# A Comparison of Low Intensity UV-C and High Intensity Pulsed Polychromatic Sources as Elicitors of Hormesis in Tomato Fruit

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## Abstract

Post-harvest hormetic treatment of mature green tomato fruit (*Solanum lycopersicum* cv. Mecano) with high intensity pulsed polychromatic light (HIPPL) significantly delayed ripening to levels comparable to those achieved using a conventional low intensity UV-C (LIUV) source. A 16 pulse HIPPL treatment reduced the ΔTCI (tomato colour index) by 50.1 % whilst treatment with a LIUV source led to a reduction of 43.1 %. Moreover, the 16 pulse treatment also induced disease resistance in the fruit to *Botrytis cinerea* with a 41.7 % reduction in disease progression compared to a 38.1 % reduction for the LIUV source. A single 16 pulse HIPPL treatment was found to significantly reduce disease progression on both mature green and ripe fruit with a 28.5 % reduction on ripe fruit in comparison to 13.4 % for the LIUV treatment. It is shown here that delayed ripening and disease resistance are local responses in side treated tomato fruit for both LIUV and HIPPL treatments. Finally, utilising a 16 pulse HIPPL treatment would reduce treatment times from 370 s for LIUV sources to 10 s per fruit - a 97.3 % reduction.

**Key words:** UV-C Hormesis; *Solanum lycopersicum*; Intense Pulsed light; Induced Resistance; Delayed Ripening; Polychromatic Light

## 1 Introduction

The portion of the electromagnetic spectrum between 10 and 400 nm is referred to as the ultraviolet light region (UV). Within this region, wavelengths between 100 and 315 nm are known as 'germicidal UV'. Germicidal UV is used extensively to directly inactivate a range of micro-organisms in a number of different media including both solids and liquids (Shama, 2014). Some three decades ago research began to be undertaken in inducing UV-C hormesis (Lu *et al.,* 1987). Since then UV-C treatment has been performed on a wide range of fresh produce, as reviewed by Shama and Alderson (2005), Ribeiro *et al.* (2012) and Turtoi (2013). Hormesis is a phenomenon in which low doses of a potentially damaging agent bring about a beneficial response in the organism receiving the treatment. The beneficial effects of UV-C hormesis have been demonstrated for numerous types of fresh produce including both climacteric and non-climacteric fruit, tubers, salads and brassicas (Ranganna *et al.,* 1997, D'Hallewin *et al.,* 1999, Costa *et al.,* 2006, Pongprasert *et al.,* 2011, Kasim & Kasim, 2012). Such effects include, but are not limited to, pathogen resistance, delayed ripening and improved nutritional content (Shama & Alderson, 2005, Ribeiro *et al.,* 2012, Turtoi, 2013).

It has been estimated that in the UK, 45 % of all purchased salad and 26 % of fruit is disposed of post retail (WRAP, 2012). Losses in storage, however, can be attributed to spoilage pathogens, senescence and transpiration (Maharaj *et al.,* 1999). Crop-dependant pre and postharvest losses of 8 - 15 % occur annually due to spoilage pathogens (Oerke, 2006). Losses of tomato fruit (S*olanum lycopersicum*), the tenth most economically important non-meat food commodity, however, are exacerbated as fruits are particularly prone to chilling injury (Morris, 1982, FAO, 2015).

UV-C hormesis has been shown to induce disease resistance against a wide range of pathogens, which is achieved through both phytoalexin production and delayed ripening (Ben-Yehoshua *et al*.,

1992, D'Hallewin 1999, D'Hallewin *et al.,* 2000, Mercier *et al.,* 2000, Romanazzi *et al.,* 2006, Charles *et al.,* 2008a). Many phytoalexins are phenolic compounds that act both as light quenchers, absorbing damaging wavelengths of light, and antioxidants that prevent reactive oxygen species (ROS) mediated cellular damage (Pietta, 2000, Sourivong *et al.,* 2007, Lev-Yadun & Gould, 2009). It would appear, therefore, that it is their dual function which allows the build-up of resistance against plant pathogens in response to UV-C stress. Furthermore, specific pathogenesis related (PR) proteins have also been shown to increase in concentration following hormetic UV-C treatment; these include chitinases and β-1,3-glucanases which interact directly with pathogens to reduce their viability (Charles *et al.,* 2009).

