INTRODUCING

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TRANSFORMATION
THE GENESIS OF GOOD HEALTH®

Enzyme Therapy Seminar

October 11-12 • Houston, TX

Hashimoto's & COVID

Where's the Connection?

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Proper Test for The Thyroid

- Inadequate testing and review is the reason so many thyroid issues go undetected, leaving the patient with a heavy symptom burden
- A blood test needs to have **ALL** of the following **EVERY TIME**:
    - TSH, TT4, FT4, TT3, FT3, T3 Uptake, RT3, **TPO & TBG Antibodies**
    - RT3 can be left off if liver, adrenal, immune, and inflammatory scores are normal
- In my office this test (without TBG antibodies) costs $250

Hashimoto's: The Enigma

- The most common autoimmune disease
- One of the most benign autoimmune disorders
- Antibodies to the TPO or TBG qualify for diagnosis (MAYO story)
- The pattern in TPO antibody Hashimoto's:
  - TSH: anywhere & variable, immune status dependent
  - T4: low to low normal
  - T3: low to low normal
  - TPO Antibodies: <30 iu/ml
- The pattern with TBG antibody Hashimoto's:
  - TSH: Normal Range 1.85-3.0
  - T4: Normal range 6-12 mlu/l (note negative feedback loop)
  - T3: Below normal 100-180 ng/dl
  - TBG Antibodies <116 IU/mL

Don't Drink The Water

- Halides accumulated over long periods may occupy iodine receptors and or restrict iodine absorption into thyroid follicular cells.
- Although molecularly similar, the halides are not capable of being used to make T1 or T2 molecules.
- Often, this accumulation may lead to free radical toxicity and then to TPO antibodies.

Chlorine, Fluoride, Bromine:

- In areas where fluoride levels in the water registered above 0.3 mg/l, the risk of having a high rate of hypothyroidism was 37% greater compared to areas that do not fluoridate.

The Gut Connection

SCFs: acetates, butyrates, propionates

- Generation of T-regulatory cells
- Downregulated by all leaky gut contributors and low microbiome
- Downregulated by spike protein inflammation

ASCA-ANCA

- anti- Saccharomyces cerevisiae antibodies-Ulcerative colitis
- antineutrophil cytoplasmic antibodies-Crohn's
- Increased in molecular mimicry with spike protein
- Antibiotic use common in hospitalization treatments

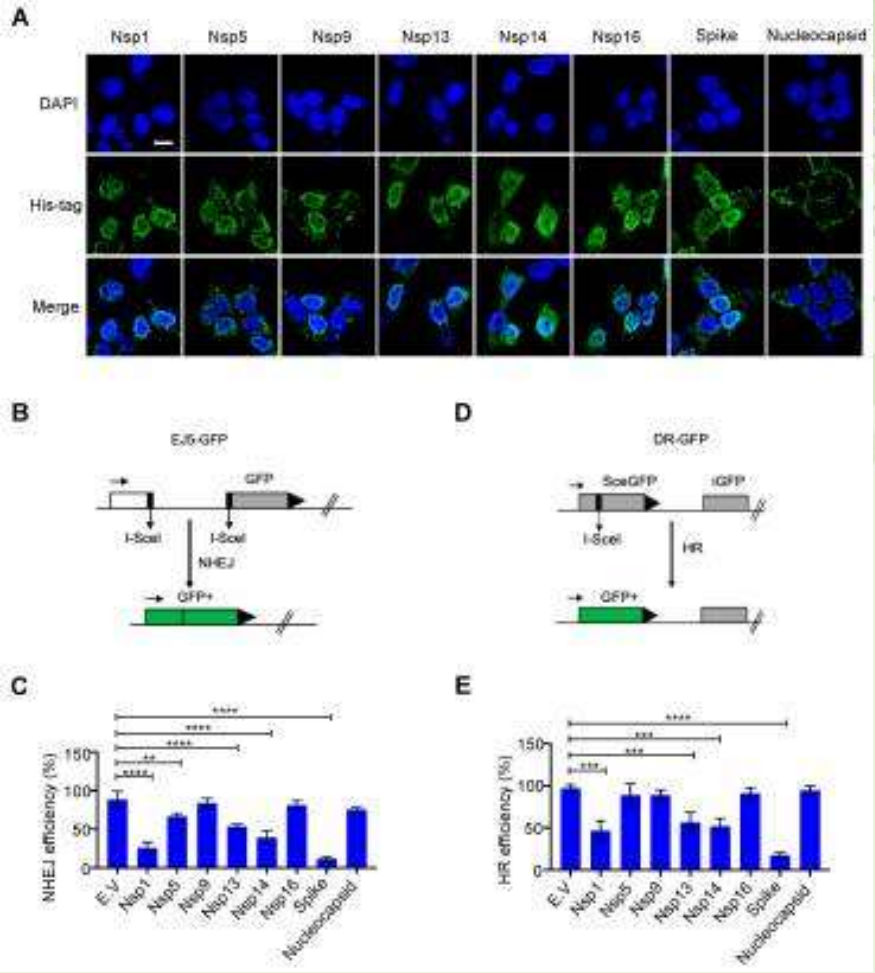
The Cardiovascular Link

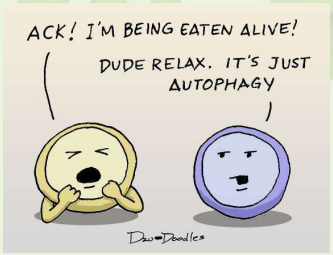
- **Circulation to the thyroid gland**
- **TBG Antibodies in the bloodstream**
  - Increased T-reg, antibody, and NK Cell immune dysregulation
- **Microclotting**
  - Interruption of normal blood flow
- **Clinical observation of greater variance of MCV and RDW**
  - Bone marrow spike protein
  - Including skull to brain

