

**QUIESCENCE OF POSTHARVEST PATHOGENS:
A FUNGAL INHIBITION PROCESS OR
AN IMMUNE RESPONSE OF THE UNRIPE HOST
FRUIT?**

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Postharvest fungal infections

Are initiated before, during, or after harvest, but often remain dormant in a quiescent stage until fruit ripening and senescence

Different from pathogens that show a full cycle in the developing plant

Ustilago tritici

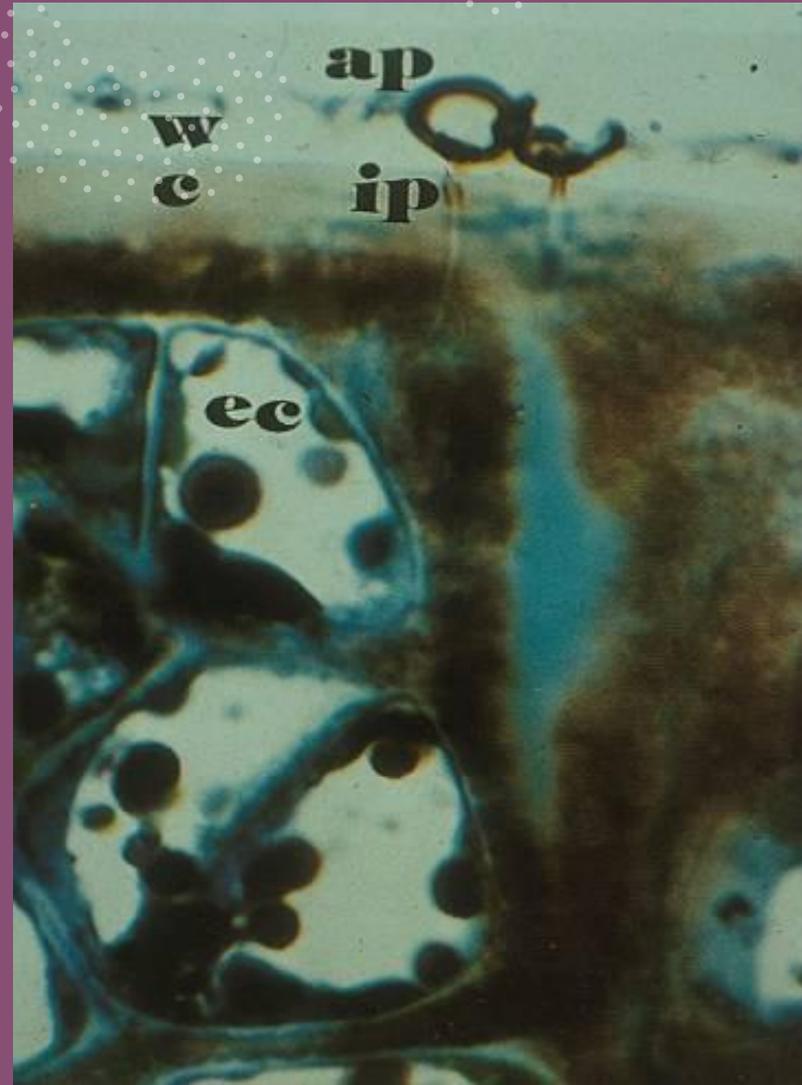


Ustilago maydis



Postharvest pathogens infect fruits and vegetables through two main mechanisms:

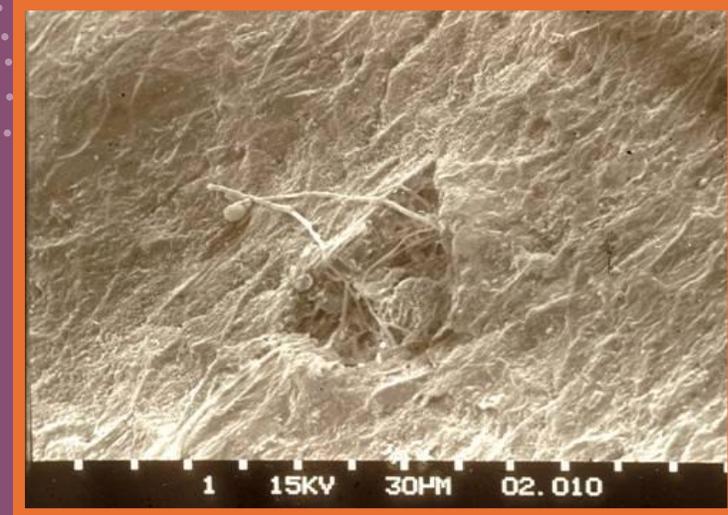
Direct penetration *Colletotrichum*



Postharvest pathogens infect fruits and vegetables through two main mechanisms:

Wound penetration in *Alternaria*

a. Lenticel penetration

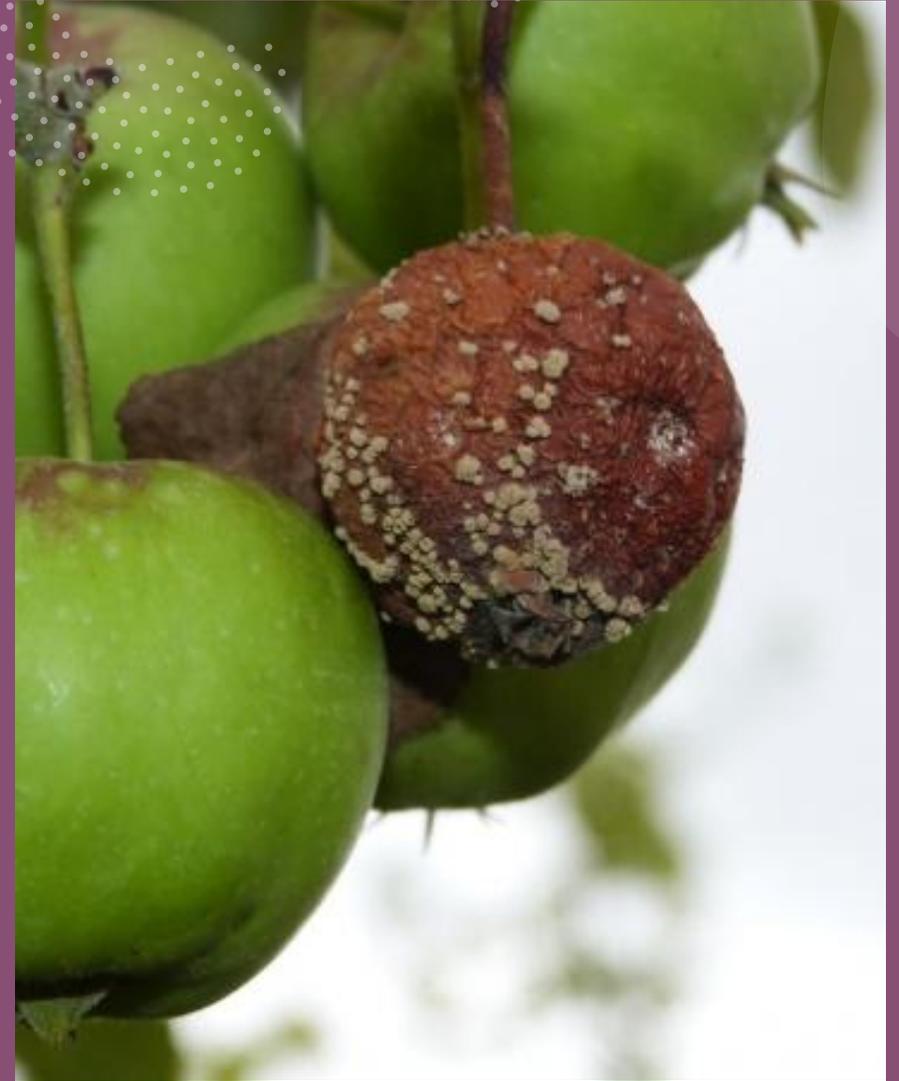


b. Initial symptoms



Postharvest pathogens infect fruits and vegetables through two main mechanisms:

Wound penetration in Monilinia





During fruit development until harvest conditions, the infections remain quiescent



Symptoms usually are not visible until prolonged cold storage or during shelf life, just before consumption

Symptoms usually become visible only after fruit maturation, with prolonged cold storage or during shelf life, just before consumption

Colletotrichum



Botrytis

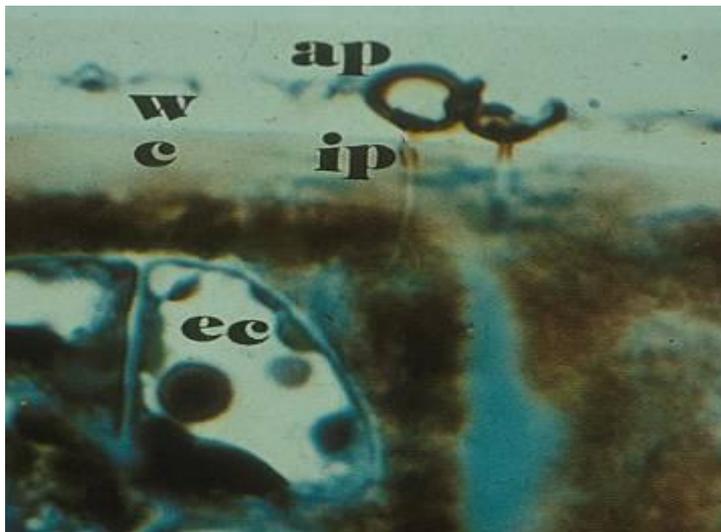
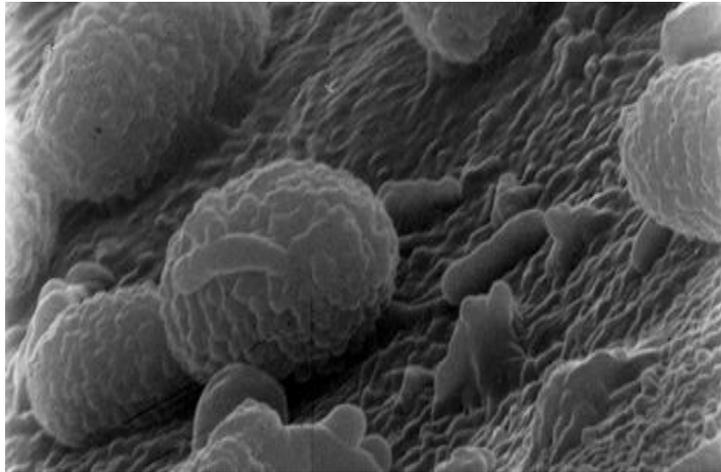


