# Chronic Infection as an associative finding in Parkinsons. Case History

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# Epidemiology

- 1 million people affected in the USA
- 50,000 new cases diagnosed annually
- Incidence increases steadily with age
- Possibly related to decline in antioxidant functionality

Perlmutter

# Parkinsonian symptoms

- Tremor
- Bradykinesia or slowness of movement
- Rigidity
- Loss of automatic movements
- Impaired balance

Considered to be downstream effects of a primary brain biochemical abnormality, the loss of dopamine production due to neuronal degeneration in the area of the brain called the substantia nigra

# Parkinson treatments

- Drugs eg levodopa which increases the amount of dopamine. LevoDopa consumes vitamin B6 and increases homocysteine elevation
- Dopamine agonists eg ropinerol
- Tremors eg propranolol
- Bacofen or Diazepam for muscle spasms
- Surgical
- Food and nutrient interventions have been shown to be more than palliative. They restore function and delay disease progression, possibly long enough for the patients to benefit from surgical restorative treatments as they become available.
- Currently there is no “recognised cure”

# Case History

Male aged 45, diagnosed with Parkinson's disease seven years previously, aged 38.

When he was aged 21, his mother died of fulminating lupus following a 10-year year illness with gradual organ failure..

Soon after this, he got married. Remains happily married. Has held down a top job until age of 45.

Referred by company as health deteriorating.

On medications, Levodopa, Ramipril for hypertension, Lansoprazole for GERD

# Gastroenteritis preceded Parkinsons by 5 years

***Severe gastroenteritis*** infection whilst on holiday ***aged 33***. He had required a drip.

Following this, suffered severe gut symptoms investigated with endoscopy,, colonoscopy and excluded coeliac disease.

Nothing was found and so he just struggled on, eventually developing ***Parkinson's disease aged 38.***

At the time of our meeting, he had the ***classical triad of shuffling gait, cogwheel rigidity and tremor***.

Diminishing help from Levodopa, breakthrough symptoms for 1/2hr prior to each dose

# TESTS

FOLLOW THE GUT...SO…. Food intolerance IgG tests. He was able to remove certain foods from his diet and his symptoms improved quite considerably. (egg white, dairy and many gluten -containing foods, which would have been sensible advice given the family history of autoimmunity).

CDSA 2.0  stool test

Metabolic screen, Vitamin D, homocysteine

Toxicity tests ( metals)

Neurogenomic  GENETIC screen

Optimal Nutritional evaluation ( deficiencies, oxidative stress, gut health, mitochondria)

Hormones; testosterone, PREGNENALONE, oestrogen, progesterone, adrenal & thyroid assessment inc antibodies & reverse T3

# Food IGG sensitivities ( CNS labs)

Dairy

Wheat

Egg

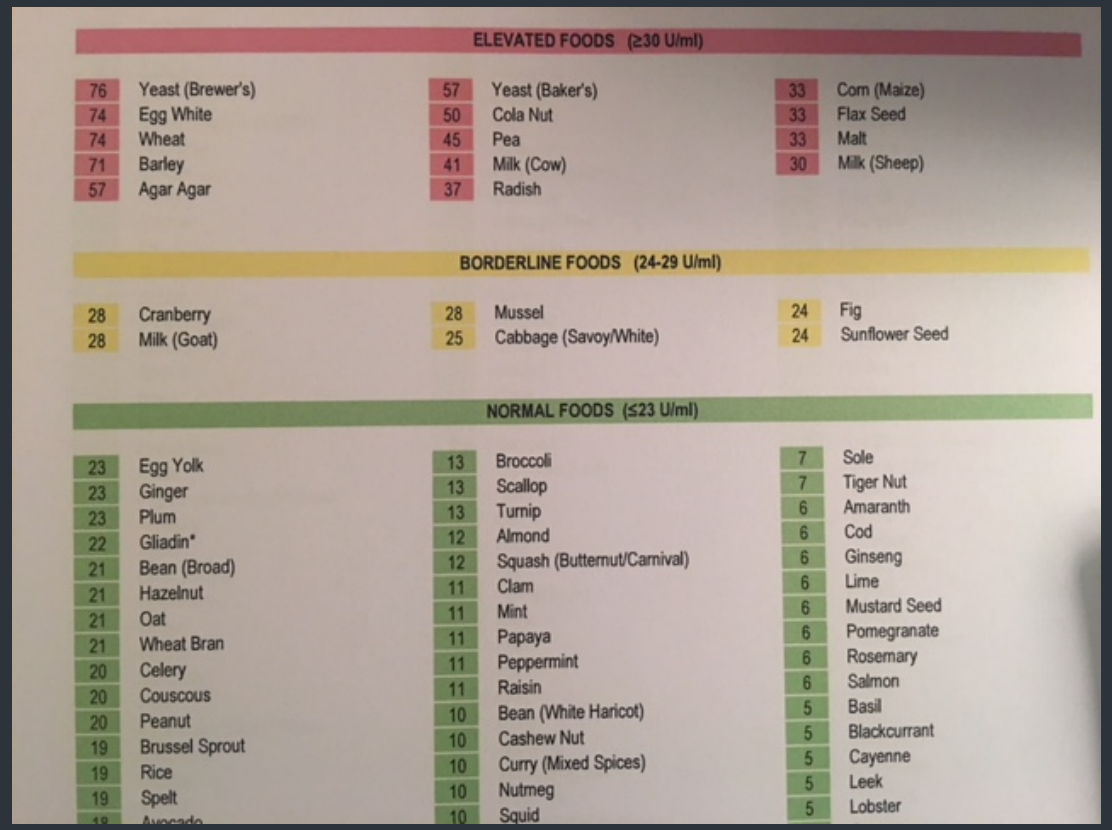
Remove the reds

Rotate the yellows

Eat any of the greens

NOT the whole story

Cause of IGG reaction maybe a combination of maldigested food; combined with metals; chemicals, biotoxins. COMPLEX; needs further assessment



**ELEVATED FOODS (≥30 U/ml)**

| | | | | | |
|---|---|---|---|---|---|
| 76 | Yeast (Brewer's) | 57 | Yeast (Baker's) | 33 | Corn (Maize) |
| 74 | Egg White | 50 | Cola Nut | 33 | Flax Seed |
| 74 | Wheat | 45 | Pea | 33 | Malt |
| 71 | Barley | 41 | Milk (Cow) | 30 | Milk (Sheep) |
| 57 | Agar Agar | 37 | Radish | | |

**BORDERLINE FOODS (24-29 U/ml)**

| | | | | | |
|---|---|---|---|---|---|
| 28 | Cranberry | 28 | Mussel | 24 | Fig |
| 28 | Milk (Goat) | 25 | Cabbage (Savoy/White) | 24 | Sunflower Seed |

**NORMAL FOODS (≤23 U/ml)**

| | | | | | |
|---|---|---|---|---|---|
| 23 | Egg Yolk | 13 | Broccoli | 7 | Sole |
| 23 | Ginger | 13 | Scallop | 7 | Tiger Nut |
| 23 | Plum | 13 | Turnip | 6 | Amaranth |
| 22 | Gliadin* | 12 | Almond | 6 | Cod |
| 21 | Bean (Broad) | 12 | Squash (Butternut/Carnival) | 6 | Ginseng |
| 21 | Hazelnut | 11 | Clam | 6 | Lime |
| 21 | Oat | 11 | Mint | 6 | Mustard Seed |
| 21 | Wheat Bran | 11 | Papaya | 6 | Pomegranate |
| 20 | Celery | 11 | Peppermint | 6 | Rosemary |
| 20 | Couscous | 11 | Raisin | 6 | Salmon |
| 20 | Peanut | 10 | Bean (White Haricot) | 5 | Basil |
| 19 | Brussel Sprout | 10 | Cashew Nut | 5 | Blackcurrant |
| 19 | Rice | 10 | Curry (Mixed Spices) | 5 | Cayenne |
| 19 | Spelt | 10 | Nutmeg | 5 | Leek |
| 19 | Avocado | 10 | Squid | 5 | Lobster |

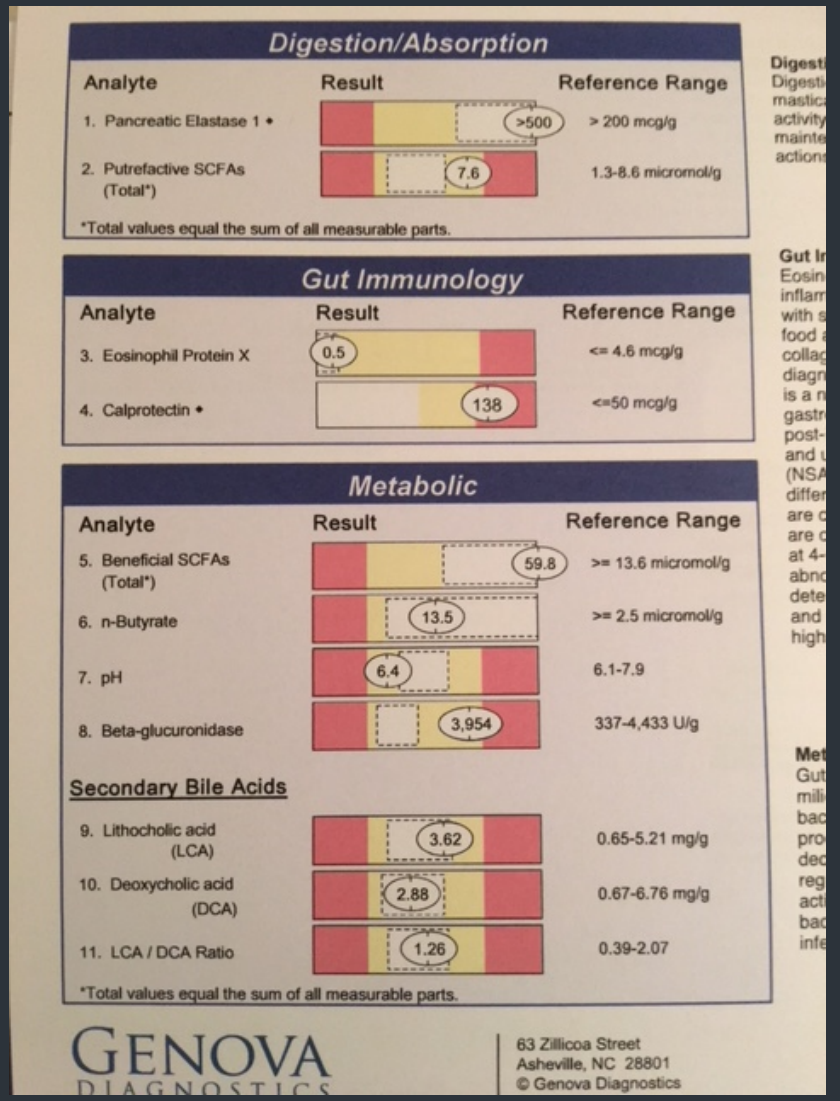
# Gluten & Auto-immunity!

- **NOW ( 2019)** *Dietary Focus shift:*
- *Keto / Paleo diet*
- *Membrane medicine ( Fatty acid assessments; focus on replenishing fatty acid deficiencies; phospholipid treatments)*
- *Replenish electrolytes & minerals*
- *Mitochondrial function & DNA adducts*

CDSA with parasitology

High calprotectin: cause? Medically investigated, possibly relates to high IGG levels

High Beta-glucuronidase (reabsorption of toxins)



## Digestion/Absorption

| Analyte | Result | Reference Range |
|---|---|---|
| 1. Pancreatic Elastase 1 • | >500 | > 200 mcg/g |
| 2. Putrefactive SCFAs (Total*) | 7.6 | 1.3-8.6 micromol/g |

*Total values equal the sum of all measurable parts.

## Gut Immunology

| Analyte | Result | Reference Range |
|---|---|---|
| 3. Eosinophil Protein X | 0.5 | <= 4.6 mcg/g |
| 4. Calprotectin • | 138 | <=50 mcg/g |

## Metabolic

| Analyte | Result | Reference Range |
|---|---|---|
| 5. Beneficial SCFAs (Total*) | 59.8 | >= 13.6 micromol/g |
| 6. n-Butyrate | 13.5 | >= 2.5 micromol/g |
| 7. pH | 6.4 | 6.1-7.9 |
| 8. Beta-glucuronidase | 3,954 | 337-4,433 U/g |
| **Secondary Bile Acids** | | |
| 9. Lithocholic acid (LCA) | 3.62 | 0.65-5.21 mg/g |
| 10. Deoxycholic acid (DCA) | 2.88 | 0.67-6.76 mg/g |
| 11. LCA / DCA Ratio | 1.26 | 0.39-2.07 |

*Total values equal the sum of all measurable parts.

GENOVA DIAGNOSTICS

63 Zillicoa Street
Asheville, NC 28801
© Genova Diagnostics

# CDSA high calprotectin

Normal SMALL BOWEL ENTEROGRAM TO EXCLUDE SMALL BOWEL IBD

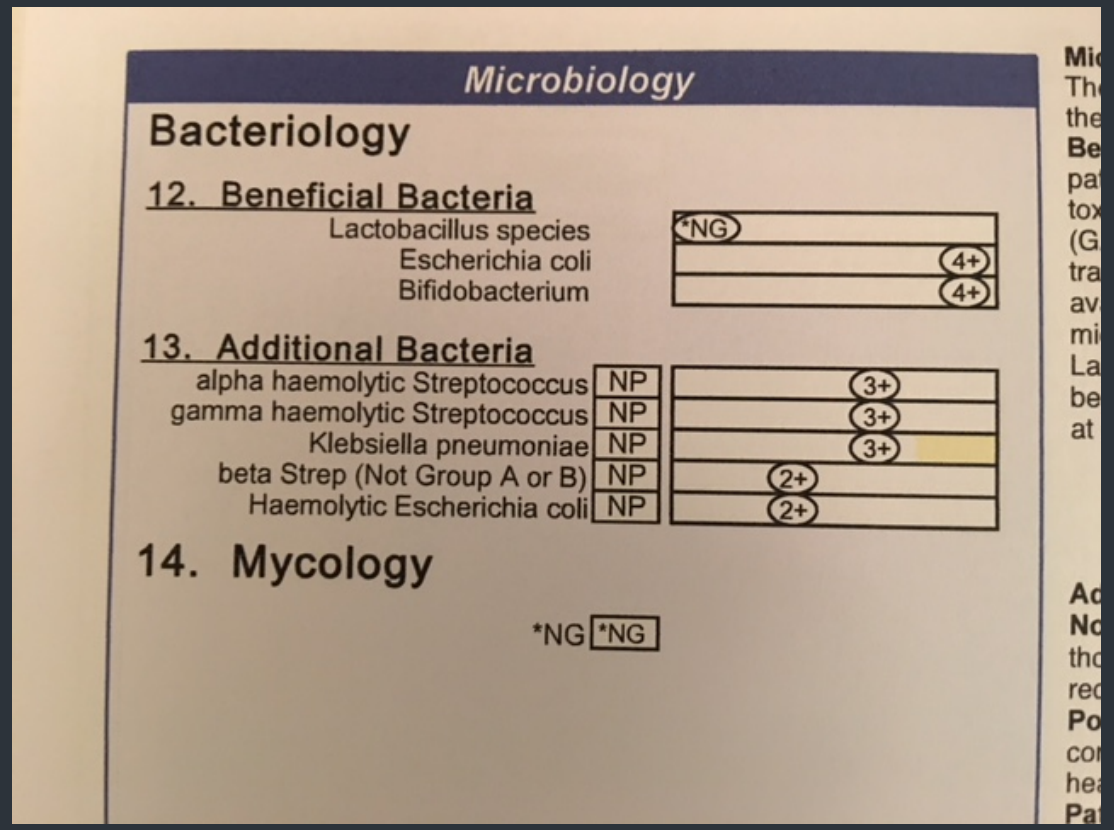
Normal Virtual colonoscopy

CDSA 2.0 + parasitology
Genova (NOW GI Map)

Low beneficial bacteria

High additional bacteria (usually no more than 2-3)

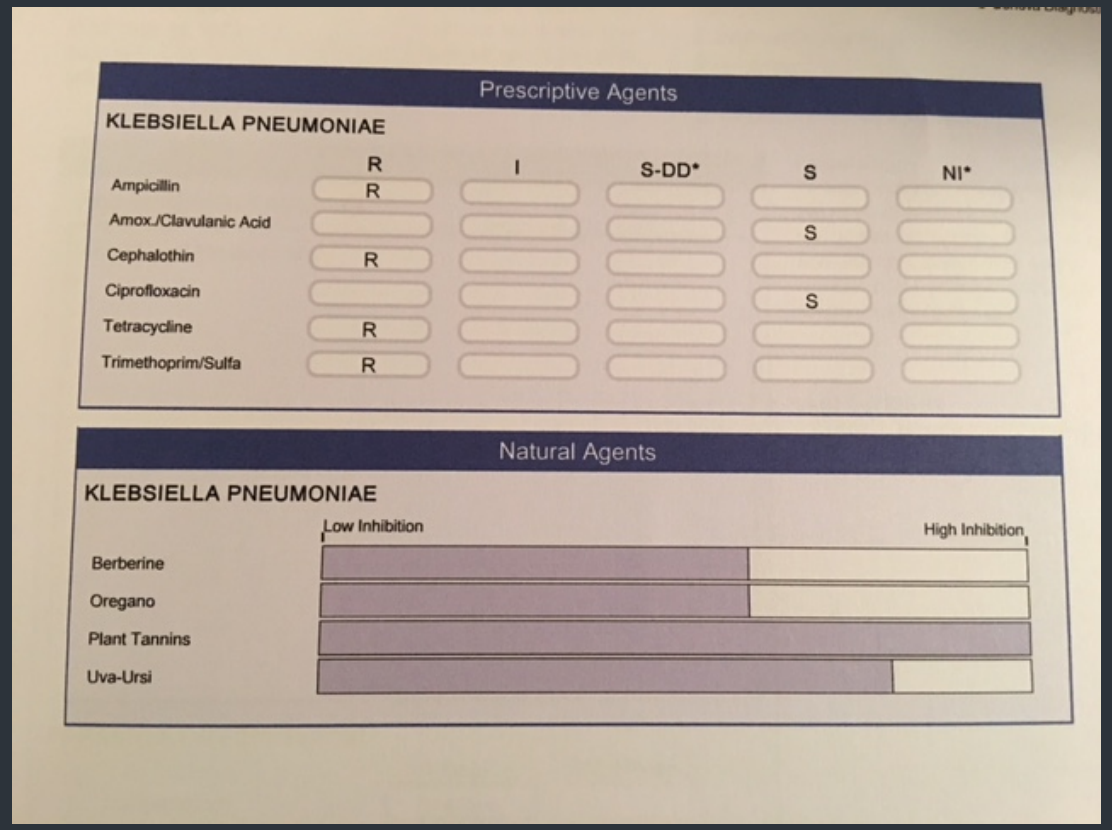
Klebsiella positive



CDSA sensitivities

Antibiotics versus natural agents; we used plant tannins & probiotics, + general support for gut health.

