

## 26 Appendix: Jarisch-Herxheimer Reaction

Jarisch-Herxheimer reaction (JHR) is an acute febrile reaction which may complicate the initiation of an effective treatment against infections due to intracellular micro-organisms<sup>2221</sup>. JHR can also be expressed as the worsening of symptoms or the appearance of new symptoms. JHR is traditionally associated with antibiotics and antivirals but may appear from anything that impacts the immune response or infections. If the action results in a pathogen being killed which releases chemicals that cause symptoms, a JHR may occur. For example, the use of NAC (Acetylcysteine) or EDTA has been reported to produce a worsening of symptoms or JHR. These two anti-biofilm compounds break down biofilms exposing pathogens to the immune system; they do not directing kill any pathogens.

Antibiotics increase the bioavailability of endotoxin from Gram-negative bacteria<sup>2222</sup>. The increase may be up to 20 fold more<sup>2223</sup>. It should be noted that the Gram-negative bacteria producing the endotoxins may be microflora.

A worsening of symptoms from something is helping is almost a trademark for CFS. Unfortunately, in many CFS communities there is concerns that any reaction may be *something* else happening; the conclusion of many patients is to “do no harm”, thus avoiding JHR totally for fear (or not wishing to go through the misery of a JHR).

JHR can include:

- ❖ Acute rise in temperature, tachycardia, tachypnea, hypoxia, hypotension<sup>2224</sup>
- ❖ Associated with Gram-positive, Gram-negative or fungal organisms<sup>2225</sup>
- ❖ Associated with high titers<sup>2226</sup>, high concentrations of TNF, IL-6, and IL-8<sup>2227 2228</sup> up to 8 fold increase over pre-antibiotic levels<sup>2229</sup>.
- ❖ Can last for hours and reoccur<sup>2230</sup>
- ❖ Caused by release of endotoxin-like material and cytokine elevation<sup>2231 2232 2233</sup>.
- ❖ Delusional behavior, fever, rigors, tachycardia and hypoxia<sup>2234 2235 2236</sup>, complex visual and auditory hallucinations<sup>2237</sup>

<sup>2221</sup> <http://www.ncbi.nlm.nih.gov/pubmed/9733392> (1998)

<sup>2222</sup> <http://www.ncbi.nlm.nih.gov/pubmed/7619330> (1995)

<sup>2223</sup> <http://www.ncbi.nlm.nih.gov/pubmed/1445982> (1992)

<sup>2224</sup> <http://www.ncbi.nlm.nih.gov/pubmed/19040755> (2008 \*)

<sup>2225</sup> <http://www.ncbi.nlm.nih.gov/pubmed/12122518> (2002)

<sup>2226</sup> <http://www.ncbi.nlm.nih.gov/pubmed/20825309> (2010)

<sup>2227</sup> <http://www.ncbi.nlm.nih.gov/pubmed/16455348> (2006)

<sup>2228</sup> <http://www.ncbi.nlm.nih.gov/pubmed/9093599> (1997 \*)

<sup>2229</sup> <http://www.ncbi.nlm.nih.gov/pubmed/1569394> (1992)

<sup>2230</sup> <http://www.ncbi.nlm.nih.gov/pubmed/19040755> (2008)

<sup>2231</sup> <http://www.ncbi.nlm.nih.gov/pubmed/15896248> (2005)

<sup>2232</sup> <http://www.ncbi.nlm.nih.gov/pubmed/9733392> (1998)

<sup>2233</sup> <http://www.ncbi.nlm.nih.gov/pubmed/9511083> (1998)

<sup>2234</sup> <http://www.ncbi.nlm.nih.gov/pubmed/22707695> (2012)

## Chronic Fatigue Syndrome Remissions – Facts 2012

- ❖ Developed disseminated intravascular coagulation<sup>2238</sup>.
- ❖ Feeling cold with worsening headache and chills<sup>2239</sup>.
- ❖ Improves with antipyretic and anti-inflammatory agents<sup>2240</sup>.
- ❖ Improves with dexamethasone treatment<sup>2241</sup>.
- ❖ Improves with olanzapine treatment<sup>2242</sup>.
- ❖ Improves with pre-treatment with anti-tumour necrosis factor antibodies<sup>2243 2244 2245</sup>.
- ❖ Incidence varies by antibiotic and dosage<sup>2246</sup>.
- ❖ Increase of IgG and IgM by 4x<sup>2247</sup>.
- ❖ May show up as worst MRIs<sup>2248 2249</sup>.
- ❖ No standard for determining a JHR.
- ❖ Not uncommon to confuse drug allergy with JHR<sup>2250</sup>.
- ❖ Pentoxifylline does not have any effect<sup>2251</sup>.
- ❖ Potentially lethal<sup>2252</sup>.
- ❖ Rates seen between 0.8%<sup>2253</sup> - 1.4%<sup>2254</sup> - 9%<sup>2255</sup> - 15%<sup>2256</sup> - 32%<sup>2257</sup> - 34%<sup>2258</sup> - 40%<sup>2259</sup> - 43%<sup>2260 2261</sup> - 47%<sup>2262</sup> - 54%<sup>2263</sup>
- ❖ Rise in body temperature (1 C)<sup>2264</sup>

---

2235 <http://www.ncbi.nlm.nih.gov/pubmed/20664452> (2010)

2236 <http://www.ncbi.nlm.nih.gov/pubmed/9610974> (1998)

2237 <http://www.ncbi.nlm.nih.gov/pubmed/12604286> (2002)

2238 <http://www.ncbi.nlm.nih.gov/pubmed/12182387> (2002)

2239 <http://www.ncbi.nlm.nih.gov/pubmed/16288069> (2005)

