Lit Lunch 5-8-15

Minsoo

1. Cell Res. 2015 May;25(5):621-33. doi: 10.1038/cr.2015.46. Epub 2015 Apr 24.

Deficient plastidic fatty acid synthesis triggers cell death by modulating mitochondrial reactive oxygen species.

Wu J(1), Sun Y(2), Zhao Y(1), Zhang J(1), Luo L(1), Li M(1), Wang J(1), Yu H(1), Liu G(1), Yang L(1), Xiong G(1), Zhou JM(1), Zuo J(1), Wang Y(1), Li J(1).

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Programmed cell death (PCD) is of fundamental importance to development and defense in animals and plants. In plants, a well-recognized form of PCD is hypersensitive response (HR) triggered by pathogens, which involves the generation of reactive oxygen species (ROS) and other signaling molecules. While the mitochondrion is a master regulator of PCD in animals, the chloroplast is known to regulate PCD in plants. **Arabidopsis Mosaic Death 1 (MOD1), an enoyl-acyl
carrier protein (ACP) reductase essential for fatty acid biosynthesis in chloroplasts, negatively regulates PCD in Arabidopsis**. Here we report that **PCD in
mod1 results from accumulated ROS and can be suppressed by mutations in**

mitochondrial complex I components, and that the suppression is confirmed by pharmaceutical inhibition of the complex I-generated ROS. We further show that **intact mitochondria are required for full HR and optimum disease resistance to the Pseudomonas syringae bacteria.** These findings strongly indicate that the ROS generated in the electron transport chain in mitochondria plays a key role in triggering plant PCD and highlight an important role of the communication between chloroplast and mitochondrion in the control of PCD in plants.
2. Cell. 2015 Apr 23;161(3):459-69. doi: 10.1016/j.cell.2015.03.051.

Selective elimination of mitochondrial mutations in the germline by genome editing.

Reddy P(1), Ocampo A(1), Suzuki K(1), Luo J(1), Bacman SR(2), Williams SL(2), Sugawara A(1), Okamura D(1), Tsunekawa Y(3), Wu J(1), Lam D(1), Xiong X(4), Montserrat N(5), Esteban CR(1), Liu GH(6), Sancho-Martinez I(1), Manau D(7), Civico S(7), Cardellach F(8), Del Mar O'Callaghan M(9), Campistol J(9), Zhao H(4), Campistol JM(10), Moraes CT(11), Izpisua Belmonte JC(12).

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Mitochondrial diseases include a group of maternally inherited genetic disorders caused by mutations in mtDNA. In most of these patients, **mutated mtDNA coexists**
with wild-type mtDNA, a situation known as mtDNA heteroplasmy. Here, we report on
a strategy toward preventing germline transmission of mitochondrial diseases by
inducing mtDNA heteroplasmy shift through the selective elimination of mutated
mtDNA. As a proof of concept, we took advantage of NZB/BALB heteroplasmic mice,
which contain two mtDNA haplotypes, BALB and NZB, and selectively prevented their
germline transmission using either mitochondria-targeted restriction
endonucleases or TALENs. In addition, we successfully reduced human mutated mtDNA
levels responsible for Leber's hereditary optic neuropathy (LHOND), and
neurogenic muscle weakness, ataxia, and retinitis pigmentosa (NARP), in mammalian
oocytes using mitochondria-targeted TALEN (mito-TALENs). Our approaches represent

a **potential therapeutic avenue for preventing the transgenerational transmission**
of human mitochondrial diseases caused by mutations in mtDNA.

3. Methods in Molecular Biology. Plant Mitochondria: Methods and protocols
Editors: Whelan, James, Murcha, Monika W. (Eds.)

