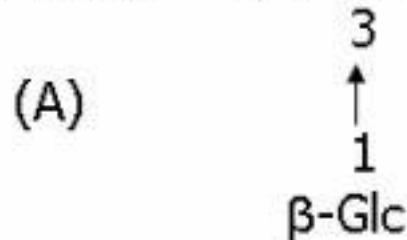


Major oligosaccharides recognized by plants:

(A) oligoglucans, (B) oligogalacturonide, (C) chitin-oligomer (D)
chitosan-oligomer. Glc, glucose; GalUA, galacturonic acid; GlcNAc, N-acetyl
glucosamine; GlcN, N-glucosamine.

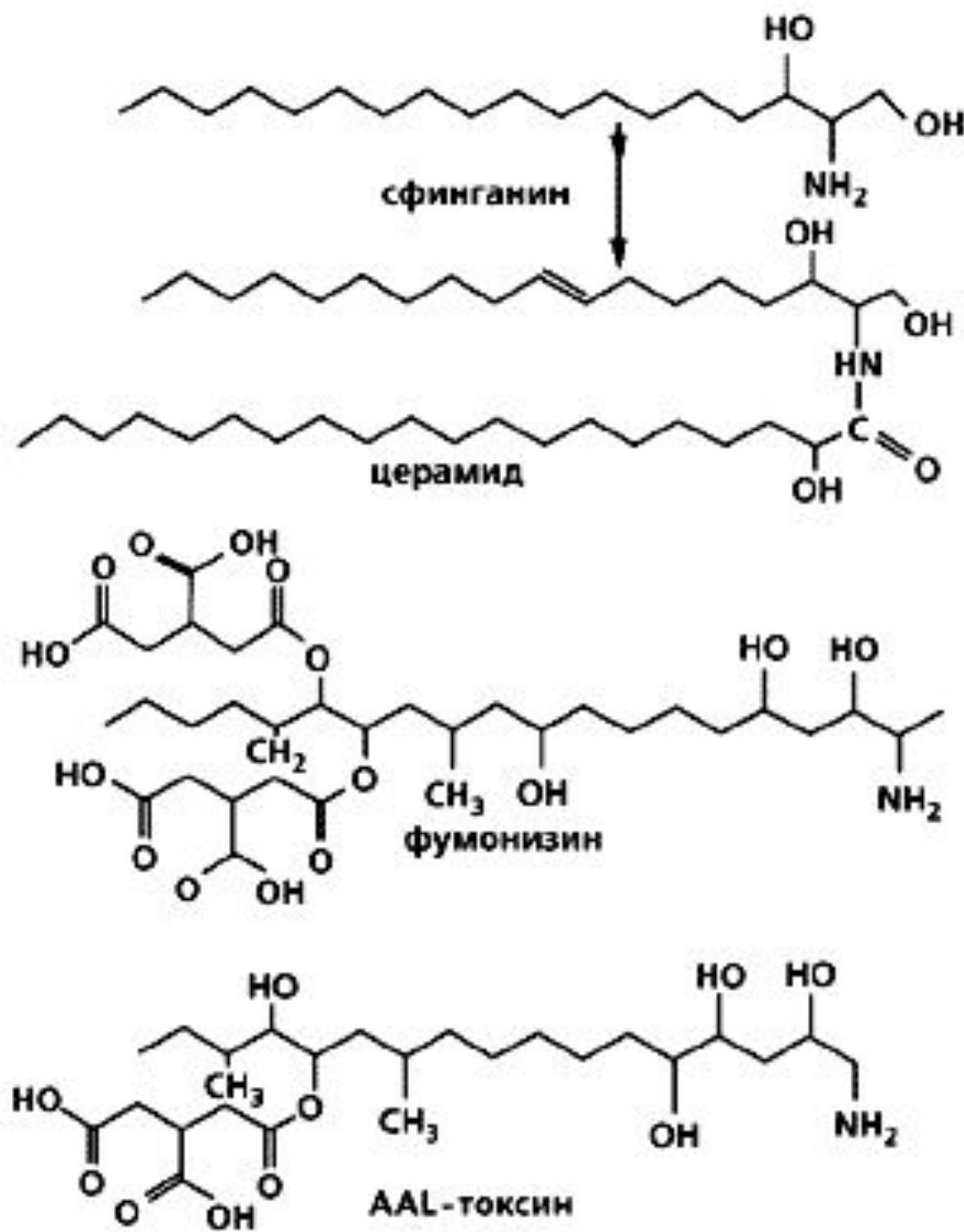
$\beta\text{-Glc}\text{ (1}\rightarrow\text{6)}$ $\beta\text{-Glc}\text{ (1}\rightarrow\text{6)}$ $\beta\text{-Glc}\text{ (1}\rightarrow\text{6)}$ $\beta\text{-Glc}\text{ (1}\rightarrow\text{6)}$ $\beta\text{-Glc}\text{ (1}\rightarrow\text{6)}$



(B) $\alpha\text{-GalUA}\text{ (1}\rightarrow\text{[4})$ $\alpha\text{-GalUA}\text{ (1}\rightarrow\text{]}_n\text{ 4})$ $\alpha\text{-GalUA}$

(C) $\beta\text{-GlcNAc}\text{ (1}\rightarrow\text{[4})$ $\beta\text{-GlcNAc}\text{ (1}\rightarrow\text{]}_n\text{ 4})$ $\beta\text{-GlcNAc}$

(D) $\beta\text{-GlcN}\text{ (1}\rightarrow\text{[4})$ $\beta\text{-GlcN}\text{ (1}\rightarrow\text{]}_n\text{ 4})$ $\beta\text{-GlcN}$



Синтез церамида (регулятора клеточного цикла и апоптоза) из сфинганина и структура грибных антиметаболитов фумонизина и AAL-токсина.

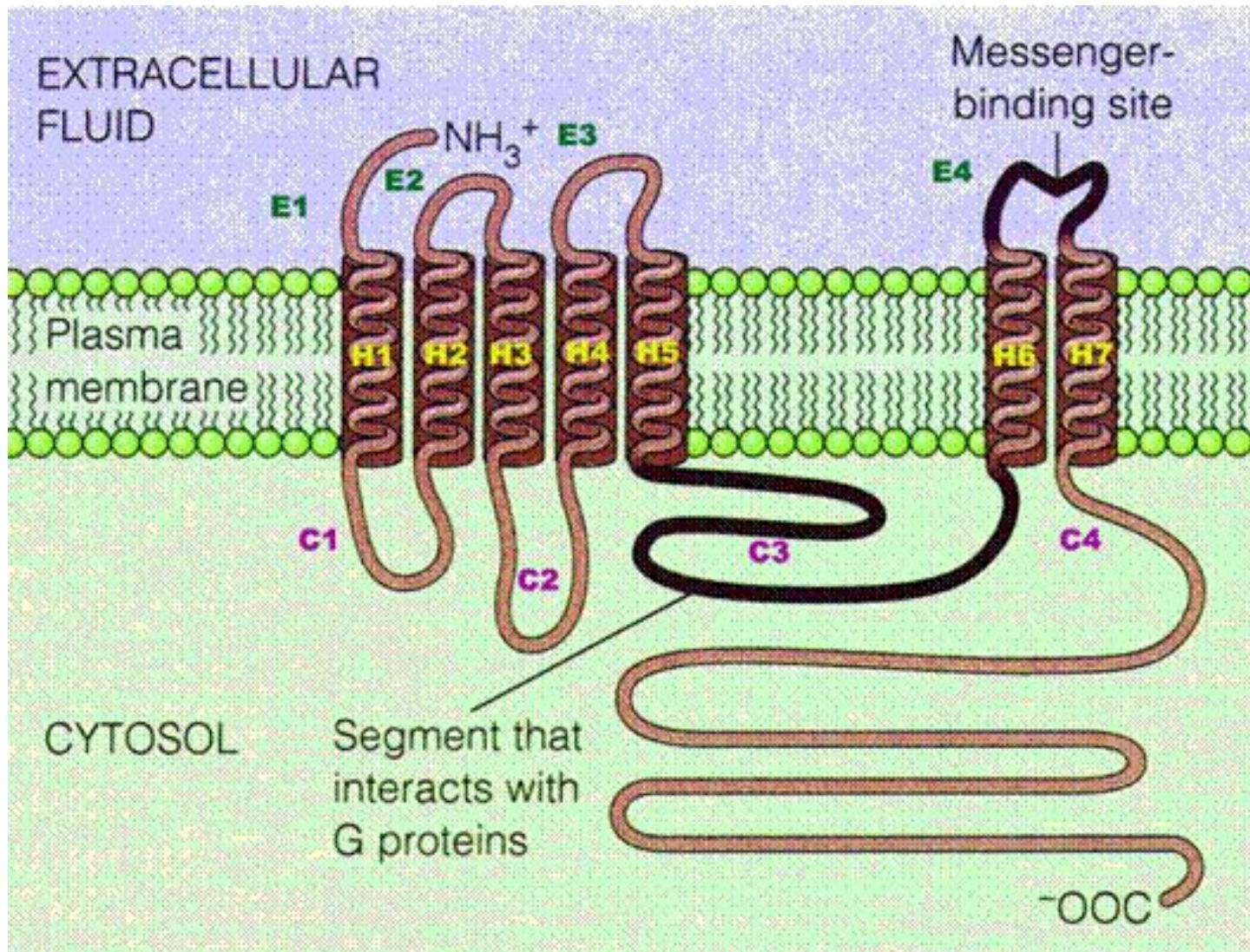
У растений эти токсины подавляют защитные реакции и транспорт сахаров, приводят к неопластическому росту и некрозам.

У человека и сельскохозяйственных животных фумонизин вызывает гепатотоксикозы, разные формы неоплазмозов и гибель клеток.

Сигнальные системы передачи сигнала для возбуждения экспрессии защитных генов:

1. циклоаденилатная,
2. МАР-киназная (mitogen-activated protein-kinase),
3. фосфатидокислотная,
4. кальциевая,
5. липоксигеназная,
6. НАДФ-Н-оксидазная (супероксидсингтазная),
7. NO-синтазная.

Structure of GPCRs



Classification of GPCRs:

Class A (1) (Rhodopsin-like)

Class B (2) (Secretin receptor family)

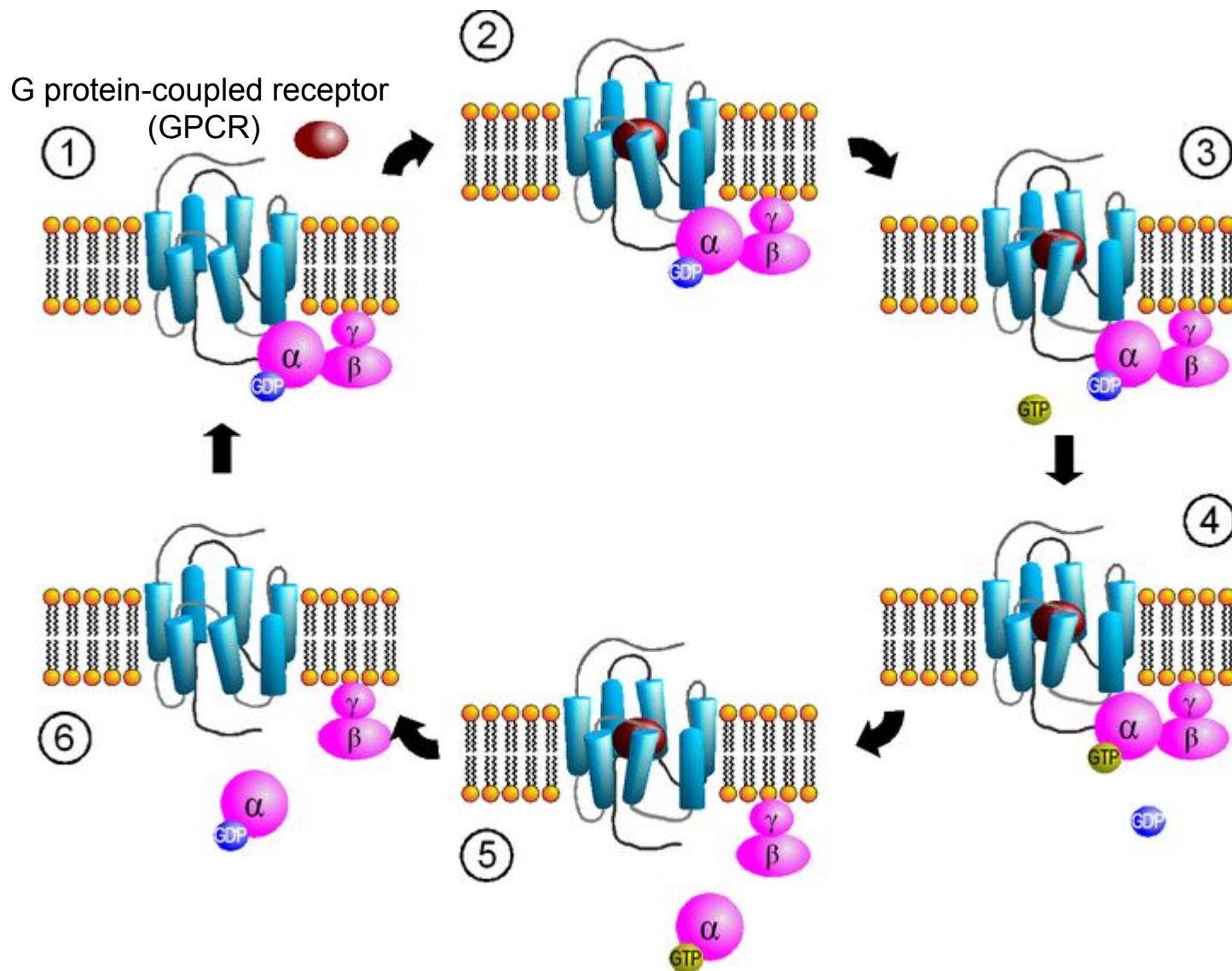
Class C (3) (Metabotropic glutamate/pheromone)

Class D (4) (Fungal mating pheromone receptors)

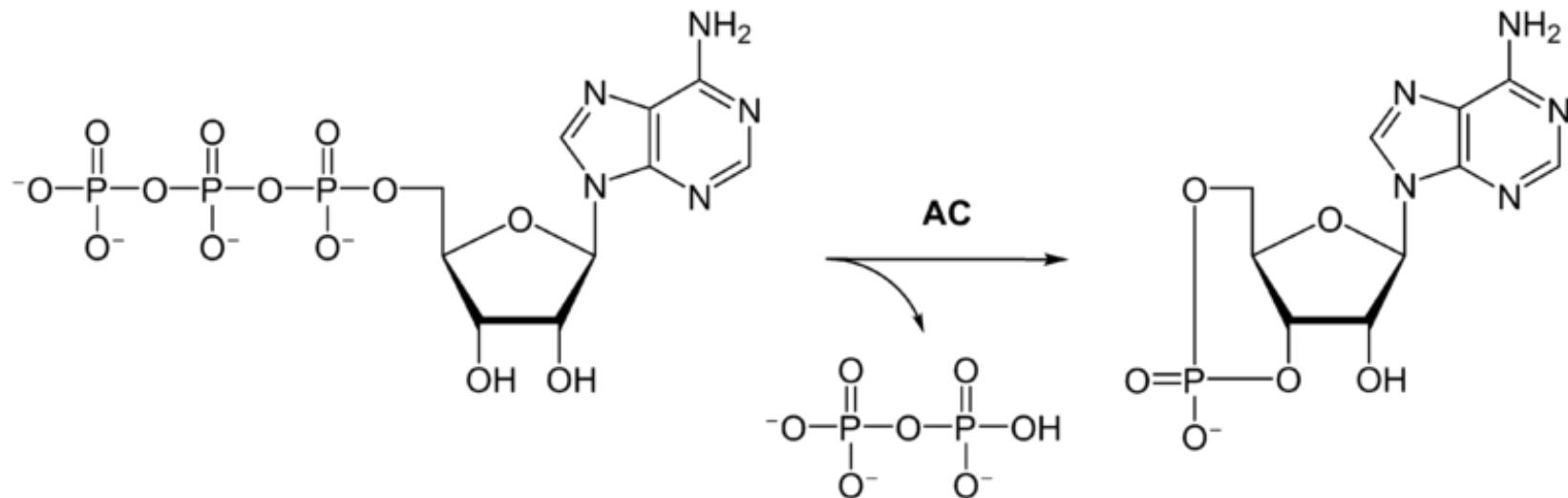
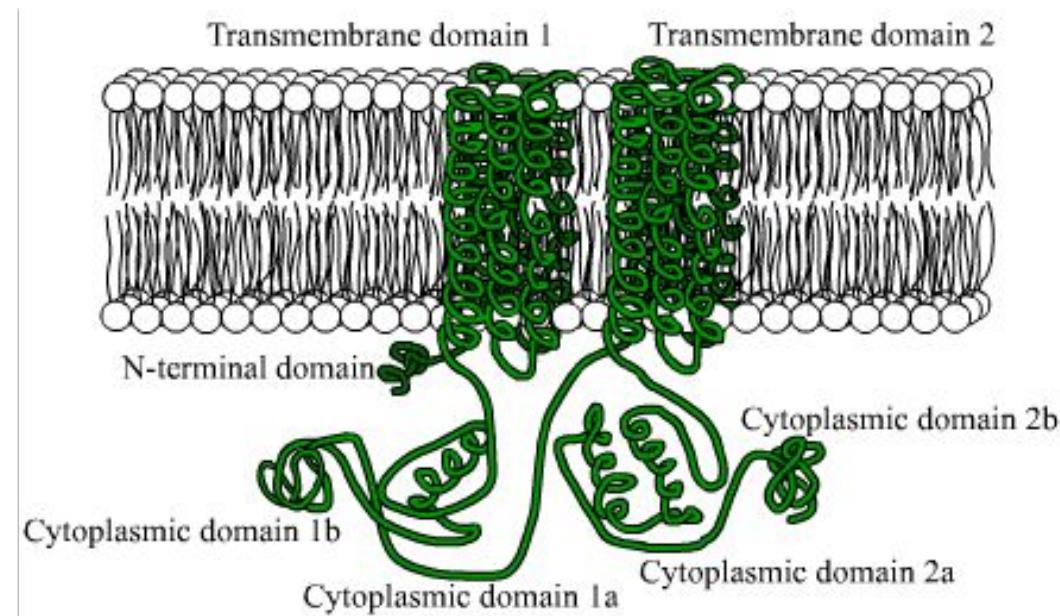
Class E (5) (Cyclic AMP receptors)

Class F (6) (Frizzled/Smoothened)

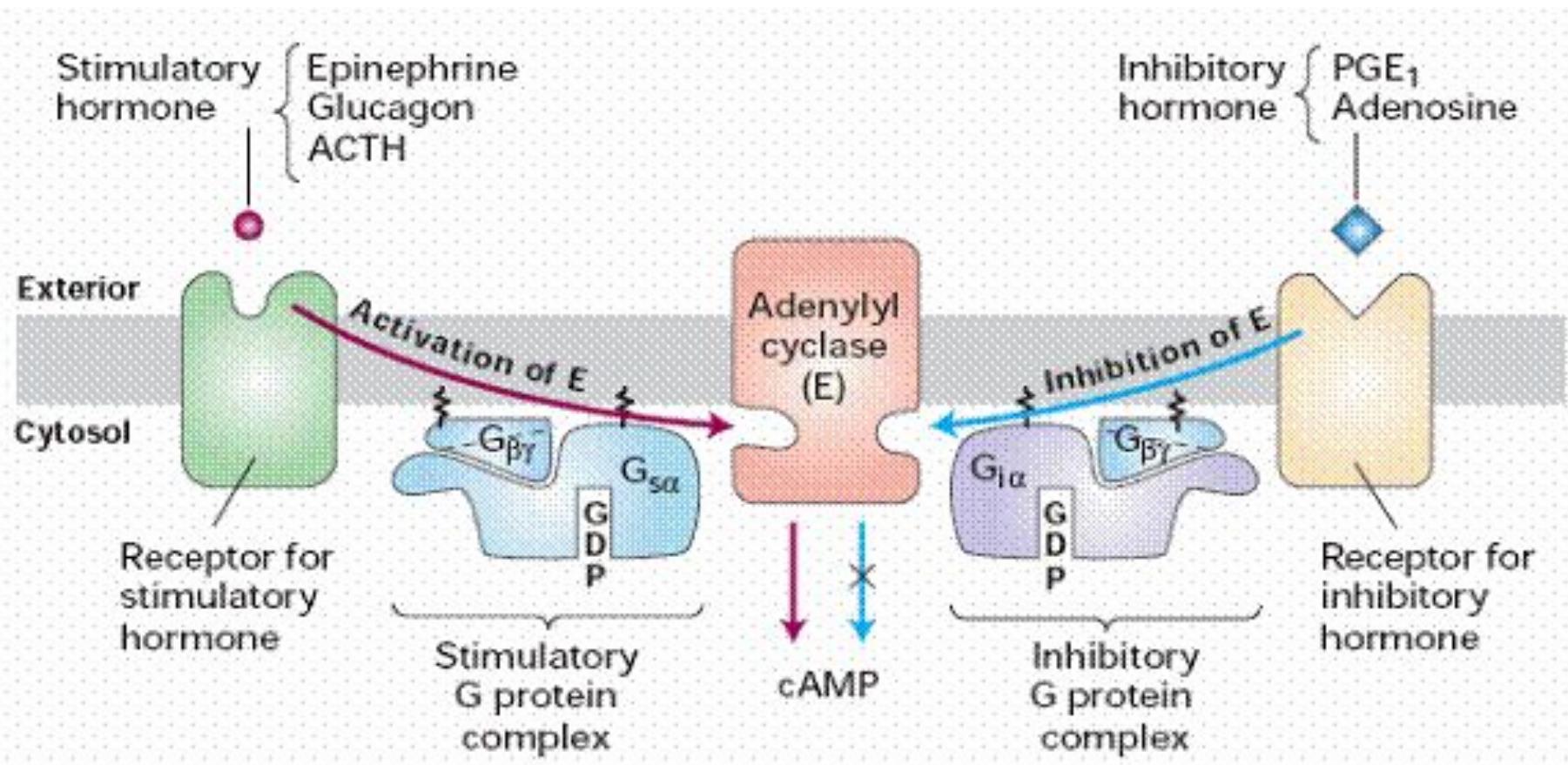
G-proteins



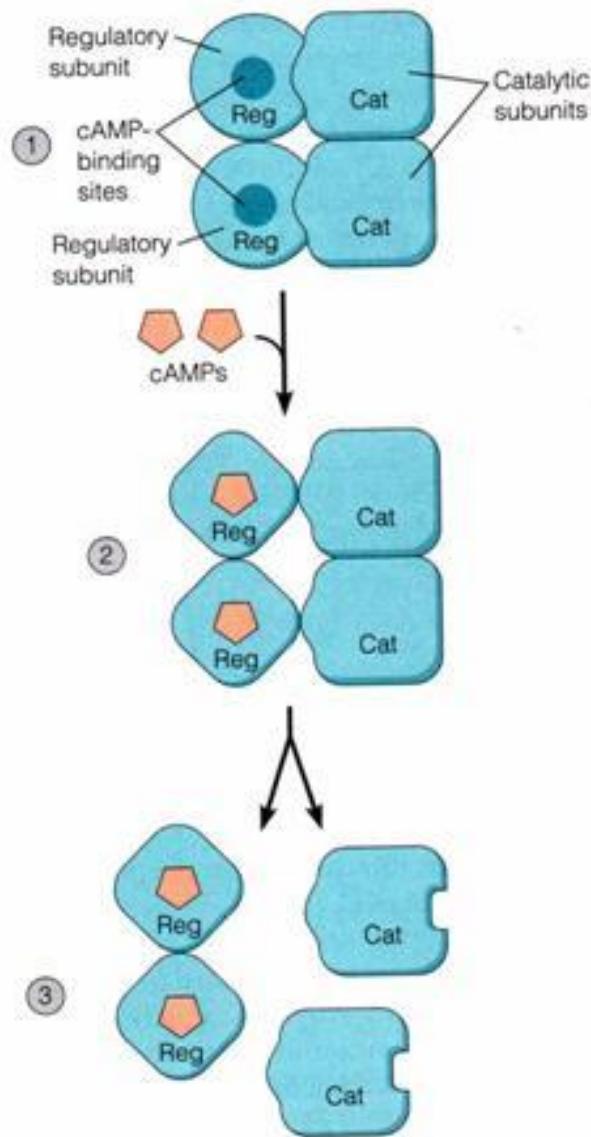
Adenylate Cyclase catalyzes the conversion of ATP to 3',5'-cyclic AMP

