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Putting the Vicious Cycle to the Test: Evidence for the Cognitive Behavioral Model of Persistent Somatic Symptoms From an Online Study

Alexander H. J. Sahm, MSc, Michael Witthöft, PhD, Josef Bailer, PhD, and Daniela Mier, PhD

Objective: In clinical practice, persistent somatic symptoms are regularly explained using a cognitive-behavioral model (CBM). In the CBM, predisposing, perpetuating, and precipitating factors are assumed to interact and to cause the onset and endurance of somatic symptoms. However, these models are rarely investigated in their entirety.

Methods: We conducted an online survey during the COVID-19 pandemic. A total of 2114 participants from the general German population completed questionnaires that measured different factors of the CBM. We used state negative affectivity and neuroticism as predisposing factors, fear of a COVID-19 infection as the precipitating factor, and somatic symptoms, misinterpretation of bodily symptoms, attention allocation to bodily symptoms, and health anxiety as perpetuating factors. Moreover, we added safety and avoidance behavior as end points to the model. We conducted a psychological network analysis to exploratively study the relationships between the model's different factors and tested the assumptions of the CBM by evaluating a structural equation model (SEM) that incorporated all factors of the model.

Results: Network analyses revealed clustering in our data: Health anxiety and different cognitive factors are closely related, whereas somatic symptoms and state negative affectivity are strongly associated. Our SEM showed adequate fit.

Conclusions: Our findings from an exploratory and a confirmatory approach give empirical support for the CBM, suggesting it as a suitable model to explain bodily symptoms in the general population and to possibly guide clinical practice. The network model additionally indicates the necessity to apply an individualized CBM for patients, depending on a preponderance of either persistent somatic symptoms or health concerns.

Key words: somatic symptom disorder, health anxiety, COVID-19, cognitive behavioral therapy, network analyses

Abbreviations: CBM = cognitive-behavioral model, **COVID-19** = coronavirus disease 2019, **DSM** = Diagnostic and Statistical Manual of Mental Disorders, **eBIC** = extended Bayesian information criterion, **gLASSO** = graphical least absolute shrinkage and selection operator, **IAD** = illness anxiety disorder, **ICD** = International Classification of Diseases, **MUS** = medically unexplained symptoms, **PHQ-15** = Patient Health Questionnaire-15, **PHQ-4** = Patient Health Questionnaire-4, **PSS** = persistent somatic symptoms, **SEM** = structural equation model, **WLSMV** = weighted least square mean and variance adjusted

(*Psychosom Med* 2024;86:569–575)

INTRODUCTION

Persistent somatic symptoms (PSS) are common in the general public, with 22.1% of the population reporting at least one debilitating somatic symptom over the past 7 days (1). Such PSSs[1] are the hallmark of a multitude of clinical diagnoses, including, but not limited to, somatoform disorder (ICD-10), bodily distress disorder (ICD-11), somatic symptom disorder (DSM-5), and several functional somatic syndromes (e.g., fibromyalgia) (2). This plethora of diagnoses is mirrored in numerous explanatory models for PSS, among which classical conditioning (e.g., (3)), cognitive (e.g., (4,5)), psychobiological (e.g., (6)), sensitization (e.g., (7)), and predictive processing (e.g., (8,9)) explanations can be differentiated (cf. (10)). The cognitive-behavioral model (CBM) (11) explaining PSS and health anxiety is perhaps the most frequently used model in clinical practice. However, this model has never been empirically evaluated in its entirety. The present study was conducted to explore the CBM by network as well as structural equation modeling in the general population. Data were collected during the COVID-19 pandemic, which is associated with an increase in somatic symptoms and health anxiety in the general population (12).