The chapters compiled in this detailed **collection outline a number of methods used to study plant mitochondria today**, starting from the isolation of mitochondria to detailed analyses of RNA, protein and enzymatic activities. Given that the ability to uncover mitochondria's unique features is underpinned by current methodology, this book explores the subject from morphology to detailed molecular mechanisms. Written in the highly successful Methods in Molecular Biology series format, chapters include introductions to their respective topics, lists of the necessary materials and reagents, step-by-step, readily reproducible laboratory protocols and tips on troubleshooting and avoiding known pitfalls.

Keith
Wild-type Human γD-crystallin Promotes Aggregation of Its Oxidation-mimicking, Misfolding-prone W42Q Mutant

May 1, 2015 The Journal of Biological Chemistry, 290, 11491-11503.

Eugene Serebryany and Jonathan A. King[1]

Department of Biology, Massachusetts Institute of Technology, Cambridge, Massachusetts

Non-native protein conformers generated by mutation or chemical damage template aggregation of wild-type, undamaged polypeptides in diseases ranging from amyotrophic lateral sclerosis to cancer. We tested for such interactions in the natively monomeric human eye lens protein γD-crystallin, whose aggregation leads to cataract disease. The oxidation-mimicking W42Q mutant of γD-crystallin formed

non-native polymers starting from a native-like state under physiological conditions. Aggregation occurred in the temperature range 35–45 °C, in which the mutant protein began to lose the native conformation of its N-terminal domain. Surprisingly, wild-type γD-crystallin promoted W42Q polymerization in a catalytic manner, even at mutant concentrations too low for homogeneous nucleation to occur. The presence of wild-type protein also downshifted the temperature range of W42Q aggregation. W42Q aggregation required formation of a non-native intramolecular disulfide bond but not intermolecular cross-linking. Transient WT/W42Q binding may catalyze this oxidative misfolding event in the mutant. That a more stable variant in a mixture can specifically promote aggregation of a less stable one rationalizes how extensive aggregation of rare damaged polypeptides can occur during the course of aging.

Damian

Plant Journal

The *Chlamydomonas* heat stress response (pages 466–480)

Michael Schroda, Dorothea Hemme and Timo Mühlhaus

Article first published online: 27 MAR 2015 | DOI: 10.1111/tpj.12816

Significance Statement

Heat waves occurring with increased frequency as a result of global climate change threaten crop yield safety. Countermeasures may lie in the genetic engineering of crop plants toward higher thermotolerance, for which a thorough understanding of how plants sense heat and respond to it is imperative. This review gives a comprehensive overview to this issue with a strong focus on data from *Chlamydomonas reinhardtii*.

Cell Press

A Small Molecule Inhibitor of ATPase Activity of HSP70 Induces Apoptosis and Has Antitumor Activities

Sung-Kyun Ko, Jiyeon Kim, Deuk Chae Na, Sookil Park, Seong-Hyun Park, Ji Young Hyun, Kyung-Hwa Baek, Nam Doo Kim, and others

Chemistry & Biology, Vol. 22, Issue 3, p391–403

Published online: March 12, 2015

Hsp70 Forms Antiparallel Dimers Stabilized by Post-translational Modifications to Position Clients for Transfer to Hsp90

Nina Morgner, Carla Schmidt, Victoria Beilsten-Edmands, Ima-obong Ebong, Nisha A. Patel, Eugenia M. Clerico, Elaine Kirschke, Soumya Daturpalli, and others

Cell Reports, Vol. 11, Issue 5, p759–769

Published online: April 23, 2015

Phase Transition of a Disordered Nuage Protein Generates Environmentally Responsive Membraneless Organelles

Timothy J. Nott, Evangelia Petsalaki, Patrick Farber, Dylan Jervis, Eden Fussner, Anne Plochowietz, Timothy D. Craggs, David P. Bazett-Jones, and others

Molecular Cell, Vol. 57, Issue 5, p936–947

Published in issue: March05, 2015

SoNar, a Highly Responsive NAD^+/NADH Sensor, Allows High-Throughput Metabolic Screening of Anti-tumor Agents

