Summary

Polar auxin transport provides essential directional and positional information for many developmental processes in plants. At the cellular level, it is realized by both the passive diffusion and the active transport through the membrane proteins - AUX1/LAXes, PINs and ABCBs. The aim of this thesis was to characterize the role of ABCB1, ABCB4 and ABCB19 proteins in polar auxin transport using transformed tobacco BY-2 cell lines. It was shown that localization of the ABCB1, 4 and 19 is not polar on the plasma membrane (PM). The ABCB4 was also more stable on PM after the treatment with auxin influx inhibitors 1-NOA, 2-NOA and CHPAA; making use of ABCB4-cell line helped to uncover new characteristics of markers of endocytosis – the FM-dyes. The induction of ABCB19 has led to the decrease in ^{3}H-NAA accumulation with characteristic cell phenotype (cells ceased to divide, started to elongate and there was formed an increased amount of starch granules), similar to the PIN7 overexpressing cell line. Thus, also the ABCB-based enhanced auxin efflux resulted in depletion of auxin from cells, and concomitantly changed their developmental program. The auxin starvation phenotype in the cell line with induced overexpression of PIN7 could be rescued by application of the auxin efflux inhibitor NPA; however, the accumulation of auxin in the ABCB19-overexpressing cell line was less sensitive to NPA, and the rescue of the auxin starvation phenotype was ineffective there. Importantly, the unique property of the ABCB4 was demonstrated: It displayed the dual, auxin-concentration-dependent auxin transport activity in *Arabidopsis* roots, tobacco BY-2 and yeast cells. The results also suggested that the non-competitive inhibition of the ABCB4-mediated auxin efflux contributes to the herbicidal effects of the synthetic auxin analogue 2,4-D.

Besides intercellular transport, there is another process with the potential to modify auxin level, the metabolism. Auxin metabolic profiles together with data from auxin transport assays were produced, and they allowed mathematical modelling of auxin transport on the cellular level by utilizing real quantitative experimental data. It was shown that NAA is strongly and rapidly metabolized in BY-2 cells and the predominant metabolite was identified as NAA glucosyl ester. This metabolite was retained in cells, thus raising apparent intracellular concentration of NAA previously measured during auxin accumulation experiments. This might lead to serious underestimation of auxin efflux carriers transport capacity for NAA as well as IAA measured in the past. The mathematical modelling using both experimental data on accumulation of auxins together with metabolic profiling is currently in progress.