Research Article

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1-Methylcyclopropene (1-MCP) effects on natural disease resistance in stored sweet potato

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Abstract

BACKGROUND: The potential of 1-methylcyclopropene (1-MCP) to maintain postharvest storage of sweet potato was studied. In two separate experiments, the orange-fleshed sweet potato cv. Covington was treated with 1-MCP (1.0 μL L^{-1}, 24 h) and roots stored at 15 °C. During storage, samples were evaluated for the respiration rate, sprout growth, weight loss, incidence of decay and changes in dry matter. The roots were further assayed for the temporal changes in individual non-structural carbohydrates and phenolic compounds in the skin and flesh tissues of the proximal (stem end), middle and distal (root end) regions.

RESULTS: 1-MCP treatment reduced root weight loss and decay but respiration rate and non-structural carbohydrates were not affected. No sprouting was recorded irrespective of the treatment. 1-MCP transiently suppressed the accumulation of individual phenolic compounds, especially in the middle and distal segments. This accentuated the proximal dominance of phenolic compounds. Isochlorogenic acid A and chlorogenic acid were the dominant phenolics in the skin and flesh tissues, respectively.

CONCLUSION: 1-MCP treatment may have an anti-decay effect and reduce weight loss. Therefore, storage trials that involve the use of continuous ethylene supplementation to inhibit sprout growth may be combined with 1-MCP to alleviate ethylene-induced weight loss and decay in sweet potato.

Supporting information may be found in the online version of this article.

Keywords: respiration; decay; weight loss; sugars; phenolics

INTRODUCTION

Sweet potato (*Ipomoea batatas* (L.) Lam.) has great potential for improving food security in the Tropics owing to the numerous agronomic advantages and nutritional benefits.[1] In many parts of Africa, sweet potato, especially the *β*-carotene-rich orange-flesh varieties, is being promoted as a public health tool for improved child and maternal nutrition. 'Covington' is an orange-fleshed, smooth-skinned and rose-coloured sweet potato variety developed by North Carolina State University. It is moderately resistant to many diseases and has an agreeable flavour, making it a preferred table stock[2] and for food security initiatives.

Despite the growing importance, sweet potato roots have an inherently short postharvest storage life of about 2–3 weeks at ambient temperature.[3] Root decay represents the most significant cause of economic loss in sweet potato.[4] In addition, the dormancy phase is readily abbreviated by storage under favourable growth conditions; this is followed by excessive sprout growth, which reduces the marketable value. Sprout growth in both potato (*Solanum tuberosum*) and sweet potato can be inhibited by sustained exposure to exogenous ethylene.[5–7] However, ethylene has detrimental effects on both crops. In potato, the use of ethylene to suppress sprout growth can trigger the accumulation of sugars and leads to darkening of the fry colour. This deleterious effect can be reduced by manipulating ethylene scheduling.[5,8] Alternatively, pre-treatment of potato with the ethylene binding inhibitor 1-methylcyclopropene (1-MCP; 1.0 μL L^{-1}) can alleviate the darkening effect.[5,6] Other studies demonstrate that treatment of the onion cv. Sherpa with 1-MCP (1.0 μL L^{-1}) before or after curing elicits a similar effect of suppressing sprout growth to using ethylene alone.[9,10]

In sweet potato, the use of ethylene to suppress sprouting also promotes root decay.[7,11] The potential effect of 1-MCP on sweet potato storage was investigated by Cheema *et al.*,[12] who found that a concentration of 625 nL L^{-1} applied for 24 h and storage at 25 °C inhibited sprout growth in sweet potato cvs Bushbuck and Ibees for at least 4 weeks. This offers a potential alternative approach to continuous exposure to exogenous ethylene. Thus the work reported herein investigated the effects of 1-MCP on sweet potato decay and physiology in relation to the temporal and spatial flux of major individual carbohydrates and phenolic compounds during storage.

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EXPERIMENTAL

Plant material

Experiments were conducted on two different consignments of sweet potato cv. Covington to elucidate the effect of 1-MCP during cold storage at 15 °C. Both old and freshly stored roots were tested. Roots for Experiment 1 were cultivated in the USA (Barnes Farming) and delivered through Barfoots of Botley Ltd (Bognor Regis, UK). After harvesting in November 2010, the roots were cured (30 °C, 95% relative humidity) and stored at 14 °C for ca. 7 months before being received (July 2011) at Cranfield University (CU), UK. Roots for the second experiment were sourced from Portugal, where they were planted in June 2011, harvested in October 2011 and also cured under similar conditions to those described earlier and according to standard commercial practice, and stored at 14 °C at Barfoots of Botley for 2 weeks before being delivered to CU for the trial.

Experimental design

The consignment for each experiment was initially divided into two lots; the lots were further divided into three replicates each and treated separately in water-sealed 264 L polypropylene boxes at 15 °C. In each case, one lot (in triplicate) was treated with 1-MCP (1.0 μL L^{-1}, 24 h) while the other (also in triplicate) served as the untreated control. Each replicate box contained two subsamples designated as destructive subsamples ($n = 15$ and 30 in Experiment 1 and Experiment 2, respectively) and non-destructive subsamples ($n = 10$ and 20 in the respective experiments). The non-destructively evaluated subsamples were distinguished by tagging individual roots and assessed periodically for weight loss, decay and sprouting. The destructive subsamples were further assessed for respiration rate, followed by biochemical assays (non-structural sugars and phenolics). Baseline samples (as received at the beginning of the experiment) were assessed before 1-MCP treatment and then at periodic intervals after the treatment.

