UV Hormesis in Fruits: A Concept Ripe for Commercialisation

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ABSTRACT

Hormesis is the application of potentially harmful agents at low doses to living organisms in order to induce stress responses. When fruit are exposed to low doses of UV a number of changes are induced including the production of anti-fungal compounds and delays in ripening. Both of these responses could be exploited by the horticultural sector to reduce postharvest losses. We review the results of UV treatment of a variety of fruits and the work done in identifying chemical changes in them. The prospects for treating fruits with UV on a commercial scale are considered.

INTRODUCTION

Fruits are highly perishable products particularly once they have been harvested. So called 'postharvest losses' can arise by various means. For example, attack by fungi and other organisms, or the premature induction of ripening may result from physical damage to the fruit during processing or transportation, or by incorrect storage. The scale of such losses, recently estimated as constituting one third of all harvested produce, is significant cause for concern in developed countries but can be catastrophic for developing countries (Stevens et al., 1997). This review is concerned with one particular strategy – the application of low doses of ultraviolet radiation - for protecting fruits against attack by pathogenic fungi.

The conventional approach to the control of fungi has been the use of synthetic anti-fungal compounds. However, since the 1960s doubts have increasingly been expressed about the safety of many fungicides, and it has emerged that a significant number of commonly used fungicides pose a threat to human health (Wilson, Wisniewski, Biles, McLaughlin, Chalutz & Droby, 1991). Outright bans of chemical fungicides can only sensibly be made once safe and effective alternatives have been identified and much effort is currently being invested by the research community in investigating 'biological control' measures. Biological control has been defined as 'the decrease of inoculum or disease-producing activity of a pathogen accomplished through one or more organisms, including the host plant, but excluding man' (Baker, 1987). Mari and Guizzardi (1998) have recently reviewed emerging technologies in this field and categorised existing approaches; these are, the use of antagonistic organisms, natural defence mechanism enhancement and the use of natural anti-fungal substances. Under this system of classification, hormesis, the subject of this review, comes under the category of natural defence mechanism enhancement.

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HORMESIS

Hormesis has been defined as ‘stimulation by low doses of any potentially harmful agent’ (Luckey, 1980). Hormetic phenomena have been reported since the early 1880s and evidence of such effects has now been accumulated for a very wide range of living organisms. One author has included the term ‘counterintuitive’ in his definition of hormesis (Calabrese, 2002), presumably to reflect the controversy regarding the effects of low doses of ionising radiation on humans. However, such concerns lie outside the scope of the present work.

Although hormetic effects can be induced by both ionising and non-ionising radiation (e.g. UV), it is effects caused by the latter that will be examined here. The UV portion of the electromagnetic spectrum ranges from approximately 10 to 400 nm, however the phenomena described here are concerned with effects induced by UV-C, i.e. wavelengths in the range 100 to 280 nm. These wavelengths are also referred to as ‘far-UV’ and henceforth the abbreviation ‘UV’ will be intended to signify wavelengths within this narrower range.

Luckey (1980) proposed a mechanism for hormesis in which he suggested that low doses of UV could inflict repairable damage to DNA, and that this slight trauma would activate repair mechanisms for radiation-induced DNA damage. This suggests that sub-lethal radiation may stimulate vital processes inside the cells and create a positive change in the homeostasis of a plant.

HORMETIC EFFECTS ON FRUIT

Table 1 is a compilation of recent work on low-dose UV treatment of fruit.

Fruit (Cultivar)	Targeted Pathogen	UV Dose/Dose Range Investigated kJ m^{-2}	Optimal UV Dose kJ m^{-2}	Additional Details	Reference
Apple (Red Delicious)	*Penicilliu m expansum*	7.5	Not determined	The earliest application of UV treatment (96 hours) before inoculating with *P.expansum* provided the best defence against disease. Combining UV irradiation with other disease prevention measures, harpin, chitosan and yeast antagonists *Candida saitoana* and *C. oleophilia* offered no advantages.	De Capdeville, Wilson, Beer & Aist (2002)
Cactus Pear (Gialla)	Not specified	0.75	Not determined	UV treatment did not reduce the incidence of decay. Skin damage observed following irradiation.	Piga, D'hallewin, D'Aquino & Aggabio (1997)
Cherry (several un-named culivars)	*Botrytis cinerea, Monilinia fructigena*	0.5-15.0	-	UV treatment had no affect either on fungal development or fruit quality.	Marquenie et al. (2002)
Grape (Italia)	*Botrytis cinerea*	0.125 – 4.0	0.125 – 0.5	Grapes irradiated 24-48 hours before inoculating with *B. cinerea* showed a lower disease incidence than those inoculated immediately before irradiation. Doses above 1.0 kJ m^{-2} resulted in skin discolouration. Treatment within the optimum range did not significantly reduce the numbers of epiphytic yeasts that showed antagonism towards pathogenic moulds.	Nigro , Ippolito & Lima (1998)
Grapefruit	*Penicilliu m*	0.5 – 3.0	0.5	Quality and disease resistance determined after storage at 7°C for 4 weeks followed by 1 week	D'hallewin, Schirra, Pala & Ben-Yehoshua (2000)

