Sleep Medicine Reviews 46 (2019) 108–123

Contents lists available at ScienceDirect

Sleep Medicine Reviews

journal homepage: www.elsevier.com/locate/smrv

CLINICAL REVIEW

Light therapies to improve sleep in intrinsic circadian rhythm sleep disorders and neuro-psychiatric illness: A systematic review and meta-analysis

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ARTICLE INFO

Article history:
Received 9 November 2018
Received in revised form
2 April 2019
Accepted 23 April 2019
Available online 30 April 2019

Keywords:
Bright light treatment
Dark treatment
Dawn simulation
Circadian rhythm sleep disorders
Co-morbid insomnia
Mental illness
Dementia
Delayed sleep phase disorder
Affective disorders
Psychosis

SUMMARY

Circadian dysregulation causes sleep disturbance and impacts quality of life and functioning. Some interventions target circadian entrainment through modifying light exposure, but existing reviews of light interventions for sleep improvement include few studies in psychiatric populations. We examined effect of light interventions on sleep quality, duration and timing, and effect moderators. We included controlled studies in intrinsic circadian rhythm disorders (such as advanced or delayed sleep) and in neuropsychiatric disorders with assumed high prevalence of circadian dysregulation (such as affective and psychotic disorders). Articles were identified through database searching: 40 studies reporting 49 relevant intervention comparisons met inclusion criteria. Meta-analysis showed improvements in sleep continuity (ES = −0.23, p = 0.000), self-reported sleep disturbance (ES = −0.32, p = 0.014), and advancement of delayed sleep timing (ES = −0.34, p = 0.010). Although the small number of studies limited meta-regression, evening light avoidance was associated with greater increase in total sleep time. Effects of light on sleep and circadian outcomes have received limited attention in studies in psychiatric disorders, but results were promising in these groups. These findings invite further refinement and testing of light interventions to improve sleep in psychiatric disorders, with improved assessment and specification of problems, and the development and implementation of light schedule interventions for delayed sleep.

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Introduction

Circadian rhythms exert a strong influence on sleep propensity, alertness and performance, modulating these each day [1]. Circadian rhythms are entrained to the external day-night cycle by environmental cues, of which the light-dark cycle is by far the strongest influence [2]. When circadian rhythms are well entrained, the suprachiasmatic nucleus (SCN) oscillates in time with the external day-night cycle, and all other bodily tissues and organs are synchronised by the SCN's rhythmic signal. Circadian dysregulation has been connected to various negative physical and mental health consequences, and of course, to poor sleep [3]. As circadian rhythms are known to be entrained by light exposure, interventions that modify light exposure patterns to reduce sleep disturbance have strong theoretical support. Light and darkness also influence sleep and arousal processes more acutely, through 'direct', non-circadian pathways, which may contribute toward therapeutic effects [4].

In circadian rhythm sleep disorders (CRSDs) such as delayed sleep phase disorder (DSPD) or advanced sleep phase disorder (ASPD), interventions may aim to normalise timing of sleep. Interventions may equally aim to improve sleep quality or duration by improving the synchronisation between circadian phase and sleep-wake timing where these are out of phase [5]. Interventions may aim to increase the amplitude of the SCN rhythm, which can become reduced in older age and in neurodegenerative disorders [6]. Symptoms of circadian dysregulation also frequently occur in

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https://doi.org/10.1016/j.smrv.2019.04.012
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Glossary

Suprachiasmatic nucleus (SCN)	the brain's internal master clock, cells in the SCN oscillate with a period of approximately 24hr, and are entrained to external time by environmental cues.
Zeitgeber	from German "time-giver"; environmental cues which entrain circadian rhythms; light is the strongest zeitgeber for entraining circadian rhythms.

the context of neuropsychiatric disorders; for instance irregular sleep wake disorder (ISWD) is common in dementia [7]. 'Free-running' rhythms (non 24hr sleep wake disorder) most commonly occur due to eye or optic nerve damage. Non 24hr rhythms can be observed in time-cue free environments in sighted participants [8], but occur under free-living conditions in sighted people with schizophrenia [9] and rapid-cycling bipolar disorder [10].

Circadian dysregulation impacts negatively on quality of life, on occupational and social engagement [11–13], and on cognitive functioning [14]. Although causality is challenging to investigate, evidence suggests circadian dysregulation and poor sleep exacerbate psychiatric symptoms [15–17]. Circadian dysregulation very often precedes other symptoms, and some evidence suggests a causal role in development of illness [18,19]. There is much hope that treating circadian dysregulation and reducing sleep disturbance might improve functioning and reduce suffering in a range of conditions. Indeed, some studies which reduced insomnia via psychological intervention, reported a reduction in psychiatric symptoms [20–22]; these effects have been shown to be mediated by reductions in sleep disturbance [20].

Historically sleep problems have been neglected in groups with neuropsychiatric disorders due to diagnostic overshadowing [23], and assumptions that sleep problems are purely secondary to psychiatric symptoms. Unfortunately sleep problems often persist even if affective or psychotic symptoms are well-controlled [24,25]. There is increasing recognition that sleep problems require independent attention irrespective of co-morbid conditions. In accordance with this the 'primary'/'secondary' insomnia distinction was removed from DSM-5 and ICSD-3 [26]. Circadian dysregulation disorder definitions have not been similarly modified; the ICSD-3 stipulates for diagnosis of CRSD the sleep disturbance must not be "better explained" by another medical, neurologic or mental disorder. Further, it contains no category for CRSD secondary to another disorder [27]. Studies which examine circadian dysregulation in samples with neuropsychiatric disorders find high prevalence of patterns similar to ASPD, DSPD, ISWD and non-24hr [6,9,24], but usually CRSD terminology is not applied.

Previous meta-analyses of studies of light treatment for sleep improvement in CRSD (without co-morbidity) and in dementia were inconclusive, and limited by small participant and trial numbers [28,29]. Despite the high levels of circadian dysregulation symptoms in populations with severe mental illness, no meta-analysis has yet included studies in these groups. In the meta-analysis by Van Maanen et al. (2016) [30] broad inclusion criteria were used, including any reported or diagnosed sleep disorder or complaint. The authors identified studies in groups as diverse as renal patients, brain injury, and mid-winter insomnia in the subarctic, yet no studies targeting sleep in samples with co-morbid mental illness which met inclusion criteria were found.

Evidence syntheses of the effects of light in seasonal and non-seasonal mood disorders have shown effects on improvement in mood [31,32], but the effects on sleep are less well understood.

This review presents a synthesis of the effects of light schedule interventions on circadian rest-activity patterns and sleep, across dementia, CRSD, and psychotic, affective and personality disorders. Acknowledging the lack of measurement of circadian dysregulation and application of CRSD terminology in existing research in psychiatric disorders, our inclusion criteria are operationalised specifically to ensure studies in these groups are not excluded.

Our aims are: 1) To examine the effect of interventions altering light exposure patterns on sleep and rest-activity parameters, in populations diagnosed with, or at risk of, circadian dysregulation. 2) To examine predictors of effect size.

Methods

The protocol for this review was prospectively published on Prospero, and is available at: http://www.crd.york.ac.uk/PROSPERO/display_record.php?ID=CRD42017072387. For changes from protocol see Supplement S1.

Search, screening and inclusion

Systematic searches were performed using keywords and subject headings in MEDLINE, Embase, PsycInfo, and AMED (via Ovid), and CINAHL (via EBSCO). Searches were run in August 2017, from database inception. Truncation (*), wildcards, and the (adj) adjacent operator were used to capture terminology variants.

Searches were limited to human studies, and a trial filter was applied; 10% of articles excluded by the trial filter were screened to confirm no relevant studies were being filtered. Database searches were supplemented by reference checking relevant reviews and contacting key authors in the field. Only peer-reviewed publications of primary research were included. For an example search strategy see Supplement S2.

We included populations with intrinsic CRSDs, and those with other conditions strongly associated with circadian dysregulation. We identified and specified a list of conditions a priori based on the background literature; amongst them were dementia, psychotic disorders and affective disorders (see Tables 1 and S3).

