Restless REM sleep in insomnia disorder and its detrimental effects on regulation of emotional distress

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2019

document version
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Chapter 3.

Overnight worsening of emotional distress indicates maladaptive sleep in insomnia

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In press, 2019 in:

Sleep, doi: 10.1093/sleep/zsy268

Abstract

Study Objectives: Mechanisms underlying the distress of hyperarousal in people with insomnia remain enigmatic. We investigated whether insomnia impedes the overnight adaptation to emotional distress.

Methods: We induced the distressful self-conscious emotion of shame four times across three consecutive days by exposing 64 participants to their often embarrassingly out-of-tune singing, recorded earlier during a Karaoke session. Perceived physical, emotional, and social distress was assessed with the Experiential Shame Scale.

Results: Compared to exposures followed by wakefulness, exposures followed by sleep resulted in overnight *relief* of physical component of shame in normal sleepers, but in a striking opposite overnight *worsening* in people with insomnia.

Conclusions: Our findings are the first to experimentally show that the benefits of sleep are not only lost when sleep is poor; people with insomnia experience a maladaptive type of sleep that actually aggravates physically perceived distress. Maladaptive sleep could shed new light on posttraumatic stress disorder (PTSD) and on diurnal mood fluctuation and the counterintuitive favourable effects of sleep deprivation in depression.

Introduction

Insomnia Disorder (ID) is the second most common mental disorder in Europe (Wittchen *et al.* 2011), and the primary modifiable risk factor for the development of major depressive disorder (Manber *et al.* 2008, Baglioni *et al.* 2011, Blom *et al.* 2015, Christensen *et al.* 2016, Blom *et al.* 2017). Insomnia moreover contributes to the risk of most other major societally burdening chronic disorders and their underlying pathology (Van Someren *et al.* 2015). ID is diagnosed when problems falling asleep or maintaining sleep, accompanied by daytime distress, occur three times or more each week for at least three months, and cannot be attributed to unfavourable sleeping conditions (American Psychiatric Association 2013, Diagnostic Classification Steering Committee 2014). The treatment of choice is Cognitive Behavioral Therapy for Insomnia (CBT-I), which unfortunately does not have satisfying results for every individual. CBT-I works in about 70% of the cases, yet ameliorates complaints only by about 30% (Harvey and Tang 2003, Morin *et al.* 2009). Pharmacological treatment for insomnia is discouraged (National Institutes of Health 2005). The current status of treatment effectiveness thus leaves ample room for improvement, which is hampered by an insufficient understanding of underlying mechanisms of sleep complaints and the accompanying characteristic daytime physical distress known as hyperarousal.

Surprisingly, little support has been found so far for malfunctioning of sleep-regulatory neurobiological pathways in ID. For example, the genetic risk of insomnia is only modestly correlated with the genetic risk of other sleep traits (Hammerschlag *et al.* 2017, Jansen *et al.* 2018). Rather, both genetically and phenotypically, insomnia is most markedly associated with anxiety disorders (Hammerschlag *et al.* 2017). Moreover, functional annotation analysis found enrichment of expression of insomnia risk genes in the limbic brain circuitry, once more pointing to key involvement of emotion regulation rather than sleep regulation *per se* (Jansen *et al.* 2018). Conceptually, ID can indeed best be characterized as a chronic state of hyperarousal, which manifests itself across many physiological and psychological dimensions (Baglioni *et al.* 2010, Bonnet and Arand 2010, Riemann *et al.* 2010), and resembles the state that healthy volunteers show during short-lived anxiety or other emotional distress. The upcoming hypothesis that anxiety disorders can mechanistically be regarded disorders of

emotional memory (Kindt 2018), may apply equally well to insomnia (Wassing *et al.* 2016). The current study addressed the idea that ID may involve a deficiency to downregulate emotional distress across the night.

