Carolina Berglund was born in 1980 in Stockholm and she moved to Örebro when she was 20 years old to begin her biomedical studies at the Örebro University. The interest and fascination for bacteria and infectious diseases was raised at an early age and Carolinas first paper on the subject was written already during junior high school. Her specific studies of staphylococci began 2003 as a degree project at the Department of Clinical Microbiology, Örebro University Hospital, and subsequently continued with doctoral studies of methicillin-resistant *Staphylococcus aureus*, that are resistant to treatment with the staphylococcal penicillins. During her doctoral studies, Carolina has been working as a guest researcher at the Juntendo University in Tokyo, Japan, in 2006-2007, with the group supervised by Prof. Keichi Hiramatsu and Dr. Teruyo Ito, who are world-leading researchers in the fields of molecular biology and antibiotic resistance. In addition, detailed studies of the Panton Valentine leukocidin (PVL) were performed in 2007 at the Université Louis Pasteur in Strasbourg, France, under supervision of Dr. Gilles Prévost and colleagues.

Methicillin-resistant *S. aureus* (MRSA) arise by the acquisition of the *mecA* gene located on the staphylococcal cassette chromosome *mec* (SCCmec), and this pathogen has shown a unique capability to adapt to almost every situation, as demonstrated by the ability to accumulate resistance to almost all groups of antibiotics. The recent change in epidemiology and increase of MRSA in the healthy population outside the hospitals may be a serious future threat, especially since these community-acquired MRSA (CA-MRSA) may possess virulence genes, e.g. PVL, that confers a higher pathogenic potential in comparison with the nosocomial MRSA or methicillin-susceptible *S. aureus*.

This thesis describes the molecular epidemiology of MRSA with focus on community-acquired isolates that represent more than half of the MRSA cases in Örebro County. The results of this thesis contribute to the insight of the evolutionary origin of MRSA and the understanding of the epidemiology of CA-MRSA. In conclusion, nosocomial MRSA have disseminated throughout the whole world by colonized or infected persons following healthcare contacts, while in contrast, the CA-MRSA appear to arise spontaneously by horizontal acquisition of SCCmec into the chromosome of previously susceptible *S. aureus* from a donor, possibly methicillin-resistant CoNS, that colonize the human skin, and may serve as a reservoir for SCCmec.