PMP

2025

7) Physiology and signaling of plant hormones

a) Auxins

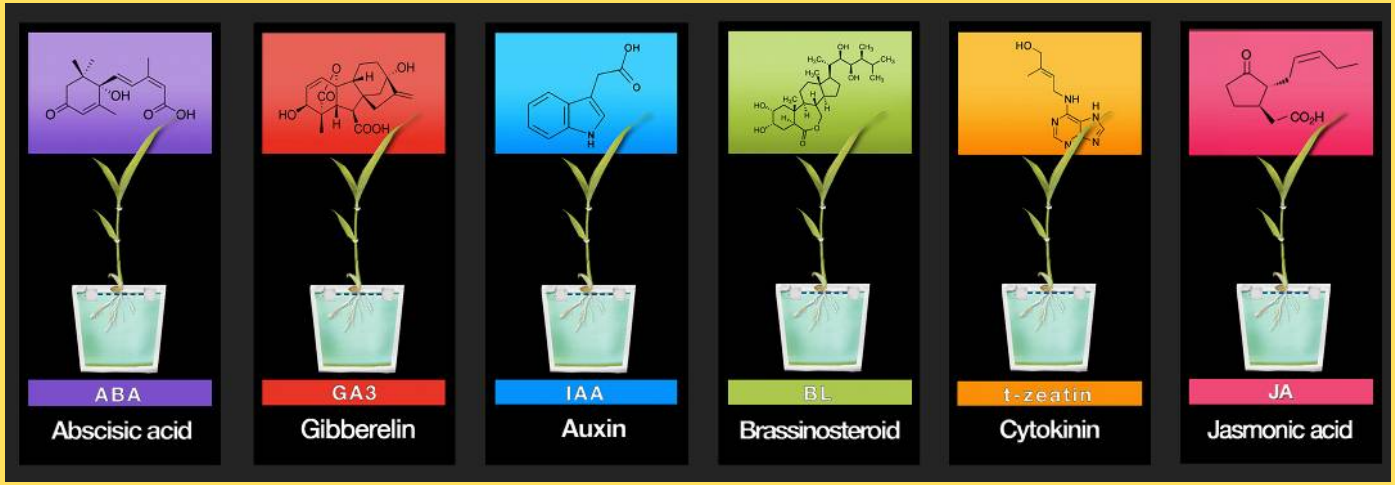
b) Brassinosteroids

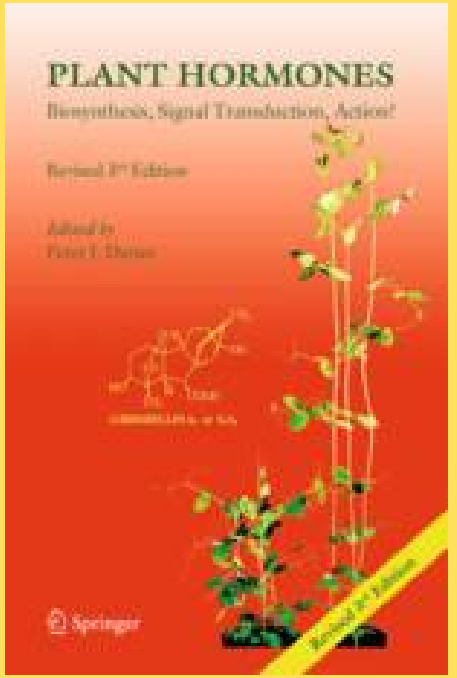
c) Ethylene

d) Abscisic acid (ABA)

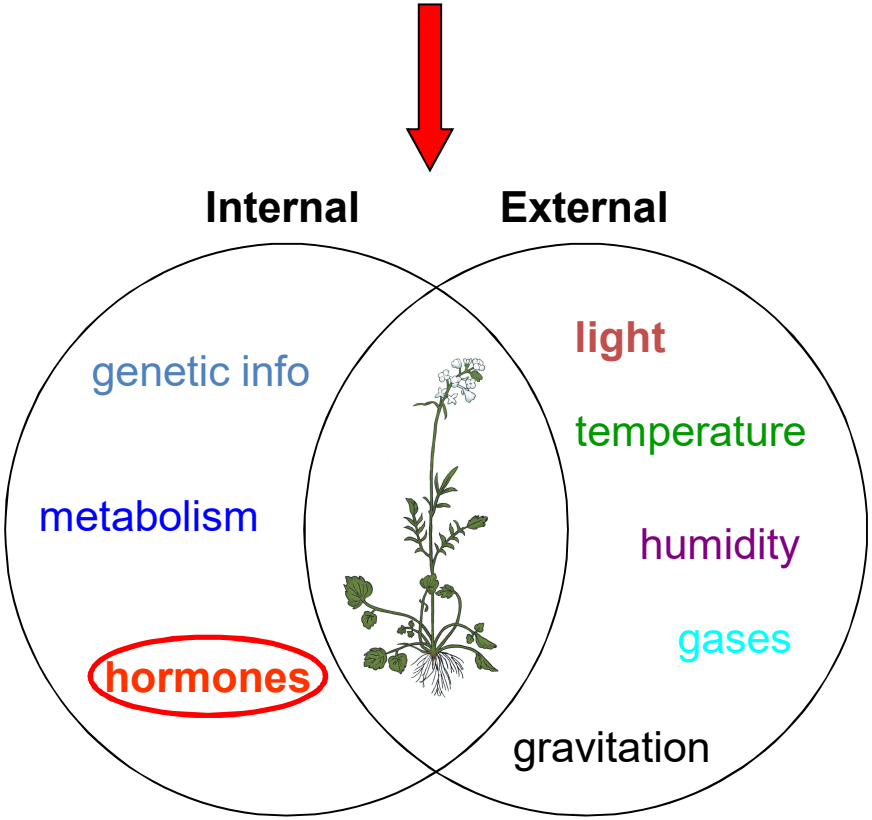
e) Salicylic acid (SA)

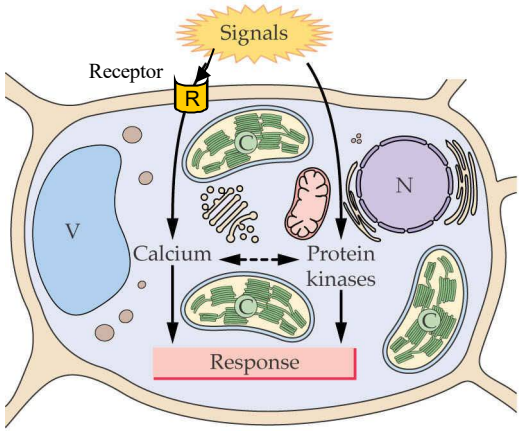
f) Jasmonic acid (JA)





Development of organism is regulated by signals





Signal

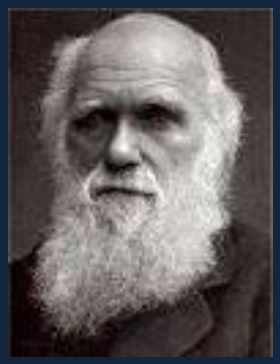
Receptor

Secondary messenger

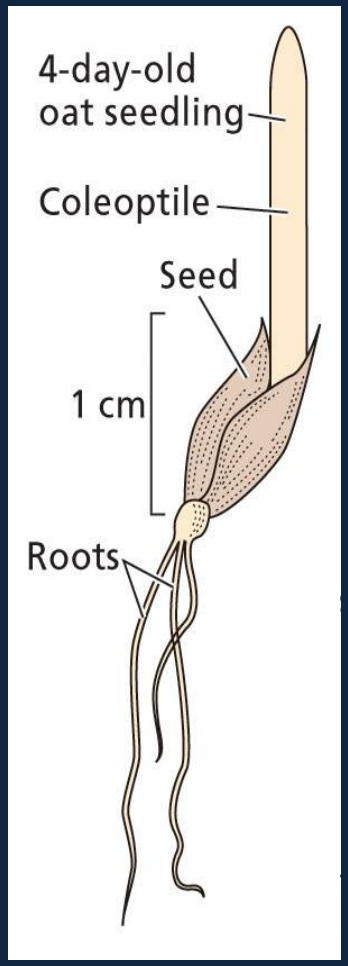
Response
(e.g. gene expression)

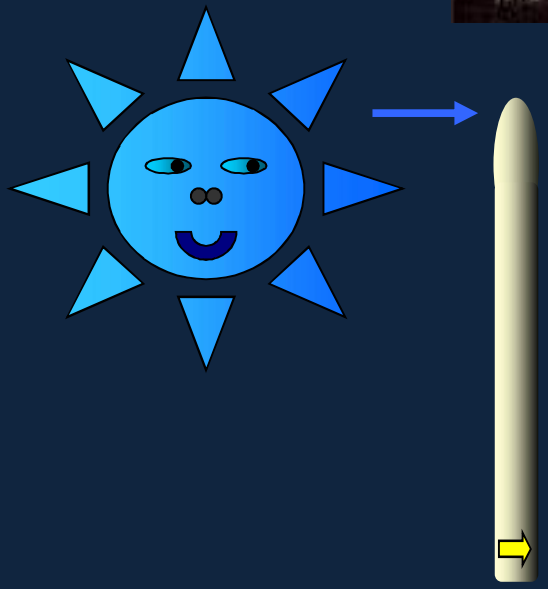
a) Auxins

Phototropism (1880)



Ch. Darwin





Auxin gradient

~ 180 minutes

Coleoptile bending

Auxin stimulates cell expansion more in the shaded side than in the lighted side of the coleoptile => asymmetric growth and bending.

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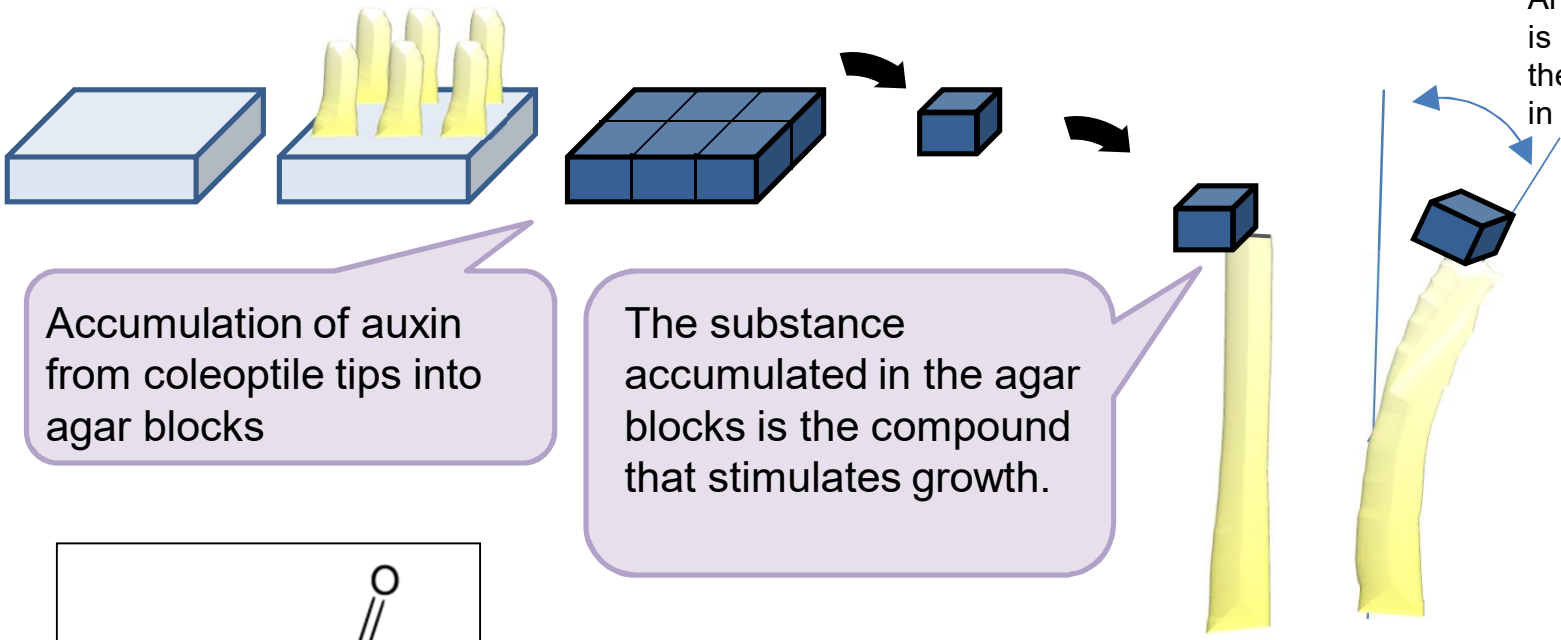
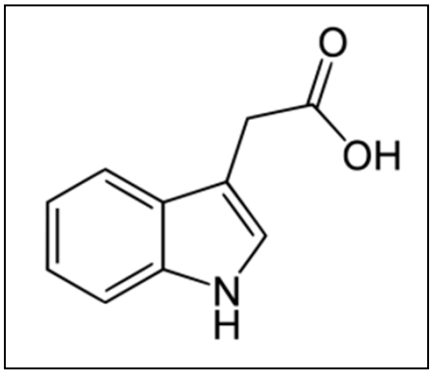
In 30th auxin has been isolated and it was shown that it stimulates growth.





IAA - Indole-3-acetic acid

This experiment showing auxin-induced growth stimulation was used as a basis for auxin purification.

Redrawn from Went, F.W. (1935) Auxin, the plant growth-hormone. Bot. Rev. 1: 162-182.

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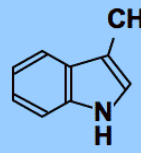
Auxins – important plant hormones involved in spread spectrum of growth and developmental processes.

Naturally Occurring Auxins

indole-3-acetic acid

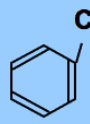


IAA

4-chloroindole-3-acetic acid

4-Cl-IAA

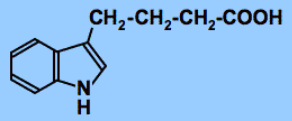
phenylacetic acid



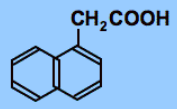
PAA

Commonly Used Synthetic Auxins

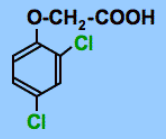
indole-3-butyric acid



naphthalene acetic acid



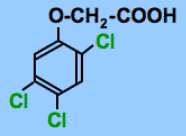
2,4-dichlorophenoxy-acetic acid



2-methoxy-3,6-dichlorobenzoic acid

4-amino-3,5,6-trichloropicolinic acid

2,4,5-trichlorophenoxy-acetic acid



- embryogenesis
- stem elongation
- apical dominance
- photo- and gravitropism
- lateral root formation

Cellular level:

- cell division
- cell expansion
- cell differentiation

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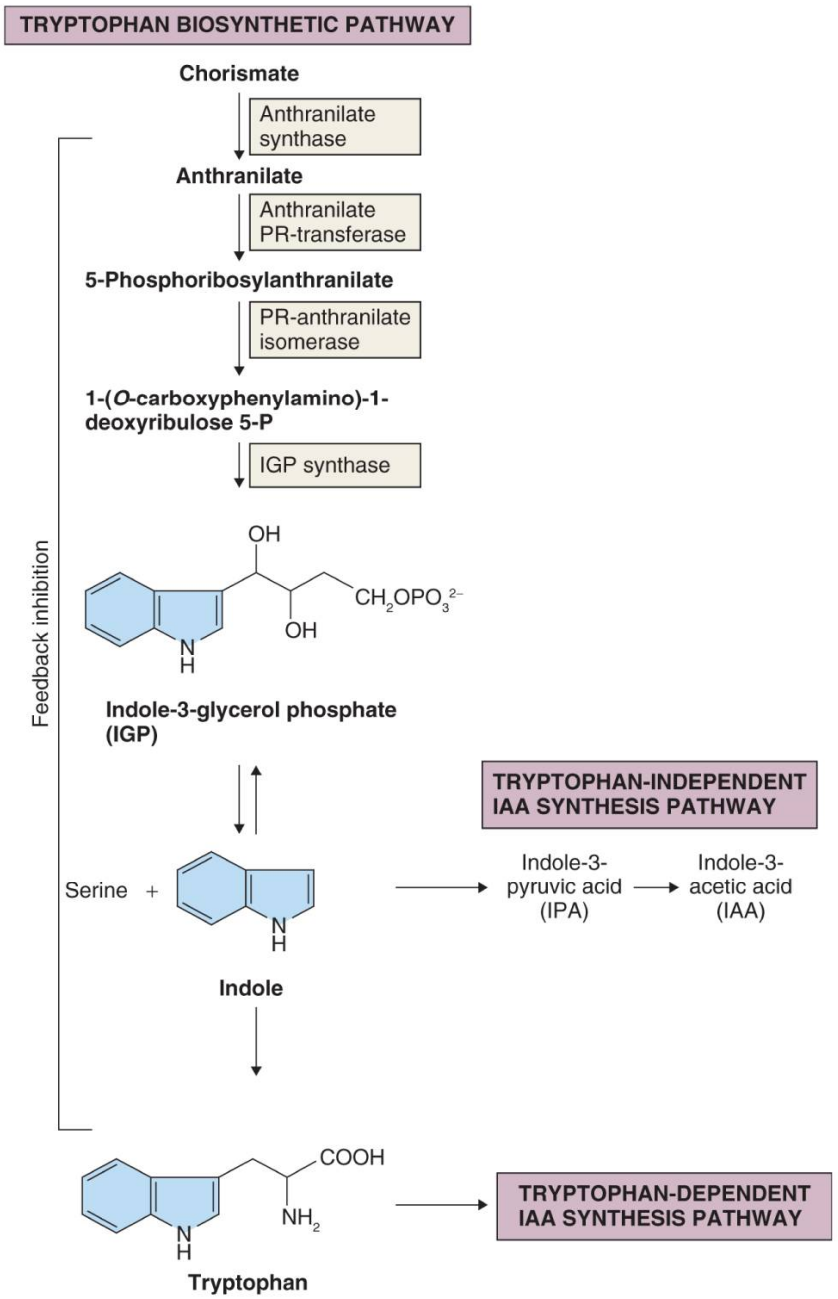
IAA biosynthesis

- Indole pathway
- Tryptophan pathway

Young developing tissues and meristems are main place of auxin biosynthesis.



Conjugates

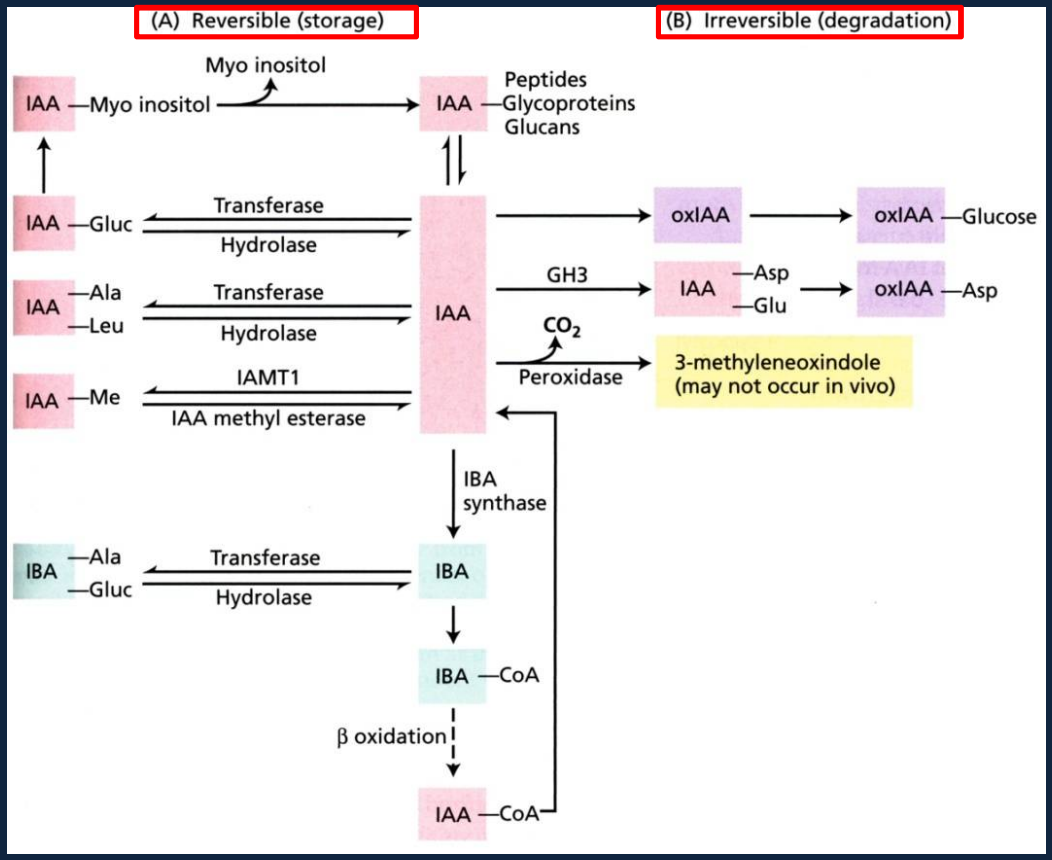
Covalently bound IAA (seeds and storage organs) – untransportable inactive forms of auxin

(A) Reversible

IAA-myo-inositol, IAA-glucosa, IAA-AMK, methylester-IAA

(B) Irreversible

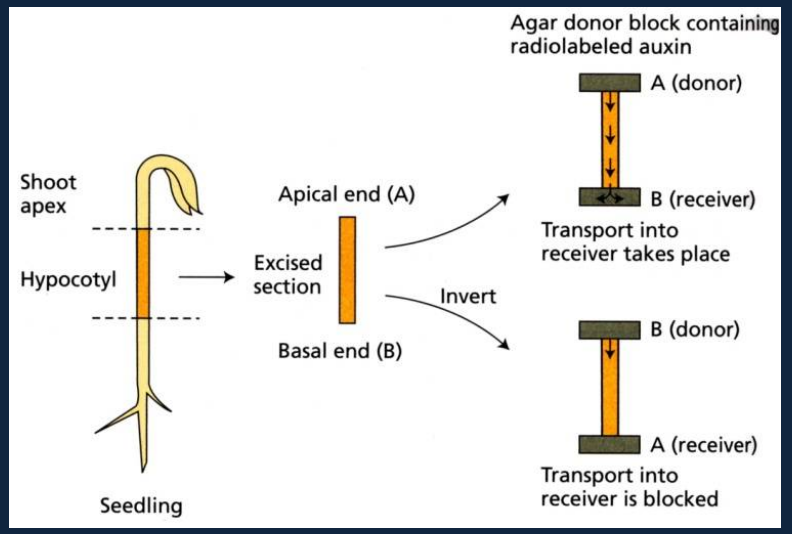
IAA-aspartate, IAA-glutamate



Degradation: oxidation of IAA (substrate – IAA and irreversible conjugates)

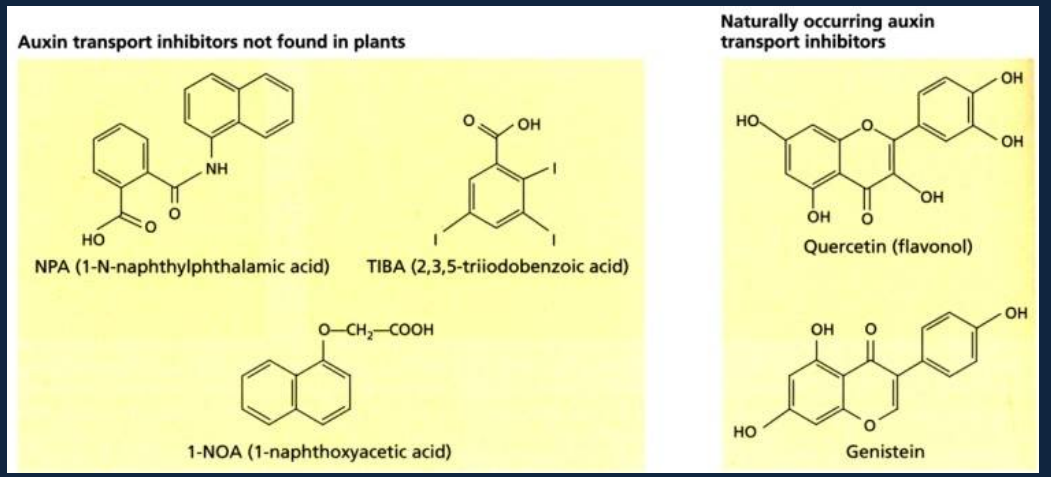
Transport of auxin

- Polar (basipetal) transport from apex, coleoptile; in roots acropetal transport predominates (is not affected by orientation)
- Apoplastic transport, xylem and phloem transport



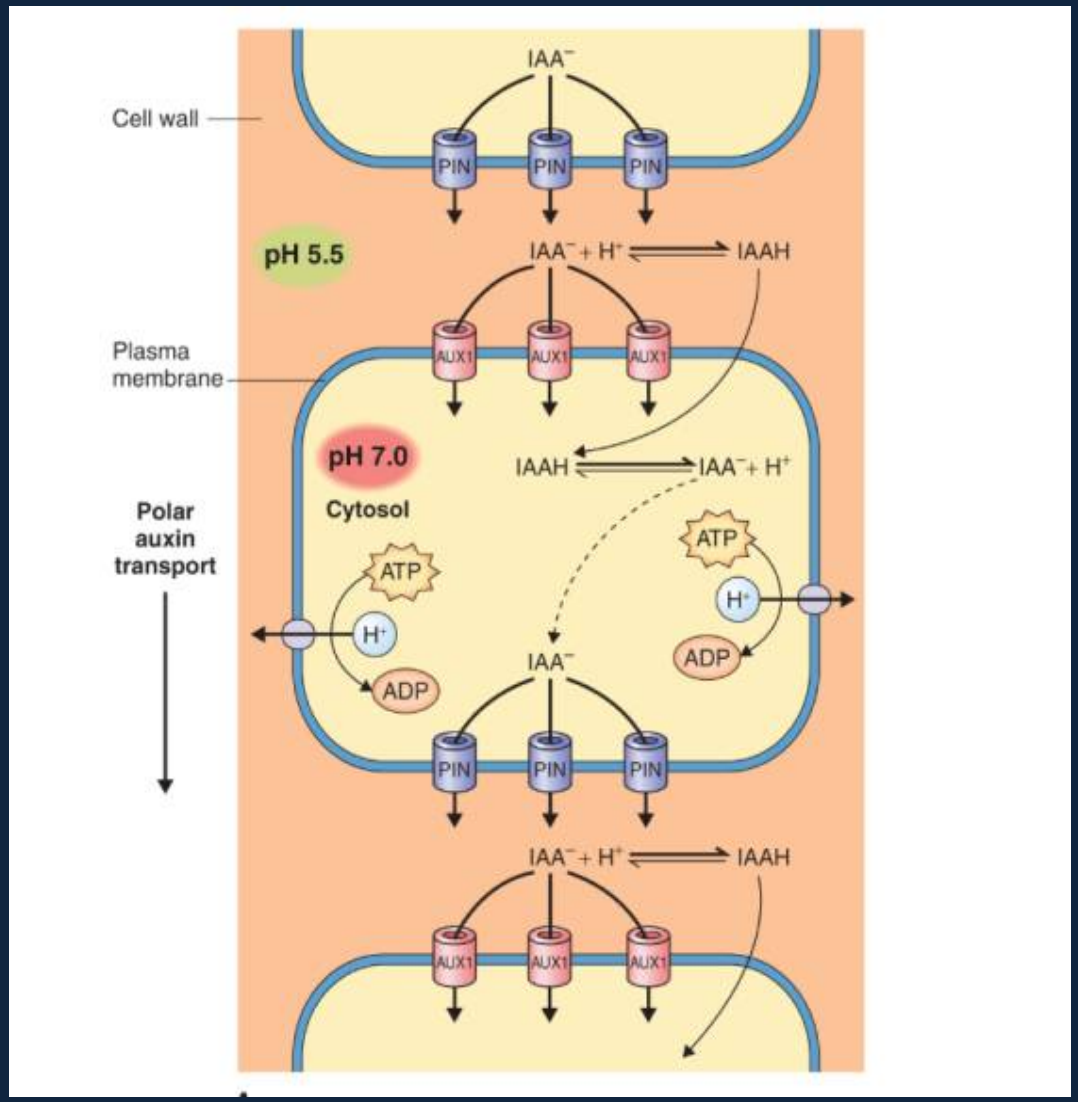
Transport inhibitors

NPA: 1-N-Naphthylphthalamic acid
TIBA: 2,3,5-triiodobenzoic acid
1-NOA: 1-naphthoxyacetic acid
quercetin, genistein



