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Induced resistance to control postharvest decay of fruit and vegetables

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Highlights

- Several treatments applied to fruit and vegetables can trigger host defenses
- Induced resistance can be systemic acquired resistance or induced systemic resistance
- Biocontrol agents, physical means, and natural compounds elicit host defense system
- Induced resistance is a strategy that contributes to control of postharvest decay
- Induced resistance can increase amounts of beneficial antioxidant compounds

Contents

Abstract

More than one third of harvested fruit and vegetables are lost and do not reach the customers mainly due to postharvest decay. During the last decade, several postharvest fungicides have been excluded from the market, or their allowed residues have been significantly decreased. Therefore, there is growing interest in eco-friendly and safe alternatives to synthetic fungicides. Induced resistance has gained increasing attention as a sustainable strategy to manage postharvest decay of fruit and vegetables. Their natural resistance can be increased by various means, such as biocontrol agents or their secreted elicitors. Alternatively, physical means, such as UV-C, ozone, and heat treatment, can prime plant resistance through abiotic stress. Moreover, various defense-related phytohormones, biological elicitors, non-organic elicitors, and volatile organic compounds have been shown to induce plant resistance. During the last decades, new technologies have enabled the evaluation of gene expression, such as quantitative real time PCR and the most recent next-generation sequencing, and thus the quantification of physiological changes, which have revealed new knowledge about preharvest and postharvest induced resistance in response to various treatments. These techniques allow optimization of postharvest application of the control means, although these data cannot disregard the evaluation of in vivo effectiveness. The elicitation of host defenses prevents the appearance of resistant isolates of pathogens. Induced resistance can lead to increased levels of phenolic compounds in the plant tissues, which often have antioxidant properties that are highly beneficial to humans. Moreover, induced resistance preserves the natural microflora, which is rich in potential biocontrol agents, and which provides a combined approach in the control of postharvest decay that is sustainable and safe for both growers and consumers. This approach meets the requirements of integrated disease management on sustainable use of pesticides that in the EU is implemented through Directive 128/2009. This review summarizes recent achievements and knowledge of the elicitation of host defenses to control postharvest decay of fruit and vegetables, and provides an outlook on the new challenges in this fascinating subject.

Keywords: biostimulants, elicitors, induced systemic resistance, resistance inducers, systemic acquired resistance

1. Introduction

Recent investigations have shown that more than one third of harvested fruit and vegetables are lost (FAO, 2011; USDA, 2014; OECD, 2014). Most losses occur due to pathogen infections in the field or after harvest, which lead to postharvest decay, when fruit ripen and vegetables senesce. Moreover, during the last decade, several postharvest fungicides that often had wide spectra of targets have been withdrawn from the market, due to: (i) selection of resistant fungal isolates; (ii) toxicity to humans and the environment; (iii) increasing consumer concern toward risk of pesticide residues on products, with the consequent strict requirements from several major supply chains for the quantity and number of active ingredient(s) on foodstuffs, as percentages of maximum residue limit; and (iv) increasing costs of registration and re-registration (Romanazzi et al., 2016a). Therefore, there is growing interest in finding cheap, safe, and eco-friendly alternatives to synthetic fungicides for the control of postharvest decay of fresh produce. Induction of plant resistance by biological, chemical, or physical means is considered a sustainable strategy to manage postharvest decay of fruit and vegetables. This approach has gained increasing interest during recent years, in which we can see a high trend in papers dealing with induced resistance, from few ones recorded 30 years ago to more than 800 recorded yearly in 2013-2015 (Fig. 1), and due to new tools, further knowledge has been obtained on host responses to various methods of control (Hershkovitz et al., 2013; Gapper et al., 2014).

The beneficial effects of induced resistance in the postharvest environment were originally demonstrated about two decades ago. For example, the use of heat treatment to decrease chilling injury and disease incidence in fruit through the induction of host resistance has been extensively studied (Lurie and Pedreschi, 2014). Ultraviolet-C (UV-C) irradiation and exposure to sunlight have been shown to induce resistance to pathogens and chilling tolerance in many harvested commodities (Wilson et al., 1994; Ruan et al., 2015; Sivankalyani et al., 2016). More recently, different inducers, such as cell-wall components, plant extracts, compounds of biological origin, and synthetic chemicals, have been shown to trigger plant resistance to pathogen attack locally and systemically (Walters and Fountaine, 2009). Moreover, biological control agents can induce plant resistance to pathogens (Vallad and Goodman, 2004; Da Rocha and Hammerschmidt, 2005; Lyon, 2007). However, to correctly induce resistance in different plants, it is necessary to know and understand the host–microbe interactions, and the effects on postharvest physiology and handling of the different fruit and vegetables (Da Rocha and Hammerschmidt, 2005).

Here, we review the different biological, physical, and chemical inducers that have been shown to control postharvest diseases of fruit and vegetables, and highlight their proposed mechanisms of action.

2. Mechanisms involved in induced resistance

Various biotic inducers (e.g., fungi, bacteria, viruses, phytoplasma, insects) and abiotic stresses (e.g., chemical and physical inducers) can trigger resistance in plants, which is known as 'induced resistance' (Pieterse et al., 2012; Walters et al., 2013; Pieterse et al., 2014). These can produce rapid expression of defense responses (Conrath et al., 2002; Fu and Dong, 2013). Examples of treatments able to induce resistance in host tissues and of representative mechanisms involved are reported in Fig. 2. We can imagine induced resistance as produced by an array of treatments that elicit a cloud of defense responses. There are two types of induced resistance in plants: systemic acquired resistance (SAR) and induced systemic resistance (ISR). Both of these mechanisms can induce defenses that confer long-lasting protection against a broad spectrum of microorganisms, and are mediated by phytohormones, such as salicylic acid (SA), jasmonic acid (JA), and ethylene (ET). SAR requires the signal molecule SA and is associated with accumulation of pathogenesis-related (PR) proteins, which are believed to contribute to resistance (Durrant and Dong, 2004). Instead, the ISR pathway functions independently of SA, while it is dependent on JA and ET (van Wees et al., 1999).

This induced resistance does not directly activate plant defense responses, but activates the plant to a state of 'alertness', so that a future pathogen attack will be strongly and efficiently responded to. This phenomenon is also known as the 'priming effect' (Conrath et al., 2006; Jung et al., 2009), and one of the most known priming effects is root colonization by plant-growth-promoting rhizobacteria (PGPR), which induce plant development and ISR-mediated resistance (Vallad and Goodman, 2004; Verhage et al., 2010). While PGPR induces ISR, other inducers can activate SAR or both of these systems.

2.1. Systemic acquired resistance (SAR)

The mechanisms of SAR are based on SA-mediated defense. The transcription factor Nonexpressor of pathogenesis-related genes 1 (NPR1) is considered to be the master regulator of SA and SAR. Here, biotic, abiotic, chemical, and physical inducers can trigger defense responses locally, and can also induce the production of suggested mobile immune signals, including SA, methyl salicylic acid (MeSA), azelaic acid (AzA), glycerol 3-phosphate, and abietane-diterpenoid-dehydroabietinal (Park et al., 2007; Chaturvedi et al., 2012). One or more of these signals can lead to systemic defense 'memory' that can last for weeks to months, to protect the plant from future infection (Jung et al., 2009).

Cellular redox and reactive oxygen species (ROS) are modified during SAR. Both primary and secondary oxidative bursts are required for the onset of SAR (Alvarez et al., 1998). Furthermore, cellular redox has been shown to be modified during SAR, as initially oxidized, and then reduced. NPR1, a master regulator of SA and SAR, was shown to be reduced by thioredoxin. The NPR1 oligomer is disrupted and its monomers enter the nucleus, and activate SA-mediated defense (Tada et al., 2008). The transcription factor NPR1 and the activated SA-mediated defense response result in SAR and the activation of approximately 10% of the plant transcriptome (Fu and Dong, 2013). This defense response includes direct targets of the WRKY domain transcription factor family, and synthesis and secretion of various PR proteins, with activation of the mitogen-activated protein kinase (MAPK) cascade, the DNA repair machinery, histone modifications, and a whole arsenal of defense-related transcripts (Fu and Dong, 2013).

2.2. Induced systemic resistance (ISR)

Induced systemic resistance is known to reprogram plant-induced mechanisms based on JA and ET defenses that alleviate physiological, abiotic, and biotic stresses. Elicitors and effectors known as ‘microbe-associated molecular patterns’ can be identified by plant receptors (Bent and Mackey, 2007). This recognition has a key role in activation of innate immunity. The ISR responses to biotic or abiotic stresses are diverse and can elicit plant defense responses. These responses include: MAPK signaling, generation of ROS, the octadecanoic pathway (which synthesizes oxidized fatty acid signals known as oxylipins), the phenylpropanoid pathway (which is involved in terpenoid and phytoalexin biosynthesis), increased levels of phenolic compounds, lignification at the site of pathogen infection, and cell-wall metabolism (Shoresh et al., 2010; Lloyd et al., 2011). ISR activates hydrogen peroxide producers on the one hand, such as oxalate oxidase and glucose oxidase, and antioxidants on the other hand, such as peroxidase (POD) and superoxide dismutase (SOD) (Shoresh et al., 2010). The balance between these two determines the ROS levels, and high ROS levels can lead to lipid peroxidation (Mittler, 2002). Additionally, several key transcripts, such as lipoxygenase (LOX1), phenylalanine ammonia lyase (PAL), and heat-shock proteins (HSPs), have been shown to be induced during ISR (Bi et al., 2007; Shoresh et al., 2010).

2.3. Other mechanisms of induced resistance

While the main induced resistance mechanisms are SAR and ISR, some processes of induced resistance combine these two resistance mechanisms in various ways. For example, β-aminobutyric acid (BABA)-induced resistance involves both SA-dependent and abscisic acid (ABA)-dependent defense mechanisms (Buonaurio et al., 2009; Pieterse et al., 2009). The relative importance of these phytohormone-dependent defenses varies according to the nature of the challenge pathogen. Indeed, BABA-induced resistance against *Botrytis cinerea* resembles SAR and requires SA accumulation (Zimmerli et al., 2000), while the ABA-dependent pathway, which is associated to callose deposition, is necessary against *Hyaloperonospora parasitica* and *Plectosphaerella cucumerina* (Zimmerli et al., 2000; Ton and Mauch-Mani, 2004).

3. Induced resistance by biocontrol agents

Numerous reports have indicated that biocontrol agents, such as antagonistic yeast, can increase fruit resistance against postharvest diseases (Ippolito et al., 2000; Tian et al., 2006; Janisiewicz et al., 2008; Droby et al., 2016; Spadaro and Droby, 2016) (Tab. 1). As defense responses in plants are complex and involve both biochemical and structural barriers, the mechanisms of biocontrol agents are usually multiple. One is the secretion of extracellular lytic enzymes, as for *Pichia membranifaciens* and *Cryptococcus albidus*, where these can attach and degrade the hyphae of *Monilinia fructicola, Penicillium expansum* and *Rhizopus stolonifer*, both in vitro and in vivo (Chan and Tian, 2005). The second is the accumulation of host PR proteins (Jijakli and Lepoivre, 1998). These are strongly induced in response to wounding or infection by pathogens, and they accumulate abundantly at the site of infection, to contribute to SAR (Ryals et al., 1996). Treatment with *Cryptococcus laurentii* noticeably stimulated expression of the β-1,3-glucanase (*Glu-1*) gene in jujube fruit (Tian et al., 2007), which suggested that *Glu-1* has a role in defense responses to fungal pathogens. *C. laurentii* and *P. membranifaciens* have been shown to reduce disease incidence in pears (Tian et al., 2006), peaches (Xu et al., 2008a), and table grapes (Meng and Tian, 2009), via enhancement of defense-related enzyme activities, such as chitinase (CHT), β-1,3-glucanase (GLU) and PAL. *Aureobasidium pullulans* induced the production of CHT, GLU and POD in apple tissues starting 24 h after treatment, which reached maximum levels 48 h and 96 h after treatment (Ippolito *et al.*, 2000). Then, the induction of host antioxidant enzymes and specific proteins has a relevant role. *P. membranifaciens* can induce host hydrogen peroxide metabolism, to enhance the resistance of sweet cherry against blue mold by *P. expansum* (Chan and Tian, 2006). Additionally, four antagonistic yeasts (*P. membranifaciens*, *C. laurentii*, *Candida guilliermondii*, *Rhodotorula glutinis*) can stimulate catalase (CAT) and POD activity, and reduce the levels of protein carbonylation in response to ROS caused by *M. fructicola* in peach fruit (Xu et al., 2008b). These results suggest that yeast treatments can alleviate protein carbonylation and pathogen-induced oxidative damage, which implies that the antioxidant defense response is involved in the mechanisms of microbial biocontrol agents against fungal pathogens. Other yeasts, such as *P. membranifaciens, C. guilliermondii,* and *R. glutinis,* have also been shown to stimulate the expression of both PR proteins and redox enzymes (e.g., CHT, GLU, CAT, POD) in peach fruit during all storage periods (Xu et al., 2008b). *Candida saitoana* induced postharvest systemic resistance in apple fruit against *B. cinerea*, with increased activities of CHT and GLU reported (El Ghaouth et al., 2003b). *Candida intermedia* induced strawberry fruit defense mechanisms, which included β-1,3-exoglucanase, and was effective in postharvest control of *B. cinerea* (Huang et al., 2011a). Higher PAL, CHT and GLU activities were observed when *Pichia guilliermondii* and *C. laurenti* were applied to harvested tomatoes (Zhao et al., 2008).

