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Link between Gastrointestinal Disorder, Memory, Eating Behaviour and Stress

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Abstract

One in five people is affected by irritable bowel syndrome (IBS), and while there is no definitive treatment, there are ways to take control of the condition and minimize symptoms. IBS is an invisible, fluctuating disease with potentially embarrassing symptoms. The possible causes of IBS such as diet, stress, anxiety, depression are mutually exclusive. Most of the conditions of IBS are multi-causal and causes interact together to trigger symptoms. This retrospective study mostly focuses on illustrating the multi-dimensional view of gastrointestinal disorders (specifically IBS). The aim is to determine the association of irritable bowel syndrome, stress, eating behaviour and memory. In this study, a symptomology questionnaire is designed to assess the type of gastrointestinal disorder based on symptoms, 2 sets of questionnaires are used to measure the level of stress and pattern of eating behaviour, and paired associate learning (PAL) test is conducted in CANTAB to estimate short-term memory. The possible hypothesis for the study is to find a positive interlink between gastrointestinal (GI) disorders (particularly IBS), stress, eating behaviour and short-term memory, as well as to assess and illustrate the level of association and its effect on immunity.

Keywords: Gastrointestinal Disorder • Memory • Eating Behavior • Stress

Introduction

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 [1]. 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, behavioural 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 [2]. 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. 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 [3].

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 [4]. 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 [5].

Biopsychosocial model

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 [6]. 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 (Figure1) [7].

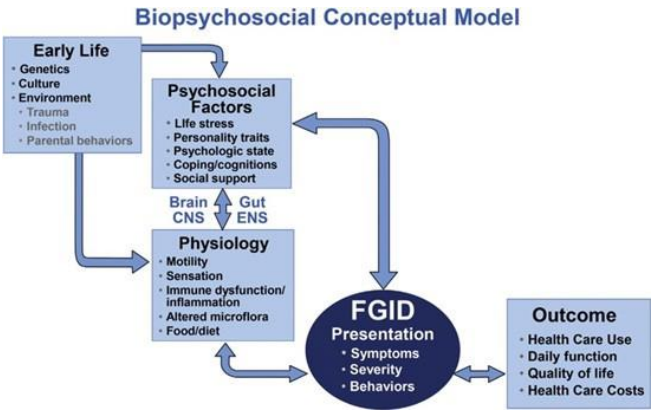

Figure 1. Biopsychosocial Model.

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Neurogastroenterology

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 [8]. 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 [9]. 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 (Figure2) [10].

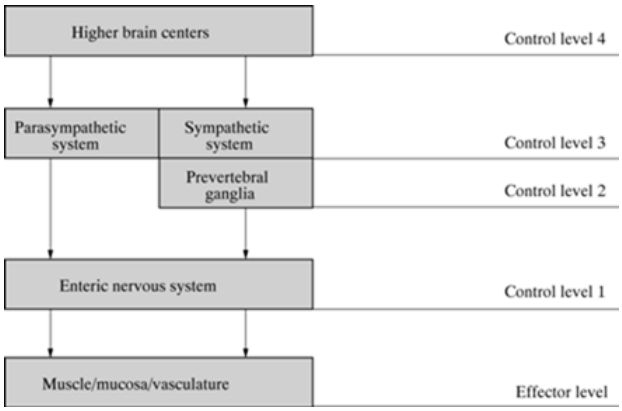

Figure 2. Neurogastroenterology.

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 (Figure 3) [11]. 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.

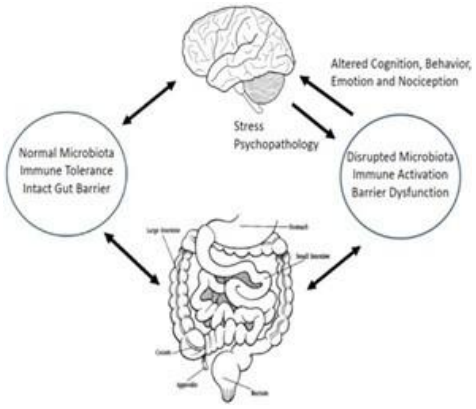

Figure 3. Gut-Brain Axis and the Microbiome.

Impact of the gut-brain axis dysfunction on Memory:

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 [12]. 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 [13]. 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 [14]. Recent studies demonstrate that efferent and afferent nerves facilitate the neuronal interactions between the brain and GI tract [15]. 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. 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 [16]. 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 studies suggest that increase in levels of cytokines in patients with IBS and depression has an impact on cognitive performance [17].