A supportive role of sleep in overnight emotion regulation has been demonstrated in healthy volunteers; re-exposure to an emotional stimulus after a period of sleep elicits less self-reported emotional distress, less autonomic arousal, and less activation of the amygdala, than when the emotional stimulus is repeated across a comparable period without sleep (Sterpenich *et al.* 2007, Pace-Schott *et al.* 2011, Van Der Helm *et al.* 2011, Talamini *et al.* 2013). If sleep-dependent memory reactivation and synaptic reorganization can occur within a critical time window of about 6 hours, emotional distress is downregulated (Nader *et al.* 2000, Agren *et al.* 2012). Such downregulation fails when sleep is perturbed (Van Der Helm *et al.* 2011, Vanderheyden *et al.* 2014). Because the sleep electroencephalography (EEG) of people with ID is characteristically perturbed by increased high frequency activity and fragmentation due to brief arousals (Baglioni *et al.* 2014), we hypothesized that they might show insufficient overnight adaptation to emotional stimuli (Wassing *et al.* 2016), or even an overnight worsening of emotional reactivity (Vanderheyden *et al.* 2014). We repeatedly induced the self-conscious emotion of shame to evaluate if changes in perceived distress across two subsequent exposures depended on whether the first exposure was immediately followed by sleep or not. We hypothesized a stronger beneficial effect of immediate sleep in normal sleepers than in people suffering from ID.

Methods

Study participants

Participants were recruited via an emailed newsletter sent to participants of the Netherlands Sleep Registry (NSR; Benjamins *et al.* 2017). Inclusion criteria were an age between 18 and 70 years, availability for morning and evening sessions on five consecutive days, and access to a computer with microphone and headphone. Exclusion criteria were chronic use of medication and diagnosis of any neurological or mental disorder other than insomnia. The study was approved by the ethical committee of the University of Amsterdam, the Netherlands.

Table 3.1. Mean sleep quantity and quality estimated from five nights of sleep diary data

Differences between insomnia disorder and normal sleepers were evaluated using mixed-effects general linear models.

	NS n = 42, mean (SE)	**ID** n = 22, mean (SE)	***t*-statistic**	***p*-value**
Bed time (hr:mm)	23:52 (00:11)	23:07 (00:15)	-2.41	0.02
Lights out time (hr:mm)	00:19 (00:09)	23:40 (00:13)	-2.52	0.01
Final wake time (hr:mm)	07:44 (00:08)	06:45 (00:12)	-4.12	5.0×10^{-5}
Get up time (hr:mm)	08:10 (00:08)	07:31 (00:11)	-2.82	0.005
SOL (min)	13.0 (3.9)	41.3 (5.6)	4.15	4.4×10^{-5}
Awakenings (N)	1.6 (0.2)	2.7 (0.3)	3.15	0.002
WASO (min)	15.3 (4.4)	58.5 (6.2)	5.68	3.2×10^{-8}
TIB (min)	497.9 (10.5)	503.9 (14.8)	0.33	0.74
TST (min)	416.8 (9.7)	325.0 (13.6)	-5.50	8.3×10^{-8}
SE (%)	84.2 (1.3)	64.8 (1.9)	-8.27	4.4×10^{-15}
Self-reported sleep quality[a]	3.9 (0.1)	2.8 (0.1)	-6.79	6.1×10^{-11}

NS: normal sleepers, ID: insomnia disorder, SOL: sleep onset latency, WASO: wake after sleep onset (time spend awake between sleep onset and final awakening), TIB: time in bed, TST: total sleep time, SE: sleep efficiency. [a] Self-reported sleep quality was assessed on a 5-point Likert-type scale.

According to the Insomnia Severity Index cutoff ≥10 that is optimized for classification of ID in community samples (86.1% sensitivity and 87.7% specificity; Morin *et al.* 2011), 22 participants suffered from ID (mean age (SD) = 52.7 (10.7) years, 17 women (77.3%)), and 42 were normal sleepers (48.5 (14.9) years, 36 women (85.7%)). We used mixed effects general linear models to evaluate differences in sleep diary assessments between cases with ID and normal sleepers, which confirmed the classification as indicated by pronounced group differences in quantitative and qualitative aspects of sleep, most significantly so for sleep efficiency (table 3.1).

Procedure

Volunteers participated at their own home, making use of a dedicated webpage on the NSR website. On the first day, participants recorded a karaoke-style sing-along of one of three songs: "Gloria in Excelsis Deo", "Silent Night, Holy Night", or the Dutch national anthem, counterbalanced either in the morning or in the evening. These songs were selected because of their familiarity and the likely difficulty to maintain pitch when performing them.

