
NMI
SUMMIT
2024
An Energetic View:
Mitochondrial Nutrition
for Fatigue, the Brain,
& Healthy Ageing
Friday 11th October
Featuring Professor Nick Lane, Dr. Iain Hargreaves, Dr. Joseph Pizzorno,
Dr. Nina Fuller-Shavel, Dr. Deanna Minich and Benjamin Brown
An event by:
Nutritional Medicine
Institute
Platinum sponsors:
regenerus labs
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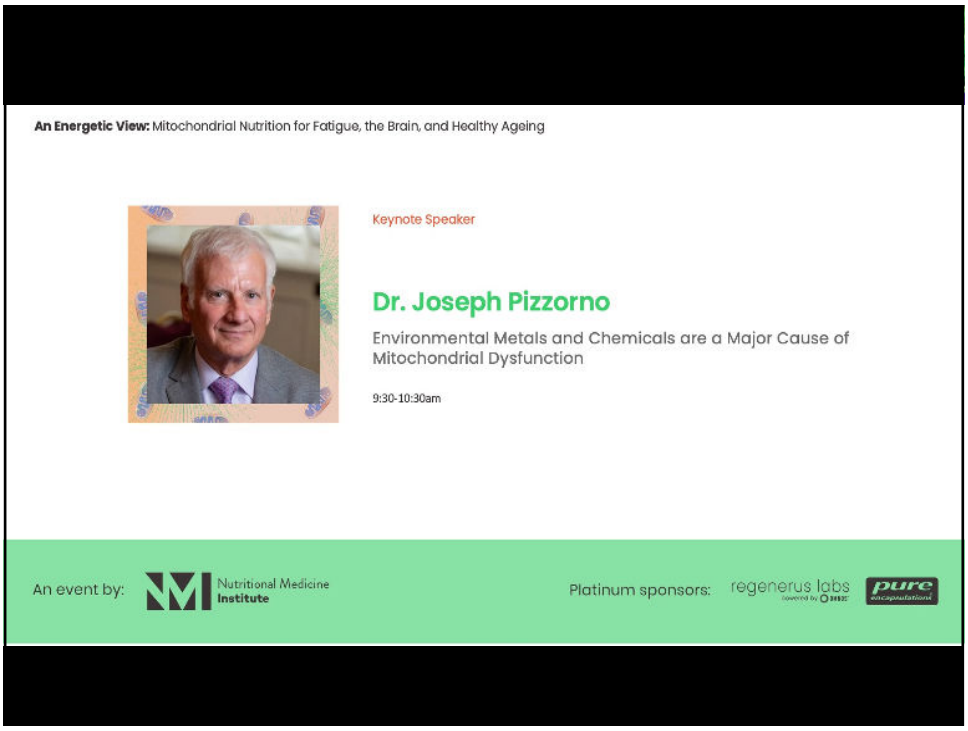
An Energetic View: Mitochondrial Nutrition for Fatigue, the Brain, and Healthy Ageing
Keynote Speaker
Dr. Joseph Pizzorno
Environmental Metals and Chemicals are a Major Cause of
Mitochondrial Dysfunction
9:30-10:30am
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Environmental Toxins and Mitochondrial Function

Dr. Joseph Pizzorno, ND
Co-Author *Clinical Environmental Medecine*.
Founding President, Bastyr University
Editor-in-Chief, *Integrative Medicine: A Clinician's Journal*
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Disclosure

Dr. Pizzorno is on the Scientific Advisory Board for Bioclinic Naturals which sells dietary supplements for healthcare professionals. No BCN products are recommended in this lecture.

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Overview

- Mitochondrial Function
- Mitochondria are Damaged by Environmental Metals and Chemicals
  - Assessment of Body Load
  - Ways to Increase Elimination
- Assessment of Mitochondrial Function
- Resources
- Summary

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How much ATP does the body at rest produce every day?

70 KG!

How about during maximal exercise?

0.5-1.0 KG/MIN!

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Mitochondrial Structure and Function

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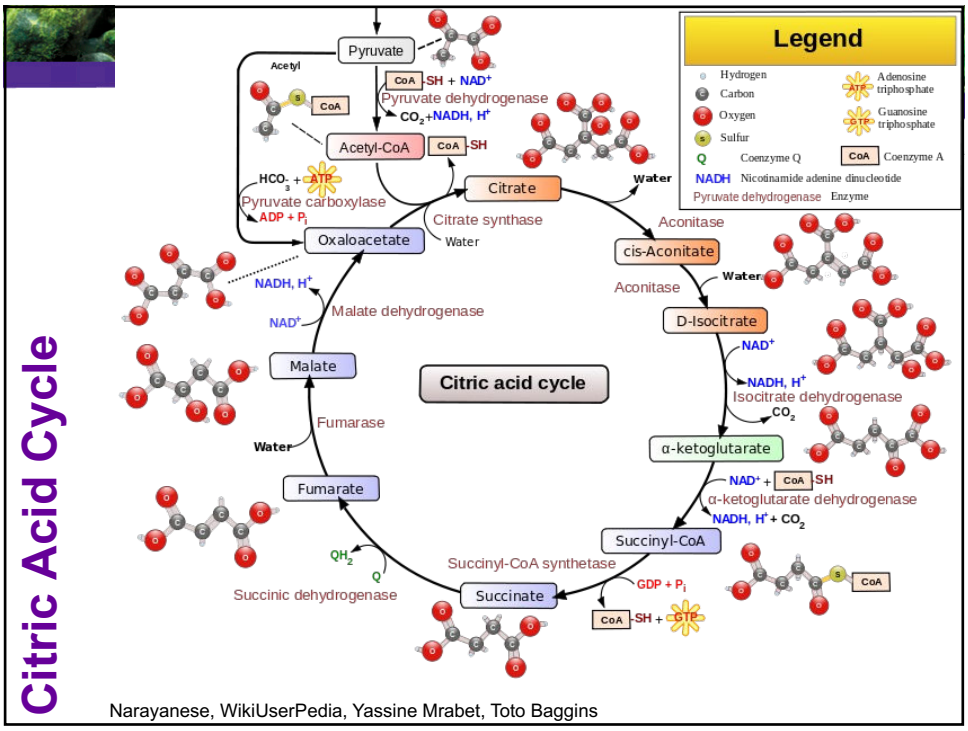

Narayanese, WikiUserPedia, Yassine Mrabet, Toto Baggins

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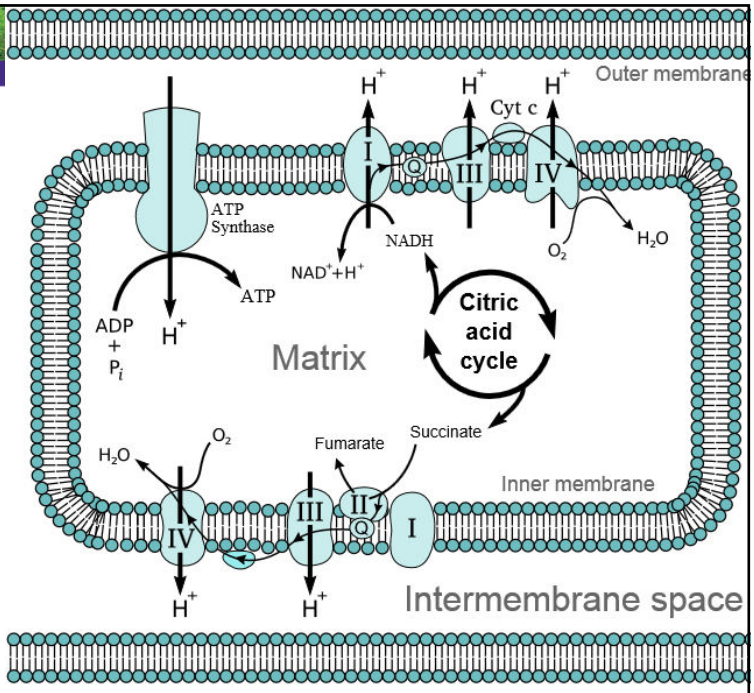
Citric Acid Cycle Feeds Electron Transport Chain



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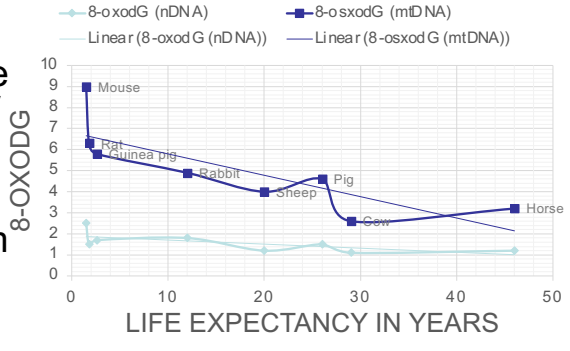
Mitochondrial Damage & Lifespan

- 8-oxo-7,8-dihydro-2'-deoxyguanosine (8-oxodG), marker of oxidative stress
- Inverse relationship between maximum lifespan and 8-oxodG mtDNA – not observed with nuclear DNA



Barja G, Herrero A. Oxidative damage to mitochondrial DNA is inversely related to maximum life span in the heart and brain of mammals. FASEB J. 2000 Feb;14(2):312-8.

