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ORIGINAL ARTICLE

WILEY

The effect of altered sleep timing on glycaemic outcomes: Systematic review of human intervention studies

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Abstract

Aims: Alterations in sleep timing can lead to disturbances in glycaemic control, although the evidence is inconsistent. Therefore, this systematic review summarizes results from human intervention studies of altered sleep timing on glycaemic outcomes.

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Materials and Methods: As part of a broader search on the effect of altering timing of sleep, physical activity and dietary intake, Medline and Embase were searched from inception to February 2023, and subsequent reference searches were done. With the help of a machine learning–aided program 'ASReview', we selected any type of intervention study in the general adult population, which acutely delayed sleep by ≥2 h for at least one night, while the total time in bed was the same between early and late sleep. Quality assessment was done using the quality assessment tool for quantitative studies.

Results: In total, 14 studies (159 adults with normal or increased weight) were identified. Methodological quality was high ($n = 4$), moderate ($n = 7$) or low ($n = 3$). Acute delays of sleep onset showed unfavourable effects in 10 out of 27 measured glycaemic outcomes (one–six studies reported on each outcome) with outcomes mostly measured in the postprandial period, compared to (early) nighttime sleep.

Conclusions: Acutely delaying sleep timing might have unfavourable effects on glycaemic outcomes, compared to (early) nighttime sleep. Future research does however need better controlled trials, also measuring and controlling sleep quantity, sleep quality, physical activity and dietary intake, with longer follow-up periods, consistent outcomes and designs and more diverse populations to provide targeted advice regarding the optimal timing for sleep.

Protocol registration: This review is part of a larger search 'The effect of altering timing of physical activity, sleep and energy intake on glycaemia and Type 2 Diabetes risk in humans', of which the protocol was registered in the PROSPERO database on 27 November 2021 under number: CRD42021287828.

KEYWORDS
circadian clocks, circadian dysregulation, glucose metabolism, glycemic control, sleep

1 | INTRODUCTION

In today's society, the standard circadian sleep–wake cycle is often disrupted in many people. For example, approximately 15%–20% of the working population worldwide is engaged in shift work,[1] which is associated with increased risk of type 2 diabetes and worse glycaemic control.[2,3] Shift work results in shifting the timing of dietary intake, physical activity and sleep, which can all cause misalignment of our intrinsic central circadian clock. Since the circadian clock governs several neural, endocrine and behavioural processes,[4,5] a misaligned circadian clock may result in altered rhythms of downstream processes including autonomic nervous system processes, cortisol and melatonin secretion as well as temperature regulation.[6] These misalignments in turn lead to alterations in the regulation of the peripheral clocks of the gut, muscle, liver, fat and pancreas and, as a result, lead to impaired endocrine function, including disturbances in glycaemic outcomes.[6–13]

Circadian misalignment can thus be caused by shifting the timing of different behavioural factors. In this review, we will focus specifically on the timing of sleep, while other reviews focus on the timing of physical activity and dietary intake (published[14] or in progress). Although numerous studies[15–17] have investigated the association between sleep quality and sleep quantity and glycaemic markers, only a few studies specifically investigated the effect of altered sleep timing on glycaemic outcomes, and these showed inconsistent results.[18–22] Therefore, a systematic review, summarizing the current available evidence with regard to altered sleep timing, will help to understand the impact on a broader level and provide recommendations regarding future research. This systematic review thus aims to summarize results from human intervention studies of altered sleep timing on glycaemic outcomes.

2 | MATERIALS AND METHODS

2.1 | Data sources and search strategy

A systematic review was conducted in accordance with the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines.[23] This review was part of a series of four systematic reviews and meta-analyses, of which the protocol was registered in the PROSPERO database under number CRD42021287828 on

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27 November 2021. The series investigates the effect of altering timing of physical activity, sleep and dietary intake on glycaemia and (pre) diabetes risk in human trials. In this review, we focus exclusively on timing of sleep.

