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Induced Resistance in Fruits as an Eco-friendly Strategy for Postharvest Disease Management: A Review

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Abstract

Postharvest diseases cause substantial losses in fruit production worldwide, with fungal pathogens being the primary causal agent. Traditional control methods rely heavily on chemical fungicides, which pose environmental and health concerns while promoting pathogen resistance. Induced resistance offers a sustainable alternative by activating the fruit's natural defense mechanisms rather than directly targeting pathogens. This review examines two main types of induced resistance: Systemic Acquired Resistance (SAR), triggered by chemical treatments and providing long-lasting protection, and Induced Systemic Resistance (ISR), activated by beneficial microorganisms with faster response times. Various elicitors can trigger these defenses, including physical treatments (UV-C light, heat, modified pressure), chemical compounds (salicylic acid, jasmonic acid, brassinosteroids), and biological agents (chitosan, harpin, oligandrin, beneficial microbes like *Bacillus, Pseudomonas, Trichoderma*, and antagonistic yeasts). Each elicitor category offers unique advantages for disease management. Application methods such as dipping, spraying, and coating enable practical implementation in commercial systems. While challenges exist regarding timing, formulation stability, and variability across fruit types, combining different elicitors and integrating them with other control strategies shows promise. As regulations tighten and consumer demand for safer produce grows, induced resistance is emerging as a key component of sustainable postharvest disease management.

***Key words*:** Postharvest, Disease, Systemic acquired resistance, Induced systemic resistance, Elicitors

Postharvest diseases pose significant challenge to fruit production worldwide, leading to extensive economic losses and decreased food security. In countries like India, which ranks second globally in fruit production, approximately 30-40 percent of fruits and vegetables are lost between harvest and consumption [34]. These losses are primarily due to fungal, bacterial, and viral infections that affect the quality, shelf life, and marketability of fruits. The scale of these losses represents not only a substantial economic burden for producers and suppliers but also contributes to global food waste concerns and threatens the availability of nutritious food for growing populations. Among the various pathogens, fungal pathogens play the major role in causing significant postharvest losses. Fungi such as *Botrytis cinerea* (grey mould), *Penicillium* (blue mold), *Colletotrichum* (anthracnose), *Monilinia fructicola*, *Alternaria*, *Aspergillus*, *Fusarium*, *Mucor*, and *Rhizopus* are among the most important ones causing various rots and decays of fruits and infect fruits during harvesting, handling, or storage [78]. Each of these pathogens has distinct characteristics and infection strategies. For instance, *Botrytis cinerea* thrives in cool, humid conditions and can spread rapidly through fruit lots, while *Aspergillus* species are particularly concerning due to their ability to produce harmful mycotoxins that pose serious food safety risks. The diversity of these pathogens and their varying environmental requirements make disease management particularly challenging in commercial postharvest systems.

Conventional methods for controlling postharvest diseases heavily rely on synthetic chemical fungicides [45], but their excessive use leads to fungicide resistance in pathogens, environmental pollution, and health risks for consumers. The development of fungicide-resistant pathogen strains has become increasingly common, with some populations showing resistance to multiple fungicide classes simultaneously. Furthermore, consumer awareness of pesticide residues on fresh produce has grown substantially, driving demand for residue-free or organically produced fruits. Regulatory agencies worldwide have responded by implementing stricter limits on fungicide residues and withdrawing certain active ingredients from approved use. These factors collectively highlight the urgent need for alternative, eco-friendly approaches to enhance the natural defense of fruits against pathogens.

One such promising strategy is induced resistance (IR), a phenomenon where plants develop an enhanced defensive capacity against pathogens following exposure to certain biotic or abiotic stimuli [45]. By harnessing the plants' immune system, induced resistance offers a sustainable solution for managing postharvest diseases. Unlike direct pathogen-targeting treatments, IR strengthens the plants' ability to resist

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or mitigate infections by activating defense-related enzymes, secondary metabolites, and antimicrobial proteins [53]. There are mainly two types of induced resistance- Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR). Both of them are similar phenotypically but they vary in their mechanisms. This distinction is important because it reveals that plants have evolved multiple pathways to achieve induced resistance, each optimized for different types of threats and environmental conditions. Understanding the differences between SAR and ISR helps in selecting appropriate resistance-inducing treatments for specific fruit-pathogen combinations and storage conditions. Various physical, chemical, and biological methods can trigger defense mechanisms against diseases in postharvest fruits and vegetables. Treatments or substances that activate these protective responses in fruits and vegetables are referred to as inducers or elicitors [46]. Physical elicitors like ultraviolet-C (UV-C) irradiation, thermal treatments, and manipulation of atmospheric pressure through hypobaric or hyperbaric storage, have been demonstrated to effectively reduce postharvest decay and extend the storage life of fruits [36], [40], [51], [59]. Commonly used chemical elicitors include salicylic acid, jasmonic acid, brassinosteroids, benzothiadiazole, 2,6 dichloronicotinic acid [62]. Microbial effectors like harpin, oligandrin, chitosan and biocontrol agents like antagonist yeasts, *Bacillus* and *Pseudomonas* are also used as elicitors [79], [11]. All these elicitors suppress postharvest pathogens through both direct and indirect modes of action. Directly, they inhibit pathogen growth, spore germination, or cell integrity by disrupting membranes or metabolic processes. Indirectly, elicitors activate host defense mechanisms, including the induction of pathogenesis-related proteins, antioxidant enzymes, and phenolic compounds, thereby enhancing systemic resistance against infection. This approach represents a fundamental shift from attempting to directly kill pathogens to empowering the fruit itself to defend against infection. In this context, the present review critically summarizes induced resistance in postharvest systems, its types with emphasis on the major classes of elicitors used in fruits.

Physiology of fruit development and postharvest diseases

Fruits require a complex set of interacting genes and signaling pathways for proper development. Fruit growth and maturation include three separate stages: (a) fruit set, (b) development, and (c) ripening and senescence [24]. Understanding these developmental stages is crucial for comprehending how fruits become susceptible to postharvest diseases and how resistance mechanisms can be activated.

