The effect of isoprenoid growth regulators on avocado (Persea americana Mill. cv Hass) fruit growth and mesocarp 3-hydroxy-3-methylglutaryl coenzyme A reductase (HMGR) activity was investigated during the course of fruit ontogeny. Both normal and small-fruit phenotypes were used to probe the interaction between the end products of isoprenoid biosynthesis and the activity of HMGR in the metabolic control of avocado fruit growth. Kinetic analysis of the changes in both cell number and size revealed that growth was limited by cell number in phenotypically small fruit. In small fruit a 70% reduction in microsomal HMGR activity was associated with an increased mesocarp abscisic acid (ABA) concentration. Application of mevastatin, a competitive inhibitor of HMGR, reduced the growth of normal fruit and increased mesocarp ABA concentration. These effects were reversed by co-treatment of fruit with mevalonic acid lactone, isopentenyladenine, or N-(2-chloro-4-pyridyl)-N-phenylurea, but were not significantly affected by either gibberellic acid or stigmasterol. However, stigmasterol appeared to partially restore fruit growth when co-injected with mevastatin in either phase II or III of fruit growth. In vivo application of ABA reduced fruit growth and mesocarp HMGR activity and accelerated fruit abscission, effects that were reversed by co-treatment with isopentenyladenine. Together, these observations indicate that ABA accumulation down-regulates mesocarp HMGR activity and fruit growth, and that in situ cytokinin biosynthesis modulates these effects during phase I of fruit ontogeny, whereas both cytokinins and sterols seem to perform this function during the later phases.