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Abstracts

Stem Cells and Regeneration

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Cooperation of activin, BMP and FGF signals controls mouse incisor epithelial stem cell proliferation

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Mouse incisors grow continuously throughout life and contain stem cells in the cervical loop area at their proximal end, particularly in the central core of stellate reticulum compartment. Dental epithelial stem cells proliferate and migrate towards the tooth apex and differentiate into the enamel-forming ameloblasts. The labial side cervical loop is thick and contains abundant epithelial stem cells, whereas the lingual cervical loop is very thin with only a few stem cells between the epithelial sheet. Here we show that the proliferation of these epithelial stem cells is regulated by a complex signaling network involving activin, BMP, and FGF, which mediate reciprocal interactions between mesenchymal and epithelial cells in the stem cell niche. Epithelial FGFs induce Fgf3 and activin in the dental mesenchyme. BMP4 inhibits the expression of Fgf3 and subsequent epithelial cell proliferation, whereas activin counteracts this effect. Fgf3 and Fgf10 stimulate epithelial stem cell proliferation. Activin is expressed more intensely on the labial side, leading to the asymmetric expression of Fgf3 only in the labial dental mesenchyme. This generates a bigger cervical loop labially with more proliferating stem cells. Fine-tuning of these signals is responsible for the characteristic asymmetry of mouse incisors and controls the production of dental epithelial progenitors.

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Skin and hair abnormalities in the rough coat mice

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The rough coat (*rc*) mutation arose spontaneously in the C57BL/6J inbred mouse strain. Homozygous *rc* mice were born indistinguishable from their normal littermates but showed unkempt looking coat by weaning age and developed cyclic and progressive hair loss. The *rc* mutation was inherited in an autosomal recessive mode and was previously mapped to chromosome 9. The purpose of this study is to further characterize the skin and hair abnormalities of the *rc/rc* mice and identify the gene mutation underlying such abnormalities. Histological analysis showed severe sebocyte hyperplasia in the *rc/rc* mice. Additionally, more than half of the *rc/rc* mice over 10 months old developed spontaneous and persistent wounds in the skin, suggesting a defect in epidermal maintenance. Because the multipotent keratinocyte stem cells are the source for the cyclic growth of the hair follicle root sheaths and the proliferating, basal cells of the sebaceous glands, as well as epidermal maintenance upon wounding, our observations suggest a possible over-commitment of hair follicle stem cells to sebaceous differentiation in the *rc/rc* mice. To understand the genetic basis of the rc phenotype development, we carried out positional cloning in backcross mice. We identified a mapping interval for the rc locus and reduced it to 246 kb and identified a mutation in a candidate gene. We are currently analyzing the expression of this gene during skin and hair development in normal and *rc/rc* mice. (This study was funded by the NIH and the Ingeborg v.F. McKee Fund through the Hawaii Community Foundation.)

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Inhibition of mammalian muscle differentiation by blastema extract of *S. macrurus*

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Most animals can replace lost tissues through stem cell activation and/or dedifferentiation, that is, the reentry of differentiated cells into the cell cycle. Regeneration capacity in mammals, however, is much diminished and this may reflect a limited dedifferentiation potential. McGann et al. (2001) reported that mouse muscle cells cultured in newt regeneration