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REVIEW

Sleep and Circadian Rhythm Disturbances in Diabetes: A Narrative Review

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Abstract: Sleep and circadian rhythm disturbances are less-known risk factors for the development and suboptimal outcomes of diabetes. The goal of this narrative review is to highlight the importance of sleep and circadian rhythm disturbances in the development and outcomes of type 1 diabetes (T1D) and type 2 diabetes (T2D), assess current treatment options and the possible mediating mechanisms. We performed a literature search using PubMed and selected relevant English and Dutch papers. Disturbances of sleep and circadian rhythm are common in people with diabetes. They are associated with an increased risk of developing T2D as well as with suboptimal diabetes outcomes (including higher HbA_{1c} levels and reduced quality of life) for T1D and T2D. Preliminary data suggest that treatment of sleep and circadian rhythm disturbances could improve diabetes outcomes in people with T1D and T2D. Finally, the association with medical parameters appears to be mediated by disturbance in hormones, and by suboptimal self-care including forgetting or postponing glucose monitoring or medication use as well as higher consumption of high fat/high sugary foods. Diabetes may also disturb sleep, for example through nocturnal hypoglycemia and nocturia. We concluded that sleep and circadian rhythm disturbances are closely linked with diabetes. More attention to sleep in regular diabetes care is warranted, while further research is needed on treatment of sleep and circadian rhythm disturbances in the prevention of diabetes and its suboptimal outcomes.
Keywords: sleep, sleep disorders, circadian rhythm, type 1 diabetes, type 2 diabetes

Introduction

Diabetes is a common metabolic non-communicable condition with an increasing prevalence. Almost 90% of the people with diabetes have type 2 diabetes (T2D) and the biggest part of the remaining 10% has type 1 diabetes (T1D).[1] T1D and T2D have different pathophysiology; however, both types include altered glucose metabolism, which can lead to microvascular and macrovascular complications and premature mortality.[2,3] Diabetes and its daily self-management may negatively affect a person's quality of life and increase the risk of depression and other psychological problems.[4,5]

Apart from non-modifiable factors such as increasing age and genetic predisposition, the development of T2D is associated with obesity and lifestyle factors such as unhealthy eating, sedentary behavior and smoking.[6] In both T1D and T2D, these lifestyle factors play a role in suboptimal diabetes outcomes, such as high HbA_{1c} levels throughout the course of treatment.[7,8] Lifestyle factors that are not often mentioned in this context, are sleep and circadian rhythm.

Sleep is defined as a state of inactivity with a reduced responsiveness to all external stimuli.[9] It is a complex phenomenon, as it involves several dimensions (eg, disorders, deficiencies, descriptives) and levels of analysis (eg, self-report, objective measures such as polysomnography).[10] Most sleep studies to date have focused on duration, quality and sleep timing (the placement of sleep in a day).[11] The latter is important as sleep timing serves as a zeitgeber for the circadian rhythm. The circadian rhythms are the physical, mental, and behavioral changes that follow a 24-hour cycle. The endogenous rhythm responds primarily to light and dark. In this modern society with 24/7 light access, many face

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disturbances in sleep and circadian rhythm.[12] This asynchrony between endogenous rhythms and behavioral sleep-wake schedules is termed circadian misalignment.[13]

The goal of this narrative review is to highlight the importance of sleep and circadian rhythm disturbances in the development and outcomes of T1D and T2D, current treatment options and the possible mediating mechanisms.

Materials and Methods

A literature search was performed in PubMed from inception until August 2022, using MeSH and tiab search terms indicating sleep: eg, "sleep", "sleep duration", "sleep disorder", "sleep deprivation", "circadian", "social jeltag", "insomnia", "obstructive sleep apnea", "restless legs syndrome", "shift work", "jetlag"; and diabetes mellitus: eg, "dm 1", "insulin dependent", "dm 2" and "non-insulin dependent". Additionally, search terms for prevalence, treatment and health outcomes were used: eg, "morbidity", "prevalence", "chronobiology", "sleep drug", "sleep medication" and "health status". All relevant English or Dutch language original and review studies were read by the authors and summarized. Observational (cross-sectional and longitudinal), experimental and interventional studies were included.

