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Mitochondrial "Collateral Damage" Thanks to Big Pharma

Iatrogenic Drug and Vaccine-induced Mitochondrial Disorders

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"Mitochondrial damage is now understood to play a role in a wide range of seemingly unrelated disorders such as schizophrenia, diabetes, Parkinson's disease, chronic fatigue syndrome, and nonalcoholic steatohepatitis. Recently it has become known that iatrogenic (physician or treatment-caused) mitochondrial damage explains many adverse reactions from medications." — John Neustadt, MD and Steven Pieczenik, MD

"All classes of psychotropic drugs have been documented to damage mitochondria, as have statin medications, analgesics such as acetaminophen, and many others." – John Neustadt, MD and Steven Pieczenik, MD

Several years ago I attended a conference that was sponsored by the United Mitochondrial Disease Foundation (UMDF), an organization which seems to be a combination patient advocacy group and a funding organization for mitochondrial researchers.

The conference centered entirely upon the rare congenital/inherited forms of mitochondrial disorders that are first diagnosed in infancy and which comprise about 10 – 15 % of cases of known mitochondrial disorders.

Nothing was said by the presenters about the 85 – 90 % of acquired forms of mitochondrial disorders, which could, of course, be preventable if knowledge of the root causes were transmitted to us physicians and patients.

During the Q & A, a mitochondrial research scientist in the audience got up and talked about a colleague of his that had written an academic paper that identified 72 commonly-prescribed drugs that were mitochondrial poisons. He mentioned Pfizer's Lipitor and Zoloft as two examples. The author had not been able to get her paper published, and I have found no evidence that it was ever published. No comments were forthcoming from the UMDF expert that was leading the conference, and the discussion went back to the rare hereditary forms of the disease.

Being naturally suspicious of "experts" who may have professional or financial conflicts of interest, my curiosity was aroused; so I talked to the researcher who raised the obviously unwelcome question. He gave me his email address, but my several attempts to contact him by email failed to get any response. I later discovered that the researcher had at one time received research grants from Pfizer.

Ever since that suspicious episode I have maintained an interest in mitochondrial disorders, and since then I have discovered many articles in the basic science literature that have

dealt with drug and vaccine-induced mitochondrial disorders, none of which ever gets published in the mainstream medical journals, at least those that take advertising money from pharmaceutical companies.

Interestingly, UMDF has a convenient privacy policy that keeps it from revealing who are their donors, although five pharmaceutical or genetic testing companies (Reata, Transgenomic, Courtagen, Raptor and Stealth BioTherapeutics) have their logos displayed, but no discussion about acquired or iatrogenic mitochondrial disorders could be found on its website. I could find only one statement (on www.Mitoaction.org's website) about non-inherited mitochondrial disorders. It said that "Medicines or other toxic substances can trigger mitochondrial disease." No elaboration or links to more information were provided. I smelled a rat, and so should we all.

So this Duty to Warn column is about the multitude of common iatrogenic (drug- or doctor-caused) diseases that can be caused by the commonly prescribed drugs and/or commonly injected vaccine ingredients that are making many of us highly drugged, malnourished, environmentally-toxic and also thoroughly vaccinated. We Americans (infants, children, adolescents and adults) are among the sickest, most chronically-ill people in the developed world.

I include excerpts from just three examples from a multitude of peer-reviewed medical journal articles that have been trying to tell us clinicians (and our most aware patients) that there are many common, preventable disorders that the powers-that-be want us to believe are either the fault of the patient-victim ("shame-on-you") or are simply inherited from our guilty parents (and thus neither preventable nor curable).

Many of these disorders (see list below) are actually caused by prescription drugs, vaccines and/or other toxic chemicals that are poisoning the mitochondria in our brains, nerves, muscles and other organs. Thus we are being afflicted by preventable, iatrogenic- or industry-caused diseases. Both realities are taboo subjects in the current era of mind-control by America's powerful, profit-motivated, multinational corporations in BigPharma, BigChemical, BigMedicine, BigMedia, BigFood and BigAgribusiness industries. That pervasive group prefers our ignorance, and each of them spends unlimited amounts of money to ensure it.

The avarice of these industries for larger market-share, higher share price, bigger profits, lower wages and more aggressive wealth extraction knows no bounds, and their brain-disabling products makes their goals ever easier to attain.

The first excerpt below is about the injectable, toxic aluminum adjuvants that have been added to virtually all infant and adult vaccines for the past 70+ years There is no safe dose of aluminum or mercury, and neither have any nutritional value. (Aluminum is poorly absorbed when swallowed [0.5% absorption] but is 100% absorbed into the blood stream when injected.) The CDC/AAP (American Academy of Pediatrics)-mandated immunization schedule ensures that a total of nearly 5,000 micrograms of the mitochondrial toxin aluminum will be injected into the average American baby by the time he or she reaches 18 months (before which, by the way, is when many of the alleged "inherited" mitochondrial diseases become manifest)!

