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BRIEF REPORT

Clarifying the Latent Structure and Correlates of Somatic Symptom Distress: A Bifactor Model Approach

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Distressing somatic symptoms are ubiquitous both in mental disorders and medical diseases. From a psychometric perspective, the structure of somatic symptom distress is unclear, and little is known about the strengths of associations to related constructs, such as health anxiety and somatosensory amplification. To clarify the structure of somatic symptom distress and to explore associations to health anxiety, somatosensory amplification, and functional somatic syndromes, data sets of 2 samples of college students from Germany (N = 1,520) and Switzerland (N = 3,053) were investigated with confirmatory factor analysis with robust estimation. A bifactor model (with 1 general and 4 orthogonal specific symptom factors—gastrointestinal, fatigue, cardiopulmonary, and pain symptoms) revealed the best model fit. Medium-sized associations were found among latent factors of general somatic symptom distress, health anxiety, and depression. First evidence for the construct validity of the latent variables within the proposed bifactor structure was gained by observing (a) strong associations between the general somatic symptom distress factor and somatosensory amplification and (b) significant associations between both the general somatic symptom factor as well as the symptom-specific factors with functional somatic syndromes. The results offer a theoretically and psychometrically plausible model for the structure of somatic symptom distress and suggest a distinction between cognitive-affective and sensory aspects of symptom perception. The findings are compatible with current cognitive psychological and neuropsychological approaches to symptom perception and imply that somatic symptom distress is a multidimensional phenomenon that is both strongly linked to but also clearly separable from related constructs.

Keywords: somatic symptom disorder, medically unexplained symptoms (MUS), somatoform disorders, functional somatic syndromes, bifactor model

Between two thirds and three quarters of distressing somatic symptoms presented in primary medical care are not fully explainable by current medical knowledge and consequently represent "medically unexplained" symptoms (MUS; Körber, Frieser, Steinbrecher, & Hiller, 2011). A considerable amount of people with MUS (10–30%) develop chronic and distressing symptom patterns which fulfill the diagnostic criteria of functional somatic syndromes (e.g., fibromyalgia syndrome; Fischer, Gaab, Ehlert, & Nater, 2013) and/or a somatoform disorder according to *DSM–IV*. For the sake of clarity, we apply the term somatic symptom distress in this article. It remains largely unknown which factors contribute to a chronic development of somatic symptom distress and the even more fundamental question of the latent structure of somatic symptom distress remains largely unanswered. Without exact knowledge regarding the type of latent structure and an adequate measurement model, somatic symptom distress remains poorly specified, and research into its causes and correlates is therefore hampered (Deary, 1999). Recent psychometric evidence from taxometric analyses suggests that somatic symptom distress should be considered as a continuous construct (e.g., Jasper, Hiller, Rist, Bailer, & Witthöft, 2012). This implies that the etiology of

