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Sleep recordings in individuals with borderline personality disorder before and after trauma therapy

Sara Lena Weinhold[1] · Robert Göder[1] · Astrid Pabst[1] · Anna-Lena Scharff[1] · Maggie Schauer[3] · Paul Christian Baier[1] · Josef Aldenhoff[1] · Thomas Elbert[3] · Mareen Seeck-Hirschner[2]

Abstract Most individuals diagnosed with borderline personality disorder (BPD) have been exposed to severe and traumatic stressors and thus frequently present with symptoms of a posttraumatic stress disorder (PTSD). Severe sleep disturbances often accompany these complex cases, but changes of sleep parameters during therapy and the impact of sleep on treatment response have barely been studied. Narrative Exposure Therapy (NET) is an evidence-based approach for the treatment of trauma-related psychological disorders. To investigate the effect of NET on sleep in patients with BPD and comorbid PTSD, we screened 45 inpatients and outpatients who met the inclusion criteria of both diagnoses according to DSM-IV and who had a minimum of 2 weeks' stable medication. Patients were allocated to NET ($N = 13$) or treatment as usual (TAU; $N = 8$) in blocks. Polysomnographies and psychological questionares were performed before, directly and 6 months after the last therapy session. The aim of this pilot study was to investigate the effectiveness of trauma therapy by NET on sleep quantity (total sleep time) and sleep continuity (sleep efficiency and awakenings) in patients with comorbid BPD and PTSD. Participants of the NET group compared with those who received TAU showed an increased reduction in sleep latency from baseline to the end of therapy and a reduction in arousals over time. Patients with longer pre-treatment total sleep time and pre-treatment REM sleep duration showed a better outcome of NET with respect to PTSD symptoms. NET seems not lead to a change in sleep for the worse during therapy and seems to improve sleep as good as treatment as usual. Furthermore, our results provide evidence of an influence of sleep structure at baseline on treatment success later on.

Keywords Borderline personality disorder · Narrative exposure therapy · Sleep · Polysomnography · Psychotherapy

S. L. Weinhold and R. Göder contributed equally to this article.

✉ Robert Göder
robert.goeder@uksh.de

1 Department of Psychiatry and Psychotherapy, Christian Albrechts University Kiel, Niemannsweg 147, 24105 Kiel, Germany

2 Department of Psychiatry, Diakonissen Hospital, Flensburg, Germany

3 Department of Psychology, University of Konstanz, Constance, Germany

Introduction

Borderline personality disorder and posttraumatic stress disorder

According to DSM V borderline personality disorder (BPD) is characterised by a pervasive pattern of instability in interpersonal relationships, self-image, and affects, as well as markedly impulsive behaviour (American Psychiatric Association 2013). The development of BPD has not been completely understood until now. Genetic factors and adverse life events seem to interact and lead to the disorder (Leichsenring et al. 2011). Furthermore, there is a lot of evidence indicating a link between negative life events in childhood, such as trauma and sexual abuse, and the development of BPD (Ogata et al. 1990; Herman et al. 1989; Terr 1991; Zweig-Frank and Paris 1991; Silk et al. 1995; Yen et al. 2002). Consistent with this observation, there is a high comorbidity of posttraumatic stress disorder

(PTSD) and BPD (Driessen et al. 2000; Yen et al. 2002; Gunderson and Sabo 1993). That leads to the discussion of BPD as a complex and chronic form of early-onset PTSD after multiple and chronic traumatisation in childhood and adolescence. However, this concept has been revised by newer studies (Zanarini et al. 1998; Zimmerman and Mattia 1999; Golier et al. 2003).

Sleep of patients with borderline personality disorder

Sleep changes in BPD are not as clearly defined as in PTSD, where disturbance of sleep is already defined by the DSM V criteria. In studies with BPD patients, up to 95.5 % of investigated patients reported having subjective sleep problems, mainly difficulties falling asleep and sleep interruptions (Asaad et al. 2002; Semiz et al. 2008; Plante et al. 2013b). Defining sleep problems of patients with BPD is difficult because of high rates of comorbidity with other psychiatric disorders known to be associated with sleep problems (i.e. depression or PTSD, see above). However, even without comorbidity there is a significant correlation between subjective sleep disturbance and borderline symptoms (Harty et al. 2010). In addition, patients with BPD have a high rate of hypnotic and sedative medication use which gives further evidence for sleep problems (Plante et al. 2009). Important for this study are the findings of a higher remission rate in BPD patients without subjective sleep problems (Plante et al. 2013a, b). However, because of discrepant results in subjective and objective methods, sleep problems need further evaluation by objective measurements (Philipsen et al. 2005). Polysomnographic studies in BPD are listed in Fig. 1. Most studies have reported increases in sleep onset latency and decreases in total sleep time and sleep efficiency. Some studies have also described decreases in REM sleep latency and increases in the duration of REM sleep (see Table 1).