4. Induced resistance by physical means

Several physical means, such as ultraviolet-C (UV-C) light, heat, hypobaric and hyperbaric treatments are known to be effective in controlling postharvest decay of fruit and vegetables (Usall et al., 2016). The advantage of most of these relies on direct effects on the pathogen without leaving residues on the fruit (Sanzani et al., 2009a). Moreover, these can induce several changes in host tissues, including increased resistance to abiotic and biotic stress (Tab. 2).

4.1. UV-C irradiation

The eliciting effects of UV-C irradiation have been studied for different fruit and vegetables (Charles and Arul, 2007). The first studies on the effects of UV-C irradiation on host tissues were carried out with carrots, the phytoalexin content of which was increased by the treatment (Mercier et al., 1993). Strawberries exposed to UV-C at 0.50 kJ m^{-2} and 1.00 kJ m^{-2} increased their PAL activity 12 h after treatment (Nigro et al., 2000). Peach fruit treated with UV-C showed increases in PAL, CHT and GLU activities (El Ghaouth et al., 2003a). Tomatoes exposed to UV-C and later inoculated with *R. stolonifer* showed 40% reduction in polygalacturonase (PG) activity, as compared to the control 72 h after the challenge (Stevens et al., 2004). The application of UV-C to harvested table grape berries increased the content of trans-resveratrol and catechin in the skin (Cantos et al., 2000; Romanazzi et al., 2006). A higher production of both compounds occurred when the berries were treated with chitosan 48 h before harvest and later exposed to UV-C irradiation (Romanazzi et al., 2006).

4.2. Heat treatment

Temperature (both high and low) is one of the oldest means to control postharvest diseases of fruit and vegetables. In particular, the use of low temperature is the most diffuse means of control, and nowadays imperfect management of this cold chain can cause heavy losses of fresh produce (Romanazzi et al., 2016a). The physiological changes in host tissues induced by heat treatment were well described by Lurie and Pedreschi (2014). In peaches exposed to hot water treatment, cell-wall genes involved in ripening, such as β-galactosidase, pectin lyase, PG, and pectin methyl esterase, showed general decreased expression, while ROS scavenging genes and PAL, CHT, and HSP70 showed increased expression (Spadoni et al., 2014). A microarray analysis run on apples treated at 45 °C for 45 min and inoculated with *P. expansum* showed up-regulation of HSP, HS cognate protein, and HS transcription factor genes, which were linked to resistance of the fruit to the heat stress (Spadoni et al., 2015). Thus, this short hot water treatment has been shown to induce resistance to chilling and pathogens in various fruit (Fallik, 2004; Lurie and Pedreschi, 2014).

4.3. Hypobaric and hyperbaric treatment

Hypobaric and hyperbaric treatments rely on the use of pressure different from atmospheric pressure over a short period of time. These treatments do not have direct effects on the pathogen, although they affect the host tissues (Romanazzi et al., 2008). For hyperbaric treatment the occurrence of physiological changes is presumed, but not yet clarified, while the variations in host tissues induced by hypobaric applications have been well known since the middle of the last century, when they were applied to fresh fruit to delay ripening (Burg and Burg, 1966). The reduced pressure affects ET metabolism, which reduces respiration, delays ripening, and makes the fruit less prone to decay (Lougheed et al., 1978). Short hypobaric treatment has been shown to be an effective means of control of postharvest decay of strawberries, sweet cherries, and table grapes (Romanazzi et al., 2001), and increased activities of PAL, CHT, and POD were observed in strawberry exposed to 0.5 atm for 4 h (Hashmi et al., 2013). In contrast, the mechanisms of action of hyperbaric treatments are still not clear. However, this treatment changed lycopene accumulation in tomatoes, as it reduced during storage and increased during ripening (Liplap et al., 2013).

5. Induced resistance by natural and synthetic chemicals

5.1. Phytohormones and chemical elicitors

Phytohormones are well-known in the control of defense responses to pathogens and in the modulation of plant induced resistance (Alkan and Fortes, 2015). A central role in the regulation of plant immune responses has been ascribed to the defense hormones SA, JA, ABA, and ET in the regulation of plant–pathogen interactions (Fujita et al., 2006; Spoel and Dong, 2008). Gibberellic acid, auxin indolacetic acid, brassinosteroids (BR), and cytokinines have recently emerged as important modulators of plant defenses against microorganisms, mostly based on vegetative tissue data and on the lifestyle of the infecting pathogen (Robert-Seilaniantz et al., 2011). The SA and JA signaling pathways are generally considered as antagonistic and are dependent on NPR1 (Spoel et al., 2007; Spoel and Dong, 2008; Pieterse et al., 2012). This interplay between SA and JA was suggested to optimize the host response to the pathogen lifestyle (Glazebrook, 2005; Spoel and Dong, 2008; Pieterse et al., 2012; Siciliano et al., 2015). In vegetative tissues, it is commonly postulated that effective responses to biotrophic pathogens are typically mediated by SA and programmed cell death, and responses to necrotrophic pathogens, which benefit from host cell death, involve JA and ET signaling (Glazebrook, 2005; Spoel and Dong, 2008). Examples of changes in gene expression or enzyme activities in response to application of natural and synthetic chemicals are reported in Tab. 3.

5.1.1. Salicylic acid and its analogs

SA participates in the mechanisms of defense, plant development, fruit ripening, and responses to various abiotic factors. SA application to an active necrotrophic infection with *Colletotrichum gloeosporioides* led to programmed cell death and increased susceptibility (Alkan et al., 2012). Similarly, infection with *C. gloeosporioides* on a ripe *NahG* tomato fruit mutant that lacked SA responses showed increased tolerance to *C. gloeosporioides* (Alkan and Fortes, 2015). Also, preharvest and postharvest treatments with SA for latent infection of *C. gloeosporioides* effectively reduced the occurrence of anthracnose of mango (Zainuri et al., 2001). Application of SA at 0.14 mg/mL by preharvest spraying or soaking before storage induced resistance to gray mold in kiwi fruit (Poole et al., 1998). SA at 0.05 mM in combination with ultrasound induced higher disease resistance to blue mold in peach fruit (Yang et al., 2011). Treatment with 0.5 mM SA reduced incidence and severity of decay caused by *P. expansum* on sweet cherries (Chan and Tian, 2006).

Treatment with SA and its analogs induces the accumulation of ROS, which can kill pathogen cells (Baker and Orlandi, 1995; Mittler et al., 2011). ROS levels are determined by the activities of enzymes, such as NADPH oxidase, that are designated as ‘respiratory burst oxidase homologs’, and antioxidants, such as SOD, ascorbate peroxidase (APX), glutathione reductase (GR), and CAT (Sharma et al., 2012). The induced host resistance was closely related to the levels of hydrogen peroxide and the activities of antioxidant enzymes (Liu et al., 2005; Ren et al., 2012; Dickman and Fluhr, 2013; Ge et al., 2015). On the other hand, activation of NADPH oxidase promotes the SA defense response (Alkan et al., 2012).

SA increased the activities of CHT, PAL, GLU and GR, and reduced the activities of CAT and APX in pears (Cao et al., 2006). A combination of SA and an antagonistic yeast significantly increased the activities of polyphenol oxidase (PPO), PAL, and GLU activities in cherries (Qin et al., 2003). Additionally, in cherry, increases in the activities of CAT, POD, APX, and SOD were observed after application of MeSA (Valverde et al., 2015).

Proteome research has shown that antioxidant and PR proteins, as well as enzymes associated with sugar metabolism, are involved in resistance of peach and sweet cherry fruit treated with SA (Chan et al., 2007). Therefore, the induced resistance by SA in fruit and vegetables activates a global defense response, which includes activation of the phenylpropanoid pathway, induction of accumulation of ROS and antioxidants, and production of PR proteins (Liu et al., 2014; Wang et al., 2015b).

5.1.2. Benzothiadiazole (BTH)

Benzothiadiazole (benzo(1,2,3)-thiadiazole-7-carbothioicacid S-methyl ester, also known as BTH or ASM) is perhaps the most potent synthetic elicitor discovered to date (Terry and Joyce, 2004; Bi et al., 2007). BTH is a light-insensitive functional analog of SA that induces resistance against a broad range of pathogens through activation of SAR in plants. Preharvest and postharvest BTH treatments have effectively reduced latent infections and induced resistance to diseases in fruit and vegetables, including strawberries (Terry and Joyce, 2004; Mazaro et al., 2008; Cao et al., 2011; Feliziani et al., 2015), pears (Cao et al., 2006), peaches (Liu et al., 2005), melons (Ren et al., 2012; Liu et al., 2014; Li et al., 2015b), and potatoes (Bokshi et al., 2003).

Preharvest BTH treatment significantly reduced *Alternaria* rot and blue mold of pears during storage (Cao et al., 2005). The same authors reported that the activities of PR proteins such as POD, CHT, and GLU were significantly enhanced in pears treated with BTH. Similar results were also observed in potatoes (Bokshi et al., 2003), peaches (Liu et al., 2005), and melons (Bi et al., 2006a).

Postharvest application of BTH to strawberries induced gene expression and increased activity of a range of enzymes that included several that are linked to biotic stress resistance (Landi et al., 2014). BTH-induced disease resistance enhanced gene expression of PPO and POD, and up-regulation of these genes was related to accumulation of total phenolic compounds, in harvested mango fruit (Lin et al., 2011). Postharvest dipping with BTH at 100 mg/L reduced artificial and natural infections in melons, while concentrations greater than 300 mg/L failed to promote resistance and caused phytotoxicity (Bi et al., 2006a).

5.1.3. 2,6-Dichloronicotinic acid (INA)

2,6-Dichloronicotinic acid (INA) is a synthetic compound that is a structural and functional analog of SA, and it has been reported to mediate resistance against a broad spectrum of pathogens and its induced resistance has been suggested to have long-lasting effects (Lucas, 1999).

Preharvest foliar spray of INA at 50 mg/L significantly reduced postharvest diseases of melons (Bokshi et al., 2006). The resistance against *C. gloeosporioides* in mango was noticeably enhanced by preharvest treatment with INA (Santiago et al., 2006). INA at 0.5 g/L also effectively reduced the disease spot sizes on the peel of banana fruit when inoculated with *C. musae* (Huang et al., 2011b).

5.1.4. Jasmonic acid (JA) and methyl jasmonate (MeJA)

Application of JA and methyl jasmonate (MeJA) can control decay incidence of several fruit. Postharvest JA treatment at 0.01 mM reduced green mold of grapefruit and orange (Porat et al., 2002). The optimal concentration of JA or MeJA varies for different fruit, and for different diseases on the same fruit. The effective concentration of MeJA to control tomato anthracnose was 0.0448 mM (Tzortzakis, 2007), while it was 10 mM for the control of tomato gray mold (Zhu and Tian, 2012). When applied by infiltration, the concentration of MeJA was reduced to 0.1 mM (Yu et al., 2009).

JA stimulated production of signaling molecules related to resistance and accumulation of antimicrobial compounds, and strengthened the structural barriers that restrict pathogen infection (Tian et al., 2007). MeJA treatment promoted higher PAL activity and increased total phenolics, flavonoids, and anthocyanins (Wang et al., 2009b). MeJA treatment promoted early accumulation of hydrogen peroxide, and increased gene expression of Cu-Zn SOD, CAT, and APX, at the same time. MeJA treatment also enhanced the contents of ascorbic acid and glutathione, which can scavenge excess ROS to alleviate protein oxidation injury (Zhu and Tian, 2012). MeJA treatment induced resistance against *Penicillium citrinum* by priming defense responses, and up-regulated the hydrogen peroxide burst and enhanced translation levels of defense-related proteins and the contents of antimicrobial compounds in Chinese bayberries (Wang et al., 2014).

5.1.5. Other chemical elicitors

Brassinosteroids (BRs) are a group of phyto-steroidal hormones that have crucial roles in a wide spectrum of biochemical, physiological, growth, and developmental processes in plants. A remarkable feature of BRs is their potential to increase resistance to a wide spectrum of stress in plants (Krishna, 2003). BRs at 5 μM effectively inhibited development of blue mold rot and enhanced the activities of defense-related enzymes in jujube fruit. BRs did not show in vitro antimicrobial activity against *P. expansum* (Zhu et al., 2010).

Although BABA is only rarely found naturally in plants, it has been shown to be a potent inducer of acquired resistance and has a broad spectrum of activity against many pathogens (Conrath et al., 2001). BABA induced resistance of mango fruit to postharvest anthracnose caused by *C. gloeosporioides*, and enhanced the activity of fruit defense mechanisms (Zhang et al., 2013). BABA caused short-lasting activation of CHT and POD after a first spray, and a boost after a second spray, although to a lower level than that caused by INA (Bokshi et al., 2006).