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chronic somatic symptom distress most likely follows a complex and multicausal process and that the mechanisms are not qualitatively different from the mechanisms of milder variants of transient somatic symptom experiences. Further knowledge of the factor analytic structure is also directly related to the question whether it is reasonable to distinguish among different kinds of somatic symptom distress, in terms of separate diagnoses or different functional somatic syndromes (e.g., fibromyalgia or chronic fatigue syndrome), or whether the similarities among different somatic symptom distress patterns outweigh their differences (e.g., Wessely, Nimnuan, & Sharpe, 1999). In this latter case, broader diagnostic terms, as recently proposed with the novel diagnosis of somatic symptom disorder in *DSM–5* (American Psychiatric Association, 2013), would be empirically justified. Previous studies on the structure of somatic symptom distress (e.g., Deary, 1999) mostly relied on three kinds of models: (a) a general factor model; (b) a correlated factor model consisting of correlated symptom-specific factors; and (c) a hierarchical model in which the variability of symptoms is explained by lower-order symptom-specific factors, and the associations among these latent symptom-specific factors are accounted for by a higher order general factor. Recently, a fourth type of model has been proposed which can be considered as a mixture of the general model and the correlated subfactor approach. In this so called *bifactor* model (e.g., Brunner, Nagy, & Wilhelm, 2012), every single symptom is explained by two latent factors: a general factor that is related to every symptom, and a second symptom-specific factor that is related to specific groups of symptoms (e.g., pain symptoms or gastrointestinal symptoms). The different latent factors are orthogonal in this model; that is, each latent factor explains the unique variability of a given symptom distress level. A bifactor approach was recently proposed to represent the psychometrically best-fitting measurement model in the realm of somatic symptom distress (Thomas & Locke, 2010; Witthöft, Hiller, Loch, & Jasper, 2013). Although evidence of the statistical superiority of the bifactor model is growing, data on the construct validity of the proposed latent (general and specific) somatic symptom-distress factors is still missing. Thus, the meaning of the different factors remains unclear, that is, how they relate to constructs that are potentially involved in the pathogenesis of somatic symptom distress and theoretical models of symptom perception. According to cognitive-psychological models (e.g., Brown, 2004; Leventhal, 1986), somatic symptom experiences are the consequence of the activation of prior formed symptom representations (*schemata*) in memory. Accordingly, symptom perception represents a constructive cognitive process driven by automatic selection and attention allocation processes to potential bodily signs of symptoms involving a complex interplay of sensory and affective-motivational components. Certain psychological traits that are related to increased body and symptom focused attention are strongly suspected to maintain and amplify the perception of somatic symptom distress. In this regard, health anxiety (i.e., the unsubstantiated and disproportionate fear or conviction to suffer from a severe illness) represents one of the most important related constructs. Additionally, the construct of somatosensory amplification (i.e., the trait-like disposition to experience somatic reactions as more intense and to habitually evaluate them as more negative, noxious, and as evidence of a physical disease) has been proposed as an explanatory construct to account for the development and maintenance of both chronic somatic symptom distress and health anxiety (Barsky, Wyshak, & Klerman, 1990). Theoretically, somatosensory amplification as a rather general construct regarding symptom perception and should be strongly related to the general factor of somatic symptom distress within the bifactor model.

The central aims of this study are twofold: First, the latent structure of somatic symptom distress will be explored by comparing the bifactor model to alternative structural models (the general factor model, correlated factor model, and the hierarchical model).[1] Second, we aim at investigating the construct validity of the proposed latent variables by exploring associations to health anxiety, somatosensory amplification, and functional somatic syndromes (e.g., fibromyalgia, chronic fatigue syndrome, irritable bowel syndrome). Specifically, we hypothesize (a) that the general factor reflects the affective component of somatic symptom distress and should therefore reveal stronger associations to health anxiety and somatosensory amplification compared with the symptom-specific factors and (b) that functional somatic syndromes are equally well predicted by the general somatic distress factor and the symptom-specific factors.

Method

Participants and Measures

Sample 1 (S1) consisted of a total of 1,604 participants who completed a set of questionnaires in the years 2004 to 2005 in the waiting area of the Office of Student Enrollment at a German University. They were asked to take part in a study on environment and well-being. Of the participants, 60.1% were female, and the mean age was 21.8 (SD = 5.81). Most of the participants were university students (83.5%), with chemistry (17.2%) and economics (11.7%) reported as the most frequent academic majors. The study was approved by the local ethics committee. Sample 2 (S2) consisted of participants of a web-based survey at a University in Switzerland. Invitations to take part in the study were sent out via administrators of public colleges and universities in the German-speaking part of Switzerland. A link to the survey website was posted on several student Internet platforms. The participants were asked to take part in a survey on physical and mental well-being. A total of 6,206 participants visited the website and about 51% of them finished the survey. After the exclusion of implausible datasets (regarding duration, age, etc.) and incomplete responses on the Somatosensory Amplification Scale (SSAS), N = 3,053 datasets remained for further analyses. About 73% of the participants were female and the mean age was 24.6 (SD = 5.60). About 76% had at least a high school diploma, 22% a university degree, and the remaining 2% lower school degrees.

Patient Health Questionnaire-15 (PHQ-15; S1 and S2). The PHQ-15 (Kroenke, Spitzer, & Williams, 2002) represents a continuous self-report measure of somatic symptom distress over the previous 4 weeks. The PHQ-15 consists of 15 symptoms (e.g.,

[1] Some of the models that we compared are nested within one another (i.e., hierarchical model within the bifactor and g-factor model within the bifactor model). This implies that one model may be seen as the extension of the other model. Thus, a g-factor model or a hierarchical model do not necessarily contradict a bifactor model. Rather, they should be seen as extensions that help to model the data structures even better.

headaches, dizziness) with three response categories (*not bothered at all, bothered a little,* or *bothered a lot*).

