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A randomized controlled trial of a behavioral intervention for decreasing bedtime procrastination using a wait-list control group in a non-clinical sample of young adults

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ABSTRACT

Study objectives: Bedtime Procrastination (BP) is defined as the behavior of going to bed later than intended, without external reasons. This study is a randomized controlled trial aiming to establish efficacy of a behavioral intervention to decrease BP in a non-clinical sample.
Method: This was an open-label trial that was conducted in sixty participants (mean age 21.33 ± 2.35 years, 86.7% females) without insomnia or psychopathology who endorsed frequent BP. They were randomized to either the treatment group (n = 32) or wait-list control group (n = 28). Participants completed the Bedtime Procrastination Scale (BPS), the Epworth Sleepiness Scale (ESS), the Insomnia Severity Index (ISI), Center for Epidemiologic Studies Depression Scale (CES-D), Morningness-Eveningness Questionnaire (MEQ), and a weekly sleep diary. Functional analysis was conducted to investigate the function of BP. Linear mixed models were used for analyses.
Result: The treatment group showed significant improvement on the BPS (35.56% decrease, $d = 2.19$, bedtime procrastination duration based on the sleep diary (−46.29 min, $d = 1.22$), and sleep efficiency (5.70% increase, $d = 1.25$) compared to the wait-list control group following the intervention. There were also significant reductions in time spent from bedtime to lights out, and wake time to time out of bed, in addition to improvements in ISI and ESS scores in the treatment group compared to the control group. Functional analysis results indicated emotional regulation (31.3%), compensation (26.5%), and social interaction and belongingness (18.1%) as the most frequent functions of bedtime procrastination.
Conclusion: This study shows promising results for a behavioral intervention targeting BP and sleep. In addition, this study demonstrated various functions of BP as a sleep-interfering behavior. We expect that these findings could be used in future studies and clinical settings to decrease BP.

1. Introduction

Bedtime procrastination is defined as “going to bed later than intended, without having any external factors for doing so” [1]. Recently, bedtime procrastination has been considered a significant health behavior because of its associations with insufficient sleep and insomnia, and other psychological variables such as depression and anxiety [1,2]. From a socio-cultural context, there has been a general increase in media use [3,4], which has also resulted in increased media use before bedtime, with more individuals using smartphones and computers that cause delays in their bedtime [2,3].

We have previously reported that individuals who engage in high levels of bedtime procrastination mainly engage in more smartphone use 3 h before bedtime compared to those who have low levels of bedtime procrastination [2]. While there are many effective non-pharmacological interventions to improve sleep, many of the techniques used in these interventions were developed during a time that does not reflect the modern sociocultural context and updates in technology. Many sleep interventions aimed to improve sleep, such as sleep hygiene [5], stimulus control [6], sleep restriction [7], relaxation techniques [8] were developed during a time when ubiquitous personal electronic media use was

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Abbreviations:

BP	Bedtime Procrastination
BPD	Bedtime Procrastination Duration
BPS	Bedtime Procrastination Scale
BT	Bedtime;
CBTI	Cognitive Behavioral Therapy for Insomnia
CES-D	Center for Epidemiologic Studies Depression Scale
DBSM	Diplomate of Behavioral Sleep Medicine;
ESS	Epworth Sleepiness Scale
FRESH	Feeling Refreshed Upon Awakening
ISI	Insomnia Severity Index
LMM	Linear Mixed Models
LO	Lights Off
MEQ	Morningness-Eveningness Questionnaire;
RCT	Randomized Controlled Trial
SE	Sleep Efficiency
SOL	Sleep Onset Latency
SQ	Sleep Quality
TIB	Time In Bed
TST	Total Sleep Time;
TTM	Transtheoretical Model
WASO	Wake After Sleep Onset;
WT	Wake Time

not an issue, especially at bedtime. Therefore, with the fast-changing landscape of universal media use and the effect that these devices have on postponing sleep, there is a need for interventions that target bedtime procrastination as a serious health-interfering behavior [9].

For such reasons, we developed a behavioral intervention for reducing bedtime procrastination (BED-PRO) based on the stage model [10,11]. This intervention was developed based on the theoretical framework of the transtheoretical model (TTM) using motivational interviewing techniques and behavioral modification principles. During the first stage, our research team verified the feasibility and acceptability of the behavioral intervention developed to specifically target bedtime procrastination using a single-group pre-post and follow-up design study, resulting in a 63.8% reduction (Δ51 min) in bedtime procrastination compared to baseline, in addition to significant improvements in wake after sleep onset, sleep efficiency, and other self-report scores of insomnia and daytime sleepiness [9].

Based on our previous study, the current study aims to establish efficacy of the developed intervention utilizing a more rigorous study design. We implemented an open label randomized controlled trial comparing the intervention group to a control group. In addition, the current aim included a functional analysis of bedtime procrastination to identify the emotional or behavioral purpose of engaging in bedtime procrastination. Recent studies have suggested that procrastination can be used as an emotional regulation strategy [12,13]. Thus, the present study hypothesized that emotion regulation would be one of the main functions of bedtime procrastination.

2. Methods

2.1. Participants and procedures

Participants were recruited between March 2019 to July 2020 in Seoul, South Korea using advertisements through online community postings in 23 universities in Seoul. Offline fliers using the same content was also used to advertise in the community. Sixty participants free of insomnia or psychopathology who endorsed that they frequently engage in bedtime procrastination were selected to participate in the study. While we did not have eligibility criteria for age to participate in the study, we actively advertised the study targeting young adults in their 20s, as previous studies have noted that general procrastination behavior was most common in early adulthood [14]. Individuals who scored 33 or higher on the bedtime procrastination scale (BPS) were included in the study, based on previous studies using this cut-off score [15].

