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**NO GRAIN
NO PAIN**





Autoimmune 101:

Understanding the origins of an immune system gone rogue...

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What is Autoimmunity?

According to the



“An autoimmune disease is an illness that causes the immune system to produce antibodies that attack normal body tissues. Autoimmune is when your body attacks itself. It sees a part of your body or a process as a disease and tries to combat it.”

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Autoimmunity Statistics



- An estimated 50 million Americans suffer with autoimmune disease (AID) compared to 9 million with cancer and 22 million with heart disease.
- AID is the #1 cause of death in women under the age of 65
- There are more than 100 forms of AID currently recognized by research.
- Symptoms cross many specialties and can affect all body organs.
- Medical education provides minimal learning about autoimmune disease.
- Specialists are generally unaware of interrelationships among the different autoimmune diseases.
- Initial symptoms are often intermittent and unspecific until the disease becomes acute.
- Research is generally disease-specific and limited in scope. More information-sharing and crossover among research projects on different autoimmune diseases is needed.

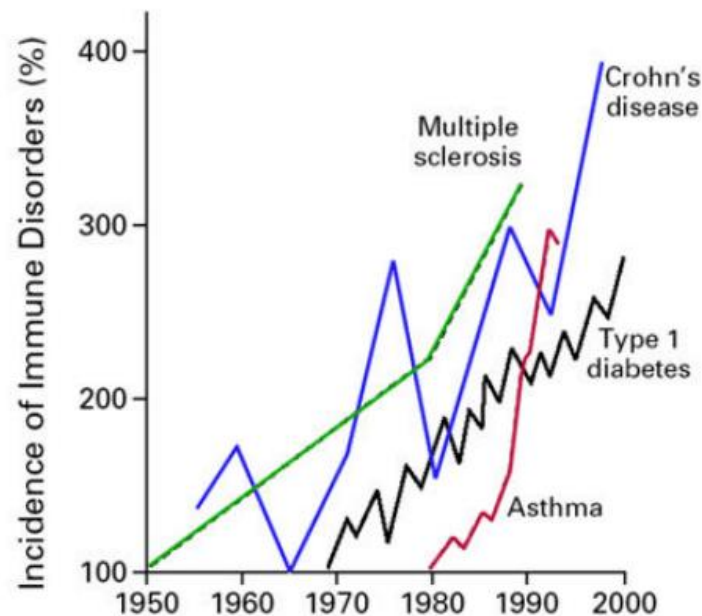
Source: [American Autoimmune Related Disorders Association](http://www.aar-da.org)

Examples of Autoimmune Disease

- Hashimoto's hypothyroidism
- Rheumatoid Arthritis
- Celiac Disease
- Ulcerative Colitis
- Type I Diabetes
- Asthma
- Sjogren's
- Lupus
- Vitiligo
- Psoriasis
- Reactive Arthritis
- Scleroderma
- Dermatomyositis
- Vasculitis
- Raynaud's phenomenon
- Transverse myelitis
- Sarcoidosis
- Autoimmune hepatitis
- Dysautonomia
- Myocarditis
- Narcolepsy
- Multiple Sclerosis
- Alopecia Areata
- Addison's Disease
- Graves' Disease
- Fibromyalgia
- Endometriosis
- Eosinophilic Esophagitis
- Nephropathy (kidney damage)
- TPP
- Guillain-Barre syndrome
- Restless leg syndrome
- Uveitis
- Crohn's disease

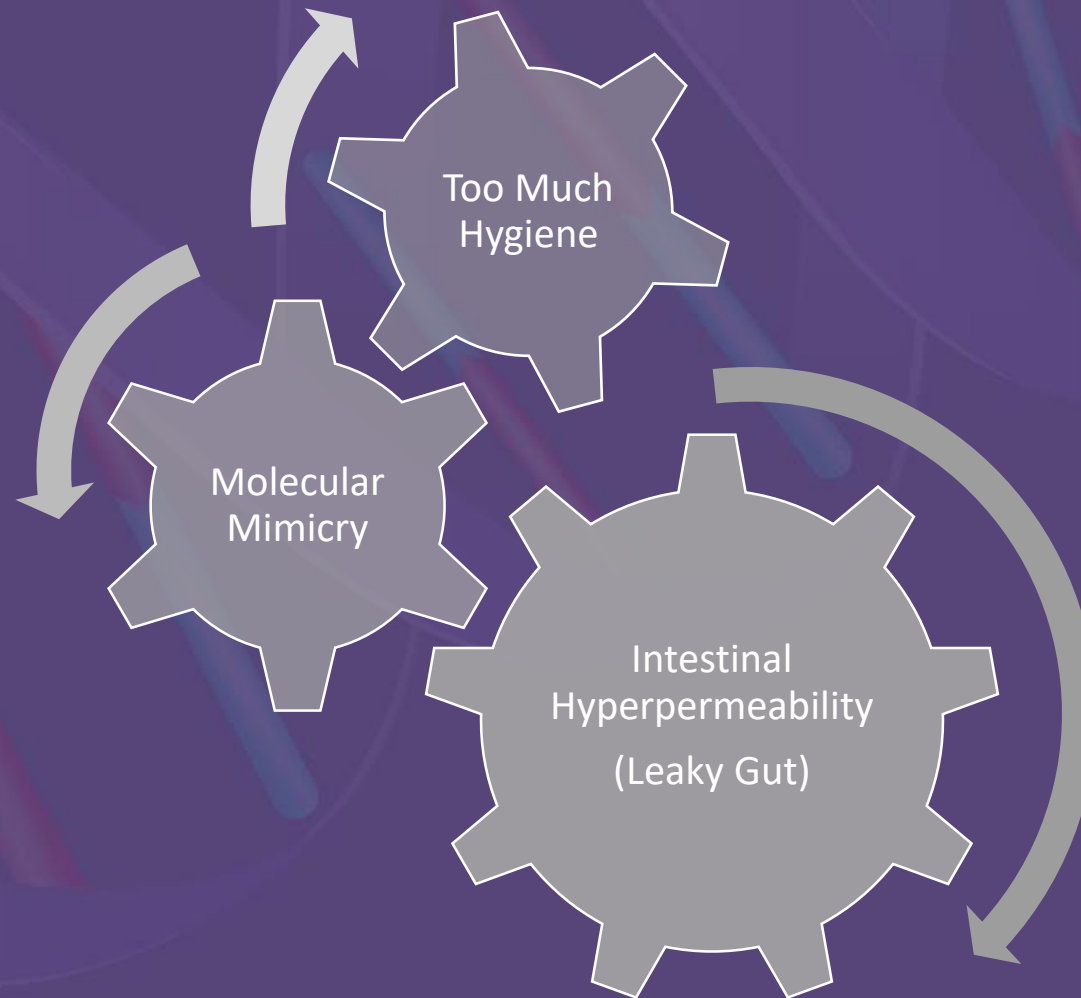
The problem is getting worse...

Figure 1: Rising Incidence of Autoimmune Disorders



From: Bach JF. The effect of infections on susceptibility to autoimmune and allergic diseases. N Engl J Med. Sep 2002;347(12):911-920.

Scientifically Recognized Central Mechanisms Involved in AID

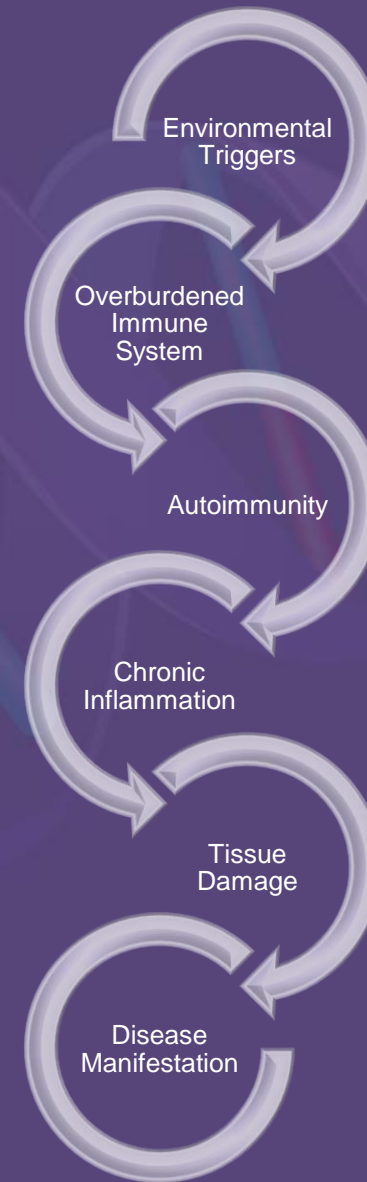


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Leaky Gut = Pre-Autoimmunity

- Intestinal Hyperpermeability
- 70-80% of the immune system is in the gut



KNOWN CAUSES OF LEAKY GUT



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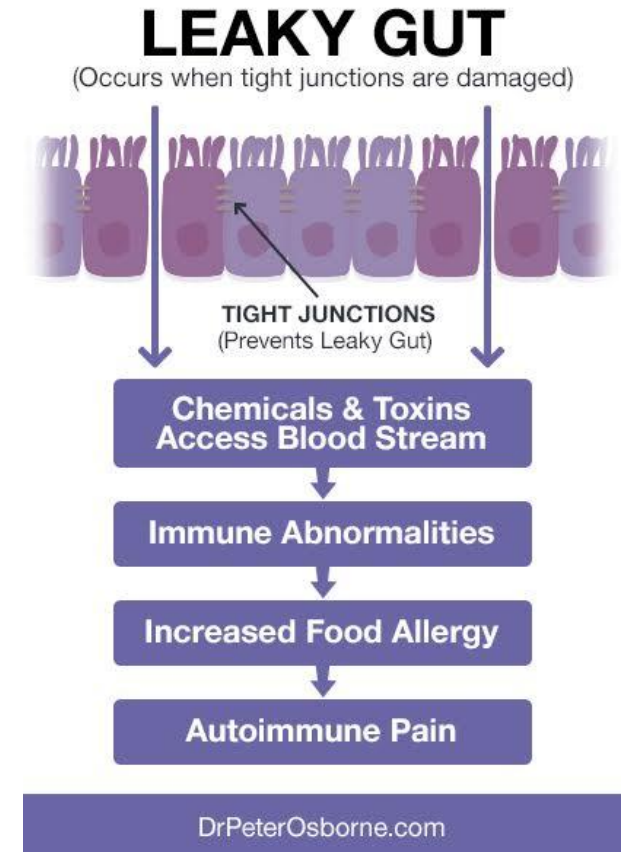
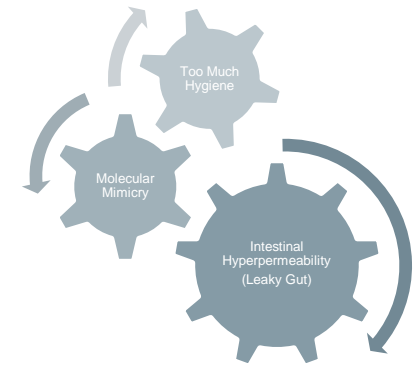
Gluten Free
SOCIETY
www.GlutenFreeSociety.org

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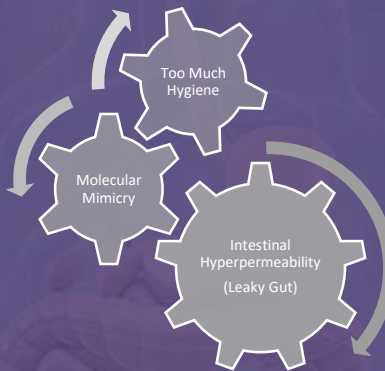
What Does Leaky Gut Damage Do?

- Overstimulates the immune system
- Allows bacterial and viral toxins access to the central circulation
- Causes allergic hypersensitivity to foods
- Sets the stage for **Molecular Mimicry**
- Linked to abnormal microbiome
- Inflammation of the GI Tract
- Malabsorption of vitamins, minerals, and other nutrients
- **Triggers Systemic Leaky Barrier Syndrome**



What Causes AID

We have 1 model of AID where the scientific consensus agrees on the cause.



Celiac Disease – Gluten

Food & Nutrition

Allergens Nutrients Too much of too little

Chemicals in Food

Pesticides, herbicides
Food dyes Preservatives
Flavoring agents

Infections

Bacteria Fungus, mold, yeast
Virus Parasite

Lack of Sunshine

Vitamin D deficiency
Melatonin Deficiency

Excessive Stress

Relationships Work
Life purpose
Time management

Air Quality

Smoking Indoor pollutants
Outdoor pollutants EMF Air “fresheners”

Water Quality

Fluoride
Chlorine & chloramine Medications

Medical Interventions

Vaccines Amalgams
Medications Surgical implants

Lack of Sleep

Artificial light sources
Caffeine over utilization

Lack of Physical Activity

Sedentary lifestyle
Convenience

**There is no magic bullet.
AID is multifactorial.**

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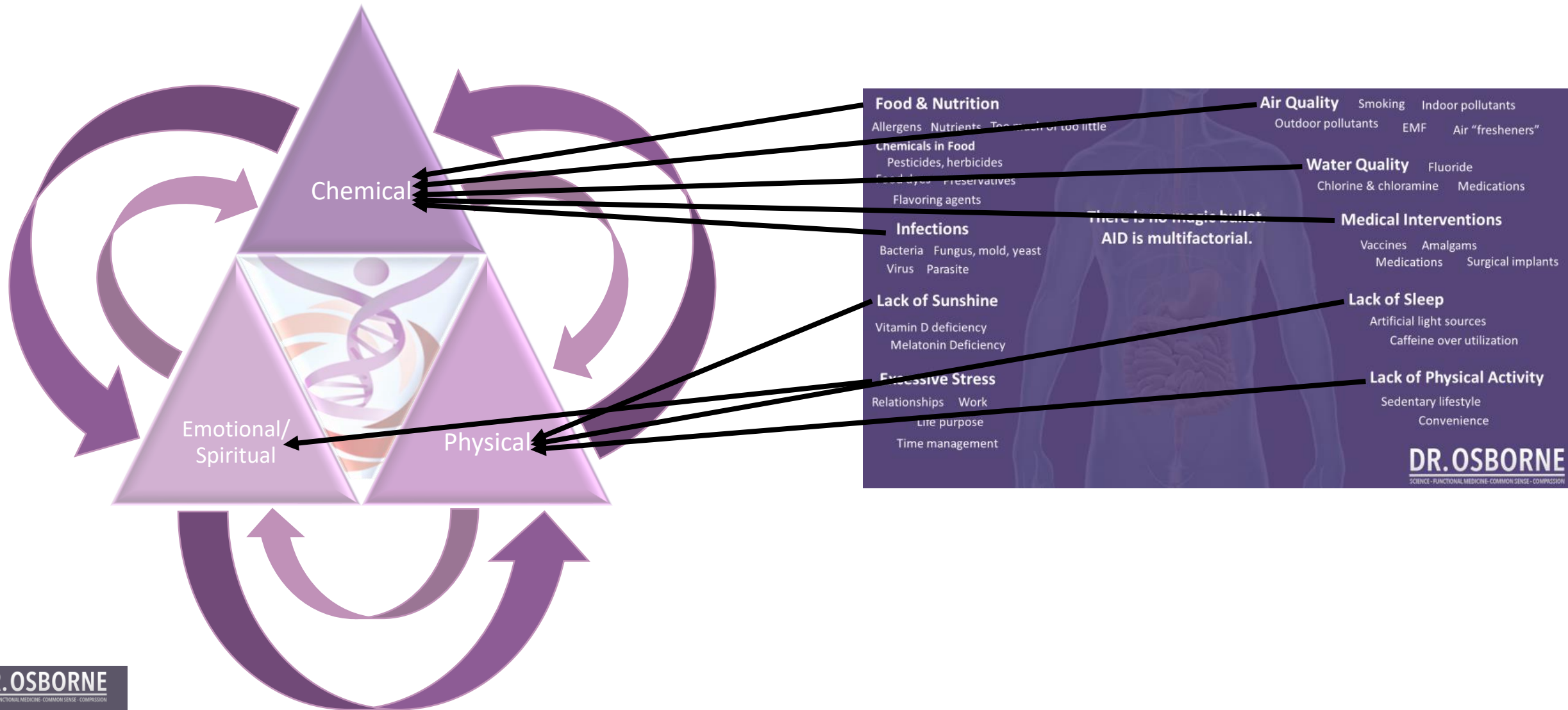
Should We Be Worried?