The application of oxalic acid has been shown to induce systemic resistance against postharvest diseases (Zheng et al., 2005). Oxalic acid induced defense-related enzymes and priming of defense responses, enhanced the contents of antimicrobial compounds and modulated antioxidant enzymes in muskmelon (Deng et al., 2015). Oxalic acid at 5 mM increased antioxidant levels and PPO activity, which was beneficial for delayed ripening and enhanced disease resistance in peach fruit during storage at low temperature (Zheng et al., 2005).

L-arginine induced disease resistance via its effects on nitric oxide (NO) biosynthesis and defensive enzyme activity in tomato fruit (Zheng et al., 2011).

Riboflavin at 1.0 mM inhibited development of *Alternaria* rot, enhanced the activities of defense-related enzymes, such as PAL, PPO and POD, and increased accumulation of flavonoids, phenolics, and lignin (Li et al., 2012b).

1-Methylcyclopropene induced resistance against postharvest decay in jujube fruit, and increased PAL, PPO, CAT, and SOD activities (Zhang et al., 2012).

5.2. Biological elicitors

5.2.1. Bacterial effectors

5.2.1.1. Harpin

Harpin is an acidic, heat-stable, glycine-rich, 44-kDa protein encoded by the *hrpN* gene, and it was first described in *Erwinia amylovora*, which causes fire blight of *Rosaceae*. Bacterial harpin has been shown to elicit the hypersensitive response and to induce SAR in plants (Baker and Orlandi, 1995). This elicitor has also been shown to induce resistance in some postharvest fruit and vegetables (Bi et al., 2007). Postharvest treatment with harpin at 0.04 mg/L to 0.16 mg/L inhibited incidence of blue mold in apple (De Capdeville et al., 2003). Field spraying with harpin at 50 mg/L reduced latent infections in muskmelons caused by *A. alternata* and *Fusarium* spp., with reductions proportional to the levels of applied harpin, up to 90 mg/L (Wang et al., 2011b). However, harpin higher than 90 mg/L failed to promote resistance in melons (Bi et al., 2005). Harpin reduced lesion diameter in both treated and untreated halves of the same melon, which indicated that SAR was induced by harpin (Bi et al., 2005). Some of the defense reactions in melons elicited by harpin were identified as alterations in the levels of preformed antifungal substances, such as phenols and flavonoids, accumulation of PR proteins, such as CHT and GLU, induction of enzyme activities, such as PAL and POD, modulation of metabolism of ROS, such as SOD, CAT, and hydrogen peroxide, and reinforcement of cell walls and lignin (Bi et al., 2005; Wang et al., 2011b; Zhu and Zhang, 2016).

5.2.1.2. Oligandrin

Oligandrin is an elicitin-like protein with molecular mass of >10 kDa that has been shown to be secreted by *Pythium oligandrum*. Oligandrin is known to induce resistance against a number of plant diseases. The treatment of tomato fruit with oligandrin at 10 μg/mL significantly reduced incidence and severity of gray mold, increased the activities of the defense-related enzymes, like PAL, PPO, and POD, and the mRNA levels of the genes encoding PR proteins, and activated the ET-dependent signaling pathway (Wang et al., 2011a).

5.2.2. Fungal effectors

5.2.2.1. Chitosan

Chitosan (poly-β-(1,4)-N-acetyl-d-glucosamine) is a natural biopolymer with wide antimicrobial properties that can elicit defense responses in fruit and vegetables (El Ghaouth et al., 1992; Romanazzi et al., 2002). This chemical and its derivatives, such as oligochitosan and glycol chitosan, can be used in solution, as powders, and as edible coatings (Romanazzi et al., 2016b). Preharvest and postharvest treatments with chitosan and its derivatives tend to suppress storage rots in many commodities, such as strawberry (El Ghaouth et al., 1992; Reddy et al., 2000), jujube (Yan et al., 2012), sweet cherries (Feliziani et al., 2013a), citrus fruit (Fajardo et al., 1998; Zeng et al., 2010), apples (Felipini and Di Piero, 2009), banana (Meng et al., 2012), table grapes (Romanazzi et al., 2002; Meng et al., 2008), and tomatoes (Liu et al., 2007; Badawy and Rabea, 2009), and for many other vegetables (Miranda-Castro, 2016).

Several studies have shown that chitosan has multiple mechanisms of action, with direct antimicrobial properties, film-forming activities, and induction of host defenses (Romanazzi et al., 2016b). There was a significant increase in CHT and GLU activities in banana and jujube treated with oligochitosan (Meng et al., 2012; Yan et al., 2012). Chitosan and oligochitosan treatments induced significant increases in the activities of PPO (Liu et al., 2007), POD (Liu et al., 2007; Yan et al., 2012), and PAL (Romanazzi et al., 2002; Meng et al., 2012; Landi et al., 2014) in several harvested products. Chitosan treatment enhanced the total content of phenolics, flavonoids, and other antifungal substances (El Ghaouth et al., 1992; Yan et al., 2012), and accumulated ROS through regulation of the activity of metabolic enzymes, such as SOD, CAT, and APX (Zeng et al., 2010; Yan et al., 2012; Landi et al., 2014). Spraying with chitosan increased the activities of PPO and PAL in table grapes, thus promoting protection from latent infection of *B. cinerea* (Romanazzi et al., 2002; Meng et al., 2008). Next-generation sequencing with chitosan-treated avocado has defined more genes as up-regulated than down-regulated (Gutiérrez-Martínez et al., 2016). CHT and ROS production in table grape berries varied according to the formulation of chitosan applied (Feliziani et al., 2013b). Structural defense responses, such as preservation of pectin binding sites and the intense and regular cellulose distribution over host cell walls were observed in the first tissue layers beneath the ruptured cells in bell peppers treated with chitosan (El Ghaouth et al., 1997).

5.2.2.2. Burdock fructooligosaccharide

Burdock fructooligosaccharide (BFO) is a fructosan oligomer that effectively inhibited postharvest diseases and reduced incidence of gray mold in tomato. BFO increased mRNA levels of genes encoding PR proteins, such as PR-1a, PR-2a (extracellular GLU), PR-2b (intracellular GLU), PR-3a (extracellular CHT), and PR-3b (intracellular CHT), and induced accumulation of PAL mRNA in tomatoes (Wang et al., 2009a). BFO also effectively controlled postharvest diseases in grapes, apples, banana, kiwi fruit, citrus fruit, strawberries, and pears (Sun et al., 2013).

5.2.3. Other biological elicitors

Peach fruit pretreated with yeast saccharide activated CHT, GLU, PAL, and POD. Moreover, yeast saccharide triggered endogenous NO in peaches during storage (Yu et al., 2012). The flavonoid quercetin significantly reduced blue mold in apples, and genes differentially expressed in quercetin-treated apples revealed high similarities with different classes of PR proteins (i.e., RNase-like PR10, PR8), and with proteins expressed under stress conditions (Sanzani et al., 2009b; Sanzani et al., 2010). The resistance inducer protein hydrolysates were effective against green mold of citrus fruit and gray mold of table and wine grapes (Lachhab et al., 2015, 2016).

5.3. Inorganic elicitors

5.3.1. Silicon (Si)

Silicon (Si) is the second most abundant element in the Earth lithosphere, and it is as important as phosphorus and magnesium in the biota (Exley, 1998). Si is also considered to be biologically active and to trigger more rapid and extensive deployment of plant natural defenses. Guo et al. (2007) reported that Si oxide and sodium silicate suppressed pink rot in muskmelons. Sodium silicate at 100 mM reduced rots in melons caused by *A. alternata*, *F. semitectum*, and *T. roseum* (Bi et al., 2006b; Li et al., 2012a).

Sodium silicate has been shown to be effective for suppression of pathogen growth and for induction of resistance to postharvest diseases in fruit and vegetables. Si treatment did not affect the activities of POD, PPO, PAL, and GLU, or the content of total phenols and flavonoids in potato tuber, although these were significantly accumulated after a challenge with *F. sulphureum* (Li et al., 2009). These results indicated that a priming state was induced by Si in potato tubers (Conrath et al., 2001). In Hami melons, Si treatments caused activation of POD and CHT (Bi et al., 2006b). The effects of

Si on postharvest diseases of muskmelons were associated with elicitation of the antioxidant system (Li et al., 2012a).

5.3.2. Nitric oxide (NO)

Nitric oxide (NO) is an important bioactive molecule that serves as a signal in plants, in particular for maturation and senescence (Leshem et al., 1998). Lai et al. (2011) reported that NO treatment activated antioxidant enzymes and induced resistance against gray mold in tomato. NO increased accumulation of phytoalexin rishitin in potato tubers (Niritake et al., 1996), and induced resistance against dry rot in potato tubers (Hu et al., 2014a). Moreover, NO treatment modulated anthracnose levels and induced defense-related enzymes in mangoes (Hu et al., 2014b).

5.3.3. Sodium carbonate

Sodium carbonate and bicarbonate were effective in the control of green mold of citrus fruit (Youssef et al., 2014). These salts exerted direct antifungal effects on *Penicillium digitatum*, and increased the activities of resistance enzymes, such as GLU, POD, and PAL. Moreover, citrus peel extracts showed increased levels of sugars and phytoalexins, with sucrose and scoparone being the most represented.

6. Induced resistance by disinfecting agents

6.1. Ozone

Since its recognition in 1997 by the US Food and Drug Administration as a safe food disinfectant, both gaseous ozone (O_3) and ozonated water have gained particular attention for the control of postharvest diseases of fruit and vegetables. The most recent examples of its use are those reported for blueberries (Crowe et al., 2012), persimmons (Ikeura et al., 2013), papaya (Ali et al., 2014), and table grapes (Feliziani et al., 2014). Ozone is a strong oxidizing agent; however, as the O_3 disinfecting activity is limited to surface-contaminating microflora, and as various microbes show different susceptibilities (Pascual et al., 2007), further modes of action appear to be involved (Feliziani et al., 2016). Artes-Hernandez et al. (2007) reported that continuous flow of O_3 increased total flavonol and hydroxycinnamates contents in cold-stored ‘Autumn Seedless’ table grapes. Moreover, it has been reported that O_3 boosts maintained the total polyphenols, and greatly increased the phytoalexin resveratrol content in cold-stored ‘Napoleon’ table grapes (Artés-Hernández et al., 2003). Similarly, O_3 at 0.1 μmol/mol increased total phenolic content in red bell peppers (Glowacz et al., 2015). A proteomic analysis conducted on O_3-treated kiwi fruit identified 102 differentially expressed proteins that were mainly involved in energy, protein metabolism, defense, and cell structure (Minas et al., 2012). A set of candidate kiwifruit proteins was defined as sensitive to protein carbonylation, which was induced by ripening and depressed by O_3. Moreover, O_3 treatment transiently increased expression of CHT and PAL in ‘Redglobe’ and ‘Sugraone’ table grapes (Duarte-Sierra et al., 2016) (Tab. 4).

6.2. Electrolyzed water

Electrolyzed water (EW) is obtained by adding a small amount of an electrolyte to tap water, which is traditionally sodium chloride, and passing an electrical current across an anode and a cathode. EW has high oxidation–reduction potential with strong direct effects against decay causing fungi (Guentzel et al., 2010). However, recently, it has shown resistance inducer potential. In particular, it caused 30% increase in the total phenolic content of broccoli (Navarro-Rico et al., 2014) and maintained fresh-cut cilantro firmness (Hao et al., 2015), thus improving their resistance to pathogen attack. More recently, other electrolytes have been tested to improve EW performance and to avoid production of chlorine by-products. Encouraging results were obtained using $NaHCO_3$ against postharvest rots of citrus fruit, which also induced host defense responses (Fallanaj et al., 2016). In particular, up-regulation of defense-related genes that encode CHT, POD, and PAL was observed at 6 h to 12 h post-treatment, with increased activity of the related enzymes and of GLU (Tab. 4). As this was observed at 12 h post-treatment, this suggested an early host response against *P. digitatum* by limiting tissue colonization.

6.3. Ethanol

The effects of ethanol dipping as a surface disinfectant that can reduce pathogen populations without impairing product quality are well known (Lichter et al., 2002; Mlikota Gabler et al., 2004; Lee et al., 2015). However, its mode of action appears to be much more complex. Ethanol induction of resistance to postharvest anthracnose in loquat fruit was demonstrated by Wang et al. (2015a). Ethanol at 300 μL/L inhibited anthracnose caused by *Colletotrichum acutatum*, and maintained overall quality. Moreover, it increased SOD activity, thus resulting in higher levels of hydrogen peroxide, which can activate disease resistance. Meanwhile, ethanol treatment significantly enhanced the activities of defense-related enzymes, including PAL, POD, PPO, CHT, and GLU (Tab. 4). Recently, ethanol treatments were shown to decelerate the ripening process and down-regulate expression of major lipoxygenase-encoding genes involved in melon fruit ripening, thus contributing to its increased resistance to biotic and abiotic stress (Zhang et al., 2015).

7. Induced resistance by microbial and plant volatile organic compounds (VOCs)

Biotic agents that contribute to plant disease management include PGPR, growth promoting fungi, and fungi that work as biocontrol agents (Lyon, 2007). In addition to the use of microorganisms and chemical compounds in plant protection, there is an important developing area of research in the control of postharvest pathogens in fruit and vegetables based on the use of volatile organic compounds (VOCs), which can be produced by microorganisms (MVOCs) or by plants (PVOCs) (Mari et al., 2016).

7.1. Induced resistance by microbial volatile organic compounds (MVOCs)

Microbial volatile organic compounds (MVOCs) are mixtures of carbon-based compounds that are highly volatile or are vapors (Morath et al., 2012). Fungi, yeasts, and bacteria produce MVOCs as primary and secondary metabolites (Jijakli and Lepoivre, 1998; Korpi et al., 2009). MVOCs have shown applicative potential as biofumigants and have motivated great interest, mainly because they occur naturally without chemical synthesis (Li et al., 2015a). MVOCs can be signaling substances for regulation and control of some physiological actions, which include induction of systemic resistance against pathogens (Ryu et al., 2004).

Some defense mechanisms activated by biocontrol bacteria are induced in fruit and leaves by MVOCs, including production of phytoalexins, PR proteins, such as CHT and GLU, and protein inhibitors (Conrath et al., 2006; Li et al., 2012c). *Bacillus* spp. VOCs (2-3-butanediol and lipopetides) induced over-expression of the surfactin and fengicin genes, which caused metabolic changes in host tissues (Ryu et al., 2004; Ongena et al., 2007).

7.2. Induced resistance by plant volatile organic compounds (PVOCs)

The eliciting activities of PVOCs, such as MeSA, were reported in section 5.1.1. Citrus fruit produce VOCs that are actively involved in defense systems before pathogen attacks. The presence and variation of VOCs depend on the type of produce and of its development phase. VOCs occur naturally in plant systems and can be associated with the biochemistry of constitutive defense mechanisms (Wightwick et al., 2010). Structures in the petals of citrus flowers, called osmophores, can release more than 60 VOCs, such as phenols, terpenes, and lipophilic compounds, which have been recognized as antifungal agents (Caccioni et al., 1995; Lattanzio et al., 2006). It was reported that limonene and linalool have antifungal actions against *C. acutatum* and are associated with constitutive biochemical responses and can be used in the control of pathogens (Rodrigues Marques et al., 2015).

Essential oils produce their activity through vapor, and thyme and cinnamon essential oils increased PPO, PAL, CHT, GLU activities in peach fruit (Cindi et al., 2016). In tomato, it has been reported that VOCs are induced and modified in defense responses, and ripening processes, and by wounding. In a microarray analysis of tomato fruit at different stages of ripening, more than 4,000 differentially expressed genes have been reported. Genes related to defense mechanisms were expressed in the early stages of ripening, and genes related to VOCs changed during late maturation. Overall, more than 40 VOCs were detected, and their profiles were characterized along the fruit ripening stages (Baldassarre et al., 2015).

8. Induced resistance perspectives

A large amount of data has been generated during the last few years that are related to the triggering of host defenses during postharvest (see as examples, Tables 1, 2, 3 and 4, and Fig. 1). The elicitation of host defenses has a central role nowadays in integrated disease management strategies, and this is welcome for the reasons summarized in Tab. 5. The effects on plants can last for weeks or months. Induced resistance has a wider spectrum of targets than synthetic fungicides. Induced resistance does not lead to the appearance of resistant strains, due to involvement of various modes of action. Induced resistance is usually applied before the appearance of the symptoms, so there are no side effects on nontarget organisms and on humans, in terms of farmers, people working in the packinghouses, retailers, or consumers. Moreover, induced resistance can lead to increased levels of phenolic compounds in the plant tissues, which often have antioxidant properties that are highly beneficial to humans. However, we cannot forget the weaker points linked to the application of strategies based on induced resistance, such as possible inconsistent results or difficulties in their implementation in packinghouse practices. Recently, a high number of biostimulants have appeared on the market, and are considered as part of the effective treatments. Usually these biostimulants have combinations of direct activities on pathogens and indirect activities on the host. Increasing interest in this novel approach that is based on the triggering of host defenses satisfies consumer demands and the guidelines on sustainable approaches to plant protection, which in European Union is implemented through Directive 128/2009 on sustainable use of fungicides. New tools, such as the -omics sciences, allow better understanding of the changes in host physiology and provide information on gene functions. This information will provide optimization of the application of alternative treatments to control postharvest decay.

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References

Ali, A., Ong, M.K., Forney, C.F., 2014. Effect of ozone pre-conditioning on quality and antioxidant capacity of papaya fruit during ambient storage. Food Chem. 142, 19-26.

Alkan, N., Fluhr, R., Prusky, D., 2012. Ammonium secretion during *Colletotrichum coccodes* infection modulates salicylic and jasmonic acid pathways of ripe and unripe tomato fruit. Mol. Plant-Microbe Interact. 25, 85-96.

Alkan, N., Fortes, A.M., 2015. Insights into molecular and metabolic events associated with fruit response to postharvest fungal pathogens. Front. Plant Sci. 6.

Alvarez, M.E., Pennell, R.I., Meijer, P.J., Ishikawa, A., Dixon, R.A., Lamb, C., 1998. Reactive oxygen intermediates mediate a systemic signal network in the establishment of plant immunity. Cell 92, 773-784.

Artés-Hernández, F., Artes, F., Tomas-Barberan, F.A., 2003. Quality and enhancement of bioactive phenolics in cv. Napoleon table grapes exposed to different postharvest gaseous treatments. J. Agric. Food Chem. 51, 5290-5295.

Artés-Hernández, F., Aguayo, E., Artes, F., Tomas-Barberan, F.A., 2007. Enriched ozone atmosphere enhances bioactive phenolics in seedless table grapes after prolonged shelf life. J. Sci. Food Agric. 87, 824-831.

Badawy, M.E.I., Rabea, E.I., 2009. Potential of the biopolymer chitosan with different molecular weights to control postharvest gray mold of tomato fruit. Postharvest Biol. Technol. 51, 110–117.

Baker, C.J., Orlandi, E.W., 1995. Active oxygen in plant pathogenesis. Annu. Rev. Phytopathol. 33, 299–321.

Baldassarre, V., Cabassi, G., Spadafora, N., Aprile, A., Müller, C., Rogers, H., Ferrante, A., 2015. Wounding tomato fruit elicits ripening-stage specific changes in gene expression and production of volatile compounds. J. Exp. Bot. 66, 1511–1526.

Bent, A.F., Mackey, D., 2007. Elicitors, effectors, and R genes: The new paradigm and a lifetime supply of questions. Annu. Rev. Phytopathol. 45, 399-436.

Bi, Y., Tian, S.P., Zhao, J., Ge, Y.H., 2005. Harpin induces local and systemic resistance against *Trichothecium roseum* in harvested Hami melons. Postharvest Biol. Technol. 38, 183–187.

Bi, Y., Ge, Y.H., Li, Y.C., Wang, J.J., Miao, X.Y., Li, X.W., 2006a. Postharvest acibenzolar-S-methyl treatment suppresses decay and induces resistance in Hami melons. Acta Hortic. 712, 393–399.

Bi, Y., Tian, S.P., Guo, Y.R., Ge, Y.H., Qin, G.Z., 2006b. Sodium silicate reduces postharvest decay on Hami melons: induced resistance and fungistatic effects. Plant Dis. 90, 279–283.

Bi, Y., Li, Y., Ge, Y., 2007. Induced resistance in postharvest fruits and vegetables by chemicals and its mechanism. Stewart Postharvest Rev. 3, 1-7.

Bokshi, A.I., Morris, S.C., Deverall, B.J., 2003. Effects of benzothiadiazole and acetylsalicylic acid on β-1,3-glucanase activity and disease resistance in potato. Plant Pathol. 52, 22–27.

Bokshi, A.I., Morris, S.C., McConchie, R.M., Deverall, B.J., 2006. Pre-harvest application of 2,6-dichloroisonicotinic acid, beta-aminobutyric acid or benzothiadiazole to control post-harvest storage diseases of melons by inducing systemic acquired resistance (SAR). J. Hortic. Sci. Biotechnol. 81, 700–706.

Buonaurio, R., Iriti, M., Romanazzi, G., 2009. Induced resistance to plant diseases caused by Oomycetes and fungi. Petria 19(3), 130-148.

Burg, S.P., Burg, E.A., 1966. Fruit storage at subatmospheric pressures. Science 153, 314-315.

Caccioni, D., Deans, L., Ruberto, G., 1995. Inhibitory effect of citrus oil components on *Penicillium italicum* and *P. digitatum*. Petria 5, 177-182.

Cantos, E., Garcia-Viguera, C., de Pascual-Teresa, S., Tomas-Barberan, F.A., 2000. Effect of postharvest irradiation on resveratrol and other phenolics of cv. Napoleon table grapes. J. Agric. Food Chem. 48, 4606-4612.

Cao, J.K., Jiang, W.B., He, H., 2005. Induced resistance in Yali pear (*Pyrus bretschneideri* Rehd.) fruit against infection by *Penicillium expansum* by postharvest infiltration of acibenzolar-S-methyl. J. Phytopathol. 153, 640-646.

Cao, J.K., Zeng K.F., Jiang, W.B., 2006. Enhancement of postharvest disease resistance in Yali pear (*Pyrus bretschneideri*) fruit by salicylic acid sprays on the trees during fruit growth. Eur. J. Plant Pathol. 114, 363-370.

Cao, S.F., Hu, Z., Zheng, Y., Yang, Z., Lu, B., 2011. Effect of BTH on antioxidant enzymes, radical-scavenging activity and decay in strawberry fruit. Food Chem. 125, 145–149.

Chan, Z.L., Tian, S.P. 2005. Interaction of antagonistic yeasts against postharvest pathogens of apple fruit and possible mode of action. Postharvest Biol. Technol. 36, 215-223.

Chan, Z.L., Tian, S.P. 2006. Induction of H_2O_2-metabolizing enzymes and total protein synthesis by antagonistic yeast and salicylic acid in harvested sweet cherry fruit. Postharvest Biol. Technol. 39, 314-320.

Chan, Z.L., Qin, G.Z., Xu, X.B., Li, B.Q., Tian, S.P. 2007. Proteome approach to characterize proteins induced by antagonist yeast and salicylic acid in peach fruit. J. Proteome Res. 6, 1677-1688.

Charles, M.T., Arul, J., 2007. UV treatment of fresh fruits and vegetables for improved quality: a status report. Stewart Postharvest Rev. 3, 6.

Chaturvedi, R., Venables, B., Petros, R.A., Nalam, V., Li, M.Y., Wang, X.M., Takemoto, L.J., Shah, J., 2012. An abietane diterpenoid is a potent activator of systemic acquired resistance. Plant J. 71, 161-172.

Cindi M.D., Sivakumar D., Romanazzi G., Soundy P., 2016. Differential defense responses and brown rot control after essential oil fumigation in two *Prunus persica* cultivars during storage. Postharvest Biol. Technol. 119, 9-17.

Conrath, U., Thulke, O., Katz, V., Schwindling, S., Kohler, A., 2001. Priming as a mechanism in induced systemic resistance of plants. Eur. J. Plant Pathol. 107, 113–119.

Conrath, U., Pieterse, C., Mauch-Mani, B., 2002. Priming in plant–pathogen interactions. Trends Plant Sci. 7, 210–216.

Conrath, U., Beckers, G.J.M., Flors, V., Garcıa-Agustın, P., Jakab, G., Mauch, F., Newman, M.A., Pieterse, C.M.J., Poinssot, B., Pozo, M.J., Pugin, A., Schaffrath, U., Ton, J., Wendehenne, D., Zimmerli, L., Mauch-Mani, B., 2006. Priming: getting ready for battle. Mol. Plant-Microbe Interact. 19:1062–1071.

Crowe, K.M., Bushway, A., Davis-Dentici, K., 2012. Impact of postharvest treatments, chlorine and ozone, coupled with low-temperature frozen storage on the antimicrobial quality of lowbush blueberries (*Vaccinium angustifolium*). LWT - Food Sci. Technol. 47, 213-215.

Da Rocha, A., Hammerschmidt, R., 2005. History and perspectives on the use of disease resistance inducers in horticultural crops. HortTechnology 15: 518–529.

De Capdeville, G.., Beer, S.V., Watkins, C.B., Wilson, C.L., Tedeschi, L.O., Aist, J.R., 2003. Pre- and postharvest harpin treatments of apples induced resistance to blue mold. Plant Dis. 89, 39–44.

Deng, J., Bi, Y., Zhang, Z., Xie, D., Ge, Y., Li, W., Wang, Y., 2015. Postharvest oxalic acid treatment induces resistance against pink rot by priming in muskmelon (*Cucumis melo* L.) fruit. Postharvest Biol. Technol. 106, 53-61.