A total of 134 volunteers were screened for the study, and 74 potential participants were selected based on inclusion criteria. Next, telephone screening interviews were conducted to ensure that participants met inclusion criteria. During in the telephone screening, potential participants who did not meet DSM-5 criteria for insomnia disorder were selected. In addition, other mental illnesses (psychosis, bipolar disorder, and other sleep disorders) that affect participants' sleep were screened. For the same reason, individuals who were currently participating in sleep-related interventions such as CBT-I or taking medications were not included in this study.

Specific exclusion criteria were: (a) scores of 15 or higher on the Insomnia Severity Index and individuals who met criteria for insomnia disorder based on the DSM-5, as they may have different reasons for procrastinating their bedtime (e.g., aversion to going to bed due to inability to sleep) compared to individuals without insomnia; (b) history of suicide attempts; (c) previous diagnosis of bipolar disorder, schizophrenia, and other sleep disorders; (c) currently taking sleep-related medication or participating in psychotherapy; and (d) being a shift worker. Fig. 1 presents a flowchart of the recruitment procedure.

2.2. Study design and procedure

The current study was a randomized, open label trial with two study arms. This study was approved by Sungshin Women's University Institutional Review Board and was registered with the Clinical Research Information Service (CRIS, cris.nih.go.kr, KCT0007337).

Participants were sequentially randomly allocated to the treatment group or the wait-list control group using simple randomization. The simple randomization table was created using Excel by two researchers who conducted recruitment and treatment of participants using a random number generation function (=RAND). These random numbers were arranged in ascending order, with half of the participants who were sorted by random numbers being assigned to the treatment group and the other half to the control group. After randomization, participants were assigned a subject ID. This study was an open label trial. Therefore, there is a possibility that participants were aware of which conditions they were randomized in, although participants were not informed about the conditions they were allocated. After randomization, both groups received the same following description of the study during their Visit1: “This study is being conducted to prove the effectiveness of a behavioral intervention for reducing bedtime procrastination.” Participants assigned to the wait-list control groups received the same intervention after their control period.

During visit 1, participants completed the baseline questionnaire that consisted of demographic information, self-report questionnaires, and sleep diaries. The treatment group participated in the intervention for four weeks after their first visit. After

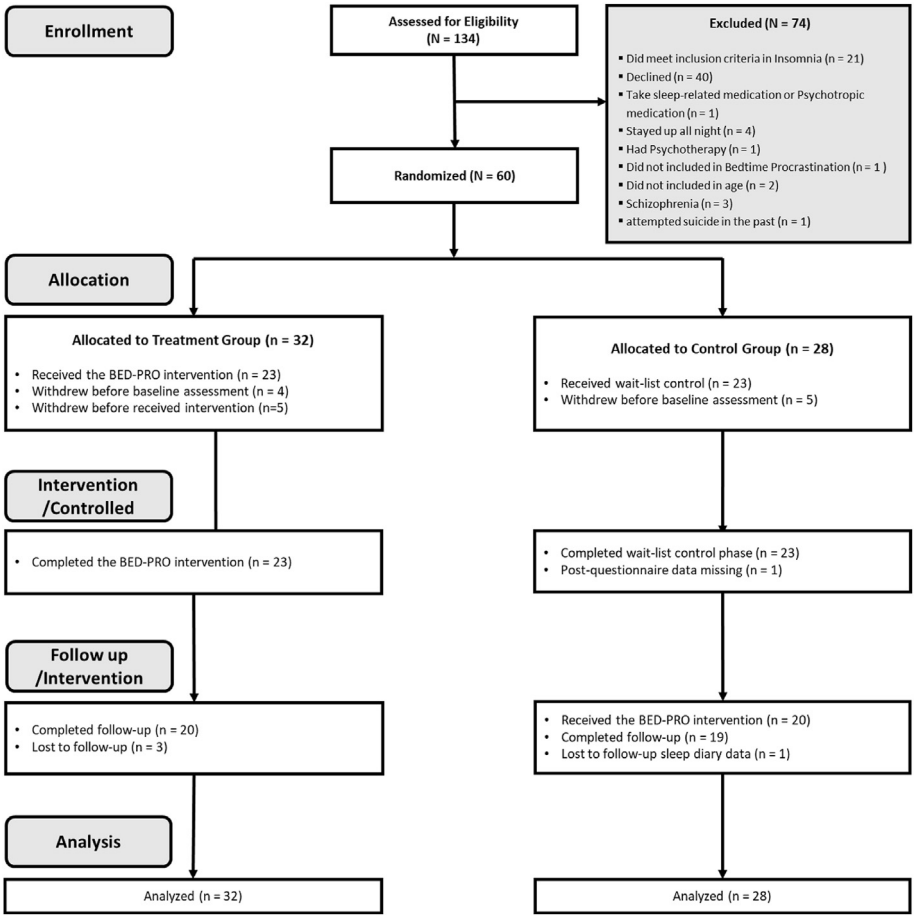


Fig. 1. Flowchart of study Participants in the TRT group were allocated to 4 weeks of BED-RPO intervention, followed by assessments at post-treatment and 1-month follow-up. The CTRL group was not treated during the 4 weeks that the TRT group received the intervention, After the end of the controlled period, they received the same BED-PRO intervention as the TRT.

the booster call session, all post-intervention data was collected. A month after the booster call session, participants were asked to complete the follow-up assessment. The wait-list control group received no treatment for four weeks, and after that period they were provided with the same intervention as the treatment group. Assessments were performed at baseline (T1) and after the intervention (T2). The treatment group also completed one-month follow-up assessment (T3). The wait-list control group received the intervention in a cross-over design after T2, and received their one-month follow-up assessment (T3) after receiving the intervention.

Nine individuals withdrew from the study after being randomized and prior to baseline assessment. Since most individuals received their baseline assessment within one week of their screening, we used screening data of these nine individuals as a proxy for baseline assessment. The procedure of collecting data during the study process is presented in Fig. 1.

