THE EFFECT OF PERCEPTUAL TRAINING ON SOMATOSENSORY DISTORTION IN PHYSICAL SYMPTOM REPORTERS

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ABSTRACT

Title: The effect of perceptual training on somatosensory distortions in physical symptom reporters.

Objective: The perceptual mechanisms underlying the development and maintenance of excessive physical symptom reporting (i.e. "somatisation") are poorly understood. Research with non-clinical participants suggests that high and low symptom reporters perform differently when detecting somatosensory signals and have different false alarm rates in which the presence of a signal is incorrectly reported when no signal is present. High symptom reporters often incorrectly report the presence of a signal particularly when a stimulus in a different sensory modality is presented. Previous research has shown that it may be possible to reduce false alarm rates by perceptual training using bi-modal visuo-tactile stimuli pairing. The current was designed to test this hypothesis.

Methods: Seventy non-clinical participants scoring either high or low on the Patient Health Questionnaire (PHQ-15; a measure of somatisation) completed the Somatic Signal Detection Task (SSDT), a novel perceptual paradigm that purports to measure individual differences in somatosensory distortion. Prior to the SSDT, approximately two thirds of the sample completed either a "low" or "high" perceptual training protocol in which a suprathreshold tactile and visual stimuli were paired either infrequently (25%) or frequently (75%), with the intention of training participants to discriminate tactile signal from noise more effectively. The remaining participants received no perceptual training. Factors known to be highly associated with somatisation were controlled for. Negative affectivity was controlled for using the State-Trait Anxiety Inventory Trait Version (STAI-T; Spielberger, Gorsuch & Lushene, 1970), somatosensory amplification was controlled for using the Somatosensory Amplification Scale (SSAS; Barsky, Goodson, Lane & Cleary, 1988), the Patient Health Questionnaire (PHQ-9; Kroenke, Spitzer, & Williams, 2001) was used to control for depression and the Health Anxiety Inventory-Short Version (SHAI; Salkovskis, Rimes, Warwick & Clark, 2002) was used to control for hypochondriacal factors with the Patient Health Questionnaire-Generalised Anxiety Disorder (PHQ-GAD-7; Spitzer, Kroenke, Williams & Löwe, 2006) being used to control for anxiety.

Results: The high PHQ-15 group reported significantly more false alarms and had a significantly higher response criterion than the low PHQ-15 group in the no perceptual training conditions. The perceptual training reduced the false alarm rate for the high PHQ-15 group but did not alter response criterion. Although the findings were in the predicted direction, neither of these findings reached significance. The effect size indicated that this was due to low power.

Conclusions: The findings were suggestive of the effect of perceptual training reducing false alarm rates; however, low power meant that it was impossible to draw firm conclusions. Further research with a larger sample is required.

DECLARATION

No portion of the work referred to in this thesis has been submitted in support of an application for another degree or qualification of this or any other university or other institute of learning.

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CHAPTER ONE: INTRODUCTION

The purpose of this review is to provide an overview of the literature regarding the perceptual mechanisms underlying physical symptom reporting and somatisation. The review will begin by discussing the many different definitions and diagnoses in this area and will then discuss a series of psychological models that explain the development and maintenance of somatisation. The review will conclude with an overview of exploratory studies that have aimed to test these psychological models using analogue paradigms. A description of the current study will then be provided.

Keywords: Functional somatic syndromes; Medically unexplained symptoms; Perception; Signal detection theory; Somatisation; Somatoform disorders.

1. Physical symptom reporting and medically unexplained symptoms

According to the Concise Oxford Dictionary, a symptom is "a perceptible change in the body or its function indicating injury or disease" (Sykes, 1983, p. 1083). Although apparently sensible, this definition misses two important issues: (i) the matter of who decides whether the symptom is indicative of a disease; and (ii) the fact that the patient's belief in the presence of injury or disease may be at odds with the results of medical tests and opinion. Most healthy people perceive changes in their body from time to time which cause them limited distress. In some cases, these changes are perceived as possible signs of disease and a physician is consulted. If no pathology is identified by medical investigation, the expectation is that the patient will be reassured and desist from further help-seeking. Sometimes, however, the symptom persists and the patient remains distressed, often seeking further medical reassurance from specialist services to no avail. A range of terms have been used in the literature to describe symptoms of this sort, including medically unexplained symptoms (Mayou, 1991), functional somatic syndromes (Trimble, 1982) and somatisation symptoms (Kellner, 1985). For clarity, this review will use the term somatisation throughout. Common symptoms of this sort in primary care are fatigue, dizziness and pain as well as gastrointestinal problems and sexual dysfunction (Kroenke & Mangelsdorff, 1989; Kirkwood et al., 1982). Symptoms seen in secondary care can include more dramatic symptoms, such as gait disturbance and seizures.

Research shows that psychological factors underlie somatisation to a large degree. According to Watson and Pennebaker (1989), for example, individuals with high levels of trait negative affectivity or "neuroticism" are more likely to perceive

minor physical symptoms or somatic changes as symptoms that need direct medical treatment. In some cases, symptoms may be somatic symptoms related to diagnosable psychiatric disorders such as anxiety and depression (Kirmayer & Robbins, 1991; Kirmayer &Taillefer, 1997.) The term *presenting somatisation* is used to describe "the predominantly or exclusively somatic presentation of psychiatric disorder, most commonly depression and anxiety" (Kirmayer & Robbins, 1991). This is similar to Lipowski's (1968) definition of somatisation as "a tendency to experience and communicate somatic distress in response to psychological distress and to seek medical help for it" (p. 1358).

Presenting somatisation can be contrasted with the concept of *hypochondriacal somatisation*, developed from a study of clinical illness behaviour (Pilowsky, 1990). It has been proposed that individuals experiencing hypochondriacal somatisation have an amplifying somatic style such that they experience and express high levels of somatic distress when experiencing normal physiological processes or mild physiological changes (Barsky & Wyshak, 1989, 1990). Hypochondriacal somatisation is characterised by illness worry and anxiety.

A third category, which many specialists regard as the definition of "true" medically unexplained symptoms (Brown, 2007), is Kirmayer and Robbins' (1991) concept of *functional somatisation*. According to Kirmayer and Robbins (1991), this category of somatisation is characterised simply by high levels of medically unexplained symptomatology that cannot be explained by anxiety, depression or hypochondriasis. The three definitions do overlap to some degree but Kirmayer and Robbins (1991) propose that the definitions describe different groups of patients. Complaints characterised by functional somatisation have been classified through psychiatric definitions and medical definitions. Therefore, depending on the clinician the patient presents to, the same symptoms will be defined differently and given a different diagnostic label. For example, in medical settings, many different "functional somatic syndromes" have been delineated (See table 1; Kanaan, Lepine, & Wessely, 2007), such as chronic fatigue syndrome (CFS), irritable bowel syndrome (IBS) and fibromyalgia, to name a few. Such medical definitions fall on a continuum of severity and, as such, a patient may be diagnosed with a relatively minor case of IBS, for example, and another may be diagnosed with severe IBS with multiple other functional syndromes.

In contrast, psychiatric diagnoses of functional somatic symptoms are categorised along a continuum of somatisation disorder as defined by Diagnostic and

Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). At the most extreme end of this continuum are somatisation disorders and at the less severe end are somatoform disorders not otherwise specified.

Table 1: Characteristics of common functional somatic syndromes (Kanaan, Lepine, & Wessely, 2007; Brown, 2007).

Speciality	Common unexplained Symptoms	Syndrome label
Gastroenterology	Abdominal pain; Diarrhoea; constipation	Irritable bowel syndrome
Gynaecology	Pelvic pain; Dysmenorrhoea; painful urination	Chronic pelvic pain
Rheumatology	Joint pain; fatigue	Fibromyalgia
Cardiology	Chest pain; palpitations	Atypical chest pain
Infectious diseases	Fatigue; poor concentration	Post-viral fatigue syndrome
Respiratory medicine	Breathlessness; rapid breathing	Hyperventilation syndrome

Due to the number of services the individual may be referred to given the nature of their symptoms, it is possible that they may receive different diagnoses for essentially the same symptoms (Mayou, Kirmayer, Simon, Kroenke, & Sharpe, 2005). These diagnostic overlaps are likely to result in confusion for both the patient and the clinician and may increase the stress associated with the symptoms, potentially leading to increased disability, health resource utilisation and associated psychopathology (Katon, Lin, Von Korff, Russo, Lipscomb, & Bush, 1991). Controversy exists over whether the psychiatric and medical definitions do actually refer to the same thing (Mayou et al., 2005; Rief & Hiller, 1999) or whether there is overlap but to a lesser degree (Brown, 2004).

The current thesis addresses the area of functional somatisation as defined by Kirmayer and Robbins (1991a, 1991b), which includes all of the DSM-IV somatoform

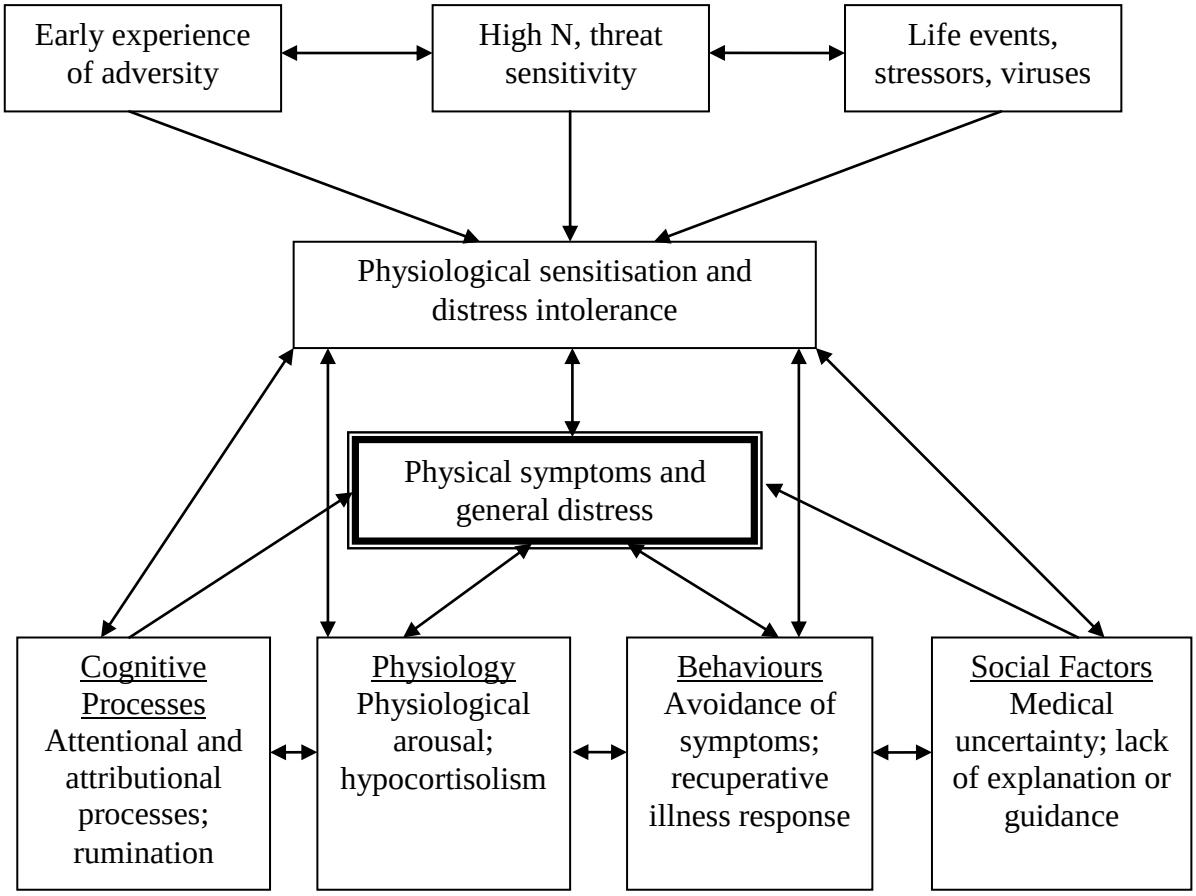
disorders with the exceptions of hypochondriasis and body dysmorphic disorder with functional somatic syndromes also included in the review.

2. Cognitive behavioural and bio-psychosocial theories of somatisation

As a preface to a discussion of perceptual processing in somatisation, a generic framework that is often used to understand these complaints will be described, based on the cognitive behavioural therapy (CBT) models proposed by Deary, Chalder and Sharpe (2007), Kirmayer and Taillefer (1997) and Sharpe, Peveler, & Mayou (1992), which describe similar factors underlying somatisation.

Deary, Chalder and Sharpe (2007) reviewed literature focusing on the predisposing, perpetuating and precipitating factors underlying somatisation in general, and irritable bowel syndrome (IBS) and chronic fatigue syndrome (CFS) more specifically, to develop a contemporary model of somatisation (Figure 1).

Figure 1: Deary, Chalder & Sharpe's (2007) model of somatisation



By this view, symptoms result from a range of predisposing, precipitating and perpetuating factors that interlink to form vicious cycles that maintain symptoms.

2.1. Predisposing factors

Although research in this area is limited, there is some evidence to suggest a genetic factor in the development of somatisation (Kendler et al., 1995; Farmer et al., 1999; Hickie, Kirk, & Martin, 1999). However, it could be argued that learned illness behaviour from previous experiences of illness in self and others also influence the development of somatisation (Sharpe, Peveler & Mayou, 1992; Hotopf, 2003). Attachment style has also been proposed as a significant predisposing factor for the development of somatisation (Noyes, Stuart, & Watson, 2008). The authors suggest that individuals with a preoccupied attachment style tend to seek more medical care and those with an ambivalent or dismissing attachment style are less likely to be compliant with treatment. When faced with a lack of medical reassurance, individuals with dismissing attachment style are at a high risk of developing somatisation.

Watson and Pennebaker (1989) suggest that a neurotic personality trait predisposes an individual to experience "somatopsychic distress". Henningsen, Zimmermann and Sattel (2003) found consistent correlations between somatisation and high levels of anxiety and depression and, neurotic personality traits have been found to relate to anxiety and depression (Matthews, Deary, & Whiteman, 2003), heightened sensitivity to general stressors (Bolger & Schilling, 1991) and increased incidence of physical illness (Huovinen, Kaprio, & Koskenvuo, 2001; Denollet & Van Heck, 2001). Neurotic personality traits have also been found to be associated with somatisation (Kirmayer, Robbins, & Paris, 1994; De Gucht, Fischler, & Heiser, 2004a; De Gucht, Fischler, & Heiser, 2004b; Deary, 2001; Hazlett-Stevens, Craske, Mayer, Chang & Naliboff, 2003). Health anxiety and worry can be de-motivating for a person and have been shown to be increased in patients with functional somatisation (Rief & Broadbent, 2007). It could be argued that individuals experiencing somatisation may feel anger, demoralisation or apathy which impedes on activities in their daily life. They may become focussed on finding a means to alleviate the symptoms such that motivation for any other activities reduces significantly (Young, 2008).

2.2. Precipitating factors

Deary, Chalder and Sharpe (2007) suggest that adverse life events are the main precipitating factors of somatisation discussed in the literature. It has been argued that for some individuals who have experienced trauma, those with psychiatric disorders, individuals with high levels of life stress and individuals whose relatives have reinforced their symptoms and illness behaviours have a higher propensity to experience

somatisation (Kellner, 1991; Ford, 1983; Stuart & Noyes, 1999). In the field of CFS research, there is a large body of research indicating that major life events often occur prior to the onset of CFS (Salit, 1997; Chalder, 1998; Theorell, Blomvkist, Lindh, & Evengard, 1999). Similar findings have also been obtained for chronic pain (Craufurd, Credd, & Jayson, 1990) and IBS (Creed, Craig, & Farmer, 1988).

As discussed above, attachment style developed with parents in early childhood may be carried forward into childhood and adulthood and insecurity-promoting attachment styles may play a part in the development of somatisation (Young, 2008).

Social factors that increase stress levels for the individual play a role in the development of somatisation. A lack of social support and relationships may increase dependency on medical care and levels of stress (Young, 2008).

2.3. Perpetuating factors

The attributions and beliefs the person has about symptoms determine how they are perceived and experienced. Such beliefs stem from personal experiences of illness and from other peoples' responses to illness. Within the cognitive-behavioural model cognitions determine the emotional, behavioural and attentional responses a person may have to their physical sensations (Sharpe, Peveler, & Mayou, 1992) and the outcome. Of particular interest, Deale, Chalder & Wessely (1998) found that negative beliefs about activity were related to poorer outcome in CFS and Lackner (2005) found that similar beliefs in IBS patients led to avoidance behaviours and increased anxiety and symptom maintenance. Furthermore, the more an individual is convinced that their symptoms are pathological and serious, the more intense and prolonged their symptoms become (Barsky & Borus, 1999). A large body of research supports this claim and has found that increased illness attributions and high estimates of symptom vulnerability, together with limited normalising attributions, increase symptom experience and related illness behaviours whilst mixed attributions predict better outcomes (e.g. Sensky, MacLeod & Rigby, 1996; Moss-Morris & Petrie, 2000). It could be argued, therefore, that it is the exclusivity and rigidity of the attributions an individual makes regarding their symptoms rather than just the type of attribution that is made that contributes to the maintenance of the symptom.

Beliefs about illness can also bias recall of past illness experiences (Less-Haley & Brown, 1992). Croyle and Sande (1988) found that informing healthy controls that they had been tested positive for a disease caused them to recall symptoms that were said to be related to the disease and recall more behaviours that were indicated to be risk factors for the illness. It could be argued that this relates to the role of expectations in

somatisation which will be discussed in more detail below in relation to specific perceptual models of somatisation.

Studies have found that it is the way the individual responds to a symptom or illness that is important in the maintenance of symptoms (Deary et al., 2007). In relation to specific functional somatic syndromes, Candy, Chalder, Cleare, Wessely and Hotopf (2004) found that a gradual return to activity rather than rest following glandular fever reduced the chance of developing CFS. However, some behavioural responses to functional somatic symptoms can often be dysfunctional as they maintain the symptoms rather than reducing them (Sharpe, Peveler & Mayou, 1992). Adoption of a "sick role" can amplify symptoms through reduction in activity and increased disability, which causes reinforcement of the symptoms for the individual and strengthening of expectations of future illness and related distress (Young, 2008). Furthermore, people may avoid exacerbating the symptoms (Philips, 1987) by reducing exercise and social activities. In chronic pain, avoidance of activities has been found to be equally predictive of disability as pain itself (Rief & Nanke, 1999). Other "illness behaviours" are seeking medical reassurance and reassurance from family members, which are thought to maintain dysfunction through heightened attention to symptoms and through non confirmation of the patient's illness beliefs. Rief and Broadbent (2007) propose that reassurance seeking can maintain health anxieties through negative reinforcement. Symptom checking may develop as a behavioural response to symptoms which again increases the attention given to the symptoms and alters their perception.

Stress and illness worry are known to exacerbate and maintain physical symptoms, lower the threshold for medical reassurance seeking and heighten the propensity to conclude that ambiguous symptoms are pathological (Kellner, 1986; Rahe & Arthur, 1978). Stress serves to exacerbate symptoms in two ways. First, when an individual is under stress, they are more likely to attribute normal bodily symptoms to disease rather than normal bodily changes as they might otherwise do if they were not experiencing stress. Second, life stressors cause anxiety and depression which also have their own somatic and autonomic factors. For example, anxiety is linked to physiological changes in the body (Sharpe, Peveler & Mayou, 1992) related to an increase in activation of the sympathetic nervous system, reflected in increased heart rate, sweating and shaking, which can be interpreted by the person as further signs of illness (Warwick & Salkovskis, 1990). Anxiety has been found to lower the threshold and tolerance of pain (Sternbach, 1978). Along with depression, anxiety has been found to increase hypervigilance and alter the perception of symptoms as noxious and

worrying (Salovey & Birnbaum, 1989; Larsen, 1992; Kirmayer, Robbins & Paris, 1994). In CFS, illness worry was found to be highly associated with symptom attribution, neuroticism and depression (Taillefer, Kirmayer, Robbins & Lasry, 2003). Depression also produces autonomic symptoms, such as reduced energy, fatigue and pain (Simon, 1990). Negative beliefs about a symptom cause the depressed individual to recall illness-related memories and to perceive their current and future health negatively (Barsky, 1992). Similarly to anxiety, depression influences attention towards to the symptom and increases bodily preoccupation. A cycle then develops in which further emotional distress and physical sensations develop to alter symptom perception. The more the somatic symptoms are perceived as threatening, incapacitating and pathological and the more distress associated with them, the more intense, disabling and chronic they become (Struewing & Gray, 1990; Decoufle, Holgreen, Boyle, & Stroup, 1992).

