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Sex Differences and Gender Bias in SSD

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ABSTRACT
Hysteria originally was characterized by a cluster of physical symptoms thought to be caused by the uterus moving throughout the body. One of the most modern iterations of hysteria in the DSM-V is Somatic Symptom Disorder (SSD), a psychiatric disorder characterized by excessive psychological response to physical symptoms. Though many changes have been made over the years, SSD, like its predecessor, is still diagnosed mainly in women. This diagnostic discrepancy can be explained by a combination of medical gender bias in the treatment of women's symptoms as primarily psychological in origin and differences in the underlying neurobiological mechanisms that govern pain response. Due largely to the societal stereotype that women are more emotional and psychologically unstable than men, many medical practitioners seem to assume a certain level of exaggeration and unreliability when listening to women describe their symptoms. This is evidenced by various studies showing that doctors and nurses tend to judge women's pain as less severe than men's and prescribe women antidepressants and referrals for psychotherapy for pain and men surgery, painkillers, and physical therapy. While evidence suggests women experience pain more frequently and intensely than men, this difference seems to be due mainly to underlying neurobiological differences rather than psychological ones. Sex differences have been noted in neural immune response to pain, as well as in the roles of sex hormones in pain analgesia. These differences, however, have only been known about for the last few decades due to a deficit in women's health research. As a result, practitioners have been overly focused on proposed psychological gender differences in pain experience that are not upheld by empirical research. Future medical practice must account for the unique neurobiology of pain experience in the sexes while also evading the long held belief that women's symptoms are more psychologically based.

KEYWORDS
Pain, Somatic Symptom Disorder, Gender Bias, Sex Differences in Pain, Medical Sexism, Hysteria, Sex as a Biological Variable, Illness

INTRODUCTION

Somatic Symptom Disorder (SSD), a disorder classified by disproportionately large distressing psychological reactions to physical symptoms, is diagnosed in women much more frequently than men, with an estimated ratio of 10:1 (Kurlansik and Maffei 2016). In this paper, I present multifaceted evidence to argue that this discrepancy can be partly explained by a historical bias in the treatment of women's symptoms as psychologically-based, as well as by neurobiological sex differences in physical symptom experiences.

I begin by providing a historical analysis of SSD. Specifically, I examine how the medical meaning of "hysteria" has changed over the past 3000 years and how we can trace these changes in meaning to today's definition of SSD. This historical analysis provides evidence for a gender bias in SSD diagnosis that seems to stem from a centuries-old tendency to treat women's symptoms as psychological in origin. I give a thorough explanation of the current diagnostic criteria for the disorder, offering a brief description of the five main theories for the cause of SSD. I then explore current gender bias in medical treatment, honing in on the continuing inclination to treat women's symptoms as psychologically-based and men's symptoms as physically-based that can partially explain the diagnostic disparity in SSD.

In order to also consider the possibility that women are genuinely at higher risk for SSD, I then look at differences in the way men and women experience symptoms, especially pain. Because pain is a multifaceted phenomenon involving both psychological and biological aspects, I consider whether there are gender differences in symptom experience relating to both facets. The research suggests that though there are differences in the way men and women experience symptoms, they are mainly neurobiological rather than psychological in nature. Specifically, if there exist differences at all, I argue that they are such that women experience more physical pain than men. This contrasts strikingly with how men and women are treated for their symptoms; the research suggests that men are often treated for physical pain, whereas women are often treated as psychiatric patients.

Finally, I review how the diagnostic discrepancy in SSD, a primarily psychological disorder, acts as an example for the failures and successes in how we factor gender and sex into medical diagnosis and treatment. I consider how research into sex differences in pain experience may inform future healthcare

practice while also stressing the danger of making assumptions about pain and psychological status based on gender and gender stereotypes**.**

SSD AS A WOMAN'S ILLNESS: A HISTORICAL ANALYSIS

Many scholars attribute the creation of hysteria to Hippocrates in Ancient Greece (Gilman 1993). While Hippocrates seems to be the first individual to coin the term "hysteria" in the fifth century BC from the Greek word for uterus "hysteros", hysteria actually has origins in Ancient Egypt with the Kahun and Eber Papyrus Scrolls, dating back to 1900 BC and 1600 BC respectively. These documents did not yet assign the malady a name, but described a series of disorders with various symptoms attributed to movement of an unhappy, sick uterus, thus diagnosed exclusively in women (Tasca et al. 2012). This idea was partially born out of the sense that women were lesser beings than men and thus uniquely afflicted by certain medical problems. This definition of hysteria, that physical symptoms in women arose from unhappy, moving uteruses, remained unchanged for centuries.

