Journal of Psychosomatic Research 200 (2026) 112468

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

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

Clarifying the role of somatic and depressive symptoms in suicide risk: Evidence from a cross-sectional analysis

Ronja Volz [a,b,1], Robert Philipp Kosilek [a,1], Carolin Haas [c], Patricia Dolp [a,b], Caroline Jung-Sievers [b,d,e], Tobias Teismann [f], Peter Henningsen [g], Peter Falkai [b,h,i], Jochen Gensichen [a,b,i], Karoline Lukaschek [a,b,j,*], for the POKAL Study Group[2]

[a] Institute of General Practice and Family Medicine, LMU University Hospital, LMU Munich, Munich, Germany
[b] Graduate Program "POKAL – Predictors and Outcomes in Primary Care Depression Care"(DFG-GrK 2621), Munich, Germany
[c] Institute of Psychology, University of the Bundeswehr Munich, Neubiberg, Germany
[d] Institute for Medical Information Processing, Biometry, and Epidemiology, Faculty of Medicine, LMU Munich, Munich, Germany
[e] Pettenkofer School of Public Health, Munich, Germany
[f] Ruhr-University Bochum, Mental Health Research and Treatment Center, Faculty of Psychology, Massenbergstraße 9-13, Bochum 44787, Germany
[g] Department of Psychosomatic Medicine and Psychotherapy, Klinikum Rechts der Isar, Technical University of Munich, Munich, Germany
[h] Department of Psychiatry and Psychotherapy, LMU University Hospital, LMU Munich, Munich, Germany
[i] German Center for Mental Health (DZPG), Munich/Augsburg, Germany
[j] Division of Occupational Dermatology, Department of Dermatology, University Hospital Heidelberg, Heidelberg, Germany

ARTICLE INFO

Keywords:
Depressive disorder
Somatic symptom disorder
Suicidal ideation
Primary health care
Screening and assessment

ABSTRACT

Background: Somatic symptoms are common in psychiatric and primary care populations, and their relationship with depression and suicidal ideation is complex. This study investigates the association between somatic symptoms and suicidal ideation, accounting for comorbid depressive symptoms.
Methods: This cross-sectional study included 521 in- and outpatient with PHQ-9 $\geq$ 6 or a positive response to item 9, indicative of suicidal thoughts. Somatic and depressive symptoms were assessed using the PHQ-15 and PHQ-2 or PHQ-9, respectively. Suicidal ideation was measured using the risk subscale of the SuPr-10; the protective subscale was included as a covariate. Associations were examined using correlation matrices and regression models.
Results: The sample was 68 % female with a mean age of 41 years; 52 % screened positive for suicidal ideation. PHQ-9 (mean = 14.8, SD = 5.0) and PHQ-15 scores (mean = 12.4, SD = 5.0) were moderately elevated. In a two-part regression model, higher depressive symptoms were associated with a reduced likelihood of reporting no suicidal ideation (OR = 0.50, 95 % CI: 0.41–0.62) and increased severity of suicidal ideation (IRR = 1.17, 95 % CI: 1.09–1.27). Somatic symptoms showed no meaningful direct effects but were indirectly associated with suicidal ideation via depressive symptoms (indirect effect OR = 1.05, 95 % CI: 1.03–1.07).
Conclusions: In individuals with somatic complaints, co-occurring depressive symptoms are strongly associated with suicidal ideation. Systematic depression screening is essential in somatically presenting patients.

1. Background

Suicide is one of the most important preventable causes of premature mortality worldwide and a significant public health challenge [1]. Identifying individuals at increased risk continues to be a major clinical and research priority. While depression is recognized as one of the strongest predictors of suicidal ideation and behavior [2], additional factors, such as somatic symptoms, hopelessness, self-harm, and prior suicide attempts, may also contribute to suicide risk [3,4]. Successful prevention depends not only on advancing psychological interventions [5], but also on precise and dependable risk assessment. While current methods in primary care and general practice provide a foundation,

* Corresponding author at: Institute of General Practice and Family Medicine, LMU University Hospital, Nußbaumstr. 5, 80336 Munich, Germany.
E-mail address: Karoline.Lukaschek@med.uni-muenchen.de (K. Lukaschek).
[1] Shared first authorship.
[2] Group information: All POKAL Group members are listed at the end of this article.

https://doi.org/10.1016/j.jpsychores.2025.112468
Received 26 September 2025; Received in revised form 18 November 2025; Accepted 19 November 2025
Available online 20 November 2025

there remains meaningful potential to enhance their accuracy and effectiveness [6].

In primary care, somatic complaints are among the most frequent reasons for consultation, affecting nearly one-third of patients [7]. Common symptoms include fatigue [8], chronic pain [9], sleep disturbances [9], and gastrointestinal issues [10]. Importantly, somatic symptoms are frequently comorbid with psychiatric conditions, especially depression and anxiety [9], and frequently serve as the primary motivation to seek help [11]. Their presence can also contribute to the underrecognition or misdiagnosis of depression, particularly in brief consultations where mental health concerns are not always explicitly explored [12].

