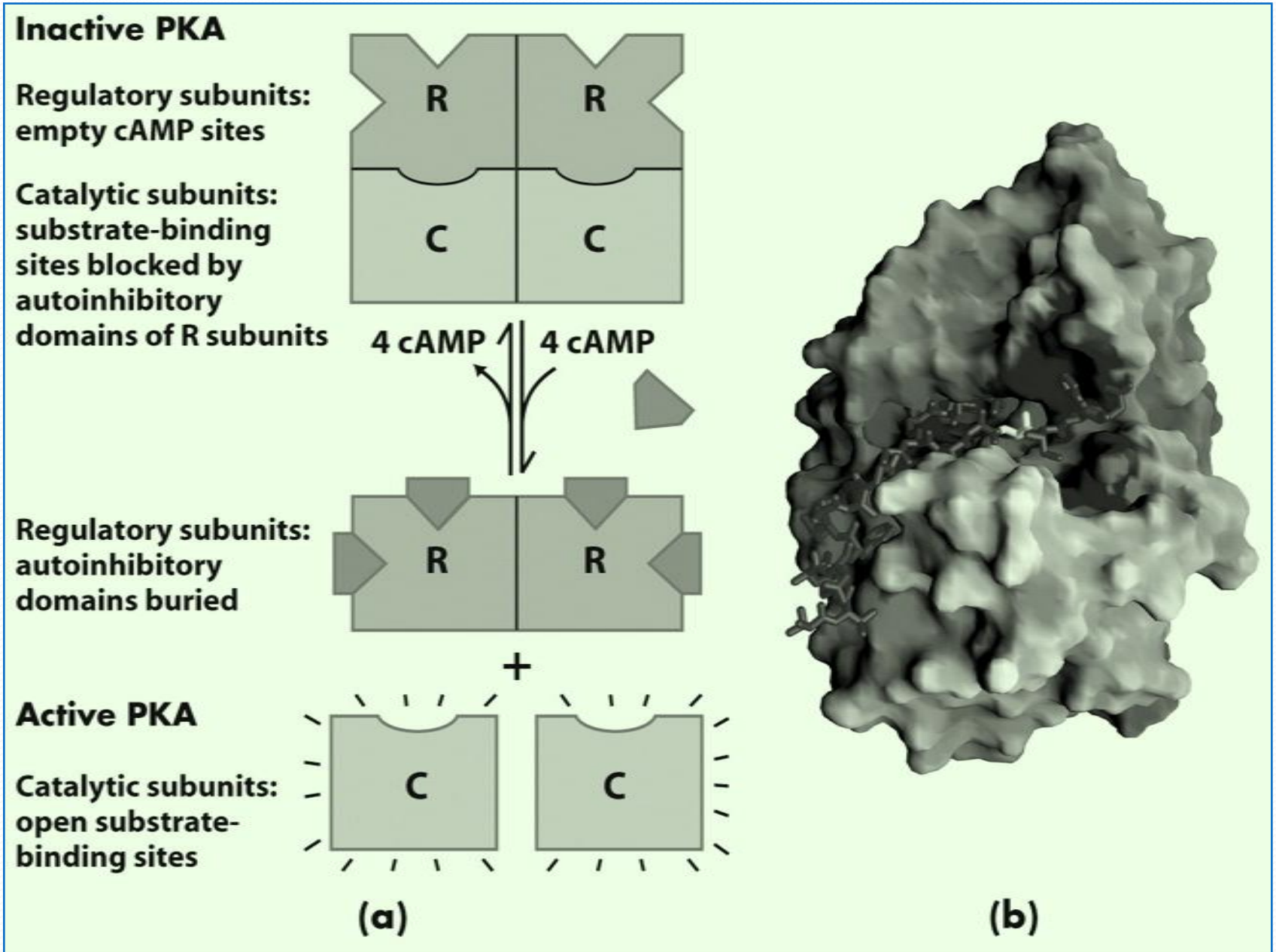
Activation of PKA by cAMP

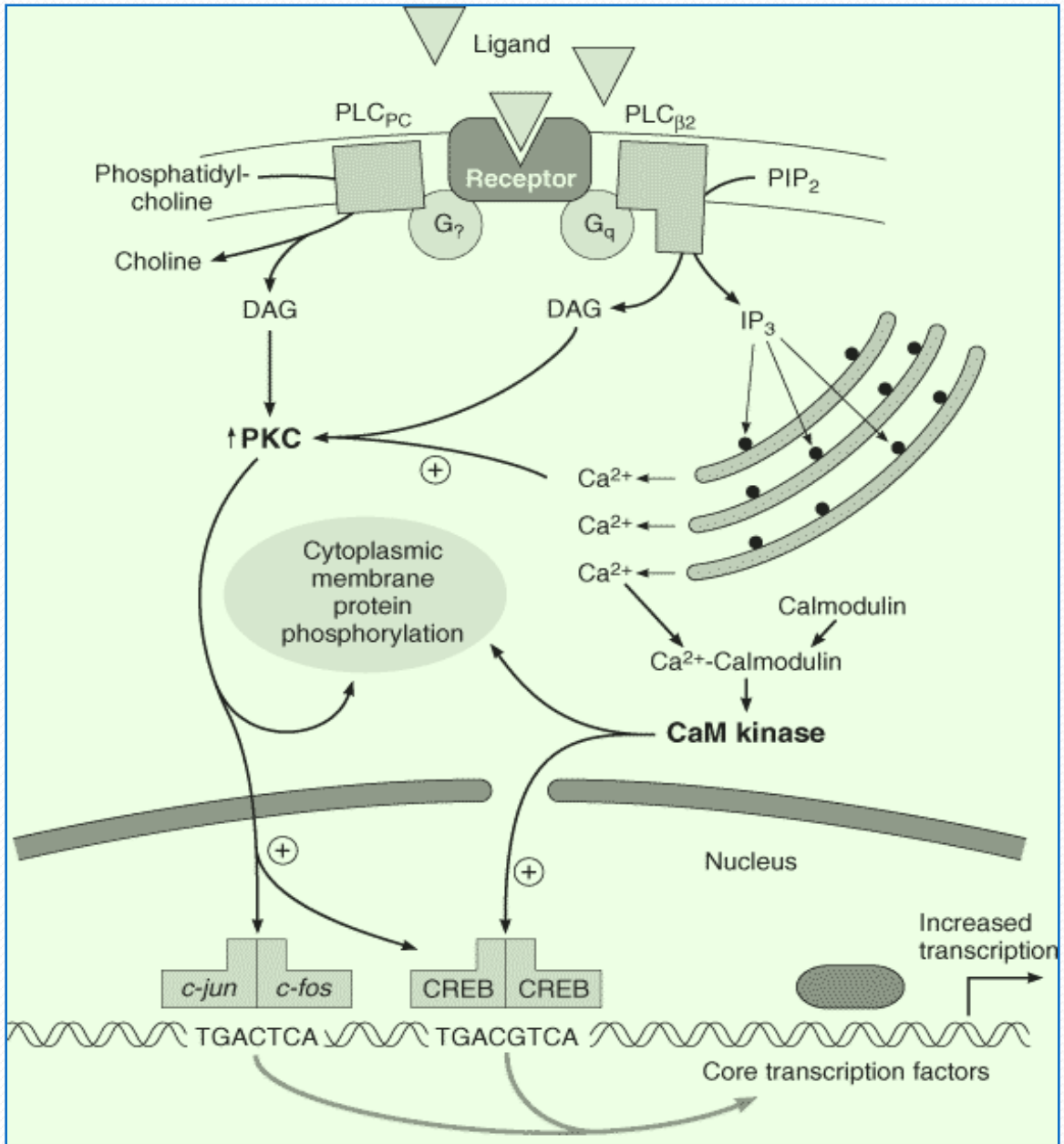


G protein Transducers (2)

