Mitochondria Communication (A4)

Organizer(s) Jared Rutter, Cole M. Haynes and Marcia C. Haigis
January 14–18, 2017
Sagebrush Inn & Suites • Taos, New Mexico USA
Discounted Abstract Deadline: Sep 20, 2016
Abstract Deadline: Oct 13, 2016
Scholarship Deadline: Sep 20, 2016
Discounted Registration Deadline: Nov 14, 2016

Supported by the Directors Fund

This meeting took place in 2017

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Summary of Meeting:
Understanding how mitochondria communicate within the cell will provide important clues to elucidate its roles in normal cell physiology, as well as numerous diseases such as diabetes, neurodegeneration and cancer. The classical view of mitochondria is of an organelle that interacts with other features of cell biology primarily through the provision and consumption of metabolic intermediates and energetic products. Emerging evidence from many areas of science is accumulating to suggest that mitochondria play much more active roles in communication with other organelles, determining cell and organismal behavior. This conference will focus on these integrated behaviors and the resulting communication. The sessions will be organized around the different nodes, modes and destinations of that signaling. It will bring together people and ideas from different areas of cell biology and physiology who typically do not attend the same conferences, resulting in the development of new collaborations and concepts.

View Scholarships/Awards

No registration fees are used to fund entertainment or alcohol at this conference

Conference Program

Print | View meeting in 12 hr (am/pm) time

The meeting will begin on Saturday, January 14 with registration from 16:00 to 20:00 and a welcome mixer from 18:00 to 20:00. Conference events conclude on Wednesday, January 18 with a closing plenary session from 17:00 to 19:30, followed by a social hour and entertainment. We recommend return travel on Thursday, January 19 in order to fully experience the meeting.

SATURDAY, JANUARY 14

16:00–20:00	**Arrival and Registration**	Sagebrush Lobby & Cantina
18:00–20:00	**Welcome Mixer** No registration fees are used to fund alcohol served at this function.	Sagebrush Lobby & Cantina

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SUNDAY, JANUARY 15

07:00–08:00	**Breakfast**	Sagebrush Lobby & Cantina
08:00–09:00	**Welcome and Keynote Address** **Meeting has ended...abstracts no longer viewable online.** *** Jared Rutter,** University of Utah, USA **Johan Auwerx,** Ecole Polytechnique Fédéerale de Lausanne - EPFL, Switzerland *Cross-Species Genetic Mapping of Targets in Mitochondria, Metabolism and Aging*	Chamisa Ballroom 1
09:00–11:30	**Epigenetic Signaling and Regulation** **Meeting has ended...abstracts no longer viewable online.** *** Marcia C. Haigis,** Harvard Medical School, USA **Eyal Gottlieb,** Technion Integrated Cancer Center, Israel *The Onco-Metabolic Role and Liabilities of the TCA Cycle in Cancer* **Matthew D. Hirschey,** Duke University, USA *Short Talk: Lipids Reprogram Metabolism to Become a Major Carbon Source for Histone Acetylation* **Coffee Break** **William G. Kaelin, Jr.,** Dana-Farber Cancer Institute, USA *Molecular Pathogenesis of IDH Mutant Cancers* **Atan J. Gross,** Weizmann Institute of Science, Israel *Short Talk: MTCH2: A Critical Regulator of Mitochondria Function and Communication* **Kathryn E. Wellen,** University of Pennsylvania, USA *Linking Metabolism to DNA Damage Repair*	Chamisa Ballroom 1
11:30–17:00	**On Own for Lunch**	
11:30–13:00	**Poster Setup**	Chamisa Ballroom 2
13:00–22:00	**Poster Viewing**	Chamisa Ballroom 2
14:30–16:30	**Workshop 1** *** Valentina Perissi,** Boston University School of Medicine, USA **Adam L. Orr,** Weill Cornell Medical College, USA *Novel Site-Specific Inhibitors of Mitochondrial ROS Production Define ROS-Mediated Events in Health and Disease* **Amanda E. Brinker,** University of Kansas Medical Center, USA *Differences in Mitochondrial Haplotype Influence Metastatic Efficiency and Expression of Related Nuclear Genes* **Oleh Khalimonchuk,** University of Nebraska, USA *Loss of Mitochondrial Protease OMA1 Alters Proliferative Properties and Promotes Metastatic Growth of Breast Cancer Cells* **Yoshiyuki Tsujihata,** Takeda Pharmaceutical Company, Ltd., Japan *Novel Approach for Exploring Small Molecule Regulators of Mitochondrial ATP under Hypoxia in Cardiomyocytes by Live Cell High Content Screening using ATP Biosensor* **Allen Kaasik,** University of Tartu, Estonia *Miro Proteins are Required for Priming Mitochondria for PINK1*	Chamisa Ballroom 1

