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A longitudinal bifactor approach to modelling somatic symptom development in psychosomatic treatment

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ABSTRACT

Background: Persistent somatic symptoms display a large public health problem and are often treated or addressed alongside other conditions in psychosomatic medicine units. With growing evidence pointing to unfavorable central nervous processing in persistent symptom perception, we aimed at modelling somatic symptom clusters together with an overarching general factor (g-factor) longitudinally during psychosomatic treatment.

Methods: This study analyses data from the Multicenter Effectiveness Study of Inpatient Psychosomatic-Psychotherapeutic Treatment in German University Hospitals (MEPP, 19 academic psychosomatic medicine departments with inpatient and day clinics, $N = 2094$ patients with ICD diagnoses between F30 and F69). The PHQ-15 was used to construct bifactor models of symptom perception. We used structural equation models to investigate how the g-factor and symptom-group clusters developed from treatment intake over discharge to a one year follow-up. Factor scores were used to evaluate treatment effects on the individual components.

Results: As expected, the highest treatment effects, from a bifactor model perspective, were found for the overarching affective-motivational component ($\eta_{part}^2 = 0.277$) and for fatigue ($\eta_{part}^2 = 0.250$). All components predicted themselves at future timepoints: autoregressions were high, significant, and explained large proportions of the variance (all $\beta > 0.69, p < .001$).

Conclusions: The bifactor model of symptom perception can be longitudinally applied in a large clinical sample with various diagnoses. Psychosomatic treatment showed effects on all components, both those similar to an overarching g-factor and those related to the perception of physiological signals, even when somatic symptoms were not the main target of the treatment.

Many people experience bothersome bodily symptoms in their daily lives. In primary care, these persistent somatic symptoms that are typically not fully explained by clear-cut biomedical causes make up approximately two-thirds of all symptoms that patients report to their doctors [33,50]. If these symptoms are experienced as distressing or disruptive, persist for 6 or more months, and lead to excessive thoughts, feelings and behaviors related to these symptoms, they may merit the diagnosis of a somatic symptom disorder according to the DSM-5 [1], bodily distress disorder according to the ICD-11 [63], or functional somatic disorder according to the European Research Network to Improve Diagnosis, Treatment, and Healthcare for Patients with Persistent Somatic Symptoms [6].

In Germany, functional disorders – often associated with somatic disease, trauma-related and personality disorders – are common diagnoses in psychosomatic-psychotherapeutic treatment settings, and persistent somatic symptoms frequently accompany other disorders seen in this setting. Psychosomatic medicine is an established discipline which employs an integrative, biopsychosocial model of health [16]. It has its own specialist training program; usually operates inpatient, day hospital, and outpatient settings; and works together with other tertiary care units by providing consultation-liaison services [10,64]. The Multicenter-Effectiveness Study of Psychosomatic Medicine and Psychotherapy (MEPP, [12]) was the first concerted effort to assess the inpatient treatment effects of psychosomatic university clinics in Germany. The goal of the MEPP project was to systematically compare different patient groups (e.g., personality disorders, mood disorders, etc.) before, directly after, and one year after their treatment by using standardized instruments and structured interviews. The pre-post treatment effect sizes (measured in terms of domain-specific symptom reduction) are medium to high [13,31], and this success story continues at follow-up [24].

In recent years, persistent somatic symptoms have received more attention [20] and promising neuroscientifically-based explanatory models have been proposed [8,15,39]. The Predictive Processing Framework of brain function posits that our (bodily) experiences are shaped by two sources of information: our previous experiences and resulting expectations in similar situations (“priors” or “internal model”)

