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Forever Young: Linking Genome Size to Regeneration in Salamanders

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Salamanders are unexcelled among vertebrates in their ability to regenerate body parts throughout life. Salamanders also stand out in the accumulation of massive amounts of non-coding genomic DNA, mostly transposable elements, that has occurred repeatedly and independently in nearly every salamander lineage, with extensive variation in genome size between species. The selective advantage of regeneration is obvious, but that of large and variable genomes is not. In fact, large genomes can impose significant morphogenetic constraints in the form of cell size relative to body size, rates and patterns of reproduction and somatic development, and even life history traits, some of which appear to be maladaptive. Here, we explore the idea that both the ability to regenerate and genome expansion reflect a key developmental trait that evolved in the common ancestor of all living salamanders: systemic developmental retardation (SDR), such that salamander tissues are populated throughout life with weakly differentiated cells. This idea is supported by experimental evidence indicating that adult salamander tissues are populated with cells that can easily convert to stem cells capable of re-activating developmental programs necessary for regeneration. Thus salamander tissues are “histologically paedomorphic”. We hypothesize that SDR has been achieved by allowing introns to grow through the accumulation of transposable elements and other kinds of selfish DNA leading to a global slowdown of gene expression across the genome (“intron delay”). This suggests a novel function for introns as non-coding regulators of gene expression, not just by their location, but also by their physical size (length). This also presents a novel view of genome expansion and its phenotypic consequences in salamanders as unavoidable trade-offs for the prodigious ability of salamanders to regenerate lost body parts.