



Associations of nocturnal sleep with experimental pain and pain catastrophizing in healthy volunteers

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ABSTRACT

Strong alterations of night sleep (e.g., sleep deprivation, insomnia) have appeared to affect pain in inducing hyperalgesic changes. However, it has remained unclear whether everyday variations of night sleep in healthy individuals have any influence on pain processing. Forty healthy subjects were studied by portable polysomnography (PSG) and sleep questionnaire during two non-consecutive nights at home. Experimental pain parameters (pressure pain threshold, temporal summation = TS, conditioned pain modulation = CPM) and situational pain catastrophizing (Situational Catastrophizing Questionnaire = SCQ) were always assessed the evening before and the morning after sleep recording in a pain laboratory. Linear regression analyses were computed to test the prediction of overnight changes in pain by different sleep parameters. Significant prediction of changes in pain parameters by sleep parameters was limited (2 out of 12 analyses), indicating that everyday variations in sleep under non-pathological and low stress conditions are only weakly associated with pain.

1. Introduction

It is to date widely accepted that sleep alterations affect pain. Evidence for this belief stems mainly from studies in which the effects of sleep deprivation or substantial sleep fragmentation on experimental pain parameters were investigated (Karmann, Kundermann, & Lautenbacher, 2014; Kundermann and Lautenbacher, 2007; Lautenbacher, Kundermann, & Krieg, 2006). Insomnia as a clinical condition with sleep fragmentation as a symptom has appeared to corroborate this impression (Haack et al., 2012). These findings allow for the assumption that poor night sleep enhances pain sensitivity but not for the determination of the mechanisms of action.

Sleep is a highly complex state with multiple processes and different stages; thus, it appears unlikely that all sleep-indicative variables are equally linked with pain. For identification of the critical variables, more specific manipulations were used instead of total sleep deprivation. Lentz, Landis, Rothermel, & Shaver (1999) selectively disrupted slow wave sleep (SWS) with little effect on the total sleep duration and produced a substantial decrease in pain threshold. Onen, Alloui, Gross, Eschallier, & Dubray (2001) also interrupted SWS, again with the result of a decrease in pain threshold. These findings raised hope that a specific delta-wave related mechanism might be identified, which was,

however, frustrated by a study by Older et al. (1998), who could not change pain threshold with three days of delta-wave interruption. The findings by Engström et al. (2013, 2014); of inconsistent correlations between SWS duration and pain threshold also suggest that there might not be an easy answer claiming variations in SWS as major mediator of changes in pain processing.

Which other candidates are available and have been tested? Rapid eye movement (REM) sleep deprivation led to a decrease in pain threshold (Onen et al., 2001) in one study; however, in another study no changes in laser evoked potentials and ratings were observed (Azevedo et al., 2011). Roehrs, Hyde, Blaisdell, Greenwald, & Roth (2006) may have found an explanation for this inconsistency by demonstrating a rapid attenuation of the effect of REM sleep deprivation on pain after only one night. Landis, Lentz, Rothermel, Buchwald, & Shaver (2004) found a correlation of pain threshold with sleep spindle activity, with less activity being associated with lower thresholds, which is a finding awaiting further replication.

The impression that there are still open questions as regards the specific mechanisms implicated in sleep effects on pain was also supported by studies in which the conditioned pain modulation (CPM) paradigm for the study of pain inhibition was used. It seems very likely that CPM becomes deficient after sleep deprivation. However, it is still

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unclear which sleep stages are critically responsible for the naturally occurring nocturnal restoration of pain inhibition (Edwards et al., 2009; Smith, Edwards, McCann, & Haythornthwaite, 2007).

In summary, there is considerable evidence that substantial sleep interruption, either induced experimentally by total sleep deprivation or occurring as a consequence of insomnia, definitely leads to hyperalgesia. However, when less powerful interventions were used or non-pathological covariations were studied, results were much less consistent. One might assume that there is only a loose covariation between sleep and pain, which requires major changes of sleep to affect pain. Such an association might be functionally adaptive to avoid that smallest sleep disturbances can already dysregulate the pain system. Under this perspective, everyday variations of nocturnal sleep may have no impact on pain; it may be that the two functions remain disconnected as long as sleep varies within normal and non-pathological limits.

To test this assumption, we studied healthy individuals (sleep and pain disorders were explicitly excluded) as regards their pain psychophysics (pain threshold, temporal summation, CPM) before and after having had night sleep at home, i.e., in a familiar and non-stressful situation. We added pain catastrophizing as subjective state variable known to influence both sleep quality and pain processing (Byers, Lichstein, & Thorn, 2016; Campbell et al., 2015). Night sleep was recorded via portable polysomnography and sleep quality was assessed via questionnaires. We hypothesized that sleep parameters would not substantially relate to pain parameters in our healthy sample under these non-pathological and low stress conditions.

2. Materials and methods

2.1. Subjects

The 40 participants (female: $N = 20$) between the ages of 19 and 59 years (mean age: 38.8 years; $SD = 13.5$) were recruited via university news posted in the local newspapers. Exclusion criteria were acute and chronic pain, psychological disorders or medical diseases, including sleep disorders. Participants taking psychotropic drugs or analgesics were also excluded from participation. A trained psychologist verified the exclusion criteria in a standardized clinical interview. All participants provided written informed consent before testing and received monetary compensation for their participation. The study protocol was approved by the ethics committee of the University of Bamberg.

2.2. Procedures

Nocturnal sleep quality was assessed in two non-consecutive nights (1–13 days interval) at the participant's home via objective (portable polysomnography (PSG) recordings) and subjective (questionnaire) measures. In addition, four procedurally equal test sessions were run in the pain laboratory at the University of Bamberg in order to assess several parameters of pain processing. These sessions took always place at 6 p.m. before and at 8 a.m. after the test nights. The test protocol for these sessions included the assessment of several physiological, behavioral and psychological measures. At the start and end of each session, subjects provided saliva samples for determination of cortisol levels. In a second part, the participants completed a dot-probe task and an eye tracking paradigm, both presenting emotional facial stimuli (in a randomized order). The last part of each session – which will be the subject of the current publication – was run to measure pain processing. First, pressure pain thresholds were assessed. The assessment of temporal summation of pressure pain and conditioned pain modulation (CPM) followed. Given this protocol, the impact of stimuli slowly increased, starting with pain cues (pictorial facial expression of pain), being followed by slightly painful stimuli (pain threshold) and ending with moderately painful stimuli (temporal summation, CPM), in order to minimize order effects. After pain stimulation, participants were asked

to fill out the Situational Catastrophizing Questionnaire (SCQ; 17), whereupon the last cortisol sampling followed.

2.3. Assessment of pain-related measures

2.3.1. Apparatus

Pressure stimuli were administered with a computer-controlled pressure algometer (Noxitest Biomedical, Aalborg, Denmark; see also Nie, Arendt-Nielsen, Andersen, & Graven-Nielsen, 2005 for a detailed description). A rounded aluminum foot plate with a padded probe area of 1 cm^2 was fixed to the tip of a piston, which was moved by an electric motor. The pressure stimulation was controlled by a built-in force transducer. Pressure stimuli were applied to the fingertip of the middle and index finger of the left hand. The pressure algometer was mounted on a table in front of the participants in such a way that the participant could place her/his fingertips comfortably below the probe.

A heat stimulus was administered as conditioning stimulus in the CPM paradigm by using a circulating water bath (Witeg GmbH, WiseCircu WCB-22, Wertheim, Germany), containing $46\text{ }^\circ\text{C}$ hot water. The subject immersed her/his hand up to 2 cm above the wrist in this water bath. The water temperature was controlled by a thermostat, and the water was stirred with a force and suction pump to avoid layers of lower temperature around the hand. The heat stimulus was always applied to the right hand.

2.3.2. Assessment of pressure pain thresholds

Pressure pain thresholds were assessed using the method of limits. The piston was lowered until the probe touched the skin of the fingertip. Then, the pressure increased at a rate of 50 kPa/s until the subjects felt the stimulus to be slightly painful and responded by pressing a stop button. Each time they pressed the button, the probe lifted and returned the pressure to zero. After two practice trials, five trials were presented at each finger (middle and index fingers) with an inter-stimulus interval (ISI) of $> 8\text{ s}$. These 5 trials were averaged to get the estimate of pressure pain threshold for each finger. For the correlation analysis with sleep parameters, the average pain threshold computed over both fingers was used.

2.3.3. Assessment of temporal summation

Temporal summation was tested by comparing the sensations evoked by single pulses of pressure stimulation to sensations evoked by a series of five pulses (only the last pulse was rated), which were applied with a repetition frequency of 0.5 Hz . The series of five pulses was always delivered 60 s after the single pulse. Stimulus intensity was tailored to the individual pain threshold (50% above threshold) and increased with a rate of rise of 75% of the target intensity per second. The stimuli had a saw-tooth shape with stimulus duration at maximum of only 0.1 s . The three runs of single pulses and pulse series were separated by intervals of 60 s. In each run, either the index or the middle finger were stimulated, alternating always with the other finger in the next run. This sequence was counterbalanced over the participants, with half of the participants starting with the index finger. The three runs were presented once in each of the two experimental conditions (baseline, CPM).

2.3.4. Assessment of conditioned pain modulation (CPM)

The CPM effect was tested using water of painful heat ($46\text{ }^\circ\text{C}$) as conditioning stimulus whereas the pressure stimuli (single and series) served as test stimuli. The perceived intensity of the latter was supposed to be modulated by the former stimulus. For assessing this CPM effect, the ratings evoked by the pressure stimuli during concurrent presentation of the conditioning stimulus (CPM condition) were compared to ratings without conditioning stimulation (baseline condition). The temperature of $46\text{ }^\circ\text{C}$ was selected as the painful intensity of the conditioning stimulus based on the results of previous studies (Lautenbacher, Roscher, & Strian, 2002; Willer, Roby, & Le Bars, 1984).

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