Indu

1: Nelson BR, Makarewich CA, Anderson DM, Winders BR, Troupes CD, Wu F, Reese AL,
McAnally JR, Chen X, Kavalali ET, Cannon SC, Houser SR, Bassel-Duby R, Olson EN. Muscle physiology. A peptide encoded by a transcript annotated as long noncoding RNA enhances SERCA activity in muscle. Science. 2016 Jan 15;351(6270):271-5. doi: 10.1126/science.aad4076. PubMed PMID: 26816378.

2: Toyama EQ, Herzig S, Courchet J, Lewis TL Jr, Losón OC, Hellberg K, Young NP, Chen H, Polleux F, Chan DC, Shaw RJ. Metabolism. AMP-activated protein kinase mediates mitochondrial fission in response to energy stress. Science. 2016 Jan 15;351(6270):275-81. doi: 10.1126/science.aab4138. PubMed PMID: 26816379.

3: Xuan W, Band LR, Kumpf RP, Van Damme D, Parizot B, De Rop G, Opdenacker D, Möller BK, Skorzinski N, Njo MF, De Rybel B, Audenaert D, Nowack MK, Vanneste S, Beeckman T. Cyclic programmed cell death stimulates hormone signaling and root development in Arabidopsis. Science. 2016 Jan 22;351(6271):384-7. doi: 10.1126/science.aad2776. PubMed PMID: 26798015.

4: Tabebordbar M, Zhu K, Cheng JK, Chew WL, Widrick JJ, Yan WX, Maesner C, Wu EY,
Xiao R, Ran FA, Cong L, Zhang F, Vandenberghe LH, Church GM, Wagers AJ. In vivo gene editing in dystrophic mouse muscle and muscle stem cells. Science. 2016 Jan 22;351(6271):407-11. doi: 10.1126/science.aad5177. Epub 2015 Dec 31. PubMed PMID:
26721686.

Minsoo

**1. Mol Cell. 2016 Jan 21;61(2):199-209. doi: 10.1016/j.molcel.2015.12.002. Epub 2015
Dec 24.**

TCA Cycle and Mitochondrial Membrane Potential Are Necessary for Diverse Biological Functions.

Martínez-Reyes I(1), Diebold LP(1), Kong H(1), Schieber M(1), Huang H(2), Hensley CT(3), Mehta MM(1), Wang T(4), Santos JH(4), Woychik R(4), Dufour E(5), Spelbrink
JN(6), Weinberg SE(1), Zhao Y(2), DeBerardinis RJ(3), Chandel NS(7).

Author information:

(1)Department of Medicine, Northwestern University Feinberg School of Medicine, Chicago, IL 60611, USA. (2)Ben May Department of Cancer Research, The University of Chicago, Chicago, IL 60637, USA. (3)Children Medical Center Research Institute, University of Texas Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, TX 75390, USA. (4)Division of Extramural Research and Training, National Institute of Environmental Health Sciences (NIEHS), National Institutes of Health (NIH), Department of Health and Human Services (DHHS), Research Triangle Park, NC 27709, USA. (5)BioMediTech and Tampere University Hospital, University of Tampere, Biokatu 8, 33520 Tampere, Finland. (6)Department of Pediatrics, Nijmegen Center for Mitochondrial Disorders, Radboud University Medical Centre, Geert Grooteplein 10, P.O. Box 9101, 6500 HB, Nijmegen, The Netherlands. (7)Department of Medicine, Northwestern University Feinberg School of Medicine, Chicago, IL 60611, USA. Electronic address: nav@northwestern.edu.

Mitochondrial metabolism is necessary for the maintenance of oxidative TCA cycle function and mitochondrial membrane potential. Previous attempts to decipher whether mitochondria are necessary for biological outcomes have been hampered by
genetic and pharmacologic methods that simultaneously disrupt multiple functions linked to mitochondrial metabolism. Here, we report that inducible depletion of mitochondrial DNA (ρ(o) cells) diminished respiration, oxidative TCA cycle function, and the mitochondrial membrane potential, resulting in diminished cell proliferation, hypoxic activation of HIF-1, and specific histone acetylation marks. Genetic reconstitution only of the oxidative TCA cycle function specifically in these inducible ρ(o) cells restored metabolites, resulting in re-establishment of histone acetylation. In contrast, genetic reconstitution of the mitochondrial membrane potential restored ROS, which were necessary for hypoxic activation of HIF-1 and cell proliferation. These results indicate that distinct mitochondrial functions associated with respiration are necessary for cell proliferation, epigenetics, and HIF-1 activation.