and the actual somatic input [35]. In healthy people, the prediction errors between internal model and somatic input are incorporated into future predictions by updating the internal model. However, persistent somatic symptoms are characterized by high negative affect [19,56], which seems to be a necessary but not sufficient condition of the symptoms [3]. In conditions with high negative affect, the internal model of symptoms being health threats can be especially active, leading to cognitive avoidance, decontextualization, no error reduction, and compromised memory. This is described by the Better Safe Than Sorry Model, which demonstrates how this pattern of symptom perception can turn into a vicious circle and lead to chronified/persistent somatic symptoms [54]. This model implies that people with persistent somatic symptoms or functional disorders may also be less accurate at perceiving the exact sensory details of their symptoms [5,47,48,52], while having higher general symptom distress. Experimental studies have found evidence of unfavorable sensory processing in persistent somatic symptoms [4,57]. One experimental paradigm to test the interplay of the internal model and sensory input is the eye-head gaze-shift paradigm, which not only focusses on perceptual processes, but also takes motor planning and execution into account [28]. In this paradigm, participants wear a weighted helmet while performing large gaze shifts, meaning that they need to integrate this newly added inertia into their internal models of motor planning and control to be able to successfully complete the gaze shift. Patients with functional neurological disorders face difficulties shifting away from their unfavorable internal model and show variations in sensorimotor processing [29,43] – their heads oscillated at the end of the gaze shifts, meaning they did not update their internal models despite prediction errors. However, this finding was not successfully replicated in chronic pain, indicating that the modality of the sensory input (here: head movement) needs to fit to the disorder (i.e., functional neurological disorder, functional dizziness) to demonstrate the prediction errors [42].

From a psychometric perspective, the processes forming bodily symptom experiences can be captured well in the bifactor model of symptom perception [61,62]. Here, an overarching general factor (dubbed g-factor) containing the affective component of symptom perception can be distinguished from specific sensory factors of symptom perception. This g-factor is thus not specific to any group of symptoms but instead reflects common underlying mechanisms of somatic symptom perception. It is related to health anxiety, trait negative

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affect, depressiveness, and somatosensory amplification, amongst others [58,61]. This is in line with the conceptualization of somatic symptom perception as an active, constructional process that consists of top-down elements (g-factor) and predominantly bottom-up (sensory) information (symptom-group factors) [58,60]. The bifactor model has been validated in different contexts [7,49,51,65], however, little is known about the longitudinal course of these factors. This would be especially interesting, as it could shed light on how different symptom perception components change during and after psychosomatic treatment. This could have implications for theoretical models of symptom perception, such as the predictive processing models.

The current study aimed at assessing persistent somatic symptoms in psychosomatic patients longitudinally by applying the bifactor models of somatic symptom perception addressing the g-factor as well as symptom-group factors across the three timepoints of the MEPP study. This approach could answer how psychosomatic treatment affects the different components of the bi-factor model of somatic symptom distress. Given that the g-factor of somatic symptom distress is highly affective-motivational, we expected the largest treatment effects on the g-factor compared to the specific, sensory symptom-group factors of symptom perception.

1. Methods

1.1. Design

Per STROBE guidelines, this study is an observational study (Elm et al., 2007) [14].

This study is a secondary data analysis from the Multicenter Effectiveness Study of Inpatient Psychosomatic-Psychotherapeutic Treatment in German University Hospitals [13], which aimed at evaluating the effectiveness of inpatient and day hospital treatment in 19 German university departments of Psychosomatic Medicine and Psychotherapy. Specifically, this comprised patients with affective disorders (ICD-10 codes F30-F39), neurotic, stress-related and somatoform disorders (ICD-10 codes F40-F48), behavioral syndromes associated with physiological disturbances and physical factors (F50-F59), and disorders of adult personality and behavior (F60-F69). The participants were assessed at three timepoints: at intake (T0), in the week of discharge (T1), and at a 1-year follow-up (T2).

Ethical approval was obtained from the ethics committee of the medical faculty of the Ruhr-University Bochum (ID: 18-6388) and confirmed by the ethics committees of the other participating universities. This trial was registered with the German Clinical Trials Register (<https://drks.de/search/de/trial/DRKS00016412>).

Participants were recruited from 01/2019 to 12/2020. Eligible participants who gave informed consent were interviewed by trained clinicians and were asked to fill in a variety of questionnaires.