As discussed, emotional arousal and the development of illness behaviours can increase the amount of attention given to symptoms whereas distraction away from the symptoms can diminish the amount of attention given (Barsky, 1992). The amount of attention given to symptoms can vary greatly (Kirmayer & Taillefer, 1997). Rief, Hiller and Margraf (1998) found that individuals who perceived themselves as more vulnerable to developing illness or the symptom as more dangerous were more likely to attend to the symptoms. Research has also found that decreased attention to CFS symptoms increased the effect of treatment to some degree (Moss-Morris, Wash, Tobin & Baldi, 2005) and that threat sensitivity increased attentional processes which led to increased pain in IBS patients (Lackner, 2005).

It is often difficult to disentangle and isolate the perpetuating variables discussed above as each interacts with one another. Cognitive behavioural models of somatisation tend to agree that all of these variables play a significant role in the development and maintenance of somatisation but perhaps place emphasis on different areas and specific variables. Furthermore, although there is evidence for each of the factors that influence the development and maintenance of somatisation within a CBT framework, the amount of evidence varies considerably (Deary, Chalder, & Sharpe, 2007). Moreover, there is much individual variation in each of these factors.

Whilst these generic CBT models of somatisation provide a useful overview of the interactions between psychological and physiological processes underlying somatisation and the feedback loops that help to maintain the symptoms, they do not provide a detailed account of the processes involved in the actual development of

symptoms. Other models have, however, focused on more specific aspects of the perceptual processes involved in the development of somatisation. These will be discussed below.

3. Perceptual processing models of somatisation

3.1. Barsky's somatosensory amplification model

According to Barsky (1992) people perceive physical sensations differently due to different levels of perceptual sensitivity. By this view, somatosensory "amplifiers" are people who have increased sensitivity and therefore experience bodily sensations as particularly intense, unpleasant and disturbing (Barsky, 1992), which may be a perceptual trait that can be learned from early experiences or have a genetic link. Barsky (1992) also argues that somatosensory amplification can be a transient state in which the same sensation may be experienced differently on different occasions. Three aspects of somatosensory amplification have been identified: firstly, physiological hypervigilance that increases attention to unpleasant sensations; secondly, a tendency to selectively attend to relatively mild sensations; and thirdly, the tendency to attribute these sensations to serious pathology and disease rather than normal physiological changes. In accordance with other cognitive approaches to somatisation, Barsky's (1992) model also assumes that the perception and maintenance of a symptom can be influenced by the belief that the individual has a disease, negative expectations about the prognosis of the disease, the sick role, and stressful events. As such, symptoms can be amplified when they are attributed to a serious pathology rather than a benign cause. Furthermore, situational context influences how a symptom is perceived and how much meaning is placed on the symptom. Attention also influences symptom perception with more symptom-focused attention amplifying the perceived presence and intensity of the symptom. Therefore, anything that may influence the process of amplification, such as anxiety and depression, will alter the perception of symptoms and maintain somatisation.

The cognitive-perceptual style of somatosensory amplification appears to be a common risk factor for many different types of somatic symptoms (Hiller, Cuntz, Rief, & Fichter, 2001). Support for this model has been found in patients with IBS who amplify mild gut dysfunction and are hypersensitive to gut distension, leading to increased pain during medical examinations (Ritchie, 1973). Many studies have used the self-report Somatosensory Amplification Scale (SSAS; Barsky, Wyshak, & Klerman, 1990) to assess individual differences in amplification. Barsky, Orav,

Delamater, Clancy, and Hartley (1998), for example, found that SSAS scores were related to heightened symptom perception for patients with respiratory tract infections. Furthermore, there is evidence to support the claim that amplification is related to depression and anxiety as well as hypochondriacal beliefs (Barsky, Goodson, Lane, & Cleary, 1988). However, there are conflicting opinions regarding whether amplification has an independent effect on symptom perception or whether it is simply mediated by negative affect (Young, 2008).

3.2. Rief and Barsky's perception-filter model of somatoform symptoms

Rief and Barsky (2005) also claim that somatoform disorders are disorders of perception but argue that dysfunctional filtering of bodily signals may be a key component of these complaints, rather than the amplification of those signals as such. Their model proposes that symptoms develop via a three stage process, encompassing the initial generation of sensations and their subsequent selection by the cognitive system. The first two stages of the model occur prior to conscious awareness with the third stage occurring after the symptom is perceived. For the current purposes, only the initial pre-conscious stages and their influence on symptom perception are relevant. During the first stage, sensory signals are constantly being sent from the periphery to the brain, with various factors affecting the number and quality of those signals. During the second stage, the signals are filtered by neural filtering processes and selected for conscious attention. For healthy people most of these sensory signals are filtered out; for people experiencing somatoform symptoms, however, the signals are not adequately filtered and irrelevant sensations enter conscious awareness. By this view, factors such as maladaptive health beliefs, anxiety, depression and attentional dysfunction cause the filter system to become dysfunctional, thereby increasing the likelihood of symptom perception. Another important factor in this model is habituation. Habituation is described as a decrease in physiological activity through repeated presentation of signals (Rief and Auer, 2001) and is a process that is ordinarily expected in healthy individuals. However, for people with somatisation, amplification of symptoms combined with uncertainty regarding the origin of the symptoms hinders this habituation process and maintains symptom perception (Rief & Auer, 2001; Rief & Barsky, 2005).

3.3 Sensitisation

Deary, Chalder and Sharpe (2007) suggest that sensitisation is an underlying perceptual mechanism of somatisation. Sensitisation is the tendency to experience an

increased response to stimuli over time due to prior exposure to those stimuli. Johnson (2008) describes sensitisation as taking place in both the peripheral and central nervous system and that it has both biological and psychological components. Rygh, Svensden, Fiska, Haugan, Hole and Tjolsen (2005) found that long term potentiation, a sensitisation mechanism, reduced the perceptual threshold for future pain stimulation when it had been induced in pain pathways by prior experience of pain stimuli. Deary et al. (2007) drew on this work to further explain the effect of long term potentiation on neural thresholds and symptom perception. Through sensitisation, sensory receptor sites come to fire more readily and almost automatically without any additional input beyond peripheral noise. The central processes in sensitisation include neural pathways forming tight circuits that also fire more readily and incorporates a large network of associated connections which amplifies the impact of sensitisation. The way in which perception of symptoms is altered is due to the feedback from altered proprioceptive and kinaesthetic sensations, which serves to heighten the activity within the peripheral and central neural networks in which they are integrated. The increase in signal activity within the peripheral and central neural networks would interact with other perpetuating factors discussed above to alter symptom perception and thereby amplify symptoms. Once the sensitisation process is established, an individual has an increased propensity to generalise bodily complaints, thereby increasing stress levels (Young, 2008).

3.4 Brown's integrative conceptual model of somatisation

Brown's (2004) model draws from cognitive psychology and previous models of somatisation to explain the role of perception and cognition in somatisation. The model proposes that medically unexplained symptoms are alterations in symptom perception generated by information (broadly speaking, memories) stored in the cognitive system. Brown (2004) uses the term "rogue representations" to refer to this information and suggests that it can be acquired from various different sources: 1) direct exposure to physical states in oneself; 2) indirect exposure to physical states in others; 3) sociocultural transmission; and 4) verbal suggestion.

Brown (2004) proposes that somatisation occurs when the activation levels of these rogue representations becomes high enough for them to be selected by attentional systems as current illness experiences, creating a distortion in body perception. In this account, symptoms are maintained through repeated re-activation of these rogue representations via the perpetuating factors identified in other models.

Brown's (2004) model links both perceptual and memory processes to the experience of somatisation. Consistent with this, there is some evidence that there is memory bias and

inaccurate recollection of bodily symptom related information. Such memories influence the expectations related to future symptoms and therefore alter symptom perception (e.g. Bayer, Coverdale, Chiang, & Bangs, 1997; Rief and Broadbent, 2007; Van Damme, Crombez and Eccleston, 2004). Lim and Kim (2005) found evidence for a memory bias for physical threat words in explicit but not implicit memory tasks. In addition, Rief, Heitmüller, Reisberg and Rüddel (2006) found that patients with somatisation syndrome were more likely to remember the probabilities linked to health-related information rather than the information itself. Another factor to consider in this model is the effect of future expectations and suggestibility on symptom perception. Perception is guided by our expectations of what we will experience next. Therefore, suggestion can alter perception by changing our expectations of what we will experience in the future. Research on placebo effects (Colloca, Sigaudo, & Benedetti, 2008; Price, Finniss, & Benedetti, 2008) demonstrates the role of expectancy on perception. Patients provided with a placebo have expectations of the future responses (Kirsch, 1985) and respond accordingly. Vase, Robinson, Verne and Price (2003) demonstrated this effect in patients with irritable bowel syndrome. Patients were administered a painful stimulus under two conditions: local anaesthetic and placebo. In the first study, participants were told they would receive an active or a placebo agent and in the second study they were told they would receive the agent that is known to reduce pain in some patients. Larger placebo effects were observed in the second study which had more definitive instructions.

Central to the Brown (2004) model is the idea that somatic symptoms are distortions of body representation, influenced by top-down factors. There are numerous examples of how the body image can be distorted. In the rubber hand illusion, for example, stroking someone's hand whilst simultaneously stroking a rubber hand in their line of vision can lead to them experiencing the rubber hand as part of their body (Botvinick & Cohen, 1998). Research suggests that both top-down and bottom-up factors influence experience of the illusion. For example, the illusion does not work when the rubber hand and the real hand are stroked consecutively rather than simultaneously (demonstrating the influence of bottom-up factors) or when another object other than a hand is used or if the rubber hand is in an unusual position (demonstrating the influence of top-down factors; Tsakiris & Haggard, 2005). Miles, Poliakoff and Brown (in press) investigated the effects of the rubber hand illusion on high and low symptom reporters. The authors proposed that if responsiveness to such bodily illusions represents individual differences to everyday bodily experiences and

perceptions then bodily illusions may be a more objective way to investigate perceptual distortions in clinical populations. The study found that the high symptom reporters responded more highly to the rubber hand illusion than the low symptom reporters. Brown (2004) proposes that somatisation occurs due to over reliance on top-down cognitive factors during the processing of bodily representations. The rubber hand illusion essentially "tricks" this top-down process by providing discrepant sensory information which distorts perception. The study therefore found that high symptom reporters are more susceptible to this than low symptom reporters (Miles, Poliakoff, & Brown, in press).

4. Analogue paradigms to test the perceptual mechanisms of somatisation

Perceptual illusions provide one way in which perceptual mechanisms underlying somatisation can be investigated. The remainder of this review will discuss an alternative method, using a novel paradigm (Lloyd, Mason, Brown and Poliakoff, 2008) based on signal detection theory, to specifically investigate perceptual mechanisms discussed by Brown (2004). The discussion will begin with an overview of signal detection theory before discussing the study conducted by Lloyd et al. (2008).

Signal detection theory provides a mathematical analysis of an individual's signal sensitivity and response bias (Green & Swets, 1966; Harvey, 1992; McNicol, 1972). According to signal detection theory, the detection of a stimulus involves a decision-making process in which an individual's perceptual sensitivity, the nature of the stimulus itself and other cognitive factors influence the decision of whether or not a stimulus is perceived as being present or absent. McNicol (1972) describes sensitivity as the tendency to correctly detect the presence or absence of a signal. Sensitivity varies according to the stimulus' probability of occurrence, intensity and imminence (McNicol, 1972). As such, individuals perform differently when detecting signals and this may be influenced by factors other than the sensitivity of sense receptors. The theory also analyses response bias or the extent to which one response is favoured over another (Harvey, 1992). Response bias can be influenced by beliefs held by the individual about the stimulus and the goals the individual has when making that response (Green & Swets, 1966), particularly in relation to the severity and consequence of the false alarm response. Signal detection theory provides methods that allow an individual's level of signal sensitivity and response bias to be analysed separately. Signal detection theory proposes that individuals make decisions regarding the presence or absence of a stimulus based on information provided by two distributions and that neurones are constantly sending this information to the brain. However, the ratio of

signal to “noise” (irrelevant internal or external information) is constantly oscillating, introducing a degree of ambiguity to the decision-making process. The first distribution; the signal absent distribution, relates to the situation that only a background level of noise is present and the actual signal is absent. Therefore, in the case of the tactile sensory modality, for example, if the tactile stimulus is absent any perception of a tactile stimulus would be incorrect and the decision would have been guided solely by the background “noise” information provided to the brain. In contrast, the signal present distribution represents an experience in which there is an increase in the noise level due to the presence of a signal.

During signal detection tasks, series of trials are presented in which a stimulus may be present or not and after each trial, the participant must report whether they perceived a signal or not. The theory proposes that during each trial, the participant will perceive a certain degree of the signal; however, the participant must decide whether their perception is due to the presence of the signal or merely due to noise. Reponses to these trials are categorised as “hits” (a correct “yes” response to the presence of a signal), misses (an incorrect “no” response to the presence of a signal), false alarms (an incorrect “yes” response to the presence of a signal when the signal is absent), and correct rejections (a correct “no” response to the absence of a signal when the signal is absent). From these responses, the theory proposes that it is possible to measure how likely an individual is to report the presence of a signal even if the signal is absent. The theory assumes that each individual has a criterion level when attempting to detect a signal which can be considered a measure of the readiness to respond that the signal is present in an ambiguous situation. If a high stimulus is presented, it is argued that the signal strength is higher than the criterion level and the stimulus is reported as being present. Likewise, if no stimulus is presented or it is low, the signal strength is said to be below the criterion level and is reported as being absent. Therefore, the theory purports that the level of the criterion value determines how many hits and false alarms are reported and thereby altering the criterion level changes the proportions of such responses being made. An individual’s perceptual sensitivity (d prime; *d*’) to the signal can also be measured. This is a measure of an individual’s ability to discriminate between signal and noise on Signal Present and Signal Absent trials. Finally, a measure of response bias (*c*) can be assessed. The response biascan be described as the tendency to favour one response above another, i.e. signal present over signal absent. If more hits and false alarms are reported then a lower criterion level and a reduced bias towards reporting “yes” to the presence of a signal is observed with the *c* value being negative.

Likewise, if fewer hits and false alarms are reported then there is an increase in criterion level, a positive c value is observed and therefore a increase in the tendency to say "no" to the presence of a signal.

Johnson, Burton and Ro (2006) used signal detection theory to investigate the effect of presenting bi-modal visuo-tactile stimuli on the detection and response criteria of the tactile stimulus. In their study, Johnson et al. (2006) presented participants with four randomly ordered trial types: (i) a visual stimulus (a short flash on an LED light positioned above the participant's finger, (ii) a tactile stimulus (a short electrical vibration delivered to the finger), (iii) the visual stimulus followed sequentially by the tactile stimulus, or (iv) no stimulus. After each trial, participants were required to report whether they had felt the tactile stimulus or not. Johnson et al. (2006) used this method to investigate the effect of visual stimuli on tactile stimuli and found that participants were more likely to detect a near perceptual threshold tactile stimulus when it was presented simultaneously with a supra-threshold visual stimulus (light) compared to when the tactile stimulus was presented alone. Furthermore, participants were more likely to report a touch in the absence of the tactile stimulation (i.e. a "false alarm") when the visual stimulus was present. Lovelace, Stein and Wallace (2003) used the signal detection task with auditory stimuli together with the non-informative light and found that the presence of the light increased participants' auditory sensitivity. Both Johnson et al. (2006) and Lovelace et al. (2003) found that when the non-informative light was presented in the absence of the signal, participants continued to make some reports of signal perception. These studies demonstrate that perceptual sensitivity can be increased in one sensory modality when stimulation in another parallel sensory modality is presented. Furthermore, when the non-informative light is presented, the propensity to report the presence of the signal in its absence is increased.

Lloyd, Mason, Brown and Poliakoff (2008) argued that these false alarm responses on the task are similar to somatisation symptoms and might involve comparable mechanisms, suggesting that a paradigm based on this approach could be used to test the perceptual distortion processes described in the Brown (2004) model. With this in mind, Lloyd et al. (2008) developed an experimental paradigm (the Somatic Signal Detection Task; SSDT) with a view to measuring individual differences in tactile sensitivity and response bias in healthy control participants with the aim of validating an laboratory analogue paradigm to test patients with somatisation. The authors propose that the presence of the non-informative light at the same time as the presence of the

signal (i.e. tactile stimulation) during trials increases the activation of the memory of the tactile stimulation and therefore increases the number of false alarms reported.

McKenzie, Poliakoff and Lloyd (2010) investigated the reliability of illusory touch reports on the SSDT over time and whether there are robust individual differences in the tendency to report illusory touch experiences (i.e. false alarms) on the task. Their findings replicated those of previous studies using the SSDT (Brown et al., in press; Johnson et al 2006; Lloyd et al 2008; Mirams et al 2010) showing that presenting bi-modal visuo-tactile stimuli enhances the detection of the near perceptual threshold tactile stimulus. Furthermore, the study found that the tendency to report false alarms was stable over time (in spite of variation in tactile sensitivity), indicating that the tendency to experience illusory touch has a trait-like component (McKenzie et al., 2010) that is reliably captured using the SSDT.Further studies using the paradigm have identified that false alarm rate on the SSDT is increased for individuals who score highly on symptom report measures. Brown, Brunt, Poliakoff, & Lloyd (2010) distinguished between participants who had high or low scores on the somatoform dissociation questionnaire (a proxy measure of somatoform symptoms) and found that high symptom reporters exhibited more false alarms and a lower response bias than low symptom reporters. These findings remained when controlling for depression, negative affect and somatosensory amplification which are known to be associated with somatisation (Brown et al., 2010; Brown, Skehan, Chapman, Perry, McKenzie, Lloyd, Babbs, Paine & Poliakoff, submitted).

McKenzie, Lloyd, Brown, Plummer and Poliakoff (submitted) have recently investigated the influence of prior knowledge of visuo-tactile bimodal stimuli on the number of false touch responses in light-present trials on the SSDT. In their initial experiment, a similar method to Johnson et al. (2006) was used. The authors found that participants reported more false alarms in light-present trials despite having no prior experience of the experimental visuo-tactile pairing. It was suggested that this finding was a consequence of participants having an existing association between simultaneous visual and tactile stimuli, developed from everyday multi-sensory experiences.

In a second study, McKenzie et al. (submitted) varied the reliability of the light as a predictor of the tactile stimulus during a training phase prior to the SSDT, with the prediction that this would influence the frequency of false touch reports on the task in light-present trials. During the training, the light was paired with a supra-threshold tactile stimulus either frequently (i.e. 75% of trials; high association group) or infrequently (i.e. 25% of trials; low association group). It was predicted that participants

in the high association group would make more false alarms in the light-present trials than participants in the low association group and a no training control group. Contrary to expectation, however, the high association group exhibited a similar false alarm rate to the no training controls, while the low association group actually experienced fewer false alarms than the control group in both light-present and light-absent trials. This seems to provide further support for the notion that the effect of light is reliant upon a life-long association established from everyday experiences where visual and tactile stimuli occur in close proximity (Johnson et al., 2006). The results of McKenzie et al.'s (submitted) study suggested that the effect of the manipulation was probably less about the perceived contingency between the light and touch, and more about giving people practice at identifying the presence of a touch either in more (i.e. multisensory) or less ambiguous (i.e. unisensory) conditions. McKenzie et al (submitted) proposed that participants in the low association group became more "stimulus driven" in their perceptual decision-making following the training and therefore relied less on top-down information from the visual stimulus and their expectations. The current study therefore predicted that the low perceptual training would reduce the number of the number of false alarms.