1-MCP treatment

1-MCP was applied as described by Chope *et al.*,[13] with slight modifications. To release the concentration of 1.0 μL L^{-1} of 1-MCP gas, 1.47 g 1-MCP powder (SmartFresh 0.14%, Rohm & Haas, Philadelphia, PA, USA) was weighed into three 50 mL conical flasks and sealed with Nescofilm (Bando Chemical Ind. Ltd., Kobe, Japan). To each flask, 5 mL warm water (~50 °C) was injected through the Nescofilm using a needle and syringe, shaken to mix and quickly placed in the storage boxes for 24 h. An 8 × 8 cm electric fan (Nidec beta SL, Nidec, Kyoto, Japan) was installed in each box to circulate the released gas. Periodically, the headspace gas in the boxes were analysed by repeated withdrawal and injection into a gas chromatograph (GC model 8340, DP800 integrator, Carlos Erba Instruments, Hemel Hempstead, UK) equipped with a flame ionization detector (FID). The 1-MCP treatments achieved a mean concentration of 1.02 μL L^{-1} (data not shown). After 24 h, the boxes were opened and the roots transferred to air storage in a temperature-controlled room at 15 °C. Alongside the 1-MCP treatment, the control roots were kept in other boxes under identical conditions.

Non-destructive assessments

The non-destructively tested roots were individually numbered and repeatedly assessed for weight loss, cumulative decay and sprout growth as described by Amoah *et al.*[7] In Experiment 2, some control roots were also stored at 25 °C to check the effect of temperature on sprouting.

Measurement of respiration rate

Roots ($n = 9$ and 12, in Experiments 1 and 2, respectively) selected for biochemical assays were initially assessed for their respiration rates using a Sable Respirometry System (model 1.3.8 Pro, Sable Systems International, Las Vegas, NV, USA) as described by Amoah *et al.*[7]

Sample preparation for biochemical assays

After measuring respiration rates, roots were immediately washed in running tap water and dried with a soft paper towel. Each root was then cut into the proximal, middle and distal segments (each segment was approximately a third of the total root length). The segments were manually peeled with a sharp knife to obtain two types of tissue: skin (comprising the periderm, ~1.0–1.5 mm deep) and flesh (comprising the cortex and pith). The skin and flesh tissues were snap frozen in liquid nitrogen, each divided into two and stored at −40 °C and − 80 °C, respectively, until further analysis. Approximately 10 g of the frozen tissue (−40 °C) was weighed (fresh weight, FW) and freeze dried (Scan Vac, Västerås, Sweden) in the dark for 7 days. After lyophilization, the samples were reweighed to obtain the dry weight (DW). The dry matter content (%) was subsequently calculated on a fresh weight basis. The samples were then ground to powder and stored at −40 °C in readiness for individual non-structural carbohydrates and phenolics assays.

Extraction and quantification of non-structural carbohydrates and phenolic compounds

Individual soluble sugars and phenolic compounds were extracted and quantified according to the protocols described by Amoah *et al.*[7] Each phenolic compound in the samples of Experiment 1 was calibrated against chlorogenic acid and measured in terms of chlorogenic acid equivalence on a fresh weight basis. In Experiment 2, however, the individual phenolic compounds were calibrated with their respective pure standards.

Statistical analysis

All data were first subjected to Shapiro–Wilk normality test and plotted for residuals to verify the assumptions for the analysis of variance (ANOVA). The ANOVA tables were generated using GenStat for Windows, Version 14 (VSN International Ltd, Hemel Hempstead, UK) and used to identify statistically significant trends. The means between treatments were separated with least significant difference (LSD) at $P = 0.05$.

RESULTS

1-MCP had no effect on respiration rate and sprouting

Respiration in the older root stock (Experiment 1: measured 70 days after 1-MCP treatment) remained fairly constant at a mean value 3.87×10^{-6} g kg^{-1} s^{-1} (Fig. 1). On the other hand, initial respiration rate of the much fresher roots (Experiment 2) was higher than that of the older roots but it gradually declined from the mean value 8.90×10^{-6} g kg^{-1} s^{-1} at the beginning of storage to 3.67×10^{-6} g kg^{-1} s^{-1} after 130 days of storage. In both experiments, however, no effect of 1-MCP treatment was observed on