The vast majority of previous studies on UV-C hormesis have been conducted with low pressure mercury sources that emit UV light with a peak emission at 254 nm at relatively low intensities, henceforth referred to as low intensity UV-C (LIUV). The long treatment times required by LIUV sources explains in part why there has been reluctance by the horticulture sector to adopt this form of treatment. To take a specific example, there is consensus on the average UV-C dose (3.7 $kJ/m^2$) necessary to induce hormetic effects in tomato fruit, (Liu, *et al.,* 1993 & Maharaj *et al.,* 1999). Using low pressure mercury sources at an intensity of 20 $W\ m^{-2}$ would require an exposure time of approximately six min per fruit. Furthermore, the requirement for complete surface irradiation to induce the beneficial effects on certain types of produce both complicates the treatment procedure and extends the treatment time (Mercier *et al.,* 2000). The recent advent of high intensity pulsed polychromatic sources (HIPPS) with considerable emission in the UV-C region could result in a substantial reduction in treatment times from minutes to seconds.

Treatment of fresh produce with HIPPL has been shown to increase the concentration of anthocyanins and total phenolics along with improving colour in nethouse grown fig, *Fiscus carica* (Rodov *et al.,* 2012). Both LIUV and HIPPL treatments have been shown to significantly increase the total lycopene, carotenoid and phenolic content as well as antioxidant activities of tomato fruit (Liu *et al.,* 2009, Liu *et al.,* 2012 & Pataro *et al.,* 2015). HIPPL has also been shown to increase

77 anthocyanin and Vitamin $D_2$ levels in mushrooms, *Agaricus bisporus* (Oms-Oliu *et al.,* 2010,
78 Koyyalamudi *et al.,* 2011).
79 The aim of this study was to investigate whether HIPPL sources were able to delay colour change
80 during ripening and induce resistance against *B. cinerea* on mature green tomato. Treatments were
81 also conducted with a LIUV source as a basis for comparison. Experiments were also undertaken to
82 establish whether it was necessary to irradiate the entire fruit surface for successful elicitation of
83 delayed colour change and disease resistance. Additionally, treatments using both types of source,
84 HIPPL and LIUV, were conducted to assess their ability to induce disease resistance on red ripe fruit,
85 as an increasing number of tomato growers are harvesting at this stage due high consumer demand.
86
87 **2. Materials and Methods**
88
89 **2.1 Plant Material**
90 Mature green and red ripe tomato fruit, cv. Mecano, were grown in the glasshouse at APS Salads
91 (UK) and delivered at ambient temperature to the University of Nottingham within 24 h of
92 harvesting. Fruit were then sorted to remove fruit showing deviation from the desired
93 developmental stage or uniformity of size. Fruit showing any surface damage were also discarded.
94
95 **2.2 UV Treatment**
96 Upon arrival tomatoes were randomly assigned to treatment groups and treated at room
97 temperature on the same day. LIUV treatments were carried out using a U-shaped amalgam UV
98 source (UVI 12OU2G11 CP15/469) obtained from Dr Hőnle AG, Gräfelfing, Germany, with peak
99 emission at 254 nm and housed within an anodised aluminium parabolic reflector. Doses of 3.7
100 $kJ/m^2$ were delivered at an intensity of 20 W $m^{-2}$ following the procedures of Charles *et al.* (2008a).
101 Intensity was measured with a portable radiometer (Model UVX, UVP Instruments, Cambridge) fitted
102 with a 254 nm sensor.

HIPPL treatments were carried out with a XENON LH-840 16" ozone free B lamp powered and controlled by RT-847 cabinet and RC-802 controller, supplied by Lambda Photometrics (Harpenden, Herts). The source produced 505 J of energy per pulse with a pulse width of 360 µs at 3.2 pulses per second. Spectral emissions of the source were between 240 nm and 1050 nm. Fruit were placed at a distance of 10 cm from the window of the lamp housing. Though extrapolation of the manufacturer's data an estimated 4.6 kJ/m$^2$/pulse was delivered at fruit level.

Fruit received exposure on two sides through 180 ° axial rotation. For experiments aimed at determining whether full tissue exposure was necessary for inducing disease resistance, fruit were treated from only one side. Following treatment fruit were immediately stored in the dark until sterilisation. For sterilisation tomatoes were immersed in 2 % Sodium hypochlorite (Sigma-Aldrich) for approximately 5 – 10 seconds; to prevent growth of naturally occurring microorganisms during the incubation period. Fruit were then rinsed three times in sterile distilled water (SDW), dried and immediately incubated in the dark at 13 °C to prevent photoreversal. Fruit were stored for 10 d in high humidity boxes with relative humidity > 98 %.

**2.3 Colour measurement**

Tomato colour was monitored to determine ripening progression (Lopez Camelo & Gomez, 2004, Corcuff *et al.,* 2012). Measurements were conducted using a calibrated CR-200 Chroma meter (Konica Minolta) in L*a*b* mode. Readings were taken at a single point directly facing the source and at a 90 ° axial rotation from that point. A second colour measurement was taken using the same reference points at 10 d post treatment (DPT). Tomato colour index (TCI, Eq.1) was then calculated (Hobson, 1987). The two measurements were then used to calculate the change in TCI over 10 d.

$$\mathrm{TCI} = \frac{2000(a)}{\sqrt{L(a^2 + b^2)}}$$

**Equation 1.** Tomato colour index (TCI) formula where L= lightness, a= red-green and b = blue-yellow values (Hobson, 1987).

128 **2.4 Pathogen Maintenance and Inoculum Preparation**

129 A *Botrytis cinerea* culture, originally isolated from a plant of the genus *Rosa*, was supplied from
130 The University of Nottingham's collection. Cultures were grown at room temperature on potato
131 dextrose agar (Sigma-Aldrich) supplemented with Penicillin G sodium salt (Sigma-Aldrich) at 33 mg/L
132 and Streptomycin sulphate salt (Sigma-Aldrich) at 133 mg/L. A calibrated spore solution was made
133 from 10-14 d old cultures. Briefly, Petri dishes were flooded with 15 mL of SDW supplemented with
134 0.03 % Tween 20. Spores were released by gentle agitation and then filtered through a double layer
135 of muslin cloth and vortexed vigorously to release conidia from conidiophores. The spore solution
136 was then centrifuged at 184 g in a Centaur 2 (MSE) for 10 min and the supernatant discarded. The
137 pellet was re-suspended in SDW, vortexed and centrifuged again at 184 g for a further 10 min, the
138 supernatant was discarded. The pellet was re-suspended in SDW and a haemocytometer was used
139 to obtain the desired spore concentration.

140

141 **2.5 Inoculation and Lesion Measurement**

142 At 10 DPT fruit were inoculated with *B. cinerea*. This interval was selected on the basis of the
143 work of Charles *et al.* (2008) who showed near optimal induction of resistance occurred at 10 DPT.
144 Fruit were wounded with a sterile hypodermic needle to a depth of 3 mm. Ripe fruits were then
145 inoculated with 5 µL of spores at 1 x $10^5$ per mL. Green fruits, however, were inoculated with 5 µL of
146 1 x $10^6$ spores per mL due to decreased levels of susceptibility shown in preliminary work. For direct
147 tissue exposure experiments fruit were either inoculated on a treated or untreated side with one
148 inoculation point per fruit.

149 Total lesion diameter, including all sunken lesions, splitting and tissue maceration, were then
150 measured with digital Vernier callipers at 3 and 4 d post inoculation. Measurements were used to
151 calculate the area under the disease progression curve (AUDPC, Equation 2) (Jeger and Viljanen-
152 Rollinson, 2001).

153 $$\mathrm{AUDPC} = \sum_{i=1}^{n-1} \frac{y_i + y_{i+1}}{2} (t_{i+1} - t_i)$$

154 **Equation 2.** Area Underneath the Disease Progression Curve formula where n= total number of
155 observations, i= observation, y= disease score and t= time (Jeger and Viljanen-Rollinson, 2001).

156

157 **2.6 Experimental Design and Statistical Analysis**

158 All data presented here was collected from two independent replicate experiments. For the
159 experiments concerning delayed ripening and disease resistance 15 fruit were used in each
160 treatment group, per experiment (n = 30). Ten fruit per group, per experiment (n=20) were used for
161 experiments on the necessity for direct tissue exposure.

162 Analysis was performed using statistical software package SPSS 22 (IBM). One-way ANOVA with
163 Tukey's post-hoc testing was performed. Where the homogeneity of variances assumption could not
164 be met Welch's robust ANOVA was performed followed by the Games-Howell post-hoc test.
165 Statistical significance is here defined as $p \leq 0.05$.

166

167 **3 Results and Discussion**

168

169 **3.1 Delayed Ripening**

170 The induction of delayed ripening in mature green tomatoes is an established beneficial effect
171 following hormetic LIUV treatment (Stevens *et al.,* 1998a, Corcuff *et al.,* 2012). Furthermore, colour
172 is the key external indicator for ripening progression on tomato fruit (Lopez Camelo and Gomez,
173 2004). Changes in TCI were, therefore, used to monitor the progression in ripening; with lower TCI
174 values indicating a greener tomato.

