The role of somatic amplification in physical symptom reporting

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THESIS ABSTRACT

Despite their apparent ubiquity across healthcare settings medically unexplained symptoms (MUS) remain poorly understood. Theoretical models have proposed several different ways in which altered sensory and perceptual processes may contribute to the development and/or maintenance of MUS. The narrative review presented in Chapter 1 explores sensory and perceptual processes relevant to MUS and physical symptom reporting in general and considers whether current empirical findings lend support to a particular model or hypothesis. One conclusion of the review is that there is a paucity of research using objective methods to measure the putative role of somatic amplification during the perception of physical symptoms, and the influence of negative affect on this process. To address this shortfall, Chapter 2 presents an original study that attempts to test the effect of negative affect on somatic amplification, using a novel paradigm derived from signal detection theory (SDT), the somatic signal discrimination task (SSDiT). On the SSDiT, subjects are required to discriminate between a series of "weak" and "strong" vibrations. Nonclinical "high" and "low" symptom reporters completed this task prior to and following either a neutral or negative mood induction. Contrary to expectation, there was limited support for differences between symptom reporting groups on the SSDiT task at baseline. However, there was some suggestion that highs and lows may drift differentially on this task over time, such that lows showed a tendency to become more conservative than highs over time. Potential implications for theoretical models are discussed.

Finally, Chapter 3 presents a critical evaluation in which the literature review and empirical study are placed within a wider context, allowing implications of the research process as a whole to be drawn out further. This chapter also identifies potential directions for future research, discusses limitations of the present thesis and offers personal reflections about the research process as a whole.

This thesis follows the paper-based format, such that Chapters 1 and 2 are formatted as stand-alone papers considered suitable for publication, prepared in accordance with submission requirements for Clinical Psychology Review (Chapter 1) and Journal of Abnormal Psychology respectively (Chapter 2; see Appendix 1 for submission guidelines).

DECLARATION

I hereby declare that this thesis is my own original work and no portion of the work referred to

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Chapter 1: Literature review

Sensory and perceptual processes in functional somatization: A narrative review of the literature.

Abstract

A number of conceptual models have identified alterations in sensory and perceptual processing as central to the development and maintenance of functional somatization (i.e., medically unexplained symptoms; MUS). At present it is unclear which, if any, of these models is most consistent with the available empirical evidence. Accordingly, the present review aims to bring together relevant literature from different fields to evaluate the evidence for altered sensory and perceptual processes in patients with functional somatization and to establish if evidence supports a particular theoretical approach. Although a large body of literature has focused on searching for physiological or biological markers, the majority of such studies have neglected the role of psychological factors during measurement of perceptual processes. Key psychological factors that need to be considered include negative affect, expectancy/anticipation effects, response bias, and attention. In general, current models do not adequately integrate psychological and physiological factors, nor provide sufficient detail about how cognitive-perceptual factors are altered in MUS, making it difficult to identify specific experimental predictions which would enable theories to be differentiated.

KEYWORDS: perception; medically unexplained symptoms; functional symptoms; somatization; somatosensory amplification

Introduction

Patients frequently present with physical symptoms for which no medical explanation can be found (Gureje, Simon, Ustun, & Goldberg, 1997; Kroenke, 2003; Nimnuan, Hotopf, & Wessely, 2001). In psychiatric settings, these medically unexplained symptoms (MUS) are commonly diagnosed as somatoform disorders (DSM-IV, APA, 1994), while across general medical settings, the same cluster of symptoms may be labeled as a functional somatic syndrome (see Table 1 for common diagnostic labels). The noticeable overlap of symptoms challenges the assumption that these labels represent distinct disorders (Whitehead, Palsson, &

Jones, 2002) and patients often meet criteria for both (Aaron & Buchwald, 2001), indicating that the dichotomy between medical and psychiatric explanations may be redundant.

Table 1 Diagnostic labels for common functional conditions used across medical specialties (adapted from Brown, 2007)

Specialty	Diagnostic labels	Functional symptoms
Allergy/toxicology	Multiple chemical sensitivity/ idiopathic environmental intolerance Chemical intolerance	Odor hypersensitivity to common chemical agents Headache
Cardiology	Non-cardiac chest pain/ atypical chest pain	Persistent chest pain often exacerbated by ingestion or exercise; heartburn; muscle and joint aches
Dentistry	Temporomandibular joint disorder	Pain, clicking, grating in the jaw joint; headache; restricted movement of the jaw
Gastroenterology	Irritable bowel syndrome Non-ulcer dyspepsia Interstitial cystitis	Abdominal bloating and pain lessened by defecation; constipation and /or diarrhoea; change in frequency and/ or consistency of stools
Gynaecology	Chronic pelvic pain Interstitial cystitis/ painful bladder syndrome	Pain during sex; abdominal and pelvic pain; dysmenorrhoea; detrusor instability; change in bladder capacity, frequency and urgency
Infectious disease	Chronic fatigue syndrome/ myalgic encephalomyelitis/ post-viral fatigue syndrome	Widespread muscle and joint pain; persistent fatigue; sleep disturbance; mental exhaustion; headaches
Military medicine	Gulf war syndrome	Fatigue, headaches, muscle pains, neurological symptoms, poor concentration
Neurology	Conversion disorder (formerly known as hysteria) Non-epileptic Attack Disorder	Loss or alteration of motor or sensory function; motor weakness and tremor, paralysis, impaired vision or hearing Seizures
Otolaryngology	Functional dysphonia	Hoarseness
Psychiatry	Somatization Disorder/ Somatoform Disorder (e.g., conversion disorder, pain disorder, undifferentiated somatoform disorder, or somatoform disorder not otherwise specified)	Depends on classification
Rheumatology	Fibromyalgia	Chronic widespread pain and tenderness; sleep disturbance

While initially conceived in relation to physical symptoms resulting specifically from psychological distress (Lipowski, 1988), the term *somatization* has become synonymous with MUS and is commonly used to refer to physical symptoms without a discernable medical cause. This use of such a broad definition of somatization, coupled with the existence of separate, yet

overlapping, medical and psychiatric taxonomies, has made it difficult to know to whom empirical findings in the field are relevant (Brown, 2007; Kirmayer & Robbins, 1991). In describing three forms of somatization, Kirmayer and Robbins (1991) have usefully differentiated "Functional Somatization" (physical symptoms for which there is no discernible medical or psychiatric cause) from the somatic presentation of a psychiatric disorder such as anxiety or depression (i.e., "Presenting Somatization": Bridges & Goldberg, 1985), and the catastrophic worry about, and misattribution of, normal bodily sensations characteristic of "Hypochondriacal Somatization". In line with emerging opinion in the field, the terms MUS and functional somatization are used here interchangeably to refer to the somatoform disorders and the functional somatic syndromes.

Although significant adverse consequences are associated with MUS, such as emotional distress, functional disability and high health care utilization (Gureje & Simon, 1999; Henningsen, Zimmerman, & Sattell, 2003), their development and maintenance remain poorly understood. One reason for the lack of progress in this area may be the conflation of different forms of somatization within research: despite the useful distinction offered by Kirmayer and Robbins (1991), few empirical studies identify the form of somatization under discussion. Lack of clarification and poor control of potential confounds makes it difficult to know whether empirical findings pertain to one or more of these forms, or even to the presence of other diagnosed medical conditions (Brown 2007).

While it is known that psychological factors, such as trait negative affectivity (NA: Costa & McCrae, 1987) are associated with inflated physical symptom reports (Watson & Pennebaker, 1989; Simon, Gater, Kisely, & Piccinelli, 1996), it is not clear how such factors exert their effects. The idea that sensory or perceptual processes are altered in some way is implicated across theories of MUS; however, investigation of perceptual phenomena has been relatively neglected compared to the study of cognitive-attributional, affective, and attentional components (Rief & Broadbent, 2007). Moreover, it is unclear whether the available evidence pertaining to sensory and perceptual processing in functional somatization is consistent with one or more of these theories. To address this shortfall, the present review draws together literature from various sources to examine whether sensory and/or perceptual processes are altered in

people with MUS and the implications of this for current theorizing concerning these conditions.

A number of perceptual theories of MUS have been described in the literature and a detailed review of these is beyond the scope of this paper. Instead, we begin with a brief overview of the main models that emphasize sensory and perceptual processes; for more comprehensive descriptions the reader is referred to an existing theoretical review (Rief & Broadbent, 2007). Parameters for the literature search are then described and empirical findings pertaining to perception in MUS are summarized and evaluated. Distinct sensory and perceptual features that are considered for altered function include: the threshold at which signals from the body are detected; the strength of sensory signals from the body; the level of sensory "noise"; and response criterion (i.e., the general tendency to report sensory signals as present regardless of whether they are present or absent). Within each of these categories, further controversy exists about the mechanisms involved and possible explanations will be explored. Empirical data from the following sources is considered: investigations of sensory thresholds; central nervous system dysfunction (i.e., evidence for 'sensitization' and /or 'habituation'); sham manipulations and expectancy effects; imaging studies on neural correlates; autonomic nervous system (ANS) and endocrine dysfunction; and manipulations within analogue samples relevant to symptom reporting in general.

Current models of MUS

Somatosensory amplification

One concept that has received widespread attention in relation to somatization is that of *somatosensory amplification* – defined as "the tendency to experience somatic and visceral sensation as intense, noxious, and disturbing" (Barsky, 1992, p. 28). Although originally conceived in relation to hypochondriacal somatization, it is thought that amplification occurs in symptom perception more generally (Barsky, 1992; Barsky & Borus, 1999) and may involve both state and trait processes (Barsky, Goodson, Lane, & Cleary, 1988). Research has attempted to measure somatic amplification primarily using the self-report Somatosensory Amplification Scale (SSAS; Barsky, Wyshak, & Klerman, 1990), which asks respondents to rate on a five-point scale the extent that they are bothered by ten common bodily sensations (for example, "I

hate to be too hot or too cold" and "I can sometimes hear my pulse or my heartbeat throbbing in my ear"). However, reliance on subjective assessment tools has made it difficult to determine the nature of the relationship between negative affect and somatic amplification: the SSAS shows strong relationships with other subjective measures of negative affect (Wise & Mann, 1994) and findings from attempts to objectively capture somatic amplification (e.g., via heartbeat detection), indicate the SSAS is simply another measure of psychological distress (Aronson, Barrett, & Quigley, 2001). Furthermore, the SSAS confounds three distinct processes thought to underpin somatic amplification, creating difficulty in understanding the specific role that each process may play in symptom experience. The three mechanisms thought to amplify *state* perception of somatic sensations are: heightened attention to the body (i.e., bodily hypervigilance) that increases the detection of sensations; selective focus on detected sensations; and the attribution of sensations to noxious rather than benign causes. This model also proposes that enhanced detection due to hypervigilance for, and subsequent misattribution of, benign bodily sensations amplifies the future perception of physical signals, eventually leading to more enduring (i.e., trait) perceptual differences (Barsky & Wyshak, 1990). The concept of somatosensory amplification has been widely influential and offers more than one route through which higher-order cognitive and attentional processes can influence perceptual ones. Most theories of MUS now embrace the idea that persons with MUS have amplified perceptual experiences, albeit with different emphases placed on the relative contribution of attentional, affective, and cognitive-attributional factors.

Biological sensitization and habituation

It has been suggested that amplification in MUS may reflect physiological alterations in the strength of sensory signals, such as those related to pain (Eriksen & Ursin, 2002; Ursin, 1997). Ursin and Eriksen (2006) identify two potential mechanisms, largely studied in relation to chronic pain, by which sensory signals may be altered: i) sensitization, referring to increased neuronal reactivity (i.e., "wind-up") to sensory input over time resulting in abnormal sensory sensitivity and, ii) a failure to habituate to stimuli, whereby repeated exposure to a stimulus fails to lead to a diminished response habituation. These phenomena are thought to be associated with different neural processes (Kleinbohl, Trojan, Konrad, & Holzl, 2006). Although this

perspective acknowledges that psychological factors may be important (largely in relation to behavioral, cognitive, and effective responses to changes in sensitization or habituation), they essentially explain perceptual amplification purely on the basis of altered sensory input and/or basic neuronal changes.

Sensitization is hypothesized to occur both peripherally and centrally. Peripheral sensitization results in reduced sensory thresholds (i.e., the lowest stimulus intensity at which pain or discomfort is reported) and enhanced responsiveness of the sensory neurons (i.e., increased pain sensitivity to noxious stimuli, or hyperalgesia) that transmit information from the periphery and viscera to the central nervous system (CNS). Central sensitization occurs following abnormal neuronal excitability in the CNS, which causes increased responsiveness to inert stimuli (observed as sensitivity at the periphery), termed allodynia. Such an account may be of greater relevance to the maintenance of MUS, rather than to their initial development (Rief & Broadbent, 2007). Although such an account has been linked to other symptoms such as fatigue in chronic fatigue syndrome (CFS), it is unclear how this explanation might apply to more unusual functional somatic complaints, such as pseudoneurological symptoms.

Cognitive sensitization

Based on proposed similarities between cognitive bias in anxiety and neuronal sensitization, Brosschot (2002) has attempted to describe cognitive bias as a higher-order *cognitive* sensitization process relevant to the experience of MUS. He argues, however, that a fundamental difference lies in the sensitization of associative neuronal networks (such as cognitive semantic ones), rather than individual neuronal sensitization to painful stimuli. Brosschot (2002) proposes that increased activation in particular illness-related cognitive networks (e.g., those associated with previous illness experiences or illness representations) reduces the activation threshold for that network, making the representations encoded therein more robust and easily activated. In turn, this results in a pre-conscious bias towards processing threat-related information and attribution of ambiguous information to threat-relevant rather than benign causes. Consequently, more illness-relevant cues will be detected, further increasing the strength and number of neuronal traces.

Attention and competition for cues

Perception of the body is generally thought to involve a constructive process that is influenced by information arising from the sense organs and from internal information such as knowledge, beliefs, and expectations (Pennebaker & Hoover, 1984): it is the combination of information from both sources that determines an individual's representation of what they 'think' is happening inside their body (Pennebaker, 1982). Based on the observation that people report more physical symptoms following increased attention to their bodies and in non-stimulating environments (Pennebaker & Watson, 1991), Pennebaker (1982) argued that reduced stimulation from the environment both lowers the threshold for detection of sensory signals (by decreasing the amount of competing information) and increases attention to internal stimuli (increasing available signal), so that previously unnoticed bodily sensations are brought into conscious awareness. Following Pennebaker's (1982) assertion that self-focused attention increases availability of sensory information, Cioffi (1991) argues that self-focused attention also increases the salience of detected sensory signals and may increase perception of symptoms via enhanced attention to interpretations of symptoms (i.e., what the symptom means), rather than to the sensation itself.

Signal-filtering

Following the gate-control theory of pain (Melzack & Wall, 1965), which assumes that peripheral sensory input is normally filtered by the brain so that the most relevant signals enter conscious awareness, the perception-filter model (Rief & Barsky, 2005; Rief & Broadbent, 2007) proposes that MUS arise from disturbances in the perceptual processes responsible for filtering sensory 'signal' from irrelevant 'noise'. By this view, symptoms could result from one of: amplification of sensory signals from the periphery; erroneous selection of irrelevant somatic signals for conscious processing; or altered cortical perception, influenced by factors such as memory and expectation. Several factors that might contribute at each of these levels are identified in the model, but how the various processes actually operate is left unspecified.

Symptom representations

In contrast to models proposing that sensory signals always arise from the body itself, Brown's (2004) model of MUS suggests that they can also arise from the activation of mental

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representations of the body within memory. In this account, MUS arise when representations of previous symptom experiences are erroneously selected by the attentional system instead of those corresponding to "true" sensory signals. Repeated re-activation of these "rogue" representations increases their strength and likelihood of future selection, until they eventually dominate bodily awareness. Thus, in contrast to the suggestion that MUS result from increased sensitivity to sensory signals (e.g., Pennebaker, 1982), Brown's model suggests reduced sensitivity to sensory stimuli in MUS patients, because perception is based on rogue representations rather than what is actually happening in the body. This model offers a useful explanation of how cognitive factors (e.g., memory) may influence pre-conscious perceptual ones.

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Evidence for perceptual alterations in MUS

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The purpose of the present review is to describe and evaluate the available empirical evidence concerning the role of sensory and perceptual processes in MUS and their implications for current theories of these conditions. Fundamental differences in experimental methodologies and techniques used to investigate perceptual processes, and the sheer volume of literature, made direct comparison of findings difficult. Rather than presenting a systematic review, therefore, the current paper provides a narrative account of the literature that provides a broad overview of the main findings from sensory and perceptual research in the main functional somatic syndromes and somatoform disorders, while highlighting important discrepancies and current controversies within each area.

Search Methodology

PsycINFO, PsycARTICLES, and Medline databases (accessed via OvidSP) were searched for published studies investigating perceptual processes in functional somatization. To identify relevant literature, key perceptual terms were cross-referenced with clinical ones. Thus studies were identified by separately linking each of "perceptual," "perception," "habituation," "sensitiz(s)ation," and "amplification" with the following terms (abstracts only): "hysteria," "conversion disorder," "chronic fatigue syndrome," "fibromyalgia," "irritable bowel syndrome," "tempromandibular joint dysfunction," "non-ulcer dyspepsia," "non-cardiac chest pain," "somatoform disorders," "functional somatic syndromes," "somatiz(s)ation" and "medically

unexplained symptoms";[1] this identified 859 articles. Abstracts were inspected by hand and the following exclusion criteria were applied: review articles; treatment studies; book chapters; conference abstracts; non-English articles; non-adult populations; animal studies. Papers relating to body dysmorphic disorder and hypochondriasis, which are distinct from functional somatization but part of the somatoform disorders category, were removed. Reference lists of selected papers were also searched and all remaining abstracts were considered to inform the present review.

Sensitization and habituation

An extensive body of literature has investigated whether or not patients with functional symptoms detect pain, discomfort, and normal sensation at significantly lower thresholds than healthy controls. Sensory thresholds have been measured at both symptom-specific sites (e.g., colon in irritable bowel syndrome, IBS; jaw in temporomandibular joint disorder, TMJD; 'tender' points in fibromyalgia, FM) and non-related sites (e.g., arm; fingertip). In general, researchers have interpreted the presence of reduced thresholds at non-related sites (i.e., allodynia) and at symptom-specific sites as biological evidence of peripheral and central sensitization respectively (e.g., by Mertz, Naliboff, Munakata, Niazi, & Mayer, 1995; Ness, Powell-Boone, Cannon, Lloyd, & Fillingim, 2005). Following Eriksen and Ursin's (2002) sensitization theory, the majority of these threshold-based studies assume that individuals are accurately perceiving an elevated sensory response, caused by increased reactivity to stimuli. The validity (or otherwise) of this assumption is discussed in more detail later. Across syndromes, the majority of studies have focused on the perception of pain, presumably because this is the most prevalent MUS reported in primary care and is evident across the functional somatic syndromes (Khan, Khan, Harezlak, Tu, & Kroenke, 2003).

Despite some discrepancy, empirical findings appear to indicate reduced perceptual thresholds in at least some patients with MUS. For example, lowered thresholds have been reported in symptom-related pain and discomfort in IBS, FM, TMJD,CFS, and functional

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[1] "Chronic pain" was excluded from the search terms in light of controversy regarding whether chronic pain syndromes should be considered truly medically unexplained, given that such conditions often develop in association with identifiable injury or disease. Pain symptoms in other functional syndromes were nevertheless considered relevant.

dyspepsia (e.g., Awad, Camacho, Martin, & Rios, 2006; Chang et al., 2006; Corsetti, Caenepeel, Fischler, Janssens, & Tack, 2004; Lautenbacher, Rollman, & McCain, 1994; Mertz, et al., 1995; Montoya, Larbig, Braun, Preissl, & Birbaumer, 2004; Naliboff et al., 1997; Park, Clark, Kim, & Chung, 2010; Ritchie, 1973: Trimble, Farouk, Pryde, Douglas, & Heading, 1995; van Laarhoven et al., 2007; Vecchiet et al., 1996). Similarly, reduced non-symptom thresholds have been noted in patients with IBS, TMJD, and FM (e.g., Mountz et al., 1995; Park et al., 2010; Zhou, Fillingim, Riley, & Verne, 2010).

It is well established that there is an increased incidence of MUS in women (e.g., Drossman et al., 1993; Nimnuan et al., 2001), although not all studies take this factor into account during their analyses or consider their findings within this context. In IBS, while reduced thresholds have largely been evidenced in women they have also been reported in male patients (Kim et al., 2006), although not all studies report lowered thresholds in men with IBS (e.g., Chang et al., 2006). Evidence from one PET study suggested that gender related differences in IBS result from differential connectivity of emotional-arousal neural networks rather than different visceral afferent processing (Labus et al., 2008).

Some studies have reported reduced symptom-related thresholds in patients compared to controls with comparable sensory thresholds at non-symptom related sites. For example, Ness et al. (2005) reported reduced bladder sensory thresholds in patients with functional cystitis (i.e., interstitial cystitis/painful bladder syndrome; IC/PBS) compared to controls, while there was no difference between groups in pain thresholds at non-symptom related sites (i.e., arm pain). Furthermore, not all studies have found any evidence for reduced sensory thresholds in patients compared to control groups (e.g., Whitehead, Crowell, Davidoff, Palsson, & Schuster,1997), or demonstrated lowered thresholds for all MUS patients within samples. Lowered thresholds were seen in only 33% of IBS patients in one study (Van der Veek, Van Rood, & Masclee, 2008) and, despite greater reports of pain and discomfort compared to controls, there was no evidence of altered thresholds in others (Elsenbruch et al., 2010; Whitehead et al., 1997). Elsewhere, there was no evidence that thresholds for persons with multiple functional somatic symptoms differed from those of healthy controls (Kuzminskyte, Kupers, Videbech, Gjedde, & Fink, 2010).

Discrepant findings are particularly prevalent in studies relating to colonic thresholds in IBS, possibly reflecting the range of different methodological approaches used to measure sensory thresholds (typically balloon distension paradigms in the rectum/lower bowel), which are wrought with difficulties (Whitehead & Palsson, 1998). Indeed, a variety of methodological inconsistencies may have contributed to discrepant findings across conditions.

Arguably, however, attempting to establish whether methodological differences between studies can account for these discrepancies may detract from a more serious problem: proponents of central and/or peripheral pain sensitization theories have relied on the assumption that reduced thresholds automatically infer enhanced neural sensitivity. However, focusing exclusively on the level or threshold at which individuals detect certain stimuli as an indicator of sensitization is misleading because sensory and pain thresholds are also influenced by psychological factors (Whitehead & Palsson, 1998). Notably, across studies there is a systematic failure to control for cognitive-perceptual biases to respond in certain ways, such as the tendency to report as signal present regardless of whether it is or not. As such, lowered thresholds may reflect a perceptual bias towards reporting a stimulus as present rather than enhanced sensitivity.

One method that clearly separates out physiological and perceptual determinants of sensory thresholds relies on the principles of signal detection theory (SDT: MacMillan & Creelman, 1991), which proposes that each decision about whether a stimulus (signal) is present or absent is subject to the influences of sensitivity (i.e., ability to accurately discriminate signal from noise or d') and response criterion (i.e., tendency to report signal as present or c). Most studies that have applied this approach have done so within the context of IBS, and in general show that apparent differences in sensitivity diminish or disappear once response criterion is accounted for (e.g., Dorn et al., 2007; Naliboff et al., 1997). That is, detecting stimuli at a lower level (i.e., an apparent reduction in sensory threshold) may be due to a general bias to report signal rather than increased accuracy or sensitivity. In contrast, one study showed some evidence for enhanced sensitivity in persons with IBS compared to controls in the absence of response bias (Corsetti, Ogliari, Marino, & Basilisco, 2005), although this finding was not evident across all stimuli intensities and may have resulted from methodological differences

between studies (i.e., looking at discomfort thresholds rather than using individual pain thresholds). Findings suggest that, at the very least, future studies should be controlling for response criterion during threshold testing, as well as extending their remit to conditions other than IBS. Only then, will it be possible to establish the true determinants of sensory thresholds across different types of functional symptoms.

It is possible that psychological factors may also influence 'sensitivity' itself, as suggested by Brosschot's theory (2002) of cognitive sensitization. As such, evidence of increased sensitivity may actually indicate disturbance of top-down processes rather than necessarily inferring enhanced bottom-up ones. Anticipation and expectancy effects, for example, are emerging as important influences on top-down perceptual processes. Evidence supporting their potential role in the development and maintenance of MUS is discussed later.

Investigations of whether or not MUS are associated with a failure to habituate (i.e., reduced reactivity to a stimulus over time; Glaser, 1966) have also yielded mixed results. The tendency to habituate is thought to be adaptive in that it enables repeated presentation of non-noxious or irrelevant stimuli to be disregarded, preventing over-stimulation (Thompson & Spencer, 1966). A variety of methods have been used to examine potential anomalies in habituation in patients with functional symptoms at both symptom-relevant and unrelated sites, including measurement of rate and/or amplitude of neuronal firing in relevant somatosensory pathways using laser evoked potentials (LEPs), thresholding paradigms, and physiological arousal variables (e.g., heartbeat).

In a recent comparison of LEPs to repeated laser stimulation of tender points and non-tender points, deTommaso et al. (2011) found evidence of increased amplitude, and decreased habituation over time, of vertex LEPs (i.e. late components) in patients with FM compared to healthy controls. The authors interpreted this finding as evidence of reduced habituation in FM patients, suggesting that it may indicate altered cortical excitability in the sensory cortex associated with increased depressive symptoms. However, this method does not provide any information about the causes of these differences (e.g., whether they relate to signals coming from the body, or occur in response to top-down processes) nor gives any indication about whether any differences predate the onset of symptoms. Event-related potentials (ERPs) have

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shown reduced attenuation over time in FM patients compared to controls in response to somatosensory stimuli but not auditory stimuli (Montoya et al., 2006). Montoya and colleagues suggest that FM patients exhibit disrupted "sensory gating", whereby the brain's ability to filter out irrelevant information in the presence of a repetitive stimulus is disturbed and that this is specific to processing of somatosensory information rather than indicating a general information processing disturbance. In another study, Horvath, Friedman, and Meares (1980) argued that a failure to habituate to auditory stimuli (measured via heart rate and skin conductance) evident in the majority of their "hysterical" participants relative to anxious controls indicated either an inability to filter out irrelevant information or to form accurate central representations of past stimuli against which new stimuli are matched (Horvath et al., 1980). Rief and Auer (2001), in contrast, found that somatization disorder patients exhibited smaller decreases in heart rate between trials of an attentional task compared to healthy controls, indicating reduced or delayed habituation in relation to physiological arousal in the clinical group.

Evidence from thresholding studies has also been considered relevant to habituation processes. For example, differential patterns of habituation were shown in FM patients and controls towards repeated delivery of heat and cold pain stimuli (Smith et al., 2008). While habituation occurred in both FM patients and healthy controls for heat pain, it was significantly stronger across trials in healthy controls, and while patients gradually habituated in the cold pain condition, FM patients evidenced decreased cold pain thresholds over time. The authors argued that this indicated increased sensitization over time. Decreased habituation to nonnoxious electrical and thermal stimuli has also been suggested in patients with interstitial cystitis or painful bladder syndrome (IC/PBS: Lowenstein et al., 2009; Fitzgerald, Koch, & Senka, 2005): despite showing no evidence of difference in perceptual thresholds across a range of volumes, Lowenstein et al. (2009) found poorer habituation to repetitive bladder fills than in controls. However, measurement of habituation over time also needs to control for psychological factors during perception and potentially decreased thresholds over time may reflect response criterion not adapting over time, whereby individuals remain vigilant to non-threatening information.

In sum, solely relying on reduced or disturbed thresholds as evidence of sensitization or habituation is problematic because it does not take into account psychological influences during

perception. Poor consideration of both physiological and psychological factors across studies has meant that it is difficult to draw conclusions about what causes lowered or altered thresholds and whether these are evident at both symptom-related and non-symptom-related sites. Studies that include a role for response bias suggest this is an important factor that should be accounted for during investigation of perceptual processes. However, it is not clear at present which factors may alter response criterion or sensitivity and how these factors may exert an effect, and furthermore whether such influences are likely to be temporary or more enduring. Potentially a number of psychological factors may exert an influence on response criterion and sensitivity during somatic, including expectation/anticipation of negative effects, subtle threat-related primes, attention (e.g., selective attention/ bodily hypervigilance), and psychological mood states (e.g., negative affect or anxiety) and associated autonomic nervous system activity: these are considered in more detail in the next section.

Psychological factors

  Expectancy effects and anticipation. Negative anticipation or expectation of worsening of symptoms is emerging as a potential top-down influence during symptom perception, which may offer one route to explain the development and maintenance of functional symptoms. Expectation effects are evident in both placebo and nocebo responses. During the placebo phenomenon, expectation that an inert stimulus will have a positive effect is associated with subsequent improvement in symptoms (Price, Finniss, & Benedetti, 2008). Conversely, conscious expectation of negative or aversive consequences (e.g., pain) from a "sham" (i.e., inert) stimulus has been associated with subsequent appearance or worsening of symptoms, the so-called *nocebo* effect. Nocebo-like effects also occur following the mere suggestion of negative changes in the absence of a stimulus (Benedetti, Lanotte, Lopiano, & Colloca, 2007). Experimental investigations of nocebo effects have induced negative expectations by verbally informing subjects about forthcoming pain (or other negative experiences) from a sham stimulus, and compared subsequent neural activation in patients to healthy controls during both the period of anticipation of negative effects and during delivery of sham or active stimuli.

  Both verbal expectancy and conditioning processes have been implicated as possible mediating factors during perception in the placebo and nocebo effects (Enck, Benedetti, &

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Schedlowski, 2008), but uncertainty exists over the precise mechanisms involved. Moreover, while placebo and nocebo responses appear conceptually similar, in that direction of expected outcome influences the direction of subsequent responses (i.e. improvement or deterioration in symptoms), the way in which expectations are induced in the perceptual system in these two phenomena may result from fundamentally different processes (Petrovic, 2008). In a well-designed study, Colloca, Sigaudo, and Benedetti (2008) demonstrated that both hyperalgesic and allodynic responses could be induced in healthy controls via verbal suggestion of increased pain (i.e., negative expectation) using a sham electrode. Comparison of placebo analgesic effects with nocebo hyperalgesic ones indicated that learning via previous experience (i.e., *conditioning*), is more important in placebo than nocebo responses (Colloca et al., 2008). The observation that nocebo effects can occur in the absence of a stimulus altogether (i.e. apparent allodynia) has led many to argue that they are not determined by the same conditioning processes thought to underpin the placebo effect (Colloca et al., 2008). Unfortunately, these findings cannot exclude a role for conditioning in nocebo responses, due to low statistical power. Nonetheless, they highlight the importance of verbal suggestion (and potentially threat-related cues) in the nocebo effect, which appears more affected by the semantic system than the placebo effect.

As well as influencing immediate subjective ratings of pain (that may be highly susceptible to reporting biases), negative expectations of pain have also been shown to have lasting effects on pain perception without further learning (Rodriguez-Raecke et al., 2010). In their study, Rodriguez-Raecke and colleagues found that negative expectations that a constant heat pain (delivered at pain-threshold; Engen, 1971) would increase over time prevented habituation in comparison to controls who were told that the pain would remain the same over an eight day period. In other words, negative expectation that pain would increase over time took precedence over actual (repetitive) sensory information during perception; that is, participants' internally generated cognitive representations of what they thought was happening was more important than the real perceptual signal.

Extending earlier findings from Colloca and colleagues (2008) showing that learning or conditioning process are not necessary in immediate nocebo effects, expectancy effects also

appear to have longevity in the absence of further suggestion or learning. fMRI showed differential activation after eight days in the parietal operculum, a structure thought to be important in cortical representation of touch and pain (Treede, Apkarian, Bromm, Greenspan, & Lenz, 2000). Elsewhere, nocebo effects have shown increased activation in the anterior cingulate cortex (ACC) and insular cortex (IC; Sawamoto et al., 2000), neural structures that have been linked with the affective-motivational aspect of pain perception (Vogt, Derbyshire, & Jones, 1996), possibly related to the integration of affect, cognition and response selection (Devinsky, Morrell, & Vogt, 1995). The insula has been associated with memory for previous pain experiences (Lenz, Gracely, Zirh, Romanoski, & Dougherty, 1997).

Anticipation of sensory input has been associated with activation of similar neuronal networks (e.g., the primary and secondary somatosensory cortices) to those activated by actual sensory input (Carlsson, Petrovic, Skare, Petersson, & Ingvar, 2000). This seems to suggest that, as well as processing actual sensory input, these networks enable the generation of a cognitive representation about predicted incoming sensory information determined by verbal suggestion (i.e., neural activation of these areas can be driven by top-down processes, including anticipation of a stimulus; Carlsson et al., 2000). Interestingly, increased activation in selective parts of the somatosensory cortex during anticipation/expectation associated with the attended process is coupled with decreased activity outside of these areas (e.g., Carlsson et al., 2000). Potentially this may reflect a narrowing of attention to anticipated information, or could indicate deficient signal-filtering – actual information from the body is suppressed and cognitive representation of what think is happening is favored for conscious processing. Carlsson et al. (2000) suggested that this ability to predict the content of incoming sensory information enhances processing efficiency by allowing incoming sensory signals to be matched with what has been predicted. In a recent review, Benedetti, Lanotte, Lopiano, and Colloca (2007) suggest that negative verbal suggestions of pain cause anticipatory anxiety which activates the hormone cholecystokinin thought to enhance the transmission of pain (Benedetti, Amanzio. Vighetti, & Asteggiano, 2006), thereby offering one explanation of how negative expectancy may be translated into pain. Thus, in relation to functional somatization, it might offer one route by

which psychological factors such as threat-perception influences symptom experience even in the absence of clinical anxiety.

In persons with MUS, evidence from sham experimental manipulations has shown that anticipation of symptoms involves broadly similar neural networks as actual stimulus experiences. For example, in one study anticipation and exposure to 'fake' mobile phone radiation were associated with increased activation in the ACC, insular cortex, and fusiform gyrus (indicating enhanced processing of threat-relevant information/sensory cues) of subjectively electrosensitive patients but not controls (Landgrebe et al., 2008). In contrast, presentation of a heat stimulus, which is presumably not threat-relevant in this sample, resulted in similar neural activations in both groups. Initially, it had been thought that exposure to electromagnetic fields could actually trigger symptoms in IEI, however evidence does not support this theory (for a review see Rubin, Nieto-Hernandez, & Wessely, 2010) and a fairly large body of literature reliably suggests involvement of nocebo effects (e.g., Szemerszky, Koteles, Lihi, & Bardos, 2010; Rubin et al., 2010; Rubin, Das Munshi, & Wessely, 2005).

Activation of the ACC and anterior insula has also been demonstrated in other functional syndromes like multiple chemical sensitivity (MCS; i.e., hypersensitivity or reactive symptoms to unrelated chemicals or pollutants which ameliorate following stimulus withdrawal; Hillert, Musabasic, Berglund, Ciumas, & Savic, 2007). Using PET in female MCS subjects with odor sensitivity, Hillert et al. (2007) found processing of an odorant stimuli in MCS patients was associated with increased regional cerebral blood flow (rCBF) in the ACC and cuneus-precuneus compared to healthy controls, despite normal baseline rCBF. Furthermore, MCS patients also showed decreased activation of actual odor-processing brain regions. The study authors propose that this differential activation indicates top-down regulation of odor-response involving the ACC.

Anticipation of or sham bowel distensions have also been investigated in patients with IBS and there is some evidence of differential neural activation during anticipation in persons with IBS compared to controls. For example, Berman et al. (2008) observed reduced inactivation of non-relevant areas during cued anticipation of distension in patients compared to controls, including the insular cortex, an area implicated in memory of previous pain

experiences (Lenz et al., 1998). Subsequent increases in activation following the active stimulus were larger in patients in the ACC and dorsal brainstem. In one study, Silverman et al. (1997) found that anticipation of stimuli was associated with activation of the ACC and other structures in healthy controls but not in patients with IBS, who instead exhibited activation of the left dorsolateral prefrontal cortex, an area that has been associated with somatic and visceral pain. The authors argue that increased activation in this area, together with inhibited ACC activity, is associated with vigilance towards expected stimuli which may lead to a more liberal response criterion. One study reporting that subjective reports of pain were increased in patients with IBS during both sham and active trials (Galati, McKee, & Quigley, 1995) highlights the importance of controlling for expectancy effects during threshold assessments that rely on subjective responses of when participants can feel pain.