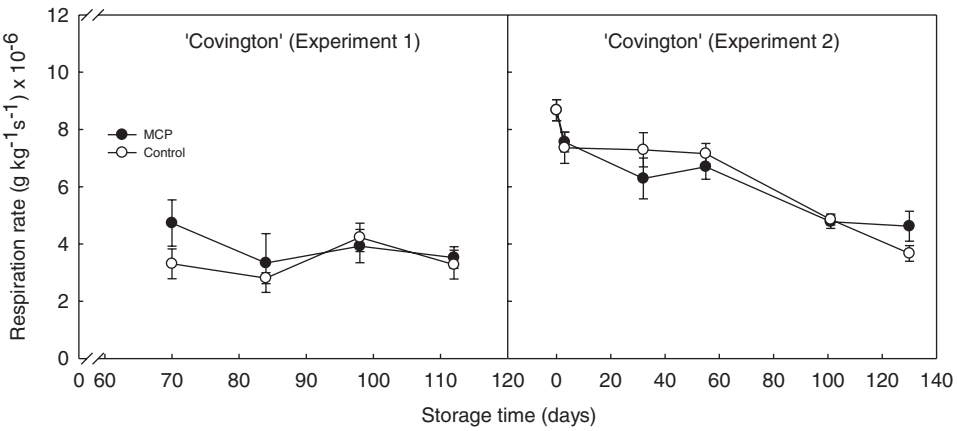

Figure 1. Respiration rate (g kg^{-1} s^{-1}) of sweet potato during storage with or without 1 µL L^{-1} 1-MCP. Respiration rate was measured in terms of the evolution of CO_2. Each data point is the mean of three replicated treatments consisting of three roots per replicate for Experiment 1 ($n = 9$) and four roots per replicate for Experiment 2, respectively. Roots in Experiments 1 and 2 were treated 255 and 22 days, respectively, after harvest. The bars represent ± standard error of the mean.

the respiration rate of the roots. Also, in both 1-MCP-treated and untreated roots, no incidence of sprout emergence was observed as long as roots remained in cold storage at 15 °C.

1-MCP reduced root decay

Root decay was significantly reduced by 1-MCP in both experiments. This was more profound in the older root stock in Experiment 1 where, at 84 days after treatment with 1-MCP, disease severity was reduced by half to about 23% compared to 47% in the untreated roots (Fig. 2). A similar pattern was observed in Experiment 2, although decay was less pronounced. In this case, decay was only marginally reduced by 1-MCP during the first 100 days after treatment. During subsequent storage, however, 1-MCP (5% decay at 112 days) significantly decreased root decay compared to the control (20% decay at 112 days). The earliest incidence of decay in Experiment 1 was 3 weeks after 1-MCP treatment, and decay was observed in both 1-MCP-treated and control roots. However, the fresher roots of Experiment 2 started decaying much later, at 7 weeks after 1-MCP treatment and subsequent storage. Generally, two distinct types of root decay were observed: tip (proximal) rot and end (distal) rot. Both types of rot initiated from the respective root ends and progressed through the middle to the opposite ends.

1-MCP reduced weight loss

Weight loss in 1-MCP treatment was reduced by ~1.8-fold in the old root stock (Experiment 1) and by ~1.3-fold in the fresher roots (Experiment 2; Fig. 3). Thus at 84 days the untreated roots in Experiment 1 had lost ~41% weight as against ~23% in the 1-MCP-treated roots. Likewise, in Experiment 2, the control roots lost weight at a much faster rate (~32% at 112 days) than the 1-MCP-treated roots (~21% at 112 days). The rate of root decay was correlated with the weight loss. It was noted that decay increased concomitantly with weight loss (Experiment 1: $R^2 = 0.90$; Experiment 2: $R^2 = 0.86$).

Effect of 1-MCP on dry matter

The effect of 1-MCP on dry matter content (percentage of fresh root weight) was not consistent between the two experiments (supplementary Fig. 1, supporting information). In the older root stock (Experiment 1), dry matter weight of 1-MCP-treated roots (211 g kg^{-1}) was not significantly different from that of the untreated roots (206 g kg^{-1}). A contrary effect was, however, observed in the fresh roots in Experiment 2 as dry matter was significantly lower in roots treated with 1-MCP (243 g kg^{-1}) than untreated roots (253 g kg^{-1}). Skin dry matter content (223 and 262 g kg^{-1} in Experiments 1 and 2, respectively) was significantly higher than the respective flesh dry weights (194 and 237 g kg^{-1} in Experiments 1 and 2, respectively). Roots in Experiment 1 were further assayed for the spatial gradient of dry matter across the root length. The mean dry matter content (for pooled skin and flesh tissues) at the proximal (210 g kg^{-1}), middle (206 g kg^{-1}) and distal (209 g kg^{-1}) root sections was not significantly different.

1-MCP did not affect non-structural carbohydrates

Individual non-structural carbohydrates were analysed separately in flesh and skin tissues across the proximal, middle and distal sections. During storage, all reducing sugars declined ($P < 0.001$) in both 1-MCP-treated and control roots whereas sucrose content remained relatively stable. In both experiments, no effect of 1-MCP was observed on the content of sugar across the spatial gradient of the roots (supplementary Figs 2 and 3, supporting information) except maltose in Experiment 1, which was more concentrated in the skin of roots treated with 1-MCP than the control ($P = 0.004$).

The mean fresh weight concentration of maltose, fructose, glucose and sucrose in the flesh tissues at the start of Experiment 1 were 2420, 5510, 8340 and 46 930 mg kg^{-1}, respectively. Their corresponding concentrations in the skin were 2770, 3080, 3210 and 38 050 mg kg^{-1}, respectively. Thus their corresponding values in the skin were lower except for maltose. During subsequent storage, however, maltose was found in an approximately similar concentration in the skin to the flesh tissues. The mean concentrations of sugars in the roots from Experiment 2 were significantly higher than those in Experiment 1. Initial fructose, glucose and sucrose content in the flesh tissues were 13 710, 15 420 and 40 660 mg kg^{-1}, whereas the corresponding values in the skin were 8110, 6850 and 41 860 mg kg^{-1}, respectively. However, maltose was not detected in Experiment 2, irrespective of treatment.

Effect of 1-MCP on phenolic compounds

Experiment 1 (old root stock of 'Covington').