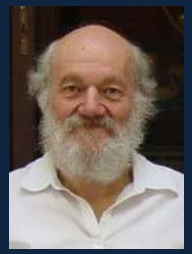
Influx

- Passive diffusion of IAAH (lipophilic)
- Active permease - AUX1 carrier (symport $2H^+/IAA^-$)



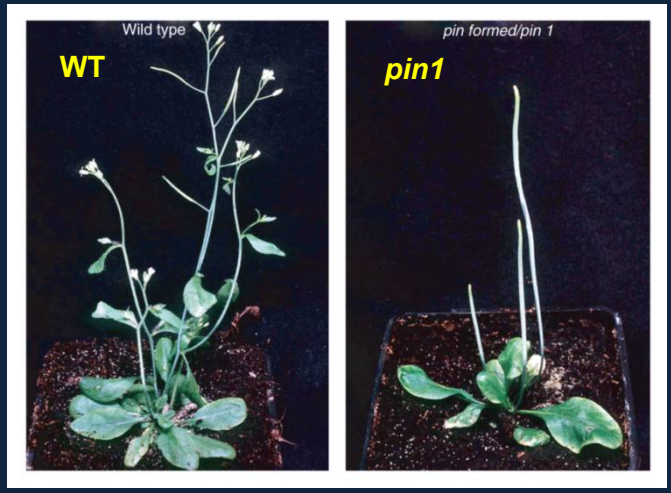
Efflux

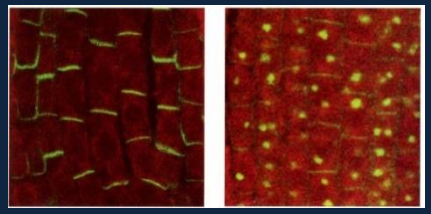
- Active - carrier: PIN proteins, P-glycoproteins (ATP-dependent carrier)



John Raven

Mary Helen Goldsmith

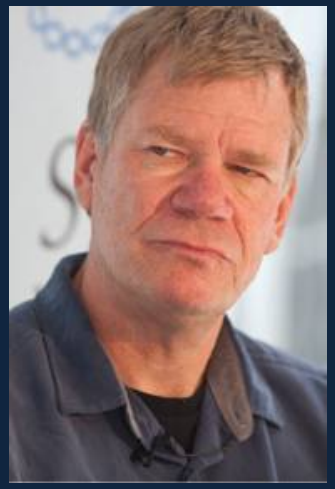




Accumulation of PIN1 on plasma membrane

Accumulation of PIN1 in nucleus

Auxin signaling



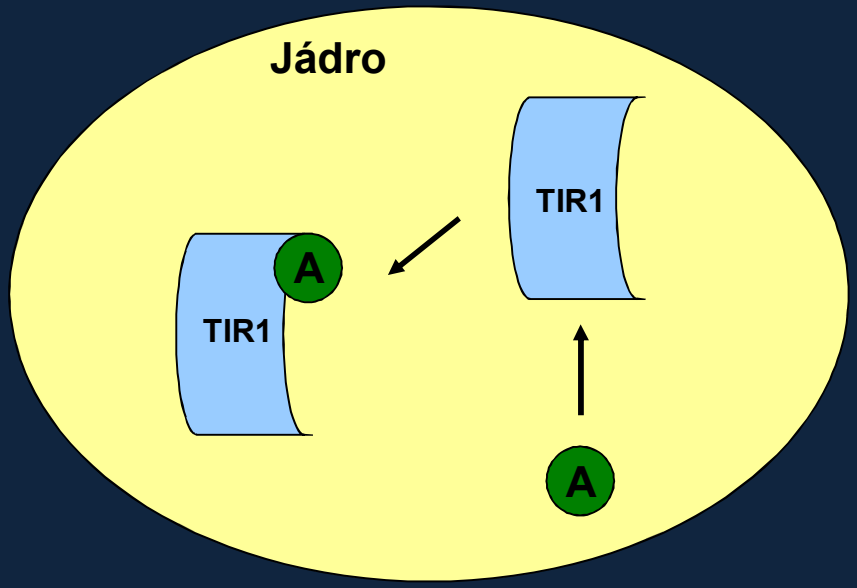
Auxin receptor TIR1

Intracellular receptor TIR1 (Transport Inhibitor Response 1) in *Arabidopsis*

Kepinski and Leyser 2005

Dharmasiri *et al*. 2005

Auxin binds directly to TIR1 in nucleus



1) First intracellular auxin receptor

2) Receptor mediates transcriptional responses to auxin

A – auxin

TIR1 – F-box subunit of E3-ubiquitin ligase; auxin receptor

AUX/IAA – repressor of transcription of auxin-induced genes

ARF – transcription factor (expression activator)

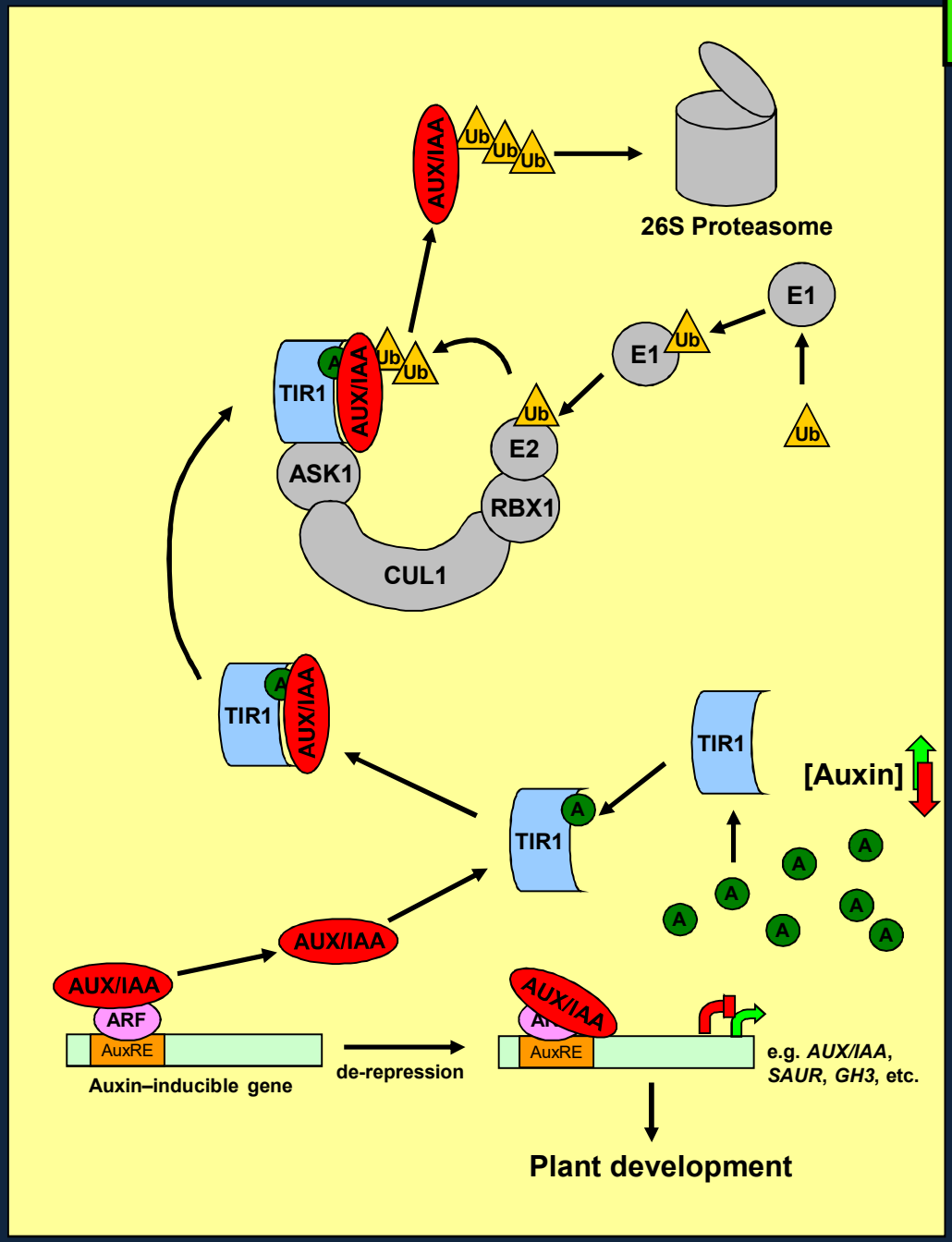
AuxRE – auxin-responsive element

Ub Ubiquitin

E1 Ubiquitin-activating enzyme

E2 Ubiquitin-conjugation enzyme

Auxin functions as „a glue“ enabling connection of TIR1 with substrates.



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Signaling pathway through the receptor TIR1 is not the only auxin signaling pathway.

Homologs TIR1: AFB1, AFB2, AFB3 – the same functions as TIR1

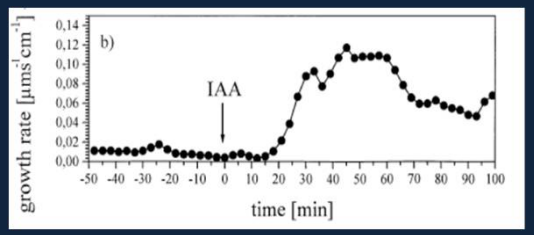
However: 1) Quadruple mutant – functional plant → ReceptorsTIR1 and AFB are not essential

Dharmasiri *et al.* 2005b

2)

Auxin induces cell elongation with lag phase 8-15 min => rapid response excludes involvement of TIR1, i.e. gene expression and protein synthesis

Auxin functions via signaling pathway involving another receptor → Receptor ABP1



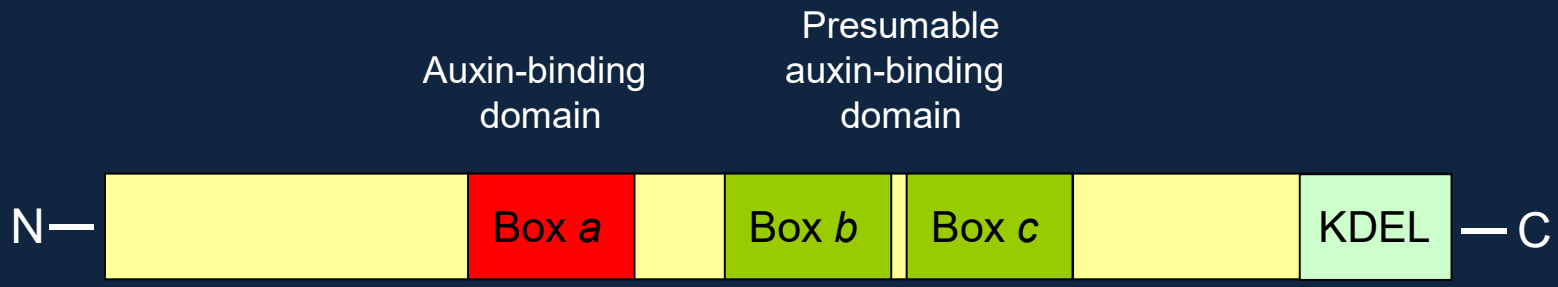
Steffens and Lüthen 2000

Auxin receptor ABP1

1972 – identification of ABP1 (Auxin-Binding Protein 1) in isolated membranes of maize coleoptile cells; binding of radioactive auxin in the membrane

1985 – isolation of protein ABP1 in maize; 22 kD

End of 80th – cloning and structure of ABP1



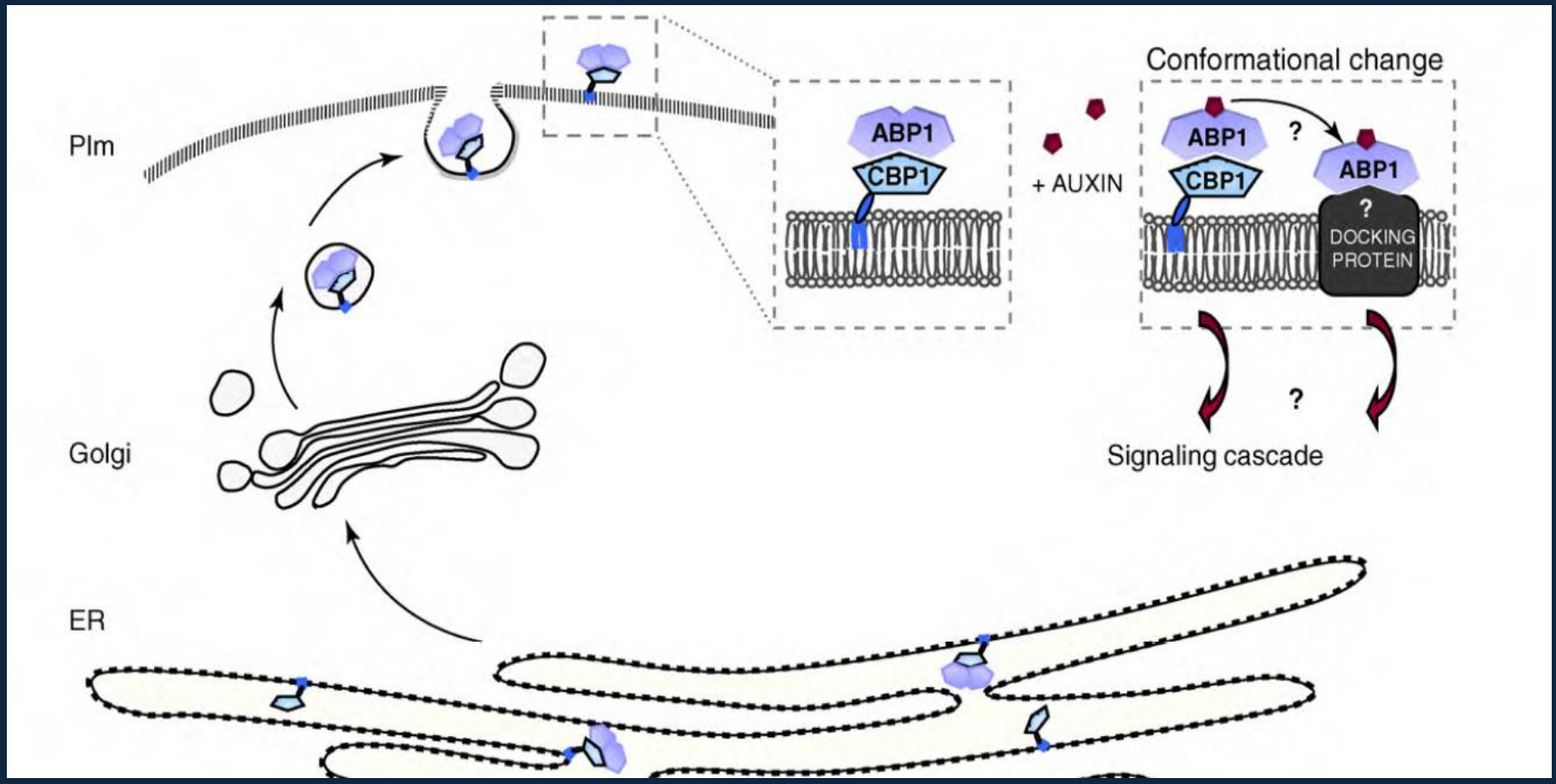
Auxin binds to plasma membrane fraction.

BUT ?

Protein ABP1 has not transmembrane domain, but has s KDEL domain.

Transport of ABP1 from ER and „Docking“ model

Transport of ABP1 from endoplasmic reticulum to apoplast and binding to a docking protein.

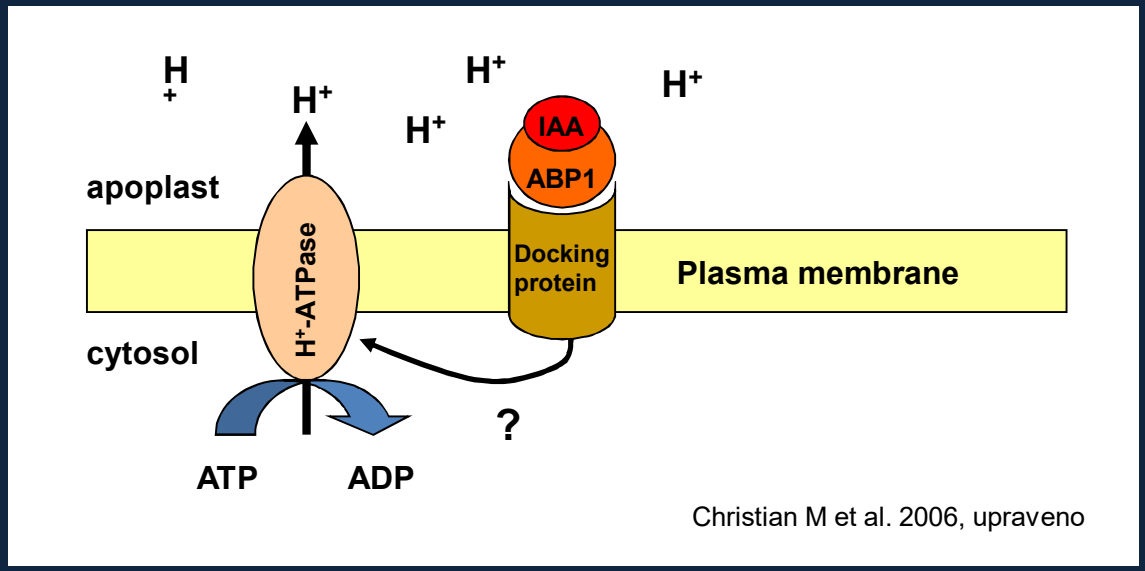


Acid growth theory, proton pump and K^+ channels

Rayle D and Cleland R (1970)

Auxin → Excretion of H^+ to apoplast → Lowering of pH in apoplast

Activation of enzymes (expansins) loosening the cell wall



Christian M et al. 2006, upraveno

Binding of auxin to ABP1

↓

Stimulation of H^+-ATPase

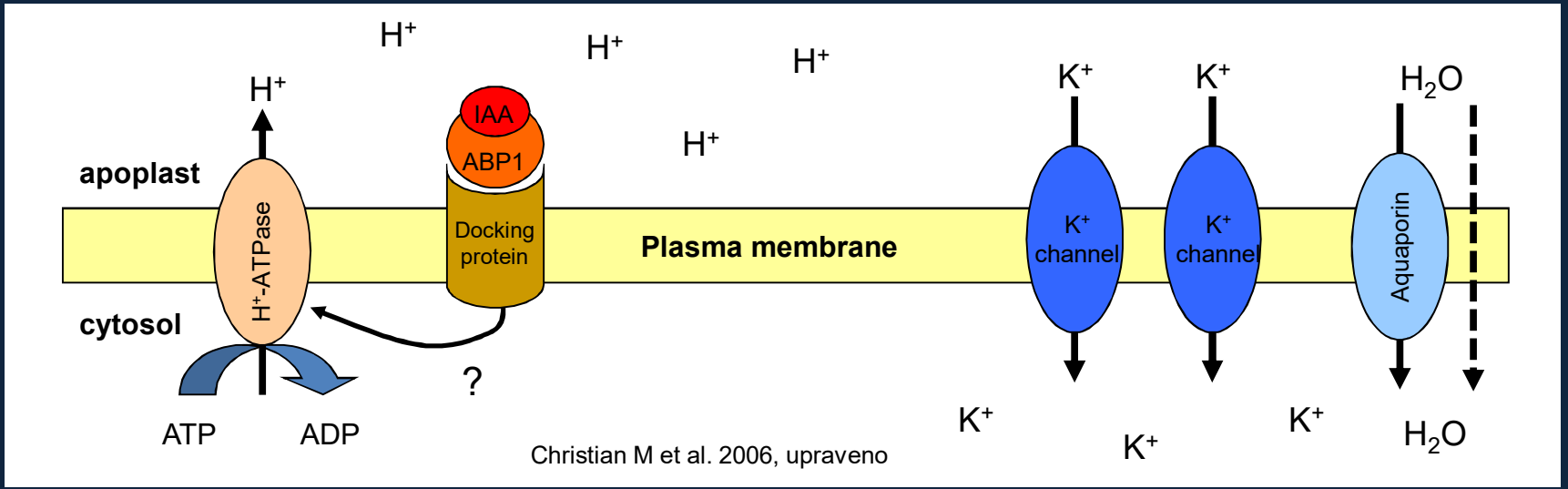
↓

Plasma membrane hyperpolarization
Apoplast acidification

The conditions for growth is a turgor. But auxin itself does not increase turgor.

Accumulation of H^+ in apoplast → Compensation of charge in cytosol

Inward-rectifying K^+ channels

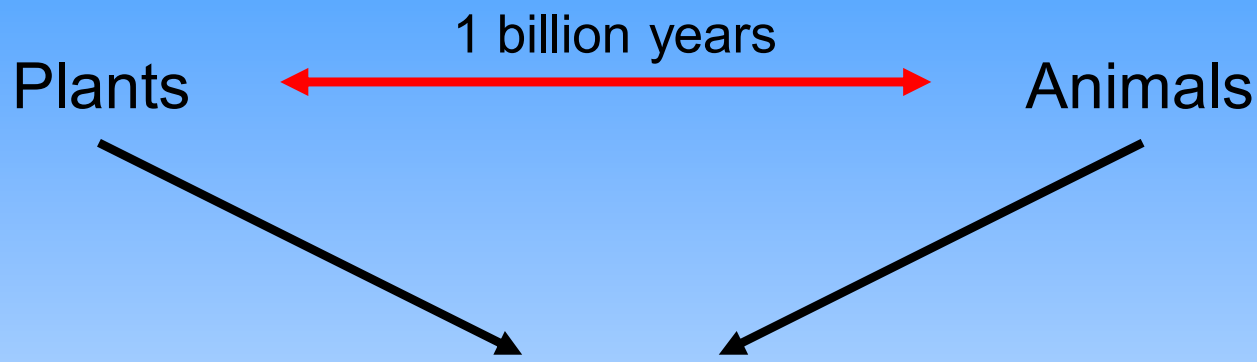


Christian M et al. 2006, upraveno

K^+ accumulation in cytosol → Transport of H_20 into cell → Turgor → GROWTH

Presence of K^+ : conditions of sustained acidification and growth

b) Brassinosteroids



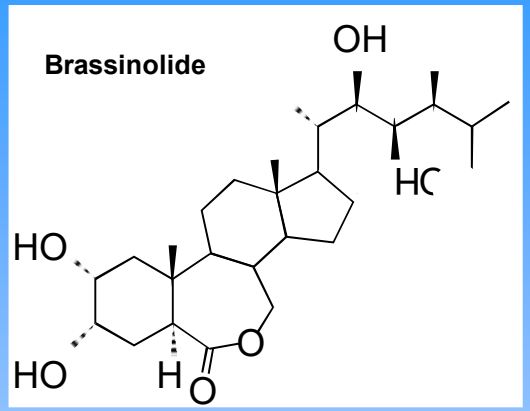
Hormones – polyhydroxylated steroid molecules

BR and steroids control:

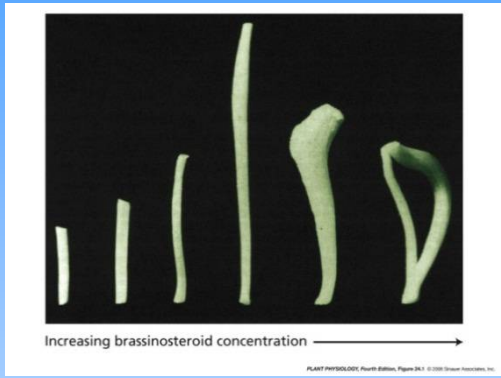
- Regulation of gene expression
- Cell division
- Cell expansion
- Cell differentiation
- PCD
- Homeostasis

1979 – brassinolide (BL) – final product of biosynthetic pathway

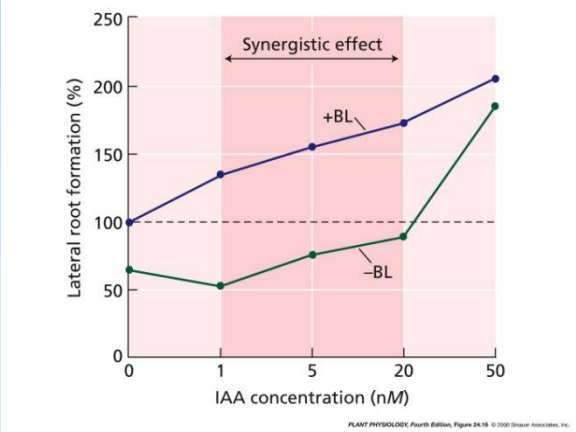
- Stimulates stem elongation
- Stimulates root elongation (low concentration)
- Inhibits root elongation (high concentration)
- Stimulates seed germination and growth of leaves



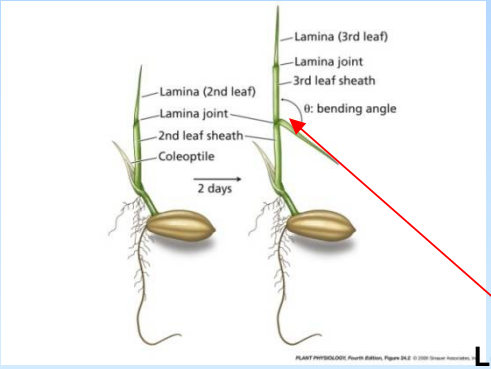
Elongation of 2nd internode of bean stem



Brassinosteroids and auxins have synergistic effects. Stimulation:



- Formation of lateral roots
- Stem elongation
- Growth of pollen tube
- Leaf angle and epinasty
- Activity of proton pump
- Xylem differentiation



Leaf angle

Auxin – fast effects Brasinolid – slow effect

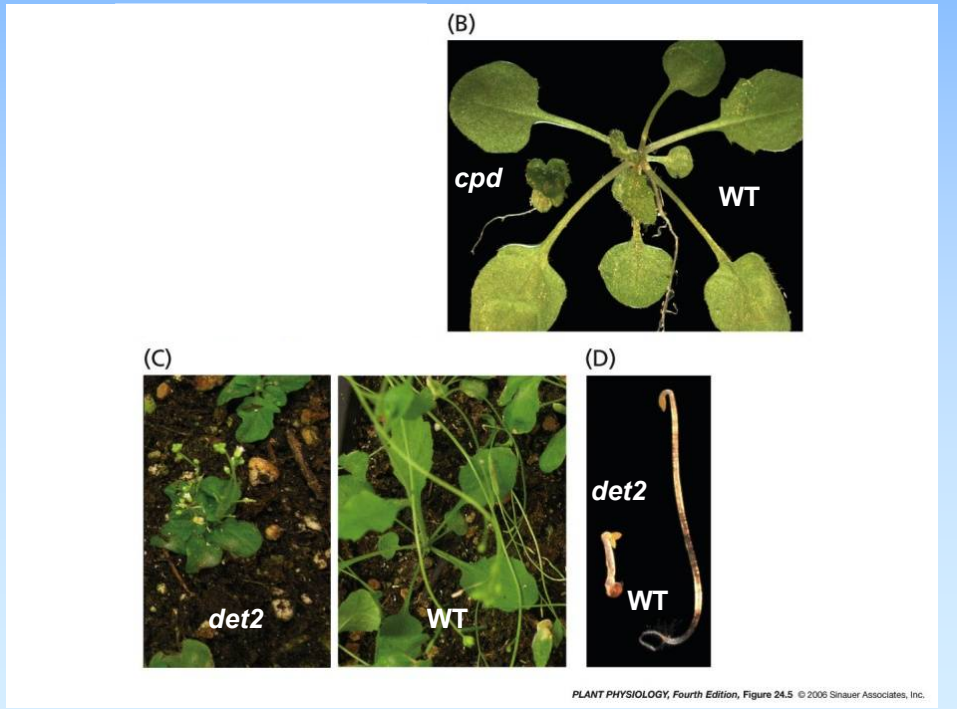
Genetic approach – physiological, biochemical and molecular characterization of mutants

Selection of brassinosteroid-deficient mutants (tomato, pea, rice, *Arabidopsis*)

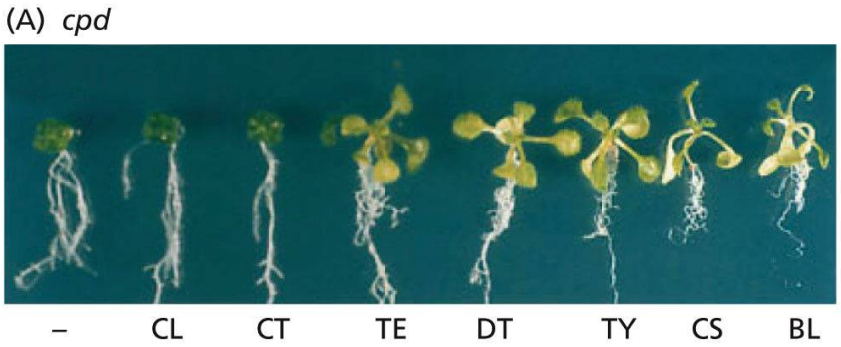
Distinct phenotype of mutants; *Arabidopsis* – pleiotropic mutation effect

Dark: short growth stature, swelled hypocotyl, open bigger cotyledons, presence of primary leaf buds

Light: dwarf, dark green, pollen sterility, delayed senescence of chloroplasts and leaves, altered responses to light



Application of exogenous BR results in development of WT phenotype.



