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EDITORIAL

# Stress, ROS, and actin—a volatile *menage à trois*?

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Life is not easy—this is especially true for plant cells that cannot run away, when they do not like their environment. The impressive capability of plant cells to cope with stress factors is a central element of their survival even under adverse conditions. In many organisms, stress responses involve the production of reactive oxygen species. These occur, for instance, when mitochondrial function is impaired such that the mitochondrial transmembrane potential decreases. Reactive oxygen species (ROS) have been found to be a central component of stress-induced programmed cell death in a couple of organisms, such as animals, yeast, and plants. The production of ROS, and the initiation of programmed cell death is under control of actin in many organisms (for review see Franklin-Tong and Gourlay 2008). Plants produce high levels of ROS as part of their normal metabolism, for instance to trigger cross-linking of cell wall, i.e., outside of a stress context. It is not surprising that, here, the link between stress responses, ROS, and actin has remained a bit elusive. Two contributions of the present issue might help to get insight into this triangle.

One reason for the controversial debate on the actual role of ROS in the stress response has been the difficulty to localize them at the subcellular level. The work by Darehshouri and Lütz-Meindl deals with the ultrastructural detection of hydrogen peroxide in the model alga *Micrasterias*. They administer osmotic and salt stress and show that by the fluorescent dye H2DCFDA that ROS production is strongly induced by salt stress. In the next step, they use $CeCl_3$ that forms a precipitate with peroxide and identify Cerium in the labelled structures by TEM-coupled electron energy loss spectroscopy. They demonstrate that chloroplast are the strongest source of peroxide production, followed by mitochondria and cytoplasm, whereas the cell walls do not play a role.

The work by Malerba et al. investigates the cellular response to heat stress (a topic, whose relevance is emphasized by the failure of the Copenhagen conference...) in the tobacco BY-2 system. They focus on the actin cytoskeleton and the endoplasmic reticulum and show disintegration of actin microfilaments and a reorganization of the endoplasmic reticulum in response to heat shock. This response is accompanied by accumulation of the HSP70 Binding Protein that can be suppressed cobalt, an inhibitor of ethylene synthesis. Actin filaments have been shown to be an evolutionary conserved central element of apoptosis and programmed cell death. The molecular players transmitting the actin signal towards the apoptotic machinery are far from being understood and seem to differ partially between the kingdoms. However, a direct interaction of actin filaments with membrane topology has been demonstrated in both directions. Actin reorganization has been observed during developmentally induced cell death such as somatic embryogenesis or self-incompatibility, and in a previous paper, the authors could show that actin drugs can manipulate the extent of fusicoccin-induced cell death in a cell culture system. The present work confirms, but also extends the previous findings by showing that actin plays also a role in the response to a physical signal, and by the involvement of the stress hormone ethylene.

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In both contributions, the triggering signal is a physical stimulus, which means that it has to be translated into a biochemical cascade. Salt and osmotic stress presumably act by mechanically challenging the plasmamembrane, heat stress could be sensed through changes in membrane fluidity. How are these still physical changes transduced into the cell interior? Actin is intimately linked with the membrane and might act as such a transducer. Interestingly, chloroplasts and mitochrondria that seem to be the major source of ROS-production travel along actin track and therefore might sense when these tracks are disorganized or even disassembled.

## Reference

Franklin-Tong VE, Gourlay CW (2008) A role for actin in regulating apoptosis/programmed cell death: evidence spanning yeast, plants and animals. Biochem J 413:389–404