The relationship between somatic symptom burden – the intensity and frequency of physical symptoms – and suicidal ideation remains complex and insufficiently understood. A key gap in the literature is the lack of distinction between direct and indirect effects of somatic symptoms on suicide risk. A systematic review by Torres et al. [13] reported elevated rates of suicidal ideation (24–34 %) and prior suicide attempts (13–67 %) among individuals with somatic symptoms and related disorders (e.g., somatoform disorders, functional disorders, multiple pain complaints and general physical complaints), independent of comorbid depression or anxiety, suggesting a potential direct association. Other studies however indicate that somatic symptoms (e.g., pain conditions) may have mediated effects within a broader network of depressive psychopathology, indirectly influencing suicidal ideation or behavior through their association with affective symptoms [14,15]. Supporting this view, Jeon et al. [16] found that patients with major depressive disorder who experienced suicidal thoughts reported significantly more severe somatic symptoms (e.g., pain, muscle tension) than their non-suicidal counterparts. However, the existing studies are highly heterogeneous and show a wide range in design, populations, and methods used to assess somatic symptoms, which limits the comparability and generalizability of outcomes. While these findings suggest an association between somatic symptom severity and suicidality, the directionality of this relationship remains unclear due to the cross-sectional nature of most studies. It remains uncertain whether somatic symptoms directly contribute to suicidal ideation, or if they are part of an intensified depressive syndrome, where both somatic and suicidal symptoms worsen together as depression severity increases.

Some authors argue that somatic symptoms may play a secondary role compared to cognitive–affective symptoms of depression. For instance, Keilp et al. [17] found that the persistence of suicidal ideation was more strongly tied to core mood symptoms and self-critical cognitions, and that risk remained high even when somatic or vegetative symptoms had improved during treatment. Similarly, Hawton et al. [2] identified several factors, such as male sex and previous suicide attempts, as significantly associated with elevated suicide risk in individuals with depression, but did not highlight somatic symptoms as relevant risk factors.

In summary, while many studies report an association between somatic symptoms and suicidality, it remains unclear whether this relationship represents a distinct and independent pathway or is primarily mediated by the severity of depressive symptoms. Clarifying this distinction has practical relevance for suicide risk detection in primary care, where patients often present with physical complaints while affective symptoms remain underreported. Notably, although approximately 45 % of individuals who die by suicide have contact with a primary care physician within one month of their death, fewer than one-third disclose their suicidal thoughts to a healthcare professional [18]. Available research highlights the need for structured approaches that effectively incorporate somatic symptoms into suicide risk assessments to improve identification and intervention strategies [13,15].

This study aims to address these gaps in the literature examining both direct and indirect associations between somatic symptoms and suicidal ideation. Using validated measures and appropriate statistical methods, this study seeks to clarify the role of somatic symptom burden in suicide risk to support clinical decision-making in primary and psychiatric care.

2. Methods

2.1. Study design and participants

This study is a secondary analysis of the validation dataset for the Suicide Prevention in Primary Care questionnaire (SuPr-10). The original study was designed to validate a 10-item instrument assessing suicidal risk and protective factors in outpatient and psychiatric clinical populations. A total of 521 adults were recruited from outpatient practices, day care clinics, and psychiatric inpatient wards across multiple sites in Germany and Austria (Supplementary Fig. 1; enrollment flowchart). The main inclusion criteria were a PHQ-9 score ≥ 6 or a positive response to item 9 (suicidal ideation). All participants provided informed consent, and the study was approved by the LMU Munich ethics committee (#22–0028, May 9th, 2022). Participants were recruited over a 20-month period (July 2022 to February 2024) using both traditional and online strategies [19]. Traditional recruitment involved direct engagement with physicians and therapists and collaboration with 39 outpatient and inpatient facilities across Germany (primarily Bavaria) and Austria. Participants recruited online were screened in person at the Institute of General Practice of LMU Munich by a study psychologist or physician and, if eligible, were subsequently enrolled in the study [20]. These sites comprised 20 general medical practices, 12 psychotherapy practices, five psychiatric inpatient wards, and two daycare clinics, covering a range of settings from urban to rural regions. Online recruitment, implemented in parallel, utilized digital platforms to reach potential participants [18]. The recruitment flowchart, including treatment settings, is provided in the supplementary material (suppl. Fig. A.1). Full study details are reported in the primary publication [18].

2.2. Measures

Suicidality was assessed using the SuPr-10 questionnaire. It comprises 10 items, with items 1–4 assessing protective beliefs (e.g., future orientation) and items 5–9 capturing risk indicators (e.g., suicidal ideation). Each item is scored on a 4-point Likert scale (0–3). The final item (10) assesses suicide attempts within the past two weeks (yes/no), followed by questions on reasons that prevented such actions if attempts were denied. The SuPr-10 protective and risk subscales showed good to excellent internal consistency in the validation study, with McDonald's Omega values of 0.817 and 0.928, respectively [19]. Items 5–9 of the risk subscale were analyzed as a total count score and as a binary outcome, where any score above zero indicated increased risk of suicidal behavior, consistent with prior validation [19].

Somatic symptom burden was assessed using the Patient Health Questionnaire-15 (PHQ-15), which captures the frequency and severity of 15 physical complaints commonly associated with depression [21]. Total scores range from 0 to 30, with burden categorized as minimal (0–4), low (5–9), medium (10–14), and high (15–30). While the dataset did not include clinical diagnoses to distinguish medically explained from functional symptoms, this distinction is no longer required under DSM-5 criteria for somatic symptom disorder [22]. The PHQ-15 is validated as a general measure of somatization severity [23].