“Of all of man’s interventions in the natural order, none is accelerating quite so alarmingly as the creation of chemical compounds....There is, however, a price to pay for an industrial society that has come to rely so heavily on chemicals: almost 35,000 of those used in the U.S. are classified by the Environmental Protection Agency (EPA) as being either definitely or potentially hazardous to human health”

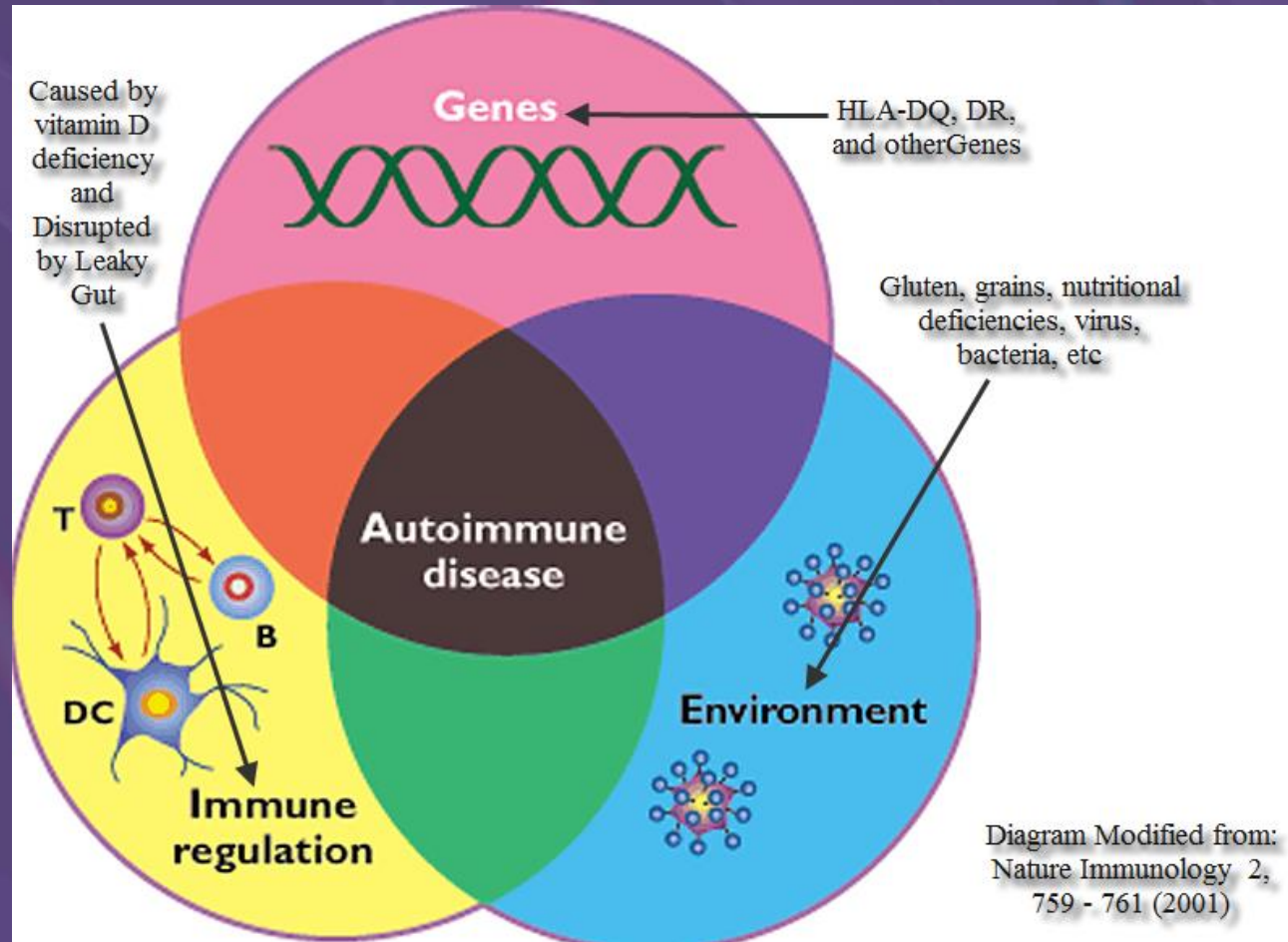
Douglas M. Costle,
Administrator of the EPA



Phenotypic Expression = You + Your Gene Interaction



Is Autoimmunity The Perfect Storm?



There is HOPE...

- Genes do not make you sick...
- Subjecting your genes to the wrong environment does.
- You have the capacity to:
 - Educate yourself
 - Take action on what you learn
 - ...and by doing so alter the outcome of your health.

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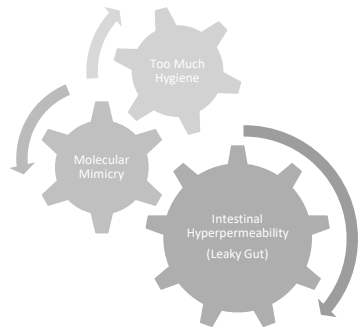
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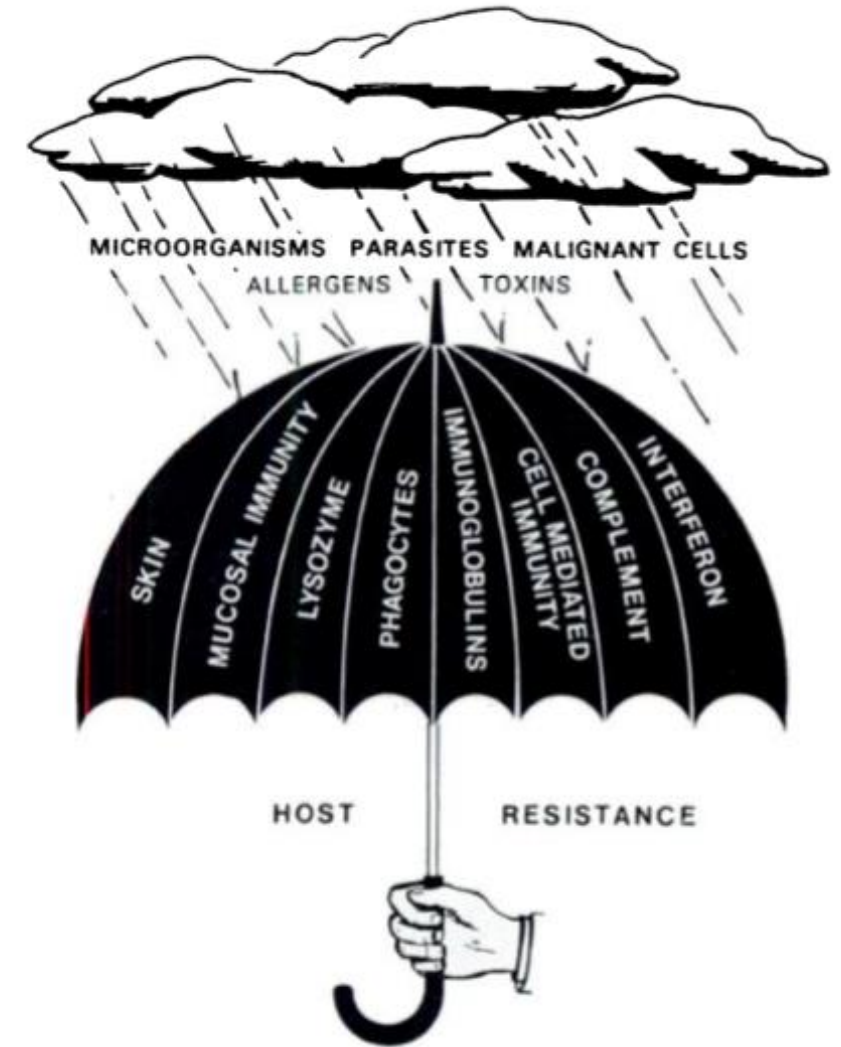
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Nutrition and the Immune System...



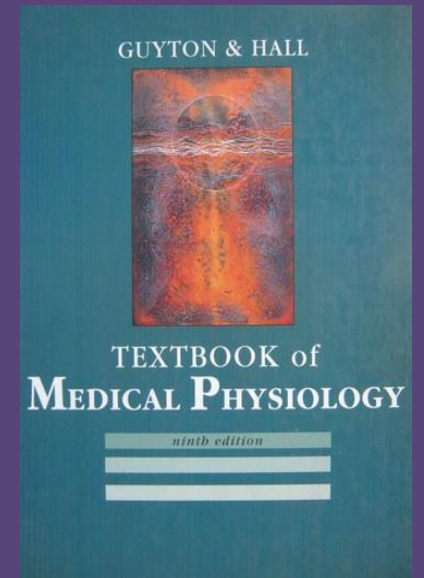
Am J Clin Nutr 1991;53:1087-1101.

Biochemistry = Nutrition

All of the known factors involving autoimmune disease are affected by nutrition

“Each of the 100 trillion cells in the human being is a living structure that can survive indefinitely and, in most instances can even reproduce itself, provided it's surrounding fluids contain appropriate nutrients.”

Guyton, A. Textbook of Medical Physiology 9th edition. P. 11, W.B. Saunders Co., Philadelphia, 1996.

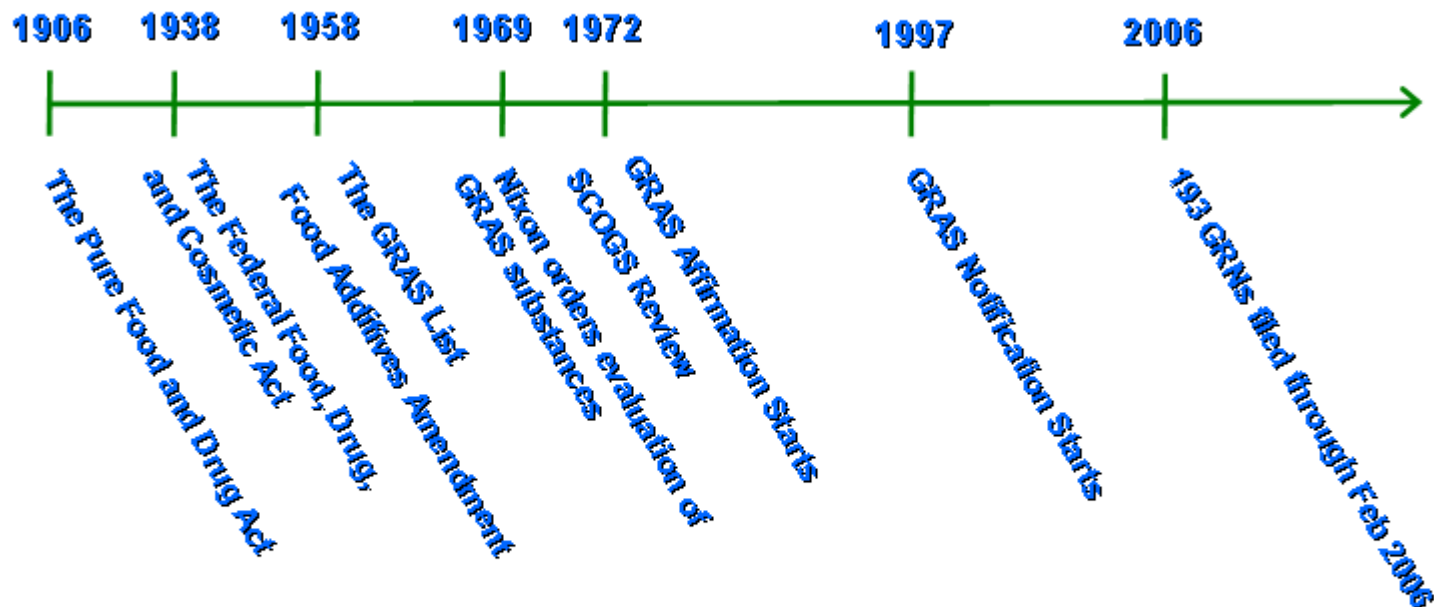


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Food Additives

A GRAS Timeline



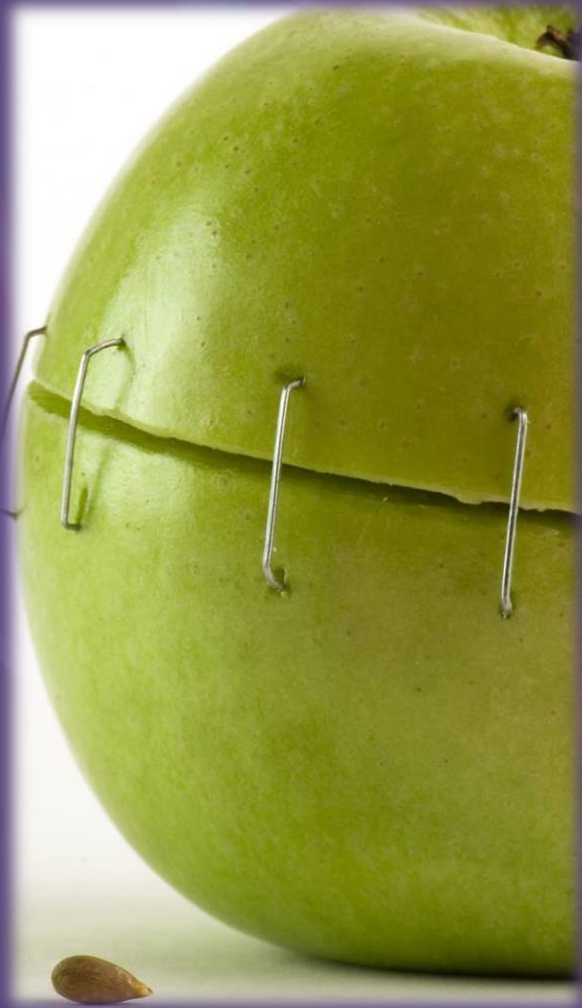
This picture is a graphical representation of a timeline starting in 1906 when the Pure Food and Drug Act was passed. Milestones along the way to the present include the 1938 Federal Food Drug and Cosmetic Act, and the Food Additives Amendment and the GRAS list in 1958. In 1969, President Nixon ordered an evaluation of GRAS substances and in 1972 the GRAS Affirmation process began. The GRAS Notification Program started in 1997 and by the end of 2006, 193 GRAS Notices were filed.

Source: FDA.gov

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Frood...



- Aside from the possibility of reacting to real food...
- The FDA has approved approximately 3,000 food additives, preservatives, and colorings.
- The average person ingests 150 lbs. of additives every year.
- Many commonly eaten foods are genetically modified or contain genetically modified ingredients.

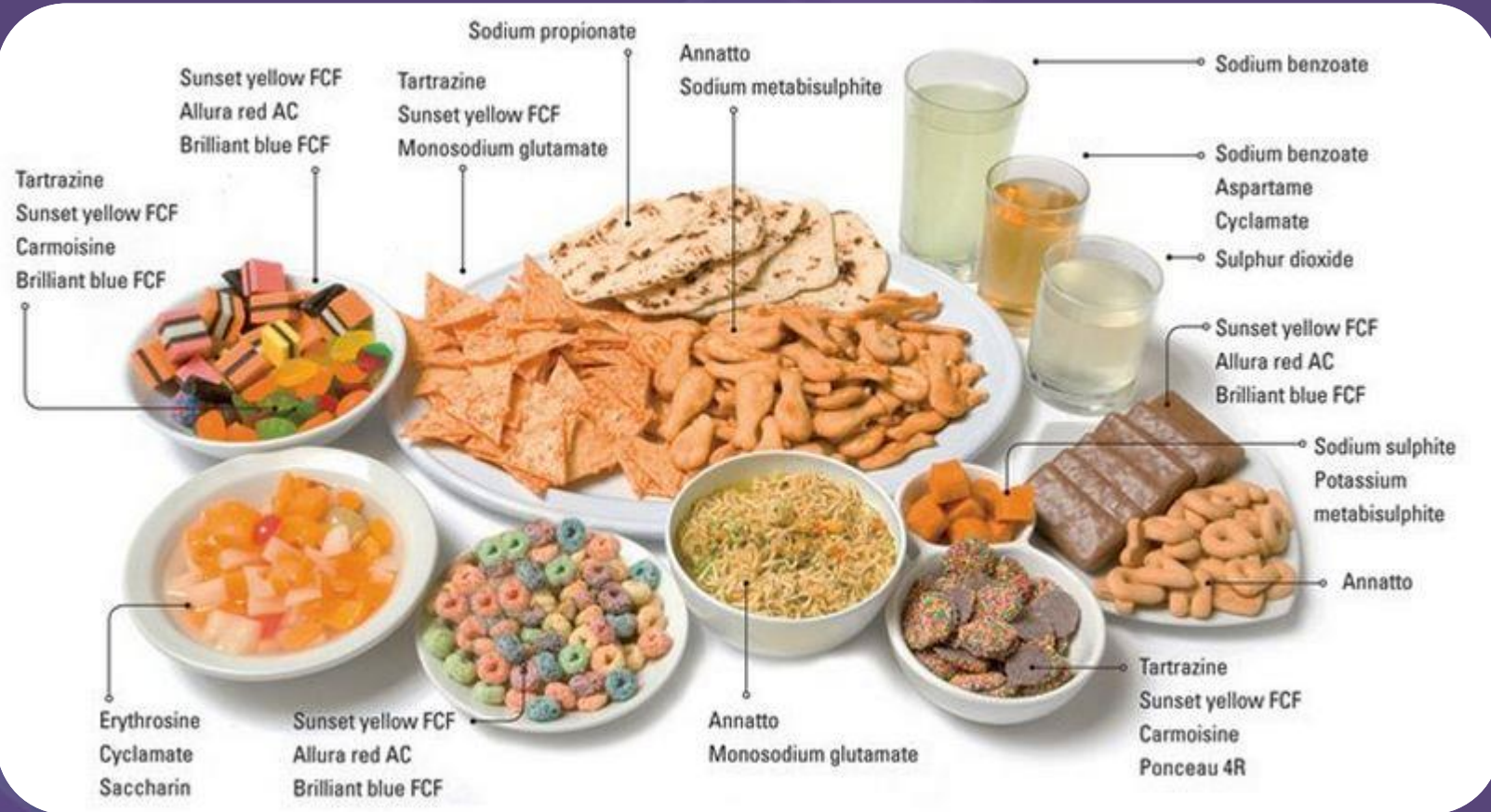
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TOXINS IN FOOD - Herbicides, Pesticides, Steroids, Hormones, Antibiotics, and Excitotoxins

- It takes approximately 5 to 8 pounds of chemically sprayed grain to produce 1 pound of beef. Therefore you will ingest considerably more cancer causing chemicals from meat than from fruit and vegetables.
- On average, one glass of inorganic, store-bought milk contains the residue of about a hundred different antibiotics. Once in our bodies, these antibiotics impact the microbiome and subsequently disrupt the immune system.



