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Session-level emotional processing and subsequent somatic symptom change in online ISTDP for treatment-resistant somatic symptom disorder: A lagged within-person secondary analysis

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ABSTRACT

Objective: Difficulties in emotional processing are implicated in the development and maintenance of Somatic Symptom Disorder (SSD) and may represent a target for therapeutic change. This study examined whether session-level emotional processing, operationalized as "rise in complex feelings", predicts subsequent reductions in somatic symptoms during online Intensive Short-Term Dynamic Psychotherapy (ISTDP) for treatment-resistant SSD.

Methods: Twenty-five participants with moderate to severe SSD and non-response across two prior intervention phases received up to 16 sessions of online ISTDP ($M = 14.1$) delivered by 17 therapists. After each session, therapists rated rise in complex feelings (0–6), reflecting emotional processing within the therapeutic relationship. Somatic symptom severity was assessed weekly using the Patient Health Questionnaire–15 (PHQ-15). Lagged multilevel models tested whether rise predicted the subsequent PHQ-15 assessment, with person-mean centering isolating within-person effects. A one-sided test evaluated the a priori directional hypothesis.

Results: Higher-than-usual rise in complex feelings was associated with lower somatic symptom severity at the subsequent assessment ($b = -0.22$, 95% CI [−0.43, −0.02], one-sided $p = .016$, two-sided $p = .032$). The association was robust across sensitivity analyses, including reverse temporal ordering. Adjustment for patient-rated emotional activation and therapeutic alliance only modestly attenuated the estimate. The association was observed within-person; between-person differences in average rise were unrelated to symptom outcomes ($b = -0.03$, $p = .809$).

Conclusions: Session-level emotional processing was associated with subsequent reductions in somatic symptoms in online ISTDP, providing preliminary evidence that emotional processing may represent a session-level marker of symptom change in SSD.

1. Introduction

Somatic Symptom Disorder (SSD) is characterized by one or more persistent and distressing physical symptoms accompanied by excessive thoughts, feelings, or behaviors related to those symptoms, regardless of medical explanation [1]. SSD is associated with substantial functional impairment, reduced quality of life, and high healthcare utilization [2]. Psychiatric comorbidity is common, particularly depression and anxiety [3], and chronic presentations are often considered difficult to treat [4].

Growing evidence suggests that difficulties in emotional awareness and regulation may play an important role in SSD. Alexithymia has repeatedly been associated with somatic symptom severity [5], and systematic reviews indicate that individuals with SSD often show impairments in emotional clarity and adaptive emotion regulation strategies [6]. Developmental adversity may contribute to these vulnerabilities. Childhood maltreatment and neglect have been linked to increased somatic symptom severity and somatization in adulthood [7], potentially through disrupted emotion regulation, attachment insecurity, and impaired mentalization capacities [8].

These findings suggest that difficulties in emotional processing may represent an important therapeutic target in SSD. Consistent with this perspective, growing evidence suggests that psychotherapies that focus

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explicitly on emotional processing may improve symptoms and functioning in patients with SSD and related conditions [9–11].

One such approach is Intensive Short-Term Dynamic Psychotherapy (ISTDP), an experiential-psychodynamic model grounded in emotion-focused and attachment-informed treatment principles [12,13]. ISTDP is based on the premise that unprocessed and conflicted emotions—often linked to disruptions in early attachment relationships—can contribute to psychological distress and somatic symptom formation. Such complex feelings (i.e., love, grief, rage and guilt about rage) may activate anxiety and defensive processes that block emotional awareness and manifest through somatic channels such as muscle tension, smooth muscle activation, or cognitive–perceptual disruption.

Compared to traditional forms of psychodynamic psychotherapy, ISTDP is characterized by a more active and emotion-focused stance, with continuous attention to emotional activation, anxiety, and defensive responses in the here-and-now therapeutic relationship. Treatment length varies depending on patient characteristics and capacity, although it is typically shorter than traditional long-term therapy; time-limited formats such as the 16-session online treatment in the present study are common in research settings. Rather than following predefined session themes, treatment is guided by moment-to-moment affective and interpersonal processes emerging during sessions. The therapist actively identifies maladaptive defenses and regulates anxiety to facilitate emotional processing. Within ISTDP theory, facilitating the direct experience of previously avoided complex feelings in the therapeutic relationship is considered a central therapeutic process that may contribute to symptom change.

A growing body of research suggests that ISTDP can reduce somatic symptoms in SSD and related functional disorders, including chronic pain, gastrointestinal disorders, irritable bowel syndrome, and mixed presentations [14]. A recent randomized controlled trial [15] reported large between-group effect sizes ($d = 0.94$ at 8 weeks and $d = 1.54$ at end of treatment) compared with a waitlist control in an emergency department sample of patients with SSD. However, while research indicates that ISTDP can reduce somatic symptoms, it remains unclear whether the emotional processing mechanism proposed by the model is associated with changes in somatic symptoms session-to-session.

Within the ISTDP framework, the depth of emotional processing achieved during therapy has been operationalized using the concept of "rise in complex feelings." Abbass [12] formalized this construct as reflecting the degree of emotional mobilization and breakthrough occurring within a particular session. At lower levels of rise, anxiety and defensive processes dominate, and evidence of patient emotional mobilization is absent or minimal. As rise increases and the combination of resistance and anxiety lessens, the unconscious therapeutic alliance becomes more prominent, allowing patients to experience increasingly intense emotional responses related to significant attachment figures and conflicts. In the model, rise reflects the dynamic balance between resistance and emotional activation during therapy and represents fluid in-session process rather than a stable patient characteristic.

Although the concept of "rise in complex feelings" occupies a central place in ISTDP theory and clinical practice, empirical research examining its role as a mechanism of change remains limited [16]. Previous studies have found that higher levels of rise—often operationalized as an "unlocking of the unconscious"—are associated with favorable treatment outcomes in depression [17], generalized anxiety [18], and mixed psychiatric samples [19,20]. However, most studies have focused on peak levels of rise during treatment rather than session-to-session variations in emotional processing. In one notable exception, Town and colleagues [21] found that higher-than-usual in-session emotional experiencing predicted lower depressive symptoms one-week later, but only when patient personality severity and session-level insight or alliance were considered. Consequently, the theorized link between rise in complex feelings and symptom reduction remains unclear. To our knowledge, no study has examined whether session-level variations in emotional processing predict subsequent changes in somatic symptoms specifically.