CL - campesterol

CT - cathasterone

TE - teasterone

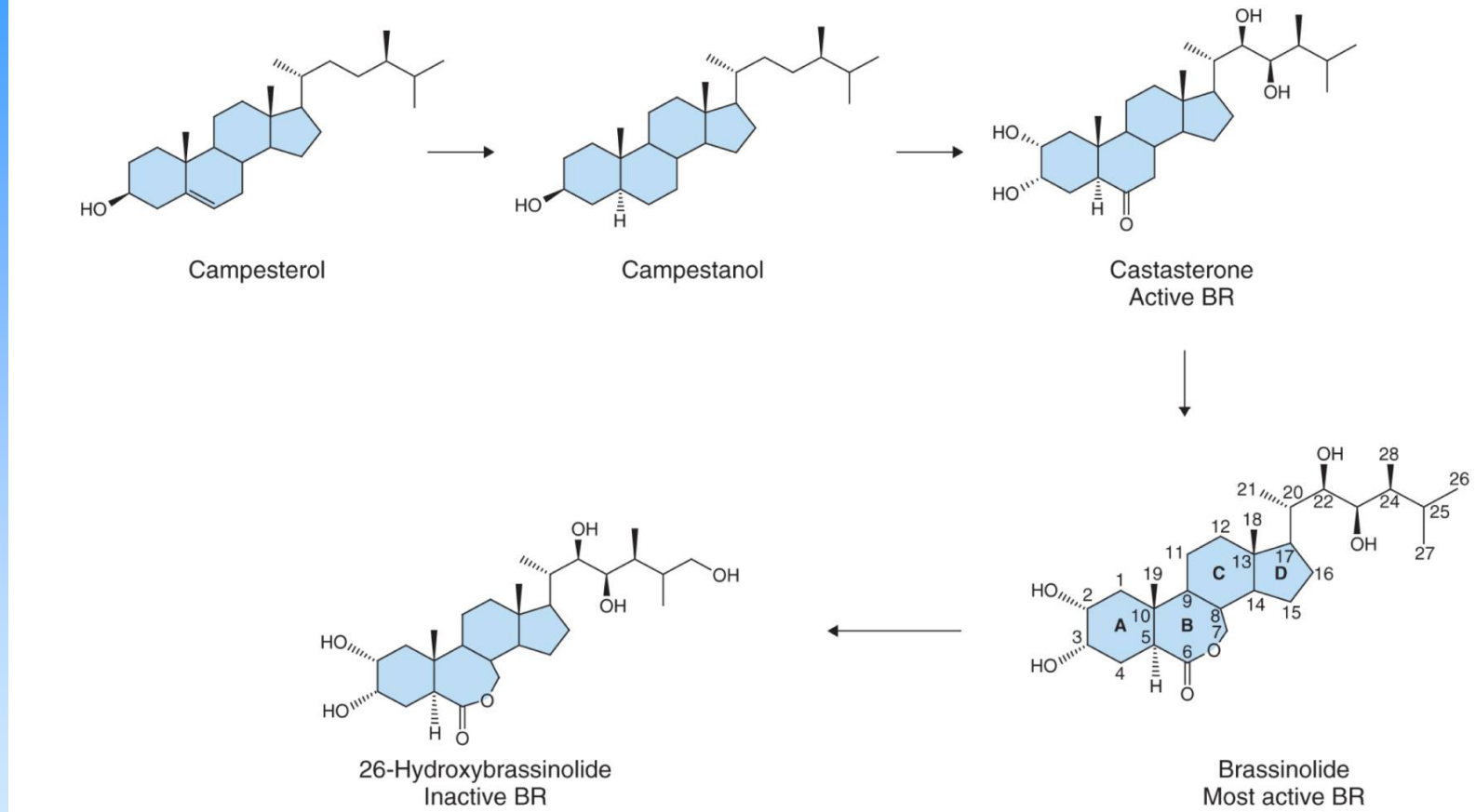
DT - dehydroteasterone

TY - typhasterol

CS - castasterone

BL - brassinolide

Biosynthesis of brassinosteroids



Likely in all organs **Two biosynthetic pathways C28 BR**

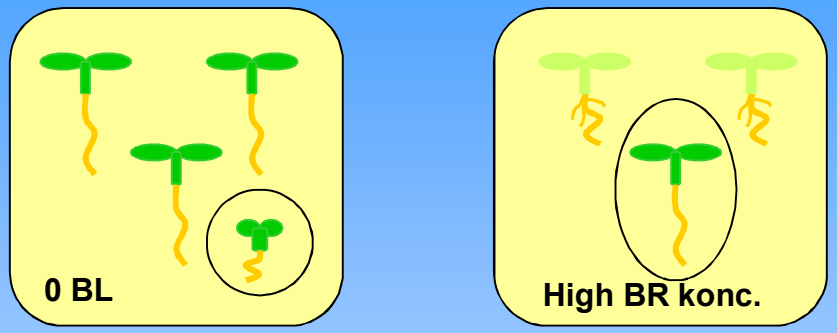
early C-6 oxidation

late C-6 oxidation

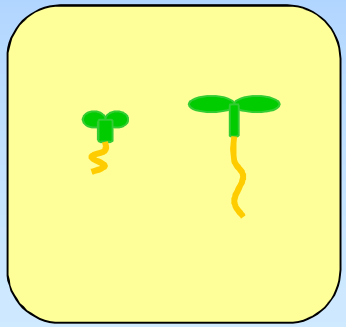
Brassinosteroid signaling

Identification of BR-insensitive mutants

Mutated population of plants

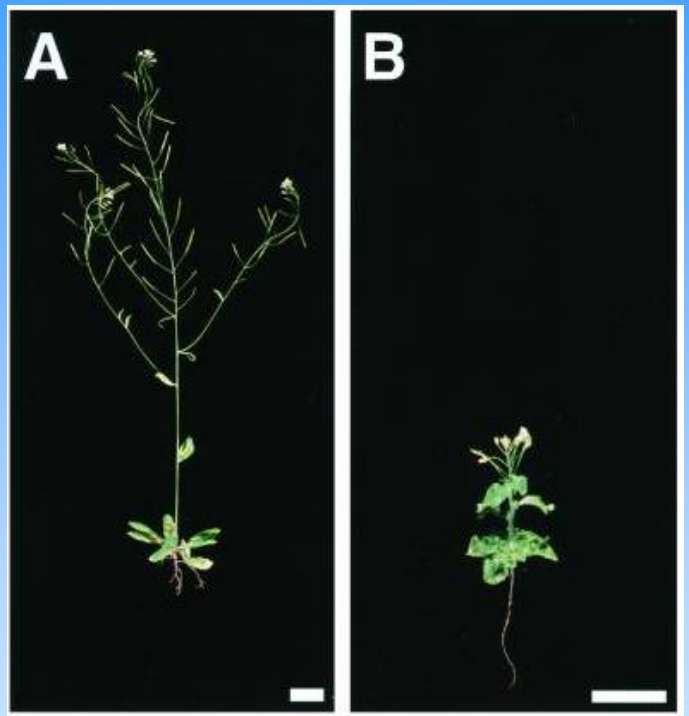


Likely BR-insensitive mutants



rice

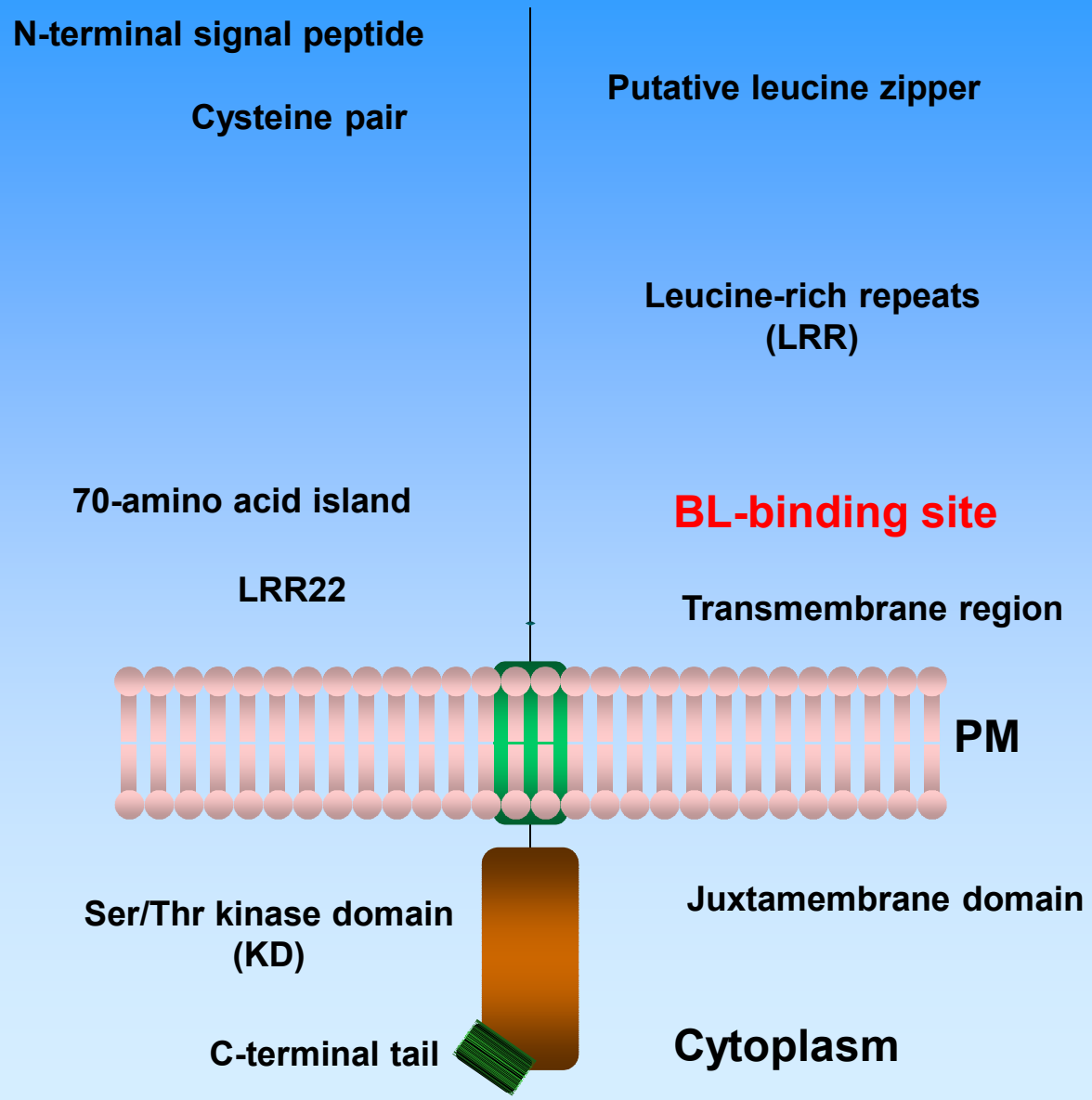
WT *brd2*



Mutant *bri1*
(*brassinosteroid-insensitive1*)

***BRI1* codes for brassinosteroid receptor**

Structure of BRI1 receptor

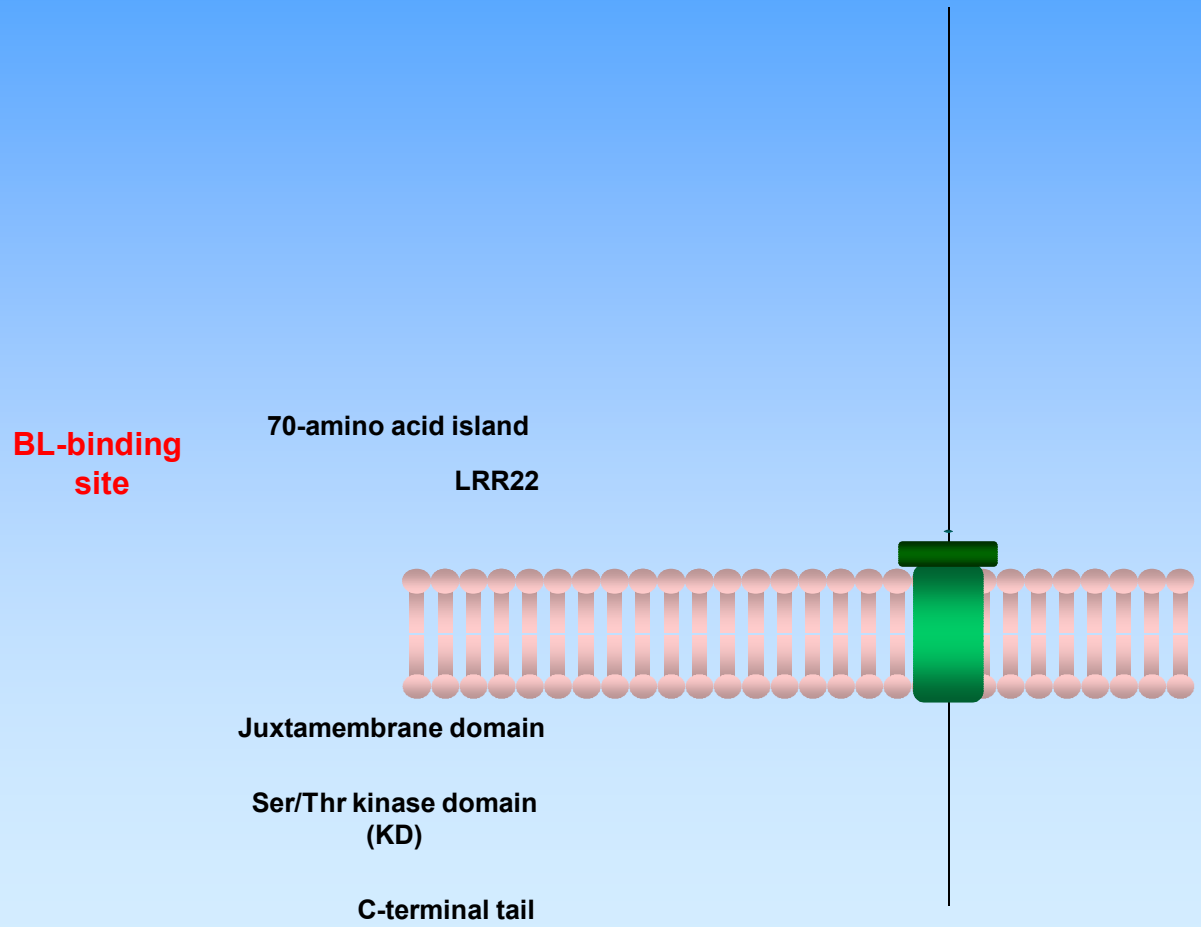


BRI1 **codes leucine-rich repeat (LRR) transmembrane receptor-like kinase (RLK) (LRR-RLK)**

Localization: plasma membrane (expression of BRI1-GFP)

Phosphorylation of BRI1 receptor

Analysis of BRI1-defective mutants → Identification of domains essential for signal transduction



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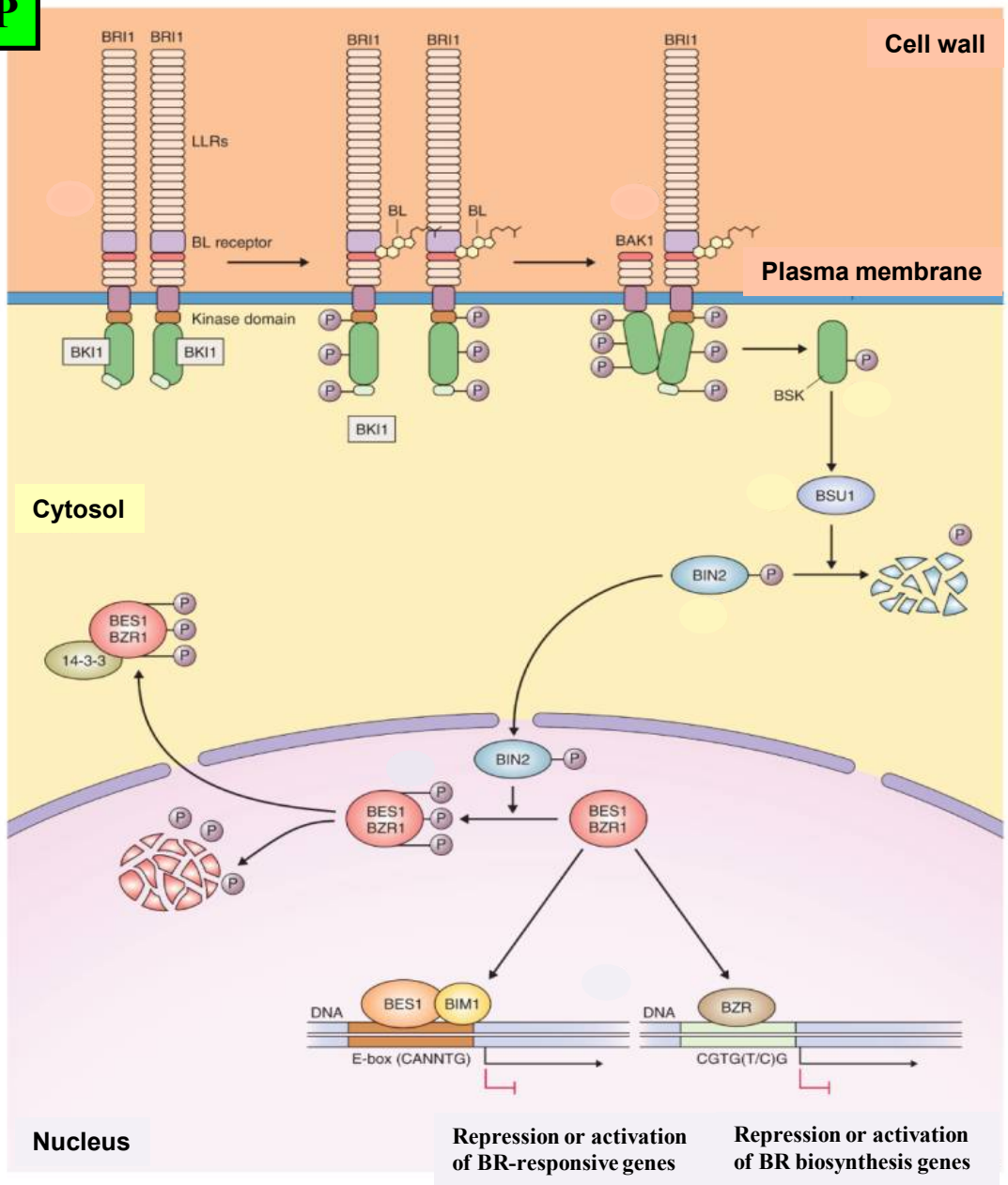


Presence of BL = BL binding to receptor

Activated BRI1/BAK1 heterodimer inhibits activity of BIN2 kinase via **BSK** (**B**rassinosteroid-**S**ignaling **K**inases) and **BSU1** (**bri1 SU**ppressor **1**)

+

Direct activity of BSU1

↓

Accumulation of dephosphorylated form of BES1 and BZR1 in the nucleus

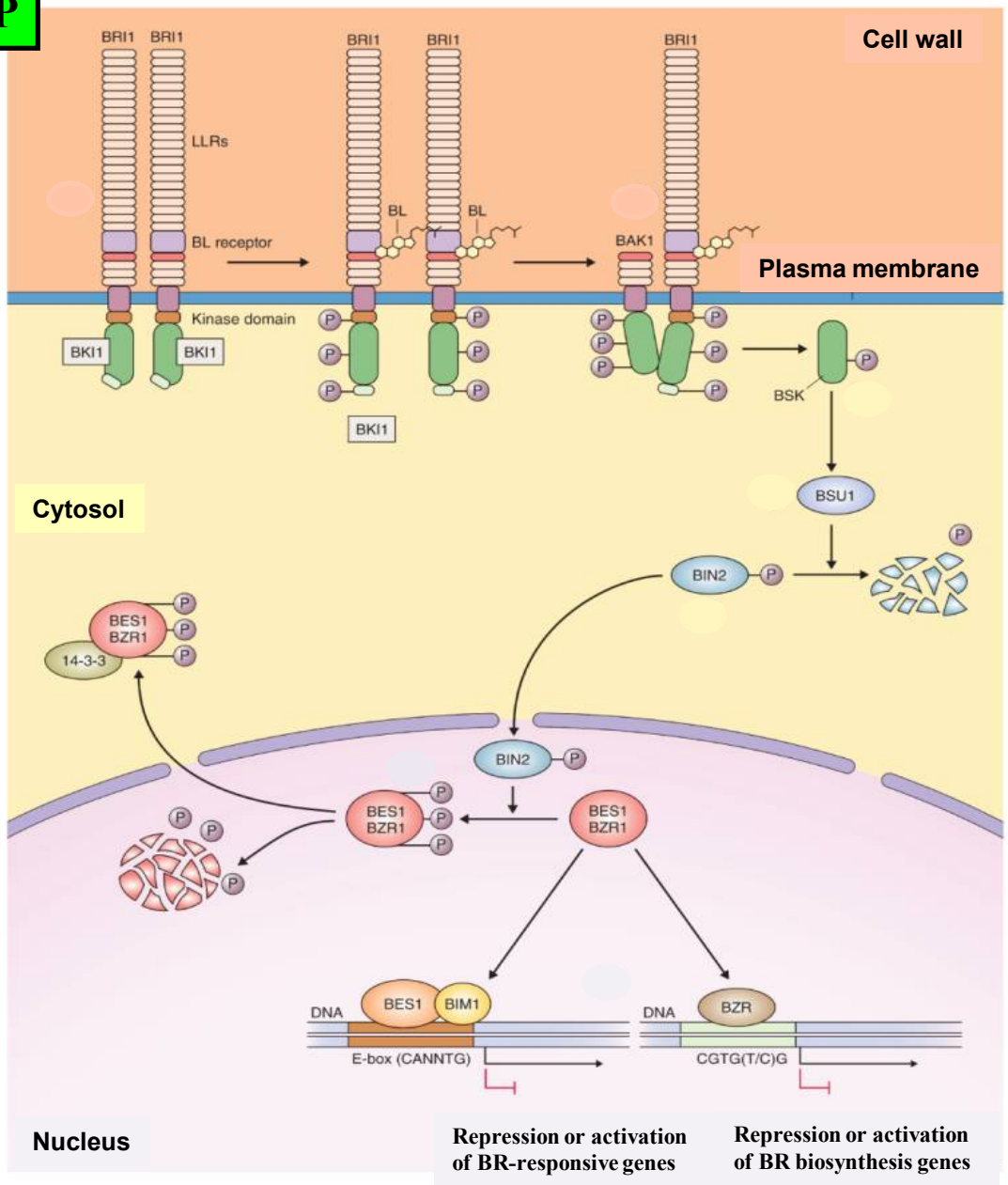
BES1 (bri1-EMS-suppressor 1)

BZR1 (brassinazole-resistant 1)

BES1 and BZR1 – transcriptional factor of BR-induced genes; short-life proteins; degradation in 26S proteasome

↓

Activation or suppression of gene expression



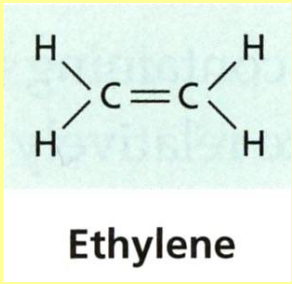
Absence of BR:

Phosphorylation of proteins BES1 and BZR1 localized in nucleus.

Phosphorylated BES1 and BZR1 cannot bind to DNA and are degraded

BR-induced genes are not expressed.

c) Ethylene



- Gaseous hormone

- D. Neljubov (1901) – ethylene-triple response identified.

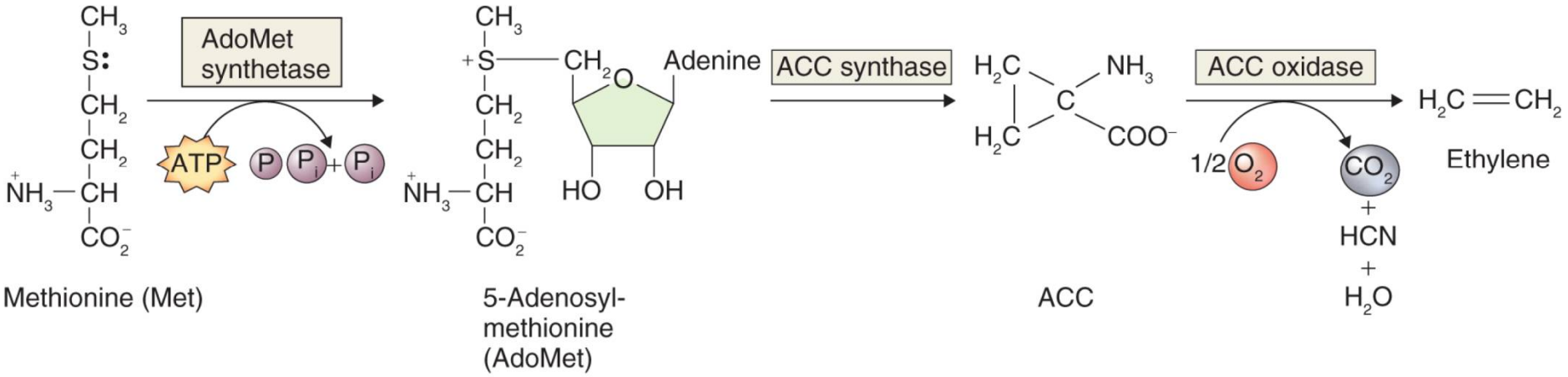
- H. Cousins – 1910 – ethylene is natural substance; gas produced by stored oranges

- R. Gane (1934) – chemical identification of ethylene as a product of plant metabolism

- 1958 – ethylene recognized as plant hormone

Triple response.

