ANNUAL REVIEWS

Annual Review of Phytopathology

Induced Resistance in Fruit and Vegetables: A Host Physiological Response Limiting Postharvest Disease Development

Dov Prusky[1] and Gianfranco Romanazzi[2]

[1]Department of Postharvest Science, Agricultural Research Organization, The Volcani Institute, Rishon LeZion, Israel; email: dovprusk@agri.gov.il

[2]Department of Agricultural, Food and Environmental Sciences, Marche Polytechnic University, Ancona, Italy; email: g.romanazzi@univpm.it

Annu. Rev. Phytopathol. 2023. 61:279–300

First published as a Review in Advance on May 18, 2023

The *Annual Review of Phytopathology* is online at phyto.annualreviews.org

https://doi.org/10.1146/annurev-phyto-021722-035135

Keywords

fruits, vegetables, induced resistance, physiological factors, postharvest, ripening, maturation delay

Abstract

Harvested fruit and vegetables are perishable, subject to desiccation, show increased respiration during ripening, and are colonized by postharvest fungal pathogens. Induced resistance is a strategy to control diseases by eliciting biochemical processes in fruits and vegetables. This is accomplished by modulating the progress of ripening and senescence, which maintains the produce in a state of heightened resistance to decay-causing fungi. Utilization of induced resistance to protect produce has been improved by scientific tools that better characterize physiological changes in plants. Induced resistance slows the decline of innate immunity after harvest and increases the production of defensive responses that directly inhibit plant pathogens. This increase in defense response in fruits and vegetables contributes to higher amounts of phenols and antioxidant compounds, improving both the quality and appearance of the produce. This review summarizes mechanisms and treatments that induce resistance in harvested fruits and vegetables to suppress fungal colonization. Moreover, it highlights the importance of host maturity and stage of ripening as limiting conditions for the improved expression of induced-resistance processes.

CURRENT KNOWLEDGE

Postharvest losses have been estimated at 5–25% in developed countries and 20–50% in developing countries, depending on the commodity, cultivar, and marketing and handling practices (64). Consequently, the reduction of food loss and food waste is a major societal, economic, nutritional, and environmental challenge worldwide. The FAO reported that 14% of all food produced around the world is lost, not including at the retail and consumption levels, where 17% is wasted (40). Technically, food losses include what occurs from the grower to retail, whereas food waste begins with the retail market and ends in the consumer home. Food loss and waste can be caused by senescence, physiological breakdown, mechanical injury, enhanced ripening, simple water loss to compositional changes, and deterioration of the crop by decay organisms. The development of innovative, global cultivars with improved color, shape, or taste is of limited commercial value without proper storage conditions that provide improved resistance to postharvest decay. Moreover, the removal from the market or the limitations on the use of broad-spectrum synthetic fungicides have increased the incidence of decay-causing fungi previously considered minor pathogens on most crops (e.g., fungal pathogens in the genera *Rhizopus*, *Mucor*, *Alternaria*, *Aspergillus*, and *Penicillium*) (119).

With the trend toward healthier and widely available supplies of fresh fruits and vegetables, an ability to suppress postharvest diseases has become one of the most important limiting constraints to extending the storability of crops. In response, research investigating induced resistance in stored produce has increased greatly in the past 25 years (**Figure 1**), which has led to the practical deployment of induced-resistance technologies as significant alternatives to synthetic fungicides to control postharvest diseases. The induction of host defenses in fruits and vegetables includes the application of exogenous physical, chemical, and biological technologies that cause physiological changes in the plant to increase defenses against rot-causing fungal pathogens. Importantly, induced resistance confers protection toward a broad spectrum of postharvest rot pathogens during storage and shelf life, which gives it a central role in integrated disease management strategies. Progress in the development of scientific tools (e.g., molecular biology, genomics, etc.) has allowed better biological process monitoring than in the previous decades (25). This process monitoring has increased the holistic understanding of the postharvest quality of fresh fruits and vegetables, which is influenced by combinations of technologies applied before and during storage (e.g., growth regulators and fungicides) as well as storage temperatures, storage atmosphere, and pre- and postharvest packinghouse protocols. Consequently, holistic tools that can provide an integrated depiction of how a range of biotic and abiotic factors alter host resistance become central to the improvement of crop quality and consumer health (123). This review highlights how these tools have allowed the unraveling of effects of host maturity, ripening processes, and senescence on the mechanism of induced resistance to postharvest disease.

POSTHARVEST PHYSIOLOGY OF FRUITS AND THEIR SUSCEPTIBILITY TO POSTHARVEST PATHOGENS

Fruits and vegetables require a complex set of interacting genes and signaling pathways for their proper development. Fruit growth and maturation include three separate stages: (*a*) fruit set, (*b*) development, and (*c*) ripening and senescence. Of these, the ripening process triggers a complex set of biochemical pathways that make fruit attractive, desirable, and edible to consumers but at the same time susceptible to pre- and postharvest diseases (49, 50, 108). Postharvest diseases frequently do not show symptoms during the early stages of fruit set and development but only during the period of ripening. Fungal infections that occur during flowering and early

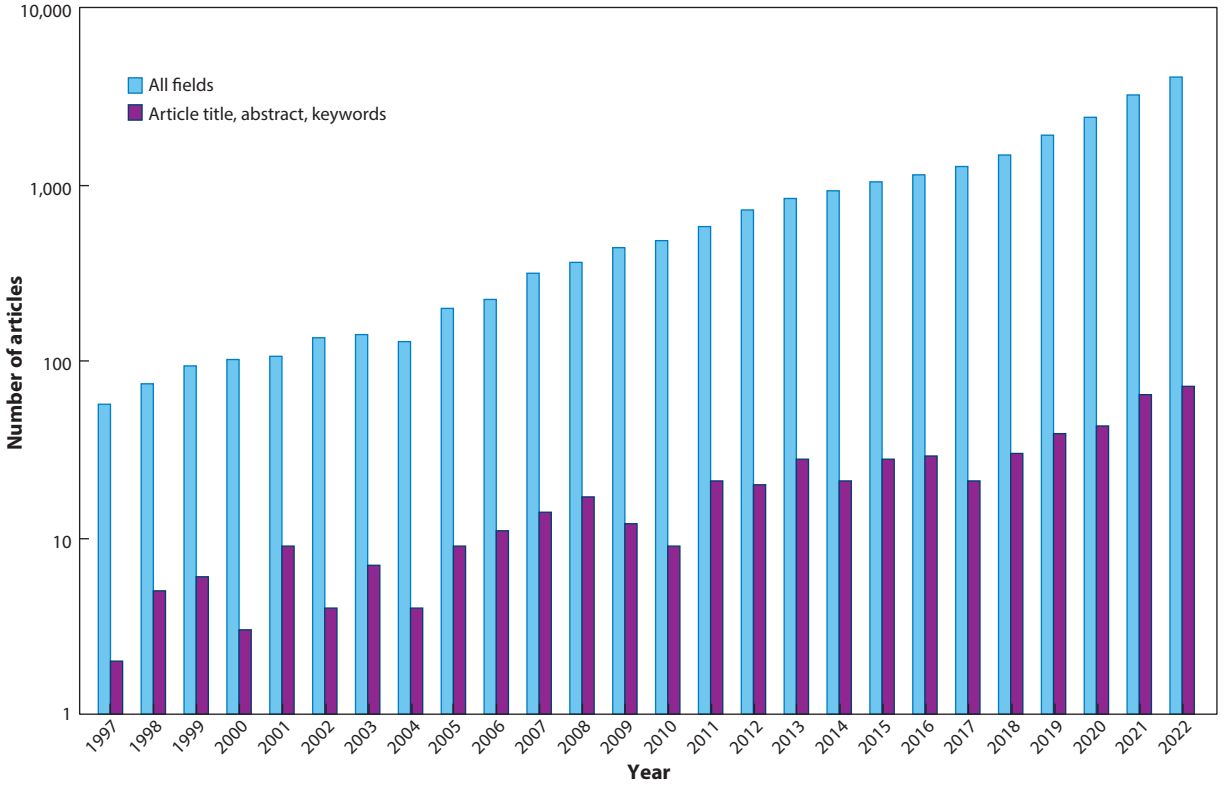

Figure 1

Number of articles available through Scopus (**https://www.scopus.com/**, accessed April 11, 2023) over the past 25 years using the search keywords of "induced resistance postharvest." Adapted from Romanazzi et al. (123).

fruit development commonly become quiescent (dormant) until ripening due to fruit's innate capacity to resist pathogen challenge (107, 108).

During fruit ripening, components of the plant immune system gradually lose either their effectiveness or the ability to activate resistance processes, which happens concurrently with a reduction of defense hormone production and signaling and downstream transcriptional responses. Ripening processes lead to cell wall breakdown, simple sugar accumulation, changes in pH and secondary metabolite composition, and increased production of and sensitivity to the phytohormone ethylene (ET). All these processes can affect the fruit's capability to respond to the process of induced resistance and prevention of fungal infection (2, 109). Understanding how physiological and biochemical change during ripening and senescence limits elicitation of induced resistance has become a primary goal of postharvest plant pathology research (123).

