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Hypomanic Personality as a Moderator of Sleep Quality Effects on Affective Instability: An Ecological Momentary Assessment Study Using a Wearable Device

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This study investigated the influence of sleep quality on daily affect and affective instability, emphasizing the moderating role of hypomanic personality. Forty-two adults (≥19 years old), 21 with elevated hypomanic personality traits and 21 controls, participated in a 14-day ecological momentary assessment study. The participants completed affect assessments four times daily, and sleep indices, including sleep onset latency, total sleep time, wake after sleep onset, and sleep efficiency, were recorded using Mi Band 5 actigraphy. Compared with controls, the hypomanic personality group exhibited longer sleep onset latency and wake after sleep onset, shorter total sleep time, and lower sleep efficiency. They also reported higher levels of positive and negative affect and affective instability. Multilevel analyses revealed that hypomanic personality moderated the sleep–affect relationship: poor sleep quality predicted lower positive affect, higher negative affect, and greater affective instability, with significantly stronger effects in the hypomanic group. These findings suggest that poor sleep is a vulnerability factor for affective instability, particularly in individuals with elevated hypomanic traits. These results underscore the importance of sleep in understanding the affective processes relevant to bipolar spectrum risk and provide implications for preventive clinical interventions targeting sleep in high-risk populations.

Keywords: hypomanic personality, sleep quality, affective instability, ecological momentary assessment, wearable device

Introduction

Sleep disorders, including short or long sleep duration, poor sleep quality, and irregular sleep patterns, are among the most commonly reported psychiatric symptoms in the general population, with a high prevalence rate of 20–41.7% (Ohayon, 2011; Roth, 2007). Sleep disorders have been shown to be highly associated with mood disorders, particularly bipolar disorder recurrence and suicide attempts (Hsu et al, 2006). Poor sleep quality has been reported to increase affective instability, and insufficient sleep has been shown to elevate affective intensity and variability (Bowen, et al., 2013; Gujar et al, 2011).

The relationship between sleep and affect is complex and bidirectional (Kahn et al., 2013; Lovato & Gradisar, 2014), with previous research supporting reciprocal relationships between sleep and mood disturbances (Alvaro et al., 2013; Babson & Feldner, 2010). However, recent studies using ecological momentary assessment suggest temporal asymmetry: sleep quality strongly predicts next-day affect, while affect shows weaker or inconsistent prediction of next-day sleep (de Wild-Hartmann et al., 2013; Triantafillou et al., 2019). Consistent with this emerging evidence, the pres-

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ent study adopts a unidirectional approach examining the sleep-to-affect pathway. Beyond directionality, a methodological limitation of previous studies has been reliance on retrospective or self-report sleep measures (Bower et al., 2010). The present study addresses this limitation by employing wearable actigraphy devices to provide objective sleep assessment with improved validity and reliability (Concheiro-Moscoso et al., 2021).

Most research on sleep-affect relationships has focused on clinical populations meeting diagnostic criteria for mood disorders, while research on hypomanic personality groups remains limited (Sperry & Kwapil, 2017; Tokumitsu et al., 2021). Hypomanic personality, assessed by the Hypomanic Personality Scale (HPS; Eckblad & Chapman, 1986), is a widely used tool for identifying individuals at risk for bipolar spectrum disorders (Berson et al., 2022; Camacho et al., 2018). Hypomanic personality is conceptualized as a stable trait rather than a transient state (Nagel et al., 2022), characterized by elevated mood, increased energy, heightened sociability, and reduced need for sleep (e.g., "I feel restless and unable to keep still"; "I can go for days with very little sleep"). It reflects persistent behavioral patterns that can be considered an endophenotype for bipolar risk (Osher et al., 2019). Both theoretically and empirically, hypomanic personality connects to bipolar disorder through shared vulnerabilities including reward hypersensitivity, circadian dysregulation, and impulsivity (Alloy et al., 2017; Johnson et al., 2012, 2018), with longitudinal studies showing 20-30% conversion rates to bipolar spectrum disorders over 3-13 years (Kwapil et al., 2000; Walsh et al., 2015). Individuals with elevated hypomanic traits exhibit shorter sleep duration and poorer sleep quality (Sperry & Kwapil, 2017, 2022; Stanton et al., 2019), and report higher negative affect when sleep is reduced (Hensch et al., 2019), suggesting hypomanic personality may moderate sleep's impact on affective experiences. By examining how hypomanic personality moderates the sleep-affect relationship in a high-risk, non-clinical sample, the present study aims to inform early intervention strategies targeting sleep as a modifiable mechanism for preventing affective instability in individuals at risk for bipolar disorder (Vieta et al., 2018).