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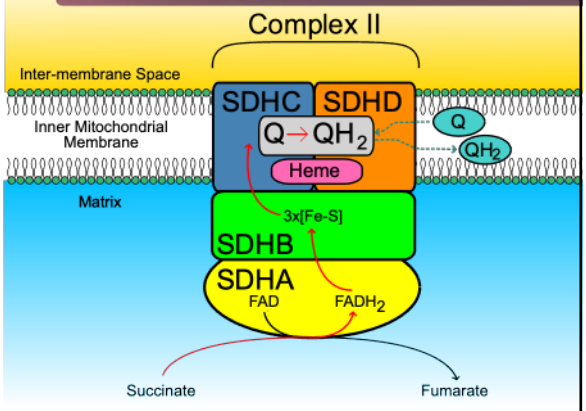
Succinate Dehydrogenase

- 4 subunits
- Both CAC and OXPHOS
- Succinate is a carcinogen
- SDH is a tumor suppressor



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Jawahar Swaminathan and MSD staff at the European Bioinformatics Institute - http://www.ebi.ac.uk/pdbe-srv/view/images/entry/1puz600.png, displayed on http://www.ebi.ac.uk/pdbe-srv/view/entry/1puz/summary, Public Domain, https://commons.wikimedia.org/w/index.php?curid=5929813

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Succinate Dehydrogenase Deficiency

- Mitochondrial myopathies:
  - 23% have SDH dysfunction
  - Activity only 34% of normal
- COVID
  - Succinate levels increase during COVID
  - In an animal model, increasing succinate dehydrogenase activity protects lungs from acute respiratory distress syndrome (ARDS).
- Neurodegenerative disease
  - Alzheimer's, Parkinson's and Huntington's
  - Mutations in SDH correlate with onset of neurodegenerative disorders

Vladutiu GD, Heffner RR. Succinate dehydrogenase deficiency. Arch Pathol Lab Med. 2000;124(12):1755-1758

Vohwinkel, Christine U et al. Targeting alveolar-specific succinate dehydrogenase A attenuates pulmonary inflammation during acute lung injury. FASEB journal : official publication of the Federation of American Societies for Experimental Biology vol. 35,4 (2021): e21468

Jodeiri Farshbaf M, Kiani-Esfahani A. Succinate dehydrogenase: Prospect for neurodegenerative diseases. Mitochondrion. 2018;42:77-83.

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Example Causes of SDH Dysfunction

- Genetic (mitochondriopathies) - Rare
- Arsenic, fungicides, DDT
- Vitamin C deficiency
- Succinate increased by COVID and cancer cells

Karimzadeh P, Keramatipour M, Karamzade A, Pourbakhtyaran E. Succinate Dehydrogenase Deficiency: A Treatable Neurometabolic Disorder. Iran J Child Neurol. 2020;14(4):111-116.

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Environmental Metals and Metalloids Damage Mitochondria

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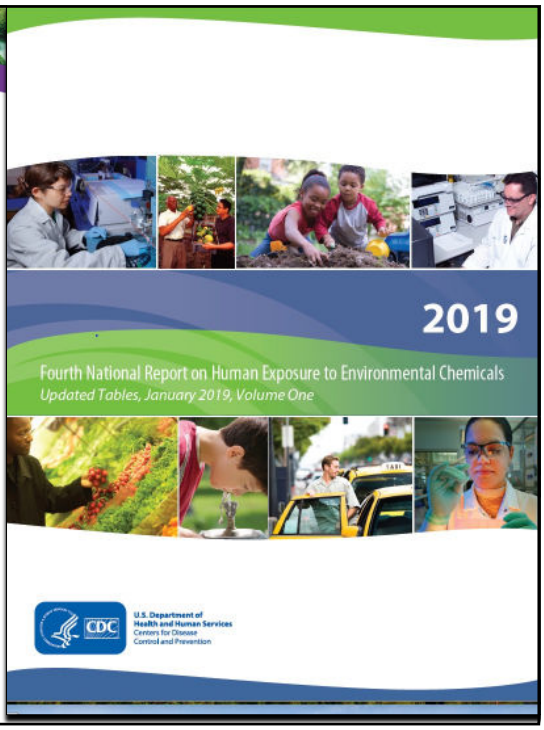
Source Population Toxicant Load

- US data only
- No other country appears to be systematically measuring population load
- 2 volume set
- >1,000 toxins



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Toxins

Non-Persistent

- Arsenic
- Bisphenols (BPx)
- Glyphosate
- Polycyclic aromatic hydrocarbons (PAHs)
- Parabens
- Phthalates
- Solvents

Drugs!

Persistent

- Cadmium, lead mercury
- Halogenated chemicals
  - Organochlorine pesticides
  - Organophosphate pesticides
  - Perfluorocarbons
  - Polybrominated diphenyl ethers (PBDEs)
  - Polychlorinated biphenyls (PCBs)
- Pyrethroid pesticides

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Arsenic

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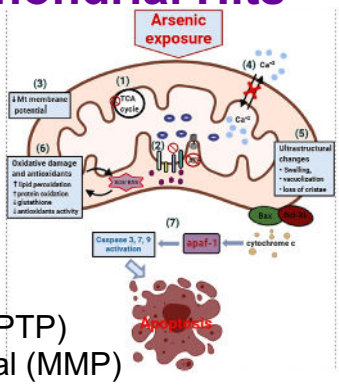
Arsenic: Multiple Mitochondrial Hits



- Inhibits:
  - Isocitrate dehydrogenase
  - α-ketoglutarate dehydrogenase
  - Succinate dehydrogenase
  - Complex I and IV
- Decreases ATP production
- Increased ROS generation
- Opens permeability transition pores (PTP)
- Breaks down mito membrane potential (MMP)
- Can inducepoptosis

Prakash C, et al. Mitochondrial Dysfunction in Arsenic-Induced Hepatotoxicity: Pathogenic and Therapeutic Implications. Biol Trace Elem Res. 2022 Jan;200:261-270.

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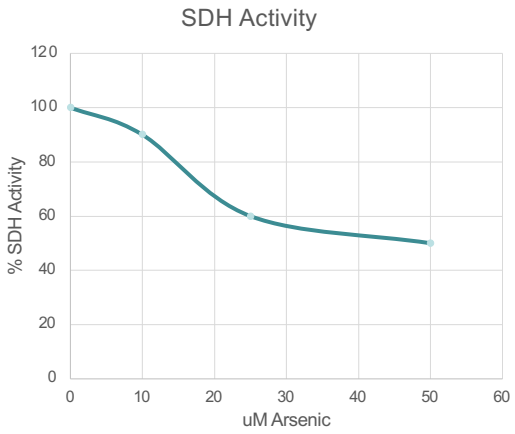
Arsenic

- Cell culture
- **Average** person has 1.3-7.5 uM arsenic in various tissues



Hosseini MJ, Shaki F, Ghazi-Khansari M, Pourahmad J. Toxicity of Arsenic (III) on Isolated Liver Mitochondria: A New Mechanistic Approach. Iran J Pharm Res. 2013;12(Suppl):121-138.
Yamauchi H, Yamamura Y. Concentration and chemical species of arsenic in human tissue. Bull Environ Contam Toxicol. 1983;31(3):267-270

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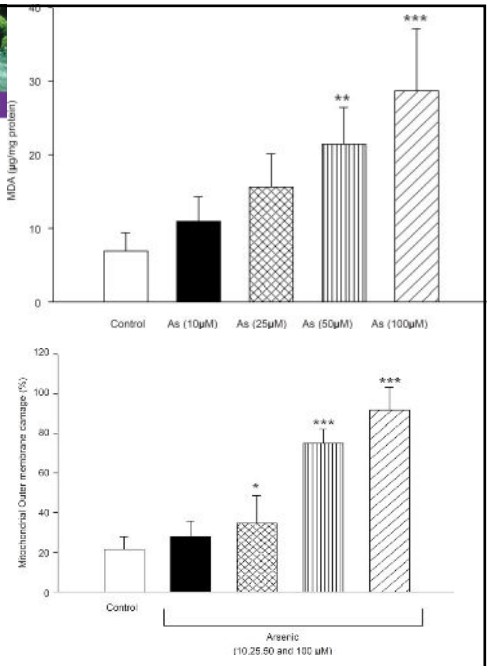
Arsenic & ROS Damage

- Increase:
  - H_2O_2
  - Superoxide
  - RNS formation
- Results in:
  - Lipid peroxidation (MDA/ malondialdehyde)
  - GSH depletion
  - mtDNA and membrane damage



Hosseini MJ, et al. Toxicity of Arsenic (III) on Isolated Liver Mitochondria: A New Mechanistic Approach. Iran J Pharm Res. 2013 Winter;12(Suppl):121-38.

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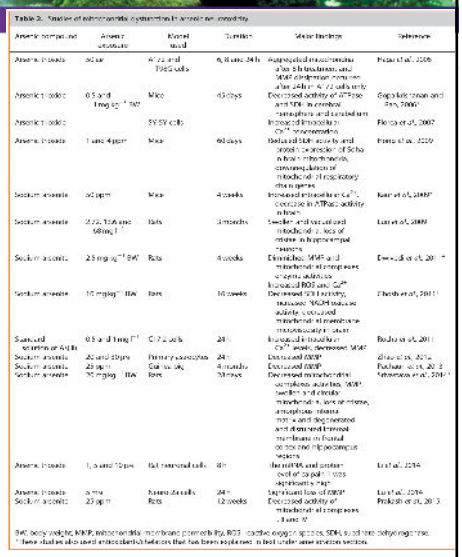
Arsenic Neurotoxicity due to Mitochondrial Damage

- Arsenic can cross the blood–brain barrier (BBB), and accumulate in the brain (all forms of arsenic)
- Arsenic triggers neuronal cell death through a mitochondria–dependent apoptosis pathway (JNK/ERK activation)



Prakash C, et al. Mitochondrial oxidative stress and dysfunction in arsenic neurotoxicity: A review. J Appl Toxicol. 2016 Feb;36(2):179-88.

