



Napping promotes inter-session habituation to emotional stimuli

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ABSTRACT

The effects of a daytime nap on inter-session habituation to aversive visual stimuli were investigated. Healthy young adult volunteers viewed repeated presentations of highly negative and emotionally neutral (but equally arousing) International Affective Picture System (IAPS) photographs during two afternoon sessions separated by 2.5 h. Half of the photographs were shown at both sessions (Repeated Sets) and half differed between sessions (Novel Sets). For each stimulus presentation, evoked skin conductance response (SCR), heart-rate deceleration (HRD) and corrugator supercilii EMG response (EMG), were computed and range corrected using respective maximum session-1 responses. Following each presentation, subjects rated each photograph on dimensions of pleasantness and arousability. During the inter-session interval, Nap subjects had a 120-min polysomnographically monitored sleep opportunity, whereas Wake subjects watched a non-stimulating video. Nap and Wake subjects did not differ in their subjective ratings of photographs. However, for Repeated-Set photographs, Nap subjects demonstrated greater inter-session habituation in SCR and EMG but a trend toward lesser inter-session habituation in HRD. These group differences were absent for Novel-Set photographs. Group differences across all measures were greater for negative stimuli. Occurrence of SWS during the nap was associated with greater inter-session habituation of EMG whereas occurrence of REM was associated with lesser inter-session habituation of SCR to negative stimuli. Sleep may therefore promote emotional adjustment at the level of somatic responses. Physiological but not subjective inter-session habituation to aversive images was enhanced by a daytime nap.

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1. Introduction

It is often hypothesized that sleep plays an emotional regulatory role in healthy humans (Walker, 2009; Walker & van der

Helm, 2009) and that disruption of sleep negatively impacts mood (Dinges et al., 1997; Haack & Mullington, 2005; Zohar, Tzischinsky, Epstein, & Lavie, 2005). Moreover, it is widely suggested that sleep disruption and waking emotion mutually interact in the etiology and perpetuation of mood and anxiety disorders (Franzen & Buysse, 2008; Germain, Buysse, & Nofzinger, 2008; Kim & Dimsdale, 2007; Mellman, 2006; Nielsen & Levin, 2007; Peterson & Benca, 2006). In humans, sleep may play a role in moderating emotional aspects of higher cognitive functions such as resolution of intrapersonal conflict (Cartwright, Lutten, Young, Mercer, & Bears, 1998), affectively guided decision making (Killgore, Balkin, & Wesensten, 2006a), moral reasoning (Killgore et al., 2006) and recognition of facial expressions of emotion (van der Helm, Gujar, & Walker, 2010). Similarly, sleep may modulate the impact of emotion on human declarative memory (Payne, Stickgold, Swanberg, & Kensinger, 2008; Walker, 2009). However, sleep may also moderate emotions by phylogenetically primitive learning mechanisms such as habituation and extinction (Pace-Schott et al., 2009). Moreover, sleep may be essential for the homeostatic equilibration of stress, reward, autonomic and neuroendocrine systems that is essential

Abbreviations: ANOVA, analysis of variance; BPM, beats-per-minute; CS, conditioned stimulus; DCS, D-Cycloserine; DSM-IV-TR, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision; EEG, electroencephalogram; EMG, electromyogram; ESS, Epworth Sleepiness Scale; HRD, heart-rate deceleration; Hz, Hertz (cycles per second); IAPS, International Affective Picture System; ISI, inter-stimulus interval; KOhm, kilo Ohm; μ S, microSiemens; μ V, microVolts; NEO-PI-R, Revised NEO Personality Inventory; NMDA, N-methyl D-aspartate; NREM, non-rapid eye movement sleep; PANAS, Positive and Negative Affect Schedule; PSQI, Pittsburgh Sleep Quality Index; PSG, polysomnography; REM, rapid eye movement sleep; SAM, Self-Assessment Manikin; SCL, skin conductance level; SCR, skin conductance response; SD, standard deviation; SEM, standard error of the mean; SSS, Stanford Sleepiness Scale; STAI, Spielberger State-Trait Anxiety Index; SWS, slow wave sleep (NREM stages 3 and 4); US, unconditioned stimulus.

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to maintaining a euthymic waking mood (McEwen, 2006; Mullington, Haack, Toth, Serrador, & Meier-Ewert, 2009).

Habituation is an evolutionarily ancient mechanism of neuronal plasticity and non-associative learning whereby behavioral and physiological responses during initial exposure to a stimulus diminish with repeated presentations of that stimulus (Grissom & Bhatnagar, 2009; Leussis & Bolivar, 2006; Thompson & Spencer, 1966). Habituation studies in rodents differentiate between intra-session habituation, the decline in a behavior during an exposure session, and inter-session habituation, maintained low or further reduced responses from one session to the next (Leussis & Bolivar, 2006). Both processes are believed to reflect memory phenomena that are to some extent dissociable by their responses to experimental lesions (e.g., hippocampal) and pharmacological manipulations as well as by their genetic determinants (Bolivar, 2009; Leussis & Bolivar, 2006).

