

Article

Autonomic Arousal and Perseverative Cognition Are Associated with Depression and Suicidal Ideation: A Moderated-Mediation Model

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Abstract

Perseverative cognition—frequently expressed through obsessive–compulsive symptoms—is a recognized trans-diagnostic risk factor for psychopathology, with documented links to depression and suicidal ideation. While the literature suggests that altered autonomic arousal in depression may relate to suicidal risk, the conditional architecture linking these variables remains insufficiently explored. This study examined whether suicidal ideation mediates the association between depression and autonomic arousal and whether obsessive–compulsive symptoms moderate the initial pathways within this mechanism. A sample of 120 university students (61.8% female; $M_{age} = 28.6$, $SD_{age} = 10.71$) completed the Symptom Checklist-90-Revised (SCL-90-R), from which the depression, obsessive–compulsive, and suicidal ideation scores were derived; notably, the suicidal ideation score was calculated from specific items (15 and 59) embedded within the depression subscale. All participants underwent a psychophysiological evaluation to record baseline Electrodermal Activity (EDA) as an index of tonic autonomic arousal. Results indicated that depression significantly predicted suicidal ideation ($B = 0.05$, $SE = 0.006$, $p < 0.001$, 95% CI [0.03, 0.06]), which, in turn, was a significant predictor of autonomic arousal ($B = 0.17$, $SE = 0.08$, $p = 0.03$, 95% CI [0.02, 0.33]). Additionally, the mediation analysis revealed that depression showed an indirect statistical association with autonomic arousal through suicidal ideation (Bootstrapped 95% CI [0.01, 0.12]). Concurrently, obsessive–compulsive symptoms significantly moderated the psychological link between depression and suicidal ideation ($B = 0.20$, $SE = 0.04$, $p < 0.001$, 95% CI [0.04, 0.37]), accounting for 38.40% of the variance ($F(4, 115) = 22.17$, $p < 0.001$). Sensitivity analyses, conducted by re-calculating the models after excluding items 15 and 59 from the depression score, confirmed the robustness of these findings against potential psychometric overlap. However, the formal Index of Moderated Mediation was not statistically significant (0.0000 (95% Boot CI [−0.0001, 0.0001])), explicitly demonstrating that this psychological moderation did not translate into a significant conditional indirect effect on peripheral autonomic arousal. These findings suggest that while obsessive cognitive patterns significantly intensify the psychological burden of depression into thoughts of death, their concurrent association with peripheral physiological parameters remains strictly exploratory in non-clinical cohorts, underscoring a nuanced underscoring a nuanced ‘blocking’ effect—both emotional and behavioral—where obsessive rigidity functions as a mechanism of paralysis that, while generating profound subjective distress, fails to translate into a corresponding reactive physiological response of the autonomic nervous system.



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1. Introduction

According to a 2019 World Health Organization report, suicide is among the leading causes of death worldwide, with 703,000 people dying by suicide each year. In Italy, approximately 4000 young people kill themselves every year [1]. By way of illustration, for every completed suicide, an estimated 20 suicide attempts occur [2], and suicidal ideation is a significant risk factor for suicide attempts [3].

Yale Medicine defined suicidal ideation as a set of thoughts, fantasies, or contemplation about ending one's own life. The intensity of these thoughts moves along a spectrum, going from passive (described as a general desire to die, but without any intent or plan) [4] to active suicidal ideation (supported by actual intentions and plans about dying), eventually leading to effective suicidal behaviours [5].

From a psychological point of view, internal entrapment (perception of entrapment by one's own thoughts and feelings) and perceived burdensomeness (the feeling of being a burden to others) seem to be among the most significant factors that characterize suicidal ideation [6].

These highly negative experiences generally occur during peak exacerbation of depressive symptoms [7] and are associated with intense distress [8]. In the clinical setting, this phenomenon is frequently referred to as "psychache" [9]. In 95% of cases, suicide occurs in individuals with a history of at least one mental disorder [10]. Specifically, suicidal ideation is common in depressive and anxiety disorders, which are also the most commonly diagnosed psychiatric categories [11,12].

At a physiological level, suicidal ideation is characterized by a state of hyperarousal of the Sympathetic Nervous System (SNS), the branch of the Autonomic Nervous System (ANS) that activates the fight-or-flight response [13]. Autonomic arousal can be described using specific psychophysiological indices, such as the electrodermal activity (EDA) that reflects the activity levels of the eccrine sweat glands and, therefore, the activity of the SNS [14,15].

There are contrasting opinions in the literature regarding the relationship between depressive symptoms and ANS. Some studies reported the presence of hyperarousal, while others linked it to hypoarousal [16,17]. Nevertheless, depression is considered an antecedent of suicidal ideation [18].

Another factor linked to both suicidal ideation and depression is perseverative cognition, a transdiagnostic construct encompassing thinking styles characterized by repetitive, intrusive, and distressing thoughts [19]. It is characterized by a propensity for negative cognitive content, poorer problem-solving skills, and the development of an attentional bias toward negative information, ultimately leading to increased perception of negative stimuli [18,19]. Manifesting through obsessive-compulsive symptoms [18,19], perseverative cognition captures the component of cognitive inflexibility and the risk of using compulsion as a maladaptive mechanism to neutralize the tension generated by thoughts [20]. Perseverative cognition is widely recognized as a transdiagnostic risk factor [20,21] as it has been associated with the severity of depressive symptoms, fueling suicidal ideation [22,23]. Nevertheless, at the psychophysiological level, perseverative cognition is linked to autonomic hyperarousal as measured by EDA [24].