A full overview of the material and methods is described in a previously published review in this series[14] and provided in full in supplementary file S1. In short, a search was done using Medline via Ovid and Embase.com up until 24 February 2023. The search was restricted to clinical trials, with adult human participants (with no exclusion on health status), and measuring glycaemic outcomes (overview of the search strategy in supplementary file S2). Studies were included if the timing of sleep was acutely delayed by ≥2 h, either delayed sleep for just a few hours (e.g. sleep between 00.00–08.00 h and 03.30–11.30 h[19]) or entirely shift towards daytime sleep (e.g. sleep between 23.00–07.00 h and 07.00–15.00 h[24]). The minimum study duration was at least one night. Due to limited number of studies, heterogeneity between study designs and outcome measures and limited response from authors, results could not be meta-analysed. Furthermore, the lack of consistency in the way outcomes were measured, lack of reporting effect sizes and heterogeneity between studies prevented us from reporting the results in forest plots. More specifically, most studies did not provide sufficient data to calculate mean differences between study conditions (e.g. only a *p*-value or the association reported only in a figure) or provided different ways to measure a certain outcome (e.g. postprandial mean glucose was measured as mean value in some studies, but as area under the curve in others, which prevented us from combining these data in one forest plot). Therefore, we decided to plot the results in harvest plots, as also done in previous studies and suggested by Cochrane's recommendations.[25–28] Harvest plots allow to explore trends in the data across multiple outcomes, while including the quality of the studies. As harvest plots are solely reflecting the direction of the effect, these can combine different ways of measuring a glycaemic outcome, as well as include studies that do not report sufficient data to calculate a difference between groups. To make the harvest plots, vote counting was used: per type of glycaemic outcome, the amount of studies that reported on this outcome were counted and added in a harvest plot based on the direction of the effect.[27,28] This was done by two independent reviewers and discussed until consensus or resolved through discussion with a third reviewer.

3 | RESULTS

3.1 | Description of included studies

A total of 55 569 publications were identified from the systematic literature search, 20 732 publications were excluded as duplicates and 30 249 were marked as not relevant by the screening tool ASReview.[29] After manually screening 4588 publications based on title and abstract, 383 full-text publications were read and screened for the topics in our series of systematic reviews and meta-analyses. Of these, 33 were potentially relevant for this review and 12 publications met the inclusion criteria. Reasons for exclusion after full-text screening are listed in supplement S3. Two relevant publications were found in the reference lists of included studies, which were not in the original search, making data extraction possible for a total of 14 studies (Figure 1).

A total number of 159 participants (72% were men, mean age ranged between 20 and 34 years) were included. All studies included a population with no known metabolic condition other than overweight in one study.[19] Three studies[30–32] included shift workers, seven studies[19,20,22,33–37] included non-shift workers without extreme bed-times and/or no travel across time zones and three studies[18,24,38] did not specifically mention usual bed-times, chronotype or shift work. In most studies, participants were encouraged to either maintain their usual activity level or refrain from any physical activity other than their normal activities. Almost all studies included time in bed of 8 h, except for three studies, with 4 h,[20] 5 h[33] and 12 h[31] in bed, respectively. Bedtimes were shifted acutely for ±3.5 h in two studies,[19,20] while all other studies acutely shifted by 8–15 h in one night. Study duration per trial arm ranged from 24 h to 8 days. A detailed overview of the study characteristics is provided in Table 1.

Finally, methodological quality was considered high in four studies,[19,30,31,33] moderate in seven studies[18,20,24,32,34,35,38] and low in three studies[22,36,37] (Table 2). The most common reason for predisposing studies to bias was the lack of information about withdrawals and dropouts.