Monilinia

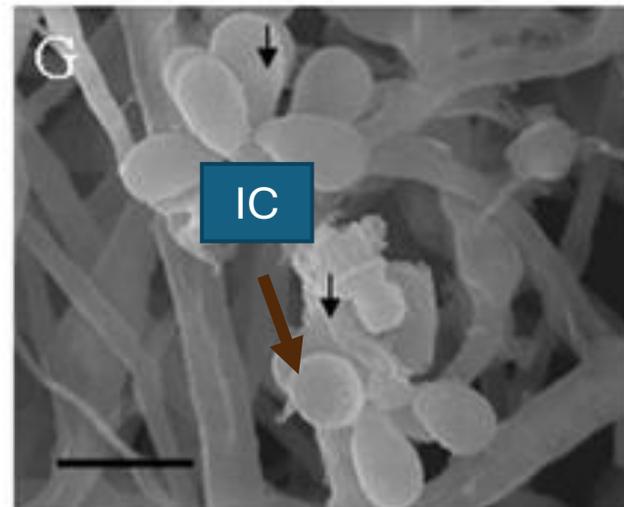
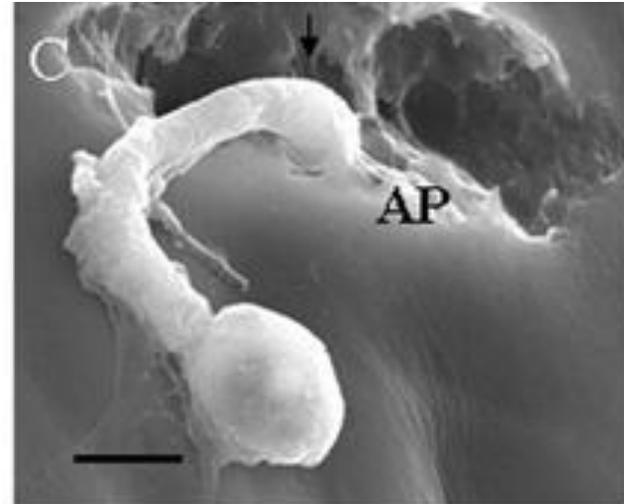


Infection mechanisms of postharvest pathogens that become quiescent

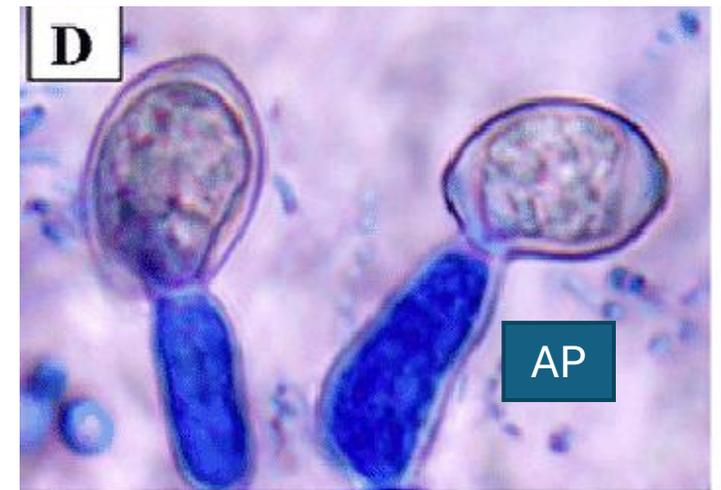
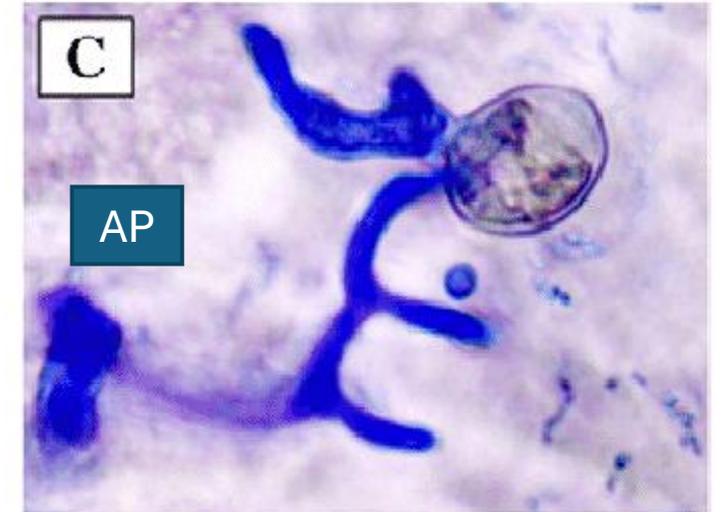
Colletotrichum



Botrytis



Monilinia



The road map of the infection of Botrytis in leaves until the first line of defense in plants, (**P**attern-**T**riggered Immunity, PTI), which imparts a baseline level of disease resistance during the first 48 h after infection, includes:

Confirmed processes:

C-Cell death-inducing proteins (CDIPs)

