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Review

The Role of 1-methylcyclopropylene (1-MCP) and Salicylic Acid (SA) in Induced Resistance of Postharvest Fruits

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Abstract: Postharvest diseases cause huge postharvest losses of horticultural fresh produce. Cooling and synthetic fungicide are used as traditional postharvest preservation technology. Recently, induced resistance has been thought to be an optional and perhaps alternative preservation technology. 1-methylcyclopropylene (1-MCP) and salicylic acid (SA) are two more common chemical agents used mostly as a preservative for harvested fruit in order to achieve better quality and better taste. Many reports have also proven that 1-MCP and SA could induce postharvest fruit resistance. The purpose of this review is to summarize the role of 1-MCP and SA in postharvest fruit resistance, including the effect of 1-MCP and SA on the induced resistance as well as its involved mechanism; the effects of 1-MCP and SA on firmness, phenolic metabolism, membrane lipid metabolism, and reactive oxygen species in fruit after harvest; and the effects of 1-MCP and SA on disease resistance-related defense enzymes, proteins, signaling synthesis, and signaling pathways as well as the combined effect of 1-MCP and SA on the induced resistance and its mechanism. Meanwhile, we prospect for the future direction of increasing postharvest fruit resistance by 1-MCP and SA in more depth.

Keywords: 1-MCP; salicylic acid; induced resistance; postharvest; fruit

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1. Introduction

Although the harvested fruit loses the supply of water and nutrients from the mother, it maintains life activities using its own stored nutrients. During postharvest storage, transport, and sale, a series of biochemical reactions occur, the metabolism changes from synthesis to hydrolysis, and the susceptibility to pathogens increase. Pathogens infecting fruits before or after harvest can cause postharvest decay during storage, transport, and marketing, and the occurrence and development of diseases depends on both fruit physiological characteristics and storage environmental conditions after harvest [1]. It was reported that 10 to 30% of fresh fruit in developed countries rotted after harvest, and 30 to 50% in developing countries decayed due to lacking storage technique [2]. Citrus, for example, is the world's largest fruit industry. Citrus postharvest diseases are mainly caused by fungi. The rot rate was 10% to 30% and even up to 50%, causing serious economic losses in China [3].

Fungicides are widely used for fruit disease control. Currently, there is growing consumer concern about fungicide residues in fruit and the emergence of fungicide-resistant strains. It is necessary to explore effective and harmless fungicides or treatments that can induce resistance. With the treatment of biotic or abiotic elicitors, postharvest fruits can be indued with a natural resistance to pathogenic microorganism through activation of their own defense system, effectively resisting and killing pathogenic microorganisms, which is called postharvest induced resistance. The induced resistance is characterized by persistence and broad-spectrum to green mold, blue mold, gray mold, anthracnose, and several fugal diseases, conferring resistance to many kinds of pathogen infection without

leading to the appearance of resistant strains, indicating a potential application perspective in the control and prevention of postharvest disease in fruit [4]. Treatment with yeast, heat, UV, 1-methylcyclopropylene (1-MCP), ethylene, and aminobutyric acid has been widely used to induce resistance in postharvest fruit.

1-MCP is an ethylene competitive inhibitor that can irreversibly occupy the ethylene binding receptor and therefore delay fruit senescence. For its efficiency, simplicity to operate, low cost, and good economic benefits, 1-MCP has been used widely in fruit storage. It can be used as a preservative alone or mixed with other preservatives or treatments, such as heat treatment, ClO_2, salicylic acid (SA), jasmonic acid (JA), etc., to achieve the preservation effect. However, some literature also reported that 1-MCP could induce fruit resistance, for example, in apple, fragrant pear, strawberry, peach, and persimmon [5,6]. Therefore, in addition, 1-MCP can be used as an abiotic elicitor to induce resistance and prevent postharvest disease in fruits after harvest.

SA is a common, simple phenolic compound in plants. It can not only produce antagonistic substances with many pathogenic microorganisms such as bacteria, fungi, and viruses but also act as a plant growth regulator to improve the resistance of crops to adverse environmental factors and promote photosynthesis. SA has important influence on plant disease resistance as well as on fruit maturity, horticultural produce preservation, and seed germination [7]. SA is an endogenous signaling molecule to activate the system-acquired disease resistance (SAR) and enhance plant defense and protection mechanisms [8]. It has been reported that exogenous SA has the potential to significantly improve harvest fruit resistance [9].

This review focuses on the role of 1-MCP and SA in the induction of postharvest fruit resistance, including their effects on reactive oxygen metabolism, phenolic metabolism, defense enzymes and proteins, resistance-related hormone synthesis and the signal transduction pathway, and their combined use in the control of postharvest diseases of fruits. This study sheds important light on the role of 1-MCP and SA in postharvest fruits resistance and facilitates the development of new and safer strategies for elevating postharvest fruit resistance against pathogenic microorganism. The outlook for further application of 1-MCP and SA on postharvest storage of fruit is also presented in this study.

2. The Role of 1-MCP in Induced Resistance of Postharvest Fruits

2.1. Effect of 1-MCP on Induced Resistance and Its Involved Mechanism

As shown in Table 1, some certain concentrations of 1-MCP can improve fruit disease resistance and, ultimately, disease performance. Li et al. showed that treatment with 1-MCP at a concentration of 5 μL/L effectively inhibited postharvest blue mold caused by *Penicillium expansum* in apple fruit [10]. Further, 1 μL/L 1-MCP significantly reduced the incidence of apple gray mold and inhibited the expansion of disease spots compared with the control [11], and 1 μL/L 1-MCP treatment could also reduce the disease of blue mold, gray mold, and anthracnose in golden delicious apples [12]. Jiang et al. showed that the main disease of "Everest" strawberry was fruit rot caused by a natural infection of the roots in strawberry, and treatment with 1 μL/L 1-MCP reduced disease occurrence [13], while 5 μL/L 1-MCP could effectively inhibit the increase of spot diameter, significantly reduce the disease incidence, and enhance the disease resistance to *Penicillium digitatum* in citrus fruit [14]. Additionally, 1 mg/L 1-MCP treatment could also prevent and control postharvest fruit diseases such as in peach and jujube [15,16]. In this way, 1-MCP can induce resistance to blue mold (*Penicillium expansum*), gray mold (*Botrytis cinerea*), anthracnose (anthracnosis), and other diseases in apple, strawberry, kumquat, peach, jujube, and other fruits. 1-MCP at 1 μL/L for 24 h is generally the best choice for treatment.