Psychotherapy of patients with borderline personality disorder and PTSD

While the standard treatment of BPD is Dialectic Behavioural Therapy (DBT), which includes a stabilization phase with skills training before any exposure to memories of traumatic experiences in a second step (Linehan et al. 1994), numerous studies have shown that various forms of exposure offer effective treatment of PTSD (Benish et al. 2008; Bisson and Andrew 2007). In cases of comorbidity, selection of the best treatment strategy seems to be difficult. One reason for this is that most investigations of therapy outcome exclude patients with BPD because of their high drop-out rate. Only few studies of patients with comorbid PTSD and BPD are available. Whereas Clarke et al. reported on the benefit of therapy in patients with

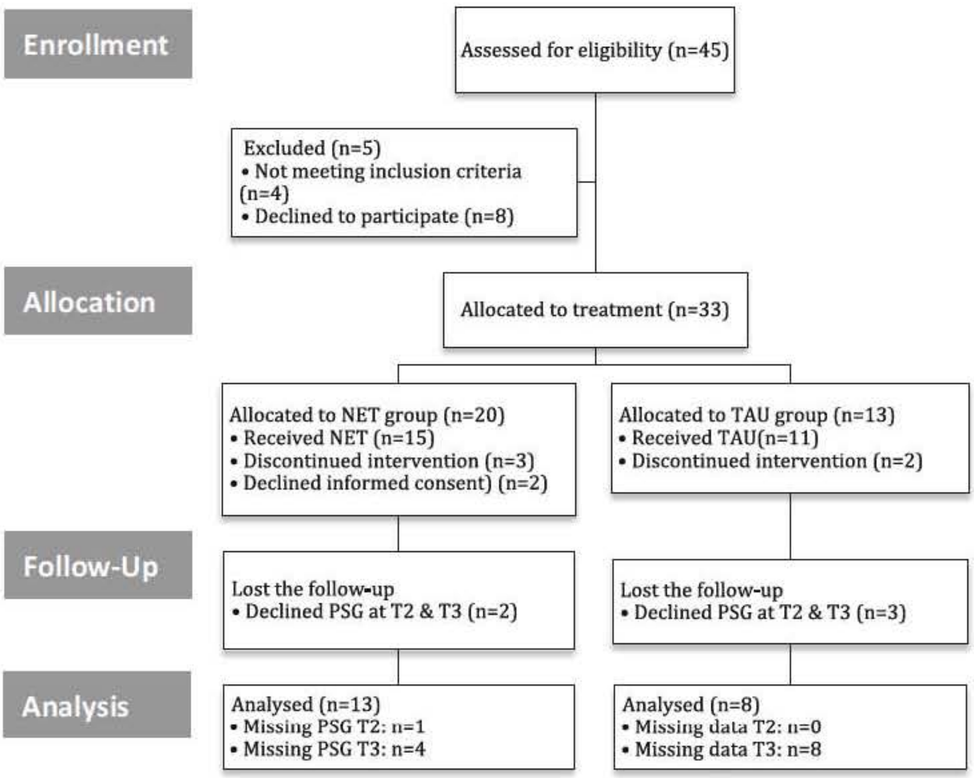

Fig. 1 Flow chart of study participants

Table 1 Shown are results of polysomnography studies in BPD with healthy control subjects

Author	Year	*N*	Comorbidity of BPD patients	Sleep lat	TST	SE	Arousals	REM lat	REM	SWS
Reynolds et al.	1985	10	/	↓	↓	↓	/	ns	ns	ns
Benson et al.	1990	10	Substance abuse	ns	↓	ns	/	ns	ns	↓
Battaglia et al.	1993	10	Substance abuse, histrionic personality disorder	↑	/	↓	/	↓	ns	ns
De La Fuente et al.	2001	20	Substance abuse	↑	↓	/	/	ns	↑	↓
Asaad et al.	2002	20	/	↑	/	↓	ns	↓	↑	↓
De La Fuente et al.	2004	20	Depression, substance abuse	↑	↓	/	/	/	↑	↓
Philipsen et al.	2005	20	Anxiety and eating disorder	ns	ns	ns	ns	↘	ns	ns
Hornung et al.	2008	9	Depression	ns	ns	/	/	ns	↗	ns
Bastien et al.	2008	12	/	↑	↓	↓	/	ns	ns	ns
Schredl et al.	2012	27	PTSD, anxiety, eating and adjustment disorder	↑	/	↘	↑	↓	ns	ns

N number of patients in the borderline group; sleep lat sleep latency, *TST* total sleep time, *SE* sleep efficiency, *REM* lat latency from falling asleep to first REM sleep, *N1* nonREM sleep 1, *N2* nonREM sleep 2, *SWS* slow wave sleep, *ns* no significant differences, *WASO* wake after sleep onset

↑ significantly higher in BPD, ↓ significantly lower in BPD, ↗ trend for higher values in BPD, ↘ trend for lower values in BPD, / = no data

BPD and PTSD by exclusive PTSD treatment also in view of their BDP symptoms, McDonagh et al. found no benefit of PTSD treatment in such a patient collective (Clarke et al. 2008; McDonagh et al. 2005). First analysis of our patient collective outcome showed improvement of PTSD symptoms, depression, and dissociation by trauma therapy (Pabst et al. 2012).

Concept of narrative exposure therapy

Narrative Exposure Therapy (NET) is an evidence-based approach for the treatment of trauma-related psychological disorders (Schauer et al. 2011). In NET, the patient with the assistance of the therapist constructs a chronological narrative of his/her life story with a focus on the traumatic experiences. After a structured clinical interview, patients were requested to place the most important life events into chronological order. In the following therapy sessions, the therapist guides the patients through a chronological narration of his/her life. Fragmented reports of the traumatic experiences are thusly transformed into a coherent narrative. Empathic understanding, active listening, congruency, and unconditional positive regard are key components of the therapist's behaviour. For traumatic stress experiences, the therapist asks in detail about emotions, thoughts, sensory information, and physiological responses. The patient is encouraged to relive these emotions while narrating without losing the connection to the "here and now": by using permanent reminders that the feelings and physiological responses result from memories, the therapist links the experiences to episodic facts, i.e. time and place. Given its focus on the autobiographical elaboration of traumatic experiences, NET is particularly suited for populations affected by multiple and continuous traumatic experiences.

The aim of this pilot study was to investigate the effectiveness of trauma therapy by NET on sleep quantity (total sleep time) and sleep continuity (sleep efficiency and awakenings) in patients with comorbid BPD and PTSD. In this context, we expected NET to improve sleep at least as well as treatment as usual directly after treatment (primary hypothesis). Furthermore, we investigated the effect of NET on sleep over time up to 6 months after treatment (secondary hypothesis). Additionally, we wanted to analyse sleep parameters as a predictor for treatment outcome. As a third exploratory hypothesis, we supposed that sleep parameters would be able to predict the outcome of NET therapy in comorbid borderline and PTDS.

Materials and methods

Subjects

We originally screened 45 patients between January 2009 and June 2011 from the patient collective of the Department of Psychiatry and Psychotherapy in Kiel. Inclusion criteria were a comorbid diagnosis of a borderline personality disorder and a posttraumatic stress disorder according to DSM-IV and a minimum of 2 weeks' stable medication. For this pilot study, we used a small sample size to demonstrate reasonability for studies with lager sample sizes and multi-center experiments. Furthermore, we needed this study to determine the effect size for sufficient sample size calculation for future studies.

Exclusion criteria were severe mental disorders (i.e. drug abuse, psychoses), current use of psychoactive substances other than those prescribed for medical purposes, simultaneous participation in other studies, pregnancy or nursing, acute suicidal behaviour and a BMI lower than 18. Pharmacological therapy was not an exclusion criterion but the medication was adjusted prior to therapy and had to remain unchanged during treatment. Subjects were blinded regarding the purpose of the study.

Study design

The local ethics committee approved the study and subjects' informed consent was obtained according to the Declaration of Helsinki. The ClinicalTrials.gov Identifier is NCT01033708. To evaluate the effect of NET on sleep, we used a quasi-experimental design. Because of the pilot character of the study instead of determination of sample size based on effect sizes we planned for a period of 2.5 years to finish data collection.

