



CDB SEMINAR

Speaker: **Masazumi Tada**

< Department of Anatomy and Developmental Biology
University College London >

Title: **"Genetic cascades that regulate gastrulation cell movements in zebrafish"**

Date:	Monday, December 15
Time:	16:30 P.M.~17:30 P.M.
Place:	7th floor Conference Room of Building A, CDB

Summary :

During vertebrate gastrulation, mesodermal and ectodermal cells undergo convergence and extension (CE), a process characterised by prominent cellular rearrangements in which polarised cells intercalate along the medio-lateral axis leading to elongation of the antero-posterior axis. Recent work has implicated that a non-canonical Wnt/Frizzled (Fz)/Dishevelled (Dsh) signalling pathway, related to the planar cell polarity (PCP) pathway in flies, regulates CE during vertebrate gastrulation. It, however, remains to be addressed how PCP gene products regulate CE at the cellular level in the gastrula embryo. We have been characterising zebrafish homologues of *Drosophila prickles* (*pk*) and *flamingo* (*fmi*), genes implicated in the regulation of PCP.

pk1 encodes an intracellular protein with a conserved PET domain and 3 LIM domains and is expressed maternally and in migrating mesodermal precursors. Abrogation of Pk1 function by morpholino oligonucleotides leads to defective CE movements, enhances the *silberblick* (*slb*)/*wnt11* and *pipetail*(*ppt*)/*wnt5* phenotypes and suppresses the ability of Wnt11 to rescue the *slb* phenotype. Gain-of-function of Pk1 also inhibits CE movements and enhances the *slb* phenotype, most likely due to the ability of Pk1 to block the Fz7-dependent membrane localisation of Dsh.

fmi encodes a 7-pass transmembrane protein with extracellular cadherin repeats and two *fmi* genes are expressed broadly on the dorsal side of the gastrula embryo. In order to dissect signalling function of Fmi from cell adhesion function, we constructed a mutant form of Fmi in which the intracellular domain of Fmi is fused with a membrane localisation signal (Lyn-Fmi). Over-expression of *lyn-fmi* leads to severely defective CE movements in the gastrula embryo, reminiscent of the *slb/ppt* double mutant phenotype.

I will discuss our results with respect to mechanisms by which Pk and Fmi regulate CE movements in relation to the non-canonical Wnt/PCP pathway.

Host **Masahiko Hibi** Vertebrate Axis Formation, CDB

E-mail hibi@cdb.riken.jp Tel: 078-306-3135

RIKEN Center for developmental Biology <http://www.cdb.riken.go.jp/>