2.3. Sample size calculation

The sample size calculation was conducted using G*Power [16]. A priori power analyses based on t-tests showed that 23 participants were needed in each group. Specifically, the sample size was calculated considering the following parameters: effect size = 0.86, $\alpha = 0.05$, power = 0.80, and number of groups = 5. The effect size was defined through the previous study that used the bedtime procrastination scale as a primary outcome [17]. Furthermore, in the previous study of dropouts from group cognitive-behavior therapy for insomnia (CBT-I), nearly 7% of patients terminated prior to the fourth session. Therefore, considering the dropout rate, we made a final sample size of 60 participants for the study [18]."

2.4. Intervention

The behavioral intervention developed to specifically target bedtime procrastination intervention is a short-term psychological intervention consisting of a total of four sessions. Participants had three weekly face-to-face sessions and one booster call session (via telephone) with their therapist. Each session was 50 min per week, with the booster call session typically lasting 20 min. During the study, the COVID-19 pandemic was announced, which made it difficult to maintain face-to-face sessions. *Therefore, some sessions during lockdown were conducted as online sessions. Specifically, one participant participated online for Visit 1 and two sessions, one participant participated all three sessions online, and one participant participated online for two sessions.*

The behavioral intervention developed to specifically target bedtime procrastination intervention was based on the trans-theoretical model [19] using motivational interviewing techniques, and behavior modification principles. A review of the intervention can be found here [9]. The intervention consisted of motivational interviewing techniques by connecting behavioral change (e.g., decreasing bedtime procrastination) with their values. Functional analysis was also conducted utilizing a cognitive-behavioral

approach to bedtime procrastination by identifying thoughts and feelings that preceded the problem behavior (e.g., bedtime procrastination) and explored factors that increase the likelihood of engaging in bedtime procrastination (reinforcers), which occurred during Session 1. Functional analysis consists of the following: Antecedents(A): situations, thoughts, and emotions before going to bed; Behaviors(B): specific behavior during delaying bedtime, and Consequences(C): Reinforcement that increases bedtime procrastination. Researchers used structured questions to investigate the function of bedtime procrastination. For example, "What thoughts and emotions did you notice before engaging in bedtime procrastination?", "What kind of behaviors do you engage in during delayed bedtime?", "What did you get out of engaging in bedtime procrastination?". Every participant who received the intervention was asked to record the results of the functional analysis on paper with the therapist as an interactive activity. After conducting functional analysis to identify the reinforcers that were causing the problem behavior, the therapist and participant would work together to identify alternative behaviors that were more adaptive but had the same function as the bedtime procrastination. Participants conducted this procedure guided by therapists and found alternative behaviors to replace activities that had higher likelihood of bedtime procrastination.

For actual behavioral changes, participants planned a weekly bedtime schedule and signed a behavioral contract with the therapist. For example, participants were asked to identify their problem behavior (e.g., spending 3 h on phone prior to bedtime), identify antecedents of the problem behavior based on functional analysis (e.g., "I start scrolling on my phone when I feel depressed to avoid ruminating", "I felt lonely because I didn't talk to anyone today"), and identify a different and potentially more desirable behavior following the antecedent (e.g., "I took a warm bath to treat myself). Other behavioral changes involved identifying the function of bedtime procrastination for that individual (e.g., feeling lonely), and trying to pre-emptively schedule the day to fulfill the function that was driving bedtime procrastination at night (e.g., schedule a quick coffee break during the day). Imagery training using the planned weekly bedtime schedule consisting of series of behaviors before bedtime activities was implemented to increase likelihood of going to bed at the planned time.

2.5. Therapist and intervention integrity

The intervention was led by a Ph.D. level licensed clinical psychologist with Diplomate of Behavioral Sleep Medicine (DBSM) certification and two master's level graduate students receiving clinical supervision trained in behavioral sleep medicine. The graduate students who participated in this study had previous training in behavioral sleep medicine and behavioral therapy and was specifically trained for this intervention by DBSM certified licensed psychologist (S.S.). The intervention was structured and manualized. First, all study therapists received training about the protocol of the intervention. The next step involved observing the licensed clinical psychologist with the protocol. Finally, all students practiced all intervention modules with four mock participants and received subsequent supervision prior to starting the study. The graduate students received ongoing clinical supervision throughout the study.

All sessions were audio-recorded, and 31 recordings of the sessions were assessed by two independent research assistants to evaluate equivalence and integrity of the content. A checklist for each session was rated based on an experimenter-derived fidelity checklist [20] coding the presence/absence of 23 essential components.

2.6. Functional analysis of bedtime procrastination

A functional analysis of bedtime procrastination was conducted during the first session of the intervention.

Based on previous studies of general procrastination and bedtime procrastination [9,12,13,21,22], functions of bedtime procrastination were classified as follows: (1) emotion regulation [12,13]; (2) rewards [21]; (3) sleep inducing [21]; and (4) social interaction and belongingness [15]. Next, functions of bedtime procrastination for each individual were determined through verbatim records and recording tapes, and these functions were classified based on the primary function. Subsequently, researchers discussed the need to add additional functions of bedtime procrastination to the primary classification, and a consensus was reached through group discussion. During this process, additional secondary functional areas were established based on the content and words repeatedly reported by the participants. The functions of bedtime procrastination that were added to the original list were as follows: (5) acquisition of information and knowledge; (6) accomplishment; and (7) pleasure.

Finally, the functions of bedtime procrastination were classified using the following 7 categories: (1) emotion regulation: reducing or avoiding negative emotions through delayed bedtime behavior; (2) rewards: to use "having 'me' time" or "rewarding oneself" for working hard during the day through bedtime procrastination; (3) social interaction and belongingness: to gain feelings of belonging or interaction to more than one person or groups of people through bedtime procrastination; (4) acquisition of information and knowledge: to gain information and knowledge from any material or news by doing bedtime procrastination; (5) sleep inducing: de-arousing through bedtime procrastination; (6) accomplishment: to gain accomplishment through bedtime procrastination and (7) pleasure: to gain pleasure through bedtime procrastination.