Dickman, M.B., Fluhr, R., 2013. Centrality of host cell death in plant-microbe interactions. Annu. Rev. Phytopathol. 51, 543-570.

Droby, S., Wisniewski, M., Teixidó, N., Spadaro, D., Jijakli, H.M., 2016. The science, development, and commercialization of postharvest biocontrol products. Postharvest Biol. Technol. (this issue).

Duarte-Sierra, A., Aispuro-Hernández, E., Vargas-Arispuro, I., Islas-Osuna, M.A., González-Aguilar, G.A., Martínez-Téllez, M. Á., 2016. Quality and PR gene expression of table grapes treated with ozone and sulfur dioxide to control fungal decay. J. Sci. Food Agric. 96, 2018-2014.

Durrant, W., Dong, X., 2004. Systemic acquired resistance. Annu. Rev. Phytopathol. 42, 185–209.

El Ghaouth, A., Arul, J., Grenier, J., Asselin, A., 1992. Antifungal activity of chitosan on two postharvest pathogens of strawberry fruits. Phytopathology 82, 398-402.

El Ghaouth, A., Arul, J., Wilson, C., Benhamou, N., 1997. Biochemical and cytochemical aspects of the interactions of chitosan and *Botrytis cinerea* in bell pepper fruit. Postharvest Biol. Technol. 12, 183–194.

El Ghaouth, A., Wilson, C.L., Callahan, A.M., 2003a. Induction of chitinase, β-1,3 glucanase, and phenylalanine ammonia-lyase in peach fruit by UV-C treatment. Phytopathology 93, 349-355.

El Ghaouth, A., Wilson, C.L., Wisniewski, M., 2003b. Control of postharvest decay of apple fruit with *Candida saitoana* and induction of defense responses. Phytopathology 93, 344-348.

Exley, C., 1998. Silicon in life: a bioinorganic solution to bioorganic essentiality. J. Inorg. Biochem. 69, 139-144.

Fajardo, J.E., McCollum, T.G., McDonald, R.E., Mayer, R.T., 1998. Differential induction of proteins in orange flavedo by biologically based elicitors and challenged by *Penicillium digitatum* Sacc. Biol Control 13, 143-151.

Fallanaj, F., Ippolito, A., Ligorio, A., Garganese, F., Zavanella, C., Sanzani, S.M., 2016. Electrolyzed sodium bicarbonate inhibits *Penicillium digitatum* and induces defence responses against green mould in citrus fruit. Postharvest Biol. Technol. 115, 18-29.

Fallik, E., 2004. Prestorage hot water treatments (immersion, rinsing and brushing). Postharvest Biol. Technol. 32, 125-134.

FAO, 2011. Global food losses and food waste – extent, causes and prevention. In: Gustavsson, J., Cederberg, C., Sonesson, U., van Otterdijk, R., Meybeck, A. Rome (http://www.fao.org/docrep/014/mb060e/mb060e00.pdf).

Felipini, R.B., Di Piero, R. M., 2009. Reduction of the severity of apple bitter rot by fruit immersion in chitosan. Pes. Agropec. Bras. 44, 1591-1597.

Feliziani, E., Santini, M., Landi, L., Romanazzi, G., 2013a. Pre- and postharvest treatment with alternatives to synthetic fungicides to control postharvest decay of sweet cherry. Postharvest Biol. Technol. 78, 133-138.

Feliziani, E., Smilanick, J.L., Margosan, D.A., Mansour, M.F., Romanazzi, G., Gu, H., Gohil, H.L., Rubio Ames, Z., 2013b. Preharvest fungicide, potassium sorbate, or chitosan use on quality and storage decay of table grapes. Plant Dis. 97, 307-314.

Feliziani, E., Romanazzi, G., Smilanick, J.L., 2014. Application of low concentrations of ozone during the cold storage of table grapes. Postharvest Biol. Technol. 93, 38-48.

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, 111-117.

Feliziani, E., Lichter, A., Smilanick, J.L., Ippolito, A., 2016. Disinfecting agents for controlling fruit and vegetable diseases after harvest. Postharvest Biol. Technol. (this issue).

Fu, Z.Q., Dong, X., 2013. Systemic Acquired Resistance: turning local infection into global defense. Annu. Rev. Plant Biol. 64, 839-863.

Fujita, M., Fujita, Y., Noutoshi, Y., Takahashi, F., Narusaka, Y., Yamaguchi-Shinozaki, K., Shinozaki, K., 2006. Crosstalk between abiotic and biotic stress responses: a current view from the points of convergence in the stress signaling networks. Curr. Opin. Plant Biol. 9, 436-442.

Gapper, N.E., Giovannoni, J.J., Watkins, C.B., 2014. Understanding development and ripening of fruit crops in an ‘omics’ era. Hortic. Res. 1, 14034; doi:10.1038/hortres.2014.34.

Ge, Y., Deng, H., Bi, Y., Li, C., Liu, Y., Dong, B., 2015. Postharvest ASM dipping and DPI pre-treatment regulated reactive oxygen species metabolism in muskmelon (*Cucumis melo* L.) fruit. Postharvest Biol. Technol. 99, 160-167.

Glazebrook, J., 2005. Contrasting mechanisms of defense against biotrophic and necrotrophic pathogens. Annu. Rev. Phytopathol. 43, 205-227.

Glowacz, M., Colgan, R., Rees, D., 2015. Influence of continuous exposure to gaseous ozone on the quality of red bell peppers, cucumbers and zucchini. Postharvest Biol. Technol. 99, 1-8.

Guentzel, J.L., Lam, K.L., Callan, M.A., Emmons, S.A., Dunham, V.L., 2010. Postharvest management of gray mold and brown rot on surfaces of peaches and grapes using electrolyzed oxidizing water. Int. J. Food Microbiol. 143, 54-60.

Guo, Y.R., Liu, L., Zhao, J., Bi, Y., 2007. Use of silicon oxide and sodium silicate for controlling *Trichothecium roseum* postharvest rot in Chinese cantaloupe (*Cucumis melo* L.). Int. J. Food Sci. Technol. 42, 1012–1018.

Gutiérrez-Martínez, P., Chacón-López, A., Xoca-Orozco, L.A., Ramos-Guerrero, A., Velázquez-Estrada, R., Aguilera-Aguirre, S., 2016. Chitosan and changes in gene expression during fruit–pathogen interaction at postharvest stage. In: Bautista-Baños, S., Romanazzi, G., Jiménez-Aparicio, A. (Eds.), Chitosan in the preservation of agricultural commodities, Academic Press, Elsevier, London, UK, pp. 299-311.

Hao, J., Li, H., Wan, Y. Liu, H., 2015. Effect of slightly acidic electrolyzed water (SAEW) treatment on the microbial reduction and storage quality of fresh-cut cilantro. J. Food Proc. Preserv. 39, 559-566.

Hashmi, M.S., East, A.R., Palmer, J.S., Heyes, J.A., 2013. Hypobaric treatment stimulates defence-related enzymes in strawberry. Postharvest Biol. Technol. 85, 77-82.

Hershkovitz, V., Sela, N., Taha-Salaime, L., Liu, J., Rafael, G., Kessler, C., Aly, R., Wisniewski, M., Droby, S., 2013. De-novo assembly and characterization of the transcriptome of *Metschnikowia fructicola* reveals differences in gene expression following interaction with *Penicillium digitatum* and grapefruit peel. BMC Genomics 14, 168.

Hu, L., Li, Y., Bi, Y., Li, J., Bao, G., Liu, J., Yu, X., 2014a. Effects of nitric oxide on growth of *Fusarium sulphureum* and its virulence to potato tubers. Eur. Food Res. Technol. 238, 1007-1014.

Hu, M., Yang, D., Huber, D. J., Jiang, Y., Li, M., Gao, Z., Zhang, Z., 2014b. Reduction of postharvest anthracnose and enhancement of disease resistance in ripening mango fruit by nitric oxide treatment. Postharvest Biol. Technol. 97, 115-122.

Huang, R., Li, G.Q., Zhang, J., Yang, L., Che, H.J., Jiang, D.H., Huang, H.C., 2011a. Control of postharvest Botrytis fruit rot of strawberry by volatile organic compounds of *Candida intermedia*. Phytopathology 101, 859-869.

Huang, X.M., Zhang, C., Pang X.Q., Zhang Z.Q., 2011b. Early changes of reactive oxygen species in 2,6-dichloroisonicotianic acid inducing tolerance in postharvest banana fruits. Acta Hortic. Sin.38, 265–272.

Ikeura, H., Hamasaki, S., Tamaki, M., 2013. Effects of ozone microbubble treatment on removal of residual pesticides and quality of persimmon leaves. Food Chem. 138, 366-371.

Ippolito, A., El Ghaouth, A.E., Wilson, C.L., Wisniewski, M., 2000. Control of postharvest decay of apple fruit by *Aureobasidium pullulans* and induction of defense responses. Postharvest Biol. Technol. 19, 265-272.

Janisiewicz, W., Bastos Periera, I., Almeida, M.S., Roberts, D.P., Wisniewski, M., Kurtenbach, E., 2008. Improved biocontrol of fruit decay fungi with *Pichia pastoris* recombinant strains expressing Psd1 antifungal peptide. Postharvest Biol. Technol. 47, 218-225.

Jijakli, M.H., Lepoivre, P., 1998. Characterization of an exo-β-1,3-glucanase produced by *Pichia anomala* strain K, antagonist of *Botrytis cinerea* on apples. Phytopathology 88, 335-343.

Jung, H.W., Tschaplinski, T.J., Wang, L., Glazebrook, J., Greenberg, J.T., 2009. Priming in systemic plant immunity. Science 324, 89-91.

Korpi A., Järnberg J., Pasanen A., 2009. Microbial volatile organic compounds. Crit. Rev. Toxicol. 39, 139–193.

Krishna, P., 2003. Brassinosteroid-mediated stress responses. J. Plant Growth Reg. 22, 289-297.

Lachhab, N., Sanzani, S.M., Fallanaj, F., Youssef, K., Nigro, F., Boselli, M., Ippolito, A., 2015. Protein hydrolysates as resistance inducers for controlling green mould of citrus fruit. Acta Hortic. 1065, 1593-1598.

Lachhab, N., Sanzani, S.M., Bahouaoui, M.A., Boselli, M., Ippolito, A., 2016. Effect of some protein hydrolysates against gray mould of table and wine grapes. Eur. J. Plant Pathol. 144, 821-830.

Lai, T.F., Wang, Y.Y., Li, B.Q., Qin, G.Z., Tian, S.P., 2011. Defensive responses of tomato fruit to exogenous nitric oxide during postharvest storage. Postharvest Biol. Technol. 62, 127–132.

Landi, L., Feliziani, E., Romanazzi, G., 2014. Expression of defense genes in strawberry fruit treated with different resistance inducers. J. Agric. Food Chem. 62, 3047-3056.

Lattanzio, V., Cardinali, A., Lattanzio, V., 2006. Role of phenolics in the resistance mechanisms of plants against fungal pathogens and insects. In: Imperato, F. (Ed), Phytochemistry: advances in research. Research Signpost, Kerala, India, pp. 23-67.

Lee, J.-S., Kaplunov, T., Zutahy, Y., Daus, A., Alkan, N., Lichter, A., 2015. The significance of postharvest disinfection for prevention of internal decay of table grapes after storage. Scientia Hortic. 192, 346-349.

Leshem, Y.A.Y., Wills, R.B.H., Ku, V.V.V., 1998. Evidence for the function of the free radical gas - nitric oxide (NO•) -as an endogenous maturation and senescence regulating factor in higher plants. Plant Physiol. Biochem. 36, 825–833.

Li, Y.C., Bi, Y., Ge, Y.H., Sun, X.J., Wang, Y., 2009. Antifungal activity of sodium silicate on *Fusarium sulphureum* and its effect on dry rot of potato tubers. J. Food Sci. 74, 213–218.

Li, W.H., Bi, Y., Ge, Y.H., Li, Y.C., Wang, J.J. Wang, Y., 2012a. Effects of postharvest sodium silicate treatment on pink rot disease and oxidative stress-antioxidative system in muskmelon fruit. Eur. Food Res. Technol. 234, 137–145.

Li, Y.C., Yin, Y., Bi, Y., 2012b. Effect of riboflavin on postharvest disease of Asia pear and the possible mechanisms involved. Phytoparasitica 40, 261-268.

Li, Q.L., Ning, P., Zheng, L., Huang, J.B., Li, G.Q., Hsiang, T., 2012c. Effects of volatile substances of *Streptomyces globisporus* JK-1 on control of *Botrytis cinerea* on tomato fruit. Biol. Control 61, 113-120.

Li, Q., Wu L., Hao J., Luo L., Cao Y., Li J., 2015a. Biofumigation on post-harvest diseases of fruits using a new volatile-producing fungus of *Ceratocystis fimbriata*. PLoS ONE 10(7).