Testing whether in-session rise in complex feelings predicts symptom change requires a within-person analysis. The relevant question is not whether patients who generally achieve higher rise improve more (a between-person comparison), but whether a patient who achieves higher-than-usual rise in a specific session subsequently shows greater symptom reduction. This distinction is important because between-person associations may reflect therapist calibration, patient characteristics, or other factors unrelated to the proposed mechanism [22,23]. Examining session-to-session fluctuations within individual treatment courses therefore provides the most direct test of the process–outcome link predicted by ISTDP theory.

In the present study, we test the a priori directional hypothesis that higher session-level rise is associated with subsequent reductions in patient-reported somatic symptoms. Using data from a recent trial of online ISTDP for treatment-resistant SSD [24], we estimate lagged within-person multilevel models with person-mean centering to separate session-level fluctuations from between-person differences. By examining session-level variations in emotional processing and their temporal relationship to symptom change, this study tests a process–outcome association proposed by ISTDP theory while also contributing to broader research on emotional processing in psychotherapy for somatic symptoms [25].

2. Method

2.1. Design

This study is part of the KOSMOS project, a longitudinal research program evaluating sequential emotion-focused online interventions for individuals with persistent physical symptoms. The present analysis uses data from KOSMOS 3, which examined online ISTDP for participants who had not improved during earlier project phases (i.e. KOSMOS 1 and 2) [24].

KOSMOS 3 was preregistered on ClinicalTrials.gov (NCT06682104) and used an interrupted time-series design in which each participant served as their own control. The current secondary process-outcome analyses were not separately preregistered but were guided by a priori hypotheses derived from ISTDP theory. This manuscript was prepared in accordance with STROBE recommendations for observational research.

2.2. Participants

Twenty-five adults meeting criteria for moderate to severe SSD participated in KOSMOS 3. All participants had completed the earlier KOSMOS treatment phases, consisting of a psychiatric assessment, a structured Life Stress Interview and an internet-delivered Emotional Awareness and Expression Therapy program [26,27].

KOSMOS 3 was offered to participants who showed the least favorable symptom trajectories during the KOSMOS 1 and 2 treatment periods (approximately 60 weeks in total) and were reassessed for eligibility before entering the ISTDP phase. Participants continued their usual medical care throughout the project, including psychopharmacological treatment when prescribed, but were not permitted to engage in concurrent psychotherapy outside the KOSMOS project. The sample consisted primarily of middle-aged women with longstanding somatic symptoms, including fatigue, chronic pain, gastrointestinal complaints, migraine or severe headaches. Psychiatric comorbidity was common and Global Assessment of Functioning scores indicated moderate impairment (see [24] for further details regarding participant selection and sample characteristics).

2.3. Treatment and therapists

ISTDP was delivered via videoconferencing in a time-limited format,

comprising one extended initial session (up to 120 min) followed by 15 weekly sessions (50–60 min).

Scheduling flexibility was permitted within a 21-week treatment period. Rather than following predefined session themes, treatment was guided by therapists' moment-to-moment assessment of affective and interpersonal processes within sessions, with the aim of facilitating emotional processing in relation to individual patients' presenting symptoms. The therapists followed a treatment manual [28] and had access to a weekly 2-h online peer supervision group led by the first author. Supervision involved review of recorded sessions, including discussion of rise in complex feelings.

A total of 17 therapists (mean age = 46.3 years; 58.8% female) provided the treatment, including 14 psychologists and 3 physicians. All had completed a three-year ISTDP core training before entering the study. Nine therapists treated one participant each and eight treated two participants, accounting for all 25 participants. Participants attended an average of 14.1 ISTDP sessions (SD = 3.9; range = 3–16); three participants dropped out during treatment, whereas 22 completed the treatment phase and provided post-treatment assessment data.

3. Measures

3.1. PHQ-15

The Patient Health Questionnaire–15 (PHQ-15) [29] is a self-report measure of somatic symptom severity. It includes 15 items assessing how bothersome common somatic symptoms (e.g., headaches, back pain, gastrointestinal distress) have been, rated on a 0–2 scale. The standard PHQ-15 uses a two-week recall window; in this study, the recall period was shortened to one week to match the weekly assessment schedule. Sum scores range from 0 to 30, with established severity thresholds (0–4 minimal, 5–9 low, 10–14 medium, 15–19 high, 20–30 very high).

Participants completed the PHQ-15 pre-treatment, weekly during the ISTDP treatment phase (16 assessments), and at post-treatment via an online platform. The weekly assessments were distributed according to a fixed schedule determined by the research protocol rather than by the timing of therapy sessions. Of 450 possible observations (25 participants × 18 assessment occasions: pre-treatment, 16 weekly assessment, and post-treatment), 412 (91.6%) were available. Internal consistency varied between $\alpha = 0.48$–0.78 across assessments.

3.2. Rise rating

Rise is a therapist-rated measure of in-session emotional activation based on the ISTDP framework [12]. Therapists rated each session on a 0–6 ordinal scale representing progressively intense levels of emotional processing, ranging from minimal emotional activation (0, low rise) through increasing levels of mobilization (1–2) to breakthroughs involving conscious experience of complex feelings toward attachment figures (3–6; see Supplementary Table S1). In the present sample, the observed range was 0–5. Rise was treated as a continuous variable in the statistical models, consistent with common practice for ordinal scales with sufficient number of ordered categories. Rise ratings were available for 346 of 400 possible session observations (25 participants × 16 sessions).

Ratings were completed by the 17 therapists immediately after each session. Inter-rater reliability was not available because therapists rated only their own sessions. The analyses focus on within-person fluctuations in rise and use person-mean centering (see Statistical Analysis), which removes stable between-therapist calibration differences from the within-person estimates. However, session-level biases within therapists (e.g., halo effects or expectancy, such as rating rise higher after sessions perceived as successful) cannot be ruled out. Because the analyses rely on session-to-session variation in rise, valid inference depends on therapists' ratings reflecting genuine variation in emotional processing rather than overall impressions of session quality. To assess whether rise primarily varied at the session level rather than reflecting stable participant or therapist differences, we conducted a three-level variance decomposition of rise scores (sessions nested within participants within therapists; see Supplement). The decomposition suggested that 70.1% of variance was at the session level, 29.9% at the therapist level, and 0.0% at the patient level. The predominance of session-level variance is consistent with the intended use of rise as a session-level process measure, although the absence of independent blinded ratings prevents direct assessment of inter-rater reliability or session-level halo effects. Additional analyses also controlled for patient-rated emotional activation and therapeutic alliance to assess alternative explanations (see below).

3.3. Patient-rated emotional activation

After each session, participants rated their general emotional activation on a visual analogue scale ranging from 0 ("*This week's session evoked no feelings at all*") to 10 ("*This week's session evoked very strong feelings*"). This measure was included in exploratory analyses examining the extent to which the rise–symptom association was distinct from general emotional arousal.