5. The current study

The current study aims to further investigate the effects of perceptual training on the experience of somatosensory distortion (McKenzie et al., submitted) and the possibility that these effects may be different for high and low symptom reporters. This would provide further evaluation of the link between symptom reporting and false alarm rates at baseline and further investigate underlying perceptual mechanisms presented in Brown's (2004) model. As Brown's (2004) model is based on the idea that regular cognitive processes occur in a dysfunctional manner to distort symptom perception rather than on pathology per se, it is appropriate to carry out the study's aims using a non-clinical population. As a non-clinical sample was to be recruited, it was necessary to identify individuals with a tendency to experience somatic symptoms. The Patient Health Questionnaire (PHQ-15; Kroenke, Spitzer, & Williams, 2002) was used as it is a well validated measure of generic somatic symptoms (Kroenke et al., 2002), such as, headaches, stomach pain, dizziness etc. and measures total number of symptoms as well as symptom severity.

Students from the University of Manchester were invited to complete the PHQ-15 online as well as measures of somatosensory amplification and health anxiety. Individuals identified as either high or low symptom reporters on the PHQ-15 were then

invited to participate in an experimental session. Those who consented to take part completed measures of depression, generalised anxiety and negative affectivity and were randomly assigned to one of three perceptual training groups (i) no training, (ii) low training, or (iii) high training. The training conditions were the same as those used by McKenzie et al. (submitted) with the light paired with a supra-threshold tactile stimulus either frequently (i.e. 75% of trials; high association group) or infrequently (i.e. 25% of trials; low association group). After having their perceptual threshold established and undergoing the training conditions where appropriate, the participants performed the SSDT. Somatosensory amplification, health anxiety, generalised anxiety, depression and negative affectivity were used as covariates to control for and eliminate the influences of these factors known to be associated with somatisation. The following research questions and hypotheses were addressed.

Research question 1: Are there baseline differences between high and low symptom reporters in terms of false alarm rate and response criterion?
Hypothesis 1: The current study will replicate the findings of Brown et al (2010) of a difference between high and low symptom reporters in terms of false alarms and response criterion at baseline (i.e., no perceptual training condition).

Research question 2: Can any such baseline differences be reduced through perceptual training?
Hypothesis 2: The current study predicts an interaction between symptom reporting and perceptual training such that there would be a significant difference in false alarms and response criterion between the PHQ-15 groups in the baseline condition but not in the training conditions.

The current study will also aim to replicate previous findings with the SSDT in terms of the effect of the light and the overall effect of perceptual training (regardless of PHQ-15 group), with a view to establishing the reliability of the paradigm in this study.

CHAPTER TWO

The following chapter consists of a paper written for submission to the Journal of Psychosomatic Research according to journal guidelines (Appendix I).

The effect of perceptual training on somatosensory distortion in physical symptom reporters

Abstract

Objective: The perceptual mechanisms underlying the development and maintenance of excessive physical symptom reporting (i.e. "somatisation") are poorly understood. Research with non-clinical participants suggests that high and low symptom reporters perform differently when detecting somatosensory signals and have different false alarm rates in which the presence of a signal is incorrectly reported when no signal is present. High symptom reporters often incorrectly report the presence of a signal particularly when a stimulus in a different sensory modality is presented. Previous research has shown that it may be possible to reduce false alarm rates by perceptual training using bi-modal visuo-tactile stimuli pairing. The current study was designed to test this hypothesis.

Methods: Seventy non-clinical participants scoring either high or low on the Patient Health Questionnaire (PHQ-15; a measure of somatisation) completed the Somatic Signal Detection Task (SSDT), a novel perceptual paradigm that purports to measure individual differences in somatosensory distortion. Prior to the SSDT, two thirds of the sample completed either a "low" or "high" perceptual training protocol in which suprathreshold tactile and visual stimuli were paired either infrequently (25%) or frequently (75%), with the intention of training participants to discriminate tactile signal from noise more effectively. The remaining participants received no perceptual training.

Results: The high PHQ-15 group reported more false alarms and had a higher response criterion than the low PHQ-15 group in the no perceptual training conditions. The

perceptual training reduced the false alarm rate for the high PHQ-15 group but did not alter response criterion. Although the findings were in the predicted direction, neither of these findings reached significance. The effect size indicated that this was due to low power.

Conclusions: The findings were suggestive of the effect of perceptual training reducing false alarm rates; however, low power meant that it was impossible to draw firm conclusions. Further research with a larger sample is required.

Keywords: Functional somatic syndromes; Medically unexplained symptoms; Perception; Signal detection theory; Somatisation; Somatoform disorders.

Introduction

Excessive physical symptom reporting or *somatisation* is a growing problem for health services and the economy [1]. Research shows that the more physical symptoms reported by an individual, the greater their level of distress, disability and medical resource utilisation [1-3]. For a large proportion of patients who report high numbers of physical symptoms, a medical explanation for those symptoms cannot be found [4-6]. Psychological factors are thought to be central to excessive symptom reporting and psychological interventions using cognitive-behavioural therapy [7] and psychodynamic psychotherapy have shown limited effectiveness in reducing the impact of this phenomenon [8,9]; however, a clear understanding of the specific psychological processes involved in somatisation remains lacking [10].

A recent integrative model [10] proposes that excessive distortions in symptom perception can develop through the over-activation of illness information stored in memory, that intrude into awareness as current perceptions. The model proposes that there may be basic perceptual differences between individuals that influence how susceptible they are to these somatosensory distortions.

A novel paradigm, the Somatic Signal Detection Task [SSDT; 11], was designed to induce and measure tactile sensitivity change and response bias in health control participants with the aim of developing a paradigm that can test such changes in somatic symptoms and to specifically investigate perceptual mechanisms discussed by Brown [10]. Lloyd, Mason, Brown and Poliakoff [11] proposed that the false alarms (i.e. reports of the presence of a tactile stimulation when no such stimulation is presented) demonstrate the same mechanisms proposed by Brown [10] and are therefore an example of somatosensory distortion. Lloyd et al. [11] argued that these false alarm responses on the task are similar to somatisation symptoms and might involve comparable mechanisms, suggesting that a paradigm based on this approach could be used to test the perceptual distortion processes described in the Brown [10] model. McKenzie, Poliakoff, Brown and Lloyd [12] investigated the reliability of using the SSDT as a paradigm to test illusory tactile sensations in a laboratory. Their findings replicated those of previous studies using the SSDT [11,13,14,16] showing that presenting bi-modal visuo-tactile stimuli enhances the detection of the near perceptual threshold tactile stimulus. Furthermore, the study found that the tendency to report false alarms or illusory touch experiences was stable over time and has a trait-like component [12] suggesting that the measurement of reported false alarms and response criterion on the SSDT are robust phenomena and a reliable way of testing signal perception in the laboratory.

It has been shown that false alarm rate on the SSDT is increased for individuals who score highly on symptom report measures, and that this effect remains when controlling for self-reported depression, negative affect and somatosensory amplification, which are known to be associated with somatisation [13,14].

McKenzie, Lloyd, Brown, Plummer and Poliakoff [15] have recently investigated the influence of prior knowledge of visuo-tactile bimodal stimuli, stored in memory, on the number of false touch responses in light-present trials on the SSDT.

The authors investigated the effect of the level of association between the visual and the tactile stimuli on the light-evoked false alarms. The authors propose that the presence of the non-informative light at the same time as the presence of the signal (i.e. tactile stimulation) during trials increases the activation of the memory of the tactile stimulation and therefore increases the number of false alarms reported. A training task was used prior to the SSDT to vary the reliability of the light as a predictor of the tactile stimulus and thereby strengthen the association between the light and the tactile stimuli. According to the Brown model [10] these associations would be stored in memory and overactivated during the SSDT as misrepresentations of the tactile stimulus which would predict that the false alarm rate would increase as a consequence. During the training, the light was paired with a supra-threshold tactile stimulus either frequently (i.e. 75% of trials; high association group) or infrequently (i.e. 25% of trials; low association group)[15]. Contrary to prediction, the authors found that the high association group did not experience more false alarms than a no training control group, but that the low association group experienced fewer false alarms than the controls. The findings therefore were not explained by the memorial factors described in Brown [10] but that there was a training effect in which participants' ability to discriminate signal (tactile stimulation) from noise (visual stimulation) through a forced decision making process was increased. It appears that this study raises the possibility that perceptual training might be able to reduce the tendency to experience somatosensory distortion and thereby symptom experience.

The current study was designed to further investigate the effects of perceptual training on the experience of somatosensory distortions [15] and the possibility that training may have different effects for high and low symptom reporters. The same perceptual training conditions were used as the McKenzie et al. [15] study in which the level of association between light and touch stimuli was varied. The results of that study suggested that the effect of the manipulation was probably less about the perceived

contingency between the light and touch, and more about giving people practice at identifying the presence of a touch either in more (i.e. multisensory) or less ambiguous (i.e. unisensory) conditions. The current study therefore predicted that the low perceptual training would reduce the number of false alarms. McKenzie et al [15] proposed that participants in the low association group became more "stimulus driven" in their perceptual decision-making following the training and therefore relied less on top-down information from the visual stimulus and their expectations. In the current study, therefore, the training protocol aimed to make participants more stimulus driven and that the training would have a greater effect on the high PHQ-15 group because they are less stimulus driven to begin with.

As Brown's [10] model is based on the idea that regular cognitive processes occur in a dysfunctional manner to distort symptom perception rather than on pathology per se it is appropriate to carry out the study's aims using a non-clinical population. High and low symptom reporters were identified using the Patient Health Questionnaire [PHQ-15; 17] The effects of perceptual training were assessed using the same training procedures as in McKenze et al. [15] prior to the SSDT, however, a no perceptual training condition was added to the current study.

The current study therefore aims to further investigate any baseline differences between high and low symptom reporters on the SSDT and predicts that high symptom reporters will report more false alarms than low symptom reporters [18]. The main aim of the study is to further investigate whether it is possible to reduce any such differences through perceptual training [15]. The current study predicts an interaction between symptom reporting and perceptual training such that there would be a significant difference in false alarms and response criterion between the PHQ-15 groups in the baseline condition but not in the training conditions.

The current study will also aim to replicate previous findings with the SSDT in terms of the effect of the light and the overall effect of perceptual training (regardless of PHQ-15 group), with a view to establishing the reliability of the paradigm in this study.

Method

Design

Ethical approval was obtained from the local ethics committee. A 3 x 2 x 2 mixed design was used with PHQ-15group (low vs. high) and condition (no training vs. low perceptual training vs. high perceptual training) as between-subjects factors and light (absent vs. present) as a within-subjects factor. The primary dependent variable was false alarm rate on the SSDT; the secondary dependent variable was response criterion on the task.

Participants initially completed an online battery of questionnaires including the PHQ-15 [17] and the Somatosensory Amplification Scale [SSAS; 19]. Participants who scored in the specified range on the PHQ-15, as well as demonstrating right handedness on the Edinburgh Handedness Inventory [EHI; 20], were invited to participate in the experimental phase of the study. Participants within each PHQ-15 group were randomly allocated to one of the three perceptual training conditions prior to arrival at the testing session. The session began with participants completing a consent form, followed by the measures discussed below. Participants in the no-training group completed the thresholding procedure followed by the SSDT. Participants in the low and high perceptual training groups completed the relevant training phase, followed by the thresholding procedure then the SSDT proper. The low and high perceptual training conditions were designed to investigate the effect of the level of intensity between the pairings of the visual and tactile stimuli on false alarm rate. The experiment lasted 30-45 minutes depending on group allocation and the length of time required for thresholding.

Participants

The initial sample consisted of 256 University students and staff (209 female, 47 male; mean age = 21.4 years; S.D = 4.57 years) who responded to poster or email invitations and provided informed consent to participate. Of this sample, right-handed individuals scoring either above 10 (i.e. high symptom reporters; n=66) or below 5 on the PHQ-15 (i.e. low symptom reporters; n=92) were deemed eligible for the study and approached about participating in the experimental phase. As a score of ≥15 on the PHQ-15 is regarded as indicative of clinically significant levels of symptom reporting [17], a score of ≥10 was used here to identify an analogue sample of high symptom reporters. Recent research suggests that such a score has both sensitivity and specificity as a screening cut-off in primary care settings [21]. A score of ≤5 was taken as indicative of low symptom reporting, following Kroenke, Spitzer and Williams [17].

Eighty one participants (48 low PHQ-15, 33 high PHQ-15) took part in the experimental phase and received £5 or course credits. Eleven participants did not achieve a perceptual threshold between 40-60% and therefore did not complete the remainder of the experiment. Seventy participants (39 low PHQ-15, 31 high PHQ-15) completed all stages of the experiment and were included in the final analysis.

Questionnaire measures

Patient Health Questionnaire-15 [17]. The PHQ-15 was used to measure physical symptom reporting, operationalised as how often the individual has felt distressed by 15 common somatic symptoms in the past 4 weeks. The symptoms account for 90% of physical symptoms presented in outpatient settings [17], as well as 14 of the 15 most prevalent DSM-IV somatisation disorder symptoms [22]. Participants are asked to rate how much they have been bothered by each symptom on a scale from 0 ("not bothered at all") to 2 ("bothered a lot"). Total scores range from 0 to 30. Scale reliability (Cronbach's α = .86) was comparable to the original validation report [α = .80; 17].

Somatosensory Amplification Scale [SSAS; 19]. The SSAS was used to control for individual differences in the tendency to experience somatic sensations as unpleasant and to identify them as symptoms of illness. The SSAS asks respondents to rate the degree to which ten statements about unpleasant bodily sensations relate to them in general, on a scale from one ("not at all true") to five ("extremely true"). Total scores range from 10 to 50. Scale reliability for the current study (α = .84) was superior to the original report [α = 0.70; 19].

State-Trait Anxiety Inventory Trait Version [STAI-T; 23]. Negative affectivity was assessed using the STAI-T to control for the correlation between trait anxiety and physical symptom reporting [24]. Participants are asked to rate 20 statements about their experience of cognitive and affective components of anxiety using a Likert scale ranging from one ("almost never") to four ("almost always"). Total scores range from 20-80. The STAI-T has been validated with students [23,25,26]. The scale validity (α = .85) was comparable to the original study [α = .89 – .92; 24].

Patient Health Questionnaire-Generalised Anxiety Disorder [27]. The PHQ-GAD-7 was used to control for individual differences in anxiety symptoms. Participants are required to rate the frequency of seven aspects of generalized anxiety on a scale ranging from 0 ("not at all") to 3 ("nearly every day") over a two week period. Total score range from 0 to 21. The PHQ-GAD-7 predicts diagnoses of generalised anxiety disorder with accuracy [28]. The current study showed good internal reliability for the measure (α = .83).

Patient Health Questionnaire [PHQ-9; 29]. The PHQ-9 is a 9 item self report for measuring depression. Depression was measured as a covariate due to the links between depression and somatoform disorders [30,31]. Participants are asked to rate the frequency of nine aspects of depression on a scale ranging from 0 ("not at all") to 3 ("nearly every day") over a two week period. Total scores range from 0 to 27. The scale

validity for the PHQ-9 (α = .82) was comparable to the original study [α = 0.86-0.89; 29].

Health Anxiety Inventory-Short Version [SHAI; 32]. The SHAI is an 18 item self-report measure used to control for individual differences in hypochondriacal anxiety. The participant is presented with 18 sets of four items and asked to identify which of these they agree with most highly in the past six months; statements are allocated a score of 0-3, with higher score demonstrating higher levels of health anxiety. Scores range from 0 to 54. The scale validity of the SHAI for the current study (α = .83) was comparable to the original validation report [α = 0.89; 32].

Somatosensory Signal Detection Task

Participants sat in a light attenuated room approximately 50 cm in front of the stimulus apparatus, comprising a polystyrene block mounted with a 4-mm diameter red light emitting diode (LED) and a 1.6 x 2.4cm vibrating bone conductor. The participant's left index finger was fixed to the surface of the bone conductor using a double sided adhesive strip. Vibrations were generated through a square wave generator and sent to the bone conductor, controlled by E-Prime software [33]. Instructions were presented to participants on a computer monitor located approximately 10cm behind the stimulus apparatus. A visual cue of a green arrow pointing directly to the participants' finger and indicating the beginning of each trial was presented on the screen. Participants listened to white noise through headphones to prevent them hearing external noise or the vibrations themselves. Participants used their right hand on the computer keyboard to respond "yes" or "no" on the thresholding procedure and "definitely yes", "maybe yes", "definitely no" or "maybe no" on the experimental task, with key order counterbalanced between participants.

Perceptual Training Procedure

During the perceptual training phase the light was paired with a supra-threshold tactile stimulus on a varying number of trials. Supra-threshold tactile stimuli were three

times more intense than the tactile stimulus used at the beginning of the threshold procedure. In the low perceptual training condition, the light and tactile stimuli were paired infrequently (25% of trials) and for the high perceptual training condition, the stimuli were paired frequently (75% of trials)(Table 1). The perceptual training protocol aimed to train participants to discriminate the vibration (i.e. signal) from noise (i.e. light).

The training groups differed only by the number of light only trials they were presented with. Both groups were presented with the same number of vibrations during this phase of the study. Participants were instructed to report when they did *not* feel a vibration on each trial, so as to make the task different to that in the SSDT. Feedback concerning accuracy was provided to the participant on the screen after each trial.

Participants in the no-training condition did not receive any training and began the experiment with the thresholding procedure.

Table 1: Number of trials in each perceptual training condition for one block of trials.

Light	Touch	Low perceptual training	High perceptual training
Absent	Absent	10	30
Absent	Present	30	10
Present	Absent	30	10
Present	Present	10	30

Thresholding procedure

Before commencing the SSDT, each participant's perceptual threshold was assessed using a staircase procedure [34] by the end of which they reported feeling the tactile stimulus in 40-60% of trials. Each participant was presented with an initial block of trials consisting of 10 vibration-present trials and 3 vibration-absent trials, which

were randomly presented to minimise guessing. The blocks of trials were repeated until performance was in the 40-60% range for three consecutive blocks.

Each trial began with a 250-ms visual cue of a green arrow pointing to the participant's finger, followed by a 1020-ms stimulus period. During vibration-present trials, a 20-ms vibration was delivered with a 500-ms empty window pre and post vibration; during vibration-absent trials, an empty period of 1020-ms was presented. After each trial a 500-ms on-screen message prompted participants to report whether they had perceived a vibration or not by responding "Y" (for yes) or "N" (for no) on the keyboard. Depending on the participant's responses, the intensity of the tactile stimulus was adjusted by the experimenter at the end of each block of trials.

SSDT proper

The SSDT consisted of two blocks of 80 trials with four trial types: vibration only; vibration with light; light only; no stimulus. Each trial type was randomly presented 20 times in each block. The vibration was presented at the participant's perceptual threshold, as determined during the thresholding procedure. Each trial began with a 250-ms visual cue of a green arrow pointing to the participant's finger followed by a 1020-ms stimulus period. During vibration only trials, a 20-ms vibration was delivered with a 500-ms empty window pre- and post-vibration. On vibration with light trials, the 20-ms vibration was presented at the same time as a 20-ms illumination of the LED. During light only trials, the LED was illuminated for 20-ms in the middle of the trial interval but no vibration was presented. On no stimulus trials, an empty interval of 1020-ms was presented.

In order to maximise the number of false alarms on the task, participants were asked to report the certainty with which they perceived the presence of a vibration on each trial: "yes definitely"; "yes maybe"; "no definitely"; "no maybe". These categories were collapsed into "yes" and "no" as response confidence levels were not measured in

this study. No further instructions were given and participants were naive as to the true purpose of the study.

Statistical analysis of the SSDT data

Following the SSDT, responses were classified as hits (a correct "yes" response to vibration present trials), misses (an incorrect "no" response to vibration present trials), false alarms (an incorrect "yes" response to vibration present trials), and correct rejections (a correct "no" response to vibration absent trials). The signal detection statistic *d'* (z(hit)-z(FA)) and *c* (-.5[z(hit)+z(FA)]) were calculated as indices of perceptual sensitivity and response bias (i.e. the tendency to report stimuli as present) respectively. The higher the value of c, the more conservative the response criterion. A score less than 0 means the participant is more likely to say 'yes' than 'no'; a score of more than 0 means they are more likely to say 'no' than 'yes'.