175 The 3.7 kJ/m$^2$ LIUV, 16 and 24 pulse treatments showed significantly lower ripening progression,
176 Δ TCI, in comparison to the control (Figure 1). Fruit treated with 8 pulses did not ripen at a rate
177 significantly different from the control. Representative samples of tomato fruit are shown in Figure

2. All of the data here supports the successful induction of delayed ripening with either HIPPL or LIUV. This data contradicts recently published work by Pataro *et al*. (2015) who observed no effect for either LIUV or HIPPL treatments on the ripening of tomato fruit of cv. San Marzano. The HIPPL source used by Pataro *et al.* (2015) gave comparable pulse length (360 µs) and spectral emission (200 to 1100 nm) to that produced by the source used here. The spectral irradiance, i.e. intensity of specific wavelengths, however, may have differed to the source used in this study. Furthermore, different experimental protocols used by Pataro *et al.,* (2015) may have led to the failure to detect a significant difference in colour change for LIUV and HIPPL treated fruits. Specifically, the use of a 14 / 10 h day and night light cycle during fruit storage may have affected the induction of delayed ripening.

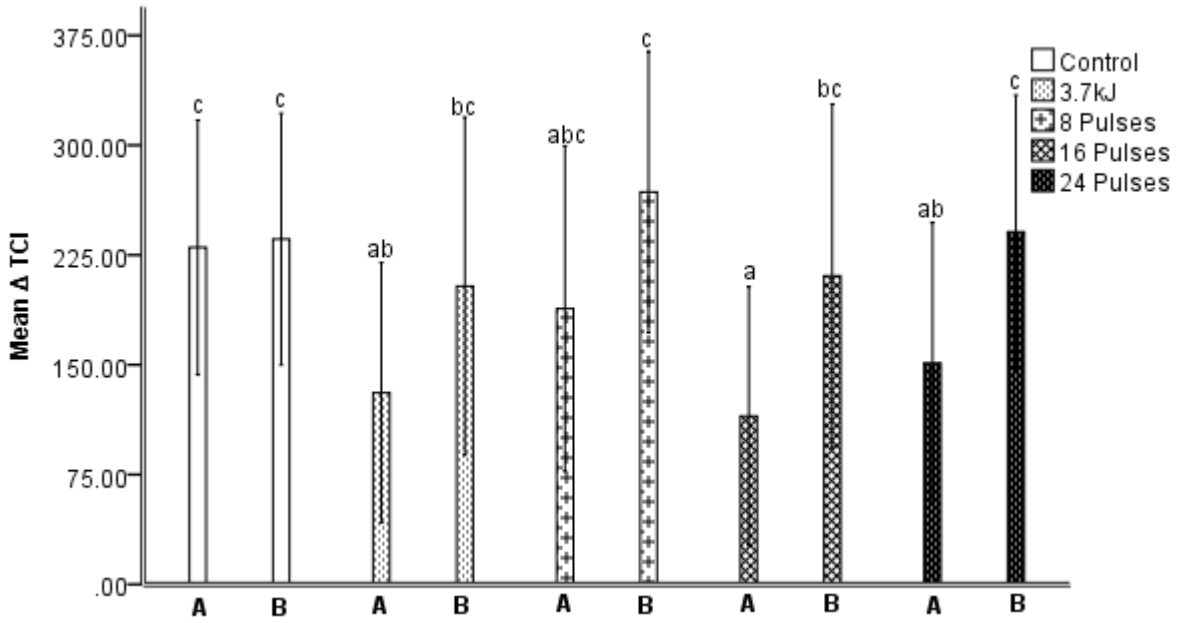


**Figure 1.** The Δ TCI (tomato colour index) from day 0 - 10 of mature green fruit from cv. Mecano. Fruit were treated with a hormetic LIUV treatment of 3.7 kJ/m$^2$ from a low intensity source with peak emissions at 254 nm and three high intensity pulsed polychromatic light (HIPPL) treatments of 8, 16 and 24 pulses. TCI measurements were taken from tissue directly facing the light source (**A**) and at 90 $^o$ from the source (**B**). Error bars show ± 1 standard deviation; n = 30. Labelling indicates statistical significance. Means sharing the same label are not significantly different from each other

at $p < 0.05$.

