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Greg Macpherson, BPharm
CEO/President

MITOCHONDRIA AND MITOQ – A RESEARCH UPDATE

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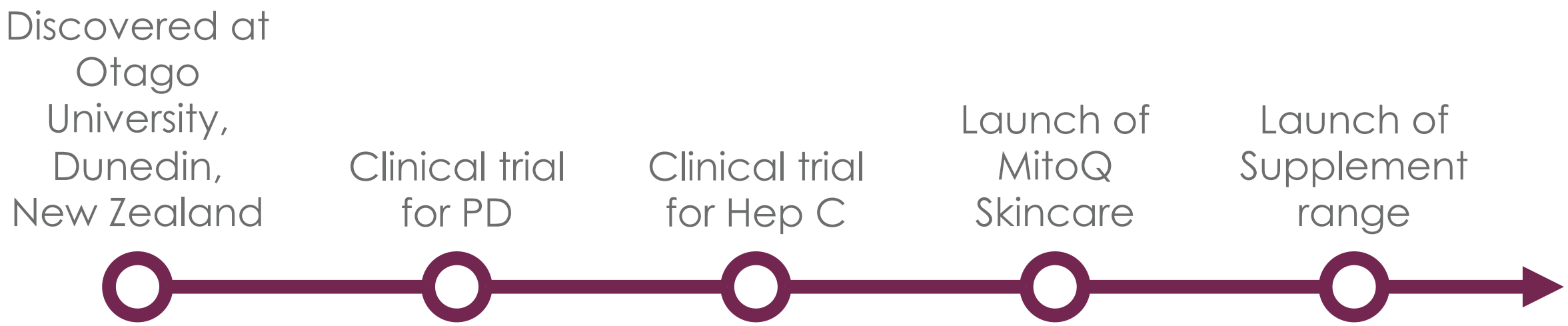

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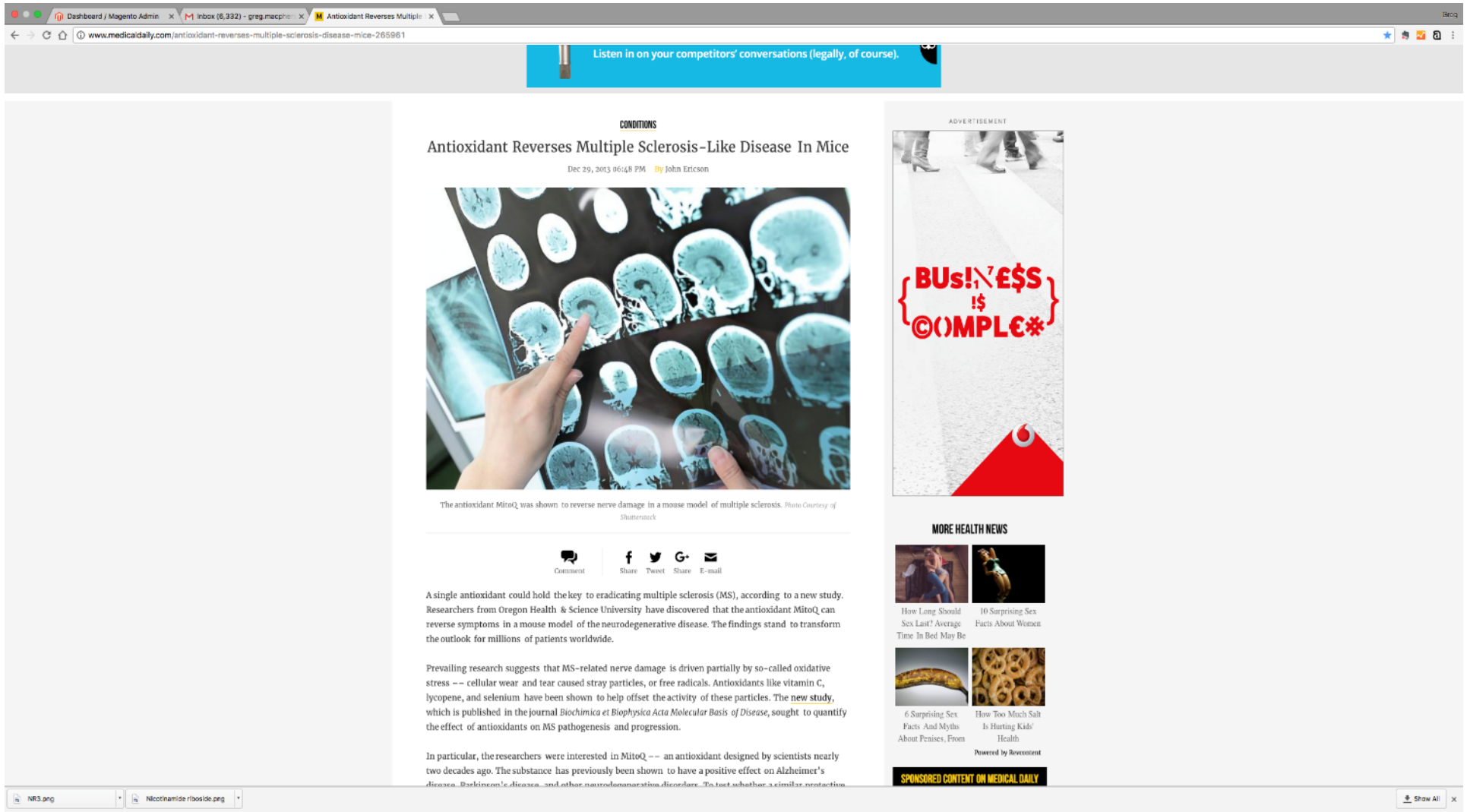

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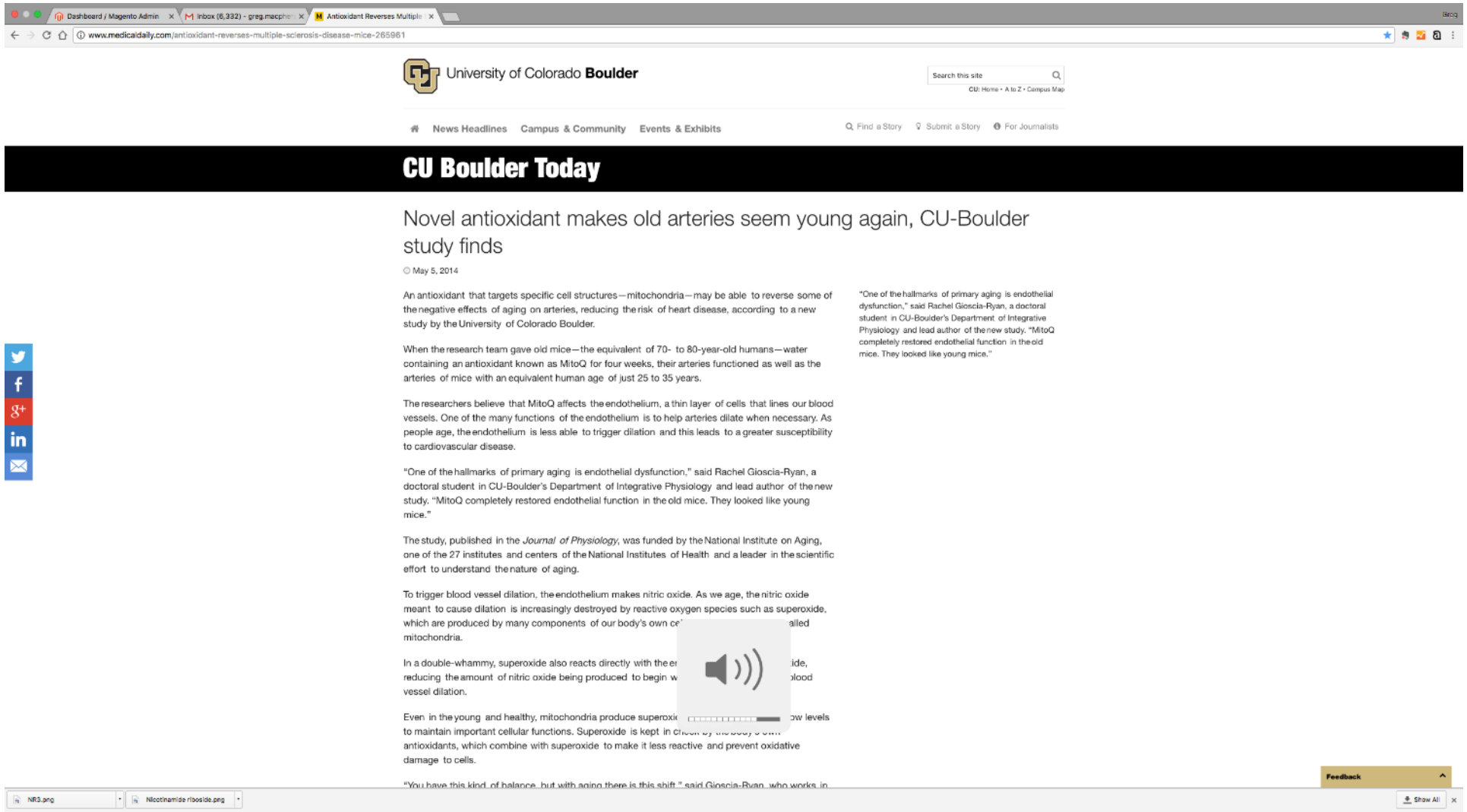

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CU Boulder Today

Novel antioxidant makes old arteries seem young again, CU-Boulder study finds

May 5, 2014

An antioxidant that targets specific cell structures—mitochondria—may be able to reverse some of the negative effects of aging on arteries, reducing the risk of heart disease, according to a new study by the University of Colorado Boulder.

When the research team gave old mice—the equivalent of 70- to 80-year-old humans—water containing an antioxidant known as MitoQ for four weeks, their arteries functioned as well as the arteries of mice with an equivalent human age of just 25 to 35 years.

The researchers believe that MitoQ affects the endothelium, a thin layer of cells that lines our blood vessels. One of the many functions of the endothelium is to help arteries dilate when necessary. As people age, the endothelium is less able to trigger dilation and this leads to a greater susceptibility to cardiovascular disease.

"One of the hallmarks of primary aging is endothelial dysfunction," said Rachel Gioscia-Ryan, a doctoral student in CU-Boulder's Department of Integrative Physiology and lead author of the new study. "MitoQ completely restored endothelial function in the old mice. They looked like young mice."

