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Waking Up to the Importance of Sleep in Type 2 Diabetes Management: A Narrative Review

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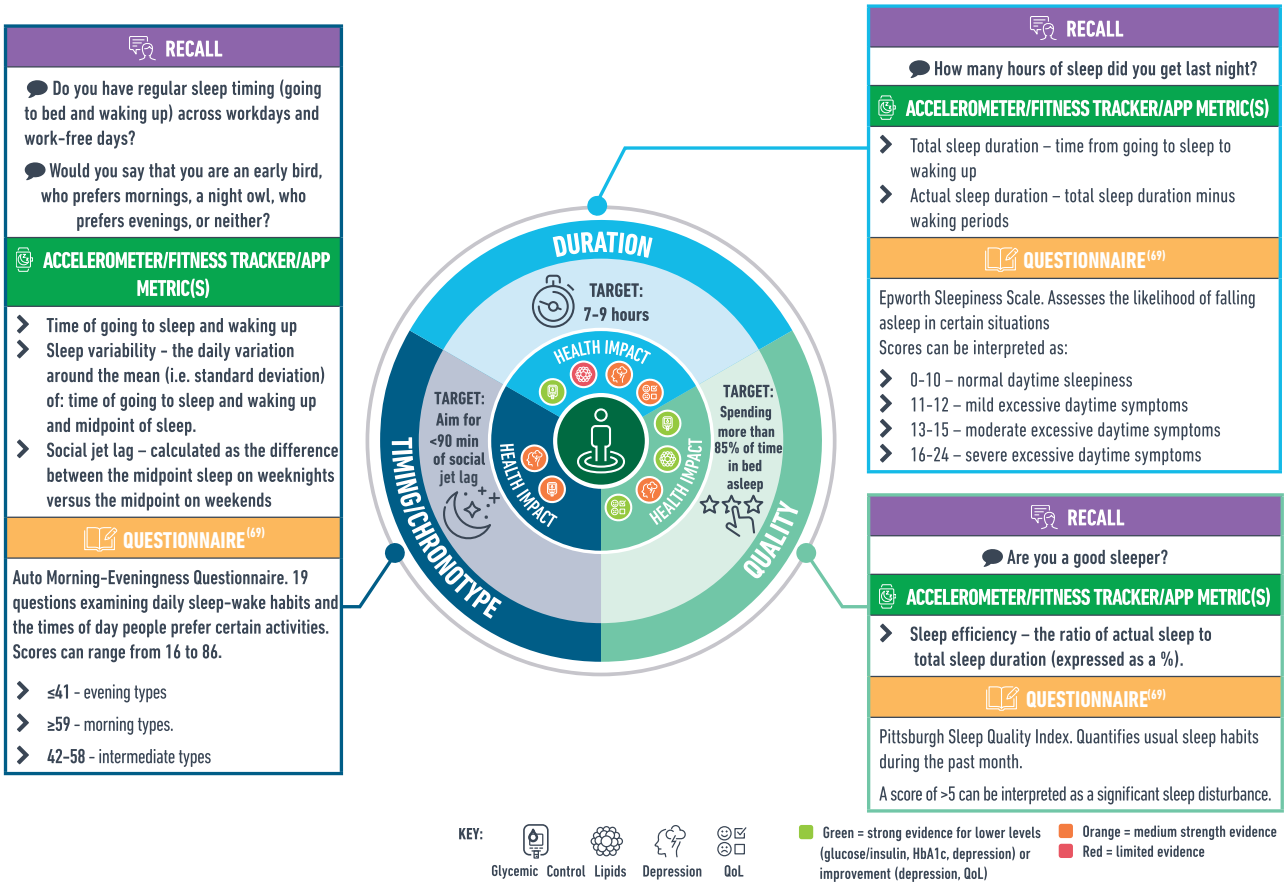

Auto, automated; QoL, quality of life.

ARTICLE HIGHLIGHTS

- **Why did we undertake this study?**
  Sleep quantity, quality, and timing are associated with markers of glycemia, cardiovascular disease risk, and mortality in those living with type 2 diabetes, with the strongest evidence seen for sleep quantity.
- **What is the specific question(s) we wanted to answer?**
  Despite the burgeoning observational data, the evidence base for improving sleep in those living with type 2 diabetes is limited, potentially restricting its applicability.
- **What did we find?**
  Sleep should always be discussed as part of a holistic approach to lifestyle behavior in diabetes care. To facilitate sleep self-management, health care professionals should consider using wearables/home-based assessments/self-reported measures of sleep.

Waking Up to the Importance of Sleep in Type 2 Diabetes Management: A Narrative Review

Joseph Henson,[1] Alix Covenant,[1] Andrew P. Hall,[2,3] Louisa Herring,[1,2] Alex V. Rowlands,[1,4] Thomas Yates,[1] and Melanie J. Davies[1]

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For the first time, the latest American Diabetes Association/European Association for the Study of Diabetes (ADA/EASD) consensus guidelines have incorporated a growing body of evidence linking health outcomes associated with type 2 diabetes to the movement behavior composition over the whole 24-h day. Of particular note, the importance of sleep as a key lifestyle component in the management of type 2 diabetes is promulgated and presented using three key constructs: quantity, quality, and timing (i.e., chronotype). In this narrative review we highlight some of the key evidence justifying the inclusion of sleep in the latest consensus guidelines by examining the associations of quantity, quality, and timing of sleep with measures of glycemia, cardiovascular disease risk, and mortality. We also consider potential mechanisms implicated in the association between sleep and type 2 diabetes and provide practical advice for health care professionals about initiating conversations pertaining to sleep in clinical care. In particular, we emphasize the importance of measuring sleep in a free-living environment and provide a summary of the different methodologies and targets. In summary, although the latest ADA/EASD consensus report highlights sleep as a central component in the management of type 2 diabetes, placing it, for the first time, on a level playing field with other lifestyle behaviors (e.g., physical activity and diet), the evidence base for improving sleep (beyond sleep disorders) in those living with type 2 diabetes is limited. This review should act as a timely reminder to incorporate sleep into clinical consultations, ongoing diabetes education, and future interventions.

Although lifestyle modifications are fundamental therapeutic components in the management of type 2 diabetes, national and international guidelines have predominantly focused on pharmaceutical therapies. The prominence and integration of lifestyle modifications into guidelines and resources for clinical decision-making have paled in comparison, epitomized by fewer citations and less defined clinical impact. Now, for the first time, the latest American Diabetes Association/European Association for the Study of Diabetes (ADA/EASD) consensus guidelines have broken with tradition by incorporating a growing body of evidence linking health outcomes associated with type 2 diabetes to the movement behavior composition over the whole 24-h day (1,2). In this context, a 24-h day comprises a sequence of movement behaviors distributed on a continuum ranging from limited/no movement to high-intensity activities. The five S's (sleep, sitting, stepping, sweating, and strengthening) encapsulate these physical behaviors, and their inclusion represents an important milestone in bridging the gap between current knowledge around 24-h behaviors and clinical care. Of note, the importance of sleep as a key lifestyle component in the management of type 2 diabetes is promulgated, which

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complements recent reviews (3–5), and is placed, for the first time, on a level playing field with the importance of physical activity. This is important key because despite sleep occupying approximately one-third of the day for most people and modulating a variety of metabolic, endocrine, and cardiovascular processes, it only appears in ∼40% of clinical practice guidelines (6). Given the fundamental role of sleep in the health and well-being of those living with type 2 diabetes, there is a need to increase its exposure and applicability for health care professionals, policymakers, and individuals with lived experience. As such, we highlight some of the key evidence underpinning the importance of sleep in the management of type 2 diabetes, outline practical advice about initiating conversations in clinical care, and propose future directions for sleep research in the management of type 2 diabetes. As a frame of reference for the reader, the common terminology and definitions used throughout this article can be found in Table 1.

