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The impact of the classical, lectin and alternative pathways of complement activation on protective immunity against *Streptococcus pneumonia* infection following vaccination with pneumococcal vaccines

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S.pneumoniae (pneumococcus) is a pathogen that can cause serious infectious diseases that can be life-threatening, including meningitis, septicaemia and pneumonia. The complement system plays a major role in the immune response to *S. pneumonia* infections. The complement system is a part of the innate immune system that protects the body from invading pathogens, including *S. pneumoniae*. The aim of this work is to determine the impact of selective deficiencies of the classical, the lectin and the alternative pathways of complement activation on the vaccination response to pneumococcal polysaccharide vaccines. I am comparing these results with those achieved using CRM197 (non-toxic mutant of diphtheria toxin) which elicits an immune response of a different kind than that achieved with pneumococcal polysaccharide vaccines. In addition to the i.p. immunisation route, WT mice, C1q-/- and MASP-2-/- mice were immunized subcutaneously (s.c./i.p.) with 20 µg of CRM197 or with 1µg of PneumovaxII (pneumococcal polysaccharide vaccine). These experiments aimed to insure and compare the antibody response to CRM197 and to PneumovaxII in WT mice and complement deficient mice deficient of the components, C1q-/- and MASP-2-/- mice. When immunized with single or 3 spaced doses of CRM197 or PneumovaxII all mice generally responded with high antibody titres for this vaccine protein compound (CRM197) and for PneumovaxII. My results indicate that strong increase in the antibody response to CRM197, occurs immediately after third immunization. However, the strong antibody response to PneumovaxII, occurs immediately after first immunisation. My result also suggests that the antibody response to CRM197 is independent of the presence or absence of C1q (the classical pathway) or MASP-2 (the lectin pathway). However, the antibody response to PneumovaxII is dramatically altered in C1q deficient mice.

Biography

Youssef Ali D Alaofi is pursuing his PhD at the University of Leicester, UK in the field of infection, immunity and inflammation. He received a scholarship from government of Saudi.

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