The second excerpt talks about how poisonous mercury is to the mitochondria that are in human brain, nerve, muscle and body cells. Over the last 20 years there have been at least

a hundred peer-reviewed medical journal articles that have been warning physicians about the neurotoxicity of mercury, the second-most toxic metal known to man (plutonium is first).

Mercury, in the form of Eli Lilly & Company's Thimerosal, has been in most infant and adult vaccines for several generations and was only removed from a number – but not all – of them when the AAP pleaded with the vaccine manufacturers to remove it from all vaccines because many concerned pediatricians were rightfully convinced that the rapidly escalating autism epidemic was at least partially caused by the rapidly escalating dosing of vaccines: and they were correct. But the neurotoxic aluminum, often given in multiple inoculations simultaneously, remained in the over-vaccination schedule, and the epidemic of chronic, autoimmune disorders among fully vaccinated children continued.

Nevertheless, the pharmaceutical companies, the CDC and the AAP continue to recommend annual (aluminum and mercury-containing) flu shots for immature, immune-vulnerable, brain-undeveloped babies as young as 6 months of age, and for their pregnant mothers! What could possibly go wrong? One must ask: who are the benefactors and who are the victims?

The third article below consist of extracts from a literature review of the subject of mitochondrial damage and the role of medications, chemicals, pesticides, metals, drugs, vaccine ingredients and other mitochondrial poisons that put every cell in our bodies at increased risk of permanent damage. It is titled "Medication-induced Mitochondrial Damage and Disease". Alarmingly, no mitochondrial patient advocacy website that I could find has links to this or any of the scores of articles that discuss acquired or iatrogenic mitochondrial disorders. Go figure.

1) Aluminum-induced Defective Mitochondrial Metabolism Perturbs Cytoskeletal Dynamics in Human Astrocytoma Cells

By J. Lemire, R. Mailloux, S. Puiseux-Dao, and V. D. Appanna

Published in the Journal of Neuroscience Research 87:1474–1483 (2009)

Posted at: http://onlinelibrary.wiley.com/doi/10.1002/jnr.21965/abstract

Abstract

Although aluminum (Al), a known environmental toxin, has been implicated in a variety of neurological disorders, the molecular mechanism responsible for these conditions is not fully understood. In this report, we demonstrate the ability of Al to trigger mitochondrial dysfunction and ineffective adenosine triphosphate (ATP) production. This situation severely affected cytoskeletal dynamics. Whereas the control cells had well-defined structures, the Al-exposed astrocytoma cells appeared as globular structures. Creatine kinase (CK) and profilin-2, two critical modulators of cellular morphology, were markedly diminished in the astrocytoma cells treated with Al. Antioxidants such as a-ketoglutarate and N-acetylcysteine (NAC) mitigated the occurrence of the globular-shaped cells promoted by Al toxicity. Taken together, these data reveal an intricate link between ATP metabolism and astrocytic dysfunction and provide molecular insights into the pathogenesis of Al-induced neurological diseases.

2) Thimerosal-Derived Ethylmercury Is a Mitochondrial Toxin in Human Astrocytes

By M. A. Sharpe, A. D. Livingston, and D. S. Baskin – Published online 6/28/2012 in the Journal of Toxicology, (posted at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3395253/)

Abstract

Thimerosal generates ethylmercury in aqueous solution and is widely used as a *(bactericidal)* preservative. We have investigated the toxicology of Thimerosal in normal human astrocytes, paying particular attention to mitochondrial function and the generation of specific oxidants. We find that ethylmercury not only inhibits mitochondrial respiration leading to a drop in the steady state membrane potential, but also concurrent with these phenomena increases the formation of superoxide, hydrogen peroxide, and Fenton/Haber-Weiss generated hydroxyl radical. These oxidants increase the levels of cellular aldehyde/ketones. Additionally, we find a five-fold increase in the levels of oxidant damaged mitochondrial DNA bases and increases in the levels of mtDNA nicks and blunt-ended breaks. Highly damaged mitochondria are characterized by having very low membrane potentials, increased superoxide/hydrogen peroxide production, and extensively damaged mtDNA and proteins. These mitochondria appear to have undergone a permeability transition, an observation supported by the five-fold increase in Caspase-3 activity observed after Thimerosal treatment.