A BRIEF OVERVIEW OF SLEEP AND CURRENT RECOMMENDATIONS

Sleep is a metabolically active and dynamic process that involves complex behavioral and physiological processes. Sleep has a typical underlying architecture underpinned by a rhythmic alternation between nonrapid eye movement (NREM) and rapid eye movement (REM) stages (7). In particular, the deeper stages of NREM sleep (also termed stage N3) are the most refreshing and restorative, allowing the body to repair cells, tissues, and muscles (8). Over the course of the night, total sleep is made up of several rounds of the sleep cycle, which is composed of four individual stages. A visual representation (hypnogram) and description of the various stages within a sleep cycle can be found in Fig. 1 and Table 2.

Despite healthy sleep consisting of adequate duration, quality, timing, and regularity, along with the absence of sleep disturbances or disorders, guidelines and recommendations have typically focused on duration. General recommendations suggest at least 7 h of sleep per night regularly for optimal health (9,10). Specific recommendations for those living with type 2 diabetes have not been developed and as such should defer to these global recommendations.

SLEEP CHARACTERISTICS HIGHLIGHTED IN THE ADA/EASD CONSENSUS REPORT

Historically, the research in type 2 diabetes has focused on sleep disorders and deficiencies, with the most prevalent reported in Table 1. Such disturbances can increase the risk of developing type 2 diabetes and its associated micro- and macrovascular complications (e.g., neuropathy, nephropathy, and retinopathy), alongside several health-related quality of life domains (4,11). However, a range of outcome measures (i.e., beyond sleep disorders) can be used to characterize "optimal sleep" (Table 1). Discussion of each characteristic is beyond the scope of this article; therefore, we primarily focus on the three overarching constructs outlined in the latest ADA/EASD consensus report—quantity, quality, and timing (i.e., chronotype) of sleep—as they represent important and underrecognized components of type 2 diabetes management (1,2). However, we also acknowledge that these sleep behaviors usually coexist and interact with each other in a compensatory manner. For ease of interpretation, we highlight each construct in turn, while outlining their role in the incidence of type 2 diabetes and effects on glycemic control, cardiovascular disease (CVD) risk, and mortality. These outcomes were chosen as they represent the most robust evidence to date (1,2). That said, we recognize that sleep also impacts on other markers of interest (e.g., depression), which is likely to become more prominent as the evidence base evolves.

An overall summary of the meta-analyses published within each construct can also be found in Table 3. However, due to the varying level of available evidence this narrative review presents data across the research hierarchy spectrum (i.e., from systematic reviews/meta-analysis to single, cohort studies).

QUANTITY OF SLEEP

Sleep Quantity and the Incidence of Type 2 Diabetes

There is now established evidence for a U-shaped association between sleep duration and type 2 diabetes incidence, with the nadir typically occurring at 7 h per day, with short (typically defined as <6 h) and long (typically defined as >9 h) sleep duration having up to a 50% increase in the risk of type 2 diabetes, including progression from prediabetes (12). Dose-response analysis has also demonstrated in comparison with 7 h/night, each hour decrease or increase in sleep is associated with a 9–14% increase in risk of type 2 diabetes (13–16)

Despite allowing significant advancement in our knowledge, studies to date have mostly used self-reported and single time point assessments of sleep. Use of objective measures of sleep duration or genetics-based analyses has continued to support an association for sleep as a risk factor for metabolic dysfunction but has produced equivocal evidence of an association for long sleep. In the UK Biobank cohort studies, for example, although use of objective measures of sleep duration demonstrated that sleeping >8 h/night is associated with increased CVD, cerebrovascular, and mood disorders, there was no evidence of a relationship with type 2 diabetes (17). A recent meta-analysis, alongside Mendelian randomization studies, also failed to support a higher risk of cardiometabolic dysfunction with longer sleep (12,18,19) with some evidence suggesting that longer sleep may even be protective (18). This suggests that deleterious associations with longer sleep may be explained by confounding or reverse causation, whereas associations with shorter sleep may be causal in nature and represent a target for intervention. Indeed, this is supported by emerging interventional research that has shown that sleep extension interventions in short sleepers result in improved insulin sensitivity and reduced daily energy intake (20,21).

Associations of Sleep Quantity, Glycemic Control, CVD Risk, and Mortality in Those With Type 2 Diabetes

Subjectively quantified sleep is associated (U-shaped) with HbA_{1c} and fasting plasma glucose in those with type 2 diabetes, with both long (>8 h/night) and short (<6 h/night) sleep durations adversely influencing glycemic control (16). Recent clinical evidence also extends beyond glycemic control in demonstrating that short sleep duration is also associated with CVD risk and mortality in those living with type 2 diabetes (20,21). For example, data from the UK Biobank cohort

Table 1—Definitions of common sleep disorders, terminology, and interventions in type 2 diabetes

