Does hypervigilance predict Functional Gastrointestinal Disorder symptoms over and above known psychosocial predictors?

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Abstract

Functional Gastrointestinal Disorders (FGIDs) are chronic medically unexplained disorders of the gut. Research has shown that, broadly, FGIDs are caused by dysregulation of the neurohormonal brain-gut axis. Biopsychosocial conceptualisations of the brain-gut axis imply a need to treat not only biological but also social and psychological contributors to dysregulation. Past research suggests that attentional bias might play a role in dysregulation. We investigated the relationship between hypervigilance and FGID symptoms over and above known psychosocial predictors of symptoms: anxiety, stress, neuroticism, pain catastrophising and self-efficacy. Electroencephalography was used to record Event Related Potentials (ERPs) to measure attention for any bias in FGID-sufferers. Participants also filled out scales measuring known psychosocial predictors of symptoms. FGID symptoms were tracked using a 14-day daily diary in which participants reported daily on pain, mood and non-pain symptoms. Averages and standard deviations in these indices across 14 days served as the outcome variables in regression models (12 in total). It was found that conscious attentional bias was marginally statistically significant when predicting mood. However, stress and neuroticism together predict significant variation in mood but not in symptom-related (pain and non-pain) daily diary variables. We also observed an effect of self-efficacy on fluctuations in pain. Daily Diary measures seem to be difficult to predict with existing survey measures and they do not seem to be predicted by measures of attention. With respect to daily diary measures, mood is a variable for future research to consider further.

Declaration

This thesis contains no material which has been accepted for the award of any other degree of diploma in any University, and, to the best of my knowledge, this thesis contains no material previously published except where due reference is made. I give permission for the digital version of this thesis to be made available on the web, via the University of Adelaide's digital thesis repository, the Library Search and through web search engines, unless permission has been granted by the School to restrict access for a period of time.

Benjamin Owen-Thomas

27/9/21

Contribution Statement

The current project was based on an existing data set. Before receiving ethics approval from Macquarie University to be added to the project, I worked with a list and description of the available variables to conduct an initial literature search and generate research questions. My supervisor provided only general guidance on the process of generating research questions. During this time, I also, with guidance from my supervisor, wrote up a pre-registration protocol and worked with a toy dataset to generate analysis templates. Upon receiving ethics permission to use the anonymised version of the actual data, I merged one set of daily diary results into the main dataset to obtain the final csv file. At this point, I generated the final analysis scripts for the analyses and ran them.

I was responsible for then writing the thesis and conducting any remaining analyses (e.g., descriptive statistics).

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27/9/21

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Lastly, I would like to thank the participants of this project who gave their time and energy to make this project possible.

Does hypervigilance predict FGID symptoms over and above known psychosocial predictors?

Functional gastrointestinal disorders (FGIDs) are chronic medically unexplained disorders of the gut (Talley, 2020). The disorders are classified and diagnosed based on the Rome IV diagnostic manual (Whitehead, 2016), and the two most common disorders are Irritable Bowel Syndrome (IBS) and Functional Dyspepsia (FD; Talley, 2020). Central to a diagnosis of IBS is recurrent abdominal pain, whereas FD can be diagnosed with non-pain symptoms such as bothersome early satiety, postprandial fullness or bloating (Whitehead, 2016). Symptomology can vary depending on the person and the FGID. For example, females tend to feel more fatigue, depression, anxiety and have a lower quality of life than male counterparts. Females are also twice as likely to seek medical care (Kim & Kim, 2018).

FGIDs are common in the general population, negatively impact quality of life, and cost the health sector billions of dollars annually (Sperber et al., 2021; Talley, 2020). A recent study found that 40% of people worldwide are affected by FGIDs (Sperber et al., 2021) with another study finding that 40% of referrals to gastroenterologists are for FGIDs (Noddin et al., 2005). The majority of the countries surveyed by Sperber et al. (2021) had between 3 and 5% of people being affected by IBS, with FD affecting between 4 and 7%. Past research has placed these numbers as high as 45% for IBS and 57% for FD (El-Serag & Talley, 2004; Palsson et al., 2016).

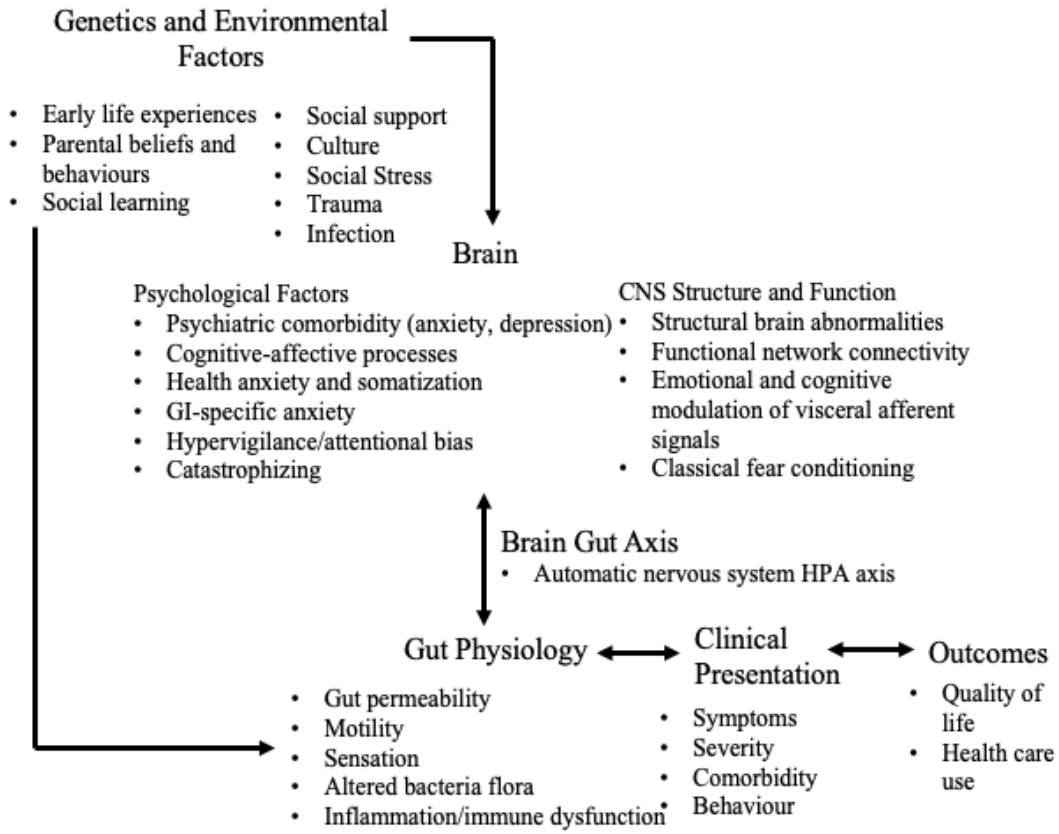
FGIDs are medically unexplained, in that the exact biological mechanisms giving rise to them have not been defined. However, a common general explanation of FGIDs is that they are caused by dysregulation of the brain-gut axis, a bidirectional neurohormonal communication system between the brain and the gut (Van Oudenhove et al., 2016). Structurally, the brain-gut axis hosts direct connections between the central nervous system and the myenteric plexus, the main nerve running through the gastrointestinal tract. Within

this structure, negative emotional states can affect gastrointestinal functioning. Analogously, signals in the opposite direction – from the gut to the brain – are modulated by the brain's cognitive and emotional centres. The affected centres, mainly the emotional ones, can impact upon our mood. Our mood is determined by our emotions. A mood is considered to be a group of persistent feelings that are associated with evaluations and cognitions of our emotions. Our mood can then influence thoughts, feelings and actions (Amado-Boccara et al., 1993). As a consequence of the bidirectionality and evaluative nature of mood, it can affect the symptomology of FGIDs but also be affected by symptoms. Accordingly, FGIDs have consistently been associated with a comorbid mood disorder (Van Oudenhove et al., 2016).

Theory and treatment of FGIDs informed by the biopsychosocial model

The bidirectional and integrated nature of the brain-gut axis implies a need to treat not only the biological aspects of FGIDs but also the cognitive aspects. Meeting this need, the conceptualisation of FGIDs has shifted from purely biological to 'biopsychosocial' over the past two decades (Drossman, 2016). The adoption of the biopsychosocial model with respect to FGIDs has led to more in-depth research on the psychological, social and coping-related variables that influence communication between the brain and the gut. According to the model, illustrated in Figure 1, these variables affect symptom severity and probability of onset of disorder. Consequently, there are reductions in the quality of life in the sufferer and increased use in health care needs.

Figure 1. Biopsychosocial model of factors influencing FGIDs. Adapted from Van Oudenhove et al. (2016).



Research motivated by the biopsychosocial model depicted in Figure 1 has shown that between 30 and 50% of FGID-sufferers have an anxiety disorder affecting symptom severity and probability of onset (Van Oudenhove et al., 2016). It has also been shown that higher levels of the personality trait of Neuroticism are associated with a higher probability of FGID symptom onset (McKinnon et al., 2015). A further finding has been that stress causes disruptions in the brain-gut axis, adding to FGID symptom severity and probability of onset (Drossman, 2016). Research has further shown that utilisation of coping strategies has the potential to mitigate or exacerbate symptoms (Drossman, 2016). For example, pain catastrophising, a maladaptive coping strategy that involves magnifying the seriousness of the symptoms and, thus, perceiving oneself as less able to reduce those symptoms, has been

linked to exacerbation of FGID symptoms (Drossman et al., 2000). Further, lower scores on adaptive coping strategies, such as self-efficacy, have been associated with stress and exacerbation of symptoms. Self-efficacy is the belief that one can attain a goal or in the context of chronic pain may be aimed at improving health. (Dąbek-Drobny et al., 2020).

The set of findings just outlined illustrates that the 'Psychological Factors' in the biopsychosocial model can be roughly classified into 'traits' (e.g., Neuroticism), 'states' (e.g., stress) and 'coping mechanisms' (e.g., pain catastrophising and self-efficacy). Traits are stable features of personality, while states are more transient and variable (Geiser et al., 2017; Ladd & Gabrieli, 2015). Past research has found that anxiety can take the form of a more stable state or a more variable trait (Vagg et al., 1980). Coping mechanisms are how people view, respond to and learn from stressful situations (Skinner, 2016). Coping mechanisms can curb or exacerbate negative states depending on which ones are used.

Table 1 below lists studies published since the year 2000 that indicate a relationship between FGID symptom severity or onset and the traits, states and coping mechanisms that are shown in Figure 1. These studies were located by checking the reference lists of four recent reviews of the literature: reviews by Van Oudenhove et al. (2016), Drossman (2016), Sperber et al. (2021) and Talley (2020). Some informal searches were also undertaken in Scopus, as described in the footnote to the table. It is clear from the table that anxiety, stress, neuroticism, pain catastrophising and self-efficacy emerge as predictors of symptomology in the largest number of studies relative to other traits, states and coping mechanisms.

Table 1. Studies published in the year 2000 or later˜ that have found a relationship between states, traits or coping strategies and FGID symptom onset and severity Studies with N > 300 highlighted.

Environmental or social predictor	FGID symptom onset likelihood	FGID symptom severity	Sample Size (N)
Anxiety	Koloski et al. (2012)		1775
		Soderquist et al. (2020)	576
	Sibelli et al. (2016)		4810
	Jones et al. 2017		4966
	Sykes at al. (2005)		188
	Aro et al. (2015)		3000
		Simren et al. (2019)	407
	Locke et al. (2004)		222
	Hazlett-Stevens et al (2002)		1021
	Seres et al. (2008)		156
	Dunlop et al (2003)		90
	Camilleri et al. (2008)		
	Bulut et al. (2018)		109
		Shang & Xu. (2018)	210
	Lee et al. (2020)		99
	Jang et al. (2017)		1217
	Ly et al. (2015)		259
	Vu et al. (2014)		912
	Hosseinzadeh et al. (2011)		54
Stress	Locke et al. (2004)		222

	Murray et al. (2004)		36
	Suarez et al. (2010)		668
		Dąbek-Drobny et al. (2020)	129
	Duan et al. (2021)		3838
	Jang et al. (2017)		1217
	Chen et al. (2021)		2520
	Pozos-Radillo et al. (2018)		561
Neuroticism	Charles et al. (2008)		21676
	Dąbek-Drobny et al. (2020)		129
	Farnam et al. (2007)		150
	Hazlett-Stevens et al. (2002)		1021
	Drossman et al. (2000)		174
	Tanum & Malt. (2001)		111
	Beath et al. (2019)		210
	Sharbafchi et al. (2019)		4763
	Zarpour & Besharat. (2011)		170
Pain-Catastrophising	Lackner & Quigley (2005)		186
	Drossman et al. (2000)		174
	Seres et al. (2008)		156
		Lackner et al. (2004)	244
Self-efficacy (including self-management)	Dąbek-Drobny et al. (2020)		129

	Drossman et al. (2000)		174
		Dorn et al. (2015)	40
		Everitt et al. (2013)	123
		Hunt et al. (2009)	54
		Ljotsson et al. (2011)	195
		Ljotsson et al. (2010)	85
		Perderson (2015)	123
		Lackner et al. (2008)	75
		Oerlemans et al. (2011)	76
		Shahabi et al. (2015)	27
		Van Tillburg et al. (2009)	19
		Ghiyasvadndian et al. (2016)	119
		Moss-Morris et al. (2010)	64
		Robinson et al. (2006)	420
		Sanders et al. (2007)	28
		Jarrett et al. (2009)	176
		Jarrett et al. (2016)	85
		Labus et al. (2013)	69
Depression		Soderquist et al. (2020)	576
	Sibelli et al. (2016)		4810
	Jones et al. 2017		4966
		Simren et al. (2019)	407
	Locke et al. (2004)		222
	Seres et al. (2008)		156

