

Abstract

The major human pathogen *Streptococcus pneumoniae* is a unique model for the study of eukaryotic-type serine/threonine protein kinases and its cognate phosphatases in bacteria, since it encodes only a single signaling pair composed of the StkP protein kinase and PhpP phosphatase. This signaling pair plays a role in several cellular processes, mainly in cell wall biosynthesis and cell division. StkP and PhpP proteins with a pleiotropic effect appear to regulate a complex signaling cascade by phosphorylation of many substrates. However, only a few have been characterized so far. Using MS analysis, we have identified about 90 phosphopeptides that are potential substrates for the StkP kinase and PhpP phosphatase.

This diploma thesis is focused on the characterization of the new substrate Spr0929 and its role in pneumococcal physiology. One of the objectives was to investigate cell morphology of strains carrying deletion of the *spr0929* gene in different genetic backgrounds. It turned out that the role of Spr0929 in cell morphology is strain specific. The growth curves of strains with this deletion were compared to that of the wild type in various physiological conditions as well.

As Spr0929 contains a nucleoid-associated domain called NdpA, determination of its cell localization was an important objective, too. Using translation fusion of Spr0929 with GFP, its localization near bacterial nucleoid was found.

The results obtained also confirmed the assumed phosphorylation site of this protein on threonine 255. StkP-mediated phosphorylation at this site was confirmed in both *in vitro* and *in vivo* conditions.

Taken together, the results presented in this diploma thesis help to decipher the regulation of a new signaling cascade controlled by StkP and PhpP.