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Authors
Gordon, Amie M
Del Rosario, Kareena
Flores, Abdiel J
et al.

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Authors: Amie M. Gordon, Kareena Del Rosario, Abdiel J. Flores, Wendy Berry Mendes, Aric A. Prather

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Bidirectional Links Between Social Rejection and Sleep

Amie M. Gordon, PhD
Kareena Del Rosario, BA
Abdiel J. Flores, BA
Wendy Berry Mendes, PhD
Aric A. Prather, PhD

1Department of Psychiatry, University of California, San Francisco
2Department of Psychology, Columbia University

Author’s Information:
Address correspondence to: amie.gordon@ucsf.edu or aric.prather@ucsf.edu

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Abstract

**Objective:** This set of studies examines the bi-directional links between social rejection and sleep, a ubiquitous and increasingly problematic health behavior.

**Methods:** In Study 1, a multi-day field experiment, 43 participants completed a neutral task just prior to sleep on night 1 and a social rejection task on night 2. Objective and subjective sleep, post-rejection affect, and physiological responses were measured. In Study 2, 338 participants reported typical sleep quality prior to coming to the laboratory where they received social rejection or social acceptance feedback from a stranger. Physiological and affective responses were measured throughout the session.

**Results:** In Study 1, after social rejection, participants took longer going to bed \([M(\text{SD}) = 38.06(48.56) \text{ vs. } 11.18(15.52), t(42) = 3.86, p < .001]\) and had shorter sleep durations \([6:46(1:27) \text{ vs. } 7:19(1:38), \text{ } t(41) = 2.92, p = .006]\) compared to the baseline night. Trait rumination moderated these effects, with high ruminators taking the longest to go to bed post-rejection \(t(38) = 2.90, p = .006\). In both studies, there was (inconsistent) evidence that sleep influences reactions to rejection: some sleep measures predicted physiological reactivity during the rejection task in Study 1 and greater negative affect after social rejection in Study 2.

**Conclusions:** These studies provide evidence that social rejection may affect sleep outcomes, particularly for trait ruminators, and poor sleep in turn may exacerbate affective responses to
social rejection. Given the mixed findings, small sample size, and no active control condition more work is needed to confirm and build on these findings.

**Keywords:** Social Rejection, Sleep, Physiology, Affect, Rumination

**Abbreviations:** IBI = Interbeat Interval; HRV = Heart Rate Variability; PSQI = Pittsburgh Sleep Quality Index; RSA = Respiratory Sinus Arrhythmia; SOL = Sleep Onset Latency
INTRODUCTION

Social rejection plays an important role in our waking lives, influencing health and well-being. Humans are a social species with a strong need to belong; thus, rejection can signal a threat to survival (1). When faced with rejection, people experience both mental and physical distress (2), including greater negative affect (1) and increased cardiovascular, hormonal, and inflammatory responses (3-6). Although much work has focused on the mental and physical consequences of rejection, little research has linked it with sleep, a ubiquitous health behavior with major social, physiological, and affective consequences (7,8). Evidence from both the rejection and sleep literatures suggest that these two phenomena might influence each other.

Sleep and Health

Sleep problems are on the rise, with more than 69% of adults getting less sleep than they need (9). When people sleep poorly, there are numerous physical and mental health consequences, from increased mortality, cardiovascular disease, and depression to decreased quality of life and productivity (7). Given the consequences of insufficient sleep, researchers and health officials are calling for attention to be paid to understanding processes linked to sleep. Recently, we highlighted the need to consider the links between sleep and social processes (8). A handful of studies have shown that interpersonal processes, particularly negative ones, influence sleep. For example, people subject to more racial discrimination have more disrupted sleep and greater daytime fatigue (10-12) and lonely individuals spend more time awake at night (13,14) and report lower sleep quality (15). In work on close relationships, researchers have found that
people sleep worse after interpersonal conflict (16), and sleep better when feeling connected and cared for (17,18). Most relevant to the current studies, research on social stress has shown that anticipation of a social stressor, such as giving a speech to a panel of evaluators, leads to disrupted sleep (19). Building on this nascent literature, we anticipated that experiences of social rejection would disrupt sleep.

The Effect of Social Rejection on Sleep

Which aspects of sleep are affected by social rejection? Findings from the literature on sleep and negative social processes point to delayed sleep, more time awake after sleep onset, more fragmented sleep, and worse subjective sleep quality (20,21). From the rejection literature, research has shown that social rejection impairs the ability to self-regulate (22,23), which is essential for high-quality consistent sleep and good bed-time behavior (i.e, not delaying bed-time) (24). In addition, the physiological arousal caused by social rejection may delay sleep and affect sleep quality (particularly people’s perceptions of their sleep) (25,26). Rejection also leads to a variety of physiologically-arousing negative emotions, such as shame, loneliness, anxiety, and anger (27), all of which have been linked to disrupted sleep (28). People often experience these emotions, particularly shame, right before bed, a time when people frequently review their actions and experiences from that day (28,29). Taken together, this literature suggests that after rejection, people may be particularly likely to take longer to go to bed and fall asleep, and once asleep, they may have more fragmented sleep and poorer perceived sleep quality upon awakening.
Individual Differences in Responses to Rejection

Are some individuals particularly susceptible to the effects of social rejection on sleep? Several studies point to the likelihood that people who are higher in rumination—a tendency to passively and repetitively focus on the self and negative affect—will show the strongest effects of social rejection on sleep. Trait ruminators have worse sleep outcomes, and this seems to be particularly true after experiencing a social stressor (30-32). Linking rejection and rumination, work has shown that individuals sensitive to rejection increase their ruminative thinking over time (33), and that rumination is one pathway through which loneliness influences sleep quality (34). Thus, we expected that individuals prone to rumination would be the ones who had the most disrupted sleep following social rejection.

