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Evaluating the Role of Sleep Duration in Managing Glycemic Control in Type 2 Diabetes: A Systematic Review and Meta-Analysis

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is one of the most common metabolic disorders that produces a persistent hyperglycemic state due to insulin resistance and β-cell dysfunction. The lifestyle, especially sleep duration, has also become a possible glycemic control modulator. Both insufficient and excessive sleep time have been associated with poor glucose metabolism although the correlation is irregular among the studies. This association is therefore vital in coming up with sound non-pharmacological interventions to enhance glycemic control in T2DM patients. **Objectives:** This is a systematic review and meta-analysis we conducted to assess the relationship between sleep duration and glycemic control as indicated by glycated hemoglobin (HbA1c) levels in adults with T2DM. **Methodology:** An electronic search of PubMed, Scopus, Web of Science, and Google Scholar was conducted using a combination of predetermined keywords with the term sleep duration, glycemic control, and type 2 diabetes. Inclusion criteria: Original observational or intervenient studies evaluating connection between sleep duration and glycated hemoglobin (HbA1c) or fasting blood glucose (FBG) in adults with diabetes type 2. Titles, abstracts, and full-text screening, data extraction, and measurement of the quality of the studies in the NewcastleOttawa Scale were independently conducted by two reviewers. A random-effects model was used to compute pooled mean differences in HbA1c between short (<6 h) or long (>9 h) sleepers and normal (6-8 hours) sleepers by meta-analysis. I 2 statistics were used to measure heterogeneity and Egger test was used to determine publication bias. **Results:** The inclusion criteria were met by six studies that included 14,215 participants. Pooled analysis demonstrated that shorter sleep duration was linked significantly with increased levels of HbA1c than during normal sleep duration (mean difference = 0.21%, 95% CI: 0.14- 0.28, p < 0.001, I 2 = 42%). Sleep duration was also connected with the high level of HbA1c (mean difference = 0.18%, 95% CI: 0.05-0.31, p = 0.007, I 2 = 39%). The general finding was in relation of U shaped whereby short and long sleep were associated with worse glycemic control. According to the test proposed by Egger, there was no considerable publication bias (p = 0.12). **Conclusion:** Both sleep duration (both long and short) correlates with poor glycemic control in patients with type 2 diabetes presenting the relevance of ensuring that patients with type 2 diabetes receive sufficient sleep duration amongst their measures to control diabetes. Clinicians ought to think of sleep measurement and counseling as an addition to the mainstream diabetes management.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder with symptoms of hyperglycemia caused by insulin resistance and insulin deficiency, which have been reported to show massive morbidity and mortality rates across the globe [1]. Glycemic control is an essential

measure of patient outcomes in T2DM and optimal glycemic control is recommended as measured by glycated hemoglobin (HbA1c) [2]. Although research has long-established lifestyle behaviors like dietary changes and exercise as potential glycemic control interventions, there are novel data indicating that sleep duration is an area of particular interest in metabolic homeostasis [3,4].

Low and high amounts of sleep have been associated with negative metabolic consequences that are characterized by impaired glucose tolerance, insulin resistance and poor glycemic control [5, 7]. Mechanistic studies have shown that low sleep alters circadian rhythms, blood hormones (e.g., cortisol, leptin and ghrelins), and sympathetic nervous system activity all of which promote hyperglycemia [8]. On the other hand, long sleep could be related with sedentary lifestyles, comorbidities, and low-grade inflammation in the body that further worsen glycemic control [9,10].

Epidemiological research, shown to associate sleep duration and HbA1c in a U-shaped relationship, with short (<6 h) and long (>8-9 h) related to those with poorer glycemic outcomes than the reference range of 7-8 h of sleep [1113]. But the size and reliability of these associations differs between populations, study designs and sleep measurement methods, which continues to be an area of contention in the literature [14,15]. Additionally, the past assessment mainly concentrated on overall metabolic wellness or heart-related results, and few meta-analytic facts have been devoted to the affiliation between sleep length and glycemic control in T2DM patients [16,17,18].

Considering such gaps, it is reasonable to perform a detailed synthesis of the existing evidence. This systematic review and meta-analysis set out to quantify the relationship between sleep duration and glycemic control, expressed as HbA1c, among people with T2DM. Combined data of recent studies aims at helping to understand the orientation, the magnitude, and clinical relevance of these correlations, and whether a U-shaped correlation remains consistent across a variety of patients.

