Zinc is an essential micronutrient for plants. It acts as a catalytic or structural co-factor in a large number of enzymes and regulatory proteins. Thus, it is of great interest for cells to tightly control Zn homeostasis, using complex regulatory pathways. Up-to-now, numerous of plants proteins regulating transport and chelation processes involved in the response to Zn-excess, contributing to zinc acquisition from soil, mobilization between organs and tissue, and sequestration within cellular compartments, were recognized. However, the identify of Zn-sensors receptors at the cellular level were not yet done.

The recent studies of research group which I belong, showed that the zinc cellular sensors should be localized in apoplast (continuum of plants cell walls). Experiments performed on transgenic plants characterized by the higher Zn concentration in apoplast in comparison to the wild type plants, demonstrated that in transgenic plants previously manifest the Zn-toxicity symptoms. In transgenic plants earlier was occurred the Zn-storage mesophyll (one of the leaf tissue) cell differentiation, and consequently the formation of necrosis (a group of dead leaf cells with high Zn concentration accumulated inside). Up-to-date, there is nothing known about the apoplastic mechanisms (molecular and/or biochemical) of generation and perception the signal, which causing the described above Zn-toxicity symptoms.

There are indications, that WAKs (Wall-Associated Kinases) family members could play a role in the apoplastic Zn-level perception in plant cells. It is known that WAKs are located in the cell membrane and that, have apoplastic pectin binding domain. It has been proven that the degree of esterification of pectin binding to receptors, determines the type of the following signal processes inside the cell. What is interesting, it was demonstrated that exposure plants to high Zn concentration causing the changes in level of pectins esterification in cell walls. Taking this together, it appears that wall-associated kinases (WAKs) may be involved in the perception of signal of Zn-level in apoplast; signal generated through the pectins molecules in different degree of esterification.

To verify above assumption, for the beginning of the proposed project the expression level of all so far known *WAKLs* (*Wall-Associated Kinases Like*) genes in leaves of tobacco exposed to high Zn concentration, in comparizon to leaves of tobacco exposed to optimal Zn concentration will be examined. Based on the gene expression analysis, to the further study one of the WAKs receptor will be chosen. To check whether the selected receptor is involved in formation the Zn-toxicity symptoms (Zn-storage mesophyll cell differentiation and consequently the necrosis formation), the localization of chosen WAK in relation to Zn localization in the mesophyll cells will be investigated. In addition, to confirm the pectins role in formation the Zn-toxicity symptoms, the co-localization of WAKs receptor in mesophyll cells and the pectins of different degrees of esterification in cell walls will be examined. The cellular localization of WAK and the pectins in relation to different Zn level in apoplast will be studied –the high and the optimal Zn concentration in plants grown medium are planned to comply, also transgenic plants with higher Zn level in apoplast compared to wild type plants will be used.

The results of proposed project will allow to answered the question how plants can sensed the toxic Zn concentration in apoplast, thus Zn-status in the cell. In general, this project may contribute to the better understanding of the mechanisms of heavy metals' tolerance in plants.