

Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia (Review)

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[Intervention Review]

Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

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ABSTRACT

Background

Rest-activity and sleep-wake cycles are controlled by the endogenous circadian rhythm generated by the suprachiasmatic nuclei (SCN) of the hypothalamus. Degenerative changes in the SCN appear to be a biological basis for circadian disturbances in people with dementia, and might be reversed by stimulation of the SCN by light.

Objectives

The review assesses the evidence of effectiveness of light therapy in managing cognitive, sleep, functional, behavioural, or psychiatric disturbances associated with dementia.

Search methods

The Specialized Register of the Cochrane Dementia and Cognitive Improvement Group (CDCIG), *The Cochrane Library*, MEDLINE, EMBASE, PsycINFO, CINAHL and LILACS were searched on 4 March 2008 using the terms: “bright light*”, “light box*”, “light visor*”, “dawn-dusk*”, phototherapy, “photo therapy”, “light therapy” “light treatment”, light* . The CDCIG Specialized Register contains records from all major health care databases (*The Cochrane Library*, MEDLINE, EMBASE, PsycINFO, CINAHL, LILACS) as well as from many trials databases and grey literature sources.

Selection criteria

All relevant, randomized clinical trials in which light therapy, at any intensity and duration, was compared with a control group for the effect on managing cognition, sleep, function, behavioural, or psychiatric disturbances (as well as changes in institutionalization rates or cost of care) in people with dementia of any type and degree of severity.

Data collection and analysis

Three reviewers independently assessed the retrieved articles for relevance and methodological quality, and extracted data from the selected studies. Statistically significant differences in outcomes between the treatment and control groups at end of treatment and follow-up were examined. Each study was summarized using a measure of effect (e.g. mean difference).

Main results

Eight trials met the inclusion criteria. However, three of the studies could not be included in the analyses because of inappropriate reported study analyses or inability to retrieve the required data from the investigators. This review revealed no adequate evidence of the effectiveness of light therapy in managing cognition, sleep, function, behaviour, or psychiatric disturbances associated with dementia.

Authors' conclusions

There is insufficient evidence to assess the value of light therapy for people with dementia. Most of the available studies are not of high methodological quality and further research is required.

PLAIN LANGUAGE SUMMARY

There is insufficient evidence to determine whether light therapy is effective in the management of cognitive, sleep, functional, behavioural or psychiatric disturbances in dementia

Rest-activity and sleep-wake cycles are controlled by the endogenous circadian rhythm generated by the suprachiasmatic nuclei (SCN) of the hypothalamus. Degenerative changes in the SCN appear to be a biological basis for circadian disturbances in people with dementia, and might be reversed by stimulation of the SCN by light. The light sources in the included studies were: a light box placed approximately one metre away from the participants at a height within their visual fields; a light visor worn on their heads; ceiling mounted light fixtures; or dawn-dusk simulation that mimics outdoor twilight transitions. Eight studies met the inclusion criteria. However, three trials were not included in the analyses because of inappropriately reported analyses or inability to retrieve the required data from the original investigators. The studies included in the analyses revealed no adequate evidence of the effectiveness of light therapy in managing cognitive, sleep, functional, behavioural, or psychiatric disturbances associated with dementia.

BACKGROUND

Description of the condition

Dementia is defined as acquired impairment in short- and long-term memory, associated with impairment in abstract thinking, judgement, and other disturbances of higher cortical function, or personality changes (APA 1995; McKhann, 1984). This definition of dementia is the most widely used in practice (Robillard 2007). According to the Global Burden of Disease estimates prepared by the World Health Organization (WHO 2003), the disability weight for dementia was higher than for any other condition except spinal cord injury and terminal cancer. The prevalence of dementia increases with age, from 8% of people aged 65 and over to 34.5% over the age of 85 years (CSHA 2000). Alzheimer's disease is the most common cause, accounting for 64% of all individuals with dementia (NACA 1999). As world populations age, and specifi-

cally as the "baby boomers" reach old age, the number of people affected by dementia could triple by the year 2031 (NACA 1999). Advanced dementia results in severe cognitive impairment, functional disability, behavioural disturbances, and total dependence on caregivers (Herrmann 2007). All of these symptoms reduce the quality of life of the individual with dementia, while sleep disruptions and behavioural disturbances also contribute to the burden on family and formal caregivers. The stress that such disturbances place on family caregivers is an important factor in the decision to institutionalize their family member with dementia (Ancoli-Israel 1994; Gallager-Thompson 1992; Pollak 1991; Strang 2006). In addition, there are cost implications for persons with dementia, their family caregivers and health care systems (Hux 1998).

Description of the intervention

The light sources were usually a light box placed approximately one metre away from the participants at a height within their visual fields; a light visor worn on their heads; ceiling mounted light fixtures; or 'naturalistic' light therapy, known as dawn-dusk simulation, that mimics outdoor twilight transitions. Even if light therapy is efficacious, the minimum and optimum intensities and durations of light therapy that best manage disturbances of cognition, sleep, function, behaviour, or psychiatric changes associated with dementia are unknown. While organizations such as the American Academy of Sleep Medicine have drawn up practice recommendations in a number of areas of sleep medicine, there are currently no practice recommendations with regard to people with dementia known to the authors.

How the intervention might work

With normal ageing, there is functional deterioration of the SCN and circadian rhythms are phase-advanced and decreased in amplitude, leading to an altered timing of nocturnal sleep (Campbell 1998; van Someren 1993). More than 50% of people aged 65 years and over experience sleep changes such as fragmented nocturnal sleep, multiple and prolonged awakenings in the second half of the night, and increased daytime napping (Campbell 1988). These abnormalities appear to be even more pronounced in elderly people with Alzheimer's disease (McCurry 2000). In a comparison with healthy elderly people, Satlin 1991 reported lower amplitude and delayed acrophase (time of peak daily activity) of the circadian rhythm in individuals with Alzheimer's disease. Other evidence of disordered circadian rhythmicity in individuals with Alzheimer's disease emerges from studies of rhythms of sleep and endocrine secretion (Ancoli-Israel 1997; Prinz 1982; Touitou 1982). Neuropathological studies have noted loss of vasopressin-secreting neurons in the SCN of the hypothalamus (Liu 2000; Swaab 1985). Vasopressin is one of the major neuropeptides in the SCN and is involved in the synchronization of the circadian rhythm. However, Liu 2000 emphasizes that the loss of vasopressin-secreting neurons in the SCN does not necessarily mean that the neurons have died; they may still be present but inactive. The disorder of circadian rhythmicity common in non-Alzheimer's dementias is also likely to be due to deterioration of the SCN.

Reactivation of SCN cells was shown to be possible in studies of aged rats. These studies revealed that exposure to bright light appeared to reverse age-associated disturbances of circadian sleep-wake rhythm (Witting 1993) and to prevent the age-associated decrease in the number of vasopressin-secreting neurons in the SCN (Lucassen 1995). In humans the neurons in the SCN decrease during normal ageing and even more so in individuals with dementia. As in the studies of aged rats, stimulation with light may positively affect the SCN neurons in humans.

A decreased ability to maintain a stable circadian pattern of daytime arousal and nocturnal quiescence may contribute to cognitive dysfunction, behavioural disturbances, sleep disruptions, and

depression associated with dementia (Haffmans 2001; Mishima 1999; Satlin 1992). The rest-activity and sleep-wake cycles are controlled by the endogenous circadian rhythm generated by the suprachiasmatic nuclei (SCN) of the hypothalamus (Harper 2001). The SCN, considered to function as a biological clock, synchronize internal rhythms with the environmental light-dark cycles predominantly by responding directly to retinal input (van Someren 1996; van Someren 1999). Light impinging on the retina is transduced into neural activity that reaches the SCN through the retinohypothalamic and possibly the geniculo-hypothalamic tracts. Light leads to changes in the firing rates of specialized neurons in the SCN that in turn affect circadian rhythms (Chesson 1999).

In addition to the internal regulatory loss, elderly people (especially those with dementia) experience a reduction in sensory input because they are visually less sensitive to light, and have less exposure to bright environmental light. They also, typically, have fewer social contacts. Reduced sensory input is likely to lower the 'general level of excitement' that is thought to play an important role in the entrainment of circadian rhythms (van Someren 1993).

Why it is important to do this review

Several studies have examined the effectiveness of light therapy in managing disturbances of cognition, sleep, function, behaviour or psychiatric disturbances in individuals with dementia (e.g. Ancoli-Israel 2003a; Colenda 1997; Dowling 2007; Gasio 2003; Graf 2001; Ito 2001; Lovell 1995; Lyketos 1999; Mishima 1998; Satlin 1992; Riemersma 2008; Thorpe 2000; van Someren 1997). There is preliminary evidence from some studies (e.g., Gasio 2003; Lyketos 1999) that light therapy improves nocturnal sleep, while other studies (e.g., Dowling 2008) demonstrate no improvement in people with dementia. Thus, it is important to test the hypothesis that degenerative changes in the SCN are the biological basis of circadian disturbances in people with dementia that may be reversed by stimulation of the SCN by light (Liu 2000). There is therefore a need for a systematic review of studies that examines the effectiveness of light therapy in managing cognition, sleep, function, behaviour, or psychiatric disturbances associated with dementia.

OBJECTIVES

The objectives of the systematic review are:

- to assess the quality of studies that measure the effectiveness of light therapy in managing cognitive, sleep, functional, behavioural, or psychiatric disturbances associated with dementia;

- to make recommendations to consumers, practitioners, and researchers regarding the effectiveness of light therapy in managing cognitive, sleep, functional, behavioural, and/or psychiatric disturbances associated with dementia.

METHODS

Criteria for considering studies for this review

Types of studies

Randomized controlled trials (RCTs) in which light therapy of any intensity and duration is compared with a control group for the management of cognitive, sleep, functional, behavioural, or psychiatric disturbances associated with dementia were included. Since the intervention consisted of bright light, single-blind RCTs were expected; double-blind RCTs would be difficult to achieve.

Types of participants

The participants in a study must have a diagnosis of dementia (Alzheimer's disease, Dementia with Lewy Bodies, Vascular Dementia, or dementia due to another cause) according to accepted criteria such as the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R, DSM-IV) (APA 1995), the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (NINCDS-ADRDA) (McKhann 1984), or ICD-10 (WHO 1992). Severity of dementia should be assessed by the use of standardized instruments such as the Mini-Mental State Examination (Folstein 1975). Level of severity of dementia, age, and sex were not inclusion criteria.

Types of interventions

Any form of intervention involving the use of bright light.

Types of outcome measures

Objective outcome measures sensitive to changes in cognition, sleep, function, behaviour, or psychiatric disturbances were of interest to this review. These measures could be obtained at baseline, during the light therapy, immediately following, or at any interval of time after the treatment. Both dichotomous and continuous data were accepted. Outcome measures that assessed at least one of the following were included:

- changes in deterioration of cognition (e.g., memory)
- changes in the incidence or frequency of sleep-wake disturbances

- change in level of functional decline (e.g., activities of daily living)
- changes in the incidence, severity or frequency of behavioural disturbances (e.g., agitation)
- changes in incidence, severity or frequency of psychiatric disturbances (e.g., depression)
- changes in rate of institutionalization
- impact on cost of care

Search methods for identification of studies

The Specialized Register of the Cochrane Dementia and Cognitive Improvement Group (CDCIG) was searched on 4 March 2008 for all years up to December 2005. This register contains records from the following major healthcare databases *The Cochrane Library*, MEDLINE, EMBASE, PsycINFO, CINAHL and LILACS, and many ongoing trial databases and other grey literature sources. The following search terms were used: "bright light*", "light box*", "light visor*", "dawn-dusk*", phototherapy, "photo therapy", "light therapy" "light treatment", light* *The Cochrane Library*, MEDLINE, EMBASE, PsycINFO, CINAHL and LILACS were searched separately on 4 March 2008 for records added to these databases after December 2005 to January 2008. The search terms used to identify relevant controlled trials on dementia, Alzheimer's disease and mild cognitive impairment for the Group's Specialized Register can be found in the Group's module on *The Cochrane Library*. These search terms were combined with the following search terms and adapted for each database, where appropriate: "bright light*", "light box*", "light visor*", "dawn-dusk*", phototherapy, "photo therapy", "light therapy" "light treatment", light*

On 4 March 2008, the Specialized Register consisted of records from the following databases:

Healthcare databases:

The Cochrane Library: (2006, Issue 1);
MEDLINE (1966 to 2006/07, week 5);
EMBASE (1980 to 2006/07);
PsycINFO (1887 to 2006/08, week 1);
CINAHL (1982 to 2006/06);
SIGLE (Grey Literature in Europe) (1980 to 2005/03);
LILACS: Latin American and Caribbean Health Science Literature (<http://bases.bireme.br/cgi-bin/wxislind.exe/iah/online/?IsisScript=iah/iah.xis&base=LILACS&lang=i&form=F>) (last searched 29 August 2006).

Conference proceedings:

ISTP (<http://portal.isiknowledge.com/portal.cgi>) (Index to Scientific and Technical Proceedings) (to 29 August 2006);
INSIDE (BL database of Conference Proceedings and Journals) (to June 2000);

Theses:

Index to Theses (formerly ASLIB) (<http://www.theses.com/>) (UK and Ireland theses) (1716 to 11 August 2006);
Australian Digital Theses Program (<http://adt.caul.edu.au/>): (last update 24 March 2006);
Canadian Theses and Dissertations (<http://www.collectionscanada.ca/thesescanada/index-e.html>): 1989 to 28 August 2006);
DATAD - Database of African Theses and Dissertations (<http://www.aau.org/datad/backgrd.htm>);
Dissertation Abstract Online (USA) (<http://www.lib.umi.com/dissertations/gateway>) (1861 to 28 August 2006).

Ongoing trials:

UK

National Research Register (<http://www.update-software.com/projects/nrr/>) (last searched issue 3/2006);
ReFeR (<http://www.refer.nhs.uk/ViewWebPage.asp?Page=Home>) (last searched 30 August 2006);
Current Controlled trials: Meta Register of Controlled trials (mRCT) (<http://www.controlled-trials.com/>) (last searched 30 August 2006) :
ISRCTN Register - trials registered with a unique identifier
Action medical research
Kings College London
Laxdale Ltd
Medical Research Council (UK)
NHS Trusts Clinical Trials Register
National Health Service Research and Development Health Technology Assessment Programme (HTA)
National Health Service Research and Development Programme 'Time-Limited' National Programmes
National Health Service Research and Development Regional Programmes
The Wellcome Trust
Stroke Trials Registry (<http://www.strokecenter.org/trials/index.aspx>) (last searched 31 August 2006);

Netherlands

Nederlands Trial Register (<http://www.trialregister.nl/trialreg/index.asp>) (last searched 31 August 2006);

USA/International

ClinicalTrials.gov (<http://www.ClinicalTrials.gov>) (last searched 31 August 2006)
(contains all records from <http://clinicalstudies.info.nih.gov/>);
IPFMA Clinical trials Register: www.ifpma.org/clinicaltrials.html.
The Ongoing Trials database within this Register searches <http://www.controlled-trials.com/isrctn>, <http://www.ClinicalTrials.gov> and <http://www.centerwatch.com/>. The ISRCTN register and Clinicaltrials.gov are searched separately. Centerwatch is very dif-

ficult to search for our purposes and no update searches have been done since 2003.

The IPFMA Trial Results databases searches a wide variety of sources among which are:

<http://www.astrazenecaclinicaltrials.com> (seroquel, statins)
<http://www.centerwatch.com>
<http://www.clinicalstudyresults.org>
<http://clinicaltrials.gov>
<http://www.controlled-trials.com>
<http://ctr.gsk.co.uk>
<http://www.lillytrials.com> (zyprexa)
<http://www.roche-trials.com> (anti-beta antibody)
<http://www.organon.com>
<http://www.novartisclinicaltrials.com> (rivastigmine)
<http://www.bayerhealthcare.com>
<http://trials.boehringer-ingelheim.com>
<http://www.cmrinteract.com>
<http://www.esteve.es>
<http://www.clinicaltrials.jp>.

This part of the IPFMA database is searched and was last updated on 4 September 2006;

Lundbeck Clinical Trial Registry (<http://www.lundbecktrials.com>) (last searched 15 August 2006);
Forest Clinical trial Registry (<http://www.forestclinicaltrials.com/>) (last searched 15 August 2006).

The search strategies used to identify relevant records in MEDLINE, EMBASE, PsycINFO, CINAHL and LILACS can be found in the Group's module on *The Cochrane Library*.

Data collection and analysis

1. Three reviewers (DF, IC and AL) independently assessed the relevance of the retrieved articles. The relevance criteria consisted of the following questions:

- Does the article describe an evaluation of the effectiveness of light therapy in managing cognitive, sleep, functional, behavioural, or psychiatric disturbances associated with dementia using a randomized controlled trial design?
- Does the study measure at least one of the following patient/resident outcomes: cognition, sleep-wake disturbances, function, behavioural disturbances, psychiatric disturbances, or cost?

Relevant criteria had to be met for the study to be included in the next stage of assessment. Disagreements were resolved by discussion and agreement was reached.

2. Three reviewers (DF, IC and AL) then independently assessed the selected studies for methodological quality using criteria adapted from the Cochrane Handbook for Systematic Reviews of Interventions (Higgins 2008) and Moher 2008. The following factors were assessed: sequence generation, allocation conceal-

ment, attrition rate, compliance, control of potential confounders, blinding for data collection and outcome measures, presence of point estimates, and measures of variability for the outcomes.

3. Three reviewers (DE, IC, and AL) independently extracted data from the studies selected for inclusion. Information regarding the publication date; authors; country; study design; characteristics of the study population including setting; type, duration, intensity, frequency, and time of day of light therapy; control intervention; concurrent interventions; and measures of outcomes were extracted, recorded, and entered into RevMan5.

4. A Continuous Data Table (number of participants in each group, means and standard deviations for the outcomes in each group) was developed. Attempts were made to collect missing data from the original authors. Each study was summarized using a measure of effect (e.g., mean difference). The studies were examined for degree of heterogeneity, to determine the possibility of combining the results. Statistical heterogeneity was assessed using the I^2 test that measures the degree of inconsistency across studies (if I^2 equals 0% then there is no apparent heterogeneity. Larger values [70% and greater] indicate greater heterogeneity and caution should be used in interpreting the meta-analysis). Both the fixed-effects and random-effects models were used. We have exercised caution with values greater than or equal to 50% and selected a random effects model for those values. If the value was less than 50%, a fixed effects model was selected. Unfortunately, the sample sizes were not large enough to conduct subgroup analyses to explore potential differences that may have influenced the results. Lastly, sensitivity analyses would have been conducted to determine how sensitive the results of the analyses were to changes in the way they were conducted, if the number of included studies had been larger.

5. All reviewers discussed and reached agreement on the interpretation of the results. The consumer editor and other reviewers commented on the draft review prior to its submission to the CDCIG.

RESULTS

Description of studies

See: [Characteristics of included studies](#); [Characteristics of excluded studies](#); [Characteristics of ongoing studies](#).

Please see Table *Characteristics of included studies*

An updated search strategy on March 6, 2008 and an external reviewer together revealed four new RCTs which were published as seven articles (Dowling 2005a; Dowling 2005b; Dowling 2007; Dowling 2008; McCurry 2005; McCurry 2006; Riemersma 2008) and three non-RCTs (Hickman 2007; Skjerve 2004; Sloane 2007). The non-RCTs and the McCurry 2005; McCurry 2006 articles did not meet our relevance criteria and were therefore not included

in the updated review. As well, the Dowling 2005a article appeared to report preliminary results, the full reported study was found in Dowling 2005b. Thus, three new trials (four articles) were added to our previous review (Dowling 2005b; Dowling 2007; Dowling 2008; Riemersma 2008). In total, eight trials (ten articles; Ancoli-Israel 2003a; Ancoli-Israel 2003b; Dowling 2005b; Dowling 2007; Dowling 2008; Gasio 2003; Graf 2001; Lyketsos 1999; Mishima 1998; Riemersma 2008) met the relevance and risk of bias criteria and were included in the review.

The articles included in this review were published between 1998 and 2008. Four of the trials were conducted in the United States (Ancoli-Israel 2003a; Ancoli-Israel 2003b; Dowling 2005b; Dowling 2007; Dowling 2008; Lyketsos 1999), one was conducted in Japan (Mishima 1998), one in the Netherlands (Riemersma 2008), one in Switzerland (Gasio 2003), and one in Austria (Graf 2001). All of the participants were residents of long-term care facilities of varying descriptions: assisted living (Riemersma 2008), nursing homes (Ancoli-Israel 2003a; Ancoli-Israel 2003b; Graf 2001; Dowling 2008), chronic care facilities (Dowling 2005b; Dowling 2007; Lyketsos 1999), specialized wards (Mishima 1998), and nursing wings for residents with dementia (Gasio 2003).

Consent was obtained from the residents and/or from their relatives (Dowling 2005b; Dowling 2007; Dowling 2008; Graf 2001; Mishima 1998; Riemersma 2008) as well as from their physicians (Ancoli-Israel 2003a; Ancoli-Israel 2003b; Gasio 2003). Consent was not mentioned in one study (Lyketsos 1999). The number of residents who agreed to participate in the included studies was relatively small, with a total of 373 participants. Of these participants, 278 to 279 completed the protocol (the range reflects the different outcomes measured in the same study; Ancoli-Israel 2003a; Ancoli-Israel 2003b).

The participants met the DSM-IV or NINCDS-ADRDA criteria for Alzheimer's disease (Ancoli-Israel 2003a; Ancoli-Israel 2003b; Dowling 2005b; Dowling 2007; Dowling 2008; Mishima 1998; Riemersma 2008), Vascular Dementia (VD) (Mishima 1998; Riemersma 2008) or dementia (Lyketsos 1999; Riemersma 2008). In one study, the participants were included only if their Mini-Mental State Examination (MMSE) score was no more than 23 (Graf 2001). In all but two studies (Dowling 2005b; Dowling 2007), the MMSE was used to measure the severity of dementia at baseline. The mean MMSE scores of the participants in the included studies ranged from severe to moderate levels of dementia: 5.7 (SD 5.6) (Ancoli-Israel 2003a; Ancoli-Israel 2003b), 6.4 (SD 6.8) (Lyketsos 1999), 8.45 (range 3-17) (Mishima 1998), 9.3 (SD 7.9) (Dowling 2008), 13.92 (SD 5.37) (Gasio 2003), 14.4 (SD 6.6) (Riemersma 2008), and 15.9 (SD 5.90) (Graf 2001). In Graf 2001, subtypes of dementia were diagnosed by assessing whether the progress of the dementia was steady suggesting Alzheimer's disease, or was step-wise suggesting Vascular Dementia; and whether there was evidence of focal neurological deficits, essential hypertension, or vascular brain disease on computerized tomographic

scan suggesting Vascular Dementia. In [Mishima 1998](#) all participants underwent brain magnetic resonance imaging (MRI) and computerized tomographic (CT) examinations; and residents with mixed type dementia (usually Alzheimer's disease with Vascular Dementia) were excluded from the study. Overall, 310 (83%) of the participants in the included studies were diagnosed with probable Alzheimer's disease. The remainder were diagnosed with either Vascular Dementia (n=39, 10%) or another type of dementia (n=24, 6%).

Light therapy was usually administered from a Brite-LiteTM box (Apollo Light Systems, Orem, Utah) which was 24 inches wide by 12 inches high by 3 inches deep and placed approximately one metre from the participant's head. The Brite-LiteTM utilized cool-white fluorescent, non-ultra-violet, full-spectrum light bulbs with special ballast to augment the brightness. The treatment groups received light therapy ranging from 2,500 to 10,000 lux and the control groups received dim red light or dim, low-frequency blinking light, less than 300 lux; either in the morning or evening, for one to two hours, for ten days to ten weeks. There were two exceptions: the use of dawn-dusk simulation (maximum 400 lux) or placebo dim red light (< 5 lux) ([Gasio 2003](#)) and the use of ceiling mounted light fixtures ([Riemersma 2008](#)). The Dawn-Dusk SimulatorTM included a computer algorithm that drove an electronic controller connected to an overhead halogen lamp placed behind a diffusing membrane behind participants' bed. In [Riemersma 2008](#), residents were exposed to light by means of ceiling-mounted fixtures with plexiglas diffusers containing an equal amount of Philips TLD840 and TLD940 fluorescent tubes, which were installed in the common living area. The lights were kept on between approximately 0900 and 1800 hours with the aim of an exposure of ± 1000 lux ([Riemersma 2008](#)).

Rest-activity cycles were documented using small wrist-mounted activity monitors such as the ActillumTM (Ambulatory Monitoring, Inc., Ardsley, New York, cited in [Ancoli-Israel 2003a](#)), ActigraphTM (AMI, Ardsley, Inc. New York, cited in [Mishima 1998](#)) and the ActiwatchTM ([Dowling 2008](#); [Gasio 2003](#); [Riemersma 2008](#)). The ActillumTM, for example, records activity level with

a linear accelerometer and a microprocessor, and light exposure is collected via a photosensitive cell. Both activity and light data were sampled every 10 seconds and stored every minute on a 32 K byte memory chip. The reliability of the ActillumTM for estimation of sleep and wake in nursing home residents has been found to be 0.81 (p< .005) for maximum activity and 0.91 (p<.001) for mean activity levels ([Ancoli-Israel 2003a](#)). In addition, sleep logs generated by nurses recorded hours of sleep ([Gasio 2003](#), [Lyketsos 1999](#)).

Memory, cognitive impairment, and progression of dementia were measured by the Consortium to Establish a Registry for Alzheimer's Disease (CERAD) ([Morris 1989](#)) in [Gasio 2003](#) and the MMSE ([Folstein 1975](#)) in [Gasio 2003](#), [Graf 2001](#), and [Riemersma 2008](#). Function was measured by the Nurse-Informant Activities of Daily Living (NI-ADL) scale ([Holmes 1990](#)), an adaptation of the Katz Activities of Daily Living Scale ([Katz 1963](#)) in [Riemersma 2008](#). Disruptive behaviours were measured by the Behavioral Pathology in Alzheimer Disease scale (Behav-AD) ([Reisberg 1987](#)) in [Lyketsos 1999](#); Cohen-Mansfield Agitation Inventory (CMAI) ([Cohen-Mansfield 1989](#)) and Agitated Behavior Rating Scale (ABRS) ([Bliwise 1993](#)) in [Ancoli-Israel 2003b](#); Neuropsychiatric Inventory (NPI) ([Cummings 1994](#)) in [Gasio 2003](#) and [Riemersma 2008](#); and Neuropsychiatric Inventory - Nursing Home Edition (NPI-NH) ([Iverson 2002](#)) in [Dowling 2007](#). Psychiatric disturbances were measured by the Cornell Scale for Depression in Dementia (CSDD) ([Alexopoulos 1988](#)) in [Lyketsos 1999](#), the Philadelphia Geriatric Centre Affect Rating Scale (PG-CARS) ([Lawton 1996](#)) in [Riemersma 2008](#), and the Geriatric Depression Scale (GDS) ([Sheikh 1986](#)) in [Gasio 2003](#). See Additional Table *Description of rating scales used in the included studies* (Table 1).

