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Role of interleukin-10 gene promoter polymorphism in the severity of group A *Streptococcus* infection

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Group A Streptococci (GAS) with several virulence factors contributing to their pathogenesis are responsible for a variety of human illnesses. The variability of illnesses from mild uncomplicated infections to severe invasive diseases proposes that host genetic variation is the determining factor that modulates disease manifestations and outcomes. Unbalanced inflammatory response against streptococcal super antigens (Strep-SAgs) with avalanche of cytokine release is the main reason for severe manifestations. Interleukin 10 (IL-10), a Cytokine Synthesis Inhibitory Factor (CSIF), plays a pivotal role in controlling the process of inflammation. The production of IL-10 is controlled by 3 Single Nucleotide Polymorphisms (SNPs) in the promoter region, -1082G/A, -819C/T and -592C/A. The association of these SNPs with the severity of GAS infection was investigated. Alleles and haplotypes, known to be associated with low production of IL-10, were found to be more represented in individuals that were subjected to early death due to GAS infection.

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Broadly neutralizing antibodies to dengue virus

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Dengue is a rapidly emerging, mosquito borne viral infection with an estimated 400 million infections occurring annually; of these approximately one quarter are clinically apparent or symptomatic. The majority of these result in a self limited but none the less unpleasant febrile illness, dengue fever. 1-5% of infections lead to a more severe disease, dengue hemorrhagic fever, which is characterized by a severe vascular leak, hypovolemia and in extreme cases shock and hemorrhage. Dengue exists as four highly divergent serotypes differing in sequence by some 30-35%; infection with one serotype does not provide protection against the other three. In endemic areas serotypes frequently co-circulate and repeat infections are common. Interestingly, severe disease is much more common in secondary as opposed to primary infections, implying a role of the acquired immune system in disease pathogenesis. Understanding this immune enhancement of disease is crucial for the design of safe and effective vaccines. Through clinical collaborations in Thailand and Vietnam, we have been studying the immune response to dengue in cohorts of infected children. We have characterized 145 human monoclonal antibodies (mAbs) and identified a previously unknown epitope, the envelope dimer epitope (EDE) that bridges two envelope protein subunits that make up the 90 repeating dimers on the mature virion. We will describe the antibody response to the two virion surface glycoproteins prM and E and discuss the E dimer epitope (EDE), a novel site bridging the 90 basic head to tail envelope dimers making up the virion surface. The antibodies directed to the EDE are both potent and broadly neutralizing across the dengue sero complex suggesting the EDE could be a target for a new generation of subunit vaccines against dengue.

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