Introduction

Thimerosal is a preservative that is widely used in medical products, including as a preservative in vaccines, immunoglobulin preparations, skin test antigens, antivenins, ophthalmic and nasal products, and tattoo inks, and is composed of 49.6 percent ethylmercury by weight. The widespread use of Thimerosal exposes many to its potential toxic effects, especially *in utero* and in neonates. We report the results of a series of experiments using cultured normal human astrocytes (NHA) exposed to Thimerosal to study the compound's effect on astrocyte mitochondria.

Oxidative Stress and Brain

The brain utilizes 20% of the oxygen consumed by the body but constitutes only 2% of the body's mass. <<snip>>

3) Medication-induced Mitochondrial Damage and Disease

By John Neustadt and Steve R. Pieczeni

Published in Molecular Nutrition and Food Research. 2008, 52, pp 780 – 788

This article is posted in its entirety at: http://psychrights.org/research/Digest/NLPs/DrugsCauseMitochondrialDamage.PDF

Abstract

Since the first mitochondrial dysfunction was described in the 1960s, the medicine has advanced in its understanding the role mitochondria play in health and disease. Damage to mitochondria is now understood to play a role in the pathogenesis of a wide range of seemingly unrelated disorders such as schizophrenia, bipolar disease, dementia, Alzheimer's disease, epilepsy, migraine headaches, strokes, neuropathic pain, Parkinson's disease, ataxia, transient ischemic attack, cardiomyopathy, coronary artery disease, chronic fatigue

syndrome, fibromyalgia, retinitis pigmentosa, diabetes, hepatitis C, and primary biliary cirrhosis.
Medications have now emerged as a major cause of mitochondrial damage, which may explain many adverse effects.
All classes of psychotropic drugs have been documented to damage mitochondria, as have statin medications, analgesics such as acetaminophen, and many others. While targeted nutrient therapies using antioxidants or their precursors (e. g., N-acetylcysteine [NAC]) hold promise for improving mitochondrial function, there are large gaps in our knowledge. The most rational approach is to understand the mechanisms underlying mitochondrial damage for specific medications and attempt to counteract their deleterious effects with nutritional therapies. This article reviews our basic understanding of how mitochondria function and how medications damage mitochondria to create their occasionally fatal adverse effects.

Introduction

Mitochondria are the powerhouses of our cells. They are responsible for generating energy… <<snip>> …mitochondria are the only other subcellular structure aside from the nucleus to contain DNA. However, unlike nuclear DNA (nDNA), mitochondrial DNA (mtDNA) are not protected by histones. nDNA wraps around histones, which then physically shield the DNA from damaging free radicals and are also required to repair DNA breaks. Since mtDNA lacks the structural protection of histones and their repair mechanisms, they are quite susceptible to damage. <<snip>>

Mitochondria Structure and Function

Cellular energy requirements control how many mitochondria are in each cell. A single somatic cell can contain from 200 to 2000 mitochondria, while human germ cells such as spermatozoa contain a fixed number of 16 mitochondria and oocytes have up to 100 000. The largest number of mitochondria are found in the most metabolically active cells, such as skeletal and cardiac muscle and the liver and brain. Mitochondria are found in every human cell except mature erythrocytes (red blood cells).

Acquired Conditions in which Mitochondrial Dysfunction has been Implicated (as of 2007

- Diabetes
- Huntington’s disease
- Cancer including hepatitis-C virus-associated hepatocarcinogenesis
- Alzheimer disease
- Parkinson’s disease
- Bipolar disorder
- Schizophrenia
- Aging and senescence
- Anxiety disorders

Nonalcoholic steatohepatitis (NASH – late stage of nonalcoholic fatty infiltration of the liver)

Cardiovascular disease, including atherosclerosis

Sarcopenia (muscle-wasting disease, mainly of the elderly)

Exercise intolerance

Fatigue, including chronic fatigue syndrome, fibromyalgia, and myofascial pain

Medications Documented to Induce Mitochondrial Damage (as of 2007)

http://psychrights.org/research/Digest/NLPs/DrugsCauseMitochondrialDamage.PDF

Alcoholism medications Ex: Antabuse

Alzheimer's dementia drugs Ex: Tacrine (Cognex), Galantamine

Analgesics (for pain) and anti-inflammatory drugs, Ex: Aspirin, acetaminophen (Tylenol), indomethacin, Naproxen

Anesthetics Ex: lidocaine, propofol (also general anesthetics likehalothane. isoflurane, sevoflurane)

Angina medications Ex: amiodarone

Antiarrhythmic (regulates heartbeat) Ex: amiodarone (also beta blockers)

Antibiotics Ex: tetracycline (also chloramphenicol, Cipro)

