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CLINICAL REVIEW

Psoriasis and sleep disorders: A systematic review

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SUMMARY

Psoriasis is an immune-mediated chronic inflammatory disorder which manifests as dermatologic lesions, and psoriatic arthritis (PsA) in about 30% of cases. Psoriasis is associated with multiple comorbidities including metabolic syndrome, hypertension, diabetes, cardiovascular events, obesity and psychiatric disorders, which can all affect the course of sleep disorders. A systematic review of the literature on the relationship between psoriasis, PsA, and formal sleep disorders identified 33 studies. There is an increased prevalence of obstructive sleep apnea (OSA) with 36%–81.8% prevalence in psoriasis versus 2%–4% in the general population. There was also an increase in the prevalence of restless legs syndrome of 15.1%–18% in psoriasis versus 5%–10% in European and North American samples. The wide variety of insomnia criteria used in studies resulted in an insomnia prevalence of 5.9%–44.8% in psoriasis, which is insufficient to show an elevated prevalence when the general population has a 10% prevalence of chronic insomnia and 30–35% prevalence of transient insomnia. There is evidence that symptoms of insomnia in psoriasis are directly mediated by pruritus and pain. Treatments that decrease the cutaneous symptoms in psoriasis were successful in mitigating insomnia, but did not show improvements in OSA where the relationship with psoriasis is multifactorial.

Introduction

Psoriasis is a chronic inflammatory disease that is associated with both genetic and environmental factors and affects about 2%–4% of the population worldwide [1,2]. In about 80% of cases, psoriasis presents as symmetrical, sharply demarcated, erythematous, dry, scaling, pruritic plaques affecting the skin. Psoriasis has a predilection for presenting on the scalp, extensor surfaces of the limbs, hands and feet, sacral and genital regions, but the total body surface area affected can vary. Psoriatic plaques may persist for months to years, and periods of complete remission are possible. Psoriasis is often disfiguring and painful and pruritus or itching is one of the most bothersome symptoms of psoriasis. Painful nail changes are also seen in up to 40% of patients, and about 30% of psoriasis patients have psoriatic arthritis (PsA) that commonly affects the distal interphalangeal joints. There are also less common presentations of psoriasis. Erythrodermic psoriasis is characterized by generalized erythema and scaling affecting over 90% of the body surface area [1,2]. Guttate psoriasis, most commonly seen in children, presents as numerous small disseminated papules and plaques seen in <2% of patients and is often preceded by a streptococcal throat infection. Pustular psoriasis can present as generalized pustules and pustulosis of the palms and soles and usually follows an infection or medications such as lithium.

Psoriasis has been associated with disturbed sleep, but its association with formal sleep disorders has not yet been fully investigated [3]. Psoriasis may have a direct effect on the development of sleep disorders due to the cutaneous symptoms of the disorder. The skin acts as a primary circadian mediator of core body temperature (CBT), and a decrease in CBT in the late evening is an important mechanism for sleep initiation. The CBT decreases due to decreased metabolic heat generation, increased cutaneous blood flow and vascular dilation distally, leading to increased distal-to-proximal gradient in skin temperature, dissipation of heat and trans-epidermal water loss (TEWL). Psoriasis has been associated with problems with thermoregulation and reduced ability to dissipate heat, which may disrupt sleep initiation [4]. Pruritus, a primary contributor to sleep disturbance, is also regulated by circadian mechanisms. The threshold for pruritus is lowered in the evening due to complex circadian-

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Abbreviations

AD	atopic dermatitis
ADA	adalimumab
AHI	apnea hypopnea index
CASPAR	classification criteria for psoriatic arthritis
CBT	core body temperature
CENTRAL	Cochrane central register of controlled trials
CPAP	continuous positive airway pressure
ESS	Epworth sleepiness scale
ETN	etanercept
FOSQ	functional outcomes of sleep questionnaire
ICSD	International classification of sleep disorders
IL	interleukin
INF-γ	interferon-γ
MSLT	multiple sleep latency test
NHS	nurses' health study
OR	odds ratio
OSA	obstructive sleep apnea
PASI	psoriasis area severity index
PLMD	periodic limb movement disorder
PsA	psoriatic arthritis
PSG	polysomnography
PSQI	Pittsburgh sleep quality index
RLS	restless legs syndrome
RoB	risk of bias
TEWL	trans-epidermal water loss
Th	T-helper cell
TNF-α	tumor necrosis factor α

mediated factors such as lower cortisol levels, decreased epidermal barrier function, and increased distal-to-proximal gradient in skin temperature [5]. Pruritus in psoriasis typically manifests or exacerbates mainly in the evening and worsens nocturnally, often disrupting sleep [5]. These skin-related circadian factors can therefore affect sleep both as a result of decrease in the threshold for pruritus and also by affecting thermoregulation and sleep onset.

Psoriasis may be indirectly linked to sleep disorders through its association with systemic inflammatory disorders. Individuals with psoriasis have a higher prevalence of metabolic syndrome, hypertension, diabetes, obesity, tobacco smoking, depression, anxiety and suicidality (Table 1) [6–10]. They also have a higher incidence of diabetes, obesity, myocardial infarction, stroke and cardiovascular mortality (Table 1) [6,11,12]. Psoriasis is primarily mediated by interferon-γ (INF-γ), interleukin (IL)-23, IL-17, tumor necrosis factor α (TNF-α), as well as increases in T-helper cells (Th1 and Th17). The inflammatory state results in an increase in IL-6, increased antigen presenting dendritic cells, and upregulation of adhesion molecules in the vasculature [13,14]. These pro-inflammatory molecules are also markers of the systemic inflammatory disorders that are commonly co-morbid with psoriasis [14]. Diabetes and hypertension have shown associations with insomnia [15,16], while all of the above comorbidities (Table 1) have associations with obstructive sleep apnea (OSA) [17–21].

A lack of evaluation of the sleep of psoriasis patients by formal criteria has left doubt that there is a consistent association between psoriasis and sleep disorders [22]. Psoriasis is likely to disrupt sleep directly via nocturnal itch and pain resulting in disorders of movement, circadian rhythmicity, or interrupted sleep. The inflammatory pathogenesis of psoriasis and its contribution to the development of systemic diseases like depressive disease, hypertension, adverse cardiac events, diabetes, metabolic syndrome and obesity are likely to indirectly give rise to sleep-disordered breathing [6–8,11,12,23]. The heightened pro-inflammatory state in conditions such as OSA and insomnia could in turn lead to exacerbations of psoriasis. Here, we conduct a systematic review to determine if psoriasis is associated with the sleep disorders meeting some of the major diagnostic criteria of the International classification of sleep disorders (ICSD) [24,25].

Objectives

To examine the relationship between psoriasis, PsA, and sleep disorders.

Methods

This review was conducted in accordance with the PRISMA guideline for systematic reviews [26].

Eligibility criteria

Types of studies

This review included studies of the prevalence and characteristics of sleep disorders in psoriasis and PsA (case–control, cohort and cross-sectional observational studies were included) and intervention studies where a sleep disorder was one of the outcome measures (case reports, single-assignment trials and randomized trials using both parallel-group and crossover design were included). Studies were excluded if they were non-English language studies, review articles, or animal studies.