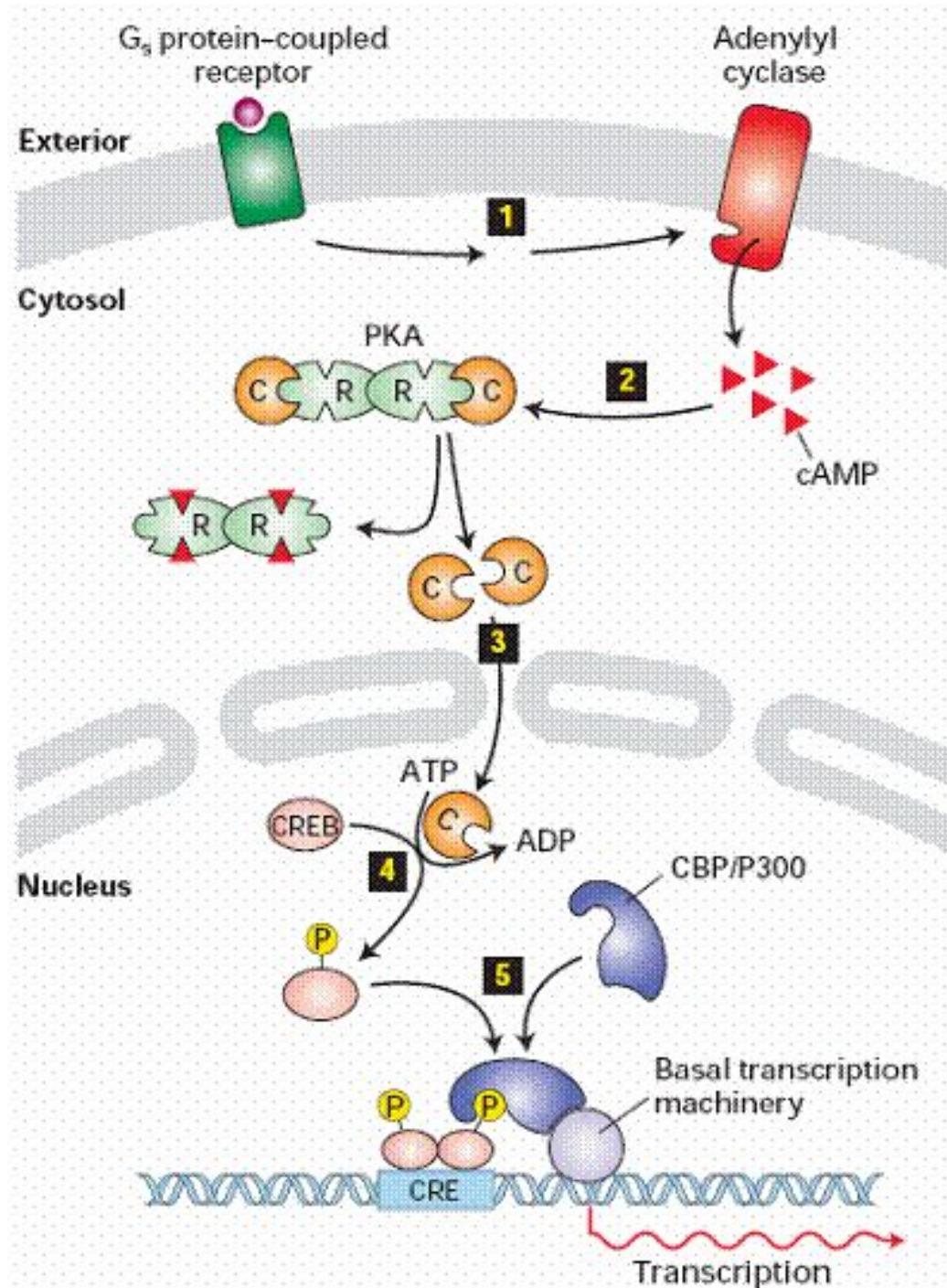


Regulation of Adenylate cyclase



Activation of PK-A

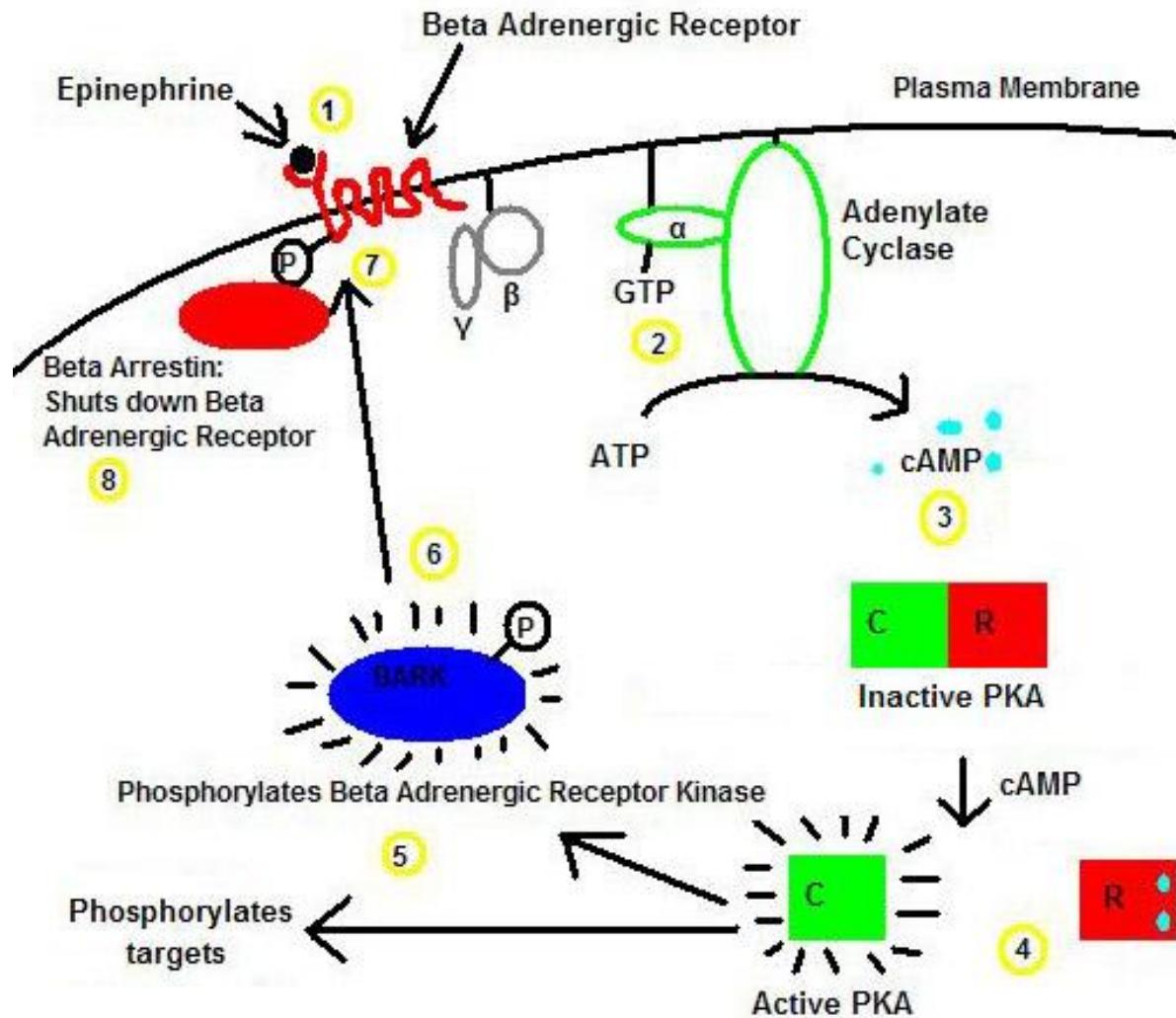




CREB (cAMP response element binding)

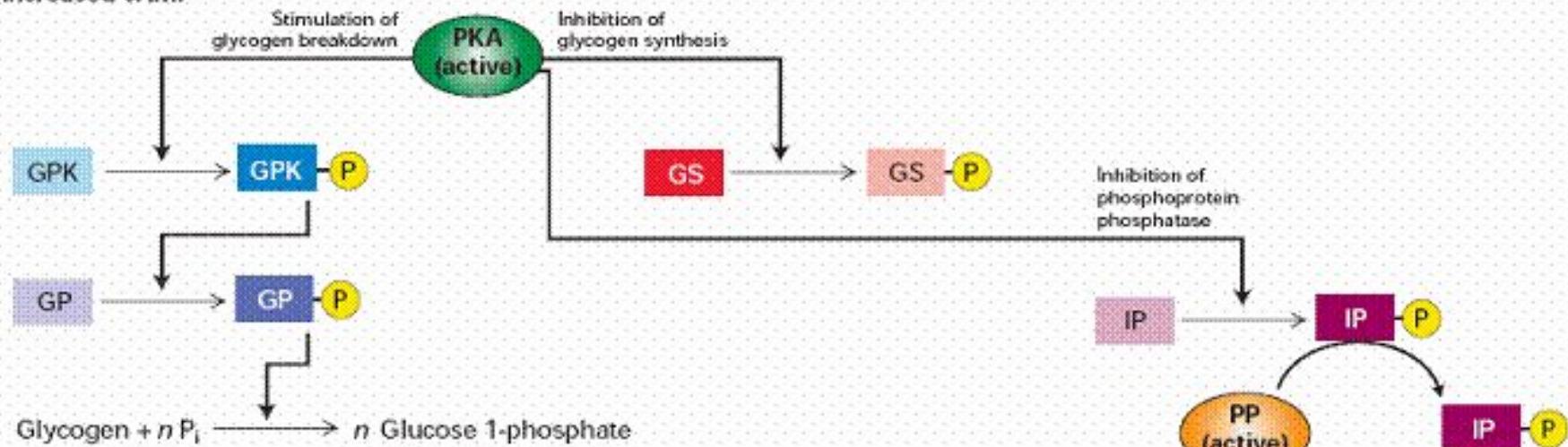
Phosphorylated CREB then binds with CBP/P300 (co-activator) and forms activator for cyclic AMP response element (CRE). This activator then binds with CRE and express various genes and proteins.

Beta adrenergic receptor kinase pathway

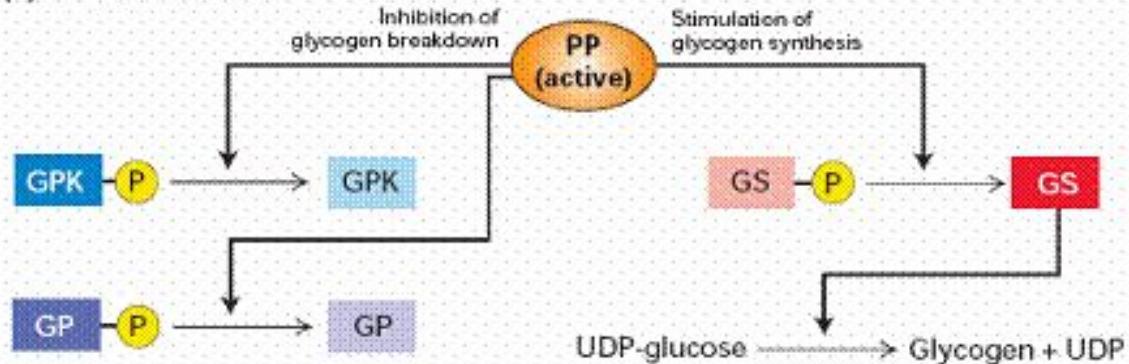


Regulation of glycogen metabolism by cAMP

(a) Increased cAMP



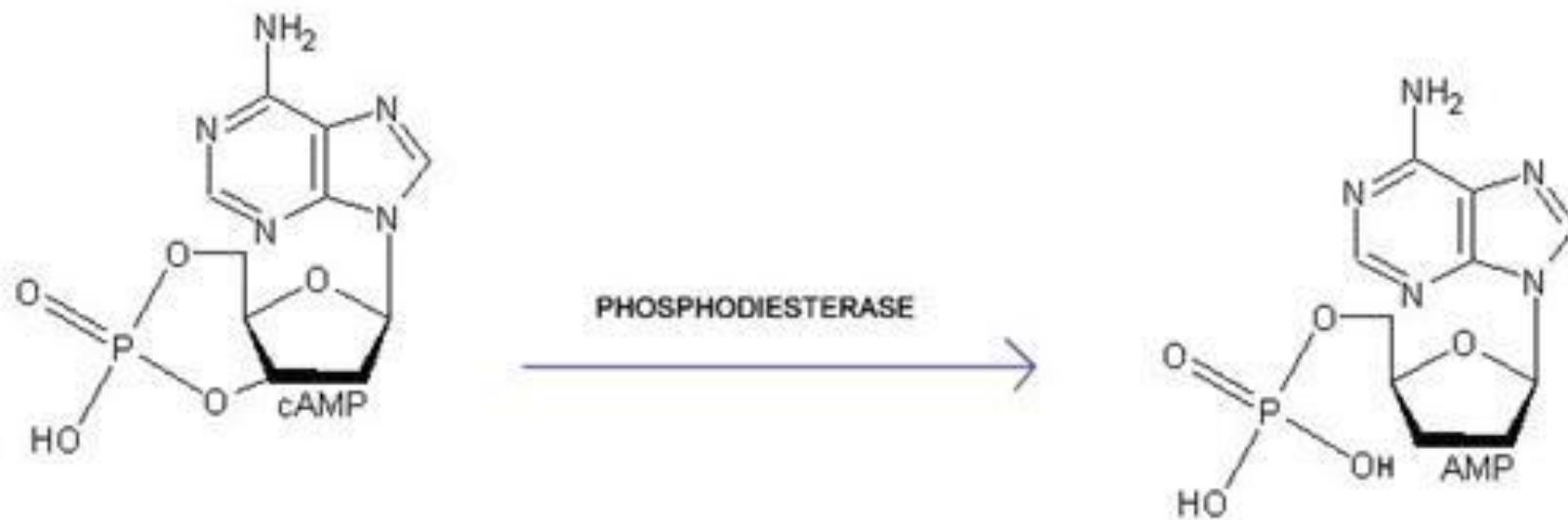
(b) Decreased cAMP

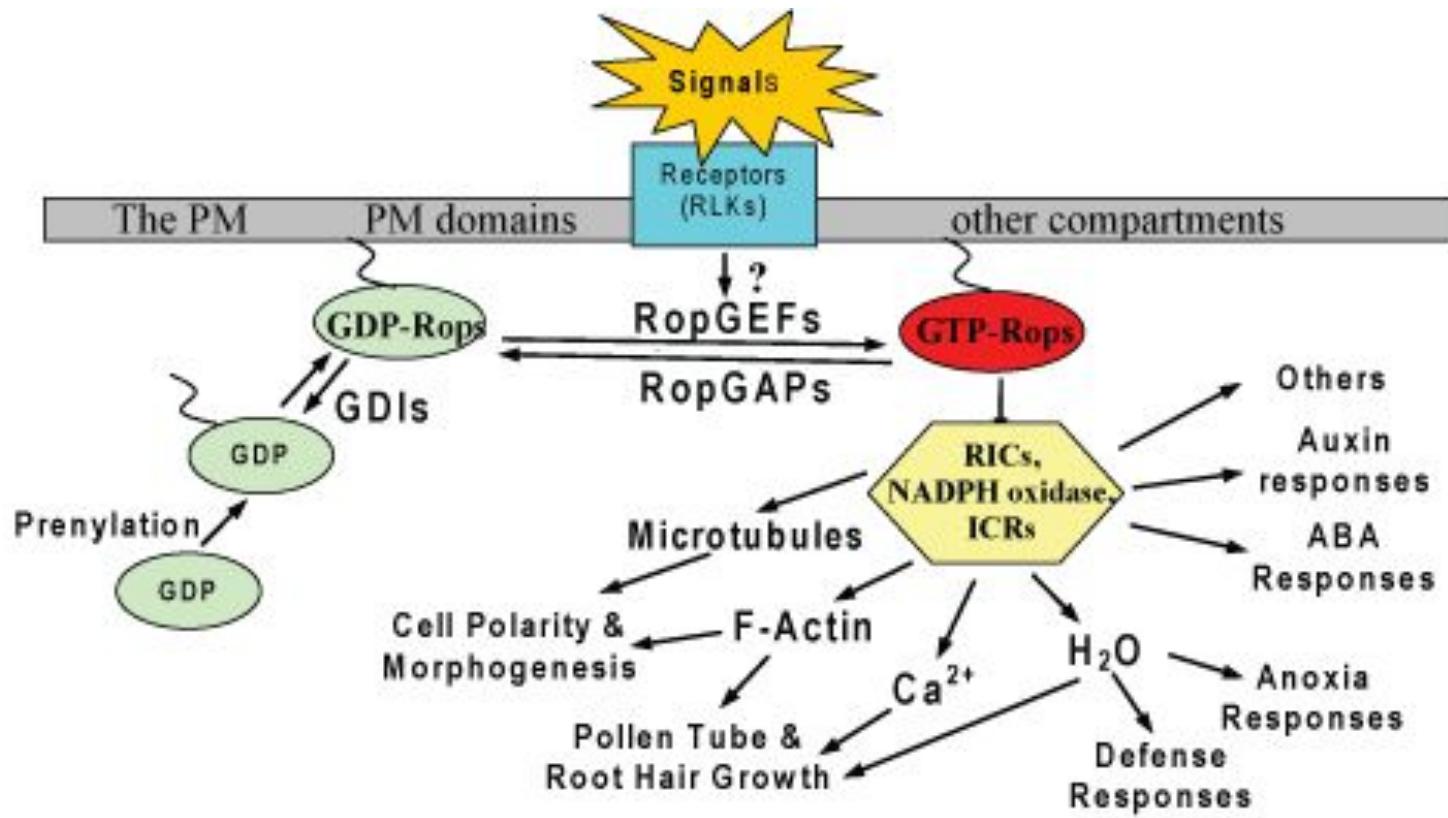


Abbreviations:

PKA	Protein kinase A
PP	Phosphoprotein phosphatase
GPK	Glycogen phosphorylase kinase
GP	Glycogen phosphorylase
GS	Glycogen synthase
IP	Inhibitor of phosphoprotein phosphatase

