P.2.4-004
The nuclear-encoded plant pentatricopeptide protein MEF31 is involved in RNA editing at two near sites in the mitocondrial transcript encoding subunit C of the twin-arginine translocation pathway

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RNA editing is one of the major post-transcriptional RNA maturation events in plant mitochondria and chloroplasts. In flowering plants, about 40 chloroplast and more than 400 mitochondrial Cs are deaminated to Us, most changes occurring in mRNA coding regions and changing their coding potential. A region between −20 or −25 and +6 relative to the C to be edited is generally sufficient and necessary for editing, and the region upstream of the edited C in these *cis* elements is recognized by specificity factors belonging to the largest protein family in angiosperms: the pentatricopeptide repeat (PPR) proteins. Here we describe the novel mitochondrial editing factor 31, an E-PPR protein involved in editing at two sites in the same transcript, which encodes subunit C of the twin-arginine translocation (tat) pathway. MEF31 is required for C581 editing, which changes a Pro CCA codon to a Leu CUA codon, and application of a recently proposed amino acid code for modular RNA recognition by PPR proteins shows that MEF31 likely directly targets the C581 *cis* sequence. Our data also demonstrates that MEF31, although not absolutely required, influences C586 editing. MEF31 probably acts by modifying the C586 *cis* sequence through editing at site C581, increasing its affinity for a putative unknown second PPR protein. A model for MEF31 action on the *tatC* transcript will be presented.

This work was supported by grants from Fondecyt-Chile (n° 1141197), Millenium Nucleus for Plant Systems and Synthetic Biology (NC130030, ICM-MINECON, Chile) and the Deutsche Forschungsgemeinschaft.

P.2.4-005
Vesicular elongator regulates axonal transport via a-tubulin acetylation

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The Elongator complex (ELP1-ELP6) regulates distinct cellular processes, including transcription, translation and post-translational modifications. A growing body of evidence supports an emerging role for mutations that disrupt the activity of the Elongator complex role in various pathologies characterized by synaptic defects, ranging from familial dysautonomia, amyotrophic lateral sclerosis, epilepsy, to intellectual disabilities and autism.

Recent findings have shown that loss of Elongator activity correlates with a reduction in a-Tubulin acetylation and that ELP1 co-localizes with synaptic vesicle markers. Here, we aimed to integrate these findings in order to decipher whether vesicular Elongator could control axonal transport. To this end we applied a multidisciplinary approach *in-vitro* and *in-vivo* using biochemical, proteomic, electron microscopy and live imaging analyses of human, mice, and Drosophila models. Our results demonstrate that 1) ELP1 is required for Elongator complex recruitment to axonal vesicles; 2) Elongator transport defects are associated with reduction in microtubules acetylation and could be rescued by inhibition of a-Tubulin deacetylase HDAC6; 3) brain vesicles derived from *Elp3* cKO mice show lower acetylation activity of a-Tubulin in cell free *in-vitro* assay. Altogether these results uncover the mechanism through which Elongator controls axonal transport and places axonal vesicles as predominant drivers of a-tubulin acetylation. *These authors contributed equally to the work

P.2.4-006
Systematic identification and functional characterization of contact site resident proteins in *Saccharomyces cerevisiae*

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Contact sites are areas of close apposition between organelle membranes that enable the transfer of lipids, metabolites and small molecules in an efficient manner. In recent years, several new membrane contact sites have been described and the tethering proteins that hold the two membranes together have been identified. Despite these advances we are still far from having a deep understanding of the function and regulation of most contact sites. To mechanistically characterize a contact site it is essential to know its entire repertoire of resident proteins yet very few proteins that are enriched in any contact site have, to date, been described. To systematically characterize the proteome of contact sites we have decided to utilize a panel of split fluorescence sensors for a diversity of contact sites in *Saccharomyces cerevisiae*. With this sensor one part of a fluorophore is fused to the outer membrane of one organelle while the second is fused to another organelle's membranes. If a contact site is present between both membranes, a fluorescent signal is emitted. We have taken sensors for 15 contacts between several organelles such as mitochondria, peroxisomes, lipid droplets, vacuoles, the plasma membrane and the endoplasmic reticulum, and crossed these split-tagged strains with a novel library of mCherry tagged yeast proteins. By analyzing co-localization events we have discovered a large number of new, previously unappreciated, contact site residents. Following up on these proteins, especially those that are conserved to humans, should

The FEBS Journal **284** (Suppl. 1) (2017) 104–392 DOI: 10.1111/febs.14174