The Whitely Index (WI; S1). The WI represents the most prominent self-report measure for a dimensional assessment of health anxiety. It consists of 14 dichotomous items (*yes* or *no*) and a two dimensional structure (Factor 1: health anxiety; Factor 2: symptoms and illness convictions; Schwarz, Witthöft, & Bailer, 2007).

The Patient Health Questionnaire–9 (PHQ-9; S1 and S2). The PHQ-9 (Kroenke, Spitzer, & Williams, 2001) comprises nine 4-point items that are based on the criteria for depressive disorders in *DSM–IV*. The response format ranges from *not at all* to *nearly every day*. The PHQ-9 is regarded as a valid and reliable scale for depression (Kroenke et al., 2001).[2]

Somatosensory Amplification Scale (SSAS; S2). The German version of the SSAS (e.g., Jasper et al., 2013) was used, which asks the respondent to what extent each of the 10 items is "characteristic of you in general" (Barsky et al., 1990, p. 325) on a 5-point scale, ranging from 1 (*not at all true*) to (*extremely true*). The 10 items mainly ask for uncomfortable bodily sensations, such as Item 5, "Sudden loud noises really bother me" (Barsky et al., 1990, p. 327).

Questionnaire for the Assessment of Functional Somatic Syndromes (FFSS; S2). The FFSS (Nater, Fischer, Latanzio, Ruoss & Gaab, 2011) assesses 17 functional somatic syndromes according to their existing research criteria.[3]

Data Analysis

Confirmatory factor analyses (CFA) and structural equation modeling (SEM) were performed with MPlus Version 6.11 (Muthén & Muthén, 2010). The analyses were conducted with the robust mean and variance adjusted weighted least squares (WLSMV) procedure.[4] Because the chi-square test is sensitive to the sample size and the complexity of the model, we used other descriptive fit measures for the evaluation of the model fit. As an absolute fit index, we chose the root mean square error of approximation (RMSEA). Furthermore, the comparative fit index (CFI) and the TLI (Tucker–Lewis Index) are reported as incremental fit indices. RMSEA values close to .06 and CFI/TLI values close to .95 are considered as indicators of a good model fit (Hu & Bentler, 1999). All coefficients reported (i.e., factor loadings and regression coefficients) represent standardized coefficients. In a first step, CFAs were used to test the model fit of a bifactor model of somatic symptom distress in both samples (S1 and S2). In a second step, SEMs consisting of measurement models and a structural model were computed in order to determine the size of associations between the bifactor model and relevant other constructs (i.e., health anxiety, somatosensory amplification, and functional somatic syndromes). Finally, latent regression models were used to statistically test the specificity of the observed associations by statistically controlling for possible confounding influences of depressive symptoms.

Results

The Latent Structure of Somatic Symptoms in the PHQ-15

Several CFA models were tested on the latent structure of somatic symptom distress: The bifactor model (see Figure 1), consisting of a general symptom distress factor and four orthogonal symptom-specific factors (pain-, gastrointestinal-, cardiopulmonary-, and fatigue-related symptoms) showed an excellent model fit in both samples, S1: $\chi^2(54) = 88.38$, $p = .002$, (CFI = .992; TLI = .989; RMSEA = .020; 90% CI [.012, .028]); S2: $\chi^2(54) = 140.04$, (CFI = .992; TLI = .988; RMSEA = .023; 90% CI [.018, .028]). This model fitted the data significantly better than a hierarchical factor model with one higher order and four lower order factors, S1: $\chi^2(64) = 402.03$, $p < .001$, (CFI = .926; TLI = .909; RMSEA = .059; 90% CI [.054, .065]); χ^2-difference test: $\chi^2(10) = 221.20$, $p < .001$; S2: $\chi^2(64) = 598.28$, $p < .001$, (CFI = .949; TLI = .938; RMSEA = .052; 90% CI [.049, .056]); χ^2-difference test: $\chi^2(10) = 327.23$, $p < .001$. The assumption of a general factor model resulted in rather poor model fit in both samples, S1: $\chi^2(65) = 900.56$, $p < .001$, (CFI = .816; TLI = .780; RMSEA = .092; 90% CI [.087, .097]); S2: $\chi^2(65) = 1658.01$, $p < .001$, (CFI = .849; TLI = .818; RMSEA = .090; 90% CI [.086, .093]).