		Lackner et al. (2004)	244
	Dunlop et al. (2003)		90
	Koloski et al. (2012)		1775
	Camilleri et al. (2008)		163
	Bulut et al. (2018)		109
		Shang & Xu. (2018)	210
	Lee et al. (2010)		279
	Lee et al. (2020)		99
	Vu et al. (2014)		912
	Singh et al. (2012)		382
Trauma/abuse	Baccini et al. (2003)		260
	Drossman et al. (2000)		174
		Sherman et al. (2015)	268
	Lee et al. (2020)		99
Extraversion	Dąbek-Drobny et al. (2020)		129
		Sharbafchi et al. (2019)	4763
Openness	Farnam et al. (2007)		150
		Sharbafchi et al. (2019)	4763
Conscientiousness	Farnam et al. (2007)		150
		Sharbafchi et al. (2019)	4736
Agreeableness		Sharbafchi et al. (2019)	4763
Decreased self-perceived ability to decrease symptoms	Drossman et al. (2000)		174

Resilience	Dąbek-Drobny et al. 2020		129
Social Support		Lackner et al. (2013)	235
		Lackner et al. (2010)	105
Negative social relationships	Lackner et al. (2013)		235
	Jang et al. (2017)		1217
Passive coping	Jones et al. (2005)		177
Less planful problem solving	Suarez et al. (2010)		668
Less task-orientated coping	Suarez et al. (2010)		668
Less positive appraisal	Suarez et al. (2010)		668
Escape avoidance	Suarez et al. (2010)		668
Negative self esteem	Grodzinsky & Walter. (2015)		353
Anger	Bulut et al. (2018)		109
	Tanum & Malt. (2001)		111
Mindfulness		Beath et al. (2019)	210
		Teh et al. (2021)	28
Job demand	Jang et al. (2020)		1217
	Popa et al. (2018)		76
Organisational system	Jang et al. (2020)		1217
Lack of reward	Jang et al. (2020)		1217
Panic disorder	Singh et al. (2012)		382
Alexithymia	Mazaheri et al. (2012)		237

˜Studies were located by searching through the reference lists of four reviews: Van Oudenhove et al. (2016), Drossman (2016), Sperber et al. (2021) and Talley (2020). Follow-

up searches were conducted in Scopus. The search terms consisted of the name of the relevant psychosocial variable followed by the term 'functional gastrointestinal disorder'.

Attentional bias: A contributing and possibly unconscious psychological factor?

Studies of anxious people have found that sufferers have an attentional bias, or hypervigilance, towards threatening words or stimuli. The hypervigilance shown can exacerbate or maintain features of the disorder (Cisler et al., 2009; Schoth et al., 2012). Attentional bias, or hypervigilance, refers to attention allocation to certain stimuli at the expense of attention paid to other stimuli (Posserud et al., 2009). Hypervigilance exhibited by sufferers exacerbates symptoms by burdening cognitive resources and reinforcing avoidant behaviours toward certain stimuli or activities that they perceive as symptom inducing (Mogg, & Bradley, 2016; Schoth et al., 2012; Todd et al., 2015). Cognitive resources become burdened when patients perceive the symptoms as being painful and develop a sense of fear or catastrophic thinking. Further, if sufferers perceive the symptoms as not being within their control they will engage in more fearful and catastrophic thinking adding to the cognitive load and exacerbating symptoms.

A study conducted by Dorn et al. (2007) found that people with IBS reported pain and discomfort to balloon distension in the rectum or colon at abnormally low volumes of pressure. Concurrent functional magnetic resonance imaging (fMRI) and Positron Emission Tomography (PET) indicated that IBS sufferers also showed similar abnormal cortical responses for actual and sham distentions. Subsequent studies using distension and concurrent fMRI and PET scans have produced similar results (Van Oudenhove et al., 2010; Elsenbruch et al., 2010). Together with findings of high levels of comorbidity between anxiety and FGIDs, these studies suggest that attentional biases play a role in FGID symptom

onset and severity. The biopsychosocial model lists hypervigilance and attentional bias among psychological contributors to brain-gut axis dysregulation.

Some of the most commonly used tests of attentional bias are the emotional Stroop, the dot-probe, and the visual probe, which are usually administered on a computer (Mathews & MacLeod, 1985; Williams et al., 1996). The emotional Stroop task involves seeing various words, including threat-related words, displayed on a computer screen in different font or colours. The participant is required to indicate the colour of the word. A longer response time represents greater attention allocation (Mathews & MacLeod, 1985; Williams et al., 1996). Other measures of attentional bias, similarly, use reaction time as the main indicator of attention allocation.

The hypothesised automaticity of signalling in the central nervous system and the brain-gut axis has led researchers to hypothesise that attentional biases in FGID-sufferers might be unconscious (Cisler & Koster, 2010; Sass et al., 2010; Van Oudenhove et al., 2016). Three studies that examined attentional biases and their conscious accessibility in people with FGIDs suggest that the biases are unconscious. Afzal et al. (2006), using the emotional Stroop task, found that IBS patients allocated more attention to symptom words over neutral words compared to controls, but only when words were shown subliminally (i.e., for a very short period: 15ms). A dot-probe study by Martin and Chapman (2010) similarly found that FGID-sufferers showed attentional bias towards briefly presented (100ms) social-threat words relative to neutral words. Evidence of unconscious attentional bias was also obtained in a subsequent dot-probe task administered by Chapman and Martin (2011). In this study it was found that when subliminal cues were presented to IBS sufferers there was a faster response toward pain-related words compared to a control group of participants.

Two meta-analyses conducted by Crombez et al. (2013) and Schoth et al. (2012) on attentional biases and chronic pain found evidence of conscious attentional bias. They

concluded that attentional biases in chronic pain sufferers are present when words are presented for a longer period rather than subliminally.

The emotional Stroop, dot probe and other common measures of attentional bias are based on reaction time. As conscious processes, reaction behaviours do not capture a complete time course of attentional biases; that is, they do not capture how conscious and unconscious processes contribute to the bias. The same is the case for the fMRI- and PET-based measures of cognitive processing used by Dorn et al. (2007). These measures have a time resolution of a few seconds. Moreover, fMRI and PET studies of reactions to distension have tended to be highly invasive, and therefore inherently stress-inducing.

More precise measurement of the stages of attentional bias

A pilot study conducted by Ejova et al. (2021) used electroencephalography (EEG) – a method with millisecond time resolution – to determine if FGIDs are characterised by unconscious or conscious attentional bias. Ejova et al. (2021) showed FGID-sufferers and healthy controls word sequences consisting of emotionally neutral, emotionally negative and symptom-related words and captured four event related potentials (ERPs). ERPs are voltages generated by the brain in response to stimuli (Sur & Sinha, 2009). They are captured by the EEG and are time-locked amplitudes in specified regions of the brain.

The ERPs that were examined in the current project were the P100, Early Posterior Negativity (EPN), N400 and Late Positive Potential (LPP). For the P100, a larger negative going amplitude represents greater unconscious attention. For the EPN, a larger positive amplitude indicates greater unconscious attention. For the N400, a smaller positive amplitude is indicative of greater conscious attention, and, for the LPP, more positive values represent greater conscious attention. Ejova et al. (2021) found that FGID-sufferers had marginally significantly higher occipital EPN amplitudes for all words, indicating marginally higher

unconscious attention to the task. Furthermore, FGID-sufferers but not healthy controls recorded a lower amplitude in the central N400 toward emotionally negative words as opposed to emotionally neutral and symptom-related words. This finding indicated greater conscious bias towards threat among FGID-sufferers.

The Current Project

The current project aims to investigate the relationship between unconscious and conscious attentional biases captured in Ejova et al. (2021), and subsequent physical and psychological symptoms of FGIDs. We hypothesise that pain symptoms, non-pain symptoms and mood will be significantly predicted by occipital EPN and central N400 over and above known psychosocial variables. Given that the psychosocial influences on symptoms summarised in Figure 1 fall roughly into the categories of 'states', 'traits' and 'coping mechanisms', and given the pattern of findings summarised in Table 1, where large studies indicated predictive effects for anxiety, stress, neuroticism, pain catastrophising and self-efficacy, the following control variables were selected: anxiety (a mixture of state and trait), stress (a state), neuroticism (a trait), pain catastrophising (a maladaptive coping strategy), and self-efficacy (an adaptive coping resource). Pain catastrophising has not been explored in very large sample sizes, but is a coping strategy that relates specifically to health and is known to be related to numerous maladaptive coping strategies (Quartana et al., 2009; Gatchel & Neblett, 2017; Petrini & Arendt-Nielsen, 2020). These variables span all three categories and have the largest associated bodies of evidence indicating that they are predictive of symptomatology. A measure of an adaptive coping resource – self-efficacy – is included among the control variables because Ejova et al. (2021) showed that ERP amplitudes correlate with some adaptive coping strategies. This means that attentional biases could reflect adaptive rather than maladaptive tendencies. Without the inclusion of an adaptive coping strategy as a

control variable, we cannot assess the usefulness of ERPs for tracking adaptive tendencies that might predict symptomatology over and above maladaptive predictors.

The current project used pain, non-pain symptom and mood ratings from 14-day daily diaries as the outcome variables. In Ejova et al.'s (2021) study, participants completed these diaries after the EEG session. Daily diaries were used by Ejova et al. (2021) because little concordance has been found between retrospective reporting of FGID symptom severity and daily-diary-based ratings (Jones et al., 2019).

According to Lischetzke (2014), daily diary methodologies are preferred when the research is aimed at capturing real time or close to real time representations of momentary experiences, such as FGID symptoms on a particular day. Capturing these representations relies on episodic memory. Episodic memories are memories about specific events during a particular time. Shortly after an experience, people have greater access to the episodic memories. However, as the interval between the experience and recall increases these episodic memories become inaccessible. Episodic memories degrade without rehearsal and when people are asked to recall the memories that have degraded there is a shift to semantic memory or generalised memory (Lischetzke, 2014; Robinson & Clore, 2002). Semantic memories are generic and context-free. Capturing experiences regarding specific FGID symptom information at a specific time is ideally done with prospective daily diaries when there is still access to episodic memory. Further, Jones et al. (2019) concluded that prospective collection of symptoms and other measures is preferable to other approaches due to the avoidance of the mentioned memory biases.

Ejova et al.'s (2021) results indicated that FGID-sufferers exhibited an attentional bias toward emotionally negative words. Whilst it would be more intuitive to expect greater vigilance towards symptom-related words in FGID-sufferers, the emotionally negative words still represent threat. Martin and Chapman (2010) similarly observed attentional bias towards

emotionally negative rather than pain-related words among FGID-sufferers. This may be due to the fact that not all symptoms are experienced equally by all FGID-sufferers. Alternatively, as Martin and Chapman (2010) argue, FGID-sufferers experience functional impairments that, over time, impact their abilities to successfully overcome negative emotions in daily life. For the current project this means that the attention being paid to emotionally negative words will serve as the measure for hypervigilance. Analyses for symptom-related words will be presented as a supplement.

Overall, the current research project will consider the following research questions:

1. Does unconscious hypervigilance to emotionally negative nouns, as indicated by EPN amplitude in the occipital region, predict daily diary *average* (a) pain levels, (b) non-pain symptom levels, and (c) mood in FGID-sufferers over and above known psychosocial predictors?
2. Does unconscious hypervigilance to emotionally negative nouns, as indicated by EPN amplitude in the occipital region, predict daily diary *fluctuation* (standard deviation) in (a) pain levels, (b) non-pain symptom levels, and (c) mood in FGID-sufferers over and above known psychosocial predictors?
3. Does conscious hypervigilance to emotionally negative nouns, as indicated by N400 amplitude in the central region, predict *average* (a) pain levels, (b) non-pain symptom levels, and (c) mood in FGID-sufferers over and above known psychosocial predictors?
4. Does conscious hypervigilance to emotionally negative nouns, as indicated by N400 amplitude in the central region, predict *fluctuation* (standard deviation) in (a) pain

levels, (b) non-pain symptom levels, and (c) mood in FGID-sufferers over and above known psychosocial predictors?

Method

Participants

All participants (*N = 29)* were female undergraduate psychology students recruited from Macquarie University, Sydney. Initially participants filled out a screener questionnaire (Appendix 1) to determine if they met the inclusion criteria to be in the FGID sufferers' group. Twenty-nine (96.7%) met the Rome IV criteria (Whitehead, 2016; Appendix 2) for IBS or FD, with 14 (46.7%) meeting the criteria for IBS, 5 (16.7%) meeting the criteria for FD, and 6 (20%) meeting the criteria for both disorders. One of the participants (3.3%) met all but one of the criteria for IBS. The diagnostic criteria for IBS require that pain be related to changes in stool or defecations on at least 30% of occasions but this participant reported that the co-occurrence took place on 20% of occasions.

The screener questionnaire also contained the Gastrointestinal Symptom Rating Scale (GSRS). The GSRS is an instrument of 15 items that gathers information on Reflux, Abdominal pain, Indigestion, Diarrhoea and Constipation. It has a 7-point Likert scale with 1 representing absence of troublesome symptoms to 7 which represents very troublesome symptoms (Kulich et al., 2008).

