



## **Abscissic Acid (ABA) hormone Stimulates Protein Kinase Gene in Cell Signaling for Cell, tissue and plant Growth and Development**

**ABM Sharif hossain<sup>1</sup>, Musamma M Uddin<sup>2</sup>**

<sup>1,2</sup> Program of Biotechnology, Department of Biology, Faculty of Science, University of Hail, Hail, KSA, Saudi Arabia

<sup>2</sup> Biotechnology Program, Institute of Biological Science, Faculty of Science, University of Malaya, Kuala Lumpur, Malaysia

### **Abstract**

The review study was carried out from different research data to find out the innovative latest information on stimulated protein kinase gene in cell signaling in cell, tissue of plant or animal growth and development affected by abscissic Acid (ABA) hormone. From the review study results, ABA stimulated ACPK1 in defined concentration of treatment and the ACPK1 expression as well as enzyme activities altered using ABA concentrations during fruit development. The other phytohormone like gibberellic acid (GA) was also ineffective in this ACPK1 stimulation. ABA sensitivity adapted to drought conditions better than their wild-type species. ABA was required for tolerance to freezing which occurred through the induction of dehydration-tolerance genes. Moreover, cell division and differentiation, genetically modified (GM) might not be occurred by the concentration of ABA hormone. ABA regulated genes were expressed and found new genes like At5g06760, LT130, RD29A.

**Keywords:** Abscissic Acid, protein kinase gene, cell signaling, growth

### **Introduction**

Over the past decades, more than 100 mediators involved in ABA signaling have been identified using molecular genetics, physiological, biochemical and pharmacological studies [1]. The identification of receptors capable of sensing ABA and relaying this signal to other mediators in the signaling pathway. Several reports have claimed to identify the ABA receptors [2]. It was reported that Steroidogenic acute regulatory protein (Star) and related lipid-transfer (START) of proteins were as candidate of ABA receptors [3].

It was stated that abscissic acid (ABA) keeps many significant roles during plant growth and in plants adaptation to their environment. During vegetative growth, ABA took a part as a central signal of plant response to various environmental challenges including drought, salt, and cold stresses [4, 5, 6]. Besides, ABA was responsible for the seed storage reserve synthesis, acquisition of desiccation tolerance and dormancy, and induction of stress tolerance [7].

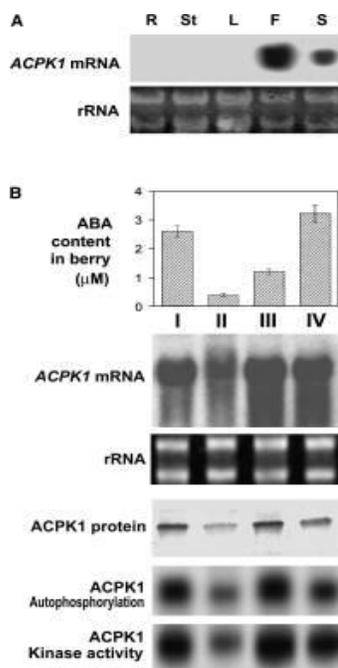
It had been observed that fleshy fruits were the essential portions of reproductive organs and economically important harvest organs. In fleshy fruits as in seeds, ABA regulated various processes concerning assimilate uptake and metabolism to enhance reserve accumulation [8, 9, 10]. The ripening of grape berry was considered to be independent of the hormone ethylene but to be triggered essentially by ABA [11]. Reversible protein phosphorylation, catalyzed by protein kinases and phosphatases had been caused central roles in ABA signal transduction. Calcium-modulated protein phosphatases (PPs) 2C ABI1 and ABI2 are the two most characterized, homologous negative [12, 13, 14]. A nuclear-localized transcription factor homeodomain protein ATHB6 had also been identified as a more downstream component of the ABI1 and ABI2 [15]. In comparison to PP2Cs ABI1 and ABI2, a PP 2A, RCN1, had been identified as a positive

regulator of ABA response involved in early events of ABA signaling [16]. In addition, ABA responded as a kinase substrate of CIPK5 [17].

