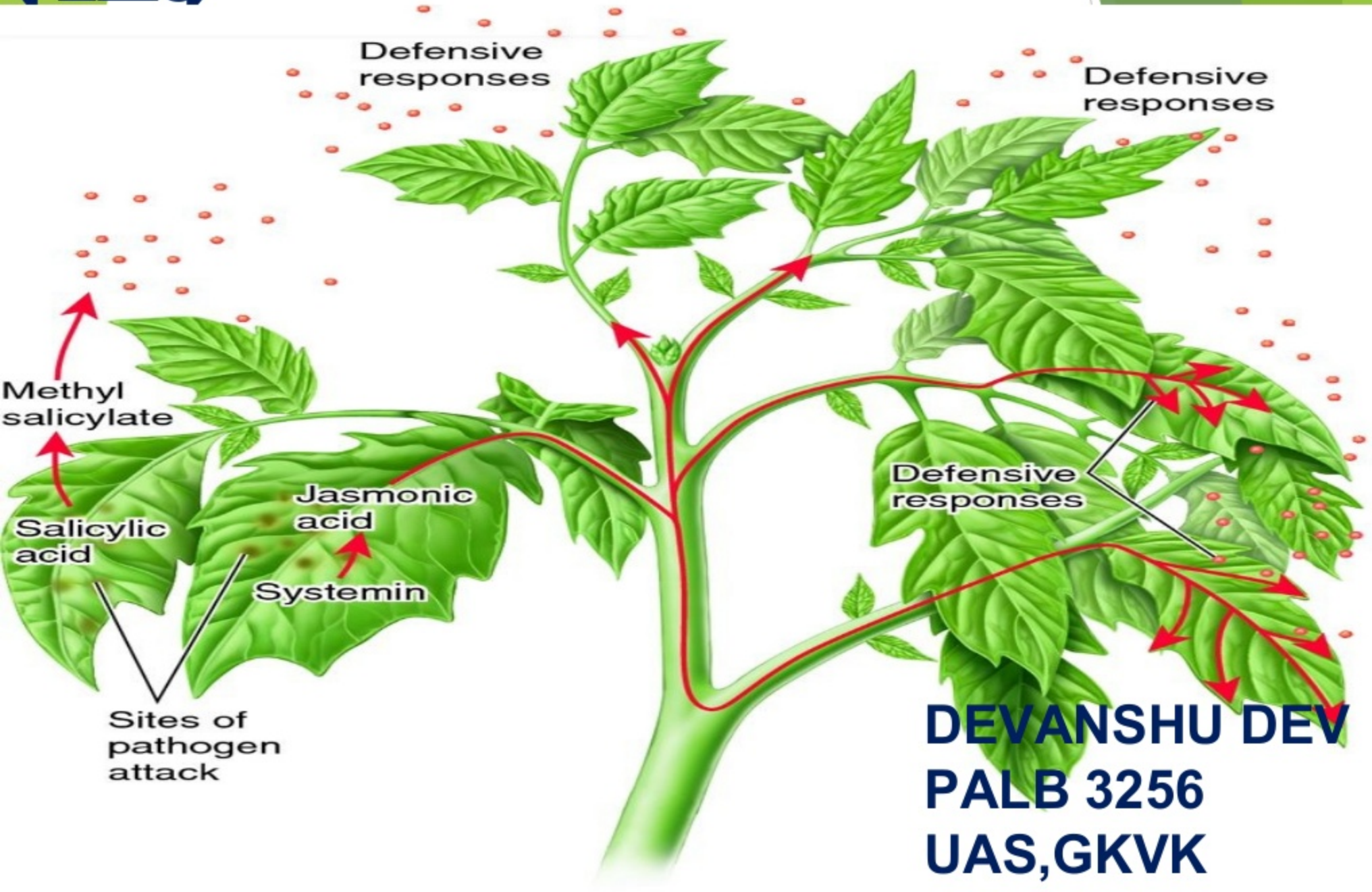
Induced Biochemical Defenses (HR)