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 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 [18]. 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 [19].

Hypothesis

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 [20]. 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 [21].

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 [22]. 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 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 [23].

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 [24].

Method

Participants

Voluntary participants (n=72) aged between 18-90 years old were invited to participate in the study by handing out flyers and posting a digital image of this flyer on social media. Based on the eligibility criteria participants were grouped into Control, healthy and Clinical, people with GI symptoms mimicking IBS or patients diagnosed with IBS [25].

Eligibility Criteria:

1) Inclusion

- Clinically diagnosed for irritable bowel syndrome
- People who experience 1 or more symptoms of irritable bowel syndrome
- Normal or corrected vision

2) Exclusion

- The presence of any neurological disease
- GI symptoms due to any surgical intervention
- Clinically diagnosed with other GI disorder
- Visual defect
- History of Learning/Reading Disorder
- Drug abuse or alcohol abuse
- Psychological illness.

The exclusion criterion was designed to rule out all the factors that may affect the reliability and validity of the study [26]. Considering that the participants have to answer questionnaires in written form and work on a digital memory task, participants with learning or reading disorders, visual defects other than corrected vision (e.g., colour blindness), drug or alcohol abuse, and history of neurological disease and psychological illness were not included [27].

Design

The current study is designed to determine statistically significant evidence between the two independent groups (control and clinical group) among the associated population [28].

The independent variable is clinical status of an individual and the dependent variables are the psychometric measures [28].

Method

This research was carried out by adapting three sets of questionnaires symptomology questionnaire, Dutch Eating Behaviour Questionnaire (DEBQ) and Perceived Stress Scale (PSS), and digital visuospatial memory test [29].

Symptomology questionnaire

The 12-item symptomology questionnaire was designed based on NHS guidelines for assessing the clinical condition of GI manifestations. This questionnaire involves leading questions about the present and past medical history of the participant related only to the GI system, such as What symptoms you have?; If they come and go?; How often you get them?; How long you've had them?; and When you get them (for example, after eating certain foods)? [30]. The participants were also asked about any previous health issues to rule out the exclusion criteria with direct questions, such as Are you clinically diagnosed with any gastrointestinal disorder?; Do you have any neurological disease?; Do you have any surgical history?; Do you have any visual defect?; Do you have any history of learning/reading disorder?; History of drug abuse or alcohol abuse?; and Do you have any psychological illness. Qualitative analysis of this questionnaire is done to allocate the participants to either control or clinical group [31].

Dutch Eating Behaviour Questionnaire (DEBQ)

On one side of the questionnaire is 33 item predesigned Likert scale to assess the rough scores of scales and subscales of eating behaviour. Participants were given five options for each item-never, rarely, sometimes, often or very often to correctly rate the questions in a way that corresponds to them. These questions assess the pattern of eating behaviour in relation to stress, fear, anxiety, worry, emotion, mood, temptations, and perception or conscience about one's personality. For example, do you have the desire to eat when you are irritated? If you put on weight, do you eat less than you usually do? If you see or smell something delicious, do you have a desire to eat it? And Do you have a desire to eat when you are frightened? This questionnaire measures scores on the scales such as diffuse emotion, clearly labelled emotions, emotion eating, external eating, and restrained eating. The other side of the questionnaire is consists of demographic details (e.g., age, sex, height and weight) [32].

Perceived Stress Scale (PSS)

The most commonly used 14 item stress scale to estimate the level of perceived stress within time period of one month (Cohen, 1988; Lee, 2012). Participants were asked to rate their feelings and thoughts felt during last month on a scale of 0-4 stating never, almost never, sometimes, fairly often, and very often respectively. For example, in the last month, how often have you been upset because of something happened unexpectedly? ; How often have you felt that things were going your way? ; And how often have you been able to control irritations in your life. Participants were also made aware to answer each item fairly quick to avoid the counting of the number of times they felt a particular way and alternate it with a reasonable estimate [33].