Total phenolics content (measured as chlorogenic acid equivalence on fresh weight basis) in the skin (2290 mg kg^{-1}) was

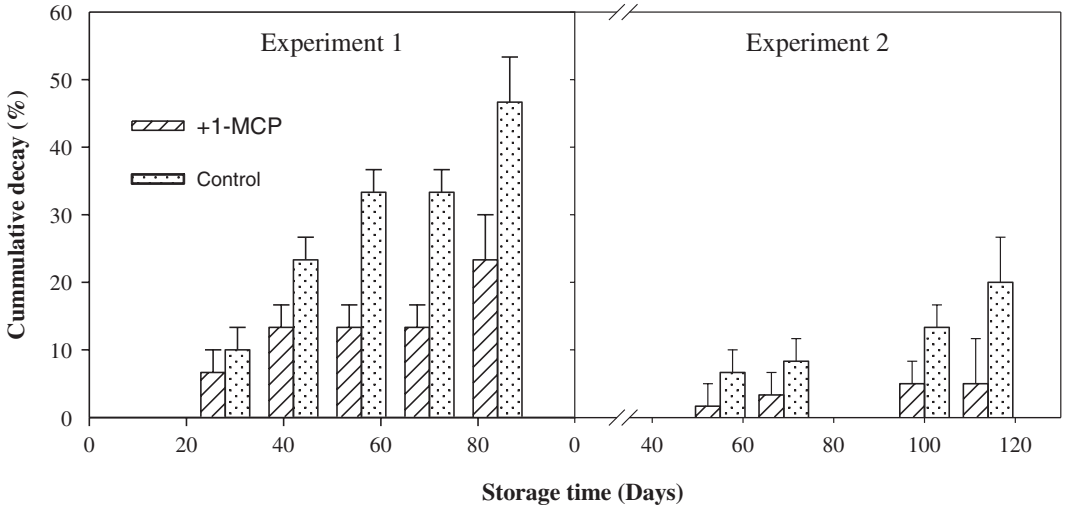

Figure 2. Decay of sweet potato cv. Covington during storage at 15 °C with or without 1.0 μL L^{-1} 1-MCP. Each data point is the mean of three replicated treatments containing 10 roots per replicate in Experiment 1 and 20 roots per replicate in Experiment 2, respectively. Roots in Experiments 1 and 2 were treated with 1-MCP 255 and 22 days, respectively, after harvest. The bars represent ± standard error of the mean.

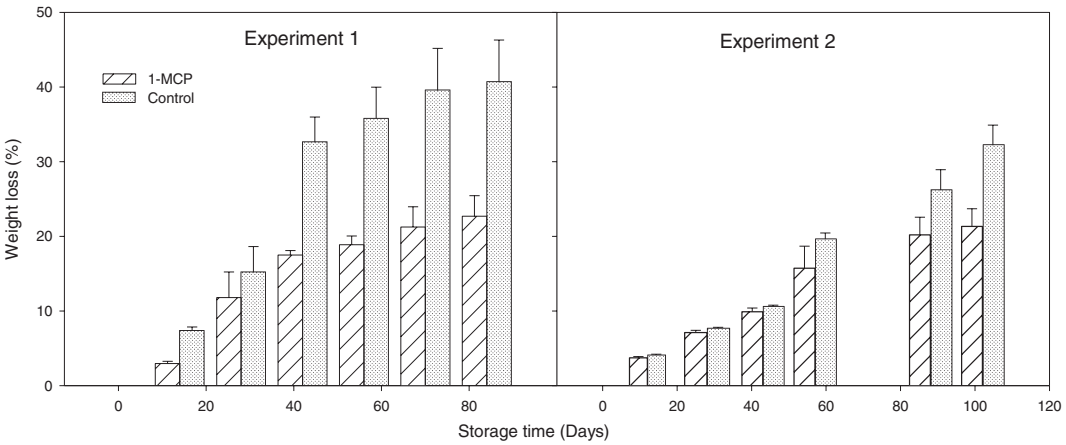

Figure 3. Weight loss of sweet potato cv. Covington during storage at 15 °C with or without 1.0 μL L^{-1} 1-MCP. Each data point is the mean of three replicated treatments containing 10 roots per replicate in Experiment 1 and 20 roots per replicate in Experiment 2, respectively. Roots in Experiments 1 and 2 were treated with 1-MCP 255 and 22 days, respectively, after harvest. The bars represent ± standard error of the mean.

~11-fold greater than the flesh content (200 mg kg^{-1}). Also, total phenolics concentration in the proximal section (2960 mg kg^{-1}) of the roots was greater ($P < 0.001$) than either the middle (1890 mg kg^{-1}) or distal sections (2030 mg kg^{-1}) (Fig. 4). The individual phenolic compounds were also not uniformly distributed across the spatial gradient (Fig. 5 and supplementary Fig. 4, supporting information). Isochlorogenic acid A (3,5-di-*O*-caffeoylquinic acid) was the most abundant phenolic compound in the periderm (960 mg kg^{-1}), followed by chlorogenic acid (5-*O*-caffeoylquinic acid; 450 mg kg^{-1}). Conversely, chlorogenic acid was the dominant phenolic in the flesh tissues (130 mg kg^{-1}), followed by isochlorogenic acid A (60 mg kg^{-1}) (Fig. 5 and supplementary Fig. 4). Ferulic and coumaric acids were only present in trace quantities. Chlorogenic acid and its isomers were more concentrated in the proximal section of the roots ($P < 0.05$) except for isochlorogenic acid C (4,5-dicaffeoylquinic acid), whose content in the flesh tissue did not vary across the root sections ($P = 0.315$). The skin content of caffeic acid was evenly distributed between the proximal and distal sections (560 mg kg^{-1}), with the middle section accumulating the lowest concentration. However, in the flesh tissues, caffeic acid was more abundant in the proximal than the middle and distal sections of the roots (Fig. 5).

