



SAN

**Sociedad Argentina de Investigación
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P126.-Neuroendocrine control of puparium morphogenesis

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Drosophila larvae undergo a dramatic change in body shape at the end of the larval growth period when the thin, flexible and transparent cuticle of the larvae is transformed into the puparium. This remodeling is achieved by a series of muscular contractions such as retraction of the anterior segments and body contraction, and accompanied by structural remodeling of the cuticle. Even though the onset of the metamorphosis is known to be under the control of ecdysone, other molecular players have been shown or hypothesized to act downstream of it to mediate different aspects of these behavioral and morphogenetic processes. Serendipitously, we observed that animals lacking the relaxin-receptor like G-protein coupled receptor Lgr3 produce a thin and elongated puparium, indicating that Lgr3 is required for proper puparium morphogenesis. This activity is separable from the previously described role for Lgr3 during larval development, where it has been shown to act in a subpopulation of CNS neurons to coordinate growth with developmental timing by inhibiting ecdysone biosynthesis, in a *Drosophila* insulin-like peptide 8 (Dilp8)-dependent fashion. Rather, our results are consistent with Lgr3 acting in a distinct population of neurons that respond to a developmentally-triggered surge of carcass-derived Dilp8 peptide that occurs at the onset of pupariation. Hence, the Dilp8 and Lgr3 constitute a new neuroendocrine pathway directly contributing to puparium morphogenesis.