2.7. Measures

2.7.1. Baseline questionnaires

Demographic Information. Demographic data were collected at baseline. Participants completed the following basic demographic questions: gender, age, education, marital status, employment status.

Morningness-Eveningness Questionnaire (MEQ). The Morningness-Eveningness Questionnaire (MEQ) was developed by Horne and Östberg (1976) [23]. In this study, the Korean version of MEQ validated by Lee and colleagues (2014) [24] was used. The MEQ consists of 19 items and total scores range from 16 to 86, with scores above 59 classifying individuals as morning type, scores ranging from 42 to 58 as intermediate type, and scores below 41 as evening type. Cronbach's α for the MEQ was 0.61 in this study.

2.7.2. Primary outcome measures

The Bedtime Procrastination Scale (BPS). The BPS was developed by Kroese and colleagues (2014) [1] and measures the degree of bedtime procrastination. We used the validated Korean version for this study [25]. The BPS consists of 9 items that describe sleep-related behaviors and habits that reflect level of bedtime procrastination. The items are rated on a five-point Likert scale from 1 (almost never) to 5 (almost always). The BPS total score range is 9–45 points, with higher scores reflecting higher levels of bedtime procrastination. The Cronbach's α was 0.54 in this study.

Sleep diary. Participants were asked to keep a sleep diary for four weeks after the first visit. They were asked to record bedtime procrastination duration (BPD), which was operationally defined as the difference from the time initially planned to go to bed and lights off (LO). Additional sleep parameters such as sleep onset latency

(SOL), wake after sleep onset (WASO), total sleep time (TST), time in bed (TIB), bedtime (BT), light off time (LO), wake time (WT), sleep efficiency (SE), sleep quality (SQ), duration of bedtime to light off time (BT-LO) and feeling refreshed upon awakening (FRESH, scale 1–5) were also collected.

2.7.3. Secondary outcomes measures

The Insomnia Severity Index (ISI). The ISI was developed by Bastien, Vallières, and Morin (2001) [26] and is composed of 7 items that measure the severity of insomnia during the past two weeks. The ISI is rated on a five-point Likert scale from 0 to 4. The total score range is 0–28, and higher scores reflect greater insomnia severity. In the present study, participants who scored above 15 were excluded [27]. Cronbach's α was 0.41 in this study.

The Epworth Sleepiness Scale (ESS). The Epworth sleepiness scale (ESS) consists of 8 items that measure excessive daytime sleepiness. This scale was developed by Johns (1991) [28] and the items are rated on a 4-point Likert scale from 0 to 3. The total score ranges from 0 to 24 points, and higher scores reflect higher levels of daytime sleepiness. Cronbach's α for the ESS was 0.72 in this study.

Center for Epidemiologic Studies Depression Scale (CES-D). The CES-D is a self-report questionnaire measuring depressive symptoms over the past 7 days [29]. This scale consists of 20 items, and is rated on a 4-point scale ranging from 0 (rarely or none of the time) to 3 (most or all of the time). Total scores range from 0 to 60 points, with scores above 24 being classified as severe. Cronbach's α was 0.82 in this study.

2.8. Statistical analysis

Analyses followed the intention-to-treat principle that includes all available data from all randomized participants. Baseline characteristics were investigated with an independent sample *t*-test for continuously distributed variables and a chi-square test for categorical variables.

A linear mixed model analysis (LMM) fitted with full information maximum likelihood estimation [30] was used to analyze changes between the treatment group and the wait-list control group at pre-intervention and post-intervention points. Mixed models include non-independence among repeated measures data and models individual differences in change over time by including person-specific growth parameters (i.e., random effects). In this study, the best model was determined using commonly used procedures for linear mixed-effects models. In this model, both the fixed effects (group average effects) and random effects (within and between individual variability) were included. For the comparison of the treatment group and wait-list controlled group, the fixed effects included the linear effect of group, time, and a group by time interaction. Furthermore, a COVID variable was also included in this model as a fixed effect to verify that format of the sessions due to the COVID pandemic did not affect the intervention. The error terms were held equal across time for all analyses. In this study, the primary interest was the group X time effect.

The maintenance of treatment effect was assessed using simple marginal contrasts comparing outcomes three-time points that pre-assessment, post-assessment, and 1-month follow-up. In addition, the treatment effect for the wait-list control group was assessed using simple marginal contrasts comparing outcomes before intervention to post-intervention.

In addition, the post-assessment mean score and standard deviation of the two groups were used to estimate the effect size (Cohen's *d*) of the BED-PRO intervention on reducing bedtime procrastination behavior.

A qualitative analysis of the function of bedtime procrastination was conducted in both the treatment group and the wait-list control group to estimate the function of bedtime procrastination. A total of 43 participants, after excluding two members who dropped out of the intervention, were used for the final analysis. The functions were classified into 7 categories. Subsequently, each participant's functional areas were coded 1 if they had the function and were coded 0 if they did not have the function. Finally, a frequency analysis of multiple responses was performed to estimate the function of bedtime procrastination. All analyses were conducted with SPSS version 21.0 (IBM Corp., Armonk, NY, USA).

3. Results

3.1. Demographic information

A total of 60 participants were enrolled in this study (see Fig. 1 for study flow). The average age of the participants was 21.33 years (SD = 2.35), and 86.7% of the sample was female. Demographic information is presented in Table 1. There were no significant differences between groups on any of these demographic variables. Preliminary inspection of the data revealed no outliers or significant differences on any of the primary outcome measures at baseline to indicate a need for entering additional covariates into the analytic model.