The fruit set stage begins after successful pollination and fertilization, when the ovary starts to develop into a fruit. During this initial phase, cell division is the primary growth mechanism, establishing the basic structure and cell number that will determine final fruit size. Hormonal signals, particularly auxins and gibberellins, play critical roles in initiating and maintaining fruit development. This stage is characterized by high metabolic activity and rapid tissue expansion [20]. The development stage involves both continued cell division and, more prominently, cell expansion through water uptake and accumulation of storage compounds. During this period, fruits accumulate starches, organic acids, and structural compounds while maintaining relatively high levels of defensive compounds. The fruit epidermis develops its protective cuticle layer, and various anatomical features that contribute to disease resistance are established. At this stage, fruits generally exhibit robust resistance to pathogen infection due to high levels of antimicrobial compounds, firm tissue structure, and active defense mechanisms. Of these three stages, the ripening process triggers a complex set of biochemical pathways that make fruit attractive, desirable, and edible to consumers but at the same time susceptible to pre and postharvest diseases. Ripening represents a highly coordinated developmental program involving changes in color, texture, flavor, and aroma. The process is regulated by complex hormonal interactions, with ethylene playing a central role in climacteric fruits such as apples, bananas, and tomatoes, while non-climacteric fruits like strawberries and citrus rely more heavily on abscisic acid signaling. During ripening, chlorophyll breaks down while other pigments such as carotenoids and anthocyanins accumulate, giving fruits their characteristic colors. Cell wall polysaccharides are degraded by enzymes like polygalacturonase and pectinase, causing tissue softening. Starch converts to simple sugars, organic acids decline, and volatile compounds that contribute to characteristic fruit aromas are synthesized. These changes make fruits more palatable and nutritious but simultaneously increase their vulnerability to pathogen attack [6].

Relationship between ripening and disease susceptibility

Postharvest diseases frequently do not show symptoms during the early stages of fruit set and development but only during the period of ripening. This phenomenon is known as quiescent or latent infection, where pathogens establish initial infections during early fruit development but remain dormant until conditions become favorable for disease development [47], [67]. Fungal infections that occur during flowering and early fruit development commonly become quiescent (dormant) until ripening due to fruit's innate capacity to resist pathogen challenge. The mechanisms maintaining quiescence include high concentrations of preformed antifungal compounds, low pH that inhibits fungal enzyme activity, firm tissue structure that resists pathogen penetration, and active defense responses that limit pathogen growth. During fruit ripening, components of the plant immune system gradually lose either their effectiveness or the ability to activate resistance processes, which happens concurrently with a reduction of defense hormone production and signaling and downstream transcriptional responses. Salicylic acid levels, which are associated with defense responses, typically decline during ripening, while ethylene production increases to promote ripening processes. This hormonal shift can suppress certain defense pathways while activating ripening-related genes. Additionally, the expression of genes encoding antimicrobial proteins and enzymes involved in defensive compound synthesis decreases during ripening and senescence. All these processes can affect the fruit's capability to respond to the process of induced resistance and prevention of fungal infection [48]. The declining defensive capacity during ripening means that resistance-inducing treatments must be carefully timed and optimized to achieve maximum effectiveness. Treatments applied too early may lose their effectiveness by the time fruits reach full ripeness, while treatments applied too late may be insufficient to protect fruits that have already entered advanced stages of ripening. Understanding the intricate relationship between fruit physiology and disease susceptibility is therefore essential for developing effective induced resistance strategies in postharvest systems.

Induced resistance

The phenomenon of Induced Resistance (IR), where plants develop an increased resistance to pathogen attack following localized pathogen infection, has been recognized for over 100 years. Early observations by researchers noted that plants that survived disease outbreaks often showed enhanced

resistance to subsequent infections, suggesting the existence of an inducible immune memory. However, scientific investigation of this phenomenon only gained momentum in the latter half of the 20th century with advances in plant pathology and biochemistry.

The first empirical evidence for biological activation of IR was presented by Ross [54] who demonstrated that localized inoculation of tobacco leaves with tobacco mosaic virus (TMV) resulted in the development of resistance to TMV in distal non-inoculated leaves. This landmark study established that resistance could be induced systemically throughout the plant following local stimulus, introducing the concept of systemic resistance. Ross's work opened new avenues of research into the mechanisms underlying this phenomenon and its potential applications in disease management.

Induced resistance is the phenomenon in which a plant, once appropriately stimulated, exhibits an enhanced resistance upon "challenge" inoculation with a pathogen [66]. This definition emphasizes several key features of induced resistance. First, it requires an initial stimulus or priming event that prepares the plant for subsequent pathogen challenge. Second, the enhanced resistance manifests only after this priming, distinguishing it from constitutive resistance that exists without prior stimulation. Third, the resistance is typically broad-spectrum, protecting against multiple pathogens rather than being specific to a single disease. Fourth, the induced state can persist for extended periods, ranging from days to weeks depending on the plant, stimulus, and environmental conditions. Pathogen infection at low intensity, avirulent pathogens, certain non-pathogenic bacteria, and certain chemicals can be used to stimulate the plants [75]. Low-intensity pathogen infection provides genuine pathogen-associated molecular patterns that trigger defense responses without causing significant disease. Avirulent pathogens lack the full complement of virulence factors needed to cause disease but retain the molecular signatures that activate plant immunity. Non-pathogenic bacteria, particularly those that colonize plant surfaces and promote plant growth, can stimulate resistance through various mechanisms including competition with pathogens and production of defense-inducing compounds [22].

Types of induced resistance

There are mainly two types of induced resistance - Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR). Both of them are similar phenotypically but they vary in their mechanisms. This distinction is important because it reveals that plants have evolved multiple pathways to achieve induced resistance, each optimized for different types of threats and environmental conditions. Understanding the differences between SAR and ISR helps in selecting appropriate resistance-inducing treatments for specific fruit-pathogen combinations and storage conditions.

SAR and ISR share several common features that define them as forms of systemic induced resistance. Both involve systemic signaling that spreads from the site of stimulus to distant tissues, both result in priming of defense responses so that subsequent pathogen challenges elicit faster and stronger defenses, both provide broad-spectrum protection against multiple pathogen types, and both can persist for extended periods after the initial inducing stimulus. However, the molecular mechanisms, signaling pathways, and types of defenses activated differ significantly between these two forms of resistance [56]. The phenotypic similarity between SAR and ISR means that both result in enhanced resistance to pathogen infection, reduced disease severity when infection occurs, and activation of similar defense mechanisms including antimicrobial compound production and defense enzyme activation. However, molecular analysis reveals that different signaling pathways and regulatory genes control these two types of resistance, leading to subtle differences in the timing, magnitude, and specificity of defense responses.