2240 <http://www.ncbi.nlm.nih.gov/pubmed/16288069> (2005)

2241 <http://www.ncbi.nlm.nih.gov/pubmed/18302644> (2008)

2242 <http://www.ncbi.nlm.nih.gov/pubmed/18635694> (2009)

2243 <http://www.ncbi.nlm.nih.gov/pubmed/15896248> (2005)

2244 <http://www.ncbi.nlm.nih.gov/pubmed/9093599> (1997 \*)

2245 <http://www.ncbi.nlm.nih.gov/pubmed/8663853> (1998 \*)

2246 <http://www.ncbi.nlm.nih.gov/pubmed/7784810> (1995)

2247 <http://www.ncbi.nlm.nih.gov/pubmed/8508816> (1993)

2248 <http://www.ncbi.nlm.nih.gov/pubmed/21042805> (2011)

2249 <http://www.ncbi.nlm.nih.gov/pubmed/18302644> (2008)

2250 <http://www.ncbi.nlm.nih.gov/pubmed/16288069> (2005)

2251 <http://www.ncbi.nlm.nih.gov/pubmed/8769625> (1996)

2252 <http://www.ncbi.nlm.nih.gov/pubmed/21803390> (2011)

2253 <http://www.ncbi.nlm.nih.gov/pubmed/19411042> (2009)

2254 <http://www.ncbi.nlm.nih.gov/pubmed/22607395> (2012)

2255 <http://www.ncbi.nlm.nih.gov/pubmed/21297087> (2010)

2256 <http://www.ncbi.nlm.nih.gov/pubmed/17412541> (2007)

2257 <http://www.ncbi.nlm.nih.gov/pubmed/12380884> (2002)

2258 <http://www.ncbi.nlm.nih.gov/pubmed/20825309> (2010)

2259 <http://www.ncbi.nlm.nih.gov/pubmed/9794683> (1998)

2260 <http://www.ncbi.nlm.nih.gov/pubmed/16222003> (2005)

2261 <http://www.ncbi.nlm.nih.gov/pubmed/8511813> (1993)

2262 <http://www.ncbi.nlm.nih.gov/pubmed/7784810> (1995)

2263 <http://www.ncbi.nlm.nih.gov/pubmed/9455520> (1998 \*)

2264 <http://www.ncbi.nlm.nih.gov/pubmed/1569394> (1992)

- ❖ Seen with tetracyclines and penicillin<sup>2265</sup>, ciprofloxacin<sup>2266</sup>, sulfamethoxazole-trimethoprim<sup>2267</sup>.
- ❖ Tachycardia, hypotension, and thrombocytopenia, elevated serum cardiac troponin<sup>2268</sup>

**PO:** I will often develop headaches (never have otherwise), dizziness, sore throat and cough from effective antibiotics, antivirals or probiotics – as well as much increased malaise.

## 26.1 Understanding and Managing JHR is Essential

I will get on a soap box here because I often have seen non-compliance with patients stopping a protocol due to JHR. A well controlled JHR results in patients becoming positive about the protocol and wishing to continue with the protocol. A few personal examples may help:

- ❖ In 2001, I was sick 24 hrs a day from starting doxycycline @ 3 times/day, but by day 5, I was out at the herx about 1 hr before the next dosage was due. Getting a few hours each day being productive and feeling better convinced me that antibiotics was the right path.
- ❖ In 2012, when I started E.Coli Nissle 1917, each day had a worst herx then the prior day. I was starting to seriously consider stopping. On day 3-4, I found my eyes tearing and suddenly realized that it had eliminated one dominant symptom – dry eye (and dry mouth disappeared a few days later). It would be two weeks before the JHR had reduced to give me 1 hr per day of productive time.
- ❖ Later in 2012 when I increased E.Coli Nissle 1917, I initially took it just before bed and had a horrible night including waking with a throat that felt like the Sahara at 3am. The next day I took it at noon and noted the symptoms that resulted and when they occurred. Nissle herx was very different then antibiotic herx. The result was to shift it until about 5 pm so that I would be awake to handle the intense dry mouth that showed up about an hour later.

There is no medical need to keep someone in a JHR continuously. A JHR, even for just 1 hr, is a positive sign suggesting that the treatment is appropriate. Jadin's practice of pulsing antibiotics likely help with patient compliance – they have a short sprint of JHR to endure and then some time to see their impact. I have seen this psychological benefit with someone doing a 5 day pulse of EDTA, by day 3 they were horrible but knowing that it would be just two more days kept them taking it.

## 26.2 Managing JHR

In this appendix I will describe how I often manage JHR to reduce its impact on my day. Whenever I start an antibiotic, I avoid all potentators for 72 hours prior to start. I will attempt to find out the half-life of the antibiotic (time until the concentration is 50% less) and when the peak concentration after taking occurs (TMax). Peak concentration is generally delayed if taken with food. I use this information to schedule peak antibiotic concentration just *after* bed-time and then hope that I will sleep through most of any JHR. I will time things to have less JHR occurring during my waking day.