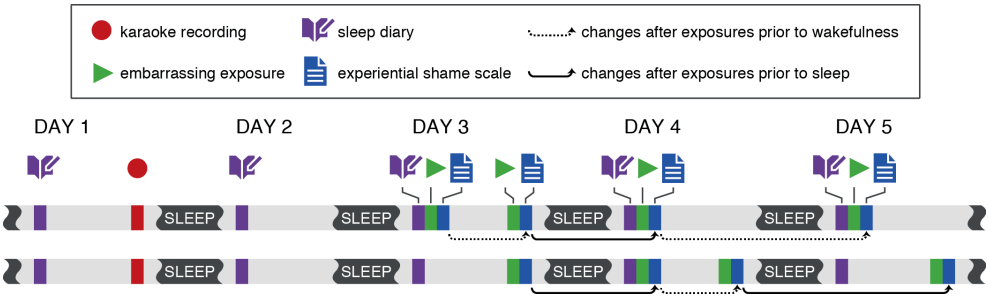

Figure 3.1. Examples of two individual schedules out of twelve possible counterbalanced schedules

On the first day, a karaoke style sing-along of Christmas songs or the national anthem was recorded, counterbalanced either in the morning or in the evening (red bar, morning not shown). Over the course of day three to five, the participants were exposed to the recording of their solo singing 4 times (green bars). Exposures were immediately followed by assessment of perceived physical, emotional and social distress using the Experiential Shame Scale (blue bars; Turner 2014). Analyses compared changes in experienced shame after exposures immediately followed by sleep (solid arrows) with exposures immediately followed by a period of wakefulness (dotted arrows). Participants filled out sleep diaries (Carney *et al.* 2012) each morning between 6 am and 10 am (purple bars). The examples show an AM-12hr-PM-12hr-AM-24hr-AM sequence (top) and a PM-12hr-AM-12hr-PM2-4hr-PM sequence (bottom).

To make the audio recording, participants heard instrumentation and vocals of others over their headphones while singing along with the lyrics presented in a Karaoke-style video. Their own voice was not presented over the headphones to impede pitch correction and thus promote out-of-tune singing, even for trained individuals. Participants received no feedback on their singing performance afterwards. The audio file was stored on the NSR data-server.

Over the course of day three to five, the self-conscious emotional distress of shame was experimentally induced by exposing people to the recordings of their own solo-singing unaccompanied by instruments and vocals of others. Participants were exposed for a total of four times across three consecutive days, commencing either in the morning (between 6 am and 10 am) or in the evening (between 6 pm and 10 pm) with two intervals of ±12 and one interval of ±24 hours (figure 3.1). The twelve possible schedules were counterbalanced between participants. The 2'12" to 2'47" duration recordings were offered from the NSR webpage and played through headphones. To illustrate the likely feelings of shame, the supplementary video "Singing Silent Night" (online material or https://youtu.be/G3gWyua3grE)

provides a recording example sung by the corresponding author. Listening might give others *Schadenfreude*[1].

Outcome measures

Immediately following each exposure, participants rated the shame they experienced using the 11-item Experiential Shame Scale (ESS; Turner 2014). The scale is validated to assess state shame rather than guilt. Importantly, participants are unlikely to discern the intent of the scale, which is advantageous to prevent bias, social desirability and the induction of coping strategies. Each item of the ESS is presented as two opposing states, for example, pale (left) versus flushed (right), with seven circles between the two words (range 0-6). Participants are instructed to select the circle that best describes how they feel at that moment when comparing the two opposite word-states.

The ESS provides three components of shame (physical, emotional and social) by averaging the items. The three items of the physical component ask about somatic symptoms: feeling very warm versus very cool, and flushed versus pale, and experiencing a rapid versus normal heartbeat. The four items of the emotional component assess the extent to which people are feeling bad versus good, confused versus clear, distressed versus content, and highly agitated versus calm. The four items of the social component ask whether participants felt like hiding versus being sociable, being quiet versus talkative, being looked at by others versus unobserved, and wanting to talk to others about the recording or not. As intended by the ESS, this last item was adapted to the context of this experiment as follows: "*I AM [...]*" versus "*I AM NOT willing to talk about the recording with an acquaintance right now*". In addition, to characterize sleep and insomnia complaints, participants filled out the Insomnia Severity Index (Morin *et al.* 2011) once, and the Consensus Sleep Diary (Carney *et al.* 2012) every morning during the protocol (figure 3.1).

