

Patrick

Science:

- A subcellular map of the human proteome (www.proteinatlas.org), Science 11 May 2017: DOI: 10.1126/science.aal3321
- Mysterious unchanging DNA finds a purpose in life, Science 02 Jun 2017: Vol. 356, Issue 6341, pp. 892 DOI: 10.1126/science.356.6341.892

1.

Front. Plant Sci., 11 April 2017 | <https://doi.org/10.3389/fpls.2017.00524> [Titel anhand dieser DOI in Citavi-Projekt übernehmen]

Seed Biology Updates – Highlights and New Discoveries in Seed Dormancy and Germination Research

Hiroyuki Nonogaki*

Department of Horticulture, Oregon State University, Corvallis, OR, USA

2.

S-nitrosylation triggers ABI5 degradation to promote seed germination and seedling growth

Pablo Albertos, María C. Romero-Puertas, Kiyoshi Tatematsu, Isabel Mateos, Inmaculada Sánchez-Vicente, Eiji Nambara & Oscar Lorenzo

Nature Communications 6, Article number: 8669 (2015)

doi:10.1038/ncomms9669

Minsoo

1. Brain. 2017 Jun 1;140(6):1595-1610. doi: 10.1093/brain/awx094.

ATAD3 gene cluster deletions cause cerebellar dysfunction associated with altered mitochondrial DNA and cholesterol metabolism.

Desai R(1), Frazier AE(2), Durigon R(3), Patel H(4), Jones AW(3), Dalla Rosa I(3), Lake NJ(2), Compton AG(2), Mountford HS(2), Tucker EJ(2), Mitchell ALR(3), Jackson D(4), Sesay A(4), Di Re M(5), van den Heuvel LP(6), Burke D(7), Francis D(8), Lunke S(8),(9), McGillivray G(1),(8), Mandelstam S(2),(10),(11), Mochel F(12),(13), Keren B(13),(14), Jardel C(14),(15), Turner AM(16),(17), Ian Andrews P(17),(18), Smeitink J(6), Spelbrink JN(6), Heales SJ(7),(19), Kohda M(20), Ohtake A(21), Murayama K(22), Okazaki Y(20),(23), Lombès A(1),(6), Holt JJ(1),(3),(24), Thorburn DR(2),(8), Spinazzola A(3),(25).

Author information:

(1)MRC Laboratory, Mill Hill, London NW71AA, UK. (2)Murdoch Childrens Research Institute, Royal Children's Hospital and Department of Paediatrics, University of Melbourne, Melbourne VIC 3052, Australia. (3)Department of Clinical Neurosciences, Institute of Neurology, Royal Free Campus, University College

London, NW3 2PF, UK. (4)Bioinformatics and Biostatistics, Francis Crick Institute, 1 Midland Road, London NW1 1AT, UK. (5)Mitochondrial Biology Unit, Hills Road, Cambridge, CB2 0XY, UK. (6)Radboud Center for Mitochondrial Medicine, Radboud University Medical Center, Nijmegen, The Netherlands. (7)Department of Genetics and Genomic Medicine, Institute of Child Health, University College London, London, UK and Laboratory Medicine, Great Ormond Street Hospital, London, UK. (8)Victorian Clinical Genetics Services, Murdoch Children's Research Institute, Melbourne VIC 3052, Australia. (9)Department of Pathology, University of Melbourne, Melbourne 3052, Australia. (10)The Florey Institute of Neuroscience and Mental Health Melbourne, Australia. (11)Departments of Radiology and Paediatrics, University of Melbourne, Melbourne, Australia. (12)AP-HP, Department of Genetics, GHU Pitié-Salpêtrière, Paris, F-75651 France. (13)Inserm U975; CNRS UMR 7225, ICM; F-75013, Paris, France. (14)AP-HP, Service de Biochimie Métabolique et Centre de Génétique moléculaire et chromosomique, GHU Pitié-Salpêtrière, Paris, F-75651 France. (15)Inserm U1016; CNRS UMR 8104; Université Paris-Descartes-Paris 5; Institut Cochin, 75014 Paris, France. (16)Department of Clinical Genetics, Sydney Children's Hospital, Sydney, NSW, Australia. (17)School of Women's and Children's Health, University of New South Wales, Kensington, NSW, Australia. (18)Department of Paediatric Neurology, Sydney Children's Hospital, Sydney, NSW, Australia. (19)Department of Molecular Neuroscience, Institute of Neurology, University College London, Queen Square, London, UK. (20)Division of Translational Research, Research Center for Genomic Medicine, Saitama Medical University, Hidaka-shi, Saitama, Japan. (21)Department of Pediatrics, Saitama Medical University, Moroyama-machi, Iruma-gun, Saitama, Japan. (22)Department of Metabolism, Chiba Children's Hospital, Chiba, Japan. (23)Division of Functional Genomics and Systems Medicine, Research Center for Genomic Medicine, Saitama Medical University, Hidaka-shi, Saitama, Japan. (24)Biodonostia Health Research Institute, 20014 San Sebastián, Spain. IKERBASQUE, Basque Foundation for Science, 48013 Bilbao, Spain. (25)MRC Centre for Neuromuscular Diseases, UCL Institute of Neurology and National Hospital for Neurology and Neurosurgery, Queen Square, London WC1N 3BG, UK.

