

Molecular mechanisms of acquired thermotolerance in plants

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Sessile organisms such as plants experience daily heat-shocks, with temperature variations rising up to 30°C between 2 am and 2 pm. Diurnal temperature variations may be particularly extreme for organisms that evolved far from large temperate water bodies, such as oceans, and populated interperate inland habitats exposed to abrupt environmental stresses, such as freezing, heating, excess light, excess oxygen radicals and to more gradual seasonal stresses, such as dehydration, nutriment starvation and salt stress. To cope with a noxious heat shock, exposed organisms need to timely develop appropriate molecular defenses that can prevent and repair heat damages, mostly in labile lipids and proteins. A mild rise of ambient temperature transiently increases the fluidity of the plasma membrane, which in turn, activates the transient opening and depolarization of specific heat-sensitive Ca²⁺ channels. Members of the cyclic nucleotide gates channels (CNGCs) were recently identified as being the primary heat-sensors of land plants. Depending on the rate of temperature increase, the duration and intensity of the heat priming preconditions, terrestrial plants may thus acquire an array of heat shock protein-based thermotolerance mechanisms against upcoming, otherwise lethal, extreme heat waves. Quantitative proteomics reveals absolute changes of cellular protein concentrations during heat-shock treatments.

Key words: Heat, stress, Hsp, acquired thermotolerance