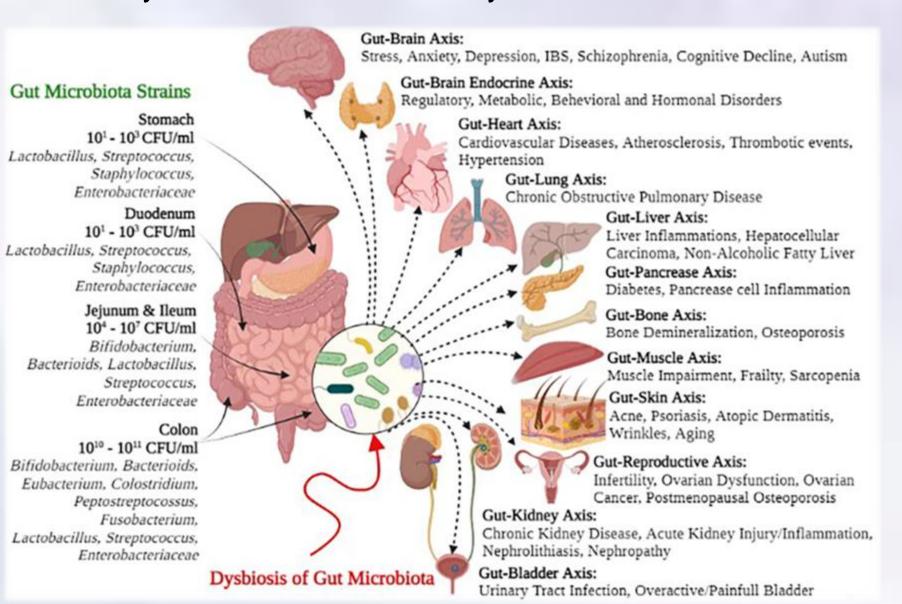
New era in medicine or dead end: can microbes save humanity?

Abstract

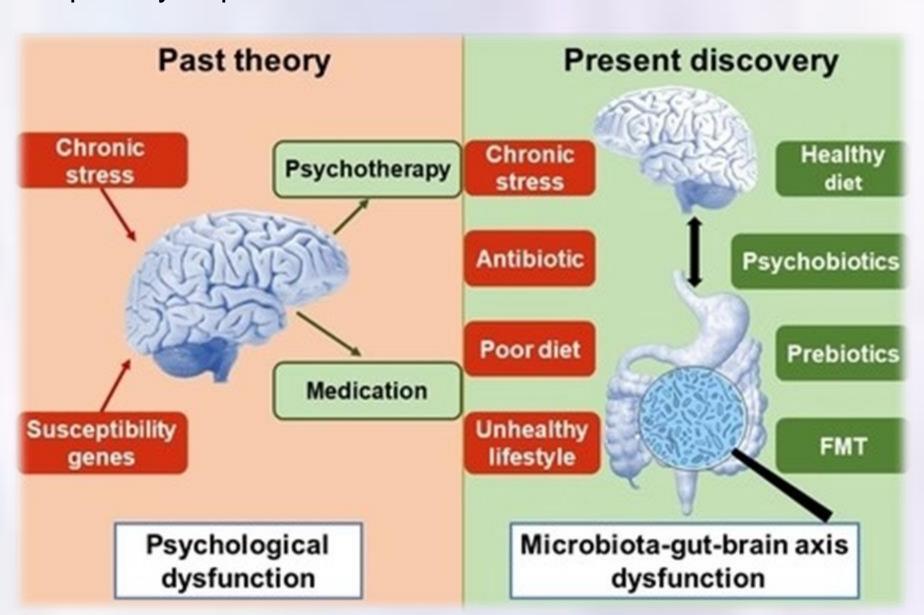
The search for a safe and effective cure for chronic diseases such as diabetes, obesity, and mental disorders has long been a goal of scientists. Recent research has shown that the interaction between microbiota and the body's immune system is a significant factor in the development or prevention of these diseases. The gutbrain axis also plays a critical role. Microbiota can influence the brain by producing neurotransmitters, enzymes, and other chemically active elements. Altering the species composition of microbiota or implementing a strict diet could potentially lead to a radical cure for chronic diseases by regulating important body functions such as im-munity and biochemical activity.