Biosynthesis of ethylene



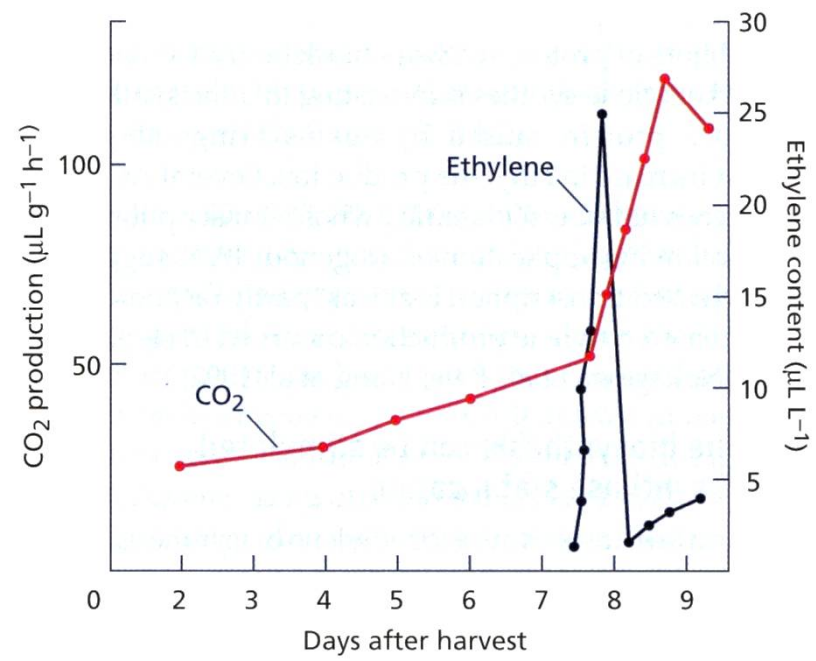
Flammable gas, easily oxidizable

ACC - 1-aminocyclopropane-1-carboxylic acid (ethylene precursor)

Ethylene is produce by ripened fruits, at senesce, at injury and physiological stresses. Concentration of ethylene occurs in ml/l.

Physiological effects of ethylene

Ethylene stimulates ripening some fruits, i.e. climacteric fruits: bananas, apple, tomato, avocado



never ripe WT

Non climacteric fruits: limes, pine apple, water melon,..

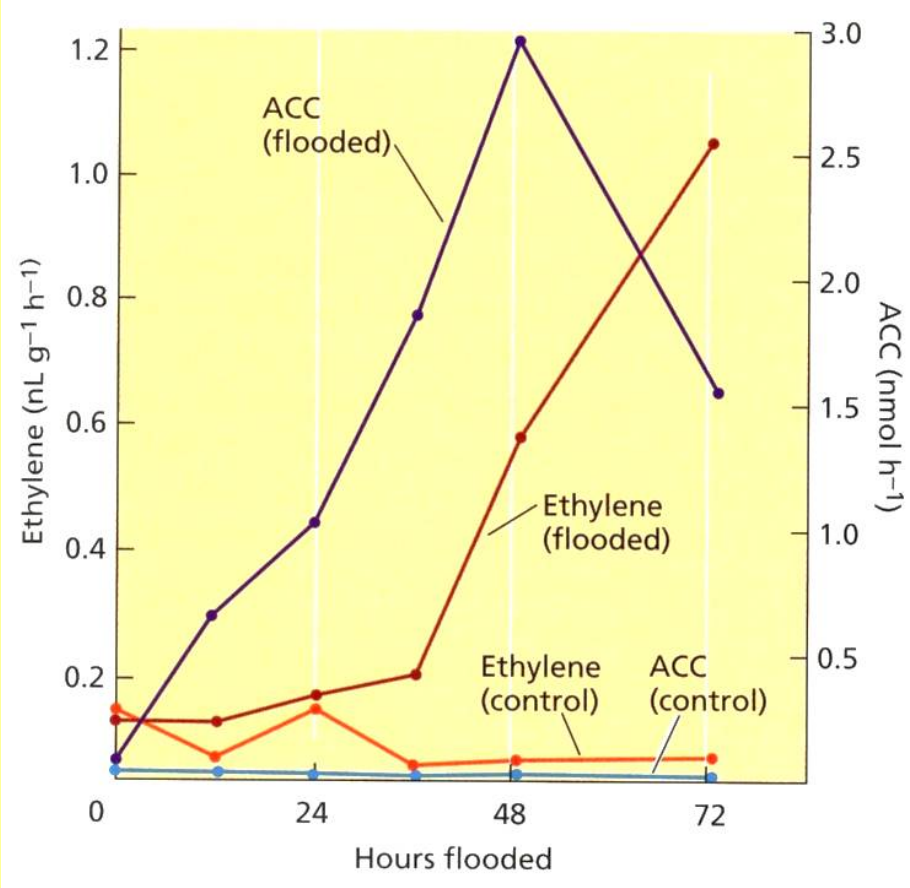
Influence ethylene on leaf epinasty

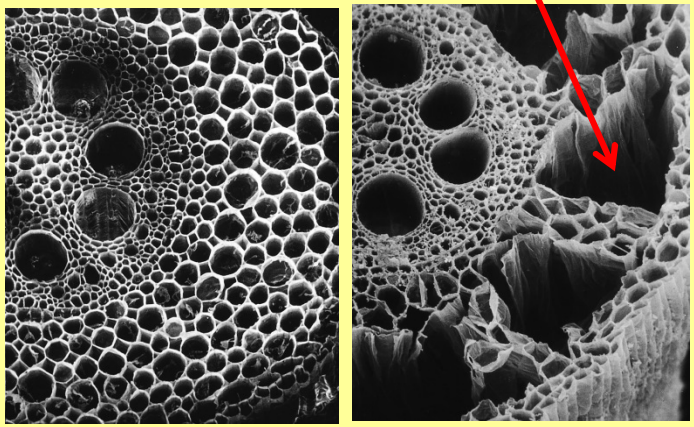
Epinastic leaves of tomato = bent towards down

Cells of upper part of petiole grow faster than the cells of lower part.

Interaction of auxin-ethylene → auxin induces ethylene production

Concentration of ethylene increases in stressed plants – e.g. inundation (flood)

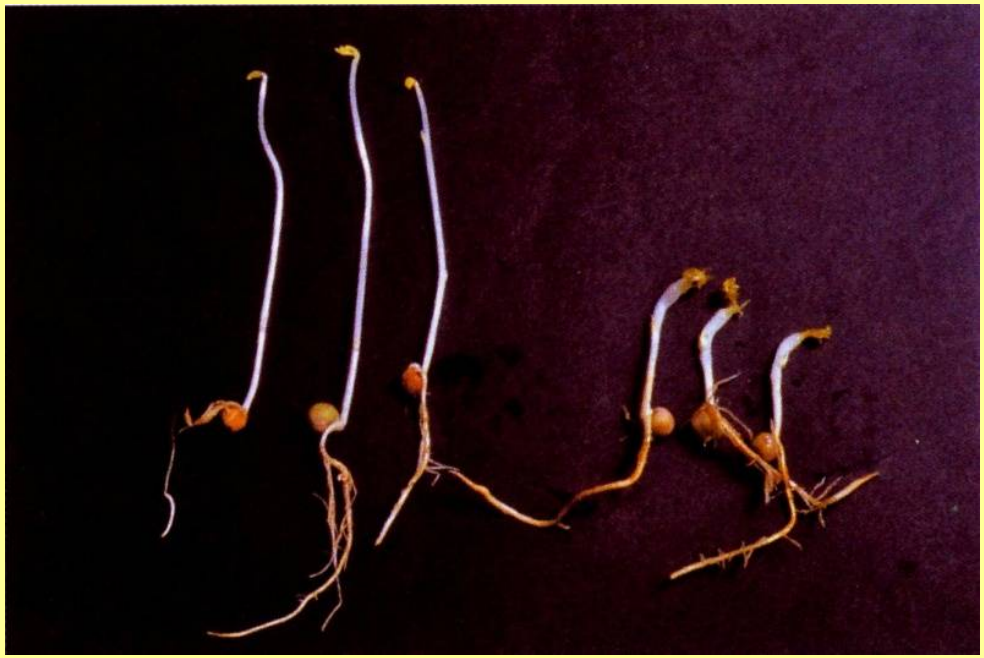




In submerged plants, the roots suffer from hypoxia (oxygen deficiency). Hypoxia in roots stimulates formation of ethylene. Ethylene increases cytosolic concentration of Ca^{2+}, which triggers PCD of cortex cells – formation of aerenchyma.

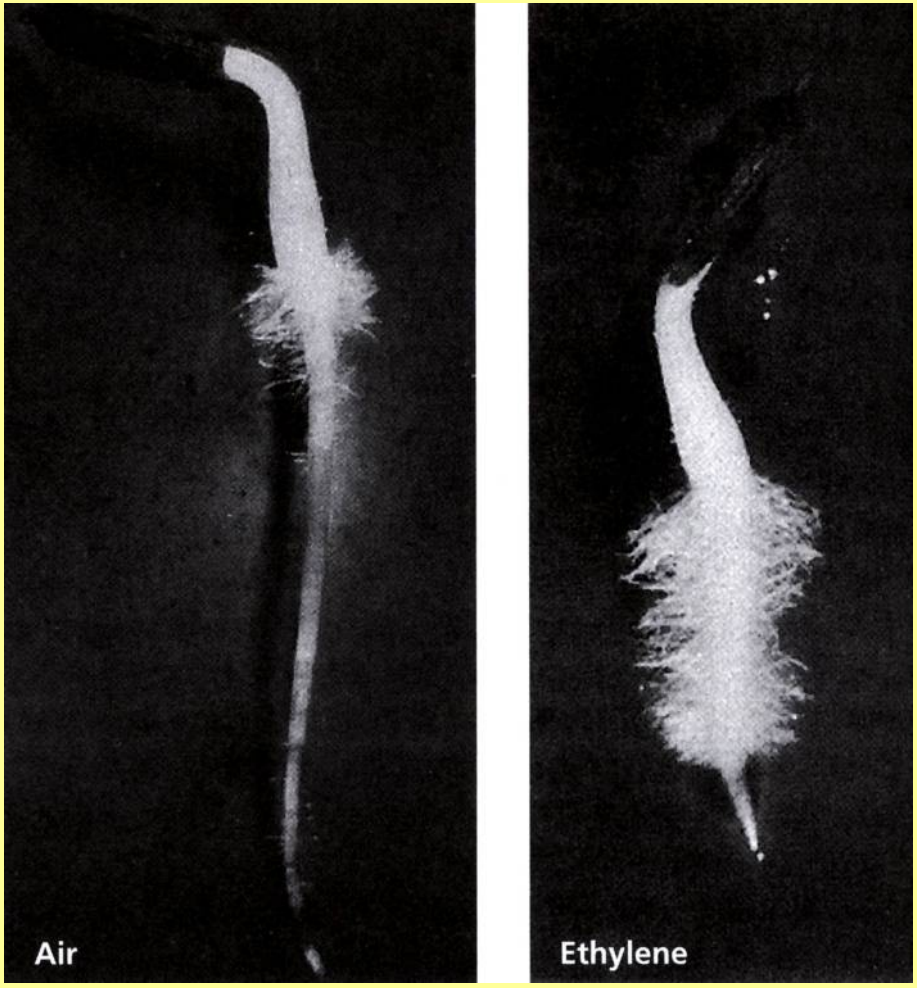
Transport of ACC from roots, which are in anaerobic conditions.

Triple response = changes of three parameters of etiolated plants

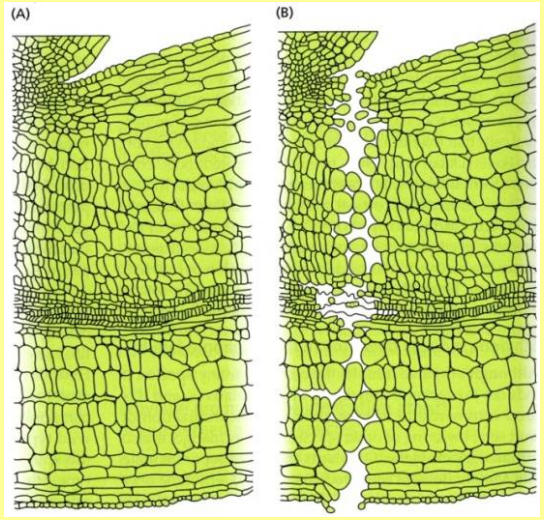


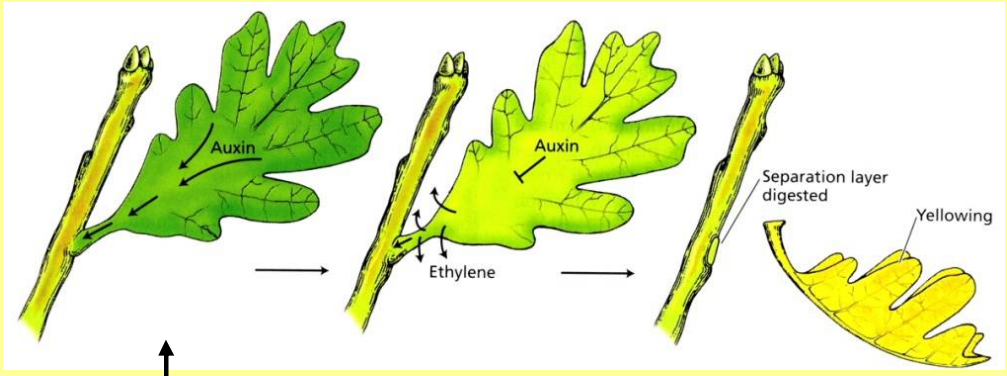
1. Hypocotyl thickening
2. Hypocotyl shortening
3. Apical hook formation

Ethylene induces formation of adventitious roots and root hairs.



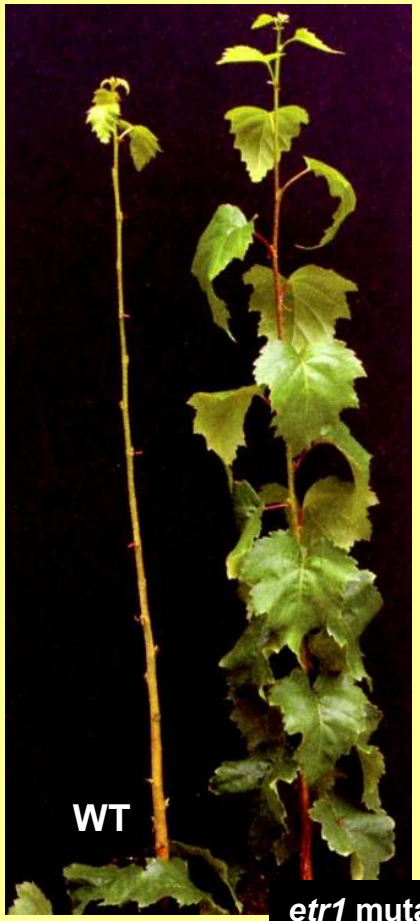
Ethylene speeds up leaf senescence and fall – *etr1* or *ein2* mutants (receptor-affected mutants) show extend length of plant life by 30%





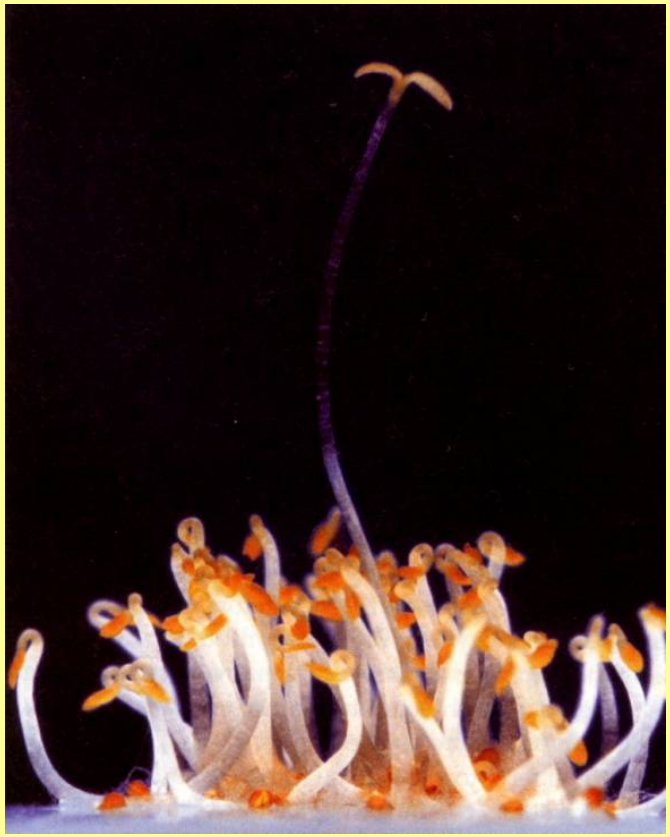
Auxin ↑ → Sensitivity to ethylene ↓

Auxin ↓ → Sensitivity to ethylene ↑, Production of ethylene ↑



etr1 mutant – defect in ethylene receptor

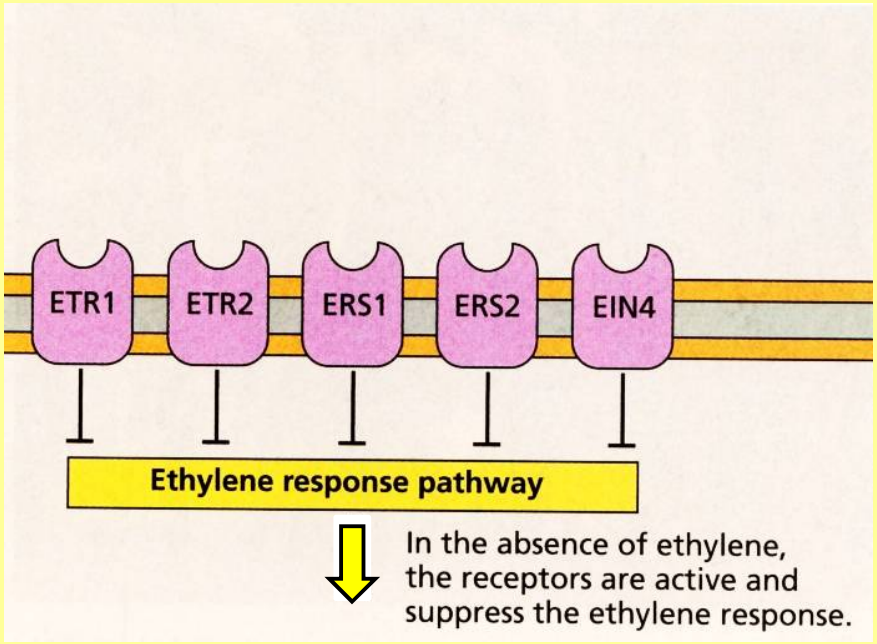
Ethylene signaling

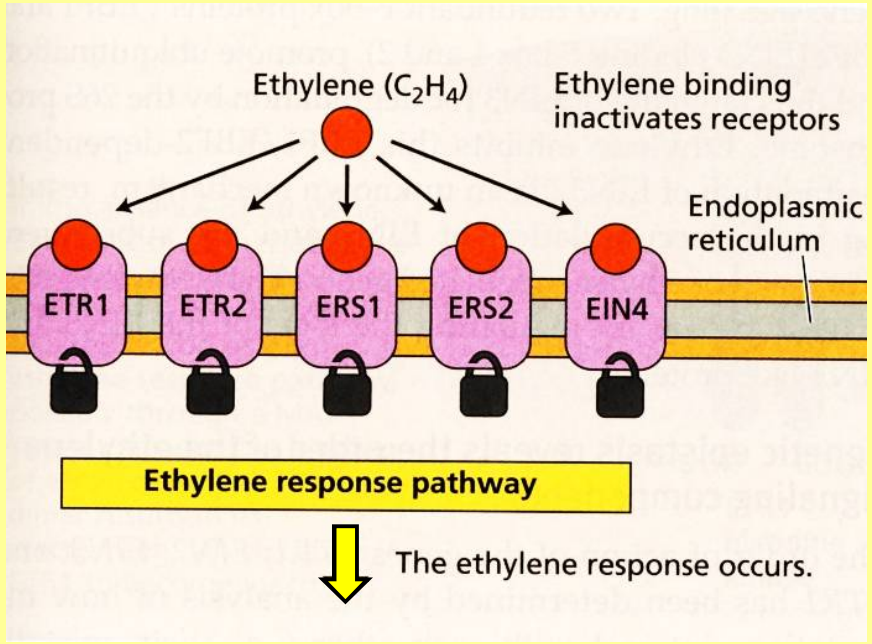


Selection of *etr1* mutant insensitive to ethylene.

Receptor of ethylene

In the absence of ethylene, the receptors on the membrane of endoplasmic reticulum are active and block signaling pathway.





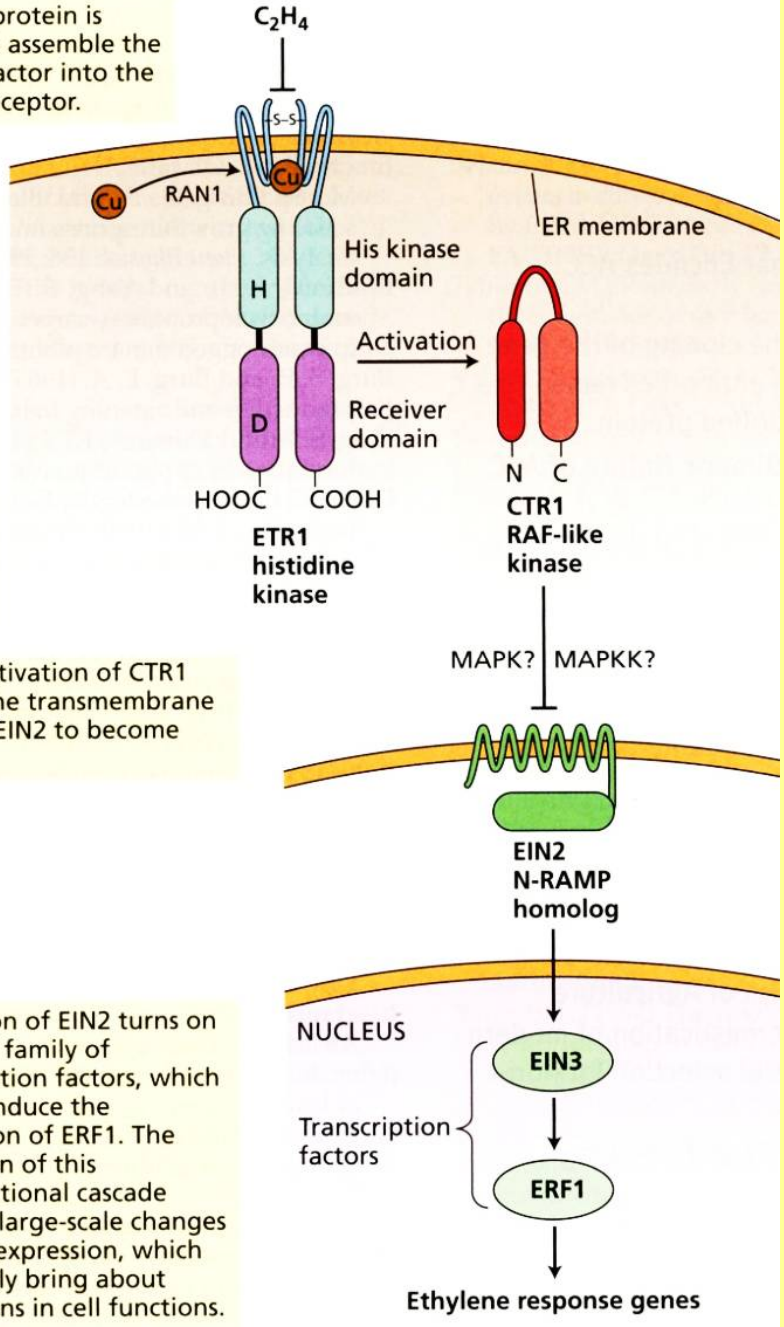
Binding of ethylene to receptor results in suppression of the receptors. By blocking the receptor, other signaling elements are released and ethylene signal is transduced.

The RAN1 protein is required to assemble the copper cofactor into the ethylene receptor.

In the absence of ethylene, ETR1 and the other ethylene receptors activate the kinase activity of CTR1. This leads to a repression of the ethylene response pathway, possibly through a MAP kinase cascade. The binding of ethylene to the ETR1 dimer results in its inactivation, which causes CTR1 to become inactive.

The inactivation of CTR1 allows the transmembrane protein EIN2 to become active.

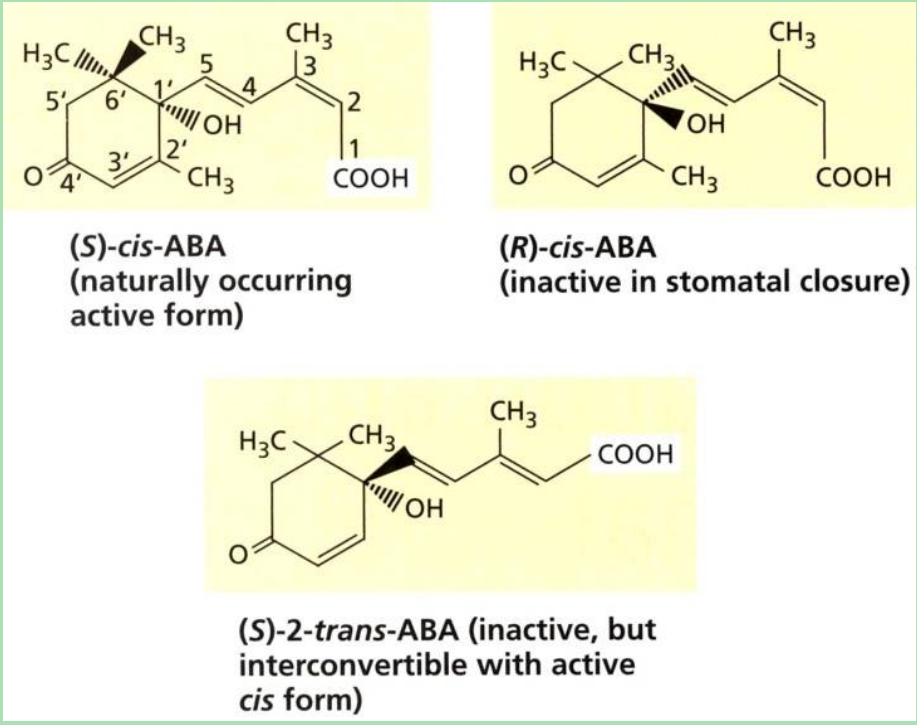
Activation of EIN2 turns on the EIN3 family of transcription factors, which in turn induce the expression of ERF1. The activation of this transcriptional cascade leads to large-scale changes in gene expression, which ultimately bring about alterations in cell functions.



