Both short and long sleep durations are associated with type 2 diabetes, independent from traditional lifestyle risk factors—The Maastricht Study

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ABSTRACT

Objectives: This study examined the cross-sectional association between sleep duration, prediabetes, and type 2 diabetes, and its independence from the traditional lifestyle risk factors diet, physical activity, smoking behavior, and alcohol consumption.

Methods: Cross-sectional data from 3561 people aged 40-75 years recruited into The Maastricht Study between 2010 and 2018 were used (1:1 female: male and mean age: 60.1 years [standard deviation: 8.6]). Sleep duration was operationalized as in-bed time, algorithmically derived from activPAL3 accelerometer data (median 7 nights, IQR 1). Glucose metabolism status was determined with an oral glucose tolerance test. Multinomial logistic regression was used to assess the association of sleep duration as restricted cubic spline with prediabetes and type 2 diabetes. We adjusted for sex, age, educational level, the use of sleep medication or antidepressants, and the following lifestyle risk factors: diet quality, physical activity, smoking behavior, and alcohol consumption.

Results: A U-shaped association between sleep duration and type 2 diabetes was found. Compared to those with a sleep duration of 8 hours, participants with a sleep duration of 5 and 12 hours had higher odds of type 2 diabetes (OR: 2.9 [95% CI 1.9 to 4.4] and OR 3.2 [2.0 to 5.2], respectively). This association remained after further adjustment for the lifestyle risk factors (OR: 2.6 [1.7 to 4.1] and OR 1.8 [1.1 to 3.1]). No such association was observed between sleep duration and prediabetes.

Conclusions: Both short and long sleep durations are associated positively and independently of lifestyle and cardiovascular risk factors with type 2 diabetes, but not with prediabetes.

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Introduction

Type 2 diabetes mellitus is a disease characterized by hyperglycemia, which is caused by variable degrees of insulin resistance and impaired insulin secretion. It is a prominent and growing cause of disability and death, having a pronounced economic impact on societies worldwide. As such, the United Nations has designated type 2 diabetes as one of four noncommunicable diseases, which should be targeted with the highest priority. Westernized and sedentary lifestyle behaviors have been identified as a major driver of the growth of type 2 diabetes incidence.

Traditionally, smoking behavior, alcohol consumption, diet, and physical activity were the lifestyle behaviors of primary interest in type 2 diabetes research. Another behavior that has garnered substantial interest in health research in recent years is sleep. Efforts are ongoing to explore the roles of various aspects of sleep play in the promotion of health and the underlying mechanisms involved and clarify what defines healthy sleep. Multiple aspects of sleep, such as duration, quality, regularity, disturbance, and timing, appear to be associated with type 2 diabetes. Current guidelines regard 7-9 hours of sleep as a healthy sleep duration for adults with an age between the ages of 18 and 64 years, with a lower upper limit of 8 hours for adults over 65 years of age. Sleep durations under 6 hours and over 10 hours are viewed as outside the healthy range.

There are indications that the number of adults sleeping less than 6 hours is growing, with unfavorable health effects, and developments at individual, social, and societal levels may increasingly compromise sleep duration.

Meta-analyses of prospective studies involving self-reported sleep have suggested a higher risk for type 2 diabetes with both short and long sleep durations. Two recent studies found associations between sleep measured with polysomnography and glycemic control. There remains a paucity of studies that make use of objectively measured sleep in relation to type 2 diabetes. In addition, some studies investigate associations only in men or in women, and most studies rely on self-reported outcome measures or include a limited set of confounding factors. Furthermore, pre-diabetes is not considered in most studies. Finally, although there has been a comparison of type 2 diabetes risk between sleep-related measures and traditional risk factors, it is not well-understood whether sleep duration is a risk factor for diabetes independent of these lifestyle factors.