1-MCP suppressed both total and individual phenolic contents in the tissues across the root sections, except the proximal skin tissues (Fig. 5). Thus, the inhibition of phenolics by 1-MCP was more profound in the middle and distal tissues, accentuating the proximal dominance of phenolics. However, the suppression of phenolic compounds by 1-MCP was transient as the effect diminished with time (Fig. 5 and Supplementary Fig. 5, supporting information).

Experiment 2 (fresh root stock of 'Covington').

As observed in Experiment 1, isochlorogenic acid A was the dominant phenolic in the skin tissues (1870 mg kg^{-1}), followed by chlorogenic acid (1010 mg kg^{-1}) (Fig. 6). Also, chlorogenic acid (130 mg kg^{-1}) was the most abundant phenolic compound in the flesh, followed by isochlorogenic acid A (60 mg kg^{-1}). Caffeic acid (200 mg kg^{-1}) and isochlorogenic acid C (190 mg kg^{-1}) were observed in the skin but not in the flesh tissues. There was ~15-fold greater concentration of total phenolics in the skin

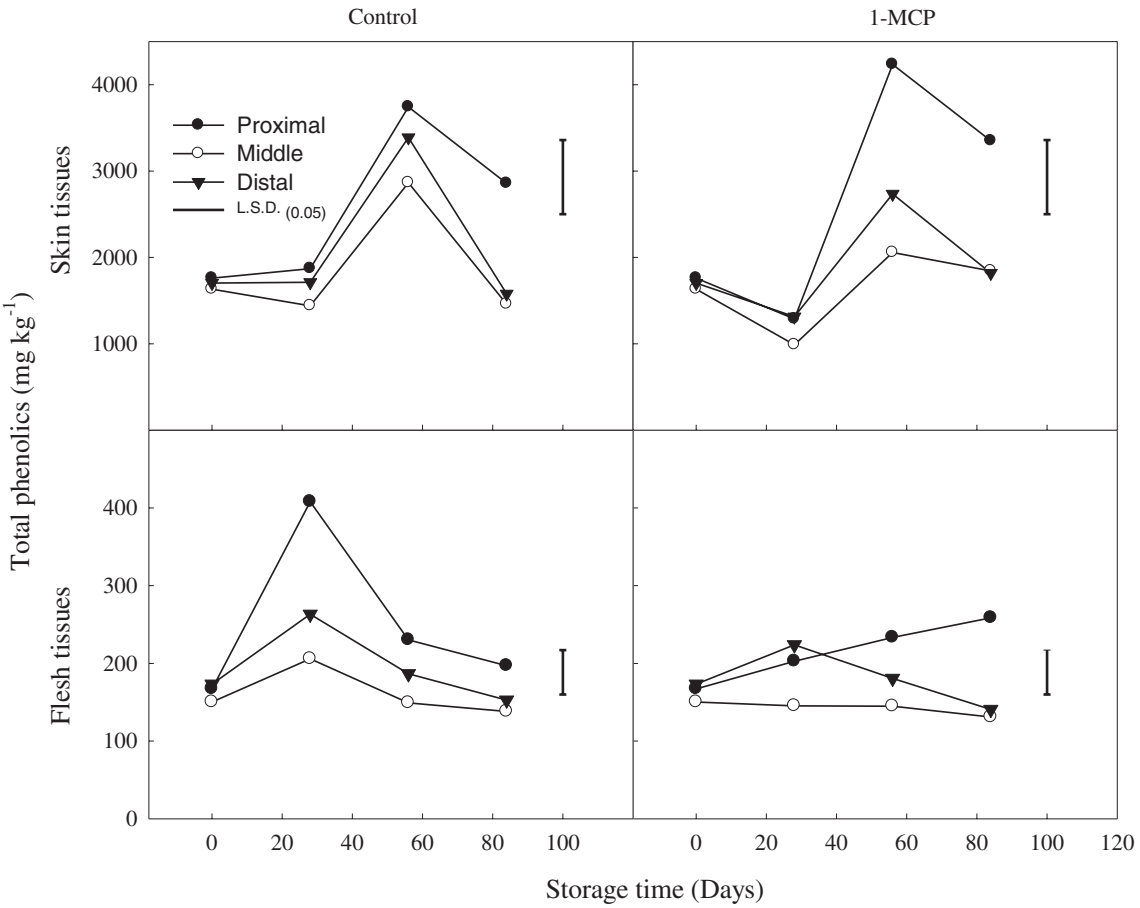

Figure 4. Spatial concentrations of total phenolics (chlorogenic acid equivalence on fresh weight basis) in sweet potato cv. Covington (Experiment 1) during storage at 15 °C with or without 1 μL L^{-1} 1-MCP. Each data point is the mean of three replicated treatments consisting of three roots per replicate ($n = 9$). The roots were treated 255 days after harvest. The bars represent LSD (0.05).

compared to the flesh tissues. All the individual phenolic compounds increased in abundance during the initial period (up to ca. 55 days) of storage and tended to decline thereafter. Contrary to the old 'Covington' roots in Experiment 1, no effect of 1-MCP was observed on the phenolic compounds in the fresh roots in Experiment 2.