**Optimise gut health**



Prescriptive Agents

KLEBSIELLA PNEUMONIAE

| | R | I | S-DD* | S | NI* |
|---|---|---|---|---|---|
| Ampicillin | R | | | | |
| Amox./Clavulanic Acid | | | | S | |
| Cephalothin | R | | | | |
| Ciprofloxacin | | | | S | |
| Tetracycline | R | | | | |
| Trimethoprim/Sulfa | R | | | | |

Natural Agents

KLEBSIELLA PNEUMONIAE

| | Low Inhibition | High Inhibition |
|---|---|---|
| Berberine | | |
| Oregano | | |
| Plant Tannins | | |
| Uva-Ursi | | |

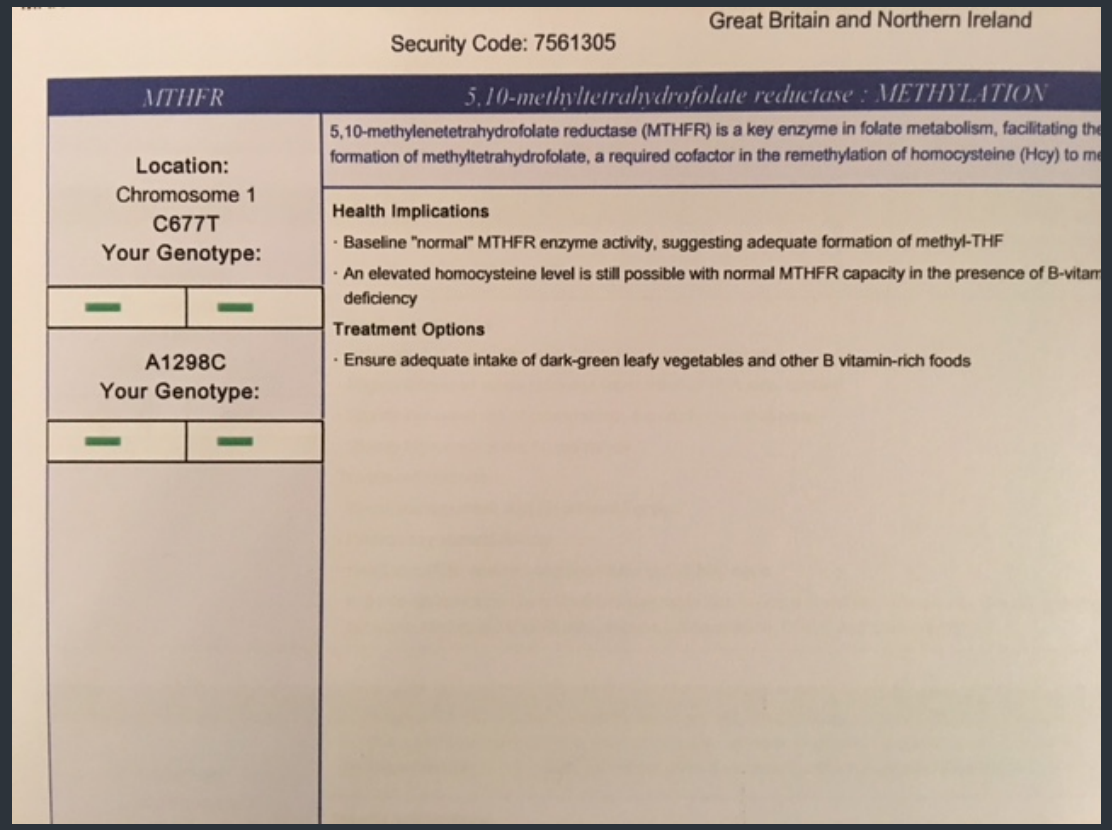
# Metabolic results

- Homocysteine level 25mmol/l ( ideal less than 8)
- Folate 2.5ug /L ( normal above 3.9)
- B12 46pmol/l ( normal range 25-165)
- Free testosterone  5.7 ( normal range 4-30)
- Reverse T3 25 ( normal less than 24, ideal less than 16)

- FBC, liver, kidney, inflammatory markers, other thyroid markers; other sex hormones, adrenal function all ok)

Neurogenomics

# No SNPs on MTHFR gene



Great Britain and Northern Ireland

Security Code: 7561305

| MTHFR | 5,10-methyltetrahydrofolate reductase : METHYLATION |
|---|---|
| Location:<br>Chromosome 1<br>C677T<br>Your Genotype: | 5,10-methylenetetrahydrofolate reductase (MTHFR) is a key enzyme in folate metabolism, facilitating the formation of methyltetrahydrofolate, a required cofactor in the remethylation of homocysteine (Hcy) to me |
| — — | **Health Implications**<br>· Baseline "normal" MTHFR enzyme activity, suggesting adequate formation of methyl-THF<br>· An elevated homocysteine level is still possible with normal MTHFR capacity in the presence of B-vitam deficiency |
| A1298C<br>Your Genotype: | **Treatment Options**<br>· Ensure adequate intake of dark-green leafy vegetables and other B vitamin-rich foods |
| — — | |

# Pro inflammatory SNPS IL6 (Th2 cytokine), IL-1B (inflammatory cytokine produced by macrophages in response to LPS)

Advice re lowering inflammation:

Mediterranean diet

Curcumin

Omega 3 Fats

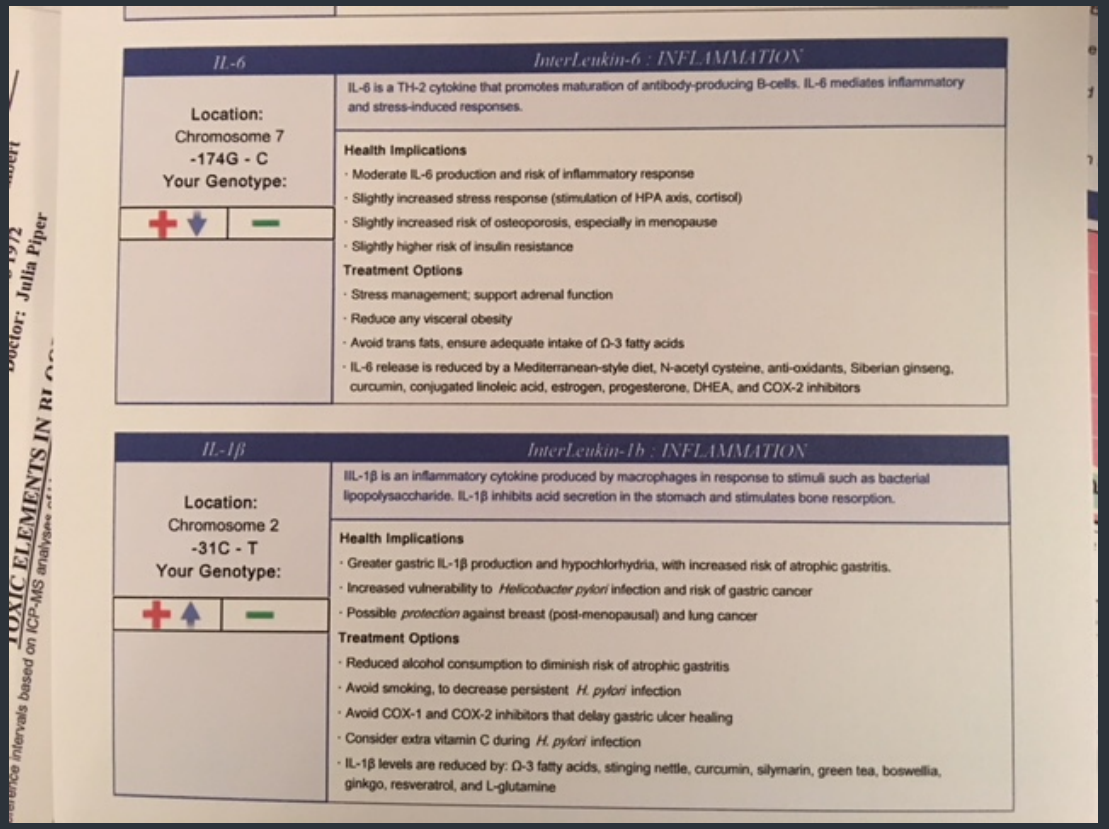
Silymarin

Green tea

Resveratrol

NAC

Hormones Oestrogen ,Progesterone & DHEA



*IL-6* — *InterLeukin-6 : INFLAMMATION*

**Location:**
Chromosome 7
-174G - C
**Your Genotype:**

IL-6 is a TH-2 cytokine that promotes maturation of antibody-producing B-cells. IL-6 mediates inflammatory and stress-induced responses.

**Health Implications**
- Moderate IL-6 production and risk of inflammatory response
- Slightly increased stress response (stimulation of HPA axis, cortisol)
- Slightly increased risk of osteoporosis, especially in menopause
- Slightly higher risk of insulin resistance

**Treatment Options**
- Stress management; support adrenal function
- Reduce any visceral obesity
- Avoid trans fats, ensure adequate intake of Ω-3 fatty acids
- IL-6 release is reduced by a Mediterranean-style diet, N-acetyl cysteine, anti-oxidants, Siberian ginseng, curcumin, conjugated linoleic acid, estrogen, progesterone, DHEA, and COX-2 inhibitors

*IL-1β* — *InterLeukin-1b : INFLAMMATION*

**Location:**
Chromosome 2
-31C - T
**Your Genotype:**

IIL-1β is an inflammatory cytokine produced by macrophages in response to stimuli such as bacterial lipopolysaccharide. IL-1β inhibits acid secretion in the stomach and stimulates bone resorption.

**Health Implications**
- Greater gastric IL-1β production and hypochlorhydria, with increased risk of atrophic gastritis.
- Increased vulnerability to *Helicobacter pylori* infection and risk of gastric cancer
- Possible *protection* against breast (post-menopausal) and lung cancer

**Treatment Options**
- Reduced alcohol consumption to diminish risk of atrophic gastritis
- Avoid smoking, to decrease persistent *H. pylori* infection
- Avoid COX-1 and COX-2 inhibitors that delay gastric ulcer healing
- Consider extra vitamin C during *H. pylori* infection
- IL-1β levels are reduced by: Ω-3 fatty acids, stinging nettle, curcumin, silymarin, green tea, boswellia, ginkgo, resveratrol, and L-glutamine

TOXIC ELEMENTS IN BL
Doctor: Julia Piper

# Other Phase 2 SNPs SOD COMT ACETYLATION & glutathione

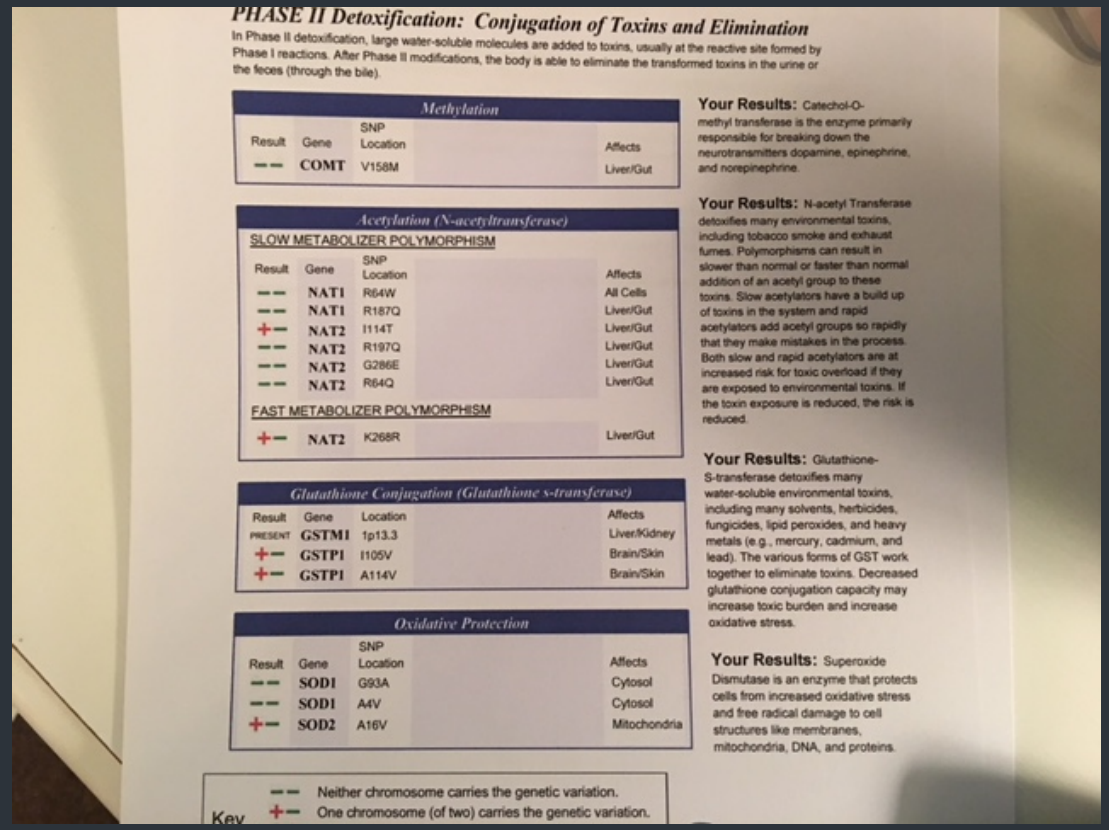
COMT negative ; MTHFR -/-

Many other genes surrounding methylation ( not measured here) try LIFECODE

**Glutathione S transferase x2 SNPs ( MASTER ANTI INFLAMMATORY)**

**SOD (Mitochondrial, membrane and DNA protection)**

**NAT2 : increased toxin build up**



## PHASE II Detoxification: Conjugation of Toxins and Elimination

In Phase II detoxification, large water-soluble molecules are added to toxins, usually at the reactive site formed by Phase I reactions. After Phase II modifications, the body is able to eliminate the transformed toxins in the urine or the feces (through the bile).

### Methylation

| Result | Gene | SNP Location | Affects |
|---|---|---|---|
| −− | COMT | V158M | Liver/Gut |

**Your Results:** Catechol-O-methyl transferase is the enzyme primarily responsible for breaking down the neurotransmitters dopamine, epinephrine, and norepinephrine.

### Acetylation (N-acetyltransferase)

SLOW METABOLIZER POLYMORPHISM

| Result | Gene | SNP Location | Affects |
|---|---|---|---|
| −− | NAT1 | R64W | All Cells |
| −− | NAT1 | R187Q | Liver/Gut |
| +− | NAT2 | I114T | Liver/Gut |
| −− | NAT2 | R197Q | Liver/Gut |
| −− | NAT2 | G286E | Liver/Gut |
| −− | NAT2 | R64Q | Liver/Gut |

FAST METABOLIZER POLYMORPHISM

| Result | Gene | SNP Location | Affects |
|---|---|---|---|
| +− | NAT2 | K268R | Liver/Gut |

**Your Results:** N-acetyl Transferase detoxifies many environmental toxins, including tobacco smoke and exhaust fumes. Polymorphisms can result in slower than normal or faster than normal addition of an acetyl group to these toxins. Slow acetylators have a build up of toxins in the system and rapid acetylators add acetyl groups so rapidly that they make mistakes in the process. Both slow and rapid acetylators are at increased risk for toxic overload if they are exposed to environmental toxins. If the toxin exposure is reduced, the risk is reduced.

### Glutathione Conjugation (Glutathione s-transferase)

| Result | Gene | Location | Affects |
|---|---|---|---|
| PRESENT | GSTM1 | 1p13.3 | Liver/Kidney |
| +− | GSTP1 | I105V | Brain/Skin |
| +− | GSTP1 | A114V | Brain/Skin |

**Your Results:** Glutathione-S-transferase detoxifies many water-soluble environmental toxins, including many solvents, herbicides, fungicides, lipid peroxides, and heavy metals (e.g., mercury, cadmium, and lead). The various forms of GST work together to eliminate toxins. Decreased glutathione conjugation capacity may increase toxic burden and increase oxidative stress.

### Oxidative Protection

| Result | Gene | SNP Location | Affects |
|---|---|---|---|
| −− | SOD1 | G93A | Cytosol |
| −− | SOD1 | A4V | Cytosol |
| +− | SOD2 | A16V | Mitochondria |

**Your Results:** Superoxide Dismutase is an enzyme that protects cells from increased oxidative stress and free radical damage to cell structures like membranes, mitochondria, DNA, and proteins.

Key
−− Neither chromosome carries the genetic variation.
+− One chromosome (of two) carries the genetic variation.

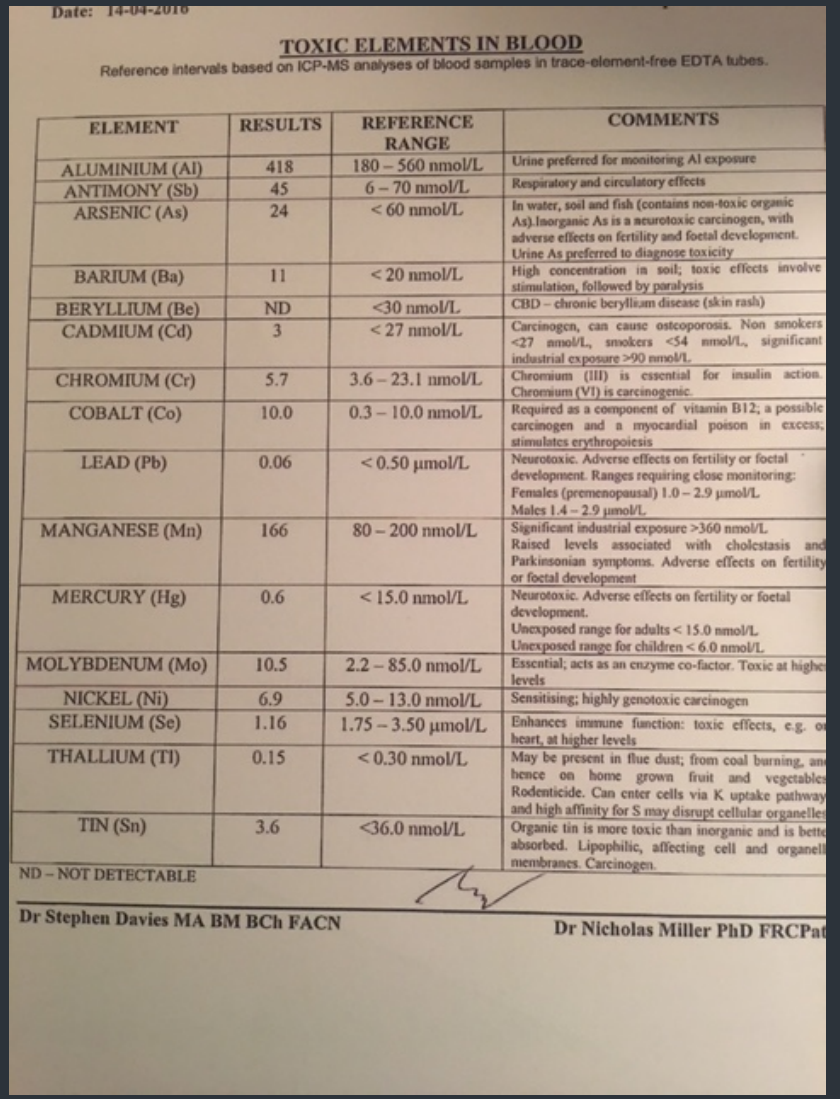
Biolab Toxic Element Screen

Not a provocation test

On the face of it negative

Keep an open mind on metal as it is tightly bound in the cells

**CONSIDER ACUMEN TEST WITH TRANSLOCATION STUDIES & dna ADDUCTS**



Date: 14-04-2016

**TOXIC ELEMENTS IN BLOOD**

Reference intervals based on ICP-MS analyses of blood samples in trace-element-free EDTA tubes.

| ELEMENT | RESULTS | REFERENCE RANGE | COMMENTS |
|---|---|---|---|
| ALUMINIUM (Al) | 418 | 180 – 560 nmol/L | Urine preferred for monitoring Al exposure |
| ANTIMONY (Sb) | 45 | 6 – 70 nmol/L | Respiratory and circulatory effects |
| ARSENIC (As) | 24 | < 60 nmol/L | In water, soil and fish (contains non-toxic organic As).Inorganic As is a neurotoxic carcinogen, with adverse effects on fertility and foetal development. Urine As preferred to diagnose toxicity |
| BARIUM (Ba) | 11 | < 20 nmol/L | High concentration in soil; toxic effects involve stimulation, followed by paralysis |
| BERYLLIUM (Be) | ND | <30 nmol/L | CBD – chronic beryllium disease (skin rash) |
| CADMIUM (Cd) | 3 | < 27 nmol/L | Carcinogen, can cause osteoporosis. Non smokers <27 nmol/L, smokers <54 nmol/L, significant industrial exposure >90 nmol/L |
| CHROMIUM (Cr) | 5.7 | 3.6 – 23.1 nmol/L | Chromium (III) is essential for insulin action. Chromium (VI) is carcinogenic. |
| COBALT (Co) | 10.0 | 0.3 – 10.0 nmol/L | Required as a component of vitamin B12; a possible carcinogen and a myocardial poison in excess; stimulates erythropoiesis |
| LEAD (Pb) | 0.06 | < 0.50 µmol/L | Neurotoxic. Adverse effects on fertility or foetal development. Ranges requiring close monitoring: Females (premenopausal) 1.0 – 2.9 µmol/L Males 1.4 – 2.9 µmol/L |
| MANGANESE (Mn) | 166 | 80 – 200 nmol/L | Significant industrial exposure >360 nmol/L Raised levels associated with cholestasis and Parkinsonian symptoms. Adverse effects on fertility or foetal development |
| MERCURY (Hg) | 0.6 | < 15.0 nmol/L | Neurotoxic. Adverse effects on fertility or foetal development. Unexposed range for adults < 15.0 nmol/L Unexposed range for children < 6.0 nmol/L |
| MOLYBDENUM (Mo) | 10.5 | 2.2 – 85.0 nmol/L | Essential; acts as an enzyme co-factor. Toxic at higher levels |
| NICKEL (Ni) | 6.9 | 5.0 – 13.0 nmol/L | Sensitising; highly genotoxic carcinogen |
| SELENIUM (Se) | 1.16 | 1.75 – 3.50 µmol/L | Enhances immune function: toxic effects, e.g. on heart, at higher levels |
| THALLIUM (Tl) | 0.15 | < 0.30 nmol/L | May be present in flue dust; from coal burning, and hence on home grown fruit and vegetables Rodenticide. Can enter cells via K uptake pathway and high affinity for S may disrupt cellular organelles |
| TIN (Sn) | 3.6 | <36.0 nmol/L | Organic tin is more toxic than inorganic and is better absorbed. Lipophilic, affecting cell and organelle membranes. Carcinogen. |

ND – NOT DETECTABLE

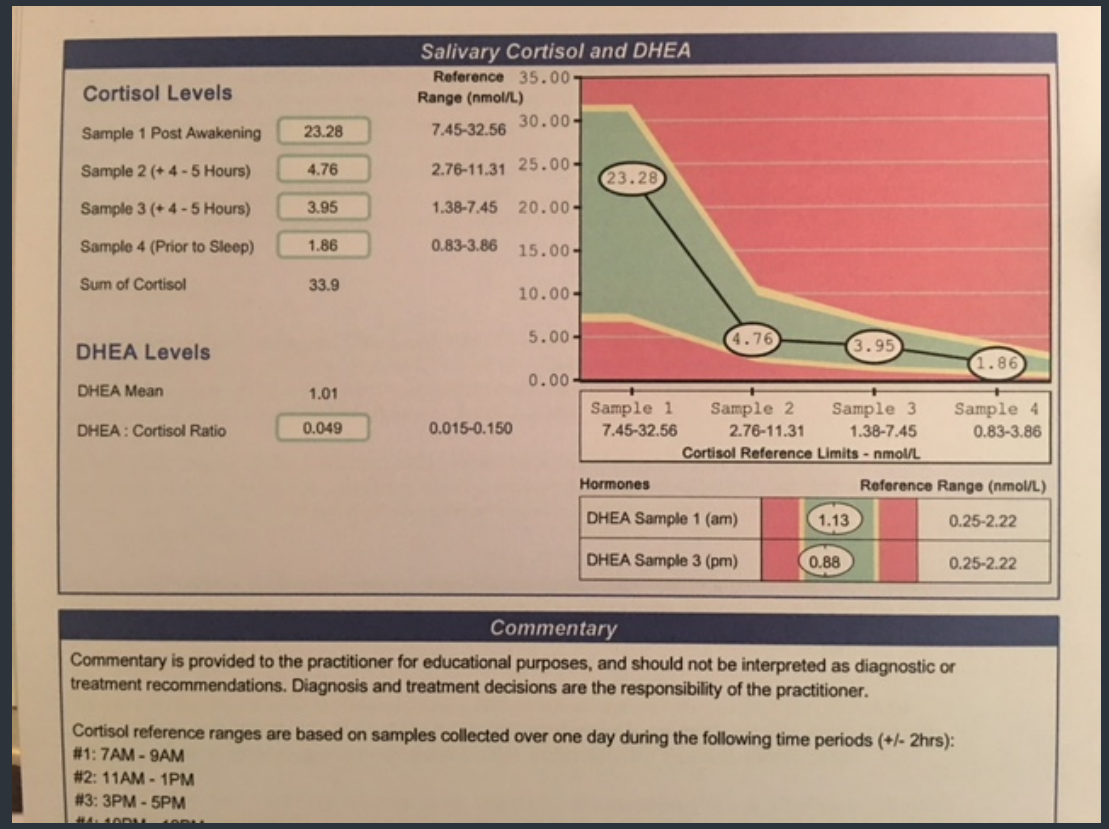
Dr Stephen Davies MA BM BCh FACN

Dr Nicholas Miller PhD FRCPath

HPA axis (Genova Labs)

Diurnal rhythm optimal for cortisol

DHEA IN NORMAL RANGE BUT ON LOW SIDE



Salivary Cortisol and DHEA

| Cortisol Levels | | Reference Range (nmol/L) |
|---|---|---|
| Sample 1 Post Awakening | 23.28 | 7.45-32.56 |
| Sample 2 (+ 4 - 5 Hours) | 4.76 | 2.76-11.31 |
| Sample 3 (+ 4 - 5 Hours) | 3.95 | 1.38-7.45 |
| Sample 4 (Prior to Sleep) | 1.86 | 0.83-3.86 |
| Sum of Cortisol | 33.9 | |

| DHEA Levels | | |
|---|---|---|
| DHEA Mean | 1.01 | |
| DHEA : Cortisol Ratio | 0.049 | 0.015-0.150 |



| Hormones | | Reference Range (nmol/L) |
|---|---|---|
| DHEA Sample 1 (am) | 1.13 | 0.25-2.22 |
| DHEA Sample 3 (pm) | 0.88 | 0.25-2.22 |

Commentary

Commentary is provided to the practitioner for educational purposes, and should not be interpreted as diagnostic or treatment recommendations. Diagnosis and treatment decisions are the responsibility of the practitioner.

Cortisol reference ranges are based on samples collected over one day during the following time periods (+/- 2hrs):
#1: 7AM - 9AM
#2: 11AM - 1PM
#3: 3PM - 5PM

# Test results & management April 2016

- Dietary changes & exclusions as per IGG results; gluten & dairy removal. Mediterranean diet ( now would be keto/paleo
- Testosterone low normal; replacement organised for general mood, muscle strength; immune modulation
- Neurogenomics tests showed two polymorphisms on his glutathione S transferase genes but no polymorphisms in the rate limiting step of methylation, the MTHFR. NAC & Lipoic acid suggested **( now liposomal glutathione)**
- Optimal nutritional evaluation April 2016, mild deficiency in magnesium. Interestingly, his oxidative stress markers were normal. Epsom salt baths recommended.
- Comprehensive stool analysis from Genova : low protective bacteria, high Beta glucuronidase. Probiotics suggested; Meta i3c; Betaine HCL/ pepsin & other digestive enzymes; exclusion diet, glutamine/ IPS (Spanish Moss) (GUT protocol)
- Low folic acid; B12; high homocysteine level of 25, despite his excellent ONE test results. Treated with methylated folate and methyl B12.
- Low vitamin D level; treated with high dose vitamin D supplements, 5000 units a day

# Referral to Neurology

I referred him to  an excellent neurologist who revamped his Parkinson's medication and this reduced his re-ignition of symptoms as each dose of L-Dopa wore off.

Propranolol for tremor

Reduced dose of Ramipril

# DAT scans; early diagnosis

- Differentiating between Parkinson's and essential tremor – in an individual whose sole symptom is tremor, it can be difficult to make a definitive diagnosis. DAT scans are abnormal in patients with Parkinson's, but normal in patients with essential tremor.
- Differentiating between Parkinson's disease and drug-induced parkinsonism – anti-psychotic medications used to treat psychiatric illnesses (including schizophrenia) work by blocking dopamine receptors. DAT scans are normal in drug-induced parkinsonism.
- Differentiating between Parkinson's disease and psychogenic parkinsonism – occasionally individuals have motor symptoms that arise from psychological rather than neurodegenerative causes. DAT scans are normal in individuals with psychogenic parkinsonism.

# 1.Subsequent review approximately 1 year later. Clinical progress mixed

- He was not referred back to me again until June 2017 as the company were considering retiring him early. Initially he had been very much better even able to do an 18 round game of golf;  but he persisted with abdominal symptoms including bloating, nausea and sickness when eating sugary foods and that he was very disabled by this. Now only able to do 9 holes of golf; difficulty getting off a chair when meds wore off.
- ?SIBO. I organised for this to be tested with Aero diagnostics. Unfortunately, he did not complete the test.
- December 2017, SIBO test still not carried out. Clinical diagnosis of SIBO made, Rx: high-dose rifaximin 550mg bd  for one month. He felt vastly better with this treatment;

# 2. Further gut tests organised, ( November 2017). then chronic infection tests ( January 2018)

- Nov 2017 Advanced Intestinal Barrier Assessment showed HIGH LPS & Zonulin ? Infection present. H Pylori negative.
- January 2018 Armin Chronic infection test recommended.
- Armin chronic infection test confirmed the presence of Borrelia, showing a weak positive Elispot and positive sera spot tests for several different strains, nine in total. His CD57 measured 93/ul
- He was also positive for reactivating infections chlamydia pneumoniae and many viruses

Advanced Intestinal Barrier Assessment ( Invivo)

High zonulin ( gluten; infection)

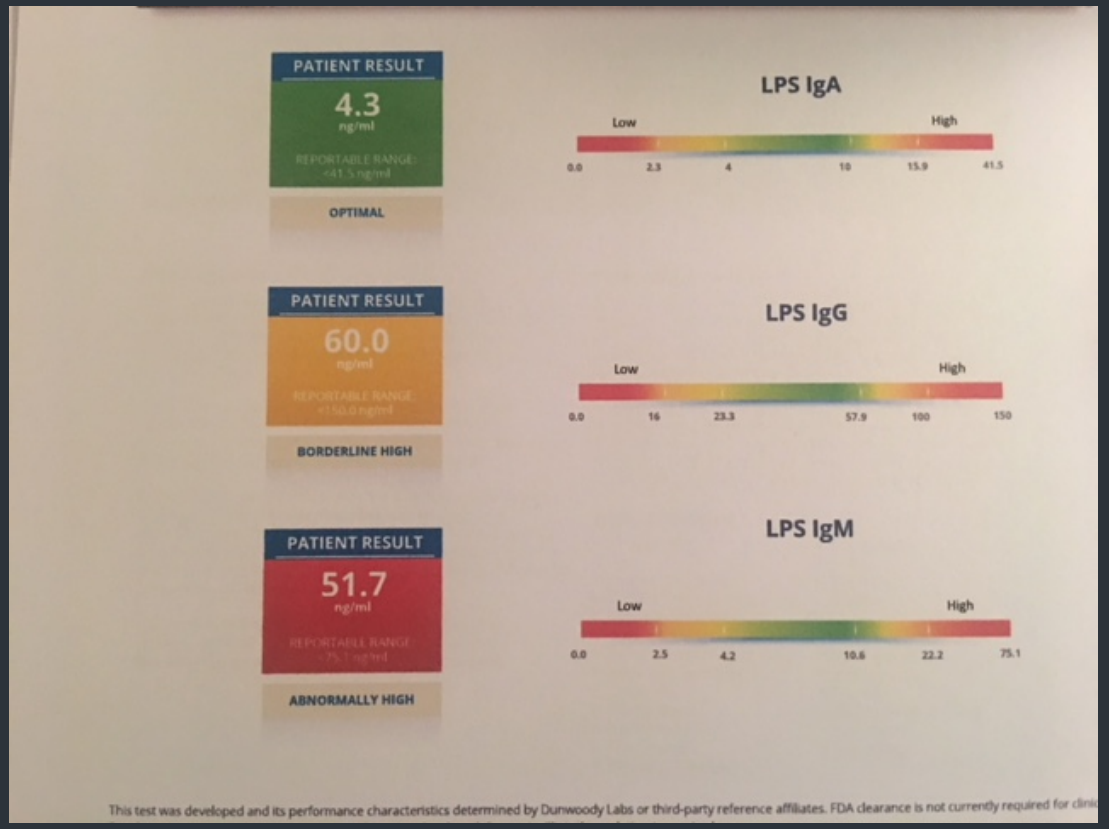
Borderline histamine but adequately  dealt with by high DAO

Lipo Polysaccharides (LPS)

Highly inflammatory Endotoxins from intraluminal gut bacteria

LPS IGM very high!

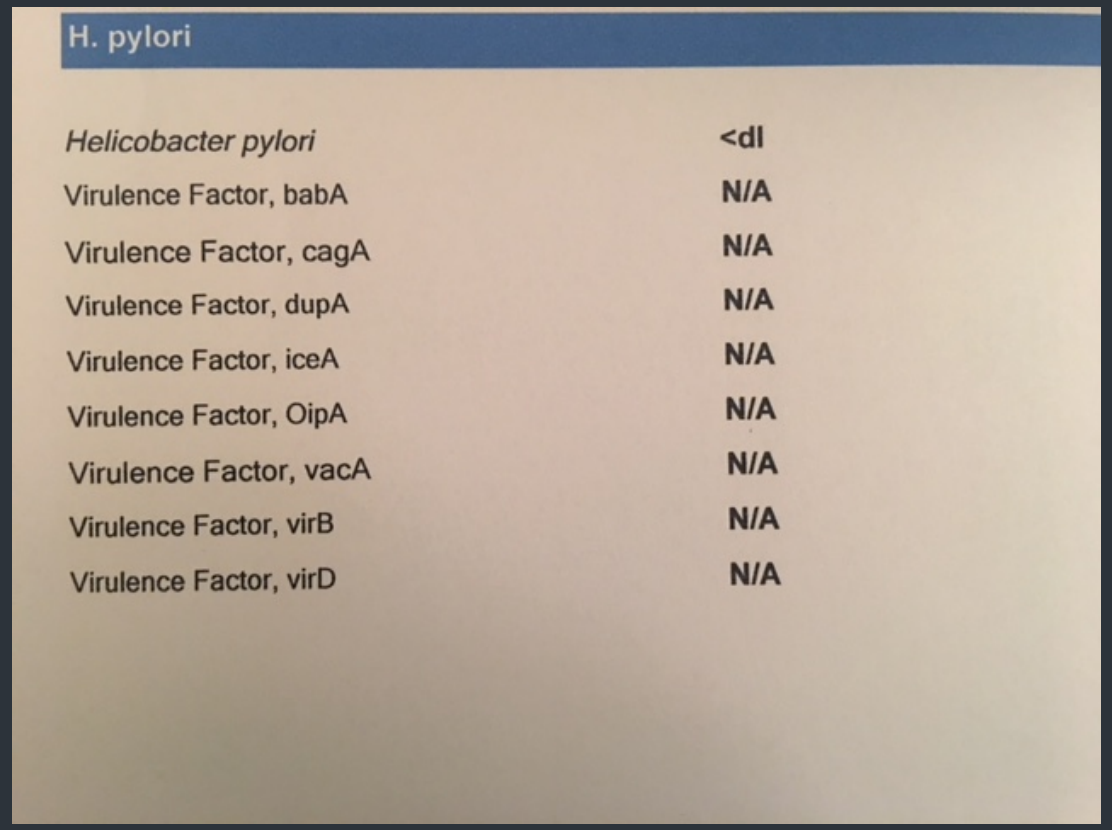
Highly inflammatory endotoxaemia



# The Fire of Inflammation

H Pylori ( Invivo)

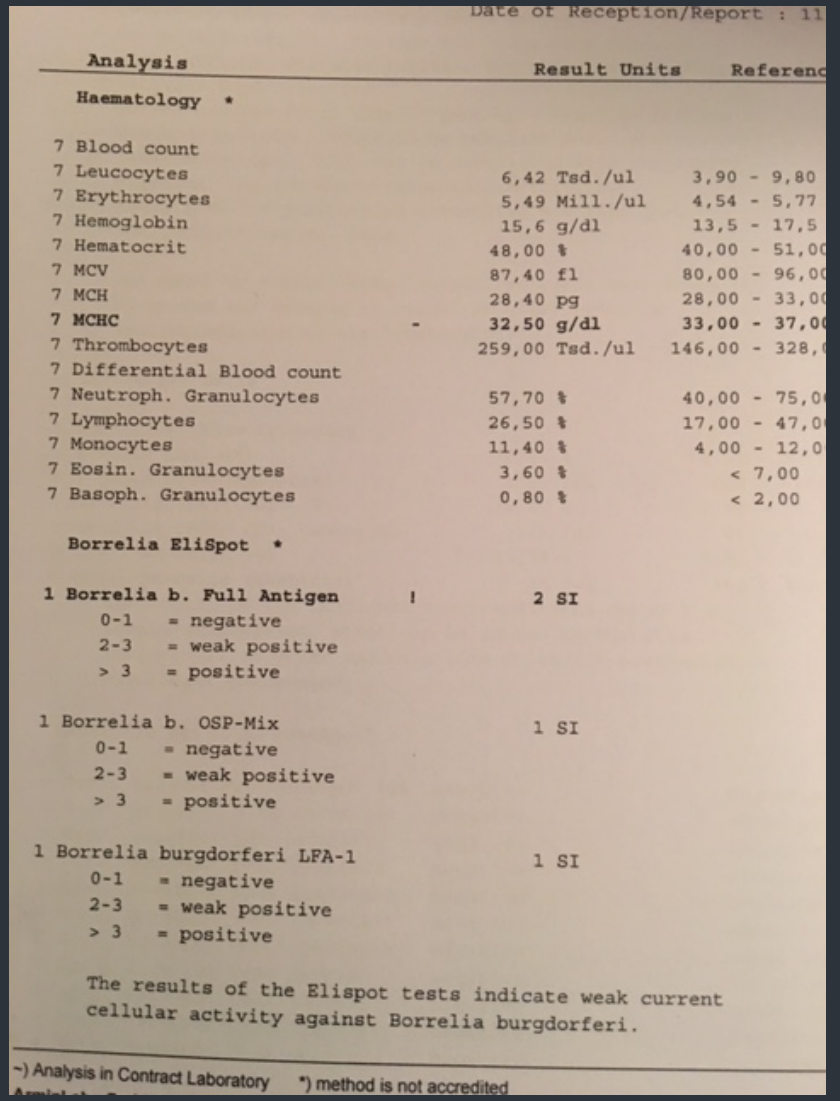
# Negative



| H. pylori | |
|---|---|
| *Helicobacter pylori* | <dl |
| Virulence Factor, babA | N/A |
| Virulence Factor, cagA | N/A |
| Virulence Factor, dupA | N/A |
| Virulence Factor, iceA | N/A |
| Virulence Factor, OipA | N/A |
| Virulence Factor, vacA | N/A |
| Virulence Factor, virB | N/A |
| Virulence Factor, virD | N/A |

Armin Chronic Infections test

Borrelia weak Elispot positive



Date of Reception/Report : 11

| Analysis | | Result | Units | Referenc |
|---|---|---|---|---|
| **Haematology** * | | | | |
| 7 Blood count | | | | |
| 7 Leucocytes | | 6,42 | Tsd./ul | 3,90 - 9,80 |
| 7 Erythrocytes | | 5,49 | Mill./ul | 4,54 - 5,77 |
| 7 Hemoglobin | | 15,6 | g/dl | 13,5 - 17,5 |
| 7 Hematocrit | | 48,00 | % | 40,00 - 51,0 |
| 7 MCV | | 87,40 | fl | 80,00 - 96,0 |
| 7 MCH | | 28,40 | pg | 28,00 - 33,0 |
| **7 MCHC** | - | **32,50** | **g/dl** | **33,00 - 37,0** |
| 7 Thrombocytes | | 259,00 | Tsd./ul | 146,00 - 328, |
| 7 Differential Blood count | | | | |
| 7 Neutroph. Granulocytes | | 57,70 | % | 40,00 - 75,0 |
| 7 Lymphocytes | | 26,50 | % | 17,00 - 47,0 |
| 7 Monocytes | | 11,40 | % | 4,00 - 12,0 |
| 7 Eosin. Granulocytes | | 3,60 | % | < 7,00 |
| 7 Basoph. Granulocytes | | 0,80 | % | < 2,00 |
| **Borrelia EliSpot** * | | | | |
| **1 Borrelia b. Full Antigen** | ! | **2** | **SI** | |
| 0-1 = negative | | | | |
| 2-3 = weak positive | | | | |
| > 3 = positive | | | | |
| 1 Borrelia b. OSP-Mix | | 1 | SI | |
| 0-1 = negative | | | | |
| 2-3 = weak positive | | | | |
| > 3 = positive | | | | |
| 1 Borrelia burgdorferi LFA-1 | | 1 | SI | |
| 0-1 = negative | | | | |
| 2-3 = weak positive | | | | |
| > 3 = positive | | | | |

The results of the Elispot tests indicate weak current cellular activity against Borrelia burgdorferi.