Yuzheng Zhao, Qingxun Hu, Feixiong Cheng, Ni Su, Aoxue Wang, Yejun Zou, Hanyang Hu, Xianjun Chen, and others

Cell Metabolism, Vol. 21, Issue 5, p777–789

Published in issue: May05, 2015

Accumulation of Basic Amino Acids at Mitochondria Dictates the Cytotoxicity of Aberrant Ubiquitin

Ralf J. Braun, Cornelia Sommer, Christine Leibiger, Romina J.G. Gentier, Verónica I. Dumit, Katrin Paduch, Tobias Eisenberg, Lukas Habernig, and others

Cell Reports, Vol. 10, Issue 9, p1557–1571

Published online: March 5, 2015

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Fionn

Plant cell

Arabidopsis ROOT PHOTOTROPISM2 Contributes to the Adaptation to High-Intensity Light in Phototropic Responses

Ken Hagaa, Tomoko Tsuchida-Mayamab, Mizuki Yamadac and Tatsuya Sakaic,1

Abstract

Living organisms adapt to changing light environments via mechanisms that enhance photosensitivity under darkness and attenuate photosensitivity under bright light conditions. In hypocotyl phototropism, phototropin1 (phot1) blue light photoreceptors mediate both the pulse light-induced, first positive phototropism and the continuous light-induced, second positive phototropism, suggesting the existence of a mechanism that alters their photosensitivity. Here, we show that light induction of ROOT PHOTOTROPISM2 (RPT2) underlies photosensory adaptation in hypocotyl phototropism of Arabidopsis thaliana. rpt2 loss-of-function mutants exhibited increased photosensitivity to very low fluence blue light but were insensitive to low fluence blue light. Expression of RPT2 prior to phototropic stimulation in etiolated seedlings reduced photosensitivity during first positive phototropism and accelerated second positive phototropism. Our microscopy and

biochemical analyses indicated that blue light irradiation causes dephosphorylation of NONPHOTOTROPIC HYPOCOTYL3 (NPH3) proteins and mediates their release from the plasma membrane. These phenomena correlate closely with the desensitization of phot1 signaling during the transition period from first positive phototropism to second positive phototropism. RPT2 modulated the phosphorylation of NPH3 and promoted reconstruction of the phot1-NPH3 complex on the plasma membrane. We conclude that photosensitivity is increased in the absence of RPT2 and that this results in the desensitization of phot1. Light-mediated induction of RPT2 then reduces the photosensitivity of phot1, which is required for second positive phototropism under bright light conditions.

Nathen

The massive mitochondrial genome of the angiosperm Silene noctiflora is evolving by gain or loss of entire chromosomes

Zhiqiang Wua , Jocelyn M. Cuthberta , Douglas R. Taylorb , and Daniel B. Sloana,1 a Department of Biology, Colorado State University, Fort Collins, CO 80523; and b Department of Biology, University of Virginia, Charlottesville, VA 22904 Edited by John P. McCutcheon, University of Montana, Missoula, MT, and accepted by the Editorial Board February 11, 2015 (received for review December 3, 2014)

Across eukaryotes, mitochondria exhibit staggering diversity in genomic architecture, including the repeated evolution of multichromosomal structures. Unlike in the nucleus, where mitosis and meiosis ensure faithful transmission of chromosomes, the mechanisms of inheritance in fragmented mitochondrial genomes remain mysterious. Multichromosomal mitochondrial genomes have recently been found in multiple species of flowering plants, including Silene noctiflora, which harbors an unusually large and complex mitochondrial genome with more than 50 circular-mapping chromosomes totaling ∼7 Mb in size. To determine the extent to which such genomes are stably maintained, we analyzed intraspecific variation in the mitochondrial genome of S. noctiflora. Complete