If students had a current diagnosis of a chronic illness (e.g., Crohns Disease or rheumatoid arthritis) or a psychiatric disorder (e.g. schizophrenia, depression or anxiety), they were excluded from the study. After providing informed consent, participants received course credit for their involvement (Ejova et al. 2021).

Healthy controls (*N=30*) were selected if they met no more than two of three of the Rome IV criteria for IBS, none of the criteria for FD postprandial distress and no more than one criterion for FD epigastric pain.

Procedure and Materials

Days prior to the EEG recording session, participants filled out an online questionnaire through *Qualtrics*. The questionnaire measured demographic, health-related and psychosocial variables. The associated measures and the FGID-sufferers' scores on them are described in Appendix 3 and compared to scores for the healthy control group.

For the psychosocial measures relevant to the present analysis, anxiety and stress were each operationalised as the average on 7 items of the Depression Anxiety and Stress scale (Lovibond & Lovibond, 1995). An example item (for anxiety) is "I was aware of dryness in my mouth". Responses could range from *Did not apply to me at all* (0) to *Applied to me very much or most of the time* (3). Neuroticism was operationalised using the mean of responses to the 10-item International Personality Item pool (Goldberg, 1999). Items included "I am very pleased with myself", and responses could range from *very inaccurate* (1) to *very accurate* (5). Pain catastrophising was operationalised using the mean responses to 13 questions on the Pain Catastrophising Scale (Sullivan et al., 1995). An example question is "I become afraid that the pain will get worse" with responses ranging from *Not at all* (0) to *All the time* (4). Self-efficacy was operationalised using the mean of responses to 10 items on the Generalised Self-Efficacy Scale (Schwarzer & Jerusalem, 1995). An example question is "I can solve most problems if I invest the necessary effort", with responses ranging from *Not at all true* (1) to *Exactly true* (4).

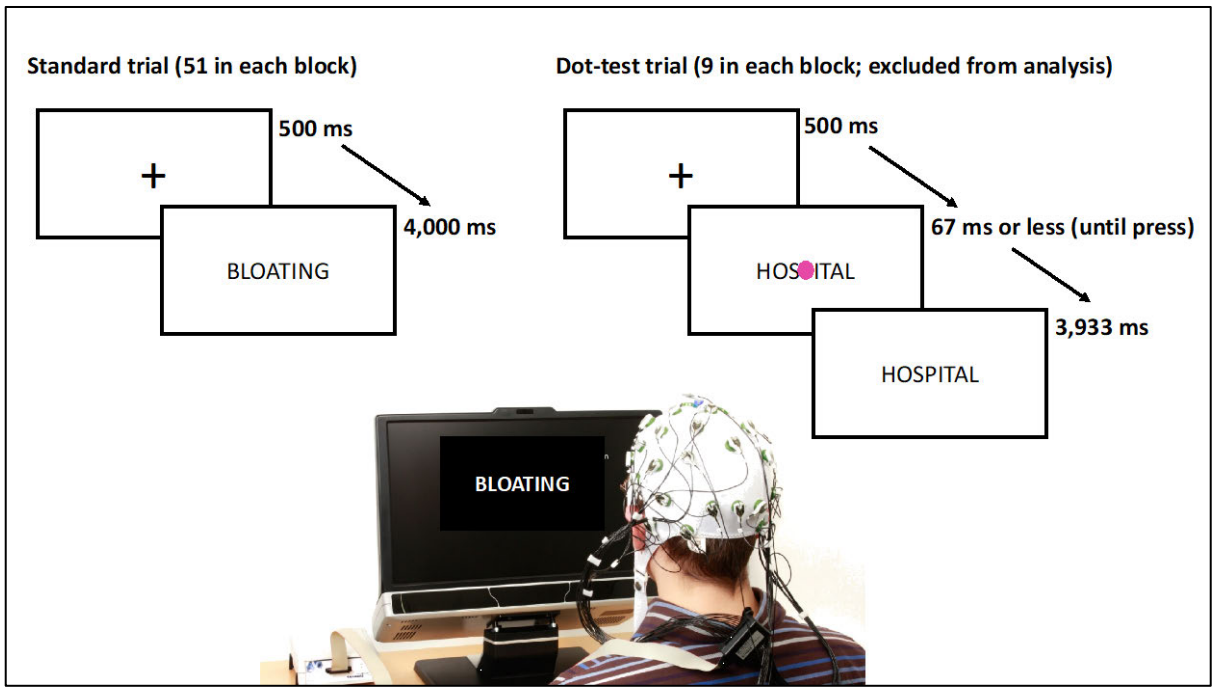
Notably, due to a technical issue, one participant was missing measures of all psychosocial variables, and an additional participant was missing a pain catastrophising score.

EEG session

During the EEG session, participants undertook a silent reading task modelled on one conducted by Wabnitz et al. (2016). The task involved participants randomly viewing one of 60 words for 4000ms after a 500ms fixation cross. Twenty of the words were symptom-related, 20 were emotionally neutral and 20 were emotionally negative. All of the words that were presented were nouns. As described in full detail by Ejova et al. (2021), words were matched on length, orthographic neighbourhood, frequency of occurrence in corpora of English text, emotional valence, and emotional arousal. More specifically, words were selected in such a way that the means and standard deviations of these characteristics were the same across noun types.

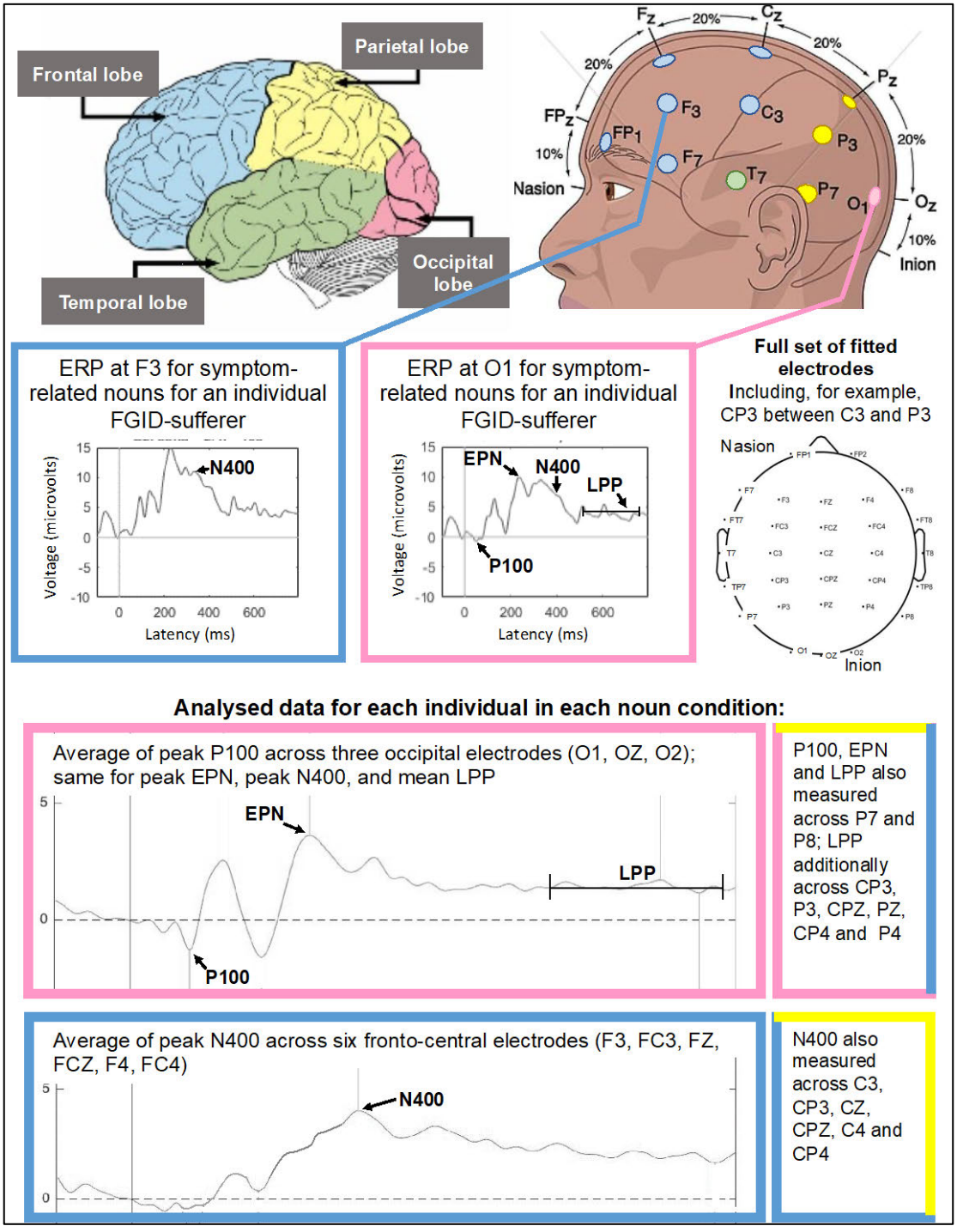
Figure 2 depicts one trial of the silent reading task. Participants viewed the 60 words six times, in six blocks. There were breaks in between as required. To maintain focus participants pressed the space bar on the keyboard as soon as possible when a red dot appeared in the centre of the screen. In each block, there were nine random trials in which the dot appeared. Participants had 67ms to respond to the dot before it disappeared. Participants received feedback at the end of each block regarding the percentage of dots to which they reacted to in time.

Figure 2. Description of a typical trial (with a symptom-related noun) in the silent reading task. The press refers to a pressing of the spacebar using both index fingers. Source: Ejova et al. (2021).



Recording and processing of the EEG signal was conducted as described by Ejova et al. (2021). Figure 3 summarises the placement of the Neuroscan EasyCap 32-electrode cap with the layout of electrodes being in accordance with the International 10-20 system. In Figure 3 there are also examples of peaks and time bands of the selected ERPs.

Figure 3. Examples of peaks analysed in this study, and the locations of those peaks with reference to the lobes of the brain.



Daily Diaries

Following the EEG session participants filled out a daily diary over 14 days (starting on the day after the EEG session). The *RealLifeExp* app was used to administer the questionnaire (LifeData, 2021). At a random time between 2pm and 9pm each day, participants received a notification to fill out the diary before midnight. Each daily diary took approximately 3 minutes to complete.

The daily diary consisted of the following questions; (1) At what time did you finish you last major meal? (2) What was your last major meal? Breakfast/Lunch/Dinner. (3) How would you rate any gastro pain you have experienced so far today? Rated on a sliding scale of 0 to 10, moving from *no pain* to *worst pain*. If a participant's response on the pain rating scale was greater than 0, they moved to question (4) how much did your gastro pain interfere with your planned activities? Rated on a sliding scale of 0 to 10, moving from *not at all* to *very much*. (5) How would you rate any non-pain gastro symptoms you have experienced so far today? Rated on a sliding scale of 0 to 10, moving from *no symptoms* to *worst symptoms*. If a participant's response on the non-pain rating scale was greater than 0, they moved to question (6) How much did your non-pain gastro symptoms interfere with your planned activities? Reponses were rated from 0 to 10, moving from *not at all* to *very much*. (7) How would you rate your mood right now? Reponses were rated on a sliding scale from -5 to 5 moving from *negative* to *positive*. Responses to these questions were averaged to determine average pain, non-pain symptoms and mood scores across the 14-day period. Standard deviations over the 14-day period indicated fluctuations in symptoms, non-pain symptoms and mood.

On the fourteenth day of the daily dairy process, participants were presented with an additional question asking about their average level of symptoms from the preceding 14 days. Responses to this question were not analysed in this study.

Statistical Analysis

For all research questions, multiple regression models were fitted to the data. *R* (v. 4.0.4; R Core Team, 2021) was used for all analyses, with the *lm* function (in the *base* package; R Core Team, 2021) being used for the regressions. All parts of the analysis except the *Preliminary analysis* were pre-registered (see https://osf.io/q9ye2/).

Preliminary analysis. A preliminary examination of the correlations between all the variables of interest to the study indicated that there was a strong relationship between stress and anxiety. As a result, anxiety was removed from the analysis. Anxiety can be conceived of as a state or a trait, so removing it enabled us to retain predictors spanning all categories of psychological factors in Van Oudenhove et al.'s (2016) biopsychosocial model: traits (Neuroticism), states (stress), maladaptive coping strategies (pain catastrophising), and adaptive coping strategies (self-efficacy).

Main analysis relating to research questions. For Research Question 1a, a multiple regression model was fitted to data from FGID-sufferers ($N = 29$) with the 14-day average of pain ratings as the outcome variable, and six variables as predictors: stress, neuroticism, pain catastrophizing, self-efficacy, and occipital EPN. The first five predictors are the psychosocial variables being controlled, with the incremental effect of occipital EPN being the chief effect of interest. For research questions 1b and 1c, the same model was be fitted, except with average non-pain symptoms and average mood as the outcome variables, respectively.

Research Questions 2a to 2c was analysed in the same way as 1a to 1c, except with standard deviation in pain, non-pain symptom and mood ratings as the outcome variables, rather than means.

For Research Questions 3 and 4, concerning the incremental effects of central N400, the analyses were the same as for Research Questions 1 and 2, except that the incremental effect of central N400 was the chief effect of interest.

Assumption checks. Subsequent to running visual checks on regression model residuals using box plots and histograms, the assumptions of the regression models were checked in line with the pre-registered analysis protocol (see https://osf.io/q9ye2/). Box-Cox transformations were applied to predictor variables that were shown by Tukey tests to have violated the linearity assumption.

