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Exposing and Measuring Suppressed HIV Specific Humoral Immunity

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Detection of HIV infection by serum antibodies is hindered by a delay in antibody production, post infection, as can be seen by the relatively long window period between infection and seroconversion. This delay could potentially mask the initial set of primed cells at the onset of the infection.
We used an in-vitro stimulation technology to induce the production of HIV specific antibodies at the seronegative state of the infection in order to detect those early infected individuals and analyze the profile of those early antibodies. Fresh blood samples from 200 very high risk individuals were tested for HIV antibodies with and without the pre-stimulation of the blood sample. Twenty one were seropositive without pre-stimulation, and 25 (21+additional 4) were positive after the pre-stimulation. All positives were confirmed by second ELISA assays. Using PCR, integrated HIV-1 was detected in 5/5 additional positives.
The antigenic profile of the antibodies in the serum of seropositive samples was compared to that of induced antibodies form the early, seronegative, stages of the infection. It was found that while most of the antigenic targets were the same, there were several peptides against which there were antibodies only after in-vitro stimulation. Thus the antibody repertoire in the seropositive individuals does not represent the whole "story" of the initial encounter with the HIV. Peptides from two strains of HIV were used and for both the epitops "seen" at the earliest stages of the infection were of peptides from the variable regions.
These findings could shed light not only on the early immune recognition of HIV but also on epitops against which the immune response has been "silenced" to be detected only by in-vitro stimulation that overcomes that suppression or peripheral tolerance. Thus it could have an impact both on HIV vaccine design and its clinical research.