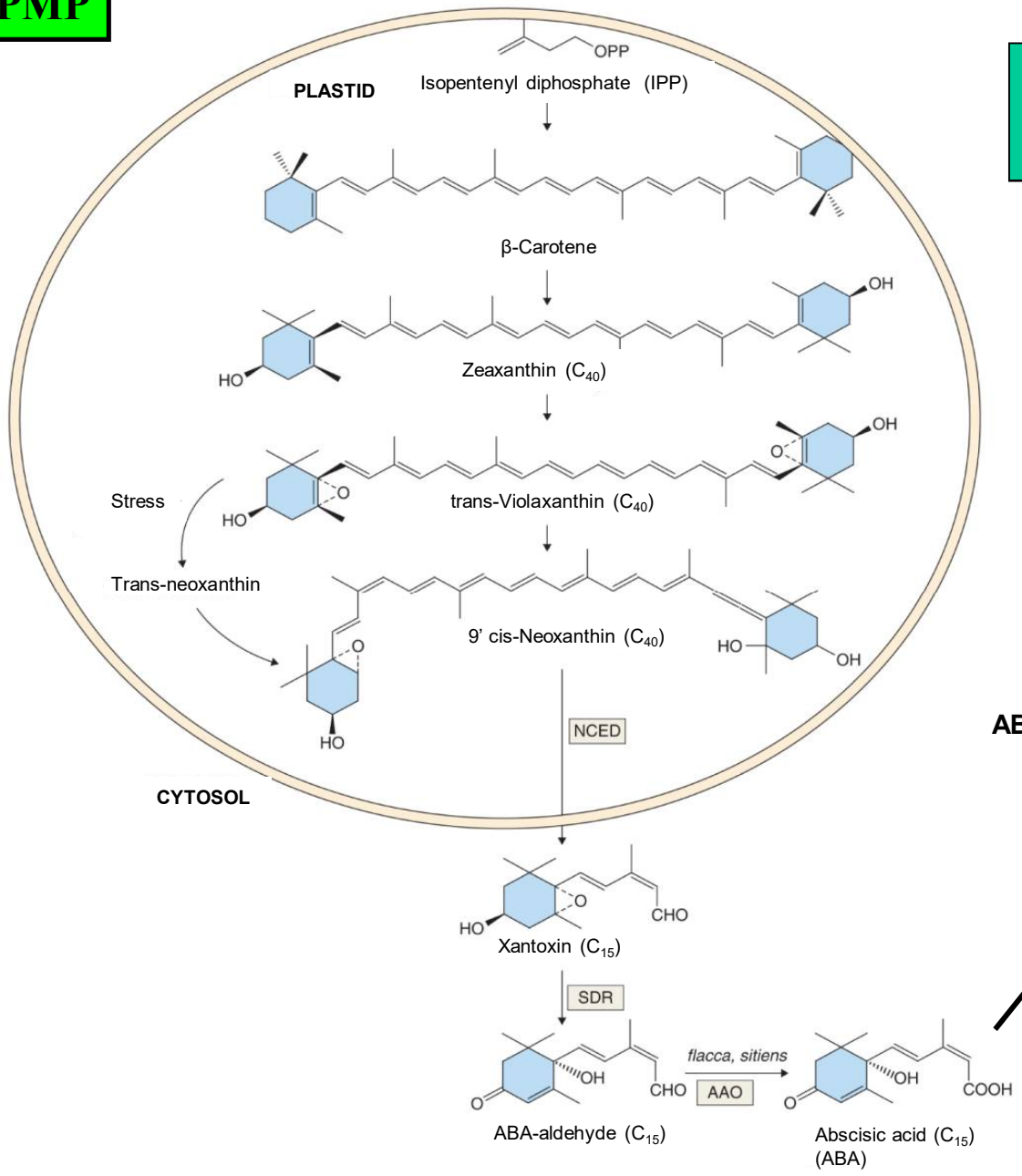
d) Abscisic acid (ABA)

- **Abscisic acid (ABA) (formerly called abscisin II or dormin) is inhibitory phytohormone. It slows plant growth, prepares plant for period of vegetative rest. Structurally belongs to sesquiterpenes.**

- **ABA has a range of isomers – S-(*cis*)-form is natural and active form**

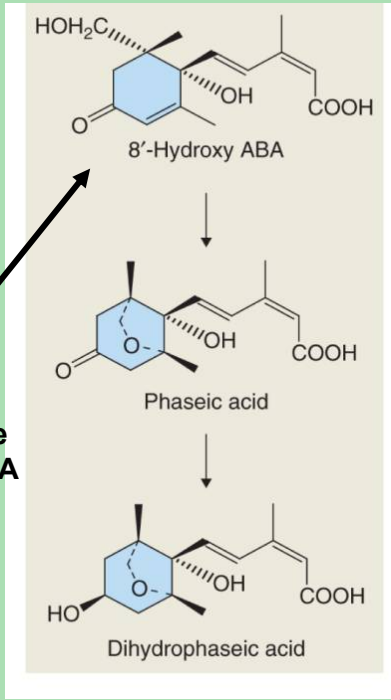


ABA biosynthesis and metabolism



ABA inactivation by oxidative processes.

Enzyme CYP707A



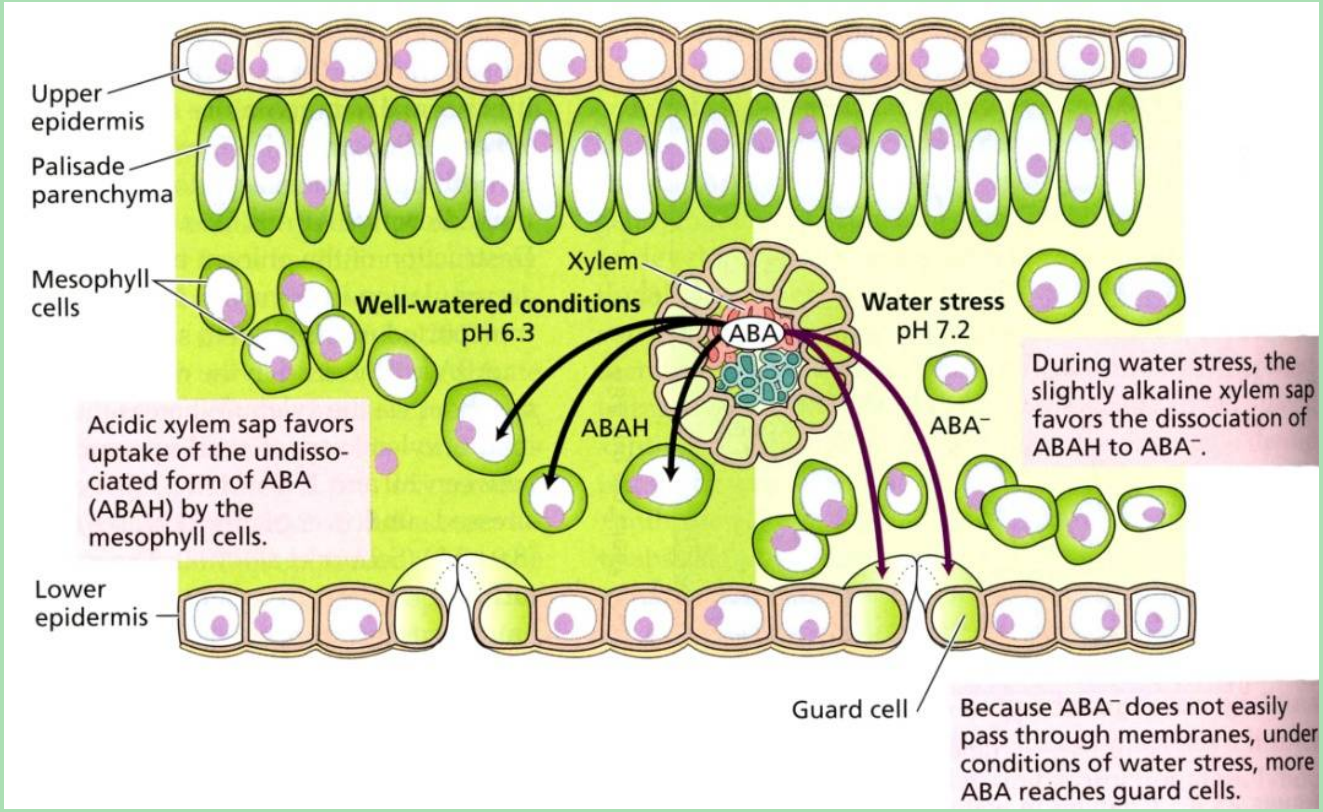
Physiological function of ABA

ABA inhibits precocious germination (vivipary), i.e. ABA induces seed dormancy

In ABA-deficient mutants (*vp2, vp5, vp7, vp9, vp14*), application of ABA results in partial restoration of normal phenotype.

ABA regulates accumulation of storage proteins and proteins responsible for seed drying (proteins LEA – late-embryogenesis-abundant)

ABA closes stomata in dependence of water stress



Hydrated plants: **pH of xylem sap is around 6.3 => absorption of ABA by mesophyll cells, because ABA occurs in undissociated (protonated) form ABAH.**

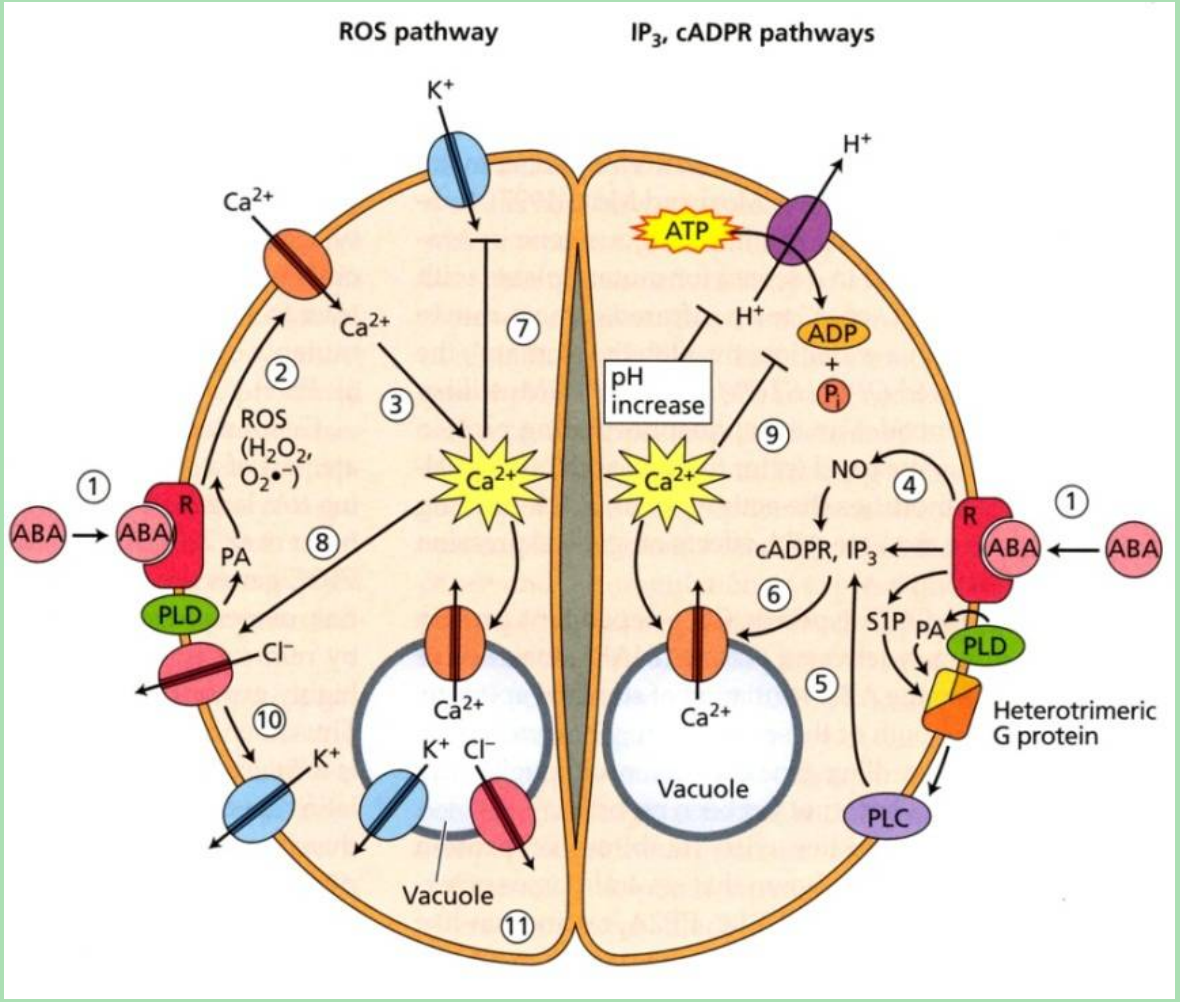
Water stress: **pH of xylem sap increases to 7.2 => stress-induced alcalinization of apoplast results in formation of dissociated form of ABA (ABA^-). ABA^- does not easily pass membrane of mesophyll cell => more ABA^- reaches guard cells, where it binds to membrane receptor.**

PMP

Mechanism of ABA action in guard cells

Water stress:

1. ABA binds to receptor.
2. Binding ABA induces flow of Ca^{2+} into the cell.
3. Intracellular konc. Ca^{2+} increases and further flow of Ca^{2+} from vacuole is induced.
4. ABA stimulates induction of NO and cADPR.
5. ABA increases level of IP_3 via G protein.
6. IP_3 stimulates flow of Ca^{2+} from vacuole.
7. High level of Ca^{2+} blocks flow of K^+ to the cell.
8. High level of Ca^{2+} stimulates flow of Cl^- from the cell and causes membrane depolarization.
9. High level of Ca^{2+} blocks proton pump and amplifies membrane depolarization.
10. Depolarization of membrane activates flow of K^+ from the cell.
11. K^+ and Cl^- flow from vacuole to cytosol.



ROS - Reactive oxygen species

IP_3 - Inositol-triphosphate

cADPR – cyclic ADP-ribose

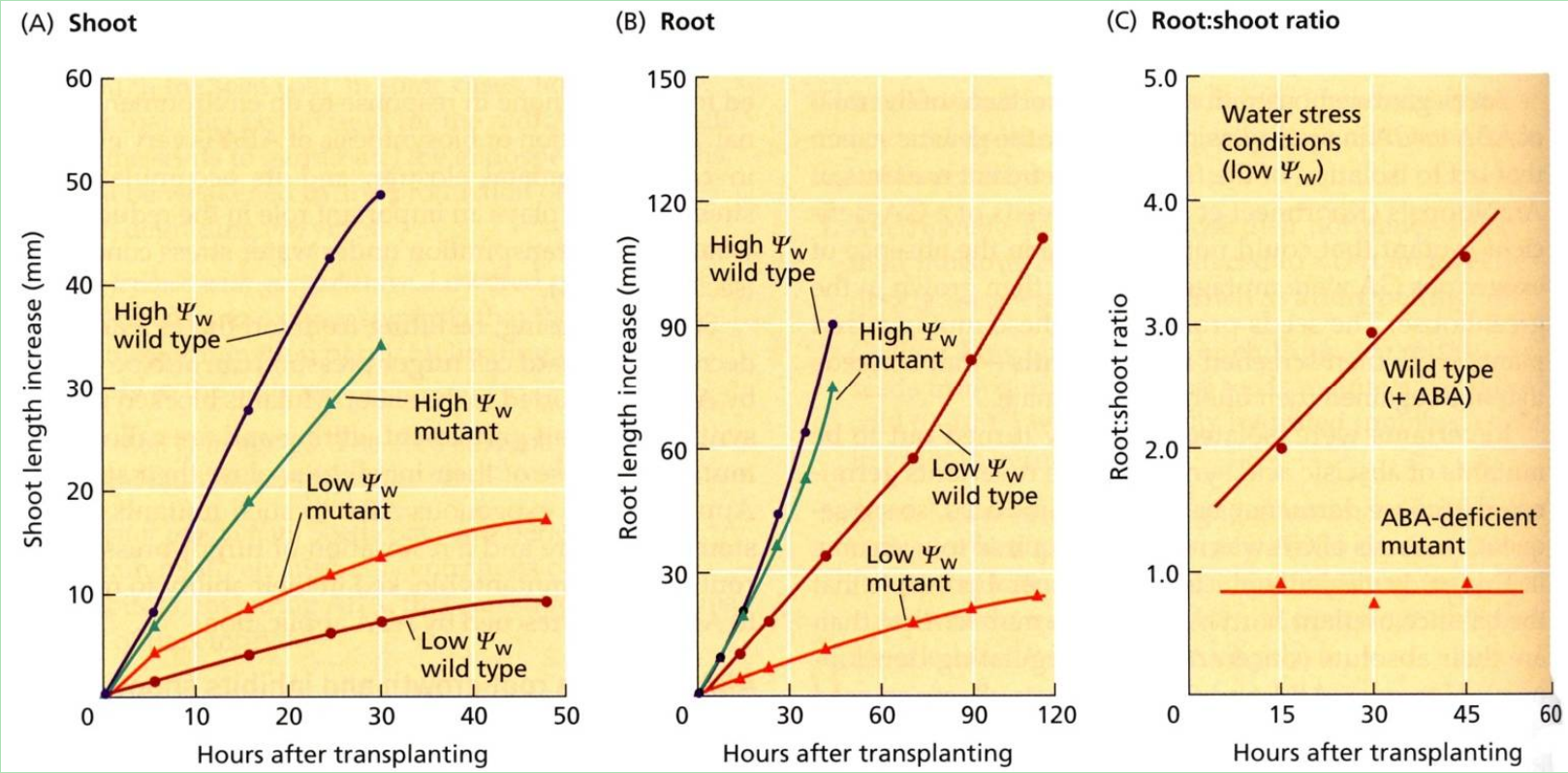
NO – nitric oxide

PLC – phospholipase C)

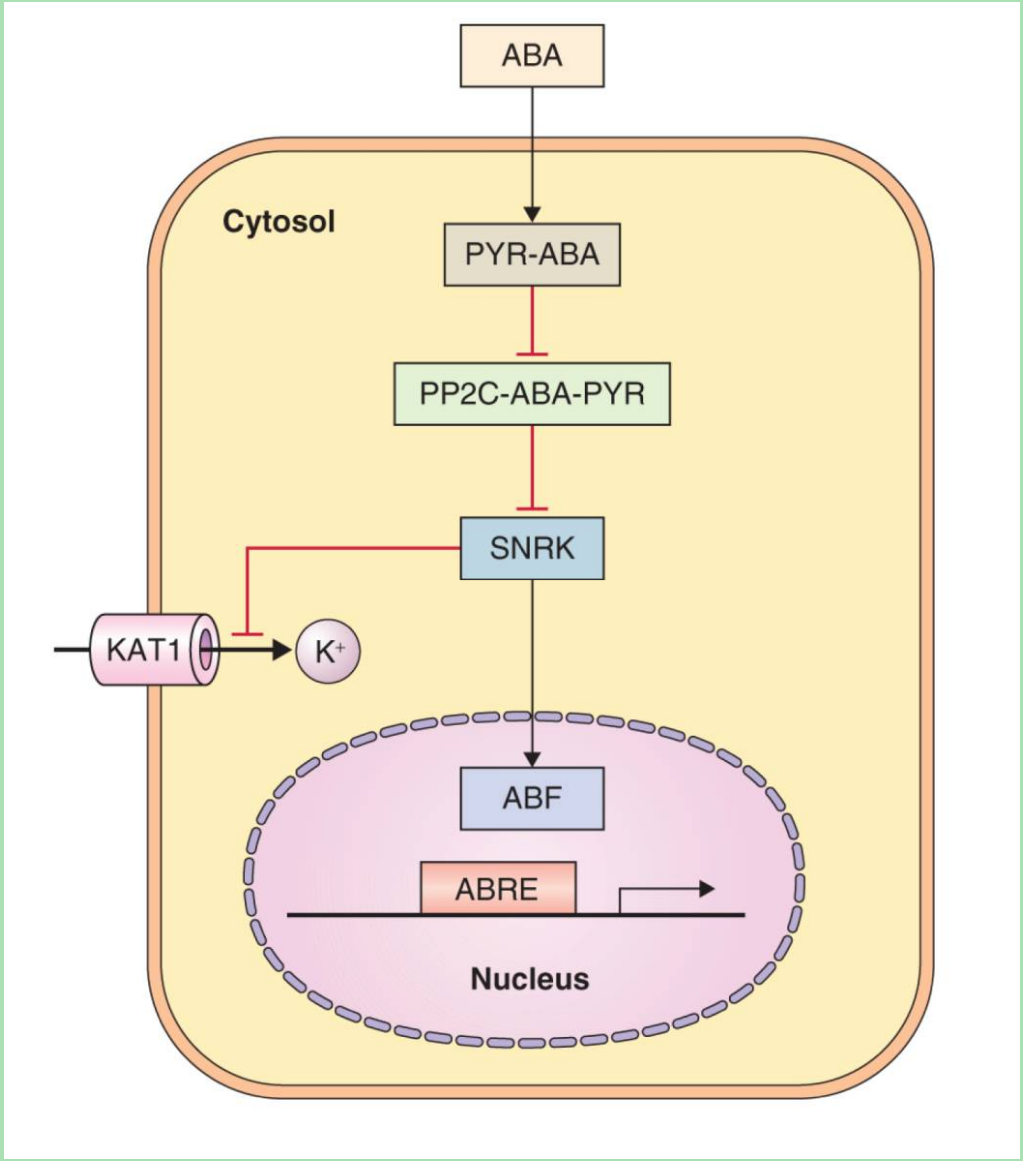
PLD – phospholipase D)

PA – phosphatidic acid)

At low water potential (ψ_W; water deficit) ABA stimulates root growth and inhibits stem growth



ABA signaling



ABA receptors:
PY*rabactin* ***R****esistance* ***1*** (***PYR1***) (homologs **PYL** and **PY**R-**L**ike).

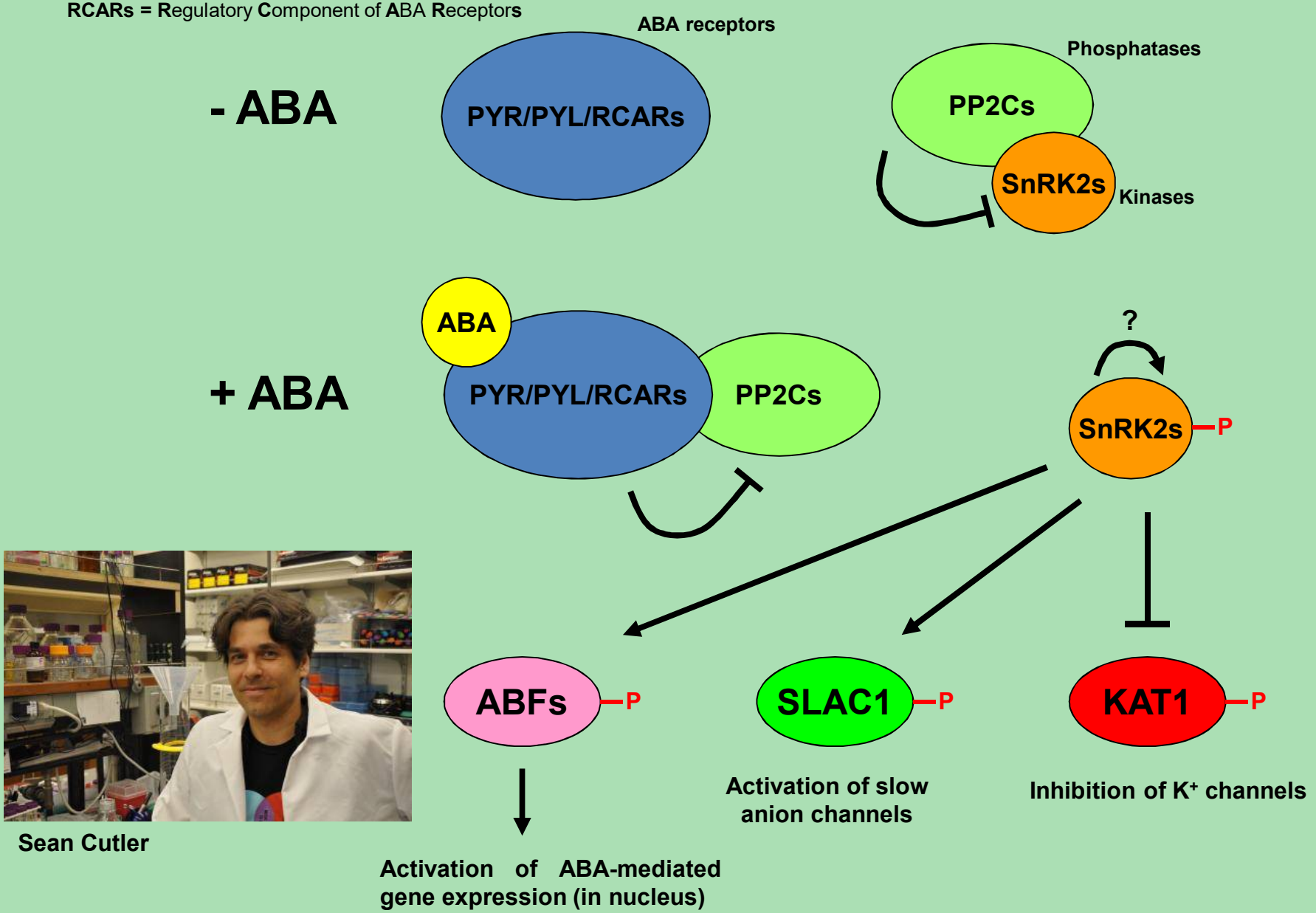
Binding of ABA to PYR1 results in inhibition of 2C type (**P**hos**P**hatase type **2C**, **PP2C**)

Proteins PP2C target proteins called **SN**F1-**R**elated protein **K**inase 2, **SNRK2**.

Absence of ABA: Phosphatase PP2C (ABI1, ABI2) is active and de-phosphorylates SNRK2. SNRK2 kinase is inactivated.

Presence of ABA: Phosphatase PP2C is inhibited and SNRK kinases are active.

