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## **A comparison of low intensity UV-C and high intensity pulsed polychromatic sources as elicitors of hormesis in tomato fruit**

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

3  
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10  
11 **Abstract**

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

24

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

27

## 28 **1 Introduction**

29

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

43 It has been estimated that in the UK, 45 % of all purchased salad and 26 % of fruit is disposed of  
44 post retail (WRAP, 2012). Losses in storage, however, can be attributed to spoilage pathogens,  
45 senescence and transpiration (Maharaj *et al.*, 1999). Crop-dependant pre and postharvest losses of 8  
46 - 15 % occur annually due to spoilage pathogens (Oerke, 2006). Losses of tomato fruit (*Solanum*  
47 *lycopersicum*), the tenth most economically important non-meat food commodity, however, are  
48 exacerbated as fruits are particularly prone to chilling injury (Morris, 1982, FAO, 2015).

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

51 1992, D'Hallewin 1999, D'Hallewin *et al.*, 2000, Mercier *et al.*, 2000, Romanazzi *et al.*, 2006, Charles  
52 *et al.*, 2008a). Many phytoalexins are phenolic compounds that act both as light quenchers,  
53 absorbing damaging wavelengths of light, and antioxidants that prevent reactive oxygen species  
54 (ROS) mediated cellular damage (Pietta, 2000, Sourivong *et al.*, 2007, Lev-Yadun & Gould, 2009). It  
55 would appear, therefore, that it is their dual function which allows the build-up of resistance against  
56 plant pathogens in response to UV-C stress. Furthermore, specific pathogenesis related (PR) proteins  
57 have also been shown to increase in concentration following hormetic UV-C treatment; these  
58 include chitinases and  $\beta$ -1,3-glucanases which interact directly with pathogens to reduce their  
59 viability (Charles *et al.*, 2009).

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

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

77 anthocyanin and Vitamin D<sub>2</sub> 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 120U2G11 CP15/469) obtained from Dr Hönle 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<sup>2</sup> were delivered at an intensity of 20 W m<sup>-2</sup> 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.

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

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

117

### 118 **2.3 Colour measurement**

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

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

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

## 127 **2.4 Pathogen Maintenance and Inoculum Preparation**

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

139

## 140 **2.5 Inoculation and Lesion Measurement**

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

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

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

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

154

## 155 **2.6 Experimental Design and Statistical Analysis**

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

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

164

## 165 **3 Results and Discussion**

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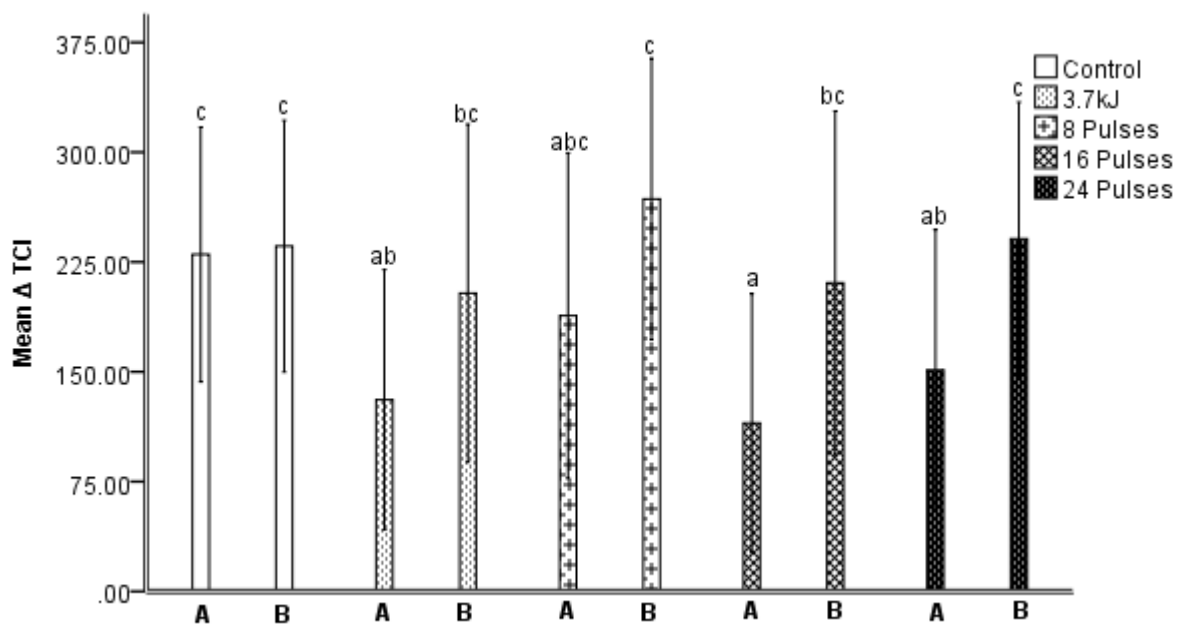
### 167 **3.1 Delayed Ripening**