Associations Between the PHQ-15 Bifactor Model, Health Anxiety, and Depression (S1)

To test the strength of associations between the different somatic symptom factors derived from the bifactor model and health anxiety (assessed with the WI), we computed a SEM consisting of the PHQ-15 bifactor model and a hierarchical model of the WI consisting of a higher order factor (general health anxiety) and the two lower-order factors "health anxiety" and "symptoms and illness convictions." The model revealed a good fit to the data, $\chi^2(213) = 511.37$, $p < .001$, (CFI = .968; TLI = .962; RMSEA = .030; 90% CI [.027, .034]). The health anxiety general factor showed strong associations to the somatic symptom general factor ($r = .622$, $p < .001$, $SE = 0.053$). Additionally, weaker but significant associations were observed between the general health anxiety and the cardio-pulmonary symptom factor ($r = .264$, $p = .001$, $SE = 0.077$), as well as between the general health anxiety and the gastrointestinal symptom factor ($r = .219$, $p < .001$, $SE = 0.047$). Associations between the general health anxiety and the pain symptom factor ($r = .067$, $p = .219$, $SE = 0.054$) and the fatigue symptom factor ($r = .092$, $p = .137$, $SE = 0.062$) did not reach significance. To explore the specificity of the observed associations between somatic symptom distress and health anxiety, we added depressive symptoms to the model by computing a latent regression model in which the PHQ-15 general factor was regressed onto a health anxiety and a depression factor (see Figure 2). The results indicated good model fit, $\chi^2(382) = 899.61$, $p < .001$, (CFI = .965; TLI = .960; RMSEA = .030; 90% CI [.027,

[2] Because the PHQ-9 contains two items ("trouble sleeping" and "feeling tired/low energy") that are also part of the PHQ-15, we used only the remaining seven items for the depression score to avoid item overlap.

[3] (Tension-type headache; globus hystericus; whiplash-associated disorders; temporomandibular disorders; persistent idiopathic facial pain; chronic low back pain; fibromyalgia syndrome; chronic fatigue syndrome; multiple chemical sensitivity; irritable bowel syndrome; functional dyspepsia; chronic abacterial prostatitis; chronic pelvic pain; premenstrual syndrome; premenstrual dysphoric disorder; functional chest pain of presumed esophageal origin; hyperventilation syndrome.)

[4] For model comparisons, we used the DIFFTEST option in MPlus which takes into account that the distribution of the WLSMV based chi-square differences is not itself chi-square distributed.

