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THE ROLE OF ALEXITHYMIA AND EMOTION REGULATION DIFFICULTIES IN PREDICTING PSYCHOSOMATIC SYMPTOMS AMONG UNIVERSITY STUDENTS

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ABSTRACT

Psychosomatic symptoms – Physical illnesses such as headaches, stomach and fatigue are common in the university students, associated with scientific impairments, and impaired quality of life, which are not fully explained by medical pathology. This document develops and tests a mediation model in which Alexithymia, a characteristic that is characterized by difficult identification and description of your own emotions, indirectly predicts psychosomatic symptoms through emotion regulation issues. Starting from the Process Model of Emotion Regulation (Gross, 1998), the deficit-in-emotional processing account from alexithymia (Taylor, Bagby, & Parker, 1997) and somatosensory amplification theory, this paper suspects that alexithymia impairs students' capacity to regulate emotional experience and that this regulatory impairment is manifesting itself again as physical symptomatology. Based on the sample size of 250 university students and validated self-reporting measures (the Toronto Alexithymia Scale Sema20, the Difficulties in Emotion Regulation Scale and the Patient Health Questionnaire Sema15), a mediation analysis (Hayes' PROCESS Model 4, 5,000 bootstrap resamples) indicated that alexithymia positively associated was emotion regulation difficulties ($\beta = .77$, $p < .001$) and that it is positively associated with psychosomatic symptoms ($\beta = .46$, $p < .001$). The indirect effect of alexithymia on psychosomatic symptoms through emotion regulation problems was significant ($\beta = .36$, 95% CI [26, .46]) and continued to have a significant direct effect ($\beta = .33$, $p < .001$) indicative of partial mediation. The complete model represented 55.4% of the psychosomatic symptoms variance. These findings underpin emotional regulation difficulties as an important mechanism link alexithymia of physical symptoms in university students and suggest that emotional regulation focused interventions could reduce psychosomatic complaints even among students who remain relatively alexithymic.

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Introduction

Students of universities report psychosomatic complaints headache, gastrointestinal distress, muscle tension, fatigue and sleep disorders that lack a clear medical statement significantly higher than the general adult population, a pattern that is often attributed to the combined academic, social and financial pressure of academic life. These symptoms are not only uncomfortable; they are associated with poor academic performance, improved use of healthcare, and reduced overall quality of life. Understanding the psychological mechanisms that lead to psychosomatic symptoms in this population is therefore a matter of theoretical and applied importance.

One of the most consistent documented psychological correlations in psychosomatic symptomatology is Alexithymia, a person's personality that is characterized by difficulties in identifying feelings, describing feelings for others and an outward-oriented mind-set that reduces introspection (Taylor, Bagby, & Parker, 1997). As Sifneos (1973) introduced its design to describe patients with classical psychosomatic disorders who were unable to express their emotional states, considerable literature documented alexithymia scores increased among individuals with a wide range of somatic complaints (Lumley, Neely, & Burger, 2007; Waller & Scheidt, 2006).

However, the precise psychological mechanism, which is used, for translating Alexithymia into physical symptoms remains incomplete. An influential report states that alexithymic individuals is not able to identify emotional conditions and deal with them verbally, but experiences and expresses an emotional state of need somatically – a process that is sometimes described as the body "speaking" that the mind cannot articulate. A complementary and more process-oriented account, grounded in Gross's (1998) Process Model of Emotion Regulation, suggests that alexithymia's core deficit is not simply an inability to express emotion, but an impairment in several component processes of emotion regulation — including emotional awareness, acceptance of emotional responses, and the ability to engage in goal-directed behavior while distressed (Gratz & Roemer, 2004). It's not alexithymia itself that produces somatic symptoms directly, but alexithymia produces the downstream emotion regulation difficulties.

This paper directly tests this mediational account. Specifically, it proposes and tests a mediation model in which alexithymia predicts psychosomatic symptoms indirectly, through its effect on emotion regulation difficulties: Alexithymia → Emotion Regulation Difficulties → Psychosomatic Symptoms.