The physiological and biochemical processes during ripening divide types of fruit into one of two broadly defined categories, climacteric and non-climacteric, based on their respiratory profile as well as the way in which they produce and respond to ET (127). Non-climacteric fruits respire and produce ET at basal levels throughout fruit maturation and senescence. This mode of ET production is termed System 1 (S1) and includes the autoinhibition of ET production, resulting in non-climacteric behavior. Non-climacteric fruits, which include cherries, berries, citrus, and others, are harvested ripe and do not ripen after harvest or exhibit increasing levels of ET production during ripening (21). In contrast, ripening in climacteric fruit and vegetables, such as apple,

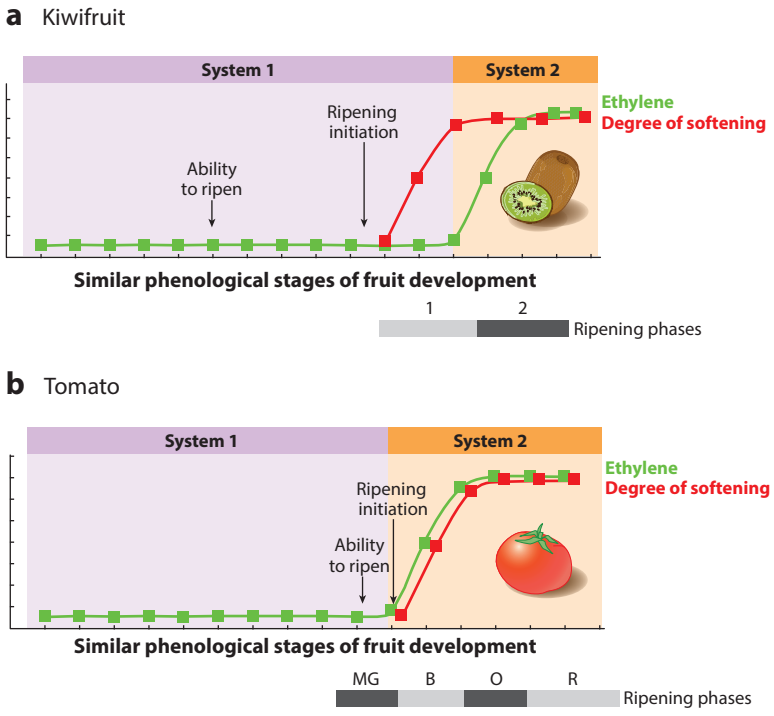

Figure 2

Different models of ripening behavior in kiwifruit and tomato, both climacteric fruits. In kiwifruit, the ability to soften occurs well before ethylene ripening initiation. Ripening initiation and the initial softening period (ripening phase 1) are accompanied by nonautocatalytic ethylene production (System 1) and are separated from the late-ripening period (ripening phase 2) that is accompanied by autocatalytic ethylene production (System 2). In tomato fruit, the ability to soften (responsiveness to exogenous ethylene) coincides with the mature green (MG) stage and is closely followed by ethylene ripening initiation and autocatalytic ethylene production. Different stages of fruit development: mature green (MG), breaker (B), orange (O), and red (R). Adapted from Nieuwenhuizen et al. (93).

pear, banana, papaya, avocado, mango, tomato, and others, is characterized by a burst of respiration and a substantial increase in ET biosynthesis as a fruit transitions from System 1 (S1) to System 2 (S2) (autoinduction of ET production) (**Figure 2**) (18, 21, 76, 97).

Transcriptional and phytohormonal regulation of ET-dependent ripening of fruits has been extensively reviewed (21, 23). After the respiratory burst in climacteric fruit, ripening progresses rapidly and irreversibly to senescence, during which host susceptibility to fungal attack is enhanced (59, 77). Fruit ripening is genetically modulated with senescence partially overlapping the ripening process. Attributes of senescence include cell decompartmentalization, membrane weakening, and demolition of cell functions. Current research is directed at understanding the difference between the physiological and biochemical processes that modulate the induced resistance during the preclimacteric, climacteric, and postclimacteric stages. Specifically, why is resistance induction after initiation of ripening in fruit showing the climacteric phase less efficient in terms of the magnitude of the defense response than in freshly harvested fruits (**Figure 3**)?

In the regulatory network involved in non-climacteric ripening, abscisic acid (ABA) and polyamines, rather than ET, play essential roles (58, 77). Comparative studies with strawberry and tomato indicate that the split between non-climacteric and climacteric ripening responses lies

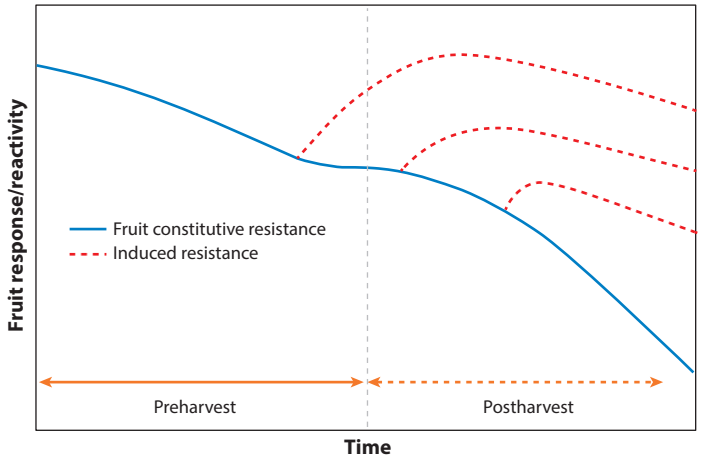

Figure 3

Change of constitutive host resistance in fruit over time and modulation of this response/reactivity following preharvest and postharvest induced resistance.

in the way that *S*-adenosyl-L-methionine (SAM) is preferentially utilized as a precursor to ET or as a substrate for polyamine biosynthesis (72). One question is whether ripening in climacteric fruit triggers increased susceptibility to fungal infection and disease (80). This is unlikely because susceptibility to fungal pathogens is also observed in non-climacteric ripening fruit (68, 107).

Consequently, parsing physiological regulation of fruit ripening and a shifting susceptibility to disease is complex. Results indicate that only selected ripening events and pathways are required to facilitate pathogen attack and that pathogens may modify their infection strategy as fruit ripens (10, 11, 15, 16). Fruits and vegetables have a natural defense against postharvest pathogens that is expressed during early fruit development and changes according to the stage of ripening, i.e., higher before harvest and then declining (**Figure 3**). For example, for fungal pathogens that infect prior to the ripening phase, it is common for the invader to enter what is termed a quiescent phase, which becomes activated when natural resistance declines. This decline typically occurs after harvest, when the fruits and vegetables are transferred to the shelf or often stored at cool temperatures for long periods. At this temperature, fungal pathogens (e.g., *Botrytis cinerea*) continue to progress slowly, and the fruit is less responsive during senescence (124). To achieve induced resistance, the magnitude of the response depends on the reactivity (or response) of the fruit, which is highest before and soon after harvest and in environmental conditions that allow the host to respond before being shifted to conditions (e.g., long cold storage or shelf life) where the pathogen has an advantage. **Figure 3** highlights the hypothetical physiological factors that determine the magnitude and diversity of defense responses to biotic or abiotic stress at the different stages of fruit ripening.

PHYSIOLOGICAL MECHANISMS OF HOST RESISTANCE TO PATHOGENS DURING FRUIT RIPENING

To compare mechanisms of induced resistance in ripening fruit, we have to understand the possible mechanism(s) that lead to this enhanced state. In tomato, for both decay-resistant and decay-susceptible cultivars, profiles of host defense gene expression to fungal infection in unripe and ripe fruit have been identified (128). Genes expressed commonly in all profiles include receptor-like kinases, the leucine-rich repeat classes, WRKY transcription factors (22) (repressors and activators

of plant processes), and the family of ET transcription factors, including ET response factors (ERFs) (148). ERFs play vital regulatory roles in developmental processes and stress responses in plants and integrate the salicylic acid (SA) and the ET/jasmonic acid (JA) pathways. Wasternack & Song (141) also reported the involvement of JA biosynthesis responses that regulate plant growth, development, secondary metabolism, defense against pathogen infection, and tolerance to abiotic stresses as well as chitin catabolism (150). These profiles of gene expression are closely related to those known to be associated with innate immunity (127). For example, genes that appear in both the resistant and susceptible profile responses of tomato fruit were identified previously as components of the innate immune response, including the JA biosynthesis gene *LoxD* (149), the subtilisin-like protease SBT3 (92), the peroxidase (POD) CEVI-1 (91), and the chitinase (CHT) CHI9 (31). Given that the responses occur in both resistant and susceptible interactions, it is likely that host genotypes show differential levels of expression during fungal attack. In contrast, the gene expression profiles revealed several well-known defense genes, such as *WRKY33* and the ERF *PTI5*, that were expressed only in susceptible fruit (51, 55, 144). Although the gene profile in the resistant cultivar did contain some defense genes not present in the susceptible profiles (128), the findings indicate that many of the genes in the resistant-stage fruit profile response were either functionally similar to those in the susceptible profile response or expressed at lower levels than in the susceptible interaction. Therefore, the inability to induce resistance in susceptible fruit or at susceptible stages of ripening is likely not because the cultivar lacks the genes for pathogen defense.

If the expression of defense genes at resistant and susceptible stages of ripening does not determine the outcome of the interaction in tomato fruit, the question is what other factors associated with ripening fruit may instead govern susceptibility (15). Ripening processes in tomato have been studied using the mutants *ripeninginhibitor* (*rin*) and *nonripening* (*nor*) and nonripening mutants with altered cell wall architecture, e.g., *Colorless nonripening* (*Cnr*). The *rin* mutation results in full-sized firm tomato fruit that remains green, does not produce or ripen in response to ET, and shows the same susceptibility to *B. cinerea* as wild-type fruit (139). The *nor* mutation, which also does not undergo most of the changes associated with ripening, produces fruits that are entirely resistant to *B. cinerea* infection at both the unripe and ripe stages. Unlike *rin* and *nor* fruit, *Cnr* fruit have altered cell wall architecture and are susceptible to *B. cinerea* when both in unripe and ripe stages (39, 95). Consequently, the *Cnr* phenotype is evidence that factors that are not ripening-related contribute to the resistance of the unripe fruits.

Ripening mutants of tomato were utilized by Blanco-Ulate et al. (9) and Silva et al. (127) to further examine how ripening contributes to susceptibility. Their data indicated that with ripening, preformed defenses declined and correspondingly, susceptibility factors increased. Most interestingly, the decline of preformed defenses appeared to be regulated by genes involved in the mediation of reactive oxygen species (ROS) levels. Host activation of ROS levels at early stages of fungal penetration is critical for the activation of defense signaling against the pathogen, whereas detoxification of ROS at later periods of the interaction is important for pathogenicity by *B. cinerea* (75, 142). The initial importance of ROS for improved resistance was observed when *B. cinerea* was not able to colonize an ABA-deficient tomato mutant. In this case, the controlled ROS production in the ABA-deficient tomato mutant promotes cell wall fortification, which prevents fungal colonization by *B. cinerea* (5, 30). In addition, engineered tomato lines with high amounts of the antioxidant anthocyanin in the fruit are also resistant to gray mold (153). This may indicate that during ripening, loss of control of ROS levels may represent the reduction of an important preformed defense.

Some features of ripening have the potential to be affected by a susceptibility factor such as the ET burst that accompanies ripening in climacteric fruit. Although ET is known for its

involvement in defense activation toward necrotrophic fungi (135), its induction of the ripening program catalyzes downstream events of climacteric fruits that also can be favorable to pathogen invasion. Previous research suggests that blocking of ET receptors in the immature-resistant fruit stage can increase or decrease resistance to gray mold, depending on the concentration of the inhibitor used (11). Thus, ET-mediated resistance may be dependent on careful regulation of ET levels.

