A genetic mosaic analysis with a repressible cell marker screen to identify genes involved in tracheal cell migration during Drosophila air sac morphogenesis

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Branching morphogenesis of the Drosophila tracheal system relies on the fibroblast growth factor receptor (FGFR) signaling pathway. The Drosophila FGF ligand Branchless (Bnl) and the FGFR Breathless (Btl/FGFR) are required for cell migration during the establishment of the interconnected network of tracheal tubes. However, due to an important maternal contribution of members of the FGFR pathway in the oocyte, a thorough genetic dissection of the role of components of the FGFR signaling cascade in tracheal cell migration is impossible in the embryo. To bypass this shortcoming, we studied tracheal cell migration in the dorsal air sac primordium, a structure that forms during late larval development. Using a mosaic analysis with a repressible cell marker (MARCM) clone approach in mosaic animals, combined with an ethyl methanesulfonate (EMS)-mutagenesis screen of the left arm of the second chromosome, we identified novel genes implicated in cell migration. We screened 1123 mutagenized lines and identified 47 lines displaying tracheal cell migration defects in the air sac primordium. Using complementation analyses based on lethality, mutations in 20 of these lines were genetically mapped to specific genomic areas. Three of the mutants were mapped to either the Mhc or the stam complementation groups. Further experiments confirmed that these genes are required for cell migration in the tracheal air sac primordium.
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