Outlier data was measured as data falling in the top 5% and bottom 5% of the distribution which would identify any extreme scores [35]. No outliers were identified. However, false alarm rate (light-absent condition: Shapiro-Wilk W=.833, p<.05; light-present condition: Shapiro-Wilk W=.817, p<.05) and PHQ-9 scores were not normally distributed (Shapiro-Wilk W=.856, p<.05), therefore, parametric statistical analyses with these variables were conducted using square-root transformed data. HAI scores (Shapiro-Wilk W=.803, p<.05) were not normally distributed and parametric analyses with this variable were conducted using logarithm transformed data. Finally, the STAI-T scores (Shapiro-Wilk W=.845, p=.05) were not normally distributed and parametric statistical analyses with this variable were conducted inverse-square-root transformed scores (Pallant, 2005). The *c* statistic in the light condition was also not normally distributed (Shapiro-Wilk W=.700, p<.05) but transformations were unsuccessful. Parametric analyses were used rather than non-parametric analyses in order to analyse

the data using factorial ANOVA analysis. ANOVA is regarded as robust in the face of deviation from normality but this will reduce the power of analysis [36].

To test between group differences on the questionnaires, MANOVA and f-tests were used as appropriate. The SSDT statistics were then analysed using 2x2x3 mixed model ANOVAS with follow-up tests as appropriate, using an alpha level of .05 and two-tailed p values. These analyses were then repeated using HAI, STAI-T, PHQ-9, PHQ-GAD-7 and SSAS scores as covariates, with the Delaney and Maxwell correction for repeated measures as appropriate [37]. The current study also aimed to replicate previous findings with the SSDT in terms of the effect of the light and the overall effect of perceptual training (regardless of PHQ-15 group), with a view to establishing the reliability of the study methods.

Results

Comparability of groups on questionnaire measures

There were no significant differences in age between PHQ-15 group, [$F(1,64)=.21$, $P=.65$, partial $\eta^2=.003$] or perceptual training conditions, [$F(1,64)=2.15$, $P=.13$, partial $\eta^2=.063$]. Furthermore, there was no significant PHQ-15 group x perceptual training group interaction for age [$F(2,64)=1.275$, $P=.286$, partial $\eta^2=.038$]. Chi square analyses also revealed no significant association between gender and PHQ-15 group, [$\chi^2=.22$, $P=.64$] and no significant association between gender and perceptual training condition, [$\chi^2=1.07$, $P=.59$].

There were no differences between the high PHQ-15 participants who completed the experimental phase and those who were identified during screening but did not proceed to the experimental phase on the PHQ-15 [$F(1,67)=.025$, $P=.874$, partial $\eta^2=.000$] or SSAS [$F(1,67)=.361$, $P=.550$, partial $\eta^2=.005$]. Likewise, there were no differences between the low PHQ-15 participants who did and did not complete the experimental phase on the PHQ-15 [$F(1,93)=.002$, $P=.962$, partial $\eta^2=.000$] or SSAS [$F(1,93)=.528$, $P=.469$, partial $\eta^2=.006$].

Table 2 presents descriptive statistics for the questionnaires. MANOVA revealed a statistically significant main effect of PHQ-15 group [$F(5,60)=8.00$, $P<.001$, partial $\eta^2=.400$]. The high PHQ-15 group scored significantly higher than the low PHQ-15 group on the HAI ($F(1,64)=14.00$, $P<.001$, partial $\eta^2=.180$), PHQ-9 ($F(1,64)=36.54$, $P<.001$, partial $\eta^2=.363$), STAI-T ($F(1,64)=14.93$, $P<.001$, partial $\eta^2=.189$), PHQ-GAD-7 ($F(1,64)=20.80$, $P<.001$, partial $\eta^2=.245$), and SSAS [F(1,64)=5.23, P<0.01, partial $\eta^2=.076$. The main effect of perceptual training group [F(10,120)=.737, P=.688, partial $\eta^2=.058$] and PHQ-15 group x perceptual training condition interaction did not reach significance [F(10,120)=.527, P=868, partial $\eta^2=.042$].

Table 2: Medians and interquartile ranges of questionnaire scores for the six groups

PHQ-15 group	Training condition (N)	PHQ-15	SSAS[a]	HAI	PHQ-9	PHQ-GAD-7[a]	STAI-T
Low	No (14)	3.5 (3.3)	24.5 (7.2)	11.0 (8.8)	4.0 (4.0)	3.0 (3.0)	37.5 (13.8)
	Low (14)	4.0 (2.3)	22.9 (7.2)	10.0 (4.3)	3.5 (3.5)	2.7 (2.1)	34.5 (10.8)
	High (11)	4.0 (4.0)	23.5 (7.9)	11.0 (8.0)	4.0 (3.0)	2.7 (2.2)	34.0 (6.0)
High	No (10)	11.0 (2.3)	26.6 (6.0)	11.5 (9.0)	7.5 (8.3)	5.0 (3.4)	42.0 (16.3)
	Low (10)	11.0 (3.0)	29.3 (8.2)	16.5 (8.8)	10.0 (6.5)	7.4 (4.3)	44.0 (10.0)
	High (11)	13.0 (7.0)	27.0 (6.3)	17.0 (5.0)	8.0 (4.0)	6.2 (3.4)	43.0 (15.0)

[a] Means and standard deviations reported due to normality of the data.

SSDT analyses

Assessment of Hypothesis 1

Table 3 suggests that high PHQ-15 participants in the group report more false alarms than low PHQ-15 participants in the no perceptual training condition in the light-present and light-absent trials. This main effect of PHQ-15 group on false alarm rate at baseline was significant on light-present trials [t(22)=-2.18, P<.05, eta squared=.178] and on light-absent trials [t(22)=-2.13, P<.05, eta squared=.171]. However, there was no

significant difference in response bias between high and low PHQ-15 scorers at baseline on light-present trials [t(22)=.017, P=.986, eta squared=.001] or light-absent trials [t(22)=.24, P=.811, eta squared=.003]. A power calculation indicated that 6008 participants would be needed in each group to have an 80% chance of detecting a significant difference at the 0.05 alpha level (2-tailed). Therefore, lack of effect of group on response bias is not due to low power. This finding, coupled with a significant between group difference for false alarms suggests that the group difference is specific to false alarms and not to response criterion more generally. This supports the predictions of the Brown [10] model.

Table 3: Mean hit and false alarm rates, tactile sensitivity index and response criterion statistics (± SD) for light present and light absent conditions across PHQ-15 groups and perceptual training conditions.

PHQ-15 group	Training condition	Light	% Hit Rate	% FA Rate[b]	d'	c[b]
Low	No	Present	58.53 (30.95)	12.20 (15.85)	1.48 (1.19)	0.41 (0.82)
		Absent	48.43 (26.47)	7.32 (11.59)	1.38 (0.91)	0.81 (0.77)
	Low	Present	59.23 (21.46)	8.54 (19.51)	1.47 (0.75)	0.42 (0.79)
		Absent	44.42 (22.28)	7.32 (20.12)	1.26 (0.79)	0.81 (0.66)
	High	Present	69.51 (13.04)	13.41 (14.63)	1.76 (0.64)	0.34 (0.53)
		Absent	49.33 (15.35)	8.54 (4.88)	1.36 (0.59)	0.74 (0.28)
High	No	Present	45.37 (24.46)	26.83 (24.39)	0.58 (0.96)	0.61 (0.77)
		Absent	35.12 (19.08)	17.07 (24.39)	0.50 (0.61)	0.70 (0.71)
	Low	Present	54.39 (23.50)	9.76 (14.02)	1.39 (1.14)	0.49 (0.61)
		Absent	46.83 (22.62)	7.32 (18.29)	1.13 (0.97)	0.61 (0.46)
	High	Present	64.86 (15.21)	15.85 (12.20)	1.62 (0.82)	0.41 (0.21)
		Absent	52.22 (10.02)	8.54 (12.20)	1.48 (0.64)	0.65 (0.27)

[b]Median and interquartile range reported due to the non-normality of the data.

Assessment of Hypothesis 2

Table 3 shows that high PHQ-15 participants who completed either of the perceptual training protocols reported fewer false alarms than the high PHQ-15 group who did not receive any perceptual training. In contrast, the false alarm rate appears relatively stable across perceptual training conditions for the low PHQ-15 group. However, the PHQ-15 group x perceptual training interaction was not statistically significant [F(2,64)=1.416, P=0.250, partial η^2=.042]. The medium effect size (Pallant, 2005) suggests that this finding may be non-significant due to the small sample size. The analysis showed that 223 participants (37 in each condition) would be needed to have 80% power to calculate this interaction effect at the 0.05 level.

Table 3 shows that the perceptual training does not influence response criterion for either low or high symptom reporters. The PHQ-15 group x perceptual training interaction on response criterion was not statistically significant [F(2,64)=.002, P=.998, partial η^2=.000].

Secondary analyses

Effect of light

Across groups, there were significantly more false alarms [F(1,64)=11.46, P<0.01, partial η^2=.152] and hits [F(1,64)=87.90, P<0.001, partial η^2=.579] reported when the light was present. Tactile sensitivity was increased in the presence of light [F(1,64)=10.87, P<0.01, partial η^2=.145], as was response criterion [F(1,64)=45.756, P=.000, partial η^2=.417].

Effect of perceptual training

The main effect of perceptual training on false alarm rate [F(2,64)=1.22, P=.303, partial η^2=.037] and on hit rate [F(2,64)=1.953, P=.150, partial η^2=.058] did not reach significance. The main effect of perceptual training on tactile sensitivity approached

significance [F(2,64)=2.718, P=.074, partial η^2=.078]. However, a main effect of perceptual training on response bias was not significant [F(2,64)=.431, P=.652, partial η^2=.013].

Discussion

The findings indicate that high symptom reporters reported more false alarms than low symptom reporters in both light conditions at baseline. As such, these findings replicate the main effect of group on false alarm rate from previous research [18]. However, there was no difference in response criterion between the high and low symptom reporter groups in the no perceptual training condition across both light conditions. This does not reflect Brown et al.'s [18] finding that the high symptom reporters had a lower response criterion than the low symptom reporters on both light trials. This finding is not due to a small sample size as the power calculation shows that 6008 participants would be needed in each group to find a significant effect of PHQ-15 group on response criterion. This finding supports the predictions of the Brown [10] model.

The non-significant between groups finding may also be attributed to methodological differences between this study and that reported in Brown et al [18]. The current study identified high and low symptom reporters using the PHQ-15 whereas Brown et al [18] divided participants according to scores on the SDQ-20. It may be that the two screening measures are, in fact, measuring different concepts leading the experiment to test different somatisation constructs and therefore yielding different findings as a result. The PHQ-15 is a well validated measure of somatic symptoms and is used by general practitioners in clinical practice. Previous studies using the PHQ-15 questionnaire [13] have found a correlation between false alarm rate on the SSDT and symptom reporting, suggesting that the current findings are more likely to be attributable to lack of statistical power than the screening method. Nevertheless, many

of the items pertain to autonomic sensations and could therefore be confounded by presenting or hypochondriacal somatisation, however the current study controlled for effects of depression, anxiety and health anxiety so this confounding effect would be unlikely. In contrast, the SDQ-20 is a well validated measure of positive and negative pseudoneurological symptoms pertaining to somatoform dissociation. In light of the differences between the phenomena measured by these tools, some of the differences in findings between the current study and previous studies using the SDQ-20 may be attributed to this.

The findings also indicated that the perceptual training protocol did not have an effect on the number of false alarms reported or the response criterion in the high and low symptom reporter groups. These findings do not replicate the findings of McKenzie et al., [15]. Based on the findings of McKenzie et al [15] it was assumed that perceptual training would reduce the number of false alarms reported for both low and high perceptual training groups. One possible explanation for the differences in findings is due to low power. The analysis showed that 223 participants (37 in each condition) would be needed to have 80% power to calculate this interaction effect at the 0.05 level. The perceptual training protocol was intended to increase participants' ability to distinguish the vibration from the light on light-present trials during the SSDT by presenting similar tactile stimulation at a suprathreshold level when paired with a non-informative light. The low perceptual training involved presenting the visual and tactile stimuli pairing infrequently (25% of trials) and the high perceptual training involved pairing the stimuli frequently (75% of trials). It was predicted that there would be a greater reduction in false alarms following the high perceptual training condition than the low perceptual training condition. Due to the low power in the study, no firm conclusions can be drawn from this finding.

Although the current study did not replicate previous findings [15] it is important to reflect upon the fact that the previous findings of McKenzie et al. [15] were

counterintuitive to their predictions which proposed that the training protocol would cause individuals in the high association group to report more false alarms in the light-present trials than the low association group. Johnson et al. [14] claimed that participants often rely on visual information when making decisions about ambiguous tactile stimulation. McKenzie et al. [15] proposed that when the individual perceives the tactile stimulus as ambiguous on the SSDT, the light provides a resolution to this ambiguity by forcing the decision-making process. One possible limitation of the procedure in the current study and McKenzie et al. [15] was that the suprathreshold tactile stimulation might not have been intense enough to make the task of differentiating light from touch difficult enough thereby reducing the force exerted on the decision-making process.

It is also important to consider the influence of the perceptual training protocol on participants' perceptual threshold level. As already discussed, the perceptual training protocol was designed to train people to better distinguish between signal (i.e. vibration) and noise (i.e. light) using suprathreshold stimuli. A by-product of this training, however, may have been that tactile sensitivity in general could have been increased which would consequently increase the participant's perceptual threshold. Although the finding did not reach significance and needs to be treated with caution, the current findings demonstrate to some extent a main effect of perceptual training on tactile sensitivity with sensitivity being higher following the high perceptual training than the low perceptual training in light-absent trials. These findings contrast with McKenzie et al. [15] who found that tactile sensitivity was higher in the low association group compared to the high. These differences may be due to low power with further research with a large sample being indicated.

Due to the analogue nature of the study, the generalisability of the findings is limited. The non-clinical sample was recruited from students with a large proportion being Psychology undergraduates who obtained course credits for participating.

Moreover, participants were identified using scores on the PHQ-15, which only provides a proxy measure of somatisation in a clinical population. It may, therefore, be argued that the findings are more generalisable to physical symptom reporting rather than somatoform disorders in a clinical setting. However, there is some evidence that the findings may generalise to clinical samples with somatisation, given that the mean score on the PHQ-15 for high symptom reporters was comparable to the scores demonstrated by Körber, Frieser, Steinbrecher & Hiller [21] for individuals with somatic symptoms presenting in primary care settings. Furthermore, validity of the high and low symptom reporting groupings in the current study is provided by the high symptom reporting group scoring highly on measures of depression, negative affectivity and anxiety which are known to be associated with somatisation [38].

In summary, this study provides initial findings regarding the effect of perceptual training on somatosensory distortion in physical symptom reporters; although the findings are suggestive, further research with larger sample sizes is needed before firm conclusions can be drawn .

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CHAPTER THREE: CRITICAL EVALUATION

This chapter begins by presenting a summary of each of the main findings in relation to the research aims and hypotheses, followed by a detailed interpretation of the findings in relation to previous literature. Methodological considerations will then be reviewed followed by a review of the limitations of the study. The chapter will conclude by reviewing the clinical implications of the findings and making suggestions for future research.

1. Hypothesis 1: Summary and interpretations of findings

(For statistical analyses see Appendix II)

The study found that there was a significant difference in false alarm rate between high and low symptom reporters in the no perceptual training (i.e. baseline) condition in both light conditions. As such, these findings replicate the main effect of group on false alarm rate from previous research (Brown et al, 2010). However, there was no difference in response criterion between the high and low symptom reporter groups in the no perceptual training condition across both light conditions. This does not reflect Brown et al.'s (2010) finding that the high symptom reporters had a lower response criterion than the low symptom reporters on both light trials.

A power calculation indicated that 6008 participants would be needed in each group to have an 80% chance of detecting a significant difference at the 0.05 alpha level (2-tailed). Therefore, lack of effect of group on response bias is not due to low power. This finding, coupled with a significant between group difference for false alarms suggests that the group difference is specific to false alarms and not to response criterion more generally. This supports the predictions of the Brown (2004) model.

The non-significant between groups finding may also be attributed to methodological differences between this study and that reported in Brown et al (2010). The current study identified high and low symptom reporters using the PHQ-15 (Appendix III) whereas Brown et al (2010) divided participants according to scores on the SDQ-20 (Nijenhuis, Spinhoven, Van Dyck, Van der Hart, & Vanderlinden, 1996). It may be that the two screening measures are, in fact, measuring different concepts leading the experiment to test different somatisation constructs and therefore yielding different findings as a result. The PHQ-15 is a well validated measure of somatic symptoms and is used by general practitioners in clinical practice. Previous studies using the PHQ-15 questionnaire (Brown et al., submitted) have also found a correlation between false alarm rate on the SSDT and symptom reporting, suggesting support for the use of the PHQ-15 as ascreening measure for physical symptom reporting.

However, many of the items in this measurepertain to autonomic sensations and could therefore be confounded by presenting or hypochondriacal somatisation, however the current study controlled for effects of depression using the PHQ-9 (Appendix IV), anxiety using the PHQ-GAD-7 (Appendix V), and the STAI-T (Appendix VI) and health anxiety using the HAI (Appendix VII) and somatosensory amplification using the SSAS (Appendix VIII) so this confounding effect would be unlikely. In contrast, the SDQ-20 is a well validated measure of positive and negative pseudoneurological symptoms pertaining to somatoform dissociation. In light of the differences between the phenomena measured by these tools, some of the differences in findings between the current study and previous studies using the SDQ-20 may be attributed to this.

Brown (2004) argues that for high symptom reporters, their current awareness of the tactile stimuli was altered by their tactile memories and the model therefore predicts lower tactile sensitivity for the high symptoms reporters on this basis. Alternatively, it may predict no change in tactile sensitivity as there may be an increase in hits reported for the same reason.

In contrast to Johnson et al.'s (2006) claims that high symptom reporters require less evidence than low symptom reporters to make a decision regarding the presence of a tactile stimulus, Brown's (2004) model proposes that high and low symptom reporters require exactly the same amount of evidence to make these decisions. Brown's (2004) models therefore accounts for differences in false alarm rates between groups by proposing that high symptom reporters have increased activation levels of symptom memories and hypervigilance to these which impact on and distort their current perception of the tactile stimulus. Furthermore, the model argues that low symptom reporters do not experience this over-activation of memories and therefore do not experience distortion of perception, meaning a lower false alarm rate. It has been argued that the increase in false alarms and response bias towards yes on the SSDT are as a result of the same underlying perceptual mechanisms underlying somatisation (Brown et al., 2010). Specifically, that the overactivation of memories and hypervigilance to sensations provide further evidence to the participant that they have experienced a tactile event and false alarms are reported and response bias towards yes increases. Brown (2004) also proposes that high symptom reporters demonstrate a memory bias and tend to inaccurately recollect bodily symptom related information. Such memories influence the individual's expectations of future symptoms and therefore alter symptom perception.

Somatosensory amplification theory (Barsky 1992) suggests that the perception of somatic symptoms is amplified through hypervigilance to bodily sensations, which increases the perceived intensity of the symptom, and heightened sensitivity to physiological sensations more generally (i.e. a perceptual trait either with a genetic link or acquired from early experiences). Barsky (1992) would predict that high PHQ-15 scorers would attend more to their bodily sensations and therefore be more likely to misinterpret both internal and external tactile stimulation as the tactile stimulation on the SSDT. Similarly to other models of somatisation, this model would predict that such increased attention to bodily sensations would increase both hits and false alarms, increase tactile sensitivity and also increase response bias towards yes.