Results

Development of Diabetes

Sleep Duration

In the past decade, quite a large amount of evidence has been collected that shows an association between short sleep duration (often defined as <6h) and the development T2D. A recent meta-analysis of 21 experimental studies showed that sleep restriction reduced insulin sensitivity assessed by several methods, including glucose tolerance tests, homeostatic model assessment of insulin resistance and the hyperinsulinemic euglycemic clamp.[14] In a second meta-analysis of observational studies, when compared to other lifestyle factors such as physical inactivity, short sleep duration was associated with a 1.48 higher odds (95% CI: 1.25, 1.76) of incident diabetes.[15] An older meta-analysis of prospective observational studies with a follow-up duration of more than three years reported a relative risk (RR) of 1.28 (95% CI 1.03–1.60, p=0.024).[16] A recent umbrella review focusing on all possible health outcomes of short sleep did not show a significant association of sleep duration with diabetes development, however there were only few meta-analyses with diabetes as an outcome included.[17] The umbrella review did show limited evidence for a higher risk of obesity, an important T2D risk factor.[17]

In addition to short sleep, long sleep duration (often defined as >9h) has also been associated with the development of T2D. In the Anothaisintawee et al meta-analysis, when compared to other lifestyle factors such as physical inactivity, long sleep duration was associated with a 1.36 higher odds (1.12, 1.65) of incident diabetes.[15] Cappuccio et al found a RR of 1.48 (95% CI 1.13–1.96, p=0.005).[16] In the Gao et al umbrella review, long sleep was not associated with diabetes, but they did show a 5-times increased risk of stroke, dyslipidemia and cardiovascular mortality.[17] Overall, there seems to be a U-shaped association between short and long sleep in the development of T2D, with the lowest risk at 7–8 hours per day.[18]

Sleep Quality

In addition to sleep duration, there has been quite some evidence on the role of suboptimal sleep quality (often measured as difficulty falling asleep, frequent waking up during the night, low self-reported sleep quality) as well as sleep disorders (ie, insomnia, sleep apnea, restless leg syndrome) and the development of T2D. First, a recent meta-analysis including four studies that fragmented sleep or suppressed certain sleep stages showed that sleep fragmentation had no effect on markers of insulin sensitivity, while certain types of sleep stage (especially slow wave sleep) suppression did show reductions in insulin sensitivity, glucose tolerance and beta-cell function.[14] Second, in an older systematic review by Cappuccio et al, they showed a relative risk of T2D incidence for difficulty in initiating sleep of 1.57 (1.25–1.97) and for difficulty in maintaining sleep, of 1.84 (1.39–2.43).[16] In the Anothaisintawee et al meta-analysis, compared to other lifestyle factors, obstructive sleep apnea syndrome (OSA) (sleep-disordered breathing with repetitive upper airway collapse) was associated with a RR of 1.49 (95% CI: 1.27, 1.75) of incident diabetes, while difficulty maintaining sleep and difficulty initiating sleep were associated with a RR of 1.74 (95% CI: 1.30, 2.34) and 1.55 (95% CI: 1.21,

1.99).[15] In the Gao et al umbrella review, suboptimal sleep quality was not significantly related to diabetes development, however there were only few meta-analyses included with diabetes as an outcome, reducing statistical power.[17]

Circadian Rhythm

In the past years, there has been more attention to not only sleep duration and quality, but also the role of sleep timing in metabolic functioning. In a recent meta-analysis of 5 experimental studies, which exposed participants to circadian misalignment, a negative association with insulin sensitivity was shown.[14] Many observational studies have focused on the extreme form of circadian rhythm disturbance, which is shift work. A recent meta-analysis of 19 studies indicated that shift work was associated with an increased risk of T2D (relative risk = 1.10, 95% CI 1.05–1.14).[19] In the Anothaisintawee et al meta-analysis, compared to other lifestyle factors, shift work was associated with a RR of 1.60 (95% CI: 1.20, 2.14) of incident diabetes.[15]