Cambridge Neuropsychological Test Automated Battery (CANTAB)-Paired Associate Learning (PAL) test

CANTAB, an automated battery of neuropsychological tests, is predesigned to assess the executive and cognitive functions of an individual [34]. In this study, the 8-pattern Paired Associate Learning (PAL) test is used to measure the accuracy and speed of response as to estimate an individual's visuospatial memory [35]. In this test, square-shaped boxes open and close randomly within 500ms in a pattern to show the visual icons hidden in it [36]. The task is to observe and remember the visible location of the object/s on the screen and select the pattern in the order requested by the battery [37]. The type of PAL test is selected for this study is an 8-pattern (Figure 1) [38]. To make it easy yet challenging for all age groups within a range of 18-90 years old. Ideally, this test is chosen as an opt way to measure the visuospatial memory because-

- It demands less time (approximately 8-10 mins).
- It combines the visual and spatial information which requires the functioning of hippocampus.
- The test is often taken as a rewarding game after answering the series of questionnaires.
- The battery gives a digitalised summary report of the test for each participant [39].

This sensitive test assesses list learning, new learning, and list memory. Studies indicate that PAL has demonstrated delicate modifications in the function of hippocampal brain areas and involves a front-parietal network

during encoding phases and posterior cingulate and left cuneus areas during recovery phases [40].

Procedure

Participants were initially given the information sheet to inform them about the details of the current study followed by a consent form informing them that the study is non-invasive, simple questionnaire-based and holds anonymous data collection [41]. Upon voluntary participation, they were asked to answer a series of questionnaires symptomology questionnaire, Dutch Eating Behaviour Questionnaire, and Perceived Stress Scale followed by short-duration memory test [42]. Participants were given the debriefing sheet and supporting material (if needed) after the study to make them aware of the dimensions of irritable bowel syndrome and navigate their way towards recovery with better understanding [43].

Ethics

This study has been approved by the Psychology Research Ethics Panel [44]. Participants had the right to change their decision from the moment of entering the room until stepping out of the chamber after data collection, but participant cannot retrieve the data after submitting the questionnaires and task as the data collected is anonymous [45]. If the participants seem to have any negative impact, they have full rights to withdraw from participation any time before the completion of the tasks [46]. Participants can also ask to remove the data before moving out of the room with or without any explanation [47]. All data collected was confidential and anonymous [48]. Any physical copies of the information provided were kept in a secure filing cabinet, and digital data was stored within an encrypted folder [49]. All data was only accessed by the researcher and their supervisor. Data from the whole study was then assessed and published as the result of analysis in the final report [50].

Results

Before commencing data collection, assuming þ=0.05, G-power calculated to attain ≥0.8 effect size (δ) and 90% of power to correctly reject the null hypothesis indicated a requirement of a minimum of 34 participants in each group. Participants (N=72) were randomly selected from the population based on the inclusion criteria and divided into two categories a group with symptoms of GI (N=36) and control group (N=36) [51]. The raw data was then scrutinized for missing data and reverse scores to prepare refined data for statistical analysis [52].

Missing Data

Instead of excluding the complete datasheet of the participant, the missing data were replaced by an assumption to secure the target size of the sample. Among 72 participants, 6 participants skipped randomly one of the questions in the 33-item Dutch Eating Behaviour Questionnaire [53]. So, the mean value of the scores of the other 32 answers was replaced as the missing value for one of the questions [54].

Scoring Data

Symptomology questionnaire

Each intestinal symptom and extra-intestinal symptom was provided 2 and 1 point respectively on the grounds of ROME IV criteria [55]. Overall scores of all symptoms were calculated under the severity score [56]. Based on the severity rating, only respondents who scores<2 were included in the control group [57].

Dutch Eating Behaviour Questionnaire (DEBQ)

For this questionnaire, each response is given a value based on the Likert scale (1-5) to calculate the outcome for the following subscales — diffuse emotions, clearly labelled emotions, emotional eating, internal eating, and restrained eating [58].

Perceived Stress Scale (PSS)

The scoring of 14-item perceived stress scale is also based on the Likert scale (from 0-4) [59]. But, the order of scoring is reversed for 7-items (4, 5, 6, 7, 9, 10 and 13) to preserve the reliability of the scale [60]. After recording the scale with reverse scores, the total sum of scores is calculated to estimate the numerical value of the stress level perceived by an individual [61].

CANTAB-PAL test

In this scale, the sensitive visuospatial memory changes are recorded in the form of the average time taken to respond to the stimuli (response time), the number of stages completed in the first trial, and the summary report of total errors adjusted (i.e., raw score, standard score, and performance percentile) as suggested by the Cambridge Cognition Support Team [62]. Performance score is calculated for every range of performance percentile with a common difference of 5 was given a value from 1-20 as the percentile increases from 0%-5% to 95%-100% to feasibly analyse the memory performance scale during the task [63].