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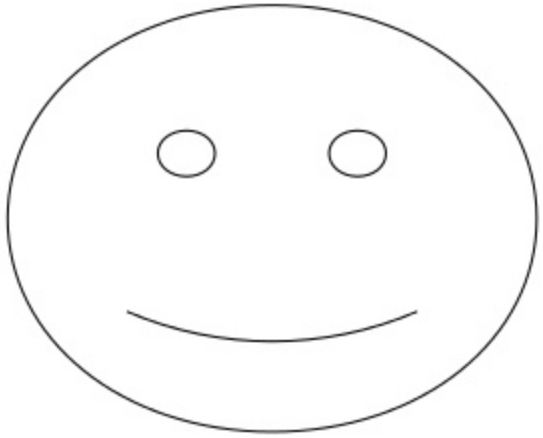
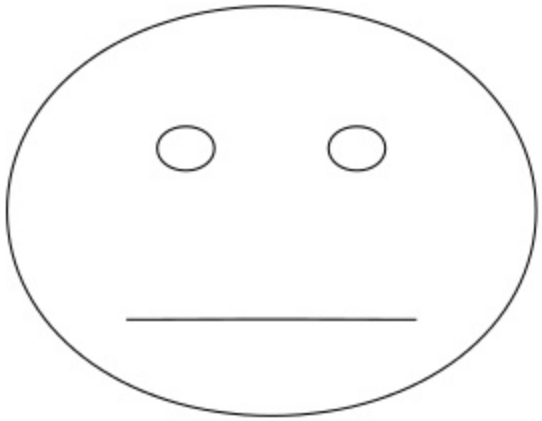
Plant defense response



Hypersensitive Response



Cell death

How do plants defend against bacteria that enter the cell?

- **Plants have a general response to infection**
  - anti-microbial molecules (secondary metabolites, phytoalexins)

- **Plants respond to specific infections through the Hypersensitive Response (PCD)** rapid accumulation of reactive oxygen species (directly kill pathogen)

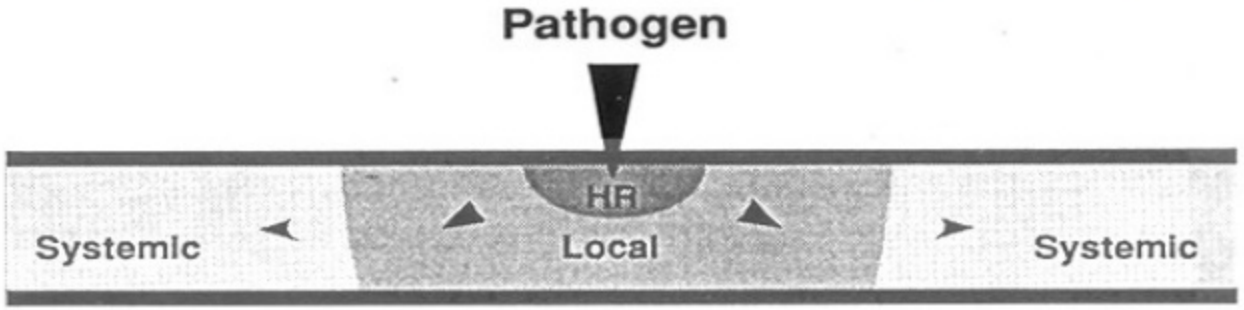
  - Induction of defense genes (pathogenesis-related proteins)

Plant Defense Response

- Hypersensitive response
- Production of reactive oxygen species
- Cell wall fortification
- Production of antimicrobial metabolites (phytoalexins)
- Defense signal transduction
- Synthesis of enzymes harmful to pathogen (eg. chitinases, glucanases)

Plant Defense Response

Compatible interaction → disease
Incompatible interaction → resistance



plant defense gene activation

3 aspects of response:

1. Hypersensitive
2. Local
3. Systemic

	Relative Timing
Responses of invaded cell(s) Browning/autofluorescence Oxidative reactions Callose deposition Intracellular rearrangements Hypersensitive cell death etc.	rapid
Local gene activation Phenylpropanoid pathways Pathogenesis-related (PR) proteins Phytoalexins etc.	rapid/ intermediate
Systemic gene activation 1.3-β-Glucanases Chitinases Other PR proteins etc.	intermediate/ slow

Elicitors of defense responses

- Any substance that has the capability of activating defense responses in plants
- Include components of the cell surface as well as excreted metabolites