3.4. Patient-rated therapeutic alliance

Therapeutic alliance was assessed after each session using the patient-rated Session Rating Scale (SRS; [30]), a four-item measure assessing the relationship bond, agreement on goals and topics, and the overall therapeutic approach. Items are rated on visual analogue scales and summed to yield a total score ranging from 0 to 40, with higher scores indicating stronger alliance. This measure was included in exploratory analyses examining alternative explanations of the rise–symptom association.

4. Procedure and data preparation

The weekly assessments of PHQ-15 were distributed via an online platform on a fixed schedule determined by the research protocol. ISTDP sessions were scheduled approximately weekly, although the actual spacing varied across participants and over the course of treatment (median inter-session interval = 7 days; range = 1–25 days). As a result, PHQ-15 assessments and therapy sessions were not always synchronized, and the PHQ-15 for a given nominal week could be completed several days before or after the corresponding therapy session.

To align the two time series for lagged analyses, PHQ-15 values were linearly interpolated to each participant's therapy session dates (see Supplement for details). This produced a dataset with one observation per participant per session containing both the observed rise rating and the estimated PHQ-15 value for that date. Interpolation quality was high: 96% of session dates had a PHQ-15 observation within 7 days (median gap = 1 day), and only 3 of 346 observations (0.9%) required extrapolation.

After synchronization of rise ratings and PHQ-15 assessments, 346 session observations were available across the 25 participants. For each session, the outcome was the next available PHQ-15 assessment. For participants who completed treatment, the post-treatment PHQ-15 served as the outcome for the final treatment session. Participants who dropped out of treatment before post-treatment assessment contributed no final-session outcome. This yielded the primary lagged analytic sample of 343 observations. The derivation of the analytic sample is summarized in Supplementary Table 2.

4.1. Statistical analysis

To examine whether emotional processing predicted subsequent symptom change, we estimated lagged multilevel models predicting the

subsequent PHQ-15 assessment from rise at session *s*, establishing temporal ordering between process and outcome variables. Within- and between-person effects were disaggregated by decomposing predictors into within-person components (session-level deviations from each participant's mean) and between-person components (participant means) using person-mean centering [22,23]. The outcome variable was also person-mean centered so coefficients reflect session-to-session covariation within individuals rather than stable between-person differences (see Supplement for details).

We fit two-level models (sessions nested within participants) including three predictors: PHQ-15 at session *s* (autoregressive term) controlling for current symptom level, rise at session *s* as the predictor of primary interest, and session number capturing linear time trends across treatment. The derivation of the lagged analytic sample from the 25 × 16 = 400 possible session observations, including missing rise ratings, interpolation versus extrapolation, and the pairing of each participant's final session with the post-treatment assessment, is summarized in Supplementary Table 2.

Three model specifications were estimated. Model 1 used grand-mean centered predictors without within–between decomposition. Model 2, the primary prespecified model, implemented the full within–between decomposition described above: the outcome and lagged PHQ-15 were person-mean centered, rise was decomposed into within- and between-person components, and person means of both rise and PHQ-15 were included as between-person covariates. Model 3 was a simplified within-person specification including centered rise, centered lagged PHQ-15, and session number without between-person covariates.

ISTDP theory predicts that deeper emotional processing (higher rise) should be associated with subsequent symptom reduction [12,13]. We therefore specified a one-sided test of the within-person rise coefficient, testing the hypothesis that higher-than-usual rise at session *s* is associated with lower PHQ-15 at the subsequent assessment. One-sided *p*-values are reported for the rise coefficient, while the conventional two-sided *p*-value is also reported for the primary model for transparency. All other predictors were tested two-sided. All models were estimated using restricted maximum likelihood (REML), with Satterthwaite degrees of freedom.

4.2. Sensitivity analyses and tests of alternative explanations

We conducted several sensitivity analyses to evaluate the robustness of the results (see Supplement for details). These included residual diagnostics, exclusion of outliers (observations with |scaled residual| > 3), inclusion of a random slope for the within-person rise term, adjustment for the number of days between sessions, testing for lag-1 autocorrelation of residuals, and person-specific detrending of both rise and PHQ-15 to reduce the possibility that shared temporal trends accounted for the observed association.

In addition, we examined three alternative explanations for the rise–PHQ-15 association (see Supplement for details). First, to evaluate reverse temporal ordering, we estimated a reverse-lag model predicting rise at session $s + 1$ from PHQ-15 at session *s*. Second, to examine whether the rise association was related to general emotional activation, we re-estimated the primary model controlling for patient-rated in-session emotional activation. Third, to examine the relationship between rise and therapeutic alliance, we re-estimated the primary model controlling for session-level patient-rated therapeutic alliance (Session Rating Scale; SRS) and tested a Rise × SRS interaction to examine whether rise and alliance operated synergistically.

4.3. Software

All analyses were conducted in R version 4.5.2 [31] using the *tidyverse* [32], *lme4* [33], and *lmerTest* [34] packages.

5. Results

5.1. Descriptive statistics

Table 1 presents sample and treatment characteristics. At baseline, the mean PHQ-15 score was 13.0 (*SD* = 3.1; range = 9–20), corresponding to medium somatic symptom severity. At post-treatment, the mean was 10.1 (*SD* = 4.2), with a mean pre–post change of −2.9 (*SD* = 3.7) among the 22 participants who completed the treatment phase and provided post-treatment assessment data. Participants contributed a median of 16 sessions with rise data (range = 3–16). The most common rise rating was 1 (mid rise; 34.1%), followed by 4 (partial unlocking; 21.1%) and 3 (first breakthrough; 18.5%). Overall, 41.6% of sessions achieved at least a first breakthrough (rise ≥3).

Fig. 1 displays individual and mean trajectories for both interpolated PHQ-15 and rise across sessions.

5.2. Descriptive association between rise and subsequent PHQ-15 change

Table 2 and Fig. 2 present the mean interpolated PHQ-15 change from session *s* to the subsequent assessment as a function of rise level at session *s*. After low and mid rise sessions (0–1), symptoms were essentially unchanged or slightly worsened (*M* = +0.41 and +0.14, respectively). After sessions rated 2 or higher, symptoms decreased on average, with mean changes ranging from −0.33 to −0.69. This pattern suggests that higher rise levels tended to be followed by symptom reduction, although session-to-session variability was large relative to the mean differences (*SD*s = 1.72–3.13).

5.3. Lagged multilevel models

Table 3 presents the results of the three lagged multilevel models. In the primary model (Model 2), the within-person rise effect was associated with lower subsequent PHQ-15 scores ($b = -0.22$, $SE = 0.10$, 95% CI [−0.43, −0.02], one-sided $p = .016$, two-sided $p = .032$). This indicates that when rise in a session was one unit higher than the

Table 1
Sample and treatment characteristics.

