Analysis of molecular mechanisms of ciliary beat regulation in *Paramecium*

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SUMMARY

Radial spokes and dynein arms are major ciliary structures, and it assumed that they have important roles for motility of cilia. In fact, there are several reports that defects of these structures lead to abnormal motility. However, the functions of individual proteins composing these structures are still unknown. We attempted to analyze phenotypes of knockdown cells in which particular proteins were silenced with the RNAi feeding method. First, we identified six genes encoding radial spoke proteins, both by comparative genomics and proteomics. Of the six genes, three are highly identical to radial spoke head protein of *Tetrahymena* and *Chlamydomonas*. Silencing of *Paramecium* radial spoke head like 1 (PtRSHL1) led to increased swimming speed, but silencing of PtRSHL2 resulted in decreased swimming speed and a reduced rate of growth. However, silencing both genes results in high sensitivity to Ca^{2+} ion. Silencing PtRSHL3 did not give definite phenotypic changes. Five genes encoding inner dynein arm components were also found from ciliary proteomics. *Paramecium* inner dynein arm intermediate chain 1(PtIC1)-silenced cells are motile but swim forward slowly. However, they did not exhibit backward swimming under KCl and $BaCl_2$ treatment. This result suggests that PtIC1 might be the switch for ciliary reversal induced by internal Ca^{2+} increase.