It had been reported that ABA affected the transcription of most chloroplastic genes [18]. Mutation of chloroplastic isoform CKA4 in *Arabidopsis* showed a phenotype of reduced sensitivity to ABA during seed germination and seedling growth and increased stomatal aperture and leaf water loss [19]. Moreover, these effects were attributed to the down regulation of ABA-responsive genes, including OST1, a representative SnRK2 kinase central to ABA signaling. The similar work suggested that CK2 was involved in retrograde signaling from chloroplast to nucleus, since the expression levels of the transcription factor *ABI4* directly involved in retrograde and ABA signaling were reduced in the *cka4* mutant under ABA treatment [20]. It was stated that recent work analyzed CK2A4, RNAi lines in the CK2 $\alpha$  triple mutant background confirmed the importance of this gene in the regulation of ABA response, lateral root formation and flowering time, in a process that were regulated by retrograde signaling [21]. A different type of protein kinase, mitogen-activated protein kinase (MAPK), had been reported to be activated by ABA [22, 23, 24]. An *Arabidopsis* MAPK, AtMPK3, and a rice MAPK, OsMAPK5, had been identified as ABA-activated MAPKs. The former mediates post germination arrested of development by ABA [25]. and the latter is involved in disease resistance and abiotic stress tolerance [26].

Besides, calcium-dependent histone-phosphorylating activity was evaluated to be activated by ABA in rice seedling, but the CDPK gene responsible for this activity was not identified. The transcripts of a CDPK gene, *NiCDPK1* in tobacco, were enhanced unspecifically by various phytohormones ABA, indole-3-acetic acid (IAA), GA<sub>3</sub>, and synthetic cytokinin benzyladenine

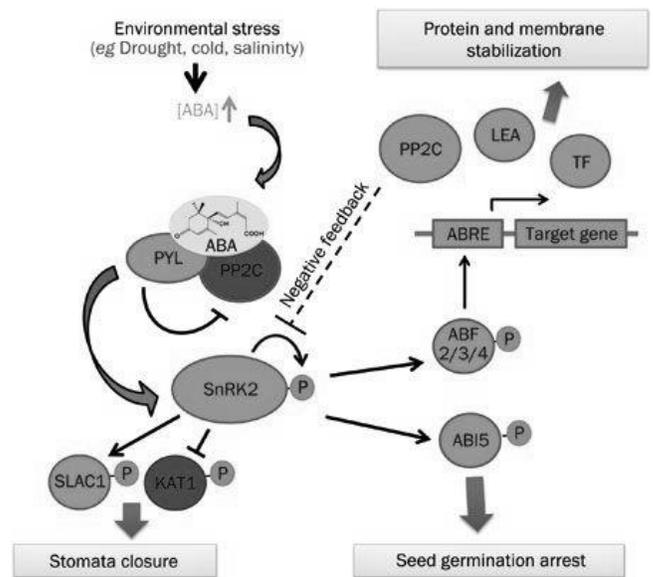
and other growth substances such as jasmonic acid, but it is unknown whether the NtCDPK1 was activated by ABA in its kinase activities [27]. It was reported that expression of ABA response *ACP1* gene in different tissues from root, young stem, leaf, flesh or seed of berry [28].



**Fig 1:** ACPK1 Expression in different tissues of fruit development. A. *ACP1* Expression gene in different tissues. Total RNA (20 µg) from root (R), young stem (St), leaf (L), flesh (F), or seed (S) of berry, transferred on a nylon membrane after electrophoresis in an agarose gel. [28].

