

Rashes are a Sign of Inflammation

- Rashes caused by Viruses, Bacteria, and CWD Forms
 - Enzymes of pathogens cause lesions (sores)
 - Enzymes are bacterial spreading factor
 - Enzymes are viral penetrating factor
- Multiple infections' enzyme action is multiplied
 - Viruses boost bacteria (sore throat & Arthritis flare)
 - Proteinase, Hyaluronidase, Colligenase lyse 3-D tissues
 - HHV6 & Group A Streptococcus, => Flesh Eating Bacteria
- Dietary enzymes (papain, bromilain) disable foreign proteins
 - Kill gut worms
 - Promote sports injury healing
 - Topically disable bee/spider venom
- Vitamin C disables Hyaluronidase

Mycoplasma HyperAllergic State

Noted in Mycoplasma pneumonia vaccine trials that failed.

JAMA Vol 199, 1967 Feb 6 353-358 *Inactivated M. pneumonia Vaccine*,
C.B. Smith, et al.

- 21 men received vaccine and a booster injection,
- 24 unvaccinated controls, some with prior antibodies
- 19/21 had no M. pneumonia antibodies to start
- Treatment of infection: Tetracycline or Erythromycin
- 8 of 19 vaccinated failed to develop antibodies and were infected
- 10 of 19 vaccinated developed antibodies.
- Where vaccine antibodies failed to develop, there was no protection and illness was more severe, 3 developed pneumonia.

One might conclude:

With M. pneumonia (live and vaccine),

- Antibodies often may not normally develop ~50% of cases
- When antibodies fail, testing finds persons with a hyper sensitive (secondary) immunity mode
- These persons may have chronic mycoplasma infection with continued negative antibody test results and hyper allergic reaction to mycoplasma

Crohn's Vs Jonne's Disease in Cattle

- **Mycobacterium paratuberculosis at >90% level**
- Similar to tuberculosis but different
- Respiratory phase followed by Gut inflammation
- Food Borne Infection
 - M. paratuberculosis is heat resistant
 - Pasteurization sometimes fails
- Jonne's is epidemic in dairy herds 10-35%
- Cross Species ~1/100 virulence decrease
- Genetic factors enhance human susceptibility
- Other bacteria, viruses make IBD worse
- Susceptable to multi antibiotic treatment
- No vaccine and you can get it again.

CytoMegalovirus (CMV)

CMV and HHV6 infect Monocytes persistently

They also alter the hematopoietic development process
from stem cells to developed immune system
white blood cells

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=9247037&dopt=Abstract

CMV infects/disrupts mononuclear phagocytes (Monocytes)

Also macrophages, bacteria specific polymorphonucleocytes (PMNs),
endothelial cells and sinusoidal lining cells. Bone marrow may be source of
latency (similar to leukemia).

PMN disruption may promote specific bacterial persistence.

Endothelial infection may lead to sclerosis of tissues.

CMV causes colitis

Anti Viral ganciclovir is used to suppress CMV

CMV HHV5

- Transmitted in utero, during birth, from kissing, breast milk, sex
- ~ 80% test positive for infection
- Makes invaded cells enlarge
- Kidney, salivary gland, retina,
- Lungs deadly pneumonia,
- GI tract perforation

CROHN'S/IBD Complicated by Microbes:

- Candidae, and other yeasts, fungal forms,
- Coxsackie/Norwalk/HHV6/ CMV
- Myobacteria/mycobacteria (para)Tuberculosis**
- Celiac sprue = gluten intolerance,
Tropical sprue = Unknown infection
- Klebsiella Pneumonia,
- E. coli, e.g., hemolytic strain,
- Clostridium difficile
- Yersinia enterocolitica => systemic infiltration**

Diet *may help* control microbes

- Sugar(s) promote bacterial and **yeast** proliferation.
Candida filaments puncture gut;
Unfiltered food enters blood;
Immune system reacts generating food antibodies;
Antibodies amplify inflammation at inflamed sites.
- Probiotics suppress yeast (Candida);
- Cut out sugar (Atkins ketosis) starve your parasites
- Excess estrogen, (soy) promotes Candida.

Ultimately, antibiotics are needed to control bacteria.

But antibiotics are not effective against dormant forms.

Long-term, pulsed, low-level tetracycline antibiotics.

AntiBiotic Protocols

- Medical Establishment is Anti- Antibiotic

 - Adverse to long term AB treatments

 - Adverse to multi antibiotics at same time

 - Likes pills, not shots, patches, or intravenous

 - Gut administration convenience builds AB resistance

 - Intravenous protocols are overpriced

 - Vaccine Costs are mostly liability insurance premiums

- The Math of Multi-AB Protocols (A, B, C)

 - Protocol A = 98%, B is 97%, C is 95%

 - Combined $1/[(.02 \times .03 \times .05) = 1/.00003 \Rightarrow 99.997\%$

 - Probability of drug resistance is much much lower.