Elicitors

General

a) Oligosaccharide elicitors

b) Protein/peptide elicitors

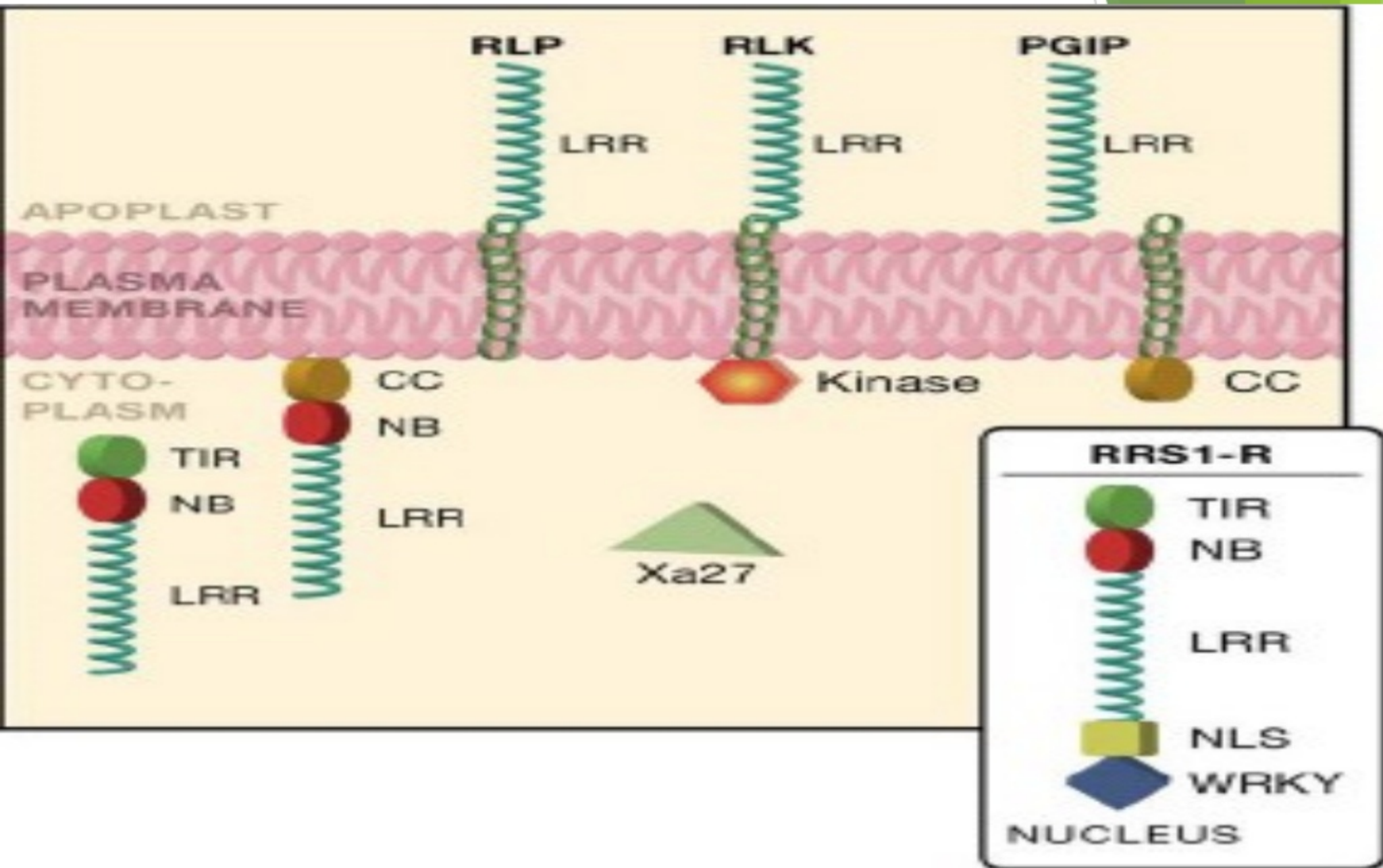
Race specific

a) *avr gene products*

Plant disease resistance genes

- Encode proteins that recognize *Avr*-gene-dependent ligands
- Activate signaling cascade(s) that coordinate the initial plant defense responses to impair pathogen ingress
- Capacity for rapid evolution of specificity
- Common feature of resistance proteins is a leucine-rich repeat

Classes of resistance proteins



Gene-for-gene resistance

- For resistance to occur, complementary pairs of dominant genes, one in the host and the other in the pathogen, are required (incompatibility)
- A loss or alteration to either the plant resistance (*R*) gene or the pathogen avirulence (*Avr*) gene leads to disease (compatibility)

Interactions involved in *R* gene -*Avr* gene incompatibility

		Host plant genotype	
		R1 r2	*r1 R2*
Pathogen genotype	*Avr1, avr2*	I	C
	avr1, Avr2	C	I

Deviations from gene-for-gene concept

- One R gene may confer specificity to more than one ligand
  - *RPM1* in Arabidopsis confers resistance against *P.syringae* expressing either avrRpm1 and avrB

- More than one R gene may exist for a given *Avr* gene
  - *Pto* and *Prf* genes encode biochemically distinct components of the same pathway
  - Two genes at the *Cf-2* locus furnish identical functions

(Bent, 1996)

Guard hypothesis

- Key points

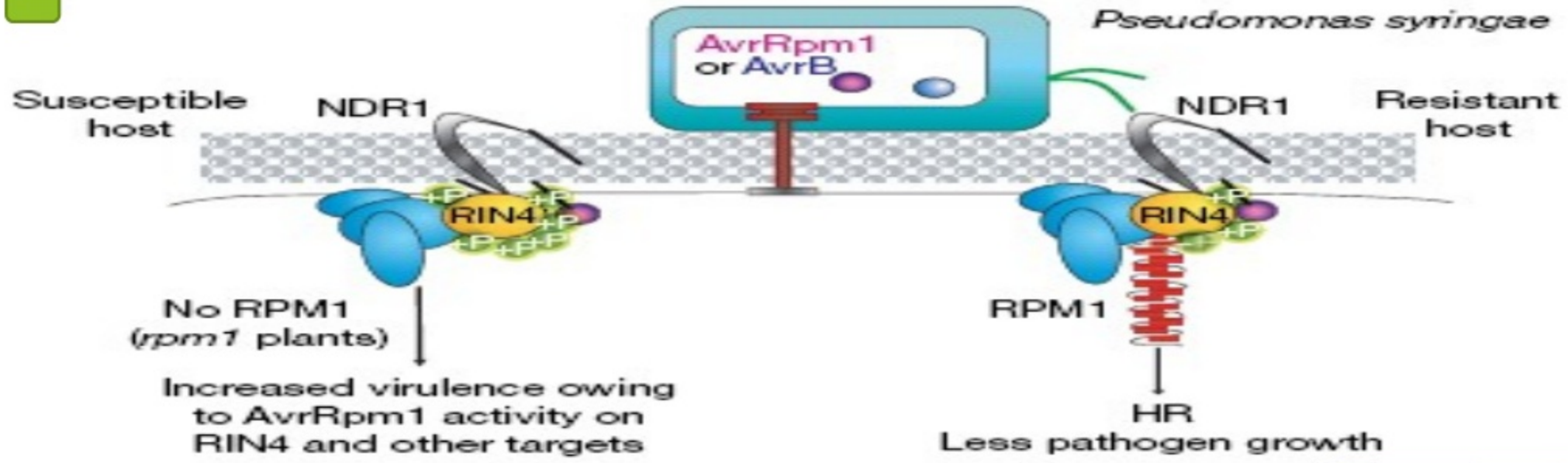
a) An effector acting as a virulence factor has a target(s) in the host

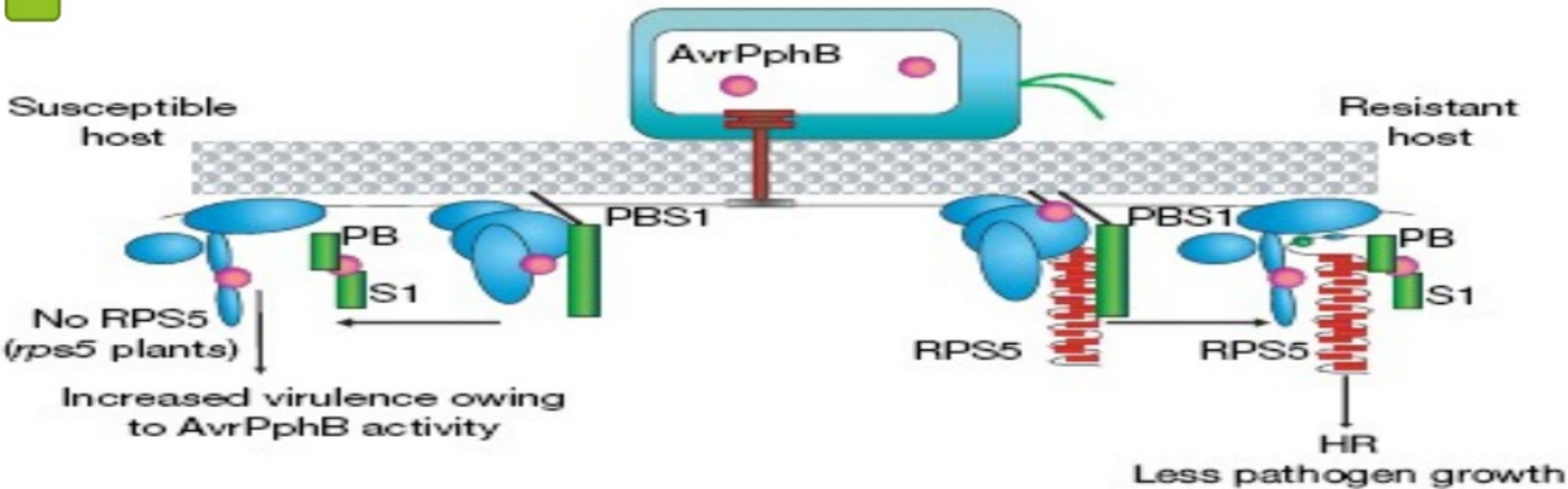
b) By manipulating or altering this target(s) the effector contributes to pathogen success in susceptible host genotypes

c) Effector perturbation of a host target generates a “pathogen induced modified self” molecular pattern, which activates the corresponding NB-LRR protein, leading to ETI