Based on existing bipolar vulnerability models, hypomanic personality is expected to moderate the sleep-affect relationship through several theoretical mechanisms. These mechanisms help account for why individuals with elevated hypomanic traits exhibit greater affective reactivity to sleep disturbances than those with lower levels of such traits. Individuals with elevated hypomanic traits tend to exhibit circadian rhythm dysregulation, reflected in irregular sleep–wake timing and heightened sensitivity to circadian disruption (Alloy et al., 2017). Notably, healthy individuals scoring high on the HPS often display sleep patterns that resemble those observed in bipolar disorder (Hensch et al., 2015). Additionally, the reward hypersensitivity theory of bipolar spectrum disorders suggests that individuals with hypomanic traits exhibit heightened reactivity to both rewarding and punishing stimuli (Alloy et al., 2015), and this heightened reactivity may extend to sleep-related physiological signals, making these individuals more sensitive to the affective impact of sleep disturbance (Johnson et al., 2012). Furthermore, individuals with hypomanic personality demonstrate elevated arousal and stress sensitivity (Johnson et al., 2012; Levenson et al., 2013), and difficulties in arousal regulation may reduce capacity to regulate emotions following sleep disruption. Together, these mechanisms–circadian dysregulation, reward hypersensitivity, and arousal sensitivity–suggest that hypomanic personality represents a vulnerability factor that increases susceptibility to the affective consequences of poor sleep quality (Johnson et al., 2012).

To address the limitations of previous research on sleep-affect relationships, the present study employed objective sleep measurement via wearable actigraphy devices and ecological momentary assessment (EMA) methodology to examine these relationships in non-clinical individuals with hypomanic personality traits. This combination of objective sleep assessment through actigraphy and intensive longitudinal measurement through EMA in a high-risk, non-clinical sample represents a methodological advancement over prior research, which has largely relied on retrospective self-report in clinical populations (Bower et al., 2010; Kahn et al., 2013).

Specifically, we examined whether hypomanic personality moderates the impact of sleep quality on daily affective experiences. Although hypomanic personality reflects a relatively stable trait vulnerability and is not itself a direct target for intervention, identifying sleep as a modifiable mechanism that disproportionately affects high-risk individuals may guide the development of sleep-focused preventive strategies (e.g., cognitive-behavioral therapy for

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insomnia, sleep hygiene education) for those at risk for bipolar disorder (Vieta et al., 2018). By testing this moderation effect in a non-clinical high-risk sample, the present study aims to provide evidence that can inform early intervention efforts targeting sleep as a pathway to reducing affective instability.

Research Questions and Hypotheses

Research Question 1: Will hypomanic personality have a moderating effect on the relationship between sleep quality and affect?

Research Hypothesis 1-1: The degree of hypomanic personality will moderate the relationship between sleep quality and negative affect.

Research Hypothesis 1-2: The degree of hypomanic personality will moderate the relationship between sleep quality and positive affect.

Research Hypothesis 1-3: The degree of hypomanic personality will moderate the relationship between sleep quality and affective instability.

Methods

Study Participants

Participants were adults who voluntarily applied through online announcements between January and February 2022. They had no history of mental illness and were not taking psychotropic medications affecting the sleep-wake cycle.

Participants completed demographic information and self-report scales via Google Docs. A structured clinical interview (*SCID-5*) was conducted by two trained master's students. Participants were classified into two groups based on Hypomanic Personality Scale (HPS) scores. The hypomanic personality (high-risk) group included participants scoring 30 points or above, while the control group included those scoring 22 points or below (Kim & Oh, 1996). Participants scoring between 23 and 29 points were excluded to ensure clear group separation. A recent validation of the Korean HPS (Oh et al., 2018) reported a mean of 13.17 ($SD = 7.52$) in Korean adults ($N = 230$), indicating that the high-risk threshold (≥ 30) represents approximately 2.2 standard deviations above the Korean normative mean, and the control group upper boundary (≤ 22) represents approximately 1.2 standard deviations above this mean.

A total of 74 participants were recruited. After excluding 25 participants based on clinical interviews and 7 additional participants for various reasons, the final sample consisted of 42 participants: 21 in the HPS hypomanic personality group and 21 in the control group. The average age was 23.74 years ($SD = 2.56$, range = 20–30 years).

Measurement Tools

Hypomanic Personality Scale (HPS)

A 48-item tool screening hypomanic tendencies in non-clinical populations. Responses are "yes/no," with scores ranging from 0–48 points. Internal consistency (Cronbach's α) was .81 in the current sample.

Pittsburgh Sleep Quality Index (PSQI)

A 19-item self-report scale measuring sleep quality over the past month through seven subfactors. Scores range from 0–21 points, with higher scores indicating poorer sleep quality. Internal consistency (Cronbach's α) was .80 in the current sample.

Center for Epidemiologic Studies–Depression Scale–Revised (CES–D–R)

A 20-item scale measuring depressive symptoms on a 5-point Likert scale (0–80 points). Internal consistency (Cronbach's α) was .86 in the current sample.

Sleep and Affective Diaries

Participants completed daily sleep diaries at 9 AM and affective diaries four times daily for 14 days. Sleep variables included sleep onset latency, wake after sleep onset, total sleep time, and sleep efficiency. Sleep efficiency was calculated as (Total Sleep Time / Time in Bed) × 100, using values extracted from Mi Band 5 data. Sleep diaries were used to replace data from days when participants failed to wear the wearable device or reported inaccurate measurements, with sleep efficiency computed using the same formula for diary-based data.

