MitoMatters
The Official Newsletter of the Mitochondria Research Society

Volume 4, Issue 2, 2005

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Time to renew your 2006 MRS membership and receive uninterrupted subscription to the Mitochondrion journal and this newsletter.
Dear Colleagues,

It is with great pleasure that we announce the Mitochondria Research Society’s journal, *Mitochondrion*, is now fully indexed and appearing on PubMed. *Mitochondrion* was born out of our belief that research involving all the different aspects of mitochondria biology is substantial enough and deserving of a specialized platform for scientific research on mitochondria. Hundreds and hundreds of papers are published every year that deal directly or indirectly with mitochondrial metabolism, genetics and role in cell survival and death, only to name a few areas. This large body of literature, however, is dispersed throughout a great number of journals. In this context, *Mitochondrion* emerges as a dedicated venue that integrates basic and clinical mitochondrial research.

The addition of *Mitochondrion* to PubMed will surely increase the visibility of the journal and, we hope, attract more and more submissions, and as such contribute to our goal of making this journal the best forum for all interested in mitochondria research.

We invite you to submit your papers to *Mitochondrion* and work with us to make this journal the best reference for all things mitochondrial.

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**Front cover image:** Bovine pulmonary artery endothelial cell labeled with a mouse monoclonal anti-histone antibody and visualized with green-fluorescent Alexa Fluor® 500 goat anti–mouse IgG antibody (pseudocolored yellow). F-actin was labeled with blue-fluorescent Alexa Fluor® 350 phalloidin (pseudocolored purple), and mitochondria were stained with red-fluorescent MitoTracker® Red CMXRos (pseudocolored green). Image contributed by Michael Janes, Molecular Probes, Inc.
The journal *Mitochondrion* was recently added to Pubmed. Current and all previous issues are now available to search through that system. Below you will find the top 25 most viewed articles published in the journal. The variety of research areas covered by these papers highlights the extensive appeal of the journal.

1. Nuclear and mitochondrial DNA repair: similar pathways? • Review article
   *Mitochondrion*, Volume 5, Issue 2, 1 April 2005, Pages 89–108
   Larsen, N.B.; Rasmussen, M.; Rasmussen, L.J.

2. Mitochondrial dysfunction and its role in motor neuron degeneration in ALS • Review article
   *Mitochondrion*, Volume 5, Issue 2, 1 April 2005, Pages 77–87
   Manfredi, G.; Xu, Z.

3. The machinery of mitochondrial fusion, division, and distribution, and emerging connections to apoptosis • Review article
   Hales, K.G.

4. Mitochondrial dysfunction in reproduction • Article
   *Mitochondrion*, Volume 4, Issue 5–6, 1 September 2004, Pages 577–600
   Jansen, R.P.S.; Burton, G.J.

5. Physical, occupational, respiratory, speech, equine and pet therapies for mitochondrial disease • Article
   *Mitochondrion*, Volume 4, Issue 5–6, 1 September 2004, Pages 403–415
   Shanske, S.; Wong, L.-J.C.

6. The role of mitochondrial transport in energy metabolism • Review article
   *Mitochondrion*, Volume 2, Issue 5, 1 April 2003, Pages 319–343
   Passarella, S.; Atlante, A.; Valenti, D.; de Bari, L.

7. Mitochondrial genome polymorphisms associated with type-2 diabetes or obesity • Article
   *Mitochondrion*, Volume 5, Issue 1, 1 February 2005, Pages 15–33
   Guo, L.-J.; Oshida, Y.; Fuku, N.; Takeyasu, T.; Fujita, Y.; Kurata, M.; Sato, Y.; Ito, M.; Tanaka, M.

8. Mitochondrial pharmaceutics • Article
   *Mitochondrion*, Volume 3, Issue 4, 1 March 2004, Pages 229–244
   Weissig, V.; Cheng, S.-M.; D’Souza, G.G.M.

9. Mitochondrial function, zinc, and intermediary metabolism relationships in normal prostate and prostate cancer • Review article
   Costello, L.C.; Franklin, R.B.; Feng, P.