Degradation of cAMP

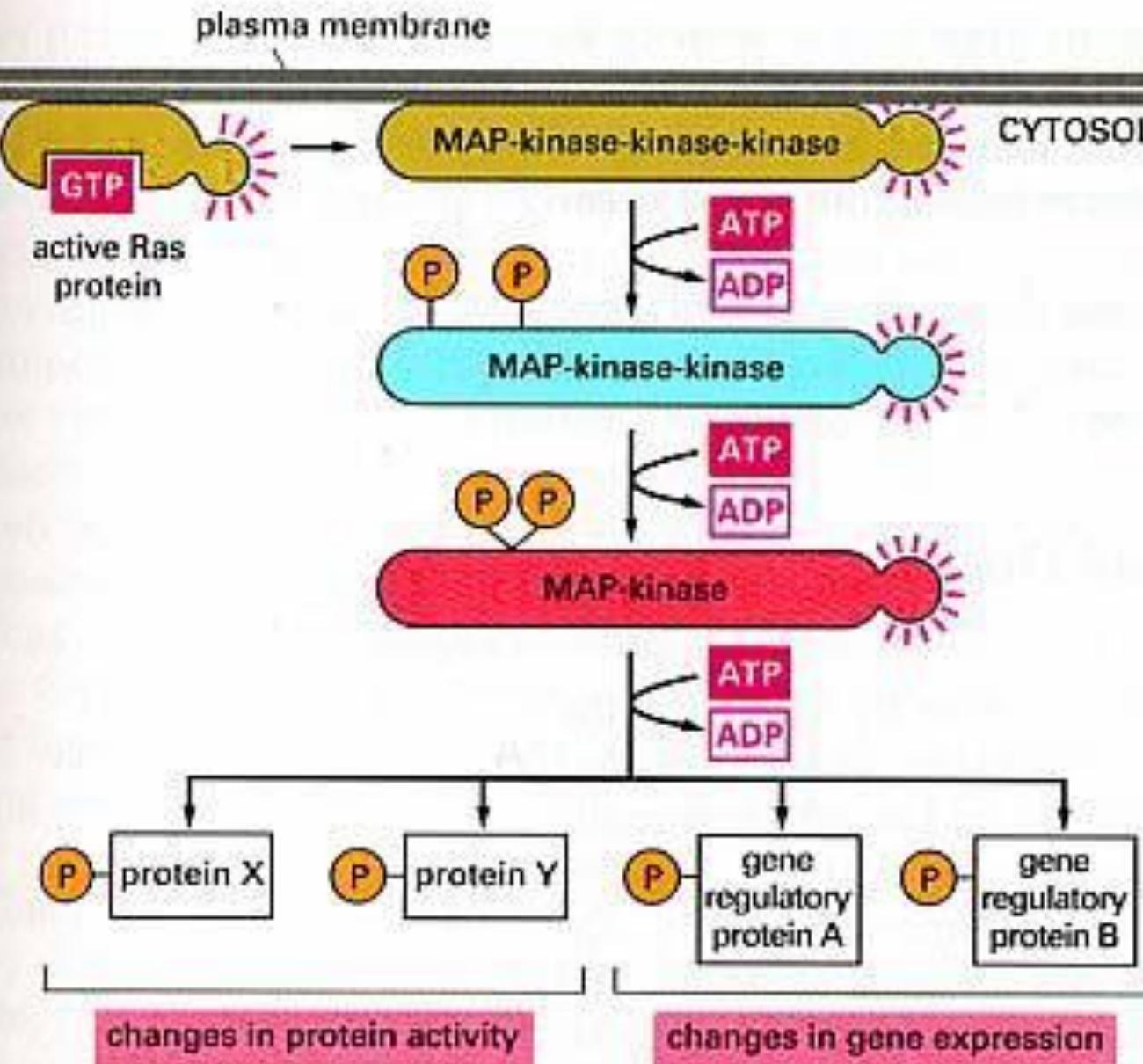




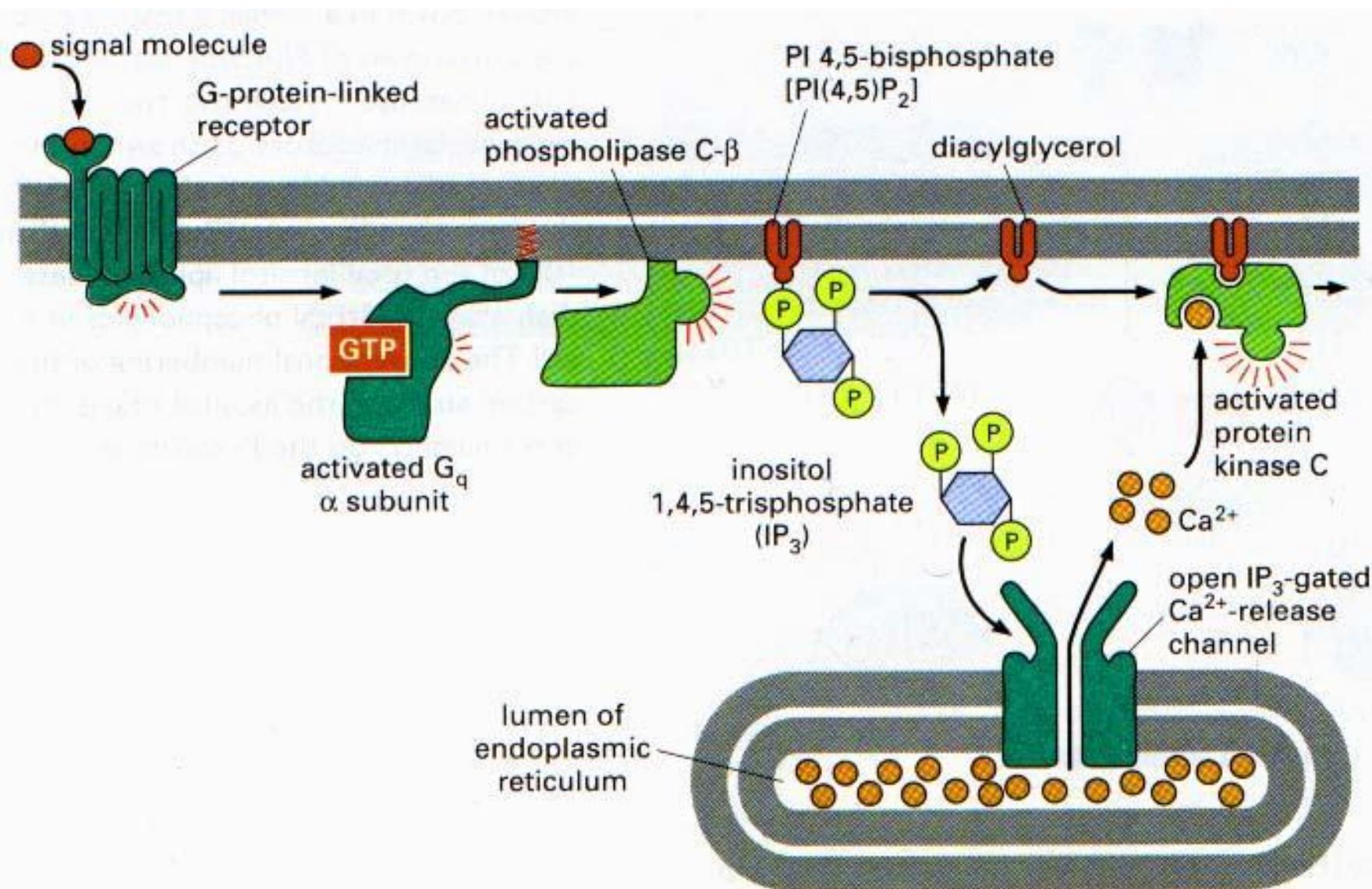
A generalized scheme illustrating the role of Rop GTPase as a signaling switch and a “hub” for controlling signaling networks.

RLK, receptor-like ser/thr kinases; GEF, guanine nucleotide exchange factor; GDI, guanine nucleotide dissociation inhibitor; RopGAP, Rop GTPase activating protein. RIC, Rop-interacting CRIB-containing proteins. ICR, interactor of constitutively active ROPs.

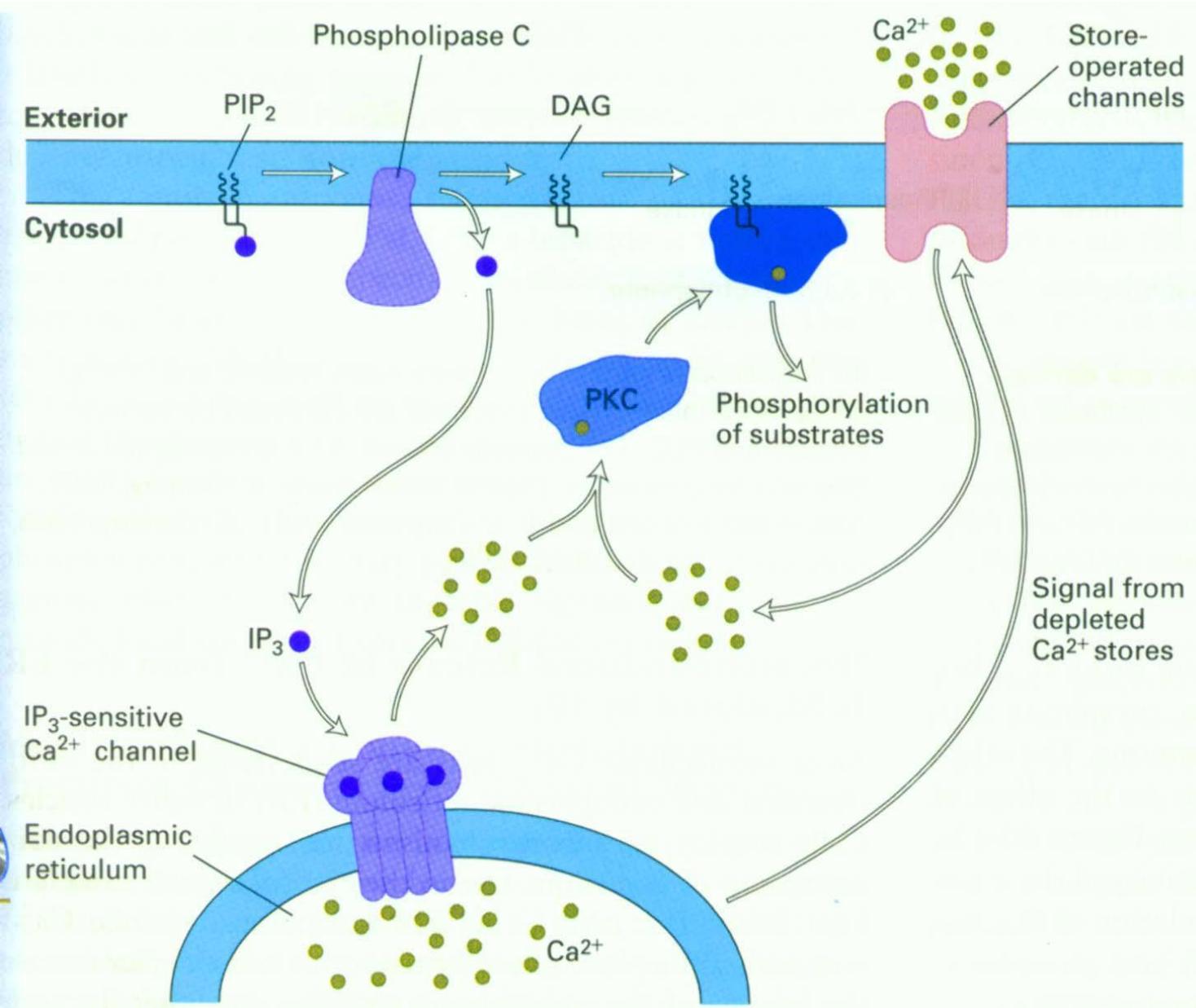
MAP-kinase serine/threonine phosphorylation pathway activated by Ras



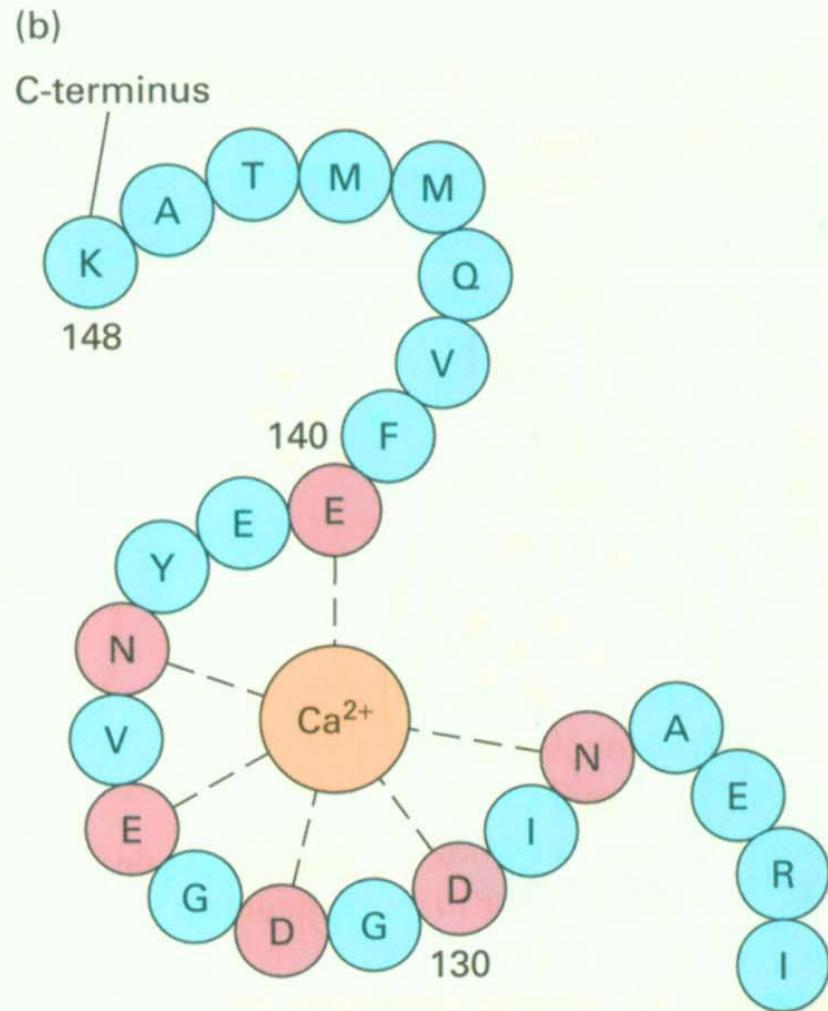
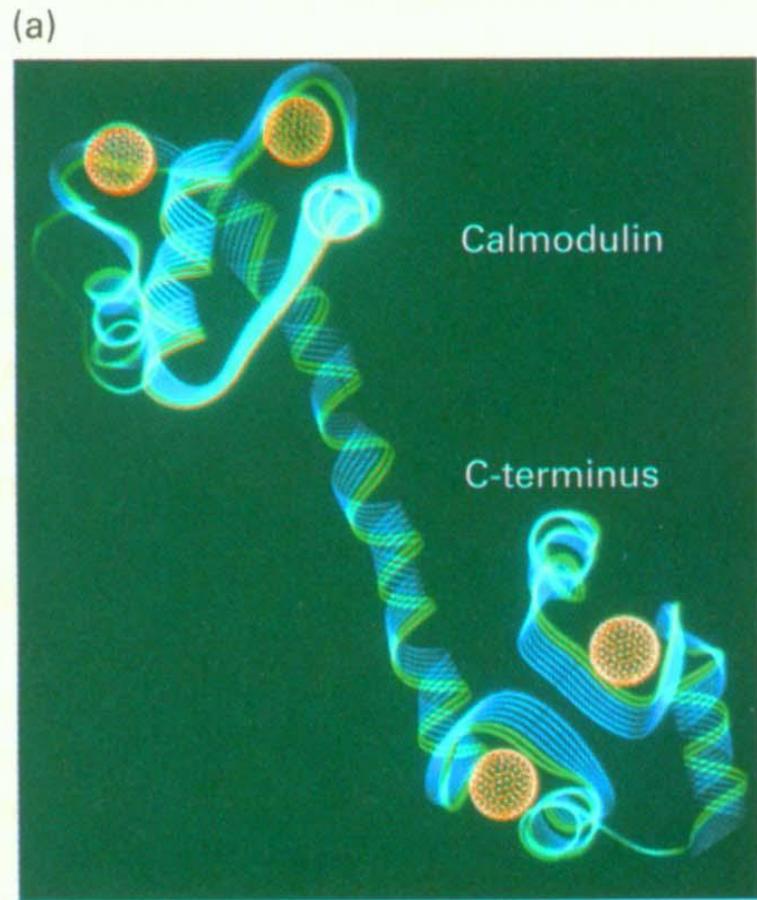
The pathway through phospholipase C results in a rise in intracellular Ca²⁺