Epidemiological studies with objectively assessed sleep have mostly used wrist-worn accelerometer data to estimate sleep duration. More recently, algorithms for deriving “bed time” using data from thigh-worn accelerometers have been developed. A method to estimate sleep time from thigh-worn accelerometer data performed almost as well as the traditional wrist-worn accelerometers. A recent study comparing sleep duration measurements from thigh-worn accelerometer data to polysomnography concluded that thigh-worn devices were able to measure total sleep time with sufficient accuracy.

The aim of the present study is to examine the association between sleep duration measured by thigh-worn accelerometry and the presence of prediabetes and type 2 diabetes in a contemporary Dutch sample and to understand if any such association is independent of the traditional lifestyle risk factors for type 2 diabetes.

Material and methods

Study design and population

We used data from The Maastricht Study, an observational prospective population-based cohort study. Detailed rationale and methods have been described previously. In brief, the study focuses on the etiology, pathophysiology, complications, and comorbidities of type 2 diabetes mellitus and is characterized by an extensive phenotyping approach. Eligible for participation were individuals aged between 40 and 75 years and living in the southern part of the Netherlands. Participants were recruited through mass media campaigns and from the municipal registries and the regional Diabetes Patient Registry via mailings. Recruitment was stratified according to known type 2 diabetes status, with an oversampling of individuals with type 2 diabetes, for reasons of efficiency. The examinations of each participant were performed within a time window of three months.

The study has been approved by the Institutional Medical Ethical Committee (NL 31329.068.10) and the Minister of Health, Welfare and Sports of the Netherlands (Permit 131088–105234-PG). All participants gave written informed consent.

We had cross-sectional data from 7689 participants who had completed the baseline survey between November 2010 and January 2018. The present study included 5561 participants in a complete-case analysis, after the exclusion of participants because they had diabetes other than type 2 (n = 50), had missing sleep duration data due to the accelerometer not being available at the start of the study or invalid measurements (n = 1434), or had missing other covariates (n = 644), as shown in more detail in Fig. 1.

Measures

Glucose metabolism status

Glucose metabolism status was determined according to the World Health Organization diagnostic criteria. All participants underwent a standardized 2-hour 75-g oral glucose tolerance test after overnight fasting. Blood samples were collected at baseline and 15, 30, 45, 60, 90, and 120 minutes after participants had consumed the 75-g glucose drink. Participants who were on insulin therapy and participants with a fasting glucose level higher than 11.0 mmol/l (as determined by finger prick) did not undergo this test for safety reasons. Prediabetes was defined as impaired fasting glucose (fasting plasma glucose 6.1-7.0 mmol/l and 2-hour plasma glucose < 7.8 mmol/l) or impaired glucose tolerance (fasting plasma glucose < 7.0 mmol/l and 2-hour plasma glucose ≥ 7.0-11.1 mmol/l). Type 2 diabetes was defined as fasting plasma glucose ≥ 7.0 mmol/l or 2-hour plasma glucose ≥ 11.1 mmol/l. Participants without type 1 diabetes using blood glucose-lowering medication were classified as having type 2 diabetes.

Sleep duration

Sleep duration was operationalized in terms of minutes in bed-time. This was algorithmically derived from data recorded by a thigh-worn activPAL3 accelerometer (PAL Technologies, Glasgow, U.K.). The activPAL3 is a small (53 mm × 35 mm × 7 mm), lightweight (15 g) device that records movement in the vertical, anteroposterior, and mediolateral axes and determines posture (sitting or lying, standing, and stepping) based on acceleration information. After the device had been waterproofed using a nitrile sleeve, it was attached directly to the skin on the front of the right thigh with transparent Tegaderm (3M, Maplewood, MN, USA) tape. Participants were asked to wear the accelerometer for eight consecutive days, without removing it at any time. To avoid inaccurately identifying nonmovement time, participants were asked not to reattach the device once removed. Data were uploaded using the activPAL software and processed using customized software written in MATLAB 2019b (MathWorks, Natick, MA, USA). Data from the first day were excluded from the analysis because participants performed physical function tests at the research center after the device was attached. In addition, data from the final wear day providing ≤ 14 wear hours of data were excluded from the analysis. The algorithm to determine the in-bed time was described in more detail elsewhere. In brief, it identified different wake and bed times for each day for each
participant based on the number and duration of sedentary periods within a moving time window between 19:00 and 12:00 hours.\textsuperscript{20} There was high agreement between self-reported sleep diary times and the algorithmically derived sleep duration.\textsuperscript{20}