Checks of interaction effects. The effects of any uncovered significant ERP-based predictors of ERPs in the main analysis and the supplementary analysis for symptom-related words were checked in relation to whether they are stronger among FGID-sufferers as compared to healthy controls. To do this, regression models with the following terms were fitted to data from both groups of participants ($N = 60$) for the relevant outcome variable:

- ERP found to be significant in primary or supplementary analysis
- Group
- ERP x Group

Sensitivity analysis. Given the small sample size (*N=29)* of our experiment, we ran a sensitivity analysis. For our study to have the conventional statistical power of 0.8 with 6 predictors, our effect size (f^2) must be .60, which roughly corresponds to a correlation of .77 between the ERP and the relevant outcome variable.

Supplementary Analysis. Our supplementary analysis consisted of the entire statistical analysis outlined above, albeit with ERPs for symptom-related words as the variable predicting outcome variables.

Results

Participant Characteristics

In Table 2 we can see the descriptive statistics for the sample (N = 29). The means and standards deviations are presented in Table 2 and indicate that variability around means for the majority of variables was not excessive, in that standard deviations were generally less than half the size of the means. However, Table 2 reveals that mean scores for fluctuations in pain, as represented by the standard deviation for the average in pain, was almost equal to the scores in average pain for FGID sufferers. Fluctuations and averages in pain for healthy controls as seen in Appendix 3 also had substantial variability; however, were much lower.

In Appendix 3 we can see that when sufferers were compared to controls there was no difference between groups on key demographics such as age, previous diagnosis of psychiatric conditions and past chronic illness rates. However, as expected, FGID-sufferers reported more severe gastrointestinal symptoms as well as psychological symptoms, such as depression, anxiety, health anxiety and somatic symptoms. Sufferers also differed significantly from the control group on all daily diary variables except fluctuations in mood.

They reported higher pain and non-pain symptoms, and lower mood, as well as greater fluctuation in pain and non-pain symptoms.

Table 2. FGID sufferers scores (N = 29: mean and standard deviations) for ERPs, psychosocial variables and daily diaries.

Variable	FGID-sufferers[#]: Mean (SD)
EPN event related potential (Symptom-related Words)	6.830 (3.470)
EPN event related potential (Emotionally Negative Words)	6.820 (3.160)
EPN event related potential (Neutral Words)	6.490 (3.260)
N400 event related potential (Symptom-related Words)	1.730 (3.750)
N400 event related potential (Emotionally Negative Words)	0.830 (3.630)
N400 event related potential (Neutral Words)	1.700 (3.660)
Anxiety	0.724 (0.489)
Stress	1.192 (0.621)
Neuroticism	2.648 (0.360)
Pain-Catastrophising	1.289 (0.893)
Self-Efficacy	2.783 (0.452)
Average Pain	2.672 (1.396)
Fluctuations in pain	2.084 (0.815)
Average Mood	5.212 (1.108)
Fluctuations in mood	1.737 (0.659)
Non-Pain Symptoms	2.660 (1.465)
Fluctuations in non-pain symptoms	1.920 (0.760)
GSRS	3.187 (1.253)

[#]Due to a technical issue, one participant in this group was missing measures of all psychosocial variables, and an additional participant was missing a pain catastrophizing score.

Preliminary Analysis

Correlations between all the variables of interest to the study are presented in Figures 4, 5 and 6. Noticing the high correlation between stress and anxiety, we removed anxiety as a regression predictor (see *Statistical Analysis* for more details).

Figure 4. Spearman Correlations between average pain (gpain_level), fluctuations in pain (gpain_level_sd), anxiety, stress, neuroticism, pain catastrophising (pcs), self-efficacy (self-effic), EPN, N400 and Gastro Symptom Rating Scale in FGID sufferers.

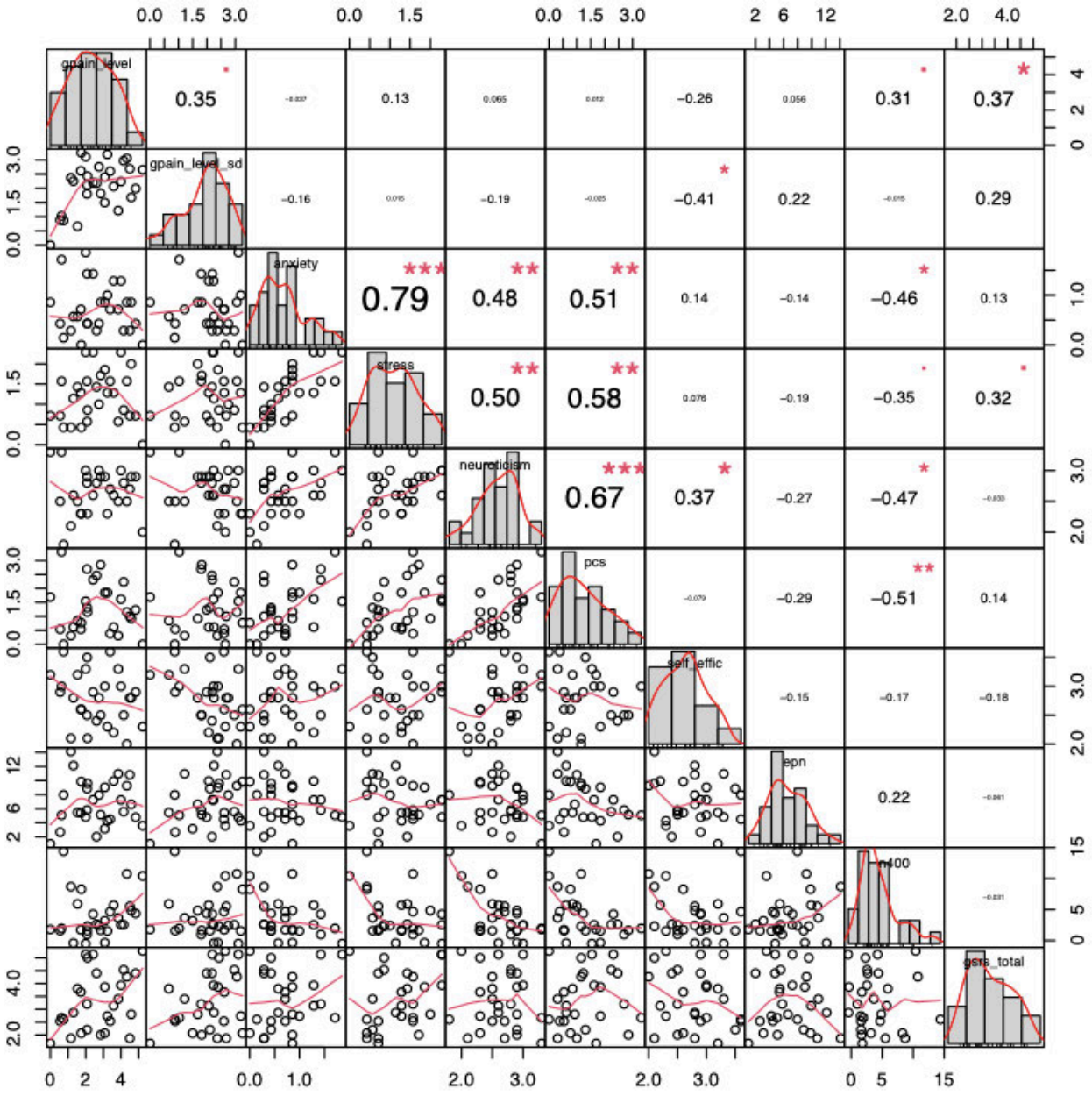


Figure 5. Spearman Correlations between average mood (curr_mood), fluctuations in mood (curr_mood_sd), anxiety, stress, neuroticism, pain catastrophising (pcs), self-efficacy (self-effic), EPN, N400 and Gastro Symptom Rating Scale in FGID sufferers.

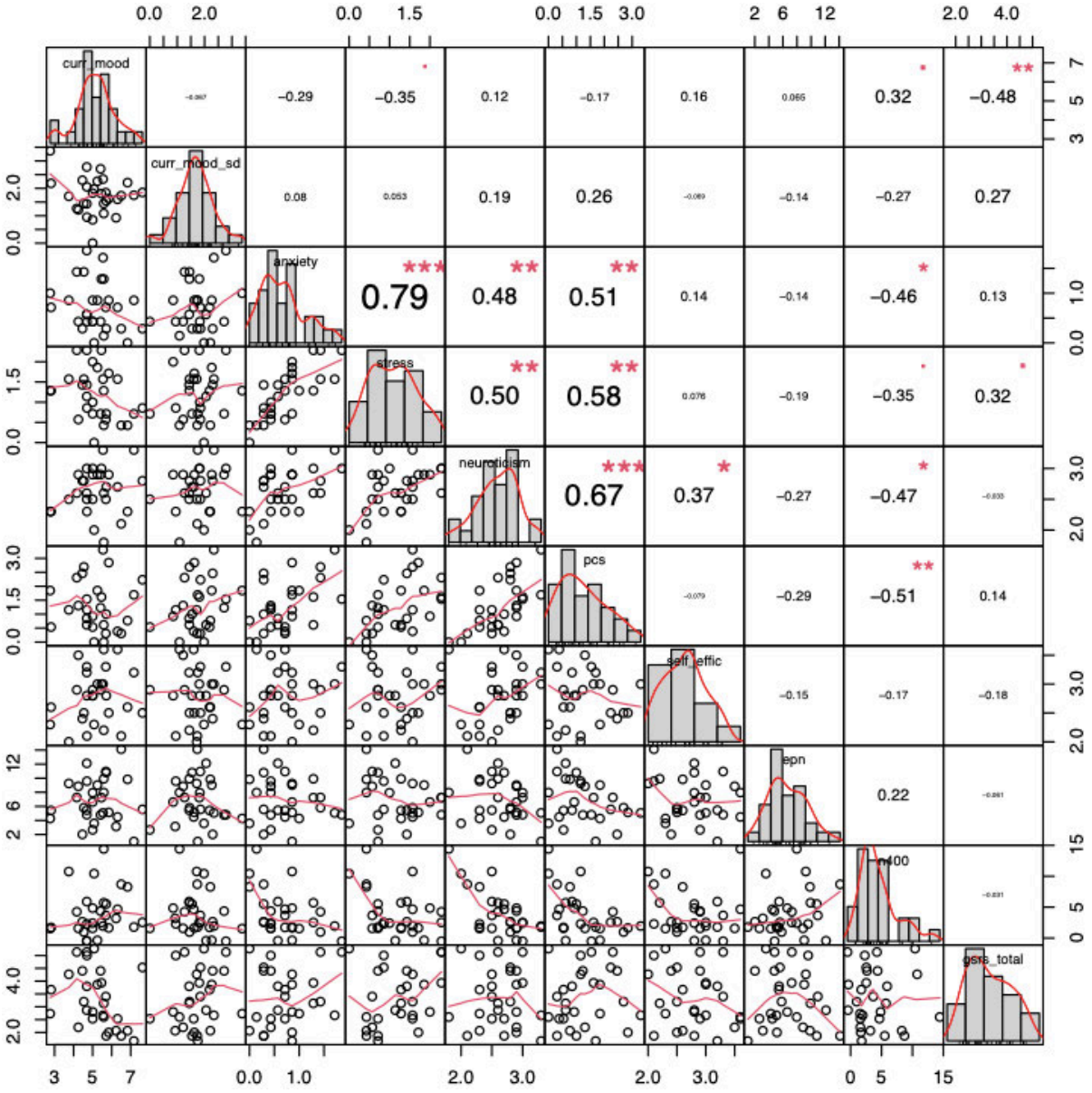
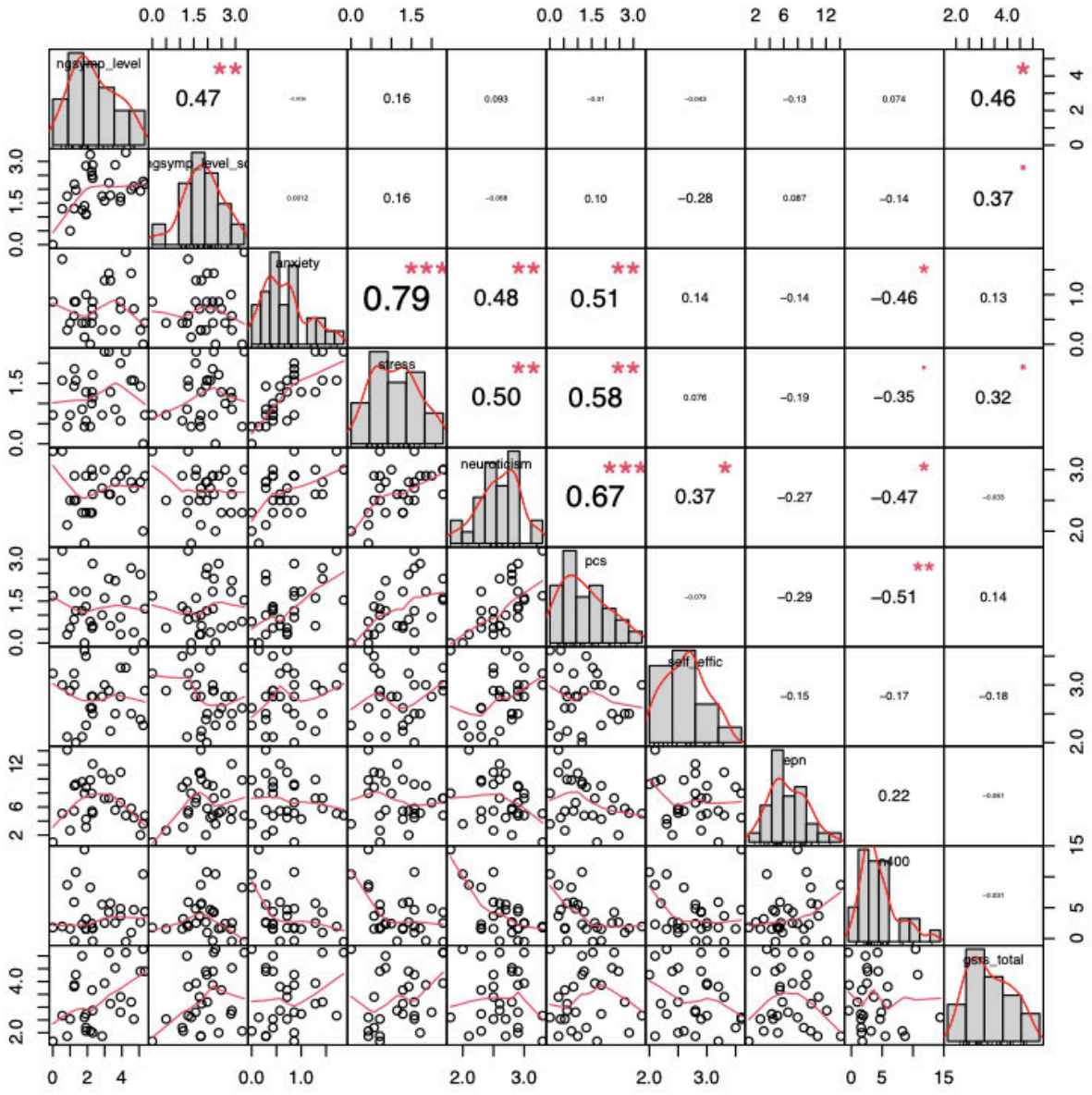


