Hair follicle dermal papilla cells at a glance

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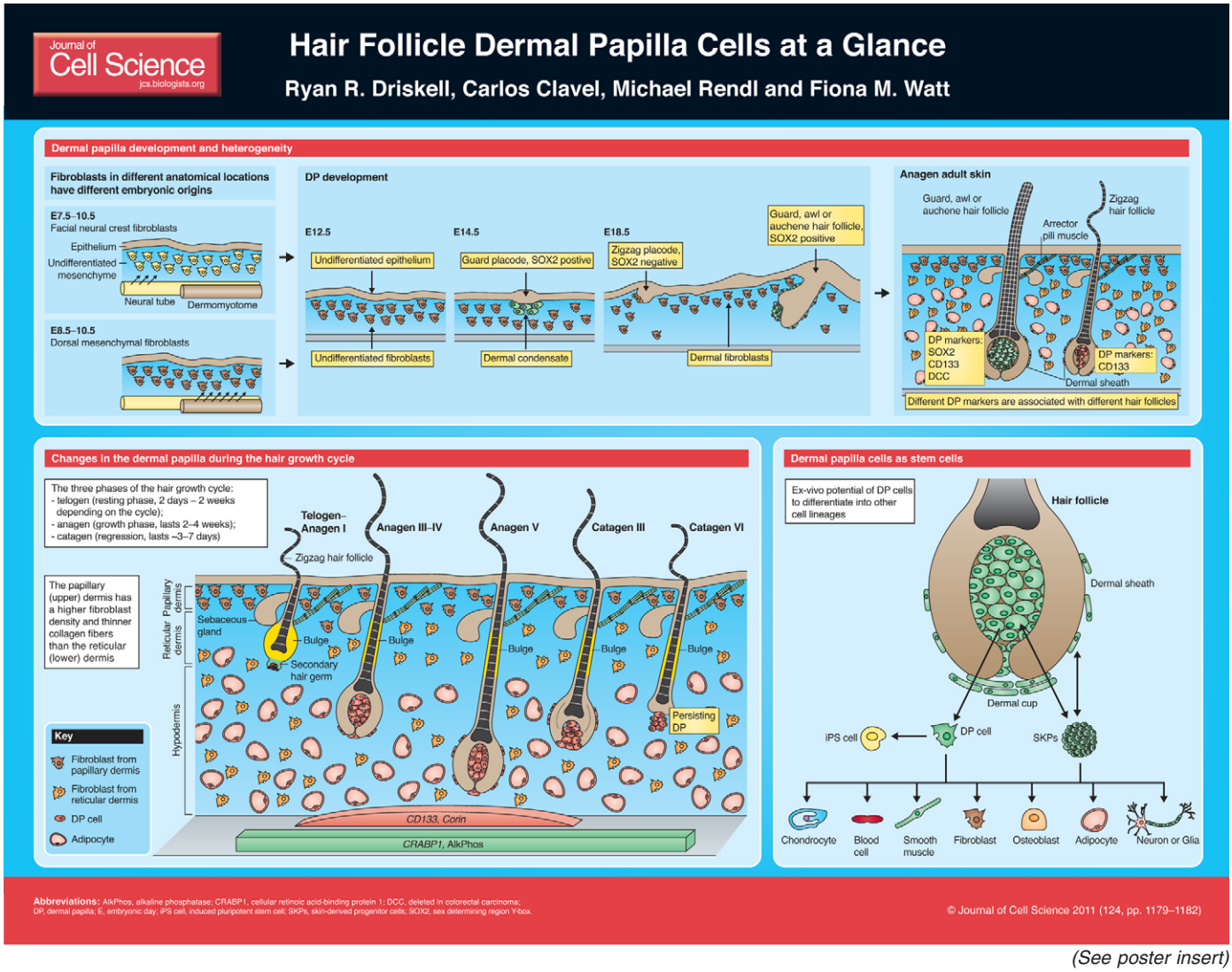
Introduction

Mammalian skin is a highly tractable tissue in which to explore epithelial–mesenchymal interactions during development and in postnatal life (Blanpain and Fuchs, 2009; Müller-Röver et al., 2001; Schmidt-Ullrich and Paus, 2005; Watt and Jensen, 2009). One population of mesenchymal cells in the skin, known as dermal papilla (DP) cells, is the focus of intense interest because the DP not only regulates hair follicle development and growth, but is also thought to be a reservoir of multipotent stem cells. In this article and the accompanying poster we review the origins of the DP during skin development, and discuss DP heterogeneity and the changes in the DP that occur during the hair growth cycle. We also consider the different cell lineages along which DP cells can differentiate as well as potential clinical applications of DP cells.

