Losing Control: Sleep Deprivation Impairs the Suppression of Unwanted Thoughts

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Abstract

Unwanted memories often enter conscious awareness when we confront reminders. People vary widely in their talents at suppressing such memory intrusions; however, the factors that govern suppression ability are poorly understood. We tested the hypothesis that successful memory control requires sleep. Following overnight sleep or total sleep deprivation, participants attempted to suppress intrusions of emotionally negative and neutral scenes when confronted with reminders. The sleep-deprived group experienced significantly more intrusions (unsuccessful suppressions) than the sleep group. Deficient control over intrusive thoughts had consequences: whereas in rested participants suppression reduced behavioural and psychophysiological indices of negative affect for aversive memories, it had no such salutary effect for sleep-deprived participants. Our findings raise the possibility that sleep deprivation disrupts prefrontal control over medial temporal lobe structures that support memory and emotion. These data point to an important role of sleep disturbance in maintaining and exacerbating psychiatric conditions characterised by persistent, unwanted thoughts.

Introduction

Memories of unpleasant experiences and thoughts can intrude into conscious awareness when we confront reminders to them. Individuals suffering from psychiatric conditions such as post-traumatic stress disorder (PTSD) and major depressive disorder (MDD) typically experience a disproportionate number of unwanted memory intrusions, and difficulties in limiting the duration and recurrence of these intrusions compound negative mood and affective dysregulation (Brewin, Gregory, Lipton, & Burgess, 2010; Mihailova & Jobson, 2018; Moritz et al., 2014; Newby & Moulds, 2011). Our capacity for inhibiting intrusive thoughts might therefore play a fundamental role in maintaining mental health and wellbeing (Gagnepain, Hulbert, & Anderson, 2017).

The ability to control intrusive memories and thoughts can be studied in the laboratory by measuring people's success at suppressing the memory retrieval process, when confronted with reminders to unwanted thoughts. For example, one widely used task, known as the Think/No-Think (TNT) paradigm (Anderson & Green, 2001), requires participants to either actively engage ('Think') or suppress ('No-Think') memory retrieval when presented with reminder cues to associated memories, often aversive images. An intrusion occurs when participants' attempts to suppress retrieval during 'No-Think' trials fail, and the reminder cue triggers an involuntary retrieval of the associated memory. Presenting a reminder cue typically elicits the intrusion of an unwanted memory less than 50 percent of the time (Gagnepain et al., 2017; Hellerstedt, Johansson, & Anderson, 2016; Levy & Anderson, 2012; van Schie & Anderson, 2017), but people vary widely in memory control ability (Levy & Anderson, 2008). The factors contributing to this variability are poorly understood. Identifying determinants of successful retrieval suppression could contribute to our understanding of vulnerability to affective disorders characterised by intrusive thoughts (Brewin et al.,

2010; Moritz et al., 2014; Streb, Mecklinger, Anderson, Johanna, & Michael, 2016), and inform the development of novel treatments.

Researchers have hypothesized that retrieval suppression ability is intrinsically linked to inhibitory control. According to the inhibitory deficit hypothesis (Levy & Anderson, 2008), individual differences in regulating intrusive memories originate from variation in underlying inhibition ability. This hypothesis predicts that conditions that strain inhibitory control will likewise undermine the ability to suppress unwanted thoughts. Sleep deprivation in healthy adults impairs both cognitive (Alhola & Polo-Kantola, 2007; Walker, 2009; Wild, Nichols, Battista, Stojanoski, & Owen, 2018) and executive (Drummond, Paulus, & Tapert, 2006; Nilsson et al., 2005) functioning, making sleep loss an important candidate factor mediating fluctuations in thought control. Indeed, mental fatigue arising from sustaining an effortful task can increase the frequency of intrusions during the TNT task (van Schie & Anderson, 2017). Critically, sleep loss disrupts functioning in the prefrontal – medial temporal lobe (MTL) networks involved in retrieval suppression (Benoit, Hulbert, Huddleston, & Anderson, 2015; Gagnepain et al., 2017; Levy & Anderson, 2012; Yoo, Gujar, Hu, Jolesz, & Walker, 2007), suggesting that losing sleep may heighten people's vulnerability to intrusive thoughts (Chee, 2004; Mazur, Pace-Schott, & Hobson, 2002; Thomas et al., 2000, 2003; van Schie & Anderson, 2017; Yoo et al., 2007). Notably, chronic sleep disturbance is a formal symptom of most psychiatric conditions, particularly PTSD (Maher, Rego, & Asnis, 2006) and MDD (Riemann, Berger, & Voderholzer, 2001).

These observations led us to consider whether the established link between psychiatric conditions and disturbed sleep may be mediated partially by sleep deficits compromising a person's ability to regulate emotion by suppressing retrieval of aversive thoughts. For example, suppressing retrieval of aversive scenes reduces

people's emotional response to those scenes later on, as revealed by changes in subjective affect ratings for the suppressed stimuli, and the relationship of those changes to prefrontally-driven downregulation of the amygdala during memory intrusions (Gagnepain et al., 2017). This impact of retrieval suppression on perceived emotion (hereinafter referred to as affect suppression) suggests that suppression contributes to affective homeostasis by reducing the negative tone of unpleasant events. If sleep loss compromises memory control, it may diminish the impact that retrieval suppression has on affective responses to unwanted thoughts, a possibility consistent with the well-documented negative consequences of sleep loss on mood (Dinges et al., 1997; Short & Louca, 2015; Zohar, Tzischinsky, Epstein, & Lavie, 2005). Whether sleep loss disrupts affect suppression arising from memory control, and whether affect suppression effects are mirrored in psychophysiological reactivity to suppressed thoughts, has never been examined.

To determine whether sleep deficits could contribute to the pathogenesis and maintenance of intrusive symptomatology, we investigated the impact of sleep deprivation on memory control in healthy young adults. Participants learned associations between faces and emotionally negative or neutral scenes before an overnight interval of sleep or total sleep deprivation (Figure 1a). The following morning, participants completed a TNT task for the face-scene associations (Figure 1b). On each trial of this task, a face was presented alone in either a green or a red frame, instructing participants either to actively retrieve ('Think') or to suppress ('No-Think') the associated scene, respectively. Attempts to suppress retrieval of the scene often initially fail, leading the scene to intrude into participants' awareness involuntarily, despite efforts to stop it. Because involuntary retrievals in this paradigm (as in real life) are unobservable events private to the individual, it was necessary to identify their

occurrence through intrusion reports, done after each individual trial. Participants reported whether the associated scene had entered awareness on the preceding trial on a three-point scale ("never", "briefly", "often"). We then quantified variations in memory control success by using the proportion of 'No-Think' trials that triggered any awareness of the associated scene, reflecting an initial failure of retrieval suppression (i.e., reports of "briefly" or "often"; hereinafter referred to as intrusions). Intrusion reports provide a validated index of involuntary retrieval. Using intrusion reports, prior functional magnetic resonance imaging studies have established that in rested individuals, intrusion trials (i.e. unsuccessful suppression attempts) trigger greater activation in the right dorsolateral prefrontal cortex (rDLPFC), greater negative coupling of rDLPFC with both the hippocampus and amygdala, and greater downregulation of activity in the latter structures, consistent with a reactive engagement of top-down inhibitory control to suppress the intrusive content (Benoit et al., 2015; Gagnepain et al., 2017; Levy & Anderson, 2012). EEG studies, moreover, establish that trials which attract intrusion ratings of "never" (i.e. successful suppression attempts), show significant increases in beta frequency power linked to successful inhibitory control, consistent with successful proactive control of retrieval (Castiglione, Wagner, Anderson, & Aron, 2019). Using these intrusion indices, we predicted that sleep-deprived participants would report more intrusions for 'No-Think' scenes than would participants who slept, and exhibit impaired ability to reduce the frequency of intrusions over time.

