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Calcineurin instructs when to stop regenerating once the correct size of zebrafish appendages is reached

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Animals that regenerate organs and appendages control the growth of stem and progenitor cells to reform lost structures to the same dimensions as the original structures. This proportional regeneration involves coordinating rapid allometric (disproportional) growth with the restoration of isometric (proportional) cell proliferation once the correct tissue dimensions are reached. It is unknown what executes this coordinated control. We show that the calcium-dependent phosphatase calcineurin regulates this control. Calcineurin inhibition results in continued allometric outgrowth of regenerating fins beyond their original dimensions. Congruent with these results, calcineurin activity is low when the rate of progenitor cell proliferation is highest and its activity increases as the regeneration rate decreases. Furthermore, inhibition of calcineurin in uninjured adult fins switches isometric growth into allometric growth demonstrating that calcineurin regulates appendage allometry. Previous results showed that the rate of regenerative outgrowth is controlled by position along the proximodistal axis but it is unknown what this positional control is? Our growth rate measurements and morphometric analysis of proximodistal asymmetry indicate that calcineurin inhibition shifts fin regeneration from a distal isometric growth program to an allometric proximal program. This shift is associated with the promotion of retinoic acid signaling, a signal transduction mechanism that affects positional information along the proximodistal axis. Furthermore, we provide evidence that calcineurin regulates potassium conductance via a potassium leak channel that has been shown to promote allometric growth. In summary, we identified a calcineurin-mediated mechanism that operates as a molecular switch between distal isometric growth and proximal allometric growth.

Biography

Christopher L Antos is a Group Leader (PhD Cell and Molecular Biology) who Heads his laboratory at the DFG-Center for Regenerative Therapies Dresden, an Excellence Cluster at the Technische Universität Dresden. He got his PhD at Southwestern Medical Center at Dallas, University of Texas (UTSW) and subsequently pursued a Postdoctoral fellowship at the Max-Planck Institute for Developmental Biology in Tubingen, Germany, where he started researching regeneration biology using zebrafish as a model. Current research in his lab is focused on the molecular biology that regulates progenitor cell behavior during regeneration of zebrafish structures: Principally what is involved in inducing cells in the residual stump tissues to become progenitor cells and what is involved in instructing these progenitor cells to grow and pattern correctly. From this work, his lab uncovered that the previously uncharacterized molecule Smp is required for progenitor cell participation in regeneration and embryonic development is involved in the Wnt signal transduction by regulating β-catenin nuclear localization.

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