During screening we encountered studies where participants were not formally diagnosed with DSPD or ASPD, but where inclusion criteria specified suggested that most participants had DSPD or ASPD. After discussion we included these studies and planned a sensitivity analysis on the effect of their removal. We excluded groups at-risk of (not currently presenting with) mental disorders, 'extrinsic' CRSDs, specifically jet-lag disorder and shift work disorder, and problems occurring due to very unusual light environments (e.g., space flight, arctic winter).

Results were de-duplicated and screened; one third of results were independently screened by another researcher. Authors of relevant conference abstracts were contacted to seek publications. Potentially relevant studies were assessed for eligibility using the full text. All those where there was ambiguity were assessed by another researcher. An additional 10% of randomly selected full texts were independently assessed by another researcher to check consistency in application of the inclusion criteria. After familiarisation we found inconsistency only between categories "include"/"unsure" and "exclude"/"unsure" – not between "include/"exclude", therefore we were satisfied that all relevant studies were being identified. Uncertainty was resolved through discussion, and consulting a third researcher where needed. Where multiple papers reported on the same study all were included and data were combined.

Table 1
Inclusion criteria.

Study type	Intervention study (not observational and correlational studies) Exclude: case studies (<5 people)
Population	Intrinsic CRSD (DSPD, ASPD, ISWD or Non-24hr)[a] OR Psychiatric or neurodegenerative disease with high levels of circadian dysregulation: dementia, psychotic disorders, personality disorder, affective disorders (bi-polar or unipolar, seasonal or non-seasonal) AND Over 70% of the sample meet the above criteria, or subgroups reported Adults or adolescents (over 13)
Intervention	Interventions altering light exposure (amount, timing, or spectral qualities) as a core (not optional) component, with primary or secondary aim of improving sleep. Examples: light boxes, light visors, dark treatment, amber glasses, increasing daylight exposure, increasing or decreasing indoor lighting.
Comparison	Control group without the light/dark intervention
Outcome	Self-reported, clinician reported, or objectively measured sleep or rest-activity variables (Sleep onset latency, maintenance, quality, depth, timing, duration, rest-activity patterns)

[a] CRSD = circadian rhythm sleep disorder, DSPD = delayed sleep phase disorder, ASPD = advanced sleep phase disorder, ISWD irregular sleep wake disorder, Non-24hr = non-24hr sleep wake phase disorder, SCN = suprachiasmatic nucleus.|

Data extraction

Population, setting, intervention and outcomes data were extracted and cross-checked by another researcher. Mean, standard deviation (SD) and sample size were extracted, or where required calculated (from confidence intervals (CIs) and standard errors (SEs)), or requested from authors. Where outcome measures were scaled in the opposite direction (e.g., higher = better, higher = worse) effect sizes were reversed. Clock times (hours, minutes) were converted to decimal hours or total minutes.

Risk of bias assessment

Methodological quality in controlled studies was appraised during data-extraction using the Cochrane risk of bias tool. Participant blinding is a pervasive difficulty in light/dark treatment (interventions are by nature perceptible) and this affected overall risk of bias assessments across studies. It was therefore decided to use gradations of 'low/medium/high', to capture more information than 'low/high' quality ratings. Studies were independently assessed by a second researcher, discrepancies were resolved through discussion.

Analysis

There are various sleep outcomes, many of which are dissimilar, and therefore not amenable to pooling. The relevance of outcomes in each group was based upon the known complaints of that population and relevance was noted a-priori to guide synthesis (see Table S4).

To reduce risk of bias in outcome extraction, a hierarchy of outcome measures for each construct was determined a priori, in case multiple measures were reported (e.g., TST actigraphy and self-report), so the preferred measure for that construct was pre-determined (see Table S5).

Where studies included multiple relevant comparisons (for instance morning light/afternoon light/placebo) each was included individually, as differences in effects depending on intervention features were of interest. To avoid double counting the control group was split accordingly [33].

Meta-analyses and meta-regressions were run in Stata 14. Standardised mean differences (SMDs) were used due to inclusion of multiple outcome measures, the random effects model was used to allow for heterogeneity [34]. Meta-regression covariates were planned in advance. To avoid applying meta-regression with too few trials [35] we set a lower limit of 10 comparisons. Covariates were tabulated and visually inspected for collinearity and confounding to avoid misinterpretation of associations (e.g., if only studies in certain diagnoses used afternoon light, population effects and intervention effects cannot be separated). We did not run meta-regressions for participant characteristics for which we had only a study mean, such as age or mean baseline sleep quality, because without individual patient data results may be misleading [35].

The following sensitivity analyses were run for each meta-analysis:

1) Removing and examining any striking outliers
2) Removing studies at high risk of bias
3) Removing studies with neuropsychiatric diagnosis but no sleep inclusion criteria.
4) Removing studies where diagnosis was assumed not formally assessed

Sub-group analyses and differences in outcomes between populations

It was our assumption on embarking on this review that there is some commonality in mechanisms of sleep disturbance between groups and therefore that there may be commonality in intervention effects. Research in animal models and from genetic studies suggests the mechanisms causing circadian dysregulation may be similar across diagnostic groups [36,37]. Although our primary aim was to examine effects across diagnostic groups, we have examined sub-group differences [38]. We examined differences first using overall tests for heterogeneity between sub-groups for each meta-analysis. We then examined sub-grouped forest plots and for those where CIs did not overlap [33] we examined differences statistically using the method described by Bornstein et al. [39].

Results

Search results

3319 unique results were returned, resulting in 320 potentially relevant studies for which full-text was retrieved and assessed for eligibility. 48 articles reporting on 40 studies met inclusion criteria. Fig. 1 shows the flow of articles through search and screening.

Study characteristics

There were 40 controlled studies reporting on 49 relevant intervention comparisons (see Table 2). Studies in non-dementia populations were conducted in participant's own homes, with the exception of five inpatient studies [40–44]. Studies in dementia

were based in institutions, with the exception of four in private residences [45–48]. The most commonly used intervention was the light box, followed by bright indoor light, outdoor light, or light from a combination of sources. Light visors, dawn simulation, and light avoidance were infrequently used. Many interventions were single component. The most common additional components were sleep schedule recommendations and sleep hygiene advice. Some studies combined morning light exposure and evening light avoidance. A small minority of studies combined light exposure with exogenous melatonin [49,50], cognitive behavioural therapy for insomnia (CBT-i) [40,51] or wake therapy [44]. Many interventions did not describe including any maintenance advice or treatment.

Population groupings were decided after data-extraction and before analysis. The two studies in seasonal affective disorder (SAD) both included hypersomnia, hence the category "SAD with hypersomnia". Certain outcomes and measurement methods were more common within certain diagnostic groups, for instance actigraphy was most commonly used in dementia (Table S6 summarises outcomes and measurement methods per study).

Risk of bias assessment

Many studies attempted participant blinding, but difficulties were understandably common. To counter this some studies assessed the participants' intervention expectations to determine whether this accounted for outcomes. Rates of attrition were highly variable. Allocation methods were sometimes unclearly reported (see Table S7).

Publication bias

Funnels plots were generated for each analysis. None appeared skewed [52] and statistical testing suggested no publication bias [53]. However, due to the small number of studies and limited variation in trial size for many analyses, it is difficult to accurately interpret these plots and tests.

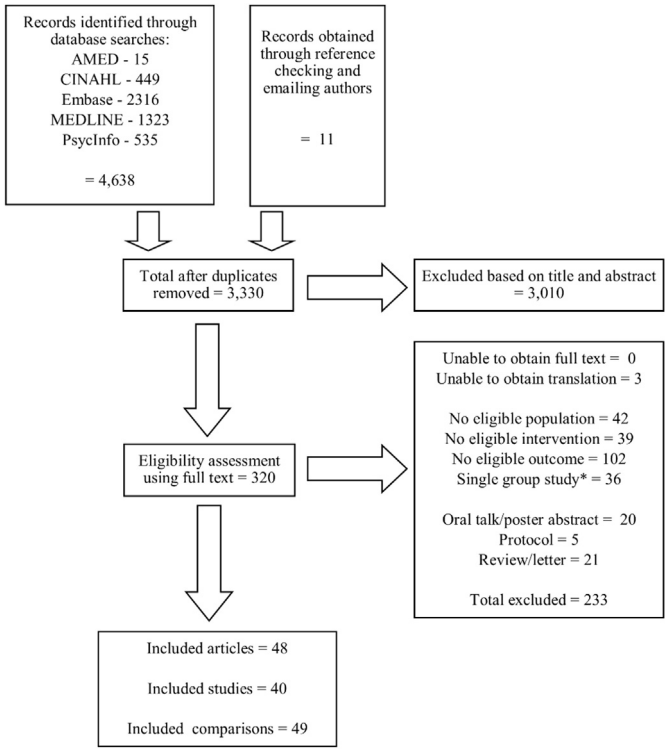

Fig. 1. PRISMA flow diagram for search, screening and inclusion. *Includes controlled trials with the wrong comparison, e.g., both groups gave the same light exposure and modified another component.