Induced Parkin Translocation

Nicholas R. Weir, Harvard University, USA
Msp1 Antagonizes Integration of Membrane Proteins into Lipid Bilayers

16:30—17:00 **Coffee Available** Chamisa Lobby

17:00—19:15 **Mitochondrial/ER Communication** Chamisa Ballroom 1
Meeting has ended...abstracts no longer viewable online.

*** Maya B. Schuldiner,** Weizmann Institute of Science, Israel

Jodi Nunnari, University of California, Davis, USA
ER/Mitochondria Contact Sites as Organizers of Mito Function

Thomas Langer, CECAD Research Center, Germany
Proteolytic Control of Mitochondrial Function

Luca Scorrano, University of Padova, Italy
Unbiased Genetic and Proteomic Screenings Unveil the Mitochondria-Endoplasmic Reticulum Contacts Machinery

György Hajnóczky, Thomas Jefferson University, USA
Calcium and ROS Regulation at the ER-Mitochondrial Interface

19:15—20:15 **Social Hour with Lite Bites** Chamisa Ballroom 2
No registration fees are used to fund alcohol served at this function.

19:30—22:00 **Poster Session 1** Chamisa Ballroom 2

MONDAY, JANUARY 16

07:00—08:00 **Breakfast** Sagebrush Lobby & Cantina

08:00—11:00 **ROS Signaling** Chamisa Ballroom 1
Meeting has ended...abstracts no longer viewable online.

*** Adam L. Hughes,** University of Utah, USA

Navdeep S. Chandel, Northwestern University, USA
Mitochondria as Signaling Organelles

Gerald S. Shadel, Yale School of Medicine, USA
Adaptive Responses to Mitochondrial Stress

Heide Christine Patterson, Whitehead Institute, USA
Short Talk: Mitochondrial ROS-Induced Syk Signaling Mediates Brown Adipocyte Development and Thermogenesis

Coffee Break

Heinrich Jasper, Buck Institute for Research on Aging, USA
Mitochondrial Stress Signaling, Stem Cells and Lifespan Extension: Lessons from Drosophila

Danica Chen, University of California, Berkeley, USA
Mitochondrial UPR-Mediated Metabolic Checkpoint, Stem Cell Aging and Rejuvenation

11:00—12:00 **NIH Funding Opportunities for Mitochondrial Research: Conversation with an NCI Program Officer** Chamisa Ballroom 1

Michael Graham Espey, NCI, National Institutes of Health, USA

12:00—17:00 **On Own for Lunch**

12:00—13:00 **Poster Setup** Chamisa Ballroom 2

13:00—22:00 **Poster Viewing** Chamisa Ballroom 2

16:30—17:00 **Coffee Available** Chamisa Lobby

17:00—19:00 **Mitochondria/Lysosome Communication** Chamisa Ballroom 1
Meeting has ended...abstracts no longer viewable online.

*** Oleh Khalimonchuk,** University of Nebraska, USA

David M. Sabatini, Whitehead Institute for Biomedical Research, USA
Amino Acid Sensing and Mitochondrial Function

Adam L. Hughes, University of Utah, USA
Pathways of Mitochondria-Lysosome Crosstalk

Maya B. Schuldiner, Weizmann Institute of Science, Israel
Keeping in Touch – Discovering and Characterizing New Contact Sites between Organelles

19:00—20:00 **Social Hour with Lite Bites** Chamisa Ballroom 2
No registration fees are used to fund alcohol served at this function.