The Cognitive-Behavioral Model of PSS

In their review, Deary and colleagues (11) differentiate the CBM from other cognitive or psychobiological models by pointing toward its eclectic nature, simultaneously entailing cognitive, behavioral, and biological components. The identifying characteristic of the CBM is a self-perpetuating dynamic of predisposing, perpetuating, and precipitating factors. In that regard, the CBM of PSS mirrors similar "vicious cycle" models for other diseases, for example, generalized anxiety disorder (13). Vicious cycle models often entail a broadly defined

[1]While a large part of the existing literature uses the term *medically unexplained symptoms* (MUS), we prefer the usage of the term *persistent somatic symptoms* (PSS), as it encompasses both medically unexplained and underexplained somatic symptoms and is, thus, more inclusive. The term also better fits the current state of classificatory systems; namely the definitions of somatic symptom disorder in DSM-5 and bodily distress disorder in ICD-11.

From the Department of Psychology (Sahm, Mier), University of Konstanz, Konstanz; Department of Clinical Psychology, Psychotherapy, and Experimental Psychopathology (Witthöft), Johannes Gutenberg-University Mainz, Mainz; and Department of Clinical Psychology, Central Institute of Mental Health (Bailer), University of Heidelberg/Medical Faculty Mannheim, Mannheim, Germany.

Address correspondence to Alexander H. J. Sahm, MSc, Box 905, Universitaetsstrasse 10, 78457 Konstanz, Germany. E-mail: Alexander.Sahm@uni-konstanz.de

ORCID IDs: 0000-0002-1401-4329 (A.H.J.S.); 0000-0002-4928-4222 (M.W.); 0000-0002-2196-2482 (J.B.); 0000-0003-2518-7492 (D.M.).

Article Editor: Harald Gündel

Received for publication June 16, 2023; revision received February 27, 2024.

Supplemental digital content is available for this article.

ISSN: 0033-3174

DOI: 10.1097/PSY.0000000000001313

predisposition for a disorder that, in combination with a triggering event (most often: stress), leads to first symptoms, which are then iteratively strengthening themselves in feedback loops (11). In clinical practice, this flexibility allows therapists to create individual disease models that may be used for tailoring psychotherapeutic interventions to the individual patient.

Although the CBM has been mainly developed for functional somatic symptoms, here we apply it to somatic symptoms in general. An association of negative affectivity and somatic symptoms has frequently been shown both in healthy samples (e.g., (14,15)), as well as in patients with fibromyalgia/chronic fatigue syndrome (e.g., (16)) or irritable bowel syndrome (reviewed in Ref. (17)). These and other results have led to the change of somatoform disorder to somatic symptom disorder in DSM-5 (18), which does not differentiate between medically explained and unexplained symptoms (cf. (19)).

Predisposing Factors

The predisposing factors of PSS encompass personality characteristics like neuroticism, as well as state and trait negative affectivity. In addition to affective and anxiety disorders (20), neuroticism has frequently been associated with somatic symptoms (21,22) and health anxiety (23). Importantly, neuroticism was related to perpetuating factors such as somatosensory amplification (24), as well as other predisposing factors, such as alexithymia and state negative affectivity (25). State negative affectivity has been related to somatic symptoms in clinical samples (16), and trait negative affectivity has been found to predict healthy anxiety (26). Experiments have also shown that individuals with high trait negativity show a negative bias in reaction to negative affective cues during interoception (27). In agreement, it was shown during the COVID-19 pandemic that neuroticism was associated with general anxiety, as well as corona fear (28), and that corona fear was related to the experience of somatic symptoms (29,30). Moreover, neuroticism was associated with the perceived stress during the pandemic (31).

Precipitating Factors

In their review, Deary and colleagues (11) discuss general "life events" as a repeated and ongoing strain on bodily stress systems, which ultimately give way to somatic symptoms. Regarding precipitating factors, Hatcher and House (32) demonstrated that individuals with chronic fatigue syndrome had a nine times higher risk for stressful life events within the last 3 months before symptom onset than healthy controls. However, longitudinal evidence shows that this close relationship between stress and somatic symptoms does not hold for all patients with functional somatic symptoms (33). Recent evidence suggests that chronic stress may be a mediator between different stress-related predisposing factors and somatic symptoms, suggesting that chronic rather than incidental stressors may trigger the vicious cycle (34). During the COVID-19 pandemic, those individuals with enhanced distress before the outbreak also had the highest increase in symptom distress during the outbreak (35,36), showing the role of prolonged or repeated stressors in the onset of somatic symptoms.

