Prime Time for Transgenerational Defense

Protection of precious offspring against potentially life-threatening conditions is marked in the genes of virtually all species. Because plants are sessile organisms and practically unable to communicate with their offspring, they need ingenious ways to "inform" them about the potential dangers in their environment. Plants are continuously exposed to a variety of biotic and abiotic stresses, and the chances are high that progeny are exposed to the same stress conditions as the parents have encountered. During evolution, the process of survival of the fittest genetically equipped plants species to endure the stress conditions that frequently occur in every generation. However, less frequent stress situations may only persist for only 1 or a few generations, which is often too short for genetic adaptations to establish in the population. In the past decade, evidence accumulated that suggests that environmental stress conditions can cause effective adaptations in the next plant generation. To pass on an acquired stress adaptation to the next generation, the stress condition must be memorized in a form that is propagated through mitotic and meiotic divisions, even when the stress is no longer present. Epigenetic changes in genetic material, such as changes in DNA methylation patterns and histone modification, can heritably and reversibly modify the expression of genes without changing the DNA sequence. Also, small interfering RNAs (siRNAs) have been shown to be able to carry the memory of an experienced stress situation to the next.

Examples of epigenetic and heritable adaptive responses to protect future generations against biotic stress are scarce. In this issue of *Plant Physiology*, three independent studies provide novel insights into transgenerationally induced resistance. Induced resistance is often associated with priming for enhanced defense. Priming can be triggered biologically, such as in healthy plant parts of pathogen-infected or herbivore-damaged plants, or chemically by low doses of the defense hormones salicylic acid (SA) or jasmonic acid or the nonproteinaceous amino acid β-aminobutyric acid. In primed plants, cellular defense responses are not activated directly by the priming agent but are memorized and expressed in an accelerated manner after perception of a second biotic stress signal, a process that is likely to have an epigenetic basis.

In the first study, by Slaughter et al. (pp. 835–843), Arabidopsis (*Arabidopsis thaliana*) plants were primed with β-aminobutyric acid or an avirulent isolate of the bacterial pathogen *Pseudomonas syringae*. The progeny of the primed parental plants displayed a potentiated expression pattern of SA-dependent, defense-related genes and an enhanced level of resistance to infection by virulent *P. syringae* and the downy mildew pathogen *Hyaloperonospora arabidopsidis*. Interestingly, the transgenerationally primed plants were also "primed to be primed" because their offspring showed an even stronger priming phenotype than their primed parents.

In the second study (pp. 844–853), Luna et al. induced the primed state in parental Arabidopsis plants by repeated inoculations with virulent *P. syringae*. Also in this study, the primed state was passed on to the next generation and was even sustained over 1 stress-free generation, indicating that the phenomenon is truly epigenetically regulated. Transgenerational acquired resistance was blocked in the SA signaling mutant *nonexpressor of pathogenesis-related genes1* (*npr1*), revealing a central role of NPR1 in next-generation immune priming. Interestingly, transgenerationally induced resistance was shown to be associated with chromatin modifications at the promoters of the SA-responsive genes *PATHOGENESIS-RELATED1*, *WRKY6*, and *WRKY53* in the progeny of primed plants. In addition, evidence was provided for a role of DNA methylation because the hypomethylated DNA status of the DNA methylation-related *drm1drm2cmt3* triple mutant, which is mutated in *DOMAINS REARRANGED METHYLASE1 AND 2 AND CHROMOMETHYLASE3*, mimicked the transgenerationally acquired resistance phenotype of the progeny of primed parental plants. The authors conclude that transgenerational immune priming, as triggered by bacterial infections, is transmitted by hypomethylation of genes that direct priming of SA-dependent genes in following generations.

In the third study, Rasmann et al. (pp. 854–863) investigated the transgenerational persistence of herbivore-induced resistance in Arabidopsis and tomato (*Solanum lycopersicum*). The authors show that herbivory-induced resistance was passed on to subsequent generations and that this phenomenon required jasmonic acid signaling. Intriguingly, Arabidopsis mutants impaired in the biogenesis of siRNAs did not transmit the herbivore-induced resistance to the next generation, indicating an important role for siRNAs in epigenetic and heritable resistance to insect herbivory.

Within-generation induced resistance is a well-characterized phenomenon that is believed to have evolved to save energy under enemy-free conditions because it only involves costs when defenses are activated upon attack by the enemy. The three studies in this issue clearly demonstrate that priming for enhanced resistance also extends to next generations and that epigenetic regulatory mechanisms, such as DNA methylation, chromatin remodeling, and siRNAs, play a central role the regulation of these transgenerational plant immune responses. These exciting discoveries pave the way to further understand the molecular details of how plants are able to heritably protect their offspring against potential enemies in their environment without making changes in their DNA sequence.

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