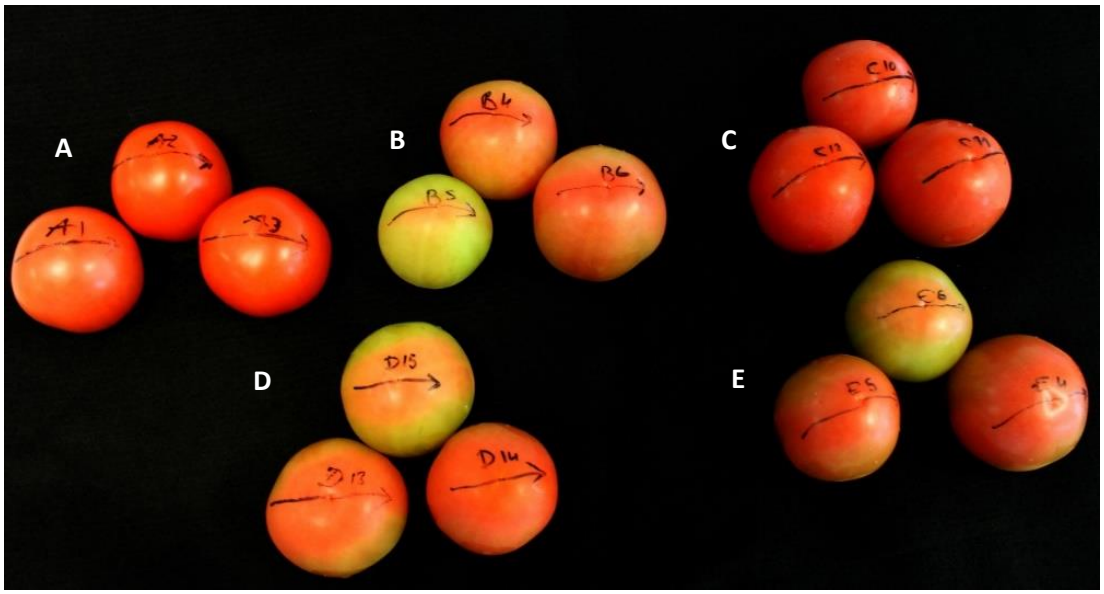


**Figure 2.** Representative samples of tomato fruit of the cultivar Mecano at 10 d post treatment. Groups show the **c**ontrol fruit (**A**), the 3.7 kJ/m$^2$ LIUV treatment with peak emissions at 254 nm (**B**) and fruit treated with the high intensity pulsed polychromatic light (HIPPL) light source at 8 (**C**), 16 (**D**) and 24 (**E**) pulses.

Allowing the fruit to become exposed to visible wavelengths of light following treatment may have led to photoreversal - a phenomenon in which the effects of UV-C induced responses are negated by subsequent exposure to visible light (Kelner, 1949). It had previously been shown by Stevens *et al.* (1998b) that peaches, *Prunus persica*, exposed to 48 h of visible light following UV-C treatment no longer exhibited a reduction in brown rot lesions caused by *Monilinia fructicola.* The influence of photoreversal on the ripening progression of tomato fruit, has not been investigated.

**3.2 Direct Tissue Exposure and Delayed Ripening**

During preliminary work it was noted that the effects of delayed ripening were more pronounced on tissue directly facing the HIPPL and UV-C sources. To establish whether LIUV and

HIPPL delayed ripening is a local response, Δ TCI was also calculated for tissue at 90 $^{o}$ from that directly exposed to the source. For all groups the tissue at 90 $^{o}$ from the source showed no significant difference in ripening progression. When compared with directly exposed tissue, however, tissue at 90 $^{o}$ from the 16 and 24 pulse treatments showed a significantly greater progression in ripening to that of the directly exposed tissue (Figure 1). Tissue at 90 $^{o}$ for the LIUV treatment ripened faster than directly exposed tissue but was not statistically significant from directly exposed tissue or the control. The data presented here indicate that direct exposure to both LIUV and HIPPL is required for the induction of delayed ripening. This is in line with observations by Mercier *et al.* (2000) who showed the local accumulation of phytoalexin 6-methoxymellein in carrot, *Daucus carota,* following LIUV treatment.

It has, however, been shown by Stevens *et al.,* (2005) that alterations in treatment orientation may facilitate systemic signal translocation utilising the fruit's vasculature. Stevens *et al.,* (2005) showed that treatment at the calyx resulted in systemic disease resistance on apples (*Malus domestica*), peaches (*Prunus persica*) and tangerines (*Citrus reticulate*). Alternative treatment orientations were, therefore, performed to establish whether directing treatments at either the blossom end or calyx would allow the translocation of a systemic signal to delay ripening. All treatment orientations, however, produced uneven ripening progression, Figure 3.

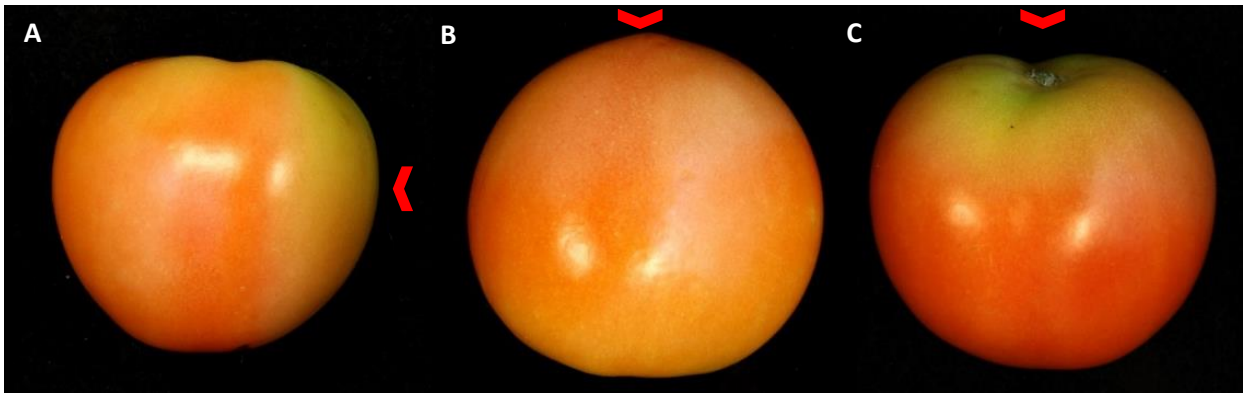


**Figure 3.** Representative samples of tomato fruit exposed to polychromatic light from different orientations. Fruit, cv. Mecano, were treated with 16 pulses of high intensity pulsed polychromatic light (HIPPL) and photographed at 10 d post treatment. Red arrows indicate the positioning of the HIPPL source. **A**) Treatment from the side. **B**) Treatment from the blossom end. **C**) Treatment from the calyx.

### 3.3 Disease Resistance on Mature Green Fruit

LIUV has previously been shown to induce disease resistance against *B. cinerea* on tomato fruit (Charles *et al.,* 2008a). The possibility of inducing resistance with HIPPL was, therefore, investigated. HIPPL and LIUV treated fruit showed reductions in mean AUDPCs indicating reduced disease progression (Table 1). Welch's ANOVA showed that disease progression for all treated groups was significantly lower than the control. No significant differences were observed between HIPPL treatments and the LIUV treatment. However, a significant difference between the AUDPCs of the 8 and 16 pulse treatments was observed showing increased disease resistance for the 16 pulse treatment.

**Table 1.** Area underneath the disease progression curve (AUDPC) from mature green fruit cv. Mecano treated with a conventional low intensity UV-C (LIUV) source, with peak emissions at 254 nm, and an high intensity pulsed polychromatic light (HIPPL) source. Inoculations were performed with *B. cinerea* at 10 d post treatment; n = 30.

| Treatment | Treatment time (s) | Mean AUDPC | Standard deviation | Mean AUDPC Reduction (%) |
|---|---|---|---|---|
| Control | 0 | 70.74 | 14.00 | - |
| 3.7 kJ/m$^2$ | 370 | 43.76$^{ab}$ | 25.13 | 38.14 |
| 8 Pulses | 5 | 56.05$^{b}$ | 16.82 | 20.76 |
| 16 Pulses | 10 | 41.21$^{a}$ | 17.09 | 41.74 |
| 24 Pulses | 15 | 45.15$^{ab}$ | 22.91 | 36.17 |

Superscript labelling indicates statistical significance. Means sharing the same superscript are not significantly different from each other at $p < 0.05$.