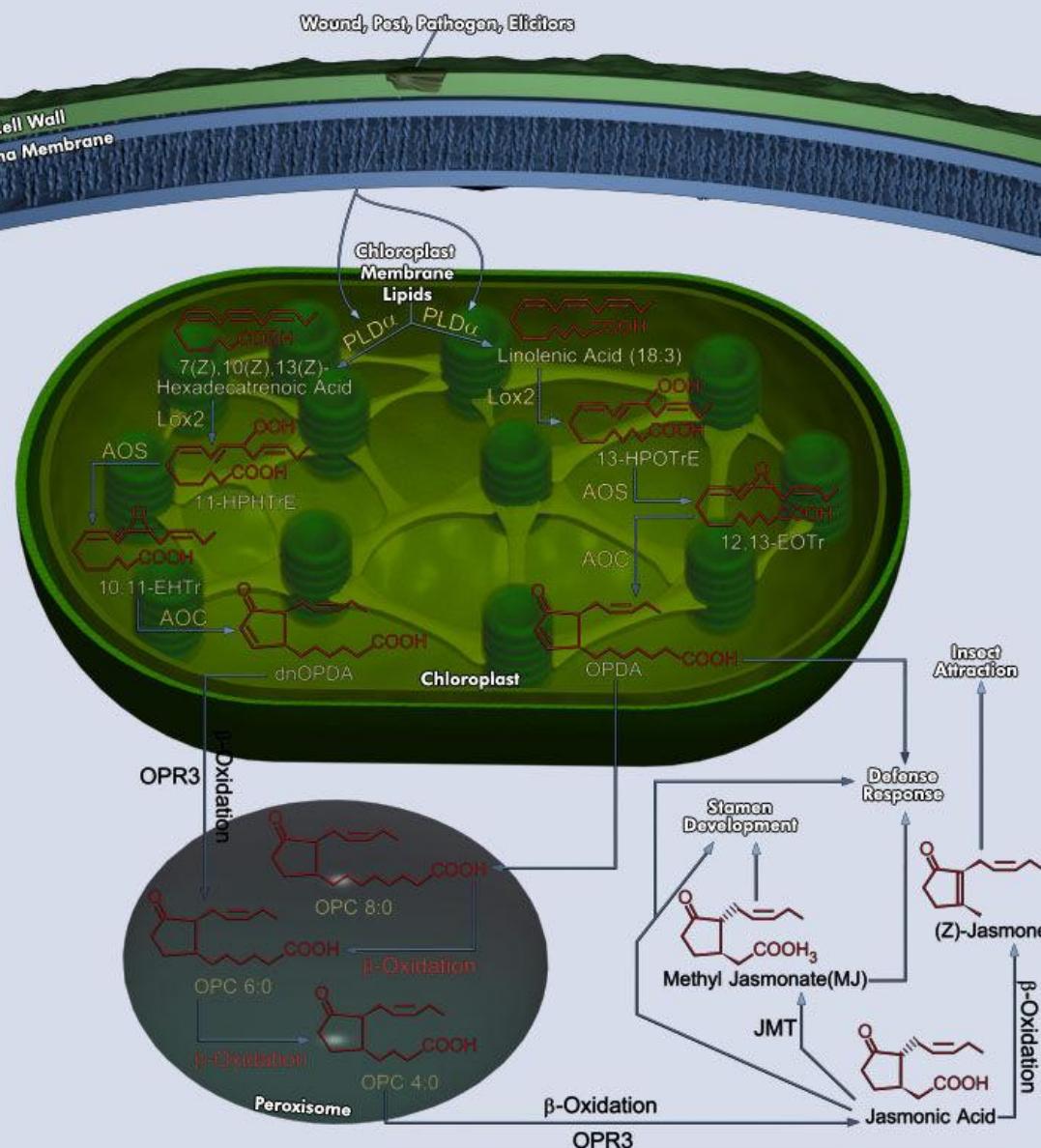
Elevation of cytosolic Ca^{2+} via the IP₃ signaling pathway

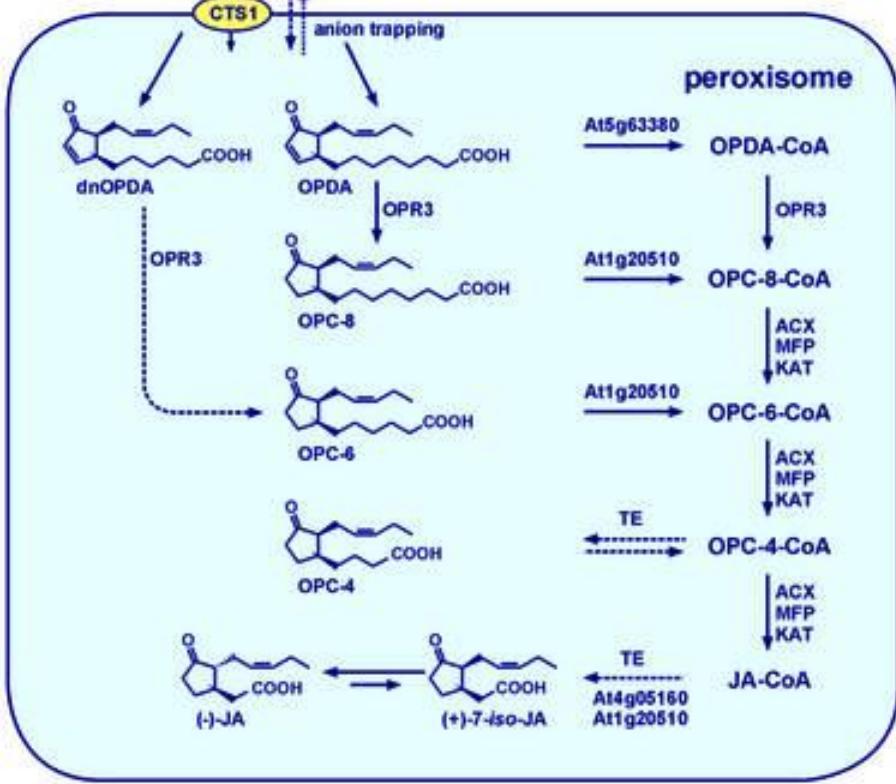
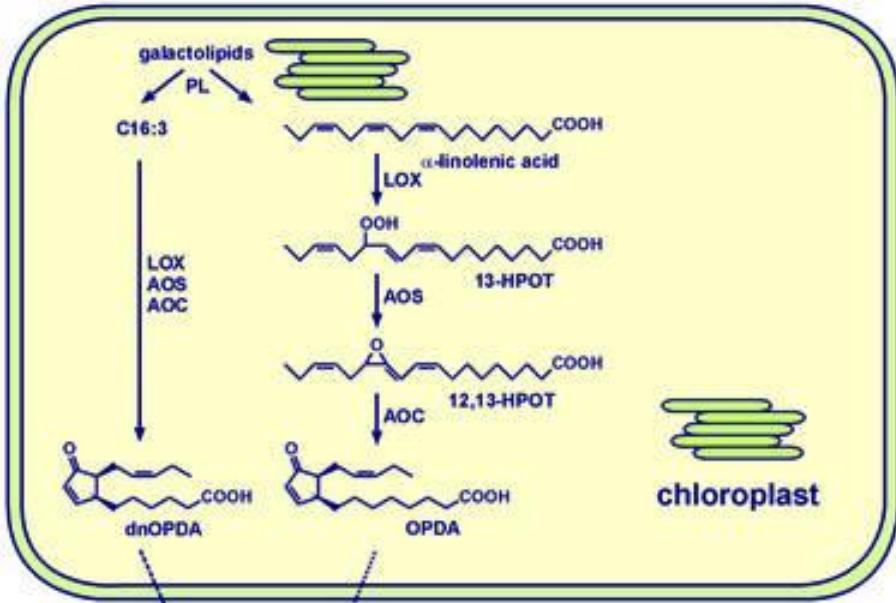