1.2. Participants

Participants were recruited upon admission to inpatient or day hospital treatment on a psychosomatic ward for any diagnoses between ICD-10-GM F30 to F69 (affective disorders, neurotic, stress-related, and somatoform disorders; behavioral syndromes associated with physiological disturbances and physical factors; and disorders of adult personality and behavior). Participants were eligible if they were above 18 years of age and had sufficient German knowledge. Exclusion criteria were acute psychotic disorders, organic mental disorders, and substance dependency with current use and intoxication (excluding tobacco and prescribed medications). The final sample size was $N = 2094$ participants.

1.3. Measures

At all timepoints, a battery of questionnaires addressing mental

health and biological markers addressing allostatic load were assessed. To answer our research question, we used the Patient Health Questionnaire-15 (PHQ-15) data from the MEPP-study. The PHQ-15 was designed by Kroenke et al. [27] with the goal of creating an assessment tool for the severity and psychological burden of somatic symptoms (somatic symptoms distress). It consists of 15 questions on somatic symptoms in the last 4 weeks and has a response format ranging from 0 (not bothered at all) to 2 (bothered a lot). In this study, we did not use the “menstrual pain” item, as it only applies to menstruating people, and the “fainting spells” item, as it was also omitted in previous literature due to psychometric issues [61]. The cut-off scores of 5, 10, and 15 correspond to low, medium, or high symptom severity, respectively. The German translation is by Löwe et al. [32]. We measured an internal consistency of $\alpha = .79$ at T0.

1.4. Statistical analysis

Data were analyzed using SPSS Versions 23, 27, and 29 (IBM [21–23]), as well as R version 4.4.1 [40]. Within R, we used packages *psych*, *foreign*, *lavaan*, *dplyr*, and *string* [41,45,46,59].

After calculating demographic parameters, we computed a structural equation model with PHQ-15 bifactor models according to [61], at the three timepoints. Each bifactor model consists of an overarching latent factor, called the g-factor, which is constructed out of all 13 remaining PHQ-15 items (as “menstrual pain” and “fainting spells” were omitted due to psychometric reasons), and, orthogonal to this overarching factor, four sensory symptom-group subfactors. These 4 subfactors together are the sensory factor and the second part of the bifactor model. These are:

- Pain, which consists of *back pain, headaches, and pain in arms, legs or joints*
- Gastroenterological and related symptoms, constructed from *stomach pain, pain or problems during sexual intercourse, nausea/gas/indigestion, and constipation, loose bowels, or diarrhea*
- Cardiorespiratory symptoms: *chest pain, dizziness, feeling your heart pound or race, and shortness of breath*
- Fatigue symptoms: *trouble sleeping and feeling tired or having low energy*

Note that these 5 factors are all latent factors, meaning they do not correspond to sum scores but instead are constructed based on the shared covariance of their underlying manifest items. To model the bifactor components longitudinally, the latent factors were used to predict themselves at later timepoints. As bifactor models are orthogonal within one timepoint [44], we decided not to model cross-lagged regressions and to focus on autoregressions. We used a weighted-least squares mean- and variance adjusted estimator (WLSMV) and theta parametrization because of the categorical data. Note that as our goal was to extend findings of the model put forward by [61], we did not inspect modification indices or other potential adaptations that would change the model structure. Additionally, we inspected longitudinal invariance as suggested by Lodder et al. [30].

To better understand the course of the latent components over time, we then computed the latent factor scores and compared the timepoints using ANOVAs. Values were Greenhouse-Geisser corrected if the Mauchly-test for sphericity was significant.

2. Results

2.1. Demographic data

At T0, $N = 2094$ participants were recruited, and we received PHQ-15 data from $N_0 = 2036$ participants. Of these, $N_1 = 1765$ had some PHQ-15 data at discharge. At T2, this dropped to $N_2 = 1270$. While some people dropped out of the trial, other people did not fill in some

questions or instruments. For example, there were $n = 18$ people who did not fill in the PHQ-15 at T0 but did at T1 or even T2.