19:30–22:00	**Poster Session 2**	Chamisa Ballroom 2

TUESDAY, JANUARY 17

07:00–08:00	**Breakfast**	Sagebrush Lobby & Cantina
08:00–11:00	**Communicating Mitochondrial Dysfunction** **Meeting has ended...abstracts no longer viewable online.** *** Heidi M. McBride,** McGill University, Canada **Cole M. Haynes,** University of Massachusetts Medical School, USA *Preferential Propagation of Toxic mtDNAs by the UPRmt* **Marcia C. Haigis,** Harvard Medical School, USA *Mitochondrial Sirtuin Networks* **Sebastien Herzig,** The Salk Institute for Biological Studies, USA *Short Talk: Regulation of Mitochondrial Dynamics by AMPK* **Coffee Break** **Matt Kaeberlein,** University of Washington, USA *Mechanisms Linking Severe Mitochondrial Disease and Normative Aging* **Jared Rutter,** University of Utah, USA *Stress Responsive Mitochondrial Protein Degradation*	Chamisa Ballroom 1
11:00–17:00	**On Own for Lunch**	
11:00–13:00	**Poster Setup**	Chamisa Ballroom 2
13:00–22:00	**Poster Viewing**	Chamisa Ballroom 2
16:30–17:00	**Coffee Available**	Chamisa Lobby
17:00–19:00	**Mitochondria and Inter-Cellular Communication** **Meeting has ended...abstracts no longer viewable online.** *** Cole M. Haynes,** University of Massachusetts Medical School, USA **William B. Mair,** Harvard School of Public Health, USA *Mitochondrial Plasticity Is Required for AMPK-Mediated Longevity* **Michael Ristow,** ETH Zürich, Switzerland *Mitochondrial Control of Healthy Aging* **Heidi M. McBride,** McGill University, Canada *The Cell Biology of Metabolic Flux* **Meng C. Wang,** Baylor College of Medicine, USA *Short Talk: Microbes Tune Mitochondrial Dynamics to Regulate Host Metabolic Adaptation to Environmental Variations*	Chamisa Ballroom 1
19:00–20:00	**Social Hour with Lite Bites** No registration fees are used to fund alcohol served at this function.	Chamisa Ballroom 2
19:30–22:00	**Poster Session 3**	Chamisa Ballroom 2

WEDNESDAY, JANUARY 18

07:00–08:00	**Breakfast**	Sagebrush Lobby & Cantina
08:00–11:00	**Mitochondria and Fuel Catabolism** **Meeting has ended...abstracts no longer viewable online.** **Tony Hui,** Princeton University, USA *Exchange of Mitochondrial Substrates Between Tissues* **Ralph J. DeBerardinis,** University of Texas Southwestern Medical Center, USA *Mitochondrial Metabolism and its Importance in Health and Disease* **Desiree Schatton,** University of Cologne, Germany *Short Talk: The RNA-Binding Protein CLUH is a Master Regulator of Mitochondrial Catabolic Programme Activated upon Nutrient Deprivation* **Coffee Break** *** Dave Pagliarini,** Morgridge Institute for Research at University of Wisconsin-Madison, USA *Short Talk: Diverse Mitochondrial Protein Functions Revealed by*	Chamisa Ballroom 1

Multi-Omic Mass Spectrometry Profiling

Benjamin Tu, University of Texas Southwestern Medical Center, USA
Adaptive Catabolism during Cell Proliferation

11:00–17:00 **On Own for Lunch**

14:30–16:30 **Workshop 2** Chamisa Ballroom 1

*** Danica Chen,** University of California, Berkeley, USA

Yi Zhang, NHLBI, National Institutes of Health, USA
Selective Protein Synthesis on the Mitochondrial Surface Drives the mtDNA Selection