(Jones *et al.*,2006)

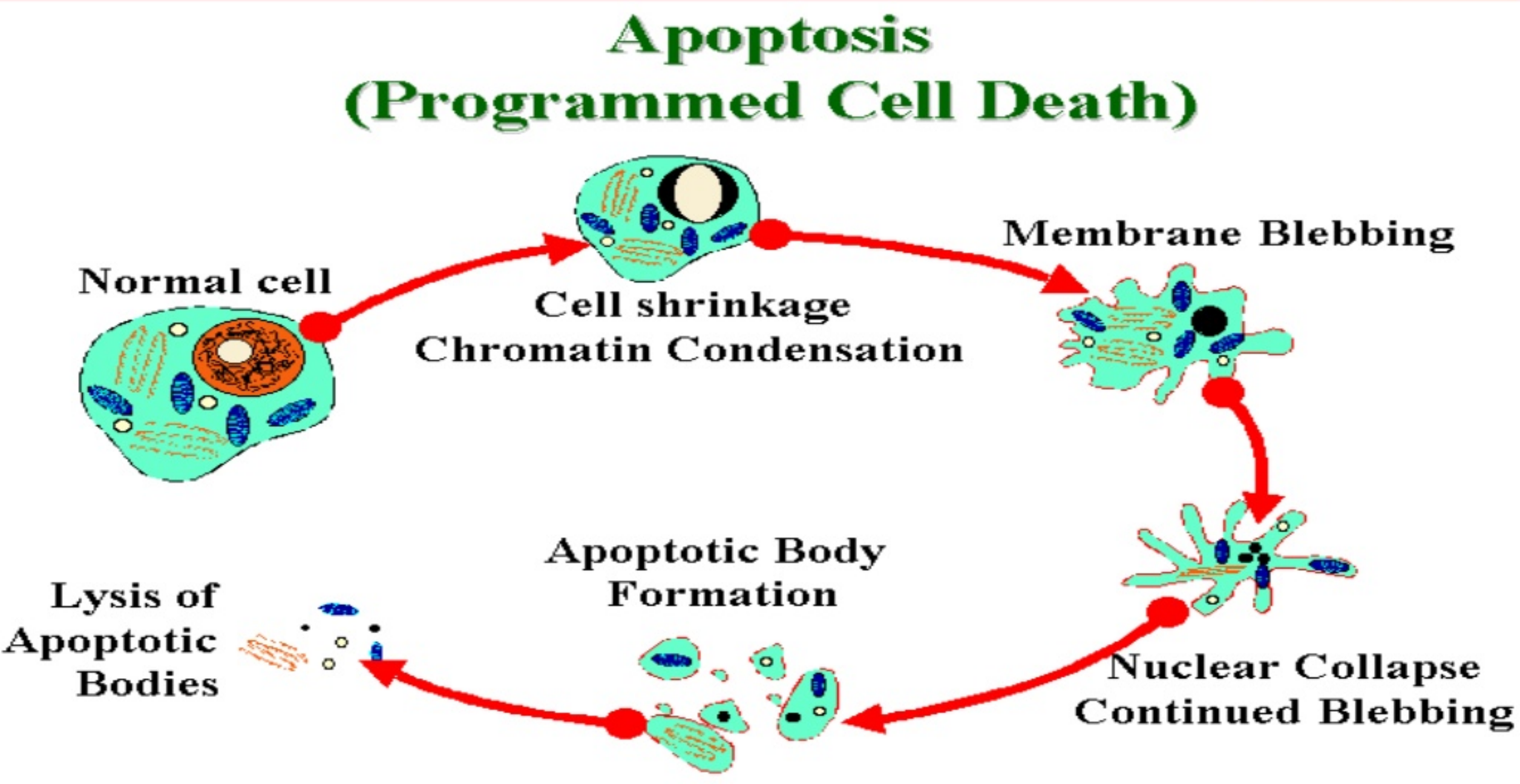
Guard hypothesis



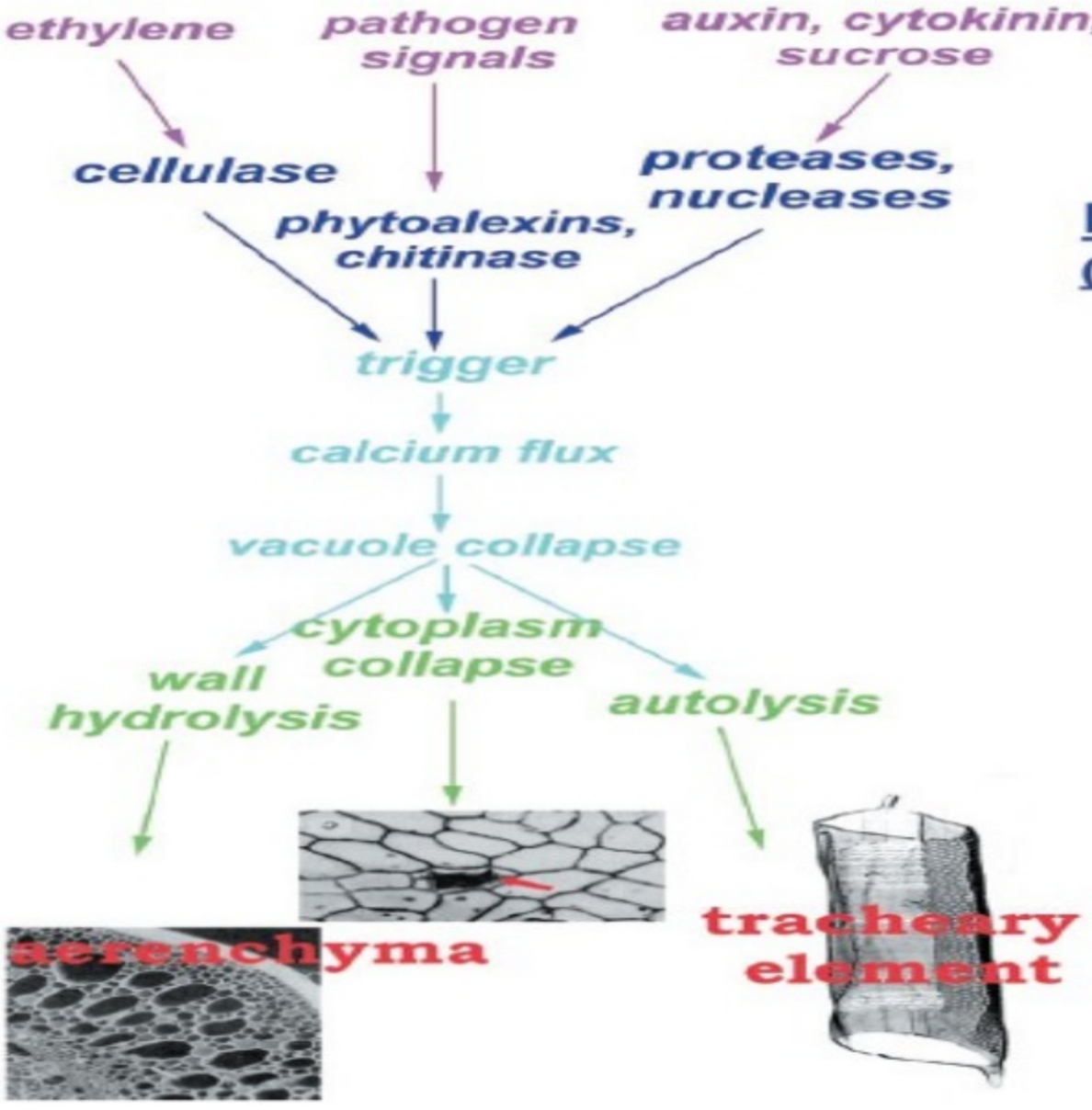