In fact, expectancy effects have also been evidenced in interstitial cystitis/painful bowel syndrome (IC/PBS Twiss et al., 2009; Kilpatrick et al., 2009). Twiss et al. (2009) measured acoustic startle responses (ASRs) via eye blink reflexes to look at responses during sham application of electrodes to the lower abdomen in women with IC/PBS and controls. Patients had increased ASRs during non-imminent conditions (i.e., at baseline and in the cued safety condition where no stimulation was possible) while both groups had increased ASRs during context trials (imminent threat of stimulation) although this was higher in patients (group differences were not attributable to differences in anxiety and depression). Twiss et al. (2009) interpreted these findings as evidence of enhanced perception of bladder signals in IC/PBS due to increased activation of threat-related emotion networks in response to threat-relevant contextual cues. Context enhanced ASRs have been associated with activation of the amygdala, ACC and insular cortexes amongst other areas (Alvarez, Biggs, Chen, Pine, & Grillon, 2008).

Evidence that expectations or anticipation may be triggered by semantic primes suggests one pre-conscious route through which perception may be biased (Meerman, Verkuil, & Brosschot, 2011). In their well-designed study, Meerman et al. (2011) found that subliminal illness-related primes delivered to healthy control participants resulted in reduced pain tolerance during a cold pressor task, compared to delivery of non-threat, or emotion, related words. This study demonstrates that contextual-threat information may lower pain thresholds, which has

important implications for study methodologies across the board. Specifically whether this information alters sensitivity or response criterion, and whether or not differences exist between healthy controls and persons with MUS, could be addressed in future research. Elsewhere, emotional priming has been shown to increase response bias towards reporting a heat stimulus present (Kirwilliam & Derbyshire, 2008)

Further evidence for the importance of contextual cues or expectancy processes in MUS comes from two studies using "rebreathing" trials to induce altered respiration (Bogaerts et al., 2008; Bogaerts et al., 2010). Bogaerts et al. (2008) showed that high symptom reporters have worse interoceptive accuracy during recovery from breathlessness following negative but not neutral contextual cues compared to low symptom reporters. This study illustrates how even very subtle language difference (i.e., describing a forthcoming experience as a "symptom" versus a "sensation") may result in altered sensory perception. More recently, Bogaerts and colleagues (2010) showed that when physiological input was relatively weak, the relationship with subjective symptoms was weaker in MUS patients than controls suggesting that in MUS, symptom perception may be more largely determined by cognitive representations of the body rather than by what is actually occurring, especially when incoming information is more ambivalent.

In sum, nocebo effects offer one route to explain the development and maintenance of functional symptoms: negative expectation of symptoms may activate pain-related and memory pathways while at the same time inhibiting neural networks associated with stimuli-relevant processing (e.g., odour; Hillert et al., 2007). Activation of memory related structures (e.g., insular cortex) during expectation is likely to interact with incoming sensory information or could even provoke symptoms in the absence of stimuli in a top-down fashion (Landgrebe et al., 2008). This highlights the importance of controlling for these features and casts doubt over the validity of interpretations or conclusions made by authors who have not considered the impact of anticipation or expectation effects, which presumably may be critical in understanding whether observed effects or perceptual experiences are resulting from bottom-up (from the body) or top-down (from the brain) processes. For example, although altered neuronal responses have been interpreted as evidence for increased reactivity to sensory stimuli (e.g.,

Gracely, Petzke, Wolf, & Clauw, 2002), such studies are also highly susceptible to the influence of anticipation or expectancy effects making it difficult to know what mechanisms underlie differences. While the presence of functional symptoms are likely well-established by the time patients receive a diagnosis of somatoform disorder or functional somatic syndrome, findings of longevity of nocebo effects (Rodriguez-Raecke et al., 2010) with suggestion that symptoms may be triggered by even subtle primes (Bogaerts et al., 2008; Meerman et al., 2011), highlights the importance of information that physicians provide about symptoms which may have effects via expectancy.

In general, the nocebo effect has received much less attention than the placebo effect and investigation of these effects has been complicated by ethical considerations, in that suggestion is likely to actually result in a worsening of symptoms (Benedetti et al., 2007). Although exploration of nocebo effects is highly relevant to functional somatic symptoms the majority of research has focused on understanding the nocebo effect in healthy control subjects.

Attention and self-monitoring. The role of attention in MUS has been investigated in a number of different ways, presumably due to the broad application of the term "attention" and the variety of ways in which attentional processes may be affected. For example, studies have investigated general attention to the body (hypervigilance), selective attention to symptoms, attention to threat-related information, and self-monitoring of attentional processes.

Potentially, attention to the body may result in a cognitive-perceptual response bias or even augment perceptual sensitivity directly. Studies investigating the role of body-focused attention on somatic perception have produced conflicting results: increased vision of the body has been linked separately to both increased and reduced accuracy of symptom detection (Weisz, Balazs, & Adam, 1988; Levine & McDonald, 1981) Evidence from one recent study indicates that discrepant findings may have resulted from differences in the type of attention under investigation: interoceptive (internally focused) and exteroceptive (externally focused) attention have been associated with more liberal, and more stringent, response criterion respectively, but not altered sensitivity (Mirams, Poliakoff, Brown, & Lloyd, *submitted*). Mirams and colleagues suggest that interoceptive attention may increase sensory noise in the perceptual system making it harder to accurately perceive signal, while exteroceptive attention

may lead to a reduction in sensory noise making discrimination between signal and noise easier. However, it is not known from this study whether these effects generalize to threat-relevant symptoms for persons with MUS.

McDermid, Rollman, and McCain (1996) argued that FM patients show general hypervigilance or alertness towards unpleasant stimuli, compared to patients with organic pain (rheumatoid arthritis) and control subjects, indicating an amplifying perceptual style that is not limited to pain (here observed also in response to auditory stimuli). More recently, based on a similar pattern of findings with gentle pressure in FM and TMJD patients, Hollins et al. (2009) argue that pain-related stimuli are amplified as a result of habitual attention paid towards them not on the basis that the sensation is unpleasant.

Evidence from emotional Stroop tasks suggests that people with MUS may exhibit increased attentional-bias towards threat related stimuli. For example, Witthöft, Gerlach, and Bailer (2006) found that attention in persons with IEI was biased towards IEI related words compared to control subjects and patients with somatoform disorder. In both patient groups, there was also evidence of more general health related biases towards general symptom words. However, there was no difference between groups on a dot probe task, which may suggest that these tasks capture different processes. Findings from another study that attempted to separate out preattentive and conscious influences on processing by looking at the effect of subliminal (i.e., below the threshold for conscious perception) and supraliminal (i.e., above the threshold for conscious perception) primes indicated that biases were not operating preattentively (Lim & Kim, 2005). These findings are open to interpretation, however, as delayed response latencies on tasks such as the emotional Stoop and dot probe may be due to other influences on perceptual processing (e.g., increased anxiety) than attentional bias.

It has been suggested that medically unexplained neurological (i.e., conversion disorder) symptoms may involve disruption of late-stage attentional processes and active inhibition prior to conscious awareness (Sierra & Berrios, 1999), coupled with normal early-stage processing (e.g., in functional hearing loss; Fukuda et al., 1996). Consistent with this, evidence suggests that conversion paralysis may result from impaired higher-level intentional processes and

disruption of self-monitoring, rather than lower-level attentional processes (Roelofs et al., 2001).

However, findings in this area should be interpreted cautiously: a recent review of available literature concludes that it is difficult to reliably interpret findings given the small sample sizes and variety of techniques that have been used (Browning, Fletcher, and Sharpe, 2011).

Negative affect. The relationship between stable psychological factors, such as trait negative affectivity (NA; Costa & McCrae, 1987) and alexithymia (i.e., difficulty in understanding and expressing emotions; Sifneos, 1973), with increased somatic symptom reporting is well-documented (DeGucht & Heisser, 2003; Watson & Pennebaker, 1989). In addition, many people with MUS have co-morbid anxiety and depression (Henningsen, Zimmerman, & Sattell, 2003; Löwe et al., 2008) and *state* negative affect (a transient mood factor which corresponds with trait NA; Watson & Pennebaker, 1989) has also been associated with inflated somatic symptom reports (De Gucht, Fischler, & Heiser, 2004). Based on stronger relationships between physical symptom reports and state negative affect, it has been suggested that trait NA leads to increased state negative affect which is then the main influence on symptom experience (Brown & Moskowitz, 1997); potentially, high scores on measures of trait NA may indicate a retrospective memory bias to remember symptoms as worse than they actually were (Larsen, 1992; Watson & Pennebaker, 1989) rather than exerting an effect on the perceptual process per se. More recently, findings from Howren and Suls (2011) suggest that state anxiety and depression affect different aspects of physical symptom reporting, with stronger relationships between depression and retrospective reports and anxiety and momentary reports.

It is not clear whether this relationship between state negative affect and symptom experience reflects a reporting bias, a perceptual response bias, augmented sensitivity, or another alteration of sensory and perceptual processes (e.g., increased deficiency in filtering signal from noise). Lack of progression in understanding this relationship is likely due to the paucity of research using appropriate methods to investigate this relationship. For example, a number of studies looking at the role of mood in somatic amplification have relied on subjective

assessments of somatosensory amplification (such as the SSAS – described earlier; Barsky et al., 1990), which is problematic because it is not clear if this measure accurately captures tendency to amplify somatic experiences or is simply another measure of psychological distress (Aronson et al., 2001). Future studies aiming to clarify the role of negative affect on response criterion and sensitivity need to find objective means of measuring somatic amplification.

From the observation that neural activation is altered in cognitive-emotional networks during experience of MUS related-symptoms (e.g., Twiss et al., 2009), it seems plausible that state negative affect directly distorts the perceptual process. State negative affect involves a number of physiological changes associated with activation of the sympathetic nervous system and it has been suggested across models of MUS that this will alter somatic perception.

In a study investigating whether state negative affect is associated with deregulated stress-physiological systems, Houtveen and van Doorenen (2007) monitored somatic complaints, mood and physiological measures of arousal during the day using electronic diaries: after controlling for baseline activity levels, there was no evidence of dysregulation in stress-physiological systems in persons with MUS compared to healthy control participants. Elsewhere, Van Marle, Hermans, Qin, and Fernández (2009) found that induced acute stress increased sensitivity and reduced specificity of amygdala responses (i.e., enhanced amygdala responses were observed in relation to both threat-related stimuli and stimuli with positive valence post stress induction), arguing that stress induced a general state of hypervigilance that may increase risk of false negatives (i.e., reporting signal present when absent).

Although research has tended to investigate the roles of attention and negative affect separately, emerging evidence suggests that negative affect and anxiety may actually operate by increasing attention to the body or to specific somatic experiences (Stegen, Van Diest, Van de Woestijne, & Van den Bergh, 2001; Wells & Matthews, 1994). The joint impact hypothesis suggests that negative affect exerts an effect on physical symptoms in general by increasing attention to the body (Gendolla, Abele, Andrei, Spurk, & Richter, 2005). In support of this hypothesis, Gendolla et al. (2005) found that negative affect only augments symptom perception when self-focused attention is simultaneously increased.

Across models of MUS it is assumed that negative affect will augment symptom reports although models do not offer specific predictions concerning the effect of negative affect on somatic perception. A lack of research using objective methods and measures of the relationship between mood and somatic amplification has made it difficult to know exactly how mood exerts an effect (e.g., whether mood enhances perceptual sensitivity, or alters response criterion).

Theoretical implications

This section explores whether or not the evidence reviewed above lends support to one or more of the theories of MUS outlined earlier. At present, methodological limitations have made it difficult to interpret findings from thresholding studies. This is due in part to the widespread neglect of perceptual response bias and poor consideration of anticipation or expectancy effects across studies. There were some discrepancies across findings reported when these variables were controlled for potentially suggesting that experimental factors may have been distorting findings. Therefore, from available findings there seems to be little support for the notion of biological sensitization or disturbed habituation processes (Ursin, 1997; Eriksen & Ursin, 2002; Ursin & Eriksen, 2006).

Instead, evidence from expectancy and anticipation literature is suggesting that top-down processes may place a critical role in the generation and maintenance of MUS. The observation that expectancy or anticipation (i.e., nocebo effects) may be induced with subtle threat-related primes (Meerman et al., 2011), is in keeping with Brown's (2004) suggestion that "rogue" illness-related representations can be activated in the perceptual system via memory. Although this may also seem similar to Brosschot's (2002) notion of cognitive sensitization, his model suggests that signals originate from the body which the anticipation literature is suggesting need not be the case. It is difficult to evaluate whether or not the literature relating to attention supports one model over another because models are poor at describing where and how different attentional processes may be altered.

The process of attempting to fit evidence with a particular model has highlighted a number of potential shortcomings across theories and research being carried out in the field. In general, discussion regarding implications for theoretical models was noticeably scant across the majority of papers, with a few notable exceptions (e.g., Bogaerts et al., 2010). This was

especially evident across those studies on biological sensitization, where it was difficult to evaluate the potential implications of findings given the widespread failure to take psychological factors into account. Broadly speaking, models do not provide adequate detail of how different factors contribute to perceptual construction and how this may be altered in MUS. For example, although Rief and Barsky (2005; see also Rief & Broadbent, 2007) identify numerous factors as potential influences on signal filtering, their model does not link these together in a meaningful way such that it is difficult to extract specific hypotheses that could be tested to provide differential support for their model. However, for progress to be made in understanding how perceptual processes might be altered (i.e., in relation to sensitivity or response criterion or both) and to understand mechanisms thought to subsume these changes (i.e., biological vs. cognitive sensitization), it is imperative for studies to exert greater control over their experimental paradigms.

At the same time as apparent neglect of psychological factors across medical literature, in general, models lack cohesive integration with neuro-biological theories of MUS (Roelofs & Spinhoven, 2007) and do not tend to incorporate findings from neuroimaging and electrophysical approaches, with few exceptions (e.g., Brown, 2004). This likely parallels the dichotomy between medical and psychiatric approaches, but means that until recently, fields have been attempting to progress in relative isolation of each other.

Conclusions

Evidence indicates that while perceptual thresholds may be altered in some persons with functional symptoms, this is certainly not true for all observed cases, and a group difference between patients with MUS and healthy controls is not a reliable finding. This may reflect methodological differences, particularly related to the fact that studies investigating sensation and perception in functional symptoms differ substantially in their consideration of psychological and physiological factors. There was some evidence that when psychological factors such as perceptual response bias is accounted for, apparent threshold differences or reduced sensitivity is significantly lessened, although again there were exceptions to this finding. Studies need to control systematically for this factor during assessment of apparent sensitization as part of a methodologically sound design. The lack of consideration for

psychological influences during perception has likely fuelled existing controversy about whether or not functional somatic symptoms result from basic physiological differences or represent part of the spectrum of somatization, because many authors have prematurely concluded that lowered thresholds are tantamount to enhanced sensitivity (and therefore basic biological differences) in MUS.

Given the ethical limitations involved with investigating the nocebo effect, the majority of research that has been carried out has focused on healthy volunteers rather than MUS patients and this is clearly one area that would benefit from further attention. Despite recent developments in understanding these cognitive-perceptual effects, the role of negative expectations during symptom experience is not routinely considered in empirical studies purporting to investigate perceptual differences in MUS. The observation that anticipating an adverse stimulus is associated with similar neural networks as for actual adverse experiences has important implications for recruitment methods and information given to subjects about what to expect during experiments (Rodriguez-Raecke, et al., 2010). Potentially, even subtle suggestions or vocabulary differences may activate neural networks associated with cognitive representations of pain so that subjects are responding to an internal perceptual construction rather than sensory signals that are actually coming from body. This may be particularly noticeable within some methodological approaches, for example in studies that used standard increases in pain over time as these may become predictable to subjects, and further may have particular salience for people with functional symptoms so that similar effects are not observed in control subjects. Studies on context information show how even subtle differences (e.g., the use of the term "symptom" instead of "sensation"; Bogaerts et al., 2008) can create pre-attentive perceptual biases such that apparently minor methodological differences relating to information given to participants may influence their expectations. The suggestion that perception of symptoms could be dominated by a cognitive memory representation activated by even subtle pre-conscious primes in persons with MUS clearly also has implications for practising clinicians in terms of their interactions with patients and information they provide about symptom causes and prognosis.

One clear shortfall highlighted by this review of existing literature is the focus on the perception of pain across the functional somatic syndromes, presumably because this is the most frequently reported MUS in primary care (Khan et al., 2003) and possibly the easiest to study. Experience of other symptoms observed in the functional somatic syndromes (e.g., urgency in IBS), may involve similar perceptual alterations (i.e., a more liberal response criterion). Alternatively, different symptoms may be associated with disturbances in different parts of the perceptual process. Until these findings are replicated or investigated more thoroughly, it is difficult to draw firm conclusions about disturbance of common perceptual processes across different types of functional symptoms. In addition, most samples are predominantly female, most likely due to the increased prevalence of MUS in females (Nimnuan et al., 2001). However, this makes it difficult to know to what extent these findings are applicable to MUS observed in men and future studies should attempt to recruit more males to their studies.

Importantly, studies that attempt to investigate perception of such symptoms should account for both physiological and psychological aspects of perception to facilitate understanding of at what point the perceptual process is altered.

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A brief preface to the empirical study

As identified during review of the literature in Chapter 1, poor specificity across theoretical models of exactly how the perceptual process may be altered for MUS has made it difficult to know what predictions different models might make during somatic perception and thus to differentiate between them.

Some variant of somatosensory amplification (Barsky et al., 1988) is incorporated across models, although the lack of available standardised methods to measure such amplification has made it somewhat difficult to evaluate the validity of different models. To address this shortfall, Chapter 2 presents an empirical study that has used a novel objective paradigm to measure somatic amplification in nonclinical "high" and "low" symptom reporters, with a view to evaluating predictions from different models of MUS.