As mentioned above, the literature on suicidal ideation documented a close association between internal entrapment and an increased risk of progressing to suicidal ideation [25]. In particular, internal entrapment has been linked to the desire to escape from internal feelings and emotions [26]. There is also scientific evidence demonstrating how perseverative cognition, a characteristic of obsessive–compulsive symptoms, can maintain the body in a state of prolonged stress through the absence of habituation [27]. In individuals with high obsessive–compulsive symptoms, the internal threat posed by suicidal ideation appears to be amplified by the tendency towards perseverative cognition [28]. Consequently, the inability to divert attention from the suicidal trigger might lead to autonomic hyperarousal, suggesting that compulsive traits might act as catalysts that transform a transient thought into a persistent state of physiological emergency [29].

Nonetheless, it is also necessary to emphasize that suicidal ideation itself can represent an internal phobic stimulus capable of mediating psychological distress, favoring the transition from depression to autonomic arousal [30]. In agreement with the internal phobic trigger hypothesis, while depression is often characterized by emotional dulling or reduced reactivity, the emergence of explicit thoughts of death can act as an acute psychological stressor [28]. These thoughts may represent an internal threat that triggers an immediate autonomic alarm, as evidenced by the significant increase in EDA [27].

Despite extensive scientific evidence about the relationships between depression, suicidal ideation, autonomic arousal, and perseverative cognition (i.e., obsessive–compulsive symptoms), to date, little is known about the interaction mechanisms linking these variables of interest.

The present study was conducted precisely to analyze the moderating role of perseverative cognition and the mediating role of suicidal ideation in the relationship between depression and autonomic arousal.

The following hypotheses were formulated to guide the study: (1) depression directly predicts suicidal ideation and indirectly predicts autonomic arousal; (2) suicidal ideation mediates the relationship between depression and autonomic arousal; (3) perseverative cognition (i.e., obsessive–compulsive symptoms) moderates the relationship between depression and suicidal ideation.

Shedding light on the psychological and psychophysiological pathways linking depression to autonomic arousal might be useful to offer relevant implications for suicide risk prevention and for clinical interventions targeting emotional and physiological regulation.

2. Materials and Methods

2.1. Study Design

The study was exploratory and cross-sectional.

2.2. Participants

Participants were recruited consecutively through invitations sent via the university's institutional mailing list and posters offering a link to schedule an appointment at the Clinical Psychology, Clinical Psychophysiology, and Clinical Neuropsychology Laboratories of the Department of Medicine and Surgery at the University of Parma. After describing the purpose of the study and the experimental procedure, the researchers offered participants the opportunity to obtain personalized feedback on the results.

Inclusion criteria included a minimum age of 18 years, completion of an informed consent form, and the absence of neuropsychiatric (e.g., previous head trauma, epilepsy, etc.) and/or physical (e.g., sensory deficits in vision and/or hearing) conditions that could compromise the proper implementation of the experimental protocol. However, participants were not taking any psychotropic medications that affected the ANS in the

past three months (e.g., tricyclic antidepressants, antipsychotics, etc.). Lastly, participants were included if they provided complete psychometric assessments and if their EDA signal was free of excessive motion artifacts during the recording.

The research was conducted following the 2020 Guidelines for Good Research Practice of the University of Parma. All procedures were conducted following the 1964 Declaration of Helsinki, as revised in 2024, as well as the 2005 Universal Declaration on Bioethics and Human Rights of the UNESCO regarding research involving human participants. The protocol was approved by the Ethics Committee of the University of Parma (protocol number: 0311528-2025 and date of approval 29 October 2025).

2.3. Measures

Depression and obsessive–compulsive symptoms were investigated through the Symptom Checklist-90-Revised (SCL-90-R) [31], a widely used standardized instrument designed to assess the presence and severity of psychological symptoms. It comprises 90 items, each rated on a 5-point Likert scale (1 = “not at all” to 5 = “extremely”), reflecting the frequency of internalizing and externalizing experiences over the past seven days. Among others, the SCL-90-R includes 13 items for depressive symptoms and 10 items for obsessive–compulsive symptoms. Normative values are distributed on a T-point scale with a mean equal to 50 and a standard deviation (SD) equal to 10. Following the caseness criterion, values of 63 or higher (+1.5 SD) indicate psychological distress. Internal consistency ranges from acceptable to excellent, with Cronbach’s α coefficients equal to 0.93 for Obsessive–Compulsive and to 0.95 for Depression.