Affect was assessed using items from the Korean version of the Positive and Negative Affect Schedule (PANAS; Ahn & Kim, 2017). Based on emotion frequency data from Korean young adults, the top 10% most frequently experienced emotions were

identified. Following expert review, four positive affect items (excited, relaxed, satisfied, thrilling) and four negative affect items (difficult, irritating, sad, anxious) were selected. Each item was rated on a 7-point Likert scale (1 = not at all, 7 = extremely). Affective instability was calculated using the Mean Square Successive Difference (*MSSD*).

Xiaomi Mi Band 5

Sleep was objectively measured using the Xiaomi Mi Band 5, a wrist-worn actigraphy device. The device employs three sensors: (1) a triaxial accelerometer to detect body movements, (2) a proximity sensor to confirm skin contact, and (3) a photoplethysmography (PPG) sensor to monitor heart rate continuously (Benavides et al., 2023).

The device uses a proprietary algorithm that integrates movement patterns from the accelerometer and heart rate data from the PPG sensor to automatically detect sleep-wake states and calculate sleep parameters. Sleep onset is detected when minimal movement and reduced heart rate are sustained over time, while wake periods are identified through increased movement and heart rate elevation. Although the Mi Band 5 can differentiate sleep stages (light sleep, deep sleep, REM sleep) based on movement intensity and heart rate variability patterns, the present study utilized only sleep continuity parameters.

Four sleep parameters were extracted: sleep onset latency (time from lying down to sleep onset), wake after sleep onset (minutes awake after initial sleep onset), total sleep time, and sleep efficiency. Sleep efficiency was operationalized as (Total Sleep Time/Time in Bed) × 100, using device-recorded values.

Participants wore the device for 14 consecutive nights. To verify measurement validity in the present sample, we examined correlations between Mi Band 5 and sleep diary data, which showed moderate to high agreement: sleep onset latency ($r = .78$, $p < .01$), wake after sleep onset ($r = .56$, $p < .01$), total sleep time ($r = .81$, $p < .01$), and sleep efficiency ($r = .74$, $p < .01$). When device malfunction or non-wear occurred, data were replaced with sleep diary reports following systematic verification (2-5% of nights).

Procedures

Selected participants attended orientation sessions and received training on EMA participation using Mi Band and smartphones. After a one-day practice session, the main study was conducted for 14 days. Participants completed sleep diaries once daily at 9 AM and mood diaries four times daily between 10 AM and 10 PM at 3-hour intervals. All diaries were completed via personalized Google Docs links sent via text message. Responses submitted more than 1 hour after the prompt were excluded from analysis.

Objective sleep data from Mi Band 5 underwent systematic quality control. When participants failed to wear the device or reported device malfunction, sleep diary data were used as substitutes. Participants uploaded daily screenshots of Mi Band data, which research staff reviewed for measurement discrepancies (e.g., unrealistic sleep times). Device data with confirmed errors were replaced with sleep diary data. Nights with both device failure and participant recall difficulties were excluded as missing data. Compliance was high: the average EMA participation rate was 94% (65.8 out of 70 prompts per person, $SD = 2.84$). For objective sleep measurement, Mi Band data were available for the vast majority of nights, with sleep diaries substituting for approximately 2-5% of nights when device data were unavailable or unreliable. Strong convergent validity between Mi Band and sleep diary measures supported the reliability of diary-based substitution, with correlations of $r = .81$ for total sleep time, $r = .78$ for sleep onset latency, $r = .74$ for sleep efficiency, and $r = .56$ for wake after sleep onset (all $p < .01$). All participants received monetary compensation based on participation rate.

The use of sleep diaries as valid alternatives to actigraphy when device data are unavailable has been widely discussed and supported in the sleep research literature. Convergence between diary and objective sleep measures is often interpreted as good when correlations are in the higher range (around $r = .70$), although findings vary across studies (Lauderdale et al., 2008; Buysse et al., 2007). In ecological momentary assessment studies, substituting diary-based estimates for a small proportion of missing device data can be justified when missingness is low and convergent validity is high, as in the present study ($r = .74$–$.81$ across sleep parameters). Importantly, sleep diary substitution does not introduce outcome-predictor contamination because sleep measures (whether device-based or diary-based) served as Level 1 predictors of next-day affect, not as outcomes themselves. The temporal or-

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dering–previous night's sleep predicting next-day affect–ensures that sleep measurements (regardless of source) preceded affective assessments, eliminating the possibility of contamination. Additionally, sleep diaries and affective assessments were completed at different times (sleep diary at 9 AM, affect assessments throughout the day), further preventing overlap between predictor and outcome measurement.

All procedures were approved by the Institutional Review Board of Duksung Women's University.

Data Analysis

IBM SPSS 20 was used for demographic analyses and correlations. Multilevel modeling using HLM 7.0 was conducted to analyze intensive longitudinal data, with the *restricted maximum likelihood (REML)* method applied. Data were mean-centered: Level 1 (*within-individual*) variables were centered on individual means, and Level 2 (*between-individual*) variables were centered on overall means.

The study examined whether hypomanic personality moderated the effect of the previous night's sleep quality on the next day's affective intensity and instability.