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

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



176 2. All of the data here supports the successful induction of delayed ripening with either HIPPL or  
 177 LIUV. This data contradicts recently published work by Pataro *et al.* (2015) who observed no effect  
 178 for either LIUV or HIPPL treatments on the ripening of tomato fruit of cv. San Marzano. The HIPPL  
 179 source used by Pataro *et al.* (2015) gave comparable pulse length (360  $\mu$ s) and spectral emission  
 180 (200 to 1100 nm) to that produced by the source used here. The spectral irradiance, i.e. intensity of  
 181 specific wavelengths, however, may have differed to the source used in this study. Furthermore,  
 182 different experimental protocols used by Pataro *et al.*, (2015) may have led to the failure to detect a  
 183 significant difference in colour change for LIUV and HIPPL treated fruits. Specifically, the use of a 14 /  
 184 10 h day and night light cycle during fruit storage may have affected the induction of delayed  
 185 ripening.



186 **Figure 1.** The  $\Delta$  TCI (tomato colour index) from day 0 - 10 of mature green fruit from cv. Mecano.  
 187 Fruit were treated with a hormetic LIUV treatment of 3.7 kJ/m<sup>2</sup> from a low intensity source with  
 188 peak emissions at 254 nm and three high intensity pulsed polychromatic light (HIPPL) treatments of  
 189 8, 16 and 24 pulses. TCI measurements were taken from tissue directly facing the light source (**A**)  
 190 and at 90 ° from the source (**B**). Error bars show  $\pm$  1 standard deviation; n = 30. Labelling indicates  
 191 statistical significance. Means sharing the same label are not significantly different from each other

192 at  $p < 0.05$ .

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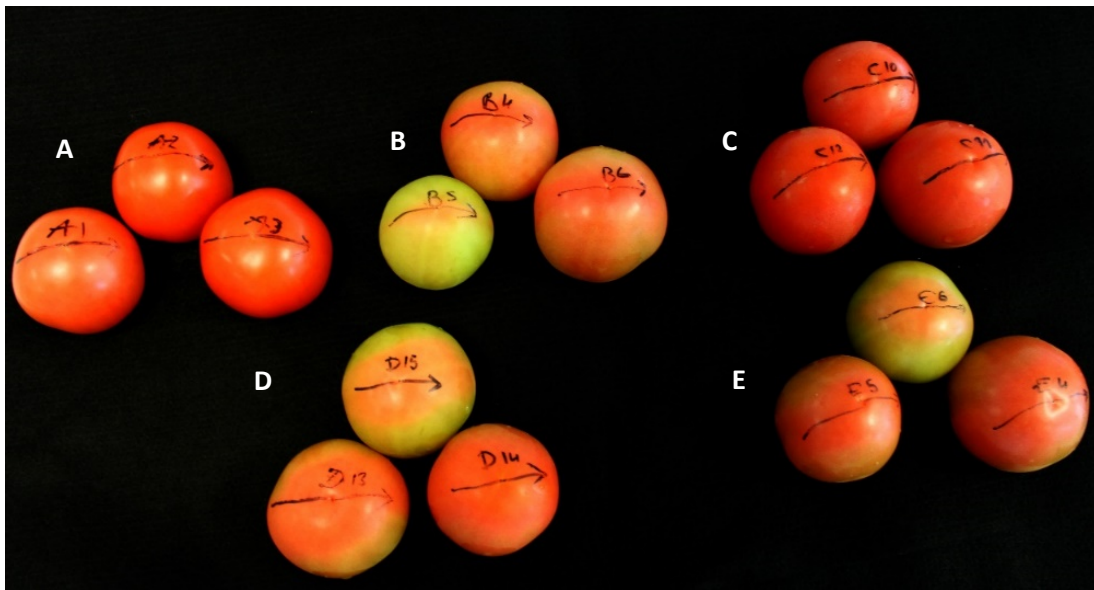
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202 **Figure 2.** Representative samples of tomato fruit of the cultivar Mecano at 10 d post treatment.