Although mitochondrial disorders are clinically heterogeneous, they frequently involve the central nervous system and are among the most common neurogenetic disorders. Identifying the causal genes has benefited enormously from advances in high-throughput sequencing technologies; however, once the defect is known, researchers face the challenge of deciphering the underlying disease mechanism. Here we characterize large biallelic deletions in the region encoding the ATAD3C, ATAD3B and ATAD3A genes. Although high homology complicates genomic analysis of the ATAD3 defects, they can be identified by targeted analysis of standard single nucleotide polymorphism array and whole exome sequencing data. We report deletions that generate chimeric ATAD3B/ATAD3A fusion genes in individuals from four unrelated families with fatal congenital pontocerebellar hypoplasia, whereas a case with genomic rearrangements affecting the ATAD3C/ATAD3B genes on one allele and ATAD3B/ATAD3A genes on the other displays later-onset encephalopathy with cerebellar atrophy, ataxia and dystonia. Fibroblasts from affected individuals display mitochondrial DNA abnormalities, associated with multiple indicators of altered cholesterol metabolism. Moreover, drug-induced perturbations of cholesterol homeostasis cause mitochondrial DNA disorganization in control cells, while mitochondrial DNA aggregation in the genetic cholesterol

trafficking disorder Niemann-Pick type C disease further corroborates the interdependence of mitochondrial DNA organization and cholesterol. These data demonstrate the integration of mitochondria in cellular cholesterol homeostasis, in which ATAD3 plays a critical role. The dual problem of perturbed cholesterol metabolism and mitochondrial dysfunction could be widespread in neurological and neurodegenerative diseases.

2. Cell. 2017 Jun 1;169(6):1142-1155.e12. doi: 10.1016/j.cell.2017.04.032. Epub 2017 May 18.

Bypassing Negative Epistasis on Yield in Tomato Imposed by a Domestication Gene.

Soyk S(1), Lemmon ZH(1), Oved M(2), Fisher J(2), Liberatore KL(3), Park SJ(4), Goren A(5), Jiang K(1), Ramos A(6), van der Knaap E(6), Van Eck J(7), Zamir D(2), Eshed Y(5), Lippman ZB(8).

Author information:

(1)Cold Spring Harbor Laboratory, Cold Spring Harbor, NY 11724, USA. (2)Faculty of Agriculture, Hebrew University of Jerusalem, Rehovot 76100, Israel. (3)Cold Spring Harbor Laboratory, Cold Spring Harbor, NY 11724, USA; Watson School of Biological Sciences, Cold Spring Harbor Laboratory, Cold Spring Harbor, NY 11724, USA. (4)Division of Biological Sciences and Research Institute for Basic Science, Wonkwang University, Iksan, Jeonbuk 54538, Rep. of Korea. (5)Department of Plant and Environmental Sciences, Weizmann Institute of Science, Rehovot 76100, Israel. (6)Institute of Plant Breeding, Genetic & Genomics, University of Georgia, Athens, GA 30602, USA. (7)The Boyce Thompson Institute, Ithaca, NY 14853, USA. (8)Cold Spring Harbor Laboratory, Cold Spring Harbor, NY 11724, USA; Watson School of Biological Sciences, Cold Spring Harbor Laboratory, Cold Spring Harbor, NY 11724, USA. Electronic address: lippman@cshl.edu.