Systemic acquired resistance

Systemic acquired resistance (SAR) is a form of induced resistance that is activated throughout a plant after being exposed to elicitors from virulent, avirulent, or non-pathogenic microbes, or artificial chemical stimuli such as chitosan or salicylic acid [12]. SAR is characterized by several distinctive features that make it particularly valuable for disease management. It develops slowly over several days following the inducing stimulus, allowing time for the establishment of defense mechanisms throughout the plant. Once established, SAR provides long-lasting protection that can persist for weeks or even months, depending on the plant species and environmental conditions.

Systemic acquired resistance (SAR) provides enhanced, long-lasting systemic immunity to secondary infection by a range of biotrophic, hemibiotrophic, and necrotrophic pathogens that have diverse modes of infection. This broad-spectrum protection is one of the most valuable features of SAR, as it means that a single inducing treatment can provide protection against multiple diseases simultaneously. The effectiveness of SAR against different pathogen lifestyles reflects the activation of multiple defense mechanisms that target various aspects of pathogen biology. Against biotrophic pathogens that require living host cells, SAR activates hypersensitive cell death responses that deprive the pathogen of living tissue. Against necrotrophic pathogens that kill host cells and feed on dead tissue, SAR enhances cell wall fortification and antimicrobial compound production that limit pathogen spread.

Salicylic acid (SA) is widely involved in signal transduction in the induced resistance of fruits and vegetables SA functions as the primary signaling molecule in SAR, and its accumulation is both necessary and sufficient to trigger the development of systemic resistance [37], [4], [29]. The central role of SA in plant immunity has been established through numerous studies showing that plants unable to accumulate SA fail to develop SAR, while exogenous application of SA can induce resistance even without pathogen challenge. SA and its SA analogues benzothiadiazole and 2,6-dichloroisonicotinic acid can induce SAR production in plants [68], [21], [31], [13], [26]. These synthetic analogues were developed to overcome some limitations of SA itself, including its photosensitivity and potential phytotoxicity at high concentrations. Benzothiadiazole (also known as acibenzolar-S-methyl or BTH) has been particularly successful as a commercial product for inducing resistance in various crops. These compounds mimic SA's defense-inducing activity while offering improved stability and reduced negative effects on plant growth and fruit quality.

On application of any elicitor, there is an increased production of salicylic acid in plants and it will catalyze the conversion of transcription factor NPR1 (Non expressor of pathogenesis-related gene 1: key regulator of the systemic acquired resistance pathway in plants) from a polymer to a monomer by activating thioredoxin and transfers it from the cytoplasm to the nucleus. NPR1 exists in the cytoplasm as an inactive oligomer held together by intermolecular disulfide bonds. When SA accumulates in response to elicitor treatment or pathogen challenge, it triggers changes in cellular redox

status that activate thioredoxin enzymes. These thioredoxins reduce the disulfide bonds holding NPR1 oligomers together, releasing active NPR1 monomers that can enter the nucleus. Then, NPR1 interacts with TGACGTCA cis-element-binding protein (TGA) to regulate the expression of downstream defense genes such as the PR gene [30]. In the nucleus, NPR1 monomers serve as transcriptional co-activators by binding to TGA transcription factors. TGA factors recognize and bind to specific DNA sequences in the promoters of defense-related genes, but they require interaction with NPR1 to activate transcription. The NPR1-TGA complex recruits additional proteins involved in chromatin remodeling and transcriptional activation, leading to increased expression of hundreds of defense-related genes. These genes encode antimicrobial proteins, enzymes involved in defensive compound synthesis, signaling proteins that amplify defense responses, and proteins involved in cell wall fortification and other structural defenses. Therefore, nonexpressor of pathogenesis-related genes 1 (NPR1) is considered to be the master regulator of SAR located downstream of SA, which regulates and induces SAR by interacting with transcription factors such as TGA [16]. The central role of NPR1 has been confirmed through genetic studies showing that mutations in NPR1 completely abolish SAR even when SA accumulates normally. Conversely, overexpression of NPR1 can enhance disease resistance even without additional SA accumulation. Understanding the NPR1-mediated signaling pathway has provided valuable insights into how resistance-inducing treatments activate defense responses and has identified potential targets for enhancing induced resistance through genetic or chemical manipulation [15].

The effectiveness of SAR-inducing treatments in postharvest systems depends on several factors. The timing of treatment relative to harvest is critical, as treatments applied too long before harvest may lose effectiveness by the time fruits are actually stored, while treatments applied after harvest may not allow sufficient time for resistance mechanisms to develop before pathogen challenge. The concentration of elicitors must be optimized for each fruit species, as concentrations that are effective for inducing resistance in one species may be insufficient or cause phytotoxicity in another. Storage conditions, particularly temperature, influence both the development of induced resistance and its persistence during storage. One challenge in applying SAR in postharvest systems is that the ripening process itself tends to suppress SA signaling and favor other hormonal pathways such as ethylene signaling. This means that SAR-inducing treatments must overcome the natural suppression of SA responses during ripening. Researchers have explored various strategies to address this challenge, including combining SA treatments with compounds that delay ripening, using higher concentrations of elicitors to overcome suppression, or applying multiple treatments to maintain elevated defense responses throughout storage.