Statistical analysis

Analyses evaluated case-control differences in the sleep-related change for each of the shame components (physical, emotional, and social) and were performed using linear mixed-effect models with a random intercept (MATLAB and Statistics and

[1] German noun: pleasure derived by someone from another person's misfortune.

Machine Learning Toolbox, R2016a, The MathWorks, Inc., Natick, MA, United States). Separate models were run for each of the three shame components as outcomes (equation 3.1). In line with previous work showing effects primarily if sleep immediately follows the stimulus (Talamini *et al.* 2008, Van Der Werf *et al.* 2009), we compared changes after exposures immediately followed by sleep with exposures first followed by a period of wakefulness. The significance of regression coefficients was evaluated with two-tailed *t*-test thresholded at $\alpha = 0.05$.

Equation 3.1.

$$shame_{ij} \sim \beta_0 + \beta_1 \times group_{ij} + \beta_2 \times time_{ij} + \beta_3 \times sleep_{ij} + \beta_4 \times (group_{ij} \times time_{ij}) + \beta_5 \times (group_{ij} \times sleep_{ij}) + \gamma_{0j} + \varepsilon_{ij}$$

exposures *i* are nested within participants *j*,
shame represents one of the three shame components, each analysed separately,
time represents the elapsed time since the first exposure,
group is a dichotomous variable indicating normal sleepers (0) or cases with ID (1),
sleep is a dichotomous variable indicating intervals commencing with wakefulness (0) or sleep (1),
γ_{0j} is a random intercept for each participant, and
ε_{ij} are the residuals.

Results

Four participants missed one exposure, and one participant missed two. Four participants reported incidental sleep medication use on one night, and one participant on two nights. These exposures were removed, resulting in a dataset with 244 out of 256 possible observations (95.3%). ID and normal sleepers did not significantly differ in the intensity of perceived shame after the first exposure: mean (SD) physical component of shame (ID: 2.9 (0.9), NS: 2.8 (0.7); $t(58) = 0.37$, $p = 0.71$), emotional component of shame (ID: 2.5 (1.5), NS: 1.9 (1.4); $t(58) = 1.62$, $p = 0.11$), social component of shame (ID: 3.3 (0.8), NS: 3.1 (1.0); $t(58) = 0.92$, $p = 0.36$).

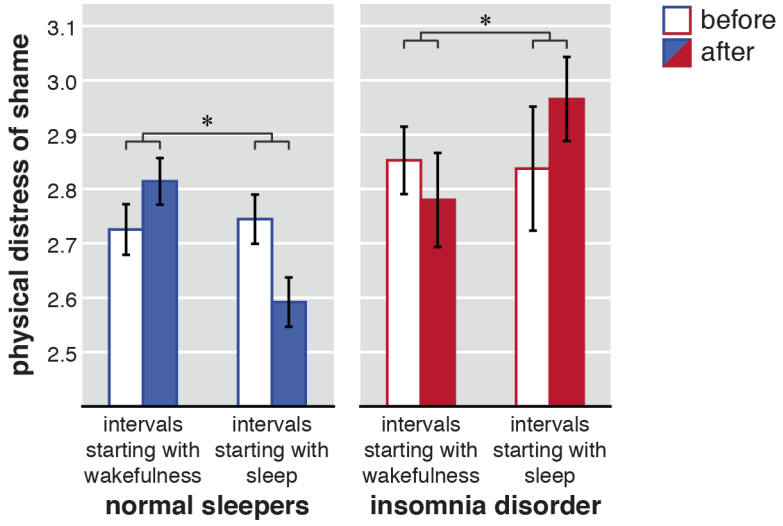

Figure 3.2. Changes in the perceived physical distress of shame

If exposures were not immediately followed by sleep (left bars), the next exposure elicited a comparable amount of physical distress. In NS, exposures prior to immediate sleep were followed by a significantly stronger decrease in the physical distress they elicited than exposures not immediately followed by sleep ($p = 0.03$). Strikingly opposite ($p = 0.001$), in ID, exposures prior to immediate sleep were followed by a significantly stronger *increase* in the physical distress they elicited than exposures not immediately followed by sleep ($p = 0.03$). Error-bars show within-subject standard errors. * $p < 0.05$.

