for your interest, and to take copies if you wish.

First I think I must tell you how I came to link the **'forgotten disease'** of Rickettsia with the crop of new diseases.

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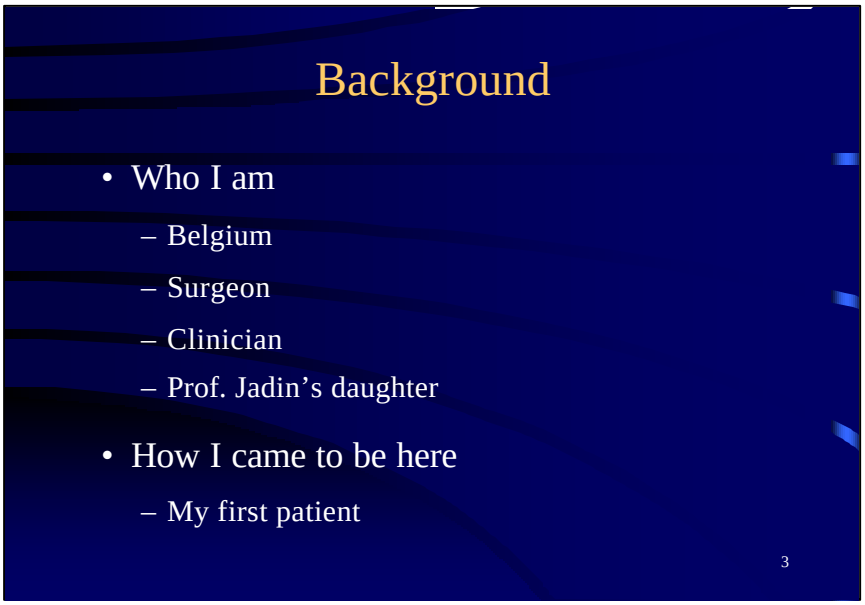

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I am originally from Belgium, but I have been practising in South Africa for the last 17 years. I am a surgeon by profession.

In South Africa, in addition to practising as a surgeon, I also assisted my husband in his general practice. **For the last 7 years**, I have been focusing on the subject of my paper and **my approach has naturally been that of a clinician**, and it is in this context that I am presenting my paper.

I wish to explain something of my background, so that you understand why I took the Rickettsial approach. I was born in the then Belgian Congo, because my father was Professor JB Jadin, who undertook groundbreaking research on tropical diseases, among them Rickettsial infection, with Professor Paul Giroud in Central Africa, South Africa, the Near East, and in Europe, developing the work started in the Pasteur Institute of Tunisia, with Professor Charles Nicolle, who was a disciple of Louis Pasteur. Thus I was familiar with those germs from an early age and my work represents the results of teamwork through the last 100 years.

¿

12 years ago, one of my friends became unable to walk and was diagnosed as having ME. For 4 years I suggested the diagnosis of Rickettsial Infection, therefore the Weil-Felix test was performed several times in South Africa but the results were negative.

One day, she came to see me with an acute appendicitis. After I removed her appendix, upon her request, I sent her serum to my father to test for Rickettsiae, **and it was positive**. I treated her with Tetracyclines and **3 weeks later, she was riding her horse again**.

I was sceptical.

But this case brought me a couple of 100 patients and the publicity surrounding an investigation of my methodology by the South African Medical Council brought me several thousand more.

So I began to focus on the Rickettsial Approach.

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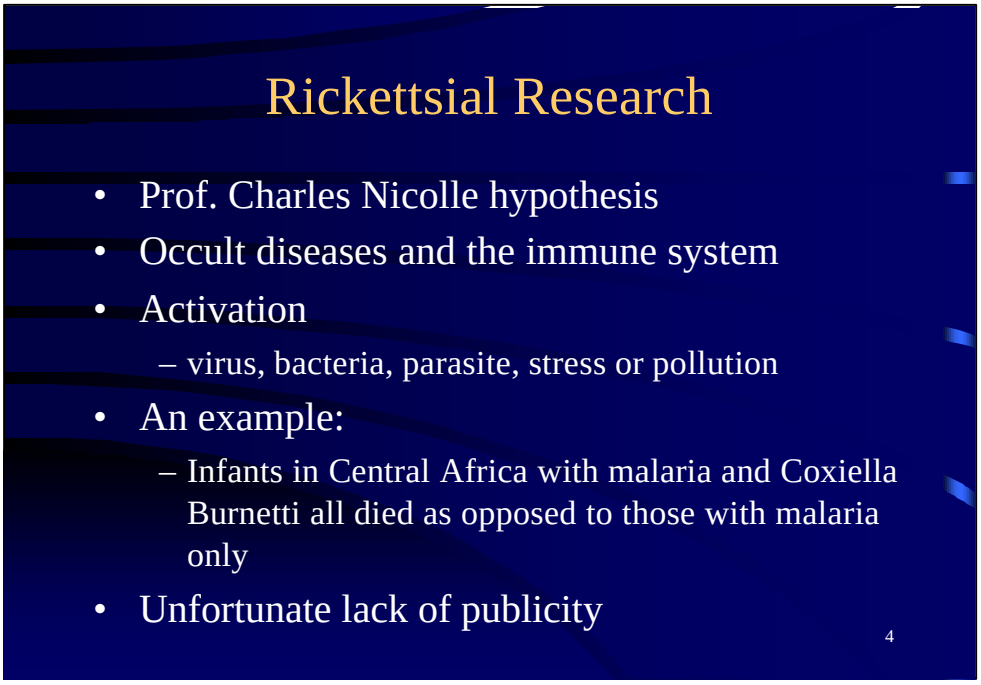

Research on Rickettsioses was originally developed by French, Polish and Russian scientists.

¿

They followed the hypothesis of Charles Nicolle: - Pasteur Institute, winner of the Nobel Prize for medicine in 1933

¿

which is that **occult diseases** are a reality and their cohabitation in the same host will lead to the bankruptcy of the immune system. By **occult** disease, Charles Nicolle implies the **asymptomatic** stage of the disease, where the **agent** is **present** in the host, but **dormant.**

¿

The emergence of a virus, bacteria, parasite, stress or pollution can **activate** this agent, which leads to the **symptomatic** stage.

¿

An example of this cohabitation is the infant mortality rate described by J.B. Jadin in Central Africa. Neonates diagnosed with malaria **and** Coxiella Burnetti **all died** as opposed to those with malaria only.

¿

The numerous publications of these authors are **unfortunately** all in French, so their circulation was limited. They also, as academics, excluded the media. Therefore the **real importance of their discovery is still to be made widely known**.

Let us now look at the **possible** links between **Rickettsia** and **CFS**.

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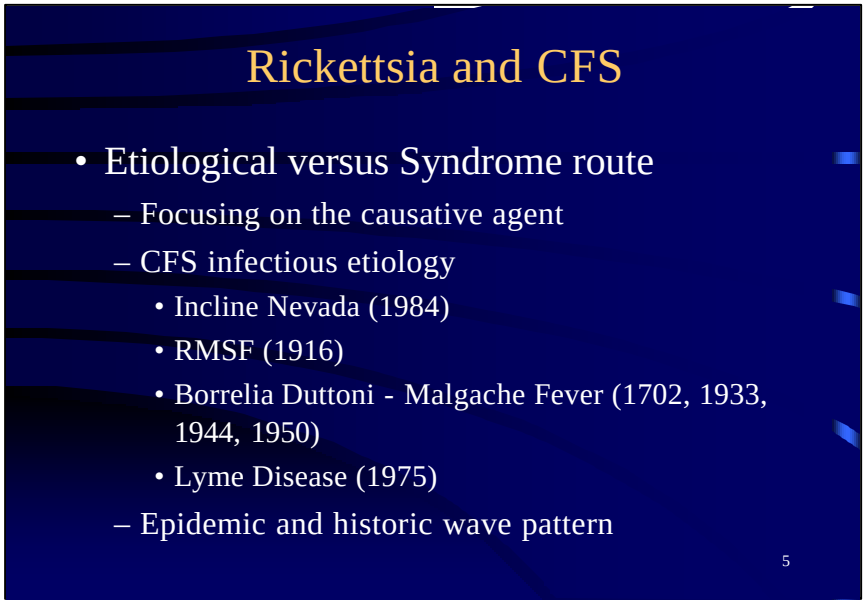

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The fairly recently recognised entity of **CFS** gives us a **perfect opportunity** to try the etiological route to understand this disease. ……Along this route, we will automatically enter other medical fields, inviting us to consider an infectious etiology in cardiology, in psychiatry, in neurology, and in rheumatology, rather than **describing** the symptoms and **gathering** them into syndromes .

¿

Obviously one germ can cause many diseases - depending on a selective topicality for one or more particular tissues. Also, one disease can be caused by **different germs alone or simultaneously**.

Therefore we would like to concentrate **on the causative agent** rather than **on the name of,** and the **criteria to classify**, the diseases.

There are **many** reasons suggesting the infectious etiology and, more specifically, Rickettsial-like organisms of CFS. Amongst those reasons:

¿

CFS was first reported in Incline, Nevada in 1984 and developed into epidemic proportions.

- Rocky Mountain Spotted Fever originated from the same place in 1916
- The spirochete Borrelia Duttoni, first blamed for causing the recurrent Malgache fever described in the journals written by Drury in 1702 in Madagascar, then by Scheltz in the Belgian Congo in 1933, by Palakov in Cape Town in 1944, by Heisch in Kenya in 1950.
- Lyme Disease appeared (or reappeared?) more recently in Lyme, Connecticut in 1975.

Could these be new names for old diseases?

¿

All of the above highlights the life of a germ as an individual emerging and disappearing in a wave pattern epidemically and historically. Like us, germs have to adapt, producing new variations of themselves, (not new species), that may or may not survive on their own, with or without the help of another germ. This is circumstance-dependent, and these particular circumstances will never reoccur. Some of those variations will acquire specific and consistent characteristics.

