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Degree Objective: Ph.D. Endocrinology and Reproductive Physiology

Background: BS Molecular Biology Univ Wisconsin-Madison, Madison, WI

Current Research Project:

A large number of umbilical-associated defects are associated with abnormalities in the fetal gastrointestinal system (Stevenson and Hall, 2006). However, the mechanism that unifies the umbilical cord and hindgut are unknown. The goal of my thesis proposal is to elucidate the developmental mechanisms involved in umbilical-associated gut abnormalities. For this, I will characterize posterior visceral endoderm and its role in development of the allantois, or pre-umbilical tissue, and the hindgut. Recently, visceral endoderm, a temporary and nutritive tissue, has been shown to induce hematopoietic cells and pattern the anterior neurectoderm (Belaoussoff et al., 1998; Thomas and Beddington, 1996). In our laboratory, preliminary data suggest that a small segment of posterior visceral endoderm induces the formation of the allantois and establishes a stem cell niche there (Downs et al., 2009). Over the next several hours, the allantois-associated extraembryonic visceral endoderm (AX) undergoes morphological changes, and appears to become a conduit through which primordial germ cells migrate to the hindgut (M. Mikedis and K. Downs, in preparation). Whether the AX actually becomes a part of the hindgut is not known. My immediate goal is to fate map the AX via application of Dil, a lipophilic dye compatible with cell survival, culture the embryos, photobleach the Dil to form a solid precipitate, and carry out histological analysis to discover the whereabouts of the labeled cells. My preliminary data, achieved during my rotation and during which I optimized the photobleaching protocol, provided tantalizing evidence that the AX becomes the midline ventral portion of the hindgut. My goals now are to fate map the visceral endoderm in this posterior region at multiple sites, and then examine the effects of removal of the AX and/or other segments of visceral endoderm on gut formation and primordial germ cell behavior. Results will then be combined with analysis of the Sox17 mutant, which is thought to have defects in gut formation (REF). However, the role of Sox17 in allantoic development is not known.

Honors:

National Science Scholarship (PhD) 2010, Singapore

Grants Received:

National Science Scholarship (PhD) 2010, Singapore

Publications:



National Presentations:

Other Presentations:

ERP Service: