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Day-to-day associations between pain intensity and sleep outcomes in an adult chronic musculoskeletal pain population: A systematic review

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ABSTRACT

Background: In individuals with chronic musculoskeletal pain, a reciprocal relationship between sleep and pain across short and long-term evaluations exists. Sleep influences pain levels, while the level of pain also impairs sleep. However, given the day-to-day variability of both sleep and pain intensity, assessing this relationship within a daily time frame should be considered.
Objectives: To systematically review the literature concerning the bidirectional day-to-day relationship between night-time sleep variables and day-time pain intensity in individuals with chronic musculoskeletal pain.
Methods: A systematic search (final search on October 12, 2023) in four databases (PubMed, Web of Science, Embase, PsycInfo) identified eligible articles based on pre-defined criteria. Three independent reviewers executed data extraction and risk of bias assessment using the "Quality In Prognosis Studies" tool. The study findings were synthesized narratively.
Results: Eleven articles (1014 study participants; 83 associations) were included. A bidirectional relationship between pain intensity and sleep was found. Nine articles indicated night-time sleep quality to be a more consistent predictor for next day pain intensity than vice versa.
Conclusion: Nonetheless the bidirectional day-to-day sleep-pain relationship in individuals with chronic musculoskeletal pain, results suggest that self-reported sleep quality has a stronger predictive value on pain intensity then vice versa.

1. Introduction

Chronic musculoskeletal pain (CMP) stands as a critical global health concern [1]. CMP can be defined as ongoing or recurrent pain lasting more than 3 months and experienced as arising from musculoskeletal tissue(s) including muscle, bone, joints and/or connective tissues. CMP may be secondary to persistent inflammation or structural changes due to musculoskeletal pathology, or defined as chronic primary musculoskeletal pain, as classified according to the International Association for the Study of Pain and International Classification of Diseases 11th Revision [22,23]. It is the foremost contributor to disability on a

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Abbreviations

CMP	Chronic musculoskeletal pain
EMA	Ecological momentary assessment
IASP	International Association for the Study of Pain
NRS	Numerical Rating Scales
QUIPS	Quality In Prognosis Studies
SE	Sleep efficiency
SOL	Sleep onset latency
SQ	Sleep quality
SWiM	Synthesis Without Meta-Analysis guidelines
TST	Total sleep time
VAS	Visual Analogue Scales
VRS	Verbal Rating Scales
WASO	Wake after sleep onset

worldwide scale, impacting daily activities, social interactions, quality of life and other aspects of an estimated 1.71 billion individuals' life globally [1–3]. Furthermore, significant socioeconomic consequences arise as CMP is correlated with absenteeism, presenteeism, and premature retirement [1,2,4].

Sleep problems have been identified as a significant risk factor for CMP patients [5–8]. Approximately 58.7 % of individuals with CMP experience comorbid sleep problems and disorders [8–10]. Moreover, research indicates a bidirectional relationship between sleep and CMP, where pain disrupts sleep continuity and quality, and individuals with sleep problems face an elevated risk of developing CMP [6,8–11]. Recent studies emphasize the predictive role of sleep disturbances in long-term future pain, suggesting that they might be stronger predictors compared to the reverse [5,12,13].

The relationship between sleep and CMP gains complexity given the day-to-day variability in both the level of sleep disturbance and the level of pain. Therefore, assessments that capture this variability are recommended [14]. To examine day-to-day associations between sleep and pain, ecological momentary assessment (EMA) proves instrumental [14]. EMA is characterized by four key aspects: it assesses phenomena as they occur, it requires precise timing of assessments, measurements are conducted in subjects' natural environments, and it typically involves a significant number of repeated observations, though not always [15]. This allows the examination of individuals' real-time experiences and behaviors in their natural environment, instead of relying on retrospective self-reports or laboratory-based assessments, providing real-life data [14]. This method might be more appropriate due to the day-to-day variability of sleep disturbance and the level of pain. Furthermore, the day-to-day interaction between pain and sleep could be influenced by unmeasured confounding processes.

While there is a growing interest in EMA application within the field of CMP, to examine pain and other factors of interest [15–21], a systematic overview of the current state of the research of the day-to-day association between various night-time sleep outcomes and daytime pain intensity in individuals with CMP is lacking [14]. Therefore, the objective of this review is to systematically review the literature concerning the bidirectional predictive value between different night-time sleep outcomes and next day pain intensity in individuals with CMP.

2. Material and methods

The review was preregistered in the PROSPERO database and the PRISMA, SWiM and PERSiST guidelines were followed [24,25].

2.1. Study design and inclusion criteria

The following eligibility criteria were defined for study inclusion.

1) Daily assessments of daytime pain intensity and night-time sleep outcomes were used. Synonyms for this methodology are high intensity longitudinal data sampling, micro-longitudinal assessment, experience sampling methodology, day-to-day assessments, diary assessments, etc.; 2) The association between night-time sleep outcomes and daytime pain intensity was assessed, with 'pain intensity' also including the constructs 'pain level' or 'pain severity', as they are often used interchangeably.; 3) Written in English.; 4) Enrolment of adult individuals (>18 years old), with the whole or majority of the study sample consisting of individuals with CMP. Chronic primary musculoskeletal pain (chronic limb, low back, thoracic, and cervical pain) and chronic widespread pain (fibromyalgia) according to the International Association for the Study of Pain (IASP) classification of chronic primary pain [22] are included in the CMP definition used in this review. Also individuals with chronic musculoskeletal pain associated with structural changes (i.e. osteoarthritis and spondylitis), defined according to the IASP classification of chronic secondary musculoskeletal pain [23], or pain from temporomandibular disorder are included in this study's definition of CMP.

This means that studies with study samples (mainly) consisting of individuals suffering from complex regional pain syndrome, orofacial pain, chronic primary headache, or chronic visceral pain were excluded. Also studies on individuals with chronic pain associated with persistent inflammation or disease of the nervous system are excluded. Lastly, non-musculoskeletal pain diagnoses, such as cancer, diabetes mellitus, sickle cell disease, chronic fatigue syndrome, mental and psychological disorders are excluded.

Assessments of (confounding) factors on the association between a night-time sleep outcome and daytime pain intensity are not required for inclusion. See Fig. S1 for a visual overview of the study populations was included in this review.

2.2. Search strategy

Four databases—PubMed (including Medline), Web of Science, Embase, and PsycInfo (via ProQuest)—were searched for eligible studies. The final search was conducted on October 12, 2023. Textword and Medical Subject Heading (MeSH) terms covered the outcomes of interest ("sleep" and "pain") and the study design ("micro-longitudinal study", "micro-longitudinal design", "ecological momentary assessment", "experience sampling methodology", "diary", "ambulatory assessment", "sleep diary", and "day-to-day"). The specific search strategy for each database are available in Table S1.

2.3. Study selection

Preceding the selection process, duplicates from the database search were removed using Endnote (version X9). The remaining articles were transferred to Rayyan for screening [26]. Three independent reviewers (A.V.S., J.V., Z.G.) carried out the study selection. In instances of disagreement, consensus was reached through discussion with a fourth reviewer (L.D.B.). The inclusion of articles followed a two-step screening process. First review was based on the title and abstract following the eligibility criteria. Second, the remaining articles were screened based on the full text. Additionally, potential eligible studies were identified by the reference lists of the included articles.

2.4. Risk of bias assessment

Three independent reviewers (A.V.S., J.V., Z.G.) assessed the risk of bias using the "Quality In Prognosis Studies" (QUIPS) tool, an electronic spreadsheet (MS Excel) as provided by Hayden et al. (2013) [27] (see Table S4). A fourth reviewer (L.D.B.) was present to discuss potential disagreements. Based on the QUIPS, the following six domains were

Table 1
Study characteristics.

Reference	Country	Pain population	Total sample size (N)	Mean age (SD)	Female N (%)	Daily pain measurements	Follow-up period	Sleep variable (measurement instrument)	Pain intensity (measurement)
Abeler et al., 2021 [39]	Norway	Chronic primary musculoskeletal pain: Cervicalgia (12), Low back pain (11), Pain in thoracic spine (1), Other dorsalgia (2), Dorsalgia, unspecified (11), Myalgia (10), Pain in limb (3), Fibromyalgia (6)	56	41.7 (10.8)	42 (75 %)	1	7 days	**Subjective:** • SQ (VAS) • First sleep attempt • Final morning awakening **Objective:** • TST (actigraphy) • SOL (actigraphy) • WASO (actigraphy) • SE (actigraphy) • Midsleep (actigraphy)	**Subjective:** • Pain intensity (BPI; NRS) o Worst, least and average pain oCurrent pain before bedtime oMean score of these 4 items
Affleck et al., 1996 [31]	US	Primary fibromyalgia syndrome	50	43.86 (8.23)	50 (100 %)	3	30 days	**Subjective**: • SQ (electronic sleep interview)	**Subjective**: • Pain intensity (electronic pain interview), scored as the sum across 14 body regions
Alsaadi et al., 2014 [7]	Australia	Non-specific low back pain; Acute low back pain (25.23 %), Chronic low back pain (52.68)	80	43.9 (15.4)	39 (51 %)	2	7 days	**Subjective**: • Time went to bed (Pittsburg sleep diary) • Lights out time (Pittsburg sleep diary) • SOL (Pittsburg sleep diary) • Time of final waking (Pittsburg sleep diary) • Method of final waking (Pittsburg sleep diary) • WASO (Pittsburg sleep diary) • Reason(s) for WASO (Pittsburg sleep diary) • SQ (Pittsburg sleep diary) • Alertness on final waking (Pittsburg sleep diary) • TST (Pittsburg sleep diary) • SE(Pittsburg sleep diary) **Objective:** • TST (Actigraphy) • SOL (Actigraphy) • WASO (Actigraphy) • SE (Actigraphy)	**Subjective**: • Pain intensity at awaking (Pittsburg sleep diary; NRS) • Average day-time pain intensity (Pittsburg sleep diary; NRS)
Gerhart et al., 2017 [32]	US	Chronic low back pain	105	46.30 (12.1)	51 (49 %)	4	14 days	**Subjective:** • SQ (5-point likert-type scale)	**Subjective**: • Pain intensity (0–8 scale)
Kothari et al., 2015 [33]	US	Fibromyalgia	220	51.25 (11.02)	195 (89 %)	4	21 days	**Subjective**: • SQ (4 items of Pittsburgh SQ Index)	**Subjective**: • Late morning pain intensity (0–100 scale; NRS)
Liszka-Hackzell et al., 2005 [34]	US	Chronic low back pain	18	52 (10.5)	8 (44 %)	9	6 days	**Objective:** • SQ (Actigraphy)	**Subjective**: • Pain level (0–10 scale; every 90 min

(*continued on next page*)

Table 1 (*continued*)

Reference	Country	Pain population	Total sample size (N)	Mean age (SD)	Female N (%)	Daily pain measurements	Follow-up period	Sleep variable (measurement instrument)	Pain intensity (measurement)
								• Actual Sleep Percentage (Actigraphy) • SE (Actigraphy) • Wake Bouts (Actigraphy) • Number of Minutes Immobile (Actigraphy) • Number of Immobile Phases (Actigraphy) • Movement/ Fragmentation Index (Actigraphy)	between 08:00 h and 22:00 h; NRS) • Mean daytime pain level (0–10 scale; every 90 min between 08:00 h and 22:00 h; NRS) • Difference between the total pain level and the daily pain level (0–10 scale; every 90 min between 08:00 h and 22:00 h; NRS)
Mun et al., 2022 [37]	US	Temporomandibular disorder and insomnia complaints	144	36 (11.1)	144 (100 %)	2	2 weeks	**Subjective**: • Bedtime (Daily sleep diary) • Lights-out time (Daily sleep diary) • Final wake-up time (Daily sleep diary) • Out-of-bed time (Daily sleep diary) **Objective:** • Sleep continuity (Actigraphy) • TST (Actigraphy) • SOL (Actigraphy) • WASO (Actigraphy)	**Subjective**: • Pain severity (Pain diary; NRS) • Morning pain (Pain diary; NRS) • Overall daily pain severity (Pain diary; NRS)
O'Brien et al., 2011 [35]	US	Chronic pain; Chronic facial pain (8), Chronic back pain (8), Fibromyalgia (6)	22	43.77 (14.13)	22 (100 %)	2	2 weeks	**Subjective**: • Bedtime (Morning sleep diary) • Rise time (Morning sleep diary) • SOL (Morning sleep diary) • Wake time (Morning sleep diary) • WASO (Morning sleep diary) • SQ (Morning sleep diary) • Factors interfering with sleep, e.g., pain or worries (Morning sleep diary) **Objective:** • SOL (Actigraphy) • WASO (Actigraphy) • SE (Actigraphy) • TST (Actigraphy)	**Subjective**: • Pain intensity (0–10 VAS; at bedtime) • Upon awaking (0–10 VAS; at bedtime)
Tang et al., 2012 [38]	UK	Chronic pain and insomnia complaints: <u>Chronic pain locations:</u> - Lower back (72.8 %), leg (54.4 %), neck (37.7 %), shoulder (32.5 %), knee (35.1 %), arm (21.1 %), upper back (21.9 %), joint (21.9 %) pain. - More than 1 location (86.8 %) <u>Cause of pain:</u> - Disc/cartilage damage (52.9 %), followed by trauma/ injury (32.8 %), arthritis (7.6 %), migraine and headaches	119	46 (10.09)	88 (74 %)	3	7 days	**Subjective**: • Bedtime (Electronic sleep diary; upon waking) • Rise time (Electronic sleep diary; upon waking) • SOL (Electronic sleep diary; upon waking)	**Subjective**: • Pain intensity (electronic diary; 0–10; NRS) • Pain upon waking (electronic diary; 0–10; NRS) • Pain during the first half of the day (electronic diary; 0–10; NRS at midday)

(*continued on next page*)

Table 1 (*continued*)

Reference	Country	Pain population	Total sample size (N)	Mean age (SD)	Female N (%)	Daily pain measurements	Follow-up period	Sleep variable (measurement instrument)	Pain intensity (measurement)
		(5.9 %), fibromyalgia (5 %), endometriosis (2.5 %), ankylosing spondylitis (1.7 %), tendinitis (1.7 %), sickle cell anemia (1.7 %), neurofibromatosis (0.8 %), irritable bowel syndrome (0.8 %), leg ulcer (0.8 %), spina bifida occulta (0.8 %), and no specific cause for their pain (10.9 %). More than one cause (>50 %).						• WASO (Electronic sleep diary; upon waking) • WASO duration (Electronic sleep diary; upon waking) • TST (Electronic sleep diary; upon waking) • SE (Electronic sleep diary; upon waking) • SQ (NRS 0–10) **Objective:** • SE (Actigraphy)	• Presleep pain (electronic diary; 0–10; NRS) • Pain during the second half of the day (electronic diary; 0–10; NRS before bedtime)
Whibley et al., 2019 [36]	US	Osteoarthritis	160	71 (4.5)	99 (62 %)	5	5 days	**Subjective**: • SQ (calculated of 2 questions; 0–4 scale) **Objective:** •TST (Actigraphy) •SOL (Actigraphy) •SE (Actigraphy) •WASO (Actigraphy)	**Subjective**: • Pain intensity, at different times of the day (0–10; NRS; awakening, 11 a.m., 3 p.m., 7 p.m., Bedtime)
Wilson et al., 1998 [40]	Canada	Chronic musculoskeletal pain: - Back pain (67.5 %) - Pain in the cervical region (15 %) - Pain in the shoulder and upper limbs (7.5 %) - Pain in the lower limbs (7.5 %) - Pain in the abdominal region (2.5 %)	40	44.9 (7.9)	21 (53 %)	2	2 days	**Subjective**: • Bedtime (Daily sleep diary) • Rise time (Daily sleep diary) • TIB (Daily sleep diary) • TST (Daily sleep diary) • SE (Daily sleep diary) • SOL (Daily sleep diary) • WASO (Daily sleep diary) • SQ (0–5 scale) • Sleep restfulness (0–5 scale) **Objective:** • TIB (Actigraphy) • TST (Actigraphy) • SE (Actigraphy) • SOL (Actigraphy) • WASO (Actigraphy)	**Subjective:** • Pain severity upon awaking (0–5 scale) • Pain severity at bedtime (0–5 scale)

Legend: BPI, Brief Pain Inventory; NRS, Numerical Rating Scales; SE, sleep efficiency; SQ, sleep quality; TIB, time in bed; TST, total sleep time; VAS, Verbal Analogue Scales; WASO, wake after sleep onset.

assessed: study participation, study attrition, prognostic factor measurement, outcome measurement, study confounding, statistical analysis and reporting [27,28].

An overall risk of bias score was obtained by assessing two levels– the previous mentioned domains, which were again divided in sub-items. First, sub-items were scored using a four point scale (yes, no, partial and unsure). When a sub-item was scored 'unsure' due to missing results, it was interpreted as no (i.e. high risk). Additionally, the sub-items 'target population', 'clear population description', 'valid and reliable measurement of prognostic and outcome factors', and 'measurement of confounders' were labelled as important and thereby received a higher weight in the risk of bias assessment. Second, the percentage of low risk (i.e., yes) scored on sub-items was calculated to obtain a domain risk of bias score. For this review, a cut-off point of 70 % was defined. Thus, achieving 75 % or more "yes" responses indicated a low risk of bias, as it ensures that only a small portion (up to 25 %) of the responses are negative ("no"). When the proportion of "yes" responses was between 70 % and 75 %, the risk of bias of the domain was considered moderate. This range allows for up to 30 % "no" responses, suggesting a balanced but slightly concerning level of bias. If "yes" responses comprised 70 % or less of the total, the domain had a high risk of bias. This threshold means that over 30 % of the responses are "no," indicating a significant potential for bias in the domain [29]. An overview of the Risk of Bias assessment was created with Review manager (5.4.1.).

2.5. Data-extraction

Two reviewers executed a standardized data extraction in parallel (A.V.S., J.V.) and their results were compared and adjusted accordingly. The predetermined data-extraction form included publication demographics, population, study design, assessed variables, outcomes (p-values and β-coefficients), and conclusion.

2.6. Data synthesis

Conducting a meta-analysis was determined as not feasible after examining the included studies, due to methodological constraints. More specifically, the studies showed significant heterogeneity in terms of clinical conditions, statistical methods, outcome measures, measurement instruments, amount of daily pain measurements, follow-up periods, and study populations. Consequently, a narrative synthesis was conducted following the Synthesis Without Meta-Analysis (SWiM) [30]. These guidelines were used as an extension to PRISMA and comprise nine categories of information to be included when quantitative synthesis is not achievable [30]. This review adhered to the categories where applicable.

The studies were grouped by direction of prediction: the predictive value of night-time sleep variables on daytime pain intensity and the predictive value of daytime pain intensity on night-time sleep variables. Night-time sleep data were grouped according to five outcome measures: sleep quality (SQ), total sleep time (TST), sleep onset latency (SOL), wake after sleep onset (WASO) and sleep efficiency (SE). Whether night-time sleep predicts daytime pain intensity and vice versa was visualized using tables and examined using a vote-counting method. A sensitivity analysis was performed to assess the robustness of the synthesized results, in which studies considered having a high risk of bias were excluded.

Informal approaches were used to explore heterogeneity in the findings, including the creation of a table delineating methodological characteristics (such as study design) and subpopulations (including pain population, country, and sex).

2.7. Protocol changes

The eligibility criterion describing that 'studies are eligible for inclusion when the majority of the study sample consisted of individuals suffering from the predefined pain problems' was added after the protocol was published. We decided to include these studies as their samples largely represent our intended target population. Furthermore, we only count each study once (e.g. when the study gives different values and sets of adjustments, we only used the unadjusted results for the counting). Additionally, a sensitivity analysis which excluded articles with high risk of bias was carried out to assess the robustness of the results and an additional database (PsycInfo (via ProQuest)) was added to the search.

3. Results

The database search resulted in 1946 unique articles. 108 articles were read in full, resulting in eight included articles. The reference list screening of the included articles ensued three additional included articles. In total, 11 eligible articles were included in this systematic review. Fig. S2 visualizes the selection process.

3.1. Study characteristics

Seven studies were conducted in the United States [31–37], one in Australia [7], one in the United Kingdom [38], one Norway [39], and one in Canada [40] and were published between 1966 and 2022. The studies encompassed a range of CMP populations, including fibromyalgia [31,33,35,38,39], temporomandibular disorder [37], osteoarthritis [36], and chronic low back pain [7,32,34,39,40]. Four studies were considered eligible for inclusion because the majority of the study sample consisted of individuals suffering from the predefined CMP problems [7,35,38,40]. Due to the heterogeneity, data were also grouped by pain population (See Table S2). All but four studies used a significance level of 0.05. One study used a significance level of 0.01 [38] and three studies did not report the significance level [31,34,36]. We used a significance level of 0.05 for all studies. For multiple analysis corrections, Bonferroni correction was used in one study [32], while ten studies did not report any corrections [7,32–40]. Sample sizes ranged from 18 to 220 participants, number of assessment days ranged between 2 and 30 days and a total of 83 associations were examined. Table 1 provides a summary of the study characteristics.

3.2. Risk of Bias

The included articles showed varying risk of bias, with two studies rated as low risk of bias [33,38], two studies as moderate risk of bias [7, 39], and seven studies as high risk of bias [31,34–37,40,41]. Please see Fig. S3 for an overview. The domain 'study attrition' showed moderate to high risk of bias in all included studies. Four studies missed data concerning 'proportion of baseline sample available for analysis' (sub--item study attrition) [31,35,39,40] and 'outcome and prognostic factor information on those lost to follow-up' (sub-item study attrition) was only reported by one study [41].

'Study confounding' was the second most common source of bias, with only four studies performing well [7,35,38,39]. 'Methods used for missing data' (sub-item of study confounding) was not reported by four studies [7,31,37,39]. Four studies showed low risk of bias in the domain of 'prognostic factor measurement' (referring to sleep) [33,34,36,38]. Five studies performed well in the 'outcome measurement' domain (referring to pain) [7,33,34,36,38]. Both domains are crucial factors in this systematic review, as it examines the association between sleep and pain.

3.3. Measures of night-time sleep variables

Sleep was either objectively assessed (e.g. actigraphy, polysomnography) or subjectively using self-reported instruments. Six studies used both methods [7,35,36,38–40], three studies used only self-reported outcomes [31,33,41], and the remaining two studies used only objective measures [34,37]. Ten studies assessed SQ (nine by self-report [7,31,33,35,36,38–41] and one by actigraphy, where SQ was derived using a sleep software package [34]), TST in seven studies (two by self-report and actigraphy [7,40], one by self-report [38], four via actigraphy only [35–37,39]), SOL in six studies (two by self-report and actigraphy [7,40], one by self-report [38], four by actigraphy only [36–39]), WASO in seven studies (three by self-report and actigraphy [7, 35,40], one by self-report [38], three by actigraphy only [7,35,40]), and SE in six studies (3 by self-report and actigraphy [7,38,40], three by actigraphy only [34,36,39]).

3.4. Measures of daytime pain intensity

Numerical Rating Scales (NRSs), Visual Analogue Scales (VASs), and Verbal Rating Scales (VRSs) assessed daytime pain intensity. Six studies used a 0–10 NRS [7,34,36,38,39,42], Six studies used a 0–10 NRS [7,34, 36,38,39,42], with anchors of "worst" to "least pain" [39], "no pain" to "worst possible pain" [7,34], "no pain" to "worst pain imaginable" [37], "no pain at all" to "a lot of pain" [38] and "no pain at all" to "pain as bad as I can imagine" [36]. Besides, one study used a 0–100 NRS with anchors of "no pain" and "pain as bad as it can be" [33]. One study used a 0–10 VAS scale, with anchors of "least intense pain sensation imaginable" and "most intense pain sensation imaginable" [35]. Three studies used different VRSs, such as a 0–9 scale with anchors of "not at all" to "extremely" [32], a 0–6 scale anchored verbally from "none" to "severe"

Table 2
The results on the predictive value of night-time sleep variables on daytime pain intensity.

Reference	SQ	TST	SOL	WASO	SE
Self-reported					
Abeler et al. 2021 [39]	$p = 0.063$; $\beta = 0{,}003$				
Affleck et al. 1996 [31]	**$p < 0.001$; $\beta = -0.05$**				
Alsaadi et al. 2014 [7]	**$p < 0.001$; $\beta = -0{,}026$**	$p = 0.85$; $\beta = 0.000$	**$p = 0.02$; $\beta = 0.009$**	$p = 0.06$; $\beta = 0.006$	**$p = 0.05$; $\beta = -0{,}019$**
Gerhart et al. 2017 [32]	8:50 a.m. **$p < 0.001$; $\beta = -0.42$** 11:50 a.m. **$p < 0.01$; $\beta = -0.15$** 2:50 p.m. $p > 0.05$; $\beta = -0.07$ 5:50 p.m. $p > 0.05$; $\beta = -0.04$ 8:50 p.m. $p > 0.05$; $\beta = -0.09$ Total day **$p < 0.001$; $\beta = -0.16$**				
Kothari et al. 2015 [33]	**$p < 0.01$; $\beta = -0.111$**				
O'Brien et al. 2011 [35]	**$p < 0.01$; $\beta = -0.11$**				
Tang et al. 2012 [38]	Upon awakening **$p < 0.001$** first half of the day **$p < 0.001$** second half of the day **$p < 0.05$**				Upon awakening **$p < 0.001$** first half of the day $p = 0.34$ second half of the day $p = 0.96$
Whibley et al. 2019 [36]	11 a.m. **$p < 0.001$; $\beta = -0.77$** 3 p.m. **$p = 0.007$; $\beta = -0.54$** 7 p.m. **$p = 0.007$; $\beta = -0.55$** Bedtime $p = 0.34$; $\beta = -0.19$ Total day **$p < 0.001$; $\beta = -0.48$**				
Actigraphy/PSG					
Abeler et al. 2021 [39]		$p = 0.11$; $\beta = -0.04$			p = NS; $\beta = -0.004$
Alsaadi et al. 2014 [7]		$p = 0.82$;	$p = 0.42$;	**$p = 0.01$;**	**$p = 0.007$;**
Mun et al. 2022 [37]		$\beta = 0.000$ **$p < 0.05$; $\beta = -0.10$**	$\beta = 0.003$	**$\beta = 0.006$ $p < 0.05$, $\beta = .086$**	**$\beta = -0.022$**
O'Brien et al. 2011 [35]	p = NS				
Tang et al. 2012 [38]					Upon awakening $p = 0.60$ first half of the day $p = 0.07$ second half of the day **$p < 0.01$**
Whibley et al. 2019 [36]		11 a.m. $p = 0.56$; $\beta = -0.20$ 3 p.m. $p = 0.61$; $\beta = 0.18$ 7 p.m. $p = 0.55$; $\beta = -0.21$ Bedtime **$p = 0.02$; $\beta = -0.58$** Total day $p = 0.21$; $\beta = -0.00$	11 a.m. $p = 0.62$; $\beta = -0.04$ 3 p.m. $p = 0.06$; $\beta = 0.16$ 7 p.m. **$p < 0.001$; $\beta = 0.31$** Bedtime **$p = 0.003$; $\beta = 0.25$** Total day $p = 0.46$; $\beta = 0.001$	11 a.m. $p = 0.22$; $\beta = 0.17$ 3 p.m. **$p = 0.006$; $\beta = 0.37$** 7 p.m. **$p < 0.001$; $\beta = 0.50$** Bedtime $p = 0.27$; $\beta = 0.15$ Total day $p = 0.13$; $\beta = 0.004$	11 a.m. $p = 0.90$; $\beta = -0.05$ 3 p.m. $p = 0.32$; $\beta = 0.43$ 7 p.m. $p = 0.94$; $\beta = 0.03$ Bedtime $p = 0.79$; $\beta = 0.12$ Total day $p = 0.40$; $\beta = -0.004$

Legend: Significant associations are indicated in bold; all coefficients are unstandardized and unadjusted beta weights. SE, sleep efficiency; SQ, sleep quality; TST, total sleep time; WASO, wake after sleep onset.

[31] and a 0–5 scale with anchors of "no pain" to "very intense pain" [31,40,41].

The number of daily pain measurements varied among the included studies. Two studies were limited to one measurement per day [39,40]. Three studies measured pain twice per day [7,35,37]. Two studies used three measurements per day: upon awakening, around midday, and before bedtime [31,38]. Two studies measured pain four times per day [32,33]. One study measured pain five times per day [36]. One study performed a pain intensity measurement every 90 min between 8 a.m. and 10 p.m [34]. Moreover, the articles applied different approaches to the characterization of "momentary" pain. The first was a momentary model, intended to capture the current pain (e.g., "What is your pain level right now?" [7]). Seven studies used this momentary model [7,31,34–37,40]. The second approach was a coverage model, intended to capture a short period (e.g., "Rate your level of pain with reference to the first half of the day." [38]). The latter covers a fuller range of within-day momentary experiences [43]. Four studies used the coverage model [33,38,39,41]. A full overview of the sleep and pain outcomes for each study can be found in Table S3.

Table 3
The results on the predictive value of daytime pain intensity on night-time sleep variables.

Reference	SQ	TST	SOL	WASO	SE
Self-reported					
Abeler et al. 2021 [39]	***p* = 0.003; *β* = 2.81**	*p* = 0.079; *β* = 0.07			*p* = NS; *β* = −0.11
Affleck et al. 1996 [31]	***p* < 0.05; *β* = -0.10**				
Alsaadi et al. 2014 [7]	***p* < 0.001; *β* = -0.49**	*p* = 0.83; *β* = - 0.59	***p* = 0.03; *β* = 2.07**	***p* = 0.03; *β* = 2.46**	***p* = 0.02; *β* = -0.78**
O'Brien et al. 2011 [35]	***p* < 0.001; *β* = -0.33**				
Tang et al. 2012 [38]	*p* = 0.09				**p < 0.01**
Wilson et al. 1998 [40]	***p* < 0.001; *β* = -0.92**	***p* < 0.05; *β* = -35.6**	***p* < 0.05; *β* = 10.8**	*p* = NS	*p* = NS
Actigraphy/PSG					
Alsaadi et al. 2014 [7]		*p* = 0.61; *β* = −1.58	*p* = 0.39; *β* = 1.02	***p* = 0.005; *β* = 5.83**	***p* = 0.004; *β* = -1.08**
Liszka-Hackzell 2005 [34]	*p* = NS				*p* = NS
O'Brien et al. 2011 [35]	*p* = NS				
Tang et al. 2012 [38]					*p* = 0.16

Legend: Significant associations are indicated in bold; all coefficients are unstandardized and unadjusted beta weights. SE, sleep efficiency; SQ, sleep quality; TST, total sleep time; WASO, wake after sleep onset.

3.5. Study results

All articles reported a significant relationship between night-time sleep outcomes and daytime pain intensity, except for Liszka-Hackzell et al. (2005), who found no relationship between actigraphy sleep parameters and daytime pain intensity in patients with chronic low back pain [34]. See Tables 2 and 3 for an overview of the study results. Additionally, the outcome of the sensitivity analysis is displayed in Table S7. The sensitivity analysis supports the overall findings, except for the one concerning the predictive value of actigraphy based measures in the second half of the day. This should be interpreted with caution as one of the two studies examining this is considered to have a high risk of bias [36,38].

4. The predictive value of night-time sleep on daytime pain intensity

4.1. Sleep quality

Eight studies investigated the role of self-reported night-time sleep quality (SQ) in predicting daytime pain intensity [7,31,33,35,36,38,39,41]. Seven out of eight studies reported that self-reported SQ predicted next day pain intensity in patients with fibromyalgia and chronic low back pain [7,31–33,35,36,38,39].

Furthermore, three studies indicated that higher self-reported SQ was linked to lower pain intensity upon awakening the next day ($p < 0.001$) and reduced pain intensity during the first half of the day in patients with chronic low back pain ($p < 0.001$) [36,38,41]. However, this association did not hold for pain intensity during the second half of the day ($p < 0.05$) [38].

Self-reported SQ did not predict daytime pain intensity in patients with chronic neck pain ($p = 0.063$) [39].

4.2. Total sleep time

Four studies examined night-time TST predicting daytime pain intensity [7,36,37,39]. While there was no significant evidence supporting the predictive value of self-reported TST in all studies, two studies suggested that TST measured by actigraphy predicted daytime pain intensity close to bedtime in patients with osteoarthritis ($p = 0.02$) and overall pain intensity in patients with temporomandibular disorder ($p < 0.05$) [36,37].

4.3. Sleep onset latency

Two studies analysed the predictive value of night-time SOL on daytime pain intensity [7,36].

One study found that night-time SOL measured by self-report predicted next day pain intensity in patients with chronic low back pain ($p = 0.02$), but this was not found while measured with actigraphy ($p = 0.42$) [7]. While the other study found that night-time SOL measured via actigraphy predicted next day pain intensity around 7 p.m. ($p = 0.007$) and close to bedtime ($p = 0.02$) in patients with osteoarthritis [36].

4.4. Wake after sleep onset

Three studies investigated WASO predicting daytime pain intensity [7,36,37]. There was no evidence supporting the predictive value of self-reported WASO [7]. Contrary, findings for WASO measured by actigraphy did predict next day pain intensity ($p = 0.01$) in patients with chronic low back pain [7] and temporomandibular disorder [37]. And one study found that night-time WASO measured via actigraphy predicted pain intensity around 3 p.m. ($p = 0.006$) and 7 p.m. ($p < 0.001$) in patients with osteoarthritis [36].

4.5. Sleep efficiency

Four studies examined the predictive value of night-time SE on next daytime pain intensity, providing evidence for self-reported SE predicting daytime pain intensity and mixed findings for SE measured by actigraphy predicting daytime pain intensity [7,36,38,39].

Two studies found that both self-reported and actigraphy-derived SE predicted next daytime pain intensity in patients with chronic low back pain ($p = 0.05$ and $p = 0.007$, respectively) [7,38].

Interestingly, one of these studies self-reported SE only predicted next day pain intensity upon awakening ($p < 0.001$) [38]. However, night-time SE measured via actigraphy only predicted next day pain intensity in the second half of the day ($p < 0.01$) [38].

SE measured via actigraphy did not predict overall next day pain intensity in patients with chronic neck pain ($p = 0.40$) [39] and osteoarthritis [36].

5. The predictive value of daytime pain intensity on next night sleep

5.1. Sleep quality

Seven studies examined daytime pain intensity predicting next night SQ, resulting in mixed findings for daytime pain intensity predicting both self-reported SQ [7,31,34,35,38–40].

Five studies found that daytime pain intensity predicted self-reported SQ in patients with chronic neck pain, chronic low back pain and fibromyalgia ($p < 0.05$) [7,31,35,39,40]. Contrary, Two studies found that daytime pain intensity did not predict self-reported SQ ($p = 0.09$) [38] nor SQ measured via actigraphy patients with chronic low back pain [35].

5.2. Total sleep time

Three studies examined daytime pain intensity predicting next night TST, providing mixed findings for daytime pain intensity predicting both self-reported and actigraphy-derived TST [7,39,40].

One study found that daytime pain intensity did not predict self-reported or actigraphy-derived TST in patients with chronic back pain ($p = 0.83$ and $p = 0.61$, respectively) [7]. One study found that daytime pain intensity predicted self-reported TST in patients with chronic back pain ($p < 0.05$) [40]. Another study found that daytime pain intensity did not predict self-reported TST ($p = 0.079$) in patients with chronic neck pain [39].

5.3. Sleep onset latency

Two studies examined the predictive value of daytime pain intensity on next night SOL, resulting in evidence for daytime pain intensity predicting self-reported SOL, but not actigraphy-derived SOL [7,40].

Two studies found that daytime pain intensity predicted self-reported SOL in patients with chronic back pain ($p < 0.05$) [7,40]. However, the study of Alsaadi et al. (2014) did not find this relationship with SOL measured via actigraphy ($p = 0.39$) [7].

5.4. Wake after sleep onset

Two studies examined daytime pain intensity predicting next night WASO in patients with chronic back pain [7,40]. These studies provided mixed findings for daytime pain intensity predicting both self-reported and actigraphy-derived WASO.

Alsaadi et al. (2014) found that daytime pain intensity predicted both self-reported and acrigraphy-derived WASO ($p = 0.03$; $p = 0.005$) [7]. The other study did not find these results for self-reported WASO [40].

5.5. Sleep efficiency

Five studies examined daytime pain intensity predicting next night SE, providing mixed findings for daytime pain intensity predicting both self-reported and actigraphy-derived SE [7,34,38–40].

Two studies found that daytime pain intensity predicted self-reported SE in patients with chronic back pain ($p = 0.02$) [7,38]. However, the study of Alsaadi et al. (2014) found that daytime pain intensity predicted SE measured via actigraphy ($p = 0.004$), while Tang et al. (2012) found that daytime pain intensity did not predict SE measured via actigraphy ($p = 0.16$) [7,38]. Two other studies found that daytime pain intensity did not predict self-reported SE in patients with chronic back pain and chronic neck pain [39,40]. Additionally, one study found that daytime pain intensity did not predict SE measured via actigraphy in patients with chronic back pain [34].

6. Discussion

This study is the first to provide a systematic overview of the evidence on the day-to-day associations between various sleep outcomes and pain intensity in adults with CMP. Eleven articles were included and a total of 83 associations were examined.

There seems a bidirectional relationship between pain intensity and sleep in a CMP population [7,31–33,35–37,39,40,44], yet our results indicate that self-reported SQ and SE might be a more consistent predictor of next day pain intensity than vice versa [7,31,33,36–39,41], even after removing studies with a high risk of bias [7,33,38,39]. The diminishing of predictive effect in the afternoon might be due to additional variables associated with daytime pain intensity, introduced during the day [38,45]. Our results support the idea of a recuperative nature of an appropriate self-reported SQ and SE in the morning, with potentially decreased attention to pain and more positive affect after a night of good sleep [38,46]. Thus, while good sleep quality may be an important indicator for less intense pain, it seems insufficient to predict lower pain intensity throughout the day [32].

Interestingly, other actigraphy based measures such as SE, TST, SOL and WASO show an opposite trend, with a predictive value mainly present in the second half of the day [36,38]. This is consistent with previous research, showing that sleep fragmentation (which relates to lower values on these variables) increases spontaneous pain reports [38]. Nevertheless, this should be interpreted with care as one of the two studies examining this was considered to have a high risk of bias.

Additionally, the association between sleep variables and pain intensity varies even when examined in the same CMP population. Some studies yielded *p*-values close to 0.05 [7,36,38,39], and five out of 11 studies had sample sizes of 56 or less [31,34,35,39,40]. Furthermore, consistent issues with study attrition limits these studies' ability to detect anything beyond moderate to large associations. Additionally, some studies fail to provide precise estimates of effect sizes, further complicating result interpretation [32,34,35,39,40].

Several methodological aspects should be considered as well when interpreting the results of this review. First, only three studies specifically included individuals with CMP and comorbid insomnia complaints. This might underestimate the day-to-day influence of sleep outcomes on pain intensity and vice versa. Therefore, future studies should include samples of individuals with CMP and comorbid sleep disorders. Moreover, comparative studies should investigate CMP alongside various sleep disorders to reveal associations and delve into the nuanced differences among different types of sleep disturbances, enhancing our understanding of their interplay with CMP. Yet, it seems equally crucial to examine CMP in isolation from sleep disorders to gain a comprehensive perspective, as specific chronic pain populations might have an influence on the daily association between sleep variables and pain intensity, explaining the differences seen in the results.

Second, the association between night-time sleep variables and next morning pain intensity is mainly true when sleep is assessed by self-report (which assesses the perception of sleep quality or sleep duration) instead of actigraphy, even when studies considered with a high risk of bias were excluded. This result emphasizes the impact of an individual's beliefs in shaping his/her experienced sleep quality and related pain intensity, which objective assessments may not capture such as feelings of restfulness upon awakening [47]. Furthermore, objective measures might influence the subjective sleep quality of a participant, while subjective measurements of sleep and pain are important in the clinical setting [47]. Additionally, while actigraphy estimates sleep parameters based on biometric data including arm movement [34], they are known to overestimate sleep and underestimate wakefulness [36]. Thus, the less frequently observed association between night-time objective sleep quality and next day (morning) pain intensity might be due to the suboptimal assessment of sleep/wake behavior by actigraphy. Polysomnography, which is the goldstandard for sleep assessment, might be more appropriate to reliably assess the day-to-day sleep-pain association. Wearable sleep EEG devices appropriate for multiple nights recording are available, and provide opportunities for further research, also including data on sleep architecture [48].

Third, apart from the assessment of sleep, it is also important to consider the assessment of pain via EMA. Today, there are no established standards for the development of effective EMA pain questions, leading to variable approaches used to characterize "momentary" pain, variable types of pain rating scales, and variable numbers of pain questions per day and their timing within the day [43,49]. In this review, both a momentary model (intending to capture the current pain, e.g., "What is your pain level right now?") [35] and a coverage model (intending to capture a short period, e.g., "Rate your level of pain with reference to the first half of the day") were used to characterize pain levels [38]. Since the latter covers a fuller range of within-day momentary experiences, it might be more prone to pain beliefs than the momentary approach,

Practice points

1. Untreated sleep problems in individuals with CMP might initiate a vicious cycle of sleep and pain problems, which forms a barrier for chronic pain management.
2. Patients experiencing heightened pain levels primarily in the first half of the day may derive benefits from sleep interventions to improve their subjective sleep quality and efficiency.
3. Routine screening for sleep disturbances and sleep disorders in individuals with CMP should become standard practice.
4. Educate patients on sleep-pain relationship and habits.

leading to different pain intensity results [43].

Importantly, it is impossible to ask questions about pain intensity without increasing someone's focus on bodily symptoms and attention to pain (which in turn relates to pain intensity). Therefore, the number of 'pain intensity' questions per day can in itself influence the outcome of a pain intensity measure, and as such the results on the association between day-to-day sleep quality and pain intensity. This phenomenon is called assessment reactivity and forms an issue in EMA studies [21,50]. Moreover, participants might also alter their daily routine because of the frequent assessments of pain intensity [35,54]. Given that the number of pain intensity questions per day varied between one and eight in the included studies, this might have influenced the results. In future, the use of self-report outcomes in a high-frequency way, as is typical for the EMA methodology, that may increase the attention to (negative) bodily signals, like pain, should always be well-considered in context of the research question. Indeed, the increased attention to pain might increase the threat-value of pain and related distress. Lastly, as already mentioned, the timing of the pain intensity question influenced the day-to-day association between sleep and pain intensity [36,38,41]. The finding that the predictive value of sleep variables on pain intensity diminishes depending on the time of the day confirms the importance of timing in measurement [36,38].

This overview of factors influencing the day-to-day relationship between sleep and pain intensity underscores the necessity for standardized and normed EMA measurement instruments in the field of sleep and pain research [43]. Consistency in the application of measurement methods and outcomes will facilitate comparisons across studies, thereby advancing the field. Furthermore, ensuring adherence to time-based EMA notifications through mobile devices seems straightforward [51]. However, the momentary aspect of EMA may be compromised if not all survey items are completed. Despite concerns regarding the impact of EMA burden on adherence, it remains unclear which EMA factors specifically affect adherence. Future research should focus on identifying and understanding barriers to participation and adherence [52].

6.1. Strengths and limitations

Apart from limitations of the included studies in this review, limitations of the review itself should also be considered. Although a systematic search was performed, and reference lists were screened, it is possible that relevant papers were missed. Further, we could not perform a meta-analysis due to the heterogeneity of the included studies, their statistical analysis, and reporting. However, this review also has several strengths. A pre-determined and registered protocol was followed, and independent and blinded reviewers were used for the selection process, data extraction, and Risk of Bias assessment. Lastly, findings were reported in line with the PRISMA and SWiM guidelines [25,30].

6.2. Clinical implications

The findings of this systematic review have several clinical implications. Given the day-to-day bidirectional relationship between self-reported *sleep quality, sleep efficiency, total sleep time, wake after sleep onset,* and *sleep onset* and pain intensity, untreated sleep problems in individuals with CMP might initiate a vicious cycle of sleep and pain problems, which additionally forms a barrier for chronic pain management [53]. It also shows that a biopsychosocial approach which includes sleep in general may benefit patients with CMP. Therefore, routine screening for sleep disturbances and sleep disorders in patients with CMP should become standard practice. Patients experiencing heightened pain levels may benefit from sleep interventions aimed at enhancing their subjective sleep quality and efficiency. However, in case of suspected sleep disorder, patients should be referred to a sleep clinic or a certified sleep expert.

In this context, Ecological Momentary Interventions and Just-In-Time Adaptive Interventions, using real-time information from EMA, offer opportunities for more personalized and timely care [54]. Indeed, it provides opportunities for immediate personalized treatment, to forecast periods of susceptibility and subsequent timely interventions [54]. Although these approaches are in the early stages of development, they show considerable potential for advancing pain management and research in future [54]. Opposingly, it is important to temper expectations regarding immediate pain reduction throughout the entire day following sleep improvement. Encouraging patients to refrain from self-criticism for daily fluctuations in pain and sleep, given their natural variability, is essential.

Research agenda

1. Develop standardized and normed EMA measurement instruments in the field of sleep and pain research.
2. Include both subjective and objective outcomes in research.
3. Comparative studies should explore associations between CMP and various sleep disorders to understand their interplay, while also examining CMP in isolation to grasp specific chronic pain populations' influence on the daily relationship between sleep variables and pain intensity.
4. Consider the potential impact of high-frequency self-reporting in EMA methodologies, particularly regarding the assessment reactivity phenomenon.

7. Conclusions

There is a bidirectional day-to-day relationship between pain intensity and various sleep outcomes in CMP populations, as established by examining 83 associations. The association between self-reported night-time sleep and next-day pain intensity is mainly present in the first half of the day and fades as the day progresses. These results can have important implications for clinical practice, where a vicious cycle can be broken by targeting both sleep and pain in the treatment of individuals with CMP. Furthermore, this review underscores the necessity for standardized and normed EMA measurement instruments in the field of sleep and pain research.

Declaration of AI and AI-assisted technologies in the writing process

During the preparation of this work the author(s) used ChatGPT 3.5 in order to improve language and readability, recognizing the potential influence of English not being their native language. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

Declaration of competing interest

The authors declare no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.smrv.2024.102013.

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