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ORIGINAL ARTICLE

# Linking eveningness to depression and anxiety: the mediating role of impulsivity and resilience

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ABSTRACT

Objective: This study investigated the interrelationships between chronotype, impulsivity, resilience, and affective symptomatology in individuals with Major Depressive Disorder (MDD) in remission. Specifically, it examined whether impulsivity and resilience mediate the association between eveningness and depressive and anxiety symptoms, and whether sleep quality moderates these pathways.

Materials and Methods: This cross-sectional study was conducted between February and April 2025 at Istanbul Tuzla State Hospital, Türkiye, and included 203 patients diagnosed with MDD in remission. Participants were assessed using validated psychometric instruments, including the Morningness-Eveningness Questionnaire (MEQ), Beck Depression Inventory (BDI), Beck Anxiety Inventory (BAI), Psychological Resilience Scale (PRS-33), Barratt Impulsiveness Scale (BIS-11), and the Pittsburgh Sleep Quality Index (PSQI). Analyses were conducted using SPSS 22.0, including correlations, multiple linear regression, and PROCESS-based mediation and moderation models.

Results: Eveningness was significantly associated with increased severity of both depressive and anxiety symptoms. Non-planning and attentional impulsivity partially mediated the relationship between eveningness and depressive symptoms. Resilience also partially mediated the link between eveningness and depression, indicating a protective psychological buffer. Sleep quality moderated the chronotype–depression association, such that poor sleep exacerbated depressive symptoms in evening types, but it did not moderate the chronotype–anxiety link.

Conclusion: Chronotype influences mental health outcomes through intricate cognitive-affective pathways. Evening-type individuals are more vulnerable to affective symptoms due to heightened impulsivity and reduced resilience. These findings emphasize the need for multidimensional interventions that address not only circadian misalignment but also impulsivity regulation and resilience enhancement to improve psychological outcomes in mood disorder populations.

Keywords: anxiety, chronotype, depression, impulsivity, resilience

## INTRODUCTION

Major depressive disorder (MDD) remains one of the most significant global health burdens, necessitating a comprehensive understanding of its multifaceted etiology [1]. While genetic predispositions, environmental stressors, and neurobiological mechanisms are well-established contributors emerging research underscores the critical influence of individual differences in neurobiological rhythms and psychological traits—particularly chronotype, impulsivity, and resilience—on vulnerability to MDD and overall psychological well-being [2].

Chronotype, defined as an individual's biological preference for activity and alertness in the morning or evening, has gained attention as a chronobiological factor linked to emotional and psychiatric outcomes. A growing body of research suggests that eveningness is associated with greater susceptibility to depression, anxiety, and impulsivity, likely due to circadian misalignment and sleep disruptions [3,4]. In contrast, morningness has been linked to increased psychological resilience and reduced vulnerability to mood disturbances [5,6]. Importantly, chronotype effects extend beyond this dichotomy, as circadian rhythm amplitude and stability also play a vital role in coping mechanisms, affective regulation, and mental health outcomes [7].

Psychological resilience—the capacity to adapt to and recover from adversity—acts as a protective buffer against psychiatric disorders, including depression and anxiety [8]. Resilient individuals tend to regulate emotions more effectively and cope better with stress, maintaining psychological well-being despite life challenges. Recent studies suggest that resilience may mediate the impact of chronotype on psychopathology, potentially mitigating the negative consequences of eveningness [6,9,10]

Impulsivity, characterized by difficulties in self-regulation, delayed gratification, and increased risk-taking, is another psychological trait closely associated with mood disorders. Individuals with an evening chronotype tend to exhibit higher levels of impulsivity, which can exacerbate emotional dysregulation, increase susceptibility to depression and anxiety, and promote maladaptive behaviors [4,11]. Additionally, personality traits such as neuroticism and low conscientiousness may moderate the chronotype–impulsivity–depression pathway, further emphasizing the need to consider trait-level individual differences [12].

Anxiety symptoms frequently co-occur with depressive disorders, and comorbidity rates are particularly high among individuals diagnosed with MDD. Research indicates that nearly half of patients with MDD also meet criteria for an anxiety disorder, a co-occurrence that significantly increases illness severity, functional impairment, and the risk of poor treatment outcomes [13].

Despite extensive evidence linking chronotype, impulsivity, and resilience to mental health, inconsistencies persist regarding their direct and indirect effects. Some studies challenge the assumption that eveningness is inherently maladaptive, suggesting its impact may be context-dependent and shaped by environmental, cognitive, and social factors [14]. Moreover, the stability of chronotype in clinical populations and the potential for therapeutic circadian interventions remain topics of ongoing investigation. Other contributing factors, such as cognitive rumination, emotional dysregulation [3], and genetic variations (e.g., PER3 gene [15]), may further mediate these complex interactions.

While previous research has established links between chronotype and mood disorders [3,4], the current study aims to make several unique and important contributions. Specifically, we focus on individuals with Major Depressive Disorder (MDD) in remission—a clinically relevant yet relatively understudied population in circadian rhythm research. Investigating this group is crucial, as remitted patients often continue to experience residual cognitive and emotional vulnerabilities [3], including poor sleep and diminished resilience, which can predict relapse. Studying MDD in remission therefore offers a strategic opportunity to identify underlying risk pathways that persist beyond symptom resolution. To our knowledge, this is the first study to simultaneously examine both impulsivity and resilience as key mediating

factors in the relationship between chronotype and affective symptoms (both depressive and anxiety) in a remitted MDD patient group. Most prior research has typically focused on either a single mediator [4] or broader associations between chronotype and mood symptomatology [5], or has examined bipolar populations [11]. Our integrated approach incorporates both mediation and moderation analyses, enabling a more nuanced understanding of how sleep quality interacts with chronobiological preference and cognitive-affective traits. This framework may help uncover distinct and potentially overlapping pathways linking eveningness to depressive and anxiety symptoms, thereby offering novel insights for the development of more tailored and targeted interventions for individuals with mood disorders.

## MATERIALS AND METHODS

### Study population and sample

This study was conducted on a clinical sample of patients diagnosed with Major Depressive Disorder (MDD) based on the criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5). The diagnosis of MDD was confirmed using the Structured Clinical Interview for DSM-5 Disorders (SCID-5), a gold-standard diagnostic tool for psychiatric assessment. Participants were recruited from the outpatient psychiatry clinics of Istanbul Tuzla State Hospital. A total of 203 patients who met the eligibility criteria and provided written informed consent were included in the study. Data collection was conducted between February 2025 and April 2025.

At the time of assessment, all participants were in remission, defined by the absence of active depressive symptoms based on clinical evaluation and SCID-5 criteria. However, we acknowledge that residual symptoms—particularly sleep disturbances—may persist during remission, as noted in previous research [16].

### Inclusion and exclusion criteria

To ensure a homogeneous sample, this study implemented a set of strict inclusion and exclusion criteria. Participants were eligible for inclusion if they were between the ages of 18 and 65 years, literate, and capable of independently completing self-report questionnaires. Additionally, all participants had a confirmed diagnosis of Major Depressive Disorder (MDD), which was established using the Structured Clinical Interview for DSM-5 Disorders (SCID-5). At the time of assessment, participants were required to be in remission, as determined by clinical psychiatric evaluation, ensuring the absence of active depressive symptoms. Furthermore, only individuals who were receiving routine follow-up care at the outpatient clinic were considered for inclusion. Prior to their participation, all individuals provided written informed consent after receiving comprehensive information about the study, including its objectives, procedures, and their rights as participants.

To eliminate potential confounding factors and maintain the validity of the findings, several exclusion criteria were applied. Patients who were currently experiencing an active major depressive episode at the time of assessment were excluded, as were those with a history of intellectual disability (IQ < 70), dementia, or other neurocognitive disorders that could impair their ability to comprehend or complete the study assessments. Furthermore, individuals with severe and potentially confounding psychiatric disorders were excluded. These encompassed psychotic disorders such as schizophrenia, schizoaffective disorder, and delusional disorder; bipolar and related disorders including bipolar I disorder, bipolar II disorder, and cyclothymic disorder, due to the distinct affective and circadian rhythm profiles characteristic of these conditions; and severe personality disorders—such as borderline or antisocial personality disorder—when, in the judgment of the evaluating psychiatrist, these were likely to significantly compromise the accuracy of self-report measures or introduce marked emotional instability beyond the scope of MDD.