The idea that women were inferior beings prone to sin and sickness flourished in the middle ages as Christianity rose to power in Europe. Hysterical women became pariahs, witches who were plagued with an illness from the devil. If a physician could not understand or find the cause to physical illness, it was assumed that the devil was responsible. As a result, the focus of treating hysteria veered from helping treat the patient's symptoms to eradicating the devilish presence. This attribution of hysteria to witchcraft remained into the sixteenth century, until the Dutch physician, Johann Weyer, theorized that hysterical "witches" were mentally ill. Although hysteria continued to be associated with the uterus (and thereby with women), Weyer's theory was highly influential in shifting views of hysteria as a mental malady (Tasca et al. 2012). Hysteria began to take shape as a mental disorder resulting in physical symptoms that was unique to women.

In the 1800s, the idea that medically unexplained symptoms were attributed to the devil faded away in favor of the idea that medically unexplained symptoms were due to some underlying psychological abnormality. Similarly, the disorder was no longer associated with movement of the uterus but instead with some problem involving the nervous system or the brain. Jean-Martin Charcot declared it to be an inheritable nervous system disorder while Robert B. Carter assigned it two primary markers or criteria, the first being that hysteria arose from too much emotion in the nervous system and the second being that once afflicted, those

with the disorder had something to gain from being sick (Crimlisk and Ron 1999). As medical understanding increased, the idea that the disorder was caused by a migratory uterus became largely obsolete, but the disorder was still diagnosed almost solely in women. This new definition of hysteria strengthened the idea that not only were women's physical symptoms often psychological in origin, but that these reported symptoms might have been exaggerated to reap some sort of benefit, whether it be in the form of financial gain or simply more attention.

A significant change in the understanding of hysteria occurred in the latter half of the nineteenth century. Sigmund Freud revolutionarily crafted the idea of "male hysteria", even diagnosing himself with the disorder. Freud suggested sexual frustration as a possible cause of the disorder and described hysteria as the transformation of these unconscious desires into physical symptoms. He defined this transformation as the patient's "primary gain" because the patient was able to ignore his or her psychological traumas in favor of treating physical symptoms (Tasca et al. 2012). Despite these changes, hysteria still remained mainly a female disorder. With the World Wars of the twentieth century came mass epidemics of hysteria, still afflicting mainly women. The psychological stress of the war was thought to result in a peak in cases of the disorder. After the wars, however, the medical use of the term "hysteria" fell out of use. Some concluded that Freud had essentially cured the world of hysteria, but in reality, it still existed, just under other names (Gilman 1993).

The term "hysteria" was ruled largely obsolete in medical practice in 1980 when it was absent from the DSM-III. It instead reappeared under a various number of names and classifications, most clearly in the form of Somatization Disorder (APA 1980). Somatization Disorder in the third and fourth editions of the DSM was characterized by a list of physical symptoms rather than psychological ones. Diagnosis was generally only made when symptoms had no known medical explanation. The fourth edition specified that patients with the disorder tended to descibe their symptoms in overexaggerated, colorful terms with an unclear symptom timeline. It warned that patients tend to seek out the advice of many doctors and may undergo unnecessary medical testing and treatment (APA 1994). Somatization Disorder was diagnosed at a much higher rate in women than men, with some physicians even arguing it to be nonexistant in men (Golding, Smith, and Kashner 1991). In other words, some practicioners still openly viewed this

new iteration of hysteria, Somatization Disorder, as solely a women's disorder, even into the 1990s.

When crafting the DSM-V section of Somatoform Disorders, the task force seemed to keep in mind the critiques of the DSM-III and DSM-IV's diagnostic criteria for Somatization disorder; that they were too specific and strict, that they seemed to emphasize an archaic dualistic approach to mind-body separation, and that these problems made physicians uncomfortable with making a diagnosis (Dimsdale and Levenson 2013; Kurlansik and Maffei 2016). The focus shifted from the symptoms of the body to the symptoms of the mind. In doing so, the actual name of the disorder changed in 2013. Somatization Disorder, Pain Disorder, and Somatoform Disorder Not Otherwise Specified were dissolved into one disorder: Somatic Symptom Disorder. (Rief & Martin 2014).