The current mechanism of resistance of 1-MCP-induced fruits includes maintaining firmness and delaying senescence to maintain natural resistance, which inhibits the increase in conductivity and malondialdehyde content, reduces the accumulation of reactive oxygen species (ROS), impairs phenolic metabolism, and increases the activity of certain key

antioxidant enzymes and the induction of higher defense-related enzymatic activity such as chitinase and β-1, 3-glucanase.

Table 1. Studies on the induced resistance effect of 1-MCP treatment on different types of fungus in postharvest fruits.

Fruits	Treatment Concentration	Fungi	Reference
"Fuji" apple	1 μL/L	*Botrytis cinerea*	[6]
"Everest" strawberry	1 μL/L	*Botrytis cinerea*	[13]
Jujube	1 μL/L	*Botrytis cinerea*	[16]
"Golden Delicious" apple	0.3 μL/L	*Colletotrichum acutatum*	[12]
Cactus pear	1 μL/L	*Colletotrichum gloeosporioides*	[17]
Carambola	0.6 μL/L	*Colletotrichum gloeosporioides*	[18]
Loquat	50 nL/L	*Colletotrichum gloeosporioides*	[19]
"Anxi" persimmon	1.35 μL/L	*Gloeosporium kaki*	[20]
"Dangshan" pear	1 μL/L	*Penicillium digitatum*	[21]
"Emerald" pear	1 μL/L	*Penicillium digitatum*	[22]
Citrus	5 μL/L	*Penicillium digitatum*	[14]
"Fuji" apple	5 μL/L	*Penicillium expansum*	[10]
"Golden Delicious" apple	0.3 μL/L	*Penicillium expansum*	[23]
Peach	0.6 μL/L	*Penicillium expansum*	[15]
"Hongyang" kiwifruit	0.8 μL/L	*Phomopsis* sp.	[24]

2.2. Effect of 1-MCP on Reactive Oxygen Metabolism

Reactive oxygen species generally refer to some oxygen metabolites and their derivatives that contain oxygen atoms but have more active chemical reactivity than oxygen [25], such as superoxide anion radicals (O_2^-), hydroxyl radicals (OH), hydrogen peroxide (H_2O_2), lipid peroxides (ROO^-), and singlet oxygen ($^1O_2$). Among them, superoxide anions, hydroxyl radicals, and hydrogen peroxide exert their main effects on plant physiological activities. Producing reactive oxygen species is the inherent biological feature in plants. The concentration of reactive oxygen species is quite low under normal physiological conditions: not enough to damage the plant because plants have formed a perfect defense system of removing reactive oxygen species in the process of long-term evolution, which has caused the production and clearance of reactive oxygen species to be kept at a dynamic equilibrium, and the reactive oxygen species will not accumulate. Once the plant is stressed, however, the metabolism of oxygen in the plant changes, the production of reactive oxygen species is accelerated, and the function of the scavenging system is diminished, leading to the accumulation of reactive oxygen species in the body, causing damage to the structure and function of the plant cell and even resulting in death; i.e., the plant is damaged by reactive oxygen species. The antioxidant removal system can transfer or eliminate free radicals and oxidative intermediates, and it is important in the ripening and aging of fruit [26]. Reactive oxygen species can also transmit signals to the disease-infected plants, thus activating the plant defense system and obtaining resistance to pathogens in time.

The excessive accumulation of reactive oxygen species was eliminated by controlling the activity of superoxide dismutase (SOD) and catalase (CAT) so that the reactive oxygen species in fruit was in a dynamic equilibrium, which is the mechanism of 1-MCP in scavenging reactive oxygen species. SOD is widely found in aerobic prokaryotes and eukaryotes and is also the first antioxidant enzyme to function in a reactive oxygen species scavenger system. SOD makes superoxide anion radicals in order to generate hydrogen peroxide and molecular oxygen, which play an important role in the protection of cells from oxidative damage. High SOD activity and lower values of ion leakage were detected in apricots treated with 1 MCP, indicating that 1 MCP reduced ethylene production, enhanced antioxidant capacity, and improved disease resistance [27]. SOD and CAT activity as well as higher levels of ascorbate were significantly increased by 1-MCP treatment, which

enhanced the antioxidant capacity and improved the disease resistance in "Conference" pear fruit [28]. 1-MCP treatment significantly enhanced SOD activity, effectively extended fruit storage time, and increased disease resistance and antioxidant capacity in "Hayward" and "Zihong" kiwifruit [29]. Xia et al. found that by treating red-fleshed kiwifruit with 1-MCP (0.8 μL/L), fruit decay mainly caused by *Phomopsis* sp. was effectively reduced, increased SOD and CAT activity was found to improve the antioxidant capacity of the fruit, and phenolic compound contents and defense-related enzyme activity was elevated [30]. Shi et al. found that 1-MCP had a significant effect on non-climacteric fruit as well. They treated strawberries with 1-MCP, which increased the activity of a variety of antioxidant enzymes such as SOD and increased the protein content, which helped to lower the toxicity of the reactive oxygen species generated during catabolic activity, improving strawberry quality and increasing strawberry disease resistance [31]. Zhang et al. found that 1-MCP did indeed limit the development of the blue rot lesion diameter, causing significant reductions in the incidence of natural rot and increases in SOD activity in jujube fruits, which suggests that 1-MCP-induced resistance in jujube fruit was associated with increased enzymes involved in scavenging reactive oxygen species [16].