There are several studies, showing that a more subtle form of circadian disturbance, namely sleep variability (varying the time of going to bed and varying the time of getting up during the week, excluding shift work) is related to development of T2D. In a narrative review, Zuraikat et al 2020 showed that higher standard deviations (SD) across nights of sleep duration and onset or midpoint of sleep were associated with increased odds of having the precursor of T2D, namely the metabolic syndrome, as well as with higher Hba_{1c} levels. Conversely, greater regularity of rest-activity patterns related to lower risk for T2D.[20]

Finally, there is a highly prevalent form of sleep variability and circadian disturbance, namely social jetlag. Social jetlag is defined as the discrepancy between work schedules, social obligations and biological need for sleep.[21–23] Especially evening types, people with a late chronotype, have social jetlag.[23,24] In our recent (unpublished) meta-analysis, we showed that in high-quality studies social jetlag was significantly associated with higher BMI as well as higher HbA_{1c} levels, compared to no social jetlag. No statistically significant associations were observed for T2D development.[25]

Overall, limited to no evidence has been found on the role of (maternal) sleep (duration or quality) as well as circadian disturbance in the development of T1D. A prospective observational study found an association of sleep initiation and maintenance with adult-onset autoimmune diabetes (hazard ratio 95% CI 1.01–2.22), but there were only a limited number of people with "classical" T1D in this group.[26] Another preliminary observational study suggests that maternal obesity, in the absence of maternal diabetes, is a risk factor for T1D in the offspring.[27] Similarly, in a large observational study among adolescents, body mass index was associated with increased risk for incident T1D in early adulthood.[28] Given the close link of obesity with sleep and circadian rhythm disturbances,[29] these factors may indirectly contribute to T1D development. More research is, however, needed to confirm that hypothesis and enlighten the mechanisms.

Prevalence

Sleep and circadian rhythm disturbances are common in people with diabetes. The highest prevalence estimates even run over 90%, with problems varying from inadequate sleep, sleep deprivation, nocturia, apnea and leg symptoms.[30] The present section focuses on prevalence comparisons with general sleep recommendations, people without diabetes and across diabetes types.

Sleep Duration

Many children <5 years with T1D did not meet the recommended sleeping time, which is approximately 10–13 hours per day, even if naps and overnight sleeping times were combined.[31] Also, studies including older children between 5 and 12 years old demonstrated an average total sleeping time at the lower end of the recommended time in children with T1D.[32] Similar patterns of short sleeping times were observed in adolescents or adults with T1D, when compared to peers without diabetes.[33,34] Furthermore, a small meta-analysis showed that adults with T1D with optimal HbA_{1c} levels (<53 mmol/mol (7%)) had a longer sleep duration (17.3 minutes), compared to those with a suboptimal HbA_{1c} levels.[35] Short sleep is not limited to T1D. Research demonstrated the prevalence of insufficient sleep (compared to age recommendations) ranged from 38% to 97% among children, adolescents and adults with T2D.[36,37]

Sleep Quality

Among parents of 2- to 12-year-old children with T1D, 67% reported their child met criteria for suboptimal sleep quality.[38] In the meta-analysis by Reutrakul et al, adults with T1D reported more pronounced suboptimal sleep quality (MD 0.51; 95% CI = 0.33, 0.70), compared to those without T1D.[35] This is in line with findings among people with T2D, as for example shown by Birhanu et al 2020, who found a prevalence of suboptimal sleep quality of 47.2% (95% CI: 42.5–52.1).[39] In the Dutch Diabetes MILES study, we directly compared suboptimal sleep quality (PSQI-score >5) prevalence across diabetes types, and reported 31% suboptimal sleep in adults with T1D and 42% in adults with T2D.[40] Finally, in a small study by Barone et al, adolescents with T1D had higher sleep variability, compared to their healthy peers.[41]

With regard to sleep problems, we have provided an elegant review on the topic last year.[42] For example, we found the pooled prevalence of insomnia (symptoms) in people with T2D to be 39% (95% confidence interval, 34–44),[43] while the prevalence of obstructive sleep apnea (OSA) in adults with T1D was 51.9% (95% CI = 31.2, 72.6).[35] Overall, we showed lower sleep quality in people with diabetes, while the prevalence of sleep problems is higher than the general population.