Figure 6. Spearman Correlations between average non-pain symptoms (ngsymp_level), fluctuations in non-pain symptoms (ngsymp_level_sd) anxiety, stress, neuroticism, pain catastrophising (pcs), self-efficacy (self-effic), EPN, N400 and Gastro Symptom Rating Scale in FGID sufferers.



Main Analysis

In Tables 3 and 4 we can see that there was only one regression that returned a significant adjusted R-squared result. This regression modelled the incremental effect of the N400 on mood. In this model, however, the significant predictive effects were shown by stress and neuroticism and the N400 was only marginally statistically significant as a predictor.

Further, Tables 3 and 4 indicate that significant predictive effects were shown by stress in the model of the incremental effects on the EPN on average mood. Significant predictive effects were also shown by self-efficacy in the model of the incremental effects of the N400 on fluctuations in pain.

Table 3. Results from six regressions run with occipital EPN for emotionally negative nouns as the main predictor variable of interest and average pain, fluctuations in pain, average mood, fluctuations in mood, average non-pain symptoms and fluctuations in non-pain symptoms as the outcome variables (N = 29)

	b	***b SE***	***β***	***p*-value**
Average pain: R^2 = 0.160; Adj. R^2 = -0.030; *p* = 0.536				
Stress	0.439	0.546	0.192	0.430
Neuroticism	1.253	1.232	0.313	0.320
PCS	-0.725	0.506	-0.449	0.166
Self-Efficacy	-1.313	0.721	-0.418	0.082
EPN~	-0.010	0.098	-0.021	0.922
Fluctuations in Pain R^2 = 0.302; Adj. R^2 = 0.144; *p* = 0.134				
Stress	0.376	0.285	0.287	0.200
Neuroticism	-0.385	0.643	-0.168	0.555
PCS	-0.038	0.264	-0.041	0.888
Self-Efficacy	-0.718	0.376	-0.400	0.069
EPN	0.048	0.051	0.184	0.360
Average Mood R^2 = 0.316; Adj. R^2 = 0.160; *p* = 0.114 ~				
Stress	-0.990	0.386	-0.553	0.018*
Neuroticism	1.684	0.870	0.537	0.066
PCS	-0.039	0.357	-0.031	0.915
Self-Efficacy	0.244	0.509	0.099	0.637
EPN	0.063	0.069	0.178	0.372
Fluctuations in Mood R^2 = 0.078; Adj. R^2 = - 0.132; *p* = 0.863				
Stress	-0.009	0.263	-0.008	0.974
Neuroticism	-0.008	0.594	-0.004	0.990
PCS	0.184	0.244	0.247	0.459
Self-Efficacy	-0.127	0.347	-0.088	0.718
EPN	-0.008	0.047	-0.040	0.860
Average Non-pain symptoms R^2 = 0.083; Adj. R^2 = - 0.126; *p* = 0.847				
Stress	0.462	0.596	0.1930	0.447
Neuroticism	0.138	1.346	0.0329	0.919
PCS	-0.448	0.552	-0.265	0.426
Self-Efficacy	-0.346	0.787	-0.106	0.664
EPN	-0.125	0.107	-0.265	0.253
Fluctuations in Non-pain symptoms R^2 = 0.211; Adj. R^2 = 0.031; *p* = 0.353				
Stress	0.411	0.278	0.341	0.154
Neuroticism	-0.872	0.628	-0.413	0.179
PCS	0.172	0.258	0.202	0.513
Self-Efficacy	-0.185	0.367	-0.112	0.619
EPN	0.034	0.050	0.141	0.508

* Statistically significant result
~ Regressions in which the linearity assumption was violated, but in which BoxCox transformations did not change the significance of predictors

Table 4. Results from six regressions run with central N400 for emotionally negative nouns as the main predictor variable of interest and average pain, fluctuations in pain, average mood, fluctuations in mood, average non-pain symptoms and fluctuations in non-pain symptoms as the outcome variables (N = 29)

	b	***b SE***	***β***	***p*-value**
Average pain: R^2 = 0.215; Adj. R^2 = 0.037; p = 0.339				
Stress	0.511	0.530	0.223	0.345
~Neuroticism	1.925	1.304	0.480	0.154
PCS	-0.649	0.475	-0.401	0.186
Self-Efficacy	-1.324	0.684	-0.422	0.066
N400	0.125	0.101	0.317	0.226
Fluctuations in Pain R^2 = 0.290; Adj. R^2 = 0.128; p = 0.156				
Stress	0.374	0.288	0.285	0.208
Neuroticism	-0.614	0.710	-0.267	0.397
PCS	-0.120	0.259	-0.129	0.648
Self-Efficacy	-0.777	0.373	-0.433	0.049*
N400	-0.037	0.055	-0.164	0.506
Average Mood R^2 = 0.390; Adj. R^2 = 0.251; p = 0.041*				
Stress	-0.882	0.365	-0.493	0.024*
Neuroticism	2.344	0.900	0.747	0.016*
PCS	-0.055	0.328	-0.044	0.868
Self-Efficacy	0.130	0.472	0.053	0.786
N400	0.132	0.070	0.427	0.070
Fluctuations in Mood R^2 = 0.088; Adj. R^2 = - 0.120; p = 0.828				
Stress	-0.028	0.263	-0.027	0.916
Neuroticism	-0.140	0.647	-0.076	0.831
PCS	0.182	0.236	0.244	0.449
Self-Efficacy	-0.110	0.339	-0.076	0.748
N400	-0.026	0.050	-0.143	0.609
Average Non-pain symptoms R^2 = 0.026; Adj. R^2 = - 0.196; p = 0.987				
Stress	0.418	0.617	0.175	0.505
Neuroticism	0.303	1.518	0.072	0.844
PCS	-0.274	0.553	-0.162	0.626
Self-Efficacy	-0.175	0.797	-0.053	0.828
N400	0.016	0.117	0.038	0.896
Fluctuations in Non-pain symptoms R^2 = 0.222; Adj. R^2 = 0.046; p = 0.317				
Stress	0.397	0.277	0.330	0.166
Neuroticism	-1.143	0.683	-0.542	0.108

PCS	0.103	0.249	0.121	0.682
Self-Efficacy	-0.223	0.358	-0.135	0.540
N400	-0.047	0.053	-0.226	0.382

* Statistically significant result
~ Regressions in which the linearity assumption was violated, but in which BoxCox transformations did not change the significance of predictors

Interaction effects

A pre-registered follow-up analysis of the result in which the N400 had a marginally significant incremental effect on average mood revealed that, in the whole sample ($N = 60$) there was no significant interaction between Group and N400 (see Table 5).

Table 5. Results from a regression (N = 60) involving average mood as the outcome, and central N400 for emotionally negative nouns, Group and the interaction between Group and central N400 as predictors

	b	***b SE***	***β***	***p*-value**
Average mood (N400*Group): $R^2 = 0.170$; Adj. $R^2 = 0.123$; $p = 0.019$*				
N400	0.072	0.055	0.207	0.197*
Group	-0.944	0.527	-0.324	0.079*
N400*Group	-0.021	0.091	-0.046	0.818

* indicates significant results

Supplementary Analysis

Results of the supplementary analyses (modelling the incremental effects of ERPs for symptom-related words), as seen in Appendix 4 and Appendix 5 were almost identical to the results for the main analysis. The main difference was that the Adjusted R-squared for the regression model that investigated the incremental effect of the EPN on average mood was marginally statistically significant. The effects of stress were also statistically significant and the effect of the EPN was marginally statistically significant. The model that examined the incremental effect of the N400 on mood had a significant adjusted R-squared. Again, it was stress and neuroticism that had a significant predictive effect and the effect of the N400 was

only marginally significant. As in the main analysis, there was no significant interaction between Group and the N400 (see Appendix 6).

Discussion

The current project investigated whether hypervigilance predicted FGID symptoms over and above known psychosocial variables. We specifically investigated whether unconscious or conscious attentional biases predicted averages and/or fluctuations in a) pain, b) mood, c) non-pain symptoms.

Afzal et al. (2006), Martin and Chapman (2010) and Chapman and Martin (2011) found evidence of unconscious attentional biases in FGID-sufferers. Ejova et al. (2021) also found evidence of unconscious attentional bias in FGID-sufferers. The meta-analyses of Crombez et al. (2013) and Schoth et al. (2012) found that chronic pain sufferers exhibited conscious attentional bias to stimuli when that stimuli was presented supraliminally. Ejova et al. (2021) also found that FGID sufferers exhibited a conscious attentional bias. The current project built upon these findings and examined the effect that attentional biases have upon FGID symptoms. As far as we are aware the current project is the first to use the results from an EEG recording with a properly delineated time course of attentional processes to predict FGID symptoms over and above known psychosocial predictors. These preliminary results call for further investigation into the relationship between hypervigilance and FGID symptoms.

Findings regarding the incremental effects of EEG-based indices of attention

Of the 12 regressions that were run, only one result, returned a significant Adjusted R-squared. The significant Adjusted R-squared result was found when we investigated whether the N400 (conscious attentional bias) had a predictive effect on average mood.

However, it was stress and neuroticism that had significant predictive effects, with the N400 being marginally statistically significant. The regression examining interaction effects between group and ERP was also non-significant. The absence of an interaction effect suggested that the effects of N400 on mood were not different for sufferers as compared to controls. This further means that the current results do not provide evidence that the n400 is an attentional predictor of mood related symptoms. Surprisingly, the relationship between the N400 and mood was positive.

One possible explanation of this is the heightened level of cytokines observed among FGID sufferers. Cytokines are small proteins released by cells that effect the communication between cells (Zhang & An, 2007). IBS and other FGIDs have been characterised not only by recurrent symptoms, but also by an over active hypothalamic-pituitary-adrenal axis (HPA). The HPA is the main endocrine system that houses vital links between the brain and gut and regulates cytokine levels. Dysregulation of the HPA system through stress, cognitive overload or psychological distress can lead to unregulated release of cytokines (Dinan et al., 2006). One of their roles within the body is to modulate the immune system and its responses. Accordingly, the unregulated actions of these cytokines have been associated with oxidative stress, where there are imbalances between production and accumulation of oxygen, which leads to inflammation and dysregulation in the brain, specifically the hippocampus and amygdala. The hippocampus and amygdala are key players in the regulation of mood and alterations in these areas have been associated with greater mood disorder. Simultaneously, raised cytokine levels have an excitotoxic effect which interferes with the amino acid glutamate which is involved in neuroplasticity and neurogenesis, both vital processes within the brain and maintenance mood and health (Mudyanadzo et al., 2018). Lastly, alterations in cytokine behaviour have been implicated in dysregulation of Corticotropin-Releasing-Factor (CRF) within the HPA axis. CRF is the main regulator of the

HPA which has many roles to play, one being the organiser of responses to stress. Hence, our responses to stress become dysregulated and prolonged, impacting upon mood (Slominski, 2009).

Similar to the findings above, Seminowicz et al. (2010) found that within a sample of female IBS sufferers brain networks that were concerned with attention and emotions were altered as a result of decreased areas of gray matter. Van Oudenhove et al. (2016) concluded that such changes in gray matter and the results found by Seminowicz et al. (2010) are indicative of the close relationship between mood and IBS. Consequently, the heightened attentional biases found in FGID sufferers may be maintaining the features of the disorder which alters areas of the brain which are involved in emotional control thusly, impacting upon mood.

Whilst the current project found evidence that suggested mood is not predicted by conscious attentional bias, the findings and reasons above propose that altered brain function, pathways and structure as a result of cytokine activity and FGID related distress is a possible explanation for how mood can be impacted. Hence, why the current project found a marginally significant predictive effect of conscious attention on mood.