To examine whether hypomanic personality moderated the effects of sleep quality on daily affective experiences, we constructed two-level hierarchical linear models. The collected data consisted of nested data: Level 1 (within-person) repeated measurements nested within Level 2 (between-person) individuals ($N = 42$). Such nested data structures require mean-centering procedures for appropriate multilevel analysis. At Level 1 (within-person), daily observations included the previous night's sleep parameters (sleep onset latency, wake after sleep onset, total sleep time, and sleep efficiency) as time-varying predictors of next-day affective outcomes (positive affect, negative affect, positive affective instability, and negative affective instability). All Level 1 predictors were person-centered (i.e., each individual's mean was subtracted from their daily values), allowing the model to isolate pure within-person associations between sleep and affect while controlling for between-person differences in average sleep levels. At Level 2 (between-person), group membership (0 = control group, 1 = hypomanic personality group) was modeled as a predictor of both intercepts and slopes and was grand-mean centered. Cross-level interactions between Level 1 sleep parameters and Level 2 group membership were included to test moderation effects.

Model Specification

Prior to testing the moderation models, we estimated unconditional models (intercept-only models without predictors) to calculate intraclass correlation coefficients (ICCs) for each affective outcome. The ICC represents the proportion of total variance attributable to between-person differences, with higher values indicating greater stability of individual differences and lower values indicating greater within-person variability.

For each sleep parameter, we tested: (1) the main effect of sleep quality on next-day affect at Level 1, (2) the main effect of group membership at Level 2, and (3) the cross-level interaction between sleep quality and group membership. Significant interactions were probed using simple slope analyses to examine group-specific effects.

Results

Demographic Characteristics and Group Differences

As shown in Table 1, there were no significant differences between groups in age ($t(40) = -.179$, $p = .859$) and education level ($\chi^2(1) = .171$, $p = 1.00$). However, the hypomanic personality group had a

Table 1. *Demographic Characteristics of Study Participants (N = 42)*

Variable	Hypomanic Personality Group ($N = 21$)	Control Group ($N = 21$)	t/χ^2
Age (year)	23.81 (2.71)	23.67 (2.46)	$t(40) = -.179$
Gender (Male:Female)	1 (4.8%):20 (95.2%)	8 (38.1%):13 (61.9%)	$\chi^2(1) = 6.929^*$
Education (High school:College)	17 (81.0%):4 (19.0%)	18 (85.7%):3 (14.3%)	$\chi^2(1) = .171^a$
HPS Score	32.81 (3.43)	13.57 (6.25)	$t(40) = -12.367^{***}$

Hypomanic personality group = 21, Control group = 21.
[a]*Fisher's exact test result.*
*$p < .05$. ***$p < .001$.*

significantly higher proportion of women (95.2%) than the control group (61.9%) ($\chi^2(1) = 6.929$, $p = .009$).

Correlations Between Variables

The HPS showed significant positive correlations with the sleep quality scale (PSQI; $r = .517$, $p < .001$) and with the depression scale (CES-D; $r = .378$, $p = .014$). In EMA-measured variables, HPS correlated positively with sleep latency ($r = .390$, $p = .011$), wake after sleep onset ($r = .650$, $p < .001$), negative affect ($r = .353$, $p = .022$), positive affect ($r = .434$, $p = .004$), and affective instability (negative: $r = .648$, $p < .001$; positive: $r = .391$, $p = .010$), and negatively correlated with sleep efficiency ($r = -.584$, $p < .001$). Sleep problems were closely related to affective instability, with wake after sleep onset showing positive correlations with positive affect and negative affective instability, while sleep efficiency showed a negative correlation with negative affective instability (see Supplementary Table 1 for complete correlation matrix).

Group Differences in Self-Report Measures

The hypomanic personality group had significantly poorer sleep quality ($M = 10.62$, $SD = 3.51$) than the control group ($M = 6.43$, $SD = 3.43$) ($t(40) = -3.91$, $p < .001$, d = 1.21). Depression levels were also significantly higher in the hypomanic personality group ($M = 13.67$, $SD = 8.87$) than controls ($M = 6.67$, $SD = 6.79$) ($t(40) = -2.87$, $p = .006$, d = .88).

EMA Data Differences Between Groups

As presented in Table 2, multilevel model analysis revealed significant differences between groups in all sleep variables. The hypomanic personality group had significantly longer sleep latency (B = .68, $p = .002$) and wake after sleep onset (B = .29, $p < .001$), shorter total sleep time (B = -.75, $p = .005$), and lower sleep efficien-

Table 2. *Group Differences in Sleep Variables from Ecological Momentary Assessment (n = 42)*

Variable	Sleep Onset Latency		Wake After Sleep Onset		Total Sleep Time		Sleep Efficiency	
	B	*(SE)*	*B*	*(SE)*	*B*	*(SE)*	*B*	*(SE)*
Fixed effects								
Level-1								
Intercept	.91***	(.10)	.25***	(.02)	7.10***	(.13)	83.50***	(.95)
Level-2								
Group	.68**	(.20)	.29***	(.03)	-.75**	(.25)	-10.92***	(1.89)
	Variance (*SD*)		Variance (*SD*)		Variance (*SD*)		Variance (*SD*)	
Random effects								
Random intercept	.40***	(.63)	.01**	(.07)	.53***	(.73)	33.92***	(5.82)
Error term	.54	(.73)	.11	(.33)	2.71	(1.65)	81.64	(9.04)