This is their **'civilisation'**. We only see them when they succeed, and only then do new avenues of investigation open up, while others are abandoned.

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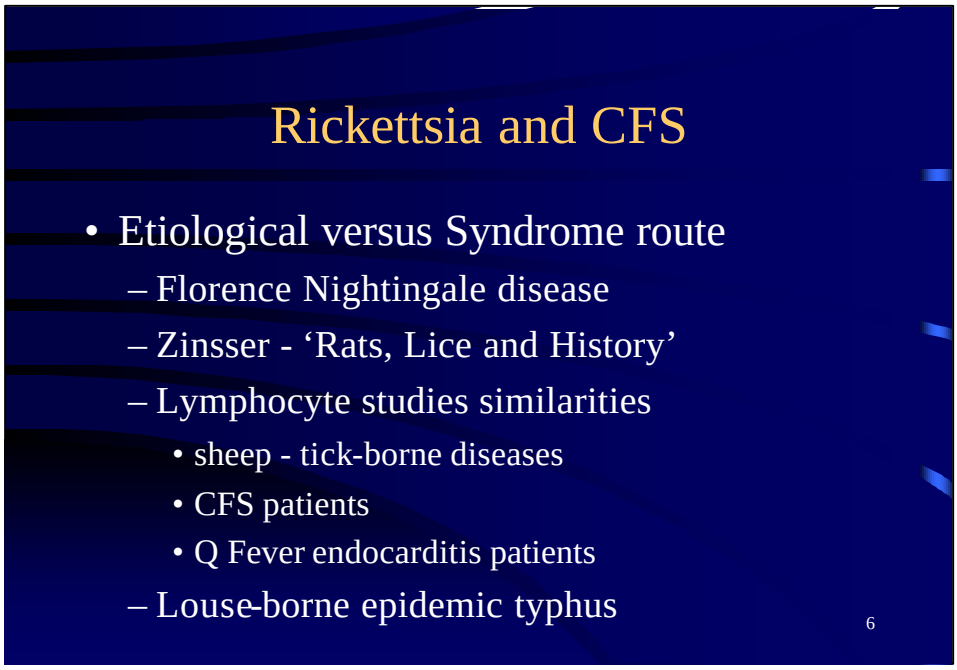

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Continuing the discussion on the Etiological versus the Syndrome route:

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A link has been established between Florence Nightingale disease and CFS. The fact that she was working surrounded by lice, fleas and ticks, treating soldiers with wounds and with epidemic typhus during the Crimean war, could be a logical explanation as to why she was **terribly tired** during the last 2 decades of her life; and **possibly** has relevance to Gulf War illnesses.

¿

Zinsser has developed the same concept in his classic book "Rats, Lice and History". He contends:

"Soldiers have rarely won wars. Typhus and other infectious diseases have decided the outcome of more military campaigns than Caesar, Hannibal, Napoleon and all generals in history. Depending on the outcome for each warring faction, either the epidemics were blamed for defeat, or the generals were credited with victory".

More examples of this phenomenon were reported by JB Jadin.

¿

Lymphocyte studies conducted on sheep with tick-borne diseases, CFS patients, and patients with Q Fever endocarditis, are showing **amazingly similar** results.

Coincidentally, the new name suggested in the Lancet for CFS is PQFS (Post Q Fever Syndrome) in April 1996.

¿

During the First World War, an estimated **25 million** Russians contracted Louse-borne epidemic typhus, resulting in **3 million** deaths. Why not before or after? It **could** suggest that the stress factor **reactivates** the virulence of Typhus Prowazeki. In the medical history of CFS patients, **stress has often been described** as the start of the illness.

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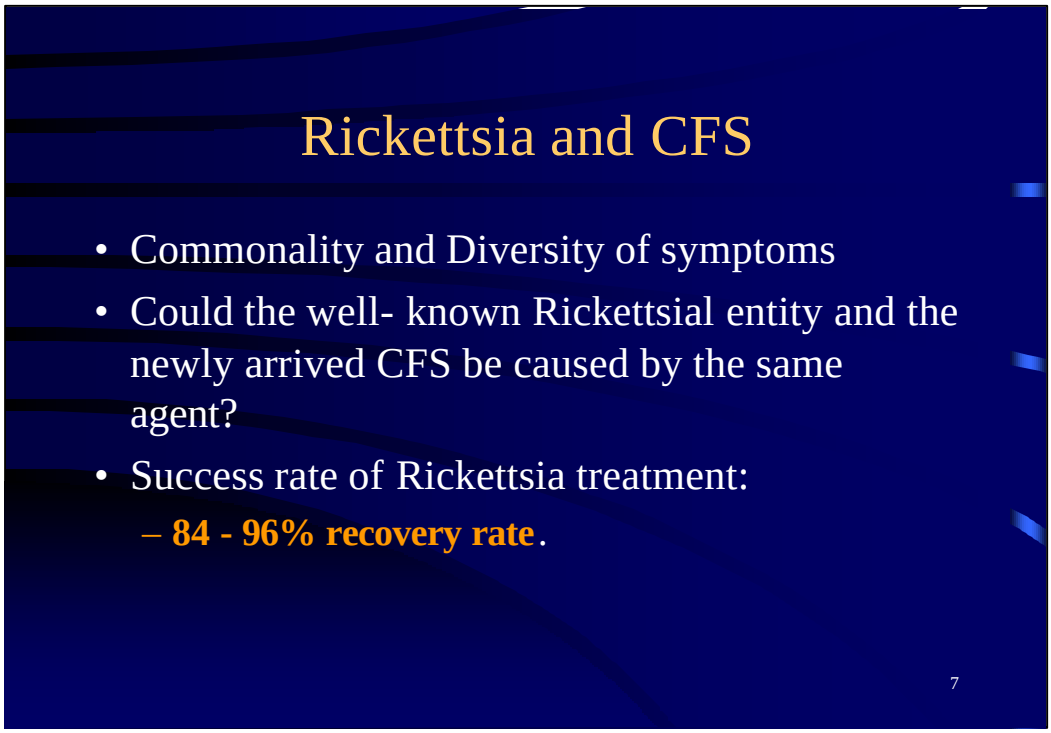

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The symptoms displayed by CFS, Fibromyalgia, RA, depression and even neurological patients as MS, show the **same diversity** of symptoms as Rickettsial patients.

How many scientists blamed the **diversity of symptoms** for misleading unprepared practitioners in the diagnosis of chronic Rickettsial infection? That **same diversity** could have contributed to the delay in recognising CFS.

¿

French authors (Giroud, Jadin, Legag) attribute those multiple aspects to a generalized micro-vascular invasion. They widely demonstrated the persistence of Rickettsiae in the vessels. The suggestion here is that the **well-known, well-documented** entity of Rickettsial disease, showing the **same symptoms** as the **newly arrived** CFS, might simply, partially or totally be caused by the **same agent.**

¿

The last, but **not the least** reason, is the **success rate** of the Rickettsia treatment, Tetracycline, applied on **CFS, Fibromyalgia etc.** patients. Dr Phillipe Bottero on 180 patients, Dr Peter Tarbleton on 300 patients and myself on a much larger number of patients, maintain an **84% - to 96%** recovery rate.

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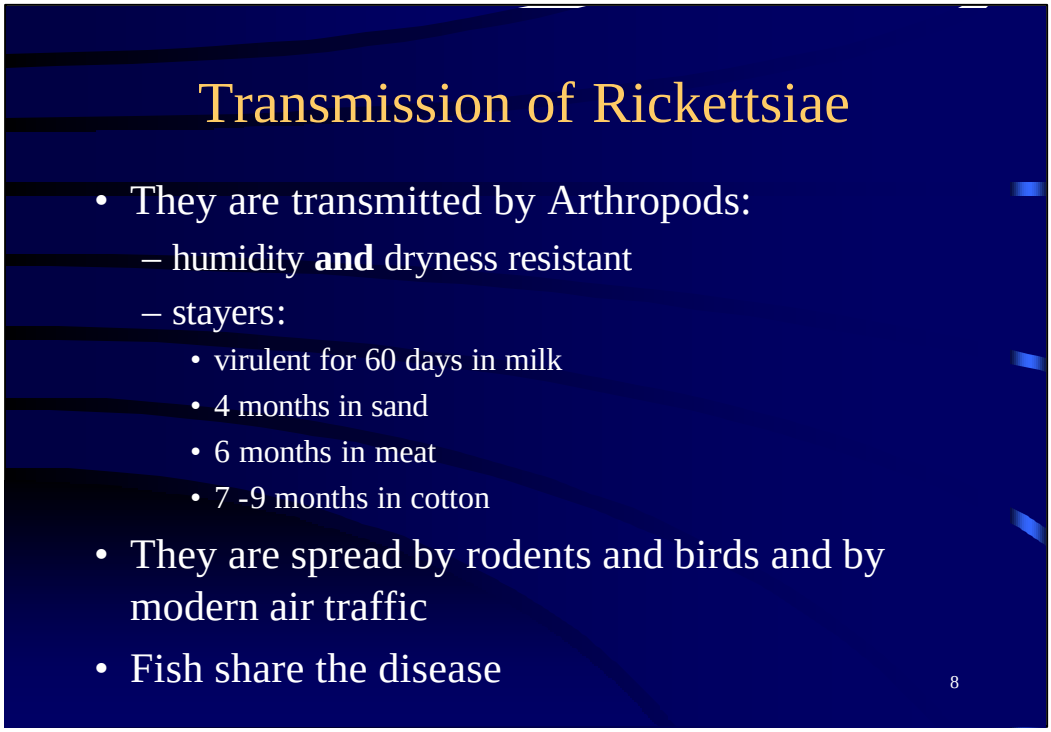

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Rickettsiae are transmitted by arthropodes, except for Q Fever, which does not really need vectors;

-they are resistant to humidity and to dryness

-they will stay virulent for 60 days in milk

-4 months in sand

-6 months in meat

-7 - 9 months in cotton

¿

They are spread by rodents and birds. Through the centuries, bird migration has been responsible for changing the geographical distribution of disease - but this is nothing compared to the effect of the explosion of these diseases due to the cocktail effect created by distribution through global air traffic.