Studies that employ the TNT paradigm often measure the consequences of thought suppression for memory accessibility via a recall test administered after the TNT assessment phase. These studies have repeatedly shown that suppressing a memory impairs its later accessibility (i.e. suppression-induced forgetting; for review

see: Anderson & Hanslmayr, 2014). However, here our objective was to test how sleep deprivation affected the ability to downregulate unwanted thoughts and, consequently, affect suppression for the intruding content. Because measuring affect suppression required that we re-expose all of the scenes after the TNT phase, any memory test following this measurement would be contaminated by scene re-exposures. Hence, suppression-induced forgetting following active retrieval suppression was not addressed here in favour of examining affect suppression. Specifically, to investigate how the predicted failures in intrusion control following sleep loss influenced affect suppression, we acquired emotional ratings for the scenes both before the overnight delay and also after the TNT phase the following morning (Figure 1c). We also measured skin conductance responses (SCRs) to scene presentations to examine whether psychophysiological measures of sympathetic arousal mirrored suppression-induced changes in subjective affect. We predicted that retrieval suppression would attenuate both subjective and psychophysiological reactivity to negative scenes in the sleep group, limiting their tendency to induce unpleasant affect. Critically, however, if our hypothesis about the role of sleep loss in the pathogenesis of psychiatric disorders is correct, this salutary effect of suppression on negative affect should be significantly reduced in otherwise healthy participants randomly assigned to our sleep deprivation group.

We also measured the physiological correlates of memory control by collecting heart rate variability (HRV) recordings both before and after the TNT assessment phase. Spectral analysis of HRV has previously identified two reliable components: high-frequency HRV (HF-HRV; 0.15-0.40 Hz) and low-frequency HRV (LF-HRV; 0.04-0.15 Hz). Importantly, superior executive functioning has been linked to higher HF-HRV (for review, see: Thayer & Lane, 2009), including superior memory control (Gillie,

Vasey, & Thayer, 2014), whereas LF-HRV instead increases with fatigue (Tran, Wijesuriya, Tarvainen, Karjalainen, & Craig, 2009). Given these observations and given the hypothesized disruption to inhibitory control with sleep deprivation, we predicted that higher HF-HRV would be associated with superior affect suppression and intrusion control in the sleep group, but not in participants deprived of sleep. We further predicted that LF-HRV would be significantly higher following sleep deprivation than after a night of sleep, providing physiological confirmation of extreme fatigue.

Methods

Participants

Sixty healthy individuals were recruited for this study and randomly assigned to a sleep group (*n*=30) or a sleep deprivation group (*n*=30). Of these participants, one was excluded for failing to follow task instructions. The reported data relate to the remaining fifty-nine participants (sleep group: *n*=29, 13 male, mean age = 19.79 years, SD = 1.63 years; sleep deprivation group: *n*=30, 12 male, mean age = 20.20 years, SD = 1.75 years) who participated in return for £40 payment or BSc Psychology course credit (University of York). Participants reported no history of neurological, psychiatric, attention, or sleep disorders, and typically maintained a regular sleep/wake pattern. We requested that participants refrain from consuming alcohol or caffeine for 24 h prior to the experiment. Wristwatch actigraphy was used to ensure that participants did not nap during the day preceding the overnight phase. Written informed consent was obtained from all participants in line with the requirements of the University of York's Department of Psychology Research Ethics Committee, who approved the study.

Stimuli

Fourty-eight emotionally neutral face images (half male, half female) served as cues in the TNT task. An equal number of scene images (half negative, half neutral) selected from the International affective picture system (IAPS; Lang, Bradley, & Cuthbert, 1997) served as targets. Negative scenes received lower affect ratings compared to neutral scenes from participants in the current study during the first affect evaluation task [$F(1,57) = 630.89$, $p < .001$, $\eta_p^2 = .92$], demonstrating that the scene images elicited the expected emotional response. Face-scene pairs were created by randomly assigning each face cue to a target scene. Three lists of 16 pairs (8 negative; 8 neutral) were created from the 48 pairs to allow three within-subjects TNT conditions ('Think', 'No-Think', 'Baseline'). The assignment of pairs to TNT conditions was counterbalanced across participants for each of the sleep groups. Twelve additional face-scene pairs (6 negative; 6 neutral) were created to serve as fillers that were also used in the practice phases. The same 60 scene images (48 experimental + 12 practice) were also used for the affect evaluation tasks, which featured an additional 8 filler scenes (4 negative, 4 neutral).

Procedure

A schematic representation of the study procedure is shown in Figure 1a. Participants completed two experimental sessions that were separated by an overnight delay containing either sleep or sleep deprivation. At 09:00 on the first experimental day, participants collected an actigraphy watch that they wore until 20:15 when they arrived at the University of York's Emotion Processing and Offline Consolidation (EPOC) laboratory.

Affect evaluation task

Participants provided emotional ratings for 68 IAPS images using a pictorial scale that ranged from a frowning face on the far-left side of the scale to a smiling face on the far-right side of the scale (Figure 1c). On each trial, an image was presented for 6.5 s. Participants were asked to focus their attention on the image for the entire time it was on the screen. Following a 2 s blank screen delay, the affect rating scale was displayed until participants provided their rating on a scale from 1 (corresponding to the frowning face on the far-left side of the scale) to 9 (corresponding to the smiling face on the far-right side of the scale) using a single key press. Participants were told that the extreme left side of the scale should be used for pictures that made them feel *completely* unhappy, annoyed, unsatisfied, melancholic, despaired or bored, and the extreme right side of the scale should be used for pictures that made them feel *completely* happy, pleased, satisfied, contented and hopeful. Participants were given 15 s to provide their affect rating for each picture, although they were asked to respond quickly and spontaneously. The trial terminated once an affect rating had been provided or the 15 s time-limit expired, and participants then viewed a blank screen for 5 s, followed by a fixation cross for a random interval of either 0.5 s, 1 s, 1.5 s, or 2 s, followed by the next image. This pattern continued until all scenes had been viewed and rated. The affect evaluation task was repeated after the TNT assessment phase. Skin conductance responses (SCRs) were recorded throughout the affect evaluation tasks.

TNT task

Next, participants completed the learning phase of the TNT task. Participants initially encoded face-scene pairs by studying them for 6 s, one at a time. To reinforce learning and ensure adequate knowledge of the face-scene pairs, participants then completed

a test phase. Here, faces were displayed individually for up to 4 s, and participants indicated whether or not they were able to *fully visualize* the associated scene image. If the participant indicated that they were indeed able to visualize the associated scene, they were next presented with the correct scene alongside two additional foil scenes that they had seen previously in the learning phase but were not paired with that particular face. Participants were required to select the scene associated with the face. If the participant failed to identify the correct scene, or they indicated that they could not visualize the scene associated with the face, their memory for the face-scene pair was probed again later in the test phase. Regardless of whether or not the participant was able to correctly identify the scene paired with a particular face, the correct face-scene pairing was presented for 3.5 s after each trial. Participants were instructed to use this feedback as an opportunity to reinforce their knowledge of the pairs. The test phase continued until each face-scene pair had been correctly identified once. Following completion of the test phase, a second, identical, test phase was administered to reinforce learning. This overtraining procedure was employed to ensure that participants would experience difficulty in preventing scene images from intruding into consciousness when presented with face cues on 'No-Think' trials of the TNT assessment phase. However, the overtraining procedure also precluded any meaningful examination of suppression-induced forgetting due to ceiling effects (Gagnepain et al., 2017). Before the overnight delay, participants carried out a mock version of the TNT assessment phase to ensure that they had opportunity to practice inhibiting memory intrusions before completing the task proper. In the mock task, participants completed 24 trials (12 'Think', 12 'No-Think') which used 12 filler items (each suppressed or retrieved twice) that were different to those used in the TNT

assessment phase proper. Participants then went to sleep or remained awake overnight – details about the overnight delay are provided below.

The following morning, participants completed a memory refresher phase in which the face-scene pairs were presented at a rate of 1.5 sec per pair, offering participants the opportunity to reinforce their knowledge of the pairs. The TNT assessment phase proper was then administered in five blocks, each lasting approximately 8 min. During each block, two repetitions of 16 'Think' (8 negative and 8 neutral face cues) and 16 'No-Think' (8 negative and 8 neutral face cues) items were presented in pseudorandom order, with the two repetitions of each item appearing at least three trials away from each other. Accordingly, participants completed a total of 320 trials (32 trials x 2 conditions x 5 blocks). Face cues appeared inside a green frame on 'Think' trials and inside a red frame on 'No-Think' trials. For green framed faces, participants were instructed to visualize, in as much detail as possible, the scene associated with the face for the entire 3 s it was on the screen. For red framed faces, participants were instructed to focus their attention on the face for the entire 3 s, but simultaneously prevent the associated scene from coming to mind at all (Figure 1b). They were told to accomplish this by making their mind go blank, rather than by replacing the unwanted scene with another image, thought or idea. If the scene came to mind automatically, participants were asked to actively push the scene out of mind.