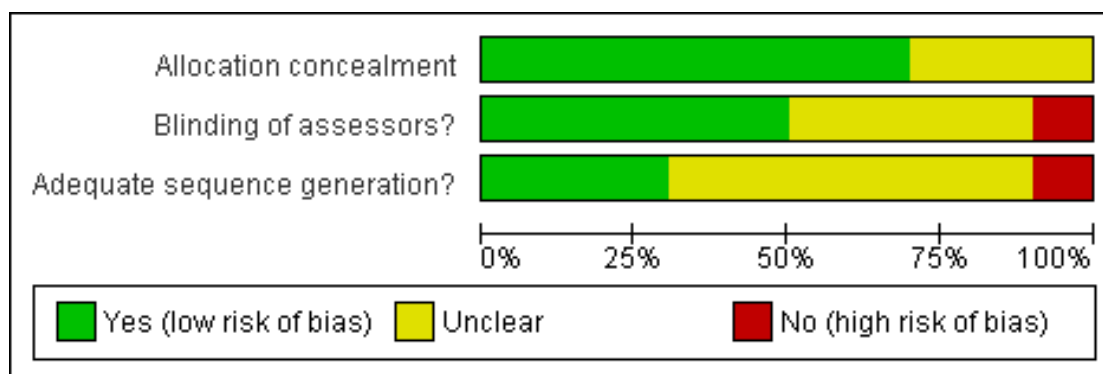
Risk of bias in included studies

Please see Additional Table *Description of Risk of Bias of Included Studies* (Table 2) and *Summaries of Risk of Bias of Included Studies* Figure 1, Figure 2.

Figure 1. Methodological quality summary: review authors' judgements about each methodological quality item for each included study.

	Allocation concealment	Blinding of assessors?	Adequate sequence generation?
Ancoli-Israel 2003a	+	+	?
Ancoli-Israel 2003b	+	+	?
Dowling 2005b	+	-	?
Dowling 2007	+	?	?
Dowling 2008	+	?	?
Gasio 2003	?	?	+
Graf 2001	?	+	-
Lyketsos 1999	+	+	+
Mishima 1998	?	?	?
Riemersma 2008	+	+	+

Figure 2. Methodological quality graph: review authors' judgements about each methodological quality item presented as percentages across all included studies.



The process of randomization was assessed based on how the authors generated the allocation sequence of participants to either a treatment or control group. Investigators who used a computer generated sequence program, random number tables, lot drawing, coin tossing, shuffling cards, or throwing dice were rated as 'adequate'. Those who used case number, date of birth, date of admission, or alternation were rated as 'inadequate'. If the randomization process was not adequately described in the article and the investigators did not respond to requests for clarification, then the study received an 'unclear' rating. The studies were also rated on concealment of allocation sequence. If the investigators used central randomization or envelopes that were sealed, opaque, and sequentially numbered, then the study was rated as 'adequate'. If open allocation sequence was used or the procedure was based on inadequate generation, then the study was rated as 'inadequate' for allocation concealment. If the process was not adequately described in the article and the investigators did not provide clarification, then the study received an 'unclear' rating. All but one of the authors of the included studies were contacted to determine the method of randomization and allocation concealment, as the description in the published articles was incomplete.

Riemersma 2008 randomized sequence generation and concealed allocation to group assignment through the use of the Microsoft WordTM random number function. This study was rated as 'adequate' for selection and allocation. The sequence generation and allocation processes in the Dowling 2005b, Dowling 2007, and Dowling 2008 articles were not described. Dr. Dowling was contacted for clarification and responded on October 28, 2008 that a permuted blocking procedure was used in which the numbers of patients allocated to each group was forced to be equal after an

a priori defined "balancing" number of participants had been enrolled in the study. This study was rated as 'unclear' for sequence generation due to the potential for selection bias but 'adequate' for concealment of allocation sequence. Further clarification about the sequence generation procedure has been requested but to date not received. Another trial utilized block-stratified randomization using pre-assignment by order of entry into strata; stratification was determined by sex and by quartiles of the categorical sleep spread score or by time of agitation (Ancoli-Israel 2003a; Ancoli-Israel 2003b). This study was rated as 'unclear' for sequence generation due to the potential for selection bias but 'adequate' for concealment of allocation sequence. Graf 2001 used date of admission to prospectively randomize participants to either the treatment or control group (personal communication, Alexander Neumeister, August 5, 2003). This study was rated as 'inadequate' for sequence generation. A table of random numbers was used to generate the participants and a sealed envelope was used to conceal the allocation sequence in Lyketos 1999. Gasio 2003 also used a random number generator (personal communication, Anna Wirz-Justice, June 10, 2003 regarding Gasio 2003; further clarification about the concealment of assignment to groups was requested on June 14, 2003 but to date not received). These two studies were rated 'adequate' for sequence generation and the Lyketos 1999 study was also rated 'adequate' for concealment of allocation to groups. The processes of sequence generation and concealment of allocation to groups were not described in the Mishima 1998 study and the authors have not responded to requests for this information that were made on May 29, 2003 and August 13, 2003. This study was rated as 'unclear' for both sequence generation and allocation

concealment.

The exclusion criteria of the studies ensured that many of the potential confounders were eliminated. For example, residents who were blind or severely visually impaired or had severe motor symptoms or primary psychiatric disorders, were not included in the studies (Ancoli-Israel 2003a; Ancoli-Israel 2003b; Dowling 2005b; Dowling 2007; Dowling 2008; Graf 2001; Gasio 2003; Lyketsos 1999; Mishima 1998; Riemersma 2008).

Participants' medications were stabilized for various periods of time prior to initiating the trials: 12 weeks (Mishima 1998), one month (Graf 2001), and one week (Lyketsos 1999). In addition, Dowling 2005b, Dowling 2007, Dowling 2008 and Mishima 1998 excluded participants who were taking melatonin, sedatives, hypnotics or antipsychotics. Riemersma 2008 and Gasio 2003 kept the medications as constant as possible and listed each of the medications in a table. Ancoli-Israel 2003a and Ancoli-Israel 2003b did not report if and how medication use was dealt with. Four of the included studies had small sample sizes ranging from 13 to 23 participants at baseline, and 8 to 13 participants at completion (Gasio 2003; Graf 2001; Lyketsos 1999; Mishima 1998). The four remaining included studies had larger sample sizes, ranging from 50 to 92 participants at baseline, and 50 to 72 at completion (Ancoli-Israel 2003a; Ancoli-Israel 2003b; Dowling 2005b; Dowling 2007; Dowling 2008) and 26 participants after two years of the intervention (Riemersma 2008).

Attrition rates varied from 0% to 47% in the included studies. Often studies did not report whether the drop out rates related to the treatment or control groups. Compliance with the light therapy and/or wearing the activity monitor was an issue in some of the studies. Two studies reported that participants received 77% (Ancoli-Israel 2003b) and 82% (SD 17%; Dowling 2008) of the light therapy. The range of compliance with wearing the activity monitors was 75% to 100% of the participants (Ancoli-Israel 2003a; Dowling 2005b; Gasio 2003). Dowling 2008 reported that of a total possible 108 hours, on average 105 ±8 hours of valid data for baseline and 107 ±3 hours of valid data at the end of the intervention were collected. Mishima 1998 did not report compliance with the actigraph (information requested August 11, 2003). Riemersma 2008 did not report the participants' exposure

to the light therapy.

One trial reported double-blinding, as both the assessors and the participants were not aware of the treatment condition (Riemersma 2008). Another trial reported that the study staff, nursing home staff, and participants were blinded to the melatonin intervention but were not blinded to the bright light (Dowling 2008). Three trials reported that those who assessed the outcomes were blind to group allocation (Dowling 2005b; Dowling 2007; Graf 2001; Lyketsos 1999). In other studies, nursing and research staff (Ancoli-Israel 2003a; Ancoli-Israel 2003b) or residents and staff (personal communication, Anna Wirz-Justice, June 10, 2003 regarding Gasio 2003) were informed that both the white and red coloured light conditions were expected to show improvement and that the study was examining which colour was better.

Effects of interventions

Several outcomes were measured: cognition, function, sleep, behavioural and psychiatric disturbances. These are each discussed below.

Cognition

Three studies (Gasio 2003; Graf 2001, Riemersma 2008) used the MMSE to measure cognition. Evening bright light (3,000 lux) was compared with evening dim light (100 lux) in Graf 2001, all day bright light (1,000 lux) was compared with dim light (300 lux) in Riemersma 2008, and dawn-dusk simulation with light up to 400 lux was compared with dawn-dusk simulation with dim red light (<5 lux) in Gasio 2003. Only the data in the Riemersma 2008 and Graf 2001 studies were combined because their light intensities were both considered bright light. The pooled data revealed no effect following 10 to 42 days of treatment (MD=1.10, 95% CI -1.37 to 3.57, p=.38; Figure 3). Riemersma 2008 data revealed similar results after 1 year of treatment (MD=1.70, 95%CI -1.03 to 4.43, p=.22; Figure 4), and after 2 years of treatment (MD=3.60, 95%CI -1.05 to 8.25, p=.13; Figure 5). Graf 2001 administered evening bright light for 10 days that had no effect on cognition (MD=0.70, 95% CI -4.90 to 6.3, p=.81; Figure 6). Similarly, the Gasio 2003 study revealed no effect at endpoint (MD=.46, 95% CI -14.14 to 15.06, p=.95; Figure 7) and at follow up (3 weeks after treatment) (MD=-0.50, 95% CI -10.68 to 9.67, p=.92; Figure 8).

Figure 3. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: I.I Cognition at endpoint (MMSE; 10-42 days).

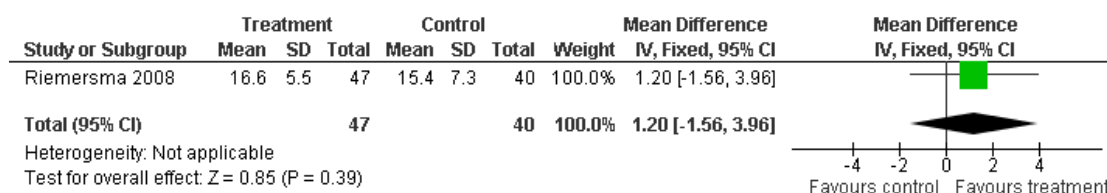


Figure 4. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.2 Cognition at endpoint (MMSE; 1 year).

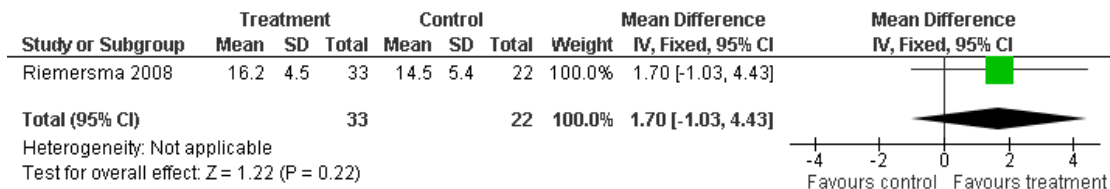


Figure 5. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.3 Cognition at endpoint (MMSE; 2 years).

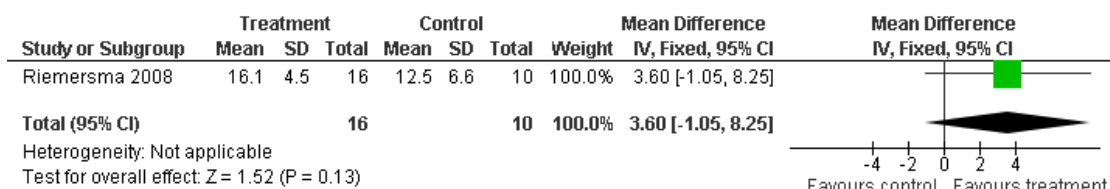


Figure 6. Forest plot of comparison: 2 Evening/afternoon bright light vs control, outcome: 2.1 Cognition at endpoint (MMSE; 10 days).

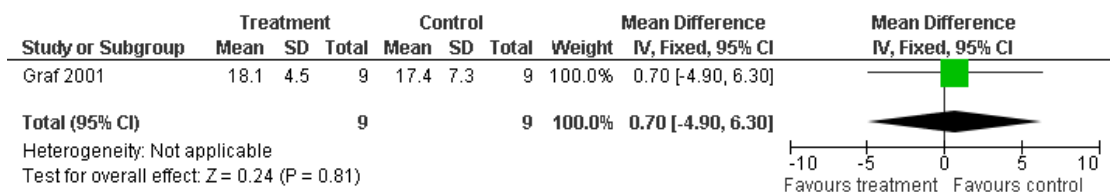


Figure 7. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.1 Cognition at endpoint (after 3 weeks of treatment (MMSE scores)).

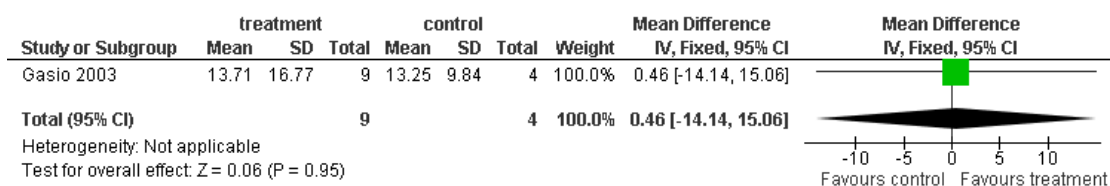
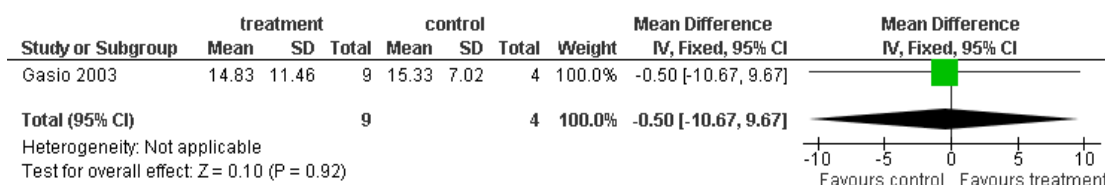


Figure 8. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.2 Cognition at follow-up (3 weeks after treatment (MMSE scores)).



Function

One study (Riemersma 2008) measured functional limitations using NI-ADL after 6 weeks, 1 and 2 years of treatment. After 6 weeks of treatment, light therapy had a positive effect in attenuating the increase in functional limitations (MD= -5.00, 95% CI -9.87 to -0.13, p=.04; Figure 9). After 1 year of treatment, there was no significant effect (MD -5.00, 95% CI -11.16 to 1.16, p=.11; Figure 10), however, a significantly less steep increase in functional decline was seen after 2 years of light therapy (MD= -16.00, 95% CI -26.21 to -5.79, p=.002; Figure 11).

Figure 9. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: I.4 Function at endpoint (NI-ADL; 42 days).

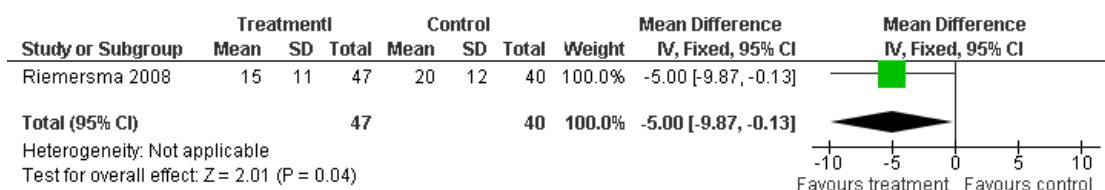


Figure 10. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: I.5 Function at endpoint (NI-ADL; 1 year).

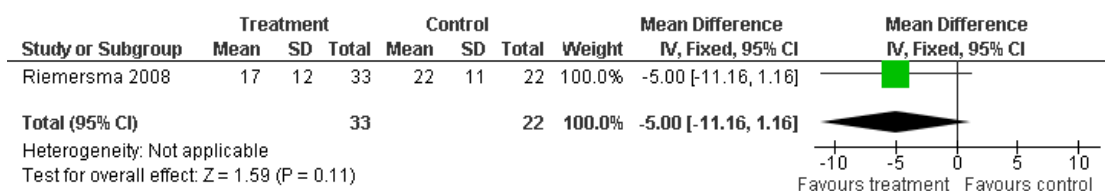
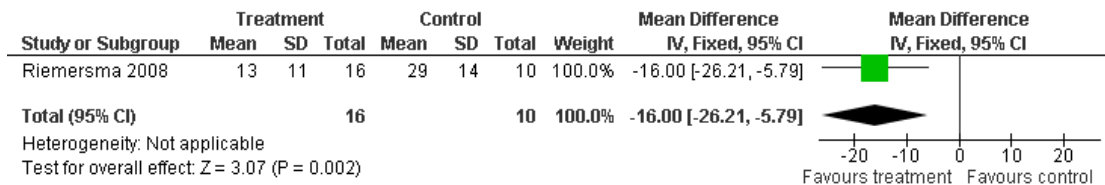


Figure 11. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.6 Function at endpoint (NI-ADL; 2 years).



Sleep

Sleep latency, defined as the amount of time between reclining in bed and the onset of sleep (Davis 2001) was measured in Gasio 2003 and Riemersma 2008. However the data from these two studies could not be pooled due to differences in light intensity. Findings from Riemersma 2008 revealed that there were no significant improvements in sleep onset latency after 6 weeks of treatment (MD=6.00, 95% CI -12.34 to 24.34, p=.52; Figure 12), 1 year of treatment (MD=5.00, 95% CI -24.79 to 34.79, p=.74; Figure 13) and after 2 years of treatment (MD=10.00, 95% CI -11.33 to 31.33, p=.36; Figure 14). Similarly, data from Gasio 2003 revealed that “naturalistic light” did not significantly reduce sleep latency after 3 weeks of treatment (MD -79.00, 95% CI -327.17, 169.17, p=.53; Figure 15) and after 3 weeks of follow-up (MD -62.00, 95%CI -216.55 to 92.55, p=.43; Figure 16).

Figure 12. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.7 Sleep onset latency (42 days).

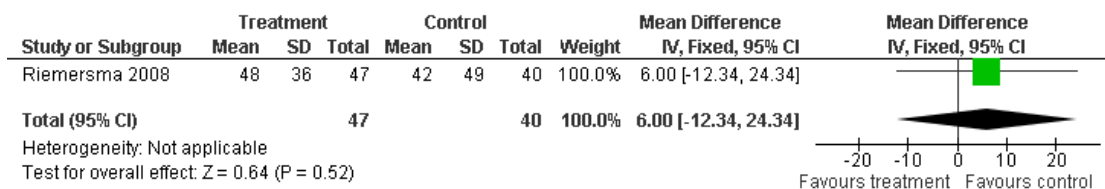


Figure 13. Forest plot of comparison: 1 Day time bright light vs control, outcome: 1.8 Sleep onset latency (1 year).

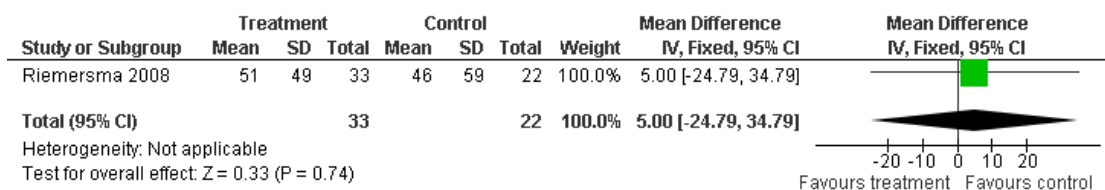


Figure 14. Forest plot of comparison: 1 Day time bright light vs control, outcome: 1.9 Sleep onset latency (2 years).

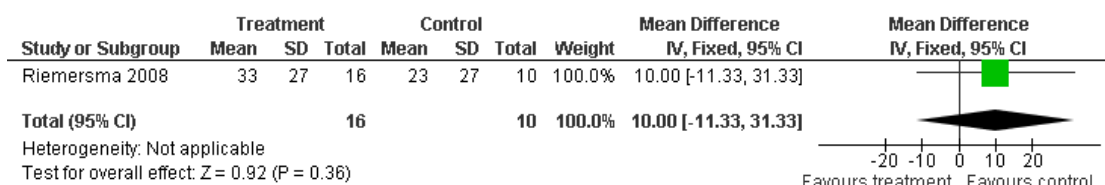


Figure 15. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.3 Sleep onset latency (minutes) at endpoint (after 3 weeks of treatment).

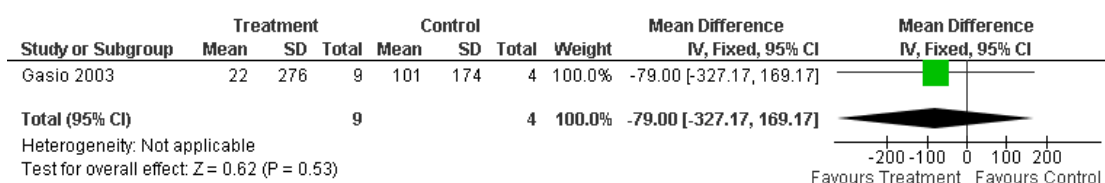
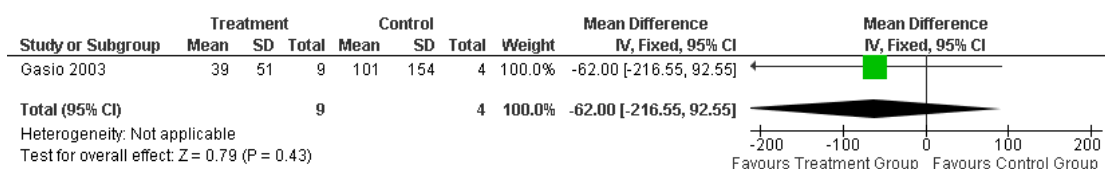


Figure 16. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.4 Sleep onset latency (minutes) at follow-up (3 weeks after treatment).



Six studies measured total night sleep duration following 10 days (Ancoli-Israel 2003a), 3 weeks (Gasio 2003), 4 weeks (Lyketso 1999), 10 weeks (Dowling 2005b; Dowling 2008), and 1 and 2 years of treatment (Riemersma 2008) that consisted of bright light therapy ($\geq 2500 - 10,000$ lux) for one hour in the morning (Ancoli-Israel 2003a; Lyketso 1999; Dowling 2008) or evening (Ancoli-Israel 2003a; Dowling 2005b), all day bright light (1000 lux) (Riemersma 2008), or dawn-dusk simulation (400 lux) morning and evening (Gasio 2003). The treatment groups were compared with control groups who received dim light. Unfortunately, Ancoli-Israel 2003a reported only the combined findings of both

the bright light therapy and dim red light groups because there were no significant differences between the groups. Requests for group or individual data (Oct. 29, 2008) have not been forthcoming. Thus, the data from this study could not be included in the analysis. In addition, the study by Lyketso 1999, which was a cross-over design, does not appear to have utilized analyses appropriate to a paired design. Group data prior to the cross-over were requested (August 12, 2003), but have not yet been provided. Thus, the findings from Lyketso 1999 also had to be excluded from the analyses. Dowling 2005b, Dowling 2008 and Riemersma

2008 combined data revealed no effect of morning to all day bright light on total night sleep duration (MD= 10.25, 95%CI -21.05 to 41.54, p= .52; Figure 17). Evening bright light (Dowling 2005b) revealed similar findings (MD=10.00, 95%CI -59.22 to 79.22, p=.78; Figure 18). Similarly, data from Riemersma 2008 revealed that bright light had no effect on night sleep duration after 1 year (MD= -36.00, 95% CI -84.21 to 12.21, p=.14; Figure 19) and 2 years of treatment (MD= -36.00, 95% CI -121.69 to 49.69, p=.41; Figure 20). Data from Gasio 2003 were analysed separately due to the lower intensity of treatment light. No effect was found after 3 weeks of treatment (MD=143.00, 95% CI -637.66 to 923.66, p=.72; Figure 21) or at follow-up (MD=110.00, 95% CI -77.22 to 297.22, p=.25; Figure 22).

Figure 17. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.10 Total sleep duration (mins; 6-50 days).

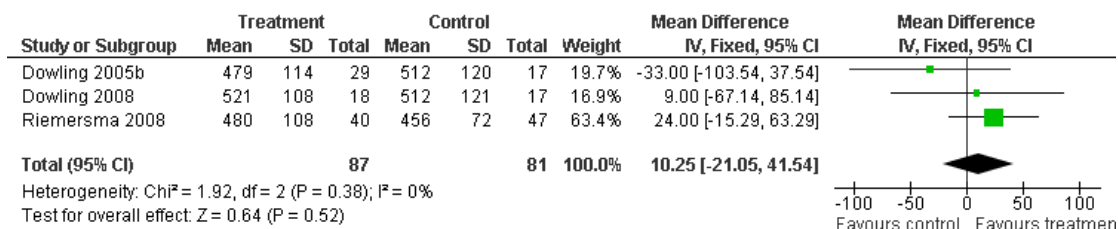


Figure 18. Forest plot of comparison: 2 Evening bright light vs control, outcome: 2.1 Total sleep duration (minutes) at endpoint.

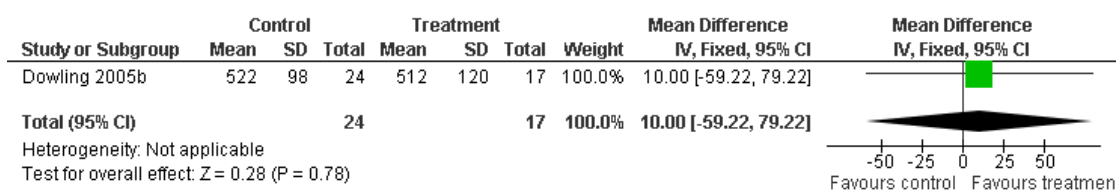


Figure 19. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: I.11 Total sleep duration (mins; 1 year).