Pesticides

- **3 million tons of pesticides** are used each year worldwide --
More than **1,600 chemicals** are used in their production!
- **Most have not been tested for their toxic effects on humans.**
Exposure has been linked to:

Autoimmune Disease
Nervous system disorders
Immune system suppression
Childhood Cancer
Breast Cancer
Diabetes

Reproductive damage
Hormone problems
Asthma
ADHD - Autism
Migraine Headaches
Developmental delays

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Air Quality

- Our indoor environment is two to five times *more toxic* than our outdoor environment.

Environmental Protection Agency

- In some cases, the air measurements indoors have been found to be 100 times more polluted.

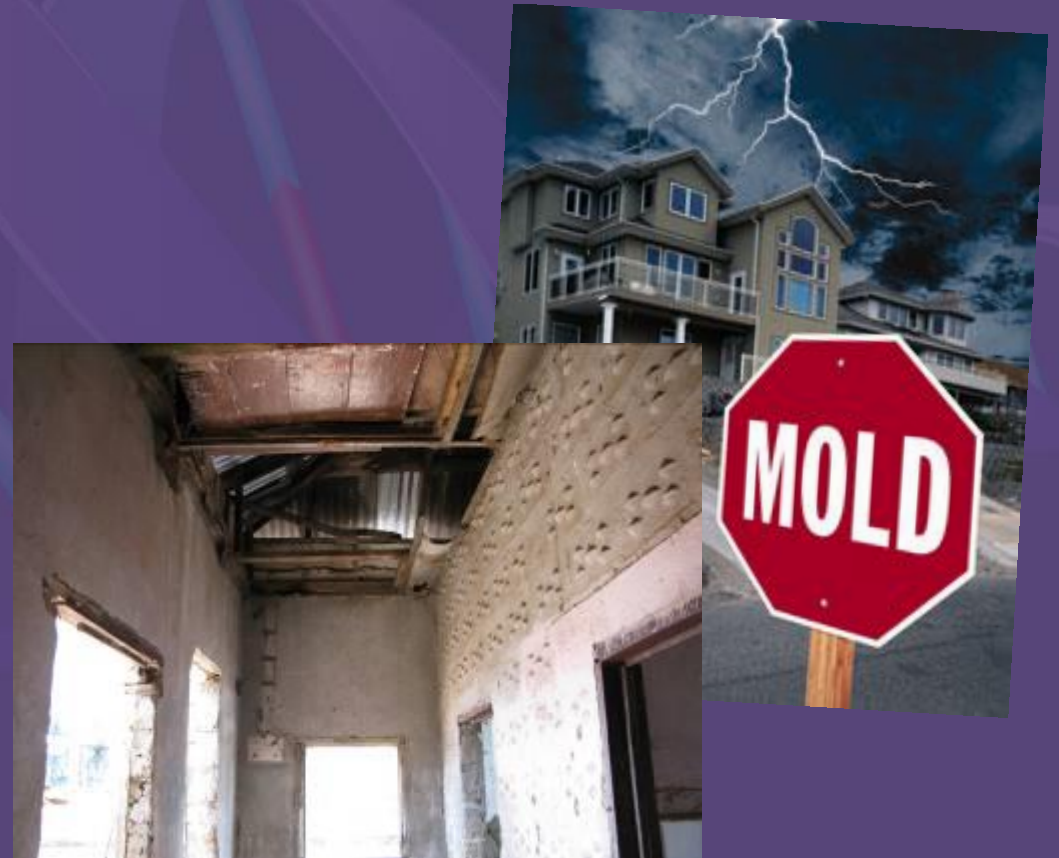
Environmental Protection Agency
Office Radiation and Indoor Air
Indoor Environment Division
www.epa.gov/iaq/indexold.html

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Indoor Air Quality

- Modern Construction
 - Chemical VOC's
- Invention of HVAC
- Aging buildings – office & school infrastructure
- Mold Morphology
 - 24% genetic susceptibility
- EMF emissions



Outdoor Air Quality

- Ozone
 - Automobile exhaust
 - Power plant emissions
 - Gasoline vapor and solvents
 - Sulfur dioxide
 - Outdoor allergens
-
- Increase the risk of asthma, upper respiratory infections, lung cancer, & heavy metal exposures

Source: Emory University School of Medicine.

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Water Quality

Tap water contains unhealthy contaminants including microorganisms, heavy metals, agricultural run-off, pharmaceuticals, household chemicals, lawn chemicals, gasoline, dry cleaning solvents, drugs, chlorine, fluoride, radioactive particles, lead and other impurities. Many of these are known carcinogens.



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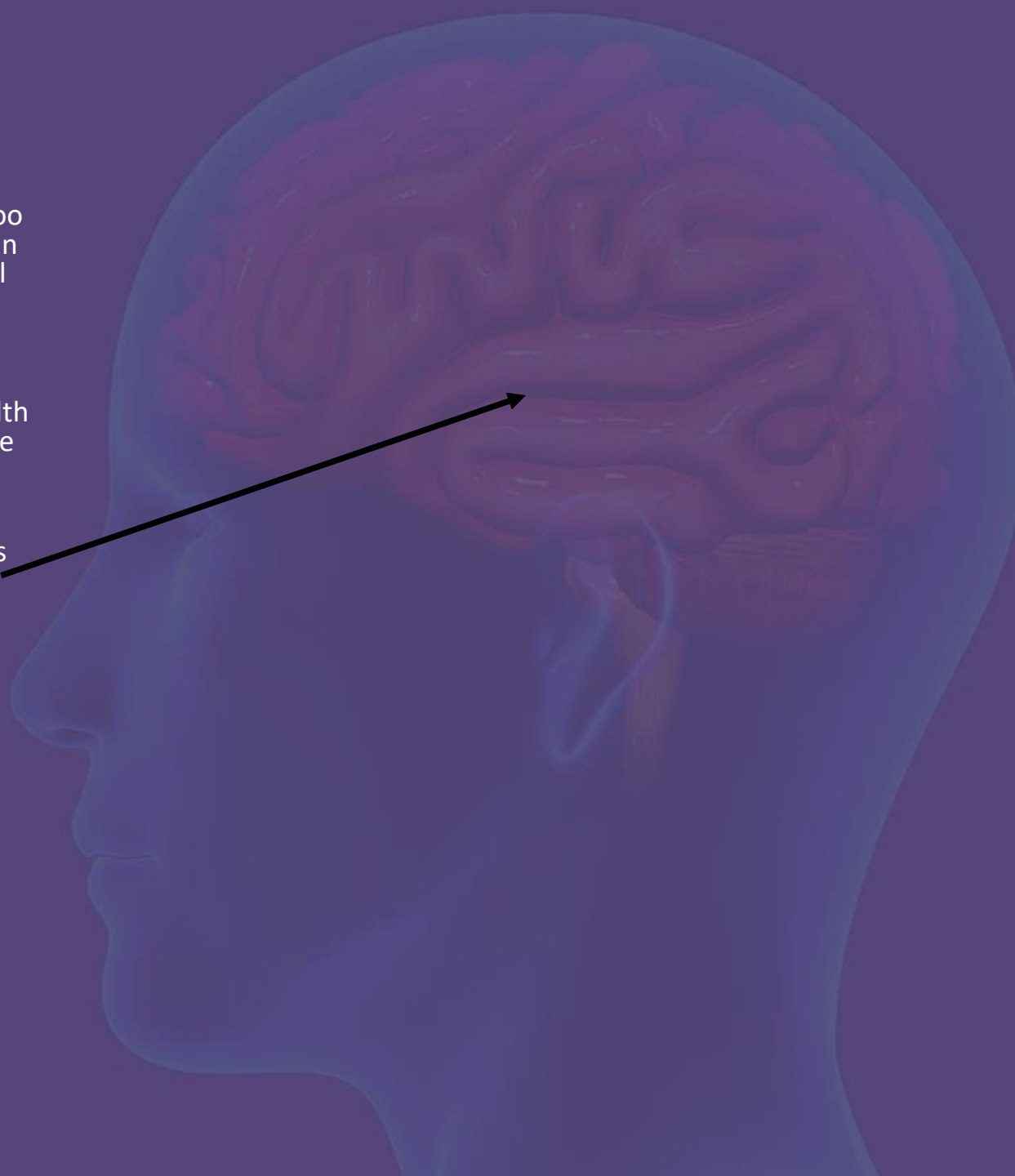
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Chlorine in Water

- Chlorine was introduced to public drinking water in 1908 in Chicago and was used to eliminate waterborne disease such as cholera and typhoid fever. Wide spread use started in 1914.
- In the 1970's it was discovered that chlorine when added to water forms trihalomethanes (chlorine by-products) by combining with certain naturally occurring organic matter, such as vegetation and algae.
- The National Cancer Institute estimates cancer risks for people who consume chlorinated water to be up to 93% higher than people who are not exposed to chlorinated water.

Fluoride

- Americans are consuming too much fluoride because it is in large quantities in municipal water, toothpaste, mouthwash, and other products.
- The U.S Department of Health plans to lower fluoride in the drinking water for the first time in 50 years.
- Studies show that fluoride is very damaging to the brain. Fluoride affects a section of the brain that regulates reactions to stressful circumstances, making human beings easier to control.
- The introduction of fluoride into the municipal water systems was first used by Hitler. He used it on concentration camp prisoners.



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Infections and Autoimmune Disease Links...

Coxsackievirus B (virus) – myocarditis

Streptococcus pyogenes (bacteria) – heart inflammation

Trypanosoma cruzi (parasite)- cardiomyopathy

Borrelia burgdorferi – Arthritis, myelitis

Escherichia coli – Uveitis

Campylobacter jejuni – Guillain-Barre Syndrome

Chlamydia pneumoniae – Multiple Sclerosis

Epstein-Barr

Klebsiella

Pseudomonas

Candida

Mycotoxins

Enterobacter

Clinical & Experimental Immunology
The Journal of Translational Immunology

doi:10.1111/j.1365-2249.2008.03834.x

Clinical and Experimental Immunology

British Society for immunology

STELLAR REVIEW

The role of infections in autoimmune disease

A. M. Ercolini and S. D. Miller
Department of Microbiology-Immunology and Interdepartmental Immunobiology Center, Northwestern University Feinberg School of Medicine, Chicago, IL, USA

Accepted for publication 27 October 2008
Correspondence: S. D. Miller, Department of Microbiology-Immunology, Northwestern University, Tarry 6-718, 303 E. Chicago Avenue, Chicago, IL 60611, USA.
E-mail: s-d-miller@northwestern.edu

Summary

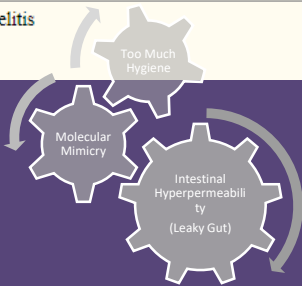
Autoimmunity occurs when the immune system recognizes and attacks host tissue. In addition to genetic factors, environmental triggers (in particular viruses, bacteria and other infectious pathogens) are thought to play a major role in the development of autoimmune diseases. In this review, we (i) describe the ways in which an infectious agent can initiate or exacerbate autoimmunity; (ii) discuss the evidence linking certain infectious agents to autoimmune diseases in humans; and (iii) describe the animal models used to study the link between infection and autoimmunity.

Keywords: autoimmune disease, molecular mimicry, virus infection

Table 1

Examples of Human Autoimmune Diseases with Possible Molecular Mimicry as a Mechanism

Human Diseases	Target	T Cells/Abs	Human Antigen Mimicked	Organism	Ref(s)
Spondyloarthropathies (SpAs), ankylosing spondylitis, psoriatic arthritis, reactive arthritis and undifferentiated SpA	Lumbar spine and sacroiliac joints	Abs	HLA-B27	<i>Klebsiella pneumoniae</i> , <i>Shigella</i> , <i>Chlamydia trachomatis</i> and other gram-negative bacteria	[71–73]
Antiphospholipid syndrome	Fetal loss and thromboembolic phenomena	Abs	β2-glycoprotein I	Bacteria, viruses, yeast, and tetanus toxin	[74]
Autoimmune chronic gastritis (AIG) (gastric atrophy, hypochloridria and pernicious anemia)	Stomach epithelium cells or parietal cell canaliculi	T cell/Abs	H ⁺ , K ⁺ -ATPase, parietal cell canaliculi	<i>Helicobacter pylori</i>	[75]
Cogan's syndrome	Eye and ear	Abs	SSA/Ro; (DEP-1/CD148); connexin 26	Reovirus III major core protein lambda 1	[76]
Autoimmune thrombocytopenic purpura	Platelet	Abs	Platelet; platelet-associated immuno-globulin G (PAIgG)	<i>Helicobacter pylori</i>	[77]
Behçet's disease	Eyes, skin, oral cavity, joints, genital system, CNS and blood vessels	T cell	HSP 60, HSP 65, HSP70, alpha-tropomyosin, S-antigens	Mycobacterial HSP, <i>Plasmodium falciparum</i>	[78–82]
Cardiomyopathy (myocarditis)	Heart	T cell/Abs	Cardiac myosin	Coxsackie virus, group A streptococci, chlamydia or <i>Trypanosoma cruzi</i>	[83]
Celiac sprue (celiac disease)	Small intestine	T cell	Transglutaminase	Gliadin (gluten), perinatal infections, adenovirus 12, hepatitis C virus (HCV)	[84, 85]
Chagas disease	Heart	T cell	Cardiac myosin	<i>Trypanosoma cruzi</i> B13 protein	[86, 87]
Chronic inflammatory demyelinating polyneuropathy	Schwann cells	Abs	Monosialoganglioside GM2	Melanoma, <i>Campylobacter jejuni</i>	[88, 89]
Crohn's disease	Gastrointestinal tract	T cell	Unknown	Gram-positive bacterial peptidoglycans	[90]
Dermatomyositis (juvenile)	Skin and muscle	T cell	Skeletal myosin	<i>Streptococcus pyogenes</i> M5 protein	[91]
Essential mixed cryoglobulinemia	B cell	Abs	IgG-Fc	HCV	[92]
Guillain-Barré syndrome	Gangliosides and peripheral nerve	Abs	Peripheral nerve	<i>Campylobacter jejuni</i>	[93]
Insulin dependent diabetes (type I)	Pancreas	T cell	Islet antigens (GAD 65, proinsulin carboxypeptidase H)	Coxsackie B virus, rubella, rotavirus, herpes, rhinovirus, hantavirus, flavivirus and retrovirus	[94–96], reviewed in [97], [98–100]
Systemic lupus erythematosus	Systemic	Abs	60 Kda Ro	Epstein-Barr virus (EBV nuclear antigen-1)	[101]
Multiple sclerosis	Myelin	T cell	Myelin basic protein	EBV, measles and HHV-6	[11, 35,102]
Primary biliary cirrhosis	Liver (intrahepatic bile ducts)	Abs/B and T cell	PDE2, GP210, human pyruvate dehydrogenase complex-E2 (PDC-E2), HLA-DR	Gram-negative bacterium, <i>Escherichia coli</i> , <i>Helicobacter pylori</i> , <i>Pseudomonas aeruginosa</i> , cytomegalovirus and <i>Haemophilus influenza</i>	[103–107]
Psoriasis	Skin	T cell	Epidermal keratins	<i>Streptococcus pyogenes</i> (streptococcal M protein)	[108]
Rheumatic fever	Heart	Abs/ T cell	Cardiac myosin	M protein (major virulence factor of group A streptococci) and streptococcus carbohydrate epitope GlcNAc	[12, 109–111]
Rasmussen's encephalitis	CNS	Abs	Antigliutamate receptor (GLUR3)	Microorganisms	[112, 113]
Acute disseminating encephalomyelitis	CNS	T cell	Myelin basic protein	Measles virus, rabies vaccine, HHV-6, coronavirus, influenza virus hemagglutinin, EBV, Semliki Forest virus	[114, 115], reviewed in [116]



Source: Clin Rev Allergy Immunol. 2012 February ; 42(1): 102–111.