Developmental origins of dermal papilla cells

The precursor of the hair follicle is a local thickening, also known as placode, of the embryonic epidermis, which is detectable at embryonic day 14.5 (E14.5) of mouse development. Soon after, a local condensation (dermal condensate) of fibroblasts forms beneath the placode. Reciprocal signalling between the condensate and the placode leads to proliferation of the overlying epithelium and downward extension of the new follicle into the dermis (Millar, 2002; Schneider et al., 2009; Ohyama et al., 2010; Yang and Cotsarelis, 2010). After the initial downward growth, the epithelial cells envelope the dermal condensate, thereby forming the mature DP. The DP then instructs the surrounding epithelial cells, now called matrix cells, to proliferate, move upward and differentiate into the multiple layers of the outgrowing hair shaft and the channel surrounding the hair shaft, called the inner root sheath (Millar, 2002; Schneider et al., 2009).

Tissue recombination studies have shown that mouse dermis contains the activity necessary to



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induce hair follicle formation as early as E12.5, before the dermal condensate has developed (Dhouailly, 1973; Song and Sawyer, 1996). Dermis from hair-forming regions of skin can induce follicles in both hair- and non-hair-forming epithelium, whereas dermis from non-hair-forming sites cannot support the formation of hair follicles. Several of the pathways that are involved in reciprocal signalling between the epithelial cells and DP of the developing follicle have been identified, with reciprocal Wnt signalling emerging as one of the earliest and most-important (Kishimoto et al., 2000; Millar, 2002; Schneider et al., 2009; Ohyama et al., 2010; Yang and Cotsarelis, 2010). However, relatively little is known about how dermal condensate and DP cells become hair-inducing specialised fibroblasts (Schneider et al., 2009; Ohyama et al., 2009; Yang and Cotsarelis, 2009).

Fibroblasts and, therefore, DP in different body sites have different embryonic origins (Fernandes et al., 2004; Rendl et al., 2005; Ohtola et al., 2008; Wong et al., 2006; Jinno et al., 2010). Head and facial fibroblasts are derived from the neural crest, whereas dorsal and ventral trunk fibroblasts come from the dermomyotome of somitic and lateral plate origin, respectively. Cell autonomous, site-specific homeobox (*Hox*) gene expression confers positional memory on fibroblasts from different body sites and has a role in specifying the gene expression profile of the overlying epidermis (Rinn et al., 2008).

Dermal papilla heterogeneity

Mouse skin contains several distinct hair follicle types that differ in length, thickness and the shape of the hair shaft, i.e. straight or kinked. In back skin, the most abundant follicles form zigzag hairs, which have two kinks in the shaft, whereas the hairs of other follicle types (guard, awl and auchene) have longer shafts that are either straight or have a single kink (Schlake, 2007). Guard hairs develop during the first wave of hair follicle morphogenesis around E14.5; awl and auchene hairs form in the second wave around E16.5; and zigzag hairs form during the third wave, starting at E18.5 (Schlake, 2007). The DP of zigzag hairs are smaller than those of the other follicle types (Elliott et al., 1999). Between E14.5 and E16.5, all developing DP (that is, those associated with guard, awl or auchene follicles) express the transcription factor sex determining region Y-box 2 (*Sox2*), but SOX2 is undetectable in the DP of zigzag hairs, which develop from E18.5 onwards (Driskell et al., 2009).