3.2 | Vote counting

A total of 27 different outcomes could be extracted and analysed using vote counting, as depicted in Figure 2. Ten of the 27 glycaemic outcomes showed unfavourable effects in the group that had delayed sleep timing, compared to (early) nighttime sleep. More specifically, postprandial glucose response and postprandial insulin response were higher in delayed sleep, compared to (early) nighttime sleep in six out of eight studies in both outcomes.[18,19,24,30–32,34] The remaining studies showed no clear direction in both outcomes.[24,31,38] Disposition index (DI), a measure for the glucose-insulin control system, showed unfavourable effects of delayed sleep in four out of five studies[19,31,33,34]; the remaining study[20] showed no direction. Last, postprandial glucose peak,[18,34] time to postprandial glucose peak,[31] time to postprandial insulin peak,[31] insulin sensitivity (SI),[33,34] postprandial insulin secretion rate (ISR) response,[34] insulin-stimulated glucose disposal[22] and non-oxidative glucose disposal (NOGD)[22] were measured in one or two studies per outcome, and these all showed unfavourable effects of delayed sleep. In contrast, glucose response during bedtime (which was altered to the actual time in bed instead of the clock time)[24,35,37] and Matsuda index[19] showed favourable effects of delayed sleep in three out of four studies and one single study, respectively. All other outcomes showed no clear differences between delayed and early sleep time, namely 24 h glucose (sampling interval 10–60 min), fasting glucose, glucose peak over 24 h, fasting insulin,

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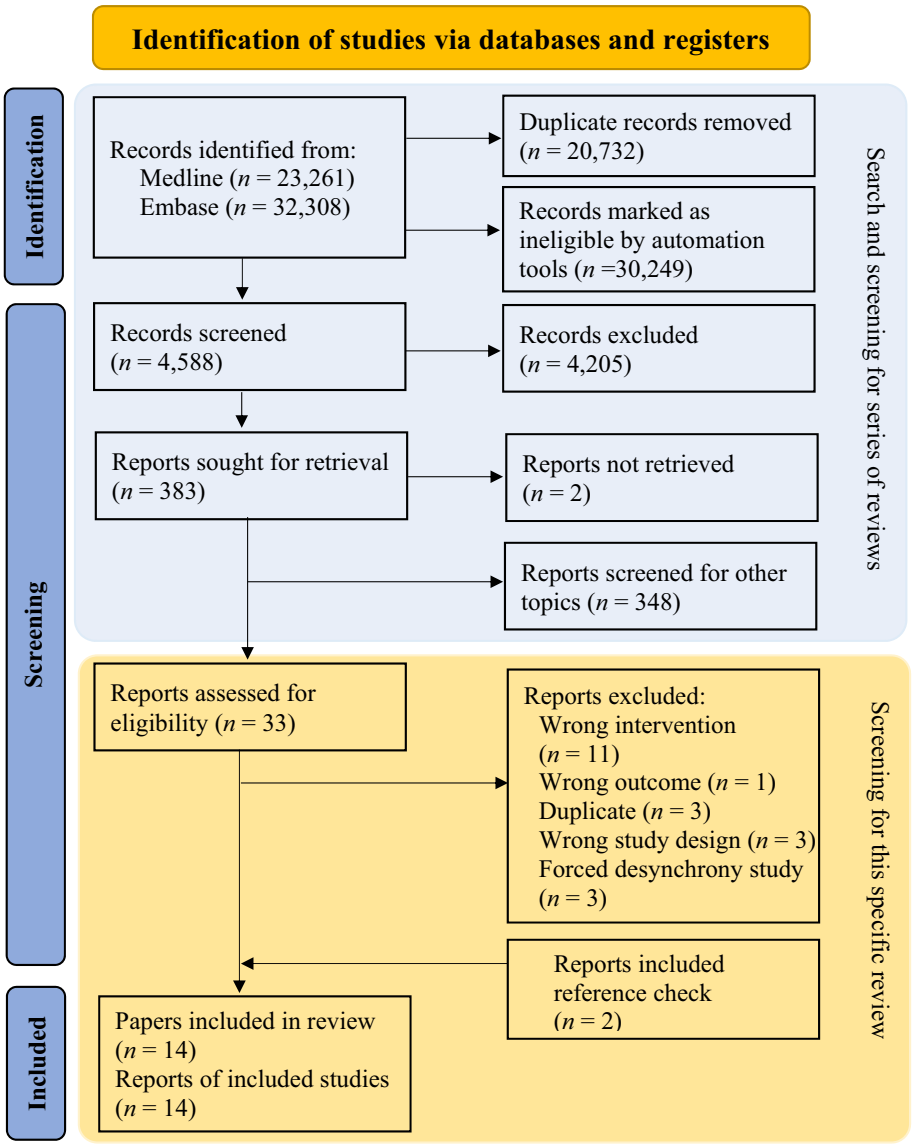

FIGURE 1 Flow chart of the search and selection process. This study is part of a series of systematic reviews and meta-analyses. The flow chart therefore partly consists of data from the series (highlighted in blue). From 'Reports assessed for eligibility', only papers for this specific meta-analysis are reported (highlighted in yellow).

postprandial insulin peak, 24 h insulin (sampling interval 30–60 min), insulin during bed time, acute insulin response to glucose (AIRg), fasting ISR, 24 h ISR, glucose oxidation, ISR during bed time, peak ISR over 24 h and M-values.

4 | DISCUSSION

This systematic review aimed to summarize results from human intervention studies of altered sleep timing on glycaemic outcomes. The 14 included studies showed that delaying sleep timing may have unfavourable effects on some glycaemic outcomes, specifically postprandial outcomes, compared to (early) nighttime sleep. However, studies were rather heterogeneous, resulting in inconclusive results in other outcomes. Furthermore, no studies reported on more long-term outcomes, such as development of (pre) diabetes, as all included studies looked into short-term effects (between 1 and 8 days) which prohibits providing information on chronic effects. Finally, most studies had a high to moderate quality, strengthening the interpretation of our findings.

Our results are supported by several narrative reviews,[6,8,12,13] which generally concluded that altered sleep influences the biological clock and therefore increases glycaemia and reduces insulin sensitivity. A few studies, included in our systematic review, studied possible mechanisms driving the association between delays in sleep timing and adverse glycaemic outcomes. These include, for example, altered secretion of cortisol and melatonin as a result of changed sleep timing.[21,40,41] Namely, delaying sleep towards early in the morning or daytime sleep results in misalignment between the sleep–wake cycle and the light–dark cycle. The circadian rhythm, generated by the central biological clock in the suprachiasmatic nuclei, is synchronized to the environmental light/dark cycle by light and governs the circadian rhythms of melatonin and cortisol.[42,43] During the dark night, a period where we normally sleep, melatonin is at its highest and cortisol is low, which reduces insulin sensitivity and glucose tolerance.[41,44] When sleep is delayed by more than 6–8 h, the body is active and

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TABLE 1 Characteristics of included studies.

First author, year	Study design	Type of patients	*N* (M/W ratio)	Age: mean (± SD)	Follow-up time	Intervention	Control	Glycaemic outcomes	Comments
Hampton et al.[18]	Single-arm trial	Healthy	9 (6/3)	22 ± 2.46	Intervention was 15 days, but 3 nights C and 3 nights I	8 h time in bed14:30–22:30 h; Mealtimes shifted in parallel with the light/darkness cycle	8 h time in bed 23:30–7:30 h; mealtimes shifted in parallel with the light/darkness cycle	Fasting, AUC, peak response, time to peak response of glucose and insulin, glucose/insulin ratio	
Leproult et al.[33]	Non-randomized parallel group design	Healthy; no shiftwork or travel across time zones during the past 2 months.	C: 13 (10/3) I: 13 (9/4)	C: 23 (21.5–25.5)[a] I: 22 (21.5–24.5)[a]	Two parallel 8-day interventions	5 h time in bed 9:00–14:00 h on day 5, 6, 8 and 9 and 0:30–5:30 h on the other days; standardized meals	5 h time in bed 0:30–5:30 h; standardized meals	SI, AIRg, DI (IVGTT)	
Lund et al.[32]	Non-randomized crossover	Healthy shift workers	12 (10/2)	28 ± 6.6	3 test meals during a 21-day period, 7 days per shift in sleep times	8 h time in bed 8:00–16:00 h; standardized test meals	8 h time in bed 0:00–8:00 h; standardized test meals	Postprandial glucose and insulin AUC	
Morris et al.[34]	Randomized crossover	Healthy; no shiftwork >3 years ago, < 6 months shiftwork in lifetime, > 3 months no travel across time zones	14 (8/6)	28 ± 9	4 days per trial (2–8 weeks washout period)	8 h time in bed 11:00–19:00 h; Isocaloric diet	8 h time in bed 23:00–7:00 h; Isocaloric diet	Fasting and AUC of glucose, insulin and ISR, peak glucose response, 24 h mean of glucose and insulin	The paper of Qian et al.[39] reports on the same study and includes SI and DI
Morris et al.[30]	Randomized crossover	Healthy chronic shift workers; shifts in sleep/wake schedule on days off	9 (3/6)	34 ± 8	3 days per trial (3–8-week washout period)	8 h time in bed 11:00–19:00 h; isocaloric diet	8 h time in bed 23:00–7:00 h; isocaloric diet	Fasting, postprandial and 24 h mean of glucose and insulin	
Pizinger et al.[19]	Randomized crossover	Overweight; only intermediate chronotype	6 (4/2) 1 W failed to complete all phases	25.1 ± 3.9	5 nights per trial (3 weeks washout period)	8 h time in bed 3:30–11:30 with isocaloric meals 1, 5, 11 and 12.5 h after awakening	8 h time in bed 00:00–8:00 h with isocaloric meals 1, 5, 11 and 12.5 h after awakening	SI, AIRg, DI (FSIVGTT); glucose and insulin AUC, Matsuda index (MMT)	Also two trials with late meal times are conducted
Rehman et al.[24]	Non-randomized crossover	Healthy	7 (7/0)	25 (22–32)[b]	25 h per trial (three occasions, no information on washout)	8 h time in bed 07:00–15:00 h; isocaloric meals at 22:00 h and 16:00 h, 02:00 h and 06:00 h	8 h time in bed 23:00–7:00 h; isocaloric meals at 22:00 h and 16:00 h, 8:00 h and 12:00 h	Postprandial, 24 h, mean during bed for glucose and insulin	
Ribeiro et al.[38]	Single-arm trial	Healthy	12 (4/8)	24.1 ± 2.01	14 days (4 days baseline, 5 days phase shift, 5 days back to baseline)	8 h time in bed 14:30–22:30 h; mealtimes shifted in parallel with the light/darkness cycle	8 h time in bed 23:30–7:30 h; mealtimes shifted in parallel with the light/darkness cycle	Fasting and postprandial AUC of glucose and insulin	

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TABLE 1 (Continued)

First author, year	Study design	Type of patients	*N* (M/W ratio)	Age: mean (± SD)	Follow-up time	Intervention	Control	Glycaemic outcomes	Comments
Simon et al.[35]	Randomized crossover	Healthy; normal routines of work, meals and sleep	7 (7/0)	20–28[c]	24 h per trial (1–2 month washout period)	8 h time in bed 07:00–15:00 h; continuous enteral nutrition	8 h time in bed 23:00–07:00 h; continuous enteral nutrition	24 h mean glucose and ISR, mean glucose during bed	
Simon et al.[36]	Randomized crossover	Healthy day-active; normal routines of work, meals and sleep	8 (8/0)	23–30	48 h per trial (1 month washout period)	One night of 8 h time in bed 23:00–07:00 h and then an acute shift with 8 h time in bed 07:00–15:00 h; continuous enteral nutrition	Two night of 8 h time in bed 23:00–07:00 h; continuous enteral nutrition	24 h mean glucose and ISR, peak glucose	Also in night-shift workers
Sharma et al.[31]	Randomized crossover	Healthy nurses performing rotational shiftwork	12 (2/10)	25 ± 3.46	3 days per trial (2–6 weeks washout period)	Two consecutive 12 h shifts 19:00–7:00 h. admission to clinical research unit after these 2 days, 12 h darkened conditions during the day; Standardized mixed meals with glucose infusion	Two consecutive 12 h shifts 7:00–19:00 h. admission to clinical research unit after these 2 days, 12 h darkened conditions during the night; standardized mixed meals with glucose infusion	Fasting, postprandial AUC, peak response, time to peak of glucose and insulin, DI	
Van Cauter et al.[37]	Single-arm trial	Healthy; no shiftwork or travel across time zones <60 days before study	8 (8/0)	22–27	2 days in total	8 h time in bed 11:00–19:00 h; continuous dextrose infusion for 57 h	8 h time in bed 23:00–07:00 h; continuous dextrose infusion for 57 h	Peak response and 24 h mean of glucose and insulin, 24 h mean ISR	
Wefers et al.[22]	Randomized crossover	Healthy; regular bedtimes (11 PM ± 2 h)	14 (14/0)	22.4 ± 2.8	4 days per trial (4–10 weeks washout period)	Day 1 8 h time in bed 23:00–7:00 h, day 2 4 h bedtime 15:00–19:00 h, day 3–4 8 h bedtime 11:00–19:00 h; isocaloric meals	8 h time in bed 23:00–7:00 h; isocaloric meals	Fasting glucose and insulin, EGP, insulin-stimulated glucose disposal, NOGD, glucose oxidation (CLAMP)	
Wilms et al.[20]	Randomized crossover	Healthy; no shiftwork or travel across time zones <4 weeks before study	15 (15/0)	24.6 ± 2.71	1 day per trial, 3 weeks washout	4 h time in bed 02:15–06:45 h; standardized diner at 20:15 h	4 h time in bed 22:30–03:00 h; standardized diner at 20:15 h	Fasting glucose, SI, DI	Also a condition with 8 h sleep

Abbreviations: AIRg, acute insulin response to glucose; AUC, area under the curve; C, control condition; CLAMP, two-step hyperinsulinemic euglycemic clamp; DI, disposition index; EGP, endogenous glucose production; (FS)IVGTT, (frequently sampled) intravenous glucose tolerance test; HOMA-IR, homeostatic model assessment for insulin resistance; I, intervention condition; ISR, insulin secretion rate; M, men; MMT, mixed meal test; N, number of participants; NOGD, nonoxidative glucose disposal; SD, standard deviation; SI, insulin sensitivity; W, women.

[a]Median (25th–75th percentiles).

[b]Mean (range).

[c]Range.

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TABLE 2 Quality assessment of included studies.

First author, year	SD	BL	RSB	RWD	CF	DC	DA	RP	Overall
Hampton et al.[18]	S	NR	M	W	S	S	S	M	Moderate
Leproult et al.[33]	S	NR	M	S	M	S	S	S	High
Lund et al.[32]	S	NR	M	W	S	S	S	M	Moderate
Morris et al.[34]	S	NR	M	W	S	S	S	S	Moderate
Morris et al.[30]	S	NR	M	S	S	S	S	S	High
Pizinger et al.[19]	S	NR	M	M	S	S	S	S	High
Rehman et al.[24]	S	NR	M	W	S	S	S	M	Moderate
Ribeiro et al.[38]	S	NR	M	W	S	S	S	M	Moderate
Simon et al.[35]	S	NR	M	W	S	S	S	M	Moderate
Simon et al.[36]	S	NR	W	W	S	S	S	M	Low
Sharma et al.[31]	S	NR	M	S	S	S	S	S	High
Van Cauter et al.[37]	S	NR	W	W	S	S	S	M	Low
Wefers et al.[22]	S	NR	M	W	S	W	S	S	Low
Wilms et al.[20]	S	NR	M	W	S	S	S	S	Moderate

Abbreviations: BL, blinding; CF, confounding; DA, data-analysis; DC, data-collection; M, moderate; NR, no rating; RP, reporting; RSB, representativeness with regard to selection bias; RWD, representativeness with regard to withdrawals/dropouts; S, strong; SD, study design; W, weak.

food is consumed in a period that levels of melatonin are high and cortisol is low, which causes disturbances in glycaemic control.[45] However, following the circadian disruption hypothesis, metabolic health is influenced by different daily rhythms, including behavioural, hormonal and autonomic nervous system rhythms. These rhythms oscillate in synchrony, and all influence the central and different peripheral clocks in the body.[6] More research is needed to clarify these mechanisms and conclude the combination of pathways through which the timing of sleep is associated with glycaemic control.