Habituation of shame across time

Mixed-effect models indicated that, neither the physical nor the emotional component of shame showed an overall linear change across the days (resp. β (SE) = −0.05 (0.06), $t(238) = -0.80$, $p = 0.43$, and β (SE) = 0.03 (0.09), $t(238) = 0.36$, $p = 0.72$). The social component of shame showed a nonsignificant trend to decrease over time (β (SE) = −0.12 (0.06), $t(238) = -1.94$, $p = 0.053$), and this effect was not different for normal sleepers and ID (β (SE) = 0.07 (0.10), $t(238) = 0.66$, $p = 0.51$).

Changes in shame depend on sleep and insomnia

Changes in perceived shame depended on whether the previous exposure was immediately followed by sleep or by wakefulness. In normal sleepers, the physical component of shame decreased more steeply after exposures followed by sleep than after exposures followed by wakefulness (β (SE) = −0.19 (0.08), $t(159) = -2.22$, $p = 0.028$; figure 3.2). This effect was also observed for the emotional component of shame (β = −0.35 (0.15), $t(159) = -2.34$, $p = 0.021$) but not for the social

component of shame (β = 0.06 (0.09), $t(159)$ = 0.68, p = 0.49). In contrast, a remarkably different change was seen in people suffering from insomnia: exposures prior to sleep were followed by a steeper *increase* in the physical component of shame than the change following exposures prior to wakefulness (β = 0.37 (0.17), $t(79)$ = −2.22, p = 0.029; figure 3.2), while sleep timing did not matter for the emotional (β = −0.06 (0.19), $t(79)$ = −0.29, p = 0.77) and social (β = 0.08 (0.16), $t(79)$ = 0.51, p=0.61) components of shame.

A significant interaction effect confirmed that cases with ID and normal sleepers differ with respect to the effect of immediate sleep on the subsequent change in the physical component of shame (β = 0.56 (0.17), $t(238)$ = 3.32, p=0.001; figure 3.3). Cases with ID and normal sleepers did not differ significantly with respect to sleep timing-specific changes in the emotional (β = 0.28 (0.25), $t(238)$ = 1.12, p = 0.26) or social (β = 0.03 (0.17), $t(238)$ = 0.16, p = 0.87) components of shame.

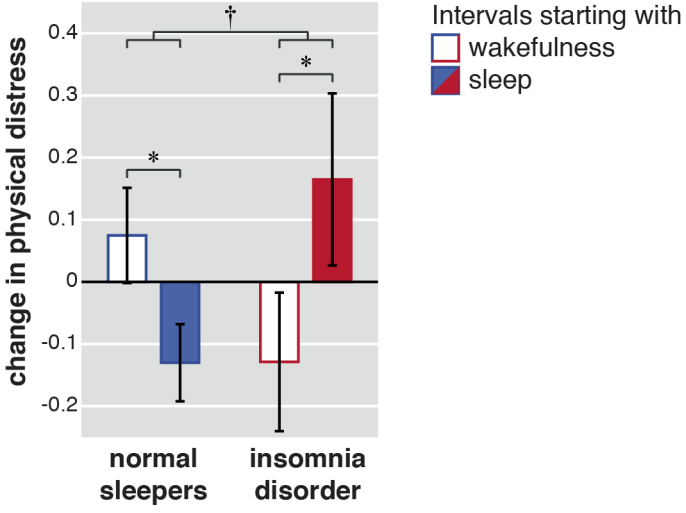

Figure 3.3. Visualization of physical distress changes from one to the next exposure

Changes differ depending on whether the first exposure was immediately followed by sleep, but in a strikingly opposite way (p = 0.001) in people with normal sleep (NS, left bars) and those with Insomnia Disorder (ID). Whereas immediate sleep ameliorates subsequent distress in NS, it rather boosts subsequent distress in ID. Bars indicate average changes adjusted for time effects. Error-bars show within-subject standard errors, * $p < 0.05$, † $p = 0.001$.

These findings indicate that insomnia impedes overnight emotion regulation, but this effect could be confounded by diurnal changes in mood or emotional reactivity or by group-differences in habituation over time. Importantly however, perceived shame was not significantly different on morning or evening exposures, and the time-of-day effect was not significantly different between ID and NS ($0.18 \leq p \leq 0.92$; supplementary material). Furthermore, we found no moderation of the habituation over time by ID for the physical component of shame ($\beta = -0.15$ (0.10), $t(238) = -1.58$, $p = 0.11$), nor for the emotional ($\beta = -0.12$ (0.15), $t(238) = -0.79$, $p = 0.43$), or the social component of shame ($\beta = 0.07$ (0.10), $t(238) = 0.66$, $p = 0.51$).