Autoimmunity | Autophagy | Autoimmunity

- We are inherently autoimmune
- Normal antibody production and cell removal is defined as apoptosis or autophagy
- Out of range antibody and cytotoxic activity is autoimmunity
- Predisposed apoptotic targets are converted into autoimmune victims by accelerated T-cell & NK cells
- There are 3 phases of progression in autoimmunity
    1) Genetic & Cell Wall Alteration
    2) Molecular Mimicry
    3) Epitope Spreading/ Bystander Activation

Genetic & Cell Wall Alteration | Phase 1





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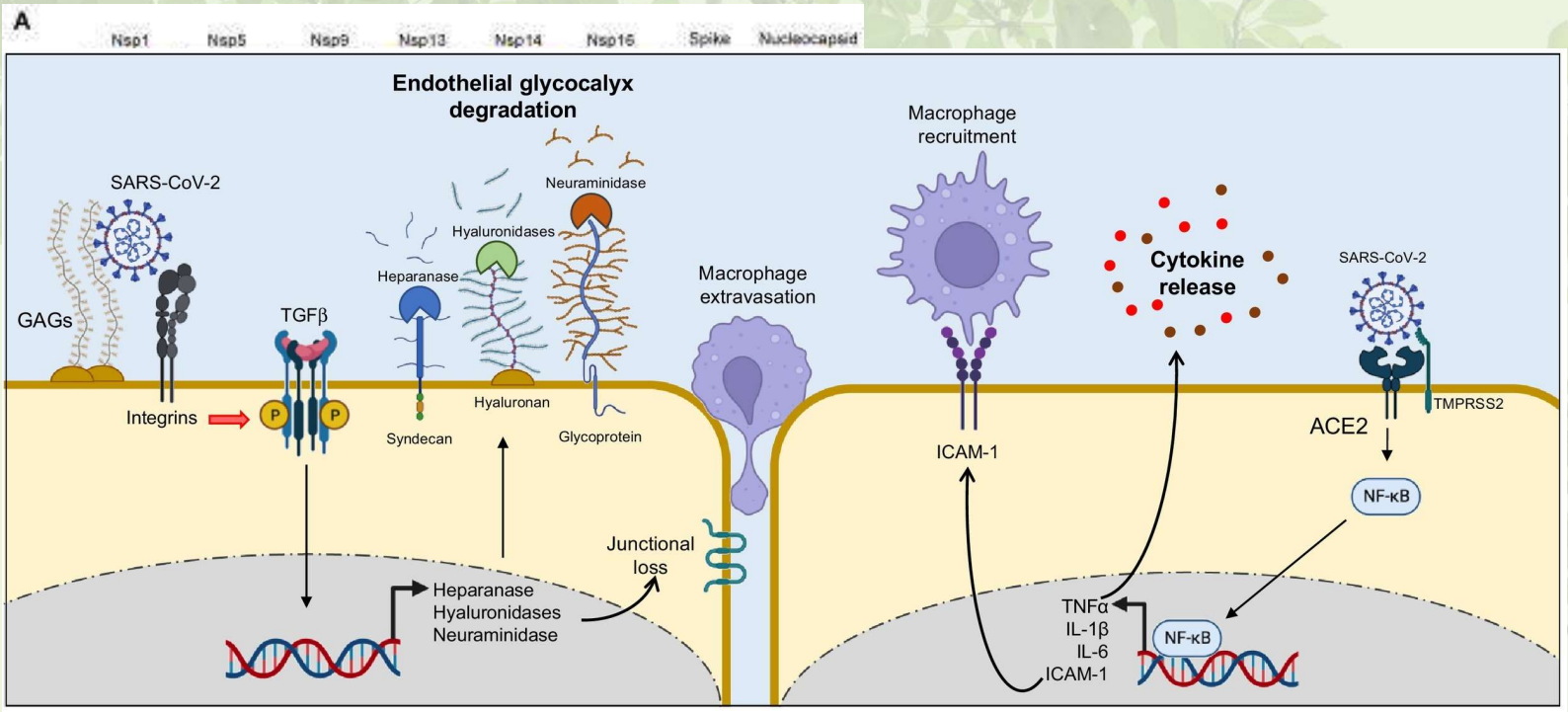
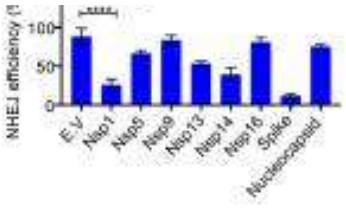
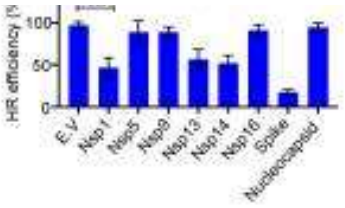
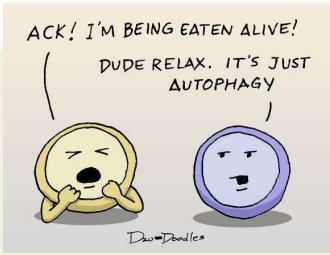
Genetic & Cell Wall Alteration | Phase 1









