J. Sleep Res. (2007) **16,** 285–296

Adverse childhood experiences associated with sleep in primary insomnia

KLAUS BADER[1], VALÉRIE SCHÄFER[1], MAYA SCHENKEL[1], LUKAS NISSEN[1] and JÜRG SCHWANDER[2]

[1]Psychiatric University Clinics, Basel and [2]Clinic for Sleep Medicine, Lucerne, Switzerland

Accepted in revised form 22 June 2007; received 15 January 2007

SUMMARY The objectives were to explore the association between self-reported adverse childhood experiences (ACE) and sleep in adults suffering from primary insomnia and to examine the impact of presleep stress on this relationship. Fifty-nine patients with primary insomnia, aged 21–55 years, were administered the Childhood Trauma Questionnaire (CTQ) and then divided into two groups according to the achieved scores: with moderate/severe or low/no reports of ACE. The participants spent three consecutive nights in the sleep laboratory in order to record polysomnographic and actigraphic sleep parameters. A stress induction technique was administered by activating negative autobiographical memories immediately before sleep in the second or third night. Results show that 46% of the insomniac patients reported moderate to severe ACE. This group exhibited a significantly greater number of awakenings and more movement arousals compared to patients with low or no reports of ACE. Actigraphic data also indicated more disturbed sleep and increased nocturnal activity for the high-ACE group. On the other hand, no specific group differences were found with regard to stress condition. The results support the assumption that it is possible to identify a subgroup among patients with primary insomnia who has experienced severe maltreatment in childhood and adolescence. This subgroup appears to differ in several sleep parameters, indicating a more disturbed sleep compared to primary insomniacs with low or no reports of ACE. With regard to sleep-disturbing nightly patterns of arousal, parallels between individuals with high ACE and trauma victims as well as post-traumatic stress disorder-patients suggest themselves.

KEYWORDS actigraphy, adverse childhood experiences, polysomnography, primary insomnia, sleep, trauma

INTRODUCTION

Adverse experiences during childhood and adolescence such as physical, emotional and sexual abuse seem to be an important risk factor for mental and physical health problems in later adulthood (Felitti *et al*., 1998; Goodwin and Stein, 2004; Spertus *et al*., 2003). Adults with a history of adverse childhood experiences (ACE) show higher rates of depression, anxiety disorders, alcohol abuse, ischemic heart disease and certain psychosomatic symptoms like chronic gastrointestinal distress and recurrent headaches(Chapman *et al*., 2004; Dong *et al*., 2004; Dube *et al*., 2002).

Sexually and physically abused children frequently show symptoms of post-traumatic stress such as hypervigilance or sleep disturbances (Ackerman *et al*., 1998) which can persist for many years after the original traumatic event and may never fully remit (Zlotnick *et al*., 1999). Changes in the nervous and endocrine system in the course of repeated trauma or long-lasting experiences of stress appear to render adults victimized in childhood more vulnerable to stress-related disorders and more reactive when confronted with stressors. According to Barlow (2002), hypervigilance (i.e. hyperarousal) in traumatized individuals may be interpreted to reflect the promptness and preparation to deal with potentially negative events.

Correspondence: Dr Klaus Bader, Psychiatric University Clinics UPK, Wilhelm Klein-Str. 27, CH-4025 Basel, Switzerland. Tel.: +41 61 325 51 19; fax: +41 61 325 56 71; e-mail: klaus.bader@upkbs.ch

Hyperarousal is assumed to play a central role in the development and maintenance of sleep disturbances (Morin, 1993; Perlis *et al*., 1997). Stressful negative life events seem to be the most common precipitating factors of insomnia (Bastien *et al*., 2004). The vulnerability to react to these stressors with hyperarousal and to maintain this arousal at a certain level may be related to a variety of biological and/or psychological factors (Espie, 2002). A long history of neglect and abuse in childhood or other ACE may lead to an adaptive process of the organism and predispose a patient to hyperarousal reactions when confronted with acute stressors in adulthood. In this sense, early trauma history can be understood as a risk factor for the development of insomnia in the course of a lifetime.

Sleep disruption and nightmares are presumed to be associated with childhood physical and sexual abuse. In a study by Sadeh *et al*. (1995) physically and sexually abused children from a psychiatric inpatient unit were examined using actigraphy. Abuse history was found to be significantly related to sleep measures. Physically abused children had decreased sleep efficiency and spent proportionally less time in quiet-motionless sleep compared with non-abused inpatients or sexually abused children. Glod *et al*. (1997) compared actigraphic sleep parameters of abused, normal and depressed children. Abused children, whether or not they fulfilled diagnostic criteria for post-traumatic stress disorder (PTSD), displayed higher activity levels during the night and more disruption in sleep initiation and continuity than both the non-abused normal volunteers and the depressed children. In an own study (Bader *et al*., 2007) 39 adults suffering from primary insomnia were monitored for seven consecutive nights at home with wrist actigraphs to evaluate objective sleep-related activity. ACE proved to be important predictors of actigraphically assessed sleep-onset latency, sleep efficiency, number of body movements and moving time. Furthermore, in various samples of healthy persons, we found significant correlative relationships between subjective sleep quality ratings and stressful experiences in childhood (Bader *et al*., 1999). We assume that adults with an early history of severe negative life experiences (e.g. childhood maltreatment) are more vulnerable to react to acute stressors with sleep disturbances, which, under certain conditions, may lead to manifest insomnia.

The goal of the present study was to explore the relationship between self-reported ACE and sleep in a sample of non-treated adults suffering from primary insomnia. We hypothesized that such an association does exist and that it should become particularly apparent following a presleep stress induction, i.e. the recall of negative autobiographic memories.

METHODS

Participants

Thirty-nine persons participating in this study also were part of another study in this field as described in Bader *et al*. (2007).

Participants were recruited via newspaper advertisements. Inclusion criteria were (1) age 20–55 years; (2) reported insomnia, defined as difficulty initiating or maintaining sleep or as non-restorative sleep for at least 1 month; and (3) reports that the insomnia was causing significant distress or clearly affecting daytime functioning. Exclusion criteria were (1) presence of other sleep disorders such as narcolepsy, sleep apnea, restless legs syndrome, circadian sleep disorders or parasomnias; (2) evidence that the insomnia was related to a somatic disorder; (3) presence of major depression, anxiety disorder, alcohol or substance abuse, or any other psychopathology; (4) currently in psychotherapy; and (5) regular use of sleep medication or use of other sleep-affecting medication (e.g. sedatives, anxiolytics, antidepressants, neuroleptics, beta-blockers) for the last 4 weeks prior to study entry. Persons with irregular use of sleep medication (i.e. less than twice weekly) were permitted to participate in the study after a drug washout period of at least 7 days. These criteria are consistent with those of the DSM-IV [American Psychiatric Association (APA), 1994] for primary insomnia.

Ninety persons attended the screening interview. Thereafter, 13 withdrew and 12 were excluded because they did not meet full insomnia criteria (1 person), met criteria for an additional axis-I disorder (2 persons), or were suspected of suffering from sleep-related breathing disorder or restless legs syndrome (9 persons). Of the 65 persons who then attended the sleep laboratory investigations, 6 were excluded from the sample because they either suffered from a sleep-related breathing disorder (3 persons), suffered from bruxism (1 person), or cancelled the study after the second night (2 persons).

