Opinion
Volume 08:05, 2024

Research and Reports in Medical Sciences

ISSN:2952-8127 Open Access

Trauma, Memory, Eating Habits and Gastrointestinal Issues

Reamy Grace*

Department of Surgery, University of Arkansas for Medical Sciences, Arkansas, USA

Introduction

An enigmatic reaction connected to emotion, embarrassment, and shame is intestinal function and malfunction. Every population's perception of GI problems was thought to have a distinct source. For instance, some people thought it was hallucinations, while others from a lower socioeconomic background did not see GI clinical aspects as symptoms. Nonetheless, current research indicates that worry, stress, depression, and nutrition may all contribute to gastrointestinal symptoms, which is supported by the physiological, behavioral, and psychosocial analysis of Functional Gastrointestinal Disorder (FGID). Mood and intestinal motility may be related, according to other research that uses emotion as stress on both healthy and IBS-affected participants. For instance, it was discovered that emotional and aggressive moods were linked to changes in intestinal motility [1].

The inability to calculate the reciprocal impact of gut physiology on brain functioning was another drawback. Subsequent research reveals that the neurological systems of the gut and brain are interconnected and share an embryonic neural crest, indicating that the gut physiology is sensitive to stressful environmental inputs and emotions. Psychosocial and stress factors are strongly linked to intestinal function and malfunction, GI symptoms, and sickness, according to brain-gut interactions. Therefore, the biopsychosocial and neurogastroenterology model, which explains the connection between stress, nutrition, and FGIDs through the brain-gut axis, is hypothesized as a unified knowledge of health and disease. According to the biopsychosocial model, GI symptoms arise from multi-level interactions between biological, social, and psychological subsystems, while neurogastroenterology represents the structural and physiological aspects of the biopsychosocial model [2].

Description

By claiming that GI disturbance is the consequence of multi-level interactions between social, psychological, and biological subsystems, the biopsychosocial model explains the clinical experience, pathophysiology, and repercussions of FGID. By balancing the disparities between clinical and biomedical observations, measuring physiological integrity with patient behavior and perception, evaluating primary and secondary complications of acute or chronic gastrointestinal symptoms other than death, and assessing control for all biopsychosocial variables using multivariate statistical methods for treatment protocol development, the model provides an advantage in understanding the illness.

On the other hand, neurogastroenterology, or the Brain-Gut axis, describes the clinical research and application and illustrates the connection between the physiological and structural components of the biopsychosocial model. Results indicate that nutrition has a substantial impact on the gut microbiota, either favorably or unfavorably [3].

The Central Nervous System (CNS), Autonomous Nervous System (ANS), parasympathetic and sympathetic branches, Enteric Nervous System (ENS), neuroimmune and neuroendocrine pathways, and neuronal interactions of the efferent and afferent nerves facilitate the efficient operation of the microbiota-gut-brain axis. GI microbiome is therefore important for brain health maintenance. When visceral pain is stimulated, scientists believe that people with IBS display aberrant brain activity in regions related to endogenous pain modulation and pain processing. IBS patients may have both emotionally regulated cognitive changes mediated by the hippocampus and amygdalar regions, as well as non-emotional visuospatial episodic memory, according to cognitive function in IBS research. Additionally, attentional biases in responses were observed in IBS patients [4].

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. 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 [5].

Conclusion

In conclusion, this study highlights the complex interplay between gastrointestinal symptoms, stress, and dietary behaviors, which may contribute to the development or exacerbation of IBS-like manifestations. Stress and eating habits seem to significantly influence the severity of GI symptoms, creating a feedback loop that may further worsen both physical and psychological health. Moreover, the study suggests that these factors could also have a detrimental impact on cognitive functions, particularly visuospatial memory. Understanding these interconnections could provide valuable insights into the management of IBS and related conditions, potentially leading to more effective treatment strategies.

Acknowledgement

None.

Conflict of Interest

There is no conflict of interest by author.

References

1. McKee, Ann C., Robert C. Cantu, Christopher J. Nowinski and E. Tessa Hedley-Whyte, et al. "Chronic traumatic encephalopathy in athletes: Progressive tauopathy after repetitive head injury." *J Neuropathol Exp Neurol* 68 (2009): 709-735.
2. Goldstein, Lee E., Andrew M. Fisher, Chad A. Tagge and Xiao-Lei Zhang, et al. "Chronic traumatic encephalopathy in blast-exposed military veterans and a blast neurotrauma mouse model." *Sci Transl Med* 4 (2012).
3. Masson, Françoise, P. Maurette, L.R. Salmi and J. F. Dartigues, et al. "Prevalence of impairments 5 years after a head injury, and their relationship with disabilities and outcome." *Brain Inj* 10 (1996): 487-498.

***Address for Correspondence:** Reamy Grace, Department of Surgery, University of Arkansas for Medical Sciences, Arkansas, USA; E-mail: grace@23edu.com*

Received: 29 August, 2024, Manuscript No. rrms-24-155125; **Editor Assigned**: 31 August, 2024, PreQC No. P-155125; **Reviewed**: 14 September, 2024, QC No. Q-155125; **Revised**: 19 September, 2024, Manuscript No. R-155125; **Published**: 26 September, 2024, DOI: 10.37421/2952-8127.2024.8.186

4. Omalu, Bennet I., Steven T. DeKosky, Ryan L. Minster and Cyril H. Wecht, et al. "Chronic traumatic encephalopathy in a national football league player." *Neurosurg* 57 (2005): 128-134.

5. Ardila, Alfredo. "Normal aging increases cognitive heterogeneity: Analysis of dispersion in wais-iii scores across age." *Arch Clin Neuropsychol* 22 (2007):1003-1011.

How to cite this article: Grace, Reamy. "Trauma, Memory, Eating Habits and Gastrointestinal Issues." *Res Rep Med Sci* 8 (2024): 186.