Hypomanic personality group = 21, Control group = 21. Group: Hypomanic personality group = 1, Control group = 0.
***p < .01. ***p < .001.*

Table 3. *Group Differences in Affective Variables from Ecological Momentary Assessment (n = 42)*

Variable	Negative Affect		Positive Affect		Negative Affective Instability		Positive Affective Instability	
	B	*(SE)*	*B*	*(SE)*	*B*	*(SE)*	*B*	*(SE)*
Fixed effects								
Level-1								
Intercept	2.35***	(.11)	4.03***	(.12)	.76***	(.05)	1.14***	(.11)
Level-2								
Group	.61**	(.22)	.73**	(.24)	.76***	(.11)	.56*	(.22)
	Variance	(*SD*)	Variance	(*SD*)	Variance	(*SD*)	Variance	(*SD*)
Random effects								
Random intercept	.48***	(.70)	.59***	(.77)	.07***	(.27)	.35***	(.59)
Error term	.51	(.72)	.72	(.85)	.83	(.91)	2.63	(1.62)

Hypomanic personality group = 21, Control group = 21. Group: Hypomanic personality group = 1, Control group = 0.
**p < .05. **p < .01. ***p < .001.*

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cy (B = -10.92, $p < .001$).

Table 3 shows results for affective variables. The hypomanic personality group demonstrated significantly higher negative affect (B = .61, $p = .007$), positive affect (B = .73, $p = .004$), and both negative (B = .76, $p < .001$) and positive affective instability (B = .56, $p = .015$) compared to controls.

Moderating Effects of Hypomanic Personality

Multilevel modeling examined whether hypomanic personality moderated the relationship between sleep quality and affective variables. The ICCs indicated substantial individual differences in baseline affective levels: negative affect (ICC = .60), positive affect (ICC = .52), negative affective instability (ICC = .22), and positive affective instability (ICC = .14). These values confirmed the appropriateness of multilevel modeling, with 52-60% of variance in affect intensity and 14-22% of variance in affective instability attributable to stable between-person differences. Random intercepts were statistically significant for all models (all $p < .001$).

1) Sleep Latency: Longer previous-day sleep latency predicted higher next-day negative affect, negative affective instability, positive affective instability, and lower positive affect (all $p < .001$). Hypomanic personality significantly moderated the effect on negative affective instability (interaction: B = .37, $p = .028$). Simple slope analyses indicated that each additional minute of sleep onset delay predicted a .52-unit increase in instability for the hypomanic group (B = .52, $p < .001$), whereas this association was non-significant in controls (B = .15, $p = .08$), representing a 3.5-fold difference in effect magnitude.

2) Wake After Sleep Onset: Greater wake after sleep onset predicted higher negative affect and positive affective instability. However, the interaction with hypomanic personality was not significant.

3) Sleep Efficiency: Higher sleep efficiency predicted higher positive affect and lower negative affect and affective instability (all $p < .001$). Hypomanic personality moderated these effects on negative affect (interaction: B = -.02, $p = .042$) and negative affective instability (interaction: B = -.06, $p < .001$). These significant interactions indicate that improvements in sleep consolidation conferred disproportionately stronger protective benefits against negative affectivity in the hypomanic group, with poor sleep efficiency predicting substantially greater affective disturbance compared to controls.

4) Total Sleep Time: As shown in Table 4 and Figure 1, longer sleep time predicted higher positive affect and lower negative affect and affective instability (all $p < .001$). Hypomanic personality significantly moderated these effects for negative affect (interaction: B = -.14, $p = .012$), positive affect (interaction: B = .17, $p = .020$), and negative affective instability (interaction: B = -.30, $p < .001$). Simple slope analyses revealed that hypomanic personality consis-

Table 4. *Moderating Effects of Hypomanic Personality on the Relationship Between Total Sleep Time and Affective Variables (n = 42)*

Dependent Variable	Negative Affect		Positive Affect		Negative Affective Instability		Positive Affective Instability	
	B	*(SE)*	*B*	*(SE)*	*B*	*(SE)*	*B*	*(SE)*
Fixed Effects								
Level-1 Intercept	2.36***	(.12)	4.03***	(.16)	.77***	(.08)	1.22***	(.18)
Time	-.00	(.01)	.00	(.01)	-.00	(.01)	-.00	(.01)
Total Sleep Time	**-.24*** **	**(.03)**	**.28*** **	**(.04)**	**-.25*** **	**(.03)**	**-.38*** **	**(.07)**
Level-2 Group	.63**	(.22)	.71**	(.22)	.76***	(.11)	.47*	(.21)
Group × Total Sleep Time	**-.14***	**(.05)**	**.17***	**(.07)**	**-.30*** **	**(.07)**	-.23	(.14)
	Variance	(*SD*)	Variance	(*SD*)	Variance	(*SD*)	Variance	(*SD*)
Random Effects								
Random Intercept	.47***	(.69)	.91***	(.95)	.13*	(.36)	.82***	(.90)
Random Slope	.02***	(.14)	.04***	(.20)	.03**	(.18)	.20***	(.44)
Error Term	.35	(.59)	.46	(.68)	.57	(.76)	1.97	(1.40)

