

Lit Lunch 3/13/15

Damian:

Gopi K. Kolluru, Shuai Yuan, Xinggui Shen, Christopher G. Kevil, Chapter Fifteen - **H₂S Regulation of Nitric Oxide Metabolism**, In: Enrique Cadenas and Lester Packer, Editor(s), *Methods in Enzymology*, Academic Press, 2015, Volume 554, Pages 271-297, ISSN 0076-6879, ISBN 9780128015124, <http://dx.doi.org/10.1016/bs.mie.2014.11.040>.

Nitric oxide (NO) and hydrogen sulfide (H₂S) are two major gaseous signaling molecules that regulate diverse physiological functions. Recent publications indicate the regulatory role of H₂S on NO metabolism. In this chapter, we discuss the latest findings on H₂S–NO interactions through formation of novel chemical derivatives and experimental approaches to study these adducts. This chapter also addresses potential H₂S interference on various NO detection techniques, along with precautions for analyzing biological samples from various sources. This information will facilitate critical evaluation and clearer insight into H₂S regulation of NO signaling and its influence on various physiological functions.

Fionn:

Plant cell

Tudor Staphylococcal Nuclease Links Formation of Stress Granules and Processing Bodies with mRNA Catabolism in Arabidopsis

Emilio Gutierrez-Beltran,^a Panagiotis N. Moschou,^{a,1} Andrei P. Smertenko,^{b,c} and Peter V. Bozhkova

Tudor Staphylococcal Nuclease (TSN or Tudor-SN; also known as SND1) is an evolutionarily conserved protein involved in the transcriptional and posttranscriptional regulation of gene expression in animals. Although TSN was found to be indispensable for normal plant development and stress tolerance, the molecular mechanisms underlying these functions remain elusive. Here, we show that *Arabidopsis thaliana* TSN is essential for the integrity and function of cytoplasmic messenger ribonucleoprotein (mRNP) complexes called stress granules (SGs) and processing bodies (PBs), sites of posttranscriptional gene regulation during stress. TSN associates with SGs following their microtubule-dependent assembly and plays a scaffolding role in both SGs and PBs. The enzymatically active tandem repeat of four SN domains is crucial for targeting TSN to the cytoplasmic mRNA complexes and is sufficient for the cytoprotective function of TSN during stress. Furthermore, our work connects the cytoprotective function of TSN with its positive role in stress-induced mRNA decapping. While stress led to a pronounced increase in the accumulation of uncapped mRNAs in wild-type plants, this increase was abrogated in TSN knockout plants. Taken together, our results establish TSN as a key enzymatic component of the catabolic machinery responsible for the processing of mRNAs in the cytoplasmic mRNP complexes during stress.

Keith:

Cyclin-dependent Kinase 5 Phosphorylation of Familial Prion Protein Mutants Exacerbates Conversion into Amyloid Structure

February 27, 2015 The Journal of Biological Chemistry, 290, 5759-5771.

Raphaël Rouget[‡], Gyanesh Sharma^{‡ §}, and Andréa C. LeBlanc^{‡ §1}

[‡]Lady Davis Institute for Medical Research, Sir Mortimer B. Davis Jewish General Hospital, Department of Neurology and Neurosurgery, McGill University, Montréal, Québec H3T 1E2, Canada and [§]Department of Neurology and Neurosurgery, McGill University, 3775 University Street, Montréal, Québec H3A 2B4, Canada

Familial prion protein (PrP) mutants undergo conversion from soluble and protease-sensitive to insoluble and partially protease-resistant proteins. Cyclin-dependent kinase 5 (Cdk5) phosphorylation of wild type PrP (pPrP) at serine 43 induces a conversion of PrP into aggregates and fibrils. Here, we investigated whether familial PrP mutants are predisposed to Cdk5 phosphorylation and whether phosphorylation of familial PrP mutants increases conversion. PrP mutants representing three major familial PrP diseases and different PrP structural domains were studied. We developed a novel in vitro kinase reaction coupled with Thioflavin T binding to amyloid structure assay to monitor phosphorylation-dependent amyloid conversion. Although non-phosphorylated full-length wild type or PrP mutants did not convert into amyloid, Cdk5 phosphorylation rapidly converted these into Thioflavin T-positive structures following first order kinetics. Dephosphorylation partially reversed conversion. Phosphorylation-dependent conversion of PrP from α -helical structures into β -sheet structures was confirmed by circular dichroism. Relative to wild type pPrP, most PrP mutants showed increased rate constants of conversion. In contrast, non-phosphorylated truncated PrP Y145X (where X represents a stop codon) and Q160X mutants converted spontaneously into Thioflavin T-positive fibrils after a lag phase of over 20 h, indicating nucleation-dependent polymerization. Phosphorylation reduced the lag phase by over 50% and thus accelerated the formation of the nucleating event. Consistently, phosphorylated Y145X and phosphorylated Q160X exacerbated conversion in a homologous seeding reaction, whereas WT pPrP could not seed WT PrP. These results demonstrate an influence of both the N terminus and the C terminus of PrP on conversion. We conclude that post-translational modifications of the flexible N terminus of PrP can cause or exacerbate PrP mutant conversion.

