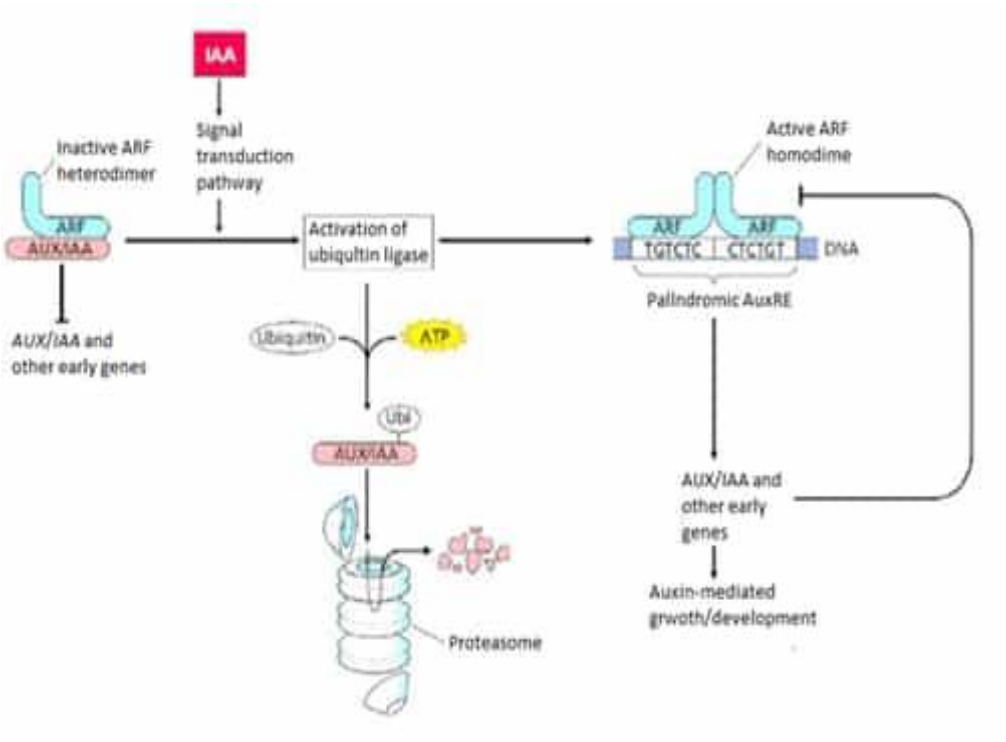
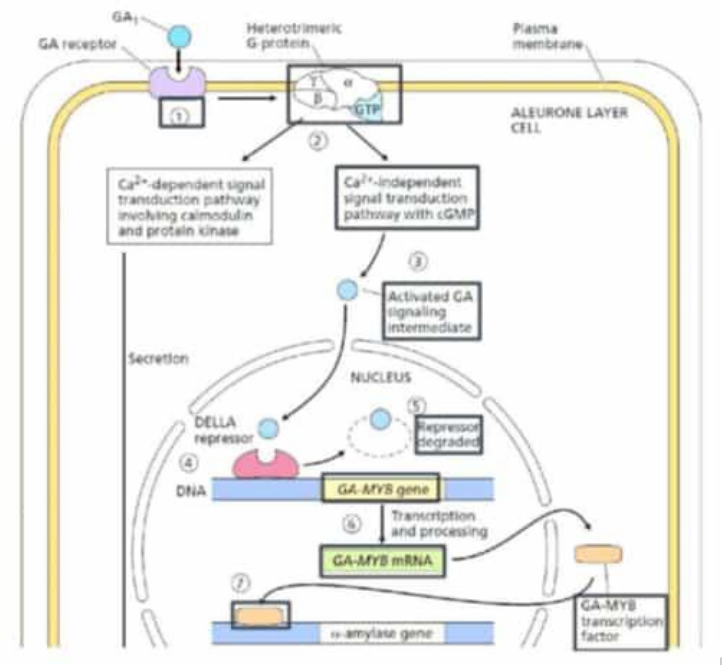
CSIR NET Life Science Unit 6