Trends in Microbiology

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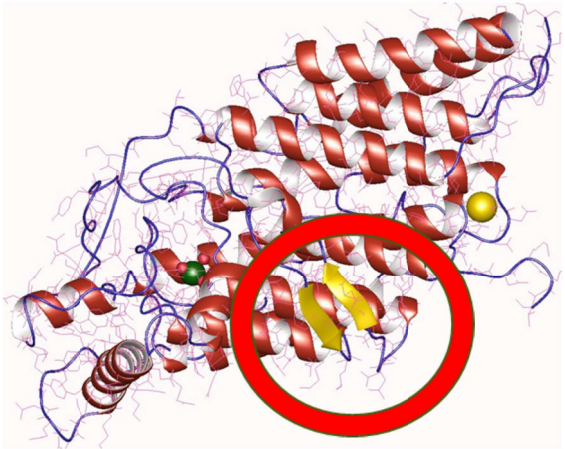
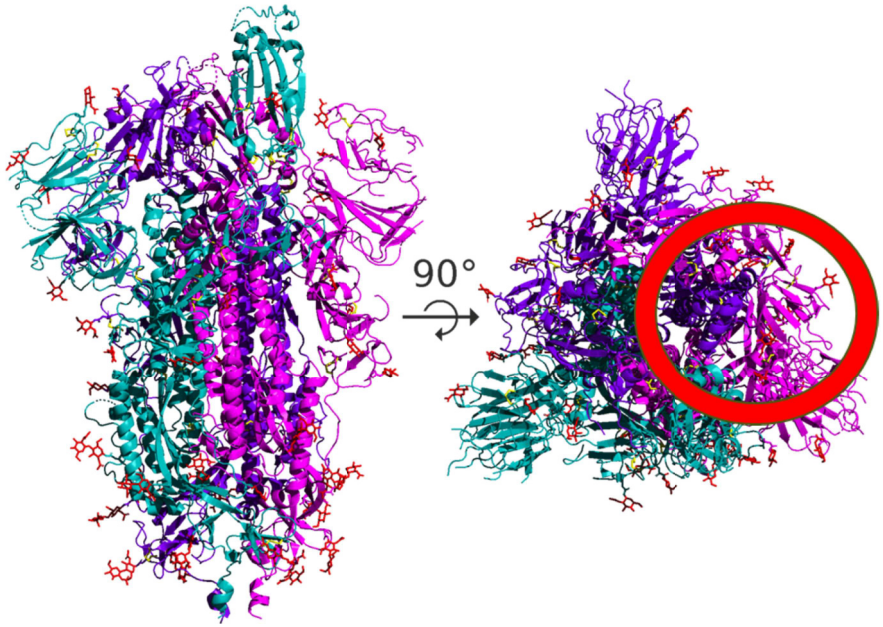
Molecular Mimicry-Protein Specific | Phase 2



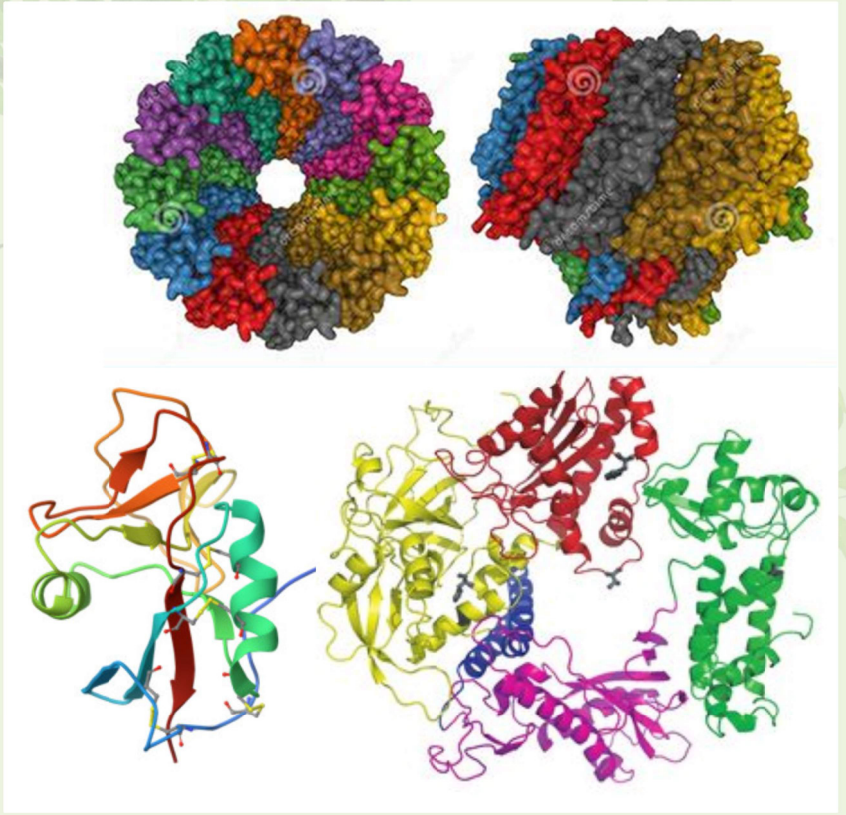
TPO Enzyme Structure



SARSCov-2 Protein Structure

Epstein-Barr, HHV-6, and Covid

- Epstein-Barr and HHV-6 have long been known as autoimmune instigators for many autoimmune disorders.
- Molecular mimicry is the leading theory as to why.
- Endemic viruses downregulate vitamin D receptors and interferon production to decrease immune recognition.
- Protein synthesis and autophagy is decreased in older populations.



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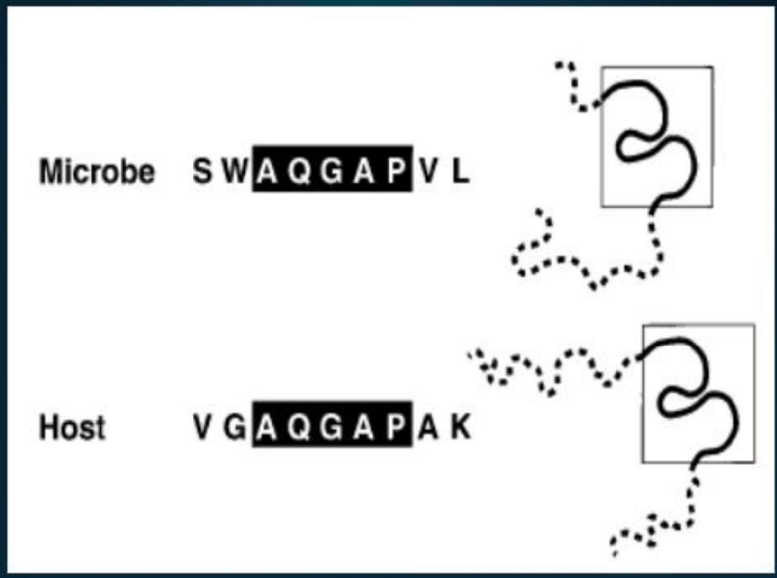
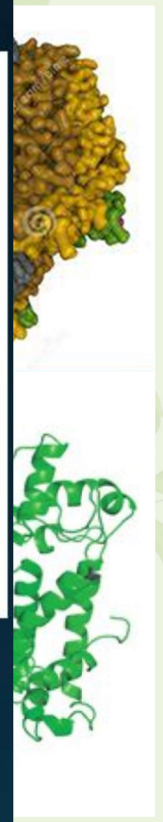
Molecular Mimicry