The remaining sample included 59 participants (45 women and 14 men) with a mean age of 43.6 years (SD = 8.8; range: 21–55). The sample comprised 50 employed workers, 4 unemployed participants, 3 students and 2 housewives. The average insomnia duration was 10.3 years (SD = 8.3). Four participants needed a drug-washout period: two because of the use of antidepressants and two because of an irregular use of sleep medication. They stopped taking their medication 4 and 1 weeks, respectively, before study entry.

Materials

Polysomnography

The basic polysomnography (PSG) montage included eight electroencephalograms (EEGs) placed according to the International 10/20 System (F3-A2, F4-A1, C3-A2, C4-A1, P3-A2, P4-A1, O1-A2, O2-A1), a right and left electrooculogram, a bipolar submental electromyogram (EMG) and an electrocardiogram. In addition, respiration (nasal and oral airflow, thoracic and abdominal respiratory effort and oxygen saturation) and left and right anterior tibitalis EMG were recorded to rule out the presence of occult sleep disorders (e.g. sleep apnea and/or periodic limb movements in sleep). Participants were also observed via a video monitor. All electrophysiological

signals were recorded by a polygraphic amplifier (Braintronics, Almere, The Netherlands), digitized and transmitted via fiber-optic cables to a personal computer containing Deltamed PSG software (Deltamed, Paris, France). EEG was amplified with a time constant of 0.3 s and a low pass filter at 100 Hz. Data were sampled with a frequency of 512 Hz, digitally filtered and stored with a resolution of 128 Hz.

The PSG recordings were manually scored in 30-s epochs according to the standardized criteria by Rechtschaffen and Kales (1968) by two clinicians specifically trained in PSG scoring. Arousal scoring was based on the definition of the American Sleep Disorders Association (ASDA) (1992). According to the ASDA (1992), an arousal is defined as 'an abrupt shift in EEG frequency, which may include theta, alpha and/or frequencies greater than 16 Hz but no spindles'. Furthermore, 10 s of continuous sleep must precede the arousal and a minimum of 10 s of intervening sleep is necessary to score a second arousal. The arousal must last ≥3 s and must be accompanied by an increase in chin EMG if it occurs during rapid eye movement (REM)-sleep. Arousal indexes were calculated by dividing the number of arousals by the total sleep time. For the purpose of this study, we discriminated three different types of arousals: (1) cortical arousals were defined as spontaneous shifts in EEG frequency to alpha or theta ≥3 s but < 30 s duration, (2) movement arousals were defined as shifts in EEG frequency to alpha or theta ≥3 s but < 30 s duration, associated with a rise in chin EMG amplitude and/or rise in leg EMG activity, and (3) respiratory arousals were defined as shifts in EEG frequency to alpha or theta ≥3 s but < 30 s duration, associated with a respiratory event such as apnea, hypopnea or snoring.

For the purpose of this study, the following sleep variables were derived from PSG scoring: sleep-onset latency (SOL; amount of time elapsed in minutes from 'lights off' to the first occurrence of stage 2); sleep efficiency (SE; percentage of time spent asleep whilst in bed); number of awakenings (NWAK; number of awakenings, excluding the final awakening before the final arising; awakening was defined as an instance of more than 30 s of wakefulness occurring after sleep onset); percentages of stages 1, 2, slow wave sleep (stages 3 and 4 combined) and REM sleep; latency to REM sleep; cortical, movement and respiratory arousals.

Actigraphy

The rest–activity cycle was recorded over three consecutive nights in the sleep laboratory with a small activity monitor (Actiwatch®; Cambridge Neurotechnology Ltd, Cambridge, UK) worn on the non-dominant wrist. This monitor contains a piezo-electric linear accelerometer; activity counts are accumulated at selected time intervals and data are downloaded into a computer. Acceleration signals of movements were collected in 1-min epochs. Participants were given an explanation of the monitor's function and instructed to record bedtime and rise time by pressing the event button on the actigraph. All actigraphic data were checked for potential quality problems by one of our research coordinators. Days with data gaps or spurious values were excluded from the statistical analyses.

Actigraphic data were analyzed by using the algorithm supplied by the Actiwatch Sleep Analysis Software (Actiwatch Sleep Analysis 2002; Cambridge Neurotechnology Ltd). Using the default medium sensitivity that was chosen for this study, an integrated activity count ≥40 within a 1-min epoch designates the epoch as being 'awake'. For automatic determination of sleep start, the algorithm searches for a period of at least 10 min consecutively recorded immobile data, with no more than one epoch movement within this period, following the lights-off time. The start of this defined period is classified as sleep start. For sleep end, the algorithm searches for a 10-min consecutive period of activity around the lights-on time, and then works back to find the last epoch of immobility before the start of such a sequence and classifies that as sleep end.

The following actigraphic sleep measures calculated by the Actiwatch Sleep Analysis Software were used in the present study: SE, percentage of number of body movements (percentage of total number of activity counts during the sleep period), and moving time [percentage of minutes spent moving (i.e. number of minutes where scores of greater than zero were recorded) during the sleep period].

Adverse childhood experiences

For the assessment of traumatic experiences in childhood and adolescence the German version of the 'Childhood Trauma Questionnaire' (Bernstein and Fink, 1998) was used. The CTQ is a 28-item self-administered questionnaire to identify childhood maltreatment. It comprises five subscales (five items each) that assess different types of childhood trauma: physical, sexual, and emotional abuse, as well as physical and emotional neglect. The questionnaire also includes a Minimization/Denial scale to detect individuals who may be under-reporting traumatic events. Respondents are presented with a series of statements about childhood experiences (e.g. 'When I was growing up I was punished with a belt, a board, or some other hard object') and are asked to choose from responses on a 5-point Likert-type scale that ranges from 'never true' to 'very often true'. For each CTQ-subscale a scale total score is calculated by summing the respective items, which ranges from 5 to 25. The higher the score, the more childhood maltreatment is being reported. Validation studies of the CTQ have been conducted in seven different clinical and non-referral samples with more than 2200 respondents (Bernstein and Fink, 1998). These studies have demonstrated that self-reports on the CTQ-scales are highly stable over time and show good convergent and divergent validity with traumatic histories that have been ascertained by other measures. The CTQ-scales are highly sensitive to identifying adolescents with verified histories of abuse and neglect. Guidelines are established for classifying subscale scores depending on the severity of the abuse and/or neglect. These guidelines specify the range of scores that constitute 'none to minimal', 'low to moderate',

'moderate to severe', and 'severe to extreme' for each subscale. According to Bernstein and Fink (1998) these ranges are based on data from a non-clinical sample and are successful in identifying 'cases' of these specific types of abuse and neglect, with therapist interview ratings as criteria.

In an additional explorative questionnaire, further information was obtained concerning particularly negative and particularly positive events in the life of the participants. Participants were asked to recall those three events in their lives which they perceived as the most negative and to describe these events in note form. They were also asked to specify the duration of the event ('How long did the negative event last?'), the symptoms perceived during the time of the event ('By which symptoms did you realize you were experiencing stress at the time?'), as well as the coping strategies used to overcome the stress reaction as well as their effectiveness ('Did you make attempts to cope with the situation? If so, what were they and were they successful?'). In addition, participants were asked if the events were foreseeable ('Was the occurrence of the event foreseeable or unforeseeable by you?') or controllable ('Was it possible for you to control the event in any way?'), and whether they were still affected by the event ('Do you still feel affected by the event?'). Furthermore, participants were asked to recall the three most positive events in their lives and to describe them in written form. These reports were used as basis for presleep activation of autobiographic memories.