Depressive symptoms were assessed using the Patient Health Questionnaire-9 (PHQ-9) [24]. Total scores range from 0 to 27, with higher scores indicating greater symptom severity. Standard clinical cutoffs were applied to describe burden categories (mild: 5–9, moderate: 10–14, moderately severe: 15–19, severe: 20–27). Although the full PHQ-9 was collected in the trial, our primary analyses focused on the PHQ-2, a validated brief screener comprising the first two PHQ-9 items, capturing the two core affective symptoms of depression: anhedonia and depressed mood [25]. It ranges from 0 to 6 points with a cut-off at ≥ 3

points indicating depression with excellent screening validity and similar performance compared to the full PHQ-9 [26]. This choice was made to reduce conceptual and statistical overlap with the PHQ-15, as several PHQ-9 items (sleep, fatigue, appetite changes) measure somatic symptoms also captured by PHQ-15. Furthermore, PHQ-9 item 9 directly assesses suicidal ideation, which overlaps with the SuPr-10 risk scale, potentially confounding analyses. By focusing on the PHQ-2, we aimed to isolate core depressive affect and thereby better distinguish its relationship with somatic symptoms and suicidal ideation. To ensure robustness, we conducted sensitivity analyses using the full PHQ-9, provided as supplementary material.

Together, the three instruments provide domain-specific assessment of depressive affect, somatic burden, and suicidal ideation with strong validity and conceptual specificity. Covariates included sex, age, education (CASMIN levels [27]) and treatment setting (outpatient, day-care, and inpatient general or acute psychiatry) and protective beliefs (SuPr-10 items 1–4).

2.3. Statistical analysis

Analyses were conducted in Stata 19.0 (StataCorp, TX, USA). Missing data were minimal at <1 % for all variables of interest. We summarized continuous variables using means (SD) or medians (IQR), and categorical data using frequencies and percentages. Pairwise Spearman correlations were calculated across symptom and demographic variables and visualized in a heatmap. A density plot was created using the *heatplot* package [28], illustrating how PHQ-9 and PHQ-15 scores relate to mean SuPr-10 risk scores. We then fit a two-part zero-inflated negative binomial (ZINB) model to account for the overdispersed distribution of the SuPr-10 risk subscale with excess zero scores. The ZINB model includes a count component predicting the total risk score expressed as incidence rate ratios (IRR) and an inflation component estimating a logistic model for the odds of scoring zero, expressed as odds ratios (OR). PHQ-2, PHQ-15, SuPr-10 protective scores, and demographic covariates were included. While the PHQ-2 modeling approach reduces variance compared to the full PHQ-9, it offers a conservative and conceptually specific measure of the affective components of depression. A sensitivity analysis was run utilizing the full PHQ-9 score in the ZINB model. Finally, we estimated a generalized structural equation model (GSEM) to examine indirect associations. The model specified an ordinal logit path from PHQ-15 to the PHQ-2 total score, and logistic regression paths from both PHQ-15 and PHQ-2 to the binary suicidal ideation indicator (SuPr-10 risk >0). Indirect, direct, and total effects were calculated using the product-of-coefficients method with robust standard errors. The proportion mediated was derived as the ratio of the indirect effect to the total effect.

3. Results

3.1. Sample characteristics

Descriptive statistics are presented in Table 1. The sample included 521 participants, with a mean age of 40.9 years (SD = 14.3); 67.8 % were female. Most participants had intermediate (44.3 %) or high (38.4 %) education, and the majority were recruited in the outpatient setting (75.6 %). In the PHQ-2 depression screener, 73.5 % of participants exceeded the threshold of ≥3 points. The mean PHQ-9 score was 14.8 (SD = 5.0), indicating moderate depressive symptom severity, with 19 % meeting criteria for severe depression (PHQ-9 ≥ 20). PHQ-15 scores averaged 12.4 (SD = 5.0), and 35.3 % reported high somatic symptom burden (PHQ-15 ≥ 15). On the SuPr-10 instrument, the median protective subscale score was 5.0 (IQR: 3–6), while the risk subscale median was 1.0 (IQR: 0–4). Notably, 51.6 % of participants had a non-zero score on the risk subscale, indicating increased risk for suicidal behavior based on the instrument's validation criteria.

Table 1
Sample characteristics ($N = 521$).

Sex, % (No.)	
Female	67.8 % (353)
Male	31.7 % (165)
N/A	0.6 % (3)
Age (years), mean (SD)	40.9 (14.3)
Education (CASMIN levels), % (No.)	
Low (1a-1c)	16.9 % (88)
Intermediate (2a-2c)	44.3 % (231)
High (3a-3b)	38.4 % (200)
N/A	0.4 % (2)
Setting, % (No.)	
Outpatient care	75.6 % (394)
Psychiatric day care	14.2 % (74)
Psychiatric inpatient care	10.2 % (53)
PHQ-2 Score, median (IQR)	4 (2–5)
PHQ-2 Score ≥ 3, % (No.)	73.5 % (383)
PHQ-9 Score, mean (SD)	14.8 (5.0)
PHQ-9 Burden, % (No.)	
Mild (5–9)	16.7 % (87)
Moderate (10–14)	33.0 % (172)
Mod. severe (15–19)	30.9 % (161)
Severe (20–27)	19.0 % (99)
N/A	0.4 % (2)
PHQ-15 Score, mean (SD)	12.4 (5.0)
PHQ-15 Burden, % (No.)	
Minimal (0–4)	5.8 % (30)
Low (5–9)	26.3 % (137)
Medium (10–14)	32.4 % (169)
High (15–30)	35.3 % (184)
N/A	0.2 % (1)
SuPr-10 Protective Subscale (Items 1–4), median (IQR)	5 (3–6)
SuPr-10 Risk Subscale (Items 5–9), median (IQR)	1 (0–4)
SuPr-10 Increased Risk (Items 5–9 > 0), % (No.)	51.6 % (269)
SuPr-10 Item 10a: Suicide attempt (< 2 weeks), % (No.)	1.2 % (6)
SuPr-10 Item 10b: Protective Reasons (1–8), median (IQR)	3.0 (2.0–4.0)