2.3. Effect of 1-MCP on Phenolic Metabolism

Phenolic metabolism forms an integral part of secondary metabolism and has an important role in plant defense responses. Phenolic bacteriostat, mainly biosynthesized by benzene propane metabolic pathway, including simple phenolic acids, flavonoids, benzene propane, and polyphenol, is closely related to plant disease resistance. PPO (polyphenol oxidase), POD (peroxidase), and PAL (phenylalanine ammonialyase) are the enzymes involved in the metabolism of phenolic compounds in plants, and they play an important role in the induced disease resistance of fruits.

The mechanism of 1-MCP on phenolic metabolism is to effectively accumulate PAL, PPO, lignin, total phenol, and other disease-resistant substances in pulp through the influence of phenylpropane pathway in fruit, thus improving the postharvest resistance of fruit. PAL is the key enzyme involved in the metabolic pathways of benzene propane, which is involved in the biosynthesis of phenols, flavonoids, phytoalexins, lignins, and other chemical resistance compounds. The activity level of PAL is often regarded as a biochemical index of plant disease resistance. 1-MCP treatment increased PAL, POD, SOD, and CAT in jujube fruit, which enhanced resistance. This suggests that enhanced fruit resistance was associated with increased activity of phenylalanine ammonialyase. Combined 1-MCP and ClO_2 treatment improved PAL activity, with dual physiological and antibacterial effects, and effectively improved the resistance of strawberries [21].

Polyphenol oxidase is a key enzyme in the phenolic metabolism pathway, which is involved in enzymatic browning as well as in the biodefense reactions in fruits. PPO mainly oxidizes phenols into highly toxic material, directly limiting or poisoning the invasive pathogenic microorganism. Peroxidase is mainly involved in the generation of lignin and phytoalexin and can also remove H_2O_2 and O^{2-} from tissue cells to avoid their excessive accumulation in cells. Zhang et al. found that immersion of mature avocado in aqueous solution with 1-MCP significantly retarded the accumulation of total soluble phenolics, flavonoids, and total antioxidant capacity [32]. 1-MCP treatment inhibited postharvest rot of "Anxi" persimmons, delayed the synthesis of phenolic substances, increased PPO enzyme activity, and increased the strength of the postharvest fruit [20].

2.4. Effect of 1-MCP on Disease Resistance-Related Defense Enzymes and Proteins

Plants respond to attacks by pathogens by producing a wide range of pathogenesis related (PR) proteins [33,34]. The first family of these proteins (PR-1) was identified in the early 1980s [35]. Since then, a crowd of reports have documented similar PR-1 proteins originating from numerous species of monocot and dicot plants [36–40]. Transgenic expression in plants confirmed the defensive function of some PR-1 proteins [41–45]. These PR proteins have now been classified into 17 different families [46], including β-

1,3-glucanases (PR-2), chitinases (PR-3, -4, -8, and -11), osmotins with proteins similar to proteins (PR-5), defensins (PR-12), thionins (PR-13), lipid-transfer proteins (PR-14), etc. It is well-established that signaling compounds such as abscisic acid (ABA), ethylene, jasmonic acid (JA), salicylic acid (SA), mechanical wounding, UV light exposure, osmotic stress, and microbial infection are involved in the regulation of PR expression [47–51]. PR proteins have been shown to be closely related to systemic acquired resistance (SAR) and systemic induced resistance (ISR) [52].

1-MCP can improve fruit resistance to pathogens by inducing a wide range of pathogenesis-related (PR) proteins in fruit. Chitinase (CHI) acts on the fungal cell wall to degrade its chitin, to destroy the cytoskeleton of the fungus, and to achieve antifungal effect [53]. After fungal infection, rice was treated with 1-MCP, and the rice's chitinase activity increased; the germination, growth, and reproduction of fungal spores was inhibited; and the rice's resistance was enhanced [54]. Using a transcriptome study, Li et al. found that 1-MCP alleviated the overall transcriptome changes after refrigeration in cactus pear fruit [17]. Functional classifier analysis revealed that the most significant effect of 1-MCP was to avoid the large-scale downregulation of transcripts belonging to the stress, RNA and transcription, signaling, and cellular classes. In addition, exposure to 1-MCP significantly reduced the levels of phytase and chlorophyll enzyme transcripts as well as increased chitinase transcript levels, thereby providing molecular evidence for its observed effects in delaying pericarp staining and increasing resistance to pathogens. β-1,3-glucanases (GLU) belong to the PR-2 class and play an important role in disease resistance in plants [55]. β-1,3-Dextran is an important structural component of the fungal cell wall, exposed on the surface of many fungal hyphal tips and capable of being directly attacked by β-1,3-glucanase [56]. β-1,3-glucanase is often expressed in concert with chitinase in plant defense responses, thus enhancing plant disease resistance [57]. Cao et al. found that 1-MCP treatment significantly reduced the rot incidence of loquat fruit by significantly inhibiting the accumulation of superoxide radicals and hydrogen peroxide, inducing higher activity of chitinase and β-1,3-glucanase and maintaining natural resistance by delaying senescence [19]. Treatment with 1-MCP inhibited fruit decomposition in postharvest "Anxi" persimmon likely because 1-MCP promoted disease resistance by increasing the activity of chitinase and β-1,3-glucanase and by retaining higher amounts of substances related to disease resistance. 1-MCP + PL treatment also induced an increase in PAL activity and increased intracellular lignin and total phenolic content, which is conducive to the formation of structural barriers with indirect resistance to pathogens. The disease index of the 1-MCP + PL-treated fruit was significantly lower than control. The 1-MCP treatment alleviated the occurrence of disease during fruit storage and reduced postharvest decay loss in melo fruit [58]. Lin et al. found that treatment with paper leaf 1-MCP effectively reduced disease index in the carambola fruit; produced increased CHI, GLU, PAL, PPO, and POD activity; and maintained a high total phenolic content. [18].