Differential Sox2 expression can be used to isolate DP cells originating from different hair follicle types in early postnatal (P2) mouse skin (Driskell et al., 2009). At this stage, all DP cells express CD133 (also known as PROM1) and alkaline phosphatase (Handjiski et al., 1994), but DP of zigzag hairs are SOX2-negative and guard, awl and auchene follicles are SOX2-positive. When different DP populations are sorted and used in skin reconstitution assays, SOX-positive DP cells are found to be necessary for the formation of guard, awl or auchene follicles (Driskell et al., 2009). By contrast, DP expression of SOX is required for the formation of zigzag hair follicles (James et al., 2003). This indicates different roles for two SRY transcription factors in specifying hair follicle type during development.

Gene expression profiling of DP cells isolated from developing mouse skin has resulted in the definition of a core DP 'signature' of 184 genes (Rendl et al., 2005), and in signatures that are specific for SOX2-positive and -negative DP types (Driskell et al., 2009). These signatures are beginning to provide information about the signalling pathways that control DP and hair follicle function, in particular the importance of Wnt, bone morphogenetic protein (BMP) and fibroblast growth factor (FGF) (Greco et al., 2009; Kishimoto et al., 2000; Rendl et al., 2008). In addition, comparison of the properties of hair follicles in different body sites reveals differences in the properties of their DP. One example, in human skin, is the observation that androgens stimulate hair follicle growth in the face but cause follicle miniaturisation in the scalp. DP cells express androgen receptors and 5α-OH-reductase – a key enzyme in androgen metabolism – and DP from different body sites differ in their responsiveness to androgen (Rutberg et al., 2006).

The role of the DP in the hair growth cycle

In postnatal life the hair follicles undergo cyclical growth. The resting phase is known as telogen, the growth phase as anagen and the regression phase as catagen (Müller-Röver et al., 2001; Schmidt-Ullrich and Paus, 2005; Ohyama et al., 2010; Yang and Cotsarelis, 2010). During catagen, the epithelial cells at the base of the follicle undergo apoptosis, but the DP remains intact and is pulled or migrates upwards, until it comes to rest next to the stem cells of the hair follicle bulge. This situation persists during telogen. In anagen, cells at the base of the follicle start to proliferate, which results in downward growth of the follicle and envelopment of the DP. DP cells themselves are thought to not divide. However, the number of cells in the DP increases during anagen, possibly as a result of replenishment from neighbouring cells of the dermal sheath (Tobin et al., 2003; Chi et al., 2010).

At the onset of anagen the DP activates stem cells in the secondary hair germ leading to new downward growth of follicles. In the hairless mutant mouse, DP cells become stranded deep in the dermis during catagen and lose contact with the bulge; the follicles in these mice cannot undergo anagen and eventually degenerate (Panteleyev et al., 1999). Interruption of β-catenin signalling in the DP results in reduced proliferation of cells at the base of the follicle, which induces catagen and prevents anagen induction (Enshell-Seijffers et al., 2010). β-catenin activity in the DP regulates a number of other signalling pathways, including the FGF pathway, which mediate the inductive effects of the DP on the hair follicle epithelium (Enshell-Seijffers et al., 2010). *Fgf7* and *Fgf10* are expressed in the DP and stimulate proliferation of the adjacent epithelial cells of the hair follicle (Greco et al., 2009).

Some DP markers, such as alkaline phosphatase and cellular retinoic-acid-binding protein 1 (*CRABP1*), are expressed throughout the hair growth cycle (Collins and Watt, 2008). Others, such as the serine protease *Corin*, are upregulated during anagen (Enshell-Seijffers et al., 2008). In adult mouse skin *Sox2* expression in the DP varies during the hair growth cycle and is only detectable during anagen (Biernaskie et al., 2009).

Potential therapeutic applications of DP cells in restoring hair growth

Hair loss (alopecia) is a common and distressing problem for men and women, and there is therefore considerable interest in treatments that can prevent or reverse it. Harnessing the ability of the differentiated and highly specialised fibroblasts of the DP to induce neighbouring epidermal cells to differentiate along the hair follicle lineages is an attractive approach to treating alopecia.