Rather than focusing on the physical symptoms themselves, the diagnostic criteria for SSD focused on the psychological impact of physical symptoms. Criterion "A", for instance, requires that only one "distressing" physical symptom exist, without specification about bodily area affected, age of onset, or whether or not it has a known medical explanation. Criterion "B" consists of three manifestations of "excessive thoughts, feelings or behaviors" relating to the symptoms. These include having thoughts about the seriousness of one's illness or conditions that are disproportionate to the actual nature of the illness, having persistently high levels of anxiety about the nature of these symptoms, and devoting excessive time and energy to the symptoms. Only one of these manifestations need be present for a diagnosis of SSD. Severity of the disorder is dependent on how many of the manifestations are present (Rief and Martin 2014). Criterion "C" specifies that although the one particular physical symptom in Criterion "A" need not be persistent, the patient must be persistently symptomatic for more than six months (APA 2013; see Appendix 1).

Interestingly, there is currently no single etiological theory of SSD that is universally agreed upon. Based on the literature, there are at least five main theories for describing the causes of SSD: somatosensory amplification, the "vicious cycle effect", alexithymia, catastrophizing, and hypervigilance. Although there is some overlap in the description of these theories (e.g. somatosensory amplification is often co-morbid with alexithymia, and its symptoms and diagnostic are very similar to those of hypervigilance) there are also clear distinctions (Wise and Mann 1994).

Somatosensory amplification and the vicious cycle effect are reliant largely on the existence of proposed biological abnormalities in SSD afflicted patients. SSD may be caused by somatosensory amplification, during which an SSD patient perceives normal sensations as more noxious than healthy patients due to overactive sensory pathways (Harvey, Stanton, and David 2006). A patient with SSD may interpret a typically innocuous experience like digestion, for example, as more intense and painful than the average person. fMRI, PET, SPECT, and structural MRI imaging techniques have pointed to possible abnormalities in a variety of nervous system and cortical structures related to somatosensory processing in patients with SSD and related disorders. These include striatal and amygdalar abnormalities, bilateral caudate-putamen hypometabolism, decreased amygdalar volume, and possible differences in the lamina 1 spinothalamic cortical pathway (Perez et al. 2015). The next theory, the "vicious cycle effect", occurs when a patient's negative mood and unpleasant physical symptoms feed off each other, causing a worsening of both. A patient may feel sick and decide to stay in bed, leading them to isolate themselves, exacerbate their depression, and exacerbate their perception of their symptoms. Often, thinking about increased symptom experiences and worsened health continues the cycle (Cooper, Booker, and Spanswick 2003). Notably, this cycle is not unique to patients with SSD but has been identified in many patients with a variety of chronic health conditions and comorbid mental illness leading to negative affect, like depression or anxiety (Gatchel 2004; Katon, Lin, and Kroenke 2007; Teasdale 1983)resulting from a paradigm shift from an outdated biomedical reductionism approach to a more comprehensive biopsychosocial model, which emphasizes the unique interactions among biological, psychological, and social factors required to better understand health and illness. This biopsychosocial perspective is important in evaluating the comorbidity of mental and physical health problems. Psychiatric and medical pathologies interface prominently in pain disorders. Important topics in the biopsychosocial approach to comorbid chronic mental and physical health disorders, focusing primarily on pain, are presented. Though this biopsychosocial model has produced dramatic advances in health psychology over the past 2 decades, important challenges to moving the field forward still remain.","ISSN":"0003-066X","shortTitle":"Comorbidity of Chronic Pain and Mental Health Disorders","language":"en","author":[{"family":"Gatchel","given":"Robert J."}],"issued":{"date-parts":[["2004",11]]}}},{"id":320,"uris":["http://zotero.org/

users/3294891/items/8KCJUDPZ"],"uri":["http://zotero.org/users/3294891/items/8KCJUDPZ"],"itemData":{"id":320,"type":"article-journal","title":"The association of depression and anxiety with medical symptom burden in patients with chronic medical illness","container-title":"General Hospital Psychiatry","page":"147-155","volume":"29","issue":"2","source":"ScienceDirect","abstract":"Background\nPrimary care patients with anxiety and depression often describe multiple physical symptoms, but no systematic review has studied the effect of anxiety and depressive comorbidity in patients with chronic medical illnesses.\nMethods\nMEDLINE databases were searched from 1966 through 2006 using the combined search terms diabetes, coronary artery disease (CAD. Though there is a lack of research into the exact nature of the vicious cycle effect in SSD, researchers have suggested that negative affect is the most accurate predictor of a patient's reported symptom severity, more telling than the actual nature of the disease (Dimsdale and Levenson 2013; Van den Bergh et al. 2017).

The last three of these theories are rooted in proposed psychological and cognitive abnormalities in the way SSD patients process their pain and emotions. Alexithymia, defined as an inability to recognize and name one's own emotions (Taylor, Bagby, & Parker 1991), may lead SSD patients to have difficulty assigning a psychological meaning to their distressing emotions. Rather than being able to say he is feeling sad and alone, for example, a patient may instead complain of back pain. When asked if he can think of emotional or mental reasons for this pain, the patient is unable to name any. Past research has bolstered this, showing that patients with SSD identify themselves as having more difficulty giving their emotions name and cause than the average person (Erkic et al. 2018). Catastrophizing is based primarily on unhealthy thought patterns and abnormal cognition. Consistent with the "excessive worry" criterion for SSD, a patient who catastrophizes may take a small problem and "blow it out of proportion", giving it much more attention and worry than necessary. For example, a patient with intermittent chest pain caused by a known benign health condition may cause themselves undue stress by attributing their chest pain to heart attacks. Interestingly, multiple studies have connected catastrophizing to a low pain tolerance, low pain thresholds, and increased somatosensory cortex activation during painful stimuli (Rief and Martin 2014). Similarly, SSD patients are thought to have an increased awareness of their bodily functions caused by constantly "checking in" on how parts of the body are feeling and functioning, leading them to become obsessive and paranoid,

often attributing normal bodily functions to sign of disease. This is known as "hypervigilance", the third etiological theory of SSD. In fact, cognitive behavioral therapy (CBT) is effective at lowering hypervigilance in SSD patients by training the patient to stop this "checking-in" behavior and become more comfortable with "abnormal" sensations of the body. (Rief and Martin 2014). The success of CBT in lessening physical symptoms, disability, and psychological distress in SSD patients suggests hypervigilance could play a major role in the disorder (Kurlansik and Maffei 2016).

GENDER BIAS IN SOMATIC SYMPTOM DISORDER

For thousands of years, hysteria, now SSD, was a woman's illness. Because its very name comes from the Greek word for "uterus," it is difficult to detach from "female." Moreover, although the name has since changed, the ideas underlying it have remained essentially the same. Patients with SSD are frequently diagnosed with comorbid personality disorders, especially histrionic personality disorder. Other proposed risk factors include childhood neglect, sexual abuse, history of substance abuse, and "chaotic lifestyle" (Kurlansik and Maffei 2016). The latter factor is reminiscent of the middle ages idea that women were plagued with hysteria as a result of sinful behavior (Tasca et al. 2012). The 1800s idea of "secondary gain," that women with hysteria had something to gain from acting sick, seems to still be alive today in the description of attention-seeking, "doctor-shopping", histrionic behavior often attributed to SSD (Crimlisk and Ron 1999; Gerger et al. 2015).

The central critique of most papers on the topic of the DSM-V creation of SSD is that the diagnostic criteria are too vague. Although the change was intended to make the criteria more inclusive to a wider variety of physical symptoms and shift the focus to psychological symptoms, many argued that the change made the criteria too "loose", allowing for false diagnoses to be made (Frances and Chapman 2013). It is estimated that one in six heart disease patients, one in six cancer patients, one in four IBS patients, and one in four chronic widespread pain patients qualify for the diagnosis based on the current criteria, with a seven-percent false positive rate in the general population (Frances 2013). Because of the subjective nature of the criteria, the personal philosophy of the physician plays one of the biggest roles in whether or not a patient with a difficult case ends up receiving a medical diagnosis or a psychiatric one (Rief and Martin 2014).

This burden falls especially strongly on women, who are frequently judged as exaggerating their symptoms and pain (Frances 2013).

Moreover, due in part to societal stereotypes of gender, there is an inclination in medicine to believe that women are more emotional and thus have a tendency to exaggerate their symptoms while men are more stoic and thus have a tendency to describe their symptoms as less severe than they really are (Hoffmann and Tarzian 2001; Keogh 2018). A 2001 review paper stated that women were less likely to be admitted to the hospital, less likely to receive anesthesia, and less likely to have follow up tests than men. While men tended to receive referrals for specialty pain clinics from their general practitioner, women tended to have to go through the extra hoop of seeing a specialist. Doctors were found to prescribe less pain medication to women than men recovering from the same surgery and of a study of 300 nurses, the vast majority thought that women were less sensitive to pain than men (Hoffmann and Tarzian 2001). A 2019 Danish study of nearly seven million patients found that women consistently had to wait longer to receive a diagnosis than men, with an average of 2.5 years later for cancer and 4.5 years later for metabolic diseases (Westergaard et al. 2019).

Virtual patient studies have demonstrated that both physicians and nurses tend to judge the patient's pain differently and prescribe different medication depending on the patient's gender (Hirsh et al. 2014; Wandner et al. 2014). Even though all the virtual patients were coded to give the exact same description of pain quality, location, effect on daily life, and duration with the same facial expressions, participants judged the female patients as having lower pain intensities than the male patients. In addition, female patients were more likely to be prescribed antidepressants for their pain while men were more likely to receive pain medication like opioids (Hirsh et al. 2014). A 2015 study involving the medical records of 589 female and 262 male chronic pain patients found that women were significantly less likely than men to receive recommendations for further rehabilitation and medical testing. Men were more likely to receive surgery and physical therapy while women were more likely to receive medications and psychotherapy (Stålnacke et al. 2015).

Together, this evidence suggests the presence of a bias in how medical practitioners treat women's symptoms. While women's pain is often judged to be psychological in origin, men's pain is more frequently thought to be physical in origin. This tendency, in combination with the deeply gendered history of

SSD, indicates that the diagnostic discrepancy in SSD can be at least partially explained by a gender bias. Should there be differences in the way men and women experience symptoms, however, perhaps the bias is not a bias at all but a warranted difference in the way practitioners diagnose different genders. Because pain has psychological and physical aspects, it is important to look at the possibility of gender and sex differences in both facets, especially those differences relevant to the five proposed causes of SSD as mentioned earlier.

SEX-BASED NEUROBIOLOGICAL DIFFERENCES IN SYMPTOM EXPERIENCE

Many studies point to the fact that women seem to experience and report more physical symptoms with more severity than men overall (Fillingim et al. 2009; Hoffmann and Tarzian 2001; Keogh 2018; Ramírez-Maestre and Esteve 2014; Unruh 1996). Women also experience anxiety and depression more frequently than men, which could possibly act as a confounding variable to explain this difference in physical symptoms (Klonoff, Landrine, and Campbell 2000). However, even when comorbid mental illness is controlled for, women have been found to report an average of 1.1 more physical symptoms than men, regardless of if these symptoms have a known medical cause of not (Kroenke and Spitzer 1998). Despite the fact that women have a longer life span than men in the United States, they are significantly more prone to autoimmune diseases and other chronic conditions, especially chronic pain, than men and utilize healthcare services more frequently than men. 78% of autoimmune disease sufferers, including those afflicted by Multiple Sclerosis, Lupus, Rheumatoid Arthritis, and more, are women (Fairweather, Frisancho-Kiss, and Rose 2008). Women are effectively the "sicker sex"(Turabian 2017).

In addition to experiencing more pain in everyday life, laboratory studies have consistently shown women to have significantly lower pain thresholds and tolerances than men (Fillingim et al. 2009; Hoffmann and Tarzian 2001; Ramírez-Maestre and Esteve 2014; Unruh 1996). In addition, researchers have observed that women experience more activation in the contralateral prefrontal cortex, contralateral insula, and thalamus in response to painful stimuli than men (Hoffmann and Tarzian 2001). These studies have also pointed to a wide variety of possible neurobiological differences in how women and men experience symptoms. Firstly, estrogen seems to play a major role in both mechanisms of analgesia involving

opioid receptors and inflammatory response to pain. A woman's response and tolerance to ischemic pain changes throughout her menstrual cycle as her estrogen spikes and drops (Iacovides, Avidon, and Baker 2015). Overall low levels of estrogen have been linked to osteoporosis, tempromanidibular joint disorder, and other joint pain disorders (Hoffmann and Tarzian 2001). Similarly, rapid drops in estrogen levels have been associated with increased symptom severity in patients with Rheumatoid Arthritis (Sorge and Totsch 2017).

Estrogen-dependent analgesic mechanisms have been found in female mice, which may explain why pain tolerance shifts alongside hormones (Hoffmann and Tarzian 2001). Estrogen has even been found to have an effect on externally administered opioids. The hormone reduces available opioid binding sites on the cell membrane and can "uncouple" morphine binding, making it less effective (Averitt et al. 2018). Interestingly, progesterone seems to act as a counterpart to estrogen, possibly working as a therapeutic agent to neuropathic pain. Rat studies have shown progesterone administration to lessen the harmful electrophysiological changes in peripheral nerves relating to peripheral neuropathy and decrease the quantity and severity of neuropathic pain related behaviors (Coronel et al. 2016; Jarahi et al. 2014).

In addition to estrogen and progesterone mediated differences in pain experience, research has pointed to possible sex-based differences in parts of the PNS and CNS devoted to pain experience. Researchers have observed differences in tissue thickness and sensory receptor count in the peripheral nervous system of men and women, suggesting women may have lower pain thresholds and tolerances because they are actually sensing larger quantities of painful stimuli than men (Fillingim and Maixner 1995). A 2016 study found that immune response in the spinal cord, the over-activity of which is often associated with chronic pain, is primarily mediated by T-cells in female rats and microglia in male rats (Mapplebeck, Beggs, and Salter 2016). This difference may be associated with the fact that male rats in the study were able to recover from spinal cord injury much faster than female rats. Sex-based differences in immune response have also been observed in the rodent peripheral nervous system (Sorge and Totsch 2017). A 2019 study involving human dorsal root ganglion neurons found evidence for the existence of sex-differential gene expression that could possibly be related to sex-specific neuropathic pain (North et al. 2019). The female subjects in this study had a different set of spinal-cord injury related gene expressions when compared to the

male subjects that may have resulted in differing pain experiences. These studies provide strong evidence not only for disparities in the amount and strength of pain experienced by men and women but also for numerous notable differences in the underlying biological mechanisms of pain sensation and perception between the sexes.

Somatosensory amplification, the idea that SSD is caused by the patient perceiving normal sensations as more intense than the average person, is based primarily in proposed neurobiological irregularities. There is little research into the question of whether the specific neural differences thought to be related to somatosensory amplification, such as striatal and amygdalar abnormalities and differences in the lamina 1 spinothalamic cortical pathway, are more common in women or men. There is evidence, however, that women generally experience pain more strongly than men and that this may be a result of sex hormone modulation of pain sensation and increased activation of pain-responsive areas in the brain (Hoffmann and Tarzian 2001). In this sense, this is evidence for natural somatosensory amplification of pain in most women as compared to men, independent of SSD.

Given that women exhibit both higher levels of chronic and everyday pain and higher prevalence of mood and anxiety disorders, it is not illogical to infer that women may be more prone to falling into the vicious cycle effect, another etiological theory of SSD in which a negative psychological mindset feeds into the worsening of physical symptoms and vice versa, than men (Riecher-Rössler 2017). These factors being considered, it is quite possible that women genuinely exhibit the symptoms of SSD more often than men. It is important to ask, however, how a person with comorbid depression and chronic illness would differ in presentation to a person with SSD. In addition, it is important to ask how a psychiatrist would differentiate between these two situations and how they would differently inform treatment. Might the gender of the patient determine their diagnosis?

GENDER-BASED PSYCHOSOCIAL DIFFERENCES IN SYMPTOM EXPERIENCE

Many of the proposed psychological differences between how women and men experience pain are reliant on gender roles and stereotypes. The social constructs of "man" and "woman" have vast and complex meanings that may vary across cultures, but generally, men are made to feel ashamed of their feelings, including

pain, while women are encouraged to be more vocal due to a community-based perception of the world (Keogh 2018; Kroenke and Spitzer 1998). As a result, men tend to suppress their feelings of pain and wait until they interfere significantly with work and daily life to seek medical attention and support. Women, meanwhile, are generally more socially and emotionally oriented, leading them to more readily seek support (Hoffmann and Tarzian 2001). In addition, there seems to be a societal assumption that pain is inevitably a bigger part of a woman's life than a man's due to childbirth and menstruation (Keogh 2018). As a result of these stereotypical differences, men may feel a pressure to hide their pain, causing them to falsely report lower pain tolerances and thresholds and ignore possible chronic pain conditions. Despite these assumed stereotypical differences in the way men and women experience symptoms psychologically, studies have shown mixed evidence as to whether or not these differences are upheld by empirical results.

For example, alexithymia, the theory that individuals with SSD are unable to name their emotions, is found to be more common in men than women (Unruh, Ritchie, and Merskey 1999). In a study of 2018 depressed patients, 891 male and 1127 female, male patients were significantly more likely to experience alexithymia than female patients. Among these patients with depression, 12.8% of men and 8.2% of women demonstrated alexithymia (Berger et al. 2005). Other studies have also shown alexithymia to be more common in men than women (Guvensel et al. 2018; O'Loughlin et al. 2018). Perhaps due to male gender role expectations that discourage men from showing and seeking help for their emotions, men are more prone to alexithymia than women, especially men who feel insecure in their masculinity (Berger et al. 2005). This evidence suggests alexithymia likely cannot explain the gender diagnostic discrepancy in SSD.