3.2. Primary outcomes

Mean and standard deviation of the treatment group and the wait-list control group at pre- and post-assessment, including 1-month follow-up are presented in Table 2.

The results of LMM are presented in Table 3. There was a significant group × time interaction for the bedtime procrastination scale (BPS; $p < .001$), such that the treatment group showed significantly greater rates of reduction on the BPS (35.56%) compared to the wait-list control group (1.85%, d = 2.19). The treatment group had an average reduction in BPS score of 12.98 from pre-assessment to post-assessment, compared to the wait-list control group who had an average reduction of 0.67 (see Fig. 2). Post hoc comparisons were conducted for the treatment group compared to wait-list control group. As a result, the treatment group had an average reduction in BPS score of 12.98 points, which was significantly greater than the wait-list control group ($p < .001$).

There was also a significant group × time interaction for bedtime procrastination duration (BPD) based on the sleep diary comparing the average effects of the treatment group to the wait-list control group (p = .002), such that the treatment group showed significantly greater reduction in BPD (46.29 min) compared to the wait-list control group (4.55 min; d = 1.22; see Fig. 2). Post hoc comparisons showed that the treatment group had an average reduction in BPD of 46.29 min, which was significantly greater than the wait-list control group ($p < .001$)

There was also a significant group × time interaction for sleep efficiency (SE; d = 1.25) and duration of bedtime to lights off (BT-LO; d = 1.13).

3.3. Secondary outcomes: sleep and clinical outcomes

There were no significant interaction effects for the following sleep parameters: SOL, WASO, TST, TIB, BT, LO, or WT. However, the intervention had a significant effect for time spent between BT and LO (p = .002) and time spent between wake time and time out of bed (p = .036).

There was a significant group × time interaction for ISI scores (p = .001), such that the treatment group showed significantly greater rates of reduction in ISI (38.54%) compared to the wait-list control group (10.49%; d = 1.17). Post hoc comparisons were

Table 1
Baseline characteristics (N = 60).

	Total(N = 60)	TRT(n = 32)	CTRL(n = 28)	$x^2/t(p)$
Gender				
Female	52(86.7%)	28(87.5%)	24(85.7%)	.041(.839)
Male	8(13.3%)	4(12.5%)	4(14.3%)	
Age	21.33(±2.35)	21.06(±2.50)	21.64(±2.18)	−.951(.245)
Education				
High school graduation	2(3.3%)	1(3.1%)	1(3.6%)	1.229(.746)
University student	51(85.0%)	28(87.5%)	23(82.1%)	
Bachelor' degree	7(11.7%)	3(9.4%)	4(14.3%)	
Marital status				
Sigle	60(100.0%)	32(100.0%)	28(100.0%)	–
Employment status				
Unemployed	3(5.0%)	–	3(10.7%)	7.692(.104)
Student	53(88.3%)	30(93.8%)	23(82.1%)	
Employed	4(6.7%)	2(6.3%)	2(7.2%)	
Circadian types (MEQ)				
Neither type	12(23.5%)	9(32.1%)	3(13.0%)	2.560(.110)
Evening type	39(76.5%)	19(67.9%)	20(87.0%)	
(Missing)	9(15.0%)	4(12.5%)	5(17.9%)	

TRT, Treatment group; CTRL, Control group.

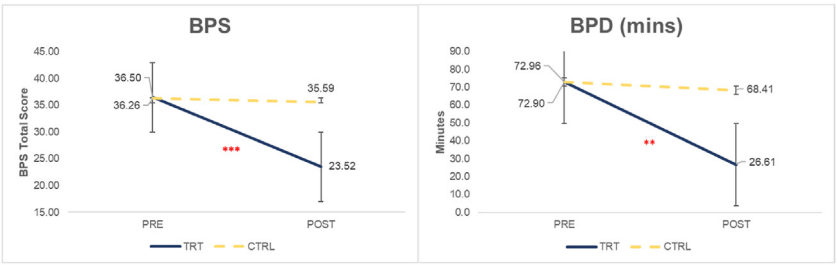
Table 2
Means of the TRT and CTRL groups by time point (N = 46).

		TRT			CTRL			Effect Size(Cohen's d)
		T1	T2	T3	T1	T2	T3	
		M(SD)	M(SD)	M(SD)	M(SD)	M(SD)	M(SD)	
Questionnaire	BPS	36.50(3.24)	23.52(6.54)	24.62(5.20)	36.26(3.89)	35.59(4.18)	23.60(5.10)	2.19
	ISI	10.25(2.86)	6.30(2.12)	5.48(2.02)	10.87(2.67)	9.73(3.61)	6.75(3.93)	1.17
	ESS	9.39(3.25)	6.00(3.30)	5.29(2.72)	7.22(3.95)	7.64(4.27)	6.20(4.57)	0.43
	CES-D	13.89(7.28)	11.00(9.28)	10.67(8.17)	13.39(5.48)	14.55(8.33)	10.70(6.73)	0.40
Sleep diary	BPD (min)	72.90(57.24)	26.61(24.57)	24.44(21.12)	72.96(33.91)	68.41(41.88)	32.84(33.42)	1.22
	SOL (min)	11.64(12.06)	7.93(6.45)	8.66(8.67)	12.87(9.19)	8.02(7.05)	12.53(10.32)	0.01
	WASO (min)	4.23(4.08)	4.65(5.93)	3.42(3.57)	6.65(7.13)	9.46(17.49)	8.08(14.68)	0.37
	BT-LO (min)	35.40(32.31)	11.50(14.08)	18.22(21.08)	54.22(56.38)	58.34(57.17)	22.16(26.39)	1.13
	TST (min)	406.38(48.54)	435.67(49.78)	448.02(73.37)	414.04(60.35)	433.31(57.20)	423.24(62.52)	0.04
	TIB (min)	472.66(70.87)	473.14(55.63)	496.51(71.62)	499.50(87.15)	525.51(89.92)	481.81(77.46)	0.70
	SE (%)	86.71(7.57)	92.41(4.03)	90.38(4.87)	83.91(9.21)	83.62(9.07)	88.06(6.91)	1.25
	SQ	3.38(0.61)	3.79(0.58)	3.72(0.59)	3.26(0.71)	3.45(0.79)	3.42(0.66)	0.49
	FRESH	2.91(0.59)	3.54(0.63)	3.46(0.62)	2.87(0.59)	3.10(0.84)	3.25(0.68)	0.59
	BT	2:07:58(1:26:43)	1:52:01(0:59:37)	1:50:40(0:54:46)	1:51:15(1:07:42)	1:21:20(1:05:53)	1:46:25(0:52:39)	0.49
	LO	2:43:38(1:22:40)	2:03:50(1:02:33)	2:09:03(0:57:24)	2:45:37(0:53:16)	2:19:54(1:17:40)	2:08:41(1:00:28)	0.22
	WT	9:43:47(1:12:42)	9:32:05(1:20:21)	9:49:09(1:13:29)	9:59:10(1:11:00)	9:59:48(1:22:29)	9:32:33(1:19:56)	0.34
	WT-TOB (min)	16.85(16.42)	13.06(10.35)	18.04(13.79)	11.58(8.90)	16.15(17.28)	15.68(10.79)	0.22