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Mitochondrial DNA copy number (mtDNAcn)

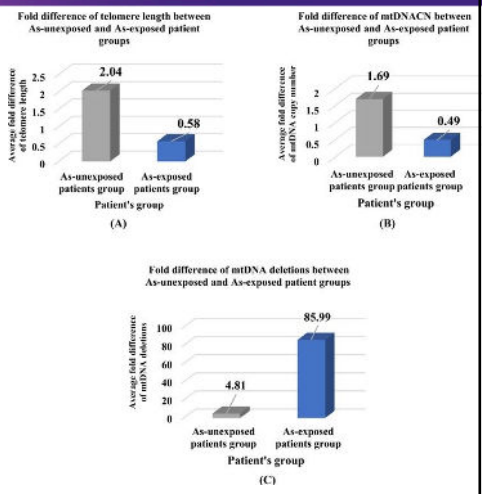
- 272 arsenicosis patients
  - 155 cases without liver damage and 117 cases with liver damage
- 218 participants not exposed to arsenic
  - 155 cases without liver damage and 63 cases with liver damage
- Peripheral blood mtDNAcn in patients with arsenic-induced liver damage was significantly decreased and negatively correlated with serum ALT, AST, and GGT
- Mutations in mtDNA combined with mtDNAcn may together determine mitochondrial fitness

Wang Q, et al. Reduced Peripheral Blood Mitochondrial DNA Copy Number as Identification Biomarker of Suspected Arsenic-Induced Liver Damage. Biol Trace Elem Res. 2023 Nov;201(11):5083-5097.

22

mtDNA Deletions & CN

- Arsenic-affected and unaffected areas in Bangladesh, patients having heart surgery
- Arsenic exposure (assessed in nails and cardiac tissues) associated with
  - 1.2-fold decreased mtDNAcn
  - 81-fold higher amount of mitochondrial DNA deletion
  - 1.5-fold shorter telomere length



Khaleda L, et al. Arsenic-Induced Cardiovascular Diseases and their Correlation with Mitochondrial DNA Copy Number, Deletion, and Telomere Length in Bangladeshi Population. Cardiovasc Toxicol. 2024 Jan;24(1):27-40.

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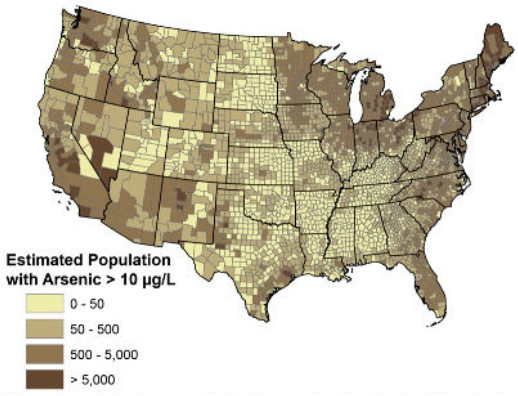
Portion of Population with >10 ugL Arsenic Levels from Water



https://www.usgs.gov/mission-areas/water-resources/science/arsenic-and-drinking-water?qt-science_center_objects=0#qt-science_center_objects (accessed 2022-02-06)

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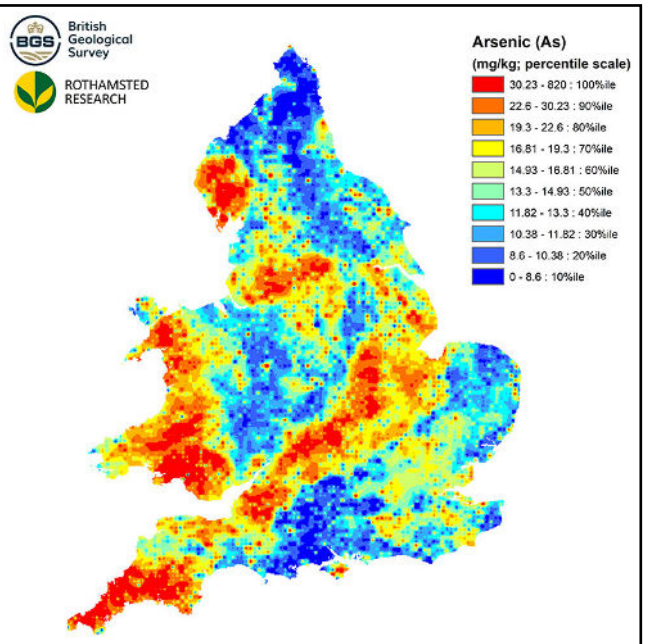
Arsenic in UK Soil



Advanced soil geochemical atlas of England and Wales | UK Soil Observatory | UK Research and Innovation (Accessed 2024-09)

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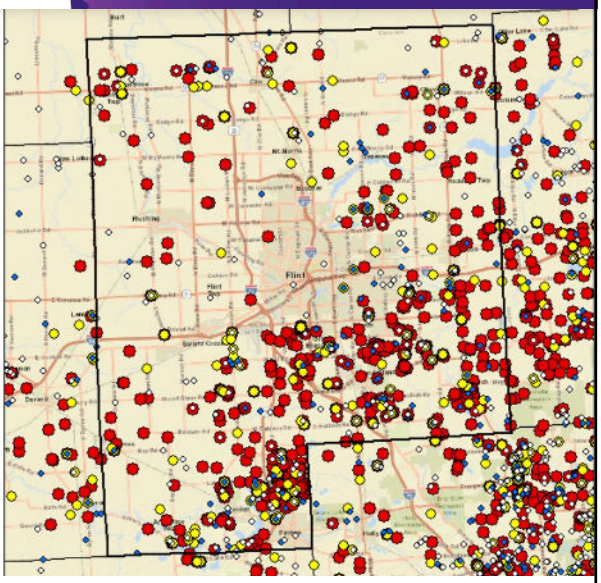
Arsenic in Flint Drinking Water



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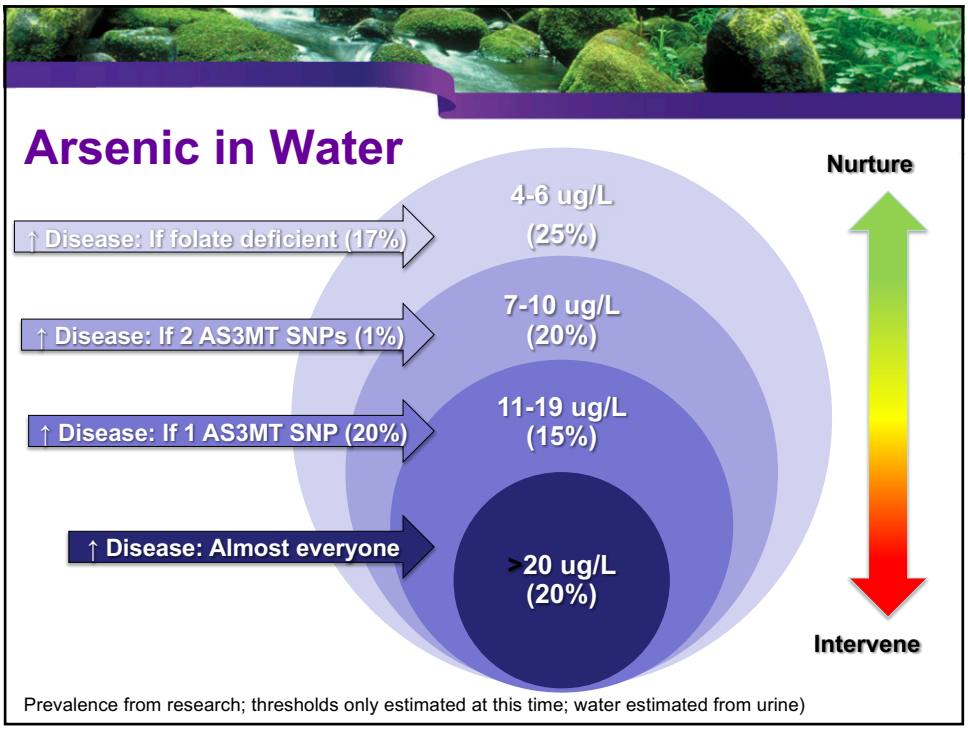

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Clinical Takeaways

Arsenic

- Major diseases: Gout, cancer,diabetes, cardiovascular disease
- Primary sources: Water, rice, chicken
- Best measure: First morning urine
- Best intervention:
  - Avoidance
  - Folates (not folic acid)