203 Groups show the control fruit (A), the 3.7 kJ/m<sup>2</sup> LIUV treatment with peak emissions at 254 nm (B)

204 and fruit treated with the high intensity pulsed polychromatic light (HIPPPL) light source at 8 (C), 16

205 (D) and 24 (E) pulses.

206

207

208 Allowing the fruit to become exposed to visible wavelengths of light following treatment may

209 have led to photoreversal - a phenomenon in which the effects of UV-C induced responses are

210 negated by subsequent exposure to visible light (Kelner, 1949). It had previously been shown by

211 Stevens *et al.* (1998b) that peaches, *Prunus persica*, exposed to 48 h of visible light following UV-C

212 treatment no longer exhibited a reduction in brown rot lesions caused by *Monilinia fructicola*. The

213 influence of photoreversal on the ripening progression of tomato fruit, has not been investigated.

214

### 215 3.2 Direct Tissue Exposure and Delayed Ripening

216 During preliminary work it was noted that the effects of delayed ripening were more

217 pronounced on tissue directly facing the HIPPPL and UV-C sources. To establish whether LIUV and

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

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

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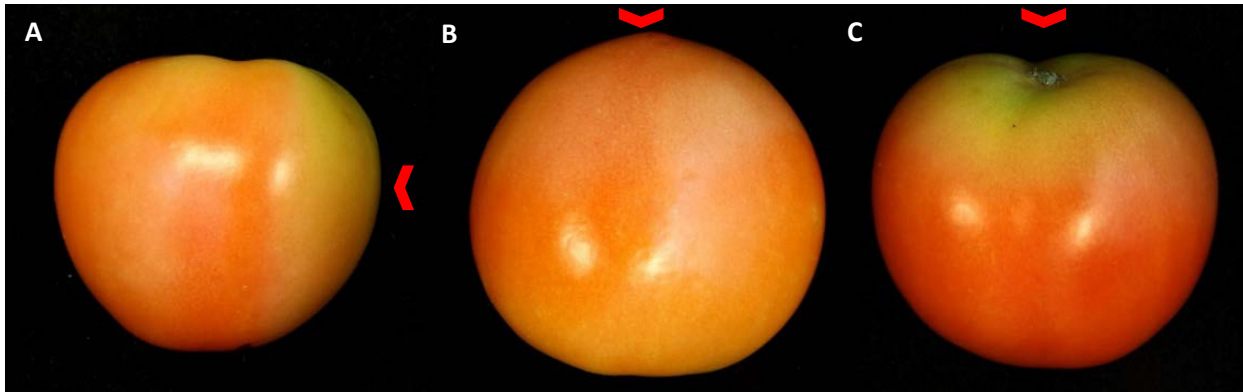
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**Figure 3.** Representative samples of tomato fruit exposed to polychromatic light from different orientations. Fruit, cv. Mecano, were treated with 16 pulses of high intensity pulsed polychromatic light (HIPPL) and photographed at 10 d post treatment. 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.