2. Biochim Biophys Acta. 2016 Jan 19. pii: S0005-2728(16)30001-9. doi: 10.1016/j.bbabio.2016.01.009. [Epub ahead of print]

Plant mitochondrial Complex I composition and assembly: A review.

Subrahmanian N(1), Remacle C(2), Hamel PP(3).

Author information:
(1)The Ohio State University, Department of Molecular Genetics, 500 Aronoff Laboratory, 318 W. 12th Avenue, Columbus, OH 43210, USA. (2)Institute of Botany, Department of Life Sciences, University of Liège, 4000 Liège, Belgium. (3)The Ohio State University, Department of Molecular Genetics, 500 Aronoff Laboratory, 318 W. 12th Avenue, Columbus, OH 43210, USA; The Ohio State University, Department of Biological Chemistry and Pharmacology, 500 Aronoff Laboratory, 318

W. 12th Avenue, Columbus, OH 43210, USA. Electronic address: hamel.16@osu.edu.

In the mitochondrial inner membrane, oxidative phosphorylation generates ATP via the operation of several multimeric enzymes. The proton-pumping Complex I (NADH:ubiquinone oxidoreductase) is the first and most complicated enzyme required in this process. Complex I is an L-shaped enzyme consisting of more than 40 subunits, one FMN molecule and eight Fe-S clusters. In recent years, genetic and proteomic analyses of Complex I mutants in various model systems, including plants, have provided valuable insights into the assembly of this multimeric enzyme. Assisted by a number of key players, referred to as "assembly factors", the assembly of Complex I takes place in a sequential and modular manner. Although a number of factors have been identified, their precise function in mediating Complex I assembly still remains to be elucidated. This review summarizes our current knowledge of plant Complex I composition and assembly derived from studies in plant model systems such as Arabidopsis thaliana and Chlamydomonas reinhardtii. Plant Complex I is highly conserved and comprises a significant number of subunits also present in mammalian and fungal Complexes I. Plant Complex I also contains additional subunits absent from the mammalian and fungal counterpart, whose function in enzyme activity and assembly is not clearly understood. While 14 assembly factors have been identified for human Complex I, only two proteins, namely GLDH and INDH, have been established as bona fide assembly factors for plant Complex I. This article is part of a Special Issue entitled Respiratory complex I, edited by Volkerzickermann and Ulrich Brandt.

Mary

ATPase-Modulated Stress Granules Contain a Diverse Proteome and Substructure
Saumya Jain4, Joshua R. Wheeler4, Robert W. Walters, Anurag Agrawal, Anthony Barsic, Roy Parker

Stress granules are mRNA-protein granules that form when translation initiation is limited, and they are related to pathological granules in various neurodegenerative diseases. Super-resolution microscopy reveals stable substructures, referred to as cores, within stress granules that can be purified. Proteomic analysis of stress granule cores reveals a dense network of protein-protein interactions and links between stress granules and human diseases and identifies ATP-dependent helicases and protein remodelers as conserved stress granule components. ATP is required for stress granule assembly and dynamics. Moreover, multiple ATP-driven machines affect stress granules differently, with the CCT complex inhibiting stress granule assembly, while the MCM and RVB complexes promote stress granule persistence. Our observations suggest that stress granules contain a stable core structure surrounded by a dynamic shell with assembly, disassembly, and transitions between the core and shell modulated by numerous protein and RNA remodeling complexes.

Keith

High-fidelity CRISPR–Cas9 nucleases with no detectable genome-wide off-target effects

Nature 529, 490–495 (28 January 2016)

Benjamin P. Kleinstiver, Vikram Pattanayak, Michelle S. Prew, Shengdar Q. Tsai, Nhu T. Nguyen, Zongli Zheng & J. Keith Joung

Molecular Pathology Unit, Center for Cancer Research, and Center for Computational and Integrative Biology, Massachusetts General Hospital, Charlestown, Massachusetts 02129, USA

Department of Pathology, Harvard Medical School, Boston, Massachusetts 02115, USA

Department of Biomedical Sciences, City University of Hong Kong, Hong Kong, China