Immediately after each face cue presentation, participants reported the extent to which they thought about the associated scene by pressing a key corresponding to one of three options: "never", "briefly", and "often". Participants were given 10 s to make this rating, however, they were instructed to provide their rating quickly without dwelling too much on their decision. The trial terminated once an intrusion rating had been provided. These intrusion ratings were collected to ascertain how competent

participants were at suppressing scenes associated with faces for 'No-Think' trials. Although we collected intrusion reports on a 3-point scale (never, briefly, often) for every trial, in practice, people rarely give "often" ratings in 'No-Think' trials (M = 3.15% of 'No-Think' trials in the current study, SEM = 0.62%). For simplicity, we therefore followed prior work, combining "briefly" and "often" responses (Benoit et al., 2015; Gagnepain et al., 2017; Hu, Bergström, Gagnepain, & Anderson, 2017; van Schie & Anderson, 2017), rendering the judgment binary (intrusion or non-intrusion). Participant responses were followed by a jittered fixation cross lasting between 0.5 and 9 s, before the next face cue was presented. Note that 16 scenes that were included in the TNT learning and test phases, and the affect evaluation tasks, did not appear in the TNT assessment phase. These 'Baseline' items provided an estimate of generalised changes in emotional affect that could be directly compared with changes resulting from mnemonic suppression (i.e. 'No-Think' scenes). Immediately before and after the assessment phase of the TNT task, an 8 min ECG recording was collected for the purposes of obtaining resting HRV.

Overnight delay

Electrodes were attached to participants who had been assigned to the sleep group following completion of the first experimental session (i.e. after the TNT learning phase) to allow for overnight polysomnographic recording. Lights were turned out at approximately 22:45 and participants were awoken at 06:45, providing an 8 h sleep opportunity. Before the morning session began, participants were given the opportunity to shower and eat breakfast.

Participants in the sleep deprivation condition remained awake throughout the overnight delay under the supervision of at least one researcher. Participants were tested in groups of two or three, who were allowed to communicate, read, use the PC,

watch TV, or play games. Snacks were provided, but participants were not permitted to consume caffeine. Once the second session began, participants had been awake for > 24 h.

Equipment and data processing

Behavioural

All aspects of the TNT task were written and implemented using Presentation 20.0 (Neurobehavioral Systems, Albany, CA, USA). The affect evaluation task was administered with E-Prime 2.0 (Psychology Software Tools, Pittsburgh, PA, USA). Behavioural tasks ran on a desktop computer and visual aspects were displayed on a 20" flat-screen monitor. Participant responses were collected using the computer keyboard. Behavioural data were analysed using IBM SPSS Statistics 24 (SPSS Inc., Chicago, IL, USA).

Intrusion proportion. In accordance with previous studies (e.g. Benoit et al., 2015; Gagnepain et al., 2017; Hu et al., 2017; van Schie & Anderson, 2017), intrusion ratings provided during the TNT task were used to classify each trial as either eliciting an intrusion (i.e. "briefly" or "often" responses) or not (i.e. "never" responses), in a binary fashion. Unwanted retrieval events were separated into "briefly" and "often" in the TNT assessment phase to ensure that all intrusions, irrespective of persistence or fortitude, were reported. Using these binary intrusion ratings, we calculated the proportion of trials that evoked a memory intrusion, separately for each participant, trial block (1-5), TNT condition ('Think', 'No-Think'), and image valence (neutral, negative). Overall intrusion proportion scores were also calculated by averaging mean intrusion proportion scores across trial blocks, separately for each TNT condition and image valence.

Intrusion slope scores. To measure changes in intrusion frequency for 'No-Think' trials across TNT blocks, we calculated proportionalised intrusion slope scores, separately for each participant and image valence (neutral, negative). Slope scores were calculated by taking the slope of the intrusion frequencies across the five TNT trial blocks. This value was divided by intrusion frequency in the first block, to account for the fact that initial intrusion rates varied and participants with more initial intrusions had more room to decrease their intrusion frequency. We then multiplied the values by -1 to render the (primarily) negative scores as positive, with increasingly positive scores reflecting increasing levels of control at downregulating the frequency of intrusions. In accordance with previous research (Hellerstedt et al., 2016; Levy & Anderson, 2012), this measure was z-normalized within that participant's TNT pair list counterbalancing group. Z-normalizing within each counterbalancing group allows us to quantify a participant's intrusion slope score with respect to a group of participants receiving precisely the same items in the TNT task. To investigate the correlation between total sleep time and intrusion slopes in the sleep group, both measures were z-normalized within each counterbalancing group without including the sleep deprivation group.

Relapse probability. Each item that appeared in the TNT task was presented 10 times (twice in each TNT block). This repetition allowed us to investigate how well a participant's efforts to suppress a particular item on one trial then carries forward to the next trial involving that same item. The frequency of relapses for 'No-Think' items was calculated as the number of 'No-Think' trials in repetitions 2-10 where an item elicited an intrusion, but that specific item had been successfully suppressed on its immediately preceding presentation. This was done separately for each participant, trial transition (1 to 2, 2 to 3 … 9 to 10), and image valence (neutral, negative). Each

value was divided by the frequency of successfully suppressed items (i.e. non-intrusions) during the first repetition of that trial transition (e.g. repetition 3 of transition 3 to 4) to produce relapse probability scores (van Schie & Anderson, 2017). This was to account for the fact that individuals with fewer intrusions on the first repetition of each trial transition had more relapse potential.

Affect suppression. Affect ratings gathered during the affect evaluation tasks were used to measure overnight changes in subjective emotional reactivity to the scene images. Mean affect rating values were calculated for each participant, session (pre-delay, post-delay), TNT condition ('Think', 'No-Think', 'Baseline'), and image valence (neutral, negative). Affect suppression scores were then calculated by subtracting the averaged values at session one from those at session two. To account for individual differences in initial emotional responses, affect suppression scores in each condition were divided by the mean affect rating at session one to produce proportionalised affect suppression scores. Greater scores reflect more positive affect evaluations at session 2 compared to session 1.

Electrodermal activity

Electrodermal activity was recorded using a BIOPAC MP36R data acquisition system and AcqKnowledge (ACQ) 4.4.1 software (sampling rate = 2KHz). During the affect evaluation tasks, E-Prime-triggered square pulse outputs were transmitted to the MP36R unit via a BIOPAC STP35A interface enabling precise alignment of each stimulus onset to the SCR data. Two BIOPAC EL507 disposable adhesive electrodes were attached to the fingertips of the index and middle fingers of the non-dominant hand. The data were imported and preprocessed using PsPM (version 4.0.2; Bach & Friston, 2013).

A unidirectional first-order Butterworth high-pass filter with cut-off frequency 0.05 Hz was used to filter the data to account for the change of baseline activity during the duration of the recording sessions. The time series averaged over corresponding trials for each experimental condition (e.g. 'No-Think', negative valence) were then extracted, for each subject, for each of the two sessions (pre-TNT and post-TNT). An average 'session-specific' skin conductance level (SCL) was computed for each subject for each session. SCL was the skin conductance value measured for the first second after the presentation of the stimuli, averaged across all conditions. This first 1 s period after stimulus presentation is widely considered to be the 'latency' period for event-related evoked SCRs (Bach, Flandin, Friston, & Dolan, 2010; Braithwaite, Watson, Robert, & Mickey, 2013; C. L. Lim et al., 1997). This 'baseline' SCL was then subtracted from the rest of the measured skin conductance activity, which was deemed to belong to a canonical evoked SCR, taken for the entirety of the time that the stimulus was presented on screen during a trial, after excluding the first second (i.e. 5.5 seconds). The area under the curve for each condition was then computed for each subject, for each of the two sessions. This is akin to an analysis approach shown earlier for spontaneous skin conductance fluctuations, except here we have used it to analytically quantify event-related evoked SCRs (Bach, Friston, & Dolan, 2010). The average SCRs elicited by images in each TNT condition and valence category were used to calculate the difference in SCRs across sessions (dSCR; SCR post-TNT – SCR pre-TNT). The same analysis pipeline was used for both the sleep and the sleep deprivation groups. Data from 2 participants were unavailable due to technical issues (sleep group n=1; sleep deprivation group n=1). Furthermore, we excluded data from 3 participants in the sleep group who were SCR non-responders.