Equally the transport of insects compared to the import and export of livestock - as in the case of the import of 10,000 parrots from Paraguay to Belgium when some 2,000 died, leaving the virus well and alive behind them, (identified by my father as Neo-Rickettsia Bedsonia).

This world distribution does not include Antarctica, where they do not survive.

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Fish also share this disease, as Erlichioses is, according to breeders, a common problem. ……

I would now like to discuss my patients and their diagnosis.

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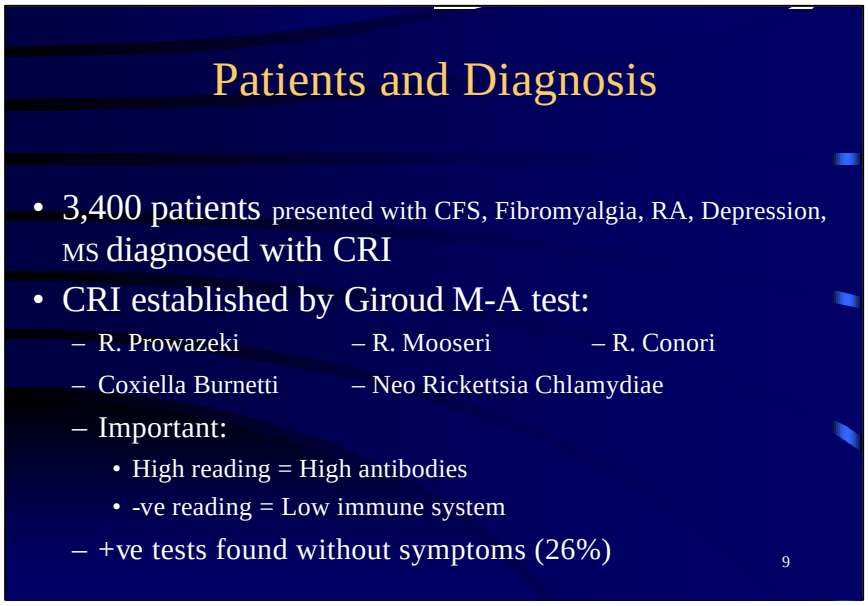

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3,400 patients presented with CFS, Fibromyalgia, RA, depression and MS have been diagnosed as suffering from **Chronic Rickettsial Infection** (CRI) after eliminating other diseases as a cause such as diabetes, cancer etc.

The majority of my patients report a flu-like infection, with often an elevated temperature and severe headaches. This lasts for a few days, disappears **or** reoccurs, and then leaves them with a chronic condition of CFS, Fibromyalgia etc. as mentioned above.

¿

Diagnosis of CRI is established by Giroud's Micro-Agglutination test against these **five strains of Rickettsiae.**

R. Prowazeki is the epidemic type of Typhus. **R. Mooseri** is endemic. **R. Conori** belongs to the Spotted Fever group.

C. Burnetti is well known as Q Fever: It has 2 phases, influenced by the host, Phase II is pathogenic. **Chlamydiae Q18** falls into the group of Neo-Rickettsia.

Important Points to note are that:

• A high reading means a high serological level of antibodies - a negative reading in endemic areas reflects the poverty of the immune system

• Agglutination happens or does not - therefore there is no possibility of personal interpretation. Test quality depends on Antigen quality

• Positive tests can be found in people who display no symptoms according to Giroud and Jadin; 26% are positive according to Drancourt.

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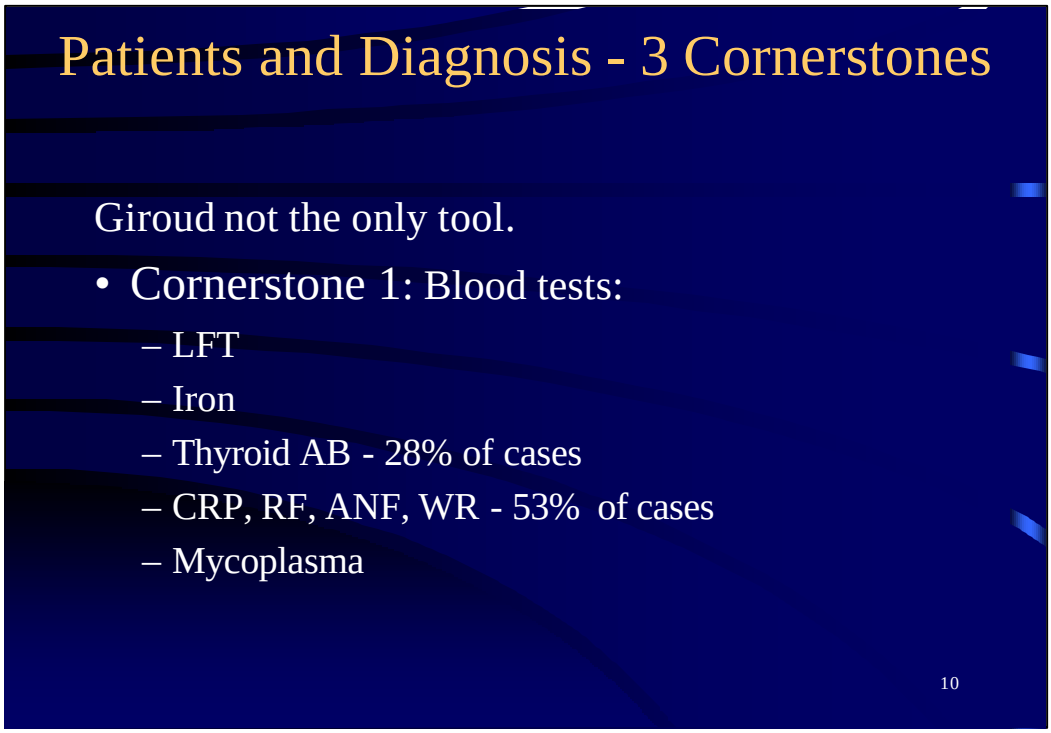

We can say that the diagnosis of Rickettsia rests on **3 cornerstones**.

¿

The Micro-Agglutination test of Giroud is not our only tool to establish the diagnosis of Rickettsial infections.

¿

Because we find the following blood tests most relevant:

• **LFT**: the hepatotoxicity of Rickettsiae has been reported as early as 1937 by Derrick in Q Fever, followed by many others - Giroud, Lenette, Legag, Brezina, Perron, Kelly, Raoult, etc. In these cases, Tetracyclines are improving or normalizing liver function.

• **Iron study** showed 50% of abnormalities corrected with Tetracyclines only and when necessary with a short course of iron supplement.

• **Thyroid AB** rather than **TFT**, although the **TFT** show abnormalities in 3% of patients, the Thyroid AB are elevated in **28%** of cases and improve or normalise **rapidly** with treatment.

• **CRP, RF, ANF, WR** positive in 53% of patients and also improved with treatment, and often normalised.

• **Mycoplasma** - I only started to research after the Manly conference in February 1998).

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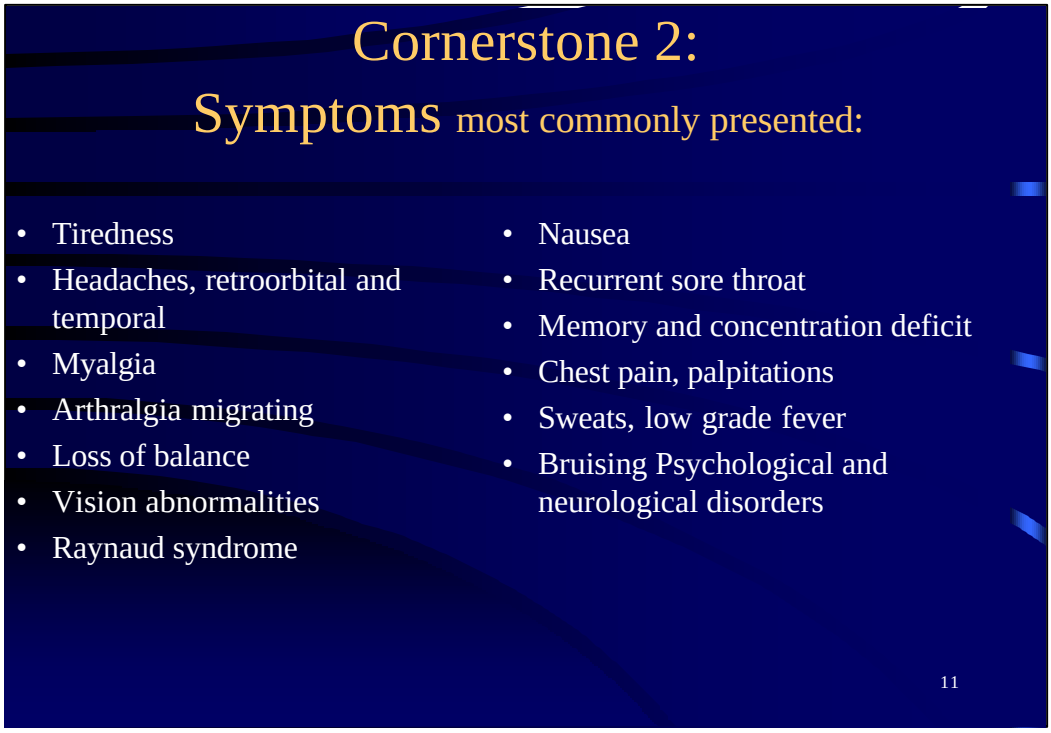

The **second cornerstone** is the Patient's symptoms. These are the symptoms most commonly exhibited.

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Headaches, retroorbital **and** temporal, seem to be worst after prolonged horizontal position or mental effort.