Statistical Analysis

The control group (N=36) was associated with severity score M=0.19 (0.4), p<.001; clearly labelled emotions 14.2 (5.86), p<.001; emotional eating 19.9 (7.80), p<.001; response time

2641 (2113), p<.001; raw score 13.3 (12.4), p<.001; standard score 0.45 (0.47),p<.001; performance score 13.7 (5.64), p<.001; stages completed in the first trial 5.94 (1.09), p=.001; BMI 27.1 (6.24), p=.03; diffuse emotions 8.02 (3.32), p=.01; restrained eating 21.4 (8.32),

p=.04; external eating 27.5 (7.16), p=0.27 and stress score 23.3 (9.15), p=0.69. By comparison, the clinical group (N=36) was associated with numerically smaller BMI 25.1 (5.44), p>.001; response time 2498 (1654), p>.001; raw score 11.2 (8.8), p=0.01 and standard score 0.53 (0.34),p=0.02 of total errors adjusted, and numerically larger severity score 5.58 (4.28), p<.001; diffuse emotions 10.4 (2.97), p=0.29; clearly labelled emotions 19

(7.84), p=.006; emotional eating 26.5 (9.47), p=0.03; external eating 31 (5.35), p=0.42;

restrained eating 23.9 (8.4), p=0.49; stress score 28.5 (6.88), p<.001 and performance score

14.2 (4.49), p=0.04. Whereas, the clinical group showed no numerical difference when compared to control group in regards of stages completed in the first trial 5.94 (1.04), p=.004 [64]. Furthermore, in comparison to the control group, the mean value of BMI was within the normal range for both males (M=25.4, SD=4.86) and females (M=24.7, SD=6.08) and age group exhibiting GI symptoms were 30-35 years (i.e., males (M=33, SD=8.81) and females (M=30.4, SD=10.4) [65].

An independent t-test was conducted to test the hypothesis that the control and clinical group were associated with statistically significantly different mean variables [66]. As can be seen in Table 1 (a-Control group; b-Clinical group), the control and clinical group distributions were sufficiently normal for the purpose of performing a non-parametric independent t-test (i.e., Skew<|3.3| and Kurtosis<|10.8|) [67].

Table 1a. Descriptive statistics: Control group.

Variables	Control Group (N=36)				
	Mean(M)	SD	Skewness	Kurtosis	Shapiro- Wilk p
Severity Score	0.19	0.4	1.61	0.63	<.001
BMI	27.1	6.24	0.89	0.38	0.03
Diffuse Emotion	8.02	3.32	0.63	-0.44	0.01
Clearly Labelled Emotions	14.2	5.86	1.18	0.61	<.001
Emotional Eating	19.9	7.8	1.07	0.33	<.001
External Eating	27.5	7.16	0.07	-0.66	0.27
Restrained Eating	21.4	8.32	0.42	-0.79	0.04
PSS total score	23.3	9.15	-0.15	-0.53	0.69
Response time	2641	2113	3.3	10.8	<.001
Stages completed in first trial	5.94	1.09	-0.3	-0.94	0.001
Raw score	13.3	12.4	1.1	0.06	<.001
Standard score	0.45	0.47	-1.18	0.23	<.001
Performance score	13.7	5.64	-0.67	-0.99	<.001

Note: **p<.01, **p<.001

Table 1b. Descriptive statistics: Clinical group.

Variables	Clinical Group (N=36)				
	Mean(M)	SD	Skewness	Kurtosis	Shapiro-Wilk p
				4.22	<.001
	Mean(M)	SD	Skewness	Kurtosis	Shapiro-Wilk p
Diffuse Emotion	10.4	2.97	0.59	0.88	0.29
Clearly Labelled Emotions	19	7.84	1.03	0.76	0.006
Emotional Eating	26.5	9.47	0.93	1.08	0.03
External Eating	31	5.35	0.36	-0.36	0.42
Restrained Eating	23.9	8.4	0.51	-0.18	0.49
PSS total score	28.5	6.88	0.44	-0.63	0.24
Response time	2498	1654	2.65	6.98	<.001
Stages completed in first trial	5.94	1.04	-0.21	-0.16	0.004
Standard score	0.53	0.34	-0.98	0.74	0.02
Performance score	**14.2**	**4.49**	**-0.54**	**-0.62**	**0.04**

Note: **p<.01, **p<.001

Moreover, correlation matrix displayed positive and strong association between severity score, diffuse emotions, clearly labelled emotions, emotional eating and stress [68]. However, weak associations were also noticeable between diffuse emotion, emotional eating and memory (standard score of total errors adjusted) and BMI, restrained eating and memory (stages completed in first trial) (Table 2) [69].

Table 2. Positive correlation between different variables.

a.Variables	Diffuse emotion	Clearly labelled	Mean(M)	Mean(M)
Severity score	Rho=.35, p=.003	Rho=.37, p=.001	Rho=.38, p=.001	Rho=.25, p=.03

b.Variables	Restrained eating	Stages completed in first trial
BMI	Rho=.28, p=.01	Rho=.23, p=.05

c. Variables	Diffuse emotion	Clearly labelledemotions	Emotional eating
Stress score	Rho=.40, p=<.001	Rho=.34, p=.004	Rho=.38, p=<.001

D. Variables	Diffuse emotion	Emotional eating
Standard score	Rho=.26, p=.03	Rho=.23, p=.05

Note: *p<.01, **p<.001

Additionally, Levene's F test was performed to test and satisfy the assumption of homogeneity of variances,

Severity score, F (1)=25.2, p=<.001* BMI, F (1)=1.63, p=0.20

Diffuse emotions, F (1)=1.16, p=0.28

Clearly labelled emotions, F (1)=1.27, p=0.26 Emotional eating, F (1)=0.45, p=0.51

External eating, F (1)=2.92, p=0.09

Restricted eating, F (1)=0.07, p=0.79

Stress score, F (1)=0.71, p=0.40

Response time, F (1)=0.17, p=0.68

Stages completed in the first trial, F (1)=1.03, p=0.31 Total errors adjusted:

Raw score, F (1)=5.47, p=0.02* Standard score, F (1)=4.81, p=0.03* Performance score, F (1)=3.87, p=0.05 (*equality of variance not assumed)

The Mann-Whitney U test was associated with a statistically significant affect illustrating 4, 3, 5, 7, 4, and 5 points difference for severity score, diffuse emotions, clearly labelled emotions, emotional eating, external eating and stress score respectively as follows:

Severity score, U=0, p=<.001, 95% CI (-5.0) – (-3.0), r=1.8

Diffuse emotions, U=369, p=.002, 95% CI (-4.0) – (-1.0), r=0.8

Clearly labelled emotions, U=382, p=.003, 95% CI (-7.0) – (-1.0), r=0.7

Emotional eating, U=361, p=.001, 95% CI (-10.0) – (-2.0), r=0.8

External eating, U=452, p=.03, 95% CI (-7.0) – (-5.0), r=0.7

Stress score, U=421, p=.01, 95% CI (-9.0) – (-1.0), r=0.7

Therefore, participants with GI symptoms show statistically significantly higher effect of stress level and eating behaviour on severity score when compared to the healthy participants [70]. Cohen's d was estimated more than 0.5 (0.7-1.8) indicating large effect size based on Cohen's guidelines [71].The following descriptive plots represent graphs of the means and 95% confidence intervals for each variable [72].

Discussion

In ancient times, physiological and morphological abnormalities of the gastrointestinal (GI) system visceral hypersensitivity; motility disturbances;

altered gut microbiota; altered mucosal and immune function; and altered central nervous system [73]. On further investigation of the effect of biophysical, physiological, psychosocial and behavioural factors on gut function and dysfunction lead to better identification, clarification, and categorization of functional gastrointestinal disorders [74]. Findings from these studies suggest a link between emotions and intestinal dysfunctions [75]. The contents of intestine are noxious to the sight, smell, sense, and touch which leads to avoidant emotional responses, vomiting, and nausea [76]. Scientific based evidence also suggests that brain-gut interactions explain how psychological and stress factors relate to intestinal function and dysfunction, and gastrointestinal disorders and symptoms [77]. Biopsychosocial and neuro gastroenterology is the latest clinical approach to understand gastrointestinal health and disease [78].

Evaluation of the current study

In this study, we tested the hypothesis that the severity of the gastrointestinal symptoms or gastrointestinal disorder (IBS, irritable bowel syndrome) is associated with stress [79]. Based on the statistical outcome, when compared to the control group, patients with severe GI symptoms illustrated a demand of selective eating pattern, specifically the involvement of diffuse emotions, clearly labelled emotions, emotional eating and external eating [80]. Concerning several other studies, this study also demonstrates that in general population patients with acute or chronic GI symptoms that may trigger IBS are at high risk to develop disordered eating practices [81]. The qualitative analysis of the symptomology questionnaire suggests that people experiencing GI symptoms were found to be sensitive to carbohydrates (i.e., wheat, whole grain), gluten, citrus fruits, milk and bakery products, spicy food, caffeine and junk food [82]. The severity of GI symptoms, however, was not correlated to age and BMI.