In addition to ET, JA is known to mediate resistance to necrotrophic pathogens in plants (98, 140). The enrichment of JA biosynthesis genes is seen in the ripe-susceptible stage response as well as the response to *B. cinerea* in all *nor*, *rin*, and *Cnr* mutant fruit at both stages of susceptibility. Basal levels of JA in healthy fruit are highest in *nor* fruits at the susceptible stage, where they are nearly twice as high as levels in wild-type susceptible fruits. Moreover, *nor* fruits that are resistant to infection by gray mold at the resistant unripe and susceptible ripe stage (128) are the only fruits in which JA signaling/response genes are enriched in response to *B. cinerea* infection at both stages. The interplay between ET and JA and their impact on ripening-associated susceptibility require further study.

Other features of ripening can increase susceptibility to fungal diseases such as the weakening of plant cell walls leading to fruit softening. Cell wall polysaccharide remodeling, breakdown, and solubilization in ripening fruit occur as the result of various cell wall–degrading enzymes, particularly those that act on pectin (12). The cell wall integrity and fortification improve tomato fruit resistance to infection by *B. cinerea* and other pathogens (9, 16, 31, 86). Silva et al. (128) described remarkable similarities in the cell wall polysaccharide changes caused by both infections of unripe fruit and ripening of healthy fruit, particularly in the increased accessibility of pectic polysaccharides. This again raised the question of why the response of the ripening fruit cannot induce resistance if infected unripe fruit can do it. Their conclusion was that in those cases the modulation of virulence was closely related to enhanced ripening of the host, the ability to infect the host, and the capability of pectin degradation. This may indicate that induced resistance depends on the interaction of the fungus with the host's ability to initially inhibit the degradation of host pectin. This type of interaction was supported by the development of a clustered regularly interspaced short palindromic repeat (CRISPR) pectate lyase mutant, where the reduced rate of softening was accompanied by reduced susceptibility to *B. cinerea* in ripe susceptible fruit (128). Furthermore, if *B. cinerea* double knockout Δbc polygalacturonase1-Δbc polygalacturonase2 was inoculated into unripe fruit, the fungus was incapable of emerging from quiescence even after the fruit was fully ripe (129). This differential fruit response to inoculated tissue may indicate that the changes in the host following fungal attack are the result of the host's capability to modulate factors affecting the softening process. In conditions of delayed ripening of the fruits, a reduced colonization pattern was observed (129).

When biotrophic *Colletotrichum gloeosporioides* infects unripe, resistant tomato fruit (3), the fungus breaches the cuticle and remains quiescent until ripening begins. During quiescence, the pathogen activated chromatin remodeling genes and began to alkalize the surrounding environment. Host defense reactions were further intensified by activation of pathways involved with lignification and production of glycoalkaloid components of the phenylalanine pathway and the activation of preformed defense compounds. Subsequently, initiation of the ripening process (similar to the tomato fruit; see **Figure 2**) also initiated expression of fungal cell wall–depolymerization enzymes that contribute to fungal hyphae growth.

Conversely, an ET treatment applied to avocado fruits increased both the rate of fruit ripening and levels of defense compounds but did not result in immediate lesion development by *C. gloeosporioides* (45, 112). *Alternaria alternata* inoculated onto unripe, unharvested mango fruits

was able to colonize the flesh of peeled fruit (still at a preclimacteric stage) (35), whereas the fungus did not colonize the peel of the same fruit that contained a high concentration of defense compounds. These results again demonstrate that fruit ripening and susceptibility are not completely correlated physiological processes. Consequently, the ability of induced resistance during the ripening of a susceptible host may be dependent on retaining or increasing preformed antifungal defenses and on slowing the rate of cell wall depolymerization observed in ripening fruits.

COMPARISON OF PHYSIOLOGICAL CONDITIONS OF FRUIT CONTRIBUTING TO RESISTANCE INDUCTION IN CLIMACTERIC AND NON-CLIMACTERIC FRUITS

A primary thesis of this review is that although decay development usually occurs during ripening, how ripening processes specifically influence fruit susceptibility is not always clear (107, 108). For example, ET is required for ripening but its role in regulating the fruit's ability to resist pathogen colonization and/or the induction of resistance or susceptibility has yielded contrasting observations. Pristijono et al. (106) reported that pathogens isolated from climacteric fruits showed inhibited growth in the presence of exogenous ET (87). However, most of the fungal pathogens tested in their study had a very broad host range, including both climacteric and non-climacteric fruit, indicating that perhaps it is the host that determines the colonization pattern of the pathogen. In contrast, technologies to suppress respiration and ET production are used widely to delay fruit senescence and, indirectly, reduce fruit susceptibility to pathogens (80). In avocado, rapid disease development takes place in harvested fruit after the ET burst, but several other factors affect susceptibility, including loss of preformed defenses and softening of the fruit (109). A high CO_2 storage atmosphere slows respiration, delays the initiation of ET production and fruit ripening, enhances retention of preformed defense compounds, and retards avocado senescence, enhancing its resistance (111). This indicates that fruit response to the external environment is a factor that modulates the elicitation of immune activity against the pathogen. Under high CO_2 conditions, the initial colonization of *C. gloeosporioides* induces a significant increase in ROS (8) and antifungal defenses (111), but these responses become attenuated when the treatment occurs on ripe, post-climacteric fruit. Similarly, when avocado was treated with 1-methylcyclopropene (1-MCP), an ET action inhibitor that delays fruit ripening, fruits showed susceptibility to *C. gloeosporioides* before full ripening. This contrast, i.e., high CO_2 delays ET and susceptibility compared to 1-MCP's inhibition of ET and enhanced susceptibility in unripe fruit, indicates fruit susceptibility is modulated by different mechanisms that are correlated with ET production but not regulated by it.

In non-climacteric fruits such as strawberries, ET is generally considered to have little or no effect on ripening (101). Nonetheless, a short-term hypobaric treatment (0.5 atm for 4 h) reduced gray mold of strawberry; this result was interpreted as the hypobaric treatment having an effect on the host through a decrease in ET concentration in fruit tissues, which delayed ripening and made the fruit more resistant to disease (80, 121). This interpretation was confirmed by exposing the strawberry fruit to exogenous ET, which decreased fruit firmness and enhanced gray mold development (38). In other cases, as with climacteric avocado fruit, disease development was enhanced by treatment with 1-MCP. Also, in non-climacteric citrus and grapes, a 1-MCP treatment increased susceptibility to blue and gray mold, respectively (105). This effect in avocado was the result of the need for low concentrations of ET to induce the preformed compounds, and in grapes, it was the result of the inhibited accumulation of anthocyanins associated with ripening of berries (24, 42, 53). These examples indicate the complex behavior of ET in the modulation of inducible factors modulating fruit resistance.

MECHANISMS OF RESISTANCE INDUCTION IN FRUIT: TREATMENT TARGETS AND HOST RESPONSES

Various biotic inducers (e.g., fungi, bacteria, viruses, phytoplasma, pests) and abiotic stresses (e.g., chemical and physical treatments) can trigger induced resistance in plants (1, 104, 137), causing rapid expression of defense responses (27, 46, 71) and limiting fruit colonization after harvest. Two types of mechanisms of induced resistance in plants are systemic acquired resistance (SAR) and induced systemic resistance (ISR). Both these mechanisms can induce defenses that confer long-lasting protection against a broad spectrum of microorganisms and are mediated by phytohormones, such as SA in SAR and JA and ET in ISR (133). ISR occurs following an application of a biocontrol agent or other microorganism (e.g., mycorrhizal fungi) to the roots (133), and its effects on postharvest decay have not been extensively investigated. SAR is associated with accumulation of pathogenesis-related (PR) proteins, which contribute to resistance (36). Induced resistance does not always directly activate plant defense responses but can place the plant in a state of alertness such that a future pathogen attack will be strongly and efficiently responded to. This phenomenon is known as the priming effect (26, 63). This response is the result of the capacity to sensitize the plant immune system for a better expression of induced defense mechanisms (26). Priming can be first established by stimuli that have environmental, biological, or chemical origins. After perception, fruit maintain a priming phase in which molecular and biochemical changes occur but where there is not a direct activation of defense mechanisms (89).

On table grape berries, chitosan treatment showed a priming effect, because by itself the compound did not increase the amount of *trans*-resveratrol in the berries, but when applied prior to exposure of UVc irradiation, it increased the content of this compound as compared to the UVc alone (117). Priming, theoretically, may divert part of the resources of the plant from primary (growth and production) to secondary metabolism (defense). This may generate a potential fitness cost; however, in practice, it is not very costly to fruit undergoing maturation (14, 90, 120, 133). The priming response phase has been shown to be short- to long-lasting, and in some examples, it has been transmitted to subsequent generations, meaning that plants exposed to stress stimuli produce progeny that display sensitized defense mechanisms (120, 133). Upon subsequent attack, priming allows a faster and stronger activation of defense mechanisms, which results in broad-spectrum disease protection.

Activation of SAR also involves cellular redox modification in the host tissues. Both primary and secondary oxidative bursts are required for the onset of SAR (4). SAR is predominantly dependent on the activity of NPR1 (nonexpressor of PR gene 1) (126). NPR1 confers resistance through a transcriptional cascade, which includes transcription activators and repressors, leading to the massive induction of antimicrobial genes. Upon pathogen challenge, the NPR1 is phosphorylated and degraded and its turnover appears to be required for its full transcriptional activity and the activation of SA-mediated defense, which results in the expression of a battery of *PR* genes (130). The transcription factor NPR1 and the activated SA-mediated defense response result in SAR and the activation of approximately 10% of the plant transcriptome (46). Several studies suggested the requirement of lipid signals such as JA-derived molecules for SAR (46, 81) and a putative lipid transfer protein in challenged tissue to initiate translocation of the SAR mobile signal. Because SA is light sensitive, several stable and more efficient structural and functional analogs of SA have been synthesized to induce resistance [e.g., benzothiadiazole (BTH)] (44, 69, 78, 120). The involvement of different mechanisms of resistance was reported in some cases. For example, β-aminobutyric acid (BABA)-induced resistance involves both SA-dependent and ABA-dependent defense mechanisms (13, 103). The relative importance of these phytohormone-dependent defenses varies according to the nature of the challenge pathogen.

Indeed, BABA-induced resistance against *B. cinerea* resembles SAR and requires SA accumulation (154), whereas the ABA-dependent pathway, which is associated with callose deposition, is necessary against other pathogens (132, 154). The expression of defense-related genes correlates with the reduction of disease incidence and/or severity, demonstrating the contribution of induced resistance to postharvest disease management (123). However, it is clear that the mechanism(s) of response can be affected by maturity and degree of ripening of fruit.