Sleep disorders	
Insomnia	Regular difficulty initiating and maintaining sleep or waking up earlier than desired despite adequate opportunity to sleep
OSA	A sleep-related breathing disorder characterized by complaints such as nonrestorative sleep, sleepiness, snoring, or obstructive respiratory events
Restless leg syndrome	A neurological disorder characterized by uncomfortable sensations in the extremities and an overwhelming urge to move one's legs, especially in the evening and during periods of inactivity
Circadian rhythm disorders including (but not limited to) delayed or advanced sleep phase type, irregular sleep-wake type, jet lag type, shift work type	These disorders arise when the desired timing of sleep does not match the underlying circadian rhythm in sleep propensity (i.e., the timing of sleep is either earlier or later than desired, sleep timing is irregular from day-to-day, and/or sleep occurs at the wrong circadian time)
Sleep terminology	
Sleep quantity	The total amount of sleep per 24 h
Sleep quality	How well an individual is sleeping (often measured subjectively)
Timing	The placement of sleep within the 24-h day
Alertness/sleepiness	The ability to maintain attentive wakefulness
Catecholamines	Neurohormones responsible for the body's "fight-or-flight" response
Chronotype	The internal circadian rhythm or body clock of an individual that influences the cycle of sleep and activity over a 24-h period
Circadian misalignment	A range of processes including (but not limited to) inappropriately timed sleep and wake time, misalignment of sleep/wake with feeding rhythms, or misaligned central and peripheral rhythms
Cortisol	A hormone produced by the adrenal gland that plays an important role in the stress response
Nocturia	The need to wake and pass urine at night
NREM sleep	Four sleep stages in which there is an absence of REM
REM sleep	Presence of desynchronized brain wave activity and bursts of rapid eye movements
Sleep architecture	Cyclical sleep pattern involving different stages (e.g., REM and NREM sleep)
Sleep continuity	The amount and distribution of sleep vs. wakefulness in a given sleep period
Sleep debt	The cumulative effect of not getting enough sleep
Sleep efficiency	The time asleep as a percentage of the time in bed (with the intention to sleep)
Sleep latency	Time taken to fall asleep
Sleep variability	The daily variation around the mean for sleep parameters. Often measured over multiple days
Social jet lag	The discrepancy between biological time and social time, which often culminates from two separate, distinct sleeping patterns. This disparity usually occurs between separate weekday and weekend routines
WASO	Periods of wakefulness occurring after sleep onset
Zeitgebers	Environmental variables that can act as circadian time cues
Interventions to change sleep behavior	
CBTi	A psychosocial intervention approach to confront and modify the irrational thoughts and beliefs that are most likely at the root of maladaptive behavior (i.e., poor sleep quality). Includes elements of sleep hygiene, education, and stimulus control
Sleep education	A program that may include information on sleep health, sleep cycles, or consequences of insufficient sleep or sleep hygiene tips. Often delivered using a variety of methods (e.g., group-based education, webinars, apps)
Melatonin	A hormone that is produced by the pineal gland in the brain that regulates the body's sleep-wake cycle
OSA-specific interventions	
CPAP	A continuous pressure of air that is delivered into the airway during sleep
Mandibular advancement devices	An oral device that holds the mandible and tongue forward, away from the back of the throat, thus holding the upper airway open
Positional therapy	Techniques/devices that prevent individuals from lying in a supine position and promote side sleeping
Hypoglossal nerve stimulation	An implanted medical device used to target moderate and severe OSA by electrically stimulating the hypoglossal nerve, which is responsible for tongue movement
Upper airway surgery	Targets upper airway expansion and/or stabilization and/or removal of the obstructive tissue

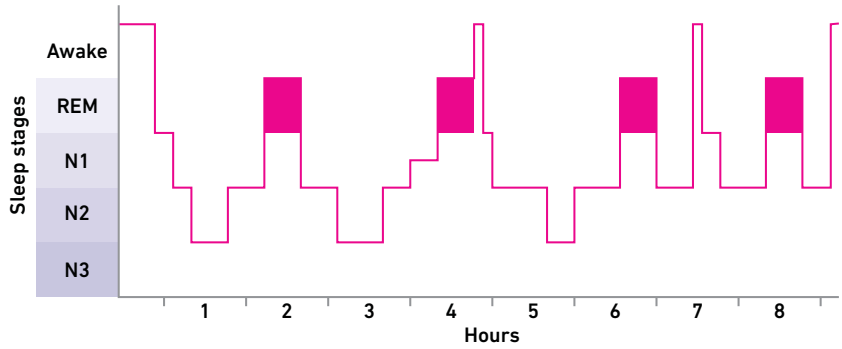

Figure 1—A visual representation (hypnogram) and description of the various stages within a sleep cycle.

demonstrated that short (≤5 h/night) sleep duration was associated with a 42–70% higher risk of ischemic stroke and CVD mortality in comparisons with 7 h/night (22). These results mirror the J-shaped association previously observed between all-cause and CVD mortality and sleep duration, where ≤4 h sleep was associated with a 41% increased risk of all-cause mortality and 54% increased risk of CVD mortality vs. 7 h sleep (21).

QUALITY OF SLEEP

Sleep Quality and the Incidence of Type 2 Diabetes

Sleep quality has recently been defined as "an individual's self-satisfaction with all aspects of the sleep experience" (23). This is underpinned by four attributes: sleep efficiency, sleep latency, sleep duration, and wakefulness after sleep onset (WASO) (Table 1). Many factors (e.g., environmental, behavioral, psychological, and physiological) can contribute to poor or insufficient sleep quality that impact health outcomes, including those associated with type 2 diabetes.

A recent analysis using seven cycles of National Health and Nutrition Examination Survey (NHANES) data (n = 16,517) demonstrated that sleep quality has declined from 2005–2006 to 2017–2018 and that the highest prevalence of diabetes was consistently observed in the low sleep quality group (24). These findings corroborate previous meta-analysis, where poor sleep quality was associated with a 40–84% increased risk of developing type 2 diabetes (14,25).

Associations of Sleep Quality, Glycemic Control, CVD Risk, and Mortality in Those With Type 2 Diabetes

Epidemiological studies have suggested that there are associations between sleep disturbances and glycemic control in those living with type 2 diabetes. For example, Knutson et al. (26) demonstrated that sleep quality was a significant predictor of HbA_{1c} in those individuals with at least one diabetes-related complication. Indeed, the predicted increase in HbA_{1c} level for a 5-point increase in Pittsburgh Sleep Quality Index (PSQI) score in those with complications and taking insulin was 1.9% (0.7 mmol/mol) (i.e., moving from 8.7 to 10.6% [71.6 to 92.4 mmol/mol]). As the change is proportional, the increase in PSQI score may have a greater effect at a higher HbA_{1c}. A 2017 meta-analysis that included a small number of studies (n = 5) also showed that in those with type 2 diabetes who had difficulty in initiating or maintaining sleep, poorer sleep quality was associated with higher HbA_{1c} levels (16).

There is limited evidence on the association between sleep disturbance and risk of incident CVD in those living with type 2 diabetes. However, data from a large, single cohort study (n = 36,058) demonstrated that disturbances in sleep were associated with increased risk for all CVD (hazard ratio [HR] 1.24 [95% CI 1.06–1.46]) and coronary heart disease (1.24 [1.00–1.53]) events in those living with newly diagnosed type 2 diabetes (<6 months) (27).

Studies examining sleep disturbances in relation to mortality have shown significantly increased risk (27,28). More specifically, in a recent study with use of prospective data from UK Biobank (n = 487,728) investigators examined associations of frequent sleep disturbances, diabetes, and risk of all-cause mortality (28). In examination of sleep

Table 2—Stages of sleep

Stage	Physiological processes	Duration
NREM stage N1: falling asleep	Heart rate and breathing slow down, muscles begin to relax, light changes in brain activity	A few minutes
NREM stage N2: light sleep	Heart rate and breathing slow down even further, brain waves show a new pattern and eye movement stops, body temperature drops	10–25 min during the first sleep cycle. Collectively, ∼50% of total sleep time is spent in this stage
NREM stage N3: slow wave sleep	Deepest sleep state. No eye movements; muscle tone, heart rate, and breathing rate decrease as the body relaxes even further; tissue repair, growth, and cell regeneration	20–40 min. As sleep continues, these stages get shorter
REM	Primary dreaming stage. Eye movements become rapid, brain activity is markedly increased, body experiences temporary paralysis of the muscles. Essential to cognitive functions (e.g., memory)	While the first REM stage may last only a few minutes, later stages can last for ∼60 min. REM stages make up ∼25% of total sleep

Table 3—Overview of meta-analyses examining associations of sleep duration, quality, and timing with incidence of diabetes and glycemic control

Authors	Year of publication	Number of participants (and studies)	Type of sleep variable	Measurement of sleep	Outcome measure(s) of interest	Main results
Shan et al.	2015	482,502 (10)	Duration	Self-reported	Incidence of diabetes	In comparisons with 7 h/day, each hour decrease in sleep was associated with a 9% increased risk of diabetes, vs. 14% for every hour increase
Cappuccio et al.	2010	107,756 (10)	Duration and quality	Self-reported	Incidence of diabetes	Short (≤5–6 h/night) and long (>8–9 h/night) sleep durations increase the relative risk of developing type 2 diabetes (RR 1.28 [95% CI 1.03–1.60] and 1.48 [1.13–1.96], respectively). Difficulty in initiating or mainlining sleep also increased the relative risk of developing type 2 diabetes (1.57 [1.25–1.97] and 1.84 [1.39–2.43]).
Lu et al.	2021	737,002 (17)	Duration	Self-reported	Incidence of diabetes	Short (<6 h/night) and long (≥9 h/night) sleep increased the risk of type 2 diabetes in comparison with normal sleep duration (RR 1.22 [95% CI 1.15–1.29] and 1.26 [1.15–1.39])
Anothaisintawee et al.	2016	1,061,555 (36)	Duration and quality	Self-reported	Incidence of diabetes in those with sleep disorders	The pooled RR was 1.48 (95% CI 1.25–1.76) for ≤5 h/night and 1.36 (1.12–1.65) for ≥9 h/night. Poor sleep quality, OSA, and shift work were associated with diabetes, with a pooled RR of 1.40 (1.21–1.63), 2.02 (1.57–2.61), and 1.40 (1.18–1.66).
Lee et al.	2017	29,649 (7)	Duration	Either a single survey item or extracted from PSQI	Glycemic control (HbA_{1c} and fasting glucose)	Short (<6 h/night) and long (>8 h/night) sleep duration were associated with increased HbA_{1c} (WMD 0.23 and 0.13%) and higher fasting plasma glucose (WMD 0.22 and 0.44 mmol/L) vs. normal sleep duration (6–8 h/night).
Mostafa et al.	2022	20,139 (3)	Duration	Self-reported	Progression of diabetes from prediabetes	Short sleep duration was associated with a greater risk of progressing from prediabetes to type 2 diabetes (HR 1.59 [95% CI 1.29–1.97])
Lee et al.	2017	1,808 (5)	Quality	PSQI	Glycemic control (HbA_{1c})	Poorer sleep quality was associated with higher HbA_{1c} levels (WMD 0.35% [95% CI 0.12–0.58])
Gan et al.	2015	226,652 (12)	Timing	Self-reported	Incidence of diabetes	Individuals exposed to shift work have a 9% increased risk of type 2 diabetes compared with those with no shift work experience
Gao et al.	2020	Not specifically reported (21)	Timing	Self-reported	Incidence of diabetes	A 10% increased risk of type 2 diabetes was demonstrated for shift work (mostly nights) in comparison with daytime working
Bouman et al.	2023	Sample sizes ranged from 33 to 58,370 (68)	Timing	Subjective and objective (accelerometers)	HbA_{1c}	Social jet lag was associated with higher HbA_{1c} (0.42% [95% CI 0.12–0.72]); however, the results are limited by high heterogeneity (I^2 = 100%).

WMD, weighted mean difference; RR, relative risk.

disturbances (28% prevalence) and diabetes, the presence of both was associated with increased risk of all-cause mortality (HR 1.87 [95% CI 1.75–2.01]) in comparisons with subjects who had either or neither condition (28).

CHRONOTYPE AND TIMING

The subset of sleep management concerning an individual's chronotype, or what most people understand as being an early bird or a night owl, can influence sleep timing and consistency. In humans, the circadian clock is divided into two distinct parts, the master clock in the suprachiasmatic nucleus of the hypothalamus and peripheral clocks, situated in the peripheral tissues (29). From a biological perspective, sleep timing depends on two processes: sleep debt (i.e., the difference between the amount of sleep we need and the amount we get) and an internal circadian clock that synchronizes biological sleep/wake rhythms to our 24-h day, aided by zeitgebers ("time givers") and neurohormonal pathways (including melatonin). However, many facets of modern life, such as work schedules (i.e., night/rotating shifts), can lead to sleep/wake schedules that are misaligned relative to our internal biological clock. Our bodies appear to have evolved to cope with day-to-day variability in sleep timing within certain thresholds, beyond which unfavorable responses occur (Fig. 2). This misalignment (termed social jet lag) occurs when different endogenous circadian rhythms are not synchronized with one another and/or with external cues or social pressures, such as work pattern (30).

Chronotype, Timing, and the Incidence of Type 2 Diabetes

Chronotype preference has been linked with many chronic diseases, including type 2 diabetes (31–34). For example, for those with a preference for evenings (i.e., going to bed late and getting up late) there was a 2.5-fold higher odds ratio for type 2 diabetes as compared with morning types (i.e., going to bed early and getting up early), independent of sleep duration and sleep sufficiency (34). Moreover, investigators of a recent cohort analysis showed that after accounting for multiple lifestyle and sociodemographic factors, middle-aged nurses with an evening chronotype demonstrated a 19% increased diabetes risk compared with morning chronotypes (35).

Shift Work

There is also a growing body of evidence suggesting a chronotype-dependent association between work hours (i.e., shift work) and metabolic disease (36–38). For instance, findings of a meta-analysis of observational studies indicate that individuals exposed to shift work have up to a 10% increased risk of type 2 diabetes compared with those with no shift work experience (39,40). Similarly, in those with established type 2 diabetes who work night shifts, glycemic control is more likely to be impaired compared with people with type 2 diabetes performing day work (41). Findings of a 2015 article also showed that women who were late chronotypes without any history of rotating night shift work had a 1.5-fold increased risk of type 2 diabetes (odds ratio 1.51 [95% CI 1.13–2.02]) (42).