E-Enzymes

T- toxins,

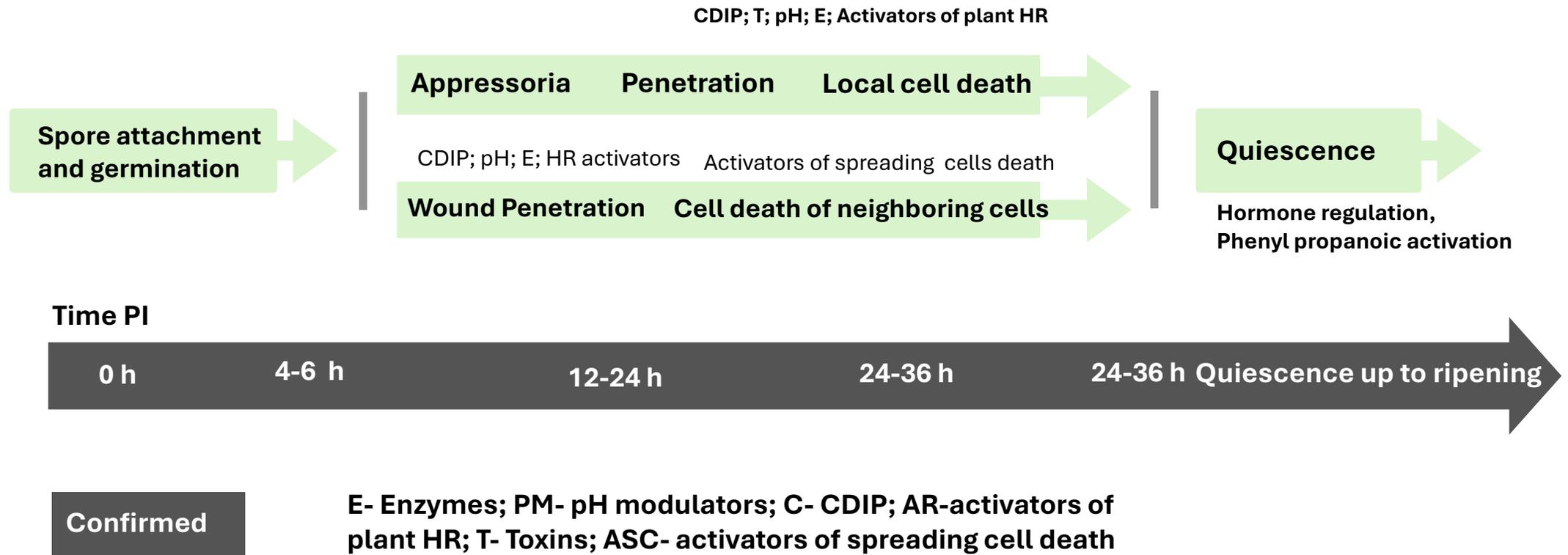
PM- pH modulators,

AR- activators of plant HR

ASC-activators of spreading cell death

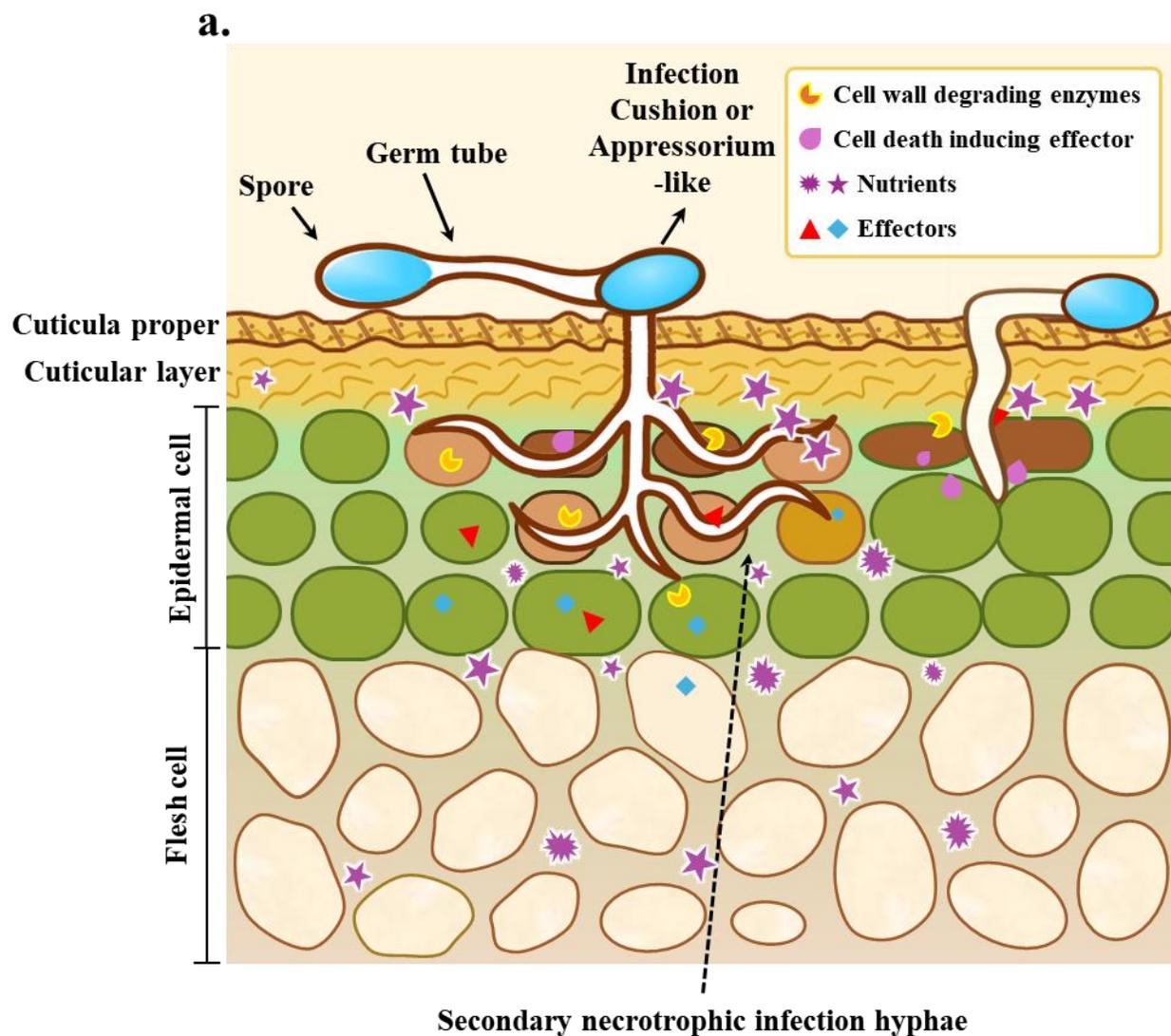
SAR-Suppressors of autophagy

A series of processes during the early quiescence of Botrytis in strawberries and grapes