<sup>2265</sup> <http://www.ncbi.nlm.nih.gov/pubmed/21803390> (2011)

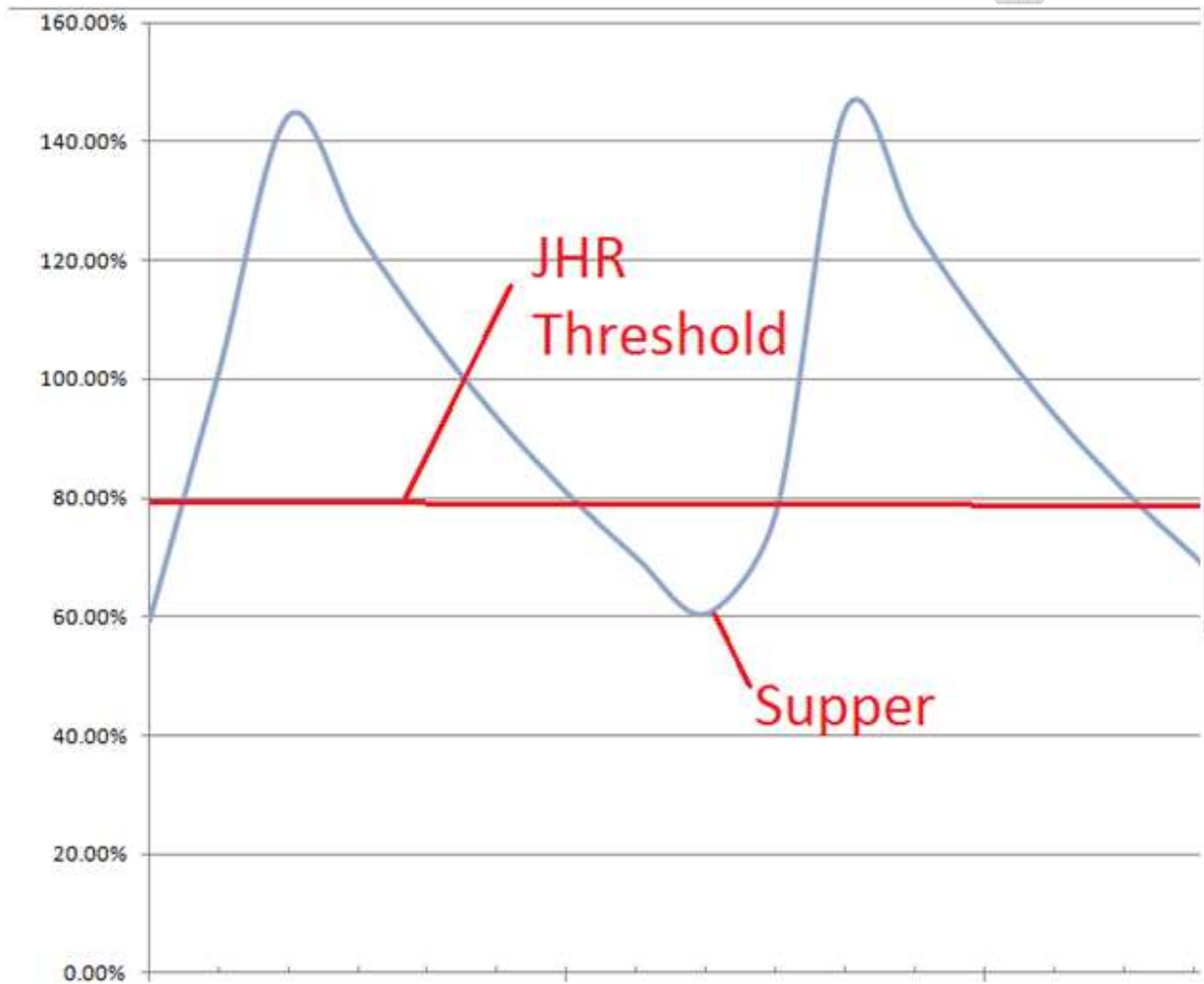
<sup>2266</sup> <http://www.ncbi.nlm.nih.gov/pubmed/12182387> (2002)

<sup>2267</sup> <http://www.ncbi.nlm.nih.gov/pubmed/11721494> (2001)

<sup>2268</sup> <http://www.ncbi.nlm.nih.gov/pubmed/22567483> (2011 \*)

### 26.3 Basic Antibiotic JHR Curve

The curve below illustrates the concentration of an antibiotic over time. The first antibiotic is taken without food and results in TMax at 2 hrs, the second one with food with TMax at 4 hrs. At some concentration level, JHR is produced (shown as JHR Threshold on the chart). This level may change over time because JHR is not a direct result of the antibiotic, but a result of the volume of pathogens eliminated. As the volume of remaining pathogens drop, the line will raise. Eventually, there will be no apparent JHR resulting from the antibiotics. I term this event, the “1<sup>st</sup> level clearance of pathogens”.



