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Pro-active application of UV-B light could protect strawberry fruits against the postharvest gray mold

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ABSTRACT

The severity of *Botrytis cinerea* growth could be significantly inhibited in mature strawberry fruits by priming fungal resistance in harvested berries after the proactive application of UV-B light (312 nm). Harvested fruits were irradiated by accumulative UV-B light amounts (0, 0.33, 0.65, and 1.3 mJoule/cm^2) before being artificially infected with *B. cinerea*. PCR assay could estimate a reduction ratio of 50 % in fungal DNA in fruits proactively exposed to 1.3 mJoule/cm^2 of UV-B light compared to non-UV-B treated fruits (0 mJoule/cm^2), which were harshly rotted with velvety gray mold growth. To reveal a side of possible molecular mechanisms behind UV-B induced immunity in ripe berries against *B. cinerea*, the expression profiles of some resistance-related genes were quantified using real-time PCR on day 1 and day 2 after infection. UV-B significantly sharpens the expression of strawberry defense gene FaBG2-1 which encodes the fungal cell wall degrading enzyme β-1-3-glucanase. Furthermore, jasmonic acid (JA) biosynthesis key gene FaAOS was strongly up-regulated after exposure to UV-B in infected or non-infected fruits on day 2. Equally important, the expression of FaNES1, volatile terpenoids linalool/nerolidol synthase gene, was moderately elevated. Abscisic acid (ABA) signaling gene FaPYR1 was found to be more responsive on day 2 after UV-B irradiation. Collectively, we hypothesize that UV-B proactive irradiation can encourage the enhancement of the molecular immune system in mature strawberry fruits against possible *B. cinerea* infection postharvest. Further empirical studies are needed to reveal UV-B potential as a sustainable priming agent to plant resistance against diseases.

Keywords: Strawberry; *Botrytis cinerea*; UV-B; gene expression

INTRODUCTION

Strawberry (*Fragaria* × *ananassa* Duch.) is a high-value cash crop all over the world. Furthermore, its short growth and fruiting period could facilitate conducting systematic studies on growth and developmental processes, which makes it a reliable model plant for Rosaceae (Han *et al*., 2019). Nevertheless, ripe harvested fruits are very soft and characterized by a short shelf life postharvest, often less than 5 days. It is very susceptible to rapid dehydration, physiological disorders, mechanical damage, and infections caused by several pathogens that can quickly reduce the quality of ripe fruits. These difficult factors limit strawberry production and prevent desirable export market success (Sallato *et al.,* 2007).

Fungi are the main microorganisms that can attack strawberry plants, causing critical acute diseases at various developmental stages (Garrido *et al*., 2016). Gray mold (caused by the necrotrophic fungus *Botrytis cinerea*) is one of the most serious problems for strawberry production and cultivation worldwide, leading to economic losses in the field, at harvest time, during marketing and exportation (Embaby *et al*., 2016). Unlike immature green or white fruits, mature red strawberry fruits are extremely vulnerable to *B. cinerea* infection. It is highly thought that strawberry fruit ripening process is accompanied by a severe reduction in defenses against *B. cinerea* thus increasing susceptibility to fungal development (Williamson *et al*., 2007). Several physiological and structural changes occur during the ripening process and lead to a severe attenuation in fungal resistance as a result of a reduction in cell wall firmness, increased sugar content, anthocyanin, water, and organic volatile compounds (van Kan, 2006; Haile *et al*., 2019; Petrasch *et al*., 2019; Neri *et al*., 2014).

Although they are more susceptible to gray mold fungal infection compared to immature fruits, mature strawberry fruits adopt several defense mechanisms relying on pathogen sensing and trigger defense responses (Amil-Ruiz *et al*., 2011; Petrasch *et al*., 2019). For example, the expression of FaGIP (encodes polygalacturonase inhibiting protein) was highly induced in mature strawberry fruits after *B. cinerea* infection (Mehli *et al*., 2004). *B. cinerea* necrotizing activity largely relies on producing a variety of pectinases for degrading plant cell walls, including exo and endo polygalacturonases (PGs) for absorbing water and sugars (Kars *et al.,* 2005). Thus, inhibiting such enzymes is essential for controlling fungal growth severity (Walton, 1994). Strawberry mature fruits could also resist fungal virulence and inhibit fungal growth rates by generating antifungal compounds as secondary metabolites or reactive oxygen species (Petrasch *et al*., 2019). Nevertheless, so far, no monogenic resistant strawberry line has been achieved, which raises the difficulty of developing resistant strawberry cultivars through classical and molecular breeding programs (Bestfleisch *et al*., 2015).

Despite their well-known negative effects on health and the environment, chemical synthetic fungicides are still the most widely used crop protection strategy worldwide and appear to be essential for securing the global food supply (Badmi *et al.,* 2019). The periodic and continuing use of fungicides (ex: dicarboximide, iprodione, and hydroxyanilide fenhexamid) has led to the development of resistant fungal strains all over the globe (Grabke, 2014; Xu *et al*., 2019). Therefore, it is of

great significance to develop sustainable and efficient alternatives to fungicides to help control crop diseases and, in parallel, keep the environment free of toxic hazards. UV-B light (280–315 nm) is a type of solar radiation that reaches the earth and is harmful to all living organisms, including plants (Robaa, 2004). Ground levels of UV-B are elevated due to the depletion of the ozone (O_3) protective layer as a result of global emissions of ozone-depleting substances (Williamson *et al.*, 2014). While UV light is generally known as a germicide physical agent for reducing fungal growth in strawberry tunnels (Onofre *et al.,* 2022), UV-B light can specifically activate a series of defensive molecular signaling cascades in plants, thus altering their response to surrounding pathogens (Meyer *et al*., 2021). In this study, we conducted phenotypical and molecular studies to validate the hypothesis of whether UV-B light could control the fungal infection severity of gray mold in ripe strawberry fruits postharvest, not as a mere germicide but as a priming agent for plant biological natural resistance.

MATERIALS AND METHODS

Plant materials:

Strawberry (*Fragaria* x *ananassa* Duch.) plants cv. Fertona was cultivated and nursed in net-house unit at Agricultural Genentic Engineering Research Institute (AGERI), Agriculture Research Center (ARC), Giza, Egypt . The experiment's location latitude is 30º; 01'; 27'' N, and the longitude is 31º; 12'; 11.3''E. The daughter plants were cultivated in two rows with 30 cm in between under a drip irrigation system. All cultural practices were performed according to Ministry of Agriculture and Land Reclamation (MALR) Egypt. Mature ripe healthy fruits were manually harvested and individually (one by one) washed under very weak current tap water to remove any mud residues by hanging each fruit from the peduncle to facilitate the rinsing process without causing outer tissue damage. Subsequently, the fruits were subjected to several rounds of gentle rinsing in sterile distilled water under hood insulating conditions. The fruits were allowed to be air-dried for a maximum of 30 min under the hood and then subjected to UV-B irradiation or artificial infection treatments.

Fungal suspension preparation:

The strain of *Botrytis cinerea* was isolated from rotten strawberries showing rotted fruit with velvety gray mold growth, by placing infected tissues onto a solid PDA (potato dextrose agar) medium and transferring fungal mycelium onto a new PDA plate. Finally, a single spore (obtained by the dilution method) of isolated purified fungi was transferred to a PDA slant medium and kept at 5°C. Isolated fungi were identified in the Plant Pathology Department, Faculty of Agriculture, Cairo University (Jarvis, 1977; Barnett and Hunter, 1987).

Serial doses of UV-B radiation:

The UV-B source was derived from UV-B lamps (312 nm) installed in the cross-linker GS Gene Linker® UV chamber (BioRad-USA). Strawberry ripe fruits were positioned in the center of the chamber with an area of 763.97 cm^2 at a distance of 15.2 cm from the UV lamps. Control fruits were kept in the dark for 17 min (the same period of UV-B treatment of 1.3 mJoule/cm^2) and were referred to as 0 mJoule/cm^2 of UV-B dose treatment. The UV-B-treated fruits were subjected to the following doses: 0.33, 0.65, and 1.3 mJoule/cm^2 of UV-B light energy. The energy of applied UV-B light was measured by the cross linker and divided by the chamber area to calculate light energy units in mJoule per cm^2. The control and UV-B irradiated fruits were subjected to the artificial infection of *B. cinerea* by applying 30 µl of freshly prepared conidial suspension (1 x 10^6 conidia/ml) to the surface of the fruits. Following that, all treated fruits were incubated for 5 days at 23 °C until infection symptoms appeared. The incubation time was extended to 7 days with a decreased amount of spores suspension by pipetting 5 µl of 1 x 10^6 conidia/ml suspension on the surface of ripe strawberry fruits. The infection solution was deposited by dropping it on an intact fruit surface or inside a small aperture (5 x 5 mm, diameter x depth), which was manually made by a sterile yellow tip bottom.

Phenotypic evaluation of damage assessment of *B. cinerea*:

The exogenous shape of the treated fruits was documented after 5 and 7 days of the treatments using a high-resolution camera (Nikon d7500, Japan). In addition, rotted spots that appeared on the surface of infected fruits were visually examined under the stereoscope SZ61 (Olympus, Japan) using a magnification power of 25X.

Molecular diagnoses of *B. cinerea* fungal infection:

Isolation of fungi/strawberry mixture DNA from fruits

At the end of the incubation period (5 days), all strawberry fruits were divided longitudinally into two halves, and then the upper infected halves were dissected and immediately flash-frozen in liquid nitrogen and subjected to genomic DNA extraction procedures by the DNeasy plant mini kit (QIAGEN, Germany) according to manufacturer's protocol.

Molecular quantification of *B. cinerea* infection:

The molecular quantification of *B. cinerea* in fruits was carried out by the quantification of fungal DNA levels relative to host plant DNA. This was achieved by quantifying the fungal-specific Cutinase A signal PCR fragment (Z69264) relative to that of strawberry FaGAPDH2 (Aubert *et al*., 2015). The sequences of forward and reverse primers are provided in Table 1. For setting up PCR reactions, a 50 µl mixture was prepared as follows: 25 µl of amaR PCR master mix (GeneDireX, Taiwan), 3 µl of genomic DNA mix, 0.3 µl forward primer (10 µM), 0.3 µl reverse primer (10 µM), and nuclease-free water was added up to 50 µl. The Thermal cycling protocol used with T100™ Thermal Cycler (Biorad, USA) was as follows: initial denaturation step 95 ℃ for 2 minutes, 35 cycles of denaturation step at 95 ℃ for 1 min, annealing step at 58 ℃ for 1 min, extension step at 72 ℃ for 1 min. The final extension step was done at 72 ℃ for 10 min. The reaction was stopped by incubating the tubes at 4 ℃ for at least 30 min. The PCR product was electrophoresed and then visualized on a 1.5 % agarose gel stained with Ethidium Bromide (10 mg/ml). The agarose gel was photographed by the gel documentation system Bio-Rad, USA. The quantification analysis was performed according to Hazman (2022). DNA isolated from *B. cinerea* mycelium was used as a positive control for validating reaction specificity.

Table 1. The sequences of forward and reverse primers for the genes investigated in this study, in addition to gene accession number and product size for each PCR amplicon.

