Lit Review – 2/14/17
Patrick

1. **Astrategy for dissecting the architectures of native macromolecular assemblies**

Yi Shi[1], Riccardo Pellarin[2–5], Peter C Fridy[6], Javier Fernandez-Martinez[6], Mary K Thompson[6], Yinyin Li[1], Qing Jun Wang[7], Andrej Sali[2–4], Michael P Rout[6] & Brian T Chait[1]

It remains particularly problematic to define the structures of native macromolecular assemblies, which are often of low abundance. Here we present a strategy for isolating complexes at endogenous levels from GFP-tagged transgenic cell lines. Using cross-linking mass spectrometry, we extracted distance restraints that allowed us to model the complexes' molecular architectures.

2. **Nanobodies: Natural Single-Domain Antibodies**

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Abstract
Sera of camelids contain both conventional heterotetrameric antibodies and unique functional heavy (H)-chain antibodies (HCAbs). The H chain of these homodimeric antibodies consists of one antigen-binding domain, the VHH, and two constant domains. HCAbs fail to incorporate light (L) chains owing to the deletion of the first constant domain and a reshaped surface at the VHHside, which normally associates with L chains in conventional antibodies. The genetic elements composing HCAbshave been identified, but the in vivo generation of these antibodies from their dedicated genes into antigen-specific and affinity-matured bona fide antibodies remains largely underinvestigated. However, the facile identification of antigen-specific VHHs and their beneficial biochemical and economic properties (size, affinity, specificity, stability, production cost) supported by multiple crystal structures have encouraged antibody engineering of these single-domain antibodies for use as a research tool and in biotechnology and medicine

Ian

J Biol Chem. 2013 Sep 13;288(37):26497-504. doi: 10.1074/jbc.R113.461368. Epub 2013 Jul 16.

Causes and consequences of cysteine S-glutathionylation.

Grek CL[1], Zhang J, Manevich Y, Townsend DM, Tew KD.

Jesse

The Plant Cell. Jan 2017.

Arabidopsis Seed Mitochondria Are Bioenergetically Active Immediately upon Imbibition and Specialize via Biogenesis in Preparation for Autotrophic Growth

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Seed germination is a vital developmental transition for production of progeny by sexual reproduction in spermatophytes. Quiescent cells in nondormant dry embryos are reawakened first by imbibition and then by perception of germination triggers. Reanimated tissues enter into a germination program requiring energy for expansion growth. However, germination requires that embryonic tissues develop to support the more energy-demanding processes of cell division and organogenesis of the new seedling. Reactivation of mitochondria to supply the required energy is thus a key process underpinning germination and seedling survival. Using live imaging, we investigated reactivation of mitochondrial bioenergetics and dynamics using Arabidopsis thaliana as a model. Bioenergetic reactivation, visualized by presence of a membrane potential, is immediate upon rehydration. However, reactivation of mitochondrial dynamics only occurs after transfer to germination conditions. Reactivation of mitochondrial bioenergetics is followed by dramatic reorganization of the chondriome (all mitochondrial in a cell, collectively) involving massive fusion and membrane biogenesis to form a perinuclear tubuloreticular structure enabling mixing of previously discrete mitochondrial DNA nucleoids. The end of germination coincides with fragmentation of the chondriome, doubling of mitochondrial number, and heterogeneous redistribution of nucleoids among the mitochondria, generating a population of mitochondria tailored to seedling growth.

Minsoo

1. Cell. 2016 Sep 8;166(6):1553-1563.e10. doi: 10.1016/j.cell.2016.08.042.

Neuroendocrine Coordination of Mitochondrial Stress Signaling and Proteostasis.

Berendzen KM(1), Durieux J(1), Shao LW(2), Tian Y(1), Kim HE(1), Wolff S(1), Liu Y(2), Dillin A(3).

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During neurodegenerative disease, the toxic accumulation of aggregates and misfolded proteins is often accompanied with widespread changes in peripheral metabolism, even in cells in which the aggregating protein is not present. The mechanism by which the central nervous system elicits a distal reaction to proteotoxic stress remains unknown. We hypothesized that the endocrine communication of neuronal stress plays a causative role in the changes in mitochondrial homeostasis associated with proteotoxic disease states. We find that an aggregation-prone protein expressed in the neurons of C. elegans binds to

mitochondria, eliciting a global induction of a mitochondrial-specific unfolded protein response (UPR(mt)), affecting whole-animal physiology. Importantly, dense core vesicle release and secretion of the neurotransmitter serotonin is required for the signal's propagation. Collectively, these data suggest the commandeering of a nutrient sensing network to allow for cell-to-cell communication between mitochondria in response to protein folding stress in the nervous system.

