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Simplet is required for nuclear localization of beta-catenin and for progenitor cell proliferation and patterning during zebrafish early embryogenesis and tissue regeneration

Christopher L Antos[1], Caghan Kizil[1], Beate Küchler[1], Jia-Jiun Yan[1], Günes Özhan-Kizil[1], Enrico Moro[2], Francesco Argenton[2], Michael Brand[1] and Gilbert Weidinger[1,3]

[1]Technische Universität Dresden, Germany
[2]University of Padova, Italy
[3]Universität Ulm, Germany

Tissue formation and regeneration requires the coordinated contribution of stem and progenitor cells that proliferate and pattern. We show that the gene simplet (smp) is required for both proliferation and patterning of progenitor cells in the blastemas of regenerating zebrafish fins. Furthermore, we determined that Simplet/Fam53B (Smp) is required for Wnt-signaling by positively regulating beta-catenin nuclear localization. In zebrafish embryos, the loss of smp blocks the activity of two beta-catenin-dependent reporters and endogenous target genes as well as precludes nuclear accumulation of beta-catenin. Conversely, overexpression of smp enhances beta-catenin nuclear localization and transcriptional activity. Expression of a mutant Smp protein lacking its nuclear localization signal reveals that the translocation of Smp into the nucleus is essential for beta-catenin-dependent Wnt signaling. We further provide evidence that beta-catenin and Smp interact and that Smp retains beta-catenin in the nucleus. In the mouse intestine, the SMP protein localizes to the crypt cells, which are a pool of stem and progenitor cells that are highly dependent on Wnt signaling. Our findings identify a previously unknown, evolutionary conserved regulator of beta-catenin-dependent Wnt signal transduction that is involved in the regulation of stem and progenitor cells.

christopher.antos@crt-dresden.de