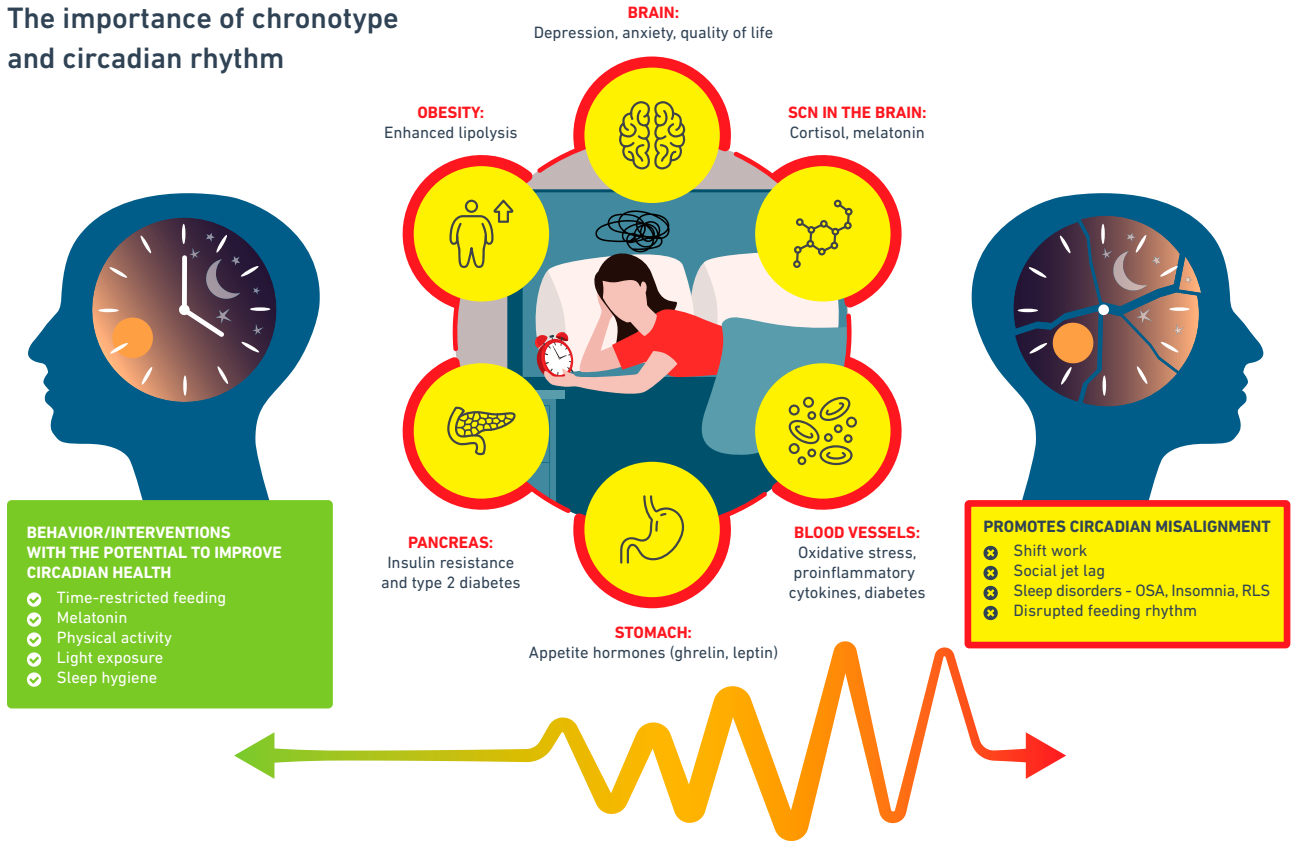

Figure 2—Impact of circadian misalignment on health outcomes in type 2 diabetes. RLS, restless leg syndrome; SCN, suprachiasmatic nucleus.

Interestingly, the investigators observed an interaction between chronotype and shift work, where late chronotypes had a significant increase in type 2 diabetes risk only when their shift schedule did not involve night work. Conversely, although early chronotypes had a lower risk of type 2 diabetes, this increased with the length of rotating night shift work, possibly driven by more circadian misalignment during night shifts (42). Therefore, these results suggest that if work times interfere with sleep timing, shift and day workers may be at an increased risk for type 2 diabetes.

Associations of Chronotype and Sleep Timing With Glycemic Control, CVD risk, and Mortality in Those With Type 2 Diabetes

Although, limited by high heterogeneity between studies, a recent systematic review and meta-analysis demonstrated that social jet lag (vs. no social jet lag) is associated with higher HbA_{1c} levels in those living with type 2 diabetes (0.42% mean difference [95% CI 0.12–0.72]) (43). When chronotype, social jet lag, and glycemic control are considered together, there also appears to be a significant relationship between later chronotype and HbA_{1c} levels, but only for patients with $>$90 min of social jet lag (44). Other sleep disturbances (insufficient sleep, poor sleep quality, and sleep apnea) influencing glycemic control may also induce a degree of social jet lag. For instance, objectively measured variability in sleep duration, which may reflect partial sleep deprivation alternating with sleep compensation, was most strongly associated with HbA_{1c} in 172 individuals with type 2 diabetes (when compared with total sleep duration, subjective sleep quality, and sleep efficiency) (45). The difference in average HbA_{1c} between participants in the lowest and highest quartile of variability in sleep duration was 1.0% (45).

Findings of a prospective cohort analysis including those living people with type 2 diabetes (n = 3,147) demonstrated that disrupted circadian-activity rest rhythms (defined with use of accelerometer-derived average activity during wake and sleep) were associated with higher risks of CVD (HR 1.38 [95% CI 1.03–1.84]), ischemic heart disease (2.49 [1.71–3.64]), and CVD-related mortality (3.98 [1.76–9.00]). Similar associations were also observed for all-cause mortality (1.75 [1.14–2.71]) (46). Although not confined to those with type 2 diabetes, definite evening types have been shown to have a significantly increased risk of all-cause mortality (HR 1.10 [1.02–1.18]) compared with definite morning types (47). This analysis, conducted in 433,268 UK Biobank individuals over a 6.5-year period, also demonstrated that the effect size across different chronotypes was similar to the effect observed for BMI, renal, musculoskeletal, and gastrointestinal/abdominal disorders (47).

POTENTIAL MECHANISMS LINKING SLEEP ARCHITECTURE AND TYPE 2 DIABETES

The bidirectional link between sleep complaints and type 2 diabetes likely occurs via multiple physiological and behavioral mechanisms (Fig. 2). Indeed, sleep restriction is associated with several hormonal changes that are known to impact insulin resistance and insulin secretion. For example, melatonin and cortisol, which are produced from the hypothalamic-pituitary-adrenal axis modulate the sleep-wake cycle and display an inverse relationship (48). However, these patterns change with short sleep duration (both habitual and experimental) as sleep restriction causes elevated markers of sympathetic activation and catecholamines (49). As a result, cortisol levels become higher in the evening (50) and display a lower rate of decline (51). Such changes can lead to a lower response of β-cells to glucose and reduce insulin sensitivity (driven by lower glucagon-like peptide 1 levels) (52). Short sleep duration and sleep deprivation are also associated with elevated levels of proinflammatory cytokines, changes in adipokines secreted from adipose tissue (53,54), enhanced lipolysis (55), and increased hunger and appetite, largely driven by changes in leptin (decrease) and ghrelin (increase) (56).

Circadian misalignment also plays an important role in the etiology of type 2 diabetes. The circadian rhythm is driven by circadian clock genes, controlling physiological and behavior processes over a 24-h period (57). Indeed, polymorphisms in many of the well-established core clock genes (e.g., *CLOCK*,*BMAL1*, *CRYO*) have been shown to increase the risk of type 2 diabetes (58), with gene-behavior interaction studies also demonstrating interactions between diet and clock gene mutations that affect fasting glucose (59), insulin resistance (60), and type 2 diabetes (61).