To further minimize the influence of undiagnosed psychiatric comorbidity, all participants underwent the SCID-5, and only individuals with a primary diagnosis of MDD in remission and no comorbid psychiatric disorders at the diagnostic level were included. Consequently, no subgroup reanalysis for psychiatric comorbidity was required.

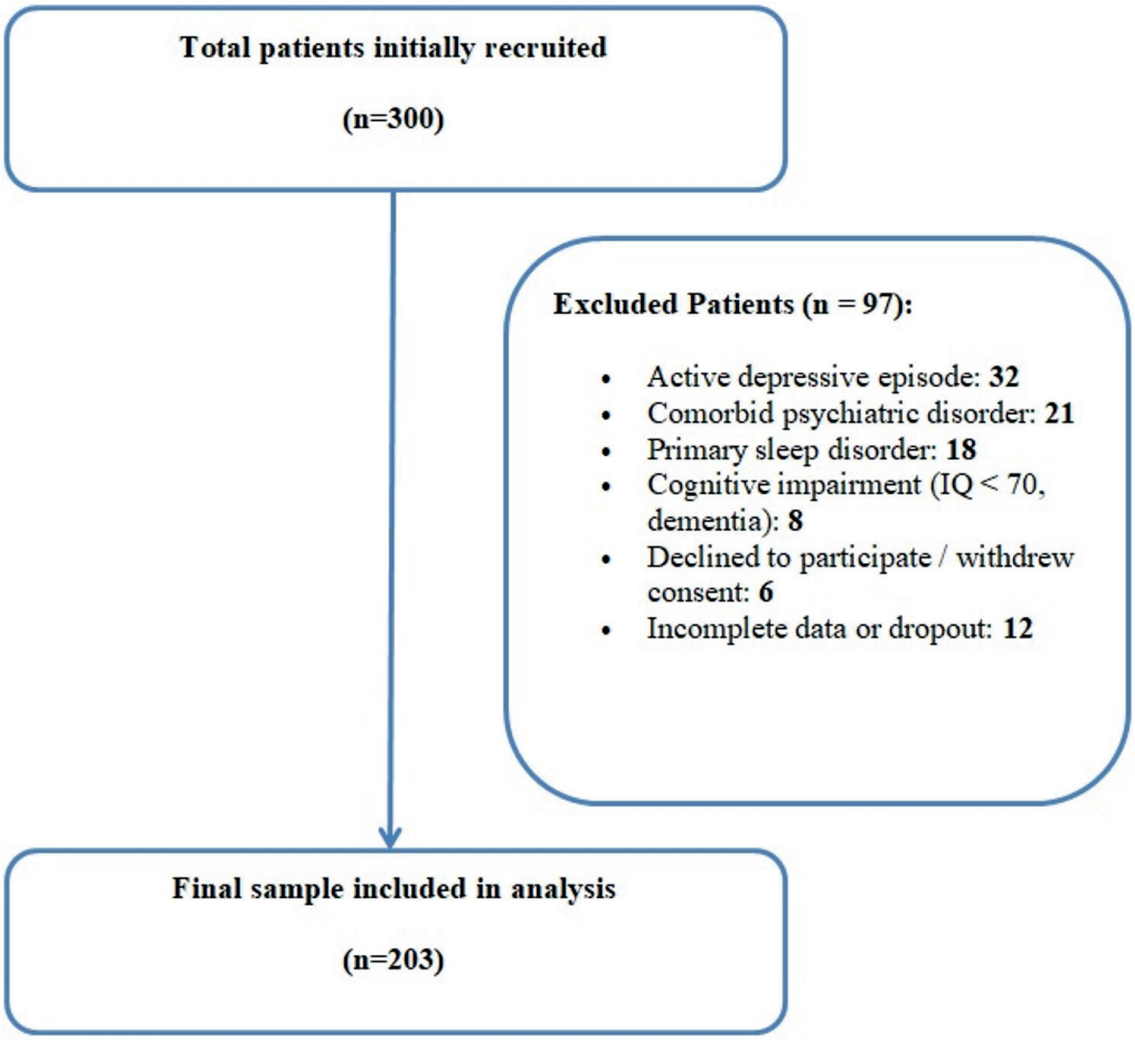


**Figure 1.** Flowchart of participant recruitment and exclusion criteria

In addition, individuals with primary sleep disorders—including insomnia, obstructive sleep apnea, or restless legs syndrome—diagnosed independently of depressive symptoms, were excluded, as these conditions could independently influence key psychological variables such as mood, sleep quality, and impulsivity. Lastly, participants who were unable or unwilling to provide informed consent were not included in the study (see Figure 1).

## Measures

A combination of structured clinical interviews and validated psychometric instruments was employed to assess the key psychological constructs relevant to the study.

Sociodemographic and clinical characteristics—including age, gender, education, marital status, illness onset, duration, psychiatric history, suicide attempts, family psychiatric history, and substance use—were collected via a structured researcher-developed questionnaire. To assess depressive and anxiety symptoms, participants completed the Beck Depression Inventory (BDI) and the Beck Anxiety Inventory (BAI), both widely validated self-report measures that assess symptom severity [17,18]. Psychological resilience was measured using the Psychological Resilience Scale for Adults (PRS-33), which evaluates resilience across six dimensions [19].

Chronotype was assessed using the Morningness-Eveningness Questionnaire (MEQ), a validated measure that classifies individuals into morning, evening, or intermediate types based on their sleep-wake preferences [20]. Sleep quality was evaluated using the Pittsburgh Sleep Quality Index (PSQI), a self-report instrument assessing subjective sleep quality over the past month [21]. To

measure impulsivity, the study utilized the Barratt Impulsiveness Scale - Short Form (BIS-11-SF), which assesses impulsivity across attentional, motor, and non-planning dimensions [22].

All psychometric instruments used in this study have been adapted into Turkish, with their validity and reliability confirmed through prior research [23-27].

### Procedure

Participants who met the eligibility criteria were invited to participate in the study. After obtaining written informed consent, they completed self-report questionnaires under the supervision of trained research staff. The assessments were conducted in a standardized and structured manner to ensure consistency and minimize potential biases.

Clinical and demographic data were gathered through structured interviews and supplemented with a review of hospital medical records. The SCID-5 interview was conducted by trained psychiatrists to confirm the diagnosis of MDD. The absence of active depressive symptoms was clinically evaluated, ensuring that all participants were in remission.

### Statistical analysis

An a priori power analysis was performed using GPower (version 3.1.9.7) to estimate the minimum sample size needed for detecting a moderate effect size ($f^2 = 0.08$) in a multiple linear regression model with six predictors, including chronotype, sleep quality, impulsivity subscales, and resilience. With an alpha level of 0.05 and desired power of 0.80, the required sample size was calculated to be 160 participants. The final sample of 203 exceeded this threshold, ensuring adequate statistical power to detect hypothesized effects.

The Kolmogorov–Smirnov test was applied to evaluate the normality of data distributions for all continuous variables. Descriptive statistics, including mean ± standard deviation (SD) for normally distributed variables and median (minimum–maximum) for non-normally distributed variables, were used to summarize continuous demographic and clinical characteristics. Categorical variables were summarized using frequencies and percentages (n, %). Pearson's correlation coefficients were calculated to assess bivariate relationships among key study variables, including chronotype, sleep quality, impulsivity, resilience, and affective symptoms. To examine the predictive power of chronotype, impulsivity, and resilience on depressive and anxiety symptoms, multiple linear regression analyses were performed. To test indirect and conditional effects, mediation and moderation analyses were conducted using the PROCESS macro for SPSS (Model 4 and Model 1, respectively; Hayes, 2018). Mediation models assessed whether impulsivity (non-planning and attentional) or resilience served as mediators in the relationship between chronotype and affective symptoms. Moderation models explored whether sleep quality (PSQI) or impulsivity dimensions moderated the effect of chronotype on depression or anxiety outcomes. All analyses were performed using IBM SPSS Statistics (Version 22.0; IBM Corp., Armonk, NY, USA). Statistical tests were two-tailed, and significance was set at $p < 0.05$.