Heart rate variability

ECG recordings were administered before and after the TNT assessment phase for the purposes of resting HRV assessment. Three BIOPAC EL503 ECG electrodes were attached to the midline of the left and right clavicle and the lower left rib. ECG was recorded for eight successive minutes. The first 2 min and last 1 min of each recording was discarded, and HRV was calculated for the remaining five successive minutes. The ECG signal was analysed offline using ACQ and Kubios Standard 3.0.2 software.

R-peaks were automatically detected using ACQ's QRS detection algorithm and visually inspected for accuracy. Peaks that the algorithm missed were inserted manually. The interbeat-interval time series was then imported to Kubios for analysis. To obtain frequency-domain-specific indices of HRV we used autoregressive estimates of low-frequency (0.04-0.15 ms^2/Hz) and high-frequency (0.15-0.40 ms^2/Hz) power. Autoregressive algorithms are generally preferable to Fourier transform based algorithms for spectral analysis of HRV(Thayer, Hansen, & Johnson, 2008), partly because they have better spectrum resolution when using short data frames (Miranda et al., 2012). LF-HRV reflects both sympathetic and parasympathetic control over heart rate, whereas HF-HRV reflects parasympathetic or vagal modulation (Laborde, Mosley, & Thayer, 2017). In accordance with previous research (Gillie et al., 2014; Park, Vasey, Van Bavel, & Thayer, 2014), values of LF-HRV and HF-HRV were transformed logarithmically (base 10). One participant in the sleep group exhibited atypical ECG patterns, and was thus removed from HRV analyses.

Polysomnography

Sleep monitoring was carried out using an Embla N7000 PSG system (Embla Systems, Broomfield, CO, USA). Gold-plated electrodes were attached using EC2 electrode cream after the scalp was cleaned with NuPrep exfoliating agent. Scalp electrodes were attached at eight standard locations according to the international 10-

20 system (Homan, Herman, & Purdy, 1987): F3, F4, C3, C4, P3, P4, O1, and O2, each referenced to the contralateral mastoid (A1 or A2). Left and right electrooculogram, left, right and upper electromyogram, and a ground electrode (forehead) were also attached. All electrodes were verified to have a connection impedance of < 5 kΩ. All signals were digitally sampled at a rate of 200 Hz.

Sleep data was divided into 30 s epochs and scored as wakefulness, N1 sleep, N2 sleep, slow-wave sleep (SWS) or rapid eye movement (REM) sleep according to standardised criteria (Iber, Ancoli-Israel, Chesson, & Quan, 2007), using REM Logic 3.4. PSG data was unavailable for 4 participants due to reference electrodes becoming detached during the night.

Data analysis

Intrusion control measures were analysed for 'No-Think' trials with a 2 (Valence: Negative/Neutral) x 2 (Group: Sleep/Sleep Deprivation) mixed ANOVA. Analyses of intrusion proportion and relapse probability featured the additional factors Trial Block (1-5) and Trial Transition (1 to 2, 2 to 3… 9 to 10), respectively. Affect suppression was analysed for negative images with a 2 (TNT Condition: 'No-Think'/'Baseline') x 2 (Group: Sleep/Sleep Deprivation) mixed ANOVA. Subjective and physiological measures were analysed using identical techniques. HRV measures were analysed using a 2 (Time: Pre-TNT/Post-TNT) x 2 (Group: Sleep/Sleep Deprivation) ANOVA. Note than ANOVA is robust against deviations from normality (Glass, Peckham, & Sanders, 1972; Harwell, Rubinstein, Hayes, & Olds, 1992; Schmider, Ziegler, Danay, Beyer, & Bühner, 2010). Where appropriate, ANOVAs were followed up with post-hoc t-tests. All reported correlations used Pearson's correlation coefficient. Where relevant, Fisher's *Z* transformation was used to compare correlations.

Results

Sleep deprivation impairs the suppression of intrusive thoughts

First, we analysed how well participants controlled intrusive thoughts and investigated whether this was affected by sleep. Overall intrusion scores were robustly higher for 'Think' trials (M = 92.76% of trials, SEM = 1.38%) compared to 'No-Think' trials (M = 28.88% of trials, SEM = 2.52%) [$t(58) = 19.73$, $p < .001$, $d_z = 2.57$], confirming that participants largely succeeded in suppressing thoughts about the scenes associated with red-framed faces. Because we primarily sought to examine intrusions for scenes that participants attempted to suppress, our analyses focus on 'No-Think' trials hereinafter, unless otherwise stated.

Of key interest is whether depriving participants of sleep disrupted their ability to stop unwanted thoughts from coming to mind, given reminders. Critically, sleep-deprived participants reported more intrusions than did the sleep group [$F(1,57) = 5.55$, $p = .022$, $\eta_p^2 = .09$], demonstrating that sleep deprivation impairs memory control (Figure 2a). Strikingly, the sleep deprivation group suffered a proportional increase in intrusions of nearly 50% relative to the rested group, revealing how deficient control may be a pathway to hyper-accessible thoughts. Participants overall showed decreasing intrusions across TNT trial blocks [$F(3.16,180.33) = 29.81$, $p < .001$, $\eta_p^2 = .34$; Greenhouse-Geisser corrected] (Figure 2b), indicating that repeatedly inhibiting retrieval was, in general, highly effective at stopping memories from intruding on future trials. Intrusion rates were comparable for negative and neutral scenes [$F(1,57) = 0.11$, $p = .738$], and the detrimental impact of sleep deprivation on intrusion rate did not vary with the valence of scenes [all $p > .05$].

Although suppression robustly reduced intrusions over blocks across all participants, we were also interested in examining whether sleep deprivation altered

the rate at which suppression attempts led intrusions to decline. Consistent with deficient inhibitory control, depriving participants of sleep significantly disrupted their ability to downregulate intrusive thoughts over blocks compared to participants who had slept, as reflected in lower intrusion slope scores [$F(1,55) = 9.38$, $p = .003$, $\eta_p^2 = .15$; Figure 2c]. Slope scores did not differ between negative and neutral scenes [$F(1,55) = 0.08$, $p = .777$], and the intrusion slope deficit in the sleep deprivation group did not vary with scene valence [$p > .05$]. Within the sleep group, total sleep time predicted intrusion slopes, with participants sleeping longer showing a trend towards better ability to reduce intrusions over repetitions [$r = -.349$, $p = .08$].

We next considered whether sleep-deprived participants were more vulnerable to intrusion relapses for scenes that they had previously suppressed, as compared to participants that had slept. Sleep-deprived participants exhibited significantly higher relapse probability scores than did the sleep group [$F(1,57) = 7.08$, $p = .010$, $\eta_p^2 = .11$; Figure 2d], indicating that after initially gaining control over an intrusive thought, they suffered intrusion relapses more than participants who had slept. As with intrusions overall, relapses also became less frequent across trial transitions [$F(6.39,364.12) = 2.49$, $p = .020$, $\eta_p^2 = .04$; Greenhouse-Geisser corrected; Figure 2e]. Relapse probability was comparable for negative and neutral memories [$F(1,57) = 0.07$, $p = .800$], and sleep deprivation affected negative and neutral relapses to a similar degree [$p > .05$]. Sleep stage data is available in Table 1.

Sleep deprivation nullifies affect suppression

Although the foregoing findings show that sleep deprivation disrupts intrusion control, they do not address whether losing sleep alters how suppression affects emotional responses. Recent work indicates that successfully suppressing negative scenes renders them less aversive when they are later re-encountered (Gagnepain et al.,

2017). Building on this finding, we tested whether sleep deprivation affected the relationship between retrieval suppression and our behavioural index of affect suppression. Behavioural affect suppression refers to the difference in affect ratings of scenes between the first (pre- delay and TNT) and second (post- delay and TNT) session. Positive affect suppression scores indicate that participants felt less negative about the scenes in the second affect evaluation task.

Consistent with earlier reports using this procedure (Gagnepain et al., 2017), we found that, across all participants, successful retrieval suppression (i.e. fewer intrusions) was associated with greater affect suppression for negative scenes [r = -.32, p = .015; Figure 3a]. In other words, greater success at suppressing retrieval of aversive memories predicted a larger change in their perceived affect, rendering them less negative when participants next encountered the scenes. We observed no such relationship for neutral 'No-Think' scenes, or 'Think' scenes of either valence type [all p > .05]. The relationship between intrusion control and affect suppression did not differ significantly between the sleep [r = -.33, p = .079] and sleep deprivation groups [r = -.17, p = .367; Z = 0.62, p = .533].