3.2. Correlation and exploratory visualization

Spearman correlations (Fig. 1, suppl. Table A.1) showed a moderate association between PHQ-15 and PHQ-9 ($r = 0.40$), which was attenuated with the PHQ-2 ($r = 0.22$). A similar pattern could be observed for PHQ-9 / PHQ-2 and the SuPr-10 risk subscale ($r = 0.53$ vs 0.43), while

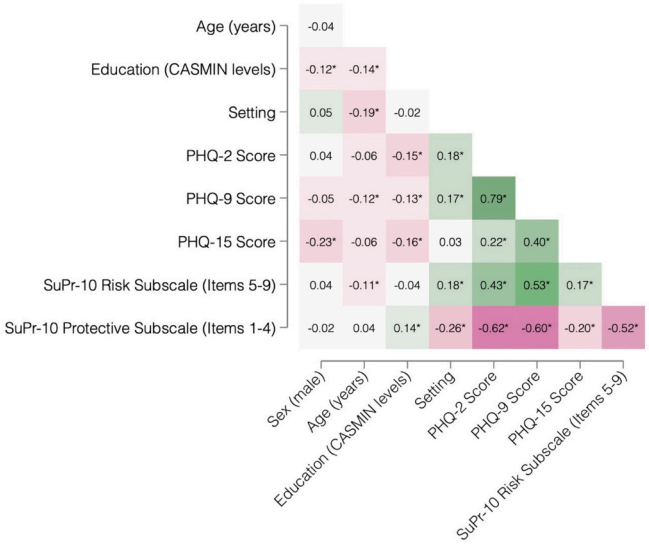

Fig. 1. Correlation heatmap of mental health and demographic variables. Pairwise Spearman correlations. Color scale indicates strength and direction of pairwise correlations; green for positive, red for negative. * $p < .05$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

the protective subscale shows a comparable substantial correlation of about −0.60. The PHQ-15 as well as sociodemographic variables showed overall weaker associations with symptom scores. The density plot (Fig. 2) revealed that higher mean SuPr-10 risk scores clustered predominantly among participants with PHQ-9 scores above 15, regardless of somatic symptom burden.

3.3. Main predictors of suicidal ideation

Table 2 presents results from zero-inflated negative binomial regression models predicting both the severity and likelihood of suicidal ideation based on the SuPr-10 risk subscale (items 5–9). We used the PHQ-2 as the primary measure of depressive symptoms to address potential overlap with somatic symptoms and the SuPr-10 risk scale. Sensitivity analyses using the full PHQ-9 scores yielded overall consistent results (suppl. Table A.2).

In Model 1, higher PHQ-2 scores were associated with increased suicidal ideation severity (count model IRR = 1.17, 95 % CI: 1.09–1.27) and substantially reduced odds of scoring zero on the risk scale (inflation model OR = 0.50, 95 % CI: 0.41–0.62). In contrast, PHQ-15 scores showed no significant association with either outcome component.

After introducing the SuPr-10 protective subscale and sociodemographic covariates (Model 2 and Model 3), the association between PHQ-2 and ideation severity was attenuated and no longer statistically significant (IRR ≈ 0.96), while its association with the zero-risk group remained significant but reduced (OR ≈ 0.65–0.68). Notably, the protective subscale was strongly associated with lower suicidal ideation severity (IRR ≈ 0.81–0.85) and increased odds of zero risk (OR ≈ 1.33). Inpatient treatment setting was also strongly linked to increased severity and decreased zero risk odds.

PHQ-15 remained mostly non-significant, except for a small positive signal with severity in the fully adjusted model (IRR = 1.02). Higher education was associated with reduced odds of suicidal ideation in the inflation component. Age and sex did not show meaningful effects.

To illustrate the impact of affective symptoms on suicidal ideation, we used Model 1 to estimate predicted SuPr-10 risk scores at different PHQ-2 levels, holding PHQ-15 constant. At a PHQ-2 score of 2, the predicted SuPr-10 item count was about 1.0; at a score of 4, it increased nearly threefold to 2.7; and at 6, it rose to approximately 4.9. This sharp increase is driven by both components of the model, i. e. higher intensity among those at risk and fewer individuals remaining in the zero-risk group, resulting in a combined effect that exceeds a simple multiplicative increase.

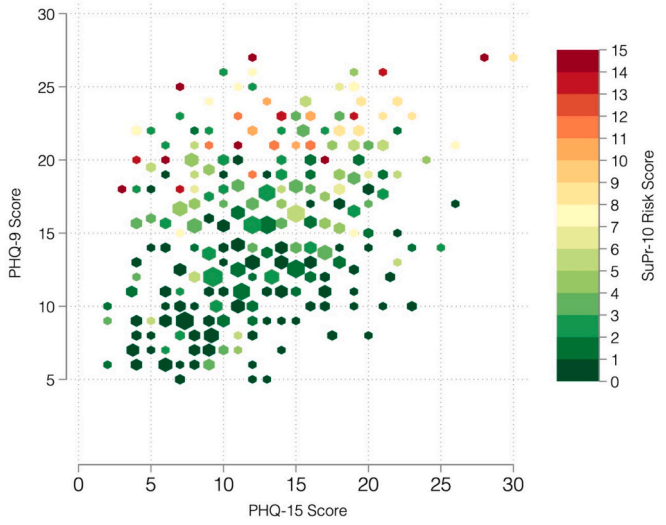

Fig. 2. Density plot of SuPr-10 risk score over PHQ-9 and PHQ-15 scores. The color scale reflects the mean SuPr-10 risk score per hexagonal bin. Bin size is proportional to the number of observations. Empty areas reflect sparse or no data.