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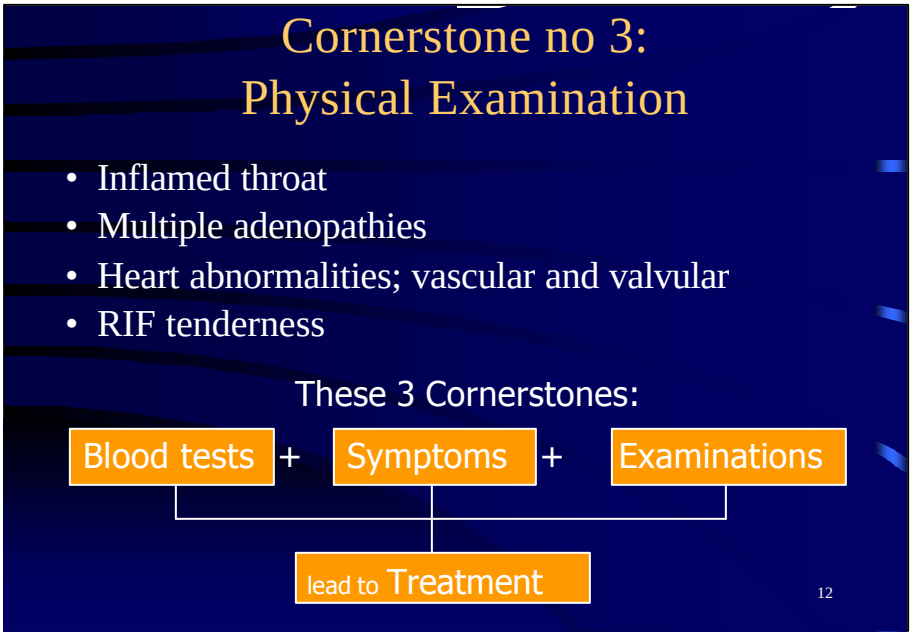

The Third corner stone is the Physical Examination, which is quite a constant guideline, because it often shows the following:

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- Inflamed throat,
- Multiple adenopathies,
- Heart abnormalities - vascular and valvular impact
- RIF tenderness.

¿

After establishing these **3 cornerstones**

-Blood tests

-Symptoms

-Physical examination

treatment is administered:

- Guided by our predecessors, (Giroud, Jadin, Legag etc.)
- Refined by our contemporaries, (Bottero and Raoult)
- and by my own daily, private lessons (each patient is one).

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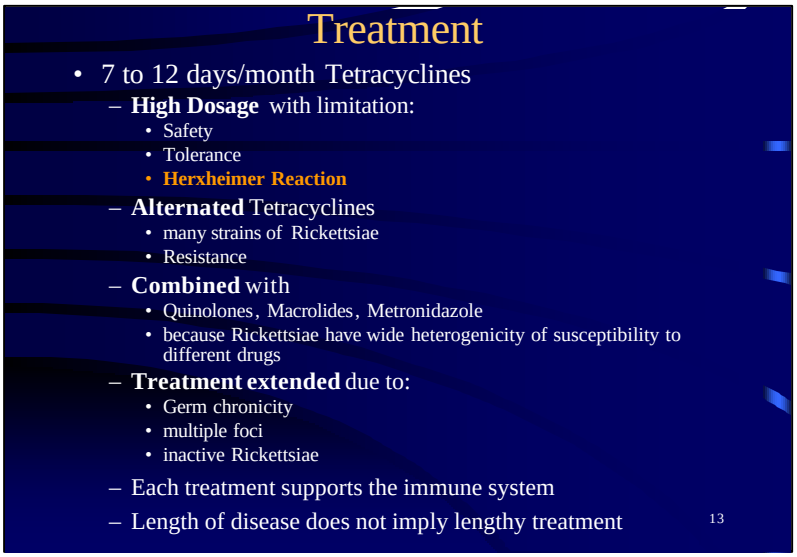

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The treatment consists of 7 to 12 days per month of a **specific Tetracycline.** The monthly treatment aims to follow the Rickettsial development in the cell.

¿

A **high dosage** is required with the limitation of:

•**Safety:** Goodman et al highlights irreversible hepatotoxicity in intravenous administration only. Our experience was that when liver functions were normal to start with, they stay normal. If they were abnormal, they will improve during treatment and generally return to normal. Cases of fatty acid depots (as shown by liver scan, before and after 6 months to 1 year of treatment) have disappeared (1 MS, 4 ME). This confirms the fact that Rickettsiae are more hepatotoxic than Tetracyclines.

•**Tolerance:** The gastric intolerance will be successfully prevented by using a gastric pump inhibitor during and if necessary before and after the administration of the Tetracyclines.The tolerance of the treatment is directly related to the **Herxheimer** reaction, which is a reactivation of old symptoms and/or exacerbation of present symptoms, that occurs on antibiotherapy. Its presence has a very important diagnosis and prognosis value. They might or might not be parallel to a serological reactivation. It will fade with the number of treatments received. When very severe, the **Herxheimer** is treated with Probenecid.

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The Tetracyclines are **alternated** because: A patient is frequently contaminated by many strains of Rickettsiae and different Rickettsiae have different sensitivity to different Tetracyclines, or combinations. A patient might build resistance to each Tetracycline. Patients show individual sensitivity to different Tetracyclines or combinations and there is very often a privileged reaction to a specific treatment.

¿

The Tetracyclines are **combined** with Quinolones, Macrolides or Metronidazole, because Rickettsiae present a wide heterogenicity of susceptibility to different drugs

¿

The treatment is often **long** due to: The chronicity of the germ, The multiple foci of Rickettsiae and The fact that Rickettsiae have a slow evolution and some foci are dormant, encapsulated and therefore protected from antibiotherapy. Only when they become active can they be treated.

¿

Each treatment will allow the immune system to produce and maintain a proper and efficient level of antibodies. This happens each time the antigen Rickettsiae are released from the cell to the blood stream while on antibiotherapy.

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The **length** of the disease should logically imply a **lengthy** treatment. In our experience, this point is **not always true**. Patients, ill for many years, may recover after a **few months** treatment.

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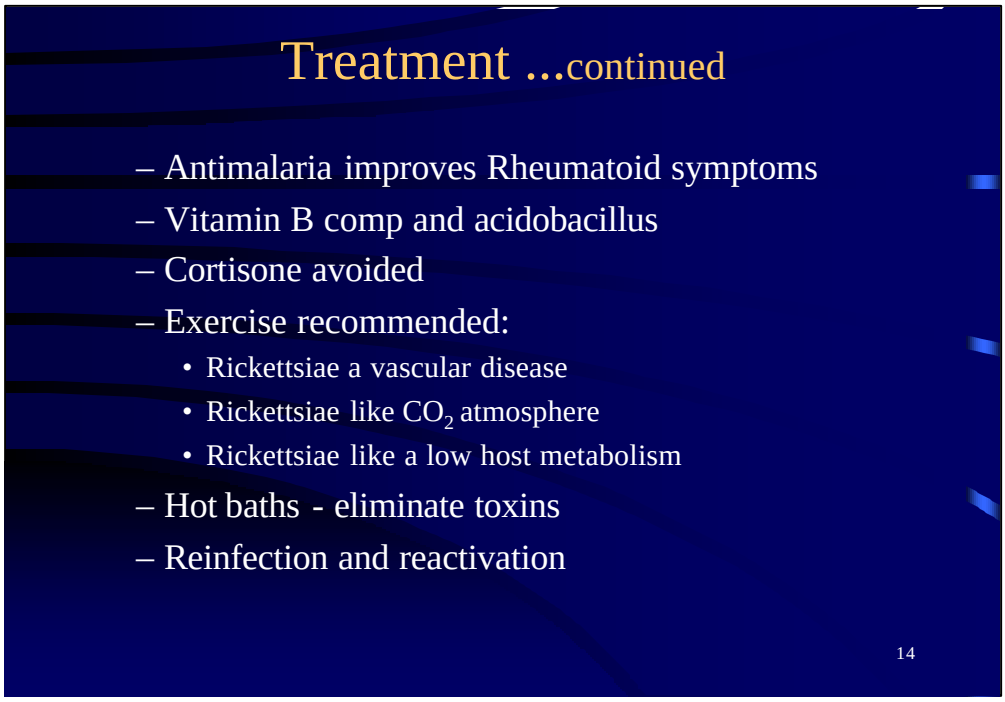

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Antimalaria has been found efficient to improve Rheumatoid symptoms and Rheumatoid biological findings (see patients' files).

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Adjuvants such as Vitamin B complex and acidobacillus are also used.

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Cortisone is avoided as much as possible as it is known to weaken the Immune System in general and also to reactivate the disease in experiments on guinea-pigs. Cortisone has been accused of interfering with the diagnosis of Rickettsia by masking the antibody level.

¿

Exercise is recommended, for 3 reasons:

- Rickettsial infection is a vascular disease and exercise, properly done, will improve the smooth peri-vascular muscle function, as well as develop our biggest muscle, the heart.
- The fact that strains of Rickettsiae grow better in vitro when maintained in a CO2 enriched atmosphere (34).
- The suggestion that Rickettsiae grow best when the metabolism of the host cell is low (38).

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Hot baths are important to eliminate toxins via the skin, produced by Rickettsiae antigens when liberated in the bloodstream by antibiotherapy.

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Reinfection may obviously occur. Reactivation (called so rather than relapse) may also happen due to the interaction of bacteria, virus, stress, pollution, etc. causing the Rickettsiae forms' change to active from dormant.