10. Mitochondrial nitric oxide synthase • Review article
    Brookes, P.S.

11. Mitochondrial dysfunction and possible treatments in Parkinson’s disease—a review • Article
    Shults, C.W.

12. Cyclical mitochondrial $\Delta\psi_m$ fluctuations linked to electron transport, $F_0F_1$ ATP-synthase and mitochondrial $\text{Na}^+$/Ca$^{2+}$ exchange are reduced in Alzheimer’s disease cybrids • Article
    *Mitochondrion*, Volume 5, Issue 2, 1 April 2005, Pages 109–119
    Thiffault, C.; Bennett, J.P.

13. Molecular analysis for mitochondrial DNA disorders • Article
    *Mitochondrion*, Volume 4, Issue 5–6, 1 September 2004, Pages 403–415
    Shanske, S.; Wong, L.-J.C.

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14. A century of mitochondrial research: achievements and perspectives • Article
_Mitochondrion_, Volume 1, Issue 1, 1 June 2001, Pages 3–31
Scheffler, I.E.

15. Respiratory chain enzyme analysis in muscle and liver • Article
_Mitochondrion_, Volume 4, Issue 5–6, 1 September 2004, Pages 363–375
Thorburn, D.R.; Chow, C.W.; Kirby, D.M.

16. Developing a systematic approach to the diagnosis and classification of mitochondrial disease • Article
_Mitochondrion_, Volume 4, Issue 5–6, 1 September 2004, Pages 351–361
Naviaux, R.K.

17. DNA-binding proteins of mammalian mitochondria • Article
_Mitochondrion_, Volume 5, Issue 1, 1 February 2005, Pages 35–44
Kutsyi, M.P.; Gouliaeva, N.A.; Kuznetsova, E.A.; Gaziev, A.I.

18. Mitochondrial signal transduction in accelerated wound and retinal healing by near-infrared light therapy • Article
_Mitochondrion_, Volume 4, Issue 5–6, 1 September 2004, Pages 559–567

19. Mitochondrial dysfunction in AIDS and its treatment • Article
_Mitochondrion_, Volume 4, Issue 5–6, 1 September 2004, Pages 763–777
Gerschenson, M.; Brinkman, K.

20. Immunological approaches to the characterization and diagnosis of mitochondrial disease • Article
_Mitochondrion_, Volume 4, Issue 5–6, 1 September 2004, Pages 417–426
Capaldi, R.A.; Murray, J.; Byrne, L.; Janes, M.S.; Marusich, M.F.

21. l-Carnitine and acetyl-l-carnitine in the treatment of complications associated with HIV infection and antiretroviral therapy • Review article
_Mitochondrion_, Volume 4, Issue 2–3, 1 July 2004, Pages 163–168
Ilias, I.; Manoli, I.; Blackman, M.R.; Gold, P.W.; Alesci, S.

22. Mitochondrial dysfunction in cancer • Article
_Mitochondrion_, Volume 4, Issue 5–6, 1 September 2004, Pages 755–762
Modica-Napolitano, J.S.; Singh, K.K.

23. Structure and patterns of sequence variation in the mitochondrial DNA control region of the great cats • Article
_Mitochondrion_, Volume 1, Issue 3, 1 October 2001, Pages 279–292

24. Oxidative phosphorylation analysis: assessing the integrated functional activity of human skeletal muscle mitochondria-case studies • Article
_Mitochondrion_, Volume 4, Issue 5–6, 1 September 2004, Pages 377–385
Puchowicz, M.A.; Varnes, M.E.; Cohen, B.H.; Friedman, N.R.; Kerr, D.S.; Hoppel, C.L.

25. Mitochondrial dysfunction in osteoarthritis • Article
_Mitochondrion_, Volume 4, Issue 5–6, 1 September 2004, Pages 715–728
Blanco, F.J.; Lopez-Armada, M.J.; Maneiro, E.
1. New association between mitochondria and immune response: Multicellular organisms developed several mechanisms to counteract the deleterious effects of infectious agents. A viral infection in higher organisms results in a rapid and complex response that involves the activation of several signaling pathways. Two recent papers published in *Cell* and *Molecular Cell* identified a new pathway in which a mitochondrial protein called MAVS, for Mitochondrial AntiViral Signaling, is necessary for interferon production, and thus protection against viral infection. This protein indicates a new role of mitochondria in innate immunity and in controlling susceptibility for diseases. The commentary cited below, by Maniatis and colleagues addresses the implications of this finding.