Types of participants

Participants in this study must have a clinical diagnosis of psoriasis or PsA. Participants from both pediatric (<18 y) and adult (≥18 y) populations were included.

Types of interventions

This review considered drugs, devices like continuous positive airway pressure (CPAP) and lifestyle interventions for the treatment of co-morbid sleep and dermatologic disorders.

Types of outcome measures

The outcome measures considered included the prevalence and symptoms of sleep disorders (See Table 2 for criteria for definition of a sleep disorder) in observational studies. In intervention studies, the outcome measures included a sleep disorder (Table 2) as one of the indices of treatment efficacy, compliance, tolerability, or treatment emergent adverse events.

Table 1
Association between psoriasis and systemic disease.

	Prevalence OR (95%CI)	Incidence RR or HR (95%CI)
Metabolic syndrome [8]	2.26 (1.70–3.01)	–
Hypertension [7]	1.58 (1.42–1.76)	HR: 1.09 (1.05–1.14) HR: 1.17 (1.06–1.30)
Diabetes [6]	1.59 (1.38–1.83)	RR: 1.27 (1.16–1.40)
Obesity [11]	1.66 (1.46–1.89)	HR: 1.18 (1.14–1.23)
Cardiovascular mortality [12]	–	RR: 1.39 (1.11–1.74)
Depression [9]	2.40 (1.67–3.47)	–
Anxiety [9]	2.18 (1.68–2.82)	–
Suicidality [9]	1.94 (1.33–2.82)	–

		Mild psoriasis	Severe psoriasis
Myocardial Infarction [12]	–	RR: 1.29 (1.02–1.63)	RR: 1.70 (1.32–2.18)
Stroke [12]	–	RR: 1.12 (1.08–1.16)	RR: 1.56 (1.32–1.84)

CI, confidence interval; HR, hazard ratio; OR, odds ratio; RR, risk ratio.

Information and sources

Electronic searches

The Cochrane central register of controlled trials (CENTRAL) in The Cochrane Library;
MEDLINE via OVID (from 1946);
EMBASE via OVID (from 1947);
PsycINFO via OVID (from 1806) and
The full search strategy can be found in Table S1.

Study selection

Studies were screened for the evaluation of psoriasis or PsA and a sleep disorder. The full text articles were evaluated for inclusion by two independent reviewers (FS and KK or MAG), and disagreements were resolved by discussion to reach a consensus. In cases where participants were evaluated in more than one report, the articles are grouped under a single study identifier.

Data extraction

Data extraction was performed using two data collection templates, one for prevalence studies and one for intervention studies. Data extraction was performed by one reviewer and verified by a second reviewer. The data template was piloted before use. The prevalence template included: study identifier, study design, number of participants per group, baseline characteristics, sleep disorder criteria, prevalence, and outcome measures. The intervention template included: study identifier, study design, sleep disorder criteria, number of participants per group, baseline characteristics, intervention, treatment regimen and duration, treatment success and failure, efficacy, safety, morbidity and mortality, tolerability, compliance, number of patients lost to follow-up, and the duration of follow-up. Disagreements were resolved with discussion, or if agreement could not be reached, a third author assisted in the resolution of any discrepancies. The data were entered by a single author (FS) and verified by two reviewers (FS and MAG).

Risk of bias

The risk of bias (RoB) for observational studies was evaluated using a modified version of the Agency for Healthcare Research and Quality scale [27]. Questions 4, 10 and 11 were omitted as they were not relevant to the included studies. Intervention studies were evaluated using the Cochrane RoB scale [28]. Each included study was evaluated for RoB in duplicate, by FS and KK or MAG. Disagreements were resolved with discussion.

Results

Included studies

The search, finalized on January 13, 2015, identified 687 articles related to psoriasis and sleep disorders. Of these, 35 records including 33 studies discussed co-morbid sleep and dermatological disorders (Fig. 1; Tables S2) [29–63]. The characteristics of the included studies can be found in Table 3.

Excluded studies

There were 67 excluded full text records (Table S3). The most common reason for exclusion was lack of a sleep disorder, followed by an ineligible skin disorder. The reason for exclusion for each record can be found in Table S3.

Study design

The observational studies included nine case control studies [31,33,34,36,42,46,51,52,59], twelve cross-sectional studies [29,30,32,35,38,40,43,47–49,60,63], five nested case–control studies [41,45,57,61,62], one non-comparative study [50], and one prospective cohort study [37]. The intervention studies included one interrupted time series with comparator group [53,54] and four randomized controlled trials (RCTs) [39,44,55,56,58].

Insomnia

Eleven observational studies were identified concerning insomnia in patients with psoriasis (Table 4) [33,35,36,40,43,46,49,51,52,61,63]. Seven studies had a high RoB [33,35–37,46,49,52], four studies had moderate RoB [40,43,51,63] and one study had a low RoB [61]. The prevalence of insomnia was 5.9%–44.8% in psoriasis and 15.1% in PsA [33,35,46]. Psoriasis patients with self-reported psoriasis had an odds ratio (OR) of 1.44 (95% CI: 1.19–1.75) for developing insomnia [51], whereas patients with physician-diagnosed chronic plaque psoriasis had an OR of 4.3 (95% CI: 1.7, 11.2) [51]. Sleep disturbance in psoriasis and PsA was most commonly linked to poor quality of life (QoL), increased depression and anxiety severity, pruritus, and pain [35,36,43,46,49,63]. Only one study did not find a correlation between psoriasis severity and

Table 2
Sleep disorder criteria [24,25].

Sleep disorder	Criteria
Insomnia	A. The patient reports, or the patient's parent or caregiver observes, one or more of the following: 1. Difficulty initiating sleep. 2. Difficulty maintaining sleep. 3. Waking up earlier than desired. 4. Resistance to going to bed on appropriate schedule. 5. Difficulty sleeping without parent or caregiver intervention. B. The patient reports, or the patient's parent or caregiver observes, one or more of the following related to the nighttime sleep difficulty: 1. Fatigue/malaise. 2. Attention, concentration, or memory impairment. 3. Impaired social, family, occupational, or academic performance. 4. Mood disturbance/irritability. 5. Daytime sleepiness. 6. Behavioral problems (e.g., hyperactivity, impulsivity, aggression). 7. Reduced motivation/energy/initiative. 8. Proneness for errors/accidents. 9. Concerns about or dissatisfaction with sleep. This included participants evaluated using scales that assessed A and B criteria like the PSQI and the DLQI.
Narcolepsy	A. The patient has daily periods of irrepressible need to sleep or daytime lapses into sleep occurring for at least three months. B. A mean sleep latency of ≤8 min and two or more SOREMPs are found on a MSLT performed according to standard techniques. A SOREMP (within 15 min of sleep onset) on the preceding nocturnal PSG may replace one of the SOREMPs on the MSLT.
Obstructive sleep apnea	**(A and B) or C satisfy the criteria** A. The presence of one or more of the following: 1. The patient complains of sleepiness, nonrestorative sleep, fatigue, or insomnia symptoms. 2. The patient wakes with breath holding, gasping, or choking. 3. The bed partner or other observer reports habitual snoring, breathing interruptions, or both during the patient's sleep. 4. The patient has been diagnosed with hypertension, a mood disorder, cognitive dysfunction, coronary artery disease, stroke, congestive heart failure, atrial fibrillation, or type 2 diabetes mellitus. B. PSG or OCST demonstrates: 1. Five or more predominantly obstructive respiratory events (obstructive and mixed apneas, hypopneas, or RERAs) per hour of sleep during a PSG or per hour of monitoring (OCST). **OR** C. PSG or OCST demonstrates: 1. Fifteen or more predominantly obstructive respiratory events (apneas, hypopneas, or RERAs) per hour of sleep during a PSG or per hour of monitoring (OCST).
Periodic limb movement disorder	A. PSG demonstrates PLMS, as defined in the most recent version of the AASM manual for the scoring of sleep and associated events. B. The frequency is >5/h in children or >15/h in adults. C. The PLMS cause clinically significant sleep disturbance or impairment in mental, physical, social, occupational, educational, behavioral, or other important areas of functioning. D. The PLMS and the symptoms are not better explained by another current sleep disorder, medical or neurological disorder, or mental disorder (e.g., PLMS occurring with apneas or hypopneas should not be scored).
Restless legs syndrome	A. An urge to move the legs, usually accompanied by or thought to be caused by uncomfortable and unpleasant sensations in the legs. These symptoms must: 1. Begin or worsen during periods of rest or inactivity such as lying down or sitting; 2. Be partially or totally relieved by movement, such as walking or stretching, at least as long as the activity continues; and 3. Occur exclusively or predominantly in the evening or night rather than during the day. B. The above features are not solely accounted for as symptoms of another medical or a behavioral condition (e.g., leg cramps, positional discomfort, myalgia, venous stasis, leg edema, arthritis, habitual foot tapping). C. The symptoms of RLS cause concern, distress, sleep disturbance, or impairment in mental, physical, social, occupational, educational, behavioral, or other important areas of functioning.
Shift-work disorder	A. There is a report of insomnia and/or excessive sleepiness, accompanied by a reduction of total sleep time, which is associated with a recurring work schedule that overlaps the usual time for sleep. B. The symptoms have been present and associated with the shift work schedule for at least three months. C. The sleep and/or wake disturbance are not better explained by another current sleep disorder, medical or neurological disorder, mental disorder, medication use, poor sleep hygiene, or substance use disorder.

AASM, American academy of sleep medicine; DLQI, dermatology life quality index; MSLT, multiple sleep latency test; OCST, out of center sleep testing; PLMS, periodic limb movement syndrome; PSG, polysomnography; PSQI, Pittsburg sleep quality index; REM, rapid eye movement; RERA, respiratory effort related arousal; RLS, restless legs syndrome; SOREMP, sleep onset REM period.

sleep disturbance as measured by the Pittsburgh sleep quality index (PSQI) [52].

Four intervention studies on the treatment of insomnia in psoriasis were identified (Table 7) [39,53–56,58]. Two studies had a high RoB [39,55,56], one had a moderate RoB [53,54] and one had a low RoB [58]. Three studies examined the effects of etanercept (ETN), two examined different dose regimens for ETN [39,55,56] and one was a placebo-controlled trial [58]. In all three trials ETN improved sleep domains, QoL, depression and fatigue scores. In the placebo-controlled trial, improvements in joint pain were associated with decreased fatigue [58]. A single study examined the impact of adalimumab (ADA) on sleep in psoriasis [53,54]. After 16 wk, Strober et al. found that ADA improved PSQI scores 15% from baseline which was partially explained by improvements in psoriasis area severity index (PASI) score. ADA also improved QoL, pain, and work productivity.

Obstructive sleep apnea

Eleven observational studies were identified concerning OSA in patients with psoriasis (Table 5) [30,31,34,38,42,47,48,50,57,60,62]. Two studies were at a high RoB [30,42], five

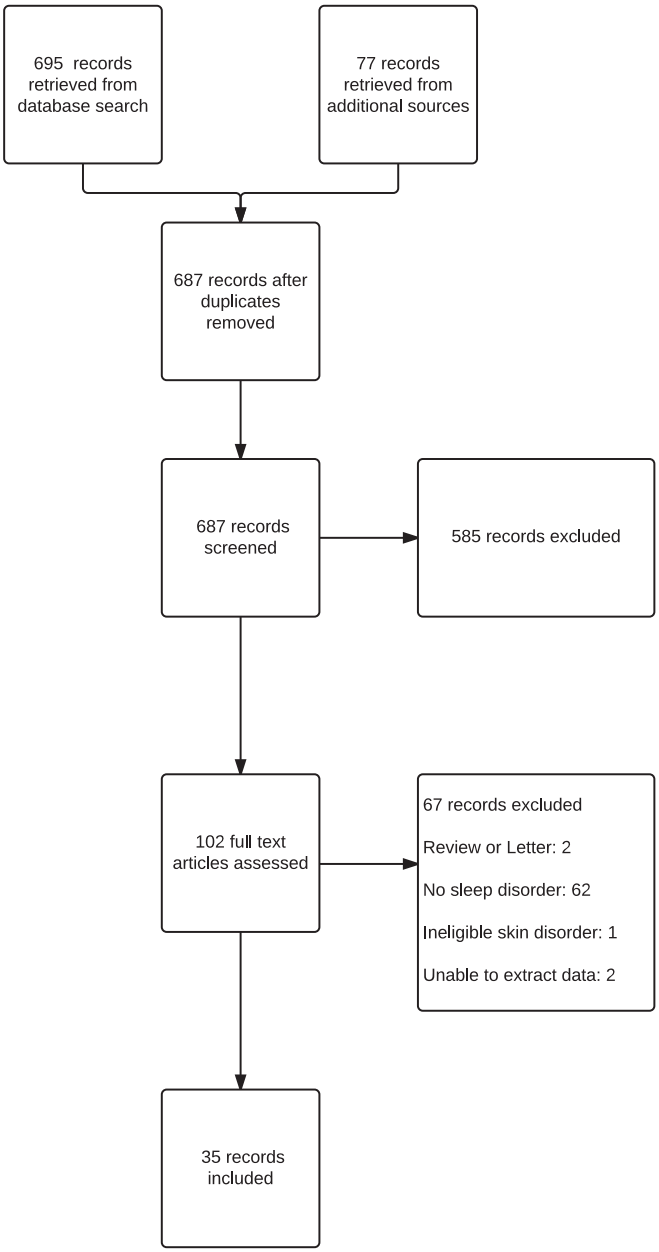

Fig. 1. Systematic review flow diagram.

studies had a moderate RoB [31,34,38,48,60], and three had a low RoB [47,57,62]. The prevalence of OSA in psoriasis ranged from 36 to 81.8% [30,31,38,48,60]. Tsai et al. reported a risk ratio of 3.86 (95% CI: 2.26–6.71) of developing OSA in patients with psoriasis [57]. Yang et al. reported a hazard ratio of 2.30 (95% CI: 1.13–4.69) for the development of psoriasis in patients with OSA [62]. Two studies reported a higher mean apnea hypopnea index (AHI) in patients with psoriasis than controls [34,42]. The prevalence of OSA in PsA was reported in a single study with a prevalence of 100% [60].

One intervention study with a low RoB was identified for OSA (Table 7) [44]. Maari et al. examined the effect of TNF-α antagonist ADA on OSA in psoriasis. At follow-up, there was no difference in AHI, Epworth sleepiness scale (ESS) score or functional outcomes of sleep questionnaire (FOSQ) score between patients treated with ADA or placebo. Patients treated with ADA had a greater reduction in PASI score than those treated with placebo.

Restless legs syndrome (RLS) and periodic limb movement disorder (PLMD)

Four observational studies were identified concerning RLS or PLMD in psoriasis (Table 6) [29,32,33,59]. All four studies had a high RoB. The rate of RLS in patients with psoriasis was reported as higher than the general population. In 2009, Duffin et al. reported that the rate of RLS was higher in patients with PsA (15.1%) and psoriasis (6.4%) than controls (4.1%) [33]. Cicek et al. reported a prevalence of RLS of 15.1%, which was lower than in patients with atopic dermatitis (AD) (40.8%), but higher than in controls (10.8%) [32]. Bilgic et al. reported a similar prevalence of RLS of 15.9% [29]. A single study reported on symptoms of PLMD in psoriasis (Table 2). Wong et al. conducted the sole study on PLMD and found that psoriasis patients had an elevated periodic limb movement index when compared to controls (44.5 $\pm$ 48.7 vs. 22.2 $\pm$ 27.1) [59].

Narcolepsy and shift work disorder

A single study with a moderate RoB was identified for narcolepsy in psoriasis [45]. Martinez-Orozco et al. examined the incidence of inflammatory diseases in patients with narcolepsy diagnosed by polysomnography (PSG) and a multiple sleep latency test (MSLT). The rate of psoriasis in narcolepsy was 1.3% and the rate of AD was 1.9% [45].

A single study with a low RoB was identified concerning psoriasis in shift work disorder [41]. Li et al. assessed the incidence of psoriasis in nurses who worked night shifts over a ten year period using the Nurses' Health Study I and II and a follow-up psoriasis survey. The multivariate-adjusted hazard ratio for nightshift workers developing psoriasis in 10 y was 1.23 with a 95% confidence interval of 1.03–1.47.

Discussion

Summary of evidence

The primary goal of this systematic review was to assess the relationship between psoriatic conditions and sleep disorders. The prevalence of sleep disorders in individuals with psoriasis is one of the primary components of this relationship. There was a clear elevation in the prevalence of OSA (36%–81.8%) in psoriasis, given that the prevalence of OSA in the general population is 2% for women and 4% for men using the diagnostic criteria of an apnea-hypopnea index (AHI) $\geq$5, with a complaint of excessive daytime sleepiness [25]. The prevalence of OSA was also higher than the general population rate of 9% for women and 24% for men when only an AHI $\geq$ 5 criterion is used to diagnose OSA [25]. The associated risk and hazard ratios for the development of OSA in psoriasis and vice versa were also elevated [57,62]. There was also an increased prevalence of RLS (15.1–18%) in patients with psoriasis compared to the 5–10% prevalence reported in European and North American populations [25,29,32,33]. The prevalence of insomnia ranged from 5.9%–44.8% in psoriasis which is inconclusive evidence given that the prevalence of chronic insomnia and transient insomnia are 10% and 30–35%, respectively [33,35,46]. The likelihood of developing insomnia in psoriasis ranged from 1.44 to 4.3 (OR) [25,51,61]. There were insufficient prevalence data for other sleep disorders.

The second factor in determining the relationship between psoriatic conditions and sleep disorders is determining if treatment of psoriasis results in decreased sleep disturbance. Both ETN and ADA therapy improved the dermatologic lesions in psoriasis.

Table 3
Characteristics of included studies.

Study identifier	Study design	Psoriasis characteristics	Controls	n	Age (Mean ± SD)	M/F	Outcome measures	RoB
Bilgic 2013 [29]	Cross-sectional	NR	–	44	46.43 ± 14.62	21/23	Prevalence of RLS, blood tests	High
Bissonnette 2012 [30]	Cross-sectional	Chronic plaque psoriasis >5% BSA and AHI >15	–	20	NR	NR	PSG and PLMI	High
Buslau 1999 [31]	Case-control	Ps and two symptoms of OSA	–	Ps: 25 CB: 19	NR	Ps: 25 CB: 19	Prevalence, PSG	High
Cicek 2012 [32]	Cross-sectional	Active or inactive Ps	Active or inactive AD	AD-A: 65 AD-I: 55 Ps: 50 Con: 83	AD-A: 33.33 ± 8.4 AD-I: 30.65 ± 8.8 Ps: 35.1 ± 12.8 Con: 34.36 ± 11.1	AD-A: 29/36 AD-I: 23/32 Ps: 21/29 Con: 32/51	Prevalence and severity of RLS	High
Duffin 2009 [33]	Case-control	Ps alone or with PsA	–	Ps: 188 PsA: 86 Con: 169	NR	NR	PSQI, GFS, RLS	High
Duffin 2012 [34]	Case-control	Untreated moderate to severe plaque Ps	Age, gender and BMI matched controls	Ps: 16 Con: 16	Ps: 43.1 Con: 42.4	NR	PSG	Moderate
Duruoz 2013 [35]	Cross-sectional	CASPAR	–	40	46.06	14/26	PSQI, PsAQoL, NHP, MASES	High
Gezer 2014 [36]	Case-control	CASPAR	Healthy controls	PsA: 41 Con: 38	PsA: 41.36 ± 12.60 Con: 38.02 ± 9.79	PsA: 16/25 Con: 13/25	PSQI, HADS, PsAQoL, PASI, VAS	High
Gupta 1989 [37]	Prospective cohort	Consecutive patients with Ps at a dermatology inpatient unit	–	127	NR	NR	SCL-R, SAAST, SSTPI, CRDS	High
Karaca 2013 [38]	Cross-sectional	Ps for >5 y, diagnosed by biopsy, referred for PSG	–	33	47.64 ± 15.66	21/12	PASI, DLQI, BMI, PSG, ESS	Moderate
Kemeny 2013 [39]	RCT	Moderate to severe Ps	–	171	NR	NR	EQ-5D, DLQI, FACIT, HADS, MOS, WPAI	High
Kimball 2013 [40]	Cross-sectional	Moderate to severe plaque Ps	–	142	NR	NR	ItchVAS, MOS-S, WPAI	Moderate
Li 2009 [42]	Case-control	Patients with Ps	Controls with and without psychological factors	Ps: 70 Con: 32	NR	NR	β-EP, SAS, SDS, PSG	High
Li 2013 [41]	Nested case control	Participants in NHS and NHS-II reporting a medical diagnosis of Ps and night shift work	Participants without Ps	NHS: 62,487 NHS II: 95,561	NR	NR	Incidence of Ps	Low
Ljosaa 2012 [43]	Cross-sectional	Plaque Ps	–	Pain: 58 Discomfort: 51 None: 30	Pain: 49.7 ± 13.8 Discomfort: 51.8 ± 13.6 None: 54.1 ± 11.3	Pain: 23/35 Discomfort: 19/32 None: 18/12	PASI, GSDS, IPQ-R, DLQI	Moderate
Maari 2014 [44]	RCT	Adults with chronic plaque Ps covering at least 5% of the body surface area and an AHI ≥15	–	ADA: 10 Placebo: 10	ADA: 55.7 ± 11.8 Placebo: 49.0 ± 10.9	ADA: 9/1 Placebo: 9/1	PSG, FOSQ, ESS, daytime sleep latency	Low
Martinez-Orozco 2014 [45]	Nested case–control	Inflammatory diseases and PSG/MSLT diagnosed NC	NC without AD	156	39.1 ± 17.8	NR	Immuno-pathological diseases	Moderate
Mossner 2009 [46]	Case-control	Chronic plaque Ps	Other skin conditions	Ps: 135 Con: 55	NR	NR	PASI, DLQI, HAM-D	High
Papadavid 2010 [48]	Cross-sectional	Adult with mild to severe Ps	–	15	49	9/6	PSG, BMI	Moderate
Papadavid 2013 [47]	Cross-sectional	Chronic plaque Ps	–	35	48.9 ± 13.06	23/12	PASI, DLQI, PSG, ESS, Framingham score	Low

Pärna 2015 [49]	Cross-sectional	Ps	AD, SD or Acne	Ps: 40 AD: 41 Acne: 40 SD: 15 Con: 40	Ps: 41.8 ± 12.2 AD: 47.4 ± 13.5 Acne: 24.5 ± 5.23 SD: 34.9 ± 14.1 Con: 42.1 ± 13.9	Ps: 22/18 AD: 16/25 Acne: 6/34 SD: 8/8 Con: 17/23	DLQI, RAND-36, EST-Q	High
Patel 2009 [50]	Non-comparative	Ps	–	1	59	0/1	PSG, ESS	N/A
Shutty 2013 [51]	Case-control	Chronic plaque Ps for at least 6 mo	Healthy controls recruited from the dermatology clinic	79	44.2 ± 14.6	46/33	PSQI, PHQ-9, ISS, ISI, ESS	Moderate
Stinco 2013 [52]	Case-control	Out-clinic and hospitalized patients with a diagnosis of Ps	Controls recruited at nevi check-ups	Ps: 202 Con: 202	Ps: 50.75 ± 16 Con: 52.23 ± 12	Ps: 118/84 Con: 100/102	PASI, PSQI	High
Strober 2012 [53,54]	Interrupted time-series without comparator group	Chronic plaque Ps for ≥6 mo and suboptimal response to prior therapy	–	152	47.6 ± 13.7	91/61	MOS-S, DLQI, WPAI, PASI	Moderate
Thaci 2014 [55,56]	Randomized, parallel-group trial	Patients with active, clinically stable, chronic plaque Ps involving ≥10% of BSA or PASI ≥ 10	–	270	A: 43.9 ± 12.7 B: 44.0 ± 12.7	A: 101/36 B: 89/47	MOS-S, DLQI, EQ-5D, FACIT	High
Tsai 2011 [57]	Nested case–control	One outpatient visit or admission claim with an ICD-9-CM code 696.0 or 696.1	Matched case controls	Ps: 51,800 Con: 170,948	Ps: 46.6 ± 18.6 Con: 46.1 ± 19.1	Ps: 31,923/19,877 Con: 102,644/68,304	Prevalence of OSA	Low
Tyring 2006 [58]	RCT	Patients with active, clinically stable plaque Ps with ≥10% BSA, a PASI ≥10 and at least one prior therapy	–	ETN: 311 Placebo: 307	ETN: 45.8 ± 12.8 Placebo: 45.6 ± 12.1	ETN: 203/108 Placebo: 216/91	PASI, FACIT, BDI, HAM-D	Low
Wong 2009 [59]	Case-control	Patients with Ps	Controls from a Sleep/Wake Center	Ps: 13 Con: 1338	Ps: 57.9	Ps: 7/4	PSG	High
Woodcock 2010 [60]	Cross-sectional	Patients with Ps or Ps and PsA.	–	Ps: 12 PsA: 3	OSA-: 39 OSA+: 45	NR	PASI, BMI, DLQI, PSG	Moderate
Wu 2008 [61]	Nested case–control	Self-report of psoriasis by BSA	Age, gender, region and race matched controls	Ps: 1127 Con: 1127	Ps: 53.1 ± 15.1 Con: 52.5 ± 15.1	Ps: 522/605 Con: 522/605	BSA, comorbidities	Low
Yang 2012 [62]	Nested case–control	OSA (ICD-9-CM codes 780.51, 780.53, 780.57 or 327.23) diagnosed by PSG in 2001–2005. Ps was identified using ICD-9-CM codes 696.1 or 696.0	Members of the cohort without OSA.	OSA: 2258 Con: 11,255	40.9 ± 10.74	8484/5029	Incidence of Ps	Low
Zachariae 2012 [63]	Cross-sectional	Patients with Ps	Patients with AD, U, GP, NP or controls with non-pruritic vascular malformations.	AD: 20 Ps: 20 U: 20 GP: 12 NP: 11 Con: 20	AD: 31.4 ± 12.7 Ps: 51.6 ± 17.0 U: 44.5 ± 17.0 GP: 48.8 ± 12.5 NP: 60.2 ± 20.6 Con: 41.2 ± 15.0	AD: 8/12 Ps: 13/7 U: 6/14 GP: 4/8 NP: 5/6 Con: 4/16	ISS, BDI, BSI, DLQI, PSQI, PASI, SCORAD	High

AD, atopic dermatitis; ADA, adalimumab; AHI, apnea hypopnea index; β-EP, β-endorphin; BAI, Beck anxiety inventory; BDI, Beck depression inventory; BMI, body mass index; BSA, body surface area; BSI; brief symptom inventory; CASPAR, classification criteria for psoriatic arthritis; CB, chronic bronchitis; Con, controls; CRSD, Carroll rating scale for depression; DLQI, dermatology life quality index; EQ-5D, EuroQoL-5D; ESS, Epworth sleepiness scale; EST-Q, emotional state questionnaire; ETN, etanercept; FACIT, functional assessment of chronic illness therapy; FOSQ, functional outcomes of sleep questionnaire; GFS, general fatigue scale; GP, genital pruritus; GSDS, general sleep disturbance survey; HADS, hospital anxiety and depression scale; HAM-D, Hamilton rating scale for depression; ICD-9-CM, international classification of diseases, ninth revision, clinical modification; IPQ-R, illness perception questionnaire; ISI, insomnia severity index; ISS, itch severity scale; MASES, Maastricht ankylosing spondylitis enthesitis score; MOS-S, medical outcomes study-sleep; NC, narcolepsy; NHP, Nottingham health profile; NHS, nurses' health study; NP, nephrogenic pruritus; NR, not reported; PASI, psoriasis area severity index; PHQ-9, patient health questionnaire; PLMI, periodic leg movement index; Ps, psoriasis; PsA, psoriatic arthritis; PsAQoL, psoriatic arthritis quality of life scale; PSG, polysomnography; PSQI, Pittsburgh sleep quality index; OSA, obstructive sleep apnea; RAND-36, RAND-36-item health-related quality of life survey; RCT, randomized controlled trial; RLS, restless legs syndrome; RoB, risk of bias; SAAST, self-administered alcoholism screening test; SAS, self-rating anxiety scale; SCL-R, symptom checklist–revised version; SCORAD, scoring atopic dermatitis; SD, seborrheic dermatitis; SDS, self-rating depression scale; SSTPI, Spielberger state-trait personality inventory; U, urticaria; VAS, visual analog scale; WPAI, work productivity and activity impairment.