It was suggested that ABA sensitivity adapted to drought conditions better than their wild-type counterparts [29]. At the osmotic stress conditions, ABA promoted stomatal closure which prevented water loss through transpiration and the accumulation of osmo-compatible solutes to retain water. It had also been exhibited that ABA was required for tolerance to freezing which occurred through the induction of dehydration-tolerance genes [31]. It was reported [32] that ABA accumulation induced by stress signals, activated PYL ABA receptors to inhibit group A PP2Cs. PP2C inhibition in turn allowed SnRK2 activation through auto phosphorylation. Active SnRK2s mediated the ABA response through the phosphorylation of downstream targets. In addition, SnRK2s phosphorylated the SLAC1 and KAT1 ion channels, resulting in stomatal closure to prevent transpirational water loss in guard cells. In seeds, ABI5 phosphorylation by SnRK2s led to the inhibition of seedling growth.

They also observed that phosphorylation of the AREB1 (ABF2), AREB2 (ABF4), and ABF3 transcription factors activated the transcription of target genes such as Late Embryogenesis Abundant (LEA)-class genes and transcription factors (TF) involved in stress tolerance. Besides, transcriptional increase in the expression of group A PP2C genes might function as a negative feedback loop in the ABA response pathway by inhibiting SnRK2 activity. Positive regulators of the ABA signaling pathway are shown in green, whereas negative regulators are shown in red [32].



**Fig 2:** Abscisic acid perception and signaling: structural mechanisms [32].

It had been exhibited [33] that ABA 2000 ppm had showed the highest inhibition of the peach shoot and bark phloem tissue growth. This might be due to the effectiveness of the given concentration. About 100% inhibition was found in these concentration. Cell division and differentiation might not be occurred and showed dwarfism [34] and genetically modified occurred by the concentration of hormone. Root and shoot growth was inhibited 100% by ABA 2000 ppm concentration [35,36, 37,38]. It was reported that growth of the different organs of peach plant was inhibited by using ABA 1000 ppm. They also reported that shoot and root growth were inhibited 46% at 1000 ppm ABA. The ABA regulated genes were expressed by northern blot [37] and found new genes like At5g06760, LTI30, RD29A.



**Fig 3:** Inhibition of Peach plant growth leaf, root, shoot and phloem tissue inhibition) as dwarf trees using genetically modified technique by ABA hormone [39]

It was reported [39, 40] that high protection rates associated with a significant decrease in the multiplication of *R. solanacearum* in plants pre-inoculated with a DhrpB mutant strain. Neither salicylic acid, nor jasmonic acid/ethylene played a role in the establishment of this resistance. It also showed that 26% of the up-regulated genes in protected plants were involved in the biosynthesis and signaling of abscisic acid (ABA). In addition 21% of these genes were constitutively expressed in the irregular xylem cellulose synthase mutants which presented a high level of

resistance to *R. solanacearum*. They also suggested that inoculation with the DhrpB mutant strain.

Some of these genes were also expressed during the normal embryogenic program when seeds desiccate and embryos become dormant<sup>[41]</sup>. Although different sets of ABA-responsive genes exhibit different patterns of developmental and tissue-specific expression, some of them appear to be part of a general reaction to osmotic stress. This system is a normal part of the embryogenic program but is inducible in vegetative tissues at other times in the plant life cycle. Several ABA-responsive genes have now been isolated<sup>[42]</sup>. It was stated<sup>[43]</sup> that *Diacylglycerol Pyrophosphate* (DGPP) content was increased consecutively to ABA treatment and the application of dioleoyl DGPP was able to trigger the expression of RAB18. In addition, application of dioleoyl DGPP also induced expression of genes<sup>[43]</sup>.

## 2. Conclusion

It can be concluded that abscissic Acid (ABA) hormone effectively stimulated Protein Kinase Gene in Cell Signaling in fruit development in plant. Protein kinase genes expression like ACPK1, OST1, SnRK2, CK2, CK2A4, CK2 $\alpha$ , At5g06760, LT130 and RD29A are new information in this study. Therefore it can be suggested that abscissic Acid (ABA) hormone may be used for stimulating Protein Kinase Gene in Cell Signaling in animal and human growth and development.

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