Rief and Barsky (2005) would interpret a correlation between false alarm rate and PHQ-15 scores as evidence of dysfunctional perceptual filtering. They propose that during the pre-conscious stage sensory signals are constantly being sent from the periphery to the brain and the signals are filtered by neural filtering processes and selected for conscious attention. For healthy people most of these sensory signals do not come into consciousness (Gallagher, 2005), however, for people experiencing somatoform symptoms, the signals are not adequately filtered and more sensations are brought into conscious awareness which can be related to the findings of increased false alarms on the SSDT. As such, Rief and Barsky (2005) would predict more false alarms and demonstrate poorer tactile sensitivity as individuals would be less able to discriminate signal from noise.

The role of sensitisation may be a possible explanation for the correlation between false alarm rate and PHQ-15 scores. Deary, Chalder and Sharpe (2007) suggest that sensitisation is the tendency for a heightened response to stimuli due to prior experience and knowledge of those stimuli. Johnson (2008) described sensitisation as taking place in both the peripheral and the central nervous system, and that it had both biological and psychological components. Through sensitisation, sensory receptor sites come to fire more readily and almost automatically without any additional input beyond peripheral noise. The peripheral noise when applying this theory to the SSDT could be represented by the light, sensations from the skin or heart beat so that over a short period of time the sensory receptor sites fire more automatically and the individual misperceives the presence of the tactile stimulus. Using signal detection theory, the increase in false alarms reported by the high PHQ-15 group may be explained through a more lenient response criterion. It is important to note that response criterion according to signal detection theory is a mathematical construct calculated from hit and false

alarm rate, however, this discussion focuses on the psychological construct of response criterion. Johnson et al. (2006) considers response criterion to be the amount of evidence the individual requires to decide that a stimulus is present amongst irrelevant stimuli and therefore views response bias as a measure of how cautious the individual feels about making this decision. As such, Johnson et al. (2006) suggest that high symptom reporters are less cautious in this decision making and therefore have a lower response criterion and an increase response bias towards yes when compared to low symptom reporters. Johnson et al.'s (2006) research would predict that, if high symptom reporters need less evidence to make a decision on the presence of a tactile signal, more hits would be reported on tactile stimulus present trials on the SSDT. However, additional analyses did not show an increase in hit rate in the current study [$t(22)=1.12$, $P=.276$] which supports previous research (Brown et al., 2010). Evidence to support the link between somatisation and more lenient response criterion has been found to be inconsistent (e.g. Garralda, 2005; Willinger & Aushauer, 2005).

In summary, the current finding that high symptom reporters report more false alarms than low symptom reporters supports the findings of previous studies and therefore provides further support for the validity of the SSDT as an analogue paradigm to investigate somatisation in a laboratory setting (Lloyd et al., 2008; Brown, Brunt, Poliakoff & Lloyd, 2010; Mirams, Poliakoff, Brown & Lloyd, 2010).

2. Hypothesis 2: Summary and interpretations of findings

The current findings indicate that the perceptual training protocol did not have an effect on the number of false alarms reported or response criterion in the high and low symptom reporter groups. It was predicted that the greatest effect of perceptual training would be for the high symptom reporters rather than the low symptom reporters. One possible explanation for this non-significant finding is the effect size and sample size. The power calculation demonstrated that 223 participants (37 in each cell) would be required to obtain a significant PHQ-15 group x perceptual training interaction with the effect size obtained here.

The current findings show that the false alarm rate was similar for the three conditions for the low PHQ group, suggesting that the training had no effect for this group. For the high PHQ group, in contrast, both training conditions are associated with fewer false alarms, but the high training seems to have had less of an effect in light present trials. It is important to treat these discussion points with caution, however, as the findings were non-significant.

The current study aimed to improve the design of McKenzie et al.'s (submitted) study to include a control group that receive no perceptual training and to distinguish between high and low symptom reporters . McKenzie et al (submitted) compared their findings to a control group from a previous data set who had not received any training but had performed on the SSDT. The findings of the current study are similar in some respects to those of McKenzie et al., (submitted). In their study, they investigated the effect of varying the reliability of the light as a predictor of the tactile stimulus on the frequency of false touch reports on the SSDT in the presence of light (McKenzie et al., submitted). The same training protocols were used in the current study. McKenzie et al. (submitted) predicted that individuals in a high association group would make more false alarms in the light-present trials than the low association group. However, the authors found that the high association group did not have an increased number of false alarms, whereas the low association group experienced fewer false alarms reported in both light-present and light-absent trials, a similar effect to that observed here. In the current study there was no increase in false alarms following the high perceptual training in either light conditions but that the low perceptual training reduced false alarm rate in both light conditions.

Furthermore, the current study did not find a significant difference in response criterion for the perceptual training conditions, unlike McKenzie et al. (submitted) who found an increased response bias towards yes following the training protocol in light-present trials but no main effect of training group or group x light interaction. The current study also found that a main effect of perceptual training on tactile sensitivity approached significance when controlling for covariates. This is similar to McKenzie et al. (submitted) who found that tactile sensitivity was higher for the low association group than the high association group.

A possible explanation for the perceptual training protocols influencing false alarm rate across both light conditions is that similar perceptual decision-making processes are occurring during both of the light conditions. Furthermore, it could be argued that the way in which the participants responded to light-present trials influenced how they responded to the light-absent trials (McKenzie et al., submitted) and therefore the light influences the decision-making process at a more general level. Using non-human primates, de Lafuente and Romo (2005, 2006) found that subjective experience was highly correlated with activity in the medial prefrontal cortex whilst activity occurring earlier in the somatosensory processing chain was highly related to stimulus attributes. Therefore, both feed-forward and feed-back mechanisms are involved in

perceptual decision making. On this basis, McKenzie et al. (submitted) suggest that the reduction in false alarms for the low association group led them to become less reliant on top-down information from the light and their own expectations.

McKenzie et al. (submitted) suggest that one mechanism underlying this finding is how readily the representation of the tactile stimulus is activated by the light or their own expectations. The authors propose that the training may have lowered this activation for the low association group. This can be related to Brown's (2004) model which proposes that somatisation is maintained through over-activation of symptom representations. These "rogue representations" are developed by top-down cognitive processes through prior learning and stored in memory. Brown's (2004) model might predict that over activation of rogue representations between the light and the tactile stimulus is occurring during the SSDT. Johnson et al., (2006) would propose that such associations have been developed through prior multisensory experiences. It could be hypothesised that if these representations could be altered through the perceptual training then the number of false alarms would subsequently reduce.

Similarly, if symptom reporters were better able to distinguish signal from noise, they may be less prone to developing such rogue representations, thereby reducing the likelihood of symptoms in the future. Powers, Hillock and Wallace (2009) found that, following training with feedback, participants' ability to distinguish auditory stimuli from visual stimuli improved over time. It could be argued that the perceptual training on the SSDT breaks down previous visuo-tactile associations developed from prior learning and allows the individual to learn lower associations between the sensory modalities that allow signals to be perceived more accurately.

Another factor to consider in this is the effect of future expectations and suggestibility on symptom perception. Symptom perception is guided by our expectations of what we will experience next. Suggestion (e.g. in hypnosis, but also more generally) can alter perception by changing our expectations of what we will experience in the future. The role of expectation links with the predictions made by Brown (2004) as the perceptual training protocols would be predicted to alter the individual's expectations of the tactile sensation being presented when the light is presented, thereby reducing the false alarm rate.

Barsky's (1992) somatosensory amplification model does not make clear predictions about the effect of such perceptual training. Indeed, two opposing hypotheses might be drawn from this model. The first hypothesis would predict that the perceptual training would increase the number of false alarms reported as it would

increase the amount of attention placed on the tactile stimulus by the individual. Participants in the high PHQ group may be particularly sensitive to this, although individuals in the low PHQ-15 group may also exhibit an increase in false alarms, but not to the same extent. It would be expected that this would also increase the hit rate. Another hypothesis might be that perceptual training would reduce false alarms due to the light drawing attention away from that tactile stimulus, thereby reducing amplification of the tactile stimulus and a reduction in the hit rate in the light present condition.

Another possible explanation for the findings can be drawn from Rief and Barsky's (2005) perceptual filtering model. They would predict that the perceptual training would improve individuals' filtering ability and reduce false alarm rate. The model proposes that sensory signals are constantly being sent from the periphery to the brain, with various factors affecting the number and quality of those signals. It could be hypothesised that the light stimulus is a factor that alters the quality of the encoding of the tactile signals. Following this encoding process the signals are filtered by neural filtering processes and selected for conscious attention, however, high symptom reporters have a dysfunctional perceptual filtering system that allows too many signals to be brought into conscious awareness. Brown et al. (submitted) provides further explanation for this and suggests that false alarms on the SSDT are related to dysfunctional filtering of sensory noise which would also be responsible for increased symptom reporting. They propose that these two concepts may not be mutually exclusive from one another and that the dysfunctional filtering system may reduce the reliability of the somatosensory signal as a source of information and therefore force the cognitive system to rely more on top-down factors when processing the somatic experience. Brown et al. (submitted) argue that when the top-down factors contradict normal sensory information, more somatic distortions will occur. The model would therefore predict that perceptual training would have an effect on both groups but there would be a greater effect on high symptom reporters than low symptom reporters. As demonstrated, a reduction in false alarms was observed following both low and high perceptual training in the high PHQ-15 group. Therefore, the Rief and Barsky model (2005) provides some explanation for the current findings.

3. Additional findings: Summary and interpretations

3.1 Effect of light

The current study found that false alarm and hit rates were increased in the presence of light. Moreover, tactile sensitivity and response bias towards yes were increased in light present trials.

Lloyd et al. (2008) found that the presence of light increased the number of false alarms (or illusory touch experiences) reported and the response bias to report the presence of a tactile stimulus on the SSDT. Subsequent studies using the SSDT paradigm also support these findings (Lloyd et al., 2008; Brown, Brunt, Poliakoff & Lloyd, 2010; Mirams, Poliakoff, Brown & Lloyd, 2010). Lloyd et al. (2008) propose that this effect occurs due to the light increasing the activation of tactile representations as proposed in Brown's (2004) model of MUS. Moreover, they propose that the presence of the light increases activation levels of memorial tactile representations through associative mechanisms.

Johnson et al. (2006) hypothesised that the mechanisms underlying the enhancement of false alarm rate and increased response bias towards yes in the presence of the light are produced through multisensory facilitation. More specifically, they purport that the presence of the light in the SSDT enhances the detection of tactile stimuli via cues from multisensory modalities (e.g. Lovelace et al., 2003). Johnson et al. (2006) report that people's experience of the environment around them is developed from multisensory information but that vision is the most relied upon sensory modality and, as such, can dominate and alter other senses. Furthermore, when visual and tactile stimuli provide conflicting information the visual modality not only overrides the tactile perception but alters it also (Pavani, Spence & Driver, 2000). Johnson et al. (2006) argue that the presence of light reduces individuals' response criterion to increase false alarm rate in these trials.

Moreover, studies related to body focused attention in somatisation also offer an explanation to increased detection of tactile stimuli (Kennett, Taylor-Clarke & Haggard., 2001; Taylor-Clarke, Kennett & Haggard, 2004; Schaefer, Flor, Heinze & Rotte, 2006) as well as research demonstrating tactile attention shifts towards close proximal body parts in the presence of neutral visual stimuli (e.g. Kennett, Spence & Driver, 2002). This body of research, therefore, suggests that the effects of the presence of non-informative light in the current study are due to increased attention to the body part (e.g. finger) and thus increased body-focused attention and sensitisation. Through sensitisation, sensory receptor sites come to fire more readily and almost automatically

without any additional input beyond peripheral noise. The peripheral noise when applying this theory to the SSDT could be represented by the light, sensations from the skin or heart beat so that over a short period of time the sensory receptor sites fire more automatically and the individual misperceives the presence of the tactile stimulus.

Zhou and Fuster (1997) found that when the monkeys memorised a visual cue preceding a tactile choice, cells in the primary somatosensory cortex were activated when the visual cue was presented alone. Moreover, similar findings have been demonstrated in human studies. Goldberg, Perfetti, & Schneider (2006), using neuroimaging, found that retrieval of tactile knowledge into working memory during a semantic decision making task indexing tactile knowledge activated the primary and secondary somatosensory cortices and the posterior parietal cortex. This body of research demonstrates that specific cerebral areas and neural pathways play a significant role in the activation of tactile memories which impact on current experience. In the current study, the presence of light can be regarded as the stimulus activating these cerebral areas.

In the case of long term tactile memories, other cerebral areas have been found to play significant roles. Such cerebral regions include the medial temporal and medial prefrontal cortex as well as the perirhinal cortex (Bonda, Petrides, & Evans, 1996; Burton & Sinclair, 2000). Similar to the processes involved in the activation of short-term tactile memories, the light serves to activate long-term tactile memories through neural pathways in these cerebral regions. More specifically, research has shown that reciprocal connections between these cerebral regions and the primary and secondary somatosensory cortices lead to the activation of memories (e.g. De Lafuente & Romo, 2006). This may help to explain the findings of increased false alarm rate and response bias in previous studies in both light conditions. Interestingly, brain areas responsible for tactile perception overlap considerably with those responsible for short term tactile memory, which is related to the mechanisms described in the Brown (2004) model.

4. Methodological considerations

4.1 Ethics

The current study was approved by the School of Psychological Sciences Research Ethics Committee at the University of Manchester (Project number 616/07P; Appendix IX). No specific ethical issues were raised.

4.2 Identifying somatisation

It is generally considered to be difficult to distinguish somatisation from symptoms resulting from general medical conditions. In some cases, the unusual physiological nature of the symptoms is considered by the clinician to be consistent with somatisation and a diagnosis is made; however, this is rare. Many patients who present to their general practitioner are considered to be the "worried well" and their symptoms resolve without intervention. It is therefore unclear what proportion of these symptoms are caused by mild pathology and which are somatic in nature. In more extreme cases, somatisation is identified after medical investigations have failed to identify an organic pathology for the symptoms; however, such investigations can take significant periods of time and can increase the individual's distress and disability. One of the difficulties here is that many patients may not receive a diagnosis of somatisation due to not being referred to secondary services for investigation. Therefore, there may be an underestimation of prevalence of somatisation patients presenting in primary care services. Furthermore, Brown (2007) argues that the diagnosis of somatisation or medically unexplained symptoms is one of exclusion rather than inclusion and such terms imply that the symptoms are solely psychological or psychiatric in nature which cannot be concluded simply from excluding organic illness.

In light of a potential underestimation of somatisation in primary care patients, it is important to consider whether excessive symptom reporting is different to somatisation and indeed organic illness. It can be argued that there is a large overlap between these terms and that they may in fact be the same thing with different terminologies. Research has shown that individuals with organic illness can still be diagnosed with somatisation and therefore demonstrates that these terms are not mutually exclusive (Aragonès, Labad, Piñol, Lucena, & Alonso, 2005). Furthermore, patients whose physical symptoms are related to a medical illness may also receive a diagnosis of somatisation if their level of disability exceeds the level expected for their condition irrespective of the fact that reports related to expected disability are subjective and difficult to validate (Brown, 2007). It could therefore be argued that patients who receive a diagnosis of somatisation are in fact those who report a large number of symptoms and high levels of distress and disability.

These issues highlight the difficulty of identifying individuals with a tendency towards somatisation in research such as that described in this thesis. A non-clinical population was used for this study with high and low symptoms reporters being identified by scores on the PHQ-15, as used in previous studies (Brown et al., submitted). Moreover, it is difficult to distinguish the different forms of somatisation

identified by Kirmayer and Robbins (1991b) as presenting, hypochondriacal and functional forms of somatisation overlap to some degree. Of particular importance in this thesis is the need to distinguish between functional somatisation and physical symptoms that are associated with psychiatric problems, such as anxiety and depression. Furthermore, comorbidity between depression and anxiety and somatisation has been found to be very high (Smith et al., 2005). It was therefore important to take this into account when recruiting participants for this study. A battery of self-report measures was identified to control for any influence of comorbid psychiatric disorders on symptom reporting.

Another methodological consideration is whether there are alternative methods that might be a better measure of somatosensory distortion than the SSDT. In chapter one studies using placebo and nocebo paradigms were discussed as well as studies using perceptual illusions. Brown (2004) specifically claims that somatoform symptoms are distortions of somatic awareness due to over-activation of symptom representations in memory and, as such, reflect the fact that bodily experiences are interpretations rather than direct representations of sensory information based on both top-down and bottom-up factors. The influence of top-down factors, such as, hypervigilance are discussed in other models as well as Brown's (2004), however, these models do not assume that somatic symptoms are distortions in awareness due to over activation of representations. Therefore, the use of perceptual illusions may be a useful way to investigate such perceptual distortions. Indeed there have been many perceptual illusions that have demonstrated how bodily experiences can be distorted. The rubber hand illusion has been used extensively to demonstrate distortions in bodily perceptions (Botvinick & Cohen, 1998; Tsakiris & Haggard, 2005; Durgin, Evans, Dunphy, Klostermann, & Simmons, 2007; Miles, Poliakoff, & Brown, in press). In the rubber hand illusion, stroking a rubber hand whilst simultaneously stroking the participant's hand in their line of vision can cause the participant to perceive the rubber hand as part of their body (Botvinick & Cohen, 1998). Recently, Miles, Poliakoff and Brown (in press) have used this perceptual illusion to investigate the effects of the rubber hand illusion on high and low symptom reporters. The authors proposed that if responsiveness to such bodily illusions represents individual differences to everyday bodily experiences and perceptions then bodily illusions may be a more objective way to investigate perceptual distortions in clinical populations. The study found that the low symptom reporters responded more highly to the rubber hand illusion than the high symptom reporters than the low symptom reporters which supports suggestion by Brown (2004) that

somatisation occurs due to over reliance on top-down cognitive factors during the processing of bodily representations. The rubber hand illusion essentially "tricks" this top-down process by providing discrepant sensory information which distorts perception. The study therefore found that high symptom reporters are more susceptible to this than low symptom reporters (Miles, Poliakoff, & Brown, in press) and provides an interesting starting point for using perceptual illusions to test somatoform distortion further.

The role of memory and expectations are central factors in the Brown (2004) model and therefore placebo and nocebo research paradigms may be a useful way of measuring somatoform symptoms. Research on placebo effects (Colloca, Sigaudo, & Benedetti, 2008; Price, Finniss, & Benedetti, 2008) has demonstrated the role of expectancy on perception and placebo research on functional somatic syndromes, such as, irritable bowel syndrome has shown that these patients are highly influenced by the placebo effect (Vase, Robinson, Verne and Price, 2003).

A final methodological consideration to take into account is the extent to which a harmless vibration really mimics an unpleasant physical symptom, which raises the question of whether it would be possible to develop a version of the SSDT that uses painful stimuli. Such research raises ethical issues and it would be important to establish the SSDT as a reliable and valid method of testing somatoform symptoms before developing further variants of the task.

4.4 Self-report questionnaire measures

It is common for researchers to use self-report measures to identify symptom reporters and measure psychiatric disorders. Although many of the measures used demonstrate good reliability and validity, self-report measures rely on the individual's recollection of symptoms which are often unreliable (Schrag, Brown, & Trimble, 2004; Simon & Gureje, 1999) and may lead to an overestimation of somatic symptoms (Kroenke, 2001). Each of the measures used in the current study will be discussed separately below.

The current study used the Patient Health Questionnaire-15 (PHQ-15) to identify high and low symptom reporters. The PHQ-15 measures how much the individual has been bothered by 15 somatic symptoms during the past four weeks. The symptoms account for 90% of physical symptoms presented in outpatient settings (Kroenke, Spitzer, Janet & Williams, 2002), as well as 14 of the 15 most prevalent DSM-IV somatization disorder symptoms (Liu, Clark, & Eaton, 1997) and as such is a well validated measure of somatic symptoms (Kroenke et al., 1998a). Scale reliability for the

current study was good (Cronbach's α = .86) and comparable to the original validation report (α = .80; Kroenke, Spitzer, Janet & Williams, 2002). It was appreciably higher than in some previous research (α = .56; Katzer et al., in press). Although the PHQ-15 measures the total symptom count and severity of a broad range of somatic symptoms, it also measures symptoms that may be related to autonomic sensations associated with depression and anxiety and scores may therefore be influenced by presenting and hypochondriacal somatisation. However, the current study controlled for these influences. An important point to make here is that statistical control of these factors does not eradicate the influence of these factors and therefore methodological control would have been more beneficial. Such methodological control could involve exclusion of people who are presenting and hypochondriacal somatisers. However, this would have further reduced the sample size for the current study and therefore further reduced the power of the findings.