Circadian Rhythm

To our knowledge, there have been no reviews on the prevalence of circadian disturbances among people with diabetes. In studies among adults, the prevalence of social jetlag (≥1h) was 46% in T1D[44] and 58% in those with T2D.[45] However, given the age-dependence of social jetlag these numbers cannot be automatically generalized to the wider population. To illustrate, social jetlag appears highest in adolescents with T1D (mean, SD: 2.5, 1.2 hours)[46] and young adults with T1D (mean, SD: 1.62, 0.87 hours),[47] followed by adults with T1D (mean, SD: 53, 53 minutes;[44] median, interquartile range: 49, 24–79 minutes[48]) and adults with T2D (median, range 15, 0–304 minutes[49] and mean 35 minutes).[50] One study directly compared sleep timing parameters between adults with and without T2D, finding a difference in self-reported social jetlag (median 43 vs 23 minutes in people with versus without T2D), suggesting there might be a difference in the prevalence.[51]

Diabetes Outcomes

The following section will summarize evidence related to the association of sleep and circadian rhythm disturbances with suboptimal diabetes outcomes (glycemic measures, diabetes complications and mortality) in people with diabetes.

Sleep Duration

In recent years, there has been quite an extensive line of research on the association between short sleep duration and suboptimal diabetes outcomes. For example, children with T1D who have insufficient sleep have significantly higher HbA_{1c} levels, compared to children with T1D with sufficient sleep duration.[52] A systematic review of Ji et al demonstrated shorter sleep to be associated with higher HbA_{1c} levels in adolescents with T1D. Additionally, a review of Reutrakul et al found that adults with T1D who have short nights of sleep have a significantly higher HbA_{1c} level (+0.24%), compared to adults with T1D sleeping >6h per night.[35]

These higher $HbaA_{1c}$ levels could be explained by decreased insulin sensitivity. An experimental study of Donga et al investigated the effect of a single night of sleep restriction (4 hours) on glucose tolerance and insulin sensitivity in people with T1D.[53] The results showed that a single night of sleep restriction decreased insulin sensitivity with a reduction of insulin-stimulated glucose uptake by 14–21%. These findings are supported by other studies also in adults with T1D.[11]

For people with T2D, two meta-analyses containing >15 prospective studies showed a U-shaped association between sleep duration and HbA_{1c} levels. Short sleep was associated with a 0.23% higher HbA_{1C} levels and long sleep was associated with a 0.13% higher HbA_{1c} levels.[54,55] In addition to glycemic measures, the study of Meng et al demonstrated a higher risk of complications in adults with T2D and short sleep, including diabetic kidney disease and cardiovascular complications.[56] Finally, a meta-analysis showed short sleep duration also to be associated with the occurrence of diabetic retinopathy (OR = 1.49, 95% CI 1.15–1.94).[57]

Sleep Quality

The review of Reutrakul et al suggested that participants with T1D reporting high sleep quality had lower HbA_{1c}, compared to those with low sleep quality (MD = −0.19%; 95% CI = −0.30, −0.08).[35] With regard to diabetes complications, a recent meta-analysis of 7 articles including more than 4500 people with diabetes found that low sleep quality related to a higher occurrence of diabetic retinopathy.[57] With respect to sleep problems, our meta-analysis showed that in those with T2D having insomnia (symptoms), there are higher HbA_{1c} levels (mean difference, 0.23% [0.1–0.4]) and higher fasting glucose levels (mean difference, 0.40 mmol/L [0.2–0.7]),[43] compared to those without insomnia. In addition, among people with T1D with moderate-to-severe OSA there was a trend toward higher HbA_{1c} levels (MD = 0.39%, 95% CI = −0.08, 0.87).[35]

Circadian Rhythm

Also, circadian disturbance has been related to suboptimal glycemic outcomes. First, compared with day work, shift work was associated with significantly higher HbA_{1c} levels (shift workers had higher mean HbA_{1c} levels than non-shift workers; B=0.67, p<0.05) in both those with T1D[58] (ref) and (B = 0.059, p = 0.044) T2D.[59] Second, higher sleep variability was significantly associated with suboptimal glycemic outcomes, including glucose levels and medication use (B = 0.100, p = 0.004) in people with T1D.[60] Finally, also in our meta-analysis we showed that social jetlag was associated with suboptimal glycemic (ie, glucose) and metabolic outcomes (ie, hypertension) in people with diabetes (both types).[25]