TRT, Treatment group; CTRL, Control group.T1, Pre-Assessment; T2, Post-Assessment; T3: Follow Up-Assessment for Treatment group and After Intervention Assessment for Control group.
Effect Size was calculated by comparing TRT and CTRL in T2.BPS, bedtime procrastination scale; ISI, insomnia severity index; ESS, Epworth sleepiness scale; CES-D, Center for epidemiologic studies depression scale; BPD, bedtime procrastination duration; SOL, sleep onset latency; WASO, wake after sleep onset; BT-LO, duration of bedtime to light off time; TST, total sleep time; TIB, time in bed; SE, sleep efficiency; SQ, sleep quality; FRESH, feeling refreshed upon awakening; BT, bedtime; LO, lights off; WT, wake time; WT-TOB, morning procrastination.

Table 3
Linear mixed models comparing treatment effects in the TRT and CTRL groups for primary outcomes (N = 60).

	Variable	Effects	Estimate	*SE*	*t*	*p*
Questionnaire	BPS	GROUP	−12.06	1.65	−7.31	.000***
		TIME	1.20	1.36	0.89	.379
		GROUPxTIME	12.00	1.89	6.34	.000***
	BPD (min)	GROUP	−43.55	12.32	−3.54	.001**
		TIME	4.55	9.45	0.48	.632
		GROUPxTIME	43.28	13.28	3.26	.002**
	SE (%)	GROUP	9.08	2.27	3.99	.000***
		TIME	0.28	1.26	0.22	.824
		GROUPxTIME	−6.34	1.78	−3.56	.001**
	WT-TOB (min)	GROUP	−3.36	4.04	−0.83	.407
		TIME	−4.56	2.90	−1.57	.123
		GROUPxTIME	8.82	4.08	2.16	.036*



TRT, Treatment group; CTRL, Control group.
PRE: Pre-Assessment; POST: Post-Assessment for treatment group or After Intervention Assessment for Control group
BPS, bedtime procrastination scale; BPD, bedtime procrastination duration.

Fig. 2. Changes in BPS and BPD for TRT and CTRL groups
TRT, Treatment group; CTRL, Control group.PRE: Pre-Assessment; POST: Post-Assessment for treatment group or After Intervention Assessment for Control groupBPS, bedtime procrastination scale; BPD, bedtime procrastination duration.

conducted for the treatment group compared to wait-list control group. As a result, the treatment group had an average reduction in ISI score of 3.95 points, which was significantly greater than the wait-list control group ($p < .001$).

Significant group × time interactions were also found for the Epworth sleepiness scale (ESS; $p < .001$), with the treatment group showing significantly greater rates of reductions compared to the wait-list control group ($d = 0.43$). There were no significant group × time interactions for CESD scores ($p = .051$).

3.4. Treatment maintenance effects

The treatment group's pre-post assessment scores and one-month follow-up scores were compared to determine whether the treatment effect was maintained at one-month (see Supplemental Table S2). Participants in the treatment group reported an average of 11.88 points decrease in BPS from pre-assessment to the one-month follow-up ($p < .001$). Participants in the treatment group also reported an average of 48.46 min decrease in BPD from pre-assessment to one-month follow-up ($p = .001$). Thus, both BPS and BPD were maintained at one-month follow-up following the intervention.

For SE, there was an average of 5.70% increase from pre- and post-assessment ($p = .007$). However, there was no significant difference between the pre-assessment and one-month follow-up ($p = .168$).

For secondary outcomes, significant differences were also found. For ISI, there was an average of 4.77 points decrease from pre-assessment to one-month follow-up ($p < .001$). Participants reported an average reduction of ESS score of 4.10 from pre-assessment to the one-month follow-up ($p < .001$). However, there were no significant difference in CES-D from pre-assessment to one-month follow-up ($p = .479$).

3.5. Treatment effects for the wait-list control group

After the control period, the wait-list control group received the BED-PRO intervention, identical to the treatment group (see Supplemental Table S3). Significant changes were found for BPS with participants in the wait-list control group reporting an average reduction of 11.99 points from pre-intervention to post-intervention ($p < .001$). Significant changes were also found BPD (based on the sleep diary), with participants in the wait-list control group reporting an average reduction of 35.57 min from pre-intervention to post-intervention ($p = .004$). A significant change was also found for ISI scores ($p = .015$) and BT-LO ($p = .001$). However, there were no significant differences for SE, ESS, and CES-D.