Selection for inflorescence architecture with improved flower production and yield is common to many domesticated crops. However, tomato inflorescences resemble wild ancestors, and breeders avoided excessive branching because of low fertility. We found branched variants carry mutations in two related transcription factors that were selected independently. One founder mutation enlarged the leaf-like organs on fruits and was selected as fruit size increased during domestication. The other mutation eliminated the flower abscission zone, providing "jointless" fruit stems that reduced fruit dropping and facilitated mechanical harvesting. Stacking both beneficial traits caused undesirable branching and sterility due to epistasis, which breeders overcame with suppressors. However, this suppression restricted the opportunity for productivity gains from weak branching. Exploiting natural and engineered alleles for multiple family members, we achieved a continuum of inflorescence complexity that allowed breeding of higher-yielding hybrids. Characterizing and neutralizing similar cases of negative epistasis could improve productivity in many agricultural organisms.

lan

[Free Radic Biol Med.](#) 2017 May 19;110:19-30. doi: 10.1016/j.freeradbiomed.2017.05.014. [Epub ahead of print]

Chronicles of a reductase: Biochemistry, genetics and physio-pathological role of GSNOR.

[Rizza S](#)¹, [Filomeni G](#)².

Corey

MtLAX2, a Functional Homologue of the Arabidopsis Auxin Influx Transporter AUX1, Is Required for Nodule Organogenesis

Most legume plants can form nodules, specialized lateral organs that form on roots, and house nitrogen-fixing bacteria collectively called rhizobia. The uptake of the phytohormone auxin into cells is known to be crucial for development of lateral roots. To test the role of auxin influx in nodulation we used the auxin influx inhibitors 1-naphthoxyacetic acid ([1-NOA](#)) and 2-NOA, which we found reduced nodulation of *Medicago truncatula*. This suggested the possible involvement of the AUX/LAX family of auxin influx transporters in nodulation. Gene expression studies identified MtLAX2, a paralogue of Arabidopsis (*Arabidopsis thaliana*) AUX1, as being induced at early stages of nodule development. MtLAX2 is expressed in nodule primordia, the vasculature of developing nodules, and at the apex of mature nodules. The MtLAX2 promoter contains several auxin response elements, and treatment with indole-acetic acid strongly induces MtLAX2 expression in roots. *mtlax2* mutants displayed root phenotypes similar to Arabidopsis *aux1* mutants, including altered root gravitropism, fewer lateral roots, shorter root hairs, and auxin resistance. In addition, the activity of the synthetic DR5-GUS auxin reporter was strongly reduced in *mtlax2* roots. Following inoculation with rhizobia, *mtlax2* roots developed fewer nodules, had decreased DR5-GUS activity associated with infection sites, and had decreased expression of the early auxin responsive gene ARF16a. Our data indicate that MtLAX2 is a functional analog of Arabidopsis AUX1 and is required for the accumulation of auxin during nodule formation in tissues underlying sites of rhizobial infection.

Keith

Specific Sequences in the N-terminal Domain of Human Small Heat Shock Protein HSPB6 Dictate Preferential Heterooligomerization with the Orthologue HSPB1.

[J Biol Chem.](#) 2017 May 9.

[Heirbaut M](#)¹, [Lermyte F](#)², [Martin EM](#)³, [Beelen S](#)¹, [Sobott F](#)², [Strelkov SV](#)¹, [Weeks SD](#)⁴.