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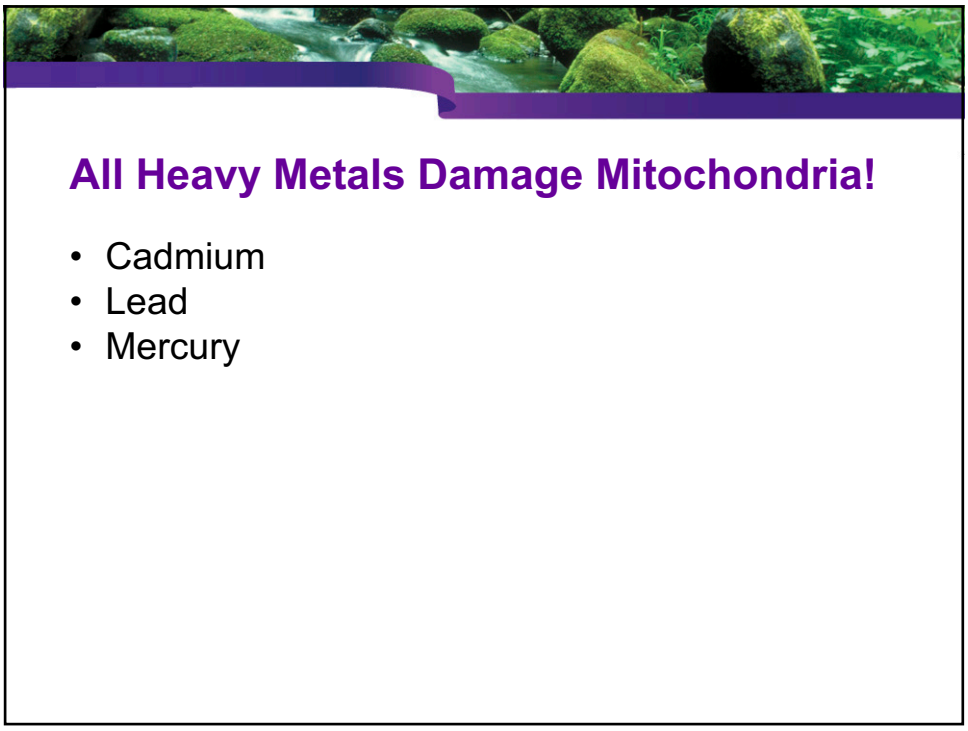

All Heavy Metals Damage Mitochondria!

- Cadmium
- Lead
- Mercury

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Environmental Chemicals that Damage Mitochondria

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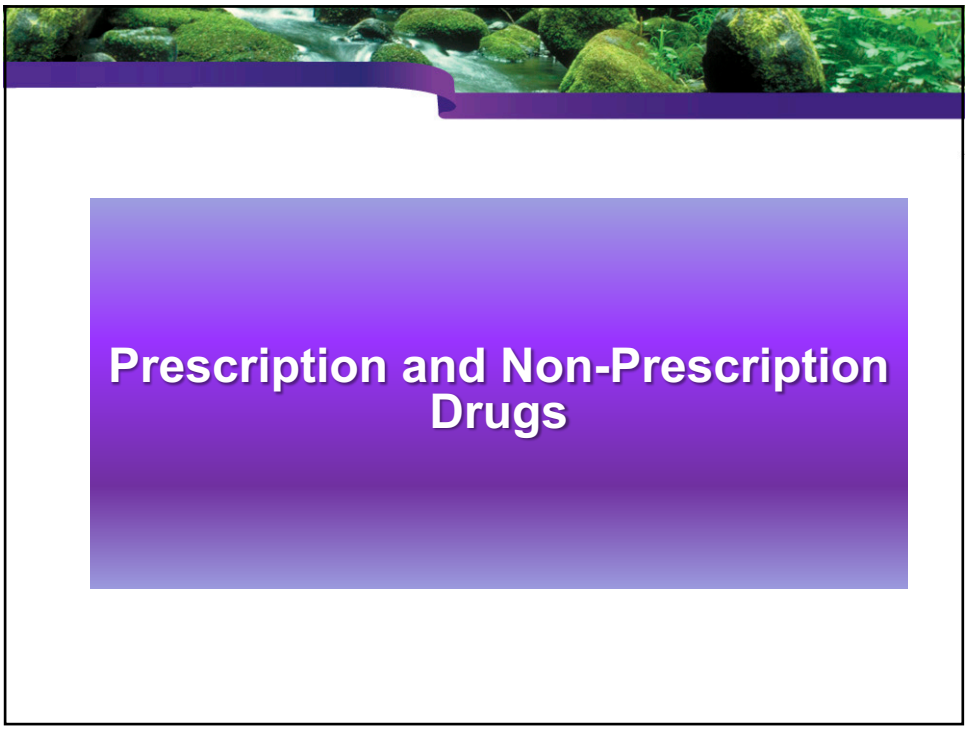
Prescription and Non-Prescription Drugs

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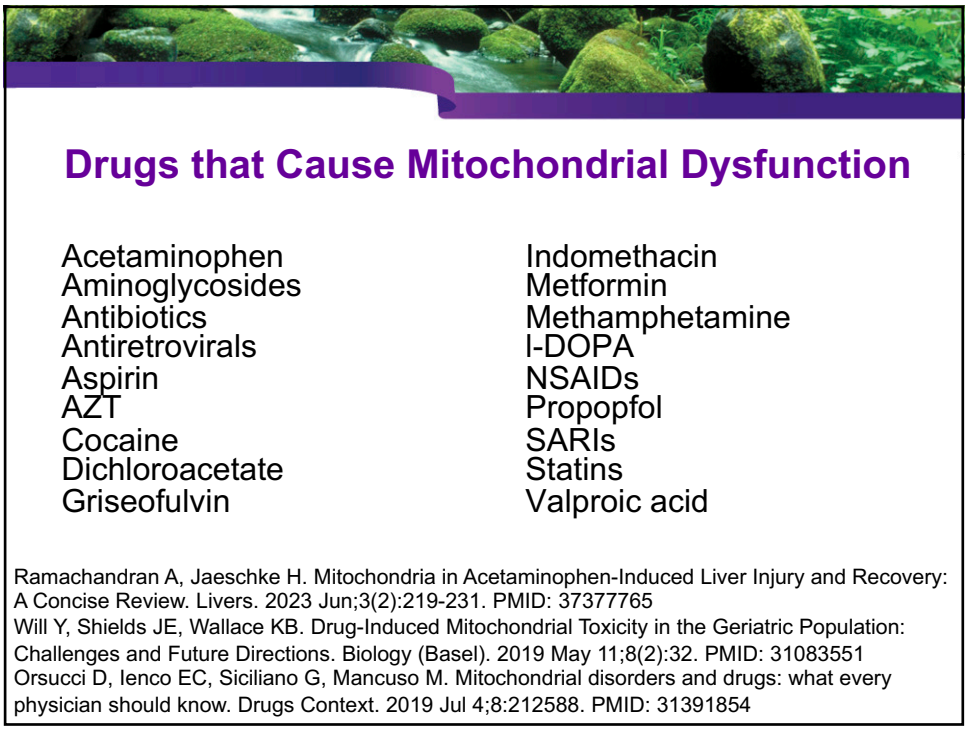
Drugs that Cause Mitochondrial Dysfunction
Acetaminophen
Aminoglycosides
Antibiotics
Antiretrovirals
Aspirin
AZT
Cocaine
Dichloroacetate
Griseofulvin
Indomethacin
Metformin
Methamphetamine
l-DOPA
NSAIDs
Propopfol
SARIs
Statins
Valproic acid
Ramachandran A, Jaeschke H. Mitochondria in Acetaminophen-Induced Liver Injury and Recovery: A Concise Review. Livers. 2023 Jun;3(2):219-231. PMID: 37377765
Will Y, Shields JE, Wallace KB. Drug-Induced Mitochondrial Toxicity in the Geriatric Population: Challenges and Future Directions. Biology (Basel). 2019 May 11;8(2):32. PMID: 31083551
Orsucci D, Ienco EC, Siciliano G, Mancuso M. Mitochondrial disorders and drugs: what every physician should know. Drugs Context. 2019 Jul 4;8:212588. PMID: 31391854

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Drugs That Poison Mitochondria

Drug or Toxin	Mechanism of Activity
Acetaminophen	Poisons mitochondria
Arachidonic acid	Inhibits complex I
Aspirin	Uncouples electron transport
AZT	Inhibits mitochondrial replication
Cocaine, Ethanol	Uncouples OXPHOS, ↑radical production
Griseofulvin	Interferes with mitochondrial replication
Methamphetamine	Uncouples electron transport chain
L-DOPA	Inhibits Complex I
NSAIDs	Uncouples electron transport
Rotenone and other pesticides	Inactivates Complex I NADH dehydrogenase
Statins	Interferes with synthesis of CoQ10
Tobacco	Uncouples OXPHOS, ↑free radical production
Valproic, Adipic and Benzoic Acids	Increases mitochondrial permeability

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Antibiotics Poison Mitochondria

After 4 days	Ciplofloxan	Ampicillin	Kanamycin	Tetracycline
ATP Production	-90%	-75%	-80%	-20%
ROS	+250%	+200%	+240%	+40%
MDA	+90%	+80%	+75%	+20%
8-OHdG	+100%	+720%	+400%	230%

Mitochondrial DNA repair limited

- Cell study, reproduced in animals
- Damage decreased 50% with NAC supplementation

Kalghatgi S, et al. Bactericidal antibiotics induce mitochondrial dysfunction and oxidative damage in Mammalian cells. Sci Transl Med. 2013 Jul 3;5(192):192

34

Statins Decrease CoQ_{10} → ↑ Electron Leakage

- HMG-CoA Reductase Inhibitors
- Adverse effects appear to be related to mitochondrial dysfunction, including cognitive loss, neuropathy, pancreatic and hepatic dysfunction, myalgia, cancer, and sexual dysfunction
  - Meta-analysis did not find significant mortality statin benefit for treatment of high risk primary prevention (i.e. those without established CHD).
- Most trials show benefit of CoQ10 for myalgia from statins
- Statins dose-dependent reduction in blood and lymphocyte CoQ10
- Plasma CoQ10 predictor of mortality in patients with heart failure

Ray KK, et al. Statins and all-cause mortality in high-risk primary prevention: a meta-analysis of 11 randomized controlled trials involving 65,229 participants. Arch Intern Med. 2010 Jun 28;170(12):1024-31.
Golomb BA, Evans MA. Statin adverse effects : a review of the literature and evidence for a mitochondrial mechanism. Am J Cardiovasc Drugs. 2008;8(6):373-418.
Mortensen SA, et al. Dose-related decrease of serum coenzyme Q10 during treatment with HMG-CoA reductase inhibitors. Mol Aspects Med 1997;18:137-44
Molyneux SL, Florkowski CM, George PM, et al. Coenzyme Q10: an independent predictor of mortality in chronic heart failure. J Am Coll Cardiol. 2008 Oct 28;52(18):1435-41.
Caso G, et al. Effect of coenzyme q10 on myopathic symptoms in patients treated with statins. Am J Cardiol. 2007 May 15;99(10):1409-12.