The literature review also highlighted a dearth of studies investigating the role of state negative affect on somatic amplification under controlled experimental conditions. Therefore, the study was also designed to explore the effects of induced negative affect on aspects of the perceptual process (i.e., response bias and sensitivity).

Chapter 2: Empirical paper

Using signal detection theory to investigate the effect of state negative affect on somatosensory amplification in nonclinical high and low physical symptom reporters

Abstract

Theory suggests that negative affect augments the tendency to amplify somatic sensations in high symptom reporters, although there is a shortage of research that has used objective measures of somatosensory amplification. To address this shortfall, eighty-three participants with high or low physical symptom scores on the Patient Health Questionnaire (PHQ-15) were compared on a somatic signal discrimination task (SSDiT) where subjects had to differentiate weak and strong tactile vibrations, both before and after either a neutral or negative mood induction. Contrary to expectation, there were no baseline differences in response criterion between high and low symptom reporting groups. Over time, for response criterion, low and high symptom reporters became more and less conservative, respectively, such that in the neutral condition lows were significantly more conservative at T_2 than highs. There was a trend for low symptom reporters (but not highs) to become more conservative at T_2 compared to baseline, although this finding did not reach significance. There was no evidence of change over time for highs, or between groups following the negative mood induction, suggesting that negative affect may prevent drift in response criterion that would otherwise occur.

Keywords: Somatosensory/ somatic amplification; physical symptom reporting; negative affect; sensitivity; response criterion; somatic signal discrimination task

Introduction

At least one third of physical symptoms reported in primary care do not have an identifiable medical cause (Kroenke, 2003). Despite increased emotional distress and functional disability for patients and high health care utilization, mechanisms responsible for these medically unexplained symptoms (MUS) are poorly understood. The term MUS (Mayou, 1993) encompasses a range of related conditions and phenomena, including the somatoform disorders, functional somatic syndromes (e.g., irritable bowel syndrome, chronic fatigue syndrome) and

the more general tendency for excessive physical symptom reporting (so-called *somatization*; Kirmayer & Robbins, 1991)[2]. Psychological factors are widely thought to contribute to the development and maintenance of these conditions. For example, trait negative affectivity (NA; i.e., general tendency to experience negative emotions; Costa & McCrae, 1987) is associated with poorer subjective, but not objective, health status (Watson & Pennebaker, 1989). *State* negative affect has also been associated with inflated somatic symptom reports (De Gucht, Fischler, & Heiser, 2004), although how mood exerts its effect is not well understood.

One process implicated in symptom perception that may be susceptible to the influence of mood is that of *somatosensory amplification.* Defined as "a tendency to experience somatic and visceral sensation as unusually intense, noxious, and disturbing" (Barsky, Wyshak, & Klerman, 1990, p. 323), somatosensory amplification broadly comprises three components: excessive attention to the body (so-called "hypervigilance") and thereby increased detection of somatic sensations, selectively focusing on detected sensations, and attribution of sensations to noxious rather than benign causes. Although originally conceptualised in relation to hypochondriasis (i.e., excessive health worry), somatic amplification is also thought to influence symptom perception more generally (Barsky, 1992) and theoretically occurs at both trait (as an enduring perceptual style) and state (as a transient process) levels. Barksy (1992) suggests that the presence of state negative affect is likely to further augment somatic complaints.

Studies in this area have typically relied on the self-report Somatosensory Amplification Scale (SSAS; Barsky et al., 1990), which asks respondents to rate how much they are bothered by ten bodily-related experiences. While the SSAS has acceptable convergent validity with other symptom reporting measures (Barsky, Goodson, Lane, & Cleary, 1988; Barsky & Wyshak, 1990), evidence from more objective measures of somatic amplification (e.g., heartbeat detection accuracy) indicates that the scale simply measures NA, rather than somatic sensitivity *per se* (e.g., Aronson, Barrett, & Quigley, 2001). Other research suggests that it may also capture somatic avoidance (Brown, Poliakoff, & Kirkman, 2007). As such, it is difficult to evaluate the validity of the somatosensory amplification hypothesis.

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[2] Although controversy exists about exactly what constitutes a MUS, this paper follows convention and assumes that this diverse array of conditions belong to the same general domain.

The somatosensory amplification model implies that individual differences exist in somatic perception. The term perception covers a range of different processes, some of which have been studied in relation to physical symptom reporting. For example, numerous studies have investigated pain thresholds in functional syndrome patients, suggesting that basic biological differences exist in our reactivity to sensory stimuli (i.e., sensitization; Ursin, 1997), which might account for elevated symptom reports. Unfortunately, most studies have focused solely on the detection of internal physiological changes: focusing exclusively on the level or threshold at which individuals detect certain stimuli is misleading because it does not control for relevant psychological factors (e.g., biases to respond in a certain way; Lloyd & Appel, 1976). Studies using measures derived from signal detection theory (SDT: MacMillan & Creelman, 1991) indicate that apparent differences in detection thresholds diminish once this response bias is accounted for (see e.g., Whitehead & Palsson, 1998).

A novel approach to perception and symptom reporting based on the principles of signal detection theory (SDT) is the Somatic Signal Detection Task (SSDT; Lloyd, Mason, Brown, & Poliakoff, 2008). During this task, participants complete a series of trials where they report whether a tactile stimulus (vibration) is present or absent, with the vibration present on only 50% of trials. Responses are classified as false alarms (vibration reported present when absent), hits (vibration reported present when present), correct rejections (vibration reported absent when absent), and misses (vibration reported absent when present). These classification data are then used to estimate perceptual sensitivity (d') and response criterion (c; a measure of bias) using standard signal detection formulae. According to the authors, false alarms on the SSDT can be thought of as illusory touch experiences that are analogous to unexplained physical symptoms such as those seen in the functional somatic syndromes.

The SSDT paradigm was developed to test the Brown (2004) model of MUS, which suggests that symptoms are intrusions in awareness that result from the over-activation of symptom representations: the observed elevated false alarm rate was interpreted as evidence in support of this hypothesis. Consistent with this, research with both non-clinical and clinical samples suggests that physical symptom reports are associated with false alarm rate on this task, leading to a more liberal response criterion for high symptom reporters (Brown, Brunt,

Poliakoff, & Lloyd, 2010; Brown et al., submitted). Alternatively, this increased false alarm rate may indicate somatic amplification, whereby high symptom reporters are more likely to detect other sensations in the fingertip (e.g., pulse) and misattribute those to the vibration (as suggested by Mirams, Poliakoff, Brown, & Lloyd, 2010). Lloyd and colleagues (2008) showed that the simultaneous presentation of a light leads to a more liberal response criterion, increasing both hit rate and false alarms on the task. The authors suggested that the light may orient attention to the hand, increasing awareness of sensory noise (e.g., heartbeat) that is then attributed to the vibration. In support of this interpretation, Mirams et al. (2010) found that viewing the hand in the presence of the light led to an increased number of false alarms compared to when the hand was not visible.

This study uses an adapted version of this task which offers a more explicit way of examining somatic amplification and its relationship to symptom reporting. During the Somatic Signal Discrimination Task (SSDiT), participants complete a series of trials where they are required to differentiate between "weak" and "strong" vibrations in light-absent and light-present conditions. Following SDT, strong vibrations are regarded as "signal present" and weak vibrations as "signal absent", with responses being classified as hits (correct identification of strong vibrations), false alarms (weak vibrations reported as strong), misses (strong vibrations reported as weak), and correct rejections (correct identification of weak vibrations) accordingly. Response criterion as such, provides an objective measure of individual situational differences in somatosensory amplification, whereas sensitivity indicates internal differences in ability to discriminate sensory signal from noise. As in the SSDT, a simultaneous light flash occurs on 50% of trials to maximize the false alarm rate. Using this paradigm, we aimed to investigate the effect of negative affect on somatosensory amplification in a non-clinical population of "high" and "low" symptom reporters.

Potentially, findings here may help to differentiate between models of MUS, as these would make different predictions in relation to SSDiT performance. Broadly speaking, negative affect is expected to increase physical signals from the body and increase focus on bodily symptoms, thereby further augmenting any symptom-reporting group differences. The somatosensory amplification model (Barsky & Wyshak, 1990) might predict heightened

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sensitivity in high-symptom reporters together with a more liberal response criterion (proposing that enhanced detection, and misattribution, of bodily sensations amplifies future perception of physical signals): high-symptom reporters may be more likely to detect the increase in physical signals associated with negative affect. Conversely, Rief and Barsky's signal-filtering model (2005) where MUS arise from distorted filtering of sensory input, would predict poor sensitivity in high-symptom reporters, which may be further reduced in the presence of increased signal from negative affect. Sensitization theory (Ursin, 1997) argues that perceptual amplification predicts enhanced sensitivity with no effect on response criterion, suggesting increased hit rates in the absence of change to response criterion in response to negative affect. Brown's model (2004), where symptoms arise from the activation of mental representations of the body rather than from the body itself might suggest an elevated response criterion coupled with either similar or reduced sensitivity compared to non-reporters. Negative mood may increase the activation of rogue representations, whereby high symptom reporters in the negative mood condition may report more false detections than high- (and low-) symptom reporters in the neutral condition, observed primarily via a more liberal response criterion.

Given that the models make different predictions for SSDiT performance, we simply predicted that there would be (i) significant baseline differences in tactile sensitivity and/or response criterion between high and low symptom reporters; (ii) an interaction between symptom reporting group and mood condition, such that high and low symptom reporters would respond differently to the neutral and negative affect conditions.

Method

Design

A 2 x 2 x 2 mixed experimental design was used with mood condition (neutral vs. negative), and PHQ-15 group (low vs. high) as between-subjects factors and light (present vs. absent), as a within-subjects factors. The main dependent variables were change in tactile sensitivity (d') and response bias (c). Ethical approval was obtained from the University of Manchester School of Psychological Sciences Research Ethics Committee.

Participants

Five hundred and sixty-three students completed an online version of the Patient Health Questionnaire (PHQ-15; Kroenke, Spitzer, & Williams, 2002; see Appendix 2) to identify relatively low and high symptom reporters using the following cut-offs: "lows" (score < 5), "highs" (score $\geq$ 10). Although a score of $\geq$ 15 has been suggested as clinical criterion elsewhere (Kroenke et al., 2002), a score of $\geq$ 10 has been shown to be optimal in terms of sensitivity and specificity when identifying patients with somatoform disorders in outpatient settings (Körber, Frieser, Steinbrecher, & Hiller, in press) and was considered suitable as we aimed to recruit an analogue sample of relative high symptoms reporters from the general population rather than a clinical sample as such. Of 198 low (35.17%) and 120 high (21.31%) symptom reporters invited to participate in the experimental session, 83 took part (22 male; mean age = 21.76 years, SD = 5.25 years)[3]. There was no difference in age between groups and conditions, although there was an uneven distribution of males across groups and conditions ($\chi^2_{(3, n = 83)}$ = 21.41, $p < .001$; neutral highs = 1, neutral lows = 11, negative highs = 1, negative lows = 9).

All subjects were right-handed according to the Edinburgh Handedness Inventory (EHI: Oldfield, 1971, Appendix 3), and used their left index finger for the SSDiT to minimise differences in tactile sensitivity and response times between dominant and non-dominant hands (Goldblatt, 1956).

Materials

Questionnaires. The Patient Health Questionnaire somatic symptom scale (PHQ-15; Kroenke et al., 2002) was used to identify low and high symptom reporters. This measure captures the most common physical symptoms observed in primary care (Körber et al., in press; Kroenke et al., 2002). Respondents were asked to rate how much they had been bothered by 15 somatic symptoms in the past four weeks from 0 (*not bothered at all*) to 2 (*bothered a lot*). The measure had good internal reliability here (α =.83).

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[3] Four additional participants consented to take part but were not included in the final analyses: two participants withdrew their consent during the mood induction and two participants performed at ceiling across the SSDiT.

The Somatosensory Amplification Scale (SSAS; Barksy et al., 1990; Appendix 4) was included to explore the relationship between subjective and objective somatic amplification. Although the SSAS has demonstrable convergent validity with a number of subjective measures (e.g., hypochondriacal symptoms; Barksy & Wyshak, 1990), the extent to which this measure is associated with objectively measured somatic sensitivity remains questionable (Aronson et al., 2001). Respondents were asked to rate how characteristic each of 10 items regarding their experience of uncomfortable bodily sensations was on a five-point Likert scale ranging from 1 (*not at all true*) to 5 (*extremely true*). The authors demonstrated good test-retest reliability (*r* =.79) and internal consistency ($\alpha = .82$). Internal consistency was slightly lower in the present sample ($\alpha = .70$) but still acceptable.

The trait version of the State and Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970; Appendix 5) was administered to control for trait anxiety (i.e., NA), known to be associated with increased physical symptom reports (Watson & Pennebaker, 1989). Participants were asked to rate how they generally feel on 20 items pertaining to anxiety using a four-point Likert scale ranging from 1 (*not at all*) to 4 (*very much so*). The STAI is a widely used measure with concurrent validity with other anxiety measures (*r*'s ranging from .73-.85), and recognised test-retest reliability (trait scale *r* = .73). Reliability was excellent here ($\alpha =.90$).

The Depression Anxiety Stress Scales – 21- item (DASS21; Appendix 6), an abbreviated version of the 42- item DASS (Lovibond & Lovibond, 1995), was used to measure anxiety and depression symptoms over the preceding week. The DASS21 comprises three 7-item scales yielding separate scores for anxiety, stress, and depression. Respondents indicated to what extent they had experienced each of 21 items over the past week from 0 (*did not apply over the past week*) to 3 (a*pplied most of the time over the past week*). In this sample, reliability was adequate: overall score ($\alpha = .89$); depression ($\alpha = .89$); anxiety ($\alpha = .69$); and stress ($\alpha = .80$).

Three 11-point Likert scales were completed pre- and post- mood induction as a manipulation check (see Appendix 7). Participants were asked to rate how they were feeling "at the present moment" from 0 (*not at all*) to 10 (*extremely*) for "anxious," "depressed," and "disgust". These were combined to form total mood rating scores ($\alpha = .76$): difference scores (total mood score T_2 – total mood score T_1) were used in further analyses. A modified version of

the Symptom-checklist (SCL; Pennebaker, 1982; Appendix 8), incorporating two symptoms common in primary care ("pain" and "fatigue": Kirkwood et al., 1982), was used to capture changes in physical symptoms. Participants rated present-moment experience of all 14 symptoms on seven-point Visual Analogue Scales ranging from 0 to 6. Items were summed, yielding an overall score between 0 and 84, with good internal consistency (α = .85).

Mood manipulation. Images from the International Affective Picture System (IAPS: Lang, Bradley, & Cuthbert, 2005) were used to manipulate mood. The IAPS comprises a large set of visual images (~900) standardised for emotional arousal and valence (ranging from 1 to 9). Despite differences in experimental methodologies across studies (e.g., number of pictures displayed; length of time presented), the IAPS has shown consistency in inducing negative affect (e.g., Lincoln, Lange, Burau, Exner, & Moritz, 2010; Van Dillen, Heslenfeld, & Koole, 2009).

For the negative mood condition, unpleasant threat-related pictures (n = 20) pertaining to bodily symptoms or health with negative valence (M = 2.26, SD = 0.66) and high arousal (M = 6.07, SD = 0.63) were selected from the database; for the neutral condition, picture sets unrelated to health or bodily symptoms with neutral valence (M = 5.00, SD = 0.26) and low arousal (M = 2.86, SD = 0.76) were chosen (n = 20).

Pictures were presented sequentially on a monitor screen. Each picture was displayed for 6 seconds. To augment processing of stimuli, all pictures were preceded by a 10 second text description of the image content (Appendices 9 & 10). Providing such contextual references influences the manner in which visual stimuli are interpreted and enhances the impact of emotionally evocative images (Tomarken, Davidson, & Henriques, 1990).

Somatic Signal Discrimination Task (SSDiT). Participants sat at a desk in front of the stimulus array in a dark university laboratory lit by a desk lamp. For the SSDiT their left index finger was fixed to a vibrotactile bone conductor (Oticon Limited, B/C 2-PIN 100 Ohm, Hamilton, UK) using double-sided adhesive tape. A 5mm red light-emitting diode (LED) was attached to the side of the conductor and these were mounted together on a polystyrene base. Vibrations were produced by driving the bone conductor using the amplified sound output from a desktop computer. The weak and strong vibrations were both 20ms in duration and 100Hz, but

the strong vibration was three times stronger than the weak. The experiment was run using *E-Prime* software (Psychology Software Tools Inc., Pittsburgh, PA). A monitor placed behind the stimulus array was used to present the green arrow cue and written instructions. Instructions were also delivered verbally.

Participants were given examples of both vibrations and asked to confirm that they could feel all vibrations to ensure vibrations were above each participant's perceptual threshold before proceeding to the practice trial. White noise was played through headphones to mask the sound of the vibrations. First, participants completed a practice phase consisting of 20 vibrations, which they were asked to identify as weak or strong using a keyboard. A green arrow was presented on the left side of the monitor screen at the same time as the vibration and participants were instructed to look at their left hand. The LED flashed on half of the experimental trials on each block (but not during the practice) to maximise the number of false alarms. Participants were told to respond to the vibration rather than the light. Participants who performed at chance level or below during the practice trials had to repeat this phase prior to continuing into the experimental phase. The vibration intensities were increased for one participant who remained at chance level after repeating the practice block, using the same intensity ratio between weak and strong vibrations. Then the experimental phase was split into three blocks comprising 80 trials each, with participants able to rest briefly between blocks. For each block, 20 trials were presented in random order across each of the four conditions [strength (weak, strong) x light (present, absent)]. The experimental phase was repeated post-mood induction.

Procedure

The study was advertised to University students via posters, a website, and an experiment credit system (Appendix 11). The SSAS was administered with the PHQ-15 as part of the online screen. Prior to beginning, all participants confirmed that they had read the information sheet (Appendix 12) and were given a verbal description of the procedure before providing written consent (Appendix 13). Participants were not informed of the objective of the mood induction until completion. Participants received either £5 or course credits for participation.