\section*{Covariates}

Additional data were collected for descriptive purposes or inclusion in main analyses as confounders. Age in years, sex (male, female), educational level (low: no education, primary education, and lower vocational education; medium: general secondary education, general vocational education, higher secondary, and pre-university education; and high: higher vocational education and university education), occupational class (self-report that best fits current or past job: not employed, self-employed, lower occupational class, intermediate occupational class, higher occupational class, or other), and history of cardiovascular disease (yes, no) were assessed by questionnaire. Income was calculated by dividing self-reported net household income per month (the midpoint of one of 19 categories, ranging from 0 to >5000€ per month) by the square root of household size. Lifestyle factors included smoking behavior, alcohol use, diet, and physical activity. Smoking behavior (never, former, or current smoker) was assessed by questionnaire. Alcohol use and diet were evaluated with a food frequency questionnaire and operationalized as energy intake (mean kcal/day), diet quality (as the Dutch Healthy Diet index, a measure of adherence to dietary guidelines) for diet, and grams of alcohol per day for alcohol use.\textsuperscript{25,26} Physical activity was operationalized as moderate-to-vigorous physical activity in min/day and defined as stepping time with a step frequency $\geq$ 100 steps/min, based on the activPAL3 data. Medication use was ascertained in an interview where generic name, dose, and frequency were registered. Data on the use of sleep medication, antidepressants, lipid-modifying medication, blood pressure-lowering medication, or glucose-lowering medication were available as dichotomous variables (use: yes, no). The presence or absence of a current depressive episode was assessed with the MINI diagnostic interview and used as a dichotomous variable.\textsuperscript{27} Neuropathic pain was defined as a score $\geq$ 3 on the DN4-interview and used as a dichotomous variable.\textsuperscript{28} Body mass index (BMI) was calculated from measurements of weight and height to the nearest 0.5 kg or 0.1 cm. Peripheral vibration perception was tested with a Horwell Neurothesiometer (Scientific Laboratory Supplies, Nottingham, U.K.). Vibration perception thresholds were tested three times at the distal phalanx of the hallux on both feet. Impaired vibration perception (yes, no) was defined as having a vibration perception threshold at one or both toes above the predicted 97.5 percentile. The predicted 97.5 percentile was based on the vibration perception threshold of

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{flowchart.png}
\caption{Flowchart of participant inclusion and missing values}
\end{figure}
the right toe of a healthy reference population as described previously. Blood pressure was used dichotomously, based on the average blood pressures that were measured multiple times during the visits to the research center (high blood pressure if blood pressure-lowering drugs were used or if the systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg). Total cholesterol and HDL cholesterol were measured in the fasting blood samples with an automatic analyzer (Beckman Synchron LX20, Beckman Coulter, Brea, CA, USA), as described previously.

### Statistical analysis

We computed descriptive statistics for the total study population and according to sleep duration as means (standard deviation [SD]), median (25%–75%), or percentages as appropriate. Characteristics of included and excluded participants were compared statistically with χ²-tests for categorical, one-way analysis of variance for normally distributed, and Kruskal-Wallis tests for not-normally distributed continuous variables.

As previous research suggested, a U-shaped association between sleep duration and diabetes. Linear or second-degree polynomial terms, or a restricted cubic spline with knots at percentiles 10, 50, and 90 was considered potentially suitable functional forms for the representation of sleep duration. Descriptive statistics and locally weighted scatterplot smoothing plots suggested a U-shaped association between sleep duration and prediabetes, and type 2 diabetes. Likelihood ratio tests of crude logistic regression models, including linear, second-degree, and third-degree polynomial terms, indicated a statistically significant advantage for the model, including the second-degree term. A visual comparison of predicted probability plots based on crude models, including the second-degree polynomial term or the restricted cubic spline and a locally weighted scatterplot smoothing plot, did not show large discrepancies. Comparison the Akaike information criterion implied a small statistical advantage for the restricted cubic spline. Consequently, the restricted cubic spline was chosen to represent sleep duration in prespecified regression models.