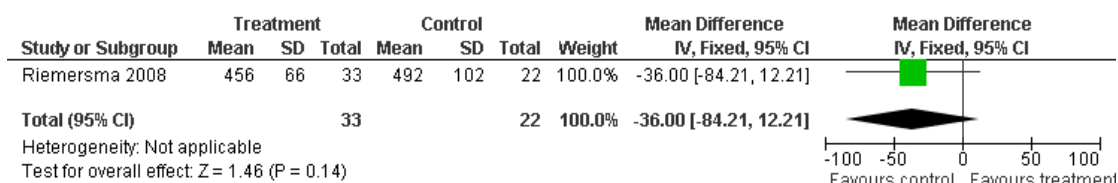


Figure 20. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: I.12 Total sleep duration (mins; 2 years).

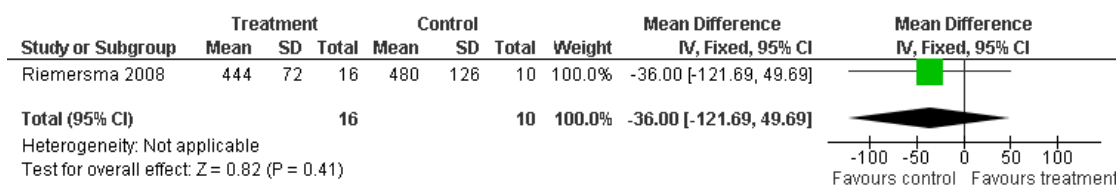


Figure 21. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.5 Total sleep duration (minutes) at endpoint (after 3 weeks of treatment).

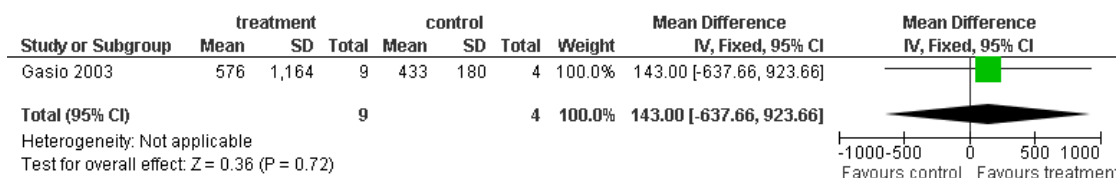
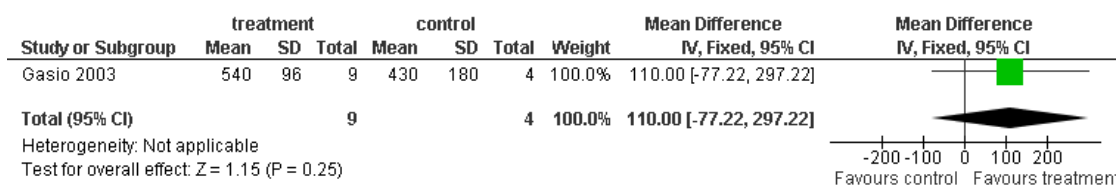


Figure 22. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.6 Total sleep duration (minutes) at follow-up (3 weeks after treatment).



Four studies (Ancoli-Israel 2003a, Dowling 2005b, Gasio 2003, Mishima 1998) measured night-time activity counts. Unfortunately, the findings from Ancoli-Israel 2003a cannot be included in the analyses for reasons described above. In addition, the study by Mishima 1998, which was a cross-over design, does not appear to utilize analyses appropriate to a paired design. Group data prior to the cross-over were requested (August 13, 2003), but have not yet been provided. Thus, the findings from this study cannot be included in the analyses. The findings from Dowling 2005b and Gasio 2003 could not be combined due to the differences in intensity of the light therapy. Dowling 2005b measured activity scores per night for both morning and afternoon treatment groups compared with control groups after 10 weeks of treatment. No effect on night time activity scores was found when bright light

was administered in the morning (MD= 855.78, 95% CI -867.84 to 2579.40, p=.33; Figure 23) or afternoon (MD= -78.60, 95% CI -627.17 to 469.97, p=.78; Figure 24). In Gasio 2003, activity for each participant was averaged in one-hour bins and then over seven consecutive days of baseline, treatment, and follow-up. No effect on night activity was found after three weeks of treatment (MD= -20.60, 95% CI -46.52 to 5.32, p=.12; Figure 25) and after 3 weeks of follow-up (MD=-24.70, 95% CI -52.70 to 3.30, p=.08; Figure 26). Dowling 2005b and Dowling 2008 also measured the number of nighttime awakenings. Again, there was no effect on the number of nighttime awakenings after 10 weeks of treatment in either the morning bright light exposure (MD= -2.37, 95% CI -8.75 to 4.01, p=.47; Figure 27) or evening exposure (MD= -4.38, 95%CI -11.61, 2.86, p=.24; Figure 28).

Figure 23. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.20 Activity score (per night) at endpoint.

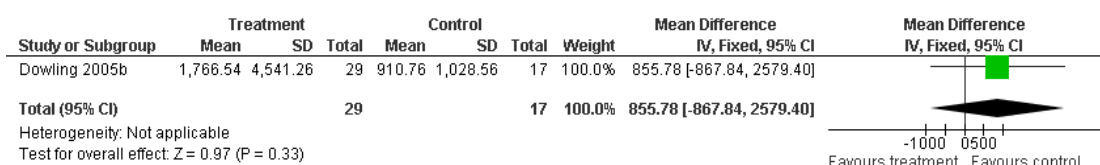


Figure 24. Forest plot of comparison: 2 Evening bright light vs control, outcome: 2.7 Activity score (per night) at endpoint.

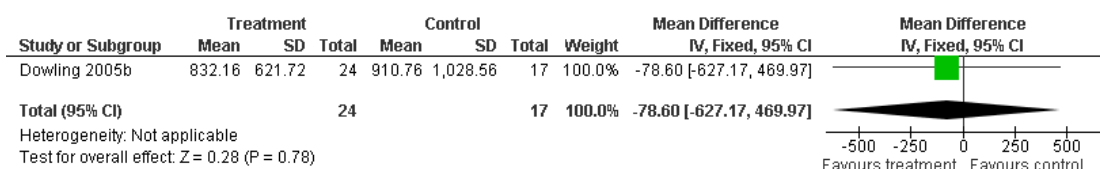


Figure 25. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.7 Nighttime activity counts (per night) at endpoint (after 3 weeks of treatment).

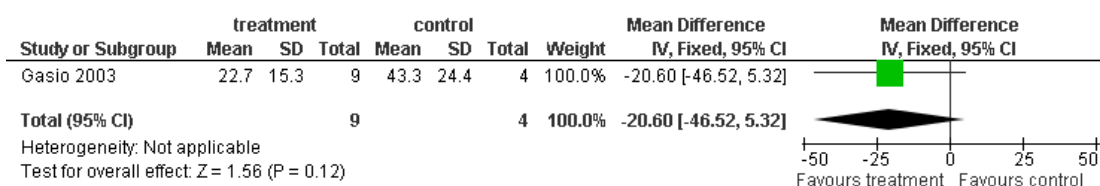


Figure 26. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.8 Nighttime activity counts (per night) at follow-up (3 weeks after treatment).

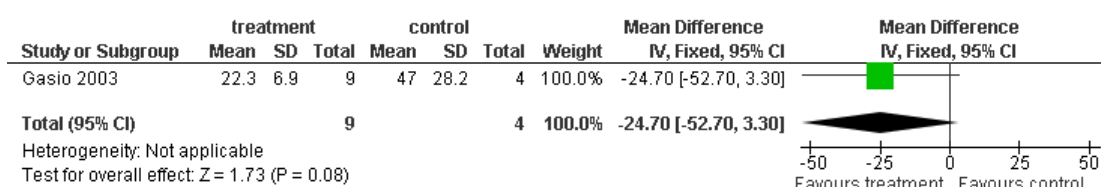


Figure 27. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.13 Number of night-time awakenings at endpoint.

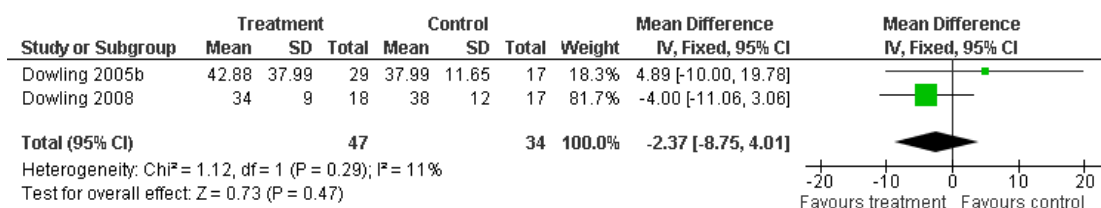
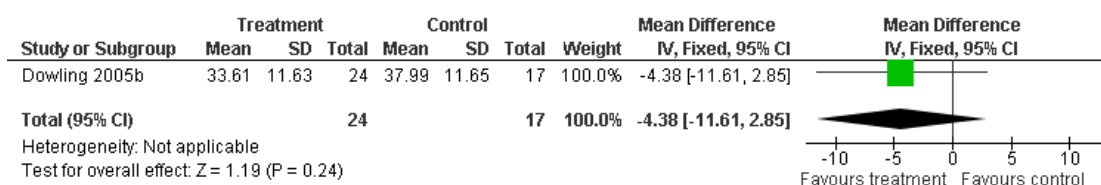


Figure 28. Forest plot of comparison: 2 Evening bright light vs control, outcome: 2.2 Number of nighttime awakenings at endpoint.



Behavioural Disturbances

Behavioural disturbances (e.g., agitation) were measured in five studies using several instruments: the ABRS (Ancoli-Israel 2003b), Behave-AD scale (Lyketsos 1999), NPI scale (Gasio 2003,

Dowling 2007) and CMAI (Riemersma 2008). In two studies (Ancoli-Israel 2003b; Dowling 2007) behavioural disturbances were compared between morning light therapy exposure and af-

ternoon/evening light therapy and assessed in the morning and evening shifts (Ancoli-Israel 2003b). The findings from Lyketsov 1999 could not be included in the analyses as data prior to the cross-over were requested on August 12, 2003, but have not yet been provided.

With light therapy administered during the morning or day time, behavioural disturbances measured by ABRS scores (Ancoli-Israel 2003b), NPI scores (Dowling 2007) and CMAI scores (Riemersma 2008) were pooled. The results revealed that light therapy administered during the morning or daytime had no effect on behavioural disturbances (SMD=-0.02, 95%CI -0.45 to

0.40, p=.91; Figure 29) following 10 to 50 days of light therapy. Similarly, no effect on behavioural disturbances was observed in the evening assessment following 10 days of treatment (MD=0.11, 95%CI -0.23 to 0.45, p=.52; Figure 30; Ancoli-Israel 2003b) after 5 days of follow-up measured in the morning (MD 0.02, 95%CI -0.23 to 0.27, p=.87; Figure 31; Ancoli-Israel 2003b), in the evening (MD 0.07, 95%CL -0.26, 0.40, p=.67; Figure 32; Ancoli-Israel 2003b), following one year of treatment (MD=-2.00, 95%CI -11.71 to 7.71, p=.69; Figure 33; Riemersma 2008), and after two years of light therapy (MD=-9.00, 95%CI -21.34 to 3.34, p=.15; Figure 34; Riemersma 2008).

Figure 29. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: I.14 Agitation at endpoint (NPI, ABRS, CMAI; morning assessment; 6-50 days).

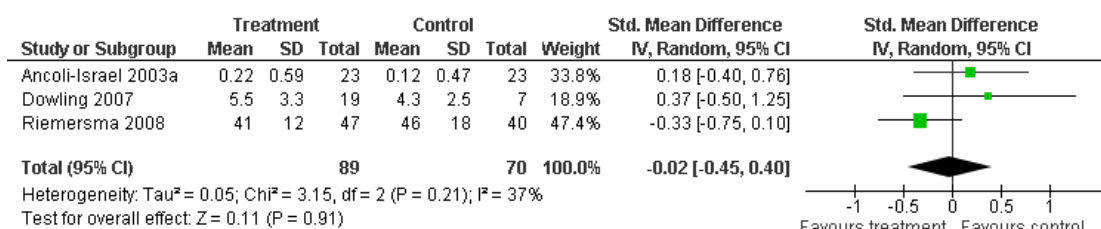


Figure 30. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: I.15 Agitation at endpoint (ABRS; evening assessment; 6-10 days).

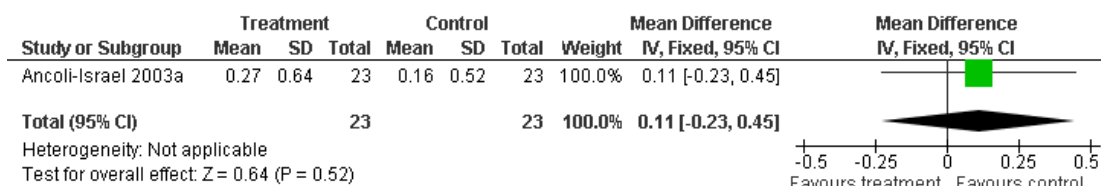


Figure 31. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: I.17 Behavioural disturbances at follow-up (ABRS; morning assessment; after 5 days).

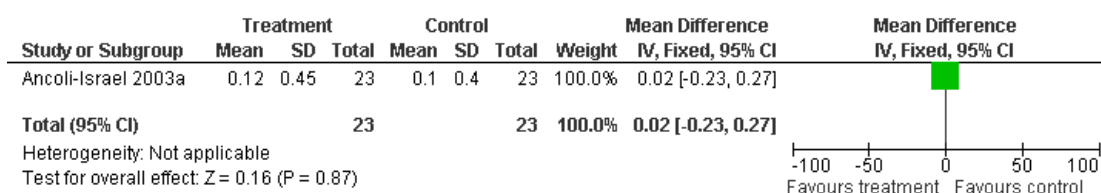


Figure 32. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: 1.18 Behavioural disturbances at follow-up (ABRS; evening assessment; after 5 days).

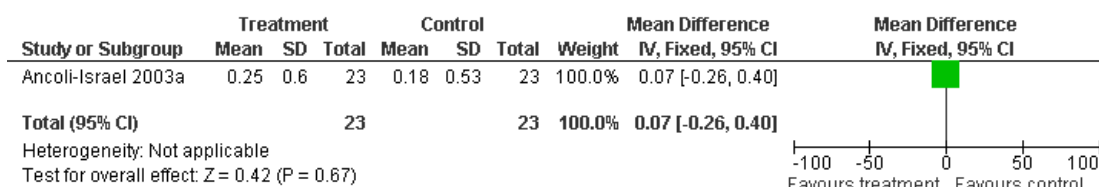


Figure 33. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: 1.16 Agitation at endpoint (CMAI; 1 year).

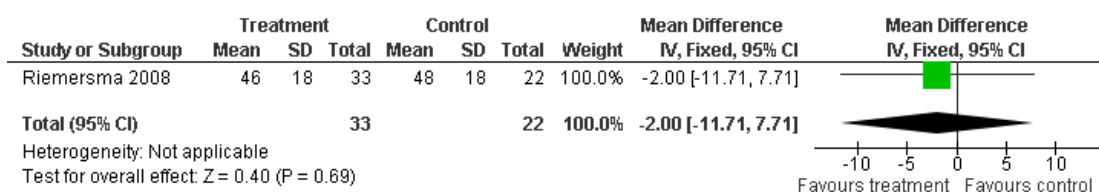
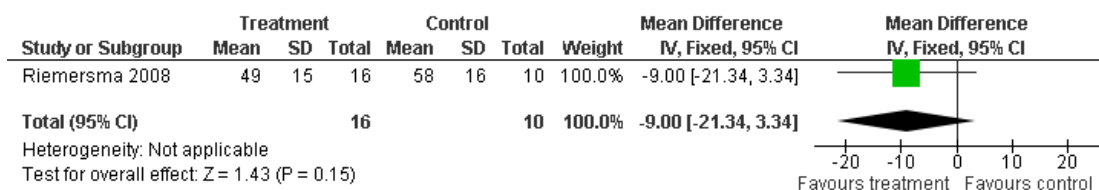


Figure 34. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: 1.19 Agitation at follow-up (CMAI; after 2 years).



To assess behavioural disturbances following the administration of afternoon or evening light therapy, ABRS scores (Ancoli-Israel 2003b) and NPI scores (Dowling 2007) were pooled. The results revealed that light therapy administered in the afternoon or evening had no effect on reducing behavioural disturbances when assessed during the morning (SMD=0.16, 95%CI -0.31 to

0.64, p=.50; Figure 35) following 10 to 50 days of light therapy (Ancoli-Israel 2003b; Dowling 2007) or when assessed during the evening (MD 0.07, 95%CI -0.26 to 0.40, p=.67; Figure 36) following 10 days of treatment (Ancoli-Israel 2003b). Similar results were found after five days of follow-up during morning

assessments (MD 0.10, 95%CI -0.16 to 0.36, p=.46; [Figure 37](#); [Ancoli-Israel 2003b](#)) and during evening assessments (MD 0.11, 95%CI -0.23 to 0.45, p=.53; [Figure 38](#); [Ancoli-Israel 2003b](#))

Figure 35. Forest plot of comparison: 2 Evening bright light vs control, outcome: 2.3 Agitation at endpoint (NPI, ABRS, CMAI; morning assessment; 6-50 days).

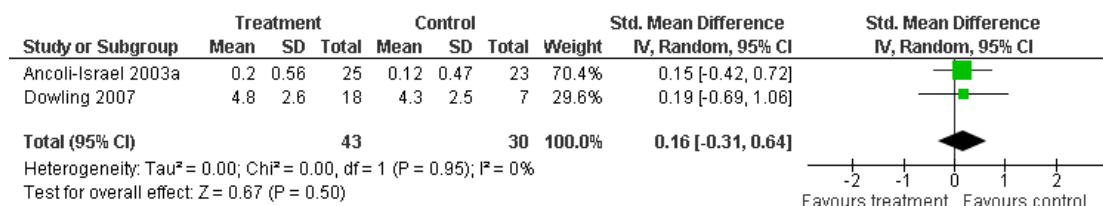


Figure 36. Forest plot of comparison: 2 Evening bright light vs control, outcome: 2.4 Agitation at endpoint (ABRS; evening assessment; 6-10 days).

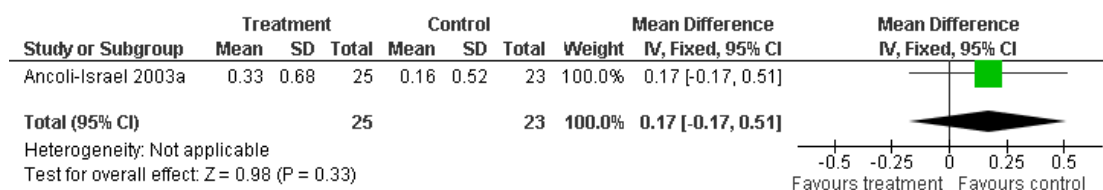


Figure 37. Forest plot of comparison: 2 Evening bright light vs control, outcome: 2.5 Agitation at follow-up (ABRS; morning assessment; 5 days).

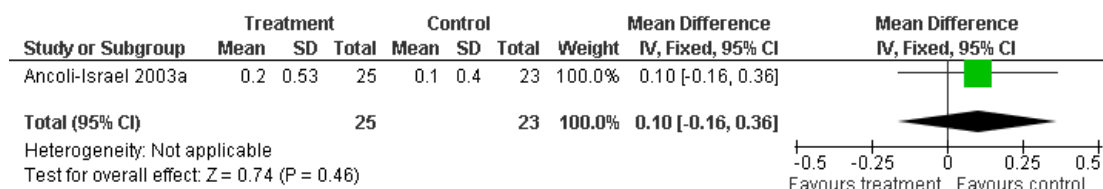
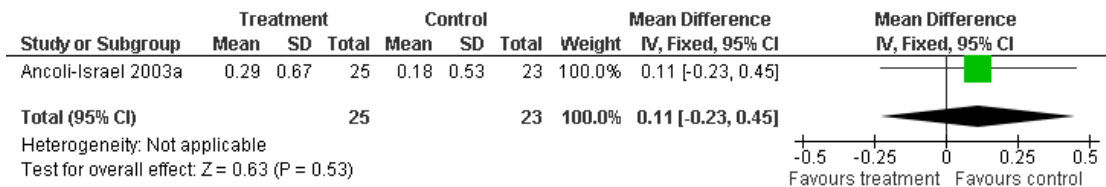


Figure 38. Forest plot of comparison: 2 Evening bright light vs control, outcome: 2.6 Agitation at follow-up (ABRS; evening assessment; 5 days).



Psychiatric Disturbances

Two studies (Dowling 2007, Riemersma 2008) used the Neuropsychiatric Inventory (NPI) that comprises ten behavioral domains: delusions, hallucinations, dysphoria, anxiety, agitation/aggression, euphoria, disinhibition, irritability/lability, apathy, and aberrant motor activity to measure psychiatric disturbances. No effect on changing psychiatric disturbances was observed after 42 to 50 days of treatment (MD=1.77, 95%CI -6.34 to 9.87, p=.67; Figure 39), after one year (MD=-0.30, 95%CI -2.73 to 2.13, p=.81; Figure 40), and after 2 years of light therapy (MD= -3.30,

95%CI -7.03 to 0.43, p=.08; Figure 41). In addition, there was no effect when light therapy was administered in the afternoon (MD=7.90, 95%CI, -0.46 to 16.26, p=.06; Figure 42 Dowling 2007). Gasio 2003 used the NPI to examine psychiatric symptoms following 3 weeks of dawn-dusk simulation or dim red light therapy. No effect was observed following the treatment (MD=-3.19, 95%CI -9.83 to 3.45, p=.35; Figure 43) and after 3 weeks of follow-up (MD =-4.17, 95%CI -13.37 to 5.03, p=.37; Figure 44; Gasio 2003).

Figure 39. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.21 Psychiatric symptoms at endpoint (NPI domain subscores; 42-50 days).

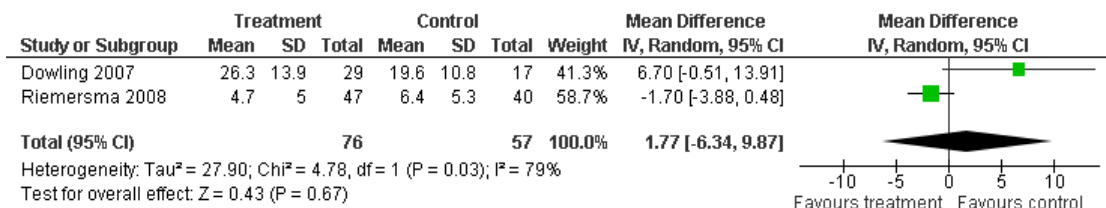


Figure 40. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.22 Psychiatric symptoms at endpoint (NPI domain subscores; 1 year).

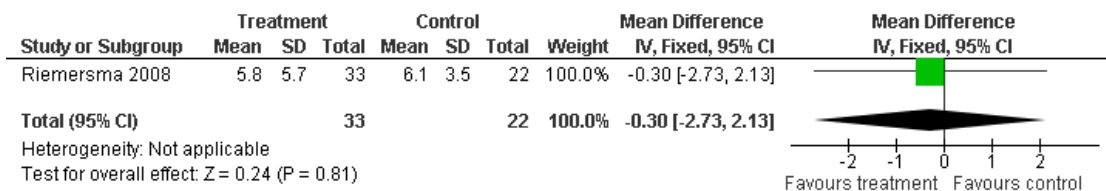


Figure 41. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.23 Psychiatric symptoms at endpoint (NPI domain subscores; 2 years).

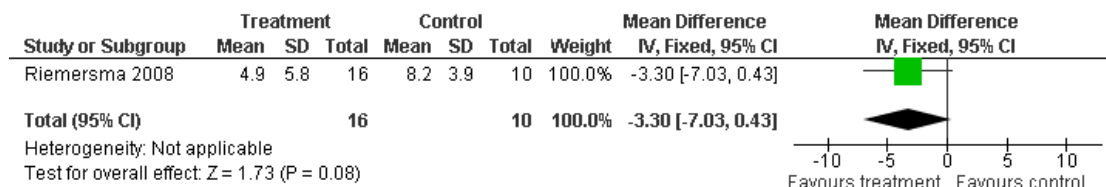


Figure 42. Forest plot of comparison: 2 Evening bright light vs control, outcome: 2.8 Psychiatric symptoms at endpoint (NPI domain scores).

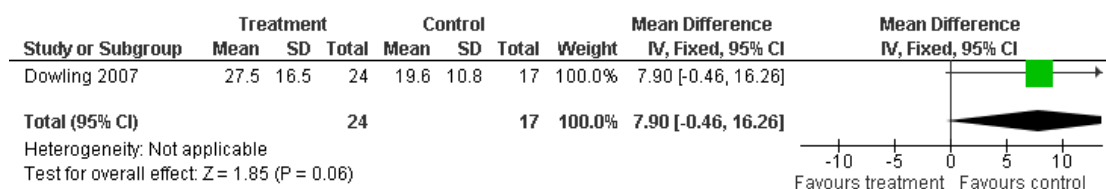


Figure 43. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.9 Psychiatric symptoms at endpoint (after 3 weeks of treatment, NPI domain subscores)).

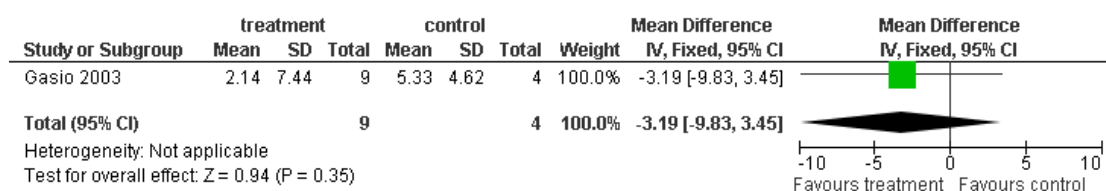
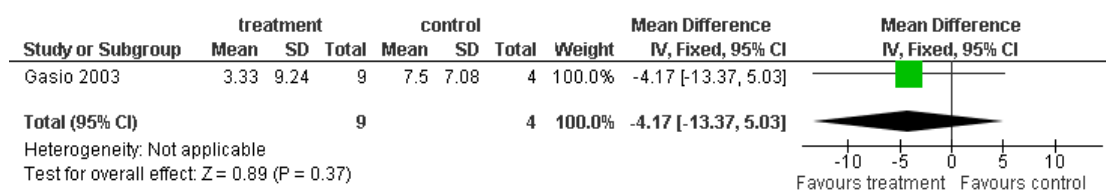


Figure 44. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.10 Psychiatirc symptoms at follow-up (3 weeks after treatment, NPI domain scores)).