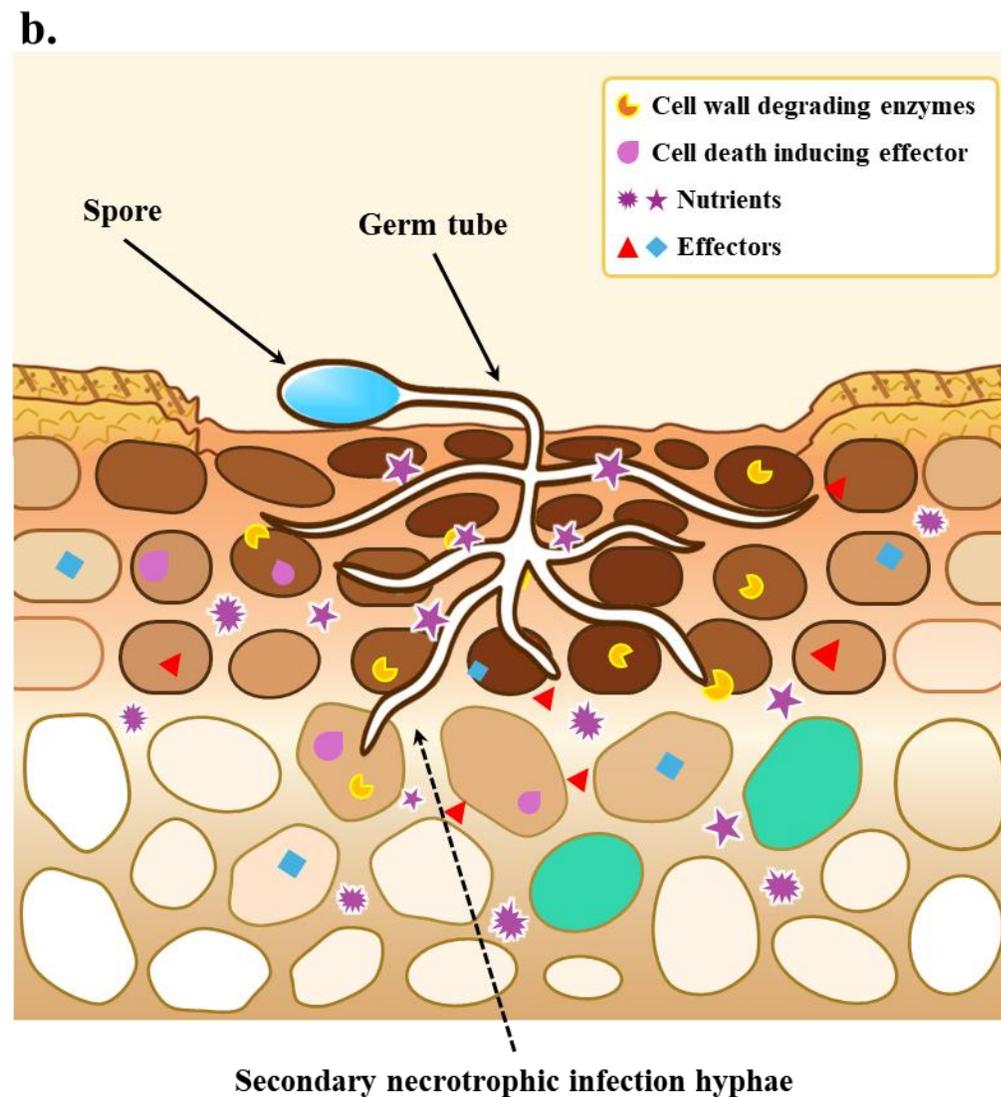


Quiescence of *Botrytis cinerea* with a very short biotrophic stage

DIRECT CUTICLE PENETRATION



WOUND PENETRATION



The road map of infection of Botrytis in leaves during the first 48 h includes:

Confirmed processes:

C-Cell death-inducing proteins (CDIPs)

E-Enzymes

T- toxins,

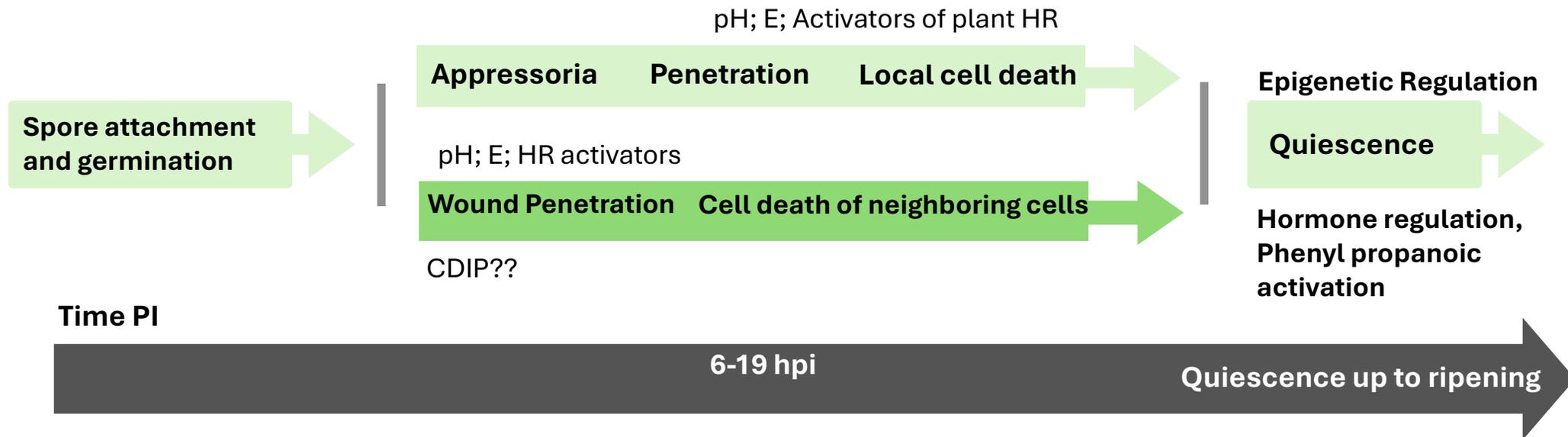
PM- pH modulators,

AR- activators of plant HR

ASC-activators of spreading cell death

SAR-Suppressors of autophagy

A series of processes during early quiescence in Colletotrichum



Confirmed

E- Enzymes; PM- pH modulators; AR- activators of plant HR;

