A Psychobiological Model for the Neurological Symptoms in Somatic Symptom Disorder

Yutong Xu
Keystone Academy, Beijing 101318, China

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Abstract: Current research lacks in synthesis of psychological and biological approaches when studying the mechanism of somatic symptom disorder (SSD), which impedes the development of effective treatment intervention. Primarily due to the conflicting perspectives of the two fields. This paper derived a model that combines both psychological and biological factors by analyzing literature about factors causing development of SSD. Three neurological symptoms of SSD were analyzed in this paper (headache, dizziness, and weakness) using 19 pieces of studies. The result shows that the development of SSD follows the path of external stimuli – physiological change – cognitive processing – response – recurrence. Based on the result, a psychobiological model of SSD development is established. It will help to find a more precise target for SSD treatment.

1 INTRODUCTION

Although modern medicine has made remarkable progress in curing diseases over the past century, there is still a range of symptoms that cannot be fully resolved. Somatic symptom disorder (hereinafter referred to as SSD) is a typical category. When patients report discomfort, but a medical inspection cannot determine its cause, the complaint may be ascribed to the psychological status of patients and diagnosed as SSD. Terms such as somatization, somatization disorder, somatoform disorder, and somatization syndrome, have all been used in early studies to describe SSD. Currently, SSD is defined as "distressing somatic symptoms plus abnormal thoughts, feelings, and behaviors in response to these symptoms" (American Psychiatric Association, 2013). A diagnosis of other medical conditions might or might not be present for patients with SSD. It is believed that SSD is caused by the interaction of biological and psychological factors, but the specific mechanism behind SSD still needs more understandings due to the miscellaneous physical expressions and interlaced causes of SSDs.

The diagnostic criteria of SSD in DSM-V are shown in Table 1. It was improved from the criteria in DSM-IV, which attributes SSD mainly to the psychological factors and suggests that somatic symptoms of patients are illusions caused by their mental disorder since their somatic discomfort lacks medical explanation. According to DSM-V, the patient's self-reported somatic distress is real, regardless of the presence or absence of medical explanation. Moreover, diagnosis of SSD does not conflict with other medical diagnoses – in fact, SSD is usually accompanied by other medical conditions (American Psychiatric Association, 2013).

Table 1: Diagnostic Criteria of Somatic Symptom Disorder in DSM-V (American Psychiatric Association, 2013).

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 or symptoms. 3. Excessive time and energy devoted to these symptoms or health concerns.
C. Although any one somatic symptom may not be continuously present, the state of being symptomatic is persistent (typically more than 6 months).

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Common symptoms of SSD may be categorized into 1) pain, 2) neurological symptoms, 3) digestive symptoms, 4) sexual symptoms (Cleveland Clinic, 2018). Pain has been the most commonly reported symptom, hence most thoroughly studied by previous researchers. This paper would therefore discuss the neurological symptoms of SSD. Neurological symptoms of SSD include headaches, weakness, dizziness, and abnormal movements (Cleveland Clinic, 2018). Compared to localized pain, these symptoms are non-specific, which means the causes of these symptoms can be associated with a greater variety of factors.

As mentioned before, SSD is undeterminable by objective inspections, psychiatrists have to rely on self-reported experiences of patients, which makes diagnosis and treatment of SSD problematic. Due to the complicated nature of SSD, we need to have a comprehensive understanding of its causes, so that a better treatment/prevention of SSD could be developed. Since SSD is a combination of mental disorders and medical conditions, it is of concern to both psychiatrists and physicians. However, because of the discrepancies between professions, previous researchers have been using different approaches to study the causes of SSD – psychological and biological – which are sometimes disagreeing.

Researchers using psychological approaches explain the cause of SSD mainly by patients' mental processing of information and stimuli. Eifert, Lejuez and Bouman argued that SSD is caused by individuals' belief about the threatening outcomes of physiological changes, which is strongly related to their past learning, including misinterpretation of medical information, past experiences, perception of illness, etc (Eifert, 1998). Witthoft and Hiller added up to that opinion, suggesting that SSD is caused by individuals' excessive focus on bodily sensations and exaggerated illness outcomes and that excessive focus is primarily due to their neuroticism and suggestibility (Witthöft, 2010).

Researchers using biological approaches explain the cause of SSD mainly by patient's physiological changes. Rief and Barsky proposed a filter system, in which they argued that SSD is strongly influenced by biological factors such as physiological arousal, endocrine imbalance, neurotransmitter disorder, etc (Rief, 2005). The individual's selective attention and pre-existing mental disorder either made them ignore the primary physiological signal or over-amplify the signal, thereby causing the symptoms in SSD. Dimsdale and Dantzer argued more radically, suggesting that SSD is probably a misdiagnosis due to ignorance of patients' history, an unrecognized disease, or misdiagnosis due to not using modern diagnostic technology (Dimsdale, 2007).

The disagreement between psychological and biological approaches is not beneficial for the study of SSD because a single-factor model is not enough to induce the developmental path of SSD, which may have been allowed more precise and effective treatment interventions. The modern behavioral medicine approach considered both psychological and biological factors in the mechanism of SSD by indicating a mutually reinforcing relationship between physiological disturbance and emotional arousal, but the focus was on treatment intervention (Looper, 2002). The model needs more details on the mechanism of SSD, as well as more specific connections to symptoms.

One integrated model was created by Flor, Birbaumar, and Turk in 1990 for the development of chronic pain. According to their model, the cause of chronic pain can be divided into four stages (Flor, 1990):

1. Predisposing factor including genetic defect, previous social learning, trauma, etc;
2. Precipitating stimuli, which are external and internal stimuli that arouse discomfort feeling;
3. Precipitating responses, referring to the over-perception of physical symptoms or inadequate perception of internal stimuli;
4. Maintaining processes, which are the conditional learning of pain-related fear and physiological responses as a result of conditional learning.

Researchers then suggested: recurring stress and pain episode leads to increased muscle tension, which leads to insufficient blood and oxygen in affected muscles, consequently releasing pain-related substances, finally resulting in muscular and sympathetic hyperactivity, thereby forming a vicious cycle (Flor, 1990).

In this paper, an integrated model for the development of neurological symptoms in SSD is established based on literature analysis. This paper will start by reviewing theories with empirical supports from both psychological and biological perspectives, then identify major factors that play a role in the development of each symptom. Finally, a general developmental path of neurological symptoms of SSD will be drawn.

2 METHOD

Literature referenced in this paper is drawn from three databases (PubMed, UpToDate, and NCBI) by searching "somatization cause", "somatoform cause", "somatic symptom cause", "chronic headache", "muscle weakness", and "chronic dizziness". Papers are excluded if: (1) irrelated with the cause of SSD; (2) individual case without other empirical supports; (3) did not refer to any of the three neurological symptoms; (4) is specific to a group of patients; (5) published before 2000. The potential factors causing the three symptoms are extracted from selected literature and explained in the following section.

3 RESULTS

3.1 Chronic Headache

3.1.1 Pre-Existing Health Issue

Headache is one symptom of anxiety. As patients with anxiety demonstrate stress responses and are more vulnerable to external stimuli. Lindsay Allet and Rachel Allet stated that anxiety level is widely recognized in relation to headaches (Allet, 2006). Shim, Aram Park, and Sung-Pa Park did a statistical analysis to determine whether alexithymia, somatization, anxiety and depression are factors causing chronic headaches (Shim, 2018). They found that these factors are statistically significant in effect on headache patients. Anderson, Maes, and Berk stated that somatic symptoms such as pain and muscular tension, are major comorbidities of depression (Anderson, 2012).

3.1.2 Cognitive Processing

Shim, Aram Park, and Sung-Pa Park found that people with alexithymia are more likely to be associated with tension-induced chronic headaches (Shim, 2018). Since alexithymia patients are more self-affective (Saito, 2016) and hence pay more focus on their own feeling, a more sensitive perception toward physical change may be expected, thereby leading to a more frequent report of headache. Cappucci and Simons measured anxiety sensitivity by patients' self-report and discovered that SSD patients have high anxiety sensitivity (Cappucci, 2014). They hypothesized a model that anxiety sensitivity leads to fear of pain and consequently a stronger experience of pain-related disability.