Calmodulin, a cytosolic protein of 148 amino acids that bind Ca^{2+} ions

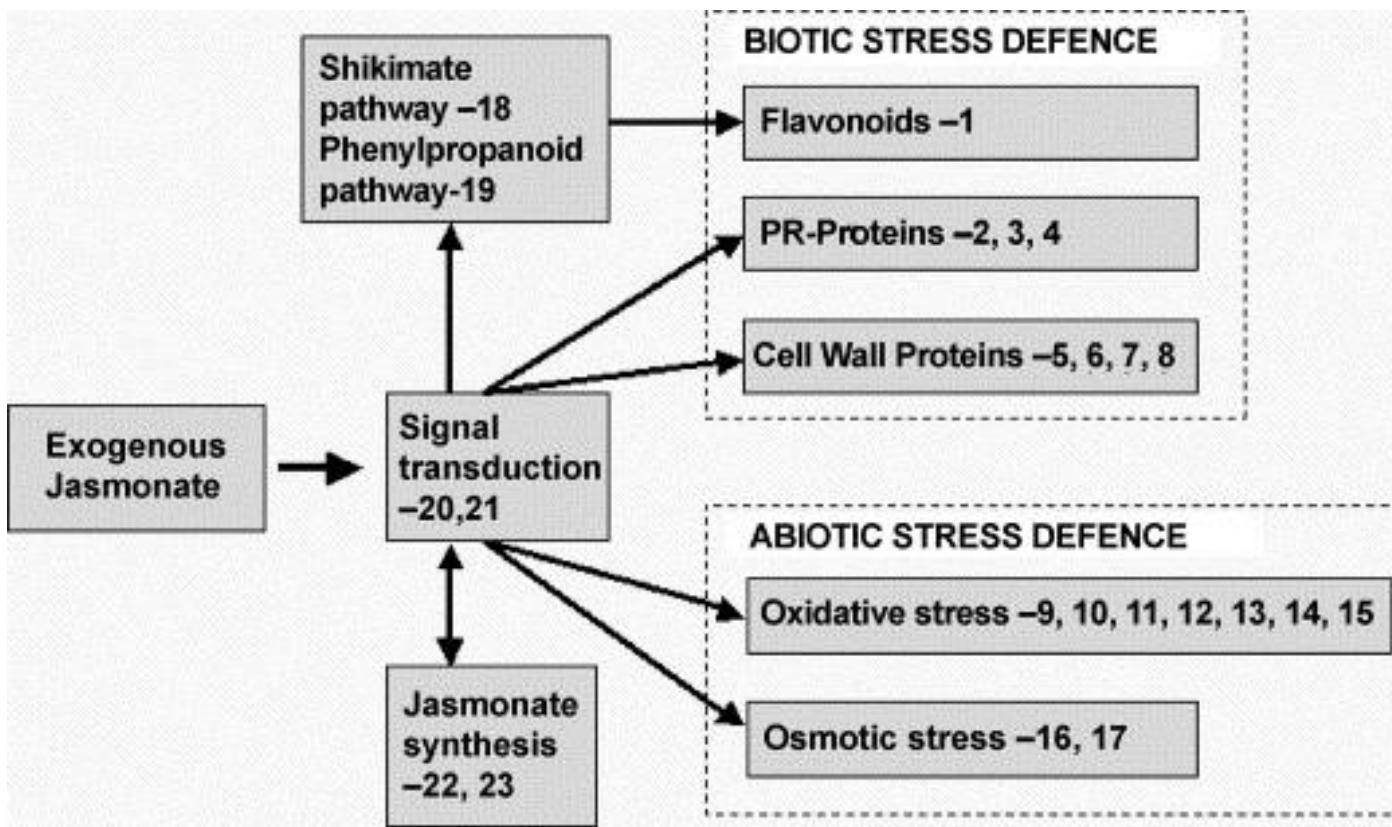


Jasmonate Biosynthesis in *Arabidopsis*



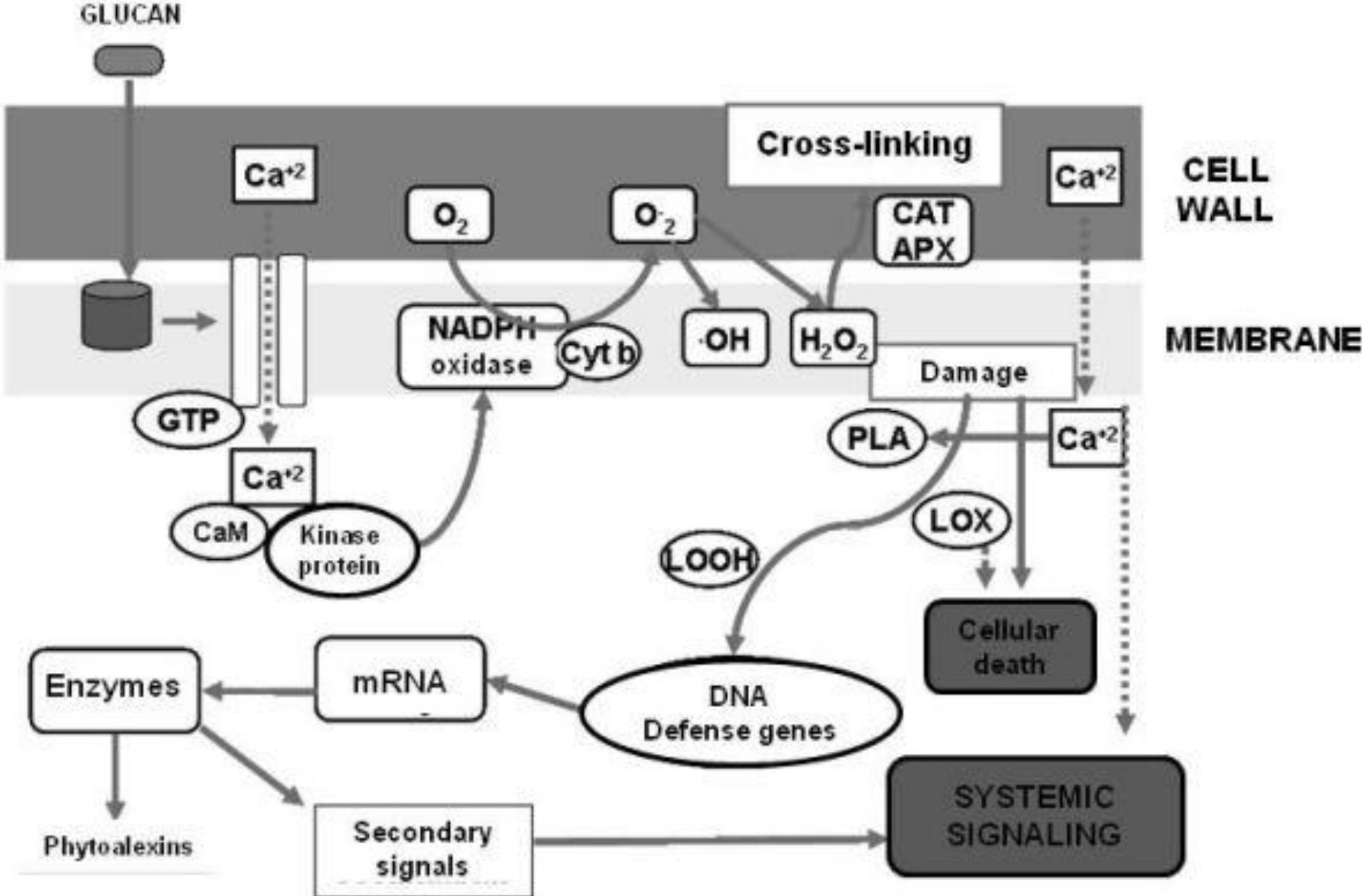


Possible gene network that is activated following application of exogenous methyl jasmonate



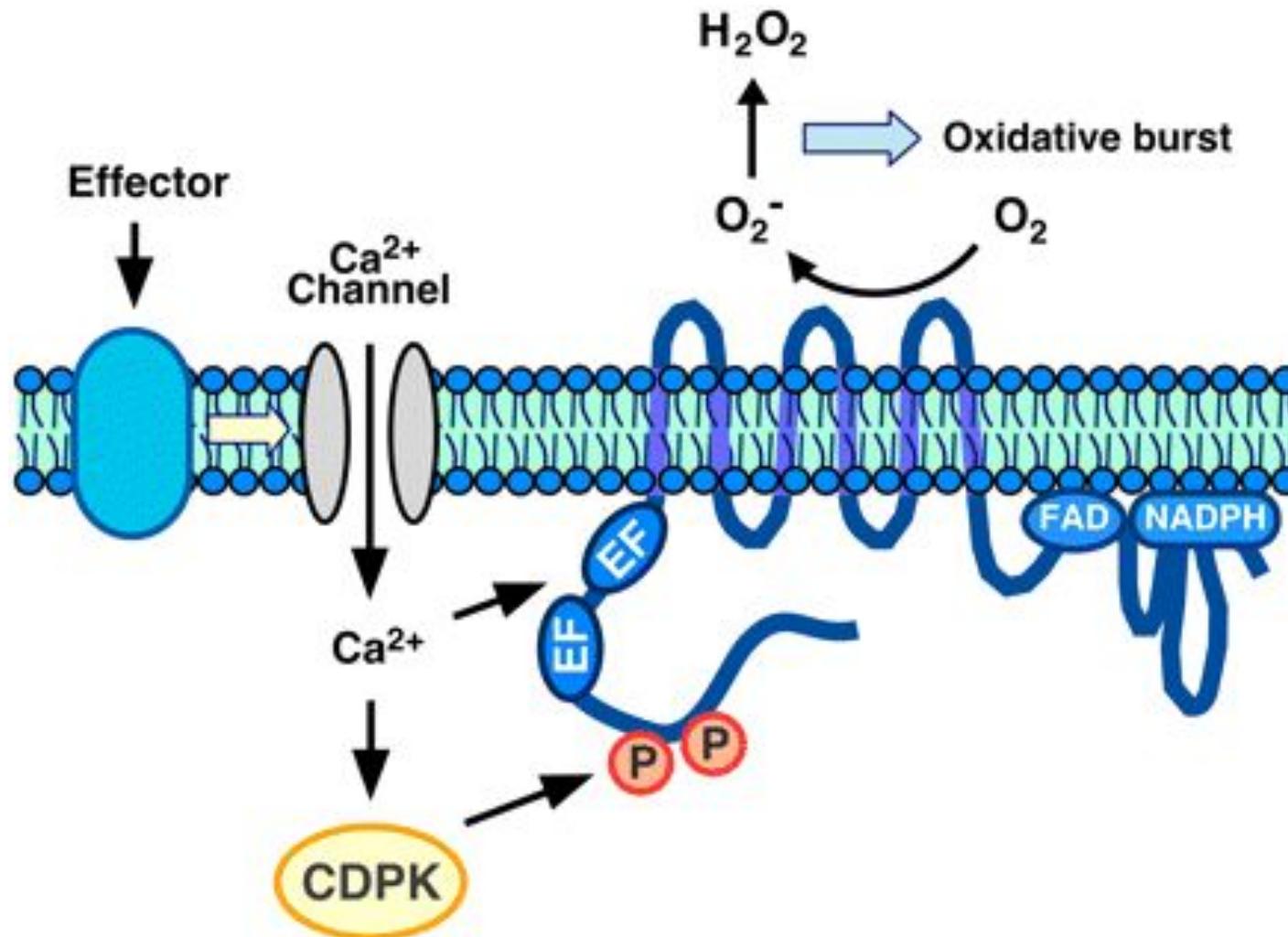
(1) Chalcone synthase; (2) pathogenesis-related (PR) protein 5; (3) PR-protein 10; (4) benzothiadiazole-induced protein; (5) dirigent; (6) glycine-rich protein; (7) proline-rich protein; (8) actin; (9) glutathione-S-transferase; (10) ferredoxin; (11) haemoglobin; (12) DNA repair protein; (13) aldose reductase; (14) dTDP-glucose 4,6-dehydratase; (15) methionine synthase; (16) phosphoethanolamine *N*-methyltransferase; (17) trehalose-6-phosphate synthase; (18) DAHP synthase; (19) phenylalanine ammonia lyase; (20) Myb transcription factor; (21) receptor-like protein; (22) patatin lipase-like protein; (23) lipoxygenase.

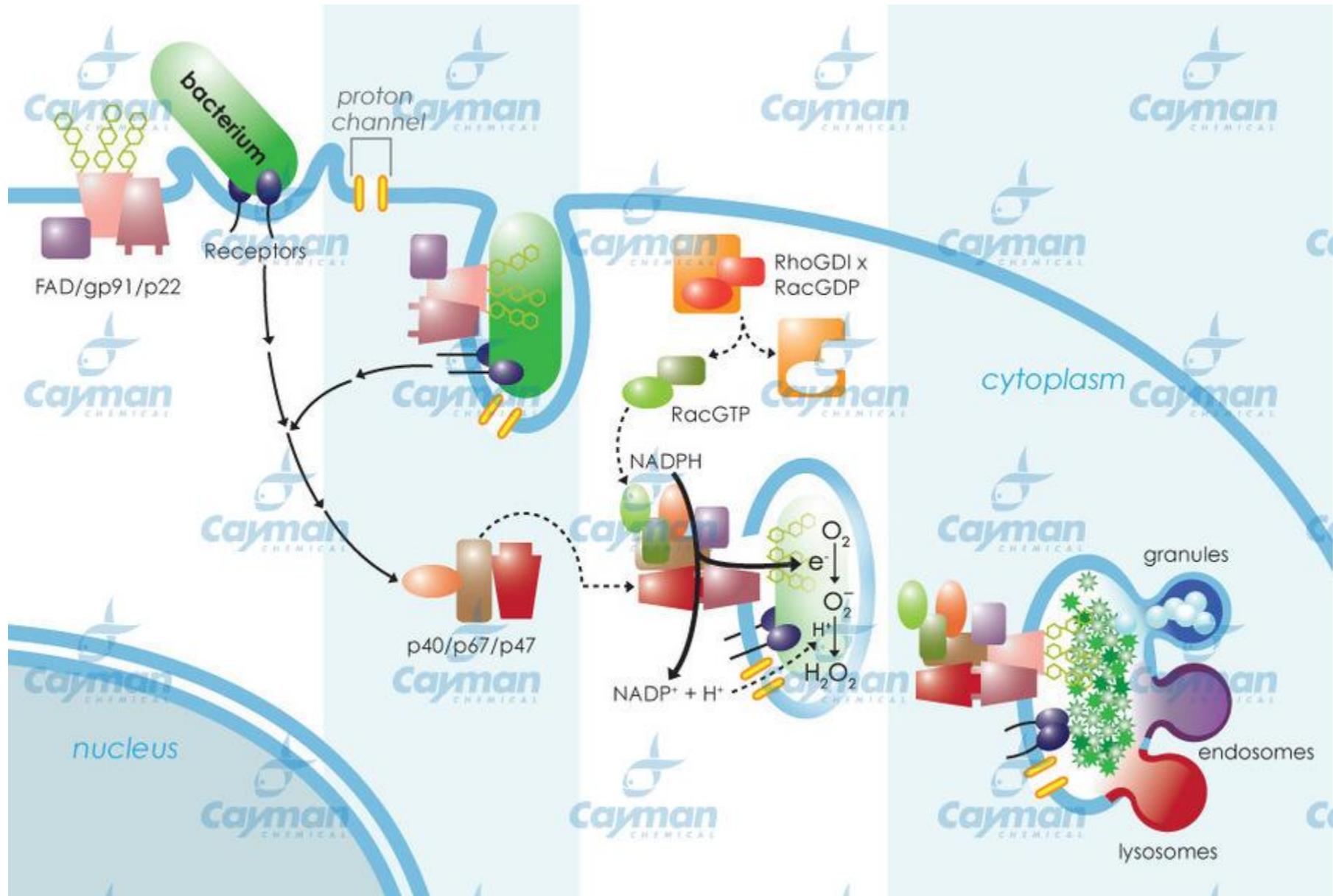
Oligoglucans action mechanism in plants



Model for St RBOHB Regulation by CDPK.

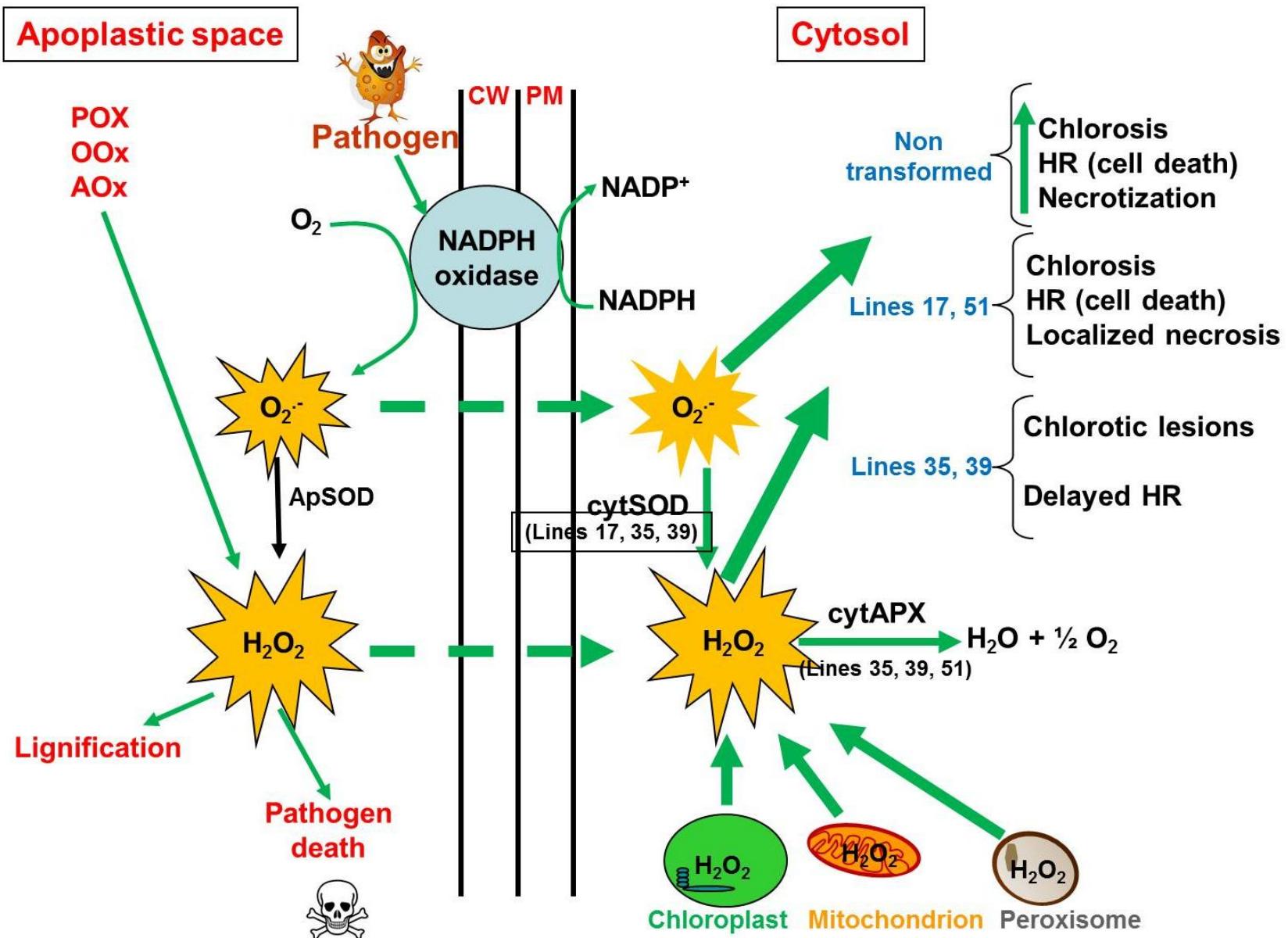
The elicitor induces Ca^{2+} influx. Increase of intracellular Ca^{2+} concentration provokes Ca^{2+} binding to EF-hand motifs of **CDPK** (calcium-dependent protein kinases) and the **RBOH** (Respiratory Burst Oxidase Homolog) N-terminal region. Phosphorylation of St RBOHB by CDPK results in ROS production.