### Ethical considerations

This study was approved by the Istanbul Medipol University Noninterventional Clinical Research Ethics Committee (Protocol No: E-10840098-202.3.02-1257; Date: 13.02.2025) and conducted in accordance with the Declaration of Helsinki (2013). Written informed consent was obtained from all participants. Confidentiality and anonymity were ensured through coded data and secure storage procedures.

## RESULTS

Table 1 provides a comprehensive overview of the sociodemographic and clinical characteristics of the patient sample, offering insights into the composition and clinical profiles of the study participants. The sample consisted of 203 patients, with a mean age of 39.01 ± 10.13 years, representing a broad adult age range. A majority of participants were female (64.5%), and more than half were unmarried (53.7%), a factor often associated with increased psychological vulnerability in clinical populations. Further details are summarized in Table 1.

**Table 1.** Sociodemographic and clinical characteristics of patients

| | n (%)<br>mean ± SD<br>median (min-max) |
|---|---|
| **Gender** | |
| Female | 131 (64.5) |
| Male | 72 (35.5) |
| **Age** | 39.01 ± 10.13 |
| **Education (Years)** | 9.97 ± 2.69 |
| **Marital Status** | |
| Single/Divorced/Widowed | 109 (53.7) |
| Married | 94 (46.3) |
| **Work Status** | |
| Employed | 99 (48.8) |
| Not Working | 51 (25.1) |
| Retired | 37 (18.2) |
| Student | 16 (7.9) |
| **Smoking Status** | |
| Smoker | 112 (55.2) |
| Non-Smoker | 91 (44.8) |
| **Age of Disease Onset** | 31(13-62) |
| **Illness Duration (Years)** | 6 (1-20) |
| **History of Suicide Attempt** | |
| Yes | 14 (6.9) |
| No | 189 (93.1) |
| Other Medical Comorbidities | |
| Yes | 118 (58.1) |
| No | 85(41.9) |
| Family History of Psychiatric Disorder | |
| Yes | 65 (32.0) |
| No | 138 (68.0) |

Values presented as mean ± SD (standard deviation), median (min-max) and n (%).

Participants had a mean MEQ (chronotype) score of 43.46 ± 11.97, indicating a tendency toward eveningness. Average scores were 7.89 ± 2.19 on the BAI and 5.13 ± 1.15 on the BDI. The total PRS-33 averaged 114.45 ± 10.56, with subscale means as follows: Self-Perception (18.93 ± 4.39), Future Orientation (13.22 ± 3.99), Structured Style (13.96 ± 2.90), Social Competence (22.15 ± 5.43), Family Cohesion (20.11 ± 4.50), and Social Resources (26.08 ± 4.74). Sleep quality, measured by the PSQI, had a mean score of 7.54 ± 2.67. Impulsivity subscales of the BIS-11 yielded mean scores of 25.85 ± 4.16 for Non-Planning, 21.70 ± 5.64 for Motor, and 23.02 ± 5.09 for Attentional impulsivity.

Chronotype (MEQ) showed significant negative correlations with anxiety (BAI), depression (BDI), and all impulsivity subscales, while positively correlating with resilience (PRS-33). Poor sleep quality (PSQI) was positively associated with depressive symptoms but unrelated to anxiety or impulsivity. Resilience was negatively related to both depression and impulsivity. For detailed correlation coefficients and significance values, see Table 2.

Prior to conducting the multiple linear regression, a series of univariate linear regression analyses were performed to assess the individual predictive capacity of each independent variable on depressive symptoms, as measured by the BDI. The results indicated that chronotype, assessed by the MEQ, significantly predicted depressive symptoms ($\beta = -0.207$, $p = 0.003$). Sleep quality, measured by the PSQI, was also a significant predictor ($\beta = 0.289$, $p < 0.001$). All three subdimensions of the BIS-11 were significantly associated with depressive symptoms: Non-Planning Impulsivity ($\beta = 0.239$, $p = 0.001$), Motor Impulsivity ($\beta = 0.144$, $p = 0.040$), and Attentional Impulsivity ($\beta = 0.165$, $p = 0.019$). In terms of resilience, the total score of the PRS-33 was negatively associated with depressive symptoms ($\beta = -0.228$, $p = 0.001$), along with the following subdimensions: Self-Perception ( $\beta = -0.176$, $p = 0.012$), Future Orientation ($\beta = -0.165$, $p = 0.019$), and Family Cohesion ($\beta = -0.144$, $p = 0.040$). Conversely, the subdimensions Structured Style ($\beta = -0.001$, $p = 0.984$), Social Competence ($\beta = -0.037$, $p = 0.599$), and Social Resources ($\beta = -0.020$, $p = 0.776$) did not significantly predict depressive symptomatology (Table 3).

A multiple linear regression analysis was conducted to examine the predictive capacity of MEQ, PSQI, BIS-11-NP, BIS-11-M, BIS-11-A, and PRS-33 on depressive symptoms, as measured by the BDI. The overall model was statistically significant, $F(6, 196) = 8.544$, $p < 0.001$, explaining 20.7% of the variance in BDI scores ($R^2 = 0.207$, Adjusted $R^2 = 0.183$). Among the predictors, PSQI ($\beta = 0.322$, $p < 0.001$), BIS-11-NP ($\beta = 0.208$, $p = 0.003$), and PRS-33 ($\beta = -0.148$, $p = 0.031$) were significant predictors of depressive symptoms. In contrast, MEQ ($\beta = -0.009$, $p = 0.904$), BIS-11-M ($\beta = 0.051$, $p = 0.441$), and BIS-11-A ($\beta = 0.133$, $p = 0.046$) did not significantly contribute to the model (Table 3).

**Table 2.** Correlation matrix of chronotype, impulsivity, resilience, and psychological symptoms

| | | MEQ | BAI | BDI | BIS-11-NP | BIS-11-M | BIS-11-A | PRS-33 |
|---|---|---|---|---|---|---|---|---|
| MEQ | r | 1.000 | -0.241 | -0.207 | -0.259 | -0.147 | -0.147 | 0.311 |
| | p | - | 0.001** | 0.003** | <0.001** | 0.037* | 0.037* | <0.001** |
| PSQI | r | -0.224 | 0.039 | 0.289 | -0.117 | 0.059 | -.0104 | -0.004 |
| | p | 0.001 | 0.585 | <0.001 | 0.098 | 0.407 | 0.138 | 0.953 |
| BAI | r | -0.241 | 1.000 | 0.079 | 0.024 | -0.094 | 0.230 | -0.052 |
| | p | 0.001** | | 0.264 | 0.737 | 0.183 | 0.001** | 0.459 |
| BDI | r | -0.207 | 0.079 | 1.000 | 0.239 | 0.144 | 0.165 | -0.226 |
| | p | 0.003** | 0.264 | | 0.001** | 0.040* | 0.019* | 0.001** |
| BIS-11-NP | r | -0.259 | 0.024 | 0.239 | 1.000 | 0.171 | 0.164 | -0.244 |
| | p | <0.001** | 0.737 | 0.001** | - | 0.015* | 0.020* | <0.001** |
| BIS-11-M | r | -0.147 | -0.094 | 0.144 | 0.171 | 1.000 | 0.185 | -0.089 |
| | p | 0.037* | 0.183 | 0.040* | 0.015* | | 0.008** | 0.207 |
| BIS-11-A | r | -0.147 | 0.230 | 0.165 | 0.164 | 0.185 | 1.000 | -0.138 |
| | p | 0.037* | 0.001** | 0.019* | 0.020* | 0.008** | - | 0.049* |
| PRS-33 | r | 0.311 | -0.052 | -0.226 | -0.244 | -0.089 | -0.138 | 1.000 |
| | p | <0.001** | 0.459 | 0.001** | <0.001** | 0.207 | 0.050* | |

MEQ: Morningness-Eveningness Questionnaire, PSQI: Pittsburgh Sleep Quality Index, BAI: Beck Anxiety Inventory, BDI: Beck Depression Inventory, BIS-11-NP: Barratt Impulsiveness Scale-11 - Non-Planning Impulsivity, BIS-11-M: Barratt Impulsiveness Scale-11 - Motor Impulsivity, BIS-11-A: Barratt Impulsiveness Scale-11 - Attentional Impulsivity, PRS-33: Psychological Resilience Scale for Adults, r indicates Pearson correlation coefficient, ** p< 0.01, *p<0.05.