INDUCED RESISTANCE BY POSTHARVEST TREATMENTS

Various treatments that have proved to induce resistance in plants following infection by a pathogen have been applied to harvested fruits and vegetables (**Figure 4**; **Tables 1** and **2**) (123). The application of these physical, natural, and synthetic chemicals induces physiological changes strongly linked to defense mechanisms in the host tissues. These responses influence fruit resistance or susceptibility and are dependent on the level of response in the interaction with the pathogen. They can be characterized into key groups: (*a*) accumulation of PR proteins and hormone-dependent signaling; (*b*) decrease in membrane lipid metabolism and improvement in ROS scavenging ability by activation of the antioxidant machinery, including enzymes such as catalase (CAT), POD, ascorbate peroxidase (APX), and superoxide dismutase (SOD); and (*c*) synthesis of antimicrobial enzymatic activity of fruit–phenolic compounds, lignin, and enzymes such as CHT, glucanases (GLU), and phenylalanine ammonia-lyase (PAL). Host defense responses that limit pathogen colonization also affect several critical physiological processes; for example, they retard ripening and senescence, which affects fruit taste and rate of softening (80, 123).

Chitosan, which is a biopolymer produced in crab shells, is one of the main resistance inducers, and it has a threefold property that includes antimicrobial, eliciting, and film-forming activities

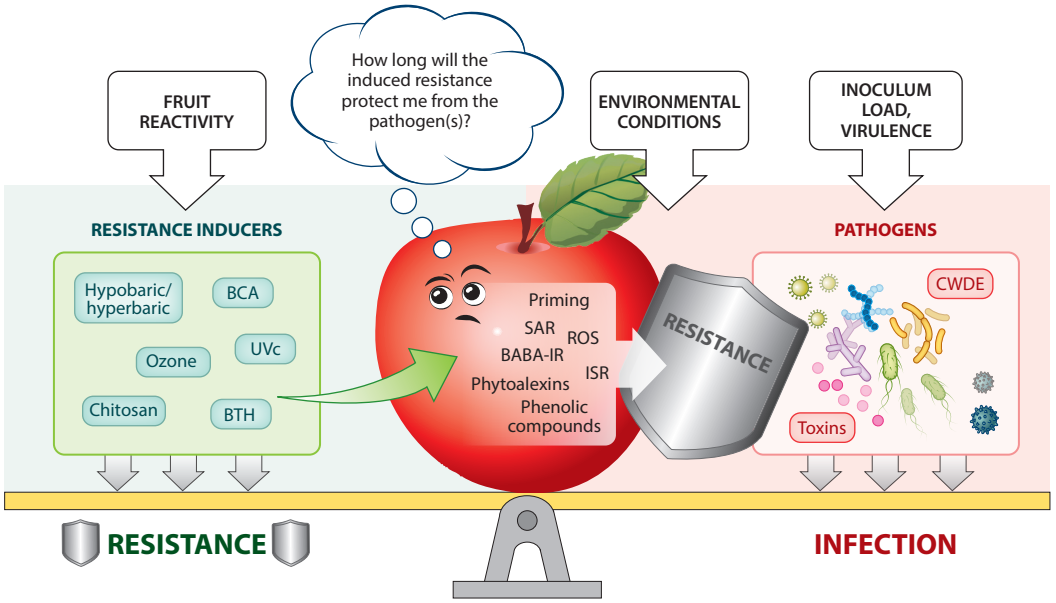

Figure 4

The dynamic balance of induced host resistance and progress of fungal decay in harvested fruits and vegetables. Abbreviations: BABA-IR, β-aminobutyric acid–induced resistance; BCA, biocontrol agents; BTH, benzothiadiazole; CWDE, cell wall–degrading enzyme; ISR, induced systemic resistance; ROS, reactive oxygen species; SAR, systemic acquired resistance; UVc, ultraviolet C.

Table 1 Examples of differential gene expression or enzyme activities in response to physical treatment applied to fruit to delay ripening and/or suppress postharvest diseases

Treatment	Genes and/or enzymes						Reference(s)
	PAL	CHT	CAT	POD	SOD	PPO	
UVc irradiation	NA	NA	+	NA	+	NA	152
Ozone	+	+	NA	NA	NA	NA	see 123[a]
Electrolyzed water	+	+++	NA	+	NA	NA	see 123[a]
Heat treatment	+/−	−	NA	NA	NA	NA	see 123[a]
Hypobaric treatment	+	+	NA	+	+	+	145; see 123[a]

[a]References therein and tables 2 and 4.

Abbreviations: +, overexpressed up to threefold; ++, overexpressed from fourfold to tenfold; +++, overexpressed more than tenfold; −, downregulated up to threefold; CAT, catalase; CHT, chitinase; NA, not applicable; PAL, phenylalanine ammonia-lyase; POD, peroxidase; PPO, polyphenol oxidase; SOD, superoxide dismutase.

(116, 122). The eliciting activity is produced in host–pathogen interactions when the enzymes of the pathogens attack the host cell wall and are then considered endogenous elicitors that cause plants to perceive them as a signal of pathogen infection and therefore raise defenses (115). The biopolymer can directly induce plant defense enzymes and synthesis of secondary metabolites, such as polyphenolic compounds, lignin, flavonoids, and phytoalexins, in several plant species (28, 85, 119) as well as increase the antioxidant capacity (17, 71, 102, 114, 116). In addition to being a priming agent, chitosan produces a coating on the treated surface that preserves fruit freshness by reducing gas exchange and slowing respiration and the ripening process, slowing the fruit's

Table 2 Examples of differential gene expression or enzyme activities in response to application of natural and synthetic chemicals that are involved with resistance induction in fruits

Treatment	Genes and/or enzymes								Reference(s)
	SOD	CAT	POD	APX	CHT	PAL	GLU	PPO	
Methyl salicylic acid	+	++	++	+	NA	NA	NA	NA	see 123[a]
Benzothiadiazole	NA	NA	+/++	++	+	NA	+/+++	+	69; see 123[a]
Chitosan	+	−/+	+	+	−/+++	−/+++	−/+++	+	69, 71, 94, 96, 125; see 123[a]
Ruta graveolens essential oil	NA	NA	NA	NA	+/++	+/++	+/++	NA	71
Calcium and organic acids	NA	NA	NA	+	+/+++	++	++	NA	see 123[a]
Sodium carbonate	NA	NA	++	NA	−	++	++	NA	see 123[a]
Chitosan + *R. graveolens* essential oil	NA	NA	NA	NA	+/+++	+/+++	+/+++	NA	71
Chitosan + potassium sorbate	NA	NA	+/+++	NA	NA	NA	NA	+/+++	29

[a]References therein and table 3.

Abbreviations: +, overexpressed up to threefold; ++, overexpressed from fourfold to tenfold; +++, overexpressed more than tenfold; −, downregulated up to threefold; APX, ascorbate peroxidase; CAT, catalase; CHT, chitinase; GLU, β-1,3-glucanase; NA, not applicable; PAL, phenylalanine ammonia-lyase; POD, peroxidase; PPO, polyphenol oxidase; SOD, superoxide dismutase.

physiological metabolism (118, 119). To these two actions (induction of defenses and increases of antioxidant activities) chitosan showed antimicrobial activity toward a list of decay-causing fungi, reducing decay development on a list of crops and extending their shelf life (113).

Physical treatments also showed significant induction of fruit resistance. Heat-stress-exposed peach, strawberry, and mango fruit showed the induction of transcription factors that enhance fruit resistance (60, 84) and delay fruit ripening. Heat-treated strawberries directly triggered plant defenses with the accumulation of PAL, CHT, CAT, APX, and SOD, which reduced the size of gray mold lesions (60). Strawberry fruit exposed to a hypobaric atmosphere were shown to express induced resistance to *B. cinerea* and *Rhizopus stolonifer*, which was linked to increased CHT, PAL, and POD activity and improved fruit storability (54). The application of short-term hypobaric treatments can inhibit the senescence of table grapes, strawberries, and sweet cherries (121). In inoculated table grapes, this treatment reduced gray mold lesions compared to an inoculated control maintained at room pressure (**Figure 5**). In addition to direct physiological effects (e.g., less ET in hypobaric-treated tissues), physical treatments have been linked to the activation of SAR (26, 90). Therefore, findings indicate that the defense responses induced by biotic and/or abiotic agents that protect against pathogen development can be accompanied by critical physiological effects on the host that improve fruit storability (34, 121, 123).

What is more difficult to explain is the fact that resistance induction to prevent fungal colonization can be long-lasting (82, 83) and perhaps even transmitted to subsequent generations (138). Preharvest treatments with SA and BABA have been shown to reduce disease incidence of *Penicillium digitatum* and *B. cinerea* in orange and tomato fruit, respectively, affecting colony initiation by 3–5 days followed by a 50% inhibition in colony growth (56, 143). In the case of BABA, the long-lasting induced-resistance response correlated with the delay of fruit maturation (red fruit per plant) and with differential accumulation of metabolites putatively identified as lipids, alkaloids, terpenoids, and the plant hormone ABA (143).

The long-lasting nature of induced resistance in fruit also has been linked to epigenetic mechanisms that could fine-tune the expression of defense responses over longer periods or from one

Figure 5

Table grape bunches exposed to hypobaric treatment at 0.05 atm for 24 h (*left*) or kept at room temperature (*right*) after inoculation with a conidial suspension (10^4 spores/mL) of *Botrytis cinerea* and incubated for 5 days at 20°C.

generation to the next (82, 83). Fruit development and ripening are influenced by chromatin modifications and changes in DNA methylation (41, 47, 67, 147). Considering that fruit are mostly maternal tissues and that some preharvest treatments trigger long-lasting induced resistance, several research groups have attempted to link this induction with epigenetic change. Certainly, it is plausible that intergenerational epigenetic mechanisms could play certain roles in the priming processes of fruit produced from true seed (57). A recent investigation indicated that potato seeds obtained from primed potato plants have shown higher levels of induced wound healing and resistance to dry rot in the subsequent crop (48).

In summary, different classes of chemicals ranging from naturally occurring metabolites, inorganic compounds, and synthetic chemicals, together with several physical treatments (123), serve as examples of abiotic agents that act as resistance inducers. Chemical inducers can trigger defense responses locally in fruits and can also induce the production of mobile immune signals, including SA, methyl salicylate (MeSA), azelaic acid, glycerol 3-phosphate, and abietane-diterpenoid-dehydroabietinal (20, 62, 99).

