



## Seminar/Talk

# Potential caspase-myosin interaction as a regulator of asymmetric cell division in *Caenorhabditis elegans*

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Host: Carl-Philipp Heisenberg

During *Caenorhabditis elegans* development, 131 somatic cells invariably die in a controlled manner. Most of these cells die through a process called apoptosis and are the smaller daughter of a mother cell that divides asymmetrically. In animals with a reduced asymmetry of the mother cell division, the apoptotic fate is often not successfully executed, which results in the presence of extra cells. Thus, asymmetric cell division (ACD) and cell fate specification and execution (including apoptosis) are functionally linked. However, while the influence of ACD on daughter cell fate has been widely investigated, it remains unclear if the factors that determine daughter cell fate also, in turn, govern ACD. During my PhD, I discovered a novel role of the *C. elegans* central apoptotic pathway in promoting asymmetry in divisions of mother cells that produce apoptotic daughters. I found that proapoptotic genes not only ensure that the apoptotic cells are smaller in size but also that they inherit limited amounts of mitotic factors. This role is dependent on the protease function of CED-3 caspase. We have now identified *ect-2*, which encodes a Rho-GEF, as an interactor of *ced-3*. Our results suggest that *ect-2* acts downstream of *ced-3* and that it may mediate the regulation of ACD by *ced-3*. Preliminary results indicate that anisotropic actomyosin contractility in the mother cell may govern its asymmetric division, and, being a positive regulator of actomyosin contractility, *ect-2* is well-placed to facilitate the regulation of ACD by *ced-3*. In my talk, I will discuss the results we have obtained in our attempts to explore this potential CED-3-Myosin interaction mediated by ECT-2 in the context of asymmetric cell division.

**Monday, May 6, 2019 11:00am - 12:15pm**

Meeting room 2nd floor / Bertalanffy Bldg. (I04.2OG - LAB)

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