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Tobacco mosaic virus induces the expression of cellular factor KPILP for effective reproduction and intercellular movement

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Plants are continuously subjected to diverse range of stresses, and one of them is viral infection that could lead to severe damage of agricultural plants and cause a great loss of yield. High rate of viral genome mutations gives good opportunity for viruses to infect multiple host species. Study of the mechanisms and cellular factors involved in plant defense reactions could reveal new opportunities and instruments for crop protection. One of the factors playing a significant role in plant-virus interactions is Kunitz protease inhibitor-like protein (KPILP) gene in plants of *Solanaceae* family. It has been shown that KPILP expression increases sharply in response to *potato virus X* and *tobacco mosaic virus* (TMV) infection in *Nicotiana benthamiana* [1] and *Nicotiana tabacum* [2] respectively. KPILP is a glycoprotein that stimulates intercellular transport of macromolecules and induces the decrease of callose accumulation level at plasmodesmata - channels, through which low and high molecular weight compounds are transported between cells. Plasmodesmata are essential for the spread of viral infection. The aim of this work is to determine the role of KPILP in development of TMV infection in *N. benthamiana*. We used vector based on TMV genome, as a model for viral infection. Plasmid DNA was delivered into plant cells via agroinfiltration. Viral vector reproduction was demonstrated to induce significant increase of KPILP expression. Western blot analysis revealed the difference in KPILP electrophoretic mobility between proteins extracted from the control leaves and leaves expressing the viral vector. The additional bands with lower electrophoretic mobility were detected using KPILP-specific antibodies in samples from the infected leaves. These bands could correspond to KPILP complex with cellular or viral protein(s) formed during viral infection. When we performed KPILP knockdown using virus-induced gene silencing approach and assessed the efficiency of TMV infection in such plants we revealed 10-fold decrease in TMV RNA accumulation level compared to the control plants and significant suppression of TMV intercellular movement efficiency. Based on the obtained results we suggest that KPILP interacts with viral proteins and/or other cellular factors and stimulates viral reproduction and spread. Therefore, TMV induces KPILP expression creating favorable conditions for the development of the effective viral infection.

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References

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