Life Sciences Seminar

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

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During Caenorhabditis elegans development, 131 somatic cells invariably die in acontrolled manner. Most of these cells die through a process called apoptosis and arethe smaller daughter of a mother cell that divides asymmetrically. In animals with areduced asymmetry of the mother cell division, the apoptotic fate is often notsuccessfully executed, which results in the presence of extra cells. Thus, asymmetriccell division (ACD) and cell fate specification and execution (including apoptosis) arefunctionally linked. However, while the influence of ACD on daughter cell fate has beenwidely investigated, it remains unclear if the factors that determine daughter cell fatealso, in turn, govern ACD. During my PhD, I discovered a novel role of the C. eleganscentral apoptotic pathway in promoting asymmetry in divisions of mother cells thatproduce apoptotic daughters. I found that proapoptotic genes not only ensure that theapoptotic cells are smaller in size but also that they inherit limited amounts of promitoticfactors. This role is dependent on the protease function of CED-3 caspase. We havenow identified ect-2, which encodes a Rho-GEF, as an interactor of ced-3. Our resultssuggest that ect-2 acts downstream of ced-3 and that it may mediate the regulation ofACD by ced-3. Preliminary results indicate that anisotropic actomyosin contractility inthe mother cell may govern its asymmetric division, and, being a positive regulator ofactomyosin 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 thispotential CED-3-Myosin interaction mediated by ECT-2 in the context of asymmetric celldivision.

Monday, May 6, 2019 11:00am - 12:15pm
Meeting room 2nd floor / Bertalanffy Bldg. (I04.2OG - LAB)

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