DISCUSSION

Effect of 1-MCP on decay and weight loss

The economic losses in sweet potato postharvest chain are estimated to range between 35% and 95% in developing countries.[14] These losses are attributed mainly to decay and weight loss.[4] From studies on different sweet potato cultivars stored under East African marketing conditions, it was noted that weight loss was associated with physiological disorders,[15] and therefore the authors recommended that weight loss may be used to measure the postharvest loss of marketable value. A similar observation was made by van Oirschot.[4] This relationship was also confirmed in the current study where a striking correlation between sweet potato decay and weight loss was noted. The high rate of decay and weight loss in sweet potato are attributed to the high moisture content (50–81%) and water loss through the delicate skin, respectively. The high moisture content renders the roots vulnerable to a number of microbial infections. The rot symptoms showing on the roots in the current study implicated that *Rhizopus stolonifer* could possibly be the causal pathogen. Disease screens by Yencho *et al.*[2] showed that 'Covington' is typically susceptible to *Rhizopus* soft rot but resistant to *Fusarium* wilt and stem rots caused by *Fusarium oxysporum*. *Rhizopus stolonifer* causes a watery soft rot at the ends of injured roots, giving off fermentative odours.

Although ethylene inhibits sprout growth in sweet potato, there is growing evidence that it promotes both weight loss and decay,[7,11,16] and this may be through its interaction with the crop–pathogen system. Ethylene-induced decay characteristically initiates from the root tip.[7,11,16] 1-MCP effect on physicochemical changes in crops is normally evaluated in the context of its antagonistic response to ethylene. 1-MCP may therefore be expected to counteract ethylene-induced decay. In an unpublished report, it was found that the application of 1-MCP (1.0 μL L^{-1}) during the curing of sweet potato cvs Beauregard and Evangeline achieved 10% lower incidence of rot symptoms in the early periods (up to 133 days) of storage compared to the control (Villordon A, pers. comm.). However, the effectiveness against decay diminished during subsequent storage. In another study, Cheema *et al.*[12] found no diseases when 4-week-old sweet potato cvs. Bushbuck and Ibees were treated with 1-MCP (625 nL L^{-1}, 24 h) and stored at 25 °C for just 4 weeks. Thus the timing of 1-MCP application is important, being more effective in most crops soon after harvest.[17] The efficacy of 1-MCP against sweet potato decay is further corroborated by a separate report by Villordon[16] in which 'Beauregard' roots treated with 1-MCP (1.0 μL L^{-1}, 1 h) showed no signs of disease during 3 months' storage.