The EHI, STAI-T, DASS-21, modified SCL and state mood measures were administered first. Participants were then given examples of the weak and strong vibrations on the SSDiT and

completed the practice phase prior to the first experimental phase. Participants were then randomised to either the "negative" or "neutral" mood condition (stratified by PHQ-15 group to ensure similar numbers of high and low symptom reporters across conditions) before repeating the state mood measure and the adapted SCL. Participants then completed the second experimental phase of the SSDiT before debriefing. The experimental session lasted for 60 minutes.

Statistical Analysis

Two non-normally distributed variables (DASS-21[4]; SSAS[5]) were normalised via transformation following Tabachnick and Fidell (2001). SCL[6] ratings and mood rating scores could not be normalised. A symptom change score (SCL; T_2 - T_1) was rendered normal via reassignment of one outlier value (to the next lowest score plus 1; Field, 2005).

Responses on the SSDiT were classified as hits (correct identification of strong vibrations), false alarms (weak vibrations reported as strong), misses (strong vibrations reported as weak), and correct rejections (correct identification of weak vibrations). SDT statistics d' (tactile sensitivity) and c (response criterion) were calculated from the raw data (MacMillan & Creelman, 1991). High scores for d' and c indicated higher sensitivity and a more conservative response criterion, respectively. As the main interest during these analyses resided with d' and c, raw data were not analysed *a priori* although were used during selected *post hoc* non-parametric comparisons[7] to facilitate interpretation.

Prior to testing the main hypotheses, group characteristics were explored using between-subjects multivariate ANOVA. The Wilcoxon signed-ranks test was used to confirm significant changes in mood ratings across PHQ-15 groups and to explore changes in physical symptom reports. Pearson's correlations (r) were then used to establish whether subjective and objective measures of somatic amplification correlate (i.e., SSAS scores and c).

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[4] DASS-21 scores were positively skewed for highs; square root transformation rendered all cells normal.
[5] Data for the SSAS was positively skewed for high symptom reporters; log transformation rendered all cells normal.
[6] SCL scores were positively skewed for low symptom reporters.
[7] False alarm rates and hit rates were non-normally distributed across PHQ-15 group and mood condition and could not be normalised.

For clarity, results relating to the main hypotheses are presented separately from analyses pertaining to the light, which were considered more exploratory. To test the first hypothesis, that significant baseline differences in tactile sensitivity and/or response criterion exist between high and low symptom reporters, separate custom ANOVA were used to explore group comparability for *d'* and *c* at baseline with light as a within-subjects factor and PHQ-15 group, mood condition and gender as between-subjects factors. Gender was included to adjust for imbalances in the number of males and females across symptom-reporting groups although interactions involving gender were not explored, as this study was not designed to look for potential interactions with gender. These analyses were run first without, then with, DASS-21 and STAI-T scores as covariates (mean-centered to prevent them from rendering within-subject analyses more conservative, Delaney & Maxwell, 1981).

To evaluate the second hypothesis, that changes in SSDiT performance would be associated with an interaction between symptom reporting group and mood condition, *d'* and *c* change scores were first calculated. These difference scores did not vary across block according to either PHQ-15 group or mood condition and were collapsed across block at both time points. All values were normally distributed across groups and conditions after reassignment of one outlier value (Field, 2005). Separate custom ANOVA were then carried out for change in *d'* and change in *c* with mood condition, PHQ-15 group, and gender as between-subject factors and light as a within-subject factor, first without, then with DASS-21, STAI-T, symptom change and mood change scores as covariates (mean-centered). Mood change scores for lows in the neutral condition were non-normally distributed (high kurtosis) and could not be normalised via transformation despite the reassignment of one outlier value. All statistical analyses were carried out using SPSS Version 16.0 (SPSS Inc., Chicago, IL).

Results

Group characteristics

There was a significant effect of PHQ-15 group for questionnaire responses (Table 1), $F_{(3, 77)} = 11.68$, $p < .001$, $\eta_p^2 = .31$. High symptom reporters scored higher across questionnaires: STAI-T, $F_{(1, 79)} = 11.24$, $p = .001$, $\eta_p^2 = .13$; SSAS, $F_{(1, 79)} = 15.32$, $p < .001$, $\eta_p^2 = .16$; DASS-21, $F_{(1, 79)} = 27.64$, $p < .001$, $\eta_p^2 = .26$. Questionnaire responses were comparable for participants

across mood conditions, $F_{(3, 77)} = .33$, $p = .81$, $\eta_p^2 = .01$, and there was no effect of gender, $F_{(3, 77)} = .91$, $p = .44$, $\eta_p^2 = .03$. For PHQ-15 scores, Mann-Whitney U tests showed no baseline differences between high scorers in the neutral and negative conditions, $Z = -.24$, $p = .81$, or between low scorers in the neutral and negative condition, $Z = -.31$, $p = .76$. As expected, there was a significant difference in PHQ-15 score between highs and lows, $Z = 7.89$, $p < .001$.

Table 1 Median (*IQR*) questionnaire scores for PHQ-15 groups across mood conditions

PHQ-15 group	Mood condition	PHQ-15	STAI-T[1]	SSAS	DASS-21
Low	Neutral ($n = 20$)	1.50 (3.00)	32.85 (5.33)	20.50 (9.00)	8.50 (9.50)
($n = 41$)	Negative ($n = 21$)	2.00 (2.00)	32.19 (7.77)	21.00 (9.00)	6.00 (7.00)
	Total	**2.00 (2.00)**	**32.51 (6.62)**	**21.00 (9.00)**	**6.00 (8.00)**
High	Neutral ($n = 22$)	11.00 (3.00)	39.77 (8.59)	26.00 (8.00)	15.00 (16.50)
($n = 42$)	Negative ($n = 20$)	11.50 (3.00)	39.80 (7.42)	25.00 (4.00)	15.42 (10.75)
	Total	**11.00 (3.00)**	**39.79 (7.95)**	**26.00 (6.00)**	**15.00 (12.25)**

PHQ-15 = Patient Health Questionnaire; STAI-T = State-Trait Anxiety Inventory – Trait Version; SSAS = Somatosensory Amplification Scale; DASS-21 = Depression Anxiety and Stress Scale
[1] Means and standard deviations presented as scores were normally distributed across all cells

Manipulation check

Wilcoxon signed-ranks tests confirmed that the negative mood manipulation was successful (Table 2), with both PHQ-15 groups rating their mood as significantly worse post mood induction, highs, $Z = -3.93$, $p < .001$; lows, $Z = -3.56$, $p < .001$. In the neutral condition, both groups exhibited improvements in their mood, highs, $Z = -3.79$, $p < .001$; lows, $Z = -2.83$, $p = .005$. Mann-Whitney U tests showed that highs rated their mood as significantly worse than lows at baseline, $Z = -5.24$, $p < .001$. Mood improved significantly more for highs than lows in the neutral condition, $Z = -2.77$, $p = .006$, but there was no difference between groups in the extent of mood change in the negative condition, $Z = -1.61$, $p = .11$. In the negative condition, number of physical symptoms reported following the mood induction increased significantly for the low group, $Z = -1.95$, $p = .05$, but not for highs, $Z = -.99$, $p = .32$. For the neutral condition, symptom reports decreased over time for both groups, highs, $Z = -3.17$, $p = .002$; lows, $Z = -3.38$, $p = .001$.

Table 2 Median (*IQR*) pre- and post- mood induction mood rating scores by PHQ-15 group for neutral and negative conditions

PHQ-15 group	Mood condition	Pre mood rating[1]	Post mood rating[1]	Mood difference	SCL (pre)	SCL (post)	SCL difference[2]
Low	Neutral	2.00 (3.00)	0.00 (2.00)	0.00 (0.00)	7.00 (9.00)	5.00 (6.75)	-2.58 (2.47)
	Negative	1.00 (3.50)	6.00 (7.00)	-4.00 (5.50)	6.00 (11.39)	8.00 (6.50)	1.61 (3.66)
High	Neutral	6.50 (6.25)	3.00 (5.25)	0.00 (3.25)	20.50 (14.50)	13.50 (14.12)	-3.95 (4.50)
	Negative	5.00 (7.00)	13.50 (7.00)	-6.50 (5.00)	17.50 (15.29)	19.00 (15.50)	1.72 (6.71)

PHQ-15 = Patient Health Questionnaire; SCL = Adapted Symptom Checklist
[1] A high score indicates poorer mood ratings
[2] Mean scores (*SD*) presented as scores were normally distributed

Subjective and objective somatic amplification

Pearson's correlations revealed that SSAS scores were not significantly correlated with *c* at baseline, $r_{(81)} = .12$, $p = .27$, or at T_2, $r_{(81)} = .16$, $p = .16$. However, SSAS scores were positively correlated with the DASS-21, $r_{(81)} = .48$, $p < .001$, and the STAI-T, $r_{(81)} = .23$, $p = .04$, suggesting that the SSAS provides another indicator of psychological distress. For this reason, SSAS scores were excluded from remaining analyses.

Evaluation of hypothesis 1

Descriptive statistics for tactile sensitivity (*d'*) and bias (*c*) pre- and post- mood induction for both light conditions are presented in Table 3 alongside raw data (false alarm rates and hit rates).

Response criterion (*c*). There was no effect of symptom-reporting group, $F_{(1, 79)} = 2.17$, $p = .15$, mood condition, $F_{(1, 79)} = .14$, $p = .71$, or gender, $F_{(1, 79)} = 3.38$, $p = .07$. After the addition of covariates (DASS-21, STAI-T), the pattern of results for *c* remained unchanged.

Tactile sensitivity (*d'*). There was no effect of PHQ-15 group, $F_{(1, 79)} = .79$, $p = .38$, mood induction group, $F_{(1, 79)} = 0.01$, $p = .94$, or gender, $F_{(1, 79)} = 2.93$, $p = .09$. Following addition of covariates (DASS-21, STAI-T), results largely remained the same except for a trend towards an interaction involving the light (see below).

Table 3 False alarm and hit rates (Median, *IQR*) and tactile sensitivity, *d',* and response criterion, *c, (*Mean, *SD*) across PHQ-15 group, mood condition and light condition

PHQ-15 group	Mood condition	Light condition	Pre-induction false alarm rate (%)	Post-induction false alarm rate (%)	Pre-induction hit rate (%)	Post-induction hit rate (%)	Pre-induction d'	Post-induction d'	Pre-induction c	Post-induction c
Low	Neutral	Present	18.85 (18.85)	13.11 (13.11)	95.08 (81.97)	93.44 (10.25)	2.69 (0.77)	2.64 (0.66)	-0.38 (0.43)	-0.20 (0.52)
		Absent	4.10 (1.64)	3.28 (4.51)	85.25 (19.26)	85.25 (21.72)	2.77 (0.77)	2.80 (0.63)	0.39 (0.35)	0.42 (0.49)
		Total	**11.48 (13.00)**	**8.61 (11.00)**	**90.98 (11.00)**	**90.98 (16.00)**	**2.73 (0.72)**	**2.72 (0.59)**	**0.01 (0.35)**	**0.11 (0.47)**
	Negative	Present	13.93 (18.03)	12.30 (15.57)	95.90 (98.36)	97.54 (7.38)	2.58 (0.69)	2.92 (0.75)	-0.35 (0.57)	-0.38 (0.50)
		Absent	4.10 (3.28)	4.10 (5.74)	76.23 (29.51)	89.34 (22.13)	2.64 (0.71)	2.98 (0.74)	0.47 (0.45)	0.34 (0.45)
		Total	**9.02 (16.00)**	**7.38 (9.00)**	**82.79 (36.00)**	**94.26 (14.00)**	**2.61 (0.66)**	**2.95 (0.65)**	**0.06 (0.41)**	**-0.02 (0.43)**
High	Neutral	Present	15.57 (20.49)	13.93 (16.39)	95.90 (9.15)	94.26 (8.20)	2.57 (0.72)	2.61 (0.90)	-0.34 (0.38)	-0.20 (0.53)
		Absent	2.46 (3.28)	4.10 (10.25)	72.95 (29. 10)	79.50 (18.44)	2.68 (0.77)	2.60 (0.84)	0.64 (0.35)	0.47 (0.45)
		Total	**10.25 (11.00)**	**9.43 (13.00)**	**83.61 (16.00)**	**86.48 (16.00)**	**2.62 (0.68)**	**2.61 (0.82)**	**0.15 (0.31)**	**0.13 (0.44)**
	Negative	Present	13.93 (15.98)	12.30 (17.62)	97.54 (6.14)	95.90 (7.38)	2.77 (1.01)	2.90 (0.93)	-0.43 (0.18)	-0.36 (0.47)
		Absent	2.46 (8.20)	3.28 (6.56)	77.87 (22.13)	86.07 (27.05)	2.68 (0.82)	2.61 (0.88)	0.46 (0.52)	0.40 (0.44)
		Total	**9.02 (13.00)**	**9.02 (14.00)**	**88.11 (17.00)**	**91.39 (17.00)**	**2.72 (0.88)**	**2.76 (0.85)**	**0.02 (0.36)**	**0.02 (0.34)**

PHQ-15 = Patient Health Questionnaire

Evaluation of hypothesis 2

Descriptive statistics for change in d' and c across groups and conditions are presented in Table 4.

Table 4 Median (*IQR*) for change in false alarm rate and hit rate and estimated marginal means (*SE*) for change in tactile sensitivity (d') and response criterion (c) across group and conditions

PHQ-15 group	Mood condition	Light condition	Change in false alarm rate[1] (%)	Change in hit rate[1] (%)	Change in d'[2]	Change in c[3]
Low	Neutral	Present	-4.92 (0.12)	-0.82 (8.00)	0.09 (0.17)	0.26 (0.10)
		Absent	0.00 (3.00)	-1.64 (16.00)	-0.05 (0.15)	0.04 (0.09)
		Total	**-2.32 (0.08)**	**-1.64 (9.00)**	**0.02 (0.13)**	**0.15 (0.07)**
	Negative	Present	-1.64 (18.00)	1.64 (4.00)	0.40 (0.17)	0.08 (0.10)
		Absent	0.00 (2.60)	4.92 (18.00)	0.21 (0.15)	-0.03 (0.09)
		Total	**-0.82 (9.00)**	**2.46 (12.00)**	**0.30 (0.13)**	**0.03 (0.07)**
	Total		**-1.64 (8.00)**	**0.82 (8.00)**	**0.16 (0.09)**	**0.09 (0.05)**
High	Neutral	Present	-4.10 (15.00)	-1.64 (6.00)	-0.14 (0.19)	-0.34 (0.10)
		Absent	1.64 (7.00)	4.10 (11.00)	-0.16 (0.17)	-0.02(0.11)
		Total	**-1.64 (8.00)**	**1.64 (8.00)**	**-0.15 (0.15)**	**-0.17 (0.08)**
	Negative	Present	-0.82 (11.00)	0.00 (5.00)	-0.05(0.20)	-0.04 (0.11)
		Absent	0.82 (5.00)	1.64 (25.00)	-0.08 (0.18)	-0.10 (0.10)
		Total	**-0.82 (8.00)**	**0.41 (12.00)**	**-0.07 (0.16)**	**-0.07 (0.09)**
	Total		**-1.23 (7.00)**	**0.82 (3.90)**	**-0.11 (0.11)**	**-0.12 (0.06)**

PHQ-15 = Patient Health Questionnaire
[1]Positive and negative scores indicate increasing and decreasing number of false alarms and hits, respectively.
[2]Positive and negative scores indicate increased and decreased sensitivity over time, respectively.
[3]Positive and negative scores indicate that response criterion became more and less conservative, respectively.

Response criterion (c) change. There was no effect of PHQ-15 group, $F_{(1, 78)} = 1.37$, $p = .25$, gender, $F_{(1, 78)} = 3.54$, $p = .06$, or mood condition, $F_{(1, 78)} = 1.84$, $p = .18$, and no interaction between mood and PHQ-15 group, $F_{(1, 78)} = 2.91$, $p = .09$. Following the addition of covariates, there was a main effect of DASS-21 score, $F_{(1, 73)} = 9.60$, $p = .003$, $\eta_p^2 = .12$: participants with relatively better baseline mood tended to become less conservative over time, while persons with worse baseline mood tended to become more conservative over time.

Addition of the covariates (including baseline c) also revealed an effect of PHQ-15 group, $F_{(1, 73)} = 6.18$, $p = .015$, $\eta_p^2 = .08$: adjusted means were positive and negative in the low and high groups suggesting that participants became more and less conservative over time, respectively. There was also a trend towards an interaction between PHQ-15 group and mood condition, $F_{(1, 73)} = 3.16$, $p = .08$, $\eta_p^2 = .04$ (see Figure 1). Extent of change in c across mood conditions was not significantly different for lows ($p = .14$), or highs ($p = .23$).
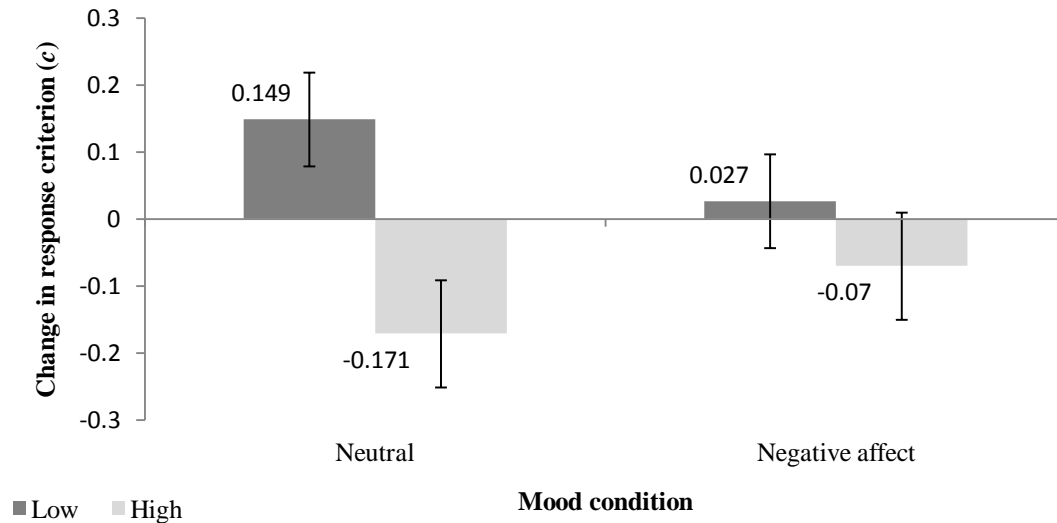
Figure 1 Estimated marginal means for response criterion change for PHQ-15 groups and mood conditions. Positive and negative scores indicate that response criterion became more and less conservative, respectively.

