Motile cilia are tiny cell protrusions that play locomotory functions. In human, motile cilia are formed in large number by epithelial cells lining respiratory tracks, brain ventricles, oviduct, epidydimis (a part of male reproductive trucks, between testis and vas deferens) and as single organelle by sperm cells. Cilia play important functions which when impaired, cause a disorder called primary ciliary dyskinesia, PCD. This genetic disorder leads to chronic infections of respiratory tracks, hydrocephalus and infertility. In about 30-35% of cases the cause of PCD is unknown, mostly due to the huge number of genes whose mutations could potentially cause PCD and genetic tests restricted to only about 30.

Although cilia were discovered already in XVII century and significant number of ciliary proteins was identified during last few decades, we still do not fully understand, how ciliary movement is regulated. We know though that such regulation involves specific structures formed within this organelle.

Motile cilium is composed of nine doublet microtubules forming a circle. On doublets are docked motor proteins, so-called dyneins, which allow microtubule sliding. Effective cilia beating requires coordination of dynein movements, which depends upon additional structures: radial spokes (RSs) and central apparatus (CA) whose interactions regulate dynein activity. Central apparatus is composed of two single microtubules forming numerous projections. Protein composition of CA is known only partially. Therefore our goal is to determine protein composition of CA and to investigate the role of these components in cilia beating regulation.

Our analyses will be conducted with use of a ciliate *Tetrahymena thermophila*, accepted model in studies of cilia structure and functions. New CA protein will be identified using mutant biotin ligase fused to known CA proteins (BioID) and mass spectrometry methods. Localization of newly identified proteins within CA will be confirmed by molecular, biochemical and microscopic methods. Function of new proteins will be revealed by analysis of the phenotype of cells with knocked out genes encoding newly identified proteins. Moreover we will perform analysis of the role of individual domains, protein posttranslational modifications and analysis of interactions between CA components and between CA and RS. Additionally we will identify interactors of STK36, a kinase related to CA, whose mutation is one of the causes of PCD.

Our studies will broaden the general knowledge of cilia structure and regulation of their functions. There is a sub-type of PCD manifests by lack of CA within cilia, but up now the causes were rarely determine. Identification of new CA proteins could contribute to the discovery as-yet unknown causes of this PCD sub-type. As result it will be possible to include new proteins in the PCD diagnosis and possibly, in the development of gene therapy.