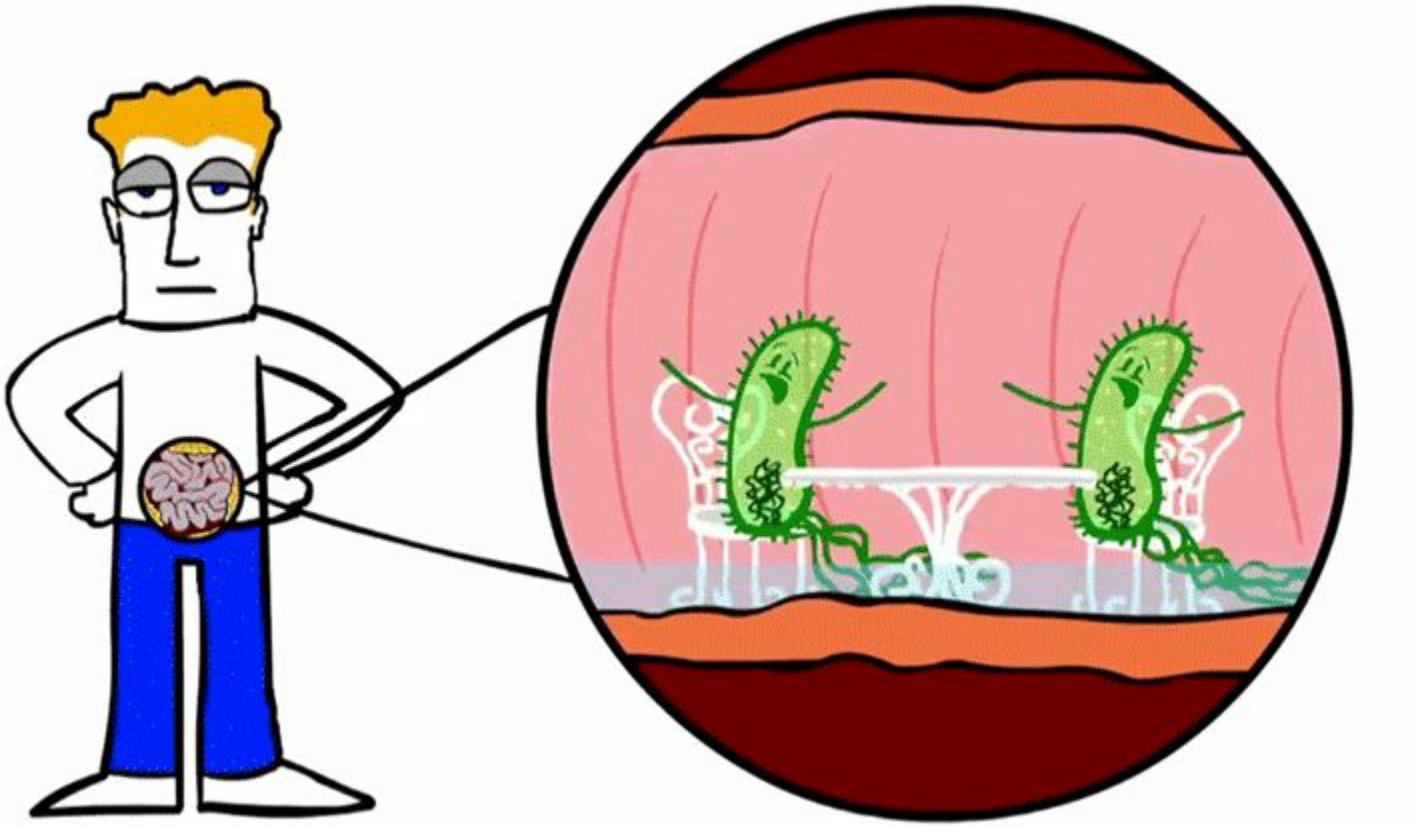
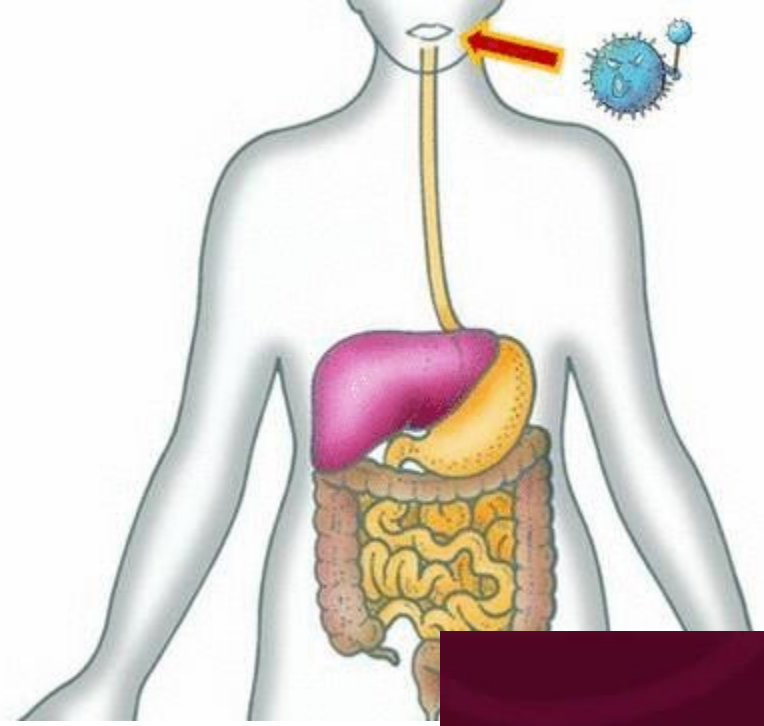
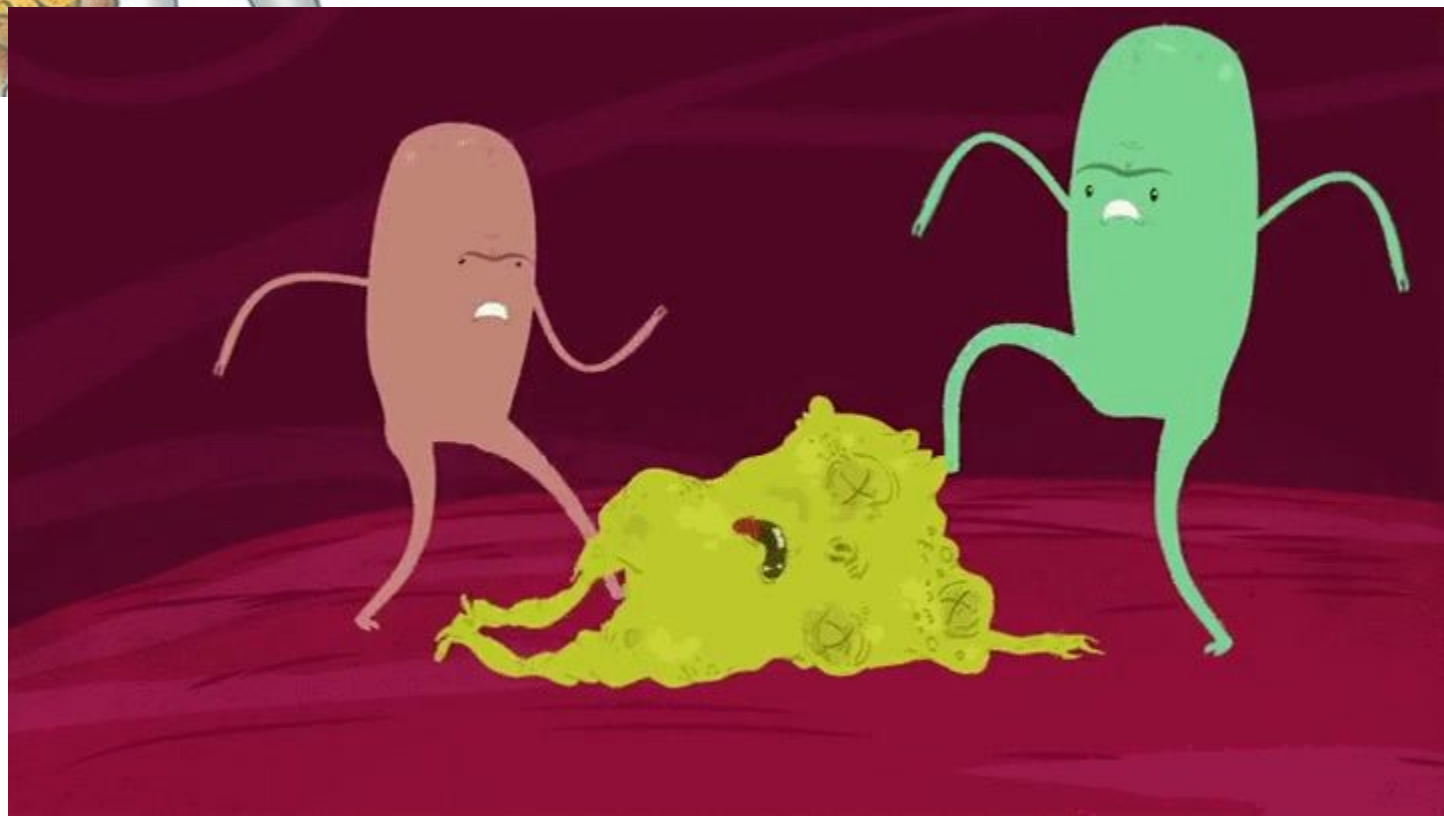
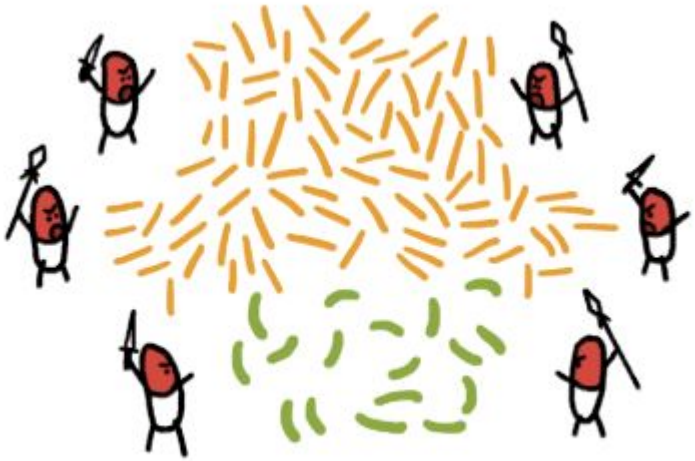
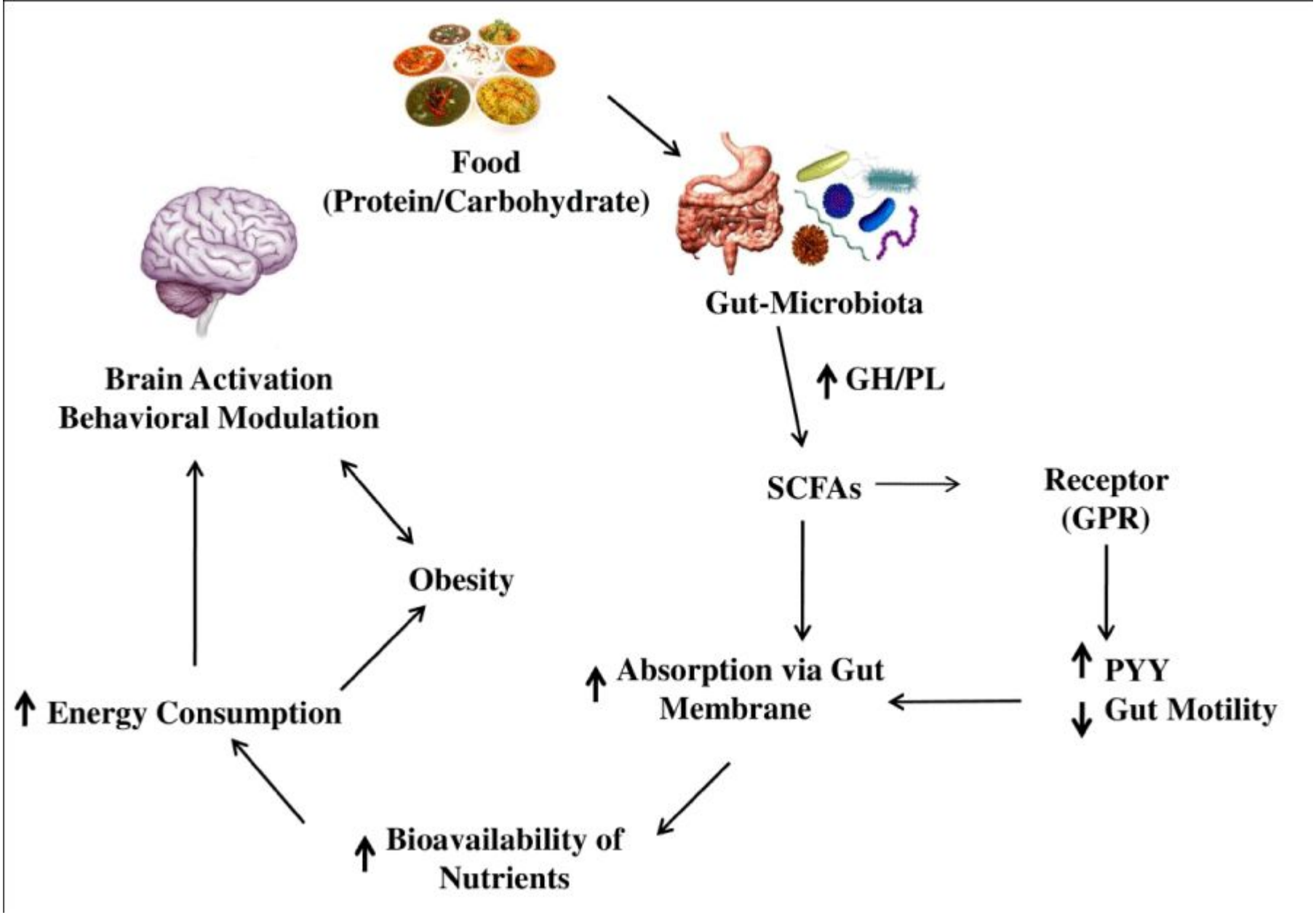


Noah Besser, Parker Street Animation









Microbial-host metabolism and the effect on behavioral function of brain. GH- Glycoside Hydrolases; PL- Polysaccharide