In plant cells, actin filaments are nucleated in two different ways: The growth of single filaments or their bundles is enabled by various types of formins, whereas branched meshworks emerge due to Arp2/3 complex activity. Mutations in genes of these nucleators lead to various phenotypic traits.

This thesis deals in the first place with influence of Arp2/3 complex subunits' dysfunction on intracellular motility (cytoplasmic streaming, *stop-and-go* movements of Golgi apparatus cisternae), since it had not been extensively studied before, and also attempts to quantify the already known impacts of mutations in genes for ARP2 and ARPC5 subunits on the vacuolar morphogenesis. For comparison, a few experiments with plants which carried a mutation in gene for FH1 formin were also realised when measuring the cytoplasmic streaming.

The experiments were conducted with a model plant *Arabidopsis thaliana*. The methods particularly included transformation with fluorescent markers by *Agrobacterium tumefaciens* (or usage of a fluorescent dye), microscopy (both standard and confocal) and subsequent evaluation of the acquired data using a computer. During the cytoplasmic streaming research, effects of cytoskeletal drug latrunculin B were studied, too.

The outputs did not prove that the Arp2/3 complex defects would manifest themselves on the behaviour of dictyosomes; nor do they cause any general changes of the cytoplasmic flow. Preliminary data indicate that neither the absence of FH1 formin does significantly influence the velocity of cytoplasmic streaming. However, vacuolar system fragmentation in the leaf epidermis cells of *arp2* and *arpc5* mutants was confirmed and former results were refined.