Four studies measured depression: Dowling 2007 used the depression/dysphoria domain of the NPI-Nursing Home version (NPI-NH), Gasio 2003 used the Geriatric Depression Scale (GDS), Lyketsos 1999 used the Cornell Scale for Depression in Dementia (CSDD), and Riemersma 2008 used the CSDD. Lyketsos 1999 reported that no significant differences in scores of depression were found between groups at each time point. However, raw data were not reported and could not be retrieved as the data were archived (personal communication, Constantine Lyketsos, May 31, 2003). Pooled data (Dowling 2007; Riemersma 2008) revealed no effect on depression following 42 to 50 days of light therapy (SMD=0.12, 95%CI 0.06 to 1.30, p=.84; Figure 45). In

addition, Riemersma 2008 data revealed no effect on depression using CSDD scores at 1 year (MD=-.30, 95%CI -4.36 to 3.76, p=.88; Figure 46) and after 2 years of treatment (MD -4.40, 95%CI -10.82 to 2.02, p=.18; Figure 47). However, administering the light therapy in the afternoon resulted in an effect after 50 days of treatment (MD=3.20, 95% CI 0.86 to 5.51, p=.007; Figure 48), favouring the control group (Dowling 2007). These results should be viewed with caution due to the small sample size (n=17). Analysis of the data provided by Gasio 2003 revealed no effect on depression scores after 3 weeks of treatment (MD=-0.82, 95%CI -4.33 to 2.69, p=.65; Figure 49) or at follow-up (MD= -1.29, 95%CL -3.99, 1.41, p=.35; Figure 50).

Figure 45. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: I.24 Depression/dysphoria (CSDD, NPI subscale; 42-50 days).

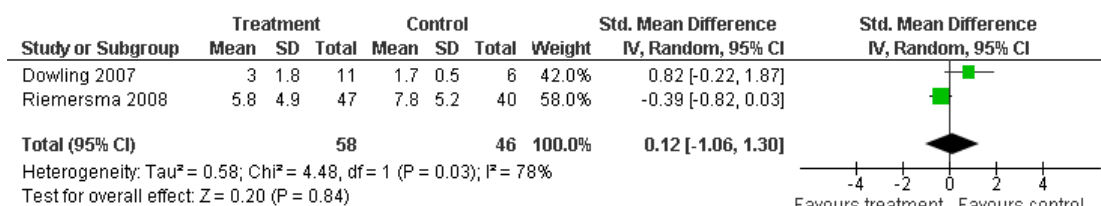


Figure 46. Forest plot of comparison: I Morning/daytime bright light vs control, outcome: I.25 Depression (CSDD; 1 year).

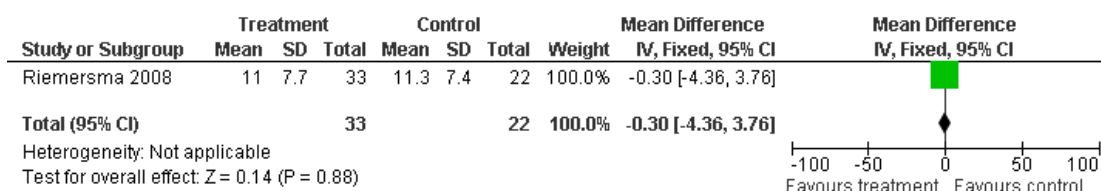


Figure 47. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.26 Depression (CSDD; 2 years).

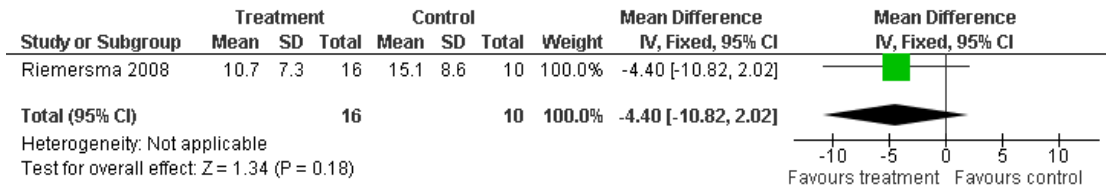


Figure 48. Forest plot of comparison: 2 Evening bright light vs control, outcome: 2.9 Depression/Dysphoria at endpoint (NPI domain subscale).

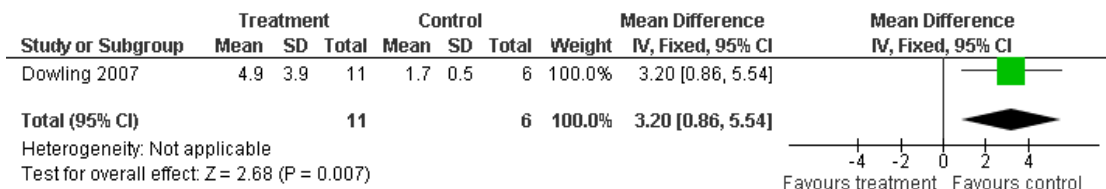


Figure 49. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.11 Depression at endpoint (after 3 weeks of treatment, GDS scores).

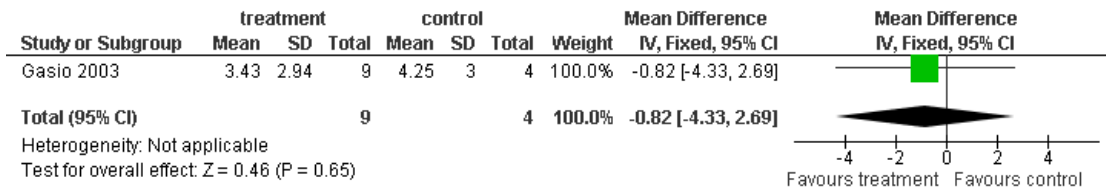
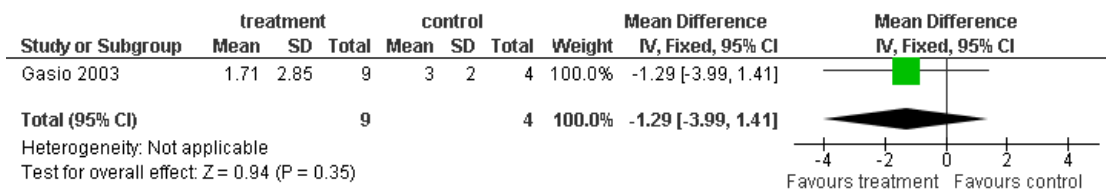


Figure 50. Forest plot of comparison: 3 Dawn-dusk simulation with bright white light vs dawn-dusk simulation with dim red light, outcome: 3.12 Depression at follow-up (3 weeks after treatment, GDS scores).



Apathy and indifference were measured using a domain of the NPI-NH following 50 days of treatment (Dowling 2007). There was no effect on apathy or indifference in either the morning administration of bright light (MD=1.00, 95%CI -2.21 to 4.21, p=.54; Figure 51) or afternoon administration (MD=0.40, 95%CI -3.00 to 3.80, p=.82; Figure 52).

Figure 51. Forest plot of comparison: 1 Morning/daytime bright light vs control, outcome: 1.27 Apathy/indifference at endpoint (NPI subscale).

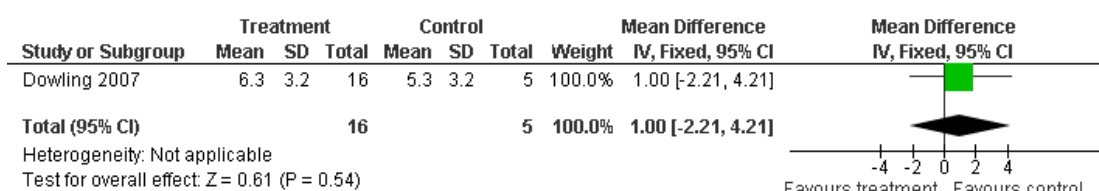
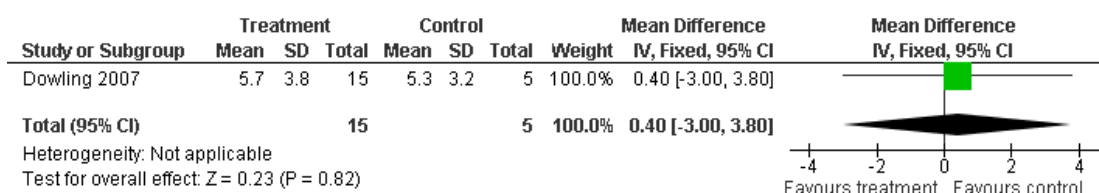


Figure 52. Forest plot of comparison: 2 Evening bright light vs control, outcome: 2.10 Apathy/Indifference at endpoint (NPI domain subscale).



DISCUSSION

This review of the effects of light therapy on cognition, function, sleep, behavioural disturbances, and psychiatric disturbances associated with dementia revealed little significant evidence of benefit. Light therapy may have an effect on two outcomes of interest. The Riemersma 2008 study revealed that light therapy had a positive effect on the treatment group in attenuating the increase in functional limitations after six weeks and after two years of light therapy. The sample size was adequate at six weeks (n=87) but by two years the sample size was only 26 participants. The Dowling 2007 study revealed that the lack of afternoon bright light therapy improved depression in the control group. However, these results should be viewed with caution as the sample size was also small (n=17). No significant evidence was found that light therapy

decreased the decline in cognition, shortened sleep latency time, increased nocturnal sleep time, decreased night-time activity, decreased behavioural disturbances, or improved psychiatric symptoms. No RCTs were retrieved that measured the other outcomes of interest, namely changes in rates of institutionalization or impact on cost of care. Only one trial (Riemersma 2008) examined adverse effects of light therapy. No adverse effects were reported, on the contrary, light therapy significantly reduced the ratings of irritability, dizziness, headache, constipation, and inability to sleep (Riemersma 2008).

The non-significant results may have been related to small sample sizes that contribute to insufficient power to detect a difference, if one is present. Notable exceptions were the Ancoli-Israel 2003a and Ancoli-Israel 2003b trials that included 92 participants and the Riemersma 2008 study that included 94 participants. Clearly further research with larger sample sizes is required that examines

all of the outcomes of interest.

Other methodological inadequacies that may contribute to bias were related to sequence generation and concealed allocation to groups. It was difficult to determine these processes for most of the trials included. Only one study [Riemersma 2008](#) reported using a computer generated randomization technique, the only safe method of sequence generation and concealed allocation. Inadequate concealment includes randomization by use of case record numbers, dates of birth, admission date, day of the week, and any procedure transparent before allocation such as an open list of random numbers ([Wild 2003](#)). Because selection bias and allocation bias are a concern, future research should use a randomized controlled trial design and ensure that participants are truly randomized by employing a computer generated randomization technique. Clinical researchers need to make a practice of providing the information and data required for a systematic review in published articles or be willing to share this information with reviewers when contacted. In addition, data must be reported for each group and, if possible, for individuals within groups.

Two studies ([Lyketos 1999](#); [Mishima 1998](#)) used cross-over designs and did not conduct analyses appropriate to a paired design. Although participants received no light treatment for one week prior to being crossed over to the other group, it is not known if there is a carry-over effect from the two to four weeks of exposure to the light therapy. Some studies (e.g. [Ancoli-Israel 2003a](#)) suggest that the effects of light therapy on nocturnal sleep persist beyond the treatment, while [McCurry 2000](#) concluded that the benefits to sleep from increased bright light decline almost immediately once exposure is discontinued. Until the evidence is stronger, participants should not be regarded as generating independent data in the two phases of a cross-over design.

Another plausible reason for the lack of significant effect of light therapy was the heterogeneity within several of the trials in terms of participants' diagnosis and severity of dementia. Three trials ([Ancoli-Israel 2003a](#), [Ancoli-Israel 2003b](#); [Dowling 2005b](#); [Dowling 2008](#)) were notable exceptions as they included only participants with Alzheimer's disease. Individuals with Vascular Dementia have heterogeneous brain pathology; their response to light therapy may depend on the areas in which Ischaemic damage has occurred. The response to light therapy of individuals with scattered lesions of Vascular Dementia ([Mishima 1998](#)) or with frontotemporal degeneration ([Harper 2001](#)) may differ from that of people with Alzheimer's disease who commonly have damage to the hippocampi and medial temporal lobes of the brain. Investigators need to be sensitive to the importance of controlling for these differences in pathology when designing studies of light therapy. Differences in severity of dementia may also influence the results. Unfortunately, because of the small sample sizes and small number in included trials, sub-group analyses could not be conducted. Thus, given the methodological shortcomings in the published studies, they do not constitute good evidence that light

therapy is ineffective.

[Gasio 2003](#) used dawn-dusk simulated light therapy that exposed the participants to natural amounts of light at dawn and dusk and [Riemersma 2008](#) used ceiling-mounted light fixtures with Plexiglas diffusers containing an equal amount of Philips TLD 840 and 940 fluorescent tubes in the common living room. These approaches to enhancing light exposure for long term care residents is less invasive and demanding of the residents than the traditional Brite-LiteTM box used in the remaining studies. Use of a Brite LiteTM box requires participants to sit in front of the box for approximately two hours, which they may find difficult, so noncompliance may be a problem. However, the intensity (<400 lux) and duration of the natural light at dawn and dusk may be insufficient to be effective in changing sleep, behaviour and/or psychiatric disturbances. Bright light (> 2,000 lux) appears to synchronize circadian rhythms ([Wever 1983](#)). Healthy older adults on average were found to be exposed (naturally) to 60 minutes of bright light a day. People with Alzheimer's disease living at home were exposed to 29 minutes a day (on average) ([Campbell 1988](#)), while institutionalized residents with dementia spent a median of 10.5 minutes per day (mean=34 minutes, SD=63, range=0-314) exposed to light above 1000 lux and a median of 4 minutes (mean=19 minutes, SD=39, range=0-242) per day in light over 2000 lux ([Shocat 2000](#)). A systematic review that includes not only RCTs but also non-RCTs (e.g., [Sloane 2007](#)) of studies that examine increasing natural ambient light exposure within long term care facilities is needed ([McCurry 2000](#)).

The best time of day to offer light therapy remains unknown although trials that administered light therapy in the morning, afternoon, evening, and all day were included in this Review. In older adults in general, circadian rhythms are phase-advanced (i.e., rhythms are shifted to an atypical early time resulting in falling asleep or waking up earlier than was habitual in earlier life) ([Touitou 2000](#)). However, individuals with Alzheimer's disease have been reported to have phase-delayed activity resulting in rhythms that are shifted to unusually late times ([Satlin 2000](#)). Exposure to morning bright light has been shown to advance circadian rhythms and thus normalize people with a phase delay, whereas evening bright light may delay circadian rhythms and normalize those with advanced rhythms ([Campbell 1995](#)). However, other studies (e.g. [Ancoli-Israel 2002](#), [Satlin 1992](#)) have not supported the expected direction of change in individuals with Alzheimer's disease. [Ancoli-Israel 2003a](#), included in this Review, hypothesized that the timing of light required to achieve a phase advance or phase delay may be different in people with Alzheimer's disease owing to the advanced deterioration of the SCN, and recommends increasing light exposure throughout the day and evening. Clearly further research is required in this area.

AUTHORS' CONCLUSIONS

Implications for practice

There is insufficient evidence of the effectiveness of light therapy in managing sleep, functional, behavioural, or psychiatric disturbances associated with dementia.

Implications for research

As there is a possible rationale for light therapy in managing important manifestations of dementia, further and better-designed research is required.

Studies should incorporate:

- 1) a randomized controlled parallel-group design with statistically appropriate analysis,
- 2) a cluster randomized trial if a randomized controlled parallel-group design is not possible,
- 3) a computer generated randomization technique,
- 4) a sample size with sufficient power to detect an effect of clinically significant magnitude, and

5) blinded and objective outcome ratings.

Further research is necessary to identify appropriate illumination intensity, frequency, interval, time of day and length of intervention for individuals with different types and severity of dementia. Exploring different light therapy approaches (e.g., dawn-dusk simulation, light visors worn on heads, ambient light) are also required to ensure that the light therapy is acceptable to the participants. Unless the participants are comfortable with the light therapy, there will be low compliance. Outcomes that contribute to the quality of life of those with dementia should be examined as well as potential adverse effects of light therapy. The cost implications of light therapy also need to be examined.

ACKNOWLEDGEMENTS

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CHARACTERISTICS OF STUDIES

Characteristics of included studies [ordered by study ID]

Ancoli-Israel 2003a

Methods	Randomly assigned (although the selection process is unclear) to morning bright light, evening bright light, or morning dim red light (control), single blind (although nursing and research staff were told that both the white and red light conditions were expected to show improvement and the study was examining which colour light would be better)	
Participants	Country: USA 92 nursing home residents (63 women, 29 men); mean age 82.3 years (SD 7.6, range 61-99); MMSE mean=5.7 (SD 5.6, range 0-22)	
Interventions	Apollo "Brite-Lite" box placed 1m from resident 1. Bright light > 2500 Lux: time of day 930-1130 or 1730-1930 2. Dim, red light < 300 Lux: time of day 930-1130 Received treatment daily Baseline data: 3 days Duration of treatment: 10 days Follow-up: 5 days post-treatment	
Outcomes	Wake after sleep onset (WASO) Total sleep time (TST) Percent sleep Percent wake Number of nighttime awakenings Average length of nighttime awakenings Number of daytime naps Duration of naps Length of time between naps	
Notes		
<i>Risk of bias</i>		
Item	Authors' judgement	Description
Allocation concealment?	Yes	
Blinding of assessors?	Yes	

Ancoli-Israel 2003b

Methods	Randomly assigned (although the selection process is unclear) by block-stratified randomization to morning bright light, evening bright light, or morning dim red light (control). Residents were stratified by time of agitation (primarily in the morning, evening or all day) Single blind although nursing and research staff were told that both the white and red light conditions were expected to show improvement and the study was examining which colour light would be better	
Participants	Country: USA 92 nursing home residents (63 women, 29 men); mean age 82.3 years (SD 7.6, range 61-99); MMSE mean=5.7 (SD 5.6, range 0-22)	
Interventions	Apollo "Brite-Lite" box placed 1m from resident 1. Bright light > 2500 Lux: time of day 930-1130 or 1730-1930 2. Dim, red light < 300 Lux: time of day 930-1130 Received treatment daily Baseline data: 3 days Duration of treatment: 10 days Follow-up: 5 days post-treatment	
Outcomes	Agitation assessed using the ABRS and CMAI	
Notes		
Risk of bias		
Item	Authors' judgement	Description
Allocation concealment?	Yes	
Blinding of assessors?	Yes	

Dowling 2005b

Methods	Phase one: randomly assigned to morning bright light or control; Phase two: randomly assigned to morning bright light or afternoon bright light	
Participants	Country: USA 70 nursing home residents (57 women, 13 men), mean age 84(SD 10) ranging from 58 to 98, MMSE 0-23 (mean=7, SD 7)	
Interventions	Bright light exposure >2,500 lux: morning (9:30-10:30am), afternoon (3:30-4:30pm) or supplemented using Apollo Brite Lite IV box placed at least 4 feet from resident Frequency: Daily, Monday through Friday Duration: 10 weeks	
Outcomes	Sleep efficiency, night sleep time, night wake time, number of nighttime awakenings, day wake time, 24-hour rest-activity rhythm	
Notes		

Dowling 2005b (Continued)

<i>Risk of bias</i>		
Item	Authors' judgement	Description
Allocation concealment?	Yes	
Blinding of assessors?	No	

Dowling 2007

Methods	Randomly assigned to morning bright light, afternoon bright light or usual indoor light (control), single blind	
Participants	Country: USA 70 nursing home residents (57 women, 13 men), mean age 84 (SD 10) ranging from 58 to 98, MMSE 0-23 (mean=7, SD 7)	
Interventions	Bright light exposure >2,500 lux: morning (9:30-10:30am), afternoon (3:30-4:30pm) or supplemented using Apollo Brite Lite IV box placed at least 4 feet from resident Frequency: Daily, Monday through Friday Duration: 10 weeks	
Outcomes	NPI-NH, occupational disruptiveness scores	
Notes		

Risk of bias

Item	Authors' judgement	Description
Allocation concealment?	Yes	

Dowling 2008

Methods	Randomly assigned to one of three groups: morning light and melatonin or placebo or usual indoor light (control). Only reported on blinding to melatonin group	
Participants	Country: USA 50 nursing home residents (43 women, 7 men), mean age 86 (SD 8), subjects in the control group were significantly younger ($82_{\pm 10}$) than subjects in the light placebo group ($89_{\pm 7}$), thus age was centred on the grand mean. MMSE mean=9.3 (SD 7.9). All subjects diagnosed with Alzheimer's Disease	
Interventions	Bright light exposure >2,500 lux in gaze direction in the morning (9:30-10:30am). Ambient light was supplemented using Apollo Brite Lite IV box placed 30-34 inches from resident Frequency: Daily, Monday through Friday Duration: 10 weeks	

Dowling 2008 (Continued)

Outcomes	Night sleep time, night wake bouts, number of nighttime awakenings, day sleep time, day/night sleep ratio, 24-hour rest-activity rhythm	
Notes		
Risk of bias		
Item	Authors' judgement	Description
Allocation concealment?	Yes	
Blinding of assessors?	Unclear	Reported that study staff, nursing home staff and subjects were blinded to melatonin treatment group. No mention of light therapy group

Gasio 2003

Methods	Randomly assigned to dawn-dusk simulation (DDS) light therapy or 'placebo' dim red light (DRL), single-blind	
Participants	Country: Switzerland 13 nursing home residents, (12 women, 1 man) mean age 85.6 years Dawn-Dusk Simulation Age mean 86.8 (SD 4.5) MMSE mean 13.8 (SD 5.9) Probable AD (n=7) Probable Vascular (n=2) Dim Red Light Age mean 83.0 (SD 5.2) MMSE mean 14.3 (SD 4.1) Probable AD (n=3) Lewy Body (n=1)	
Interventions	Dawn-Dusk Simulation using an overhead halogen lamp placed behind a diffusing membrane behind the resident's bed simulating a naturalistic form of light therapy 1. DDS max 400 Lux morning and evening 2. DRL < 5 Lux morning and evening Treatment time varied to mimic the duration and latitude of dawn and dusk Baseline data: 3 weeks Duration of the treatment: 3 weeks Follow-up: 3 weeks post-treatment	
Outcomes	MMSE NPI-NH GDS CERAD Sleep logs: measured time of going to bed and getting up	

Gasio 2003 (Continued)

Notes	
Risk of bias	
Item	Authors' judgement
Adequate sequence generation?	Yes

Graf 2001

Methods	Randomly assigned (although rated as inadequate) to dim light (control) or bright light (experimental), single-blind
Participants	Country: Austria 23 nursing home residents, (proportions of male and female not stated), mean age 81.6 years (range 65-94), diagnosed with AD (n=11) or Vascular Dementia (n=12)
Interventions	Bright light placed 90 cm from resident 1. Bright light = 3000 Lux: time of day 1700-1900 2. Dim, red dim light < 100 Lux: time of day 1700-1900 Received treatment daily. Baseline data: morning of initiation of study Duration of treatment: 10 days No follow-up
Outcomes	MMSE
Notes	
Risk of bias	
Item	Authors' judgement
Blinding of assessors?	Yes
Adequate sequence generation?	No

Lyketos 1999

Methods	Randomized, single-blind, crossover design
Participants	Country: USA 15 nursing home residents (14 women, 1 man) mean age 80.8 (SD 8.7). DSM-IV criteria for AD (n=12) or Vascular Dementia (n=3) MMSE mean: 6.4 (SD 6.8) Behave-AD: > 4 points

Lyketsos 1999 (Continued)

Interventions	Bright light placed 3 feet from resident 1. Bright light = 10,000 Lux: time of day morning 2. Dim light = Lux not specified: time of day morning Received treatment daily for 1 hour. Baseline: 1 week Duration of treatment: 4 weeks Follow-up: one week post-treatment Then received other condition for 4 weeks.	
Outcomes	Behave-AD CSDD Mean hours of total nocturnal sleep	
Notes		
<i>Risk of bias</i>		
Item	Authors' judgement	Description
Allocation concealment?	Yes	
Blinding of assessors?	Yes	
Adequate sequence generation?	Yes	

Mishima 1998

Methods	Randomized (although process unclear), single-blind, crossover design	
Participants	Country: Japan 22 nursing home residents, (13 women, 9 men) mean age 79.6 years MRI, CT, & DSM-IV criteria for AD (n=10; mean age: 78 years; MMSE: mean 9, range 3-17 or Vascular Dementia (n=12; mean age: 81 years; MMSE: mean 8, range 3-14)	
Interventions	Bright light placed 90cm from resident 1. Bright light = 5,000-8,000 Lux: time of day 900-1100 2. Dim light = 300 Lux: time of day 900-1100 Received treatment daily. Baseline: 1 week Duration of treatment: 2 weeks. Follow-up: 1 week Interval between conditions: at least 4 weeks	
Outcomes	Average daily total activity Average daytime activity Average nighttime activity Percentage of average nighttime activity to average daily total activity	
Notes		

Riemersma 2008

Methods	Double-blind, placebo-controlled randomized multi-centre trial
Participants	Country: Netherlands 94 nursing home patients (85 women, 9 men), mean age 85, 59 Alzheimer's diagnosis (NINCDS-ADRDA)
Interventions	Ceiling-mounted light fixtures with Plexiglas diffusers containing an equal amount of Philips TLD 840 and 940 florescent tubes in the common living room (aimed exposure of ± 1000 lux) Duration: 3.5 years
Outcomes	Sleep duration, latency, and efficiency MMSE CSDD PGCARS NI-ADL
Notes	

Risk of bias

Item	Authors' judgement	Description
Allocation concealment?	Yes	
Blinding of assessors?	Yes	
Adequate sequence generation?	Yes	

Characteristics of excluded studies [ordered by study ID]

Study	Reason for exclusion
Abegg 1993	Not a randomized controlled trial design.
Ancoli-Israel 1997	Not a randomized controlled trial design.
Ancoli-Israel 2002	A more recent version of this study with a larger sample size is reported in Ancoli-Israel 2003a
Colenda 1997	Not a randomized controlled trial design.
Dawson 1999	Not a randomized controlled trial design.
Dowling 2005a	Preliminary results. Full study reported in Dowling 2005b.
Fetveit 2003	Not a randomized controlled trial design.

(Continued)

Haffmans 2001	Not a randomized controlled trial design.
Hickman 2007	Cross-over trial not randomized.
Hozumi 1990	Not a randomized controlled trial design.
Ito 1999	Not a randomized controlled trial design.
Ito 2001	Not a randomized controlled trial design.
Kobayashi 2001	Not a randomized controlled trial design.
Koyama 1999	Not a randomized controlled trial design.
Lovell 1995	Not a randomized controlled trial design.
McCurry 2005	Light therapy not the only group difference.
McCurry 2006	Light therapy not the only group difference.
Mishima 1994	Not a randomized controlled trial design.
Mishima 2000	Not a randomized controlled trial design.
Okawa 1989	Not a randomized controlled trial design.
Okawa 1999a	Not a randomized controlled trial design.
Okawa 1999b	Did not measure severity of behaviour.
Okumoto 1998	Not a randomized controlled trial design.
Rheume 1998	Not a randomized controlled trial design.
Riemersma 2001	Not a randomized controlled trial design.
Satlin 1992	Not a randomized controlled trial design.
Skjerve 2004	Not a randomized controlled trial design.
Sloane 2007	Cross-over not randomized.
Thorpe 2000	Not a randomized controlled trial design.
van Someren 1997	Not a randomized controlled trial design.

(Continued)

Yamadera 2000	Not a randomized controlled trial design. All participants received light therapy
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Characteristics of ongoing studies [ordered by study ID]

Byrne 2000

Trial name or title	A randomised controlled trial of light therapy for agitation and sleep disturbance in symptoms of dementia
Methods	
Participants	Country: UK People with dementia in residential and nursing care
Interventions	1. Light therapy (dose to be determined)
Outcomes	Improvement in behavioural disturbances and sleep
Starting date	Ongoing in 2000
Contact information	Dr. J. Byrne South Manchester University Hospitals NHS Trust Dept of Mental Illness Withington Hospital Nell Lane Manchester M20 2LR E-mail: a-day@man.ac.uk Amanda Gaunt, Assistant: e-mail Amanda.Gaunt@manchester.ac.uk Feb. 2009
Notes	End date of trial was 31/12/2001 Study report requested June 4, 2003, September 15, 2003 and February 2009. Study will be published in International Psychogeriatrics, probably in August 2009. "Forthcoming" article may be available on the journal's website in April 2009

Dimond 1999

Trial name or title	Light therapy and agitated behavior in dementia
Methods	
Participants	Country: USA 63 long-term care residents in 8 Special Care Units
Interventions	1. Bright light evening 2. Placebo Two week washout was followed by crossover to light or placebo Duration of treatment was 14 days

Dimond 1999 (Continued)

Outcomes	ABRS
Starting date	Unknown
Contact information	M. Dimond School of Nursing, University of Washington, Seattle, WA 98195-7266 Tel: +1 206 685 3778 Email: dimond@u.washington.edu
Notes	Author will submit final paper/report when completed.

DATA AND ANALYSES

Comparison 1. Morning/daytime bright light vs control

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Cognition at endpoint (MMSE; 42 days)	1	87	Mean Difference (IV, Fixed, 95% CI)	1.20 [-1.56, 3.96]
2 Cognition at endpoint (MMSE; 1 year)	1	55	Mean Difference (IV, Fixed, 95% CI)	1.70 [-1.03, 4.43]
3 Cognition at endpoint (MMSE; 2 years)	1	26	Mean Difference (IV, Fixed, 95% CI)	3.60 [-1.05, 8.25]
4 Functional limitations at endpoint (NI-ADL; 42 days)	1	87	Mean Difference (IV, Fixed, 95% CI)	-5.0 [-9.87, -0.13]
5 Functional limitations at endpoint (NI-ADL; 1 year)	1	55	Mean Difference (IV, Fixed, 95% CI)	-5.0 [-11.16, 1.16]
6 Functional limitations at endpoint (NI-ADL; 2 years)	1	26	Mean Difference (IV, Fixed, 95% CI)	-16.0 [-26.21, -5.79]
7 Sleep onset latency (mins; 42 days)	1	87	Mean Difference (IV, Fixed, 95% CI)	6.0 [-12.34, 24.34]
8 Sleep onset latency (mins; 1 year)	1	55	Mean Difference (IV, Fixed, 95% CI)	5.0 [-24.79, 34.79]
9 Sleep onset latency (mins; 2 years)	1	26	Mean Difference (IV, Fixed, 95% CI)	10.0 [-11.33, 31.33]
10 Total sleep duration (mins; 6-42 days)	3	168	Mean Difference (IV, Fixed, 95% CI)	10.25 [-21.05, 41.54]
11 Total sleep duration (mins; 1 year)	1	55	Mean Difference (IV, Fixed, 95% CI)	-36.0 [-84.21, 12.21]
12 Total sleep duration (mins; 2 years)	1	26	Mean Difference (IV, Fixed, 95% CI)	-36.0 [-121.69, 49.69]
13 Activity score (per night) at endpoint	1	46	Mean Difference (IV, Fixed, 95% CI)	855.78 [-867.84, 2579.40]
14 Number of night-time awakenings at endpoint	2	81	Mean Difference (IV, Fixed, 95% CI)	-2.37 [-8.75, 4.01]
15 Behavioural disturbances at endpoint (NPI, ABRs, CMAI; morning assessment; 10-50 days)	3	159	Std. Mean Difference (IV, Random, 95% CI)	-0.02 [-0.45, 0.40]
16 Behavioural disturbances at endpoint (ABRS; evening assessment; 10 days)	1	46	Mean Difference (IV, Fixed, 95% CI)	0.11 [-0.23, 0.45]
17 Behavioural disturbances at follow-up (ABRS; morning assessment; after 5 days)	1	46	Mean Difference (IV, Fixed, 95% CI)	0.02 [-0.23, 0.27]
18 Behavioural disturbances at follow-up (ABRS; evening assessment; after 5 days)	1	46	Mean Difference (IV, Fixed, 95% CI)	0.07 [-0.26, 0.40]
19 Behavioural disturbances at endpoint (CMAI; 1 year)	1	55	Mean Difference (IV, Fixed, 95% CI)	-2.0 [-11.71, 7.71]

20 Behavioural disturbances at follow-up (CMAI; after 2 years)	1	26	Mean Difference (IV, Fixed, 95% CI)	-9.0 [-21.34, 3.34]
21 Psychiatric symptoms at endpoint (NPI total scores; 42-50 days)	2	133	Mean Difference (IV, Random, 95% CI)	1.77 [-6.34, 9.87]
22 Psychiatric symptoms at endpoint (NPI total scores; 1 year)	1	55	Mean Difference (IV, Fixed, 95% CI)	-0.30 [-2.73, 2.13]
23 Psychiatric symptoms at endpoint (NPI total scores; 2 years)	1	26	Mean Difference (IV, Fixed, 95% CI)	-3.30 [-7.03, 0.43]
24 Depression/dysphoria (CSDD, NPI subscale; 42-50 days)	2	104	Std. Mean Difference (IV, Random, 95% CI)	0.12 [-1.06, 1.30]
25 Depression (CSDD; 1 year)	1	55	Mean Difference (IV, Fixed, 95% CI)	-0.30 [-4.36, 3.76]
26 Depression (CSDD; 2 years)	1	26	Mean Difference (IV, Fixed, 95% CI)	-4.4 [-10.82, 2.02]
27 Apathy/indifference at endpoint (NPI subscale)	1	21	Mean Difference (IV, Fixed, 95% CI)	1.0 [-2.21, 4.21]

Comparison 2. Evening/afternoon bright light vs control

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Cognition at endpoint (MMSE; 10 days)	1	18	Mean Difference (IV, Fixed, 95% CI)	0.70 [-4.90, 6.30]
2 Total sleep duration (minutes) at endpoint	1	41	Mean Difference (IV, Fixed, 95% CI)	10.0 [-59.22, 79.22]
3 Activity score (per night) at endpoint	1	41	Mean Difference (IV, Fixed, 95% CI)	-78.60 [-627.17, 469.97]
4 Number of nighttime awakenings at endpoint	1	41	Mean Difference (IV, Fixed, 95% CI)	-4.38 [-11.61, 2.85]
5 Behavioural disturbances at endpoint (NPI, ABRIS; morning assessment; 10-50 days)	2	73	Std. Mean Difference (IV, Random, 95% CI)	0.16 [-0.31, 0.64]
6 Behavioural disturbances at endpoint (ABRS; evening assessment; 10 days)	1	48	Mean Difference (IV, Fixed, 95% CI)	0.17 [-0.17, 0.51]
7 Behavioural disturbances at follow-up (ABRS; morning assessment; 5 days)	1	48	Mean Difference (IV, Fixed, 95% CI)	0.1 [-0.16, 0.36]
8 Behavioural disturbances at follow-up (ABRS; evening assessment; 5 days)	1	48	Mean Difference (IV, Fixed, 95% CI)	0.11 [-0.23, 0.45]
9 Psychiatric symptoms at endpoint (NPI total scores; 50 days)	1	41	Mean Difference (IV, Fixed, 95% CI)	7.90 [-0.46, 16.26]

10 Depression/Dysphoria at endpoint (NPI domain subscale; 50 days)	1	17	Mean Difference (IV, Fixed, 95% CI)	3.2 [0.86, 5.54]
11 Apathy/Indifference at endpoint (NPI domain subscale; 50 days)	1	20	Mean Difference (IV, Fixed, 95% CI)	0.40 [-3.00, 3.80]

Comparison 3. Dawn-dusk simulation with bright white light vs dim red light

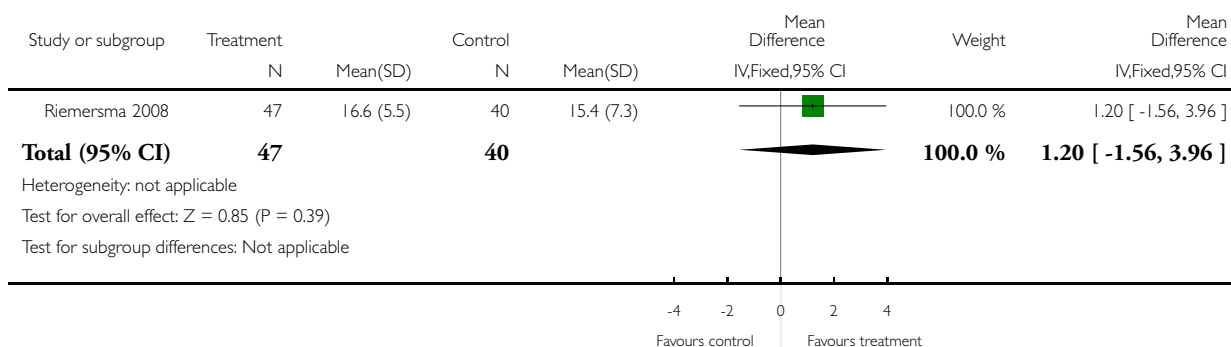
Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Cognition at endpoint (MMSE; 3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	0.46 [-14.14, 15.06]
2 Cognition at follow-up (MMSE; 3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	-0.5 [-10.67, 9.67]
3 Sleep onset latency (minutes) at endpoint (3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	-79.0 [-327.17, 169.17]
4 Sleep onset latency (minutes) at follow-up (3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	-62.0 [-216.55, 92.55]
5 Total sleep duration (minutes) at endpoint (3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	143.0 [-637.66, 923.66]
6 Total sleep duration (minutes) at follow-up (3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	110.0 [-77.22, 297.22]
7 Nighttime activity counts (per night) at endpoint (3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	-20.60 [-46.52, 5.32]
8 Nighttime activity counts (per night) at follow-up (3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	-24.7 [-52.70, 3.30]
9 Psychiatric symptoms at endpoint (NPI total scores; 3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	-3.19 [-9.83, 3.45]
10 Psychiatric symptoms at follow-up (NPI total scores; 3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	-4.17 [-13.37, 5.03]
11 Depression at endpoint (GDS; 3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	-0.82 [-4.33, 2.69]
12 Depression at follow-up (GDS; 3 weeks)	1	13	Mean Difference (IV, Fixed, 95% CI)	-1.29 [-3.99, 1.41]

Analysis 1.1. Comparison 1 Morning/daytime bright light vs control, Outcome 1 Cognition at endpoint (MMSE; 42 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 1 Cognition at endpoint (MMSE; 42 days)

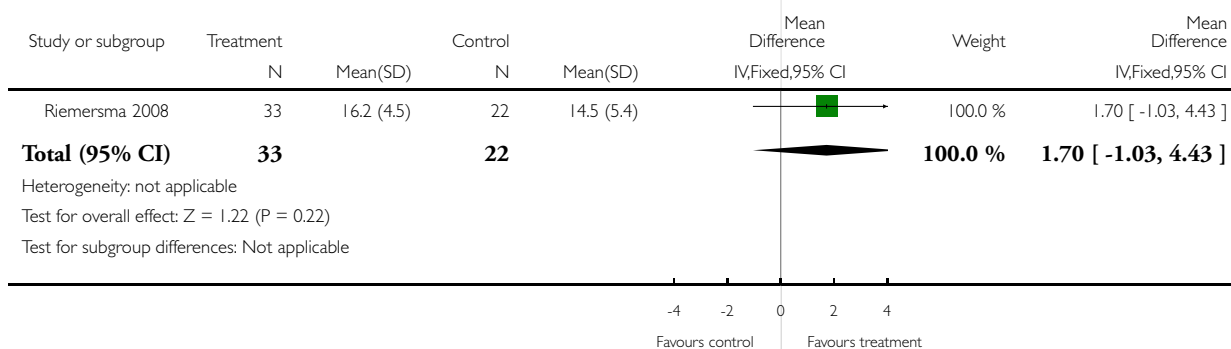


Analysis 1.2. Comparison 1 Morning/daytime bright light vs control, Outcome 2 Cognition at endpoint (MMSE; 1 year).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 2 Cognition at endpoint (MMSE; 1 year)

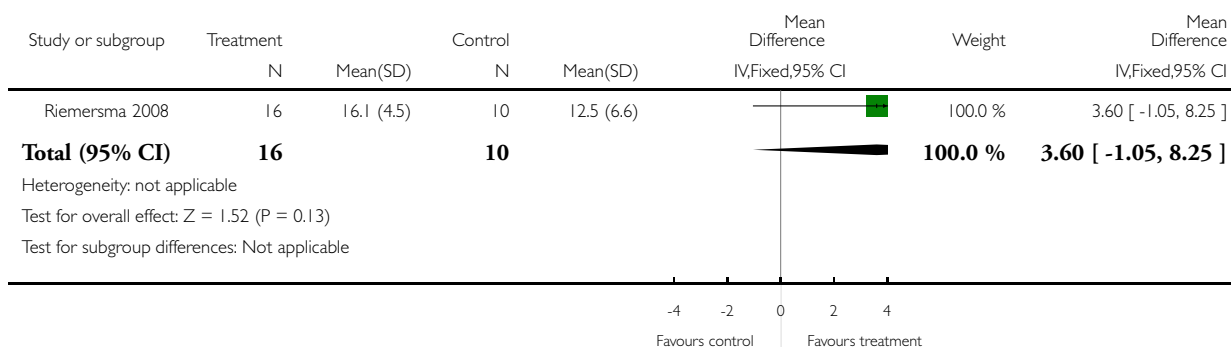


Analysis I.3. Comparison I Morning/daytime bright light vs control, Outcome 3 Cognition at endpoint (MMSE; 2 years).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: I Morning/daytime bright light vs control

Outcome: 3 Cognition at endpoint (MMSE; 2 years)

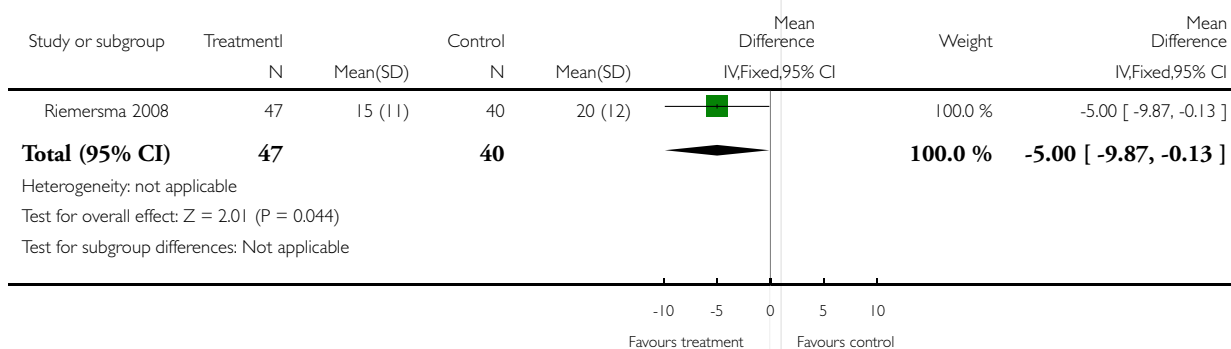


Analysis I.4. Comparison I Morning/daytime bright light vs control, Outcome 4 Functional limitations at endpoint (NI-ADL; 42 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: I Morning/daytime bright light vs control

Outcome: 4 Functional limitations at endpoint (NI-ADL; 42 days)

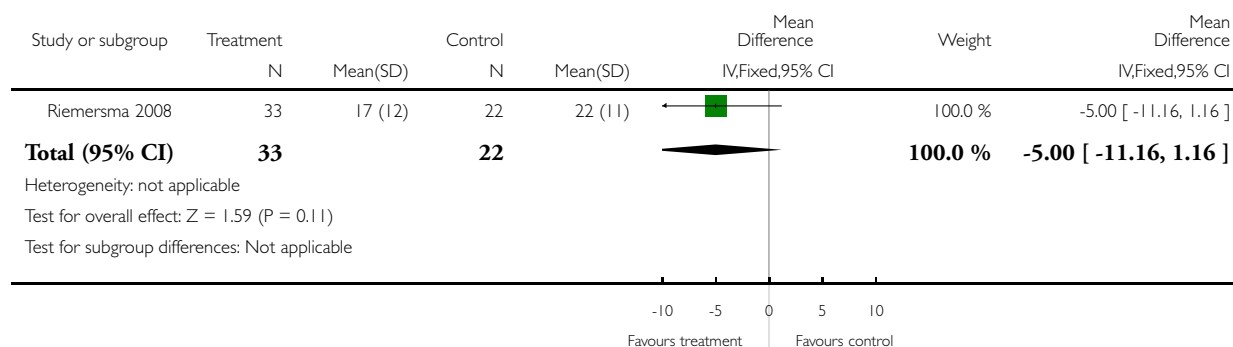


Analysis 1.5. Comparison 1 Morning/daytime bright light vs control, Outcome 5 Functional limitations at endpoint (NI-ADL; 1 year).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 5 Functional limitations at endpoint (NI-ADL; 1 year)

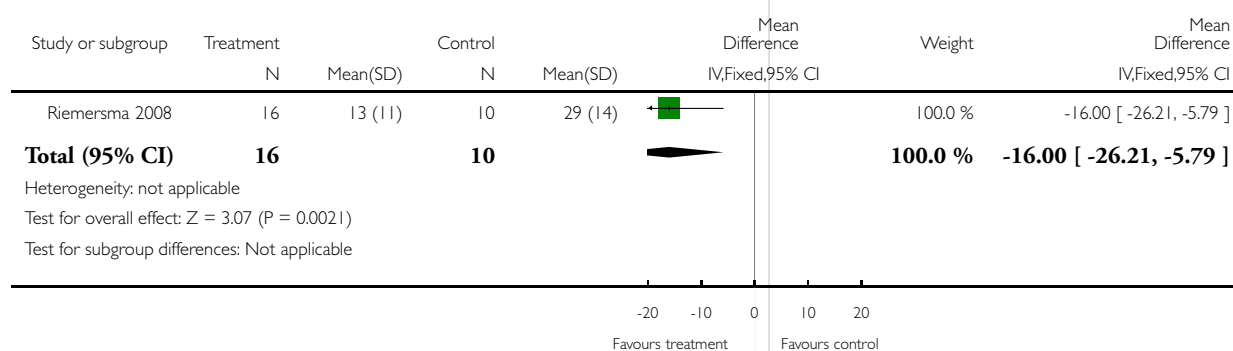


Analysis 1.6. Comparison 1 Morning/daytime bright light vs control, Outcome 6 Functional limitations at endpoint (NI-ADL; 2 years).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 6 Functional limitations at endpoint (NI-ADL; 2 years)

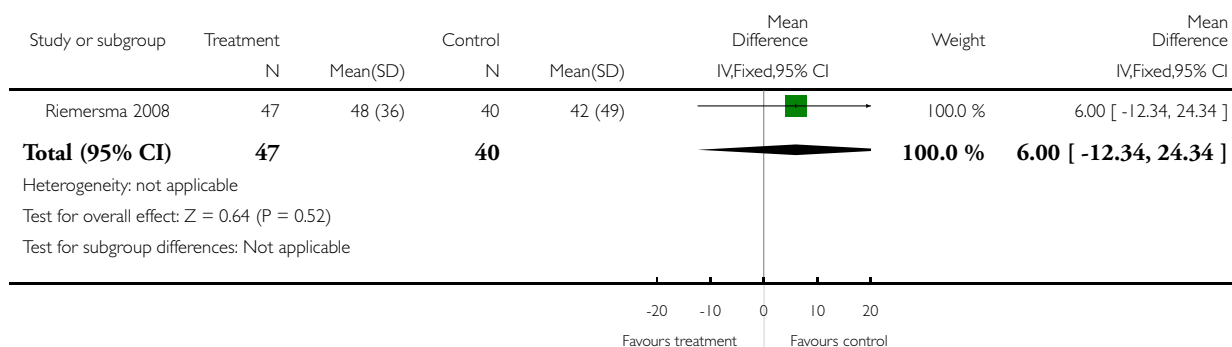


Analysis 1.7. Comparison 1 Morning/daytime bright light vs control, Outcome 7 Sleep onset latency (mins; 42 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 7 Sleep onset latency (mins; 42 days)

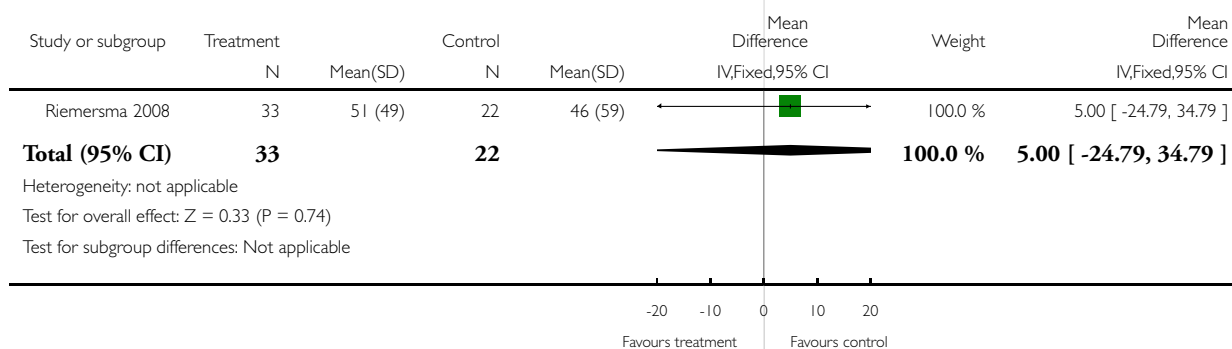


Analysis 1.8. Comparison 1 Morning/daytime bright light vs control, Outcome 8 Sleep onset latency (mins; 1 year).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 8 Sleep onset latency (mins; 1 year)

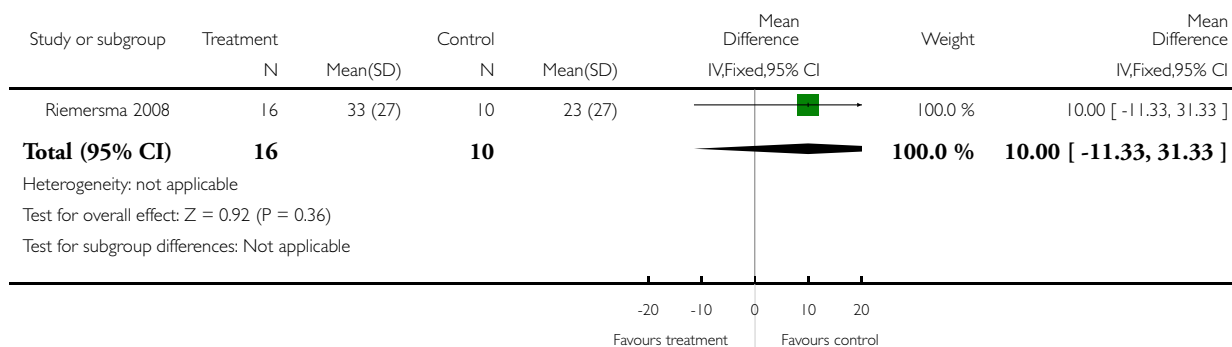


Analysis 1.9. Comparison 1 Morning/daytime bright light vs control, Outcome 9 Sleep onset latency (mins; 2 years).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 9 Sleep onset latency (mins; 2 years)

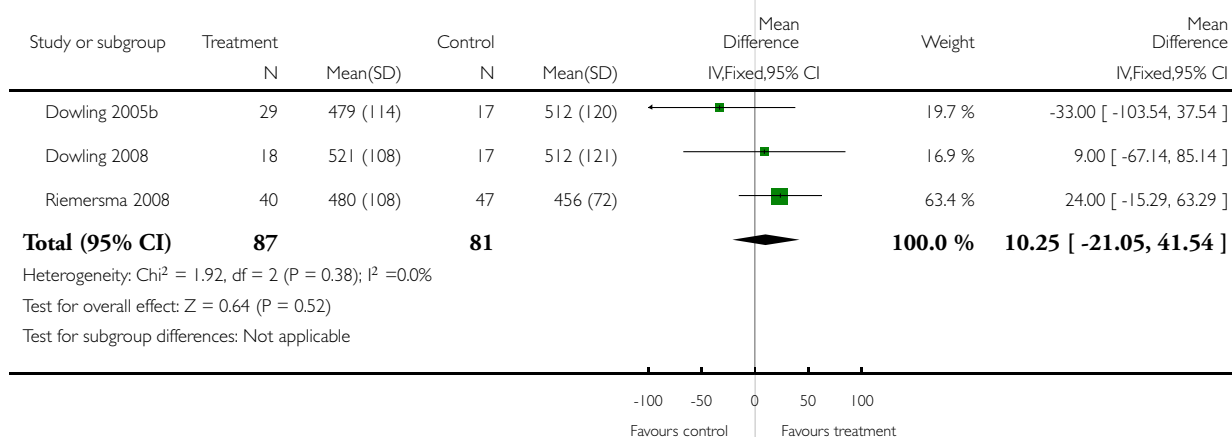


Analysis 1.10. Comparison 1 Morning/daytime bright light vs control, Outcome 10 Total sleep duration (mins; 6-42 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 10 Total sleep duration (mins; 6-42 days)

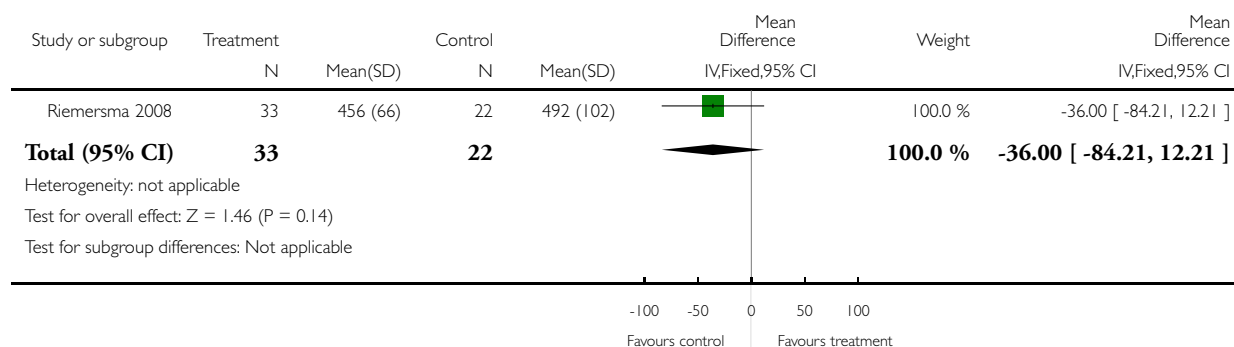


Analysis 1.11. Comparison 1 Morning/daytime bright light vs control, Outcome 11 Total sleep duration (mins; 1 year).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 11 Total sleep duration (mins; 1 year)

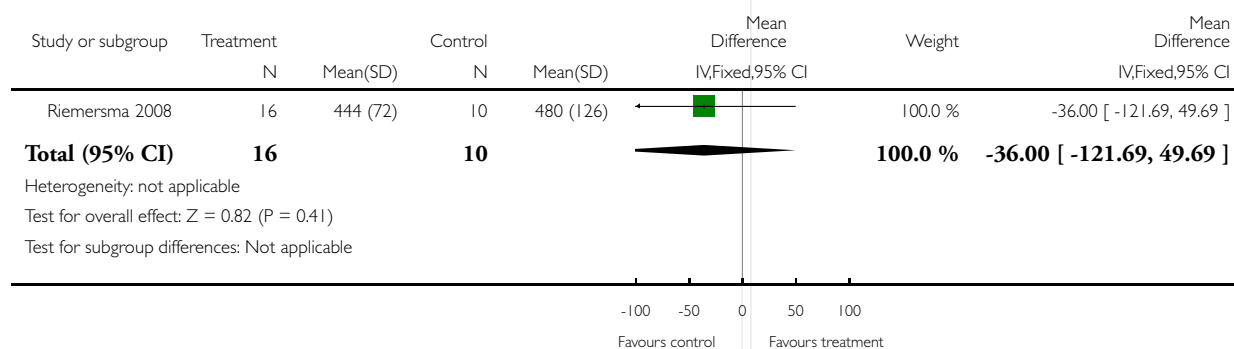


Analysis 1.12. Comparison 1 Morning/daytime bright light vs control, Outcome 12 Total sleep duration (mins; 2 years).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 12 Total sleep duration (mins; 2 years)

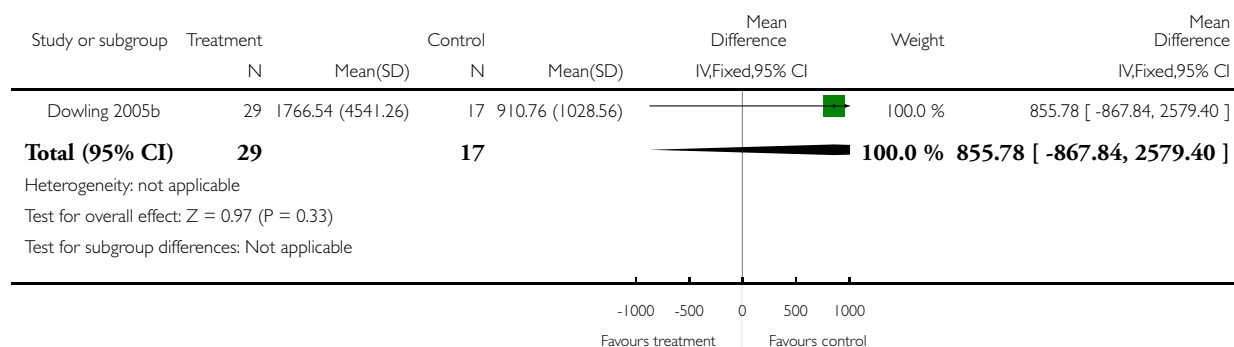


Analysis 1.13. Comparison 1 Morning/daytime bright light vs control, Outcome 13 Activity score (per night) at endpoint.

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 13 Activity score (per night) at endpoint

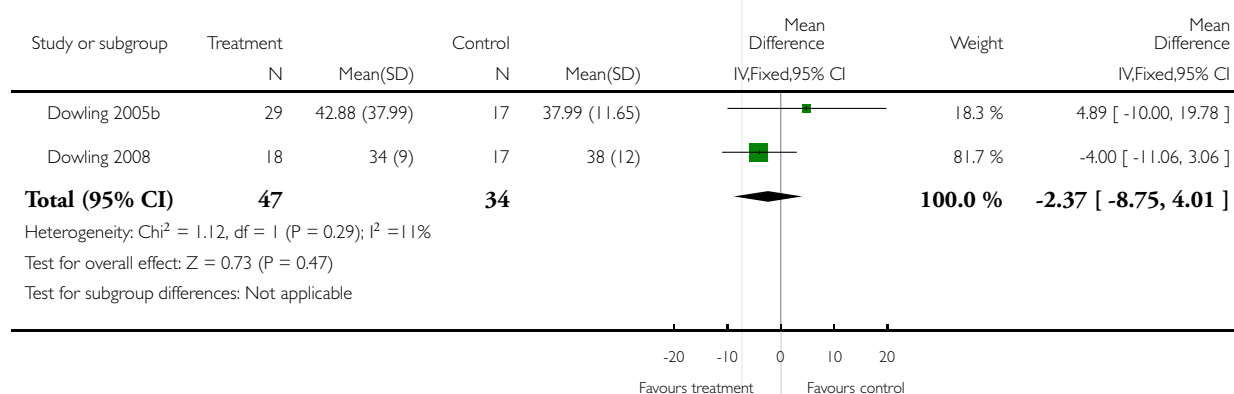


Analysis 1.14. Comparison 1 Morning/daytime bright light vs control, Outcome 14 Number of night-time awakenings at endpoint.

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 14 Number of night-time awakenings at endpoint

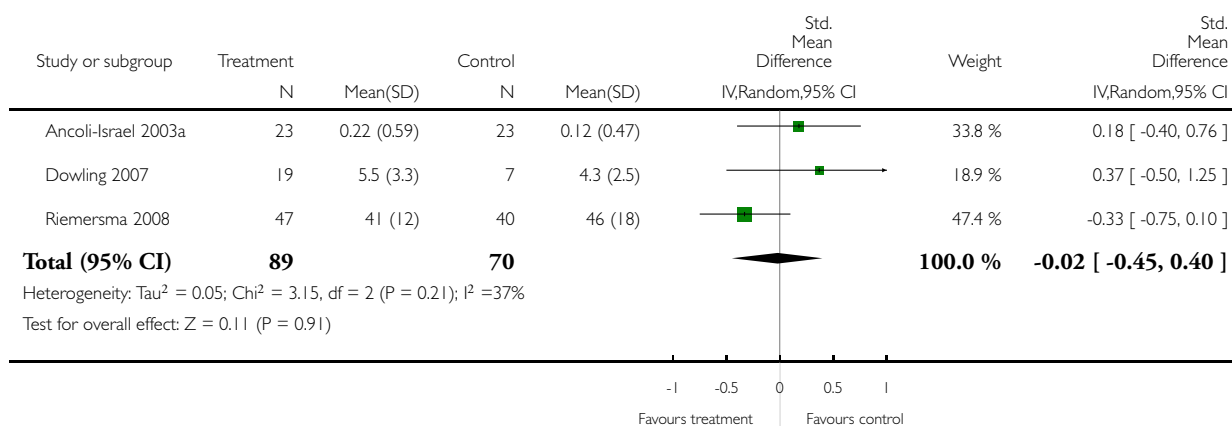


Analysis I.15. Comparison I Morning/daytime bright light vs control, Outcome I5 Behavioural disturbances at endpoint (NPI, ABRS, CMAI; morning assessment; 10-50 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: I Morning/daytime bright light vs control

Outcome: I5 Behavioural disturbances at endpoint (NPI, ABRS, CMAI; morning assessment; 10-50 days)

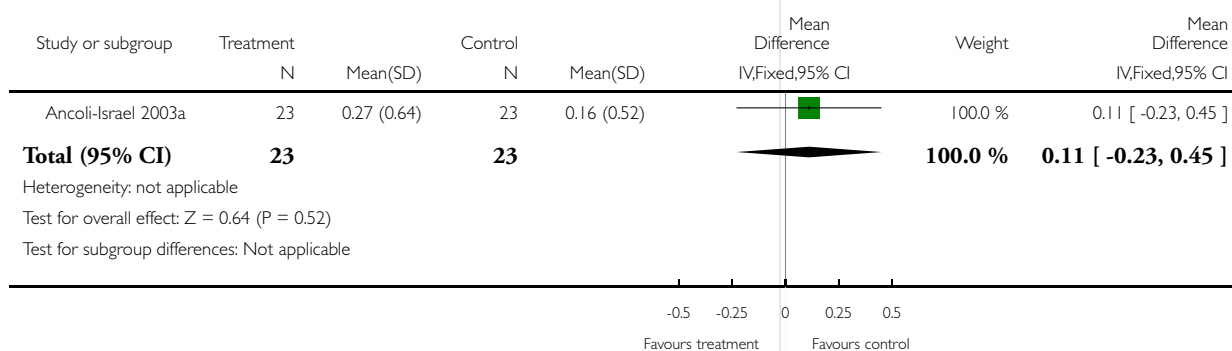


Analysis I.16. Comparison I Morning/daytime bright light vs control, Outcome I6 Behavioural disturbances at endpoint (ABRS; evening assessment; 10 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: I Morning/daytime bright light vs control

Outcome: I6 Behavioural disturbances at endpoint (ABRS; evening assessment; 10 days)

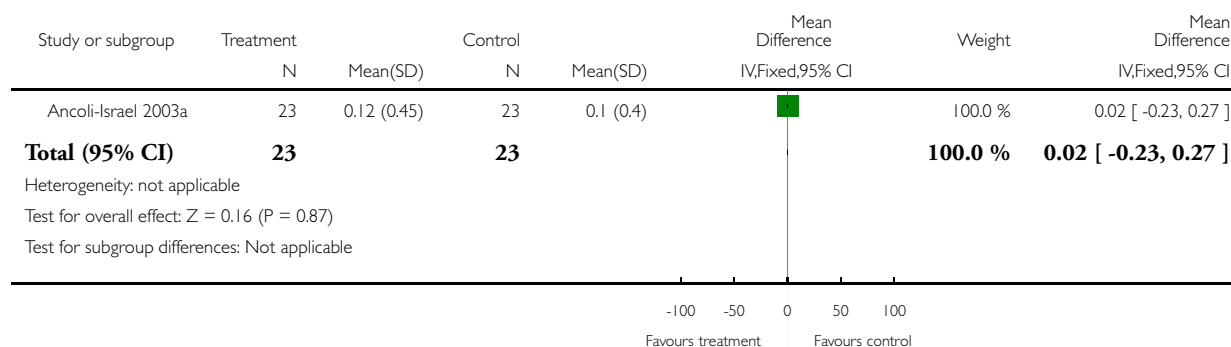


Analysis I.17. Comparison I Morning/daytime bright light vs control, Outcome 17 Behavioural disturbances at follow-up (ABRS; morning assessment; after 5 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: I Morning/daytime bright light vs control

Outcome: 17 Behavioural disturbances at follow-up (ABRS; morning assessment; after 5 days)

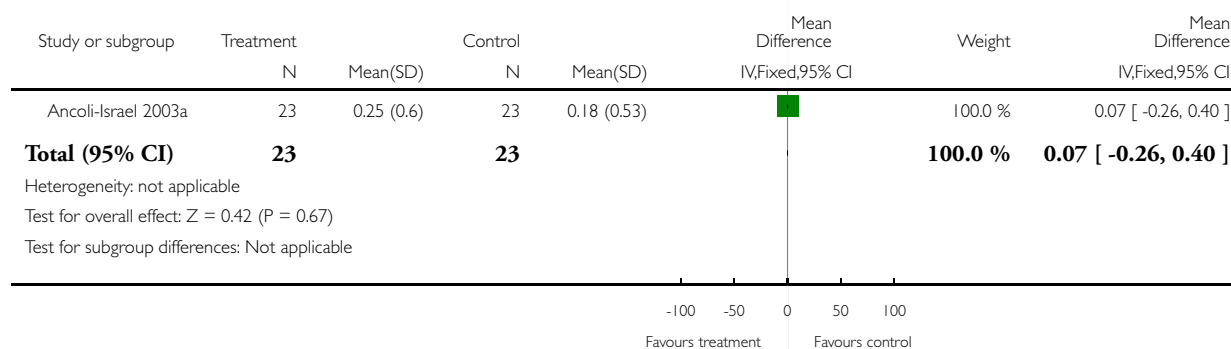


Analysis I.18. Comparison I Morning/daytime bright light vs control, Outcome 18 Behavioural disturbances at follow-up (ABRS; evening assessment; after 5 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: I Morning/daytime bright light vs control

Outcome: 18 Behavioural disturbances at follow-up (ABRS; evening assessment; after 5 days)

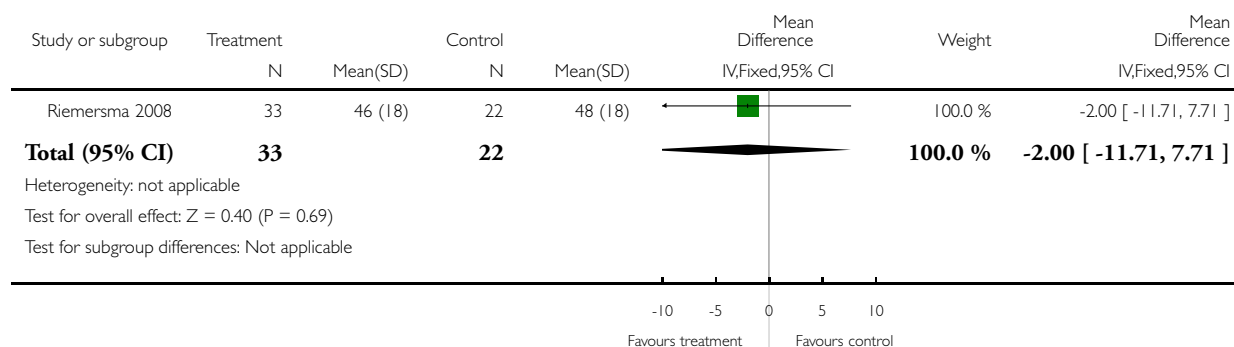


Analysis 1.19. Comparison 1 Morning/daytime bright light vs control, Outcome 19 Behavioural disturbances at endpoint (CMAI; 1 year).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 19 Behavioural disturbances at endpoint (CMAI; 1 year)

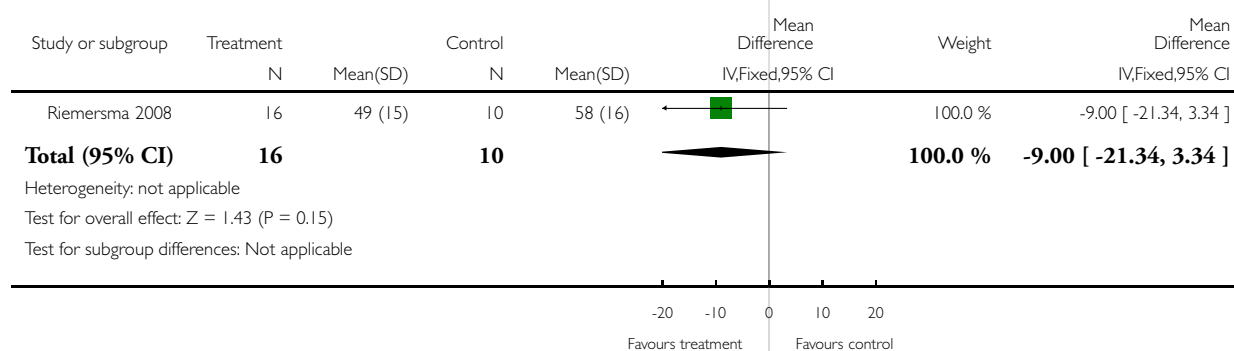


Analysis 1.20. Comparison 1 Morning/daytime bright light vs control, Outcome 20 Behavioural disturbances at follow-up (CMAI; after 2 years).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 20 Behavioural disturbances at follow-up (CMAI; after 2 years)

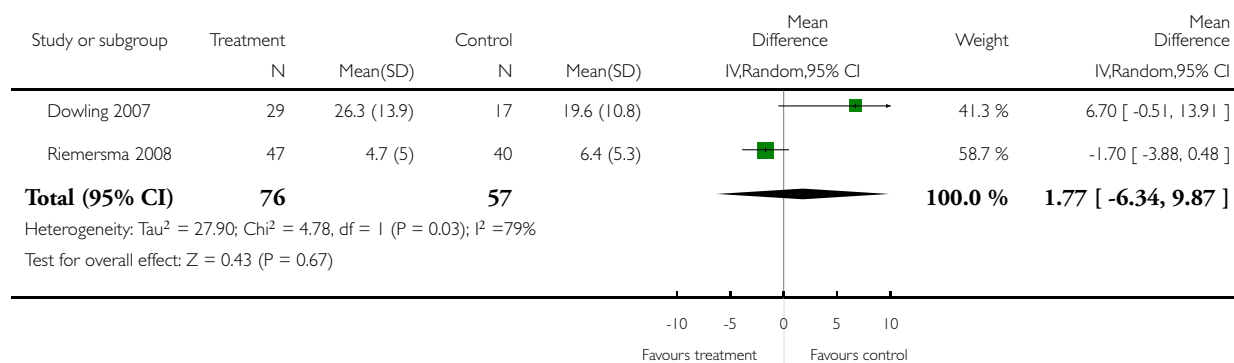


Analysis 1.21. Comparison 1 Morning/daytime bright light vs control, Outcome 21 Psychiatric symptoms at endpoint (NPI total scores; 42-50 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 21 Psychiatric symptoms at endpoint (NPI total scores; 42-50 days)

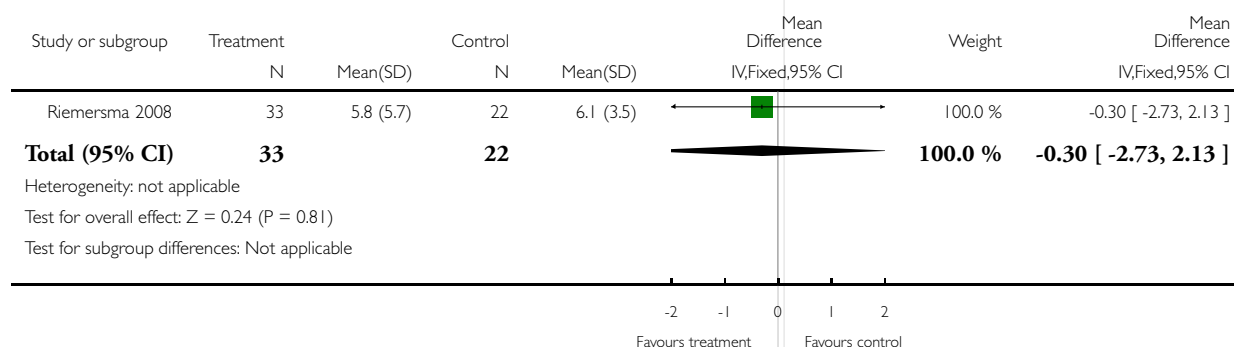


Analysis 1.22. Comparison 1 Morning/daytime bright light vs control, Outcome 22 Psychiatric symptoms at endpoint (NPI total scores; 1 year).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 22 Psychiatric symptoms at endpoint (NPI total scores; 1 year)

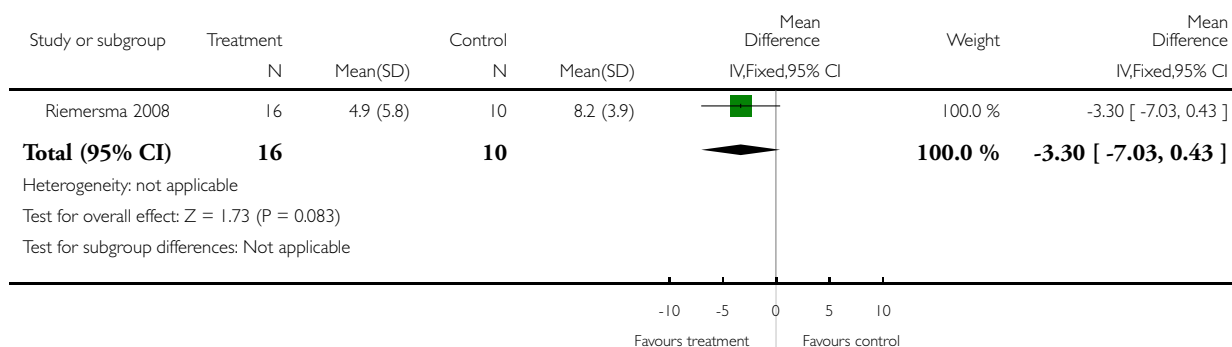


Analysis 1.23. Comparison 1 Morning/daytime bright light vs control, Outcome 23 Psychiatric symptoms at endpoint (NPI total scores; 2 years).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 23 Psychiatric symptoms at endpoint (NPI total scores; 2 years)

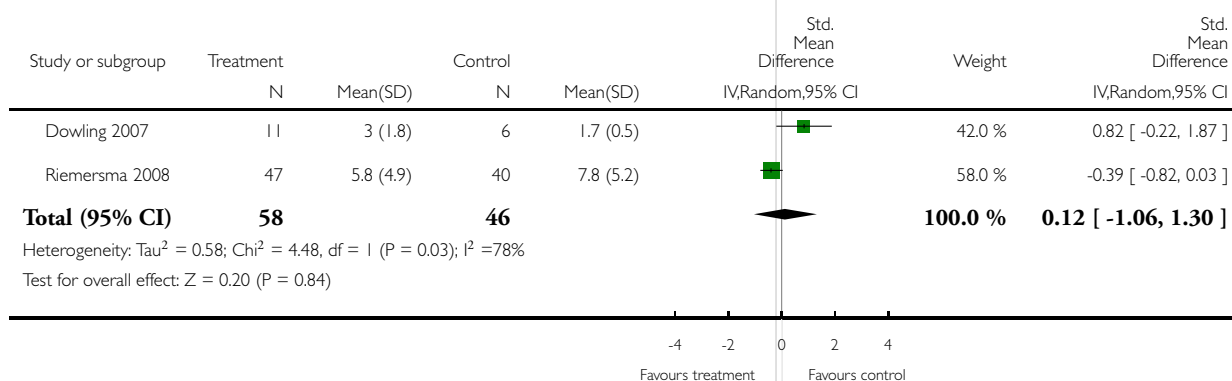


Analysis 1.24. Comparison 1 Morning/daytime bright light vs control, Outcome 24 Depression/dysphoria (CSDD, NPI subscale; 42-50 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 24 Depression/dysphoria (CSDD, NPI subscale; 42-50 days)

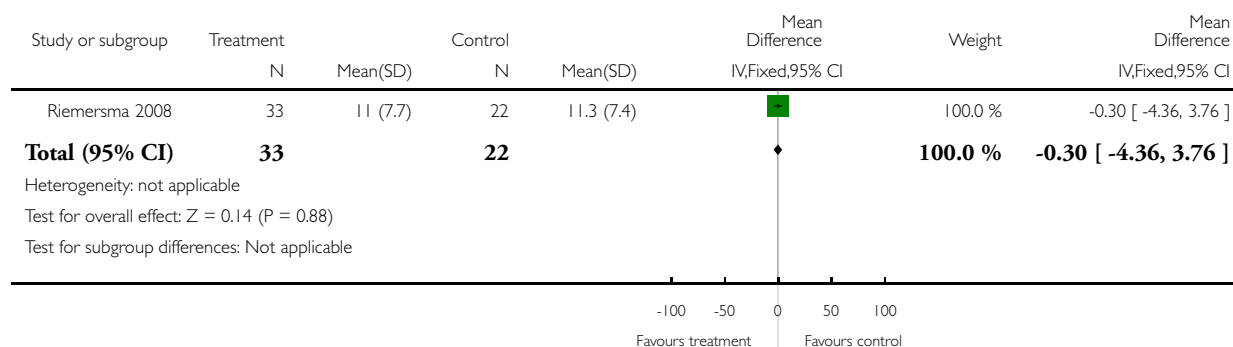


Analysis 1.25. Comparison I Morning/daytime bright light vs control, Outcome 25 Depression (CSDD; 1 year).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: I Morning/daytime bright light vs control

Outcome: 25 Depression (CSDD; 1 year)

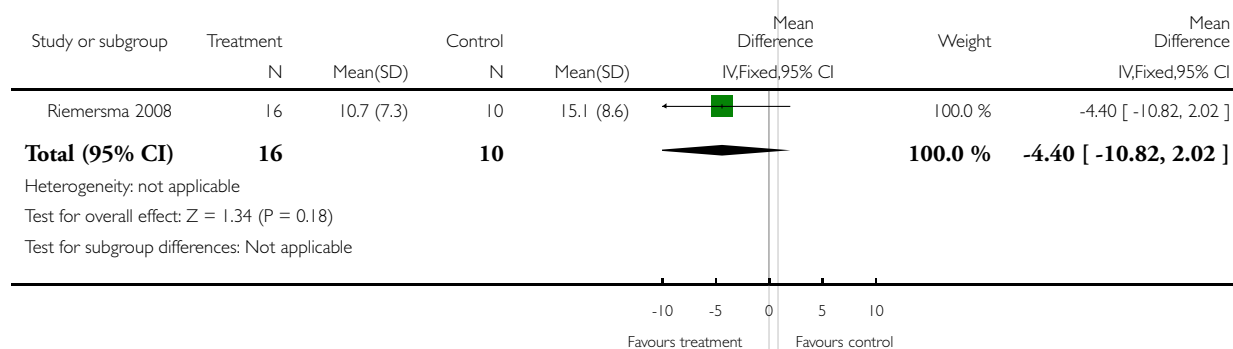


Analysis 1.26. Comparison I Morning/daytime bright light vs control, Outcome 26 Depression (CSDD; 2 years).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: I Morning/daytime bright light vs control

Outcome: 26 Depression (CSDD; 2 years)

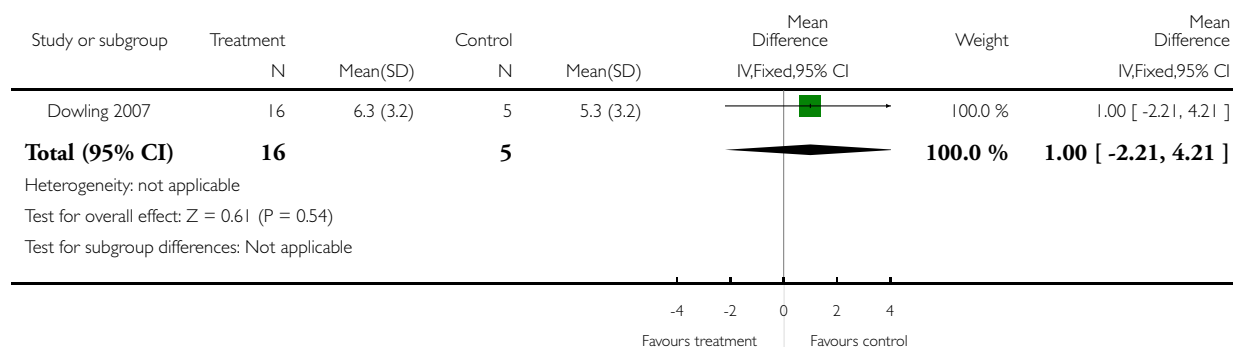


Analysis 1.27. Comparison 1 Morning/daytime bright light vs control, Outcome 27 Apathy/indifference at endpoint (NPI subscale).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 1 Morning/daytime bright light vs control

Outcome: 27 Apathy/indifference at endpoint (NPI subscale)

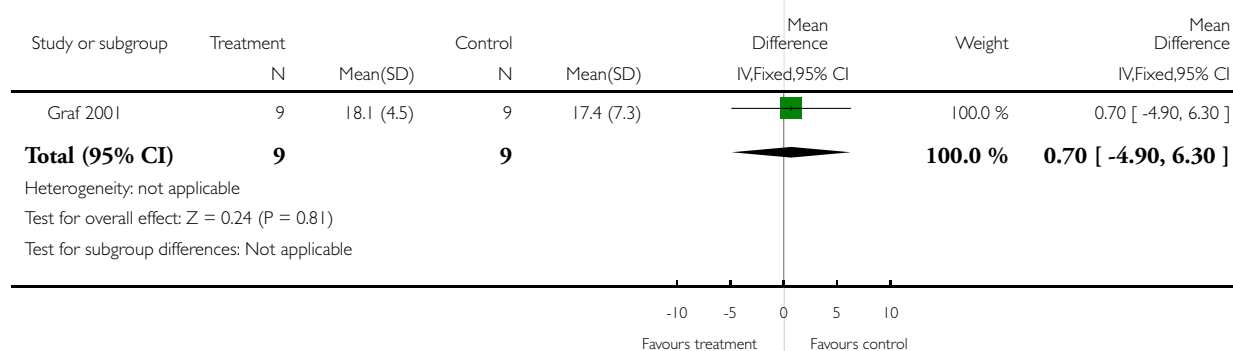


Analysis 2.1. Comparison 2 Evening/afternoon bright light vs control, Outcome 1 Cognition at endpoint (MMSE; 10 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 2 Evening/afternoon bright light vs control

Outcome: 1 Cognition at endpoint (MMSE; 10 days)

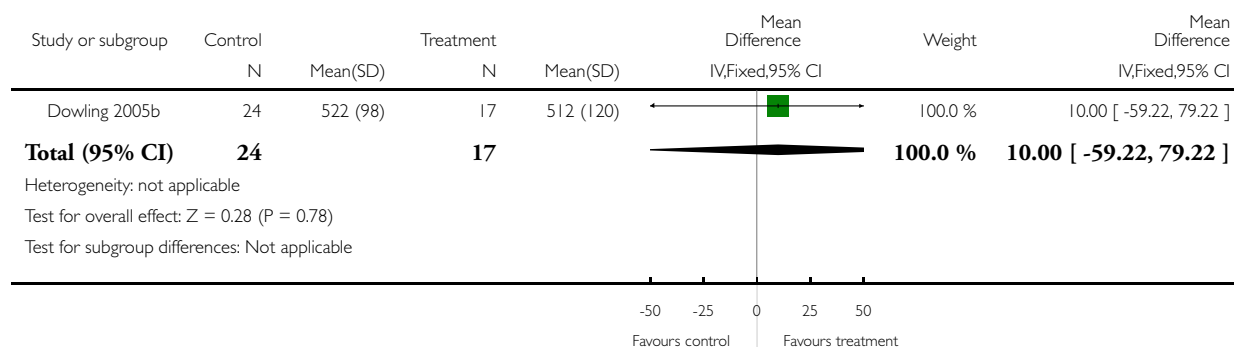


Analysis 2.2. Comparison 2 Evening/afternoon bright light vs control, Outcome 2 Total sleep duration (minutes) at endpoint.

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 2 Evening/afternoon bright light vs control

Outcome: 2 Total sleep duration (minutes) at endpoint

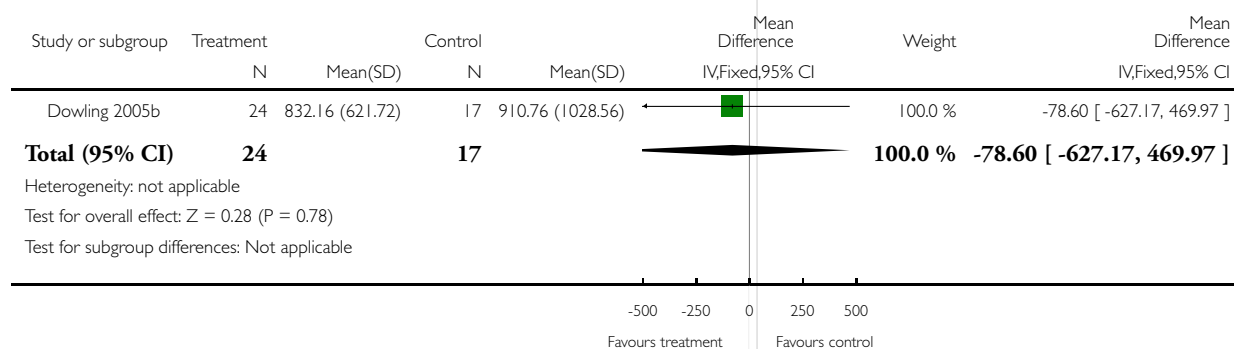


Analysis 2.3. Comparison 2 Evening/afternoon bright light vs control, Outcome 3 Activity score (per night) at endpoint.

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 2 Evening/afternoon bright light vs control

Outcome: 3 Activity score (per night) at endpoint

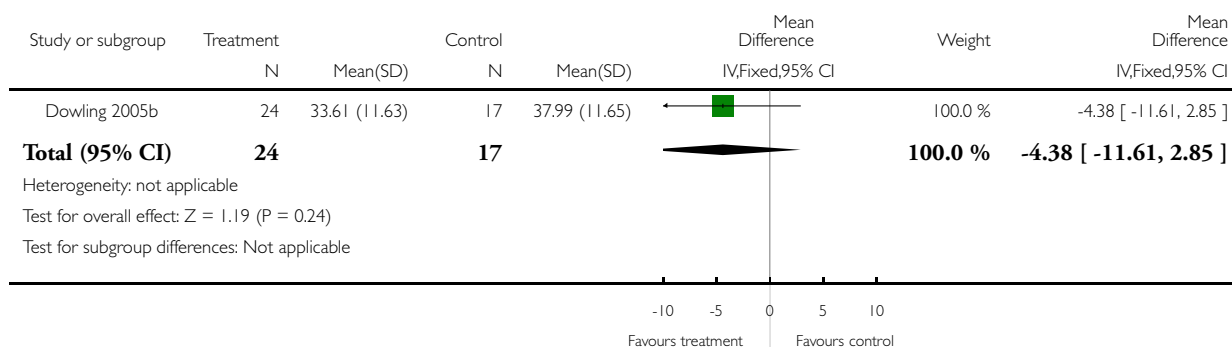


Analysis 2.4. Comparison 2 Evening/afternoon bright light vs control, Outcome 4 Number of nighttime awakenings at endpoint.

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 2 Evening/afternoon bright light vs control

Outcome: 4 Number of nighttime awakenings at endpoint

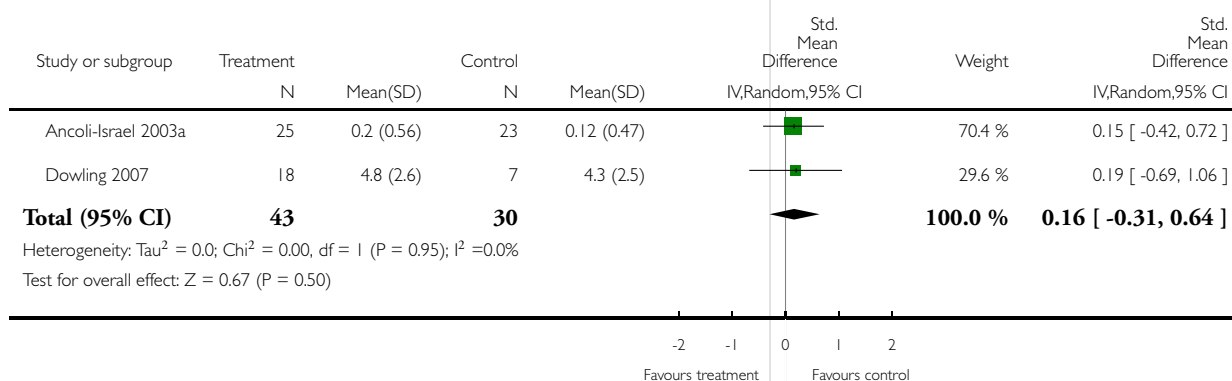


Analysis 2.5. Comparison 2 Evening/afternoon bright light vs control, Outcome 5 Behavioural disturbances at endpoint (NPI, ABRs; morning assessment; 10-50 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 2 Evening/afternoon bright light vs control

Outcome: 5 Behavioural disturbances at endpoint (NPI, ABRs; morning assessment; 10-50 days)

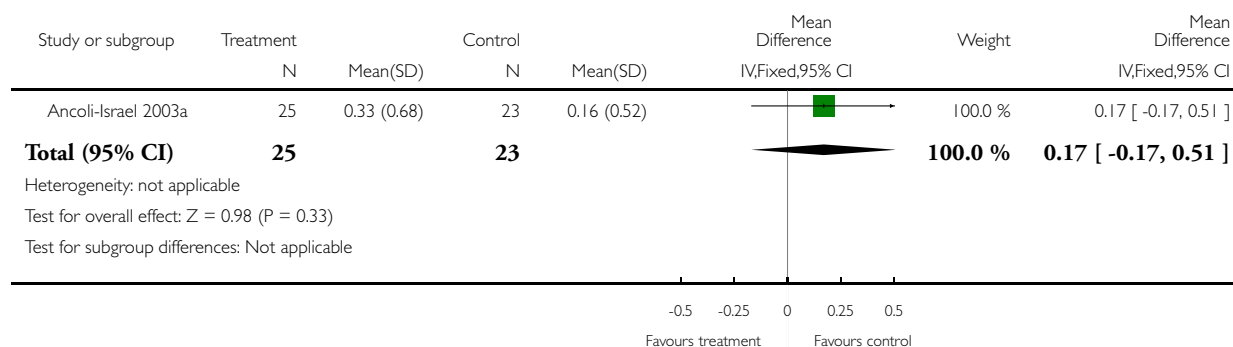


Analysis 2.6. Comparison 2 Evening/afternoon bright light vs control, Outcome 6 Behavioural disturbances at endpoint (ABRS; evening assessment; 10 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 2 Evening/afternoon bright light vs control

Outcome: 6 Behavioural disturbances at endpoint (ABRS; evening assessment; 10 days)

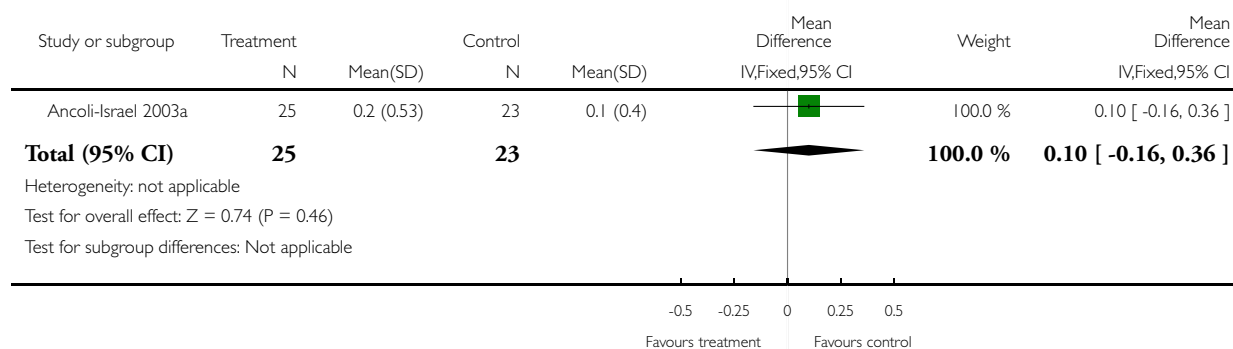


Analysis 2.7. Comparison 2 Evening/afternoon bright light vs control, Outcome 7 Behavioural disturbances at follow-up (ABRS; morning assessment; 5 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 2 Evening/afternoon bright light vs control

Outcome: 7 Behavioural disturbances at follow-up (ABRS; morning assessment; 5 days)

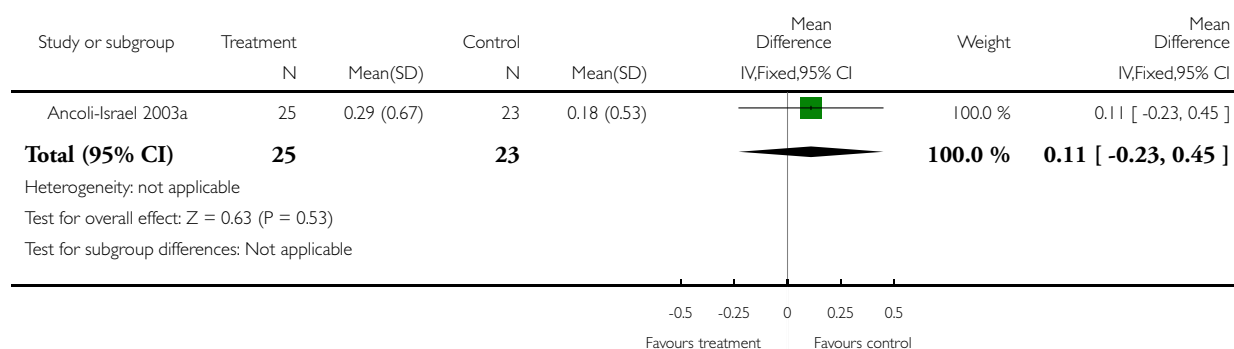


Analysis 2.8. Comparison 2 Evening/afternoon bright light vs control, Outcome 8 Behavioural disturbances at follow-up (ABRS; evening assessment; 5 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 2 Evening/afternoon bright light vs control

Outcome: 8 Behavioural disturbances at follow-up (ABRS; evening assessment; 5 days)

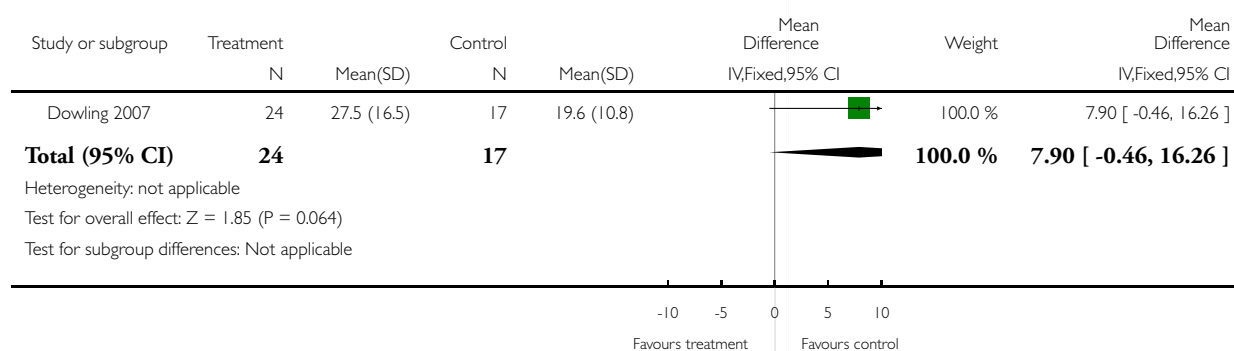


Analysis 2.9. Comparison 2 Evening/afternoon bright light vs control, Outcome 9 Psychiatric symptoms at endpoint (NPI total scores; 50 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 2 Evening/afternoon bright light vs control

Outcome: 9 Psychiatric symptoms at endpoint (NPI total scores; 50 days)

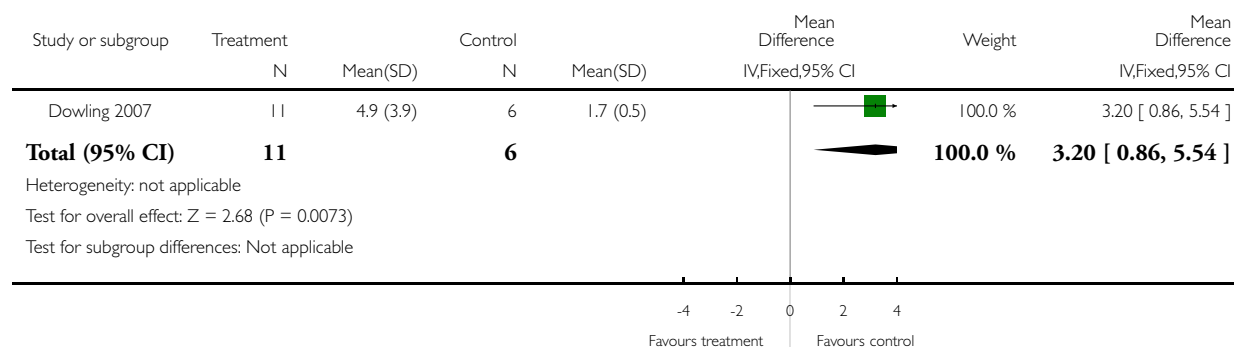


Analysis 2.10. Comparison 2 Evening/afternoon bright light vs control, Outcome 10 Depression/Dysphoria at endpoint (NPI domain subscale; 50 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 2 Evening/afternoon bright light vs control

Outcome: 10 Depression/Dysphoria at endpoint (NPI domain subscale; 50 days)

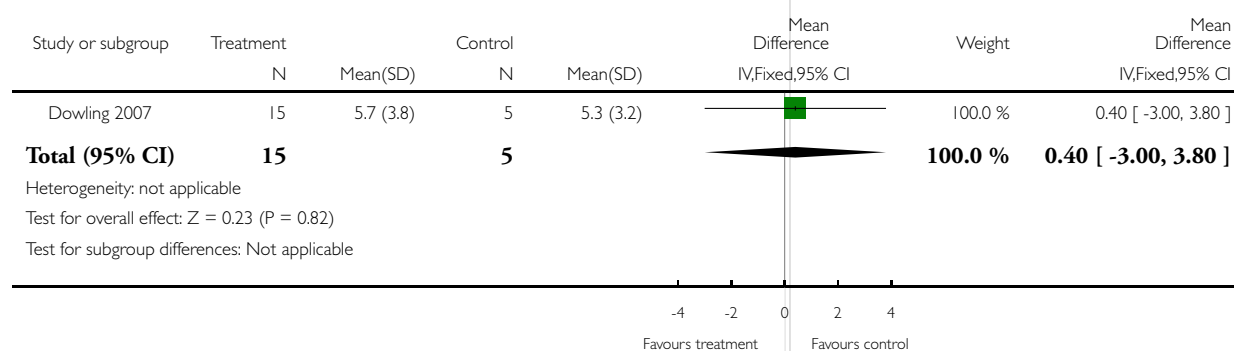


Analysis 2.11. Comparison 2 Evening/afternoon bright light vs control, Outcome 11 Apathy/Indifference at endpoint (NPI domain subscale; 50 days).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 2 Evening/afternoon bright light vs control

Outcome: 11 Apathy/Indifference at endpoint (NPI domain subscale; 50 days)

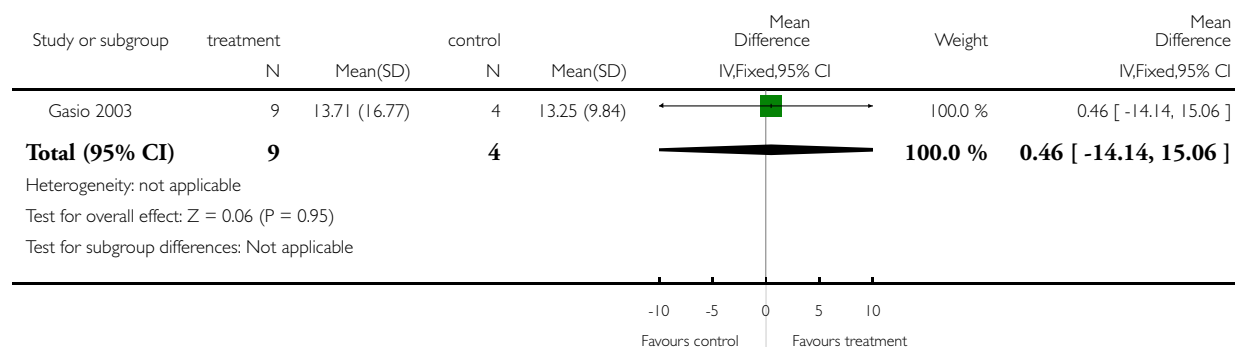


Analysis 3.1. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 1 Cognition at endpoint (MMSE; 3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 1 Cognition at endpoint (MMSE; 3 weeks)

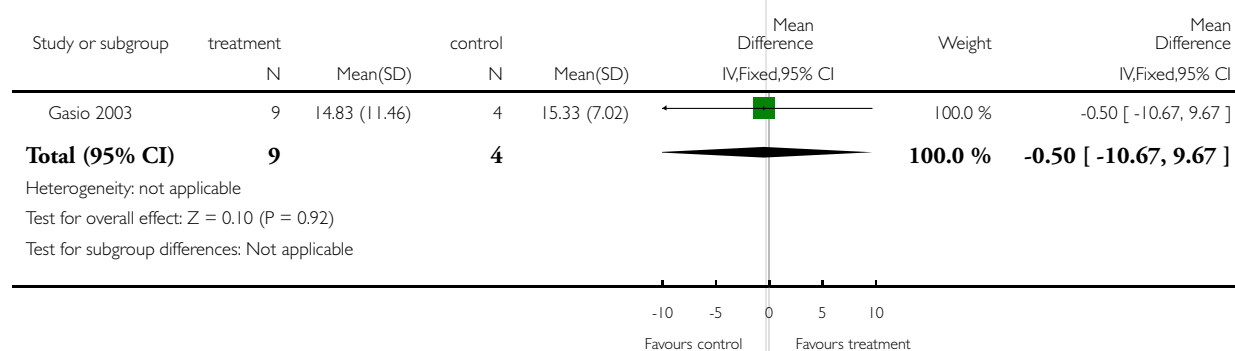


Analysis 3.2. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 2 Cognition at follow-up (MMSE; 3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 2 Cognition at follow-up (MMSE; 3 weeks)

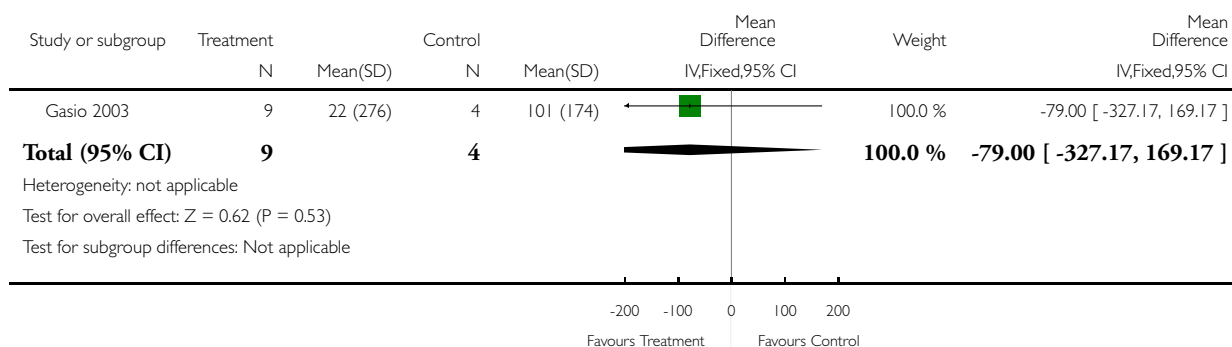


Analysis 3.3. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 3 Sleep onset latency (minutes) at endpoint (3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 3 Sleep onset latency (minutes) at endpoint (3 weeks)