Nevertheless, included studies were rather heterogeneous and resulted in some inconclusive results. This could have different explanations. First, as mentioned, all studies were short-term (1 to 83 days) and the time period may be too short to detect any changes. Namely, available studies that do support a chronic effect of altered sleep timing on glycaemia are observational studies (not included in this review), which include shift workers shifting their sleep times for multiple years, who have demonstrated a propensity towards reduced glucose tolerance and increased risk of type 2 diabetes.[2,3] Results may be altered in chronic shift workers. However, we could only include three studies[30–32] with shift workers, with limited outcomes. We therefore could not stratify between shift workers and participants working only day shifts, but this should be examined in future research. Second, although most studies included the recommended time in bed of 7–9 h,[46,47] it is expected that sleeping in the research facilities has influenced both sleep quality and quantity. Most studies only included time in bed, and we could therefore not take the actual sleep duration and sleep quality into account. Studies that did include measures on sleep mostly found no differences in total sleep time and only some minor changes in sleep stages.[19,20,24,33,36,37] Two studies[30,34] did find decreased total sleep time in delayed sleep, but this did not explain the variance found in glucose tolerance. No comparisons were made in sleep quality and sleep quantity between the free-living nights and the research visits. Previous research has shown that reduced sleep duration and sleep quality are associated with hyperglycaemia and increased risk of type 2 diabetes.[16,48–50] The reduction of sleep quantity and quality could likely increase glucose metabolism irrespective of the sleep timing and could blunt the effect of sleep timing, making it impossible to separate the effects of sleep timing, sleep quality and sleep duration. Third, as earlier mentioned, also timing of other behavioural factors could influence glycaemic variability[40,42,43] and when sleep is delayed or shifted towards daytime, the body is active and food is consumed in a period where insulin sensitivity is reduced. Some studies shifted the timing of energy intake along with the sleep times, while others did not. However, there were not enough studies to separate these results. We can thus not conclude whether results were caused by shifts in sleep time, or a combination of shifting different behavioural factors. Apart from this, Ribeiro and colleagues[38] mention that meal composition may also influence results, as they found no differences in pre- and post-phase shifts when a high-carbohydrate meal was employed, while differences were found in an earlier similar study[18] with a high-fat meal composition. This suggests that meal composition may eliminate the effect of alterations in sleep timing and result in the absence of a clear effect in some studies. Nevertheless, all but two studies used isocaloric and standardized meals or continuous dextrose infusion. The two studies that did not mention this did use standardized test meals throughout the measurements. We thus do not expect differences within studies. Fourth, most studies shifted sleep with multiple hours towards daytime sleep, while two studies[19,20] only shifted sleep by approximately 3.5 h. This may have great effect on the results, as sleep is either completely during regular daytime, or still during the night. These small shifts in sleep time are also seen when people have a late chronotype and their biological sleep time does not correspond to social and work related obligations, also called social jetlag.[51,52]

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FIGURE 2 Harvest plots of the results of the acute phase shift studies. AIRg, acute insulin response to glucose; DI, disposition index; ISR, insulin secretion rate; NOGD, non-oxidative glucose disposal; SI, insulin sensitivity.

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Included studies either did not specify chronotype or only included intermediate chronotypes.[19,22,35,36] Last, studies differed greatly in other factors of the study design, such as study duration, washout period and standardization of physical activity and sleep the days before the intervention. However, due to the relatively small amount of studies and limitations in reported outcome measures, the above-mentioned hypotheses could not be tested in separate analyses and should be taken in consideration when interpreting the results.