CRISPR–Cas9 nucleases are widely used for genome editing but can induce unwanted off-target mutations. Existing strategies for reducing genome-wide off-target effects of the widely used *Streptococcus pyogenes* Cas9 (SpCas9) are imperfect, possessing only partial or unproven efficacies and other limitations that constrain their use. Here we describe SpCas9-HF1, a high-fidelity variant harbouring alterations designed to reduce non-specific DNA contacts. SpCas9-HF1 retains on-target activities comparable to wild-type SpCas9 with >85% of single-guide RNAs (sgRNAs) tested in human cells. Notably, with sgRNAs targeted to standard non-repetitive sequences, SpCas9-HF1 rendered all or nearly all off-target events undetectable by genome-wide break capture and targeted sequencing methods. Even for atypical, repetitive target sites, the vast majority of off-target mutations induced by wild-type SpCas9 were not detected with SpCas9-HF1. With its exceptional precision, SpCas9-HF1 provides an alternative to wild-type SpCas9 for research and therapeutic applications. More broadly, our results suggest a general strategy for optimizing genome-wide specificities of other CRISPR-RNA-guided nucleases.

Small Heat Shock Proteins are Novel Common Determinants of Alcohol and Nicotine Sensitivity in Caenorhabditis elegans.

Genetics. 2016 Jan 15. pii: genetics.115.185025.

Johnson JR1, Rajamanoharan D1, McCue HV1, Rankin K1, Barclay JW2.

University of Liverpool

Addiction to drugs is strongly determined by multiple genetic factors. Alcohol and nicotine produce distinct pharmacological effects within the nervous system

through discrete molecular targets; yet, data from family and twin analyses support the existence of common genetic factors for addiction in general. The mechanisms underlying addiction, however, are poorly described and common genetic factors for alcohol and nicotine remain unidentified. We investigated the role that the heat shock transcription factor, HSF-1, and its downstream effectors played as common genetic modulators of sensitivity to addictive substances. Using Caenorhabditis elegans, an exemplary model organism with substance dose-dependent responses similar to mammals, we demonstrate that HSF-1 altered sensitivity to both alcohol and nicotine. Using a combination of targeted RNAi screen of downstream factors and transgenic approaches we identified that these effects were contingent upon the constitutive neuronal expression of HSP-16.48, a small heat shock protein (HSP) homologue of human α-crystallin. Furthermore we demonstrated that the function of HSP-16.48 in drug sensitivity surprisingly was independent of chaperone activity during the heat shock stress response. Instead we identified a distinct domain within the N-terminal region of the HSP-16.48 protein that specified its function in comparison to related small HSPs. Our findings establish and characterise a novel genetic determinant underlying sensitivity to diverse addictive substances.
stress response.

REVIEW

Protein misfolding in the endoplasmic reticulum as a conduit to human disease

Nature 529, 326–335 (21 January 2016)

Miao Wang & Randal J. Kaufman

Degenerative Diseases Program, Sanford Burnham Prebys Medical Discovery Institute, 10901 North Torrey Pines Road, La Jolla, California 92037, USA.

In eukaryotic cells, the endoplasmic reticulum is essential for the folding and trafficking of proteins that enter the secretory pathway. Environmental insults or increased protein synthesis often lead to protein misfolding in the organelle, the accumulation of misfolded or unfolded proteins — known as endoplasmic reticulum stress — and the activation of the adaptive unfolded protein response to restore homeostasis. If protein misfolding is not resolved, cells die. Endoplasmic reticulum stress and activation of the unfolded protein response help to determine cell fate and function. Furthermore, endoplasmic reticulum stress contributes to the etiology of many human diseases.

Zebrafish as a Model for the Study of Chaperonopathies.

J Cell Physiol. 2016 Jan 27.

Bellipanni G1,2,3, Cappello F2,3,4, Scalia F4, de Macario EC5, Macario AJ3,5, Giordano A1,2.

1 Sbarro Institute for Cancer Research and Molecular Medicine.
2 Department of Biology, College of Science and Technology, Temple University, Philadelphia, Pennsylvania, 19122.
3 Euro-Mediterranean Institute of Science and Technology (IEMEST), Palermo, Italy.
4 Department of Experimental Biomedicine and Clinical Neuroscience, University of Palermo, Palermo, Italy.
5 Department of Microbiology and Immunology, School of Medicine, University of Maryland at Baltimore; and IMET, Baltimore, MD.