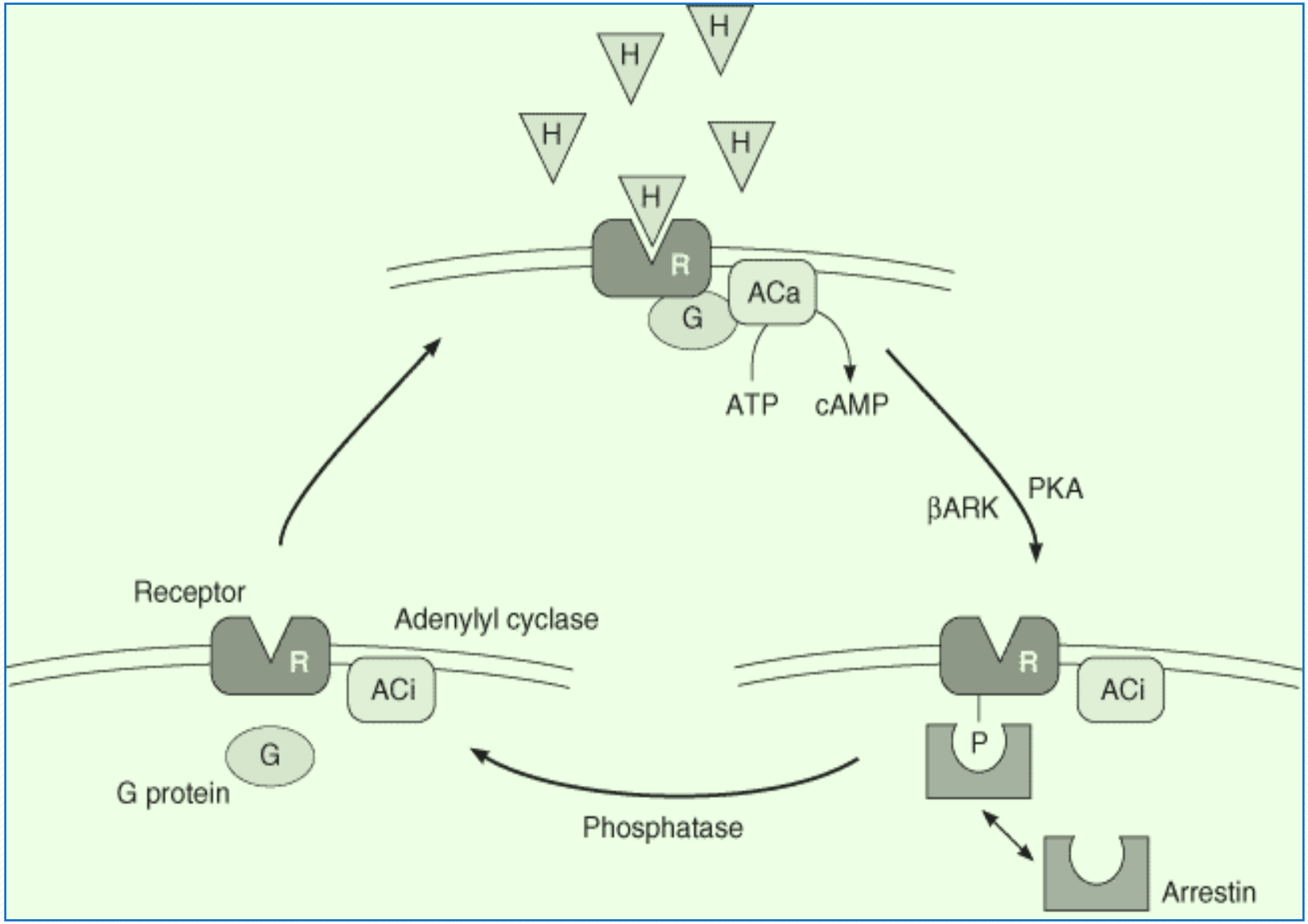
G protein disorders

- **Cholera toxin:** Covalent modification inhibits GTPase →↑CAMP.
- **Pertussis toxin:** covalent modification of Gi → inactivation.

