Staphylococcus spp. are Gram-positive and non-motile bacteria. According to their ability to coagulate rabbit plasma, staphylococci are divided into two groups, i.e., coagulase-positive (CPS) and coagulase-negative (CNS). So far, *Staphylococcus aureus* (*S. aureus*) remains most characterized but also most pathogenic CPS. Enterotoxins produced by this species are recognized cause of staphylococcal food poisoning, participate in development of wide range of clinical disorders, and are supposed to favor host colonization by staphylococci. Enterotoxins present in foodstuffs contaminated with *S. aureus* may cause symptoms of food poisoning in humans. Enterotoxins are highly stable proteins, hence their emetic activity remains unaltered even in thermally processed food. According to the EU legislation, certain food products are routinely examined for enterotoxins presence, however only if *S. aureus* is detected in food. Recent surveys revealed that also CNS are able to produce enterotoxins. CNS group comprises over 40 species traditionally considered non harmful or at worst opportunistic pathogens. Currently, it is widely documented that CNS may pose a considerable threat to human and animal health. First complete sequence of enterotoxin genes in CNS was published in 2011, netx one was recently characterized in our laboratory. However, significance of CNS enterotoxin for public health remains largely unknown.

Enterotoxin genes occurring in *S. aureus* are located on so called mobile genetic elements what implies they can move between staphylococcal strains. Very little is known on mobility of enterotoxin genes in CNS. Main scientific problem that should be currently resolved concerns the structure of genetic elements bearing enterotoxin genes in CNS, and assessment of their mobility. It is also critical to resolve whether certain CNS species are better fitted to acquire enterotoxin genes. Main direction of enterotoxin genes flow also need to be determined, to show whether it occurs from CNS to S. aureus or in opposite way.

Already published research and our results indicate that enterotoxin genes can occur in CNS in stable and unstable forms. Loss of enterotoxin genes was observed in some CNS but not in *S. aureus* strains following cultivation in microbiological media. To study this phenomenon copy number of enterotoxin genes in a range of CNS species and toxigenic *S. aureus* strains will be assessed. Visualization of enterotoxin gene-positive cells.

We found enterotoxigenic CNS frequently occurring in food and livestock animals, implying that those environments should stabilize enterotoxin genes. We have also shown that standard laboratory conditions can be inadequate to study CNS enterotoxins. Therefore, to determine factors stabilizing enterotoxin genes in CNS non-standard laboratory media and culture on food matrices will be tested.

To determine the requirements of CNS genomes to function as enterotoxin gene recipients enterotoxin-coding elements will be identified in CNS genomes. Complete genome sequences will be determined in CNS stably carrying enterotoxin genes.

Using bacteriophages frequently occurring in staphylococci we will assess the transfer of mobile genetic elements between CNS and *S. aureus*.

The ability of CNS to carry enterotoxin genes homologues was yet supported by unambiguous data. However, only some CNS were shown to stably harbour enterotoxin gens being able to secrete enterotoxins to the environment, thus potentially contributing to staphylococcal food poisoning and clinical disorders. Therefore, significance of CNS enterotoxigenicity still remains unknown. The main question is whether these genes pose a direct public health hazard acting as CNS virulence factors or their significance is indirect, since they constitute gene pool to supply pathogenic staphylococcal species with virulence factors. This project was designed to resolve a number of questions important from food safety and public health point of view. Its results will allow to assess the significance of CNS enterotoxins in pathogenicity and evolution of staphylococcal virulence.