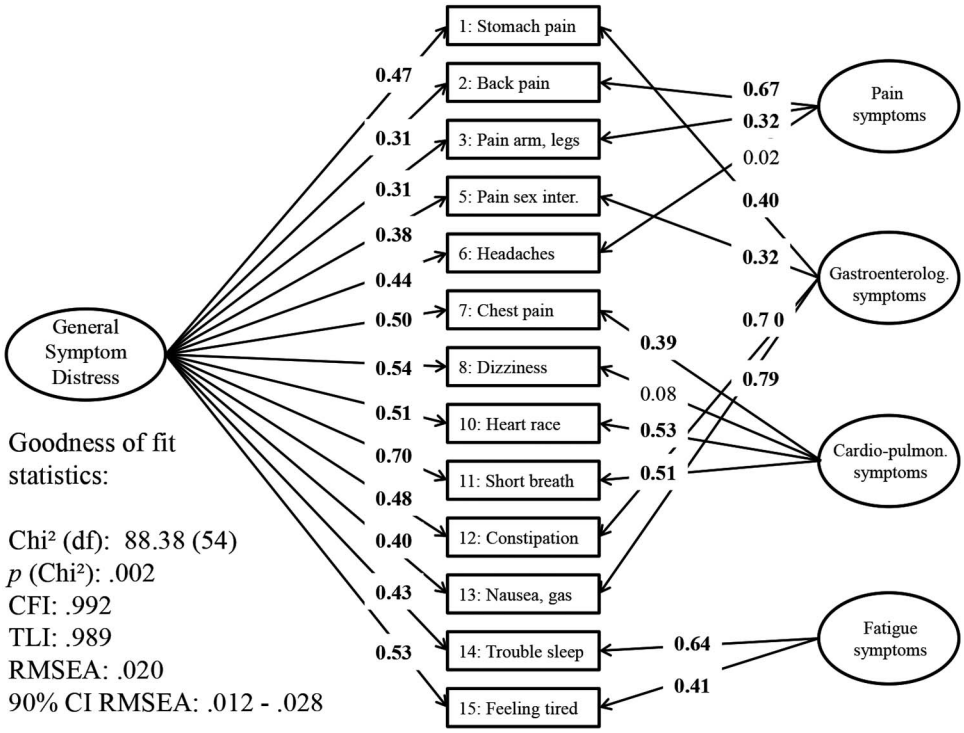

Figure 1. A bifactor model of somatic symptoms in the PHQ-15 in sample 1 (N = 1,520) with standardized factor loadings (circles represent latent variables, squares refer to manifest variables, single headed arrows represent factor loadings; all factor loading coefficients printed in bold are significant at $p < .05$; error terms of manifest variables not shown).

.032]) and yielded significant latent regression coefficients for both health anxiety (β = .407, $p < .001$, SE = 0.089) and depression (β = .542, $p < .001$, SE = 0.061). Together, both constructs account for a total of 67% of explained variance in the general somatic symptom factor. We used the Wald test to compare the strengths of association in this model (H0: the regression weights of health anxiety and depression are of equal size). The Wald test was not significant (p = .333), suggesting that health anxiety and depression represent equally powerful predictors of general somatic symptom distress.

Associations Between the PHQ-15 Bifactor Model and Somatosensory Amplification (SSA; S2)

A SEM containing the PHQ-15 bifactor model and a general factor measurement model for the SSAS obtained good model fit, $\chi^2(211) = 868.94$, (CFI = .966; TLI = .960; RMSEA = .032; 90% CI [.030, .034]), and a strong association (r = .525, $p < .001$, SE = 0.033) between the general somatic symptom factor and the SSAS. Associations between the SSAS and the specific symptoms factors were of small size (pain: r = .066, SE = 0.040; cardiopulmonary: r = .065, SE = 0.050; gastrointestinal: r = .090, SE = 0.035; fatigue: r = .076, SE = 0.041) and only reached significance for the gastrointestinal symptom factor (p = .009). To test for specific associations between SSA and somatic symptom distress beyond depression, we specified a latent regression model in which the PHQ-15 general somatic symptom factor was regressed onto SSA and the depression factor (see Figure 2). Both SSA (r = .400, $p < .001$, SE = 0.047) and depression (r = .556, $p < .001$, SE = 0.037) yielded significant latent regression coefficients and accounted for a total of 65% of explained variance in the general somatic symptom factor. Thus, SSA is specifically related to general somatic symptom distress beyond depressive symptoms.

Associations Between the PHQ-15 Bifactor Model and Functional Somatic Syndromes (S2)

To test possible associations between the different factors of somatic symptom distress and the existence of functional somatic syndromes based on the FFSS, we specified a latent regression model in which binary variables that indicated the existence of specific functional somatic syndromes (e.g., irritable bowel syndrome) and the existence of any functional somatic syndrome were regressed onto the different somatic symptom-distress factors in the PHQ-15 bifactor model. The model yielded an excellent fit to the data, $\chi^2(62) = 168.84$, $p < .001$ (CFI = .990; TLI = .986; RMSEA = .024; 90% CI [.019, .028]) and the general somatic symptom-distress factor turned out to be the strongest predictor for the existence of any functional somatic syndrome (β = .487, $p < .001$, SE = 0.046). Among the specific factors, only the gastrointestinal factor significantly contributed to the prediction of functional somatic syndromes. It was the best predictor of the presence of irritable bowel syndrome (β = .83, $p < .001$, SE = 0.109) and significantly predicted the presence of any functional somatic