1 KU Leuven, Belgium

2 University of Antwerp, Belgium.

3 University of Leeds, United Kingdom.

4 KU Leuven, Belgium

Small heat shock proteins (sHSPs) are a conserved group of molecular chaperones with important roles in cellular proteostasis. Although sHSPs are characterized by their small monomeric weight, they typically assemble into large polydisperse oligomers that vary in both size and shape but are principally composed of dimeric building blocks. These assemblies can comprise different sHSP orthologues, creating additional complexity that may affect chaperone activity. However, the structural and functional properties of such heterooligomers are poorly understood. We became interested in heterooligomer formation between human heat shock protein family B (small) member 1 (HSPB1) and HSPB6, which are both highly expressed in skeletal muscle. When mixed in vitro, these two sHSPs form a polydisperse oligomer array composed solely of heterodimers, suggesting preferential association that is determined at the monomer level. Previously, we have shown that the sHSP N-terminal domains (NTDs), which have a high degree of intrinsic disorder, are essential for the biased formation. Here we employed iterative deletion mapping to elucidate how the NTD of HSPB6 influences its preferential association with HSPB1 and show that this region has multiple roles in this process. First, the highly conserved motif RLFDQxFG is necessary for subunit exchange among oligomers. Second, a site approximately 20 residues downstream of this motif determines the size of the resultant heterooligomers. Third, a region unique to HSPB6 dictates the preferential formation of heterodimers. In conclusion, the disordered NTD of HSPB6 helps regulate the size and stability of heterooligomeric complexes, indicating that terminal sHSP regions define the assembly properties of these proteins.

Elizabeth:

Malinverni D, Jost Lopez A, De Los Rios P, Hummer G, Barducci A.
Modeling Hsp70/Hsp40 interaction by multi-scale molecular simulations and co-evolutionary sequence analysis.

Elife. 2017 May 12;6. [Epub ahead of print]

PMID: 28498104 [PubMed - as supplied by publisher]

Brodehl A, Gaertner-Rommel A, Klauke B, Grewe SA, Schirmer I, Peterschröder A, Faber L, Vorgerd M, Gummert J, Anselmetti D, Schulz U, Paluszkiwicz L, Milting H.

The novel Î±B-Crystallin (CRYAB) mutation p.D109G causes restrictive cardiomyopathy.

Hum Mutat. 2017 May 11;. [Epub ahead of print]

PMID: 28493373 [PubMed - as supplied by publisher]

French K, Robinson SA, Lia J.

Thermotolerance capacities of native and exotic coastal plants will lead to changes in species composition under increased heat waves.

Conserv Physiol. 2017;5(1):cox029.

PMID: 28491321 [PubMed - in process]

Mitchell Sontag E, Samant RS, Frydman J.

Mechanisms and Functions of Spatial Protein Quality Control.

Annu Rev Biochem. 2017 May 10;. [Epub ahead of print]

PMID: 28489421 [PubMed - as supplied by publisher]

Heirbaut M, Lermyte F, Martin EM, Beelen S, Sobott F, Strelkov SV, Weeks SD.
Specific Sequences in the N-terminal Domain of Human Small Heat Shock Protein HSPB6 Dictate Preferential Heterooligomerization with the Orthologue HSPB1.
J Biol Chem. 2017 May 9;. [Epub ahead of print]
PMID: 28487364 [PubMed - as supplied by publisher]

Hu X, Van Marion DMS, Wiersma M, Zhang D, Brundel BJJM.
The protective role of small heat shock proteins in cardiac diseases: key role in atrial fibrillation.
Cell Stress Chaperones. 2017 May 8;. [Epub ahead of print]
PMID: 28484965 [PubMed - as supplied by publisher]

Gorkovskiy A, Reidy M, Masison DC, Wickner RB.
Hsp104 disaggregase at normal levels cures many [α PSI⁺] prion variants in a process promoted by Sti1p, Hsp90, and Sis1p.
Proc Natl Acad Sci U S A. 2017 May 8;. [Epub ahead of print]
PMID: 28484020 [PubMed - as supplied by publisher]

Xu J, Driedonks N, Rutten MJM, Vriezen WH, de Boer GJ, Rieu I.
Mapping quantitative trait loci for heat tolerance of reproductive traits in tomato (*Solanum lycopersicum*).
Mol Breed. 2017;37(5):58.
PMID: 28479863 [PubMed - in process]

Lemire BD.
Evolution, structure and membrane association of NDUFAF6, an assembly factor for NADH:ubiquinone oxidoreductase (Complex I).
Mitochondrion. 2017 May 2;. [Epub ahead of print]
PMID: 28476317 [PubMed - as supplied by publisher]