Li, X., Bi, Y., Wang, J.J., Dong, B., Li, H., Gong, D., Zhao, Y., Tang, Y., Yu, X., Shang, Q., 2015b. BTH treatment caused physiological, biochemical and proteomic changes of muskmelon (*Cucumis melo* L.) fruit during ripening. J. Prot. 120, 179–193.

Lichter, A., Zutkhy, Y., Sonego, L., Dvir, O., Kaplunov, T., Sarig, P., Ben-Arie, R., 2002. Ethanol controls postharvest decay of table grapes. Postharvest Biol. Technol. 24, 301–308.

Lin, J.H., Gong, D.Q., Zhu, S.J., Zhang, L.J., Zhang, L.B., 2011. Expression of PPO and POD genes and contents of polyphenolic compounds in harvested mango fruits in relation to benzothiadiazole-induced defense against anthracnose. Scientia Hortic. 130, 85–89.

Liplap, P., Charlebois, D., Charles, M.T., Toivonen, P., Vigneault, C., Raghavan, G.S.V., 2013. Tomato shelf-life extension at room temperature by hyperbaric pressure treatment. Postharvest Biol. Technol. 86, 45-52.

Liu, H.X., Jiang, W.B., Bi, Y., Luo, Y.B., 2005. Postharvest BTH treatment induces resistance of peach (*Prunus persica* L. cv. Jiubao) fruit to infection by *Penicillium expansum* and enhances activity of fruit defense mechanisms. Postharvest Biol. Technol. 35, 263–269.

Liu, J., Tian, S.P., Meng, X.H., Xu Y., 2007. Effects of chitosan on control of postharvest diseases and physiological responses of tomato fruit. Postharvest Biol. Technol. 44, 300–306.

Liu, Y.Y., Ge, Y.H., Bi, Y., Li, Y.C., Deng, H.W., Hu, L.G., Dong, B., 2014. Effect of postharvest acibenzolar-S-methyl dipping on phenylpropanoid pathway metabolism in muskmelon (*Cucumis melo* L.) fruits. Scientia Hortic. 168, 113–119.

Lloyd, A.J., William Allwood, J., Winder, C.L., Dunn, W.B., Heald, J.K., Cristescu, S.M., Sivakumaran, A., Harren, F.J., Mulema, J., Denby, K., 2011. Metabolomic approaches reveal that cell wall modifications play a major role in ethylene mediated resistance against *Botrytis cinerea*. Plant J. 67, 852-868.

Lougheed, E.C., Murr, D.P., Berard, L., 1978. Low pressure storage for horticultural crops. HortScience 13, 21-27.

Lucas, J.A., 1999. Plant immunisation: from myth to SAR. Pesticide Sci. 55, 193–196.

Lurie, S., Pedreschi, R., 2014. Fundamental aspects of postharvest heat treatments. Horticulture Res. 1, 14030.

Lyon, G., 2007. Agents that can elicit induced resistance. In: Walters, D., Newton, A., Lyon, G. (Eds), Induced resistance for plant disease control: a sustainable approach to crop protection. Blackwell Publishing, Oxford, pp. 9–29.

Mari, M., Sivakumar, D., Bautista-Baños S., 2016. The role of microbial and plant volatile organic compounds in decay control in the postharvest system. Postharvest Biol. Technol. (This issue).

Mazaro, S.M., Deschamps, C., de Mio, L.L., Biasi, L.A., de Gouvea, A., Sautter, C.K., 2008. Postharvest behavior of strawberry fruits after pre harvest treatment with chitosan and acibenzolar-S-methyl. Rev. Brasil. Frutic. 30, 185–190.

Meng, X.H., Li, B.Q., Liu, J., Tian, S.P., 2008. Physiological responses and quality attributes of table grape fruit to chitosan preharvest spray and postharvest coating during storage. Food Chem. 106, 501–508.

Meng, X.H., Tian, S.P., 2009. Effects of preharvest application of antagonistic yeast combined with chitosan on decay and quality of harvested table grape fruit. J. Sci. Food Agric. 89, 1838-1842.

Meng, X.C., Tang, Y.X., Zhang, A.Y., Huang, X.M., Zhang, Z.Q., 2012. Effect of oligochitosan on development of *Colletotrichum musae in vitro* and *in situ* and its role in protection of banana fruits. Fruits 67, 147–155.

Mercier, J., Arul, J., Cohen, C., 1993. Effect of UV-C on phytoalexin accumulation and resistance to *Botrytis cinerea* in stored carrots. J. Phytopathol. 137, 44-54.

Minas, I. S., Tanou, G., Belghazi, M., Job, D., Manganaris, G.A., Molassiotis, A., Vasilakakis, M., 2012. Physiological and proteomic approaches to address the active role of ozone in kiwifruit post-harvest ripening. J Exp. Bot. 63, 2449-2464.

Miranda-Castro, S.P., 2016. Application of chitosan in fresh and minimally processed fruits and vegetables. In: Bautista-Baños, S., Romanazzi, G., Jiménez-Aparicio, A. (Eds.), Chitosan in the Preservation of Agricultural Commodities, Academic Press, Elsevier, London, UK, pp. 67-113.

Mittler, R., 2002. Oxidative stress, antioxidants and stress tolerance. Trends Plant Sci. 7, 405-410.

Mittler, R., Vanderauwera, S., Suzuki, N., Miller, G., Tognetti, V.B., Vandepoele, K., Gollery, M., Shulaev, V., Van Breusegem, F., 2011. ROS signaling: the new wave? Trends Plant Sci. 16, 300–309.

Mlikota Gabler, F., Mansour, M., Smilanick, J., Mackey, B., 2004. Survival of spores of *Rhizopus stolonifer*, *Aspergillus niger*, *Botrytis cinerea* and *Alternaria alternata* after exposure to ethanol solutions at various temperatures. J. Appl. Microbiol. 96, 1354–1360.

Morath, S., Hung, R., Bennett, J., 2012. Fungal volatile organic compounds: a review with emphasis on their biotechnological potential. Fungal Biol. Rev. 26, 73–83.

Navarro-Rico, J., Artés-Hernández, F., Gómez, P.A., Núñez-Sánchez, M.Á., Artés, F., Martínez-Hernández, G.B., 2014. Neutral and acidic electrolysed water kept microbial quality and health promoting compounds of fresh-cut broccoli throughout shelf life. Innov. Food Sci. Emerg. Technol. 21, 74-81.

Nigro, F., Ippolito, A., Lattanzio, V., Di Venere, D., Salerno, M., 2000. Effect of ultraviolet-C light on postharvest decay of strawberry. J. Plant Pathol. 82, 29-37.

Niritake, T., Kawakita, K., Doke, N., 1996. Nitric oxide induces phytoalexin accumulation in potato tuber tissues. Plant Cell Physiol. 37, 113–116.

OECD, 2014. Market and trade impacts of food loss and waste reduction. Okawa K. (Ed.). Paris, France (http://www.oecd.org/officialdocuments/publicdisplaydocumentpdf/?cote=TAD/CA/APM/WP(2014)35/FINAL&docLanguage=En)

Ongena, M., Jourdan, E., Adam, A., Paquot, M., Brans, A., Joris, B., Arpigny, J.L., Thonart, P., 2007. Surfactin and fengycin lipopeptides of *Bacillus subtilis* as elicitors of induced systemic resistance in plants. Environ. Microbiol. 9, 1084–1090.

Park, S.W., Kaimoyo, E., Kumar, D., Mosher, S., Klessig, D.F., 2007. Methyl salicylate is a critical mobile signal for plant systemic acquired resistance. Science 318, 113-116.

Pascual, A., Llorca, I., Canut, A., 2007. Use of ozone in food industries for reducing the environmental impact of cleaning and disinfection activities. Trends Food Sci. Technol. 18, 29-35.

Pieterse, C.M.J., Leon-Reyes, A., Van Der Ent, S., Van Wees, S.C.M., 2009. Networking by small-molecule hormones in plant immunity. Nat. Chem. Biol. 5, 308-316.

Pieterse, C.M.J., Van der Does, D., Zamioudis, C., Leon-Reyes, A., Van Wees, S.C.M., 2012. Hormonal modulation of plant immunity. Annu. Rev. Cell Dev. Biol. 28, 489-521.

Pieterse, C.M.J., Zamioudis, C., Berendsen, R.L., Weller, D.M., Van Wees, S.C.M., Bakker, P.A.H.M., 2014. Induced systemic resistance by beneficial microbes. Annu. Rev. Phytopathol. 52:347–75.

Poole, P.R., McLeod, L.C., Whitmore, K.J., Whitaker, G., 1998. Postharvest control of *Botrytis cinerea* rots in stored kiwifruit. Acta Hortic. 464, 71–76.

Porat, R., McCollum, T.G., Vinokur, V., Droby, S., 2002. Effects of various elicitors on the transcription of a β-1,3-endoglucanase gene in citrus fruit. J. Phytopathol. 150, 70–75.

Qin, G.Z., Tian, S.P., Xu, Y., Wan, Y.K., 2003. Enhancement of biocontrol efficacy of antagonistic yeasts by salicylic acid in sweet cherry fruit. Physiol. Mol. Plant Pathol. 62, 147–154.

Reddy, B.M.V., Belkacemi, K., Corcuff, R., Castaigne, F., Arul, J., 2000. Effect of pre-harvest chitosan sprays on post-harvest infection by *Botrytis cinerea* and quality of strawberry fruit. Postharvest Biol. Technol. 20, 39–51.

Ren, Y.L., Wang, Y.F., Bi, Y., Ge, Y.H., Wang, Y., Fan, C.F., Li, D.Q., Deng, H.W., 2012. Postharvest BTH treatment induced disease resistance and enhanced reactive oxygen species metabolism in muskmelon (*Cucumis melo* L.) fruit. Eur. Food Res. Technol. 234, 963–971.

Robert-Seilaniantz, A., Grant, M., Jones, J.D., 2011. Hormone crosstalk in plant disease and defense: more than just jasmonate-salicylate antagonism. Annu. Rev. Phytopathol. 49, 317-343.

Rodrigues Marques, J.P., Amorim, L., Silva-Junior, G.J., Bellato Sposito, M., Appezzato-da Gloria, B., 2015. Structural and biochemical characteristics of citrus flowers associated with defence against a fungal pathogen. AoB PLANTS 7, plu090.

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.

Romanazzi, G., Nigro, F., Ippolito, A., Di Venere, D., Salerno, M., 2002. Effects of pre and postharvest chitosan treatments to control storage grey mold of table grapes. J. Food Sci. 67, 1862-1867.

Romanazzi, G., Mlikota Gabler, F., Smilanick, J.L., 2006. Preharvest chitosan and postharvest UV irradiation treatments suppress gray mold of table grapes. Plant Dis. 90, 445-450.

Romanazzi, G., Nigro, F., Ippolito, A., 2008. Effectiveness of short hyperbaric treatment to control postharvest decay of sweet cherries and table grapes. Postharvest Biol. Technol. 49, 440–442.

Romanazzi, G., Smilanick, J.L., Feliziani, E., Droby, S., 2016a. Integrated management of postharvest gray mold on fruit crops. Postharvest Biol. Technol. 113, 69-76.

Romanazzi, G., Feliziani, E., Bautista-Baños, S., Sivakumar, D., 2016b. Shelf life extension of fresh fruit and vegetables by chitosan treatment. Crit. Rev. Food Sci. Nutr. (in press, doi: 10.1080/10408398.2014.900474).

Ruan, J., Li, M., Jin, H., Sun, L., Zhu, Y., Xu, M., Dong, J., 2015. UV-B irradiation alleviates the deterioration of cold-stored mangoes by enhancing endogenous nitric oxide levels. Food Chem. 169, 417-423.

Ryals, J.A., Neuenschwander, U.H., Willits, M.G., Molina, A., Steiner, H., Hunt, M.D., 1996. Systemic acquired resistance. Plant Cell 8, 1809-1819.

Ryu, C., Farag, A., Hu, C., Reddy, M., Kloepper, J., Paré, P., 2004. Bacterial volatiles induce systemic resistance in *Arabidopsis*. Plant Physiol. 134, 1017–1026.

Santiago, J.A., Rivera-Vargas, L.I., Rodriguez, R.d.P., Macchiavelli, R., 2006. Resistance-inducing chemicals against *Colletotrichum gloeosporioides* in mango. J. Agric. Univ. Puerto Rico, 90, 221–235.

Sanzani, S.M., Nigro, F., Mari, M., Ippolito, A., 2009a. Innovations in the control of postharvest diseases of fresh fruits and vegetables. Arab J. Plant Prot. 27, 240-244.

Sanzani, S. M., De Girolamo, A., Schena, L., Solfrizzo, M., Ippolito, A., Visconti, A., 2009b. Control of *Penicillium expansum* and patulin accumulation on apples by quercetin and umbelliferone. Eur. Food Res. Technol. 228, 381-389.

Sanzani, S. M., Schena, L., De Girolamo, A., Ippolito, A., González-Candelas, L., 2010. Characterization of genes associated with induced resistance against *Penicillium expansum* in apple fruit treated with quercetin. Postharvest Biol. Technol. 56, 1-11.