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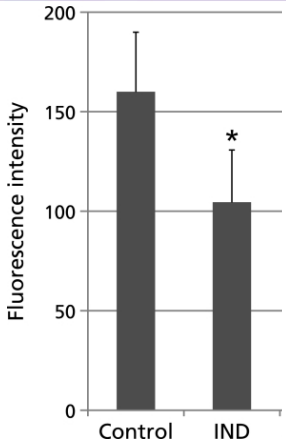
Indomethacin Poisons Mitochondria

- Concentration dependently decreases cellular ATP
- Inhibits the activity of mitochondrial complex I and causes accumulation of NADH
- Leads to overproduction of mitochondrial $O_2^{\cdot-}$



Rai 2012; Creative Commons

Carrasco-Pozo C, et al. Differential protective effects of quercetin, resveratrol, rutin and epigallocatechin gallate against mitochondrial dysfunction induced by indomethacin in Caco-2 cells. Chem Biol Interact. 2012 Feb 5;195(3):199-205
Rai K et al. Lansoprazole inhibits mitochondrial superoxide production and cellular lipid peroxidation induced by indomethacin in RGM1 cells. J Clin Biochem Nutr (2011)

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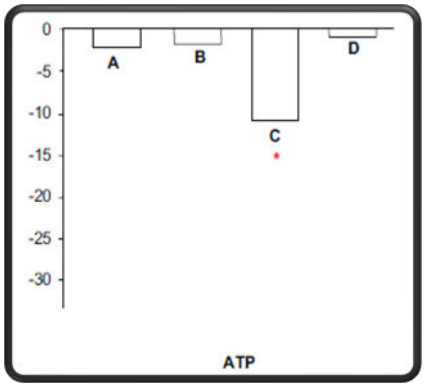
Alcohol Decreases ATP Production

- Ethanol converted to acetaldehyde
  - Mitochondrial aldehyde dehydrogenase 2 (ALDH2) converts acetaldehyde to acetate.
  - Depletes NADH
  - Increases ROS
- 40 g/d for 30d
  - Beer and wine: no effect
  - Spirits: decreased ATP production 11%



Addoloratoa G, et al. Effects of short-term moderate alcohol administration on oxidative stress and nutritional status in healthy males. Appetite 2008;50:50–56
Manzo-Avalos S, Saavedra-Molina A. Cellular and mitochondrial effects of alcohol consumption. Int J Environ Res Public Health. 2010 Dec;7(12):4281-304

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Clinical Takeaways

Prescription and non-prescription drugs

- Major diseases: 4th leading cause of death
- Primary sources: Choice
- Best measure: History
- Best intervention:
  - Avoidance
  - Glutathione support

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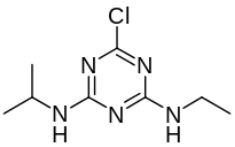

Pesticides and Herbicides

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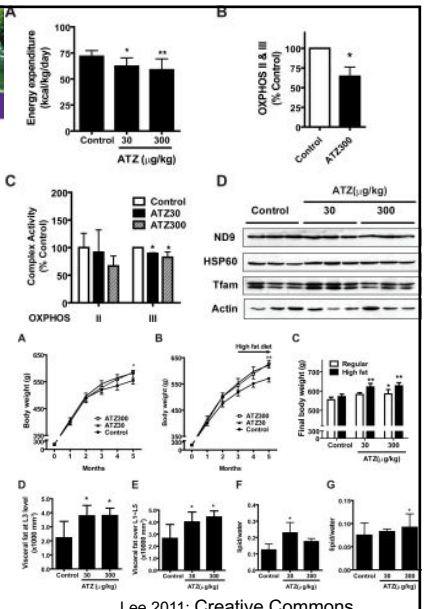
Organophosphate Pesticides Poison Mitochondria

- Atrazine has been widely used in US since 1960's, and inhibits photosynthesis and mitochondrial function
- Animal models show both mitochondrial inhibition and insulin resistance
- Induces obesity
- Overlap between obesity rates and areas of heavy use



Lee 2011; Creative Commons

Lim S, et al. Chronic exposure to the herbicide, atrazine, causes mitochondrial dysfunction and insulin resistance. PLoS One. 2009;4(4):e5186.

Lee HK. Mitochondrial dysfunction and insulin resistance: the contribution of dioxin-like substances. Diabetes Metab J. 2011 Jun;35(3):207-15.

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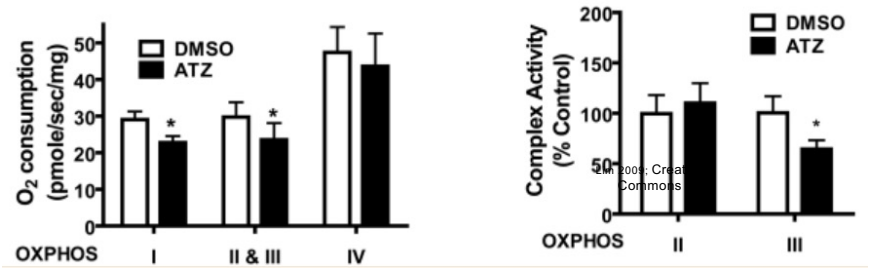
Atrazine Impairs OXPHOS

- Human cell culture



Lim S, Ahn SY, Song IC, et al. Chronic exposure to the herbicide, atrazine, causes mitochondrial dysfunction and insulin resistance. PLoS One. 2009;4(4):e5186.

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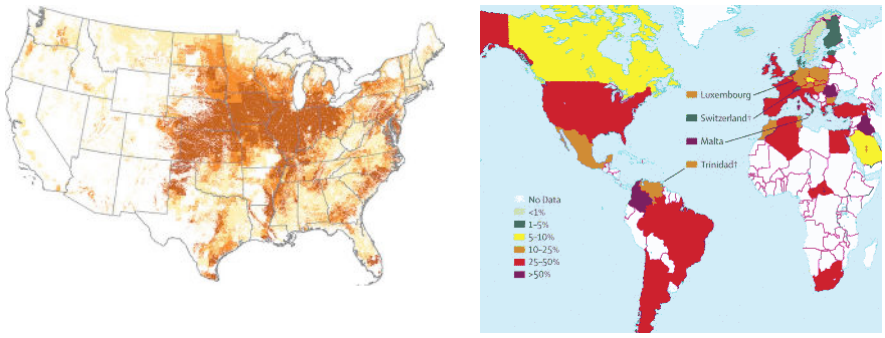
Atrazine Use Widespread



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Clinical Takeaways

Pesticides and Herbicides

- Major disease: Neurodegeneration, diabetes, cancer (incidence of virtually every chronic disease shows dose-dependent relationship)
- Primary sources: Chemically grown foods, yard spraying
- Best measure: Urinary metabolites
- Best intervention: Avoidance, fiber, glutathione support

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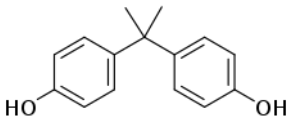

Bisphenols (BPx)

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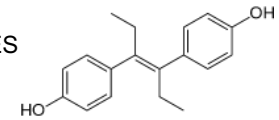
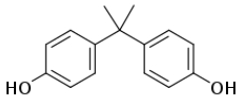
BPA — Troubled History

- BPA and DES are synthetic oestrogens developed during the 1930s:
  - DES 'won' and was prescribed for pregnant women.
  - Discontinued due to urogenital cancers in children and many other clinical problems.
  - Very similar chemical structures.

DES



BPA



- BPA 'put on the shelf' until 1950s when its ability to harden plastics was discovered.
- So widely used very difficult to avoid exposure:
  - Technically non-persistent, but practically semi-persistent.

