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# Sleep Efficiency Predicts Next-Day Glycaemia and Daytime Glycaemia Influences Sleep in Free-Living Adults at Risk of Type 2 Diabetes

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**Received:** 14 January 2026 | **Revised:** 6 March 2026 | **Accepted:** 6 March 2026

**Keywords:** clinical physiology | continuous glucose monitoring (CGM) | glycaemic control | obesity care | observational study

## ABSTRACT

**Aim:** To investigate how sleep features influence next-day diurnal glucose homeostasis and vice versa in free-living adults at risk of type 2 diabetes.

**Materials and Methods:** This observational repeated-measures study included 388 adults aged 25–65 years (50% women) with overweight or obesity (BMI  $\geq 25.0$ – $<40.0$  kg/m<sup>2</sup>). Sleep and glucose homeostasis were simultaneously assessed over 14 days using wrist-worn accelerometers and continuous glucose monitors. