ABSTRACT

ROLE OF ADA2b AND GCN5 IN *COR* GENE EXPRESSION DURING COLD ACCLIMATION IN *ARABIDOPSIS*

By

Kanchan Amol Pavangadkar

Covalent modifications of histones play important roles in the regulation of transcription. Acetylation of lysine residues on the amino-terminal tails of histones is associated with transcriptionally active genes and is catalyzed by histone acetyltransferases (HAT). GCN5 (a HAT) and ADA2 are components of coactivator complexes such as SAGA in yeast. The *Arabidopsis* genome encodes one homologue of GCN5 and two homologues of ADA2 (ADA2a and ADA2b).

Arabidopsis ADA2 and GCN5 physically interact with the transcriptional activator CBF1 *in vitro* which activates the expression of cold-regulated (*COR*) genes during cold acclimation. Cold acclimation is the process by which plants increase freezing tolerance upon exposure to low non-freezing temperatures. CBF1 binds to the cold/dehydration responsive element (CRT/DRE) present in *COR* gene promoters. *ada2b* and *gcn5* mutants show a delay in activation and a reduction in expression of *COR* genes during cold acclimation. Chromatin immunoprecipitation (ChIP) assays show that the acetylation of histone H3 at the *COR* promoters increases upon cold acclimation. Thus we hypothesized that CBF recruits GCN5 and ADA2b to the *COR* gene promoters to acetylate histones and thus help activate *COR* genes. ChIP assays on plants overexpressing CBF1 show increased acetylation of histone H3 even without cold stress

and the acetylation levels increase further upon cold acclimation. Thus, CBF is sufficient to bring about an increase in acetylation of histone H3 at the *COR* gene promoters.

The acetylation levels of histone H3 in *ada2b* and *gcn5* mutants are similar to wild type plants upon cold acclimation and thus ADA2b and GCN5 are not required for histone H3 acetylation at the *COR* gene promoters. The *Arabidopsis* genome encodes 12 potential HATs and the HAC1, HAC5, HAC12 and TAF1 HATs were tested for their ability to regulate *COR* gene expression. None of these HATs were required for the acetylation of histone H3 at the *COR* gene promoters as determined by ChIP assays. Thus we conclude that the acetylation of histone H3 at the *COR* gene promoters is not solely dependent on any of the HATs tested.