genomes from two populations revealed a high degree of similarity in the sequence, structure, and relative abundance of mitochondrial chromosomes. For example, there are no inversions between the genomes, and there are only nine SNPs in 25 kb of protein-coding sequence. Remarkably, however, these genomes differ in the presence or absence of 19 entire chromosomes, all of which lack any identifiable genes or contain only duplicate gene copies. Thus, these mitochondrial genomes retain a full gene complement but carry a highly variable set of chromosomes that are filled with presumably dispensable sequence. In S. noctiflora, conventional mechanisms of mitochondrial sequence divergence are being outstripped by an apparently nonadaptive process of whole-chromosome gain/loss, highlighting the inherent challenge in maintaining a fragmented genome. We discuss the implications of these findings in relation to the question of why mitochondria, more so than plastids and bacterial endosymbionts, are prone to the repeated evolution of multichromosomal genomes.

Elizabeth Vierling

Nature: Volume 520 Number 7549 pp585-716

Structure of the human 80S ribosome

Heena Khatter, Alexander G. Myasnikov, S. Kundhavai Natchiar & Bruno P. Klaholz

The structure of the human ribosome at high resolution has been solved; by combining single-particle cryo-EM and atomic model building, local resolution of 2.9 Å was achieved within the most stable areas of the structure.

NIK1-mediated translation suppression functions as a plant antiviral immunity mechanism

Cristiane Zorzatto, João Paulo B. Machado, Kênia V. G. Lopes, Kelly J. T. Nascimento, Welison A. Pereira+ et al.

A new mechanism that plants use to combat begomoviruses—one of the most pathogenic groups of plant viruses, causing severe disease in major crops worldwide—is uncovered: plants inhibit the transcription of genes associated with the translational apparatus, thus causing a general reduction in protein synthesis.

Theoretical perspectives on nonnative interactions and intrinsic disorder in protein folding and binding Review Article *Pages 32-42* Tao Chen, Jianhui Song, Hue Sun Chan

doi:10.1016/j.cell.2015.03.018

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Cell

Rapid Elimination of the Persistent Synergid through a Cell Fusion Mechanism

Daisuke Maruyama[1, 2, 3, 4,], Ronny Völz[5, 6], Hidenori Takeuchi[7], Toshiyuki Mori[8], Tomoko Igawa[9], Daisuke Kurihara[7], Tomokazu Kawashima[4, 10], Minako Ueda[1], Masaki Ito[11], Masaaki Umeda[12, 13], Shuh-ichi Nishikawa[14], Rita Groß-Hardt[5, 15], Tetsuya Higashiyama[1, 2, 7]

See also: Stefanie Sprunck, Thomas Dresselhaus

Three Cell Fusions during Double Fertilization *Cell, Volume 161, Issue 4, 7 May 2015, Pages 708-709*

Highlights

•The persistent synergid cell fuses with the endosperm after fertilization ; Synergid cytoplasm containing pollen tube attractants is diluted by the fusion; •FIS-PRC2 is involved in mitosis-associated synergid nuclear elimination; •Each female gamete independently controls the cell-fusion and ethylene signaling

In flowering plants, fertilization-dependent degeneration of the persistent synergid cell ensures one-on-one pairings of male and female gametes. Here, we report that the fusion of the persistent synergid cell and the endosperm selectively inactivates the persistent synergid cell in *Arabidopsis thaliana*. The synergid-endosperm fusion causes rapid dilution of pre-secreted pollen tube attractant in the persistent synergid cell and selective disorganization of the synergid nucleus during the endosperm proliferation, preventing attractions of excess number of pollen tubes (polytubey). The synergid-endosperm fusion is induced by fertilization of the central cell, while the egg cell fertilization predominantly activates ethylene signaling, an inducer of the synergid nuclear

disorganization. Therefore, two female gametes (the egg and the central cell) control independent pathways yet coordinately accomplish the elimination of the persistent synergid cell by double fertilization.