Oancea A, Georgescu E, Georgescu F, Nicolescu A, Oprita EI, Tudora C, Vladulescu L, Vladulescu MC, Oancea F, Deleanu C.
Isoxazole derivatives as new nitric oxide elicitors in plants.
Beilstein J Org Chem. 2017;13:659-664.
PMID: 28487760 [PubMed]

Li X, Zhang L, Ahammed GJ, Li ZX, Wei JP, Shen C, Yan P, Zhang LP, Han WY.
Nitric oxide mediates brassinosteroid-induced flavonoid biosynthesis in *Camellia sinensis* L.
J Plant Physiol. 2017 Apr 12;214:145-151. [Epub ahead of print]
PMID: 28482335 [PubMed - as supplied by publisher]

Mancera-Martínez E, Brito Querido J, Valasek LS, Simonetti A, Hashem Y.
ABCE1: A special factor that orchestrates translation at the crossroad between recycling and initiation.
RNA Biol. 2017 May 12;:0. [Epub ahead of print]
PMID: 28498001 [PubMed - as supplied by publisher]

Prabhakar A, Choi J, Wang J, Petrov A, Puglisi JD.
Dynamic basis of fidelity and speed in translation: Coordinated multi-step mechanisms of elongation and termination.
Protein Sci. 2017 May 8;. [Epub ahead of print]
PMID: 28480640 [PubMed - as supplied by publisher]

Liu K, Rehfus JE, Mattson E, Kaiser C.

The ribosome destabilizes native and non-native structures in a nascent multi-domain protein.

Protein Sci. 2017 May 5;. [Epub ahead of print]

PMID: 28474852 [PubMed - as supplied by publisher]

Melkina OE, Khmel IA, Plyuta VA, Koksharova OA, Zavilgelsky GB.

Ketones 2-heptanone, 2-nonanone, and 2-undecanone inhibit DnaK-dependent refolding of heat-inactivated bacterial luciferases in *Escherichia coli* cells lacking small chaperon IbpB.

Appl Microbiol Biotechnol. 2017 Jun 3;. [Epub ahead of print]

PMID: 28577028 [PubMed - as supplied by publisher]

Kumar RR, Goswami S, Shamim M, Dubey K, Singh K, Singh S, Kala YK, Niraj RRK, Sakhrey A, Singh GP, Grover M, Singh B, Rai GK, Rai AK, Chinnusamy V, Praveen S.

Exploring the heat-responsive chaperones and microsatellite markers associated with terminal heat stress tolerance in developing wheat.

Funct Integr Genomics. 2017 Jun 1;. [Epub ahead of print]

PMID: 28573536 [PubMed - as supplied by publisher]

Ko E, Kim M, Park Y, Ahn YJ.

Heterologous Expression of the β Carrot Hsp17.7 gene Increased Growth, Cell Viability, and Protein Solubility in Transformed Yeast (*Saccharomyces cerevisiae*) under Heat, Cold, Acid, and Osmotic Stress Conditions.

Curr Microbiol. 2017 Jun 1;. [Epub ahead of print]

PMID: 28573339 [PubMed - as supplied by publisher]

Wang X, Wang R, Ma C, Shi X, Liu Z, Wang Z, Sun Q, Cao J, Xu S.

Massive expansion and differential evolution of small heat shock proteins with wheat (*Triticum aestivum* L.) polyploidization.

Sci Rep. 2017 May 31;7(1):2581.

PMID: 28566710 [PubMed - in process]

Lazarev VF, Mikhaylova ER, Guzhova IV, Margulis BA.

Possible Function of Molecular Chaperones in Diseases Caused by Propagating Amyloid Aggregates.

Front Neurosci. 2017;11:277.

PMID: 28559794 [PubMed - in process]

O'Neil PK, Richardson LGL, Paila YD, Piszczek G, Chakravarthy S, Noinaj N, Schnell D.

The POTRA domains of Toc75 exhibit chaperone-like function to facilitate import into chloroplasts.

Proc Natl Acad Sci U S A. 2017 May 30;. [Epub ahead of print]

PMID: 28559331 [PubMed - as supplied by publisher]

Zhang ZW, Luo S, Zhang GC, Feng LY, Zheng C, Zhou YH, Du JB, Yuan M, Chen YE, Wang CQ, Liu WJ, Xu XC, Hu Y, Bai SL, Kong DD, Yuan S, He YK.