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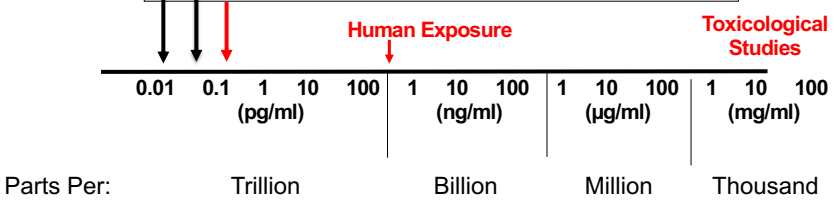
Receptor Site Activation by BPA

BISPHENOL A STIMULATES:

Rat Pituitary Cells

Human Breast Cancer Cells

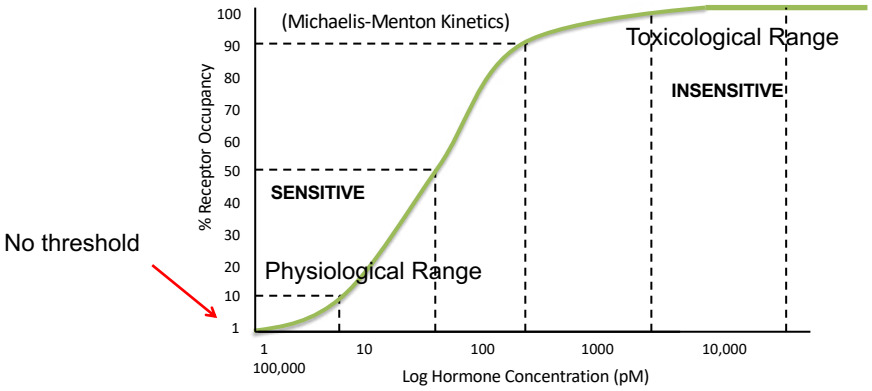
Mouse Pancreatic Insulin Secretion



Adapted from. Welshons, W.V., Nagel, S.C. and vom Saal, F.S. Large effects from small exposures. III. Endocrine mechanisms mediating effects of bisphenol A at levels of human exposure. Endocrinol. 147, S56-S6, 2006.

46

Hormone-Receptor Interactions are Highly Specific and Sensitive only within a Narrow Concentration



Adapted from: W.V. Welshons et al. EHP 111, 994-1006, 2003; D.M. Sheehan Env. Res. 100,93-99, 2006

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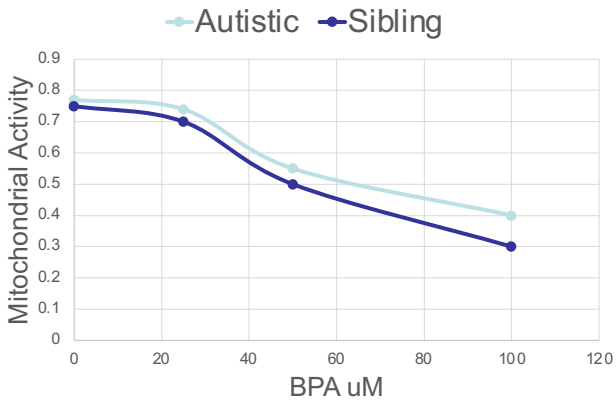
Bisphenols

- Human cell culture
- BPA is just the tip of the bisphenol iceberg
- Proportional inhibition of ATP by BPA in both autistic and controls



Kaur K, Chauhan V, Gu F, Chauhan A. Bisphenol A induces oxidative stress and mitochondrial dysfunction in lymphoblasts from children with autism and unaffected siblings. Free Radic Biol Med. 2014;76:25-33

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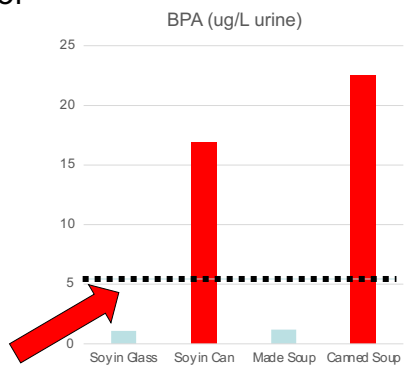
Packaging Puts BPA Into Food

- One 12oz serving daily for 1 week of either fresh soup or canned lentil soup (Progresso)
  - 12-fold increase in BPA
- 2 servings of 6 ounces Soy milk in can compared to glass
  - 16-fold increase in BPA
  - Systolic BP elevated 4.5 mm Hg
- **Diabetes 2x risk threshold?**



Carwile JL, Ye X, Zhou X, et al. Canned soup consumption and urinary bisphenol A: a randomized crossover trial. JAMA. 2011 Nov 23;306(20):2218-20.
Bae S, Hong YC. Exposure to bisphenol A from drinking canned beverages increases blood pressure: randomized crossover trial. Hypertension. 2015 Feb;65(2):313-9.

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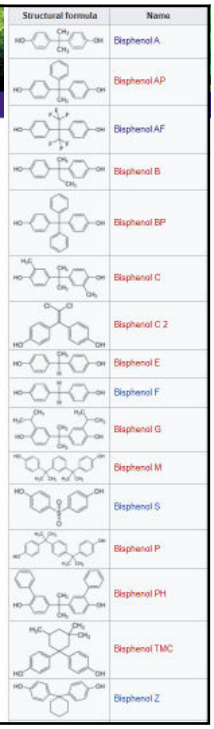
Alternatives Are NOT Safer!

- Typical substitutes are BPF, BPS, BPZ
- Very similar endocrine disruption
- Considered semi-persistent because so prevalent in society
- As BPA levels have gone down, other bisphenols have gone up in proportion



Bisphenol - Wikipedia

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Clinical Takeaways

BPA – Bisphenols!

- Major diseases: Endocrine-related disorders, diabetes, obesity, infertility
- Primary sources: Food packaging, thermal paper, toys, healthcare equipment, dental materials
- Best measure: Urine bisphenols
- Best intervention: Avoidance, antioxidants

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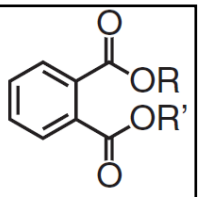

Phthalates

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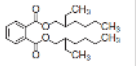
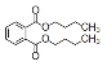
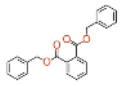
Phthalates

Phthalate	Diethyl phthalate	Diisononyl phthalate
		
Diethylhexyl phthalate	**Dibutyl phthalate**	**Dibenzyl phthalate**
		

- Worldwide high-production chemicals used in many ways:
  - Make plastics more flexible and resilient
  - Solubilize fragrances in health and beauty aids
- Weakly bound to plastics and easily released into environment
- 2 categories: high-molecular-weight (HMW) or low-molecular-weight (LMW) compounds.
- In general:
  - HMW phthalates mainly from diet
  - LMW phthalates mainly from non-food sources

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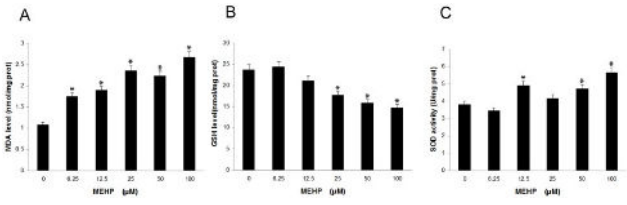
Phthalates

- Dose-dependent:
  - Increase in oxidative stress markers
  - Depletion of reduced glutathione



Ban JB, Fan XW, Huang Q, et al. Mono-(2-ethylhexyl) phthalate induces injury in human umbilical vein endothelial cells. PLoS One. 2014;9(5):e97607.

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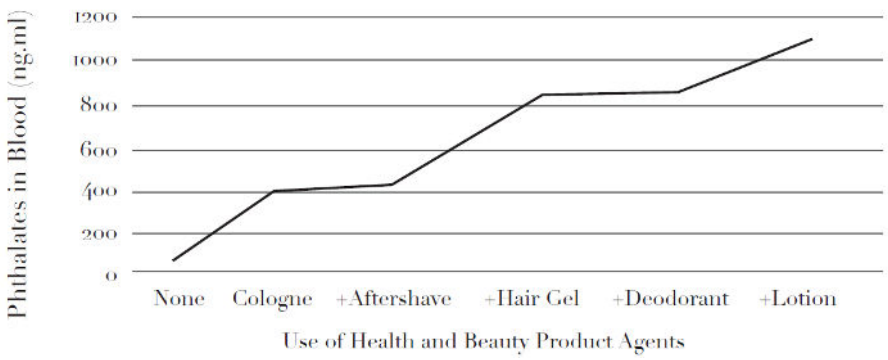
Health and Beauty Aids (HABAs) Significant Source of Phthalates



Duty SM, et al. Personal care product use predicts urinary concentrations of some phthalate monoesters. Environ Health Perspect. 2005 Nov;113(11):1530-5

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Fast Food = Lots of Phthalates

- Hamburgers, fries, chicken nuggets, chicken burritos, cheese pizza (n = 64) and gloves (n = 3) from restaurants and analyzed them for 11 chemicals
- DEHT highest concentrations in both foods (2510 µg/kg) and gloves (28-37% by weight)!
- Median DEHT concentrations were significantly higher in burritos than hamburgers (6000 µg/kg vs. 2200 µg/kg; $p < 0.0001$)
- DEHT was not detected in fries
- Cheese pizza had lowest levels of most chemicals.