The Effect of Sleep on Social Rejection

Research points to a bi-directional link between sleep and social processes (8). Research has shown that people who suffer from sleep deprivation experience more negative affect, are more reactive to negative stimuli (36), and have greater physiological arousal during social stressors (37,38). Therefore, we expected to find evidence of a reverse link between rejection and sleep, such that participants who slept poorly prior to experiencing a social rejection would have stronger affective and physiological responses to the rejection experience.
Present Research

Our primary aim was to test the effects of social rejection on sleep outcomes. We did so by utilizing a multi-day within-person field-experiment design in which we compared both objectively- and subjectively-measured sleep after a social rejection task compared to a neutral task.

To explore the physiological underpinnings of these effects, we tested whether people experienced greater physiological reactivity during and after the social rejection manipulation. We also tested whether increased physiological arousal predicted sleep outcomes. In particular, we focused on arousal in the autonomic nervous system, in line with prior work linking autonomic activation to both rejection and sleep. In addition, in line with the prior work on rumination, we tested whether trait ruminators showed the strongest effects of rejection on sleep.

We also explored the reverse association by testing whether poor sleep prior to a social rejection influenced affective and physiological responses during the rejection in both our field experiment (Study 1) and an experimental laboratory study (Study 2).
Study 1: Sleep and Social Rejection Field Study

Method

Participants

Forty-seven adults from the San Francisco Bay area participated in exchange for monetary compensation. Four participants did not complete all necessary parts of the study, leaving a sample of 43 (27 female, 16 male; 19 African Americans; 24 European Americans; $M_{age} = 27, SD = 4.75, Range = 19-34$). Participants were eligible if they (a) spoke English as their primary language, (b) lived in the United States since the age of 7, (c) had no history of psychiatric or physical disease or disorder, and (d) were not on any medications that interfere with cardiovascular responses (e.g., beta blockers) or sleep (e.g., using sleep aids more than twice per week). Data collection started in January 2013 and ended in June 2016. Study 1 and Study 2 were both approved by the UCSF Institutional Review Board (IRB#s: 12-09286 and 11-05791, respectively).

Procedure

Participants were recruited from a laboratory mailing list to participate in a 6-day study. After being deemed eligible via a phone screening, participants set up a time for experimenters to come into their homes. Two Research Assistants (RAs) went into participants’ homes at four separate times across six days (Days 1, 2, 3 and 6). On Day 1, participants completed consent
and an initial health intake questionnaire. RAs then trained participants on the study tasks and equipment. For the entire study, participants wore actigraphs that measured their rest and activity. On Days 1 and 2, participants wore heart rate monitors that captured their beat-to-beat intervals during the study tasks and throughout the night. Study tasks differed on Day 1 and Day 2. For both nights, participants were instructed to log onto the computer and begin the task 30-45 minutes before their typical sleep time. To control for spending time on a computer prior to sleep, on Day 1, participants watched a neutral 20-minute video and then completed a brief quiz about it. At a similar time to when the Day 1 task started, on Day 2 participants completed a series of online activities during which they interacted with other “participants” on-line who rejected them during several activities (the other “participants” were confederates). These activities included a cyberball task in which the other two “participants” stopped throwing a ball to the participants during an online ball-throwing game (39) and a speech delivery task where the participant was instructed to give a speech on his/her strengths and weaknesses and then answer a variety of topical questions (40). Throughout the speech task, the other two “participants” made negative comments via online chat every 10-15 seconds (these comments were chosen from a set of predetermined responses—e.g., “you are saying ‘um’ a lot;” “try to be clearer, you don’t make any sense”).

Participants were randomly assigned to be rejected either by members of their own race or members of a different race, but given the small N per cell, we did not analyze the data based on this distinction and, instead, in an initial set of analyses we entered confederate race (same-versus cross-race) as a covariate. The presence of this covariate did not change any of the effects
substantially. Final analyses do not include race as a covariate (data are available on-line including race: https://osf.io/mg264/?view_only=e433779d518249b291941bb509df9d2b).

Other activities not relevant to the current paper included completing brief questionnaires each night and reaction time tasks each morning. On Day 3, the RAs visited the participants to pick up the computer and heart rate equipment. For Days 3-6, participants completed brief sleep questionnaires each morning. On Day 6, actigraphs were collected by research assistants and participants were debriefed and compensated.

Measures

**Background Questionnaires**

As part of an online prescreening, participants completed demographics as well as a series of background questionnaires. Relevant to these analyses, participants completed the **Pittsburgh Sleep Quality Index** (PSQI; 41), which assesses sleep over the prior month using seven components of sleep: sleep duration, sleep efficiency, sleep quality, sleep disruptions, sleep onset latency, daytime dysfunction, and use of medicine. Scores for each component range from 0 (better) to 3 (worse), and were summed to create a global sleep composite (possible range 0–21). In this sample, $M = 4.79$, $SD = 2.56$, $range = 0$ to 10. Participants also completed the **Rumination-Reflection Questionnaire** (42). We focused on the rumination subscale which includes 12 items such as, “I tend to ‘ruminate’ or dwell over things that happen to me for a really long time afterward” and “Sometimes it is hard for me to shut off thoughts about myself.”
Items were reported on a 6-point scale (0= Strongly Disagree, 5=Strongly Agree) and combined into a single scale, $M = 2.52$, $SD = .92$, with $\alpha = .89$.