Mycotoxins

[Int J Toxicol](#). 2014 May;33(3):175-186. Epub 2014 Mar 12.

Environmentally Relevant Level of Aflatoxin B1 Dysregulates Human Dendritic Cells Through Signaling on Key Toll-Like Receptors.

[Mohammadi A](#)^{1,2}, [Mehrzaad J](#)^{3,2}, [Mahmoudi M](#)⁴, [Schneider M](#)⁵.

+ Author information

Abstract

Aflatoxins (AFs) are highly hazardous fungal biometabolites usually present in feeds and foods. Aflatoxin B₁ (AFB₁) is the most toxic and a known carcinogen. Toll-like receptors (TLRs), highly expressed by myeloid dendritic cells (DC), are key innate immune-surveillance molecules. Toll-like receptors not only sense pathogen-associated molecular patterns but also contribute to infections and cancer. To assess AFB₁-TLR interactions on human myeloid DC, pure CD11c⁺ DC were generated from monocytes isolated from healthy individuals and then exposed to relevant level of AFB₁ for 2 hours. Both quantitative polymerase chain reaction and flow cytometric assays were used to quantify, respectively, expression of TLR2 and TLR4 at the messenger RNA (mRNA) and protein levels in these DC. Levels of interleukin (IL) 1 β , IL-6, and IL-10 were also analyzed in AFB₁- and mock-treated DC. Compared to nontreated CD11c⁺ DC, expression levels of both TLR2 and TLR4 mRNA and proteins were significantly upregulated in AFB₁-treated cells. Further, although IL-10 levels in AFB₁-treated DC were similar to those in the mock-treated DC, the AFB₁-exposed DC secreted higher amounts of IL-1 β and IL-6. Dendritic cells are sensitive to environmentally relevant level of AFB₁, and TLR2 and TLR4 are involved in sensing AFB₁. Considering the broad roles of TLR2, TLR4, and DC in immunity and infections, our novel findings open a new door to understanding the molecular mechanisms and functional consequences of AFB₁ in inducing immunodysregulation, immunotoxicity, and thus (non)infectious diseases in humans.

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Analysis

Medical error—the third leading cause of death in the US

BMJ 2016 ; 353 doi: <https://doi.org/10.1136/bmj.i2139> (Published 03 May 2016)

Cite this as: BMJ 2016;353:i2139

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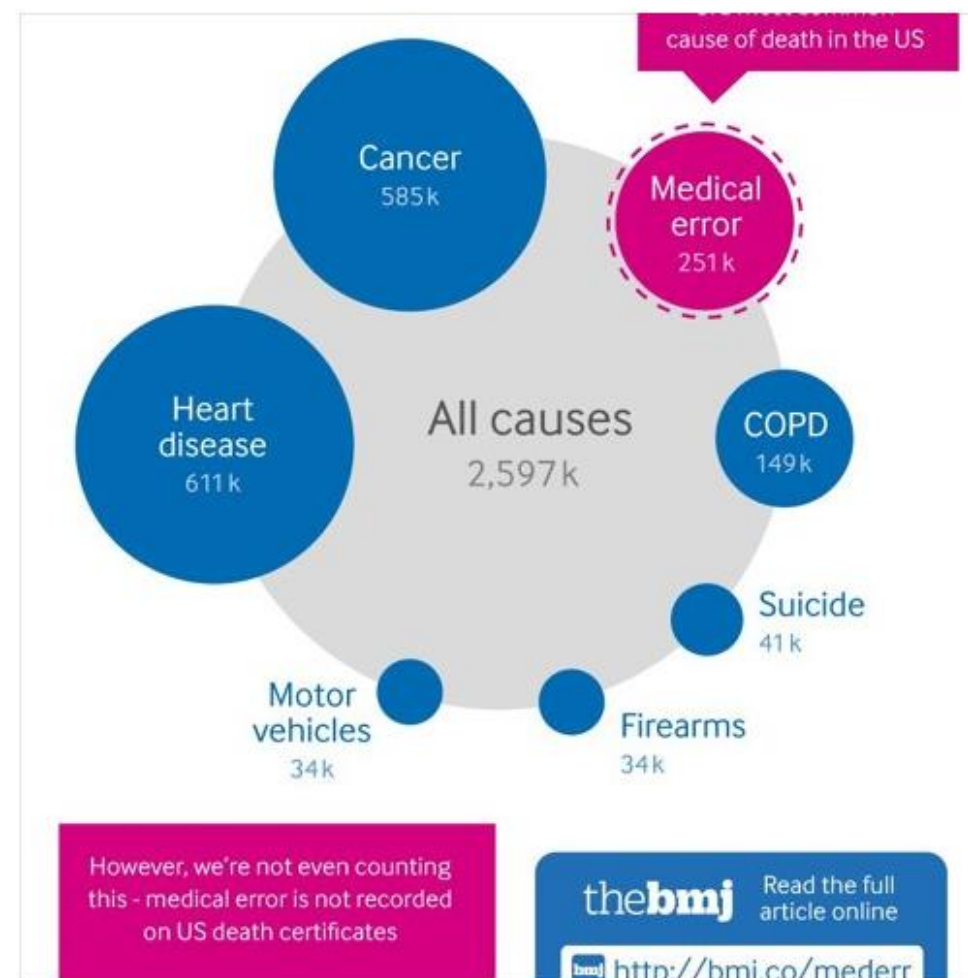
Martin A Makary, professor¹, Michael Daniel, research fellow¹

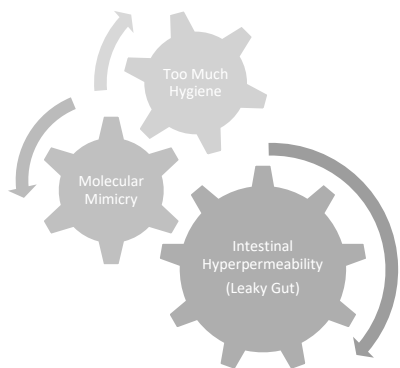
[Author affiliations ▾](#)

Correspondence to: M A Makary mmakary1@jhmi.edu

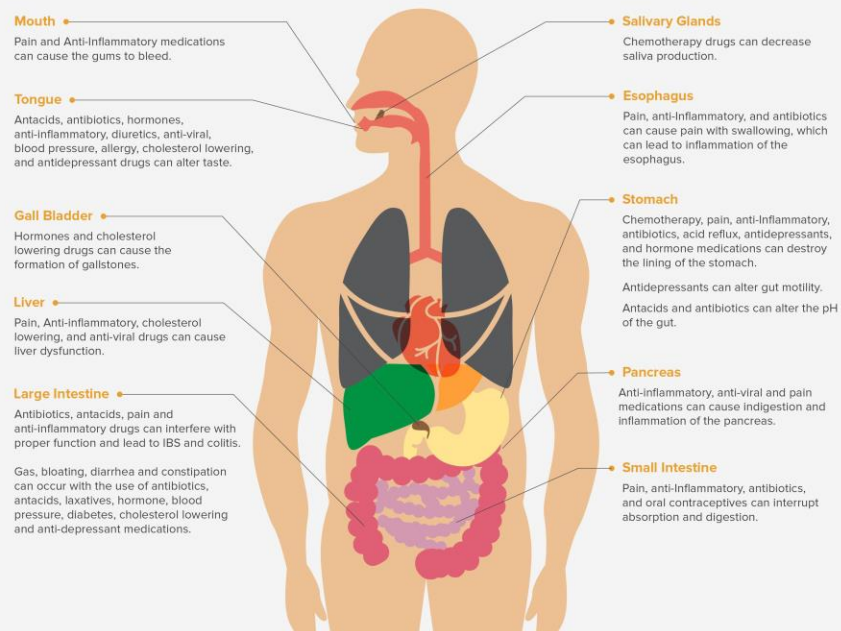
Medical error is not included on death certificates or in rankings of cause of death. **Martin Makary** and **Michael Daniel** assess its contribution to mortality and call for better reporting

Medical error—the third leading cause of death...





DRUGS THAT CAUSE GUT DYSFUNCTION



ASK YOUR DOCTOR IF YOUR MEDICATIONS WILL CAUSE THESE SIDE EFFECTS:



Alter Taste and Smell



Disrupt GI Motility Causing Constipation and Diarrhea



Cause Nutritional Deficiencies



Increase or Decrease Appetite

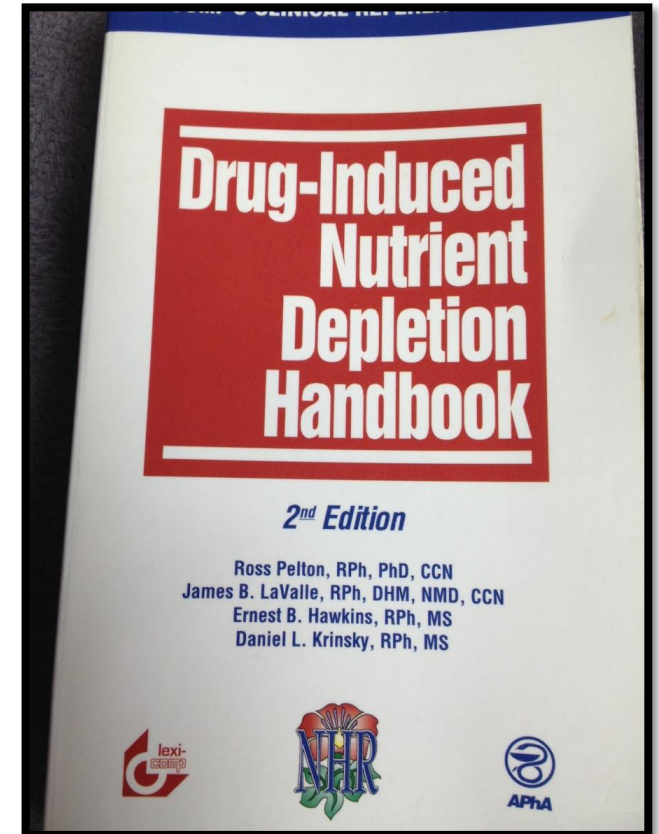
IF YES, TALK TO YOUR DOCTOR ABOUT NATURAL ALTERNATIVES.

References

1. CDC/National Center for Health Statistics. (2016). Leading cause of death. Retrieved from <https://www.cdc.gov/nchs/fastats/leading-causes-of-death.htm>
2. Hadjilabaie, et al. (2005). The Adverse Drug Reaction in the Gastrointestinal Tract: An Overview. International Journal of Pharmacology 1 (8), 1-8.
3. Norman, A. and Hawkey, C. (2011). Drug-induced gastrointestinal disorders. Medicine 29(3), 162-168.
4. Osborne, P. (2016). No Gain, No pain. Touchstone: New York, NY.
5. Richter, Joel E. (2007). Advances in Geriatric Gastroenterology & Hepatology. 3(4).
6. Tonbo, Linda G. (2002). Drug-induced Gastrointestinal Disorders. Medscape Pharmacotherapy. 4(1).

Or This...

Common medications prescribed for the treatment of autoimmune disease cause vitamin and mineral deficiencies that can hinder the immune system.



Damage caused by drugs that treat autoimmunity...

Int J Cancer. 2018 Sep 1;143(5):1062-1071. doi: 10.1002/ijc.31407. Epub 2018 Apr 16.

Tumor necrosis factor-alpha inhibitors and risk of non-Hodgkin lymphoma in a cohort of adults with rheumatologic conditions.


Calip GS^{1,2,3}, *Patel PR*⁴, *Adimadhyam S*¹, *Xing S*¹, *Wu Z*¹, *Sweiss K*⁵, *Schumock GT*^{1,2}, *Lee TA*^{1,2}, *Chiu BC*⁶.

⊕ Author information

Abstract

Based on limited evidence, the U.S. Food and Drug Administration (FDA) issued a black box warning for the use of tumor necrosis factor-alpha inhibitors (TNFIs) and risk of non-Hodgkin lymphoma (NHL). Our objective was to determine the risk of NHL associated with TNFI use by duration and type of anti-TNF agent. We performed a nested case-control study within a retrospective cohort of adults with rheumatologic conditions from a U.S. commercial health insurance database between 2009 and 2015. Use of TNFIs (infliximab, adalimumab, etanercept, golimumab and certolizumab pegol) and conventional-synthetic disease-modifying antirheumatic drugs (csDMARDs) was identified, and conditional logistic regression models were used to estimate adjusted odds ratios (OR) and 95% confidence intervals (CI) for risk of NHL. From a retrospective cohort of 55,446 adult patients, 101 NHL cases and 984 controls matched on age, gender and rheumatologic indication were included. Compared to controls, NHL cases had greater TNFI use (33% vs. 20%) but were similar in csDMARD use (70% vs. 71%). **TNFI ever-use was associated with nearly two-fold increased risk of NHL** (OR = 1.93; 95% CI: 1.16-3.20) with suggestion of increasing risk with duration (P-trend = 0.05). TNF fusion protein (etanercept) was associated with increased NHL risk (OR = 2.73; 95% CI: 1.40-5.33), whereas risk with anti-TNF monoclonal antibodies was not statistically significant (OR = 1.77; 95% CI: 0.87-3.58). In sensitivity analyses evaluating confounding by rheumatologic disease severity, channeling bias was not likely to account for our results. **Our findings support the FDA black box warning for NHL.** Continued surveillance and awareness of this rare but serious adverse outcome are warranted with new TNFIs and biosimilar products forthcoming.


Medicines can also cause autoimmune disease.



Journal of Advanced Research



Volume 7, Issue 5, September 2016, Pages 719-726


open access



Review

Aromatase inhibitors induced autoimmune disorders in patients with breast cancer: A review

George Zarkavelis ^a, Aristomenes Kollas ^a, Eleftherios Kampletsas ^a, Vasilis Vasiliou ^b, Evripides Kaltsonoudis ^c, Alexandros Drosos ^c, Hussein Khaled ^d, Nicholas Pavlidis ^a  

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<https://doi.org/10.1016/j.jare.2016.04.001>[Get rights and content](#)

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Abstract

Subacute cutaneous lupus erythematosus (SCLE) is characterized by particular cutaneous manifestations such as non-scarring plaques mainly in sunlight exposed parts of the body along with specific serum autoantibodies (i.e. antinuclear antibodies (ANA), Ro/SSa, La/SSb). It is considered either idiopathic or drug induced. The role of chemotherapeutic agents in causing SCLE has been investigated with the taxanes being the most common anticancer agents. However, recent data emerging point toward antiestrogen therapies as a causative factor not only for SCLE but also for a variety of autoimmune disorders. This is a report of a case of a 42 year old woman who developed clinical manifestations of SCLE after letrozole treatment in whom remission of the cutaneous manifestations was noticed upon discontinuation of the drug. In addition, an extensive review of the English literature has been performed regarding the association of antiestrogen therapy with autoimmune disorders. **In conclusion, Oncologists should be aware of the potential development of autoimmune reactions in breast cancer patients treated with aromatase inhibitors.**

Table 2: Drugs Implicated in the Development of Drug-Induced Lupus Erythematosus

Definite	Probable	Possible	Recent case reports

Lupus. 2014 May;23(6):545-53. doi: 10.1177/0961203314523871. Epub 2014 Feb 20.

Drug-induced lupus: Including anti-tumour necrosis factor and interferon induced.

Araújo-Fernández S¹, Ahijón-Lana M, Isenberg DA.

⊕ Author information

Abstract

Drug-induced lupus erythematosus is defined as a syndrome with clinical and serological features similar to systemic lupus erythematosus that is temporally related to continuous drug exposure and which resolves after discontinuation of this drug. More than 90 drugs, including biological modulators such as tumour necrosis factor- α inhibitors and interferons, have been identified as likely 'culprits'. While there are no standard diagnostic criteria for drug-induced lupus erythematosus, guidelines that can help to distinguish drug-induced lupus erythematosus from systemic lupus erythematosus have been proposed and several different patterns of drug-induced lupus erythematosus are emerging. Distinguishing drug-induced lupus erythematosus from systemic lupus erythematosus is important because the prognosis of drug-induced lupus erythematosus is usually good when the drug is withdrawn. This review discusses the differences between drug-induced lupus erythematosus and systemic lupus erythematosus, the mechanisms of action of drug-induced lupus erythematosus and drugs that are usually associated with drug-induced lupus erythematosus, with particular focus on the biological treatments.