- Scientists now endorse multi AB protocols 'cause they work

Human Herpies Virus HHV6

Double Stranded DNA Virus

- HHV6 Accelerates HIV, a retrovirus
- HHV6 Invades B-Lymphocytes
 - Persistent lifelong infection
 - Infected in childhood (roseola)
- **REDD** RNase deficient immune compromise
 - Epstein barr, CMV, HHV6, mycoplasma
 - <http://www.integralworld.net/redd.html>
- Est. >90% HHV6 infection rate in adults
- HHV6 Cofactor in Flesh Eating Strep (Grp A)
- HHV6 Cofactor in Juvenile RA and RA
- HHV6 Produces tissue dissolving enzymes
- HHV6 active in 89% of Multiple Sclerosis biopsies
- Antiviral HHV6 treatment has no positive results
- Avonex, Betaserson & Copaxon interferon drugs for MS
- **Treatment:** Foscamet according to Merck Manual

Epstein Barr Virus (EBV= HHV4)

Infectious mononucleosis = Kissing

Incubation period is 6 weeks

Tropism for B-Cells where it multiplies

It changes B-Cell genes immortalizing them

Acute stage mononucleosis

Fever, malaise, Fatigue (interleukin effect), Sore throat,

Lymphadenopathy esp behind ear adnoides

Lymph node infection signs, biopsy

Spleen packed with activated cells and subject to rupture.

Rash esp. if ampicillan

Recessive Gene makes it fatal (attack on Red Blood Cells)

Burkett's Lymphoma, throat and salivary cancers

Dormant after acute stage; chronic EBV is reported

EBV viral protein mimics myelin basic protein

=> MS myelin autoimmune attacks

Hodgkins Disease traces of EBV are often found, discounted

Treatment: Acyclovir, Famcyclovir, Valacyclovir acc.to Merck Manual

Coxsackie Virus

- RNA: PicoVirus Family: SubFam:
- Enterovirus (affects the gut); Similar to EchoViruses and Poliovirus
- Attacks muscles, esp. the heart www.itmonline.org/arts/coxsackie.htm
- Feeling of severe muscle tiredness, almost pain
- Attacks Brain, CNS
- Hand, foot & mouth disease, 6 mo to 3 yrs, high fever, rash, sore throat.
- Generalized disease of the newborn.
- Cross strain re-infections are possible (~30 strains) => multiple infections
- **CV A & CV B (30 Strains):** Herpangina, sore throat, lymph nodes, conjunctivitis, Diarrhea, Hemolytic-uremic syndrome, Myositis, Guillain-Barre syndrome, Reye syndrome, Mononucleosis-like syndrome, Infectious lymphocytosis, Exanthem=blooming rash, Pleurodynia, Pericarditis, **Myocarditis, Diabetes mellitus.**

Coxsackie Virus Treatment

Treatment: Pleconaril anti viral for PicornaViruses (Germany)

- **Rest** (activity worsens); anti-inflammatories; Quercetin & Vitamin C;
- **Herbals W/ anti viral properties:**
Bitter melon, St. John's Wort, Olive Leaves, Licorice root,
Ginger, Rosemary, Oregano, Garlic, Green Tea extracts
Shiitake & Porcini (boletus) Mushrooms
- **Chinese Medicine:** sophora root (*kushen*), astragalus (*huangqi*),
and ginseng (*renshen*).

Leishmania

- Infects vertebrates
- Tailed football shape with internal structures
- Invades Monocytes replicates
- Promotes prostoglandin E2, TGF beta, defective interferon-gamma, etc
- Disables immune activity
- Vector is blood feeding sandflies
- Possibly bats, rodents, birds are carriers
- <http://www.biosci.ohio-state.edu/~parasite/leishmania.html>

How HMOs Fail to Work

- **Capitation** implies funds retention by insurance companies
 - They negotiate lowest cost to sponsor so provider is squeezed
 - Medical costs have huge **liability insurance component**
 - Doctors need to see too many patients with minimum time
 - Doctors are penalized for “Overtesting” so they undertest
 - Doctors are rewarded for finding nothing, treating symptoms
 - Tests are expensive and have many false negatives
 - Positive chronic microbe test results dismissed as normal
 - Many microbes and pathological variant strains not detected
 - **Resulting in:**
 - Multiple visits until correct diagnosis, delayed root cause treatment
 - Reluctance to prescribe Antibiotics til treatment is diagnosed/codified
 - Multi-infections go untested, untreated, and parasite load increases.
- Health degrades and is not maintained at all.**

Bacterial Forms Span Huge Size Range

Many forms in complete life cycle of multi- host bacteria:

E.G. Malarial Plasmodium

- Vectors/Hosts range from Insects to Animals
- Microbe Relocation (infection) via food, air, water, bites, sex
- Microbe Forms replicate & transmit

Genetic Recipes: DNA/RNA/etc.