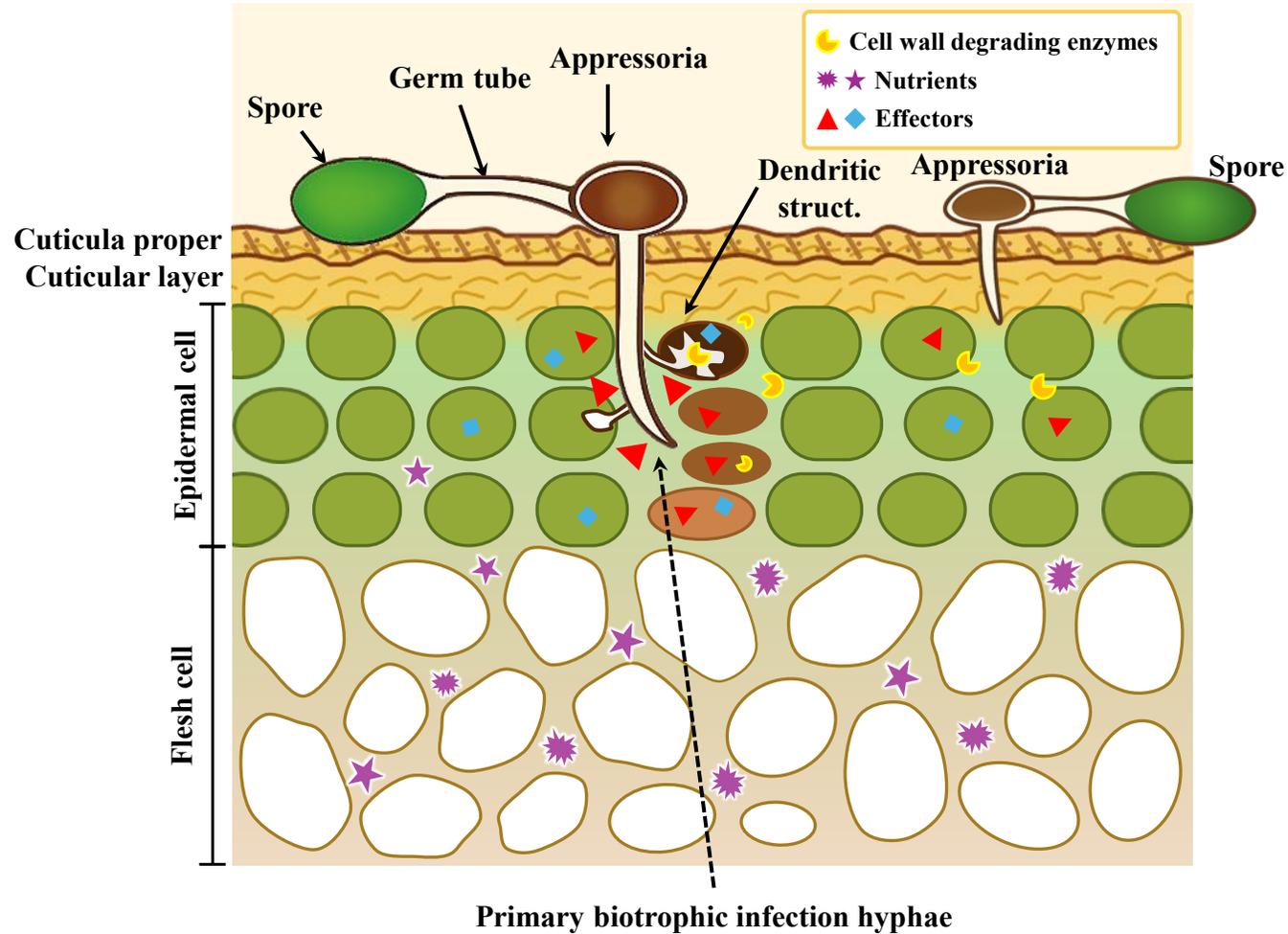
Most likely

C-Cell Death-Inducing Proteins

Quiescence of *Colletotrichum* a pathogen with long biotrophic stage