### 26.4 Potentated Antibiotic JHR Curve

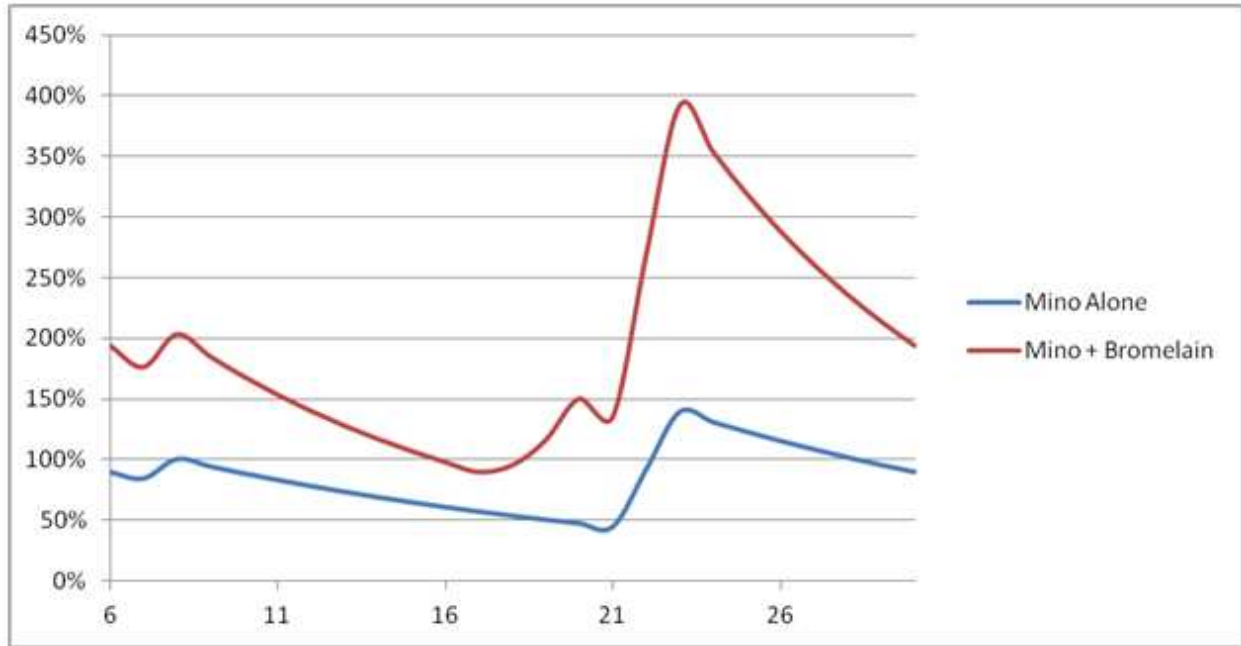
In the curve below we see how things change when a potentator is taken once a day (towards bed time). For example, taking 100 mg of minocycline twice a day and taking bromelain (which increases the penetration of minocycline by 220%<sup>2269</sup>) results in the following pattern:

- ❖ 100 mg at 6am

<sup>2269</sup> <http://www.ncbi.nlm.nih.gov/pubmed/7001087> (1980)

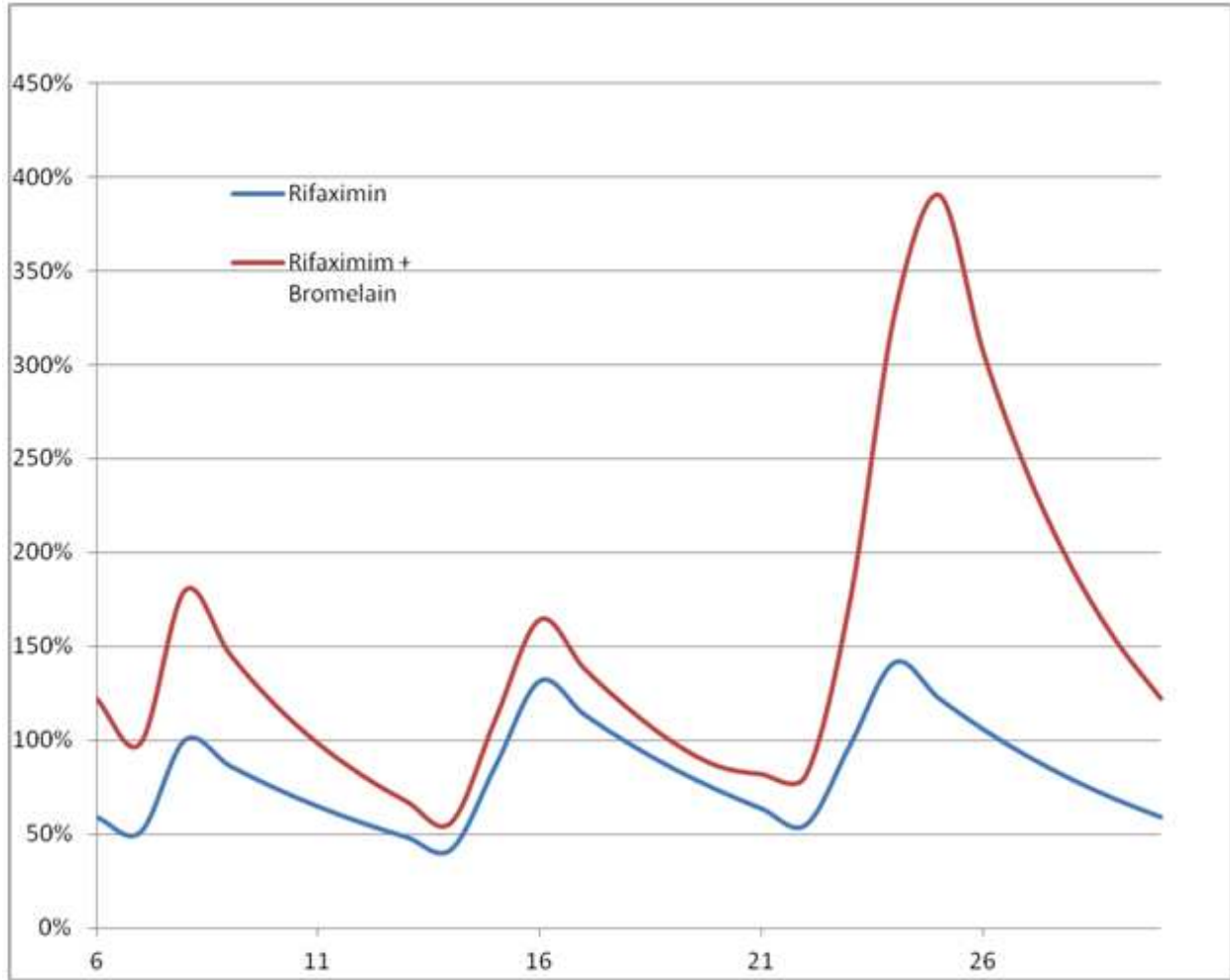
- ❖ 100 mg at 9pm
- ❖ Bromelain at 2pm

Doing some calculations in Excel, I ended up with the concentration charts shown below for minocycline during a day (6am until 6am the next day (30)). The result is a massive JHR while I sleep and a mild JHR during the day.