Having found evidence of a general relationship between intrusion control and affect suppression specifically for negative 'No-Think' scenes, we next tested whether sleep deprivation undermines suppression-induced regulation of negative affect for aversive scenes. Importantly, one-third of the scenes that were rated during the affect evaluation tasks did not appear in the TNT assessment phase ('Baseline' scenes). Cross-session affect change scores for 'Baseline' scenes index generalised overnight changes in emotional reactivity, which can be directly compared to those resulting from memory suppression (i.e. for 'No-Think' scenes). We observed a significant interaction between TNT condition ('Baseline', 'No-Think') and group [F(1,57) = 5.38, p = .024,

η_p^2 = .09]. Whereas affect change scores for aversive 'No-Think' scenes were greater in the sleep group than in the sleep deprivation group [t(57) = 2.56, p = .013, d = 0.68], we observed no difference for aversive 'Baseline' scenes [t(57) = 0.24, p = .809, d = 0.07; Figure 3b]. In the sleep group, affect change scores for negative 'No-Think' scenes were marginally more positive than those for negative 'Baseline' scenes [t(28) = 1.76, p = .089, d_z = 0.35], consistent with the view that suppression mitigates unpleasant affect in rested individuals. We observed no such difference for the sleep deprivation group [t(29) = 1.50, p = .145, d_z = 0.27].

The foregoing findings indicate that suppression reduced the perceived valence of suppressed negative scenes. However, they do not speak to whether suppression alters sympathetic arousal, and whether sleep loss impacts this form of regulation. To probe this question, we analysed dSCR scores, which refer to the difference in SCRs across sessions arising for negative 'Baseline' and 'No-Think' scenes. Negative dSCR scores reflect a decrease in SCR towards the scenes in the second affect evaluation task.

As with our behavioural index of affect suppression, we observed a significant interaction between TNT condition ('Baseline', 'No-Think') and group on dSCR scores [F(1,52) = 4.05, p = .049, η_p^2 = .07; Figure 3c]. Participants who slept exhibited a decrease in dSCR towards the negative 'No-Think' scenes compared to 'Baseline' scenes [t(24) = 2.07, p = .049, d = 0.41]. This finding is consistent with our affect suppression data, and further supports the view that successfully suppressing unpleasant memories alleviates their associated emotional charge. Sleep-deprived participants, who were less competent at controlling intrusions, exhibited comparable dSCR scores for 'Baseline' and 'No-Think' scenes [t(28) = 1.08, p = .290]. These findings suggest that the affective benefits of retrieval suppression for rested

participants were not confined to subjective reports of perceived affect, but also arose for indices of psychophysiological emotional arousal. Sleep deprivation abolished the affective benefits of suppression, and this was evident in both our behavioural and psychophysiological measures. Interestingly, we observed no reliable correlation between individual differences in our behavioural and psychophysiological suppression measures for negative 'No-Think' scenes, suggesting that the indices tap partially distinct processes [r = 0.09, p = .522]. The observed change in psychophysiological reactivity for 'No-Think' scenes in the sleep group likely reflects diminished sympathetic nervous system responses to suppressed stimuli, possibly initiated by the impact of retrieval suppression on activity in the amygdala (Gagnepain et al., 2017), suggesting tangible effects of suppression on stress responses.

Heart rate variability is linked to affect suppression and fatigue

Inhibitory control over cognition is linked to HF-HRV (Gillie et al., 2014). This finding led us to investigate whether the degree of affect suppression that a given participant could achieve is linked to their HF-HRV, and whether any relationship is altered by sleep loss. Indeed, higher HF-HRV scores (log base 10 transformed HF-HRV collected prior to the TNT phase) predicted greater affect suppression scores for negative 'No-Think' scenes when participants were well rested [sleep group, r = .44, p = .021; Figure 3d]. This relationship was absent in the sleep deprivation group, suggesting that sleep deprivation may reduce the involvement of control processes related to HF-HRV [r = .11, p = .577; Z = 1.30, p = .097]. We observed the same pattern using the HF-HRV measure collected after the TNT phase, though to a lesser degree (sleep group [r = .34, p = .077]; sleep deprivation group [r = .07, p = .724; Z = 1.03, p = .152]). In contrast, we found no relationship between HF-HRV (pre-TNT or post-TNT) and affect changes for negative 'Think' or 'Baseline' scenes, or for neutral scenes in any TNT condition,

for either group [all $p > .05$]. We found no relationship between HF-HRV scores and overall intrusion proportion for 'No-Think' scenes [both groups $p > .05$]. The LF-HRV component was not associated with affect suppression or overall intrusion proportion in any TNT condition for either group [all $p > .05$].

LF-HRV has been related to both sleep deprivation (Zhong, 2005) and mental fatigue (Egelund, 1982; Tran et al., 2009; Zhao, Zhao, Liu, & Zheng, 2012). Confirming this relationship, participants deprived of sleep exhibited higher LF-HRV scores (log base 10 transformed LF-HRV) than did those in the sleep group [$F(1,56) = 6.71$, $p = .012$, $\eta_p^2 = .11$; Figure 3e]. Along similar lines, we found significantly higher LF-HRV following the TNT phase than we did preceding it [HRV scores pre-TNT vs post-TNT: $F(1,56) = 17.96$, $p < .001$, $\eta_p^2 = .24$], reflecting task-induced fatigue, given the ~40 min duration of the TNT task (van Schie & Anderson, 2017). We observed no interaction between these factors [$p > .05$].

Interestingly, performing the TNT task also affected HF-HRV: HF-HRV was higher after the TNT task than before it [$F(1,56) = 19.62$, $p < .001$, $\eta_p^2 = .26$]. An interaction between session and group [$F(1,56) = 9.39$, $p = .003$, $\eta_p^2 = .14$] drove this difference. Whereas completing the TNT task elevated HF-HRV in the sleep group [$t(27) = 5.54$, $p < .001$, $d_z = 1.05$], it did not in the sleep deprivation group [$t(29) = 0.93$, $p = .358$, $d_z = 0.17$], primarily because HF-HRV was already high at the outset. Overall, the groups did not differ reliably in HF-HRV [$F(1,56) = 2.26$, $p = .138$]. These data suggest that fatigue may increase HF-HRV, but less reliably than LF-HRV.

Discussion

Our findings indicate that sleep deprivation substantially increases people's vulnerability to unwanted memories intruding into conscious awareness when they

confront reminders. Remarkably, sleep-deprived participants exhibited a nearly fifty percent proportional increase in intrusions relative to participants that had slept. Moreover, sleep deprivation diminished the cumulative benefits of retrieval suppression for downregulating subsequent intrusions, showing that it compromises the ability to gain control over intrusive thoughts, even after repeated efforts to do so. Indeed, even after sleep-deprived participants initially gained control over unwanted memories and prevented them from intruding, they were consistently more susceptible to relapses when reminders were confronted again later, relative to rested participants. These findings help to bridge the gap in our understanding of how sleep disturbance could contribute to the onset and maintenance of psychiatric disorders.

Deficient memory suppression following sleep loss might arise from dysfunction of the neural networks that govern inhibitory control. Retrieval suppression engages the rDLPFC, which is thought to downregulate recollection-related activity in MTL areas via inhibitory, top-down mechanisms (Benoit et al., 2015; Gagnepain et al., 2017; Levy & Anderson, 2012). The functional integrity of the rDLPFC may be particularly vulnerable to sleep loss (Mazur et al., 2002). Functional connectivity from the medial PFC (mPFC) to MTL appears to be disrupted in sleep-deprived individuals when viewing negative emotional images, potentially compromising a pathway of inhibitory control over affect (Yoo et al., 2007). Moreover, prolonged sleep restriction impairs performance in attention and working memory tasks that rely on prefrontal engagement (Chee & Choo, 2004; Frenda & Fenn, 2016; J. Lim & Dinges, 2008; Thomas et al., 2000). In the current study, sleep deprivation may have disrupted functional interactions between the rDLPFC (and possibly mPFC) and MTL structures such as the hippocampus and amygdala during retrieval suppression, impairing inhibitory control over memory and affect; increasing intrusions, and decreasing affect

suppression. Future research can employ functional brain imaging to address this theoretical hypothesis.