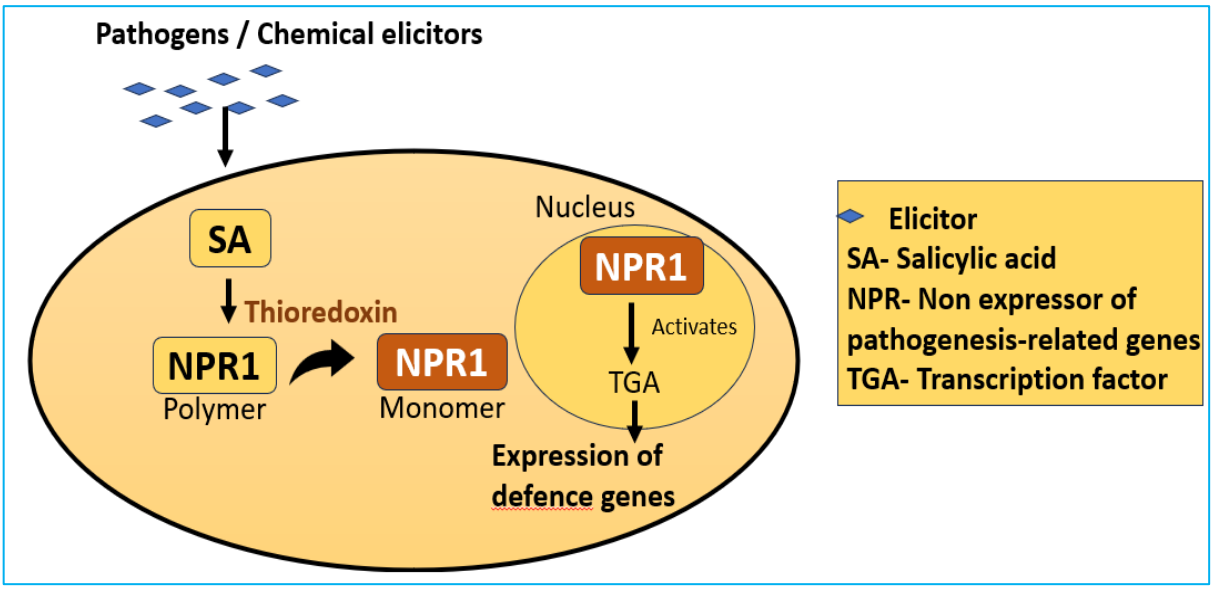

Fig 1 Role of salicylic acid in activating defence mechanisms

Induced systemic resistance

Induced Systemic Resistance is activated by beneficial microbes and depends on jasmonic acid and ethylene signaling pathways [10]. Unlike SAR, which is typically triggered by pathogen-derived signals or chemical elicitors, ISR is specifically associated with colonization of plant roots or tissues by beneficial microorganisms. These beneficial microbes include plant growth-promoting rhizobacteria such as *Pseudomonas* and *Bacillus* species, as well as beneficial fungi like *Trichoderma*. The distinction between SAR and ISR extends beyond the nature of the inducing stimulus to encompass different signaling pathways and defense mechanisms.

ISR typically develops more rapidly than SAR, with enhanced resistance detectable within hours to days following colonization by beneficial microbes. The defense responses associated with ISR are often characterized as "priming" rather than direct activation, meaning that defense mechanisms are prepared for rapid deployment but not fully activated until pathogen challenge occurs. This priming state is metabolically less costly than constitutive activation of defenses, allowing plants to maintain resistance readiness without the growth penalties associated with continuous defense activation.

JA and its volatile ester derivative methyl jasmonates (MeJA) are a class of growth regulators widely existing in plants, which act as signal molecules to induce defense gene expression and enhance the resistance of plants to biotic and abiotic stresses [65]. Jasmonic acid belongs to a family of lipid-derived signaling molecules called oxylipins, which are produced from membrane lipids in response to various stresses. The discovery that JA functions as a defense signal came from studies showing that plants treated with JA become more resistant to insect herbivores and certain pathogens, particularly necrotrophic fungi that kill host tissue during infection.

When plants are stimulated by elicitors, JA is rapidly synthesized in cells, and the synthesized JA combines with

isoleucine to produce active Jasmonic acid isoleucine complex (JA-Ile). The conjugation of JA with isoleucine is catalyzed by the enzyme jasmonate resistant 1 (JAR1) and represents a crucial activation step in jasmonate signaling. JA-Ile is the biologically active form that is recognized by cellular receptors and initiates the signaling cascade. This conjugation step provides a mechanism for regulating signal strength, as the concentration of JA-Ile can be modulated through the balance between synthesis and degradation. JA-Ile interacts with the receptor proteins COI1 and JAZ to form a complex, and then degrades the ubiquitinated JAZ protein through the 26S proteasome. COI1 is an F-box protein that functions as part of an SCF ubiquitin ligase complex. When JA-Ile binds to COI1, it promotes the interaction between COI1 and JAZ proteins, leading to ubiquitination of JAZ proteins and their subsequent degradation by the proteasome. This degradation relieves repression of downstream transcription factors, allowing defense genes to be expressed. Because the JAZ protein can inhibit the activity of the transcription factor MYC2, when JAZ is decomposed, the transcription factor MYC2 is released, activating the JA signaling pathway, thus inducing systemic resistance [9]. MYC2 is a basic helix-loop-helix transcription factor that binds to G-box elements in the promoters of JA-responsive genes. When released from JAZ repression, MYC2 activates transcription of genes encoding enzymes involved in secondary metabolite synthesis, antimicrobial proteins, and additional regulatory proteins that amplify defense responses. The JA signaling pathway also interacts with ethylene signaling, with the two pathways often working synergistically to activate defenses against necrotrophic pathogens.

