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Mucosal Immune Responses to both Immunization and Infection of Haemophilus influenzae B

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The introduction of Haemophilus influenzae B conjugate vaccines during the 1990s was followed by dramatic decreases both within the incidence of Haemophilus influenzae B related invasive disease and in nasopharyngeal carriage of the organism. The extent of this effect has been influenced by the very fact that Haemophilus influenzae B conjugate vaccines reduce nasopharyngeal carriage and induce herd immunity. Supported the success of Haemophilus influenzae B conjugate vaccines, chemical conjugation has been applied to the event of pneumococcal and meningococcal polysaccharide conjugate vaccines. Evidence has begun to accumulate that these new polysaccharide based conjugate vaccines also can reduce nasopharyngeal carriage and may induce immune responses at the local mucosal level, which can be liable for these effects. This text reviews recent studies on mucosal immune responses induced by polysaccharide based vaccines and a few protein vaccine antigens against several pathogenic nasopharyngeal bacteria, and discusses the mechanisms and functions of those immune responses which will help our understanding of mucosal immune responses to both immunization and infection [1].

Every year, many people die of infectious diseases worldwide, most of which are caused by pathogens invading the host via mucosal surfaces, including the tract. Several new mucosal vaccines against respiratory infections are under development. Live attenuated mucosal influenza vaccine has been licensed within the USA, but it'll probably be a while before others enter general use. Recent studies show that parenteral administered capsular polysaccharide (PS) based vaccines can induce mucosal immune responses. These immune responses could also be important both within the prevention of invasive diseases and within the reduction of upper respiratory carriage of pathogens.

Hemophilic influenza B, Streptococcus pneumonia, and Neisseria meningitides colonies the mucosa of the human upper tract alongside other opportunistic pathogens and commensal bacteria. The nasopharynx is presumed to be the most site of invasion into the bloodstream. The transmission of those bacteria primarily between asymptomatic carriers is thru droplet spread or contact with respiratory secretions. To be effective against colonization, vaccines must induce local immune responses, which promote elimination of the pathogen, break the chain of transmission, and induce herd immunity [2].

It has long been recognized that serum antibodies to capsular PS of some bacteria including H influenza B ,S pneumonia, and N meningitides are protective against invasive disease. Unconjugated PS vaccines are available for several years and have received some use in adults. However, because they induce a T cell independent B cell response, they're poorly immunogenic in young children, and in adults only induce relatively short term protection.

Conjugate vaccine technology, where a polysaccharide antigen is coupled chemically to a protein carrier, either by direct linkage or by indirect coupling via daimio spacer molecules, can render the PS specific immune reaction T cell dependent. With the assistance of T cell derived factors, the antigen specific B cells produce a way enhanced antibody response. Several protein carriers are used including tetanus toxoid (TT), diphtheria toxoid, mutant diphtheria toxin (CRM197), and therefore the outer membrane protein of N meningitides. Different conjugate vaccines with different protein carriers vary in their immunogenicity. Whereas some conjugate vaccines (for example, H influenzae B polyribosyl phosphate–outer membrane protein) are shown to be immunogenic after one dose in infancy, other H influenzae B vaccines with different protein carriers need two to 3 doses to possess appreciable immunogenicity. The TT carrier has been suggested to be a far better primer than CRM197 for immune responses induced by the conjugate meningococcal C vaccines. Conjugate vaccines with different carrier proteins have also been shown to induce antibody responses with varying avidity [3].

Conjugate vaccines can induce effective primary immune responses in young children and supply long-term protection through the induction of immunological memory. The introduction of H influenza B conjugate vaccines in several countries within the 1980s and 1990s was followed by a rapid reduction in H influenza B related invasive disease and nasopharyngeal carriage. After the success of H influenza B conjugate vaccines, an equivalent approach has been applied to the event of latest conjugate vaccines against S pneumoniae12 and N meningitides 13. Early results have suggested that these vaccines could also be effective against mucosal carriage of the vaccine serotypes.

Despite the effectiveness of polysaccharide–protein conjugate vaccines, protection is restricted to those serotypes of bacteria covered by the vaccine serotypes, and it's possible that they'll get replaced within the mucosa by other serotypes after immunization. Within the case of N meningitides B, the polysaccharide capsule may be a very poor immunogenic and there are theoretical risks associated with rendering it immunogenic by conjugation. For these reasons, efforts have also been made within the past few years to spot effective protein vaccine antigens that would have a broad spectrum of serotype coverage among these bacteria, especially S pneumonia, which has over 90 serotypes.

Here, we review recent studies on mucosal immune responses induced by polysaccharide based vaccines and a few protein vaccine antigens against several pathogenic nasopharyngeal bacteria, and discuss the mechanisms and functions of those immune responses which may help our understanding of mucosal immune responses to both immunization and infection [4].

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Mechanisms of Mucosal Immunity Induced By Parenteral Vaccination

Both PS IgA1 and IgA2 subclasses are detectable after vaccination.80 The IgA2 subclass of IgA antibodies may provide some functional advantage in specific mucosal immune responses over IgA1 as a results of structural differences that make IgA2 relatively immune to IgA1 protease activity.81,82 Haemophilus influenza B , S pneumonia, and N meningitides can produce IgA1 protease, which may cleave IgA1 to Fab and Fc fragments, and may therefore eliminate the Fc mediated functions of IgA1,82,83 although human secretory IgA has been shown to be immune to this protease activity. It's been suggested that in mucosal secretions, IgA antibodies against protein antigens are predominantly IgA1, whereas those directed against polysaccharides are almost equally distributed between the 2 subclasses [5].

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Conflict of interest

None

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