Discussion

Mechanisms underlying the characteristic daytime distress experienced as hyperarousal by people with insomnia have so far remained enigmatic. To experimentally investigate the role of disturbed sleep in the overnight adaptation to the experienced distress of a self-conscious emotion, we confronted participants with ID and normal sleepers with their own imperfect singing of a Christmas song or a national anthem. The findings show that people suffering from insomnia experience an adverse effect on adaptation if they sleep immediately after having been exposed to a self-conscious emotional experience. This adverse effect is strikingly opposite to the benefits of immediate sleep on adaptation that normal sleepers experience. Case-control differences in the effect of sleep timing were most pronounced for the physical distress induced by the shameful experience. In normal sleepers, physical distress decreased with re-exposure if the previous exposure had immediately been followed by sleep. People with insomnia, however, experienced the opposite: immediate sleep in fact boosted subsequent physical distress with re-exposure. Moreover, normal sleepers also experienced a decrease in emotional distress, while people with insomnia did not. Neither cases with ID nor normal sleepers experienced sleep timing-specific changes in social distress.

Hyperarousal is a multidimensional concept considered key to insomnia. Different research groups have mentioned different dimensions, including autonomous, neuroendocrine, neuroimmunological, electrophysiological, neuro-

psychological, somatic, cortical, cognitive, sleep-reactive, and emotional (Perlis *et al.* 1997, Bonnet and Arand 2010, Riemann *et al.* 2010, Spiegelhalder and Riemann 2013, Wassing *et al.* 2016). Our findings suggest that overnight adaptive consequences of the disturbed sleep in ID may surface more strongly in primary somatic reactivity than in higher order emotional and social cognitive appraisal. Indeed, primary somatic reactivity involves different brain circuits than secondary evaluation, reappraisal, and suppression (Etkin *et al.* 2011). It would be interesting to employ brain imaging studies to evaluate circuits involved in the suggested maladaptive sleep.

A first strength of the study is that, to our knowledge, it is the first experimental approach to address the overnight resolution of self-conscious emotions rather than of basic emotions addressed in previous work (Sterpenich *et al.* 2007, Pace-Schott *et al.* 2011, Van Der Helm *et al.* 2011). In psychological and psychiatric practice, self-conscious emotions like guilt, embarrassment, humiliation, pride and especially shame are more clinically relevant than basic emotions (Feiring and Taska 2005, Stuewig and Mccloskey 2005, Schalkwijk 2015). Shame, for example, increases the risk of developing depression (Stuewig and Mccloskey 2005), contributes to the development of post-traumatic stress disorder (PTSD; Au *et al.* 2017) and reversibly worsens its symptoms including hyperarousal (Feiring and Taska 2005, Øktedalen *et al.* 2015). Also, by interfering with effective coping, shame hinders therapeutic progress (Black *et al.* 2013). Our findings are the first to suggest that sound sleep could support therapeutic progress by resolving some of the somatic distress of shame, making it less difficult to work again on the shameful experience in the next therapy session. It must be noted however, that we did not induce shame from a salient autobiographical event, but rather participants were exposed to listening to their own imperfect singing, a relatively harmless, albeit distressing, stimulus. Generalization of our findings to clinical settings therefore remains to be evaluated. Future studies could, for example, start with cued recall of autobiographical memories to assess abiding of shameful emotional distress in ID.

As a second strength, the stringent exclusion criteria allow for the interpretation of insomnia-specific effects, rather than effects that are possibly confounded by use of medication or comorbid other mental disorders. At the same

time, the stringent exclusion criteria can also be regarded a limitation. Since insomnia is so well represented among people suffering from mental disorders, and since many people with insomnia resort to chronic use of sleep medication, our sample may not optimally represent the general population of people with insomnia. Moreover, the exclusion criteria limited the sample size to 22 cases, as compared to 42 normal sleepers.