The therapy sessions took place at the in Department of Psychiatry and Psychotherapy of the University Hospital of Schleswig Holstein in Kiel Germany, and the sleep recordings and psychometrical measurement were conducted at the Center of Sleep Medicine also at the University Hospital Schleswig Holstein in Kiel (in the same building). All patients underwent a diagnostic screening before study participation. The patients were allocated to a NET or a TAU group in blocks in an approximately matched style depending upon the severity of the symptoms and psychosocial circumstances (Pabst et al. 2014). Patients of the NET group participated in three experimental blocks, each with two nights under polysomnographic conditions. The first block for baseline measurement was placed before the first therapy session (T0). Four weeks after the first therapy session, patients participated in the second test block without sleep recording (T1). These data are reported elsewhere (Pabst et al. 2014). To test direct therapy effects, the next test block came directly after the end of therapy (T2). To evaluate long-term effects, patients were tested again 6 months after the end of therapy (T3). Patients of the TAU group did not participate in T3.

Each experimental block includes two nights under polysomnographic conditions. For each block, the first night was meant for adaptation; the second night was the test night. Patients were not to drink coffee, black, or green tea after 4 pm or to sleep during the day. Patients arrived at 9 pm and were then prepared for polysomnographic recording. Depending on individual sleep habits, patients switched off the lights between 9:30 pm and 12:00 am. At 6:30 am patients were woken up.

Therapy

NET The basis of the treatment was the manual of NET therapy (Schauer et al. 2005, Hogrefe). Sessions of 90 min took place once or twice a week. Experienced clinicians, who had received a 2-day training in NET by a multiprofessional team, carried out all sessions.

TAU The therapists were the same as for the NET group. The treatment included DBT and behavioural therapeutic elements, as well as other standard treatment such as psycho-education, improvement of coping skills and crisis intervention.

Polysomnography

Polysomnographic procedures were performed and rated according to the criteria of Rechtschaffen and Kales. Electroencephalographic activity (EEG), electrooculographic activity (EOG) and submental electromyographic activity (EMG) were measured (Viasys, Germany). Sleep stage scoring was performed visually by a trained rater who was blinded to the experimental conditions and the hypotheses. The following parameters were computed: sleep onset latency (min), total sleep time (min), sleep efficiency (ratio of total sleep time to time in bed in percent), number of awakenings, stage 1 sleep (min), stage 2 sleep (min), slow wave sleep (min) and REM sleep (min), REM latency (time from sleep onset to the first epoch of stage REM sleep in min) and arousals (abrupt shift in EEG frequency for 3 10 s).

Psychological tests

Psychological tests were performed before the therapy, directly after and 6 months after the last therapy session. Psychologists experienced in working with traumatised patients performed them:

Posttraumatic Stress Diagnostic Scale (*PDS*) a German version of the self-rating questionnaire by Edna Foa that consists of 49 items with a maximum of 51 points to detect and range the severity of PTSD symptoms (Foa 1995; Ehlers and Steil 1996).

Pittsburgh Sleep Quality Index (*PSQI*) is a self-rating assessment of 19 questions of sleep habits and sleep-related events in the last 2 weeks (Buysse et al. 1989).

Hamilton Depression Rating Scale (*HAMD*) a rating instrument for the external assessment of depression symptoms (Hamilton 1967).

The results of the study concerning the psychometric effects of NET versus TAU are reported elsewhere (Pabst et al. 2014).

Statistics

Descriptive data are presented as mean ± standard deviations. Analyses of the primary hypothesis were based on multifactorial univariate analyses of variances with the within factor time (T0, T2) and the between factor group (NET, TAU). In the case of missing sphericity, values were corrected using the Huynh Feldt method. The secondary outcome was determined by unifactorial univariate analyses of variances for repeated measures with the within subject factor time (T0, T2, T3). The explorative hypotheses were tested by unifactorial univariate analyses of variances with the between subject factor median split (TST and REM duration). The effect sizes were expressed by partial eta squared and its 90 % confidence intervals. Relevance of mean differences was determined by p values. The alpha level was set at 5 % to show differences and at 20 % to show equality (to reduce indirect β-error). Statistical analyses were performed by SPSS Statistics version 21.0. We disclaimed of alpha adjustment for multiple testing because of the pilot character of this study.