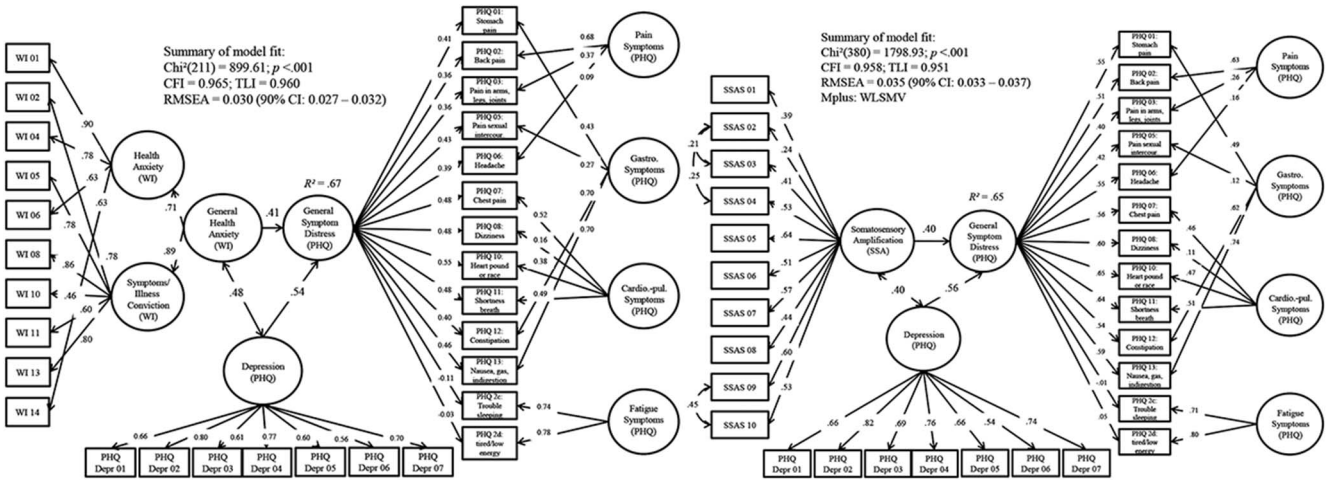

Figure 2. Left panel: Latent regression model (Sample 1) for the prediction of somatic symptom distress, by health anxiety and depressive symptoms; right panel: latent regression model (Sample 2) for somatic symptom distress, somatosensory amplification (SSA), and depression (significant association between SSA and cardiopulmonary symptoms [$r = -.17$, $p = .034$], not shown; all other correlations between SSA factor and symptom-specific factors [$r \leq .09$]; circles represent latent variables, squares refer to manifest variables, single headed arrows between manifest and latent variables represent factor loadings; single and double headed arrows between latent variables represent standardized latent regression and correlation paths, respectively).

syndrome ($\beta = .18$, $p < .001$, $SE = 0.051$). Overall, 28% of the variability of functional somatic syndromes could be explained by the different somatic symptom factors.