Importantly, sleep deprivation also disrupted people's ability to reduce the affective content of intruding thoughts through retrieval suppression. In examining this question, we first confirmed prior evidence showing that retrieval suppression reduces the negative affect reported for unpleasant scenes: across all participants, we found that the more successfully people controlled unpleasant intrusions, the greater was their affect suppression for the suppressed scenes on self-report measures of valence. Extending these findings, we further showed that, in rested participants, affect suppression effects arose in psychophysiological reactivity, with suppressed scenes eliciting significantly reduced skin conductance responses compared to scenes that were not suppressed, consistent with a reduction in sympathetic arousal. These behavioural and psychophysiological effects align well with prior work showing that indices of affect suppression are predicted by downregulation of amygdala activity by the prefrontal cortex during memory intrusions (Gagnepain et al., 2017), a process that could underlie both effects. Critically, when our sleep-deprived participants tried to suppress negative scenes, they showed markedly less affect suppression than did rested participants on both behavioural and psychophysiological measures. Indeed, on psychophysiological indices, the sleep-deprived group exhibited numerically increased reactivity, raising the possibility that suppression may be counterproductive for people suffering sleep loss. Taken together, these findings suggest that difficulties engaging inhibitory control to regulate unpleasant intrusions after sleep loss contributed to diminished affect regulation for the suppressed content.

The possibility that sleep deprivation compromised affect suppression by altering prefrontal control involvement receives indirect support from

psychophysiological indices. Among participants who slept normally, higher HF-HRV predicted better affect suppression for negative 'No-Think' scenes; this relationship was not found, however, after sleep deprivation. Critically, prior evidence links higher HF-HRV to better inhibitory control (for review, see: Thayer & Lane, 2009), including evidence relating this measure to memory control (Gillie et al., 2014). HF-HRV has been hypothesized to reflect the engagement of brain regions supporting cognitive and affective downregulation during memory inhibition, and in healthy participants blood flow in the right prefrontal cortex correlates with HF-HRV (Lane et al., 2009). Notably, however, in the current data, higher HF-HRV did not predict fewer intrusions for 'No-Think' scenes. The selective relationship of HF-HRV and affect suppression may indicate that this measure better indexes the efficiency of those components of the inhibitory control pathway that are uniquely tied to affect regulation (e.g. connectivity between mPFC and amygdala: Yoo et al., 2007). In the sleep deprivation group, extreme fatigue may have compromised these components, eliminating the relationship between HF-HRV and the emotional aftereffects of suppression, a possibility supported by research on the relationship between sleep deprivation, HF-HRV and cognitive performance (Quintana et al., 2017).

Prior work suggests that in healthy participants, acute sleep deprivation increases LF-HRV (Zhong, 2005). Replicating this prior work, our sleep-deprived participants exhibited higher LF-HRV compared to participants who slept. LF-HRV is produced by both sympathetic and parasympathetic systems, whereas HF-HRV reflects just parasympathetic activity (Shaffer & Ginsberg, 2017). Given that HF-HRV was not altered by our sleep/wake manipulation, our results suggest that sleep deprivation elevates sympathetic arousal. Previous studies have also shown that mental fatigue elicits an increase in LF-HRV (Egelund, 1982; Tran et al., 2009; Zhao

et al., 2012). Consistent with these studies, completing our cognitively-demanding retrieval suppression task elevated LF-HRV in our sample. These data suggest that measuring resting cardiovascular activity may provide a strategy for objectively predicting how well an individual will perform in a given task by indexing how well-rested or mentally fatigued they are. This finding has important implications for managing fatigue in the workplace, which are discussed in more detail below.

We found no evidence that negative scenes intruded more often than did neutral scenes, despite a highly robust difference in perceived valence reported by our participants (see Methods). This finding might at first seem counter-intuitive. However, following previous work in which the same result was observed (Gagnepain et al., 2017), we ensured that the initial training level of negative and neutral materials was carefully matched, a feature of our protocol that may nullify the benefits of emotional arousal on memory encoding and consolidation (Canli, Zhao, Brewer, Gabrieli, & Cahill, 2000; Ritchey, Dolcos, & Cabeza, 2008). Specifically, we trained participants to perfect accuracy for all face-scene pairs before the suppression phase. Failure to match initial training on negative and neutral materials in general may explain prior mixed findings regarding the relation between valence and forgetting; whereas some studies report enhanced suppression for negative relative to neutral stimuli (Depue, Banich, & Curran, 2006; Lambert, Good, & Kirk, 2010), others observed no difference (van Schie, Geraerts, & Anderson, 2013) or even the opposite effect (Nørby, Lange, & Larsen, 2010). Importantly, taken together with earlier reports (Gagnepain et al., 2017), these findings suggest that increased intrusion frequency of aversive memories (relative to neutral memories) in research on emotional memory does not reflect any intrinsic difficulty in suppressing emotional intrusions, but instead reflects superior encoding of emotional memories. When negative and neutral scenes are matched on

encoding, intrusions of each type are comparably frequent and suppressible, at least in these studies. Our findings show further that depriving people of sleep disrupts their ability to suppress both neutral and unpleasant intrusions.

Although impaired memory control and affect suppression following sleep deprivation is a valid interpretation of our data, another possibility relates to overnight memory processing. Previous work has shown that sleep promotes the consolidation of procedural skills required to complete perceptual and motor tasks (for review, see: Stickgold, 2005). As such, intrusion control may have been impaired in the sleep deprivation group as these participants did not have an opportunity for sleep-associated consolidation of the mnemonic suppression skills learned during the evening TNT practice. Although this interpretation is possible, participants were only given very brief practice with the TNT task (12 'No-Think' trials on 6 filler items) in the first session, making it an unlikely source of the substantial sleep advantage in intrusion control that we observed. Moreover, no evidence currently exists to show that intrusion control, as a procedural skill, can be developed and transferred to entirely novel suppression targets, making this possibility necessarily speculative. Nevertheless, future work should seek to distinguish the relative contributions of procedural consolidation and disrupted inhibitory control to greater intrusiveness after sleep deprivation.

Prior work suggests that sleep curtails spontaneous intrusive thoughts in the aftermath of trauma. Indeed, sleeping soon after exposure to a traumatic film clip reduces the number of spontaneous intrusions relating to that clip in the following week (Kleim, Wysokowsky, Schmid, Seifritz, & Rasch, 2016). Furthermore, greater sleep disturbance following a road traffic accident has been associated with more frequent spontaneous, accident-related thoughts during the first week post-trauma (Luik,

Iyadurai, Gebhardt, & Holmes, 2019). Although these studies associate sleep loss to variations in intrusion frequency following adverse events, they do not address the mechanistic basis of these associations. Sleep following a traumatic film, for example, could reduce intrusions over the following week by enabling better memory control, or, alternatively, by facilitating sleep-related changes to the memory trace that make it less likely to intrude. As such, prior work has not established causal evidence concerning whether sleep deprivation impairs retrieval suppression of specific, unwanted thoughts. Because we randomly assigned participants to the sleep or sleep deprivation conditions, and because we used a procedure that demands the suppression of memories of aversive content in response to reminders, the current study pointedly targets the causal status of sleep loss in compromising inhibitory control over thought.

Our findings may help to explain the relationship between sleep disturbance and vulnerability to psychiatric conditions associated with intrusive thoughts. Up to ninety percent of patients with PTSD (Maher et al., 2006) and MDD (Riemann et al., 2001) report recurrent sleep disturbances, which are major risk factors for disease onset (Maher et al., 2006; Mendlewicz, 2009). Exactly *how* disturbed sleep contributes to these disorders remains elusive, however. Our data raises the possibility that poor intrusion control may help bridge the gap between disturbed sleep and psychiatric symptoms: insufficient sleep might increase memory intrusions, whilst also nullifying the benefits of retrieval suppression for regulating affect. The onset of intrusive thoughts and affective dysfunction following bouts of poor sleep could create a vicious cycle, whereby upsetting intrusions and emotional distress exacerbate sleep problems (Talamini, Bringmann, de Boer, & Hofman, 2013), inhibiting the sleep needed to support recovery. We employed a sleep deprivation paradigm to model the sleep

disruption characteristic of psychiatric disorders. However, it should be noted that a night of total sleep deprivation is qualitatively different to the chronic sleep disturbance observed in PTSD and MDD. Future studies should utilize a prolonged partial sleep deprivation design to ascertain whether this form of sleep disruption has similar consequences for intrusion control and associated affect suppression.