Possible issues around measuring symptoms

For average pain and fluctuations in pain specifically, in both unconscious and conscious attentional bias, the subjective nature of pain may provide a possible explanation for why we received non-significant results. According to the Williams and Craig (2016) we associate pain with our personal experiences that are related to injury. However, pain often occurs in the absence of tissue damage, injury or pathological cause, although, is always unpleasant, meaning there is an emotional response. Such experiences of pain may be associated with psychological distress. The International Association of Pain has broadened

their definition of pain to include not only the sensory unpleasantness associated with pain but also the emotional unpleasantness. They have accepted that psychological and emotional distress can lead to pain in absence of tissue damage or pathophysiological causes. Including the emotional and psychological aspects of pain in their definition has allowed for pain to be discussed in a biopsychosocial context. Consequently, pain is no longer defined in terms of tissue damage and pathophysiological diagnosis. It has been broadened to also take into consideration social and psychological factors as well. Experiences of pain and how it is expressed can be influenced not only by exposure to and intensity of pain, but thoughts and feelings when in pain, how we communicate pain to others and how other people experience pain around us. Further, other influences that have been cited are the individual's thoughts when in pain that can impact experience, including emotions, decision making and social cognitions (Williams & Craig, 2016). Pain for each individual is based upon their individual experiences with sensory, emotional, cognitive and social events.

Pain has also been described as a multidimensional system whereby our experiences of pain are impacted upon by three dimensions (Moayedi & David, 2013). The first is the sensory-discriminative dimension which is the system involved in processing the intensity, location, duration and quality of the pain. The second dimension is the affective-motivational dimension which processes the unpleasantness and subsequent fight or flight response. The final dimension is the cognitive-evaluative dimension where the pain is appraised in terms of cultural values, context and cognitive states. Within this model of pain, we can see there is room for social and psychological experiences to impact. This model further highlights the multifaceted nature of pain (Moayedi & David, 2013).

Moreover, chronic pain and exacerbation of symptoms through attention, something that is inherent in FGIDs, changes the neuroplasticity of the brain. This can lead not only to

psychological issues such as depression or anxiety but it also alters the way that pain is perceived through modification of synaptic pathways (Yang & Chang, 2019).

The above literature outlining the nature pains means, for our project, that when people are asked a single question to rate their gastro pain on a scale of 0 to 10, the ratings of pain may be measuring numerous constructs or dimensions. Therefore, the pain rating may not be valid. Further, due to the subjective nature of pain, people with similar pain scores may be experiencing completely different pain. This makes pain difficult to accurately measure and be predicted, hence, how pain is measured needs to be carefully considered.

Issues with using daily diaries

Surprisingly, in the majority of regressions the known psychosocial predictors of FGIDs did not have a predictive effect on pain, mood or non-pain symptoms. One possible explanation is the use of the single item daily diary. The daily diary asked one question for each construct, those constructs being mood, pain and non-pain symptoms, with scores for interference in daily life being recorded as well. A study conducted by Hoeppner et al. (2011) found that single item questionnaires had convergent and discriminative validity as well as predicative validity. It must be mentioned that this study was conducted on self-efficacy in predicting relapse in young adults. However, there has been criticism of single item measures that may contribute to the predictor variables not predicting the daily diary scores, both fluctuations and averages. Single item questionnaires are criticised for their lack of internal consistency, are more vulnerable to random measurement errors and are prone to biases in meaning and interpretation (Hoeppner et al., 2011).

For the current study, this means that the single item on pain for example “how would you rate any gastro pain you’ve had today” could be interpreted in a multitude of ways. Gastro pain may mean different things to different people and they interpret and respond in a way that is unique to them. They may interpret gastro pain and include pain that is higher up

in the chest or throat and not limit it to the gastrointestinal tract. They may include discomfort or non-pain symptoms. Typically, multiscale items are designed to cover a greater range of the construct so as to cover more interpretations. Moreover, as discussed above, with a multifaceted construct such as pain a single question daily diary that specifically asks about gastro pain may be missing important information regarding other features of pain or measuring multiple constructs of pain, thereby reducing validity. Further, in the current study scores on the GSRS were significantly correlated with ERPs, although correlations were small. This may indicate that there is a relationship between attention and FGID symptoms that just was not captured by the daily diary measurement. As discussed above single question daily diaries lack internal validity and may measure multiple construct of pain, mood and non-pain related symptoms with a single question.

Possible issues around measuring attention

The current project found no evidence that unconscious attentional bias predicted any of the chosen FGID symptoms and only marginally statistically significant evidence of conscious attentional biases impacting upon mood. A possible reason considered by Chapman and Martin (2011) is that responses to the words used to capture hypervigilance could be affected by personal experience. They explain that symptom-related words were intuitively thought to have elicited greater responses in FGID-sufferers because they were relevant to their situation and triggered a response within them. This however, was not the case for either Ejova et al. (2021) nor Chapman and Martin (2011). Chapman and Martin (2011) posit that is may be due to the fact that symptoms are not generalised to everyone, in that, each FGID sufferer may experience different symptoms or variations of symptoms that may not have been present in the words that they were exposed to during the EEG sessions. They may not have any experience with any of the chosen symptom related words and

therefore pay limited attention or none at all. This explanation from Chapman and Martin (2011) can extend to emotionally negative words. For example, one emotionally negative word used in the current project was 'centipede'. Unless you have a connection with this word or an experience with it, then it may not elicit an emotionally negative response within you and cause you to show any attentional bias. Being unable to find words that are specific to each individual person to extract valid attention for everyone is going to affect results. For the current project this means hypervigilance scores that were captured may not have been optimal and therefore could not adequately predict any of our outcome variables.

In this paper and within the wider literature unconscious attention has been thought to be automatic, implying there are few underlying processes affecting how unconscious attention is elicited and can be altered. For example, Van Oudenhove et al. (2010) found that brain regions that were associated with top-down processing and emotional and arousal regulation were not engaged during gastric distension trials. This led them to conclude that processing of stimuli in FGID sufferers was anxiety driven, unconscious and did not involve top-down modulation. However, there is some evidence that indicates that unconscious attention can be goal orientated or altered based upon what is important to that individual (Prasad & Mishra, 2019). Ansorge et al. (2009) during a selective attention trial study where participants had to look for a coloured target after a cue-display found that the activation of the N2pc ERP which is involved in attention allocation was active for both sham and real trials. Ansorge et al. (2009) and Prasad and Mishra, (2019) argue the activation of this brain region during both sham and real trials was evidence of goal-orientated or top-down processing during unconscious stages of attention.

Within this context, the fear-avoidance and vigilance-avoidance model may aid in explaining how FGID-sufferers and their goals change their unconscious attentional bias to align with their goals. Within these models' sufferers appraise pain using catastrophic

thinking and develop a fear for the stimuli or activity. This results in avoidance of activities or stimuli and a hypervigilance. As a result of this process the sufferer is reinforcing avoidant behaviour to stimuli and is likely to overestimate the effects of the stimuli or activity in terms of causing them pain (Cosio, 2019). Consequently, if the sufferer's goal is to avoid stimuli and it is important to them to avoid that stimuli, as reinforced by their avoidant behaviour, it is entirely possible according to the literature that argues that attention can be altered based upon the goals of the person, that sufferers adapt their attention to avoid certain stimuli. This may affect how unconscious attention is elicited when capturing it with EEG recordings and affects how it predicts FGID symptoms.

Ejova et al. (2021) also theorised that heightened levels of unconscious attention, as indicated by higher EPN, might be associated with passivity. Sufferers may attend to a stimuli and engage in a passive coping strategy to avoid anxiety and other negative cognitions associated with the stimuli. In this way, unconscious attention may impact upon conscious processing and may even alter the later stages of unconscious attention. Passivity according to Walker et al. (2005) is associated with a restriction in activities and catastrophic thinking.

A further explanation for why the predictor variables were not predictive of FGID symptoms is the variability of FGIDs (Drossman, 2016). As we can see in the biopsychosocial model there are a vast number of systems and factors that influence FGID symptoms severity and onset that knowing exactly which ones are influencing which individual and their symptoms and to then control for them is something that needs further research (Van Oudenhove et al., 2016).

Findings regarding self-efficacy and fluctuation in mood

When we investigated whether conscious attention predicted fluctuations in mood, it was self-efficacy that had a predictive effect. There was a significant negative relationship,

meaning that self-efficacy may play a role in mitigating fluctuations in mood for sufferers. As mentioned self-efficacy is the belief that one can attain a goal (Dąbek-Drobny et al., 2020). Higher scores of self-efficacy can enhance human accomplishment and wellbeing. Tahmassian and Moghadam (2011) state that this outlook helps people accomplish more, reduces stress and reduces the risk of developing mood disorders. For FGID sufferers this may mean that those who have higher self-efficacy have greater regulation of their mood because they believe in themselves and their ability to overcome obstacles. They are consistent in their approaches to potentially challenging situations, thereby reducing fluctuations. In this way, challenging situations, such as managing chronic symptoms, are perceived to be manageable and within their power to overcome. They do not appraise the situation as too difficult and become vulnerable to stress and mood alterations as a result. Consequently, in our results we see a negative relationship between self-efficacy and fluctuations in mood.

Strengths

The current project was pre-registered on the Open Science Framework. The purpose of this was to maintain our academic integrity and credibility and avoid any potential for data misuse. Moreover, pre-registration aids in reducing unintentional inflation of false positive results and helps distinguish between exploratory studies and hypothesis testing (Forstmeier et al., 2017; Nosek et al., 2018). Further, when choosing the predictor variables there was an extensive process to determine which ones to choose. After reviewing the literature, the variables that were consistently found to be predictive of FGID symptoms in large studies were selected. Following this, we carefully contemplated how many predictor variables to include to make sure we could analyse the data meaningfully considering the small sample size.

Further, single question daily-diary surveys are prospective measures as opposed to retrospective and have been found to have convergent, discriminative and predicative validity.

Limitations

In the current project there were 29 female participants from Macquarie University, Sydney. Having a larger sample size is ideal as having such a small sample size meant the power of the study was reduced and increased the margin of error.

As discussed above, single item measures have strengths and limitations and as a result may not best capture FGID symptoms. Future research should consider the validity of single item measures as they may not capture what they are designed to capture. Specifically, the multifaceted nature of pain needs to be considered when choosing a tool in which to measure the construct of pain. An alternative is the GSRS. The benefits of this scale are that it captures more dimensions of pain. Further, a study conducted by Kulich et al. (2008), found that the GSRS when used as a tool to aid in diagnosis of FD was adequate with appropriate internal consistency reliability and test-retest reliability. In the current project the GSRS was moderately correlated with averages in pain, mood, non-pain-symptoms, fluctuations in non-pain symptoms and with ERPs. Future studies may consider this scale as an alternative to the single question daily diary approach.

Another limitation that is inherent in FGID studies, due to the unknown aetiology, is the presence of confounding variables. Being explained in a biopsychosocial context means that there are numerous systems and factors influencing FGID symptomology.

Implications

The findings from the current study may direct future research towards different measures for capturing FGID symptoms. The preliminary findings from this study indicate that more

research needs to be undertaken to better understand the relationship between mood and FGID symptoms with the use of daily diaries. Moreover, the predictive effect of self-efficacy implies a need to include adaptive coping strategies in future studies of attention. Adaptive coping could be associated with attention as opposed to maladaptive coping strategies.

From a clinical perspective, marginally statistically significant evidence of conscious attention predicting mood means that through cognitive behavioural therapy, conscious attentional patterns may be altered to become more adaptive. By challenging the catastrophic and fear inducing thoughts associated with FGID symptoms, cognitions surrounding them can be changed, thereby reducing avoidant behaviour and catastrophic thinking and alleviating cognitive distress. Alternatively, Van Oudenhove et al. (2016) suggest exposure therapy to help sufferers confront their symptoms and adapt their conscious and unconscious processes and fears towards negative stimuli such as FGID symptoms or symptom inducing activity.

Directions for further studies

Future studies may consider alterations in how the words used to capture attention and measure hypervigilance are identified. In the current project, symptom-related words were selected by research team consensus, with emotionally negative and neutral words being selected via corpora of English text. A more rigorous search method may be incorporated to select more appropriate words. In choosing more appropriate words future studies might consider specific FGID studies or get diary entries from sufferers and consider the frequency of words used.

The current project investigated the averages and fluctuations of pain, mood and non-pain symptoms. Future projects may consider how those pain levels change over time. Having a longer period that sufferers fill out the daily for and then measuring and tracking

the increase or decrease of symptoms severity may reveal further information regarding symptomology. It may allow participants and researchers to point to periods in time or events that occurred where symptom severity increased and/or decreased. This information may reveal how not only biopsychosocial factors influence but how personal perceptions and experiences impact upon symptomology.

To gather information on changes over time future researchers may consider administering their symptom surveys using ecological momentary assessment. This approach administers the chosen questions aimed at gathering information on symptoms multiple times a day. This minimises any biases that come with memory. Further, ecological momentary assessments have been found to minimise exaggeration of symptoms that has been found to occur when retrospective, once a day questionnaire are used (Mujagic et al., 2015). Using this type of assessment will also allow researchers to gather more information and use it to measure not only how one symptom may change over time but also how symptoms relate to each other and how strong that relationship is. This will aid in investigating, for example, the relationship between mood and pain.

Another consideration for a future study is a distinction made by Crombez et al. (2013). They conclude that attentional bias in chronic pain sufferers has always been difficult to identify, generate or replicate. One reason that they posit as a possible explanation and should be considered in the future is the difference between words that symbolically represent pain and cues of impending pain. They found that attention to predictive cues of impending pain that had been classically conditioned had a greater effect size when compared to attention paid to words. What this finding may suggest is that attention to stimuli in the form of words may not be capturing attention appropriately compared to the impending threat of pain which may represent a better method to capture attention.