Perpetuating Factors

Ursin (37) has proposed that patients experiencing PSS are more sensitive to bodily misperceptions and are increasingly prone to these misperceptions over the course of the illness. In that manner, somatic symptoms occurring after a stressful life event lead to higher sensitivity for somatic misperceptions, which in turn increase overall perceived stress and sensitivity. Importantly, a lack of adaptive coping strategies and negative cognitive expectations serve as mediators between increased sensitivity and increased somatic symptoms (7).

Among others, an alternative model has been presented by Rief and Barsky (6), who argue that somatic symptoms arise from aberrant filtering processes and increased physiological signaling of the body, due to chronic stress or sensitization. Selective attention and health anxiety may serve to further impair this filter system, for example, via somatosensory amplification (38,39). In an adaptation of this model, Brown (4) emphasizes the importance of perceptual schemas in the experience of somatic symptoms. If, e.g., a pain schema is already activated and coincides with normal bodily sensations, these are interpreted in a threating context, leading to according psychological and physiological reactions. This process subsequently fosters the activation of a pain schema in following encounters with bodily misperceptions. In agreement, converging evidence shows that somatic attribution styles are associated with the experience of PSS (40), and that patients with somatoform disorders have more somatic attributions than controls (41). Moreover, catastrophizing misinterpretations of bodily symptoms and somatosensory amplification are related to health anxiety and PSS (42–44).

Apart from cognitive factors, illness behavior may lead to the continuation of somatic symptoms and health anxiety. Illness behavior encompasses a wide range of phenomena, including increased self-checking, doctor visits, and treatment seeking, but also avoidance of physical activities and health worries (40,45). Rief and colleagues (46) found that patients with somatic symptoms showed higher expressiveness related to their symptoms, more medication and treatment seeking, avoidance of exercise, and body checking than healthy controls. Illness behaviors may thus result in the subsequent strengthening of several cognitive factors, leading to a direct interplay of behavior and cognition in the emergence of PSS.

The Present Study

Individualized disease models based on the CBM or similar models are an essential part of cognitive behavioral therapy of PSS. The effectiveness of these approaches (47) suggests a heuristic value of the CBM for understanding and treating patients' interconnected symptoms. However, to our knowledge, the CBM has never been empirically evaluated in its entirety. The dynamic and self-perpetuating nature of the CBM resonates with timely approaches that understand psychopathology as the result of a complex interaction of symptoms (48). Recently, network models have been utilized to study connections between different somatic symptoms (49) and the connection of posttraumatic stress and somatic symptoms (50). However, to our knowledge, there are no network analytic studies that examine the different components of the CBM simultaneously.

Because direct evidence of the explanatory power of the CBM is scarce, our study served a dual purpose. First, we wanted

to exploratively study the features of the CBM by examining its correlational structure without any restricting assumptions. Second, we set out to test the CBM to assess its empirical fit to real-life data.

For the present study, we included neuroticism and state negative affectivity as predisposing factors, the fear of a corona infection as a precipitating factor, and several physiological (somatic symptoms), cognitive (misinterpretation of bodily signals, aberrant attention allocation, health anxiety), and behavioral (avoidance, safety behaviors) components as perpetuating factors. As our data lack a time dimension that would allow us to study the dynamic interaction of the components, we entered them to our model in the order we would expect them to occur in a patient: With predisposing factors first, followed by the precipitating factor, and the physiological and cognitive components of the perpetuating factors in subsequent succession. The behavioral consequences of these components form the end point of our model. As the survey was conducted during the COVID-19 pandemic, we had elevated distress levels in our sample (12) and were able to use corona fear as an exogeneous trigger to the CBM (see Supplemental Digital Content, Figure S1, http://links.lww.com/PSYMED/B24, for an illustration).