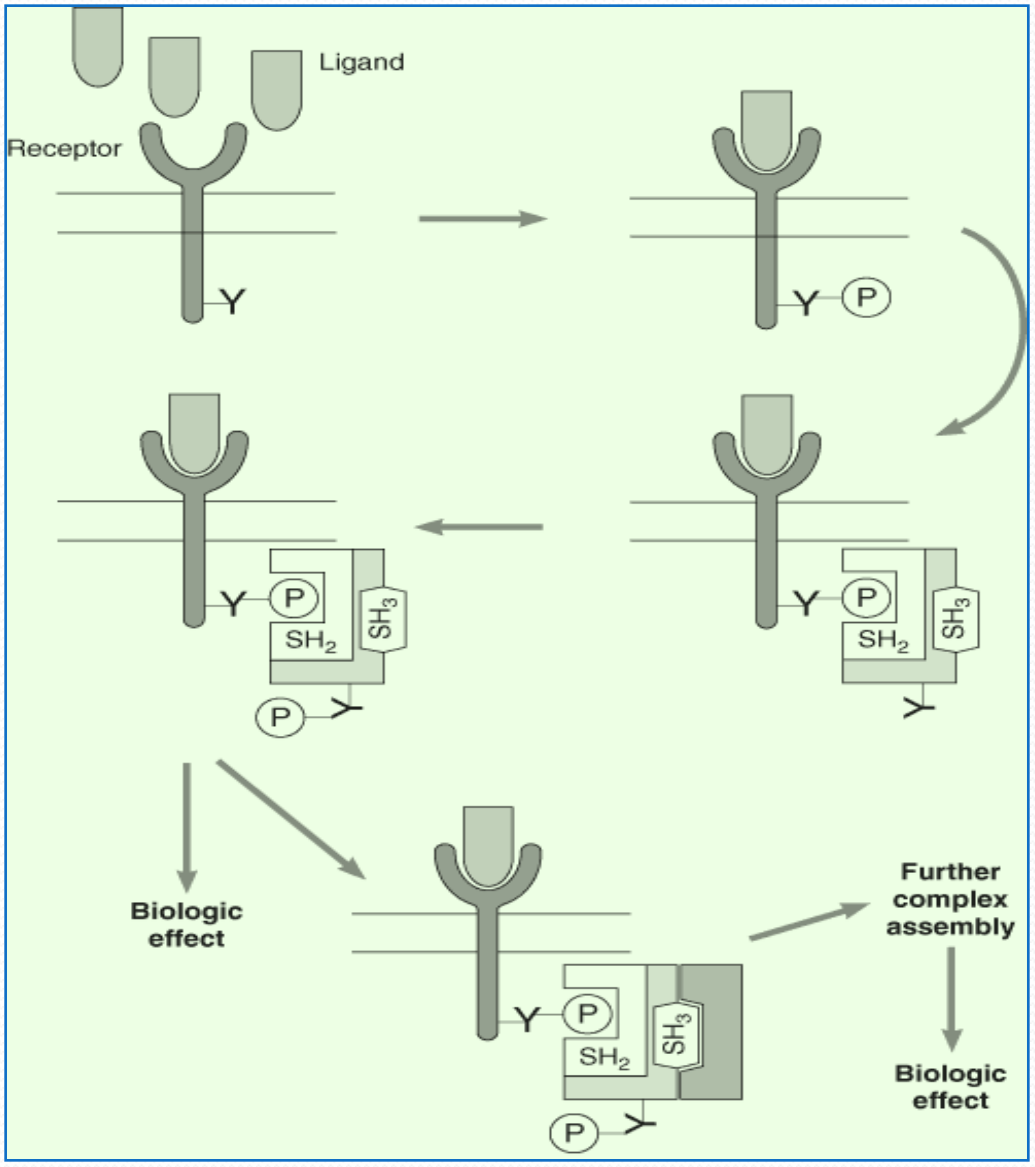
Signal Transduction-PLC



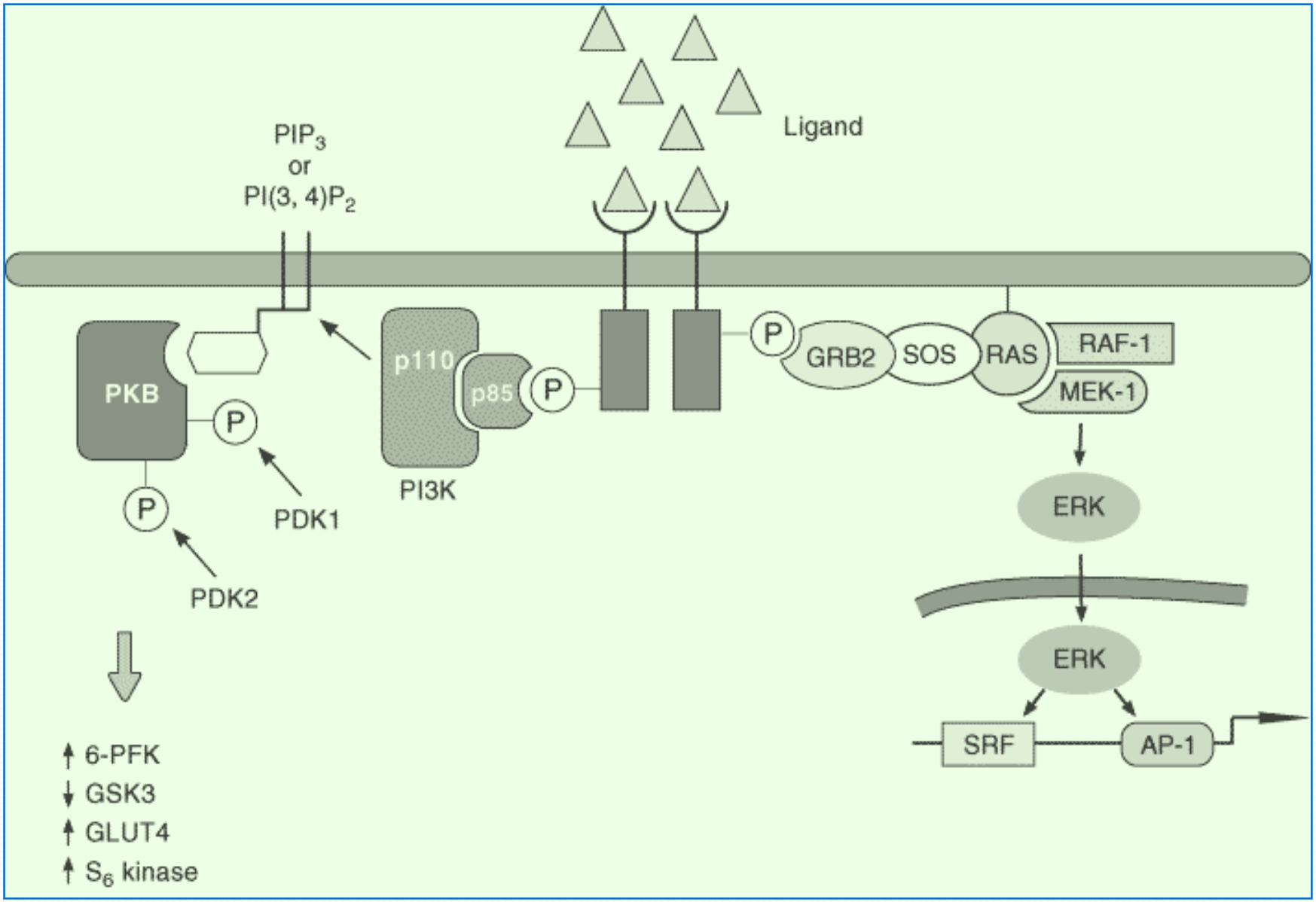
Desensitization of Ligand-Receptor Complex