Furthermore, findings also illustrate significant progression of stressor score in comparison to the control group [83]. Evidence from preclinical and clinical research indicates stress as the key factor to induce alterations in neuroendocrine-immune pathways acting on the microbiota-gut-brain axis, and brain-gut axis leading to worsening of symptoms in IBS [84]. Studies evaluating the relationship between stress and severity of IBS explain that stress activates the corticotrophin-releasing factor (CRF), which converts brain stimulus into an enhanced physical response [85]. The CRF signalling system plays a key role when the body experiences stress and act as a primary neurotransmitter or neuromodulator [86]. The CRF system stimulates hypothalamic-pituitary-adrenal (HPA) axis, to coordinate the visceral and immune efferent limbs, and activate the coeruleus locus and its noradrenergic projections [87]. It can also modulate forebrain, hindbrain and spinal sites monitoring the activity of autonomic nervous system (ANS) which leads to induction of sympathetic and sacral parasympathetic activity and release of catecholamine [88]. On further study, findings indicate that stress also has an indirect or direct impact on the growth and composition of microbiota that helps to regulate bidirectional communication of the brain-gut axis [89]. Similar to other studies results from this research also show that stress has a powerful impact on triggering GI symptoms that can lead to FGIDs (e.g., IBS), and psychosocial and physiological stressors determine the growth of IBS [90]. The perceived stress scale used in this study, however, need not be a suitable measure to evaluate the stress level in this modern age as the settings and patterns of questions were developed in 1983 that need to be updated to meet the requirements of the new age [91].

Moreover, this study also evaluates the impact of severity of GI symptoms, dietary patterns, and stress on visuospatial episodic memory through the 8-pattern stage PAL test [92]. Clinical and experimental evidence demonstrate cognitive impairment related to stress in patients with IBS [93]. Although the pathophysiology of the link between IBS and cognitive performance is not well understood, it is assumed to be due to disruption of the brain-gut axis interactions [94]. Recent study findings illustrate impaired visuospatial memory in participants with IBS mainly depicting it as a stress-related impairment [95]. However, visuospatial memory impairment was only visible for 6-pattern stage PAL test. In comparison to other studies, participants selected for this study were not diagnosed by FGID's rather participants who exhibit 2 or more symptoms that may trigger into IBS [96]. Findings from this research did not display any correlation of 8-pattern memory test performance with stress, nutritional habits, and the presence of GI symptoms likely due to pre-attentional memory differences [97]. Several studies explain that the ability to control attention can constrain the capacity of short term visual memory [98]. Evidence from a recent study suggests that working memory in response to visual stimuli is a goal-oriented mental operation to support and store the information temporarily to perform cognition and behaviour (e.g., the focus of attention; FoA) [99]. The memory for a short duration of visual stimuli can direct the FoA in two ways:

1. Automatic, an effect indexed with the recent event or act that ameliorates the recall of the last item [100].

2. Strategic, an ability to prioritize the acts into different instructions which enhance the retention of important information and boost the capacity of executive function [101].

On further study, findings propose that some components of working memory competitively maintain FoA determined by recency and internal executive control perceptual drive [102]. Overall, when compared to the control group, there was no impact on the response time, first trial memory score, and total errors adjusted [103].

Limitations

This exploratory study is, however preliminary, limiting the research to measure only visuospatial episodic memory (in 8-pattern PAL test) rather than including other cognitive measures (e.g., attentional flexibility, response inhibition) [104]. Further follow-up studies are needful to analyze the cognitive performance in patients diagnosed with IBS or patients suffering from 2 or more GI symptoms that may lead to IBS mainly by the use of neuroimaging techniques to notice morphological brain changes [105].

Conclusion

Nevertheless, this study confirms other study findings that stress and eating practices have an impact on GI manifestations. Therefore, as suggested in many studies, although there is no cure for IBS, findings also note that appropriate, adequate, and suitable diet according to personal eating habits, vitamin D supplementation, and fiber containing food can help to control the worsening of GI symptoms and use of iron replacement therapies to improve cognitive performance. It is also advisable to undergo both non-pharmacological and pharmacological approaches that target to release stress and control the exaggeration of GI symptoms, such as antipsychotics, 5HT synthesis inhibitors, antidepressants, therapies to reduce stress and miscellaneous perceptual remedies to control the symptoms.

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