Evidence suggests that practice improves intrusion control in both real-life situations and via strategic intervention. For example, undergraduate students who report having experienced moderate childhood adversity exhibit greater memory control than do those who report experiencing little to no adversity (Hulbert & Anderson, 2018). This finding suggests that prior experience in suppressing upsetting memories facilitates intrusion control even for new, unrelated memories, leading to greater resilience to adversities later in life. Moreover, providing MDD patients a cognitive strategy to aid their intrusion control prior to performing a retrieval suppression task significantly facilitates memory inhibition (Joormann, LeMoult, Hertel, & Gotlib, 2009). These observations highlight that understanding how intrusions are controlled might lead to strategies for preventing affective disorders from developing in those who are at-risk due to sleep problems, particularly given that controlling intrusive thoughts may benefit affect regulation. Research showing that non-depressed individuals with insomnia have a twofold risk of developing MDD (Baglioni et al., 2011) highlight the critical importance of developing such preventative strategies, especially given that MDD is a leading cause of disease burden in developed countries (Whiteford et al., 2013).

We found that sleep deprivation strikingly impairs the ability to prevent unwanted memories from entering conscious thought. Moreover, we found that whereas suppressing negative memories renders them less subjectively and

psychophysiologically aversive, poor intrusion control following sleep deprivation negates this affective benefit. Together with an increasingly specific understanding of the neural machinery of retrieval suppression (Anderson, Bunce, & Barbas, 2016; Anderson & Hanslmayr, 2014; Guo, Schmitz, Mur, Ferreira, & Anderson, 2017; Schmitz, Correia, Ferreira, Prescot, & Anderson, 2017), these findings point to an important neurocognitive mechanism linking sleep problems to the pathogenesis and maintenance of psychiatric conditions characterized by intrusive symptoms. Developing interventions that improve retrieval suppression in poor sleepers may be a promising avenue for averting the potentially pathogenic consequences of disordered control over distracting thoughts.

Author contributions

M.O.H and S.A.C designed the study; M.O.H and J.E.A. performed the experiments; M.O.H, S.S, M.C.A and S.A.C analysed the data; M.O.H wrote the manuscript; M.O.H, J.E.A, S.S, M.C.A and S.A.C revised the manuscript.

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Declaration of interests

The authors declare no competing interests.

Data availability

All study data have been deposited on the Open Science Framework, and will be made publicly available following publication of the final article.

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Table 1. Sleep stage data

N1 (min; %)	8.02 ± 1.07	1.96 ± 0.26
N2 (min; %)	218.14 ± 6.76	53.56 ± 1.25
SWS (min; %)	107.34 ± 3.85	26.47 ± 0.93
REM (min; %)	73.00 ± 3.87	18.00 ± 0.95
TST (min)	406.50 ± 6.78	-
Sleep efficiency (%)	97.26 ± 0.49	-

Data presented as mean ± SEM. Sleep efficiency refers to the proportion of time spent asleep between sleep onset and final awakening. Abbreviations: N1, N2, stages of non-REM sleep; SWS, slow-wave sleep; REM, rapid eye movement sleep; TST, total sleep time.

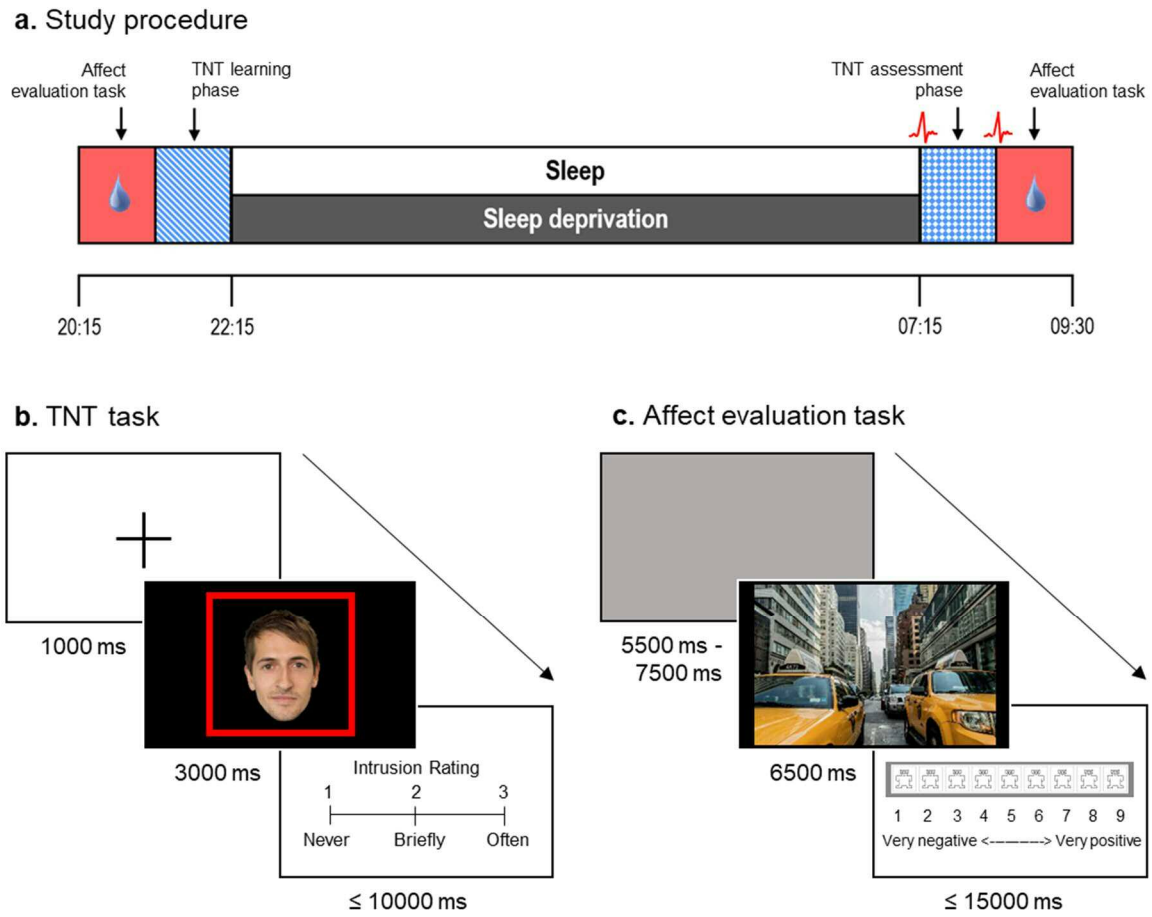

Figure 1. Study procedures and tasks. (a) All participants completed session one in the evening, which included the first of two affect evaluation tasks and the learning phase of the Think/No-Think (TNT) task. Participants in the sleep group slept in the sleep laboratory for ~8 hours. Participants in the sleep deprivation group remained awake across the night. All participants then completed session two, which included the assessment phase of the TNT task and the second affect evaluation task. We collected skin conductance responses (SCRs) to scene presentations throughout the affect evaluation tasks. We recorded resting heart rate variability (HRV) before and after the TNT assessment phase. (b) In the critical TNT assessment phase, we presented participants with faces in red or green frames. For red framed faces ('No-Think' trials), we instructed participants to avoid thinking about the associated scene without generating diversionary thoughts; for green framed faces ('Think' trials), we instructed them to visualise the associated scene. After each trial, participants reported the extent to which they thought about the paired scene (never, briefly or often). We considered reports of "briefly" or "often" as intrusions. (c) In the affect evaluation task, participants viewed negative and neutral scenes and we asked them to provide an emotional rating for each image on a scale from 1 (very negative) to 9 (very positive).