**Table 3.** Univariate and multivariable regression analyses of factors associated with depressive symptoms among patients

| | Unstandardized Coefficients | | Standardized Coefficients | CI | | t | p |
|---|---|---|---|---|---|---|---|
| | B | Std. Error | β | Lower | Upper | | |
| Univariate Analysis | | | | | | | |
| MEQ | -0.020 | 0.007 | -0.207 | -0.033 | -0.007 | -3.005 | 0.003 |
| PSQI | 0.125 | 0.029 | 0.289 | 0.067 | 0.182 | 4.288 | <0.001 |
| BIS-11-NP | 0.066 | 0.019 | 0.239 | 0.029 | 0.103 | 3.491 | 0.001 |
| BIS-11-M | 0.029 | 0.014 | 0.144 | 0.001 | 0.057 | 2.067 | 0.040 |
| BIS-11-A | 0.037 | 0.016 | 0.165 | 0.006 | 0.068 | 2.366 | 0.019 |
| PRS-33 | -0.025 | 0.007 | -0.226 | -0.039 | -0.010 | -3.282 | 0.001 |
| PRS-33-PS | -0.046 | 0.018 | -0.176 | -0.082 | -0.010 | -2.531 | 0.012 |
| PRS-33-FO | -0.048 | 0.020 | -0.165 | -0.087 | -0.008 | -2.374 | .0019 |
| PRS-33-SS | -0.001 | 0.028 | -0.001 | -0.056 | 0.055 | -0.021 | 0.984 |
| PRS-33-SC | -0.008 | 0.015 | -0.037 | -0.037 | 0.022 | -0.527 | 0.599 |
| PRS-33-FC | -0.037 | 0.018 | -0.144 | -0.072 | -0.002 | -2.070 | 0.040 |
| PRS-33-SR | -0.005 | 0.017 | -0.020 | -0.039 | 0.029 | -0.284 | 0.776 |
| Multivariate Analysis | | | | | | | |
| MEQ | -0.001 | 0.007 | -0.009 | -0.014 | 0.013 | -0.120 | 0.904 |
| PSQI | 0.139 | 0.029 | 0.322 | 0.082 | 0.196 | 4.8 | <0.001 |
| BIS-11-NP | 0.057 | 0.019 | 0.208 | 0.020 | 0.095 | 3.019 | 0.003 |
| BIS-11-M | 0.010 | 0.013 | 0.051 | -0.016 | 0.037 | 0.773 | 0.441 |
| BIS-11-A | 0.030 | 0.015 | 0.133 | 0.001 | 0.060 | 2.008 | 0.046 |
| PRS-33 | -0.016 | 0.007 | -0.148 | -0.031 | -0.001 | -2.167 | 0.031 |

MEQ = Morningness-Eveningness Questionnaire; PSQI = Pittsburgh Sleep Quality Index; BIS-11-NP = Barratt Impulsiveness Scale-11 – Non-Planning Impulsivity; BIS-11-M = Barratt Impulsiveness Scale-11 – Motor Impulsivity; BIS-11-A = Barratt Impulsiveness Scale-11 – Attentional Impulsivity; PRS-33 = Psychological Resilience Scale for Adults – Total Score; PRS-33-PS = Self-Perception subscale; PRS-33-FO = Future Orientation subscale; PRS-33-SS = Structured Style subscale; PRS-33-SC = Social Competence subscale; PRS-33-FC = Family Cohesion subscale; PRS-33-SR = Social Resources subscale. Unstandardized coefficients (B), standard errors, standardized coefficients (β), confidence intervals (CI: lower and upper bounds), t-values, and p-values are reported for each predictor.

**Table 4.** Univariate and multivariable regression analyses of factors associated with anxiety symptoms among patients

| | Unstandardized Coefficients | | Standardized Coefficients | CI | | t | p |
|---|---|---|---|---|---|---|---|
| | B | Std. Error | β | Lower | Upper | | |
| Univariate Analysis | | | | | | | |
| MEQ | -0.044 | 0.013 | -0.241 | -0.069 | -0.019 | -3.524 | 0.001 |
| PSQI | 0.032 | 0.058 | 0.039 | -0.082 | 0.146 | 0.546 | 0.585 |
| PRS-33 | -0.011 | 0.015 | -0.052 | -0.040 | 0.018 | -0.742 | 0.459 |
| PRS-33-PS | 0.027 | 0.035 | 0.054 | -0.042 | 0.096 | 0.764 | 0.446 |
| PRS-33-FO | 0.018 | 0.039 | 0.032 | -0.058 | 0.094 | 0.460 | 0.646 |
| PRS-33-SS | -0.055 | 0.053 | -0.073 | -0.160 | 0.049 | -1.042 | 0.299 |
| PRS-33-SC | -0.024 | 0.028 | -0.059 | -0.080 | 0.032 | -0.832 | 0.406 |
| PRS-33-FC | -0.043 | 0.034 | -0.089 | -0.111 | 0.024 | -1.264 | 0.208 |
| PRS-33-SR | 0.001 | 0.033 | 0.003 | -0.063 | 0.066 | 0.039 | 0.969 |
| BIS-11-NP | 0.012 | 0.037 | 0.024 | -0.061 | 0.086 | 0.336 | 0.737 |
| BIS-11-M | -0.036 | 0.027 | -0.094 | -0.090 | 0.017 | -0.337 | 0.183 |
| BIS-11-A | 0.099 | 0.029 | 0.230 | 0.041 | 0.157 | 3.351 | 0.001 |
| Multivariate analysis | | | | | | | |
| MEQ | -0.039 | 0.012 | -0.212 | -0.063 | -0.014 | -3.122 | 0.002 |
| BIS-11-A | 0.085 | 0.029 | 0.199 | 0.028 | 0.143 | 2.929 | 0.004 |

MEQ = Morningness-Eveningness Questionnaire; PSQI = Pittsburgh Sleep Quality Index; BIS-11-NP = Barratt Impulsiveness Scale-11 – Non-Planning Impulsivity; BIS-11-M = Barratt Impulsiveness Scale-11 – Motor Impulsivity; BIS-11-A = Barratt Impulsiveness Scale-11 – Attentional Impulsivity; PRS-33 = Psychological Resilience Scale for Adults – Total Score; PRS-33-PS = Self-Perception subscale; PRS-33-FO = Future Orientation subscale; PRS-33-SS = Structured Style subscale; PRS-33-SC = Social Competence subscale; PRS-33-FC = Family Cohesion subscale; PRS-33-SR = Social Resources subscale. Unstandardized coefficients (B), standard errors, standardized coefficients (β), confidence intervals (CI: lower and upper bounds), t-values, and p-values are reported for each predictor.

Prior to conducting the multivariable analysis, a series of univariate linear regression analyses were performed to evaluate the individual predictive capacity of each variable on anxiety symptoms, as measured by the BAI. The results indicated that MEQ (β = –0.241, $p = 0.001$) and BIS-11-A (β = 0.230, $p = 0.001$) were significant predictors of anxiety symptoms. In contrast, PSQI (β = 0.039, $p = 0.585$), PRS-33 (β = –0.052, $p = 0.459$), PRS-33-PS (β = 0.054, $p = 0.446$), PRS-33-FO (β = 0.032, $p = 0.646$), PRS-33-SS (β = –0.073, $p = 0.299$), PRS-33-SC (β = –0.059, $p = 0.406$), PRS-33-FC (β = –0.089, $p = 0.208$), PRS-33-SR (β = 0.003, $p = 0.969$), BIS-11-NP (β = 0.024, $p = 0.737$), and BIS-11-M (β = –0.094, $p = 0.183$) did not significantly predict anxiety symptoms (Table 4).