Basic Mechanism of Hormone Action



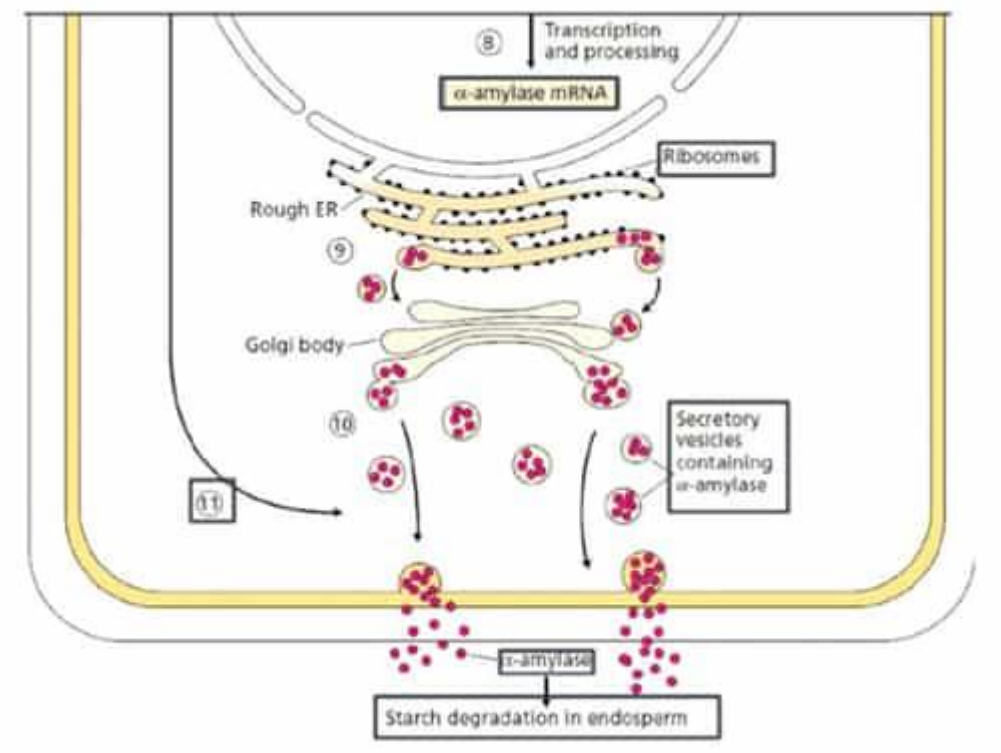
1. In the absence of IAA, the transcription factor, ARF, forms inactive heterodimers with AUX/IAA proteins.
2. Inactive hetero-dimers block the transcription of the early auxin genes. There is no audin response.
3. In the presence of auxin, AUX/IAA proteins are targeted for destruction by an activated ubiquitin
4. The AUX/IAA proteins are tagged with ubiquitin and degraded by the 265 proteasomes.
5. IAA-induced degradation of the AUX/IAA proteins allows active ARF homodimers to form.
6. The active ARF homodimers bind to palindromic AuxREs in the promoters of the early genes, activating transcription.
7. Transcription of the early genes initiates the auxin response.
8. The stimulation of AUX./IAA genes introduces a negative feedback loop.

GA MODE OF ACTION



STEPS

1. GA_1 from the embryo first binds to a cell surface receptor.
2. The cell surface GA receptor complex interacts with a heterotrimeric G-protein, initiating two separate signal transduction chains.
3. A calcium-independent pathway, involving cGMP, results in the activation of a signaling intermediate.
4. The activated signaling intermediate binds to DELLA repressor proteins in the nucleus.
5. The DELLA repressors are degraded when bound to the GA signal.
6. The inactivation of the DELLA repressors allows the expression of the MYB gene, as well as other genes, to proceed through transcription, processing, and translation.



7. The newly synthesized MYB protein then enters the nucleus and binds to the promoter genes for a-amylase and other hydrolytic enzymes.

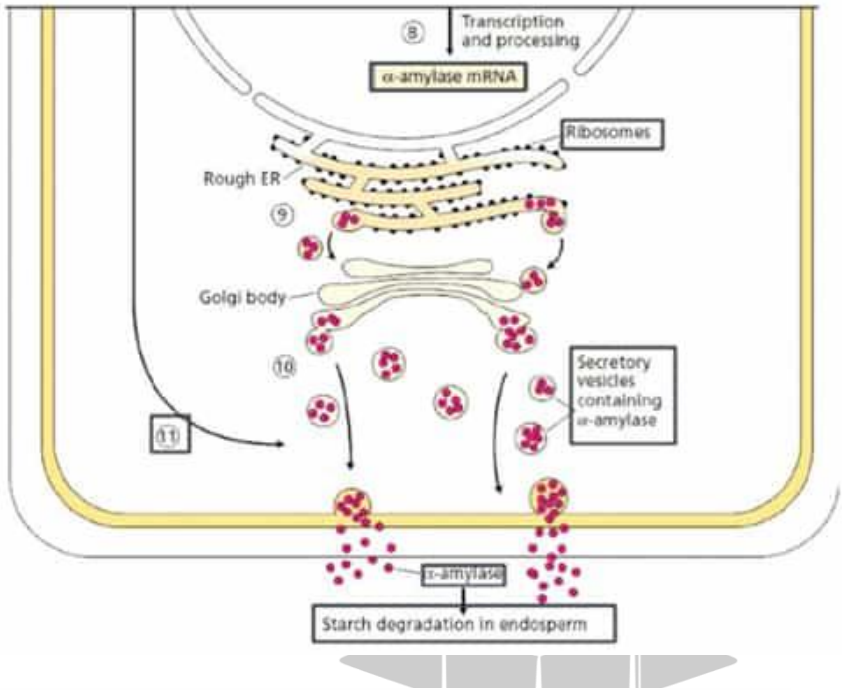
8. Transcription of a-amylase and other hydraulic genes is activated.

9. a-Amylase and other hydrolysis are synthesized on the rough ER.