When future studies consider how best to capture unconscious attention with regard to goal-orientated processing, Prasad and Mishra (2019) identified variables that need to be contemplated when presenting the cue. They identified that the type of cue, whether it is has abrupt onset (i.e., appears suddenly) or visually stands out in a scene, the relationship between the cue and the target feature and whether the task calls for engagement or attention capture can all impact upon unconscious attention to cues. Manipulation of these variables may aid in determining how best to elicit unconscious attention, therefore, giving a more valid and reliable measure of unconscious attention.

Another consideration for the future is the role that the gut microbiome plays within the brain-gut axis. Past literature has implicated altered microbiomes of sufferers as a key contributor to symptoms. Microorganisms engage in bidirectional communication with the brain via neural endocrine and immune pathways and altered composition of the microorganisms in the microbiome have been associated with IBS symptomology (Van Oudenhove et al., 2016). The bidirectional communication means that there is scope for microbiomes to play a role in affecting the brain, its neuroplasticity and emotional centres. Administration of probiotics to fix the altered composition of the microbiome has been found to alter central processing as well as resting brain connectivity. Moreover, recent studies, by Tengeler et al. (2020) Checa-Ros et al. (2021) and Stevens et al. (2019) have found that the gut microbiome plays a role in Attention-Deficit Hyperactivity Disorder. The altered microorganism within the microbiome of the gut and their bidirectional communication with the brain has been linked with possible effects on attention. These preliminary findings indicate that it could be useful to conduct a project examining the relationship between the presence of microbes relating to attention, the ERPs examined in the current project and FGID symptoms.

The current study had all-female participants. Kim & Kim (2018) identified that there are gender differences in the expression of FGID symptoms and that females are more likely to seek medical care and be available for clinical studies. A replication study with an all-male cohort should be undertaken to examine the role gender plays when investigating whether hypervigilance predicts FGID symptoms.

Conclusion

The aim of this study was to investigate whether hypervigilance predicted FGID symptoms over and above known psychosocial predictors. Our results indicated a marginally significant effect of conscious attentional bias in FGID-sufferers when predicting mood. Past literature has found that there are conscious and unconscious attentional biases in FGID sufferers. There have been four papers that have identified that sufferers do exhibit unconscious attentional bias to threat Ejova et al. (2021) Afzal et al. (2006), Martin and Chapman (2010) and Chapman and Martin (2011) with a further two meta-analyses identifying conscious attentional bias Crombez et al. (2013) and Schoth et al. (2012) with Ejova et al. (2021) also finding evidence of conscious attentional bias.

The current project was the first to use the results from an EEG recording with a properly delineated time course of attentional processes to predict FGID symptoms over and above known psychosocial variables. With only 29 participants, a future study should have a larger sample size. Moreover, a more appropriate measure that captures the various dimension and constructs of symptoms needs to be considered, the singular daily diary questions used in the current project may not have captured correct constructs regarding our outcome variables. Further research may aid in understanding the aetiology of FGIDs and identifying the specific role that hypervigilance plays. FGIDs are currently treated by trial and error that is aimed at reducing symptoms and not curing the disorder (Tally, 2020).

Identifying the relationship between hypervigilance and FGID symptomology will aid general practitioners and psychologists in creating more informed treatment plans that aim to reduce symptoms by targeting and changing behaviour regarding attention. A more complete picture of the aetiology of FGIDs through further research will help clinicians move away from a trial and error approach towards a cure.

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Appendix 1: Online screener survey.

Survey intro

Project title

An EEG study of health

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Professor Mike Jones

[redacted]

Thank you for expressing interest in this study, which is a screener survey for a larger study on how stomach symptoms might be tracked through EEG technology (a cap connected to electrodes) and a daily symptom diary (completed using a smartphone app). This short (5-minute) survey about your tummy symptoms will help us determine whether you are a perfect fit for us as a participant in the larger study. We will contact you by e-mail within 48 hours to let you know whether we are able to invite you into the EEG study. If you are invited, we will provide you with a password for signing up to a preliminary online survey.

Before starting this survey, please check that you meet the eligibility criteria in the study description on SONA.

The EEG study will take 210 minutes (150 for the EEG session itself; 30 minutes for a pre-session online survey competed at home; and 30 minutes for a post-session mobile phone survey: 2 minutes each day for 14 days). You will receive 7 credits for PSYC 104 for taking part.

It is possible that, in asking about personal issues around pain management, some of the questions in this survey might cause distress. We ask that you advise us by e-mail (anastasia.ejova@mq.edu.au) if this occurs, so that we can discuss what services are available on campus for finding the appropriate referral pathway. Should you wish to

develop over time, so, in a completely voluntary part of the study, we will send you an e-mail in 3 months' and 6 months' time, each time asking 10 minutes-worth of questions about your current symptoms.

It is possible that, in asking about personal issues around pain and anxiety management, some of the survey and diary questions might cause distress. We ask that you advise us by e-mail (anastasia.ejova@mq.edu.au) if this occurs, so that we can discuss what services are available on campus for finding the appropriate referral pathway. Should you wish to personally research referral pathways, we recommend contacting Campus Wellbeing (9850 7497; campuswellbeing@mq.edu.au) or your GP.

Ethics, confidentiality and dissemination of findings
Participation in this study is entirely voluntary. You are not obliged to participate and if you decide to participate, you are free to withdraw at any time without having to give a reason and without consequence.

Any information or personal details gathered in the course of the study are confidential, except as required by law. No individual will be identified in any publication of the results. Access to the data will be restricted to Professor Jones and approved co-investigators.

The findings of this study may be published in a peer-reviewed journal. If you leave us your e-mail address, we will forward you copies of publications arising from the study. Even before data are published (which can take some months), you are welcome to contact Dr Ejova and Professor Jones (anastasia.ejova@mq.edu.au; mike.jones@mq.edu.au) with any questions.

The ethical aspects of this study have been approved by the Macquarie University Human Research Ethics Committee. If you have any complaints or reservations about any ethical aspect of your participation in this research, you may contact the Committee through the Director, Research Ethics & Integrity (telephone (02) 9850 7854; email ethics@mq.edu.au). Any complaint you make will be treated in confidence and investigated, and you will be informed of the outcome.

Informed consent to Part A through commencement of the survey
By proceeding to the survey, you indicate that you agree to take part in Part A of this study, understanding the information above. Proceeding to the survey also indicates that any questions you might have at this point have been answered to your satisfaction. We remind you that you can withdraw from participation in this research at any time without consequence.

I have read the information above and consent to taking part in Part A of this study:

personally research referral pathways, we recommend contacting *Campus Wellbeing* (9850 7497; campuswellbeing@mq.edu.au) or your GP.

Ethics, confidentiality and dissemination of findings
Participation in this study is entirely voluntary. You are not obliged to participate and if you decide to participate, you are free to withdraw at any time without having to give a reason and without consequence. Just exit the browser.

Any information or personal details gathered in the course of the study are confidential, except as required by law. No individual will be identified in any publication of the results. Access to the data will be restricted to Professor Jones, Dr Ejova and approved co-investigators.

The preliminary screener questions begin straight after the first question, where we ask you to consent to taking part in this screener questionnaire.

I have read the information above and consent to taking part in this screener survey:

Yes
No

Demographics

In this first part of the survey, we ask a few short questions about your personal details and medical history. Any information you provide will remain completely confidential.

Please enter the following details to ensure that we can contact you with an invitation code to enrol in the study.

E-mail address
Name
Student ID (OneID)

Please indicate your age.

Are you currently on any medication?

Yes

No

Please list the medication(s) you are taking.

Are you currently diagnosed with a mental illness (e.g., depression, anxiety, or schizophrenia) by a registered health practitioner?

Yes

No

Please indicate which illness(es) you are diagnosed with.

Have you ever (in the past, but not currently) been diagnosed with a mental illness (e.g., depression, anxiety, or schizophrenia) by a registered health practitioner?

Yes

No

Please indicate which illness(es) you have been diagnosed with.

Have you ever been diagnosed with a chronic pain condition or major illness (e.g., rheumatoid arthritis, Coeliac Disease, or cancer)?

Yes

No

Please list the conditions you have been diagnosed with below.

GI symptoms 1

We now move on to a few quick questions about your gastrointestinal symptoms.

In the last 3 months, how often did you feel so full after a regular-sized meal (the amount you normally eat) that it interfered with your usual activities?

Never

Fewer than 1 day a month

1 day a month

2-3 days a month

1 day a week

2-3 days a week

Most days

Every day

Multiple times per day or all the time

Has it been 6 months or longer since you started having these episodes of fullness after meals that was severe enough to interfere with your usual activities?

No

Yes

GI symptoms 2

In the last 3 months, how often were you unable to finish a regular-sized meal because you felt too full?

Never

Fewer than 1 day a month

1 day a month

2-3 days a month

1 day a week

2-3 days a week

Most days

Every day

Multiple times per day or all the time

Has it been 6 months or longer since you started having these episodes of feeling too full to finish regular-sized meals?

No

Yes

GI symptoms 3

In the last 3 months, how often did you have pain or burning in the middle part of your upper abdomen (above your belly button but not in your chest) that was so severe that it interfered with your usual activities?

Never

Fewer than 1 day a month

1 day a month

2-3 days a month

1 day a week

2-3 days a week

Most days

Every day

Multiple times per day or all the time

Has it been 6 months or longer since you started having this pain or burning in the middle part of your upper abdomen?

No

Yes

GI symptoms 4

In the last 3 months, how often did you have pain anywhere in your abdomen?

Never

Fewer than 1 day a month

1 day a month

2-3 days a month

1 day a week

2-3 days a week

Most days

Every day

Multiple times per day or all the time

Below, are some questions about times when you have had this abdominal pain.

	0% (Never)	10%	20%	30%	40%	50%	60%	70%	80%	90%	100% (Always)
How often (i.e.., in what percentage of times that you had pain in your abdomen) did this pain happen close in time to a bowel movement (i.e., just before, during or soon after)?	○	○	○	○	○	○	○	○	○	○	○
How often did your stools become either softer than usual or harder than usual when you had this pain?	○	○	○	○	○	○	○	○	○	○	○
How often did your stools become either more frequent than usual or less frequent than usual when you had this pain?	○	○	○	○	○	○	○	○	○	○	○

Has it been 6 months or longer since you started having this pain?

No

Yes

GSRS

This survey contains questions about how you have been feeling and what is has been like DURING THE PAST WEEK. For each of the following questions, please choose the best choice that applies to you and your situation from the following options: 1 = No discomfort at all, 2 = Minor discomfort, 3 = Mild discomfort, 4 = Moderate discomfort, 5 = Moderately severe discomfort, 6 = Severe discomfort, 7 = Very severe discomfort

	1	2	3	4	5	6	7
Have you been bothered by RUMBLING in your stomach during the past week? (Rumbling refers to vibrations or noise in the stomach.)	○	○	○	○	○	○	○
Have you ever been bothered by PAIN OR DISCOMFORT IN YOUR UPPER ABDOMEN OR THE PIT OF YOUR STOMACH during the past week?	○	○	○	○	○	○	○
Have you been bothered by HEATBURN during the past week? (By heartburn we mean an unpleasant stinging or burning sensation in the chest.)	○	○	○	○	○	○	○
Have you been bothered by DIARRHEA during the past week? (Diarrhea refers to a too frequent emptying of the bowels.)	○	○	○	○	○	○	○
Have you been bothered by BURPING during the past week? (Burping refers to bringing up air or gas from the stomach via the mouth, often associated with easing a bloated feeling.)	○	○	○	○	○	○	○
	1	2	3	4	5	6	7

	1	2	3	4	5	6	7
When going to the toilet during the past week, have you had the SENSATION OF NOT COMPLETELY EMPTYING THE BOWELS? (This feeling of incomplete emptying means that you still feel a need to pass more stool despite having exerted yourself to do so.)	○	○	○	○	○	○	○
Have you been bothered by an URGENT NEED TO HAVE A BOWEL MOVEMENT during the past week? (this urgent need to go to the toilet is often associated with a feeling that you are not in full control.)	○	○	○	○	○	○	○
Have you been bothered by ACID REFLUX during the past week? (By acid reflux we mean the sensation of regurgitating small quantities of acid or flow of sour or bitter fluid from the stomach up to the throat.)	○	○	○	○	○	○	○
Have you been bothered by NAUSEA during the past week? (By nausea we mean a feeling of wanting to throw up or vomit.)	○	○	○	○	○	○	○
Have you been bothered by CONSTIPATION during the past week? (Constipation refers to a reduced ability to empty the bowels.)	○	○	○	○	○	○	○
	1	2	3	4	5	6	7

	1	2	3	4	5	6	7
Have you been bothered by PASSING GAS OR FLATUS during the past week? (Passing gas or flatus refers to the need to release air or gas from the bowel, often associated with easing a bloated feeling.)	○	○	○	○	○	○	○
Has your stomach felt BLOATED during the past week? (Feeling bloated refers to swelling often associated with a sensation of gas or air in the stomach.)	○	○	○	○	○	○	○
Have you been bothered by HUNGER PAINS in the stomach during the past week? (This hollow feeling in the stomach is associated with the need to eat between meals.)	○	○	○	○	○	○	○
Have you been bothered by LOOSE STOOLS during the past week? (If your stools (motions) have been alternately hard and loose, this question only refers to the extend you have been bothered by the stools being loose.)	○	○	○	○	○	○	○
Have you been bothered by HARD STOOLS during the past week? (if your stools (motions) have been alternately hard and loose, this question only refers to the extend you have been bothered by the stools being hard.)	○	○	○	○	○	○	○

