REGULATORY COMPONENTS INVOLVED IN COLD TOLERANCE OF BARLEY CELLS

Ildikó Vashegyi¹ – Zsuzsa Marozsán-Tóth¹ – Gábor Galiba¹ – Petre I. Dobrev² – Radomira Vankova² – Balázs Tóth¹

¹ Department of Plant Molecular Biology, Agricultural Institute, Centre for Agricultural Research, Hungarian Academy of Sciences, Brunszvik u. 2, Martonvásár 2462, Hungary, e-mail: vashegyi.ildiko@agrarr.mta.hu
² Laboratory of Hormonal Regulations in Plants, Institute of Experimental Botany, Academy of Sciences of the Czech Republic, Rozvojova 263, Prague 16502, Czech Republic

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Micro- and macroclimatic changes fundamentally determine growth rate, development, crop production and geographical distribution of plant species. The existence of successful defensive mechanisms against the damaging effects of low temperature is essential for survival and sufficient seed production of plants.

In winter-type cereals cold acclimation process is activated by low temperature, and it leads to elevated level of resistance against harmful physiological effects of suboptimal temperature. One of the most important gene expression regulator units in this mechanism is the CBF-COR system. However, cold acclimation mechanism is a very complex phenomenon, the process is influenced by many factors, e.g. falling temperature, day length, spectral composition of irradiated light, as well as local and systemic internal signals. Because of this, realignment of the gene expression pattern connected to the cold acclimation mechanism and its phenotypical effects is very difficult to investigate excluding the influence of other factors with interfering action. Basic cellular and biochemical changes caused by only the low temperature, independently of another factors mentioned above are mainly undiscovered. Therefore, elemental cold response of the CBF-COR system was compared in seedlings and dark-grown, dedifferentiated, meristemoid callus cultures of winter barley. Detailed characteristics of CBF-COR induction and effects of cold-hardening were also studied in barley callus cultures at the gene expression, hormone composition and freezing tolerance levels in the presence or absence of Dicamba, the exogenous auxin analogue used in tissue cultivation.

Our results suggest the presence of a basal, cold-responsive activation mechanism of CBF and COR genes with the highest influence on the evolvement of frost resistance, which is independent of the differentiated state of cells or chloroplast-related, light-induced and systemic signals. However, these factors seem to be required for reaching the maximum level of activation. The exogenous auxin analogue, Dicamba, seems to be rather a coinducer in this process, since it does not affect the initiation or the characteristic of the activation, only influences the magnitude of the response.

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