Discussion

The primary aims of this study were to psychometrically clarify the structure of somatic symptom distress and to determine possible associations to related constructs, specifically health anxiety, somatosensory amplification, and functional somatic syndromes. A bifactor model consisting of a general somatic symptom distress factor and four symptom-specific factors showed an excellent model fit and psychometrically outperformed alternative structural models. It is important to note, however, that the bifactor model presented here is largely data-driven and therefore not self-evidently informative regarding theoretical considerations about correlates and pathogenetic factors associated with somatic symptom distress. Further SEM analyses therefore focused on possible associations between the dimensions of symptom distress derived from the PHQ-15 bifactor model as well as other relevant constructs: a pattern of strong associations was observed between the general factor (but not the symptom-specific factors) and health anxiety, depressive symptoms, as well as somatosensory amplification. Functional somatic syndromes showed significant associations to both the general somatic symptom factor and well as the symptom-specific factors. The proposed bifactor model appears to be compatible with theoretical considerations differentiating an affective-motivational and a sensory component of symptom perception inherent in the compelling view of somatic symptoms as "homeostatic emotions" (Craig, 2003; Van den Bergh, Bogearts, & Van Diest, 2014). In this sense, the general factor most likely represents the more central affective-motivational and evaluative component of symptom perception (neurophysiologically rooted in the anterior cingulate cortex; Craig, 2003), whereas the specific-symptom factors most likely cover sensory-discriminative aspects of symptom perception which are more specific and informative regarding the exact qualities of the respective symptoms and might be linked to the anterior insular cortex (Craig, 2003). This important distinction between an affective and a sensory component of symptom perception has long been recognized in pain research (Fernandez & Turk, 1992) but somewhat neglected in the research on MUS so far. The presented bifactor model also appears to be compatible with prominent theoretical models on somatic symptom perception, such as the integrative conceptual model (ICM) proposed by Brown (2004) or the common sense model of self-regulation (CSM) by Leventhal and colleagues (e.g., McAndrew, Mora, Quigley, Leventhal, & Leventhal, 2014). Both approaches attribute the experience of somatic symptom distress to distortions in somatosensory awareness caused by the top-down activation of prior formed symptom representations in memory (*schemata*). Both sensory and affective-motivational aspects are parts of these complex cognitive–emotional symptom representations. It is tempting to speculate that the symptom-specific factors of the bifactor model should be closer related to the sensory details of previous symptom episodes, whereas the general factor might be stronger associated with the broader personality trait of negative affectivity and rather serves as an "amplification factor" for existing symptom schemata once they are triggered (e.g., by acute stressors). This hypothesis could easily be tested by linking existing medical records of past and chronic organic illnesses to the different factors of the bifactor model. Although both the ICM as well as the CSM focus on cognitive psychological aspects of symptom perception, it is also possible that the symptom-specific factors of the bifactor model rather reflect alterations in somatic physiological factors (e.g., altered composition of gastrointestinal microbiota associated with irritable bowel syndrome). Future studies should therefore use medical records as well as psychophysi-

ological indicators (including endocrinological and neuronal indicators) to decide whether the symptom-specific factors are related to cognitive sensory aspects of past illness experiences or to alterations in psychophysiology, or to a combination of both (i.e., physiological changes as a consequence of previous illness experiences).

Regarding the current nosological debate, the findings of a strong general factor of somatic symptom distress appears compatible with the broad category of somatic symptom disorder introduced in *DSM–5*. The results of the SEM analyses endorse the notion to define somatic symptom distress and health anxiety as separate entities (it is important to note that health anxiety is not stronger related to somatic symptom distress than depressive symptoms). Regarding functional somatic syndromes, the bifactor approach may represent an elegant way to reconcile the debate about whether qualitatively distinct patterns of somatic symptoms (i.e., different functional somatic syndromes) really exist, or whether the common variance among the different somatic symptoms may outweigh their differences (e.g., Wessely, Nimnuan, and Sharpe, 1999).

Limitations. Several limitations of our study have to be considered: First, and perhaps most important, the current models are based on a comparatively healthy and homogeneous sample of college students that are neither representative of the general population nor of clinical samples.[5] Because the exact factor structure partly depends on the respective sample characteristics (e.g., Haynes, Smith, & Hunsley, 2011), particularly the amount of variance for a respective construct, future studies have to test whether results observed in these rather homogeneous samples generalize to more heterogeneous populations. Although previous studies suggest that the bifactor model of somatic symptoms fits the data well also in samples of the general population and in patient samples (Thomas & Locke, 2010; Witthöft et al., 2013), future studies should explicitly and more rigorously test for the equivalence of the reported factor structure (e.g., by applying measurement invariance methodology), preferably in clinical samples including patients with diverse somatic symptom patterns. A further limitation represents the Internet-based mode of administration. No study has so far demonstrated the psychometric equivalence of the paper–pencil and the Internet version of the PHQ-15 as it has been comprehensively done for other psychometric instruments (e.g., Bagby et al., 2014). However, the observation that the described bifactor model was also found in previous paper–pencil administrations of the PHQ-15 in patients' samples and members of the general population (Thomas & Locke, 2010; Witthöft et al., 2013) endorses the notion that the proposed structure is not simply an artifact of the chosen mode of administration. Finally, the presented models are based on self-report data that do not allow for a definite distinction between medially explained and MUS. Analyses using more elaborate clinician ratings of medically explained versus unexplained somatic symptom distress may come to different conclusions.

[5] Although samples of college students are younger and have more years of formal education compared with the general population, a considerable amount of psychopathology (including MUS) has been reported in college students similar to the general population (Bailer, Schwarz, Witthöft, Stübinger, & Rist, 2008).

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