Outcomes

The effect of intervention compared to control condition was statistically significant for six outcomes; all in the desirable direction (see Table 3). Effects on sleep timing (in DSPD), self-reported sleep disturbance, and sleep continuity disruption were robust to all sensitivity analyses. Effects on sleep onset latency (SOL), total sleep time (TST), and sleep quality were statistically significant in the main analysis but not in all sensitivity analyses (see supplements S8eS20 for additional forest plots and S21 for sensitivity analyses).

Delaying sleep timing in advanced sleep phase disorder (ASPD)

Samples were middle aged or older, and community based (see Table 2). There were few studies and samples were small. All interventions gave evening light or light during early sleep, none restricted morning light. Effects were homogeneous. All studies measured the first-choice outcome – rise time. The pooled effect was non-significant, and in the wrong direction (toward even earlier sleep) (effect size (ES) = -0.15, p = 0.602). To examine if this suggested *harm*, we made post hoc examination of baseline imbalance and pre-post effect. Intervention groups indeed had earlier average rise time at baseline which persisted after intervention, and there was no significant pre-post effect in either direction in the intervention group (ES = 0.32, p = 0.111).

Advancing sleep timing in delayed sleep phase disorder (DSPD)

All studies in DSPD measured sleep timing. Samples in DSPD were young and community based, exclusion of co-morbidities varied widely. All interventions increased light exposure in the morning, and three involved some instruction to avoid or reduce evening light exposure. Effects were homogeneous (χ^2 = 6.00 (d.f. = 8), p = 0.648, I^2 = 0.0%), with the exception of Langevin et al. (2014) [63] which was an extreme outlier. This study was small (n = 10) with highly selective exclusion criteria compared to others, and used spectacle mounted LEDs rather than a light box, likely increasing 'dose' of light reaching the circadian photoreceptors (including this study increased heterogeneity, χ^2 = 16.67 (d.f. = 9), p = 0.054, I^2 = 46.0%)

The pooled effect was significant, and in the direction intended (earlier sleep timing). Sensitivity analysis removing the extreme outlier reduced the effect size slightly (−0.34 to −0.32), but it remained statistically significant (p = 0.015) (Fig. 2). This effect size can be translated to 25 min earlier sleep timing (using the pooled SD to convert overall SMD back into hours/minutes, whilst excluding SMD of the outlier in which sleep onset was 1.5 h earlier [63]).

Sleep inertia

Only three studies reported on sleep inertia: these were in DSPD (n = 2) and SAD with hypersomnia (n = 1). The summary effect was small and non-significant.

Self-reported daytime sleepiness

Only studies in DSPD reported daytime sleepiness (n = 4), all interventions supplemented morning or pre-awakening light. Effects were heterogeneous (χ^2 = 5.38 (d.f. = 3), p = 0.142,

Table 2
Characteristics of included studies, grouped by diagnosis.

Author(s) & Year	N=	Design	Population (+sleep disturbance)	Age mean (SD)/ (range)	Setting	Country	Intervention(s) * dark highlight text = light reduction component	Light timing selection	Intensity	Spectral properties/ light source	Length (days)	Control condition
Campbell et al., 1993 [54]	16	controlled	ASPD	70.4 (4.85)	at home	UK	• evening light box • watch TV during	via bio-marker	4000 lux	white	12	dim light box
Figueiro et al., 2015 [55]	8	crossover	ASPD	70 (4.5)	at home	USA	• blue flashing light mask in early sleep	via bio-marker	not stated	blue LEDs 480 nm	7	red flashing light mask 640 nm
Lack et al., 2005 [56]	25	controlled	ASPD	51.2 (36–68)	Outpatient	Australia	• evening, 4hrs • Whilst watching TV in the lab	Set time	2500 lux	White	2	<100 lux red light
Palmer et al., 2003 [57]	47	controlled	ASPD	(60–86)	at home	USA	• evening light box 2.5hrs	via sleep-timing	265 lux	white fluorescent	28	dim light box (2 lux)
Ando et al., 1999 [58]	10	controlled	DSPD	33.5 (10.7)	at home	USA	• dawn simulation light mask	via sleep-timing	500 lux	not stated	12	0.1 lux
Cole et al., 2002 [59]	54	RCT	DSPD	25 (6)	at home	USA	• bright-dawn simulation mask • advance sleep timing • avoid naps • avoid evening light	via sleep-timing	2700 lux	white	26	dim red light mask
Geerdink et al., 2016 [60]	39	RCT, quasi-random	DSPD	22.0 (6.3)	at home	Netherlands	• morning blue light pulses • advance sleep timing • avoid light at night	via sleep-timing	2306 melanopic lux	blue (460 nm, 80 nm)	9	amber light, avoid light at night, advance sleep schedule
Gradisar et al., 2011 [51]	40	RCT	DSPD	14.6 (1.0)	at home/ outpatient	Australia	• light box on awakening • advance wake time 30 min day • CBT-i	via sleep-timing	1000 lux	broad-spectrum/ outdoors	56	wait list control
Lack et al., 2007 [61]	18	RCT	DSPD	28.2 (10.6)	at home	Australia	• morning blue light via spectacle mounted LEDs	via sleep-timing	65 μW/cm^2	470 nm (blue)	7	unclear if no treatment or red light
Lack et al., 2007b [62]	16	RCT	DSPD	29 (18–56)	at home	Australia	• light box on awakening • advance wake time	via sleep-timing	2500 lux	incandescent tungsten	7	dim light
Langevin et al., 2014 [63]	10	RCT	DSPD	16.3 (15–18)	at home	Canada	• morning blue light via spectacle mounted LEDs • eliminate light after 9pm	via sleep-timing	2000 lux	400 < X < 750 nm	22	orange light of same brightness
Saxvig et al., 2014 [49,64]	40	RCT	DSPD	20.7 (3.1)	at home	Norway	• morning light box • placebo melatonin • advance rise time 1hr each day	via sleep-timing	10,000lux	not stated	14	morning dim red light, placebo melatonin, advance rise time
							• morning light box • melatonin • advance rise time 1hr each day	via sleep-timing	10,000lux	not stated		
							• morning light box • melatonin • advance rise time (re-randomised for 2nd phase)	via sleep-timing	10,000lux	not stated		no treatment
Avery et al., 1998 [58]	11	RCT	SAD	33.0 (10.0)	at home	USA	• dawn simulation lamp • darken bedroom, block out light, turn off nightlights • avoid daylight before 8am • avoid direct sunlight in the daytime (sunglasses)	set time	250 lux	white	7	red dawn, 1.5hr, 2 lux
Avery et al., 2002 [65]	50	controlled	SAD	37.3 (6.1)	at home	USA	• dawn simulation lamp • wake at 6:00am • darken bedroom • avoid morning light before 8am	set time	250 lux	white	7	dim red dawn

Bogen et al., 2016 [43,66]	57	RCT	Depression	15.4 (1.6)	inpatient	Germany	• morning light box	via MEQ	10,000lux	white	14	dim light (100–150 lux)
Esaki et al., 2017 [67]	20	RCT	Depression (+sleep)	41.6 (7.3)	at home	Japan	• amber glasses 8pm til bed, remove only once in the dark	set time	n/a	n/a	14	clear glasses
Kragh et al., 2017 [44,68]	64	RCT	Depression	38.4 (12)	inpatient	Denmark	• morning light box • wake therapy (total sleep deprivation), in a lighted area	via MEQ	10000 lux	white	63	TAU (medication, exercise, talking therapies)
Lieverse et al., 2011 [69]	89	RCT	Depression	69 (6.6)	at home	Netherlands	• morning light box • adherence monitoring via wearable device	preference	7500 lux	pale blue	21	red light (50 lux)
McEnany et al., 2005 [70]	29	RCT	Depression	37.5 (27–46) 55.9 (45–76)	at home	USA	• light visor 1hr on awakening	via sleep-timing	2500 lux	krypton incandescent	26	light blocking glasses for 1hr before bed
Barbini et al., 2005 [41]	32	controlled	Other MH – bipolar mania	38.2 (8.8)	inpatient	Italy	• enforced darkness 6pm-8am • quiet - noise deadened • staff supervision	set time	n/a	n/a	3	TAU, drug therapy alone
Bromundt et al., 2013 [71]	14	crossover	Other MH – borderline personality disorder	30.1 (6.0)	at home	Switzerland	• morning light box • rising by 9am	preference	8000 lux	not stated	21	TAU, waitlist control
Henriksen et al., 2016 [42]	19	RCT	Other MH – bipolar mania	36.0 (17.3)	inpatient	Norway	• amber glasses 6pm till bed, and before 8am	set time	n/a	n/a	7	clear glasses
Sheaves et al., 2018 [40,72]	40	RCT	Other MH – mixed acute inpatients (+sleep)	40 (13)	inpatient	UK	• morning light box/natural light • adapted CBT-i • dark in evening and at night • activity tracking wearable for motivation and feedback	via MEQ	10,000lux/ outdoors	light box/outdoor light	14	TAU
Sit et al., 2017 [73]	46	RCT	Other MH – bipolar depression	44.7 (14.5)	at home	USA	• mid-day light box; titrated from 15min to 60min per day • adherence monitoring via machine	set time	7,000lux	4,000 K white fluorescent	42	red light box (50 lux)
Ancoli-Israel et al., 2002 [74]	34	RCT	Dementia	85.7 (7.3)	nursing home	USA	• am light box • staff supervision • pm light box • staff supervision	set time set time	2,500 lux 2,500 lux	cool-white cool-white	18	dim light, less than 50 lux, red light
Burns et al., 2009 [75,76]	46	RCT	Dementia (+sleep)	83.6 (7.9)	nursing homes	UK	• am light box • staff supervision	set time	10000 lux	full spectrum	14	100 lux standard fluorescent light
Connell et al., 2007 [77]	20	RCT	Dementia	79.7 (8.3)	nursing home	USA	• daytime natural light • group outdoor activity: social, horticultural, creative, singing • facilitated by nurse	convenience	natural light	outdoor light	14	indoors, similar activities
Dowling et al., 2005 [78,79]	70	RCT (crossover in phase 2)	Dementia (+sleep)	84 [10]	long-term care	USA	• morning outdoor/indoor light • light boxes to supplement when required • afternoon outdoor/indoor light • light boxes to supplement when required • am or pm outdoor/indoor light • light boxes to supplement when required (am/pm groups merged after crossover in phase 2)	set time set time set time	~2,500 lux ~2,500 lux ~2,500 lux	not stated not stated not stated	70	indoor light (150–200lux) usual activities

(*continued on next page*)

Table 2 (*continued*)

Author(s) & Year	N=	Design	Population (+sleep disturbance)	Age mean (SD)/ (range)	Setting	Country	Intervention(s) * dark highlight text = light reduction component	Light timing selection	Intensity	Spectral properties/ light source	Length (days)	Control condition
Dowling et al., 2008 [50]	50	RCT	Dementia (+sleep)	86 (10)	long-term care	USA	• morning outdoor/indoor light • light boxes when required • melatonin	set time	2,500 lux	not stated	70	usual activities placebo melatonin
							• morning outdoor/indoor light • light boxes when required • placebo melatonin	set time	2,500 lux	not stated		
Fontana Gasio et al., 2003 [80]	13	RCT	Dementia (+sleep)	86.8 (4.5)	inpatient	Switzerland	• environmental dawn-dusk simulation, set to participants sleep timing	via sleep-timing	<400 lux	halogen with diffuser	21	dawn-dusk simulation using 15w red bulb
Friedman et al., 2012 [45]	54	RCT	Dementia	77.9 (8.1)	at home	USA	• morning light box • read or watch TV, during • sleep hygiene info • compliance aid and explanation	via sleep-timing	~4,200lux	full spectrum	14	dim red light (filter added to same light)
Lyketsos et al., 1999 [81]	15	RCT crossover	Dementia	80.8 (8.7)	residential care	USA	• morning bright light box • staff supervision	convenience	10,000lux	full spectrum	28	dim blinking light
McCurry et al., 2005 [46]	36	RCT	Dementia (+sleep)	77.8 (8.1)	at home	USA	• evening natural light/light box • sleep hygiene • regularise sleep times • reduce naps • daily walking (30min) • reduce light at night	convenience	2,500 lux	fluorescent	28	dementia education and carer support
McCurry et al., 2011 [47]	70	RCT	Dementia (+sleep)	82.2 (8.5)	at home	USA	• evening light box • sleep hygiene leaflet • reduce light at night	convenience	2,500 lux	full spectrum	56	sleep hygiene leaflet
							• evening light box • natural light • walking (30 min) • sleep hygiene leaflet • reduce light at night	convenience	natural light	full spectrum		
Mishima et al., 1998 [82]	22	RCT crossover	Dementia (+sleep)	81 & 78	inpatient	Japan	• light box 9–11am • carer supervision	set time	5000–8000 lux	full spectrum	14	dim light (300 lux)
Nowak et al., 2008 [83,84]	20	RCT	Dementia	85.9 (6.24)	nursing home	USA	• blue green light cap visor	set time	12,000lux	blue-green	14	dim red light cap visor
Ouslander et al., 2006 [85]	173	cluster crossover study	Dementia	83.2 (9.0)	nursing home	USA	• evening light box 2hrs • activity and exercise • avoid naps • regularise sleep times • reduce noise and continence related disruptions at night	convenience	1,467lux	full spectrum	17	TAU/waitlist control
Sloane et al., 2007 [86]	60	cluster crossover	Dementia	<65 = 6 65–79 = 28 >80 = 32	long-term care	USA	• indoor light 7am-11am • indoor light 4pm-8pm • all day indoor light 7am-8pm	set time set time set time	~2500 lux ~2500 lux ~2500 lux	not stated not stated not stated	21	standard indoor light (~>500 lux)
Sloane et al., 2015 [48]	17	RCT crossover	Dementia	65–79 = 6 >80 = 11	at home	USA	• all day indoor artificial light	preference	13,000 K	blue white/470 nm	42	daytime 2700 K (yellow-white) lamps & red light box
Van Someren et al., 1999 [87,88]	22	repeated measures crossover	Dementia	79 (9.4)	long-term care	Netherlands	• day time indoor artificial light	convenience	790–2190lux	not stated	28	TAU

*N = number of participants included in meta-analyses, RCT = randomised controlled trial, n/a = not applicable, TAU = treatment as usual, MH = mental health conditions, ASPD = advanced sleep phase disorder, DSPD = delayed sleep phase disorder, SAD = seasonal affective disorder, CBT-i = cognitive behavioural therapy for insomnia.

I^2 = 44.2%); mostly attributable to the same outlier as above [63] (after its removal, χ^2 = 1.40, (d.f. = 2), p = 0.496, I^2 = 0.0%). The effect was non-significant (ES = −0.34, p = 0.092).

Sleep onset latency (SOL)

Six studies reported SOL. Samples were community based. Age varied and depression samples were older than DSPD samples. Most interventions were similar, giving 30–60 min light box in the morning. Esaki et al. (2017) instead reduced evening blue light exposure using amber glasses [67]. Gradisar et al. (2011) included CBT-i and light [51], and Saxvig et al. (2014) gave melatonin in some groups [49]. Effects were heterogeneous (χ^2 = 15.51 (d.f. = 7), p = 0.030, I^2 = 54.9%).