Habituation occurs with both biologically salient and neutral stimuli (McSweeney & Murphy, 2009). In humans as well as animals, habituation occurs to aversive stimuli on both behavioral and neuroendocrine levels (Grissom & Bhatnagar, 2009) and it has been argued that such “affective habituation” functions to promote behavioral flexibility by preventing maladaptive panic-like states (Dijksterhuis & Smith, 2002). In addition, subjective and behavioral habituation to emotional stimuli is accompanied by decreasing activity in areas involved in processing and regulating emotion, especially the amygdala and prefrontal cortex (Milad et al., 2007; Mutschler et al., 2010; Wilson & Rolls, 1993; Wright et al., 2001). Habituation, like extinction, may be a mechanism contributing to the efficacy of exposure therapies (Averill, Malmstrom, Koriat, & Lazarus, 1972; Craske et al., 2008; Foa & Kozak, 1986; Jaycox, Foa, & Morral, 1998). Uniquely human cognitive mechanisms of emotion regulation, such as re-attribution of the meaning of events, may recruit the neural circuitry of these more basic mechanisms (Delgado, Nearing, Ledoux, & Phelps, 2008; Schiller & Delgado, 2010).

Sleep contributes to the consolidation of emotional memory (Hu, Stylos-Allan, & Walker, 2006; Nishida, Pearsall, Buckner, & Walker, 2009; Wagner, Degirmenci, Drosopoulos, Perras, & Born, 2005; Wagner, Gais, & Born, 2001; Wagner, Hallschmid, Rasch, & Born, 2006; Walker, 2009). Effects of sleep on emotional memory extend to adaptive modifications of relationships between different memory traces. For example, sleep enhances a trade-off in visual memory insofar as memory for emotional (but not neutral) objects is preferentially remembered (Payne et al., 2008) and sleep promotes generalization of extinction of conditioned fear (Pace-Schott et al., 2009). REM sleep may be especially important for the consolidation and modulation of emotional memory (Levin & Nielsen, 2007; Nishida et al., 2009; Wagner et al., 2001).

Two prior studies investigated sleep effects on emotional habituation using the International Affective Picture System (IAPS) (Lang, Bradley, & Cuthbert, 2008) along with self-ratings of valence (pleasantness vs. aversiveness) and arousability using the Self-Assessment Manikin (SAM) (Bradley & Lang, 1994). Wagner, Fischer, and Born (2002) examined SAM valence ratings of repeated (pre-sleep exposed) vs. novel (previously unseen) negative IAPS stimuli and compared these repeated vs. novel ratings differences between groups that had either slept or remained awake across an inter-session interval. The group that slept was further divided into sub-groups with early night (with SWS-rich sleep) and late night (with REM-rich sleep) sleep opportunities, each with its own wake control over the same time period. After an early-night period, ratings of repeated stimuli were less negative than those of novel images following sleep but more negative following wake. In contrast, after a late-night period, repeated stimuli were more negative following both sleep and wake but this difference was greater following sleep.

Similarly, Lara-Carrasco, Nielsen, Solomonova, Levrier, and Popova (2009) compared emotional reactivity to IAPS photographs seen for the second time following overnight sleep during which subjects were either minimally or substantially deprived of REM sleep. As in Wagner et al.’s (2002) study, greater reactivity was observed following the high- vs. low-REM condition. Neither study, however, employed the physiological measures that are known to capture covert or non-conscious expression of emotional responses (Bradley & Lang, 2007).

The current study examined the impact of an afternoon nap on habituation to emotionally aversive IAPS stimuli using physiological as well as subjective measures of emotion. A greater degree of intra-session habituation in comparison to studies cited above was established via repeated presentations of the same stimuli at the first session. It was hypothesized that napping vs. waking intervening between repeated viewings of the same stimuli would lead to enhanced inter-session habituation of emotional responses. It was further hypothesized that sleep facilitation of inter-session habituation would be (1) greater for negatively valenced stimuli and (2), because habituation is a form of memory, would be specific to previously viewed stimuli rather than being generalized diminished reactivity to all stimuli.

2. Methods

2.1. Participants

Candidate participants were recruited via internet and screened by telephone. Exclusion criteria were self-report of current neurological or medical conditions, use of sleep-altering drugs, night shift work or inability to keep a regular sleep schedule, excessive caffeine (>5 cups/day) or alcohol (>12 drinks/week) consumption and average sleep per night <6 or >10 h, as well as any current or history of neurological disorder, significant head trauma, DSM-IV-TR Axis 1 disorder (including alcohol or drug abuse), sleep disorder or use of psychiatric medication.

Forty-six paid volunteers (31 female), mean age = 20.78 (*SD* 2.25, range 18–27) were pseudorandomly assigned to Nap or Wake groups. Two Nap-group subjects’ data were not analyzed because they failed to sleep more than 10 min. One Wake-group subject’s data were not analyzed due to curtailed sleep the previous night. The final sample contained 22 Nap (15 females) and 21 Wake (15 females) subjects. Because extinction of fear conditioning varies with phase of the menstrual cycle (Milad et al., 2006a), female participants were scheduled for study on a date they estimated would fall during the early follicular phase (days 1–7) of their cycle (a scheduling goal that was successful in 17 of 30 females). All procedures were approved by the Beth Israel Deaconess Medical Center Committee on Clinical Investigation and all subjects provided written informed consent.

Sample size was estimated from subjective data in Wagner et al. (2002) in which early-night sleep vs. wake difference was a trend with an effect size of $d = 0.81$ that would require 25 subjects to detect a difference at $\alpha = 0.05$ with 80% power. The sleep vs. wake difference across late night was significant, with $d = 0.84$ and requiring 18 subjects per group. Our sample size fell between these figures.

2.2. Pre-study week

During the week before the experiment, subjects were asked to maintain a regular sleep schedule with ≥ 7 h in bed each night and bedtime no later than 2:00 a.m. During this week, subjects were asked to refrain entirely from alcohol, recreational drugs and daytime napping and they filled out a nightly sleep diary (Pace-Schott

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