Presleep stimulation

The first night served as an adaptation night. The following two nights, participants were instructed about 1 h before going to sleep to recall the three most positive events in their lives one night and the three most negative events the other night. The interviews were standardized and conducted by a clinical research coordinator. The answers the participants had given in the explorative questionnaire described above served as the basis for these interviews. Participants were asked to remember concrete episodes of the events and make detailed statements about certain aspects of the experiences (i.e. event triggers, duration, symptoms, emotions, body sensations and behavior during the experiences), while research coordinators made sure that participants did not digress. The interviews took about 45 min and were tape-recorded with the participants' consent.

We hypothesized that the recall of negative memories would induce stress in the participants and predicted it to affect subsequent sleep. Furthermore, we predicted that in the condition of presleep stress, the impact of ACE on sleep would become more pronounced. The recall of positive memories served as control or relaxation condition.

Effect of presleep stimulation

In order to assess the effect of presleep activation of autobiographic memories, we used salivary cortisol measurements and the 'Kurzskala Stimmung/Aktivierung' (KUSTA; Short Mood/Drive Scale; Binz and Wendt, 1986). The KUSTA is a self-rating scale developed for studying intra-individual changes in mood, alertness, and relaxation at short intervals and consists of three subscales: mood (elevated versus depressed mood), alertness (wakefulness versus sleepiness), and relaxation (calmness versus restlessness). Each subscale score ranges from 1 to 9, with higher scores indicating a more cheerful, carefree mood, more relaxation and calmness and more alertness. Lower scores indicate a more sorrowful, depressed mood, tenseness and low alertness (tiredness and lack of energy). The KUSTA was completed by participants both shortly before and after they received presleep stimulation.

To test the effect on salivary cortisol, two saliva samples were collected before and four samples after presleep activation, using the Salivette device (Bühlmann Laboratories AG, Schönenbuch, Switzerland). The first two samples were used as baseline for the subsequent stress-induced cortisol responses. Thus, they were obtained 1 and 10 min before presleep activation of autobiographic memories. Samples 3–6 (after presleep stimulation) were used to measure cortisol response to presleep stimulation, and were taken 1, 10, 20 and 30 min after presleep stimulation. According to Kirschbaum and Hellhammer (1989), salivary cortisol is a useful measure for assessing changes in free cortisol following acute stress. In the present study, we used the minimum of the two baseline samples and the maximum postmanipulation cortisol value to examine cortisol responses to the manipulations.

Current level of stress

To assess participants' current stress levels, two aspects of stress were considered: (1) daily hassles in the last month, and (2) critical life events in the last 6 months.

The German version of the 'Daily Hassles Scale' (DHS; Bodenmann, 1998) measures stress resulting from confrontation with daily hassles during the last month in the domains 'family responsibility', 'partnership', 'time pressure/work', 'social engagement' and 'external stressors'. The 37 items are rated on a five-point scale, ranging from 'not at all stressful' to 'very stressful'. The total score is averaged over all items, and ranges from 1 to 5, with higher scores indicating a greater level of stress. The German version of the DHS has good internal consistency with $\alpha = 0.90$ (Bodenmann, 1998).

The occurrence of critical life events in the last 6 months was measured by means of the self-report inventory 'Fragebogen zu kritischen Lebensereignissen' (FKL; questionnaire assessing critical life events; Bodenmann, 1998), which consists of 27 events such as unemployment, house moving, death of a close person, handicaps, illness, increased occupational demands, separation, divorce, etc. Each item is rated on a dichotomous scale ('yes'/'no') with regard to the occurrence of the critical life event in the last 6 months. The level of stress resulting from this event is rated on a three-point scale (ranging from 1 = 'slightly stressful' to 3 = 'very stressful'). The total score is averaged over the 27 items, and ranges from 0 to 3, with higher scores indicating a greater occurrence of critical life events.

The FKL has been validated in several studies and has a satisfactory discriminative validity (Bodenmann, 1998; Bodenmann *et al*., 2000). The correlation between the total scores of the DHS and the FKL amounts $r = 0.16$, which indicates independence between the two measures.

Subjective sleep measurement

In order to measure subjective quality of sleep the Pittsburgh Sleep Quality Index (PSQI; Buysse *et al*., 1989) was used. The PSQI is a 19-item, reliable, and valid self-report questionnaire that is used to assess sleep disturbances and related sequelae (use of sleep medication, daytime functioning) over a 1-month period. The PSQI-Global (PSQI-G) score was used for the subjective estimate of sleep quality. The PSQI-G ranges from 0 to 21 with scores of 6 or more indicating poor sleep. Buysse *et al*. (1989) have demonstrated that this cutoff identifies a clinically significant sleep disturbance with 89.6% sensitivity and 86.5% specificity.

Depressivity

Current depressive pathology was measured by the German short version of the 'Center for Epidemiological Studies Depression Scale' (Radloff, 1977), the 'Allgemeine Depressions-Skala – Kurzform' (ADS-K; General Depression Scale – Short version; Hautzinger and Bailer, 1993). The ADS-K is a 15-item self-report scale designed to measure depressive symptoms in the general population. The items include depressed mood, somatic complaints, attention deficit, loss of energy, motivational deficits and negative patterns of thought. Participants rate the frequency of 15 symptoms over the past week on a four-point scale (ranging from 'rarely' to 'mostly'). The total score is calculated by summing all items, and ranges from 0 to 45, with higher scores indicating greater depressivity. The ADS-K has been validated in a study with the following samples: respondents from the general population ($N = 1298$), 156 psychiatric patients mainly suffering from depression, 29 neurological patients and 105 patients suffering from chronic pain. Results support the validity and reliability of reports of current depressive symptoms obtained with the ADS-K (Hautzinger and Bailer, 1993).

Procedure

Initial screening and clinical evaluation

Participants underwent a two-step evaluation process. First, each person was interviewed by telephone to establish his or her eligibility for the study. Participants were ruled out if they did not fulfill the inclusion criteria or if they met any exclusion criteria as described above. Second (after the telephone screening), all potential participants were interviewed by one of our clinical research coordinators. The interview included a semi-structured sleep-history interview to diagnose primary insomnia according to DSM-IV (APA, 1994), the German version of the 'Structured Clinical Interview for DSM-IV/Axis I Disorders' (SCID-I; Wittchen *et al*., 1997) to evaluate the presence of other psychiatric disorders, a questionnaire to assess relevant somatic disorders, as well as a questionnaire to obtain demographic information.

The study protocol complied with the ethical principles of the Declaration of Helsinki and was approved by the Ethics Committee of Lucerne (Switzerland). At the end of the initial screening interview, details of the study protocol were discussed with each participant individually, after which participants gave their written informed consent. Prior to their first night in the sleep laboratory, participants completed at home the PSQI, CTQ and the questionnaire assessing the three most negative and most positive events in their lives.

Investigation in the sleep laboratory

Between 2 and 4 weeks after having successfully completed the screening and evaluation procedures, participants spent three consecutive nights in the sleep laboratory. They arrived at the laboratory about 3 h before their usual bedtime, typically between 19:30 and 20:30 hours. They were instructed to abstain from caffeine and alcohol for 24 h before their admission. Also, daytime naps were not permitted during the laboratory phase. Each participant was assigned his or her own room for the course of the study. Each room contained a standard hospital bed, additional furniture and a separate bath room. Participants attended the study in groups of three. In the morning, they were woken between 05:30 and 07:00 hours and received breakfast, after which they usually left the laboratory and spent the day at home.

The first night served as an adaptation night to the sleep laboratory setting. After their arrival to the laboratory, participants completed the self-report questionnaires ADS-K, DHS, and FKL. Electrodes were then attached for PSG recording. Participants spent the remaining time until lights out with various leisure activities, such as reading, conversation, watching television, etc.