Microbe Forms: Colonies, Cell Wall, L-Forms, Cell Wall Deficient (CWD), Intracellular colonies, Plasmids, Macrophages, Intracellular emergent, Dormant (seed, bleb, cyst, spore, etc)

- Smallest bacterial forms near viral sized
- They are often characterised as artifacts in microscope views
- Microscopes need ultraviolet light to “see” smallest forms.
- We cannot see ultraviolet wavelengths

Chemical Recipes are Transmitted by Invader's DNA/RNA

Viruses, Macrophages, Plasmids invade both host cells (Monocytes) and bacteria large-forms, changing DNA/ RNA

This can either be helpful or harmful to host. E;g.,

- Mycoplasma kills HIV invaded immune cells.
- Plasmids convey antibiotic recipes to bacteria.
- Mycoplasma & Parvovirus B19 ~8000 Google hits. (Lupis?)
- Leishmania and leishmaniavirus
- Plasmid Transmission crosses species in shared locations (gut, ear, urinary tract, etc)

Recipe Transmission Effects

DNA/RNA recipes control generation of bioactive host molecules, enzymes, hormones, ligands, Immune triggers, Nox, Hydrogen peroxide, etc; Contributing to lysis and inflammation.

DNA/RNA recipes in bacteria control generation of bioactive molecules, enzymes, hormones, ligands, Immune triggers, Nox, Hydrogen peroxide, etc. Spreading factor and virulence enhancement.

Plasmid recipes may activate dormant bacteria genes making them more or less virulent.

Molecular concentrations are upset affecting chemical/ hormonal/ immunal control systems, leading to disease symptoms

Harmful Synergism

Doctors assume frequently found microbes are harmless and not causal factors

Certain Combinations amplify virulence factors. However, our knowledge of cofactor mechanisms is fragmentary.

- Colds and Strep throat.
- HIV and multiple conditions
- Coxsackie, Yersinia, thyroid suppression, and diabetes, many cofactors
- Mycoplasma and parvovirus B19 linked to Lupus

Why are we getting so fat?

- Ramp up in obesity and Hyperglycemia started in recent past.
- Coincidental changed recipes for processed foods replacing Sucrose with Fructose from corn.
- Fructose is not metabolized fast by our insulin processes in body. It hangs around and is used by our microbial parasites.
- Newly discovered harmful mycoplasma strains use fructose instead of glucose/sucrose. Changed diet promotes these strains.
- Coxsackie B/B4 and other viruses CMV EBV Rubella suppress thyroid, inflame pancreas, leading to insulin production failure diabetes. Hyperglycemia immune deficiency W/out insulin failure.
- Thyroid suppression decreases resistance to mycoplasma and other fatigue symptoms. Coxsackie myocarditis enhances fatigue.
- We feel tired and lazy so we do not exercise

Marshall Antibiotic Protocol

- Similar to Protocol for RA and Fibro in my book Appx 2
- Originally to use Tetracycline against Sarcoidosis Bacteria
Addresses Herxheimer using *Benicar* to block inflammation
- Infection marked by vitamin D unbalanced concentrations
High Vitamin D active form enhances inflammation
Mild inflammation often heals infections
Chronic infections lead to persistent inflammation
- D Ratio = Active / Inactive –D forms' concentration
Highly testable, but few labs get it right
Measures size of colony(ies) and treatment progress
- Macrophage/Monocyte intracellular bacteria makes Active D
Other infections have tipped D ratios: Lyme and Fibrio and ?????
Virus and bacteria infections have different D ratios.
Explains sun sensitivity as excess D stimulating inflammation
- See www.RA-Infection-Connection-Com/macrophages.htm

Yersinia Pseudotuberculosis

- Similar to *Y. enterocolitica*
- Food borne infection
- Usually self-limiting with “recovery”
- Generates toxin that attacks Myelin in nerve sheaths
- Toxin causes myelin destruction
- Loss of neural transmission capability
- Multiple Sclerosis (MS) is affect of similar toxin/enzyme
- Web links MS sclerosis:
Mycoplasma arthrides, EBV, HHV6,
Y. enterocolitica, Y. pseudotuberculosis toxin
- Soviets moved M1 toxin gene to *Y. pestis* (plague)
- *Y. pestis* has 13% of *Y. Pseudotuberculosis* genes inactive

Yersinia/IBD Testing:

- Panel for inflammatory bowel disease reports:

IgM, IgG, IgA antibodies

Endotoxins and exotoxins

Klebsiella pneumonia

Yersinia enterocolitica

Clostridium difficile toxin

IgG for tropomyosin.

- Immunosciences Laboratories

- See:

www.RA-Infection-Connection.com/practitioners.htm

for other test labs websites.

Yersinia Enterocolitica

Colonizes monocytes disabling them

Monocytes are Immune (white) blood cells

Antibodies cause Auto immune allergies

Tropisms:

Heart, pericardial inflammation

Spine, Arthritic inflammation, destruction

Bone marrow macrophages, invasion

=> Cytokine inflammation trigger

Nerves, inflammation, excitation

Brain, Autism, excitation complications

Thyroid, react W/ H. thyrotropin receptor

HLA-B27, interaction with transcripts

Lymph nodes, infection