Catastrophizing, another proposed cause of SSD in which patients are thought to assign excessive worry to minor physical symptoms, has long been used as a theory to explain why women have lower pain tolerances than men but there is mixed evidence as to whether or not this is rooted in empirical evidence. Proponents of this view have hypothesized that women are especially prone to this cognitive dysfunction due to increased emotional vulnerability (Roth et al. 2005; Thorn et al. 2004). A 2004 laboratory study on this topic provided evidence that women may indeed experience catastrophizing more frequently than men. Even when comorbid mental illness was controlled for, women reported both more

pain and more catastrophizing than men during a thermal pain task. (Edwards et al. 2004). Other laboratory tests and field studies involving chronic pain patients have also shown women to have higher rates of catastophizing and poorer coping skills than men (Leung 2012).

A 2014 field study, meanwhile, found that men and women with the same chronic pain conditions reported no significant difference in catastrophizing mental behaviors when questioned on their coping strategies (Ramírez-Maestre and Esteve 2014). Multiple studies involving recovering whiplash patients actually found male patients to have higher levels of pain catastrophizing than female patients (Elklit and Jones 2006; Rivest et al. 2010). A 1999 field study found there to be no gender difference in emotional upset due to pain but simply an increase in the use of coping strategies in women (Unruh, Ritchie, and Merskey 1999). Based on this mixed evidence, it is unclear whether catastrophizing can account for the gender disparity in the diagnosis of SSD.

Like catastrophizing, hypervigilance, which occurs when SSD patients are overly aware of their own bodily sensations, has also been proposed as a possible model to explain why women seem to experience more pain with higher intensity. A 2003 theoretical review paper argued that hypervigilance, can be expressed through increased treatment seeking for pain, low expectations about one's own pain tolerance, and actual low pain tolerance (Rollman et al. 2004). According to this definition, because women have higher levels of pain treatment seeking and lower pain tolerances, they would overall have higher levels of hypervigilance than men. The researchers attribute this difference to a tendency for women to assign exaggerated meaning to their previous pain experiences (Rollman et al. 2004). Despite this theorization, however, there is little evidence that women actually have higher levels of hypervigilance or that low pain tolerance is a sign of hypervigilant mental behaviors. A 2014 study involving chronic pain patients found that men and women reported no significant difference in hypervigilant mental behaviors when questioned on their coping strategies. Women did, however, report higher levels of pain intensity, impaired daily functioning, and pain anxiety. This difference may also be related to overall higher levels of comorbid depression and anxiety in women and thus higher levels of negative affect (Ramírez-Maestre and Esteve 2014). Overall, there is not enough research around hypervigilance and gender to determine whether or not there is a correlation.

Although it is difficult to completely divorce the psychological aspects of pain from the physical ones, attempts to provide evidence for definite psychological differences in the way men and women experience pain have mostly been unsuccessful. More specifically, there is a lack of evidence that women's pain is in any way more psychologically influenced or oriented than men's. This includes studies aimed at examining differences in the way men and women process pain specifically related to the etiological theories of SSD, including alexithymia, catastrophizing, and hypervigilance. This is not to say that there are no psychological differences in the way men and women experience symptoms whatsoever, but that there is a lack of evidence for gender differences in the way men and women psychologically experience symptoms in relation to SSD.

CONCLUSIONS AND FUTURE DIRECTIONS

Though researchers have attempted to explain the differences in pain intensity and tolerance in men and women through cognitive differences (e.g. catastrophizing or hypervigilance), there is mixed evidence for these theories. A perhaps stronger explanation is the neurobiological differences in pain experience between men and women. The focus in research on attempting to find cognitive and psychological differences between men and women has detracted attention from the existence of neurobiological differences. As a result, differences in medical diagnosis and treatment have been based too strongly on theorized disparities in the way men and women think and process pain, many of which are at least partially based in unfounded gender stereotypes, and not strongly enough on differing underlying biological mechanisms of pain sensation and perception. The gender disparity in the diagnosis of SSD is a clear example of this.