There is considerable information on the clinical manifestations and mode of inheritance for many genetic chaperonopathies but little is known on the molecular mechanisms underlying the cell and tissue abnormalities that characterize them. This scarcity of knowledge is mostly due to the lack of appropriate animal models that mimic closely the human molecular, cellular, and histological characteristics. In this article we introduce zebrafish as a suitable model to study molecular and cellular mechanisms pertaining to human chaperonopathies. Genetic chaperonopathies manifest themselves from very early in life so it is necessary to examine the impact of mutant chaperone genes during development, starting with fertilization and proceeding throughout the entire ontogenetic process. Zebrafish is amenable to such developmental analysis as well as studies during adulthood. In addition, the zebrafish genome contains a wide range of genes encoding proteins similar to those that form the chaperoning system of humans. This, together with the availability of techniques for genetic manipulations and for examination of all stages of development, makes zebrafish the organism of choice for the analysis of the molecular features and pathogenic mechanisms pertaining to human chaperonopathies

Elizabeth

The Arabidopsis NRG2 protein mediates nitrate signaling and interacts with and regulates key nitrate regulators
Na Xu, Rongchen Wang, Lufei Zhao, Chengfei Zhang, Zehui Li, Zhao Lei, Fei Liu, Peizhu Guan, Zhaohui Chu, Nigel Crawford, and Yong Wang
Plant Cell 2016 tpc.15.00567; Advance Publication January 7, 2016;
doi:10.1105/tpc.15.00567 **OPEN**
http://www.plantcell.org/content/early/2016/01/07/tpc.15.00567.abstract

Immobilized subpopulations of leaf epidermal mitochondria mediate PEN2-dependent pathogen entry control in Arabidopsis
Rene Fuchs, Michaela Kopischke, Christine Klapprodt, Gerd Hause, Andreas J. Meyer, Markus Schwarzländer, Mark David Fricker, and Volker Lipka
Plant Cell 2016 tpc.15.00887; Advance Publication December 31, 2015;
doi:10.1105/tpc.15.00887

http://www.plantcell.org/content/early/2016/01/04/tpc.15.00887.abstract

It's time for some "site"-seeing: novel tools to monitor the ubiquitin landscape in *Arabidopsis thaliana*
Alan Walton, Elisabeth Stes, Nicolas Cybulski, Michiel Van Bel, Sabrina Inigo, Astrid Nagels Durand, Evy Timmerman, Jefri Heyman, Laurens Pauwels, Lieven De Veylder, Alain Goossens, Ive De Smet, Frederik Coppens, Sofie Goormachtig, and Kris Gevaert
Plant Cell 2016 tpc.15.00878; Advance Publication January 7, 2016;
doi:10.1105/tpc.15.00878
http://www.plantcell.org/content/early/2016/01/07/tpc.15.00878.abstract

Cell

Nitric Oxide as a Switching Mechanism between Axon Degeneration and Regrowth during Developmental Remodeling ***Pages 170-182*** **Dana Rabinovich, Shiri P. Yaniv, Idan Alyagor, Oren Schuldiner**

Cryptochromes Interact Directly with PIFs to Control Plant Growth in Limiting Blue Light *Pages 233-245*
Ullas V. Pedmale, Shao-shan Carol Huang, Mark Zander, Benjamin J. Cole, Jonathan Hetzel, Karin Ljung, Pedro A.B. Reis, Priya Sridevi, Kazumasa Nito, Joseph R. Nery, Joseph R. Ecker, Joanne Chory

A Proteome-wide Fission Yeast Interactome Reveals Network Evolution Principles from Yeasts to Human Original Research Article · *Pages 310-323*
Tommy V. Vo, Jishnu Das, Michael J. Meyer, Nicolas A. Cordero, Nurten Akturk, Xiaomu Wei, Benjamin J. Fair, Andrew G. Degatano, Robert Fragoza, Lisa G. Liu, Akihisa Matsuyama, Michelle Trickey, Sachi Horibata, Andrew Grimson, Hiroyuki Yamano, Minoru Yoshida, Frederick P. Roth, Jeffrey A. Pleiss, Yu Xia, Haiyuan Yu
Munn J.
A Potential Role for Mitochondrial Produced Reactive Oxygen Species in Salicylic Acid-Mediated Plant Acquired Thermotolerance.
Plant Physiol. 2016 Jan 22;. [Epub ahead of print] PMID: 26802040 [PubMed - as supplied by publisher]

Wang Y, Dai Y, Tao X, Wang JZ, Cheng HY, Yang H, Ma XR.
Heat Shock Factor Genes of Tall Fescue and Perennial Ryegrass in Response to Temperature Stress by RNA-Seq Analysis.
Front Plant Sci. 2015;6:1226. PMID: 26793208 [PubMed]