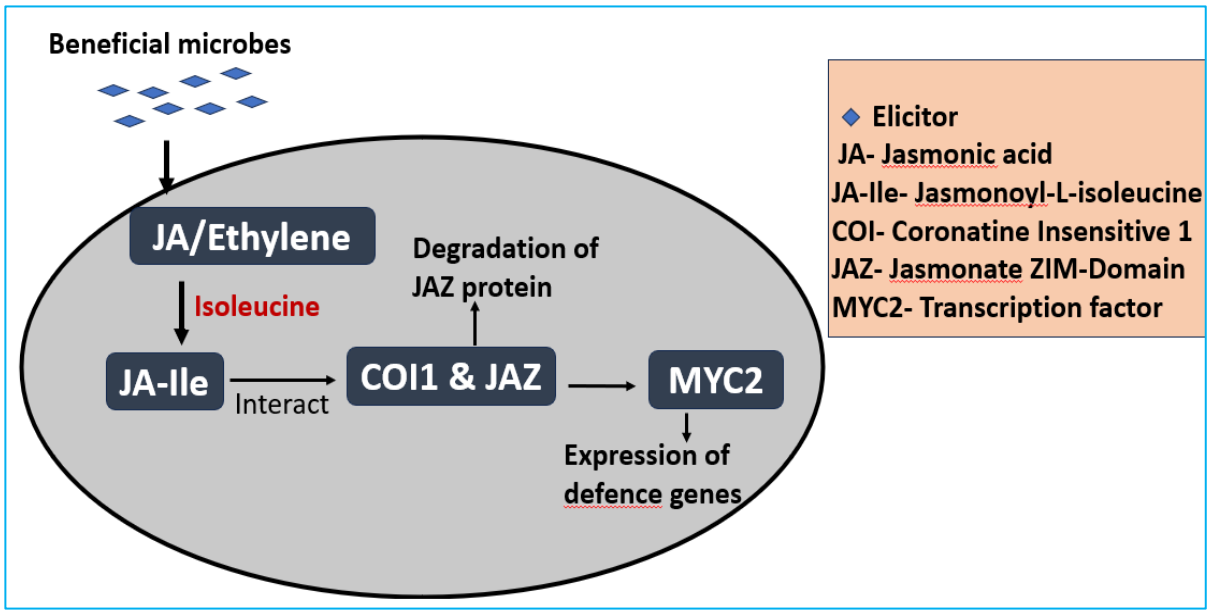

Fig 2 Role of jasmonic acid in activating defence mechanisms

Elicitors

An elicitor is defined as a compound that, in small concentrations, can activate different plant responses, such as endogenous protection responses, including the production of different secondary metabolites [46]. Elicitors initiate SAR or ISR in the host plant. The term "elicitor" derives from the Latin "elicere" meaning "to draw out," reflecting the concept that these compounds draw out or activate the plant's latent defense capabilities. The effectiveness of elicitors depends on their recognition by plant receptors, the strength of the defense response they trigger, and their stability under storage conditions.

These molecules are recognized by receptors present within or on the plant and activate the defense signaling pathways [2]. Pattern recognition receptors on the plant cell surface detect conserved molecular patterns associated with microbes or stress, initiating signaling cascades that lead to defense activation. Some elicitors function by mimicking pathogen-associated molecular patterns, tricking the plant into mounting defense responses in the absence of actual pathogen threat. Other elicitors may work by directly affecting cellular processes that indirectly trigger defense responses, such as causing mild oxidative stress that activates stress-responsive defense mechanisms. They can be divided into physical, chemical, and biological elicitors. This classification is useful for understanding the mechanisms of action and practical considerations for application. Physical elicitors typically work by imposing stress conditions that activate stress-responsive defense pathways. Chemical elicitors function as signaling molecules or metabolic regulators that directly influence defense pathways. Biological elicitors include living organisms and biological molecules that trigger defenses through recognition of pathogen-associated patterns or through production of defense-inducing compounds.

Physical elicitors

Ultraviolet-C (UV-C) light, heat, hypobaric, and hyperbaric treatments are known to be effective in controlling postharvest decay of fruit and vegetables [64]. Physical treatments offer several unique advantages for postharvest disease management that make them attractive alternatives or complements to chemical treatments. The advantage of most of these relies on direct effects on the pathogen without leaving residues on the fruit. This absence of chemical residues is particularly important given increasing consumer concerns about pesticide residues and regulatory restrictions on chemical fungicide use. Physical elicitors may be more cost-effective, particularly in large-scale applications, as they often rely on commonly available equipment (e.g., UV lamps and heating systems). Once the initial infrastructure is established, the operating costs for physical treatments can be relatively low compared to the ongoing costs of purchasing chemical fungicides. Additionally, these can induce several changes in host tissues, including increased resistance to abiotic and biotic stress. The stress-conditioning effects of physical treatments can enhance overall fruit stress tolerance, potentially improving

storage performance beyond just disease control. Physical treatments can be easily integrated into existing postharvest handling systems with minimal modification to established procedures. They do not require specialized training for safe handling of hazardous chemicals, reducing safety concerns for workers. Furthermore, physical treatments do not contribute to the development of pathogen resistance in the same way that chemical fungicides do, as they typically work through multiple mechanisms simultaneously rather than targeting a single biochemical pathway.

UV-C radiation, defined within the range of 100 to 280 nm, promotes various biochemical and physiological responses in fruits. The germicidal properties of UV-C light have been known since the early 20th century, but its application for inducing resistance in fruits is a more recent development. UV-C is the highest energy form of ultraviolet radiation and is completely absorbed by the Earth's atmosphere, meaning plants evolved without regular exposure to UV-C and may lack specific adaptive responses to it. When exposed to UV-C light, plants can activate their defense mechanisms [41], [63], [1]. The mechanisms by which UV-C induces resistance are multifaceted. UV-C causes mild damage to DNA and proteins, triggering stress responses that overlap with defense responses. UV-C exposure leads to production of reactive oxygen species, which function as signaling molecules activating defense gene expression. UV-C also directly affects the phenylpropanoid pathway, leading to accumulation of phenolic compounds with antimicrobial properties. For example, strawberries exposed to UV-C at 0.50 kJ m^{-2} and 1.00 kJ m^{-2} induced resistance against *Botrytis cinerea* causing postharvest decay by increasing their PAL activity 12 h after treatment [39]. This landmark study demonstrated that relatively low doses of UV-C could induce significant resistance without causing visible damage to fruit quality. The increase in PAL activity indicates activation of the phenylpropanoid pathway, leading to enhanced production of phenolic compounds, flavonoids, and lignin precursors that contribute to disease resistance.

Temperature (both high and low) is one of the oldest means to control postharvest diseases of fruit and vegetables. The use of hot water treatments for disease control dates back over a century, initially focused on surface disinfestation to kill pathogen spores on fruit surfaces. However, research over the past few decades has revealed that heat treatments can also induce resistance mechanisms within fruit tissues, providing protection that extends beyond the immediate lethal effects on surface pathogens. Short hot water treatment has been shown to induce resistance to chilling and pathogens in various fruits. Heat treatments typically involve immersing fruits in hot water (typically 45-53°C) for short periods (2-5 minutes) or exposing them to hot air at similar temperatures for longer periods. These treatments are calibrated to impose stress without causing heat damage that would compromise fruit quality. The stress imposed by heat triggers heat shock responses, including production of heat shock proteins that help protect cellular proteins from denaturation and assist in maintaining cellular function under stress conditions. Applications of heat treatments have shown success in various fruit crops. Hot water treatment of mangoes reduces anthracnose disease caused by *Colletotrichum gloeosporioides* by both killing surface inoculum and inducing resistance in fruit tissues [8]. Heat treatment of apples enhances resistance to *Botrytis cinerea, Colletotrichum acutatum* and *Neofabraea vagabunda* infections and extends storage life [14]. In citrus fruits, heat conditioning before cold storage reduces chilling injury and also provides enhanced protection against pathogens [76]. The dual benefits of reducing both abiotic stress (chilling injury) and biotic stress (pathogen infection) make heat treatments particularly valuable in postharvest systems.