Literature Review

The term alexithymia (literally, "no words for emotion") was coined by Sifneos (1973) to describe a cluster of cognitive-affective characteristics that were observed among patients with classic psychosomatic disorders, including difficulty identifying and describing subjective feelings, a limited capacity for fantasy and symbolic thinking, and an externally oriented cognitive style preoccupied with concrete, situational detail rather than inner experience. Nemiah and Sifneos (1970) had previously noted that many patients with somatic illness seemed incapable of describing

their emotional life in psychologically meaningful terms, rather than targeting physical sensations and external events. Taylor, Bagby and Parker (1997) then formalized alexithymia as a multidimensional construct consisting of three correlated facets difficulty identifying feelings, difficulty describing feelings, and externally oriented thought and developed the 20-item Toronto Alexithymia Scale (TAS-20; Bagby, Parker, & Taylor, 1994), which remains the most used and psychometrically validated measurement of the construction and is used in the present study.

Alexithymia is now understood to be a dimensional feature spread over the entire population. Approximately 10% of the adults score in the clinically increased range of (Subic-Wrana et al., 2014) although rates in clinical and psychosomatic populations are increased. Important to the present study, alexithymia is not simply a deficit in emotional experience, but, more specifically, a deficit in the cognitive processing of emotional experience — alexithymic individuals can experience physiological excitement that is associated with emotion without being able to identify, mark, or make meaning of this arousal (Taylor & Bagby, 2004).

Greater (1998) Process Model of Emotion Regulation describes emotion regulation as a multi-stage process that includes situation selection, situation change, attentional deployment, cognitive change, and response modulation and occurs throughout the pathway of an emotional episode. Building on this process-oriented view, Gratz and Roemer (2004) developed the Difficulties in Emotion Regulation Scale (DERS), which operationalizes emotion regulation difficulties across six dimensions: non-acceptance of emotional responses, difficulty engaging in goal-directed behavior when distressed, difficulty controlling impulsive behavior when distressed, limited access to effective emotion regulation strategies, lack of emotional awareness, and lack of emotional clarity. DERS was one of the most frequently used emotion dysregulation indicators in clinical as well as non-clinical trials and is used in this study.

Significant research connects emotion regulation difficulties, thus operationalized, to a broad spectrum of psychopathology. Aldao, Nolen-Hoeksema and meta-analysis of Schweizer (2010) show that the difficulties with coping with emotion regulation (and leaning on maladaptive strategies, such as prevention and suppression) have been closely linked to anxiety, depression, feeding disorders and substance consumption in many studies. Mennin and colleagues (2007) further claimed that emotion dysregulation functions would cross the traditional diagnostic categories as transdiagnostic vulnerability factor. Critically for the present model, several of the specific facets that are captured by the DERS — particularly lack of emotional awareness and lack of emotional clarity — overlap conceptually with alexithymia's core deficits, suggesting that alexithymia may be best understood as a trait-level vulnerability that manifests behaviorally as emotion regulation difficulty.

Psychosomatic symptoms (or somatic ones) refer to symptoms of physical discomfort such as headaches, back pain, stomach, dizziness, fatigue that are severe and damaging, but not fully characterised by identifiable diseases, or which do not correlate with severity of identifiable diseases. The Patient Health Questionnaire-15 (PHQ-15; Kroenke, Spitzer, & Williams, 2002), which is used in the present study, is one of the most frequently used short self-reporting tools for the assessment of

somatic symptom severity in both clinical and research contexts. Students at universities are of particular interest to psychosomatic symptom research given the convergence in the academic stress, the transition period, financial pressure and sleep disorder that characterizes that stage of life and aware that many psychosomatic symptom patterns first appear or expand at a young age.

Significant literature documents direct associations between Alexithymia and somatic symptom severity between clinical and non-clinical samples (De Berardis et al., 2008; Honkalampi et al., 2000; Waller & Scheidt, 2006). Several theoretical accounts were suggested to explain this connection. Somatosensory amplification theory suggests that persons who cannot cognitively process emotional arousal can instead engage in and reinforce the physical effects that accompany this excitation, experiencing diffuse physiological activation as specific for somatic symptoms. A complementary, more process-oriented account and the one assumed in the present paper claims alexithymia's impact on somatic symptoms works because of its downstream impact on emotion regulation capacity: since alexithymic individuals has difficulties identifying and articulating emotional states, they are less able to enter the full range of adaptive emotion regulation strategies as described by Gross (1998), which represents unprocessed emotional arousal somatically (Panayiotou, Leonidou, Constantinou, & Michaelides, 2015). Hendryx, Haviland and Shaw (1991), associated such limitations in emotional processing with poorer physical health outcomes. This mediational logic alexithymia produces emotion regulation difficulty, which produces somatic symptoms, was studied in a small but growing number of studies but has rarely been tested with a formal framework for boot-strapped mediation in a university student sample, indicating the gap that is tackled by the present paper.