Other limitations deserve mention as well. A notable limitation is that we only assessed self-reported measures of emotional distress. Future studies could employ physiological assessments and brain imaging to assess underlying mechanisms. Also, although we interpret the findings as probably generic for the overnight regulation of all emotions, we only induced and evaluated one type of self-conscious emotion, and no basic emotions. Several overnight studies would be needed to evaluate the generalizability and consistency of the suggested maladaptive effect of sleep in ID across the spectrum of emotions. Moreover, we did not find significant overall habituation across the exposures. Three re-exposures across three days might be insufficient to detect possible small group differences in habituation. Future studies could assess longer intervals.

The limitation of having access to self-reported measures also holds for sleep variables, which were restricted to data from sleep diaries. Self-reported sleep variables can show remarkably low correlations with polysomnographically assessed sleep. What makes it even more complex is that the correlation itself may depend on the subjectively experienced quality of sleep, i.e. it can be especially low in people with more severe insomnia (Rosa and Bonnet 2000, Moul *et al.* 2002). We analysed the long list of associations of changes on the three subscales of experienced shame, across day and night, with all diary-derived sleep variables, both across and within groups of cases and controls. Analyses included false discovery rate correction. No associations were found (supplementary table S3.1). Furthermore, across the mornings with exposures, average final wake-up time was about an hour later in normal sleepers and they started with the task about half an hour later than participants with ID. Importantly however, interindividual differences in the time between final awakening and task start time did not explain perceived shame (supplementary material). Clearly, the current proof-of-principle

study has to be followed-up by studies including objective sleep variables assessed with polysomnography.

Memory and emotion regulation involve both non-rapid eye movement (NREM) and rapid eye movement (REM) sleep (Talamini *et al.* 2013). Recent animal studies provide important clues that explain how sleep-dependent processes can become maladaptive (Vanderheyden *et al.* 2014, Poe 2017, Swift *et al.* 2018). During sound sleep, Locus Coeruleus (LC) neurons fire at much lower rates as of the transition from slow-wave sleep to REM sleep and throughout REM sleep. The resulting low level of noradrenalin provides a time window for efficient synaptic depotentiation. Abiding LC activity during sleep is therefore particularly maladaptive for adaptive processes that depend on synaptic depotentiation (Swift *et al.* 2018). Abiding LC activity changes the electrophysiological signatures of both NREM and REM sleep (Swift *et al.* 2018), and is likely to underlie the increased high-frequency activity and abundant arousals that occur in the sleep EEG of people that have been exposed to childhood adversity (Insana *et al.* 2012); in people with PTSD (Mellman *et al.* 2002, Mellman *et al.* 2007, Germain *et al.* 2008); and in people suffering from insomnia (Feige *et al.* 2008, Riemann *et al.* 2012). Concertedly, these findings delineate the variables of interest for future studies on mechanisms underlying maladaptive sleep in insomnia, notably EEG-signatures of abiding LC activity during REM sleep and the transition to REM sleep.

A notable new finding as compared to previous work, is that sleep effects on learning, adaptation, and emotion regulation are not limited to the range of negligible to significantly beneficial as is often implicitly presumed of explicitly stated (Diekelmann *et al.* 2009, Van Der Werf *et al.* 2009, Mazzotti *et al.* 2014, Palmer and Alfano 2017). Our findings indicate that the typically restless sleep of people suffering from insomnia can actually inverse sleep-dependent adaptive processes and worsen next-days outcomes: a maladaptive kind of sleep. The findings provide clues to a mechanism that could perpetuate the chronic physically distressed state that is also known as hyperarousal and is a key characteristic of ID.

Our finding is relevant to unanswered questions in our understanding of mood and anxiety disorders. A common yet poorly understood clinical observation in major depressive disorder is diurnal fluctuation in mood, typically worsening across sleep (Gordijn *et al.* 1994). In bipolar depression, switches from mania into

depression mostly occur across sleep (Wirz-Justice 2008). The marked improvement that sleep deprivation can sort on depressive symptoms are most likely to occur in those that show such worsening of mood across the night (Wirz-Justice 2008). These findings support the possible existence of a maladaptive type of sleep, that could be worse than having no sleep at all. Indeed, the restless and fragmented sleep that characterizes insomnia is also seen in major depressive disorder and PTSD. Promising animal models to understand sleep characteristics and neuronal mechanisms underlying such maladaptive sleep are currently being developed (Vanderheyden *et al.* 2014), and could suggest that perturbations in REM sleep, in particular, could be the most malicious factor of maladaptive sleep. The protocol we here present can be used to complement such animal studies with e.g. overnight brain imaging studies in humans and in ID in particular.