The pooled effect was significant (p = 0.033), with ES -0.27; equivalent to 5.5 min shorter SOL. Significance was altered by sensitivity analysis excluding assumed (not diagnosed) DSPD (p = 0.078) (see Table S21). Sub-group analysis suggested larger effects in DSPD (ES = −0.46, p = 0.006), than in depression (ES = −0.01, p = 0.946).

Sleep quality

Self-reported sleep quality (visual analogue scale; VAS) was measured in DSPD and depression. Most samples were young. Most interventions were morning or pre-awakening light, some included nighttime light avoidance. One intervention instead used amber glasses in the evenings [67].

Effects were heterogeneous, even within diagnostic groups (χ^2 = 12.62 (d.f. = 6) p = 0.049, I^2 = 52.5%). Heterogeneity was reduced by removing the same outlier [63] (χ^2 = 5.50 (d.f. = 5) p = 0.358, I^2 = 9.0%). The pooled ES of 0.28 toward improved sleep quality was statistically significant (p = 0.046), but was altered by sensitivity analyses (ES 0.18 to 0.38, p = 0.013 to 0.266). The ES of 0.28 translates to 0.25 points improvement on a 5-point Likert scale (using the pooled SD to convert overall SMD back to VAS).

Self-reported sleep disturbance (composite measures)

Composite measures of self-reported sleep disturbance (e.g., insomnia severity index (ISI)) were used in DSPD, inpatient mental illness, and bipolar depression (inpatients). Most interventions gave early morning bright light (>2500 lux), except Sit et al. (2017) gave early afternoon bright light [73]. Saxvig et al. (2014) added melatonin in some groups [49], and Sheaves et al. (2018) also gave CBT-i [89]. Effects were homogeneous (χ^2 = 5.15 (d.f. = 6), p = 0.525, I^2 = 0.0%).

The pooled effect was an ES of −0.32 (lower sleep disturbance) (p = 0.015) (Fig. 3), which was robust to all sensitivity analyses, and can be translated to a 1.26 points reduction in ISI score.

Total sleep time (TST)

TST was measured in most studies. Results were heterogeneous across diagnostic groups (χ^2 = 68.21 (d.f. = 33), p = 0.000, I^2 = 51.6%), and within all groups except dementia (χ^2 = 4.34, p = 0.996, I-squared = 0.0%) and depression (χ^2 = 0.89, p = 0.642, I^2 = 0.0%). The populations with largest ES were most heterogeneous. Heterogeneity remained after removal of two extreme positive outliers [41,63]. There was a small ES of 0.15 toward longer sleep (p = 0.015) (Fig. 4). Sensitivity analyses removing positive outliers reduced significance (p = 0.097), and as did removing studies in samples without sleep disturbance inclusion criteria (perhaps counterintuitively) (p = 0.209). Removing studies at high risk of bias increased the ES (=0.20) and significance (p = 0.002). Additional post hoc sensitivity analysis excluding studies with longer average TSTs at baseline had minimal effect on results (adequate examination of the impact of baseline TST would require individual patient data).

Sleep efficiency (SE%)

All diagnostic groupings except 'other mental health conditions' measured SE%. Effect sizes were relatively homogenous despite clinical heterogeneity in intervention and population characteristics (χ^2 = 18.98 (d.f. = 18) p = 0.393, I^2 = 5.1%). The pooled effect was small and non-significant (ES = 0.14, p = 0.063). Sensitivity analyses affected the overall result slightly as statistical significance varied (ES = 0.10 to 0.15, p = 0.045 to 0.330).

Sleep continuity disruption

Sleep continuity disruption was measured in many diagnostic groups. Light intensity and duration varied, one study used amber glasses [42]. Results were heterogenous between groups, whilst within the dementia sub-group results were homogeneous (χ^2 = 10.59 (d.f. = 17) p = 0.877, I^2 = 0.0%).

The pooled ES of −0.23 was highly statistically significant (p = 0.000) (reduced sleep disruption), this translated to 6.1 min less wake time after sleep onset, or 1.44 less awakenings per night (using pooled SD to convert back from SMD). ES varied significantly between diagnostic groups (see section below), with larger ES in mental illness and ASPD (−1.45, −0.75 and −0.50), and homogeneous, small, non-significant effects in dementia (ES = −0.12, p = 0.089) (Fig. 5). Sensitivity analyses made little difference to results (ES = −0.20 to −0.27, p = <0.000 to p = 0.038).

Rhythmicity of rest activity rhythm

Rhythmicity was only measured in dementia. Light administration, timing, intensity and duration varied; none reduced light as the primary intervention. Rhythmicity was always derived from actigraphy, but algorithms varied including non-parametric and parametric approaches (extended cosine model). Results were reasonably homogenous (χ^2 = 9.54 (d.f. = 9), p = 0.389, I^2 = 5.6%). The pooled effect was non-significant (p = 0.585) and close to the line of no effect (ES = −0.06).

Amplitude of rest activity rhythm

Amplitude was only measured in dementia. Metrics varied; relative amplitude was most common, but other metrics such as percentage of activity in night-time were also reported. Results were not significantly heterogeneous (χ^2 = 16.94 (d.f. = 15) p = 0.323, I^2 = 11.4%), nor was the effect significant (ES = 0.03, p = 0.644).

Carer reported daytime sleep propensity

Self-reported sleepiness was not available for samples with dementia, so carer reported daytime sleep propensity was examined (assessed through behavioural observations suggesting sleepiness or nodding off). Heterogeneity was low (χ^2 = 15.01 (d.f. = 13) p = 0.307, I^2 = 13.4%) and the pooled effect non-significant (ES = −0.13, p = 0.096) despite the large number of comparisons (n = 13) and participants (438/299) included.

Table 3
Effect of interventions altering light exposure patterns on sleep outcomes.

Outcome	Included			Effect				Heterogeneity	
	N_s	N_c	n (Ix/Cx)	Direction of effect with Ix	SMD (g)	95% CI	p=	I^2%	p=
Sleep timing in ASPD	4	4	52/52	Earlier (undesirable) (n.s)	−0.15	−0.54; 0.23	0.439	0.0	0.602
Sleep timing in DSPD	8	10	137/118	**Earlier (desirable)**	**−0.34**	−0.60, −0.08	**0.010**	46.0	0.054
Sleep inertia	3	3	74/69	Less sleep inertia (n.s)	−0.26	−0.59, 0.08	0.132	37.9	0.200
Self-reported daytime sleepiness	4	4	55/49	Less daytime sleepiness (n.s)	−0.34	−0.74, 0.06	0.092	44.2	0.146
Sleep onset latency (SOL)	6	8	138/131	**Shorter SOL**	**−0.27**	−0.52, −0.02	**0.033**	54.9	0.030
Sleep quality (on VAS)	5	7	113/107	**Higher sleep quality**	**0.28**	0.00, 0.55	**0.046**	52.5	0.049
Self-reported sleep disturbance (questionnaire)	5	7	119/116	**Less sleep disturbance**	**−0.32**	−0.58, −0.06	**0.015**	0.0	0.525
Total sleep time (TST)	27	34	703/492	**Longer TST**	**0.15**	0.03, 0.27	**0.015**	57.6	0.000
Sleep Efficiency (SE%)	15	19	427/364	Higher SE% (n.s)	0.14	−0.01, 0.28	0.063	5.1	0.393
Sleep continuity disruption	20	24	672/509	**Less sleep disruption**	**−0.23**	−0.36, −0.10	**0.000**	30.9	0.125
Rhythmicity of rest activity rhythm	6	10	252/123	Lower rhythmicity (n.s.)	−0.06	−0.28, 0.16	0.585	5.6	0.389
Amplitude of rest activity rhythm	11	16	463/314	Higher amplitude (n.s)	0.03	−0.11, 0.18	0.644	11.4	0.323
Carer reported daytime sleep propensity	10	14	439/299	Less daytime sleepiness (n.s)	−0.13	−0.28, 0.02	0.096	13.4	0.307

N_s = number of studies, N_c = number of comparisons, n (Ix/Cx) = number of participants in combined intervention and control groups, n.s. = not significant, ASPD = advanced sleep phase disorder, DSPD = delayed sleep phase disorder. Bold text = statistically significant effect on outcome.

Follow up points

Few studies presented data for follow-up points. Studies varied in terms of inclusion of maintenance therapy or advice, and many ceased intervention completely, so these results could not be synthesised.

Differences between outcomes depending on population

Results for sub-groups are presented in Table 4. CIs for outcomes overlapped substantially in all cases except TST and sleep continuity. The overall test for heterogeneity between sub-groups was likely to be invalid due to within group heterogeneity. Where CIs did not overlap, or only just overlapped, we tested for differences between subgroups using the method described in Bornstein et al. (2009), and found no statistically significant differences.

Intervention effect moderators

There were too few studies to run meta-regressions for some outcomes, and sometimes planned covariates varied insufficiently or were too confounded with other features. Insufficient reporting of season prevented meaningful examination of latitude or season (without season, latitude does not describe baseline light environment). Features related to intervention 'dose' (intensity, duration) were mostly well reported, and varied, allowing meta-regression; but we found no significant associations between 'dose' features and effects (see Table S22). Spectral composition of light, or bulb type was reported too infrequently to permit analysis (see supplement S1). Variability in effects based on intervention type (e.g., light box, natural light, light restriction) was examined using sub-group analysis, results are presented in supplements S23–34. Due to small numbers and many confounders further interpretation was resisted. Pm light exposure (versus am) was associated with a greater reduction in carer reported daytime sleep propensity in dementia (Coefficient = −0.5007139, p = 0.019). Interventions with a sleep schedule component were also associated with less deterioration of rest activity rhythm amplitude in dementia (Coefficient = 0.4587271, p = 0.010).

The strongest association was between interventions including light avoidance/reduction, and greater increase in TST (Coefficient = 0.6477418, p = 0.004). The association was greatest when light was avoided not just at night but from afternoon or evening (Coefficient = 1.48552, p = 0.000) (Fig. 6). On other outcomes, interventions including light avoidance also performed

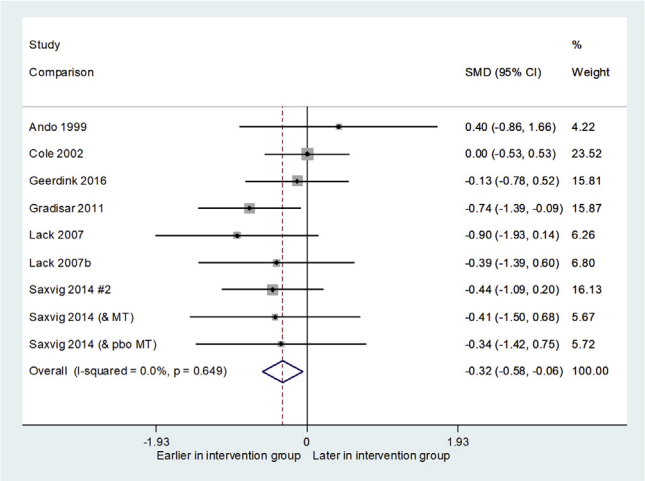

Fig. 2. Effect of morning light interventions on sleep timing in DSPD.

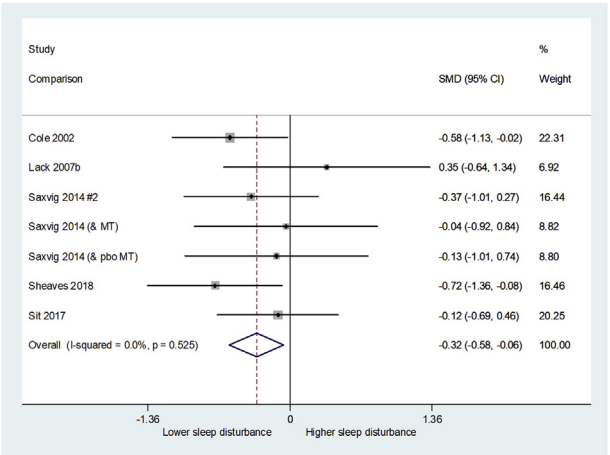

Fig. 3. Effect of light schedule interventions on self-reported sleep disturbance.

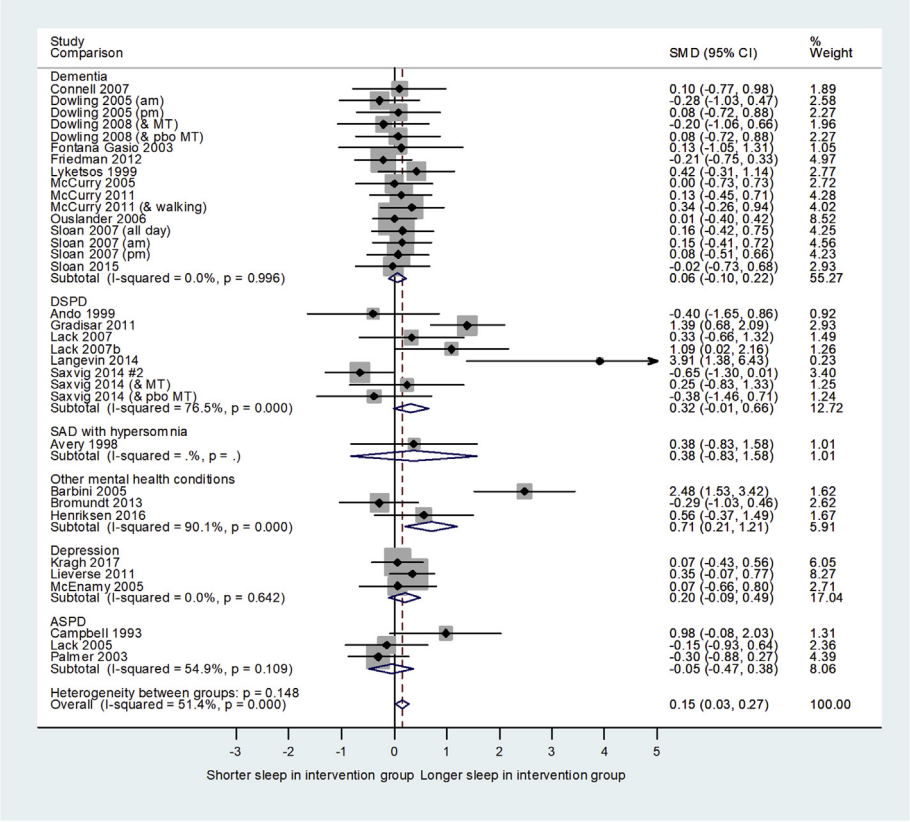

Fig. 4. Effect of light schedule intervention on total sleep time (TST).

better but associations were non-significant. To examine whether differences in effects on TST were population related, meta-regressions were re-run excluding groups where no interventions involved light reduction; associations remained highly significant (Coefficient = 0.6944778, p = 0.006, and, Coefficient = 1.69882, p = 0.009).

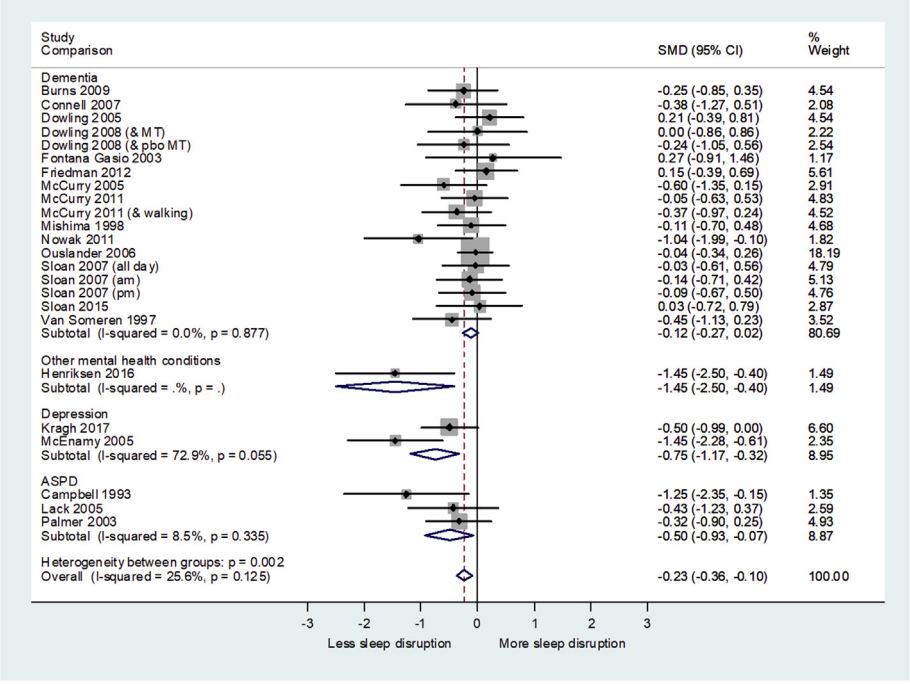

Fig. 5. Effect of light schedule interventions on sleep continuity disruption.

Table 4
Intervention effects in diagnostic sub-groups.

	Outcome (only listing those relevant and with data)	Studies reporting/total studies	SMD (g)	95% CI	p=
ASPD	Sleep timing	4/4	−0.15[a]	−0.54, 0.23	0.439
	TST	3/4	−0.05[a]	−0.47, 0.38	0.823
	SE%	3/4	−0.02[a]	−0.47, 0.43	0.929
	Sleep continuity disruption	3/4	−0.50	−0.93, −0.07	0.023
	Rhythmicity of rest activity	0/4			
DSPD	Sleep timing	8/8	−0.34	−0.60, −0.08	0.010
	Sleep inertia	2/8	−0.04	−0.45, 0.37	0.845
	Self-reported daytime sleepiness	4/8	−0.34	−0.74, 0.06	0.092
	SOL	4/8	−0.46	−0.79, −0.13	0.006
	Sleep quality (on VAS)	3/8	0.21	−0.13, 0.55	0.217
	Self-reported sleep disturbance	4/8	−0.29	−0.61, 0.04	0.087
	TST	6/8	0.31	−0.03, 0.65	0.070
	SE%	2/8	−0.07[a]	−0.47, 0.32	0.720
	Rhythmicity of rest activity	0/4			
Depression	SOL	2/5	−0.01	−0.39, 0.36	0.946
	Sleep quality (on VAS)	2/5	0.39	−0.07, 0.84	0.094
	TST	3/5	0.20	−0.09, 0.49	0.174
	SE%	2/5	0.27	−0.09, 0.64	0.139
	Sleep continuity disruption	3/5	−0.75	−1.17, −0.32	0.001
SAD with hypersomnia	Sleep inertia	1/2	−0.68	−1.28, −0.11	0.02
	TST	1/2	0.38[a]	−0.83, 1.58	0.541
Other mental health conditions	Self-reported sleep disturbance	2/4	−0.39	−0.82, 0.04	0.078
	TST	2/4	0.71	0.21, 1.21	0.005
	Sleep continuity disruption	1/4	−1.45	−2.50, −0.40	0.007
Dementia	TST	10/16	0.06	−0.10, 0.22	0.460
	SE%	7/16	0.17	−0.01, 0.36	0.066
	Sleep continuity disruption	14/16	−0.12	−0.27, 0.02	0.089
	Rhythmicity of rest activity	6/16	−0.06[a]	−0.28, 0.16	0.585
	Amplitude of rest activity rhythm	11/16	0.03	−0.11, 0.18	0.644
	Carer report daytime sleep propensity	6/16	−0.13	−0.28, 0.02	0.096

TST = total sleep time, SE% = sleep efficiency percentage, SOL = sleep onset latency, VAS = visual analogue scale, DSPD = delayed sleep phase disorder, ASPD = advanced sleep phase disorder.
[a] Opposite direction to desired effect.

Discussion

Summary of findings

We found studies of light schedule interventions to improve sleep in a range of populations, but there were relatively few studies in samples with mental illnesses which targeted and measured sleep and circadian disturbances. This despite the fact that circadian dysregulation is acknowledged to be important in this group [36]. Many studies in mental illness were excluded because they reported no sleep outcomes, and when present sleep outcomes were usually secondary. Interventions predominantly involved a circumscribed period of light exposure, most commonly from a light box at a set time each day. Some interventions were more complex and included modifications to both morning and evening light levels, personalisation of light schedules, or light from multiple sources.

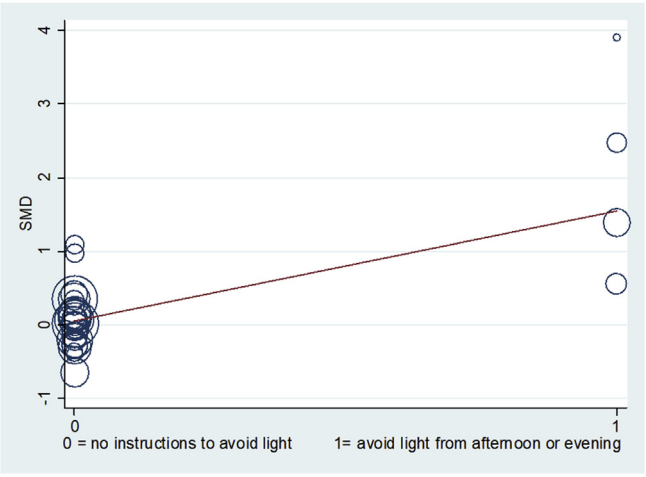

Fig. 6. Bubble plot - light avoidance from afternoon or evening associated with greater increase in total sleep time.

Overall, the effects of light schedule interventions on sleep outcomes were positive. We found evidence of small but statistically and clinically significant improvements in sleep continuity disruption, self-reported sleep disturbance, and sleep timing in DSPD, that were robust to sensitivity analyses. We found less conclusive evidence of improvements in SE%, SOL, and self-reported sleep quality; and a small increase in TST. These findings are broadly in agreement with previous meta-analytic findings where different sleep outcome types were pooled, and where study inclusion required diagnosis or specification of sleep problems at baseline [30]. The current review contributes a synthesis of evidence in psychiatric diagnoses, and separate analysis of various sleep and rest-activity outcomes.

Effects in sleep phase disorders

Basic research has found reasonably consistent effects of light interventions on circadian phase, the direction and magnitude of which depend on timing, intensity, spectral properties, and duration of light exposure [90–92]. Our findings confirm that the phase advancing effects of morning light established in laboratory settings can be replicated to some degree in the home in DSPD. This finding builds on previous syntheses in DSPD where reported effects were also positive but less conclusive [29]. We found a shortening of SOL, and an average advance in sleep timing of 25 min, suggesting morning light intervention may be sufficient

for some participants, whilst others will require some additional intervention. Basic research suggests that increasing morning light for phase advance, or evening light for phase delay, should be more effective if light is also avoided during the opposite period (morning/evening) [93]. Whilst across all studies those including light avoidance gave greater increase in TST, we were not able to definitely establish superiority of effects for adding evening light avoidance to morning light exposure from the studies in DSPD.

Our findings did not support increased evening light exposure to phase delay sleep in ASPD, and no studies in ASPD included morning light avoidance, but the total number of participants in this analysis was small.

Effects on sleep continuity and sleep disturbance

Light exposure interventions improved sleep continuity, self-reported sleep disturbance, and sleep quality (although the latter was affected by removing an outlier, and by removing studies without sleep inclusion in sensitivity analysis). This is consistent with findings in shift work: attempts to sleep out of phase with circadian rhythm are understood to incur increased disruptions [94], whilst increasing circadian alignment can improve sleep quality [95]. Sleep continuity should be considered as a potential outcome in more studies, because it is important to patients [13], has been linked to next-day symptoms [96], may be amenable to change, and can be assessed with actigraphy or self-report.

Effect on total sleep time (TST)

The most commonly reported parameter was TST (27/40 studies). Our primary analysis showed a small average effect toward longer sleep (ES = 0.15, p = 0.015). This effect size is similar to the effect sizes found by Van Maanen et al. (2016) (effect of light therapy on 'all sleep problems' and 'other sleep problems'). Changes to mean TST are difficult to interpret without individual patient data, or sub-grouping by baseline sleep duration. In many diagnostic groups, an individual's sleep may be short, long, or already of normal (optimal) duration but poor quality, thus the desirable direction of change varies. Chiu et al. (2018) stratified participants with schizophrenia and other psychoses by sleep duration at baseline (<6hrs, 6–10hrs, >10hrs) and found that intervention with CBT-i (compared to control condition) increased TST in those with short sleep and reduced TST in those with long sleep (both desirable), the mean value however obscured both effects. We recommend that studies of light schedule interventions consider stratifying participants by baseline TST, and that a future review on this topic might examine this outcome using individual patient data.