These results show that HIPPL can induce resistance to *B. cinerea* on mature green tomatoes to similar levels to that of LIUV treatment. This is in contrast to the results obtained by Marquenie *et al.* (2003) who reported no effect of pulsed light on the disease progression of *B. cinerea* on strawberries, *Fragaria ananassa*. This could be due to the employment of a different plant species or to differences in the spectral emission of the HIPPL sources. The HIPPL source used by Marquenie *et al.* (2003) produced 30 µs pulses at 15 pulses per second (15 Hz). The source in this study, however, produces 360 µs pulses at 3.2 pulses per second. Furthermore, the authors reported that the percentage of light falling within the UV region was 50 % of a 7 J pulse in contrast to the output obtained here (1 % of a 505 J pulse).

The 16 pulse treatment, here, provides comparable levels of disease resistance to the 3.7 kJ/m$^2$ LIUV treatment with 41.5 % and 38.1 % reductions in AUDPC, respectively. The total duration of the treatment times for both the HIPPL and LIUV sources are 10 s and 370 s, respectively. This equates to a 97.3 % reduction in exposure time or a 37-fold increase in the number of tomatoes that could

be treated with HIPPL compared to a LIUV treatment. Such a reduction could help overcome one of the factors - lengthy treatment times - that has militated against the adoption of LIU hormesis in commercial horticulture.

**3.4 Direct Tissue Exposure and Disease Resistance**

Following the observation that delayed ripening was a local response for both HIPPL and LIUV, section 3.2, tests were conducted to establish whether disease resistance was also a local response. To date, no data concerning this has been published for either LIUV or HIPPL induced resistance on tomato. Further investigation was performed to ascertain whether full tissue exposure is required to induce resistance in tomato fruit. Inoculations were performed on directly exposed and un-exposed tissue; the latter is henceforth referred to as 'systemic'.

Systemic tissue inoculations showed no reduction in AUDPC and similar levels of disease progression to that of the control (Figure 4). The directly exposed tissue, however, showed significant reductions following both HIPPL and LIU treatment as previously shown (section 3.3). It can therefore be stated that HIPPL and LIUV sources require direct tissue exposure to successfully induce resistance to *B. cinerea*. This is in agreement with previous findings (Stevens *et al.,* 1998a, Charles *et al.,* 2008, Liu *et al.,* 2011) who routinely rotated the fruit during LIUV treatment to ensure that the entire surface area of the fruit was irradiated, although they but did not specifically set out to show that failure to do so would not result in systemic resistance. The results presented here are therefore the first to confirm that side focused treatments require full surface exposure for LIUV induced disease resistance on tomato fruit. Similarly, it was reported by Mercier *et al.* (2000) that LIUV treatment leads to a local response in carrot. HIPPL-induced disease resistance is also a local response, and therefore cannot overcome the requirement for fruit rotation during treatment or an alternative arrangement of light sources.

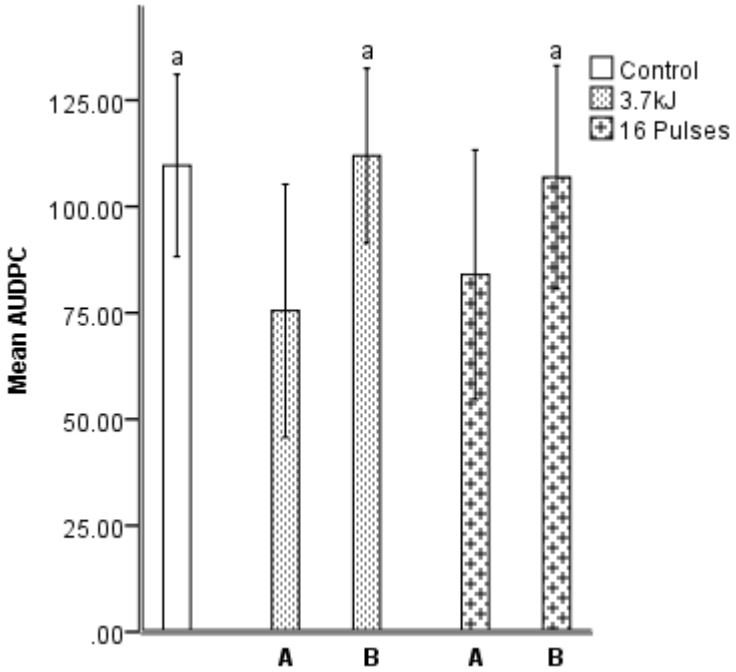


**Figure 4.** Area underneath the disease progression curve (AUDPC) of tomatoes, cv. Mecano, treated on a single side and inoculated with *B. cinerea* at 10 d post treatment (DPT). Fruit were treated with an established low intensity UV-C (LIUV) treatment of 3.7 kJ/m$^2$, peak emissions at 254 nm, and a high intensity pulsed polychromatic light (HIPPL) treatment of 16 pulses. Exposed tissue (**A**) or systemic tissue (**B**). Error bars show ± 1 standard deviation; n = 20. Labelling indicates statistical significance. Means sharing the same label are not significantly different from each other at $p < 0.05$.