The study, published in the *Journal of Physiology*, was funded by the National Institute on Aging, one of the 27 institutes and centers of the National Institutes of Health and a leader in the scientific effort to understand the nature of aging.

To trigger blood vessel dilation, the endothelium makes nitric oxide. As we age, the nitric oxide meant to cause dilation is increasingly destroyed by reactive oxygen species such as superoxide, which are produced by many components of our body's own ce alled mitochondria.

In a double-whammy, superoxide also reacts directly with the e ide, reducing the amount of nitric oxide being produced to begin w lood vessel dilation.

Even in the young and healthy, mitochondria produce superoxi ow levels to maintain important cellular functions. Superoxide is kept in c antioxidants, which combine with superoxide to make it less reactive and prevent oxidative damage to cells.

"You have this kind of balance, but with aging there is this shift," said Gioscia-Ryan, who works in

"One of the hallmarks of primary aging is endothelial dysfunction," said Rachel Gioscia-Ryan, a doctoral student in CU-Boulder's Department of Integrative Physiology and lead author of the new study. "MitoQ completely restored endothelial function in the old mice. They looked like young mice."

Feedback

NR3.png | Nicotinamide riboside.png | Show All

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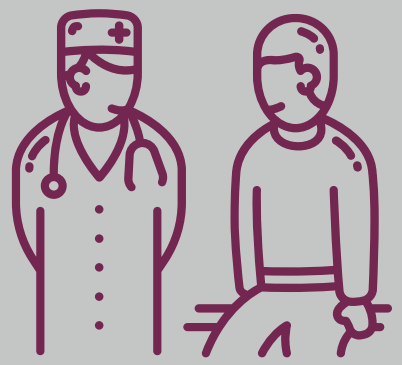

200,000
patient months experience

100+
countries

FDA

SA GRAS

12
SKUs and growing

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Company Mission

Our mission is to raise awareness of mitochondria and the link between optimal mitochondria function health and longevity

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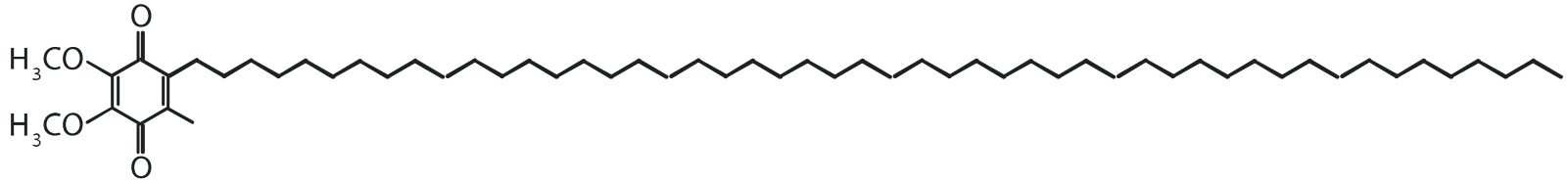
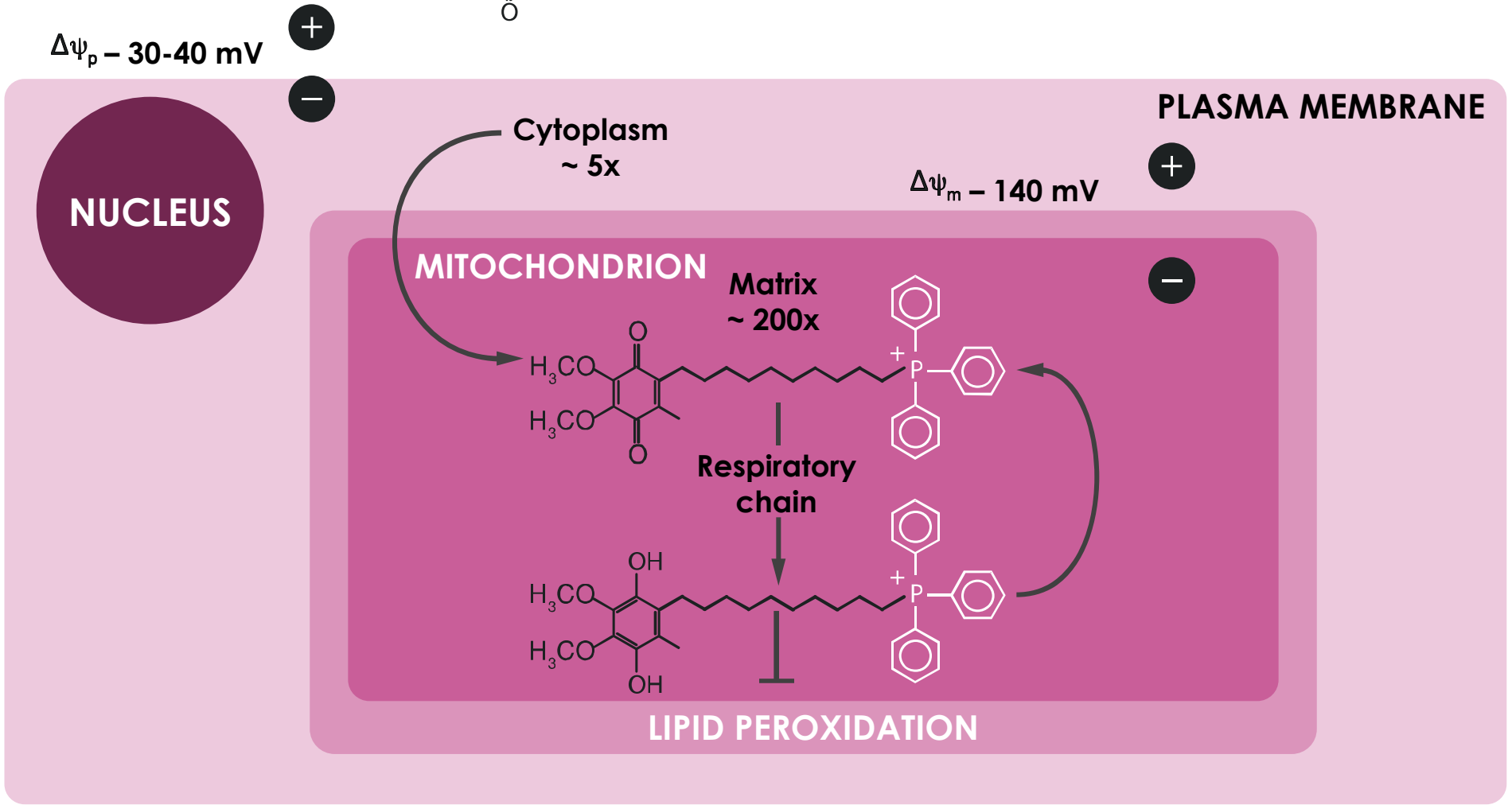
What is MitoQ?