PR1 is a marker gene of SAR. The molecular mechanism of the SA-regulating *PR1* gene in response to pathogenic microorganism infection has been widely studied in model plants. Studies in *Arabidopsis* showed that the nonexpressor of pathogenesis-related gene-1 (*NPR1)*, as a key contributor to SA signaling, interacts with TGACG motif-binding transcription factor (TGA) transcription factors, activating SA response elements in the promoter of PR1 [59]. Later, the researchers successively confirmed the prevalence of *PR1* in plants and its role in the defense process in the plant leaves. Niki et al. found that overexpression of *AtPR1* in *Arabidopsis* enhanced resistance to downy mildew [60]. Li et al. found that expression of *VvPR1* in response to tobacco wildfire infection and *PsPR1* expression increased within 24 h [61]. To our knowledge, there was no report on induced expression of *PR1* by 1-MCP treatment in postharvest fruits. However, in soft spring wheat (*Triticum aestivum* L.), 1-MCP induced disease resistance to leaf spots (*Septoria nodorum* Berk.) by increasing zeatin, hydrogen peroxide, and accumulation of gene transcripts (*PR-1* and *PR-2*). All of the defense responses studied were suppressed, and pathogen development was more concentrated in control and ethylene-treated wheat. Cytokinins

were localized to the mesophyll cells and cell walls of wheat leaves treated with 1-MCP. The cell walls of ET-treated leaves were devoid of zeatin, and the hormone was concentrated in the developing hyphae of the pathogen. These findings make it possible to hypothesize that wheat resistance is controlled by an antagonistic interplay of salicylic acid and ethylene signaling pathways with cytokinin participation [62].

2.5. Effect of 1-MCP on Fruit Firmness

Texture softening mostly occurs in the process of fruit ripening, which makes the fruit flavor quality and taste reach its best state but makes the fruit more susceptible to microbial infection and physical damage [63]. 1-MCP improves fruit resistance to pathogens by inhibiting the degradation of cell walls. It was shown that 1-MCP could inhibit the enzyme activity related to softening in the ripening process (exo-polygalacturonase, pectin methylesterase, β-galactosidase, and α-L-arabinofuranosidase), thus inhibiting the degradation of cell wall and delaying fruit softening in apple fruits [64]. Lohani et al. determined the activity changes of pectinesterase (PE), polygalacturonase (PG), and cellulase (Cx) in banana fruits during storage and found that 1-MCP was able to effectively inhibit the effect of ethylene on cell wall hydrolase as well as reduce fruit softening. The addition of 1-MCP could significantly reduce the rate of starch conversion in harvested apple; inhibit degradation of pectin, cellulose, and hemicellulose; and retard softening [65]. 1-MCP could significantly reduce the conversion rate of starch in harvested apple; inhibit the degradation of pectin, cellulose, and hemicellulose, and delay softening [66]. 1-MCP treatment could delay the peak emergence of amylase and PE activity in kiwi fruit, inhibit the degradation of starch and pectin, significantly maintain fruit firmness, and delay softening and aging [67]. Ethylene induced the expression of the PG gene *DkPG1* in persimmon fruit, while 1-MCP treatment suppressed gene expression of *DkPG1*, reduced the PG enzyme activity, and maintained a high pulp firmness [68]. It is possible that 1-MCP may effectively inhibit the activity of softening-related enzymes such as PE and PG and delay the process of softening after ripening in "Santa Rosa" plum fruit [69].

2.6. Effect of 1-MCP on Increasing Fruit Disease

In some studies, 1-MCP was reported to be able to reduce postharvest fruit strength and promote disease occurrence. For example, treatment with 1-MCP reduced resistance in Japanese prickly pear, with more severe symptoms of black spots [70]. Porat et al. found that 1-MCP increased rot caused by penicillium in "Shamouti" oranges because ethylene inhibited mold growth, while 1-MCP inhibited ethylene [23]. Janisiewicz et al. found that 1-MCP treatment increased the severity of bitter rot and blue molds, reducing disease resistance of "Golden Delicious" apples [71]. It was shown that although 1-MCP treatment delayed fruit ripening, it also changed the component of the fruit, which increased tissue morbidity [21]. Sun et al. found that after storage for 120 days, the rot rate of 1-MCP-treated fruit was significantly higher than control and increased with the treatment concentration. This indicates that 1-MCP may reduce the resistance of Dangshan pear to Penicillium and increase the disease [22]. Jiang et al. found that the effect of 1-MCP treatment on strawberry fruit was related to the treatment dose: low doses of 1-MCP treatment inhibited rot, while high doses of 1-MCP treatment promoted fruit rot occurrence [13]. Similarly, the 0.5 μL/L 1-MCP-treated group in "Emerald" pear reduced the fruit decay rate, and the fruit decay rate (mainly fungal diseases) in the 1.0 μL/L-treated group was higher than that of control group and the 0.5 μL/L-treated group. The results showed that the high concentration of (1.0 μL/L) 1-MCP treatment aggravated the occurrence of fruit fungal disease in "Emerald" pear [72].

Taken together, 1-MCP can enhance fruit resistance or reduce fruit resistance, which depends on different fruit species and treatment concentration. The reason for reducing resistance is that in some fruits, ethylene participates in fruit defense reaction induced by fungi or bacteria, and if 1-MCP combines with the ethylene receptor, ethylene cannot participate in the fruit defense reaction, eventually leading to more serious disease. Therefore,

when inducing resistance with 1-MCP, we should pay attention to determine the effect of 1-MCP on disease resistance in different fruit species and varieties and optimize the concentration of 1-MCP.