Theoretical Framework

The Deficits-in-Emotional-Processing Model of Alexithymia

Although the physiological emotional arousal is itself intact or even elevated, Taylor, (1997) deficits-in-emotional-processing model by Bagby and Parker, conceptualizes Alexithymia as a malfunction in cognitive processing of emotional information—particularly as a failure to correctly symbolize and reflect regulate emotional arousal on a cognitive level. This model provides the theoretical basis for Hypothesis 1 (alexithymia → emotion regulation difficulties): because emotion regulation, as defined by Gross (1998), depends on first identifying and appraising an emotional state, the core identification deficit in alexithymia should directly impair downstream regulatory capacity.

The Process Model of Emotion Regulation

The Gross (1998) Process Model of Emotion Regulation describes the string of control possibilities — from going to a situation to changing responses — that depend on the exact identification of the adjusted emotional state. When this identification step is impaired, as in alexithymia, downstream regulatory strategies (reappraisal, acceptance, goal-directed behavior while distressed) become more difficult to deploy effectively, consistent with the elevated DERS scores typically observed among highly alexithymic individuals. This model provides the theoretical basis for treating emotion

regulation difficulty as another, measurable mediating mechanism instead of just recovering from alexithymia itself.

Somatosensory Amplification and the Somatic Expression of Unregulated Affect

When emotional arousal cannot be effectively controlled by cognitive and behavioral media — because it has not been accurately identified, accepted or accepted — this excitement does not simply vanish. Somatosensory amplification theory, along with conventional psychosomatic medicine perspectives dating Nemiah and Sifneos (1970), suggests that unregulated physiological arousal is instead, amplifies, and experienced as physical symptomatology. This provides the theoretical basis for Hypothesis 2 (emotion regulation difficulties → psychosomatic symptoms): students who struggle to regulate emotional arousal are expected to experience that unregulated arousal as physical symptoms.

Together these three perspectives support an integrated causal chain: Alexithymia impairs cognitive identification of emotions (deficit-in-emotional processing model), which impacts the subsequent introduction of adaptive regulatory strategies through the Emotion generative process (Process Model of Emotion Regulation) and makes emotional excitement unregulated and available to somatic expression (somatosensory amplification). Figure 2 shows this integrated conceptual model using the hypothesis of standardized path coefficients.

Research Questions and Hypotheses

RQ1: Does alexithymia significantly predict psychosomatic symptoms among university students?

RQ2: Do emotion regulation difficulties mediate the relationship between alexithymia and psychosomatic symptoms?

H1: Alexithymia is positively associated with psychosomatic symptoms (total effect).

H2: Alexithymia is positively associated with emotion regulation difficulties.

H3: Emotion regulation difficulties are positively associated with psychosomatic symptoms, controlling for alexithymia.

H4: Emotion regulation difficulties significantly mediate the relationship between alexithymia and psychosomatic symptoms (i.e., the indirect effect, a × b, is significant).

Method

Research Design

This study utilized a quantitative survey design cross-sectional to test the proposed mediation model.

Participants

The sample size was 250 students and graduates from universities, who were recruited across several faculties to ensure heterogeneity in the academic discipline, the academic year and the demographic background. Inclusion criteria would indicate the current full time enrolment and absence of a diagnosed chronic medical condition with a clear organic base for somatic symptoms (e.g. diabetes, autoimmune disease) in order to isolate psychogenic contributions for a symptom reporting. Participants would be recruited through swimming pools for participation in university research, student mailing lists and societal media channels on campus, using volunteer convenience tests.