270 **Table 1.** Area underneath the disease progression curve (AUDPC) from mature green fruit cv.  
 271 Mecano treated with a conventional low intensity UV-C (LIUV) source, with peak emissions at 254  
 272 nm, and an high intensity pulsed polychromatic light (HIPPL) source. Inoculations were performed  
 273 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 <sup>2</sup>	370	43.76 <sup>ab</sup>	25.13	38.14
8 Pulses	5	56.05 <sup>b</sup>	16.82	20.76
16 Pulses	10	41.21 <sup>a</sup>	17.09	41.74
24 Pulses	15	45.15 <sup>ab</sup>	22.91	36.17

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

276

277

278 These results show that HIPPL can induce resistance to *B. cinerea* on mature green tomatoes to  
 279 similar levels to that of LIUV treatment. This is in contrast to the results obtained by Marquenie *et al.*  
 280 (2003) who reported no effect of pulsed light on the disease progression of *B. cinerea* on  
 281 strawberries, *Fragaria ananassa*. This could be due to the employment of a different plant species or  
 282 to differences in the spectral emission of the HIPPL sources. The HIPPL source used by Marquenie *et al.*  
 283 (2003) produced 30 µs pulses at 15 pulses per second (15 Hz). The source in this study, however,  
 284 produces 360 µs pulses at 3.2 pulses per second. Furthermore, the authors reported that the  
 285 percentage of light falling within the UV region was 50 % of a 7 J pulse in contrast to the output  
 286 obtained here (1 % of a 505 J pulse).

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

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

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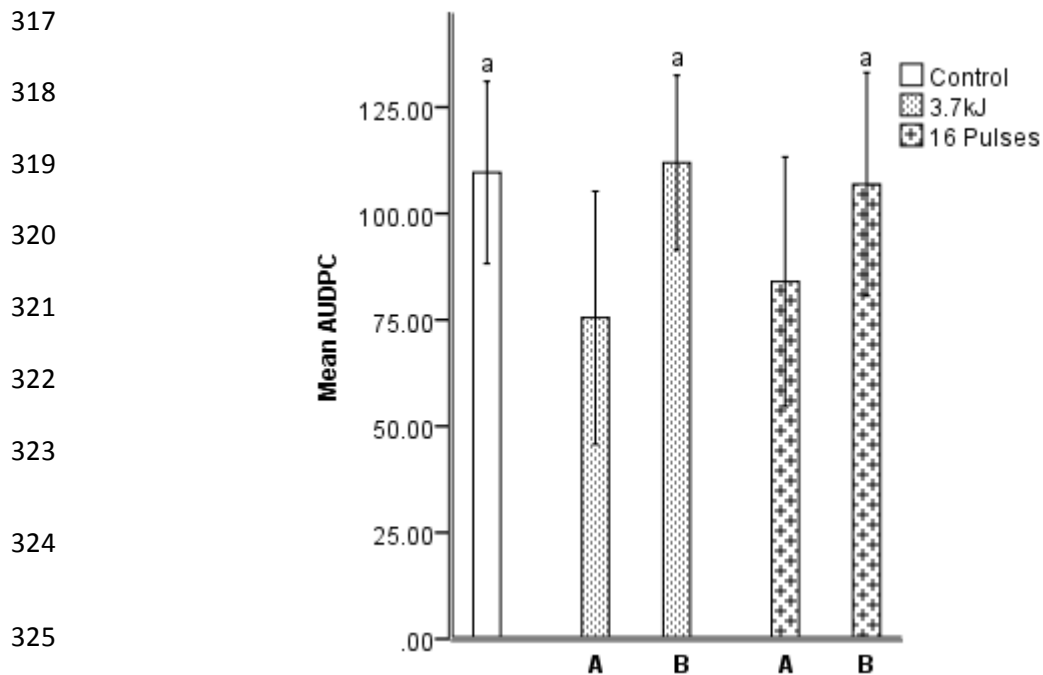
### 295 **3.4 Direct Tissue Exposure and Disease Resistance**

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

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

315

316



**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<sup>2</sup>, 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.

### 342 3.5 Disease Resistance of Ripe Fruit

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

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

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

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

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



363 **4. Conclusions**

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

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

380

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386

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