10. Proteins are secreted via the Golgi.

11. The secretory pathway requires GA stimulation via a calcium-calmodulin-dependent signal transduction pathway.

CYTOKININ MODE OF ACTION



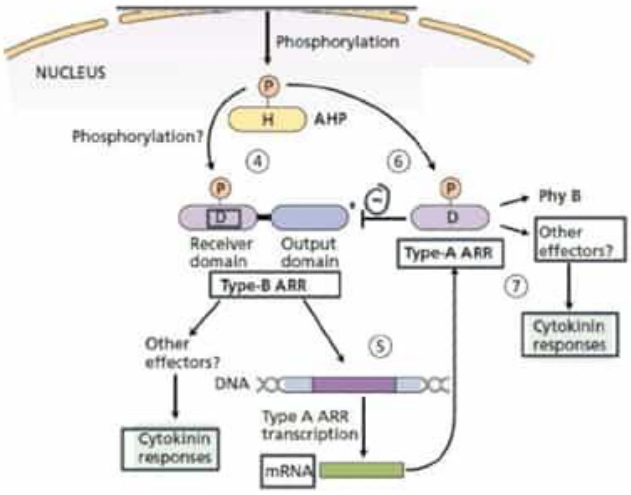


FIGURE : Model of cytokinin signaling. The near future should see significant refinement of this model, the tools are now in hand to analyze the interactions among these elements.

STEPS

1. Cytokinin binds to CRE 1, which is likely to occur as a dimer, Cytokinin binds to an extracellular portion of CRE1 called the CHASE domain. Two other hybrid sensor kinases (AHK2 and AHK3) containing a CHASE domain are also likely to act as cytokinin receptors in *Arabidoposis.*

2. Cytokinin binding to these receptors activates their histidine kinase activity. The phosphate is transferred to an aspartate residue (D) on the fused receiver domains.

3. The phosphate is then transferred to a conserved histidine present in an AHP protein.

4. Phosphorylation causes the AHP protein to move into the nucleus, where it transfers the phosphate to an aspartate residue located within the receiver domain of a type B ARR.

5. The Phosphorylation of the type-B ARR activates the output domain to induce transcription of genes encoding type-A ARRs.

6. The type-A ARRs are likely also to be phosphorylated by the AHP proteins.

7. The phosphorylated type-A ARRs interact with various effectors to mediate the changes in cell function appropriate to cytokinin (indicated in the model as *cytokinin responses*).

ABA MODE OF ACTION

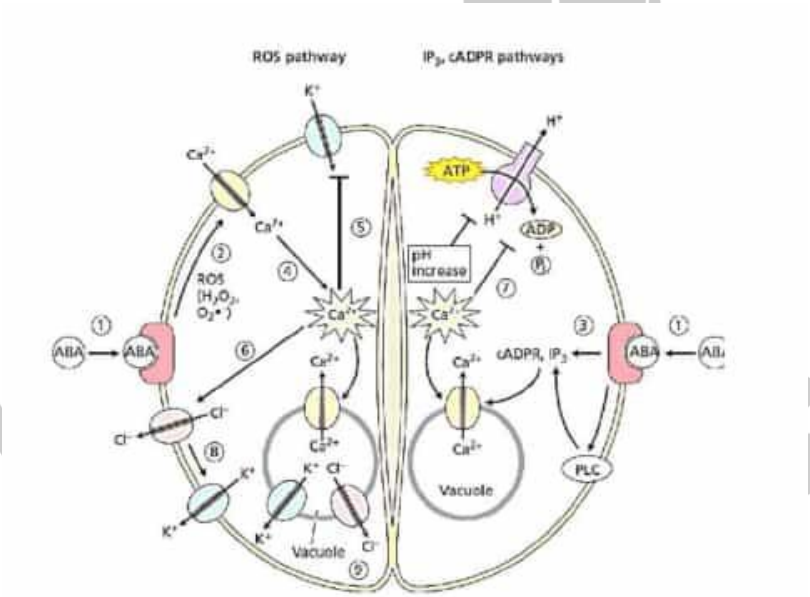


FIGURE Simplified model for ABA signaling in stomatal guard cells. The net effect is the loss of potassium and its anion (Cl^- or $malate^{2-}$) from the cell. (R = receptor; ROS = reactive oxygen species; cADPR = cyclic ADP-ribose; G-protein = GTP-binding protein; PLC = phospholipase C.)

STEPS

1. ABA binds to its receptors.

2. ABA binding induces the formation of reactive oxygen species, which activate plasma membrane Ca^{2+} channels.

3. ABA increases the levels of cyclic ADP-ribose and IP_3. Which activates additional calcium channels on the tonoplast.

4. The influx of calcium initiates intracellular calcium oscillations and promotes the further release of calcium from vacuoles.
5. The rise in intracellular calcium blocks K¢$_{im}$ channels
6. The rise in intracellular calcium promotes the opening of Cl^-_{out} (anion) channels on the plasma membrane, causing membrane depolarization.
7. The plasma membrane proton pump is inhibited by the ABA-Induced increase in cytosolic calcium and a rise in intracellular pH, further depolarizing the membrane.
8. Membrane depolarization activates K^+_{out} channels.
9. K^+ and anions to be released across the plasma membrane are first released from vacuoles into the cytosol.