Total sleep time is person-centered within individuals. Group: Hypomanic personality group = 1, Control group = 0. Time = Measurement occasion (Days 1-14).
**p < .05. **p < .01. ***p < .001.*

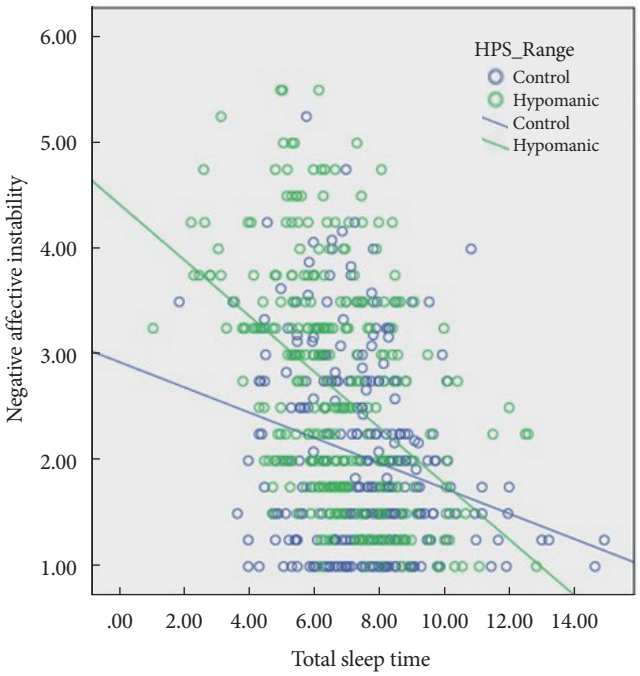

Figure 1. *Moderating effect of hypomanic personality on the relationship between total sleep time and negative affective instability.*

tently amplified sleep's impact on affective experiences. For negative affect, each additional hour of sleep produced a .38-unit reduction in the hypomanic group (B = -.38, $p < .001$) compared to .24 units in controls (B = -.24, $p < .001$)–a 1.6-fold difference. Similarly, for positive affect, the hypomanic group showed 1.6 times greater benefit from adequate sleep (.45- unit increase per hour, B = .45, $p < .001$) compared to controls (.28 units, B = .28, $p < .001$). The strongest moderation emerged for negative affective instability, as illustrated in Figure 1.

Figure 1 displays individual daily observations across the 14-day study period, with the control group (blue circles) and hypomanic personality group (green circles) showing distinct patterns. The regression lines demonstrate that both groups exhibited negative associations between sleep duration and next-day instability; however, the hypomanic group displayed a steeper slope. Quantitatively, each one-hour reduction in sleep duration predicted a .55-unit increase in negative affective instability for the hypomanic group (B = -.55, $p < .001$) versus .25 units for controls (B = -.25, $p < .001$), representing a 2.2-fold difference. This pattern indicates that individuals with elevated hypomanic traits experienced greater affective destabilization from reduced sleep, whereas controls maintained relative stability across varying sleep durations.

Supplementary Figures 1 and 2 illustrate the parallel moderation patterns for negative and positive affect, with the hypomanic group consistently showing steeper slopes (approximately 1.6 times larger) than controls across all affective outcomes.

Discussion

This study examined whether hypomanic personality moderates the sleep-affect relationship using objective sleep measurement via wearable actigraphy and ecological momentary assessment over 14 days. Results confirmed that poor sleep quality predicts next-day negative affect, reduced positive affect, and greater affective instability, with significantly stronger effects in individuals with elevated hypomanic personality traits. These findings deepen theoretical understanding of sleep-affect dynamics and underscore the need for targeted preventive interventions for those at risk for bipolar disorder.

This study demonstrates that hypomanic personality fundamentally alters how sleep disturbances impact daily affective experiences. Individuals with elevated hypomanic traits showed 1.6 to 3.5 times stronger associations between poor sleep and next-day affective disturbances compared to controls, indicating that sleep functions as a modifiable vulnerability mechanism in bipolar risk. This amplified reactivity can be understood through three converging theoretical frameworks: circadian rhythm dysregulation, reward hypersensitivity, and arousal sensitivity characteristic of hypomanic personality (Alloy et al., 2017; Johnson et al., 2012, 2018).

First, circadian rhythm instability models (Alloy et al., 2017; Murray et al., 2017) propose that individuals with hypomanic traits exhibit fundamental dysregulation in biological rhythms, creating heightened sensitivity to sleep disruption. Even modest reductions in sleep quality trigger disproportionate affective consequences in high-risk individuals, as sleep disturbance acts as a proximal trigger activating underlying circadian vulnerability. Research showing that high-HPS individuals exhibit sleep patterns similar to those with bipolar disorder (Hensch et al., 2015) supports this interpretation.