The people who completed T0 and T1 had almost identical demographic characteristics (People with T0 data: $M_{age_at_T0} = 39.8(SD = 14.2)$, people with T1 data: $M_{ageT0} = 39.8, SD = 14.2$), and those who answered some of the PHQ-15 at T2 were similar ($M_{ageT0} = 40.4, SD = 14.23$). We compared the data between people who answered the PHQ-15 at all timepoints (“completers”) and people who missed at least one timepoint (“non-completers”) and did not find significant differences between the two groups (Welch’s $t(1808.1) = 1.756, p = .079$).

At T0, 68% of participants were assigned female at birth (T1 = 68%, T2 = 69%). Participants were frequently well educated, with approximately a third having a tertiary-entrance high school degree (T0: 30.3%; T1: 30.4%, T2: 33.2%), and about a quarter having a high school degree with vocational training (T0: 23.7%, T1, 23.5%, T2: 22.7%). Most participants were German nationals (90%–91% across all timepoints), followed by Turkish nationals (~1% across timepoints). Most participants received a diagnosis of depressive disorder (83.3%), while 43.2% received a diagnosis of somatoform disorder.

The demographic data are described in more detail in Doering et al. [12].

2.2. Structural equation models

Before checking our hypothesis, we checked the fit as a prerequisite (Fig. 1). The fit was acceptable ($\chi^2_{scaled} = 5399.38, df = 668, p < .001, CFI_{scaled} = 0.89, TLI_{scaled} = 0.88, RMSEA_{scaled} = .06, SRMR = .072$).

As expected, all autoregressions were high – between $\beta = 0.69$ (fatigue T1 ~ T2; 95%CI = [0.62; 0.77], $p < .001$) and $\beta = 0.94$ (pain T0 ~ T1, 95%CI = [0.92; 0.96], $p < .001$). Subsequently, the explained variance was relatively high. The explained variance in the g-factor at T1 was $R^2 = 82.5\%$, meaning that general symptom burden at the end of the treatment can be predicted very well from general symptom burden at intake; at T2, it was $R^2 = 66\%$. Pain was slightly higher at $R^2 = 89\%$ at T1 and $R^2 = 60\%$ at T2. Cardiorespiratory and gastric symptoms at T1 were around 65% explained, and at T2 51% (see Supplement 1). Fatigue was less predictable: at T1, it was $R^2 = 48\%$ explained and at T2, $R^2 = 48\%$ was explained by the preceding timepoint.

Regarding the latent constructions, the relative influence of the individual items stayed similar across the three timepoints, indicating that the bifactor models are quite robust in their construction. Naturally, some items contribute more variance to the latent factors than others – in the case of the g-factor, the headache item seems relatively influential ($\beta_{T0} = 0.66, 95\%CI = [0.62; 0.70], p < .001$, similar values at T1 and T2).

When inspecting longitudinal invariance, we found configural

invariance ($\chi^2_{scaled} = 691.458, df = 171, p < .001, CFI_{scaled} = 0.983, TLI_{scaled} = 0.976, RMSEA_{scaled} = .043, SRMR = .038$). We did not find metric or scalar invariance.

2.3. Factor scores and descriptive data

When looking at the sum scores of the untransformed, non-factorized PHQ-15 sum scores, participants reported symptom intensities of $PHQ - 15_{T0} = 13.97(SD = 5.46), PHQ - 15_{T1} = 10.82(SD = 5.52)$, and $PHQ - 15_{T2} = 11.21(SD = 5.90)$.

