IL-17 producing T cells in the control of skin inflammation and subcutaneous adipose wasting during chronic *Trypanosoma brucei* infection

African trypanosomes colonise the skin in a process critical for disease transmission. However, the immunological barriers that these parasites must overcome to ensure transmission are far from being fully understood. Here, we addressed this gap in knowledge by applying a combination of spatially resolved single cell transcriptomics, and *in vivo* murine models of infection. We observed a significant expansion of Vγ6$^+$ $\gamma\delta$ T cell and T$_H$17 T cells in the chronically infected mouse skin compared to healthy controls, both of which produce significant levels of the inflammatory cytokine IL-17. *In silico* cell-cell interaction analysis suggests that the activation of these IL-17-producing T cells is mediated *via Cd40, Il6, Il10,* and *Tnfsf18* signalling derived from subcutaneous adipocytes. *In vivo*, we first observed that the absence of Vγ6$^+$ $\gamma\delta$ T cells results in an exacerbated dermal inflammation during infection, characterised by a heightened frequency of IFNγ-producing cytotoxic CD8$^+$ T cells. Interestingly, we also found that global deletion of IL-17 prevents the characteristic weight loss associated with this disease. Unexpectedly, we found that abrogation of IL-17 signalling exclusively on adipocytes results in a limited adipocyte turnover over time, characterised by a significant accumulation of *Dpp4*$^+$ *Pi16*$^+$ interstitial immature preadipocytes and a higher burden of extravascular parasites in the subcutaneous adipose tissue, demonstrating that IL-17 signalling is crucial for controlling preadipocyte fate, subcutaneous adipose tissue replenishment, and local parasite burden. These studies highlight a previously unappreciated crosstalk between subcutaneous adipocytes and $\gamma\delta$ T cells during chronic *T. brucei* infection, orchestrated by IL-17. In the context of *T. brucei* infection, IL-17 signalling plays pleiotropic roles in the skin, preventing excessive CD8$^+$ T cell activation and modulating subcutaneous adipose tissue remodelling to prevent wasting. Altogether, these studies reveal mechanisms of $\gamma\delta$ T cells-mediated immunity in the skin in the context of African trypanosome infection, as well as a novel role for adipocytes as regulators of skin immunity during chronic infection.