Hypobaric or hyperbaric treatments (use of pressure different from atmospheric pressure over a short period of time) can be also used as elicitors. Hypobaric conditions (reduced pressure) create a partial vacuum that reduces oxygen availability, while hyperbaric conditions (elevated pressure) increase oxygen and other gas concentrations around the fruit. Short hypobaric treatment has been shown to be an effective means of control of postharvest decay of strawberries, sweet cherries, and table grapes [51]. Hypobaric storage involves holding fruits under reduced atmospheric pressure, typically 0.1 to 0.3 atmospheres. While the reduced oxygen might be expected to slow pathogen growth, research suggests that the primary benefit comes from induced resistance responses in the fruit tissue.

Chemical elicitors

Chemical elicitors represent a sophisticated approach to activating endogenous defense mechanisms in harvested fruits. These bioactive molecules function by mimicking pathogen-associated signals, thereby priming the fruit's innate immunity without direct antimicrobial action. The strategic deployment of chemical elicitors offers a sustainable alternative to conventional synthetic fungicides, addressing growing concerns about pesticide residues and environmental contamination.

Salicylic acid (SA) stands as one of the most extensively studied phytohormones involved in plant immunity. This phenolic compound orchestrates systemic acquired resistance (SAR) by modulating the expression of defense-related genes and triggering downstream signaling cascades [35], [25]. When applied during critical developmental periods- particularly fruit set and harvest- SA induces physiological adaptations that enhance resistance to biotic stressors. Field applications have demonstrated efficacy against diverse pathogens affecting economically important crops. In mango (*Mangifera indica*), SA treatments significantly reduce anthracnose incidence caused by *Colletotrichum gloeosporioides* [27-28]. Similarly, peach (*Prunus persica*) [70], sweet cherry [74], [72] strawberry [5], [55] and citrus fruits exhibit enhanced resistance to fungal decay following SA application. The optimal concentration typically ranges from 0.5 to 2.0 mM, though this varies depending on the fruit species, cultivar characteristics, and targeted pathogen. However, SA deployment faces inherent limitations that constrain its commercial viability. Excessive concentrations trigger phytotoxic responses, manifesting as tissue browning, altered organoleptic properties, and accelerated senescence. These adverse effects compromise marketability and consumer acceptance. Additionally, SA exhibits pronounced photolability, ultraviolet radiation catalyzes its degradation into inactive derivatives, substantially diminishing field persistence and biological activity. To circumvent these constraints, researchers have developed photostable synthetic analogues that retain SA's defense-eliciting properties while overcoming its inherent instability. Benzothiadiazole (BTH), commercially available as acibenzolar-S-methyl, represents a breakthrough in this domain. This synthetic compound activates SA-dependent signaling pathways without undergoing photodegradation, providing prolonged protection [23]. Similarly, 2,6-dichloroisonicotinic acid (INA) functions as a potent SAR inducer with enhanced stability under field conditions. These analogues have demonstrated efficacy against broader pathogen spectra, including bacterial and viral diseases that prove recalcitrant to conventional SA treatments [18].

Jasmonic acid (JA) and its volatile methyl ester, methyl jasmonate (MeJA), constitute a distinct class of lipid-derived signaling molecules that govern plant responses to mechanical wounding and necrotrophic pathogens. Unlike SA, which primarily mediates defense against biotrophic organisms, jasmonates activate defense pathways effective against postharvest decay fungi that derive nutrients from dead host tissues. The jasmonate signaling network operates through the JAZ-COI1 receptor complex, triggering transcriptional reprogramming that upregulates genes encoding protease inhibitors, secondary metabolite biosynthetic enzymes, and structural proteins. Postharvest applications have yielded promising results across multiple commodities. [38] demonstrated that JA treatments substantially curtailed green and blue mold development in citrus fruits caused by *Penicillium digitatum* and *P. italicum*. The protective effect stems from enhanced activity of polyphenol oxidase and peroxidase. Concentration optimization remains critical for successful jasmonate deployment. Insufficient doses fail to activate defense responses, while excessive concentrations may accelerate ripening and senescence through ethylene-dependent pathways.

Brassinosteroids (BRs) constitute a unique family of plant steroid hormones structurally analogous to animal steroid hormones. These polyhydroxylated compounds regulate diverse physiological processes spanning cell elongation, vascular differentiation, reproductive development, and stress adaptation. Among approximately 70 naturally occurring BRs, brassinolide, castasterone, and 24-epibrassinolide exhibit the highest biological activity. BR perception occurs through the membrane-localized BRI1-BAK1 receptor kinase complex, initiating phosphorylation cascades that ultimately regulate transcription factors controlling hundreds of target genes. This hormonal system uniquely integrates growth promotion with stress tolerance, a feature that distinguishes BRs from other phytohormones that typically exhibit growth-defense trade-offs. In postharvest applications, BRs demonstrate remarkable capacity to simultaneously maintain fruit quality while enhancing disease resistance. [73] reported that BR treatments on jujube fruit (*Ziziphus jujuba*) significantly suppressed *Alternaria alternata* infection while preserving firmness and nutritional content. Similarly, mango fruits treated with 24-epibrassinolide exhibited reduced anthracnose severity alongside delayed ripening and extended shelf life [58]. The mechanisms underlying BR-mediated disease resistance involve multiple pathways. BRs enhance antioxidant enzyme activities, scavenge reactive oxygen species, stabilize membrane integrity, and induce the expression of pathogenesis-related proteins. Notably, BRs can synergize with other phytohormones; combined SA-BR treatments often yield superior protection compared to individual applications, suggesting convergence of distinct signaling networks. Nanoscale BR formulations represent an emerging frontier, enhancing bioavailability and reducing required doses. Nanoencapsulated BRs exhibit improved stability, controlled release kinetics, and enhanced tissue penetration, addressing limitations of conventional aqueous formulations.

