

Shortwave Ultraviolet Irradiation for Control of Decay Caused by *Botrytis cinerea* in Bell Pepper: Induced Resistance and Germicidal Effects

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ABSTRACT. Shortwave ultraviolet radiation (UV-C) was tested for controlling natural infections and inducing resistance to fungal decay caused by *Botrytis cinerea* Pers.: Fr. (gray mold rot) in bell pepper [*Capsicum annuum* L. var. *annuum* (Grossum Group)] fruit. All UV-C doses tested (0.22, 0.44, 0.88, or 2.20 kJ·m⁻²) caused a reduction in the number of natural infections occurring during storage at 13 °C. A UV-C dose of 0.88 kJ·m⁻² controlled most effectively natural infections in peppers stored at both 13 or 20 °C. Although UV-C was found to be highly germicidal to *B. cinerea* conidia exposed on agar or on fruit wounds, it did not prevent infection of fruit inoculated with the pathogen 24 hours before exposure to UV-C. However, fruit which were exposed to UV-C 24 hours before inoculation with *B. cinerea* had a lower percentage of infections. For this reason, UV-C appears to act mainly as an inducer of disease resistance in this crop rather than a sanitizing agent. UV-C was effective in inducing resistance to *B. cinerea* in fruit at various stages of maturity, from green to red. Disease resistance was also induced in fruit which had been stored for 7 days before UV-C treatment. The effect of UV-C doses was found to be additive as two successive exposures at 0.44 kJ·m⁻² had an equivalent effect as one exposure to the optimal dose of 0.88 kJ·m⁻². However, two successive exposures to 0.88 kJ·m⁻² were less effective than one exposure to this dose.

Fresh bell pepper fruit [*Capsicum annuum* var. *annuum* (Grossum Group)] are highly perishable after harvest with a storage life of <3 weeks under optimum storage conditions of 7 to 13 °C and 90% to 95% relative humidity (RH) (Hudson et al., 1985). Microbial deterioration of bell pepper can occur during storage or shipping and gray mold rot caused by *Botrytis cinerea* was reported as the most common fungal disease in bell pepper shipments arriving at the New York City market (Ceponis et al., 1987). Bell pepper can also be infected by other fungi such as *Alternaria* Nees (alternaria rot), *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. in Penz. f. (anthracnose), *Fusarium solani* (Mart.) Sacc. (fusarium fruit rot), *Penicillium* Link: Fr., and *Sclerotinia sclerotiorum* (Lib.) de Bary (sclerotinia rot) (Ceponis et al., 1987).

Currently, there is no fungicide registered for postharvest treatment of bell pepper in Canada and nonchemical methods would be desirable to improve storage life of this commodity. Since bell pepper harvested at the mature-green stage suffer chilling injury at temperatures <7.5 °C (Hughes et al., 1981; Wang, 1977), low temperature storage cannot be fully exploited. Modified atmosphere packaging has been used for storing pepper fruit but elevated CO₂ concentrations can increase the incidence of microbial decay (Hughes et al., 1981; Meier et al., 1995). Recently, Fallik et al. (1996) reported that hot water treatment of

red bell pepper could be beneficial in controlling infections by *B. cinerea* and *Alternaria alternata* (Fr.: Fr.) Keissl. (alternaria fruit rot).

Prestorage exposure to shortwave ultraviolet radiation (UV-C) has been shown to control fungal decay in several stored commodities (Baka et al., 1999; Lu et al., 1987; 1991; Stevens et al., 1990). Control of storage diseases by UV-C could result from induction of disease resistance as well as killing or inactivation of pathogens by irradiation. In stored carrots (*Daucus carota* L.), citrus (*Citrus* L. sp.), and tomatoes (*Lycopersicon esculentum* Mill.), induced resistance to storage pathogens by UV-C was shown to be related to accumulation of phytoalexins (Ben-Yehoshua et al., 1992; Charles et al., 2001; Mercier et al., 1993a, 2000; Rodov et al., 1992). Although control of natural infections by UV-C could result from the germicidal properties of UV-C on plant pathogens, this possibility does not appear to have been investigated. Previously, induction of disease resistance was reported in bell pepper fruit treated with a fungal wall hydrolysate (Adikaram et al., 1988). Inducing resistance with UV-C could be a more practical way of making use of natural defenses of a host for improving shelf life of this commodity.

The objective of this study was to investigate the possibility of controlling gray mold rot of stored bell pepper fruit with UV-C. Two possible modes of action of UV-C, induced disease resistance and germicidal effects on the pathogen, were investigated. Furthermore, factors such as treatment delay after harvest, fruit maturity and intermittent exposure to UV-C, which can affect the induction of disease resistance, were also studied.

Materials and Methods

PLANT MATERIAL. ‘Delphin’ or ‘Bell Boy’ bell pepper fruit were harvested from greenhouse-grown plants at Laval University. Fruit of ‘Bell Boy’ were used for experiments on control of natural infection. Fruit of ‘Delphin’ were used for all other experiments. Plants were grown in peat with an extended pho-

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toperiod of 16 h, with high-pressure sodium lamps as source of supplemental lighting. Plants were fertigated daily with a complete nutrient solution (electrical conductivity = 2.0 to 2.8 mS·cm⁻¹, pH 5.5 to 6.0) using a drip irrigation space system. The greenhouse was maintained at minimal days/nights of 22/17 °C. Fruit were picked manually and selected for uniform size, color, and absence of external blemishes and visible infection. Fruit were used for experiments after 6 h storage at 13 or 20 °C, unless stated otherwise.

PATHOGEN CULTURE. *Botrytis cinerea* was isolated from infected strawberries (*Fragaria × ananassa* Duchesne) in our laboratory and maintained on potato dextrose agar (PDA) at 4 °C. Conidia of *B. cinerea* were obtained from 8-d-old PDA cultures incubated at 25 °C. To obtain conidia, the cultures were flooded with sterile distilled water containing 0.1% Tween 80 and rubbed gently with a glass rod. Hyphae were removed from the suspensions by filtering through two layers of cheese cloth. Conidia from the filtrates were recovered by centrifugation, washed three times with sterile distilled water, and finally suspended in sterile distilled water. The spore count was determined with a hemacytometer, and the concentration was adjusted with distilled water to 5 × 10⁵ conidia/mL.

UV-C IRRADIATION. Fruit were irradiated with UV-C lamps with peak emission at 254 nm (G3T8, General Electric, Mississauga, Ontario, Canada). Each fruit was rotated manually in four different positions to ensure exposure of the entire surface of the fruit to UV-C. UV irradiance was measured using a UV digital radiometer (Ultraviolet Products Inc., San Gabriel, Calif.), and the exposure time was varied to achieve the desired UV-C doses. Treated fruit were stored in the dark at 13 or 20 °C in plastic containers which were ventilated continuously with humidified air to maintain RH of ≈95%.

CONTROL OF NATURAL INFECTION BY UV-C. ‘Bell Boy’ fruit that had not been washed or surface-sterilized were treated with a UV-C dose of 0.0, 0.22, 0.44, 0.88 or 2.2 kJ·m⁻² and stored at 13 °C. The fruit were inspected daily to determine the number of infected fruit. Fruit showing any sign of infection were removed from their container to avoid spread of infection to adjacent fruit. Identification of the causal agents was made by observing lesions for signs of the pathogen, as well as microscopic examination. Disease incidence was expressed as the percentage of infected fruit.

The control of natural infections by UV-C was tested at 13 and 20 °C. Fruit were kept at 13 or 20 °C for 6 h before exposure to UV-C at 0.88 kJ·m⁻². They were then maintained at their respective temperature, except one group, which was treated at 20 °C but stored at 13 °C. For all experiments on disease control by UV-C, there were three replications for each treatment, and each replication consisted of eight fruit placed in separate containers in a completely randomized design.

PRE- AND POSTINOCULATION EFFECT OF UV-C. The possibility of fruit decontamination and induced disease resistance as mechanisms of action of UV-C were examined with fruit which were inoculated before or after UV-C treatment at 0.88 kJ·m⁻². In order to minimize natural infection, fruit of ‘Delphin’ were surface-sterilized for 3 min with 1% sodium hypochlorite and were subsequently rinsed three times in sterile deionized water and allowed to dry in ambient air. There were three treatment groups: one group was treated with a UV-C dose of 0.88 kJ·m⁻² and then inoculated with a conidial suspension of *B. cinerea* 24 h after treatment; the second group was inoculated with a spore suspension of the fungus and treated with 0.88 kJ·m⁻² 24 h after inoculation; and the third group of fruit serving as a control was

inoculated with a spore suspension 24 h after harvest. Each treatment consisted of three replications of eight fruit each in a completely randomized design. Inoculation of the fruit was performed on fresh wounds which were made by puncturing at 10 locations on one side with a sterile 0.5 mm diameter needle. Twenty microliters of conidial suspension (5 × 10⁵ conidia/mL) was introduced into each wound to a depth of 0.1 mm with a sterile needle. Infection was estimated by measuring the diameter of lesions on each inoculated fruit.

