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The severity of disease out-come is believed to depend on the virulence factors of the infectious agent and immune response of each individual. Host genetic factors have been found to play pivotal role in explaining why some individuals respond with severe, life threatening symptoms, while others display a quiescent progression, or are even asymptomatic carriers for infection with the same strain of pathogen. This thesis investigates the role of two gene variants of the NLRP3 inflammasome signaling in the response to bacterial infections. NLRP3 inflammasome; a key component of the innate immune system, can be activated by a number of diverse stimuli, including exposure to whole pathogens, bacterial toxins, endogenous danger signals and environmental irritants. Activation of the NLRP3 inflammasome results in the release of potent proinflammatory cytokines that triggers the inflammatory response of the host. Polymorphisms Q705K and C10X are two gene variants of the NLRP3 inflammasome that combined or per se have been associated with higher risk and severity of chronic inflammation and excessive production of proinflammatory cytokines. The aims of this thesis were to investigate the association of these polymorphisms with bacterial infections and the inflammatory response. Moreover, to determine the inflammasome activation state in healthy carriers of these polymorphisms. A special focus was set on the bacteria *Neisseria meningitidis* infection that remains a major cause of bacterial meningitis and sepsis globally.

ISSN 1652-4063
ISBN 978-91-7529-154-3

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2016

Doctoral Dissertation

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Medical Science with a specialisation in Biomedicine