Nitric oxide induces monosaccharide accumulation through enzyme S-nitrosylation.

Plant Cell Environ. 2017 May 26;. [Epub ahead of print]

PMID: 28556250 [PubMed - as supplied by publisher]

Plant, Cell & Environment Content Alert: 40, 7 (July 2017)

The occurrence and control of nitric oxide generation by the plant mitochondrial electron transport chain (pages 1074–1085)

Nicole A. Alber, Hampavi Sivanesan and Greg C. Vanlerberghe

Version of Record online: 27 MAR 2017 | DOI: 10.1111/pce.12884

Abstract

Nitric oxide has emerged as an important plant stress-signalling molecule but the pathways responsible for nitric oxide synthesis and scavenging remain relatively poorly understood. We provide evidence that electron pressure in the Q-cycle of Complex III of the mitochondrial electron transport chain can result in the generation of nitric oxide from nitrite. Further, alternative oxidase, by acting as a non-energy-conserving electron sink upstream of the Q-cycle, is able to reduce this electron pressure and hence nitric oxide generation. This places alternative oxidase as a potentially key regulator of nitric oxide signalling from the plant mitochondrion.

PlantCell

Noncanonical Alternative Polyadenylation Contributes to Gene Regulation in Response to Hypoxia

Laura de Lorenzo, Reed Sorenson, Julia Bailey-Serres, and Arthur G. Hunt

Plant Cell 2017 tpc.16.00746; Advance Publication May 30, 2017; doi:10.1105/tpc.16.00746 **OPEN**

<http://www.plantcell.org/content/early/2017/05/30/tpc.16.00746.abstract>

EMBO J

Mfn2 is critical for brown adipose tissue thermogenic function

Marie Boutant, Sameer S Kulkarni, Magali Joffraud, Joanna Ratajczak,

Miriam Valera-Alberni, Roy Combe, Antonio Zorzano, and Carles Cantó

Published online 27.03.2017

Mitofusin 2 (Mfn2) is critical for brown adipose tissue thermogenic function and participates in the functional relationship between mitochondria and lipid droplets.

<http://EMBOJ.embopress.org/content/36/11/1543?etoc>

ER-mitochondria contacts control surface glycan expression and

sensitivity to killer lymphocytes in glioma stem-like cells

Esen Yonca Basso, Atsuko Kasahara, Valentina Chiusolo, Guillaume

Jacquemin, Emma Boydell, Sebastian Zamorano, Cristina Riccadonna, Serena

Pellegatta, Nicolas Hulo, Valérie Dutoit, Madiha Derouazi, Pierre Yves

Dietrich, Paul R Walker, and Denis Martinvalet

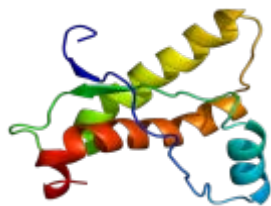
Published online 10.03.2017

The increased immune sensitivity of glioma stem-like cells is linked to mitochondrial morphology and dynamics, which affect presentation of sialylated glycan markers.

<http://EMBOJ.embopress.org/content/36/11/1493?etoc>

BMC Biology

Why is prion protein so highly conserved



The function of prion protein is less obvious than its role in disease. Adriano Aguzzi and colleagues review what we do know, and how the introduction of rigorously controlled animal models might clarify what we don't.

REVIEW

The biological function of the cellular prion protein: an update

Marie-Angela Wulf, Assunta Senatore and Adriano Aguzzi

Plant Cell and Environment

Strigolactones, karrikins and beyond

Carolien De Cuyper, Sylwia Struk, Lukas Braem, Kris Gevaert, Geert De Jaeger and Sofie Goormachtig
Accepted manuscript online: 30 MAY 2017 12:35PM EST | DOI: 10.1111/pce.12996

Strigolactones are involved in various aspects of plant development and (a)biotic stress responses. Currently, the general picture of strigolactone signaling has been depicted. One striking feature is that this signaling shares components with that of the smoke-derived karrikins or plant-derived karrikin-like compounds. Here, we aim at untangling both pathways and discuss their importance in diverse aspects of plant development.