3.6. Treatment fidelity

The intervention was provided as a manualized treatment. Utilizing an experimenter-derived fidelity checklist of 23 essential a behavioral intervention for reducing bedtime procrastination (BED PRO) components, the recorded tapes of Sessions 1–3 (all of tapes across all clients and therapists) were rated by two individuals who coded the presence/absence of components. As a result, average kappa interrater reliability was 0.499 (99.6% agreement), indicating moderate level of agreement [20].

3.7. Functional analysis

Qualitative analysis of the function of bedtime procrastination was performed in both the treatment group and the wait-list control group. Data from 43 participants were used for analysis, but since duplicate responses were allowed, the total frequency of responses was 83. The sum of the response ratios for each item was 193%. Among the total frequency of responses, emotional regulation (31.3%) and rewards (26.5%) showed the most frequent responses for engaging in bedtime procrastination. These results are presented in Table 4.

4. Discussion

This randomized controlled trial (RCT) for 60 young adults aimed to verify the efficacy of an intervention to reduce bedtime procrastination. In addition, the function of bedtime procrastination was investigated in this study. The main results and implications are as follows.

4.1. Treatment effects of the intervention

The main results of the study indicated that a behavioral intervention to reduce bedtime procrastination was efficacious in a non-clinical sample. This was true for both scores on the BPS measure and on sleep diaries. There was a 35.56% reduction on the BPS in the treatment group compared to the control group. Additionally, BPD, operationally defined as the discrepancy between desired bedtime and actual bedtime based on sleep diaries, was reported to be an average 72.90 min per day in the treatment group, which decreased by 26.61 min−24.44 min following the intervention (66.47% decrease). These effects were maintained until the one-month follow-up. Furthermore, these decreases of BPS and BPD were also found in the wait-list control group when they received the same intervention. To date, this is the first psychological intervention developed to specifically target bedtime procrastination.

Table 4
Function of bedtime procrastination (N = 43).

	n(%)	Percentage of cases (%)
Function		
Emotion regulation	26 (31.3)	60.5
Rewards	22 (26.5)	51.2
Social interaction and Belongingness	15 (18.1)	34.9
Acquisition of information and knowledge	11 (13.3)	25.6
Sleep inducing	5 (6.0)	11.6
Accomplishment	2 (2.4)	4.7
Pleasure	2 (2.4)	4.7
Total	83 (100.0)	193

The intervention in our study also demonstrated reductions for insomnia severity and daytime sleepiness. These reductions might have been caused by having more regular sleep schedules as a result of the intervention, similar to previous studies [31,32]. In addition, previous studies have suggested that media use before bedtime was associated with an increase in insomnia severity [33–36]. A significant difference in duration between bedtime to lights off (BT-LO) was found between the treatment group and the wait-list control group. This result suggests that the treatment group, which received the intervention, had significant reductions in the time window between getting into bed and intending to sleep, which is often when many individuals in the study reported engaging in bedtime procrastination. Future studies are needed to investigate whether this reduction in postponing sleep intention directly correlates with decrease in electronic media use. In addition, further investigation of whether individuals who participated in the intervention were more likely to increase their self-regulation of getting to bed at an optimal time, or improve their emotion regulation skills through the intervention, which may have helped make the decision to attempt to sleep earlier.

The intervention also had an effect on sleep efficiency, but the effects were not maintained at follow-up. SE increased 4.23% following the intervention, but baseline to one-month follow-up was not significant. These results suggest that perhaps beneficial changes due to a sleep intervention may require longer sessions to confer to a long-term habit. Literature from health psychology suggests a discrete habit takes 18 days to 36 weeks to form, which may be a reason the change was not maintained through follow-up (Harvey et al., 2021) [37].

The intervention did not yield significant changes in other sleep parameters, including SOL, WASO, BT, LO, WT, or TST. The intervention, while helpful in decreasing bedtime procrastination, did not help the participants sleep earlier or more. The reason for these results might be that this study was conducted with non-clinical individuals without sleep disorders, in a sample of young adults who most likely had flexible sleep schedules. Indeed, WASO was already very low during the baseline period (below 10 min in both groups). Other sleep parameters were also within normal range for non-clinical populations. It is possible that the psychological intervention, which was efficacious in decreasing bedtime procrastination, may work to decrease avoidant behavior associated with negative emotions before bedtime, thus facilitating better emotional regulation/resolution of negative emotions prior to bedtime. This is possible considering the main function of bedtime procrastination was found to be emotion regulation. Thus, this raises the question as to whether the concept "bedtime procrastination", which rather insinuates a negative connotation, is the correct term for the behavior if it is aiding certain individuals to get into an optimal emotional place to sleep. Further studies on the mechanisms of the intervention will be needed.

These results are consistent with previous studies [9], and the reason for these results might be that this study was conducted with young adults in their 20s. Early adulthood is known to have more evening types than morning types [23], as circadian rhythms are delayed compared to other age groups [38]. Furthermore, the reason for these results might be that most participants were young adults who had flexible sleeping schedules which allowed them to wake up late. These participants went to bed quite late and arose quite late, particularly in comparison to other adults who may have full-time jobs during the day.

While the study was conducted in individuals free of sleep disorders and other psychopathology, the study has clinical implications. In a previous study that conducted cognitive behavioral therapy for insomnia (CBT-I), results indicated that CBT-I alone was effective in reducing insomnia symptoms but not effective in reducing bedtime procrastination [17]. This suggests that more targeted approach for bedtime procrastination may be needed as an adjunctive module in clinical settings, especially for insomnia patients. While past studies of bedtime procrastination have viewed the behavior as a phenomenon, past studies from our research team and results from the functional analysis of this study suggest the importance of approaching bedtime procrastination as a sleep-interfering health behavior that merits attention through interventions [9]. Future studies comparing the treatment effects of evidence-based treatments (e.g., CBT-I) with adding a bedtime procrastination adjunctive module may be useful in ascertaining whether treatment effects can be enhanced for existing treatments.