Pic. 1 Gut microbial strains and negative health outcomes of gut microbial dysbiosis. (Afzaal et al., 2022)

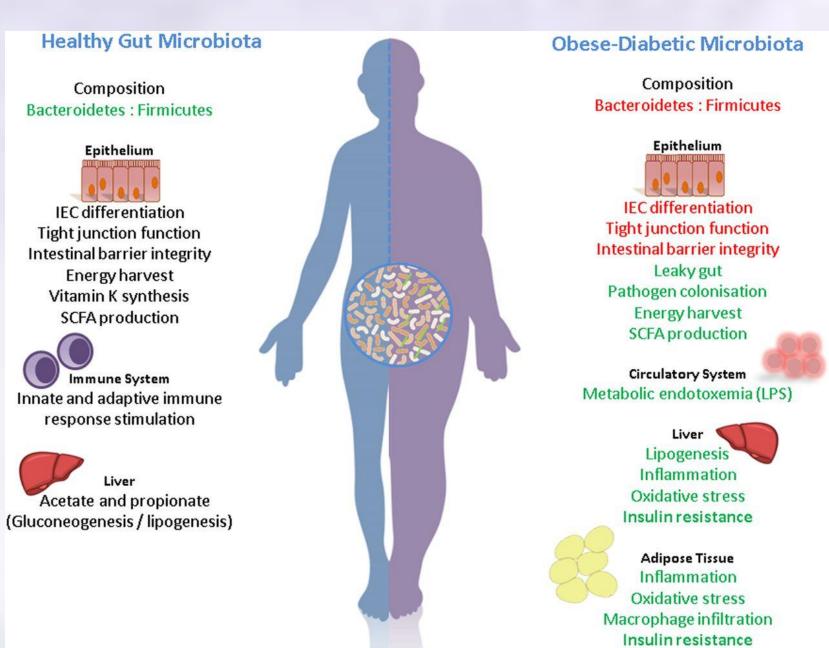
Introduction

The formation of intestinal microflora begins in the uterus and stabilizes around the age of three or four. Microbiota stimulates the newborn's immune system, contributes to the expansion of the "friend or foe" recognition system, and participates in the differentiation of natural killers and Thelpers. Also, the human body and billions of living organisms of the intestine interact actively and strive to maintain a balance to preserve their homeostasis. Therefore, any disturbance in the regulation and communication system between the microbiota and the body can be turned to being a part of pathogenesis for the development of many diseases, including the most socially important-diabetes, obesity, and mental disorders, especially depression.



Pic.2 Graphic differences of pathogenesis of depression(Liang et al. (2018)

- and diabetes.
- gut microbiota.
- diabetes.



Pic. 3 Compositional and functional alterations in the healthy gut microbiota versus the obese-diabetic microbiota. The metabolic processes in peripheral organs leading to increased adiposity, inflammation, oxidative stress, insulin resistance and lipogenesis are associated with the altered microbiota profile associated with the obese-diabetic phenotype. IEC, intestinal epithelial cell; LPS, lipopolysaccharide; SCFA, short chain fatty acid (Patterson et al. 2016).

This research paper is a result of summarizing, synthesizing, and critiquing about 95 scholar articles that have been discovered during a literature search. The criteria for inclusion were the study of the relationship of the microbiota with the human body and attempts to change it in humans and laboratory animals. Studies showing differences in microbiota species composition between healthy populations and those diagnosed with diabetes, obesity, and depression. Also, the criterion was the publication no earlier than 2019 for 90% of articles. Majority of the work show conclusion about promising breakthroughs in the future for the treatment of diseases that cause disability and death today. And at the same time, the researchers emphasize that this will take years, as the species diversity of the intestinal microbiota and its unique composition for each person requires further research.

Objectives

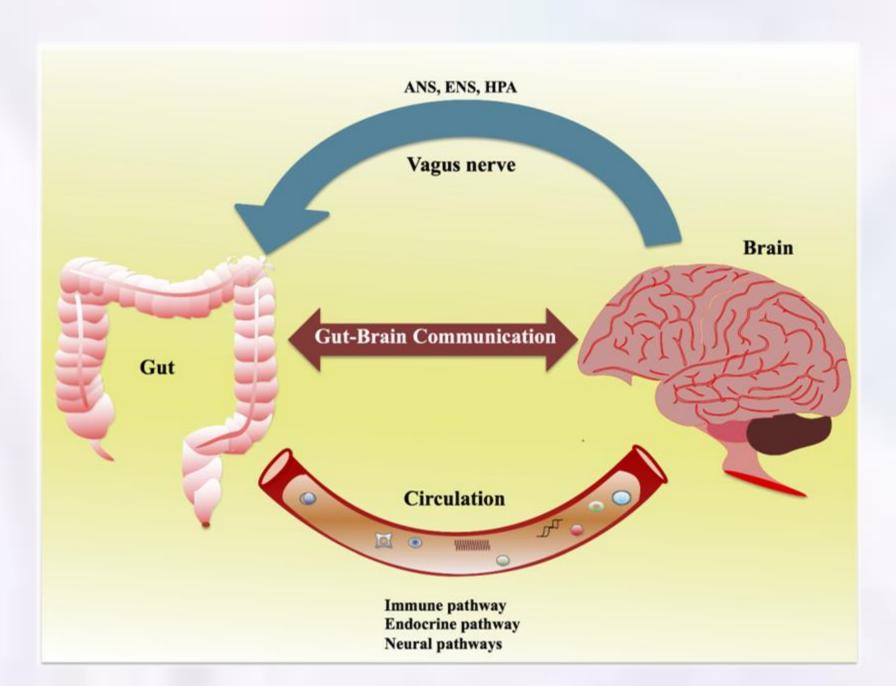
• Explore the potential role of the intestinal microflora in the pathogenesis of mental health disorders, obesity,

• Investigate the gut-brain axis and its connection to the

• Review the evidence linking alterations in the gut microbiota to mental health disorders, obesity, and diabetes can have curable effects.

