Program/Abstract # 318

Human MOB2 participates in cells migration through Erk signaling pathway
Cheng-Han Lin, Cheng-Po Hu, Seng-Sheen Fan
Tunghai University, Taichung, Taiwan

Cell migration plays important roles in proper embryonic development, wound healing, and tumor metastasis. Cell migration is a dynamic process which involves in reorganization of the cytoskeleton and membrane ruffling. The molecular mechanism of cell migration has been extensively studied but remains unclear. In this study, we identified human MOB2 protein which plays a significant role in promoting cell migration through the Erk signaling pathway in hepatocarcinoma cells. Our results showed that MOB2 was expressed at the leading edge in migrating Mahlavu cells but not in the non-migrating B2 cells. To study the function of MOB2 in cell migration, we manipulated MOB2 expression using RNA interference and overexpression. We then evaluated how MOB2 affected cell migration. We found that knockdown MOB2 expression suppressed cell migration, whereas overexpression of MOB2 facilitated cell migration in a wound-healing and transwell assay. We also found that expression of phosphorylated Erk was increased in MOB2 overexpressing cells. The inhibition of Erk phosphorylation by PD98059 blocked the cell migration in Mahlavu cells. Together, these results provide novel evidence to support the role of human MOB2 in cell migration.

doi:10.1016/j.ydbio.2011.05.274

Program/Abstract # 319

FAK is required for assembly of podosome rosettes
Yi-Ru Pan, Hong-Chen Chen
National Chung Hsing University/Department of Life Science, Taichung, Taiwan

Podosomes are dynamic, actin-enriched membrane structures that play an important role in invasive cell motility and extracellular matrix degradation. They are often found to assemble into large rosette-like structures in highly invasive cells. However, the mechanism for this assembly remains obscure. In this study, we identify focal adhesion kinase (FAK) as a key molecule necessary for the assembly. Moreover, phosphorylation of p130Cas and suppression of Rho signaling by FAK are found to be important for FAK to induce assembly of podosome rosettes. Finally, we find that suppression of vimentin intermediate filaments by FAK may facilitate the assembly of podosome rosettes. Collectively, our results strongly suggest a link between FAK, podosome rosettes, and tumor invasion and unveil a negative role for Rho signaling and vimentin filaments in podosome rosette assembly.

doi:10.1016/j.ydbio.2011.05.275

Program/Abstract # 320

Somatic gonad precursor migration in C. elegans
Monica Rohrschneider*, Jeremy Nance*

In many species, including mouse, Drosophila, and C. elegans, the somatic gonad precursor cells (SGPs) and the primordial germ cells (PGCs) are born at a distance from one another, and must migrate in order to coalesce and form the proper structure of the gonad. In C. elegans, the SGPs migrate nearly half the length of the embryo in order to reach the PGCs. This migration is critical, as the SGPs are required for survival and proliferation of the germ cells. However, little is known about what drives the SGPs to migrate, and what triggers them to stop. To address these questions, we are constructing transgenic strains that will allow us to better characterize the migration of the SGPs and their interactions with neighboring cells. We have found that the SGPs extend projections as they migrate posteriorly along the edge of the endoderm cells, and when they reach the PGCs, their projections appear to wrap around the PGCs. We are using both genetic and physical ablations of the PGCs and of endoderm cells to test the hypothesis that these cells are required for normal SGP migration. Surprisingly, SGP migration is grossly normal in mes-1 mutants which lack PGCs, suggesting that PGCs do not provide a long-range attractive cue to the SGPs. However, SGP migration is disrupted in end-1 end-3 mutants, which lack endoderm, suggesting that endoderm development or morphogenesis is required for normal SGP migration.

doi:10.1016/j.ydbio.2011.05.276

Program/Abstract # 321

Cytoskeletal polarization during collective cell migration in the Drosophila egg chamber
Maureen Cetera, Sally Horne-Badovinac
Chicago, IL, USA

Collective cell migration is critical for proper morphogenesis of developing organisms. The Drosophila egg chamber provides a novel system in which to study collective cell migration of an entire epithelial cell layer. During oogenesis, the egg chamber elongates from a spherical precursor to form a mature elliptical egg. At this time, the migratory follicular epithelium rotates circumferentially around the egg chamber’s anterior–posterior axis. The molecular mechanisms underlying this migration are currently unknown. In a single motile cell, cytoskeletal polarization is essential for proper migration. Similarly, we predict a migrating epithelium will require planar polarization of its cytoskeleton. In the follicle cells, we observe polarized actin-based protrusions extending from one side of each cell with uniform sinistral or dextral chirality. Through live and fixed cell imaging, we have shown the protrusions form the leading edge of the migrating cells, and the VASP protein Enabled localizes at the protrusion tips where it appears to regulate actin dynamics. Additionally, Myosin II is known to relieve focal adhesions at the back of a single motile cell, allowing forward migration. Through live imaging of Myosin II GFP fusion proteins, we have observed planar polarized enrichment of Myosin II along filamentous actin at the trailing edge of the migrating follicle cells. These studies are providing insight into the cytoskeletal dynamics underlying a novel mode of collective cell migration.

doi:10.1016/j.ydbio.2011.05.277

Program/Abstract # 322

SMN: A role in axon growth/fasculation and retaining MMC(m) motor neurons in the ventral neural tube
Catherine E. Krull*, Fengyun Su*, Mustafa Sahin*

SMN is the gene that is responsible for human Spinal Muscular Atrophy (SMA). In humans and primates, there are two SMN genes but in mice, chicks, zebrafish and flies, there is one gene. We have taken a loss-of-function approach to determine the role of SMN in chick motor neurons, using specific SMN shRNAs combined with