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ORIGINAL PAPER

The relation between sleep and pain among a non-clinical sample of young adults

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Abstract Poor sleep is linked to poorer daily functioning and increased risk of psychiatric symptoms. With respect to pain, the relation is bi-directional; poor sleep exacerbates pain, while greater pain adversely affects sleep. Moreover, perception of pain is subject to cognitive-emotional processes. Surprisingly, no data are available from non-clinical samples of young adults. The aim of the present study was therefore to investigate the relation between sleep and pain as a function of quality of life and depressive symptoms in young adults. The direction of influence between sleep and pain was statistically tested with two different structural equation models (SEMs). A total of 862 participants (639 women, 223 men; mean age: 24.67; SD = 5.91) completed a series of validated self-report questionnaires assessing sleep, quality of life, depressive symptoms and cognitive-emotional elaboration of pain. Sleep, pain, quality of life, and depressive symptoms were interrelated. The first SEM suggested both a direct and an indirect influence of pain on sleep, whereas the second SEM suggested that sleep had only an indirect influence on pain. Irrespective of the SEM, the relation between sleep and cognitive-emotional elaboration of pain was mediated by quality of life and depressive symptoms. For a non-clinical sample of young adults, findings did support the bi-directional relation between poor sleep and increased cognitive-emotional elaboration of pain, though other cognitive-emotional processes such as depressive symptoms and quality of life should be taken into account.

Keywords Sleep · Pain · Depressive symptoms · Quality of life · Cognitive-emotional elaboration of pain

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Introduction

Poor sleep is negatively related to quality of life, routine daily functioning [5, 32], memory [52], emotional intelligence [31], and academic performance [15]. Moreover, almost by definition, poor sleep is linked to psychiatric symptoms such as depressive disorders [16, 54], and to generalized [55], specific [42], and state-trait anxiety disorders [30]. Among adolescents, anxiety disorders seem to predict subsequent insomnia, but insomnia also predicts later depressive disorders [29]. Among adults, an overlap has been observed between major depressive disorders, fatigue, dyssomnias, and organic diseases [37]. Most importantly, neuropathic pain also seems to be highly intertwined with both psychiatric diseases and impaired sleep quality [37, 38].

The relation between pain and sleep is bi-directional (cf. [33, 48, 51]). Total sleep deprivation has a hyperalgesic effect. That is, sleep deprivation leads to increased sensitivity to pain (for a comprehensive overview, see [33, 48, 51]). Onen et al. [40] showed in nine pain-free healthy men that 40 h of sleep deprivation reduced their mechanical pain threshold by 8%. Similarly, Roehrs et al. [49] were able to show that both total and partial sleep deprivation reduced pain threshold, though it remained unclear whether sleepiness during the day and mood were possible additive or confounding factors. Finally, Raymond et al. [45], investigating inpatients with burn injuries, found that subjective quality of sleep predicted pain intensity on the

following day, whereas pain intensity did not predict the sleep quality on the following night. Their conclusion was that disturbed sleep seemed to increase pain. To summarize, both laboratory and field studies have shown that sleep loss leads to increased pain perception.

Complementary to this direction of influence, there is also evidence that pain impairs sleep. For example, in an early epidemiological study of 3,201 Swedish men, Gislason and Almquist [20] reported that experience of pain led to sleep disturbances. Sleep has also been found to be reduced in clinical samples of patients suffering from headache [28], with 55% of patients suffering from headache also reporting less sleep [41].

In a community-based study investigating about 19,000 people in five European countries, Ohayon [37] showed that painful chronic physical conditions played a major role on insomnia. The author concluded that such conditions affect sleep as much as mood disorders [37]. Consistent with this, reduced sleep has also been reported for patients suffering from chronic pain such as neuropathic pain and musculoskeletal pain (for overview, see [48]).

Taken together, these studies show that the experience of pain leads to deteriorated sleep. At the same time, a wealth of studies provides evidence for a bi-directional relation between sleep and pain.

When dealing with pain, one of the most challenging issues in researching pain is its subjective nature,, That is to say: setting aside possible objective measurements of the sources of pain (e.g., degree of heating or cooling; voltage of electric shocks; distortion of bones, ankles, organs, dimension of lesions, etc.), the experience and appraisal of pain is a highly cognitive-emotional process [23, 44]. To mention just a few of the many studies focusing on cognitive-emotional elaboration of pain (CEEP), female and male ballet dancers showed higher pain tolerance than controls, as measured in seconds until pain became intolerable in a bowl of ice water [53]. A repeatedly referenced study is that by Henry Beecher [9]. During the Italian campaign in World War II Beecher was engaged as anesthetist in a forward hospital receiving severely wounded soldiers. He observed that these soldiers often reported experiencing little pain; only 25% of those directly questioned indicated that their pain was severe enough to need morphine. By contrast, after returning to civilian life, Beecher noted that injuries comparable to those he had observed and treated among soldiers were reported as very painful by civilians. He concluded that the "context" (understood as cognitive-emotional processes) must have been the reason for the differences in experience. Whereas for a wounded soldier, taken from the battlefield and brought to a safe location, injuries must have been interpreted in terms of life-saving conditions, for a civilian similar injuries in a non-threatening environment must have been appraised as highly painful critical life events leading to severe consequences (see [9, 17, 34]).

Jelicic and Kempen [27] investigated 111 older adults with a fracture of the extremities and observed that experience of pain two months after the injury was predicted by pain at baseline and post-injury anxiety, suggesting that experience of pain perception was at least in part influenced by cognitive-emotional processes such as anxiety. Similarly, both genetic and cognitive-emotional processes (in this case: "pain catastrophizing"; [19]) predicted shoulder pain in 58 patients three to five months after operation [19].

The degree to which pain is subject to cognitive-emotional appraisals is further well documented in the cognitive-behavioral treatment of chronic pain (cf. [25]).

In this regard, there is a substantial overlap between perceived pain and psychiatric disorders. Thus, patients suffering from depressive [37, 59], anxiety [22], and somatoform disorders [50] often complain about physical pain. These patterns of associations suggest that experience of pain is a common feature of psychiatric disorders.

Taken together, there is strong evidence that the experience of pain is a highly cognitive-emotional process. As a consequence, when dealing with the relation between sleep and pain, it seemed reasonable to focus on these cognitive-emotional processes. To this end, two questionnaires (Whiteley-Index, WI; [43]; German adaptation: [47]); Somatosensory Amplification Questionnaire (SAQ [6]) were introduced for tackling these issues. By definition, dysfunctional cognitive-emotional processes result in people who suffer from hypochondriacal symptoms, interpreting normal physiological sensations as evidence of physical illness (DSM-IV, [1]). These cognitive-emotional dysfunctional processes are also prominent in people amplifying somatosensory sensations. The questionnaires have in common a focus on the cognitive-emotional elaboration of pain (see also [46]).

Surprisingly, little is known about the relation of sleep and experience of pain in a non-clinical sample of young adults. However, we believe it is important to fill this gap in knowledge because both sleep disturbances [36] and chronic pain [39] are two complaints of major public concern.

Two hypotheses were formulated. First, following Ohayon [37, 38] and Wise et al. [59], we anticipated a strong correlation between depressive symptoms, quality of life, and cognitive-emotional elaboration of pain (CEEP). Second, we expected a relation between sleep and CEEP. However, with respect to the direction of the relation, we explored whether CEEP (along with depressive symptoms, and the quality of life) predicted reduced sleep quality, as observed by Jennum and Jensen [28], Paiva et al. [41], and Ohayon [37], or whether instead or in addition reduced sleep quality (along with depressive symptoms, and the quality of life) predicted CEEP, as reported in Raymont

et al. [45], Lautenbacher et al. [33], Roehrs and Roth [48], and Smith and Haythornthewaite [51].

Method

Participants

The participants were 862 students ($M = 24.67$, SD = 5.91 years) from the German-speaking North-Western part of Switzerland (223 men: $M = 24.48$, SD = 4.78 years; 639 women: $M = 24.74$, SD = 6.26 years), who were recruited from the University of Basel (sport science: $n = 250$, psychology: $n = 83$, psychiatry: $n = 22$, medicine: $n = 201$) and from the North-Western University of Applied Sciences (psychology: $n = 66$, tourism: $n = 17$, pedagogy: $n = 127$, gymnastics: $n = 96$). Participants were informed about the purpose of the study and the voluntary basis of their participation. All participants were assured of the confidentiality of their responses and gave written informed consent. Participation involved completion of several psychological, sleep and sleep-related questionnaires as detailed in the following paragraphs. The study was approved by the local ethical committee, and therefore, the study was executed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

Assessment of sleep

Two instruments were used, the Insomnia Severity Index (ISI [7]) and the Pittsburgh Sleep Quality Index (PSQI [12]).

To assess sleep quality, participants completed the Insomnia Severity Index (ISI [7]), which is a brief screening measure of insomnia and an outcome measure for use in treatment research. The seven items of the ISI, answered on 5-point rating scales (0 = not at all, 4 = very much), refer in part to DSM-IV criteria for insomnia [1] by measuring difficulty in falling asleep, difficulties maintaining sleep, early morning awakening, increased daytime sleepiness, low daytime performance, low satisfaction with sleep, and worrying about sleep. The higher the overall score, the more the respondent is assumed to suffer from insomnia (Cronbach's alpha = .85).

Sleep quality and quantity was assessed with the Pittsburgh Sleep Quality Index (PSQI [12]); the German adaptation was taken from a conventional and widely used manual for psychological treatment of sleep complaints [4]. Participants answered questions on an 8-point visual analogue scale for evenings about: sleepiness during the day (1 = very sleepy), and concentration during the day (1 = very low concentration), and for mornings, about sleep quality (1 = very bad sleep quality), mood at awakening (1 = very bad mood) and feeling of restoration (1 = not at all restored); Cronbach's alpha = .87. In addition, details of sleep onset latency (in min) and the number of awakenings were requested.

Depressive symptoms

Von Zerssen's Depression Scale [57] is a current and widely used self-rating questionnaire. It consists of 16 items asking about depressive symptoms such as depressed mood ("I feel more depressed in the mornings"), lack of satisfying leisure and social activities ("criticism does hurt me more than before"), hopelessness ("I often feel abysmal"), and sleep complaints. However, to avoid artificial correlations with other instruments related to sleep, the two items related to sleep complaints were omitted. Answers are given on a 4-point rating scale ranging from 1 (=not at all true) to 4 (=definitively true). Higher scores reflect more marked depressive symptoms (Cronbach's alpha = .89).

Cognitive-emotional elaboration of pain

Two instruments were used to assess the cognitive-emotional elaboration of pain (CEEP), the Whiteley-Index (WI [43]; German adaptation: [47]), and the Somatosensory Amplification Questionnaire (SAQ [6]).

The Whiteley-Index (WI [47]) consists of 14 items and assesses hypochondria. From this questionnaire, we extracted three items focusing on cognitive-emotional elaboration of pain: "Are you bothered by many aches and pains?"; "Do you find that you are often aware of various things happening in your body?"; "Do you often have the symptoms of very serious illnesses?". Answers are "yes" (=1) or "no" (=0), with higher sum scores reflecting a greater inclination to overestimate bodily sensations (Cronbach's alpha = .85).

The Somatosensory Amplification Questionnaire (SAQ [6]) consists of ten items focusing on the tendency to experience somatic and visceral sensation as unusually intense and disturbing. The questionnaire involves bodily hypervigilance, the predisposition to focus on certain weak and infrequent bodily sensations, and a tendency to appraise them as pathological and symptomatic of disease, rather than normalizing them. Typical items are: "When I bruise myself, it stays noticeable for a long time"; "Even something minor, like an insect bit or a splinter, really bothers me", or "I have a low tolerance for pain". Answers are given on a 5-point rating scale ranging from 0 (=not at all true) to 4 (=completely true), with higher mean scores reflecting an increased somatosensory amplification (Cronbach's alpha = .89).

Quality of life

To assess quality of life, the SEL (Skalen zur Erfassung der Lebensqualität [3]; [Scales to assess quality of life]) was used. The questionnaire (short version) consists of 14 items asking about somatic complaints (e.g., "I have difficulties with the digestion"), somatic condition (e.g., "Within the last two weeks I often felt tired and exhausted"), general condition (e.g., "Today, I'm feeling tensed"), the degree of negative social contacts (e.g., "I have nobody to whom I can talk about my current concerns and complaints"), and global physical rating (e.g., "My impression is that my global physical condition is bad"). Answers are given on a 5-point rating scale ranging from 1 (=never/not at all true) to 5 (=always/completely true), with higher mean scores reflecting a decreased perceived quality of life (Cronbach's alpha = .79).

Statistical analyses

First, exploratory factor analyses (EFA) were performed (cf. [11]) to explore the factor structure of the Depression Scale [57]. The 14 items yielded 14 factors; the first three had Eigenvalues higher than 1, together accounting for 78% of the overall variance. The Eigenvalue of the first factor, labeled "Depressive thoughts and feelings", was 4.70; the Eigenvalue of the second factor, labelled "Anxiety", was 1.26; the Eigenvalue of the third factor, labeled "Slowed and decreased concentration", was 1.01 (all based on principal component analysis with Varimax factor rotation). Correlations between the psychological dimensions were assessed by Pearson's *r*.

To enter all psychological dimensions and sleep simultaneously, confirmatory factor analyses (CFA) and structural equation modeling (SEM) were conducted using AMOS 6.0 [2]. Parameter estimation was conducted using maximum-likelihood (ML). As generally recommended, multiple goodness-of-fit indices were considered to examine how well the theoretical model fitted the empirical data [26, 35]: AGFI should be $\geq$.95, PClose $>$ .50, CFI $>$ .90, RMR $<$ .08, and RMSEA $\leq$ .05.

Results

Table 1 shows both the descriptive statistics and intercorrelations between variables related to demographics (sex, age, BMI) and to sleep, quality of life, depressive symptoms, and cognitive-emotional elaboration of pain (CEEP). In general, the statistical significance of the many low correlations, those between demographic variables and sleep, quality of life, depressive symptoms, and perceived pain, reflects the large sample size. Therefore, sex, age, and BMI were not regarded as confounding variables. However, there were strong correlations between sleep, quality of life, depressive symptoms, and perceived pain variables, suggesting a reciprocal dependence.

Structural equation model (SEM) with the dimensions sleep, quality of life, depressive symptoms, and CEEP; sleep complaints as a function of CEEP

Figure 1 shows the structural equation model (SEM), with the variable sleep as a function of CEEP. With respect to the criteria (in [squared brackets]) proposed by Hu and Bentler [26] and by McDonald and Ho [35], the model represented an excellent fit: $\chi^2/df = 1.538$, AGFI = .970 [$\geq$.95], PClose = 1.00 [$>$.50], CFI = .991 [$>$.90], RMR = .073 [$<$.08] and RMSEA = .025 [$\leq$.05].

The model revealed that the dimension Low quality of life was the strongest predictor of poor sleep, followed by Depressive symptoms and CEEP. Low quality of life, CEEP, and Depressive symptoms were highly interrelated, suggesting that CEEP may lead to poor sleep via low quality of life and via depressive symptoms. Thus, CEEP seems to influence sleep both directly and indirectly.

Structural equation model (SEM) with the dimensions sleep, quality of life, depressive symptoms, and CEEP; CEEP as a function of sleep complaints

Figure 2 shows the structural equation model (SEM), with the variable perceived pain (CEEP) as a function of sleep. With respect to the same criteria (indicated in [squared brackets]), this model also achieved an excellent fit: $\chi^2/df = 1.241$, AGFI = .976 [$\geq$.95], PClose = 1.00 [$>$.50], CFI = .996 [$>$.90], RMR = .067 [$<$.08] and RMSEA = .017 [$\leq$.05].

The model again revealed that the dimension Depressive symptoms, Low quality of life, and CEEP were interrelated. Here, a direct influence of poor sleep on CEEP was not observed. As the model suggests, poor sleep influenced CEEP concomitantly via depressive symptoms and low quality of life. Thus, an indirect, but not direct, influence of poor sleep on CEEP is indicated.

Discussion

The key findings of the present study are that among a nonclinical sample of young adults, low quality of life, depressive symptoms, poor sleep, and cognitive-emotional elaboration of pain (CEEP) are related, that CEEP influences poor sleep both directly and indirectly, while poor sleep only influences CEEP indirectly.

Two hypotheses were formulated, and each of these is now considered in turn.

Table 1 Descriptive statistics and correlations between demographic variables, depressive symptoms, quality of life, perception and cognitive-emotional elaboration of pain, and sleep

		1	2	3	4	5	6	7	8	9	10	*M*	SD
1	Sex	–				−.23**	.01	.21**	−.15**	−.10**	.00	[f: 639; m: 223]	
2	Age	−.02	-									24.67	5.91
3	BMI	.32**	.08*	–								21.57	2.33
Depressive symptoms													
4	Depressive feelings	−.05	.01	.00	–							1.19	.34
5	Anxiety	−.23**	−.03	−.06	.55**	–						1.5	.51
6	Lack of concentration	.01	.02	.03	.55**	.43**	–					1.2	.41
Quality of life													
7	Somatic complaints	−.21**	−.04	−.07	.43**	.37**	.31**	–				1.82	.59
8	Somatic condition	−.15**	−.00	−.04	.40**	.35**	.28**	.59**	–			2.04	.86
9	Negative social contacts	−.1**	.01	−.02	.54**	.47**	.36**	.31**	.34**	–		1.76	.86
10	General condition	.00	−.04	.01	.27**	.18**	.18**	.15**	.11**	.18**	–	2.65	.37
11	Global physical rating	.06	.05	.07	.45**	.34**	.34**	.47**	.59**	.36**	.11**	3.90	.86
Cognitive-emotional elaboration of pain (CEEP)													
12	Whitley Index (WI) sum score	−.03	.00	.03	.36**	.32**	.24**	.41**	.38**	.25**	.13**	1.23	.59
13	Somatosensory Amplification questionnaire (SAQ) Sum score	−.31**	.10**	−.05	.13**	.24**	.16**	.27**	.24**	.15**	.01	23.32	5.34
Sleep													
14	ISI sum score	−.11	−.01	−.00	.46**	.42**	.40**	.53**	.52**	.33**	.16**	13.55	4.31
15	PSQI mood	−.07*	.00	.01	.33**	.27**	.28**	.37**	.37**	.25**	.13**	2.63	1.63
16	PSQI sleep quality	−.04	−.06	.01	.37**	.33**	.28**	.38**	.47**	.30**	.12**	3.59	1.64
17	PSQI feeling of being restored	.04	−.11**	.04	.27**	.20**	.18**	.28**	.29**	.21**	.05	3.68	1.78
18	PSQI concentration dd	−.04	−.09**	−.00	.32**	.28**	.27**	.37**	.47**	.211**	.13**	3.74	1.54
19	PSQI sleepiness dd	.02	−.06	−.00	.39**	.30**	.39**	.36**	.44**	.272**	.14**	3.25	1.38

		11	12	13	14	15	16	17	18	19	*M*	SD
Cognitive-emotional elaboration of pain (CEEP)												
12	Whitley Index (WI) sum score	.34**	–								1.23	.59
13	Somatosensory Amplification questionnaire (SAQ) Sum score	.16**	.28**	–							23.32	5.34
Sleep												
14	ISI sum score	.44**	.33**	.27**	–						13.55	4.31
15	PSQI mood	.35**	.21**	.17**	.63**	–					2.63	1.63
16	PSQI sleep quality	.37**	.24**	.12**	.54**	.57**	–				3.59	1.64
17	PSQI feeling of being restored	.25**	.17**	.1**	.28**	.34**	.50**	–			3.68	1.78
18	PSQI concentration dd	.38**	.16**	.16**	.48**	.39**	.55**	.33**	–		3.74	1.54
19	PSQI sleepiness dd	.39**	.19**	.16**	.51**	.4**	.51**	.32**	.73**	–	3.25	1.38

Notes: SO, sleep onset; ISI, insomnia severity index; PSQI, Pittsburgh sleep quality index; dd, during the day; BMI, body mass index. Sex: 1, male; 2, female

* $p < .05$, ** $p < .01$

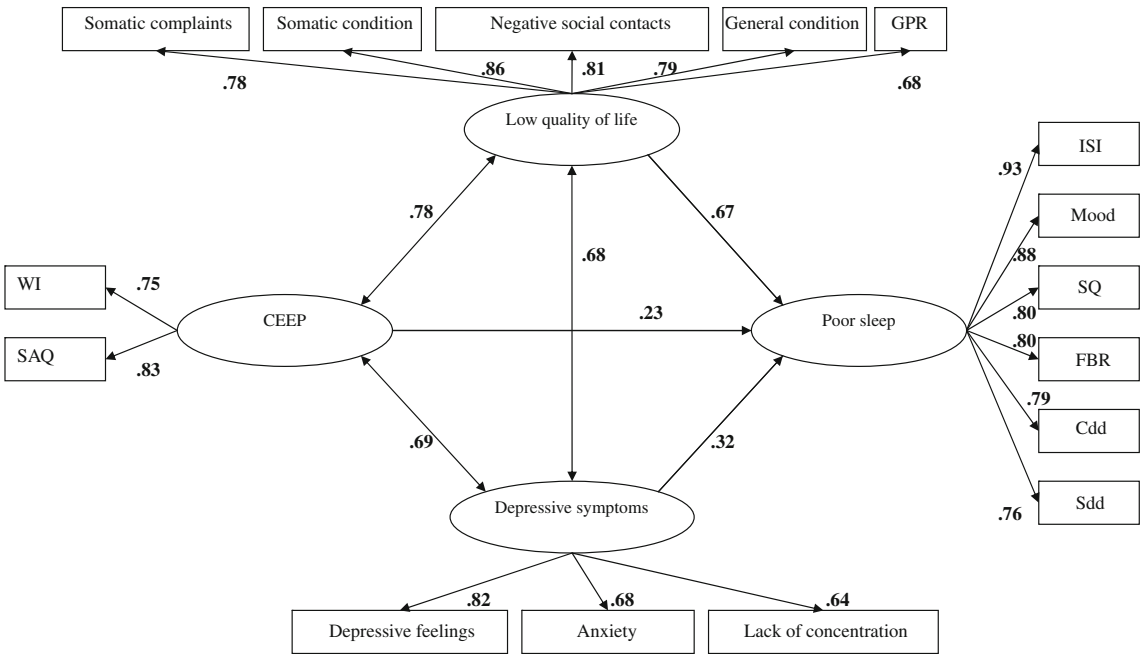

Fig. 1 WI, Whitley index; SAQ, somatosensory amplification questionnaire; CEEP, cognitive-emotional elaboration of pain; GPR, global physical rating; ISI, insomnia severity index; SQ, sleep quality; FBR, feeling of being restored; Cdd, concentration during the day; Sdd, sleepiness during the day. *Arrows* pointing in two directions indicate intercorrelations; *arrows* in one direction indicate that independent variables predict dependent variables

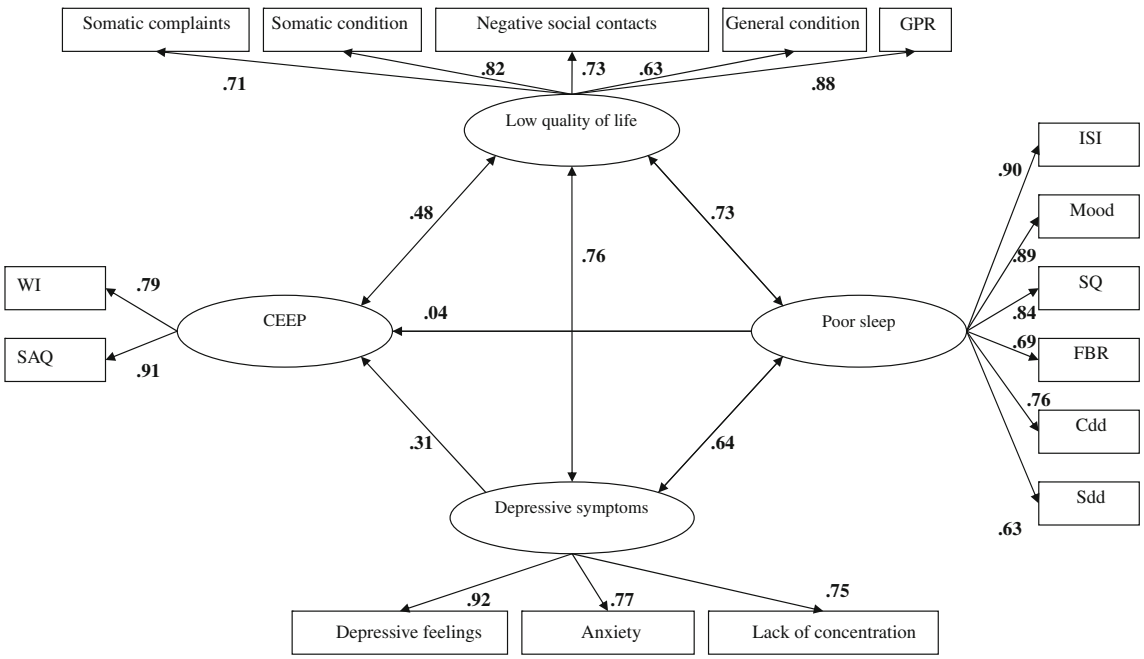

Fig. 2 WI, Whitley index; SAQ, somatosensory amplification questionnaire; CEEP, cognitive-emotional elaborated pain; GPR, global physical rating; ISI, insomnia severity index; SQ, sleep quality; FBR, feeling of being restored; Cdd, concentration during the day; Sdd, sleepiness during the day. *Arrows* pointing in two directions indicate intercorrelations; *arrows* in one direction indicate that independent variables predict dependent variables

First, we anticipated strong associations between depressive symptoms, quality of life, and cognitive-emotional elaboration of pain (CEEP), and correlations confirmed this. Our pattern of results fits well with the wealth of studies pointing to the interdependence of these psychological dimensions. Moreover, results also confirm

that sleep is highly related to psychological functioning (e.g., [5, 15, 37, 38]. Thus, the present findings are also consistent with research in the field of psychopathology which emphasizes the relation between poor sleep and depressive disorders [16, 54].

With the second hypothesis, we expected a relation between sleep and CEEP. However, with respect to the direction of the relation, we considered both possibilities, that CEEP predicts reduced sleep quality, and that reduced sleep quality predicts CEEP, as reported in Raymont et al. [45], Lautenbacher et al. [33], Roehrs and Roth [48], and Smith and Haythornthwaite [51].

Applying SEMs, the first model confirmed that CEEP predicted poor sleep; this is consistent with previously reported findings (e.g. [28, 38, 41]). However, the first SEM also revealed both direct and indirect relations, with CEEP additionally influencing poor sleep indirectly via low quality of life and depressive symptoms. Thus, the present findings contribute to research evidence on the relation between pain and sleep, indicating that additional factors such as depressive symptoms and quality of life should be taken into account. In this regard, our findings also mirror the high overlap between perceived pain and psychiatric disorders observed in patients suffering from depressive [37, 57], anxiety [22], and somatoform pain disorders [50].

With respect to the second hypothesis, we tested for an influence of sleep on CEEP. The SEM suggested no direct relation. Rather, poor sleep may influence CEEP via depressive symptoms and low quality of life. In this respect, the findings are at odds with previous research (cf. [33, 45, 48]). The reason for this lack of consistency with other research remains unclear. However, Lautenbacher et al. [33] and Roehrs and Roth [48], for example, reported results derived primarily from laboratory studies involving application of acute and standardized sources of pain while measuring single night sleep-EEG profiles. Thus, though speculative, one may claim that results from laboratory studies with healthy pain-free volunteers might not necessarily match results from field studies using self-rating questionnaires. One field study [45] that did show a direct influence of poor sleep on perceived pain involved victims of burn injuries; it is unclear whether these data can be generalized or compared with data from a non-clinical sample of young adults. In this respect, one might claim that direct consequences of poor sleep such as decreased mood, increased depressive symptoms and decreased quality of life may be the more important direct causes of increased pain. In this view, this assumption would mirror those studies reporting a close association between poor sleep and depressive disorders (e.g., [16, 24, 54]).

A question arises as to why cognitive-emotional processes should be associated with pain. The underlying neuronal processes could not be assessed in the present study, though there is evidence that, besides the sensory-discriminative afferent pathway bringing pain signals from the periphery to the central nervous system ("Where does it hurt?"), an affective-motivational afferent pathway ("How much does it hurt?") involves brain regions responsible for emotional-cognitive processes (cf. [44]). Simply put, pain signals via the affective-motivational afferent pathway are elaborated in the anterior circular cortex (ACC), insula cortex, prefrontal cortex (PFC), and the amygdala [13]. Activation of these brain centers is typically associated with mood, attention, and fear, and these patterns of activation may explain the high overlap between perception of pain, depressive symptoms, hyper-focus, and anxiety. In particular, the ACC is more closely related with unpleasantness of pain than other cortical structures. Thus, ACC-prefrontal cortical processes seem to add further cognitive evaluations to emotions related to pain [44]. To summarize, neuropsychological processes do modulate cognitive-emotional elaboration of pain, and there is reason to assume that such processes were also active within participants in the present study.

Limitations

Several considerations do warrant against overgeneralization of the findings. First, participants were all students recruited in the German-speaking part of Switzerland. This non-clinical sample is therefore not representative of the general population in early adulthood.

Second, one may claim that students are a rather marginal and healthy population, and that possible ceiling and floor effects may have contributed to the lack of any direct influence of sleep on pain. With regard to psychological functioning, however, there is evidence that students are as much at risk for developing psychiatric symptoms as are other populations [8, 58].

Third, no objective sleep data were collected. Research has repeatedly underlined the usefulness of sleep-EEGs [18, 21, 56] and actigraphs. Self-reports may, in contrast, be more susceptible to expectancy effects. However, there is evidence that retrospective self-reported and consecutive diary-reported sleep patterns are as valid as actigraphically estimated sleep behaviors [60], and that subjective estimates may correspond very closely to objective sleep-EEG recordings [10].

Fourth, the questionnaires used (Whiteley-Index; SAQ) might not accurately reflect pain, although the focus was on cognitive-emotional elaboration of pain. Last, there is the possibility of a response bias: people complaining about poor sleep may be also inclined to complain about other symptoms such as depressive symptoms, increased pain, and stress (cf. [14]). Though, this objection might have

some merit, the same claim may be made about many investigations in the field.

Conclusions

Findings based on a non-clinical sample of young adults did support the bi-directional relation between poor sleep and increased cognitive-emotional elaboration of pain, as has previously been reported for clinical samples, though the present findings indicate that other cognitive-emotional processes such as depressive symptoms and quality of life should also be taken into account.

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