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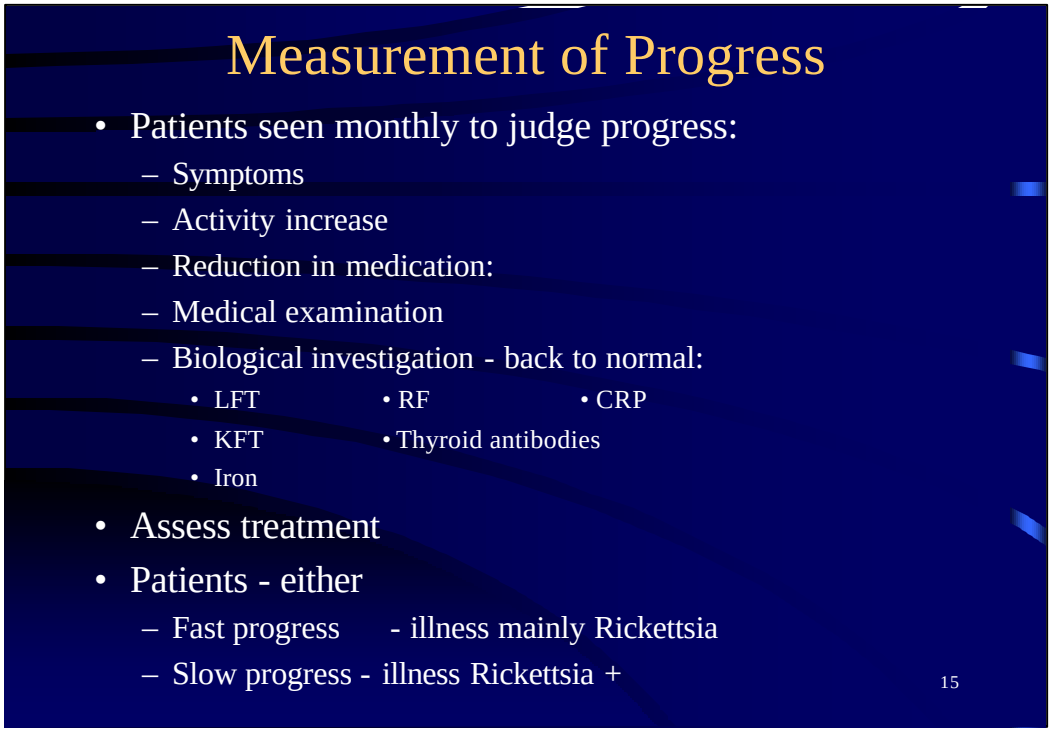

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Patients are seen monthly to judge progress on their symptoms, activity increase - from bedridden to back to exercise or back to work; - from being treated by painkillers, antidepressants, sedatives, cortisone to none. They are given a medical examination, and a biological investigation to measure the progress back to normal, or nearly so of: LFT - RF -, CRP -, ANF -, KFT -, Thyroid antibodies -, Iron.

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Based on the assessment, the treatment is prolonged or stopped. The time period can be 3 months to 2 years; and is 8 months on average. However, as previously mentioned, the length of treatment is not directly correlated to the length of illness:

. ¿

Therefore patients can be divided into 2 categories:

1. Fast progress - their illness was mainly Rickettsia
2. Slow progress - their illness was Rickettsia plus other factors.

NEXT SLIDE . ¿

Progress to health

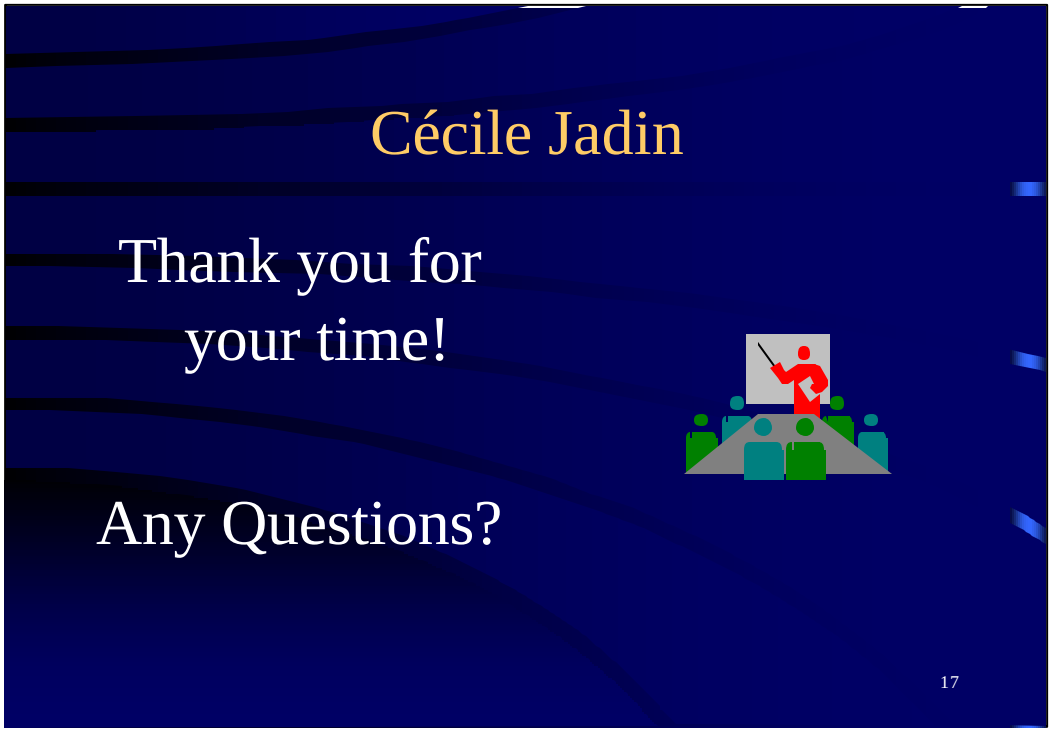
"La santé est comme une mongolfière : il faut parfois lâcher du lest"[16]

My suggestion is that by controlling Rickettsia, we can also assist in repairing the Immune system, either quickly or slowly….

Health is like a hot air balloon.

You have to get rid of excess burdens to keep it in the air. Rickettsia is the easy one to lose.

NEXT SLIDE ¿



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Ladies and gentlemen,

I thank you for your time and for this opportunity..

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I believe I have some time left for questions?

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DO NOT REMOVE:

After Questions, Leave the Podium!!

From Cellular Anoxemia caused by the presence of Rickettsiae to CFS

Padua, September 1999

CL Jadin MD MBBCh Johannesburg South Africa

Rickettsial infection was discovered in 1909, when Ricketts saw and described the germ that causes R M S F in man. Ricketts, as well as another scientist, Prowazek, contracted Typhus and died.

1. The epidemic forms of Rickettsiae were described by Zinsser in his classic book "Rats, Lice and History", in which he contends that soldiers have rarely won wars. Typhus and other infectious diseases have decided the outcome of more military campaigns than Caesar, Hannibal, Napoleon (2) and all generals in history. Depending on the outcome for each warring faction, either the epidemics were blamed for defeat, or the generals were credited with victory. It has contaminated an estimated 25 million Russians, causing 3 million deaths during the 1st World War.

2. Nowadays, following on from these historical memories, there are forms less virulent, evolving slowly, but able to induce vascular and neurological pathologies (52).

3. Rickettsiae are found in ticks, lice, fleas, mites, meat, milk, stools and dust. From the entry into the skin, the lungs, conjunctives, and the digestive mucosa, Rickettsiae spread via the bloodstream to infect vascular endothelium. These organisms grow and multiply by binary fission in and only in the cytoplasm of the host cell until the number of Rickettsiae is so great that the cell bursts, releasing hundreds of them. This invasion will impair or paralyse the vascular function, acting like a sponge between blood and organs. They will enlarge the endothelial cells of small vessels with partial or complete occlusion of the vascular lumen. They are known for long survival in various organs and lymphatic tissue. According to which vessel they invade, they might display an amazing constellation of diseases:

    - CFS, Fibromyalgia, where they cause a cellular anoxemia (41,47).
    - Cardio-vascular diseases (3,4,5,11,12,27,39,42,45).
    - Neurological diseases (from acute encephalitis to MS, epilepsy etc) (3,5,9,43).
    - Abdominal diseases (appendicitis, cœliac disease and others) (23).
    - Ocular diseases (uveitis, retinal angiopathy, optic nevritis sometimes a long time after a general infection) (3,29).
    - AutoImmune diseases (41,47).

    Rickettsiae release into the bloodstream angiotropic, endotoxins, producing inflammations, allergies and demyelinisation (H Perron).

4. Rickettsiae:

    - are resistant to humidity and to dryness
    - will stay virulent for;
        - 60 days in milk
        - 4 months in sand
        - 6 months in meat
        - 7 - 9 months in cotton (4).

    They are spread by rodents and birds. Through the centuries, bird migration has been responsible for changing the geographical distribution of disease (27) - but this is nothing compared to the effect of the explosion of these dis eases due to the cocktail effect created by distribution through global air traffic (26).

    Equally the transport of insects compared to the import and export of livestock - as in the case of the importation of 10,000 parrots from Paraguay to Belgium when some 2,000 died, leaving the virus well and alive behind them (27), (identified by JB Jadin as Neo-Rickettsia Bedsonia).

    This world distribution does not include Antarctica, where they do not survive.

    Fish also share this disease, as Erlichioses is, according to breeders, a common problem (31). They have been found in oysters by Deltreil.

5. 3,600 patients presented with CFS, Fibromyalgia, RA, depression and MS have been diagnosed as suffering from Chronic Rickettsial Infection (CRI) after eliminating other diseases as a cause (diabetes, cancer etc.).

    The majority of my patients report a flu-like infection, with often an elevated temperature and severe headaches. This lasts for a few days, disappears or reoccurs, and then leaves them with a chronic condition of CFS, Fibromyalgia etc. as mentioned above.

6. Diagnosis of CRI is established by Giroud's Micro -Agglutination test against five strains of Rickettsiae:
    - R. Prowazeki: the epidemic type of Typhus
    - R. Mooseri, which is endemic
    - R. Conori, which belongs to the spotted fever group
    - Coxiella Burnetti, which is well known as Q Fever. It has 2 phases; Phase II is pathogenic
    - Neo Rickettsia Chlamydiae which has an affinity for uteral mucosa, and will be the cause of many abortions.