(Star Ruby)	*digitatum*			at 20° C. Scoparone and scopoletin levels were increased at all UV doses. Rind browning and tissue necrosis occurred at UV doses > 1.5 kJ m^{-2}.	
Kumquat (Nagami)	*Penicilliu m digitatum*	0.2 - 15	1.5	Scoparone levels increased following irradiation at all UV exposures. After 2 weeks of storage at 17° C UV-treated fruit showed signs of damage, however at lower temperatures UV damage was practically absent even at the highest dose used.	Rodov, Ben-Yehoshua, Kim, Shapiro & Ittah (1992)

Fruit (Cultivar)	Targeted Pathogen	UV Dose/Dose Range Investigated kJ m^{-2}	Optimal UV Dose kJ m^{-2}	Additional Details	Reference
Lemon (Eureka)	*Penicilliu m digitatum*	0 - 15	5	UV was only effective in suppressing decay in fruit that had been irradiated at least 24 h before inoculation with *P. digitatum*. Increased levels of scoparone were found in irradiated fruits.	Ben-Yehoshua, Rodov, Kim & Carmeli (1992)
Mango (Tommy Atkins)	Not specified	4.9 and 9.9	4.9	Quality and disease resistance determined after storage at 5° C for 14 days followed by 7 days at 20° C. Treatment at 4.9 kJ m^{-2} resulted in improved appearance and texture of fruit. Irradiation induced spermidine and putrescine. The higher dose induced senescence.	Gonzalez-Aguilar, Wang, Buta & Krizek (2001)
Orange (Biondo Comune, Washingt	Not specified	0.5 – 3.0	Not determined	Quality and disease resistance determined after storage at 7°C for 4 weeks followed by 1 week at 20° C. Peel quality was affected in all cultivars with the exception of Valencia L. Percentage of damaged fruit at the higher	D'hallewin, Schirra, Manueddu, Piga & Ben-Yehoshua (1999)

on Navel, Tarocco, Valencia Late)				dosages decreased as the season progressed. UV irradiation at 0.5 kJ m^{-2} was effective in reducing decay development. The higher dose of 1.5 kJ m^{-2} was more effective but only in early harvested fruit. Concentrations of scoparone and scopoletin increased in all varieties with increasing dose.	
Orange (Shamouti, Valencia)	*Penicilliu m digitatum*	0.2 - 15	9.0	After 2 weeks of storage at 17° C UV-treated fruit showed signs of damage, however at lower temperatures UV damage was practically absent even at the highest dose used. Scoparone levels increased following irradiation at all UV exposures.	Rodov et al. (1992)
Peach (Elberta, Loring)	*Monilinia fructicola*	7.5	7.5	Several treatments compared. Best results achieved with UV treatment in combination with *Debaromyces hansenii* and $CaCl_2$. Protection achieved under these conditions was comparable to that achieved with antifungal agent benomyl.	Stevens et al (1997)

Fruit (Cultivar)	Targeted Pathogen	UV Dose/Dose Range Investigated kJ m^{-2}	Optimal UV Dose kJ m^{-2}	Additional Details	Reference
Peach (Elberta)	*Monilinia fructicola*	0.84 - 40	7.5	Exposure to UV delayed ripening, suppressed ethylene production and increased phenylalanine ammonia-lyase activity. Doses of 40 kJ m^{-2} increased susceptibility to brown rot. Irradiation resulted in increased numbers of the antagonist yeast *Debaryomyces hansenii* on the surface of the fruit.	Stevens et al. (1998)

Pepper (Bell Boy, Delphin)	Natural infections and *Botrytis cinerea*	0.22 – 2.20	0.88 for *Botrytis cinerea*	All doses tested provided protection against natural infection. UV provided protection against *B. cinerea* only when artificial inoculation occurred after irradiation but not before. Two successive exposures at 0.44 kJ m^{-2} were equivalent to a single exposure at 0.88 kJ m^{-2}.	Mercier, Baka, Reddy, Corcuff & Arul (2001)
Strawberry (Kent)	*Botrytis cinerea*	0.25-1.0	0.25	Treatment resulted in an extension to shelf life of 4-5 days. Fruits treated at the lower UV dose showed a lower rate of senescence. Some evidence obtained that damage caused at the highest dose tested.	Baka, Mercier, Corcuff, Castaigne & Arul (1999)
Strawberry (Elsanta)	*Botrytis cinerea*, *Monilinia fructigena*	0.5- 15.0	0.5	Fungal development reduced over the entire dose range examined. Fruit treated at the higher doses maintained their firmness better than untreated controls, however browning and drying of the calyx was also observed at these doses. Thermal treatment alone at temps above 45° C caused damage to strawberries but combined treatment at lower temps. enabled lower UV doses to be used.	Marquenie et al. (2002)