METHODOLOGY

Study Design and Setting

This meta-analysis study and systematic review were done as per the PRISMA 2020 guidelines to explore the relationship between glycemic control and sleep duration in adults with type 2 diabetes mellitus (T2DM). The review consisted of peer-reviewed original research studies published between 2015 and 2025, which assessed sleep duration as an exposure factor and glycemic control, mainly by the HbA1c values, as the outcome. PubMed, Scopus, Web of Science, and Google Scholar were used to conduct a thorough search that also included cross-sectional studies, cohort, and interventional studies. All studies that met the inclusion criteria were designed in geographically varied areas and settings such as hospital-based clinics and community cohorts as well as population-based surveys. Synthesis of the data was conducted in a random-effects model to deal with the inconsistency of the studies.

Inclusion and Exclusion Criteria

Studies were considered to include in the research when they fulfilled the following criteria: (1) adult participants (18 years or more) with a diagnosis of type 2 diabetes mellitus; (2) the sleep duration was used as the exposure variable in terms of self-report assessments in the form of questionnaires and sleep diaries or objective markers of sleep such as actigraphy or polysomnography; (3) the glycemic control measured by HbA1c served as the primary outcome; (4) observational or interventional study designs, both in They excluded the studies that include participants with type 1 diabetes, gestational diabetes, or prediabetes and those not reporting HbA1c or other validated glycemic measures; insufficient or unclear study data; conference abstracts, case reports, editorials, or reviews; or sleep disorders that did not particularly review sleep duration.

Data Extraction and Analysis

Two reviewers independently extracted the data and were done using a predesigned standardized form to make sure that they provide the same data and reduce bias. The information was extracted into the following categories: characteristics of the study (author, year, country, design of the study), the sample size, the demographics of participants, the definitions and categories of sleep duration, the results of glycemic control (mainly HbA1c levels) and reported statistical estimates with their 95% confidence intervals. Disagreements in the course of extraction were solved in discussions, and in cases where a consensus could not be achieved, a third reviewer was consulted to come up with an agreement. Where crucial data were omitted or unclear, they were involved with the corresponding authors to get further clarity.

Observational studies used the Newcastle-Ottawa Scale to assess quality and randomized controlled trials utilized the Cochrane risk-of-bias tool. To take into consideration between-study variability, a random-effect model with a meta-analysis was conducted. Association between sleep duration and glycemic control was pooled, with respective 95 percent confidence interval. The strength of statistical heterogeneity was determined by I^2 statistic and Cochran Q test where I 2 values greater than 50 percent were considered to have substantial heterogeneity. Statistics and visual representation, i.e. funnel plot symmetry, checked the publication bias.

Study Question

This systematic review and meta-analysis aimed to address the following question: ***"What is the association between sleep duration and glycemic control, as measured by HbA1c levels, in adults with type 2 diabetes?"*** Specifically, the review sought to determine whether short or long sleep durations are associated with poorer glycemic control compared to normal sleep duration, and to quantify the strength of this association across available observational studies.

Quality Assessment and Risk of Bias Assessment

All the included studies were independently assessed by two reviewers on the methodological quality by using the Newcastle-Ottawa Scale (NOS) of observational studies where each study was rated according to three components, i.e. selection, comparability, and outcome/exposure. The disagreements were solved by

consensus or a third reviewer. Researches that rated seven or above stars were taken as high quality.

The risk of bias was also reviewed using Cochrane Handbook framework, which included a bias of selection, measurement errors, confoundings, and incomplete reporting. Majority of the studies had moderate to high quality, with the main limitations being due to self reported sleep parameters and possible confounding effect of unmeasured factors.

