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EDITORIAL

Green signals for life and death

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Multicellularity allows to assign different functions to individual cells which is advantageous for the entire population of cells forming an organism. For the individual cell, differentiation represents a risky investment, because it implies that specific tasks have to be upregulated at the cost of downregulating others (Lintilhac 1999). The downregulated functions have to be compensated by corresponding 'hypercellular' output from neighbouring cells. This culminates in a situation where the individual cells cannot survive outside the organismal context. The ultimate state of cellular 'altruism' is reached when individual cells sacrifice themselves for the sake of the whole. This programmed cell death has probably evolved in colony-forming single cell organisms such as bacteria, yeast or slime molds to effectively survive starvation. Nutrients released from a portion of 'altruistic' cells undergoing programmed cell death were redistributed to ensure survival of the remaining cells that would be the founder inoculum ensuring the survival of the colony. Later, programmed cell death was integrated as essential element into developmental differentiation. Already the earliest stages of multicellularity involved a separation between the (immortal) germ line and the (differentiating and mortal) soma in a programmed 'genetic suicide' of somatic cells (Weismann 1892). Thus, death seems to be an innate element of life, and it is clear that this transition has to be controlled tightly during development. Several contributions in the current issue deal with the plant-derived signals that control this transition either in the plant itself or in a medical context.

The differentiation of lignified vasculature from parenchymatic tissues is central for architectural integration of all higher land plants and represents a classical example for programmed cell death in plants. The work by Mulisch et al. (2013) in the current issue investigates the role of cysteine proteases in this process. The CP1 protease from white clover is processed from an inactive proform in leaves and petioles and localises to the xylem. Prior to vacuolar burst in protoxylem cells, this protein can be found at cisternae of the ER and, upon tonoplast disruption seems to degrade cellular material in the cytoplasm. Thus, it predicts ensuing cell death, which might indicate that it is not only a part of execution machinery, but also might play a role for the initiation of programmed cell death.

The plant endosperm is a triploid storage tissue that has the task to nourish the developing embryo by self-sacrifice. The endosperm has to be filled with assimilates, a function that is achieved by so-called transfer cells that form already early after pollination. In adaptation to this function, the transfer cells form cell wall protrusions, increasing the surface through which transport can occur and have been described by the classical work of Gunning and Pate (1969) in this journal. After they have completed their job, the transfer cells die, along with most of the endosperm that frees the stage for the developing embryo. The work by Monjardino et al. (2013) in the current issue extends now a series of fine structural studies that have discovered so-called flange ingrowths (Wardini et al. 2007; Talbot et al. 2002) with respect to the cellular mechanisms using maize as experimental system. They show for the first time that in addition to flange ingrowths, some of the transfer cells at the base of the endosperm show reticulate ingrowths. This difference is evident as early as 5 days after pollination, indicating that the signals that drive differentiation and subsequent cell death must act earlier in development than thought so far.

Plant signals that control cell death are also of relevance for medical applications. This is demonstrated by the work of Xie et al. (2013). They work on plant compounds that support the transplantation of mesenchymal stem cells into heart muscles to treat patients with cardiovascular degeneration diseases. They search for alternatives to chemotherapy with its often harsh side effects and recur to the rich phytotherapy resources developed in Traditional Chinese Medicine. Tanshinone IIA,

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the active compound from *Salvia miltiorrhiza* Bunge has been found to quell apoptotic cell death of cardiomyocytes subjected to oxidative stress. In the current work, they analyse the underlying cellular mechanisms and find that this compound stimulates expression of the chemokine receptor CXCR4, which stimulated cellular migration towards sources of the CXCR4 ligand, the stromal cell-derived factor 1α. This allowed mesenchymal stem cells injected into rats with acute myocardial infarction to locate the heart muscle in a more efficient manner and thus to better evade apoptosis.

Conflict of interest The author declares that there is no conflict of interest.

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