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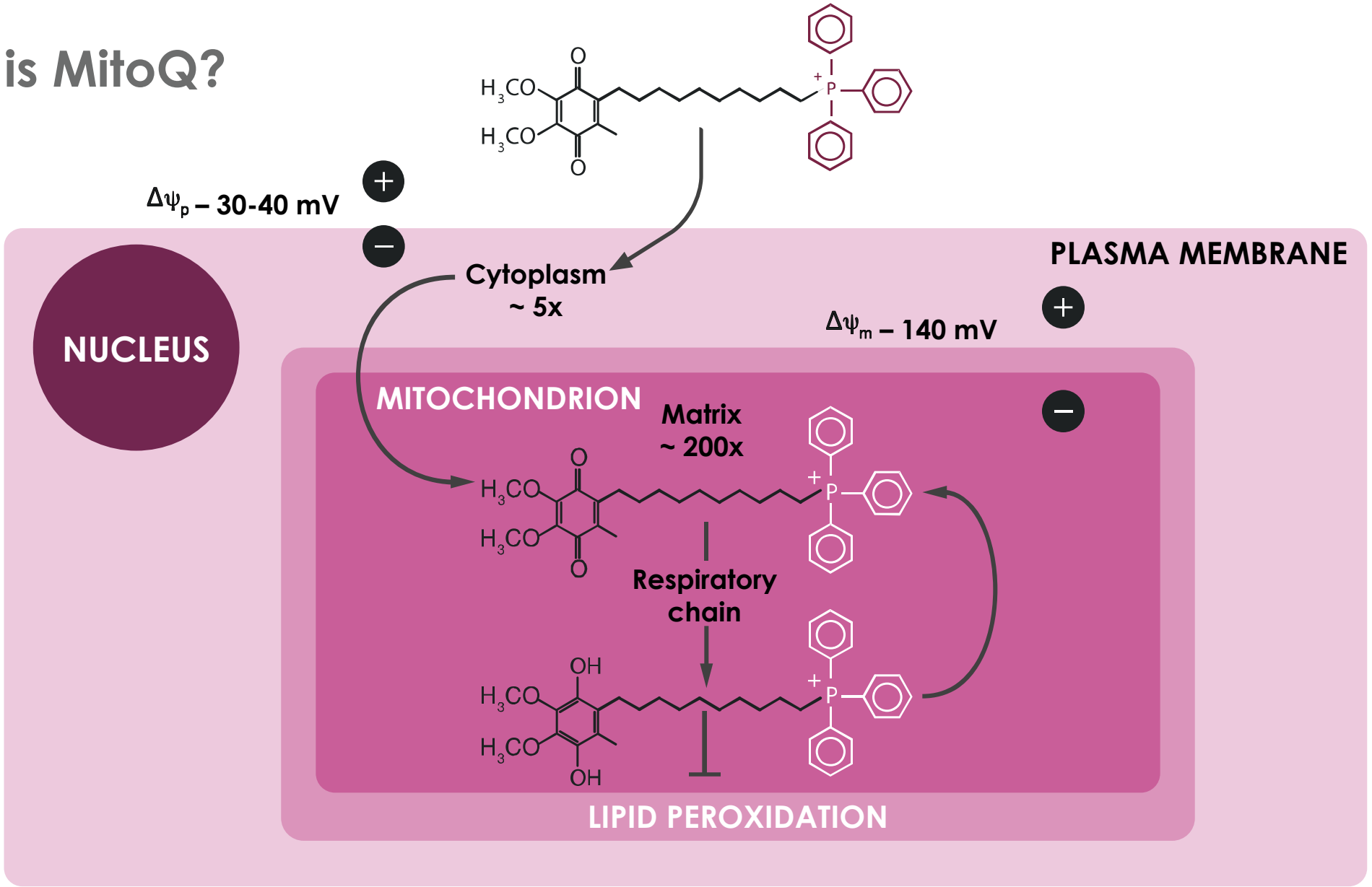
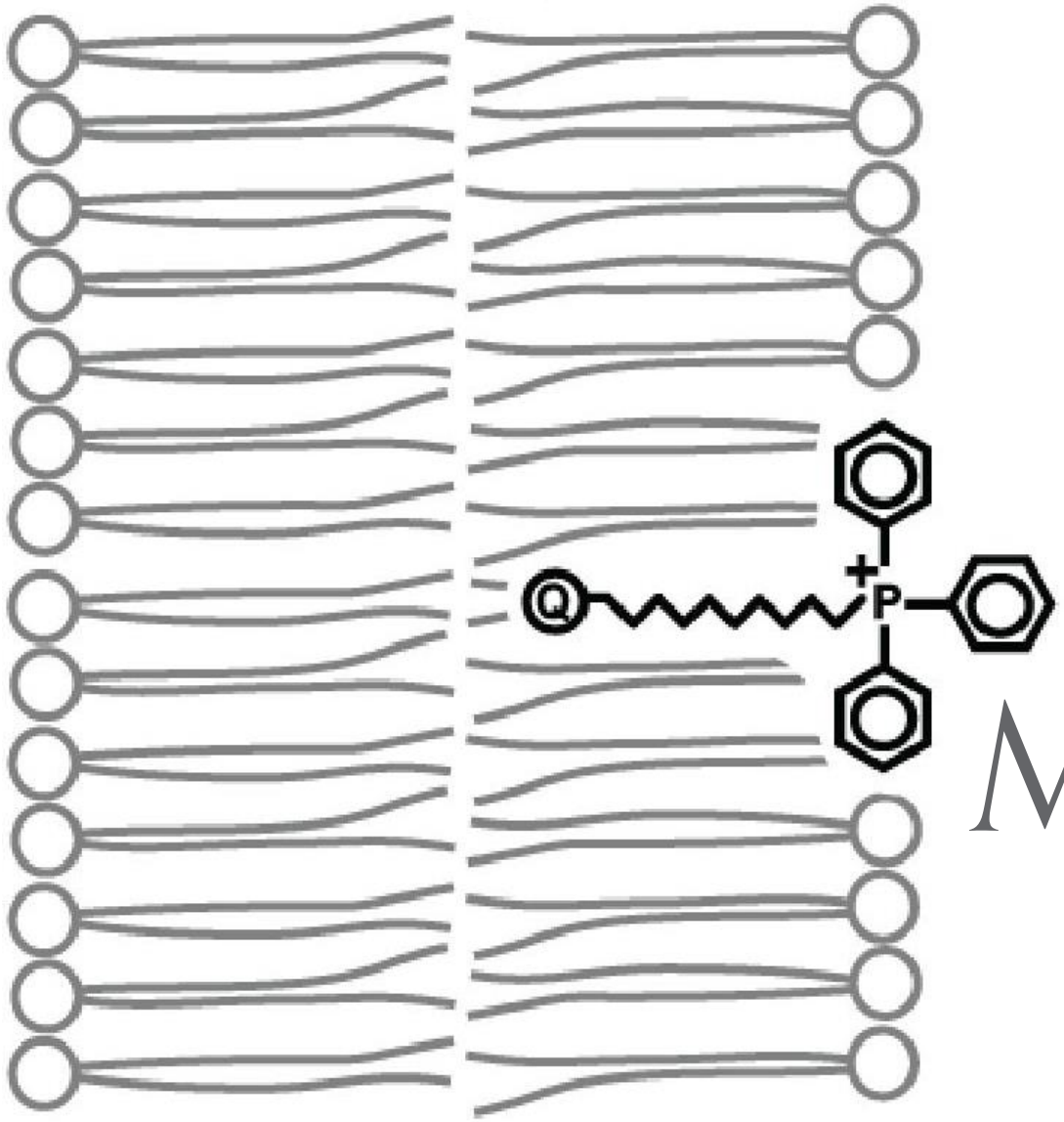


Mitochondrial Membrane



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What happens in the inner mitochondrial membrane?

Electron transport chain
NADH dehydrogenase (ubiquinone)
Electron-transferring-flavoprotein dehydrogenase
Electron-transferring flavoprotein
Succinate dehydrogenase
Alternative oxidase
Cytochrome bc1 complex
Cytochrome c
Cytochrome c oxidase
F-ATPase
ATP–ADP translocase
ATP-binding cassette transporter
Cholesterol side-chain cleavage enzyme
Protein tyrosine phosphatase
Carnitine O-palmitoyltransferase
Carnitine O-acetyltransferase
Carnitine O-octanoyltransferase
Cytochrome P450
Translocase of the inner membrane
Glutamate aspartate transporter
Pyrimidine metabolism
Dihydroorotate dehydrogenase
Thymidylate synthase (FAD)
HtrA serine peptidase 2
Adrenodoxin reductase
Heme biosynthesis
Protoporphyrinogen oxidase
Ferrochelatase
Uncoupling protein

What makes MitoQ Different?

ONE

Mitochondria -targeted antioxidant

TWO

Selective vs Broad Spectrum

THREE

Recycling antioxidant

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Why is this important?

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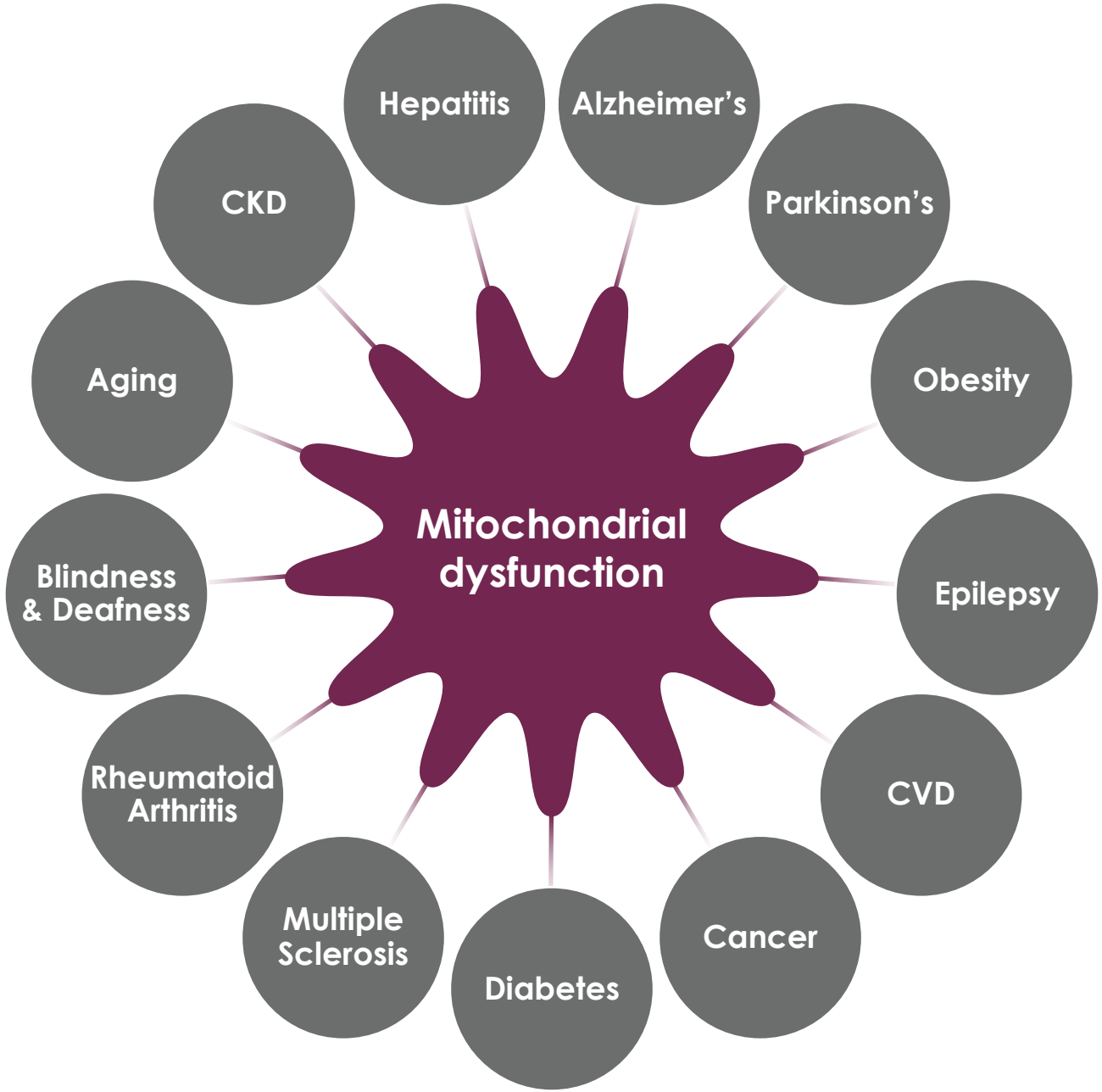

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MitoQ Research

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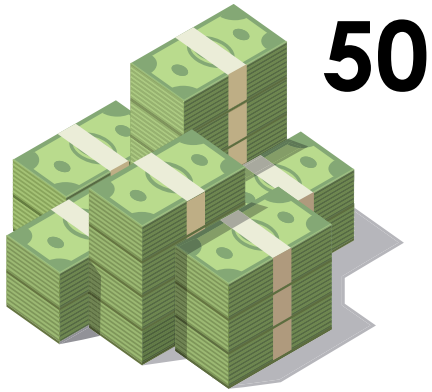
Research



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NIA funded Interventions Testing Program