Suicidal ideation was assessed by extrapolating two specific items from the depression scale of the SCL-90-R [32]. Specifically, item 15 (“Thoughts of ending one’s life”) and item 59 (“Thoughts of death or dying”) were considered simultaneously as they are believed to be comparable to the Columbia Suicidal Severity Rating Scale, which assesses suicidal ideation by asking “Have you ever wanted to die or wanted to go to sleep and never wake up?” and “Have you ever had thoughts of suicide in your life?” [33]. To maintain the psychometric integrity of the SCL-90-R, these items were retained in their standardized form within both the depression subscale (X) and the mediator (M). To address potential concerns regarding part–whole correlation inflation, a sensitivity analysis was performed by recalculating the mediation model using a depression subscale score that explicitly excludes items 15 and 59. Additionally, for descriptive purposes and to identify high-risk profiles, suicidal ideation was operationally defined as a binary categorical variable, where any non-zero score on both items was considered indicative of suicidal distress. For the moderated mediation analysis, a continuous score was calculated by summing the raw scores of these two items (ranging from 0 to 8), providing a measure of the severity of suicidal ideation. Finally, to ensure the robustness of the mediation findings against the specific distributional characteristics of these items (i.e., zero-inflation), a three-level clinical gradient was employed (Absent, Low, and High ideation), providing a more granular assessment of the indirect effects across different levels of severity [34,35].

Autonomic arousal was measured by recording the EDA during a Psychophysiological Evaluation [36]. To ensure signal stability and control for potential confounding variables, a rigorous psychophysiological protocol was implemented. Participants were instructed to abstain from caffeine consumption and smoking for at least 2 h before the assessment. Participants were asked to sit quietly to allow for autonomic stabilization. To minimize movement artifacts, participants were instructed to remain as still as possible, with their feet placed on the ground at a 45-degree angle and their arms resting comfortably on the armrests. Environmental conditions were strictly controlled, with the room temperature maintained between 19 °C and 21 °C throughout the entire recording duration. Following

a 4-min adaptation period to allow for signal stabilization, a 5-min resting baseline was recorded. During this phase, participants were instructed to remain still and breathe naturally to capture tonic autonomic activity in a neutral state. EDA was continuously recorded at a sampling rate of 256 Hz using a Biograph ProComp Infiniti 5.0 system (Thought Technology, Montreal, Quebec). Two silver-plated electrodes were placed on the distal phalanges of the index and middle fingers of the non-dominant hand. For data analysis, EDA signals were visually inspected for movement artifacts and corrected via linear interpolation where necessary. The Skin Conductance Level (SCL) was extracted as the mean tonic value of the baseline period. To meet the assumptions of normality and address the positive skewness of the raw data, all EDA values were log-transformed ($\log_{10}[\text{EDA} + 1]$) before inferential statistical analysis.

2.4. Statistical Analysis

All statistical analyses were performed using SPSS (Version 28.0.1.0; IBM Corp, Armonk, NY, USA). Descriptive statistics of the SCL-90-R scores and log-EDA values were performed with the calculation of the mean (M) and standard deviation (SD).

A consistent threshold of +1.5 SD was adopted across all measures to identify high-risk profiles. In the SCL-90-R, this cut-off aligns with the standard clinical T-score of 63–65 recommended for case identification [31]. Similarly, this conservative criterion was applied to log-EDA values to operationally define excessive autonomic arousal. Thus, the formula used corresponds to $\text{mean} + (\text{SD} \times 1.5)$. Utilizing such distributional cut-offs allows for a rigorous differentiation between typical and atypical stress responses within a non-clinical sample [35,37], ensuring methodological integration between psychometric and physiological dimensions.

Pearson's correlation analysis was conducted to examine the association between depressive and obsessive–compulsive symptoms, suicidal ideation, and log-EDA values.

To test the research hypotheses, a moderated mediation analysis was performed using Model 8 of the PROCESS macro [38] for SPSS. This specific model was selected a priori as the most appropriate statistical framework to represent the internal phobic trigger hypothesis and the perseverative cognition framework. In this model, depression was entered as the independent variable (X), suicidal ideation as the mediator (M), and log-EDA values (i.e., autonomic arousal) as the dependent variable (Y). Obsessive–compulsive symptoms (i.e., perseverative cognition) were included as the moderator (W) of the relationship between depression and suicidal ideation. Additionally, to evaluate the robustness of the mediation model against the zero-inflated distribution of the suicidal ideation score, a sensitivity analysis was performed. The mediator was re-coded into an ordinal variable with three levels: Absent (score = 0), Low (score between 1 and 4), and High (score between 5 and 8) ideation. Furthermore, as previously mentioned, a formal sensitivity analysis was conducted to test the model's stability against potential item overlap, re-running the mediation path with a depression score excluding items 15 and 59. This three-tier categorization was adopted to simultaneously manage the zero-inflation of the data and to respect the clinical distinction between the absence of symptoms, occasional thoughts, and more persistent ideation, ensuring the effect is not driven solely by a binary distinction [35,38]. The significance of the indirect effects was assessed using 5000 bootstrap resamples to generate 95% bias-corrected confidence intervals (CIs). An effect was considered significant if the 95% CI did not include zero.

3. Results

3.1. Descriptive Statistics and Correlation

A total of 120 students (61.8% were females) of the University of Parma (Northern Italy), aged between 19 and 45 years old ($M_{age} = 28.6$, $SD_{age} = 10.71$) were included. The mean age of the sample was slightly higher than that of typical undergraduate populations; the inclusion of 24 working students (16.7% of the total sample) contributed to an increase in the overall age distribution. Participants had a bachelor's degree (46.5%) or a diploma (53.5%), and the majority were single (79.8%) (only 20.2% were cohabiting or married).