~) Analysis in Contract Laboratory *) method is not accredited

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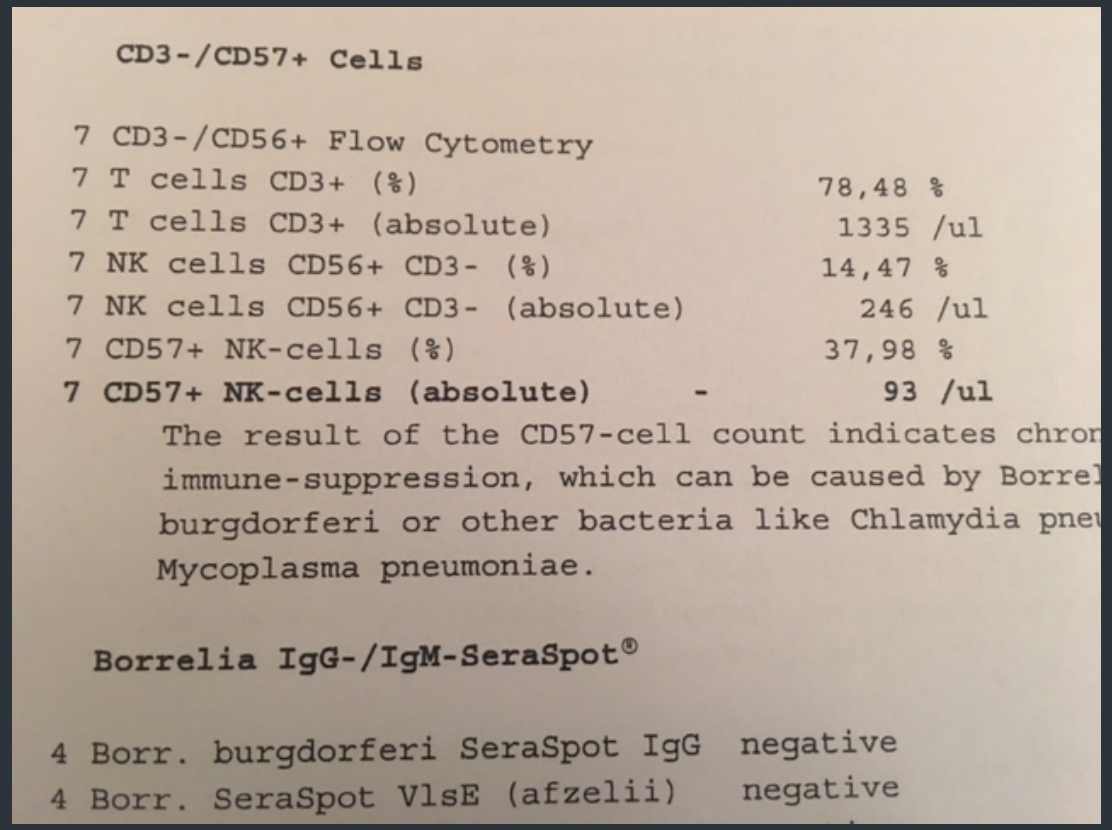
Lowish CD 57

Indicates chronic immune suppression by Borrelia, chlamydia pneumoniae; or mycoplasma pneumoniae; or viruses eg EBV



**CD3-/CD57+ Cells**

| | | | |
|---|---|---|---|
| 7 | CD3-/CD56+ Flow Cytometry | | |
| 7 | T cells CD3+ (%) | 78,48 | % |
| 7 | T cells CD3+ (absolute) | 1335 | /ul |
| 7 | NK cells CD56+ CD3- (%) | 14,47 | % |
| 7 | NK cells CD56+ CD3- (absolute) | 246 | /ul |
| 7 | CD57+ NK-cells (%) | 37,98 | % |
| **7** | **CD57+ NK-cells (absolute)** - | **93** | **/ul** |

The result of the CD57-cell count indicates chron
immune-suppression, which can be caused by Borrel
burgdorferi or other bacteria like Chlamydia pneu
Mycoplasma pneumoniae.

**Borrelia IgG-/IgM-SeraSpot®**

| | | |
|---|---|---|
| 4 | Borr. burgdorferi SeraSpot IgG | negative |
| 4 | Borr. SeraSpot VlsE (afzelii) | negative |

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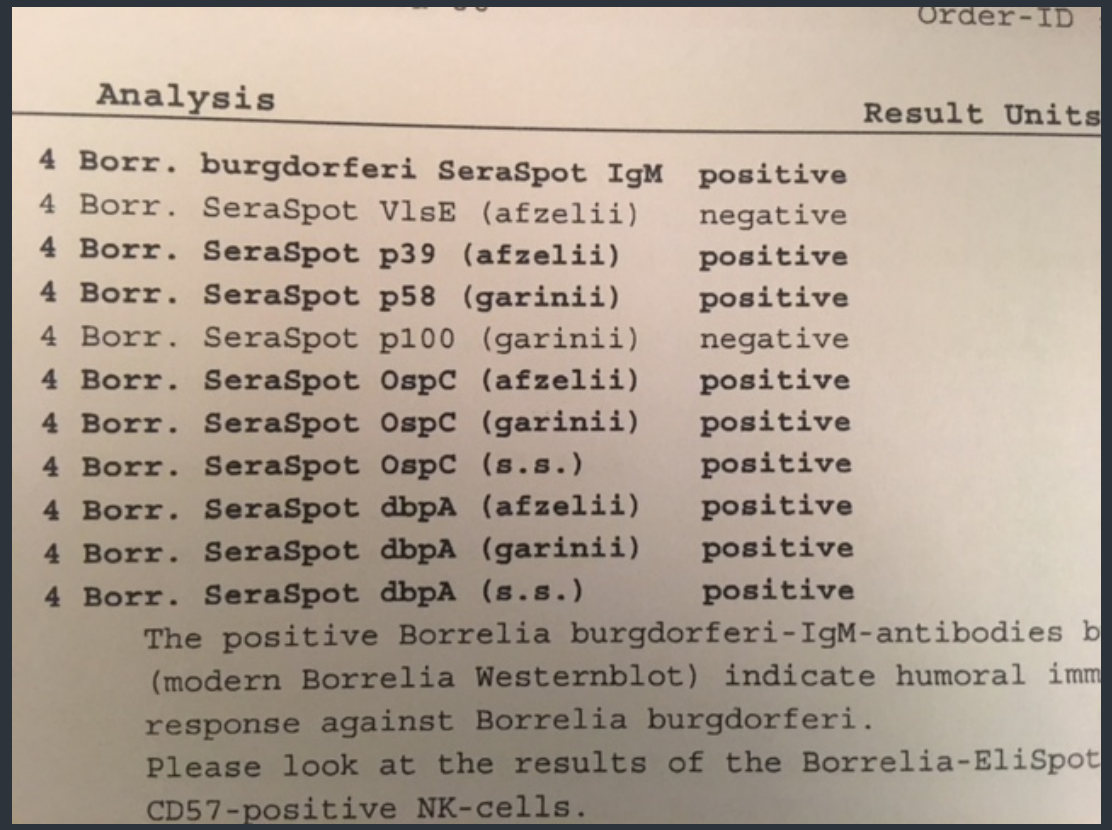
# 9 BORRELIA Seraspot positive results IGM

(humoral response)



Order-ID

| Analysis | Result | Units |
|---|---|---|
| 4 Borr. burgdorferi SeraSpot IgM | positive | |
| 4 Borr. SeraSpot VlsE (afzelii) | negative | |
| 4 Borr. SeraSpot p39 (afzelii) | positive | |
| 4 Borr. SeraSpot p58 (garinii) | positive | |
| 4 Borr. SeraSpot p100 (garinii) | negative | |
| 4 Borr. SeraSpot OspC (afzelii) | positive | |
| 4 Borr. SeraSpot OspC (garinii) | positive | |
| 4 Borr. SeraSpot OspC (s.s.) | positive | |
| 4 Borr. SeraSpot dbpA (afzelii) | positive | |
| 4 Borr. SeraSpot dbpA (garinii) | positive | |
| 4 Borr. SeraSpot dbpA (s.s.) | positive | |

The positive Borrelia burgdorferi-IgM-antibodies b
(modern Borrelia Westernblot) indicate humoral imm
response against Borrelia burgdorferi.
Please look at the results of the Borrelia-EliSpot
CD57-positive NK-cells.

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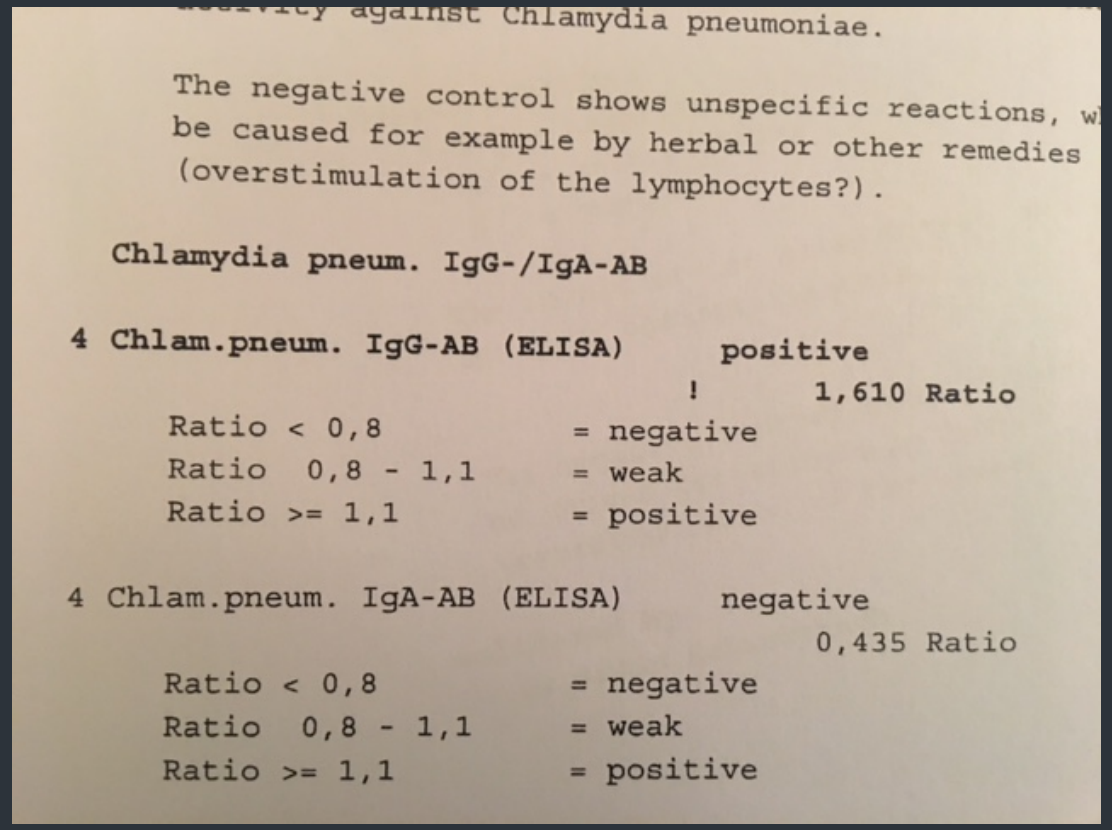
# Chlamydia IGG positive

? Reactivating infection



activity against Chlamydia pneumoniae.

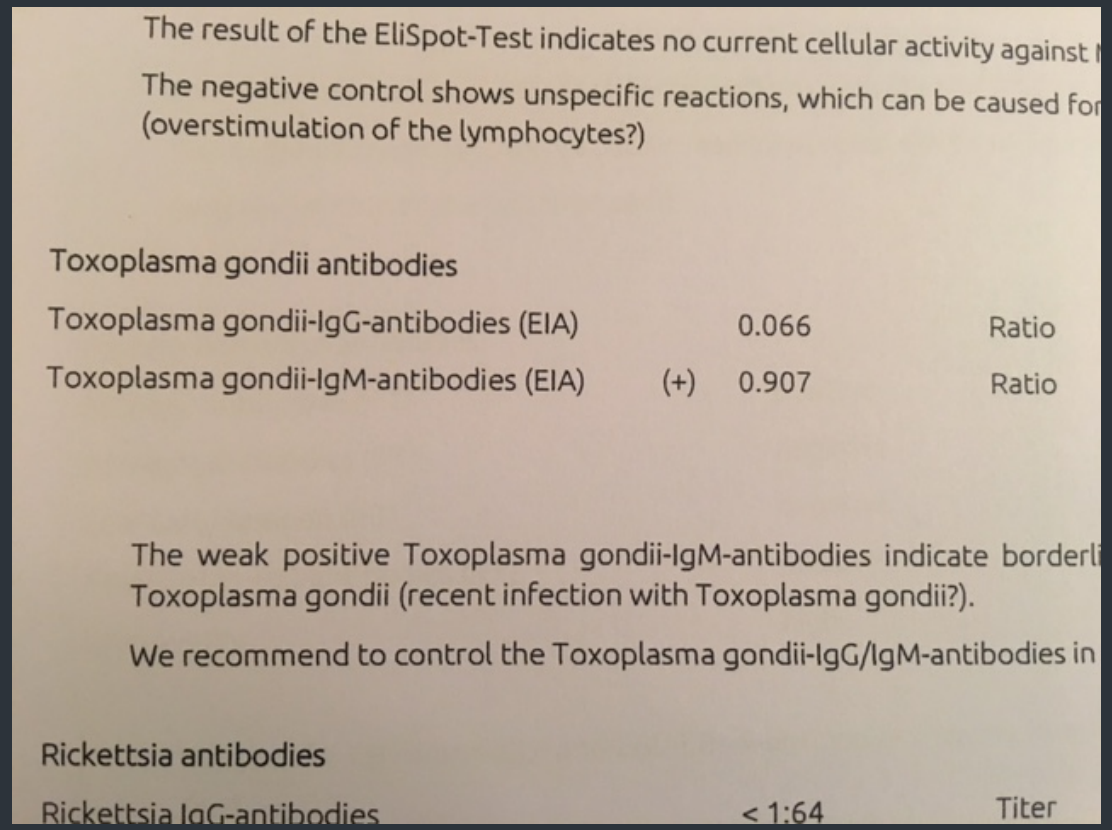
The negative control shows unspecific reactions, w
be caused for example by herbal or other remedies
(overstimulation of the lymphocytes?).

**Chlamydia pneum. IgG-/IgA-AB**

| | | | |
|---|---|---|---|
| **4** | **Chlam.pneum. IgG-AB (ELISA)** | **positive** | |
| | | **!** | **1,610 Ratio** |
| | Ratio < 0,8 | = negative | |
| | Ratio 0,8 - 1,1 | = weak | |
| | Ratio >= 1,1 | = positive | |
| 4 | Chlam.pneum. IgA-AB (ELISA) | negative | |
| | | | 0,435 Ratio |
| | Ratio < 0,8 | = negative | |
| | Ratio 0,8 - 1,1 | = weak | |
| | Ratio >= 1,1 | = positive | |

Toxoplasmosis

# Weak positive; borderline current infection



The result of the EliSpot-Test indicates no current cellular activity against

The negative control shows unspecific reactions, which can be caused for (overstimulation of the lymphocytes?)

| Toxoplasma gondii antibodies | | | |
|---|---|---|---|
| Toxoplasma gondii-IgG-antibodies (EIA) | | 0.066 | Ratio |
| Toxoplasma gondii-IgM-antibodies (EIA) | (+) | 0.907 | Ratio |

The weak positive Toxoplasma gondii-IgM-antibodies indicate borderli Toxoplasma gondii (recent infection with Toxoplasma gondii?).

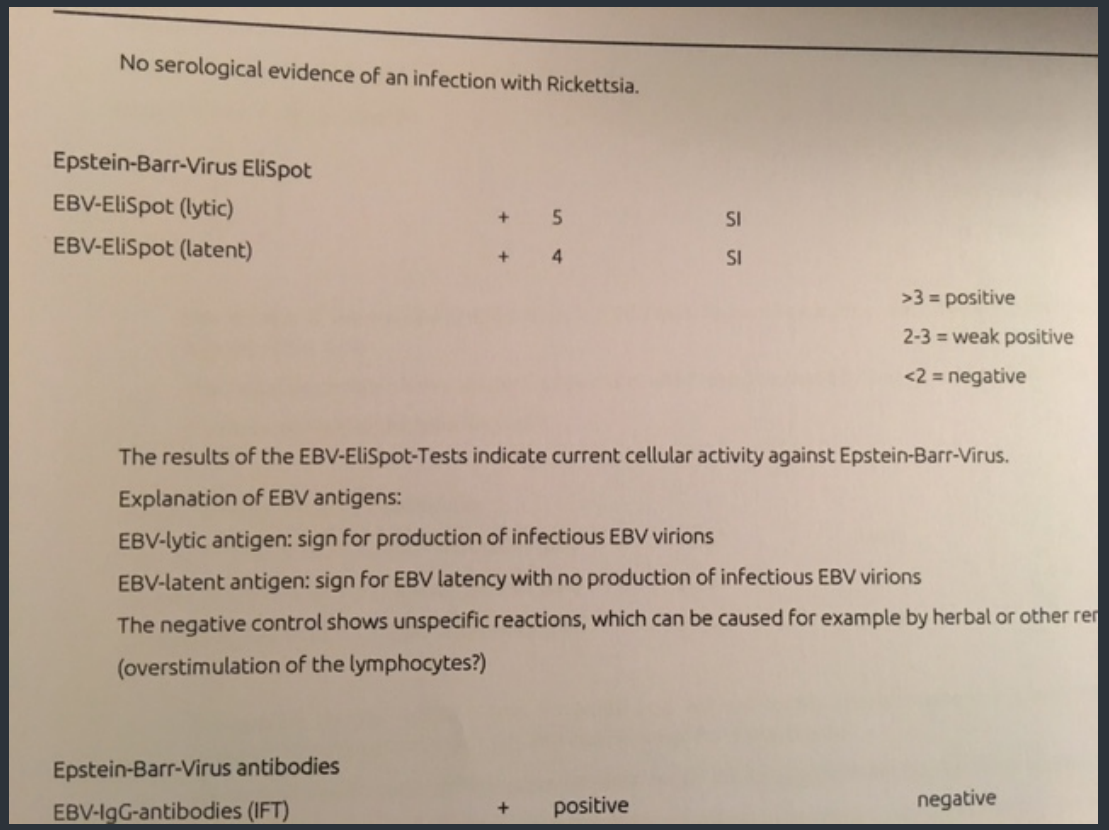
We recommend to control the Toxoplasma gondii-IgG/IgM-antibodies in

| Rickettsia antibodies | | | |
|---|---|---|---|
| Rickettsia IgG-antibodies | | < 1:64 | Titer |

Elispot Epstein Barr Virus

# Current cellular activity

# Negative control showed unspecified reactions ? Herbal overstimulation of lymphocytes



No serological evidence of an infection with Rickettsia.

| | | | |
|---|---|---|---|
| Epstein-Barr-Virus EliSpot | | | |
| EBV-EliSpot (lytic) | + | 5 | SI |
| EBV-EliSpot (latent) | + | 4 | SI |

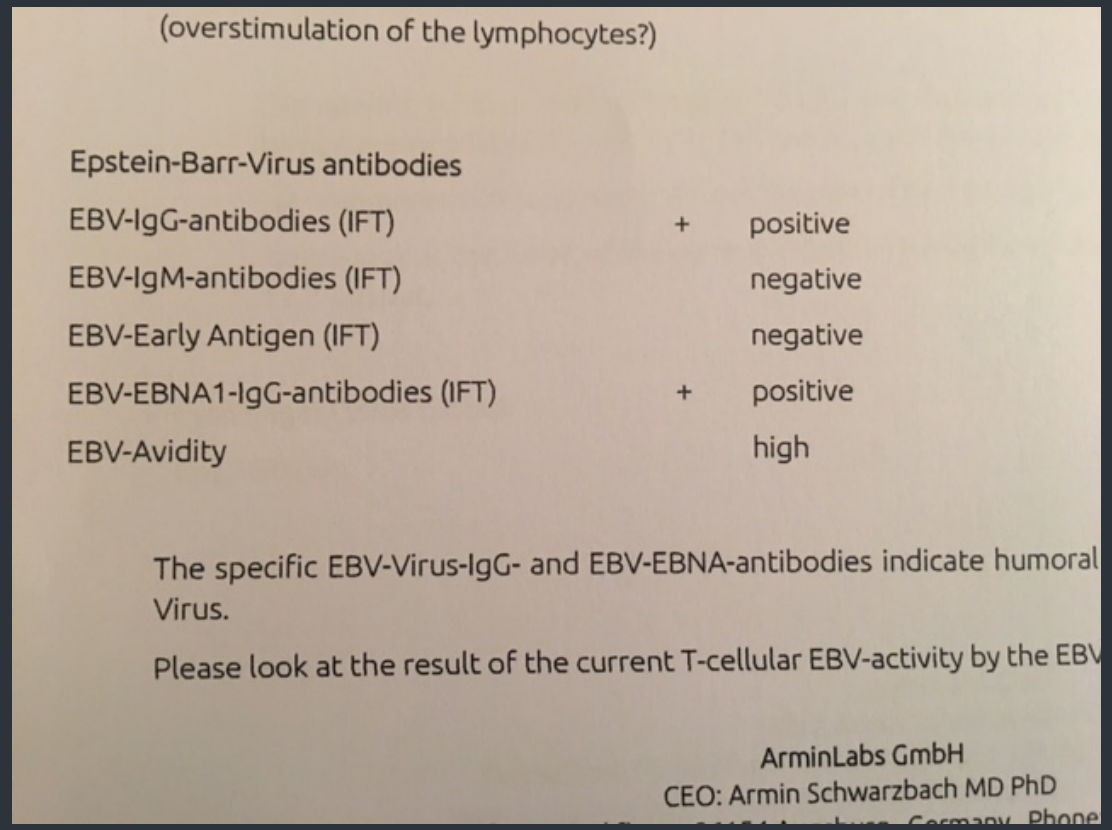
>3 = positive
2-3 = weak positive
<2 = negative

The results of the EBV-EliSpot-Tests indicate current cellular activity against Epstein-Barr-Virus.
Explanation of EBV antigens:
EBV-lytic antigen: sign for production of infectious EBV virions
EBV-latent antigen: sign for EBV latency with no production of infectious EBV virions
The negative control shows unspecific reactions, which can be caused for example by herbal or other rer
(overstimulation of the lymphocytes?)

| | | | |
|---|---|---|---|
| Epstein-Barr-Virus antibodies | | | |
| EBV-IgG-antibodies (IFT) | + | positive | negative |

EBV

# Positive humoral response



(overstimulation of the lymphocytes?)

| Epstein-Barr-Virus antibodies | | |
|---|---|---|
| EBV-IgG-antibodies (IFT) | + | positive |
| EBV-IgM-antibodies (IFT) | | negative |
| EBV-Early Antigen (IFT) | | negative |
| EBV-EBNA1-IgG-antibodies (IFT) | + | positive |
| EBV-Avidity | | high |

The specific EBV-Virus-IgG- and EBV-EBNA-antibodies indicate humoral
Virus.