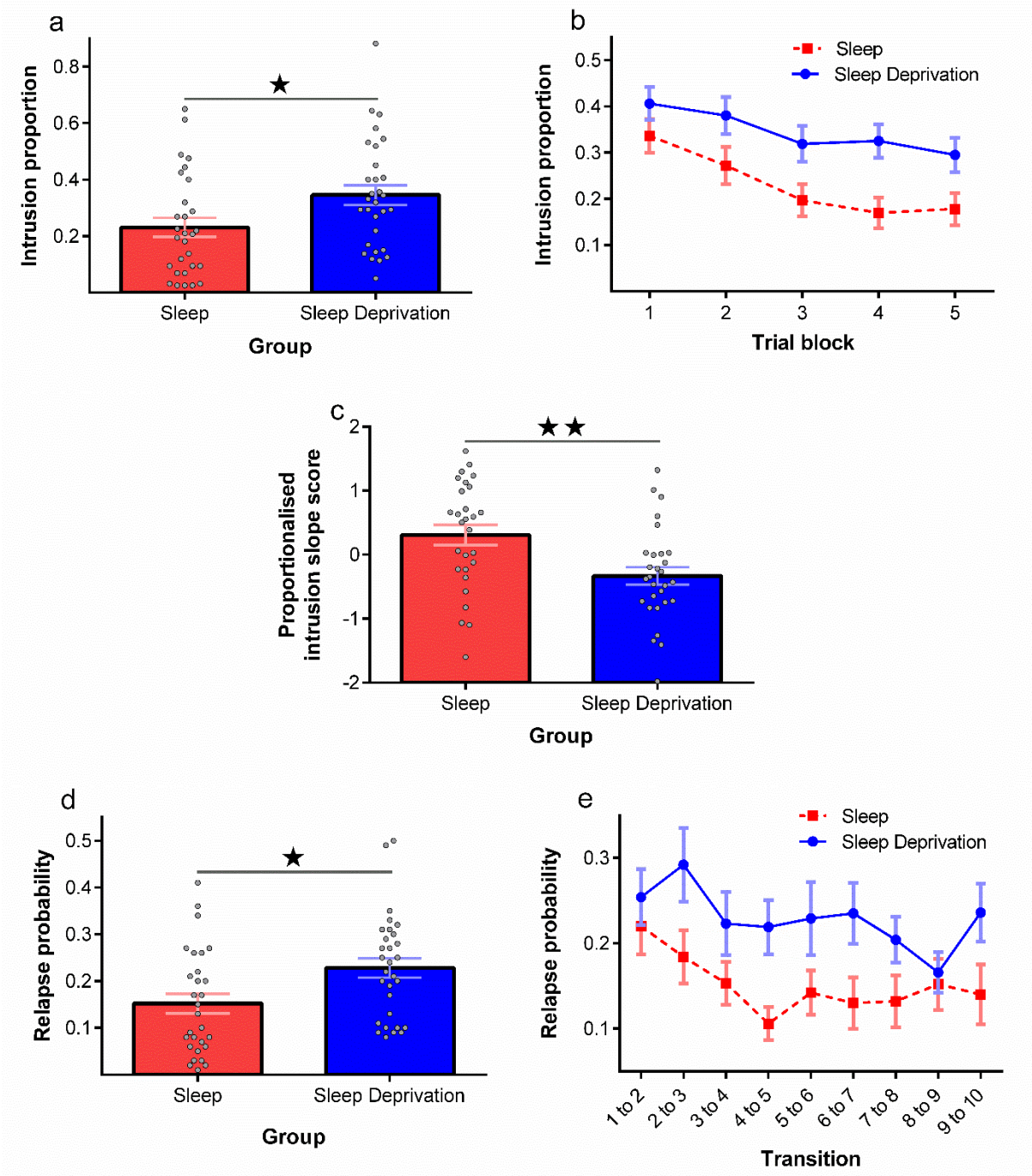

Figure 2. Sleep deprivation impairs the suppression of intrusive thoughts. (a) Intrusion proportion (i.e. proportion of 'No-Think' trials for which participants reported awareness of the associated scene), averaged across TNT blocks. (b) Intrusion proportion in each TNT block. (c) Proportionalised intrusion slope scores (i.e. the rate at which intrusions declined across the five TNT blocks for 'No-Think' items, divided by the number of intrusions in the first TNT block). Greater slope scores reflect more effective downregulation of intrusions across trial blocks. (d) Relapse probability (i.e. proportion of 'No-Think' trials where successful control on repetition N was followed by failed control on repetition N+1), averaged across trial transitions. (e) Relapse probability for each trial transition. Grey dots represent individual participants; data are shown as mean ± SEM; ★ represents $p < .05$; ★★ represents $p < .01$.

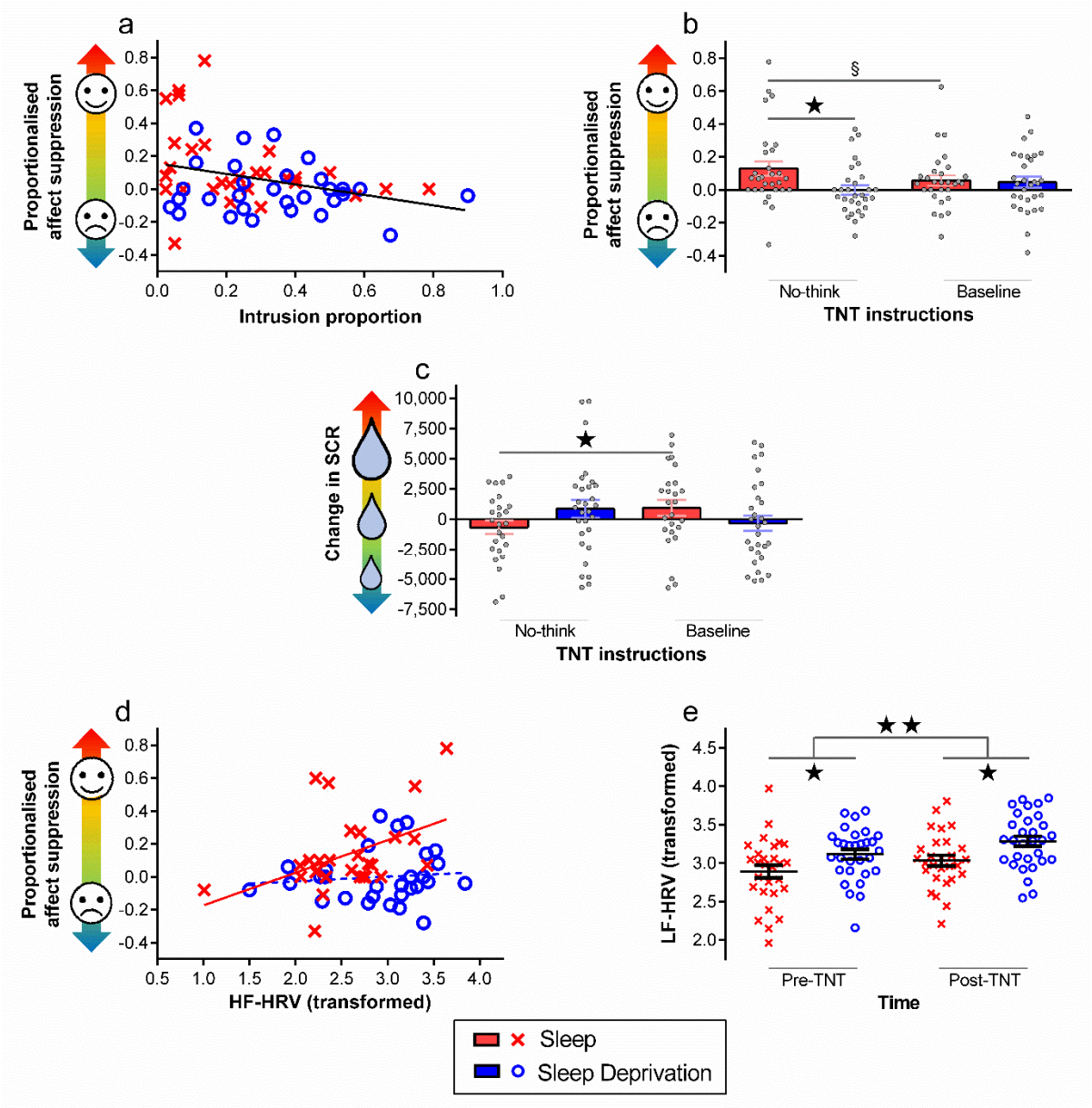

Figure 3. Sleep deprivation influences affect suppression and heart rate variability. (a) Correlation between intrusion proportion and affect suppression scores for negative 'No-Think' scenes. (b) Affect suppression scores for negative scenes. (c) Change in skin conductance response (dSCR) towards negative scenes. (d) Correlations between log-transformed resting HF-HRV (pre-TNT) and affect suppression for negative 'NoThink' scenes. (e) Mean log-transformed LF-HRV in each group. Stars represent main effects of group (sleep, sleep deprivation) and time (pre-TNT, post-TNT). Data points represent individual participants; data are shown as mean ± SEM; § represents $p < .10$; ★ represents $p < .05$; ★★ represents $p < .001$.