Retrovirology

Poster presentation

P19-24. Evaluation of recombinant influenza-SIV vaccines in macaques

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Background

HIV vaccines that induce mucosal immunity are urgently needed. Common mucosal compartments enable vaccines to be administered to the upper respiratory tract for concomitant immunity in the vagina and rectum. We investigated the potential of recombinant influenza as a live-attenuated vaccine to induce SIV-specific T cell immunity in a non-human primate model of HIV infection.

Methods

Recombinant H1N1 and H3N2 influenza A strains were engineered to express SIV CD8 T cell epitopes and evaluated for their capacity to induce primary and booster responses following administration to the respiratory tract of Pig-tail macaques. Two naïve macaques and eight SIV infected macaques were vaccinated with two doses of H1N1-expressing SIV KP9 followed by two doses of H3N2-expressing SIV KP9 or vice-versa and evaluated for influenza infection, SIV epitope-specific and influenza-specific T cell responses. Naïve macaques were subsequently challenged with SIVmac251. Three influenza viruses expressing different SIV CD8 epitopes were also evaluated.

Results

Influenza infection was asymptomatic however macaques seroconverted, generated T cell responses to influenza proteins and virus was detected in the respiratory tract. SIV KP9-specific CD8 T cell responses were primed in naive animals and a proportion expressed the mucosal homing marker β7 integrin. SIV challenge resulted in a large anamnestic recall CD8 T cell response but immune escape rapidly ensued and there was no impact on chronic SIV viremia. Vaccination of SIV infected macaques resulted in the boosting of existing KP9 responses. Sequential vaccination with different influenza strains produced only a limited boost in immunity, probably reflecting T cell immunity to internal influenza proteins. Vaccinating with multiple SIV epitopes augmented responses to each epitope.

Conclusion

These results augur well for the development of influenza to deliver HIV antigens. Broader antigen cover will be needed to limit CTL escape. We are now developing constructs which express whole SIV proteins.