Although women are the primary consumers of healthcare, they are not the primary target of healthcare research. Even in the second half of the twentieth century, the focus of medical research was men, specifically white, upper-class, older men. In 1993, legislation was created that mandated the inclusion of women and minorities in NIH research (Hoffmann and Tarzian 2001). This legislation was last amended in 2017 to better clarify what kind of studies were subject to the requirement. While this legislation is an important step forward, it has only existed for the last 20 years, suggesting there is a large gap in research between that focusing on white men's health and that focusing on everyone else's. Researchers have really only begun to identify that there could be differences

between women's and men's healthcare needs other than gynecological aspects in the last few decades. For example, in 2016, the NIH created a policy that all applicants applying for funding for studies using invertebrate animals must explain how their experiment will account for sex as biological variable. The reasoning for this policy stems from the general exclusion of female subjects in past animal research and the resulting lack of generalizability. This bias has been found to be especially prominent in neuroscience and pharmacology research. (Shansky and Woolley 2016)we present reasons to be optimistic that this new policy will be valuable for neuroscience, and we suggest some ways for neuroscientists to think about incorporating sex as a variable in their research.","DOI":"10.1523/JNEUROSCI.1390-16.2016","ISSN":"0270-6474, 1529-2401","note":"PMID: 27881768","journalAbbreviation":"J. Neurosci.","language":"en","author":[{"family":"Shansky","given":"Rebecca M."},{"family":"Woolley","given":"Catherine S."}],"issued":{"date-parts":[["2016",11,23]]},"PMID":"27881768"}],"schema":"https://github.com/citation-style-language/schema/raw/master/csl-citation.json"} Scientific research still has a long way to go to make up for these deficiencies. With these policies in place, hopefully future innovations in science will produce sex-specific pain therapies that account for neurobiological differences like sex hormones and neural mediation of immune response.

Perhaps more important than accounting for the newfound neurobiological differences in the way men and women experience symptoms is eradicating the centuries-old bias in treating women's symptoms as primarily psychological in origin. There is little to no evidence showing that women's pain is actually more often psychologically-based and influenced than men's pain is. More likely is that women generally experience more pain than men, and because the medical model has been historically based on men with the flawed assumption that women are essentially biologically identical, practitioners assumed the reported differences in pain severity and frequency were due to psychological differences rather than physical ones. As a result of this assumption, there is an ongoing tendency to treat women's pain reporting as exaggerated, influenced by supposed cognitive dysfunctions like catastrophizing and hypervigilance. This has led to an inevitable mistreatment of women's symptoms and pains as psychological in origin, a mistreatment that has lasted centuries. Future medical practitioners must work to undo this bias and treat women's pain as no less psychologically influenced than

men's while also working towards developing sex-specific pain treatment that will account for underlying neurobiological differences.

Pain is a multifaceted phenomenon involving both psychological and physical aspects, yet pain treatment and diagnosis tend to focus mainly on the psychological aspect in women and the physical aspect in men. As a result, individuals are facing misdiagnoses that can have much larger ramifications, including being denied access for necessary care. As described earlier, women must wait longer for a diagnosis than men and tend to be referred to specialists less often, instead receiving psychiatric treatment. Because it takes longer for women to get viable answers and treatment for their symptoms, they likely face a worsening of symptoms and possible escalation of underlying disease in the waiting period. This leads to larger healthcare costs in the long run and an overall worse quality of life. On the other hand, men who suffer from psychiatric disorders likely are not easily getting the treatment or diagnosis they require either. Pain and symptom treatment should be multifaceted, covering both the underlying disease and biology and the psychological distress that may arise. The increased specialization of healthcare means that patients are too often receiving care only for either the psychological aspect, affecting especially women, or the biological one, affecting especially men. In this way, the current Western medical system is failing to adequately treat both women and men.

APPENDIX:

1. The official 2013 DSM-V Criteria for Somatic Symptom Disorder are as follows:
    - A. One or more somatic symptoms that are distressing or result in significant disruption of daily life.
    - B. Excessive thoughts, feelings, or behaviors related to the somatic symptoms or associated health concerns as manifested by at least one of the following:
        1. Disproportionate and persistent thoughts about the seriousness of one's symptoms.
        2. Persistently high level of anxiety about health of symptoms.
        3. Excessive time and energy devoted to these symptoms or health concerns

C. Although any one somatic symptom may not be persistent, the state of being symptomatic is persistent (more than 6 months)

Specify if:

With predominant pain (previously pain disorder): This specifier is for individuals whose somatic symptoms predominantly involve pain.

Specify if:

Persistent: A persistent course is characterized by severe symptoms, marked impairment, and long duration (more than 6 months).

Specify current severity:

Mild: Only one of the symptoms specified in Criterion B is fulfilled.

Moderate: Two or more of the symptoms specified in Criterion B are fulfilled

Severe: Two or more of the symptoms specified in Criterion B are fulfilled, plus there are multiple somatic complaints (or one very severe somatic symptom). (APA 2013)

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