- Microbe and Host Cell:
  - Share of a linear amino acid sequence
  - Share of conformation fit
- Host immune response against the microbe reacts if the host sequence comprises a biologically important domain
- Autoimmunity may occur



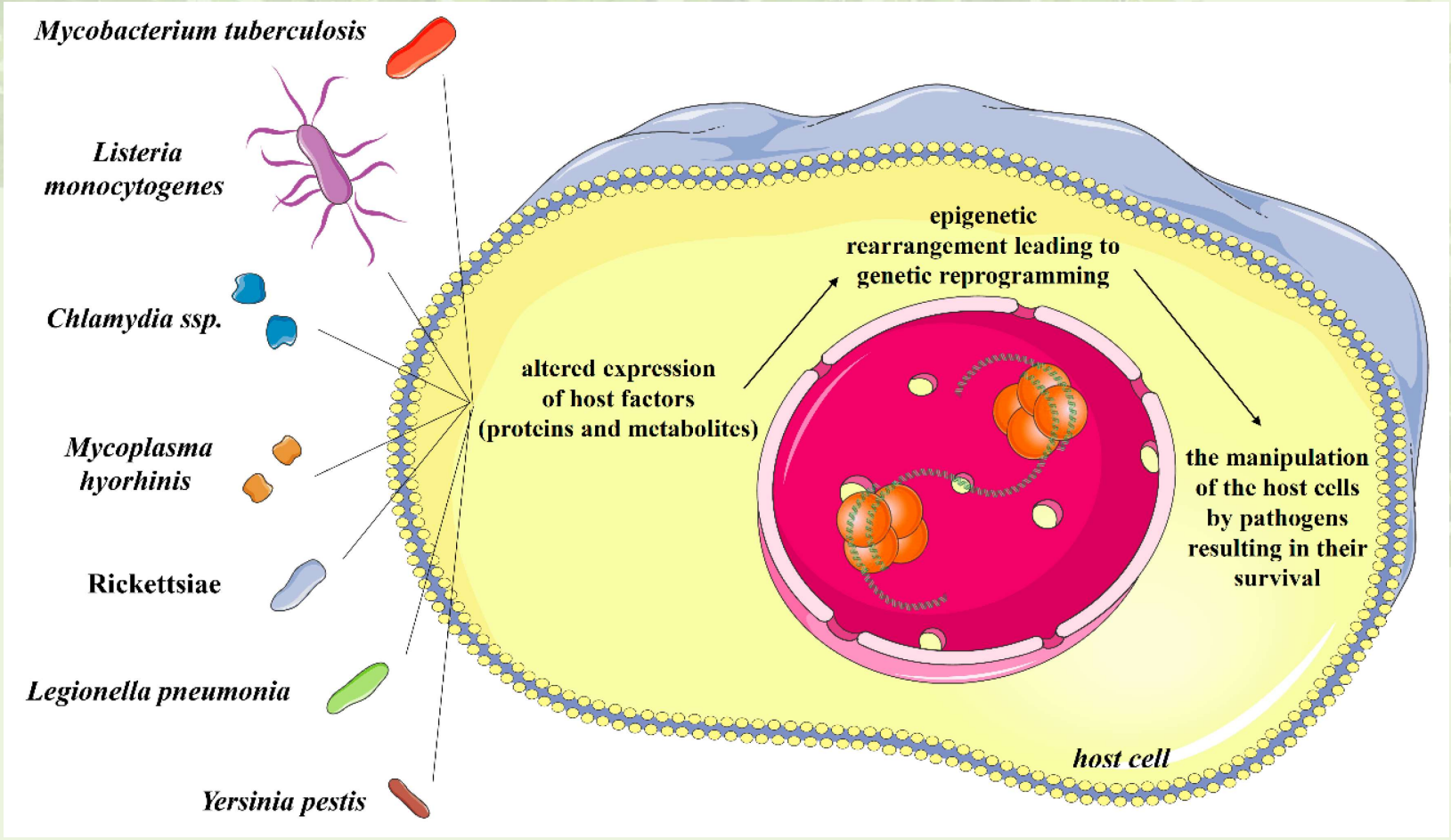
(Oldstone, 1998)



Mycoplasmic Infections

Intercellular Mycoplasms

m. pneumoniae implicated in GBS, scleroderma, RA, ALS, SLE



Epitope Spreading | Phase 3

(aka “Bystander Activation”)

- Epitope (determinant) spreading is the development of immune responses to endogenous epitopes secondary to the release of self antigens during a chronic autoimmune or inflammatory response.
- The past year has seen considerable advances in our understanding of the contribution of epitope spreading to the chronic pathogenesis of experimental T-cell-mediated and antibody-mediated autoimmune diseases.
- Most significantly, conclusive functional evidence for a major role for epitope spreading in the chronic athogenesis of murine relapsing-remitting experimental autoimmune encephalomyelitis, a CD4 + T-cell-mediated model of multiple sclerosis, was forthcoming.

Epitope Spreading | Phase 3

(aka "Bystander Activation")

- Epitope (determinant) spreading is the development of immune responses to endogenous epitopes secondary to the release of self antigens during a chronic autoimmune or inflammatory response.

PLP178–191 is primarily responsible for relapses in SJL/J mice primed with PLP139–151.