Autobiographic memory recalls were carried out the second and third night. After having had the electrodes attached, about 1 h before going to sleep, participants were randomly instructed to recall either extremely positive or extremely negative life events. In addition, participants rated their mood, alertness, and relaxation on the KUSTA shortly before and after these manipulations. To assess the impact of the presleep manipulation on salivary cortisol levels, two salvia samples were collected 1 and 10 min before the manipulations, and four samples were obtained 1, 10, 20, and 30 min afterward. On all three nights, polysomnographic and actigraphic recordings were carried out. These recordings typically began between 22:30 and 23:30 hours and ended between 05:30 and 07:00 hours. Nursing staff registered rising and bedtime. These were defined as light on in the morning and light out in the evening, so that reading or resting in bed before actual sleep intention was not considered a part of the night-time period.

Data analysis plan

Data were analyzed with the Statistical Package for Social Sciences Version 13.0 (SPSS Inc., Chicago, IL, USA). First, the sample was divided into two groups based on statements made in the CTQ: persons with reports of traumatic childhood experiences of moderate to extreme severity and persons with reports of traumatic childhood experiences of low severity or with no such reports. Second, descriptive analyses were computed for the two groups and the groups were compared in terms of various psychometric parameters. Subsequently, Wilcoxon tests or Student's *t*-tests for paired samples were carried out in order to test the effects of autobiographic memory recall on subjective mood, relaxation and alertness, and salivary cortisol. To examine the effects of presleep stimulation on polygraphically and actigraphically assessed sleep, 2×2 multivariate analysis of variance (MANOVA) or covariance (MANCOVA; controlling for age) with repeated measures were used with group as the between-subject factor and memory activation condition as the within factor. Because the memory activation conditions were randomized across the two test nights, night 1 versus night 2 effects were not considered in statistical analysis. For follow-up analyses, analyses of variance (or covariance) of the sleep data over the two experimental nights were conducted. α-level was set at 0.05 for all analyses.

RESULTS

Participants' reports of childhood trauma

Participants were divided into two groups based on statements made in the CTQ: persons with reports of adverse experiences in early life of moderate to extreme severity (high-ACE group) and persons with no reports or reports of ACE of low severity (low-ACE group). According to the guidelines established by Bernstein and Fink (1998) for classifying scores on each CTQ-subscale respecting severity of abuse and neglect, a participant was allocated to the high-ACE group if he or she reported at least on one subscale a traumatic event of at least moderate severity. Persons reporting no traumatic childhood experiences or experiences of only low severity were allocated to the low-ACE group. Twenty-seven participants (22 women and 5 men) were allocated to the high-ACE group, and 32 (23 women and 9 men) to the low-ACE group. Frequencies of types of childhood trauma based on the cut-offs recommended by Bernstein and Fink (1998) are presented in Table 1. Means and standard deviations of subscale scores are presented in Table 2. As can be seen in Table 1, in our sample, the most traumatic experiences of moderate to severe level were reported in the domains emotional and physical neglect. Thirteen participants of the high-ACE group reported only one type of traumatic experience of moderate to extreme level, nine reported having experienced childhood traumas of at least moderate severity in two domains, three in three domains, and one person each in four and five domains.

Table 1 Number of participants fulfilling the respective criterion for the severity of a traumatic experience in the CTQ subscales ($N = 59$)

	Severity of the traumatic experience			
Type of traumatic experience	*None or minimal*	*Low to moderate*	*Moderate to severe*	*Severe to extreme*
Emotional abuse	45 (76.3)	11 (18.6)	3 (5.1)	0 (0.0)
Physical abuse	51 (86.4)	4 (6.8)	2 (3.4)	2 (3.4)
Sexual abuse	46 (78.0)	6 (10.2)	3 (5.1)	4 (6.8)
Emotional neglect	17 (28.8)	18 (30.5)	10 (17.0)	14 (23.7)
Physical neglect	30 (50.8)	18 (30.5)	10 (17.0)	1 (1.7)

Values are given as *n* (%).

Global characteristics of the sample

Global characteristics of the two groups are described in Table 2. Groups did not differ in sex, subjective sleep quality, depressivity, daily hassles, and critical life events in the last 6 months. All participants scored more than 5 on the PSQI indicating poor overall sleep quality. The two groups differed significantly in age with the high-ACE group being slightly older on average.

Trends related to age and to the duration of the insomnia disorder

To evaluate in which way age and duration of insomnia affect the relevant psychological, polysomnographic and actigraphic measures, Pearson and Spearman correlations between these measures and the participants' age were calculated. None of the psychological and actigraphic measures were significantly correlated with age. However, age was significantly correlated with most polysomnographic measures with older participants tending to have a more disturbed sleep according to PSG. Therefore, age was covaried in the respective analyses. As duration of insomnia was not significantly correlated with any of the psychological and sleep-related variables, it was not taken into account for further analyses.

Effect of the autobiographic memory activation on mood, relaxation, alertness, and salivary cortisol

Table 3 depicts results for the subjective and neuroendocrine measurements immediately before and after the recall of negative and positive autobiographic memories. According to Mann–Whitney *U*-tests and *t*-tests, respectively, there were no differences between the two groups in baseline values.

Concerning subjective measurements, the recall of positive memories was not associated with a change in any of the variables measured, apart from a marginally significant increase in relaxation in the low-ACE group. After the recall of negative memories a significant decrease in subjective mood, relaxation and alertness was found in the high-ACE group. In the low-ACE group, there was a significant decrease only in subjective alertness.

Table 2 Demographic and psychometric data of the two groups ($N = 59$)

	High-ACE group (n = 27)	*Low-ACE group* (n = 32)	*Difference* (t-, U- or χ^2-test)
Sex, *n* (%)			
Female	22 (81.5%)	23 (71.9%)	$\chi^2 = 0.75$; df = 1; $P = 0.388$
Male	5 (18.5%)	9 (28.1%)	
Age	46.9 (6.8)	40.8 (9.5)	$t = 2.88$, $P = 0.006$**
Traumatic childhood experiences (CTQ)			
Emotional abuse	8.3 (2.8)	6.1 (1.5)	$z = -3.70$, $P = 0.001$***
Physical abuse	6.8 (2.9)	5.3 (0.6)	$z = -2.29$, $P = 0.022$*
Sexual abuse	7.3 (4.4)	5.2 (0.5)	$z = -2.14$, $P = 0.032$*
Emotional neglect	17.7 (2.6)	9.1 (3.0)	$t = 11.59$, $P = 0.001$***
Physical neglect	8.9 (1.8)	6.2 (1.3)	$t = 6.96$, $P = 0.001$***
Subjective sleep quality (PSQI)	9.9 (2.9)	9.3 (2.7)	$t = 0.78$, $P = 0.437$
Depressivity (ADS-K)	9.8 (6.6)	9.4 (5.0)	$t = 0.27$, $P = 0.785$
Daily hassles, last month (DHS)	1.6 (0.4)	1.6 (0.3)	$t = 0.33$, $P = 0.743$
Critical life events, last 6 months (FKL)	0.2 (0.1)	0.2 (0.2)	$t = -0.69$, $P = 0.497$

Values are given as mean (SD). CTQ, Childhood Trauma Questionnaire; ADS-K, General Depression Scale – Short version; DHS, Daily Hassles Scale; FKL, Questionnaire Assessing Critical Life Events. *$P \leq 0.05$; **$P \leq 0.01$; ***$P \leq 0.001$; tested two-tailed.

