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Review

Molecular Aspects Revealed by Omics Technologies Related to the Defense System Activation in Fruits in Response to Elicitors: A Review

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Abstract: Fruit losses and wastage are mainly due to postharvest diseases; their control is reduced with pesticides. The excessive use of synthetic fungicides has caused harmful effects on human health and the environment, so it is therefore necessary to reduce their use. The development of new innocuous strategies has led to the use of compounds of natural or biological origin with the capacity to induce the plant defense system, which improves the fruit's response against future pathogen attacks in addition to reducing the incidence of postharvest diseases. These compounds are known as "elicitors". Although the use of molecular tools such as RT-qPCR or the measurement of the enzymatic activity of molecular markers makes it possible to determine the activation of the plant defense system in response to the application of an elicitor compound, in recent years, omics technologies such as the transcriptome, proteome, or metabolome have provided new and interesting information that helps to elucidate the molecular aspects involved in the activation of the plant defense system in response to the application of elicitors. This review summarizes recent advances in molecular aspects, highlighting the contribution of omics technologies to a better understanding of fruit defense mechanisms induced by different elicitors.

Keywords: induced resistance; postharvest; elicitor; defense system; omics technologies

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1. Introduction

Fruit diseases caused by pathogenic microorganisms (e.g., bacteria, fungi, and viruses) lead to massive economic losses worldwide [1]. In this context, inorganic pesticides have been used to control diverse phytopathogenic diseases during the postharvest of fruits; however, their frequent use has promoted microbial resistance and pesticide residuals in the fruits, promoting unhealthy effects and environmental risks [2]. Therefore, collaborative efforts by some government policies and the Food and Agriculture Organization have recommended that the use of inorganic pesticides in agriculture be restricted [3]. According to the literature, several alternatives have been explored to replace pesticides and control pathogenic fruit diseases that are low-cost, safe, and eco-friendly [4]. In that sense, applying elicitors or resistance inducers is an alternative that has attracted the scientific community's attention for over two decades. Physical elicitors such as ultraviolet C irradiation and ozone, chemical elicitors such as plant hormones, polysaccharides, and essential oils, and biological elicitors such as antagonistic yeasts and vegetative growth-promoting bacteria

have been evaluated on fruit during the postharvest stage [5]. Among the biological elicitors, it has been reported that the elicitors differ in their chemical structure, and this depends on the source and nature of the molecule; they are made up of molecules such as lipids, proteins, peptides, and oligosaccharides [6].

An elicitor can enhance resistance against plant pathogens, and its application could be a strategy to control postharvest diseases by activating the immune system in fruits and vegetables [7]. In fruit postharvest treatments, the elicitors improve fruit response considerably against future attacks by pathogens and generate a protective effect [5]. Moreover, elicitors trigger the synthesis of secondary metabolites such as phenolic compounds, flavonoids, lignin, and others, which have an antimicrobial effect and improve fruits' antioxidant capacity [8]. However, the mechanisms involved in activating the defense system in response to applying an elicitor are unclear. Therefore, in recent years, the scientific community has investigated different elicitors and proposed a possible mechanism of action for each case in question [7].

During the last decade, omics technologies such as genomics, transcriptomics, proteomics, and metabolomics have expanded knowledge about the function of genes or proteins involved in the defense system of fruits and vegetables [9]. For instance, Xoca-Orozco et al. (2017) [10] performed a transcriptomic analysis of the avocado fruit-chitosan-*Colletotrichum* interaction system, reporting that chitosan acts as an inducer molecule able to activate multiple metabolic responses in the fruit that collectively implement a defense system capable of counteracting the infection by *Colletotrichum gloeosporioides*. In another work, a metabolomic study in mandarin fruit mediated by preventive applications of cyclic lipopeptides from *Bacillus subtilis* showed an increase in secondary metabolite accumulation, such as serotonin and tyramine [11]. Similarly, the transcriptomic and biochemical analysis highlighted the induction of phenylpropanoid pathway metabolism in citrus fruit in response to salicylic acid, *Pichia membranaefaciens*, and oligochitosan [12].

This review summarizes the molecular aspects of applying the different elicitors to fruits during the postharvest period. Moreover, the mechanisms of defense activation and the molecular pathways induced in fruits by elicitors are also discussed. In this context, it is appropriate to begin by addressing the subject of the plant defense system.

2. Plant System Immunity

Plants possess a complex and sophisticated defense system that activates their response to counteract the damage caused by pathogenic microorganisms, insects, or environmental factors [13,14]. Understanding the activation of the defense system is essential to elucidate how elicitors can activate it and protect plants under different stress situations. It is also essential to highlight the key aspects of the molecular mechanisms of the immune system in plants under biotic stress.

The plant defense response is divided into preformed and induced responses [15]. The preformed response involves physical barriers inherent in the plant and phytochemical compounds that provide primary defense against pathogenic agents [16]. During the preformed response, the physical barriers of the plant, such as the cuticle, play an important role. It has been reported that the cuticle has two layers rich in cutin: the inner is composed of intracuticular polysaccharides and waxes, and the outer layer is rich in epicuticular waxes that generate a mechanical barrier to prevent the proliferation of pathogens [17]. Phytochemical compounds with antimicrobial effects are also essential components of the plant defense system; among the main antimicrobial phytochemicals are phenolic compounds, flavonoids, coumarins, lignins, terpenoids, alkaloids, glucosinolates, and stilbenes [13,18]. The activity of phenolic compounds and flavonoids is based on their ability to inhibit pathogen growth by inducing membrane lipid peroxidation, which disrupts pathogen cell membrane permeability and mitochondrial function [17]. However, the innate plant defense response is triggered when pathogens penetrate the physical barriers by modifying or degrading the host cell wall [13].