Biological elicitors

Biological elicitors encompass diverse molecules of microbial origin that activate plant immunity through recognition of conserved pathogen-associated molecular patterns (PAMPs) or damage-associated molecular patterns (DAMPs). These elicitors offer advantages of low environmental impact, minimal residue concerns, and compatibility with organic production systems.

Harpin proteins represent a family of heat-stable, glycine-rich, cysteine-lacking bacterial effectors initially characterized from *Erwinia amylovora*, the causative agent of fire blight in rosaceous plants. The prototypical HrpN protein exhibits an approximate molecular mass of 44 kDa and possesses unusual biochemical properties maintaining biological activity after autoclaving at 100°C for extended periods. Despite being produced by a pathogen, harpin lacks intrinsic toxicity to plant cells. Instead, it functions as a potent elicitor, triggering hypersensitive response-like reactions in non-host plants and inducing systemic resistance in diverse crops. Upon application to fruit surfaces, harpin activates pattern recognition receptors, initiating mitogen-activated protein kinase (MAPK) cascades and calcium influx hallmarks of early defense signaling. Commercial harpin formulations have undergone extensive field testing. Treatment of apple and pear orchards pre- and post-bloom reduces fire blight incidence while simultaneously enhancing fruit set [44]. Postharvest applications of harpin reduces brown rot caused by *Monilinia* species [79], [61] The protein's heat stability facilitates formulation and storage, providing practical advantages over labile biological control agents requiring refrigeration.

Oligandrin represents an elicitin-like protein secreted by *Pythium oligandrum*, a mycoparasitic oomycete that parasitizes other fungi and oomycetes [7]. This extracellular protein possesses a molecular mass exceeding 10 kDa and shares structural homology with cryptogein and other well-characterized elicitins from pathogenic *Phytophthora* species. The elicitor properties of oligandrin derive from its recognition by plant immune receptors that evolved to detect oomycete invasion. Upon binding to high-affinity membrane receptors, oligandrin triggers rapid alkalinization of the extracellular matrix, oxidative burst generation, and transcriptional activation of defense genes [42-43]. The resulting induced resistance proves effective against taxonomically diverse pathogens, including fungi, bacteria, and viruses demonstrating broad-spectrum efficacy. Applications of purified oligandrin or *P. oligandrum* bioformulations have demonstrated protective effects in various fruit crops [71], [60].

Chitosan, a partially deacetylated derivative of chitin, stands as one of the most extensively researched biological elicitors for postharvest applications. This linear polysaccharide consists of β-(1,4)-linked N-acetyl-D-glucosamine and D-glucosamine residues, with the degree of deacetylation influencing its physicochemical properties and biological activities. Chitosan exhibits multifaceted mechanisms of action combining direct antimicrobial effects with potent elicitor properties [50] . The antimicrobial activity stems from chitosan's polycationic nature at acidic pH; positively charged amino groups interact electrostatically with negatively charged microbial cell surfaces, disrupting membrane integrity and causing leakage of intracellular contents. Molecular weight and degree of deacetylation critically influence antimicrobial potency generally, lower molecular weight chitosans (oligochitosan, <10 kDa) exhibit superior antimicrobial activity due to enhanced cell penetration. Beyond direct antimicrobial action, chitosan activates plant defense responses through recognition by pattern recognition receptors (PRRs) that detect chitin fragments released during fungal cell wall degradation. [17] pioneered research demonstrating that chitosan treatments induce chitinase, β-1,3-glucanase, and other pathogenesis-related proteins in treated tissues. This dual mode of action, direct inhibition plus induced resistance provides robust protection against postharvest decay. Chitosan applications employ diverse formulations tailored to specific commodities and handling systems. Dip treatments typically utilize 0.5-2.0%

chitosan solutions, with exposure durations ranging from 1 to 5 minutes. Spray applications deliver chitosan in fine droplets, ensuring uniform coverage while minimizing solution volume. Edible coatings incorporating chitosan create semipermeable barriers that regulate gas exchange, reduce moisture loss, and prevent the establishment and reduce the incidence of postharvest pathogens [52], [67], [33].

Microbial biocontrol agents as elicitors

Beyond purified molecular elicitors, whole microbial biocontrol agents can activate plant defense responses while simultaneously antagonizing pathogens through competition, antibiosis, and parasitism.

Antagonistic yeasts particularly *Candida, Metschnikowia,* and *Pichia*, have demonstrated exceptional biocontrol efficacy against postharvest decay fungi [49], [77]. These organisms rapidly colonize fruit wounds, depleting nutrients and occupying niches that would otherwise support pathogen establishment. Additionally, yeasts produce volatile organic compounds (VOCs) with antimicrobial properties and cell wall components that elicit plant defenses.

Spore-forming bacteria of the Bacillus genus offer robust biocontrol solutions due to exceptional environmental tolerance and production of diverse antimicrobial metabolites. *Bacillus subtilis, B. amyloliquefaciens*, and *B. licheniformis* synthesize lipopeptide antibiotics (surfactin, iturin, fengycin) that directly inhibit fungal growth [3]. Concurrently, bacterial cell wall components, lipopolysaccharides, flagellin, peptidoglycans function as microbe-associated molecular patterns (MAMPs) recognized by fruit tissue receptors, initiating defense signaling [19]. The longevity of *Bacillus* spores enables extended shelf life in commercial formulations, facilitating practical deployment.

Pseudomonas fluorescens is a rhizosphere-colonizing bacterium that produces antifungal compounds including 2,4-diacetylphloroglucinol (DAPG), phenazines, and hydrogen cyanide. Beyond direct antagonism, *Pseudomonas fluorescens* induces systemic resistance in plants through secretion of siderophores and lipopolysaccharides that prime defense pathways [69], [39]. Applications to fruit surfaces establish protective biofilms that physically exclude pathogens while continuously stimulating tissue defenses. Strains producing pyoverdine, a fluorescent siderophore, exhibit particularly robust biocontrol activity.

