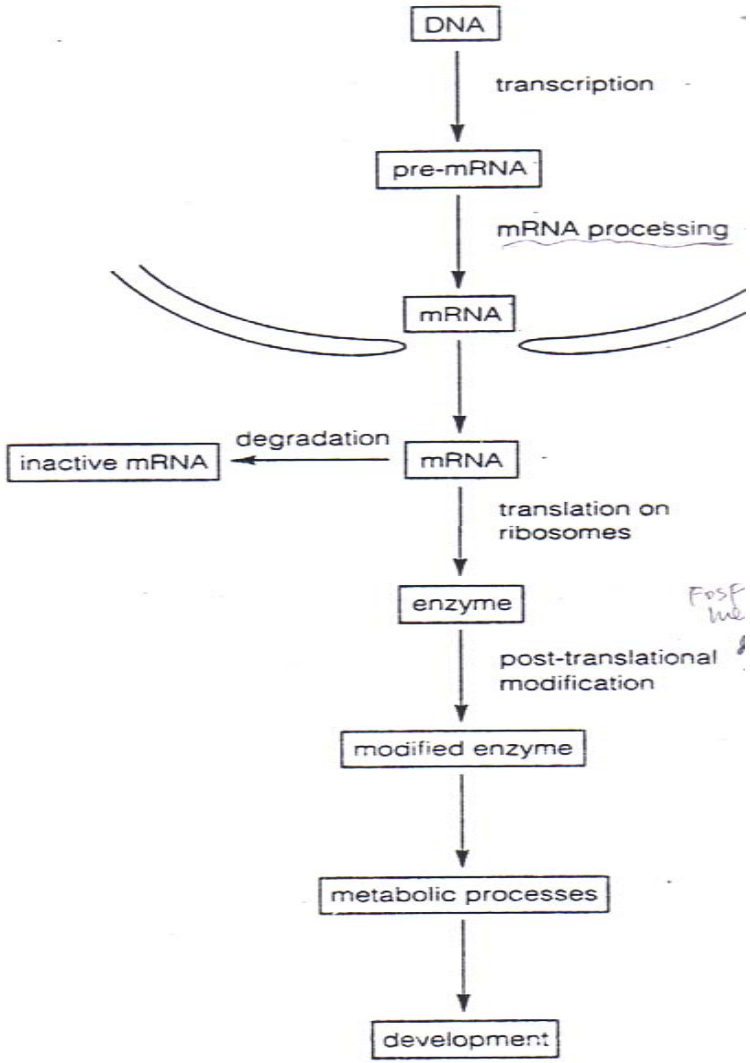
# MODE OF ACTION and MECHANISM OF PLANT HORMONES

**ANY FITRIANI**
**131964921**

# POSSIBLE SITE OF HORMONAL CONTROL



# NUCLEID ACID BIOSYNTHESIS

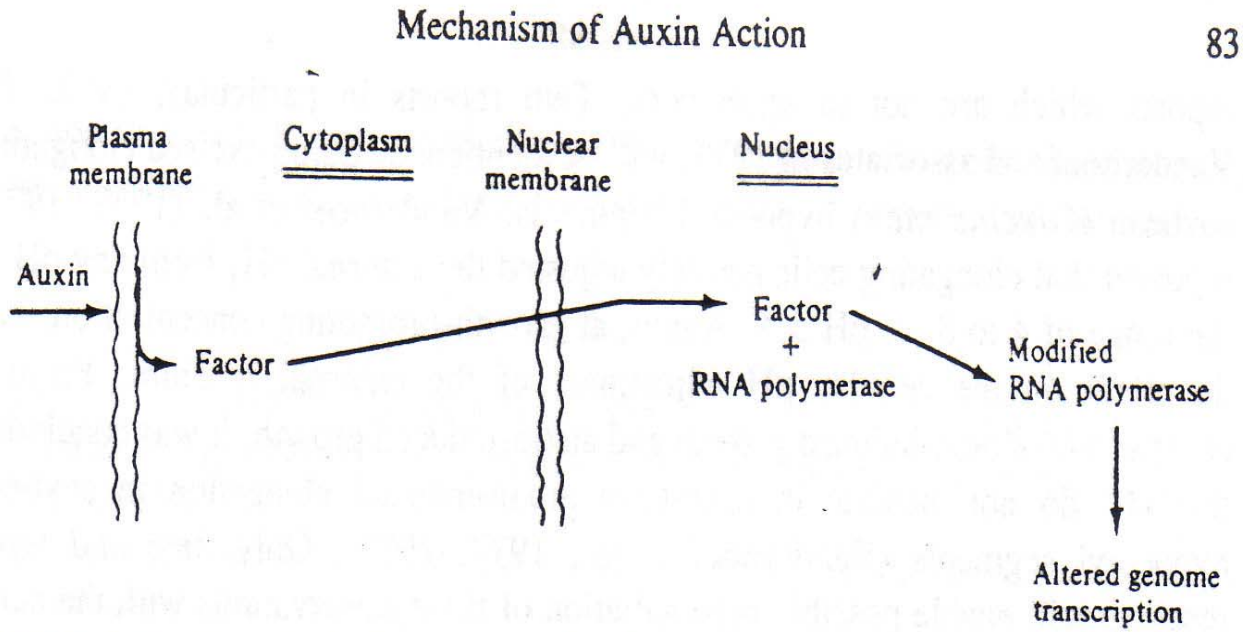



FIGURE 2.42. Hypothesis for auxin-regulated nucleic acid biosynthesis. It is proposed that auxin interacts with a factor within the plasma membrane. The factor is then transported through the nuclear membrane into the nucleus where it regulates the activity of RNA polymerase. (Redrawn, with permission, from Hardin et al., 1972.)