When plants recognize pathogens, their innate immune system is activated [19]. This system is composed of pathogen-associated molecular patterns (PAMPs) or damage-associated molecular patterns (DAMPs), pathogen-triggered immunity (PTI), followed by effector-triggered immunity (ETI), which detect and protect plants from pathogen attack [20]. Plants detect pathogens through pattern recognition receptors (PRRs), and these receptors have conserved domains that recognize both PAMPs and DAMPs and activate PTI. Plant PRRs are receptor-like kinases (RLKs), or receptor-like proteins (RLPs) located on the plasma membrane (some receptor examples are shown in Figure 1). RLKs have an ectodomain, a transmembrane region, and a cytoplasmic kinase domain, whereas the structure of RLPs is similar but without the cytoplasmic kinase domain [21]. PAMPs/DAMPs such as chitin, polygalacturonase, and others are recognized by specific PRRs, leading to the production of reactive oxygen species (ROS), activation of mitogen-activated protein kinases (MAPKs), G-proteins, ubiquitin, calcium signaling, hormone signaling, transcription factors (TFs), and epigenetic modifications, regulating the expression of pathogenesis-related (PR) genes [13,19].

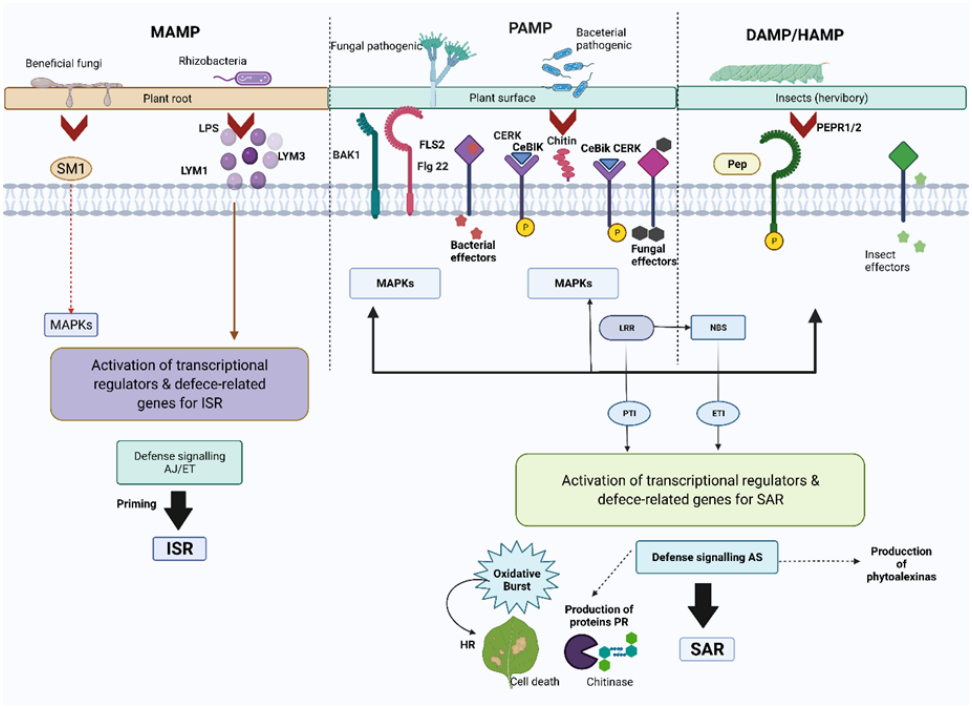

Figure 1. Overview of defense signaling pathways activated by the recognition of pathogen-associated molecular patterns (PAMP)-triggered immunity (PTI), and effector-triggered immunity (ETI) for different types of molecular patterns produced by pathogenic and non-pathogenic microorganisms and insects. The perception of microbe-associated molecular patterns (MAMPS) as flagellin and chitin by the pattern recognition receptors (PRRs; FLS2 and CERK) activates several signaling events, such as MAP kinases (MAPKs). Pathogens or insects deliver effector molecules into a plant to suppress signaling events. Nevertheless, the plants can recognize effectors with the help of R proteins (LRR and NBS) to induce a hypersensitive response (HR) and systemic acquired resistance (SAR), limiting pathogen infection and priming plants against future attacks. Similarly, endogenous phytohormones, such as salicylic acid (SA), jasmonic acid (JA), and ethylene, are induced and contribute to plant immunity. Lipopolysaccharides (LPS) triggered by rhizobacteria activate signaling pathways involving JA and ET, which trigger induced systemic resistance (ISR) and activate protective mechanisms such as *priming*. Adapted from Abdul Malik et al. (2020) [21]. Figure created using BioRender (https://biorender.com/, accessed on 1 December 2022).

Once pathogens are perceived, they can produce effector molecules that act as indicators of pathogenic potential or virulence factors and trigger ETI, which accelerates the

activation of the immune system. The ETI response is induced in the second phase of plant innate immunity. R proteins recognize effector molecules in plants through nucleotide-binding sites or leucine-rich repeats (NB-LRRs or NLRs) [20].

Initiating ETI triggers a hypersensitive response (HR) in the infection site, which prevents the infection from spreading to other parts of the plant, resulting in programmed cell death (PCD) [13]. If the plant resists the disease, it may develop increased resistance to subsequent attacks [17]. This protection is known as systemic acquired resistance (SAR), whose resistance is activated when the plant perceives some biotic and abiotic stress, triggering the response through signaling molecules that activate resistance throughout the plant (Figure 1) [13].

Plants tend to activate different resistance mechanisms to defend themselves: systemic acquired resistance (SAR), herbivore-induced resistance (HIR), and induced systemic resistance (ISR) [21]. Signaling in SAR is mainly mediated by salicylic acid (SA), and its regulation is mediated by a related non-expressor of pathogenesis (NPR1), activating the expression of PR genes involved in defense responses and furthermore of TFs, such as WRKY, NAC, and MYB, that form a complex regulatory network. ISR is mainly activated by jasmonic acid (JA) and ethylene (ET), without the decisive participation of PR gene activation. Another difference is that plant pathogens activate SAR, while plant growth-promoting microorganisms activate ISR [17]. In addition, ISR can be activated by other inducers, such as polysaccharides such as ulvans, laminarans, carrageenans, and fucans isolated from algae [22].

Hormones such as SA, JA, ET, abscisic acid (ABA), nitric oxide (NO), cytokinins (CK), gibberellin (GA), auxin, and brassinosteroids (BR) participate in the signaling cascades during the activation of the defense system [23]. SA is central to local and systemic resistance responses to biotrophic and hemi-biotrophic pathogens. SA and MAPK cascades are regulated through a complex network of interactions; when a pathogen is present, signaling occurs to activate the transcription factors involved in the expression of defense-related genes [21,24]. JA and ET play a vital role in the response of plants to necrotrophic pathogens and herbivorous insects; for example, JA and ET can regulate the emission of volatile compounds in response to herbivores such as caterpillars; oral caterpillar secretions initiate this process [20].

The perception of bacterial flagellin increases ET production as a signaling mechanism. Without ET, ethylene-insensitive TF 3 (EIN3) is degraded by F-box protein-mediated ubiquitination and proteasome activity. Nevertheless, upon ET presence, constitutive triple response protein1 (CTR1) is inactivated, a repressor of ET response. When CTR1 is inactive, the FTs EIN2 and EIN3 cascade down and are activated. These FTs are positive regulators of expression genes involved in defense against phytopathogens [20,25].

The roles of ABA, NO, auxin, CK, GA, and BR in immunity and plant development demonstrate that defense and growth molecules are closely related. ABA is involved in several plant stresses, including repression and promotion of resistance responses to abiotic stress. Stomata closure involves ABA signaling to regulate water loss, gas exchange, and pathogen access to tissues. Likewise, GA deficiencies increase pathogen resistance [23].

When the defense system is activated, there is an increase in the levels of several secondary metabolites involved in plant defense mechanisms, such as phytoalexins, which are toxic to pathogens [17]. A plant with an active defense system increases the activity of defense-related enzymes such as phenylalanine ammonia lyase (PAL), superoxide dismutase (SOD), peroxidase (POD), and polyphenol oxidase (PPO). SOD, POD, and PPO stop the progression of membrane lipid peroxidation and oxidative stress triggered by pathogen attack. SOD is the first enzyme that acts against oxidative stress and regulates other defense enzymes. POD is also involved in cell wall polysaccharide metabolism, catalyzing phenol oxidation and lignification processes that protect plant tissues against pathogen invasion; in turn, PPO is involved in the oxidation of polyphenols to quinones, which have antimicrobial activity [26].

Moreover, abiotic factors such as light, temperature, osmotic, or hydric stress affect the plant-microbe interactions that modulate plant immunity; in this sense, it has been reported that plants under stress caused by drought, salt, heat, or cold show different patterns of cytosolic calcium level fluctuations that induce signaling regulated by the hormone ABA, which is vital in multiple roles in stress abiotic and biotic responses [27].

In fruits, information on the activation of the defense response is crucial. Recent studies have made use of omics technologies (e.g., genomics, transcriptomics, and metabolomics) to elucidate how the defense system has activated the fruit during the pathogen infection [9,28]. Postharvest fruit diseases are mainly caused by fungi. Essential aspects of the activation defense system in fruit during pathogen fungi infection are deciphered below.

3. Fruits Defense System Activation by Pathogenic Fungi

Fruit consumption represents an essential contribution of nutrients, such as vitamins, minerals, and antioxidants, among others, that benefit human health. However, recent data show that about 50% of fruit production is lost or wasted worldwide [3]. Postharvest diseases caused by pathogenic fungi mainly cause these losses [1]. Among the primary postharvest pathogenic fungi are *Botrytis cinerea*, *Penicillium* spp., *Monilinia* spp., *Alternaria alternata*, *Aspergillus* spp., *Rhizopus stolonifer*, *Trichothecium roseum*, *Fusarium* spp. and *Colletotrichum* spp. [29], as well as bacteria of the genus *Pseudomonas* and *Erwinia,* and viruses such as the ring spot virus [30,31]. Although diseases caused by bacteria and viruses also cause losses in postharvest crops, they are minor compared with fungi pathogens [31]. The principal factor determining fruit resistance to pathogens is the ripening stage. Fruits infected by fungal pathogens develop disease symptoms after harvest and during storage, even if the infection occurred at the pre-harvest stage [16].

Consequently, the scientific community has constantly investigated both the processes of pathogenesis and fruit resistance to pathogen attacks [16,29,32]. The infection process typically starts with spores that reach the fruit surface; postharvest pathogenic fungi follow three pathways of penetration into fruit tissue: (1) through wounds caused by biotic and abiotic agents; (2) through natural plant openings, such as the pedicel-fruit interface and stomata; and (3) through direct rupture of the fruit cuticle. Pathogens remain quiescent on the cuticle of immature fruit and are not visually perceived until the fruit ripens [16,32]. Mature fruit is more prone to infection mainly due to the onset of senescence, which is associated with weakened defense systems, softening of tissues, and increased ethylene production. The fungal conidia attach themselves to the fruit surface and start germination; they initiate its development and successfully colonize the tissue by mechanical force or the production of hydrolytic enzymes (e.g., cutinase, polygalacturonase, and lipase) [15,16]. The quiescent pathogens, principally *Colletotrichum, Alternaria, Botrytis, Monilinia, Lasiodiplodia, Phomopsis,* and *Botryosphaeria,* cause symptoms such as necrosis due to their capacity to kill the host cell and obtain nutrients from the host, causing the decomposition of fruit tissue and decay. However, some infection processes by fungi such as *Botrytis cinerea*, *Monilinia laxa,* and *Lasiodiplodia theobromae* initially occur in floral parts but stay quiescent until ripe fruit.

With the contribution of omics sciences, both genomics and transcriptomics, the molecular events triggered during the fungal infection have been elucidated [33]. According to Ngolong Ngea et al. (2021) [32], the transmission signals from pathogens during infection involve the activation of metabolic pathways such as mitogen-activated protein kinase (MAPK), cyclic adenosine monophosphate (cAMP), nonfermenting sucrose-activated protein kinase 1/AMP (SFN1/AMPK), and high osmolarity glycerol (HOG). Transcription factors also play an important role in downstream signaling events, regulating gene expression essential for triggering pathogen virulence [32].

Regarding the fruit response to pathogen attack, it has been documented that fruits can activate the defense system through the SAR response, which triggers signaling processes and activation of defense genes reported in plants. For example, tomato fruits infected with

C. gloeosporioides induced genes encoding for PAMP receptors and genes related to fatty acid biosynthesis, elongation, and cutin and wax synthesis in the fruit [16].

ROS production plays an essential role in fruit defense mechanisms against pathogen attack, a process known as oxidative burst, where the production of superoxide anion, hydroxyl radical, and H_2O_2 is increased in response to wounding or pathogen attack. In addition, there is the activation of different enzymes that can regulate oxidative stress [32]. For instance, studies conducted during the orange-*P. digitatum* interaction showed that the antioxidant activities of SOD, catalase (CAT), ascorbate peroxidase (APX), and glutathione reductase decrease at different rates as the fungus advances around the tissue entirely colonized by the pathogen. In contrast, in the apple-*P. expansum* interaction, genes encoding ROS detoxification enzymes, such as SOD, APX, and POD, were induced [33].

Other processes, such as lignification and the synthesis of compound phenolics, are regulated by PAL, a key enzyme in the phenylpropanoid pathway [17]. In the orange-*P. digitatum* pathosystem, the maximum expression of several phenylpropanoid-related genes was detected 48 h after inoculation, and the expression of PAL1, caffeic acid O-methyltransferase, and POX1 genes were induced. In the apple-*B.cinerea* pathosystem, enzymes key in the pathway phenylpropanoids, such as PAL, cinnamate 4-hydroxylase (C4H), 4-coumarate coenzyme A ligase (4CL), and cinnamyl alcohol dehydrogenase (CAD), showed an increment in activity, which up-regulates the biosynthesis of phenolic acid, flavonoids, and lignin [34].

Another defense mechanism is activated by PR proteins, which are induced in the fruit defense response [33]. Studies have described that enzymes such as chitinase and glucanase, were increased in tomato fruit in response to *A. alternata* infection and in grapes infected by *B. cinerea* [33]. An analysis of proteomic data revealed 196 differentially accumulated proteins in kiwifruit associated with the response to the infection *B. cinerea,* ubicated in pathways such as MAPK cascades, ROS signaling, and PR proteins that play a crucial role in modulating the resistance of the host against the pathogen [35].

Hormones such as SA, ET, and JA play an essential role in infection signaling processes; in that sense, it has been described that in tomato fruits infected by *B. cinerea* and *C. gloeosporioides*, the ethylene biosynthesis pathway was induced, in addition to TFs such as non-ripe (NOR), ripening inhibitor (RIN), never ripe (NR), and several ethylene-regulated defense genes [16]. Salicylic acid and JA signaling pathways are generally antagonistic and dependent on NPR1 expression levels and hormone concentration. The interaction between SA and JA has optimized the host response to the pathogen's lifestyle. In vegetative tissue, effective responses to biotrophic pathogens are commonly mediated by SA and programmed cell death, in addition to responses to necrotrophic pathogens, which benefit from host cell death and involve JA signaling [16].

Several authors consider this information of great interest. Understanding how fruits activate their defense mechanisms in the presence of pathogens and generate resistance is of great interest to know how the fruit activates its immune system against future attacks by pathogens. This information has given a guideline to establish why the application of resistance inducers represents an effective strategy for disease control, especially in the postharvest stage [17].

Much research is based on this information to evaluate marker genes and enzyme activity by applying an elicitor. Therefore, the use of elicitors as a strategy for postharvest disease control has increased, and several elicitors have been evaluated. Since elicitors have different natures, a classification is necessary to identify them according to source or structure.

4. Classification of the Elicitors to Induced Resistance in the Postharvest Stage

According to dos S. Costa et al. (2022) [36], elicitors may be defined as molecules that play a role in the triggering or stimulating of defense mechanisms of the plants; also, elicitors are known as plant resistance inducers, resistance activators or defense activators. The elicitors can be classified as biological, chemical, and physical; their perception is

mediated by specific receptors that give information to the host for initiating the cascade of signaling and causing the induction of different types of immunity in the host [6]. Induced resistance is a new strategy that could effectively control postharvest diseases by activating the immune ability in fruits and vegetables, thereby increasing resistance against pathogens [7]. The molecules elicitors are recognized by receptors present in the host and initiate SAR or ISR by the expression of PR genes [20].

Previous information about the types of elicitors has been investigated principally in plants [6]. It has been classified according to their chemical structure in proteins, oligosaccharides, glycopeptides, lipids, lipopeptides, small molecules such as metabolites, and chemical compounds that are produced from several sources (animals, plants, microbes or their metabolites, active molecules produced during interaction plant-pathogen, or natural/synthetic compounds) [37]. Some proteins and peptides considered elicitors are found in flagellin, harpin, xylanase, elicitin, RNAase, cellulose, aldose 1-epimerase, and peptides such as phytosulfokine, AtPep1, PIPs, and GmPeps. Carbohydrates such as exopolysaccharides, chitin, xyloglucan, and oligochitosan. Lipids such as lipopolysaccharides, ergosterol, eicosapentaenoic acid, and arachidonic acid. Chemical compounds such as benzothiadiazole, 2,6-dichloro isonicotinic acid, probenazole, and dufulin are some elicitors that activate plant immunity [37].

According to the bibliography, in the last five years, in the postharvest stage of fruit, there has been the development of safe strategies for postharvest disease control, and the scientific publications are research articles that deal with elicitors tested on fruits. Most of the time, the application of elicitors is focused on treating postharvest diseases, preserving shelf life, and increasing the concentration of secondary metabolites. As mentioned above, the elicitors can be classified into physical, chemical, and biological origins; therefore, the relevant information about using elicitors in postharvest is described below.

5. Biocontrol Agents with Elicitor Potential

According to Köhl et al. (2019) [38], biological control agents are typically recognized as PAMPs, which induce defense pathways in the plant to increase the host's resistance against an opportunistic pathogen. Among the most studied biocontrol agents are *Bacillus* strains since they can provide resistance through the induction of defense mechanisms; for instance, recently, the use of a new *Bacillus atrophaeus* TE7 showed a biocontrol efficacy of 85.56% in mango fruits; this treatment was effective in controlling the development of *Cladosporium cladosporioides* [39]. Yeasts have also demonstrated efficacy in disease control. The effectiveness of treatment with *Pichia guillermondi* and *Kloeckera apiculata* on plum fruit has been established. These two yeasts were shown to control infection by *Monilinia fruticula*, and the colonization of these yeasts in the fruit activated the phenylpropanoid pathway through the activation of enzymes such as PAL and POD. These enzymes enhance the biosynthesis of some metabolites such as lignin, flavonoids, and phenolic compounds, which can prevent the development of pathogens [40].

For this reason, different biocontrol agents have been tested on fruits and vegetables, some examples of which are shown in Table 1.