**Sleep**

Objective sleep times were collected via wrist actigraphy using Actiwatch-2 devices (Philips Respironics) which were worn on the nondominant wrist. Wrist actigraphs collect behavioral data to infer rest/wake times and have been shown to be a reliable and valid measure of sleep (43). Data were collected in 30-sec epochs and sleep patterns were derived using validated Minimitter software algorithms. Bed times and wake times were set via a triangulation method which utilized participant’s reported bed time and wake time from the actigraphy watch, participant’s reported bed time and wake time from the sleep diary, as well as the bed time and wake time calculated by actiware. Actigraphy watch-reported times were prioritized, but when they were missing or more than 30 minutes from the sleep diary times, then the final time was set based on the most reasonable sleep time given the actigraphy data. Objective measures analyzed in this study include: **Sleep duration** (length of sleep period in minutes), and **fragmentation** (an index of sleep discontinuity). We chose not to include sleep onset latency or sleep efficiency given concerns that they are not reliably estimated by actigraph (48). Participants also reported their subjective **sleep quality** each morning (How would you rate the quality of your sleep last night?) on a 4-point scale (Very Bad to Very Good).
Interbeat-Interval and Heart Rate Variability

We obtained interbeat-intervals (IBIs) and heart rate variability (HRV) on Day 1 and Day 2 using the Polar® heart rate monitor wrist watch (Model RS800CX) with a chest strap (44,45). Women were provided a sports bra with embedded chest strap to prevent against the chest strap slipping during sleep. IBIs represents the milliseconds between heartbeats and are influenced by both sympathetic and parasympathetic nervous systems. HRV represents the variability in IBIs (46).

Data were downloaded using polar watch software and the IBI series were extracted. The first and second author visually inspected every bin of data for artifacts and the first and second to last author developed algorithms to identify points that were physiologically implausible (see Supplemental Digital Content for a complete list of rules for cleaning artifacts, http://links.lww.com/PSYMED/A534). Once data were edited, we extracted 1-minute bins. We calculated average IBI and HRV using open-access software, CMetX (47). HRV was operationalized as the natural log of the variance of the IBI time series. This measure includes both sympathetic and parasympathetic influence, though it is highly correlated with RSA during both relaxed and stressful tasks ($r = .84-.90$; 47).

We collected physiological data for five-minutes prior to computer log-on each night as a baseline resting period. Participants were expected to be seated at their computers waiting for instructions during this time. Physiological measures were collected throughout the tasks on both nights. To examine physiological reactivity during the tasks, a baseline score was subtracted
from each minute during the task. If a participant was missing the last minute of baseline (minute 5), minute 4 of baseline was used. If participants were missing minute 4 and 5, we took the closest clean minute of data.

We continued to collect physiological measures after the task, which we categorized as *post-task awake* and *sleep* (after the software determined the participant was asleep). Participants often lost signal during the night, so we cleaned and analyzed the first 20 minutes of sleep data.

*Post-Rejection Affect*

After the social rejection task, participants reported on their *positive* and *negative affect* using the Positive and Negative Affect Schedule (PANAS; 49) plus two additional negative affect items: *Disrespected* and *Alone*. Participants reported on how they were currently feeling using a 5-point scale (1 = *Not at all*, 5 = *A great deal*). Positive items were combined into a single scale ($M = 2.49$, $SD = .87$; $\alpha = .89$), as were negative items, $M = 1.74$, $SD = .63$; $\alpha = .83$.

*Statistical Analyses*

Data analyses were performed using SPSS Statistics Version 25 (IBM Corporation), with significance levels set to $p < .05$ (two-tailed). Using box plots, two participants were identified as outliers for task-to-bed latency. Careful inspection of their sleep data provided no evidence that their scores were not real, thus they are included in reported analyses. However, additional analyses using adjusted scores for participants greater than 2.6 SD above the mean (adjusting
them down to one percent greater than the next smallest value) as well as analyses without these participants produced similar results (these variables are included in the datasets which are available online: https://osf.io/mg264/?view_only=e433779d518249b291941bb509df9d2b).

To examine the effect of nightly task (neutral versus social rejection) on sleep outcomes, we used paired samples t-tests. To examine the effect of task on physiological outcomes, we accounted for the repeated physiological measures within each night and within each individual using multilevel models (MLMs) with auto-regressive covariance structures of the residuals (repeated physiological measures within night and individual) and random intercept and slope for the individual. Given that we had only two nights of data, when examining whether physiological reactivity during the tasks predicted sleep outcomes on each night, we adopted marginal models (nights repeated within participants) with unstructured matrices for the residuals. To examine whether rumination moderated the effect of task on sleep, we used the same marginal model described above, regressing sleep outcomes onto grand-centered rumination, task (neutral = -.5, rejection = .5), and their interaction term. For MLM and marginal models, degrees of freedom are calculated using the Satterthwaite approximation which yields degrees of freedom that are somewhere between the number of repeated measures and the number of individuals.

In our final set of analyses, we examined the reverse association between rejection and sleep, testing whether prior night sleep as well as self-reported sleep over the prior month influenced participants’ physiological and affective responses to the social rejection task. We looked at prior night sleep duration, fragmentation, and self-reported sleep quality. For sleep
over the prior month, we used the global score from the PSQI, with higher scores indicating worse sleep. Our prior analyses were all within-person, making it unnecessary to control for individual difference covariates. These analyses, however, were between-person, presenting the possibility that any effects of prior sleep may be due to demographic differences. Thus, we conducted follow-up analyses controlling for race, gender, age, and BMI. Analyses were conducted using MLM with task physiology on night 2 nested within individuals (with an autoregressive structure for residuals and a random intercept).