# HORMONE TRANSDUCTION

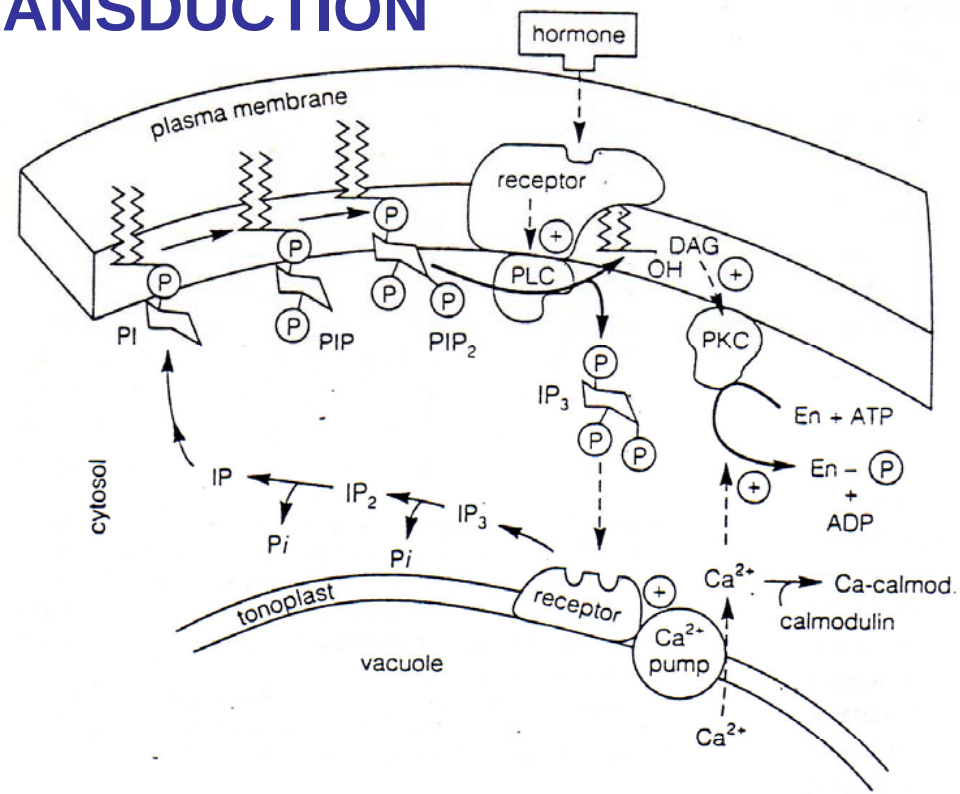



**Figure 17-2** Model for initial hormone transduction at the plasma membrane. Binding of a hormone to its receptor causes activation (+) of nearby phospholipase c (PLC). PLC hydrolyzes a membrane lipid, phosphatidylinositol-4,5-bisphosphate ($PIP_2$) to release inositol-1,4,5-trisphosphate ($IP_3$) and a diacylglycerol (DAG). $IP_3$ moves to the tonoplast in plant cells, where it combines with a receptor that activates (+) a $Ca^{2+}$ pump or transporter that moves $Ca^{2+}$ from the vacuole to the cytosol. DAG, which remains membrane-bound, activates protein kinase c (PKC). PKC is also activated by $Ca^{2+}$ released from the vacuole, so various enzymes become phosphorylated by PKC. Calcium also activates other protein kinases and other enzymes, when free or bound with calmodulin. $IP_3$ loses phosphates by hydrolysis to form $IP_2$ and IP, which is then converted back to phosphatidylinositol (PI) and other phosphoinositide lipids (PIP and $PIP_2$) in the plasma membrane. (Modified from various sources.)