NIH National Institute on Aging

Clinical Research underway


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Mitochondrial Research

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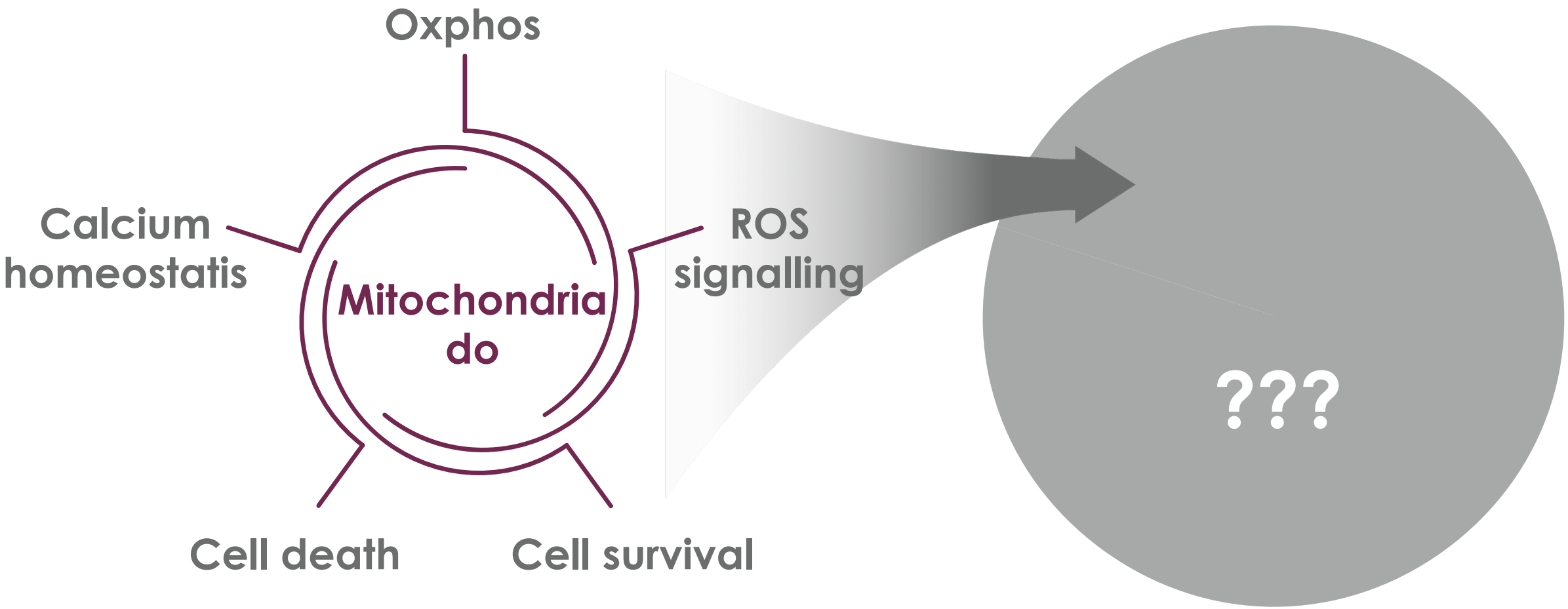
Mitochondrial research is only starting



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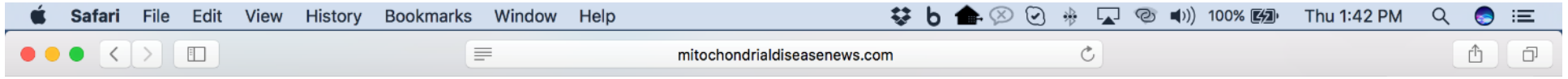

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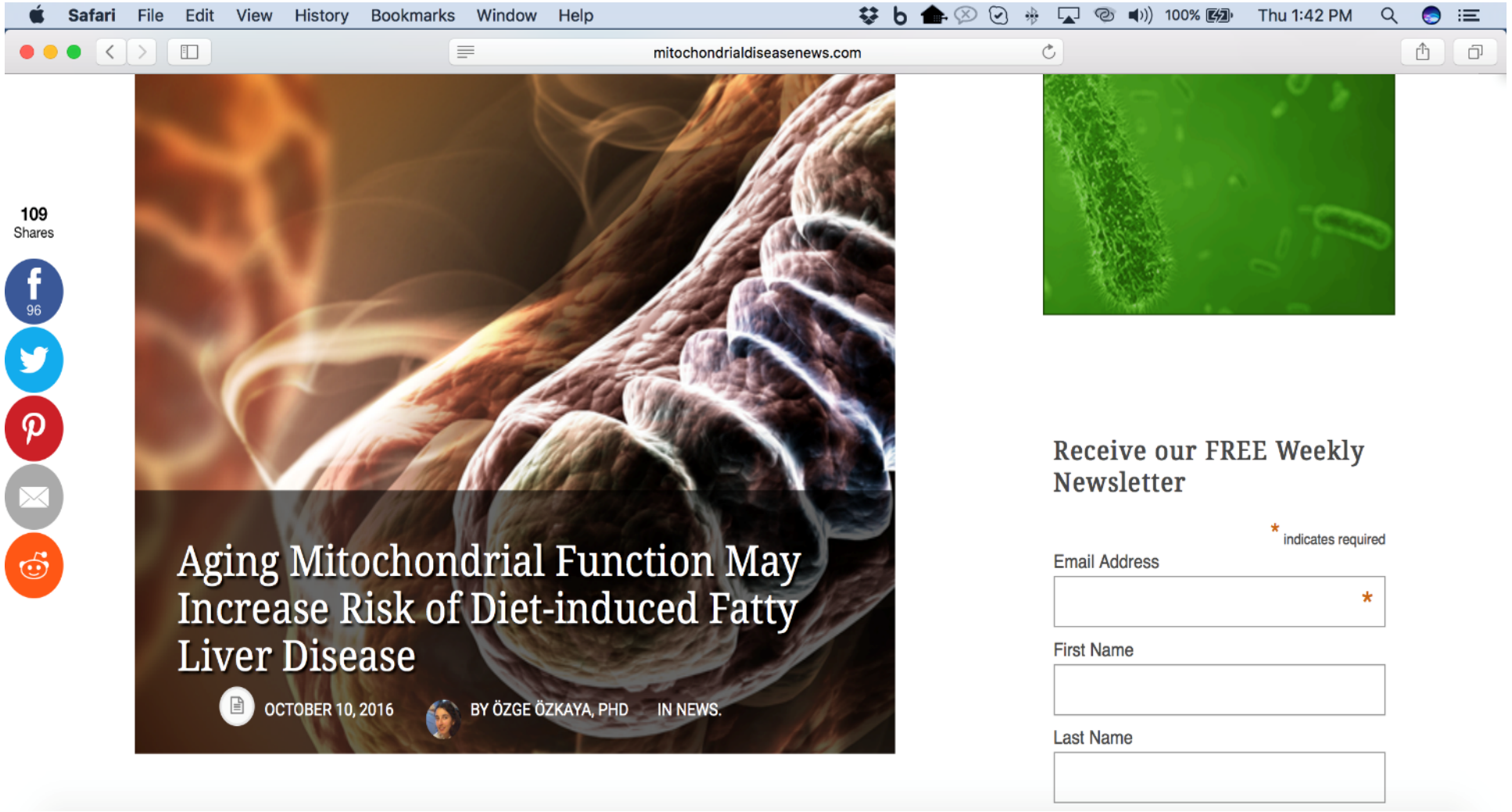
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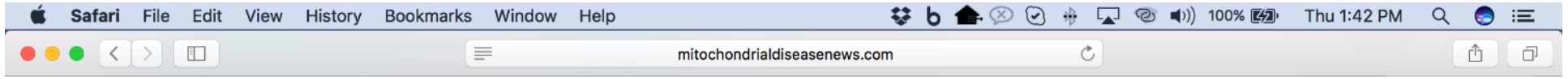

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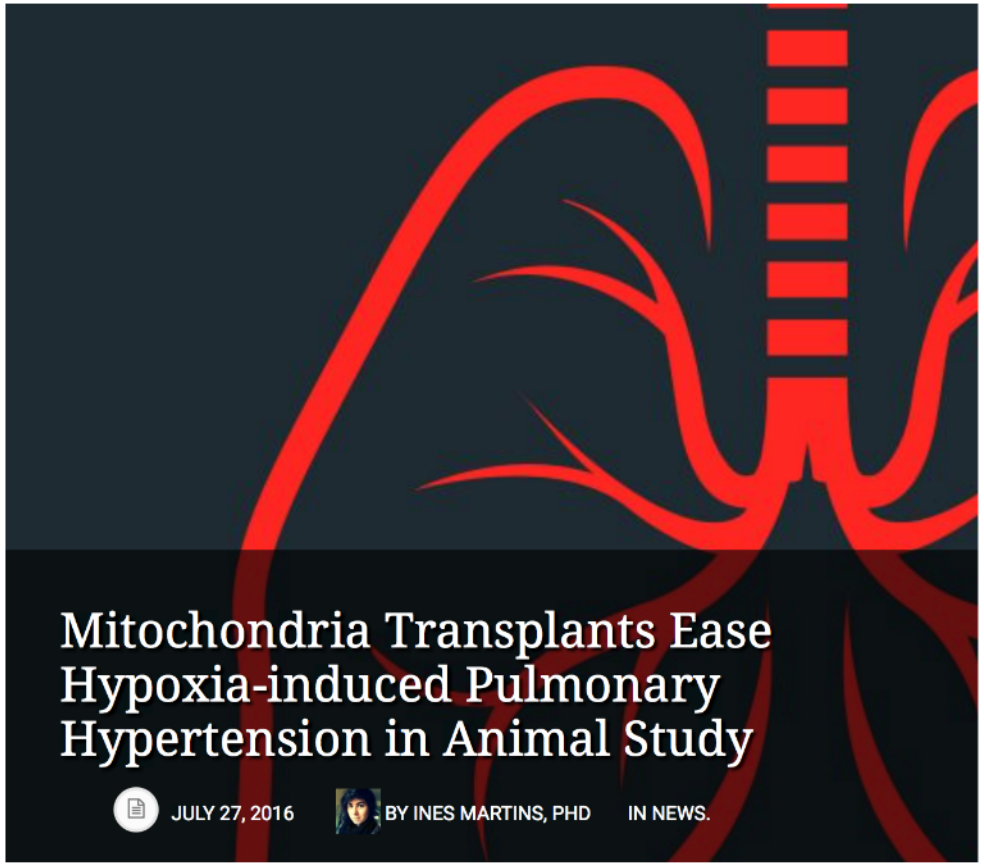