The findings of the current study suggest that the PHQ-15 was a successful proxy measure of somatisation and excessive physical symptom reporting. The high PHQ-15 group demonstrated higher levels of health and generalised anxiety (as measured by the HAI and the PHQ-GAD-7), depression (as measured by the PHQ-9), negative affectivity (as measured by the STAI-T) and somatosensory amplification (as measured by the SSAS). This study supports previous evidence of these constructs correlating with somatisation (e.g. Barsky et al., 1988; Watson & Clark, 1984) as well as supporting the findings of Brown et al. (2007) who found correlations between the STAI-T, the SSAS and somatisation.

A cut off score of 10 was chosen for the high PHQ-15 group as this score was used as a screening cut-off in primary care settings (Körber, Frieser, Steinbrecher & Hiller, in press) and therefore was comparable to a sub-clinical population. The findings discussed above highly suggest that this reduced cut-off was acceptable. All participants completed the SSDT experiment within a month of completing the PHQ-15 and therefore it was not considered necessary for participants to complete the measure again. However, this design is not ideal and that a more accurate estimate of current somatisation may have been obtained if the measure had been repeated during the experimental session. The benefits of this would be that the sample would contain current somatisers that were currently scoring high on the measure and participants who were currently scoring low on the measure and therefore reduce the risk of testing people who no longer fall into these scoring groups. However, a potential problem with

repeating the measure is that the duration of the experiment will be further extended which will potentially increase fatigue and impact on performance on the SSDT.

An alternative measure of somatic symptoms that could have been used to distinguish between high and low symptom reporters is the Somatoform Dissociation Questionnaire (SDQ-20; Nijenhuis, Spinhoven, Van Dyck, Van der Hart, & Vanderlinden, 1996). The SDQ-20 measures somatoform dissociation by identifying positive and negative pseudoneurological symptoms and therefore may be a more specific measure of functional somatisation per se (Brown et al., 2007; Brown et al., 2010) . Somatoform dissociation is defined by a lack of integration between somatic components of experience such as bodily reactions and functions (Nijenhuis, Spinhoven, Vanderlinden, Van Dyck, & Van der Hart, 1998). Similarly to Nijenhuis et al (1998), Brown (2004) identifies somatisation as a form of dissociative phenomenon. The Brown (2004) model assumes that somatoform symptoms develop due to a horizontal dissociation between the different levels of cognitive processing. This reflects Janet's (1889) claims that somatic symptoms develop due to a distortion in awareness occurring from information becoming fixed in the cognitive processing system. However, the Brown (2004) model rejects some of the ideas of the original dissociation theory, primarily the idea that somatoform symptoms are the product of a pathological process. Brown (2004) argues that such symptoms are normal psychological phenomena that develop as a result of small disruptions in the cognitive processes involved in the everyday control of perceptual experience and behaviour (Escobar, Waitzkin, Cohen Silver, Gara, & Holman, 1998; Fink, Sørensen, Engberg, Holm, & Munk-Jørgensen, 1999).

The current study aimed to recruit a large sample of non-clinical participants who were representative of the patients presenting with somatic symptoms to their GP. It was therefore decided that the PHQ-15 measure would be more appropriate in identifying a non-clinical representative of this population. Chapman (2009) has demonstrated that the PHQ-15 measure has better psychometric properties than the SDQ-20 in a study using a non-clinical population.

As described in the literature presented in chapter one, somatosensory amplification is a perceptual mechanism underlying somatisation. The Somatosensory Amplification Scale (SSAS; Barsky, Goodson, Lane & Cleary, 1988*)* was designed to measure individuals' tendencies to experience somatic sensations as unpleasant and also correlates with individuals diagnosed with hypochondriasis within DSM-III-R (APA, 1987) (Barsky et al., 1990) and as such controlled for hyponchondriacal somatisation.

This expanded upon previous research (Brown, Brunt, Poliakoff & Lloyd, 2010) suggesting that more stringent controls for types of somatisation needed to be incorporated into future research.

Watson and Pennebaker (1989) purport that high trait negative affective individuals tend to report a large number of health complaints and may be more sensitive to uncomfortable sensations.. Furthermore, a neurotic personality trait relates to anxiety and depression (Matthews, Deary, & Whiteman, 2003), heightened sensitivity to general stressors (Bolger & Schilling, 1991) and increased incidence of physical illness (Huovinen, Kaprio, & Koskenvuo, 2001; Denollet & Van Heck, 2001). Neurotic personality traits have also been found to be associated with somatisation (Kirmayer, Robbins, & Paris, 1994; De Gucht, Fischler, & Schweitzer, 2004a; De Gucht, Fischler, & Schweitzer, 2004b) and more specifically, functional dysphonia (Deary, Scott, & Wilson, 1997), chronic fatigue syndrome (Deary, 2001) and irritable bowel syndrome (Hazlett & Stevens et al., 2003). The current study, therefore, controlled for negative affectivity by using several measures which also controlled for different categories of somatisation.

The current study compliments and supports growing research demonstrating the SSDT to be a useful laboratory paradigm to investigate individual's tendency to experience somatosensory symptoms (e.g. Lloyd et al., 2008; McKenzie et al., 2010; Brown et al., 2010; Mirams et al., 2010). Furthermore, the current study expands upon the findings of McKenzie et al., (2010) to suggest that perceptual training may be beneficial for patients to aid them to discriminate symptoms from "clinical noise" and therefore reduce excessive physical symptom reporting. However, due to the low statistical power of the current study, such claims are made with caution.

5. Limitations of the study

Participants were recruited to the study through poster advertisement (Appendix X) and email advertisements (Appendix XI) via the University research volunteering website. Every student within the university receives research volunteering email regularly. Therefore the study was advertised widely. Although great efforts were made to recruit a large sample, due to the time constraints and inclusion criteria of the current study, only 56% of those individuals who responded to the adverts were invited to take part in the experimental phase of the study. Of those who were invited to take part, only 58% consented to and completed the study. One of the reasons for this can be attributed to difficulties regarding communication with participants. Once participants completed

the online questionnaires, the experimenter contacted those who were eligible to take part via email to organise an appointment for the experiment session. One of the problems with this was that participants could take long periods of time to respond to the email and therefore testing was delayed or no further contact was made. Another explanation for this poor attrition rate was due to 11 participants failing to reach perceptual threshold on the SSDT. Data was not recorded in order to compare the participants who did not reach perceptual threshold with participants who did and therefore it is not possible to investigate whether these groups are different in any way. As a consequence of these factors, the sample sizes for each of the six experimental conditions were small. Power calculations revealed that 37 participants were required in each condition to demonstrate a main effect of perceptual training on false alarms. The small sample sizes therefore contributed to the lack of positive findings. As large sample sizes are needed in order to demonstrate effects of perceptual training on false alarms, prospective studies need to find ways to improve recruitment and retention rates. One possible way of doing this would be to have each participant complete the screening measures and the SSDT in one session so as to reduce the risk of the participant only completing the first stage of the study. A potential difficulty of this, however, is that not all participants who volunteer to take part are eligible for the study which would pose an ethical problem as participants are being tested measures (e.g. the SSDT) that are not relevant to the study and this would place more demand on the experimenter.

Due to the analogue nature of the study, the generalisability of the findings is limited. The non-clinical sample was recruited from students from a university population with a large proportion being Psychology undergraduate students who obtained course credits for taking part. Moreover, participants were identified using scores on the PHQ-15, which only provides a proxy measure of functional somatisation. It may, therefore, be argued that the findings are more generalisable to analogue physical symptom reporting rather than somatisation in a clinical setting. However, there is some evidence that the findings may be related to the underlying mechanisms of somatisation as the mean score on the PHQ-15 for high symptom reporters was comparable to the scores demonstrated by Körber, Frieser, Steinbrecher & Hiller, (in press) in primary care settings. It is also important to discuss the validity of symptom reporting relating to performance on the SSDT and whether perceptual training can alter this. McKenzie, Poliakoff and Lloyd (2010) investigated the reliability of using illusory touch reports on the SSDT over time and whether there are robust individual differences

in the tendency to report illusory touch experiences or false alarms on the SSDT. Their findings replicated those of previous studies using the SSDT (Brown et al., in press; Johnson et al 2006; Lloyd et al 2008; Mirams et al 2010) showing that presenting bi-modal visuo-tactile stimuli enhances the detection of the near perceptual threshold tactile stimulus. Furthermore, the study found that the tendency to report false alarms and therefore to experience illusory touch was stable over time and has a trait-like component (McKenzie et al., 2010) suggesting that the measurement of reported false alarms and response criterion are robust phenomena and a reliable way of testing signal perception in the laboratory. However, prospective research is warranted to establish a further evidence base for the validity of the SSDT in somatoform disorders, using clinical samples, identified using a more detailed method of evaluating symptom experiences, for example, medical records.

Several methodological limitations were observed and will be discussed separately. The overall duration of the SSDT experiment varied according to the length of time required to complete the thresholding procedure and whether the participant was allocated to a perceptual training protocol. Therefore, the SSDT task lasted between 45-60 minutes. The SSDT task is repetitive in nature and, coupled with the relatively long duration of the task, concentration and performance on the task may have been reduced due to fatigue. Measures to encourage prolonged concentration on the task could be integrated into prospective studies, for example, prompts on the computer screen to remind participants of the importance of the task and incorporation of more breaks into the SSDT program. However, incorporating more breaks in the program may cause the perceptual threshold to alter due to any alight movements of the participants' fingers during the break. Furthermore, a large battery of self-report measures was used during the study which added additional time onto the study and may have contributed to any fatigue experienced by the participants.

During the thresholding procedure, the intensity of the vibration was varied on each block of trials. The participants were required to press the "Y" key on the keyboard if they did feel the vibration and "N" if they did not. At the end of each block of the thresholding procedure, the experimenter increased the intensity of the vibration if the participant reported feeling the vibration less than 40% of the time and decreased the intensity of the vibration if the participant reported the presence of the vibration on more than 60% of the trials. In order to vary the intensity of the vibration, the experimenter manually turned a dial on the amplifier equipment; however, the dial did not display a numerical range to indicate how far the dial had been moved. Therefore,

the experimenter was not able to record the exact intensity of the vibration at any one point during the thresholding procedure. As such, a degree of bias was introduced using this methodology.

This thresholding procedure poses another potential limitation to the methodology as the participant is asked to respond to the presence or absence of the vibration using a one-interval response (i.e. yes or no) directly after the trial, therefore, the selected perceptual threshold is dependent not only on tactile sensitivity but also on response bias towards responding "yes" (Katzer, Oberfeld, Hiller & Witthöft, in press). For those participants in the perceptual training conditions, they received such training prior to the thresholding procedure and therefore, their tactile sensitivity may have been altered and impacted on their performance on the threshold procedure. The training phase was designed to increase participants' perception of tactile stimulation by receiving the tactile stimulation at a suprathreshold level as well as receiving feedback regarding whether their response was correct or incorrect to the presence of the vibration. Consequently, it is unclear whether their perceptual threshold may have been reduced or increased (Katzer, Oberfeld, Hiller & Witthöft, in press). A more precise method of selecting perceptual threshold levels, as proposed by Katzer et al. (in press), would be to administer three blocks of threshold trials. The first block, a practice block, of ten trials is presented at maximum vibration intensity (i.e. 100 on the scale) to familiarise the participant with the vibration. After two consecutive correct responses the intensity was decreased by 10 units. Likewise, if an incorrect response was reported then the intensity would be increased by 10 units. This procedure continues until there is a correct response rate of 70.7% (Levitt, 1971). The second block of thresholding trials used the same procedure as the first block but did not include the first ten trials at maximum vibration intensity. In this second phase of thresholding, the direction of the stimulus level sequence alternates from up to down or vice versa (i.e. "reversal"). When eight reversals have been presented the block is terminated and the mean intensity of the trials is taken as the threshold level. A further phase of thresholding then occurs at the end of the SSDT to assess reliability.

Furthermore, the number of thresholding periods was reduced from two, as used by McKenzie et al. (submitted) to one in the current study. Although this reduced the overall duration of the experiment and the demands placed on the participant, it restricted the opportunity to re-assess the individual's perceptual threshold throughout the experiment. Therefore, perceptual threshold may have altered during the SSDT due to factors such as movement and fatigue and, as such, impacted on the findings. This

may provide an explanation for hit rate being below 40% for one of the cells (high PHQ-15 group, no perceptual training condition, light absent trials).

In the current study, the number of blocks of trials during the SSDT experiment was reduced from four, as used in McKenzie et al. (submitted) to two in order to reduce the overall duration of the experiment. McKenzie et al. (submitted) did not find a main effect of block on false alarm rate in their study using four blocks and as such it was deemed appropriate to reduce the number of blocks to reduce experimental demands such as duration. However, this reduction will have resulted in fewer false alarms and hits being recorded, as well as the other SSDT statistics, which may have resulted in a reduction in the variability of the false alarm rate demonstrated and consequently reduced the chances of obtaining a significant finding.

The start of each trial in the current study was signalled by a visual start cue (i.e. a green arrow pointing toward the finger). It is unclear as to how this may interfere with the effect of the non-informative light used as the visual stimuli in the SSDT and whether there is an argument for using a start cue derived from another sensory modality, for example, an auditory cue (Katzer et al., in press). However, McKenzie at al., (2010) found that there was no difference in performance when an auditory stimulus was used instead of a visual stimulus. Further investigation of this phenomenon is needed.

6. Clinical implications

The health care costs associated with somatisation are considerable with some estimations showing that treatment costs are nine times greater than for "average" primary care patients (Smith, Monson, & Ray, 1986). The majority of these treatment costs are due to medical investigations rather than psychiatric treatment or psychological intervention (Rost, Kashner, & Smith, 1994). It is therefore important to consider the clinical implications of the current research and indication for prospective research to guide the development of effective treatment packages to reduce the financial burden on the health care services and reduce the distress and disability caused by the experience of somatosensory distortions and symptoms.

The current findings are suggestive of the potential for the perceptual training protocols to be used clinically although further research is indicated to make firm conclusions. However, certain modifications to the training protocol would be needed as it is unlikely that the patients' difficulties are due to difficulties discriminating touch from non-touch. A more useful training protocol may be one in which the stimuli are more reflective of their normal symptoms.

Alternatively, psycho-education techniques focusing on the psychological definition of response criterion and the factors that may affect this may help to change the criteria they use to judge whether a symptoms is present or absent. Furthermore, Brown (2004) suggests that clinical assessment should use a biopsychosocial framework to link prior symptom episodes or adverse life experiences to current symptoms which may help to normalise the symptom experience and improve therapeutic engagement. The current findings demonstrate that factors other than direct sensory information can influence perceptions of signals. Brown (2004) would suggest that analogies and examples of how perceptual illusions can distort subjective experience may be useful in reducing the generation of inaccurate symptom perceptions. Perceptual illusions such as the "Pinocchio illusion" (Lackner, 1988) and a tailored form of the rubber hand illusion (Botvinick & Cohen, 1998) may be useful therapeutic tools. Likewise, analogies using placebo and nocebo effects (e.g. Spiegel, 1997) may be effective, as might demonstrating hypnotic phenomena (Brown, 2004).

Alternatively, the perceptual training protocol may be a more effective research paradigm than a therapeutic tool and the evidence gleaned from such studies can be incorporated into tailored treatment packages, such as, cognitive behavioural therapy. It has been suggested by Brown (2004) that attentional training techniques (Wells, 1990; 2000) may be used to decrease symptom memory activation and to modify factors that increase symptom-memory focused attention. However, further research is needed to establish the efficacy of this technique in somatisation. Likewise, behavioural experiments could be tailored to patients to aid them to distinguish symptoms from peripheral factors by increasing the ambiguity between these factors to force the decision-making process during therapy sessions.

The potential of the PHQ-15 as a useful clinical measure of functional somatisation was highlighted in the current study as high scorers reported more false alarms on the SSDT and therefore demonstrated more illusory touch sensations than low scorers. However, further clinical studies are needed to investigate its clinical utility as one measure that may aid accurate diagnosis of somatisation.

7. Prospective research

Some recommendations for future research and modifications of the SSDT have been suggested throughout the discussion and will be discussed further here. Due to the low power of the current study, further research is indicated to repeat the study with a much larger analogue study in order to decide whether the findings are high enough to

warrant conducting a much more difficult and time-consuming study with a clinical population.

Prospective research should refine the thresholding procedure to reduce the effect of response bias on selection of perceptual threshold. One possible method is proposed by is to use a two-alternative-forced-choice task which allowed two observation levels to reduce the effects of response bias on perception threshold on the SSDT (Green & Swets, 1966; Katzer et al., in press). They combined the SSDT paradigm with a procedure used in psychophysics known as the transformed up-down adaptive procedure (Levitt, 1971) to identify perceptual threshold and vibration intensity which corresponded to a performance level of 70.7% correct responses and was therefore identical for all participants.

Further neuropsychological research is indicated to further understand the neural mechanisms underlying somatisation and the findings of the current study. Such research should incorporate a range of fMRI studies that would investigate areas of activation during the SSDT which would expand understanding of the perceptual mechanisms underlying somatisation. Imaging studies would also help to further delineate somatisation theories. Indeed, some research in this area has already been demonstrated in non-human research. An example of this work is provided by De Lafuente and Romo (2005) who demonstrated that when decisions regarding the presence or absence of a near-perceptual threshold tactile stimulus to the finger were being made, the medial parietal cortex was activated. Furthermore, approximately 70% of the false alarms recorded in this study were predicted due to an increase in activity in this cerebral area. Further imaging research could include investigating the cerebral areas activated in different types of somatisation and functional somatic syndromes, for example, chronic pain and IBS when performing on the SSDT. The Brown (2004) model would predict no difference in performance between symptom types and different types of somatisation.

An interesting research area to investigate would be whether the findings of the current study and previous studies looking at visual and tactile sensory modalities on the SSDT (Brown et al., 2010; Brown et al., submitted; Lloyd et al., 2008; McKenzie et al., submitted; Mirams et al., 2010) can be replicated using alternative combinations of sensory modalities, for example, auditory and tactile. Brown (2004) would argue that the same effects would be observed regardless of sensory modality.

Finally, exploration into alternative ways of measuring somatosensory distortion would be useful. As discussed above, research designs using placebo and nocebo paradigms and perceptual illusions may be useful alternatives to the SSDT.

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APPENDICES

APPENDIX I: JOURNAL GUIDELINES

Journal of Psychosomatic Research
Official Journal of the European Association for Consultation-Liaison Psychiatry and Psychosomatics and affiliated with the International College of Psychosomatic Medicine
Guide for Authors

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Full Length Papers. Full length research papers will not normally be more than 4000 words in length and will preferably be shorter. Submission of a paper to the **Journal of Psychosomatic Research** will be held to imply that it represents original research not previously published (except in the form of an abstract or preliminary report), that it is not being considered for publication elsewhere, and that if accepted by the **Journal of Psychosomatic Research** it will not be published elsewhere in the same form in any language without the consent of the Publisher. Major papers of topical content will be given priority in publication.

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Manuscripts should conform to the uniform requirements known as the 'Vancouver style' (International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. N Engl J Med 1997; 336:309-315). *The Editors and Referees attach considerable importance to a succinct and lucid prose style and well organized tables. Authors whose native language is not English are advised to seek help before submission. Statistical procedures should be clearly explained.*

Manuscripts should be typed with wide margins, double-spaced on one side of standard A4 or 8.5" x 11" paper. The format should be as follows:

Title page. This should contain (a) the **title** of the article; (b) a short **running head;** (c) name of **department** where the work was conducted; (d) **names of the each author** with highest academic degree; (e) name, address, phone and fax of **author responsible for correspondence** and to whom requests for reprints should be addressed; (f) up to six **keywords** should be listed in alphabetical order after the abstract. These terms should optimally characterize the paper.