Thanks

Thank you! We will be in contact within 48 hours with information about whether you qualify for the EEG study and details on how to sign up for it after completing a preliminary

Appendix 2. List of relevant Rome IV criteria for IBS and FD. Source: Ejova et al. (2021)

FGID	Criterion number	Criterion description	Meet criterion if:
IBS	C1.1	Recurrent abdominal pain	Pain occurs at least once a week
	C1.2	Pain correlates	Pain is associated with two or more of the following: • defecation on at least 30% of occasions • change in form (appearance) of stool on at least 30% of occasions • change in frequency of stool on at least 30% of occasions
	C1.3	Symptom onset	Pain is experienced at least six months prior to survey
	A diagnosis requires meeting all three diagnostic criteria.		
FD: Postprandial distress syndrome	B1a.1	Bothersome postprandial fullness (i.e., severe enough to impact on usual activities)	Fullness is experienced at least 2-3 days per week for the last three months, and began at least six months prior to survey
	B1a.2	Bothersome early satiation (i.e., severe enough to prevent finishing a regular size meal)	Early satiation is experienced at least 2-3 days per week for the last three months, and began at least six months prior to survey
	(B1a.3) Not assessed	Lack of comorbid organic condition	There is no evidence of organic, systemic, or metabolic disease that is likely to explain the symptoms on routine investigations including at upper endoscopy.
	A diagnosis requires meeting the third diagnostic criterion and one of the first two, although, in the current study, the third criterion was not formally assessed, so meeting one of the first two criteria while reporting no current comorbid conditions was sufficient.		
FD: Epigastric pain syndrome	B1b.1	Bothersome epigastric pain (i.e., severe enough to impact on usual activities)	Epigastric pain is experienced at least weekly for the last three months
	B1b.2	Bothersome epigastric burning (i.e., severe enough to impact on usual activities)	Epigastric burning is experienced at least weekly for the last three months
	B1b.4	Symptom onset	Pain or burning began at least six months prior to survey

(B1b.3) Not assessed	Lack of comorbid organic condition	There is no evidence of organic, systemic, or metabolic disease that is likely to explain the symptoms on routine investigations including at upper endoscopy.

A diagnosis requires meeting the last two diagnostic criteria and one of the first two. In the current study, the third criterion was not formally assessed, so meeting one of the first two criteria and the fourth while reporting no current comorbid conditions was sufficient.

Appendix 3: Participants' psychological, clinical characteristics and daily dairy scores, by group, as well as details of associated measures. Adapted from: Ejova et al. (2021)

Characteristic and measure	Internal consistency (α)	*M*(*SD*) or percentage	
		Healthy controls (n = 28)~	FGID-sufferers (n = 29)#
Age		22.533 (7.664)	21.700 (6.577)
Past diagnosed psychiatric disorder (% yes)		13% (n = 4)	27% (n = 8)
Past chronic illness (% yes)		3% (n = 1)	0%
Currently diagnosed functional gastrointestinal condition (% yes)		23% (n = 7)	0%
Gastrointestinal symptom severity (past week): Gastrointestinal Symptom Severity Index (Kulich et al., 2008)	.926	1.902 (0.959)	3.297 (1.119)***
Depression symptoms: Depression, Anxiety and Stress Scale 21 (Lovibond & Lovibond, 1995)	.872	0.291 (0.362)	0.655 (0.534)**
Anxiety symptoms: Depression, Anxiety and Stress Scale 21	.725	0.403 (0.356)	0.724 (0.499)**
Stress: Depression, Anxiety and Stress Scale 21	.840	0.694 (0.409)	1.192 (0.621)**
Neuroticism: International Personality Item Pool (Goldberg, 1999)	.841	2.425 (0.438)	2.648 (0.36)*
Pain catastrophising: Pain Catastrophising Scale (Sullivan,	.945	1.217 (0.896)	1.288 (0.893)

Bishop & Pivik, 1995)			
Somatic symptoms: PHQ-15 (Kroenke, Spitzer & Williams, 2002)	NA (categorical responses)	3.214 (2.544)	7.241 (4.050)***
Health anxiety: Whiteley Index (Pilowsky, 1967)	NA (binary responses)	4.107 (3.485)	6.897 (3.040)***
Coping through denial: Brief Cope (Carver, 1997)	.564	1.232 (0.419)	1.259 (0.592)
Coping through distraction: Brief Cope	.799	2.893 (0.798)	2.810 (0.87)
Coping through disengagement: Brief Cope	.786	1.268 (0.419)	1.448 (0.632)
Coping through venting: Brief Cope	.548	2.107 (0.774)	2.241 (0.83)
Coping through self-blame: Brief Cope	.827	1.911 (0.806)	2.603 (1.081)**
Coping through substance-use: Brief Cope	.974	1.196 (0.438)	1.586 (0.856)*
Active coping: Brief Cope	.710	3.000 (0.707)	2.724 (0.714)
Coping through planning: Brief Cope	.790	2.875 (0.835)	2.793 (0.861)
Coping through acceptance: Brief Cope	.651	3.000 (0.745)	2.638 (0.706)
Coping through seeking instrumental support: Brief Cope	.889	2.893 (0.809)	2.586 (1.009)
Coping through seeking emotional/ social support: Brief Cope	.869	2.839 (0.903)	2.741 (0.96)
Coping through positive reframing: Brief Cope	.528	2.750 (0.811)	2.586 (0.835)
Coping through humour: Brief Cope	.901	1.893 (1.075)	2.017 (0.84)

Self-efficacy: Generalized Self-Efficacy Scale (Schwarzer & Jerusalem, 1995)	.863	2.786 (0.570)	2.783 (0.452)
SF-12 quality of life: physical (Ware, Kosinski & Keller, 1995)		58.780 (5.593)	50.207 (10.639)
SF-12 quality of life: mental		38.307 (5.333)	33.523 (7.888)
Daily Dairy Question: average pain.		0.843 (1.342)	2.672 (1.396) ***
Daily Dairy Question: fluctuations in pain		0.961 (0.944)	2.084 (0.815) ***
Daily Dairy Question: average mood		6.284 (1.623)	5.212 (1.108) **
Daily Dairy Question: Fluctuations in mood		1.632 (0.874)	1.737 (0.659)
Daily Dairy Question: average non-pain symptoms		0.900 (1.355)	2.660 (1.465) ***
Daily Dairy Question: Fluctuations in non-pain symptoms		0.931 (1.104)	1.920 (0.760) ***
EPN event related potential (Symptom-related Words)		5.250 (4.050)	6.830 (3.470)
EPN event related potential (Emotionally Negative Words)		4.590 (3.800)	6.820 (3.160)*
EPN event related potential (Neutral Words)		5.320 (3.780)	6.490 (3.260)
N400 event related potential (Symptom-related Words)		1.700 (4.330)	1.730 (3.750)
N400 event related potential (Emotionally Negative Words)		1.420 (3.900)	0.830 (3.630)

N400 event related potential (Neutral Words)	0.670 (4.510)	1.700 (3.660)

p-value in independent-samples *t*-test or chi-square test for counts: ***$p \leq .001$, **$.001 < p \leq .01$, *$.01 < p \leq .05$

~Due to a technical issue, two participants in this group were missing measures of all psychosocial variables.

#Due to a technical issue, one participant in this group was missing measures of all psychosocial variables, and an additional participant was missing a pain catastrophizing score.

Appendix 4: Results from six regressions run with occipital EPN for symptom related nouns as the main predictor variable of interest and average pain, fluctuations in pain, average mood, fluctuations in mood, average non-pain symptoms and fluctuations in non-pain symptoms as the outcome variables.

	b	***b SE***	***β***	***p*-value**
Average pain: R^2 = 0.161; Adj. R^2 = -0.030; p = 0.535				
Stress	0.448	0.555	0.192	0.428
Neuroticism	1.256	1.230	0.313	0.319
PCS	-0.731	0.512	-0.448	0.167
Self-Efficacy	1.317	0.722	-0.418	0.082
EPN	-0.011	0.088	-0.021	0.905
Fluctuations in pain: R^2 = 0.313; Adj. R^2 = 0.157; p = 0.118				
Stress	0.333	0.288	0.254	0.259
Neuroticism	-0.400	0.637	-0.174	0.537
PCS	-0.014	0.265	-0.015	0.958
Self-Efficacy	-0.702	0.374	-0.391	0.074
EPN	0.051	0.046	0.215	0.281
Average mood: R^2 = 0.371; Adj. R^2 =0.228; p = 0.055				
Stress	-1.089	0.376	-0.608	0.008*
Neuroticism	1.676	0.834	0.534	0.057
PCS	0.051	0.347	0.040	0.885
Self-Efficacy	0.320	0.489	0.130	0.520
EPN	0.101	0.060	0.313	0.106
Fluctuations in mood: R^2 = 0.081; Adj. R^2 = -0.128; p = 0.853				
Stress	-0.029	0.267	-0.027	0.915
Neuroticism	0.002	0.592	0.001	0.997
PCS	0.218	0.246	0.293	0.386
Self-Efficacy	-0.094	0.348	-0.065	0.790
EPN	0.013	0.043	0.070	0.759
Average non-pain symptoms: R^2 = 0.076; Adj. R^2 = -0.134; p = 0.870				
Stress	0.541	0.609	0.226	0.384
Neuroticism	0.185	1.349	0.044	0.892
PCS	-0.464	0.561	-0.275	0.417
Self-Efficacy	-0.345	0.792	-0.105	0.667
EPN	-0.106	0.097	-0.247	0.284
Fluctuations in non-pain symptoms: R^2 = 0.222; Adj. R^2 = 0.046; p = 0.317				
Stress	0.376	0.281	0.312	0.195
Neuroticism	-0.880	0.623	-0.418	0.172
PCS	0.195	0.259	0.230	0.459

Self-Efficacy	0.167	0.365	-0.101	0.651
EPN	0.040	0.045	0.184	0.384

* Indicates a significant result

Note. *The chief effect of interest was the incremental effect of the occipital EPN on average pain, fluctuations in pain, average mood, fluctuations in mood, average non-pain symptoms and fluctuations in non-pain symptoms. The control variables were stress, neuroticism, pain catastrophising (PCS) and self-efficacy.*

Appendix 5: Results from six regressions run with central N400 for symptom related nouns as the main predictor variable of interest and average pain, fluctuations in pain, average mood, fluctuations in mood, average non-pain symptoms and fluctuations in non-pain symptoms as the outcome variables.

Average pain: R^2 = 0.194; Adj. R^2 = 0.011; p = 0.410				
Stress	0.401	0.534	0.175	0.460
Neuroticism	1.506	1.232	0.376	0.235
PCS	-0.565	0.503	-0.349	0.273
Self-Efficacy	-1.181	0.704	-0.376	0.108
N400	0.082	0.086	0.231	0.347
Fluctuations in pain: R^2 = 0.278; Adj. R^2 = 0.113; p = 0.179				
Stress	0.402	0.290	0.307	0.179
Neuroticism	-0.457	0.668	-0.199	0.501
PCS	-0.125	0.272	-0.135	0.650
Self-Efficacy	-0.804	0.382	-0.448	0.047
N400	-0.014	0.046	-0.066	0.773
Average mood: R^2 = 0.378; Adj. R^2 = 0.237; p = 0.049*				
Stress	-1.005	0.367	-0.561	0.012*
Neuroticism	1.955	0.847	0.623	0.031*
PCS	0.065	0.345	0.051	0.854
Self-Efficacy	0.306	0.484	0.124	0.534
N400	0.104	0.059	0.374	0.090
Fluctuations in mood: R^2 = 0.078; Adj. R^2 = -0.132; p = 0.863				
Stress	-0.015	0.263	-0.014	0.954
Neuroticism	0.019	0.606	0.010	0.975
PCS	0.208	0.247	0.279	0.410
Self-Efficacy	-0.105	0.346	-0.073	0.764
N400	0.007	0.042	0.043	0.868
Average non-pain symptoms: R^2 = 0.025; Adj. R^2 = -0.196; p = 0.988				
Stress	0.405	0.614	0.169	0.516
Neuroticism	0.244	1.416	0.058	0.865
PCS	-0.268	0.578	-0.158	0.648
Self-Efficacy	-0.160	0.809	-0.049	0.845
N400	0.008	0.098	0.021	0.937
Fluctuations in non-pain symptoms: R^2 = 0.196; Adj. R^2 = 0.013; p = 0.402				
Stress	0.430	0.281	0.357	0.140
Neuroticism	-0.924	0.647	-0.438	0.168
PCS	0.109	0.264	0.129	0.683

Self-Efficacy	-0.246	0.370	-0.149	0.512
N400	-0.010	0.045	-0.053	0.827

* Indicates a significant result

Note. *The chief effect of interest was the incremental effect of the central N400 on average pain, fluctuations in pain, average mood, fluctuations in mood, average non-pain symptoms and fluctuations in non-pain symptoms. The control variables were stress, neuroticism, pain catastrophising (PCS) and self-efficacy.*

Appendix 6: Results from a regression (N = 60) involving average mood as the outcome, and central N400 for symptom-related nouns, Group and the interaction between Group and central N400 as predictors.

	b	***b SE***	***β***	***p*-value**
Average mood (n400*Group): R^2 = 0.162; Adj. R^2 = 0.147; *p* = 1.694e-06				
N400	0.062	0.031	0.183	0.046*
Group	-0.965	0.308	-0.331	0.002*
N400*Group	-0.020	0.049	-0.050	0.677