Table 2
Regression models for SuPr-10 risk score.

Outcome: SuPr-10 Risk Subscale (items 5–9)	Count model (Score > 0)	Inflation model (Score = 0)
	IRR [95 % CI]	OR [95 % CI]
	Model 1 (N = 519)	
PHQ-2 Score	1.17* [1.09–1.27]	0.50* [0.41–0.62]
PHQ-15 Score	1.01 [0.99–1.03]	0.97 [0.93–1.02]
	Model 2 (N = 519)	
PHQ-2 Score	0.96 [0.88–1.05]	0.65* [0.52–0.81]
PHQ-15 Score	1.01 [0.99–1.03]	0.98 [0.94–1.03]
SuPr-10 Protective Subscale (Items 1–4)	0.81* [0.77–0.86]	1.33* [1.18–1.50]
	Model 3 (*N* = 516)	
PHQ-2 Score	0.96 [0.88–1.05]	0.68* [0.56–0.84]
PHQ-15 Score	1.02* [1.00–1.04]	0.98 [0.93–1.03]
SuPr-10 Protective Subscale (Items 1–4)	0.85* [0.80–0.89]	1.33* [1.17–1.51]
Sex (Male)	0.98 [0.79–1.23]	0.68 [0.39–1.19]
Age (Years)	1.00 [0.99–1.00]	1.00 [0.99–1.02]
Education (CASMIN Levels)		
Intermediate vs. *Low*	0.97 [0.72–1.29]	0.37* [0.17–0.78]
High vs. *Low*	1.02 [0.75–1.39]	0.50 [0.22–1.12]
Setting (Inpatient)	1.99* [1.67–2.37]	0.20* [0.06–0.66]

Zero-inflated negative binomial regression predicting SuPr-10 risk subscale scores. The count model reports incidence rate ratios (IRRs) reflecting changes in the expected SuPr-10 risk score. The logistic inflation model estimates odds ratios (ORs) for being in the always-zero group (i.e., not at risk of scoring > 0). Robust 95 % confidence intervals shown in brackets. * $p < .05$.

3.4. Pathways between depressive, somatic, and suicidal symptoms

We used a generalized structural equation model (GSEM) to test whether depressive symptoms indirectly link the relationship between somatic complaints and suicidal ideation, defined as a score > 0 on the SuPr-10 risk subscale (Table 3). PHQ-15 was significantly associated with PHQ-2 scores (OR = 1.08, 95 % CI: 1.05–1.12), which in turn were strongly linked to elevated suicidal ideation risk (OR = 1.82, 95 % CI: 1.60–2.09).

The direct effect of PHQ-15 on suicidal ideation was not statistically significant (OR = 1.03, 95 % CI: 0.99–1.07), while the indirect effect through PHQ-2 was significant (OR = 1.05, 95 % CI: 1.03–1.07). The total effect of PHQ-15 on suicidal ideation was significant (OR = 1.08, 95 % CI: 1.04–1.13). The proportion of the indirect effect linked via PHQ-2 was estimated at 61 % (95 % CI: 31 %–91 %). These results indicate a significant indirect pathway from somatic symptoms to suicidal ideation via depressive affect, with no clear evidence for a direct effect of somatic symptoms independent of depressive symptoms.

Average predicted probabilities of suicidal ideation increased

Table 3
Path model linking somatic and depressive symptoms to increased SuPr-10 risk scores.

Path / Effect	OR [95 % CI]
Direct paths	
PHQ-15 → PHQ-2 (a)	1.08* [1.05–1.12]
PHQ-2 → Elevated SuPr-10 risk (b)	1.82* [1.60–2.09]
PHQ-15 → Elevated SuPr-10 risk (c)	1.03 [0.99–1.07]
Derived effects	
Indirect effect (a × b)	1.05* [1.03–1.07]
Total effect (a × b + c)	1.08* [1.04–1.13]
Proportion Indirect/Total: 0.61* [0.31–0.91]	

$N = 519$. Generalized structural equation model (GSEM) linking PDS-15 scores with a logit link to an elevated SuPr-10 risk score (> 0) and ordinal logit link to PHQ-2 scores. Derived effects were calculated using nonlinear combination functions. * $p < .05$.

substantially with higher PHQ-2 scores, rising from 27 % at a PHQ-2 score of 2, to 55 % at 4, and 80 % at 6, mirroring the pattern observed in the inflation component of the ZINB model.

4. Discussion

This study investigated the relationship between somatic symptom burden, depressive symptoms, and suicidal ideation in a cross-sectional clinical study drawn from both inpatient and outpatient settings. Using zero-inflated regression and structural equation modeling, we consistently observed that somatic symptoms were not directly associated with suicidal ideation. Instead, their relationship with suicidal ideation was indirectly linked via their association with co-occurring depressive symptoms.

