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**Human Gut Microbiota And Regulation Of Human Behaviour** 

## <u>ABSTRACT</u>

The human gut harbors a dynamic and complex microbial ecosystem, consisting of approximately 1 kg of bacteria in the average adult, approximately the weight of the human brain.

The evolutionary formation of a complex gut microbiota in mammals has played an important role in enabling brain development and perhaps sophisticated social interaction. Genes within the human gut microbiota, termed the microbiome, significantly out number human genes in the body, and capable of producing a myriad of neuroactive compounds. Gut microbes are part of the unconscious system regulating behavior. Recent investigation indicate that these microbs majorly impact

cognitive function and fundamental behavior patterns, such as social interaction and stress management. The gut microbiota-brain axis has become an intriguing field, attracting attention from both gastroenterologists and neurobiologists. The

hippocampus is the center of learning and memory, and plays a pivotal role in

neurodegenerative diseases, such as Alzheimer's disease(AD). Previous studies using

administration, antibiotics, probiotics, germ- free mice, and feacal analysis of normal and specific pathogen free animals have shown that the structure and function of the hippocampus are affected by the gut microbiota. Genomic and neurochemical analyses revealed significant alterations in genes and amino acids in the hippocampus of AD

Subjects following a remarkable shift in the gut microbiota.

A comprehensive 16S rRNA gene sequence analysis of Dom and Sub mice revealed a significantly different gut microbiota composition that clearly distinguishes between the two behavioral modes. Finally, fecal microbiota transplantation into germ-free mice show that Sub-transplanted mice acquired Sub microbiota and adopted their behavioral and physiological features, including depressive like and anti-social behaviors alongside reduced eWAT mass, smaller adipocytes, and a Sub-like eWAT adipokine profile.