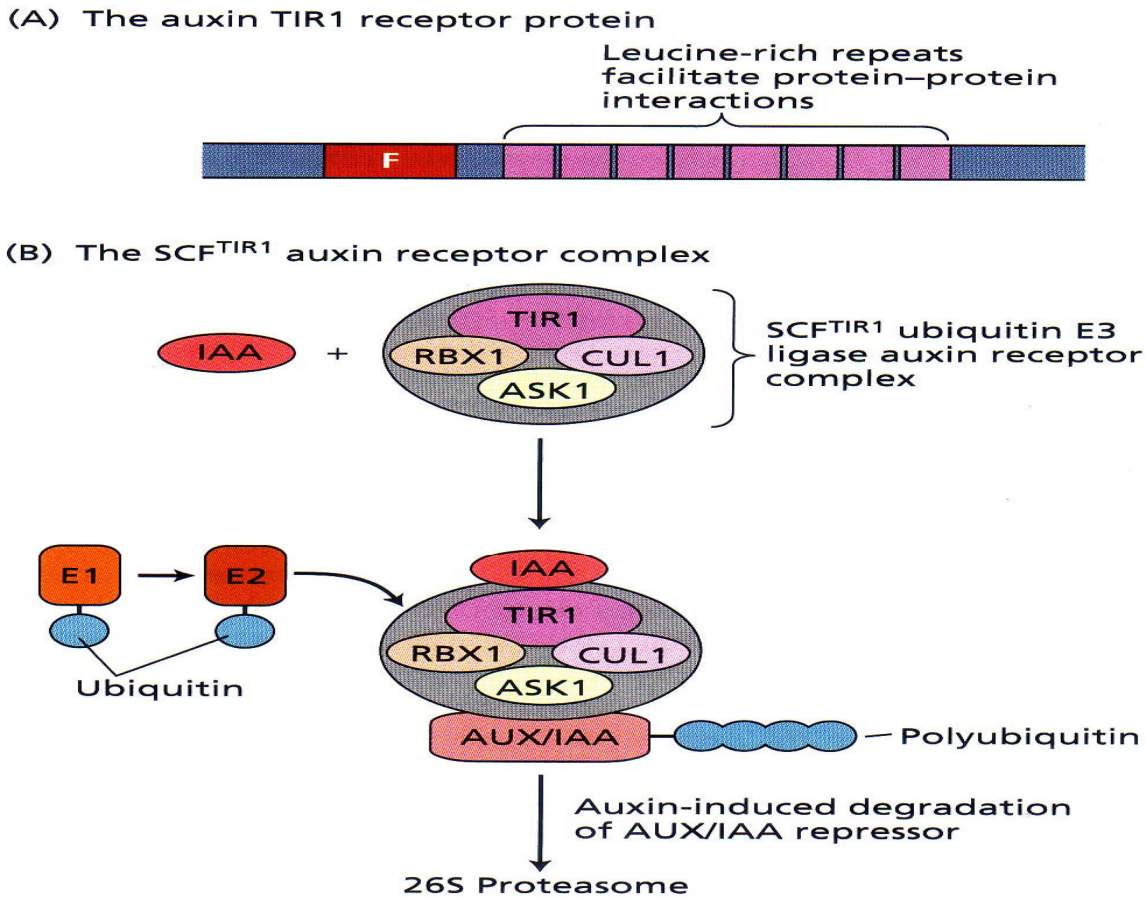



**FIGURE 19.40** The auxin receptor. (A) The auxin receptor TIR1 has an F-box motif and leucine-rich repeat sequences that facilitate protein-protein interactions. (B) TIR1 functions as a subunit of the specific ubiquitin E3 ligase complex SCF$^{TIR1}$. When auxin binds to SCF$^{TIR1}$ it activates it, resulting in the ubiquitination and proteolytic degradation of AUX/IAA repressor proteins.