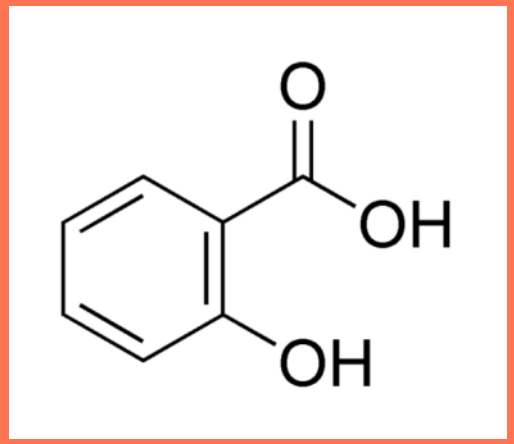
PYR = PYrabactin **R**esistance
PYL = PYR-**L**ike
RCARs = Regulatory **C**omponent of **A**BA **R**eceptor**s**



Sean Cutler

e) Salicylic acid (SA)

Salicylic acid

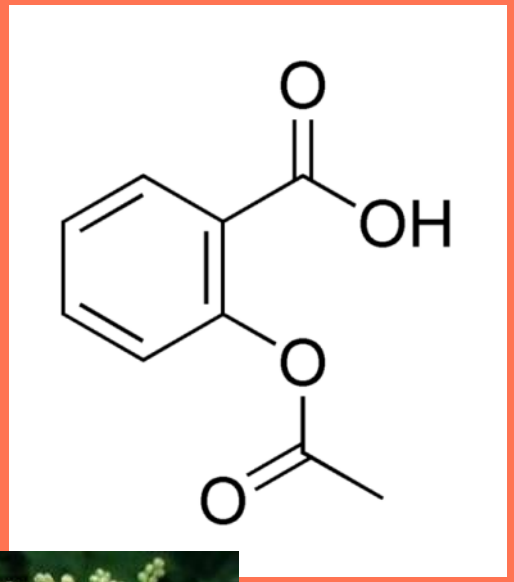


Named after *Salix alba*
(Vrba bílá)

Acetylsalicylic acid

Derivative of SA

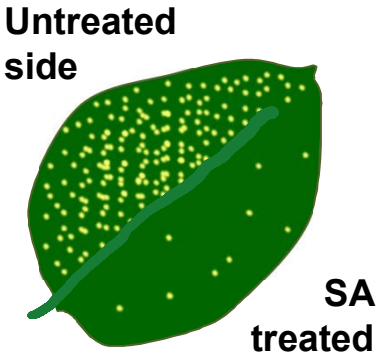
Aspirin



Named after
Spiraea (Tavolník)

History of SA

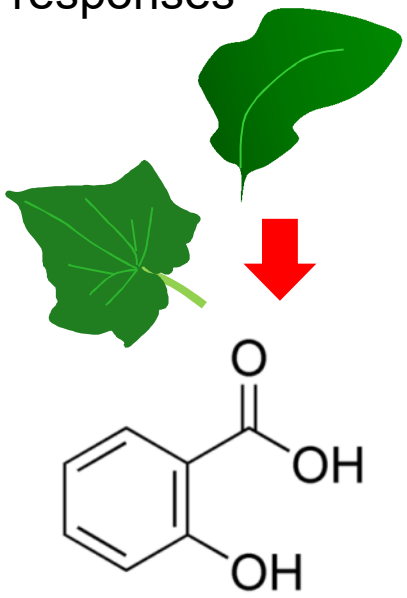
Hippocrates wrote about the use of willow to relieve pain ~ 2400 years ago



In 1979 White showed that pre-treatment of a leaf with aspirin or SA conferred resistance to tobacco mosaic virus (TMV)

In 1987 endogenous SA was shown to be responsible for heat production in *Arum* lily flowers

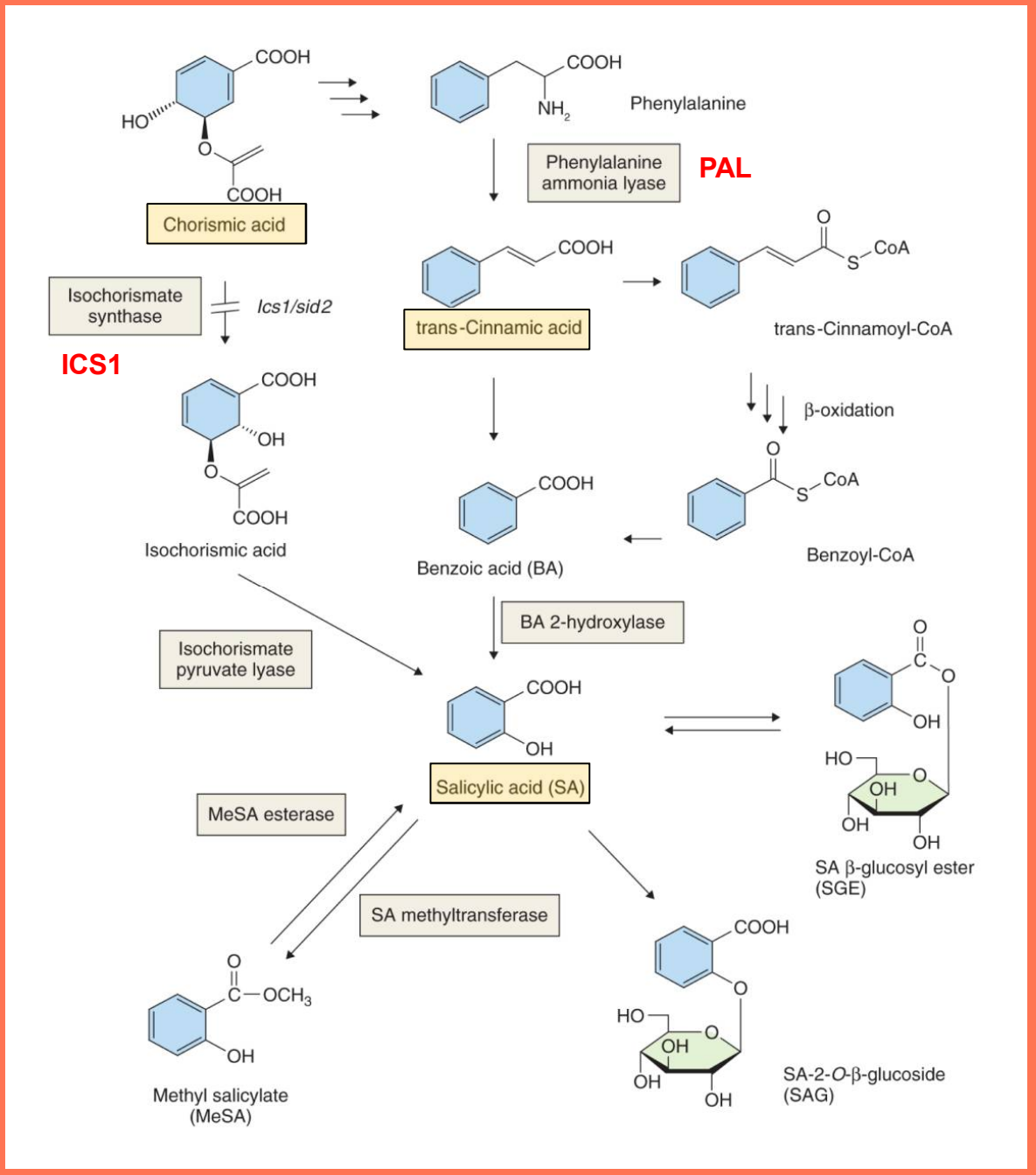
In 1990 SA was shown to be an endogenous signal in defense responses



Biosynthesis and metabolism of SA

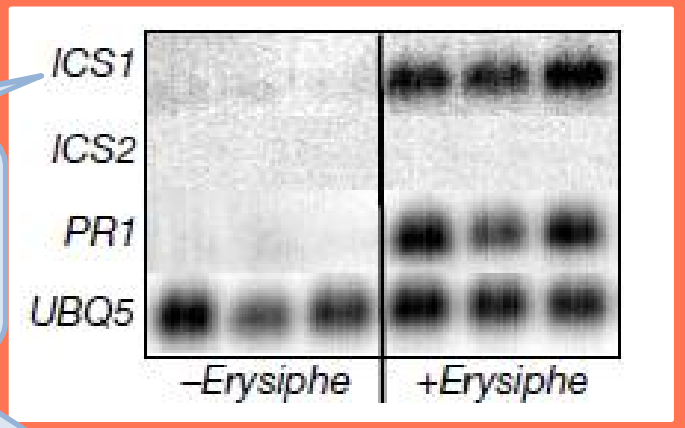
2 synthetic pathways of SA

1. Cinnamic acid
2. Chorismic acid

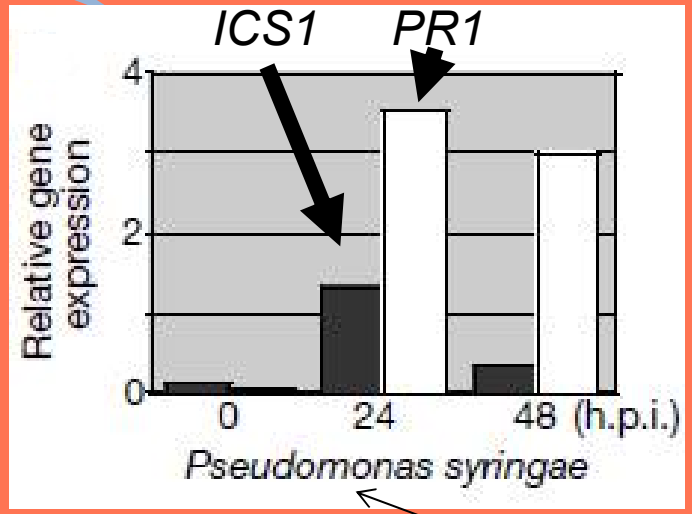


In many plant species the gene for ICS1 is upregulated by pathogen.

ICS1 gene expression increases after pathogen attack.



PR1 is SA-induced gene expressed after pathogen attack.



Gram-negative bacteria

Erysiphe **– a fungi inducing disease called mildew**

Physiological function of SA

Response to pathogens

- Local and hypersensitive response
- Systemic acquired resistance

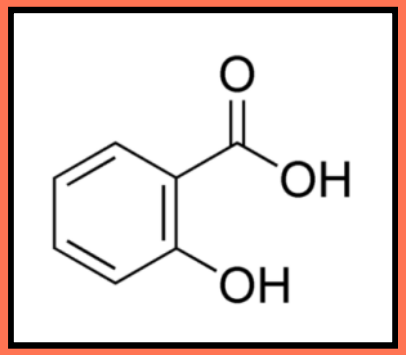
Interaction

Other functions

- Abiotic stress
- Thermogenesis

Signal (e.g. pathogen, UV, heat, developmental cues)

Increased synthesis of SA



Expression of pathogen-induced genes → PROTECTION

Responses to stress

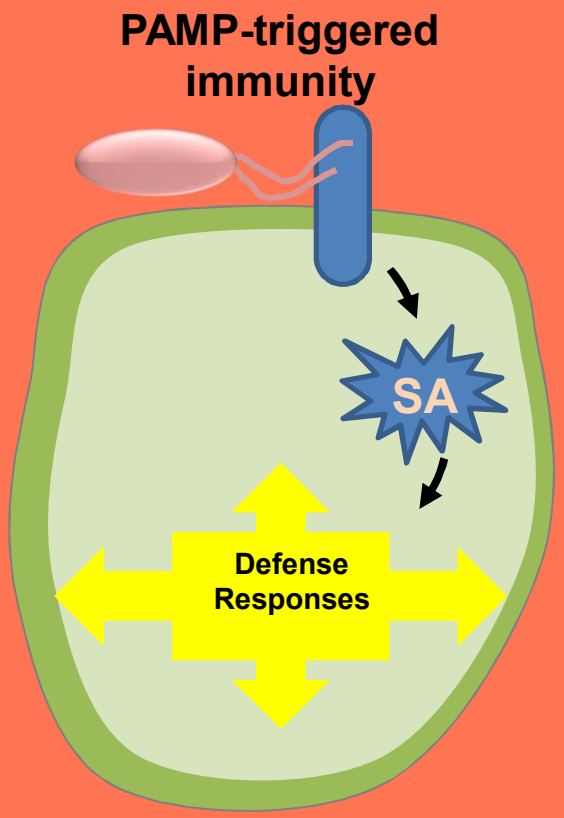
Developmental responses

Basic effect of SA in plants is ability to change expression of genes.

Pattern-triggered immunity (PTI)

Pathogen-associated molecular patterns (PAMPs) are conserved molecules like flagellin or chitin.

They are perceived by extracellular receptors called PRRs (pattern recognition receptors)



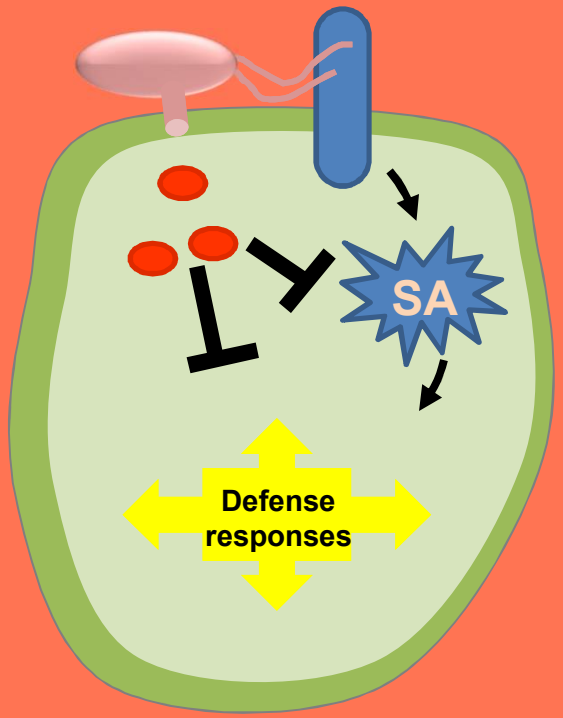
PAMP perception triggers SA synthesis and defense gene induction = pattern-triggered immunity (PTI)

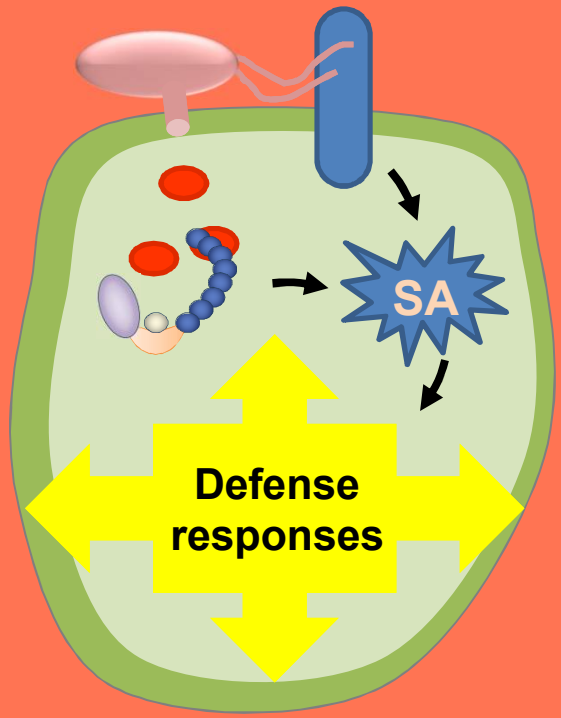
Effector-triggered immunity (ETI)

Some pathogens can overcome PTI by secreting effectors into the cell.

Buňka vytváří R-proteiny.

R-proteins recognize effectors and trigger an enhanced response.

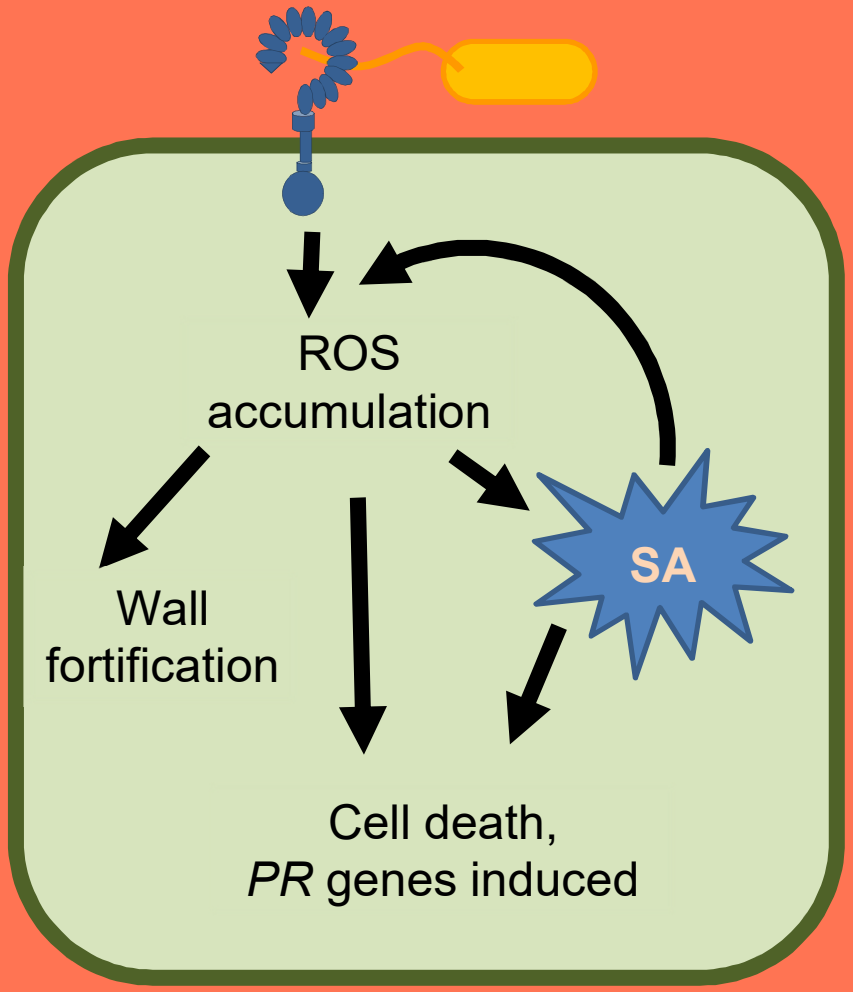




Effector-triggered immunity

Local response – hypersensitive response

Pathogen perception triggers a local hypersensitive response that includes an oxidative burst, wall fortification, induction of *PR* genes and cell death



Hypersensitive response (HR) seals the pathogen in a tomb of dead cells

The HR kills the infected cells and cells surrounding them and helps prevent the pathogen from spreading.

Systemic acquired resistance - SAR

Described in tobacco leaves infected by Tobacco Mosaic Virus (TMV)

This plant was not pre-inoculated with TMV and shows large lesions upon TMV inoculation

This experiment demonstrates systemic signaling

This plant was pre-inoculated with TMV on the three bottom leaves. Seven days later two upper leaves were inoculated. The upper leaves show tiny lesions indicating an **enhanced** resistance response

Thermogenesis

Amines + SA → Heat → Bad smell

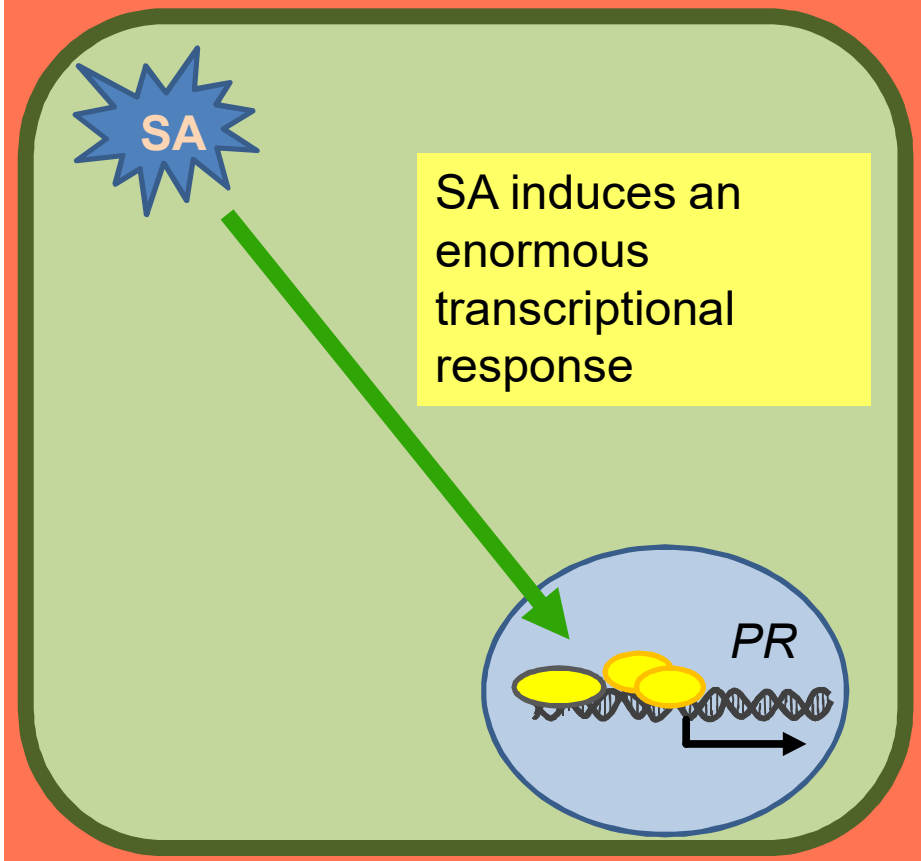
Substance inducing thermogenesis was first called **calorigen.** In 80th it was revealed that it is SA. Application of SA on the plant results in induction of thermogenesis.

Zmijovec titánský (áronova hůl)
(*Amorphophallus titanum*)

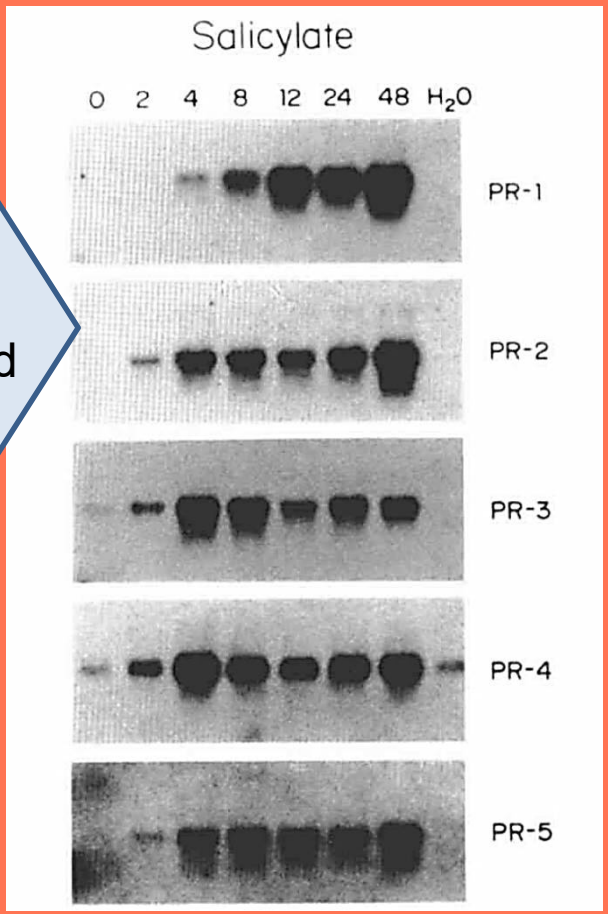
Blowflies of flower of *Helicodiceros muscivorus*

Some arum lilies smell like rotting flesh, attracting flies and carrion-eating insects as pollinators. They produce heat to carry the odor and mimic a decaying corpse

SA signaling



PR genes are pathogenesis related genes, and are involved in defense responses



Ward ER et al. (1991) Plant Cell. 3: 1085-1094

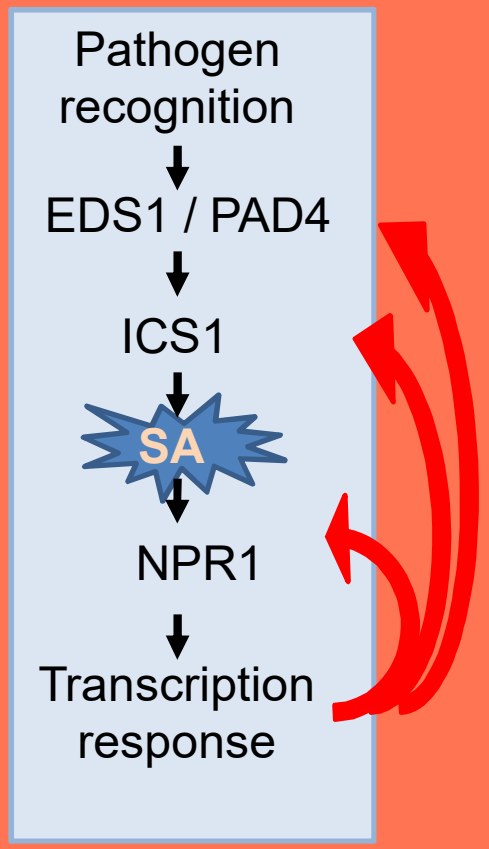
Many genes respond to SA: Early genes amplify the signal

Early

ICS1 ←
EDS1 ←
PAD4 ←
NPR1 ←
Několik *WRKY*s
(transkripční faktory)

Later

PR-1
PR-5
BGL2
NIMIN1
ATNUDT6
FRK1
Několik *WRKY*s
(transkripční faktory)



Wild-type ***npr1*** **(aka** ***nim1*****)**

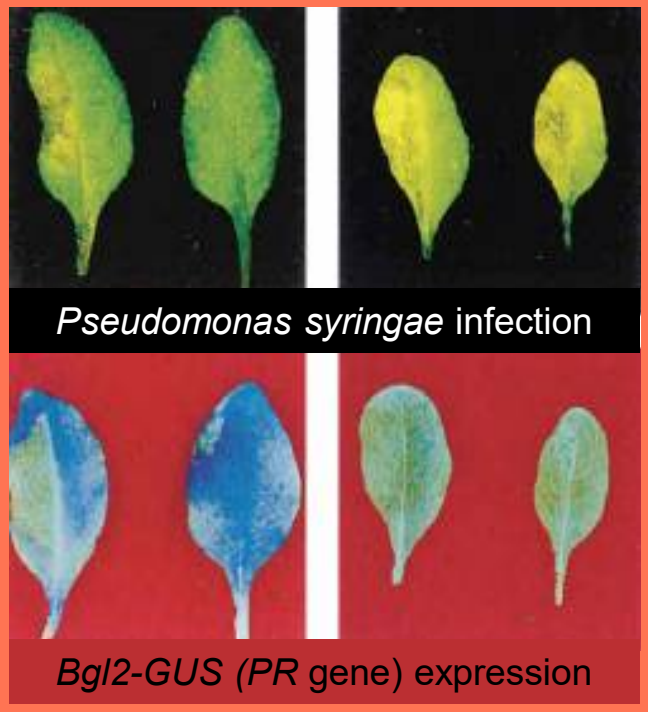
NPR1 (**N**ONEXPRESSOR OF **P**ATHOGENESIS-**R**ELATED GENES**1**) is necessary for defense responses and at the core of SA signal transduction.