Please look at the result of the current T-cellular EBV-activity by the EBV

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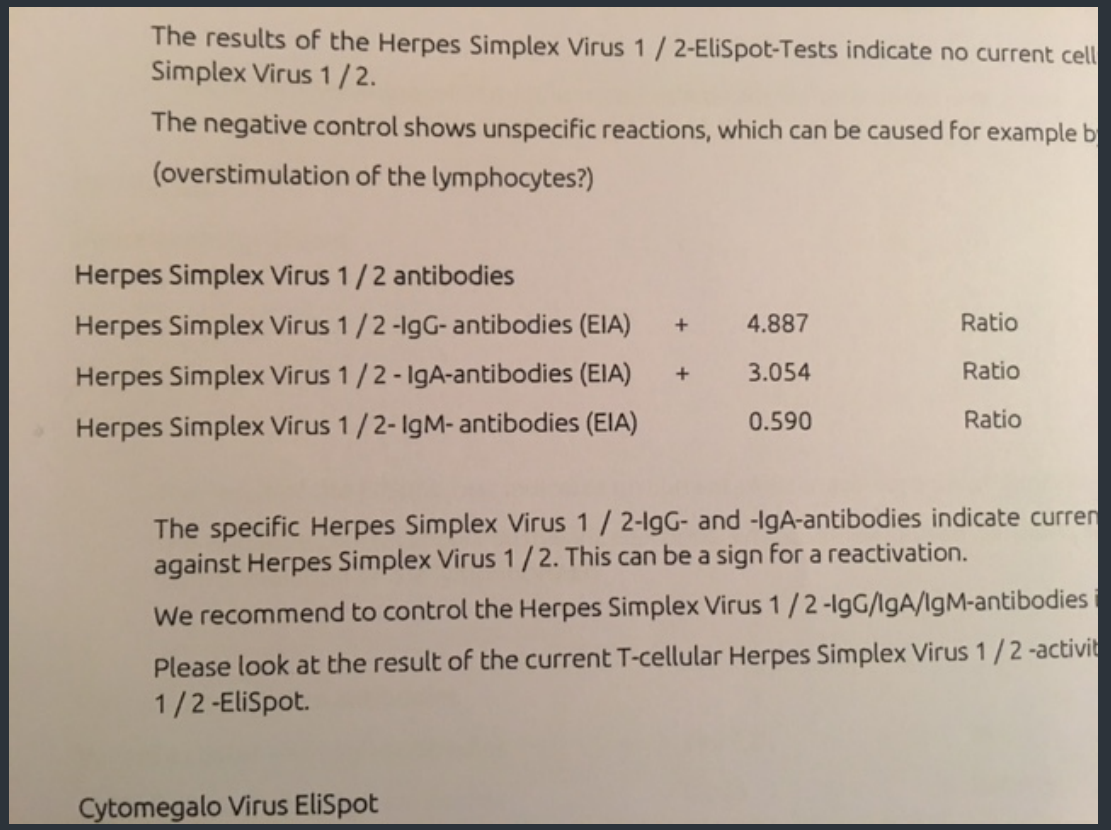
CEO: Armin Schwarzbach MD PhD

Herpes Simplex

# Current reactivation?



The results of the Herpes Simplex Virus 1 / 2-EliSpot-Tests indicate no current cell
Simplex Virus 1 / 2.

The negative control shows unspecific reactions, which can be caused for example b

(overstimulation of the lymphocytes?)

| Herpes Simplex Virus 1 / 2 antibodies | | | |
|---|---|---|---|
| Herpes Simplex Virus 1 / 2 -IgG- antibodies (EIA) | + | 4.887 | Ratio |
| Herpes Simplex Virus 1 / 2 - IgA-antibodies (EIA) | + | 3.054 | Ratio |
| Herpes Simplex Virus 1 / 2- IgM- antibodies (EIA) | | 0.590 | Ratio |

The specific Herpes Simplex Virus 1 / 2-IgG- and -IgA-antibodies indicate curren
against Herpes Simplex Virus 1 / 2. This can be a sign for a reactivation.

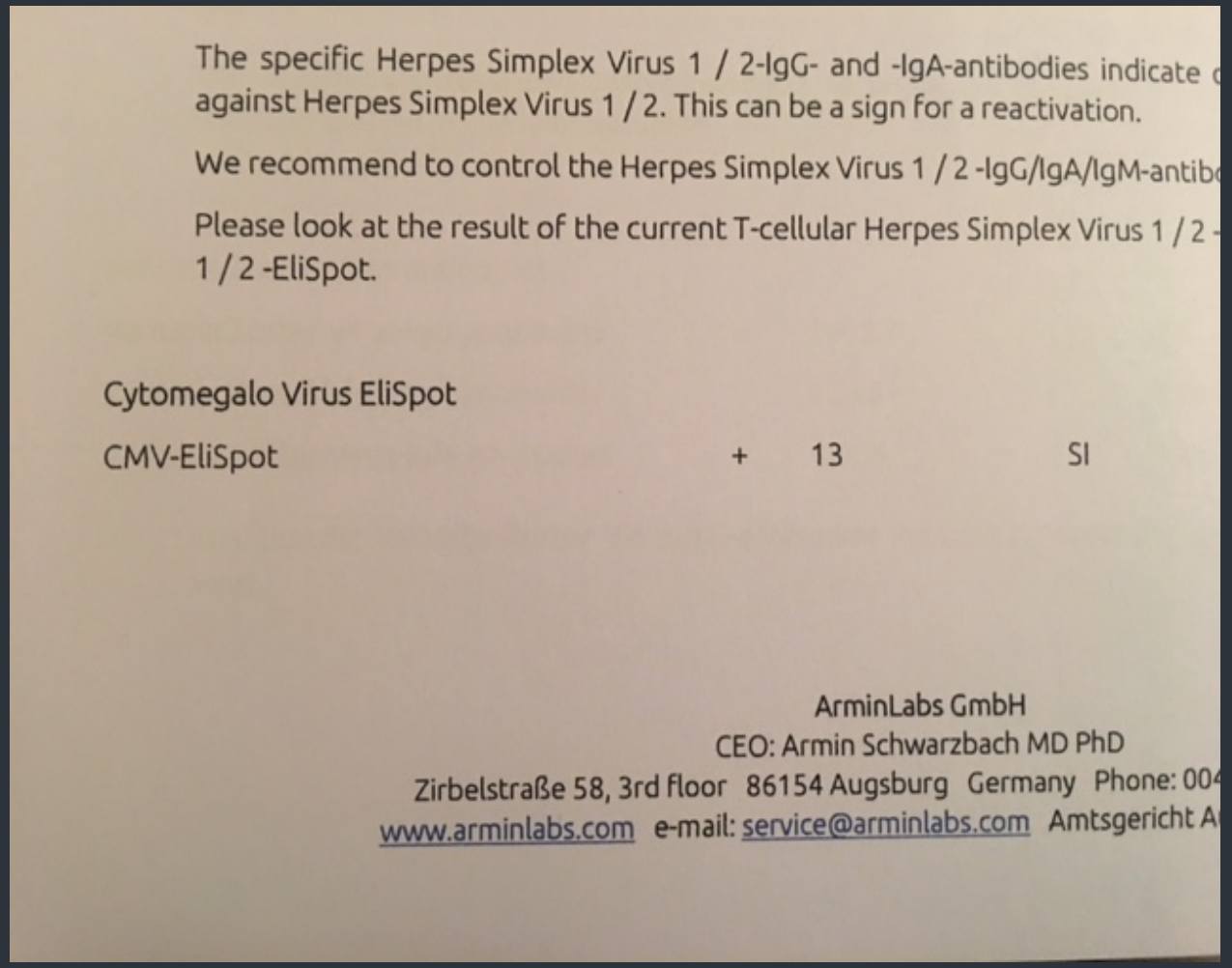
We recommend to control the Herpes Simplex Virus 1 / 2 -IgG/IgA/IgM-antibodies

Please look at the result of the current T-cellular Herpes Simplex Virus 1 / 2 -activit
1 / 2 -EliSpot.

Cytomegalo Virus EliSpot

Cytomegalovirus (CMV)

# Elispot high, current active cellular immunity



The specific Herpes Simplex Virus 1 / 2-IgG- and -IgA-antibodies indicate
against Herpes Simplex Virus 1 / 2. This can be a sign for a reactivation.

We recommend to control the Herpes Simplex Virus 1 / 2 -IgG/IgA/IgM-antib

Please look at the result of the current T-cellular Herpes Simplex Virus 1 / 2
1 / 2 -EliSpot.

| Cytomegalo Virus EliSpot | | | |
|---|---|---|---|
| CMV-EliSpot | + | 13 | SI |

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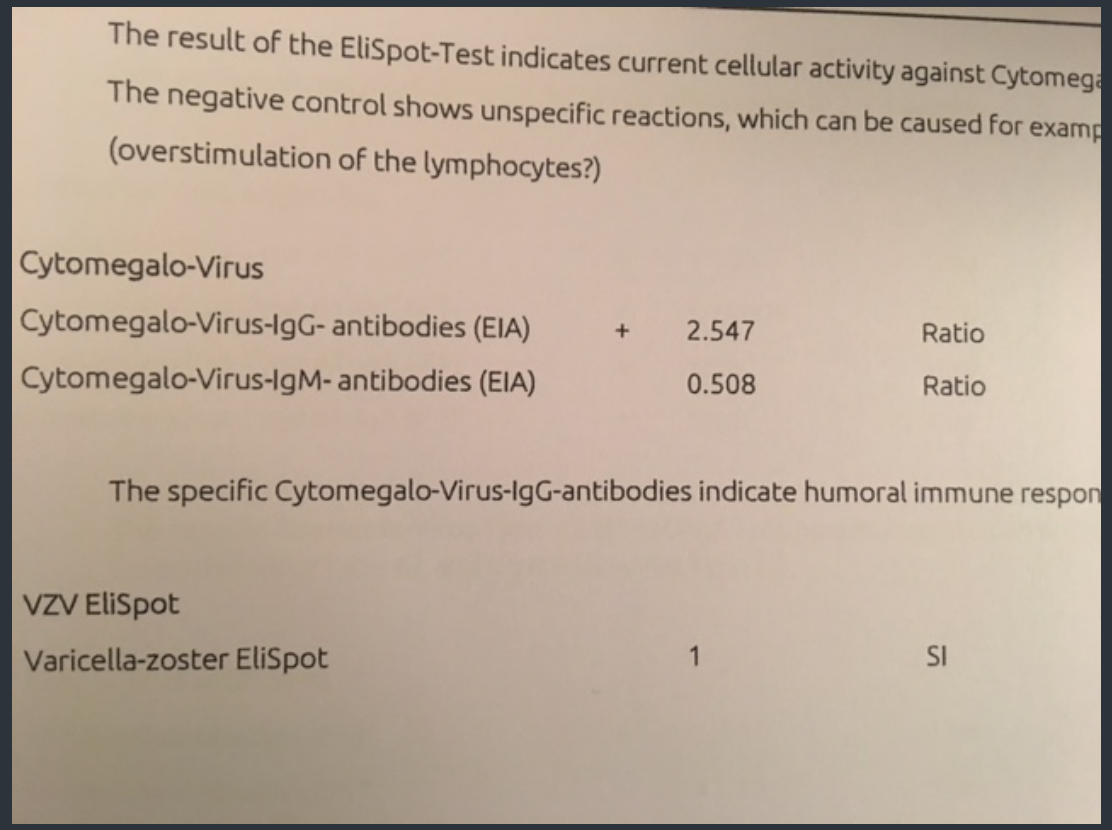
Arminlabs CMV

IGG antibodies  indicate humoral response



The result of the EliSpot-Test indicates current cellular activity against Cytomega
The negative control shows unspecific reactions, which can be caused for examp
(overstimulation of the lymphocytes?)

| | | | |
|---|---|---|---|
| Cytomegalo-Virus | | | |
| Cytomegalo-Virus-IgG- antibodies (EIA) | + | 2.547 | Ratio |
| Cytomegalo-Virus-IgM- antibodies (EIA) | | 0.508 | Ratio |

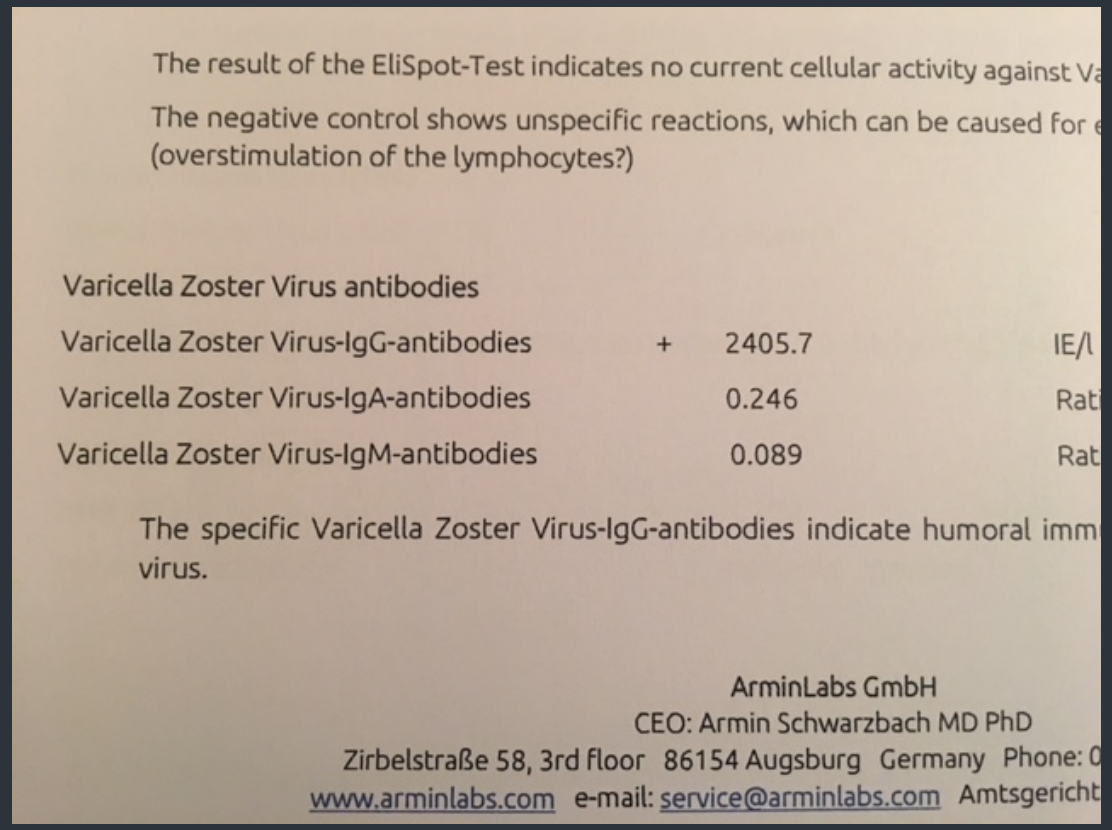
The specific Cytomegalo-Virus-IgG-antibodies indicate humoral immune respon

| | | |
|---|---|---|
| VZV EliSpot | | |
| Varicella-zoster EliSpot | 1 | SI |

Arminlabs Herpes Zoster

VZV IgG antibodies positive

Humoral response



The result of the EliSpot-Test indicates no current cellular activity against Va

The negative control shows unspecific reactions, which can be caused for e (overstimulation of the lymphocytes?)

| Varicella Zoster Virus antibodies | | | |
|---|---|---|---|
| Varicella Zoster Virus-IgG-antibodies | + | 2405.7 | IE/l |
| Varicella Zoster Virus-IgA-antibodies | | 0.246 | Rati |
| Varicella Zoster Virus-IgM-antibodies | | 0.089 | Rat |

The specific Varicella Zoster Virus-IgG-antibodies indicate humoral imm virus.

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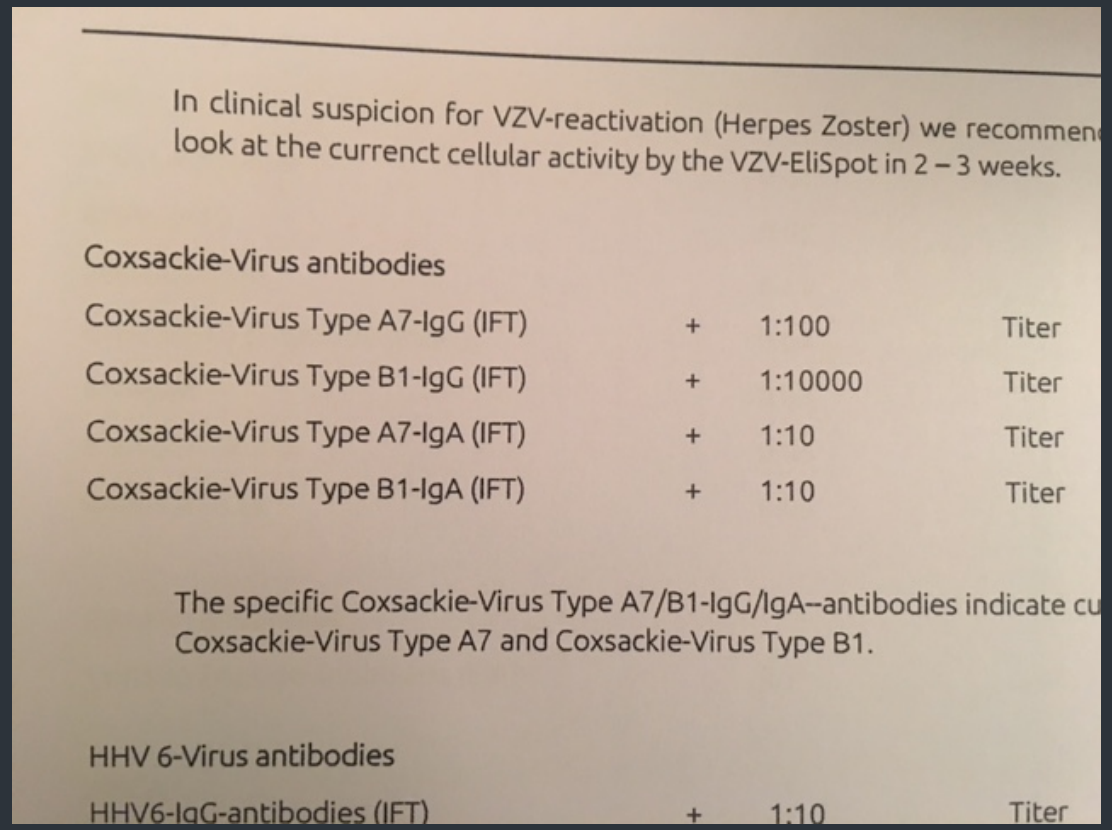
Coxsackievirus antibodies

Specific Coxsackie virus type A7/B1 – IgG/IgA, indicate current humoral activity against these organisms



In clinical suspicion for VZV-reactivation (Herpes Zoster) we recommen
look at the currenct cellular activity by the VZV-EliSpot in 2 – 3 weeks.

| Coxsackie-Virus antibodies | | | |
|---|---|---|---|
| Coxsackie-Virus Type A7-IgG (IFT) | + | 1:100 | Titer |
| Coxsackie-Virus Type B1-IgG (IFT) | + | 1:10000 | Titer |
| Coxsackie-Virus Type A7-IgA (IFT) | + | 1:10 | Titer |
| Coxsackie-Virus Type B1-IgA (IFT) | + | 1:10 | Titer |

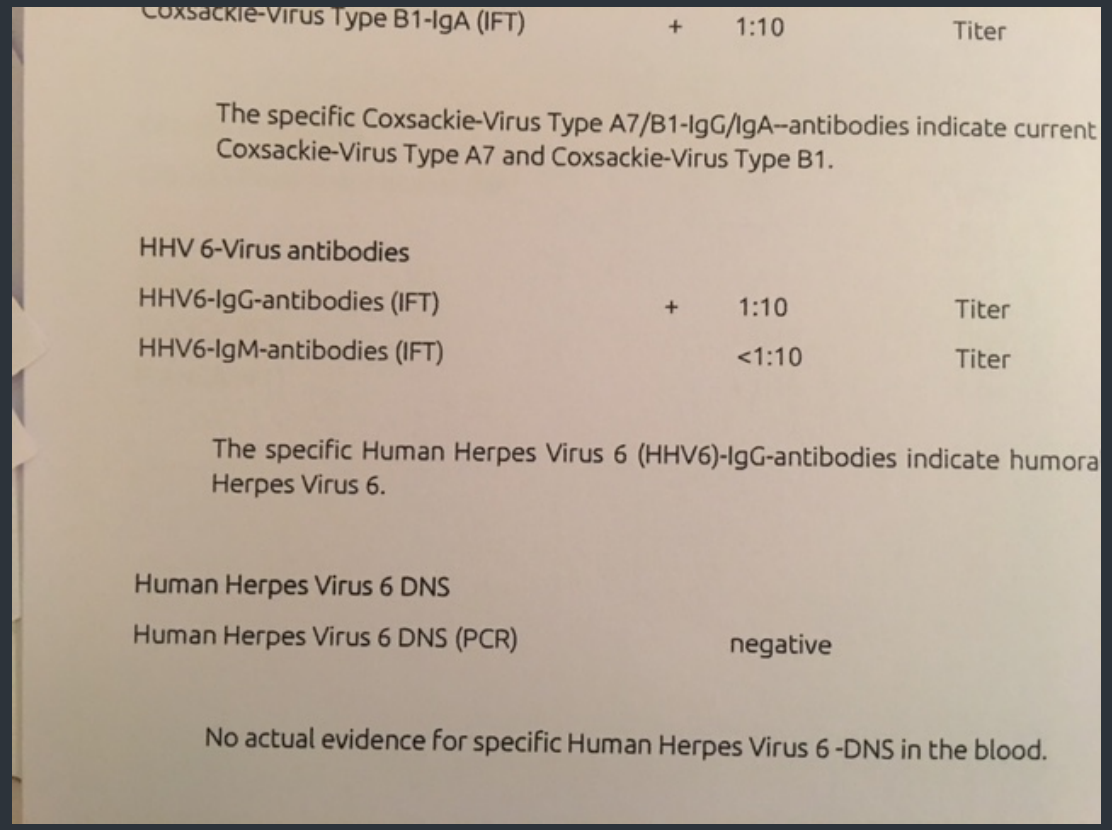
The specific Coxsackie-Virus Type A7/B1-IgG/IgA--antibodies indicate cu
Coxsackie-Virus Type A7 and Coxsackie-Virus Type B1.

| HHV 6-Virus antibodies | | | |
|---|---|---|---|
| HHV6-IgG-antibodies (IFT) | + | 1:10 | Titer |

# HHV-6 (Human herpes virus 6) antibodies

Positive, indicating humoral response against HHV6

No actual evidence for HHV-6 – DNA in the blood



| | | | |
|---|---|---|---|
| Coxsackie-Virus Type B1-IgA (IFT) | + | 1:10 | Titer |

The specific Coxsackie-Virus Type A7/B1-IgG/IgA--antibodies indicate current Coxsackie-Virus Type A7 and Coxsackie-Virus Type B1.