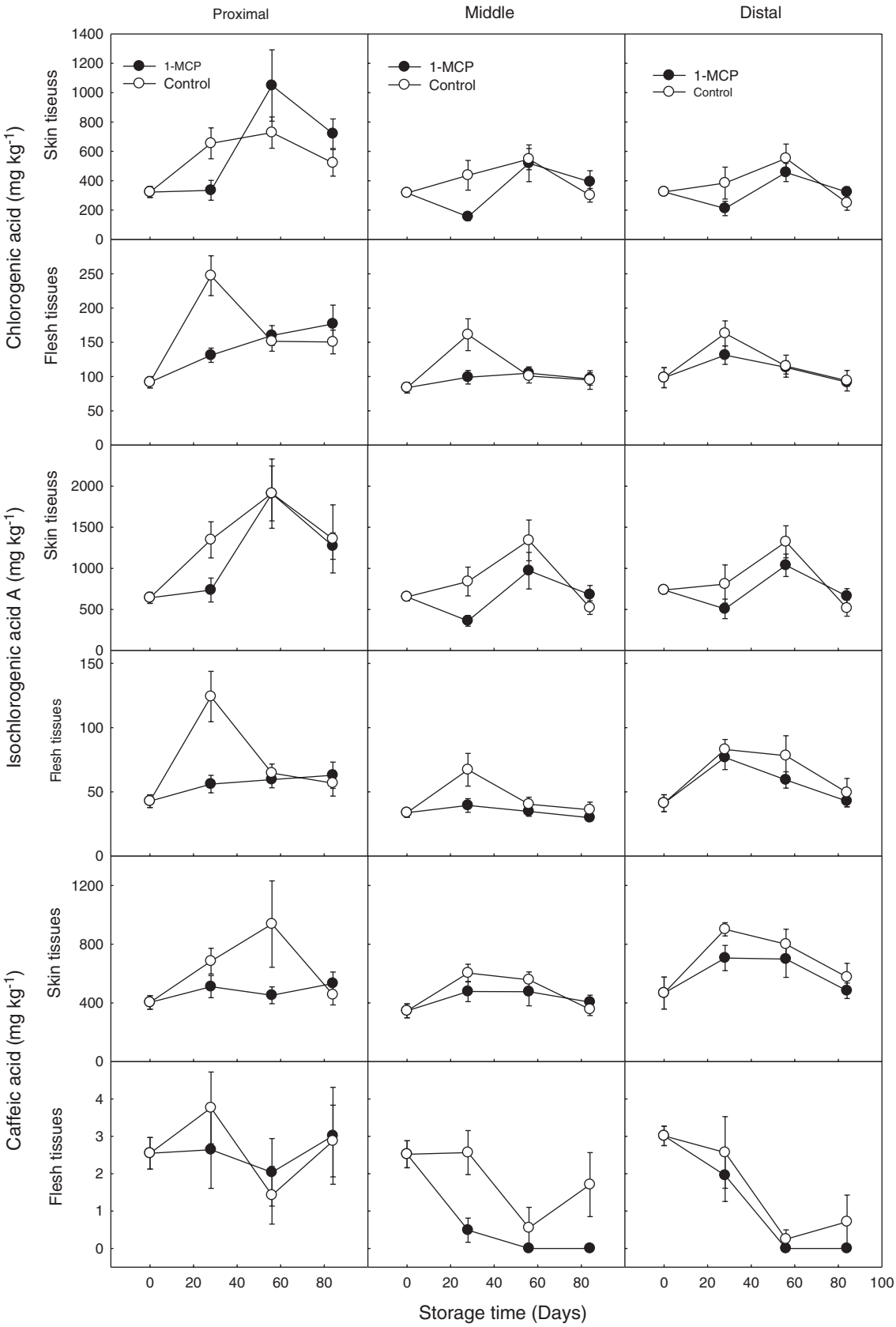

Figure 5. Spatial concentrations of chlorogenic acid, isochlorogenic acid A and caffeic acid in sweet potato cv. Covington (Experiment 1). All concentrations are expressed as chlorogenic acid equivalence on fresh weight basis. Each data point is the mean of three replicated treatments consisting of three roots per replicate ($n = 9$). The roots were treated 255 days after harvest. The bars represent ± standard error of the mean.

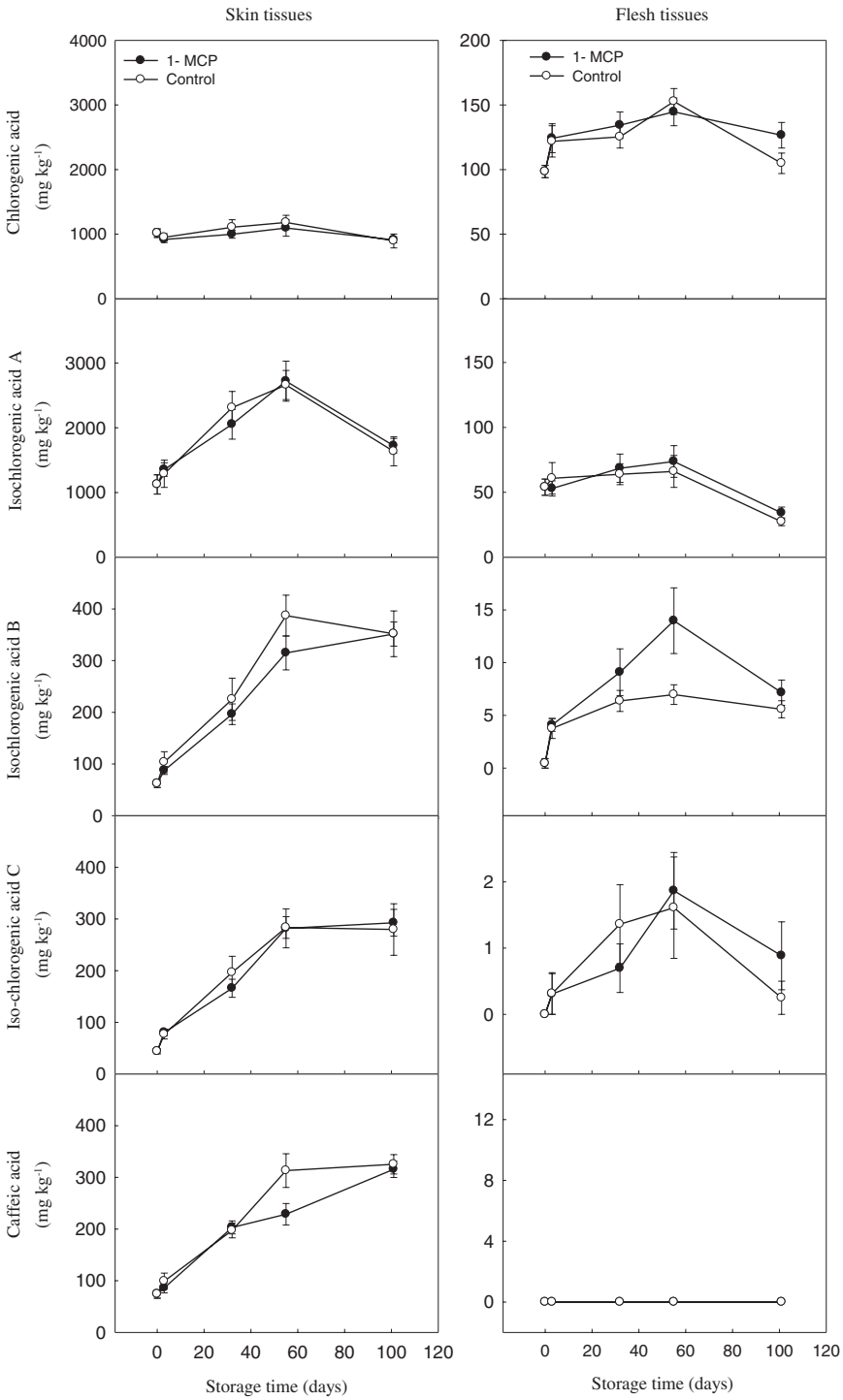

Figure 6. Spatial concentration (fresh weight basis) of phenolic compounds in sweet potato cv. Covington during storage at 15 °C with or without 1 μL L^{-1} 1-MCP (Experiment 2). Each data point is the mean of three replicated treatments consisting of four roots per treatment ($n = 12$). The roots were treated 22 days after harvest. The bars represent ± standard error of the mean.

