Gastrointestinal Disorders, Memory, Eating Habits, and Stress are All Linked

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Abstract

Irritable Bowel Syndrome (IBS) affects one in every five people, and while there is no cure, there are strategies to manage the condition and reduce symptoms. Irritable Bowel Syndrome (IBS) is an insidious, variable condition with potentially humiliating symptoms. Diet, stress, anxiety, and depression are all potential causes of IBS, but they are mutually exclusive. The majority of IBS problems are multi-causal, meaning that multiple factors combine to generate symptoms. This retrospective study is primarily concerned with demonstrating the multifaceted nature of gastrointestinal illnesses (specifically IBS). The goal is to see if there is any link between irritable bowel syndrome, stress, eating habits, and memory.

In this study, a symptomology questionnaire is used to determine the type of gastrointestinal problem based on symptoms, two sets of questionnaires are used to assess stress levels and eating patterns and a Paired Association Learning (PAL) test is used to estimate short-term memory in CANTAB. The study's possible hypothesis is to find a positive link between GI diseases (especially IBS), stress, eating behavior, and short-term memory, as well as to analyse and show the strength of linkage and its influence on immunity

Keywords: Gastrointestinal disorders • Irritable bowel syndrome • Biopsychosocial

Introduction

The inexplicable reaction of intestinal function and dysfunction is linked to emotion, embarrassment, and shame. Every population's perception of GI problems was considered to be due to a different aetiology. For example, one group thought it was hallucinations, while another group of those with lower socioeconomic position did not recognise GI clinical signs as symptoms. Modern research suggests that diet, depression, stress, or anxiety can all cause GI symptoms, which is supported by physiological, behavioural, and psychosocial studies of Functional GI Disorder (FGID). Other studies that used emotion as a stressor on healthy and IBS patients suggest that mood is linked to intestinal motility. Increases and decreases in intestinal motility, for example, were found to be linked to emotions of aggression and powerlessness, respectively. Rudimentary measuring methods and a unidirectional analysis strategy, however, hampered these investigations [1-3].

The failure to assess the reciprocal influence of gut physiology on brain performance was another flaw. Further research shows that the stomach and brain share a neurological system that is linked to one another and stems from the same embryonic neural crest, implying gut physiology responsive to emotional and stressful environmental inputs. Interactions between the brain and the gut reveal a substantial link between psychological and stress factors and intestinal function, malfunction, GI symptoms, and illness. As a result, the biopsychosocial and neurogastroenterology models, which explain the link between stress, nutrition, and FGIDs via the brain-gut axis, are hypothesised as part of a holistic view of health and disease. The biopsychosocial model proposes

That GI symptoms are the outcome of multi-level interactions between social, biological, and psychological subsystems, while neurogastroenterology represents the biopsychosocial model's physiological and structural components.

The effect of stress in the modulation of the most prevalent gastrointestinal illnesses has long been regarded a province of psychology, and the involvement of mental comorbidity has usually been bundled together. The term "stress" is commonly used by physicians to refer to psychological ("exteroceptive") stress. Stress and psychological elements have been deemed essentially independent and unconnected to the "true" biological changes producing organic disease, according to the firmly engrained Cartesian approach in medicine and gastroenterology. Recent advances in our understanding of the neurobiology of the organism's response to acute and chronic stress, as well as our growing understanding of complex brain-gut interactions and how they are modulated in health and disease, are prompting a rethinking of chronic stress's pathophysiology and management.

Model of the Biopsychosocial

The biopsychosocial model, which states that GI disturbance is the outcome of multi-level interactions between social, psychological, and biological subsystems, explains the clinical experience, pathophysiology, and effects of FGID. The model provides an advantage in understanding the illness by reconciling differences between clinical and biomedical observations, measuring physiological integrity with patient behaviour and perception, assessing control for all biopsychosocial variables using multivariate statistical methods for the development of treatment protocols, and evaluating primary and secondary complications of chronic or acute GI symptoms other than death.

Neurogastroenterology

The relationship between physiological and structural parts of the biopsychosocial model is reflected in neurogastroenterology (the Brain-Gut axis), which describes the clinical investigation and application. The findings imply that the gut microbiota, which is influenced by nutrition, has a favourable or negative impact on human health through affecting intestinal immunological and neurological processes through the gut-brain axis. The organisation of the bacterial population in the gut is rapidly altered by short-term dietary ingestion of plant or animal items, resulting in interindividual differences in microbial gene expression. The pathophysiology of mental disease is influenced by the bidirectional interaction between the resident gut microbiota and the brain, which impacts not only certain brain processes, behaviour, and brain structures associated to emotions, but also the pathophysiology of mental illness.