Sharma, P., Jha, A.B., Dubey, R.S., Pessarakli, M., 2012. Reactive oxygen species, oxidative damage, and antioxidative defense mechanism in plants under stressful conditions. J. Bot., Article ID 217037, doi:10.1155/2012/217037.

Shoresh, M., Harman, G.E., Mastouri, F., 2010. Induced systemic resistance and plant responses to fungal biocontrol agents. Annu. Rev. Phytopathol. 48, 21-43.

Siciliano, I., Amaral Carneiro, A., Spadaro, D., Garibaldi, A., Gullino, M.L., 2015, Jasmonic acid, abscisic acid and salicylic acid are involved in the phytoalexin responses of rice to *Fusarium fujikuroi*, a high gibberellin producer pathogen. J. Agric. Food Chem. 63, 8134-8142.

Sivankalyani, V., Feygenberg, O., Diskin, S., Wright, B., Alkan, N., 2016. Increased anthocyanin and flavonoids in mango fruit peel are associated with cold and pathogen resistance. Postharvest Biol. Technol. 111, 132-139.

Spadaro, D., Droby, S., 2016. Development of biocontrol products for postharvest diseases of fruit: the importance of elucidating the mechanisms of action of yeast antagonists. Trends Food Sci. Technol. 47, 39-49.

Spadoni, A., Guidarelli, M., Sanzani, S.M., Ippolito, A., Mari, M., 2014. Influence of hot water treatment on brown rot of peach and rapid fruit response to heat stress. Postharvest Biol. Technol. 94, 66-73.

Spadoni, A., Guidarelli, M., Phillips, J., Mari, M., Wisniewski, M., 2015. Transcriptional profiling of apple fruit in response to heat treatment: involvement of a defense response during *Penicillium expansum*. Postharvest Biol. Technol. 101, 37-48.

Spoel, S.H., Dong, X., 2008. Making sense of hormone crosstalk during plant immune responses. Cell Host Microbe 3, 348-351.

Stevens, C., Liu, J., Khan, V.A., Lu, J.Y., Kabwe, M.K., Wilson, C.L., Igwegbe, E.C.K., Chalutz, E., Droby, S., 2004. The effect of low-dose ultraviolet light-C treatment on polygalacturonase activity, delay ripening and Rhizopus soft rot development of tomatoes. Crop Prot. 23, 551-554.

Sun, F., Zhang, P., Guo, M., 2013. Burdock fructooligosaccharide induces fungal resistance in postharvest Kyoho grapes by activating the salicylic acid-dependent pathway and inhibiting browning. Food Chem. 138, 539-546.

Tada, Y., Spoel, S.H., Pajerowska-Mukhtar, K., Mou, Z., Song, J., Wang, C., Zuo, J., Dong, X., 2008. Plant immunity requires conformational charges of NPR1 via S-nitrosylation and thioredoxins. Science 321, 952-956.

Terry, L.A., Joyce, D.C., 2004. Elicitors of induced disease resistance in postharvest horticultural crops: a brief review. Postharvest Biol. Technol. 32, 1–13.

Tian, S.P., Wan, Y.K., Qin, G.Z., Xu, Y., 2006. Induction of defense responses against *Alternaria* rot by different elicitors in harvested pear fruit. Appl. Microbiol. Biotechnol. 70,729-734.

Tian, S.P., Yao, H.J., Deng, X., Xu, X.B., Qin, G.Z., Chan, Z.L., 2007. Characterization and expression of β-1,3-glucanase genes in jujube fruit induced by the biocontrol microbial agent, *Cryptococcus laurentii*. Phytopathology 97, 260-268.

Ton, J., Mauch-Mani, B., 2004. Beta-amino-butyric acid-induced resistance against necrotrophic pathogens is based on ABA-dependent priming for callose. Plant J. 38, 119-130.

Tzortzakis, N.G., 2007. Methyl jasmonate-induced suppression of anthracnose rot in tomato fruit. Crop Prot. 26, 1507–1513.

Usall, J., Ippolito, A., Sisquella, M., Neri, F., 2016. Physical treatments to control postharvest pathogens. Postharvest Biol. Technol. (this issue).

USDA, 2014. The estimated amount, value, and calories of postharvest food losses at the retail and consumer levels in the United States, by Buzby, J.C., Wells, H.F., Hyman, J. EIB-121, US Department of Agriculture, Economic Research Service (http://www.ers.usda.gov/publications/eib-economic-information-bulletin/eib-xxx.aspx).

Vallad, G., Goodman, R., 2004. Systemic acquired resistance and induced systemic resistance in conventional agriculture. Crop Sci. 44, 1920–1934.

Valverde, J., Giménez, M., Guillén, F., Valero, D., Martínez-Romero, D., Serrano, M., 2015. Methyl salicylate treatments of sweet cherry trees increase antioxidant systems in fruit at harvest and during storage. Postharvest Biol. Technol. 109, 106–113.

Van Wees, S.M., Luijendijk, M., Smoorenburg, I., van Loon, L., Pieterse, C.J., 1999. Rhizobacteria-mediated induced systemic resistance (ISR) in Arabidopsis is not associated with a direct effect on expression of known defense-related genes but stimulates the expression of the jasmonate-inducible gene Atvsp upon challenge. Plant Mol. Biol. 41, 537-549.

Verhage, A., Saskia, C.M., van Wees, S., Pieterse, C.M.J., 2010. Plant immunity: it’s the hormones talking, but what do they say? Plant Physiol. 154, 536-540.

Walters, D., Fountaine, J., 2009. Practical application of induced resistance to plant diseases: an appraisal of effectiveness under field conditions. J. Agric. Sci. 147, 523–535.

Walters, D.R., Ratsep, J., Havis, N.D., 2013. Controlling crop diseases using induced resistance: challenges for the future. J. Exp. Bot. 64, 1263-1280.

Wang, F.D., Feng, G.H., Chen, K.S., 2009a. Defense responses of harvested tomato fruit to burdock fructooligosaccharide, a novel potential elicitor. Postharvest Biol. Technol. 52, 110-116.

Wang, K., Jin, P., Cao, S., Shang, H., Yang, Z., Zheng, Y., 2009b. Methyl jasmonate reduces decay and enhances antioxidant capacity in Chinese bayberries. J. Agric. Food Chem. 57, 5809–5815.

Wang, A.Y., Lou, B.G., Xu, T., 2011a. Defense responses in tomato fruit induced by oligandrin against *Botrytis cinerea*. Afr. J. Biotechnol. 10, 4596-4601.

Wang, J.J., Bi, Y., Zhang, Z.K., Zhang, H.Y., Ge, Y.H., 2011b. Reducing latent infection and enhancing disease resistance in muskmelon by preharvest application of harpin. J. Agric. Food Chem. 59, 12527–12533.

Wang, K., Jin, P., Han, L., Shang, H., Tang, S., Rui, H., Zheng, Y., 2014. Methyl jasmonate induces resistance against *Penicillium citrinum* in Chinese bayberry by priming of defense responses. Postharvest Biol. Technol. 98, 90-97.

Wang, K., Cao, S., Di, Y., Liao, Y., Zheng, Y., 2015a. Effect of ethanol treatment on disease resistance against anthracnose rot in postharvest loquat fruit. Scientia Hortic. 188, 115-121.

Wang, K., Liao, Y., Cao, S., Di, H., Zheng, Y., 2015b. Effects of benzothiadiazole on disease resistance and soluble sugar accumulation in grape berries and its possible cellular mechanisms involved. Postharvest Biol. Technol. 102, 51-60.

Wightwick, A., Walters, R., Allinson, G., Reichman, S., Menzies N., 2010. Environmental risks of fungicides used in horticultural production systems. In: Carisse O. (Ed.) Fungicides. In-Tech, Croatia, pp. 273-304.

Wilson, C.L., El Ghaouth, A., Chalutz, E., Droby, S., Stevens, C., Lu, J.Y., Khan, V., Arul, J., 1994. Potential of induced resistance to control postharvest diseases of fruits and vegetables. Plant Dis. 78, 837-844.

Xu, X.B., Chan, Z.L., Xu, Y., Tian, S.P., 2008a. Synergistic effect of antagonist yeast and SA on controlling brown rot in peach fruit and its mechanism. J. Sci. Food Agric. 88, 1786-1793.

Xu, X.B., Qin, G., Tian, S., 2008b. Effect of microbial biocontrol agents on alleviating oxidative damage of peach fruit subjected to fungal pathogen. Int. J. Food Microbiol. 126, 153-158.

Yan, J.Q., Cao, J.K., Jiang, W.B., Zhao, Y.M., 2012. Effects of preharvest oligochitosan sprays on postharvest fungal diseases, storage quality, and defensive responses in jujube (*Zizyphus jujube* Mill. cv. Dongzao) fruit. Scientia Hortic. 142, 196–204.

Yang, Z.F., Cao, S.F., Cai, Y.T., Zheng, Y.H., 2011. Combination of salicylic acid and ultrasound to control postharvest blue mold caused by *Penicillium expansum* in peach fruit. Innov. Food Sci. Emerg. Technol. 12, 310-314.

Youssef, K., Sanzani, S. M., Ligorio, A., Ippolito, A., Terry, L. A., 2014. Sodium carbonate and bicarbonate treatments induce resistance to postharvest green mould on citrus fruit. Postharvest Biol. Technol. 87, 61-69.

Yu, M.M., Shen, L., Fan, B., Zhao, D.Y., Zheng, Y., Sheng, J.P., 2009. The effect of MeJA on ethylene biosynthesis and induced disease resistance to *Botrytis cinerea* in tomato. Postharvest Biol. Technol. 54, 153–158.

Yu, Q., Chen, Q., Chen, Z.W., 2012. Activating defense responses and reducing postharvest blue mold decay caused by *Penicillium expansum* in peach fruit by yeast saccharide. Postharvest Biol. Technol. 74, 100-107.

Zainuri, J., Joyce, D.C., Wearing, A.H., Coates, L., Terry, L., 2001. Effects of phosphonate and salicylic acid treatments on anthracnose disease development and ripening of 'Kensington Pride' mango fruit. Austral. J. Exp. Agric. 41, 805–813.

Zeng, K.F., Deng, Y.Y., Ming, J., Deng, L.L., 2010. Induction of disease resistance and ROS metabolism in navel oranges by chitosan. Scientia Hortic. 126, 223-228.

Zhang, Z.Q., Tian, S.P., Zhu, Z., 2012. Effects of 1-methylcyclopropene (1-MCP) on ripening and resistance of jujube (*Zizyphus jujuba* cv. Huping) fruit against postharvest disease. LWT-Food Sci. Technol. 45, 13-19.

Zhang, Z., Yang, D., Yang, B., Gao, Z., Li, M., Jiang, Y., Hu, M., 2013. β-Aminobutyric acid induces resistance of mango fruit to postharvest anthracnose caused by *Colletotrichum gloeosporioides* and enhances activity of fruit defense mechanisms. Scientia Hortic. 160, 78-84.

Zhang, C., Shao, Q., Cao, S.X., Tang, Y.F., Liu, J.Y., Jin, Y.Z., Qi, H.Y., 2015. Effects of postharvest treatments on expression of three lipoxygenase genes in oriental melon (*Cucumis melo* var. makuwa Makino). Postharvest Biol. Technol. 110, 229-238.

Zhao, Y., Tu, K., Shao, X., Jing, W., Su, Z., 2008. Effects of the yeast *Pichia guilliermondii* against *Rhizopus nigricans* on tomato fruit. Postharvest Biol. Technol. 49, 113-120.

Zheng, X.L., Tian, S.P., Li, B.Q., Xu, Y., 2005. Changes in antioxidant systems and polyphenol oxidase activity in peach fruit treated with exogenous oxalic acid during storage at low temperature. Acta Hortic. Sin. 32, 788–792.

Zheng, Y., Sheng, J.P., Zhao, R.R., 2011. Preharvest L-arginine treatment induced postharvest disease resistance to *Botrytis cinerea* in tomato fruits. J. Agric. Food Chem. 59, 6543-6549.

Zhu, Z., Zhang, Z.Q., Qin, G.Z., 2010. Effects of brassinosteroids on postharvest disease and senescence of jujube fruit in storage. Postharvest Biol. Technol. 56, 50-55.

Zhu, Z., Tian, S.P., 2012. Resistant responses of tomato fruit treated with exogenous methyl jasmonate to *Botrytis cinerea* infection. Scientia Hortic. 142, 38–43.

Zhu, Z., Zhang, X., 2016. Effect of harpin on control of postharvest decay and resistant responses of tomato fruit. Postharvest Biol. Technol. 112, 241-246.

Zimmerli, L., Jakab, C., Metraux, J.P., Mauch-Mani, B., 2000. Potentiation of pathogen specific defense mechanisms in Arabidopsis by beta-aminobutyric acid. Proc. Natl. Acad. Sci. USA 97, 12920-12925.

Figure captions

Figure 1. Number of articles available through Scopus over the last 30 years using the search keywords of “induced resistance postharvest” (accessed on June 16, 2016).