For our analyses, we did not focus on specific patient groups but conducted a large survey in the general public, which allowed us to use network models that have been successfully applied to exploratively study the interconnections of different symptoms (e.g., (51)) and to fit the complex structural models implicated by the CBM. Implications from network and structural equation models typically diverge (52) and complement each other, i.e., exploration of patterns in multivariate data without theoretical assumptions versus direct testing of theoretical assumptions.

METHODS

Sample

This investigation used data from a cross-sectional SoSci Survey online assessment of a convenience sample of 2224 people from the general population of Germany. Sample size was determined by the timing of the data collection (May to September 2020). The sample was previously used to analyze the mental health effects of the COVID-19 pandemic as published in Biermann et al. (12). We excluded participants with improbable completion times, age under 16 years, for analysis-purposes nonbinary gender, and missing values for education, resulting in an effective sample size of $N = 2114$. Participants predominantly identified as female (75.07%), were around 40 years of age ($M = 42.67$, SD = 14.26, range = 16–86), and 49% reported to have college education (see Supplemental Digital Content, Table S1, http://links.lww.com/PSYMED/B24). The study was approved by the local ethics board of the Psychology Department at the Johannes Gutenberg-University of Mainz (2020-JGU-psychEK-S010), and all participants provided written informed consent. The raw data and code supporting the conclusions of this article will be made available by the authors, under limited conditions.

Measures

The survey included established questionnaires measuring somatic (Patient Health Questionaire-15 (53)) and psychological distress (Patient Health Questionnaire-4 (54)), personality traits (Big Five Inventory-10 (55)), as well as selected items from widely used health anxiety questionnaires that aimed to measure bodily vigilance (Somatosensory Amplification Scale (56), Health Anxiety Inventory (57)), bodily misinterpretations (Fragebogen zu Koerper und Gesundheit (58)), health anxiety (Health Anxiety Inventory), and illness information avoidance and safety behavior (Fragebogen zur Erfassung von Sicherheitsverhalten bei vorliegender Hypochondrie (59)) (see Supplemental Digital Content Table S2, http://links.lww.com/PSYMED/B24, for English translations of these items and their scales of origin). In addition, a three-item scale measuring current and assumed prospective COVID-19 fear was included ("How strong is your fear of a COVID-infection?" "How strong will your fear of a COVID-infection be in four weeks?" "How strong will your fear of an COVID-infection be in eight weeks?"). All measures, except three (Cronbach's $\alpha > .60$), had good internal consistencies (Cronbach's $\alpha > .80$) (see Table S3). Because we used a forced response design for the questionnaires, we did not have missing data in our final data set. The PHQ-15 item on menstrual pain was excluded from further analyses to avoid missing data for male respondents. Because the COVID-19 fear scale had high internal consistency ($\alpha = .97$), which indicated redundancy between items, we calculated the mean of the three underlying items and used the resulting fear index in all subsequent analyses.

Network Analyses

Because most of our measures had good internal consistencies, we chose to use mean scores for our scales in all network analyses in order to avoid topological overlap (60). The resulting scores were not normally distributed (as can be seen from Supplemental Digital Content, Figure S2, http://links.lww.com/PSYMED/B24), thus violating the assumption of multivariate normality. To alleviate this issue, we applied the nonparanormal transformation to our data before estimating our models (61,62). After estimating the partial correlation network based on the transformed data, graphical least absolute shrinkage and selection operator (gLASSO) regularization with extended Bayesian Information Criterion (eBIC) model selection ($\gamma = 0.5$) was used on the data (61). These estimations were carried out with *R* and the *bootnet* package (63).