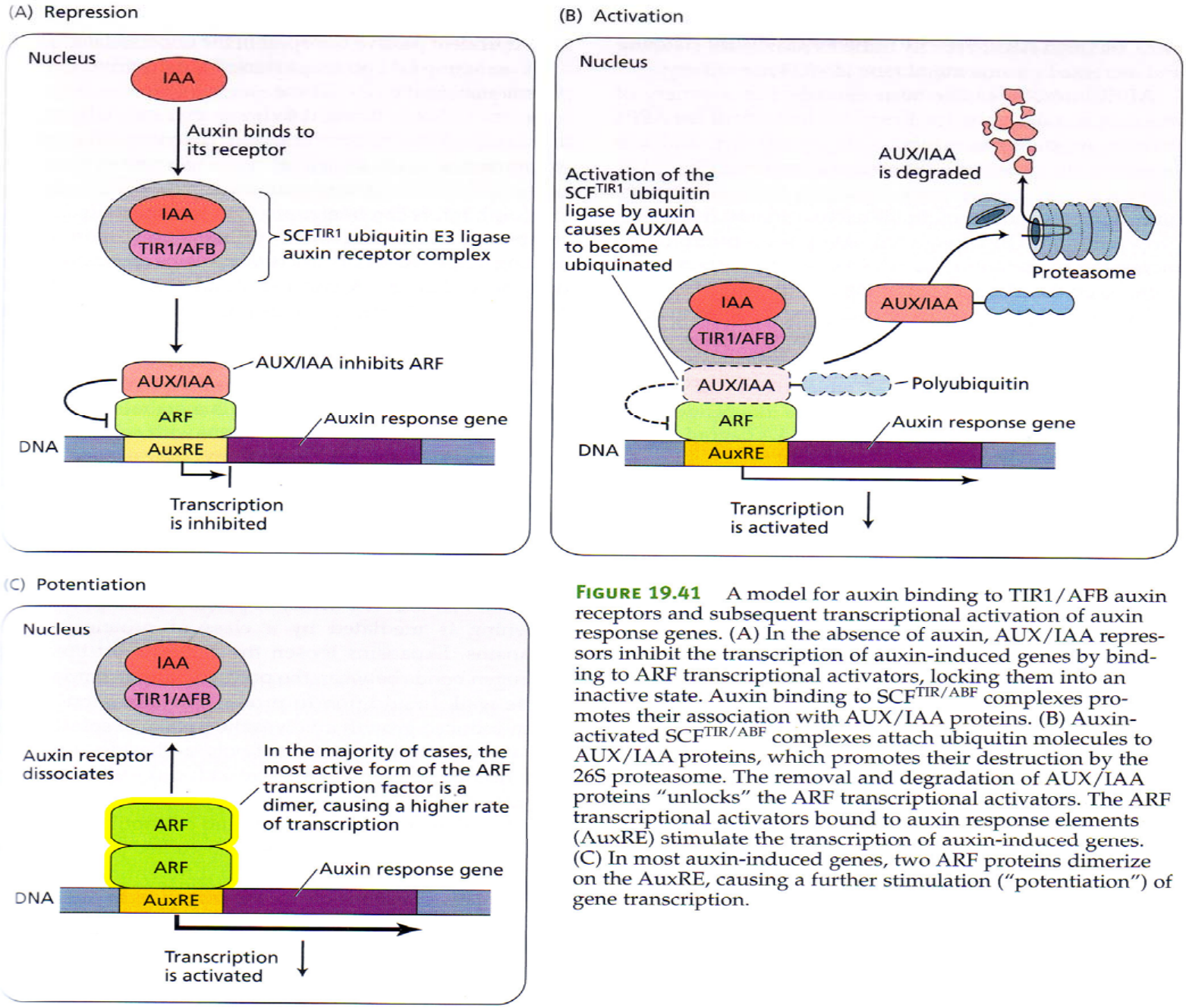



**FIGURE 19.41** A model for auxin binding to TIR1/AFB auxin receptors and subsequent transcriptional activation of auxin response genes. (A) In the absence of auxin, AUX/IAA repressors inhibit the transcription of auxin-induced genes by binding to ARF transcriptional activators, locking them into an inactive state. Auxin binding to $SCF^{TIR/ABF}$ complexes promotes their association with AUX/IAA proteins. (B) Auxin-activated $SCF^{TIR/ABF}$ complexes attach ubiquitin molecules to AUX/IAA proteins, which promotes their destruction by the 26S proteasome. The removal and degradation of AUX/IAA proteins "unlocks" the ARF transcriptional activators. The ARF transcriptional activators bound to auxin response elements (AuxRE) stimulate the transcription of auxin-induced genes. (C) In most auxin-induced genes, two ARF proteins dimerize on the AuxRE, causing a further stimulation ("potentiation") of gene transcription.

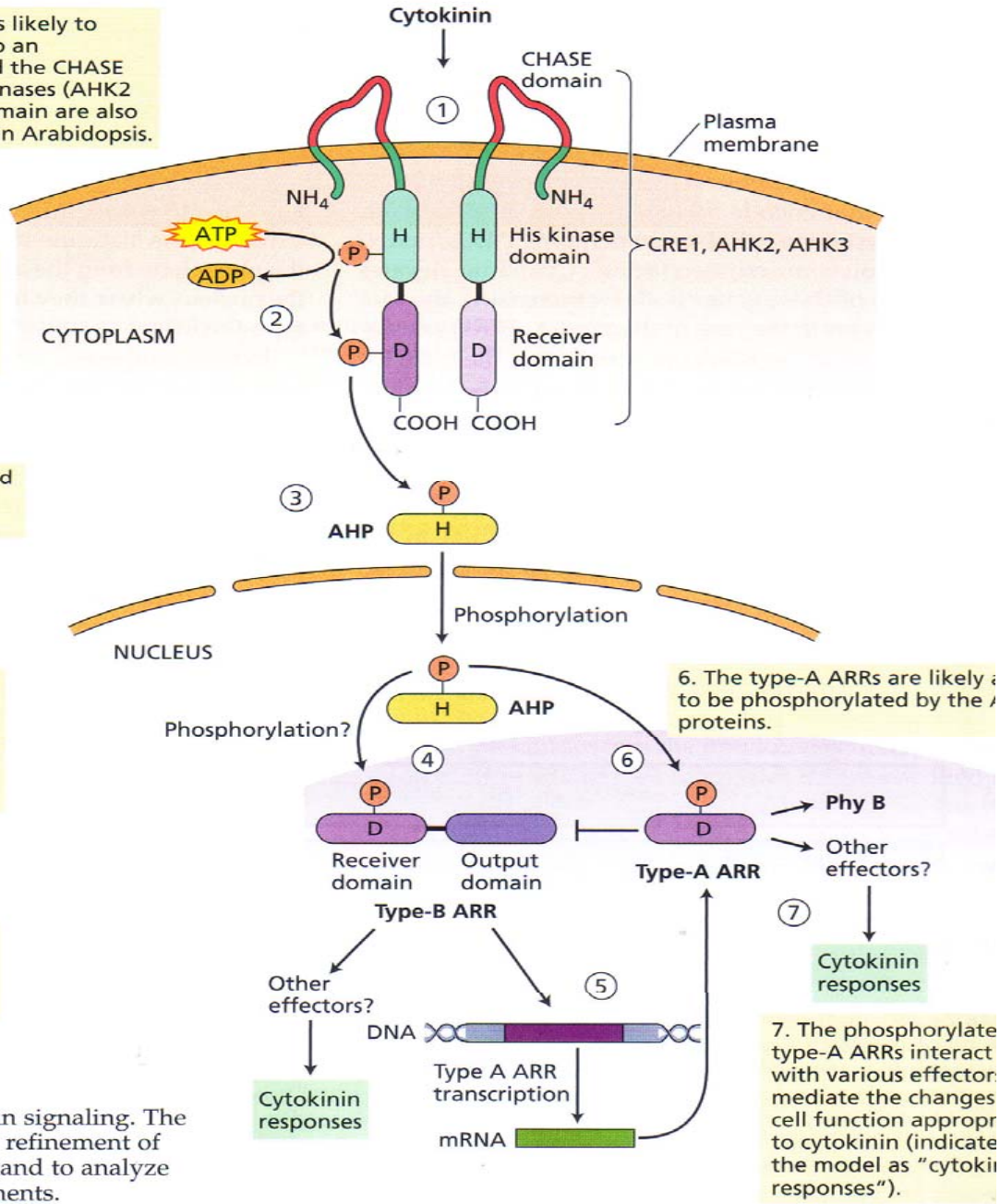
1. Cytokinin binds to CRE1, which is likely to occur as a dimer. Cytokinin binds to an extracellular portion of CRE1 called the CHASE domain.Two other hybrid sensor kinases (AHK2 and AHK3) containing a CHASE domain are also likely to act as cytokinin receptors in Arabidopsis.

2. Cytokinin binding to these receptors activates their histidine kinase activity. The phosphate is transferred to an asparate 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 asparate 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 a to be phosphorylated by the A proteins.