HHV 6-Virus antibodies

| | | | |
|---|---|---|---|
| HHV6-IgG-antibodies (IFT) | + | 1:10 | Titer |
| HHV6-IgM-antibodies (IFT) | | <1:10 | Titer |

The specific Human Herpes Virus 6 (HHV6)-IgG-antibodies indicate humora Herpes Virus 6.

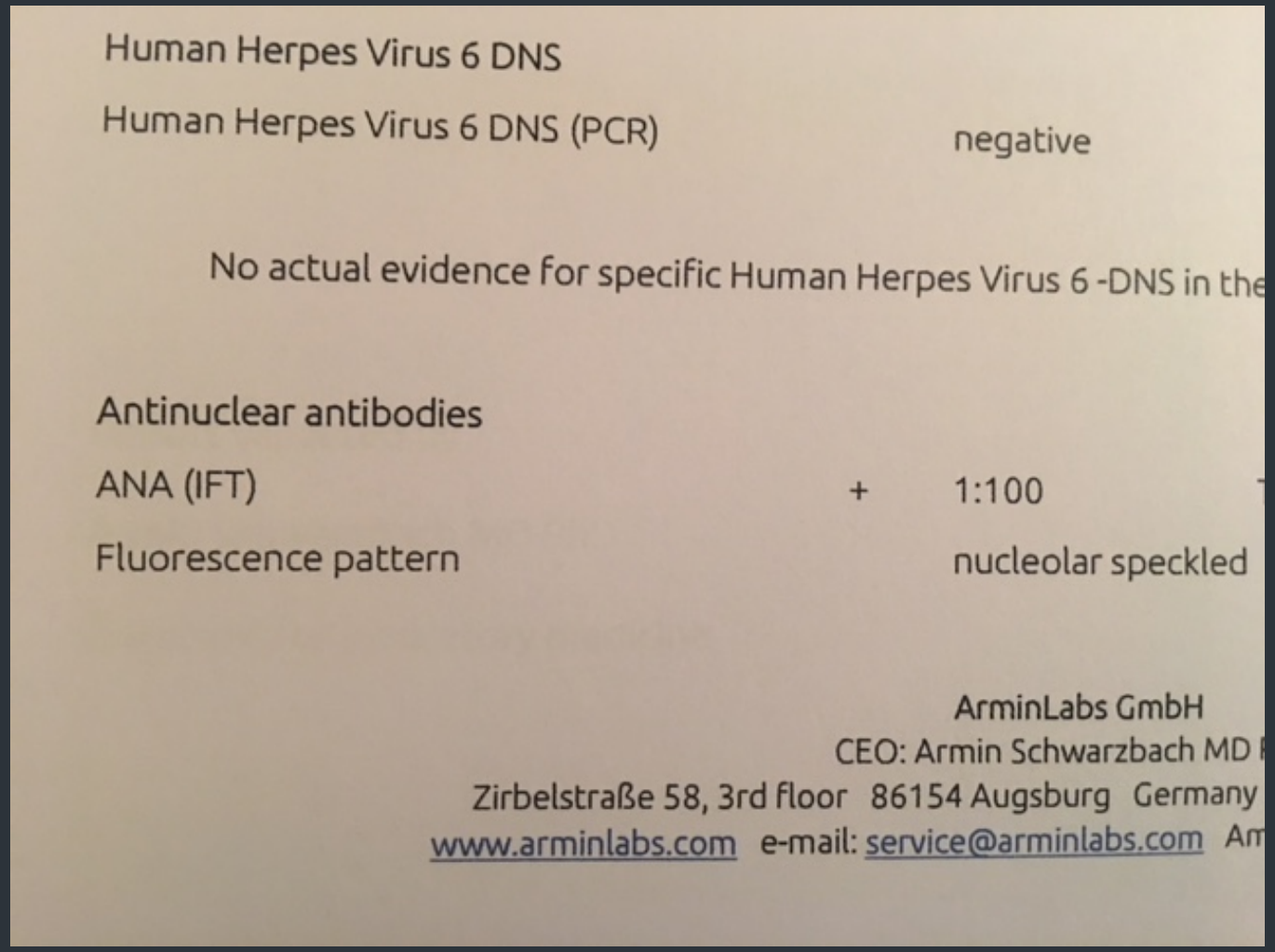
Human Herpes Virus 6 DNS

| | |
|---|---|
| Human Herpes Virus 6 DNS (PCR) | negative |

No actual evidence for specific Human Herpes Virus 6 -DNS in the blood.

# Arminlabs Autoimmunity

ANA positive



Human Herpes Virus 6 DNS

Human Herpes Virus 6 DNS (PCR) negative

No actual evidence for specific Human Herpes Virus 6 -DNS in the

Antinuclear antibodies

ANA (IFT) + 1:100

Fluorescence pattern nucleolar speckled

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# 3rd December 2017

- Further functional methylation tests
- Mitochondrial assessment

## Functional test of how methylation is actually working ( Invivo)

Low methionine

High homocysteine

Low reduced glutathione

Various types of folate & folic acid, deficient incl. folinic acid; active folate & methylated folate

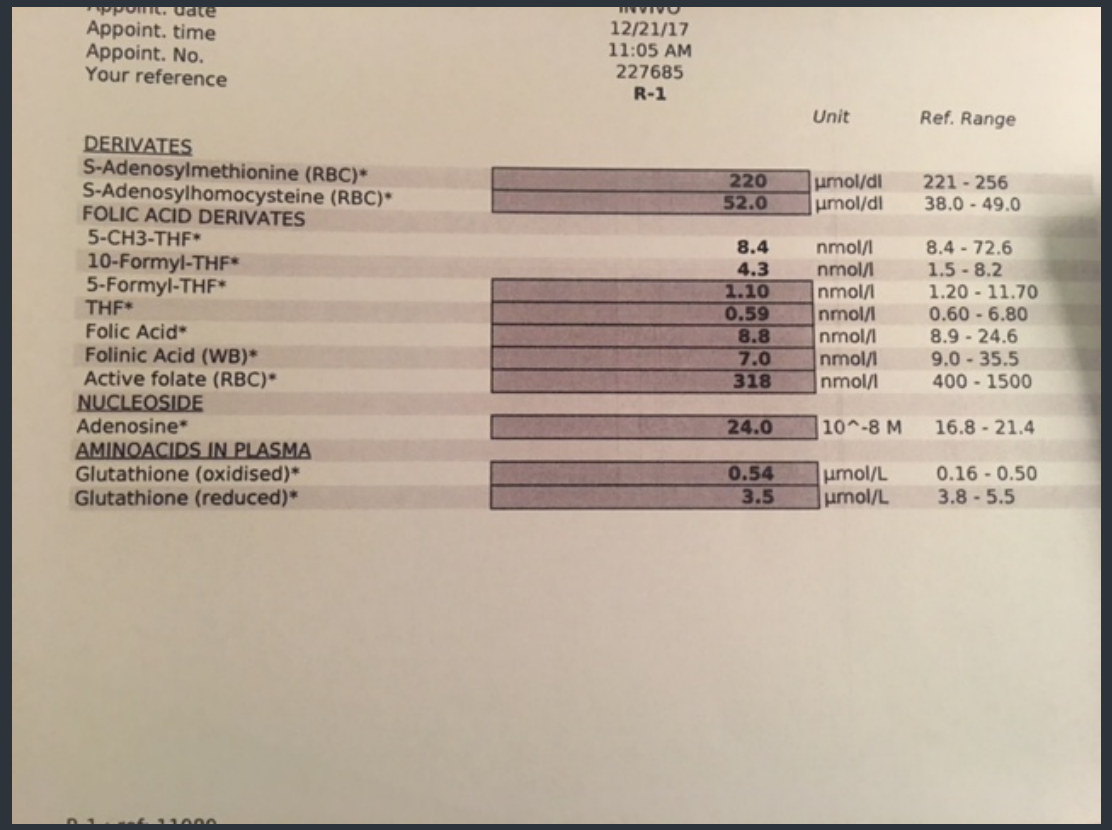
Why?

MTHFR -/- ( rate limiting)

Is there a methylation block.

? Check LIFECODE methylation genes

? Metals, infection, toxins



| | | | | |
|---|---|---|---|---|
| Appoint. date | | INVIVO | | |
| Appoint. time | | 12/21/17 | | |
| Appoint. No. | | 11:05 AM | | |
| Your reference | | 227685 | | |
| | | R-1 | | |
| | | | *Unit* | *Ref. Range* |
| DERIVATES | | | | |
| S-Adenosylmethionine (RBC)* | | 220 | µmol/dl | 221 - 256 |
| S-Adenosylhomocysteine (RBC)* | | 52.0 | µmol/dl | 38.0 - 49.0 |
| FOLIC ACID DERIVATES | | | | |
| 5-CH3-THF* | | 8.4 | nmol/l | 8.4 - 72.6 |
| 10-Formyl-THF* | | 4.3 | nmol/l | 1.5 - 8.2 |
| 5-Formyl-THF* | | 1.10 | nmol/l | 1.20 - 11.70 |
| THF* | | 0.59 | nmol/l | 0.60 - 6.80 |
| Folic Acid* | | 8.8 | nmol/l | 8.9 - 24.6 |
| Folinic Acid (WB)* | | 7.0 | nmol/l | 9.0 - 35.5 |
| Active folate (RBC)* | | 318 | nmol/l | 400 - 1500 |
| NUCLEOSIDE | | | | |
| Adenosine* | | 24.0 | 10^-8 M | 16.8 - 21.4 |
| AMINOACIDS IN PLASMA | | | | |
| Glutathione (oxidised)* | | 0.54 | µmol/L | 0.16 - 0.50 |
| Glutathione (reduced)* | | 3.5 | µmol/L | 3.8 - 5.5 |

# Acumen Labs; deeper cellular mitochondrial function

Low whole cell ATP

Low Mitochondrial ATP

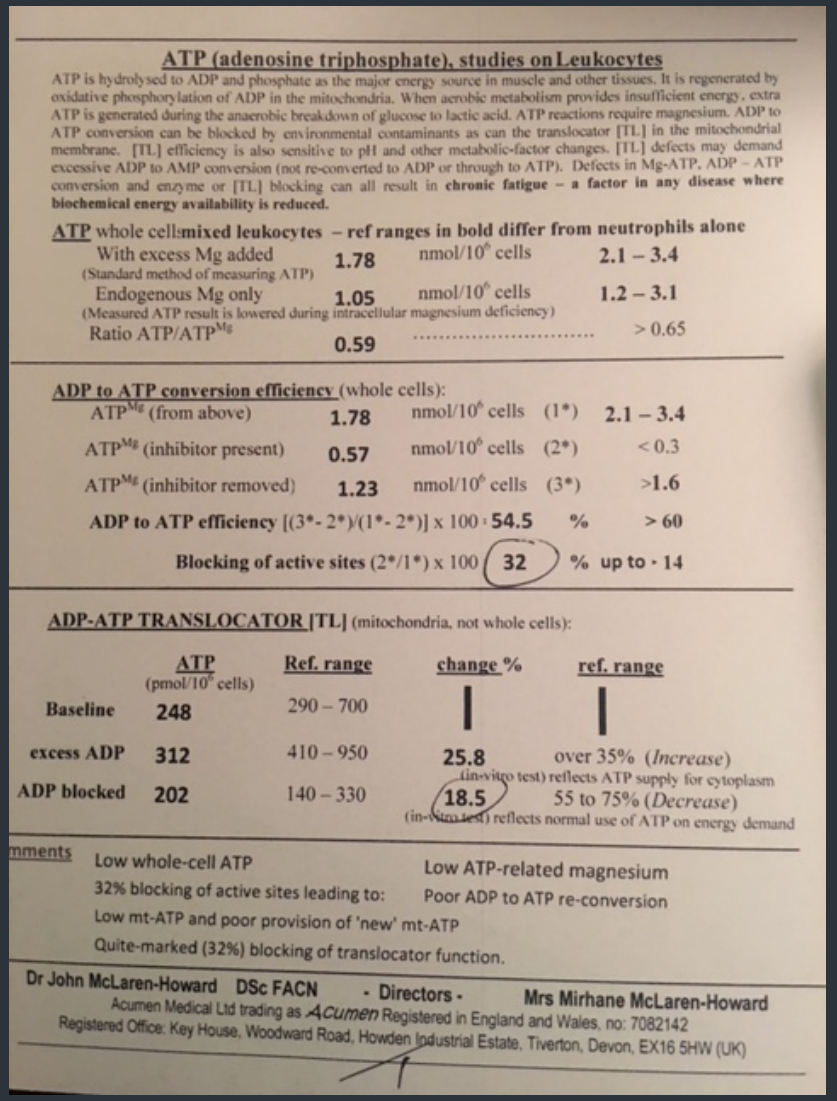
Poor conversion of new Mitochondrial ATP

Marked blocking of translocator function ( environmental contaminants? Infection/biotoxins?)

Low ATP related Magnesium

Resulting poor ADP to ATP conversion

( all this despite " normal Citric acid cycle on his ONE test…)



**ATP (adenosine triphosphate), studies on Leukocytes**

ATP is hydrolysed to ADP and phosphate as the major energy source in muscle and other tissues. It is regenerated by oxidative phosphorylation of ADP in the mitochondria. When aerobic metabolism provides insufficient energy, extra ATP is generated during the anaerobic breakdown of glucose to lactic acid. ATP reactions require magnesium. ADP to ATP conversion can be blocked by environmental contaminants as can the translocator [TL] in the mitochondrial membrane. [TL] efficiency is also sensitive to pH and other metabolic-factor changes. [TL] defects may demand excessive ADP to AMP conversion (not re-converted to ADP or through to ATP). Defects in Mg-ATP, ADP – ATP conversion and enzyme or [TL] blocking can all result in **chronic fatigue – a factor in any disease where biochemical energy availability is reduced.**

**ATP** whole cell: **mixed leukocytes – ref ranges in bold differ from neutrophils alone**

| | | | |
|---|---|---|---|
| With excess Mg added (Standard method of measuring ATP) | **1.78** | nmol/$10^6$ cells | **2.1 – 3.4** |
| Endogenous Mg only (Measured ATP result is lowered during intracellular magnesium deficiency) | **1.05** | nmol/$10^6$ cells | **1.2 – 3.1** |
| Ratio $ATP/ATP^{Mg}$ | **0.59** | ..... | > 0.65 |

**ADP to ATP conversion efficiency** (whole cells):

| | | | | |
|---|---|---|---|---|
| $ATP^{Mg}$ (from above) | **1.78** | nmol/$10^6$ cells | (1*) | **2.1 – 3.4** |
| $ATP^{Mg}$ (inhibitor present) | **0.57** | nmol/$10^6$ cells | (2*) | < 0.3 |
| $ATP^{Mg}$ (inhibitor removed) | **1.23** | nmol/$10^6$ cells | (3*) | >1.6 |
| **ADP to ATP efficiency** [(3*- 2*)/(1*- 2*)] x 100 = **54.5** | | % | | > 60 |
| **Blocking of active sites** (2*/1*) x 100 | **32** | % | | up to - 14 |

**ADP-ATP TRANSLOCATOR [TL]** (mitochondria, not whole cells):

| | ATP (pmol/$10^6$ cells) | Ref. range | change % | ref. range |
|---|---|---|---|---|
| **Baseline** | **248** | 290 – 700 | | |
| **excess ADP** | **312** | 410 – 950 | **25.8** (in-vitro test) reflects ATP supply for cytoplasm | over 35% (*Increase*) |
| **ADP blocked** | **202** | 140 – 330 | **18.5** (in-vitro test) reflects normal use of ATP on energy demand | 55 to 75% (*Decrease*) |

nments

Low whole-cell ATP

32% blocking of active sites leading to:

Low mt-ATP and poor provision of 'new' mt-ATP

Quite-marked (32%) blocking of translocator function.

Low ATP-related magnesium

Poor ADP to ATP re-conversion

Dr John McLaren-Howard DSc FACN - Directors - Mrs Mirhane McLaren-Howard
Acumen Medical Ltd trading as *Acumen* Registered in England and Wales, no: 7082142
Registered Office: Key House, Woodward Road, Howden Industrial Estate, Tiverton, Devon, EX16 5HW (UK)

**TEST RESULTS December 2017- January 2018** Positive CHRONIC INFECTION tests; IMMUNE SUPPRESSION CD 57; auto-antibodies; blocked methylation, gut permeability, mitochondrial damage.

1.BORRELIA SERASPOTS POSITIVE

2.VIRUSES positive: Toxoplasma; Epstein-Barr; CMV; Herpes zoster; Coxsackie ; HHV6.

*3.ANA positive (FH positive with Mums Lupus)*

4.Methylation functional enzyme tests, showed increased requirement for various types of folate; & glutathione.