Characteristic	Value
Sample	
Enrolled, *N*	25
Completers, *n*	22
Dropouts, *n*	3
PHQ-15 somatic symptom severity	
Baseline (*N* = 25, *M* (*SD*)	13.0 (3.1)
Baseline, range	9–20
Post-treatment (*n* = 22), *M* (*SD*)	10.1 (4.2)
Post-treatment, range	9–20
Pre–post change, (*n* = 22), *M* (*SD*)	−2.9 (3.7)
ISTDP sessions	
Sessions with rise data, M (SD)	13.8 (3.9)
Sessions with rise data, Mdn	16 (3–16)
Rise distribution across 346 sessions	
0 – Low rise, *n* (%)	26 (7.5)
1 – Mid rise, *n* (%)	118 (34.1)
2 – High rise, *n* (%)	58 (16.8)
3 – First breakthrough, *n* (%)	64 (18.5)
4 – Partial unlocking, *n* (%)	73 (21.1)
5 – Major unlocking, *n* (%)	7 (2.0)

Note. PHQ-15 = Patient Health Questionnaire–15 (range 0–30; higher = more severe). Rise = therapist-rated degree of emotional activation and breakthrough (0–5). Three participants dropped out during treatment (after 3, 4 and ~10 sessions, respectively) and did not provide post-treatment data.

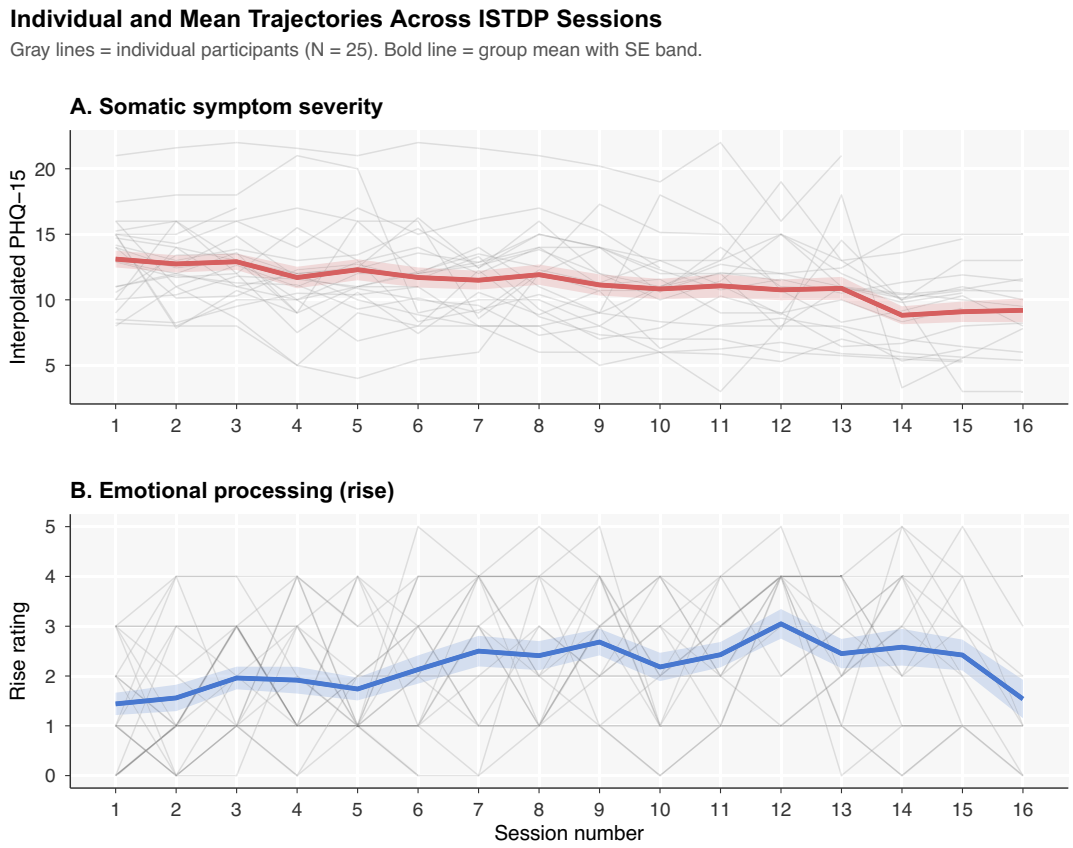

Fig. 1. Individual and mean trajectories across ISTDP sessions. Panel A shows interpolated PHQ-15 somatic symptom severity; Panel B shows therapist-rated rise. Gray lines represent individual participants ($N = 25$). Bold colored lines show the group mean with standard error bands. PHQ-15 values were linearly interpolated to session dates to synchronize with rise ratings.

Table 2
Mean interpolated PHQ-15 change from session s to the subsequent assessment by rise level at session s.

Rise	Label	*n*	*M*	*SD*
0	Low rise	26	+0.41	2.55
1	Mid rise	117	+0.14	3.13
2	High rise	57	−0.55	2.16
3	First breakthrough	63	−0.56	2.12
4	Partial unlocking	73	−0.33	2.02
5	Major unlocking	7	−0.69	1.72

Note. PHQ-15 change = PHQ-15 at the subsequent assessment minus interpolated PHQ-15 at session *s*. Negative values indicate symptom improvement. *n* = number of session-to-session transitions at each rise level across all participants.

participant's personal average, PHQ-15 at the subsequent assessment was on average 0.22 points lower. The within-person autoregressive effect was significant ($b = 0.34$, $p < .001$), indicating moderate session-to-session symptom stability. Session number was also significant ($b = -0.12$, $p < .001$), reflecting a general downward trend in symptoms across the treatment course.

The between-person terms in Model 2 were both non-significant: neither a participant's mean rise ($b = -0.03$, $p = .809$) nor their mean PHQ-15 level ($b = 0.03$, $p = .383$) predicted PHQ-15 at the subsequent assessment, indicating that the association operated at the within-person level. Model 3 (within terms only) yielded virtually identical results (rise $b = -0.22$, one-sided $p = .016$) and Model 1 also showed a significant rise effect (one-sided $p = .020$). In the centered models (Models 2–3), the random intercept variance was zero, which is consistent with the person-mean centering approach that removes between-person variance. Adding a random slope (zero variance, singular fit) and including session spacing as a covariate ($b = -0.22$, $p = .018$) all yielded virtually identical results.