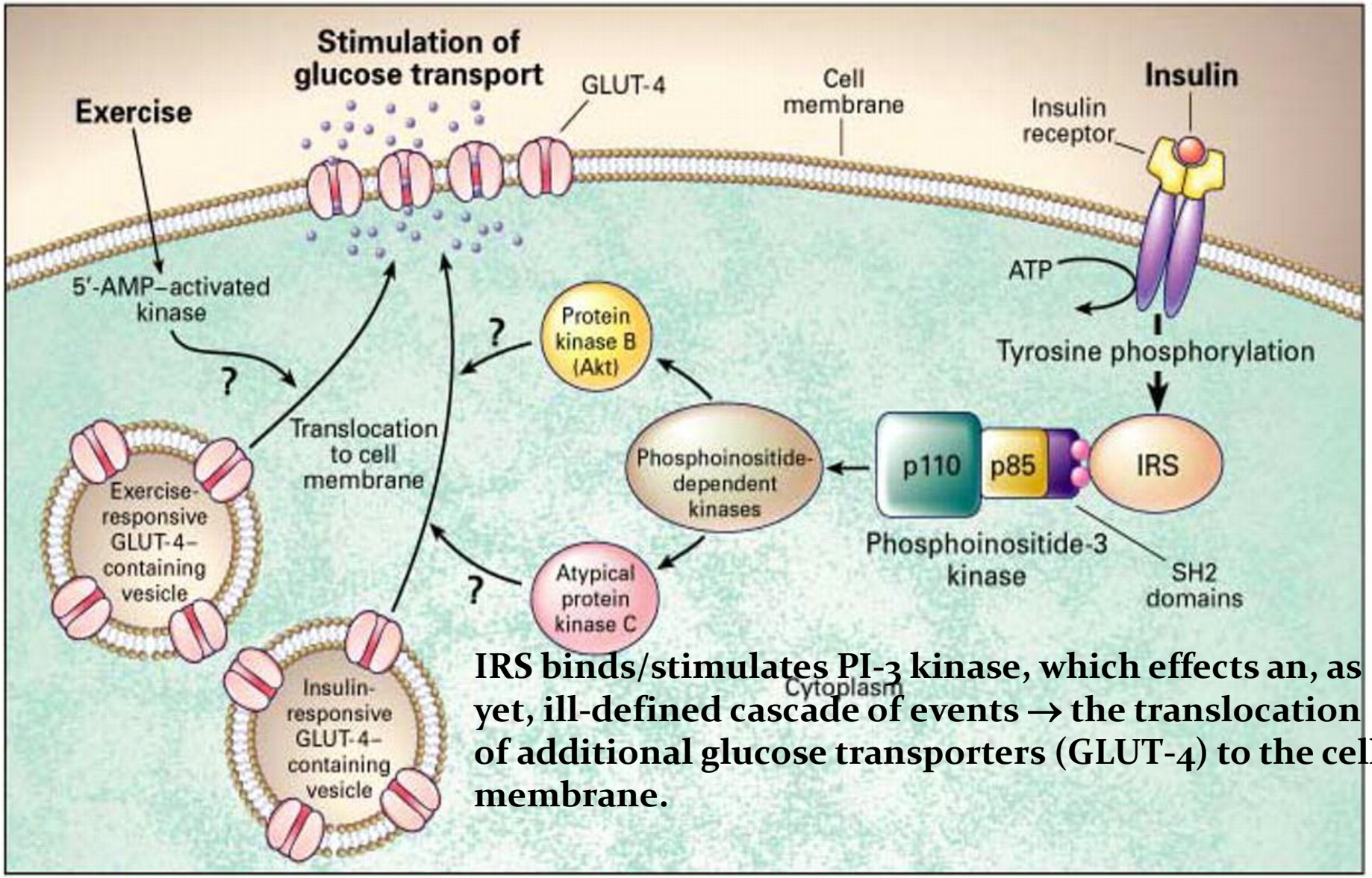
Signaling by Tyrosine Kinase-Containing Growth Factor Receptor