Lysases; SCFAs-Short-Chain Fatty Acids; GPR- G-Protein Coupling Receptors; PYY- Peptide YY.

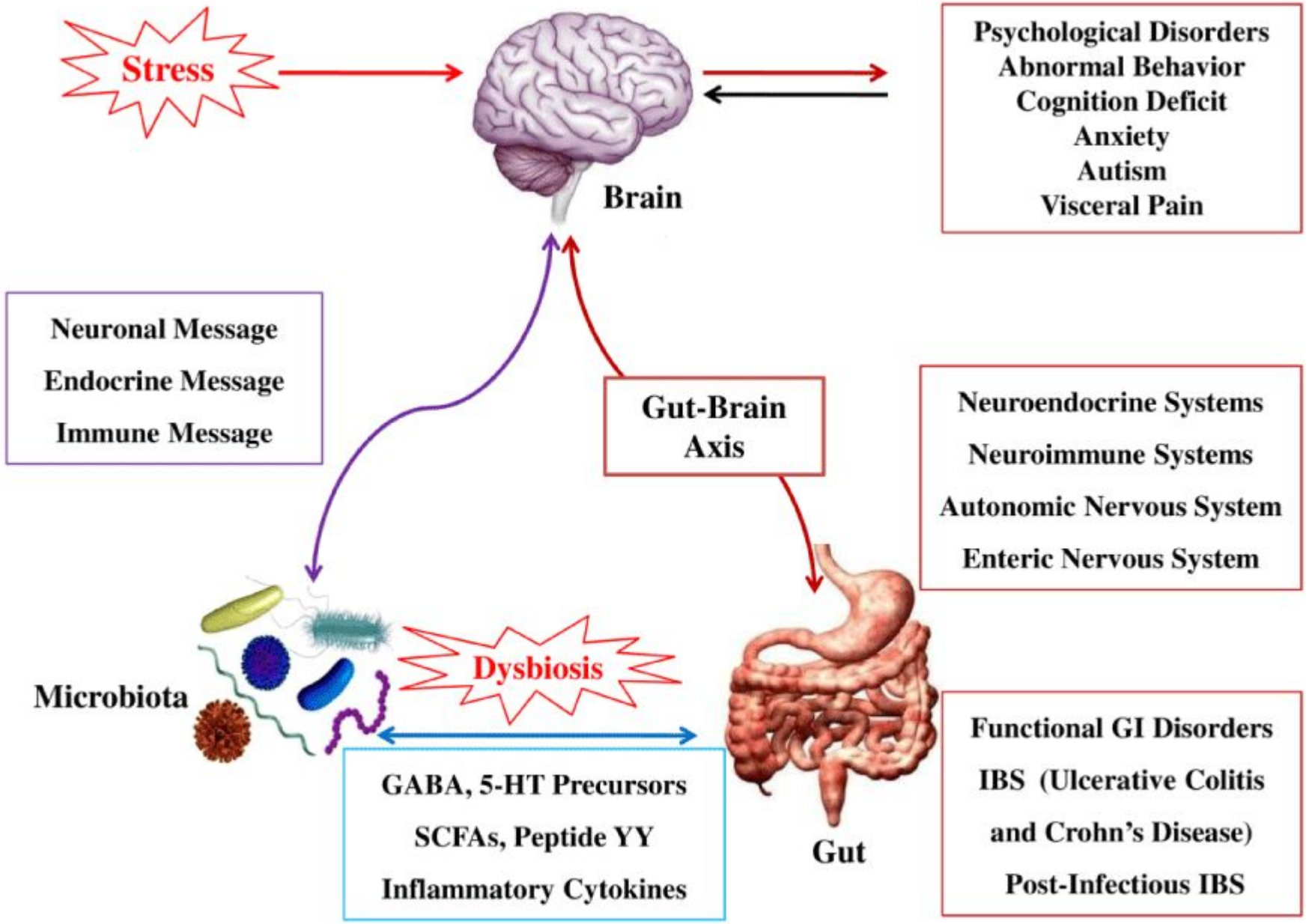

Stress
Brain
Psychological Disorders
Abnormal Behavior
Cognition Deficit
Anxiety
Autism
Visceral Pain
Neuronal Message
Endocrine Message
Immune Message
Gut-Brain Axis
Neuroendocrine Systems
Neuroimmune Systems
Autonomic Nervous System
Enteric Nervous System
Microbiota
Dysbiosis
Gut
GABA, 5-HT Precursors
SCFAs, Peptide YY
Inflammatory Cytokines
Functional GI Disorders
IBS (Ulcerative Colitis
and Crohn's Disease)
Post-Infectious IBS

Figure 2 Bidirectional communications between Gut-Microbiota and Gut-Brain Axis (GBA) in the modulation of the stress response. Microbiota communicate with the gut-brain-axis through different mechanisms viz. direct interaction with mucosal cells (endocrine message), via immune cells (immune message), and via contact to neural endings (neuronal message) to influence brain development and behavior. Stress through GBA effect on Gut-Microbiota which is responsible for functional GI disorders and dysbiosis. Similarly dysbiosis effect synthesis of several microbial by-product and precursor that gain access to the brain via the bloodstream and the area postrema, via cytokine release from mucosal immune cells, via the release of gut hormones such as 5-hydroxytryptamine (5-HT) from entero-endocrine cells, or via afferent neural pathways, including the enteric nervous system.