Additional sensitivity analyses (see Supplement) showed no substantive change. Excluding six outlying observations ($b = -0.21$, one-sided $p = .010$) and person-specific detrending—removing each participant's own linear time trends from both rise and PHQ-15—produced similar results ($b = -0.22$, one-sided $p = .017$). Fig. 3 displays the rise coefficient across all model specifications and sensitivity analyses, illustrating its consistency ($b \approx -0.22$).

Complementary person-level analyses among treatment completers showed no significant associations between overall PHQ-15 change and mean rise, maximum rise, or proportion of breakthrough sessions (see Supplement for details). Although confidence intervals were wide, this pattern was consistent with the primary finding that rise primarily operated as a within-person session-level process.

5.4. Tests of alternative explanations

Three analyses examined alternative interpretations of the rise–PHQ-15 association (see Supplement for details). A reverse-direction check showed that PHQ-15 at session *s* did not predict rise at session $s + 1$ ($b = 0.04$, $p = .155$), providing no clear evidence of reverse temporal ordering. When patient-rated general emotional activation was added as a within-person covariate ($n = 327$), the rise coefficient was modestly attenuated ($b = -0.19$, one-sided $p = .040$; an 8% reduction), while emotional activation itself did not predict subsequent symptom change ($b = -0.05$, $p = .546$). When session-level patient-rated therapeutic alliance (SRS) was added as a within-person covariate ($n = 327$), the rise coefficient was also somewhat attenuated ($b = -0.19$, one-sided $p = .039$; a 10% reduction), while within-person SRS was independently associated with lower subsequent PHQ-15.

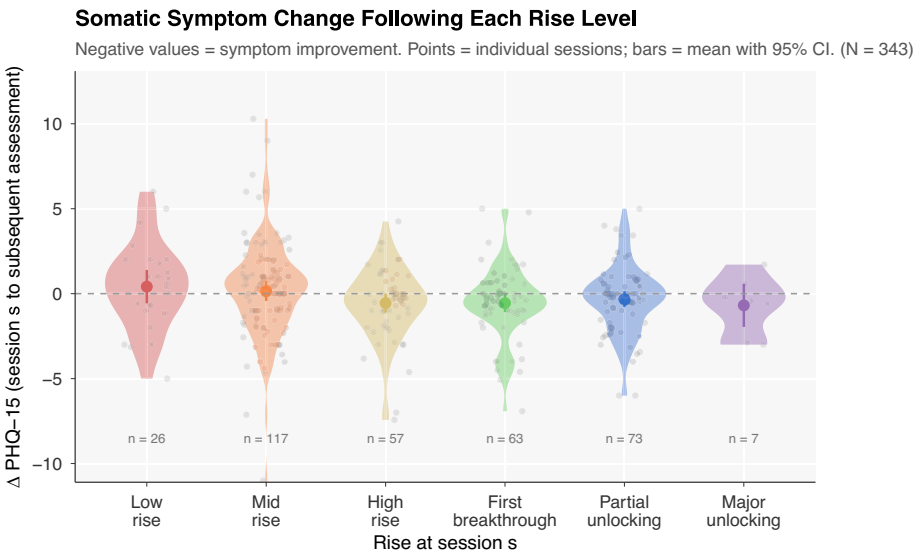

Fig. 2. Somatic symptom change following each rise level. Each point represents a session-to-session transition for an individual participant. Violin shapes show the distribution of PHQ-15 change at each rise level. Filled circles with error bars indicate the mean and 95% confidence interval. Negative values indicate symptom improvement.
After low and mid rise sessions (0–1), symptoms were essentially unchanged or slightly worsened. After sessions rated 2 or higher, symptoms decreased on average.

Table 3
Lagged multilevel models predicting the subsequent PHQ-15 assessment from current session rise.[a]

	Model 1	Model 2	Model 3
Predictor	*b (SE)*	*b (SE)*	*b (SE)*
Intercept	11.17 (0.37)***	0.43 (0.60)	0.77 (0.25)**
PHQ-15[b]	0.43 (0.05)***	0.34 (0.05)***	0.34 (0.05)***
Rise[c]	−0.22 (0.10)*	−0.22 (0.10)*	−0.22 (0.10)*
Rise (between)[d]		−0.03 (0.14)	
PHQ-15 (between)[e]		0.03 (0.04)	
Session number	−0.11 (0.03)***	−0.12 (0.03)***	−0.12 (0.03)***
$\sigma^2_{\text{intercept}}$	3.00	0.00	0.00
$\sigma^2_{\text{residual}}$	4.42	4.18	4.16

Note. $N = 343$ lagged observations from 25 participants (each participant's final session paired with the post-treatment PHQ-15; see Method). Outcome is PHQ-15 at the subsequent assessment (raw in Model 1; person-mean centered in Models 2–3). Model 1 uses grand-mean centered predictors; the intercept represents the predicted subsequent PHQ-15 at sample-average levels of all predictors. Model 2 is the primary model with full within/between decomposition. Model 3 includes only within-person terms.
$^*p < .05$. $^{**}p < .01$. $^{***}p < .001$.
Rise is tested one-sided (directed hypothesis: $b < 0$); all other predictors are tested two-sided.
[a] Primary prespecified model.
[b] Lagged PHQ-15 at session *s* (grand-mean centered in Model 1; within-person centered in Models 2–3).
[c] Rise at session *s* (grand-mean centered in Model 1; within-person centered in Models 2–3).
[d] Person mean of rise across all sessions.
[e] Person mean of interpolated PHQ-15 across all sessions.

($b = -0.08$, $p = .047$). The Rise × SRS interaction was non-significant ($p = .655$), indicating no evidence that rise and alliance interact in predicting subsequent symptoms. The rise point estimate remained in the expected direction and was only modestly attenuated in both analyses (see Supplement for details).

6. Discussion

This study examined whether session-level emotional processing is associated with subsequent somatic symptom change in online ISTDP for treatment-resistant SSD. Consistent with the directional hypothesis derived from ISTDP theory, sessions with higher-than-usual rise in complex feelings were followed by lower PHQ-15 scores at the subsequent assessment ($b = -0.22$, one-sided $p = .016$, two-sided $p = .032$). The association operated at the within-person level and was consistent across model specifications and sensitivity analyses. Between-person differences in average rise were unrelated to outcomes. To our knowledge, this is the first study to examine a session-level process–outcome association between emotional processing and somatic symptom change in ISTDP.

The within-person specificity of the finding represents a key theoretical contribution. ISTDP theory proposes that symptoms are maintained by the avoidance of complex feelings toward significant attachment figures, and that experiencing these emotions in therapy may alleviate somatic symptom formation [12,13]. Rise captures this process: the degree to which complex attachment-related feelings are mobilized and experienced within a session. The present findings indicate that it is the occurrence of such emotional processing in particular sessions—not a patient's general tendency toward breakthroughs—that is associated with subsequent symptom change.