The trend towards an interaction between PHQ-15 group and mood condition was followed up with additional analyses. Univariate ANCOVA (controlling for the relevant covariates and baseline c) revealed significant differences between high and low symptom reporters in response criterion at T_2 in the neutral condition, $F_{(1, 34)} = 9.26$, $p = .004$, $\eta_p^2 = .21$, but not in the negative condition, $F_{(1, 33)} = .26$, $p = .61$. In the neutral condition, lows were more conservative at T_2 ($EMM = 0.28$, $SE = 0.05$) than highs ($EMM = -0.10$, $SE = 0.09$). Mann-Whitney U tests showed no differences between high and low symptom reporters in the neutral condition for change in hit rates ($Z = -1.37$, $p = .17$) or false alarms ($Z = -0.95$, $p = .34$). Repeated measures ANCOVA, with time and light as within subject factors and PHQ-15 group and gender as between subject factors, was carried out to establish whether or not c changed significantly over time in the neutral condition. There was no main effect of time, $F_{(1, 36)} = .30$, $p = .72$, or PHQ-15 group, $F_{(1, 36)} = .05$, $p = .30$, although there was an interaction between time and PHQ-15 group, $F_{(1, 36)} = 6.60$, $p = .015$, $\eta_p^2 = .15$. However, response criterion was not significantly different at T_2 compared to T_1 for either lows, $F_{(1, 15)} = 3.61$, $p = .077$, or highs, $F_{(1, 17)} = .02$, $p = .88$, in the neutral condition.

Tactile sensitivity (d') change. There were main effects of PHQ-15 group, $F_{(1, 78)} = 5.39$, $p = .02$, $\eta_p^2 = .07$, and gender, $F_{(1, 78)} = 5.02$, $p = .03$, $\eta_p^2 = .06$. Tactile sensitivity changed

differently for lows and highs over time, by increasing and decreasing, respectively. Follow-up ANCOVAs (with STAI-T and DASS-21 scores as covariates) on pre- and post- tactile sensitivity scores revealed a trend towards an interaction between PHQ-15 group and time, $F_{(1, 76)} = 3.87$, $p = .052$, $\eta_p^2 = .05$. However, highs and lows were not significantly different at either time point (p's > .38), and neither highs, $F_{(1, 39)} = 0.50$, $p = .48$, nor lows, $F_{(1, 38)} = 3.07$, $p = 0.09$, changed significantly over time.

Mann-Whitney U tests showed that there was no difference between highs and lows for extent of change in false alarm and hit rates (hits, $Z = -0.01$, $p = .99$; false alarms, $Z = -1.31$, $p = .19$). In general, men reduced in sensitivity over time whereas women showed a tendency to increase ($p = .028$)[8]. There was no main effect of mood condition, $F_{(1, 78)} = 2.45$, $p = .12$, and no interaction between PHQ-15 group and mood condition, $F_{(1, 78)} = 1.26$, $p = .27$.

Following addition of the covariates (including baseline d'), the main effects of gender, $F_{(1, 73)} = 2.96$, $p = .09$, and PHQ-15 group, $F_{(1, 73)} = 3.17$, $p = .08$, were rendered non-significant. No other main effects or interactions approached significance (all p's > .21).

Exploratory findings involving the light

Baseline. For c, there was a main effect of light, $F_{(1, 79)} = 157.8$, $p < .001$, $\eta_p^2 = .67$. Mean c values were positive and negative in the light absent and light present conditions, respectively, showing that participants were more likely to say strong in the light present condition ($M = -0.37$, $SD = 0.45$) and weak in the light absent condition ($M = 0.50$, $SD = 0.43$), $F_{(1, 79)} = 156.4$, $p < .001$, $\eta_p^2 = .67$. Wilcoxon signed-ranks tests confirmed that, in general, participants had more hits in the light present condition ($Z = -7.64$, $p < .001$; light Mdn = 95.90%, IQR = 9.82%; no light Mdn = 81.15%, IQR = 24.59%) and more false alarms ($Z = -7.74$, $p < .001$; light Mdn = 15.57%, IQR = 16.39%; no light Mdn = 2.46%, IQR = 4.91%). None of the interactions with light were significant (all p's > .18). Following the addition of covariates, the pattern of results remained largely unchanged for c.

There was no effect of light on d', $F_{(1, 79)} = 0.01$, $p = .94$, and none of the interactions with light were significant (all p's > .31). Following addition of covariates, there was a trend

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[8] Further analyses could not be carried out involving gender due to the uneven distribution of men and women across cells

towards an interaction between light and PHQ-15 group, $F_{(1, 76)} = 3.65$, $p = .06$, $\eta_p^2 = .046$, with a tendency for sensitivity to be greater for lows in the absence of the light (light absent: $EMM = 2.77$, $SE = .16$; light present: $EMM = 2.60$, $SE = .14$), but greater for highs in the presence of the light (light present: $EMM = 2.89$, $SE = .17$; light-absent: $EMM = 2.76$, $SE = .16$). However, neither of these differences reached significance: lows, $F_{(1, 79)} = 2.53$, $p = .12$; highs, $F_{(1, 79)} = 2.88$, $p = .10$. Further, d' for highs and lows was not significantly different in either the light absent, $F_{(4, 78)} = 0.00$, $p = .99$, or light present, $F_{(4, 78)} = 1.65$, $p = .20$, conditions.

Change scores. For change in c, there was a main effect of light, $F_{(1, 79)} = 10.22$, $p = .002$, $\eta_p^2 = .11$. One-sample t-tests showed that extent of change was significant in both light conditions, light present, $t_{(82)} = 2.10$, $p = .04$; light absent, $t_{(82)} = 2.26$, $p = .03$. Response criterion drifted in each condition such that participants became more likely to respond strong in the light condition over time than in the light absent condition where participants became more likely to say weak over time (light present $M = -0.37$, $SD = 0.45$; light absent $M = 0.50$, $SD = 0.43$). Following the addition of covariates, the significant main effect of light remained.

For change in d', there was no main effect of light, $F_{(1, 78)} = 0.42$, $p = .52$, and there were no significant interactions with light (all p's > .30). Results remained broadly the same following addition of the covariates (STAI-T, DASS-21, SCL change score, mood rating change score).

<div align="center">

Discussion

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The findings of the present study are largely inconsistent with our initial hypotheses, in that meaningful differences in SSDiT performance were not evident between high and low symptom reporters at baseline, and highs and lows were not differentially affected by the negative mood induction. Nevertheless, there was some indication that group differences between high and low symptom reporters on this measure of somatosensory amplification do exist, but that these may only emerge over time.

In relation to the first hypothesis, after controlling for relevant covariates, we did not find baseline differences between high and low symptom reporters for either response criterion or tactile sensitivity on the SSDiT. Although this finding appears inconsistent with previous studies showing differences on the SSDT between high and low symptom reporters for response

criterion (e.g., Brown et al., 2010), this discrepancy likely reflects important differences in the paradigms used. For the SSDT, participants have to discriminate between vibration-absent and vibration-present trials (delivered at individual perceptual thresholds), while on the SSDiT participants discriminate between different types of vibration-present trials (i.e., weak vs. strong). Thus, whereas the SSDT is thought to mimic MUS as described in the Brown model (2004), the SSDiT paradigm arguably provides a test of the somatosensory amplification concept. As such, false alarms on these two tasks may actually be attributable to different processes (i.e., activation of rogue memory representations vs. individual differences in the tendency to amplify sensations). Following this line of argument, lack of significant differences between groups on the SSDiT, together with observed SSDT differences elsewhere, suggests support for the Brown (2004) model over the somatosensory amplification one (Barsky et al., 1988). Furthermore, the lack of association between SSAS scores and objectively measured somatic amplification (observed via response criterion), together with significant relationships between the SSAS and trait NA and baseline psychopathology, calls into question the validity of existing evidence for the somatosensory amplification model that has relied on the SSAS.

With respect to the second prediction that high and low symptom reporters would respond differently to the neutral and negative mood conditions, there was a trend towards an interaction between group and condition for change in response criterion (after adjusting for relevant covariates). Although groups were comparable for c at baseline, bias to respond in a certain way was differentially affected for highs and lows over time: lows became more likely to respond weak across trials whereas highs became more likely to respond strong.

In the neutral condition, lows were significantly more conservative than highs at T_2, who showed a non-significant drift towards becoming more liberal in this condition. Further, there was a trend for lows in the neutral condition to become significantly more conservative at T_2 than at baseline, although this should be interpreted with caution as the finding approached but did not reach significance. Thus, although the absence of baseline differences according to symptom reporting group is more supportive of the Brown (2004) model, these findings tentatively suggesting group differences that may take time to become apparent may be interpreted as support for the somatosensory amplification hypothesis (Barsky et al., 1988). That

highs showed a significantly greater improvement in their mood in the neutral condition than the lows (possibly related to the fact that highs had worse mood at baseline) might have accounted for this between group difference in the neutral condition – however, controlling for change in mood did alter these findings suggesting this was not the case.

Contrary to expectation, there was no difference for response criterion between highs and lows in the negative condition nor did response criterion change significantly over time in this condition. Our favoured interpretation of this finding is that negative affect keeps response bias relatively steady over time, when it would otherwise drift. Potentially, people may regress towards their "natural" bias to respond in a certain way. However, further research with larger cell sizes, possibly looking at SSDiT performance over longer time periods, is needed to confirm this suggestion.

Furthermore, we did not find evidence for altered tactile sensitivity in high symptom reporters at baseline, which might be predicted by a number of models. Lack of evidence for enhanced or diminished sensitivity in high symptom reporters is contradictory to predictions from Ursin's (1997) sensitization theory and the signal-filter model (Rief & Barsky, 2005), respectively. As for response criterion, there was some suggestion that lows and highs sensitivity changed differently over time, although this finding was rendered non-significant after controlling for relevant covariates. Furthermore, changes in tactile sensitivity from baseline to T_2 were not significant for either lows or highs, although a slight trend for significant change was evident for lows.

Exploratory analyses suggested that the presence of a simultaneous visual stimulus (light flash) resulted in a more liberal response criterion for all participants at baseline. This was not accompanied by changes in tactile sensitivity as hit rate and false alarm rate were affected in an equivalent manner: participants responded strong more across both vibration types in the light condition, regardless of which vibration was delivered. Despite task differences, a more liberal response criterion in presence of the light is consistent with findings showing that presence of a light results in a more liberal response criteria on the SSDT task (e.g., Lloyd et al., 2008; McKenzie, Poliakoff, Brown, & Lloyd, 2010). As for the SSDT, on the SSDiT the light does

not appear to interact with symptom reporting, instead suggesting a more general multisensory facilitation effect.

Furthermore, over time participants' response criterion became more liberal on light present trials where they became more likely to report a strong vibration, compared to light absent trials where participants became more likely to report a weak vibration. It appears that participants develop an illusory correlation over time whereby they came to falsely associate the light with the strong vibration. This finding is commensurate with those of Johnson et al. (2006) who reported that response criterion shifted to become more liberal in light present trials compared to light absent ones. Although SSDT performance has been found to be relatively stable over time (McKenzie et al., 2010), the current study suggests that SSDiT performance changes over time, whereby people become more likely to "amplify" and report signal present in the context of noise from other senses.

Lack of augmented sensitivity in the light present condition is somewhat at odds with existing literature showing enhanced tactile detection during the simultaneous presentation of an additional stimulus in another sensory modality (e.g., Johnson et al., 2006; Lloyd et al., 2008), although may relate to the different nature of the SSDT and SSDiT paradigms.

In line with existing literature (e.g., Van Dillen et al., 2009), the manipulation check confirmed that the negative mood induction was successful. Following the neutral pictures, participants reported improvement in their mood and fewer symptoms. As for other literature showing worse mood in persons with increased symptom reports (De Gucht et al., 2004), mood was significantly worse in high than low symptom reporters at baseline. Although there was no difference in extent of change in the negative condition between groups, this difference from the outset may have influenced performance at T_1. Lows exhibited an increase in symptoms following the negative mood induction although highs did not; this may have been due to a ceiling effect, whereby highs were more symptomatic at baseline.

The findings of the present study should be interpreted within the context of several limitations. First, the uneven distribution of males and females across groups and conditions means that observed effects may be related to gender, which is known to be associated with physical symptom reporting and somatization (Nimnuan, Hotopf, & Wessely, 2001). Although

we adjusted for this imbalance by including gender as a between-subjects factor, it was not possible to follow-up significant effects of gender found in the present study due to the appreciable differences in cell sizes – in particular, the low number of males in the sample as a whole ($n = 22$). In addition, the high hit rate percentages across groups at baseline suggested that, in general, participants were performing close to ceiling on the SSDiT suggesting that weak vibrations were substantially above individual perceptual thresholds. As for the SSDT paradigm (Lloyd et al., 2008), future research using the SSDiT should establish individual thresholds for the weak vibration to look at amplification of more ambiguous information. Given the paucity of existing means of objectively assessing somatic amplification, it is difficult to be certain that this task offers a true analogue of somatic amplification. Given that a number of findings approached, but did not reach significance, it is possible that findings were underpowered: results for response criterion and tactile sensitivity followed a similar pattern.

Finally, it is possible that the nature of the mood induction influenced the findings in the present study. Although threat-related pictures were selected to augment the impact of the mood induction for high symptom reporters, this may have differentially affected high and low symptom reporters in the negative mood condition. However, the manipulation check suggested that lows and highs were equally affected by the stimuli. Although highs presented with worse mood at baseline than lows, there was no effect of change in mood during analyses.

In conclusion, lack of significant group differences between high and low symptom reporters on the SSDiT at baseline is inconsistent with the somatosensory amplification model. Instead, taken with findings reported elsewhere (Brown et al., 2010), this finding may be more consistent with the Brown (2004) model of MUS. Conversely however, the trend towards a change in response criterion in the neutral condition, suggests that group differences do exist in somatosensory amplification but that these only emerge over time. Contrary to expectation, evidence did not support the hypothesis that mood would further augment amplification. Rather, our findings suggest that high and low symptom reporters' response criterion drifts in opposite directions although future research is needed to establish the reliability of this effect.

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Chapter 3: Critical evaluation

Summary of main findings and theoretical implications

The present thesis aimed to explore sensory and perceptual processes relevant to medically unexplained symptoms (MUS) and to physical symptom reporting more generally. The thesis also aimed to establish whether negative affect differentially influences somatosensory amplification in nonclinical "low" and "high" symptom reporters using a novel paradigm – the somatic signal discrimination task (SSDiT).

Literature review. The narrative literature review identified a fundamental flaw in the majority of research that has attempted to investigate whether or not biological sensitization (Ursin, 1997) underpins MUS by measuring differences in perceptual thresholds. In general, such studies do not sufficiently account for psychological factors and have erroneously accepted lowered perceptual thresholds as evidence for biological sensitization. However, literature which includes measurement of psychological factors highlights their importance during somatic perception. First, consideration of response criterion (i.e., overall tendency to report signal as present) during sensory or pain perception is essential because reduced thresholds for detecting stimuli may result from a tendency or bias to report a stimulus as present rather than enhanced sensitivity (i.e., accuracy at detecting a stimulus). The majority of studies that account for this variable show that apparent differences in thresholds diminish once bias to report signal as present (regardless of whether present or not) has been controlled for. Second, identification of a growing body of literature investigating the role of expectation or anticipation of adverse physical symptoms (e.g., pain) shows that these factors activate similar neural networks as those associated with actual experience of symptom or pain. Emerging evidence suggests that this cognitive representation can be activated via information stored in semantic networks (Meerman, Verkuil, & Brosschot, 2011), which is consistent with the theory that MUS are caused by activation of "rogue" representations stored in memory (Brown, 2004). Together, these factors cast doubt over the validity of conclusions drawn from threshold-based research as neither is routinely considered. The number of studies in this area is still growing as researchers continue to pursue evidence of physiological differences between patients with functional

symptoms and healthy controls. However, by neglecting the role of psychological factors, patients are undergoing uncomfortable or painful procedures for potentially invalid experiments, which clearly has ethical implications. Future thresholding studies need to control for both potential response biases and expectancy effects to ensure that they are eliciting a true measure of perceptual sensitivity. Review of the literature also identified other important influences on somatic perception, namely bodily attention and negative affect. Due to an over-reliance on subjective measures of somatosensory amplification it is somewhat difficult to draw conclusions exactly how negative affect impacts on somatic perception.

It was also clear that models of MUS need to be revised to account for this literature and to be more specific about exactly where and how they propose the perceptual process is altered in symptom generation and maintenance. This would allow predictions to be more easily drawn out and tested, thereby facilitating differentiation of models. At present, existing models are poor at explaining how psychological and physiological factors interact during the perceptual process, and how this may be disturbed in MUS.

This review makes a significant contribution to the field by integrating theoretical models of MUS with evidence pertaining to physiological and psychological influences on the perceptual process in the context of MUS, the functional somatic syndromes and the somatoform disorders. To the trainee's knowledge, no such published review yet exists. It is hoped that this paper, once published, will encourage researchers to move away from investigating biological and psychological factors in perception independently of each other. It is also hoped that the paper will facilitate research and theory concerning how the perceptual process is altered in MUS, and thereby the content of potential clinical interventions.

Empirical paper. In the empirical paper, potential differences between high and low symptom reporters were addressed using a novel paradigm (the SSDiT) based on signal detection theory (SDT). The SSDiT is adapted from the somatic signal detection task (SSDT; Lloyd, Mason, Brown, & Poliakoff, 2008), although differs conceptually in relation to what it attempts to measure: while the SSDT is thought to mimic the experience of MUS according to the Brown (2004) model, the SSDiT attempts to experimentally replicate the process of somatosensory amplification (Barsky, Goodson, Lane, & Cleary, 1988). This is the first full

study to use this paradigm as a test of the somatosensory amplification model. An important strength of this study lies in its use of an objective paradigm to test the somatosensory amplification model, where the majority of existing studies have relied on subjective measures (i.e., the SSAS; Barsky, Wyshak, & Klerman, 1990). This study also attempted to establish the influence of negative affect on objectively measured somatic amplification.