The descriptive statistics of the variables of interest are reported in Table 1 along with the correlations between them.

Table 1. Descriptive statistics and Pearson correlation matrix (N = 120).

	M (SD)	Threshold	F (%)	1	2	3	4	5
1 SCL-90-R depression (T)	65.64 (18.28)	≥ 63	49 (40.8%)					
2 SCL-90-R obsessive-compulsive (T)	67.36 (18.98)	≥ 63	69 (57.5%)	0.79 **				
3 SCL-90-R suicidal ideation (R)	0.68 (1.76)	> 0	27 (22.5%)	0.58 *	0.45 **			
4 log-EDA baseline value	0.60 (0.76)	> 1.74	5 (4.2%)	−0.02	0.002	0.18 *		
5 Gender	-	-	89 (74.2%)	0.11	0.11	−0.001	−0.18 *	
6 Age	28.6 (10.71)	-	-	−0.22 *	−0.14	−0.08	−0.23 **	0.001

Legend: F (%) = Frequency and percentage of clinical cases; log-EDA = logarithmic conversion of Electrodermal Activity values; M = mean; SCL-90-R = Symptom Checklist-90-Revised. SD = standard deviation. Note: (T) indicates T-scores (M = 50, SD = 10); (R) indicates raw scores. For Gender, F (%) represents the number and percentage of female participants. Gender was coded as 0 = male, 1 = female. Significance levels are based on two-tailed tests: * $p < 0.05$; ** $p < 0.01$.

The clinical characterization of the sample (N = 120) revealed significant levels of distress: 40.8% of participants exceeded the clinical threshold ($T > 63$) for depression, and 57.5% met the criteria for obsessive–compulsive symptoms. Notably, 22.5% of the sample reported the presence of suicidal ideation. Concerning autonomic arousal, 4.2% of participants showed a log-EDA baseline level above the threshold of 1.74.

Correlation analysis showed a strong and significant association between depression and obsessive–compulsive symptoms ($r = 0.79$, $p < 0.01$). Suicidal ideation was positively correlated with both depression ($r = 0.58$, $p < 0.05$) and obsessive–compulsive symptoms ($r = 0.45$, $p < 0.01$). Crucially, suicidal ideation was significantly and positively correlated with log-EDA baseline levels ($r = 0.18$, $p < 0.05$), indicating that higher levels of suicidal thoughts are associated with increased autonomic arousal. Finally, age showed a significant negative correlation with log-EDA ($r = -0.23$, $p < 0.01$), confirming the necessity of including it as a covariate in the subsequent moderated mediation model.

3.2. The Moderated Mediation Model

Figure 1 illustrates the paths within the moderated mediation model.

Results indicated that depression significantly predicted suicidal ideation ($B = 0.05$, $SE = 0.006$, $p < 0.001$, 95% CI [0.03, 0.06]), which, in turn, was a significant predictor of autonomic arousal ($B = 0.17$, $SE = 0.08$, $p = 0.03$, 95% CI [0.02, 0.33]). The mediation analysis confirmed a total effect: the indirect effect of depression on autonomic arousal through suicidal ideation was significant (Bootstrapped 95% CI [0.01, 0.12]), while the direct effect was non-significant ($B = -0.002$, $SE = 0.09$, $p = 0.84$, 95% CI [−0.02, 0.01]).

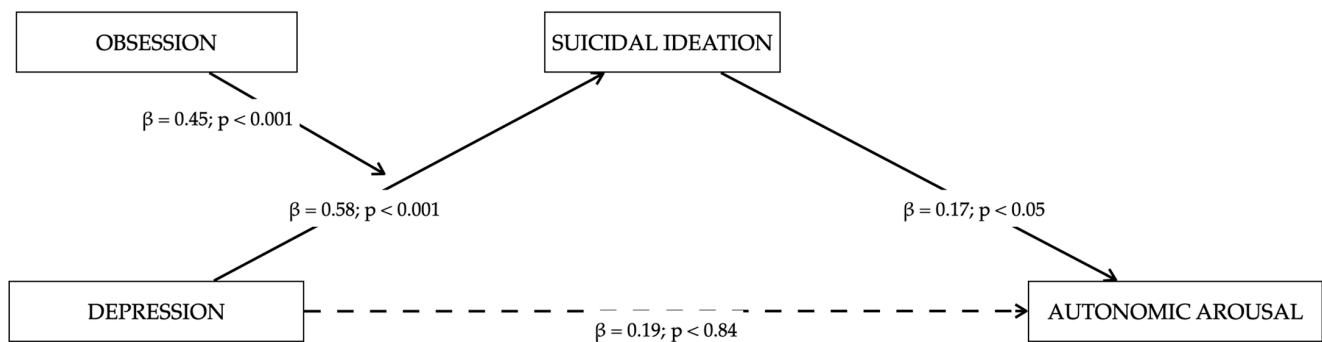


Figure 1. The mediating role of suicidal ideation in the relationship between depression and autonomic arousal and the moderating role of obsessive–compulsive symptoms between depression and suicidal ideation were described (β , p). Standardized coefficients are reported. Solid lines indicate statistically significant relationships while dashed lines indicate non statistically significant relationships.