5.*Advanced intestinal gut barrier test : high zonulin level ( ? Caused by gluten / grains & metabolites; or infection); high LPS IgM indicating significant inflammation, probable infection; and gut wall permeability.*

*6. Mitochondrial & membrane damage*

*7. IMMUNE SUPPRESSION CD57 LOW*

# Treatments 1

1.”Lyme” treatment ( Chronic infection pathologies) Abs verses Herbs ( Cowdens) He chose Abs despite my recommendations for herbal treatment. Doxycyclin; amoxicillin & tinidazole or a minimum of 3 months. +ACICLOVIR. Followed by minimum 3 month course of the COWDEN protocol. (Antibiotics suited him but he could not tolerate the herbs)

2. Viruses: Lysine 500mg bd while he remains on antibiotics

3.Mitochondrial function; Magnesium Maleate; CoQ10 300mg a day & ATP Fuel ( researched nutritional treatment that includes phospholipids) Manganese ( SOD SNP +/-)

4. Methylated folate & B12 for Methylation Block

5. NAC & lipoic acid for glutathione support GLUTATHIONE MASTER ANTI-INFLAMMATORY

5. IV glutathione recommended but he was unable to access this locally.

6. Another option for Treating the inflammation: Trifortify Liposomal Glutathione. Master antioxidant; anti- inflammatory; helpful to calm LPS. ( Potentially all the above impact to lower inflammation)

# Treatments 2

7. Strict gut protocol for autoimmunity & gut permeability.

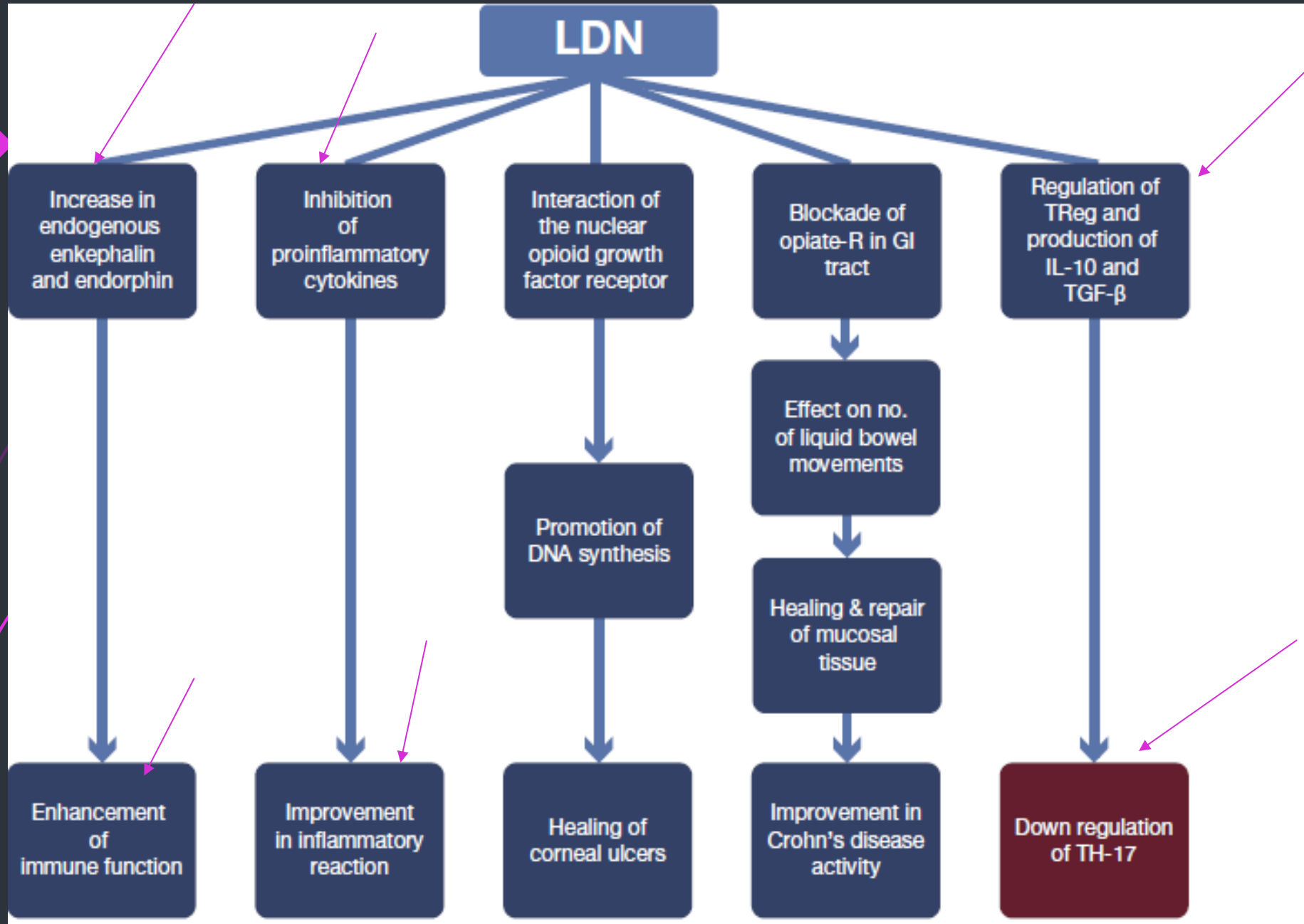
(LANSOPRAZOLE STOPPED) Included all previous gut recommendations to improve digestion; Spore probiotics; Curcumin; Perm plus ie slow release glutamine & Carnosine, ATP Fuel ,which includes phospholipids.

8.Low testosterone previously noted, treated with BIO-IDENTICAL testosterone lozenges 50MG A DAY

9. Optimisation of immune modulation: low dose naltrexone on a gradually increasing dose (see next slide), and Transfer factors from researched Nutritionals. I considered progesterone for its neuroimmune properties

# Further information on Treatments: LDN

- Low dose naltrexone 1 mg half an hour before bed month one
- Low dose naltrexone 2 mg half an hour before bed month 2
- Low dose naltrexone 3 mg half an hour before bed month three
- Thereafter 4.5 mg per day
- Side effects nightmares initially but these should gradually settle.




Dig Dis Sci. 2011 Jul; 56(7):2088-97

**Transfer Factors** Short strands of amino acids, messenger-peptides, manufactured in T-helper Cells

Transfer Factors modulate our immune system.

Transfer factors are non-allergic

# Types of Transfer Factors

- Transfer Factors targeting Lyme Co-infections
- Transfer Factors targeting mold & biotoxins
- Transfer Factors targeting Reactivating infections:
  HSV-1, HSV-2, VZV, CMV, EBV, HHV-6 A&B

| Special Dietary Usefulness for | Multimessenger | Transfer Factor L+ | Messenger N° 1 | Transfer Factor Enviro |
|---|---|---|---|---|
| Natural killer Cell - General immune support | × | | | |
| Bartonella | | x | | |
| Borrelia burgdorferi | | × | × | |
| Babesia | | × | | |
| Ehrlichia | | × | | |
| EBV | | × | × | |
| HHV6 B | | × | | |
| HHV6 A&B | | | × | |
| CMV | × | × | × | |
| Chlamydia pneumoniae | | | × | |
| Pneumocystic carinii | | | × | |
| Human TB | | | × | |
| Bovine TB | | | × | |
| Herpes 1 | | | × | |
| Herpes 2 | | | × | |
| Cryptosporosis | | | × | |
| Mycobacterium avian | | | × | |
| Hepatitis A,B,C | | | × | |
| Staphylcocci | | | × | |

| Special Dietary Usefulness for | Multimessenger | Transfer Factor L+ | Messenger N° 1 | Transfer Factor Enviro |
|---|---|---|---|---|
| Streptococci | | | × | |
| E. Coli | | | × | |
| Parvo virus B19 | | | × | |
| Varicella Zoster | | | × | x |
| Candida (multiple strains) | | | × | |
| MMR | | | × | |
| Mycoplasma - 14 strains | | | × | |
| Ureaplasma urealyticum | | | × | |
| Nanobacterium | | | × | |
| Human Papillomaviruses | | | × | |
| Penicillium | | | | x |
| Epicoccum | | | | x |
| Aspergillus fumigatus | | | | x |
| Aspergillus niger | | | | x |
| Aspergillus versicolor | | | | x |
| Cladosporium | | | | x |
| Fusarium | | | | x |
| Geotrichum | | | | x |
| Pithomyces | | | | x |
| Ustilago | | | | x |

Slide from Armin Schwarzbach Antwerp 2018. Effective antibiotics for Lyme Borreliosis

**Table 5: Effective antibiotics in Lyme borreliosis**

| Antibiotic | Effective intra-cellularly | Enters the CSF | Effective against encysted forms | Plasma half-life |
|---|---|---|---|---|
| **Betalactams** | | | | |
| Ceftriaxone | — | (+)* | — | 8 hrs |
| Cefotaxime | — | (+)* | — | 1 hr |
| Cefuroxime axetil | — | — | — | 1 hr |
| Benzathine benzylpenicillin | — | + | — | 3 days |
| Phenoxymethyl penicillin | — | — | — | 30 min |
| Amoxicillin | — | — | — | 1 hr |
| **Tetracyclines and glycylcyclines** | | | | |
| Doxycycline | + | 14% | — | 15 hrs |
| Minocycline | + | 40% | — | 15 hrs |
| **Macrolides**** | | | | |
| Clarithromycin | + | 5% | — | 4 hrs |
| Azithromycin | + | — | — | 68 hrs tissue half-life |
| **Nitroimidazoles** | | | | |
| Metronidazole | + | + | + | 7 hrs |
| **Co-drugs** | | | | |
| Hydroxychloroquine | + | + | + | 30-60 days tissue half-life |

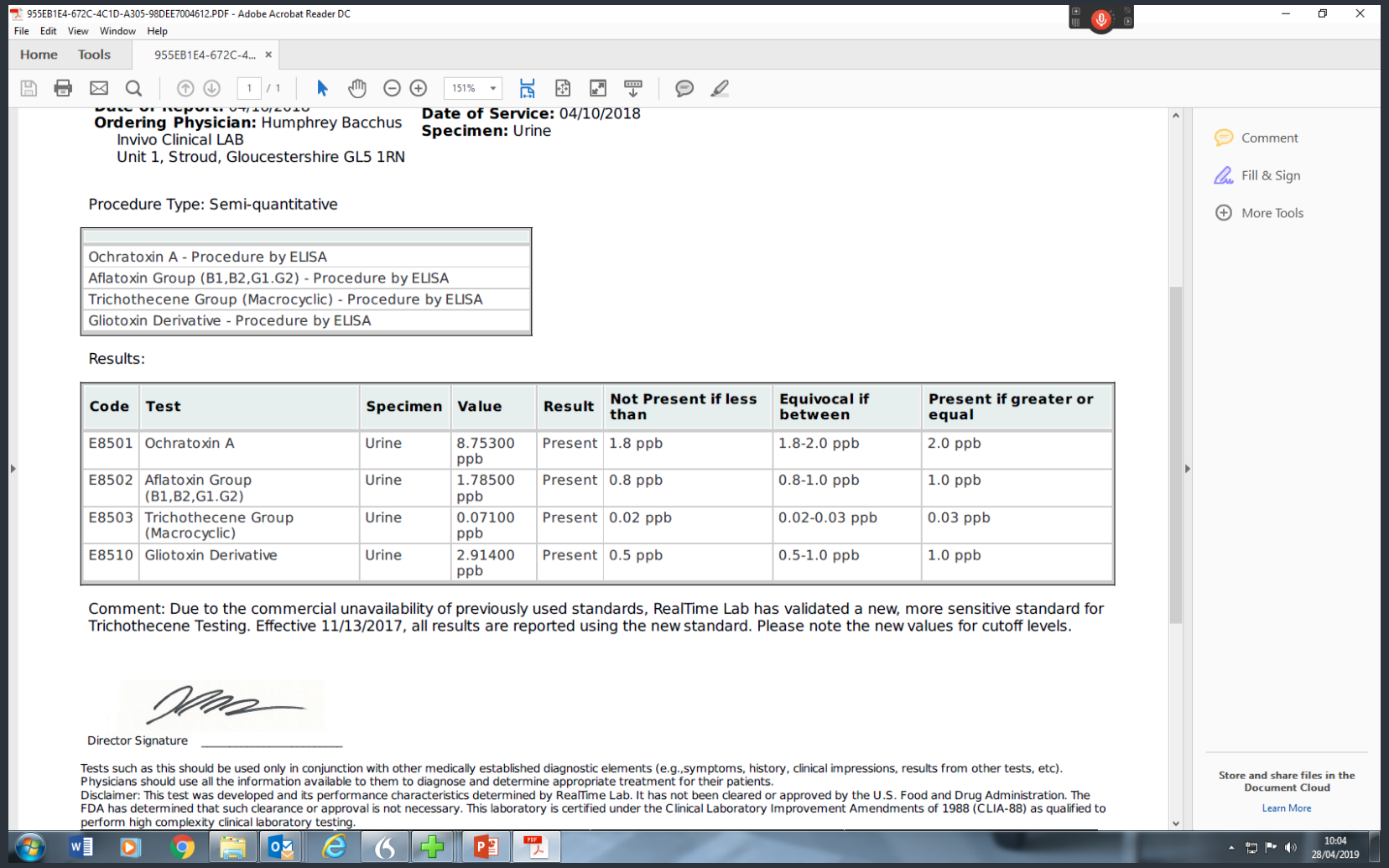
* The betalactams have a poor ability to enter the CSF but, on account of their wide therapeutic spectrum, attain concentrations in the CSF which are clearly above the minimum inhibitory concentration (MIC).[74]

** Macrolides are not used in cases of QTc intervals (frequency-corrected QT intervals) of more than 440 milliseconds with heart rates between 60 and 100 bpm.[67,68]

# Patient progress April 2018

- Coped well with antibiotics, though tinidazole brought SIBO symptoms back so he stopped this.
- Decided to have further tests under the Charity cover before stopping work. THESE WERE MOULD, MARCONS AND (chronic inflammatory response syndrome) CIRS.
- THESE WERE ALL POSITIVE; & HE AGREED TO HAVE TREATMENT FOR MARCONS, ( Nasal sprays, inc Ag EDTA & N acetyl cysteine)
- Treated with chlorella vulgaris as an initial binder for mould; with further plans for organic cholestyramine

# Further tests  April 2018 Urinary mould metabolites; all STRONGLY POSITIVE



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**Ordering Physician:** Humphrey Bacchus
Invivo Clinical LAB
Unit 1, Stroud, Gloucestershire GL5 1RN

**Date of Service:** 04/10/2018
**Specimen:** Urine

Procedure Type: Semi-quantitative

| |
|---|
| Ochratoxin A - Procedure by ELISA |
| Aflatoxin Group (B1,B2,G1.G2) - Procedure by ELISA |
| Trichothecene Group (Macrocyclic) - Procedure by ELISA |
| Gliotoxin Derivative - Procedure by ELISA |

Results:

| Code | Test | Specimen | Value | Result | Not Present if less than | Equivocal if between | Present if greater or equal |
|---|---|---|---|---|---|---|---|
| E8501 | Ochratoxin A | Urine | 8.75300 ppb | Present | 1.8 ppb | 1.8-2.0 ppb | 2.0 ppb |
| E8502 | Aflatoxin Group (B1,B2,G1.G2) | Urine | 1.78500 ppb | Present | 0.8 ppb | 0.8-1.0 ppb | 1.0 ppb |
| E8503 | Trichothecene Group (Macrocyclic) | Urine | 0.07100 ppb | Present | 0.02 ppb | 0.02-0.03 ppb | 0.03 ppb |
| E8510 | Gliotoxin Derivative | Urine | 2.91400 ppb | Present | 0.5 ppb | 0.5-1.0 ppb | 1.0 ppb |

Comment: Due to the commercial unavailability of previously used standards, RealTime Lab has validated a new, more sensitive standard for Trichothecene Testing. Effective 11/13/2017, all results are reported using the new standard. Please note the new values for cutoff levels.

Director Signature _____

Tests such as this should be used only in conjunction with other medically established diagnostic elements (e.g.,symptoms, history, clinical impressions, results from other tests, etc). Physicians should use all the information available to them to diagnose and determine appropriate treatment for their patients.
Disclaimer: This test was developed and its performance characteristics determined by RealTime Lab. It has not been cleared or approved by the U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary. This laboratory is certified under the Clinical Laboratory Improvement Amendments of 1988 (CLIA-88) as qualified to perform high complexity clinical laboratory testing.

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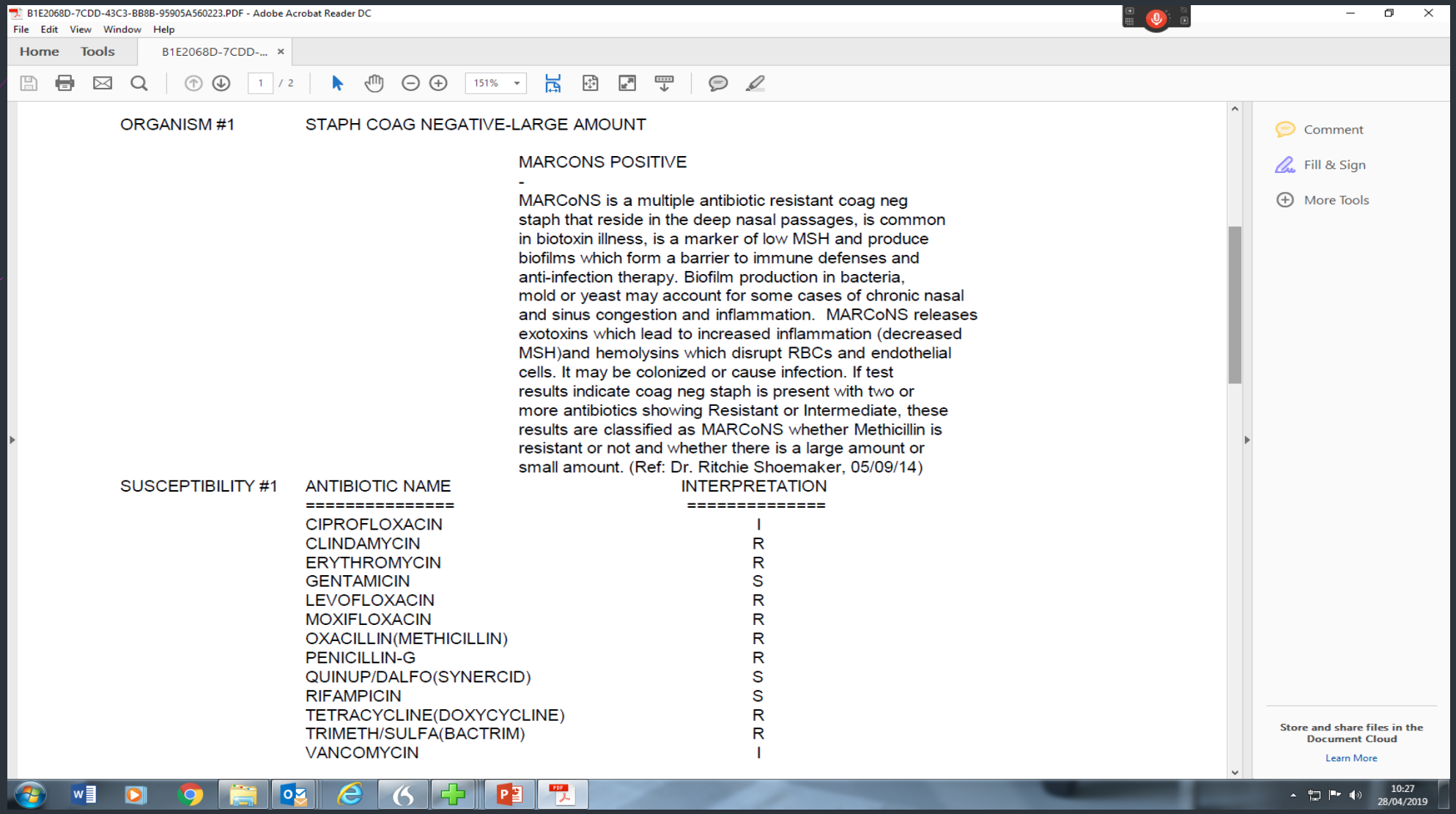
10:04
28/04/2019