Ghandi A, Adi M, Lilia F, Linoy A, Or R, Mikhail K, Mouhammad Z, Henryk C, Rena G.
Tomato yellow leaf curl virus infection mitigates the heat stress response of plants grown at high temperatures.
Sci Rep. 2016 Jan 21;6:19715. PMID: 26792235 [PubMed - in process]

Williamson JC, Edwards AV, Verano-Braga T, SchwÃ¤mmle V, Kjeldsen F, Jensen ON, Larsen MR.
High-performance hybrid Orbitrap mass spectrometers for quantitative proteome analysis: observations and implications.
Proteomics. 2016 Jan 21;. [Epub ahead of print] PMID: 26791339 [PubMed - as supplied by publisher]

Jahn M, Buchner J, Hugel T, Rief M.
Folding and assembly of the large molecular machine Hsp90 studied in single-molecule experiments.
Proc Natl Acad Sci U S A. 2016 Jan 19;. [Epub ahead of print] PMID: 26787848 [PubMed - as supplied by publisher]

Groitl B, Horowitz S, Makepeace KA, Petrotchenko EV, Borchers CH, Reichmann D, Bardwell JC, Jakob U.
Protein unfolding as a switch from self-recognition to high-affinity client binding.
Nat Commun. 2016 Jan 20;7:10357. PMID: 26787517 [PubMed - in process]

Fu X, Gao X, Ge L, Cui X, Su C, Yang W, Sun X, Zhang W, Yao Z, Yang X, Yang J.
Malonate induces the assembly of cytoplasmic stress granules.
FEBS Lett. 2016 Jan;590(1):22-33. PMID: 26787461 [PubMed - in process]

Tukaj S, WÄ™grzyn G.
Anti-Hsp90 therapy in autoimmune and inflammatory diseases: a review of preclinical studies.
Cell Stress Chaperones. 2016 Jan 20;. [Epub ahead of print] PMID: 26786410 [PubMed - as supplied by publisher]

Schymkowitz J, Rousseau F.
Protein aggregation: A rescue by chaperones.
Nat Chem Biol. 2016 Jan 19;12(2):58-9. PMID: 26784845 [PubMed - in process]

Sharma M, Laxmi A.
Jasmonates: Emerging Players in Controlling Temperature Stress Tolerance.
Front Plant Sci. 2015;6:1129. PMID: 26779205 [PubMed]

Perng MD, Huang YS, Quinlan RA.
Purification of Protein Chaperones and Their Functional Assays with Intermediate Filaments.
Methods Enzymol. 2016;569:155-75. PMID: 26778558 [PubMed - in process]

Pucciariello C, Perata P.
New insights into reactive oxygen species and nitric oxide signalling under low oxygen in plants.
Plant Cell Environ. 2016 Jan 22;. [Epub ahead of print] PMID: 26799776 [PubMed - as supplied by publisher]

Singh AP, Dixit G, Kumar A, Mishra S, Singh PK, Dwivedi S, Trivedi PK, Chakrabarty D, Mallick S, Pandey V, Dhankher OP, Tripathi RD.
Nitric Oxide Alleviated Arsenic Toxicity by Modulation of Antioxidants and Thiol Metabolism in Rice (Oryza sativa L.).
Front Plant Sci. 2015;6:1272. PMID: 26793232 [PubMed]

Li X, Pan Y, Chang B, Wang Y, Tang Z.
NO Promotes Seed Germination and Seedling Growth Under High Salt May Depend on EIN3 Protein in Arabidopsis.
Front Plant Sci. 2015;6:1203. PMID: 26779234 [PubMed]

Singh N, Bhatla SC.
Nitric oxide and iron modulate heme oxygenase activity as a long distance signaling response to salt stress in sunflower seedling cotyledons.
Nitric Oxide. 2016 Jan 14;. [Epub ahead of print] PMID: 26778276 [PubMed - as supplied by publisher]

Moore CE, Wang X, Xie J, Pickford J, Barron J, da Mota SR, Versele M, Proud CG.
Elongation factor 2 kinase promotes cell survival by inhibiting protein synthesis without inducing autophagy.
Cell Signal. 2016 Jan 18;. [Epub ahead of print] PMID: 26795954 [PubMed - as supplied by publisher]

Yutthanasirikul R, Nagano T, Jimbo H, Hihara Y, Kanamori T, Ueda T, Haruyama T, Konno H, Yoshida K, Hisabori T, Nishiyama Y.
Oxidation of a Cysteine Residue in Elongation Factor EF-Tu Reversibly Inhibits Translation in the Cyanobacterium Synechocystis sp. PCC 6803. J Biol Chem. 2016 Jan 19;. [Epub ahead of print]
PMID: 26786107 [PubMed - as supplied by publisher]