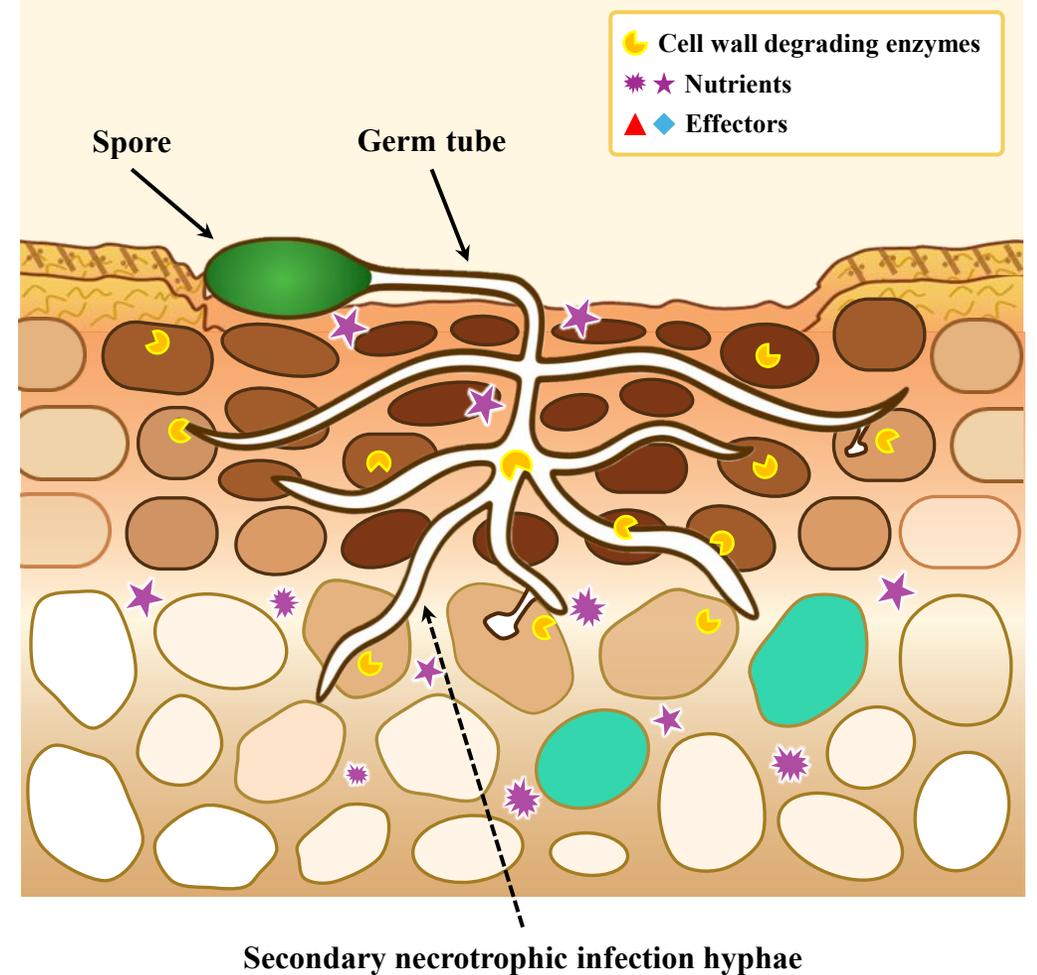
DIRECT PENETRATION OF THE CUTICLE

a.

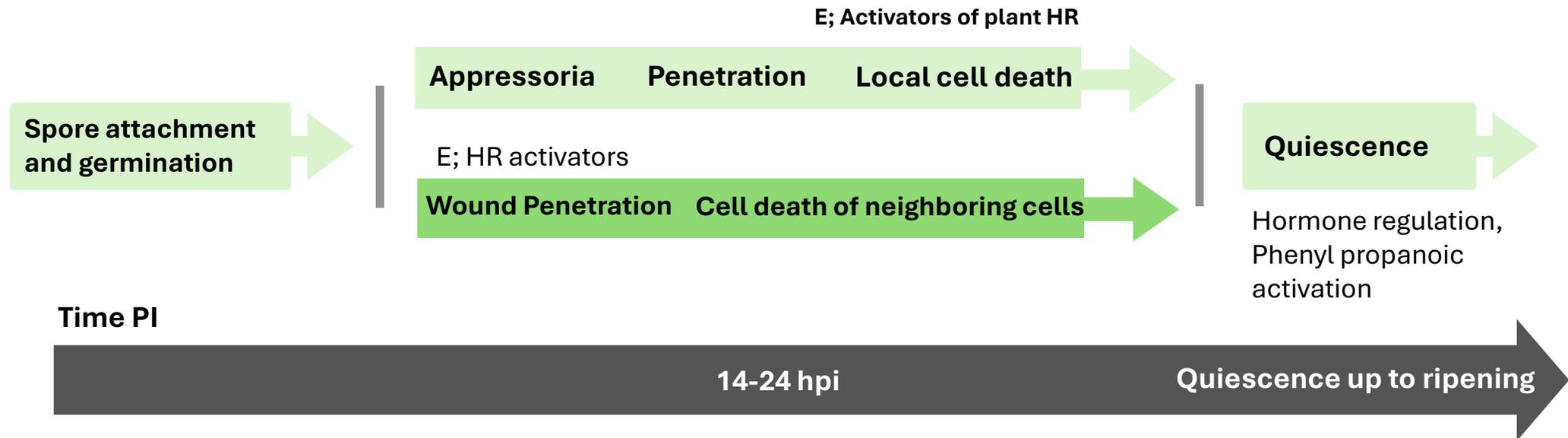


WOUND PENETRATION

b.



