



ABSTRACT

*“Defining the mitochondrial stress response:
insights into plant retrograde regulation”*

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As plants are sessile organisms and are exposed to highly variable growth conditions, their survival is determined to a large extent by the capacity to respond to these changes. Being major players in energy metabolism and biosynthesis, mitochondria are considered a prime target of stress-induced cellular damage. However, relatively little is known about how plant mitochondria respond to stress conditions and how this response is regulated. In this study we have used large scale expression data and subcellular localisation information to define a core set of highly stress responsive genes encoding mitochondrial proteins, providing model systems for exploring mitochondrial stress responses and their regulation. By studying the promoters of several of these genes, we have identified cis-acting regulatory elements (CARE's) and different regulatory pathways that control gene expression of stress-responsive mitochondrial proteins in Arabidopsis. Many of these genes respond directly to inhibition of mitochondrial function by chemical inhibition or mutations and are considered model genes for mitochondrial retrograde regulation, the feedback signalling from organelle to nucleus. Although significant breakthroughs have been made in the study of mitochondrial retrograde signalling in yeast, no components involved in plant mitochondrial retrograde signalling have been identified to date. Functional characterization of the promoter of alternative oxidase 1a (AOX1a) from Arabidopsis, a marker for mitochondrial retrograde response, identified a strong repressor element that was necessary for increased promoter activity in response to the mitochondrial complex I inhibitor rotenone. This element overlaps with a previously identified potential binding site for the transcription factor ABSCISIC ACID INSENSITIVE4 (ABI4). Binding of the ABI4 transcription factor to this region of the AOX1a promoter was demonstrated by electromobility shift and yeast one-hybrid assays. Analysis of transcript abundance for AOX1a in *abi4* mutant lines revealed significantly increased levels of AOX1a mRNA that could not be further induced by rotenone, consistent with the role of ABI4 as a repressor that is derepressed in response to rotenone. These results show that ABI4 plays a central role in mediating mitochondrial retrograde signals to induce the expression of AOX1a. Furthermore, they provide a molecular link between mitochondrial and chloroplast retrograde signalling, as ABI4 has been previously shown to act downstream of at least two chloroplast retrograde signalling pathways.