The factor scores can be seen in Table 1 and Fig. 2. We conducted repeated measures ANOVAs on the factor scores and unsurprisingly, all 5 factors were all significantly different over time at $p < .001$. General symptom severity (g-factor) had the highest effects it significantly decreased from T0 to T1, and slightly but significantly increased in the period following, $F(1.81, 1955.27) = 1691.90, p < .001, \eta^2_{part} = .610$. Pain symptoms significantly improved from pre- to post-treatment, but increased slightly but significantly afterwards, $F(1.82, 2100.10) = 202.34, p < .001, \eta^2_{part} = .149$, and this was similarly the case for cardiorespiratory symptoms, $F(1.73, 1994.93) = 454.37, p < .001, \eta^2_{part} = .282$, and gastrointestinal symptoms, $F(1.74, 1950.84) = 445.38, p < .001, \eta^2_{part} = .285$. Lastly, for symptoms of fatigue, there was a significant improvement over time: $F(1.95, 2286.33) = 1048.67, p < .001, \eta^2_{part} = .472$. Note that fatigue levels decreased from pre-treatment to discharge, and then they did not change significantly between discharge and follow-up ($p = .90$). In summary, this pattern of results shows that

Table 1
Factor scores of the bifactor components.

		M	SD	95% CI	
				low	high
T0	Cardio	2.12	1.69	1.75	2.49
	Fatigue	3.29	0.93	2.68	3.90
	Gastro	3.47	2.56	2.91	4.04
	Pain	1.78	1.43	1.24	2.32
	g-factor	10.39	4.38	10.19	10.60
T1	Cardio	0.97	0.96	0.76	1.18
	Fatigue	2.14	0.92	1.54	2.74
	Gastro	1.83	1.52	1.47	2.18
	Pain	1.12	0.91	0.78	1.46
	g-factor	4.84	2.49	4.71	4.96
T2	Cardio	1.22	1.22	0.95	1.50
	Fatigue	2.15	1.00	1.50	2.80
	Gastro	2.31	1.87	1.86	2.77
	Pain	1.37	1.04	0.98	1.77
	g-factor	7.09	3.70	6.87	7.31

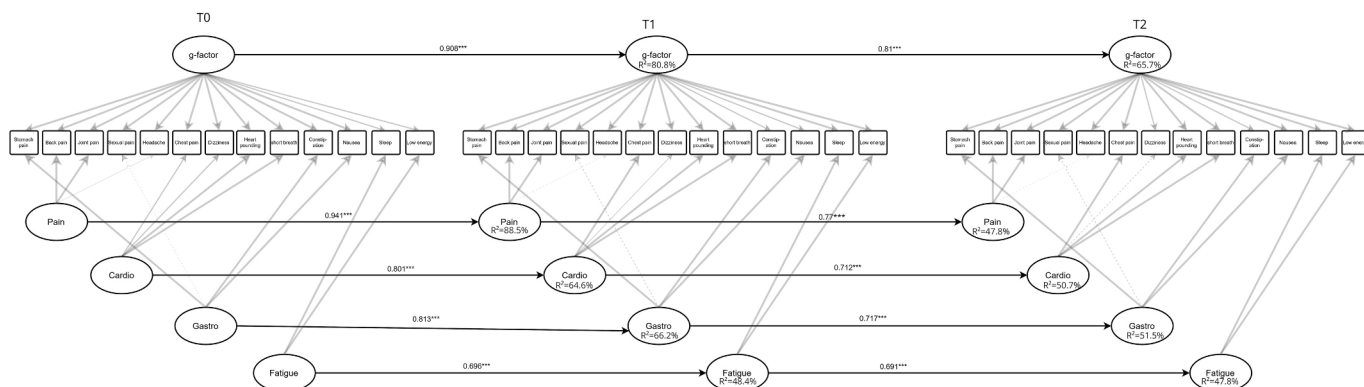


Fig. 1. Longitudinal modelling of the bifactor models and their components. Note. Grey lines signify latent constructions, black lines signify regression paths. *** $p < .001$.