A multiple linear regression analysis was then conducted to examine the combined predictive capacity of MEQ and BIS-11-A on anxiety symptoms. The overall model was statistically significant, $F(2, 200) = 10.733$, $p < 0.001$, explaining 9.7% of the variance in BAI scores ($R^2 = 0.097$, Adjusted $R^2 = 0.088$). Both MEQ (β = –0.212, $p = 0.002$) and BIS-11-A (β = 0.199, $p = 0.004$) remained significant predictors in the multivariable model, highlighting their unique and additive contributions to anxiety symptomatology (Table 4).

The moderation analysis demonstrated that the interaction between chronotype (MEQ) and sleep quality (PSQI) significantly predicted depressive symptoms (BDI) (β = –0.006, $p = 0.025$), suggesting that the impact of chronotype on depression is contingent upon sleep quality. The overall model was statistically significant, $F(3, 199) = 9.706$, $p < 0.001$, explaining 12.8% of the variance in depressive symptoms ($R^2 = 0.128$). Probing the interaction revealed that MEQ scores were not significantly associated with BDI scores at low (–1 SD) levels of sleep disturbance (β = 0.005, $p = 0.639$) or at average levels (β = –0.010, $p = 0.146$). However, at high levels of sleep disturbance (+1 SD), lower MEQ scores (i.e., eveningness) were significantly associated with increased depressive symptoms (β = –0.025, $p = 0.002$).

In contrast, sleep quality did not significantly moderate the relationship between chronotype and anxiety symptoms (BAI), as the interaction term (MEQ × PSQI) was non-significant (β = 0.003, $p = 0.610$).

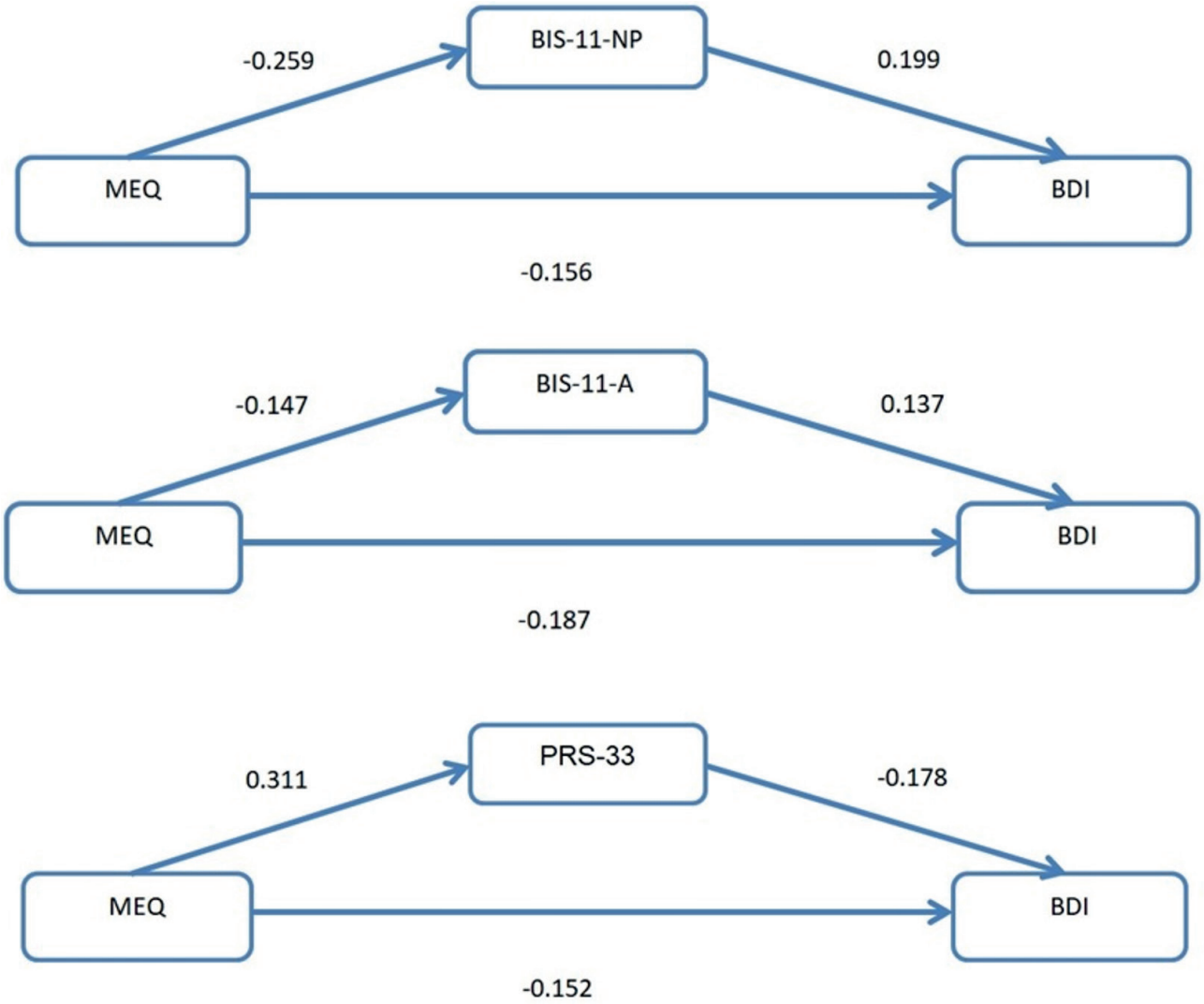


**Figure 2.** Structural model illustrating the hypothesized pathways linking chronotype (MEQ) to depressive symptoms (BDI), with psychological resilience (PRS-33) and impulsivity—non-planning (BIS-11-NP) and attentional (BIS-11-A)—as mediators. Values represent standardized coefficients.

The mediation analysis revealed that non-planning impulsivity (BIS-11-NP) partially mediated the relationship between chronotype (MEQ) and depressive symptoms (BDI). Lower MEQ scores (indicating eveningness) were significantly associated with higher levels of BIS-11-NP (β = –0.090, $p < 0.001$), which in turn predicted greater BDI scores (β = 0.055, $p = 0.005$). The indirect effect of MEQ on BDI through BIS-11-NP was statistically significant (β = –0.005, 95% CI [–0.011, –0.001]), confirming a mediation effect. Although the direct path from MEQ to BDI remained significant (β = –0.015, $p = 0.027$), the presence of this partial mediation suggests that non-planning impulsivity is a key psychological mechanism linking circadian misalignment to depressive symptomatology (Figure 2, Supplementary Table 1).

The mediation analysis revealed that attentional impulsivity (BIS-11-A) partially mediated the relationship between chronotype (MEQ) and depressive symptoms (BDI). Lower MEQ scores (indicating eveningness) were significantly associated with higher BIS-11-A scores (β = –0.062, $p = 0.037$), which in turn predicted increased BDI scores (β = 0.031, $p = 0.049$). The indirect effect of MEQ on BDI through BIS-11-A was marginally significant (β = –0.002, 95% CI [–0.005, -0.001]), suggesting a subtle mediation effect. Despite this, the direct path from MEQ to BDI remained significant (β = –0.018, $p = 0.007$), indicating that attentional impulsivity contributes to, but does not fully explain, the association between eveningness and Depression (Figure 2, Supplementary Table 2).

The mediation analysis demonstrated that psychological resilience (PRS-33) partially mediated the relationship between chronotype (MEQ) and depressive symptoms (BDI). Chronotype significantly predicted resilience (β = 0.274, p < .001), with morningness associated with higher resilience levels. In turn, resilience significantly predicted depressive symptoms (β = -0.019, p = .014), indicating that higher resilience was linked to lower depression severity. The indirect effect of chronotype on depressive symptoms through resilience was significant (β = -0.005, 95% CI [-0.011, -0.001]), suggesting that part of the protective effect of morningness on depression operates through enhanced resilience. However, the direct effect of chronotype on depressive symptoms remained significant (β = -0.015, p = 0.035), indicating that while resilience explains part of this relationship, other mechanisms may also contribute (Figure 2, Supplementary Table 3).