Fruit (Cultivar)	Targeted Pathogen	UV Dose/Dose Range Investigated kJ m^{-2}	Optimal UV Dose kJ m^{-2}	Additional Details	Reference
Tangerine (Dancy)	*Penicillium digatutum*	1.3	1.3	Several treatments compared. Best results achieved with UV treatment in combination with *Debaromyces hansenii* and $CaCl_2$.	Stevens et al (1997)
Tomato (Tuskegee 80-130, Floradade, Better Boy)	*Alternaria alternata* *Botrytis cinerea* *Rhizopus stolonifer*	1.3 - 40	3.6 – 7.5	UV doses of 3.6 and 4.8 kJ m^{-2} delayed ripening whilst doses of 40 kJ m^{-2} resulted in skin discolourization.	Liu et al.. (1993)
Tomato (Tuskegee 80-130, Floradade)	*Rhizopus stolonifer*	3.6	3.6	Several treatments compared. Best results achieved with UV treatment in combination with *Debaromyces hansenii* and $CaCl_2$.	Stevens et al (1997)
Tomato (Capello)	_	3.7 – 24.4	3.7	Study aimed at delaying senescence only. Treated fruit were stored at 16° C for 35 days. High UV doses caused abnormal browning of the surface of fruits. Treatment with doses of 3.7 kJ m^{-2} delayed ripening for 7 days. This correlated with increased amounts of putrescine in the fruits.	Maharaj, Arul & Nadeau (1999)

Table 1. Low UV Dose Treatment of Fruit

In all cases, with the exception of cactus pears and cherries, positive results were achieved as a result of treatment. This may be taken as meaning either induced resistance to pathogenic fungi or delayed ripening. In neither of the two exceptional cases, cherries (Marquennie et al., 2002) and cactus pears (Piga et al., 1997), did the authors offer explanations as to why the treatment had failed to induce beneficial effects. It should however be noted that low UV doses produced no ill effects on these particular fruit.

It is important to consider separately all the consequences of treating fruit with UV in order to assess the overall impact both on the fruit itself and on any pathogenic fungi associated with the fruit.

Direct Inactivation by UV of Surface-Associated Fungi.

Because the UV wavelengths employed to elicit hormetic effects are strongly absorbed by the DNA of living organisms, consideration must be given to the possibility that the UV doses delivered to fruit can directly inactivate fungi or fungal spores that may be present at the surface of the fruit. Indeed, the wavelength range in question is sometimes referred to as 'germicidal'. In such instances fungal inactivation occurs once a sufficiently high UV dose had been accumulated by the organism. If inactivation of this kind were to occur, it would be limited solely to the surface of the fruit as UV has extremely limited penetration into solids (Gardner and Shama, 2000).

Quantitative information on the inactivation of micro-organisms on the surfaces of solids is scarce. Most of the data available comes from work in which the organisms were irradiated in dilute suspension in a UV- transparent liquid. Microbial associations on surfaces can be quite complex and fundamentally different from those of cells in suspension in liquids. For example, microbial growth at a surface may result in a situation where the cells closest to the surface are effectively protected from the lethal effects of UV by those cells at the periphery. In addition, the surface features or 'topography' such as ridges or crevices may play a role in shielding organisms from incident UV.

Despite these factors, there is strong evidence to show that direct inactivation of surface-associated populations of micro-organisms can occur at the dose levels used to induce hormesis. Stevens et al. (1998) inoculated the surfaces of peaches with spores of the pathogenic fungus *Monilinia fructicola* and then subjected the peaches to UV exposure. At a dose of 4.8 kJm^{-2}, a decrease in viability of *M. fructicola* of approximately one order of magnitude was observed.

It seems likely that direct inactivation may have been a factor in other examples listed in Table 1, even where the possibility was not accounted for in the experimental design. A compilation of inactivation data for fungi and other organisms (Meulemans, 1987) shows a distribution ranging from approximately 0.1 to 1.0 kJm^{-2} in the UV doses needed to bring about a decrease in viability for representatives of the genera listed in Table 1 of one log order. This implies that even at the lower doses listed in the Table, at least some inactivation of particularly susceptible fungi might occur.

Interestingly, Stevens et al. (1998) also investigated the effect of UV on yeast population naturally associated with peaches. The yeast population at the surface of the fruit actually increased as a result of irradiation at UV doses which resulted in a population decline when the yeast were exposed to identical doses on the surface of paper discs. This prompted the workers to conclude that yeast growth was under the genetic control of the fruit. They went on to show that one of the yeasts, *Debaromyces hansenii*, was an antagonist of *M. fructicola* and that synergistic inhibition of the fruit pathogen could be adopted by combined application of *D. hansenii* and UV treatment.

Induced Effects

Authentic hormetic responses in fruit should be viewed as distinct from the direct effects of UV on surface-associated fungi described above. The treatment of fruit with low UV doses results in the synthesis in the fruit of a number of anti-fungal compounds. The nature of some of these compounds has been identified for certain fruits as we describe below. Synthesis is initiated by UV treatment but continues to occur for periods measured in days *after* the irradiation event. Significantly, synthesis of such compounds occurs throughout the entire fruit. Proof of this is provided by results from studies referred to in Table 1. In instances where fruit were artificially inoculated with fungal pathogens, the investigators took precautions to ensure that inoculation was made deep within the tissue of the fruit, i.e. at depths from the surface where direct-UV inactivation could be completely discounted.