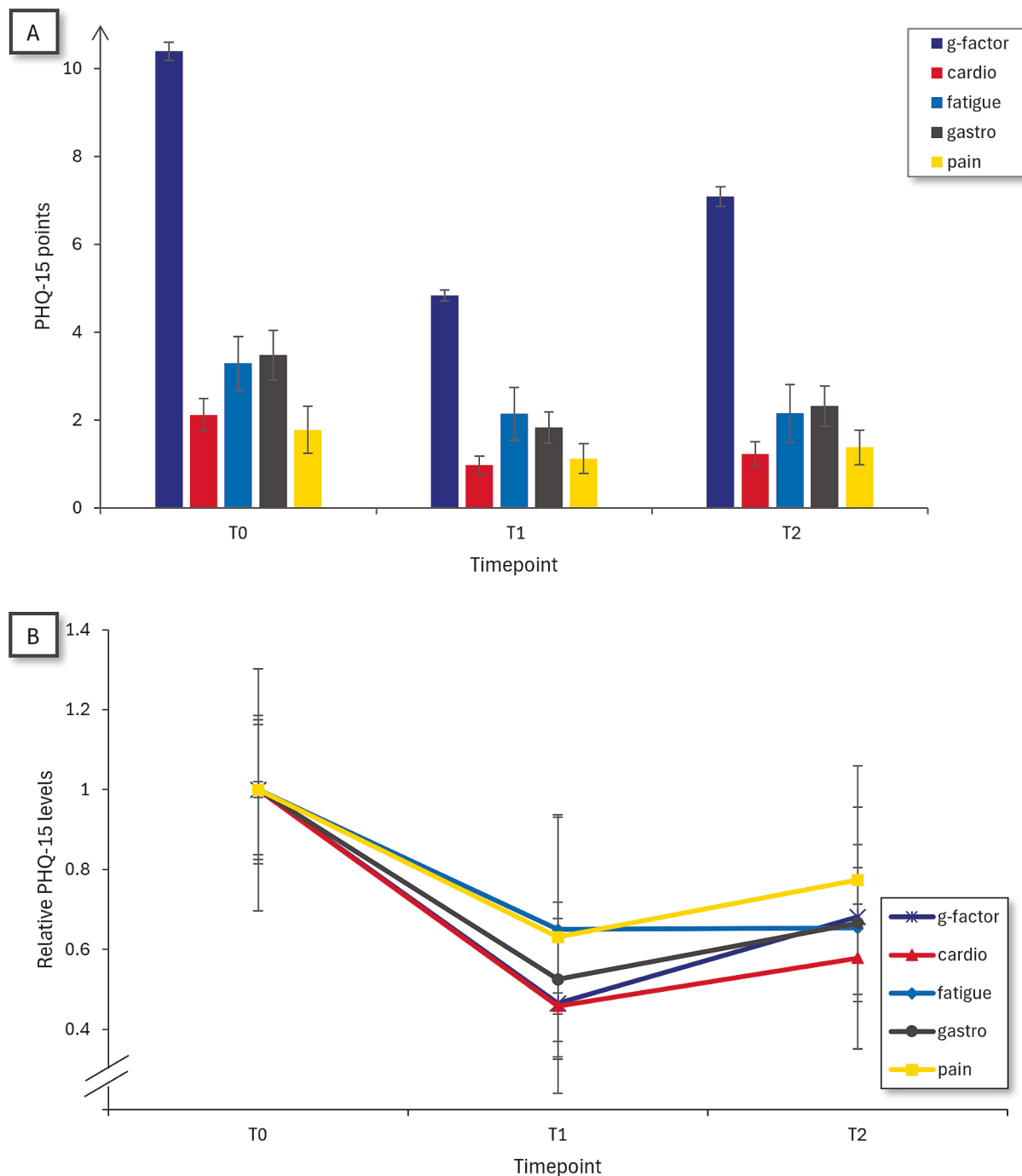


Fig. 2. Course of bifactor component factor scores over time. Note. Panel A shows absolute factor scores. Panel B shows relative PHQ-15 – T0 levels for all components were fixed to 0 and the other timepoints were calculated in relation to these values. PHQ-15: Patient health questionnaire 15; g-factor: overarching motivational-affective g-factor of somatic symptom perception.

the participant's symptom scores declined significantly during therapy, but that there was some increase in symptoms a year later – however, the participants did not have as severe symptoms as at the beginning of their treatment. The strongest treatment effects were found for the g-factor; also, a year after discharge, fatigue levels had not changed.