McWhirter et al., *Cell*, 122: 645–647, 2005

2. The mitochondrial DNA: The discovery of extranuclear DNA in animal cells, i.e., the mitochondrial DNA, was a remarkable accomplishment for a time when “molecular biology” was still in its infancy. This “inheritance unit” did not obey Mendelian inheritance rules and followed cytoplasmic inheritance patterns. In this paper, Jean-Claude Mounolou and François Lacroute give their own personal accounts of their involvement, and that of others, in such discovery. They provide a vivid and captivating historical perspective to an event that was to change biology.


3. The permeability transition pore: Opening of the mitochondrial permeability transition pore is a key event in several cellular processes and seems to be directly involved in both apoptosis and necrosis. The nature of this proteinaceous “pore” has long been a subject of intensive research, and sometimes heated arguments. Two recent papers show that cyclophilin D, a small mitochondrial protein previously identified as a prolyl isomerase, is an integral part of the permeability pore. More importantly, they show that mice lacking this protein are more resistant to several oxidative damage inducing conditions, such as cardiac and brain ischemia-reperfusion injury. These findings highlight cyclophilin D as a possible therapeutic target in several diseases that are associated with oxidative stress.


Schinzel et al., *PNAS*, 102: 12005–12010, 2005

4. The mechanisms of anxiety: Mood disorders are highly prevalent, albeit very poorly understood diseases. Uncovering some of the neurobiologic and biochemical underpinnings of anxiety disorders would certainly represent a significant step toward more effective therapeutic interventions. There is also growing evidence that mitochondria play an important role in regulating integrated central nervous system function. Further evidence for this critical role came in a recent paper showing that mice with reduced levels of Bcl-2 demonstrate a significant increase in anxiety-like behaviors, despite showing no gross behavioral abnormalities. Bcl-2 is a mitochondrial protein that is at the core of controlling mitochondrial calcium uptake and cell fate through apoptosis. These findings underscore the role of mitochondria in brain function and their association with, yet, another class of diseases.

Einat et al., *Behav. Brain Res.*, e-pub ahead of print, 2005

5. Cardioprotection via mitochondria: Preconditioning protects the heart against ischemia-reperfusion injury and thus decreases the severity of infarct damage. This protective condition can be induced by a drug called diazoxide, but the molecular mechanism involved in preconditioning is still somewhat unclear, although a role for mitochondria has been firmly established. In the paper below, researchers from Germany, Spain, and Italy show that the protein connexin 43, which is expressed in cardiomyocyte mitochondria, is necessary for preconditioning of the heart. Connexin 43 seems to mediate diazoxide-induced reactive oxygen species formation, and mice expressing lower amounts of this protein get less protection against heart damage by diazoxide. This finding may provide new therapeutic avenues in decreasing the severity of myocardial infarcts.

Mito Meetings

**DEC 15–17, 2005**  
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Tel: +81-3-3964-3241 ext 3095  
Fax: +81-3-3579-4776  
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**JUNE 14–17, 2006**  
**MASTERING THE MITOCHONDRIAL MAZE**  
The United Mitochondrial Disease Foundation in cooperation with The Mitochondria Research Society (MRS)  
E-mail: kara@umdf.org  
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**JUNE 28–30, 2006**  
**5TH FOCUS COURSE ON MITOCHONDRIAL MEDICINE**  
Nijmegen Medical Centre, Netherlands  
www.orphan-europe-academy.com/course.php?courseID=12&topic=synopsis

**JULY 22–27, 2006**  
**14TH EUROPEAN BIOENERGETICS CONFERENCE**  
E-mail: Vladimir Skulachev, PhD (info@be2006.ru)  
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