Tolerogenic treatment*	Number of relapses per mouse	Relapse frequency	Mean peak clinical score	Mean day of onset of relapse
Sham-SP	8/10	0.80	2.9	33
PLP139–151-SP	5/10	0.50	2.8	37
PLP178–191-SP	**2/10†**	**0.2†**	2.7	38
PLP139–151-SP + PLP178–191-SP	**2/10†**	**0.2†**	2.5	42

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self antigens during a

PLP178–1
Tolerogeni treatment*
Sham-SP
PLP139–1
PLP178–1
PLP139–1 PLP178–1

Article | Open access | Published: 31 October 2023

Antigen presentation by B cells enables epitope spreading across an MHC barrier

Cecilia Fahlquist-Hagert, Thomas R. Wittenborn, Ewa Terczyńska-Dyla, Kristian Savstrup Kastberg, Emily Yang, Alysa Nicole Rallistan, Quinton Raymond Markett, Gudrun Winther, Sofie Fonager, Lasse F. Voss, Mathias K. Pedersen, Nina van Campen, Alexey Ferapontov, Lisbeth Jensen, Jinrong Huang, John D. Nieland, Cees E. van der Poel, Johan Palmfeldt, Michael C. Carroll, Paul J. Utz, Yonglun Luo, Lin Lin & Søren E. Degn

Nature Communications **14**, Article number: 6941 (2023) | Cite this article

Abstract

Circumstantial evidence suggests that B cells may instruct T cells to break tolerance. Here, to test this hypothesis, we used a murine model in which a single B cell clone precipitates an autoreactive response resembling systemic lupus erythematosus (SLE). The initiating clone did not need to enter germinal centers to precipitate epitope spreading. Rather, it localized to extrafollicular splenic bridging channels early in the response. Autoantibody produced by the initiating clone was not sufficient to drive the autoreactive response. Subsequent epitope spreading depended on antigen presentation and was compartmentalized by major histocompatibility complex (MHC). B cells carrying two MHC haplotypes could bridge the MHC barrier between B cells that did not share MHC. Thus, B cells directly relay autoreactivity between two separate compartments of MHC-restricted T cells, leading to inclusion of

#auth-Gudrun-Winther-Aff1

151.

Mean peak clinical score	Mean day of onset of relapse
2.9	33
2.8	37
2.7	38
2.5	42

How SARS COV-2 is special in Hashimoto's

Checks all the boxes

☑ Phase 1

☑ Phase 2

☑ Phase 3

Infection Instigated Autoimmunity

Microbe Name	Disorder(s)	Microbe Name	Disorder(s)
Porphyromonas Gingivalis	RA	*Entamoeba Histolytica*	Bone & Brain
Streptococcus Mutans	AI Myocarditis	*Giardia Lamblia*	GI, Intestinal autoimmunity
Helicobacter pylori	GI, Brain, CNT, Hashimoto's	*Cryptosporidium Parvum*	Colon AI, Celiac, NCGS
Camphylobacter Jejuni	GI, Brain, CNT	*Blastocystis Hominis*	IBS, Fibromyalgia
Yersinia Enterocolitica	GI, Eye, Arthritis, Hashimoto's	*HSP-60/Chlamydia*	Brain, Cardiovascular
Clostridium Difficile	GI, UC, Crohn's	*Streptozymes*	OCD, PANDAS, Heart, Arthritis
Candida Albicans	GI, all autoimmune	*Mycoplasmas*	SLE, Arthritis, Phospholipid AB
Rotavirus	GI, Type 1 diabetes	*Acinobacter*	MS

Microbe Name	Disorder(s)	Microbe Name	Disorder(s)
Klebsiella	CNT, Skeletal, Eye	*HHV-6*	CFS, Fibromyalgia, SLE, Brain, Hashimoto's
Mycobacterium Avium	GI, Hashimotos, MS Type 1 diabetes	Borrelia Burgdorferi	BBB, Brain , Arthritis
Aspergillus	CFS, Fibromyalgia, Brain	Babesia, Ehrlichia, Bartonella	BBB, Brain, Arthritis
Penicillium	CFS, Fibromyalgia, Brain	Influenza	Encephalomyelitis
Stachybotrys	CFS, Fibromyalgia, Brain	Semliki Forest Virus	AI Demyelinating Disease
EBV	CNT, SLE, Brain/ MS, Hashimoto's	Herpes Simplex	AI Encephalitis
Hepatitis C	Liver, p450 Hepatocyte AI	Epstein-Barr Virus	AI hepatitis
Cytomegalovirus	Type 1 diabetes, CNT, SLE, Brain	Coxacie Virus	AI Myocarditis

SARS COV-2 Virus Autoimmunity

Microbe Name	Disorder or Tissue Name
SARS COV-2 “COVID19”	

frontiers in Immunology

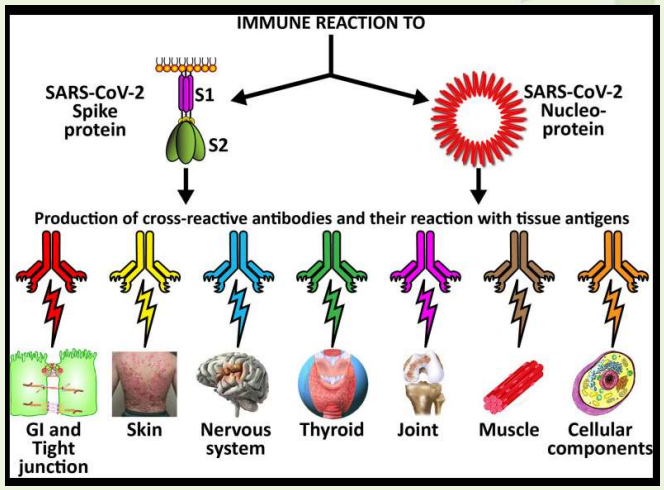
Front Immunol. 2020; 11: 617089.
Published online 2021 Jan 19. doi: 10.3389/fimmu.2020.617089
PMCID: PMC7873987
PMID: 33584709