Anna M. Schulz, Memorial Sloan Kettering Cancer Center, USA
Gcn2-Mediated Mitochondria Protection during Metabolic Stress

Jonathan Van Vranken, University of Utah School of Medicine, USA
FASII Activates Oxidative Metabolism in Mitochondria via ACP Acylation

Jessica B. Spinelli, Harvard University, USA
Evaluating the Effect of Ammonium on Cancer Cell Homeostasis

Laurent Le Cam, Institut de Recherche en Cancérologie de Montpellier, France
The MDM2 Oncoprotein Controls Complex I Activity and Mitochondrial Dynamics Independently of p53

Martin Ott, Stockholm University, Sweden
Modulation of Cellular Stress Signaling and Aging by Changes in Mitochondrial Translation Accuracy

Veena Prahlad, University of Iowa, USA
Mitochondria-Regulated Immune Pathway in C. elegans is Neuroprotective

16:30–17:00 **Coffee Available** Chamisa Lobby

17:00–19:15 **Coordination of Mitochondrial Mass and Physiology** Chamisa Ballroom 1
Meeting has ended...abstracts no longer viewable online.

*** Kathryn E. Wellen,** University of Pennsylvania, USA

J. Wade Harper, Harvard Medical School, USA
Digitizing Ubiquitin Signaling for Mitophagy

Maulik Patel, Vanderbilt University, USA
Short Talk: Homeostatic Stress Responses Regulate Selfish Mitochondrial Genome Dynamics

Richard J. Youle, NINDS, National Institutes of Health, USA
Mitophagy Regulation

Valentina Perissi, Boston University School of Medicine, USA
Short Talk: Regulation of Mitochondrial Biogenesis through Mitochondria Retrograde Signaling and Chromatin Remodeling of Nuclear-Encoded Mitochondrial Genes

Nektarios N. Tavernarakis, Foundation for Research and Technology-Hellas, Greece
Mitochondrial Turnover and Homeostasis during Aging

19:15–19:30 **Meeting Wrap-Up: Outcomes and Future Directions (Organizers)** Chamisa Ballroom 1
Meeting has ended...abstracts no longer viewable online.

19:30–20:30 **Social Hour with Lite Bites** Chamisa Ballroom 2
No registration fees are used to fund alcohol served at this function.

20:00–23:00 **Entertainment** Chamisa Ballroom 2
Entertainment is not subsidized by conference registration fees nor any U.S. federal government grants. Funding for this expense is provided by other revenue sources.

THURSDAY, JANUARY 19

Departure

*Session Chair †Invited, not yet responded.

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Keystone Symposia - Poster Abstracts
Mitochondria Communication (A4)

Current as of 12/28/2016 03:12

Only those abstracts that are listed as "viewable" are displayed here.
Authors can choose to not make his/her abstract available online.

Only the abstract text is displayed here, not the pictures or graphs.

POSTER NUMBER: 1001

Host defence to bacteria involves mitochondrial respiratory chain reorganization in macrophages

Rebeca Acín-Pérez[1,*], Johan Garaude[1,2,*], Sarai Martínez-Cano[1], Michel Enamorado[1], Matteo Ugolini[3], Estanislao Nistal-Villán[4], Sandra Hervás-Stubbs[4,5], Pablo Pelegrín[6], Leif E. Sander[3], José A. Enríquez[1,7,#] and David Sancho[1,#]
[1]Centro Nacional de Investigaciones Cardiovasculares Carlos III (CNIC), Melchor Fernández Almagro, 3, 28029 Madrid, Spain;l [2]Institute for Regenerative Medicine and Biotherapies, Institut National pour la Santé et la Recherche Médicale, U1183, 80 Avenue Augustin Fliche, 34295 Montpellier Cedex 5, France; [3]Department of Infectious Diseases and Pulmonary Medicine, Charité Hospital Berlin, Augustenburger Platz 1, 13352 Berlin, Germany; [4]Centro de Investigación Médica Aplicada, Universidad de Navarra, Pio XII, 55 E-31008 Pamplona, Spain; [5]Instituto de Investigación Sanitaria de Navarra (IDISNA), Recinto de Complejo Hospitalario de Navarra, E-31008, Pamplona, Spain; [6]Unidad de Inflamación y Cirugía Experimental, Centro de Investigación Biomédica en Red en el Área Temática de Enfermedades Hepáticas y Digestivas, Hospital Clínico Universitario Virgen de la Arrixaca, Instituto Murciano de Investigación Biosanitaria-Arrixaca (IMIB-Arrixaca), 30120 Murcia, Spain; [7]Departamento de Bioquímica y Biología Molecular y Celular. Universidad de Zaragoza. Zaragoza, Spain.
[*]These authors contributed equally to this work
[#]D.S and J.A.E share credit for senior authorship