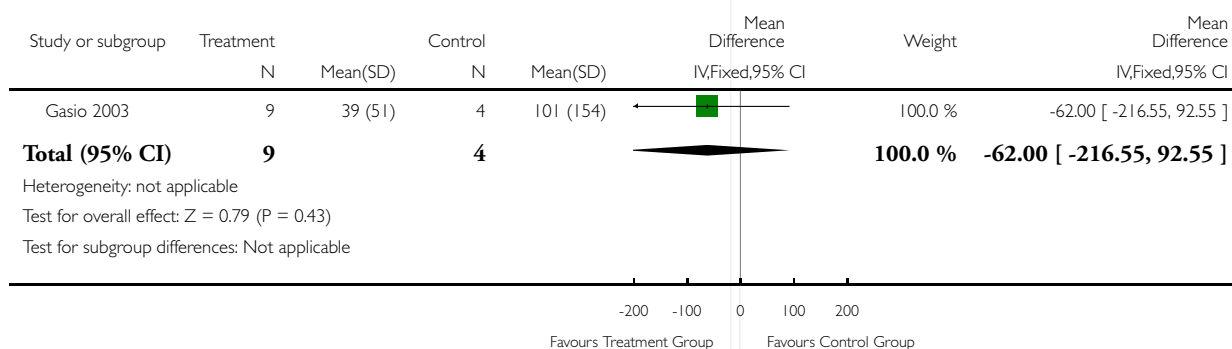


Analysis 3.4. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 4 Sleep onset latency (minutes) at follow-up (3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 4 Sleep onset latency (minutes) at follow-up (3 weeks)

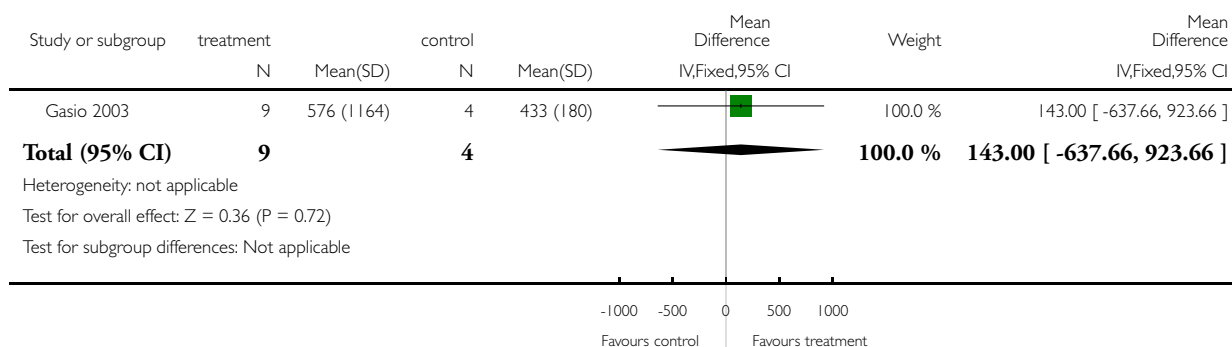


Analysis 3.5. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 5 Total sleep duration (minutes) at endpoint (3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 5 Total sleep duration (minutes) at endpoint (3 weeks)

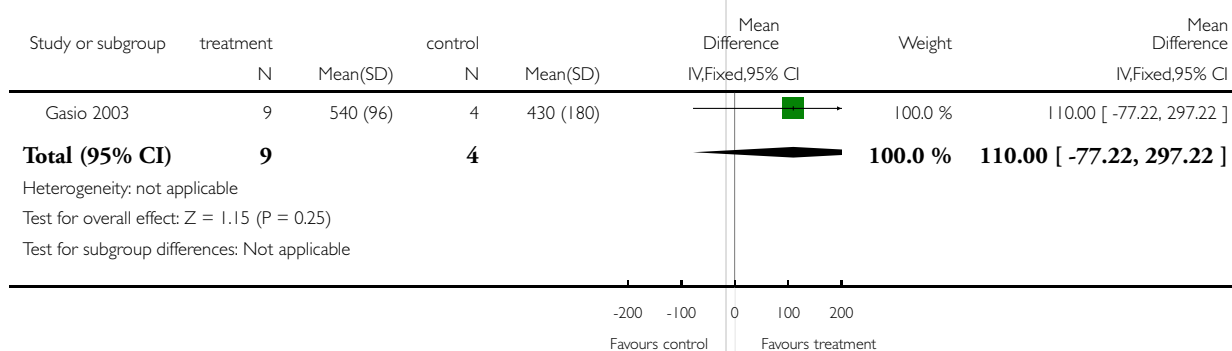


Analysis 3.6. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 6 Total sleep duration (minutes) at follow-up (3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 6 Total sleep duration (minutes) at follow-up (3 weeks)

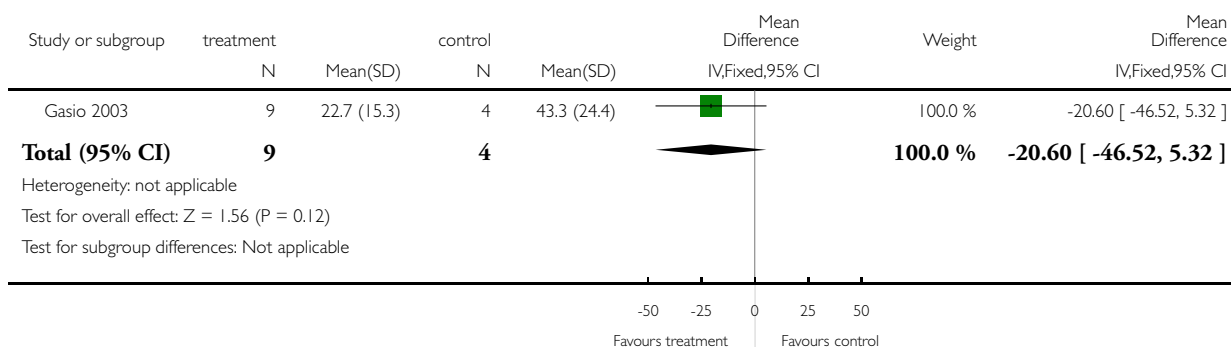


Analysis 3.7. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 7 Nighttime activity counts (per night) at endpoint (3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 7 Nighttime activity counts (per night) at endpoint (3 weeks)

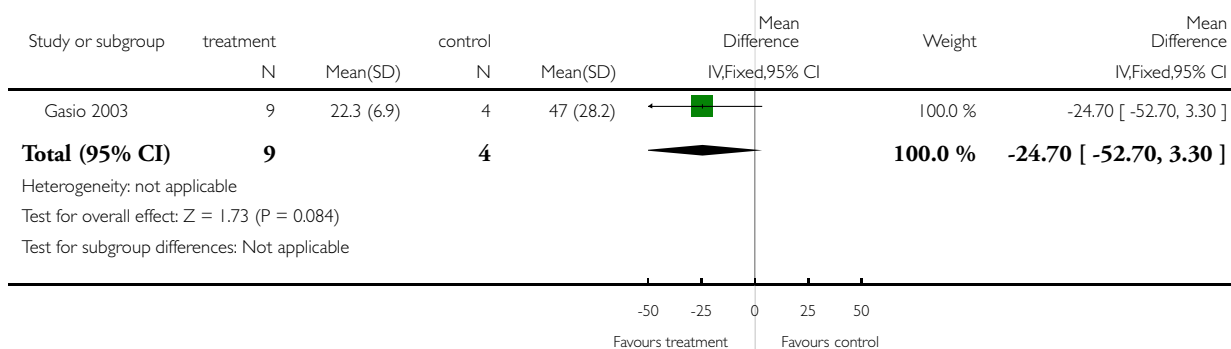


Analysis 3.8. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 8 Nighttime activity counts (per night) at follow-up (3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 8 Nighttime activity counts (per night) at follow-up (3 weeks)

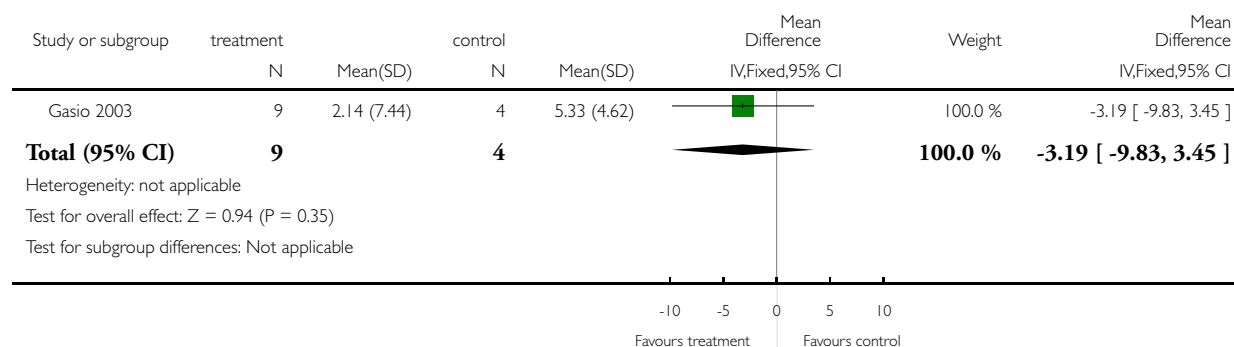


Analysis 3.9. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 9 Psychiatric symptoms at endpoint (NPI total scores; 3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 9 Psychiatric symptoms at endpoint (NPI total scores; 3 weeks)

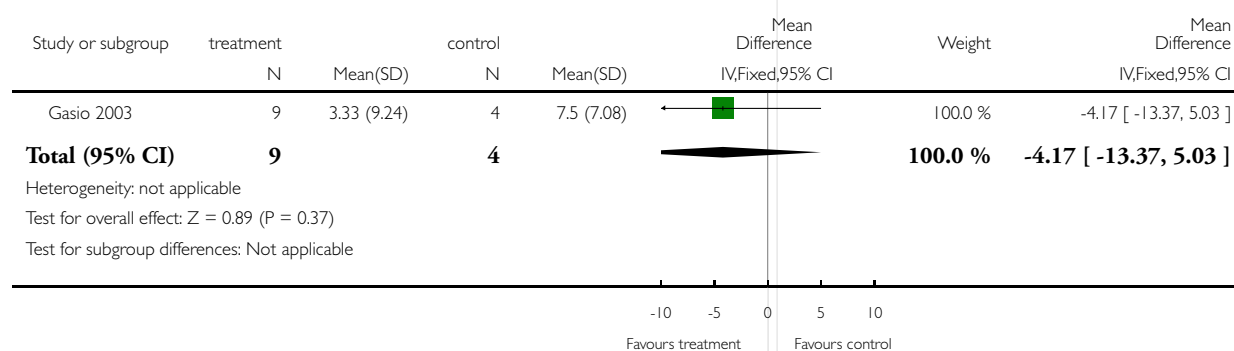


Analysis 3.10. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 10 Psychiatric symptoms at follow-up (NPI total scores; 3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 10 Psychiatric symptoms at follow-up (NPI total scores; 3 weeks)

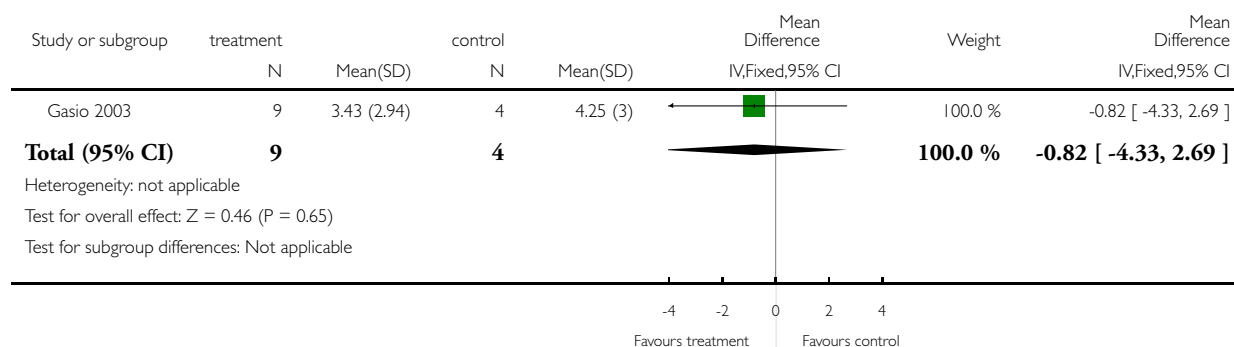


Analysis 3.11. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 11 Depression at endpoint (GDS; 3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 11 Depression at endpoint (GDS; 3 weeks)

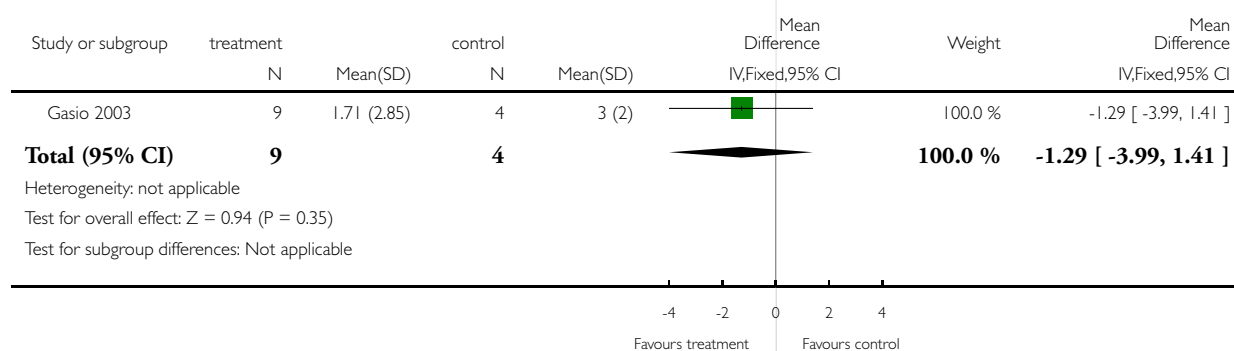


Analysis 3.12. Comparison 3 Dawn-dusk simulation with bright white light vs dim red light, Outcome 12 Depression at follow-up (GDS; 3 weeks).

Review: Light therapy for managing cognitive, sleep, functional, behavioural, or psychiatric disturbances in dementia

Comparison: 3 Dawn-dusk simulation with bright white light vs dim red light

Outcome: 12 Depression at follow-up (GDS; 3 weeks)



ADDITIONAL TABLES

Table 1. Description of Rating Scales Used in the Included Studies

Scale	Abbreviation	Description	Reference
Behavioral Pathology in AD Scale, used in Lyketsos 1999 study	Behave-AD	25 item + global rating item, 4 point scale. Categories include paranoid and delusion ideation, hallucinations, activity disturbances, aggressiveness, & anxiety and phobias. Issues of validity or reliability were not addressed in the published article	Reisberg 1987
Cohen-Mansfield Agitation Inventory, used in Ancoli-Israel 2003 and Riemersma 2008 studies	CMAI	29 item, 7 point scale. A maximum score of 203 indicates that the participant manifests agitated behavior on the average of 7 times per hour. Categories include aggressive behavior, physically non-aggressive behavior, verbal agitation, & a global rating of agitation. A caregiver's rating questionnaire that assesses the frequency of behaviour over the previous two weeks. Inter-rater agreement rates were calculated for each behavior on the CMAI (range r=.88-.92) . No mention was made of the validity of the instrument	Cohen-Mansfield 1989
Agitated Behavior Rating Scale, used in Ancoli-Israel 2003 study	ABRS	15 item, 4 point scale. Categories include agitation, manual manipulation, searching and wandering, escape behaviours, tapping and banging, and verbal agitation. The first four categories can be summarized into one physical agitation score. Higher scores indicate more frequent behaviour at high intensity. Content validity is established through the work of Cohen-Mansfield (1986), Cohen-Mansfield & Billig (1986), and Cohen-Mansfield, Marx & Rosenthal (1989). Data show high inter-rater reliability (coefficients not reported) for all components of rating scale	Bliwise 1993
Neuropsychiatric Inventory, used in Gasio 2003 study	NPI	10 behavioral domains with 7-8 sub-questions. Measures severity (0 to 3) and frequency (0 to 4). Categories include delusions, hallucinations, dysphoria, anxiety, agitation/aggression, euphoria, disinhibition, irritability/lability, ap-	Cummings 1994

Table 1. Description of Rating Scales Used in the Included Studies (Continued)

		<p>athy, & aberrant motor activity. A global score can be generated by summing the total scores (frequency multiplied by severity) of the individual sub-scales. Concurrent validity was determined by comparing the scores on the relevant sub-scales of the NPI with the appropriate scales of 2 instruments, BEHAVE-AD and the Hamilton Depression Rating Scale (coefficients not reported). To establish content validity a delphi panel was developed and asked to rate the scale items. Inter-rater reliability was found to be very high (correlation not reported) and test-retest reliability was found to be 0.79 for frequency (p=.0001) and 0.86 for sensitivity (p=.0001)</p>	
<p>Neuropsychiatric Inventory - Nursing Home version, used in Dowling 2007 study</p>	NPI-NH	<p>Modified version of the NPI, measuring 12 areas of psychiatric symptomatology: delusions, hallucinations, agitation/aggression, depression, anxiety, euphoria/elation, apathy/indifference, disinhibition, irritability/lability, aberrant motor behavior, nighttime behavior, and appetite/eating changes. Frequency (0 to 4) and severity (0 to 3) are measured. Symptom sub-scale scores are calculated as a product of frequency and severity (0 to 12). Symptom sub-scale scores are added to form the total score (0 to 144) . Higher score indicate higher levels of behavioural symptoms. Reliable change in sub-scales range from 1.29 to 5.13, depending on scale. A difference of 22 points or less may be due to measurement error. Please consult Iverson 2002 for details</p>	<p>Iverson 2002</p>
<p>Cornell Scale for Depression in Dementia, used in Lyketsos 1999 and Riemersma 2008 studies</p>	CSDD	<p>19 item, 3 point scale. Categories include mood related signs, behavioural disturbance, physical signs, cyclic functions, and ideational disturbance. The scale has adequate inter-rater reliability (kappa weighted=.67), internal consistency (coefficient alpha=.84) and sensitivity. Total scale scores were correlated with depressive subtypes of various intensity classified according to Research</p>	<p>Alexopoulos 1988</p>

Table 1. Description of Rating Scales Used in the Included Studies (Continued)

		Diagnostic Criteria (r=.83)	
Geriatric Depression Scale, used in Gasio 2003 study	GDS	30 item, or short form 15 item, with 0 indicating no sign of depression and high score suggesting severe depression. Validation study compared the two forms and found both were successful in differentiating depressed from non-depressed subjects with high correlation (r=.84, p<.001). No specific mention of the reliability of the study	Sheikh 1986
Consortium to Establish a Registry for Alzheimer's Disease, used in Gasio 2003 study	CERAD	Batteries of clinical and neuropsychological tests that measure the primary cognitive manifestations of AD and detect deterioration of language, memory, praxis, & general intellectual status. The Clinical Assessment Battery includes semi-structured interviews with both the subject and informant, general physical, neurologic, and laboratory examinations, drug inventory, depression scale, and a general medical history. The Neuropsychological Assessment Battery includes Verbal Fluency: Animal Category, Modified Boston Naming Test, Mini-Mental State Examination, Word List Memory, Constructional Praxis, Word List Recall, and Word List Recognition. Inter-rater and test-retest reliabilities were substantial. Intraclass correlation coefficients for the tests ranged from 0.92 (Constructional Praxis) to 1.0 (Word List Recall). Test-retest correlations were comparable for both mild AD and moderate AD cases. Correlation for CDR1 ranged from r=0.52-0.89 and correlation for CDR2 ranged from r=0.43-0.90. Long-term observations are in progress to determine validity	Morris 1989
Mini Mental State Examination, used in Gasio 2003, Graf 2001 and Riemersma 2008 studies	MMSE	The MMSE was developed as a short test suitable for the elderly with dementia. It concentrates on the cognitive aspects of mental function: the five sections cover orientation, immediate recall, attention and calculation, delayed recall and language. A maximum score of 30 suggests normal function. Con-	Folstein 1975

Table 1. Description of Rating Scales Used in the Included Studies (Continued)

		current validity was determined by correlating MMS scores with the Wechsler Adult Intelligence Scale, Verbal and Performance scores. For Mini-Mental Status vs Verbal IQ, Pearson $r=0.776$ ($p<0.001$) and for Mini-Mental Status vs Performance IQ, Pearson $r=0.660$ ($p<0.001$). Test re-test reliability was determined by a single examiner 24 hrs. apart ($r=0.887$), by two different examiners 24 hrs. apart ($r=0.827$), and at 28 day re-test ($r=0.988$)	
Philadelphia Geriatric Centre Affect Rating Scale, used in Riemersma 2008 study	PGCARS	Six categories (three measuring positive affect :pleasure, interest, contentment; three measuring negative affect: anger, anxiety/fear, sadness). The rater observes signs of each category; no questions are asked. Scoring of affect not described in source article. Concurrent validity suggested by comparison of PGCARS to Mattis GDS core predictive of 'positive predicted relationships' (76%). Inter-rater reliability also suggested (kappa range .76 to .89)	Lawton 1996
Nurse-Informant Activities of Daily Living, used in Riemersma 2008 study	NI-ADL	Adaptation of the Katz ADL scale. The Katz includes six items with a 3-point scale. Items include competence in: feeding, continence, transferring, going to toilet, dressing, and bathing. Higher scores indicate higher dependence. Concurrent validity claimed by comparison to paediatric texts, no statistical results reported. Reliability not discussed	Holmes 1990; Katz 1963

Table 2. Description of Risk of Bias of Included Studies

Study	Control Confounders	Attrition Rate	Compliance	Blinding of Assessor
Ancoli-Israel 2003a	Age, sex, cognitive impairment, education, vision, medication use	8.7%	Treatment: Mean 92.1min. of bright light per 120-min. bright light session. Actillumes worn by 91.3% of participants. Complete analyses were completed	Deception of research & nursing staff

Table 2. Description of Risk of Bias of Included Studies (Continued)

			on 72 of the 92 participants	
Ancoli-Israel 2003b	Age, sex, cognitive impairment, education, vision, medication use	23%	Treatment: Mean 92.1min. of bright light per 120-min. bright light session. Actillumes worn by 91.3% of participants	Deception of research & nursing staff
Dowling 2005b	Age, sex, ethnicity, cognitive impairment, medication use, uneven # of control vs. experimental participants	N/R	84% never removed Actiwatch, mean exposure to light intervention period = 76%	Unknown
Dowling 2007	Age, sex, ethnicity, cognitive impairment, medication use, uneven # of control vs. experimental participants	N/R	Patients received 76% average light exposure, some missed all or part of an intervention session (exact number not known)	Staff potentially aware of group assignment
Dowling 2008	Age, sex, vision, medication use. Subjects in the control group were significantly younger than those in the light therapy group. Analyses were conducted to control for this difference	0%	The dose of bright light received was 82% (SD 17%) . In addition, 41 of the 50 subjects did not remove the Actiwatches, of the total possible 108 hours, on average 105 +8 hours of valid data for baseline and 107 +3 hours of valid data at the end of the intervention were collected	Not reported for the light therapy group.
Gasio 2003	Age, sex, cognitive impairment, mood, vision, medication use	>20%	Dawn-Dusk Simulation or Dim Red Light received by all participants. 5/20 participants withdrew due to non-compliance with actimeter	Deception of residents & staff (unclear if assessor was blind to treatment)
Graf 2001	Age, cognitive impairment, mood, acute medical illness, medication use	>20%	Treatment/Control: 100% participants	Rater blind to treatment
Lyketsos 1999	Age, sex, cognitive impairment, education, vision, race, mood, sleep, behaviour, medication use	>20%	Treatment/Control: 100% participants	Rater blind to treatment

Table 2. Description of Risk of Bias of Included Studies (Continued)

Mishima 1998	Age, sex, cognitive impairment, physical function, schedule, medication use	<10%	Treatment/Control: 100% participants Compliance with Actigraph unknown	Unknown
Riemersma 2008	Age, sex, cognitive impairment, medication use, uneven # of control vs. experimental participants	up to 42% after 1 year	Not reported as treatment was facility light fixtures used.	Double-blind (outcome raters and caregivers were blinded to treatment allocation)

WHAT'S NEW

Last assessed as up-to-date: 2 December 2008.

Date	Event	Description
3 December 2008	New citation required but conclusions have not changed	The title of this updated review has changed
3 December 2008	New search has been performed	A new update search was performed on 4 March 2008. Some new studies were retrieved for inclusion or exclusion. Three new studies have been included in the updated review, and 4 new studies have been excluded

HISTORY

Protocol first published: Issue 4, 2002

Review first published: Issue 2, 2004

Date	Event	Description
15 May 2006	New search has been performed	New searches revealed one incomplete trial and two non-RCTs. However, none met the inclusion criteria for this review. The Results and conclusions of the review remain unchanged
11 February 2004	New citation required and conclusions have changed	Substantive amendment

CONTRIBUTIONS OF AUTHORS

-DF: All correspondence; drafting review versions; obtaining copies of reports; selection of trials; extraction of data; entry of data; interpretation of data analysis; writing review

-IC: drafting review versions; obtaining copies of reports; selection of trials; extraction of data; entry of data; interpretation of data analysis; writing review

-AL: drafting review versions; extraction of data; entry of data; interpretation of data analysis

-DM: drafting review versions; selection of trials; interpretation of data analysis

-SP: drafting review versions; selection of trials; extraction of data; entry of data; interpretation of data analysis

-JF: drafting review versions; obtaining copies of reports; entry of data

-SF: drafting review versions; assistance with data analysis

-CDCIG contact editor: Linda Clare

-Consumer editor: Yvonne Greenough

This review has been peer reviewed anonymously.

DECLARATIONS OF INTEREST

None known

SOURCES OF SUPPORT

Internal sources

- College of Nursing, University of Saskatchewan, Canada.
- Arthur Labatt Family School of Nursing, Faculty of Health Sciences, University of Western Ontario, Canada.

External sources

- No sources of support supplied

INDEX TERMS

Medical Subject Headings (MeSH)

*Phototherapy; Affect; Cognition Disorders [etiology; *therapy]; Dementia [*complications]; Depression [etiology; *therapy]; Psychomotor Agitation [etiology; *therapy]; Randomized Controlled Trials as Topic; Sleep Disorders [etiology; *therapy]

MeSH check words

Aged; Humans