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Host-microbe interactions of the tsetse fly innate immunity to control the commensal bacterium *Sodalis glossinidius* and its impact on *Trypanosoma brucei* transmission

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Tsetse flies are the sole-vectors of protozoan parasites *Trypanosoma brucei* that cause human and animal African trypanosomiasis. Additionally, tsetse flies harbor a microbiome of low diversity with interactions of varying degrees of integration. The commensal bacterium *Sodalis glossinidius* is in a transitory state from free-living towards symbiosis and comes in close proximity with trypanosome parasites, although it remains controversial in the context of trypanosome transmission. We use the tsetse-*Sodalis* model to study host-microbe interactions using the RNA seq-based approach of transcriptome profiling in symbiont-deprived flies. As a result, *Sodalis* induces 34 out of 12,968 host genes, while *E. coli* results in 1,495 altered genes. This shows that symbiont-deprived flies remain the capacity to mount a response against exogenous bacteria, while they respond differently to the closely-related *Sodalis* bacterium. Further in-depth analysis of the innate immunity genes revealed activation of Gram-negative responsive immunity genes to *E. coli*, while host immunity is more fine-tuned to the commensal. As an outcome, *Sodalis* induces less immune effectors, demonstrating that the tsetse-*Sodalis* interaction has established mechanisms to balance immunity and raises the question whether the commensal bacterium can suppress immunity after activation. In addition, we showed that long-term absence or presence of *Sodalis* does not impact the fly's immunity to septic injury, although the underlying mechanisms here remain unclear. Further functional studies with gene silencing will indicate the impact of host immunity on the commensal bacterium, and unravel its role in transmission of the trypanosome parasite to clarify the underlying mechanisms of the tsetse-*Sodalis*-trypanosome tripartite.

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