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EDITORIAL

Methusalem's mystery

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Life is limited—at least for multicellular animals. The separation between differentiating somatic cells and the germ line also delineates the separation between mortality and eternal life. We are so tantalised by induced pluricellular stem cells, because they promise the option to cross this line. However, reprogramming of developmental potential is only part of the story; life leaves traces in a cell that exceed chromatin remodelling. These traces challenge cellular homeostasis, and with time, the cell will yield, a process whose manifestation in multicellular animals is familiar to all of us as ageing. It should be clear that ageing has to be separated from developmental potency; even plants, where induced pluripotency has been developed and biotechnologically exploited already for decades, undergo ageing. Is ageing more than the mere accumulation of life-related damage, or is it rather a predefined programme? A popular theory (Harman 1956) deduces ageing from oxidative damage by radicals, especially reactive oxygen species. This sounds like a fairly general mechanism—is it thus possible to arrive at a universal theory for ageing?

If the oxidative damage model is right, the mystery of human ageing should be associated with mitochondria. In his comprehensive review, Bereiter-Hahn (2014) provides a synopsis of the current knowledge of the link between oxidative stress resulting from impaired mitochondrial activity and human age-related diseases such as cerebral dysfunctions, cardiovascular degenerations and cancer. He draws a conceptual link between mitochondrial dynamics including biogenesis, degradation, fusion and fission with healthy ageing and concludes that the underlying processes must share fundamental signatures conserved over the eukaryotes.

If this holds true, the reduction of oxidative disbalance should increase longevity also in other life forms. This is not trivial at all; at least in plants, oxidative disbalance plays a scintillating role; it is not only the manifestation of damage, and it can as well be used as a signal to sense stress factors that, otherwise, would cause oxidative damage and thus can play a role that is positive, because it triggers adaptation to adverse conditions. Moreover, compared to animal cells, the challenge of ageing is even increased, since the light reaction of photosynthesis adds a further source of reactive oxidative species. In addition, a high level of apoplastic superoxide anions is maintained by a membrane-located NADPH oxidase that plays an important role in regulating oxidative cross-linking in the cell wall. However, these oxidative species can enter the cytoplasm, probably through aquaporins and thus impose further challenges to the redox balance of plant cells. The work by Yang et al. (2014) in the current issue dissects the role of reactive oxygen species in the Desert Cherry (*Nitraria tangutorum*), a plant that can cope with the extremely harsh conditions of the Gobi Desert such as salinity, drought and dust and therefore plays an important role as a pioneer plant to halt desertification. The lifestyle of this plant is obviously based on its pronounced ability to defend internal homeostasis against environmental challenge. Previous work had shown that salinity stress increased the abundance of nitric oxide. Is this increase a mere consequence of damage, similar to a human cell that fails to maintain oxidative homeostasis in consequence of ageing, or is it adaptive? Using an artificial donor of nitric oxide, sodium nitroprusside, the authors could significantly improve viability and reduce the degree of lipid peroxidation as an indicator for oxidative damage. The accumulation of proline as a marker for osmotic stress was reduced as well as the level of superoxide anions. In contrast, the level of hydrogen peroxide, a product resulting from scavenging of superoxide anions, was increased. Although the cell biology of oxidative processes differs fundamentally from human cells, these data support a model, where longevity depends on the ability to quench oxidative imbalance. Thus, the findings from this robust desert plant support the notion by Bereiter-Hahn (2014) that Methusalem's secret might be fundamental to all multicellular forms of life.

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