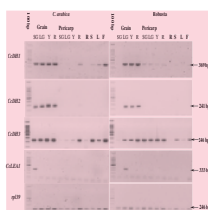


Male function fails when the heat is on

The consequences of global warming for yields of individual crop species are difficult to predict. Moderate increases in temperature affect different plant processes in different ways; further, those responses will differ between species. Thus, studies of individual species, such as the work on tomato (*Lycopersicon esculentum*) by **Sato *et al.*, Chiba and Osaka, Japan (pp. 731–738)** are important in our understanding of the effects of climate change. The authors grew plants under control conditions (28/22 °C) or at moderately increased temperatures (32/26 °C), the increase representing what is

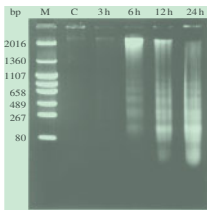
expected, according to some models, by about 2075. The first key finding was that general features of growth, including the number of flowers produced, were not affected by the temperature increase. However, the number of fruit set at the higher temperature was only 25 % of that under control conditions. This reduction was entirely ascribable to a failure of male function. Stamens were shorter than in control plants and although they produced normal amounts of pollen, the number of pollen grains released was very markedly reduced while pollen viability dropped from approx. 85 % to approx. 20 %. There were also marked changes in anther biochemistry. At the higher temperatures, sucrose accumulated at the expense of reducing sugars; this being correlated with a reduction in the amount of mRNA encoding acid invertase (although this may not be the sole enzyme regulating sucrose hydrolysis). There was also an increased accumulation of proline at the meiosis stage of pollen development. The authors ascribe pollen failure to poor transport of proline from the tapetum to the developing pollen, a suggestion supported by the decrease in the amount of mRNA encoding proline transporters. Indeed, the authors focus on the idea that a delay in tapetal breakdown, and therefore a delay in transferring nutrients to the developing pollen, may hold the key to male dysfunction at the warmer temperatures.



Genes and beans in search of coffee quality

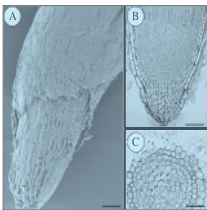
As I write this, I pause from time to time to take another sip from my cup of fair-traded Colombian coffee. But how much do we know about the biochemical features of the coffee bean that affect their taste or storage capacity? The answer, according to **Hinniger *et al.* (Nestlé Research Centre, Lyon, France and Cornell University, USA, pp. 755–765)** is 'not enough'. As a result, they have undertaken a major study of the regulation of genes involved in laying down some of the major components of the developing grain. In the current paper they focus on dehydrin proteins that are expressed late in

seed development. Dehydrins are a subgroup of the late embryogenesis abundant (LEA) proteins and may also be expressed elsewhere in the plant in response to water deficit. The authors have used information in the *Coffea canephora* EST (expressed sequence tag) database to clone cDNAs encoding three dehydrins (*CcDH1*, *CcDH2*, *CcDH3*) and another LEA protein (*CcLEA1*). Studies of the expression of the genes in *C. canephora* and *C. arabica* grains revealed slight but consistent differences between the species, but in general it can be stated that all three dehydrin genes are expressed throughout grain development and that *CcDH1* and *CcDH3* are also expressed in the pericarp and in other organs, including leaves and flowers. Analysis of the promoter of *CcDH2* (which showed a grain-specific expression pattern) revealed ABA-response elements, dehydration-response elements and sequences related to expression during seed development. Focusing on *CcLEA1*, its expression in both species was confined to a very narrow 'window' early in grain development during the phase of perisperm shrinkage and endosperm expansion, suggesting a role for this LEA in that transition. So, while these data do not specifically answer the questions raised above, they do contribute significantly to our understanding of the developmental biology of the coffee bean.



Death by drowning

In plant roots, waterlogging restricts aeration. This, in turn, affects a whole range of metabolic processes that rely on aerobic metabolism. Responses to flooding vary from species to species and according to the growth conditions and the age of the plant, as described for pea (*Pisum sativum*) by **Dan Gladish and colleagues (Oxford, Ohio, USA and Tokyo, Japan, pp. 895–902)**. Previous work by this group had shown that in plants grown at 25 °C, both 4-day-old and 5-day-old seedlings responded to flooding by forming vascular aerenchyma, a process requiring programmed cell death. However, in the 5-day-old seedlings, the aerenchyma was not extensive enough to supply the root tip with air (it did not extend far enough along the root) and the roots quickly stopped growing. Cessation of growth was associated with morphological aberrations such as tip curling, and by death of cells in the ground meristem and in the procambium. Cytological and biochemical studies of the dying cells reveal that death is an ordered process that occurs in individual cells, spreading out from the protoxylem poles. Nuclei become lobed and invaginated, and the chromatin condenses. ‘End-labelling’ techniques (‘TUNEL’) reveal extensive DNA breakage in these cells, confirmed by the observation of ‘ladders’ when extracted DNA is fractionated in agarose gels. These features are reminiscent of apoptosis in animal cells, but whether one calls this apoptosis or not may be a matter of semantics. What is clear is that this is a programmed cell death, rather than a necrotic response. Finally, it may seem rather a drastic response to kill the root tip in reaction to flooding, but the authors suggest that this leads to a diversion of resources away from the tip region and to the promotion of the growth of lateral roots from zones in the primary root that are less affected by flooding.



RAMs open the way to living borders

One of the pleasures of writing these commentaries is the discovery of topics about which I have little previous knowledge. This month I have learned about border cells, studied for several years by Martha Hawes at the University of Arizona, Tucson. Border cells are released from the outer layer of the root cap by hydrolysis of the connections with the next cell layer. The border cells remain alive in the rhizosphere where they release signalling molecules that may alter gene expression in potential pathogens and symbionts; they are thus free-living somatic plant cells. However, it is not a universal phenomenon. Amongst the dicots, some families produce no (or very few) border cells while others produce many. In plants that do not produce border cells, the outer cells of the root cap undergo programmed cell death and are sloughed off in sheets. So, what regulates the production of border cells? To start to answer the question, **Hawes has collaborated with colleagues at the University of California, Davis (Hamamoto *et al.*, pp. 917–923)**. They have found an unexpected correlation between border cells and root apical meristem (RAM) organization. Species that produce few or no border cells have closed RAMs in which it is possible to trace specific cell files back to specific initials in the RAM. Species that produce significant numbers of border cells possess open RAMs in which differentiated cell types cannot be traced back to specific initials. Why should this correlation exist? Is there something about closed meristems that also directs the programmed cell death of the outer cell layer of the root cap or is there something about open meristems that activates the expression of the genes regulating release of these cells as border cells? Do the differences reflect evolutionary history relating to how different types of plant interact with micro-organisms? This story will surely run and run.

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