We selected potential confounding factors on theoretical grounds. The conceptual causal model is shown in Fig. A1. The first regression model was adjusted for sex, age, and education. The second model was additionally adjusted for neuropathic pain, history of cardiovascular disease, use of sleep medication, or antidepressants. The third model added the other lifestyle factors: diet quality, smoking behavior, physical activity, and alcohol consumption. A potential multiplicative interaction between sleep duration and sex was explored by including an interaction term. In a sensitivity analysis, we included only participants with diabetes, who were newly diagnosed through The Maastricht Study measurements and combined using Rubin’s rules.

### Ancillary analyses

We performed ancillary analyses. To explore the sensitivity of our prespecified models to alternative variable selection, we substituted two sets of variables. First, we replaced diet quality with energy intake in model 2. Second, we replaced diet quality with energy intake in model 3. Third, we additionally adjusted for BMI and current depression in model four. BMI and current depression were entered into a separate model because of the risk of overadjustment bias: these factors may be confounders, but they may also mediate the association between sleep duration and (pre)diabetes. In addition, we added BMI and current depression separately to model 3 to probe the distinct influence of these model variables.

As suggested during peer review, we added an analysis of imputed data. Therefore, we used multiple imputations to impute covariates missing under a missing-at-random assumption. We created 32 datasets (n = 6205 complete observations each) using multivariate imputation by chained equations with 20 iterations. Imputation models used predictive mean matching for continuous variables and multinomial, ordinal, or binary logistic regression for categorical variables as appropriate. Model coefficients and their standard errors were estimated in each imputed dataset separately and combined using Rubin’s rules.

### Results

Of the 5561 participants included in this study, 3388 had normal glucose metabolism status, 832 had prediabetes, and 1341 had type 2 diabetes. Participants had a mean sleep duration of 8.3 hours (SD 0.9). A median of 7 (IQR 1) nights of accelerometer data was available per participant. A sleep duration lower than 7 hours was found in 443 participants, 4044 participants had a sleep duration between 7 and 9 hours, and 1074 participants had a sleep duration higher than 9 hours, with relatively more people with type 2 diabetes in the groups sleeping short or long. The mean age was 60.1 years (SD 8.6). Participants sleeping shorter than 7 hours were more often male than participants sleeping between 7 and 9 hours or more (63%, 50%, and 44%, respectively) and were slightly younger. Furthermore, participants sleeping shorter than 7 hours more often had type 2 diabetes, had higher physical activity levels, were more often current smokers, had a higher energy intake, and had a higher alcohol intake. Additional characteristics of the included participants are shown in Table 1.

A comparison between participants that we included in the analysis and participants that we excluded or who had missing data (tabulated in Table A1) suggested that the included participants were slightly healthier. Statistics indicated that included participants were older (60 versus 59 years) and more often female (50% versus 48%), had a lower BMI (26.9 versus 27.2), were less often current smokers (12% versus 17% current smokers), included fewer people with type 2 diabetes (24% versus 27% type 2 diabetes), and used glucose-lowering medication more often (21% versus 18%).

### Association of sleep duration with prediabetes and type 2 diabetes

Table 2 presents odds ratios and their 95% confidence intervals for prediabetes and type 2 diabetes per hour of sleep duration, relative to a reference point at 8 hours. Figs. 2 and 3 depict plots of the odds ratios, including pointwise confidence bands for models 2 and 3 across the range of observed values of sleep duration from 294 to 762 minutes for prediabetes and type 2 diabetes.