In citrus fruits, enhancement of resistance to green mould caused by *Penicillium digitatum* has been attributed to accumulation of the phytoalexins scoparone (6,7-dimethoxycoumarin) (Kim, Ben-Yehoshua, Shapiro, Henis & Camely 1991; Rodov et al., 1992; D'hallewin et al., 1999) and scopoletin (7-hydroxy-6-methoxycoumarin) (D'hallewin et al., 2000), the induction of phenylalanine amonia lyase (PAL) and peroxidase enzymes (Droby et al., 1993) and the induction of pathogenesis-related (PR) proteins (chitinases and β-1,3-endoglucanases) (Porat et al., 1999). The concentration of scoparone in lemon fruit increased up to 7 days after exposure to UV irradiation and then declined (Kim et al., 1991). Similarly, in kumquat, the phytoalexin reached a peak at 11 days and then rapidly declined (Rodov et al., 1992). With grapefruit, induced resistance seemed to develop gradually and appeared to be correlated with PAL activity in the peel of UV irradiated fruit within 24 hours of treatment and remained high at 48 hours (Chalutz, Droby, Wilson & Wisniewski, 1992). This was suggested to be an induced mechanism of resistance.

Of the PR-proteins produced by plants, chitinases, glucanases and lysozymes have the ability to hydrolyse insoluble polysaccharides from the cell walls of fungi and bacteria. Glucanases and chitinases inhibit fungi by hydrolysing β-1,3-ether linkages in β-1,3-glucans and β-1,3-1,6-glucans and hydrolysing β-1,4-ether linkages in poly-β-1,4-N-acetylglucosamine (chitin). Lysozymes and some chitinases also inhibit bacteria, with the lysozymes hydrolysing the carbohydrate component of bacterial peptidoglycan (poly-[β-1,4-N-acetylglucosamine-β-1,4-N-acetylmuramic acid]). At the tip of fungal hyphae, glucanases appear to be associated with the removal of the outer cell wall layer containing β-linked glucans and chitinases loosen and detach

fibrils from the cell wall. Consequently, fungal hyphae treated with chitinases become swollen at the tip. Mauch, Mauch-Mani & Boller (1988) working with pea pods infected with fungi observed a synergistic action of glucanases and chitinases, each providing better access for the other to their respective substrates.

In addition to their direct role as antifungal enzymes, chitinases and glucanases are thought to release from fungal cell walls elicitors of PR-genes. In this way, plants may recognise chitin fragments from invading fungi (Felix, Regenass and Boller, 1993), with the consequent production of phytoalexins.

In peach, UV irradiation resulted in induction of chitinase, β-1,3-glucanase and PAL activities as little as six hours after treatment. Activities were preceded by activation of the corresponding genes and peaked at 96 hours (El Ghaouth, Wilson & Callahan, 2003). This suggests that UV mediated these biochemical defence responses in peach. The synthesis of phenols, phytoalexins and lignins, that are all associated with local resistance processes, involves PAL as a key step in the shikimic acid pathway (Ryalls Neuenschwander, Willits, Molina, Steiner & Hunt, 1996).

Considerable recent interest has been shown in applying low UV doses to grapes. The work of Nigro et al. (1998) clearly shows that defence against *B. cinerea* occurs maximally 24 to 48 hours after UV treatment. They attributed the protective effect to a number of possible factors including PAL and peroxidase activity as well as the induction of 'stilbene-like phytoalexins' such as resveratrol (3, 5, 4′-trihydroxystilbene). Adrian, Jeandet, Douillet- Breuil, Tesson & Bessis (2000) showed that infection by *B. cinerea* could itself induce resveratrol formation and that UV irradiation increased resveratrol concentration in all the varieties of grape studied. However, where the grapes were already infected with *B. cinerea*, resveratrol induction was suppressed. They concluded that either the stilbene was metabolised by *B. cinerea* or that UV elicitation is less efficient in 'pre-induced' grapes - that is grapes that had already been subject to stress by fungal infection. Delayed induction of resveratrol and related stilbenes in grapes, including oligomers of resveratrol such as the viniferins, were demonstrated by Cantos, Espin & Tomas-Barberan (2002) following UV treatment. This group of workers had earlier (Cantos, Espin & Tomas-Barberan, 2001) gone on to advocate UV-irradiated grapes as a functional food owing to the health-enhancing properties of resveratrol. More recently they showed that the resveratrol content of wines can be increased by irradiating with UV the grapes used to produce the wines (Cantos, Espin, Fernandez, Oliva & Tomas-Barberan, 2003).

Delay of Ripening

Fruit ripening, like other plant development processes, is under the control of plant growth regulators of which ethylene plays a key role, however polyamines have also been implicated in the control of ripening (Seymour, Taylor & Tucker, 1996). In climacteric fruit, respiration increases during ripening and reaches a peak known as the 'respiratory climacteric', whereas in non-climacteric fruit respiration gradually declines during ripening. Ethylene is the first detectable sign of ripening in climacteric fruits with a characteristic burst of ethylene production that occurs during ripening and precedes the respiratory climacteric. The eventual autocatalytic ethylene

production in such fruits enhances ripening with a consequent reduction in their shelf life. In non-climacteric fruits, there is no autocatalytic production of ethylene, however exogenous application of ethylene increases the rate of respiration and consequently the rate of ripening, the whole process depending on the continuous presence and concentration of exogenous ethylene. During ripening, changes in colour and texture are under the control of ethylene, however flavour development in fruit is not. In tomato fruits, the pectin degrading enzyme polygalacturonase appears and accumulates at the onset of ripening and contributes to the softening of cell walls that is part of the changes that occur in the fruit as they ripen.

