Public Abstract
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An important aspect of brain development involves the migration, or directed movement, of neurons from their birthplaces to their permanent locations within the brain. This process is critical for the establishment of functional neural networks and distinct neural layers. Since defective neuronal migration is an underlying cause of several human diseases, and a major goal in medical research is to induce stem cell-derived neurons to migrate to exact locations in the brain, it is important to understand the mechanisms controlling neuronal migration. To better understand this process, we focus on the caudal migration of facial branchiomotor neurons (FBMNs) in the developing mouse hindbrain. Multiple proteins of the Wnt/planar cell polarity (PCP) signaling pathway had been demonstrated to regulate FBMN migration in zebrafish, but whether they were similarly required in mammals was unclear. Therefore, we analyzed FBMN migration in Wnt/PCP mutant mice.

In *Vangl2* and *Ptk7* (Wnt/PCP genes) mutant mice, FBMNs failed to initiate their caudal migration. However, FBMNs migrated normally in *Dishevelled 1/2* double mutants. Together, this suggests that the caudal migration of FBMNs in controlled by multiple components of the Wnt/PCP signaling pathway, yet may not require the central signaling molecule Dishevelled.

All previously reported FBMN migration mutants have had defects in the initiation of migration. However, we found that in *Celsr1* (another Wnt/PCP gene) mutants, many FBMNs intitiated migration but went in the wrong direction. Since this finding was so novel, we used a certain technology to determine within which cell types *Celsr1* was functioning and also found that the direction that an FBMN migrates in a *Celsr1* mutant is dependent upon its starting position before migration.