Effect on sleep efficiency (SE%)

Another commonly reported but difficult-to-interpret metric is SE%; SE% is influenced by SOL, sleep fragmentation, and unwanted early awakening. Inconsistencies in how SE% is calculated are common, and persist despite efforts at standardisation [97,98]. Furthermore, although higher SE% is an accepted intervention target in insomnia studies, it is not clear if very high SE% (>90%) should always be viewed positively. Very high SE% (mean 96.6%) was found to be a feature of sub-type "insomnia with hypersomnia" in people with psychotic illnesses, who had excessively long sleep and a subjective complaint of sleeping poorly [21].

Baseline light exposure patterns as an effect moderator

We hypothesised that if baseline light exposure patterns were already close to optimal, that alterations during intervention may make less of a difference. We were not able to examine this due to sparse reporting. Future studies should report season or months of intervention in order that future syntheses may examine whether season and latitude moderate intervention effects. Some studies in institutions examined baseline light levels in the environment, but none examined individuals' dynamic light exposure at baseline. It is possible that variability of baseline light exposure accounts for some heterogeneity of effects.

The signal received from daily light exposure patterns can be described as high or low amplitude; high amplitude being bright days and dark nights, and low amplitude being quite similar light levels throughout. The modern light environment for many individuals involves rather dimly lit days spent indoors, artificially elongated days with artificial light in the evening, and darkness only when it is time to sleep. Modelling describes how this low amplitude signal with evening light, delays and de-stabilises circadian rhythms [99]. By contrast reverting to a more natural light-dark signal was found to improve circadian rhythms and sleep [2]. This research suggests that normalising light schedules by reducing artificial evening light would improve sleep, our findings agree with this, and suggest interventions were more effective if they included evening light avoidance/reduction.

Studies in dementia compared to in other populations

There were distinct differences in approaches to intervention and outcome selection between studies in dementia and in other conditions. For instance, studies in dementia almost always used actigraphy, were more likely to specify some sleep disturbance inclusion criteria, and often set sleep as a higher priority amongst their stated aims. Only studies in dementia modified the living environment to alter overall daytime light levels, and more often used natural light, or light from a range of sources. Despite more studies focussing on sleep, intervention effects in dementia were notably smaller and less significant than in other groups (and similar to in a previous meta-analysis [28]). Caution is advised interpreting sub-group analyses of differences in treatment effects based on sample characteristics [38]; associations are observational and other factors may explain differences. The use of parametric circadian analysis may have contributed to poor measurement, as non-parametric methods have since been shown to detect change better [88]. It is also possible that interventions were insufficiently personalised, as interventions were commonly applied to whole institutions.

Studies in mental illness

Although twelve studies in mental illness were included, each only contributed a small amount of data, reporting few sleep outcomes, usually with sleep as a secondary aim. Sleep outcomes are often neglected in mental health intervention research; for instance studies in bipolar disorder seldom measure sleep, despite these interventions often containing components targeting sleep [100]. Though it was not our aim to compare effects between diagnostic groups, some of the largest effects were seen in mental illness samples (such as sleep continuity disruption in depression, ES = −0.75, TST in other mental health conditions, ES = 0.71). This suggests that light schedule interventions which have previously focused primarily on affective symptoms might consider also targeting and measuring sleep outcomes.

Research links a more regular diurnal rhythm to better mental wellbeing and functioning [11]. A regular rhythm is also described as important by people with mental health conditions, in order to support social integration, functioning, and wellbeing [13,101,102]. We had therefore identified regularity of rhythm as a relevant outcome in these groups, and planned to examine effects on regularity and amplitude, but these metrics were only reported in

dementia (see Table S6). We recommend that future studies in mental illness measure rest-activity timing and rhythm, as well as sleep quality and amount.

Co-morbid circadian dysregulation

Relatively few studies in mental illness were included despite broadening criteria to capture more studies. Without broadening criteria only 4/12 studies in mental illness, and 7/16 in dementia would have been included, as only these specified sleep inclusion criteria. Interpretation of the current review is therefore contingent on the assumption that sleep problems are prevalent enough in the included neuropsychiatric populations that sleep disruption criteria would not have changed the samples too greatly. Although sensitivity analyses found no consistent or marked difference between studies with or without sleep inclusion criteria, the relevance of studies without them can of course be questioned, and there may be a difference in effect which we were unable to detect. Furthermore, sleep problems in our included neuropsychiatric samples would most likely also have had non-circadian causes: anxiety and arousal processes of insomnia, environmental interruptions, and the impact of affective or psychotic symptoms. Better assessment and description of the different types and causes of sleep disturbance in populations with neuropsychiatric disorders would allow more targeted intervention using the most relevant components. Although more reporting of secondary sleep outcomes in mental health interventions is welcome the field needs more intervention trials targeting specific types of sleep disturbance and circadian dysregulation as primary outcomes.

Conclusions

This review highlights promising initial findings regarding the effects of altering patterns of light exposure on sleep in groups experiencing, or potentially experiencing, circadian dysregulation. This is despite some intervention protocols not appearing to be optimised according to current theoretical understanding, and despite only a few studies selecting participants specifically for presence of relevant sleep and circadian problems. Our findings suggest that small but clinically meaningful improvements in some sleep parameters can be achieved through altering light exposure patterns in some groups. To achieve greater effects, light exposure interventions may be further optimised and better targeted to particular types of sleep and circadian problems, and appropriately combined with other behavioural elements.

Practice points

- Our findings support the use of morning light exposure to advance sleep timing and hasten sleep onset in delayed sleep phase disorder; average effects are small so in many cases other intervention components may be required in addition.
- Interventions altering light exposure may be helpful for improving sleep continuity or sleep disturbance in groups with circadian dysregulation; appropriate light schedule alterations will depend upon the group.
- Enhancing evening darkness to promote sleep may be useful; evidence is as yet weak but side effects are few (as long as risk of falls is mitigated).

Research agenda

- Studies in psychiatric populations should consider targeting sleep outcomes, as findings are promising but research is sparse.
- Studies should consider altering light-dark exposure patterns over the whole day or during multiple periods.
- There should be mores studies of evening light avoidance/reduction.
- Controlled studies aiming to phase advance or phase delay sleep timing should compare the effects of using timed light exposure and timed light avoidance, with 'single component' light exposure or light avoidance.
- In groups where sleep and circadian rhythm problems are diverse, samples should be stratified by type of sleep problem, particularly where the desired effects differ (e.g., delayed versus advanced sleep, short versus long sleep).
- Studies should report season of intervention delivery; and may consider examining individual participant's baseline light exposure patterns.

Author's contributions

The review protocol was designed by SF with supervisory input from DJD, PB & RD. Search, screening, data extraction and data analysis were conducted by SF. Risk of bias assessment was conducted by SF and NM. The manuscript was drafted by SF. DJD, PB, RD and NM gave input regarding structure, content and style.

Funding

This report is independent research arising from a Clinical Doctoral Research Fellowship, awarded to Sophie Faulkner (NIHR award identifier: ICA-CDRF-2016-02-007) supported by the National Institute for Health Research and Health Education England. The views expressed in this publication are those of the authors and not necessarily those of the NHS, the National Institute for Health Research, Health Education England or the Department of Health.

Conflicts of interest

The authors do not have any conflicts of interest to disclose.

Acknowledgements

Olivia Schaff is acknowledged for second screening of titles and abstracts. Alexandra Berry is acknowledged for second eligibility assessment of full text articles. Silke Conen, Marlena Price-Williams, Julia Mueller and Yuya Mizuno are acknowledged for translation and screening of foreign language articles. Natalka Szyrokyi is acknowledged for cross-checking of data extraction. Gill Norman, Sarah Rhodes & Maggie Westby are acknowledged for advice regarding analysis and use of statistical software.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.smrv.2019.04.012.

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