Antidepressants Ex: amitriptyline, citalopram (Celexa), fluoxetine (Prozac, Symbyax, Sarafem)

Antipsychotics Ex: chlorpromazine, fluphenazine, haloperidol, risperidone, quetiapine, clozapine, olanzapine

Anxiety medications Ex: (Every benzodiazepine), including alprazolam (Xanax), diazepam (valium)

Barbiturates Ex: amobarbital, phenobarbital, pentobarbital, , propofol, secobarbital

Cholesterol-lowering medications Ex: All statins – atorvastatin, fluvastatin, lovastatin, pravastatin, rosuvastatin (Crestor), simvastatin, cholestyramine, clofibrate (Atromid-S)

Cancer (chemotherapy) medications Ex: Mitomycin C, profiromycin, adriamycin

Diabetes medications Ex: metformin, Glucophage, troglitazone, rosiglitazone, buformin

HIV/AIDS medications Ex: (AZT, zidovudine)

Epilepsy/Seizure medications Ex: valproic acid (Depakene, depakote, divalproex sodium)

Mood stabilizers Ex: lithium

Parkinson's disease medications

Vaccine Ingredients Ex: Mercury, aluminum, ethylene glycol

Mechanisms of Mitochondria-induced Injury

Damage to mitochondria is caused primarily by reactive oxygen species (ROS) generated by the mitochondria themselves. <<snip>>

As a medical concern, hyperglycemia induces mitochondrial superoxide production by endothelial cells, which is an important mediator of diabetic complications such as cardiovascular disease. Endothelial superoxide production also contributes to atherosclerosis, hypertension, heart failure, aging, sepsis, ischemia-reperfusion injury, and hypercholesterolemia. Inflammatory mediators such as tumor necrosis factor alpha (TNF-a) have been associated in vitro with mitochondrial dysfunction and increased ROS generation. <<snip>>

Vitamins, minerals, and other metabolites act as necessary cofactors for the synthesis and function of mitochondrial enzymes and other compounds that support mitochondrial function, and diets deficient in micronutrients can accelerate mitochondrial decay and contribute to neurodegeneration. For example, enzymes in the pathway for hemoglobin synthesis require adequate amounts of pyridoxine, iron, copper, zinc, and riboflavin. Deficiencies of any component of the TCA cycle or ETC can lead to increased production of free radicals and mtDNA damage. For example, low iron status decreases mitochondrial activity by causing a loss of complex IV and increasing oxidative stress.

Medication-induced Mitochondrial Damage

Mitochondrial dysfunction is increasingly implicated in the etiology of drug-induced toxicities, but mitochondrial toxicity testing is still not required by the US FDA for drug approval. Mitochondria can be damaged both directly and indirectly by medications.

Conclusions

Since the first mitochondrial dysfunction was described in the 1960s, the central role mitochondria play in health and disease has been widely documented. Mitochondrial damage is now understood to play a role in a wide range of seemingly unrelated disorders such as schizophrenia, diabetes, Parkinson's disease, chronic fatigue syndrome, and nonalcoholic steatohepatitis (late-stage fatty infiltration of the liver).

Recently it has become known that iatrogenic mitochondrial damage explains many adverse reactions from medications. Mitochondrial toxicity testing as part of the preapproval process for medications may help protect the public by identifying the most toxic medications before they are allowed to reach the market. By understanding the mechanisms underlying drug-induced mitochondrial damage, it may be possible to develop nutritional strategies to decrease the potentially toxic effects of medications.

While targeted nutrient therapies using antioxidants or their precursors (e. g., N-acetylcysteine [NAC]) holds promise for improving mitochondrial function, there are large gaps in our knowledge. The most rational approach is to understand the mechanisms

underlying mitochondrial damage for specific medications, and attempt to counteract their deleterious effects with nutritional therapies. While randomized, controlled trials are lacking in this regard, they hopefully will be designed and conducted in coming years so that clinicians will have a clearer understanding of how to best protect and treat their patients.

Dr Kohls is a retired physician who practiced holistic mental health care for the last decade of his career. Virtually all of his patients exhibited iatrogenic (prescription drug-related) syndromes such as are mentioned in the article above. In retrospect, those patients were actually manifesting iatrogenic mitochondrial diseases. His practice mainly consisted of helping his patients, through brain nutrient therapy, psycho-educational psychotherapy and the gradual reduction or elimination of the psychotropic medications that were sickening them. He now writes a weekly column for the Reader Weekly, an alternative newsweekly published in Duluth, Minnesota, USA. Many of Dr Kohls' columns are archived at http://duluthreader.com/articles/categories/200_Duty_to_Warn.