A series of processes during early quiescence in Monilinia



Confirmed

E- Enzymes; AR-activators of plant HR

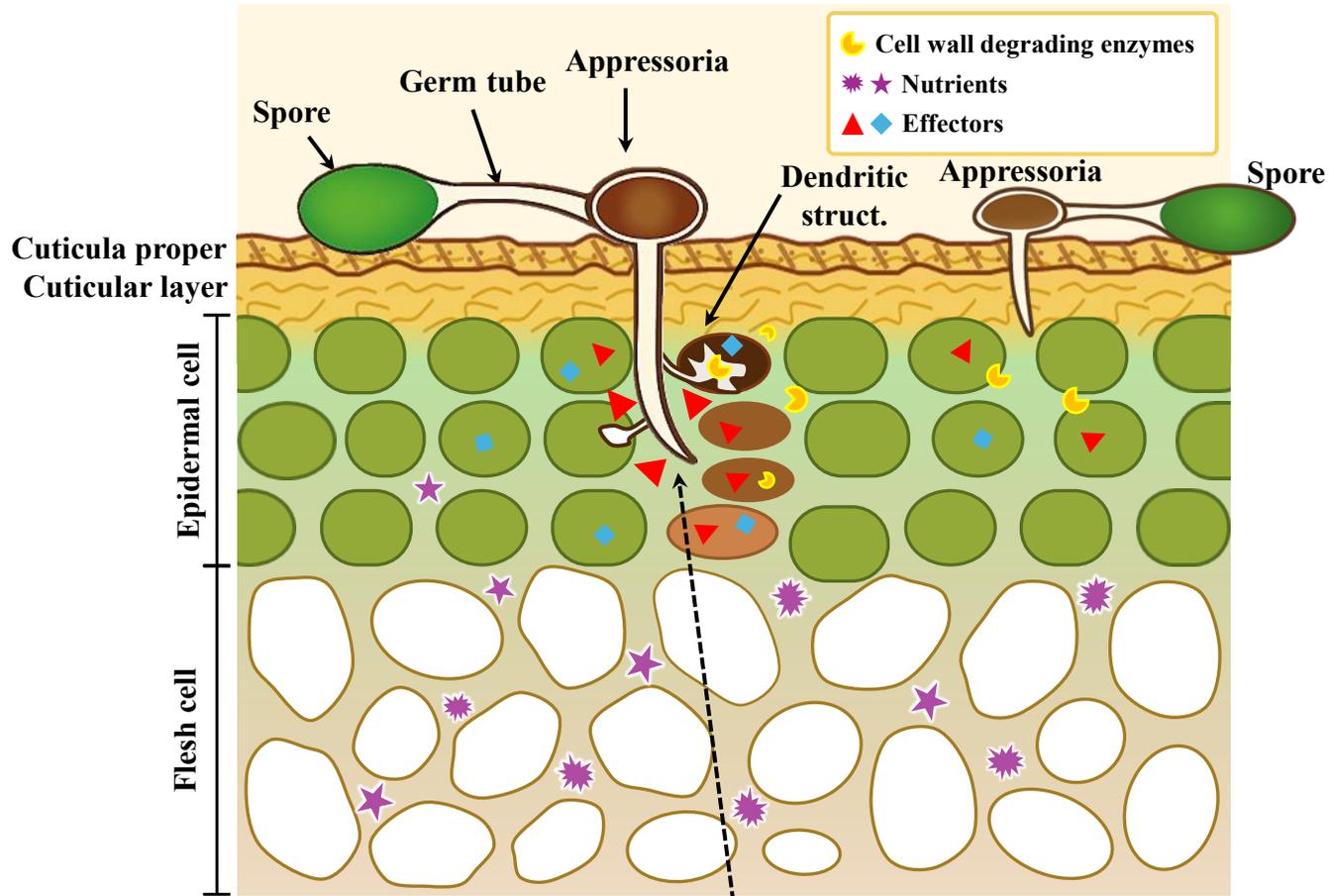
Most likely

pH regulation

Quiescence of *Monilinia* a pathogen with a short biotrophic stage

DIRECT PENETRATION OF THE CUTICLE

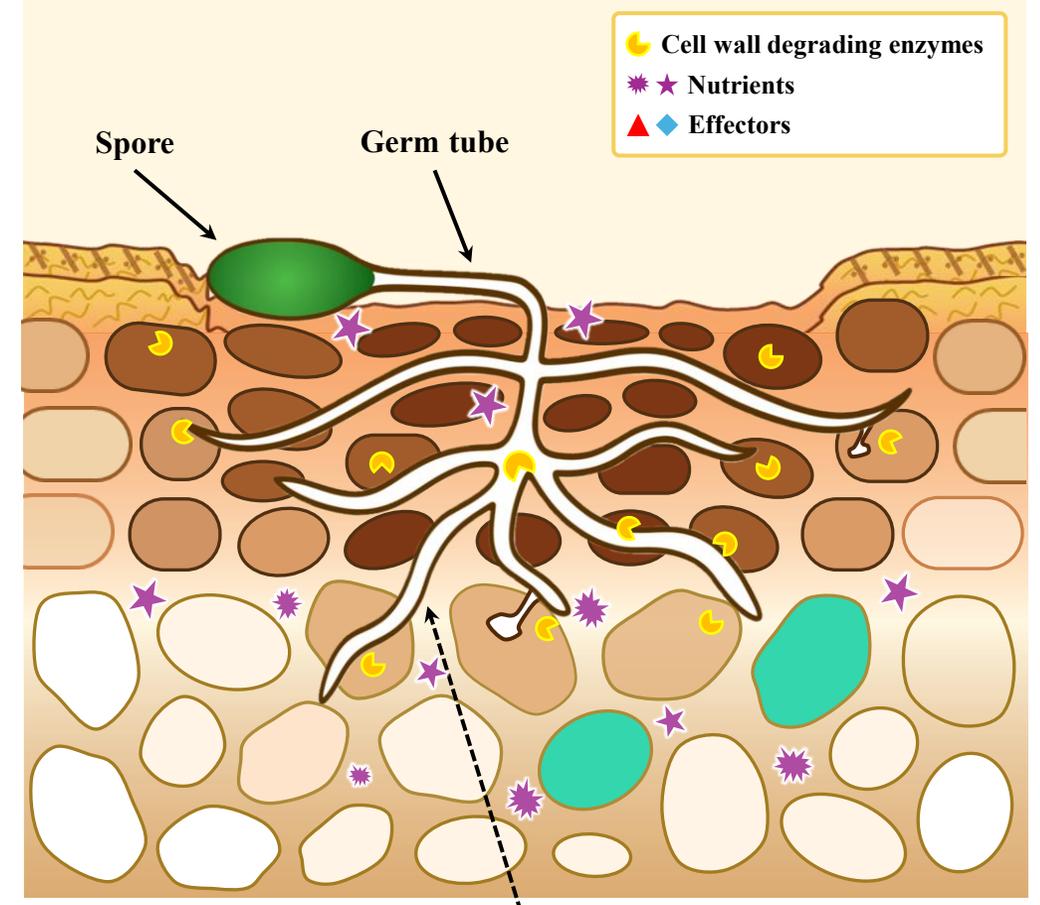
a.



Primary biotrophic infection hyphae

WOUND PENETRATION

b.



Secondary necrotrophic infection hyphae

(Balsells-Llauradó et al., 2020; 2022a, b).

In summary:

Postharvest pathogens first detect the host and then regulate diverse metabolic processes to:

- (i) Develop penetration structures
- (ii) Identify optimal entry sites
- (iii) Equip these structures with cutinase and lipases to breach the host cuticle, ensuring successful quiescence
- (iv) Inhibition of fungal colonization and host immunity until fruit ripening.

In conclusion

- These findings suggest that fruit immunity during quiescence is characterized by a rapid and synergistic interacting-processes, not all of which are described.
- They are differentially regulated across host species and developmental stages, including fruit maturity and ripening.
- This coordinated regulation enhances disease resistance and initiates a cascade of downstream defense responses, leading to fruit decay

Therefore, understanding this host-pathogen mechanism during
quiescence
and its activation proves critical for the prevention of fruit decay
throughout postharvest storage life

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