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Abstract: Bacterial endotoxic shock-like response during *Trypanosoma congolense* infection – a case of immunological mimicry?

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Bovine Trypanosomiasis due to *Trypanosoma congolense* remains a threat to livestock production in tsetse-infested pockets of Sub-Saharan Africa posing a socio-economic hurdle to farmers. Three mouse strains varying in degree of susceptibility to disease have been identified as models for study of bovine Trypanosomiasis. To understand the gene expression dynamics during *T.congolense* infection, transcriptional profiling of tissue-specific responses in tolerant (C57BL/6), moderately susceptible (Balb/c) and susceptible (A/J) mice was performed.

210 mice of each strain were randomly allocated to groups of 30 and subsequently infected with *T.congolense* parasites over 0, 3, 5, 7, 9, 13, and 17 days. Mice were sacrificed at designated timepoints, RNA was extracted from the livers and hybridized to Affymetrix GeneChip Mouse Genome 430 2.0 oligonucleotide arrays.

The results showed that the early response to *T.congolense* infection was characterized by up-regulation of inflammation genes with an expression signature akin to the endotoxic shock response elicited by the bacterial endotoxin, lipopolysaccharide (LPS). This observation was suggestive of *T.congolense*-specific structural components that mimicked bacterial LPS in induction of an inflammatory immune response. Follow-up studies in an immunologically naïve mouse macrophage culture system using *E.coli* LPS as a positive control confirmed *T.congolense* variant surface glycoprotein (VSG) and DNA to be potential inducers of LPS-like response with

the tolerant mouse model, C57BL/6 showing higher levels of inflammatory cytokines and chemokines than the susceptible model, A/J. *T.congolense*-induced inflammation may be beneficial to the host as an early response to infection but may potentially underlie associated pathology including anaemia and cachexia.