

Meckelin (MKS3) functions in the guided movement and orientation of duplicating basal bodies in *Paramecium tetraurelia* T. Picariello, M. Valentine, J. Yano, J. Van Houten

Meckelin (MKS3) functions in the development of cilia. Mutations in MKS3 lead to Meckel Syndrome (MKS), a disease caused by dysfunctional cilia. *Paramecium* is an excellent model system to study MKS3. Depletion of MKS3 mRNA levels in *Paramecium* lead to short or missing cilia across the entire cell surface and distortions of the ciliary membrane. On the dorsal surface, basal bodies were out of their usual linear rows and the cortical units of the surface were misshapen and disorganized.

The surface of *Paramecium* is organized into units that run the length of the cell. A cilium arises from a basal body that is anchored by three rootlets (post ciliary microtubules, transverse microtubules, and striated rootlets). On the dorsal side of the cell, MKS3 depletion results in clusters of basal bodies and misshapen surface units that deviate from the normal linear alignment. The rootlets that project from each basal body in the areas of misalignment have normal angles between them. However, the rootlets no longer have proper orientation and highlight that the entire basal body-rootlet complex is rotated out of normal alignment.

The striated rootlet (SR) projects toward the anterior from the basal body and traverses several cortical units. Developing basal bodies attach to the SR of the established basal body as they move toward the anterior and turn upright. We propose that, with insufficient MKS3, guidance of the basal bodies toward the anterior of the cell is lost. In further support of this hypothesis, GST pull-down experiments have identified SR proteins as potential interacting partners for MKS3.