The observed effect size (−0.22 PHQ-15 points per unit of rise) should be interpreted in context. Standardized at the within-person level ($\beta = 0.10$), the effect falls between the medium (0.05) and large (0.11) benchmarks established by Orth et al. [35] for the random intercept cross-lagged panel model—the design most analogous to our person-mean centered approach. This magnitude is particularly meaningful given two sources of systematic attenuation: somatic symptoms show substantial temporal inertia across time, and interpolating patient-reported symptom assessments to therapist-rated session dates introduces measurement noise. Both factors bias lagged associations toward zero, suggesting the present estimate is conservative. Rise, moreover, captures only one dimension of the therapeutic process—the depth of experience of complex feelings—leaving other within-session factors unaccounted for. That a single therapist-rated process variable nonetheless predicted subsequent symptom change above this attenuated floor suggests a meaningful signal. Across treatment, repeated mobilization and breakthroughs to complex feelings may contribute to the improvements observed in ISTDP trials more broadly [14,15].

Additional analyses provided tentative support for the distinctiveness of the rise construct. First, patient-rated emotional activation did

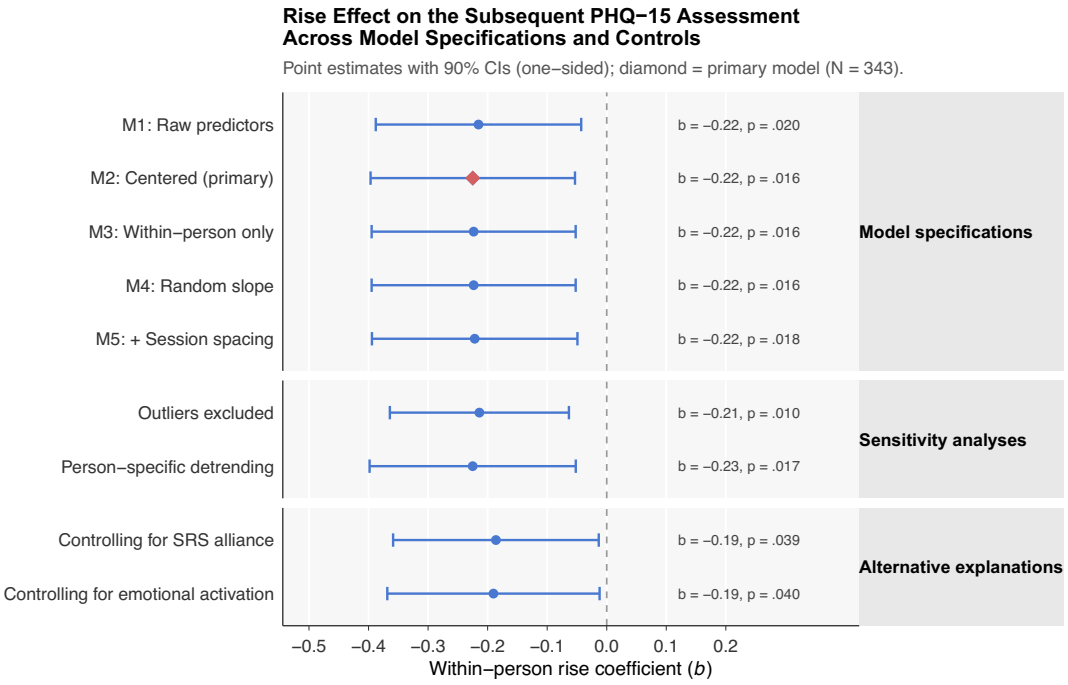

Fig. 3. Within-person rise coefficient across model specifications, sensitivity analyses, and alternative-explanation controls. Error bars represent 90% confidence intervals, corresponding to the one-sided hypothesis tests used for the rise coefficient. The diamond highlights the primary model (Model 2: fully centered within/between decomposition). The rise coefficient was virtually identical ($b \approx -0.22$) across all model specifications and sensitivity analyses (outlier exclusion, person-specific detrending). When controlling for additional session-level variables—patient-rated therapeutic alliance (SRS) and emotional activation—the coefficient was modestly attenuated but remained stable and in the predicted direction. All p-values are one-sided (directed hypothesis: $b < 0$).

not independently predict subsequent symptom change. Controlling for emotional activation modestly attenuated the rise effect (8%), leaving the estimate essentially unchanged ($b = -0.19$, one-sided $p = .040$). This pattern suggests that rise may capture aspects of emotional processing beyond what patients consciously experience as arousal, consistent with the ISTDP framework. When session-level patient-rated alliance (SRS) was included as a covariate, the within-person rise coefficient was attenuated by 10% but remained stable in direction and magnitude ($b = -0.19$, one-sided $p = .039$), while alliance independently predicted symptom reduction ($b = -0.08$, $p = .047$). The interpretation of this attenuation is ambiguous. Alliance may act as a confounder, facilitator, mediator, or broader marker of session quality depending on its temporal relationship to rise and symptom change [21]. However, the within-person correlation between SRS and rise was weak ($r = 0.12$), and the rise point estimate remained in the expected direction. The Rise × SRS interaction was non-significant, providing no evidence that rise and alliance interacted in predicting subsequent symptoms. Nonetheless, the possibility that rise partly reflects broader session quality cannot be ruled out.

These findings contribute to a growing body of evidence supporting ISTDP in somatic symptom and functional disorders [14]. While previous studies have demonstrated symptom reductions following ISTDP, they have rarely examined whether the model's proposed processes are linked to symptom change. The finding that therapist-rated rise predicted symptom change whereas patient-rated emotional activation did not suggests that the therapeutically relevant process may not simply reflect general emotional arousal but the specific experience of complex attachment-related feelings. This converges with findings from Internet-based Emotional Awareness and Expression Therapy (I-EAET), where "signs of unprocessed emotions" partially mediated somatic symptom reduction [36]. More broadly, these findings align with theoretical models linking relational adversity and impaired emotion regulation to somatization [7,8]. They are also consistent with emerging psychosomatic research highlighting the role of suppressed anger, emotional inhibition, and maladaptive emotion regulation processes in bodily distress and functional somatic symptoms [37].

Several strengths support the robustness of the findings. The within-person design with person-mean centering isolates session-level covariation from stable between-person differences. The process measure (therapist-rated) and the outcome measure (patient-reported) were collected independently, reducing the risk of shared-method variance. The treatment-resistant nature of the sample—participants showed no improvement across approximately 60 weeks prior to ISTDP [24]—makes regression to the mean or spontaneous recovery unlikely explanations. Finally, the consistency of the rise coefficient ($b \approx -0.22$) across all model specifications suggests a stable signal rather than a model-specific artefact.