Second, reward hypersensitivity theory (Alloy et al., 2015) pro-

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vides additional explanatory power. Individuals with hypomanic traits show heightened reactivity to both rewarding and punishing stimuli, and this reactivity extends to sleep-related physiological signals. Poor sleep may represent a form of physiological "punishment" to which high- HPS individuals are disproportionately sensitive, amplifying negative affective responses.

Third, difficulties in arousal regulation characteristic of hypomanic personality (Johnson et al., 2012) reduce capacity to compensate for sleep loss through emotion regulation strategies, leading to greater affective volatility following poor sleep. Together, these mechanisms suggest that sleep disturbance represents a modifiable mechanism that activates multiple vulnerability pathways in high-risk individuals.

The present findings extend Sperry and Kwapil's (2022) research showing that sleep duration variability predicts increased negative affect in hypomanic individuals. The current study demonstrates that this vulnerability extends across multiple sleep parameters (sleep onset latency, total sleep time, sleep efficiency) and affects not only negative affect but also positive affect and overall affective instability. While sleep-affect relationships existed in the general population, they were significantly amplified in the hypomanic personality group, confirming that those at risk for bipolar disorder experience greater affective difficulties due to sleep problems.

These findings have important implications for early intervention strategies. Given that sleep represents a modifiable mechanism that disproportionately affects high-risk individuals, sleep-focused preventive interventions offer a promising avenue for reducing affective instability and potentially delaying or preventing bipolar disorder onset.

Three concrete intervention approaches are suggested. First, cognitive-behavioral therapy for insomnia (CBT-I) has demonstrated efficacy in improving sleep quality and may be particularly beneficial for individuals with hypomanic traits (Harvey et al., 2015). CBT-I targets maladaptive beliefs about sleep, establishes consistent sleep-wake schedules, and teaches behavioral strategies–all of which may help buffer against circadian dysregulation. Second, sleep hygiene education emphasizing regular sleep-wake timing, appropriate caffeine and alcohol use, and pre-sleep routines may help stabilize circadian rhythms in high-risk individuals. Third, interventions targeting social rhythm regularity (e.g., Interpersonal and Social Rhythm Therapy; Frank et al., 2005) may reduce vulnerability to mood episodes by stabilizing daily routines and sleep-wake cycles.

Early identification of sleep problems in individuals with elevated hypomanic traits may serve as a valuable screening mechanism. Clinicians working with young adults showing hypomanic personality features should routinely assess sleep quality and consider sleep-focused interventions as a first-line preventive approach before more significant mood symptoms emerge.

To evaluate whether such sleep vulnerabilities manifest in daily life, the present study combined objective sleep measurement through actigraphy with intensive longitudinal assessment via EMA. However, the finding that wake after sleep onset did not show moderation effects may reflect measurement limitations of the Mi Band 5, as motion-based accelerometers show reduced accuracy with frequent movement during sleep (Lee et al., 2018).

Future research should consider several enhancements. First, integrating multimodal physiological indicators–including heart rate variability, skin temperature, and light exposure–alongside actigraphy would provide more comprehensive assessment of sleep physiology and circadian rhythms. Second, increasing EMA sampling frequency to 6-12 times daily would enable finer-grained estimation of affective variability and capture more complete affective dynamics (Shin, 2010). Third, longer assessment periods (e.g., 28 days) would allow examination of weekly and monthly patterns in sleep-affect relationships.

Several limitations should be noted. First, the sample consisted primarily of college students, limiting generalizability. Future studies should examine broader age ranges and community samples. Second, participants were non-clinical and had not yet developed full bipolar disorder. Longitudinal designs following high-HPS individuals over several years could track conversion to bipolar disorder and examine whether sleep disturbances prospectively predict mood episode onset.

Third, affective instability assessed via MSSD with four daily measurements may not fully capture complete affective dynamics; more frequent sampling (6-12 times daily) would provide more reliable estimates. Fourth, external factors affecting sleep–including caffeine intake, physical activity, alcohol use, and daily stressors–

were not systematically measured or controlled. These variables were not systematically measured. Although data collection during a vacation period minimized academic stress, the absence of daily behavioral monitoring represents a limitation that should be addressed in future research through systematic collection of these covariates. Future research should collect daily reports of these potential confounders and include them as covariates to strengthen causal interpretations.

Fifth, accelerometer-based measurement has limitations with excessive movement during sleep, potentially affecting wake after sleep onset measurements (Lee et al., 2018). Future studies should employ validated research-grade actigraphy or polysomnography. Finally, the correlational design precludes causal inferences; experimental sleep manipulation or intervention trials would provide stronger evidence for causal relationships.

Conclusion

Despite these limitations, this study makes important contributions to understanding sleep-affect relationships in daily life. Multiple aspects of sleep quality–including sleep onset latency, total sleep time, and sleep efficiency–predict next-day affective intensity and instability, with particularly strong effects in individuals with hypomanic personality traits. By identifying sleep as a modifiable mechanism that disproportionately affects high-risk individuals, this research provides a foundation for developing targeted early interventions aimed at reducing affective instability and potentially preventing mood disorder onset in vulnerable populations. The results support the important role of sleep in affective instability among those at high risk for bipolar disorder and suggest that preventive sleep interventions could contribute meaningfully to bipolar disorder prevention strategies.