7. The phosphorylate type-A ARRs interact with various effector mediate the changes cell function appropr to cytokinin (indicate the model as "cytoki responses").

**FIGURE 21.31** 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.

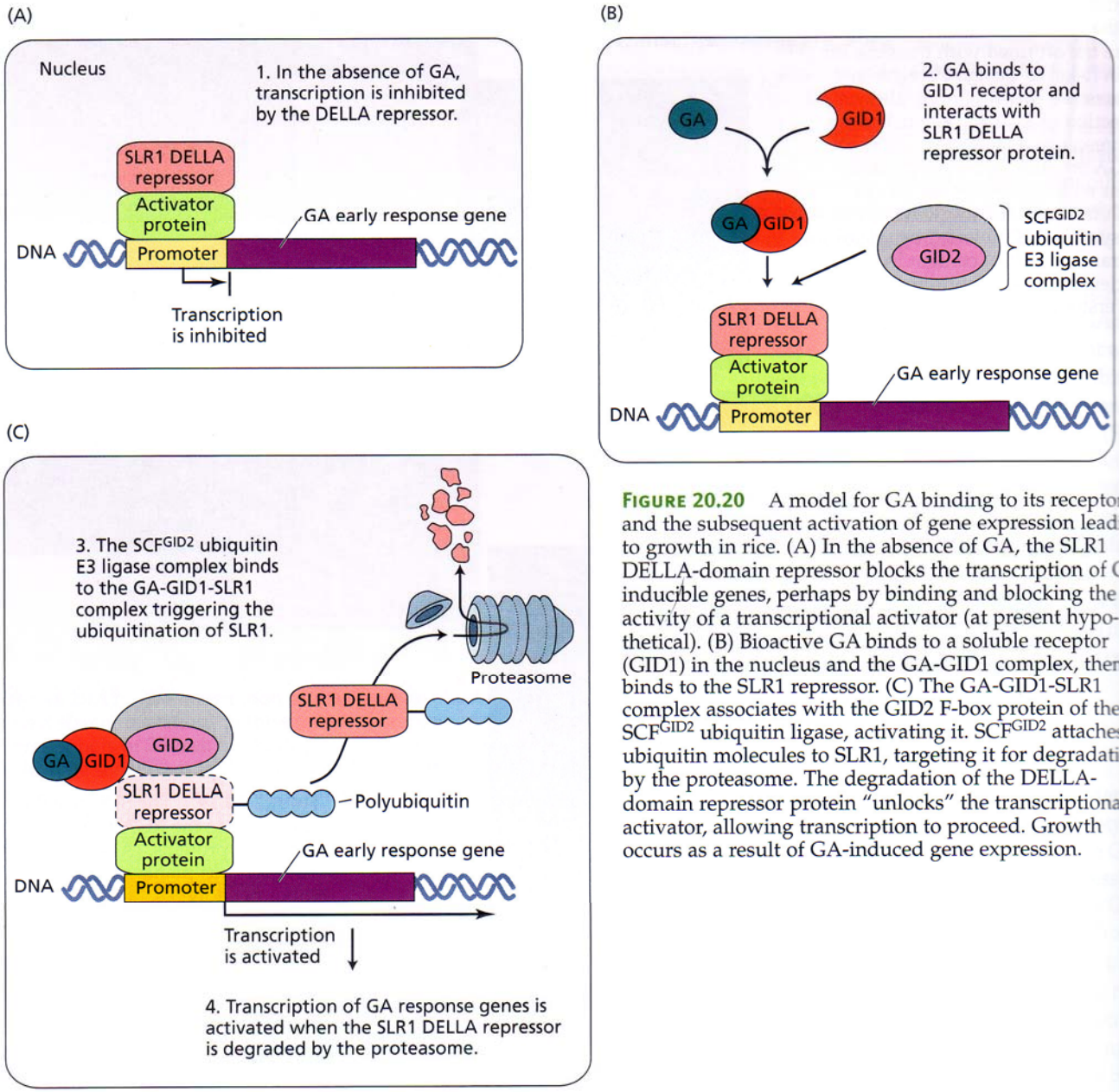
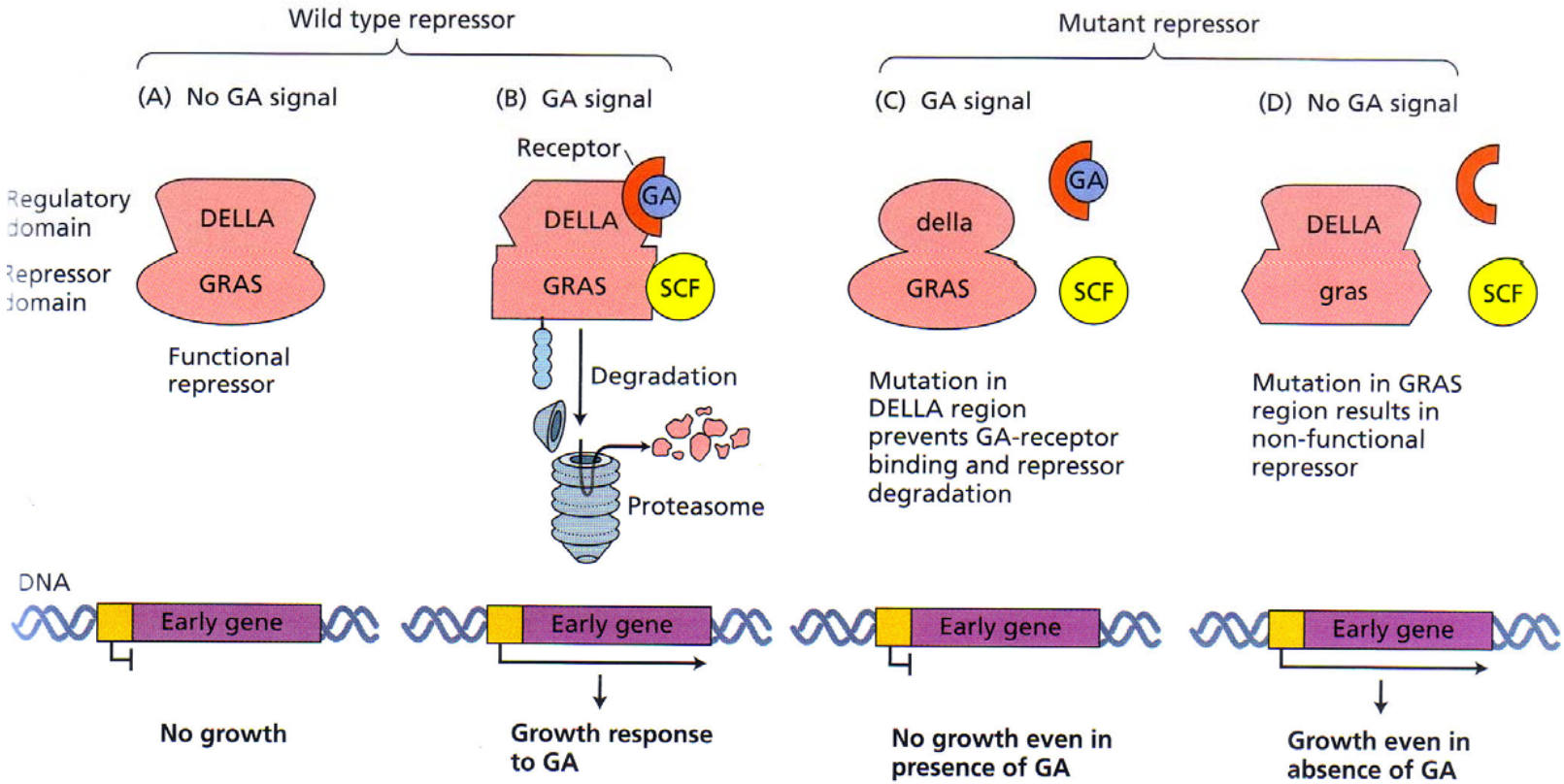