To ensure the robustness of the identified mediation model, a formal sensitivity analysis was conducted to address potential concerns regarding item overlap between the depression subscale and the mediator (suicidal ideation). Following the removal of items 15 and 59 from the depression total score, the model was re-evaluated, maintaining the original theoretical architecture. The results of this sensitivity check demonstrated that the association between depression and suicidal ideation remained statistically significant ($B = 0.14$, $SE = 0.30$, $p < 0.01$). Furthermore, the indirect effect remained consistent with the primary findings, as evidenced by the bootstrap-based 95% confidence intervals (Boot CI $[-0.03, 0.08]$).

Furthermore, obsessive–compulsive symptoms exerted a significant moderation effect on the relationship between depression and suicidal ideation ($B = 0.20$, $SE = 0.04$, $p < 0.001$, 95% CI $[0.04, 0.37]$). The final model, controlled for age ($B = 0.08$, $p = 0.48$), explained 38.40% of the variance ($F(4, 115) = 22.17$, $p < 0.001$).

This moderation effect remained consistent in the sensitivity analysis performed without items 15 and 59, where the interaction term ($B = 0.23$, $p = 0.04$) showed a similar trend, confirming that the influence of obsessive–compulsive symptoms on the depression–suicidal ideation link is not dependent on the overlap of these specific items.

To further test the robustness of the results against the distributional characteristics of the mediator, a sensitivity analysis was conducted by re-coding the ideation score into three ordinal categories: absent ideation (0; $n = 93$), low ideation (1–4; $n = 15$), and high ideation (5–8; $n = 12$). A non-parametric bootstrapping procedure (5000 resamples) was applied within an ordinal mediation framework, yielding a significant indirect effect (Effect = 0.0038, BootSE = 0.0022, 95% CI $[0.0000, 0.0085]$).

Following the confirmation of the mediation pathway, we formally evaluated the full conditional architecture of the model. To test the conditional nature of the indirect pathway, the formal Index of Moderated Mediation and the conditional indirect effects of depression on log-EDA through suicidal ideation were computed across different levels of obsessive–compulsive symptoms (16th, 50th, and 84th percentiles) using 5000 bootstrap resamples (Table 2).

The formal Index of Moderated Mediation was 0.0000 (95% Boot CI $[-0.0001, 0.0001]$). This null result, characterized by high precision (narrow confidence interval centered on zero), provides empirical evidence against a moderated mediation effect in the current sample. The sensitivity analysis performed by excluding items 15 and 59 from the depression subscale confirmed this pattern, yielding an Index of Moderated Mediation of 0.01 (95% Boot CI $[-0.03, 0.05]$). Consistently, the conditional indirect effects of depression on

log-EDA through suicidal ideation were not significant at any level of the moderator (16th, 50th, and 84th percentiles; Table 2). These findings indicate a clear decoupling between the psychological amplification of suicidal thoughts and autonomic physiological regulation in this cohort.

Table 2. Conditional indirect effects and index of moderated mediation for the digital model.

Path/Level of Moderator (SCL-90-R Obsessive-Compulsive)	Effect	Boot SE	Boot LLCI	Boot ULCI
Conditional Indirect Effects				
Low obsessive–compulsive symptoms (16th percentile = 48.00)	0.0010	0.0017	−0.0016	0.0052
Moderate obsessive–compulsive symptoms (50th percentile = 64.66)	0.0013	0.0019	−0.0023	0.0055
High obsessive–compulsive symptoms (84th percentile = 87.20)	0.0018	0.0025	−0.0034	0.0065
Index of Moderated Mediation	0.0000	0.0000	−0.0001	0.0001

Note: Bootstrap confidence intervals are based on 5000 resamples. Dependent variable: log-EDA baseline.

4. Discussion

The present study examined the concurrent associative pathways between depression, suicidal ideation, and autonomic arousal, exploring whether obsessive–compulsive symptoms moderate the cross-sectional association between depression and suicidal ideation. The results indicated a significant indirect statistical pathway wherein self-reported suicidal ideation bridges the concurrent association between depressive symptoms and autonomic arousal, as indexed by baseline electrodermal activity. Concurrently, the data suggest that obsessive–compulsive symptoms strengthen the cross-sectional association between depression and suicidal ideation.

Due to the strong correlation between depression and obsessive–compulsive symptoms, formal multicollinearity diagnostics were routinely performed for the regression model mapping onto suicidal ideation. As derived from the OLS regression metrics, both the Variance Inflation Factor and Tolerance values were safely within conservative econometric thresholds, suggesting that severe multicollinearity was not present to critically distort the regression estimates. However, we explicitly acknowledge that a degree of conceptual and measurement overlap remains an inherent limitation, given that both clinical dimensions are derived from subscales of the same self-report instrument. In line with the Italian validation study of the SCL-90-R [39], the empirical distinction between the DEP and OC subscales was rigorously maintained. Although Prunas and colleagues utilized an oblique rotation (Oblimin) precisely to accommodate the expected clinical correlation between these dimensions, their findings confirm that obsessive and depressive symptoms aggregate into independent factor components. This supports our choice to treat them as distinct variables in the moderated-mediation model, demonstrating that the observed high correlation reflects genuine clinical comorbidity rather than psychometric redundancy or statistical artifact.