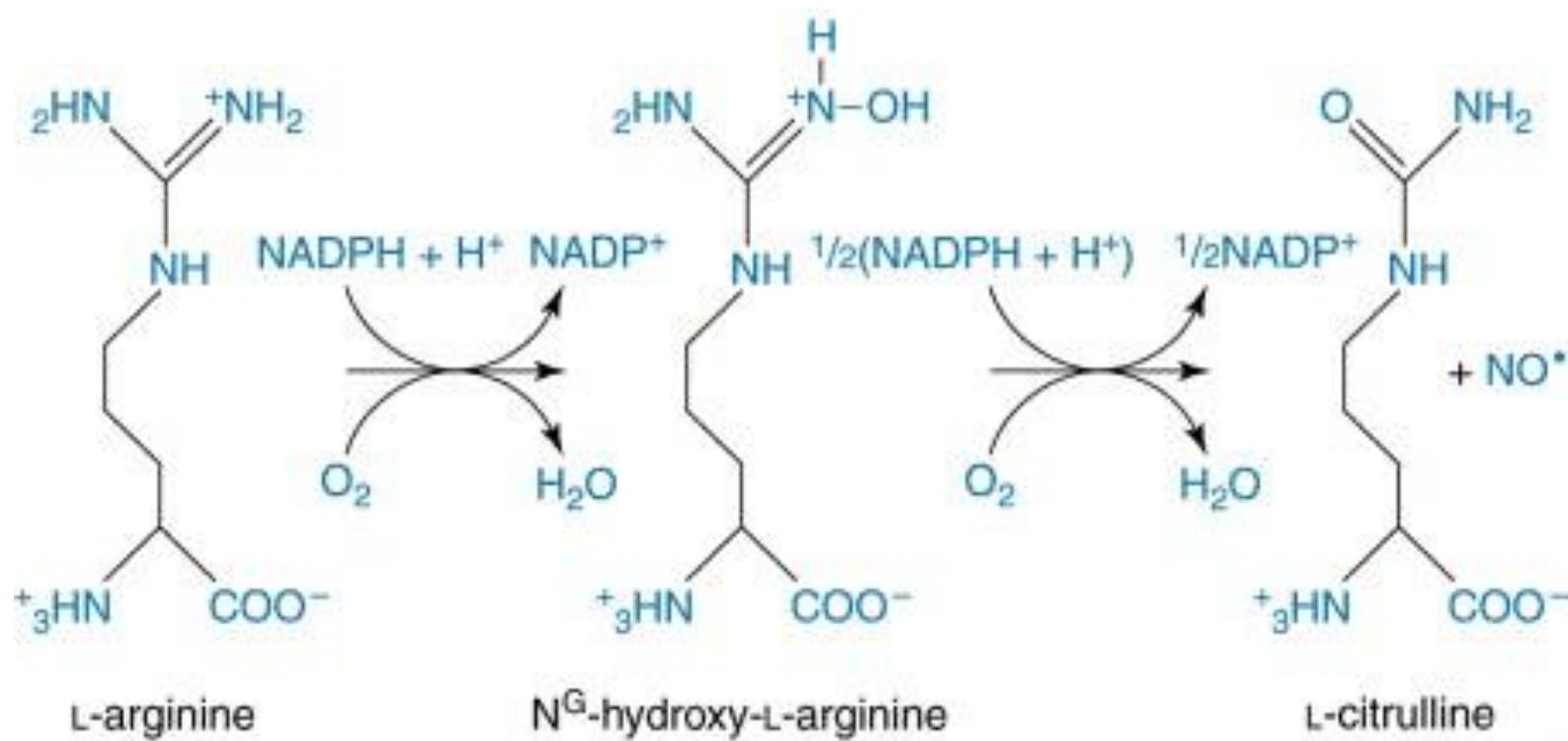


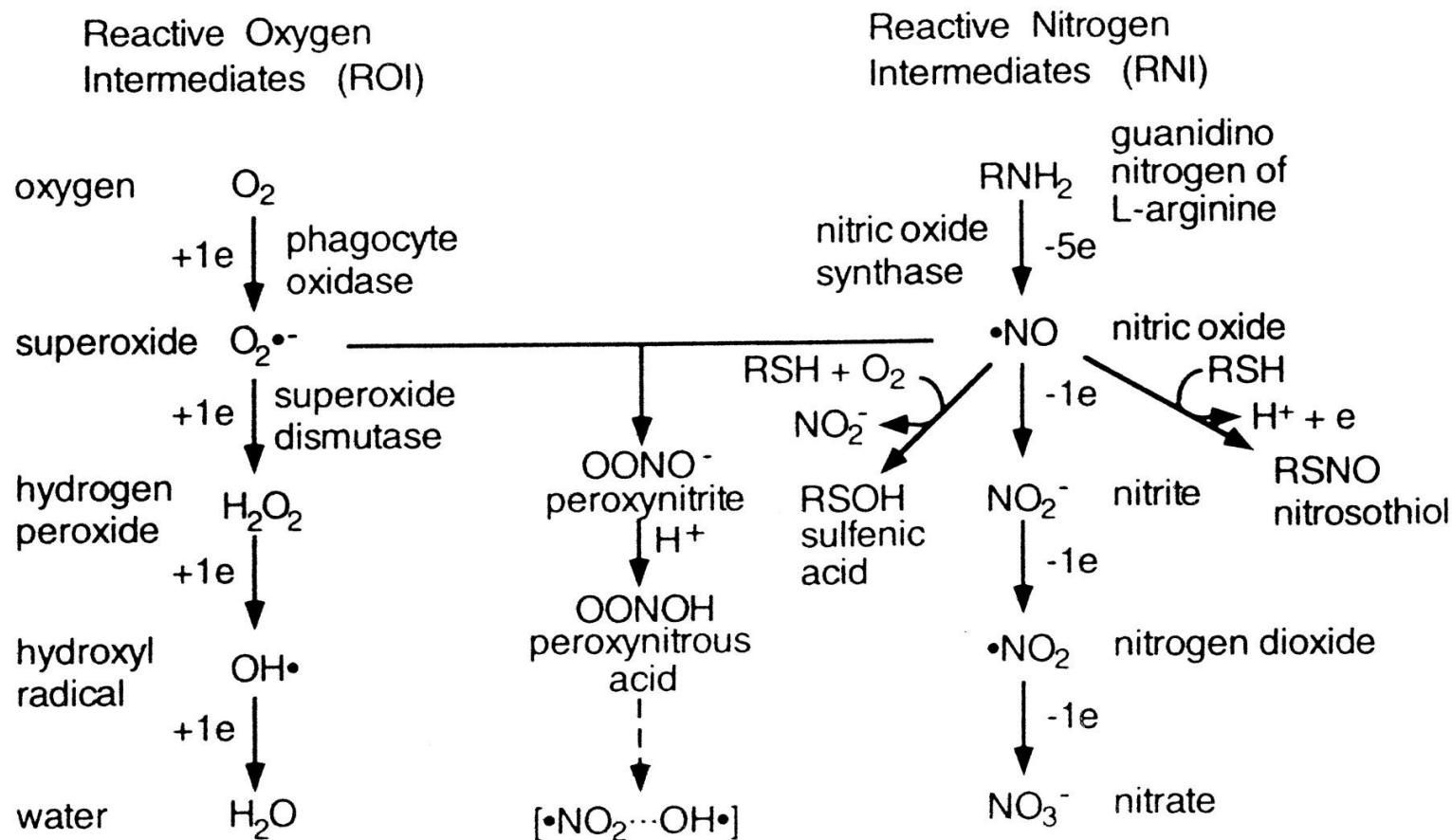
Pathogen-induced ROS generation in the apoplastic space (Lamb & Dixon 1997)

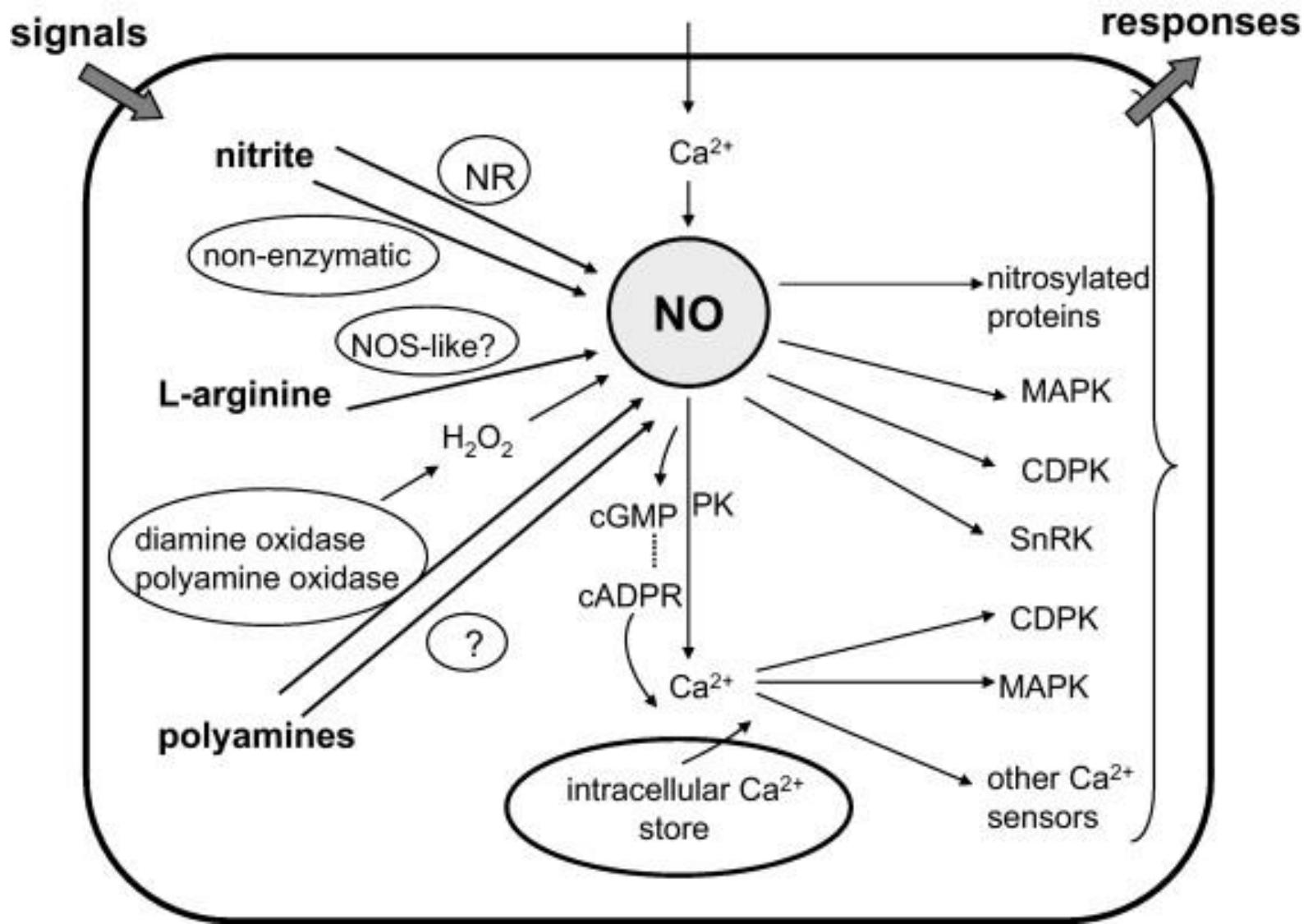
HR-hypersensitive reaction, cytSOD - cytosolic Cu,Zn - superoxide dismutase, cytAPx - ascorbate peroxidase



The reaction catalyzed by mammalian nitric oxide synthases (NOSs)