Results

Sample

We finally included 13 patients who received NET (4 exclusively outpatient and 9 partly in-patient) and 8 patients (4 exclusively outpatient and 4 partly inpatient) who received treatment as usual (TAU) to answer the hypotheses. For more detailed recruitment flow see Fig. 1. Mean age at the beginning of therapy was 31.9 ± 8.3 years in the NET and 29.5 ± 11.6 in the TAU condition ($p = 0.530$). The number of school years were 9.8 ± 1.5 in the NET group and 10.9 ± 2.4 in the TAU group ($p = 0.218$). No differences were found in PSQI score (NET: 13.4 ± 2.6; TAU: 11.6 ± 4.0; $p = 0.274$) and HAMD score (NET: 26.5 ± 6.2; TAU: 23.3 ± 6.8; $p = 0.306$) between the two groups at T0. Differences in baseline PDS score could not be excluded (NET: 35.0 ± 4.8; TAU: 38.5 ± 4.9; $p = 0.130$). Because of symptom severity, most of the patients also received pharmacological therapy. The list of medications is shown in Table 2.

In NET two patients (13 %) and in TAU three patients (27 %) discontinued their treatment. All patients had already had therapy experience (psychological and/or pharmacological), whereas none had received trauma-focused treatment before. There are missing data in the NET group as follows: one PDS at T2, 2 PSQI at T0, 5 PSQI at T2, 3 PSQI at T3, 1 sleep recording at T2, 3 sleep recordings at T3 and in TAU group 1 PSQI at T2. No subject in the TAU group participated in any test at T3.

Table 2 Number of patients who took antidepressants, antipsy chotics, GABA agonists or were not medicated in the NET and in the TAU group before and after the therapy (T0 and T2) and 6 months after the last therapy session (T3)

	NET (N 13)		TAU (N 8)	
	T0 and T2	T3	T0 and T2	T3
Antidepressant	12	10	4	4
Antipsychotic	3	3	1	2
GABA agonist	2	0	0	0
None medication	1	2	3	3

Comparison of NET and TAU over time (changes from T0 to T2)

The NET group ($N = 12$) and TAU group ($N = 8$) showed differences at T0 and T2 with respect to REM density, awakenings and arousals (significant GROUP main effect, see Table 3). The NET group showed more reduction in sleep latency from baseline (T0) to end of therapy (T2) than the TAU group. No significant changes were found in all other sleep parameters. In particular, no sleep parameters were worse after NET than after TAU. For more details see Table 3.

Changes in the NET group over the time (T0, T2, T3)

The NET group had significant changes in arousal levels over time (T0, T2, and T3). Post-hoc t tests showed a significant reduction in arousal from T0 to T3 ($p = 0.020$) and from T2 to T3 ($p = 0.007$). No further changes in sleep parameters were observed. For more details see Table 4.

Sleep as predictor for NET outcome

Patients in the NET group with longer REM duration (median split < and > 67.0 min) before therapy (T0) had lower PDS scores directly after therapy (significant differences) and a trend after 6 months (statistical trend). Furthermore there was significantly more reduction in the PDS score from T0 to T2 in the group with more REM sleep (see Table 5). In addition, the group with higher total sleep time (median split ≤ and > 370.0 min) showed trends towards more reduction in the PDS scores from T0 to T2 and from T0 to T3 (statistical trend; see Table 6). Group comparison with other sleep parameters showed no further significant differences.

Table 3 Means (*M*) and standard deviations (SD) of sleep parameters of the two therapy groups (NET and TAU) in the baseline night (T0) and the night directly after the last therapy session (T2)