Results were visualized using the *qgraph* package (64). In order to make the distance between nodes interpretable, we used multidimensional scaling of zero-order correlations (65) (see Table S4 for zero-order correlations). Further, we estimated the nodewise predictability (66) using the *mgm* package (67) and displayed the variance explained of each node by all the nodes connected to it.

The accuracy of edge estimates and centrality indexes were assessed using the bootstrapping procedures established in the *bootnet* package using 1000 nonparametric bootstrap samples.

Structural Equation Models

All latent variables were defined on the item level, except for the items from the PHQ-15 for which we built parcels for each subclass of somatic symptoms (pain, gastroenterology, cardiopulmonary, fatigue) (68). We chose all four PHQ-4 items to measure the latent variable "state negative affectivity," rather than entering anxiety and depression separately. The SEM was

estimated using robust estimation methods ("WLSMV") in the R-package *lavaan* (69). Model fit was assessed using standard criteria (70) and scaled fit statistics (71,72). We report standardized coefficients for our model.

RESULTS

Network Models

Figure 1 shows a visualization of the results of our network analysis and Table 1 the associated centrality and predictability-indicators (see Supplemental Digital Content, Table S5, http://links.lww.com/PSYMED/B24, for the underlying partial correlations). Visual inspection of the edge weight confidence intervals in Figure S3, and a high centrality stability coefficient (CS > 0.75; see also Figure S4) indicated stable results for our network model. Further, the bootstrapped differences in node strength showed that the four strongest nodes (depression, anxiety, attention allocation, and health anxiety) were more central than the other nodes in the network (see Figure S5). Correlations between strength and betweenness (r = 0.72, 95% CI = 0.16 to 0.93) and strength and predictability (r = 0.93, 95% CI = 0.72 to 0.98) were high and significant. Betweenness and predictability showed no significant correlation (r = 0.57, 95% CI = −0.09 to 0.88).

SEM

Overall, our model showed an acceptable fit (χ^2 = 2763.513; df = 285; CFI = 0.962; TLI = 0.957; RMSEA = 0.064; 90% CI = 0.062–0.066; SRMR = 0.053). Figure 2 shows the relationships between the latent variables in our model (see detailed results in Supplemental Digital Content, Table S6, http://links.lww.com/PSYMED/B24).

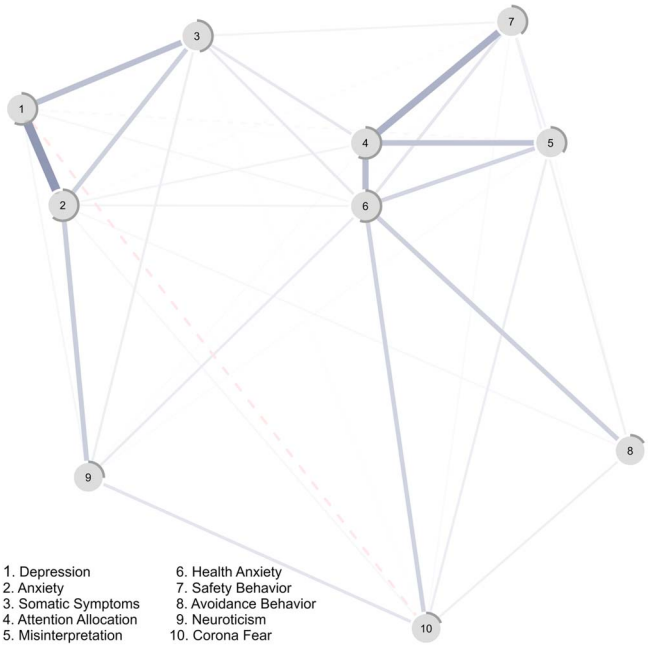

FIGURE 1. Visualization of the network model. Straight lines indicate positive partial correlations, and dotted lines indicate negative partial correlations. Pie charts for each node represent the share of variance explained at each node by all the nodes connected to it (predictability). Distance between nodes reflects their zero-order correlations. N = 2114.