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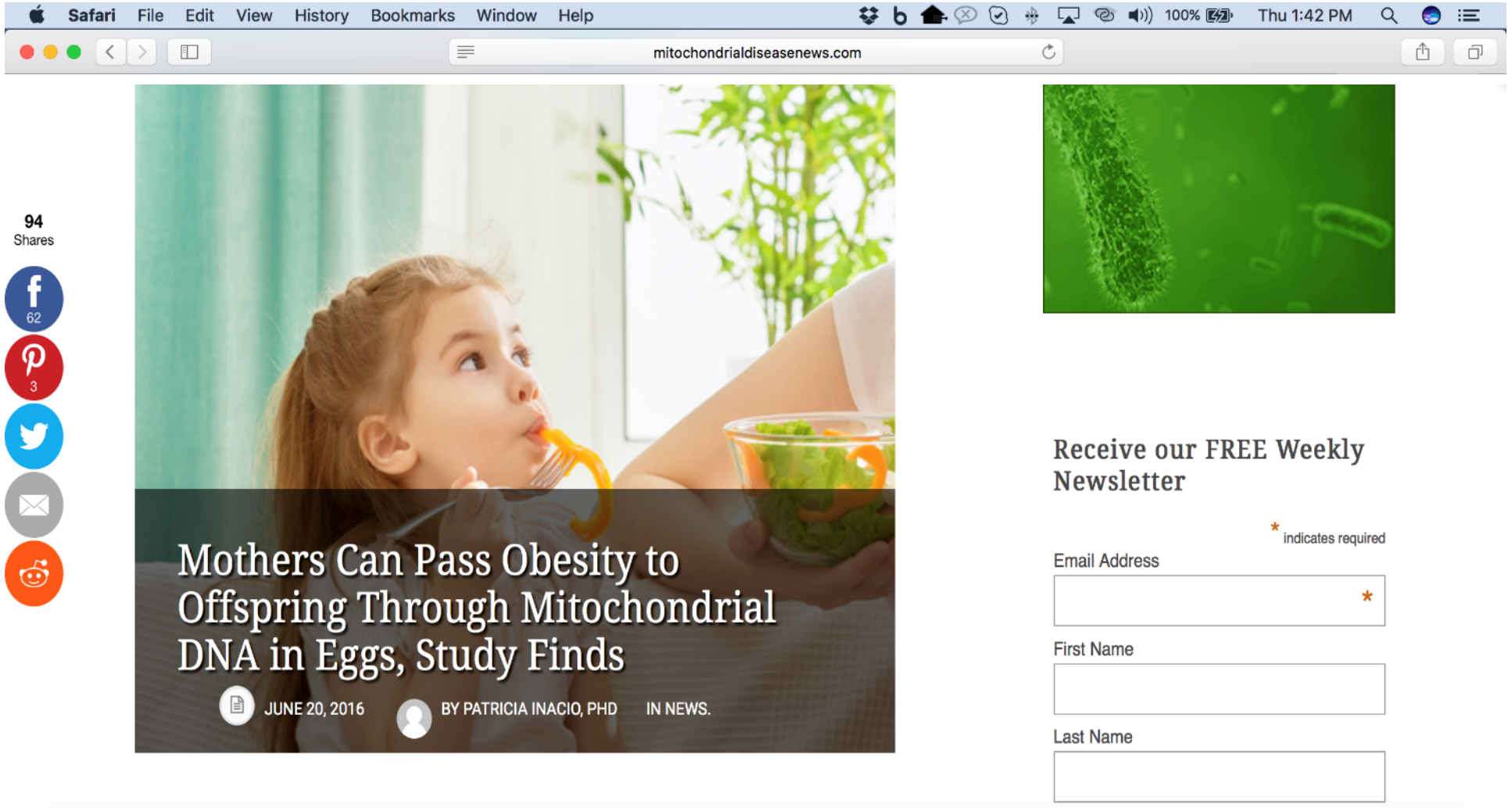
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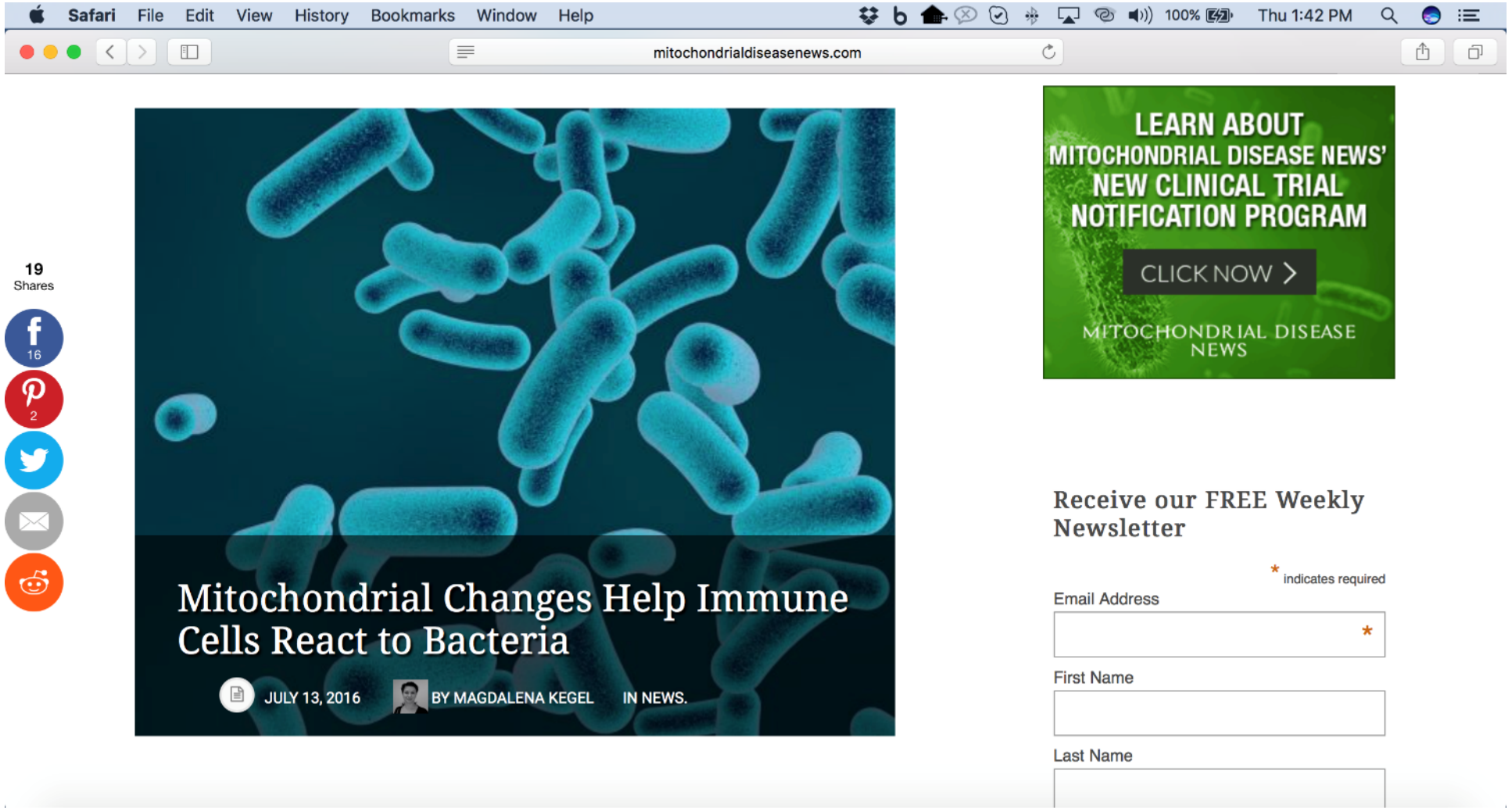
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CLINICAL IMPLICATIONS OF BASIC RESEARCH

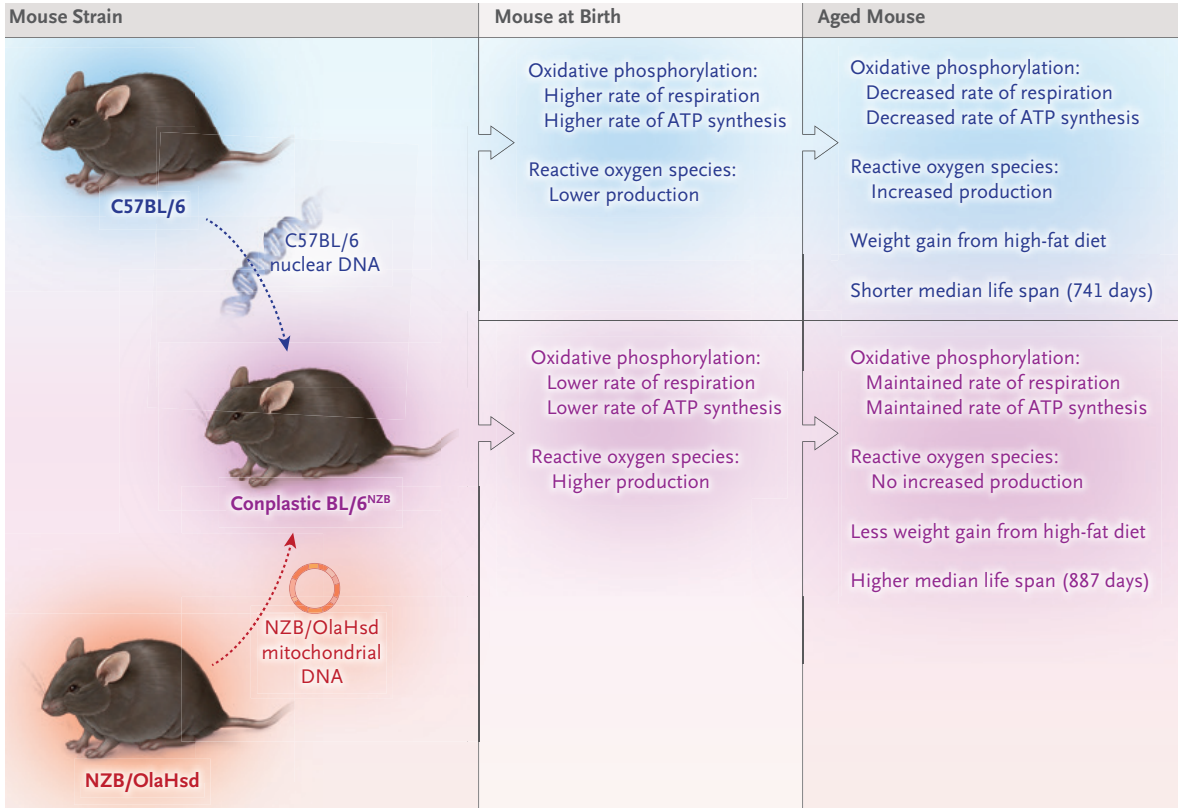
Elizabeth G. Phimister, Ph.D., *Editor*

Mitochondrial Matchmaking

Patrick F. Chinnery, M.B., B.S., Ph.D., and Massimo Zeviani, M.D., Ph.D.