Structured Abstract. This should be subdivided under the headings *Objective, Methods, Results, and Conclusion* and should not exceed 250 words.

Text. This should be divided into sections with main headings: Introduction, Method, Results and Discussion. Accepted papers will usually be between 2000 and 4000 words in length.

Acknowledgments. These must include mention of any source of funding outside the basic funding of the host institution.

References. These should be numbered consecutively in the text in the order in which they are first mentioned and be so denoted in the list. Their form should be that adopted by the US National Library of Medicine, as used in the Index Medicus and as recommended in Huth EJ, *Medical Style & Format*.

1. Ingham JC, Miller P McC. Self-referral to primary care: symptoms and social factors. J Psychosomatic Res 1986;30:49-56.

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Tables. Each should be on a separate sheet, numbered consecutively in Roman numerals.

Figures Each should be on a separate sheet, and numbered consecutively. Captions should be on a separate sheet. The number of illustrations should be kept to a minimum.

Color illustrations are not normally acceptable. Authors may be asked to support the costs of color reproduction.

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APPENDIX II: STATISTICAL ANALYSES

Table 1: Summary table for between-subjects ANOVA comparing age of participants in PHQ-15 group and perceptual training condition.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	169.967	5	33.993	1.437	.223	.101
Intercept	33700.259	1	33700.259	1424.213	.000	.957
PHQ-15 group	4.965	1	4.965	.210	.648	.003
Perceptual training condition	101.765	2	50.883	2.150	.125	.063
PHQ-15 x perceptual training condition	60.339	2	30.170	1.275	.286	.038
Error	1514.392	64	23.662			
Total	36255.198	70				
Corrected Total	1684.358	69				

Table 2: Tests of between-subjects effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	169.967	5	33.993	1.437	0223	.101
Intercept	33700.259	1	33700.259	1424.213	.000	.957
Training group	101.765	2	50.883	2.150	.125	.063
PHQ group	4.965	1	4.965	.210	.648	.003
Training group x PHQ group	60.339	2	30.170	1.275	.286	.038
Error	1514.392	64	23.662			
Total	36255.198	70				
Corrected Total	1684.358	69				

Table 3: Summary table for 2 x 2 x 3 mixed model ANOVA with one within-subjects factor (light: absent vs. present) and two between-subjects factors (PHQ-15 group: high vs. low and training condition: no training vs. low training vs. high training), taking hits as a dependent variable.

Source		Type III Sum of Squares	df	Mean Square	F	Sig	Partial Eta Squared
Light	Spherity Assumed	0.544	1	0.544	87.897	0.000	0.579
	Greenhouse-Geisser	0.544	1.000	0.544	87.897	0.000	0.579
	Huynh-Feldt	0.544	1.000	0.544	87.897	0.000	0.579
	Lower-bound	0.544	1.000	0.544	87.897	0.000	0.579
Light x Training	Spherity Assumed	0.025	2	0.013	2.031	0.140	0.060
	Greenhouse-Geisser	0.025	2.000	0.013	2.031	0.140	0.060
	Huynh-Feldt	0.025	2.000	0.013	2.031	0.140	0.060
	Lower-bound	0.025	2.000	0.013	2.031	0.140	0.060
Light x PHQ-15	Spherity Assumed	0.020	1	0.020	3.305	0.074	0.049
	Greenhouse-Geisser	0.020	1.000	0.020	3.305	0.074	0.049
	Huynh-Feldt	0.020	1.000	0.020	3.305	0.074	0.049
	Lower-bound	0.020	1.000	0.020	3.305	0.074	0.049
Light x Training x PHQ-15	Spherity Assumed	0.011	2	0.005	0.883	0.418	0.027
	Greenhouse-Geisser	0.011	2.000	0.005	0.883	0.418	0.027
	Huynh-Feldt	0.011	2.000	0.005	0.883	0.418	0.027
	Lower-bound	0.011	2.000	0.005	0.883	0.418	0.027
Error (light)	Spherity Assumed	0.396	64	0.006			
	Greenhouse-	0.396	64.000	0.006			

	Geisser Huynh-Feldt Lower-bound	0.396	64.000	0.006			

Table 4: Tests of Between-Subjects effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	37.614	1	37.614	432.52	.000	.871
Training group	.340	2	.170	1.95	.150	.058
PHQ group	.090	1	.090	1.032	.313	.016
Training group x PHQ group	.114	2	.057	.658	.522	.020
Error	5.566	64	.087			

Table 5: Summary table for 2 x 2 x 3 mixed model ANOVA with one within-subjects factor (light: absent vs. present) and two between-subjects factors (PHQ-15 group: high vs. low and training condition: no training vs. low training vs. high training), taking false alarms as a dependent variable.

Source		Type III Sum of Squares	df	Mean Square	F	Sig	Partial Eta Squared
Light	Spherity Assumed	0.093	1	0.093	11.458	0.001	0.152
	Greenhouse-Geisser	0.093	1.000	0.093	11.458	0.001	0.152
	Huynh-Feldt	0.093	1.000	0.093	11.458	0.001	0.152
	Lower-bound	0.093	1.000	0.093	11.458	0.001	0.152
Light x Training	Spherity Assumed	0.020	2	0.010	1.226	0.300	0.037
	Greenhouse-Geisser	0.020	2.000	0.010	1.226	0.300	0.037
	Huynh-Feldt	0.020	2.000	0.010	1.226	0.300	0.037
	Lower-bound	0.020	2.000	0.010	1.226	0.300	0.037
Light x PHQ-15	Spherity Assumed	0.000	1	0.000	0.047	0.829	0.001
	Greenhouse-Geisser	0.000	1.000	0.000	0.047	0.829	0.001
	Huynh-Feldt	0.000	1.000	0.000	0.047	0.829	0.001
	Lower-bound	0.000	1.000	0.000	0.047	0.829	0.001
Light x Training x PHQ-15	Spherity Assumed	0.011	2	0.006	0.691	0.505	0.021
	Greenhouse-Geisser	0.011	2.000	0.006	0.691	0.505	0.021
	Huynh-Feldt	0.011	2.000	0.006	0.691	0.505	0.021
	Lower-bound	0.011	2.000	0.006	0.691	0.505	0.021
Error	Spherity	0.519	64	0.008			

(light)	Assumed Greenhouse- Geisser Huynh-Feldt Lower-bound	0.519 0.519 0.519	64.000 64.000 64.000	0.008 0.008 0.008			

Table 6: Tests of Between-Subjects effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	16.870	1	16.870	411.103	.000	.865
Training group	.100	2	.050	1.217	.303	.037
PHQ group	.094	1	.094	2.300	.134	.035
Training group x PHQ group	.116	2	.058	1.416	.250	.042
Error	2.626	64	.041			

Table 7: Summary table for 2 x 2 x 3 mixed model ANOVA with one within-subjects factor (light: absent vs. present) and two between-subjects factors (PHQ-15 group: high vs. low and training condition: no training vs. low training vs. high training), taking d' as a dependent variable.

Source		Type III Sum of Squares	df	Mean Square	F	Sig	Partial Eta Squared
Light	Spherity Assumed	1.344	1	1.344	10.872	0.002	0.145
	Greenhouse-Geisser	1.344	1.000	1.344	10.872	0.002	0.145
	Huynh-Feldt	1.344	1.000	1.344	10.872	0.002	0.145
	Lower-bound	1.344	1.000	1.344	10.872	0.002	0.145
Light x Training	Spherity Assumed	0.227	2	0.113	0.916	0.405	0.028
	Greenhouse-Geisser	0.227	2.000	0.113	0.916	0.405	0.028
	Huynh-Feldt	0.227	2.000	0.113	0.916	0.405	0.028
	Lower-bound	0.227	2.000	0.113	0.916	0.405	0.028
Light x PHQ-15	Spherity Assumed	0.046	1	0.046	0.375	0.543	0.006
	Greenhouse-Geisser	0.046	1.000	0.046	0.375	0.543	0.006
	Huynh-Feldt	0.046	1.000	0.046	0.375	0.543	0.006
	Lower-bound	0.046	1.000	0.046	0.375	0.543	0.006
Light x Training x PHQ-15	Spherity Assumed	0.149	2	0.074	0.601	0.551	0.018
	Greenhouse-Geisser	0.149	2.000	0.074	0.601	0.551	0.018
	Huynh-Feldt	0.149	2.000	0.074	0.601	0.551	0.018
	Lower-bound	0.149	2.000	0.074	0.601	0.551	0.018
Error (light)	Spherity Assumed	7.913	64	0.124			
		7.913	64.000	0.124			

	Greenhouse-Geisser Huynh-Feldt Lower-bound	7.913 7.913	64.000 64.000	0.124 0.124			

Table 8: Tests of Between- Subjects effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	226.239	1	226.239	167.101	.000	.723
Training group	7.361	2	3.681	2.718	.074	.078
PHQ group	3.876	1	3.876	2.863	.095	.043
Training group x PHQ group	5.375	2	2.687	1.985	.146	.058
Error	86.650	64	1.354			

Table 9: Summary table for 2 x 2 x 3 mixed model ANOVA with one within-subjects factor (light: absent vs. present) and two between-subjects factors (PHQ-15 group: high vs. low and training condition: no training vs. low training vs. high training), taking c as a dependent variable.

Source		Type III Sum of Squares	df	Mean Square	F	Sig	Partial Eta Squared
Light	Spherity Assumed	2.851	1	2.851	45.756	0.000	0.417
	Greenhouse-Geisser	2.851	1.000	2.851	45.756	0.000	0.417
	Huynh-Feldt	2.851	1.000	2.851	45.756	0.000	0.417
	Lower-bound	2.851	1.000	2.851	45.756	0.000	0.417
Light x Training	Spherity Assumed	0.158	2	0.079	1.266	0.289	0.038
	Greenhouse-Geisser	0.158	2.000	0.079	1.266	0.289	0.038
	Huynh-Feldt	0.158	2.000	0.079	1.266	0.289	0.038
	Lower-bound	0.158	2.000	0.079	1.266	0.289	0.038
Light x PHQ-15	Spherity Assumed	0.083	1	0.083	1.325	0.254	0.020
	Greenhouse-Geisser	0.083	1.000	0.083	1.325	0.254	0.020
	Huynh-Feldt	0.083	1.000	0.083	1.325	0.254	0.020
	Lower-bound	0.083	1.000	0.083	1.325	0.254	0.020
Light x Training x PHQ-15	Spherity Assumed	0.064	2	0.032	0.511	0.602	0.016
	Greenhouse-Geisser	0.064	2.000	0.032	0.511	0.602	0.016
	Huynh-Feldt	0.064	2.000	0.032	0.511	0.602	0.016
	Lower-bound	0.064	2.000	0.032	0.511	0.602	0.016
Error (light)	Spherity Assumed	3.987	64	0.062			
	Greenhouse-	3.987	64.000	0.062			

	Geisser Huynh-Feldt Lower-bound	3.987	64.000	0.062			

Table 10: Tests of Between-Subjects effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	44.615	1	44.615	134.948	.000	.678
Training group	.285	2	.142	.431	.652	.013
PHQ group	.014	1	.014	.042	.839	.001
Training group x PHQ group	.001	2	.001	.002	.998	.000
Error	21.159	64	.331			

Table 11: Summary table for 2 x 2 x 3 mixed model ANCOVA with one within-subjects factor (light: absent vs. present) and two between-subjects factors (PHQ-15 group: high vs. low and training condition: no training vs. low training vs. high training), controlling for HAI, SSAS, STAI-T and PHQ-9, taking hits as a dependent variable.

Source		Type III Sum of Squares	df	Mean Square	F	Sig	Partial Eta Squared
Light	Spherity Assumed	0.517	1	0.517	94.369	0.000	0.611
	Greenhouse-Geisser	0.517	1.000	0.517	94.369	0.000	0.611
	Huynh-Feldt	0.517	1.000	0.517	94.369	0.000	0.611
	Lower-bound	0.517	1.000	0.517	94.369	0.000	0.611
Light x HAI Delaney	Spherity Assumed	0.005	1	0.005	0.843	0.362	0.014
	Greenhouse-Geisser	0.005	1.000	0.005	0.843	0.362	0.014
	Huynh-Feldt	0.005	1.000	0.005	0.843	0.362	0.014
	Lower-bound	0.005	1.000	0.005	0.843	0.362	0.014
Light x PHQ-9 delaney	Spherity Assumed	0.001	1	0.001	0.179	0.673	0.003
	Greenhouse-Geisser	0.001	1.000	0.001	0.179	0.673	0.003
	Huynh-Feldt	0.001	1.000	0.001	0.179	0.673	0.003
	Lower-bound	0.001	1.000	0.001	0.179	0.673	0.003
Light x SSAS Delaney	Spherity Assumed	0.034	1	0.034	6.114	0.016	0.092
	Greenhouse-Geisser	0.034	1.000	0.034	6.114	0.016	0.092
	Huynh-Feldt	0.034	1.000	0.034	6.114	0.016	0.092
	Lower-bound	0.034	1.000	0.034	6.114	0.016	0.092
Light x STAI-T	Spherity Assumed	0.001	1	0.001	0.200	0.656	0.003
		0.001	1.000	0.001	0.200	0.656	0.003

Delaney	Greenhouse-Geisser Huynh-Feldt Lower-bound	0.001 0.001	1.000 1.000	0.001 0.001	0.200 0.200	0.656 0.656	0.003 0.003
Light x Training	Spherity Assumed Greenhouse-Geisser Huynh-Feldt Lower-bound	0.026 0.026 0.026 0.026	2 2.000 2.000 2.000	0.013 0.013 0.013 0.013	2.373 2.373 2.373 2.373	0.102 0.102 0.102 0.102	0.073 0.073 0.073 0.073
Light x PHQ-15 group	Spherity Assumed Greenhouse-Geisser Huynh-Feldt Lower-bound	0.049 0.049 0.049 0.049	1 1.000 1.000 1.000	0.049 0.049 0.049 0.049	8.854 8.854 8.854 8.854	0.004 0.004 0.004 0.004	0.129 0.129 0.129 0.129
Light x Training x PHQ-15 group	Spherity Assumed Greenhouse-Geisser Huynh-Feldt Lower-bound	0.021 0.021 0.021 0.021	2 2.000 2.000 2.000	0.011 0.011 0.011 0.011	1.928 1.928 1.928 1.928	0.154 0.154 0.154 0.154	0.060 0.060 0.060 0.060
Error (light)	Spherity Assumed Greenhouse-Geisser Huynh-Feldt Lower-bound	0.329 0.329 0.329 0.329	60 60.000 60.000 60.000	0.005 0.005 0.005 0.005			

Table 12: Tests of Between-Subjects effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	37.334	1	37.334	406.741	.000	.871
HAI	.050	1	.050	.544	.464	.009
PHQ-9	.001	1	.001	.014	.907	.000
SSAS	.000	1	.000	.001	.973	.000
STAI-T	.011	1	.011	.120	.730	.002
Training group	.378	2	.189	2.060	.136	.064
PHQ group	.046	1	.046	.498	.483	.008
Training group x PHQ group	.127	2	.064	.694	.503	.023
Error	5.507	60	.092			

Table 13: Summary table for 2 x 2 x 3 mixed model ANCOVA with one within-subjects factor (light: absent vs. present) and two between-subjects factors (PHQ-15 group: high vs. low and training condition: no training vs. low training vs. high training), controlling for HAI, SSAS, STAI-T and PHQ-9, taking false alarms as a dependent variable.

Source		Type III Sum of Squares	df	Mean Square	F	Sig	Partial Eta Squared
Light	Spherity Assumed	0.092	1	0.092	94.369	0.001	0.162
	Greenhouse-Geisser	0.092	1.000	0.092	94.369	0.001	0.162
	Huynh-Feldt	0.092	1.000	0.092	94.369	0.001	0.162
	Lower-bound	0.092	1.000	0.092	94.369	0.001	0.162
Light x HAI Delaney	Spherity Assumed	0.014	1	0.014	0.843	0.196	0.028
	Greenhouse-Geisser	0.014	1.000	0.014	0.843	0.196	0.028
	Huynh-Feldt	0.014	1.000	0.014	0.843	0.196	0.028
	Lower-bound	0.014	1.000	0.014	0.843	0.196	0.028
Light x PHQ-9 delaney	Spherity Assumed	0.018	1	0.018	0.179	0.140	0.036
	Greenhouse-Geisser	0.018	1.000	0.018	0.179	0.140	0.036
	Huynh-Feldt	0.018	1.000	0.018	0.179	0.140	0.036
	Lower-bound	0.018	1.000	0.018	0.179	0.140	0.036
Light x SSAS Delaney	Spherity Assumed	0.000	1	0.000	6.114	0.847	0.001
	Greenhouse-Geisser	0.000	1.000	0.000	6.114	0.847	0.001
	Huynh-Feldt	0.000	1.000	0.000	6.114	0.847	0.001
	Lower-bound	0.000	1.000	0.000	6.114	0.847	0.001
Light x STAI-T Delaney	Spherity Assumed	0.014	1	0.014	0.200	0.185	0.029
	Greenhouse-	0.014	1.000	0.014	0.200	0.185	0.029

	Geisser Huynh-Feldt Lower-bound	0.014	1.000	0.014	0.200	0.185	0.029
Light x Training	Spherity Assumed Greenhouse-Geisser Huynh-Feldt Lower-bound	0.016 0.016 0.016 0.016	2 2.000 2.000 2.000	0.008 0.008 0.008 0.008	2.373 2.373 2.373 2.373	0.364 0.364 0.364 0.364	0.033 0.033 0.033 0.033
Light x PHQ-15 group	Spherity Assumed Greenhouse-Geisser Huynh-Feldt Lower-bound	0.001 0.001 0.001 0.001	1 1.000 1.000 1.000	0.001 0.001 0.001 0.001	8.854 8.854 8.854 8.854	0.736 0.736 0.736 0.736	0.002 0.002 0.002 0.002
Light x Training x PHQ-15 group	Spherity Assumed Greenhouse-Geisser Huynh-Feldt Lower-bound	0.013 0.013 0.013 0.013	2 2.000 2.000 2.000	0.006 0.006 0.006 0.006	1.928 1.928 1.928 1.928	0.449 0.449 0.449 0.449	0.026 0.026 0.026 0.026
Error (light)	Spherity Assumed Greenhouse-Geisser Huynh-Feldt Lower-bound	0.475 0.475 0.475 0.475	60 60.000 60.000 60.000	0.008 0.008 0.008 0.008			

Table 14: Tests of Between-Subjects effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	16.604	1	16.604	387.068	.000	.866
HAI	.003	1	.003	.064	.802	.001
PHQ-9	.039	1	.039	.911	.344	.015
SSAS	.000	1	.000	.007	.935	.000
STAI-T	.016	1	.016	.363	.549	.006
Training group	.114	2	.057	1.331	.272	.042
PHQ group	.018	1	.018	.417	.521	.007
Training group x PHQ group	.114	2	.057	1.330	.272	.042
Error	2.574	60	.043			

Table 15: Summary table for 2 x 2 x 3 mixed model ANCOVA with one within-subjects factor (light: absent vs. present) and two between-subjects factors (PHQ-15 group: high vs. low and training condition: no training vs. low training vs. high training), controlling for HAI, SSAS, STAI-T and PHQ-9, taking d' as a dependent variable.