Several limitations should also be considered. The sample was small ($N = 25$), limiting statistical power and the precision of the estimates. Rise is a single-item therapist rating without inter-rater reliability estimates. Person-mean centering removes stable between-therapist calibration differences, but it does not eliminate within-therapist biases. Therefore, the extent to which therapist-rated rise reflects specific emotional processing rather than broader perceptions of session success could not be directly determined. Even though therapists were well-trained in the ISTDP framework, they may rate rise higher after sessions they perceived as globally successful rather than strictly reflecting the specific emotional processing construct. The weak within-person correlation between rise and patient-rated alliance ($r = 0.12$) suggests these are largely distinct, but because they are rated by different informants this comparison cannot directly rule out therapist-level halo effects. An additional limitation is that PHQ-15 assessments and therapy sessions were not always conducted on the same dates. Although interpolation quality was high (median gap = 1 day and only 0.9% of observations required extrapolation), estimating symptom levels at session dates introduces some uncertainty regarding the precise temporal alignment of emotional processing and symptom change. Moreover, the lagged design supports temporal precedence but cannot establish causality. Although a reverse-direction check found no evidence that symptoms predicted subsequent rise, unmeasured third

variables—for example fluctuations in patients' general well-being—could influence both emotional processing and subsequent symptom reports. In addition, the findings may not generalize to broader SSD populations or routine clinical settings. The sample was small, predominantly composed of middle-aged women with treatment-resistant symptoms, treated online, and treated by therapists specifically trained and supervised in ISTDP. Generalization to other patient populations, treatment formats, or psychotherapeutic approaches should therefore be made cautiously.

Clinically, the findings suggest that rise may be useful to monitor as a session-level process indicator in ISTDP. Sessions in which complex attachment-related feelings were accessed and experienced were associated with measurable—if modest—proximal symptom reductions, whereas sessions dominated by cognitive or defensive processes were not. From a psychosomatic perspective, the findings suggest that the in-session experience of specific complex emotions may have short-term effects on somatic symptom reports, consistent with the theoretical link between emotional repression and somatic symptom formation. Future research should attempt to replicate these findings in larger samples. Studies could also examine whether the rise–symptom association is moderated by patient characteristics such as attachment style, trauma history, or personality functioning, which may identify patients for whom the processing of attachment-related emotions is particularly relevant [15].

In conclusion, this study provides initial session-level evidence that the experience of complex attachment-related feelings in therapy is associated with proximal somatic symptom change in treatment-resistant SSD. The findings are consistent with ISTDP's theoretical model, in which unprocessed emotions linked to early relational adversity are hypothesized to contribute to somatic symptom maintenance, and their conscious experience in therapy may facilitate symptom relief.

Declaration of generative AI and AI-assisted technologies

OpenAI ChatGPT and Anthropic Claude Code were used as AI assistants during writing and for writing R code scripts. All text and analyses were reviewed and approved by the authors, who take full responsibility for the final manuscript.

CRediT authorship contribution statement

Peter Lilliengren: Writing – original draft, Project administration, Methodology, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Robert Johansson:** Writing – original draft, Methodology, Investigation, Formal analysis, Conceptualization. **Joel M. Town:** Writing – review & editing, Investigation, Conceptualization. **Daniel Maroti:** Writing – review & editing, Project administration, Methodology, Funding acquisition, Data curation, Conceptualization.

Statement of ethics

The KOSMOS project, including KOSMOS 3, was approved by the Swedish Ethical Review Authority (2023-04956-01) and conducted in accordance with the Declaration of Helsinki. Participants initially provided written informed consent to participate in the KOSMOS project as a whole. Those selected for KOSMOS 3 subsequently provided additional written informed consent before entering that project phase.

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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: P. L. and J.M.T. provide ISTDP training and supervision. P.L., J.M.T., and D.M. receive occasional honoraria for ISTDP- and SSD-related lectures from non-profit and for-profit organizations. R.J. declares no conflicts of interest. 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

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychores.2026.112926.

Data availability

Data are not publicly available due to confidentiality, but de-identified datasets may be requested from the corresponding author.

References

[1] B. Löwe, et al., Somatic symptom disorder: a scoping review on the empirical evidence of a new diagnosis, Psychol. Med. 52 (4) (2022) 632–648, https://doi.org/10.1017/S0033291721004177.
[2] M.W. Petersen, et al., Welfare benefit utilization for people with functional somatic disorder: a population-based cohort study, J. Psychosom. Res. 191 (2025) 112073, https://doi.org/10.1016/j.jpsychores.2025.112073.
[3] S. Ma, et al., The central symptoms of depression, anxiety, and somatization: a network analysis, All Life 15 (1) (2022) 933–941, https://doi.org/10.1080/26895293.2022.2120091.
[4] P. Henningsen, et al., Management of functional somatic syndromes and bodily distress, Psychother. Psychosom. 87 (1) (2018) 12–31, https://doi.org/10.1159/000484413.
[5] V. De Gucht, W. Heiser, Alexithymia and somatisation: a quantitative review of the literature, J. Psychosom. Res. 54 (5) (2003) 425–434, https://doi.org/10.1016/S0022-3999(02)00467-1.
[6] K. Schnabel, T.M. Petzke, M. Witthöft, The emotion regulation process in somatic symptom disorders and related conditions: a systematic narrative review, Clin. Psychol. Rev. 97 (2022) 102196, https://doi.org/10.1016/j.cpr.2022.102196.
[7] J.L. Adamowicz, Z. Sirotiak, E.B.K. Thomas, Childhood maltreatment and somatic symptoms: examining the role of specific types of childhood maltreatment and alexithymia, Psychol. Trauma Theory Res. Pract. Policy 16 (Suppl. 1) (2024) S2–S9, https://doi.org/10.1037/tra0001315.
[8] M.M. Riem, et al., Attachment and medically unexplained somatic symptoms: the role of mentalization, Psychiatry Res. 268 (2018) 108–113, https://doi.org/10.1016/j.psychres.2018.06.056.
[9] A. Abbass, et al., Short-term psychodynamic psychotherapy for functional somatic disorders: a meta-analysis of randomized controlled trials, Psychother. Psychosom. 89 (6) (2020) 363–370, https://doi.org/10.1159/000507738.
[10] S. Frisch, H. Gündel, Emerging evidence for emotion-focused psychotherapy in patients with somatic symptom disorders and chronic pain, J. Psychosom. Res. 165 (2023) 111132, https://doi.org/10.1016/j.jpsychores.2022.111132.
[11] P. Lilliengren, et al., The efficacy of experiential dynamic therapies: a 10-year systematic review and meta-analysis update, Clin. Psychol. Psychother. 32 (3) (2025) e70086, https://doi.org/10.1002/cpp.70086.
[12] A. Abbass, Reaching through Resistance: Advanced Psychotherapy Techniques, Seven Leaves Press, Kansas City, MO, 2015.
[13] H. Davanloo, Intensive short-term dynamic psychotherapy, in: B.J. Sadock, V. A. Sadock (Eds.), Kaplan & Sadock's Comprehensive Textbook of Psychiatry 8th, Lippincott Williams & Wilkins, Philadelphia, PA, 2005, pp. 2628–2642.
[14] A. Abbass, B. Haghiri, Intensive short-term dynamic psychotherapy for functional somatic disorders: a scoping review, Clin. Neuropsychiatry 22 (2) (2025) 111–120, https://doi.org/10.36131/cnfioritieditore20250201.