Although conveniently described as the "batteries of the cell" (and, consistent with this analogy, amenable to exchange), mitochondria are complex cellular organelles assembled from proteins encoded by two distinct genomes: nuclear chromosomal DNA and the mitochondrial genome

oxygen species.[2] These observations provided a partial explanation for earlier studies in conplastic mice (i.e., mice in which the nuclear genome from one inbred strain is backcrossed into the cytoplasm of another inbred strain, with the cytoplasmic source always being the female par-



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Reversing aging: Cellular damage could be reversed by activating genes in mitochondria

Lori Dajose | November 22, 2016 | DeepStuff

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Researchers from Caltech and UCLA have developed a new approach to removing cellular damage that accumulates with age. The technique can potentially help slow or reverse an important cause of aging.

Led by Nikolay Kandul, senior postdoctoral scholar in biology and biological engineering in the laboratory of Professor of Biology Bruce Hay, the team developed a technique to remove mutated DNA from mitochondria....

There are hundreds to thousands of mitochondria per cell, each of which carries its own small circular DNA genome, called mtDNA,...The accumulation of mutant mtDNA over a lifetime is thought to contribute

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Fight Aging!

Do you want to live a longer life in good health? Simple practices can make some difference, such as exercise or calorie restriction. But over the long haul all that really matters is progress in medicine: building new classes of therapy to repair and reverse the known root causes of aging. The sooner these treatments arrive, the more lives will be saved. Find out how to help »

28 NOV 2016

Improved Quality Control of Protein Folding Extends Life in Nematode Worms

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In the paper I'll point out today, researchers map an efficient form of protein quality control from stem cells and recreate it in somatic cells, producing extended life in nematode worms as a result. Proteins are large, complex molecules, and their correct function depends on the assumption of a precise three-dimensional arrangement after creation, a process known as protein folding. Proteins can and do misfold, however, and in doing so many become actively harmful rather than merely unwanted clutter. A baroque system of chaperone proteins assists in correct folding, as well as identification and removal of misfolded molecules. The presence of misfolded proteins is effectively a form of damage: some of the molecular waste that accumulates with age and contributes to the development of age-related disease consists of misfolded proteins, such as the various forms of amyloid, for example. The gradual failure of cellular recycling systems, such as declining lysosomal function caused by the presence of metabolic waste that is hard for the body to break down, or similar failures in the proteasome, also contribute to rising levels of damaged and dysfunctional proteins. Since aging is nothing more than the accumulation of damage and the reactions to that damage, more efficient operation of chaperone and other quality control systems in cells should slow aging: the less damage there is at any one time, the less of an

First Steps

Read an Introduction to Living Longer
Read the Fight Aging! FAQ
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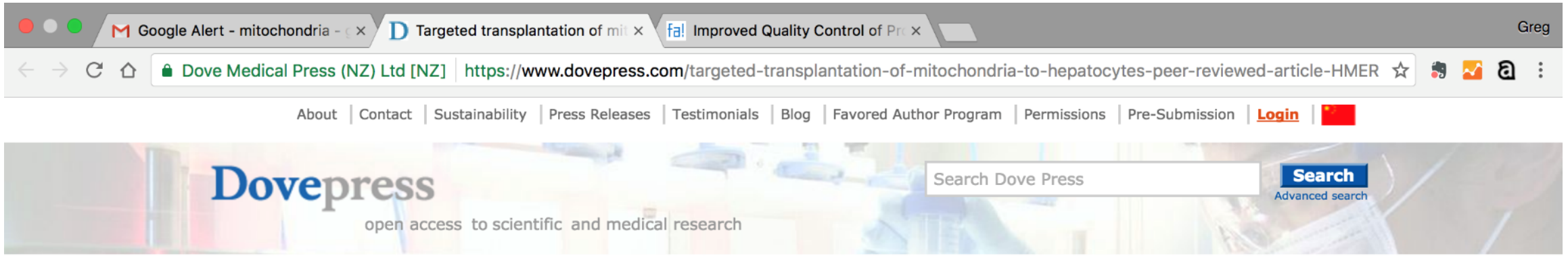
Help Researchers in Other Ways

The Root Causes of Aging

Aging is Caused by Damage
Accumulating Cross-Links
Buildup of Amyloid Between Cells



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ORIGINAL RESEARCH NOTEWORTHY

Targeted transplantation of mitochondria to hepatocytes

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Authors Gupta N, Wu CH, Wu GY

Received 10 July 2016

Accepted for publication 24 September 2016

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Peer reviewer comments 3

Editor who approved publication: Dr Gerry Lake-Bakaar

November 28, 2016

Researchers remove mutated DNA from mitochondria to slow or reverse a cause of aging

aging, antiaging, DNA, life extension, longevity, medicine, mitochondria, science

Researchers from Caltech and UCLA have developed a new approach to removing cellular damage that accumulates with age. The technique can potentially help slow or reverse an important cause of aging.

Led by Nikolay Kandul, senior postdoctoral scholar in biology and biological engineering in the laboratory of Professor of Biology Bruce Hay, the team developed a technique to remove mutated DNA from mitochondria, the small organelles that produce most of the chemical energy within a cell. A paper describing the research appears in the November 14 issue of Nature Communications.

There are hundreds to thousands of mitochondria per cell, each of which carries its own small circular DNA genome, called mtDNA, the products of which are required for energy production. Because mtDNA has limited repair abilities, normal and mutant versions of mtDNA are often found in the same cell, a condition known as heteroplasmy. Most people start off life with some level of heteroplasmy, and the levels of mutant mtDNA increase throughout life. When a critical threshold level of mutant mtDNA is passed, cells become nonfunctional or die.

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Human Research

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Mendus Trial

- Fibromyalgia and Chronic Fatigue Syndrome
- 12 week 3 arm blinded crossover trial

Key results:

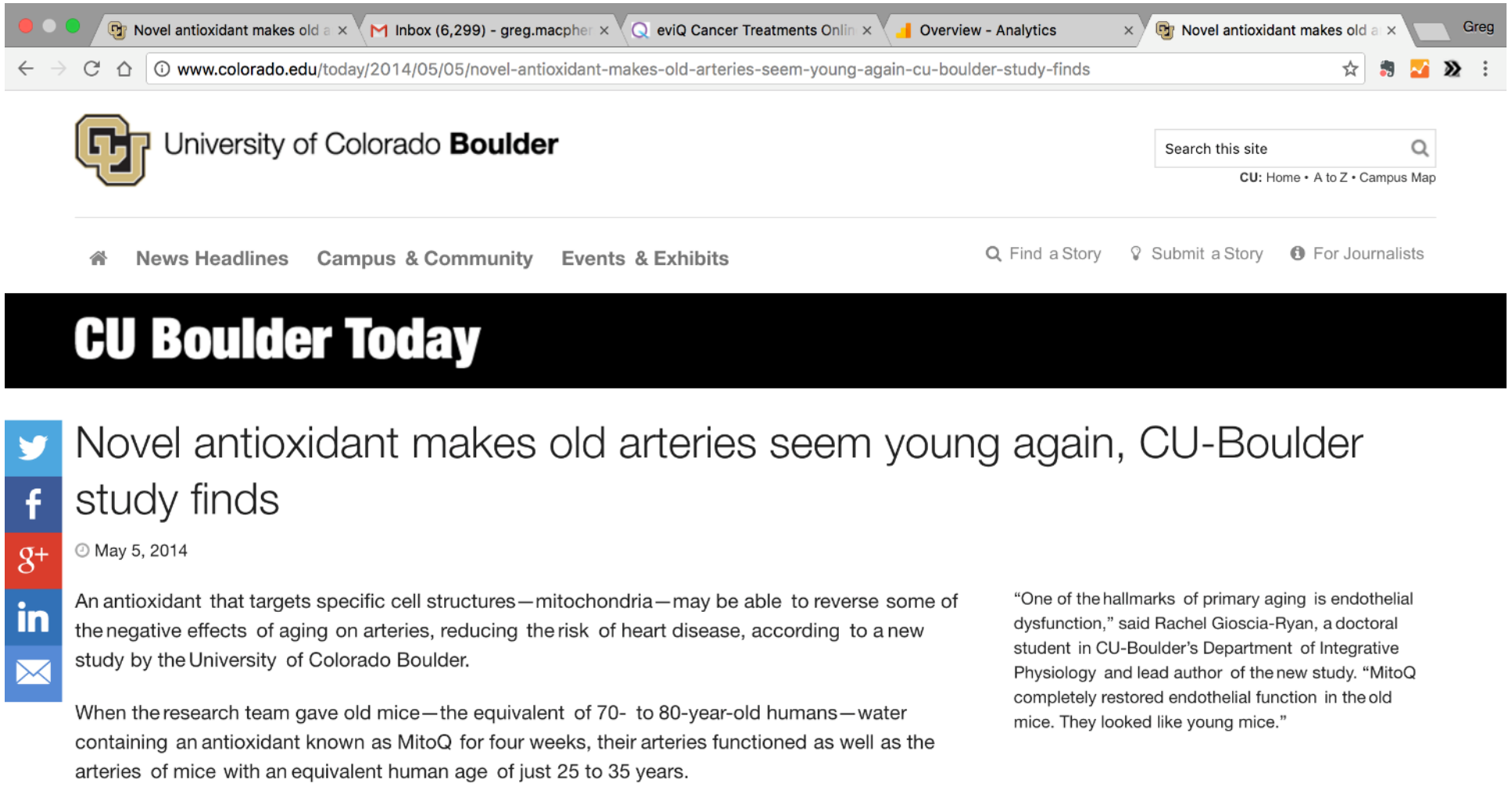
- FM Arm; 24-33% reduction in pain, 10-13% improvement in cognitive function
- CFS Blinded Arm; no significant benefit
- CFS Open Arm; including increases in energy (26% and 32%), sleep quality (17% and 35%), mental clarity (18% and 51%), activity (54% and 86%) and verbal reasoning (19% and 30%); as well as a modest reduction in pain at 6-weeks (13%)

