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"Dilp8 requires the neuronal relaxin receptor Lgr3 to couple growth to developmental timing"

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Developmental stability is the ability of an organism to buffer given traits against environmental and intrinsic perturbations and produce stable genetically determined phenotypes. The processes leading to developmental stability involve physiological, temporal or behavioral adjustments to the developmental program that have been particularly well studied in insects. For instance, if uncoordinated growth occurs in Drosophila imaginal discs, the larval precursors of adult structures, a transient delay in the onset of metamorphosis ensues, allowing extra time for all discs to achieve their specific size. We have recently identified dilp8, a fly specific insulin-like peptide that is produced in damaged imaginal discs and couples tissue growth with developmental timing. Dilp8 transiently delays the onset of metamorphosis by inhibiting the biosynthesis of the molting hormone ecdysone while simultaneously slows down growth of undamaged tissues. Thus, the prolonged larval phase allows tissue regeneration while keeping proportions with unaffected discs and results in proportionate adults. Accordingly, loss of dilp8 increases intra-individual asymmetry(1). However, which molecules and tissues sense and/or transmit this abnormal growth signal remained unknown. We have now found that mutation of Lgr3, a member of the type C1 Leucine-rich repeat-containing G-protein coupled receptors, results in body asymmetries similar to that of dilp8 mutants and the inability to delay development in response to tissue damage. By tagging the endogenous Lgr3 protein with GFP we found that it is expressed and required in a subpopulation of CNS neurons not previously linked to growth control. Our work places Dilp8 and Lgr3 as central players in the interorgan communication system that mediates plasticity to promote developmental stability in Drosophila and reveals a novel neuroendocrine circuit responsive to growth aberrations.

(1)Garelli et al, Science 2012 May 4.