INDUCED RESISTANCE IN NON-CLIMACTERIC FRUITS

A preharvest spray treatment of SA onto non-climacteric, unripe pepper fruit inhibited anthracnose in harvested fruit by inhibition of appressoria formation in *C. gloeosporioides* (73). Genetic analysis indicated that the responsive genes in pepper were regulated solely by SA and not by JA or ET. Interestingly, most of those genes were preferentially expressed in the ripe fruit, suggesting that SA-mediated transcriptional regulation modulates anthracnose development independently of ET (74). A similar response was observed in oranges, where blue mold, caused by *P. digitatum*, was inhibited by exogenous SA treatments. SA treatment of *P. digitatum*–colonized fruits enhanced expression of *CsWRKY70* and genes related to MeSA biosynthesis, including the salicylate carboxymethyltransferase (33). These results indicate that non-climacteric fruits may respond positively to SAR-inducing treatments. The mechanism of induced resistance in several commodities, such as bell pepper and strawberry fruit (37, 43, 70, 114, 116), is accompanied by the delay of ripening and senescence. Physiological responses, including delay of ripening and senescence, seem to be critical for the postponement of fungal colonization when resistance is induced by the different biotic and abiotic treatments.

INDUCED RESISTANCE IN CLIMACTERIC FRUITS

Induced resistance in freshly harvested avocado fruits was reported following the application of chitosan in combination with the amino acid phenylalanine (125). The treatment delayed decay development by *C. gloeosporioides* and enhanced the expression of heat-shock proteins, fatty acid desaturases that contribute to the synthesis of antifungal defenses, and genes involved in the biosynthesis of phenylpropanoids and PR proteins. These specific responses contributed to suppression of rot development and were accompanied by a delay in fruit ripening (110, 125, 146). This delay in the initiation of fungal attack accompanied by the inhibition of fruit ripening represents the normal way of delaying decay in climacteric fruits. In banana fruit, resistance to postharvest diseases induced by SA treatment was also accompanied by retardation of ripening (129). This retardation also suppresses ET production and may further extend the shelf life of fruit, thereby delaying the development of disease symptoms that normally develop as climacteric fruit ripen (151). In the case of banana, the degree of induction may also depend on the timing of pre- and/or postharvest elicitor treatment (61) (**Figure 3**), where levels of induction of the antifungal phytoalexin hydroxyanigorufone are higher in unripe fruit than in ripe fruit exposed to ET (65, 66).

Induced resistance of peach to brown rot can be affected by the level of ET, which is determined by the degree of fruit maturity (6). At early stages of fruit maturity, the presence of *Monilinia laxa* induces only low levels of ET insufficient to activate carbohydrate-active enzymes for cell maceration (32, 52, 88). Under these conditions, the fruit was able to suppress disease development via an oxidative burst and SAR induction that challenged the pathogen's survival (88, 134). SAR-activated enzymes act as antimicrobials in peaches by targeting β-1,3/1,6-glucans and chitin present in fungal cell walls or membranes (19, 79, 100, 131, 136). The induction of the hydroxyproline-rich glycoprotein-encoding transcripts in young peach fruit may also explain disease resistance in unripe fruits, given the lower concentration of induced hydroxycinnamates in ripe fruit that may not be sufficient to confine the disease, explaining the enhanced susceptibility to brown rot as the fruit mature (7).

Overall, the induction of defense responses in fruit aims to delay the onset and spread of fungal infections. However, the coordinated balance of responses among the multiple substances that account for enhanced resistance responses by the host may change according to the pathosystem and the physiological stage of fruit development (immature and mature fruit). This coordinated balance of response(s) may determine the fruit's resistance or susceptibility depending on the level of this response in the interaction with the pathogen (**Figure 4**). The differential responses in immature and ripening fruits are in dynamic equilibrium that can evolve toward resistance or susceptibility to postharvest decay; this differential requires further study (**Figure 4**).

If climacteric and non-climacteric fruits are responding similarly to a variety of exogenic treatments that induce resistance, and ET is not always involved in the induction process, a question is why crop losses during senescence cannot be prevented by induced resistance. Most of the published reports showing suppression of decay development by different biotic, abiotic, and physical treatments in fruit (73, 84) have also reported a corresponding delay in ripening and senescence. The possible factors affecting the responses to the induction process are likely related to the importance of (*a*) physiological maturity of the fruits, i.e., overmature fruit does not show the same response as immature fruit; (*b*) unripe fruit stored for long periods does not respond to the same degree as freshly harvested fruit; and (*c*) post-climacteric fruit does not respond to the same degree as preclimacteric fruit. Future research to address these hypotheses will have both commercial and theoretical significance to disease development in fruit.

CONCLUSIONS

Induced resistance in fruit and vegetable tissues is a tool to confer enhanced protection against postharvest decay during storage and shelf life. Application of diverse abiotic and biotic stimuli triggers physiological host responses, inducing accumulation of defense compounds that limit fungal growth, delaying fruit senescence, preserving the physiological youth of fruit for longer periods, and enhancing the plant's ability to defend itself from invading pathogens.

Therefore, induced resistance can (*a*) offer a defense strategy against many plant pathogens that are difficult to control by single resistance genes; (*b*) result in specific mechanism(s) of activation of defense responses; (*c*) modulate mechanisms that are widely present in many fruit crops; (*d*) activate mechanisms in the fruits that are considered safe and may even increase the quality of the fruits through the increase of beneficial compounds (phenols with antioxidant activity); and (*e*) be active throughout plant–fruit development, opening possibilities for both pre- and postharvest disease control (123).

We discussed optimal timings of induction of plant response to the different inducers. Harvested fruits and vegetables show a decline in responsiveness to resistance induction with the progression of ripening and senescence. Current research is focused on the discovery of new resistance inducers and understanding their mechanisms of action to apply them at the proper time.

Future research should also examine atmospheric modifications that can induce resistance by influencing host physiology and ripening. The study of biochemical mechanisms involved in the host response will benefit from technological innovations to monitor gene expression. Future efforts should also try to understand the contribution of the microbial populations (as a microbiome) to the host and pathogen interactions and verify their possible effects in induced resistance and, if positive, optimize the timing of preharvest and postharvest applications. Increasing the implementation of induced-resistance technologies will reduce the application of synthetic pesticides, moving toward a desirable sustainable approach in plant production and protection.

SUMMARY POINTS

1. Induced resistance reduces disease incidence by postharvest fungal pathogens by modulating the progression of ripening and senescence and by limiting the pathogen's ability to invade plant tissue.
2. Induced resistance was observed in both climacteric and non-climacteric fruits.
3. The process of induced resistance in disease-susceptible fruit is most strongly observed in conditions that delay ripening.
4. The interaction of constitutive resistance with induced resistance creates a fulcrum against pathogenesis that is affected by the physiological stage of the fruit and its response.

FUTURE ISSUES

1. Research should search for new stimuli of diverse origins to trigger physiological host responses that keep the produce physiologically younger for longer with a higher accumulation of nutraceutical compounds.
2. Efforts should be driven to discover new resistance inducers as compounds that improve the quality of fruits and vegetables (e.g., biostimulants) and the understanding of their possible effects on postharvest diseases.
3. Efforts should be invested to better understand the effect of the resistance-inducing potential of microbial populations on the host–pathogen interactions as possible factors that modulate fruit resistance.
4. We should characterize new induced secondary metabolites in treated fruits and vegetables, as they may affect taste and nutritional quality.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

Thanks are expressed to Barbara Blanco-Ulate, Pietro Tonutti, Marwa Moumni, Susan Lurie, and Amnon Lers for critical revision of the manuscript and to Marwa Moumni for support in drawing **Figures 3** and **4**. This research was funded by PRIMA StopMedWaste "Innovative Sustainable

technologies TO extend the shelf-life of Perishable MEDiterranean fresh fruit, vegetables and aromatic plants and to reduce WASTE," which is funded by PRIMA, a program supported by the European Union.

LITERATURE CITED

1. Adikaram NKB, Joyce DC, Terry LA. 2002. Biocontrol activity and induced resistance as a possible mode of action for *Aureobasidium pullulans* against grey mould of strawberry fruit. *Australas. Plant Pathol.* 31:223–29
2. Alkan N, Fortes AM. 2015. Insights into molecular and metabolic events associated with fruit response to post-harvest fungal pathogens. *Front. Plant Sci.* 6:889
3. Alkan N, Friedlander G, Ment D, Prusky D, Fluhr R. 2015. Simultaneous transcriptome analysis of *Colletotrichum gloeosporioides* and tomato fruit pathosystem reveals novel fungal pathogenicity and fruit defense strategies. *New Phytol.* 205:801–15
4. Alvarez ME, Pennell RI, Meijer PJ, Ishikawa A, Dixon RA, Lamb C. 1998. Reactive oxygen intermediates mediate a systemic signal network in the establishment of plant immunity. *Cell* 92:773–84
5. Asselbergh B, Curvers C, França SC, Audenaert K, Vuylsteke M, et al. 2007. Resistance to *Botrytis cinerea* in sitiens, an abscisic acid-deficient tomato mutant, involves timely production of hydrogen peroxide and cell wall modifications in the epidermis. *Plant Physiol.* 144:1863–77
6. Balsells-Llauradó M, Silva CJ, Usall J, Vall-Laura L, Serrano-Prieto S, et al. 2020. Depicting the battle between nectarine and *Monilinia laxa*: the fruit developmental stage dictates the effectiveness of the host defenses and the pathogen's infection strategies. *Hortic. Res.* 7:167
7. Baró-Montel N, Vall-Laura L, Giné-Bordonaba J, Serrano-Prieto S, Usall J, et al. 2019. Double-sided battle: the role of ethylene during *Monilinia* spp. infection in peach at different phenological stages. *Plant Physiol. Biochem.* 144:324–33
8. Beno-Moualem D, Prusky D. 2000. Early events in the development of quiescent infection of avocado fruits against *Colletotrichum gloeosporioides*. *Phytopathology* 90:553–59
9. Blanco-Ulate B, Labavitch JM, Powell ALT, Cantu D. 2016. Hitting the wall: plant cell wall implications during *Botrytis cinerea* infections. In *Botrytis*: *The Fungus*, *the Pathogen and its Management in Agricultural Systems*, ed. S Fillinger, Y Elad, pp. 361–86. Cham: Springer
10. Blanco-Ulate B, Morales-Cruz A, Amrine K, Labavitch JM, Powell A, Cantu D. 2014. Genome-wide transcriptional profiling of *Botrytis cinerea* genes targeting plant cell walls during infections of different hosts. *Front. Plant Sci.* 5:435
11. Blanco-Ulate B, Vincenti E, Powell AL, Cantu D. 2013. Tomato transcriptome and mutant analyses suggest a role for plant stress hormones in the interaction between fruit and *Botrytis cinerea*. *Front. Plant Sci.* 4:142
12. Brummell DA. 2006. Cell wall disassembly in ripening fruit. *Funct. Plant Biol.* 33:103–19
13. Buonaurio R, Iriti M, Romanazzi G. 2009. Induced resistance to plant diseases caused by oomycetes and fungi. *Petria* 19:130–48
14. Buswell W, Schwarzenbacher RE, Luna E, Sellwood M, Chen B, et al. 2018. Chemical priming of immunity without costs to plant growth. *New Phytol.* 218:1205–16
15. Cantu D, Blanco-Ulate B, Yang L, Labavitch JM, Bennett AB, Powell ALT. 2009. Ripening regulated susceptibility of tomato fruit to *Botrytis cinerea* requires *NOR* but not *RIN* or ethylene. *Plant Physiol.* 150:1434–49
16. Cantu D, Vicente AR, Greve LC, Dewey FM, Bennett AB, et al. 2008. The intersection between cell wall disassembly, ripening, and fruit susceptibility to *Botrytis cinerea*. *PNAS* 105:859–64
17. Cao D, Li H, Yi J, Zhang J, Che H, et al. 2011. Antioxidant properties of the mung bean flavonoids on alleviating heat stress. *PLOS ONE* 6:e21071
18. Cara B, Giovannoni J. 2008. Molecular biology of ethylene during tomato fruit development and maturation. *Plant Sci.* 175:106–13
19. Chakravarthy S, Tuori RP, D'Ascenzo MD, Fobert PR, Després C, Martin GB. 2003. The tomato transcription factor Pti4 regulates defense-related gene expression via GCC box and non-GCC box *cis* elements. *Plant Cell* 15:3033–50