4.2. Functions of bedtime procrastination

The most frequent function of bedtime procrastination was emotion regulation. This suggests that individuals engage in bedtime procrastination to reduce negative emotions or to avoid situations/thoughts/emotions that cause negative emotions. These results are consistent with the latest findings in studies of general procrastination that general procrastination may occur in order to find pleasure or avoid negative emotions [12,13,22]. Moreover, a recent study suggested that bedtime procrastination may be related to emotional stress [39]. This study revealed that COVID-19 related emotional stress was positively associated with bedtime procrastination. Furthermore, the association could be mediated by negative affect moderated by rumination [40]. These findings indicate that when experiencing high levels of emotional stress, such as those occurred by COVID-19, individuals may have a greater need to recover from the stressful day, and therefore have higher likelihood of engaging in bedtime procrastination.

A second frequent function of bedtime procrastination was rewards. This suggests that individuals may delay their bedtime when they feel they deserve some time for themselves and reassert control over their busy schedule to allocate time for themselves. These results are consistent with a previous study that demonstrated “deliberate procrastination”, by conducting a qualitative study to investigate the reason for bedtime procrastination [21]. A possible explanation for this type of bedtime procrastination is that

individuals may delay bedtime to recover their self-regulatory ability, which may have become depleted during the day. A previous study reported that individuals who believed their willpower was a limited resource that gets easily depleted (limited theory) may show differences in bedtime procrastination compared to individuals who believe their willpower remains regardless of their previous behaviors exerting self-control (non-limited theory). Based on this study [40], participants with limited theory procrastinated on going to sleep on stressful days, while participants with non-limited theory did not [41]. In addition, another previous study suggested that exerting self-control decreases willpower resources and triggers the goal to rest, and leads to more resting behavior in people with limited theory [42]. Iindividuals with limited theory may delay their bedtime by feeling they deserve "having 'me' time" or "reward myself" to recover their willpower, which has been depleted during the busy day. This can also be said to be similar to the recently proposed concept of "revenge bedtime procrastination".

Finally, a third frequent function of bedtime procrastination was social interaction and belongingness. According to our previous study that investigated the usage patterns of smartphone applications in the population who engaged in bedtime procrastination, the most frequently used smartphone functions before bedtime were leisure and communication [15]. These results are consistent with the previous study, and suggest that lack of social connectedness may have an effect on sleep behaviors [43–45]. These results suggest that bedtime procrastination can have varied functions for each individual, highlighting the importance of identifying the unique function of bedtime procrastination for each individual to tailor and personalize sleep interventions.

4.3. Limitations and recommendations for research

This study has some limitations. First, this study recruited fewer cases for men than for women. Since the sample consists mostly of women, the need for additional recruitment of male participants is raised.

Second, the study used a waitlist control group to verify the effectiveness of the intervention. In previous studies, study designs using a waitlist control group has been noted to overestimate intervention effects, even though this design has ethical advantages in that it allows for the provision of care (if delayed) to participants who are seeking help. Therefore, the effects of the study may have been overstated. Thus, future studies using active control designs may be a better alternative to further test intervention effects.

Third, this study proved the efficacy of the behavioral intervention developed to specifically target bedtime procrastination through comparison with the treatment group who receive the behavioral intervention and the wait-list control group who did not receive an intervention. Therefore, this study cannot completely exclude the possibility of the study participants recognizing which group they belonged to. Furthermore, there are limitations in generalizing that the significant difference between the treatment group and the wait-list control group is incurred by the efficacy of the behavioral intervention.

Fourth, in this study, objective measurement for sleep-related variables were not provided. The primary outcomes of this study are bedtime procrastination duration (BPD), which was operationally defined as the difference between the time originally planned to go to bed and the lights off (LO). However, wearable devices such as actigraphy have limitations in that they overestimate sleep, especially when individuals spend excessive time in bed awake [46]. In addition, individuals who engaged in bedtime procrastination were delayed going to sleep by engaging in activities not only before they are going to bed but also while they are already in bed [47]. Therefore, in future studies, it needs to measure objective measurements to estimate changes in sleep variables including bedtime procrastination.

Fifth, this study was studied primarily in young adults with flexible sleep schedules. These participants went to bed and arose quite late, particularly in comparison to other adults who may have full-time jobs during the day. Currently, little is known about how prevalent the issue is in different subpopulations. In the context of developmental changes, bedtime procrastination may be characterized differently in individuals with children, or after retirement. It would be helpful to expand this intervention out to other developmental groups.

Finally, this study was conducted with individuals without psychopathological disorders and sleep disorders. Therefore, there is a limit to generalizing the results of this study to clinical groups. In future studies, it is necessary to verify whether the behavioral intervention developed to specifically target bedtime procrastination is effective in the clinical group as well as in the non-clinical group. Moreover, it is to be a meaningful study if treatment modules applicable to clinical groups will be developed and verified.

5. Conclusion

Despite the above limitations, this study has the following contributions. First, this pilot study verified the efficacy of the behavioral intervention that was developed to decrease bedtime procrastination with a small sample of young adults for the first time. The results of this study demonstrated that the behavioral intervention improves not only bedtime procrastination but also insomnia severity, daytime sleepiness, sleep efficiency, and depression in non-clinical individuals.

Second, this study investigated the functions of bedtime procrastination and suggests that the most common function is emotion regulation. In addition, this study confirmed that bedtime procrastination has various functions for individuals. This is meaningful in providing the underlying data to improve the effectiveness of the behavioral intervention by understanding the mechanism for target behavior. In sum, this study was the first study to consider bedtime procrastination as a target for intervention.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2023.06.001.

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