Depressive symptom severity, measured by the PHQ-2 and PHQ-9, demonstrated a robust association with both somatic symptoms based on PHQ-15 scores and suicidal ideation as assessed by the SuPr-10 risk subscale. The SuPr-10 protective scale was inversely associated with both depressive and somatic symptoms and the SuPr-10 risk subscale. Each incremental increase of depressive symptoms based on PHQ-2 scores corresponded to a marked rise in both the likelihood and severity of suicidal ideation, with predicted risk scores more than doubling between low and elevated degrees of depressive symptoms. In contrast, somatic symptom burden, assessed using the PHQ-15, showed no meaningful association with suicidal ideation once depressive symptoms were accounted for. Graphical analyses indicated a threshold-like pattern: suicidal ideation was rarely reported in the absence of moderate or higher depressive symptom levels, regardless of somatic symptom burden. Finally, the structural equation model confirmed an indirect pathway linking somatic symptoms to suicidal ideation: while PHQ-15 showed no direct association with SuPr-10 scores, it was significantly associated with PHQ-2, which in turn predicted elevated suicidal ideation. This suggests that the impact of somatic symptoms on suicidal ideation is largely indirectly linked through core depressive affective symptoms [3].

4.1. Comparison with previous research

These results are consistent with prior literature emphasizing the co-occurrence of somatic and depressive symptoms [29,30], particularly in primary care, where somatic complaints often obscure underlying psychological distress [31]. Our findings also support theoretical models positing that depressive symptoms mediate the link between somatic symptom burden and suicidal ideation [16]. They align with earlier work highlighting the central role of cognitive–affective depressive symptoms in suicide risk, even when somatic or vegetative symptoms improve [17]. Notably, our findings diverge from reports suggesting an independent association between somatic symptoms and suicidality irrespective of depression [13]. Our findings differ from those of Torres et al. [13|, who reported elevated suicidality among individuals with somatic symptom and related disorders even after adjusting for depression and anxiety. Possible explanations include differences in diagnostic inclusion criteria, measurement tools, adjustment for psychiatric comorbidity, and the composition of the study sample [13]; in our analysis, depressive symptoms were operationalized using core affective items (PHQ-2), and somatic and suicidal symptoms were measured with distinct, non-overlapping instruments, potentially yielding a more conservative estimate of any direct somatic–suicidality association.

These discrepancies may stem from methodological differences, such as limited adjustment for psychiatric comorbidity or reliance on categorical diagnostic frameworks that do not sufficiently differentiate overlapping symptom domains [15].

Clinically, these results underscore the importance of systematic depression screening in patients presenting with somatic complaints. Given that suicidal ideation primarily occurred at higher levels of depressive symptoms, physical symptoms may mask significant emotional distress, posing a diagnostic challenge in general medical settings where mental health expertise and time are often limited. According to previous findings, up to one third of individuals with mental illness consulted a general practitioner shortly before attempting suicide, in some cases solely due to somatic symptoms [32]. Given that current clinical guidelines seldom recommend suicide risk screening in patients presenting with somatic symptoms [13], the routine implementation of validated assessment tools such as the PHQ-9, potentially supplemented by targeted instruments like the SuPr-10, should be considered as a minimum approach to improve the identification of suicide risk in this population. In this context, it has been shown that providing feedback to patients and GPs after depression screening does not significantly reduce depression severity, compared to GP-only feedback or no feedback [33]. However, providing feedback to patients plus GPs reduced, among others, the severity of somatic symptoms and improved GP–patient communication about depression, which might include thoughts about suicide. Effective treatment of depression, both pharmacological and psychological, remains important for suicide prevention [34]. Moreover, evidence suggests that training general practitioners to recognize and treat depression can lead to significant reductions in suicide rates [35]. However, effective prevention and treatment in primary care depend on thorough and systematic risk assessment. Early identification of depression, especially when masked by somatic symptoms, enables timely intervention and reduces the risk of missed opportunities.

4.2. Strengths and limitations

The study benefits from a comparatively large sample of patients from both inpatient and outpatient settings, with varying symptom severity. The use of validated instruments strengthens the generalizability and reproducibility of the findings. Importantly, by employing the PHQ-2 instead of the PHQ-9 for our primary analyses, we minimized symptom overlap and inflated associations, enabling a clear distinction between somatic, depressive, and suicidal symptoms. The analytic approach further allowed us to separate direct and indirect associations, providing a clearer understanding of how somatic symptoms relate to depression and suicidality.

However, several limitations must be acknowledged. First, the cross-sectional design limits conclusions about causality. While the structural equation model aligns with theoretical assumptions, longitudinal data are needed to confirm the direction of effects. Second, reliance on self-report instruments introduces potential response and recall biases, which may affect the accuracy of symptom reporting. Third, other established risk factors [2] such as substance misuse or emotional abuse [13] were not measured and thus, not accounted for, which could influence the observed associations. Finally, using a PHQ-9 threshold to recruit individuals with at least mild depression may have reduced variability in depressive symptom scores, particularly in relation to somatic symptom analyses. This, along with recruitment from diverse clinical and online settings, may limit generalizability to general primary care populations.

4.3. Implications and future directions

Future research should adopt longitudinal designs to clarify causal pathways between somatic symptom burden, depressive symptoms, and suicidality [36]. Further subgroup analyses based on demographic variables (e.g., sex, age) are also warranted, given possible differences in symptom presentation, help-seeking behavior and the association between affective and somatic symptoms [37]. This may offer not only a more comprehensive understanding of the association between depression and suicidal ideation, but also the mechanisms linking somatic symptom burden and suicidal ideation as well as concrete intervention approaches.