20. Chaturvedi R, Krothapalli K, Makandar R, Nandi A, Sparks AA, et al. 2008. Plastid ω-3 desaturase-dependent accumulation of a systemic acquired resistance inducing activity in petiole exudates of *Arabidopsis thaliana* is independent of jasmonic acid. *Plant J.* 54:106–17
21. Chen Y, Grimplet J, David K, Castellarin SD, Terol J, et al. 2018. Ethylene receptors and related proteins in climacteric and non-climacteric fruits. *Plant Sci.* 276:63–72
22. Chen X, Li C, Wang H, Guo Z. 2019. WRKY transcription factors: evolution, binding, and action. *Phytopathol. Res.* 1:13
23. Cherian S, Figueroa CR, Nair H. 2014. 'Movers and shakers' in the regulation of fruit ripening: a cross-dissection of climacteric versus nonclimacteric fruit. *J. Exp. Bot.* 65:4705–22
24. Chervin C, El-Kereamy A, Roustan JP, Latche A, Lamon J, Bouzayen M. 2004. Ethylene seems required for the berry development and ripening in grape, a non-climacteric fruit. *Plant Sci.* 167:1301–5
25. Clark N, Nolan TM, Wang P, Song G, Montes C, et al. 2021. Integrated omics networks reveal the temporal signaling events of brassinosteroid response in *Arabidopsis*. *Nat. Commun.* 12:5858
26. Conrath U, Beckers GJ, Langenbach CJ, Jaskiewicz MR. 2015. Priming for enhanced defense. *Annu. Rev. Phytopathol.* 53:97–119
27. Conrath U, Pieterse CM, Mauch-Mani B. 2002. Priming in plant pathogen interactions. *Trends Plant Sci.* 7:210–16
28. Coqueiro DSO, de Souza AA, Takita MA, Munari Rodriguez C, Takeshi Kishi L, Machado MO. 2015. Transcriptional profile of sweet orange in response to chitosan and salicylic acid. *BMC Genom.* 16:288
29. Coronado-Partida LD, Serrano M, González-Estrada RR, Romanazzi G, Gutierrez P. 2021. Application of GRAS compounds to control soft rot in jackfruit (*Artocarpus heterophyllus* L.) caused by *Rhizopus stolonifer*. *TIP Rev. Esp. Cien. Quím.-Biol.* 24:e327
30. Curvers K, Seifi H, Mouille G, de Rycke R, Asselbergh B, et al. 2010. Abscisic acid deficiency causes changes in cuticle permeability and pectin composition that influence tomato resistance to *Botrytis cinerea*. *Plant Physiol.* 154:847–60
31. Danhash N, Wagemakers CA, van Kan JA, de Wit PJ. 1993. Molecular characterization of four chitinase cDNAs obtained from *Cladosporium fulvum*-infected tomato. *Plant Mol. Biol.* 22:1017–29
32. De Miccolis Angelini RM, Landi L, Raguseo C, Pollastro S, Faretra F, Romanazzi G. 2022. Tracking of diversity and evolution in the brown rot fungi *Monilinia fructicola*, *M. fructigena* and *M. laxa*. *Front. Microbiol.* 13:854852
33. Deng B, Wang W, Ruan C, Deng L, Yao S, et al. 2020. Involvement of CsWRKY70 in salicylic acid-induced citrus fruit resistance against *Penicillium digitatum*. *Hortic. Res.* 7:157
34. Ding Y, Zhao J, Nie Y, Fan B, Wu S, et al. 2016. Salicylic-acid-induced chilling- and oxidative-stress tolerance in relation to gibberellin homeostasis, c-repeat/dehydration-responsive element binding factor pathway, and antioxidant enzyme systems in cold-stored tomato fruit. *J. Agric. Food Chem.* 64:8200–6
35. Droby S, Prusky D, Jacoby B. 1987. Induction of an antifungal agent in unripe mango fruit to demonstrate their involvement in latent infections of *Alternaria alternata*. *Physiol. Mol. Plant Pathol.* 30:285–92
36. Durrant WE, Dong X. 2004. Systemic acquired resistance. *Annu. Rev. Phytopathol.* 42:185–209
37. El Ghaouth A, Arul J, Grenier J, Asselin A. 1992. Antifungal activity of chitosan on two post-harvest pathogens of strawberry fruits. *Phytopathology* 82:398–402
38. El-Kazzaz MK, Sommer NF, Kader AA. 1993. Ethylene effects on in vitro and in vivo growth of certain postharvest fruit infecting fungi. *Phytopathology* 83:998–1001
39. Eriksson EM, Bovy A, Manning K, Harrison L, Andrews J, et al. 2004. Effect of the colorless non-ripening mutation on cell wall biochemistry and gene expression during tomato fruit development and ripening. *Plant Physiol.* 136:4184–97
40. FAO. 2021. *Fruit and vegetables - your dietary essentials*. Int. Year Fruits Veg. Backgr. Pap., FAO, Rome. **https://www.fao.org/3/cb2395en/cb2395en.pdf**
41. Farinati S, Rasori A, Varotto S, Bonghi C. 2017. Rosaceae fruit development, ripening and post-harvest: an epigenetic perspective. *Front. Plant Sci.* 8:1247
42. Fedorina J, Tikhonova N, Ukhatova Y, Ivanov R, Khlestkina E. 2022. Grapevine gene systems for resistance to gray mold *Botrytis cinerea* and powdery mildew *Erysiphe necator*. *Agronomy* 12:499

43. Feliziani E, Landi L, Romanazzi G. 2015. Preharvest treatments with chitosan and other alternatives to conventional fungicides to control postharvest decay of strawberry. *Carbohydr. Polym.* 132:111–17
44. Feliziani E, Santini M, Landi L, Romanazzi G. 2013. Pre- and postharvest treatment with alternatives to synthetic fungicides to control postharvest decay of sweet cherry. *Postharvest Biol. Technol.* 78:133–38
45. Flaishman MA, Kolattukudy PE. 1994. Timing of fungal invasion using host's ripening hormone as a signal. *PNAS* 91:6579–83
46. Fu ZQ, Dong X. 2013. Systemic acquired resistance: turning local infection into global defense. *Annu. Rev. Plant Biol.* 64:839–63
47. Gallusci P, Hodgman C, Teyssier E, Seymour GB. 2016. DNA methylation and chromatin regulation during fleshy fruit development and ripening. *Front. Plant Sci.* 7:807
48. Ge X, Zhu Y, Li Z, Bi Y, Yang J, Zhang J, Prusky D. 2021. Preharvest multiple fungicide stroby sprays promote wound healing of harvested potato tubers by activating phenylpropanoid metabolism. *Postharvest Biol. Technol.* 171:111328
49. Giovannoni JJ. 2001. Molecular biology of fruit maturation and ripening. *Annu. Rev. Plant Physiol. Plant Mol. Biol.* 52:725–49
50. Giovannoni JJ. 2004. Genetic regulation of fruit development and ripening. *Plant Cell* 16:S170–80
51. Gu YQ, Wildermuth MC, Chakravarthy S, Loh YT, Yang C, et al. 2002. Tomato transcription factors pti4, pti5, and pti6 activate defense responses when expressed in *Arabidopsis*. *Plant Cell* 14:817–31
52. Guidarelli M, Zubini P, Nanni V, Bonghi C, Rasori A, et al. 2014. Gene expression analysis of peach fruit at different growth stages and with different susceptibility to *Monilinia laxa*. *Eur. J. Plant Pathol.* 140:503–13
53. Haile ZM, Malacarne G, Pilati S, Sonego P, Moretto M, et al. 2020. Dual transcriptome and metabolic analysis of *Vitis vinifera* cv. Pinot Noir berry and *Botrytis cinerea* during quiescence and egressed infection. *Front. Plant Sci.* 10:1704
54. Hashmi MS, East AR, Palmer JS, Heyes JA. 2014. Strawberries inoculated after hypobaric treatment exhibit reduced fungal decay suggesting induced resistance. *Acta Hortic.* 1053:163–68
55. He P, Warren RF, Zhao T, Shan L, Zhu L, et al. 2001. Overexpression of Pti5 in tomato potentiates pathogen-induced defense gene expression and enhances disease resistance to *Pseudomonas syringae* pv. *tomato*. *Mol. Plant-Microbe Interact.* 14:1453–57
56. Iqbal Z, Singh Z, Khangura R, Ahmad S. 2012. Management of citrus blue and green moulds through application of organic elicitors. *Australas. Plant Pathol.* 41:69–77
57. Jaskiewicz M, Conrath U, Peterhänsel C. 2011. Chromatin modification acts as a memory for systemic acquired resistance in the plant stress response. *EMBO Rep.* 12:50–55
58. Jia HF, Chai YM, Li CL, Lu D, Luo JJ, et al. 2011. Abscisic acid plays an important role in the regulation of strawberry fruit ripening. *Plant Physiol.* 157:188–99
59. Jia H, Jiu S, Zhang C, Wang C, Tariq P, et al. 2016. Abscisic acid and sucrose regulate tomato and strawberry fruit ripening through the abscisic acid-stress-ripening transcription factor. *Plant Biotechnol. J.* 14:2045–65
60. Jin P, Zheng C, Huang Y-P, Wang X-L, Luo Z-S, Zheng Y-H. 2016. Hot air treatment activates defense responses and induces resistance against *Botrytis cinerea* in strawberry fruit. *J. Integr. Agric.* 15:2658–65
61. Joyce DC, Johnson GI. 1999. Prospects for exploitation of natural disease resistance in harvested horticultural crops. *Postharvest News Inf.* 10:45–48
62. Jung HW, Tschaplinski TJ, Wang L, Glazebrook J, Greenberg JT. 2009. Priming in systemic plant immunity. *Science* 324:89–91
63. Jung SC, Martinez-Medina A, Lopez-Raez JA, Pozo MJ. 2012. Mycorrhiza-induced resistance and priming of plant defenses. *J. Chem. Ecol.* 38:651–64
64. Kader AA, ed. 2002. *Postharvest Technology of Horticultural Crops*. Berkeley: Univ. Calif. Agric. Nat. Resour.
65. Kamo T, Hirai N, Iwami K, Fujioka D, Ohigashi H. 2001. New phenylphenalenones from banana fruit. *Tetrahedron* 57:7649–56
66. Kamo T, Hirai N, Tsuda M, Fujioka D, Ohigashi H. 2000. Changes in the content and biosynthesis of phytoalexins in banana fruit. *Biosci. Biotechnol. Biochem.* 64:2089–98
67. Karagiannis E, Michailidis M, Tanou G, Samiotaki M, Karamanoli K, et al. 2018. Ethylene-dependent and -independent superficial scald resistance mechanisms in 'Granny Smith' apple fruit. *Sci. Rep.* 8:11436