3. The Role of SA in Induced Resistance of Postharvest Fruits

3.1. Effect of SA on Induced Resistance and Its Involved Mechanism

Salicylic acid (SA) is an endogenous phenolic substance with low content in higher plants. As an intracellular signaling molecule, SA can regulate important metabolic processes in plants, such as inducing flowering, affecting sex differentiation, and regulating photoperiod [73]. SA plays a role in the plant resistance response to both biotic and abiotic stresses. Exogenous SA can induce resistance to fungi, bacteria, and viruses in a variety of plants, including tobacco, cucumber, tomato, potato, pea, wheat, rice, and *Arabidopsis*, which has been identified as a chemical inducer of systemic acquired resistance [74]. For example, Mandal et al. found that exogenous application of 200 μM salicylate by root feeding and leaf spray induced resistance to *Fusarium oxysporum* in tomato [75]. Exogenous SA significantly inhibited the expansion of *Penicillium expansum* during room-temperature and low-temperature storage, reduced the weight loss rate, maintained total soluble solid and titrate acid content, and enhanced preservation effect in apple fruit [76]. SA could not only produce allergic reactions (hypersensitive, HR) and systemic acquired resistance (SAR) in plants but also activate a series of important components of defense response signal transduction process after pathogen infection [77]. Different concentrations of SA induced disease resistance. Apple (*Malus domestica*) fruits were treated with 2.5 mmol/L SA and were then soaked and inoculated with penicillium fungi (*Penicillium expansum*), which could effectively inhibit the occurrence of blue mold [76,78]. Further, 3 mmol/L SA treatment reduced the occurrence of citrus (*Citrus reticulata*) fruit disease to some extent; when the SA concentration was higher than 6 mmol/L, the growth of citrus blue mold (*Penicillium digitatum*) and green mold (*Penicillium italicum*) was completely inhibited [79,80]. SA at a concentration of 2 mmol/L or higher could significantly inhibit Alternaria-induced postharvest rot and trigger defense-related mechanisms (such as enhancement of phenlypropanoid metabolism and stimulation of Chitinase and β-1,3-glucanase) in jujube fruit [81] (Table 2). Treatment with SA can enhance the production of multiple defense response mechanisms, including various reactive oxygen species, metabolism of phenols, metabolism of membrane lipid, defense-reaction-related enzymes, and pathogenesis-related (PR) protein, thus improving fruit disease resistance.

Table 2. Studies on the induced resistance effect of SA treatment to different type of fungus in postharvest fruits.

Fruits	Treatment Concentration	Fungi	Reference
Apricot	1 mmol/L	*Alternaria alternata*	[82]
Jujube	2 mmol/L	*Alternaria alternata*	[81]
Tomato	2 mmol/L	*Botrytis cinerea*	[83]
Banana	2 mmol/L	*Colletotrichum musae*	[84]
	2 mmol/L	*Colletotrichum gloeosporioides*	[85]
Mango	1 mmol/L	*Colletotrichum gloeosporioides*	[86]
	5 mmol/L	*Colletotrichum gloeosporioides*	[87]
Carambola	1 mmol/L	*Colletotrichum gloeosporioides*	[88]
Tomato	200 μmol/L	*Fusarium oxysporum*	[76]
Apple	0.2 mmol/L	*Glomerella cingulata*	[89]
Cherry	2 mmol/L	*Monilinia fructicola*	[90]
Apple	0.3 mmol/L	*Penicillium expansum*	[91]
Peach	0.05 mmol/L	*Penicillium expansum*	[83]
Citrus	2.5 mmol/L	*Penicillium expansum*	[92]
Apple	2.5 mmol/L	*Penicillium digitatum*	[76,78]
Citrus	3 mmol/L	*Penicillium digitatum*	[79]

Table 2. *Cont.*

Fruits	Treatment Concentration	Fungi	Reference
Grapefruit	2 mmol/L	*Penicillium digitatum*	[93]
Pear	0.2 mmol/L	*Physalospora piricola*	[94]
Peach	5 mmol/L	*Rhizopus stolonifer*	[80]

3.2. Effect of SA on Reactive Oxygen Metabolism

Superoxide dismutase (SOD) and catalase (CAT) are two enzymes closely related to reactive oxygen species metabolism. SA can improve the activity of the fruit superoxide dismutase (SOD), reduce the rate of production of the superoxide anion, and reduce the damage caused to the fruit. SODs act as the first line of defense to scavenge the superoxide free radicals present in fruits so that SOD activity and superoxide free radical content reach a certain balance. When the fruit is susceptible to disease, the superoxide radical content in the fruit increases, and the SOD activity also increases at this time, achieving the effect of scavenging free radicals. In addition, SOD is related to the synthesis of lignin and antiviral substance, which could enhance the resistance of fruits to various pathogenic microorganisms. CAT plays an important role in plant defense, stress response, and the control of the redox balance of cells. The main function of CAT is to catalyze the decomposition of H_2O_2 in the fruit into H_2O and O_2 and remove the hydrogen peroxide in the body so as to protect the fruit from the poisoning of H_2O_2. Similarly, ascorbate peroxidase (APX), mainly existing in the chloroplast, is the key enzyme for scavenging H_2O_2 in the chloroplast. After SA treatment, the activity of SOD, CAT, and APX enzymes was improved, and the excess reactive oxygen species were eliminated, so the reactive oxygen species in the fruit were in balance, and the resistance of the fruit to pathogens was enhanced. Gao et al. found that 0.2 mmol/L salicylic acid solution effectively improved the SOD enzyme activity and induced enhanced resistance to ring rot disease [94]. SA treatment improved the activity of SOD, POD, CAT, and APX in *Murraya paniculata* fruit; slowed down the production rate of superoxide anion (O_2^-); and reduced the cell membrane permeability [94]. Likewise, 0.01 g/L SA improved the activity of SOD and CAT while reducing the content of malondialdehyde (MDA) in chestnut [95]. Similarly, SA treatment increased the activity of SOD, POD, CAT, and APX and reduced the production rate of O_2 and MDA content in sugar apple fruit [85]. However, some studies have also shown that SA treatment could inhibit CAT activity in fruits and vegetables and increase H_2O_2 content [96–100]. Treatment of 0.3 mmol/L SA significantly induced the increase of superoxide anion and H_2O_2 content in peach fruit, increased the activity of SOD, and enhanced the resistance of apple fruit to *Penicillium expansum* [91]. Zhu et al. found that exogenous SA treatment induced H_2O_2 accumulation in citrus, showing that H_2O_2 as an important messenger molecule could induce enhanced cell wall resistance and induce the biosynthesis of plant disease resistance-related substances and the expression of defense-related genes [101].