	<ul style="list-style-type: none"> • Fluorouracil agents • Hydrochlorothiazide 		
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References:

1. Chang C, Gershwin ME. Drug-induced lupus erythematosus: incidence, management and prevention. *Drug Saf*. 2011;34:357-374.
2. Vasoo Sheila. Drug-induced lupus: an update. *Lupus*. 2006; 15:757-761.
3. Araújo-Fernández S, Ahijón-Lana M, Isenberg DA. Drug-induced lupus: Including anti-tumor necrosis factor and interferon induced. *Lupus*. 2014; 23:545-553.

[Intest Res.](#) 2017 Jul;15(3):419-421. doi: 10.5217/ir.2017.15.3.419. Epub 2017 Jun 12.

Telmisartan-induced sprue-like enteropathy: a case report and a review of patients using non-olmesartan angiotensin receptor blockers.

[Mandavdhare HS¹](#), [Sharma V¹](#), [Prasad KK¹](#), [Kumar A¹](#), [Rathi M²](#), [Rana SS¹](#).

⊕ Author information

Abstract

Recent studies have identified sprue-like illness associated with the use of the antihypertensive agent olmesartan medoxomil. However, whether this condition is specific to the use of olmesartan or is associated with the use of drugs belonging to the class of "sartans" remains to be clarified. A 45-year-old woman with chronic kidney disease along with hypothyroidism and hypertension presented with chronic diarrhea and significant weight loss. Endoscopy of the upper gastrointestinal tract showed scalloping and grooving of the duodenum, and histopathological examination showed subtotal villous atrophy. She was on telmisartan for hypertension, which was discontinued. Subsequently, diarrhea ameliorated dramatically, and she regained weight. To our knowledge, this is the first study to report **telmisartan-associated sprue-like enteropathy**. Further, we have reviewed the cases of patients with sprue-like enteropathy caused by valsartan, irbesartan, and eprosartan.

Surgery and Vaccine Induced Autoimmunity...

[Curr Opin Rheumatol](#). 2017 Jul;29(4):355-360. doi: 10.1097/BOR.0000000000000392.

Undifferentiated connective tissue disease, fibromyalgia and the environmental factors.

[Andreoli L](#)¹, [Tincani A](#).

⊕ Author information

Abstract

PURPOSE OF REVIEW: The aim of this study was to discuss the role of environmental factors in the induction and perpetuation of autoimmunity, with particular focus on **undifferentiated connective tissue disease (UCTD)** and fibromyalgia. These two entities may share undefined clinical and laboratory features and recognize environmental exposures as triggering factors. From this particular point of view, both UCTD and fibromyalgia may resemble the picture of the 'Autoimmune/Inflammatory Syndrome Induced by Adjuvants' (ASIA).

RECENT FINDINGS: A case-control study on environmental exposures showed that **patients with UCTD were significantly more exposed to several adjuvants (vaccines, metal implants, proximity to metal factories and foundries) than age and sex-matched healthy controls**. UCTD exposed to **major ASIA triggers (vaccines, silicone) displayed typical features of ASIA (general weakness, chronic fatigue, irritable bowel syndrome)** in the context of a predisposing genetic background (familiarity for autoimmunity).

SUMMARY: The induction and perpetuation of autoimmunity is a complex process that requires the interaction between the individual genetic background and the environment. Environmental factors are gaining increasing attention since the description of ASIA, a syndrome that includes symptoms typically seen in patients with fibromyalgia and UCTD. A recent case-control study focusing on environmental exposures suggested that nearly half of patients with UCTD may fall within the ASIA spectrum.

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Vaccines and AID

“Almost all types of vaccines have been reported to be associated with the onset of ASIA or Autoimmune/inflammatory Syndrome Induced by Adjuvants”

Prof. Yehyda Shoenfeld, MD, FRCP

Table 1. Association of Vaccines with Autoimmune Disease

Type of vaccine	Autoimmune disease	Reference
Influenza	GBS	Schonberger et al., 1979
Meningococcal (MCV4)	GBS	CDC, 2006
HBV	MS, SLE, RA	Geier et al., 2005
HPV	IDDM , IBD, vasculitis, SLE	Verstraeten et al., 2008; Sutton et al., 2009
MMR	ITP-like	Wraith et al., 2003
HAV, HBV, TT	Macrophagic myofasciitis	Gherardi et al., 2001

GBS, Guillain-Barré syndrome; SLE, systemic lupus erythematosus; MS, multiple sclerosis; ITP, idiopathic thrombocytopenic purpura; IDDM, insulin dependent diabetes mellitus; IBD, [inflammatory bowel disease](#); HAV, hepatitis A virus; TT, tetanus toxoid.

Source: Orbach, H, et al. Vaccines and autoimmune diseases of the adult
Discovery Medicine 9(45):90-7.

DOSES of VACCINES for U.S. CHILDREN from BIRTH-18 YEARS

1983

DTP (2 months)
OPV (2 months)
DTP (4 months)
OPV (4 months)
DTP (6 months)
MMR (15 months)
DTP (18 months)
OPV (18 months)
DTP (4 years)
OPV (4 years)
Td (15 years)

*1986:

Pharmaceutical manufacturers
producing vaccines were freed
from ALL liability resulting
from vaccine injury or death
by the Childhood Vaccine
Injury Act.

(SOURCE: www.cdc.gov)

DTP- Diphtheria, Tetanus, Pertussis (whole cell)
OPV- Oral Polio
MMR- Measles, Mumps, Rubella
Hep B- Hepatitis B
DTaP- Diphtheria, Tetanus, Pertussis (acellular)
HIB- Haemophilus influenzae Type B
PCV- Pneumococcal
IPV- Inactivated Polio
Varicella- Chicken Pox
Td- Tetanus, Diphtheria
Tdap- Tetanus, Diphtheria, and Pertussis
HPV- Human papillomavirus (Gardasil)

2016

Influenza (Pregnancy)
DTaP (Pregnancy)
Hep B (birth)
Hep B (2 months)
Rotavirus (2 months)
DTaP (2 months)
HIB (2 months)
PCV (2 months)
IPV (2 months)
Rotavirus (4 months)
DTaP (4 months)
HIB (4 months)
PCV (4 months)
IPV (4 months)
Hep B (6 months)
Rotavirus (6 months)
DTaP (6 months)
HIB (6 months)
PCV (6 months)
IPV (6 months)
Influenza (6 months)
HIB (12 months)
PCV (12 months)
MMR (12 months)
Varicella (12 months)
Hep A (12 months)
DTaP (18 months)
Influenza (18 months)
Hep A (18 months)
Influenza (30 months)
Influenza (42 months)
DTaP (4 years)
IPV (4 years)
MMR (4 years)
Varicella (4 years)
Influenza (5 years)
Influenza (6 years)
Influenza (7 years)
Influenza (8 years)
Influenza (9 years)
HPV (9 years)
Influenza (10 years)
HPV (10 years)
Influenza (11 years)
HPV (11 years)
Tdap (12 years)
Influenza (12 years)
Meningococcal (12 yrs)
Influenza (13 years)
Influenza (14 years)
Influenza (15 years)
Influenza (16 years)
Meningococcal (16 yrs)
Influenza (17 years)
Influenza (18 years)

2016

TOTAL DOSES: 69

Injections: 50

(3 Doses of Rotavirus are liquid)

1983

TOTAL DOSES: 24

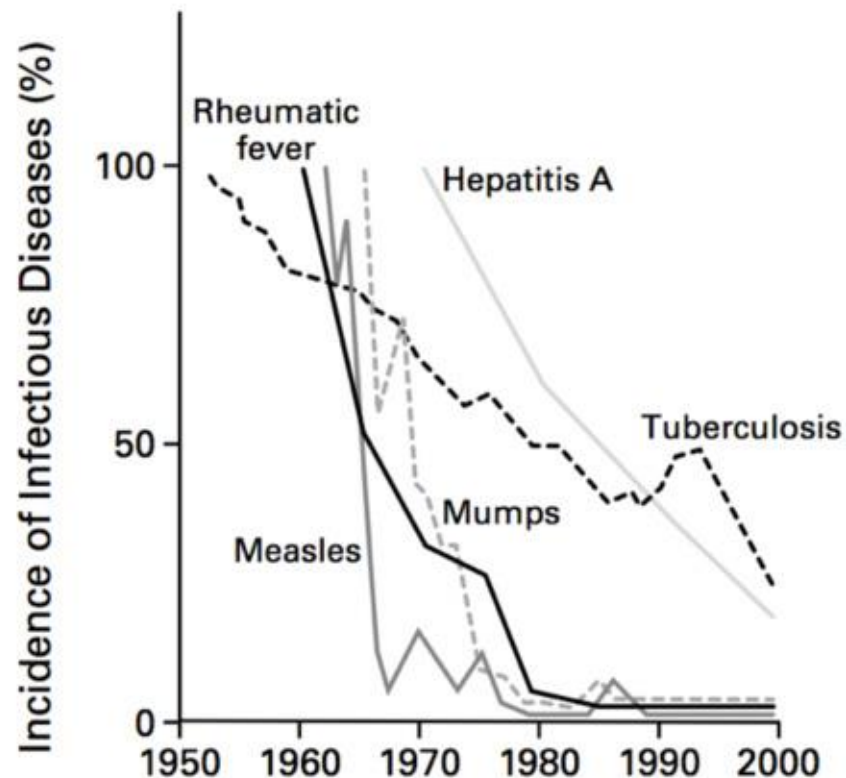
Injections: 7

(4 Doses of Polio were liquid)

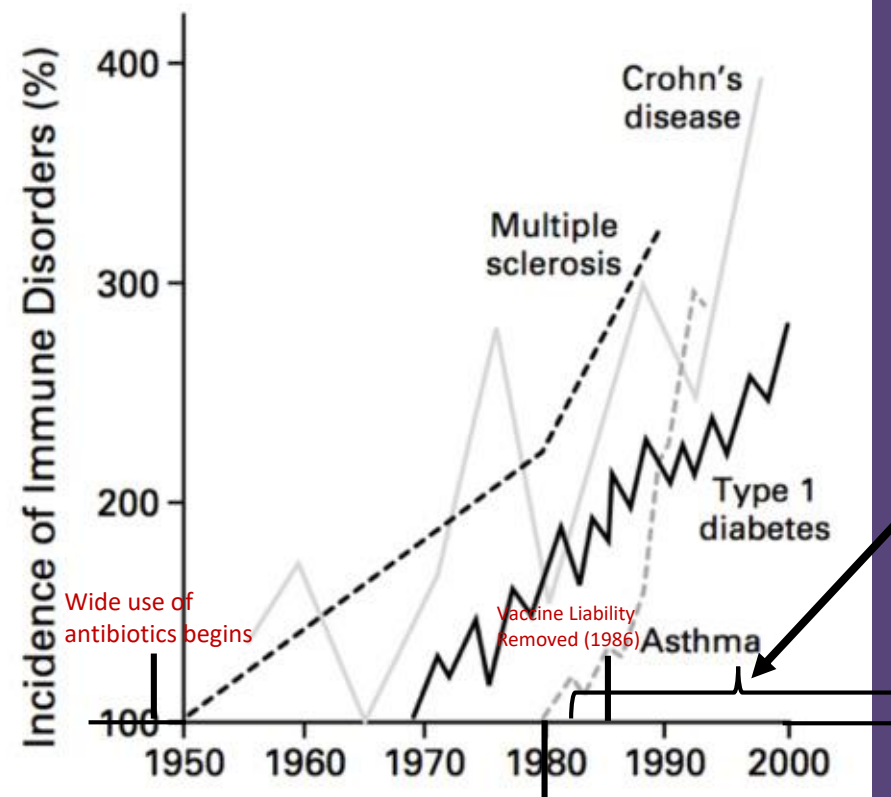
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A



B



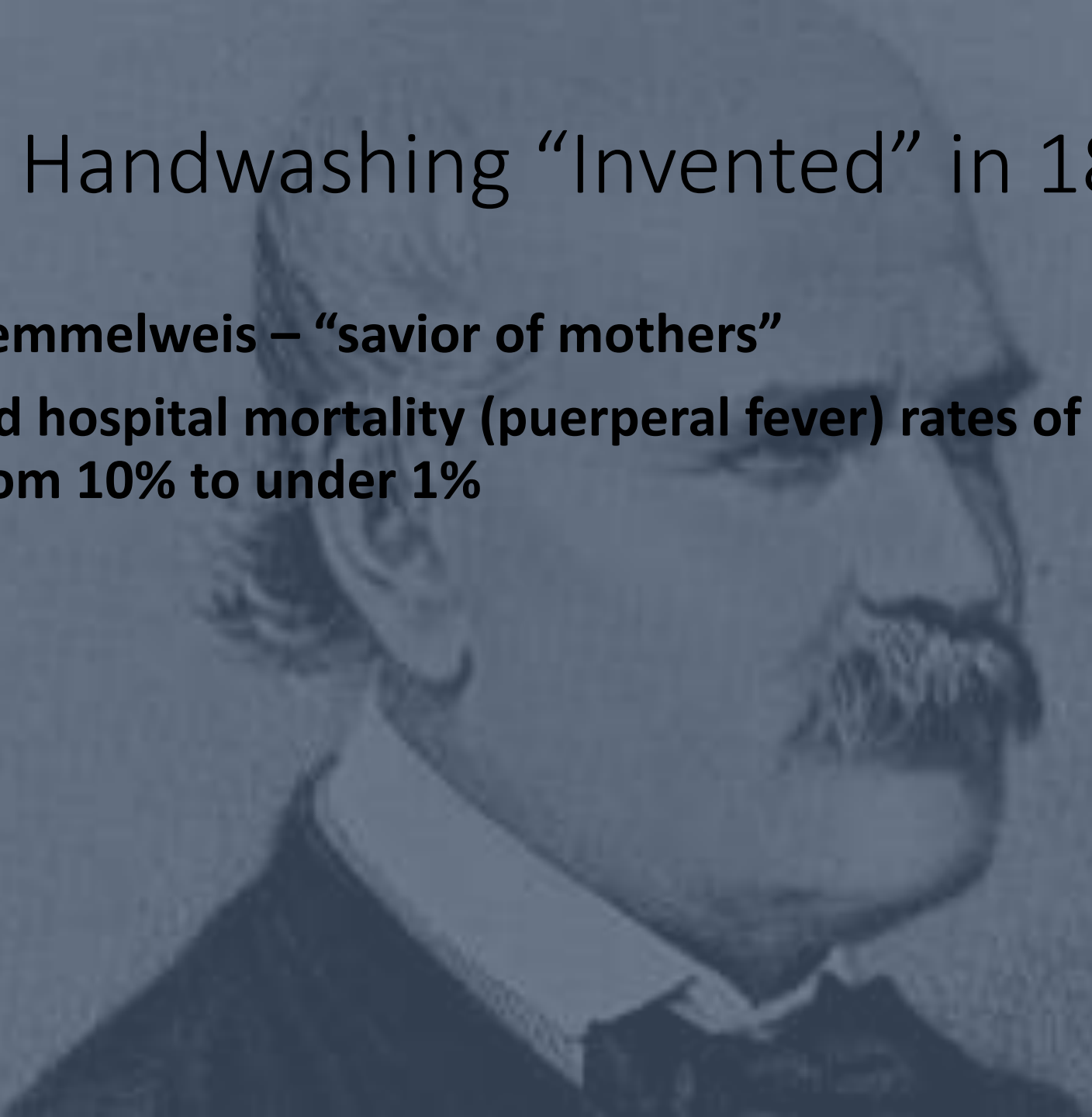
Cesarean delivery increases from 20 to 33% by 2011
While vaccine schedule increases 7 fold from 1983 to 2016.