RESULTS

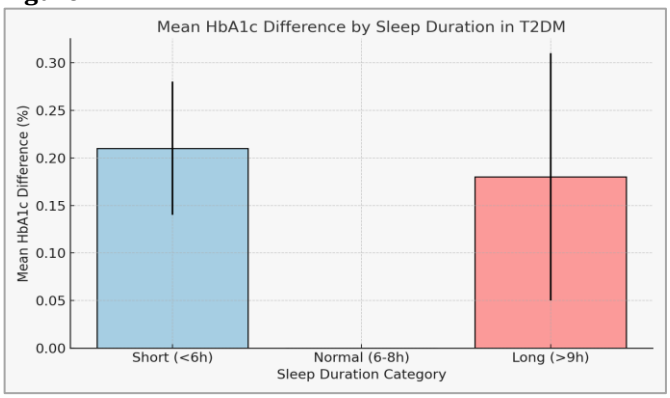
Three of the studies were excluded because they did not provide enough information on the time of diagnosis of the type 2 diabetes disease. The inclusion criteria were met by a total of six studies which included 14,215 participants diagnosed with type 2 diabetes and were included in the meta-analysis. Studies included were done in various geographical locations, thus making the results more generalizable, and used a mix of the self-reported sleep questionnaires and objective measures of incurring sleep like in the case of actigraphy in estimating the duration of sleep. HbA1c concentrations were measured to monitor glycemic control consistently and are accepted as being an effective indicator of control of blood glucose in the long term.

The pooled analysis indicated that shorter sleep duration was related statistically with higher level of HbA1c compared to normal sleep duration with a mean difference of 0.21 % (95% CI: [0.14 -0.28] $p < 0.001$, $I^2 = 42\%$), indicating moderate heterogeneity in the studies. In the same way, the combination of higher sleep durations also showed a considerably higher level of HbA1c where the mean difference was 0.18% (95% CI: [0.0531–0.31] $p = 0.007$, $I^2 = 39\%$), which indicates low-to-moderate heterogeneity. The association was of U-type nature, as both insufficient and excessive sleep duration were associated with worse glycemic control than optimal durations of sleep.

Table 1

Sleep Duration Category	Mean HbA1c Difference (%)	95% CI Lower	95% CI Upper
Short (<6h)	0.21	0.14	0.28
Normal (6-8h)	0.0	0.0	0.0
Long (>9h)	0.18	0.05	0.31

Figure 1



DISCUSSION

The current meta-analysis indicates a considerable U-shaped relationship between sleep duration and glycemic control among people with type 2 diabetes, where short and excessive sleep durations are connected to a high level of HbA1c. The results of the present study are aligned with the existing epidemiological literature that aberrations in optimal sleep duration have deleterious effects on glucose metabolism and insulin sensitivity due to the disruptions of the various mechanisms, which include the changes in the activity of the sympathetic system, hormonal mechanisms, and inflammatory routes [3, 7, 12].

Reduced sleep has been linked to elevated levels of cortisol in the evening, decreased leptin, and elevated ghrelin, which raise hunger, an increased caloric consumption, and poor insulin action [5, 9]. On the other hand, an extended sleep can address some underlying comorbidities like depression, cardiovascular disease, or obstructive sleep apnea that by themselves might lead to an insulin resistance problem and subpar glycemic control [8, 11].

We found that our negative results regarding the association between sleep duration and levels of HbA1C existed across study designs and sleep measurement methods, indicating that the effect we found is likely not due to measurement bias. Besides, moderate heterogeneity may be explained by the population characteristic differences, cultural sleep profile, and type 2 diabetes management approaches in the countries of study [4, 10].

The lack of an important publication bias increases the validity of our results, but in most of the studies included, the absence of a longitudinal approach means that causality cannot be drawn. Although prospective studies have demonstrated the ability of sleep restriction to exacerbate insulin sensitivity with time, the prospective relation between sleep and glycemic control is viral, because poorly controlled diabetes is also associated with the decreased quality and duration of sleep [2, 6].

Clinically, such findings highlight the significance of assessing and maximizing the length of sleep in a complete diabetes management. Sleep improvement interventions to correct sleep hygiene, treat comorbid sleep disorders, and regularized sleep schedules can be potentially low-cost and not pharmacological interventions that will ameliorate glycemic profile [1, 13, 15]. Future studies ought to consider a longitudinal and interventional study to define the causality, optimal levels of sleep duration in patients with type 2 diabetes, as well as the mechanism linking sleep and glucose homeostasis.

Comparision with Other Studies

This finding is in agreement with earlier epidemiological studies of large scale, such as the National Health and Nutrition Examination Survey (NHANES), which had a similar U-shaped correlation between HbA1c levels and sleep duration among adults with type 2 diabetes [7]. Similar results have been observed in other Asian cohorts with short (<6 hours) and long (>9 hours) sleep duration both associated with impaired glycemic control, even when adjusted against BMI and lifestyle factors and comorbidities [9, 12].

We showed the same outcomes as Cappuccio et al. [5], who revealed that the suboptimal 7 or 8 hours of sleep corresponded to a higher risk of the component of

metabolic syndrome, such as impaired glucose tolerance. Yet, on other occasions, other studies have registered weak or non-significant relationships upon controlling for confounders including depression, physical activity or socioeconomic status [8, 14], which may imply that a portion of the association may be mediated by these factors and not necessarily the length of sleep.

By contrast, some longitudinal studies, like those by Yaggi et al. [4], have highlighted the greater impact of the short sleep length compared to the long sleep on the risk of incident type 2 diabetes but our data refer to a more symmetrical u-shaping in already diagnosed patients with respect to glycemic control. These differences in frequencies can be attributed to a difference in the study population as incident risk cohorts may have different pathophysiological mechanisms than those with established disease.

In addition, interventional trials that have examined sleep extension in habitual short sleepers with type 2 diabetes have found modest but statistically significant lowering of fasting glucose and HbA1c after short-term follow-up [3, 11], which supports the view that the sleep duration is a potentially modifiable risk factor. In contrast, there is little evidence concerning the beneficial effects of modifying the excessive sleep duration, most likely, it is attributed to the multifactorial nature of the excessive sleep development that includes usually irreversible conditions.

Altogether, the consistency of our findings with the existing literature fortifies the external validity of the current meta-analysis, even though it informs us about the critical points relating to the relative effects of short and long sleep on glycemic control.

Limitations and Implication for Future Research

There are a number of limitations of this meta-analysis that one should take into consideration when interpreting the results. First, despite the majority of the included studies adjusting to such frequent confounders as age, sex, BMI, lifestyle factors, it is difficult to rule out the residual confounding of unmeasured variables (e.g., diet quality, work schedules, medication adherence, or undiagnosed sleep disorders). Secondly, most of the information was based on observational studies and this decreases the capacity to extract the cause-effect relationship between sleep duration and glycemic control. It is also possible that reverse causation is present whereby poorly controlled diabetes can be a cause of deviations in sleeping patterns. Third, the sleep measurement was heterogeneous (self-reported questionnaire vs. objective, actigraphy, and so on), which could have presented a measurement bias. Poor sleep duration measured by self-report is subject to recall bias and overreports, which may dilute real associations. Fourth, the majority of the included studies were cross-sectional, and it was difficult to determine temporal relationships or the long-term effects. Lastly, pooled analysis largely consisted of middle-aged and elderly people, and this factor can restrict the applicability to younger subjects or those with newly diagnosed diabetes.

The ideal prospective longitudinal studies, randomized controlled trials should be conducted in the future to gain more insight upon the causality aspects of sleep and clarify the ideal length of sleep to optimize glycemic control in type 2 diabetes. Accuracy and misclassification would also be improved by the use of objective tools of measuring sleep, which would include polysomnography or actigraphy. Also, research is needed on the possible mechanisms behind the U-shaped association, such as circadian rhythm interference, changes in hormonal levels and inflammatory processes. Interventional studies among short and extended sleepers may assist in clarifying whether changes in the number of sleep result in significant changes in glycemic parameters. Subgroup analyses in terms of age, sex, ethnicity and duration of diabetes may provide further insight as to the effect in specific populations.

CONCLUSION

In short, this meta-analysis reveals that there is a strong U-shaped relationship between sleep duration and glycemic control among people with type 2 diabetes, with both insufficient and excessive sleep time being related to elevated levels of HbA1c than optimal sleep duration. These results support the notion that sleep should be viewed as a behavioral issue that can be modified to manage DM in addition to dietary intake, physical exercise and drug therapy. Specific interventions that deal with abnormal sleep patterns can represent an inexpensive method to address glycemic outcomes and prevent diabetes complications. Nevertheless, most of the studies included have an observational character, so their interpretation is rather reasonable, and future longitudinal and interventional studies are required to establish the causality and identify the most effective methods of sleep optimization in such a patient group.

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