The mycoparasitic fungi *Trichoderma* parasitize other fungi through enzyme production, antibiotic secretion, and competition. *Trichoderma harzianum, T. viride*, and *T. atroviride* secrete hydrolytic enzymes (chitinases, glucanases, proteases) that degrade pathogen cell walls. Released fungal cell wall fragments chitin and β-glucan oligomers subsequently act as elicitors, amplifying fruit defense responses. This cascade mycoparasitism releasing elicitors that enhance host resistance exemplifies the complex interactions underlying biological control [57], [32].

Application methods of elicitors

Elicitors can be applied to fruits through several methods depending on their chemical nature, target tissue, and postharvest objectives. The choice of application method significantly influences the effectiveness of resistance induction and the persistence of protective responses during storage and handling.

Physical elicitors rely on controlled exposure to environmental cues that stimulate the fruit's innate defense system. Chemical elicitors such as salicylic acid, jasmonic acid, chitosan, and β-aminobutyric acid are widely used for postharvest treatments. They are typically applied through dipping, spraying, or coating techniques. Dipping treatment involves immersing fruits in elicitor solutions for a specific duration to ensure uniform surface contact and penetration through the cuticle or micro-wounds. This method allows rapid uptake of elicitors and is suitable for large-scale postharvest handling. Spraying methods, on the other hand, apply elicitors as a fine mist, ensuring even distribution over the fruit surface while minimizing chemical wastage. It is typically used when rapid application is required or when fruits are sensitive to immersion treatments. These coatings form semi-permeable films on the fruit surface, reducing moisture loss, acting as barriers to pathogens, and enabling sustained release of active compounds over time.

Biological elicitors include microbial extracts, cell wall fragments, or living antagonistic organisms such as *Bacillus subtilis*, *Pseudomonas fluorescens*, and *Trichoderma* spp. These agents are commonly applied through bio-coatings, inoculation, or surface colonization techniques. When used as bio-coatings, formulations containing beneficial microorganisms are spread or sprayed on fruit surfaces, allowing the microbes to establish protective biofilms that inhibit pathogen entry. Inoculation methods involve treating fruits with microbial suspensions that colonize surface tissues, competing with pathogens for space and nutrients while simultaneously triggering host defense responses through induced systemic resistance (ISR). Such biological applications are eco-friendly and provide long-term protection without chemical residues.

In practical postharvest management, combined or sequential application of elicitors is often more effective than a single method. For instance, coating fruits with chitosan incorporated with beneficial microbes or applying mild heat treatments before chemical elicitation can exert synergistic effects. Such integrated strategies enhance defense-related enzyme activity, delay senescence, and reduce pathogen infection while maintaining fruit quality during storage and transit.

In summary, the application method plays a pivotal role in determining elicitor efficiency, durability of induced resistance, and compatibility with postharvest operations. Selection should be based on fruit type, elicitor properties, and intended market shelf-life to optimize disease control and quality retention.

Challenges and future prospects

Induced resistance (IR) offers a sustainable alternative to synthetic fungicides for managing postharvest diseases by activating fruit defense mechanisms, but faces significant challenges including inconsistent protection across species and cultivars, dependence on narrow physiological windows as defense capacity declines during ripening, potential quality trade-offs affecting color and texture, technical barriers in formulating stable elicitors for large-scale application, and regulatory hurdles requiring extensive testing. Despite these obstacles, future prospects are promising due to advances in omics technologies that reveal defense signaling networks and optimal treatment windows, development of next-generation elicitors with stronger and more specific effects, smart delivery systems using nano-encapsulation and edible coatings for controlled release, and integrated approaches combining IR with biological control, physical treatments, and reduced-dose fungicides. Success depends on breeding resistant cultivars, tailoring formulations to specific commodities, developing rapid diagnostic tools for monitoring defense responses, and demonstrating economic competitiveness, which could

transform IR from a complementary tactic into a central pillar of sustainable postharvest disease management aligned with tightening fungicide regulations and consumer demand for safer products.

CONCLUSION

Induced resistance offers a promising and environmentally friendly approach to managing postharvest diseases in fruits. This strategy works by activating the fruit's natural defense system rather than directly killing pathogens, making it a sustainable alternative to chemical fungicides. Two main types of induced resistance have been identified: Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR). SAR is triggered by chemical treatments or pathogen signals and develops slowly over several days, providing long-lasting protection against various diseases. ISR is activated by beneficial microorganisms and develops more quickly, being especially effective against decay-causing fungi. Both types offer broad-spectrum protection, but they work through different pathways, allowing flexibility in choosing treatments based on specific fruit types and storage conditions. A wide range of elicitors can be used to trigger these defense responses in harvested fruits. Physical elicitors such as UV-C light, heat treatments, and modified atmospheric pressure offer the advantage of leaving no chemical residues on fruits. Chemical elicitors, including salicylic acid, jasmonic acid, methyl jasmonate, and brassinosteroids, are effective at activating specific defense pathways. Among these, salicylic acid and its synthetic analogues like benzothiadiazole have shown consistent results across many fruit types. Biological elicitors include molecules from microorganisms and living beneficial microbes themselves. Chitosan is the most widely researched, working both by directly stopping pathogen growth and by activating fruit defenses. It can be applied as dips, sprays, or coatings, making it very practical. Proteins like harpin and oligandrin trigger strong defense responses and remain stable even under harsh conditions. Living microorganisms provide another powerful option. Antagonistic yeasts colonize fruit surfaces, creating protective barriers while stimulating defenses. *Bacillus* bacteria produce natural antibiotics and activate resistance through their cell components. *Pseudomonas fluorescens* makes antifungal compounds while priming defense pathways. *Trichoderma* fungi attack pathogens directly and release molecules that further boost fruit defenses, creating a double protection effect. The practical application of these elicitors varies based on fruit type and handling systems, with methods including dipping, spraying, coating, and biofilm formation. The success of induced resistance depends on proper timing, optimal concentrations, and suitable storage conditions. While challenges remain, particularly regarding the decline of defense responses during fruit ripening and the need for standardized protocols, the combination of different elicitor types and application methods shows great promise. As consumer demand for pesticide-free produce increases and regulations on chemical fungicides become stricter, induced resistance stands as a viable strategy for ensuring fruit quality and reducing postharvest losses in a sustainable manner.

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