Table 4
Insomnia observational studies.

Study identifier	Purpose	Study population	Number of patients	Selected results
Duffin 2009 [33]	To corroborate prior evidence that pruritus, pain from lesions, PsA and diminished QoL predict sleep disturbance	Ps and PsA patients from the Utah Ps Initiative registry and control from dermatology clinics.	Ps: 188 PsA: 86 Con: 169	PSQI scores were higher in PsA but comparable in Ps and controls. The rate of insomnia was 15.1% in PsA, 5.9% in Ps and 5.9% in controls.
Duruoz 2013 [35]	To assess the relationship between sleep quality and QoL in PsA	Patients fulfilling the CASPAR criteria for PsA.	40	Sleep quality was correlated with quality of life. 44.8% of participants had poor sleep quality.
Gezer 2014 [36]	To assess sleep quality in patients with Ps.	Patients with PsA and controls.	PsA: 41 Con: 38	Sleep disturbance, daytime dysfunction and total PSQI were higher in PsA than Con. PSQI was correlated with HADS-A, PsAQoL, and pain.
Gupta 1989 [37]	To determine the association of pruritus-associated nocturnal awakenings in Ps patients with alcohol and caffeine consumption and psychological status.	Consecutive patients with Ps at a dermatology inpatient unit.	127	Patients with frequent awakenings reported greater alcohol consumption, but not caffeine consumption. They also had higher depression scores.
Kimball 2013 [40]	To determine the effects of pruritus from Ps on sleep and work productivity.	Patients with moderate to severe plaque Ps from a phase II trial of ixekizumab.	142	Work productivity decrements in patients with moderate to severe Ps are associated with sleep impairment and pruritus.
Ljosaa 2012 [43]	To associate the effect of pain and discomfort caused by Ps on sleep disturbance and psychological distress.	Adult Caucasian patients with Ps from hospital inpatients and outpatient dermatology units.	Pain: 58 Discomfort: 51 None: 30	Sleep disturbance is a primary mediator between pain and QoL.
Mossner 2009 [46]	The aim of this study was to evaluate the psychosocial morbidity in Ps patients.	Patients with chronic plaque Ps and controls with other skin conditions.	Ps: 135 Con: 55	Sleep disorder was more common in Ps at 34% than controls at 16%. Ps patients also had higher rates of depression, anxiety, and nervousness.
Pärna 2015 [49]	To evaluate the associations between chronic inflammatory skin conditions and patients' emotional state and quality of life.	Adult patients with Ps, AD, SD or acne.	Ps: 40 AD: 41 Acne: 40 SD: 15 Con: 40	Dermatology patients had higher rates of anxiety and insomnia than controls. Acne patients had the highest acne scores, whereas Ps patients had the highest insomnia scores.
Shutty 2013 [51]	To assess the relationship between Ps and the categorized levels of PSQI, ESS, ISI, and PHQ.	Outpatients with chronic plaque Ps for at least 6 mo as well as healthy controls recruited from the dermatology clinic.	79	Patients with Ps had an OR of 4.3 (95% CI: 1.19–1.75) for insomnia and 6.1 (95% CI: 2.3–16.2) for depression compared to controls.
Stinco 2013 [52]	To determine if there is a relationship between Ps severity and sleep disturbance.	Out-clinic and hospitalized patients with a diagnosis of Ps vulgaris and an equal number of age- and sex-matched healthy controls (group B) was recruited between out-clinic patients observed for nevi check-up.	Ps: 202 Con: 202	There was no correlation between PASI and PSQI score. Anti-psoriatic therapy improved PASI but not PSQI.
Wu 2008 [61]	To determine if there is a higher rate of medical comorbidity in patients with Ps.	Americans who completed the NHWS internet survey.	Ps: 1127 Con: 1127	Ps patients had an OR of 1.44 (95% CI: 1.19–1.75) for developing a sleep disorder/insomnia.
Zachariae 2012 [63]	To examine the validity of a Danish adaptation of the ISS.	Patients with AD, Ps, U, GP, NP or controls with non-pruritic vascular malformations.	AD: 20 Ps: 20 U: 20 GP: 12 NP: 11 Con: 20	Patients with Ps had similar sleep quality and sleep initiation to other pruritic disorders, but greater sleep difficulty than controls.

AD, atopic dermatitis; CASPAR, classification criteria for psoriatic arthritis; CI, confidence interval; Con, controls; ESS, Epworth sleepiness scale; GP, genital pruritus; HADS, hospital anxiety and depression scale; ISI, insomnia severity index; NHWS, national health and wellness survey; NP, nephrogenic pruritus; NR, not reported; OR, odds ratio; OSA, obstructive sleep apnea; PASI, psoriasis area severity index; PHQ-9, patient health questionnaire; Ps, psoriasis; PsA, psoriatic arthritis; PsAQoL, psoriatic arthritis quality of life scale; PSG, polysomnography; PSQI, Pittsburgh sleep quality index, RLS, restless legs syndrome; SD, seborrheic dermatitis; U, urticaria.

In insomnia, both ETN and ADA therapy improved sleep disturbance, as well as QoL, fatigue, and symptoms of depression [39,53–56,58]. In OSA, treatment with ADA did not show an improvement in AHI, indicating that it does not have a direct effect on OSA severity [44].

Limitations

The primary limitation of this systematic review is the quality of evidence. The overall RoB for the included studies is 48% high, 27% moderate and 21% low (Table 3). Secondary limitations include the variation in inclusion criteria for psoriasis and PsA, the diagnostic criteria for sleep disorders and the scarcity of evidence for many sleep disorders. The majority of the studies include patients with plaque psoriasis or who fit the classification criteria for psoriatic arthritis (CASPAR) [64]. This aids in consistency when making inter-study comparisons, but excludes the less common presentations of psoriasis, including erythrodermic, pustular, guttate and nail psoriasis. The diagnostic criteria for sleep disorders are another limitation because

Table 5
Observational studies of OSA in psoriasis.

Study identifier	Purpose	Population	Number of patients	Selected results
Bissonnette 2012 [30]	To present baseline PSG data for a trial of ADA in patients with Ps and OSA.	Patients with chronic plaque Ps (>5% body surface) and OSA (AHI>15).	20	The prevalence of OSA was 81.8%.
Buslau 1999 [31]	To compare the prevalence of OSA in Ps with the known risk group of chronic bronchitis.	Male patients with Ps or controls with chronic bronchitis >40 y of age with two signs of OSA.	Ps: 25 CB: 19	The prevalence of OSA was 36% in patients with Ps and 32% in patients with chronic bronchitis.
Karaca 2013 [38]	To determine the prevalence and association of OSA in patients with Ps.	Adults with Ps for more than 5 y diagnosed for biopsy from a hospital dermatology clinic.	33	The prevalence of OSA was 54.5%.Patients with OSA had higher PASI and ESS scores.
Duffin 2012 [34]	To determine if there is an increased prevalence of OSA in patients with Ps.	Patients with untreated moderate to severe plaque Ps and age, gender and BMI matched controls.	Ps: 16 Con: 16	The mean AHI was higher in Ps than controls (20.9 vs. 8.1).
Li 2009 [42]	To detect serum β-EP and sleep quality in patients with Ps.	Patients with Ps and controls with and without psychological factors.	Ps: 70 Con: 32	AHI and $MinSaO_2$ were higher in Ps patients than Con.
Papadavid 2010 [48]	To investigate the prevalence of OSA among Ps patients in relation to BMI, PASI score and other comorbidities, such as hypertension, hyperlipidemia, and diabetes mellitus.	Adult patients with mild to severe Ps.	15	The rate of OSA was 66.7%. Nine of ten patients were obese and eight received systemic therapies for Ps.
Papadavid 2013 [47]	To determine if the presence and severity of Ps cause an increased likelihood of OSA.	Outpatients with chronic plaque Ps.	35	BMI and hypertension, but not Ps severity, were the most accurate predictors of OSA.
Patel 2009 [50]	To report a case of OSA in a woman with familial partial lipodystrophy type 2 and numerous dermatological conditions.	Woman with familial partial lipodystrophy type 2, Ps, seborrheic keratoses, sebaceous hyperplasia and actinic changes.	1	The woman was diagnosed with OSA with and RDI of 35.9 and an ESS score of 13. CPAP reduced ESS to 8.
Tsai 2011 [57]	This study aims to describe the epidemiology of Ps and the prevalence of comorbidities in patients with Ps in Taiwan.	Patients who had at least one outpatient visit or admission claim with an ICD-9-CM code for Ps (696.0 for psoriatic arthropathy or 696.1 for other Ps) in the Taiwan National Health Insurance (NHI) claims database during 2006 and matched case controls.	Ps: 51,800 Con: 170,948	Patients with Ps have a RR of 3.86 (95% CI: 2.26–6.71) of having co-morbid OSA.
Woodcock 2010 [60]	To assess the prevalence of cardiovascular risk factors and obstructive sleep apnea in patients with Ps and PsA.	Patients with Ps or Ps and PsA.	Ps: 12 PsA: 3	OSA was present in 53% of participants and all of the individuals with PsA.
Yang 2012 [62]	This study aims to investigate the risk of Ps or PsA in patients with OSA compared with age- and gender-matched unaffected individuals, using a nationally representative population-based dataset.	Taiwan Longitudinal Health Insurance Database 2000.	OSA: 2258 Con: 11,255	The adjusted HR for Ps was 2.30 (95% CI: 1.13–4.69) for individuals with OSA.

ADA, adalimumab; β-EP, β-endorphin; BMI, body mass index; BSA, body surface area; CB, chronic bronchitis; CI, confidence interval; Con, controls; ESS, Epworth sleepiness scale; HR, hazard ratio; ICD-9-CM, international classification of diseases, ninth revision, clinical modification; OSA, obstructive sleep apnea; PASI, psoriasis area severity index; Ps, psoriasis; PsA, psoriatic arthritis; PSG, polysomnography; PSQI, Pittsburgh sleep quality index, RR, risk ratio.

Table 6
Observational studies for RLS and PLMD in psoriasis.

Study identifier	Purpose	Population	Number of patients	Selected results
Bilgic 2013 [29]	To measure the prevalence and severity of RLS in psoriasis.	Consecutive psoriasis patients from the Ps Unit at Akdeniz University Hospital.	44	The rate of RLS was 15.9%.
Cicek 2012 [32]	To determine the prevalence and severity of RLS in AD and Ps.	Patients with AD or Ps.	AD-A: 65 AD-I: 55 Ps: 50 Con: 83	RLS was present in 40.8% of AD patients, 18% of Ps patients and 10.8% of Con.
Duffin 2009 [33]	To corroborate prior evidence that pruritus, pain from lesions, PsA and diminished QoL predict sleep disturbance.	Ps and PsA patients from the Utah Ps Initiative registry and control from dermatology clinics.	Ps: 188 PsA: 86 Con: 169	The rate of RLS was 15.1% in PsA, 6.4% in Ps and 4.1% in controls.
Wong 2009 [59]	To compare sleep characteristics in patients with Ps and sleep clinic controls.	Ps patients from the Utah Ps Initiative or non-psoriatic controls from a Sleep/Wake Center.	Ps: 13 Con: 1338	Ps patients had a higher PLMI than controls at 44.5 ± 48.7 vs. 22.2 ± 27.1. Ps patients had lower mean SaO_2.

AD, atopic dermatitis; Con, controls; PLMI, periodic limb movement index; Ps, psoriasis; QoL, quality of life; RLS, restless legs syndrome; SaO_2, oxygen saturation.

Table 7
Intervention studies.

Sleep disorder	Insomnia				OSA
Authors	Strober 2012 [53,54]	Kemeny 2013 [39]	Thaci 2014 [55,56]	Tyring 2006 [58]	Maari 2014 [44]
Purpose	To assess the extent of baseline sleep impairment and the effect of ADA on sleep and other patient-reported in patients with Ps.	To determine if ETN improves QoL in Ps and PsA patients from Central and Eastern Europe, Latin America and Asia.	To quantify baseline aspects of sleep and improvement in patients with Ps receiving ETN when allowed concomitant topical medications.	To assess the impact of ETN on symptoms of Ps and associated fatigue and depression.	To determine the effect of ADA on sleep parameters in patients with Ps and OSA.
Study design	Open-label, interrupted time-series without comparator group	Randomized, parallel-group trial	Randomized, parallel-group trial	Randomized, double-blind, placebo-controlled, parallel-group trial	Single center, randomized, double-blind, placebo-controlled, parallel-group trial
Number of patients	152	171	270	ETN: 311 Placebo: 309	ADA: 10 Placebo: 10
M/F	91/61	NR	A: 101/36 B: 89/47	ETN: 216/95 Placebo: 203/106	ADA: 9/1 Placebo: 9/1
Study population	Participants in the PROGRESS trial of ADA.	Patients with moderate to severe Ps.	Patients with active, clinically stable, chronic plaque Ps involving ≥10% of the total body surface or PASI ≥10.	Patients with active, clinically stable plaque Ps with ≥10% BSA, a PASI ≥10 and at least one prior therapy.	Adults with chronic plaque Ps covering at least 5% of the body surface area and an AHI ≥15.
Age (Mean ± SD)	47.6 ± 13.7	NR	A: 43.9 ± 12.7 B: 44.0 ± 12.7	ETN: 45.6 ± 12.1 Placebo: 45.8 ± 12.8	ADA: 55.7 ± 11.8 Placebo: 49.0 ± 10.9
Intervention	ADA: 80 mg at week 0, then 40 mg every other week from week 1	A: ETN 50 mg QW for 24 wk B: ETN 50 mg BIW for 12 wk, ETN 50 mg QW for 12 wk	A: ETN 50 mg BIW for 12 wk, ETN 50 mg QW for 12 wk B: ETN 50 mg QW for 24 wk	ETN 50 mg BIW or placebo	ADA 80 mg at week 0, then 40 mg every other week from week 1 or placebo
Treatment duration	16 wk	24 wk	24 wk	12 wk	84 d
Outcome measures	MOS-S, DLQI, WPAI, PASI	EQ-5D, DLQI, FACIT, HADS, MOS, WPAI	PASI, BSA, DLQI, MOS-S, EQ-5D, HADS, FACIT, MOS	PASI, FACIT, BDI, HAM-D	PSG, FOSQ, ESS, daytime sleep latency
Relevant results	ADA improved sleep quality by 15% from baseline, as well as DLQI score, pain and work productivity. The improvement in sleep was partially explained by improvements in PASI.	ETN significantly improved EQ-5D, DLQI, FACIT and HADS. Change in EQ-5D was greater in patients with PsA.	ETN twice weekly was more effective than once weekly. Both arms improved MOS-S sleep domains. Improvements in pruritus decreased sleep disturbance which improved QoL and fatigue.	ETN improved BDI and HAM-D scores by at least 50% in a greater proportion of patients than placebo. Fatigue reduction was associated with improvements in joint pain.	ADA did not produce a difference in AHI between the two groups.
Overall risk of bias assessment	Moderate	High	High	Low	Low

