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Development of a mutated human parvovirus B19 vaccine to prevent aplastic anemia in congenital and acquired anemias

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Human parvovirus B19, the only human erythrovirus, targets a blood group globoside on progenitor red blood cells causing congenital abnormalities and aplastic anemia in a pleothra of individuals with congenital and acquired anemias. B19 recombinant capsid vaccines, that were composed of wild type VP1 and 2, have been associated with adverse side effects and low immunogenicities. Unlike other parvoviruses (eg., canine, porcine), B19 has a unique viral ligand, P-antigen binding site, that is located in the recess of the 3-fold axis of the virion. B19V virions and virus like particles (VLPs) bind to a blood group globoside, P-antigen, predominantly expressed on immature and mature red blood cells of humans. We hypothesize that binding of the B19 capsid to the blood group globoside causes a structural change in the neutralizing conformationally dependent epitopes permitting infection of erythroid precursors and development of aplastic anemia. To prevent attachment of B19 to globosides and facilitate development of an efficacious vaccine, we mutated the ligand, P-antigen binding site, on VP2 major capsid protein that is responsible for binding to precursor and mature RBCs and generated VLPs, that are incapable of inducing hemagglutination, using baculovirus expression system. We now present the structural analysis of the mutated B19 capsid, and preliminary data on its antigenicity.

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Myeloid derived suppressor cells and their role in infant responses to vaccines

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Myeloid-Derived Suppressor Cells (MDSC) have been extensively studied and strongly implicated in cancer progression and chronic inflammation. MDSC are a heterogenous collection of immature myeloid cells with suppressive T cell and NK cell activity. Their frequencies are elevated in most cancers and chronic viral infections and their numbers correlate with disease stage. We and others have shown that MDSC are also elevated at birth and remain elevated during the first weeks of life, a time when infants are known to be vulnerable to infections and do not respond optimally to vaccines. Infant MDSC potently suppress T cell and NK cell responses *in vitro*, however the role they may play *in vivo* is unknown. Here we describe data from a longitudinal study looking at the effect of MDSC play on vaccine responses. One hundred newborns were recruited at birth and followed for 1 year in Khayelitsha, Cape Town, South Africa. Responses to BCG, Hepatitis B and Tetanus Toxoid were measured at various time points and correlated to the frequency of MDSC at the time of vaccine administration. The role MDSC may play in modulating responses to vaccines and their manipulation at birth by the use of specific vaccine vectors/adjuvants may result in more efficacious infant immunity. A better understanding of the underlying immune mechanisms behind inadequate infant responses to infections or vaccines is a major goal of neonatal immunology and necessary to inform effective vaccination prior to pathogen exposure.

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