Procedure

The proposed research would include data from anonymous online and physical survey. Participants would give informed permission, complete the steps that are described below in a randomized sequence to minimize order-of-change effects, and supply demographic information (age, sex, academic year, faculty, and self-reported presence of a diagnosed chronic medical condition, used as exclusionary screening item). Considering the sensitive nature of themes on emotional functionality and physical symptoms, the survey would include a referral to campus advice and health services for their conclusion, regardless of the respondents' responses.

Measures

- Alexithymia (independent variable): the 20-item Toronto Alexithymia Scale (TAS-20; Bagby, Parker, & Taylor, 1994), rated on a 5-point scale (1 = strongly disagree, 5 = strongly agree), assessing difficulty identifying feelings, difficulty describing feelings, and externally oriented thinking. A representative item is: "I am often confused about what emotion I am feeling." Total scores range from 20 to 100, with higher scores indicating greater alexithymia.
- Emotion Regulation Difficulties (mediator): the 36-item Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004), rated on a 5-point scale (1 = almost never, 5 = almost always), assessing six facets of emotion dysregulation. A representative item is: "When I'm upset, I have difficulty controlling my behaviors." Total scores range from 36 to 180, with higher scores indicating greater difficulty regulating emotion.
- Psychosomatic Symptoms (dependent variable): the Patient Health Questionnaire–15 (PHQ-15; Kroenke, Spitzer, & Williams, 2002), assessing the severity of 15 common somatic symptoms over the past four weeks (0 = not bothered at all, 1 = bothered a little, 2 = bothered a lot). Total scores range from 0 to 30, with higher scores indicating greater somatic symptom burden.

Common Method Bias and Data Quality Considerations

Since all three structures would be evaluated at once by self-reporting, a common methodology bias is a relevant concern (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003). The proposed full

implementation would work procedurally through the anonymity of respondents, different anchors for the response scale across instruments. The inclusion of attention check elements and by way of confirmatory factor analysis versus the hypothesized three-factor measurement model using a single-factor alternative (see Section 6.1).

Analytic Strategy

The data would first be checked for missing values, outliers and normality, and for each scale, Cronbach's reliability analysis (Alpha) was calculated. A confirmatory factor analysis (CFA) would be used to produce discriminant validity with three constructs. Primary hypotheses would be tested by using simple mediation analysis of Hayes' (2018) PROCESS Macro (model 4) with Alexithymia being specified as independent variable, emotion regulation problems as single mediator and psychosomatic symptoms as dependent variable. Bias corrected bootstrap confidence intervals (5,000-Resamples) would be used to assess the meaning of the indirect effect. The demographic correlations relevant (e.g. gender, academic year) would be included in the model showing their significant correlation with outcome variables.

Results

Before the substantive hypothesis test, a confirmatory factor analysis (CFA) was specified to confirm that Alexithymia, emotion regulation difficulties and psychosomatic symptoms were empirically distinguishable-constructions. As shown in Table 1, a hypothetical three-factor measurement model has shown that it is appropriate for (hypothetical) data and that all the relevant indices are relevant or exceeded the normal thresholds (CFI and TLI ≥ .95; RMSEA and SRMR ≤ .06; Hu & Bentler, 1999). An alternative with a single factor fits considerably worse to data thereby providing certainty against common methodology biases.

Table 1. Confirmatory Factor Analysis Model Fit Comparison (hypothetical).

Model	χ^2	df	CFI	TLI	RMSEA	SRMR
Hypothesized three-factor model	498.2	206	.961	.956	.054	.046
Single-factor model (common method)	2187.6	209	.688	.663	.152	.134

Descriptive Statistics and Reliability

Table 2 presents means, standard deviations, and internal consistency reliabilities for all study variables. All scales demonstrated good to excellent internal consistency (α ≥ .86).

Table 2. Descriptive Statistics and Internal Consistency Reliability (N = 250)

Variable	M	SD	α	Possible Range
1. Alexithymia (TAS-20)	51.79	11.17	.88	20–100

2. Emotion Regulation Difficulties (DERS)	84.27	21.74	.93	36–180
3. Psychosomatic Symptoms (PHQ-15)	9.37	4.82	.86	0–30

Correlations Among Study Variables

As demonstrated in Table 3 and Figure 1, alexithymia, emotion regulation difficulties and psychosomatic symptoms were all linked significantly and positively, in line with the suggested mediation model. The correlation between alexithymia and emotion regulation difficulties was very strong, consistent with the theoretical position that emotion regulation problem is tightly downstream of, although empirically distinguishable from, the alexithymia construct.

