Blossom-end rot (BER) in pepper fruits is known as a “physiological” disease which is evoked by climatic and osmotic stresses (e.g., high temperatures and salinity) and by deficit irrigation management. The physiological and biochemical mechanisms involved in the stimulation of BER are still a matter of dispute. It has been proposed that BER occurs under cultivation conditions that accelerate fruit growth beyond calcium supply to the growing tissue. In the present study, we investigated this hypothesis by recording fruit growth and sugar metabolism under normal and BER-inducing conditions. The results showed that pepper fruits grown in a ventilated-cooled greenhouse, which ameliorated BER, had a higher initial growth rate than those grown in a non-cooled greenhouse, but had lower calcium concentrations in the fruit pericarp. Fruits that grew without ventilation-cooling had higher concentrations of apoplastic and symplastic sucrose and reducing sugars, and higher levels of symplastic starch than those that developed in a cooled greenhouse. Concomitantly, fruits that developed in a non-cooled greenhouse had lower acid invertase, sucrose synthase and fructokinase activities than those that developed in a cooled greenhouse. Accordingly, the nonnecrotic part of BER-affected fruits had higher concentrations of symplastic starch, reducing sugars and sucrose than healthy fruits. These findings indicate that the occurrence of BER in pepper fruits was associated with impaired sucrose metabolism in fruit tissues, which led to reductions in growth rate and final size. Thus, the present findings negate the notion that BER occurs when fruit growth rate is stimulated, and favour the idea that direct impairment of fruit sugar metabolism and of normal fruit growth are involved.