**FIGURE 20.20** A model for GA binding to its receptor and the subsequent activation of gene expression leading to growth in rice. (A) In the absence of GA, the SLR1 DELLA-domain repressor blocks the transcription of GA-inducible genes, perhaps by binding and blocking the activity of a transcriptional activator (at present hypothetical). (B) Bioactive GA binds to a soluble receptor (GID1) in the nucleus and the GA-GID1 complex, then binds to the SLR1 repressor. (C) The GA-GID1-SLR1 complex associates with the GID2 F-box protein of the $SCF^{GID2}$ ubiquitin ligase, activating it. $SCF^{GID2}$ attaches ubiquitin molecules to SLR1, targeting it for degradation by the proteasome. The degradation of the DELLA-domain repressor protein "unlocks" the transcriptional activator, allowing transcription to proceed. Growth occurs as a result of GA-induced gene expression.




**IGURE 20.21** The current model of GA signaling during rowth involves interactions between the GA receptor, a biquitin ligase SCF complex, and a DELLA-domain repres- or protein. The GA repressor (GAI/RGA/SLR1/SLN1) pro- eins contain two domains: the regulatory domain (DELLA) nd the repressor domain (GRAS). (A) The GRAS domain of he repressor protein is active in the absence of GA, blocking arly GA-induced gene expression and giving a dwarf eedling. (B) GA bound to its receptor binds to the repressor rotein and facilitates its association with the SCF ubiquitin ligase complex. The repressor protein is thus targeted for ubiquitination and degradation by the 26S proteasome. The destruction of the repressor proteins permits early GA-induced gene expression and seedling growth. (C) A mutation in the DELLA regulatory domain prevents it from binding the GA-receptor complex. Consequently the repressor cannot be degraded, and the mutant is a GA-insensitive dwarf. (D) Mutation in the GRAS domain gives a nonfunctional repressor, so the seedling can grow very tall (slender) even in the absence of GA.

1. ABA and a variety of transcription factors are produced in response to developmental or environmental signals.

2. Phospholipase D (PLD)-dependent signaling, regulation by microRNA (miRNA), and activation of many kinases are implicated in the activation of transcription factors by ABA. The promoters of ABA-regulated genes have different combinations of recognition sites (MYBR, MYCR, etc.) that can be bound by various members of the corresponding transcription factor families. Multiple family members for each class of the transcription factors shown participate in ABA signaling.

3. In addition to forming homo- and heterodimers within families, some of these factors interact with one another and additional components of the transcription machinery. The specific combinations determine the extent to which a given gene is activated or repressed.

4. Some of the transcription factor genes are cross- and autoregulated, in some cases enhancing the ABA response by positive feedback.