The mitochondrial electron transport chain (ETC) is a metabolic hub whose adaptations accompany fuel source fluctuations, stress responses, and innate immune signals to ensure optimal cellular functions. Macrophages tightly scale their core metabolism upon activation by innate immune receptors but the precise regulation of the ETC upon pathogen recognition and its functional implications are currently unknown. Here we show that innate immune sensing of live bacteria by macrophages elicits a profound re-organization of the ETC. This is characterized by a switch in the relative contribution of ETC complexes I and II (CI and CII) to mitochondrial respiration that is mediated by the phagosomal nicotinamide adenine dinucleotide phosphate (NADPH)-oxidase and the reactive oxygen species (ROS)-dependent Src-family tyrosine-kinase Fgr. Detection of dead bacteria does not trigger ETC adaptations while bacterial RNA, which signifies bacterial viability to innate immune cells, efficiently enhances CII activity. Furthermore, macrophages deficient for Toll-like receptor (TLR) signalling and for the NOD-like receptor (NLR) family, pyrin domain-containing protein 3 (NLRP3) inflammasome, both connected to bacterial RNA sensing and viable bacteria-specific immune responses, are unable to initiate ETC adaptations or to induce CII activity upon bacteria encounter. Consistently, the inhibition of CII in *E. coli* infected mice decreases IL-1 and increases IL-10 serum-levels to those found in mice treated with dead bacteria and impairs control of bacteria. We thus identify the innate immune receptor-mediated ETC reorganization as an early immune-metabolic checkpoint that potentially adjusts innate immune responses during bacterial infection.

POSTER NUMBER: 1002

The X protein protects against mitochondrial and neuronal defects of OPA1-deficient neurons

Macarena S. Arrázola[1], Daniel Gonzalez-Dunia[2], Marie-Christine Miquel[1] and Pascale Belenguer[1*]
[1]Centre de Recherches sur la Cognition Animale (UMR5169), CNRS/Université Paul Sabatier Toulouse III, Toulouse, France; [2]Centre de Physiopathologie de Toulouse Purpan, Inserm UMR1043, CNRS UMR5282, Toulouse, France

Mutations in the gene coding the mitochondrial fusion protein OPA1 lead to Dominant Optic Atrophy (DOA), a mitochondrial disease characterized by a reduction of visual acuity and blindness, to date without treatment. DOA mainly affects Retinal Ganglionic Cells (RGC), which axons form the optic nerve, although 20% of the patients also develop extra-ocular neuronal complications. Deficiency of OPA1 provokes mitochondrial fragmentation, alterations of mitochondrial distribution and function, leading to defects in neuronal arborization and synapse formation.

As mitochondria are dysfunctional in several neurodegenerative diseases, they appear as an attractive target for therapy. We recently showed that a viral protein called X protects neurons *in vitro* and *in vivo* against diverse

sulfoximine (BSO). These findings indicate that the p62 promotes tumorigenesis by maintaining redox homeostasis. These p62-dependent pathways reveal strategies for selective therapeutic intervention in TSC/LAM.