Polyamines are a group of nitrogen-containing compounds that accumulate in plants in response to environmental stress (Evans & Malmberg, 1989). They are low molecular weight polycations, and the polyamine putrescine is the precursor for spermidine and spermine found in all organisms. They exist in plants in both free and bound forms, i.e. conjugated to phenolic compounds. Polyamines and ethylene may compete for the intermediate S-adenosylmethionine (SAM) which provides the propylamine moiety for their biosyntheses. Polyamines have been implicated in fruit development with the observation of high levels of free and bound forms during early fruit growth, especially spermine. Changes in levels of polyamines have been linked to senescence suggesting that lowering of polyamine concentration is a step in triggering senescence or that exogenous application of polyamines inhibits senescence. The latter may be due to the possible inhibition of ethylene synthesis and to stabilisation and protection of membranes by associating with negatively charged phospholipids. Maharaj et al. (1999) also showed that optimal doses of UV produced higher levels of free and conjugated polyamines, particularly putrescine, compared with the control in mature green tomato fruits. Levels of putrescine seem to increase in plants subjected to stress and this includes UV irradiation treatments.

Fruit of tomato landrace Alcobaca, that ripen more slowly and store better than horticultural varieties, have an increase in putrescine content at later stages of development Dibble, Davies & Mutschler (1988). This genotype lacks the pattern of carbon dioxide and ethylene production normally associated with climacteric fruit and this was attributed to the elevated levels of polyamines. Thus high levels of endogenous polyamines have similar effects to those of applications of exogenous polyamines.

Liu et al. (1993) reported that UV treated tomatoes were firmer in texture and less red in colour indicating a delay in ripening. Stevens et al. (1998) attributed delayed ripening of UV treated tomatoes to high levels of putrescine and spermine. There was also a correlation between high concentration of the glycoalkaloid tomatine in the fruit at 72 hours and resistance to *Rhizopus stolonifer*. The anti-senescent activity of polyamines may also relate to their effectiveness as free radical scavengers (antioxidants) (Maharaj et al., 1999).

During the first five days after treatment of tomato fruit with UV ($3.7kJm^{-2}$), Barka, Kalantari, Makhlouf, & Arul (2000a) observed significant induction of lipid peroxidation markers, suggesting that the cell membrane was the primary target for UV irradiation. The levels then dropped lower than in the control fruit, suggesting the induction of a defence or repair mechanism. An increase in lipoxygenase and PAL activities was observed during the first five days, then it declined below the levels in

the control (Barka et al., 2001). This suggests that UV treatment results in rapid accumulation of photooxidation products and that plants react by stimulating their defence mechanisms against oxidation. Barka, Kalantari, Makhlouf, & Arul (2000b) also noted less activity of cell wall-degrading enzymes, i.e. polygalacturonase, pectin methyl esterase, cellulase, xylanase, β-D-galactosidase and protease, in tomato fruit treated with UV irradiation.

Delay of ripening by UV treatment has been reported in peaches (Stevens et al., 1998) and this was associated with enhanced activity of PAL and suppression of ethylene production. Chalutz et al. (1992) also showed that PAL was increased in citrus fruits by UV treatment. In grapefruit, the PAL activity increased within 24 hours of the treatment. In peach there was a brief initial increase in ethylene prior to the PAL activity, probably due to the UV treatment acting as a physical stress to the cells. Interestingly, the optimum doses of UV for the induction of resistance to brown rot (*M. fructicola*) and for delaying ripening in peach are almost identical, suggesting that the processes are either identical or under the same control, with potential for the control of delayed ripening in practice.

In mango, higher levels of polyamines in fruits treated for 10 minutes compared with 20 minutes was suggested to be related to the suppression of decay and softening caused by microbial growth (Gonzalez-Aguilar et al., 2001). It was proposed that UV irradiation induced and activated decay-resistance mechanisms, e.g. by increase in anti-fungal compounds in the fruit peel.

An additional positive effect of UV treatment is the enhancement of levels of anthocyanins in strawberries (Baka, Mercier, Corcuff, Castaigne & Arul, 1999) and red apples (Dong, Mitra, Kootstra, Lister & Lancaster, 1995). Accumulation of anthocyanins in apple skins occurred from *de novo* synthesis of PAL and chalcone isomerase following UV-B treatment and it was suggested that there could be a similar effect of short exposure to high energy UV-C irradiation. In grapes, accumulation occurred in the skins when berry sections were exposed to UV irradiation, leading to the suggestion that there may be benefits from UV permeability of covering materials in the protected culture of grapes (Kataoka, Sugiyama & Beppu, 2003).

Although de Capdeville et al. (2002) found that fresh apples were more responsive to treatment than were fruit stored for 3 months in a controlled atmosphere environment, relatively little is currently known about how fruit with different postharvest 'ages' respond to UV treatment. This is clearly one area in which more work is needed.

Adverse Effects

Table 1 shows that the UV doses necessary to achieve optimal beneficial effects in fruits range from 0.5 kJ/m^2, for strawberries, to 9.0 kJ/m^2 for oranges. Maximum UV doses are limited ultimately by the induction in the fruit of undesirable changes – hormetic effects are, after all, brought about by agents which are harmful at high doses. These undesirable changes include skin discoloration in tomatoes (Lui et al., 1993; Maharaj et al., 1999), browning and drying of calyxes in strawberries (Marquenie et al., 2002), increasing susceptibility to brown rot in peaches (Stevens et

al., 1998) and premature ripening in mangoes (Gonzalez-Aguilar et al., 2001). Prolonged exposure of tomato fruits to UV has been found to accelerate ripening and senescence of tomatoes (Liu et al., 1993). This may be explained by UV irradiation promoting photo-oxidation reactions in plants via production of activated oxygen species. The generated free radicals target cell membranes, nucleic acids, cell walls and enzymes resulting in acceleration of senescence (Stapleton, 1992; Foyer, Descourviers & Kunert, 1994). All of these adverse effects would contribute to postharvest losses and any proposed treatment methods should have in place measures for carefully controlling the maximum UV dose that can be delivered to individual fruits.