5. In the absence of ABA, these ABA-Insensitive (ABI) transcription factors are degraded via the proteasome.

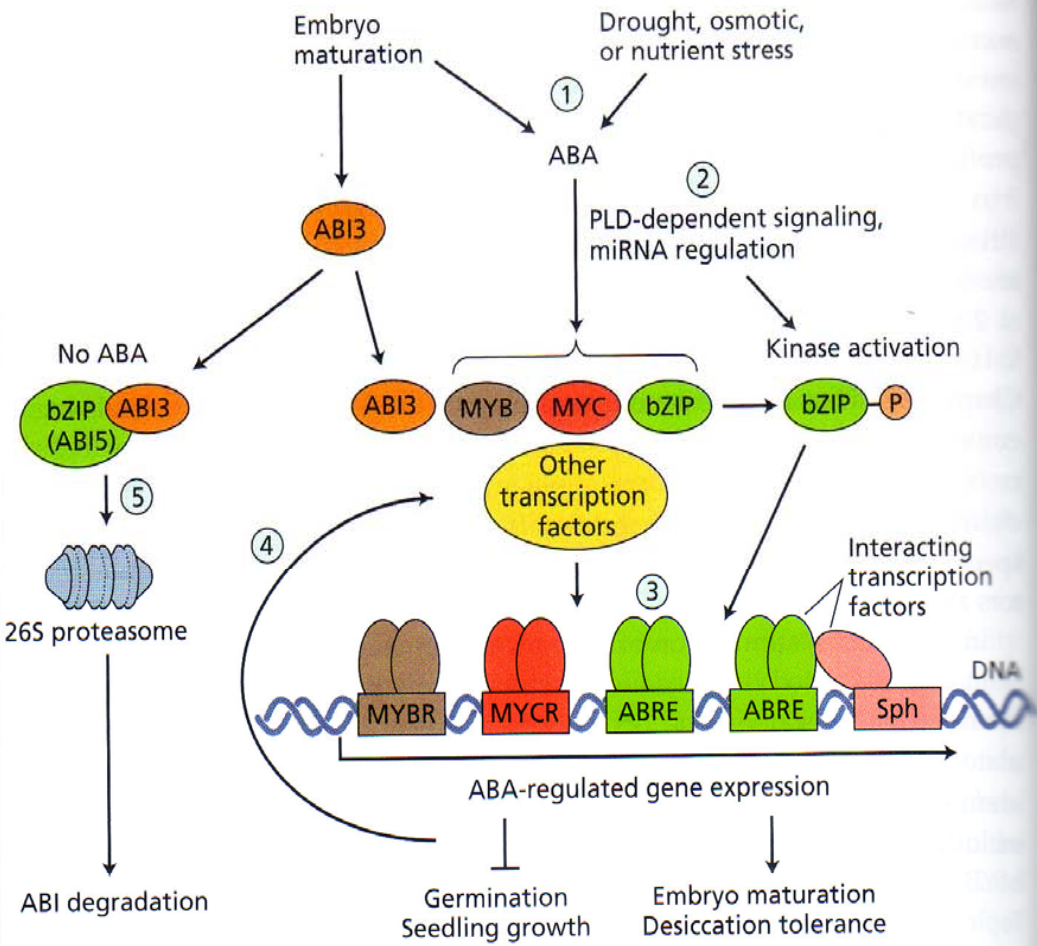
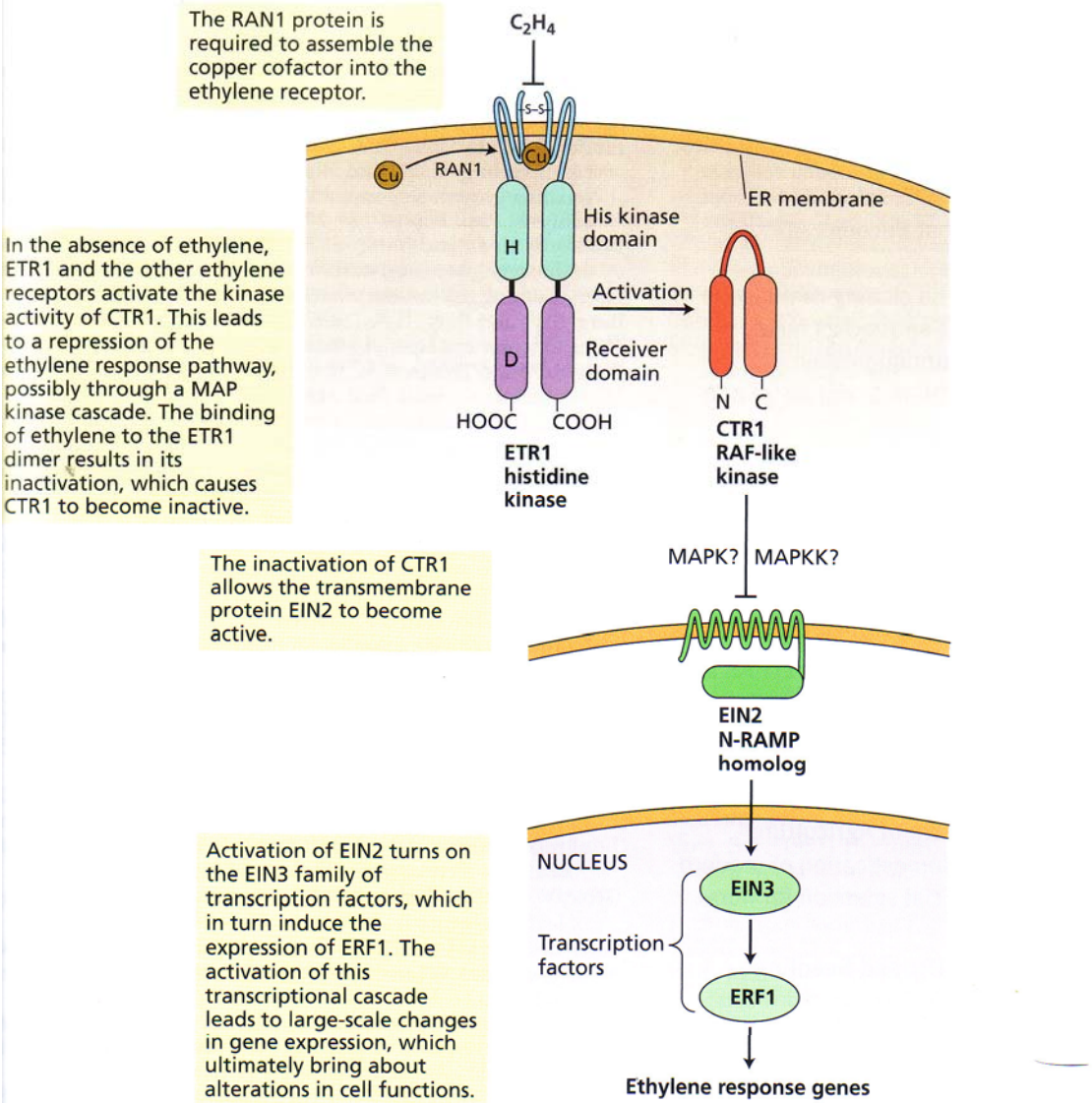