Cell
Volume 161, Issue 4, 7 May 2015, Pages 845–857

Structural Snapshots of Actively Translating Human Ribosomes

Elmar Behrmann[1, 6, 7], Justus Loerke[1, 6], Tatyana V. Budkevich[1], Kaori Yamamoto[1], Andrea Schmidt[1, 2], Pawel A. Penczek[3], Matthijn R. Vos[4], Jörg Bürger[1], Thorsten Mielke[1, 5], Patrick Scheerer[1, 2], Christian M.T. Spahn[1, ,]

Structural analysis of actively translating ribosomes
Identification of significantly populated states at close to in vivo conditions
Functional states feature localized chemical and conformational heterogeneity
Human 80S ribosome map at near-atomic resolution reveals native interactions

Macromolecular machines, such as the ribosome, undergo large-scale conformational changes during their functional cycles. Although their mode of action is often compared to that of mechanical machines, a crucial difference is that, at the molecular dimension, thermodynamic effects dominate functional cycles, with proteins fluctuating stochastically between functional states defined by energetic minima on an energy landscape. Here, we have used cryo-electron microscopy to image ex-vivo-derived human polysomes as a source of actively translating ribosomes. Multiparticle refinement and 3D variability analysis allowed us to visualize a variety of native translation intermediates. Significantly populated states include not only elongation cycle intermediates in pre- and post-translocational states, but also eEF1A-containing decoding and termination/recycling complexes. Focusing on the post-translocational state, we extended this assessment to the single-residue level, uncovering striking details of ribosome-ligand interactions and identifying both static and functionally important dynamic elements.

Volume 161, Issue 4, 7 May 2015, Pages 858–867

Mitochondrial ClpX Activates a Key Enzyme for Heme Biosynthesis and Erythropoiesis

Julia R. Kardon[1, 7], Yvette Y. Yien[2], Nicholas C. Huston[2], Diana S. Branco[2], Gordon J. Hildick-Smith[2, 8], Kyu Y. Rhee[3, 4], Barry H. Paw[2, 5, 6], Tania A. Baker[1, 7, ,]

The mitochondrial ClpX unfoldase is required for efficient heme biosynthesis

mtClpX activates a key enzyme in heme biosynthesis by catalyzing cofactor binding

mtClpX activates ALAS without committing it to degradation by mtClpP

mtClpX is important for erythropoiesis when demand for heme is high

The mitochondrion maintains and regulates its proteome with chaperones primarily inherited from its bacterial endosymbiont ancestor. Among these chaperones is the AAA+ unfoldase ClpX, an important regulator of prokaryotic physiology with poorly defined function in the eukaryotic mitochondrion. We observed phenotypic similarity in *S. cerevisiae* genetic interaction data between mitochondrial ClpX (mtClpX) and genes contributing to heme biosynthesis, an essential mitochondrial function. Metabolomic analysis revealed that 5-aminolevulinic acid (ALA), the first heme precursor, is 5-fold reduced in yeast lacking mtClpX activity and that total heme is reduced by half. mtClpX directly stimulates ALA synthase in vitro by catalyzing incorporation of its cofactor, pyridoxal phosphate. This activity is conserved in mammalian homologs; additionally, mtClpX depletion impairs vertebrate erythropoiesis, which requires massive upregulation of heme biosynthesis to supply hemoglobin. mtClpX, therefore, is a widely conserved stimulator of an essential biosynthetic pathway and uses a previously unrecognized mechanism for AAA+ unfoldases.

Volume 161, Issue 4, 7 May 2015, Pages 710–713

Minireview

An Adenine Code for DNA: A Second Life for N6-Methyladenine

Holger Heyn[1], Manel Esteller[1, 2, 3, ,]

DNA N6-methyladenine (6mA) protects against restriction enzymes in bacteria. However, isolated reports have suggested additional activities and its presence in other organisms, such as unicellular eukaryotes. New data now find that 6mA may have a gene regulatory function in green alga, worm, and fly, suggesting m6A as a potential “epigenetic” mark.

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