If your prescription is more often, then you will still see a similar pattern. For example, with a different antibiotic with three times a day, I calculated the following pattern (assuming similar amount of potentiating):

- ❖ 6 am – antibiotics
- ❖ 2 pm - antibiotics
- ❖ 10 pm - antibiotics
- ❖ 7 pm - bromelain



A rule of thumb is:

- ❖ Take the potentator 4-6 hrs before bedtime, once a day.
- ❖ Take the antibiotic 1 hr before bedtime.

## 26.5 JHR Times for common items

The table below shows the TMax and Half-Life for items that may cause a JHR. There are four classes of substances:

- ❖ Antibiotics
- ❖ Biofilms agents
- ❖ Potentators / Anticoagulation agents.

Combining their timing appropriately will allow you to manage the JHR better.

Substance	Time to Max Concentration (MaxT)	Half-Life
<b>Amoxicillin</b> <sup>2270</sup>		1 hr
<b>Azithromycin</b> <sup>2271</sup>		40 hr
<b>Bromelain</b> <sup>2272</sup>	1 hr	6-9 hrs
<b>Clarithromycin</b> <sup>2273</sup>		3 hr
<b>Doxycycline</b> <sup>2274</sup>	2.6 hr	16 hrs
<b>EDTA</b> <sup>2275</sup>		0.7 hr
<b>Erythromycin</b> <sup>2276</sup>		1 hr
<b>Heparin</b> <sup>2277</sup>	Immediate (injected)	1 hr
<b>Levofloxacin</b> <sup>2278</sup>	1 hr <sup>2279</sup>	6-8 hrs
<b>Lumbrokinase</b> <sup>2280</sup>		90 min
<b>Minocycline</b> <sup>2281</sup>	2 hr	11-23 hrs
<b>NAC</b> <sup>2282</sup>	2-3 hr	6 hr
<b>Nattokinase</b> <sup>2283</sup>		8 hrs
<b>Rifampin</b> <sup>2284</sup>	2 hr	5 hr
<b>Serrapeptase</b>	?	?
<b>Tigecycline</b> <sup>2285</sup>		37-67 hr

<sup>2270</sup> <http://www.emedexpert.com/facts/amoxicillin-facts.shtml>

<sup>2271</sup> <http://www.emedexpert.com/compare/macrolides.shtml>

<sup>2272</sup> [http://nopr.niscair.res.in/bitstream/123456789/5694/1/NPR%207\(4\)%20359-363.pdf](http://nopr.niscair.res.in/bitstream/123456789/5694/1/NPR%207(4)%20359-363.pdf)

<sup>2273</sup> <http://www.emedexpert.com/compare/macrolides.shtml>

<sup>2274</sup> <http://www.drugs.com/pro/Doxycycline.html>

<sup>2275</sup> <https://www.medical-library.net/edta-chelation-therapy.html>

<sup>2276</sup> <http://www.emedexpert.com/compare/macrolides.shtml>

<sup>2277</sup> <http://en.wikipedia.org/wiki/Heparin>

<sup>2278</sup> <http://www.emedexpert.com/facts/levofloxacin-facts.shtml>

<sup>2279</sup> <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1315976/> (2005)

<sup>2280</sup> <http://www.arrowheadhealthworks.com/bolouke.htm>

<sup>2281</sup> <http://www.drugs.com/pro/minocycline.html>

<sup>2282</sup> <http://www.benbest.com/nutrceut/NAC.html>

<sup>2283</sup> [http://www.cnmwellness.com/wp-content/uploads/2008/12/art\\_nattokinase\\_2.pdf](http://www.cnmwellness.com/wp-content/uploads/2008/12/art_nattokinase_2.pdf)

<sup>2284</sup> <http://www.drugs.com/pro/xifaxan.html>

<sup>2285</sup> <http://aac.asm.org/content/49/1/220>

**CHRONIC FATIGUE SYNDROME REMISSIONS**

**FACTS - 2012**

[KEN LASSESEN, M.SC.](#)

Preview Extract



# Chronic Fatigue Syndrome Remissions – Facts 2012

© Red Dwarf Dogs 2012

**Editorial Offices**  
892 Lake Samish Rd  
Bellingham, WA  
98229

Published in 2012 by Amazon Co

All Rights Reserved

No part of this book may be reproduced by any means,  
or transmitted, or translated into a machine language  
without the written permission of the author and/or publisher.

Library of Congress Cataloging-in-Publication Data

Lassesen, Kenneth Martinus, 1952-

Chronic Fatigue Syndrome Remission – Facts 2012  
Includes bibliographical references, appendices and index.  
ISBN (Amazon eBook)

1. Chronic Fatigue Syndrome

RB150.F37

2012

Preview Extract

*In Memory of*

*Richard A Van Konynenburg  
A Great Friend and Researcher to the  
ME/CFS, Lyme and MCS  
Communities*