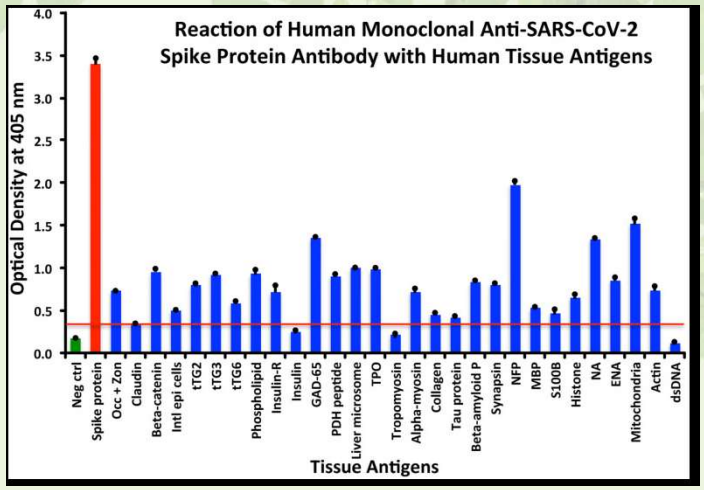
Reaction of Human Monoclonal Antibodies to SARS-CoV-2 Proteins With Tissue Antigens: Implications for Autoimmune Diseases

Aristo Vojdani, [1,2,*] Elroy Vojdani, [3] and Datis Kharrazian [2,4,5]

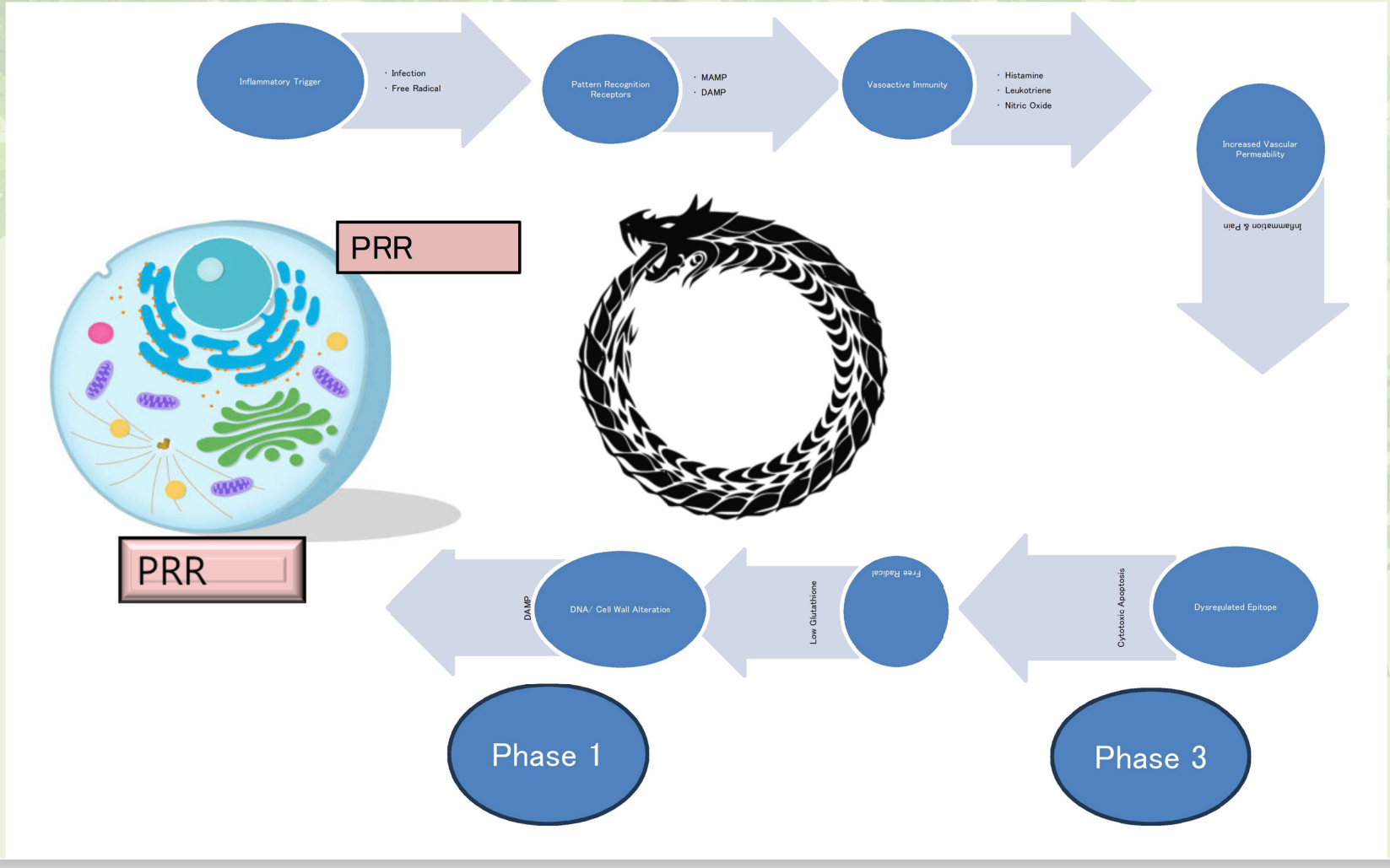
Journal Article

Author information · Article notes · Copyright and License information PMC Disclaimer





The MAMP/DAMP Progression to Autoimmune Feedback Loop



Infection vs. MRNa Gene Therapy

- Lipid Nanoparticles-serum of the “vaccines” with toxic liposomal base
  - **Moderna**
    - SM-102
    - Polyethylene glycol (PEG) 2000 dimyristoyl glycerol (DMG)
    - 1,2-distearoyl-sn-glycero-3-glycero-3-phosphocholine (DSPC)
  - **Pfizer**
    - (4-hydroxybutyl)azanediyl)bis(hexane-6, 1-diyl)bis(2-hexyldecanoate)
    - 2 [(polyethylene glycol)-2000]-N,N-ditetradecylacetamide
    - 1,2-Distearoyl-sn-glycero-3-phosphocholine
- Microclotting
- IgG4
- Development of self organizing nanobots?

