



Seminar/Talk

The tight junction protein MarvelD3 regulates eye development and neural crest induction during *Xenopus laevis* early embryogenesis

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Host: Carl-Philipp Heisenberg

Epithelial integrity is essential for organ homeostasis and tissue function. Epithelial cells adhere to each other via multi-protein complexes called junctions. Tight junctions, the most apical junction, regulate the paracellular flux, prevent the intermixing of lipids between membrane domains and act as signalling hubs to guide cell behaviour and function. Despite a well-established role for tight junction proteins in vitro, little is known about their physiological functions, and if and how they contribute to developmental processes. MarvelD3 is a tight junction transmembrane protein that, in vitro, does not affect tight junction assembly or maintenance but functions as a signalling protein that regulates c-Jun N-terminal Kinase (JNK) pathway and, thereby, cell proliferation, migration and survival. Here, we analysed the role of MarvelD3 during early *Xenopus laevis* development using a loss-of-function approach. *marveld3* expression in the dorso-lateral non-neural ectoderm overlaps laterally with the neural crest territory and anteriorly with the eye field. Injection of translation blocking morpholinos against *marveld3* disrupts eye morphogenesis through deregulation of cell proliferation and survival, and also affects the positioning of the border between the neural plate and the non-neural ectoderm, which is essential during development to determine where the neural crest is induced. We further determined that MarvelD3-induced attenuation of JNK activity is indeed required for neural crest induction. In contrast, MarvelD3 promotes JNK activation during eye morphogenesis. Thus, MarvelD3 acts as a switch that regulates the levels of JNK activity in vitro and in vivo guiding crucial cellular and developmental processes.

Monday, December 4, 2017 01:00pm - 02:00pm

Meeting room 3rd floor / Bertalanffy Bldg. (I04.3OG - LAB)



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