3. Discussion

This study shows that the bifactor model of somatic symptom distress, which models affective and sensory components of symptom

perception, can be applied to longitudinal outcomes of inpatient treatment in a psychosomatic setting. The advantage of this approach is that it allows for distinguishing changes at different levels of symptom distress, namely at the highly general level of the affective g-factor as well as at the level of the symptom and organ-specific sensory aspects of symptom perception.

In line with our hypothesis, we were able to show that psychosomatic treatment is most effective for the g-factor. The next-largest effect was for symptoms of fatigue. Remarkably, after treatment, fatigue stayed stably low, even after one year. This is in line with the argument that the

bifactor model fatigue component may reflect a depressive symptom component, as the two items used to construct it are also part of the PHQ-9 instrument for depression [26] and this factor is associated with negative affect [58]. Depressive symptoms had the highest pre-post effect sizes in psychosomatic therapy, according to Doering et al. [13]. The g-factor represents a motivational-affective component of symptom perception and might be a momentary “snapshot” of the internal symptom model in the predictive processing model [60]. The large decrease in the g-factor suggests that psychosomatic therapy could affect patients' thoughts, feelings, and behaviors related to somatic symptoms and their bodies, not merely symptom reporting.

There were slight increases in most symptom groups between discharge and follow-up, although these changes never entirely reverted entirely to baseline. This is in line with treatment effects in persistent somatic symptoms [25] and therefore not surprising – after all, the treatment was relatively short (approx. 8 weeks, depending on condition and individual severity) and in an inpatient setting. When patients return to their usual home and work surroundings, it is normal that some people struggle to apply all their new knowledge and techniques from therapy to their everyday lives. This is why it is generally recommended for inpatient treatment to be followed by outpatient sessions, to help generalizing treatment benefits to everyday life [11].

To our knowledge, this is the first study modelling the bifactor approach to somatic symptoms longitudinally. One of our research questions was to find out how the components changed over time, especially in light of treatment. The results here show that symptom severity in one domain at one timepoint is greatly determined by symptom severity in that domain at an early timepoint, as evidenced by the high R^2 values of explained variance. Note that the highest explained variance was found for pain, which was also the component with the lowest treatment effects, indicating that the perception of pain is very stable and might be hard to counteract or change. This is in line with a study in which a purportedly transdiagnostic marker of functional symptoms was not found in pain patients [42]. Seeing the high R^2 values also shows that it is unlikely that there are meaningful symptom or syndrome shifts, which was a common concern in psychodynamic therapies in the last century [17].

The bifactor model expresses what many clinicians know from their own experience: that there can be a mismatch between objective physiology and self-reported symptoms [38,56]. Computational models like the bifactor model are able to inform clinicians and give them heuristics on patient behavioral patterns and thus ideas for treatment [55]. One of the messages from the bifactor model and this paper is that the g-factor can develop independently from sensory symptom-group components, but that both aspects are important for a full clinical understanding of somatic symptom disorder. This fits to studies on the Affect and Symptom paradigm [2], in which people with persistent somatic symptoms were more likely to report more symptoms after negative affect induction [9,37]. Taking all of these findings together, this provides support that the balance between internal model and sensory components can be shifted. While it may be easier to induce or strengthen priors, which appears unfavorable for patients, our study suggests that these priors – which seem related to the g-factor – could also possibly be weakened over time and/or through treatment. Further research is needed on how the bifactor model can be translated into predictive processing terms.