In conclusion, our findings show that the disrupted sleep of people with insomnia impedes overnight emotion regulation. The common clinical advice to avoid distress in the evening may not only be beneficial to sleep, but also ameliorate the daytime distress that is an unresolved key part of the diagnosis of ID. A closer study of maladaptive sleep seems highly relevant for several mental disorders.

Acknowledgements

This work was supported by Neuropsychoanalysis grant 16.561.0001 of ZONMW; by VICI grant 453.07.001 of the Netherlands Organization of Scientific Research; by grant 253/2012 of the Bial Foundation; and by ERCADG-2014–671084 INSOMNIA and ERC-2016-PoC-737634 INSOMNIA BEAT IT grants of the European Research Council. The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Supplementary material

Timing of morning exposures relative to final wake-up time

We instructed the participants to complete morning exposures between 6 and 10 am. Across the mornings with exposures, final awakening time was over an hour later in normal sleepers (NS) (mean (SE) = 07:47 (00:11)), than in ID (06:42 (00:15); β (se) = –1.08 (0.31), $t(126)$ = –3.47, $p = 7.0\times10^{-4}$). On average, normal sleepers started with the task non-significantly later (08:58 (00:10)) than participants with insomnia disorder (ID) (08:25 (00:14); β (se) = –0.55 (0.30), $t(126)$ = –1.83, p = 0.07). As a result, the time awake between final awakening and task start time was about half an hour shorter in NS (1.16 (0.13) hours) than in ID (1.71 (0.19) hours; β (se) = 0.55 (0.23), $t(126)$ = 2.39, p = 0.02). Thus, the assessment was not within the sleep inertia zone. The interindividual differences in the time between final awakening and task start time did not explain perceived shame: physical component (β (se) = –0.04 (0.05), $t(126)$ = –0.88, p = 0.38), emotional component (β (se) = –0.08 (0.08), $t(126)$ = –0.92, p = 0.36), social component (β (se) = 0.007 (0.06), $t(126)$ = 0.11, p = 0.92).

No significant differences in perceived shame between morning and evening exposures

In order to assess whether diurnal changes in mood or emotional reactivity affected perceived shame, we performed mixed-effect general linear models as described by equation 3.1, but now including an indicator (i.e. dummy) variable for morning vs. evening as well as an indicator variable representing the interaction between this variable and insomnia. Perceived shame was not significantly different on morning or evening exposures: physical component (β (se) = 0.03 (0.10), $t(236)$ = 0.27, p = 0.79), emotional component (β (se) = –0.21 (0.15), $t(236)$ = –1.36, p = 0.18), social component (β (se) = –0.06 (0.11), $t(236)$ = –0.58, p = 0.56). There was also no significant interaction between time-of-day effect and group (ID vs. NS): physical component (β (se) = 0.02 (0.17), $t(236)$ = 0.10, p = 0.92), emotional component (β (se) = –0.19 (0.25), $t(236)$ = –0.76, p = 0.45), social component (β (se) = –0.06 (0.17), $t(236)$ = –0.36, p = 0.72).

Supplementary Table S3.1. No associations between sleep diary parameters and overnight changes in perceived physical shame

All *p*-values were non-significant after false-discovery rate correction.

	NS		ID	
	β	***FDR corrected p*-value**	***β***	***FDR corrected p*-value**
Bed time	0.15	0.78	-0.16	0.78
Lights out time	0.08	0.78	-0.13	0.86
Final wake time	-0.10	0.80	-0.25	0.64
Get up time	-0.06	0.78	-0.26	0.89
SOL	0.07	0.78	-0.31	0.78
Awakenings	-0.31	0.64	-0.28	0.89
WASO	-0.09	0.84	0.07	0.89
TIB	-0.20	0.64	-0.03	0.87
TST	-0.14	0.80	0.05	0.78
SE	0.02	0.64	0.06	0.78
Subjective sleep quality	0.11	0.36	-0.13	0.78

Abbreviations: NS = Normal Sleepers, ID = Insomnia Disorder.