<u>Important Points:</u>

a) A high reading means a high serological level of antibodies - a negative reading in endemic areas reflects the poverty of the immune system (24).
b) Agglutination happens or does not - therefore there is no possibility of personal interpretation. Test quality depends o n Antigen quality (3).
c) Positive tests can be found in people who display no symptoms (Giroud, Jadin (18); 26% according to Drancourt (39)).

7. However, the Micro -Agglutination test of Giroud is not our only tool to establish the diagnosis of Rickettsial infections. We find the following blood tests most relevant:

a) LFT: the hepatotoxicity of Rickettsiae has been reported as early as 1937 by Derrick in Q Fever (19, 29), followed by many others - Giroud, Lenette, Legag, Brezina, Perron, Kelly, Raoult, etc. In these cases, Tetracyclines are improving or normalising liver function (6).
b) Iron study (50% of abnormalities corrected with Tetracyclines only and when necessary with a short course of iron supplement).
c) Thyroid AB rather than TFT, although the TFT show abnormalities in 3% of patients, the thyroid AB are elevated in 28% of cases and improve or normalise rapidly with treatment.
d) CRP, RF, ANF, WR was positive in 53% of patients, (39) and also improved with treatment and often normalised.
e) Mycoplasma, first classified as a Rickettsia, is now considered to be an independent entity.

8. Patients' symptoms most commonly exhibited are:

- Tiredness (4,5)
- Headaches, retro-orbital and temporal, worst after prolonged horizontal position or mental effort (4).
- Myalgia (3)
- Arthralgia migrating (2,3,5)
- Loss of balance (29)
- Vision abnormalities (3,29)
- Raynaud syndrome (18)
- Nausea (8,9,18)
- Recurrent sore throat (23)
- Memory and concentration deficit (4).
- Chest pain, palpitations (8,12, 18)
- Sweats, low grade fever (4)
- Bruising (4)
- Psychological and neurological disorders(4,5,18,29,30)

9. We find quite a constant guideline in the physical examination, which often shows

- An inflamed throat
- Multiple adenopathies
- Heart abnormalities (vascular (4,12,30) and valvular impact (2, 39))
- RIF tenderness (chlamydiae 18 in appendix (23))

10. Treatment is administered:

- Guided by our predecessors, (Giroud, Jadin, Legag etc.)
- And by my own daily, private lessons (each patient is one).

The treatment consists of 7 to 12 days per month of a specific **Tetracycline**. The monthly treatment aims to follow the Rickettsial development in the cell.

a) A **high** dosage is required (4,5) with the limitation of:

- Safety (32) Goodman et al (33) highlights irreversible hepatotoxicity in intravenous administration only. Our experience was that when liver functions were normal to start with, they stay normal. If they were abnormal, they will improve during treatment and generally return to normal. Cases of fatty acid depots (as shown by liver scan, before and after 6

months to 1 year of treatment) have disappeared (1 MS, 4 ME). This confirms the fact that Rickettsiae are more hepatotoxic than Tetracyclines.

- Tolerance.
  The gastric intolerance will be successfully prevented by using a gastric pump inhibitor during and if necessary before and after the administration of the Tetracyclines.

  The tolerance of the treatment is directly related to the Herxheimer reaction (4, 6, 26, 37), which is a reactivation of old symptoms and/or exacerbation of present symptoms that occurs on antibiotherapy. Its presence has a very important diagnosis and prognosis value (4). They might or might not be parallel to a serological reactivation. It will fade with the number of treatments received. When very severe, the HR is treated with Probenecid.

b) The Tetracyclines are **alternated** because:

- A patient is frequently contaminated by many strains of Rickettsiae (5) and different Rickettsiae have different sensitivity to different Tetracyclines or combinations. (4).

- A patient might build resistance to each Tetracycline (4, 17).

- Patients show individual sensitivity to different Tetracyclines or combinations and there is very often a privileged reaction to a specific treatment (6).

c) The Tetracyclines are **combined** with Quinolones, Macrolides or Metronidazole (7), because Rickettsiae present a wide heterogenicity of susceptibility to different drugs (4).

d) The treatment is often **long** due to:

- The chronicity of the germ (4)

- The multiple foci of Rickettsiae (18)

- The fact that Rickettsiae have a slow evolution and some foci are dormant, encapsulated and therefore protected from antibiotherapy. Only when they become active can they be treated (5).

e) Each treatment will allow the immune system to produce and maintain a proper and efficient level of antibodies. This happens each time the antigen Rickettsiae are released from the cell to the blood stream while on antibiotherapy (Legag) (4).

f) The length of the disease should logically imply a lengthy treatment. In our experience, this point is not always true. Patients, ill for many years, may recover after a few months treatment.

g) **Antimalaria drugs** have been found efficient to improve Rheumatoid symptoms and Rheumatoid biological findings (see patients' files).

h) **Adjuvants** such as Vitamin B complex and acidobacillus are also used.

i) Cortisone is avoided as much as possible as it is known to weaken the Immune System in general (3) and also to reactivate the disease in experiments on guinea-pigs (39). Cortisone has been accused of interfering with the diagnosis of Rickettsia by masking the antibody level (4).

j) **Exercise** is recommended, for the following 3 reasons:

- Rickettsiae is a vascular disease and exercise, properly done, will improve the smooth peri-vascular muscle function, as well as develop our biggest muscle, the heart.
- The fact that strains of Rickettsiae grow better in vitro when maintained in a CO2 enriched atmosphere (34).
- The suggestion that Rickettsiae grow best when the metabolism of the host cell is low (38).

k) **Hot baths** are important to eliminate toxins via the skin, produced by Rickettsiae antigens when liberated in the bloodstream by antibiotherapy.

10. Reinfection may obviously occur. Reactivation (called so rather than relapse) may also happen due to the interaction of bacteria, virus, stress, pollution, etc. causing the Rickettsiae forms' to change from dormant to active (35).

11. Measurement of Progress - Patients are seen monthly to judge progress on:

a) Symptoms
b) Activity increase (From bedridden to back to exercise or back to work)
c) From being treated by painkillers, antidepressants, sedatives, cortisone to none
d) Medical examination
e) Biological investigation: from having:

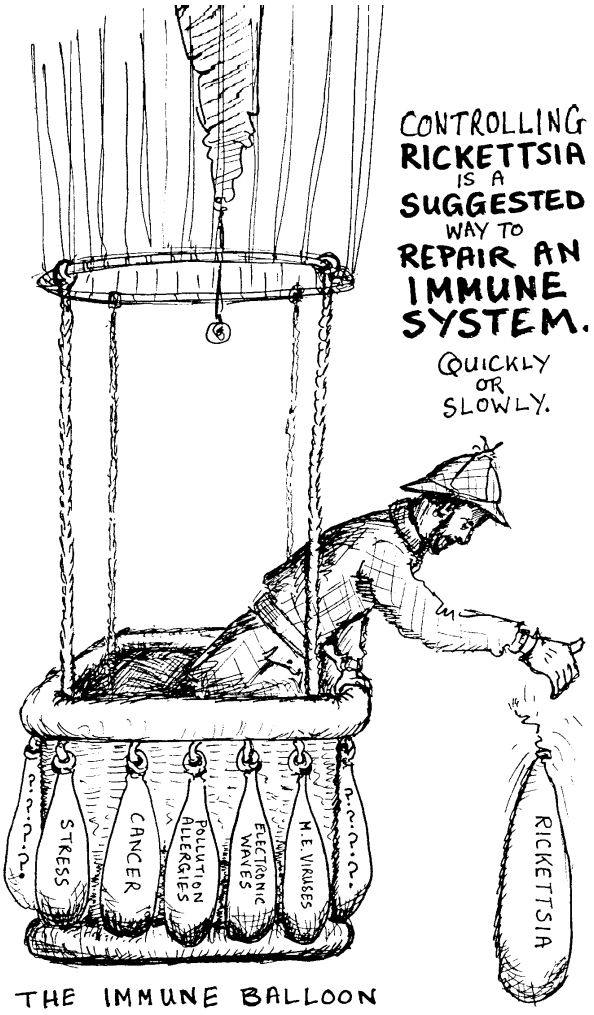
- ESR
- LFT
- RF raised
- CRP raised
- ANF raised
- KFT raised
- Thyroid antibodies raised
- Iron

} - Back to normal, or nearly so

12. Based on this assessment, the treatment is prolonged or stopped (3 months to 2 years: 8 months on average). However, as previously mentioned, the length of treatment is not directly correlated to the length of illness:

Therefore patients can be divided into 2 categories:

- Fast progress - their illness was mainly Rickettsia
- Slow progress - their illness was Rickettsia plus other factors (20).



"La salute e come un pallone d'aria calda; a volte bisogna rilasciare il peso"

Health is like a hot air balloon. You have to get rid of excess burdens to keep it in the air. Rickettsia is the easiest one to lose

Discussion

1) 12 years ago, one of my friends became unable to walk and was diagnosed as having ME. For 4 years I suggested the diagnosis of Rickettsial Infection, and therefore the Weil-Felix test was performed several times in South Africa, but the results were negative. My friend developed an acute appendicitis. After I removed her appendix, her serum was sent to Prof. JB Jadin in Belgium to test for Rickettsiae, and the result was positive. I treated her with Tetracyclines and 3 weeks later, from being in a wheelchair; she was riding her horse again. I was sceptical. But this case brought me 200 patients and the publicity surrounding an investigation of my methodology by the South African Medical Council brought me several thousand more. Thus I started to focus on the Rickettsial approach.

2) There are many reasons suggesting the infectious etiology and, more specifically, Rickettsial-like organisms of CFS. Amongst those reasons:

   a) Consider the following :

      - CFS was first reported in Incline, Nevada in 1984 (1) and developed into epidemic proportions.

      - Rocky Mountain Spotted Fever originated from the same place in 1916 (9,29).

