Cu^{2+}- treated plants. In both organs, the decrease of auxin-dependent gene expression was found, which can partly explain the growth inhibitions.

In plant organs, Cu^{2+} treatment results in severe morphological responses during which the endogenous hormonal balance and signal transduction are affected. Auxin and NO negatively regulate each other's level and NO intensifies the metal-induced cotyledon expansion, but mitigates elongation processes under short-term Cu^{2+} exposure. Besides hormonal system, nitric oxide metabolism was also influenced by copper. In the root tips, this heavy metal excess induced NO generation, while NO content in lateral roots was not affected by the treatments. Using *nia1nia2* mutants, nitrate reductase enzyme as a putative source of Cu^{2+}-induced NO was identified in *Arabidopsis* primary roots.

Moreover, ROS levels were also influenced by copper. Under copper treatment, NO might play a protective role by regulating ROS levels possibly through modulation of the antioxidant activity.

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Studies on the cellular functions of newly discovered Prion family protein Shadoo

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The SPRN gene encodes the Shadoo glycoprotein (Sho), a central nervous system-expressed member of the prion protein super family. Sho is highly conserved from fish to mammals. SPRN is conserved in mammals, as is the prion gene PRNP, but in sheep SPRN and PRNP are both marked by polymorphic variation, suggestive of a shared selection pressure within these scrapie disease-prone livestock animals. In rodent models of prion disease there are reduced levels of Sho in infected tissues, defining a form of cross-regulation between full-length Sho holoprotein and PrP^{Sc}. The similarities between Sho and PrP N-terminus are the natively unfolded nature of polypeptide chains, a hydrophobic domain and tandem repeats with positively charged residues. Indeed, scrutiny of Sho's biochemical properties in uninfected cells has revealed overlaps with the properties of PrP^{C}, these including shared protein binding partners.

Prion protein functions as a metal binding protein because divalent cations such as copper, zinc and manganese can bind to the octapeptide repeat sequences in the N-terminus of PrP^{C}. Since the binding of these metals to the octapeptide has been proposed to influence both structural and functional properties of prion protein, alterations in transition metal levels can alter the course of the disease. As a member of the prion protein super family, we thought that Sho protein may behave like PrP as a metal binding protein, although it lacks the octapeptide region.

We carried out experiments on N2a cell lines stably expressing the Sho protein applying various concentrations of transition divalent metals. We could see the membrane internalisation of Sho protein induced by Co^{+2}, Mn^{+2} and Zn^{+2} ions. Also, we observed that the Sho expressing cells showed protection against the cytotoxic effects of Mn^{+2}.

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Identification and characterization of a novel circadian clock mutant in *Arabidopsis thaliana*

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The circadian clock is a biological timing mechanism that provides rhythmicity to gene expression, metabolism, and physiology with ~24h periodicity. The central oscillator of eukaryotic clocks is based on the network of clock genes and proteins, which are interconnected by transcriptional/translational negative feed-back loops.

Current models of the plant circadian clock postulate three interlocked feedback loops. A pair of single Myb-domain transcription factors, *CIRCADIAN CLOCK ASSOCIATED1 (CCA1)* and *LATE ELONGATED HYPOCOTYL (LHY)*, plays central roles in two loops. In one loop, *CCA1* and *LHY* repress the expression of the Pseudo-Response Regulator gene *TIMING OF CAB EXPRESSION 1 (TOC1)*. *TOC1* closes the first loop by inducing *CCA1* and *LHY* transcription for the next cycle. In a second loop, *PRR7* and *PRR9*, are induced by *CCA1* and *LHY*. *CCA1* and *LHY* are subsequently repressed by *PRR7* and *PRR9*. In a third loop, *GIGANTEA (GI)* and, possibly, *PRR5* are positive regulators of *TOC1*. *GI* is negatively regulated by both *CCA1/LHY* and *TOC1*.