Plants were treated with SA, then three days later challenged with a fungal pathogen (*Hyaloperonospora arabidopsidis*)

NPR1 is necessary and sufficient for downstream signaling and defense

Loss-of-function mutant:
more sensitive

Wild-type *npr1*

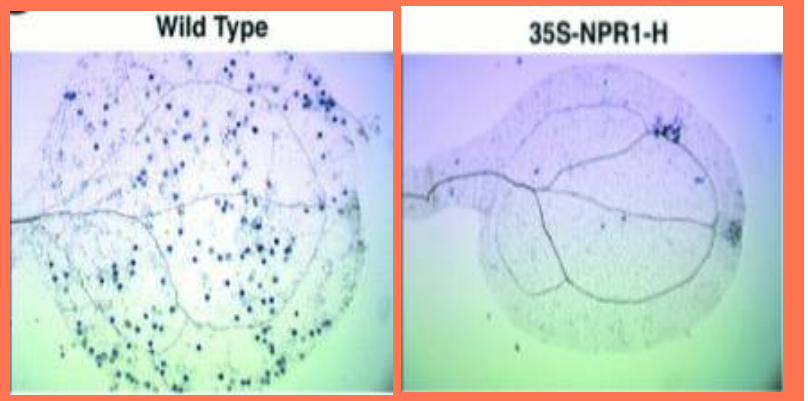


Pseudomonas syringae infection

Bgl2-GUS (PR gene) expression

SA
↓
NPR1
↓
PR gene expression
↓
Resistance

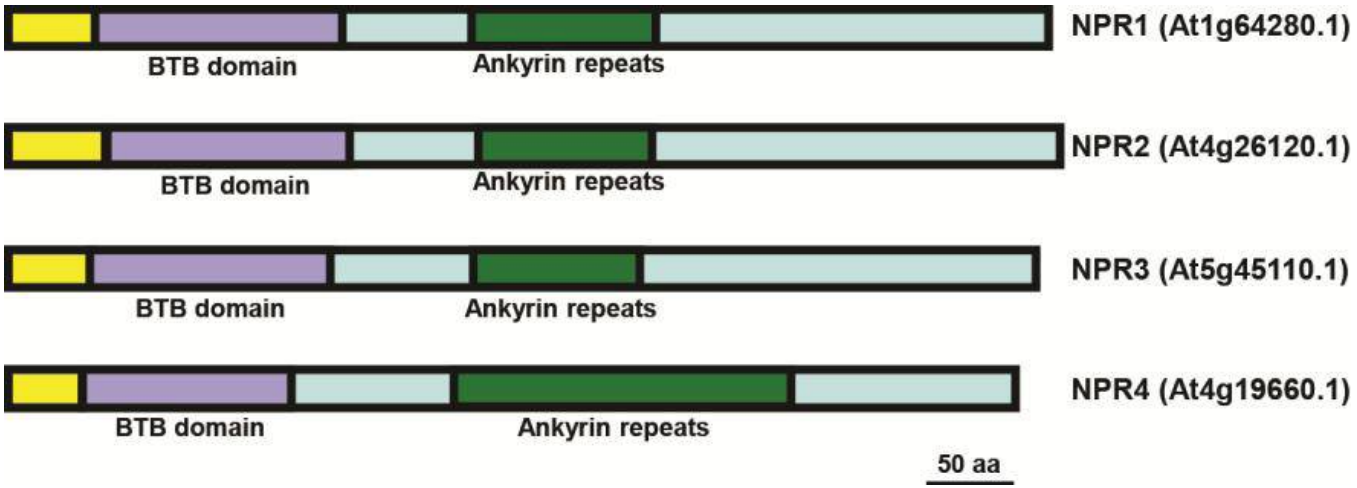
Gain-of-function mutant:
resistant



Cao H et al. (1997) Cell 88: 57-63
Cao H et al. (1998) PNAS 95: 6531-6536

NPR1, NPR3 and NPR4 are SA receptors

In 2012 two groups reported that members of the NPR family serve as SA receptors



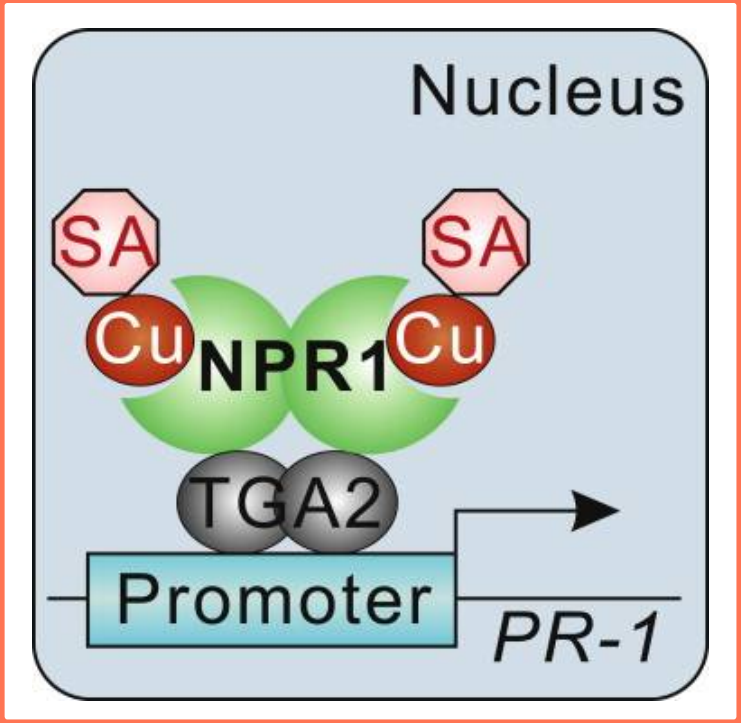
NPR1 is regulated directly by SA binding

and/or

NPR3/4 bind SA and regulate NPR1

Fu ZQ et al. (2012) Nature 486: 228–232

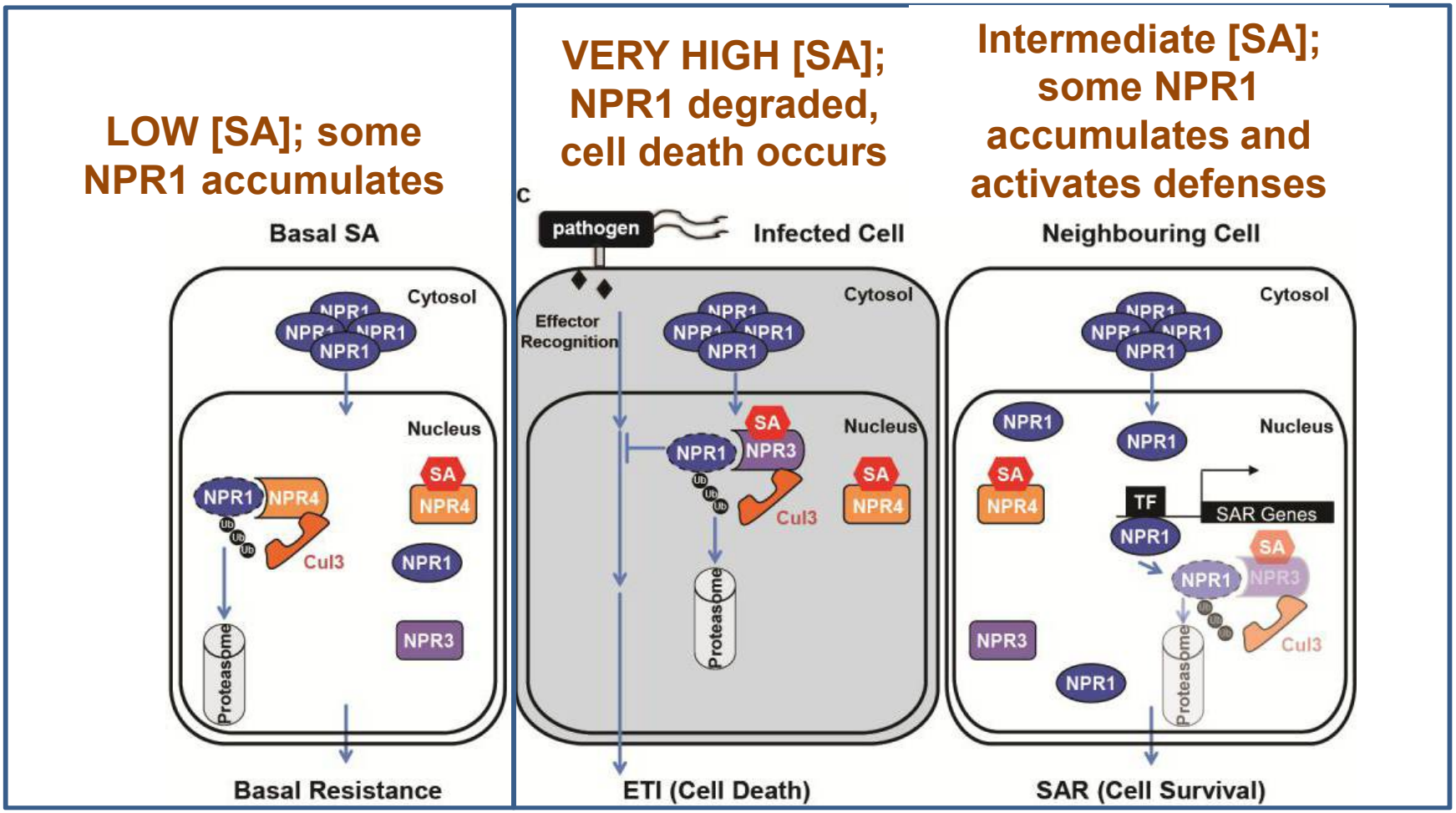
NPR1 is activated by SA



NPR1 binds SA, triggering a conformational change that releases its C-terminal activation domain from inhibition to trigger transcription.

Wu Y et al. (2012) Cell Rep. 1: 639-647

NPR3 and NPR4 regulate the proteolytic turnover of NPR1

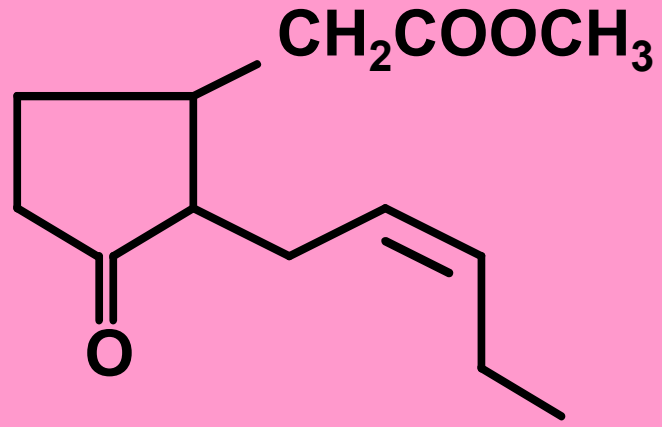


Fu ZQ et al. (2012) Nature 486: 228–232

f) Jasmonic acid (JA)

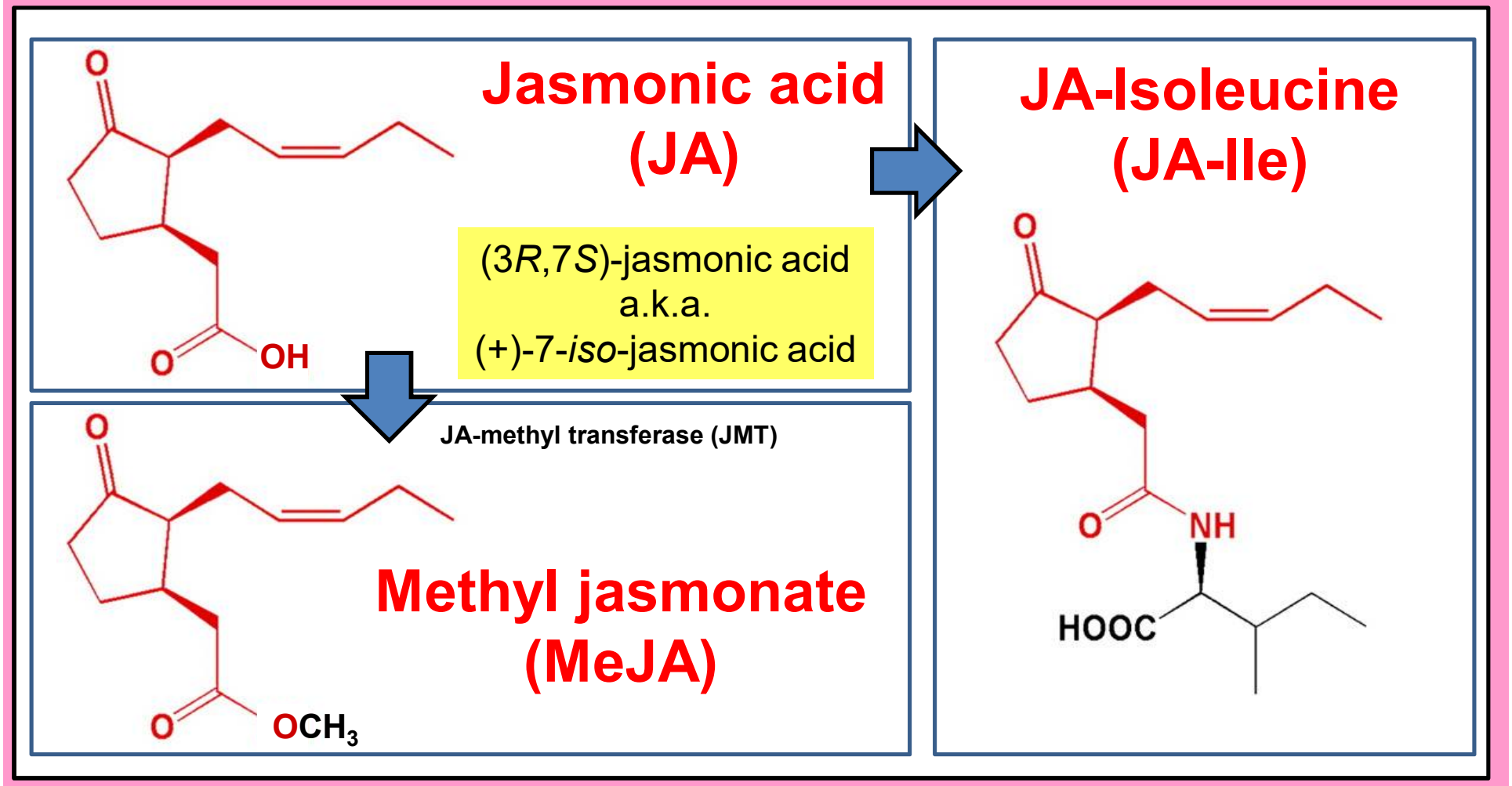
In 1962 methyl jasmonate (MeJA) was isolated from *Jasminum grandiflorium*

Like other esters, methyl jasmonate smells good – it is the dominant scent from jasmine flowers



Methyl jasmonate

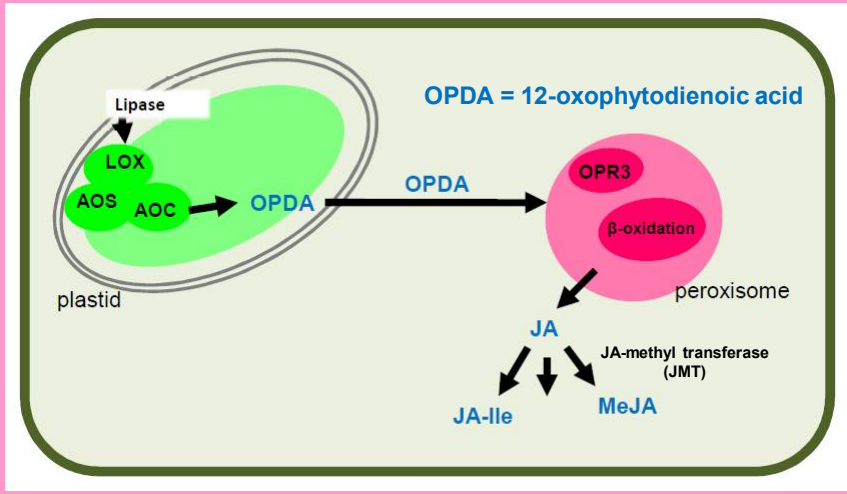
Methyl jasmonate is synthesized from jasmonic acid (JA)



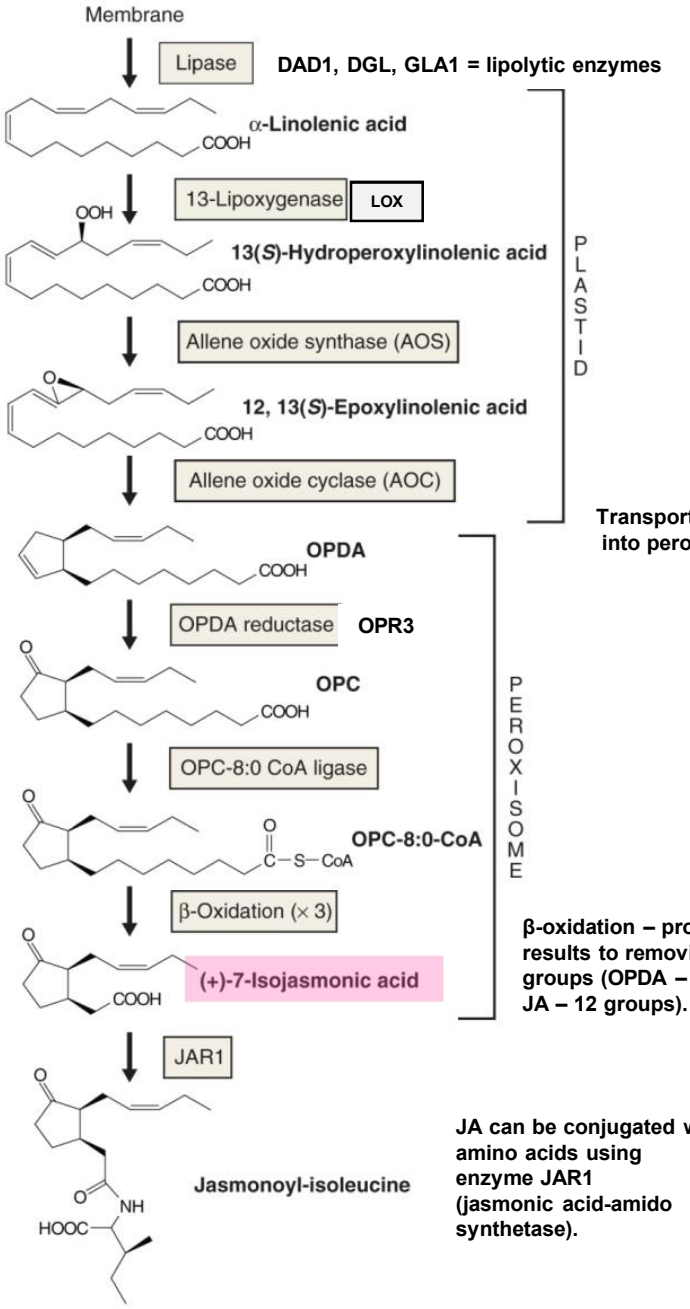
Jasmonoyl izoleucin (JA-Ile) is the most efficient jasmonate.
MeJA its transportable form of jasmonates.

JA biosynthesis

Precursors of jasmonates are derived from membrane lipids - free fatty acids.



Jasmonate synthesis occurs in the plastid, peroxisome and cytoplasm

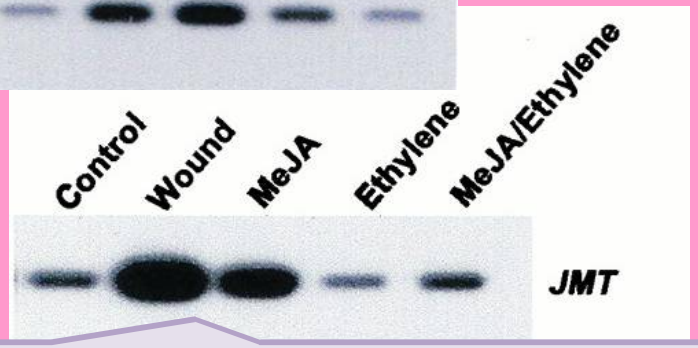


Physiological functions of JA

Production of MeJA is controlled developmentally and induced by injury.

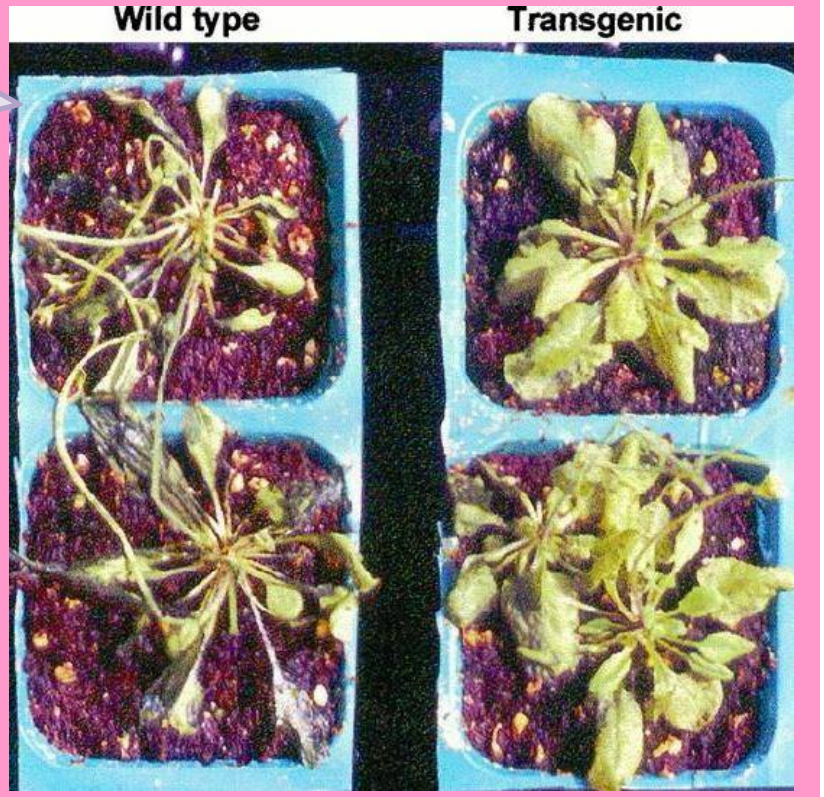
JMT expression peaks at anther dehiscence

JMT - JA-methyl transferase



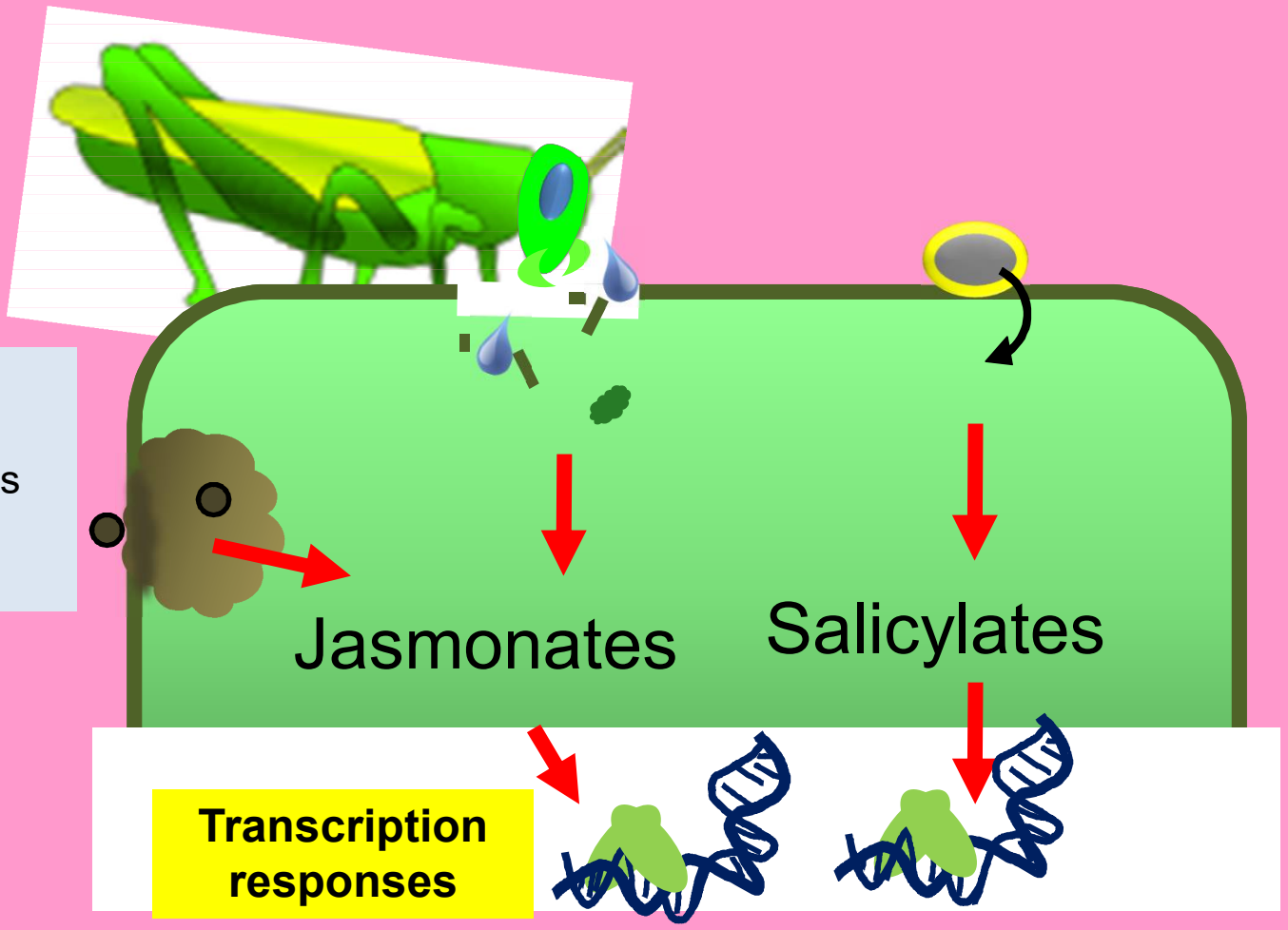
JMT is induced by wounding or MeJA

Increased MeJA production confers protection against necrotrophic pathogens



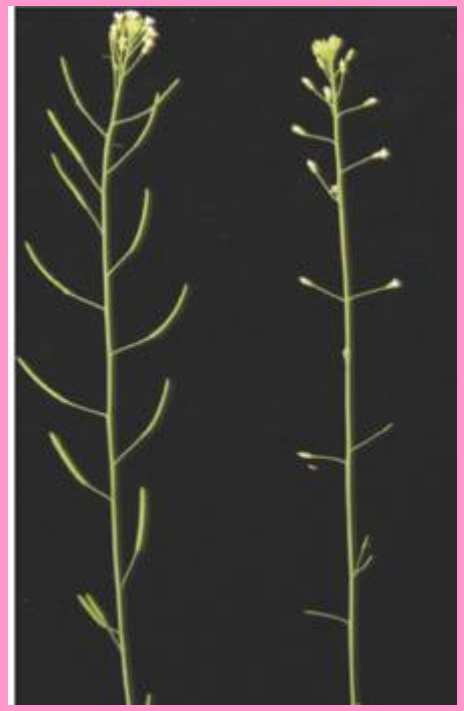
Jasmonates are involved in plant defense responses against insect and necrotrophs.

Insects and necrotrophs trigger jasmonate production, and biotrophs trigger salicylate production.



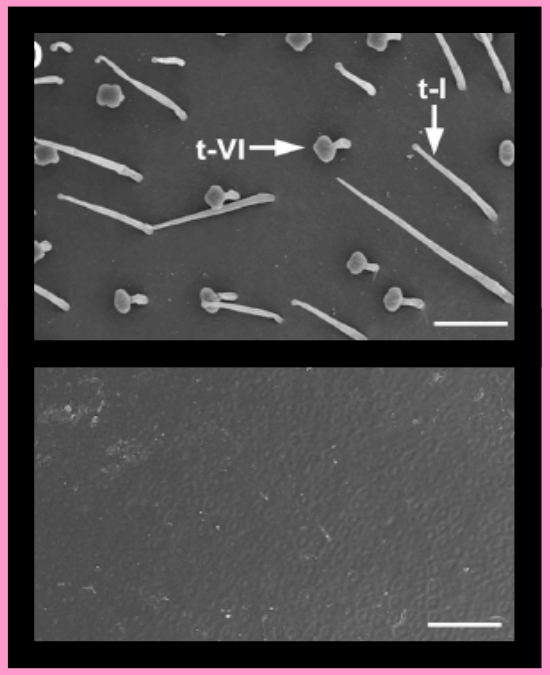
Jasmonates contribute to control of development and growth.