Results

Main Effects of Nightly Tasks on Sleep Outcomes

Does social rejection have a negative effect on going to bed and subsequent sleep? As shown in Table 1, after experiencing rejection, people slept for a significantly shorter amount of time relative to a night when they experienced no rejection. This was due to the fact that participants tended to go to bed much later after the rejection compared to the neutral, control night (i.e., there was a significantly longer task-to-bed latency). There was no significant effect of task on fragmentation. However, the moderate effect size for fragmentation ($D = .44$) suggests that people may have more disrupted sleep after social rejection. There was no evidence that social rejection impaired people’s perceptions of the quality of their sleep. In fact, people reported descriptively higher sleep quality following the social rejection compared to the prior night, although the two nights did not differ significantly from each other.
Main Effect of Social Rejection on Physiological Outcomes

Mean differences in physiological outcomes between nights are shown in Table 2 and depicted in Figures 1-4. Participants’ IBI and HRV did not differ during the baseline resting period between the neutral and rejection nights, suggesting no pre-existing differences in physiological activation prior to the nightly tasks (see Figure 1).

Not surprisingly, during the social rejection task participants were significantly more physiologically reactive compared to the neutral (i.e., video-watching) task: participants exhibited significant decreases in IBI relative to baseline ($t(61.1) = -4.19$, $p < .001$), whereas during the neutral task they exhibited small increases in IBI, $t(65.1) = 1.97$, $p = .052$. There was also a significant night by time interaction (see Figure 2), suggesting that the IBI slopes during the two tasks differed significantly from each other, ($F (1, 129.66) = 36.29$, $p < .001$), with participants experiencing decreases in IBI between the cyberball and speech tasks (i.e., greater activation/faster heart rate). HRV decreased during the social rejection task more so than during the video-watching, but the effect did not reach statistical significance. There was no evidence of a night by time interaction for HRV (see Figure 2).

To examine effects of social rejection on post-task physiology, we examined the first hour after participants completed the tasks. As shown in Figure 3, participants were significantly more physiologically activated (shorter IBIs) during the hour after the social rejection task compared to the neutral task. Post-task HRV did not differ significantly across the two nights.
Given that the time between task and bed varied across nights and across individuals, we conducted one final analysis in which we compared the first 20 minutes of sleep between the two nights. As shown in Table 2 and Figure 4, we found no significant differences in physiology during this period, suggesting that by the time participants went to bed each night, they had returned to similar levels of physiological activation.

**Effects of Physiological Arousal on Sleep Outcomes**

There was some evidence that IBI reactivity during the tasks helped explain how long people stayed up after the task. That is, shorter average IBI (greater activation) during the tasks predicted longer (but not significant) durations between task completion and going to bed, \( b = -0.03, t(25.5) = 1.82, p = .079 \). There was no effect of IBI reactivity on duration (\( b = .08, t(32.0) = 1.06, p = .30 \)), or fragmentation, \( b = -.009, t(43.3) = .973, p = .34 \). We did not look at subjective sleep quality given that people reported higher sleep quality on the second night.

**Rumination as an Individual Difference Moderator**

We anticipated that individuals prone to rumination would be more strongly affected by the social rejection task, showing longer delays in going to sleep after the rejection task compared to the neutral task, whereas those less prone to rumination would not take as long to go to bed after the social rejection compared to a night with no social rejection. There was a significant interaction effect for task-to-bed latency, \( b = 21.57, t(37.9) = 2.90, p = .006 \). As depicted in Figure 5, individuals lower in rumination (-1 SD) took a mere six minutes more, on
average, to go to bed after the social rejection task compared to after the neutral task, \( b = 6.25, t(37.9) = .64, p = .526 \). In contrast, individuals higher in rumination (+1 SD) took on average 45 minutes longer to fall asleep after being rejected compared to the neutral night, \( b = 45.70, t(37.9) = 4.79, p < .001 \).

**Effects of Prior Night Sleep on Rejection Experience**

Testing the reverse association between rejection and sleep, we found some indication that people who had more fragmented sleep the prior night were more physiologically reactive during the social rejection task (i.e., exhibited decreased IBIs relative to baseline), \( b = 3.6, t(29.29) = 2.16, p = .039 \). This effect remained similar, though less significant, when controlling for race, gender, age, and BMI, \( b = 3.54, t(25.06) = 2.02, p = .054 \). Global sleep quality over the prior month also showed some evidence of predicting physiological reactivity during the task (without covariates: \( b = -8.71, t(26) = 1.63, p = .116 \); with covariates: \( b = -13.47, t(23) = 2.19, p = .039 \)). Prior night sleep duration and sleep quality did not significantly predict IBIs during the task (duration \( b = -.19, t(28.75) = 1.33, p = .195 \); quality \( b = -6.5, t(29.94) = .30, p = .77 \), and these results did not change significantly when accounting for covariates (\( ts < 1.66, ps > .11 \)).

Regressing self-reported negative and positive emotions after the social rejection task onto sleep, we found that prior night sleep did not significantly predict people’s self-reported affective experience with or without covariates (\( ts \leq 1; \beta s \leq .16 \)). The effect of sleep over the past month was not significant either, but the effect sizes suggest this may be an issue of power, particularly for negative emotions: negative emotions (without covariates: \( b = .05, t(33) = 1.28, p = .21, \beta = .21 \); with covariates: \( b = .08, t(29) = 1.76, p = .089; \beta = .35 \)) and positive emotions (without
covariates: $b = -.07, t(33) = 1.30, p = .20, \beta = -.22$; with covariates: $b = -.05, t(29) = .87, p = .389; \beta = -.18$).

