



SPEECH INFORMATION (For Conference Program Book)

Topic	LEVERAGING A GUT MICROBE AND ITS METABOLITE TO RELIEVE STRESS-INDUCED GUT DYSFUNCTION
Abstract	<p>Stress coping is essential for survival, and its dysregulation increases the risk of brain disorders. Commensal microbiota play a critical role in stress regulation. We identified <i>Enterococcus faecalis</i> (Ef) as a bacterium that suppresses corticosterone and promotes social behavior. This study investigates Ef-derived metabolites in stress regulation using multi-omics approaches. Microbiota-deficient mice colonized with distinct Ef strains were subjected to acute stress, revealing strain-specific effects in alleviating gut dysmotility and lowering corticosterone. Chemogenetic inhibition of a brain circuit controlling sympathetic activity similarly reduced gut dysmotility, while adrenalectomy or elimination of gut tyrosine hydroxylase-positive neurons had no effect. Metabolomic profiling identified strain-dependent differences in amino acid metabolism, and delivery of Ef-derived metabolites to stressed mice alleviated gut dysmotility. Genome sequencing further revealed key Ef genes involved in amino acid metabolism. Together, these findings demonstrate that a single commensal microbe can mitigate stress via metabolites and neural circuit modulation, highlighting therapeutic potential for stress-related disorders.</p>