Male and female reproductive development



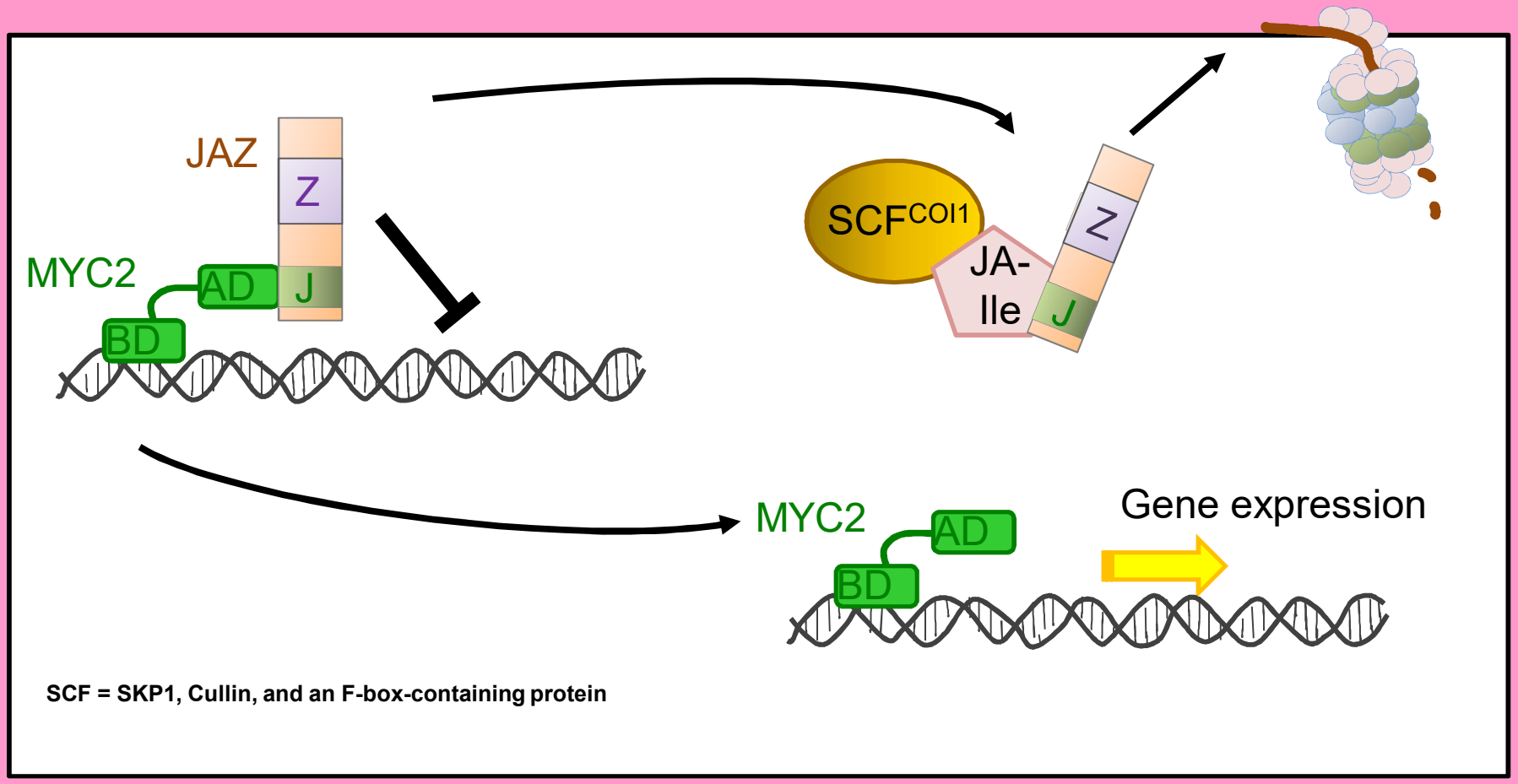
Control of cell cycle, senescence, and mutualistic interactions

Trichome formation



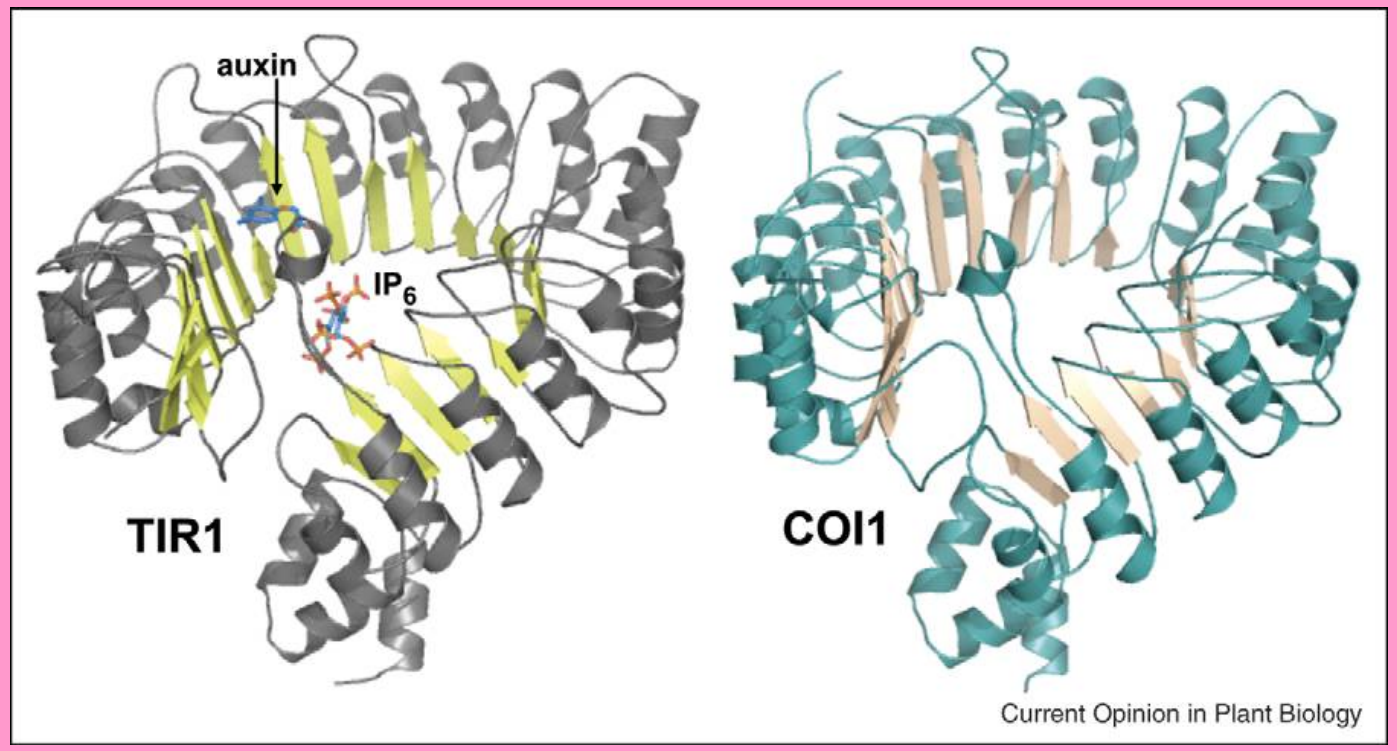
JA signaling

- JA-Ile binds to co-receptor COI1-JAZ (part of SCF complex)
- Ubiquitination and degradation of JAZ proteins occurs
- Release of transcriptional activation of transcription factors MYC2
- MYC2 activate expression of defense genes (coding for example proteins toxic for pests)



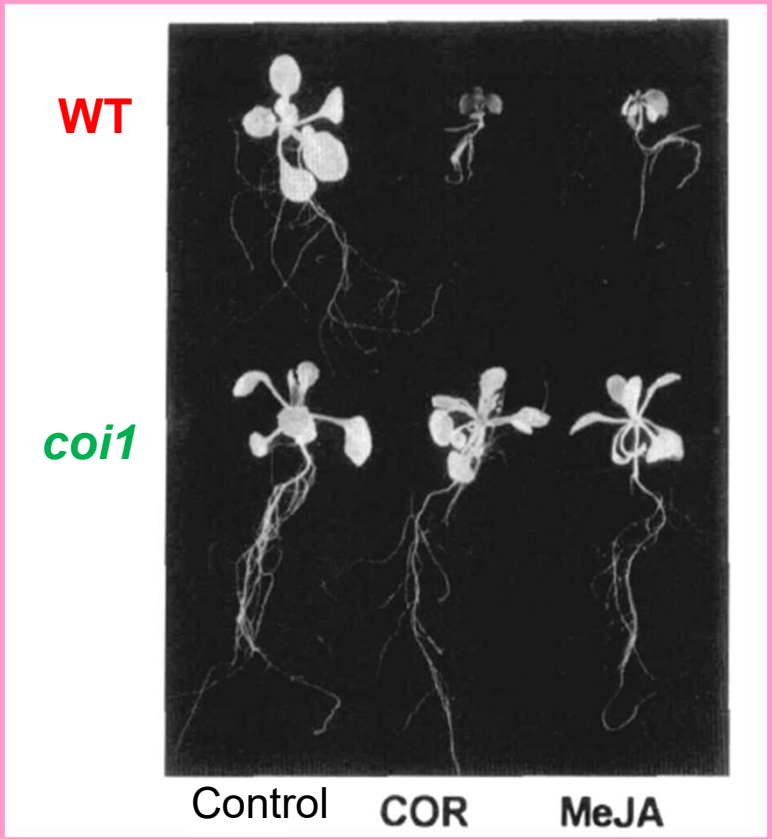
SCF = SKP1, Cullin, and an F-box-containing protein

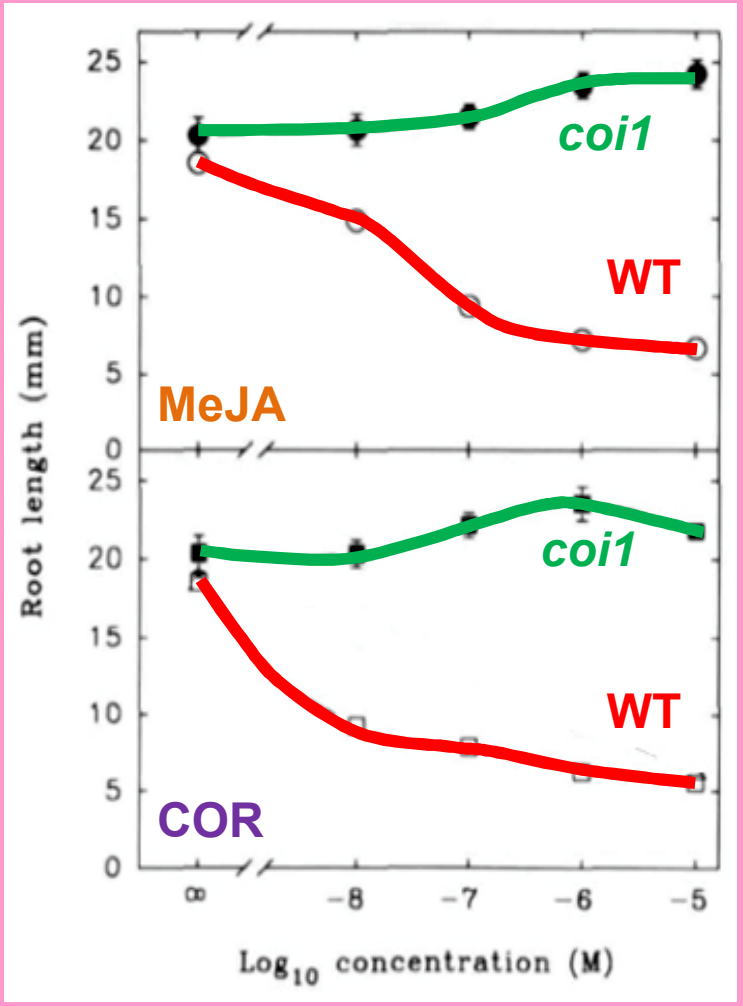
COI1 is F-box protein very similar to the auxin receptor TIR1.



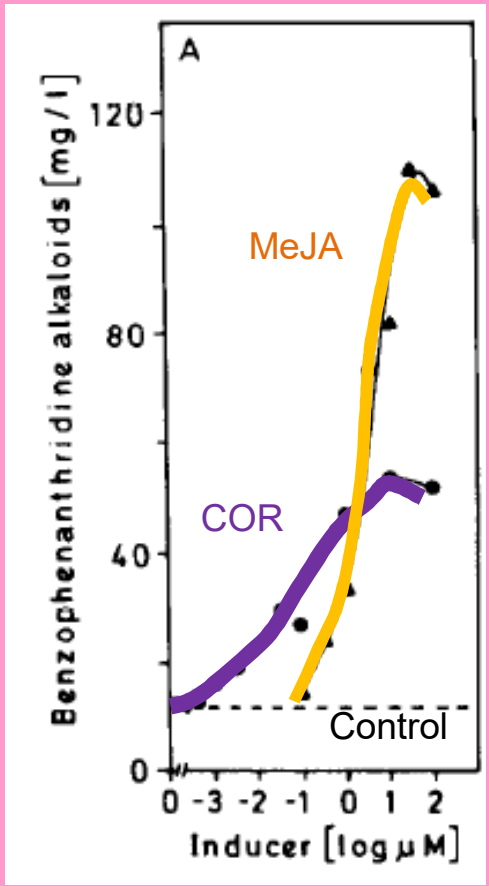
This similarity suggests a mode of action.

JA receptor COI was identified by analysis of *Arabidopsis* mutant *coi1*, which is insensitive to coronatine and MeJA





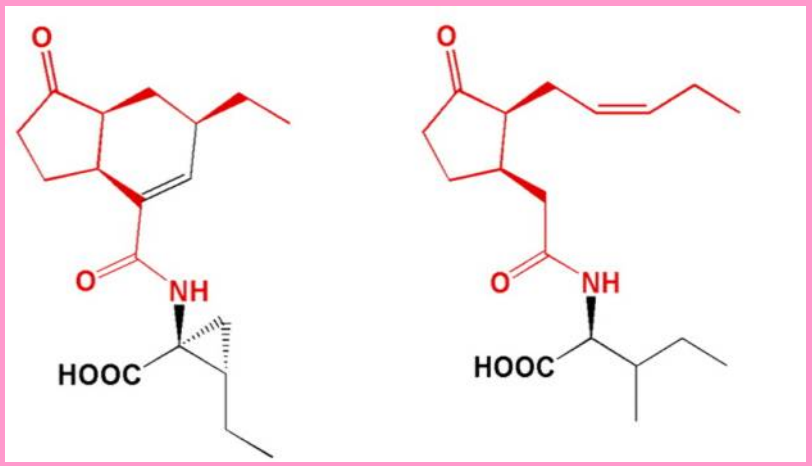
Coronatine is bacterial toxin that mimics effects of jasmonates.



MeJA or coronatine (COR) induce defense compounds in cultured cells of California poppy (kalifornského mák)

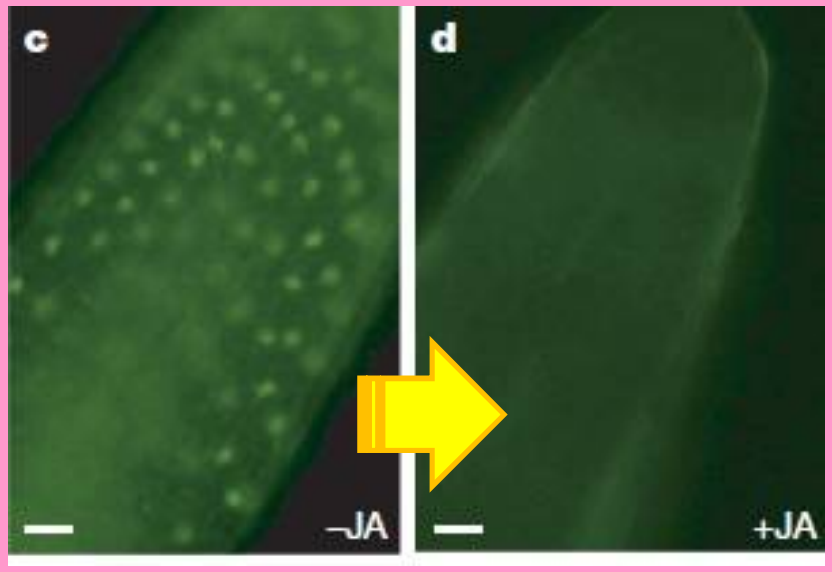
Eschscholzia californica – Sluncovka kalifornská

Coronatine is a toxin produced by some pathogenic bacteria that mimics jasmonate action and structurally resembles JA-Ile.

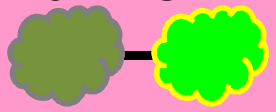


Coronatine **(3*R*,7*S*)-JA-Ile**

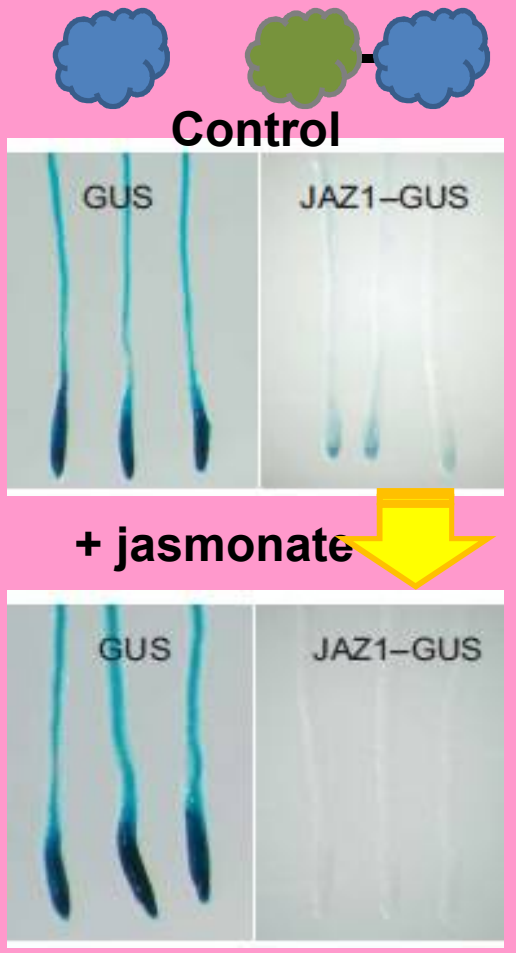
JAZ proteins are rapidly degraded in the presence of jasmonates.



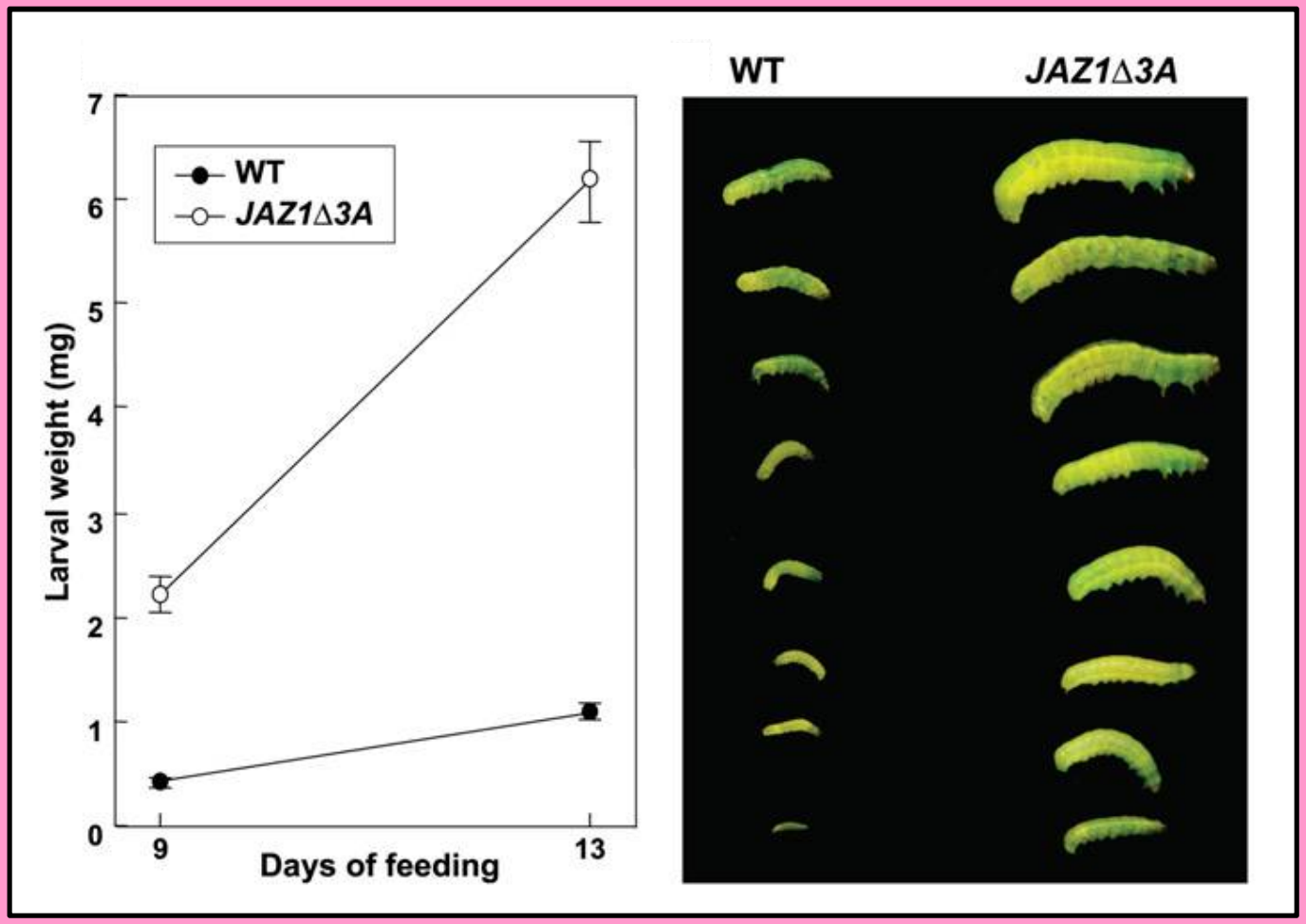
JAZ-GFP



Protein stability was assayed in transgenic plants expressing JAZ-GFP or JAZ-GUS fusions.

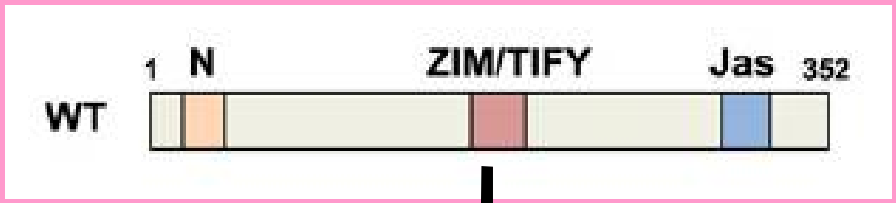


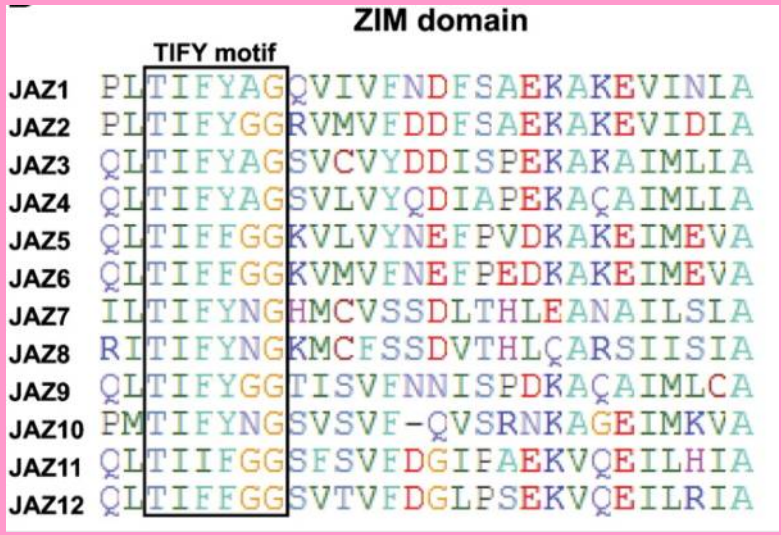
Plants expressing stabilized proteins JAZ lack defense responses against insect.



Plants with stabilized JAZ proteins have defects in defense responses. It is visible on the growth of caterpillar, which consumed these plants.

Proteins JAZ contain conserved domain Jas and domain ZIM/TIFY.

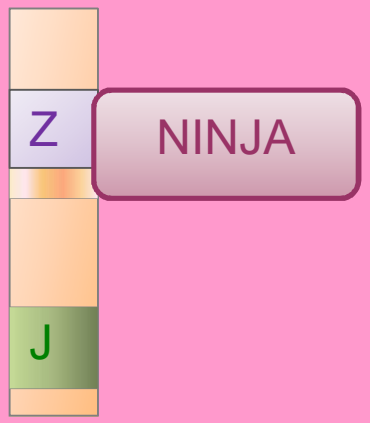




The **Jas** domains facilitate interactions with COI1 and MYC2 proteins. The **ZIM** domains (also called **TIFY** domains) are dimerization domains for interactions with other JAZ proteins and NINJA

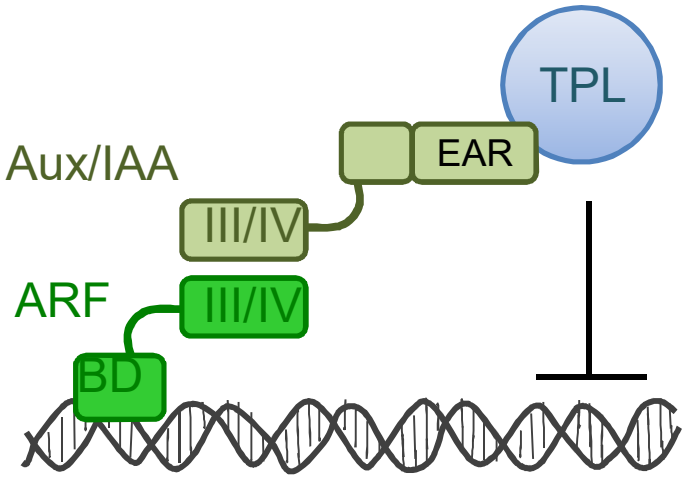
Domain ZIM of protein JAZ facilitates interaction with protein NINJA

NINJA = Novel
INteractor of JAZ

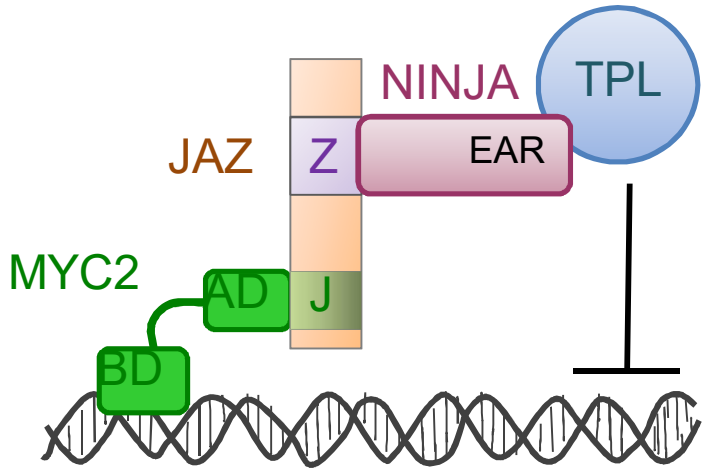


NINJA interacts with protein TOPLESS (TPL)

In the absence of hormones, protein TOPLESS suppress transcription.

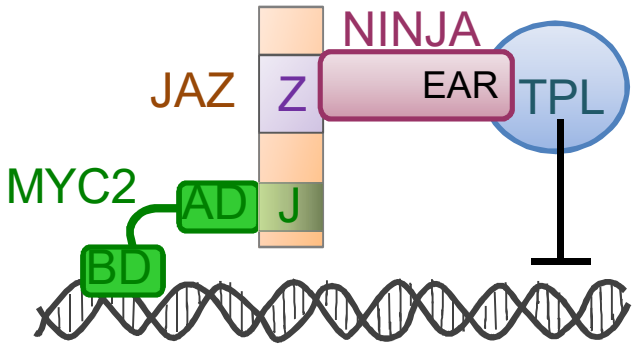


TOPLESS binds directly to EAR domain of repressor Aux/IAA.

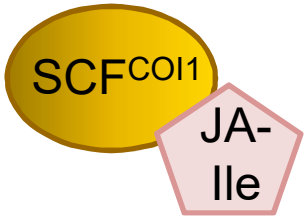


TOPLESS binds indirectly to JAZ through protein NINJA.

Jasmonate signaling overview



2. JAZ is ubiquitinated and degraded in 26S proteasome.



JA-Ile

1. JA-Ile binds to SCF^{COI1} and protein JAZ.

3. Degradation of repressor allows activation of transcription by transcription factors MYC2.