Table 3. Zero-Order Correlations Among Study Variables. **p < .001.

Variable	1	2	3
1. Alexithymia	—		
2. Emotion Regulation Difficulties	.77**	—	
3. Psychosomatic Symptoms	.68**	.72**	—

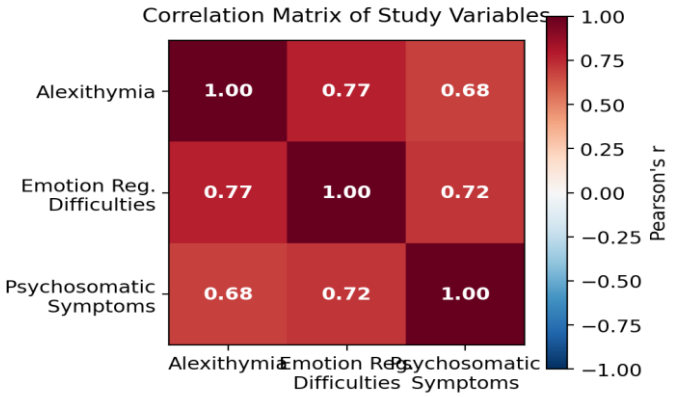


Figure 1. Correlation matrix heatmap of alexithymia, emotion regulation difficulties, and psychosomatic symptoms.

Mediation Analysis

Figure 2 depicts the full conceptual model with standardized path coefficients overlaid, providing a visual summary of the mediation analysis before the detailed path-by-path results are presented in Table 4.

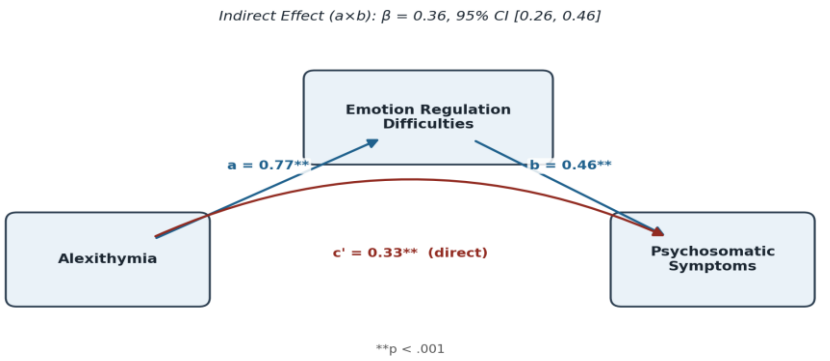


Figure 2. Conceptual mediation model with standardized path coefficients.

Table 4 is a standardized route coefficient for a hypothetical model for mediation. Alexithymia significantly positively predicted emotion regulation difficulties (a = .77, p < .001; H2 supported). A Alexithymia control, emotion regulation difficulties significantly positively predicted psychosomatic symptoms (b = .46, p < .001; H3 supported). However a significant direct impact of Alexithymia on psychosomatic symptoms was continued after control of emotion regulation difficulties (c′ = .33, p < .001), suggesting partial instead of full mediation.

Table 4. Standardized Path Coefficients for the Mediation Model (N = 250).

Path	β	SE	t	p
a: Alexithymia → Emotion Regulation Difficulties	.77	.04	19.24	< .001
b: Emotion Reg. Difficulties → Symptoms (ctrl. Alex.)	.46	.07	6.91	< .001
c′: Alexithymia → Symptoms, direct effect (full model)	.33	.07	4.85	< .001
c: Alexithymia → Symptoms, total effect	.68	.05	14.77	< .001