Table 3 Means (and standard deviations) for mood, relaxation, alertness, and salivary cortisol level before and after presleep stimulation (activation of negative and positive autobiographic memories)

	High-ACE group (n = 26)		*Low-ACE group* (n = 30)	
	Activation of negative memories	*Activation of positive memories*	*Activation of Negative memories*	*Activation of positive memories*
Mood				
Before	7.5 (1.1)	7.4 (1.1)	7.5 (1.0)	7.0 (1.5)
After	7.0 (1.2)	7.5 (1.2)	7.2 (1.1)	7.1 (1.4)
Difference	$z = -1.94$, $P = 0.026$*	$z = -0.54$, $P = 0.294$	$z = -1.15$, $P = 0.126$	$z = -0.66$, $P = 0.254$
Relaxation				
Before	7.3 (1.4)	7.0 (1.7)	7.1 (1.4)	6.5 (2.0)
After	6.8 (1.3)	7.1 (1.3)	6.9 (1.7)	6.8 (1.8)
Difference	$z = -3.13$, $P = 0.001$***	$z = -0.05$, $P = 0.482$	$z = -0.95$, $P = 0.170$	$z = -1.60$, $P = 0.055^{\dagger}$
Alertness				
Before	5.7 (1.7)	5.1 (2.0)	5.5 (1.9)	4.6 (2.1)
After	5.0 (1.9)	5.0 (1.9)	5.0 (2.0)	4.5 (1.9)
Difference	$z = -1.98$, $P = 0.024$*	$T = 0.27$, $P = 0.400$	$z = -1.97$, $P = 0.025$*	$t = 0.47$, $P = 0.320$
Salivary cortisol (nmol L^{-1})				
Before	1.5 (0.9)	2.0 (2.1)	1.5 (1.4)	1.7 (1.6)
After	2.1 (1.5)	2.5 (2.8)	2.3 (2.3)	1.8 (1.1)
Difference	$z = -1.37$, $P = 0.085^{\dagger}$	$z = -1.54$, $P = 0.062^{\dagger}$	$z = -2.89$, $P = 0.002$**	$z = -0.80$, $P = 0.212$

Mood, relaxation, and alertness scores could range from 1 to 9, with higher scores indicating a more cheerful, carefree mood, more relaxation and calmness and more alertness. Lower scores indicate a more sorrowful, depressed mood, tenseness and low alertness (tiredness and lack of energy). Differences between pre- and post-treatment are tested with Wilcoxon tests or paired Student's *t*-tests. $^{\dagger}P < 0.10$, *$P \leq 0.05$, ***$P \leq 0.001$; tested one-tailed.

To test the salivary cortisol response to the manipulations, the minimum of the two baseline samples was compared to the maximum postmanipulation cortisol value. As can be seen in Table 3, the high-ACE group exhibited marginally significant increases in salivary cortisol from pre- to postmanipulation in both conditions. In the low-ACE group, a significant difference from pre- to postmanipulation was found only after the activation of negative memories.

Additionally, for each of these measures, 2×2 analyses of variance (ANOVA) with repeated measures were computed, using time (pre- and postmanipulation) as the two repeated dimensions and group as the between factor. Because there is no nonparametric alternative to the repeated measures ANOVA, we used this type of analysis despite the non-normal distribution of most of the dependent measures. These analyses revealed: (1) significant main effects for time on mood ($F(1, 54) = 5.82$, $P = 0.019$), relaxation ($F(1, 54) = 6.54$, $P = 0.013$), alertness ($F(1, 54) = 5.52$, $P = 0.022$) and salivary cortisol ($F(1, 55) = 7.09$, $P = 0.010$) in the negative memory activation condition; (2) no significant main effects for time in the

positive memory activation condition; (3) no significant main effects for group; and (4) no significant interactions between group and time.

Polygraphic sleep data

To examine effects of the presleep activation of autobiographic memories on polygraphic sleep measures of the two groups, a 2 × 2 (i.e. group × memory activation condition) MANCOVA with repeated measures was conducted with 11 polygraphic sleep measures as the dependent variables and age as the covariate. The only significant effect this analysis revealed was for the interaction between group and polysomnographic measures, $F(10, 45) = 2.13$, $P = 0.041$. Because the two memory activation conditions did not affect polygraphic sleep measures, we further examined group differences independently of these presleep manipulations. For this purpose, mean values were derived over the two test nights, and single analyses of covariance were conducted for each of these polygraphic sleep measures, controlling for age. Table 4 displays means, standard deviations and test results. As apparent from Table 4, both groups differ significantly concerning NWAK and number of movement arousals, with the high-ACE group exhibiting a greater NWAK and more movement arousals compared with the low-ACE group. Furthermore, we found marginally significant group differences on amount of stage 1 and SWS, with the high-ACE group showing a greater amount of stage 1 and a decreased amount of SWS.

Actigraphic sleep data

Unfortunately, the study was subject to several mishaps, leading to loss of actigraphic data for 22 participants. On several occasions, the actigraph routine check was not properly carried out, leading to a range of unfavourable consequences: In three cases, the actigraphic recordings were overwritten before being saved. In four cases, defect actigraphs were used, and in another three cases, run-down batteries were not replaced in due time. Moreover, 12 temporarily saved data sets were lost due to hard-disk failure. All cases of data loss were accidental and did not happen on a systematic basis. The following analyses were performed with the data of the remaining 37 participants. However, this sub-sample did not differ significantly from the total sample in the relevant demographic and psychometric data.

To examine effects of the presleep activation of autobiographic memories on actigraphic sleep of the two groups, a 2 × 2 (i.e. group × memory activation condition) MANOVA with repeated measures was conducted with the three actigraphic sleep measures as the dependent variables. This type of analysis was used despite the non-normal distribution of the actigraphic data because there is no non-parametric alternative to the repeated measures MANOVA. We did not use MANCOVA in these analyses, because the actigraphic measures were not significantly correlated with age. This analysis revealed a significant effect for group ($F(1, 33) = 7.35$; $P = 0.011$) and a marginally significant interaction between group and actigraphic sleep measures, $F(2, 32) = 3.18$, $P = 0.055$. No significant effects were found for memory activation condition, the interaction between group and memory activation condition, as well as the interaction between memory activation condition and actigraphic measures.

Because the two memory activation conditions did not affect actigraphic sleep measures, we further examined group differences independently of these presleep manipulations. For the further analysis of the significant group effect, mean values were derived over the two test nights, and univariate analyses of variance (ANOVA) were computed for each of these sleep measures. As can be seen in Table 5, there were significant group differences on all actigraphic sleep measures, with the high-ACE group exhibiting lower SE, more body movements

Table 4 Comparisons of the polysomnographic data for the two groups: means (standard deviations) and test results

Mean values for both test nights	*High ACE-group (n = 27)*	*Low ACE-group (n = 32)*	*Group differences (ANCOVA)**
SOL (min)	12.4 (6.5)	13.9 (8.6)	$F(1, 56) = 0.09$, $P = 0.771$
SE (%)†	86.1 (9.0)	89.3 (7.0)	$F(1, 56) = 1.53$, $P = 0.222$
NWAK (%)‡	3.4 (1.9)	2.3 (1.4)	$F(1, 56) = 4.72$, $P = 0.034$
Number of cortical arousals (%)†	4.7 (2.8)	3.6 (3.0)	$F(1, 56) = 2.59$, $P = 0.113$
Number of movement arousals (%)†	5.8 (3.4)	4.4 (2.3)	$F(1, 56) = 6.81$, $P = 0.012$
Number of respiratory arousals (%)†	1.3 (2.6)	0.6 (1.2)	$F(1, 56) = 0.35$, $P = 0.559$
Stage 1 (%)†	7.6 (3.5)	5.7 (3.1)	$F(1, 56) = 3.92$, $P = 0.053$
Stage 2 (%)†	52.1 (6.4)	48.1 (6.9)	$F(1, 56) = 2.71$, $P = 0.105$
Slow wave sleep (%)†	17.9 (7.5)	23.1 (6.8)	$F(1, 56) = 3.76$, $P = 0.057$
REM sleep (%)†	22.3 (4.6)	23.2 (4.4)	$F(1, 56) = 0.94$, $P = 0.336$
REM sleep latency (min)	81.5 (23.4)	89.6 (26.7)	$F(1, 56) = 0.87$, $P = 0.356$

*Analyses of covariance, controlling for age.
†Expressed as percentage of total sleep time
‡Expressed as percentage of sleep period time.