Growth Factor-Dependent Pathway

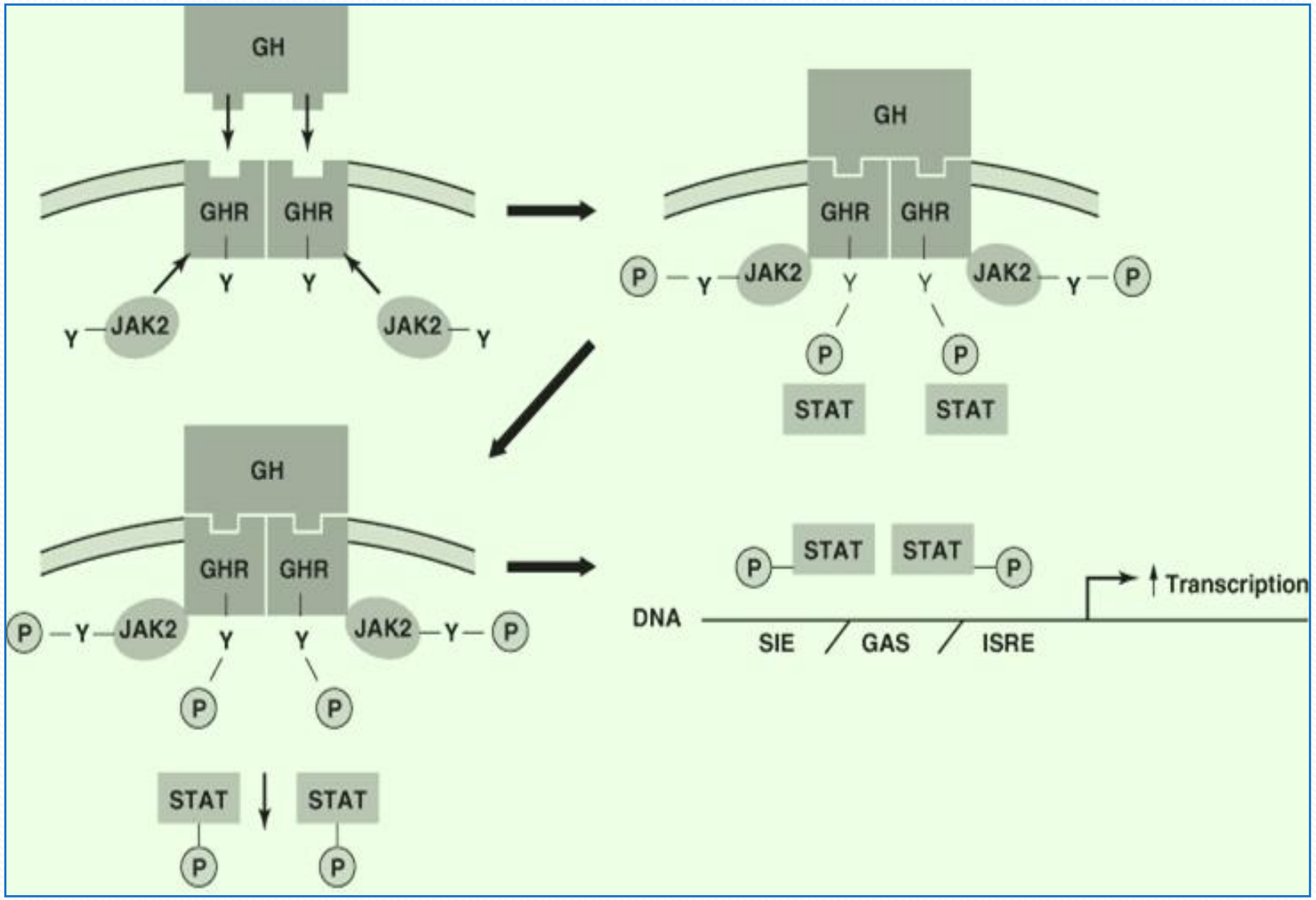


Insulin-dependent stimulation of glucose uptake occurs through a Ras-independent pathway!

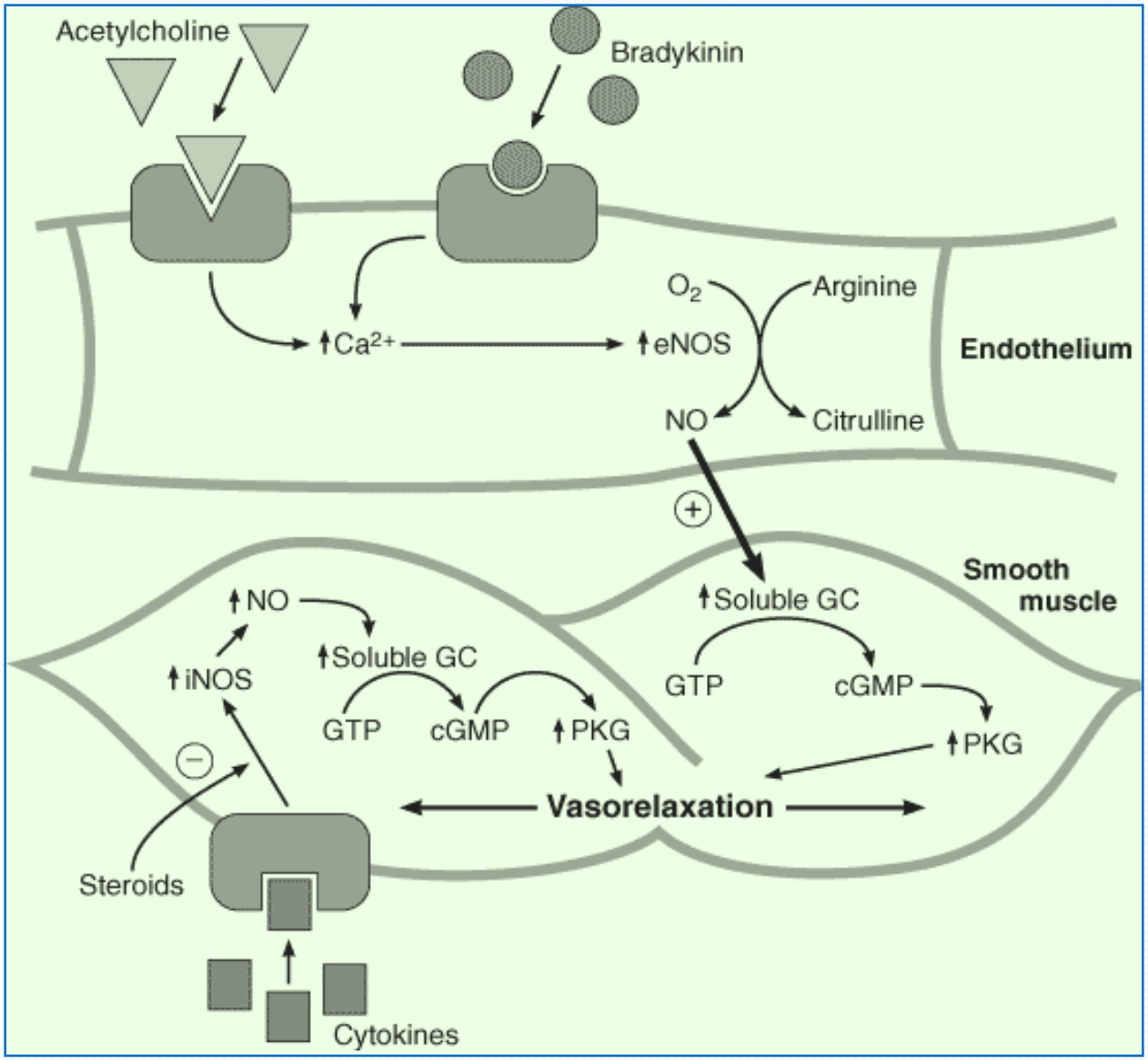


IRS binds/stimulates PI-3 kinase, which effects an, as yet, ill-defined cascade of events → the translocation of additional glucose transporters (GLUT-4) to the cell membrane.

