PO - (8383) - THE ROLE OF PLASMA B CELLS IN MYCOBACTERIUM TUBERCULOSIS INFECTION AND DISEASE

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The Role of Plasma B cells in *Mycobacterium tuberculosis* Infection and Disease

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**ABSTRACT**

**Background:** Tuberculosis (TB) is still a major global health problem with about one-quarter of the global population infected with the causative pathogen, *Mycobacterium tuberculosis* (*Mtb*). The role of T-cells in the adaptive immune response against TB has been extensively studied with little information on the role of B-cells. B-cells produce antibodies and differentiate into plasma and memory B-cells. Plasmablasts are a subset of plasma cells only present in the peripheral circulation following an ongoing infection or vaccination. Immunoglobulin G (IgG) especially IgG2 mounts more efficient immune response against bacterial infections, mainly attribute to the high affinity of IgG2 binding to the Fcγ receptor. Therefore, we hypothesised that *Mtb*-specific IgG+ plasmablasts may be a useful biomarker of TB infection status.

**Methods:** Ex-vivo B-cell Enzyme-linked immunospot (ELISPOT) was used to identify plasmablasts responses to *Mtb*-specific antigens ESAT-6/CFP-10 (EC), together with non-specific (*Mtb* purified protein derivative (PPD) and a positive (total IgG) and negative (media only) control from adults with active TB pre and post-treatment (n=20) or with latent TB infection (LTBI; n=20) in The Gambia.

**Results:** Frequencies of *Mtb*-specific plasmablasts were significantly higher in active TB cases pre-treatment compared to post treatment (p<0.0001) and LTBI with no difference seen following PPD stimulation. Interestingly, total IgG+ cells were lower in the cases at recruitment but increased following treatment indicating the relative proportion of *Mtb*-specific responses were also significantly different (p=0.034) prior to therapy.

**Conclusions:** These data show that B-cell responses are differentially modulated during active and latent TB infection, suggesting that plasmablasts may be a useful biomarker for TB infection in TB-endemic settings.

**Keywords:** Tuberculosis, B-cells, Plasmablasts, antibodies