Journal of Plant Physiology & Pathology

Editorial

A SCITECHNOL JOURNAL

Abiotic Stress Signaling in Plants: Mechanisms for Survival and Adaptation

Dr. Ravi Nair*

Department of Botany, Indian Institute of Science, India

***Corresponding author:** Dr. Ravi Nair, Department of Botany, Indian Institute of Science, India, Email: rnair@iisc.ac.in

Citation: Ravi N (2025) Abiotic Stress Signaling in Plants: Mechanisms for Survival and Adaptation. J Plant Physiol Pathol 13: 403

Received: 01-Sep-2025, Manuscript No. jppp-26-183740; **Editor assigned:** 4-Sep-2025, Pre-QC No. jppp-26-183740 (PQ); **Reviewed:** 17-Sep-2025, QC No. jppp-26-183740; **Revised:** 24-Sep-2025, Manuscript No. jppp-26-183740 (R); **Published:** 30-Sep-2025, DOI: 10.4172/2329-955X.1000403

Introduction

Plants are constantly exposed to environmental stresses that are non-living in nature, such as drought, salinity, extreme temperatures, heavy metals, and oxidative stress. These stresses, collectively called **abiotic stresses**, significantly affect plant growth, development, and productivity. Unlike animals, plants cannot escape adverse conditions, so they have evolved complex **abiotic stress signaling pathways** to perceive, process, and respond to environmental cues. Understanding these signaling mechanisms is crucial for developing stress-resilient crops and ensuring sustainable agriculture under changing climate conditions [1,2].

Discussion

Abiotic stress signaling begins with **stress perception** by specialized sensors located in the plasma membrane, cell wall, or cytoplasm. These sensors detect changes in osmotic balance, ionic concentration, temperature, or reactive oxygen species (ROS) levels. Once a stress is perceived, intracellular **signal transduction pathways** are activated, often involving secondary messengers such as calcium ions (Ca^{2+}), reactive oxygen species, cyclic nucleotides, and inositol phosphates. These messengers amplify the stress signal and relay it to downstream components [3,4].

Protein kinases, including mitogen-activated protein kinases (MAPKs) and calcium-dependent protein kinases (CDPKs), play a central role in transducing stress signals. These kinases phosphorylate transcription factors and other regulatory proteins, modulating gene expression to initiate protective responses. For instance, under drought or salinity stress, abscisic acid (ABA) accumulates and binds to its receptors (PYR/PYL/RCAR), triggering a signaling cascade that leads to stomatal closure, osmolyte accumulation, and activation of stress-responsive genes.

Transcription factors such as DREB, NAC, and bZIP regulate the expression of genes involved in osmoprotection, antioxidant defense, and heat shock protein production. These molecular responses collectively enhance **stress tolerance** by maintaining cellular homeostasis, reducing oxidative damage, and protecting proteins and membranes [5].

Recent studies have highlighted the role of **epigenetic modifications** and small RNAs in abiotic stress signaling. DNA methylation, histone modifications, and microRNAs can modulate stress-responsive gene expression, enabling plants to “remember” prior stress exposure and respond more efficiently in the future.

Conclusion

Abiotic stress signaling is a complex network of perception, transduction, and gene regulation that allows plants to sense and adapt to adverse environmental conditions. By integrating secondary messengers, protein kinases, transcription factors, and epigenetic regulators, plants coordinate physiological and molecular responses that enhance survival and productivity. Insights into these signaling pathways offer opportunities for breeding and engineering stress-resilient crops, supporting sustainable agriculture and global food security in the face of climate change.

References

1. Dalessandri D, Zotti F, Laffranchi L, Migliorati M, Isola G, et al. (2019) Treatment of recurrent aphthous stomatitis (RAS; Aphthae; canker sores) with a barrier forming mouth rinse or topical gel formulation containing hyaluronic acid: A retrospective clinical study. BMC Oral Health 19: 1-10.
2. Casale M, Moffa A, Vella P, Rinaldi V, Lopez PA, et al. (2017) Systematic review: The efficacy of topical hyaluronic acid on oral ulcers. J Biol Regul Homeost Agents 31(4 Suppl 2): 63–69.
3. Lee JH, Jung JY, Bang D (2008) The efficacy of topical 0.2% hyaluronic acid gel on recurrent oral ulcers: Comparison between recurrent aphthous ulcers and the oral ulcers of Behçet’s disease. J Eur Acad Dermatology Venereol 22: 590-595.
4. Nolan A, Baillie C, Badminton J, Rudralingham M, Seymour RA (2006) The efficacy of topical hyaluronic acid in the management of recurrent aphthous ulceration. J Oral Pathol Med 35: 461-465.
5. Koray M, Ofluoglu D, Senemtasi A, İssever H, Yaltirik M (2016) The Efficacy of Hyaluronic Acid Gel in Pain Control of Recurrent Aphthous Stomatitis. Int J Dent Oral Sci 3: 273-275.