Gene name	Accession number	Gene Function	Forward sequence (5' to 3')	Reverse sequence (5' to 3')	Amplicon size (bp)
FaBG2-1	AY170376	Degrading fungi cell walls	TTCTGTTTATGCCCGCTCTAC	TGGCTCATAAATCCTCATCCTTC	146
FaPYR1	JF268669.1	Abscisic acid signaling	CACGTGACCGGTTTCAGTATTA	CCACGTACGATTCCAGAACAA	124
FaGAPDH2	AF421493.1	Plant genomic DNA level/reference gene	CTTGAGAAGAAGGCCACCTATG	CTTCGGTGTAACCCAAGATACC	200/91
FaAOS	XM_004291875	Jasmonic acid biosynthesis	CACCCTTCCTGTCTTCTCAAC	GCTAAGGAAGAAGTAGAAGCCATT	100
FaNES1	KX450224.1	Biosynthesis of terpenoids volatile compounds	TGGGACGATTTAGGAAGTGC	TGAATGATGCTGGAAATGGA	193
Cutinase A	Z69264	Quantification of fungal DNA level in plants	GATGTGACGGTCATCTTTGCCC	AGATTTGAGAGCGGCGAGG	90

Gene expression profile of fruits defensive genes in response to *Botrytis cinerea* under the auspice of UV-B light:

The UV-B and artificial infection treatments applied to ripe strawberry fruits for gene expression analysis were designed to be as follows: control (not exposed to UV-B, not infection), UV (exposed to UV-B but not infected), Inf. (not exposed to UV-B (dark) but infected), and UV+Inf. (exposed to UV-B light then infected). The UV-B dose applied in this treatment was 1.3 mJoule/cm^2 and fungal infection was applied by positioning 30 µl of *B. cinerea* (1x10^6 conidia/ml sterile distilled water). Samples were collected at two different time points: 1 and 2 days after infection (referred here to as Day 1 and Day 2, respectively).

Total RNA isolation and cDNA synthesis:

The total RNA from all samples was isolated and genomic DNA was in-column digested using Spectrum^{TM} Plant Total RNA Kit (Sigma-Aldrich, USA) according to the manufacturer manual. cDNA was synthesized from 300 ng of total RNA using the RevertAid First Strand cDNA synthesis kit (Thermo Scientific, Lithuania) according to the supplied protocol. The synthesized cDNA was diluted 1 to 5 with nucleases-free water and used for subsequent gene expression analysis.

Real-time PCR:

Real-time PCR (quantitative PCR) was performed using Stratagene Mx3000P (Stratagene, USA). The reaction was performed in a final volume of 10 µl containing 1.5 µl of diluted cDNA, 0.5 µl of each forward primer and reverse primers (10 µM each), 5 µl 2X JumpStart Taq Ready Mix, and 2.5 µl RNase free water. The mixture was gently pipetted and briefly centrifuged then perform a thermal cycling program as follows: 94 °C for 2 min and 40 cycles (94 °C for 15 s and 66 °C for 1 min). The sequences of the forward and reverse primers for studied target genes and reference gene are listed in (Table 1). Quantification of relative expression values was calculated according to Pfaffi (2001).

Statistical analysis:

Testing of statistical significance of the mean values obtained was performed by Tukey's Honestly Significant Difference (HSD) test, with a significance level of $P \leq 0.05$. All analyzed data represented at least three biological replications.

RESULTS

Morphological response of UV-B irradiated strawberry fruits to *B. cinerea* fungal infection:

After 5 days of incubation at 23 °C, progressive UVB treatment could reduce the severity of *B. cinerea* fungal infection in ripe strawberry fruits postharvest Figs. (1 and 2). The applied UV-B doses were 0, 0.33, 0.65, and 1.3 mJoule/cm^2. The more UV-B proactive dose, the less observed fungal infection severity Figs. (1A and 2). Phenotypically, a UV-B dose of 1.3 mJoule/cm^2 leads to the most limited fungal growth compared to other lower doses. Indeed, the most noted severe fungal growth was in the case of 0 mJoule/cm^2 UV-B dose (i.e., not UV-B irradiated fruits), where the fungal growth was extremely severe and caused a full rotting of infected fruit. A UV-B dose of 0.65 mJoule/cm^2 partially diminishes fungal growth in a phenotypic appearance of between 0 and 1.3 mJoule/cm^2 Figs. (1A and 2). While UV-B light with a dose of 1.3 mJoule/cm^2 phenotypically diminished fungal growth with an infection volume of 30 µl, the same dose was able to completely prevent the appearance of fungal infection symptoms using a lower amount of fungal suspension (5 µl), yet with an extended incubation period to 7 days. Fruit protection was achieved by applying a UV-B dose of 1.3 mJoule/cm^2 where the fungal infection using 5 µl of conidia suspension takes place on the surface of the fruit or inside its tissue through a pre-made mechanical wound Fig. (3A and B).

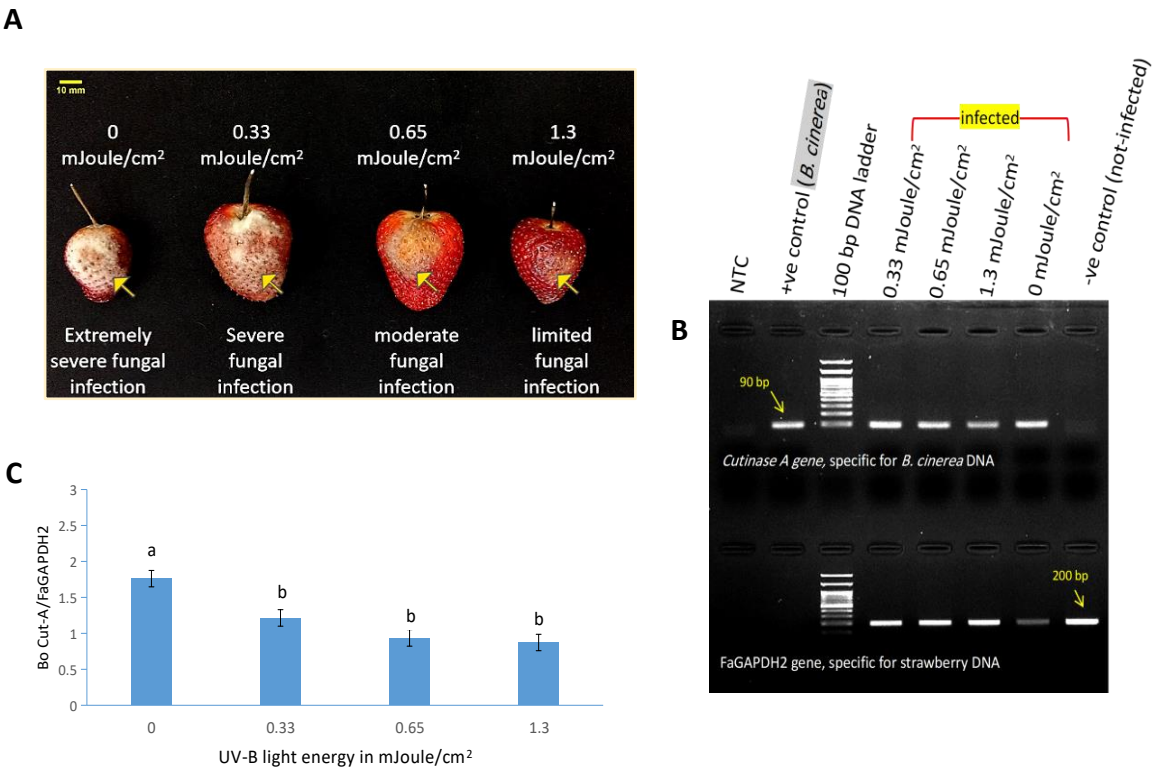

Fig. 1 The effect of UV-B priming on elevating ripe strawberry resistance level against *B. cinerea* infection. **A** phenotyping of inhibiting the severity of *B. cinerea* fungal infection on strawberry fruits by elevating UV-B priming dose. Scale bar equals 10 mm. **B** Agarose gel image representing the migration of *Cutinase A* gene PCR amplicon (90 bp) specific for *B. cinerea* (upper lane), and FaGAPDH2 PCR amplicon specific for strawberry DNA (200 bp, lower lane). **C** Quantification of in-plants growth of *B. cinerea* by conventional PCR. Values shown are the means of three replications ± SE. n= 6-8 in each treated replicate. Means with the same letters are not significantly different according to Tukey's Honest Significant Difference (HSD) test ($p \leq 0.05$).

Molecular quantification of *B. cinerea* fungal growth severity:

The level of fungal DNA relative to strawberry DNA (host plant tissue) was used to estimate fungal DNA levels in UV-B irradiated fruits after applying 30 µl of fungal spores solution and an incubation time of 5 days at 23°C. For doing so, *Botrytis cinerea* specific genomic Cutinase A fragment was successfully amplified at the size of 90 bp in all infected fruits in addition to positive control using conventional PCR assay Fig. (1B, upper lane). By increasing the applied UV-B dose, the intensity of the Cutinase A PCR amplicon band decreases. The technical credibility of the reaction was assured by considering No Template Control (NTC) and the non-infected strawberry fruit (negative control). Additionally, strawberry genomic FaGAPDH2 fragment (200 bp) was successfully amplified. Visually, the intensity of the FaGAPDH2 fragment becomes slim in 0 mJoule/cm² treated fruits compared to control and UV-B irradiated ones Fig. (1B, lower lane), which was the most rotted fruit see (Figs.1 A and 2A). The quantification of fungal DNA in different infected samples was monitored by measuring the ratio of the intensity of either PCR amplicon band (Bo Cutinase A and FaGAPDH2) using the free software ImageJ by measuring the integrated density of bands. Then calculate the ratio of *Bo* Cutinase A/FaGAPDH2. In consistent with the phenotyping results Figs. (1A and 2), fungal DNA was reduced upon elevating the UV-B light dose. DNA of *B. cinerea* was reduced by 31.25 % with 0.33 mJoule/cm², 46.7 % with 0.65 mJoule/cm², and by 50 % with 1.3 mJoule/cm² UV-B proactive dose relative to 0 mJoule/cm² Fig. (1C).

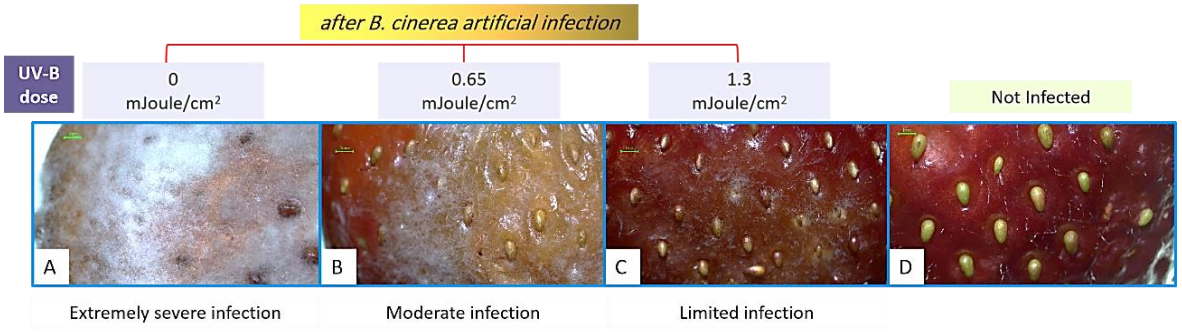

Fig. 2 Effect of UV-B proactive treatment in resistance enhancement against *B. cinerea* fungal infection. **A** severe fungal growth on strawberry fruit that has not been irradiated with UV-B but incubated in dark instead (0 mJoule/cm^2). **B** UV-B dose of 0.65 mJoule/cm^2 could clearly inhibit fungal growth comparing to "A". **C** represents a limited fungal growth of *B. cinerea* on strawberry fruits irradiated with 1.3 mJoule/cm^2 UV-B. A representative image for healthy fresh harvested non-infected strawberry fruit was presented in **D**. Images were acquired with Stereoscope Olympus SZ61 (Olympus, Japane) with 25X magnification power. Scale bar equals 1 mm.

The relative expression profiles of several defense-related genes:

As shown by investigated phenotypical and molecular confirmation tests, 1.3 mJoule/cm^2 was the most effective UV-B dose in inhibiting *B. cinerea* infection Figs. (1 and 2). Therefore, this dose was used for studying early molecular defense response against *B. cinerea* infection under the auspice of UV-B proactive treatment. The experiment comprised the following treatments: control (not UV-B irradiated, not infected), UV (UV-B irradiated but not infected), Inf. (artificially infected but not UV-B irradiated), and UV+Inf. (UV-B treated then artificially infected).

We have measured the expression of four selected genes at two time points (day 1 and day 2 after infection) using quantitative PCR assay. The transcriptional response of the anti-fungal gene FaBG2-1 (encodes β-1-3-glucanase enzyme) was quantified Fig. (4A). The enzyme β-1-3-glucanase can degrade the cell walls of several pathogenic fungi, including *B. cinerea*. As shown in Fig. 4A, FaBG2-1 expression was strongly elevated in both time points in response to UV-B irradiation, however, with a higher level on day 1 than on day 2 compared to corresponding controls (132-fold and 12.26-fold increases, respectively). The fungal infection (Inf.) heightened the expression of FaBG2-1 after 1 day of infection by 48-fold, but the expression level was severely decreased after two days. In UV+Inf. treatment, the expression was significantly elevated only on day 2 compared to control and fungal infection treatments. Furthermore, the key marker gene allene oxide synthase (biosynthesis of the plant stress hormone jasmonic acid) FaAOS expression was also studied. The transcription level of FaAOS was strongly enhanced upon UV-B treatment on day 1 and day 2 compared to corresponding controls, with 77 and 30-fold increases, respectively. The gene was not responsive to a mere fungal infection at both time points. Interestingly, UV+Inf. treatment could significantly elevate the expression of FaAOS on day 2 only. The fold of the induction ratio on day 2 compared to day 1 was 21.7 times Fig. (4B).

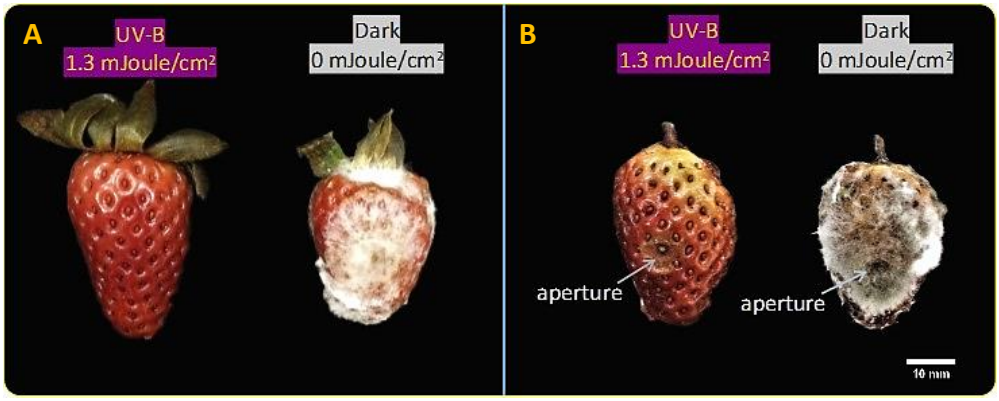

Fig. 3 Visual aspect of *B. cinerea* growth severity on the 7th day after infection under the auspices of the priming effect of UV-B of 1.3 mJoule/cm^2 using 5 µl of fungal suspension (1x10^6 spores/ml sterile distilled water). **A** and **B** demonstrate the absence of any exogenous symptoms referring to fungal growth in UV-B treated ripe strawberry fruits versus severe fungal growth in dark-incubated (0 mJoule/cm2 UV-B dose) fruit. In **A** the deposition of inoculum droplets was applied on the surface of the fruits, while in **B** a small aperture (≈ 5 x 5 mm, diameter x depth) was made for containing the fungal suspension droplet. Scale bar equals 10 mm. n= 5-8 in each treated replicate.

The relative expression of FaNES1 (a key enzyme in producing terpenoids volatile compounds) on day 1 and day 2 after infection was presented in Fig. 4C. On day 1, the gene was responsive to all treatments, with an average value of a 2-fold increase. After two days, the gene expression level was ceased in Inf. treatment by 95.6 % reduction ratio relative to day 1 Inf. treatment. On the other hand, the relative expression ratio was significantly elevated in response to UV-B treatment (UV) relative to day 1 by a 2.3-fold increase. The UV+Inf. strawberry fruits accumulated higher amounts of FaNES1 transcripts compared to Inf. treated strawberries. It is worth mentioning that the relative expression of FaNES1 increased on day 2 by 2.85-fold compared to day 1 under control conditions. Fig. 4D illustrates the expression profile of FaPYR1 (encodes for the ABA receptor pyrabactin resistance 1). On day 1, the relative expression level increased by 5.64 and 4.6-folds in response to UV-B and *B. cinerea* infection (Inf.) compared to control samples, respectively. On day 2, all applied treatments significantly down-regulated the expression level compared to control conditions. Similar to FaNES1, the level of FaPYR1 expression was significantly increased on day 2 by nearly 7-fold induction relative to day 1 in control fruits. However, the UV+Inf. treatment accumulated higher transcripts of FaPYR1 compared to Inf. treated fruits.

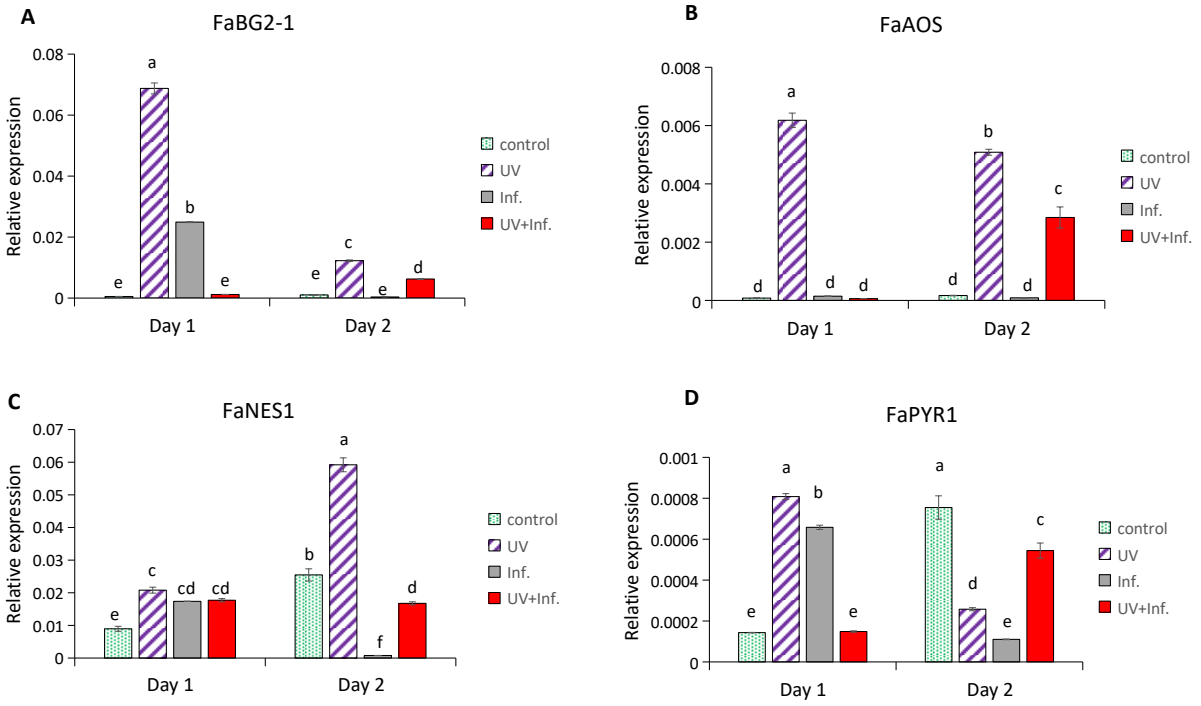

Fig. 4 Alternation in transcripts accumulation for **A**: FaBG2-1 (encodes β-1-3-glucanase enzyme), **B**: FaAOS (encodes the enzyme allene oxide synthase), **C**: FaNES1 (encodes nerolidol synthase enzyme), and **D**: PYR1 (encodes the ABA receptor pyrabactin resistance 1) in response to the artificial infection of *B. cinerea* of ripe strawberry fruits postharvest under the auspice of the priming effect of UV-B irradiation. Expression of genes was measured on day 1 and day 2 after infection treatment. Relative expression was determined using FaGAPDH2 as a reference gene. n= 4-6 in each treated replicate. Values shown are means of three replications ± SE. Means with the same letters are not significantly different according to Tukey's Honest Significant Difference (HSD) test ($p \leq 0.05$).

DISCUSSION

Gray mold, caused by the necrotrophic fungus *Botrytis cinerea*, is the most destructive postharvest disease worldwide for ripe strawberry fruits (El-Ghanam *et al*., 2015; Garrido *et al*., 2016). So far, the massive application of fungicide is the most widely adopted crop protection strategy (Badmi *et al*., 2019). Nonetheless, fungicide use is not a preferred plant protection trend due to well-documented negative effects on non-target organisms such as soil microbes, aquatic organisms, beneficial insects, and mammals (for a review, see Zubrod *et al.,* 2019). The situation is getting even more serious with the generation of a wide range of fungicide-resistant fungi strains in different agroecosystems all over the globe due to the massive applications of one kind of fungicide for an extended period (Panebianco *et al*., 2015). To cope with such a challenge, several alternative solutions were developed to offset the high spread rate of *B. cinerea* infection in strawberries. The most well-acknowledged substituting strategy for fungicide is biocontrol agents (BCAs), which include antagonistic microorganisms such as bacteria and fungi with the ability to inhibit *B. cinerea* fungal growth. Unfortunately, BCAs efficacy is not comparable to traditional organic synthetic fungicides (Pertot *et al*., 2017). Therefore, it is imperative to keep developing new efficient sustainable practices for controlling fungi infection in crop plants.

UV-C treatment (100-280 nm) is frequently used as a disinfecting physical agent to terminate some plant pathogenic fungi such as powdery mildew and *B. cinerea* growth conidia on strawberry fruits. However, the exposure of strawberry plants to UV-C postharvest might pose potential damaging effects on plants as severe oxidative stress and deformation in the reproductive organs of the flowers (Janisiewicz *et al*., 2016; Jaramillo-Sánchez *et al*., 2021; Onofre *et al*., 2021). On the other hand, UV-B (280-315 nm) light can be specifically perceived by plants through the UV-B photoreceptor UVR8 (UV-B RESISTANCE 8), leading to the triggering of molecular and biochemical defenses against wide range of pathogens and pests (Meyer *et al*., 202; Stratmann, 2003). In this study, the progressive or accumulative application of UV-B light doses (0, 0.33, 0.65, and 1.3 mJoule/cm^2) inhibited *B. cinerea* fungal growth on ripe strawberry fruits Figs (1, 2, and 3). The most effective UV-B dose was 1.3 mJoule/cm^2. Our results are in accordance with (Kanto *et al*., 2014), who reported the reduction of powdery mildew growth of ripe strawberries using UV-B light energy of ca. 2-7 kJ m^{-2} d^{-1}.

The effect of UV-B on the physiological and biochemical aspects of the fruits was not studied in this work. However, it is speculated that the irradiated fruits will not be damaged by UV-B light in the event of proper treatment. The shape of the irradiated fruit looked decent and was not deformed Fig. (3A and B). In Fig. 2, the not-spoiled area of fruits irradiated with 0.65 mJoule/cm^2 remained healthy and not stunted, providing that the incubation temperature was 23 °C for 5-7 days, where strawberry ripe fruits could be spoiled swiftly (Sallato *et al*., 2007). UV-B irradiation could even enhance free sugar levels in ripe fruits (Kanto *et al*., 2014) and caffeic acid derivatives and growth parameters in *Echinacea purpurea* callus and suspension

culture (Manaf *et al*., 2016). We hypothesize that treating ripe strawberry fruits with short-term UV-B before transportation or during it would possibly delay fruits decay due to inhibited gray mold disease severity.

The adopted artificial infection protocol was applied through the inoculation of 5 and/or 30 µl of *B. cinerea* conidia solution (1x10^6 conidia/ml sterile water) on the upper surface area of ripe fruits. For inoculated strawberry fruits with 30 µl, the quantity of conidia per fruit is 30,000 (Figs. 1 and 2), while it was 5000 conidia per fruit in the case of 5 µl inoculum droplets (Fig. 3) (Aubert *et al*., 2015; Jin *et al*., 2017). The amount of inoculum droplet affected the growth of fungi and thus infection severity. In similar UV-B treatments, many reports used lower amounts of *B. cinerea* conidia for artificial infection. (Janisiewicz *et al*., 2015) inoculated UV-C treated ripe strawberry fruits with 25 µl of 1x10^4 conidia/ml solution, which makes it 250 conidia/fruit. (Similarly, Vega *et al*., 2020) inoculated cut roses with 20 µl of 1x10^4 conidia per ml (200 spores/fruit). In a conclusion, the efficacy of the priming effect of UV-B light was greatly increased with lower to moderate fungal infection.

The molecular priming response was investigated in this work by studying the quantitative gene expression profiles of several resistance marker genes. The UV-B light energy used in this experiment was 1.3 mJoule/cm^2; the amount that showed the highest phenotypical inhibition of fungal infection Figs (1-3). Plant defense response to pathogens as fungi is very complex as it embraces structural and biochemical alternations (Simmons, 1994). Pathogenesis-related proteins (PRs) are the main biochemical barriers that plants accumulate immediately after insect attack, fungal, bacterial, and even viral infection (Amil-Ruiz *et al*., 2011). UV-B treatments activated the enzyme -1,3-glucanase, a PR protein essential for degrading invading fungi cell walls in rice and strawberries (He *et al.,* 2018; Kanto *et al.,* 2014). The expression profile of FaBG2-1 (encodes β-1-3-glucanase enzyme) showed that FaBG2-1 was strongly upregulated after one day of UV-B treatment by 132-fold compared to control conditions. The Infection treatment (Inf.) induced the gene expression by 48-fold increase Fig. (4A). (Pombo *et al.,* 2011) reported an elevated expression ratio of FaBG2-1 in UV-C irradiated ripe strawberry fruits in early storage periods (4 hours after treatment) that gets lower at the end of the storage period (48 hours). Besides ripe strawberry fruits, FaBG2-1 mRNA levels increased in UV-C treated strawberry leaves (Xu *et al*., 2019). On day 1, the relative expression level of FaBG2-1 in UV+inf. treatment was not significantly different from control. Although not certain, this might be attributed to the possibility of being suppressed by the fungus upon artificial infection. (Houterman *et al*., 2008) reported that the xylem-invading fungi *Fusarium oxysporum* can suppress the protective effect of some resistance proteins in tomatoes. The same phenomenon was observed in wheat by the fungus *Zymoseptoria tritici* (Seybold *et al*., 2020).

Jasmonic acid and its biologically active derivatives (collectively known as jasmonates or JAs) are essential molecular tools in shaping the defensive response to a broad spectrum of pathogens and insects (Hazman *et al*., 2019; Mouden *et al*., 2021). (Qi *et al*., 2018) reported that UV-B exposure for five days significantly elevated jasmonic acid hormone (JA) and synthesizing genes expression as AtOPR3 in *Arabidopsis*, and NtAOC, OsLOX, and ZmJAR1 in tobacco, rice, and maize, respectively. Here, the relative expression ratios of the key jasmonic acid biosynthesis genes allene oxide synthase FaAOS were strongly elevated in fruits exposed to UV-B light at either time point (Fig. 4B). Given the phenotype of inhibited fungal growth severity in UV-B treated fruits Figs. (1 and 2), it could be postulated that UV-B light (and UV-C) elevates plants' resistance by urging them to accumulate several defense-related molecules such as jasmonic acid and its derivatives (Escobar Bravo *et al*., 2019; Phonyiam *et al*., 2021; Qi *et al*., 2018). Recently, (Li *et al*., 2022) reported that *B. cinerea* infection in sweet cherry could be controlled through the activation of jasmonic acid biosynthesis in response to Mushroom volatile alcohol (also called 1-octen-3-ol). Interestingly, *B. cinerea* infection in the absence of UV-B light could not elevate the expression of FaAOS at either examined time point. This could be linked to two possible explanations, i) the extremely low immunity level of fully ripe strawberry fruits to *B. cinerea* infection and other pathogens (Williamson *et al*., 2007). ii) The ability of invading fungus to suppress the expression of some plant resistance key genes to facilitate infection and attenuate potential resistance (Feng *et al.,* 2020).

Volatile compounds in strawberries are extremely essential in detecting cultivar value, sweetness, and customer liking even, thus fruit price (Fan *et al*., 2021). Equally important, volatile compounds serve as both an attractant fruit marketing agent and as secondary metabolites that protect against pathogens and insects (for a review, see Amil-Ruiz *et al.,* 2011). Terpenoids (such as linalool and nerolidol) are part of a wide range of many volatile compounds that collectively produce the aromatic profile of strawberry fruits (Mishra and Kar, 2014; Yan *et al*., 2018). FaEDS1 is an important mediator in strawberry innate immunity by affecting salicylic acid signaling (Feng *et al*., 2020) and was found to be induced in response to UV-C in strawberry leaves (Xu *et al*., 2019). The applied UV-B treatment in this work induced the expression of FaNES1 (key gene in the biosynthesis of volatile terpenoids linalool/nerolidol) and with infected fruits (UV+Inf.) on day 2 Fig. (4C). Interestingly, the expression of FaNES1 in control fruits on day 2 was significantly higher than its level on day 1. This could be related to the possibility of influencing the quantitative level of the volatile compound by varying storage conditions (here at 23 °C). (According to Forney *et al*., 2000), the volatile content in fruit harvested after four days at 15 °C peaked after a 200-fold increase. Collectively, we suggested that UV-B might be able to encourage exposed strawberry fruits to accumulate higher levels of terpenoids. On the other hand, this could be beneficial by protecting ripe berries fruits from possible fungal infection, or possibly might alter the volatile profile of the fruits making them possibly more marketable. Further studies are needed for a better understanding of the effect of UV-B light on commercial characteristics of strawberry ripe fruits postharvest.

ABA (Abscisic acid) is a major plant hormone, which plays a central role in plant physiological processes, as well as responses to abiotic and biotic environmental stresses (Hu and Bidochka, 2021). FaPYR1 belongs to the ABA receptor family PYR/PYL/RCAR. These receptors are transcriptionally responsive and bind specifically with ABA during biotic stress to initiate downstream signaling mediated by ABA-activated SNF1-related kinases protein kinases or SnRK2 (García-Andrade *et al*., 2020). Both UV-B light and *B. cinerea* infection increased the relative expression level of FaPYR1 in a mature strawberry after one day of infection but then decreased after two days Fig. (4D). It was also observed that UV+Inf. treatment could enhance the gene expression on day 2 only compared to infected fruits (Inf.). These results are in accordance with (Xu *et al*., 2019). It

is worth mentioning that while some studies considered ABA accumulation as a resistance response to fungal or bacterial infections, others reported that the ABA hormone is a negative regulator that attenuates plants immunity against *B. cinerea* (García-Andrade *et al*., 2020; Lievens *et al*., 2017; Liu *et al*., 2015).

CONCLUSION

We asked whether protecting plants against the fungal disease gray mold could be achieved by urging plants themselves to trigger their biological resistance or defenses rather than applying synthetic chemical fungicides. Exposing ripe strawberry fruits to a proactive short-term course of UV-B light (280-315 nm) could inhibit *B. cinerea* fungal growth severity or prevent it. Ripe strawberry fruits could be successfully primed for fungal infection by inducing the expression of several defensive genes such as FBG2-1, FaAOS, and FaNES1 before possible fungal infection. These findings pave the way for further investigations into developing protective sustainable UV-B protocols for priming plants' defenses against different pathogens. This could help avoid the massive use of fungicides or other antifungal preservative synthetic chemicals.

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Authors' contributions

SA performed the experiments and produced the data. MH and FK proposed the idea, supervised the work, analyzed the data, and wrote the manuscript. MZ prepared fungal spores, performed infection treatments, and revised the data. SH and AH supervised the work and revised the manuscript. All authors read and approved the final manuscript.

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Conflict of Interest

The authors declare no conflict of interest.

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التفعيل الإستباقي للأشعة فوق البنفسجية- النوع ب قد يحصن ثمار الفراولة ضد العفن الرمادي بعد الحصاد

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الملخص

من خلال تلك الدراسة تم تقليل حدة نمو فطر العفن الرمادي في ثمار الفراولة الناضجة بعد الحصاد وذلك من خلال تحسين دفاعات الثمار قبل الإصابة عن طريق تعريضها لجرعات إستباقية من الأشعة فوق البنفسجية-النوع ب. لتحقيق ذلك، تم معاملة الثمار الناضجة بجرعات متراكمة من الأشعة فوق البنفسجية-النوع ب (0, 0.33, 0.65, 1.3 مل جول/سم^2) ثم إحداث العدوي معمليا بتلقيح سطح الثمار بـ30 ميكروليتر من محلول فطر بوتريتس سينيريا المسبب لمرض العفن الرمادي بتركيز 1 × 10 ^{6} كونيديا/مل. وقد وُجد ان جرعة الأشعة 1.3 مل جول/سم^2 قد تمكنت من تثبطت نمو الفطر بشكل أكبر مما في حالة الثمار المعاملة بجرعات أقل .وعلي المستوي الجزيئي، وجد أن مستوي المادة الوراثية DNA للفطر قد انخفض بنسبة قاربت الـ 50 % في حالة الثمار التي عوملت بالأشعة فوق البنفسجية-النوع ب مقارنة بتلك التي لم تعامل (0مل جول/سم^2). وبواسطة تقنية تفاعل البلمرة المتسلسل اللحظي تم تقدير التعبير الجيني كمياً لعدد من الجينات المرتبطة بدفاعات ثمار الفراولة ضد الأمراض الفطرية وهم جين FaBG2-1 وجين FaAOS1, وجين FaNES1. وقد لوحظ أن التعبير الجيني لجميع تلك الجينات يزداد بشكل معنوي في أنسجة ثمار الفراولة عند تعرضها للأشعة فوق البنفسجية-نوع ب مما قد يفسر قدرتها على تثبيط النمو الفطري بعد العدوى. وفى الختام، فإن تعريض ثمار الفراولة الناضجة لجرعات محسوبة من الأشعة فوق البنفسجية-نوع ب بشكل استباقي قبل حدوث العدوي المحتملة لفطر البوتريتس قد يساهم في حمايتها من مرض العفن الرمادي بعد الحصاد. وينبغي إتمام المزيد من الدراسات لفهم التأثير المحتمل للأشعة فوق البنفسجية-ب كعامل مستدام محفز لدفاعات النباتات ضد الأمراض الفطرية.

الكلمات المفتاحية: الفراولة، فطر البوتريتس، الأشعة فوق البنفسجية-نوع ب، التعبير الجيني