Table 5 Means (standard deviations) for the wrist actigraphy sleep scores of the two groups

Mean values for both test nights	*High-ACE group* (n = *16*)	*Low-ACE group* (n = *21*)	*Group differences* (*ANOVA*)
SE	76.5 (19.9)	87.2 (7.5)	$F(1, 35) = 5.66$, $P = 0.023$
Number of body movements*	38.8 (47.7)	12.8 (10.5)	$F(1, 35) = 6.45$, $P = 0.015$
Moving time*	23.3 (17.0)	12.7 (5.8)	$F(1, 35) = 7.76$, $P = 0.008$

*Expressed as percentage of total sleep time.

in sleep and greater moving time compared with the low-ACE group.

DISCUSSION

The aim of the present study was to examine the relationship between self-reported ACE and sleep in a sample of primary insomniacs under two presleep stimulation conditions. It was hypothesized that such a relationship exists and should become more apparent after recall of negative autobiographic memories before bedtime compared with after recall of positive memories before going to sleep.

Our results indicate that adverse experiences in childhood and adolescence seem to be associated with sleep in adults suffering from primary insomnia. Twenty-seven (46%) participants reported serious experiences of abuse and neglect in childhood and adolescence, particularly emotional and physical neglect. This group featured several sleep characteristics in two consecutive nights in the sleep laboratory, independently of the two study conditions. According to the polysomnographic measures, insomniacs with high ACE exhibited a significant greater number of awakenings and more movement arousals compared to patients with low ACE. According to the actigraph-derived sleep measures, participants with a history of abuse and neglect tended to spend a greater percentage of sleep in motion and have lower SE over two laboratory nights. These results are consistent with those of Sadeh *et al.* (1995) and Glod *et al.* (1997) which suggest that a history of sexual and/or physical abuse is associated with increased nocturnal activity in children and adolescents. Sleep disturbances as long-term effects have also been reported for children and adolescents having experienced minor head injuries (Kaufman *et al.*, 2001), road traffic accidents (Ellis *et al.*, 1998) or burns (Kravitz *et al.*, 1993). For adults, strong long-term effects of trauma on sleep have been reported for combat veterans (Neylan *et al.*, 1998) and Holocaust survivors (Kaminer and Lavie, 1991). Cuddy and Belicki (1992) studied a sample of female university students and found a higher nightmare and night terror frequency, as well as greater difficulties returning to sleep after awakenings from nightmares for those students who reported a history of sexual or physical abuse. Also, Noll *et al.* (2006) found in a longitudinal, prospective study that women that were sexually abused in childhood reported significantly greater rates of sleep disturbances in later adulthood than comparison participants.

Contrary to our assumption, group differences in sleep measures appeared independently of the two presleep stimulation conditions. Thus, the hypothesis that in insomniacs with serious childhood experiences, recall of negative memories before bedtime would irritate subsequent sleep could not be confirmed. These findings can be interpreted in different ways. First, the induced negative mood changes might not have had an impact on subsequent sleep. Similar findings were reported earlier by Lauer *et al.* (1987) who also observed only small influences on poststress sleep patterns in healthy subjects. On the other hand, these observations are at odds with those of Cluydts and Visser (1980), who found that clear variations in presleep mood were accompanied by variations in subsequent sleep. Secondly, it can be assumed that manipulation effects of the present study were too small. Decrease of mood ratings immediately after negative memory recall was statistically significant, but participants did not shift into a very negative mood state, considering the postmanipulation means of mood and relaxation (7.0 ± 1.2 resp. 6.8 ± 1.3 on a nine-point bipolar scale). On the other hand, the cortisol measurements indicate that the activation of negative autobiographic memories had an instant stress-inducing effect on both groups. Yet, this effect might have been too weak to have an effect on sleep in the longer term. This assumption is supported by the finding that the stress response measured by salivary cortisol was comparably marginal and did not reach a mean increase of 2.76 nmol L^{-1}, as proposed by several authors as pre- to postmanipulation stress response-criterion (Kirschbaum and Hellhammer, 1989). A third assumption is that participants of the present study recovered from their stress response before bedtime. Memory activation procedures lasted about 45 min. During this time, persons could probably habituate to their stressful memories. Furthermore, after memory recall, participants could spend time together until they went to bed. This might have taken their mind off negative memories and prevented them from ruminating on negative thoughts. In addition, the activation of negative memories might have had a cathartic effect on the participants and hence might have been less stressful than expected. On the other hand, the unfamiliarity of mentally reliving a negative event might possibly be perceived as a major stressor by anyone. Assuming that the previous emotional processing in the high-ACE group had been considerable, while individuals with low ACE had only experienced limited or no previous emotional processing at all, the activation of negative memories could even have been more stressful for the low-ACE group. Thus it is conceivable that the stress-induction did not produce the desired effect for the high-ACE group as the stressful memories had long before been emotionally processed.

The question whether the employed manipulation did not produce the expected stress-inducing effect or if indeed stress does not lead to more serious sleep impairments in individuals with high ACE than in individuals with low ACE must be left unanswered. The detected sleep-related group differences could be due to a fundamentally different sleep pattern in the high-ACE group which, independent of acute stressors, might be more restless and vulnerable. The results reported by Bader *et al.* (2007) support this hypothesis. In primary insomniacs who were monitored for seven consecutive nights at home with wrist actigraphs, ACE proved to be important predictors of actigraphically assessed sleep at home. Further research is required in order to investigate more thoroughly whether acute stressors have an additional disturbing effect on sleep in individuals reporting ACE. In future studies, the use of real-life stressors such as divorce, examinations or loss of a relative should be considered.

The results of our study lead to the question which mechanisms can explain a possible relationship between ACE and sleep abnormalities in adults. Hyperarousal is a very frequently observed consequence of trauma exposure, explaining the occurrence of sleep disturbances in patients suffering from PTSD. A history of early abuse and neglect is usually associated with long-standing or traumatic ordeals which lead to an adaptive process of the organism and can then result in the persistence of stress-related neurophysiologic patterns, e.g. chronically elevated levels of catecholamines (Otte *et al.*, 2005), elevation of hypothalamic–pituitary–adrenal (HPA) axis, resulting in higher stress reactivity in the course of time (Perry and Pollard, 1998). With regard to the etiology of insomnia, a comparison to PTSD-patients seems evident.