Treatment

Treatment for sleeping problems may consist of pharmacological therapy or behavioral interventions. In a recent review assessing the effect of all types of sleep interventions on glucose metabolism,[61] no meta-analysis could be conducted for sleep medication due to large heterogeneity of the studies. However, the authors did provide a narrative synthesis, suggesting mixed results. First, the studies on orexin receptor antagonists showed an increase in glucose and reduction in HOMA-IR.[62,63] Second, the studies on benzodiazepines illustrated no change in insulin sensitivity, and a lowering effect on glucose.[64,65] Third, several studies investigated melatonin, which showed no effect on glycemic parameters.[61] However, Smirnova et al[66] investigated lifestyle advice plus metformin and prolonged-release melatonin 2 mg, which showed a decrease in HOMA-IR as well as an improvement in sleep onset latency and nighttime awakenings. Overall, these studies suggest mixed results for sleep medication on glucose metabolism. Due to the highly addictive nature of the medication, the American Academy of Sleep Medicine prefers behavioral approaches, such as Cognitive Behavior Therapy (CBT) over medication for the treatment of sleeping problems.

CBT-based behavioral strategies consist of cognitive techniques (eg, reducing catastrophizing cognitions about sleep), sleep hygiene, stimulus control, relaxation and mindfulness exercises, and sleep restriction therapy. Multiple studies demonstrated positive effects of CBT-based sleeping interventions on sleep quality,[61] glucose and HbA_{1c} levels (although not always statistically significant)[61] as well as diabetes self-care management.[67] Approaches may be tailored to specific sample needs. For example, while the "Sleep Coach" intervention for adolescents with T1D includes education on sleep habits, coping techniques and relaxation/mindfulness,[68] the adapted version for children aged 5–9 years and their parents ("Sleep coach Jr") also addresses sleeping difficulties such as bedtime resistance and nighttime waking with developmentally appropriate strategies including a "bedtime pass".[69] Another possible behavioral intervention less used in regular care but investigated in research is sleep extension. The aim of sleep extension is to prolong the time in bed with 1–1.5 hours, using behavioral strategies. A recent meta-analysis from 42 studies in the general population, including people with diabetes, showed that interventions resulted in a significantly higher sleep duration, 0.80h (95% CI 0.28 to 1.31). Subgroup analyses revealed that studies directly intervening on sleep duration (ie, specifying the sleep schedule) had larger effects compared to indirect methods (coaching, educational approaches).[70] In a preliminary randomized trial among young people with T1D, intervention participants did increase sleep duration as compared to the control group (41 vs 6 minutes), however less than half were likely to continue with the changes in their sleep schedule.[71] A second review on the metabolic effects of sleep extension, also in the general population (including T1D and T2D), demonstrated a lower HOMA-IR and improved beta cell function after sleep extension.[61]

Additionally, there are treatments for specific sleep problems, such as Continuous Positive Airway Pressure (CPAP) or mandibular devices for sleep apnea, which all have been related to improved glycemic outcomes when used in people with diabetes.[42] But also pramipexole treatment for restless leg syndrome showed a decrease in related complaints as well as a change in HbA_{1c} levels of −3.2 mmol/mol (95% CI −4.4, −2.2).[72] On a more general level, there is the option of weight loss therapy, which in addition to improving insulin sensitivity, also may improve sleep and reduce severity of sleep problems such as OSA.[42]

With respect to circadian disturbances, the general consensus from the National Institute of Health is to avoid shift work in order to prevent its negative effects on health.[73] The field of interventions to restore circadian disturbance and thereby improve glycemic outcomes is limited. There are several options, including education and behavioral therapy, but none has been tested with regard to their endocrine or metabolic effects.[74] There is one interesting environmental example related to circadian disturbance that has been tested. Bright light therapy (BLT), which is known for its activating and synchronizing effects, was shown to reduce depressive symptoms as well as improve insulin sensitivity in people with T2D and depression.[75] However, much more work is needed to assess if and how treating circadian disturbances can prevent diabetes development and improve diabetes outcomes. Finally, technological developments in glucose management such as algorithm-driven partially automated insulin delivery are showing potential for improving not only glycemic outcomes but also sleep.[76]

Mechanisms

Evidence suggests that there is a bidirectional relationship of sleep and circadian rhythm disturbances with diabetes.[77,78] On the one hand, disturbances in sleep and circadian rhythm may contribute to disturbed glycemic outcomes via direct biological mechanisms (including decreased brain glucose utilization and overactivation of the HPA-axis, disturbed satiety hormones such as leptin and ghrelin)[78] and indirectly via suboptimal self-care.[79] Suboptimal sleep may negatively affect cognitive processes central to self-care, including accuracy, attention, decision-making, planning and problem solving.[80] In a study among adults with T1D, 12% and 33% reported that sleep disruptions affected bolus calculations and decision-making in diabetes care.[81] Among young people with T1D suboptimal sleep quality was associated with glucose monitoring difficulties, eg, distractibility and procrastination[82] as well as forgetfulness or "laziness" about diabetes self-care.[83] Suboptimal sleep may also lead to more unhealthy food choices, including more high fat/high sugar products, and to more sedentary behavior.[84] Conversely, increases in sleep of as little as 15–20 minutes have been associated with one additional insulin administration and fingerprick.[85] Suboptimal sleep may also hinder self-care through its negative impact on quality of life, across domains such as physical and mental health, family and school/work.[81,86] In both T1D and T2D, suboptimal sleep quality relates to higher daytime sleepiness, fatigue, diabetes-specific distress and symptoms of depression and anxiety.[40] Not surprisingly, this may create a vicious cycle between emotional distress, reduced self-care, increased glucose levels, and suboptimal sleep.[77]

On the other hand, diabetes may contribute to the development of disturbed sleep and circadian rhythm directly (nocturnal hypo- and hyperglycemia, nocturia) and indirectly through its management (eg, bodily-worn technological devices; alarms; difficulty falling asleep after correcting glucose excursions), complications (eg, neuropathic pain) and co-morbidities (eg, obesity).[11] A special mention should be made for depression, as there is large overlap between the constructs of sleep problems and depression. For example, part of the associations of sleep disturbance with diabetes development and suboptimal outcomes can be explained by depression, illustrated by a reduction in the strength of these associations when adjusting for depression status.[15] This is especially true for the associations regarding long sleep.

Discussion

This review illustrates the close link of sleep and circadian rhythm disturbances with diabetes. Suboptimal sleep is associated with an increased risk of developing T2D as well as with suboptimal diabetes outcomes for people with T1D and T2D as depicted in summary Figure 1. Preliminary data suggest that treatment of sleep and circadian rhythm disturbances in people with T1D and T2D could improve not only sleep but also diabetes outcomes. Finally, this association appears to be mediated by disturbances in hormones and changes in self-care behavior. In turn, diabetes and its management may also lead to disturbed sleep.

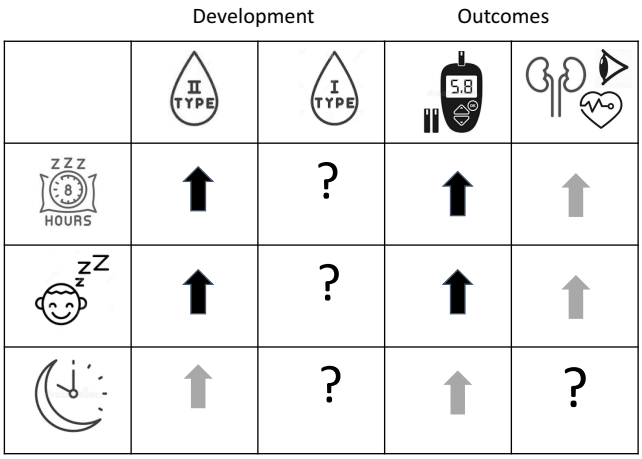

Figure 1 Summary of the literature to date on the association of sleep duration, sleep quality and sleep timing with the development as well as health outcomes (glycemic levels, complications) of type 1 and type 2 diabetes. ↑Increased risk or higher levels;?, no or insufficient data available. Bold black arrows, strong evidence based on large study sample or multiple studies; grey arrows, medium strength evidence; white arrows, evidence based on small sample or subgroup.

