1. Introduction

Anthrax is an acute disease primarily affecting herbivores but also found in other mammals including humans and even birds. Natural infections still play an important role in third world countries where both human and animal cases are regularly reported. In the past years anthrax has gained new interest as a potential agent of biological warfare. The Gulf wars and the anthrax postal attacks in the USA in 2001 have induced a recent boost in research activity.

The disease is found in three forms. The cutaneous form is the most common in humans and results from contact with infected animals or infected animal products. Pulmonary anthrax is much less common and a result of spore deposition in the lungs, while the gastrointestinal form is due to ingestion of infected meat**.** Most literature cites cutaneous disease as constituting 95% of cases, with pulmonary disease responsible for 5% and the gastrointestinal form for 1% to 5%.

The Gram-positve bacterium *B. anthracis*, the etiologic agent of anthrax, infects through intradermal inoculation, ingestion or inhalation of spores. Spores are the dormant form of *B. anthracis* and are extremely resistant to environmental stress. In the current model of respiratory infections, the spores are first taken up by macrophages where they germinate and become vegetative bacteria. Macrophages transport the bacteria to the regional lymph nodes. In these organs, the bacteria escape from the macrophage and spread through the lymphatics and blood stream causing massive septicaemia. Vegetative *B. anthracis* express two essential virulence factors: the tripartite anthrax toxin and the poly-γ-D glutamic acid capsule. The anthrax toxin protective antigen binds to its specific receptor and translocates the oedema (EF) and lethal factors (LF) into the cytosol. EF is an adenylate cyclase that causes tissue edema, whereas LF is a metalloprotease that inactivates mitogen-activated protein kinase kinase and provokes cell death.

Neutrophils are a vital component of the acute inflammatory response and play a key role in the resolution of microbial infections. They engulf microbes into a phagosome, which fuses with intracellular granules to form a phagolysosome. In the phagolysosome the bacteria are killed through the interaction of reactive oxygen species (ROS) and oxygen-independent mediators such as enzymes and antimicrobial peptides. Antimicrobial peptides are predominantly cationic and are thought to permeabilize the bacterial membrane and lyse microbes.

Inhalational and gastrointestinal *B. anthracis* infections result in sepsis and death, while cutaneous anthrax almost always remains localized. Interestingly, in untreated cutaneous cases, neutrophils surround the necrotic, bacteria-containing tissue, whereas neutrophil infiltration is rarely seen in the lung during inhalational anthrax. To date there has been no conclusive study looking at the role of neutrophils in the pathogenesis of this disease. Therefore, the aim of this study was to examine the interaction of both the vegetative form and the spores of *B. anthracis* with human neutrophils.