Contrary to the main hypotheses, this study did not find evidence that high and low symptom reporters differ in somatic amplification at baseline, nor that highs and lows respond differentially to negative affect. The lack of significant group differences at baseline, in contrast to those observed on the SSDT (e.g., Brown, Brunt, Poliakoff, & Lloyd, 2010), was interpreted as support for the Brown (2004) model over the somatosensory amplification model. Alternatively, however, the validity of this paradigm as a means of measuring somatic amplification may be questioned. There was no relationship between response criterion and the subjective Somatosensory Amplification Scale (SSAS: Barsky, Wyshak, & Klerman, 1990), which was interpreted as indicating poor validity of the SSAS. However, given the paucity of available valid measures, it is difficult to establish whether or not this task provides an experimental analogue of somatic amplification. There was a difference between high and low symptom reporters in change for response criterion over time, together with a trend towards an interaction between symptom reporting group and mood condition. It appeared that lows became more conservative over time than highs and there were trend towards the low group changing over time for both response criterion and tactile sensitivity although these analyses did not reach significance. Although these results should be interpreted cautiously, one possible interpretation considered was these findings were underpowered and actually highs and lows were drifting towards their "natural" tendency to be less and more conservative in reporting signal, respectively.

Exploratory analyses involving the light suggested that the presence of an additional visual stimulus (i.e., the light) resulted in a more liberal response criterion for all participants. The light did not appear to interact with symptom reporting but instead had a more general effect on amplification. However, without further research, it is not possible to know whether this may be disturbed or altered in some way in clinical samples. Over time, participants became

more likely to report strong in the presence of the light and weak in the absence of the light as it appeared that people came to falsely associate the presence of the light with the strong vibration. This suggests that in general, people become more liberal in the presence of increased sensory information. However, this did not differentially affect tactile sensitivity.

Research limitations and difficulties encountered

Material presented in this thesis should be considered within the context of a number of limitations. First, in relation to the literature review, the search criteria that were used could potentially have biased the papers that were identified. The search was limited to studies published in peer reviewed journals and thus did not identify any treatment studies or non-published findings, which may have yielded relevant information. It is less likely that the search terms themselves resulted in the oversight of important literature as these were fairly broad. However, it is commonplace for reviews to narrow down their search so that the number of studies identified is manageable and relevant to a specific area or question; in this case, focusing on papers from peer-reviewed journals was thought likely to identify the most widely cited and theoretically important studies. Due to the non-systematic nature of the narrative approach and the large volume of studies that met the search criteria, it is possible that the write-up was biased as papers were not given blind ratings or weightings of importance from another individual. In retrospect, given the large number of studies that were based on a fundamentally flawed assumption (that perceptual thresholds directly reflect perceptual sensitivity) a systematic approach to this literature may have been redundant in any case. Although a systematic or meta-analytic review was not possible due to substantial methodological differences which prevented direct comparison of results, the trainee endeavored to make the review process replicable via detailed description of the search parameters and by inclusion of all key findings or approaches to perception that were identified. Drawing together such a disparate literature in this manner to produce this review represents a significant achievement, which no one has attempted previously.

Although the review identified the importance of expectancy effects (and a potential mediating role of attention for negative affect) these factors were not considered in the empirical study, due to the order in which the research was carried out. A brief review that was limited to

evidence considered more directly relevant to the study was initially conducted during the design process which meant that findings from this wider literature were not incorporated. However, potentially high and low symptom reporters may have arrived at the experiment with different expectations in relation to the threat-relevant nature of the mood induction which depicted scenes of bodily harm, illness, or threat. This material may have been more salient to highs than lows, thus differentially influencing affective state mood ratings at baseline. However, this is speculative as we did not investigate these factors here. In addition, we did not measure attention to the body which has been associated with altered symptom perception (e.g., Weisz, Balazs, & Adam, 1988). This process has highlighted to the trainee the importance of carrying out a thorough and broad review of existing literature prior to designing and commencing research studies in the future.

As noted in Chapter 2 there were some limitations to the study in relation to generalisability of findings. Generalisability may be limited by the recruitment strategy and sample characteristics. The sample was recruited by advertisement and respondents may have been motivated by monetary reimbursement or by earning credits to fulfill their course requirements, factors that may have increased demand characteristics during participation. Furthermore, the sample comprised undergraduate and postgraduate students likely to have a higher than average IQ with a limited age range when compared to the general population. In terms of generalisability to persons with MUS, it is questionable whether or not the cut-offs used here with the patient health questionnaire (PHQ-15; Kroenke, Spitzer, & Williams, 2002) to identify high and low symptom reporters captured a large enough difference between groups. While the study followed cut-offs suggested by Körber, Frieser, Steinbrecher, and Hiller (*in press*) to identify highs ($\geq$ 10), elsewhere a higher cut-off of 15 has been suggested (Kroenke et al., 2002). However, the present study aimed to recruit an analogue rather than a clinical sample and Körber et al. (*in press*) have shown that a cut-off of 10 has sensitivity and specificity for identifying persons with somatoform disorders in outpatient settings. Questionnaire measures also confirmed group differences (e.g., elevated trait NA, higher SSAS scores and baseline psychopathology) associated with increased physical symptom reports (e.g., Watson & Pennebaker, 1989).

In the empirical study, there was an uneven distribution of males and females across cells due to the low number of males in the high symptom reporting group, a phenomenon representative of the increased prevalence of MUS in women (e.g., by Nimnuan, Hotopf, & Wessely, 2001). While this meant that in relation to gender, the present sample was likely representative at a population level, it generated a dilemma in how to approach this difference during statistical analyses. Gender could not simply be added as a covariate in the model because it reflected a trend in the literature rather than arising from chance (Miller & Chapman, 2001). It was considered whether gender could be added as a between subjects factor, although a full factorial ANCOVA would look for interactions with gender. This was considered problematic because any interactions between mood condition and symptom reporting group would be based on only one male in the high symptom group for each mood condition. Another alternative considered was to exclude males from the analyses entirely but this would have led to dramatically different numbers across cells, necessitating random deletion of cases to even this out. This would have resulted in a loss of over half of the data collected and substantial loss of power. To resolve this issue the trainee sought additional consultation with the available statistician about how to proceed. The statistician advised that gender was taken as a factor using a *custom* model looking only at main effects where gender was concerned, which would adjust for the uneven distribution of males and females across cells. This meant that it was not possible to do further analyses in the empirical study involving gender. Similar problems with gender are present in the wider literature and a large majority of studies exclude males altogether from their samples, making it difficult to establish what underpins this difference. Thus, for gender, the results in this study are limited in a similar way to other literature in this field and it was difficult to know what the effect this had. To address this issue, future research could aim to recruit equal numbers of male and female high symptom reporters with the specific intention of looking for effects of gender.

Although results pertaining to the second hypothesis did not reach significance, *p* values were suggestive of a trend which may have been limited by power. It has been suggested that *p* values either side of level of significance (in this case < 0.05) should be interpreted in a similar manner (Altman, Gore, Garnder, & Pocock, 1983), and in the paper the trainee discussed the

potential implications of these findings. However, any findings close to the significance level should be interpreted cautiously due to the inflated risk of making a Type 1 error (i.e., falsely reporting a non-significant finding as significant).

There were a number of problems encountered during the analysis stage. First, hit rates and false alarm rates could not be normalized via transformation. Inclusion of non-parametric analyses would have made the results section unwieldy and potentially inflated the Type 1 error rate due to the high number of comparisons required. In addition, significant differences between groups in baseline mood, together with differences in extent of mood change, created a further difficulty with carrying out the analysis intended at the outset. These issues were resolved via consultation with the statistician who advised that, instead of running a repeated measures ANCOVA to look at pre- to post- changes (with time as a within subjects factor), the trainee should create d' and c change scores and use these as dependent variables to allow a mood change score to be entered as a covariate. Although change in mood ratings were non-normally distributed, the statistician advised that while ANOVA requires the dependent variables to be normally distributed, covariates do not need to be.

To keep the empirical paper accessible to readers while including the most relevant findings, considerable thought and discussion in supervision was given to weighing up different options. Potentially, this section could have been lengthier but it seemed important to be selective about what was included. This was complicated by inclusion of the light, which was retained from the original SSDT. However, given the fact that the trainee did not make specific hypotheses in relation to the light, in retrospect it is questionable of whether it was necessary to manipulate the light during this task. Furthermore, additional analysis taking block (pre- and post-) on the SSDiT as a factor could have been carried out, although block did not interact with group or condition meaning that further exploratory analysis with this variable was considered inappropriate.

The mood induction used was selected after considering the strengths and weaknesses of different stimuli that could be used to manipulate mood. One of the main dilemmas discussed concerned the content specificity of the induction. That is, should stimuli be selected that were potentially threat-relevant to high symptom reporters or should a more general mood induction

be used (e.g., autobiographical recollections; negative mood scripts; mood-inducing films). It was thought that use of a threat-relevant induction (i.e., one pertaining to physical health and symptoms) would result in a larger increase in state negative affect for high symptom reporters, although these pictures were still expected to induce low mood in low symptom reporters. The latter part of this assumption was confirmed during analyses in Chapter 2, as both high and low symptom reporters had worse mood following the mood induction. Although numerically it appeared that high symptom reporters had a larger increase in mood following the induction, this was not significant.

Further research

Both the literature review and empirical paper have generated a number of potential areas that could benefit from further research and have highlighted the importance of controlling for numerous factors during future research in this area. From the review, it was evident that future research needs to exert careful control over psychological influences on perception (e.g. response criterion and expectancy/anticipation effects).

In relation to the empirical paper, attempting to use a novel task, the SSDiT, has highlighted a number of challenges that need to be addressed in future research attempting to apply the same paradigm. It would be interesting for future research using the SSDiT to adapt the paradigm such that weak vibrations are delivered at individual perceptual thresholds – this may give a more accurate understanding of somatic amplification during interpretation of ambiguous information and would ensure that participants were not performing at or close to ceiling level. In addition, replicating the current study with a clinical sample could yield useful information about somatosensory amplification. In particular, it may reveal whether persons with more severe presentations on the symptom reporting spectrum respond to negative affect on this task in a similar way as to high symptom reporters. Furthermore, research could attempt to establish the validity of the SSDiT paradigm as an analogue of somatic amplification, possibly by looking at the relationship between SSDiT performance and other available measures of objective amplification (e.g., heartbeat detection tasks).

Personal reflection

Throughout the process of recruitment, the trainee found it somewhat uncomfortable presenting the negative affect pictures to participants to induce low mood. Two participants became distressed during the mood induction and the study was stopped according to the standard operating procedure (see Appendix 14). A record of any minor incidents and communications with the supervisor about these were stored in the site file. These occasions highlighted to the trainee the difference between the role of a clinical psychologist in research and in clinical settings. Although both involve the use of clinical skills and general empathy, in research settings, participants are referred to other sources (e.g. university counseling services) in relation to adverse reactions.

Carrying out this research was somewhat different to the trainee's previous research experiences, primarily due to the impact of time constraints and trying to manage multiple pressures involved with the training program (for example, changing placement in the midst of writing up) whereas in previous circumstances the trainee was able to solely focus on the thesis. This experience has extended the trainee's research experiences, primarily by learning more about focus on designing an experimental paradigm, compared to previous clinical research.

Learning about up-to-date literature and current controversy surrounding MUS during the review process may be useful to the trainee during future clinical work. Reviewing an area in-depth has highlighted the importance of broad reading and referring to wider aspects of available literature and critically evaluating the validity of different methodological approaches. Due to time constraints during clinical practice in the future, this will likely be from accessing existing literature reviews. From this perspective, it feels professionally fulfilling to be able to contribute to existing reviews enabled by preparation of the paper-based thesis.

It was frustrating during the review process reading consecutive articles that had made similar errors in relation to conclusions inferred about biological sensitization and to see pursuit of a physiological difference in the functional somatic syndromes where "evidence" is derived from a flawed assumption (i.e., that altered perceptual thresholds constitute evidence of biological sensitization or habituation).

In principle, preparing the thesis in accordance with a paper based format is a good idea because it helps trainees gain experience of preparing literature for publication. However, it was somewhat frustrating to be the first year group to submit a thesis in this format because of contradictory advice that was given about how the thesis should be presented.

Conclusions

In conclusion, the present research generated a number of important contributions to existing literature in this area. First, from review of existing evidence it is clear that there are a number of methodological weaknesses across literature investigating differences in sensitivity in patients with functional symptoms. Importantly, studies do not adequately control for psychological factors during their experimental manipulations, nor consider the full range of alternative explanations leading to premature conclusions that they have found evidence of sensitization. This has broader implications, particularly in clinical practice where clinicians may be referring to these studies, without the additional knowledge that symptoms may be generated or maintained via other mechanisms. From both papers, this research has provided some support for both the Brown (2004) model of MUS and Barsky's somatosensory amplification hypothesis, over Ursin's (1997) theory of biological sensitization (1988) and generated a number of areas where future research may be useful.

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APPENDIX 1: Instructions for authors

Clinical Psychology Review

The literature review in Chapter 1 was prepared for submission to Clinical Psychology Review as a standard review article. This journal was selected due to the potential implications of the material for clinical psychologists and for its relatively high impact factor (4.901). Information about the preparation of review articles to be submitted to this journal was downloaded from the journal website

http://www.elsevier.com/wps/find/journaldescription.cws_home/652/authorinstructions

The guidelines stated that:

- The article should not exceed 50 pages (including references, graphs, tables, and figures) – there is no official word limit

- Abstracts should not exceed 200 words and should state "the purpose of the research, the principal results and major conclusions." Any references cited in the abstract should be given below the abstract although authors should try to avoid this.

- Keywords should be presented following the abstract (maximum of 6 terms)

- The paper should be formatted according to "guidelines set forth in the Publication Manual of the American Psychological Association (6th ed., 2009)."

- Tables should be numbered "consecutively in accordance with their appearance in the text" with footnotes placed below the table body and indicated with superscript lowercase letters. Vertical rules should be avoided. Tables should be used sparingly and should not duplicate information described elsewhere in the text.

- References in the main text should follow guidelines from the Publication Manual of the American Psychological Association (6th ed., 2009). References in the text should be presented in full in the reference list. "References should be arranged first alphabetically and then further sorted chronologically if necessary.

- "References should be formatted with a hanging indent (i.e., the first line of each reference is flush left while the subsequent lines are indented)."

Journal of Abnormal Psychology

The empirical study (Chapter 2) was prepared for submission to Journal of Abnormal Psychology as a regular article. This journal was considered suitable for the empirical paper as it is a high quality journal (with an impact factor of 4.515) that accepts submissions from studies testing hypotheses from psychological theories "that relate to abnormal behaviour". Information about the preparation of papers for this journal was downloaded from the journal website http://www.apa.org/pubs/journals/abn/index.aspx

The guidelines stated that:

- Regular articles "typically should not exceed 9,000 words in overall length (excluding figures)."

- Referred to APA's instruction for authors: consulted APA-VI in preparation of the paper for guidance on referencing and general formatting.

- Abstracts should not exceed 250 words and should be followed by up to five keywords or brief phrases.

- References should be listed in alphabetical order and "each listed reference should be cited in text, and each text citation should be listed in the References section."

- All tables should be created using Word's Insert Table function.

APPENDIX 2: Patient Health Questionnaire (PHQ-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 ………………………………………..	☐	☐	☐

Participant Code:_____

Date of Birth:_____

Gender:_____

Handedness:_____

Have you ever had any tendency toward left-handedness?

Yes ❑ No ❑

Please indicate your preferences in the use of hands in the following activities by putting + in the appropriate column. Where the preference is so strong that you would never try to use the other hand unless absolutely forced to, put ++. If in any case you are really indifferent put + in both columns.

Some of the activities require both hands. In these cases the part of the task, or object, for which hand preference is wanted is indicated in brackets.

Please try to answer all the questions, and only leave a blank if you have no experience at all of the object or task.

	Left Hand	Right Hand
1. Writing		
2. Drawing		
3. Throwing		
4. Using Scissors		
5. Using a Toothbrush		
6. Using a Knife (without fork)		
7. Using a Spoon		
8. Using a Broom (upper hand)		
9. Striking a Match (match)		
10. Opening a Box (lid)		
i. Which foot do you prefer to kick with?		
ii. Which eye do you use when using only one?		

APPENDIX 4: Somatosensory Amplification Scale (SSAS)

Please state the degree to which the following statements are characteristic of you in general.