Spanish trial

The mitochondria-targeted antioxidant MitoQ modulates oxidative stress, inflammation and leukocyte-endothelium interactions in leukocytes isolated from type 2 diabetic patients – Escribano-Lopez et al 2016

- 169 subjects; 98 with type-2 diabetes (T2D) and 71 control subjects
- Study aim was to examine whether MitoQ could reduce oxidative stress and affect metabolic parameters and leukocyte-endothelium interactions
- Leukocytes from T2D patients showed increased ROS (free radical) production but MitoQ treatment brought these values down to those of controls. MitoQ also increased levels of glutathione peroxidase (an ROS-neutralizing enzyme) in both patients and controls.
- MitoQ treatment significantly reduced the adhesion of leukocytes to endothelial cells in the T2D group
- MitoQ treatment also significantly reduced levels of NFκB-p65 and TNFa in the T2D group but did not change these levels in the control group.
- "Overall, our findings provide a better understanding of the pathophysiological mechanisms occurring in leukocytes/endothelium of T2D patients. They suggest that increased inflammation and oxidative stress, together with NFκB activation and increased proinflammatory cytokine TNFa, contribute to the enhanced interaction between these cells, which augments the risk of CVD. Importantly, treatment with MitoQ modulates these actions, thus preventing oxidative stress and chronic inflammation, which suggests that this compound has potential beneficial effects for preventing cardiovascular diseases in T2D"

Colorado U trial



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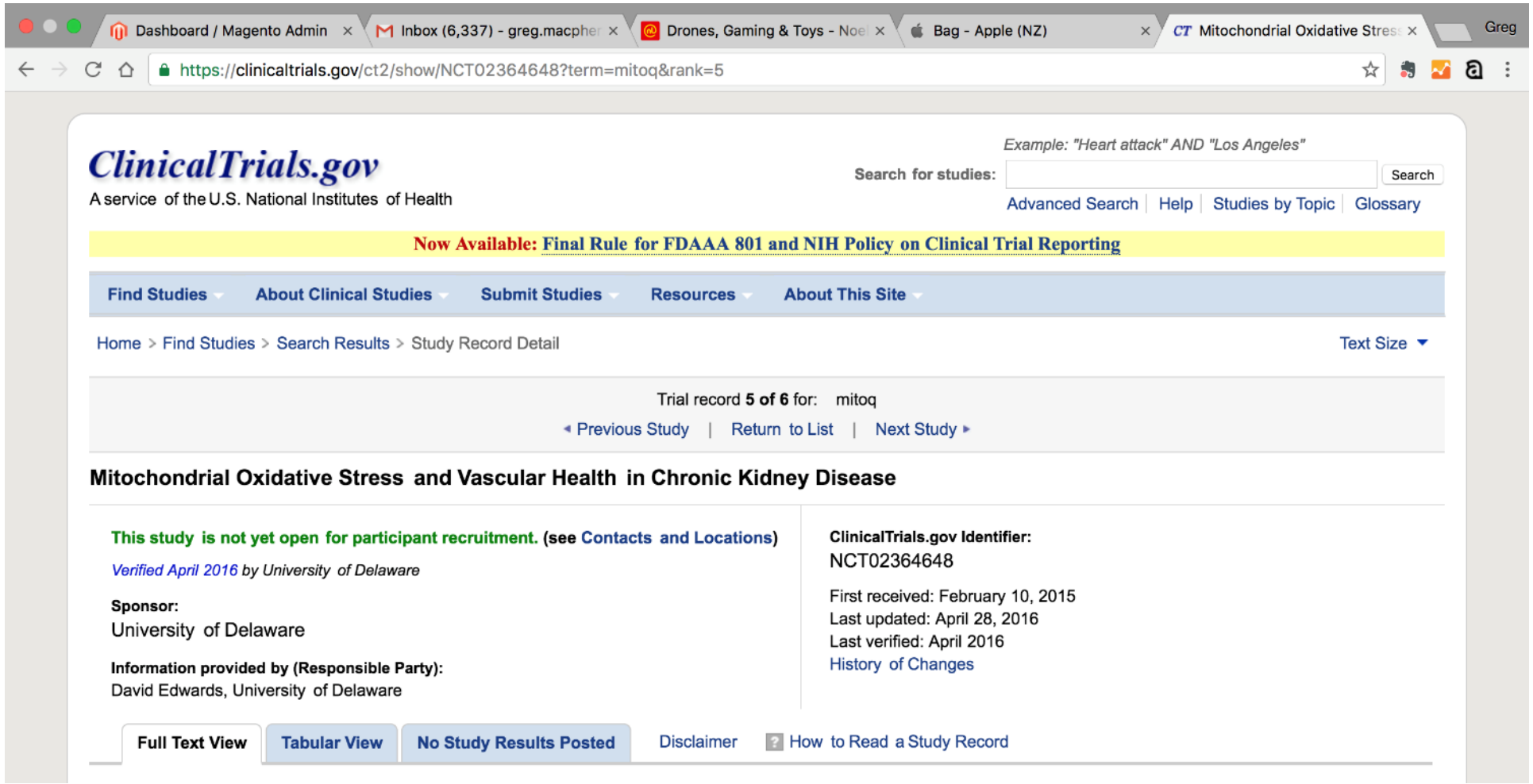
Delaware U trial



Planned Human Research

Diabetes

Multiple Sclerosis

Asthma

Mouse Model Research

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MITOCHONDRIAL SCIENCE

The mitochondria-targeted antioxidant MitoQ attenuates liver fibrosis in mice.

Authors: Rehman H et al

Abstract: Oxidative stress plays an essential role in liver fibrosis. This study investigated whether MitoQ, an orally active mitochondrial antioxidant, decreases liver fibrosis. Mice were injected with corn oil or carbon tetrachloride (CCl4, 1:3 dilution in corn oil; 1 µl/g, ip) once every 3 days for up to 6 weeks. 4-Hydroxynonenal adducts increased markedly after CCl4 treatment, indicating oxidative stress. MitoQ attenuated oxidative stress after CCl4. Collagen 1a1 mRNA and hydroxyproline increased markedly after CCl4 treatment, indicating increased collagen formation and deposition. CCl4 caused overt pericentral fibrosis as revealed by both the sirius red staining and second harmonic generation microscopy. MitoQ blunted fibrosis after CCl4. Profibrotic transforming growth factor-β1 (TGF-β1) mRNA and expression of smooth muscle a-actin, an indicator of hepatic stellate cell (HSC) activation, increased markedly after CCl4 treatment. Smad 2/3, the major mediator of TGF-β fibrogenic effects, was also activated after CCl4 treatment. MitoQ blunted HSC activation, TGF-β expression, and Smad2/3 activation after CCl4 treatment. MitoQ also decreased necrosis, apoptosis and inflammation after CCl4 treatment. In cultured HSCs, MitoQ decreased oxidative stress, inhibited HSC activation, TGF-β1 expression, Smad2/3 activation, and extracellular signal-regulated protein kinase activation. Taken together, these data indicate that mitochondrial reactive oxygen species play an important role in liver fibrosis and that mitochondria-targeted antioxidants are promising potential therapies for prevention and treatment of liver fibrosis.

Ref: Int J Physiol Pathophysiol Pharmacol. 2016 Apr 25;8(1):14-27

A mitochondrial-targeted ubiquinone modulates muscle lipid profile and improves mitochondrial respiration in obesogenic diet-fed rats.

Authors: Coudray C et al

Abstract: The prevalence of the metabolic syndrome components including abdominal obesity, dyslipidaemia and insulin resistance is increasing in both developed and developing countries. It is generally accepted that the development of these features is preceded by, or accompanied with, impaired mitochondrial function. The present study was designed to analyse the effects of a mitochondrial-targeted lipophilic ubiquinone (MitoQ) on muscle lipid profile modulation and mitochondrial function in obesogenic diet-fed rats. For this purpose, twenty-four young male Sprague-Dawley rats were divided into three groups and fed one of the following diets: (1) control, (2) high fat (HF) and (3) HF+MitoQ. After 8 weeks, mitochondrial function markers and lipid metabolism/profile modifications in skeletal muscle were measured. The HF diet was effective at inducing the major features of the metabolic syndrome--namely, obesity, hepatic enlargement and glucose intolerance. MitoQ intake prevented the increase in rat body weight, attenuated the increase in adipose tissue and liver weights and partially reversed glucose intolerance. At the muscle level, the HF diet induced moderate TAG accumulation associated with important modifications in the muscle phospholipid classes and in the fatty acid composition of total muscle lipid. These lipid modifications were accompanied with decrease in mitochondrial respiration. MitoQ intake corrected the lipid alterations and restored mitochondrial respiration. These results indicate that MitoQ protected obesogenic diet-fed rats from some features of the metabolic syndrome through its effects on muscle lipid metabolism and mitochondrial activity. These findings suggest that MitoQ is a promising candidate for future human trials in the metabolic syndrome prevention.

Ref: Br J Nutr. 2016 Apr 14;115(7):1155-66.

Selective Mitochondrial Targeting Exerts Anxiolytic Effects In Vivo.

Authors: Nussbaumer M et al

Abstract: Current treatment strategies for anxiety disorders are predominantly symptom-based. However, a third of anxiety patients remain unresponsive to anxiolytics highlighting the need for more effective, mechanism-based therapeutic approaches. We have previously compared high vs low anxiety mice and identified changes in mitochondrial pathways, including oxidative phosphorylation and oxidative stress. In this work, we show that selective pharmacological targeting of these mitochondrial pathways exerts anxiolytic effects in vivo. We treated high anxiety-related behavior (HAB) mice with MitoQ, an antioxidant that selectively targets mitochondria. MitoQ administration resulted in decreased anxiety-related behavior in HAB mice. This anxiolytic effect was specific for high anxiety as MitoQ treatment did not affect the anxiety phenotype of C57BL/6N and DBA/2J mouse strains. We furthermore investigated the molecular underpinnings of the MitoQ-driven anxiolytic effect and found that MitoQ treatment alters the brain metabolome and that the response to MitoQ treatment is characterized by distinct molecular signatures. These results indicate that a mechanism-driven approach based on selective mitochondrial targeting has the potential to attenuate the high anxiety phenotype in vivo, thus paving the way for translational implementation as long-term MitoQ administration is well-tolerated with no reported side effects in mice and humans.

