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Biological pathways discriminating African trypanotolerant and trypanosusceptible cattle breeds

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Talk / Poster title: Biological Pathways Discriminating African Trypanotolerant and Trypanosusceptible Cattle Breeds?

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Animal African Trypanosomosis, trypanotolerance, RNA-seq, pathways analysis, host*parasite interactions

Abstract (250 words maximum):

Animal African Trypanosomosis (AAT) is a vector-borne disease caused by blood protozoan parasites of the Trypanosoma genus. It represents a major constraint to the development of cattle breeding in the humid and sub-humid zones of Africa because of the high morbidity and mortality it causes. Zebu breeds and European taurine breeds are very susceptible to AAT and they usually die in the absence treatment. On the contrary, some taurine breeds in West Africa have the capacity to tolerate the disease and are called trypanotolerant.

The trypanotolerant phenotype is known to be polygenic and multifactorial, but up to now, its mechanisms remain unknown. In order to decipher the molecular bases of trypanotolerance, we chose to analyse the genes expression of blood cells of susceptible and tolerant cattle during an experimental infection, performed in Burkina Faso, in 40 cattle from five West African breeds with *T. congolense*. mRNA were extracted from blood, comprising bovine leukocytes and parasites, before and during the infection and were sequenced using a Illumina highSeq2000. We mapped the reads on the bovine and trypanosome genomes, counted the reads on the annotated genes and performed a differential expression analysis. The genes identified as differentially expressed during the infection were then analysed using the Ingenuity Pathway Analysis software in order to identify enriched functional patterns. The functional analyses highlighted upstream regulators and canonical pathways associated with the immune response, the cell proliferation and signaling. Very fine differences in the modulation of the response between trypanotolerant and susceptible cattle were observed.