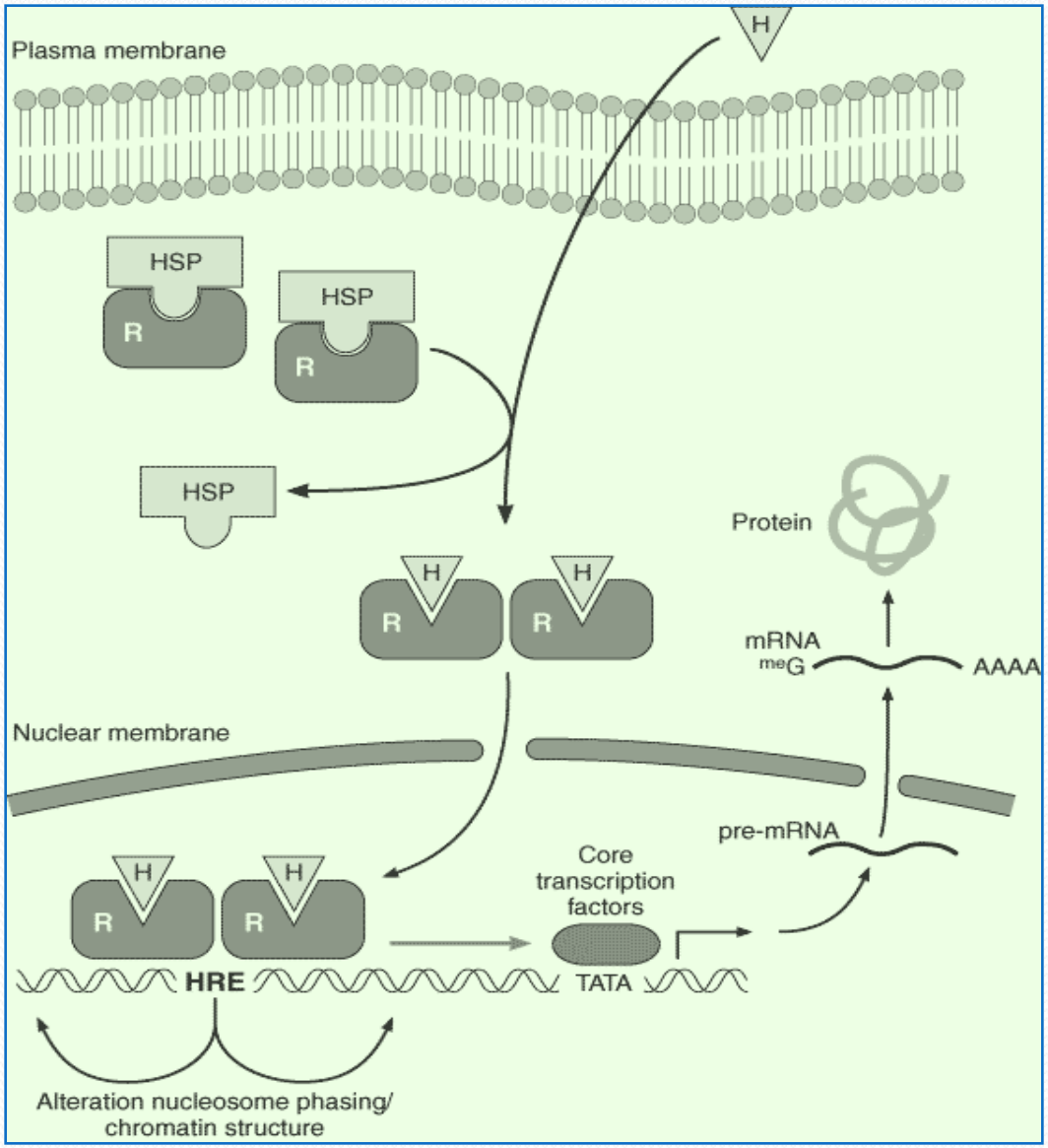
Signaling by the GH Receptor



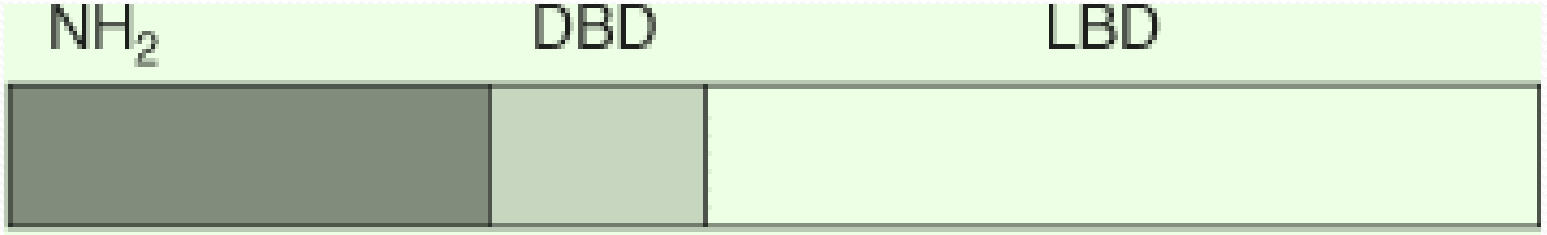
Signaling through NO



Steroid-Receptor Signaling



Steroid Receptor



Ligand-independent transactivation	DNA binding	Hetero- and homo-dimerization
	Dimerization	Ligand binding
		Ligand-dependent transactivation
		Nuclear translocation
		Association with heat shock proteins

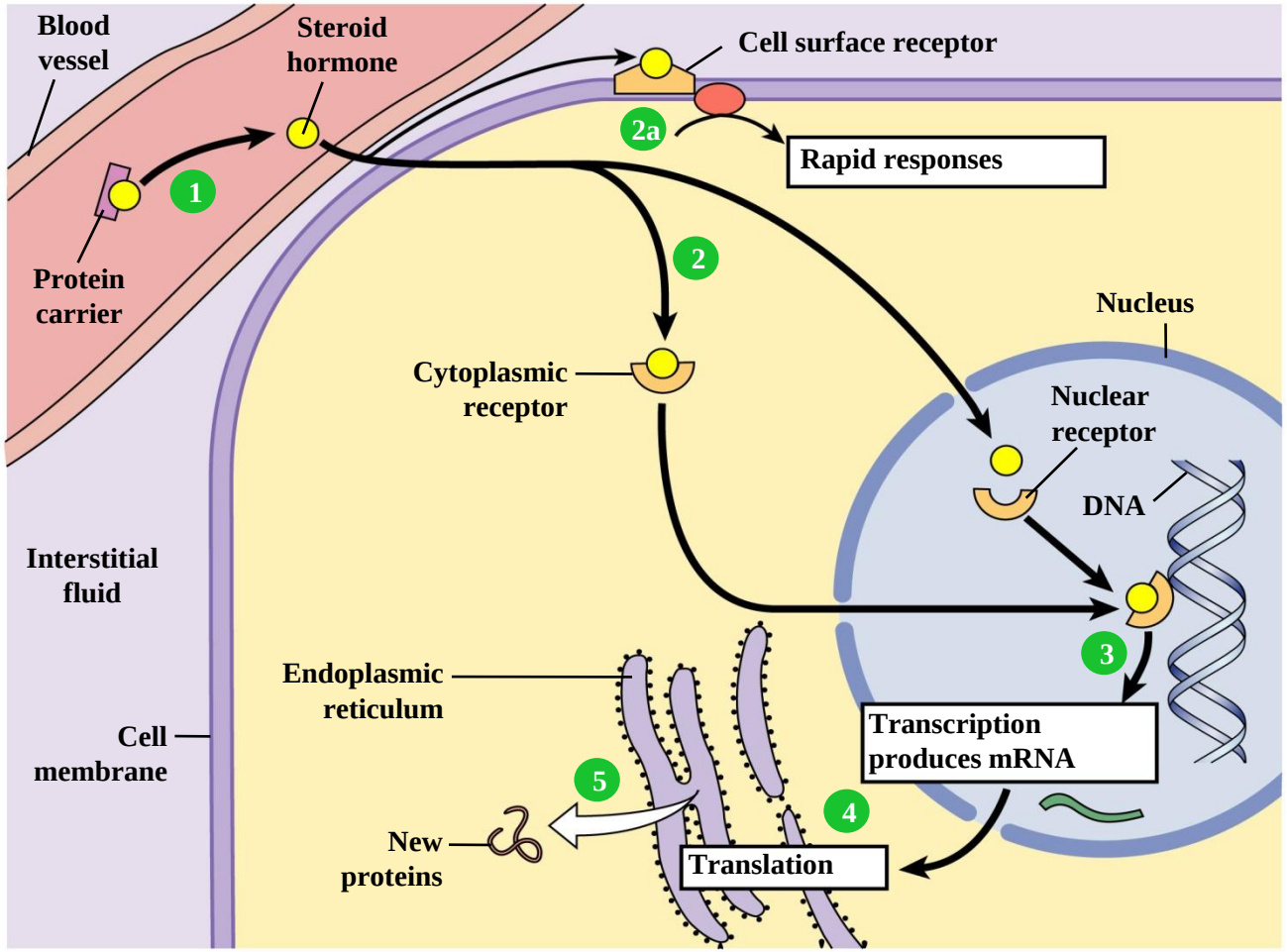
Steroid Hormones: Action

1 Most hydrophobic steroids are bound to plasma protein carriers. Only unbound hormones can diffuse into the target cell.

2 Steroid hormone receptors are typically in the cytoplasm or nucleus.

2a Some steroid hormones also bind to membrane receptors that use second messenger systems to create rapid cellular responses.

3 The receptor-hormone complex binds to DNA and activates or represses one or more genes.



4 Activated genes create new mRNA that moves into the cytoplasm.

5 Translation produces new proteins for cell processes.