Together, these data present some initial evidence for links between affective and physiological responses to social rejection and sleep: People experienced greater physiological reactivity (decrease in IBIs), took longer to go to bed, and slept for a shorter amount of time after experiencing a social rejection compared to a control/neutral night. Effects of poorer sleep was exacerbated among those who were prone to more rumination. In addition, there was some indication that prior poor sleep, particularly fragmented sleep the prior night, was associated with more physiological reactivity during the rejection task. In our second study, we further examine the link between poor sleep and responses to rejection by testing whether poor sleepers have stronger physiological and affective responses to a laboratory-based rejection task using a much larger sample than in Study 1.

**Study 2: Sleep and Social Feedback in the Laboratory**

**Method**

**Participants**

As part of a larger study, three hundred and thirty-eight adults from the San Francisco Bay area participated in exchange for monetary compensation. The larger study included 383
participants, but the first 45 participants completed this study before the PSQI was added as a background questionnaire.

We could not compute a sleep score for ten, leaving 328 participants for analyses (191 female, 135 male, 2 unknown; 133 African Americans; 193 European Americans; $M_{\text{age}} = 26, SD = 4.50, \text{Range} = 18-35$). Participants were eligible if free from (a) current or past psychiatric disorder (e.g., clinical depression or clinical anxiety), (b) significant medical illnesses (e.g., heart arrhythmia or hypertension), (c) pregnancy, (d) a pacemaker, or (d) a BMI in the obese range (body mass index > 35). Participants were asked to abstain from caffeine, alcohol, and exercise for at least two hours prior to the lab session. Data collection began in June, 2011 and ended in December, 2016.

**Procedure**

Procedures relevant to this set of analyses are described below and follow from a previous published study on social rejection and acceptance (6). In addition to manipulating social feedback (rejection or acceptance), participants were also assigned to an oxytocin manipulation (oxytocin or placebo). All analyses control for oxytocin condition (results are similar with and without this covariate). After participants completed initial intake information and physiological sensors were attached, participants’ physiological responses were recorded for five minutes while they sat quietly. During the laboratory session, participants completed a variety of tasks. First, they were told that they would be interacting with another participant (i.e., a confederate), who was in a different lab room. An audiovisual connection between the two
experiment rooms allowed for the participant and confederate to see and hear each other over large television monitors. After the brief introduction, the participant was randomly assigned to complete an **evaluated speech task** (i.e., delivering a speech on “Why I make a good friend” for 3 minutes while their partner listened to the speech). After the speech was the **feedback manipulation** in which participants received evaluation ratings from the confederate that were either mildly rejecting or accepting. Specifically, the evaluation form listed the following five statements with the partner’s ostensible rating (on a scale of -4 to +4): “I would like to work at the same business or job as my partner,” “I would like to work closely on a project or team with my partner,” “I would like to get to know my partner better,” “I would enjoy being neighbors with my partner,” and “I would like to be close friends with my partner.” Participants in the **rejection feedback condition** received generally unfavorable ratings (0 for the first three statements and -1, and -2 for the fourth, and fifth, respectively), while those in the **acceptance feedback condition** received favorable ratings on all five items (+3 for the first two statements and +4 for the rest). In order to prevent confederates or experimenters from modifying their behavior as a result of the feedback condition or trying to guess which type of feedback was delivered, no one in the lab (research assistants or confederates) knew about the feedback manipulation in this study. Only the study coordinator who set up the computer manipulation prior to the participant’s arrival and who did not interact with the participant until debriefing knew that there was a feedback manipulation in this study.

After completion of the questionnaire, the experimenter moved the confederate to the participant’s room so that they could perform two cooperative tasks together. The participant and the confederate first engaged in a task based on the game of taboo, which lasted 8 minutes. The
dyad then performed a tactile finger-spelling task for 3 minutes (50). After the completion of this task, the confederate was moved back to their original room.

Measures

_Sleep._ Participants reported on their typical sleep over the past month using the same measure as Study 1. Twenty-five participants did not have a global score on the PSQI due to partial missing data. Thus, we (1) recoded missing items as 0 for sleep disturbances and (2) imputed a global score for participants who had data for at least 5 of the 7 components by obtaining the mean for the non-missing components and multiplying it by 7. These approaches yielded data for 15 participants. In this sample, \( M = 5.40, SD = 3.14, range = 0 - 15 \).

_Cardiovascular responses._ We obtained cardiovascular responses from participants during the baseline resting period, speech task, and cooperative interactions. In line with Study 1, for these analyses we examined IBI and HRV. To obtain these measures, we used electrocardiography. Electrocardiography was recorded with two Ag/AgCl electrodes placed in a modified Lead II configuration (right upper chest, left lower rib). These signals were integrated with a Biopac MP150 data acquisition system (Goleta, CA). Data were edited and scored off-line in one-minute bins using the HRV (2.6) module from Mindware Technologies (Gahanna, OH). For heart rate variability, we focused specifically on respiratory sinus arrhythmia, RSA, which is a frequency-based measure that captures high frequency HRV. RSA is a pure measure of parasympathetic activation and can be influenced by social and affective stimuli (51). To examine how sleep and social feedback influenced cardiovascular reactivity directly following the feedback, we subtracted participants’ baseline responses obtained during the last minute of
the initial resting period from the physiological responses obtained during the first interactive

task (i.e., taboo game).

Affective responses. Participants reported on their current affect four times throughout the

study: prior to sensor application (baseline), directly after receiving social feedback from their

partner and anticipating interacting with them (interaction anticipation), after the first dyadic

interaction (post-interaction 1), and after the second dyadic interaction (post-interaction 2).