Infection vs. MRNa Gene Therapy

- …accines" with toxic liposomal base

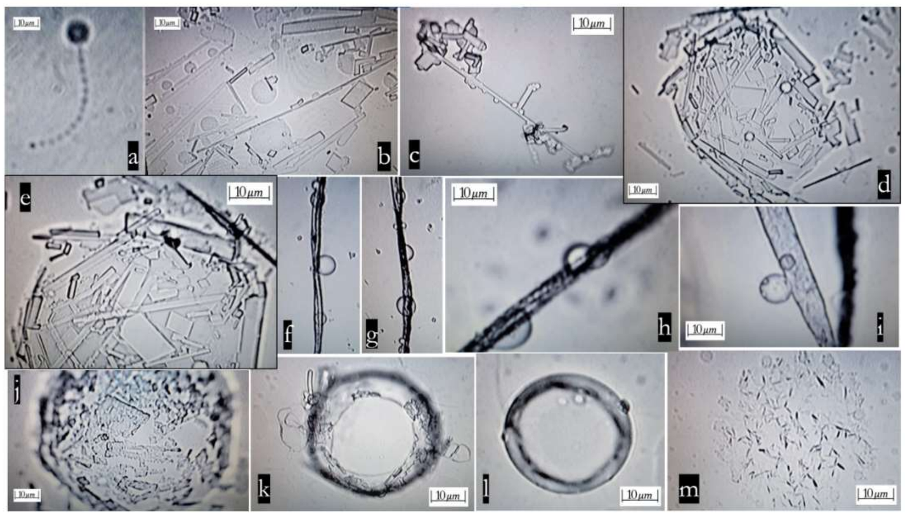


Figure 11. Findings for Pfizer incubation study for 372 days; (a) Day 22, this is what we describe as a beaded chain (at 400X magnification); (b) Day 24, 2-dimensional geometric self-assembly at the bottom (at 200X magnification) in normal saline; (c) Day 60, floating 3-dimensional detailed chip-like structures (at 400X magnification) in distilled water; (d) and (e) day 60, accumulated 3-dimensional chip-like structures within an oval shaped boundary (200X/400X) in distilled water; (f), (g), (h), (i) Floating filaments shedding bubbles inside and outside in normal solution at day 95 (100x/100x/200x/200x); (j), (k), (l), (m) Progressive degenerative changes in distilled water 200X (day 82/day 256/day 306/day 372).

…2000 dimyristoyl glycerol (DMG)

…-glycero-3-phosphocholine (DSPC)

…)bis(hexane-6, 1-diyl)bis(2-hexyldecanoate)

…00]-N,N-ditetradecylacetamide

…3-phosphocholine

International Journal of Vaccine Theory, Practice, and Research

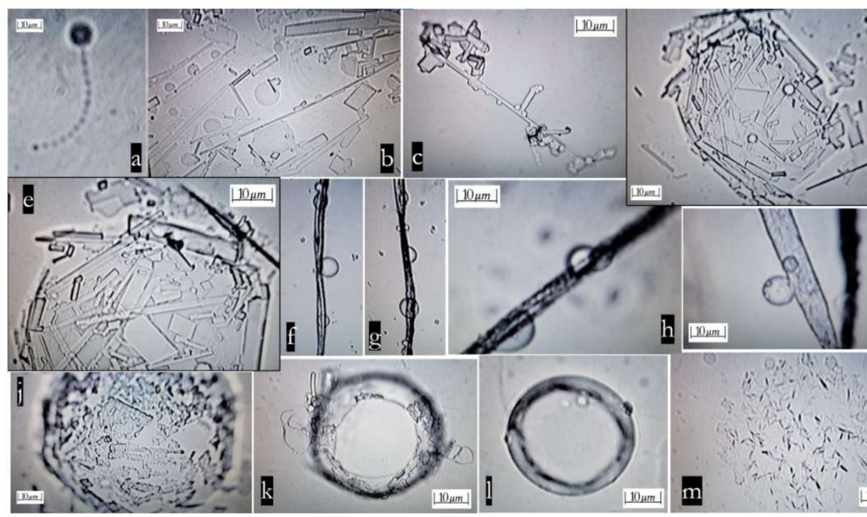
IJVTPR

Real-Time Self-Assembly of Stereomicroscopically Visible Artificial Constructions in Incubated Specimens of mRNA Products Mainly from Pfizer and Moderna: A Comprehensive Longitudinal Study

- Mic…
- IgG…
- Dev… …ing nanobots?

Infection vs. MRNa Gene Therapy

accines" with toxic liposomal base



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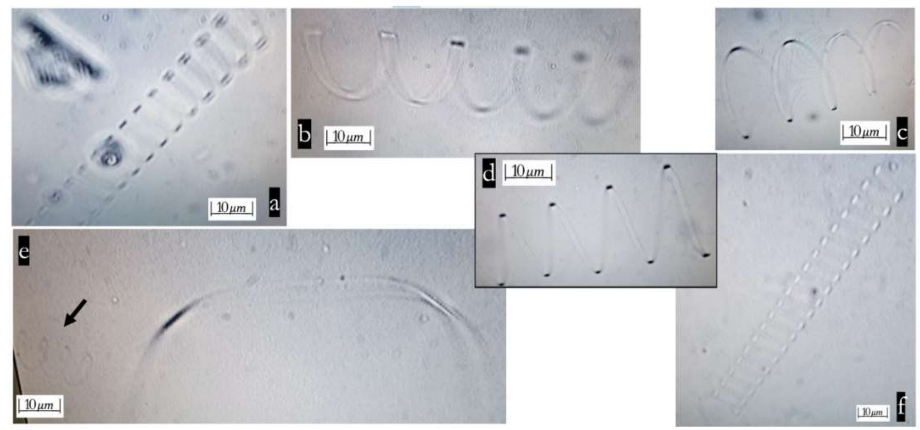

