Genome silencing, Cell Division and Phytohormone Biosynthesis in Winter Wheat (a possible relationship)

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It is not clear to what extent gene silencing plays a role in polyploid gene expression nor is it clear whether there is a mechanism to silence similar genes or all genes involved in a particular pathway. It is known that epigenetic marks such as methylation and deacetylation can influence gene expression resulting in silencing of groups of genes.

One group of genes for which deacetylation has been reported to play a key role is the set of ribosomal RNA (rRNA) genes. These genes are expressed at the Nucleolar Organizer Regions (NORs). Of the Arabidopsis histone deacetylases (HDACs), HDAC6 has been reported as playing a significant role in silencing of the rRNA genes by modifications of those genes [1]. In Figure 1, HDAC6 can be seen localized to the nucleolus where rRNA genes are normally transcribed. In this case HDAC6 switched off the rRNA genes. Early et al. [1] have also described a model that predicts the key events that allow switching from active transcription to repression of transcription. These key events involve de novo cytosine methylation and histone deacetylation.

Rather than turning off whole sets of genes, epigenetic marks can be limited to regions of one sister chromatin and not another. Such differential epigenetic marks on sister chromatids can lead to the generation of daughter cells in which one cell expresses particular genes while in the other daughter cell the genes are silenced [2]. Assuming no other controls are placed on the expression or lack of expression of these genes, it means that there will be cells in the same tissue that are expressing a particular protein and other cells that do not express that protein. As a result the cell fates are different. An additional aspect of this control is the role of stimuli in influencing the epigenetic changes leading to differential gene expression.

At the transcriptional level, there are many studies utilizing DNA microarrays and next generation RNA sequencing to identify gene networks responsive to various stimuli. This is a very complex and vast area of study. For now, I will focus the discussion on plants and response to stimuli to withstand abiotic stress. Our studies have focused on deciphering the mechanism(s) that allow plants, particularly cereals, to withstand freezing conditions. For most organisms, prior exposure to an attenuated version of the stress is known to prepare the organism to withstand the stress (acclimation). In studying freeze survival, many studies have focused on what occurs during that attenuation period termed cold acclimation. In nature, cold acclimation occurs during the autumn and can be mimicked in the laboratory with low non-freezing temperatures under dehydration conditions and low light intensity [2].

*Triticum aestivum is a hexaploid (AA, BB and DD). Therefore in our system we have to be aware as to whether we are observing gene expression from all three genomes or from a subset. This is important for many reasons including understanding dosage compensation in wheat. Firstly, I will describe the experimental system and then we will return to the role of the genomes in cold acclimation.

Having an experimental system that is very closely related and differing significantly only in survival of the stress is very valuable. We have such a system. Utilizing winter wheat lines generated by azide mutagenesis and varying in freeze survival and not vernalization, we have been able to compare expression of the cold acclimation regulon consisting of transcription factors known to map to the long arm of chromosome 5, the frost resistant (Fv) loci [3, 4]. These transcription factors belong to the group known as CBF/DREB based on identification of the binding of those proteins to C-repeat/dehydration-responsive elements (CRT/DRE) [5, 6]. We identified two of these genes Cbf-12 and Cbf-14; from the Cbf central cluster of the Fr loci [7] as associated with the difference in freeze survival between the lines. Cbf-3, 5, 6 are significantly differentially regulated between FR and FS (Figure 2). However, these Cbf genes are not located within the central cluster.

As stated earlier, it is of great value when studying gene expression in a polyploid to be able to ascertain which if any of the genomes is a key player. As seen in figure 2, Cbf-D22 is silenced in response to cold acclimation in both lines. However, Cbf-A22 and Cbf-B22 are upregulated almost to the same extent in response to cold acclimation in both lines. With respect to Cbf-22, these results suggest silencing to achieve dosage compensation to limit its expression. How and why the Cbf-D22 is silenced is not known.

The results depicted in Figure 2, also reveal that silencing is not limited to one genome. Although Cbf-B22 is activated in both lines another gene on the B genome Cbf-B10 is silenced in both lines.

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ABA dependent systems suggest an ABA/Jasmonic acid antagonistic system to maintain the meristematic crown tissue in a dormant state. Lack of cell division as reflected by the shutdown of the E2F pathway and the absence of growth during cold acclimation [3] leads to the design of a model that predicts that deacetylation and methylation events may occur to silence genes involved in cell division and biosynthetic pathways (pathways of Jasmonic Acid but not that of ABA).

**References**