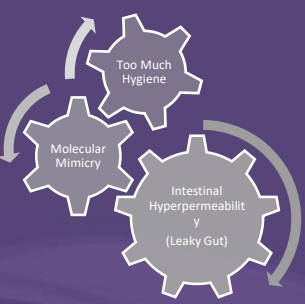
Chemical use in US reaches more than 35,000 (1980)

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Handwashing “Invented” in 1847

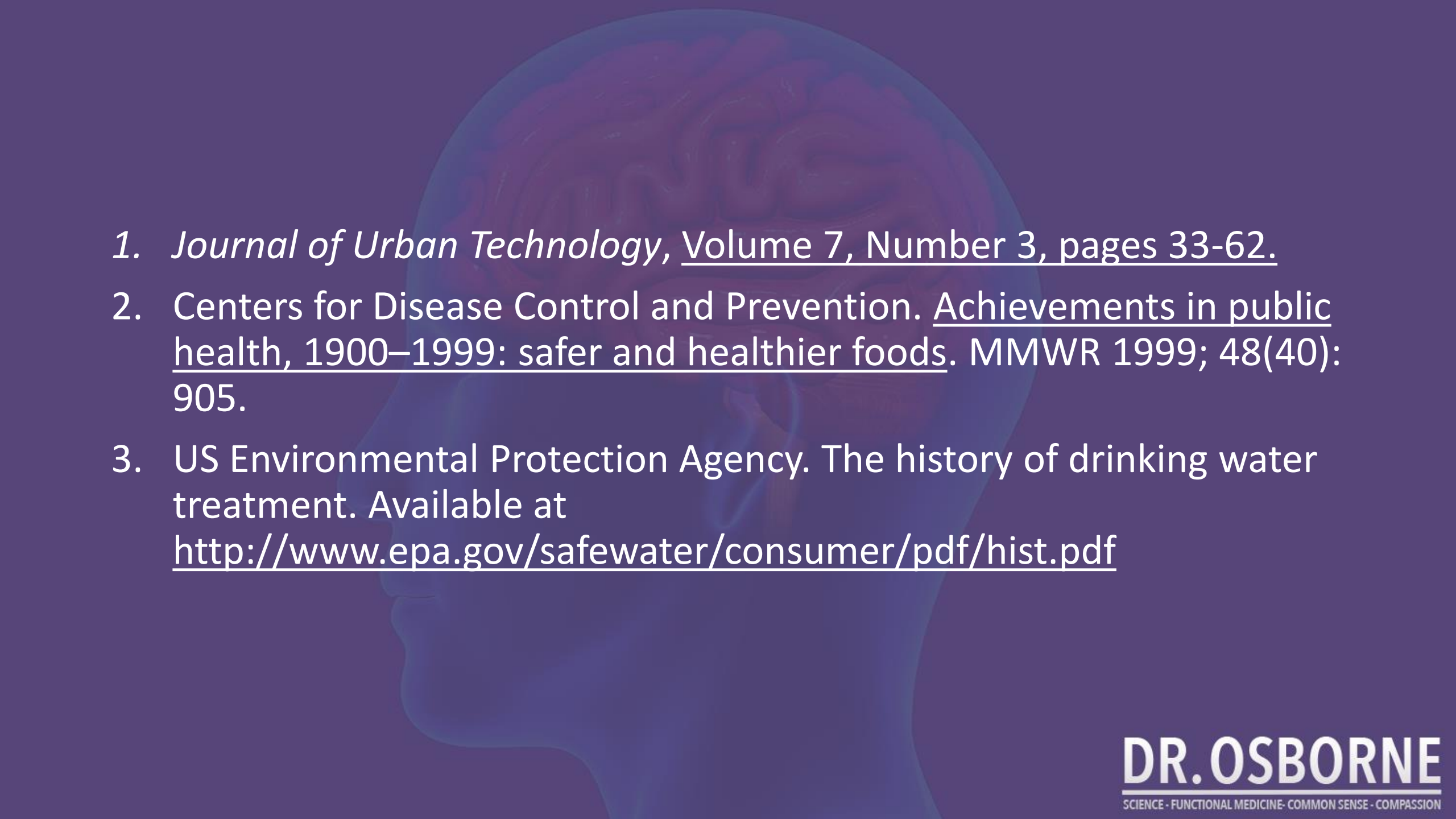
- **Ignaz Semmelweis – “savior of mothers”**
- **Reduced hospital mortality (puerperal fever) rates of mothers giving birth from 10% to under 1%**



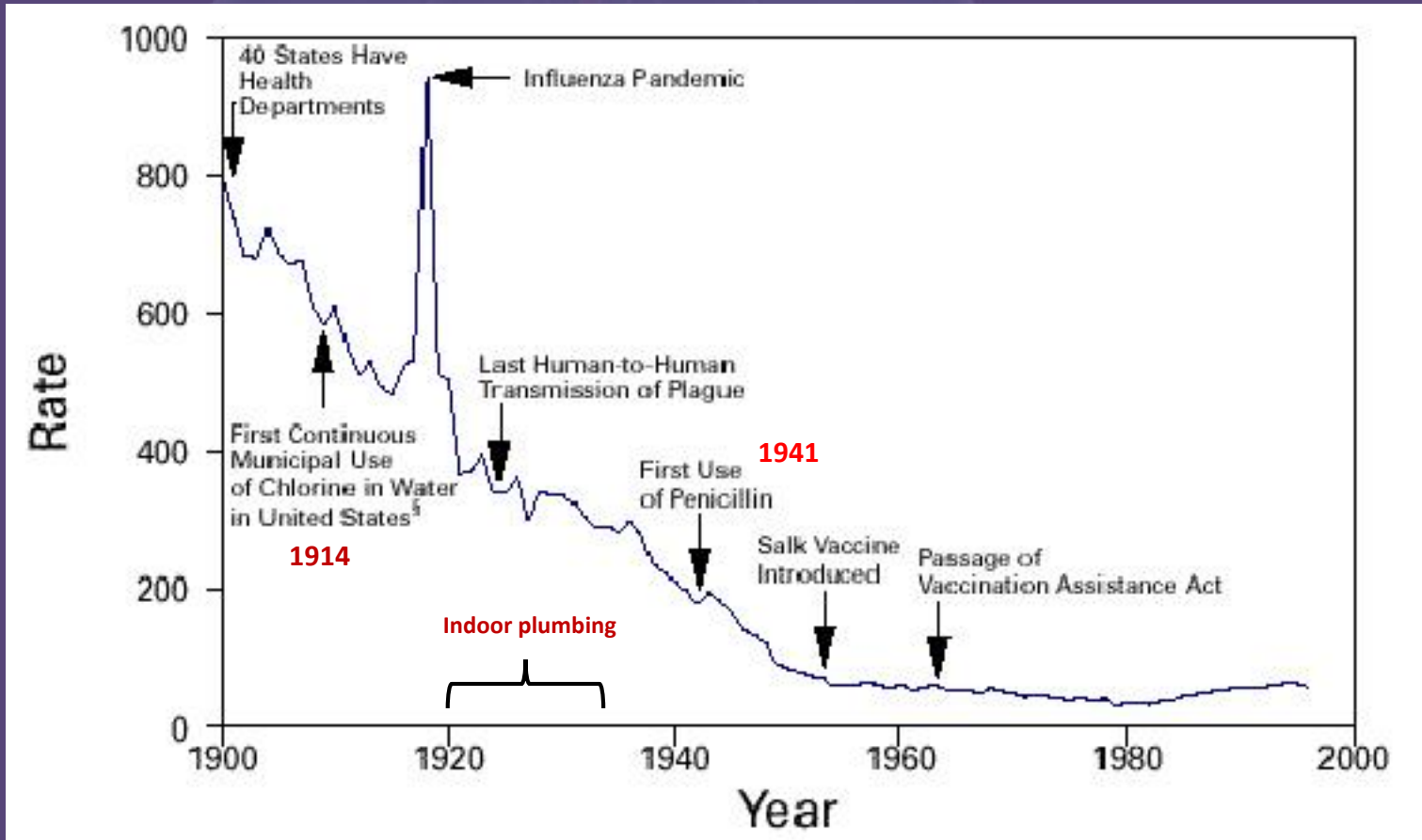


A Brief History on Water Management

- Pre 1820 water and waste water were managed by private privy and cesspools.
- 1820 – Less than 5% of US population lived in urban areas (cities with more than 8,000)
- 1820-1880 – Population boom along with urban development led to infrastructure issues around hygiene, clean water, sewage, living in close quarters
- 1854 – Physician John Snow discovers water contamination leads to infectious disease transmission.
- 1854 – Cities slowly began to build water treatment facilities
- 1885 – Louis Pasteur proves the Germ Theory of Disease
- 1914 - The U.S. Department of the Treasury enacted a set of standards, effectively requiring drinking water disinfection and leading to a dramatic increase in the use of drinking water chlorination by treatment plants.
- 1920's – New home construction in US implements standard indoor plumbing with bathrooms
- 1943 – Use of Antibiotics begins on large scale during WW2
- 1948 – Federal Water Pollution Control Act of 1948
- 1951 – US Public Health Service adopts fluoridation of public water policy
- 1970 – EPA was formed

- 
1. *Journal of Urban Technology*, Volume 7, Number 3, pages 33-62.
 2. Centers for Disease Control and Prevention. Achievements in public health, 1900–1999: safer and healthier foods. MMWR 1999; 48(40): 905.
 3. US Environmental Protection Agency. The history of drinking water treatment. Available at <http://www.epa.gov/safewater/consumer/pdf/hist.pdf>

Rate of Infections Diseases



Source: [CDC.gov](https://www.cdc.gov)

Vaccines & Antibiotics VS Sanitation...

“In conclusion, the largest historical decrease in morbidity and mortality caused by infectious disease was experienced not with the modern antibiotic and vaccine era, but after the introduction of clean water and effective sewer systems.”

The Journal of Pediatrics, December 1999, Vol. 135, No. 6, p. 663

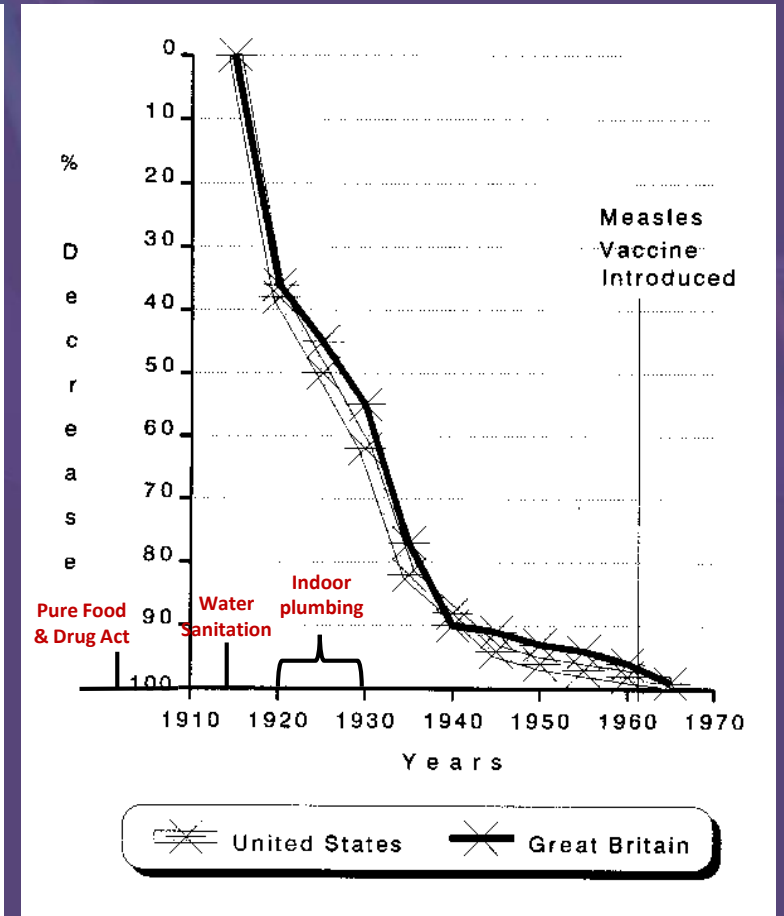
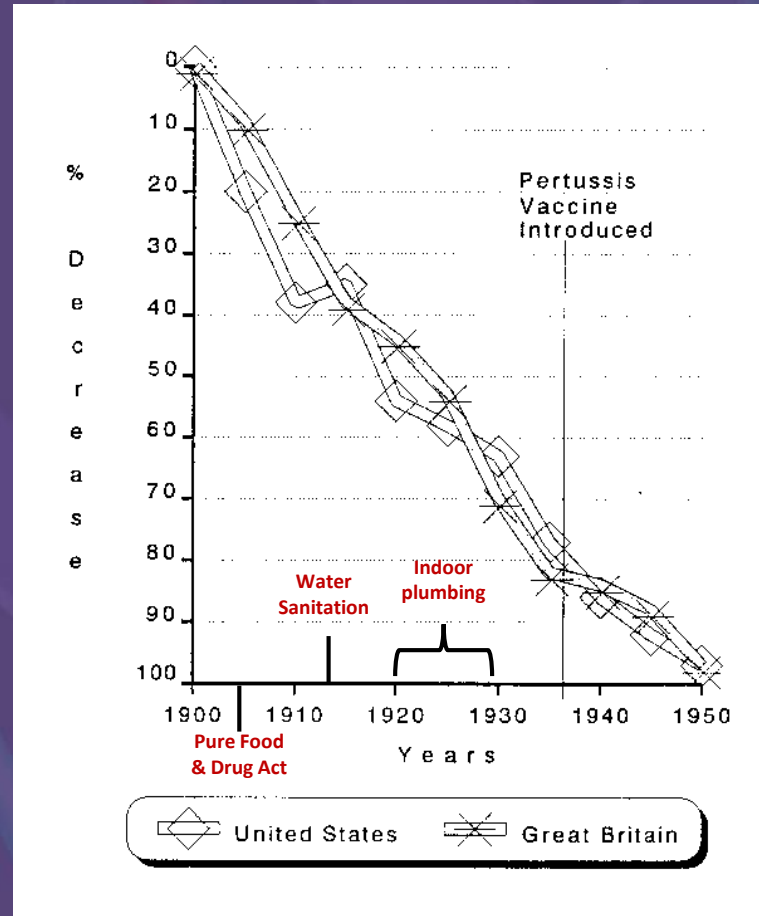
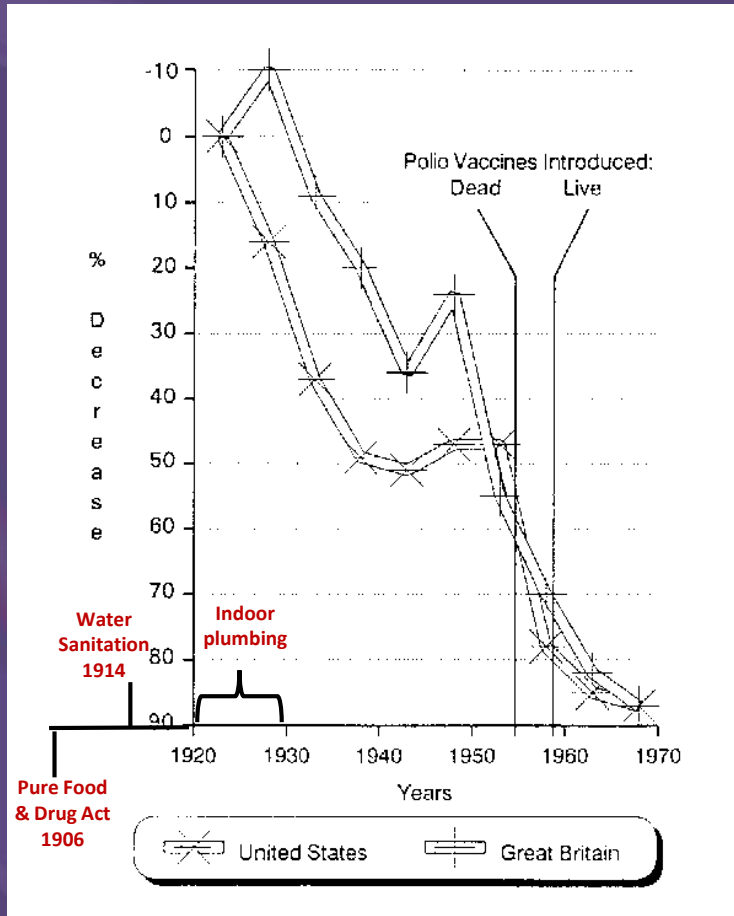
Michael Hambidge, MD

Nancy Krebs, MD

Section of Nutrition, Department of Pediatrics

DR. OSBORNE

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Source: Journal of American Physicians & Surgeons

Food & Nutrition

Allergens Nutrients Too much of too little

Chemicals in Food

Pesticides, herbicides
Food dyes Preservatives
Flavoring agents

Infections

Bacteria Fungus, mold, yeast
Virus Parasite

Lack of Sunshine

Vitamin D deficiency
Melatonin Deficiency

Excessive Stress

Relationships Work
Life purpose
Time management

Air Quality

Smoking Indoor pollutants
Outdoor pollutants EMF Air “fresheners”

Water Quality

Fluoride
Chlorine & chloramine Medications

Medical Interventions

Vaccines Amalgams
Medications Surgical implants

Lack of Sleep

Artificial light sources
Caffeine over utilization

Lack of Physical Activity

Sedentary lifestyle
Convenience

**There is no magic bullet.
AID is multifactorial.**

DR. OSBORNE

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Sunshine...

Int J Mol Sci. 2013 Jun; 14(6): 11742–11766.

Published online 2013 May 31. doi: [10.3390/ijms140611742](https://doi.org/10.3390/ijms140611742)

PMCID: PMC3709754

PMID: [23727938](https://pubmed.ncbi.nlm.nih.gov/23727938/)

Modulation by Melatonin of the Pathogenesis of Inflammatory Autoimmune Diseases

[Gu-Jiun Lin](#),¹ [Shing-Hwa Huang](#),² [Shyi-Jou Chen](#),^{3,4} [Chih-Hung Wang](#),^{3,5,6,7} [Deh-Ming Chang](#),⁸ and [Huey-Kang Sytwu](#)^{7,*}

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This article has been [cited by](#) other articles in PMC.