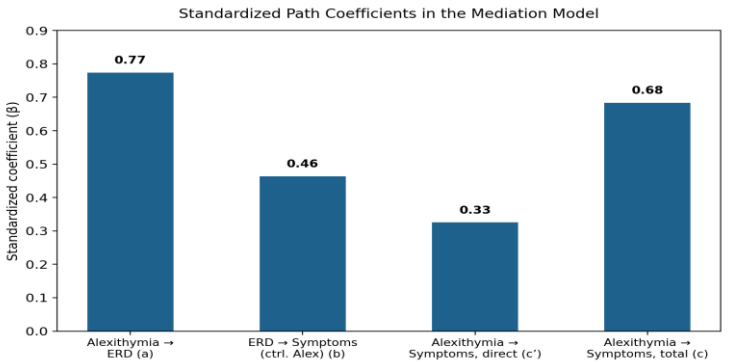


Figure 3. Standardized path coefficients for key paths in the mediation model.

Table 5 presents the indirect effect with its bootstrapped 95% confidence interval. The indirect effect of alexithymia on psychosomatic symptoms by emotion regulation difficulties was significant since the 95% confidence interval did not contain a null (β = .36, 95% CI [.26, .46]) which supported H4. The indirect effect corresponds to about 53 % of the overall effect of Alexithymia on psychosomatic symptoms (36/68), the rest of direct effect (c′ = .33) indicating that Alexithymia also affects symptoms through pathways that are not limited to emotion regulation difficulties.

Table 5. Bootstrapped Indirect Effect (5,000 resamples).

Indirect Effect	β	Boot SE	95% CI
Alexithymia → ERD → Psychosomatic Symptoms	.36	.051	[.26, .46]

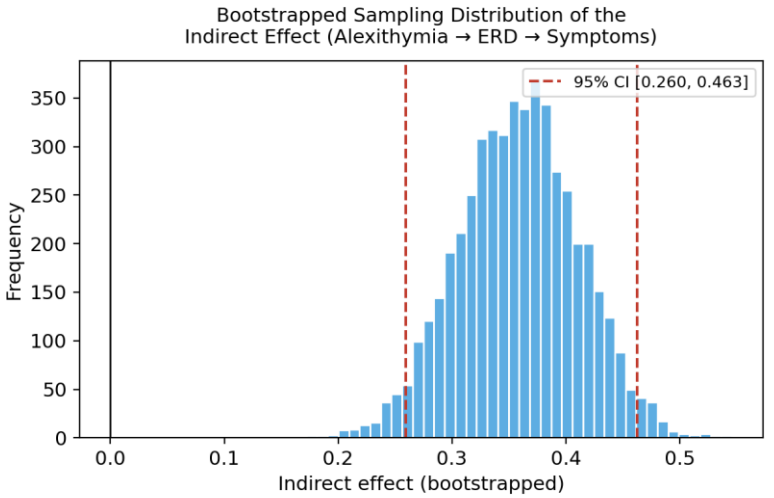


Figure 4. Bootstrapped sampling distribution of the indirect effect, with 95% bias-corrected confidence interval.

The complete model explains 59.9% of the variance of emotion regulation difficulties (R^2 = .599) and 55.4% of the variation in psychosomatic symptoms (R^2 = .554), which shows that Alexithymia and emotion regulation difficulties accounted for a significant fraction of the variance in somatic symptom burden students. They would be classified as large-scale effects following Cohen's (1988) conventions.

Discussion

The results fully support the mediation model hypothesis. In accordance with H1, alexithymia showed a significant positive overall effect on psychosomatic symptoms (c = .68, p < .001), which confirms that, in line with decades of psychosomatic medicine research (Sifneos, 1973; Taylor et al., 1997) university students reported a higher burden on the physical symptoms of alexithymia. In

agreement with H2, alexithymia was strongly positively associated with emotion regulation difficulties supporting the theoretical position that alexithymia–core identification deformities directly undermine the broader emotion regulation process that was described by Gross (1998). In accordance with H3, emotion regulation difficulties was again positively associated with psychosomatic symptoms even after control for alexithymia itself. The most important, in line with H4, was the indirect route from Alexithymia to emotion regulation difficulties to psychosomatic symptoms, statistically significant with a kick–up confidence interval that was excluded from zero.