Affect was obtained using the same measure as in Study 1. Negative affect means ranged from

1.22 (post-interaction 2) to 1.43 (interaction anticipation; SDs = .34 -.48 αs = .81 -.85). Positive

affect means ranged from 3.06 (interaction anticipation) to 3.30 (post-interaction 1; SDs = .84 -.97; αs = .90 -.94).

Statistical Analyses

Data analyses were performed using SPSS Statistics Version 25 (IBM Corporation), with

significance levels set to p < .05 (two-tailed). For all analyses we used MLM, nesting

physiological reactivity and affective responses within participants (using an autoregressive

structure for residuals and a random intercept). Sleep (grand-centered), feedback (rejection = -.5,

acceptance = .5), and their interaction term were entered into the model as predictors along with

oxytocin condition as a covariate. Degrees of freedom were calculated using the same method as

Study 1. Supplementary models were run with the same covariates from Study 1: own race,

confederate race, gender, age, and BMI, but changes to the results were negligible with the

addition of these covariates. For physiological outcomes, we focused on the first 8-minute
interaction directly after participants received feedback from their partner. For affective responses, we focused on participants’ emotions directly after receiving feedback (in anticipation of an interaction with their partner), after the first social interaction, and after the second social interaction. Baseline affect was included in models to assess affective reactivity.

**Results**

**Effects of Sleep on Physiological Outcomes**

As shown in Table 3, there was no effect of sleep nor a sleep by feedback interaction on physiological outcomes, suggesting that participant’s reported sleep over the prior month did not significantly predict their physiological reactions to interacting with a participant who had either accepted or rejected them.

**Effects of Sleep on Affective Outcomes**

As shown in Table 3 and Figure 6, there was a significant sleep by feedback interaction for negative affect. People who tended to sleep poorly had higher negative affect at baseline relative to those who tended to sleep better, $b = .03, t(325) = 4.16, p < .001$. After receiving rejecting feedback, these individuals increased in negative affect when anticipating and interacting with their partner relative to those who slept well, $b = .03, t(319.95) = 3.66, p < .001$. In contrast, the negative affect of poor sleepers who received accepting feedback decreased post-feedback, leaving them with negative affect that did not differ significantly from their well-rested
counterparts, $b = .004, t(319.40) = .65, p = .52$. Sleep did not predict positive affect at baseline, $b = -.01, t(325) = .92, p = .36$, nor did sleep moderate the effect of feedback on positive affect (see Table 3 and Figure 6).

**General Discussion**

Across two studies, we provide some initial evidence for bi-directional links between sleep and social rejection. In Study 1, we found that social rejection, compared to a neutral task, influences sleep both by affecting how late people go to bed as well as how long they sleep. That is, people took longer to go to bed and slept for a shorter amount of time after completing a social rejection task compared to a neutral task. We did not find evidence that social rejection influenced people’s subjective sleep quality. Physiological arousal, namely shorter IBIs, may be one pathway through which rejection influences sleep. People experienced significantly decreases in IBI during the social rejection task compared to the neutral task, and task IBI was a marginal predictor ($p = .079$) of how long people took to go to bed. Differences in physiological activation extended post-task, but people showed no differences in arousal by the time they were asleep.

In line with prior work on rumination (30-34), we found that people prone to ruminate showed the strongest effects of social rejection on sleep. That is, while low trait ruminators did not take significantly longer to go to bed after the social rejection task compared to the neutral task, high trait ruminators took an extra 45 minutes to go to bed after the social rejection task.
In Studies 1 and 2, we found some evidence for the reverse association, in which sleep influenced people’s responses to social rejection. In Study 1, sleep—both fragmentation the prior night and global sleep over the prior month—predicted greater physiological reactivity (i.e., decreases in IBIs) during the social rejection task, though these effects were small, and not always significant without covariates. We found no significant effect of sleep on affective responses. However, in Study 2, we found the reverse: sleep over the prior month predicted more negative affect following a social rejection (compared to acceptance), but there was no effect of sleep on physiological reactivity.

Implications, Limitations, and Future Directions

Researchers are increasingly recognizing that sleep and social processes are linked; however, there is still a dearth of research examining these links (8). This work further highlights the ways in which our waking experiences influence our sleep, and vice versa. In addition, this work builds on prior work on social rejection by highlighting another potential negative health outcome of this universal and painful social experience. In line with prior work illustrating that social stressors affect sleep, we found that when people engage in lab tasks designed to elicit rejection, this may affect their sleep. In turn, poor sleep may make people more sensitive to negative social experiences, creating the potential for a downward cycle. The effect of social rejection on sleep may also shed light on sleep as a potential pathway through which social rejection influences other mental and physical health outcomes. For example, both social rejection and sleep are linked to depression (52), setting up the possibility of a path model in
which rejection enhances depression in part through poor sleep (although the reverse is also a clear possibility—more chronic rejection might also influence sleep through depression).