Sleep parameter	Group	*N*	T0		T2		ME TIME				INT Group × time				ME Group			
			M	SD	*M*	SD	PEs	CI L	CI U	*p*	PEs	CI L	CI U	*p*	pEs	CI L	CI U	*p*
Sleep latency (min)	NET	12	30.2	16.0	24.5	14.9	0.019	0.000	0.187	0.558	0.233	0.012	0.453	0.031	0.005	0.000	0.138	0.764
	TAU	8	20.7	10.3	30.3	17.9												
Total sleep time (min)	NET	12	373.6	84.2	368.7	104.8	0.081	0.000	0.299	0.225	0.108	0.000	0.331	0.158	0.097	0.000	0.319	0.182
	TAU	8	290.3	86.8	351.9	93.2												
Sleep efficiency (%)	NET	12	82.9	11.0	84.5	10.8	0.018	0.000	0.192	0.578	0.000	0.000	0.000	0.994	0.001	0.000	0.044	0.886
	TAU	8	82.2	10.5	83.9	15.1												
REM sleep (min)	NET	12	82.0	52.4	77.4	38.7	0.014	0.000	0.180	0.626	0.045	0.000	0.248	0.369	0.006	0.000	0.147	0.738
	TAU	8	67.0	24.7	82.4	31.3												
REM density (%)	NET	12	5.5	2.6	4.6	3.1	0.003	0.000	0.106	0.822	0.075	0.000	0.297	0.257	0.233	0.009	0.458	0.036
	TAU	7	2.7	0.7	3.3	1.7												
Awakenings (*N*)	NET	12	21.4	13.8	18.0	11.3	0.084	0.000	0.303	0.214	0.000	0.000	0.000	0.994	0.291	0.037	0.502	0.014
	TAU	8	10.6	6.9	7.3	3.1												
Arousals (*N*)	NET	12	70.3	38.7	63.4	25.6	0.008	0.000	0.156	0.710	0.015	0.000	0.185	0.606	0.467	0.161	0.635	0.001
	TAU	8	27.1	19.4	28.3	9.3												
PSQI score	NET	6	14.8	1.9	11.8	4.1	0.131	0.000	0.403	0.224	0.154	0.000	0.426	0.184	0.082	0.000	0.339	0.344
	TAU	7	11.1	4.1	11.3	5.9												

p values and partial Eta squared result from an analysis of variances with the within subject factor TIME and the between subject factor GROUP

ME main effect, *INT* interaction effect, *PSQI* Pittsburgh Sleep Quality Index, *pEs* partial Eta squared, CI, *L* lower limit of the confidence interval for partial Eta squared, CI, *U* upper limit of the confidence interval for partial Eta squared

Table 4 Means (*M*) and standard deviations (SD) of sleep parameters at the 3 testing times (T0, T2, T3) of the NET group

Sleep parameter	*N*	T0		T2		T3		pEs	CI L	CI U	*p*
		M	SD	*M*	SD	*M*	SD				
Sleep latency (min)	9	31.7	16.2	26.6	16.6	64.9	66.3	0.243	0.000	0.437	0.145
Total sleep time (min)	9	362.8	93.0	370.8	120.3	339.3	90.7	0.090	0.000	0.269	0.468
Sleep efficiency (%)	9	81.2	12.0	85.4	11.5	70.7	25.0	0.324	0.006	0.507	0.069
REM sleep (min)	9	70.9	39.4	77.1	43.7	68.7	32.4	0.028	0.000	0.156	0.798
REM density (%)	8	5.4	2.7	5.3	3.3	7.6	2.9	0.252	0.000	0.452	0.131
Awakenings	9	23.3	14.2	17.9	12.9	16.9	15.7	0.121	0.000	0.310	0.357
Arousals	9	79.1	40.0	62.8	26.8	39.7	23.6	0.438	0.080	0.598	0.019
PSQI score	6	14.8	1.9	11.8	4.1	14.7	4.7	0.336	0.000	0.543	0.194

p values and partial Eta squared result from a univariate analysis of variances with the within subject factor TIME

PSQI Pittsburgh Sleep Quality Index, *pEs* partial Eta squared, CI, *L* lower limit of the confidence interval for partial Eta squared, CI, *U* upper limit of the confidence interval for partial Eta squared

Table 5 PDS scores before (T0) directly after (T2) and 6 months after NET therapy (T3) and reduction over time (T2 T0 and T3 T0) in patients with low and high REM sleep duration before therapy (median split $\leq$ and $>$ 61.5 min) in the NET group

	Low	High	pEs	CI L	CI U	*p*
PDS T0 (*N* 13)	39.0 ± 6.6	33.0 ± 4.8	0.237	0.000	0.496	0.091
PDS T2 (*N* 12)	35.5 ± 4.1	20.3 ± 8.1	0.627	0.214	0.765	0.002
PDS T3 (*N* 13)	31.1 ± 8.2	23.5 ± 7.1	0.224	0.000	0.485	0.102
PDS T2 T0 (*N* 12)	1.5 ± 4.5	12.7 ± 6.1	0.563	0.144	0.724	0.005
PDS T3 T0 (*N* 13)	7.9 ± 10.1	9.5 ± 11.2	0.007	0.000	0.197	0.786

p values and partial Eta squared result from a univariate analysis of variances with the between subject factor REM duration (low/high)