GERMICIDAL EFFECT OF UV-C ON *Botrytis cinerea*—IN VITRO. Aliquots of conidial suspensions of *B. cinerea* in sterile distilled water containing 0.1% Tween 80 were pipetted to potato dextrose agar plates and spread with a glass rod. Four groups of three plates were exposed directly to doses of UV-C at 0.0, 0.88, 2.2 or 4.4 kJ·m⁻². The plates were incubated at 22 °C in the dark for 24 h and germination of 100 spores per plate was determined microscopically. A conidium was considered germinated when the length of the germ tube was equal to or greater than the length of the conidia and germination was expressed as a percentage.

GERMICIDAL EFFECT OF UV-C ON *Botrytis cinerea*—IN VIVO. Fruit which were washed and surface sterilized as described above were wounded at three places around the stem scar using a sterile needle. Each wound was inoculated with 50 µL of conidial suspension. There were nine fruit per UV-C dose, exposed either right after inoculation (0 h) or 24 h later. After UV-C treatment, the inoculated sites were excised with a sterile razor blade and the tissue from three inoculated sites was transferred to 50 mL of sterile water in sterile plastic bags. The samples in each bag were gently ground with a Stomacher grinder (Lab blender 400, Seward Laboratory, London) for 3 min to recover the spores from the inoculated tissues. Aliquots of 50 µL were dispensed in petri plates containing potato dextrose agar and incubated at 22 °C for 24 h, after which, spore germination was determined. In the case of fruit exposed to UV-C 24 h after inoculation, the inoculated fruit were held at 13 °C and 95% RH until UV-C treatment was performed.

FACTORS AFFECTING THE INDUCTION OF DISEASE RESISTANCE BY UV-C. Three experiments were performed to determine the effect of treatment delay after harvest, fruit maturity, and intermittent exposure to UV-C on induction of disease resistance by UV-C. For these experiments, fruit of ‘Delphin’ were surface-sterilized and treated with UV-C. Wounding of the fruit and inoculation were performed as described above. Each treatment consisted of three replications of eight fruit each in a completely randomized design.

The first experiment was designed to evaluate the effect of treatment delay after harvest. Mature-green fruit were treated with UV-C (0.88 kJ·m⁻²) either 1 or 7 d after harvest, and stored in humidified containers at 13 °C. Wounding and inoculation were performed 1 d later. The second experiment was performed to study the effect of fruit maturity. Fruit were harvested at mature-green, turning or red stages, treated with a dose of 0.88 kJ·m⁻², and stored in humidified containers at 13 °C. Treated fruit were challenged with *B. cinerea* 24 h later.

The third experiment was designed to test whether UV-C treatment dispensed as two fractionated doses could be more effective than the standard single dose of 0.88 kJ·m⁻²; the second lot was treated twice 5 d apart with 0.44 kJ·m⁻² each time for a total dose of 0.88 kJ·m⁻²; the third lot was treated twice 5 d apart with a dose of 0.88 kJ·m⁻² each time for a total dose of 1.76 kJ·m⁻²; and the fourth lot received no treatment (control). Treated fruit were stored in humidified containers at 13 °C, and were inoculated with *B. cinerea* 1 d after the first exposure to UV-C.

Results

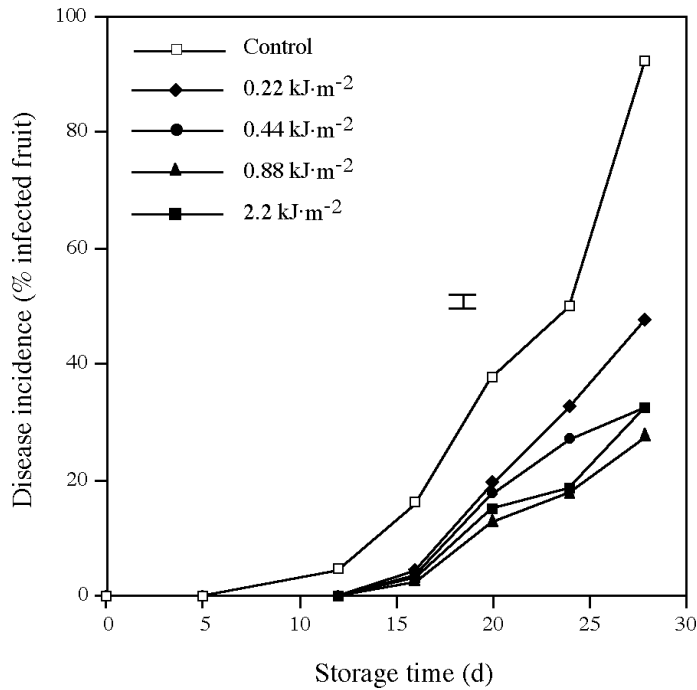


Fig. 1. Effect of UV-C treatment at different doses on control of natural infections by *B. cinerea* in 'Bell Boy' bell pepper fruit during storage at 13 °C. Vertical bar represents SE (± 0.97), $n = 3$.

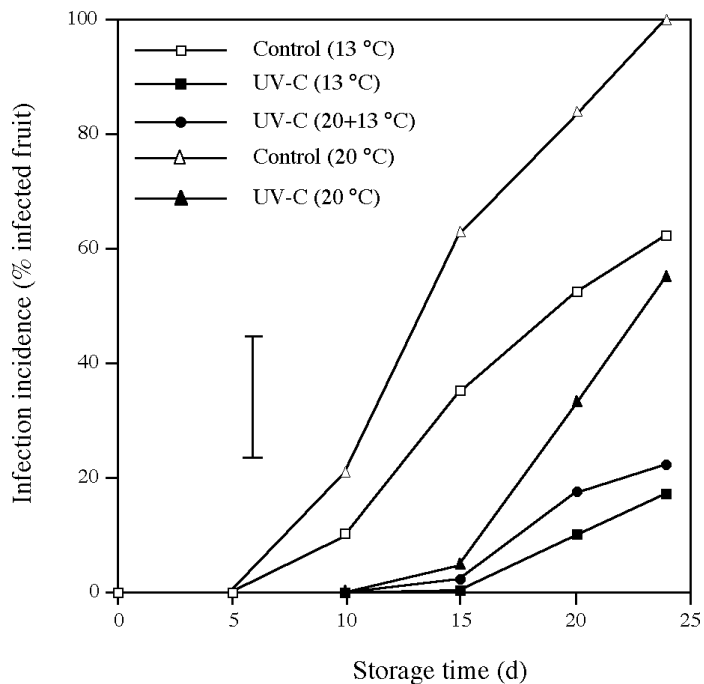


Fig. 2. Effect of UV-C at 0.88 kJ·m⁻² on control of natural infections by *B. cinerea* in 'Bell Boy' bell pepper fruit during storage at 13 or 20 °C. For the UV-C/20+13 °C treatment, fruit were exposed to UV-C at 20 °C but then placed in storage at 13 °C. Vertical bar represents SE (± 5.25), $n = 3$.

STATISTICAL ANALYSIS. A Bartlett's test was conducted to test for homogeneity of variances, and then analysis of variance (ANOVA) was performed on experimental data using a factorial split-plot design by General Linear Model Procedure (SAS Statistics Software; SAS Inst. Inc., Cary, N.C.).

EFFECT OF UV-C ON NATURAL INFECTIONS. UV-C treatment at all doses reduced significantly ($P \leq 0.01$) fungal decay of bell pepper fruit expressed as a percentage of infected fruit (Fig. 1). Inhibition of infection increased with the dose up to 0.88 kJ·m⁻². A dose of 2.2 kJ·m⁻² did not inhibit disease further and caused injury in the form of soft lesions 24 h after treatment. More than 90% of nontreated fruit showed signs of fungal decay after 4 weeks of storage at 13 °C and 95% RH. At the same time, with the 0.88 kJ·m⁻² treatment, <30% of the fruit were infected under similar storage conditions. Infection occurred more commonly on fruit which had short or damaged pedicels, although rot also appeared at the site of minor lesions on the fruit surface. Generally, once rot had started developing on the nontreated fruit, it advanced rapidly thereafter. The main cause of rot was identified as *B. cinerea*. UV-C at 0.88 kJ·m⁻² reduced significantly the percentage of fungal decay in fruit stored at both 13 and 20 °C (Fig. 2). Treating the fruit at 13 or 20 °C before storing them at 13 °C did not affect the efficacy of the UV-C treatment.

PRE- AND POSTINOCULATION EFFECT OF UV-C. Pepper fruit which were exposed to UV-C 24 h before inoculation with *B. cinerea* had lesions significantly smaller than the inoculated controls (Fig. 3). The effect of UV-C on lesion size was much weaker when the fruit had been inoculated 24 h before exposure to UV-C, indicating that UV-C treatment did not kill the pathogen in wounds. It thus appears that UV-C induces or enhances resistance in the host which becomes capable of slowing down the invasion of tissues by *B. cinerea*.

GERMICIDAL EFFECT OF UV-C ON *Botrytis cinerea*. Irradiation with UV-C killed conidia of *B. cinerea* when cultured in vitro or in vivo in fruit wounds (Table 1). On agar, UV-C killed virtually all conidia at 2.2 and 4.4 kJ·m⁻², while at 0.88 kJ·m⁻², a small proportion (14%) survived the treatment (Table 1). Similar results were obtained with conidia exposed in fruit wounds (0 h), although the survival rates was slightly higher than on agar (Table 1). In this case, 0.88 kJ·m⁻² was

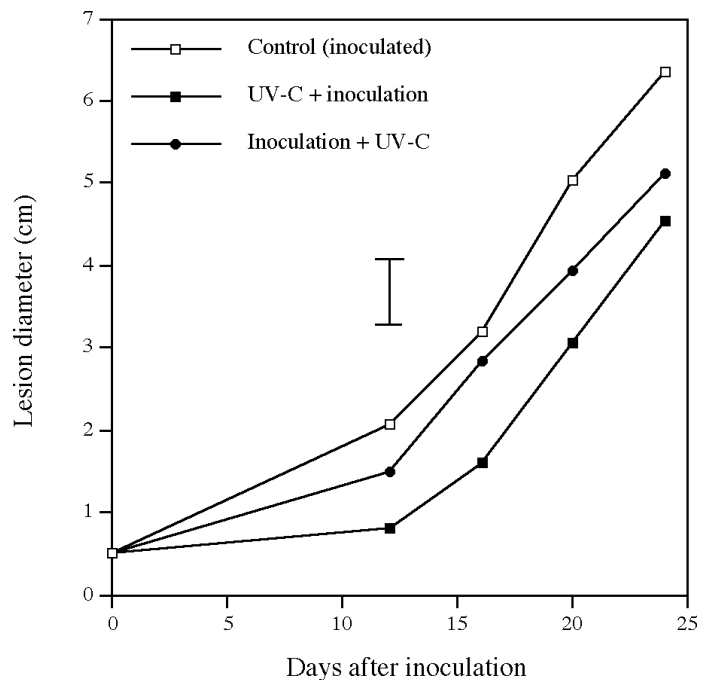


Fig. 3. Effect of exposure to UV-C at 0.88 kJ·m⁻² either 24 h before inoculation or 24 h after inoculation with *B. cinerea* on development of lesions on 'Delphin' bell pepper fruit stored at 13 °C. Vertical bar represents SE (± 0.39), $n = 3$.

Table 1. Effect of UV-C on survival of *Botrytis cinerea* when irradiated on agar or in fruit wounds, 0 h or 24 h after inoculation.

UV-C dose (kJ·m ⁻²)	In vitro spore germination (% ± SE)	In vivo fruit wounds	
		0 h Spore germination (% ± SE)	24 h Colony forming units ² (% ± SE)
0.0	97 ± 1	95 ± 2	55 ± 7
0.40	ND ³	60 ± 3	33 ± 4
0.88	14 ± 3	20 ± 3	8 ± 2
2.2	0	ND	ND
4.4	0	5 ± 0.5	0

²Expressed as percentage cfu since some germinated conidia did not survive the recovery process.

³Not determined.

effective in inactivating most of the conidia in the wounds with only 20% being able to germinate after treatment and a small proportion (5%), surviving the 4.4 kJ·m⁻² dose. When wounds were irradiated 24 h after inoculation, there was also a significant reduction in the number of colony-forming units (cfu) of the fungus, although recovery of the fungus from the wounds was generally lower, as germinated conidia and hyphae were apparently more affected by the grinding and plating than the ungerminated ones. A number of conidia which had germinated did not form colonies on agar and for that reason, these data are reported as the percentage cfu rather than percentage germination (Table 1).

FACTORS AFFECTING THE INDUCTION OF DISEASE RESISTANCE BY UV-C. Three factors affected the induction of disease resistance.

Delay after harvest. A 7 d delay after harvest did not affect the ability of UV-C of inducing resistance to *B. cinerea* (Fig. 4). However, pepper fruit which had been stored for 7 d were significantly more susceptible to the pathogen than freshly harvested ones and this difference can be seen for both control and UV-C treated fruit (Fig. 4).

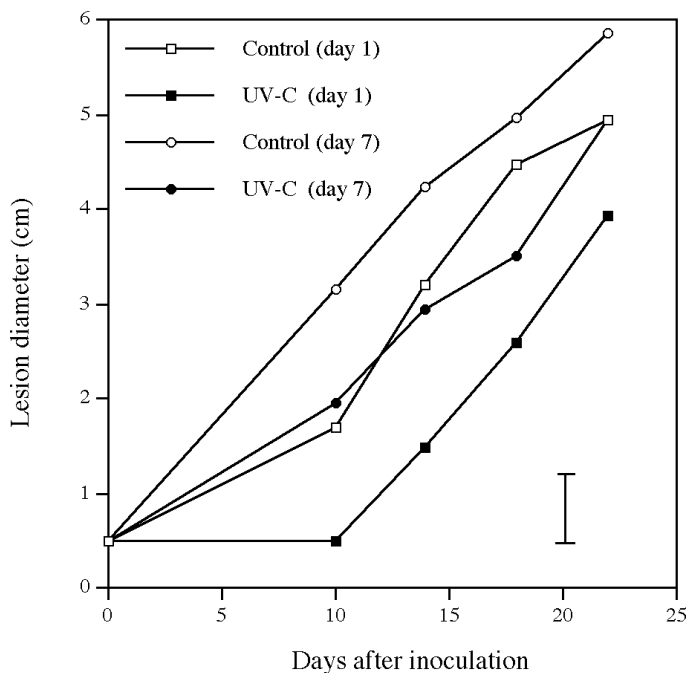


Fig. 4. Effect of delay after harvest (1 or 7 d) for UV-C treatment at 0.88 kJ·m⁻² on induction of resistance to *B. cinerea* in 'Delphin' bell pepper fruit stored at 13 °C. The fruit were inoculated with *B. cinerea* 24 h after UV-C treatment. Vertical bar represents SE (±0.37), n = 3.

Fruit maturity. UV treatment induced resistance to *B. cinerea* in bell pepper fruit regardless of their stage of maturity at the time of treatment (Fig. 5). However, the stage of maturity had a significant effect ($P \leq 0.01$) on fruit susceptibility to *B. cinerea*, more mature fruit (turning and ripe/red stages) being more susceptible than those that were green. UV-C treatment applied at the green stage was more effective in slowing down the progress of disease than at later stages (Fig. 5).

Intermittent irradiation. Treatment with a single dose of UV-C at 0.88 kJ·m⁻² was as effective as treatment with two successive doses of 0.44 kJ·m⁻² in inducing disease resistance (Fig. 6). Treatment with two successive doses of 0.88 kJ·m⁻² did not provide any advantage and even had a lower effect on disease resistance than the single dose.

Discussion

UV-C treatment proved effective in controlling naturally occurring infections (Figs. 1 and 2), as well as inducing disease

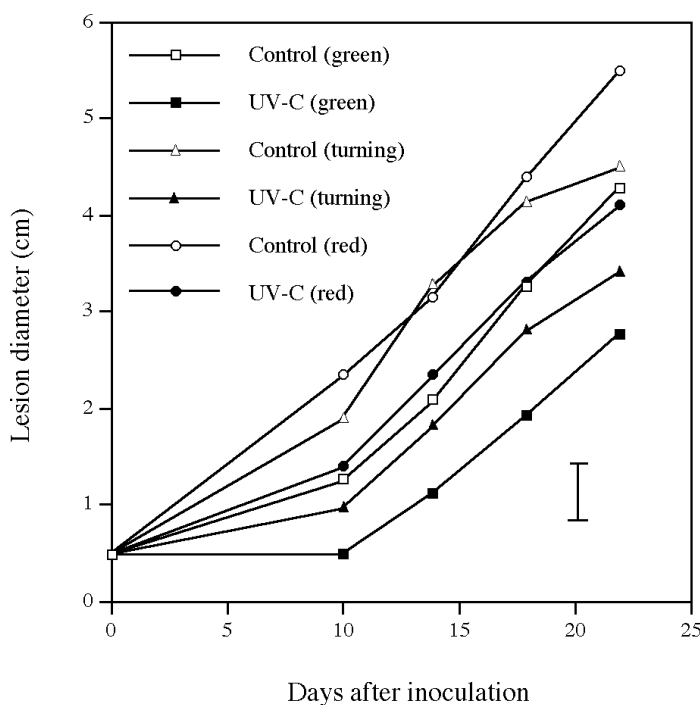


Fig. 5. Effect of maturity of 'Delphin' bell pepper fruit at harvest on induction of resistance to *B. cinerea* by UV-C at 13 °C. Fruit were exposed to 0.88 kJ·m⁻² and inoculated 24 h later with *B. cinerea*. Vertical bar represents SE (±0.30), n = 3.

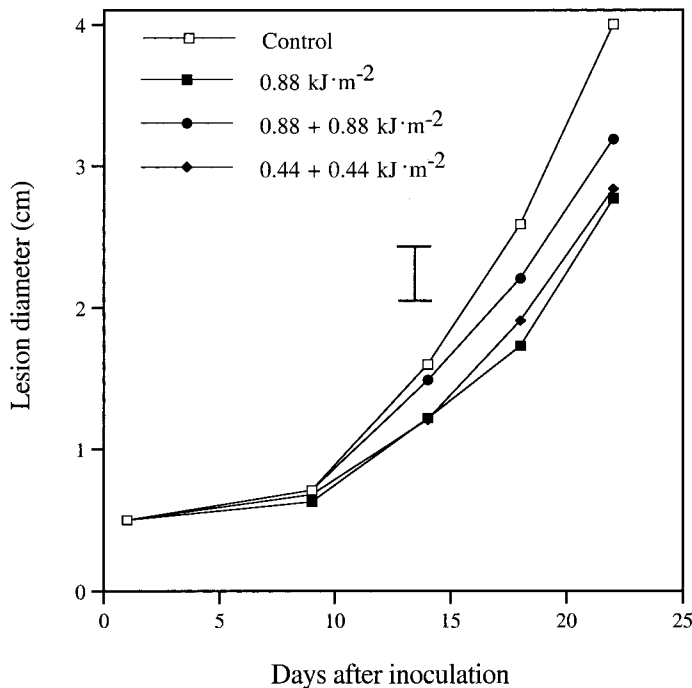


Fig. 6. Effect of intermittent UV-C treatments (0.44 or 0.88 kJ·m⁻²) compared to single standard exposure to 0.88 kJ·m⁻² on induction of resistance to *B. cinerea*. 'Delphin' bell pepper fruit were inoculated 24 h after the first exposure to UV-C. Fruit exposed twice were treated a second time 5 d after the first UV-C treatment. Vertical bar represents SE (±0.17), n = 3.

resistance in stored bell pepper fruit (Figs. 3, 4, and 5). UV-C dose—control of natural infection response was similar for both 'Bell Boy' and 'Delphin' cultivars (data not presented). UV-C dose—6-methoxymellein (phytoalexin) response was also found to be similar for five cultivars of carrot (Mercier et al., 1993c).

Until now, the main mechanism of action of UV-C studied in postharvest systems has been induced disease resistance (Ben-Yehoshua et al., 1992; Charles et al., 2001; Mercier et al., 1993a, 1993b, 2000; Rodov et al., 1992) and possible germicidal effects of UV-C on plant pathogens were not investigated. Although UV-C had a strong germicidal effect on *B. cinerea*, killing most spores exposed on agar or fruit wounds (Table 1), it was ineffective in preventing infection of fruit which had been inoculated 24 h before irradiation (Fig. 3). Similar results were obtained by Ben-Yehoshua et al. (1992), who found that UV-C did not control penicillium rot in citrus fruit inoculated 24 h or immediately before UV-C treatment. Most likely, enough spores or hyphae can survive UV treatment in wounds and initiate infections (Table 1). More spores survived exposure to 0.88 kJ·m⁻² in vivo than in vitro, presumably they evaded irradiation having been sheltered far into the wounds. Up to 20% of conidia exposed to 0.88 kJ·m⁻² on the fruit immediately after inoculation survived UV-C treatment, while still 8% survived when exposed 24 h after inoculation (Table 1). Even if UV-C could have killed a proportional number of propagules in wounds, enough inoculum could still have remained to initiate infections. Also, increasing the dose from 0.88 to 2.2 kJ·m⁻² did not provide any further control of natural infections (Fig. 1), although more spores were killed by the higher dose (Table 1). This suggests that induced resistance is the main mechanism of disease control. This phenomenon was clearly demonstrated when irradiated fruit were challenged with the pathogen 24 h later (Figs. 3, 4, and 5). However, partial decontamination of the fruit surface by UV-C could still be interesting

from a practical standpoint, as the irradiated tissues would be subject to less inoculum pressure in addition to being more disease resistant.

Induction of disease resistance by UV-C was a relatively rapid process at 13 °C, as fruit challenged 24 h after treatment already showed increased resistance to *B. cinerea* (Fig. 3). Such a rapid response makes the treatment particularly interesting as a post-harvest technology as the effect would show early during the storage period. A similar response was observed in bell pepper fruit treated with a fungal elicitor, with resistance appearing in 24 h (Adikaram et al., 1988). In this case, induction of resistance was associated with accumulation of the phytoalexin capsiannol, which was induced by the fungal elicitor. Although the induction of defense mechanisms by UV-C in bell pepper was not investigated, the induction of phytoalexins is a likely possibility, as their induction in citrus, carrot and tomato was closely associated with UV-induced resistance to storage pathogens (Ben-Yehoshua et al., 1992; Charles et al., 2001; Mercier et al., 1993a, 1993b, 2000; Rodov et al., 1992). In citrus and carrot, induction of phytoalexin was very rapid, with high amounts being produced within 48 h (Ben-Yehoshua et al., 1992; Mercier et al., 1993b; Rodov et al., 1992). However, at this point we cannot exclude induction of other defense mechanisms which could have contributed to UV-C-induced resistance in bell pepper and other systems. For example, a chitinase response to *B. cinerea* was primed by UV-C in carrot roots (Mercier et al., 2000).

Resistance responses activated by UV-C are apparently little affected by maturity or time in storage, as induced resistance was observed in fruit stored previously for 7 d or picked at the ripe stage (Figs. 4 and 5). The increase of resistance following UV-C treatment was apparently of the same magnitude when compared with controls of similar maturity, although more mature fruit were more susceptible to *B. cinerea* than freshly harvested green ones (Figs. 4 and 5). Mercier et al. (1993a) had reported previously that carrots which had been stored for 4 months, although more disease susceptible than freshly harvested carrots, could still respond to UV treatment by accumulating phytoalexin and exhibiting resistance to *B. cinerea*. The ability of inducing resistance in ripe bell pepper fruit is particularly interesting since a large number of bell peppers on the market are sold as mature red, yellow or other colored fruit, which usually command higher prices and are more perishable than green fruit. The fact that resistance can be induced after a delay of 7 d is also interesting for the applicability of the treatment for the preservation of bell peppers. This means that a small delay between harvest and UV-C treatment would not affect disease control significantly.

Results herein regarding intermittent exposure to UV-C suggest that UV-C doses are additive (Fig. 6). There is no additional benefit of fragmenting the optimal dose of 0.88 kJ·m⁻² as two successive exposures to 0.44 kJ·m⁻² had the same effect as one exposure to 0.88 kJ·m⁻², whereas two exposures to 0.88 kJ·m⁻² caused a decrease in the resistance response (Fig. 6). Apparently, once the resistance response has been induced, this response cannot be enhanced by added exposure to irradiation.

The optimum dose of UV-C for controlling infections in various crops seems to vary greatly. In the case of bell peppers, the dose appears to be quite small and is the same as the optimum dose reported for inducing disease resistance in stored carrots (Mercier et al., 1993a). However, doses as high as 3.7 kJ·m⁻² and 5.0 kJ·m⁻² were used for inducing disease resistance in tomatoes and lemon [*Citrus limon* (L.) Burm.], respectively (Ben-Yehoshua et al., 1992; Charles et al., 2001), and doses ranging between 3.6

to 7.5 kJ·m⁻² were used to control natural infection in apples [*Malus sylvestris* (L.) Mill. var *domestica* (Borkh.) Mansf.], peaches [*Prunus persica* (L.) Batsch (Peach Group)], onions [*Allium cepa* L.], and sweet potatoes [*Ipomoea batatas* (L.) Poir.] (Lu et al., 1987, 1991; Stevens et al., 1990). Damage by UV-C such as bronzing in banana [*Musa acuminata* Colla] and citrus have been reported, with unripe fruit being more susceptible (Ben-Yehoshua et al., 1992; Wade et al., 1993). Since in the present study, a high dose of UV-C (2.2 kJ·m⁻²) caused some injury in bell peppers, using the lowest effective dose would be preferable for preventing such problems. In addition, use of a low dose would allow faster treatment of the produce and make UV-C treatment easier to implement.

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