

P009 RanBPM contributes to localization of the midbody to the apical junctional complex in neuroepithelium
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In epithelia, including neuroepithelia of the embryonic nervous system, cell division and abscission are confined largely to the apical or ventricular surface. The mechanisms that direct cell abscission to this surface are not known. In neocortical neuroepithelium, citron kinase (CITK) is localized to the apical side of cells in metaphase, cleavage furrow in anaphase and midbody ring during cytokinesis. CITK null mutation results in neurogenesis defects due to cytokinesis failure. We identified RanBPM, a Ran-binding protein, as a novel interactor of CITK. In neocortical neuroepithelium, RanBPM co-localizes with junctional markers (ZO-1 and β -catenin) and partially overlaps with CITK-containing midbodies at the ventricular zone (VZ) surface. Consistent with co-localization of CITK and RanBPM, ultrastructure of E11 neocortex indicates that midbodies at the VZ surface make extensive junctional contacts. RNAi of RanBPM decreases the number of midbodies localized to the VZ surface and causes accumulation of cells in M phase. In conclusion, RanBPM recruits CITK to apical junctions and thereby contributes to localization of midbodies and progression into cytokinesis at the apical surface.