FUTURE TRENDS

If UV treatment of fruits is to become a reality, efficient means of irradiating fruits on a large scale must be found. An obvious constraint is that the cost of any such technology should not add unduly to postharvest handling costs - though it should be borne in mind that there would be cost savings in eliminating chemical fungicides. Ideally, any processing equipment developed should be able to handle a wide variety and geometries of fruits and do so without inflicting physical damage on the fruit.

At the heart of any treatment process will be the UV sources. The most convenient source of far-UV is the low pressure mercury burner (Schenk, 1987). Relatively inexpensive and operating optimally at temperatures in the region of 60° C, these sources emit UV principally at 254 nm. Sources of this kind were used in all the studies listed in Table 1. Moreover, in each of these studies an approximately even dosage over the entire surface of the fruit was achieved by manually rotating the fruit during exposure to UV. It appears to be tacitly assumed that the entire surface of the fruit should be irradiated to induce maximum protection, and whilst this has been shown to be the case for carrots (Mercier, Roussel, Charles & Arul, 2000), it does not necessarily follow that this principle holds for all fruits, and further work is needed to verify this.

The most obvious way of irradiating fruit would be to convey them past suitably arranged UV sources. One patent for achieving this was granted to Brandt and Klebaum (2000). In their invention, the conveyor, comprising one or more rollers, induces rotation of the fruit in order to ensure that a high proportion of the surface receives exposure.

An interesting proposal for irradiating plants in the field was patented by Michaloski (1991). In this invention, banks of UV sources are attached to a vehicle that can be pulled between rows of vine plants using, for example, a tractor. The sources are arranged so that individual vines receive irradiation from two lateral directions. Although the purpose was to eradicate powdery mildew from vines, it may have some relevance to the hormetic treatment of fruits such as tomatoes grown as bushes. The concept of treating fruit whist still on the plant seems not to have received attention and is an area meriting further work.

As mentioned above, clear evidence has been accumulated to show that irradiating grapes with UV can substantially increase their resveratrol content. Recent work is summarised in Table 2.

Grape Variety	Resveratrol Induction (-fold)	Reference
Crimson	100	Cantos et al. (2002)
Flame	3.4	
Napoleon	4.4	
Red Globe	2316	
Chardonnay	18.3	Adrian et al.(2000)
Gamay	16.0	
Pinot Noir	∞*	

Table 2. Resveratrol Induction in Grapes Following UV Irradiation

*Final conc., 30.2 μg/ g fresh weight of skin

The fact that in this particular case, irradiation might be carried commercially to produce what has been described as a 'functional food' (Cantos et al., 2001), rather than to protect against fungal attack is largely irrelevant; the technical challenge of irradiating grapes remains the same. In all the experimental studies cited above, the grapes were individually irradiated. It seems questionable as to whether consumers would accept grape berries detached from bunches and therefore a strategy for irradiating whole bunches may be required. This would require a method of ensuring that berries at the outside of the bunch did not receive excessive doses of UV whilst those towards the centre of the bunch received a sufficiently high dose to induce a sufficiently high concentration of resveratrol formation. This would have to be achieved without bruising the berries.

References

Adrian, M., Jeandet, P., Douillet-Breuil, A. C., Tesson, L. & Bessis, R. (2000). Stilbene content of mature Vitis vinifera berries in response to UV-C elicitation. *Journal of Agricultural and Food Chemistry*, 48, 6103-6105.

Baka, M., Mercier, J., Corcuff, R., Castaigne, F. & Arul, J. (1999). Photochemical treatment to improve storability of fresh strawberries. *Journal of Food Science*, 64, 1068-1072.

Baker, K. F. (1987). Evaluating concepts of biological control of plant pathogens. *Annual Review of Phytopathology*, 25, 67-85.

Barka, E. A. (2001). Protective enzymes against oxygen species during ripening of tomato (*Lycopersicon esculentum*) fruits in response to low amounts of UV-C. *Australian Journal of Plant Physiology*, 28, 785-791.

Barka, E. A., Kalantari, S., Makhlouf, J., & Arul, J. (2000a). Effects of UV-C irradiation on lipid peroxidation markers during ripening of tomato (*Lycopersicon esculentum* L.) fruits. *Australian Journal of Plant Physiology*, 27, 147-152.

Barka, E. A., Kalantari, S., Makhlouf, J., & Arul, J. (2000b). Impact of UV-C irradiation on the cell wall-degrading enzymes during ripening of tomato (*Lycopersicon esculentum* L.) fruit. *Journal of Agricultural and Food Chemistry*, 48, 667-671.

Ben-Yehoshua, S., Rodov, V., Kim, J.J., & Carmeli, S. (1992). Preformed and induced antifungal materials of citrus fruits in relation to the enhancement of decay resistance by heat and ultraviolet treatments. *Journal of Agricultural and Food Chemistry*, 40, 1217-1221.

Brandt, D. J., & Klebaum, C. A. (2000). *Method and apparatus for a UV light disinfection system.* US Patent, 6,132,784, Oct. 17.

Brown, J. E., Lu, T. Y., Stevens, C., Khan, V. A., Lu, J. Y., Wilson, C. L., Collins, D. J., Wilson, M. A., Igwegbe, E. C. K., Chalutz, E., & Droby, S. (2001). The effect of low dose ultraviolet light-C seed treatment on induced resistance in cabbage to black rot (*Xanthomonas campestris pv campestris*). *Crop Protection*, 20, 873-883.

Calabrese, E. J. (2002). Hormesis: changing view of the dose-response, a personal account of the history and current status. *Mutation Research*, 511, 181-189.

Cantos, E., Espin, J. C., & Tomas-Barberan, F. A. (2001). Postharvest induction modeling method using UV irradiation pulses for obtaining resveratrol-enriched table grapes: a new "functional" fruit? *Journal of Agricultural and Food Chemistry*, 49, 5052-5058.

Cantos, E., Espin, J. C., & Tomas-Barberan, F. A. (2002). Postharvest stilbene enrichment of red and white table grape varieties using UVC irradiation pulses. *Journal of Agricultural and Food Chemistry*, 50, 6322-6329.

Cantos, E., Espin, J. C., Fernandez, M. J., Oliva, J. & Tomas-Barberan, F. A. (2003). Postharvest UV-C-irradiated grapes as a potential source for producing stilbene-enriched red wines. *Journal of Agricultural and Food Chemistry*. 51, 1208-1214.

de Capdeville, G., Wilson, C. L., Beer, S. V., & Aist, J. R. (2002). Alternative disease control agents induce resistance to blue mold in harvested 'red delicious' apple fruit. *Phytopathology*, 92, 900-908.

Chalutz, E., Droby, S., Wilson, C. L., & Wisniewski, M. E. (1992). UV-induced resistance to postharvest diseases of *Citrus* fruit. *Journal of Photochemistry and Photobiology*, 15, 367-374.

D'hallewin, G., Schirra, M., Manueddu, E., Piga, A., & Ben-Yehoshua, S. (1999). Scoparone and scopoletin accumulation and ultraviolet-C induced resistance to postharvest decay in oranges as influenced by harvest date. *Journal of the American Society of Horticultural Science*, 124, 702-707.

D'hallewin, G., Schirra, M., Pala, M., & Ben-Yehoshua, S. (2000). Ultraviolet C irradiation at 0.5 kJ. M-2 reduces decay without causing damage or affecting postharvest quality of star ruby grapefruit (*C. paradisi* Macf.). *Journal of Agricultural and Food Chemistry*, 48, 4571-4575.

Dibble, A. R. G., Davies, P. J., & Mutschler, M. A. (1988) Polyamine content of long keeping alcobaca tomato fruit. *Plant Physiology*, 86, 338-340.

Dong, Y. H., Mitra, D., Kootstra, A., Lister, C., & Lancaster, J. (1995). Postharvest stimulation of skin color in Royal gala apple. *Journal of the American Society of Horticultural Science*, 120, 95-100.

Droby, S., Chalutz, E., Horev, B., Cohen, L., Gaba, V., Wilson, C. L., & Wisniewski, M. (1993). Factors affecting UV-induced resistance in grapefruit against the green mold decay caused by *Penicillium digitatum*. *Plant Pathology*, 42, 418-424.

El Ghaouth, A., Wilson, C. L., & Callahan, A. M. (2003) Induction of chitinase, β-1,3-glucanase, and phenylalanine ammonia lyase in peach fruit by UV-C treatment. *Phytopathology*, 93, 349-355.

Evans, P. T. & Malmberg, R. L. (1989) Do polyamines have roles in plant development? *Annual Review of Plant Physiology and Plant Molecular Biology*, 40, 235-269.

Felix, G., Regenass, M., & Boller, T. (1993) Specific perception of subnanomolar concentrations of chitin fragments by tomato cells: induction of extracellular alkalinization, changes in protein phosphorylation and establishment of a refractory state. *Plant Journal*, 4, 307-316.

Foyer, C. H., Descourvieres, P., & Kunert, K. J. (1994). Protection against oxygen radicals: an important defense mechanism studied in transgenic plant. *Plant and Cell Environment*, 17, 507-523.

Gardner, D. W. M. & Shama, G. (2000). Modeling UV-induced inactivation of microorganisms on surfaces. *Journal of Food Protection*, 63**,** 63-70.

Gonzalez-Aguilar, G. A., Wang, C. Y., Buta, J. G., & Krizek, D. T. (2001). Use of UV-C irradiation to prevent decay and maintain postharvest quality of ripe 'Tommy Atkins' mangoes. *International Journal of Food Science and Technology*, 36, 767-773.

Kataoka, I., Sugiyama, A., & Beppu, K. (2003). Role of ultraviolet radiation in accumulation of anthocyanin in berries of 'Gros Colman' grapes (*Vitis vinifera* L.). *Journal of the Japanese Society for Horticultural Science*, 72, 1-6.