The mediation analysis demonstrated that attentional impulsivity (BIS-11-A) partially mediated the relationship between chronotype (MEQ) and anxiety symptoms (BAI). Chronotype significantly predicted BIS-11-A scores (β = –0.062, *p* = .037), with eveningness associated with higher levels of attentional impulsivity. In turn, greater BIS-11-A scores significantly predicted elevated BAI scores (β = 0.085, *p* = .004), indicating that individuals with higher impulsivity experienced more severe anxiety symptoms.

The indirect effect of chronotype on anxiety symptoms through attentional impulsivity was significant (β = -0.005, 95% CI [-0.014, -0.001]), suggesting that attentional impulsivity plays a role in the pathway linking chronotype to anxiety. However, the direct effect of chronotype on anxiety remained significant (β = -0.039, p = .002), indicating that while impulsivity explains part of this relationship, other mechanisms may also contribute (Figure 3, Supplementary Table 4).

## DISCUSSION

The present study aimed to investigate the complex relationships between chronotype, impulsivity, resilience, depression, anxiety, and psychological well-being. Our findings suggest that chronotype—particularly eveningness—is linked to increased depressive symptoms and anxiety. This relationship appears to be mediated by impulsivity and moderated by both sleep quality and resilience. These results align with existing literature emphasizing the adverse psychological consequences of evening chronotype and underscore the importance of individual differences in circadian preference as a determinant of mental health outcomes.

Our findings reinforce the growing consensus that evening chronotype is significantly associated with elevated depressive symptoms, consistent with previous literature [3,4]. This association may be driven by circadian misalignment, which disrupts synchronization between endogenous rhythms and external social demands, compromising emotional regulation [28]. The circadian desynchrony hypothesis suggests that such misalignment may compromise neurobiological systems involved in affective regulation, thus increasing susceptibility to depressive symptoms.

In contrast to earlier studies that broadly link eveningness to depression and anxiety [3,4], our findings delineate specific cognitive-affective mechanisms—namely non-planning and attentional impulsivity, as well as psychological resilience—that mediate this association. Importantly, while much of the existing literature

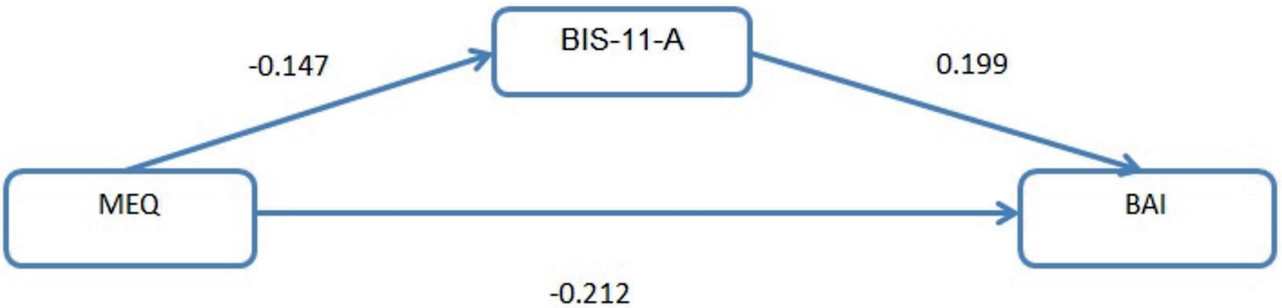


**Figure 3.** Structural model illustrating the hypothesized pathway linking chronotype (MEQ) to anxiety symptoms (BAI), with attentional impulsivity (BIS-11-A) as a mediator. Values represent standardized coefficients.

has focused on non-clinical populations or individuals with active depressive symptoms, our study uniquely examines individuals with MDD in remission, a group in whom underlying cognitive and emotional vulnerabilities may persist despite clinical improvement. For example, Tafoya et al. and Chung et al. investigated resilience in relation to chronotype in student or mixed samples, but did not assess impulsivity or employ mediation modeling [5,6]. Similarly, Hasler et al. linked eveningness to impulsivity traits but did not evaluate mood outcomes in a clinical context [4]. Our study builds on and integrates these findings, identifying distinct psychological pathways through which eveningness confers ongoing affective risk—even in the absence of active depressive episodes.

Importantly, our results extend this framework by showing that impulsivity—particularly non-planning and attentional dimensions—partially mediates this association. Individuals with an evening chronotype exhibited greater difficulties in future-oriented thinking and attentional control, both of which were linked to more severe depressive symptoms. These findings align with Hasler et al. and Gorgol et al., who argue that impulsivity may act as a key cognitive-affective pathway through which circadian preference influences mood [4,12]. In our study, non-planning impulsivity emerged as a robust mediator, suggesting that limited capacity for prospective thinking and goal-setting may exacerbate depressive risk in evening-type individuals.

Furthermore, we found that sleep quality moderated the link between chronotype and depression, such that the association was stronger among individuals with poor sleep. This interaction highlights the amplifying effect of sleep disturbances on the maladaptive impact of eveningness, reinforcing prior research that poor sleep may synergistically interact with biological predispositions to undermine psychological health [11]. Notably, eveningness was not associated with depression in individuals reporting good sleep, indicating a potential protective effect of sleep quality.

Although all participants were considered in symptomatic remission based on SCID-5 and clinical evaluation, PSQI scores varied considerably, indicating a range of subjective sleep quality. This finding aligns with prior research suggesting that sleep disturbances often persist even in remitted individuals and may continue to affect psychological well-being [16]. In our study, poor sleep quality remained a significant predictor of depressive symptoms, reinforcing the need to assess and address sleep problems even after mood symptoms have resolved. These results highlight that residual sleep disturbances may represent an ongoing vulnerability factor, with implications for relapse risk and functional recovery.

Taken together, these findings suggest that targeting impulsivity and sleep hygiene, rather than attempting to shift chronotype directly, may be a more feasible and effective approach in mitigating depressive risk. Interventions focusing on cognitive control (e.g., executive function training) and structured behavioral routines could support more adaptive functioning within an individual's chronotype constraints. This resonates with the growing movement toward personalized chronotherapeutic approaches, which aim to align therapeutic strategies with circadian and cognitive-affective profiles.

Our study found that attentional impulsivity significantly mediated the relationship between eveningness and anxiety symptoms, underscoring impulsivity as a key cognitive-affective pathway in the circadian–anxiety link (Supplementary Table 4). Specifically, individuals with an evening chronotype reported elevated levels of attentional impulsivity, which in turn predicted greater anxiety severity. This finding is consistent with prior research suggesting that cognitive dysregulation—particularly poor inhibitory control and attentional instability—may heighten emotional reactivity in evening types [4,11].

Interestingly, while a significant direct association between eveningness and anxiety was observed, sleep quality did not moderate this relationship. This contrasts with our findings in the depression model and suggests that anxiety symptoms in evening chronotypes may emerge independently of sleep disturbance, potentially through alternative pathways such as trait-level impulsivity, emotional dysregulation, or heightened cognitive reactivity. This further supports the notion that additional unmeasured moderators or neurobiological factors may be involved.

These results reinforce the idea that circadian typology not only affects physiological rhythms but also shapes the temporal structure of affective and cognitive processes. Evening-type individuals may exhibit heightened vulnerability to anxiety due to greater sensitivity to internal and external stressors, mediated by impulsive responding and reduced attentional control.

Our findings also build upon the work of Weiss et al., who demonstrated that sleep quality and genetic polymorphisms (e.g., PER3) mediate the chronotype–depression relationship, but not necessarily the chronotype–anxiety link [15]. In line with this, we found that while disrupted sleep may contribute to elevated anxiety, it does not appear to function as a moderator in this pathway. This divergence implies that depression may be more tightly coupled with physiological circadian disruptions, whereas anxiety may be more influenced by cognitive vulnerabilities associated with eveningness.

Collectively, these findings position attentional impulsivity as a promising intervention target for evening-type individuals at elevated risk for anxiety. Therapeutic strategies that enhance attentional control, emotional regulation, and executive function may reduce affective risk, especially in populations with pronounced eveningness and cognitive instability.