The odds ratios for prediabetes and type 2 diabetes in model 1, adjusted for sex, age, and educational level, increased toward both the higher and lower sleep durations. Compared to the reference at a
After further adjustment for use of sleep medication or antidepressants in model 2, the results remained statistically significant with odds ratios of 2.7 [1.7 to 4.3] at 5 hours and 3.1 [1.9 to 5.2] at 12 hours for type 2 diabetes.

Odds ratios remained statistically significant but were attenuated further in model 3, which additionally included diet quality, alcohol use, smoking behavior, and moderate-to-vigorous physical activity. The odds ratios for 5 and 12 hours in this model were 2.6 [1.6 to 4.1] and 1.8 [1.1 to 3.0] for type 2 diabetes.
Table 2
Odds ratios and 95% confidence intervals of prediabetes and type 2 diabetes by sleep duration

<table>
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<th>Outcome</th>
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<th>Odds ratio</th>
<th>95% CI</th>
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<td>3.1</td>
<td>1.9-5.2</td>
<td>1.8</td>
<td>1.1-3.0</td>
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</table>

CI: confidence interval
Model 1: adjusted for sex, age, educational level.
Model 2: adjusted for sex, age, educational level, antidepressant use, and sleep medication use.
Model 3: adjusted for sex, age, educational level, antidepressant use, sleep medication use, physical activity, diet quality, alcohol use, and smoking behavior.
* Reference sleep duration.
† Not statistically significant.

Fig. 2. Odds ratios and 95% confidence intervals of prediabetes and type 2 diabetes by sleep duration. Model 2: adjusted for sex, age, educational level, antidepressant use, and sleep medication use, reference at 8 hours

Fig. 3. Odds ratios and 95% confidence intervals of prediabetes and type 2 diabetes by sleep duration. Model 3: adjusted for sex, age, educational level, antidepressant use, sleep medication use, physical activity, diet quality, alcohol use, and smoking behavior, reference at 8 hours
Joint Wald tests for the interaction terms did not indicate a statistically significant interaction between sex and sleep duration (model 1: $\chi^2$ [df = 2]: 1.93, p = 0.38 for prediabetes and 2.86, p = 0.24 for type 2 diabetes; model 2: $\chi^2$ [df = 2]: 2.00, p = 0.37 for prediabetes and 2.05, p = 0.22 for type 2 diabetes; and model 3: $\chi^2$ [df = 2]: 1.85, p = 0.40 for prediabetes and 2.29, p = 0.32 for type 2 diabetes). When the 1113 participants with type 2 diabetes known prior to the baseline measurements were excluded from model 2, the association was attenuated on the short end of sleep and lost statistical significance, while it remained similar and retained statistical significance on the long end in that substantially smaller subgroup (OR 1.2 [0.5 to 3.2] for 5 hours and 3.4 [1.3 to 9.0] for 12 hours).

Ancillary analyses

Replacing educational level with occupational category and income in model 2 led to a lower estimate for short sleep duration (OR 2.3 [1.3 to 3.9] at 5 hours and 2.9 [1.6 to 5.3] at 12 hours); for this analysis, we had complete data for 4015 participants. Replacing diet quality with energy intake in model 3 did not substantially alter the results (OR 2.6 [1.6 to 4.2] at 5 hours and 1.7 [1.0 to 2.8] at 12 hours). After additional adjustment for BMI and current depression in model 4 (seen in Table A2), odds ratios for type 2 diabetes remained statistically significant on the short end and lost statistical significance on the long end, OR 1.8 [1.1 to 3.0] and 1.5 [0.9 to 2.7] at 5 and 12 hours, respectively. When adding current depression and BMI separately, the association between sleep duration and type 2 diabetes remained similar for the former (OR 2.5 [1.6 to 4.0] at 5 hours and 1.8 [1.0 to 3.0] at 12 hours), while it was similar to model 4 for the latter (OR 1.8 [1.1 to 3.0] at 5 hours and 1.5 [0.9 to 2.7] at 12 hours).