Effect of 1-MCP on sweet potato respiration rate and sprout growth

Ethylene boosted respiration rate in sweet potato cv. Covington by about 1.5- to 2.0-fold,[7] as was similarly the case in potato[6] and onion.[9] Therefore, the inhibition of ethylene perception by 1-MCP may be expected also to produce the opposite respiratory effect. For instance, 1-MCP suppressed respiration rate in potato.[18] 1-MCP also marginally reduced respiration in the sweet potato cvs. Bushbuck and Ibees compared to untreated roots.[12] The expected decline in respiration rate by 1-MCP treatment was, however, not evident in the current study.

1-MCP inhibited sprout growth in onions stored at 4 or 12 °C but not at 20 °C,[19] suggesting temperature dependence of 1-MCP effectiveness against sprouting. Thus the low-temperature storage used in this study may explain the low respiration rate as well as the inability of sprouts to grow. Low temperature may therefore

be sufficiently inhibitory to sprout growth in sweet potato, and the 1-MCP effect on sprout development may be better evaluated at higher storage temperatures.

Effect of 1-MCP on non-structural carbohydrates in sweet potato

1-MCP inhibits ethylene-induced sugar accumulation in potatoes.[5,6,8] In contrast to potato, however, ethylene reduces the concentration of monosaccharides in sweet potato.[7] 1-MCP would therefore be expected to elicit the opposite effect on sugars in sweet potato. The lack of effect on the sugars in any of the 'Covington' roots in the present study also confirms limited interference with natural metabolism at the low storage temperature of 15 °C.

Initial concentration of the reducing sugars and sucrose compare very well with the respective values obtained by Yencho *et al.*,[2] except for maltose, which was not detected. Maltose is scarcely present in raw sweet potatoes. The presence of maltose mainly results from starch hydrolysis, especially during cooking. Thus the detection of maltose in the roots of cv. Covington in Experiment 1 may be attributed to the breakdown of starch during long-term storage. During storage, all the monosaccharides declined ($P < 0.001$) in both tissues of the cv. Covington, whereas sucrose content remained relatively stable. Such decline in sugar levels in sweet potato has been linked with harvest age. Four sweet potato cultivars harvested at 5 months' maturity lost total soluble solids by up to 53.4% (depending on cultivar) during storage, while there were increases in samples harvested earlier, at 3 or 4 months' maturity.[20] Thus the late harvest of the cv. Covington in the present study may explain the declining trend in sugars during storage. The consistent decline in the content of maltose alongside fructose and glucose in cv. Covington also suggests restricted starch hydrolysis.

Effect of 1-MCP on phenolics in sweet potato

Some phenolic compounds are known to have an antifungal role and therefore the effectiveness of 1-MCP against decay symptoms may also be through its interaction with phenolics in sweet potato. 1-MCP has been shown to inhibit phenolics biosynthesis in many crops,[21] as opposed to ethylene-induced accumulation of phenolics. No published work is available in the literature on how 1-MCP mediates the biosynthesis of phenolics in the tissues of sweet potato. As observed in Experiment 1 of the current study, the suppression of phenolics by 1-MCP is consistent with observations in other crops. Significant suppression of phenolics by 1-MCP, however, occurred only in the early days of storage and was short lived. This may explain the earlier observations by Cheema *et al.*,[12] Harper[15] and Villordon (pers. comm.) and suggests the need for repeated application of 1-MCP during storage for full effectiveness. Phenolics content in the middle and distal sections of the roots were the most suppressed, leaving a net higher balance of individual phenolic compounds in the proximal tissues. It may therefore be speculated that the promoted concentration of phenolics in the proximal tissues by 1-MCP provides a mechanistic context to explain its suppression of ethylene-induced tip decay symptoms.

There is a marked similarity in the phenolics distribution pattern in cvs. Covington and Beauregard, since Padda[22] also observed that chlorogenic acid dominated in the flesh (cortex and pith) tissues of the sweet potato cv. Beauregard, followed by isochlorogenic acid A. That these major phenolic compounds were highly concentrated in the proximal root sections suggests that they are likely related to natural disease resistance.

CONCLUSION

At 15 °C, 1-MCP had no effect on respiration in sweet potato cv. Covington. However, there was significant improvement in the longevity of the stored roots. 1-MCP treatment may have an anti-decay effect and reduced weight loss. Therefore, storage trials which involve the use of continuous ethylene supplementation to inhibit sprout growth may be combined with 1-MCP to alleviate ethylene-induced weight loss and decay. Furthermore, the study reveals that 1-MCP suppresses phenolic compounds but that the effect is brief. The distribution of phenolic compounds and dry matter in sweet potato is skewed towards the proximal segments of the roots. The dominance of phenolics, especially the antifungal compounds chlorogenic acid and its isomers in the proximal sections, may perform a role in both defence against diseases and the commonly observed proximal sprouting in sweet potato.

ACKNOWLEDGEMENTS

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SUPPORTING INFORMATION

Supporting information may be found in the online version of this article.

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