Figure 12. Various coils, ribbons, and spirals in Pfizer, distilled water: (a) Day 60 (at 200Xmagnification); (b) ~ (e) Day 74 (at 200X magnification); (f) Day 176 (at 100X magnification).

e)

00]-N,N-ditetradecylacetamide

3-phosphocholine

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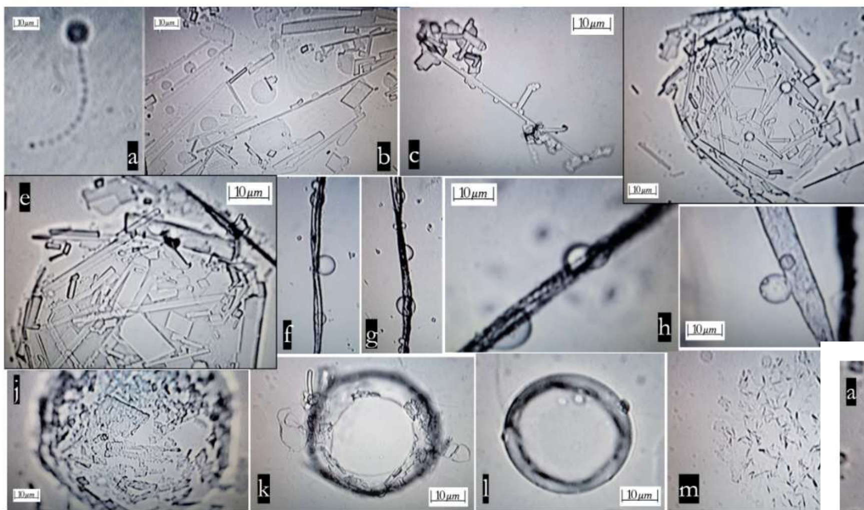

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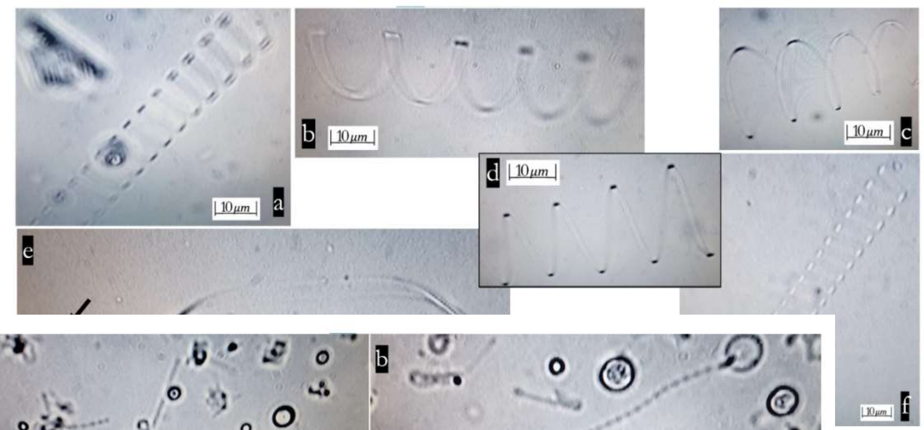

(f) Day 176 (at…

…e)

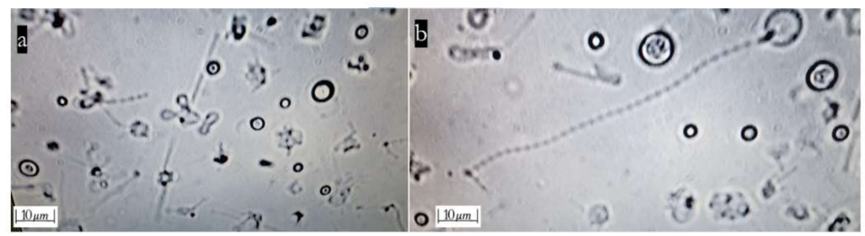

Figure 13. Beaded Chains and Assorted Structures in Pfizer Distilled Water (Day 176, 400x): (a) Various artificial satellite-like structures, (b) Long beaded chains gathered on the central surface of the medium.

- Mic…
- IgG…
- Dev… …ing…

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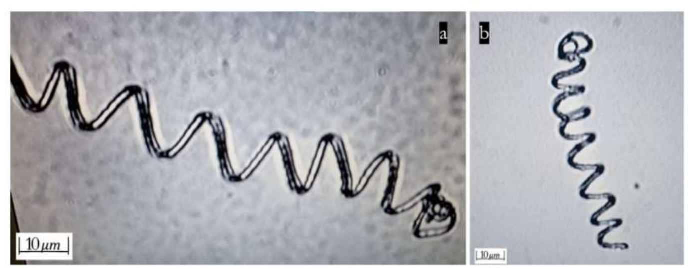

Figure 14. Typical Algae-typed Magnetic Nanobot-like Spirals in Pfizer in distilled water: (a) Day 176 (400x); (b) Day 337 (200x).

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What About Our Own Proteins?

Infections may be the triggering event for dysfunctional immune cycles.

- Cytokines are protein based and can be reduced with proteolytic enzymes.
- NFKB inhibitors reduce the signaling that releases NK cells.
- Enzymes such as the TPO and TBG are protein based, so are often targets for autoimmunity. The targeting mechanism is believed to be pH based. Alkalization of body fluids and tissues regains targeting control of both enzymes and antibodies.

Using Transformation Enzyme® Products

- **Proteolytic enzymes** - Protease, Protease 375K, Protease IFC
- **Lipolytic enzymes** - Lypo, LypoZyme
- **Immune support** to resolve infection - Immune AV
- **Anti-inflammatory** support - RepairZyme
- **Cellular repair** - Super CellZyme

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