ASPECTS OF AVOCADO FRUIT GROWTH AND DEVELOPMENT: TOWARDS UNDERSTANDING THE 'HASS' SMALL FRUIT SYNDROME

Clive Scott Moore-Gordon

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ABSTRACT

Persea Americana Mill. cv. Hass is predisposed towards producing a high proportion of undersized fruit. Reasons for phenotypically small 'Hass' fruit are obscure, but it does appear to be aggravated by adverse growing conditions. A detailed study of the metabolic control of avocado fruit growth was carried out to determine the underlying physiological reasons for the appearance of the 'Hass' small fruit phenotype. Furthermore, the application of mulch was evaluated as a possible management strategy to increase 'Hass' fruit size.

Anatomical and morphological comparisons were made between normal and small 'Hass' fruit in an attempt to characterise the 'Hass' small fruit phenotype. Small fruit always contained a degenerate seed coat and fruit size was closely correlated with seed size. Kinetic analysis of changes in cell number and size during fruit development revealed that growth was limited by cell number in phenotypically small fruit. Analysis of endogenous isopentenyladenine (IP) and abscisic acid (ABA) revealed that ABA concentration was negatively correlated with size of similarly aged fruit. Calculation of the IP: ABA ratio showed a linear relationship with increasing fruit size. Qualitative and quantitative differences in mesocarp sterol composition were observed between normal and phenotypically small fruit.

Both the normal and small-fruit phenotypes were used to probe the interaction between end-products of isoprenoid biosynthesis and activity of mesocarp 3-hydroxy-3-methylglutaryl coenzyme A reductase (HMGR) in the metabolic control of avocado fruit growth. In phenotypically small fruit, a 70% reduction in microsomal HMGR activity was associated with a substantial rise in mesocarp ABA concentration at all stages of development. Application of mevastatin, a competitive inhibitor of HMGR, via the pedicel reduced growth of phenotypically normal fruit and increased mesocarp ABA concentration. These effects were reversed by co-treatment of fruit with either mevalonate, IP or the synthetic cytokinin (CK) analogue, N-(2-chloro-4-pyridyl)-N-phenylurea, but were unaffected by gibberellic acid. Likewise, in vivo application of ABA reduced fruit growth and HMGR activity, and accelerated abscission at all stages of development, effects that were reversed by co-treatment with IP. In contrast, the effect of sterols on mevastatin-induced inhibition of fruit growth was temporally different. Application of either stigmasterol or cholesterol during phase I caused a decline in

growth, accelerated fruit abscission and exacerbated the effects of mevastatin whereas during phase II and III, stigmasterol reversed inhibition of fruit growth. Stigmasterol did not however, reverse the inhibitory effect of mevastatin on HMGR activity presumably as a result of mevastatin-induced increased endogenous ABA. It was therefore concluded that ABA accumulation downregulates mesocarp HMGR activity and that *in situ* CK biosynthesis modulates the effect of ABA during phase I of fruit growth whereas, both CK and sterols perform this function during the later stages to sustain the developmental programme.

The effect of an altered CK: ABA ratio on solute allocation, cell-to-cell communication and plasmodesmatal structure was investigated in 'Hass' avocado fruits to determine the relationship between a change in hormone balance and expression of phenotypically small fruit. Exogenous application of ABA induced early seed coat senescence and retarded fruit growth, and these effects were negated in fruit co-injected with ABA and IP. The underlying physiological mechanisms associated with ABA-induced retardation of 'Hass' avocado fruit growth included: diminution of mesocarp and seed coat plasmodesmatal branching; gating of mesocarp and seed coat plasmodesmata by deposition of apparently proteinaceous material in the neck region; abolishment of the electrochemical gradient between mesocarp and seed coat parenchyma; and arrest of cell-to-cell chemical communication. In addition, solute allocation in ABA-treated fruit resembled closely that of phenotypically small fruit confirming that elevated ABA concentration had contributed to the decline in postphloem symplastic continuity.

In a field trial in the KwaZulu-Natal midlands, root growth was substantially increased throughout three seasons by the application of coarse composted pine bark mulch. Mulching resulted in a significant 6.6% increase in mean fruit mass, in spite of 14.7% more fruits per tree. The combined effect was a 22.6% increase in overall yield. Differences in productivity between treatments closely correlated to levels of bark carbohydrate reserves. Data collated during this study to suggest that mulching at least partly ameliorated tree stress included: a reduction in the incidence of premature seed coat senescence and pedicel ring-neck, both of which are considered to be advanced symptoms of the stress syndrome; a lowering of mean foliage temperatures; and a reduction in the degree of photoinhibition during the heat of the day.