PDS Posttraumatic Stress Diagnostic Scale, *pEs* partial Eta squared, CI, *L* lower limit of the confidence interval for partial Eta squared, CI, *U* upper limit of the confidence interval for partial Eta squared

Table 6 PDS scores before (T0) directly after (T2) and 6 months after NET therapy (T3) and reduction over time (T2 T0 and T3 T0) in patients with low and high total sleep time; median split $\leq$ and $>$ 370.5 min) in the NET group

	Low (*M* ± SD)	High (*M* ± SD)	pEs	CI L	CI U	*p*
PDS T0 (*N* 13)	34.9 ± 6.0	37.8 ± 7.1	0.058	0.000	0.307	0.427
PDS T2 (*N* 12)	31.1 ± 9.5	23.4 ± 9.8	0.159	0.000	0.438	0.199
PDS T3 (*N* 13)	31.0 ± 8.3	23.7 ± 7.1	0.206	0.000	0.471	0.199
PDS T2 T0 (*N* 12)	3.7 ± 6.8	11.8 ± 7.1	0.287	0.000	0.541	0.073
PDS T3 T0 (*N* 13)	3.9 ± 10.6	14.2 ± 6.8	0.274	0.000	0.524	0.066

p values and partial Eta squared result from a univariate analysis of variances with the between subject factor total sleep time (low and high)

PDS Posttraumatic Stress Diagnostic Scale, *pEs* partial Eta squared, CI, *L* lower limit of the confidence interval for partial Eta squared, CI, *U* upper limit of the confidence interval for partial Eta squared

Discussion

Despite high rates of traumatic events in BPD, therapy with focus on exposure is not usually used in practise. Because of emotional instability and self-injury to the point of suicide in BPD, therapists do not use exposure without preceding stabilisation. This often leads to avoidance of re-exposure to the early traumata, although the effectiveness of exposure in PTSD has often been shown.

In this pilot study, we were able to show a positive effect of Narrative Exposure Therapy (NET) in comorbid BPD and PTSD on sleep. First one important result is that NET did not lead to a change of sleep for the worse during the therapy in our patients. We even observed a reduction in sleep latency at the end of NET therapy, which we did not observed after TAU. And lastly we had no higher drop-out rate despite stressful exposure during the NET therapy.

Besides the comparison of NET and TAU, this study allows assumptions of possible long-term effects of NET on sleep. We were able to show significantly reduced arousals during sleep in patients 6 months after the last therapy session compared to the beginning of the therapy and directly after the last therapy session. However, we have to interpret these results with caution due to the lacking control group. Concerning all other analysed sleep parameters, we found no changes directly and 6 months after therapy. This is in line with reviews that show only small sleep alterations in BPD (Philipsen et al. 2005).

Furthermore our results give advice for an influence of sleep structure on therapy benefit. There is slight evidence for more reduction in PTSD symptoms directly after therapy in patients with longer REM sleep duration and longer total sleep time before therapy. These data suggest an influence of objective sleep parameters on therapy benefit as it has already been shown for subjective parameters (Plante et al. 2013a, b). Thus better sleep quality seems to be an important condition for treatment response in trauma therapy. Further studies will need to investigate whether or not medications which suppress REM sleep might also lead to less therapy benefit.

Indeed, the interpretation of this study's results has some limitations, a main one being that we have no untreated control group in this study, and procedures in TAU were very manifold. Further limitations are the missing data of the TAU group at T3. In addition, effects of medication on sleep are well known, and there are some differences with respect to medications in NET and TAU group. Because of the evaluative character of this study and the small sample size, we did not adjust alpha for multiple testing. And, lastly, our sample size was very small, therefore the results of this study could be used to plan further studies with larger sample sizes and accurate randomization to the treatment groups.

Besides these limitations, we can conclude that trauma exposure therapy did not worsen sleep in patients with BPD. Indications for better sleep were associated with better treatment outcome with respect to PTSD symptoms, emphasising the importance of good sleep as a condition for successful psychotherapy.

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Compliance with ethical standards

Conflict of interest No conflict of interest exists with regard to the study presented in this paper.

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