4.1 | Strengths and limitations

The findings of this review should be seen in the light of some limitations. First, the included studies were characterized by substantial heterogeneity due to differences in study design, quality and the differences in intervention, preventing us from conducting meta-analyses or doing further sensitivity analyses to examine aforementioned hypotheses of unclear results. Although vote counting enabled us to visualize the data and was done in accordance to the Cochrane's recommendations,[27,28] this procedure has some limitations and is less powerful than a meta-analysis, as it does not include magnitude and statistical significance of the effect size. Therefore, we cannot draw firm conclusions about the clinical relevance and statistical significance of the results. Second, the clocks in the body are not only influenced by sleep, but also by other 'zeitgebers' such as light, physical activity and food intake.[13] We tried to separate these different factors in our series of reviews (other meta-analyses published[14] and in progress). However, we see that sleep, dietary intake and physical activity are often all shifted in one study, making it impossible to isolate one factor. Third, almost all participants were lean adults without pre-existing metabolic conditions, except in one study, that included overweight participants.[19] The participants were also from a relatively young population. We thus cannot compare results between different health statuses and across age ranges.

Nevertheless, there are also several strengths to this review. This is the first systematic review attempting to summarize the available knowledge on the effect of altered sleep timing on glycaemic outcomes. Additionally, we conducted this systematic review in accordance with the PRISMA guidelines, with an extensive electronic and manual search strategy, which attributes to the quality of this review. This reviews gives an overview of the current available evidence and reveals research gaps to design and conduct future research.

4.2 | Clinical implications and perspectives

The current guidelines regarding sleep indicate that adults should sleep between 7 and 9 h per night to promote optimal health.[46,47,53] These guidelines do not involve recommendations regarding the timing of sleep. The results of this systematic review suggest that there might be benefits to sleeping at night-time and that delaying our sleep timing should be prevented. However, most included studies were heterogeneous, short term and mostly completely shifting sleep between day and night. Therefore, further research is needed to create these recommendations. These should include controlled trials in which sleep during the early night is compared with sleep later at night to be able to assess the effect in a more real-life setting. These studies should also measure sleep quantity, sleep quality, physical activity and dietary intake, in order to correct for differences and isolate the effect of altered sleep time. This should be examined over multiple weeks, in diverse populations, including shift workers, different chronotypes and people with type 2 diabetes, to estimate the impact in daily living and comment on the impact on incident (pre) type 2 diabetes. Last, studies should be more transparent in their design, outcome assessment and reporting, to make comparisons between studies possible.

Furthermore, not only the timing of sleep may contribute to the growing prevalence of type 2 diabetes. Numerous studies have shown the risk of poor sleep quality and short sleep duration to the development of type 2 diabetes.[49,54,55] Many factors precede these pathways, such as, but not limited to, smartphone over-use,[56,57] caffeine intake as far as 8 h before bedtime[58] and smoking.[59] Additionally, factors such as the timing of physical activity, timing of meal intake and light exposure may also alter the human circadian clock.[6,43,44,60] The combination of these factors, rather than solely the timing of sleep, may cause circadian misalignment and increase the risk of type 2 diabetes and hyperglycaemia. The timing of sleep should thus not be seen as an isolated factor associated with type 2 diabetes incidence. We hypothesize that the combination of these factors together contributes to the growing prevalence of type 2 diabetes, and the combination should be investigated to successfully prevent type 2 diabetes and reduce hyperglycaemia.

4.3 | Conclusion

This systematic review showed that acutely delaying sleep timing might have unfavourable effects on glycaemic outcomes, compared to (early) nighttime sleep. Future research does however need better controlled trials, which should control for sleep quantity, sleep quality and keep physical activity and energy intake constant, with longer follow-up periods, consistent outcomes and designs and more diverse populations to provide targeted advice regarding the optimal timing for sleep.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

PEER REVIEW

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/dom.16104.

DATA AVAILABILITY STATEMENT

The data, code and other materials that underlie the results reported in this article are available from hoornstudy@amsterdamumc.nl upon reasonable request to researchers who provide a methodological sound proposal and after approval by the Hoorn Steering Committee.

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