3.3. Effects of SA on Membrane Lipid Metabolism

In addition, SA treatment may also alter the membrane lipid permeability of harvested fruit, resulting in improved disease resistance of the fruit. Compared with the control fruit, SA treatment reduced fruit disease index and pericarp cell membrane permeability in *P. longanae*-inoculated longans [102]. Furthermore, treatment with SA decreased activities of phospholipase D (PLD), phospholipase C (PLC), lipase, and lipoxygenase (LOX); lowered the content of saturated fatty acids (SFAs), phosphatidic acid (PA), and diacylglycerol (DAG); but suppressed the reductions in phosphatidylcholine (PC), phosphatidylinositol (PI), unsaturated fatty acids (USFAs), the ratio of USFAs to SFAs (U/S), and the index of unsaturated fatty acids (IUSFA) in the pericarp of *P. longanae*-inoculated longans. Together, these data demonstrated that SA treatment was able to retain the integrity of membrane

structures, enhance fruit disease resistance to *P. longanae*, and thus suppress disease development in *P. longanae*-inoculated longans during storage.

3.4. Effect of SA on Phenolic Metabolism

Similar to the mechanism of 1-MCP-induced resistance, salicylic acid can improve disease resistance by changing the phenolic metabolism inside the fruit. Three enzymes are closely related to phenolic metabolism, including POD, PPO, and PAL. Zeng et al. found that the activity of PAL, POD, and PPO was significantly enhanced after soaking mango fruits in SA [86]. Compared with control, PPO activity during the whole storage period (16 d) increased, PAL activity increased by 5 times at 4 d, and the resistance of mango fruit to anthrax was enhanced. After inducing tomato plants with SA, THE PAL, CAT, POD, and PPO activity in tomato leaves showed a trend of rising firstly and then decreasing and then was significantly higher than control, and the resistance of tomato to powdery mildew was improved [103]. SA treatment of tomatoes inoculated with gray mold pathogens could induce an increase in the MDA content, increase ascorbate levels and POD and PPO enzyme activity, and improve disease resistance of carambola [88]. An optimal concentration of 1.5 mM SA was used to treat chickpea, and the treated fruits showed higher induction of POD and PPO activity in addition to higher phenolics, H_2O_2, and protein accumulation, which ultimately induced plant defense [104]. Three citrus species, including "Kinnow", "Meyer" lemon, and "Mosambi", treated with salicylic and jasmonic acid, were infected with green mold (*Penicillium digitatum*) and blue mold (*P. italicum*). The activity of PPO and POD was proportional to the concentration of salicylic and jasmonic acid applied, the development of both mildew was inhibited, and the resistance of the three citrus fruits was improved [92]. It has also been found that SA combined with ultrasound treatment could improve PAL, POD, CHI, and GLU activities in mango pulp tissue; promote total phenol accumulation; and effectively inhibit mango postharvest anthrax [87]. Treatment of apricot fruits with SA spray before harvest could significantly reduce the incidence of fruit blackspot. The activity of phenylpropane metabolism key enzymes is increased, thus enhancing the resistance of apricot fruit to postharvest blackspot [82].

3.5. Effect of SA on Disease Resistance-Related Signaling Synthesis and Signaling Pathways

The SA-mediated disease resistance signaling pathway contains many genes. At present, most studies in postharvest fruits have found that the expression of *PR-1*, *PR-2* (encoding chitinase), and *PR-4* (encoding β-1,3 glucanase) are directly related with resistance, but more in-depth research is still relatively rare. Earlier studies in rice found that OsBWMK1 localized in the nucleus mediated *PR* gene expression by activating the *OsEREBP1* transcription factor [105]. It was also found that OsBWMK1 phosphorylates OsWRKY33, a WRKY transcription factor with WRKY domain (the amino acid motif WRKYGQK). The DNA binding activity of OsWRKY33 with W-box element (TTGACCA) of several *PR* gene promoters was enhanced. [106]. In postharvest banana fruit, SA and methyl jasmonate (MeJA) treatments significantly increased the content of endogenous SA and JA and resulted in higher expression levels of *MaWRKYs*, *MaPR1-1*, *MaPR2*, *MaPR10c*, *MaCHI3*, *MaCHI4*, and *MaCHIL1*. Yeast one-hybrid analysis showed that MaWRKYs could bind to the promoters of four SA and MeJA-inducible *PR* genes, i.e., *MaPR1-1*, *MaPR2*, *MaPR10c*, and *MaCHIL1*. This indicated that SA and MeJA treatment activated the banana *PRs* and *WRKYs* genes as well as the WRKY TFs bound to the *PR* promoter to induce resistance to anthracnose [85]. It was also found that *MaNAC1, MaNAC2*, and *MaNAC5* were up-regulated after *Colletotrichum musae* infection and were also significantly enhanced by SA and MeJA treatment in banana fruit. MaNAC5 cooperates with MaWRKY1 and MaWRKY2 to induce the expression of several *PR* genes [84]. Treatment of peach fruits infected with *Rhizopus stolonifer* with β-aminobutyric acid (BABA) significantly increased peach fruit resistance. This was an integrated defense response including an H_2O_2 burst, ABA accumulation, and callose deposition. Yeast two-hybrid, luciferase complementation imaging, and co-immunoprecipitation assays showed that MADS2 was an interacting

partner with NPR1. MAPK1 also participated in post-translational modification of MADS2 for signal amplification. MADS2 mediated SA-dependent *NPR1* activation to positively regulate BABA-elicited defense in peach [107]. These are relatively new discoveries in recent years, extending our understanding of transcriptional regulation associated with induction of pathogen resistance in economic fruit crops.