Edwards L, McCray NL, VanNoy BN, et al. Phthalate and novel plasticizer concentrations in food items from U.S. fast food chains: a preliminary analysis [published online ahead of print, 2021 Oct 27]. J Expo Sci Environ Epidemiol. 2021;10.1038/s41370-021-00392-8

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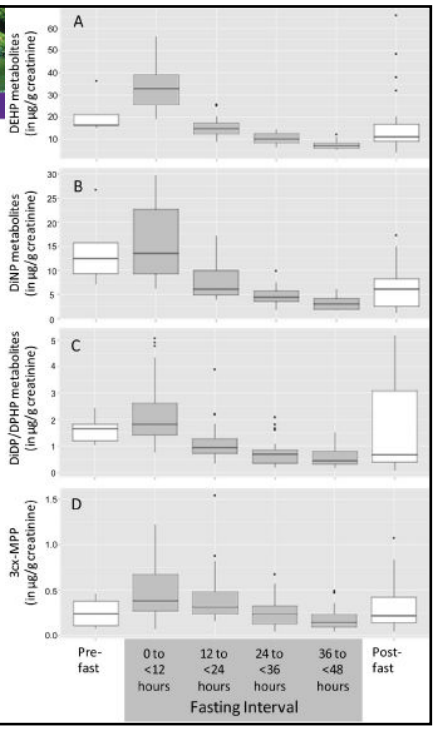
Dramatic Increase in HMW Excretion During Water Fasting



Koch HM, Lorber M, Christensen KL, et al. Identifying sources of phthalate exposure with human biomonitoring: results of a 48h fasting study with urine collection and personal activity patterns. Int J Hyg Environ Health. 2013 Nov;216(6):672-81PMID: 23333758.

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Clinical Takeaways

Phthalates

- Major disease: Infertility, low testosterone, obesity, diabetes, allergies, asthma, autism, mood disorder, reduced cognition
- Primary sources: Solid plastic products in the home, plastic-covered foods, personal care products, house dust
- Best measure: Review home for phthalate-containing products and urinary phthalate metabolites
- Best intervention: Avoidance, flavonoids, fasting

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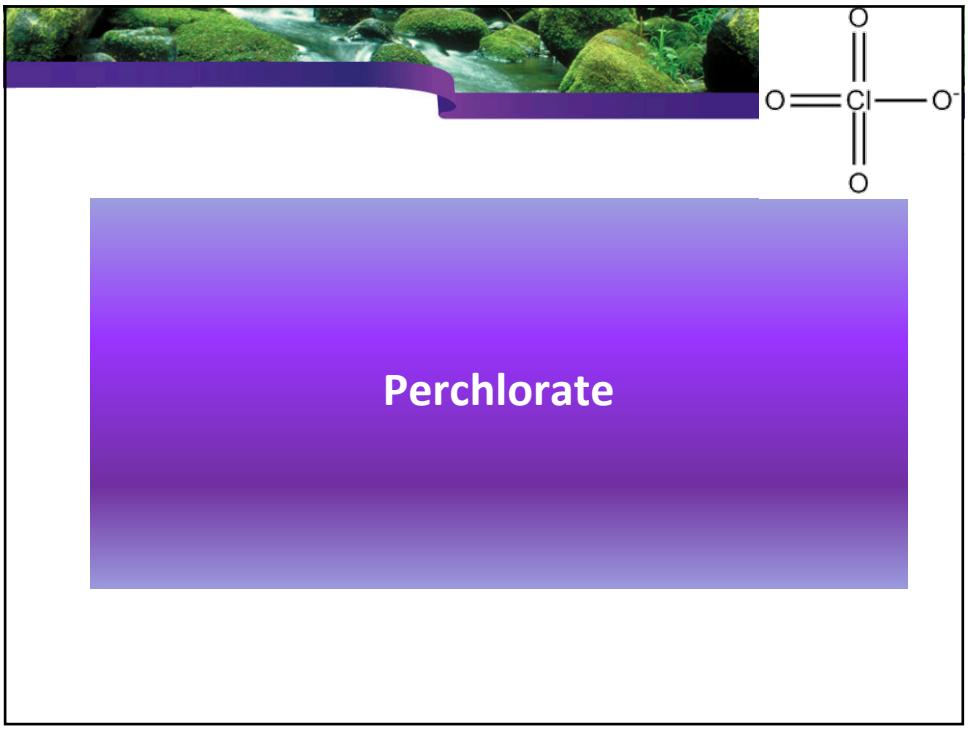

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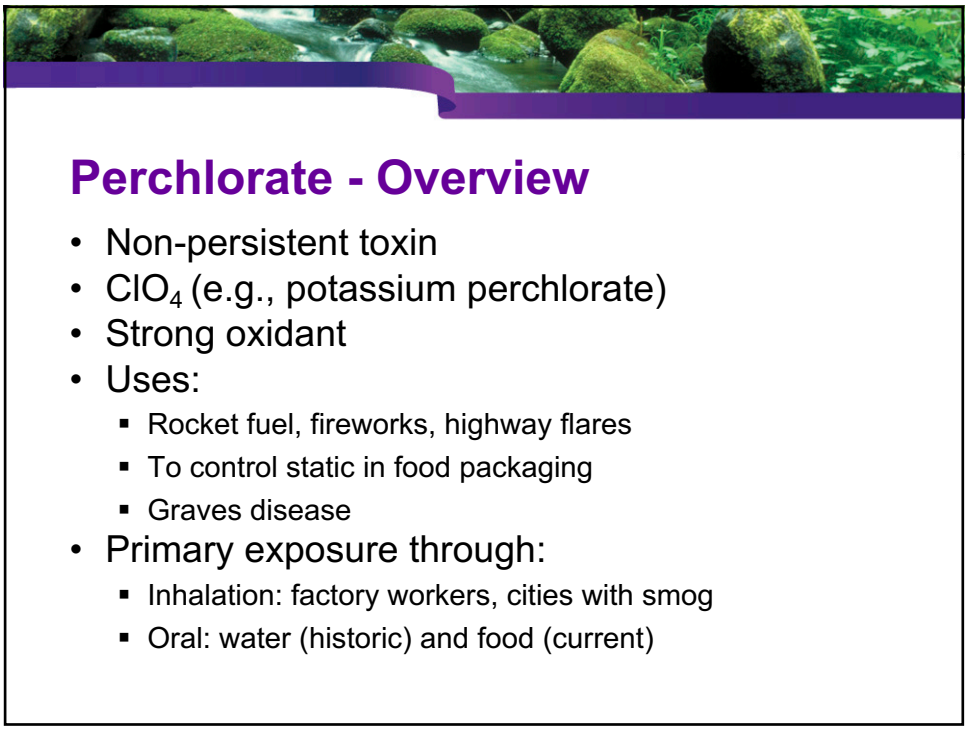

Perchlorate - Overview

- Non-persistent toxin
- ClO_4 (e.g., potassium perchlorate)
- Strong oxidant
- Uses:
  - Rocket fuel, fireworks, highway flares
  - To control static in food packaging
  - Graves disease
- Primary exposure through:
  - Inhalation: factory workers, cities with smog
  - Oral: water (historic) and food (current)

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Perchlorate – Contamination

- Sources
  - Primary source industrial waste
  - MAY be created by chlorination of water
  - Naturally produced
- Widely contaminates the environment
  - Public water supply average: 1.16 PPM
  - Public water supplies above 4.1 PPM: 4%
  - Worse near military bases, manufacturing facilities

Steinmaus CM. Perchlorate in Water Supplies: Sources, Exposures, and Health Effects. Curr Environ Health Rep. 2016 Jun;3(2):136-43. PMID: 27026358

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Perchlorate – Clinical Significance

- Binds to iodine
- Historically, potassium perchlorate was used to treat hyperthyroidism
- Human clinical research inconsistent
  - 59% of studies show clinical impact
- Inverse correlation thyroid hormone levels and perchlorate
  - But not reflected in hypothyroidism diagnosis
  - Positive correlation with TSH (women >> men)
- Dependent upon iodine status and goitrogen exposure

Niziński P, Błażewicz A, Kończyk J, Michalski R. Perchlorate - properties, toxicity and human health effects: an updated review. Rev Environ Health. 2020 Sep 4;36(2):199-222. PMID: 32887207.

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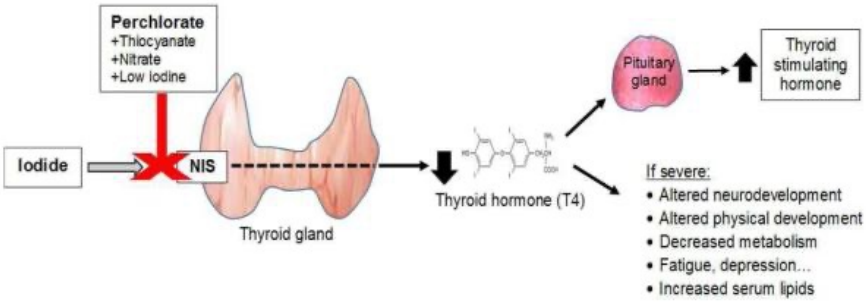
Seeing the Whole Picture



Steinmaus CM. Perchlorate in Water Supplies: Sources, Exposures, and Health Effects. Curr Environ Health Rep. 2016 Jun;3(2):136-43. PMID: 27026358

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Clinical Takeaways

Perchlorates

- Major disease: Decreased thyroid function → Decreased mitochondrial stimulation
- Primary sources: Water and food
- Best measure: Urinary
- Best intervention: Avoidance, optimize iodine intake, decrease goitrogen intake