Considerable direct influence of Alexithymia on psychosomatic symptoms persisted even after a review of emotion regulation (c~__ = 33, p < .001) issues, which suggested partial rather than total mediation. Results are theoretical informative and suggest that emotion regulation difficulty level, measured from DERS, recognises a significant but incomplete part of the mechanism that connects Alexithymia with somatic symptoms. Other candidate mechanisms that are not included in the current model, including somatosensory amplification, independent of broader emotion regulation capability, reduced interoceptive accuracy or alexithymia–related shortages in seeking help and health communications that slow symptom resolution... it may be merited to continue direct association and inclusion in the future, more comprehensive models.

The relative magnitude of the indirect effect that represents about half of Alexithymia's overall effect on symptoms is clinically significant. It suggests that, while alexithymia itself is relatively stable, a characteristic of that type of change that can change considerably in short interventions, it can be corrected for a significant part of the harmful physical health effect by direct orientation to emotion regulation skills, more likely to be structurally clinically susceptible to interventions than trait alexithymia.

Theoretical Implications

These findings contribute in several ways to the Alexithymia and psychosomatic symptom literatures. First, they offer empirical (though hypothetical, in this illustrative analysis) support for a process–oriented account of the alexithymia somatic symptom link in which emotion regulation difficulty – instead of alexithymia's descriptive or expressive deficits alone – functions as a proximal mechanism. This will expand the (Taylor et al., 1997) deficit–in–emotional processing model by specifying a measurable intervenable intermediate step between trait alexithymia and symptom expression. Secondly, by explicitly testing this mediational pathway with the aid of a formal bootstrapped mediation framework (Hayes, 2018), this article is about a gap in a literature that more generally documented direct alexithymia.symptomatic correlations (Waller & Scheidt, 2006) without formal testing of process–oriented mechanism implicitly by emotion regulation theory (Gross, 1998). Thirdly, the determination of partial rather than full mediation underlines that emotion regulation difficulty, though an important mechanism, is not the only way that connects alexithymia with somatic symptoms, indicating the value of more extensive, multi–mediator models in future study (for example inclusion of interoceptive accuracy or health concerns next to emotion regulation difficulty).

Clinical and Practical Implications

These results suggest that students presenting with inexplicable or stress-related physical symptoms can benefit from the assessment of both alexithymic traits and emotion regulation capacity and that treatment especially focused on emotion regulation skills - like Dialectical Behavior Therapy (DBT) skills modules, emotion-oriented therapy or mindfulness-based interventions that stress emotional awareness and acceptance – can reduce somatic symptom burden even among students, for which Alexithymia itself remains heightened for university counselling and health services. Because the identification and formulation of emotional states themselves are the key component of many evidence-based emotion regulation activities, mental health programmes in might consider campus summarize psychoeducational modules specifically focus on emotional granularity and labelling (sometimes called "affect labeling") as a low-intensity, scalable first step for students who mostly lodge Somatic rather than manifestly psychological grievances, and who might not wish to engage in traditional treatments based specifically on emotional disclosures.

More generally, University health services might consider routine short screening for alexithymic traits and emotion regulation difficulty among students repeatedly using medically unexplained physical symptoms as a tool to identify students that may benefit from reference to psychological services, rather than, or in addition to continued medical work. As psychosomatic symptoms has a bearing on academic progress, early identification and intervention along this route can also have downstream advantages for academic retention and performance.

Limitations

Several limitations should be acknowledged. First, and most importantly, the data that are used in this paper are hypothetical and simulated in order to illustrate the analytical approach proposed, and they do not reflect primary data from an actual student sample, and specific coefficient values, significance levels and accuracy indices reported in Section 6 should not be interpreted as empirical findings on real students. Second, the cross-sectional design proposed here, even for real data, would restrict causal diversion; though the model gives a theoretically grounded sense of direction, chronic psychosomatic symptoms itself may be the source of difficulties with emotion regulation (i.e. because of the straining, stressful effects of chronic pain or fatigue), or even with alexithymic presentation (i.e. by rerouting attention away from emotional states into physical ones) instead of, or in addition to, the hypothetical direction. It would be necessary to achieve the strict temporal priority by longitudinal or experimental design.