Stephanie:

Plant Molecular Biology

TaRAR1 and TaSGT1 associate with TaHsp90 to function in bread wheat (*Triticum aestivum* L.) seedling growth and stripe rust resistance.

Plant Mol. Biol.

Plant Mol Biol 2015 Feb 20. Epub 2015 Feb 20.

Guan-Feng Wang, Renchun Fan, Xianping Wang, Daowen Wang, Xiangqi Zhang

RAR1 and SGT1 are important co-chaperones of Hsp90. We previously showed that TaHsp90.1 is required for wheat seedling growth, and that TaHsp90.2 and TaHsp90.3 are essential for resistance (R) gene mediated resistance to stripe rust fungus. Here, we report the characterization of TaRAR1 and TaSGT1 genes in bread wheat. TaRAR1 and TaSGT1 each had three homoeologs, which were located on wheat groups 2 and 3 chromosomes, respectively. Strong inhibition of seedling growth was observed after silencing TaSGT1 but not TaRAR1. In contrast, decreasing the expression of TaRAR1 or TaSGT1 could all compromise R gene mediated resistance to stripe rust fungus infection. Protein-protein interactions were found among TaRAR1, TaSGT1 and TaHsp90. The N-terminus of TaHsp90, the CHORD-I and CHORD-II domains of TaRAR1 and the CS domain of TaSGT1 may be instrumental for the interactions among the three proteins. Based on this work and our previous study on TaHsp90, we speculate that the TaSGT1-TaHsp90.1 interaction is important for maintaining bread wheat seedling growth. The TaRAR1-TaSGT1-TaHsp90.2 and TaRAR1-TaSGT1-TaHsp90.3 interactions are involved in controlling the resistance to stripe rust disease. The new information obtained here should aid further functional investigations of TaRAR1-TaSGT1-TaHsp90 complexes in regulating bread wheat growth and disease resistance.

Affiliation

The State Key Laboratory of Plant Cell and Chromosome Engineering, Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, Beijing, 100101, China,
gfwang123@gmail.com.

Nature Plants:

- 1) **Gates Foundation backs high-risk science for big wins!** [Natasha Gilbert](#)
- 2) **Protein turnover in plant biology** [Clark J. Nelson](#) & [A. Harvey Millar](#)

The protein content of plant cells is constantly being updated. This process is driven by the opposing actions of protein degradation, which defines the half-life of each polypeptide, and protein synthesis. Our understanding of the processes that regulate protein synthesis and degradation in plants has advanced significantly over the past decade. Post-transcriptional modifications that influence features of the mRNA populations, such as poly(A) tail length and secondary structure, contribute to the regulation of protein

synthesis. Post-translational modifications such as phosphorylation, ubiquitination and non-enzymatic processes such as nitrosylation and carbonylation, govern the rate of degradation. Regulators such as the plant TOR kinase, and effectors such as the E3 ligases, allow plants to balance protein synthesis and degradation under developmental and environmental change. Establishing an integrated understanding of the processes that underpin changes in protein abundance under various physiological and developmental scenarios will accelerate our ability to model and rationally engineer plants.

Nathen:

Genomic analysis of parallel-evolved cyanobacterium *Synechocystis* sp. PCC 6803 under acid stress.

[Uchiyama J](#), [Kanesaki Y](#), [Iwata N](#), [Asakura R](#), [Funamizu K](#), [Tasaki R](#), [Agatsuma M](#), [Tahara H](#), [Matsuhashi A](#), [Yoshikawa H](#), [Ogawa S](#), [Ohta H](#).

[Author information](#)

Abstract

Experimental evolution is a powerful tool for clarifying phenotypic and genotypic changes responsible for adaptive evolution. In this study, we isolated acid-adapted *Synechocystis* sp. PCC 6803 (*Synechocystis* 6803) strains to identify genes involved in acid tolerance. *Synechocystis* 6803 is rarely found in habitats with pH < 5.75. The parent (P) strain was cultured in BG-11 at pH 6.0. We gradually lowered the pH of the medium from pH 6.0 to pH 5.5 over 3 months. Our adapted cells could grow in acid stress conditions at pH 5.5, whereas the parent cells could not. We performed whole-genome sequencing and compared the acid-adapted and P strains, thereby identifying 11 SNPs in the acid-adapted strains, including in Fo F1-ATPase. To determine whether the SNP genes responded to acid stress, we examined gene expression in the adapted strains using quantitative reverse-transcription polymerase chain reaction. *sll0914*, *sll1496*, *sll0528*, and *sll1144* expressions increased under acid stress in the P strain, whereas *sll0162*, *sll0163*, *slr0623*, and *slr0529* expressions decreased. There were no differences in the SNP genes expression levels between the P strain and two adapted strains, except for *sll0528*. These results suggest that SNPs in certain genes are involved in acid stress tolerance in *Synechocystis* 6803.