The hair-inductive ability of DP cells is not restricted to embryonic development, and DP cells from postnatal skin retain the ability to direct epithelial cells to form hair follicles (Jahoda et al., 1984). Furthermore, formation of new DP can be induced in adult skin by activating the Wnt pathway in the epidermis (Silva-Vargas et al., 2005). These observations suggest that it is possible to generate DP cells in order to treat hair loss.

One obvious strategy is to expand DP cells in culture before transplantation. DP cells not only retain the ability to form DP following in vitro culture, but they can also contribute to dermal sheath cells and non-follicle-associated fibroblasts during skin reconstitution and wound-healing (Biernaskie et al., 2009; Rendl et al., 2008). However, after a few passages cultured DP cells lose their trichogenic

properties (i.e. their ability to induce hair follicles) (Ohyama et al., 2010; Yang and Cotsarelis, 2010; Horne et al., 1986; Kishimoto et al., 2000; Lichti et al., 1993; Rendl et al., 2008). Culture media have been developed that extend the time for which DP cells can be cultured (Limat et al., 1993; Osada et al., 2007; Roh et al., 2004), and activation of Wnt and Bmp signalling pathways in mouse DP cells can delay loss of trichogenicity (Kishimoto et al., 2000; Rendl et al., 2008). Other strategies to preserve the properties of DP cells are to grow them in three-dimensional aggregates (Osada et al., 2007; Higgins et al., 2010) or to culture them together with keratinocytes on extracellular matrix substrates in order to mimic the in vivo microenvironment (Havlickova et al., 2009).

DP cells as stem cells with multi-lineage differentiation potential

Surprisingly, the therapeutic potential of DP cells extends far beyond inducing new hair follicles. To study, or eventually correct, a wide variety of degenerative disorders, induced pluripotent stem (iPS) cells are being generated from patient biopsies (Yamanaka and Blau, 2010). Plucking hair from patients is a non-invasive way to obtain cells for reprogramming, and recent studies have shown that mouse DP cells can be more readily reprogrammed into iPS cells than most other cell types (Tsai et al., 2010).

Another recent finding is that SOX2-positive DP cells are the origin of skin-derived progenitor cells (SKPs). SKPs are cells that can be cultured to form nestin-positive spheres with the capacity to differentiate into neurons, glia, smooth muscle cells, adipocytes and other cell types (Toma et al., 2001; Fernandes et al., 2004; Lavoie et al., 2009; Biernaskie et al., 2009). Since *Sox2* is also expressed in dermal sheath cells close to the DP, it is possible that sheath cells have the ability to form SKPs in culture. SKPs can be generated not only from rodent skin, but also from human hair follicle DP (Hunt et al., 2008). The multi-lineage differentiation potential of cultured DP and dermal sheath cells is not dependent on prior culture as spheroids: they can also differentiate into adipogenic, osteogenic and hematopoietic lineages under other culture conditions (Lako et al., 2002; Jahoda et al., 2003).

The observation that SKPs can be isolated from back skin (Biernaskie et al., 2009) is surprising because multipotent dermal cells have previously been identified to originate from neural crest cells (Fernandes et al., 2004; Wong et al., 2006) and dorsal skin DP arise from the dermomyotome. Recent lineage tracing studies using the *Wnt1* promoter to drive Cre recombinase expression in neural crest derivatives and the myogenic regulatory factor 5 (*Myf5*) promoter to express Cre in cells of somitic origin show that SKPs from both locations can differentiate into Schwann cells, a cell type previously thought to be exclusively derived from the neural crest (Jinno et al., 2010). This suggests that the hair follicle environment, rather than the developmental origin of the cells, induces expression of neural-crest-related genes and generates cells with the characteristics of neural crest derivatives.

Conclusions

The cells of the DP are not only essential for hair follicle development and function, but are also a reservoir of cells with the potential to differentiate into a range of cell types that are of potential therapeutic importance. Improved methods for culturing DP cells can be exploited to treat hair loss, and the ability to direct DP cells to differentiate into other lineages, in particular Schwann cells, could provide a source of cells to repair damaged nerves (Biernaskie et al., 2007).

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