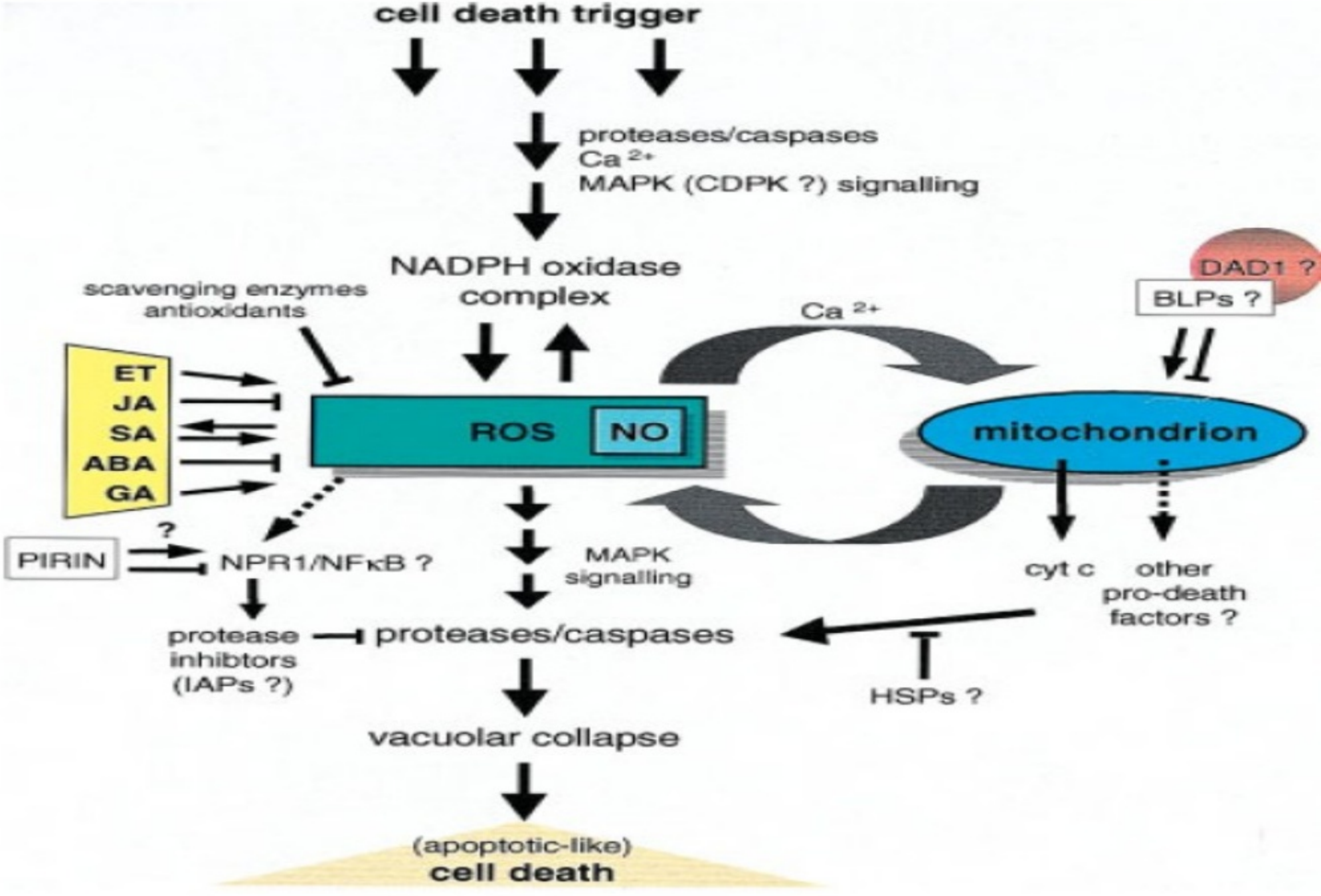
Programmed cell death

- Programmed cell death is a genetically regulated process of cell suicide that is central to the development, homeostasis and integrity of multicellular organisms