Although our findings have implications for both the sleep and rejection literatures, we see these studies as one step in testing these links and want to make note of the important limitations to this work. First, we included effect sizes in Study 1, but given our small sample it is difficult to know whether some of our null effects were due to true lack of findings or to lack of power or to what extent did we observe an effect that is not reliable. Unfortunately, budget and time constraints prevented us from collecting more useable data in this multi-day field experiment. Thus, we feel strongly that these findings, both significant and non-significant, need to be replicated in a larger sample. Second, we compared social rejection to a neutral task designed to control for light and computer exposure prior to bed. However, this task was not arousing, which leaves the possibility that our effects may be due to something about the arousing demands of the social rejection task (giving a speech) rather than the rejection itself. The rejection tasks we used are the gold-standard laboratory rejection paradigms (39,53). Cyberball and the on-line social rejection task have been validated in the lab as tasks that induce strong rejection (e.g., 40). Research has shown that the effects of these tasks are primarily due to the socio-evaluative component and that the stress and arousal associated with these tasks is an integral part of the rejection experience (5,54,55). For example, giving a speech without a socio-evaluative component does not produce the same physiological arousal as receiving negative social feedback during the speech (54). In addition, the fact that people high in rumination showed the strongest effects and the fact that physiological reactivity during the task was not a stronger predictor of sleep outcomes suggests that the sleep effects were not purely due to greater
physiological demand. Nonetheless, we cannot rule out the possibility that it was something about the task other than the rejection driving our effects. In future research, it will be critical to (a) compare this task to giving a speech without feedback or with positive feedback and (b) have a more passive induction of rejection, such as receiving a rejecting email, to ascertain whether we still see post-task physiological activation in response to a rejection manipulation that is not intrinsically arousing. Another limitation of the tasks in Study 1 was the lack of counter-balancing. Due to concerns that the study equipment, including a chest band, would be particularly disruptive to sleep on the first night, we chose not to have anyone complete the rejection task that night. We do not have any theoretical rationale as to why people would have worse sleep on the second night, nonetheless this approach means that we do not know for certain that timing did not affect our results.

One lingering question is whether we would observe the same effect of social rejection on sleep if the rejection had happened earlier in the day, as opposed to right before bed. Even if this effect is isolated to just prior to bed, it is becoming increasingly relevant as people stay engaged on social media up until they turn out the lights. However, we expect we would see this effect even if rejection happened earlier in the day. Pre-sleep cognitions are often when negative experiences from the day come back into one’s mind, and our findings with rumination suggest that some of our sleep effects are due to this exact type of ruminative processing.

In considering the reverse association between sleep and social rejection, we found differing effects across studies. Although the disconnect between affect and physiology is not surprising in light of much prior work showing a similar disconnect (56,57), given that we did
not manipulate sleep, and only had a subjective self-report measure in Study 2, we see these findings as preliminary and urge future researchers to examine the effects of a sleep deprivation experiment on important social outcomes such as social rejection. It also might be the case that the effects of sleep on social processes is simply weaker or nonexistent relative to the influence of social processes on sleep.

In conclusion, these studies provide a first step into examining the real-world implications of social rejection on sleep, suggesting that rejecting experiences may influence our bedtime behavior while poor sleep may exacerbate social rejection.
References


31. Pillai V, Drake CL. Sleep and repetitive thought: the role of rumination and worry in sleep disturbance. In Sleep and affect 2015 (pp. 201-225).


**Figure 1.** Study 1 Means for IBI and HRV during Baseline on Night 1 (Neutral) and Night 2 (Rejection). Error bars represent Standard Errors.

**Figure 2.** Study 1 Means for IBI and HRV during Tasks on Night 1 (Neutral) and Night 2 (Rejection). Error bars represent Standard Errors. X-axis represents 1-minute bins. Not all bins are contiguous due to experimenter instructions between parts of tasks. // represents a break of several minutes.

**Figure 3.** Study 1 Means for IBI and HRV during First Hour Post Task on Night 1 (Control) and Night 2 (Rejection). Error bars represent Standard Errors. Horizontal error bars represent Standard Errors for Bed Time and Sleep Time.

**Figure 4.** Study 1 Means for IBI and HRV Across First 20 Minutes of Sleep on Night 1 (Control) and Night 2 (Rejection). Error bars represent standard errors.

**Figure 5.** Individual Differences in Rumination Predicting Task to Bed Latencies across Nights in Study 1. Error bars represent estimated standard errors for simple effects.

**Figure 6.** Study 2 Affect across Lab Session as Function of Sleep and Feedback Condition. Error bars represent estimated standard errors.
Figure 1

Physiological Responses on Night 1 versus Night 2
Figure 3
Figure 4
Figure 5
Figure 6

Affective Responses Across Tasks in Study 2 by Feedback and Sleep

![Graph showing affective responses across tasks in study 2 by feedback and sleep.](image)
Table 1. Study 1 Sleep Outcomes by Condition

<table>
<thead>
<tr>
<th>Sleep Outcomes</th>
<th>N</th>
<th>Control Night M(SE)</th>
<th>Rejection Night M(SE)</th>
<th>DFs</th>
<th>t</th>
<th>p</th>
<th>Cohen’s D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Task to Bed Latency (Mins)</td>
<td>43</td>
<td>11.18 (2.37)</td>
<td>38.06 (7.41)</td>
<td>42</td>
<td>3.86</td>
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<td>1.73</td>
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<td>Sleep Duration (Hrs:Mins)</td>
<td>42</td>
<td>7:19 (15.09)</td>
<td>6:46 (13.37)</td>
<td>41</td>
<td>2.92</td>
<td>.006</td>
<td>0.34</td>
</tr>
<tr>
<td>Fragmentation</td>
<td>42</td>
<td>18.05 (1.15)</td>
<td>21.37 (2.13)</td>
<td>41</td>
<td>1.74</td>
<td>.090</td>
<td>0.44</td>
</tr>
<tr>
<td>Sleep Quality</td>
<td>38</td>
<td>2.83 (.63)</td>
<td>2.93 (.57)</td>
<td>37</td>
<td>0.89</td>
<td>.38</td>
<td>0.16</td>
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</tbody>
</table>

*Note: Cohen’s D is the difference between rejection and control night divided by the control night standard deviation.*
Table 2. Study 1 Physiological Reactivity by Night