*Departed Sept 26, 2012*

Preview Extract

Contents

1	Foreword.....	14
2	Reading Guidance .....	16
2.1	Guidance for Suspected CFS Suffer	17
2.2	Guidance for Brain Fogged CFS Patients	19
2.3	Guidance for Naturopaths	20
3	Appendix: Microflora families.....	23
3.1	Bacteroides	23
3.2	Bifidobacterium	23
3.3	Enterobacter	24
3.4	Enterococcus	24
3.5	Escherichia coli	24
3.6	Klebsiella	25
3.7	Lactobacillus	25
3.8	Staphylococcus Aureus	25
3.9	Streptococcus	25
3.10	Alteration Routes	25
3.10.1	Escherichia Coli Nissle 1917(EcN)	26
3.10.2	VSL#3	26
3.10.3	Lactobacillus Johnsonii	27
3.10.4	Lactobacillus Reuteri	27
3.10.5	Lactobacillus Rhamnosus	27
3.10.6	Saccharomyces boulardii	28
4	Appendix: Antibiotics.....	29
4.1	Reported Results	29
4.2	Antibiotics Characteristics	29
4.3	Antibiotics Annotations	31
4.3.1	Aminoglycosides Antibiotics	31
4.3.2	Beta-lactam antibiotics	31
4.3.3	Fluoroquinolones	32
4.3.4	Macrolide antibiotic	33
4.3.5	Rifamycin	34
4.3.6	Tetracyclines	34
4.4	Anti-Parasitic Drugs	35
4.4.1	Metronidazole	35
4.4.2	Tinidazole	36
4.5	Arsenic Based	36
4.6	General Practice MDs	36
4.7	Guidance for CFS MD/ND/Researcher	38
4.8	Just the facts Ma'am	38
4.9	Keeping an open mind – Collateral Effects	40
4.10	Sources of Information and Citations	41
4.11	Common Abbreviations Used	41

5	Many Facets of Chronic Fatigue Syndrome .....	43
5.1	How well do CFS patients match research definitions?	44
5.1.1	Incidence of CFS	45
5.2	Research Definitions	45
5.2.1	International CFS Case Definition	45
5.2.2	Fukuda 1994 Definition	46
5.2.3	National Institute of Health and Clinical Excellence	46
5.2.4	Canadian Definition	47
5.3	More Symptoms	48
5.4	Co-morbid Conditions:	51
5.4.1	Irritable Bowel Syndrome (IBS)	52
5.4.2	Meares-Irlen Syndrome	52
5.4.3	Multiple Chemical Sensitivity	53
5.4.4	Postural Tachycardia Syndrome	53
5.4.5	Raynaud’s Syndrome	54
5.4.6	Temporomandibular Disorder	54
5.5	Related Conditions	54
5.5.1	Autoimmune Disease Interstitial Pneumonia	54
5.5.2	Chronic Cerebrospinal Venous Insufficiency	54
5.5.3	Chronic Epstein Barr virus-infection	54
5.5.4	Chronic Lyme	55
5.5.5	Chronic Post-SARS Syndrome	55
5.5.6	Ciguatera poisoning	55
5.5.7	Sicca Syndrome (Dry Eye/Dry Mouth Syndrome)	55
5.5.8	Endometriosis	55
5.5.9	Eosinophilia-myalgia syndrome	55
5.5.10	Gulf War Syndrome	56
5.5.11	Lyme	56
5.5.12	Macrophagic myofasciitis	56
5.5.13	Young-Onset Monogenetic Parkinsonism	56
5.5.14	Pfisteria	56
5.5.15	Phosphate Diabetes	56
5.5.16	Sarcoidosis Remission	56
5.5.17	Sjögren's syndrome	57
5.5.18	Traumatic Brain Injury	57
5.6	Misdiagnosis	57
5.6.1	Differential Diagnoses	57
5.7	The Symptoms Trap	59
6	Brain Scans.....	60
6.1	Magnetic Resonance Imaging	60
6.2	Positron emission tomography	61
6.3	Transcranial Doppler Sonography	61
6.4	SPECT	61
6.5	Blood-Brain-Barrier	62
7	Exercise .....	64
7.1	Post Exertional Malaise	64

7.2	Sports and Exercise	65
8	Laboratory Manifestation .....	66
8.1	Amino Acids, Minerals and Vitamins	67
8.2	Aldosterone	67
8.3	Alpha-MSH	67
8.4	Angiotensin Converting Enzyme	68
8.5	Beta 2-Microglobulin	68
8.6	Blood	68
8.6.1	Blood Pressure	68
8.6.2	Plasma Osmolality	69
8.6.3	Plasma Renin	69
8.6.4	Red blood cells (Erythrocytes)	69
8.7	CD4/CD8 Ratio	69
8.8	Choline	70
8.9	Citric Acid	70
8.10	Circadian Rhythm	70
8.11	Coagulation	70
8.12	Cortisol	71
8.13	Cyclo-oxygenase-2	71
8.14	Cytokines	71
8.14.1	Cytokines impacts	74
8.15	C - reactive protein	74
8.16	Erythrocyte sedimentation rate (ESR)	75
8.17	Glucocorticoid	75
8.18	Heart	75
8.19	Inducible NO synthase	77
8.20	Lysozyme	77
8.21	NFkappabeta	77
8.22	Neopterin	77
8.23	Neuropeptide-Y	78
8.24	Natural Killer (NK) Cell Subsets	78
8.25	Norepinephrine	78
8.26	Pesticides	78
8.27	RNase-L	79
8.28	Serine	79
8.29	Serotonin	79
8.30	Transforming Growth Factor – beta	79
9	Pathogen and CFS .....	80
9.1	Probable Pathogens	80
9.1.1	XMRV virus	86
9.1.2	Neuroinflammatory infections	86
9.1.3	CFS Pathogens as Immune System Tricksters	86
9.1.4	Pathogens persistence	86
9.1.5	Biofilms	87
9.1.6	Infection modification of Body	88
9.1.7	The microflora dimension	88
9.2	CFS Pathogens and Onset	89