According to Perlis *et al.* (1997) hyperarousal is considered a heterogeneous phenomenon, comprised of somatic, cortical and cognitive aspects. The EEG activity of insomniacs during sleep as well as during wakefulness is characterized by a higher amount of beta activity, which is a psychophysiological feature associated with heightened attention and other cognitive functions (Perlis *et al.*, 2001). A higher level of sensory and information processing makes it difficult to (1) disengage from the environment and initiating sleep, (2) remain asleep and (3) perceive sleep 'as sleep'. From a neurocognitive perspective it may be assumed that when exposed to stressful events, maltreated and traumatized individuals show an increased risk for the activation of memories or schemas which are related to ACE. Therefore, nightmares and anxiety dreams may also be considered a result of this mental activity as well as a result of the physiological changes described above. Apart from that, sleep disturbances for their part can lead to a greater level of distress in trauma patients (Krakow *et al.*, 2004), potentially impeding the adequate emotional processing of traumatic experiences even more.

Gregory *et al.* (2006) provide another possible explanation. The authors assume that growing up in disorganized familial structures may be incompatible with good sleep hygiene (e.g. lack of sleep rituals, inappropriate levels of noise), which has a negative impact on sleep and might lead to the development of inadequate sleep patterns that can persist far into adulthood. Correspondingly, Gregory *et al.* (2005) found significant correlations between family chaos and sleep disturbances in children. As the selected sample was mainly affected by neglect (rather than abuse), it is conceivable that the participants were simply never instructed how to adopt a good sleep schedule.

Several limitations need to be considered when interpreting the results of this study. The sources of data on ACE are retrospective self-reports, and no external sources were contacted to verify childhood history. Therefore, results can only demonstrate associations between these self-reported experiences and objective sleep measures, and no conclusions about causality between real exposure and sleep may be allowed. In a recently published review by Hardt and Rutter (2004), the authors concluded that adults' retrospective reports of their own adverse experiences in childhood seem to be valid in the sense that when abuse and neglect is retrospectively reported to have taken place, these positive reports are likely to be correct. On the other hand, Hardt and Rutter (2004) found that even with well-documented serious abuse or neglect, about a third of individuals do not report its occurrence when specifically asked about it in adult life, i.e. retrospective reports are likely to provide underestimates of the incidence of abuse and neglect. Furthermore, the Childhood Trauma Questionnaire used in the present study focuses primarily on personal maltreatment experiences in the domains abuse and neglect and ignores other types of traumatic experiences someone might undergo (e.g. severe personal illness or accident, exclusion in school, mental illness, suicidal tendencies, imprisonment of a household member, violence against a household member, etc.). Thus, it is possible that in the present study persons were wrongly allocated to the low-ACE group, even though they had actually experienced severe maltreatment in early life. A third limitation refers to the fact that the study sample was comprised of highly selected persons with primary insomnia, who were participating as unpaid volunteers. Because of the fact that ACE seem to constitute a general risk factor for adult emotional disturbance, it is possible that the exclusion of other mental disorders except insomnia has curtailed existing effects. On the other hand, it was important to ensure that given effects could conclusively be traced back to insomnia and not to other forms of psychopathology. Thus, the extent to which the current findings will generalize to other populations with adverse childhood histories must await future research.

As a last concern, it should be considered that the actigraphy data must be interpreted with some caution. As several studies have documented, the actigraphy algorithm chosen for this study is only marginally reliable for distinguishing between quiet wakefulness and sleep, and it therefore tends to overestimate sleep in individuals with insomnia while they are lying quietly waiting to fall asleep (e.g. Hauri and Wisbey, 1992; Vallières and Morin, 2003). Yet, both groups in the present study were composed of insomniacs, and PSG data indicated similar results as actigraphy data. For future research, the study results by Lichstein *et al.* (2006) could be

taken into consideration, which demonstrated strong correspondence between actigraphy and PSG using the high-sensitivity algorithm of Actiwatch in a large insomnia sample.

In summary, the present study data suggest that report of childhood maltreatment is related to impaired sleep in adults suffering from primary insomnia. Based on the present results, it is impossible to draw any conclusions about the causality of the detected correlations. For this purpose, more prospective longitudinal studies in this field of research would be necessary. If a causality of this sort would be found, the findings would contain important implications for the treatment of primary insomnia and for further research, suggesting that clinicians should integrate the assessment of trauma history in their diagnostic routines for sleep disorders in order that childhood trauma history is not overlooked as a possible predisposing factor. The assessment of trauma history may provide further useful information which might contribute to amplifying the scope of conventional insomnia therapy by further treatment strategies. Elements of trauma therapy, much like they are used for patients with PTSD, might be helpful for this purpose (e.g. Krakow and Zadra, 2006).

ACKNOWLEDGEMENT

This study was supported by the Swiss National Foundation, Grant no. 1114-067146.

REFERENCES

Ackerman, P. T., Newton, J. E., McPherson, W. B., Jones, J. G. and Dykman, R. A. Prevalence of post-traumatic stress disorder and other psychiatric diagnoses in three groups of abused children (sexual, physical, and both). *Child Abuse Negl.*, 1998, 22: 759–774.

American Psychiatric Association (APA). *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*, 4th edn. APA, Washington DC, 1994.

American Sleep Disorders Association (ASDA). The Atlas Task Force; EEG arousals: scoring rules and examples. *Sleep*, 1992, 15: 174–184.

Bader, K., Goetschel, R., Caposcale, Y. and Hobi, V. Einschätzung der Schlafqualität Erwachsener im Zusammenhang mit Schwere und Dauer von Belastungen in Kindheit und Jugend. *Verhaltenstherapie*, 1999, 9(Suppl. 1): 3.

Bader, K., Schäfer, V., Schenkel, M., Nissen, L., Kuhl, H. C. and Schwander, J. Increased nocturnal activity associated with adverse childhood experiences in patients with primary insomnia. *J. Nerv. Ment. Dis.*, 2007, 195: 588–595.

Barlow, D. H. *Anxiety and Its Disorders: The Nature and Treatment of Anxiety and Panic*, 2nd edn. Guilford, New York, 2002.

Bastien, C. H., Vallières, A. and Morin, C. M. Precipitating factors of insomnia. *Behav. Sleep Med.*, 2004, 2: 50–62.

Bernstein, D. P. and Fink, L. *Childhood Trauma Questionnaire: A Retrospective Self-report*. The Psychological Corporation, San Antonio, 1998.

Binz, U. and Wendt, G. Kurz-Skala Stimmung/Aktivierung. In: CIPS (Ed.) *Internationale Skalen für Psychiatrie*. Beltz Test GmbH, Weinheim, 1986: 181–135.

Bodenmann, G. *Dyadisches Coping: Eine systemisch-prozessuale Sicht der Stressbewältigung in Partnerschaften: Theoretischer Ansatz und empirische Befunde*. Unveröffentlichte Habilitationsschrift, Universität Fribourg, Fribourg, 1998.

Bodenmann, G., Schwerzmann, S. and Cina, A. Kritische Lebensereignisse und Alltagsstress bei Depressiven und Remittierten. *Z. Klin. Psychol. Psychiatr. Psychother.*, 2000, 48: 1–17.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R. and Kupfer, D. J. The Pittsburg sleep quality index: a new instrument for psychiatric practice and research. *Psychiatry Res.*, 1989, 28: 193–213.

Chapman, D. P., Whitfield, C. L., Felitti, V. J., Dube, S. R., Edwards, V. and Anda, R. F. Adverse childhood experiences and the risk of depressive disorders in adulthood. *J. Affect. Disord.*, 2004, 82: 217–225.

Cluydts, R. and Visser, P. Mood and sleep: II. Effects of aversive presleep stimulation. *Waking Sleeping*, 1980, 4: 199–203.