Data Availability Statement

The datasets generated and analyzed during the current study are not publicly available due to privacy and confidentiality restrictions to protect research participants' identities. However, anonymized data may be available from the corresponding author upon reasonable request and in compliance with institutional review board guidelines and applicable privacy regulations.

Author contributions statement

Seulgi Lim, a graduate student at Duksung Women's University and currently a Clinical Psychological Trainee in the Department of Psychiatry at Boramae Medical Center, led the conceptualization and design of the study, conducted data collection and analysis, and drafted the manuscript. Sungwon Choi, Professor at Duksung Women's University, supervised the overall research, provided guidance on study design, and critically reviewed the manuscript. Both authors reviewed and approved the final manuscript and take responsibility for all aspects of the work.

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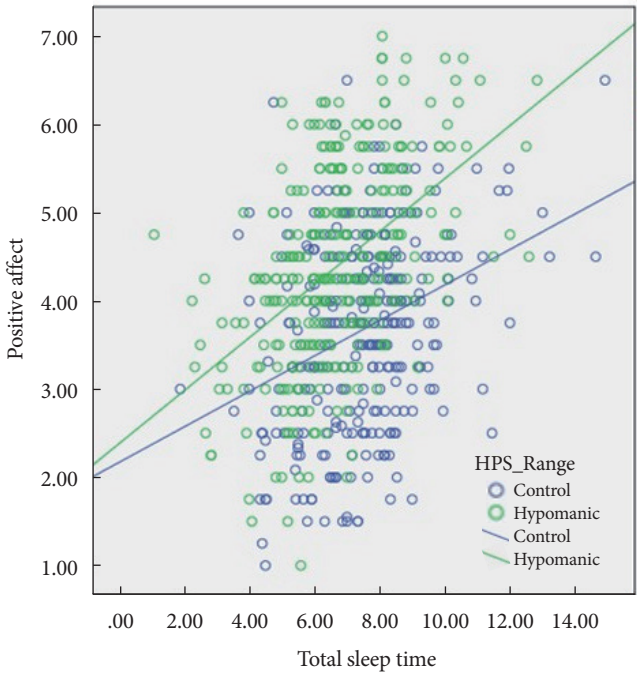
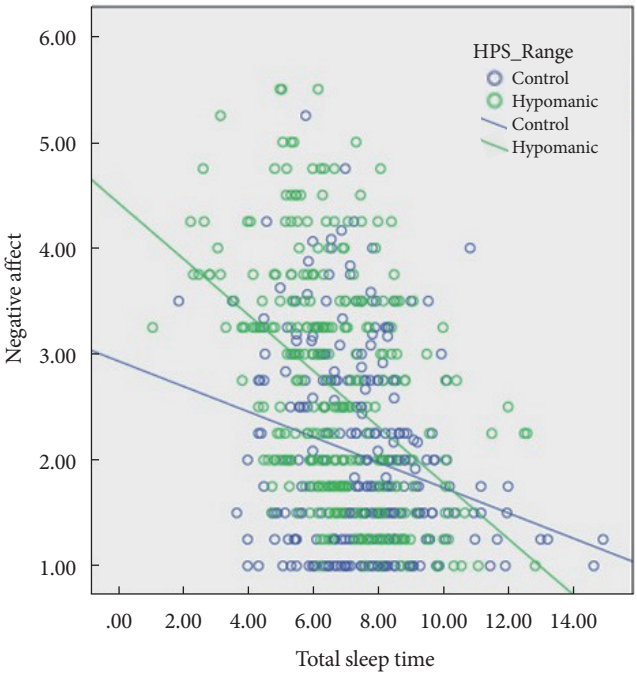
Supplementary Table 1. *Correlations Between Variables*

	1	2	3	4	5	6	7	8	9	10	11
1. Hypomanic personality scale (HPS)	1										
2. Pittsburgh sleep quality index (PSQI)	.517**	1									
3. Center for epidemiologic studies-depression scale-revised (CES-D-R)	.378*	.173	1								
4. Sleep latency	.390*	.182	.324*	1							
5. Wake after sleep onset	.650**	.502**	.217	.273	1						
6. Total sleep time	-.292	-.489**	-.181	-.019	-.287	1					
7. Sleep efficiency	-.584**	-.387*	-.427**	-.927**	-.502**	.196	1				
8. Negative affect	.353*	.181	.483**	.157	.208	-.279	-.222	1			
9. Positive affect	.434**	.023	-.105	-.181	.311*	.017	.084	-.081	1		
10. Negative affective instability	.648**	.459**	.481**	.233	.596**	-.273	-.402**	.544**	.146	1	
11. Positive affective instability	.391*	.228	.384*	.034	.259	-.076	-.174	.218	.144	.686**	1

*$p < .05$. **$p < .01$.



Supplementary Figure 1. *Moderating effect of hypomanic personality on the relationship between total sleep time and positive affect.*



Supplementary Figure 2. *Moderating effect of hypomanic personality on the relationship between total sleep time and negative affect.*

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