Bouloc P, Repoila F.
Fresh layers of RNA-mediated regulation in Gram-positive bacteria.
Curr Opin Microbiol. 2016 Jan 12;30:30-35. [Epub ahead of print] PMID: 26773797 [PubMed - as supplied by publisher]

Johnson JR, Rajamanoharan D, McCue HV, Rankin K, Barclay JW.
Small Heat Shock Proteins are Novel Common Determinants of Alcohol and Nicotine Sensitivity in Caenorhabditis elegans.
Genetics. 2016 Jan 15;. [Epub ahead of print] PMID: 26773049 [PubMed - as supplied by publisher]

EscandÃ³n M, CaÃ±al MJ, Pascual J, Pinto G, Correia B, Amaral J, MeijÃ³n M.
Integrated physiological and hormonal profile of heat-induced thermotolerance in Pinus radiata.

Tree Physiol. 2016 Jan 12;. [Epub ahead of print] PMID: 26764270 [PubMed - as supplied by publisher]

Niwa T, Fujiwara K, Taguchi H.
Identification of novel in vivo obligate GroEL/ES substrates based on data from a cell-free proteomics approach.
FEBS Lett. 2015 Dec 21;. [Epub ahead of print] PMID: 26762164 [PubMed - as supplied by publisher]

Fancy NN, Bahlmann AK, Loake GJ.
Nitric oxide function in plant abiotic stress.
Plant Cell Environ. 2016 Jan 11;. [Epub ahead of print] PMID: 26754426 [PubMed - as supplied by publisher]

Wu H, Wang C, Gong W, Wang J, Xuan J, Perrett S, Feng Y.
The C-terminal region of human eukaryotic elongation factor 1BÎ´.
J Biomol NMR. 2016 Jan 13;. [Epub ahead of print] PMID: 26762120 [PubMed - as supplied by publisher]

Torrente MP, Chuang E, Noll MM, Jackrel ME, Go MS, Shorter J.
Mechanistic Insights Into Hsp104 Potentiation.
J Biol Chem. 2016 Jan 8;. [Epub ahead of print] PMID: 26747608 [PubMed - as supplied by publisher]

Waqas M, Shahzad R, Khan AL, Asaf S, Kim YH, Kang SM, Bilal S, Hamayun M, Lee IJ.
Salvaging effect of triacontanol on plant growth, thermotolerance, macro-nutrient content, amino acid concentration and modulation of defense hormonal levels under heat stress.
Plant Physiol Biochem. 2015 Dec 24;99:118-125. [Epub ahead of print] PMID: 26744997 [PubMed - as supplied by publisher]

Choi SH, Nam JK, Kim BY, Jang J, Jin YB, Lee HJ, Park S, Ji YH, Cho J, Lee YJ.
HSPB1 inhibits the endothelial-to-mesenchymal transition to suppress pulmonary fibrosis and lung tumorigenesis.
Cancer Res. 2016 Jan 7;. [Epub ahead of print] PMID: 26744531 [PubMed - as supplied by publisher]

Arimoto-Matsuzaki K, Saito H, Takekawa M.
TIA1 oxidation inhibits stress granule assembly and sensitizes cells to stress-induced apoptosis.
Nat Commun. 2016 Jan 7;7:10252. PMID: 26738979 [PubMed - in process]

Takatsuka H, Umeda M.
Epigenetic Control of Cell Division and Cell Differentiation in the Root Apex.
Front Plant Sci. 2015;6:1178. PMID: 26734056 [PubMed]

Jung YJ, Melencion SM, Lee ES, Park JH, Alinapon CV, Oh HT, Yun DJ, Chi YH, Lee SY.
Universal Stress Protein Exhibits a Redox-Dependent Chaperone Function in Arabidopsis and Enhances Plant Tolerance to Heat Shock and Oxidative Stress.
Front Plant Sci. 2015;6:1141. PMID: 26734042 [PubMed]

Radin I, Mansilla N, RÃ¶del G, Steinebrunner I.
The Arabidopsis COX11 Homolog is Essential for Cytochrome c Oxidase Activity.
Front Plant Sci. 2015;6:1091. PMID: 26734017 [PubMed]

Andre FR, Dos Santos PF, Rando DG.
Theoretical studies of the role of C-terminal cysteines in the process of S-nitrosylation of human Src kinases.
J Mol Model. 2016 Jan;22(1):23. PMID: 26733486 [PubMed - in process]