TABLE 1. Betweenness (How Often a Node Lies on the Shortest Connecting Edge Between Two Nodes), Strength (the Sum of All Edges Connected to the Node), and Predictability (the Proportion of Variance Explained in Each Node by Regressing All Its Connected Nodes on It) for All Nodes in the Network Model (N = 2114)

		Measure		
Node	Variable	Betweenness	Strength	Predictability
1	Depression	1	1.009	0.571
2	Anxiety	2	1.122	0.608
3	Somatic symptoms	10	0.849	0.470
4	Attention allocation	11	1.137	0.573
5	Misinterpretation	0	0.678	0.353
6	Health anxiety	14	1.311	0.556
7	Safety behavior	0	0.724	0.391
8	Avoidance behavior	0	0.395	0.167
9	Neuroticism	1	0.598	0.255
10	Corona fear	0	0.621	0.192

DISCUSSION

We used a large sample from the general population to study the relationships between different parts of the CBM of somatic symptoms (11), which differentiates between predisposing, precipitating, and perpetuating factors. The COVID-19 pandemic allowed us to use the threat of a corona infection as an exogenous trigger to the model. We applied two complementary perspectives, the exploratory view of a psychological network model and the confirmatory view of a structural equation model.

Exploratory Relationships in the Network Model

To explore the relationship between the different factors of the CBM, we applied a network model. Our network model shows two major clusters: a) an affective somatic cluster with depression, anxiety, and somatic symptoms, and b) a cognitive behavioral cluster with attention allocation, misinterpretation, health anxiety, and safety behavior. This suggests a close relationship between affective and somatic symptoms on the one hand, and cognitive-behavioral symptoms and health anxiety on the other hand.

Regarding the first cluster, depression and anxiety exhibited the strongest relationship, unsurprisingly as both constitute subscales of the PHQ-4. Both anxiety and depression were related to somatic symptoms as measured in the PHQ-15, indicating an important role of the predisposing factor state negative affectivity in explaining the onset of somatic symptoms. Further, anxiety, but not depression, was related to neuroticism, suggesting (trait) anxiety as a stable predisposing factor. These associations are in line with a vast number of studies showing that negative affectivity is related to an increase in somatic symptoms (e.g., (14,16)). Future studies should apply psychophysiological measurements, as well as questionnaires on emotion processing to further understand this relationship.

The second cluster reflects health anxiety as a cognitive-behavioral process of precipitating and perpetuating factors. Here, as in the SEM, the cognitive factors were closely related to one another. Our results suggest that state negative affectivity is not directly related to health anxiety (26), but via somatic

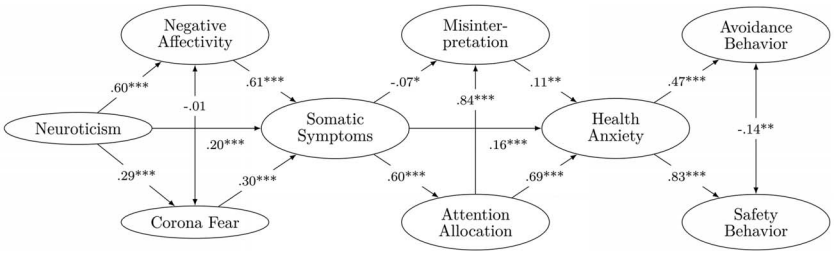

FIGURE 2. Standardized relationships between the latent variables in the model. Stars indicate statistical significance for all path coefficients: *$p < .05$, **$p < .01$, ***$p < .001$. $N = 2114$.