Our findings demonstrate that psychological resilience (PRS-33) partially mediates the relationship between chronotype and depressive symptoms, with morning-type individuals exhibiting significantly higher resilience, which in turn predicted lower depression severity (Supplementary Table 3). This supports the conceptualization of resilience as a critical psychological buffer—a protective factor that attenuates the maladaptive emotional consequences of eveningness. Resilient individuals may possess greater emotional regulation, stress tolerance, and adaptive coping capacities, making them less susceptible to mood disturbances, particularly within the context of circadian misalignment.

Importantly, while resilience significantly mediated the chronotype–depression relationship, it did not significantly predict anxiety symptoms. This divergence suggests that internal protective mechanisms such as resilience may differentially influence mood and anxiety domains, pointing to potentially distinct pathways of emotional regulation and vulnerability. Future research is warranted to explore these domain-specific resilience effects, including the possibility that trait-based resilience may be more tightly coupled to affective regulation in depression than in anxiety.

Our results are consistent with prior studies emphasizing the mediating role of resilience between circadian typology and mental health outcomes. Tafoya et al. and Chung et al. similarly found that morningness was associated with greater resilience, which helped mitigate depressive symptoms, particularly in clinical and student populations with disrupted sleep patterns [5,6]. Moreover, Jeon et al. observed that resilience buffered the adverse effects of circadian rhythm disturbances on subjective well-being [10], while Di Milia and Folkard highlighted the importance of circadian amplitude and stability in promoting coping and emotional endurance [7].

Taken together, these findings support the notion that resilience is a dynamic system—one that offers protection against both internal vulnerabilities (e.g., impulsivity) and external challenges (e.g., poor sleep quality). This reinforces the value of interventions aimed at enhancing resilience, particularly in evening-type individuals, who may be more susceptible to circadian misalignment and related affective disturbances.

From a translational perspective, stabilizing daily routines and promoting circadian regularity may simultaneously strengthen both biological and psychological resilience. Such synergy could enhance emotional stability, reduce depressive vulnerability, and contribute to long-term mental health improvement in chronotype-sensitive populations.

The present study contributes to a growing body of research linking eveningness with heightened psychological vulnerability, primarily through the mediating effects of impulsivity and reduced resilience. Evening-type individuals were more likely to exhibit elevated levels of both non-planning and attentional impulsivity, which in turn predicted increased symptoms of depression and anxiety. These findings support earlier assertions that impulsivity and circadian disruption frequently

co-occur, interacting to compound affective risk [11,12].

Moreover, the results revealed a nuanced divergence in how sleep quality interacts with affective outcomes: while poor sleep quality amplified depressive symptoms in evening chronotypes, it did not significantly influence anxiety symptoms. This pattern suggests that distinct pathways underlie the relationships between chronotype, sleep, and various mood domains. Specifically, attentional impulsivity emerged as a critical pathway linking circadian misalignment to depressive symptoms (Supplementary Table 2), highlighting the need to further investigate additional mediators such as cognitive biases, emotion regulation strategies, and neurobiological vulnerabilities.

Our findings reinforce the idea that chronotype exerts both direct and indirect effects on mood, with its impact mediated through cognitive-affective mechanisms, including impulsivity and resilience. In particular, eveningness may represent a broader psychobiological disposition characterized by reduced emotional regulation, diminished future orientation, and disrupted behavioral rhythms. From a real-world perspective, these insights can inform practical screening and intervention strategies in outpatient psychiatry and primary care settings. For instance, brief self-report measures of chronotype, impulsivity, and sleep quality can be incorporated into routine follow-ups for patients with remitted depression. This could allow clinicians to proactively identify residual cognitive-affective vulnerabilities and intervene before full relapse occurs.

Additionally, this study supports a multifactorial model of psychological well-being, one that integrates chronobiological factors, cognitive control, and trait-level psychological resilience. This aligns with findings by Kim et al., who demonstrated that eveningness, in combination with poor sleep quality, was associated with lower resilience and elevated depressive symptoms [9]. These associations underscore that chronotype is not merely a behavioral preference, but a dynamic factor shaping cognitive, emotional, and physiological processes. Implementing structured daily routines, psychoeducation on chronobiological rhythms, and targeted cognitive interventions may be feasible in outpatient practice and community mental health programs. These strategies are particularly relevant for working adults or students with evening chronotypes, whose schedules often clash with social norms, increasing stress and vulnerability.

A more comprehensive perspective on chronotype must therefore move beyond simple classifications of morningness or eveningness, and instead consider the regularity, flexibility, and stability of circadian rhythms. Such an approach allows for a deeper understanding of how biological timing systems interact with psychological traits to affect mental health —and how they can be modified in real-world therapeutic contexts.

Collectively, these findings call for multidimensional treatment approaches that target biological rhythms, impulsivity regulation, and resilience enhancement. Integrating cognitive-behavioral, chronotherapeutic, and lifestyle-based interventions may offer more effective strategies for improving mental health outcomes in individuals with evening chronotypes and affective vulnerabilities.

## Clinical implications

A key strength of this study lies in the selection of a remitted MDD population. While many studies examine active depressive states, our focus on remission provides insights into underlying trait vulnerabilities—such as impulsivity and circadian misalignment—that may remain unaddressed despite clinical improvement. This has practical implications: identifying at-risk individuals during remission allows for preventive, resilience-building, and chronotherapeutic interventions aimed at reducing long-term recurrence risk.

The current findings offer meaningful implications for clinical practice, particularly in the personalized treatment of mood disorders. The demonstrated links between evening chronotype, impulsivity, and resilience highlight key intervention targets that extend beyond traditional symptom-focused approaches.

First, enhancing psychological resilience in evening-type individuals may serve as a protective buffer against depressive symptomatology. Interventions designed to cultivate resilience—such as stress management training, mindfulness-based therapy, and emotion regulation strategies—could

empower individuals to better cope with the psychological challenges associated with circadian misalignment.

Second, the role of impulsivity as a mediator of both depression and anxiety underscores the value of cognitive-behavioral interventions aimed at improving emotional foresight, inhibitory control, and decision-making. Targeting non-planning and attentional impulsivity may reduce affective vulnerability by strengthening executive function and reducing maladaptive reactivity.

Third, the findings support the implementation of chronotherapeutic strategies that promote circadian alignment. Interventions such as sleep hygiene education, light therapy, social rhythm therapy, and structured daily routines may help optimize functioning within an individual's natural circadian profile. These strategies may be particularly effective for evening-type individuals, who often experience greater circadian misalignment and mood instability.

Taken together, these results advocate for multimodal treatment approaches that simultaneously address biological rhythm regulation, cognitive-affective vulnerabilities, and protective psychological traits. By integrating chronotype-informed care with evidence-based psychological techniques, clinicians may improve emotional outcomes and reduce relapse risk in individuals with mood disorders.

### Limitations and future directions

While this study provides important insights into the interplay between chronotype, impulsivity, resilience, and mood outcomes, several limitations warrant consideration.

First, the cross-sectional design precludes any inference of causal relationships among the observed variables. Although theoretical and empirical evidence supports directional pathways, longitudinal and prospective studies are essential to clarify the temporal dynamics and potential bidirectional influences between chronotype, impulsivity, and mood symptomatology.

Second, the sample consisted exclusively of individuals with Major Depressive Disorder (MDD) in remission, which may limit the generalizability of the findings to patients experiencing acute depressive episodes. Future studies should include diverse clinical populations, including those with active symptoms or comorbid disorders, to evaluate whether these associations remain robust under different affective states.

Third, although the study examined key mediators and moderators such as impulsivity, sleep quality, and resilience, other cognitive-affective factors—notably rumination, emotional dysregulation, and cognitive reactivity—were not directly assessed. These constructs have been previously implicated in the chronotype–mood relationship and should be systematically incorporated into future models [3].

Lastly, the reliance on self-report measures may introduce bias related to subjective reporting, particularly for constructs like sleep quality and impulsivity. Integrating objective measures and multi-informant approaches could provide a more comprehensive understanding of the mechanisms underlying circadian and affective vulnerability.