Nitric oxide induces monosaccharide accumulation through enzyme S-nitrosylation

Zhong-Wei Zhang, Sha Luo, Gong-Chang Zhang, Ling-Yang Feng, Chong Zheng, Yang-Hong Zhou, Jun-Bo Du, Ming Yuan, Yang-Er Chen, Chang-Quan Wang, Wen-Juan Liu, Xiao-Chao Xu, Yong Hu, Su-Lan Bai, Dong-Dong Kong, Shu Yuan and Yi-Kun He

Accepted manuscript online: 26 MAY 2017 04:45PM EST | DOI: 10.1111/pce.12989

The physiological effects of NO can be largely reversed by high-level (either exogenous or endogenous) sucrose treatments. NO significantly inhibits polysaccharide synthesis (by S-nitrosylation of ATP synthase and therefore decreasing ATP, ADP-glucose and UDP-glucose levels) and monosaccharide catabolism by modulating sugar metabolic enzymes (including pyruvate dehydrogenase) through S-nitrosylation. Therefore, cellular monosaccharides (glucose and fructose) accumulated substantially after NO treatments, which enhanced plant's sweetness.

Mitochondria link metabolism and epigenetics in haematopoiesis pp589 - 591

John C. Schell and Jared Rutter

doi: 10.1038/ncb3540

Due to their varied metabolic and signalling roles, mitochondria are important in mediating cell behaviour. By altering mitochondrial function, two studies now identify metabolite-induced epigenetic changes that have profound effects on haematopoietic stem cell fate and function.

Nature Cell Biology 19,626–638(2017)doi:10.1038/ncb3527

Advances in genomic profiling present new challenges of explaining how changes in DNA and RNA are translated into proteins linking genotype to phenotype. Here we compare the genome-scale proteomic and transcriptomic changes in human primary haematopoietic stem/progenitor cells and erythroid progenitors, and uncover pathways related to mitochondrial biogenesis enhanced through post-transcriptional regulation. Mitochondrial factors including TFAM and PHB2 are selectively regulated through protein translation during erythroid specification. Depletion of TFAM in erythroid cells alters intracellular metabolism, leading to elevated histone acetylation, deregulated gene expression, and defective mitochondria and erythropoiesis. Mechanistically, mTORC1 signalling is enhanced to promote translation of mitochondria-associated transcripts through TOP-like motifs. Genetic and pharmacological perturbation of

mitochondria or mTORC1 specifically impairs erythropoiesis *in vitro* and *in vivo*. Our studies support a mechanism for post-transcriptional control of erythroid mitochondria and may have direct relevance to haematologic defects associated with mitochondrial diseases and ageing.

Article preview [View full access options](#)

Nature Cell Biology | Article

The mitochondrial respiratory chain is essential for haematopoietic stem cell function

[Elena Ansó](#), [Samuel E. Weinberg](#), [Lauren P. Diebold](#), [Benjamin J. Thompson](#), [Sébastien Malinge](#), [Paul T. Schumacker](#), [Xin Liu](#), [Yuannyu Zhang](#), [Zhen Shao](#), [Mya Steadman](#), [Kelly M. Marsh](#), [Jian Xu](#), [John D. Crispino](#) & [Navdeep S. Chandel](#)

Nature Cell Biology 19,614–625(2017)doi:10.1038/ncb3529

Adult and fetal haematopoietic stem cells (HSCs) display a glycolytic phenotype, which is required for maintenance of stemness; however, whether mitochondrial respiration is required to maintain HSC function is not known. Here we report that loss of the mitochondrial complex III subunit Rieske iron-sulfur protein (RISP) in fetal mouse HSCs allows them to proliferate but impairs their differentiation, resulting in anaemia and prenatal death. RISP-null fetal HSCs displayed impaired respiration resulting in a decreased NAD^+/NADH ratio. RISP-null fetal HSCs and progenitors exhibited an increase in both DNA and histone methylation associated with increases in 2-hydroxyglutarate (2HG), a metabolite known to inhibit DNA and histone demethylases. RISP inactivation in adult HSCs also impaired respiration resulting in loss of quiescence concomitant with severe pancytopenia and lethality. Thus, respiration is dispensable for adult or fetal HSC proliferat