Source		Type III Sum of Squares	df	Mean Square	F	Sig	Partial Eta Squared
Light	Spherity Assumed	1.210	1	1.210	10.923	0.002	0.146
	Greenhouse-Geisser	1.210	1.000	1.210	10.923	0.002	0.146
	Huynh-Feldt	1.210	1.000	1.210	10.923	0.002	0.146
	Lower-bound	1.210	1.000	1.210	10.923	0.002	0.146
Light x HAI Delaney	Spherity Assumed	0.058	1	0.058	0.491	0.486	0.008
	Greenhouse-Geisser	0.058	1.000	0.058	0.491	0.486	0.008
	Huynh-Feldt	0.058	1.000	0.058	0.491	0.486	0.008
	Lower-bound	0.058	1.000	0.058	0.491	0.486	0.008
Light x PHQ-9 delaney	Spherity Assumed	0.250	1	0.250	2.130	0.150	0.034
	Greenhouse-Geisser	0.250	1.000	0.250	2.130	0.150	0.034
	Huynh-Feldt	0.250	1.000	0.250	2.130	0.150	0.034
	Lower-bound	0.250	1.000	0.250	2.130	0.150	0.034
Light x SSAS Delaney	Spherity Assumed	0.421	1	0.421	3.584	0.063	0.056
	Greenhouse-Geisser	0.421	1.000	0.421	3.584	0.063	0.056
	Huynh-Feldt	0.421	1.000	0.421	3.584	0.063	0.056
	Lower-bound	0.421	1.000	0.421	3.584	0.063	0.056
Light x STAI-T Delaney	Spherity Assumed	0.011	1	0.011	0.096	0.758	0.002
	Greenhouse-	0.011	1.000	0.011	0.096	0.758	0.002

	Geisser Huynh-Feldt Lower-bound	0.011	1.000	0.011	0.096	0.758	0.002
Light x Training	Spherity Assumed Greenhouse- Geisser Huynh-Feldt Lower-bound	0.232 0.232 0.232 0.232	2 2.000 2.000 2.000	0.116 0.116 0.116 0.116	0.987 0.987 0.987 0.987	0.379 0.379 0.379 0.379	0.032 0.032 0.032 0.032
Light x PHQ-15 group	Spherity Assumed Greenhouse- Geisser Huynh-Feldt Lower-bound	0.271 0.271 0.271 0.271	1 1.000 1.000 1.000	0.271 0.271 0.271 0.271	2.304 2.304 2.304 2.304	0.134 0.134 0.134 0.134	0.037 0.037 0.037 0.037
Light x Training x PHQ-15 group	Spherity Assumed Greenhouse- Geisser Huynh-Feldt Lower-bound	0.086 0.086 0.086 0.086	2 2.000 2.000 2.000	0.043 0.043 0.043 0.043	0.366 0.366 0.366 0.366	0.695 0.695 0.695 0.695	0.012 0.012 0.012 0.012
Error (light)	Spherity Assumed Greenhouse- Geisser Huynh-Feldt Lower-bound	7.054 7.054 7.054 7.054	60 60.000 60.000 60.000	0.118 0.118 0.118 0.118			

Table 16: Tests of Between-Subjects effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	226.685	1	226.685	160.908	.000	.728
HAI	1.067	1	1.067	.758	.388	.012
PHQ-9	.705	1	.705	.500	.482	.008
SSAS	.055	1	.055	.039	.845	.001
STAI-T	.901	1	.901	.640	.427	.011
Training group	8.555	2	4.278	3.036	.055	.092
PHQ group	1.018	1	1.018	.723	.399	.012
Training group x PHQ group	5.566	2	2.783	1.976	.148	.062
Error	84.527	60	1.409			

Table 17: Summary table for 2 x 2 x 3 mixed model ANCOVA with one within-subjects factor (light: absent vs. present) and two between-subjects factors (PHQ-15 group: high vs. low and training condition: no training vs. low training vs. high training), controlling for HAI, SSAS, STAI-T and PHQ-9, taking c as a dependent variable.

Source		Type III Sum of Squares	df	Mean Square	F	Sig	Partial Eta Squared
Light	Spherity Assumed	2.754	1	2.754	47.541	0.000	0.442
	Greenhouse-Geisser	2.754	1.000	2.754	47.541	0.000	0.442
	Huynh-Feldt	2.754	1.000	2.754	47.541	0.000	0.442
	Lower-bound	2.754	1.000	2.754	47.541	0.000	0.442
Light x HAI Delaney	Spherity Assumed	0.065	1	0.065	1.129	0.292	0.018
	Greenhouse-Geisser	0.065	1.000	0.065	1.129	0.292	0.018
	Huynh-Feldt	0.065	1.000	0.065	1.129	0.292	0.018
	Lower-bound	0.065	1.000	0.065	1.129	0.292	0.018
Light x PHQ-9 delaney	Spherity Assumed	0.082	1	0.082	1.409	0.240	0.023
	Greenhouse-Geisser	0.082	1.000	0.082	1.409	0.240	0.023
	Huynh-Feldt	0.082	1.000	0.082	1.409	0.240	0.023
	Lower-bound	0.082	1.000	0.082	1.409	0.240	0.023
Light x SSAS Delaney	Spherity Assumed	0.168	1	0.168	2.903	0.094	0.046
	Greenhouse-Geisser	0.168	1.000	0.168	2.903	0.094	0.046
	Huynh-Feldt	0.168	1.000	0.168	2.903	0.094	0.046
	Lower-bound	0.168	1.000	0.168	2.903	0.094	0.046
Light x STAI-T Delaney	Spherity Assumed	0.149	1	0.149	2.565	0.115	0.041
	Greenhouse-	0.149	1.000	0.149	2.565	0.115	0.041

	Geisser Huynh-Feldt Lower-bound	0.149	1.000	0.149	2.565	0.115	0.041
Light x Training	Spherity Assumed Greenhouse- Geisser Huynh-Feldt Lower-bound	0.156 0.156 0.156 0.156	2 2.000 2.000 2.000	0.078 0.078 0.078 0.078	1.348 1.348 1.348 1.348	0.268 0.268 0.268 0.268	0.043 0.043 0.043 0.043
Light x PHQ-15 group	Spherity Assumed Greenhouse- Geisser Huynh-Feldt Lower-bound	0.166 0.166 0.166 0.166	1 1.000 1.000 1.000	0.166 0.166 0.166 0.166	2.860 2.860 2.860 2.860	0.096 0.096 0.096 0.096	0.045 0.045 0.045 0.045
Light x Training x PHQ-15 group	Spherity Assumed Greenhouse- Geisser Huynh-Feldt Lower-bound	0.129 0.129 0.129 0.129	2 2.000 2.000 2.000	0.064 0.064 0.064 0.064	1.111 1.111 1.111 1.111	0.336 0.336 0.336 0.336	0.036 0.036 0.036 0.036
Error (light)	Spherity Assumed Greenhouse- Geisser Huynh-Feldt Lower-bound	3.475 3.475 3.475 3.475	60 60.000 60.000 60.000	0.058 0.058 0.058 0.058			

Table 18: Tests of Between-Subjects effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercep	44.731	1	44.731	128.704	.000	.682
HAI	.061	1	.061	.176	.676	.003
PHQ-9	.215	1	.215	.618	.435	.010
SSAS	.024	1	.024	.069	.794	.001
STAI-T	.003	1	.003	.008	.928	.000
Training group	.356	2	.178	.511	.602	.017
PHQ group	.019	1	.019	.054	.817	.001
Training group x PHQ group	.001	2	.001	.002	.998	.000
Error	20.853	60	.348			

APPENDIX III: PATIENT HEALTH QUESTIONNAIRE - 15

During the <u>past 4 weeks</u>, how much have you been bothered by any of the following problems?

	Not bothered at all **(0)**	Bothered a little **(1)**	Bothered a lot **(2)**
a. Stomach pain …….……..	☐	☐	☐
b. Back pain ….……..……..	☐	☐	☐
c. Pain in your arms, legs, or joints (knees, hips, etc.) ..	☐	☐	☐
d. Menstrual cramps or other problems with your periods [***Women only***] …….	☐	☐	☐
e. Headaches …….……..	☐	☐	☐
f. Chest pain …….……	☐	☐	☐
g. Dizziness ……	☐	☐	☐
h. Fainting spells …..…….	☐	☐	☐
i. Feeling your heart pound or race ……..	☐	☐	☐
j. Shortness of breath ……..……	☐	☐	☐
k. Pain or problems during sexual intercourse……	☐	☐	☐
l. Constipation, loose bowels, or diarrhea ……	☐	☐	☐
m. Nausea, gas, or indigestion ……	☐	☐	☐
n. Feeling tired or having low energy …….	☐	☐	☐
o. Trouble sleeping ……..……..	☐	☐	☐

APPENDIX IV: PATIENT HEALTH QUESTIONNAIRE – 9

Over the last <u>2 weeks</u>, how often have you been bothered by any of the following problems?

(Use ✓ to indicate your answer)

	Not at all	Several days	More than half the days	Nearly every day
1) Little interest or pleasure in doing things	0	1	2	3
2) Feeling down, depressed, or hopeless	0	1	2	3
3) Trouble falling or staying asleep, or sleeping too much	0	1	2	3
4) Feeling tired or having little energy	0	1	2	3
5) Poor appetite or overeating	0	1	2	3
6) Feeling bad about yourself – or that you are a failure or have let yourself or family down	0	1	2	3
7) Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8) Moving or speaking so slowly that other people could have noticed? Or the opposite - being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3
9) Thoughts that you would be better off dead or of hurting yourself in some way	0	1	2	3

If you checked off any problems, how difficult have these problems made it for you to do your work, take care of things at home, or get along with other people?

Not difficult at all □ Somewhat difficult □ Very difficult □ Extremely difficult □

APPENDIX V: PATIENT HEALTH QUESTIONNAIRE GENERAL ANXIETY DISORDER – 7

Over the last <u>2 weeks</u>, how often have you been bothered by any of the following problems?

(Use ✓ to indicate your answer)

	Not at all	**Several days**	**More than half the days**	**Nearly every day**
1) Feeling nervous, anxious or on edge	0	1	2	3
2) Not being able to stop or control worrying	0	1	2	3
3) Worrying too much about different things	0	1	2	3
4) Trouble relaxing	0	1	2	3
5) Being so restless that it is hard to sit still	0	1	2	3
6) Becoming easily annoyed or irritable	0	1	2	3
7) Feeling afraid as if something awful might happen	0	1	2	3

APPENDIX VI: STATE TRAIT ANXIETY INVENTORY-TRAIT VERSION

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you ***generally*** feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you ***generally*** feel.

1 = not at all 2 = somewhat 3 = moderately so 4 = very much so

21. I feel calm	1	2	3	4
22. I feel secure	1	2	3	4
23. I am tense	1	2	3	4
24. I feel strained	1	2	3	4
25. I feel at ease	1	2	3	4
26. I feel upset	1	2	3	4
27. I am presently worrying over possible misfortunes	1	2	3	4
28. I feel satisfied	1	2	3	4
29. I feel frightened	1	2	3	4
30. I feel comfortable	1	2	3	4
31. I feel self-confident	1	2	3	4
32. I feel nervous	1	2	3	4
33. I am jittery	1	2	3	4
34. I feel indecisive	1	2	3	4
35. I am relaxed	1	2	3	4
36. I feel content	1	2	3	4
37. I am worried	1	2	3	4
38. I feel confused	1	2	3	4
39. I feel steady	1	2	3	4
40. I feel pleasant	1	2	3	4

APPENDIX VII: HEALTH ANXIETY INVENTORY

Each question in this section consists of a group of four statements. Please read each group of statements carefully and then select the one which best describes your feelings, over the past six months. Identify the statement by ringing the letter next to it, i.e. if you think that statement (a) is correct, ring statement (a); it may be that more than one statement applies, in which case, please ring any that are applicable.

1. (a) I do not worry about my health.
   (b) I occasionally worry about my health.
   (c) I spend much of my time worrying about my health.
   (d) I spend most of my time worrying about my health.

2. (a) I notice aches/pains less than most other people (of my age).
   (b) I notice aches/pains as much as most other people (of my age).
   (c) I notice aches/pains more than most other people (of my age).
   (d) I am aware of aches/pains in my body all the time.

3. (a) As a rule I am not aware of bodily sensations or changes.
   (b) Sometimes I am aware of bodily sensations or changes.
   (c) I am often aware of bodily sensations or changes.
   (d) I am constantly aware of bodily sensations or changes.

4. (a) Resisting thoughts of illness is never a problem.
   (b) Most of the time I can resist thoughts of illness.
   (c) I try to resist thoughts of illness but am often unable to do so.
   (d) Thoughts of illness are so high that I no longer even try to resist them.

5. (a) As a rule I am not afraid that I have a serious illness.
   (b) I am sometimes afraid that I have a serious illness.
   (c) I am often afraid that I have a serious illness.
   (d) I am always afraid that I have a serious illness.

6. (a) I do not have images (mental pictures) of myself being ill.
   (b) I occasionally have images of myself being ill.
   (c) I frequently have images of myself being ill.
   (d) I constantly have images of myself being ill.

7. (a) I do not have any difficulty taking my mind off thoughts about my health.
   (b) I sometimes have difficulty taking my mind off thoughts about my health.
   (c) I often have difficulty taking my mind off thoughts about my health.
   (d) Nothing can take my mind off thoughts about my health.

8. (a) I am lastingly relieved if my doctor tells me nothing is wrong.
   (b) I am initially relieved but the worries sometimes return later.
   (c) I am initially relieved but the worries always return later.
   (d) I am not relieved if my doctor tells me nothing is wrong.

9. (a) If I hear about an illness I never think I have it myself.
   (b) If I hear about an illness I sometimes think I have it myself.
   (c) If I hear about an illness I often think I have it myself.
   (d) If I hear about an illness I always think I have it myself.

10. (a) If I have a bodily sensation or change I rarely wonder what it means.
    (b) If I have a bodily sensation or change I often wonder what it means.
    (c) If I have a bodily sensation or change I always wonder what it means.
    (d) If I have a bodily sensation or change I must know what it means.

11. (a) I usually feel at very low risk for developing a serious illness.
    (b) I usually feel at fairly low risk for developing a serious illness.
    (c) I usually feel at moderate risk for developing a serious illness.
    (d) I usually feel at high risk for developing a serious illness.

12. (a) I never think I have a serious illness.
    (b) I sometimes think I have a serious illness.
    (c) I often think I have a serious illness.
    (d) I usually think I am seriously ill.

13. (a) If I notice an unexplained bodily sensation I don't find it difficult to think about other things.
    (b) If I notice an unexplained bodily sensation I sometimes find it difficult to think about other things.

(c) If I notice an unexplained bodily sensation I often find it difficult to think about other things.

(d) If I notice an unexplained bodily sensation I always find it difficult to think about other things.

14. (a) My family/friends would say I do not worry enough about my health.

    (b) My family/friends would say I have a normal attitude to my health.

    (c) My family/friends would say I worry too much about my health.

    (d) My family/friends would say I am a hypochondriac.

For the following questions, please think about what it would be like if you had a serious illness of a type which particularly concerns you (such as heart disease, cancer, multiple sclerosis and so on). Obviously you cannot know for definite what it would be like; please give your best estimate of what you <u>think</u> might happen, basing your estimate on what you know about yourself and serious illness in general.

15. (a) If I had a serious illness I would still be able to enjoy things in my life quite a lot.

    (b) If I had a serious illness I would still be able to enjoy things in my life a little.

    (c) If I had a serious illness I would still be almost completely unable to enjoy things in my life.

    (d) If I had a serious illness I would be completely unable to enjoy my life at all.

16. (a) If I developed a serious illness there is a good chance that modern medicine would be able to cure me.

    (b) If I developed a serious illness there is a moderate chance that modern medicine would be able to cure me.

    (c) If I developed a serious illness there is a very small chance that modern medicine would be able to cure me.

    (d) If I developed a serious illness there is no chance that modern medicine would be able to cure me.

17. (a) A serious illness would ruin some aspects of my life.

    (b) A serious illness would ruin many aspects of my life.

(c) A serious illness would ruin almost every aspect of my life.

(d) A serious illness would ruin every aspect of my life.

18. (a) If I had a serious illness I would not feel that I had lost my dignity.

(b) If I had a serious illness I would feel that I had lost a little of my dignity.

(c) If I had a serious illness I would not feel that I had lost quite a lot of my dignity.

(d) If I had a serious illness I would not feel that I had totally lost my dignity.

APPENDIX VIII: SOMATOSENSORY AMPLIFICATION SCALE

Please indicate the degree to which each of these statements are characteristic of you in general. Circle your answer.

1 = Not at all true 2 = A little bit more 3 = Moderately true 4 = Quite a bit true
5 = Extremely true

	Not at all true	**A little bit true**	**Moderately true**	**Quite a bit true**	**Extremely true**
1) I can't stand smoke, smog, or pollutants in the air.	1	2	3	4	5
2) I am often aware of various things happening within my body.	1	2	3	4	5
3) When I bruise myself, it stays noticeable for a long time.	1	2	3	4	5
4) I sometimes can feel the blood flowing in my body.	1	2	3	4	5
5) Sudden loud noises really bother me.	1	2	3	4	5
6) I can sometimes hear my pulse throbbing in my ear.	1	2	3	4	5
7) I hate to be too hot or too cold.	1	2	3	4	5
8) I am quick to sense the hunger contractions in my stomach.	1	2	3	4	5
9) Even something minor, like an insect bite or splinter, really bothers me.	1	2	3	4	5
10) I can't stand pain.	1	2	3	4	5

APPENDIX IX: ETHICAL APPROVAL

The University of Manchester School of Psychological Sciences Research Ethics Committee

15th February 2010

Ref:	616/07P
Title:	Effect of Association Strength on illusory touch in medically unexplained symptoms.
Type:	PG research
Level:	Level 2
Research Group:	SSDT
Participants:	96
Methodology:	questionnaire and testing
Supervisor:	Richard Brown
Author1:	Laura Rowlands
Author2:	Katharyn Hall
Author3:	Ellen Poliakoff
Author4:	Donna Lloyd
Comments:	1. No changes necessary.
Decision:	**Approved**

APPENDIX X: RECRUITMENT POSTER

Project No: 616/07P
Codename:

MANCHESTER 1824

The University of Manchester

VOLUNTEERS NEEDED FOR A STUDY ON TOUCH DETECTION AND PHYSICAL SYMPTOMS

We are looking for people to take part in a study investigating the relationship between touch perception and the tendency to experience physical symptoms.

There are two parts to this study. The first part involves completing some online questionnaires about your experience of different symptoms, which should take no more than 20 minutes. Individuals who complete the first part of this study will be entered into a prize draw for a chance to win £50. Psychology undergraduates will also receive 1 academic credit.

Eligible individuals will then be invited to participate in the second part of the study. Having completed the first part of the study, you are under no obligation to complete the second part if you do not want to. The second part involves completing a simple touch perception task and some more questionnaires about various symptoms. Participants will receive £5 or 4 course credits (for psychology students) for taking part. The second part takes between 1 hour and 1hr 30 mins.

Interested in taking part?

Contact either Laura Rowlands Laura.rowlands@postgrad.manchester.ac.uk or Kate Hall Katharyn.hall@postgrad.manchester.ac.uk

This study is being carried out by Laura Rowlands and Katharyn Hall, as part of their ClinPsyD research, under the supervision of Dr Richard Brown, Dr Donna Lloyd and Dr Elllen Polliakoff from the School of Psychological Sciences.

THIS PROJECT HAS BEEN APPROVED BY THE SCHOOL OF PSYCHOLOGICAL SCIENCES RESEARCH ETHICS COMMITTEE

APPENDIX XI: RECRUITMENT EMAIL

Header: Earn £5 or 4 Credits Bodily symptoms and touch detection study

Message: We are looking people to take part in a touch detection study (Ethics Code: [to be inserted]). There are two stages to this study. The first stage involves completing an online questionnaire, which will take approximately 15 minutes. For completing the first part of this study you will be entered into a prize draw for a chance to win £50. Psychology undergraduates will also receive 1 course credit.

Eligible individuals will then be invited to take part in the second part of the study. This involves attending an appointment in the Department of Psychology, which will last approximately 1 hour. You will be asked to complete a touch detection task and some questionnaires about bodily symptoms and mood. Participants will receive £5 or 4 course credits for taking part.

If you would like to take part or would like more information, please email either

Laura Rowlands laura.rowlands@postgrad.manchester.ac.uk or
Kate Hall katharyn.hall@postgrad.manchester.ac.uk

THIS PROJECT HAS BEEN APPROVED BY THE SCHOOL OF PSYCHOLOGICAL SCIENCES RESEARCH ETHICS COMMITTEE

Thanks,
Laura

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Documents: None