



PLANT CELL BIOLOGY

When autumn falls



...a pathway of three genetic components controls age-dependent cell death.



Although the annual spectacle of leaves falling from deciduous trees is a familiar sight, the mechanisms behind the cell death events that precede autumnal leaf fall are poorly understood. Kim *et al.* now identify three components of a feed-forward loop — *ORESARA 1 (ORE1)*, *ETHYLENE INSENSITIVE 2 (EIN2)* and the microRNA miR164 — that regulate age-dependent senescence in *Arabidopsis thaliana* leaves.

The authors studied the *A. thaliana* leaf senescence mutant *ore1*, which exhibits delayed ageing-associated cell death and delayed loss of chlorophyll, a marker of leaf senescence. They found that this phenotype is due to a 5-base pair deletion in *ORE1*, which encodes a transcription factor. Wild-type *ORE1* expression was increased in senescing leaves, suggesting a key role for *ORE1* in this process.

miR164 binds and cleaves *ORE1* mRNA. As plants age, the amount of miR164 in leaf cells decreases. Consequently, the authors tested whether miR164 might negatively regulate *ORE1* mRNA and therefore age-dependent senescence. Plants that lack *MIR164* have increased levels of *ORE1* mRNA, whereas *MIR164*-overexpressing plants have decreased levels. In addition, the levels of an *ore1* mutant transcript that cannot bind miR164 are less

affected by *MIR164* overexpression. Therefore, miR164 binds and represses *ORE1* mRNA and thereby delays age-dependent senescence.

But how is the temporal expression of *MIR164* controlled such that *ORE1* becomes less repressed as a leaf ages? By looking at other mutants in which cell death and leaf fall are delayed, the authors identified *EIN2* as a further component of this genetic network. Because miR164 levels do not change with age in an *ein2* mutant, wild-type *EIN2* is thought to control the age-dependent reduction of miR164 levels. Furthermore, in older plants with the same *ein2* mutant, the level of *ORE1* mRNA is substantially reduced. This suggests that *EIN2* suppresses miR164-mediated inhibition of *ORE1* mRNA and also induces *ORE1* expression.

These observations reveal that a pathway of three genetic components controls age-dependent cell death. Because *EIN2* also has roles in cell growth control and stress response, it will be interesting to investigate whether this gene can link the genetic control of leaf cell senescence with seasonal environmental signals.

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ORIGINAL RESEARCH PAPER Kim, J. H. *et al.*

Trifurcate feed-forward regulation of age-dependent cell death involving miR164 in *Arabidopsis*. *Science* **323**, 1053–1057 (2009)