Following the confirmation of the simple statistical mediation pathway, we formally evaluated the full conditional architecture of the model to test the conditional nature of the indirect pathway. The formal Index of Moderated Mediation and the conditional indirect paths of depression on log-EDA through suicidal ideation were computed across different levels of obsessive–compulsive symptoms, probed at low, moderate, and high percentiles of the moderator using bootstrap resamples, following the macro-processes described by Hayes [38]. The results indicated that the formal Index of Moderated Mediation was not statistically significant. Consistently, the bias-corrected bootstrap confidence intervals for the conditional indirect paths encompassed zero at all levels of the moderator. Thus, while obsessive–compulsive traits significantly interact with depression at the psychological level to co-occur with higher suicidal ideation, the overall conditional indirect pathway tracking onto autonomic arousal was not statistically supported in this sample.

Crucially, the non-significant formal Index of Moderated Mediation, with confidence intervals centered precisely on zero, provides robust empirical evidence against a moderated mediation effect in this cohort. Rather than suggesting a preliminary or inconclusive finding, this result indicates a clear decoupling between the psychological amplification of suicidal thoughts and autonomic physiological regulation. While obsessive–compulsive traits significantly interact with depression at the psychological level to exacerbate suicidal ideation, this cognitive amplification does not systematically translate into a conditional indirect effect on peripheral autonomic arousal. The robust interaction observed on the mediator suggests that at a psychological level, obsessive–compulsive traits may act as a cognitive amplifier, compounding the subjective burden of depressive symptoms into self-reported thoughts of death. However, the lack of a significant conditional indirect pathway implies that this cognitive amplification is not systematically reflected in a chronic, differentiated state of sympathetic hyperarousal within this specific cohort. In a non-clinical sample of university students, tonic baseline EDA might be protected by robust homeostatic regulation and physiological resilience. Consequently, while the cognitive interaction is fully apparent, the autonomic balance may remain decoupled from stable somatic tension in healthy individuals. Alternatively, it is possible to hypothesize that the cognitive loop generated by obsessive symptoms may favor an emotional and behavioral “block” and, therefore, not translate into arousal or action. These findings underscore the necessity of treating the overall conditional architecture as strictly preliminary, highlighting that while psychological moderation occurs, its physiological translation requires larger clinical samples with established autonomic dysregulation to be fully captured.

These findings suggest that perseverative cognition—conceptually linked to ANS dysregulation—may characterize a concurrent state of vulnerability. In this regard, several studies proved that rumination is concurrently associated with the maintenance of depressive symptoms and tracks with a higher likelihood of reporting suicidal ideation [18,19,22,40]. Crucially, it must be clarified that the SCL-90-R obsessive–compulsive subscale does not directly assess perseverative cognition in its broader definitions (such as worry and rumination); rather, it should be understood strictly as a clinical proxy or a related symptomatic manifestation of rigid cognitive patterns. This maladaptive process is characterized by repetitive, egocentric, and negatively charged thoughts that maintain emotional distress and hinder adaptive self-regulation. Investigating these manifestations aligns with recent evidence highlighting how individual differences in cognitive-emotional factors, distress, and resilience shape stress-related outcomes and psychological vulnerability profiles within university student and young adult populations [41,42]. It is important to note that the present study measured perseverative cognition through the obsessive–compulsive scale of the SCL-90-R, which is most frequently associated with suicidal ideation [27] and dangerous suicide attempts [28].

From a physiological perspective, obsessive–compulsive symptoms have been associated with persistent activity of the SNS, evident in increased EDA [28]. In this context, the finding that suicidal ideation statistically bridges the relationship between depression and autonomic arousal supports the hypothesis that repetitive negative thoughts may co-occur with physiological arousal, reflecting a potential vicious cycle between mind and body [27].

This interpretation is consistent with interesting neurophysiological studies summarized in a meta-analysis conducted by Makovac et al. [43], who demonstrated that perseverative cognition, including rumination and worry, involves brain areas belonging to the Default Mode Network, such as the posterior cingulate cortex, the medial and subgenual anterior cingulate cortex, the ventromedial prefrontal cortex, the insula, and the precuneus. These regions are central to self-referential processing, emotion regulation, and autobiographical memory. In depressive states, hyperarousal of the subgenual anterior

cingulate cortex was associated with sadness, hopelessness, and suicidal ideation, whereas increased activity of the dorsal anterior cingulate and dorsolateral prefrontal areas in healthy subjects supported cognitive control and emotional flexibility. The pattern of increased sympathetic arousal observed in association with suicidal ideation in the present study may represent the physiological counterpart of this neural imbalance. In other words, excessive activity of emotional and self-referential circuits seems to be accompanied by insufficient recruitment of cognitive control networks. This dysregulation can maintain both repetitive negative thinking and physiological hyperarousal.