6. Physic Elicitors with Elicitor Potential

According to Romanazzi et al. (2016) [5], several physical stimuli, such as ultraviolet-C (UV-C) light, heat, and hypobaric and hyperbaric treatments, can induce changes in host tissues, including increased resistance to abiotic and biotic stress. The new technologies, such as high-intensity pulsed polychromatic light applied to tomatoes, delayed ripening by reducing the color index by 50.2% and induced fruit resistance to *B. cinerea* disease by 41.7% in terms of the reduction of disease symptoms. In contrast, the treatment with low-intensity UV-C light decreased 42.8% in color index and only 38.1% in removing symptoms caused by *B. cinerea* [41]. Novel technologies have been used as physical elicitors; some recent research is described in Table 2.

7. Chemical Elicitors: Naturals and Synthetics

Several authors mention that the resistance response in plants is mainly modulated by phytohormones such as SA, JA, and ET, among others, which play a central role in the regulation of defense processes [5,7,42]. In this sense, searching for new hormones that can modulate the response of the fruit to pathogen attacks has gained popularity in recent years; for example, benzothiadiazole and indole-3-acetoacetic acid have been applied in fruits as defense system elicitors [43,44].

Likewise, polysaccharides from natural sources such as chitosan, fructooligosaccharide, carrageenan, or fucans have been considered elicitors [45], and recently, agave fructans were reported as effective elicitors to control anthracnose in avocado fruit [46]. Moreover, some plant metabolites, such as epicatechin, quercetin, and essential oils, are used for postharvest disease control [47,48]. In addition, peptides such as mytichitin-CB and Epsilon-poly-l-lysine induced disease resistance in cherry tomato and apple fruits, respectively [49,50], and proteins such as harpin were adequate to control gray mold in strawberries; this was associated with the increment in the PAL activity and inducing a defense response that influenced the improvement of quality attributes in strawberries [51]. Moreover, inorganic compounds such as silicon, nitric oxide, and sodium carbonate enhance fruit responses to stress situations [52,53]. Furthermore, applying exogenous gases, such as ozone, nitrous oxide, and carbon monoxide, improves disease resistance in mandarin, grape, and jujube fruits [54–57] (Table 3).

As described above, elicitors of different types have been tested to trigger the defense response of fruits against pathogen attacks. Omics technology has contributed to a clearer understanding of the metabolic pathways involved in activating the vegetal defense system, giving us the knowledge to explore this area in more detail.

8. Omics Technologies in the Study of Fruit-Elicitor Interaction

During the last few years, there has been a growing interest in the use of "omics" technologies in biological sciences, and their use in research about the activation of defense mechanisms in fruits through the application of an elicitor has not been an exception. Omics technologies (transcriptomic, proteomic, or metabolomic) offer a global analysis that expands our knowledge and understanding of the activated metabolic processes. The following describes the most recent research (last five years) on applying these technologies to fruit in response to elicitors.

8.1. Omics Technologies in the Induction of the Defense System by Biological Elicitors

Biocontrol agents (bacteria or yeasts) have several mechanisms of action for the control of postharvest diseases, and one of them is the induction of the defense system [38]. In that sense, researchers have contributed to elucidating the mechanisms of resistance induction in fruits by relying on omics sciences as a tool.

In this sense, it has been reported that *Bacillus* is a biocontrol agent that generates secondary metabolites such as cyclic lipopeptides (CLPs) that induce the vegetal defense system; for example, CLPs from *Bacillus subtilis* ABS-S14 effectively controlled green mold disease in mandarins [58]. In addition, proteomic analysis revealed the mechanisms for activating the defense system in mandarin oranges by applying CLPs. The CLP extract increased protein production in the metabolic pathways of Ca^{2+}, ABA, glycolysis, and ROS signaling, which triggered the expression of PAL, GLU, POD, and PR1 genes or proteins, resulting in the activation of the SAR pathway [59].

When evaluating the individual effects of the lipopeptides fengicin, iturin A, and surfactin from *B. subtilis* ABS-14 on mandarin fruits, the results showed that fengicin, Iturin A, and surfactin induced the expression of crucial genes involved in the ET signaling pathway as well as genes encoding CHI proteins that are important for the ISR response in plants [60].

Metabolomic studies revealed that the metabolites induced specifically by *B. subtilis* CLPs were involved in the metabolic pathways of glycine, serine, threonine, tryptophan,

and tyrosine metabolism, which increased the production of secondary metabolites such as serotonin and tyramine, leading to the induction of mandarin fruit immunity [11].

The biocontrol capacity of *Bacillus cereus* AR156 on strawberry fruits was also investigated, and transcriptomic profiling showed that *Bacillus* AR156 increased the expression of numerous transcription factors, such as MYB, NAC, WRKY, ERF, bHLH, and bZIP, involved in the induction of the defense system. Transcription factors of the WRKY family are involved in metabolic pathways of plant-pathogen interaction in plants, which trigger the activation of the defense system. In addition, it was reported that a significant effect on the expression of genes related to flavonoid biosynthesis, which, as mentioned above, increased flavonoid concentration effectively controls pathogen development [61].

Similarly, to investigate the mechanisms of *Bacillus siamensis* induction, a comparative analysis of the mango fruit transcriptome during storage was established. Metabolic pathways such as plant-pathogen interaction, plant hormone signal transduction, phenylpropanoid, flavonoid, stilbenoid, diarylheptanoid, and gingerol biosynthesis were the most enriched pathways, indicating that these processes were involved in the response of mango to *B. siamensis*. Some genes (JAZ, BAK1, and PR1) were up-regulated by *B. siamensis* treatment, which triggered the stress response, induced phenol biosynthesis, and enhanced the disease resistance of mango fruit. In addition, some genes (WRKY22, HSP90, CNGCs, SOD, PAL, 4CL, CHS, and HCT) were up-regulated by *B. siamensis* in mango fruit, which stimulates the immune response and resistance to mango fruit disease [62].