69

POSTER NUMBER: 2018

The MDM2 oncoprotein controls complex I activity and mitochondrial dynamics independently of p53

Arena G.[1], Riscal R.[1], Cissé M.[1], Pyrdziak S.[1], Fuentes M.[1], Gayte L.[1], Bernex F.[1], Linares L.K[1,2] and Le Cam L.[1,2]
[1]Institut de Recherche en Cancérologie de Montpellier, INSERM U1194; Institut du cancer de Montpellier, ICM; Montpellier, 34298, France
[2]Co-senior and Corresponding authors

The Mouse Double Minute 2 (MDM2) oncoprotein is recognized as a major negative regulator of the p53 tumor suppressor. We recently showed that MDM2 is recruited to chromatin independently of p53 to regulate a transcriptional program implicated in amino acid metabolism and redox homeostasis (Riscal *et al.*, Mol Cell 2016). Genome-wide studies highlighted an important role for ATF3/4 transcription factors in tethering MDM2 to its target genes implicated in serine metabolism. Interestingly, our ChIP-seq analyses also revealed that MDM2 functions in metabolism extend beyond these chromatin-associated activities and that a significant fraction of MDM2 protein also localizes inside mitochondria. Our data indicate that Mitochondrial-MDM2 binds to the mitochondrial genome independently of p53 to control expression of complex I subunits. Strikingly, oxidative stress and hypoxia increase the amount of mitochondrial-MDM2, resulting in repression of its target genes and decreased Complex I activity. The in vivo relevance of these mitochondrial functions of MDM2 was confirmed in genetically engineered mouse models lacking MDM2 in skeletal muscles. I will present our latest unpublished data showing how mitochondrial-MDM2 controls the activity of the electron transport chain (ETC) and muscular activity. Taken together, our data illustrate a previously unsuspected function of the MDM2 oncoprotein in metabolism of both normal and cancer cells.

Riscal R. et al. (2016) Chromatin-bound MDM2 regulates serine metabolism and redox homeostasis independently of p53. *Molecular Cell*. Jun 16;62(6):890-902

70

POSTER NUMBER: 2055

The Role of mAAA protease transmembrane domain in dislocating mitochondrial inner membrane proteins

Seoeun Lee[1], Hunsang Lee[2] and Hyun Kim[1*]
[1]School of Biological Sciences, College of Natural Sciences, Seoul National University, Gwanak-ro, Gwanak-gu, Seoul, 08826, South Korea; [2]Donnelly Centre, 160 College St, Toronto, ON M5S3E1, Canada

In eukaryotic cells, the majority of mitochondrial proteins are nuclearly encoded and synthesized in the cytosol. Mitochondrial proteins are targeted to mitochondria via their targeting signals which guide them to specific compartments; outer membrane (OM), intermembrane space (IMS), inner membrane(IM), or matrix.
AAA-ATPases(ATP Associated with diverse cellular Activities) are known to be involved in membrane protein degradation and biogenesis. Mitochondria carry two AAA proteases, enzymatic sites of which are in opposite side of the IM; iAAA protease in the IMS and mAAA protease in the matrix. These two AAA proteases are integral membrane proteins themselves in the IM. It has been reported that the transmembrane(TM) domains of mAAA protease is required for integral membrane protein degradation, suggesting that membrane dislocation is essential for degradation as the catalytic domain of mAAA protease is exposed to the matrix side. However, how the mAAA protease recognizes and dislocates integral membrane protein remains unknown. Besides, it is also reported that the activity of mAAA protease is accelerated in cells lacking Prohibitin, a highly conserved protein complex in eukaryotes.
This study aims to elucidate the role of TM domains of mAAA protease on the membrane protein recognition and dislocation. By replacing the TM domains of Yta10 and Yta12, which are homologous subunits of mAAA protease with foreign TM segments, it is observed that when the 2nd TM of mAAA protease subunits are replaced, substrate dislocation is reduced. This suggests that the 2nd TM of mAAA proteases is critical in membrane protein dislocation. We also observed that when Prohibitin is absent, substrate dislocation is increased, implying the involvement of Prohibitin complex in dislocation activity of mAAA, and further investigation is underway.

71

POSTER NUMBER: 1057

Acetate functions as an epigenetic metabolite to promote lipid synthesis under hypoxia