      - The spirochete Borrelia Duttoni, first blamed for causing the recurrent Malgache fever described in the journals written by Drury in 1702 (24) in Madagascar, then by Scheltz in the Belgian Congo in 1933, by Palakov in Cape Town in 1944, by Heisch in Kenya in 1950.

      - Lyme Disease appeared (or reappeared?) more recently in Lyme, Connecticut in 1975 (Borrelia Burgdorferi) (25). As Lyme Disease is a new name for Malgache Fever, could CFS be a new name for Rickettsial Disease?

      All of the above highlights the life of a germ as an individual emerging and disappearing in a wave pattern epidemically and historically. Like us, germs have to adapt, producing new variations of themselves, (not new species), that may or may not survive on their own, with or without the help of another germ. This is circumstance-dependent, and these particular circumstances will never reoccur. Some of those variations will acquire specific and consistent characteristics.

      This is their 'civilisation'. We only see them when they succeed, and only then do new avenues of investigation open up, while others are abandoned.

   b) A link has been established between Florence Nightingale disease and CFS (21). The fact that she was working surrounded by lice, fleas and ticks, treating soldiers with wounds and with epidemic typhus during the Crimean war, could be a logical explanation as to why she was terribly tired during the last 2 decades of her life; and possibly has relevance to Gulf War illnesses (13).

   c) Lymphocyte studies conducted on sheep with tick-borne diseases (14), CFS patients (15,16) and patients with Q Fever endocarditis (11) are showing amazingly similar results.

d) Coincidentally, the new name suggested in the Lancet for CFS is PQFS (Post Q Fever Syndrome) in April 1996 edition (22).

e) As mentioned above, during the First World War an estimated 25 million Russians contracted Louse-borne epidemic typhus, resulting in 3 million deaths. Why not before or after? It could suggest that the stress factor reactivates the virulence of Typhus Prowazeki (2, 3, 9). In the medical history of CFS patients, stress has often been described as the start of the illness.

f) The symptoms displayed by CFS, Fibromyalgia, RA, and even neurological patients as MS, show the same diversity of symptoms as Rickettsial patients. How many scientists blamed the diversity of symptoms for misleading unprepared practitioners in the diagnosis of chronic Rickettsial infection (30)? That same diversity could have contributed to the delay in recognising CFS. French authors (Giroud, Jadin, Legag) attribute those multiple aspects to a generalised micro-vascular invasion. They widely demonstrated the persistence of Rickettsiae in the vessels (4), (18). The suggestion here is that the well-known, well-documented entity of Rickettsial disease, showing the same symptoms as the newly arrived CFS, might simply, partially or totally be caused by the same agent.

g) The last, but not the least reason, is the success rate of the Rickettsial treatment, Tetracycline, applied on CFS, Fibromyalgia etc. patients. Dr Phillipe Bottero on 100 patients, Dr Peter Tableton on 300 patients (17) and myself on a much larger number of patients, maintain an 84% to 96% recovery rate.

CFS - Rickettsial Infection: Sources of References

Presented by Dr. Cécile Jadin, Johannesburg, South Africa 1999

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From Cellular Anoxemia caused by the presence of Rickettsiae to CFS

__CL Jadin

MD MBBCh

Johannesburg

Padua September 1999

From Cellular Anoxemia caused by the presence of Rickettsiae to CFS

Topics

- Rickettsial Infection
- Distribution
- Material and Methods
- Treatment
- Measurement of Progress
- Discussion

2

Rickettsial Infection

- Discovered in 1909
- Initially epidemic forms
- Latterly slow evolving forms
- Distribution
- Vascular pathology
- A constellation of Diseases

3

Rickettsial infection was discovered in 1909, when Ricketts saw and described the germ that causes R M S F in man. Ricketts, as well as another scientist, Prowazek, contracted Typhus and died.

1. The epidemic forms of Rickettsiae were described by Zinsser in his classic book "Rats, Lice and History", in which he contends that soldiers have rarely won wars. Typhus and other infectious diseases have decided the outcome of more military campaigns than Caesar, Hannibal, Napoleon and all generals in history. Depending on the outcome for each warring faction, either the epidemics were blamed for defeat, or the generals were credited with victory. It has contaminated an estimated 25 million Russians, causing 3 million deaths during the 1st World War.
2. Nowadays, following on from these historical memories, there are forms less virulent, evolving slowly, but able to induce vascular and neurological pathologies.
3. Rickettsiae are found in ticks, lice, fleas, mites, meat, milk, stools and dust. From the entry into the skin, the lungs, conjunctives, and the digestive mucosa, Rickettsiae spread via the bloodstream to infect vascular endothelium. These organisms grow and multiply by binary fission in and only in the cytoplasm of the host cell until the number of Rickettsiae is so great that the cell bursts, releasing hundreds of them. This invasion will impair or paralyse the vascular function, acting like a sponge between blood and organs. They will enlarge the endothelial cells of small vessels with partial or complete occlusion of the vascular lumen. They are known for long survival in various organs and lymphatic tissue. According to which vessel they invade, they might display an amazing constellation of diseases:
    - CFS, Fibromyalgia, where they cause a cellular anoxemia
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    - AutoImmune diseases

    Rickettsiae release into the bloodstream, angiotropic endotoxins, producing inflammations, allergies and demyelinisation (H Perron).

Distribution of Rickettsiae

- They are:
  - humidity **and** dryness resistant
  - stayers:
    - virulent for 60 days in milk
    - 4 months in sand
    - 6 months in meat
    - 7 -9 months in cotton
  - spread by rodents and birds and by modern air traffic
- Fish and oysters share the disease

4

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Material and Methods

- 3,600 patients presented with CFS, Fibromyalgia, RA, Depression, MS diagnosed with CRI
- CRI established by Giroud M-A test:
  - R. Prowazeki – R. Mooseri – R. Conori
  - Coxiella Burnetti – Neo Rickettsia Chlamydiae
  - Important:
    - High reading = High antibodies
    - -ve reading = Low immune system
  - +ve tests found without symptoms (26%)

5

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Positive tests can be found in people who display no symptoms (Giroud, Jadin; 26% according to Drancourt).

Material and Methods (Biology)

Giroud not the only tool.

Blood tests:

- LFT
- Iron
- Thyroid AB - 28% of cases
- CRP, RF, ANF, WR - 53% of cases
- Mycoplasma

6

However, the Micro-Agglutination test of Giroud is not our only tool to establish the diagnosis of Rickettsial infections. We find the following blood tests most relevant:

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Mycoplasma, first classified as Rickettsia, is now considered to be an independent entity.

Material and Methods (Symptoms)

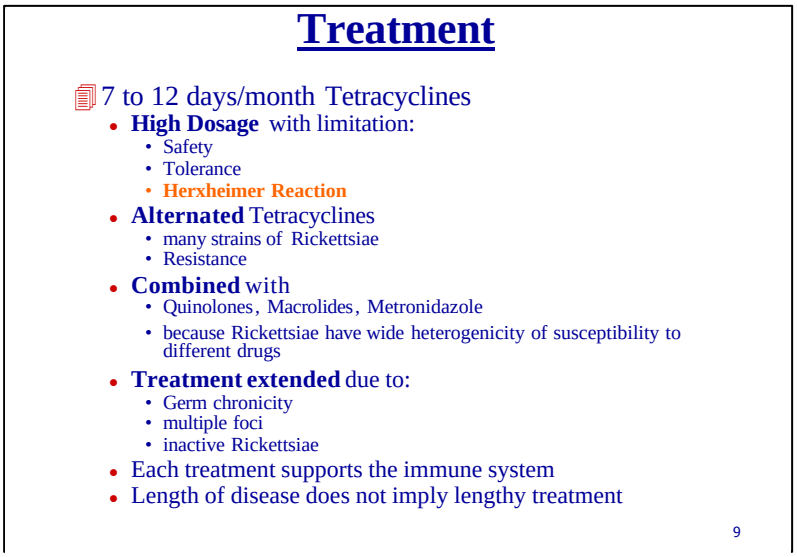
- Tiredness
- Headaches, retroorbital and temporal
- Myalgia
- Arthralgia migrating
- Loss of balance
- Vision abnormalities
- Raynaud syndrome
- Nausea
- Recurrent sore throat
- Memory and concentration deficit
- Chest pain, palpitations
- Sweats, low grade fever
- Bruising
- Psychological and neurological disorders

7

Material and Methods (Physical Examination)

- Inflamed throat
- Multiple adenopathies
- Heart abnormalities; vascular and valvular
- RIF tenderness - Chlamydiae in Appendix

8



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A **high dosage** is required with the limitation of:

•**Safety:** Goodman et al highlights irreversible hepatotoxicity in intravenous administration only. Our experience was that when liver functions were normal to start with, they stay normal. If they were abnormal, they will improve during treatment and generally return to normal. Cases of fatty acid depots (as shown by liver scan, before and after 6 months to 1 year of treatment) have disappeared (1 MS, 4 ME). This confirms the fact that Rickettsiae are more hepatotoxic than Tetracyclines.

•**Tolerance:** The gastric intolerance will be successfully prevented by using a gastric pump inhibitor during and if necessary before and after the administration of the Tetracyclines.The tolerance of the treatment is directly related to the **Herxheimer** reaction, which is a reactivation of old symptoms and/or exacerbation of present symptoms, that occurs on antibiotherapy. Its presence has a very important diagnosis and prognosis value. They might or might not be parallel to a serological reactivation. It will fade with the number of treatments received. When very severe, the **Herxheimer** is treated with Probenecid.

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The Tetracyclines are **alternated** because: A patient is frequently contaminated by many strains of Rickettsiae and different Rickettsiae have different sensitivity to different Tetracyclines, or combinations. A patient might build resistance to each Tetracycline. Patients show individual sensitivity to different Tetracyclines or combinations and there is very often a privileged reaction to a specific treatment.