On the other hand, the yeast antagonist *Yarrowia lipolytica* elicited disease resistance and proved an effective biocontrol agent against *P. expansum* in apples. The proteome and transcriptome of the yeast-treated apples and the control were analyzed [63]. The authors propose metabolic pathways, such as responses to biotic stress, defense responses, protein synthesis and storage, and signal transduction, pointing out the most dynamic categories in response to biotic stimuli and defense. The analysis of the transcriptome results proved that the induced resistance was mediated by crosstalk between the SA and ET/JA pathways. *Y. lipolytica* treatment activated the ACS1 gene, and EIN2 and 4, which are involved in the ET pathway, also activated genes such as POD, thaumatin-like protein, and CH4, elicited by *Y. lipolytica* in apples [63].

The mechanisms involved in *Pichia membranaefaciens*-induced resistance in peaches were also investigated [64]. Transcriptomic analysis revealed that the MAPK signaling pathway and the regulation of transduction signals by plant hormones such as ET, JA, and AS were activated in peaches by *P. membranaefaciens*. The results showed up-regulation of defense-related genes, including PR genes (PR1, CHI4, and major allergen Pru ar 1) and glutathione S-transferase genes (MKP11.22 and Atlg10370), in addition to genes involved in plant-pathogen interaction pathways (CML48, MUK11. 19, and ROBHA) and genes involved in the synthesis of secondary metabolites (GGPS, PK55, CHS1, CYP52B2, DRF, LDOX, PAL, PNC1, and ROMT) that contributed to improving peach tree resistance potential to diseases. This induction reflected an increase in the concentration of secondary metabolites, such as flavonoids and lignin, which help to increase disease resistance [64].

8.2. Omics Technologies in the Induction of the Defense System by Natural Chemical Elicitors

As mentioned above, carbohydrate polymers are considered elicitors, and one of the most studied is chitosan [45], as it has proven effective in controlling various postharvest diseases [65]. Chitosan has different mechanisms of action, among which stands out is its ability to induce the defense system; in this sense, transcriptomic analysis in avocado during the development of anthracnose caused by *Colletotrichum gloeosporioides* revealed that the differential genes were located in metabolic processes regulated by chitosan, including those that prevent the propagation of *Colletotrichum* [10]. Differentially expressed genes were significantly increased in different metabolic pathways involved in the defense system, e.g., cellular processes, metabolic processes, response to abiotic stress or biotic stimulus, biological processes, transport, cellular organization and biogenesis, and signal transduction. The authors found that chitosan could induce a priming state in short times

after application, which promotes effective fruit resistance against *C. gloeosporioides*, and that those fruit treatments with chitosan up-regulate some genes involved in phenylpropanoid biosynthesis such as 4CL, transcription factors such as WRKY22 and ERF, and genes involved in AFD diene biosynthesis. The results presented in this study showed that chitosan acts as a molecule capable of inducing multiple metabolic responses in avocado fruit that collectively implement a defense system capable of counteracting *C. gloeosporioides* infection [10].

Recently, transcriptomic and metabolomics analyses were used to evaluate the effect of chitosan treatment on the resistance to *B. cinerea* of two grape varieties ("Kyoho" and "Shine Muscat") that differ in their resistance to this pathogen [66]. The authors propose a model of chitosan regulating the resistance of "Kyoho" and "Shine Muscat" grapes to *Botritys cinerea* based on data from the transcriptome, metabolome, antioxidant enzyme activity, signal perception, plant hormones, and secondary metabolism. Interestingly, the model of resistance regulation by chitosan involved perception through PAMPs within the metabolic pathways for hormone regulation in plants and secondary metabolism deregulated genes such as PAL, ACS, ACO, EIN3, C4H, and CHS, among others. Secondary metabolites such as cinnamic acid, catechin, resveratrol, quercetin, and terpeptin A were significantly regulated by chitosan. However, chitosan inhibited the secondary metabolism of Kyoho and activated the secondary metabolism of Shine Muscat. With this information, they found that Shine Muscat had more vigorous resistance to *B. cinerea* than Kyoho but, based on the data, established a possible chitosan model regulating disease resistance [66].

Another carbohydrate polymer used is dextran, a complex branched glucan consisting of α-1,6 glycosidic linkages and α-1,3 linkages between glucose monomers. The application of dextran to tomato fruit inhibited gray mold caused by *B cinerea* [67]. Moreover, the transcriptomic analysis revealed that the metabolic pathways of phenylpropanoid biosynthesis, flavonoid biosynthesis, linoleic acid metabolism, stilbenoid, diarylheptanoid, and gingerol biosynthesis, plant-pathogen interaction, and plant hormone signal transduction were significantly up-regulated in response to dextran elicitor treatment. In addition, the expression of Slpa1, Slpr1, Sllox1, and genes encoding TMV resistance protein were increased in dextran-treated fruit; the authors indicate that these results support the previous hypothesis that dextran may be perceived by the β-glucan-like defense system and trigger the response against *B. cinerea* infection [67].

Other authors have evaluated different elicitors in the same fruit. Illumina sequencing technology was used to investigate the transcriptome of citrus treated with SA, *P. membranaefaciens*, and oligochitosan [12]. The results showed that these elicitors caused substantial changes in mRNA relative to control fruits by activating secondary metabolite biosynthesis in citrus responses to SA, *P. membranaefaciens*, and oligochitosan. PAL, C4H, 4CL, and POD expression levels were higher, demonstrating that all three types of elicitors are involved in gene regulation of phenylpropanoid biosynthesis during the induction of fruit resistance [12].

Furthermore, the use of omics tools to evaluate the combination of two elicitors has also been reported, such as *M. guilliermondii* combined with alginate oligosaccharide in pear fruit, which was investigated by transcriptomic analysis [68]. According to the authors, this combination of elicitors increases the expression levels of related genes in the plant-pathogen interaction pathways and the WRKY signaling pathway. WRKY transcription factors are involved in signal transduction that triggers the defense response in plants. In addition, it induces multiple disease resistance genes (RPP13, RPM1, RGA3, RGA4), defense genes (TLP1b, MLO3, and MKS1), and antioxidant stress-related genes (ASO, GSTU17, RVE1, and GLP13) to improve disease resistance and antioxidant stress capacity of pear fruit and promote the synthesis and accumulation of resistant substances in pear fruit by increasing the expression levels of genes involved in the phenylpropanoid and flavonoid biosynthesis pathways (4CL, CAD1, POD1, CHI, CHI3X1, CYP75B1, and ECMP1). In addition, increased the expression of genes related to cell wall integrity (GRP, PRP, GLP13, and CYP51) and the sphingolipid metabolism pathways (AGAL1X1, GBA2X1, ASAH2, and

SPHK1X1), which help maintain cell membrane integrity, which prevents the development of pathogens. Finally, the up-regulation of several genes closely related to plant resistance (PUB23, RGLG1, LACS4, LOX1.5, and PKS5) also plays a crucial role in enhancing pear resistance [68].