3.6. Effect of SA on Disease Resistance-Related Defense Enzymes and Proteins

Most reports found that the enzymes related to fruit resistance are basically chitinase and β-1,3 glucanase. Treatment with SA improved the activity of chitinase and β-1,3 glucanase, induced pathogenesis-related proteins, and enhanced resistance in eggplant [108]. The combination of SA and chitosan increased chitinase and β-1,3 glucanase activities, effectively activating disease resistance to green mold in grapefruit [93]. SA and chitosan treatment of grape fruit also effectively enhanced the activity of chitinase and β-1,3 glucanase, improving the resistance of grape fruit to gray mildew [109]. Treatment with SA and ultrasound effectively inhibited Penicillium-induced caries, enhancing the activity of defense enzymes such as chitinase and β-1,3 glucanase, as well as increasing disease resistance in peach fruit [83].

The relationship between *PR-1* and SA has been mentioned in the above paragraph. Many reports suggested that SA treatment could affect *PR* gene expression and induce fruit resistance. For example, SA significantly reduced the occurrence of anthrax; increased endogenous SA accumulation; increased *MaPR1-1*, *MaPR2*, and *MaPR10c* gene expression; and enhanced the resistance in banana fruit [110]. Treatment of tomato with SA and Ca^{2+} caused a significant increase in the expression of *PR* gene, resulting in increased resistance to Penicillium and gray leaf spot [111,112]. SA and JA jointly increased the transcription levels of *PR-1*, *PR-6*, and genes encoding isoperoxidase (*M21334*) and increased the resistance to advanced blight in potato (Sorokan et al. 2014). SA induced expression of five *PR* genes, including *PR-1*, *PR-2* (β-1,3 glucanase), *PR-4*(chitinase), *PR-5*, and *PR-8*, and enhanced resistance to leaf spot caused by *Glomerella cingulata* in apple [89].

4. 1-MCP and SA Synergistically Reduced the Fruit Disease

Since both 1-MCP and SA can increase the resistance effect of harvested fruit and reduce the occurrence of disease, can their combined treatment affect the resistance of harvested fruit? A 5 mM SA and 1-MCP compound treatment decreased the rate of ethylene production and incidence of flesh browning, increased texture attributes and reduced the red color of the skin, and enhanced the resistance in postharvest "Laetitia" plum fruits. Compound treatment with 1-MCP and SA in "Campbell early" table grapes increased both firmness and titratable acidity compared to control and single treatment and effectively alleviated stem browning and berry rot during the 16-day storage period of 19 °C [113]. Xu et al. found that combined 1-MCP and SA treatment inhibited the rate of respiration, ethylene production, decay incidence, MDA content, soluble sugars, and soluble solids content. The combination treatment also delayed the sweetness and color changes compared to the untreated bananas, effectively increased the activity of SOD and CAT, and inhibited the increase in POD activity in banana [114]. The compound treatment enhanced the disease resistance of bananas and reduced the occurrence of disease better than 1-MCP or SA alone. SA combined with 1-MCP treatment could effectively inhibit the occurrence of decay rate and weight loss rate in plums during storage; maintain good fruit firmness, total soluble solid, titrable acid, and ascorbic acid (VC) content; inhibit the increase of malondialdehyde and H_2O_2 content; improve the activity of peroxidase, superoxide dismutase, and catalase; and reduce O^{2-} production rate [115].

Although the compound treatment of 1-MCP and SA is better than single treatment in some fruits, the internal mechanism is still not very clear. The above-mentioned papers gave explanations for fruit firmness, phenols, resistance-related enzymes, and so on. In plants, there are primarily two types of induced resistance: systemic acquired resistance (SAR) and systemic induced resistance (ISR), which mostly interact antagonistically. In

our opinion, after compound treatment, 1-MCP competes for ethylene binding sites; the ISR pathway is inhibited, its antagonism to SAR is alleviated, and the SAR pathway is further induced by SA, both reaching a certain equilibrium point perhaps coupled with the increase of endogenous SA. This approach strongly induces the synthesis of disease resistance-related protein PR and enhances the signal expression of the defense response, jointly improving the fruit resistance. On the other hand, it was also found that SA treatment inhibited ethylene production by reducing ACC synthase (ACS) and ACC oxidase (ACO) activities [116]. This indicates that 1-MCP and SA together reduce the content of ethylene after compound treatment. In brief, salicylic-acid-, ethylene-, and jasmonic-acid-mediated fruit disease resistance signals interact. 1-MCP combined with SA is better than a single treatment, possibly due to the synergy between 1-MCP and SA in the induction of disease resistance. 1-MCP inhibits ethylene signaling, while ethylene and SA are antagonistic in disease resistance signaling, and compound treatment reduces ethylene content, reversely inducing SA production, enhancing SAR resistance pathway response, and ultimately improving fruit disease resistance. However, the mechanism of which we speculate still needs to be verified experimentally.

5. Conclusions and Prospect

Postharvest fungal diseases in fruit have received increased attention from researchers in different fields, mainly including horticulture, plant protection, and food science. Synthetic fungicides, which have discernable health or environmental risks, are still the main method used for the control of decay incidence in today's fruit warehouses. Although many non-chemical treatments, primarily including biological control agents, natural compounds, UV, ultrasound, irradiation, hot water and electrolyzed water treatment, and salts, have been used for the control of fungal diseases in fruits after harvest, these diseases still lead to enormous economic losses worldwide every year. In recent years, more chemicals have been used to improve the resistance of various harvested fruits and reduce the occurrence of disease. Induced resistance helps fruits maintain the energy level to respond to the attack of different fungi. Induced resistance is also considered to be a sustainable strategy to deal with the rigorous food safety standards. Increasing evidence shows that the use of safe and healthy chemical treatment is crucial for the development of new and safer strategies to continuously manage postharvest fruit decay. 1-MCP and SA are effective, safe, and harmless, so the role of 1-MCP and SA in induced resistance of postharvest fruits are reviewed, including the synergetic resistance between 1-MCP and SA, reactive oxygen metabolism, membrane lipid metabolism, phenolic metabolism, disease resistance-related defense enzymes and proteins, and genes related to signaling synthesis and signaling pathways (Figure 1).