Cuddy, M. A. and Belicki, K. Nightmare frequency and related sleep disturbance as indicators of a history of sexual abuse. *Dreaming*, 1992, 2: 15–22.

Dong, M., Giles, W. H., Felitti, V. J., Dube, S. R., Williams, J. E., Chapman, D. P. and Anda, R. F. Insights into causal pathways for ischemic heart disease: adverse childhood experiences study. *Circulation*, 2004, 110: 1761–1766.

Dube, S. R., Anda, R. F., Felitti, V. J., Edwards, V. and Croft, J. B. Adverse childhood experiences and personal alcohol abuse as an adult. *Addict. Behav.*, 2002, 27: 713–725.

Ellis, A., Stores, G. and Mayou, R. Psychological consequences of road traffic accidents in children. *Eur. Child Adolesc. Psychiatry*, 1998, 7: 61–68.

Espie, C. A. Insomnia: Conceptual issues in the development, persistence, and treatment of sleep disorder in adults. *Annu. Rev. Psychol.*, 2002, 53: 215–243.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M. P. and Marks, J. S. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: the adverse childhood experiences (ACE) study. *Am. J. Prev. Med.*, 1998, 14: 245–258.

Glod, C. A., Teicher, M. H., Hartman, C. R. and Harakal, T. Increased nocturnal activity and impaired sleep maintenance in abused children. *J. Am. Acad. Child Adolesc. Psychiatry*, 1997, 36: 1236–1243.

Goodwin, R.D. and Stein, M. B. Association between childhood trauma and physical disorders among adults in the United States. *Psychol. Med.*, 2004, 34: 509–520.

Gregory, A. M., Eley, T. C., O'Connor, T. G., Rijsdijk, F. V. and Plomin, R. Family influences on the association between sleep problems and anxiety in a large sample of pre-school aged twins. *Pers. Indiv. Dif.*, 2005, 39: 1337–1348.

Gregory, A. M., Caspi, A., Moffitt, T. E. and Poulton, R. Family conflict in childhood: a predictor of later insomnia. *Sleep*, 2006, 29: 1063–1067.

Hardt, J. and Rutter, M. Validity of adult retrospective reports of adverse childhood experiences: review of the evidence. *J. Child Psychol. Psychiatry*, 2004, 4: 260–273.

Hauri, P. J. and Wisbey, J. Wrist actigraphy in insomnia. *Sleep*, 1992, 15: 293–301.

Hautzinger, M. and Bailer, M. *Allgemeine Depressions-Skala (ADS): Manual*. Beltz Test, Weinheim, 1993.

Kaminer, H. and Lavie, P. Sleep and dreaming in Holocaust survivors: dramatic decrease in dream recall in well-adjusted survivors. *J. Nerv. Ment. Dis.*, 1991, 179: 664–669.

Kaufman, Y., Tzischinsky, O., Epstein, R., Etzioni, A., Lavie, P. and Pillar, G. Long-term sleep disturbances in adolescents after minor head injury. *Pediatr. Neurol.*, 2001, 24: 129–134.

Kirschbaum, C. and Hellhammer, D. H. Salivary cortisol in psychobiological research: an overview. *Neuropsychobiology*, 1989, 22: 150–169.

Krakow, B. and Zadra, A. Clinical management of chronic nightmares: imagery rehearsal therapy. *Behav. Sleep Med.*, 2006, 4: 45–70.

Krakow, B., Haynes, P. L., Warner, T. D., Santana, E., Melendrez, D., Johnston, L., Hollifield, M., Sisley, B. N., Koss, M. and Shafer, L. Nightmares, insomnia, and sleep-disordered breathing in fire evacuees seeking treatment for posttraumatic sleep disturbance. *J. Trauma. Stress*, 2004, 17: 257–268.

Kravitz, M., McCoy, B. J., Tompkins, D. M., Daly, W., Mulligan, J., McCauley, R. L., Robson, M. C. and Herndon, D. N. Sleep disorders in children after burn injury. *J. Burn Care Rehabil.*, 1993, 14: 83–90.

Lauer, C., Riemann, D., Lund, R. and Berger, M. Shortened REM latency: a consequence of psychological strain? *Psychophysiology*, 1987, 24: 263–271.

Lichstein, K. L., Stone, K. C., Donaldson, J., Nau, S. D., Soeffing, J. P., Murray, D., Lester, K. W. and Aguillard, N. Actigraphy validation with insomnia. *Sleep*, 2006, 29: 232–239.

Morin, C. M. *Insomnia: Psychological Assessment and Management*. Guilford Press, New York, 1993.

Neylan, T. C., Marmar, C. R. and Metzler, T. J. Sleep disturbances in the Vietnam generation: findings from a nationally representative sample of male Vietnam veterans. *Am. J. Psychiatry*, 1998, 155: 929–933.

Noll, J. G., Trickett, P. K., Susman, E. J. and Putnam, F. W. Sleep disturbances and childhood sexual abuse. *J. Pediatr. Psychol.*, 2006, 31: 469–480.

Otte, C., Neylan, T. C., Pole, N., Metzler, T., Best, S., Henn-Haase, C., Yehuda, R. and Marmar, C. R. Association between childhood trauma and catecholamine response to psychological stress in police academy recruits. *Biol. Psychiatry*, 2005, 57: 27–32.

Perlis, M. L., Giles, D. E., Mendelson, W. B., Bootzin, R. R. and Wyatt, J. K. Psychophysiological insomnia: the bahavioural model and a neurocognitive perspective. *J. Sleep Res.*, 1997, 6: 179–188.

Perlis, M. L., Merica, H., Smith, M. T. and Giles, D. E. Beta EEG activity and insomnia. *Sleep Med. Rev.*, 2001, 5: 363–374.

Perry, B. D. and Pollard, R. Homeostasis, stress, trauma, and adaptation. A neurodevelopmental view of childhood trauma. *Child Adolesc. Psychiatr. Clin. N. Am.*, 1998, 7: 33–51.

Radloff, L. S. The CES-D Scale: a self-report depression scale for research in the general population. *Appl. Psychol. Meas.*, 1977, 1: 385–401.

Rechtschaffen, A. and Kales, A. *A Manual of Standardized Terminology, Technique and Scoring System for Sleep Stages of Human Sleep*. United States Government Printing Office, Washington DC, 1968.

Sadeh, A., McGuire, J. P. D., Sachs, H., Seifer, R., Tremblay, A., Civita, R. and Hayden, R. M.. Sleep and psychological characteristics of children on a psychiatric inpatient unit. *J. Am. Acad. Child Adolesc. Psychiatry*, 1995, 34: 813–819.

Spertus, I. L., Yehuda, R., Wong, C. M., Halligan, S. and Seremitis, S. V. Childhood emotional abuse and neglect as predictor of psychological and physical symptoms in women presenting to primary care practice. *Child Abuse Negl.*, 2003, 27: 1247–1258.

Vallières, A. and Morin, C. M. Actigraphy in the assessment of insomnia. *Sleep*, 2003, 26: 902–906.

Wittchen, H. U., Wunderlich, U., Gruschwitz, S. and Zaudig, M. *SKID-I: Strukturiertes Klinisches Interview für DSM-IV – Achse I: Psychische Störungen*. Hogrefe, Göttingen, 1997.

Zlotnick, C., Warshaw, M., Shea, M. T., Allsworth, J., Pearlstein, T. and Keller, M. B. Chronicity in posttraumatic stress disorder (PTSD) and predictors of course of comorbid PTSD in patients with anxiety disorders. *J. Trauma. Stress*, 1999, 12: 89–100.