68. Klee HJ, Giovannoni JJ. 2011. Genetics and control of tomato fruit ripening and quality attributes. *Annu. Rev. Genet.* 45:41–59
69. Landi L, De Miccolis Angelini RM, Pollastro S, Feliziani E, Faretra F, Romanazzi G. 2017. Global transcriptome analysis and identification of differentially expressed genes in strawberry after preharvest application of benzothiadiazole and chitosan. *Front. Plant Sci.* 8:1658
70. Landi L, Feliziani E, Romanazzi G. 2014. Expression of defense genes in strawberry fruit treated with different resistance inducers. *J. Agric. Food Chem.* 62:3047–56
71. Landi L, Peralta-Ruiz Y, Chaves-López C, Romanazzi G. 2021. Chitosan coating enriched with *Ruta graveolens* L. essential oil reduces postharvest anthracnose of papaya (*Carica papaya* L.) and modulates defense-related gene expression. *Front. Plant Sci.* 12:765806
72. Lasanajak Y, Minocha R, Minocha SC, Goyal R, Fatima T, et al. 2014. Enhanced flux of substrates into polyamine biosynthesis but not ethylene in tomato fruit engineered with yeast S-adenosylmethionine decarboxylase gene. *Amino Acids* 46:729–42
73. Lee S, Hong JC, Jeon WB, Chung YS, Sung S, et al. 2009. The salicylic acid-induced protection of non-climacteric unripe pepper fruit against *Colletotrichum gloeosporioides* is similar to the resistance of ripe fruit. *Plant Cell Rep.* 28:1573–80
74. Lee WS, Rudd JJ, Hammond-Kosack KE, Kanyuka K. 2014. *Mycosphaerella graminicola* LysM effector-mediated stealth pathogenesis subverts recognition through both CERK1 and CEBiP homologues in wheat. *Mol. Plant-Microbe Interact.* 27:236–43
75. Lehmann S, Serrano M, L'Haridon F, Tjamos SE, Metraux JP. 2015. Reactive oxygen species and plant resistance to fungal pathogens. *Phytochemistry* 112:54–62
76. Lelievre JM, Latche A, Jones B, Bouzayen M, Pech JC. 1997. Ethylene and fruit ripening. *Physiol. Plant.* 101:727–39
77. Li C, Jia H, Chai Y, Shen Y. 2011. Abscisic acid perception and signaling transduction in strawberry: a model for non-climacteric fruit ripening. *Plant Signal. Behav.* 6:1950–53
78. Li S, Jiang H, Wang Y, Lyu L, Prusky D, et al. 2020. Effect of benzothiadiazole treatment on improving the mitochondrial energy metabolism involved in induced resistance of apple fruit during postharvest storage. *Food Chem.* 302:125288
79. Lorenzo O, Chico JM, Sánchez-Serrano JJ, Solano R. 2004. JASMONATE-INSENSITIVE1 encodes a MYC transcription factor essential to discriminate between different jasmonate-regulated defense responses in *Arabidopsis*. *Plant Cell* 16:1938–50
80. Lougheed EC, Murr DP, Berard L. 1978. Low pressure storage for horticultural crops. *HortScience* 13:21–27
81. Luna E, Beardon E, Ravnskov S, Scholes J, Ton J. 2016. Optimizing chemically induced resistance in tomato against *Botrytis cinerea*. *Plant Dis.* 100:704–10
82. Luna E, Bruce TJA, Roberts MR, Flors V, Ton J. 2012. Next-generation systemic acquired resistance. *Plant Physiol.* 158:844–53
83. Luna E, López A, Kooiman J, Ton J. 2014. Role of NPR1 and KYP in long-lasting induced resistance by β-aminobutyric acid. *Front. Plant Sci.* 5:184
84. Luria N, Sela N, Yaari M, Feygenberg O, Lers A, Prusky D. 2014. De-novo assembly of mango fruit peel transcriptome reveals mechanisms of mango response to hot water treatment. *BMC Genom.* 15:957
85. Malerba M, Cerana R. 2016. Chitosan effects on plant systems. *Int. J. Mol. Sci.* 17:996
86. Malinovsky FG, Fangel JU, Willats WG. 2014. The role of the cell wall in plant immunity. *Front. Plant Sci.* 5:178
87. Marcos JF, González-Candelas L, Zacarías L. 2005. Involvement of ethylene biosynthesis and perception in the susceptibility of citrus fruits to *Penicillium digitatum* infection and the accumulation of defence-related mRNAs. *J. Exp. Bot.* 56:2183–93
88. Mari M, Spadaro D, Casals C, Collina M, De Cal A, Usall J. 2019. Stone fruits. In *Postharvest Pathology of Fresh Horticultural Produce*, ed. L Palou, JL Smilanick, pp. 111–40. Boca Raton, FL: CRC Press
89. Martinez-Medina A, Flors V, Heil M, Mauch-Mani B, Pieterse CMJ, et al. 2016. Recognizing plant defense priming. *Trends Plant Sci.* 21:818–22
90. Mauch-Mani B, Baccelli I, Luna E, Flors V. 2017. Defense priming: an adaptive part of induced resistance. *Annu. Rev. Plant Biol.* 68:485–512

91. Mayda E, Marqués C, Conejero V, Vera P. 2000. Expression of a pathogen-induced gene can be mimicked by auxin insensitivity. *Mol. Plant-Microbe Interact.* 13:23–31
92. Meyer M, Huttenlocher F, Cedzich A, Procopio S, Stroeder J, et al. 2016. The subtilisin-like protease SBT3 contributes to insect resistance in tomato. *J. Exp. Bot.* 67:4325–38
93. Nieuwenhuizen NJ, Chen X, Pellan M, Zhang L, Guo L, et al. 2021. Regulation of wound ethylene biosynthesis by NAC transcription factors in kiwifruit. *BMC Plant Biol.* 21:411
94. Obianom C, Romanazzi G, Sivakumar D. 2019. Effects of chitosan treatment on avocado postharvest diseases and expression of phenylalanine ammonia-lyase, chitinase and lipoxygenase genes. *Postharvest Biol. Technol.* 147:214–21
95. Ordaz-Ortiz JJ, Marcus SE, Knox JP. 2009. Cell wall microstructure analysis implicates hemicellulose polysaccharides in cell adhesion in tomato fruit pericarp parenchyma. *Mol. Plant* 2:910–21
96. Osondu HAA, Akinola SA, Shoko T, Pillai SK, Sivakumar D. 2022. Coating properties, resistance response, molecular mechanisms and anthracnose decay reduction in green skin avocado fruit ('Fuerte') coated with chitosan hydrochloride loaded with functional compound. *Postharvest Biol. Technol.* 186:111812
97. Osorio S, Scossa F, Fernie AR. 2013. Molecular regulation of fruit ripening. *Front. Plant Sci.* 4:198
98. Pandey D, Rajendran SRCK, Gaur M, Sajeesh PK, Kumar A. 2016. Plant defense signaling and responses against necrotrophic fungal pathogens. *J. Plant Growth Regul.* 35:1159–74
99. Park S-W, Kaimoyo E, Kumar D, Mosher S, Klessig DF. 2007. Methyl salicylate is a critical mobile signal for plant systemic acquired resistance. *Science* 318:113–16
100. Penninckx IAM, Thomma BPHJ, Buchala A, Metraux JP, Broekaert WF. 1998. Concomitant activation of jasmonate and ethylene response pathways is required for induction of a plant defensin gene in *Arabidopsis*. *Plant Cell* 10:2103–13
101. Perkins-Veazie PM, Huber DJ, Brecht JK. 1996. In vitro growth and ripening of strawberry fruit in the presence of ACC, STS or propylene. *Ann. Appl. Biol.* 128:105–16
102. Petriccione M, Mastrobuoni F, Pasquariello MS, Zampella L, Nobis E, et al. 2015. Effect of chitosan coating on the postharvest quality and antioxidant enzyme system response of strawberry fruit during cold storage. *Foods* 4:501–23
103. Pieterse CM, Leon-Reyes A, Van der Ent S, Van Wees SC. 2009. Networking by small-molecule hormones in plant immunity. *Nat. Chem. Biol.* 5:308–16
104. Pieterse CM, Zamioudis C, Berendsen RL, Weller DM, Van Wees SC, Bakker PA. 2014. Induced systemic resistance by beneficial microbes. *Annu. Rev. Phytopathol.* 52:347–75
105. Porat R, Weiss B, Cohen L, Daus A, Goren R, Droby S. 1999. Effects of ethylene and 1-methylcyclopropene on the post-harvest qualities of 'Shamouti' oranges. *Postharvest Biol. Technol.* 15:155–63
106. Pristijono P, Wills RBH, Tesoriero L, Golding JB. 2018. Effect of continuous exposure to low levels of ethylene on mycelial growth of postharvest fruit fungal pathogens. *Horticulturae* 4:20
107. Prusky D. 1996. Pathogen quiescence in postharvest diseases. *Annu. Rev. Phytopathol.* 34:413–34
108. Prusky D, Alkan N, Fluhr R, Tesfaye M. 2013. Quiescent and necrotrophic lifestyle choice during postharvest disease development. *Annu. Rev. Phytopathol.* 51:155–76
109. Prusky D, Keen NT. 1993. Involvement of preformed antifungal compounds and the resistance of subtropical fruits to fungal decay. *Plant Dis.* 77:114–19
110. Prusky D, Kobiler I, Jacoby B, Sims JJ, Midland SL. 1985. Effects of inhibitors of avocado lipoxygenase: their possible relationship with the latency of *Colletotrichum gloeosporioides* on avocado fruits. *Physiol. Mol. Plant Pathol.* 27:269–79
111. Prusky D, Kobiler I, Plumbley R, Fuchs Y, Zauberman G. 1993. The effect of CO_2 levels on the symptom expression of *Colletotrichum gloeosporioides* on avocado fruits. *Plant Pathol.* 42:900–4
112. Prusky D, Wattad C, Koliber I. 1996. Effect of ethylene on the activation of quiescent infections of *Colletotrichum gloeosporioides* in avocado fruits. *Mol. Plant-Microbe Interact.* 9:864–68
113. Rajestary R, Landi L, Romanazzi G. 2021. Chitosan and postharvest decay of fresh fruit: meta-analysis of disease control and antimicrobial and eliciting activities. *Compr. Rev. Food Sci. Food Saf.* 20:563–82
114. Reddy BMV, Belkacemi K, Corcuff R, Castaigne F, Arul J. 2000. Effect of preharvest chitosan sprays on postharvest infection by *Botrytis cinerea* and quality of strawberry fruit. *Postharvest Biol. Technol.* 20:39–51