• Evaluate the evidence show a connection between changes in the gut microbiota and positive(healing) effect on to mental health disorders, obesity, and

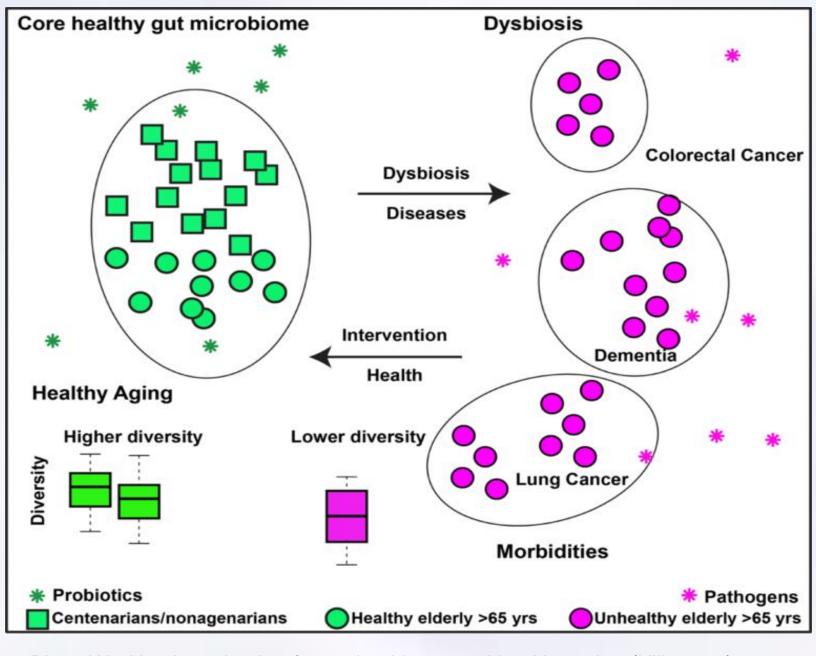
Methods



Pic.4 Schematic diagram showing the communication between the gut and brain. This is a bidirectio-nal relationship that is strongly influenced by multiple pathways, including the autonomic nervous system (ANS), enteric nervous system (ENS), hypothalamic-pituitaryadrenal (HPA), endocrine pathways immune pathways, and neural pathways. (Suganya & Koo, 2020)

Results

- The formation of intestinal microflora begins in the uterus and stabilizes around the age of three or four.
- Commensals stimulate the immune system of the newborn, contribute to the expansion of the "friend or foe" recognition system, and participate in the differentiation of natural killers and T-helpers.
- Interference with the balance of the regulation and communication system between microbiota and the body can contribute to the development of pathology.
- The gut-brain axis connects the intestine and the brain through a cascade of biochemical reactions and the parasympathetic nervous system.
- The gut microbiota can produce neurotransmitters like serotonin, SCFAs, and dopamine from tryptophan, affecting mood regulation, cognition, learning, memory, and motivation.
- A change in the quantitative and compositional balance of the flora can change the level of important brain function regulators and contribute to the development of depression.
- Managing the species composition of the intestinal microflora could potentially be a cure for chronic diseases like mental illness, diabetes, and obesity.



Student: Anastasia Ustinova Program / Major: Public Health

Pic. 5 Working hypothesis of gut microbiome and healthy aging (Hill, 2019).

Conclusion

Mental disorders, diabetes, and obesity are among the most significant health problems of the modern world. The delicate balance between the microbiota and the host plays an important role in the etiology and pathogenesis of these diseases. The gut-brain axis, which connects the microbiota and the brain, influences the production of neurotransmitters and regulates the immune response, thereby playing a crucial role in the development of mental disorders such as depression. Altering the composition of the microbiota through dietary interventions or fecal transplants can potentially improve the treatment of these diseases. However, more research is needed to fully understand the role of the microbiota in the development of these diseases and the effectiveness of microbiota-based interventions.



Pic. 6 A micrograph of various species of bacteria in human feces (Kalab et al. 2008).

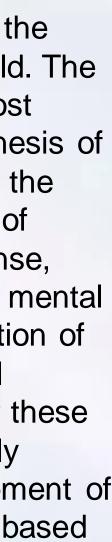
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