Analysis based on the multiply imputed datasets produced estimates similar to the complete-case analysis, as can be seen in Table A3. The inclusion of only non–shift-working participants did also not substantially change the estimates, as shown in Table A4.

Discussion

In our data, both long and short sleep durations as assessed objectively by accelerometry were associated with increased odds of type 2 diabetes. This association was independent of smoking behavior, alcohol consumption, diet, and physical activity. The association between sleep duration and prediabetes suggested a similar directionality for most models but did not achieve statistical significance.

Our findings are consistent with some but not all earlier findings. The mean sleep duration that we found (8.3 hours [SD 0.9]) is in the range of the sleep duration of 7.8 hours [0.9] for people aged 41–65 years and 7.9 hours [1.1] for people > 65 years reported by a study that used questionnaire-based data of 1.1 million people from the Netherlands, the United Kingdom, and the United States. In line with the current study, meta-analyses of prospective studies found an association between both short and long sleep durations and type 2 diabetes risk. However, some recent findings in cross-sectional studies also using objective measures to estimate sleep duration (wrist-based accelerometer data) do not align with the current study’s results. Two studies found no statistically significant association between sleep duration and type 2 diabetes prevalence, and one found an inverse association between sleep duration and type 2 diabetes prevalence. These studies included substantially smaller or different study populations (e.g., community-dwelling older individuals with overweight/obesity and metabolic syndrome, and different sleep behaviors), different sets of covariates, and used different statistical analysis techniques, which may have contributed to the divergent findings. An earlier study on the association between sleep duration and type 2 diabetes, as well as prediabetes, found an association between long and short sleep durations and type 2 diabetes, but not prediabetes, akin to this study. Studies into the associations between actigraphy-based sleep duration and glycemic control, as indicated by HbA1c levels, have found a U-shaped association or association with short sleep, also generally aligning with the findings of this study.

The mechanisms by which sleep duration or associated circadian disruption relates to prediabetes or type 2 diabetes are not fully understood. Multiple physiological pathways may link short sleep duration to type 2 diabetes directly or through obesity. Experimental evidence indicates that sleep deprivation changes adipocyte function, resulting in reduced insulin sensitivity. Other potential pathways might involve various functional systems, such as changes in the appetite-regulating hormones leptin and ghrelin leading to weight gain or changes in biological clocks. In addition, behavioral pathways could induce higher risk of type 2 diabetes. For example, both the increased opportunity to eat inherent to increased waking time and changes in appetite and decision-making capacity associated with insufficient sleep could result in unhealthy food choices and higher caloric intake, leading to type 2 diabetes. However, the inclusion of diet quality or energy intake in our regression model did not change the association between short sleep duration and type 2 diabetes. This suggests that the association is not directly linked to food choices or energy intake, but it might still be linked to the misalignment of biological clocks, and environmental and behavioral rhythms (e.g., timing of food consumption).

The mechanisms linking long sleep duration and type 2 diabetes are more speculative than the mechanisms linking short sleep duration and type 2 diabetes, and some scholars argue that these relations might be the result of underadjustment for underlying (unknown) comorbidities. A few putative mechanisms underlying an association have been outlined. Long sleep duration might be linked to type 2 diabetes through several mechanisms, involving poor sleep quality, increased physical inactivity, unhealthy dietary choices, and circadian misalignment. Since the association was attenuated but still persisted after adjusting for diet and physical activity, unhealthy dietary choices and physical inactivity might play a limited role in that association.

Adjusting for BMI and current depression attenuated the association between sleep duration and type 2 diabetes, and only the association between short sleep duration and type 2 diabetes remained statistically significant. The observation that adjusting for BMI substantially attenuates the association between sleep duration and type 2 diabetes reinforces the notion that mechanisms associated with obesity are involved. Another explanation for this finding might be that participants with obesity have lower sleep efficiency due to a higher prevalence of sleep-disordered breathing. However, these suppositions require further scientific exploration.

In our sensitivity analysis, which included only participants who were aware of their diabetes diagnosis prior to the data collection, the association with long sleep duration remained similar and statistically significant compared to the complete sample, while the association at the short end of sleep duration was attenuated and lost statistical significance. The reasons for this finding are unclear. An explanation might be that factors, such as fatigue, sleeping difficulties, or psychosocial stress, associated with participants sleeping less and seeking medical care or those participants who have sought medical care earlier, are, thus, more likely to be diagnosed with type 2 diabetes. Another possibility might be that diabetes distress might have a negative effect on sleep duration. What should be noted is that the subsample for this analysis was substantially smaller.

This study has notable strengths. The large sample size entails good potential for the detection of an association, as does the oversampling of people with diabetes. Drawing on the extensive dataset of The Maastricht Study, it was possible to include a substantial set of variables to reduce the impact of confounding.
Measurements for many of these variables, notably for both the exposure and the outcome of interest, were done objectively, which can encourage confidence in the dependability of the data. Another strength lies in the fact that the independent variable of interest was kept continuous, and thus, loss of information, issues related to the positioning of cut points, and other drawbacks inherent to categorizing continuous variables were avoided.15

This study’s limitations deserve discussion. Our study was cross-sectional. This is important, as there are plausible causal paths between sleep duration and type 2 in both directions. Therefore, it is difficult to infer the direction of a causal relationship from our study. Sleep problems are common in type 2 diabetes,36 and various type 2 diabetes complications can interfere with sleep.17–40 Additionally, current methods to estimate sleep duration from accelerometer data cannot reliably distinguish between waking and sleeping in-bed time. These issues could result in differential misclassification, with participants with diabetes having a shorter actual sleep duration than people without diabetes. Our findings might then be an underestimation of the association between short sleep duration and type 2 diabetes, and an overestimation of the association between long sleep duration and type 2 diabetes. Nondifferentially, we might have underestimated the total sleep duration, as we could not include daytime naps, and there might be some misestimation of sleep duration due to the thigh-worn accelerometer.22 We also were not able to measure long-term changes in sleep duration, while such changes might be related to a higher risk of type 2 diabetes.30,51 Another important limitation rests in the fact that, even though this study included a substantial number of confounding factors, we were not able to include, for example, sleep quality, sleep-disordered breathing measurements, dietary timing, or stress. How psychosocial stress affects the association between sleep duration and type 2 diabetes was not addressed in previous studies, and we regret that we could not address this important question. Our adjustment for socioeconomic correlates of stress might mitigate this deficiency somewhat. Nevertheless, residual confounding could be a relevant concern. The participants’ predominantly Western European ancestry, age, and cultural habits might hinder the generalizability of the results to other populations.

Conclusions

The current findings in The Maastricht Study support the idea that sleep duration could be a relevant risk factor for type 2 diabetes independent of lifestyle risk factors, including diet, physical activity, smoking behavior, and alcohol consumption, with short sleep duration showing more independence than long sleep duration. These findings underpin the importance of promoting healthy sleep habits to avoid sleep deprivation. Better insight into the relationship between sleep and type 2 diabetes requires prospective follow-up studies that include stress and sleep quality measures and confounding factors related to circadian rhythms, such as dietary timing.

Declaration of conflicts of interest

Dr. de Galan reports grants from Novo Nordisk, outside the submitted work. All other authors have nothing to disclose.

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Credit authorship contribution statement

Conceptualization: RMM and AK; Data curation: HHCMS, SK, AW, MTS, CDAS, BEdG, MMJvG, CJHvdK, SJPM, HB, NCS, and AK; Formal analysis: JDA; Methodology: JDA, RMM and AK; Supervision: AK; Visualization: JDA; Writing – original draft: JDA; and Writing – review & editing: JDA, RMM, HHCMS, SK, AW, MTS, CDAS, BEdG, MMJvG, CJHvdK, SJPM, HB, NCS, and AK; all authors have approved the final article.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.sleh.2023.06.009.

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