8.3. Omics Technologies in the Induction of the Defense System by Chemical Inorganic Elicitors

Sodium silicate (Si) effectively suppresses pathogen growth and induces postharvest disease resistance in fruits and vegetables [5]. Preventive application of Si to melon fruits activates the defense response against *Trichothecium roseum*. Proteomic changes in melon fruit mitochondria after Si treatment were analyzed using a tandem mass tag (TMT)-based comparative proteomics approach. A total of 24 mitochondrial proteins were significantly altered; a comparison of protein abundance between groups showed that 19 proteins were up-regulated. Five proteins were down-regulated: metal ion binding, transmembrane hydrogen ion transporter activity, ATPase activity, and oxidoreductase activity. The identified proteins are divided into six functional groups: energy metabolism, defense and stress response, oxidation-reduction processes, glycolytic and tricarboxylic acid cycles, and amino acid metabolism (including GABA shunting). The authors found that the proteins were differentially expressed in muskmelon fruits primed by Si treatment in response to pathogen inoculation, forming a dynamic interaction network during resistance induction. They suggest that mitochondria play an essential role during the priming of resistance against the disease by regulating energy metabolism and ROS production in Si-treated muskmelon fruits [69].

On the other hand, using gases such as carbon dioxide (CO_2) contributes to preserving the shelf life of the fruit as well as inducing defense mechanisms [70]. The cellular response of harvested strawberry fruit subjected to short-term (3 h) exposure to 30% CO_2 was investigated using transcriptomic and metabolomic analyses [71]. The CO_2 treatment reduced fruit softening and deterioration during storage at 10°C for 10 days. According to the authors, CO_2 treatment could improve fruit storage capacity by activating abiotic stress-related genes (e.g., HSPs) and down-regulating genes related to cell wall degrading enzymes (e.g., expansin, pectinesterase, and β-xylosidase). Furthermore, CO_2 treatment induced abiotic stress-related cellular responses in strawberry fruit, stimulating defense mechanisms [71].

9. The Importance of Knowing the Information Generated by Omics Technologies in the Interaction between Fruit and Elicitor

The metabolic changes in fruits after applying an elicitor are complex; however, the information generated by omics technologies so far provides a better understanding of the response of fruits to elicitor application [9]. Although the data obtained are specific to each fruit-elicitor interaction and it is complex to propose a general action mechanism for the fruit-elicitor interaction, the authors suggest, based on their results, a mechanism by which the elicitor acts and provides resistance to fruit against pathogens.

Some researchers have used the information generated to design postharvest disease control strategies; for example, Xoca-Orozco et al. (2019) [62] used information obtained from a previous transcriptome of the avocado-chitosan interaction to elucidate the metabolic pathway of phenylpropanoid biosynthesis and analyzed the changes in gene expression by quantitative PCR using specific primers to target the genes: PAL1, C4H, 4CL, CHS, and FLS. The authors mentioned that FLS participates in the biosynthesis of kaempferol, and this pathway's final product is quercetin. Quercetin and epicatechin are compounds with high antioxidant activity, which may limit the action of lipoxygenases and allow the accumulation of persin to inhibit the pathogen infection, analyze the expression of genes related to the antifungal compound 1-acetoxy-2-hydroxy-4-oxo-heneicosa-12-15-diene, and trigger resistance in avocado fruits against pathogens. Knowing that these antifungal compounds are induced, extracts were obtained from avocado exocarp previously elicited

with chitosan to evaluate their antifungal capacity. The results showed that 16 mg mL-1 of this extract could inhibit >50% of the mycelial growth of *C. gloeosporioides* [72].

Table 1. Examples of biocontrol agents with postharvest elicitor potential.

Elicitor	Fruit	Gene	Enzyme	Effect	Reference
Clonostachys rosea	Tomato		PAL PPO CAT ABA	Increases in indole acetic acid (IAA), salicylic acid (SA), and NO levels	[73]
Meyerozyma guilliermondii	Pear		POD CAT PAL	Inhibited the blue mold decay and induced disease resistance in the pear	[74]
Pseudomonas fluorescens	Grapes		PPO POD CAT PAL CHI GLU	Cell suspension of *P. fluorescens* inhibited spore germination of *B. cinerea*, and reduced the incidence of gray grape mold	[75]
Pichia guilliermondii	Peach	NPR1 AtWRKY 50 PR1 GLU CHI	SOD CAT PPO GLU PAL	Biological elicitor-activated systemic acquired resistance by the SA signaling pathway	[76]
Wickerhamomyces anomalous	Tomato		PPO POD CAT PAL	Reduced the gray mold decay without affecting cherry tomatoes' quality	[77]
Bacillus subtilis	Blueberry		CHI PAL POD PPO	Preventive treatment was more effective than the curative one in controlling gray mold-induced decay	[78]
Bacillus halotolerans	Strawberry		PPO PAL GLU CHI	The gray mold in strawberries inoculated with *B. halotolerans* was lower in comparison with that in the control fruit after 4 d of incubation	[79]
Trichoderma asperelloides	Muskmelon	CHI GLU	CHI GLU	Reduced disease severity against gummy stem blight by overexpressed PR genes and elevated enzyme activity	[80]
Burkholderia contaminans	Strawberry		PAL 4CL C4H CHI	*Bulkholderia contaminants* reduced the incidence of postharvest disease and promoted the accumulation of lignin and total phenols.	[81]
Pichia galeiformis	Citrus	PAL 4CL C4H POD CAD	PAL 4CL C4H POD PPO CAD	*P. galeiformis* reduced the disease incidence and lesion diameter without direct contact with the pathogen *P.digitatum*.	[82]

PAL = phenylalanine ammonia-lyase; PPO = polyphenol oxidase; CAT = catalase POD = peroxidase CHI = chitinase; GLU = glucanase; NPR1 = non-expressor of pathogenesis-related genes 1; PR1 = pathogenesis-related protein 1; C4H = cinnamate 4-hydroxylase; 4CL = 4-coumarate coenzyme A ligase; CAD = cinnamyl alcohol dehydrogenase.

