Nitric oxide as an epigenetic mediator of effector-triggered immunity (ETI) in potato

Late blight caused by *Phytophthora infestans* is the most devastating crop disease in the world. Moreover, in view of the annual considerable economic losses caused by potato late blight, any effort to fill the existing gaps in knowledge on the potential role of NO in the epigenetic control of host resistance after challenge inoculation with *P. infestans* is of great value. One important question that arises from the presented project is whether and how nitric oxide (NO) influences the expression of resistant genes (*R*-genes) implicated in the RNA-directed DNA methylation (RdDM) pathway. Plants have evolved hundreds of innate immune receptor genes against a broad range of various pathogen effectors. Precise regulation of R-genes is pivotal to prevent fitness cost and autoimmune responses in the absence of the pathogen. Non-coding miRNAs block a wide range of *R*-genes causing posttranscriptional gene silencing and keeping transcriptionally silent chromatin. In turn, early and rapid overexpression of *R*-genes is necessary to improve resistance under biotic stress. The boosted NO generation also occurs during the first minutes after pathogen recognition and the NO signal is rapidly translated into redox sensing targets, effective in triggering plant resistance. It is reasonable to speculate that NO could also function in the precise fine-tuning *R*-gene co-activation during the host-pathogen interaction.

The research will focus on the identification of direct targets of NO which provide a link with intranuclear signals influencing chromatin remodeling and immunity-specific gene expression. The plant material used in the proposal will involve two potato genotypes possessing *R-3a* and *Rpi-phu1* with the corresponding avirulent *Phytophthora infestans* isolates giving the hypersensitive response (HR) type of resistance. It is planned to detect NO-mediated modifications of S-adenosyl-L-homocysteine hydrolase, being a donor of the methyl group in chromatin methylation, and run a functional analysis of these changes in relation to chromatin remodeling. DNA and histone methylation governed by specific methyltransferases affects chromatin structure and its function in transcriptional regulation. Typically, pathogen ingress involves host genome hypomethylation, which can influence biogenesis of small RNAs by suppression of *R*-gene silencing and enhanced resistance. Thus**, we intend to find a connection between NO and epigenetic marks making chromatin more accessible for transcription factors in the promoter regions of the *R*-gene. Moreover, we are planning to identify peroxynitrite-mediated ($ONOO^-$) modification of mRNA and intend to explain pathophysiological consequences of these changes, probably resulting in the suppression of posttranscriptional gene silencing, downregulation of miRNA targeting the *R*-gene (*R3a* and *Rpi-phu1*) and enhanced potato resistance to late blight disease.**

In accordance with the sequence of the planned tasks, successful realization of the above-mentioned stages will be useful in clarification of the NO cross-talk with *R*-genes potentially having the epigenetic background. Whether such an interplay of NO and epigenetic marks determines resistance or host susceptibility has not yet been recognized for plants.