1 = Not At All True
2 = A Little Bit True
3 = Moderately True
4 = Quite A Bit True
5 = Extremely True

1.	When someone else coughs, it makes me cough too	1	2	3	4	5
2.	I can't stand smoke, smog, or pollutants in the air	1	2	3	4	5
3.	I am often aware of various things happening within my body	1	2	3	4	5
4.	When I bruise myself, it stays noticeable for a long time	1	2	3	4	5
5.	Sudden loud noises really bother me	1	2	3	4	5
6.	I can sometimes hear my pulse or my heartbeat 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 a splinter, really bothers me	1	2	3	4	5
10.	I have a low tolerance for pain	1	2	3	4	5

APPENDIX 5: State and Trait Anxiety Inventory – Trait Version (STAI-T)

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

1. I feel calm	1	2	3	4
2. I feel secure	1	2	3	4
3. I am tense	1	2	3	4
4. I feel strained	1	2	3	4
5. I feel at ease	1	2	3	4
6. I feel upset	1	2	3	4
7. I am presently worrying over possible misfortunes	1	2	3	4
8. I feel satisfied	1	2	3	4
9. I feel frightened	1	2	3	4
10. I feel comfortable	1	2	3	4
11. I feel self-confident	1	2	3	4
12. I feel nervous	1	2	3	4
13. I am jittery	1	2	3	4
14. I feel indecisive	1	2	3	4
15. I am relaxed	1	2	3	4
16. I feel content	1	2	3	4
17. I am worried	1	2	3	4
18. I feel confused	1	2	3	4
19. I feel steady	1	2	3	4
20. I feel pleasant	1	2	3	4

APPENDIX 6: Depression Anxiety Stress Scales – 21 item (DASS -21)

DASS21	Name:	Date:

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1	I found it hard to wind down	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I found it difficult to work up the initiative to do things	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I experienced trembling (eg, in the hands)	0	1	2	3
8	I felt that I was using a lot of nervous energy	0	1	2	3
9	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting agitated	0	1	2	3
12	I found it difficult to relax	0	1	2	3
13	I felt down-hearted and blue	0	1	2	3
14	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
15	I felt I was close to panic	0	1	2	3
16	I was unable to become enthusiastic about anything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life was meaningless	0	1	2	3

Please rate how you are feeling ***at the present moment*** for each of the items below by circling a number on each scale – please give your immediate response (gut reaction) rather than thinking about each item in great detail

ANXIOUS
Not at all anxious   Extremely anxious

| 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

DEPRESSED
Not at all depressed   Extremely depressed

| 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

DISGUST
Not at all disgusted   Extremely disgusted

| 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

APPENDIX 8: Modified version of the Symptom Checklist (SCL)

Please place a tick on each scale to indicate what you are experiencing at the present moment:

Example   Right now, at this moment, I am experiencing:

e.g. _____ _____ _____ √ _____ _____ _____
No cold hands   Cold hands

_____ _____ _____ _____ _____ _____ _____
No headache   Headache

_____ _____ _____ _____ _____ _____ _____
No watering eyes   Watering eyes

_____ _____ _____ _____ _____ _____ _____
No racing heart   Racing heart

_____ _____ _____ _____ _____ _____ _____
No congested nose   Congested nose

_____ _____ _____ _____ _____ _____ _____
No tense muscles   Tense muscles

_____ _____ _____ _____ _____ _____ _____
No upset stomach   Upset stomach

_____ _____ _____ _____ _____ _____ _____
No flushed face   Flushed face

_____ _____ _____ _____ _____ _____ _____
No sweaty hands   Sweaty hands

_____ _____ _____ _____ _____ _____ _____
No shortness of breath   Shortness of breath

_____ _____ _____ _____ _____ _____ _____
No cold hands   Cold hands

_____ _____ _____ _____ _____ _____ _____
No dizziness   Dizziness

_____ _____ _____ _____ _____ _____ _____
No ringing in ears   Ringing in ears

_____ _____ _____ _____ _____ _____ _____
No pain   Pain

_____ _____ _____ _____ _____ _____ _____
No fatigue   Fatigue

APPENDIX 9: Mean arousal and valence ratings for IAPS pictures (negative condition)

Picture No	IAPS Description	Priming text	Valence Mean (*SD*)	Arousal Mean (*SD*)	Dominance (1) Mean (*SD*)
3030	Mutilation	A man with an open wound on the side of his face	1.91 (1.56)	6.76 (2.10)	3.69 (2.10)
3051	Mutilation	A woman who has been in a car accident has cuts and bruises on her face and a broken nose covered in blood	2.30 (1.86)	5.62 (2.45)	3.92 (2.28)
3053	Burn victim	A child with life-threatening third degree burns covering their face and chest	1.31 (0.97)	6.91 (2.57)	2.33 (1.94)
3100	Burn victim	A man who has burns on his face and chest	1.60 (1.07)	6.49 (2.23)	3.00 (2.16)
3103	Injury	A man with burns and blisters on his legs from a chemical reaction is sitting in the bath	2.07 (1.27)	6.06 (2.03)	3.37 (2.00)
3140	Dead body	The body of a woman who was burnt alive	1.83 (1.17)	6.36 (1.97)	3.20 (2.17)
3150	Mutilation	A severed hand with fingers missing that is covered in blood	2.26 (1.57)	6.55 (2.20)	3.39 (2.15)
3160	Eye disease	A man with eye disease who has growths under his eyes	2.63 (1.23)	5.35 (1.79)	4.08 (1.88)
3170	Baby tumour	A baby with a cancerous tumour on one eye	1.46 (1.01)	7.21 (1.99)	2.70 (1.89)
3195	Stitches	A woman with stitches across her neck and a tube inserted into her throat to help her breathe	2.06 (1.23)	6.36 (2.25)	3.55 (2.15)
3213	Surgery	A man's thumb being cut open during an operation	2.96 (1.94)	6.82 (2.00)	3.92 (2.44)
3230	Dying man	A hospital patient with a terminal illness is struggling to breathe	2.02 (1.30)	5.41 (2.21)	2.93 (2.18)
3250	Open chest	Someone's internal organs visible during open heart surgery	3.78 (1.72)	6.29 (1.63)	4.45 (1.99)
3261	Tumour	A woman with a cancerous growth on her breast	1.82 (1.34)	5.75 (2.64)	3.57 (2.38)
9043	Teeth	A man with gum disease whose teeth have fallen out	2.52 (1.42)	5.50 (2.41)	4.29 (2.03)
9302	Toilet	Someone has been sick in a toilet and there is blood in their vomit	2.32 (1.41)	5.58 (2.43)	3.90 (2.08)
9325	Vomit	A man who had stomach pains is vomiting into the toilet	1.89 (1.23)	6.01 (2.54)	3.22 (1.96)
9405	Sliced hand	A hand that has been sliced open in two places and is covered in blood	1.83 (1.17)	6.08 (2.40)	3.40 (2.33)
9584	Dental exam	A dentist is scraping plaque from a man's gum during a dental exam	3.34 (1.57)	4.96 (2.15)	3.94 (2.41)
9592	Injection	A nurse taking blood from a man's arm with a hypodermic needle	3.34 (1.75)	5.23 (2.09)	4.14 (2.26)
Average scores			**2.26 (0.66)**	**6.07 (0.63)**	**3.55 (0.55)**

APPENDIX 10: Mean arousal and valence ratings for IAPS pictures (neutral condition)

Picture No	IAPS Description	Priming text	Valence Mean (*SD*)	Arousal Mean (*SD*)	Dominance (1) Mean (*SD*)
5500	Mushrooms	A cluster of mushrooms of varying heights are growing in the forest	5.42 (1.58)	3.00 (2.42)	6.45 (2.42)
7002	Towel	A large green bathroom towel is crumpled in a heap on a wooden surface	4.97 (0.97)	3.16 (2.00)	6.25 (1.93)
7003	Disk	A 3 ½ inch black floppy disk used to save information from a computer	5.00 (1.22)	3.07 (1.98)	6.02 (1.87)
7004	Spoon	A metal teaspoon with a pink plastic decorated handle has been left on the table	5.04 (0.60)	2.00 (1.66)	6.74 (1.99)
7006	Bowl	An empty white ceramic bowl that has just been cleaned	4.88 (0.99)	2.33 (1.67)	6.18 (1.96)
7010	Basket	An empty brown wicker basket is on the floor	4.94 (1.07)	1.76 (1.48)	6.70 (1.48)
7012	Rubberbands	A number of rubberbands of different sizes	4.98 (1.05)	3.00 (1.94)	6.06 (1.77)
7055	Lightbulb	The end of a domestic lightbulb which has a metallic screw cap fitting	4.90 (0.64)	3.02 (1.83)	5.73 (1.86)
7150	Umbrella	A blue flowered umbrella that has been opened inside to dry out after getting wet in the rain	4.72 (1.00)	2.61 (1.76)	5.55 (2.01)
7161	Pole	A large pole with a chequered black and white marble surround at the base	4.98 (1.02)	2.98 (1.99)	5.68 (2.13)
7175	Lamp	An old-fashioned table lamp with a metallic base and a large white lampshade	4.87 (1.00)	1.72 (1.26)	6.47 (2.04)
7217	Clothes Rack	A cream cardigan and two coats are hanging on a coat stand next to a window	4.82 (0.99)	2.43 (1.64)	6.25 (1.86)
7233	Plate	A white ceramic side-plate with a blue trim and swans painted on it	5.09 (1.46)	2.77 (1.92)	6.23 (2.06)
7235	Chair	A wooden dining room chair that has been placed against the wall	4.96 (1.18)	2.83 (2.00)	6.53 (2.09)
7300	Peanuts	A large pile of peanuts that need to have their shells removed before they can be eaten	5.64 (1.22)	3.25 (1.97)	6.20 (1.84)
7500	Building	A concrete building that is being supported by large pillars	5.33 (1.44)	3.26 (2.18)	5.17 (2.05)
7512	Chess	A game of chess with one player about to move a white pawn	5.28 (1.22)	3.72 (2.07)	5.84 (1.96)
7560	Freeway	Traffic driving along the freeway in the middle of the day	4.47 (1.65)	5.24 (2.03)	4.63 (2.09)
7705	Cabinet	A small storage cabinet with two drawers which have metallic handles	4.77 (1.02)	2.65 (1.88)	6.39 (2.09)
7950	Tissue	A pile of unused white tissues folded next to an open tissue box	4.94 (1.21)	2.28 (1.81)	6.30 (2.11)
Average Mean (*SD*)			5.00 (0.26)	2.86 (0.76)	6.07 (0.52)

VOLUNTEERS NEEDED

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.

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. You will also be asked to view a series of pictures depicting different scenes. Some of the pictures may depict violent or distressing scenes and some people might find this material mildly upsetting. Participants will receive £5 for taking part.

If you are interested in taking part or would like further information, please email kate.hall@postgrad.manchester.ac.uk or logon to https://www.psych-ssl.manchester.ac.uk/questionnaires/participantinformation.aspx?study_id=951

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

MANCHESTER
1824

The University
of Manchester

School of Psychological Sciences

Participant Information Sheet

Title of project:
Mood, bodily symptoms and touch detection study

Introduction
This is the first part of a two-stage study. We are interested in how mood affects people's ability to detect tactile sensations.

What will I be asked to do if I take part?
If you decide to take part, you will be asked to fill out some short online questionnaires, some of which address personal information, such as asking about your mood, and any bodily symptoms that you may have.

You may then be invited to take part in a second stage of this study. If you are invited to take part in the second stage, it is up to you to decide whether or not to continue. You will be provided with further information about what this would involve.

The online questionnaires take 15 minutes to complete, and you will receive 1 credit or be entered into a prize draw to win £50 (your choice) for taking part.

Will my data be confidential?
All information that is collected about you during the course of the research will be kept strictly confidential. Data collected about you will be stored under anonymous participant numbers. Information linking these numbers to participant names will be recorded and kept in a password-protected file (accessible only to the experimenters), for the purposes of retrieving questionnaire scores. All data collected can therefore be identified if necessary and deleted upon request.

Do I have to take part?
No. You do not have to take part in the study. If you decide to take part and then later change your mind, either before you start the study, during it or afterwards, you can withdraw without giving your reasons, and, if you wish, your data will be destroyed.

Are there any risks involved in taking part?
There are no obvious risks involved in this study.

If you are upset or concerned by any of the issues raised by this study, please contact the university counselling service at counsel.service@manchester.ac.uk or on 0161 275 2864.

Where can I obtain further information if I need it?
For further information about the study, please contact either

Kate Hall Kate.hall@postgrad.manchester.ac.uk

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This project has been approved by the

School of Psychological Sciences Research Ethics Committee

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SCHOOL OF PSYCHOLOGICAL SCIENCES

Participant Information Sheet

Title of project:
Mood, bodily symptoms and touch detection

Introduction
We are interested in how mood affects people's ability to detect tactile sensations.

What will I be asked to do if I take part?
If you decide to take part, you will be asked to fill out some questionnaires, some of which address personal information, such as asking about your mood, and any bodily symptoms that you may have.

You will then be asked to complete a short touch detection task. You will be asked to respond to painless vibrations on your index finger by pressing keys on a computer keyboard. You will be asked to indicate whether you felt a 'weak' or a 'strong' vibration. White noise will also be played via headphones to mask the noises of the vibrations. This task lasts approximately 20 minutes and is split into several blocks, with rests in between blocks if necessary.

You will also be asked to look at a series of still images on the computer. **Some of these images may depict scenes of a violent or threatening nature, for example images following a car crash, or scenes of fire or bodily harm/illness**. Each image will follow a text description of what the picture will show and will be displayed for 6 seconds. Some of the pictures will have a number on them. For those pictures with a number on you will be asked to enter the number after each picture has been displayed. **It is possible that all pictures may cause an element of distress for some people.**

Following this, you will be asked to fill out some more questionnaires about your mood.

You will then be asked to complete another short touch detection task. This task lasts approximately 20 minutes and is split into several blocks, with rests in between blocks if necessary.

The session will last for approximately 1 hour, and you will receive either £5 or 4 course credits (psychology undergraduates only) for your participation.

Will my data be confidential?

All information that is collected about you during the course of the research will be kept strictly confidential. Data collected in the experimental sessions will be stored under anonymous participant numbers. Information linking these numbers to participant names will be recorded and kept in a password-protected file (accessible only to the experimenters), for the purposes of retrieving questionnaire scores. All data collected can therefore be identified if necessary and deleted upon request.

Do I have to take part?

No. You do not have to take part in the study. If you decide to take part and then later change your mind, either before you start the study, during it or afterwards, you can withdraw without giving your reasons, and, if you wish, your data will be destroyed.

Are there any risks involved in taking part?

There are no significant risks involved in this study. However, some people may become distressed when viewing material that depicts scenes of a violent or distressing nature**. If you think that you may find this overly upsetting please do not take part in the study.**

If you are upset or concerned by any of the issues raised by this study, please contact the university counselling service at counsel.service@manchester.ac.uk or on 0161 275 2864.

Where can I obtain further information if I need it?

For further information about the study, please contact either

Kate Hall Kate.hall@postgrad.manchester.ac.uk

This project has been approved by the

School of Psychological Sciences Research Ethics Committee

Project no 573/07P	
Participant no.	

MANCHESTER 1824

The University of Manchester

SCHOOL OF PSYCHOLOGICAL SCIENCES

Consent form

Title of Project: Mood, bodily symptoms and touch detection

please delete as necessary and initial

1. Have you read the Participant Information Sheet?	**YES/NO** Initials:......
2. Have you received enough information about the study?	**YES/NO** Initials:......
3. Do you understand that you do not need to take part in the study and if you do enter you are free to withdraw:- * at any time * without having to give a reason for withdrawing * and without detriment to you?	**YES/NO** Initials:......
4. Do you agree to take part in this study?	**YES/NO** Initials:.......

Name of participant: …………..………..… **Signed:** .... **Date:** ....

Name of researcher: ………...………….. **Signed:** .... **Date:** ....

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Standard Operating Procedure for the 'Mood, bodily symptoms and touch detection study'

The mood induction for this study is designed to have an effect on participants' emotional state.

However, it is not meant to cause undue or extreme emotional distress or have a long-lasting impact on participants. This Standard Operating Procedure provides guidance for managing participants who become distressed during the 'Mood, bodily symptoms and touch detection' study.

For all participants:
The experimenter will monitor, and be alert to, participants' responses during the mood induction (neutral and anxious conditions).

The experimenter will debrief all participants at the end of the study and ask participants that they are ok to leave. The experimenter will refer to each participant's final mood state questionnaire (Likert scale) to monitor for distress.

For participants who appear uncomfortable or mildly distressed:
If participants appear noticeably uncomfortable while viewing pictures during the mood induction (e.g., turning away from the screen, covering their eyes, orally communicating distress/disgust or other similar responses) the experimenter will ask them 'are you ok to continue or would you like to stop taking part? It's completely up to you'.

If the participant replies that they are ok to continue the experimenter will monitor their distress throughout the remainder of the mood induction and study. If their distress increases – see section below 'For participants who appear moderately- extremely distressed' .

If they continue with the study, the experimenter will check that their levels of emotional distress are returning to normal by the end of the study.

If the participant withdraws their consent, the experimenter will:
- Stop the computer program immediately
- Manage their emotional distress in a calm and sensitive way
- Reassure the participant that it is completely within their rights to stop
- Apologise that the study has made them feel upset/anxious/distressed
- Normalise the participants' experience by explaining that the slides are designed to make them feel anxious and uncomfortable and reassure them that other people respond in a similar way
- Ask the participant to wait for five minutes to ensure emotional state returns to normal.
- If the experimenter is then not confident that participants are ok to leave they will follow guidelines below 'For participants who remain distressed after debriefing and five minute period'

For participants who appear moderately- extremely distressed but do not withdraw consent:
If participants become overtly distressed (agitated, crying or some other adverse emotional reaction that indicates that the participant is extremely distress) the experimenter will deem that they are too distressed to continue and automatically stop the study. At the same time they will explain that they are stopping the study but will not wait for the participant to withdraw their consent.

If the participant gets up out of their chair and leaves the room, with or without prior indication that they are emotionally distressed, the experimenter will follow them and ask them to wait for five minutes. The experimenter will:

- Manage their emotional distress in a calm and sensitive way
- Reassure the participant that it is completely within their rights to stop
- Apologise that the mood induction has made them feel upset/anxious/distressed
- Normalise the participants' experience by explaining that the slides are designed to make them feel anxious and uncomfortable and reassure them that other people respond in a similar way
- Ask the participant to wait for five minutes to ensure emotional state returns to normal.
- If the experimenter is not confident that participants are ok to leave they will follow guidelines below 'For participants who remain distressed after debriefing and five minute period'

For participants who do not appear distressed during the mood induction but disclose distress at the end of the study:
The experimenter will:

- Manage their emotional distress in a calm and sensitive way
- Apologise that the mood induction has made them feel upset/anxious/distressed
- Normalise the participants' experience by explaining that the slides are designed to make them feel anxious and uncomfortable and reassure them that other people respond in a similar way
- Ask the participant to wait for five minutes to ensure emotional state returns to normal.
- If the experimenter is not confident that participants are ok to leave they will follow guidelines below 'For participants who remain distressed after debriefing and five minute period'

For participants who remain distressed:
The experimenter will:

- Ask the participant to stay longer until they are feeling better (for up to 10 minutes)
- Provide participants with information about how to contact the University Counselling services
- Provide participants with details of how to contact CRISIS Team and/or Samaritans/ present at A&E

Payment
The experimenter will offer to pay participants that withdraw their consent to testing during the mood induction.