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Editorial

The Role of Chloroplast Signals in Cold Acclimation in Arabidopsis

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In the post-genome era, one of the main problems of plant genetics is to identify functions of genes. A massive change in gene expression is an important component of the cold acclimation process [1]. Around one thousand genes have been found to be differentially expressed following cold exposure in the model plant *Arabidopsis thaliana*. Transcriptional re-programming occurs during cold acclimation to induce expression of around 100 Cold-Regulated (*COR*) genes, responsible for producing cryoprotective molecules. Central to this transcriptional regulation are the *CBF* (C-repeat-Binding Factor) genes that encode AP2/ERF family transcription factors [2]. A general scheme of genetic control of cold stress responses can be represented as a network of transcription factors and genes that are directly responsible for morphological changes leading to cold resistance. It is well known that there are two main pathways of cold response: ABA-dependent and ABA-independent. There is also an intersection between abiotic and biotic response pathways as well as between different kinds of abiotic response pathways such as cold, dehydration and high light [3]. However, functions of most cold response genes are still not found. On the other hand, the agricultural range of many important crop species is limited by their maximum freezing tolerance capacity, and freezing stress-related damage can result in considerable crop productivity losses [4].

We pay attention in our work on the mutation in the *RpoTp* gene which product is a nuclear-encoded chloroplast RNA-polymerase. This mutation causes great cold sensitivity of the mutant plants. Interestingly, this mutation negatively affects the accumulation of *COR* gene transcripts in response to cold but strongly induces expression of *CBF* genes, stronger than in the Wild-Type (WT) plants (Novokreshchenova, unpublished data). Chloroplasts do not just carry out photosynthesis (photoreduction of carbon, nitrogen, and sulphur), but are central hubs in plant metabolism [5]. They manufacture fatty acids, aromatic and non-aromatic amino acids (essential for protein synthesis, but also for a vast array of plant secondary metabolites), purine and pyrimidine bases, isoprenoids (like carotenoids and sterols) and tetrapyrroles (like hem and chlorophyll) [6]. Plastids have retained a semi-autonomous character, minimal genetic machinery, and genes for a small number of polypeptides, the expression of which needs to be directed by the nucleus at appropriate periods of time. However, the majority of plastid proteins are encoded in the nucleus, translated in the cytosol, imported into the organelle and further targeted to one of its suborganellar compartments. Plastids also need to grow and multiply to keep pace with their 'host' cells, and increase their number by binary fission. Furthermore, plastids 'report' on their physiological status to the nucleus of the cell, to ensure coordination between the two genomes [7]. There are several mechanisms by which the chloroplast may influence nuclear gene expression, a process known as retrograde signalling. In the classical case, the retrograde signal is generated in organelle(s), then it is exported and traverses the cytosol to act in the nucleus. Several of such classical retrograde signals have been tentatively identified. Messenger roles have also been proposed for factors which display some, but not all, of the characteristics attributed to classical retrograde signals [8]. Inaba, et al. [9] believes that current model of retrograde signalling from plastid to nucleus includes GUN (Genomes Uncoupled)-dependent, ROS-dependent, and redox-dependent pathways.

At present, the retrograde or plastid signals that regulate nuclear gene expression during cold acclimation induce a great interest [10,11]. It has been suggested that chloroplasts could act as sensors of changes in temperature [12]. However, it is still unclear how they transmit information to the rest of the cell to trigger the acclimation response. The *gun* mutants of *Arabidopsis thaliana* have been used to demonstrate that Mg-protoporphyrin (Mg-Proto) acts as a plastid signal to repress the transcription of nuclear photosynthesis genes. It is unclear how Mg-Proto triggers this repression, but several components of this pathway have been recently identified [11]. GUN5 is the ChlH subunit of Mg-chelatase, which produces Mg-Proto. It is clear that there are multiple plastid-to-nucleus signaling pathways and that the original notion of a single "plastid signal" is too simplistic [13].

Kindgren, et al. also demonstrated that two independent mutant alleles of the H-subunit of Mg-chelatase [11], CHLH, *gun5-1* and *cch* in Arabidopsis showed a stronger induction of CBF1-3 expression but the expression levels of *COR* genes were generally lower compared to WT. Interestenly, no difference in freezing tolerance could be detected between wild type and *gun5* in

non-acclimated control plants using electrolyte leakage assay. The LT50 temperature was -4 °C for both WT and *gun5*. In contrast, the *cch* mutant showed a lower freezing tolerance in non-acclimated plants. After cold-acclimation for three days, WT plants were considerably more tolerant to freezing temperatures compared to the *gun5* and *cch* plants [11]. In our work we observed similar results using *gun5* mutants in expression levels and electrolyte leakage assays (Kurbidaeva, Novokreshchenova unpublised). We suggest that before making any conclusion it is necessary to perform whole plant freezing assay as the electrolyte leakage assay is limited by the leaf tissue [14].

New results regarding the involvement of retrograde signalling in cold stress response demonstrated the importance of chloroplasts for the cold acclimation process and further suggested that impaired plastid function could result in the inhibition of protein synthesis at low temperature. However the mechanism leading to such an event is still unknown.

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