Figure 2. Treatments that can induce resistance in fresh fruit and vegetables, and the mechanisms or enzymes involved. Images from http://cliparts.co/free-fruit-pictures.

INA, 2,6-Dichloronicotinic acid; HWT, Hot water treatment; GRAS, generally recognized as safe; JA, jasmonic acid; SA, salicylic acid; MeJa, methyl jasmonate; BFO, Burdock fructooligosaccharide; BTH, benzothiadiazole; EOW, Electrolyzed oxidizing water; NO, nitric oxide; UV-C, ultraviolet C irradiation; BCA, biocontrol agents; MVOCs, microbial volatile organic compounds; Si, silicon; PVOCs, plant volatile organic compounds; EtOH, ethanol; SC, sodium carbonate; SAR, systemic acquired resistance; PG, polygalacturonase; BABA, β-aminobutyric acid; GLU, β-1,3-glucanase; PR, pathogenesis related proteins; MAMP, microbe-associated molecular pattern; LOX, lipoxygenase; ROS, reactive oxygen species; CHT, chitinase; CAT, catalase; ABA, abscisic acid; PPO, polyphenol oxidase; PAL, phenylalanine ammonia lyase; ISR, induced systemic resistance; PAMP, pathogen-associated molecular pattern; SOD, superoxide dismutase; NPR1, nonexpressor of pathogenesis-related genes 1; hsp, heat shock protein.

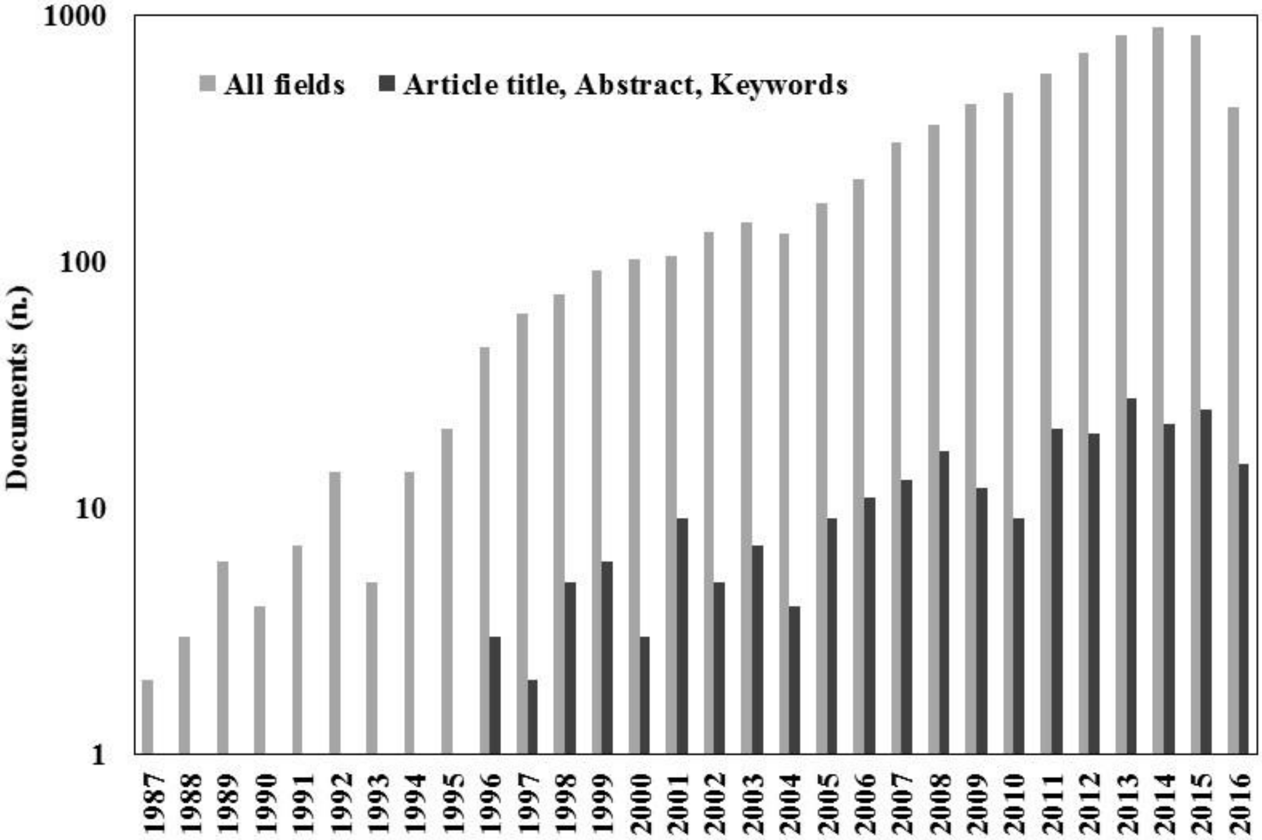
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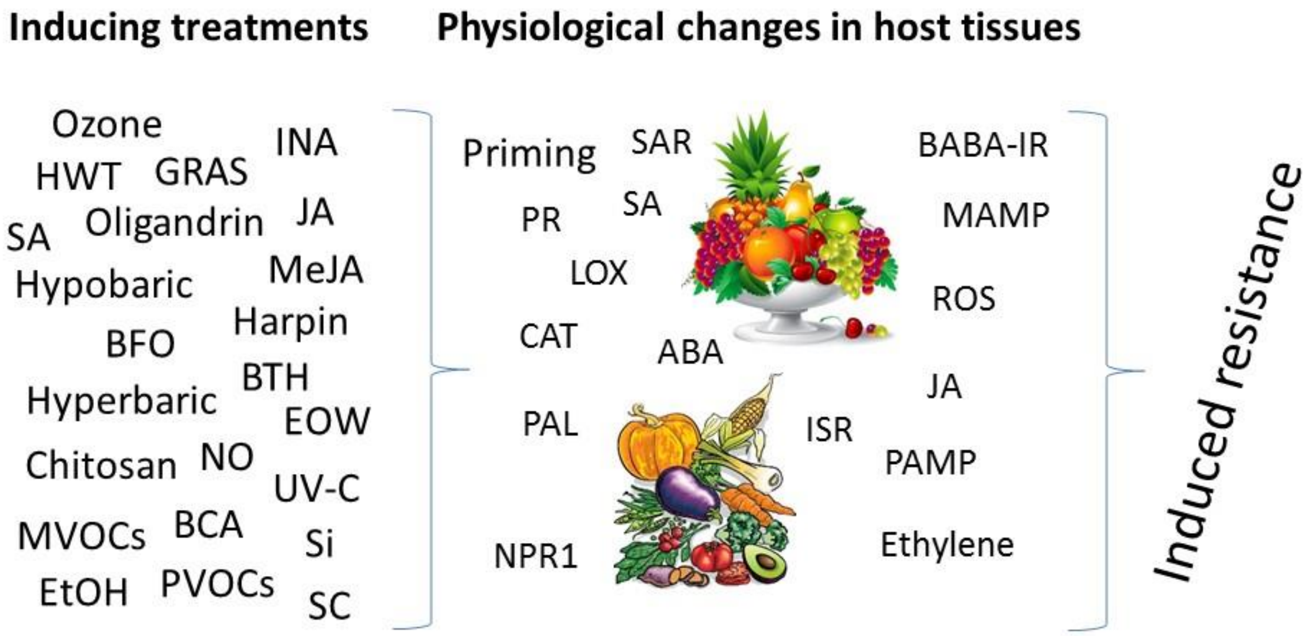
Fig. 1


Inducing treatments
Physiological changes in host tissues
Ozone
INA
HWT
GRAS
SA
Oligandrin
JA
Hypobaric
MeJA
Harpin
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BTH
Hyperbaric
EOW
Chitosan
NO
UV-C
MVOCs
BCA
Si
EtOH
PVOCs
SC
Priming
SAR
PR
SA
LOX
CAT
ABA
PAL
NPR1
ISR
BABA-IR
MAMP
ROS
JA
PAMP
Ethylene
Induced resistance

Fig. 2

Table 1. Examples of differential gene expression or enzyme activities in response to the application of biocontrol agents.

Treatment	**Genes and/or enzymes**										**Reference**
	PAL*	**CHT**	**SOD**	**CAT**	**SA**	**LOX**	**GLU**	**SURFACTIN**	**FENGICIN**	**POD**	
Bacillus subtilis						+/++**		+/+++	+/+++		Ongena et al., 2007
Candida saitoana		+/++					+				El Ghaouth et al., 2003b
Cryptococcus laurentii							+				Tian et al., 2007
Pichia membranifaciens, Candida guilliermondii Rhodotorula glutinis		+		+/+++			+/+++			+/++	Xu et al., 2008b
Pichia guilliermondii	+	+	+	-/+			+			-/+	Zhao et al., 2008
Aureobasidium pullulans		+					+			+	Ippolito et al., 2000

*PAL, phenylalanine ammonia lyase; CHT, chitinase; SOD, superoxide dismutase; CAT, catalase; SA, salicylic; LOX, lipoxygenase; GLU, β-1,3-glucanase; POD, peroxidase

**+, overexpressed up to 3-fold; ++, overexpressed from 4-fold to 10-fold; +++, overexpressed more than 10-fold; -, down-regulated up to 3-fold

Table 2. Examples of differential gene expression or enzyme activities in response to application of physical means.

Treatment	**Genes and/or enzymes**						**Reference**
	PAL*	**CHT**	**GLU**	**POD**	***Trans*-resveratrol**	**Catechin**	
UV-C irradiation	+**	++/+++	+		++/+++	++	Cantos et al., 2000; Nigro et al., 2000; El Ghaouth et al., 2003a; Romanazzi et al., 2006
Heat treatment	+/-	-					Spadoni et al., 2014, 2015
Hypobaric treatment	+	+		+			Hashmi et al., 2013

*PAL, phenylalanine ammonia lyase; CHT, chitinase; GLU, β-1,3-glucanase; POD, peroxidase

**+, overexpressed up to 3-fold; ++, overexpressed from 4-fold to 10-fold; +++, overexpressed more than 10-fold; -, down-regulated up to 3-fold

Table 3 Examples of differential gene expression or enzyme activities in response to application of natural and synthetic chemicals.

Treatment	**Genes and/or enzymes**								**Reference**
	SOD	**CAT**	**POD**	**APX**	**CHT**	**PAL**	**GLU**	**PPO**	
Salicylic acid		-**	++	-	+	++	+		Cao et al., 2006
Methyl salicylic acid	+	++	++	+					Valverde et al., 2015
Benzothiadiazole	+	+	++		+	+	++		Cao et al., 2005
β-aminobutyric acid			+		+				Bokshi et al., 2006
Riboflavin			++			+		+	Li et al., 2012b
1-Methylcyclopropene	+	+				+		+	Zhang et al., 2012
Harpin			+++		+++				Bi et al., 2005
Oligandrin			+			+		+	Wang et al., 2011a
Chitosan			+	+/++	+/++	+/+++	+/+++	+	Romanazzi et al., 2002; Liu et al., 2007; Meng et al., 2012; Yan et al., 2012; Feliziani et al., 2013b; Landi et al., 2014
Yeast saccharide			++		+	+++	+++		Yu et al., 2012
Silicon			+++		+++				Bi et al., 2006b
Sodium carbonate			++		-	++	++		Youssef et al., 2014

SOD, superoxide dismutase; CAT, catalase; POD, peroxidase; APX, ascorbate peroxidase; CHT, chitinase; PAL, phenylalanine ammonia lyase; GLU, β-1,3-glucanase; PPO, polyphenol oxidase

**+, overexpressed up to 3-fold; ++, overexpressed from 4-fold to 10-fold; +++, overexpressed more than 10-fold; -, down-regulated up to 3-fold

Table 4. Examples of differential gene expression or enzyme activities in response to application of disinfecting agents.

Treatment	**Genes and/or enzymes**						**Reference**
	PPO*	**GLU**	**PAL**	**LOX**	**POD**	**CHT**	
Ozone			+**			+	Duarte-Sierra et al., 2016
Electrolyzed water			+		++	+++	Fallanaj et al., 2016
Ethanol				-			Zhang et al., 2015

*PPO, polyphenol oxidase; GLU, β-1,3-glucanase; PAL, phenylalanine ammonia lyase; LOX, lipoxygenase; POD, peroxidase; CHT, chitinase

**+, overexpressed up to 3-fold; ++, overexpressed from 4-fold to 10-fold; +++, overexpressed more than 10-fold; -, down-regulated up to 3-fold

Table 5. Aspects related to the induction of resistance to postharvest diseases of fruit and vegetables

Negative sides	Positive sides
Complete effect is not always reproducible	Long-lasting effects
Does not provide a complete control of decay	Broad range of targets
Not easy to implement as part of farmer and packinghouse practices	Do not cause appearance of resistant isolates of the pathogen
Investigation methods are not standardized	Increasing number of biostimulants on the market
	Low side effects
	Reduction of pesticide use
	Promoted by EU Directive n. 128/2009 «Sustainable Use of Pesticides» and following National Action Plans
	Increased amounts of beneficial antioxidant compounds