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The Tetracyclines are **combined** with Quinolones, Macrolides or Metronidazole, because Rickettsiae present a wide heterogenicity of susceptibility to different drugs

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The treatment is often **long** due to: The chronicity of the germ, The multiple foci of Rickettsiae and The fact that Rickettsiae have a slow evolution and some foci are dormant, encapsulated and therefore protected from antibiotherapy. Only when they become active can they be treated.

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Each treatment will allow the immune system to produce and maintain a proper and efficient level of antibodies. This happens each time the antigen Rickettsiae are released from the cell to the blood stream while on antibiotherapy.

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The **length** of the disease should logically imply a **lengthy** treatment. In our experience, this point is **not always true**. Patients, ill for many years, may recover after a **few months** treatment.

Next Page ¿ .

Treatment ...continued

- Antimalaria improves Rheumatoid symptoms
- Vitamin B comp and acidobacillus
- Cortisone avoided
- Exercise recommended:
  - Rickettsiae a vascular disease
  - Rickettsiae like CO_2 atmosphere
  - Rickettsiae like a low host metabolism
- Hot baths - eliminate toxins
- Reinfection and reactivation

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Antimalaria has been found efficient to improve Rheumatoid symptoms and Rheumatoid biological findings (see patients' files).

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Adjuvants such as Vitamin B complex and acidobacillus are also used.

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Cortisone is avoided as much as possible as it is known to weaken the Immune System in general and also to reactivate the disease in experiments on guinea-pigs. Cortisone has been accused of interfering with the diagnosis of Rickettsia by masking the antibody level.

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Exercise is recommended, for 3 reasons:

- Rickettsial infection is a vascular disease and exercise, properly done, will improve the smooth peri-vascular muscle function, as well as develop our biggest muscle, the heart.
- The fact that strains of Rickettsiae grow better in vitro when maintained in a CO2 enriched atmosphere (34).
- The suggestion that Rickettsiae grow best when the metabolism of the host cell is low (38).

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Hot baths are important to eliminate toxins via the skin, produced by Rickettsiae antigens when liberated in the bloodstream by antibiotherapy.

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Reinfection may obviously occur. Reactivation (called so rather than relapse) may also happen due to the interaction of bacteria, virus, stress, pollution, etc. causing the Rickettsiae forms' change to active from dormant.

NEXT SLIDE ¿

Measurement of Progress

- Patients seen monthly to judge progress:
  - Symptoms
  - Activity increase
  - Reduction in medication:
  - Medical examination
  - Biological investigation - back to normal:
    - LFT • RF • CRP
    - KFT • Thyroid antibodies
    - Iron
- Assess treatment
- Patients - either
  - Fast progress - illness mainly Rickettsia
  - Slow progress - illness Rickettsia +

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Patients are seen monthly to judge progress on their symptoms, activity increase - from bedridden to back to exercise or back to work; - from being treated by painkillers, antidepressants, sedatives, cortisone to none. They are given a medical examination, and a biological investigation to measure the progress back to normal, or nearly so of: LFT - RF -, CRP -, ANF -, KFT -, Thyroid antibodies -, Iron.

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Based on the assessment, the treatment is prolonged or stopped. The time period can be 3 months to 2 years; and is 8 months on average. However, as previously mentioned, the length of treatment is not directly correlated to the length of illness:

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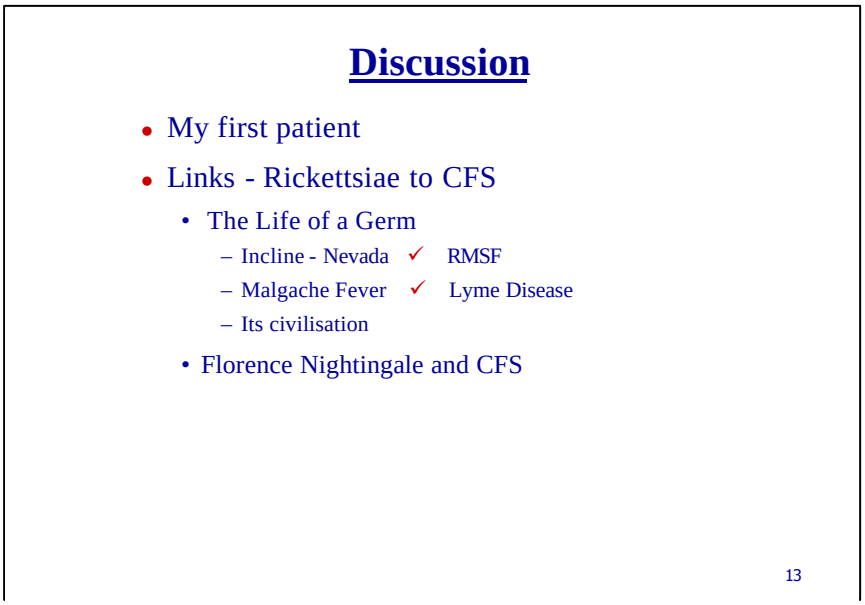
Therefore patients can be divided into 2 categories:

1. Fast progress - their illness was mainly Rickettsia
2. Slow progress - their illness was Rickettsia plus other factors.

NEXT SLIDE . ¿

"La salute e come un pallone d'aria calda; a volte bisogna rilasciare il peso"



12 years ago, one of my friends became unable to walk and was diagnosed as having ME. For 4 years I suggested the diagnosis of Rickettsial Infection, and therefore the Weil-Felix test was performed several times in South Africa, but the results were negative. My friend developed an acute appendicitis. After I removed her appendix, her serum was sent to Prof. JB Jadin in Belgium to test for Rickettsiae, and the result was positive. I treated her with Tetracyclines and 3 weeks later, from being in a wheelchair; she was riding her horse again. I was sceptical. But this case brought me 200 patients and the publicity surrounding an investigation of my methodology by the South African Medical Council brought me several thousand more. Thus I started to focus on the Rickettsial approach.

There are many reasons suggesting the infectious etiology and, more specifically, Rickettsial-like organisms of CFS. Amongst those reasons:

•CFS was first reported in Incline, Nevada in 1984 and developed into epidemic proportions.

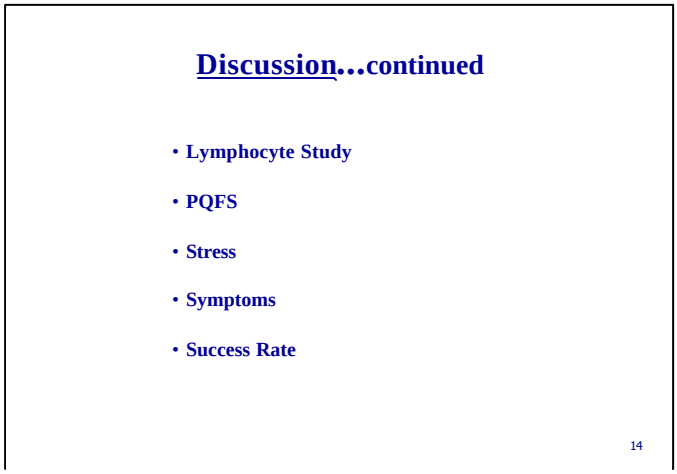
•Rocky Mountain Spotted Fever originated from the same place in 1916.The spirochete Borrelia Duttoni, first blamed for causing the recurrent Malgache fever described in the journals written by Drury in 1702 in Madagascar, then by Scheltz in the Belgian Congo in 1933, by Palakov in Cape Town in 1944, by Heisch in Kenya in 1950.

•Lyme Disease appeared more recently in Lyme, Connecticut in 1975 (Borrelia Burgdorferi). As Lyme Disease is a new name for Malgache Fever, could CFS be a new name for Rickettsial Disease?

All of the above highlights the life of a germ as an individual emerging and disappearing in a wave pattern epidemically and historically. Like us, germs have to adapt, producing new variations of themselves, (not new species), that may or may not survive on their own, with or without the help of another germ. This is circumstance-dependent, and these particular circumstances will never reoccur. Some of those variations will acquire specific and consistent characteristics.

This is their 'civilisation'. We only see them when they succeed, and only then do new avenues of investigation open up, while others are abandoned.

A link has been established between Florence Nightingale disease and CFS.The fact that she was working surrounded by lice, fleas and ticks, treating soldiers with wounds and with epidemic typhus during the Crimean war, could be a logical explanation as to why she was terribly tired during the last 2 decades of her life; and possibly has relevance to Gulf War



Lymphocyte studies conducted on sheep with tick-borne diseases,CFS patients and patients with Q Fever endocarditis are showing amazingly similar results.

Coincidentally, the new name suggested in the Lancet for CFS is PQFS (Post Q Fever Syndrome) in April 1996 edition

As mentioned above, during the First World War an estimated 25 million Russians contracted Louse-borne epidemic typhus, resulting in 3 million deaths. Why not before or after? It could suggest that the stress factor reactivates the virulence of Typhus Prowazeki. In the medical history of CFS patients, stress has often been described as the start of the illness.

The symptoms displayed by CFS, Fibromyalgia, RA, and even neurological patients as MS, show the same diversity of symptoms as Rickettsial patients. How many scientists blamed the diversity of symptoms for misleading unprepared practitioners in the diagnosis of chronic Rickettsial infection? That same diversity could have contributed to the delay in recognising CFS. French authors (Giroud, Jadin, Legag) attribute those multiple aspects to a generalised micro-vascular invasion. They widely demonstrated the persistence of Rickettsiae in the vessels. The suggestion here is that the well-known, well-documented entity of Rickettsial disease, showing the same symptoms as the newly arrived CFS, might simply, partially or totally be caused by the same agent.

The last, but not the least reason, is the success rate of the Rickettsial treatment, Tetracycline, applied on CFS, Fibromyalgia etc. patients. Dr Phillipe Bottero on 100 patients, Dr Peter Tableton on 300 patients and myself on a much larger number of patients, maintain an 84% to 96% recovery rate.