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**Title (250 Characters):** *Trichomonas* – Bacteria interactions: A Laterally Acquired Molecular Toolkit to Target the Microbiota and Potentially Enable Zoonotic Events.

**Authors:** Adam J. Hart, Jacob Biboy, Joseph Gray, Waldemar Vollmer, Robert P. Hirt

**Abstract (Max 4000 Characters):**

Species members of the *Trichomonas* genus are responsible for disease in several animal species including Humans. Most notably *Trichomonas vaginalis*, the most common non-viral STI for humans, causes trichomoniasis, which is strongly associated with increased susceptibility to other important pathogens such as HIV and HPV.

*Trichomonas* species live at various mucosal surfaces characterised by complex microbiota amongst a wide range of animal hosts. *Trichomonas* infections are associated with significant changes in the microbiota's taxonomic composition commonly referred to as dysbiosis when associated with diseases. Notably, infections with *Trichomonas vaginalis* and *Trichomonas gallinae* (a bird parasite) result in the depletion of the mutualist bacteria *Lactobacillus* spp., known to play numerous important roles in female urogenital defence against pathogens. However, the molecular and cellular basis of interactions between *Trichomonas* and members of the microbiota are poorly understood.

We exploited *Trichomonas gallinae* in co-culture with *Escherichia coli* as a model system in combination with comparative genomics and transcriptomics (RNAseq) to identify candidate enzymes and peptides targeting bacteria. A number of genes encoding homologues of bacterial cell wall targeting enzymes, including candidate lysozymes, and anti-microbial peptides, both of which are conserved throughout *Trichomonas* spp., were identified and with a number of them shown to be significantly upregulated within the co-culture system. We also observed changes in bacterial and parasite behaviour and morphology.

Integrating comparative genomics between species, transcriptomics within our model and already known interactions between *Trichomonas* and bacteria; involving for example the peptidoglycan targeting NlpC/P60 endopeptidases; illustrates a number of mechanisms for the parasites to potentially out-compete neighbouring bacteria, with a likely important role of deconstructing bacterial cell wall peptidoglycans. This could be to extract nutrients from the microbiota and/or alter the microbiota to produce a more hospitable host environment for the *Trichomonas*. This could lead to the promotion of *Trichomonas* species growth within an infected individual and through dysbiosis, could also contribute at promoting damaging host tissue inflammations. Furthermore, the conserved ability to target the bacteria from the microbiota could contribute to enabling zoonosis.

Together these findings bring new insights into the molecular and cellular basis of *Trichomonas*-Bacterial interactions and how these evolutionary conserved interactions, gained through several lateral gene transfers from bacteria, can potentially influence the zoonotic ability of *Trichomonas*.