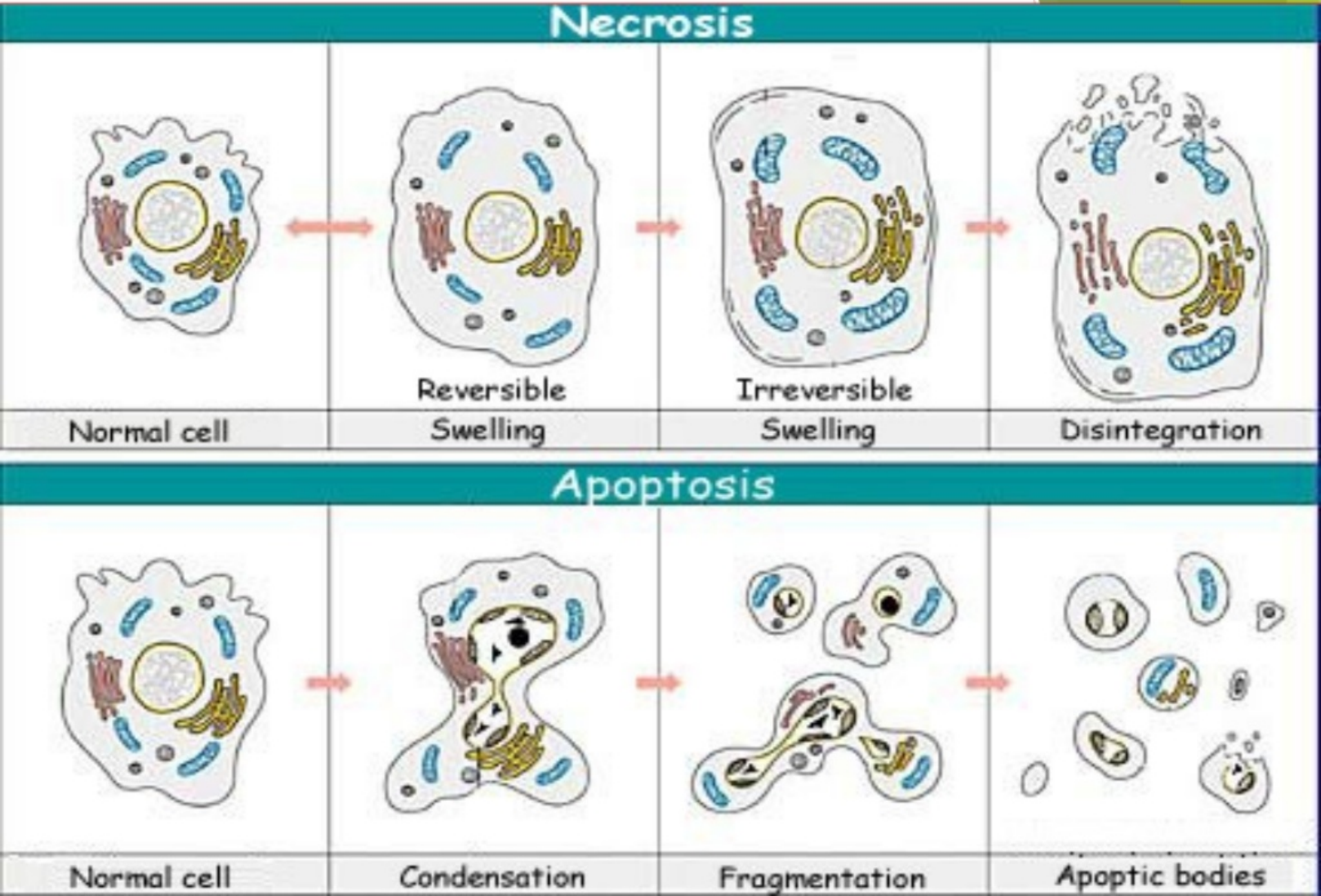


General mechanism of three PCDs in plants




cell death trigger
proteases/caspases
Ca 2+
MAPK (CDPK ?) signalling
NADPH oxidase complex
scavenging enzymes antioxidants
DAD1 ?
BLPs ?
Ca 2+
ET
JA
SA
ABA
GA
ROS
NO
mitochondrion
?
PIRIN
NPR1/NFκB ?
MAPK signalling
cyt c
other pro-death factors ?
protease inhibtors (IAPs ?)
proteases/caspases
HSPs ?
vacuolar collapse
(apoptotic-like) cell death

Necrosis vs apoptosis



Purpose of cell death

- Cells that are produced in excess
- Cell that have no function
- Cells that are produced in excess
- Cell that develop improperly
- Cell that have finished their function
- Cells that are harmful

Hypersensitive response

- Rapid, localized plant cell death upon contact with avirulent pathogens. HR is considered to be a key component of multifaceted plant defense responses to restrict attempted infection by avirulent pathogens
- Rapid - within 24 h
- Not always needed for resistance
- HR also contributes to the establishment of the long-lasting systemic acquired resistance against subsequent attack by a broad range of normally virulent **pathogens**

- HR Includes:
  - oxidative bust (production of reactive oxygen species)
  - Disruption of cell membranes
  - opening of ion channels
  - Cross linking of phenolics with cell wall component
  - Production of anti-microbial phytoalexins and PR protein
  - apoptosis (programmed cell death)

The Hypersensitive Response

- Bacteria like *Pseudomonas syringae* inject effector proteins (bacterial avirulence and virulence proteins) into plant cells using the Type-III secretion system.

- Plants that are resistant to the bacteria have resistance proteins that recognize the effector proteins and cause the infected cell to commit suicide (**apoptosis/PCD/Hypersensitive Response**)

- prevents the bacteria from infecting the rest of the plant by directly killing them and depleting nutrients