Kim, J. J., Ben-Yehoshua, S., Shapiro, B., Henis, Y., & Camely, S. (1991). Accumulation of scoparone in heat-treated lemon fruit inoculated with *Penicillium digitatum* Sacc. *Plant Physiology*, 97, 880-885.

Liu, J., Stevens, C., Khan, V. A., Lu, J. Y., Wilson, C. L., Adeyeye, O., Kabwe, M. K., Pusey, P. L., Chalutz, E., Sultana, T., & Droby, S. (1993). Application of ultraviolet-C light on storage rots and ripening of tomatoes, *Journal of Food Protection*, 56, 868-872.

Luckey, T. D. (1980). *Hormesis with Ionizing Radiation*. Boca Raton, CRC Press.

Maharaj, R., Arul, J., & Nadeau, P. (1999). Effect of photochemical treatment in the preservation of fresh tomato (*Lycopersicon esculentum* cv. Capello) by delaying senescence. *Postharvest Biology and Technology*, 15, 13-23.

Mari, M. & Guizzardi, M. (1998). The postharvest phase: emerging technologies for the control of fungal diseases. *Phytoparasitica*, 26,59-66.

Marquenie, D., Michiels, C.W., Geeraerd, A.H., Schenk, A., Soontjens, C., Van Impie, J. F., & Nicolai, B. N. (2002). Using survival analysis to investigate the effect of UV-C and heat treatment on storage rot of strawberry and sweet cherry. *International Journal of Food Microbiology*, 73, 187-196.

Mauch, F., Mauch-Mani, B., & Boller, T. (1988) Antifungal hydrolases in pea tissue. II. Inhibition of fungal growth by combinations of chitinase and β-1,3-glucanase. *Plant Physiology*, 88, 936-942.

Mercier, J., Roussel, D., Charles, M.T., & Arul, J. (2000). Systemic and local responses associated with UV- and pathogen-induced resistance to *Botrytis cinerea* in stored carrot. *Phytopathology*, 90, 981-986.

Mercier, J., Baka, M., Reddy, B., Corcuff, R., & Arul, J. (2001). Shortwave ultraviolet irradiation for control decay caused by *Botrytis cinerea* in bell pepper:

induced resistance and germicidal effects. *Journal of the American Society of Horticultural Science,* 126, 128-133.

Meulemans, C. C. E. (1987) The basic principles of UV-disinfection of water. *Ozone Science and Technology,* 9, 299-314.

Michaloski. A.J. (1991). *Method and apparatus for ultraviolet treatment of plants*. US Patent. 5,040,329, Aug. 20.

Nigro, F., Ippolito, A., & Lima, G. (1998). Use of UV-C to reduce storage rot of table grape, *Postharvest Biology and Technology,* 13, 171-181.

Piga, A., D'hallewin, G., D'Aquino, S. & Aggabio, M. (1997). Influence of film wrapping and UV irradiation on cactus pear quality after storage. *Packaging Technology and Science,* 10, 59-68.

Porat, R., Lers, A., Dori, S., Cohen, L., Weiss, B., Daus, A., Wilson, C. L., & Droby, S. (1999). Induction of chitinase and β-1,3-endoglucanase proteins by UV irradiation and wounding in grapefruit peel tissue. *Phytoparasitica*, 27, 233-238.

Rodov, V., Ben-Yehoshua, S., Kim, J. J., Shapiro, B., & Ittah, Y. (1992). Ultraviolet illumination induces scoparone production in kumquat and orange fruit and improves decay resistance, *Journal of the American Society of Horticultural Science,* 117, 788-792.

Ryalls, J. A., Neuenschwander, U. H., Willits, M. G., Molina, A., Steiner, H. Y., & Hunt, M. D. (1996) Systemic acquired resistance. *Plant Cell*, 8, 1809-1819.

Schenk, G. O. (1987). Ultraviolet sterilization. In W. Lorch, *Handbook of Water Purification,* Second Edition (pp. 530 – 595). Chichester: Ellis Horwood.

Seymour, G. B., Taylor, J. E., & Tucker, G. A. (1996). Biochemistry of Fruit Ripening. Chapman & Hall, London.

Stapleton, A. E. (1992). Ultraviolet radiation and plants: Burning questions. *Plant Cell,* 4, 1353-1358.

Stevens, C. Khan, V. A., Lu, J.Y., Wilson, C. L., Pusey, P. L., Igwegbe, E. C. K., Kabwe, K., Mafolo, Y., Liu, J., Chalutz, E., & Droby, S. (1997). Integration of ultraviolet (UV-C) light with yeast treatment for control of postharvest storage rots of fruits and vegetables. *Biological Control,* 10, 98-103.

Stevens, C., Khan, V. A., Lu, J. Y., Wilson, C. L., Pusey, P. L., Kabwe, M. K., Igwegbe, E. C. K., Chalutz, E., & Droby, S. (1998). The germicidal and hormetic effects of UV-C light on reducing brown rot disease and yeast microflora of peaches. *Crop Protection,* 17, 75-84.

Wilson, C. L., Wisniewski, M.E., Biles, C. L., McLaughlin, R., Chalutz, E. & Droby, S. (1991). Biological control of post-harvest diseases of fruits and vegetables. alternatives to synthetic fungi. *Crop Protection,* 10, 172-177.