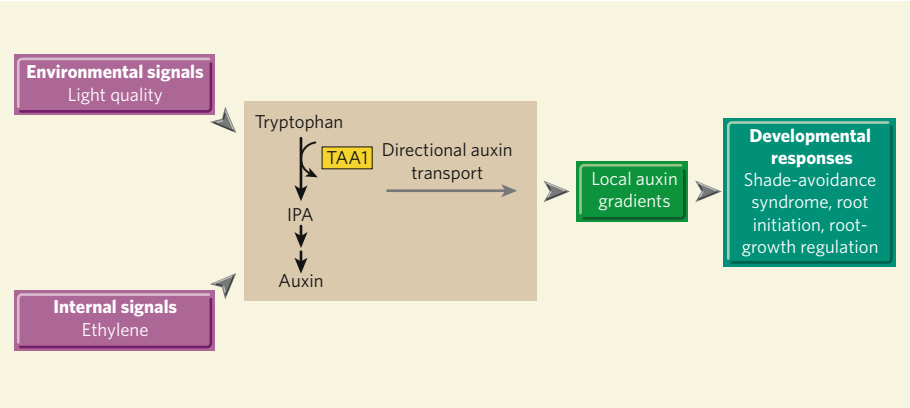

Figure 1 | Plant plasticity. Two studies[2,3] show that environmental cues (light quality) and internal signals (mediated by the hormone ethylene) can both modulate the auxin synthetic pathway that starts with the conversion of tryptophan to indole-3-pyruvic acid (IPA) in a reaction catalysed by the enzyme TAA1. TAA1-dependent local synthesis of auxin, together with the resultant directional auxin transport, leads to auxin gradients that mediate specific adaptive developmental responses in the plant.

proteins mediate directional transport of the hormone. Asymmetric subcellular localization of these transporters ensures directional auxin movement between cells and can generate uneven auxin distribution[6]. Spatially restricted peaks in auxin levels can also be generated by local auxin biosynthesis[7], although the biochemical pathways involved in this process are complex and remain poorly understood. Tao *et al.*[2] and Stepanova *et al.*[3] now identify an enzyme that is essential for local auxin biosynthesis and that markedly affects plant development.

Tao and colleagues performed a genetic screen to identify mutations in the plant *Arabidopsis thaliana* that compromise shade-avoidance responses. They detected the *TAA1* gene, which when mutated prevents plant elongation under shady conditions. This mutant also has lower auxin levels and, in contrast to its normal counterpart, does not produce more auxin when placed in shade. Supplying external auxin restores the shade-avoidance response in *taa1* mutants.

The authors' elaborate biochemical and structural analyses of TAA1 convincingly show that this enzyme catalyses conversion of the amino acid tryptophan to an auxin precursor, indole-3-pyruvic acid (Fig. 1). They also report that the expression of TAA1 is localized, being predominantly confined to the leaf margins, and that it increases under shady conditions. This finding is in line with the observed defects in shade-avoidance syndrome seen in *taa1* mutants.

In addition to confirming the role of TAA1 in auxin biosynthesis, Stepanova and colleagues[3] investigated the developmental relevance of this enzyme. They find two related genes that partially compensate for the loss of TAA1 function. But if all three genes are inactive, the consequences are disastrous: triple mutants are severely defective even during embryonic development, failing to generate a root. Only in certain combinations of the three mutations do plants survive, but at all developmental stages they still show strong, auxin-deficiency-related characteristics that are similar to the effect of mutations in *YUCCA* genes. (*YUCCA* genes encode enzymes that are also implicated in local auxin biosynthesis[7].)

In a slight twist to the story, Stepanova *et al.* identify TAA1 in a screen for tissue-specific modulators of response to another essential plant hormone, ethylene. The *taa1* mutants were insensitive to ethylene, but external auxin application restored their sensitivity, confirming earlier speculation[8–10] about crosstalk between auxin and ethylene.

Taken together, these two papers[2,3] unambiguously identify a central component of auxin biosynthesis and demonstrate its importance for specific developmental responses, as well as for hormone crosstalk. From a biochemical perspective, it will be interesting to see where, in relation to YUCCA, TAA1 resides in the auxin biosynthetic pathway, as the partially overlapping characteristics of these proteins' mutants indicate a common branch. Understanding the spatial and temporal regulation of *TAA1*, and expression of its related genes under different environmental conditions, is also of great interest for the exciting prospect of unravelling the mechanisms of signalling crosstalk in plants. ■

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50 & 100 YEARS AGO

50 YEARS AGO

Niels Henrik Abel: Mathematician Extraordinary — ... [Abel] was a very nineteenth-century genius. The son of a drunken Norwegian clergyman and his profligate wife, he faced poverty as a student; his subsequent rise to fame was hampered by the indifference, and sometimes the misunderstanding, of the established great; his hope of financial reward was frustrated by official vulnerability to pressure groups; and he died of consumption at the age of twenty-six ... The young genius of to-day will find difficulty not in getting his work published, but in getting it noticed amid the flood from his ordinary sound contemporaries. If he dies at twenty-six, it will not be of consumption, but in a mountaineering accident; and however real his genius, so dying, he will surely not leave so deep or so lasting a mark on so wide a sector of his subject as did Abel.
From *Nature* 17 May 1958.

100 YEARS AGO

"The Daylight Saving Bill"... proposes that early on the morning of each of the first four Sundays in April all the public clocks shall be set forward twenty minutes and be set back twenty minutes on each of the four Sunday mornings in September ... But Sir David Gill shows, in a letter in Tuesday's *Times*, that even to change the origin of time once for all requires careful preparation, and that to make changes in the manner proposed by the Bill must lead to confusion ... He points out that if, for example, the Bank of England could be persuaded to open business at 9 a.m. instead of 10 a.m. from April 1 to the end of September, no doubt all other banks and offices would follow suit, and if employers of labour would open their works an hour earlier in the spring and summer months the objects of the Bill would be in great part gained without difficulty and confusion.
From *Nature* 14 May 1908.

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