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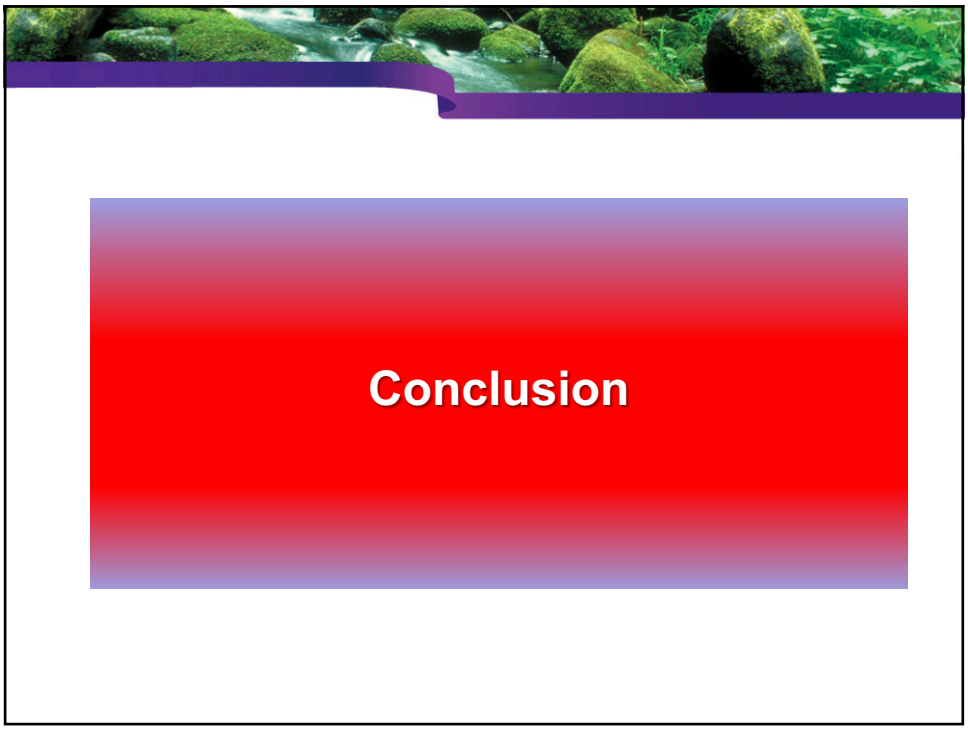
Conclusion

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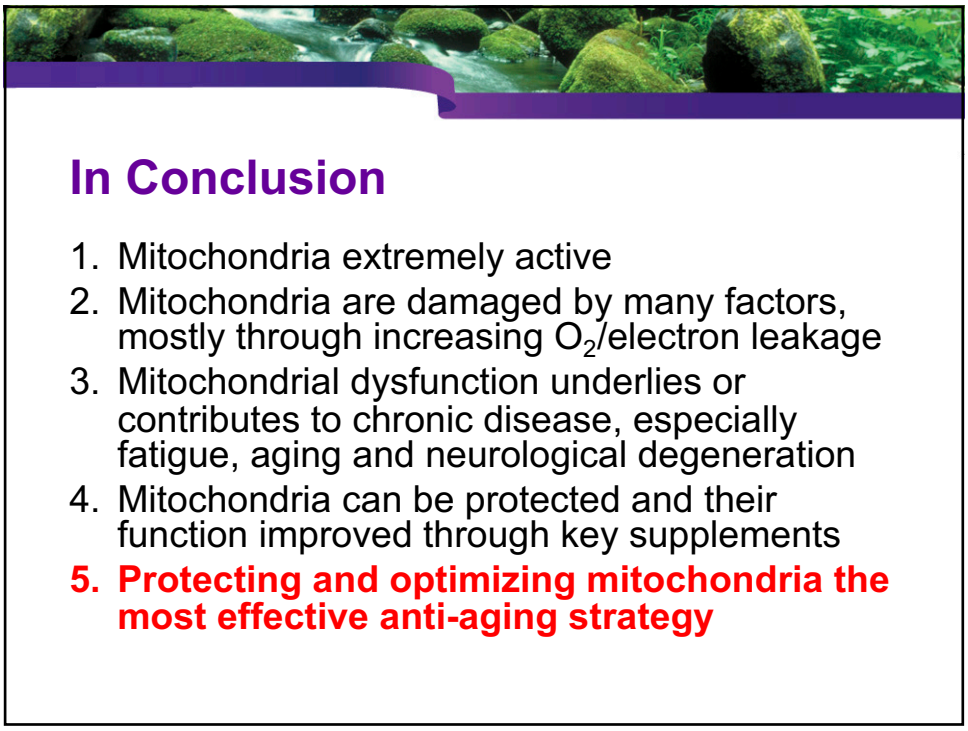
In Conclusion
1. Mitochondria extremely active
2. Mitochondria are damaged by many factors, mostly through increasing O_2/electron leakage
3. Mitochondrial dysfunction underlies or contributes to chronic disease, especially fatigue, aging and neurological degeneration
4. Mitochondria can be protected and their function improved through key supplements
5. Protecting and optimizing mitochondria the most effective anti-aging strategy

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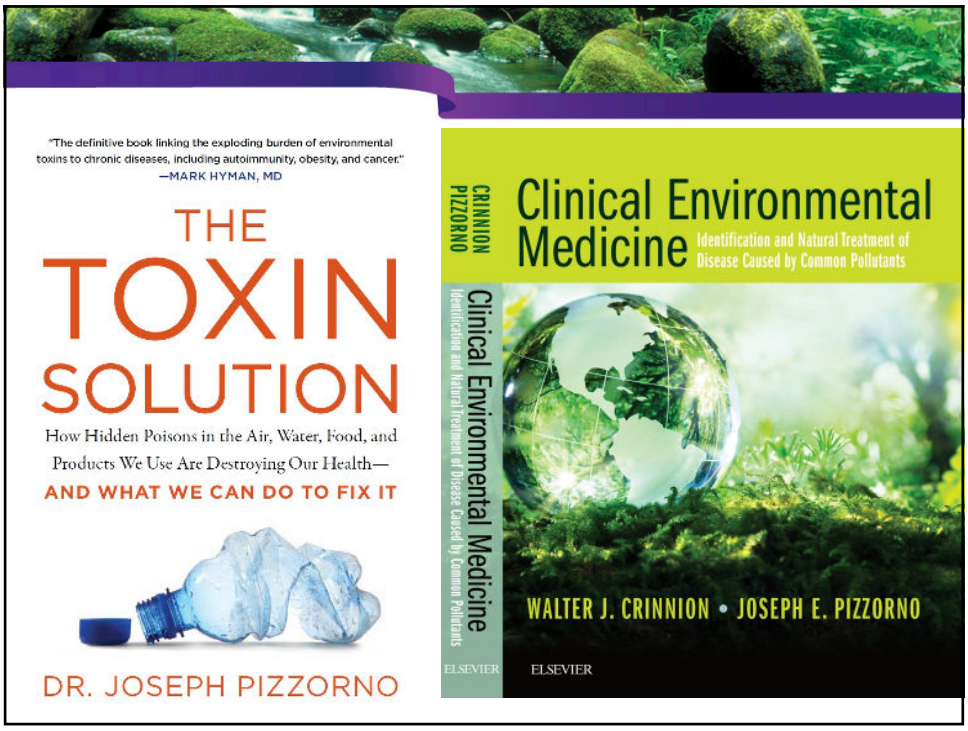
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—MARK HYMAN, MD
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How Hidden Poisons in the Air, Water, Food, and Products We Use Are Destroying Our Health—
AND WHAT WE CAN DO TO FIX IT
DR. JOSEPH PIZZORNO
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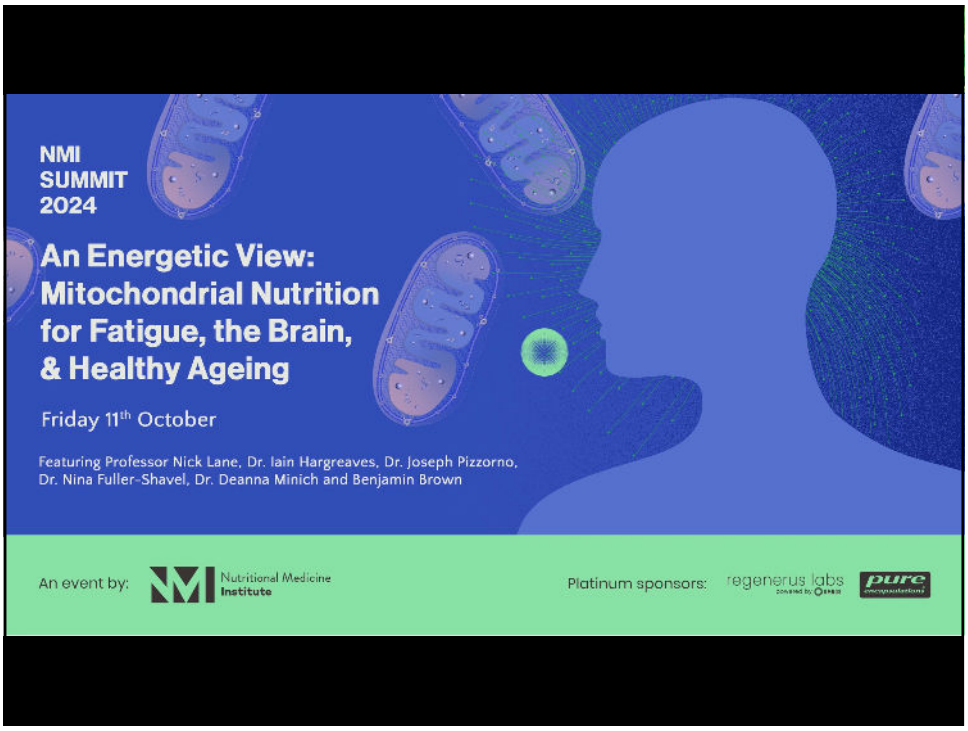
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