As the bifactor model might align with predictive processing theory on a psychometric level, one might conclude that the patients were successfully treated for their symptoms. This is in line with findings from a more traditional, clinical analysis from the same project by Doering et al. [13]. While our study cannot make conclusions about the reasons for this successful reduction in symptom perception, it could suggest that patients profit from the multimodal inpatient treatment in psychosomatic medicine, combining individual and group psychotherapy sessions with a variety of treatment types. This is likely because the biopsychosocial model can likely be applied to somatic symptoms in such a multifaceted environment, and because these clinics strongly

endorse interdisciplinary approaches. Such settings might be able to integrate predictive-processing-based therapeutic elements, specifically by adapting dysfunctional beliefs – a core tenet of cognitive behavioral treatment [53,54].

One great strength of this study is that the sample is large and representative of the psychosomatic patient population in Germany. It comprises people of various ethnic backgrounds, education levels, and ages, which is rare but important for the external validity of clinical studies. Additionally, when looking at the missing data patterns, there was no indication of problematic missingness patterns pointing to a self-selection bias – missing data seemed independent of demographic parameters.

In this study, we used the model put forward by Witthöft et al. [61], and our findings rely on the validity of that model, as we did not explore further model specifications. A limitation to our work is that we only used PHQ-15 data in this work, and while this study supports this parsimonious approach, clinicians can best understand patients' treatment needs and evaluate their progress by using many different assessment types. Additionally, the study was conducted during routine psychosomatic inpatient care and required sufficient German to understand the questionnaires, meaning that people without any knowledge of German were unable to take part in the study. Thus, this study cannot represent the psychosomatic care needs and treatment trajectories of, for example, non-German-speaking immigrants and refugees living in Germany. Furthermore, we collected sex assigned at birth and not gender, because this is the standard data the German statutory health insurances (used to) require, meaning that some aspects of patients' sex and gender may not be represented well. Also, we were not able to fit random intercept cross-lagged panel models to our data, which would have enabled us to observe within-person variations [18]. However, other authors argue that the random-intercept cross-lagged panel model only reflects temporary fluctuations around the person means and are therefore less useful and more difficult to interpret for causal longitudinal data [34]. We were also not able to detect longitudinal invariance (see [30]), which might indicate some measurement error, or a recalibrating response shift indicating changes in how bodily signals are processed and reported, which could also be a result of the treatment [36]. Lastly, having a (randomized) control group would have allowed us to discern which part of the found effects a natural fluctuation (or “regression to the mean”) and which part can be ascribed to the treatment. Undoubtedly, however, the pre-post-follow-up effects are promising and suggest show that the patients' symptoms improved during treatment.

In conclusion, this study shows that the latent components of somatic symptom perception can be modelled longitudinally, giving valuable insights into the dynamics of symptom perception components over the course of treatment and beyond. In more practical terms, psychosomatic treatments also show large pre-post effects on this level, and effective treatments should consist of elements targeting the motivational-affective side of somatic symptoms, and others targeting the sensory components.

CRediT authorship contribution statement

Tara M. Petzke: Writing – review & editing, Writing – original draft, Visualization, Methodology, Conceptualization. **Ferenc Köteles:** Writing – review & editing, Supervision, Methodology, Conceptualization. **Henrik Kessler:** Writing – review & editing, Project administration, Conceptualization. **Stephan Doering:** Writing – review & editing, Supervision, Conceptualization. **Aram Kehyayan:** Writing – review & editing, Project administration, Conceptualization. **Magdalena Pape:** Writing – review & editing, Project administration, Conceptualization. **Tobias Hofmann:** Writing – review & editing, Project administration, Conceptualization. **Matthias Rose:** Writing – review & editing, Project administration, Conceptualization. **Katrin Imbierowicz:** Writing – review & editing, Project administration, Conceptualization. **Franziska**

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Ethical standards

Ethical approval was obtained from the ethics committee of the medical faculty of the Ruhr-University Bochum (ID: 18–6388) and confirmed by the ethics committees of the other participating universities.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

Yesim Erim and Peter Henningsen are members of the editorial board of JPSR. Given their role as editorial board members, they had no involvement in the peer review of this article and had no access to information regarding its peer review. Full responsibility for the editorial process for this article was delegated to another journal editor. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

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