symptoms that are interpreted as a health threat. As proposed in the somatosensory amplification model (38), health anxiety and attention allocation were related to somatic symptoms, but no direct relationship of misinterpretation and somatic symptoms emerged. The behavioral end points of the CBM, safety and avoidance behavior were strongly related to health anxiety, thus mirroring the mediating role health anxiety had in our SEM. Further, attention allocation was strongly related to safety behavior, indicating that heightened attention toward bodily symptoms coincides with stronger behavioral responses, such as checking behavior. Several studies have demonstrated a relationship between neuroticism and corona fears and stress during the pandemic (28,31,73). We replicated this finding, but contrary to its proposed role in the SEM, corona fear as a precipitating factor was not related to somatic symptoms and state negative affectivity in the network model. However, we show a close relationship of health anxiety and avoidance behavior with corona-related fears, suggesting an indirect effect of corona fear on somatic symptoms via health worries. This mediation effect, however, warrants further research.

The centrality of the respective variables in our network model closely mirrors their clinical utility (74). Cognitive variables as health anxiety and attention allocation proved the most central parts of the model, whereas the behavioral end points and predisposing factors were peripheral. In clinical practice, cognitive variables are important to intervene on, as they form the predecessor of any behavioral consequences. Intriguingly, misinterpretation was significantly less central than health anxiety and attention allocation, suggesting that misinterpretation may be less suitable an intervention point for PSS than those other variables, but highly prominent for health anxiety. As health anxiety is defined by dysfunctional cognitions and dysfunctional coping behavior, the finding of the close link between health anxiety and misinterpretation fit cognitive-behavioral approaches in therapy. In contrast, somatic symptoms might respond rather to interventions focusing on reducing negative affect. Hence, the findings of the network model strongly suggest that PSS and health anxiety are related via somatic symptoms but differ in their interactions with affective versus cognitive processes.

Confirmatory Evidence for the Cognitive-Behavioral Model of Persistent Somatic Symptoms

In our SEM-analysis, we strived to find evidence for an adaptation of the CBM that follows the supposed temporal evolution of symptoms in patients. In line with our expectations, this structure fit the data reasonably well, presenting evidence in favor of the CBM. When looking at the different parts of the model, as expected, we found a positive relationship between neuroticism and state negative affectivity as predisposing factors, and the fear of a corona infection as a precipitating factor. State negative affectivity and corona fear were not related to one another, controlling for the effect of neuroticism on both variables. Closely mirroring the idea of an interplay between preparedness and exogenous stressors in leading to the advent of somatic symptoms, all precipitating and predisposing factors in our model were positively related to somatic symptoms, as measured by the PHQ-15. With the exemption of the negative effect of somatic symptoms on the misinterpretation of bodily signals, all our perpetuating factors (misinterpretation of bodily signals, aberrant attention allocation, health anxiety, somatic symptoms) were positively related, giving credibility to the CBM's claim of iteratively self-perpetuating relationships between those factors. The small and negative relationship between somatic symptoms and misinterpretation, however, might indicate that misinterpretation is mainly of importance in the onset of health anxiety and less so for somatic symptoms. Finally, health anxiety was positively related to the behavioral parts of our model, namely, avoidance and safety behavior, which were negatively correlated to each other, suggesting that people tend to use either avoidance or safety behavior, and not both strategies to cope with health-related concerns. This mirrors the DSM-5 categorization of patients with illness anxiety disorder (IAD) as either avoiding care, or care seeking, but not the findings of Newby and colleagues, who showed that a majority of patients with IAD switch between care seeking and care avoidance (75).

Our findings broadly support the work of other studies on PSS that found positive correlations between different predisposing factors (e.g., (25)), between predisposing and perpetuating factors (e.g., (24)), as well as between different perpetuating factors (see (42)). Kolk and colleagues (76) have investigated the relationships between exogenous factors, such as socioeconomic status or age, and several concepts related to symptom perception. Relevant to our results, they identified a pathway from negative affectivity over selective attention to bodily symptoms to physical symptoms. Although, in our model, the sequence of concepts was different (negative affectivity, followed by physical symptoms, followed by aberrant attention allocation), we similarly found positive connections between these concepts. Although our study lacked the exogenous perspective of the work of Kolk et al. (76), it is the first to simultaneously model the correlation of a number of symptoms from the three

parts of the CBM and to report evidence of an acceptable fit of the CBM to real-life data.