# MARCONS POSITIVE




ORGANISM #1 STAPH COAG NEGATIVE-LARGE AMOUNT

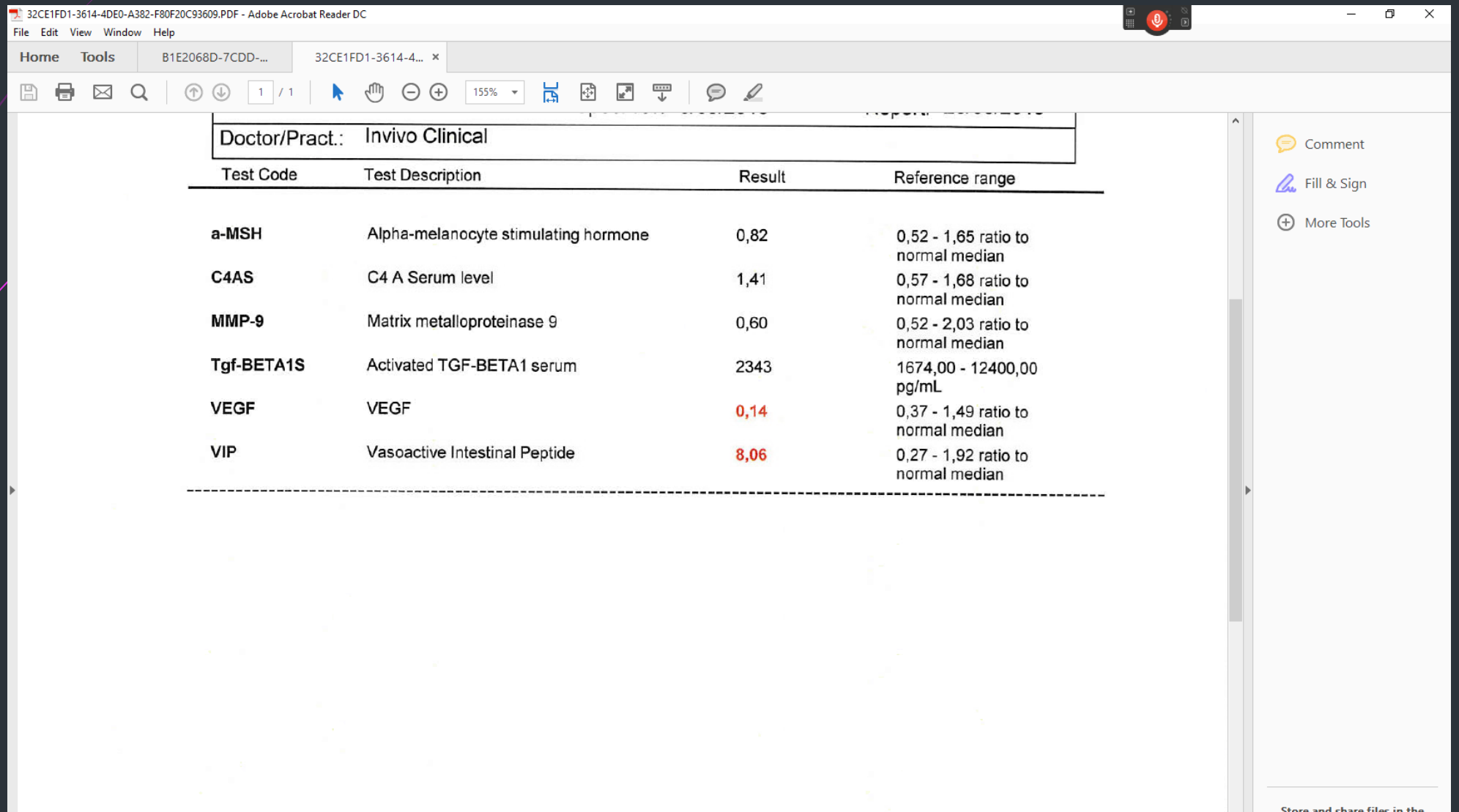
MARCONS POSITIVE

-

MARCoNS is a multiple antibiotic resistant coag neg staph that reside in the deep nasal passages, is common in biotoxin illness, is a marker of low MSH and produce biofilms which form a barrier to immune defenses and anti-infection therapy. Biofilm production in bacteria, mold or yeast may account for some cases of chronic nasal and sinus congestion and inflammation. MARCoNS releases exotoxins which lead to increased inflammation (decreased MSH)and hemolysins which disrupt RBCs and endothelial cells. It may be colonized or cause infection. If test results indicate coag neg staph is present with two or more antibiotics showing Resistant or Intermediate, these results are classified as MARCoNS whether Methicillin is resistant or not and whether there is a large amount or small amount. (Ref: Dr. Ritchie Shoemaker, 05/09/14)

| SUSCEPTIBILITY #1 | ANTIBIOTIC NAME | INTERPRETATION |
|---|---|---|
| | ========== | ========== |
| | CIPROFLOXACIN | I |
| | CLINDAMYCIN | R |
| | ERYTHROMYCIN | R |
| | GENTAMICIN | S |
| | LEVOFLOXACIN | R |
| | MOXIFLOXACIN | R |
| | OXACILLIN(METHICILLIN) | R |
| | PENICILLIN-G | R |
| | QUINUP/DALFO(SYNERCID) | S |
| | RIFAMPICIN | S |
| | TETRACYCLINE(DOXYCYCLINE) | R |
| | TRIMETH/SULFA(BACTRIM) | R |
| | VANCOMYCIN | I |

# CIRS: 2 TESTS POSITIVE




Doctor/Pract.: Invivo Clinical

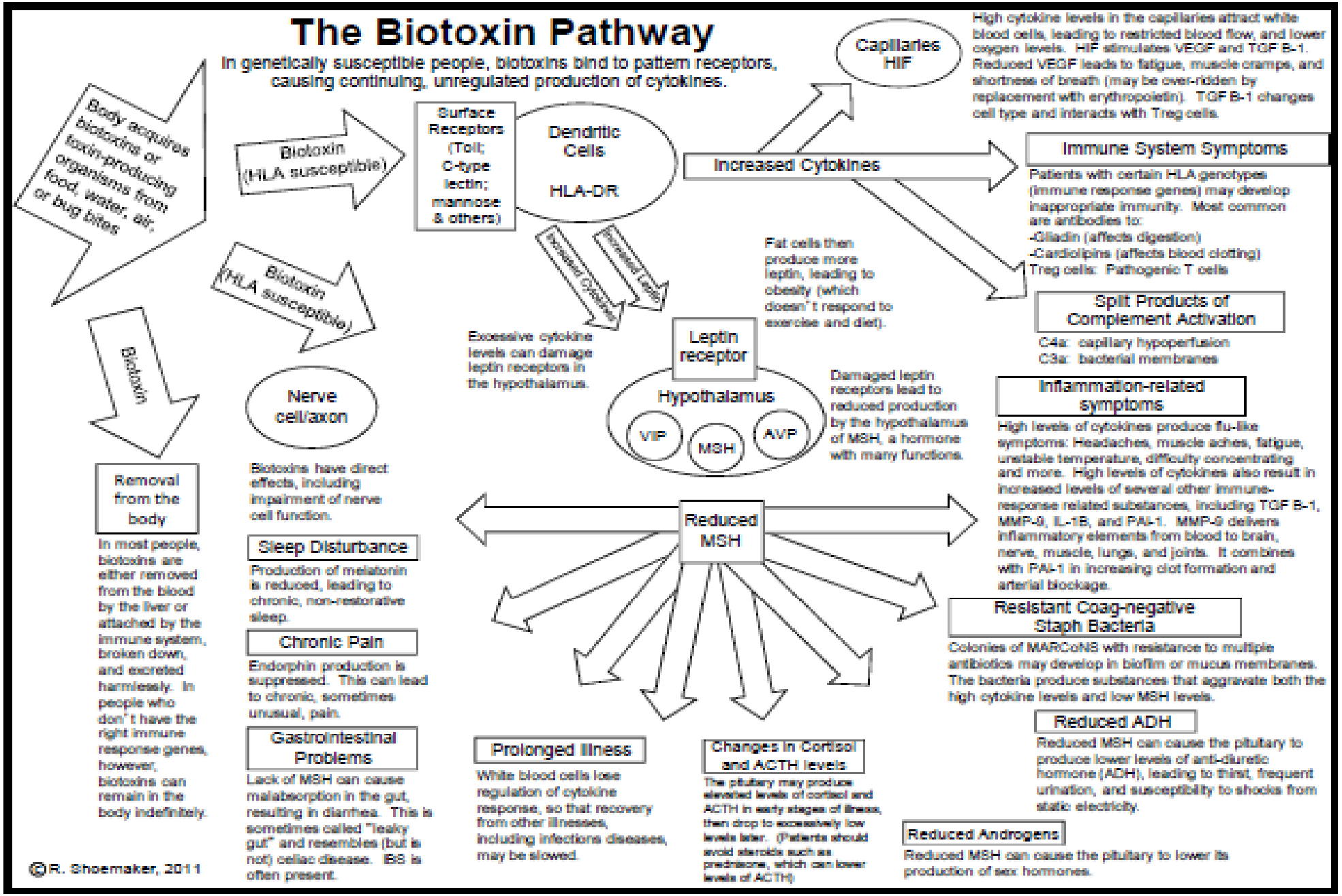
| Test Code | Test Description | Result | Reference range |
|---|---|---|---|
| **a-MSH** | Alpha-melanocyte stimulating hormone | 0,82 | 0,52 - 1,65 ratio to normal median |
| **C4AS** | C4 A Serum level | 1,41 | 0,57 - 1,68 ratio to normal median |
| **MMP-9** | Matrix metalloproteinase 9 | 0,60 | 0,52 - 2,03 ratio to normal median |
| **Tgf-BETA1S** | Activated TGF-BETA1 serum | 2343 | 1674,00 - 12400,00 pg/mL |
| **VEGF** | VEGF | 0,14 | 0,37 - 1,49 ratio to normal median |
| **VIP** | Vasoactive Intestinal Peptide | 8,06 | 0,27 - 1,92 ratio to normal median |

# VEGF vascular endothelial growth factor (& VIP relevance)

- Measurement of host response to infection
- VEGF is known for vasodilation, angiogenesis and neuroprotection
- It can be high or low in patients with CIRS
- With VEGF deficiency, there is loss of neuroprotection ; increased permeability of the blood brain barrier as well as capillary hypoperfusion. Symptoms associated include shortness of breath, cognitive different dysfunction, fatigue and muscle cramps.
- VIP (vasoactive intestinal peptide): low levels more significant in CIRS;  high levels may be compensatory. Immune regulation function, high levels may be associated with gut permeability.

# Chronic Inflammatory Response Syndrome

- Growing awareness that microbes play a role in chronic illness
- Terrain matters – impaired immunity predisposes patient to chronic infection
- Chronic infections suppress immune activity
- Other factors that alter immunity – biotoxins (metals, chemicals), inherited or acquired defects


The Biotoxin Pathway
In genetically susceptible people, biotoxins bind to pattern receptors, causing continuing, unregulated production of cytokines.
Body acquires biotoxins or toxin-producing organisms from food, water, air, or bug bites
Biotoxin (HLA susceptible)
Surface Receptors (Toll; C-type lectin; mannose & others)
Dendritic Cells
HLA-DR
Capillaries HIF
High cytokine levels in the capillaries attract white blood cells, leading to restricted blood flow, and lower oxygen levels. HIF stimulates VEGF and TGF B-1. Reduced VEGF leads to fatigue, muscle cramps, and shortness of breath (may be over-ridden by replacement with erythropoietin). TGF B-1 changes cell type and interacts with Treg cells.
Increased Cytokines
Immune System Symptoms
Patients with certain HLA genotypes (immune response genes) may develop inappropriate immunity. Most common are antibodies to:
-Gliadin (affects digestion)
-Cardiolipins (affects blood clotting)
Treg cells: Pathogenic T cells
Increased Cytokines
Increased Leptin
Fat cells then produce more leptin, leading to obesity (which doesn't respond to exercise and diet).
Split Products of Complement Activation
C4a: capillary hypoperfusion
C3a: bacterial membranes
Excessive cytokine levels can damage leptin receptors in the hypothalamus.
Leptin receptor
Hypothalamus
VIP
MSH
AVP
Damaged leptin receptors lead to reduced production by the hypothalamus of MSH, a hormone with many functions.
Inflammation-related symptoms
High levels of cytokines produce flu-like symptoms: Headaches, muscle aches, fatigue, unstable temperature, difficulty concentrating and more. High levels of cytokines also result in increased levels of several other immune-response related substances, including TGF B-1, MMP-9, IL-1B, and PAI-1. MMP-9 delivers inflammatory elements from blood to brain, nerve, muscle, lungs, and joints. It combines with PAI-1 in increasing clot formation and arterial blockage.
Biotoxin (HLA susceptible)
Nerve cell/axon
Biotoxins have direct effects, including impairment of nerve cell function.
Biotoxin
Removal from the body
In most people, biotoxins are either removed from the blood by the liver or attached by the immune system, broken down, and excreted harmlessly. In people who don't have the right immune response genes, however, biotoxins can remain in the body indefinitely.
Reduced MSH
Sleep Disturbance
Production of melatonin is reduced, leading to chronic, non-restorative sleep.
Chronic Pain
Endorphin production is suppressed. This can lead to chronic, sometimes unusual, pain.
Gastrointestinal Problems
Lack of MSH can cause malabsorption in the gut, resulting in diarrhea. This is sometimes called "leaky gut" and resembles (but is not) celiac disease. IBS is often present.
Prolonged Illness
White blood cells lose regulation of cytokine response, so that recovery from other illnesses, including infections diseases, may be slowed.
Changes in Cortisol and ACTH levels
The pituitary may produce elevated levels of cortisol and ACTH in early stages of illness, then drop to excessively low levels later. (Patients should avoid steroids such as prednisone, which can lower levels of ACTH)
Resistant Coag-negative Staph Bacteria
Colonies of MARCoNS with resistance to multiple antibiotics may develop in biofilm or mucus membranes. The bacteria produce substances that aggravate both the high cytokine levels and low MSH levels.
Reduced ADH
Reduced MSH can cause the pituitary to produce lower levels of anti-diuretic hormone (ADH), leading to thirst, frequent urination, and susceptibility to shocks from static electricity.
Reduced Androgens
Reduced MSH can cause the pituitary to lower its production of sex hormones.
©R. Shoemaker, 2011

# CIRS Shoemaker protocol

Diagnostic Process for Chronic Inflammatory Response syndrome

37 symptoms, 13 clusters of symptoms, positivity in 8 virtually diagnostic

Specific tests are involved

11 steps for treatment

# Patient email review April 2019

- Patient has been managing well.
  - "I'm doing ok thanks, my health is similar to how it was and I am still seeing the benefits from your consultations. My Parkinsons has progressed slightly in a couple of areas but not that has really impacted my life. I am working again part time 3 days a week and 6 hours each day I feel this is helping with the cognitive aspects of Parkinson's.
  - Comparing myself with others I am doing very well with my Parkinson's"

# 1. Patient telephone call & progress 28.4.19

- April 2018, took four months off of work, time to himself, went to the gym, walked a lot and recognised that the stress of a top job was too difficult for him.
- August 2018, returned to work for a different company using similar skills, pricing as an electrical contractor.
- Medication 125 mg of Levodopa 4 times a day, extra modified release dose at night. The medication was lasting 3 ½ hours but is now only lasting three hours.
- NHS Parkinson's nurses are very helpful and have advised him concerning timing of protein and food intake in order to minimise competition of receptor sites for dopamine.

# 2. Patient telephone call & progress 28.4.19

- His gut is still stable with occasional bouts of diarrhoea. His tremor is slightly worse but not a major issue for him. He is less tired. When his medications wear off he does struggle with the rigidity and shuffling gait. We discussed the possibility of cannabis helping this; as there is strong evidence for cannabis helping spasms in all kinds of neurological disorders.

- He is involved with research due to frequency of urination, also with PD stat, a research programme investigating  the benefit of statins in Parkinson's disease. This is a double blind controlled trial. He is unaware of whether he is the control or not. ( STATINS treat the raised C3a in active LYME disease) SHOULD ONLY BE USED WITH COq10 AS STATINS BLOCK ITS PRODUCTION

- I updated him on the possibility of a more formal Shoemaker approach to CIRS , also the possibilities of looking into membrane medicine and epigenetic influences of adducts which may be helped with more formalised fatty acid or phospholipid treatment; rather than the ATP Fuel regime that he had before.

Patient due to return for further discussions soon

# 1. Discussion/thoughts

- Does “essential tremor” exist? ( He had a family history of this) TREAT SYMPTOMS EARLY. CONSIDER DAT SCAN
- Which organisms may underlie Parkinson’s disease? CHECK FOR THESE
- Consider MOULD / CIRS (Chronic Inflammatory response Syndrome) formal testing and treatment via Shoemaker protocol?
- DNA adducts evaluation ( biotoxins, chemicals, metals) ? Consider IV phospholipid treatment if appropriate
- Autoimmune disease and chronic infection e.g. lyme, often run alongside one another, maternal family history of his Mother’s LUPUS very relevant.
- Neurological conditions eg ALS, MS, Schitzophrenia, Parkinsons; have differing end pathology, signs & symptoms and are given names by the medical profession but the underlying Biological systems pathology MAY be similar, with the resulting fire of neuro inflammation, affecting and damaging our brains and nervous systems. SO THINK FUNCTIONALLY

# 2. Discussion / thoughts

- Consider other types of support for immune modulation eg Low Dose Naltrexone &/or Transfer Factors
- Consider a trial of cannabis for its multiple properties (anti spasticity & tremor; relaxation properties) which may also include immuno-modulation.
- Other options to consider, hyperbaric oxygen, craniosacral therapy, liposomal melatonin.
- Consider progesterone 50 mg at night as there is evidence this can unblock the HLA chromosome 6 which is often responsible for damage to the innate immune system.

# Numerous studies have found connections of Infectious burden with Parkinson's/Parkinsonism

"Slide from Armin Schwarzbach Antwerp 2018"

Parkinsonism Relat Disord. 2015 Aug;21(8):877-81. doi: 10.1016/j.parkreldis.2015.05.015. Epub 2015 May 30.

**The association between infectious burden and Parkinson's disease: A case-control study.**

Bu XL[1], Wang X[1], Xiang Y[1], Shen LL[1], Wang QH[1], Liu YH[1], Jiao SS[1], Wang YR[1], Cao HY[1], Yi X[1], Liu CH[1], Deng B[1], Yao XQ[1], Xu ZQ[1], Zhou HD[1], Wang YJ[2].

Author information

**Abstract**

**INTRODUCTION:** The etiology of Parkinson's disea
common pathogenic infections and PD.

**METHODS:** Antibody titers to common infectious pa
type-1 (HSV-1), Borrelia burgdorferi (B. burgdorferi),
measured by ELISA in serum of 131 PD patients an
exposure to these common pathogens.

**RESULTS:** Seropositivities toward zero-two, three-four and five-six of these pathogens were found in 11%, 74% and 15% of normal controls while in 4%, 61% and 35% of PD patients, respectively. IB, bacterial burden and viral burden were independently associated with PD. Schwab and England (S&E) scores were negatively correlated with IB in patients with PD. Serum α-synuclein protein levels and inflammatory cytokines (interleukin-1β and interleukin-6) in individuals with higher IB were also significantly higher.

**CONCLUSIONS:** IB consisting of CMV, EBV,
role of infection in the etiology of PD.

**"Infectious burden consisting of CMV, EBV, HSV-1, B. burgdorferi, C. pneumoniae and H. pylori is associated with PD. This study supports the role of infection in the etiology of PD."**

of

**Drosophila-like 4 gene, which is associated with inflammation and neuronal death and is up-regulated in Parkinson's disease, was up-regulated in spirochete-stimulated tissues by 9.98-fold***

*Source: * Ramesh G et al. Interaction of the Lyme Disease Spirochete Borrelia burgdorferi with Brain Parenchyma Elicits Inflammatory Mediators from Glial Cells as Well as Glial and Neuronal Apoptosis. Am J Pathol. 2008 Nov; 173(5): 1415–1427*

# "Slide from Armin Schwarzbach Antwerp 2018

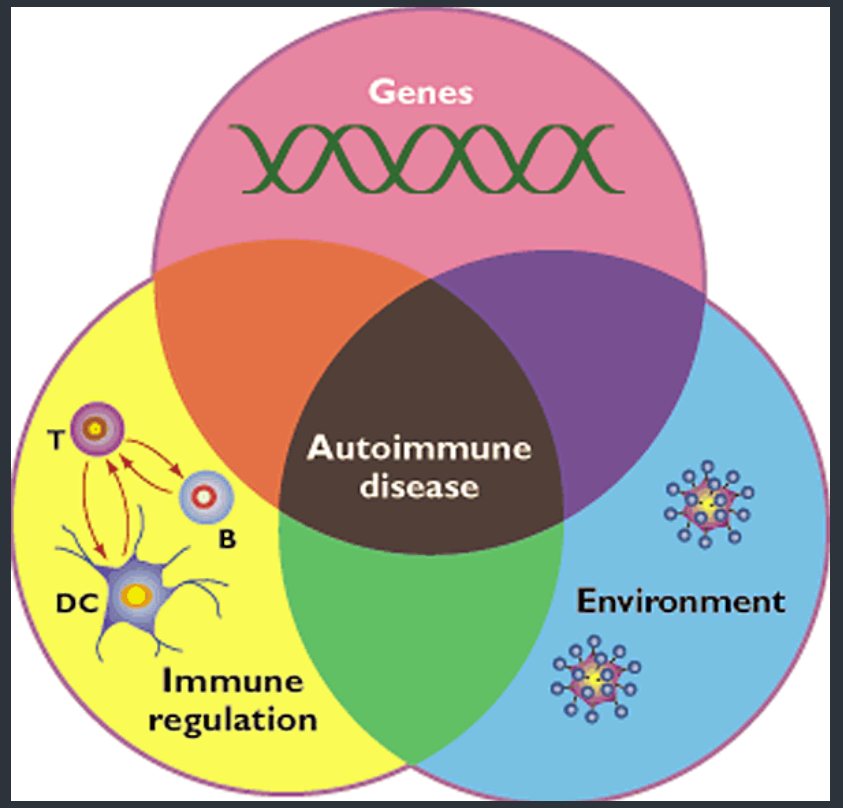
**"Borrelia can cause Parkinsonism"**
(Arch.of Path.& Lab.Med.127(9):1204-6)

# Parkinsonism possible underlying infections (this patient was positive for those in red)

1. Borrelia SeraSpot + Borrelia EliSpot + CD57 cells
2. Chlamydia pneumoniae IgG/IgA antibodies + Chlamydia pneumoniae EliSpot
3. Mycoplasma pneumoniae IgG/IgA antibodies
4. Bartonella IgG/IgM antibodies
5. Coxsackie Virus IgG/IgA antibodies
6. EBV EliSpot
7. CMV EliSpot

Slide from Armin Schwarzbach Antwerp 2018

# Multifactorial aetiology of autoimmune disease.



# Multisystems Biological Approach

Gut, microbes, neuro & general inflammation, hormones, autoimmunity & immune modulation, lifestyle to include diet, & supplements, detoxification, energy production optimisation, and where appropriate, management of relaxation, stress, exercise and sleep.

# Thank you for listening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