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Commentary

Initiation of Cyclic Hair Regeneration by Hair Follicle Stem Cells and Regenerative Potential

Mario Pascucci*

Department of Tissue Therapy, ASST Niguarda Hospital, Milan, Italy

DESCRIPTION

Initially, it was thought that during the quiescent stage of the hair follicle cycle, hair follicle stem cells were located in the secondary hair germ at the base of the Hair Follicle (HF). Later studies revealed that the bulge and Hair Germ (HG) cells' hfSCs are identical based on the expression of the cell-specific marker keratin15. The HG is in charge of starting a new hair cycle in response to molecular signals from the Dermal Papilla (DP) at the beginning of anagen, even though hfSCs are the primary source of cells in the growth phase of anagen.

The hair germ is an ephemeral region of the Hf that repeatedly developed from the hfSC population during the hair cycle's resting stage. The HG is situated between the DP and bulge hfSCs in telogen phase HFs. The exact timing at which the HG begins to specify is still up for debate. One theory ignores the evidence that cells below the bulge region can survive catagen and contends that the HG is produced directly from the hfSC population during the telogen phase.

Furthermore, HG cells behave as a closely related, extended population of the bulge hfSCs and closely resemble activated bulge hfSCs when comparing these two populations of cells at the transcriptional level. The same genetic markers, such as SCs, K15, Lgr5, Sox9, Tcf3, and Lhx2, are expressed in both the hfSC and HG populations. In contrast to hfSCs, isolated and cultured HG keratinocytes produce larger colonies more quickly, but due to their limited proliferative capacity, they can only proliferate *in vitro* for three passages at a time. Curiously, HG keratinocytes are unable to preserve SC-like characteristics *in vitro*.

HG cells activate more quickly than hfSC during the telogen to anagen transition, and they represent a more dynamic population. By gradually raising the expression level of genes involved in signal transduction pathways and cell-cycle activation, they get ready for the start of anagen. The stability of -catenin, along with other effects of canonical WNT signalling activation, is the initial indicator of HG proliferation. A group of cyclins (Ccnb1, Ccna2, Ccnd2, and Ccnd1) and cyclin-dependent kinases are among the genes heavily elevated in the network of genes implicated in cell cycle progression (Cks2 and Cdc2a).

This has a strong correlation with increased expression of the genes necessary for cell cycle progression in the N-catenin mouse model, which activates WNT signalling. Additionally, new research shows that at the start of anagen, HG swaps Tcf3/Tcf4 for Lef1 at the super-enhancers, and after Lef1 activates WNT signalling (as a result of BMP inhibition), Lef1 forms the nuclear complex with catenin, driving HG committed progenitors.

CONCLUSION

The activation of pSmad2 by Transforming Growth Factor-2 (TGF-2) at the start of anagen has also contributed to the reduced BMP signalling seen in HG proliferation. The significance of DP in this process has recently come to light. By inhibiting the bulge dependent inhibitors of WNT (such as Wif1, Frzb, Sost, Mest, Shisa3, and Igfbp4), which indirectly activate canonical WNT and Shh expression in HG progenitor cells to maintain a brief period of proliferation in the lower part of hfSCs, DP plays a significant role in this process.

Correspondence to: Mario Pascucci, Department of Tissue Therapy, ASST Niguarda Hospital, Milan, Italy, E-mail: Pascucci_mario@gmail.com

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