Endogenous rhythms are produced by the suprachiasmatic nucleus, alongside cells in peripheral organs (e.g., skeletal muscle) that also have an intrinsic circadian clock (29) (Fig. 2). These anticipatory rhythms are synchronized to light cycles, but as we stay awake for longer and are exposed to more artificial light, we subject our body to various behaviors that may exacerbate cardiometabolic abnormalities. For example, we recently showed that evening chronotypes (i.e., preferring to go to bed late and getting up late) engage in lower levels of moderate-to-vigorous physical activity levels (approximately −10 min/day, −56%) compared with morning chronotypes (i.e., preferring to go to bed early and getting up early) (62). Therefore, an advancement of the internal circadian rhythm through informed timing of physical activity and time-restricted eating may be a useful adjunct therapeutic strategy (alongside sleep interventions) to foster metabolic improvements (63,64) and chronobiological homeostasis and better align internal rhythms with the environment and standard social schedules.

Although the aforementioned mechanisms may increase the risk and impair the management of type 2 diabetes, associated comorbidities also demonstrate a bidirectional relationship with sleep. For instance, sleep complaints often present prior to the onset of a new or recurrent episode of depression (65), suggesting that sleep may be involved in its pathogenesis. Conversely, symptoms of depression have also been shown to be an important correlate of suboptimal sleep quality, with as many as 90% of those with depression also having issues with sleep quality (66), making differentiating cause-and-effect relationships problematic.

MEASUREMENT OF SLEEP

There is no single measure of sleep, as the construct spans multiple dimensions and levels of analysis. As a result, sleep can be quantified through a variety of subjective, objective/physiological, and behavioral observation methodologies. A visual summary is provided in Fig. 3. For example, questionnaires can capture chronotype, quantity, latency to sleep onset, duration, and level of daytime

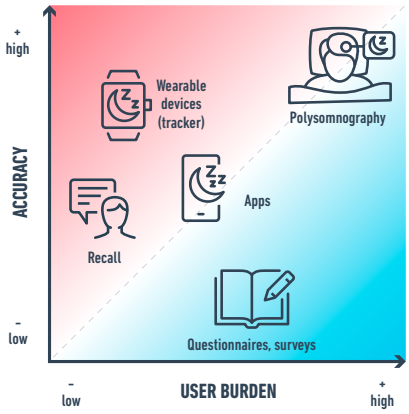

Figure 3—Accuracy and user burden of sleep measurement methodologies.

sleepiness. Examples include the Munich Chronotype Questionnaire, Morning-Eveningness Questionnaire, PSQI, and Epworth Sleepiness Scale (ESS) (67–70). Such subjective reports of sleep have informed most of the evidence base to date and are important in a clinical setting, as they can help determine whether further screening and/or treatment for a sleep complaint might be justified.

Polysomnography (PSG), which is the current gold standard for the objective measurement of sleep, provides insights into nocturnal physiology, including a recording of objective sleep architecture and measures of cardiopulmonary function (71). Actigraphy also allows physical behaviors (including sleep duration, sleep timing, and WASO) to be measured over a 24-h period (72). Indeed, the evolution of wearable technology provides individuals with an array of readily available self-assessed outcomes (e.g., duration, sleep schedules, sleep stages). A major strength is that they allow for recordings over a number of nights and in ecologic conditions. However, there are clinical implications, as although all wearables harness similar types of technology, variation in accuracy across devices and metrics exists. That said, if an individual is asleep, most wearables are 90–95% accurate for identifying this behavior (73,74). However, in determining sleep onset latency and WASO, the wearables only perform at a medium level of accuracy (∼60%), which diminishes further in trying to determine specific stages of sleep (∼50%) (74).

Other wearable and nearable examples include smart mattresses, pulse oximeters, and radar-based devices. Of particular note, electroencephalograms (EEG) are a widely used noninvasive method for monitoring neuronal action within the brain during sleep. Although usability and reliability issues with the use of EEG exist, recent advances in wearable devices mark an important step forward. Specific commercially available examples include the Dreem, Muse, and BrainBit headbands, alongside in-ear EEG devices (75). Although these devices are still to be validated at scale, they offer promising alternatives to PSG for long-term monitoring of sleep stages.

To reduce misclassification and capture sleep that is representative, individuals should be encouraged to obtain data over multiple nights (including both weekdays and weekends). Such data, although not a substitute for medical evaluations, may be used as an adjunct to an appropriate clinical evaluation or to facilitate behavior change.

INTERVENTIONS TO CHANGE SLEEP BEHAVIOR IN TYPE 2 DIABETES

Several behavioral and pharmacological sleep interventions exist; however, the majority of the evidence in type 2 diabetes focuses on the treatment of sleep disorders (in particular obstructive sleep apnea [OSA]) as opposed to diabetes-related outcomes (e.g., glycemia) per se. Discussion of them all in turn is beyond the scope of this review; therefore, we highlight some of the more prominent interventions that have been used to aid sleep management in those with type 2 diabetes. Definitions can also be found in Table 1.

Cognitive Behavioral Therapy and Cognitive Behavioral Therapy for Insomnia

Understandably, cognitive behavioral therapy (CBT) and cognitive behavioral therapy for insomnia (CBTi) are often confused. CBT is most commonly used to treat the symptoms of anxiety and depression, while CBTi is specifically designed for insomnia (76). As such, CBTi is recommended as first-line treatment for both short- and long-term insomnia (77) and includes elements of sleep hygiene, education, and stimulus control. Results of meta-analyses have shown CBTi to produce clinically meaningful improvements in sleep parameters, including sleep latency, sleep efficiency, total sleep time, WASO, and the number of awakenings (78,79). However, to date, in the majority of studies in those with type 2 diabetes investigators have used CBT, not CBTi, per se. Indeed, results of a 2021 randomized controlled trial, undertaken in 1,033 individuals living with type 2 diabetes, demonstrated improvements in glycemic control and sleep quality following lectures/discussions with trained general practitioners. Both HbA_{1c} (−0.17% at 6 months, −0.43% at 12 months) and sleep quality (−0.50 and −0.90 lower PSQI score at 6 and 12 months, respectively) were improved (80). This study, among others, was encapsulated in a 2022 meta-analysis (32 studies, n = 7,006), which included only one study specifically looking at CBTi. Nevertheless, the ability of CBT to reduce HbA_{1c} and fasting glucose was pooled at −0.14% and 0.9 mmol/L, respectively (81). However, the results of this meta-analysis and others should be interpreted with caution as individuals with type 1 diabetes and gestational diabetes mellitus were also included (82,83).

Sleep Education

Sleep education can play a fundamental role in ensuring individuals understand the relationship between sleep and overall well-being. In particular, it may include information on sleep health, sleep cycles, or consequences of insufficient sleep or sleep hygiene tips and may be delivered using a variety of methods (e.g., group-based education, webinars, apps). However, there is a paucity of evidence, particularly relating to glycemic outcomes in those living with type 2 diabetes. In a randomized pilot study in 31 adults with type 2 diabetes who did not sleep before midnight, a combination of conventional diabetes education and sleep education reduced HbA_{1c}, fasting glucose, and insulin resistance more than diabetes education alone (84). The potential utility of sleep hygiene as an intervention to improve sleep quality and glycemic control in prediabetes and diabetes has also been demonstrated (85). The intervention, which included sleep tips outlined by the American Academy of Sleep Medicine, resulted in improvements in sleep quality, time, and efficiency (10). For instance, PSQI score was 3.6 points lower in comparison with the control group. Moreover, the intervention resulted in

reductions in HbA_{1c} of 0.39% and 0.66% at 3 and 6 months, respectively (85).