<table>
<thead>
<tr>
<th>Physiological Period</th>
<th>Neutral Night M(SE)</th>
<th>Rejection Night M(SE)</th>
<th>Denom DFs</th>
<th>F</th>
<th>p</th>
<th>Effect Size (D)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline (N = 39)</td>
<td>IBI 813.5 (22.5)</td>
<td>825.7 (22.5)</td>
<td>31.2</td>
<td>.27</td>
<td>.61</td>
<td>.07</td>
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<tr>
<td></td>
<td>HRV 7.94 (.14)</td>
<td>8.02 (.14)</td>
<td>31.4</td>
<td>.27</td>
<td>.61</td>
<td>.07</td>
</tr>
<tr>
<td>Task (Reactivity) (N = 39)</td>
<td>IBI 64.6 (15.4)</td>
<td>-32.3 (16.4)</td>
<td>63.2</td>
<td>18.51</td>
<td>&lt;.001</td>
<td>.80</td>
</tr>
<tr>
<td></td>
<td>HRV .09 (.14)</td>
<td>-.25 (.15)</td>
<td>63.5</td>
<td>2.75</td>
<td>.10</td>
<td>.30</td>
</tr>
<tr>
<td>Post Task (first hr) (N = 38)</td>
<td>IBI 903.4 (25.4)</td>
<td>854.3 (25.3)</td>
<td>29.4</td>
<td>4.71</td>
<td>.038</td>
<td>.26</td>
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<tr>
<td></td>
<td>HRV 7.94 (.14)</td>
<td>7.87 (.13)</td>
<td>32.98</td>
<td>.17</td>
<td>.68</td>
<td>.06</td>
</tr>
<tr>
<td>Sleep (first 20 mins) (N = 38)</td>
<td>IBI 942.0 (27.4)</td>
<td>951.9 (27.8)</td>
<td>27.7</td>
<td>.17</td>
<td>.68</td>
<td>.05</td>
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<td></td>
<td>HRV 7.73 (.17)</td>
<td>7.69 (.18)</td>
<td>32.5</td>
<td>.04</td>
<td>.84</td>
<td>.03</td>
</tr>
</tbody>
</table>

Note: Estimated marginal means and standard errors. Effect Size (D) reflects difference between control night and rejection night divided by control night SD (estimated SE*√(df + 1)). IBI units are in milliseconds (ms); HRV units are in natural log (ln).
Table 3. Study 2 Physiological and Affective Reactivity

<table>
<thead>
<tr>
<th>Physiological Outcomes</th>
<th>$b$</th>
<th>SE</th>
<th>DFs</th>
<th>$t$</th>
<th>$p$</th>
<th>95% CI</th>
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<tr>
<td><strong>IBI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(N = 323)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Feedback</td>
<td>17.20</td>
<td>9.88</td>
<td>314.6</td>
<td>1.74</td>
<td>.083</td>
<td>(-2.25, 36.64)</td>
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<tr>
<td>Sleep</td>
<td>2.47</td>
<td>1.57</td>
<td>314.6</td>
<td>1.57</td>
<td>.118</td>
<td>(-.63, 5.56)</td>
</tr>
<tr>
<td>Feedback x Sleep</td>
<td>3.63</td>
<td>3.15</td>
<td>314.6</td>
<td>1.15</td>
<td>.250</td>
<td>(-2.56, 9.82)</td>
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<tr>
<td><strong>RSA</strong></td>
<td></td>
<td></td>
<td></td>
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<td>(N = 323)</td>
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<tr>
<td>Feedback</td>
<td>.26</td>
<td>.11</td>
<td>316.5</td>
<td>2.35</td>
<td>.019</td>
<td>(.04, .47)</td>
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<tr>
<td>Sleep</td>
<td>.01</td>
<td>.02</td>
<td>316.5</td>
<td>.66</td>
<td>.512</td>
<td>(-.02, .05)</td>
</tr>
<tr>
<td>Feedback x Sleep</td>
<td>-.03</td>
<td>.03</td>
<td>316.5</td>
<td>.78</td>
<td>.438</td>
<td>(-.10, .04)</td>
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<tr>
<td><strong>Negative Affect</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>(N = 328)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feedback</td>
<td>-.18</td>
<td>.03</td>
<td>320.4</td>
<td>5.72</td>
<td>&lt; .001</td>
<td>(-.24, -.12)</td>
</tr>
<tr>
<td>Sleep</td>
<td>.02</td>
<td>.01</td>
<td>319.8</td>
<td>3.089</td>
<td>.002</td>
<td>(.01, .03)</td>
</tr>
<tr>
<td>Feedback x Sleep</td>
<td>-.02</td>
<td>.01</td>
<td>319.6</td>
<td>2.28</td>
<td>.023</td>
<td>-.04, -.003</td>
</tr>
<tr>
<td><strong>Positive Affect</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(N = 328)</td>
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<td></td>
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<tr>
<td>Feedback</td>
<td>.11</td>
<td>.06</td>
<td>320.4</td>
<td>4.00</td>
<td>&lt; .001</td>
<td>(.12, .34)</td>
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<td>Sleep</td>
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<td>.01</td>
<td>319.8</td>
<td>.08</td>
<td>.93</td>
<td>-.02, .02</td>
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<tr>
<td>Feedback x Sleep</td>
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<td>.02</td>
<td>319.8</td>
<td>.987</td>
<td>.324</td>
<td>-.02, .05</td>
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</table>

*Note: All models control for oxytocin condition; models of affect control for baseline affect. IBI units are in milliseconds; RSA units are in millisecond-squared ms².