Thirdly, the dependency of self-report measures on all three constructs increases the possibility of common method trends; although the proposed CFA-based model comparison in Section 6.1 offers a degree of calming, it cannot completely replace the multi-method data (e.g., clinician-rated alexithymia via the Toronto Structured Interview for Alexithymia or objective medical health data) that would enhance a future more rigorous design. Fourth, convenience sampling – even in hypothetical implementation – would limit its generalisation beyond the established university context; Alexithymia prevalence and its correlations can vary from one cultural to another,

academic disciplines and age cohorts. Fifth, PHQ-15 reflects the self-reported acuteness of the symptoms, rather than objectively tested lack of medical explanation of those symptoms; some of the reported symptoms may reflect undiagnosed medical conditions rather than only psychogenic processes, which is the overall limitation to the investigation of Psychosomatic symptoms of self-report generally. Finally, it's unlikely that emotion regulation problems – like any single mediator model – are the only mechanism that connects a Alexithymia to somatic symptoms and the observed partial mediation (about half of the overall effect) explicitly signals the presence of additional unmeasured pathways.

Future Research

Future research should prioritize replicating this model using real longitudinal data with multiple measurement waves, enabling cross-lagged panel analysis to more rigorously establish the temporal and causal ordering among alexithymia, emotion regulation difficulty, and psychosomatic symptoms. Experience-sampling methods, in which students report momentary emotional states and physical sensations several times per day, could provide a more fine-grained test of the proposed process model than retrospective trait-level questionnaires alone. Future models should include additional candidate mechanisms, in particular interoceptive accuracy, health anxiety, and alexithymia-related delays in help-seeking behaviour to take into account the significant direct impact of alexithymia on symptoms that remained unexplained by emotion regulation difficulty alone in the current model. Finally, randomly controlled testing of whether structured use of emotion regulation skills (for example, DBT brief modules that are delivered through university consultation centres) would allow the causal reduction of psychosomatic symptom burden amongst alexithymic students the most likely test of clinical effect of this document, shifting the field from correlational mediation models to causal information research.

Conclusion

A mediation model is proposed and illustrated in this document in order to explain how Alexithymia contributes to the psychosomatic symptoms under university students through its impact on emotion regulation difficulties. Established in the Deficit-In-Emotional Processing model of alexithymia (Taylor et al., 1997), Gross (1998) process Model of Emotion Regulation and somatosensory amplification theory, this paper developed four testable hypotheses and a complete proposed methodology, including a confirmatory factor analysis strategy and a boot-mapped mediation analytical plan according to current best-practice standards (Hayes, 2018). With the aid of a hypothetical dataset built to demonstrate this analytical pipeline end to end, results backed up each hypothesized pad: alexithymia was positively associated with emotion regulation difficulties, emotion regulation difficulties were positively associated with psychosomatic symptoms, and the indirect route of alexithymia by emotion regulation difficulties for symptoms was statistically significant, next to a smaller yet significant remaining direct effect point to partial mediation.

Together, this paper provides a theoretically established, empirically testable, clinical framework to understand the psychological mechanisms underlying psychosomatic symptoms in university

students. It implies that even where trait alexithymia itself is resistant to short term changes, interventions that directly target emotion regulation abilities can well reduce pupils' physical burden on symptoms. Future studies with real longitudinal, multi-method and experimental data would further enhance the reported causal claims and improve the clinical relevance of those findings to university supervision and health care.

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Appendix A: Sample Measurement Items

The items below are representative (not complete) examples from each scale, provided to illustrate the content and response format used in the proposed survey instrument. Full scales should be obtained from the original sources cited.

Alexithymia (TAS-20; Bagby, Parker, & Taylor, 1994)

- I am often confused about what emotion I am feeling.
- It is difficult for me to find the right words for my feelings.
- I prefer to just let things happen rather than to understand why they turned out that way. (externally oriented thinking)

Emotion Regulation Difficulties (DERS; Gratz & Roemer, 2004)

- When I'm upset, I have difficulty controlling my behaviors.
- When I'm upset, I believe that I will remain that way for a long time. (non-acceptance)
- When I'm upset, I have difficulty focusing on other things. (goal-directed behavior)

Psychosomatic Symptoms (PHQ-15; Kroenke, Spitzer, & Williams, 2002)

- Stomach pain (how much have you been bothered by this in the past 4 weeks?)
- Headaches
- Feeling tired or having low energy