3.1.3 External Stimuli

Anderson, Maes, and Berk found that cortisol release under stress situations may increase mu-opioid transcription and affect the tryptophan pathway, tryptophan depletion causes photophobia and headache (Anderson,, 2012). Espinosa Jovel and Mejia suggested a causal relationship between individual hyperexcitability and caffeine intake (Espinosa, 2017). Moreover, excessive caffeine intake can result in depression and headaches. Another group of researchers presented a series of SSD cases after HPV vaccine injections, which are speculated to be caused by a sympathetic nervous system dysfunction stimulated by HPV injection in the susceptible population (Palmieri, 2016). Although this phenomenon was observed worldwide, data is still insufficient to draw a causal relationship between HPV vaccination and SSD.

3.2 Muscular Weakness (Functional Weakness)

3.2.1 Pre-Existing Health Issue

Shangguan et al. investigated people who developed anxiety disorder during the Covid pandemic and found a statistically significant correlation between stress and anxiety level, and subjective feeling of muscular weakness (Shangguan, 2020). Chaturvedi, Maguire, and Somashekar studied SSD in patients with cancer. Tiredness, exhaustion, and weakness are frequently reported. The researchers suggested SSDs may be the symptom of depression in cancer patients or side effects of radiation treatment (Chaturvedi, 2006). They also found an association between liver metastasis (tumor) and weakness. Liver dysfunction seems to be generally associated with energy levels (Swain, 2006).

3.2.2 Individual Condition

Shangguan et al. also discovered in their study that females have a stronger statistical association with subjective somatic symptoms (Shangguan, 2020). Stone, Warlow, and Sharpe discovered female gender dominance in cases of muscular weakness (Stone, 2010).

3.2.3 Cognitive Processing

Stone, Warlow, and Sharpe found patients with functional weakness tend to think their symptom is "a mystery" and believe that their symptom is physiological instead of psychological (Stone, 2010). Observation of medical management of patients with functional weakness done by Stone and Carson shows an improved somatic condition of patients after changing thinking processes from "the symptom is devastating" to "the symptom is curable." (Stone, 2011) The fact that change in cognitive processes can relieve a patient's condition shows an important role played by individuals' cognition in SSD.

3.2.4 External Stimuli

Jotwani and Turnbull introduced some cases suggesting central neuraxial anesthesia may cause postoperative weakness because some GABA-containing sedatives may inhibit certain neural pathways and lead to the symptoms (Jotwani, 2020). However, the author mentioned data is insufficient to draw a conclusion. They also added, in their specific case, the patient seemed to exhibit an unconscious stress response, which could result in her somatic symptom. Lack of vitamin-D intake is also related to muscle weakness (Dawson-Hughes, 2017) and this argument was backed up by an experiment done on mice, which discovers muscle weakness of mice with vitamin-D receptor removed (Girgis, 2019).

3.3 Chronic Dizziness

3.3.1 Pre-Existing Health Issue

Gupta reviewed past studies on the relationship between post-traumatic stress disorder (PTSD) and chronic dizziness (Gupta, 2013). It was concluded by previous studies that one of the features of PTSD is sleepiness and partial consciousness, which may be perceived by individuals as dizziness (Gupta, 2013). Staab stated a relationship between chronic dizziness and traumatic brain injury. He introduced that many patients who had undergone traumatic brain injury reported "subjective dizziness, imbalance, hypersensitivity to motion cues." (Staab, 2006) Cortese et al. analyzed the DNA of 95 patients with neurological symptoms of imbalance and dizziness. They discovered a disordered replication of a DNA unit named RFC1, leading to ataxia, which causes the symptoms of dizziness (Cortese, 2020). The patients usually report neurological symptoms at the sixth year of the onset of ataxia (Cortese, 2020). Hence, when investigating SSD, patients' history has to be taken into account.

3.3.2 External Stimuli

Buzhdygan et al. studied the effect of SARS-CoV-2 spike protein on patients' brain activity and found that this spike protein can affect the blood-brain barrier function, causing inflammation in endothelium and leading to neurological symptoms including chronic dizziness (Buzhdygan, 2020). Fang et al. analyzed blood samples of patients with chronic dizziness and discovered a significantly high level of oxidative stress parameters and emotional stress-related neurotransmitters (Fang, 2020). They suggested that the redox system in SSD patients may be impaired.

4 DISCUSSION

It seems that in all three neurological symptoms of SSD, the cause, or mechanism, is generally the same and are interrelated. Four significant factors had been identified: pre-existing health condition, external stimuli, cognitive processing, and individual condition.

4.1 Pre-Existing Condition

Most studies addressed depression and anxiety as the cause of individuals' strong perception of physical symptoms. Patients with depression disorder are featured in decreased interest toward the external environment and lowered self-esteem (Özen, 2010). Decreased attention to external stimuli means a higher level of attention on senses of oneself, therefore depressive individuals are more likely to experience and exaggerate somatic symptoms. Lowered self-esteem means an expectancy of negative experiences, hence negative physiological changes are more evident for depressive patients. Moreover, some depressive patients feel they need more social attention and caring. When they associate others' attention with their report of discomfort, reporting SSD would become a learned behavior. Individuals with anxiety tend to exhibit unnecessary thoughts and associate negative consequences with somatic symptoms (over-interpretation). They were found to be more often associated with hypochondriasis (Özen, 2010). Just as Shangguan et

al. discovered, individuals with anxiety tend to exaggerate and frequently report somatic symptoms (Shangguan, 2020). Also, panic attacks in anxiety disorder can directly result in sweating, lightheadedness, headache, dizziness, weakness, muscle tension, etc. (Better Health Channel, 2020) Genetic deficits such as metabolic myopathies (Johns Hopkins, 2021), which is a decrease in muscle metabolism, can be also expressed as muscular weakness. Other pre-existing medical conditions like PTSD or cancer tumors, as referenced in the last section, are all potential factors causing the symptoms of SSD.

4.2 External Stimuli

External stimuli are associated with both psychological and biological responses. A stress condition can lead to anxiety response, thus the release of stress-related chemicals, and the malfunction of neural pathways in SSD patients. It could be inferred from Anderson, Maes, and Berk's study, the amount of cortisol released by SSD patients is abnormal because normal cortisol metabolism would not induce neural pathway dysfunction (Anderson, 2012). Therefore, it is hard to tell whether the mental status of SSD patients caused them to amplify changes brought by external stimuli, or the biological difference/change in their body reacted to the external stimuli. Other external stimuli might be strenuous exercise, cold weather, strong light & sound stimuli, and sleep deprivation. These factors can lead to change in blood pressure, muscle contraction, and vascular contraction, which are all causes of headache and dizziness.

4.3 Cognitive Processing

When a physiological change is perceived, SSD patients seem to go through a different cognitive process, either consciously or unconsciously. According to the model of Eifert, Lejuez, and Bouman, past learning would cause the individual to believe in their "state of being ill" and start to use their coping skills in response to the external threatening stimuli (Eifert, 1998). The researchers suggested that illness belief and coping skills are safety-seeking behaviors. Intuitively, we have to focus on the threatening stimulus to ensure we are in a safe condition, and to respond as quickly as possible. This probably explains why SSD patients pay much attention to their somatic feeling. In Stone and Carson's case, the SSD patients received psychological counseling to change their thinking process (Stone, 2011). Their finding shows that when SSD patients do not view somatic symptoms as risk factors, their stress responses are reduced (Stone, 2011). This demonstrates the effect of cognitive processing on symptoms presented by SSD patients.

4.4 Individual Condition

Every individual is different, both biologically and psychologically. Gender is a significant factor in SSD. Female is more susceptible to emotional change and negative external stimuli, sometimes hold a more self-focused thinking process (Ingram, 1988). The premenstrual syndrome was found to be strongly connected with SSD and alexithymia when appeared together with depression (Kuczmierczyk, 1995). Other conditions include social environment, like the epidemic prevention in the society one's in during Covid; education, whether or not one knows SSD or cognitive process or the physiological change they are experiencing; immunity; fitness; etc.

4.5 A Psychobiological Model Of SSD

From the analysis of factors, a general development path of neurological symptoms in SSD can be seen:

I. Individual condition determines the one's susceptibility

II. External stimuli incite a physiological change

III. The physiological change combines with individual's pre-existing condition (if exists) and perceived by individual

IV. The cognitive processing of individual determines the strength of their response (SSD patient respond strongly due to 1/2/3)

V. Self-pressuring/worrying/prolonged symptom becomes the new stimuli

This path is presented visually in **Figure 1**.

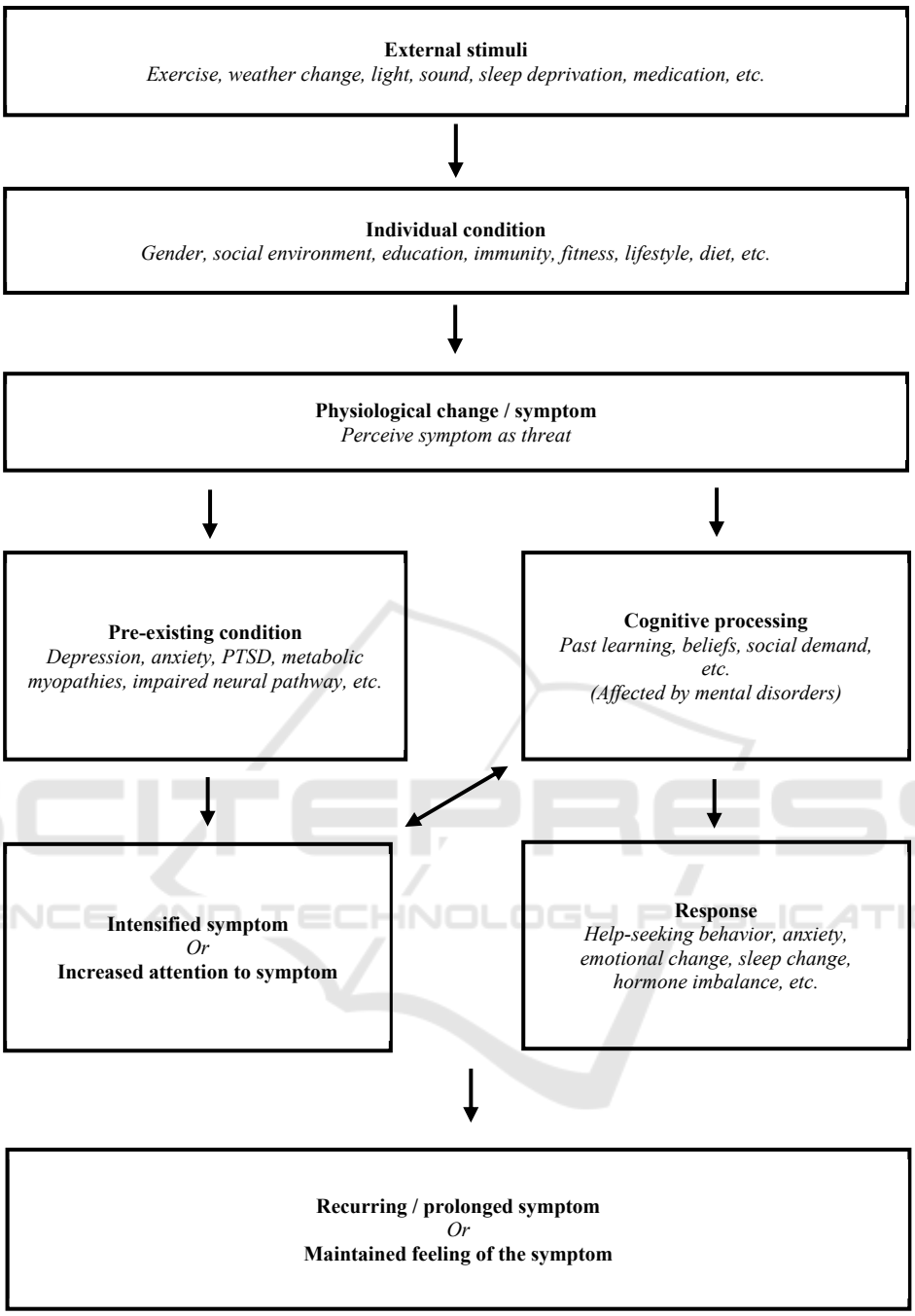

Figure 1: Model for Neurological Symptoms Development of SSD.

5 CONCLUSION

In summary, this paper reviewed previous research on SSD cause and development. This paper found that external stimuli incite a physical change in SSD patients, then biological factors, including individual differences and physical symptoms, are processed by psychological factors, such as preexisting mental disorders and cognitive differences, eventually amplifying patient's perception of their actual physical change. This work innovatively considered three types of factors and combined them into one model, thereby providing a comprehensive discussion

of SSD development. This work will help researchers to target a specific point to intervene in disease development, for example, guide the patient to have an appropriate cognition of physical discomfort. Future studies can focus on providing more empirical support so that a causal relationship between one factor such as gender, and the response, such as anxiety, may be derived.

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