Future investigations should prioritize integrated, multidimensional frameworks encompassing biological, cognitive, affective, and behavioral domains to elucidate chronotype–mental health associations. Such work will be instrumental in advancing personalized chronotherapeutic and cognitive interventions for individuals at elevated risk of mood dysregulation.

## CONCLUSION

This study underscores the complex interplay between chronotype, impulsivity, resilience, and affective symptomatology in individuals with Major Depressive Disorder. Eveningness was associated with increased depressive and anxiety symptoms, a relationship partially mediated by elevated impulsivity—particularly in the non-planning and attentional domains. Conversely, resilience emerged as a protective factor, mitigating the adverse effects of eveningness on depressive symptoms.

These findings highlight the need for targeted, multidimensional interventions that extend beyond circadian realignment alone. Clinical strategies should focus on enhancing self-regulation capacities, promoting psychological resilience,

and accommodating individual chronobiological profiles to optimize mental health outcomes. Future research integrating biological, cognitive, and behavioral frameworks will be critical to advancing personalized care for mood disorders.

## Author contribution

Study conception and design: İÖÜ, MPA, DAA and TDB; data collection: İÖÜ; analysis and interpretation of results: İÖÜ, MPA, DAA and TDB; draft manuscript preparation: İÖÜ and MPA. All authors reviewed the results and approved the final version of the manuscript.

## Ethical approval

The study was approved by the Istanbul Medipol University Noninterventional Clinical Research Ethics Committee (Protocol no. E-10840098-202.3.02-1257/13.02.2025).

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The authors declare that the study received no funding.

## Conflict of interest

The authors declare that there is no conflict of interest.

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**Supplementary Table 1.** Mediation Analysis of Non-Planning Impulsivity (BIS-11-NP) in the Relationship Between Chronotype (MEQ) and Depressive Symptoms (BDI)

| Components | Effect | Standardized Error | Standardized Coefficient | t | Lower | Upper | p |
|---|---|---|---|---|---|---|---|
| MEQ-> BIS-11-NP | -0.090 | 0.024 | -0.259 | -3.797 | -0.137 | -0.043 | <0.001 |
| BIS-11-NP->BDI | 0.055 | 0.019 | 0.199 | 2.831 | 0.017 | 0.093 | 0.005 |
| Indirect Effect | | | | | | | |
| MEQ-> BIS-11-NP->BDI | -0.005 | 0.003 | | | -0.011 | -0.001 | |
| Direct Effect | | | | | | | |
| MEQ->BDI | -0.015 | 0.007 | -0.156 | -2.221 | -0.028 | -0.002 | 0.027 |
| Total Effect | | | | | | | |
| MEQ->BDI | -0.020 | 0.007 | -0.207 | -3.005 | -0.033 | -0.007 | 0.003 |

This table presents the mediation analysis examining the indirect effect of non-planning impulsivity (BIS-11-NP) in the relationship between chronotype (MEQ) and depressive symptoms (BDI). MEQ refers to the Morningness-Eveningness Questionnaire, BIS-11-NP to the Non-Planning subscale of the Barratt Impulsiveness Scale-11, and BDI to the Beck Depression Inventory. Reported values include unstandardized coefficients (Effect), standard errors (SE), standardized coefficients (Beta), t-values, 95% confidence intervals (CI: Lower and Upper bounds), and p-values.

**Supplementary Table 2.** Mediation Analysis of Attentional Impulsivity (BIS-11-A) in the Relationship Between Chronotype (MEQ) and Depressive Symptoms (BDI)

| Components | Effect | Standardized Error | Standardized Coefficient | t | Lower | Upper | p |
|---|---|---|---|---|---|---|---|
| MEQ-> BIS-11-A | -0.062 | 0.030 | -0.147 | -2.101 | -0.121 | -0.004 | 0.037 |
| BIS-11-A->BDI | 0.031 | 0.016 | 0.137 | 1.980 | 0.001 | 0.062 | 0.049 |
| Indirect Effect | | | | | | | |
| MEQ-> BIS-11-A->BDI | -0.002 | 0.001 | | | -0.005 | -0.001 | |
| Direct Effect | | | | | | | |
| MEQ->BDI | -0.018 | 0.007 | -0.187 | -2.704 | -0.031 | -0.005 | 0.007 |
| Total Effect | | | | | | | |
| MEQ->BDI | -0.020 | 0.007 | -0.207 | -3.005 | -0.033 | -0.007 | 0.003 |

This table presents the mediation analysis exploring the indirect role of attentional impulsivity (BIS-11-A) in the association between chronotype (MEQ) and depressive symptoms (BDI). MEQ indicates the Morningness-Eveningness Questionnaire; BIS-11-A refers to the Attentional Impulsivity subscale of the Barratt Impulsiveness Scale-11; and BDI denotes the Beck Depression Inventory. The table reports unstandardized coefficients (Effect), standard errors (SE), standardized coefficients (Beta), t-values, and 95% confidence intervals (CI: Lower and Upper bounds), along with corresponding p-values.

**Supplementary Table 3.** Mediation Analysis of Psychological Resilience (PRS-33) in the Relationship Between Chronotype (MEQ) and Depressive Symptoms (BDI)

| Components | Effect | Standardized Error | Standardized Coefficient | t | Lower | Upper | p |
|---|---|---|---|---|---|---|---|
| MEQ-> PRS-33 | 0.274 | 0.059 | 0.311 | 4.638 | 0.158 | 0.391 | <0.001 |
| PRS-33 ->BDI | -0.019 | 0.008 | -0.178 | -2.488 | -0.035 | -0.004 | 0.014 |
| Indirect Effect | | | | | | | |
| MEQ-> PRS-33 ->BDI | -0.005 | 0.003 | -0.055 | | -0.011 | -0.001 | |
| Direct Effect | | | | | | | |
| MEQ->BDI | -0.015 | 0.007 | -0.152 | -2.119 | -0.028 | -0.001 | 0.035 |
| Total Effect | | | | | | | |
| MEQ->BDI | -0.020 | 0.007 | -0.207 | -3.005 | -0.033 | -0.007 | 0.003 |

This table presents the mediation analysis assessing the indirect role of psychological resilience (PRS-33) in the relationship between chronotype (MEQ) and depressive symptoms (BDI). MEQ stands for the Morningness-Eveningness Questionnaire, PRS-33 represents the total score of the Psychological Resilience Scale for Adults, and BDI denotes the Beck Depression Inventory. The table reports unstandardized coefficients (Effect), standard errors, standardized coefficients (Beta), t-values, and 95% confidence intervals (Lower and Upper bounds), along with p-values.

**Supplementary Table 4.** Mediation Analysis of Attentional Impulsivity (BIS-11-A) in the Relationship Between Chronotype (MEQ) and Anxiety Symptoms (BAI)

| Components | Effect | Standardized Error | Standardized Coefficient | t | Lower | Upper | p |
|---|---|---|---|---|---|---|---|
| MEQ-> BIS-11-A | -0.062 | 0.030 | -0.147 | -2.101 | -0.121 | -0.004 | 0.037 |
| BIS-11-A->BAI | 0.085 | 0.029 | 0.199 | 2.929 | 0.028 | 0.143 | 0.004 |
| Indirect Effect | | | | | | | |
| MEQ-> BIS-11-A->BAI | -0.005 | 0.004 | -0.029 | | -0.014 | -0.001 | |
| Direct Effect | | | | | | | |
| MEQ->BAI | -0.039 | 0.012 | -0.212 | -3.122 | -0.063 | -0.014 | 0.002 |
| Total Effect | | | | | | | |
| MEQ->BAI | -0.044 | 0.013 | -0.241 | -3.524 | -0.069 | -0.019 | 0.001 |

This table displays the mediation analysis evaluating the role of attentional impulsivity (BIS-11-A) in the relationship between chronotype (MEQ) and anxiety symptoms (BAI). MEQ refers to the Morningness-Eveningness Questionnaire, BIS-11-A denotes the attentional subscale of the Barratt Impulsiveness Scale, and BAI is the Beck Anxiety Inventory. The table includes unstandardized coefficients (Effect), standard errors, standardized coefficients (Beta), t-values, and 95% confidence intervals (Lower and Upper bounds), with associated p-values.