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Stress, Memory, Eating Behavior and Gastrointestinal Disorder

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Abstract

Intestinal function and dysfunction is a mysterious response linked to emotion, embarrassment, and shame. Perception of GI symptoms was assumed to be of different cause in every population. For example, a group of people considered it as hallucinations, whereas another group of people with lower socioeconomic status did not recognize GI clinical features as symptoms. However, modern studies suggest that diet, depression, stress, or anxiety can mutually trigger GI symptoms justified by the physiological, behavioral and psychosocial investigation of functional GI disorder (FGID). Other studies using emotion as stress on healthy subjects and subjects with IBS patients suggest that mood correlates with intestinal motility. For example, the increase and decrease in the intestinal motility were found to be associated with states of aggression and feeling of helplessness respectively. These studies, however, were limited by rudimentary measuring methods and unidirectional analysis approach.

Keywords: Hallucinations • Depression • Anxiety

Introduction

Another limitation was the failure to estimate the reciprocal effect of gut physiology on mental functioning. Further studies demonstrate that the gut and brain have a nervous system which is connected to each other and originate from the same embryonic neural crest, suggesting gut physiology responsive to emotional and stressful environmental stimuli. Brain-gut interactions reflects strong association between psychosocial and stress factors with intestinal function and dysfunction, GI symptoms and illness. Thus, a unified understanding of health and disease hypothesize the biopsychosocial and neurogastroenterology model which explain the relationship between stress, nutrition, and FGIDs via the brain-gut axis. The biopsychosocial model suggests that GI manifestations are the result of multi-level interactions between social, biological, and psychosocial subsystems; whereas neurogastroenterology reflects physiological and structural components of the biopsychosocial model [1].

Description

The concept of biopsychosocial model explains the clinical experience, pathogenesis, and effects of FGID, stating GI disturbance is the result of multi-level interactions between social, psychological, and biological subsystems. The model gives an upper hand to understand the illness that reconciles differences between clinical and biomedical observations, measures physiological integrity with patient's behaviour and perception, evaluates primary and secondary complications of chronic or acute GI symptoms other than death, and assess control for all the biopsychosocial variables using multivariate statistical methods for the development of treatment protocols.

Whereas, neurogastroenterology (the Brain-Gut axis) reflects the link between physiological and structural elements of the biopsychosocial model and outlines the clinical study and application. Findings suggest that gut microbiome which is significantly affected by diet, positively or negatively affects human health by disrupting intestinal immune and neural pathways via gut-brain axis. Short-term dietary consumption of plant or animal products rapidly alters the structure of the bacterial community in the gut producing inter individual variations in the expression of microbial genes. The bidirectional interaction between the resident gut microbiota and the brain not only influences certain brain functions and its behaviour and brain structures related to emotions but also affects the pathophysiology of mental illness [2].

The effective functioning of the microbiota-gut-brain axis is facilitated by the neuronal interactions of the efferent and afferent nerves involving the central nervous system (CNS), autonomous nervous system (ANS) parasympathetic and sympathetic branches, enteric nervous system (ENS), and neuroimmune and neuroendocrine pathways. Thus, GI microbiota plays a significant role in maintaining brain health. Intestinal microbiota has the ability to –

- Influence inflammatory reactions within the brain that modulate microglial cell activation in adult brains that affect neurogenesis and myelination.
- Indirectly or directly affect neuronal functions through neurotransmitters, vitamins and microbial neuromodulators such as short-chain fatty acids.
- Send signals to the brain using neuroendocrine and neuroimmune pathways to activate afferent sensory neurons of the vagus nerve.

Scientists suggests that patients with IBS exhibit abnormal brain activity in response to visceral pain stimulation in areas involved in endogenous pain modulation and pain processing (Mayer et al, 2009; Tillisch, Mayer & Labus, 2011). On further evaluation, cognitive function in IBS report that patients with IBS may be associated with both non-emotional visuospatial episodic memory and emotionally modulated cognitive changes mediated by hippocampus and amygdalar areas respectively. It was also noted that patients with IBS show attentional biases in response to negative valence words or stimuli related to GI symptoms suggesting consistent cognitive performance with a cognitive behavioural framework. Recent studies demonstrate that efferent and afferent nerves facilitate the neuronal interactions between the brain and GI tract. Mild hippocampal mediated visuospatial memory dysfunction and impaired cognitive flexibility in patients with IBS was explained probably due to HPA-axis functioning measured by cortisol awakening response [3]. Number of errors in the performance of memory was found to be increased with a decrease in the level of cortisol, acknowledging cognitive dysfunction associated with abnormally blunted or elevated cortisol levels. However, several clinical and preclinical studies report that dysregulation of HPA-axis negatively impacts hippocampal mediated cognitive performance suggesting an association between memory test performance and morning levels of cortisol. While other

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studies suggest that increase in levels of cytokines in patients with IBS and depression has an impact on cognitive performance [4,5].

In addition to this, IBS-induced brain functional modifications were not restricted to local changes but were also expressed at the fMRI stage. Recent Functional magnetic resonance imaging or functional MRI (fMRI) studies in IBS patients showed a decrease in amplitude of low-frequency fluctuation (ALFF) values in the right middle frontal gyrus, left superior frontal gyrus, right hippocampus, right superior temporal pole, and bilateral postcentral; while an increase in ALFF values in the left calcarine and median cingulate [6]. Analysis of functional connectivity also reveals enhanced connectivity in IBS patients between the frontal and cingulate cortex. The current study, therefore, aims at assessing visuospatial memory in people with GI symptoms mimicking IBS.

Although IBS pathophysiology is still unknown, studies often indicate IBS as a disease triggered by dysregulation of complex interactions along the gut- brain axis monitored by the microbiota. The gut microbiota consists of bacteriophages, bacteria, fungi, viruses, archaea, and protozoa; positively or negatively have an impact on human health. Further clinical and experimental studies illustrate that gut microbiota is significantly affected by diet. Research also suggests that, due to inter-individual variations in microbial gene expression, rapid changes in the gut microbiota in response to long-term or short-term plant or animal product consumption vary individually. Hence, the bidirectional communication between the brain and residual microbes of the GI tract play a vital role in maintaining human health. Moreover, studies also indicate that alterations in neuroendocrine-immune pathways due to stress intervene with the function of microbiota-gut-brain and gut-brain axis, causing flare-ups or exaggerations of the symptoms in IBS. Other studies demonstrate changes in the functioning of the hypothalamic-pituitary-adrenal (HPA) axis can be considerably affected by stress leading to dysregulation of normal gut-brain axis interactions. Several experimental studies also illustrate abnormal HPA-axis activity in IBS due to stress. Thus, considering all the suggestions from different studies, this study is similarly designed to assess the interaction between stress, diet, and GI symptoms that may trigger IBS [6].

Conclusion

The possible hypothesis of this study is - to understand the psycho-neurology of GI symptoms that may trigger IBS; find the correlation between GI symptoms, stress and diet; interlink between them; effect of stress and eating behaviour on manifestations of GI symptoms mimicking IBS and its impact on visuospatial memory.

Acknowledgement

Not applicable.

Conflict of Interest

There is no conflict of interest by the author.

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