Journal of Psychosomatic Research 101 (2017) 1–9

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

journal homepage: www.elsevier.com/locate/jpsychores

Review article

Somatosensory amplification – An old construct from a new perspective☆

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ARTICLE INFO

Keywords:
Somatization
Somatic complaints
Threat appraisals

SUMMARY

The paper reviews and summarizes the history and the development of somatosensory amplification, a construct that plays a substantial role in symptom reports. Although the association with negative affect has been supported by empirical findings, another key elements of the original concept (i.e. body hypervigilance and the tendency of focusing on mild body sensations) have never been appropriately addressed. Recent findings indicate that somatosensory amplification is connected with phenomena that do not necessarily include symptoms (e.g. modern health worries, or expectations of symptoms and medication side effects), and also with the perception of external threats. In conclusion, somatosensory amplification appears to refer to the intensification of perceived external and internal threats to the integrity of the body ("somatic threat amplification") rather than amplification of perceived or actual bodily events only. Practical implications of this new approach are also discussed.

1. The development of the construct

In a seminal article published almost 40 years ago, Arthur J. Barsky made an attempt to reconsider the phenomenon of subjective somatic symptoms from a bio-psychosocial perspective [1]. It was already well known at that time that there are marked individual differences in the phenomenology of body symptoms, which can heavily impact patients' everyday functioning and well-being [2–4]. The proneness to amplification, which was not without predecessors (e.g. sensitization [5,6], augmenting [7,8]), was characterized by Barsky as a temporally stable, generalized feature that can explain the aforementioned individual differences. Amplification was also described as an important feature of hypochondriasis (i.e. an unrealistic interpretation of physical sensations as abnormal, which leads to preoccupation with the fear of having a serious disease [9]); as the term had negative connotations, the use of the more neutral "amplifying somatic style" was proposed. Moreover, a novel approach to hypochondriasis was developed with a special emphasis on the amplification tendency [10].

A decade later, the term somatosensory amplification (SSA) was introduced and defined as *the tendency to experience somatic sensation as intense, noxious, and disturbing*[11]. SSA was assumed to consist of three components: (1) body hypervigilance, (2) focusing on rare and weak body sensations, and (3) a cognitive-emotional ("cortical") reaction to the sensations. In other words, amplification included both lower-level (sensory) and higher-level (cognitive-emotional) processes, which received serious criticism for confounding a potential explanatory mechanism (i.e. higher somatic sensitivity to sensations) with the outcome of this mechanism (i.e. the experience of symptoms as a more complex cognitive-emotional process) [12]. The first 5-item version of the Somatosensory Amplification Scale (SSAS) was also published and used [11,13,14]. Several years later, a longer 10-item version was developed (Table 1) [15]. Items were selected from a collection of statements on uncomfortable and unpleasant sensations obtained from medical outpatients [13].

Surprisingly, if one takes a look at the items, some of them partly or completely refer to exteroceptive modalities (Table 1). This issue was clearly indicated by the factor analysis of several national versions [16,17], while the majority of the studies reported a better fit with a single factor structure [18–24]. The relatively poor internal consistency (Cronbach's alpha = 0.65–0.75) reported in many studies [18,20,22,25,26] might also be the consequence of this conceptual heterogeneity, beyond the little length of the scale and the diversity of the organ systems involved.

☆ Conflicts of interest and source of funding: This research was supported by the Hungarian National Scientific Research Fund (OTKA K 109549 and K 124132). The authors wish to say thank Keith Petrie for the Wanaka meeting that substantially catalyzed the preparation of this paper.
No conflicts of interest are declared.

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http://dx.doi.org/10.1016/j.jpsychores.2017.07.011
Received 26 May 2017; Received in revised form 25 July 2017; Accepted 26 July 2017

Table 1
Items of the Somatosensory Amplification Scale and their associations with intero- and exteroception (i.e. perception of stimuli from within the body or from the environment, respectively).

Items	Interoception	Exteroception
1. When someone else coughs, it makes me cough too	Yes (secondary)	Yes
2. I can't stand smoke, smog, or pollutants in the air	Yes (secondary)	Yes
3. I find I'm often aware of various things happening in my body	Yes	
4. When I bruise myself, it stays noticeable for a long time	Yes (secondary)	Yes
5. Sudden loud noises really disturb me		Yes
6. I can sometimes hear my pulse or my heartbeat throbbing in my ear	Yes	
7. I hate to be too hot or too cold	Yes	
8. I'm quick to sense the hunger contractions in my stomach	Yes	
9. Even something minor, like an insect bite or a splinter, really bothers me	Yes (secondary)	yes
10. I have a low tolerance for pain	Yes	

1.1. Somatosensory amplification and visceral sensitivity

Somatosensory amplification as assessed by the SSAS showed a good test-retest reliability (r = 0.85 over 1.5–5 weeks, 0.79 over on average 74 days) and internal consistency (Cronbach's alpha = 0.82) in the first studies [11,15]. Its good temporal stability has been supported by later studies (r = 0.7–0.8 over 4–8 weeks) [18,22,25,27]. Concerning validity, however, only incomplete research was carried out. Of the three components described earlier, only the cognitive-emotional reaction was investigated in a systematic manner. The SSAS score showed medium level correlations with various indicators of negative affect and the perceived severity of symptoms of the upper respiratory tract [11,13–15].

