

Indu

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Fionn

Nature biotech

Overexpression of receptor-like kinase ERECTA improves thermotolerance in rice and tomato

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Abstract

The detrimental effects of global warming on crop productivity threaten to reduce the world's food supply^{1, 2, 3}. Although plant responses to changes in temperature have been studied⁴, genetic modification of crops to improve thermotolerance has had little success to date. Here we demonstrate that overexpression of the *Arabidopsis thaliana* receptor-like kinase ERECTA (ER) in *Arabidopsis*, rice and tomato confers thermotolerance independent of water loss and that *Arabidopsis* er mutants are hypersensitive to heat. A loss-of-function mutation of a rice ER homolog and reduced expression of a tomato ER allele decreased thermotolerance of both species. Transgenic tomato and rice lines overexpressing *Arabidopsis* ER showed improved heat tolerance in the greenhouse and in field tests at multiple locations in China during several seasons. Moreover, ER-overexpressing transgenic *Arabidopsis*, tomato and rice plants had increased biomass. Our findings could contribute to engineering or breeding thermotolerant crops with no growth penalty.

Nature

Autopsies reveal signs of Alzheimer's in growth-hormone patients

Alison Abbott
Plant cell

Characteristics of Plant Essential Genes Allow for within- and between-Species Prediction of Lethal Mutant Phenotypes[OPEN]
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Abstract

Essential genes represent critical cellular components whose disruption results in lethality. Characteristics shared among essential genes have been uncovered in fungal and metazoan model systems. However, features associated with plant essential genes are largely unknown and the full set of essential genes remains to be discovered in any plant species. Here, we show that essential genes in *Arabidopsis thaliana* have distinct features useful for constructing within- and cross-species prediction models. Essential genes in *A. thaliana* are often single copy or derived from older duplications, highly and broadly expressed, slow evolving, and highly connected within molecular networks compared with genes with nonlethal mutant phenotypes. These gene features allowed the application of machine learning methods that predicted known lethal genes as well as an additional 1970 likely essential genes without documented phenotypes. Prediction models from *A. thaliana* could also be applied to predict *Oryza sativa* and *Saccharomyces cerevisiae* essential genes. Importantly, successful predictions drew upon many features, while any single feature was not sufficient. Our findings show that essential genes can be distinguished from genes with nonlethal phenotypes using features that are similar across kingdoms and indicate the possibility for translational application of our approach to species without extensive functional genomic and phenomic resources.

Minsoo

1. Plant J. 2015 Aug 25. doi: 10.1111/tpj.12993. [Epub ahead of print]

Empty pericarp7 encodes a mitochondrial E-subgroup pentatricopeptide repeat protein that is required for ccmFN editing, mitochondrial function and seed development in maize.

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RNA editing, converting cytidines (C) to uridines (U) at specific sites in the transcripts of mitochondria and plastids, plays a critical role in organelle gene expression in land plants. Recently pentatricopeptide repeat (PPR) proteins were identified as site-specific recognition factors for RNA editing. In this study, we characterized an empty pericarp7 mutant (*emp7*) in maize (*Zea mays*), which confers an embryo-lethal phenotype. In *emp7* mutants, mitochondrial functions are seriously perturbed, resulting in strikingly reduced respiration rate. *Emp7* encodes an E-subgroup PPR protein that is localized exclusively in the mitochondrion. Null mutation of *Emp7* abolishes the C-to-U editing of *ccmFN* transcript solely at the position 1553. *CcmFN* is coding for a subunit of heme lyase complex in the cytochrome *c* maturation pathway. The resulting Phe to Ser substitution in *CcmFN* leads to the loss of *CcmFN* protein and a strikingly reduced the *c*-type cytochrome level. Consequently, the mitochondrial cytochrome-linked respiratory chain is impaired, due to disassembly of complex III in the *emp7* mutant. These results indicate that the PPR-E subgroup protein *EMP7* is required for C-to-U editing of *ccmFN* -1553 at a position essential for cytochrome *c* maturation and mitochondrial oxidative phosphorylation, hence essential to embryo and endosperm development in maize.

2. Proc Natl Acad Sci U S A. 2015 Aug 18;112(33):10154-61. doi: 10.1073/pnas.1421372112. Epub 2015 Jul 20.

Mitochondrial genomes are retained by selective constraints on protein targeting.

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Mitochondria are energy-producing organelles in eukaryotic cells considered to be

of bacterial origin. The mitochondrial genome has evolved under selection for minimization of gene content, yet it is not known why not all mitochondrial genes have been transferred to the nuclear genome. Here, we predict that hydrophobic membrane proteins encoded by the mitochondrial genomes would be recognized by the signal recognition particle and targeted to the endoplasmic reticulum if they were nuclear-encoded and translated in the cytoplasm. Expression of the mitochondrially encoded proteins Cytochrome oxidase subunit 1, Apocytochrome b, and ATP synthase subunit 6 in the cytoplasm of HeLa cells confirms export to the endoplasmic reticulum. To examine the extent to which the mitochondrial proteome is driven by selective constraints within the eukaryotic cell, we investigated the occurrence of mitochondrial protein domains in bacteria and eukaryotes. The accessory protein domains of the oxidative phosphorylation system are unique to mitochondria, indicating the evolution of new protein folds. Most of the identified domains in the accessory proteins of the ribosome are also found in eukaryotic proteins of other functions and locations. Overall, one-third of the protein domains identified in mitochondrial proteins are only rarely found in bacteria. We conclude that the mitochondrial genome has been maintained to ensure the correct localization of highly hydrophobic membrane proteins. Taken together, the results suggest that selective constraints on the eukaryotic cell have played a major role in modulating the evolution of the mitochondrial genome and proteome.

Mary

Aguado A, Fernandez-Higuero JA, Moro F, Muga A.

Chaperone-assisted protein aggregate reactivation: different solutions for the same problem.

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The oligomeric AAA+ chaperones Hsp104 in yeast and ClpB in bacteria are responsible for the reactivation of aggregated proteins, an activity essential for cell survival during severe stress. The protein disaggregase activity of these members of the Hsp100 family is linked to the activity of chaperones from the Hsp70 and Hsp40 families. The precise mechanism by which these proteins untangle protein aggregates remains unclear. Strikingly, Hsp100 proteins are not present in metazoans. This does not mean that animal cells do not have a disaggregase activity, but that this activity is performed by the Hsp70 system and a representative of the Hsp110 family instead of a Hsp100 protein. This review describes the actual view of Hsp100-mediated aggregate reactivation, including the ATP-induced conformational changes associated with their disaggregase activity, the dynamics of the oligomeric assembly that is regulated by its ATPase cycle and the DnaK system, and the tight allosteric coupling between the ATPase domains within the hexameric ring complexes. The lack of homologs of these disaggregases

in metazoans has suggested that they might be used as potential targets to develop antimicrobials. The current knowledge of the human disaggregase machinery and the role of Hsp110 are also discussed.

Damian

TY - JOUR

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TI - Glutathione plays an essential role in nitric oxide-mediated iron deficiency signaling and iron deficiency tolerance in Arabidopsis

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KW - Fe deficiency signaling

KW - Glutathione

KW - nitric oxide

KW - S-nitrosoglutathione

KW - Arabidopsis thaliana

PY - 2015

AB - Iron (Fe) deficiency is a common agricultural problem that affects both the productivity and nutritional quality of plants. Thus, identifying the key factors involved in the tolerance of Fe deficiency is important. In the present study, the *zir1* mutant, which is glutathione deficient, was found to be more sensitive to Fe deficiency than the wild type, and grew poorly in alkaline soil. Other glutathione deficient mutants also showed various degrees of sensitivity to Fe limited conditions. Interestingly, we found that the glutathione level was increased under Fe deficiency in the wild type. By contrast, blocking glutathione biosynthesis led to increased physiological sensitivity to Fe deficiency. On the other hand, overexpressing glutathione enhanced the tolerance to Fe deficiency. Under Fe limited conditions, glutathione deficient mutants, *zir1*, *pad2* and *cad2* accumulated lower levels of Fe than the wild type. The key genes involved in Fe uptake including *IRT1*, *FRO2* and *FIT* are expressed at low levels in *zir1*. However, a split root experiment suggested that the systemic signals that govern the expression of Fe-uptake-related genes are still active in *zir1*. Furthermore, we found that *zir1* had lower accumulation of nitric oxide (NO) and NO reservoir S-nitrosoglutathione (GSNO). Although NO is a signaling molecule involved in the induction of Fe-uptake-related genes during Fe deficiency, the NO mediated induction of Fe uptake genes is

dependent on glutathione supply in the *zir1* mutant. These results provide direct evidence that glutathione plays an essential role in Fe deficiency tolerance and NO-mediated Fe deficiency signaling in Arabidopsis.

Keith

A Mechanism of Subunit Recruitment in Human Small Heat Shock Protein Oligomers

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Small heat shock proteins (sHSPs) make up a class of molecular chaperones broadly observed across organisms. Many sHSPs form large oligomers that undergo dynamic subunit exchange that is thought to play a role in chaperone function. Though remarkably heterogeneous, sHSP oligomers share three types of intermolecular interactions that involve all three defined regions of a sHSP: the N-terminal region (NTR), the conserved α -crystallin domain (ACD), and a C-terminal region (CTR). Here we define the structural interactions involved in incorporation of a subunit into a sHSP oligomer. We demonstrate that a minimal ACD dimer of the human sHSP, HSPB5, interacts with an HSPB5 oligomer through two types of interactions: (1) interactions with CTRs in the oligomer and (2) via exchange into and out of the dimer interface composed of two ACDs. Unexpectedly, although dimers are thought to be the fundamental building block for sHSP oligomers, our results clearly indicate that subunit exchange into and out of oligomers occurs via monomers. Using structure-based mutants, we show that incorporation of a subunit into an oligomer is predicated on recruitment of the subunit via its interaction with CTRs on an oligomer. Both the rate and extent of subunit incorporation depend on the accessibility of CTRs within an HSPB5 oligomer. We show that this mechanism also applies to formation of heterooligomeric sHSP species composed of HSPB5 and HSPB6 and is likely general among sHSPs. Finally, our observations highlight the importance of NTRs in the thermodynamic stability of sHSP oligomers.