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 facilitate the effective functioning of the microbiota-gut-brain axis. As a result, the GI microbiota plays an important role in brain health [4]. The microbiota in the intestine has the power to-

- Modulate inflammatory reactions in the brain that impact neurogenesis and myelination by modulating microglial cell activation in adult brains
- Neurotransmitters, vitamins, and microbial neuromodulators such short-chain fatty acids have an indirect or direct effect on neuronal functioning
- Activate afferent sensory neurons in the vagus nerve by sending messages to the brain via neuroendocrine and neuroimmune pathways

Memory effects of a dysfunctional gut-brain axis

According to scientists, patients with IBS have aberrant brain activity in areas involved in endogenous pain modulation and pain processing in

response to visceral pain stimuli. Patients with IBS may be associated with both non-emotional visuospatial episodic memory and emotionally modulated cognitive changes, mediated through the hippocampus and amygdalar regions, according to a study on cognitive performance in IBS. Patients with IBS also exhibit attentional biases in reaction to negative valence phrases or stimuli associated with GI symptoms, implying consistent cognitive performance with a cognitive behavioural paradigm. The neural interactions between the brain and the GI tract are facilitated by efferent and afferent nerves, according to recent research. Mild hippocampal-mediated visuospatial memory impairment and poor cognitive flexibility in IBS patients were attributed to HPA-axis functioning as indicated by the cortisol awakening response. The number of memory errors was observed to increase with a reduction in cortisol levels, indicating that cognitive impairment is linked to abnormally muted or raised cortisol levels. Several clinical and preclinical research, however, have found that dysregulation of the HPA-axis has a deleterious impact on hippocampal-mediated cognitive function, implying a link between memory test performance and morning cortisol levels [5]. Other research suggests that elevated cytokine levels in patients with IBS and depression have an effect on cognitive performance.

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Stress response: Homeostasis defence at the expense of allostasis

Stress, defined as acute challenges to an organism's homeostasis, whether real (physical) or perceived (psychological), and whether posed by external or internal events, elicits adaptive reactions that serve to safeguard the internal environment and ensure the organism's existence. Surprisingly, despite the great variety of stressors, some of the main circuits underpinning the stress response under these many conditions are very similar.

Exteroceptive stressors (psychological) engage circuits in the limbic forebrain, including the lateral and medial prefrontal cortex, hippocampus, and amygdala. While the pathways involved in the activation of hypothalamic effector neurones during interoceptive stressors (gut infection, mucosal inflammation, internal haemorrhage) may be conceived as simple reflex responses, mediated at a subcortical level by the Cortical circuits are involved in modifying the stress response to the context, the physiological state of the organism, memories of previous stressful life events, and thoughts about the situation's subjective meaning. Complex neurobiological response systems have developed to orchestrate an integrated response that is best suited to respond to a specific stressor in a specific environment for a specific person. Allostasis is the ability to defend homeostasis (that is, to maintain stability) in the face of change. The physiological response systems of a healthy person are rapidly turned on and off, synchronising the physiological stress response to the length of the stressor and reducing the organism's exposure time to the stress response's potentially damaging effects. However, the degree or chronicity of the stressor, as well as the accompanying physiological response systems, can inflict damage, exacerbate current disease processes, or predispose the individual to develop new diseases-that is, this is especially true in situations where an individual's susceptibility to the negative effects of stress has already been influenced by genetic or early life events that have altered the responsiveness of physiological responses to stress and the ability to adapt, thereby biassing an individual's susceptibility to the negative effects of stress throughout life. Allostatic load, or the "wear and tear" caused by chronic overactivity or underactivity of physiological stress response systems, has been coined to describe the long-term impacts of the organism's tolerance to particular types of stress. Stressors that have been linked to such maladaptive outcomes, both acute and chronic, are referred to as pathological stressors [7]. The patient's response to pathological stress is influenced by a variety of factors, including genetics, early life experiences, cognitive factors, and contextual support, in addition to the length, severity, and type of stressor.

Allostasis regulates physiological stress reactions

This network's responsiveness and output pattern are considered to be partially genetically controlled, and it exhibits significant flexibility in response to early life events and certain types of pathological stress. For example, studies in animals and humans have clearly demonstrated that pathological stress can alter the responsiveness of feedback systems by downregulating pre and/or postsynaptic receptors (adrenergic, serotonergic, and GC receptors), as well as structural changes in certain brain regions in the most severe cases [8]. As a result, pathological stress can not only activate but also fundamentally alter the central stress circuits' responsiveness and output. Increases or decreases in target specific sympathetic outputs, increases or decreases in certain vagal outputs, up or downregulation of the HPA axis, and up or downregulation of pain perception are just a few examples of how these changes could affect the individual output pathways of the general stress response in different ways. An increase in CRF synthesis and secretion, an increase in the activity and sensitivity of central noradrenergic systems, and either downregulation or sensitisation of GC receptors and adrenocorticotropic hormone release are some of the most well-studied changes in this central adaptation to pathological stress. Secondary modifications in receptor systems in spinal or peripheral target cells of the output systems might arise as a result of these changes in the central stress circuitry [9]. As a result, life-long modifications in peripheral receptor systems may be expected in situations of pathological stress resulting in irreversible changes in the central stress circuitry. Finally, modifications in mood and affect have been linked to changes in the stress response.

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