2. Cell. 2016 Sep 8;166(6):1539-1552.e16. doi: 10.1016/j.cell.2016.08.027.

Lipid Biosynthesis Coordinates a Mitochondrial-to-Cytosolic Stress Response.

Kim HE(1), Grant AR(2), Simic MS(1), Kohnz RA(3), Nomura DK(4), Durieux J(1), Riera CE(1), Sanchez M(1), Kapernick E(1), Wolff S(1), Dillin A(5).

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Defects in mitochondrial metabolism have been increasingly linked with age-onset protein-misfolding diseases such as Alzheimer's, Parkinson's, and Huntington's. In response to protein-folding stress, compartment-specific unfolded protein responses (UPRs) within the ER, mitochondria, and cytosol work in parallel to ensure cellular protein homeostasis. While perturbation of individual compartments can make other compartments more susceptible to protein stress, the cellular conditions that trigger cross-communication between the individual UPRs remain poorly understood. We have uncovered a conserved, robust mechanism linking mitochondrial protein homeostasis and the cytosolic folding environment through changes in lipid homeostasis. Metabolic restructuring caused by mitochondrial stress or small-molecule activators trigger changes in gene expression coordinated uniquely by both the mitochondrial and cytosolic UPRs, protecting the cell from disease-associated proteins. Our data suggest an intricate and unique system of communication between UPRs in response to metabolic changes that could unveil new targets for diseases of protein misfolding.

Keith

Targeting the molecular chaperone SlyD to inhibit bacterial growth with a small molecule.

Sci Rep. 2017 Feb 8

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Molecular chaperones are essential molecules for cell growth, whereby they maintain protein homeostasis. Because of their central cellular function, bacterial chaperones might be potential candidates for drug targets. Antimicrobial resistance is currently one of the greatest threats to human health, with gram-negative bacteria being of major concern. We found that a Cu2+ complex readily crosses the bacterial cell wall and inhibits SlyD, which is a molecular chaperone, cis/trans peptidyl prolyl isomerise (PPIase) and involved in various other metabolic pathways. The Cu2+ complex binds to the active sites of SlyD, which suppresses its PPIase and chaperone activities. Significant cell growth retardation could be observed for pathogenic bacteria (e.g., Staphylococcus aureus and Pseudomonas aeruginosa). We anticipate that rational development of drugs targeting molecular chaperones might help in future control of pathogenic bacterial growth, in an era of rapidly increasing antibiotic resistance.

Single molecule force spectroscopy reveals the effect of BiP chaperone on protein folding.

Protein Sci. 2017 Feb 8.

Ramírez MP1,2, Rivera M1, Quiroga-Roger D1, Bustamante A1, Vega M1, Baez M1, Puchner EM2, Wilson CA1.

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2Laboratory of Cellular and Molecular Biophysics, School of Physics & Astronomy, University of Minnesota, Twin Cities, MN, United States.

BiP (Immunoglobulin Binding Protein) is a member of the Hsp70 chaperones that participates in protein folding in the endoplasmic reticulum. The function of BiP relies on cycles of ATP hydrolysis driving the binding and release of its substrate proteins. It still remains unknown how BiP affects the protein folding pathway and there has been no direct demonstration showing which folding state of the substrate protein is bound by BiP, as previous work has used only peptides. Here, we employ optical tweezers for single molecule force spectroscopy experiments to investigate how BiP affects the folding mechanism of a complete protein and how this effect depends on nucleotides. Using the protein MJ0366 as the substrate for BiP, we performed pulling and relaxing

cycles at constant velocity to unfold and refold the substrate. In the absence of BiP, MJ0366 unfolded and refolded in every cycle. However, when BiP was added, the frequency of folding events of MJ0366 significantly decreased, and the loss of folding always occurred after a successful unfolding event. This process was dependent on ATP and ADP, since when either ATP was decreased or ADP was added, the duration of periods without folding events increased. Our results show that the affinity of BiP for the substrate protein increased in these conditions, which correlates with previous studies in bulk. Therefore, we conclude that BiP binds to the unfolded state of MJ0366 and prevents its refolding, and that this effect is dependent on both the type and concentration of nucleotides.

Elizabeth

February 13, 2017

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