115. Romanazzi G, Feliziani E, Bautista-Baños S, Sivakumar D. 2017. Shelf life extension of fresh fruit and vegetables by chitosan treatment. *Crit. Rev. Food Sci. Nutr.* 57:579–601
116. Romanazzi G, Feliziani E, Sivakumar D. 2018. Chitosan, a biopolymer with triple action on postharvest decay of fruit and vegetables: eliciting, antimicrobial and film-forming properties. *Front. Microbiol.* 9:2745
117. Romanazzi G, Mlikota Gabler F, Smilanick JL. 2006. Preharvest chitosan and postharvest UV-C irradiation treatments suppress gray mold of table grapes. *Plant Dis.* 90:445–50
118. Romanazzi G, Mlikota Gabler F, Margosan DA, Mackey BE, Smilanick JL. 2009. Effect of chitosan dissolved in different acids on its ability to control postharvest gray mold of table grape. *Phytopathology* 99:1028–36
119. Romanazzi G, Moumni M. 2022. Chitosan and other edible coatings to manage postharvest decay, extend shelf life, and reduce losses and wastes of fresh fruit and vegetables. *Curr. Opin. Biotechnol.* 78:102834
120. Romanazzi G, Murolo S, Feliziani E. 2013. Effects of an innovative strategy to contain grapevine Bois noir: field treatment with resistance inducers. *Phytopathology* 103:785–91
121. Romanazzi G, Nigro F, Ippolito A, Salerno M. 2001. Effect of short hypobaric treatments on postharvest rots of sweet cherries, strawberries and table grapes. *Postharvest Biol. Technol.* 22:1–6
122. Romanazzi G, Orçonneau Y, Moumni M, Davillerd Y, Marchand PA. 2022. Basic substances, a sustainable tool to complement and eventually replace synthetic pesticides in the management of pre and postharvest diseases: reviewed instructions for users. *Molecules* 27(11):3484
123. Romanazzi G, Sanzani SM, Bi Y, Tian S, Gutierrez-Martinez P, Alkan N. 2016. Induced resistance to control postharvest decay of fruit and vegetables. *Postharvest Biol. Technol.* 122:82–94
124. Romanazzi G, Smilanick JL, Feliziani E, Droby S. 2016. Integrated management of postharvest gray mold on fruit crops. *Postharvest Biol. Technol.* 113:69–76
125. Saidi L, Duanis-Assaf D, Galsarker O, Maurer D, Alkan N, Poverenov E. 2021. Elicitation of fruit defense response by active edible coatings embedded with phenylalanine to improve quality and storability of avocado fruit. *Postharvest Biol. Technol.* 174:111442
126. Saleh A, Withers J, Mohan R, Marqués J, Gu Y, et al. 2015. Posttranscriptional modifications of the master transcriptional regulator NPR1 enable dynamic but tight control of immune responses. *Cell Host Microbe* 18(2):169–82
127. Silva CJ, Adaskaveg JA, Mesquida-Pesci SD, Ortega-Salazar IB, Pattathil S, et al. 2023. *Botrytis cinerea* infection accelerates ripening and cell wall disassembly to promote disease in tomato fruit. *Plant Physiol.* 191(1):575–90
128. Silva CJ, van den Abeele C, Ortega-Salazar I, Papin V, Adaskaveg JA, et al. 2021. Host susceptibility factors render ripe tomato fruit vulnerable to fungal disease despite active immune responses. *J. Exp. Bot.* 72:2696–99
129. Srivastava MK, Dwivedi UN. 2000. Delayed ripening of banana fruit by salicylic acid. *Plant Sci.* 158:87–96
130. Tada Y, Spoel SH, Pajerowska-Mukhtar K, Mou Z, Song J, et al. 2008. Plant immunity requires conformational changes [corrected] of NPR1 via S-nitrosylation and thioredoxins. *Science* 321:952–56
131. Thomma BP, Eggermont K, Tierens KF, Broekaert WF. 1999. Requirement of functional ethylene-insensitive 2 gene for efficient resistance of *Arabidopsis* to infection by *Botrytis cinerea*. *Plant Physiol.* 121:1093–102
132. Ton J, Mauch-Mani B. 2004. β-amino-butyric acid-induced resistance against necrotrophic pathogens is based on ABA-dependent priming for callose. *Plant J.* 38:119–30
133. Vallad GE, Goodman RM. 2004. Systemic acquired resistance and induced systemic resistance in conventional agriculture. *Crop Sci.* 44:1920–34
134. Vall-Llaura N, Torres R, Teixidó N, Usall J, Giné-Bordonaba J. 2022. Untangling the role of ethylene beyond fruit development and ripening: a physiological and molecular perspective focused on the *Monilinia*-peach interaction. *Sci. Hortic.* 301:111123
135. Van der Ent S, Pieterse CMJ. 2012. Ethylene: multi-tasker in plant-attacker interactions. *Annu. Plant Rev.* 44:343–77
136. van Loon LC, van Strien EA. 1999. The families of pathogenesis-related proteins, their activities, and comparative analysis of PR-1 type proteins. *Physiol. Mol. Plant Pathol.* 55:85–97

137. Walters DR, Ratsep J, Havis ND. 2013. Controlling crop diseases using induced resistance: challenges for the future. *J. Exp. Bot.* 64:1263–80
138. Wang B, He X, Bi Y, Jiang H, Wang Y, et al. 2021. Preharvest sprays with sodium nitroprusside induce resistance in harvested muskmelon against the pink rot disease. *J. Food Process. Preserv.* 45:e15339
139. Wang R, Lammers M, Tikunov Y, Bovy AG, Angenent GC, de Maagd RA. 2020. The *rin*, *nor* and *Cnr* spontaneous mutations inhibit tomato fruit ripening in additive and epistatic manners. *Plant Sci.* 294:110436
140. Wasternack C, Hause B. 2013. Jasmonates: biosynthesis, perception, signal transduction and action in plant stress response, growth and development. An update to the 2007 review in *Annals of Botany*. *Ann. Bot.* 111:1021–58
141. Wasternack C, Song S. 2017. Jasmonates: biosynthesis, metabolism, and signaling by proteins activating and repressing transcription. *J. Exp. Bot.* 68:1303–21
142. Waszczak C, Carmody M, Kangasjärvi J. 2018. Reactive oxygen species in plant signaling. *Annu. Rev. Plant Biol.* 69:209–36
143. Wilkinson SW, Pastor V, Paplauskas S, Pétriacq P, Luna E. 2018. Long-lasting β-aminobutyric acid-induced resistance protects tomato fruit against *Botrytis cinerea*. *Plant Pathol.* 67:30–41
144. Wu HL, Lv H, Li L, Liu J, Mu SH, et al. 2015. Genome-wide analysis of the AP2/ERF transcription factors family and the expression patterns of DREB genes in Moso Bamboo (*Phyllostachys edulis*). *PLOS ONE* 10:e0126657
145. Wu X, Wu H, Yu M, Ma R, Yu Z. 2022. Effect of combined hypobaric and cold storage on defense-related enzymes in postharvest peach fruit during ripening. *Acta Physiol. Plant.* 44:93
146. Xoca-Orozco L-Á, Cuellar-Torres EA, González-Morales S, Gutiérrez-Martínez P, López-García U, et al. 2017. Transcriptomic analysis of avocado Hass (*Persea americana* Mill) in the interaction system fruit-chitosan-*Colletotrichum*. *Front. Plant Sci.* 8:956
147. Xu J, Xu H, Liu Y, Wang X, Xu Q, Deng X. 2015. Genome-wide identification of sweet orange (*Citrus sinensis*) histone modification gene families and their expression analysis during the fruit development and fruit-blue mold infection process. *Front. Plant Sci.* 6:607
148. Xu ZS, Chen M, Li LC, Ma YZ. 2008. Functions of the ERF transcription factor family in plants. *Botany* 86:969–77
149. Yan Y, Borrego E, Kolomiets MV. 2013. Jasmonate biosynthesis, perception and function in plant development and stress responses. In *Lipid Metabolism*, ed. RV Baez. London: IntechOpen
150. Yang J, Zhang KQ. 2019. Chitin synthesis and degradation in fungi: biology and enzymes. *Adv. Exp. Med. Biol.* 1142:153–67
151. Zainuri JDC, Wearing AH, Coates L, Terry L. 2001. Effect of phosphonate and salicylic acid treatments on anthracnose disease development and ripening of 'Kensington Pride' mango fruit. *J. Exp. Agric.* 41:805–13
152. Zhang W, Jiang H, Cao J, Jiang W. 2021. UV-C treatment controls brown rot in postharvest nectarine by regulating ROS metabolism and anthocyanin synthesis. *Postharvest Biol. Technol.* 180:111613
153. Zhang Z, Tian CP, Zhang Y, Li CZY, Li X, et al. 2020. Transcriptomic and metabolomic analysis provides insights into anthocyanin and procyanidin accumulation in pear. *BMC Plant Biol.* 20:129
154. Zimmerli L, Jakab G, Metraux JP, Mauch-Mani B. 2000. Potentiation of pathogen-specific defense mechanisms in *Arabidopsis* by β-aminobutyric acid. *PNAS* 97:12920–25

Annual Review of Phytopathology

Volume 61, 2023

Contents

Errata

An online log of corrections to *Annual Review of Phytopathology* articles may be found at http://www.annualreviews.org/errata/phyto