9.3	Infections and cytokines production	90
9.3.1	Cytokines inhibitors and removers	90
9.4	Infections and the Immune System	92
9.5	Case Study: Chlamydia Pneumonia	92
10	Microflora .....	93
10.1	CFS Microflora	94
10.2	Related Microflora	95
10.2.1	Chronic Enteritis	95
10.2.2	Crohn’s Microflora	95
10.2.3	Sjögren's Syndrome Microflora	96
10.2.4	Rheumatoid Arthritis	96
10.2.5	IBS Microflora	96
10.2.6	Shifts with Age	97
10.3	Microflora encouraging pathogens	97
10.4	Altering Microflora	98
10.4.1	Probiotics can be deadly	98
10.5	Treatment Implications	98
11	Supplements and Drugs .....	99
11.1	5-HTP	99
11.2	Aloe	99
11.3	Alpha Lipoic Acid	100
11.4	Amino Acids	100
11.4.1	Arginine	101
11.4.2	Asparagine	101
11.4.3	Aspartic acid (Aspartate)	101
11.4.4	Carnitine	101
11.4.5	Cysteine	102
11.4.6	Glutamic acid (Glutamate)	102
11.4.7	Glutamine	102
11.4.8	Glycine	102
11.4.9	Homocysteine	102
11.4.10	Isoleucine	102
11.4.11	Leucine	103
11.4.12	Methionine	103
11.4.13	Oxitriptan (5-HTP)	103
11.4.14	Ornithine	103
11.4.15	Phenylalanine	103
11.4.16	Taurine	103
11.4.17	Tryptophan	103
11.4.18	Phenylalanine	104
11.5	Ampligen	104
11.6	Antidepressants	104
11.6.1	Fluoxetine (Prozac)	105
11.7	Aspirin	105
11.8	Ashwagandha	105
11.9	Artemisia	105
11.10	Antihistamines	106

11.10.1	Azelastine	106
11.10.2	Ketotifen	106
11.11	Bishop Weed (Ajwain Seeds)	106
11.12	Boswellia	107
11.13	Bromelain	108
11.14	Butcher’s Broom (Ruscus aculeatus)	108
11.15	Cat’s Claw (Uncaria tomentosa)	109
11.16	Chocolate (85%)	109
11.17	Cholestyramine	109
11.18	Citicoline	110
11.19	Coenzyme Q10	110
11.19.1	Ubiquinol	111
11.19.2	Idebenone	111
11.20	Dehydroepiandrosterone (DHEA)	111
11.21	D-Ribose	112
11.22	EDTA	112
11.23	Enbrel	112
11.24	Evening Primrose Oil	112
11.25	Fluoride	113
11.26	FOS	113
11.27	Galantamine	114
11.28	Ginger	114
11.29	Ginseng	114
11.30	Ginkgo biloba	114
11.31	Grape Seed Extract	115
11.32	Heparin	116
11.33	Lantana camara (Spanish Flag)	116
11.34	Licorice (Glycyrrhiza)	117
11.35	Loperamide	118
11.36	Lumbrokinase	118
11.37	Malic Acid	118
11.38	Mastic Gum	118
11.39	Mirtazapine	118
11.40	Melatonin	118
11.41	Milk Thistle (Silymarin)	119
11.42	Minerals - Metals	119
11.42.1	Aluminum	119
11.42.2	Calcium	120
11.42.3	Chromium	120
11.42.4	Copper	120
11.42.5	Iron	120
11.42.6	Magnesium	121
11.42.7	Manganese	121
11.42.8	Potassium	121
11.42.9	Selenium	121
11.42.10	Zinc	121
11.43	Monolaurin	121
11.44	Monosodium Glutamate	122

11.45	Myrrh Gum	122
11.46	NAC (Acetylcysteine)	122
11.47	NADH	123
11.48	Naltrexone	123
11.49	Nattokinase	123
11.50	Olestra	124
11.51	Olive Leaf Extract	124
11.52	Omega-3	125
11.53	Oxymatrine	125
11.54	Pleconaril	125
11.55	Probiotics	125
11.56	Prednisone	126
11.57	Quercetin	126
11.58	Racetams	126
11.59	Rituximab	127
11.60	Rosavin (Rhodiola)	127
11.61	Sambucol	128
11.62	SAM-e (ademetonine)	128
11.63	Salt (Sodium Chloride)	128
11.64	Serrapeptase	128
11.65	Silver (Colloidal)	129
11.66	Sodium Bicarbonate	129
11.67	Splenda	129
11.68	Streptokinase	129
11.69	Staphylokinase	130
11.70	St. John’s Wort	130
11.71	Sunflower Oil	130
11.72	Turmeric	130
11.73	Ubiquinol	131
11.74	Vitamins	131
11.74.1	Vitamin A	131
11.74.2	Vitamin B	132
11.74.3	Vitamin C	133
11.74.4	Vitamin D	133
11.74.5	Vitamin-E	134
11.75	Whey	134
11.76	Wobenzym	134
12	Anti-pathogens .....	135
12.1	Anti-body Therapy	135
12.1.1	Gammaglobulin	135
12.1.2	Transfer Factor	135
12.2	Anti-viral Therapy	136
12.2.1	Ganciclovir	136
12.2.2	Valacyclovir	136
12.3	Anti-Microflora	137
12.4	Anti-Biotics Therapy	137
12.4.1	Antibiotics and Remission	138



