Conclusions

The role of AUXIN BINDING PROTEIN 1 (ABP1) in the auxin management in plant cells was followed using simplified model material of suspension-cultured cells of tobacco BY-2 line. ABP1 is a putative auxin receptor considered to mediate fast non-genomic responses to auxin and it can be involved in every aspect of the regulation of auxin responses, metabolism and transport. There are four major conclusions that could be made based on the results presented in this thesis:

1) Auxin binding protein 1 mediates both cell division and expansion in tobacco BY-2 cells. In standard cultivation conditions or at lower concentrations of 2,4-D in culture medium, ABP1 overexpression had no detectable impact on cell division, cell elongation or cell growth.. 5-times increased 2,4-D concentration stimulated weakly cell elongation. . Antisense suppression of *ABP1* expression resulted in disturbance in both cell expansion and cell division intensity, suggesting that ABP1 is essential for the control of balance between cell division and cell elongation during the growth cycle. ABP1 is localized in endoplasmic reticulum of cells cultivated in standard medium supplemented with 1 μM 2,4-D and it appeared also at the plasma membrane following the IAA application.

2) ABP1 mediates intercellular auxin transport. Cells over-expressing ABP1 were less sensitive to NPA, the inhibitor of auxin efflux carrier activity on plasma membrane, but not to PBA, the inhibitor of actin dynamics. ABP1 over-expression rescued the auxin starvation phenotype induced by the over-expression of the plasma-membrane-localized PIN auxin efflux carriers and decreased the *At*PIN-mediated auxin efflux. Altogether, these findings suggest that ABP1 affects the active auxin efflux.

3) ABP1 affects vesicle trafficking processes related to PIN proteins dynamics. FRAP measurements revealed that *ABP1* inducible over-expression slowed down the PIN1-GFP fluorescence recovery at the plasma membrane. Selective application of inhibitors of anterograde vesicle trafficking (BFA) and endocytosis (Tyrphostin A23) showed that that ABP1 action was independent of anterograde vesicle transport and it stimulated endocytosis of PIN1-GFP. The *ABP1* over-expression also influenced activity of the plasma-membrane-associated phospholipases, previously reported to be important for the trafficking of PINs.

4) ABP1 mediates processes of intracellular auxin sequestration and metabolism. In the presence of IAA in cultivation medium, ABP1 rescued the effects caused by the over-expression of the endoplasmic-reticulum-localized transporter *At*PIN5 and protected free IAA

from conjugation. Thus, ABP1 contributes to the maintenance of intracellular auxin homeostasis by preventing the PIN5-dependent metabolic changes of IAA.