ADA, adalimumab; BDI, Beck depression Inventory; BIQ, twice weekly; BSA, body surface area; Con, controls; DLQI, dermatology life quality index; ESS, Epworth sleepiness scale; ETN, etanercept; FACIT, functional assessment of chronic illness therapy; HADS, hospital anxiety and depression scale; HAM-D, Hamilton rating scale for depression; MOS-S, medical outcomes study – sleep; NR, not reported; Ps, psoriasis; PSG, polysomnography; PSQI, Pittsburgh sleep quality index; PASI, psoriasis area severity index; QW, once weekly; WPAI, work productivity and activity impairment.

studies rarely use all the ICSD criteria for diagnostic purposes [24,25]. The inclusion criteria were deliberately relaxed (Table 2) to remove criteria like duration for insomnia and the presence of excessive daytime sleepiness for AHI <15 in OSA. The vast majority of the studies, included and excluded, merely measured sleep variables or used a sleep survey instead of examining true sleep disorders. Finally, there is a wide variety of sleep disorders which have never or rarely been evaluated in individuals with psoriasis and PsA including hypersomnia, parasomnias and circadian rhythm disorders.

Conclusions

This systematic review demonstrates a link between psoriasis and PsA with OSA and RLS. There is no conclusive evidence that psoriasis and PsA are associated with an elevated prevalence of insomnia, PLMD, narcolepsy, or shift work disorder. The relationship between psoriatic conditions and other sleep disorders has not been examined.

There is extensive literature on the relationship between psoriasis and insomnia due to the fact that pruritus and pain are frequent symptoms of psoriasis that may interrupt or prevent sleep. Insomnia, RLS and PLMD may also be mediated by altered circadian rhythms mediated by decreased TEWL and altered cutaneous blood flow, resulting in decreased modulation of CBT and drop in CBT that is necessary for sleep onset. It is interesting to note that the prevalence of RLS has been reported as elevated in all studies, while insomnia is highly variable, as both sleep disorders are attributed to pruritus and pain. It is possible that while RLS and PLMD are attributable to an involuntary sensation or movement and circadian factors [25], there is a greater psychological component to the development of insomnia in psoriatic conditions. The transition from transient physiological discomfort that impacts sleep to chronic insomnia may be mediated by patient expectations, co-morbid depression and anxiety, or sleep hygiene [24,25], which may be the basis for the greater variation in the prevalence of insomnia in psoriasis.

The relationship between psoriasis, PsA and OSA is likely mediated by the autoimmune nature of psoriasis and PsA. OSA is associated with obesity, hypertension, diabetes, metabolic syndrome and psychiatric disorders, all of which are feed-forward pro-inflammatory states [19,65–69]. Psoriasis also has an independent association with obesity, diabetes, hypertension, cardiovascular morbidity and depression [6–8,11,23]. The relationship between these conditions and psoriasis is too complex for the treatment of psoriasis to directly influence symptoms of OSA [44]. It is more likely that the underlying inflammation and endocrine disruption related to comorbid conditions will need to be treated in conjunction with the symptoms of psoriasis and OSA, in order to see improvement when these conditions present simultaneously.

Dermatologists and other practitioners treating patients with psoriasis and PsA should consider the possible presence of an undiagnosed sleep disorder. It is important that practitioners discuss the current sleep quality with patients during routine evaluation of psoriasis and request further sleep consultations when necessary. Complaints of poor sleep may be more extensive than sleep disruption from pruritus and pain related arousals, and include OSA, PLMD and circadian rhythm sleep-wake disorders. Patients with multiple medical comorbidities in association with their psoriasis should be considered candidates for PSG, especially if they are significantly clinically depressed, obese, hypertensive or diabetic, as some of these comorbidities are also independently associated with OSA. It is critical that treatment for psoriasis focus on mitigating pain and pruritus, and patients should be counseled on good sleep hygiene practices.

Practice points

- Patients with psoriasis are at an increased risk of OSA and they should be considered candidates for polysomnography, especially in the presence of elevated BMI, diabetes, hypertension or other known OSA risk factors.
- Patients complaining of symptoms of insomnia should receive dermatologic treatments for psoriasis in conjunction with standard insomnia therapies, as a reduction in pruritus and pain may resolve the sleep disturbance.
- Patients with insomnia or fatigue complaints should be evaluated for the possibility of periodic limb movements in sleep, especially in psoriasis patients with complaints of pain and pruritus.
- Circadian factors should be taken into consideration when managing sleep disturbance in the psoriasis patient. The impact of psoriasis and associated pruritus and pain on sleep initiation and maintenance may be mitigated by factors such as maintenance of a regular sleep-wake schedule and a cool ambient room temperature in patients with extensive psoriasis who may experience difficulty with thermoregulation and sleep onset, as a result of a reduced ability to dissipate heat peripherally.

Research agenda

- It is important that epidemiological studies in psoriasis evaluate sleep disorders following the standard ICSD-3 criteria, in order to accurately measure the prevalence and incidence of sleep disorders in this population.
- It will be critical to establish if biologics targeting immune mechanisms in psoriasis are effective in decreasing immune markers linked to additional systemic diseases that mediate the risk for sleep-disordered breathing.
- The incidence of acute vs. chronic insomnia should be measured in patients with psoriasis to determine if the sleep disturbance is acute and stems directly from pruritus and pain, or if it represents ICSD-3 chronic insomnia.

Conflicts of interest

The authors have no conflict of interest to declare.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.smrv.2015.09.003.

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