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Hair follicles arise prenatally and directly begin to produce hair fibres as they undergo morphogenesis. In mouse skin, about two weeks after birth these follicles undergo synchronised arrest of cellular proliferation and enter a brief apoptotic phase (catagen), causing the follicle to regress into a resting phase (telogen). This first catagen marks the end of morphogenesis and the beginning of the adult hair cycle which will continue through life, with the duration of active growth (anagen) phases representing the principal determinant of hair length. In the mouse, the first two hair cycles are entered synchronously across the entire body, making this an excellent system to study hair cycle control, while later cycles are locally synchronised in a travelling wave that moves through the skin.

A number of diffusible signalling proteins and their downstream transduction pathways are known to be involved in hair follicle cycling in vivo, notably those of the BMP, FGF and WNT families. In particular, the well-studied telogen to anagen transition is controlled by opposing WNT/BMP signals that regulate the behaviour of the small population of bulge stem cells (1). Anagen entry, involving WNT activation, is followed by a defined period of growth that terminates in catagen. A wave of dermal BMP signalling then prevents the resumption of anagen, locking follicles for a period in telogen, until this wave passes and they are free to reacquire anagen-inducing signals (2). Cessation of anagen is achieved at least in part through reduction of WNT/β-catenin signalling (3) but no wave directly inducing catagen has been identified. In contrast to the telogen to anagen transition, which involves activation of a small group of stem cells, the shift from anagen to catagen involves the concerted die-off of a large number of epithelial cells, but this process has received less interest and a less complete picture exists. A particularly important question is how extracellular signals are linked to the cell proliferation and apoptotic machinery to achieve rapid changes in cellular state.
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References