In general, improving sleep and circadian rhythm in people with diabetes could in turn improve glucose levels, thus providing an important aid in improving diabetes outcomes and ultimately improving quality of life.[87] Unfortunately, studies on the effect of sleep and circadian rhythm interventions specifically in people with diabetes are limited, either based on small studies or absent. Most first-line treatments of sleep problems seem effective in people with diabetes, comparable to the general population, but with additional positive effects on glycemic measures and other health outcomes, as discussed above. Of high clinical relevance are people with diabetes who partake in shift work, requiring specific guidance in terms of meal preparation and insulin schedules in order to achieve optimal glycemic outcomes.

There are also some points of discussion, for example with regard to the ways to measure sleep and circadian rhythm in practice. The literature shows variation in outcome, depending on the way it is measured: objectively using electroencephalograms or accelerometers versus subjectively using a diary or questionnaires.[88] For example, O'brien et al demonstrated this discrepancy: with actigraphy total sleeping time was approximately an hour less than self-reported sleeping time using diaries.[89] However, despite this discrepancy, we feel the direction of the associations, namely disturbance of sleep and circadian rhythm to be associated with increased risk of diabetes development as well as suboptimal diabetes outcomes, to still be the same. Finally, there are still quite some research gaps that we identified throughout the review process. For example, limited knowledge is present on the prevalence of circadian disturbances in people with diabetes, and more research is also needed to illustrate the glycemic effects of treatment of sleep or circadian disturbances in people with diabetes. But also observations from clinical practice, such as the experience of more nightmares when having low blood sugar during the night,[90] deserve further investigation.

With the advancement of measurement techniques, ecological momentary assessments (EMA) could aid the assessment of sleep, sleep disorders and circadian rhythm. EMA involves repeated sampling of participants´ behaviors and experiences in real-time and in the participants' natural environments, often using smartphone applications.[91] For example, one could ask regular questions on the status of a sleep problem and thus provide a more representative picture of the course of the condition by multiple assessments collected over a relatively short period. But also the use of activity trackers measuring sleep or even portable devices measuring sleep phases could provide enormous amounts of information on sleep and circadian rhythm in people with diabetes.[92] Using EMA allows for better evaluation of within-person changes, which in chronic conditions such as diabetes could provide valuable information for the people living with them, health-care providers and researchers.[93] A recent review of studies using EMA in people with diabetes to assess stress, anxiety, and depression showed that increases in those parameters predicted reductions in self-care behaviors[93], suggesting that, despite limited research (only 10 studies included), EMA has potential clinical utility for diabetes care to measure sleep and circadian rhythm. However, we should make sure that the use of such technologies does not widen health inequalities since these technologies might not be widely available to all who may need them.

In Practice

Given their high prevalence and adverse consequences, sleep and circadian rhythm disturbances require more attention in the prevention and treatment of diabetes. Since 2017, the Standards of Medical Care of the American Diabetes Association include a recommendation to regularly assess sleep given its association with glycemic outcomes.[94] These recommendations are yet to be widely implemented, while recent studies have further stressed the close link between suboptimal sleep and diabetes difficulties. This suggests that not only people with diabetes, but also their health professionals may benefit from more information on the reciprocal link between sleep and diabetes, including treatment options. By providing an overview of the present literature, this review contributes to increased awareness that sleep may be as central to physical and mental health as diet and physical activity.

Conclusions

We concluded that sleep and circadian rhythm disturbances are closely linked with diabetes. More attention to sleep in regular diabetes care is warranted, while further research is needed on treatment of sleep and circadian rhythm disturbances in the prevention of diabetes and its suboptimal outcomes.

Data Sharing Statement

This review did not generate any new data.

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Author Contributions

All authors made a significant contribution to the work reported, whether that was in conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agreed to be accountable for all aspects of the work.

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