Abstract

Go to: ☒

Melatonin is the major secretory product of the pineal gland during the night and has multiple activities including the regulation of circadian and seasonal rhythms, and antioxidant and anti-inflammatory effects. It also possesses the ability to modulate immune responses by regulation of the T helper 1/2 balance and cytokine production. Autoimmune diseases, which result from the activation of immune cells by autoantigens released from normal tissues, affect around 5% of the population. Activation of autoantigen-specific immune cells leads to subsequent damage of target tissues by these activated cells. Melatonin therapy has been investigated in several animal models of autoimmune disease, where it has a beneficial effect in a number of models excepting rheumatoid arthritis, and has been evaluated in clinical autoimmune diseases including rheumatoid arthritis and ulcerative colitis. This review summarizes and highlights the role and the modulatory effects of melatonin in several inflammatory autoimmune diseases including multiple sclerosis, systemic lupus erythematosus, rheumatoid arthritis, type 1 diabetes mellitus, and inflammatory bowel disease.

gene regulatory network. The bi-stable switch would enable T cells to integrate signals from pathogens, hormones, cell–cell interactions, and soluble mediators and respond in a biologically appropriate manner. Finally, unanswered questions and potentially informative future research directions are highlighted to speed delivery of etiology-based strategies to reduce autoimmune disease.

Food & Nutrition

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AID is multifactorial.**

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Sleep...

[Best Pract Res Clin Endocrinol Metab. 2010 Oct; 24\(5\): 775–784.](#)

PMID: [21112025](#)

[J Immunol Res. 2016;2016:4576012. Epub 2016 Sep 21.](#)

Blood-Brain Barrier Disruption Induced by Chronic Sleep Loss: Low-Grade Inflammation May Be the Link.

[Hurtado-Alvarado G¹](#), [Domínguez-Salazar E¹](#), [Pavon L²](#), [Velázquez-Moctezuma J¹](#), [Gómez-González B¹](#).

⊕ Author information

Abstract

Sleep is a vital phenomenon related to immunomodulation at the central and peripheral level. Sleep deficient in duration and/or quality is a common problem in the modern society and is considered a risk factor to develop neurodegenerative diseases. Sleep loss in rodents induces blood-brain barrier disruption and the underlying mechanism is still unknown. Several reports indicate that sleep loss induces a systemic low-grade inflammation characterized by the release of several molecules, such as cytokines, chemokines, and acute-phase proteins; all of them may promote changes in cellular components of the blood-brain barrier, particularly on brain endothelial cells. In the present review we discuss the role of inflammatory mediators that increase during sleep loss and their association with general disturbances in peripheral endothelium and epithelium and how those inflammatory mediators may alter the blood-brain barrier. Finally, this manuscript proposes a hypothetical mechanism by which sleep loss may induce blood-brain barrier disruption, emphasizing the regulatory effect of inflammatory molecules on tight junction proteins.

changes play key roles.

DR. OSBORNE

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Convenience

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DR. OSBORNE

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Chronic Stress

[Autoimmun Rev.](#) 2008 Jan;7(3):209-13. doi: 10.1016/j.autrev.2007.11.007. Epub 2007 Nov 29.

Stress as a trigger of autoimmune disease.

[Stojanovich L¹](#), [Marisavljevic D.](#)

⊕ Author information

Abstract

The etiology of autoimmune diseases is multifactorial: genetic, environmental, hormonal, and immunological factors are all considered important in their development. Nevertheless, the onset of at least 50% of autoimmune disorders has been attributed to "unknown trigger factors". Physical and psychological stress has been implicated in the development of autoimmune disease, since numerous animal and human studies demonstrated the effect of sundry stressors on immune function. Moreover, many retrospective studies found that a high proportion (up to 80%) of patients reported uncommon emotional stress before disease onset. Unfortunately, not only does stress cause disease, but the disease itself also causes significant stress in the patients, creating a vicious cycle. Recent reviews discuss the possible role of psychological stress, and of the major stress-related hormones, in the pathogenesis of autoimmune disease. It is presumed that the stress-triggered neuroendocrine hormones lead to immune dysregulation, which ultimately results in autoimmune disease, by altering or amplifying cytokine production. The treatment of autoimmune disease should thus include stress management and behavioral intervention to prevent stress-related immune imbalance. Different stress reactions should be discussed with autoimmune patients, and obligatory questionnaires about trigger factors should include psychological stress in addition to infection, trauma, and other common triggers.

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SCIENCE - FUNCTIONAL MEDICINE - COMMON SENSE - COMPASSION

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AID is multifactorial.**

DR. OSBORNE

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Physical Activity...

[Autoimmun Rev.](#) 2014 Sep;13(9):981-1000. doi: 10.1016/j.autrev.2014.07.001. Epub 2014 Aug 2.

Obesity in autoimmune diseases: not a passive bystander.

[Versini M](#)¹, [Jeandel PY](#)², [Rosenthal E](#)², [Shoenfeld Y](#)³.

⊕ Author information

Abstract

In the last decades, autoimmune diseases have experienced a dramatic increase in Western countries. The involvement of environmental factors is strongly suspected to explain this rise. Particularly, over the same period, obesity has followed the same outbreak. Since the exciting discovery of the secretory properties of adipose tissue, the relationship between obesity and autoimmunity and the understanding of the underlying mechanisms have become of major interest. Indeed, the fat tissue has been found to produce a wide variety of "adipokines", involved in the regulation of numerous physiological functions, including the immune response. By conducting a systematic literature review, we extracted 329 articles regarding clinical, experimental and pathophysiological data on the relationship between obesity, adipokines - namely leptin, adiponectin, resistin, visfatin - and various immune-mediated conditions, including rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), inflammatory bowel disease (IBD), multiple sclerosis (MS), type-1 diabetes (T1D), psoriasis and psoriatic arthritis (PsA), and thyroid autoimmunity (TAI), especially Hashimoto thyroiditis (HT). The strongest levels of evidence support an increased risk of RA (OR=1.2-3.4), MS (OR=2), psoriasis and PsA (OR=1.48-6.46) in obese subjects. A higher risk of IBD, T1D and TAI is also suggested. Moreover, **obesity worsens the course of RA, SLE, IBD, psoriasis and PsA, and impairs the treatment response of RA, IBD, psoriasis and PsA.** Extensive clinical data and experimental models demonstrate the involvement of adipokines in the pathogenesis of these autoimmune diseases. **Obesity appears to be a major environmental factor contributing to the onset and progression of autoimmune diseases.**

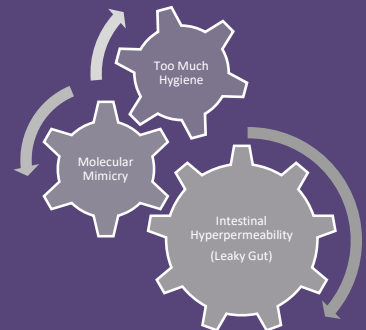
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In Summary...

- Population boom along with urban development led to infrastructure issues around hygiene, clean water, sewage, and crowded living spaces.
- This spawned an industrial revolution that led to new thoughts and technologies in chemistry and materials science, ushering in a new age of convenience and mass consumption.
- During the same time frame, a war against germs and acute infections raged on, leading to germaphobia, overuse of chemical cleaning agents, coupled with an overuse of antibiotics in our food supply as well as through prescription medications.

Our immune systems are thoroughly confused because we eat food that is not food, avoid sunshine, dismiss sleep, avoid intimacy for technology, live sedentary lives in hyper hygienic environments that are bug free, but chemically abundant, and use medicines to artificially manipulate how we feel, because the TRUTH is too painful to accept...and the thought of changing our behavior creates an illogical fear that subliminally gives us an excuse not to change.



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What are you going to do now?

- Genes do not make you sick...You can no longer use this for an excuse.
- Subjecting your genes to the wrong environment makes you sick. Change your choices, change your life.
- You will always have the capacity to:
 - Educate yourself
 - Take action on what you learn
 - ...and by doing so alter the outcome of your health and life.

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Food & Nutrition

Allergens Nutrients Too much of too little

Chemicals in Food

Pesticides, herbicides
Food dyes Preservatives
Flavoring agents

Infections

Bacteria Fungus, mold, yeast
Virus Parasite

Lack of Sunshine

Vitamin D deficiency
Melatonin Deficiency

Excessive Stress

Relationships Work
Life purpose
Time management

Air Quality

Smoking Indoor pollutants
Outdoor pollutants EMF Air “fresheners”

Water Quality

Fluoride
Chlorine & chloramine Medications

Medical Interventions

Vaccines Amalgams
Medications Surgical implants

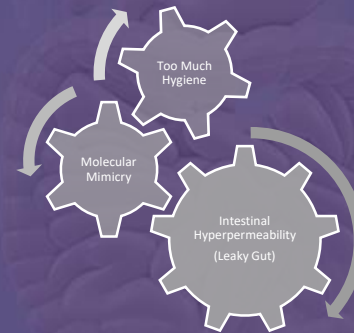
Lack of Sleep

Artificial light sources
Caffeine over utilization

Lack of Physical Activity

Sedentary lifestyle
Convenience

Get to work...



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I Love You.

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RESOURCES: Click the links below

[Pick Dr. Osborne's Brain Health Archive](#)

[Glutenology Health Matrix](#) – Yes, you need to learn more about gluten.

[Genetic Testing for Gluten Sensitivity](#)

[Air Filtration](#)

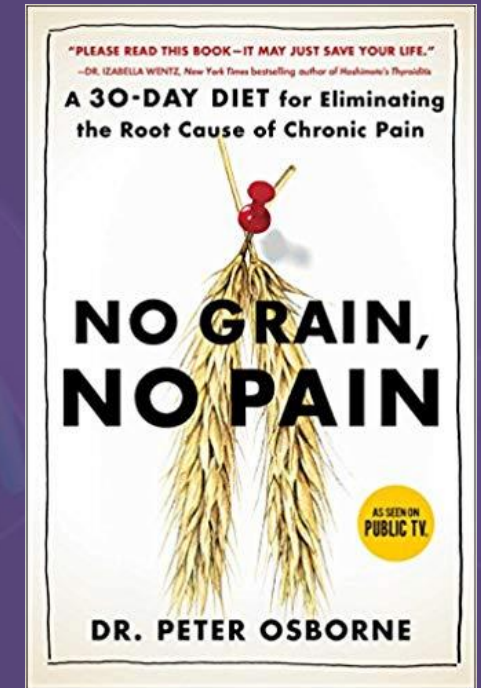
[Water Filtration](#)

[High Quality Food Sources](#)

[High Quality Supplements](#)

[The Autoimmune Revolution](#)

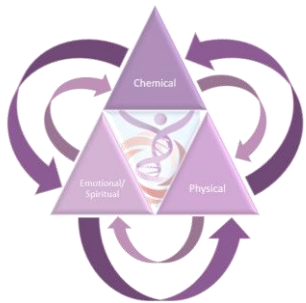
Autoimmune Matrix – A deeper dive into overcoming chronic autoimmune struggles