Transcriptional regulation mechanisms are very important as the basis for 1-MCP and SA to indirectly or directly affect disease resistance in postharvest fruits. 1-MCP induced fruit resistance by affecting the ethylene signal transduction and ISR pathway, and SA induced fruit resistance through the SAR resistance pathway. However, the mechanism of induced resistance in postharvest fruits is basically the study of physiological defense response-related mechanisms and disease resistance-related proteins at present. How is the ISR pathway regulated by 1-MCP in order to induced resistance? Since there is report about the induction of the expression of *PR1* by 1-MCP [58], and *PR1* is a marker gene of SAR, does 1-MCP treatment regulate the SAR pathway? The crosstalk model between the ISR and SAR pathways in postharvest fruits is still unknown. We should pay much attention to the molecular mechanism of induced resistance by using 1-MCP and SA treatment, especially starting with the ISR and SAR pathways.

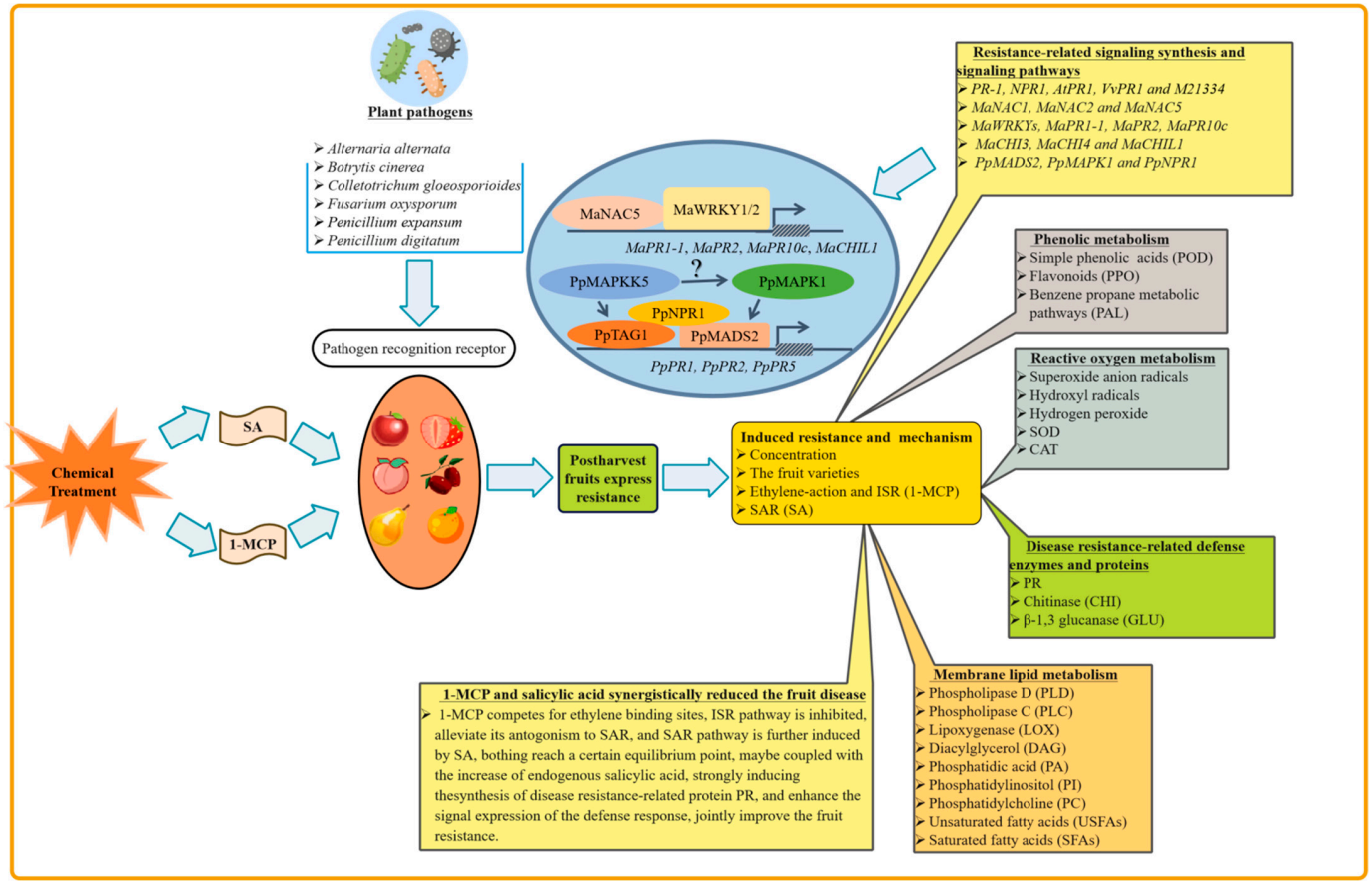

Figure 1. The induced resistance of 1-MCP and SA in postharvest fruits.

Fruits with 1-MCP or SA treatment alone show increased resistance to pathogen infection, and recent studies provide new insights into improving self-resistance by the treatment of 1-MCP in combination with SA. We should apply this compound treatment technology to preserve a wider variety of fruits and especially to improve the resistance of fruits. The preservation technique in combination with 1-MCP and SA may be a promising approach to extend shelf life. In order to explore the mechanism of the synergistic effect of 1-MCP and SA in fruit resistance, the transcriptional regulation mechanism should particularly be further studied, and new target genes for inducing resistance should be found, which will provide theoretical guidance for molecular breeding and postharvest processing.

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