Antibody Responses in Anogenital Secretions of RV305, a Late Boost Vaccination of RV144 Volunteers


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Abstract

Background

RV144 participants remain uncharacterized. Anogenital mucosae are the primary site of HIV-1 acquisition (vi). Mucosal HIV-1 specific antibodies are thought to be involved in protection of viral acquisition at mucosal sites (vii). Half of the antibodies in genital secretions originate from peripheral blood, while most antibodies in intestinal secretions are produced by plasma cells present in the lamina region.

Methods

A total of 162 healthy HIV-uninfected vaccine recipients who completed the full vaccination series from RV144 were randomized to receive one of three booster or placebo regimens in a follow-up protocol (RV305). Each group included 45 vaccine and 9 placebo recipients who were immunized at study entry and 6 months. Group I received AL VAC-HIV or AL VAC placebo. The protocol received approval from local and international Institutional Review Boards. Blood samples were collected from participants at baseline and 2 weeks post first and second injections for analysis of plasma, cervico-vaginal mucus (CVM), seminal plasma (SP), and rectal secretions (RS).

Results

The induction of gp120 and V1/V2 loop specific IgGs in CVM and SP suggests a possible mechanism by which the vaccine may function to prevent HIV-1 acquisition. Antibody responses in CVM and SP were inducible after being boosted with AIDSVAX®B/E or AL VAC-HIV/AIDSVAX®B/E. We did not detect antibody responses in SP following boosting with AL VAC-HIV/AIDSVAX®B/E. In CVM, we detected antibody responses to gp120 and gp70V1V2 CRF01_AE for both groups at the plasma concentrations tested. This study is limited by studying only baseline and two-week post injection time-points. Analysis of additional inter-injection and late time-points will allow for more detailed understanding of antibody responses.

Conclusions

The induction of IgG and IgA responses in CVM and SP following boosting with AIDSVAX®B/E or AL VAC-HIV/AIDSVAX®B/E may be a critical mechanism of vaccine-induced protection against HIV-1 acquisition. Further studies are needed to assess vaccine-induced antibodies in genital secretions and their potential role in protection against HIV-1 acquisition.

References