Ref: Neuropsychopharmacology. 2016 Jun;41(7):1751-8.

MitoQ supplementation improves motor function and muscle mitochondrial health in old male mice

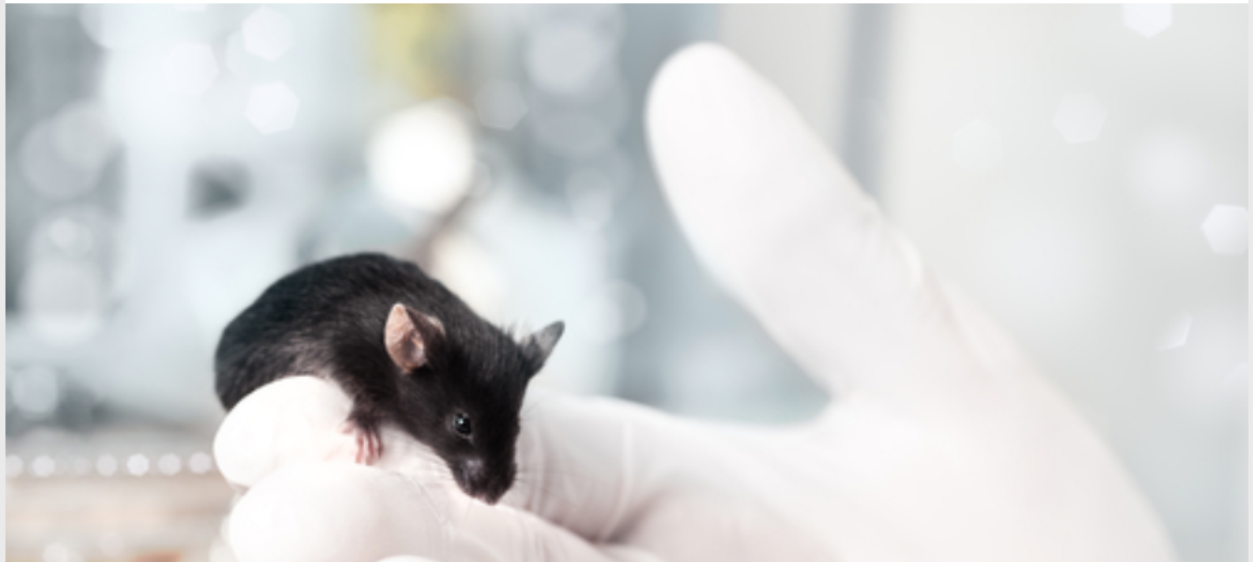
- JN-Justice Et al Colorado U
- Evaluated the role of excessive mtROS in age associated motor dysfunction

Results;

- MitoQ improved mass normalised grp-strength (+23.1%)
- Completely restored endurance rota-rod run time (+95.2%)
- Distance (+69.1%)
- In old animals supplemented with MitoQ but not old control or young male mice.
- Also saw an increased expression of SIRT-3, MnSOD and VDAC in the skeletal muscle.

Experimental MitoQ Treatment Fails Prevention of Age-Related Muscle Mass, Function Loss in Mice

NOVEMBER 7, 2016 BY ALICE MELAO IN NEWS.



Anti-aging
Research update

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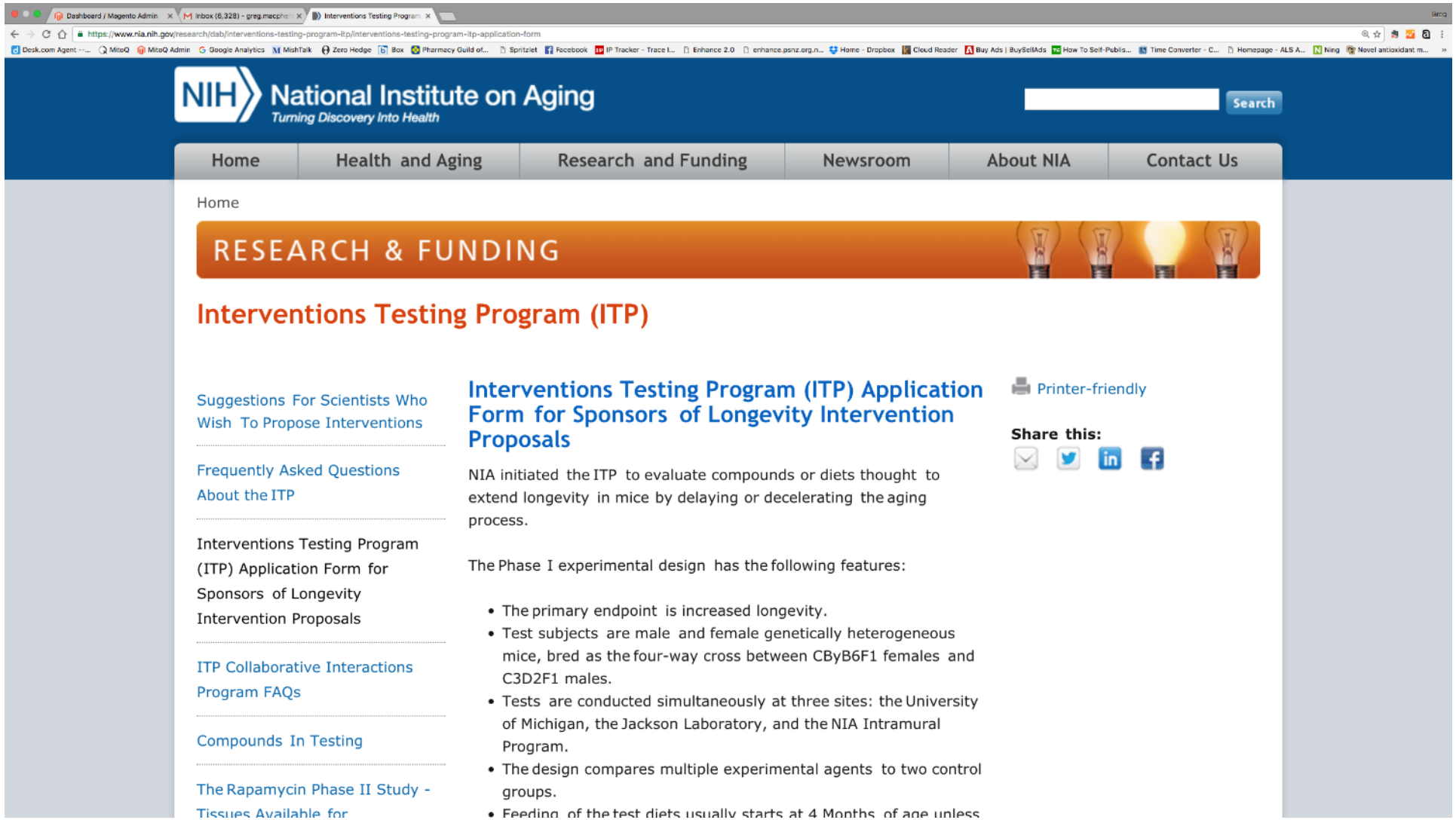
OBITUARIES
TWO YEARS YOUNGER THAN YOU
TWELVE YEARS OLDER THAN YOU
EXACTLY YOUR AGE
THREE YEARS YOUR JUNIOR
FIVE YEARS YOUR SENIOR
YOUR AGE ON THE DOT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The Rapamycin Phase II Study - Tissues Available for
Interventions Testing Program (ITP) Application Form for Sponsors of Longevity Intervention Proposals
NIA initiated the ITP to evaluate compounds or diets thought to extend longevity in mice by delaying or decelerating the aging process.
The Phase I experimental design has the following features:
The primary endpoint is increased longevity.
Test subjects are male and female genetically heterogeneous mice, bred as the four-way cross between CByB6F1 females and C3D2F1 males.
Tests are conducted simultaneously at three sites: the University of Michigan, the Jackson Laboratory, and the NIA Intramural Program.
The design compares multiple experimental agents to two control groups.
Feeding of the test diets usually starts at 4 Months of age unless
Printer-friendly
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40% reduction in telomere shortening.

Aging Cell (2003) **2**, pp141–143

SHORT TAKE

MitoQ counteracts telomere shortening and elongates lifespan of fibroblasts under mild oxidative stress

Gabriele Saretzki,[1] Michael P. Murphy[2] and Thomas von Zglinicki[1]

[1]Gerontology, Institute of Aging and Health, Newcastle University, Newcastle upon Tyne NE4 6BE, UK
[2]MRC-Dunn Human Nutrition Unit, Wellcome Trust/MRC Building, Hills Road, Cambridge CB2 2XY, UK

Key words: antioxidant; fibroblast, hyperoxia, mitoQ, oxidative stress, senescence, telomere

Oxidative damage is thought to be a major causal factor for replicative senescence and human aging (Harman, 1994). Leak-

possibility of such effects being due to non-specific interactions with mitochondria within cells can be discounted by the use of control compounds such as DPPT, which are also accumulated within mitochondria driven by the membrane potential but which do not act as antioxidants. Therefore, the blocking of a process by mitoQ but not by DPPT indicates a role for ROS production in the process and is consistent with the increased ROS production being primarily mitochondrial.

Telomeres act as 'mitotic clocks' in human fibroblasts because they shorten with each round of replication due to both the inability of DNA polymerases to replicate the very ends of chromosomes (Olovnikow, 1973) and the specific accumulation of stress-induced DNA damage (von Zglinicki, 2002). Eventually,

Meta-analysis

A review of over 200 papers resulting in the measurement of 220 significant endpoints

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Results

Increased

- Cell Survival
- Mitochondrial Membrane Potential
- Mitochondrial Respiration Rate
- **ECT activity**
- **ATP**
- **GSH**
- **Cardiolipin**
- **AMPK**
- Anti-inflammatory IL
- PGC-1a

Decreased

- **Caspase-3** activity
- Protein-carbonyl formation
- Lipid peroxidation
- ALT
- Heart rate
- Apidosity
- AST
- **TNF-a**
- **NFkB**
- **Inflammatory IL**

Thank you!

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