In our study, protective factors for suicidal ideation such as satisfaction with life, goals, and coping strategies were inversely associated with both depressive and somatic symptoms as well as with risk for suicidal ideation. Incorporating these factors into suicide risk assessments may improve screening and prevention strategies [38].

5. Conclusion

In summary, our findings provide insight into the relationship between somatic symptoms, depressive symptoms, and suicidal ideation. The cross-sectional analysis suggests that the association between somatic symptoms and suicidal ideation is largely attributable to co-occurring depressive symptoms. This highlights the need to systematically assess mood symptoms in patients presenting with physical complaints, as it may offer an opportunity for early intervention before depressive symptoms worsen and suicidal thoughts develop.

CRediT authorship contribution statement

Ronja Volz: Writing – original draft, Formal analysis, Conceptualization. **Robert Philipp Kosilek:** Writing – original draft, Supervision, Methodology, Conceptualization. **Carolin Haas:** Writing – review & editing, Project administration, Investigation, Data curation. **Patricia Dolp:** Writing – review & editing. **Caroline Jung-Sievers:** Writing – review & editing, Supervision. **Tobias Teismann:** Writing – review & editing, Supervision. **Peter Henningsen:** Writing – review & editing, Supervision. **Peter Falkai:** Writing – review & editing, Supervision. **Jochen Gensichen:** Writing – review & editing, Resources, Funding acquisition. **Karoline Lukaschek:** Writing – review & editing, Supervision, Project administration, Methodology, Investigation, Conceptualization.

Ethics approval and consent to participate

The study received ethics approval from the medical ethics committee of LMU Munich on May 9, 2022 (project no. 22–0028) and was performed in accordance with applicable guidelines and regulations according to Declaration of Helsinki–Ethical Principles for Medical Research Involving Human Subjects [39]. All participants gave written informed consent.

Funding

This study was funded by the German Research Foundation (DFG Grant: DFG-GrK 2621/POKAL-Kolleg). The funder had no role in the design and conduct of the study; collection, management, analysis and interpretation of the data; preparation and review or approval of the manuscript, or the decision to submit the manuscript for publication.

Declaration of competing interest

There are no conflicts of interest to disclose.

Acknowledgements

The POKAL-Group (PrädiktOren und Klinische Ergebnisse bei depressiven ErkrAnkungen in der hausärztLichen Versorgung (POKAL, DFG-GrK 2621)) consists of the following principal investigators: Tobias Dreischulte, Peter Falkai, Jochen Gensichen, Peter Henningsen, Markus Bühner, Caroline Jung-Sievers, Helmut Krcmar, Karoline Lukaschek, Gabriele Pitschel-Walz, Antonius Schneider, Kirsten Lochbuhler, Philipp Reindl-Spanner, Andrea Schmitt. The following doctoral students are members of the POKAL-Group: Patricia Dolp, Vildan Dogan, Christopher Ebert, Kerstin Hupe, Nana Jedlicska, Luca Kittelmann, Julia Korting, Mehring Sophie Li, Simona Maspero, Saskia Pohl, Maximilian Schumergruber, Jan Stursa, Marie Vogel, Ronja Volz, Alexandra Wieser, Susanne Winter, Melanie Wöhler.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychores.2025.112468.

Data availability

Data, analytic code, and research material supporting this study's findings are available from the DFG-GrK 2621/POKAL studies but are not publicly accessible due to licensing restrictions. They can be obtained upon reasonable request by contacting "Stiftung Allgemeinmedizin – The Primary Health Care Foundation" at www.stiftung-allgemeinmedizin.de or via email at office@stiftung-allgemeinmedizin.de.