Limitations

Several limitations hamper the interpretation of these results. First and foremost, although we have strived to validate a model for PSS, our empirical data stem from a community sample, of which participants experienced somatic symptoms in the last 4 weeks in varying degrees, as assessed in the PHQ-15. We assessed neither whether these somatic symptoms persisted for a longer period nor whether they have an organic explanation. Our results thus supported the proposed mechanisms of the CBM, but can only be cautiously applied to clinical populations. Second, in the context of SEMs, evidence in favor of a certain model is not equivalent to evidence against other possible models. In fact, many different models can lead to a comparable fit to the data (77), obscuring the interpretation of our present results. Thus, based on these data, we can only conclude that one possible operationalization of the CBM fit our data, not that this is the true data-generating process explaining the correlations between our observed variables. Third, here we modeled a dynamic, temporal process with cross-sectional data, which can greatly affect our results. In addition, between- and within-person relationships might not overlap, thus obscuring potential individual differences in the relationships of different symptoms, which has led to the call for more longitudinal data in recent years (78). Fourth, the CBM integrates processes of different time dimensions. Stressors causing negative affectivity lead to somatic symptoms. How fast this happens might depend on attentional biases toward bodily symptoms, and vulnerability to interpret bodily symptoms as signs of severe disease and, in turn, influences whether health anxiety arises. This somatosensoric amplification is thought to occur within a very short time span, whereas the maintaining effect of avoidance and safety behaviors can cover periods of month and years. Thus, the perpetuating factors happen at very different time dimensions, which was not mirrored in our data collection. Fifth, these problems also apply to our measure of negativity affectivity. The PHQ-4 has been established as a short screening instrument for state anxiety and depression and only captures the time span of the last 2 weeks (54). Thus, although it seems reasonable to assume a robust correlation of the measure and trait negativity, we cannot rule out the dominance of state variance in the measure. Sixth, the internal consistencies of three of our measures (misinterpretation, safety behavior, and neuroticism) were low but acceptable (0.65, 0.64, and 0.65), thus hampering their interpretability.

Although our SEM and network findings provide some support for the CBM and, with this, its usage for constructing individual disease models in cognitive behavioral clinical practice, replication of our analyses and the analysis of longitudinal data are necessary to affirm this conclusion. Future studies may improve on our work by estimating individualized network models to better allow individualized therapeutic care. Lastly, our analyses used a convenience sample from the general population, and we thus are unable to generalize our findings to clinical populations. In addition, it must be kept in mind that our data were acquired during a pandemic. This allowed us to assess the factors of the CBM in a convenience sample with more variance in mental distress and health anxiety than in times before the pandemic, but the results might also partially reflect the specific situation of our participants.

CONCLUSION

Our findings with a (exploratory) network model and a (confirmatory) structural equation model convergingly suggest a good fit of the CBM to real-life data. This supports the suitability of the CBM to guide clinical practice and interventions for both somatic symptoms and health anxiety. Moreover, the network model demonstrates a clustering of somatic symptoms with negative affect and a clustering of health anxiety with cognitive components, suggesting the necessity to differentiate between PSS and health anxiety and to use individualized CBM for the specific symptomatology of a patient.

The authors thank Sneha Sridhar for contributing English translations for several items of the questionnaires used in this study.

Source of Funding and Conflicts of Interest: None declared.

Transparency and Openness Promotion Disclosure: This study was not preregistered. This manuscript was posted as a preprint on psyarxiv.com on June 15, 2023. DOI: https://doi.org/10.31234/osf.io/7c4yf. The raw data and code supporting the conclusions of this article will be made available by the authors, under limited conditions.

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