13	Anti-coagulants .....	139
14	Supplements: Probiotics Species .....	140
14.1	Diet Modification of Microflora	140
14.2	Novel ways to modify Microflora	143
14.2.1	Fecal Transplants	143
14.2.2	Different Cheeses	144
14.2.3	Non-systemic Antibiotics	144
15	Genetics .....	146
15.1	Gene Expression	147
15.2	Evolutional Benefit for CFS Genes	147
16	Remissions .....	149
16.1	What is Remission	149
16.2	Low Incidence Remission via Supplements	150
16.2.1	Chocolate Remission	150
16.2.2	Licorice Remission	151
16.2.3	Olive Leaf Extract Remission	151
16.2.4	Vitamin D Remission	151
16.3	Collateral Remission	152
16.3.1	Heparin Remission	152
16.3.2	Cancer Chemotherapy	153
16.3.3	Fecal Transplant Remission	153
16.4	Anti-Pathogen Remissions	153
16.5	Summary of Remissions	154
16.6	Building a Model of how Remission happens	154
17	Models of CFS .....	155
17.1	Hypoxia	155
17.1.1	Hypoxia Predictions	157
17.1.2	Predictions Extended	157
17.2	Blood Model	159
17.2.1	Explorations	160
17.2.2	Predictions	161
17.3	CFS Model for the Brain Fogged	162
17.3.1	Steps	166
17.3.2	The pH Dimension	167
17.4	Technical Model	169
17.4.1	Onset – Failure to Restore Prior Microflora	169
17.4.2	A Pathogen Pot-Luck Street Party	169
17.4.3	Second Degree Side-Effects	171
17.4.4	Initial Onset with Stress	171
17.4.5	Reactivation or loss of control of pathogens	172
17.4.6	A new system balance is established	173
17.4.7	Treatment Implications	173
17.4.8	Paleolithic diet	174
17.4.9	Modifying microflora	174
17.4.10	Predictions	175

18	Treatments.....	176
18.1	Past Treatment Fads	179
18.1.1	Cognitive-Behavioral Therapy	179
18.1.2	Graded Exercise Therapy	180
18.1.3	Alfred Blasi Protocol	180
18.1.4	Marshall Protocol	181
18.1.5	Methylation Defect	181
18.1.6	Salt and C	182
18.1.7	Xenotropic Murine Leukemia Related Virus	182
18.1.8	Early Life Immune Insult	182
18.2	Anti-pathogen Protocols	182
18.3	Anticoagulant Treatment	184
18.4	Animals with CFS and their Physicians	184
18.5	Chemotherapy for CFS	184
19	Future Studies.....	185
19.1	Diagnosis Collection	185
19.1.1	Microflora x Pathogens	185
19.1.2	Microflora x Genetics	185
19.1.3	Genetics x Pathogens	185
19.1.4	CFS Pathogen x Microflora	185
19.1.5	Antibiotic x Microflora	185
19.1.6	Mother of all Studies	186
19.2	Treatment Trials	186
19.2.1	Mutaflor (E.Coli Nissle)	186
19.2.2	Goat Cheese and Rye-FOS	186
19.2.3	85% Chocolate x Microflora	187
19.2.4	Standard Probiotics	187
19.2.5	Therapeutic Fasting	187
19.3	Consequences of above studies	187
20	Further Information Sources .....	189
20.1	CFS Conferences	189
20.1.1	IACFS/ME	189
20.1.2	AHMF	189
20.1.3	Nation ME/Fibromyalgia Action Network	189
20.1.4	INVEST in ME	189
20.1.5	Other Conferences	190
20.2	Lyme Conferences	190
20.3	CFS Reference Libraries	190
20.4	CFS Newsletters	191
20.5	CFS User Groups	191
21	Bibliography .....	192
22	Appendix: Stress Catecholamines.....	194
23	Appendix: Microflora families.....	196
23.1	Bacteroides	196
23.2	Bifidobacterium	197

23.3	Enterobacter	197
23.4	Enterococcus	198
23.5	Escherichia coli	198
23.6	Klebsiella	199
23.7	Lactobacillus	200
23.8	Staphylococcus Aureus	200
23.9	Streptococcus	200
23.10	Alteration Routes	200
23.10.1	Escherichia Coli Nissle 1917(EcN)	201
23.10.2	VSL#3	202
23.10.3	Lactobacillus Johnsonii	202
23.10.4	Lactobacillus Reuteri	203
23.10.5	Lactobacillus Rhamnosus	203
23.10.6	Saccharomyces boulardii	204
24	Appendix: Antibiotics.....	205
24.1	Reported Results	205
24.2	Antibiotics Characteristics	205
24.3	Antibiotics Annotations	209
24.3.1	Aminoglycosides Antibiotics	209
24.3.2	Beta-lactam antibiotics	209
24.3.3	Fluoroquinolones	211
24.3.4	Macrolide antibiotic	212
24.3.5	Rifamycin	214
24.3.6	Tetracyclines	214
24.4	Anti-Parasitic Drugs	217
24.4.1	Metronidazole	217
24.4.2	Tinidazole	217
24.5	Arsenic Based	217
25	Appendix: Theoretical Protocols.....	218
25.1	Theoretical Protocol without Prescriptions	218
25.1.1	Support Foundation	218
25.1.2	Anti-Pathogen Agents	219
25.1.3	Suggested dosages and timings to Start	220
25.1.4	Suggested sequence of anti-pathogens	221
25.1.5	What are we trying to do?	221
25.2	Theoretical Protocol with Prescriptions	221
25.2.1	Antibiotics	221
26	Appendix: Jarisch-Herxheimer Reaction.....	223
26.1	Understanding and Managing JHR is Essential	225
26.2	Managing JHR	225
26.3	Basic Antibiotic JHR Curve	226
26.4	Potentated Antibiotic JHR Curve	226
26.5	JHR Times for common items	228
27	Appendix My History with CFS.....	230
27.1	1973 – Onset without PEM	230
27.2	1999 – Onset with PEM	230