CHAPTER 15

Plant transcriptional regulation in modulating cross-tolerance to stress

S.V. Ramesh[a], **Ranjeet Ranjan Kumar**[b], **Shelly Praveen**[b]
[a]ICAR-Central Plantation Crops Research Institute (ICAR-CPCRI), Kasaragod, India
[b]ICAR-Indian Agricultural Research Institute (ICAR-IARI), New Delhi, India

15.1 Introduction

Plants continuously interact with biotic and abiotic factors in a complex and ever-changing environment. These multifaceted interactions induce changes in various metabolic processes, ultimately affecting the growth and development of plants. The response of a plant to stressors depends on its ability to readjust its metabolic, biochemical, and molecular systems to maintain homeostasis within the framework of its genetic makeup.[1] The key components of stress-response mechanism include a stimulus induced by stress, signaling molecules, transducers, RNA regulators, and target genes. The concerted activity of these components culminates in stress responses causing biochemical, morphological, and physiological changes.[2] In these cellular responses, plants make use of shared components and signaling pathways. Recent studies have demonstrated that prior exposure of a plant to a particular abiotic stress enhances its tolerance to the ensuing biotic stress. Similarly, environmental changes due to climate change phenomenon have also expanded the host range of pathogens and foster rapid development of virulent pathogens/pests.[3] Additionally, the molecular response of plants exposed to multiples stresses, for instance drought and heat stress, is unique, hence the molecular nuances of individual stresses cannot be directly hypothesized for combinatorial stresses.[4] These investigations highlight that, despite sharing the molecular components, each stressor and even combination of stressors activates 'a tailored' response in plants. Hence, it is pertinent to gain insights regarding the adaptive response of plants toward a combination of stresses.

15.2 Cross-stress tolerance

Prior exposure of plants to a stress triggers enhanced tolerance to later occurring biotic or abiotic stressors, which is referred as cross-stress tolerance.[5] The molecular basis of cross-tolerance is the synergistic activation of defense related nonspecific pathways (Fig. 15.1). Interestingly, plants that are exposed to single stresses individually are more susceptible than the same plants exposed to simultaneous attack by multiple stresses. Hence, it is

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