Table 2. Examples of physical treatments with elicitor potential in postharvest.

Elicitor	Fruit	Gene	Enzyme	Effect	Reference
Gamma irradiation	Pear	PR-1 PR-3 PR-4	GLU PAL POD PPO	The gamma irradiation-induced resistance against *P. expansum*	[83]
Hot water rinse brushing and UV-C	Mango		POX PAL PPO	The defense-related enzymes induced resistance was an important mechanism involved in the control of stem-end rot in mango	[84]
UV-C	Mangosteen		PAL POD GLU	UV-C application improves the quality of mangosteen.	[85]
Light-emitting diode (LED)	Avocado	PAL LOX		LED light application can induce fruit resistance against the postharvest disease anthracnose in avocado	[86]

PAL = phenylalanine ammonia-lyase, PPO = polyphenol oxidase, CAT = catalase POD = peroxidase CHI = chitinase, GLU = glucanase, PR1,2,3 = pathogenesis-related protein 1,2,3, LOX = lipoxygenase.

Table 3. Examples of natural and synthetic chemicals with postharvest elicitor potential.

Elicitor	Fruit	Gene	Enzyme	Effect	Reference
Quercetin	Kiwi	PR1 NPR1 CHI GLU	CHI GLU PAL PPO POD	Quercetin inhibits blue mold caused by *P. expansum*, which may be associated with its toxic properties and induction of defense response.	[47]
Indole-3-acetic-acid	Pear	Endoglu9 CHI4 PR1 PR4 PAL	GLU CHI PAL	IAA induces natural resistance of pear fruit against *P. expansum* and suggests that the mechanisms may be closely related to the elicitation of enzymes and defense-related genes.	[44]
Trisodium phosphate	Apple		SOD CAT APX GR PAL POD	Enhanced disease resistance in apple fruits by TSP against *A. alternata* is associated with increasing antioxidative enzyme activities and accumulation of phenylpropane metabolites	[87]
Chitosan	Avocado	PAL CHI LOX	SOD CAT	The control of stem-end rot and anthracnose in avocados obtained with 1.5% chitosan can be ascribed to a combination of its antifungal and eliciting properties.	[88]
Salicylic acid	Longan		PLD PLC Lipase LOX	SA treatment could 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	[89]
Benzothiazole	Orange		SOD POD CAT GLU PAL CHT	BTH had promising effects on improving resistance against postharvest blue mold disease in navel orange	[43]

Table 3. *Cont.*

Elicitor	Fruit	Gene	Enzyme	Effect	Reference
β-aminobutyric acid (BABA)	Apple	EF-1α PR-1 PR-2 LOX Def		BABA reduced disease symptoms caused by *P. expansum*, in addition to the increment in the expression of the PR-1 and LOX gene and callose opposition in the cell walls that induced resistance to the pathogen.	[90]
2,6-dichloroisonicotinic acid (INA)	Citrus		GLU CHI PAL POD PPO	Treatment reduced blue and green molds and anthracnose decay in citrus	[91]
Methyl jasmonate	Sweet cherry	POD PPO SOD CAT LOX AOS OPR3 MYC2	CAT POD SOD PPO PAL CHI GLU	MeJA reduced sweet cherry fruit spoilage and is related to its induction effect rather than its fungitoxicity effect.	[92]
L-glutamate	Pear	PR1 GLU CHI3 CHI4	GLU CHI PAL POD PPO	L-glutamate at 1.00 mM induced strong resistance against blue mold rot caused by *P. expansum* in pear fruit under either 25 °C or 4 °C conditions and reduced spore germination of *P.expansum* in fruit wounds and in vitro after 24 h of treatment.	[93]
Carboxymethyl chitosan (CMSC) and *Criptococcus laurentti*	Grape		POD PPO PAL	The combination of CMSC and *C. laurentii* treatments can maintain fruit quality and control postharvest decay more effectively than a single treatment.	[94]
Chitosan and Salicylic acid	Grape		GLU POD PAL PPO	Chitosan combined with Salicylic acid reduced the lesion diameter and disease incidence, incrementing the concentration of Salicylic acid endogenous.	[95]
Pectic Oligosacharides (POs) in cold-stored	Grapes	MnSOD APX CAT GR2		POs significantly modulated the MnSOD, APX1 and CAT1 expression levels, mainly in a storage time- and temperature-dependent manner, concerning controls. By contrast, POs only significantly affected the GR2 gene expression when grapefruit were stored at non-chilling temperatures	[96]
Ozone	Satsuma mandarin	SOD GLU7 Defensin-like-protein 1		Ozone treatment effectively delayed the fruit decay, also significantly reduced fruit respiratory intensity, delayed natural fruit degreening, and prolonged shelf-life of Satsuma mandarin fruit during postharvest storage	[56]

LOX = lipoxygenase, AOS = allene oxide synthase, OPR = 12-oxo-phytodienoic acid reductase, PLD = phospholipase D, PLC = phospholipase C, GR = glutation reductase, CHT = chalcone isomerase, MnSOD = manganese superoxide dismutase, PAL = phenylalanine ammonia-lyase, PPO = polyphenol oxidase, CAT = catalase, POD = peroxidase, CHI = chitinase, GLU = glucanase, CAD = cinnamyl alcohol dehydrogenase, APX = ascorbate peroxidase.

10. Concluding Remarks

Applying elicitors to induce the defense system in fruits has gained considerable popularity as an environmentally friendly alternative to generate resistance without harming the environment or human health. The use of omics technologies has contributed to expanding knowledge and identifying specific metabolic pathways involved in activating the defense system and genes/proteins that are deregulated upon elicitor application. However, many genes/proteins can still be investigated and characterized to understand their involvement

in the activation of the defense system. The responses that each elicitor can induce in fruits comprise a complex network of genes that are deregulated to activate the defense system and protect the fruit against future pathogen attacks. The information generated by omics technologies allows knowing, in a global manner, the specific response in plants to each kind of elicitor and, based on that weigh-up, designing sustainable, eco-friendly strategies for disease control in fruits of agro-industrial interest.

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