Continuous Positive Airway Pressure and Alternative Treatments in OSA

Although continuous positive airway pressure (CPAP) is the current gold standard treatment for OSA (86), the impact on markers of glycemic control are negligible. For example, a systematic review and meta-analysis (6 studies, n = 581) showed no benefit for glycemic control (HbA_{1c} and fasting glucose) versus placebo (87). That said, CPAP should always be offered to those living with type 2 diabetes who present with OSA (regardless of the impact on diabetes-specific outcomes, given the symptomatic benefits, along with improvements in quality of life) (88).

A number of alternative therapies to CPAP also exist, including (but not limited to) lifestyle modification (e.g., weight loss), mandibular advancement devices, positional therapy, surgical procedures (e.g., upper airway), and hypoglossal nerve stimulation (see Table 1 for definitions). With such therapies improvements have been shown in apnea-hypopnea index, polysomnographic-based outcomes, daily function depressive symptoms, and quality of life, along with reduced arousals and rate of oxygen desaturation (89–92). Further research will determine whether these non-CPAP therapies are viable treatment alternatives for diabetes-specific outcomes.

Melatonin

The body naturally produces melatonin, but recently there has been interest in external sources (e.g., supplements) to aid sleep management. In those living with type 2 diabetes and insomnia, short-term use of prolonged-release melatonin has been shown to improve sleep maintenance and sleep quality (93). However, the direct impact of melatonin on glycemic parameters appears ambivalent, with a systematic review and meta-analysis showing potential benefits for fasting glucose and insulin sensitivity but not HbA_{1c} (94). That said, the potential of melatonin to influence proinflammatory pathways as well as oxidative stress state in those with diabetes (95) means that more clinical trials are needed that use combinations of melatonin with current therapeutic agents for the treatment of diabetes.

PRACTICAL ADVICE FOR CLINICIANS AND HEALTH CARE PROVIDERS

Despite health care professionals routinely asking about important indicators of health (e.g., weight, diet, and medication status), questions about sleep are often overlooked. Sleep should always be discussed as part of a holistic approach to lifestyle behavior in diabetes care. To aid discussions in clinical care, we provide a visual summary of the different methodologies, targets, and health impacts pertaining to the three sleep constructs: quantity, quality, and timing (i.e., chronotype) (Fig. 4).

Clinicians and health care professionals are also encouraged to follow the five S's for the management of sleep in clinical practice (Survey, Support, Shared decision

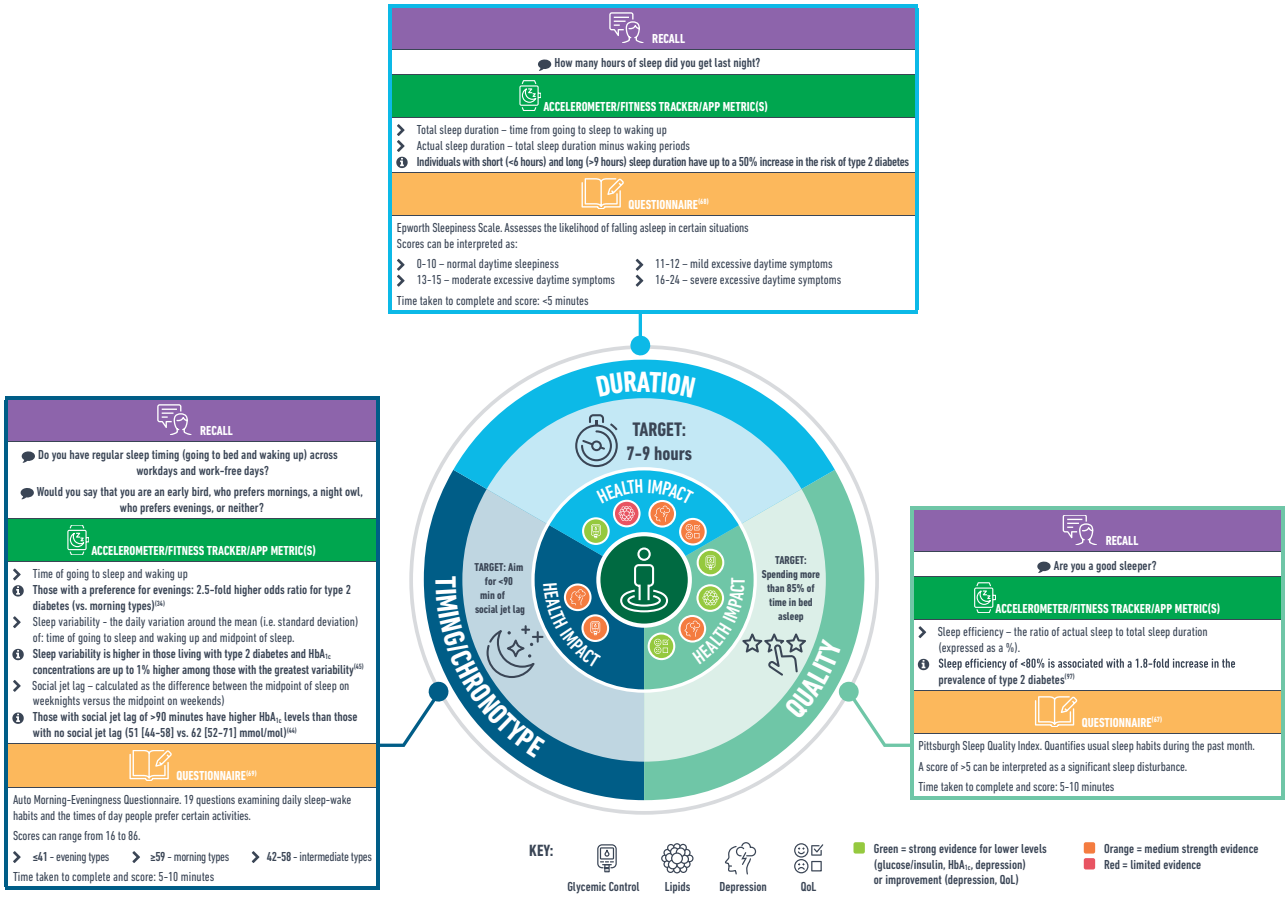

Figure 4—Methodologies, targets, and health impacts of sleep duration, quality, and timing. Auto, automated; QoL, quality of life.

making, Solutions, Signpost [Fig. 5]). Information gathered during the survey stage should inform the decision-making approach. If there is an indication that there may be an underlying sleep disorder, the patient could be fast-tracked straight to the signpost stage. If the main contributing factors to inadequate sleep health appear to be related to behavioral or lifestyle choices, they could be addressed using the processes outlined in the support, shared decision-making, and solutions stages. This may involve targeted recommendations/interventions that should be tailored to what is available in the local setting. The consultations may also need to be extended to multiple disciplines and specialties (e.g., sleep specialists, psychologists, diabetes care and education specialists).

Specific sleep behavior goals should be agreed on between the person living with type 2 diabetes and the clinical care team; shared decision-making is fundamental. SMART (specific, measurable, achievable, realistic, time-bound) goals can be used to facilitate conversations and action plans, where even small changes in sleep behavior should be acknowledged (e.g., turning off the television 15 min earlier). Realistic expectations should also be guided by self-efficacy and illness perceptions. Considering aspects of sleep, well-being, and their association with diabetes (e.g., diabetes-related distress) could support health care professionals in formulating diabetes management plans that are better tailored to the needs of the individual.

Sleep hygiene improvements can often aid the quantity and quality of sleep. It is important to emphasize that these recommendations are fundamental for the maintenance of healthy sleep, not solely as a treatment for sleep complaints. To facilitate behavior change, it is imperative that the importance of self-monitoring sleep behavior is emphasized, whether this be through objective or subjective methodologies (specific examples are highlighted in Fig. 3). Regular reviews of the data (by both the clinician and the person living with diabetes) are also pivotal to reinforce sleep behavior goals.

SUMMARY AND FUTURE DIRECTIONS

As we shift toward device-based measures of physical behaviors, future research should continue to integrate and assess the impact of sleep on glycemic control and overall health in type 2 diabetes. Given that interventional work is still in its infancy, there remain many unanswered questions the answers to which have the potential to reiterate and elevate the importance of sleep in diabetes clinical care. For example, how do lifestyle (e.g., weight loss, exercise [including timing]), and behavioral interventions affect sleep outcomes, physiological functioning, and well-being? Although weight loss results in a significant and clinically relevant improvement in OSA in a dose-response manner (96), the impact of newer classes of diabetes drugs on sleep-associated outcomes (e.g., OSA) is unknown. This is an important area for future research, particularly given their influence on body weight (especially compared with lifestyle interventions alone).

Health care professional training is pivotal, supported by established referral

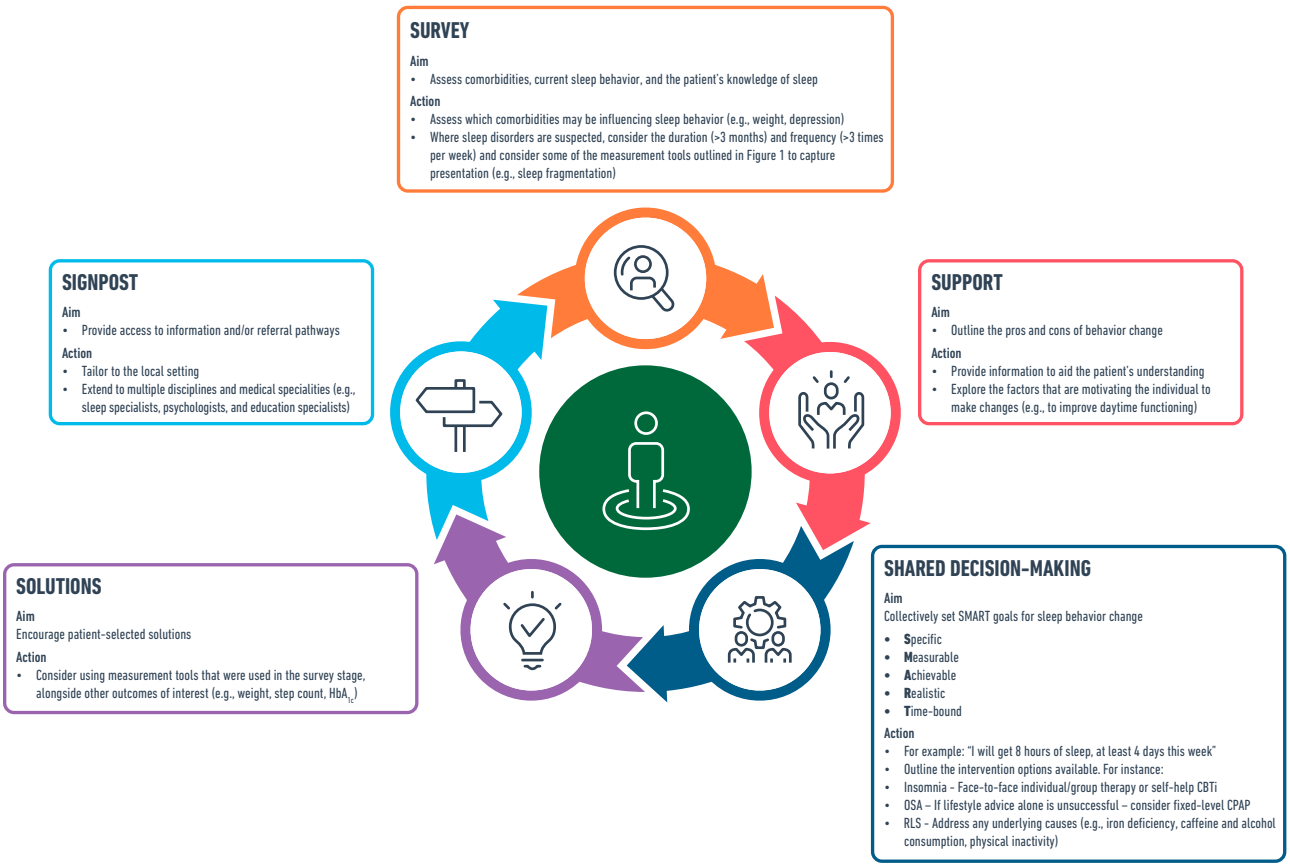

Figure 5—The five S's for the management of sleep in clinical practice. RLS, restless leg syndrome.

pathways into sleep (and other movement-based) therapies. Such training should also include appropriate behavior-change techniques and strategies to initiate and maintain healthy sleep, alongside an active lifestyle. Data management systems, coupled with self-monitoring tools and mobile health apps, are also important to capture, inform, and tailor advice around sleep. Qualitative methodologies may also shed more light on barriers to and facilitators of obtaining sufficient sleep. Ultimately, shared decision-making should contribute to the patient-centered holistic approach to diabetes management, but the recognition of the importance of sleep presents multiple interventional opportunities to induce glycemic and overall health benefits.

In summary, the latest ADA/EASD consensus report highlights sleep as a central component in the management of type 2 diabetes, placing it on a par with more traditional lifestyle factors, such as diet and exercise. Indeed, there is a plethora of observational data showing the associations between sleep and health outcomes. In contrast, although improvement in sleep characteristics (e.g., sleep extension) appears achievable in those with sleep disturbances, the impact that this has on type 2 diabetes and its associated outcomes is largely unknown. Therefore, this review should act as a timely reminder to incorporate sleep into clinical consultations and ongoing diabetes education.

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