Associations with the remaining two components of the construct (i.e. body hypervigilance and focusing on mild body sensations) were not investigated. The first study that compared SSAS with a scale assessing body awareness was published in 2001 only [28]; the reported medium level association has been replicated [29,30]. It is worth mentioning, however, that body awareness refers to perceived (subjective) body changes and states (i.e. interoceptive sensibility), which might be different from hypervigilance to body sensations.

The proneness to focus on weak and rare body sensations was approximated by visceral sensitivity (heartbeat detection ability), which is also a questionable idea as it is based on a naïve-realistic somatosensory perception model. To the surprise of the researchers, no or negative associations were found [26,31–33], and it was concluded that SSA is related more to the cognitive-emotional reaction (also called cognitive bias) to the symptoms than the detection of visceral events [26,31]. Taking into consideration the results of later studies [29,34–38], independence from visceral events appears to be more likely than a negative association. As patients with hypochondriasis showed no decreased sensory threshold for either cardiac information [31] or tactile stimulation [39], the presumed sensory amplification model of hypochondriasis was rejected [40–42].

In conclusion, the original conceptualization of the construct was only partially supported by empirical findings. However, as the SSAS is the only available questionnaire that assesses somatosensory amplification tendency, it is difficult to distinguish between issues related to the construct itself and validity issues with the scale [12]. Beyond the already mentioned conceptual problem with the construct, the scale was also criticized for (1) measuring the outcome of the amplification process and not its components, and (2) being not necessarily specific to somatic sensations (i.e. also assessing other forms of distress) [12].

1.2. Associations with hypochondriasis, somatisation, alexithymia, negative affect, and attribution style

It turned out that the association between SSA and hypochondriasis is substantial but not strong enough to equate the two constructs [17,22,43]. Components of SSA (body vigilance, intensification through emotional reaction) were finally included in the cognitive-emotional model of hypochondriasis [44,45]. Associations with health anxiety, the dimensional approach to hypochondriasis, were also reported [28,46–49].

Researcher's attention shifted to somatisation (i.e. the tendency to experience and communicate psychologic distress in the form of somatic symptoms that the patient misinterprets as signifying serious physical illness [50]), a broader concept. The overall strength of the association between SSA and somatisation was in the medium domain [51,52], which indicated that the constructs substantially overlap but yet represent distinct phenomena [53]. The existence of a moderate to strong connection between the two constructs is supported by more recent empirical findings though [23,54,55].

It was also proposed that SSA is associated with alexithymia (i.e. an impaired ability to identify and verbalize emotions [56,57]), a characteristic related to somatization. Empirical results support the existence of this connection [36,55,58–65], although null findings were also reported [66–68]. Although there seems to be an association between both constructs (possibly due to a joint overlap of both constructs with trait negative affect), alexithymia does not fully account for SSA. Moreover, findings have to be interpreted with caution because in most cases alexithymia was assessed only by self-report (mostly the Toronto Alexithymia Scale), which might be particularly problematic in case of a trait which is known to reflect problems in introspection and self-perception [69].

The possibility that SSA primarily reflects individual differences in neuroticism (i.e. dispositional emotional instability and stress reactivity) and negative affectivity (i.e. the disposition to experience aversive emotional states [70]) was also raised [34]. This approach was supported by previous data on the association between indicators of neuroticism, negative affectivity, and SSA [11,15,52,61,71]. Because these traits (neuroticism and trait negative affect) are considered as general risk factors for psychopathology, SSA is assumed to be significantly related to anxiety, depression, and general psychopathology. Several findings suggest that SSA represents a trait-like phenomenon that is related to psychopathology but appears less susceptible to change compared to symptoms of anxiety and depression. In one study, patients with fibromyalgia and major depression received antidepressant drug therapy (75 mg venlafaxine and 30–100 mg sertraline per day, respectively) over a 12-weeks period [72]. In the fibromyalgia group, both depression and anxiety showed a significant drop, while SSA did not change. In the major depression group, all three characteristics decreased; however, the significant decrease of SSA disappeared after controlling for anxiety and depression. No connection with depression and anxiety was reported in another study [28]. In conclusion, although SSA is theoretically and empirically strongly associated with neuroticism and trait negative affect, the different constructs are not identical [30].

Finally, it is worth mentioning that empirical findings concerning the associations between SSA and various attribution styles (i.e. personal preferences for one or another type of causal explanation for symptoms [73]) are not conclusive either; connections with psychological [34,65], somatic [34,74], and normalizing [74] attribution style as well as lack of association [32,55] were reported.

Although the exact mechanisms and determinants of SSA are still unknown, the scale has served well the original purpose of its development, i.e. the assessment of individual differences in symptom perception and reporting [75]. Higher levels of SSA are assumed to turn body sensations into symptoms, and increase the severity of already existing symptoms; in this vein, the term symptom amplification was