[15] J.M. Town, A. Abbass, S. Campbell, Halifax somatic symptom disorder trial: a pilot randomized controlled trial of intensive short-term dynamic psychotherapy in the emergency department, J. Psychosom. Res. 187 (2024) 111889, https://doi.org/10.1016/j.jpsychores.2024.111889.
[16] P. Hoviatdoost, et al., Mechanisms of change in intensive short-term dynamic psychotherapy: systematized review, Am. J. Psychother. 73 (3) (2020) 95–106, https://doi.org/10.1176/appi.psychotherapy.20190025.
[17] R. Johansson, et al., Intensive short-term dynamic psychotherapy for depression: treatment effectiveness and effects of unlocking the unconscious, Res. Square (2024), https://doi.org/10.21203/rs.3.rs-5404572/v1. Preprint.
[18] P. Lilliengren, et al., Intensive short-term dynamic psychotherapy for generalized anxiety disorder: a pilot effectiveness and process-outcome study, Clin. Psychol. Psychother. 24 (6) (2017) 1313–1321, https://doi.org/10.1002/cpp.2101.
[19] R. Johansson, J.M. Town, A. Abbass, Davanloo's intensive short-term dynamic psychotherapy in a tertiary psychotherapy service: overall effectiveness and association between unlocking the unconscious and outcome, PeerJ 2 (2014) e548, https://doi.org/10.7717/peerj.548.
[20] J.M. Town, A. Abbass, D. Bernier, Effectiveness and cost effectiveness of Davanloo's intensive short-term dynamic psychotherapy: does unlocking the unconscious make a difference? Am. J. Psychother. 67 (1) (2013) 89–108, https://doi.org/10.1176/appi.psychotherapy.2013.67.1.89.
[21] J.M. Town, et al., The anger-depression mechanism in dynamic therapy: experiencing previously avoided anger positively predicts reduction in depression via working alliance and insight, J. Couns. Psychol. 69 (3) (2022) 326–336, https://doi.org/10.1037/cou0000581.
[22] P.J. Curran, D.J. Bauer, The disaggregation of within-person and between-person effects in longitudinal models of change, Annu. Rev. Psychol. 62 (2011) 583–619, https://doi.org/10.1146/annurev.psych.093008.100356.
[23] E.L. Hamaker, R.M. Kuiper, R.P.P.P. Grasman, A critique of the cross-lagged panel model, Psychol. Methods 20 (1) (2015) 102–116, https://doi.org/10.1037/a0038889.
[24] P. Lilliengren, et al., Online intensive short-term dynamic psychotherapy (ISTDP) for treatment-resistant somatic symptom disorder: an interrupted time-series study, Psychother. Psychosom. (2026), https://doi.org/10.1159/000550629.
[25] A. Smakowski, et al., Psychological risk factors of somatic symptom disorder: a systematic review and meta-analysis of cross-sectional and longitudinal studies, J. Psychosom. Res. 181 (2024) 111608, https://doi.org/10.1016/j.jpsychores.2024.111608.
[26] D. Maroti, et al., A randomized cross-over trial comparing life stress interview versus basic assessment of psychiatric symptoms for patients with persistent physical symptoms, Biopsychosoc. Sci. Med. 87 (8) (2025) 584–591, https://doi.org/10.1097/psy.0000000000001424.
[27] D. Maroti, et al., Guided versus unguided internet-administered emotional awareness and expression therapy (I-EAET) for patients with persistent physical symptoms: a randomized trial, Internet Interv. 44 (2026) 100942, https://doi.org/10.1016/j.invent.2026.100942.
[28] A. Abbass, H. Schubiner, Hidden from View: A Clinician's Guide to Psychophysiologic Disorders, Psychophysiologic Press, Kansas City, MO, 2018.
[29] K. Kroenke, R.L. Spitzer, J.B.W. Williams, The PHQ-15: validity of a new measure for evaluating the severity of somatic symptoms, Psychosom. Med. 64 (2) (2002) 258–266, https://doi.org/10.1097/00006842-200203000-00008.
[30] B.L. Duncan, et al., The session rating scale: preliminary psychometric properties of a "working" alliance measure, J. Brief Ther. 3 (1) (2003) 3–12.
[31] R Core Team, R: A Language and Environment for Statistical Computing, R Foundation for Statistical Computing, Vienna, Austria, 2024. https://www.R-project.org/.
[32] H. Wickham, et al., Welcome to the *tidyverse*, J. Open Source Softw. 43 (2019) 1686, https://doi.org/10.21105/joss.01686.
[33] D. Bates, et al., Fitting linear mixed-effects models using *lme4*, J. Stat. Softw. 67 (1) (2015) 1–48, https://doi.org/10.18637/jss.v067.i01.
[34] A. Kuznetsova, P.B. Brockhoff, R.H.B. Christensen, *lmerTest* package: tests in linear mixed effects models, J. Stat. Softw. 82 (13) (2017) 1–26, https://doi.org/10.18637/jss.v082.i13.
[35] U. Orth, et al., Effect size guidelines for cross-lagged effects, Psychol. Methods 29 (2) (2024) 421–433, https://doi.org/10.1037/met0000499.
[36] D. Maroti, et al., Internet-based emotional awareness and expression therapy for somatic symptom disorder: a randomized controlled trial, J. Psychosom. Res. 163 (2022) 111068, https://doi.org/10.1016/j.jpsychores.2022.111068.
[37] S.M. Kahnert, et al., Angry body, angry mind–mediation analysis of anger suppression, psychopathology and quality of life in bodily distress, J. Psychosom. Res. (2025) 112381, https://doi.org/10.1016/j.jpsychores.2025.112381.