Another Brick in the Winter Fortress

BY KARL DANNEBERGER

Similar to fortifying a military base or regiment prior to battle, turfgrasses fortify themselves for the coming winter. Lack of fortification or cold acclimation and hardiness can cause death. For example, a plant growing at a warm temperature that's suddenly exposed to a subfreezing temperature dies. Yet, the same plant that's exposed to a period of low nonfreezing temperatures prior to subfreezing temperatures survives. Why?

In general, we know the mechanism of freeze tolerance in turfgrasses. Freeze tolerance, and conversely freeze injury, is due to how the turfgrass plant reacts to cell dehydration. Water freezes intercellulary in freezing temperatures, resulting in a decrease in water potential outside the cell. In response, the differential water potential within the cell moves toward the external ice crystals.

Simply stated, the cell dehydrates in response to freezing temperatures. The colder the temperature, the more water flows out of the cell toward the frozen water in the intercellular spaces. At 14 degrees Fahrenheit (-10 degrees Celsius), 90 percent of the osmotically active water will move out of the cell (Thomashow, 1998).

From the above description, we can see that if cells are flush with water either from succulent or rapid growth, then rapid drops in temperature can cause injury. During fall there has to be changes in the turfgrass plant so that a redistribution of water or hardiness occurs (Beard, 1973). Exposure to low but nonfreezing temperatures (less than 50 degrees F (10 C) is critical for inducing plant hardiness. Currently, the induction of cold hardiness in response to low temperatures is a hot area of research.

Scientists have identified and studied the roll of specific plant genes in freeze tolerance. A group of genes called cold-response (COR) genes apparently plays an important role. The activation of these genes requires a period of low but nonfreezing temperatures (32 to 50 degrees F (0 to 10 degrees C). The activation of these genes is then associated with the hardening or freeze tolerance of the plants. A possible reason why plants in effect die when exposed to freezing temperatures without a hardening period is due to the lack of COR gene activation. Interestingly, light in addition to low but nonfreezing temperatures is needed for gene activation (Wanner & Junttila, 1999).

GENETIC RESEARCH MIGHT UNVEIL NEW WAYS TO FORTIFY AND ACCLIMATE TURF FOR WINTER

Although none of this work has been done on turfgrass plants, the reduced or inactivation of COR genes might explain why we see freeze injury to *Poa annua* and bermudagrass turf under low light conditions. For example, increased shading on Northern golf course greens favors establishment and spread of *Poa annua*, which is susceptible to freeze injury. Reduced light in fall can negatively impact the hardening process by impacting COR gene activity. The lack or reduced state of cold hardiness increases the likelihood of freeze injury in late winter.

Continued research into the mechanisms involved in turf hardening is needed. By understanding the mechanisms involved in hardening, we can better understand why low light conditions are detrimental to turf, which will lead to better management practices to increase the turf's cold hardiness. For example, exposing a newly seeded putting green to low but nonfreezing temperatures prior to covering might help those juvenile plants reach a greater degree of cold hardiness.

Superintendents build a winter "turf fortress" through management practices. One more brick in this fortress is the role of COR genes and how light affects them.

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