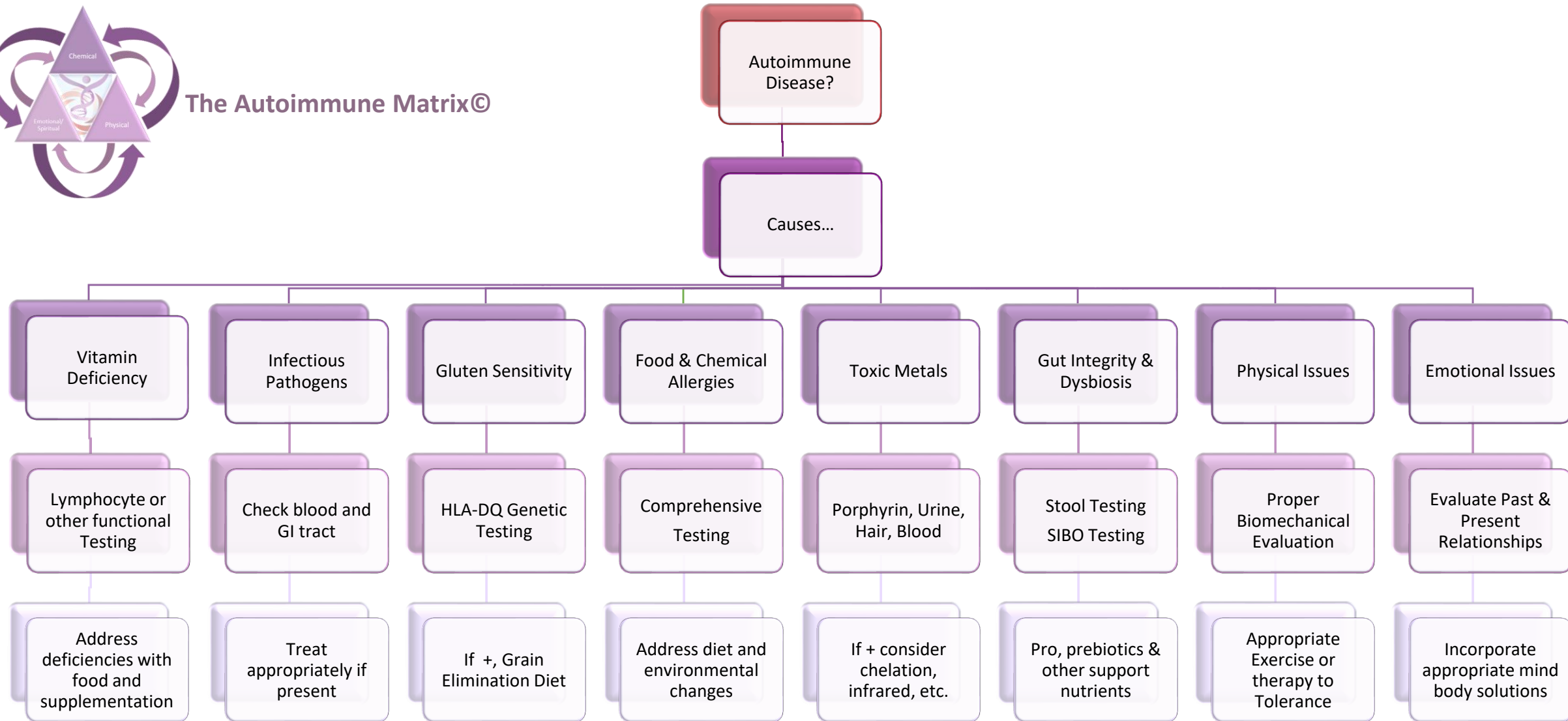


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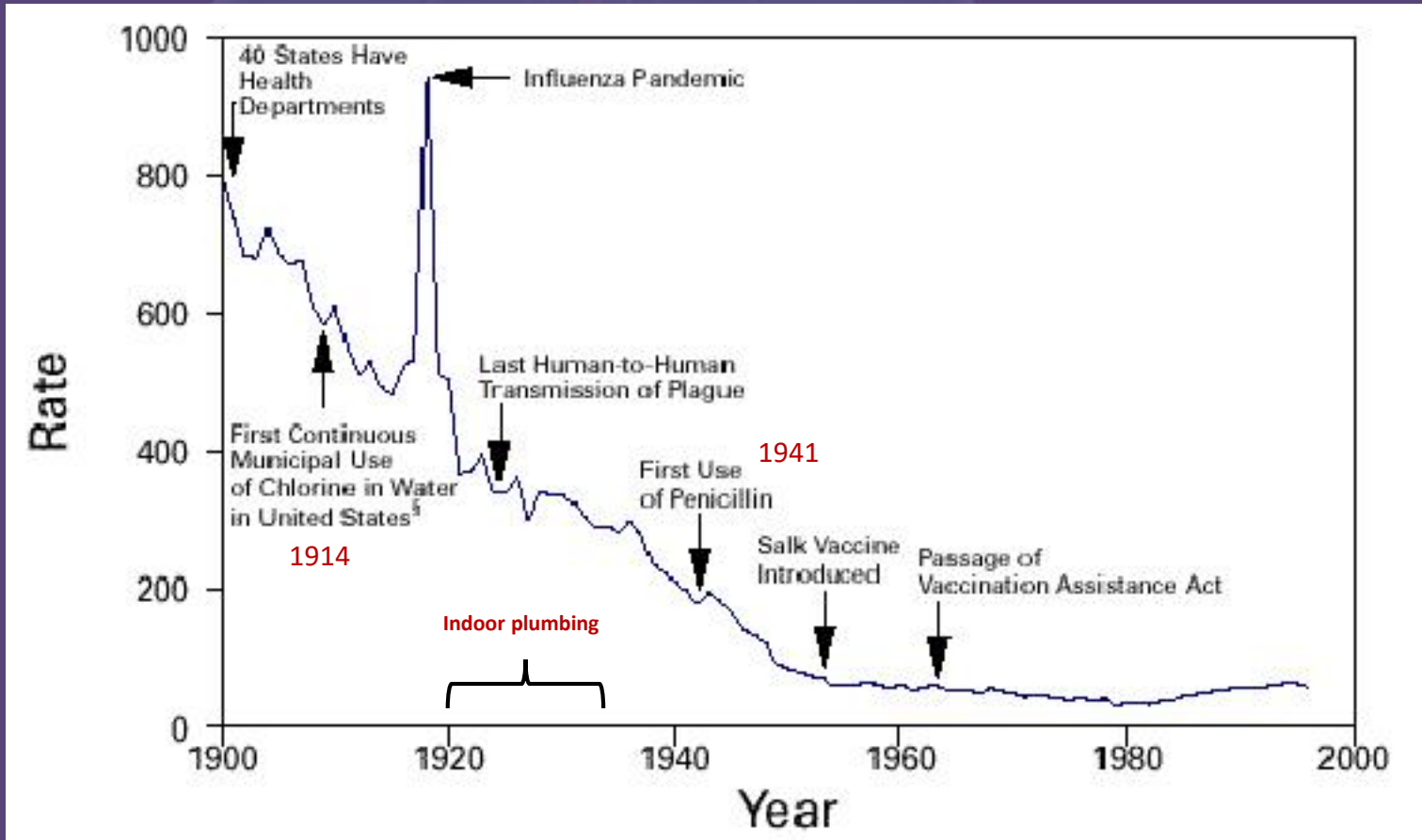
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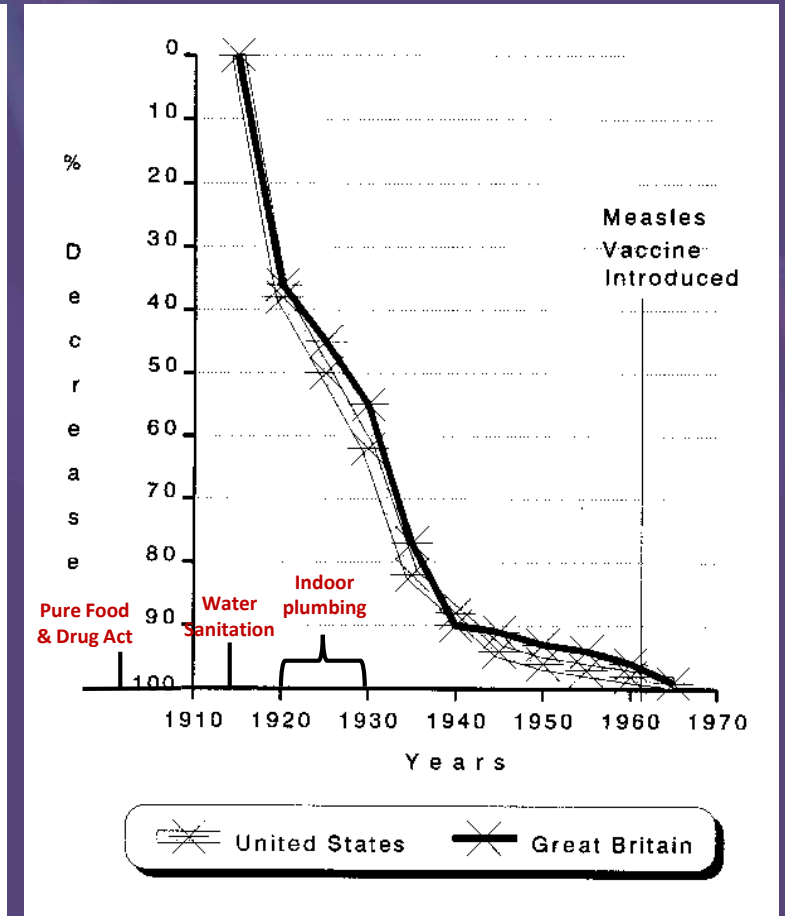
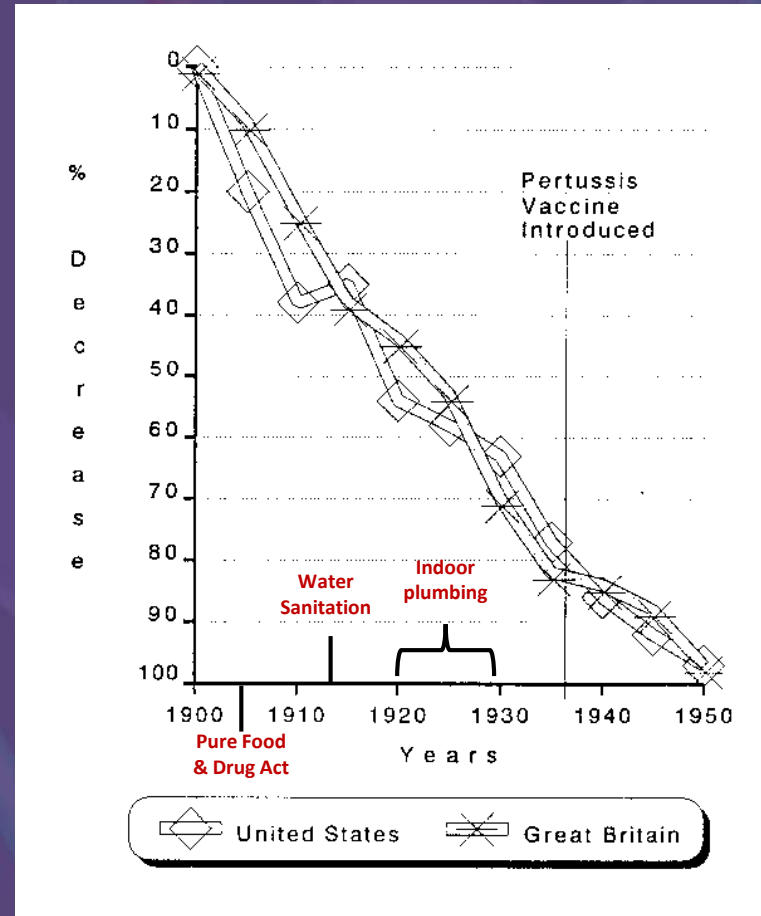
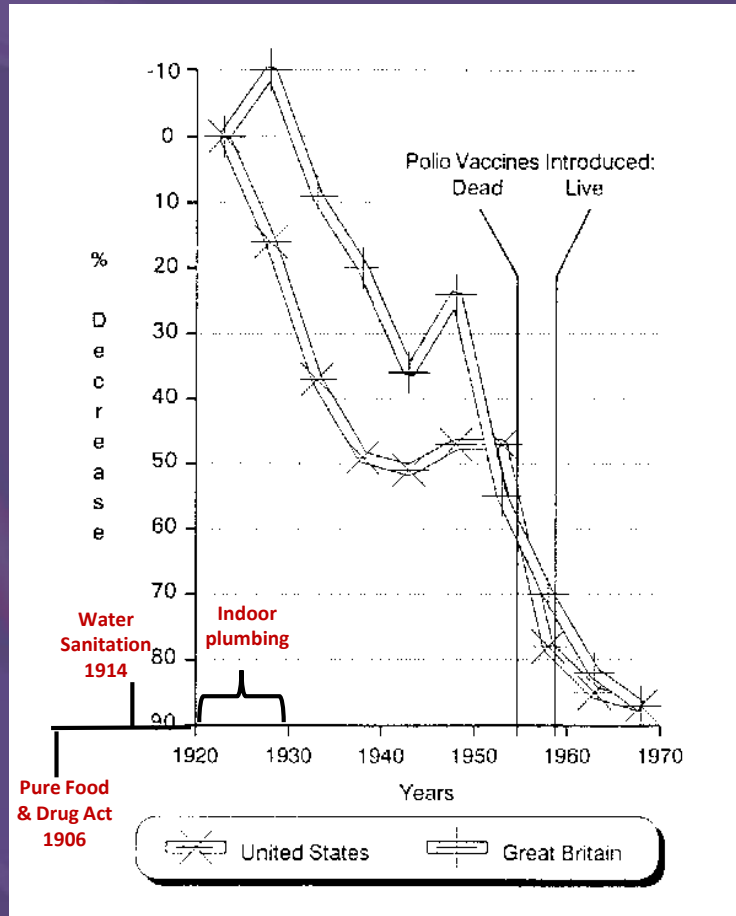
The Autoimmune Matrix©



Rate of Infections Diseases

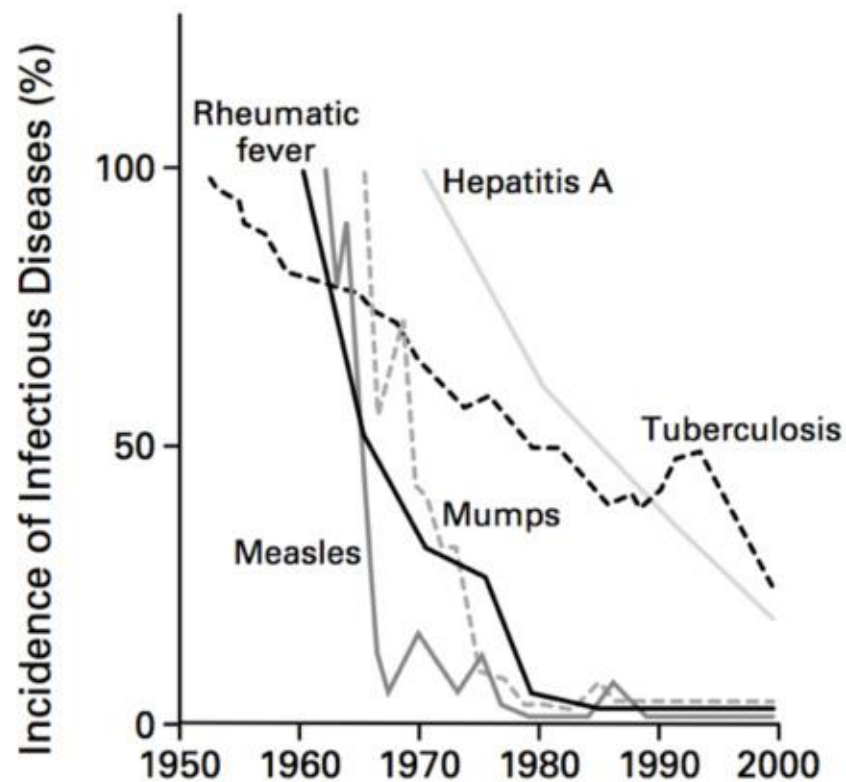


Source: CDC.gov

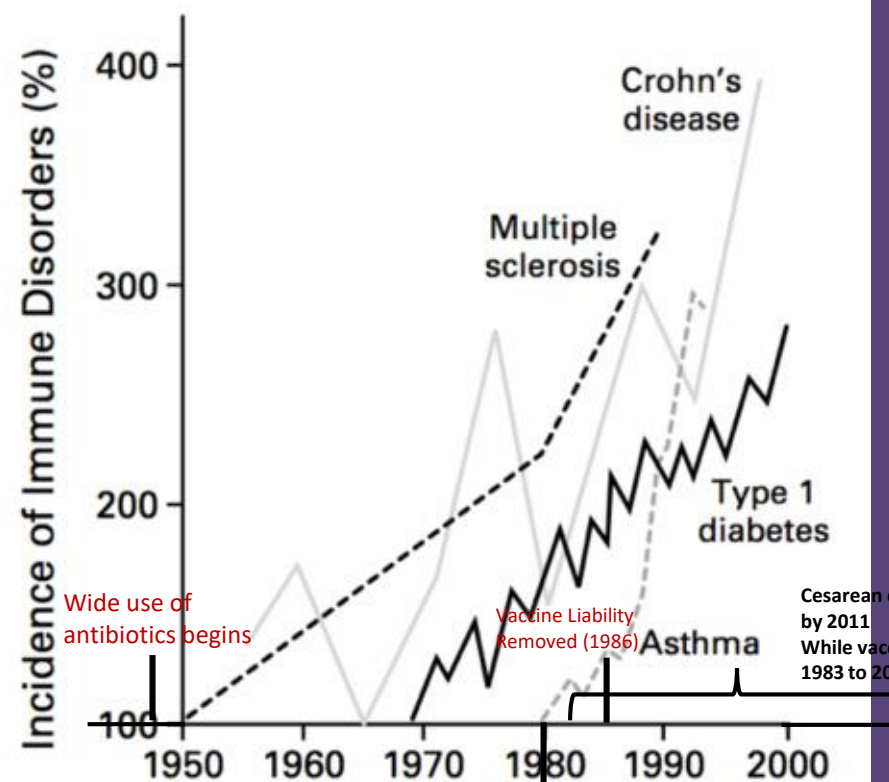


Source: Journal of American Physicians & Surgeons

A



B



Chemical use in US reaches more than 30,000 (1980)

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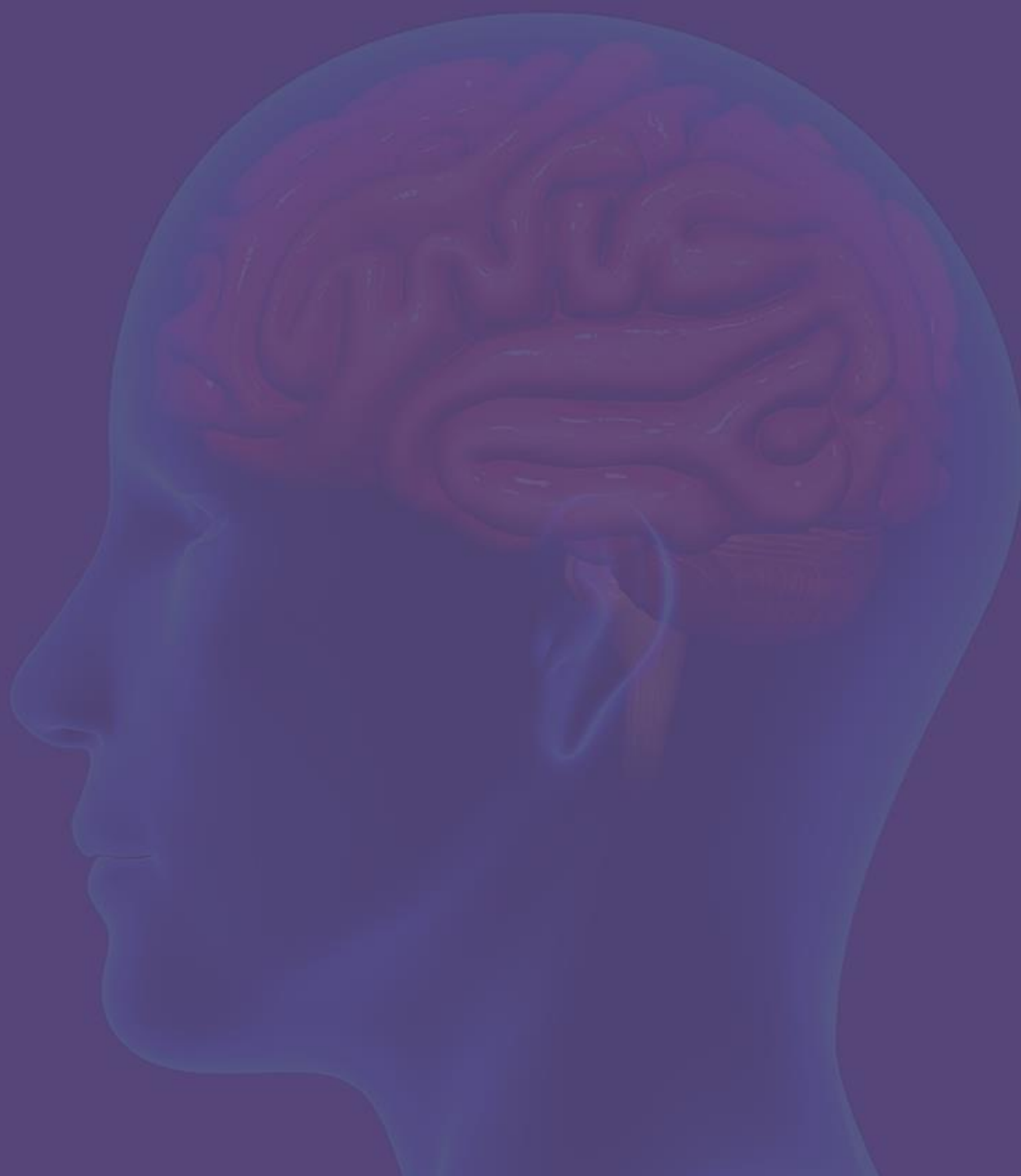
**There is no magic bullet.
AID is multifactorial.**

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What Causes AID

- We have 1 model of AID where the cause is known.
- Celiac Disease - Gluten



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Slay the Hydra



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