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Antibody responses to V2 loop are induced by CRF01_A E and not Clade B envelopes

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Background

The RV144 vaccine trial of canarypox vCP1521 (ALVAC-HIV) prime and bivalent HIV-1 envelop gp120 protein subtype B/CRF01_AE boost (AIDSVAX B/E) demonstrated a significant effect in preventing HIV-1 infection. A case-control analysis suggested that variable loops 1 and 2 (V2) of gp120 may have contributed to protection against HIV-1 acquisition. Two other vaccine trials using gp120 only– VAX003 (AIDSVAX B/E) and VAX004 (AIDSVAX B/B) failed to show protection.

Methods

Binding antibody responses induced by the RV144, VAX003 and VAX004 vaccine regimens were compared using ELISA. Recombinant gp120 envelope proteins MN (subtype B), 92TH023 (CRF01_AE), A244 (CRF01_AE) and cyclic V2 peptides were used as capture antigens.

Results

After two protein injections, VAX004 had the highest geometric mean titers (GMT) against MN (25,600), VAX003 against A244 (21,378) and RV144 against 92TH023 (6,263). Antibody responses against V2 (CRF01_AE) were detected in plasma samples from RV144 and VAX003 with GMTs of 972 and 1100, respectively. However, VAX004 failed to generate antibodies against CRF01_AE V2. None of the three vaccines generated antibodies against MN V2 after two protein immunizations.

Compared to VAX004, VAX003 had higher antibody responses against all three recombinant proteins: 2-fold (MN), 4-fold (A244) and 4-fold (92TH023) when two additional protein injections were administered. Two additional protein inoculations in the VAX trials failed to increase antibody titers against, CRF01_AE V2, but generated a small response against MN V2 (GMT, 76) in VAX003.

Conclusion

Antibody responses against V2 were induced by CRF01_A E recombinant proteins as there were no responses induced by the AIDSVAX B/B vaccine regimen. Repeated protein immunization increased the magnitude of responses against recombinant proteins in VAX003 but failed to increase titers against CRF01_AE V2. If antibodies against V2 are protective against HIV-1 acquisition, designing antigens with greater V2 antigenicity would be critical.

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