Psychophysiological evidence in the literature is in line with this interpretation. Stone et al. [44] showed that both dysphoric rumination and savoring, that is, the intentional re-experiencing of positive emotions, produce an increase in EDA, indicative of autonomic arousal. In their study, individuals with a higher tendency to ruminate remained physiologically activated even when they tried to focus on positive experiences. The pattern described supports the idea that perseverative cognition and thoughts of death share a physiological signature of stress persistence [27,28]. Our findings suggest that suicidal ideation may represent a condition of chronic sympathetic engagement, in which repetitive and intrusive thoughts about death are linked with difficulties returning to a state of physiological calm [27,28].

It is important to note that not all individuals who experience suicidal thoughts progress toward suicidal action. In this regard, the concurrent association between suicidal ideation and increased autonomic arousal must be interpreted with caution. Rather than signaling an inevitable progression toward suicidal behavior, increased autonomic arousal may index a state of sustained cognitive-emotional engagement that becomes difficult to downregulate, especially in the presence of intrusive and repetitive thoughts. In the present study, suicidal ideation should be understood as a marker of psychological distress rather than as a clear indicator of imminent suicidal behavior.

Concerning psychophysiological assessment, autonomic arousal was indexed using baseline EDA. Although stress reactivity and recovery phases are often informative in studies of autonomic regulation, baseline EDA was selected as a marker of tonic sympathetic activity, reflecting a sustained physiological state rather than phasic responses to specific tasks. This approach allows the identification of an underlying autonomic condition potentially associated with chronic cognitive-emotional engagement. Our results align with the broader psychophysiological literature, suggesting that cognitive and emotional states deeply influence autonomic modulation. Specifically, previous findings revealed that State Anxiety is significantly associated with psychophysiological indices, particularly vagal tone [45,46]. While these studies primarily focus on Heart Rate Variability (HRV) as a marker of parasympathetic activity, they provide crucial support for our theoretical framework, as perseverative cognitive states might be associated with the maintenance of the autonomic imbalance.

Another relevant result concerns the moderating role of obsessive-compulsive symptoms in the statistical relationship between depression and suicidal ideation. Obsessive-compulsive symptoms are characterized by intrusive, repetitive, and difficult-to-control thoughts, often accompanied by high anxiety and attentional disengagement difficulties. DeVylder et al. [47] observed that individuals at clinical high risk for psychosis with obsessive-compulsive symptoms, especially aggressive obsessions, exhibited higher levels of depression, greater suicidal ideation, and poorer social functioning. In our sample, obsessive-compulsive symptoms intensified the statistical link between depressive severity and suicidal ideation, confirming that obsessive cognitive patterns act as statistical amplifiers of negative thoughts within the initial stage of the concurrent model. This amplifier

effect proved robust to sensitivity analyses, maintaining consistent significance even when potential item-level overlap was systematically removed.

Notwithstanding, this study has some limitations. Crucially, the cross-sectional design strictly prevents any causal or directional inference, meaning that the temporal sequence required to establish definitive causal mechanisms cannot be confirmed. Furthermore, the non-clinical nature of the sample may limit generalizability. Future research must employ longitudinal and multimodal approaches integrating self-report, psychophysiological, and neuroimaging measures across multiple time points to formally map the temporal dynamics linking psychological suffering, autonomic arousal, and suicidal ideation. Additionally, the sample consisted predominantly of young adults, including non-traditional university students, which may limit the generalizability of the findings to other age groups. Moreover, all psychological variables were assessed using self-report measures; therefore, the present data refer to symptom severity rather than clinically established diagnoses.

Furthermore, the choice of the SCL-90-R, driven by the need for methodological consistency and the intent to use an economic model for rapid screening in institutional settings, was carefully scrutinized for potential psychometric overlap. Specifically, suicidal distress was mapped using Item 15 (“Thoughts of ending your life”) and Item 59 (“Thoughts of death or dying”), which are embedded within the broader checklist. Retaining these items in their standardized format was necessary to preserve the psychometric properties and internal consistency of the validated tool, allowing for the correct application of normative T-scores [39]. Crucially, our a priori formal multicollinearity diagnostics confirmed that the regression estimates remain perfectly stable, robust, and free from severe variance inflation, ensuring that the shared variance reflects clinical comorbidity rather than psychometric redundancy. To rigorously address this concern, a formal sensitivity analysis was conducted by re-evaluating the mediation and moderation models excluding items 15 and 59 from the depression score. The stability of the regression estimates and the consistency of the indirect and moderation effects across these models confirm that our findings are not driven by item-level redundancy but reflect a genuine clinical comorbidity.