References

[1] S. Bachmann, Epidemiology of suicide and the psychiatric perspective, Int. J. Environ. Res. Public Health 15 (7) (2018) 1425.
[2] K. Hawton, et al., Risk factors for suicide in individuals with depression: a systematic review, J. Affect. Disord. 147 (1–3) (2013) 17–28.
[3] N.M. Melhem, et al., Severity and variability of depression symptoms predicting suicide attempt in high-risk individuals, JAMA Psychiatry 76 (6) (2019) 603–613.
[4] G. Turecki, D.A. Brent, Suicide and suicidal behaviour, Lancet 387 (10024) (2016) 1227–1239.
[5] R.C. O'Connor, M.K. Nock, The psychology of suicidal behaviour, Lancet Psychiatry 1 (1) (2014) 73–85.
[6] G. Turecki, et al., Suicide and suicide risk, Nat. Rev. Dis. Primers 5 (1) (2019) 74.
[7] K. Kroenke, Patients presenting with somatic complaints: epidemiology, psychiatric co-morbidity and management, Int. J. Methods Psychiatr. Res. 12 (1) (2003) 34–43.
[8] K. Chakraborty, et al., Psychological and clinical correlates of functional somatic complaints in depression, Int. J. Soc. Psychiatry 58 (1) (2012) 87–95.
[9] A. Steffen, et al., Mental and somatic comorbidity of depression: a comprehensive cross-sectional analysis of 202 diagnosis groups using German nationwide ambulatory claims data, BMC Psychiatry 20 (1) (2020) 142.
[10] C. Zhu, et al., Association of somatic symptoms with depression and anxiety in clinical patients of general hospitals in Guangzhou, China, Gen. Hosp. Psychiatry 34 (2) (2012) 113–120.
[11] A.A. Khan, et al., Somatic symptoms in primary care: etiology and outcome, Psychosomatics 44 (6) (2003) 471–478.
[12] H.-P. Kapfhammer, Somatic symptoms in depression, Dialogues Clin. Neurosci. 8 (2) (2006) 227–239.
[13] M.E. Torres, et al., Suicide and suicidality in somatic symptom and related disorders: a systematic review, J. Psychosom. Res. 140 (2021) 110290.
[14] X. Fang, et al., The association between somatic symptoms and suicidal ideation in Chinese first-episode major depressive disorder, J. Affect. Disord. 245 (2019) 17–21.
[15] M.J. Park, et al., Multiple types of somatic pain increase suicide attempts and depression: a nationwide community sample of Korean adults, Compr. Psychiatry 90 (2019) 43–48.
[16] H.J. Jeon, et al., Gender differences in somatic symptoms and current suicidal risk in outpatients with major depressive disorder, Psychiatry Investig. 13 (6) (2016) 609.
[17] J.G. Keilp, et al., Suicidal ideation and the subjective aspects of depression, J. Affect. Disord. 140 (1) (2012) 75–81.
[18] K. Stene-Larsen, A. Reneflot, Contact with primary and mental health care prior to suicide: a systematic review of the literature from 2000 to 2017, Scand. J. Public Health 47 (1) (2019) 9–17.
[19] C. Haas, Development and Validation of the Brief Questionnaire SuPr-10 for Suicidality Assessment among Patients with Depressive Symptoms in Primary Care, Dissertation, LMU Munich, 2025.
[20] C. Haas, et al., Efficient online recruitment of patients with depressive symptoms using social media: cross-sectional observational study, JMIR Ment. Health 12 (1) (2025) e65920.
[21] K. Kroenke, R.L. Spitzer, J.B. Williams, The PHQ-15: validity of a new measure for evaluating the severity of somatic symptoms, Psychosom. Med. 64 (2) (2002) 258–266.
[22] Diagnostic and Statistical Manual of Mental Disorders, 5th ed., American Psychiatric Association, 2022.
[23] H. van Ravesteijn, et al., Detecting somatoform disorders in primary care with the PHQ-15, Ann. Fam. Med. 7 (3) (2009) 232–238.
[24] K. Kroenke, R.L. Spitzer, J.B. Williams, The PHQ-9: validity of a brief depression severity measure, J. Gen. Intern. Med. 16 (9) (2001) 606–613.
[25] K. Kroenke, R.L. Spitzer, J.B. Williams, The patient health Questionnaire-2: validity of a two-item depression screener, Med. Care 41 (11) (2003) 1284–1292.
[26] B. Löwe, K. Kroenke, K. Gräfe, Detecting and monitoring depression with a two-item questionnaire (PHQ-2), J. Psychosom. Res. 58 (2) (2005) 163–171.

[27] H. Brauns, S. Scherer, S. Steinmann, The CASMIN educational classification in international comparative research, in: J.H.P. Hoffmeyer-Zlotnik, C. Wolf (Eds.), Advances in Cross-National Comparison: A European Working Book for Demographic and Socio-Economic Variables, Springer US, Boston, MA, 2003, pp. 221–244.
[28] B. Jann, HEATPLOT: Stata Module to Create Heat Plots and Hexagon Plots, 2021.
[29] T. Lallukka, et al., Co-occurrence of depressive, anxiety, and somatic symptoms: trajectories from adolescence to midlife using group-based joint trajectory analysis, BMC Psychiatry 19 (1) (2019) 236.
[30] S. Kohlmann, et al., The overlap of somatic, anxious and depressive syndromes: a population-based analysis, J. Psychosom. Res. 90 (2016) 51–56.
[31] X. Li, et al., Predictive potential of somatic symptoms for the identification of subthreshold depression and major depressive disorder in primary care settings, Front. Psychol. 14 (2023) 999047.
[32] A. Pearson, et al., Primary care contact prior to suicide in individuals with mental illness, Br. J. Gen. Pract. 59 (568) (2009) 825.
[33] B. Löwe, et al., Clinical effectiveness of patient-targeted feedback following depression screening in general practice (GET. FEEDBACK. GP): an investigator-initiated, prospective, multicentre, three-arm, observer-blinded, randomised controlled trial in Germany, Lancet Psychiatry 11 (4) (2024) 262–273.
[34] G. Zalsman, et al., Suicide prevention strategies revisited: 10-year systematic review, Lancet Psychiatry 3 (7) (2016) 646–659.
[35] J.J. Mann, C.A. Michel, R.P. Auerbach, Improving suicide prevention through evidence-based strategies: a systematic review, Am. J. Psychiatry 178 (7) (2021) 611–624.
[36] T.J. VanderWeele, M.B. Mathur, Y. Chen, Outcome-Wide Longitudinal Designs for Causal Inference: A New Template for Empirical Studies, 2020.
[37] R. Cui, A. Fiske, Relation between depression symptoms and suicide risk in adults and older adults: a brief report, J. Appl. Gerontol. 41 (1) (2022) 176–180.
[38] T. Teismann, et al., Positive mental health moderates the association between depression and suicide ideation: a longitudinal study, Int. J. Clin. Health Psychol. 18 (1) (2018) 1–7.
[39] World Medical Association Declaration of Helsinki, Ethical principles for medical research involving human subjects, JAMA 310 (20) (2013) 2191–2194.