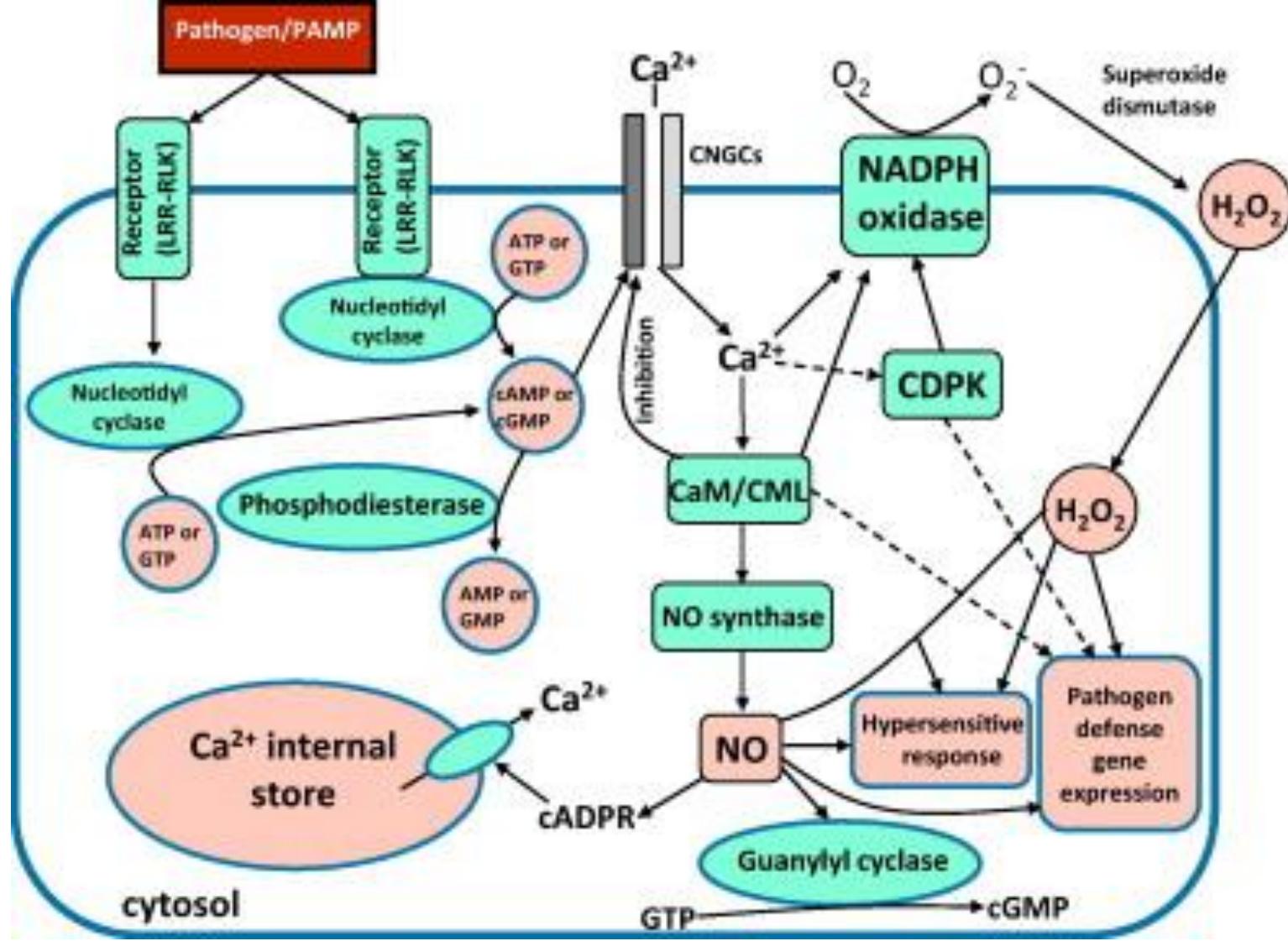






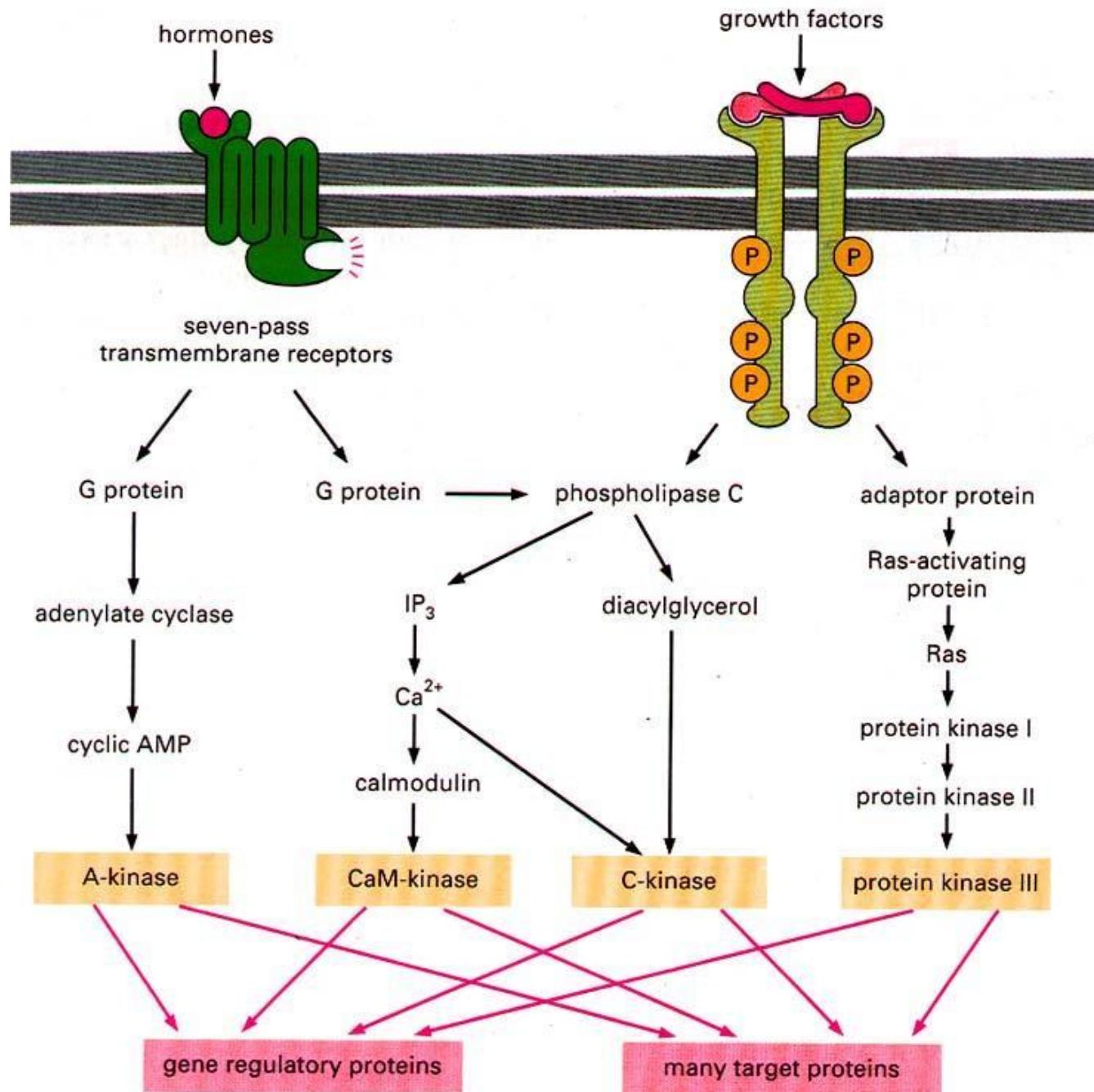
Schematic representation of NO signalling in plant cells.

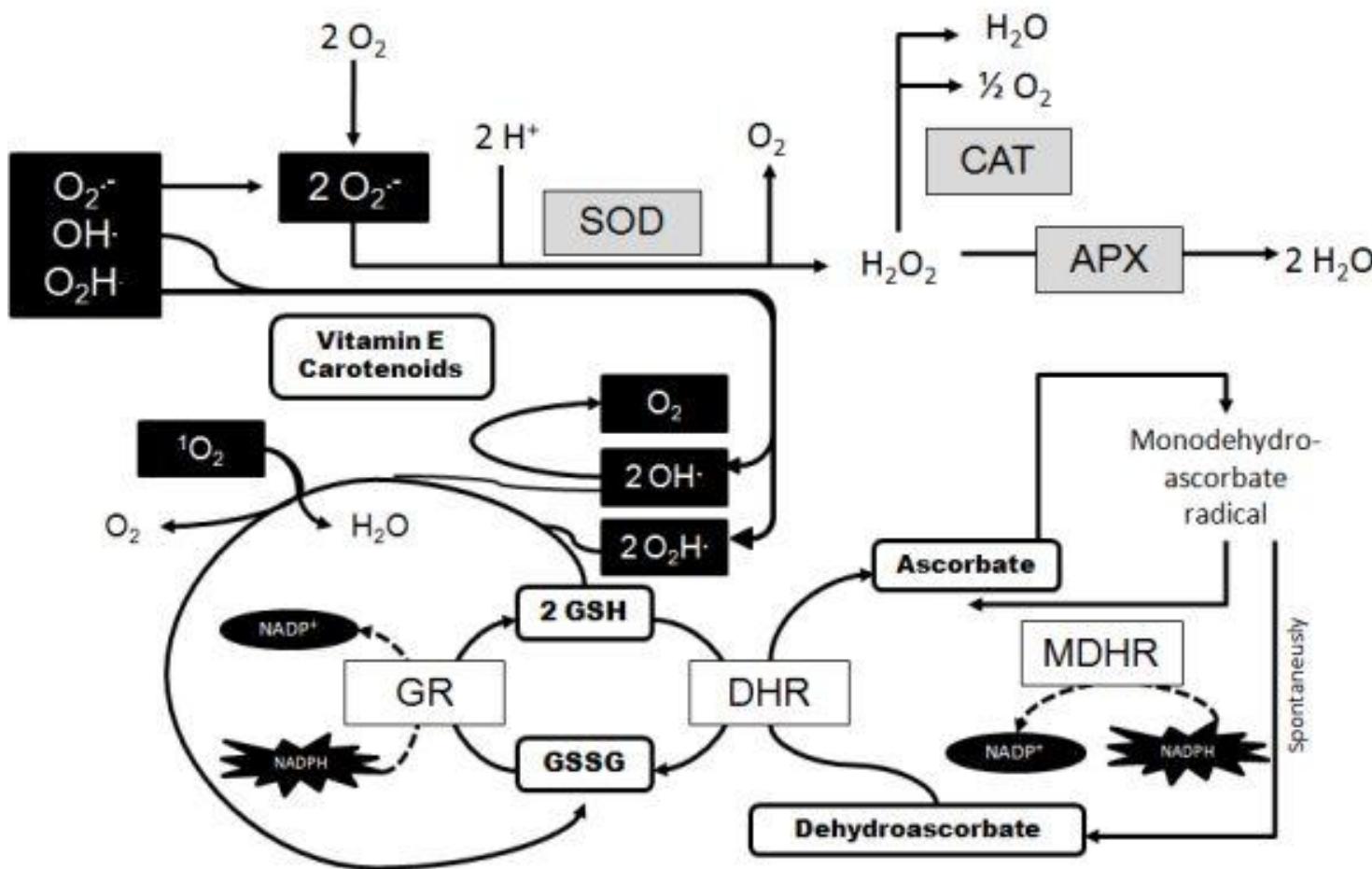
Nitrate reductase (NR), nitric oxide synthase-like enzyme (NOS-like), polyamines (PAs) DAO and PAOoxidise Pas, cyclic ADP ribose (cADPR), cyclic GMP (cGMP), protein kinases (PK), Ca²⁺ dependent protein kinases (CDPKs), mitogen activated protein kinases (MAPKs)



Model of possible early events in the plant immune response signaling cascade.

Pathogen/PAMP is recognized by a receptor which leads to an increase of cyclic nucleotide in cytosol. The rise of cyclic nucleotide leads to the activation possibly heteromeric cyclic nucleotide gated ion channels (CNGCs), resulting in Ca²⁺ influx. Cytosolic Ca²⁺ elevation results in increased amount of Ca²⁺ complexed with calmodulin (CaM) (or CaM-like protein (CML)), which leads to nitric oxide (NO) and H₂O₂ synthesis. NO and H₂O₂ are vital for hypersensitive response (HR) development. Cytosolic Ca²⁺/CaM increase competes with cyclic nucleotide and inhibits the further Ca²⁺ influx through CNGC. Arrows imply activation in all cases unless the notation 'inhibition' is shown. Some arrows are shown in broken lines for clarity.

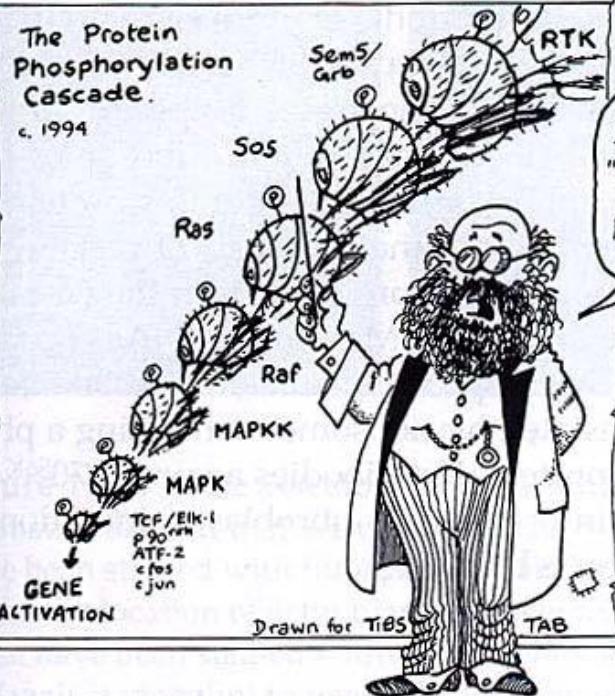




Enzymatic and non-enzymatic antioxidant system in plants.

Superoxide dismutase (SOD), catalase (CAT) and ascorbate peroxidase (APX) are the proteins responsible for eliminating ROS. While the elimination of ROS by non-enzymatic processes is carried out by vitamin E, carotenoids, ascorbate, oxidized glutathione (GSH) and reduced (GSSG). Enzymes that promote the elimination of ROS via the ascorbate-glutathione cycle are monodehydroascorbate reductase (MDHR), dehydroascorbate reductase (DHR) and glutathione reductase (GR) (Modified from [Halliwell, 2006](#)).

The Protein
Phosphorylation
Cascade.
c. 1994

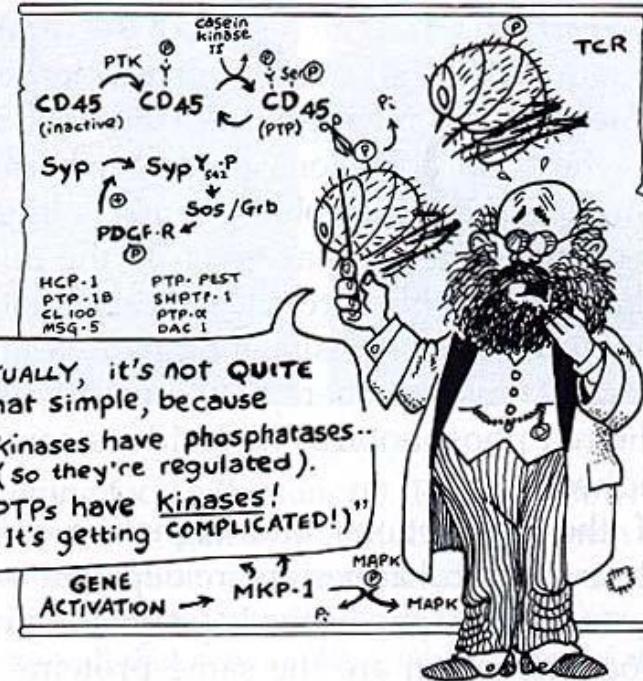


OK, CLASS!
Pay attention!
It's quite simple!
"Kinases have kinases
upon their backs to bite 'em!
Kinase Kinases have kinases--
and so-- ad infinitum?!"

GENE ACTIVATION

Drawn for TIBS

TAB



Er - ACTUALLY, it's not QUITE
that simple, because
"Some kinases have phosphatases--
(so they're regulated).
And PTPs have kinases!
(It's getting COMPLICATED!)"

GENE
ACTIVATION → MKP-1 → MAPK

This is the fourth one we've
brought in like this since the
TIBS Special Issue on Protein
Phosphorylation, George!
Do you think there might be a
link?!



"And phosphotyrosines will bind
to SH-2 domains!
Whilst proline strings bind SH-3!
... and round we go again.
Some activated proteins shift
from cytosol to membrane,
Whilst some enter the nucleus--
(I've got a pain in my brain!)"