Nonetheless, because suicidal ideation was assessed using these two checklist items rather than a dedicated suicide risk instrument, it is important to emphasize that the mediation model must be interpreted strictly as reflecting statistical associations involving self-reported suicidal thoughts rather than predictive indicators of actual suicidal risk. Although these items are comparable to the Suicidal Ideation subscale of the Columbia Suicidal Severity Rating Scale [33,34], this screening approach should be interpreted as reflecting concurrent distress patterns, aligning with established methodological precedents in the psychiatric epidemiological literature [27]. Lastly, we acknowledge that the distribution of the mediator showed a marked concentration towards the baseline value and was heavily bounded. While this poses challenges for traditional linear models, the choice to treat the variable as continuous in the original model was supported by the need to capture not only the presence of the phenomenon, but also the gradient of clinical severity expressed by the total score [48,49]. In line with the literature on suicidal risk in non-clinical samples, this variance reflects a continuum of vulnerability that linear models are effectively able to intercept as a general sample trend [36]. Furthermore, a linear approach allows for greater interpretability of the coefficients in terms of unit risk increment, facilitating the translation of results into preventive screening [49,50]. Regarding the mediation model, the sensitivity analysis corroborated the stability of the indirect effect across different distributional assumptions. Although the confidence interval bounds for the ordinal framework approached zero, the convergence across OLS, ordinal categorization, and bootstrapping frameworks provides consistent evidence for the identified mediation pathway. Far from being a statistical artifact, this indicates that the relationship between depressive symptoms,

suicidal ideation, and autonomic arousal represents a stable associative process in our sample. Future research with larger clinical samples may further delineate the boundaries of this physiological translation using more complex distributive models [51,52]. Future studies with larger clinical samples could further validate these findings using even more complex distributive models, such as Zero-Inflated Poisson regressions [53].

Overall, the present results support an associative, concurrent model in which depressive symptoms, perseverative cognitive styles, suicidal ideation, and tonic autonomic arousal are interrelated. Given the cross-sectional nature of the study, no causal or mechanistic conclusions can be drawn. Rather than identifying pathways leading directly to suicidal behavior, the data point to a concurrent psychophysiological vulnerability profile that may characterize susceptibility under specific contextual or individual conditions.

Despite its limitations, this study has crucial clinical implications. From a clinical point of view, our research underlines the importance of multidimensional assessments and integrated interventions that jointly address the concurrent cognitive and physiological components of distress. Techniques aimed at enhancing autonomic flexibility, such as biofeedback, within psychotherapeutic, pharmacological, or integrated approaches, may help to support the balance between sympathetic and parasympathetic activity and target maladaptive and repetitive cognitive mechanisms [23]. On top of this, screening for obsessive–compulsive symptoms in individuals with depressive disorders may help identify those exhibiting a higher tendency toward intrusive and repetitive thinking [20,21]. The integration of psychophysiological, cognitive, and neurobiological perspectives offers new insights into the patterns underlying suicide vulnerability and underscores the need for preventive and therapeutic strategies that integratively address both emotional and autonomic regulation.

5. Conclusions

This study provides new evidence regarding the complex, concurrent interplay between depressive symptoms, obsessive–compulsive traits, suicidal ideation, and autonomic arousal in a non-clinical student cohort. The robustness of these associations, confirmed through comprehensive sensitivity analyses, suggests that the identified pathways are not artifacts of psychometric overlap but represent stable psychological and physiological processes. The results suggest that suicidal ideation exhibits a significant indirect statistical association with baseline electrodermal activity, indicating that thoughts of death concurrently track with sustained somatic tension. Concurrently, obsessive–compulsive symptoms emerged as a significant statistical amplifier of the cross-sectional association between depression and suicidal ideation at the psychological level. Taken together, these findings support a multidimensional framework in which self-reported cognitive patterns and peripheral sympathetic tone jointly map onto profiles of psychological vulnerability.

Within this concurrent architecture, suicidal ideation seems to reflect not only an isolated cognitive process but also a state of persistent autonomic involvement, where repetitive and intrusive negative thoughts are linked to emotional distress and a disruption of psychophysiological balance. Crucially, while obsessive cognitive patterns intensify the internal cognitive burden of depressive states—strengthening their association with thoughts of death—this psychological moderation did not translate into a significant conditional indirect effect on peripheral autonomic pathways within the overall conditional architecture. This underscores a potential decoupling between cognitive amplification and stable somatic translation in resilient, non-clinical populations, positioning the moderated-mediation architecture as a robust framework that captures key concurrent dynamics, albeit within a non-causal cross-sectional design.

Clinically, these insights underscore the importance of integrated assessment and treatment approaches that simultaneously target the cognitive, behavioral, and physiological dimensions of distress. Interventions aimed at enhancing autonomic flexibility, such as biofeedback, may facilitate emotional regulation and help downregulate autonomic hyperarousal, offering functional alternatives to maladaptive coping strategies. Furthermore, screening for obsessive–compulsive symptom dimensions as a clinical proxy for rigid thinking in individuals presenting with depression may help clinicians identify those who display a higher concurrent vulnerability to severe ideation due to intrusive and perseverative thought patterns. Given the consistency of this moderating effect across all validation models, this clinical indicator appears particularly reliable for identifying subgroups at increased risk for cognitive amplification. However, given the non-clinical nature of the sample and the lack of explicit measures regarding suicidal intent, planning, or actual behavior, these findings must not be used for clinical prediction or to infer transitions toward suicidal action. Future research utilizing longitudinal and experimental designs across diverse clinical samples is strictly required to untangle the true causal and temporal dynamics linking these psychophysiological markers to actual suicide risk.

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Data Availability Statement: The data presented in this study are available on request from the corresponding author. The data are not publicly available due to ethical/privacy issues.

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