An alternative means of inducing hormetic responses in produce may be to conduct treatments pre-harvest. Obande *et al*. (2011) showed the systemic induction of delayed ripening while treating tomato fruit on the plant. The response to LIUV has been shown to be both tissue and developmental stage-specific in grapevine, *Vitis vinifera*, where biomarkers of LIUV treatment were analysed by RT Q-PCR (Petit *et al*., 2009). It could, therefore, be hypothesised that the exposure of alternative tissue such as the truss stems may allow the propagation of a systemic response. Further investigation is required to ascertain whether disease resistance is also spread systemically after pre-harvest LIUV treatment of fruit.

### 3.5 Disease Resistance of Ripe Fruit

The majority of studies on LIUV induced disease resistance have been carried out postharvest on mature green tomatoes. Treatment at this stage is not entirely relevant for the UK tomato industry where tomatoes are picked when at the red ripe stage to meet consumer preferences. Induced resistance against *B. cinerea* on red ripe tomatoes was, therefore, investigated.

LIUV treated fruit did not show significantly reduced disease progression (Table 2). Moreover, an 8 pulse treatment did result in a slight reduction of disease progression but was not statistically significant. Both 16 and 24 pulse HIPPL treatments, however, did significantly reduce the AUDPC in comparison to the control. The failure of the LIUV treatment to induce significant levels of disease resistance, here, is in accordance with the results shown by Obande *et al.* (2011) who found that pre-harvest treatments of 3 kJ/m$^2$ did not effectively reduce the disease progression of *Penicillium digitatum* on ripe tomatoes, cv. Mecano. An 8 kJ/m$^2$ dose, however, effectively reduced disease. Variation in the induction of hormetic responses for the HIPPL and conventional UV-C sources is not unexpected due to the differences in spectral emission, the intensity of dose delivery and fractionation of the dose with HIPPL sources.

**Table 2.** Area Underneath the Disease Progression Curve (AUDPC) for ripe fruit cv. Mecano treated with a conventional low intensity UV-C (LIUV) source with peak emissions at 254 nm and a high intensity pulsed polychromatic light (HIPPL) source, followed by inoculation with *B. cinerea* at 10 d post treatment; n = 30.

| Treatment | Treatment time (s) | Mean AUDPC | Standard deviation | Mean Disease Reduction (%) |
|---|---|---|---|---|
| Control | 0 | $57.98^{b}$ | 20.00 | - |
| 3.7 kJ/m$^2$ | 370 | $50.20^{ab}$ | 12.66 | 13.43 |
| 8 Pulses | 5 | $48.12^{ab}$ | 18.98 | 17.00 |
| 16 Pulses | 10 | $41.43^{a}$ | 20.04 | 28.54 |
| 24 Pulses | 15 | $41.65^{a}$ | 19.84 | 28.15 |

Superscript labelling indicates statistical significance. Means sharing the same superscript are not significantly different from each other at $p < 0.05$.

## 4. Conclusions

The data presented here shows that HIPPL can induce both delayed ripening and disease resistance against *B. cinerea* to a level comparable to that of LIUV sources, but with a significant reduction in treatment time of 97.3 %. Furthermore, the work presented here demonstrates categorically that LIUV treatments, focused on the side of fruit, induce only local responses on tomato fruit. This was shown also to be the case for HIPPL sources. In addition, a 16 pulse HIPPL treatment significantly reduced disease on both red ripe and mature green tomatoes, a feature not exhibited by the established LIUV treatment.

No studies have yet been undertaken to establish the optimum wavelengths for inducing hormetic effects in fresh produce. The spectral emission of the two types of sources used here are quite different. The HIPPL source, although rich in UV-C, has a much broader spectral output; emitting wavelengths between 180 – 1050 nm, and it should not be assumed that HIPPL will elicit the same pathways or responses as hormesis induced by conventional LIUV sources that emit over a much narrower spectral range. Future work could ascertain the importance of germicidal UV and other wavelengths in the HIPPL source. Furthermore, optimum wavelengths for inducing hormetic effects could turn out to be species-related and establishing what these are would make hormetic treatment more commercially attractive.

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