**FIGURE 23.15** Regulatory mechanisms and transcription factors that mediate ABA-regulated gene expression.




**IGURE 22.16** Model of ethylene signaling in Arabidopsis. thylene binds to the ETR1 receptor, which is an integral embrane protein of the endoplasmic reticulum membrane. Multiple isoforms of ethylene receptors may be present in a ell; only ETR1 is shown for simplicity. The receptor is a dimer, held together by disulfide bonds. Ethylene binds within the transmembrane domain, through a copper cofactor, which is assembled into the ethylene receptors by the RAN1 protein.

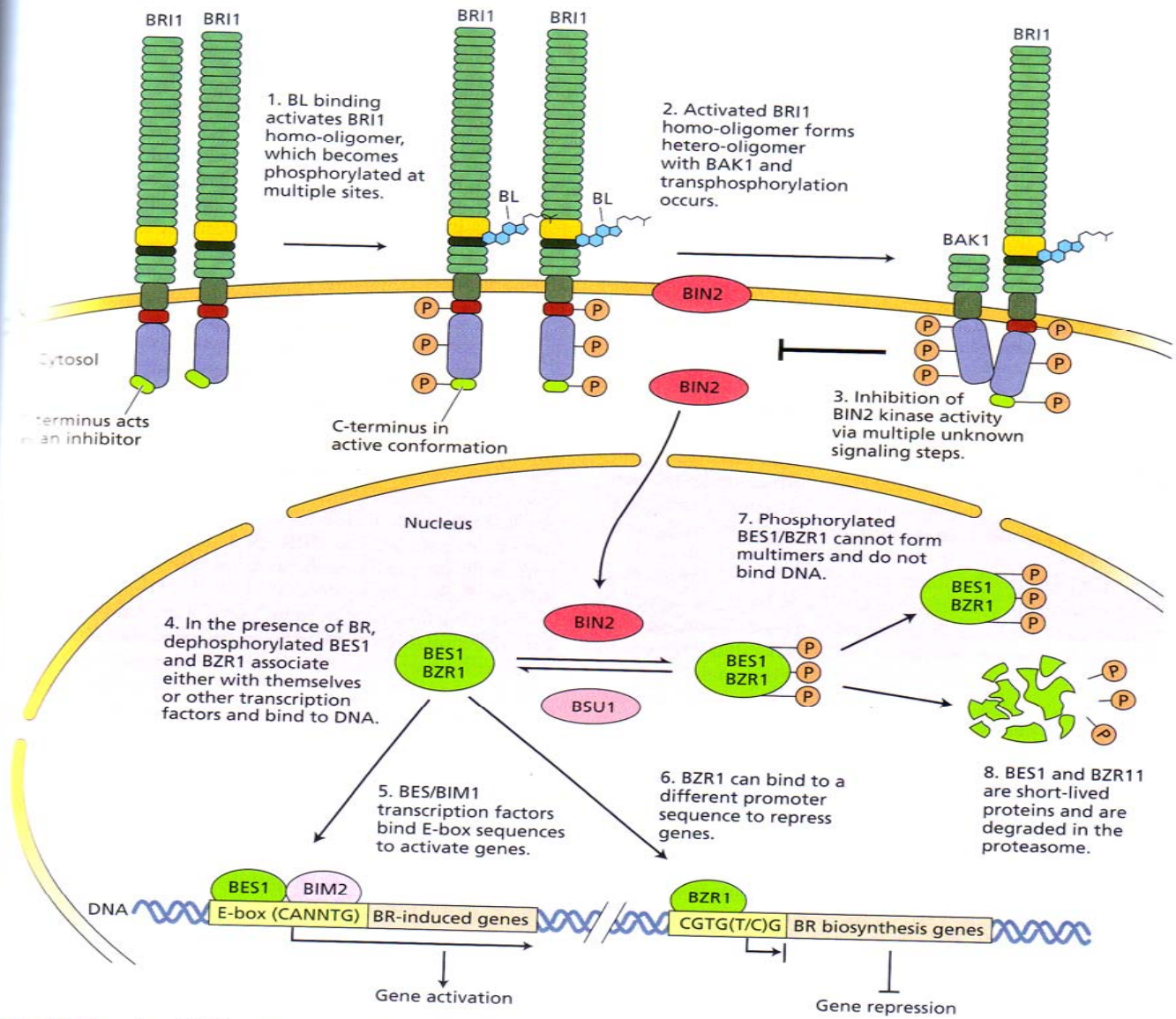



FIGURE 24.21 A model for BR signaling. Signal perception occurs at the cell surface.


THANK YOU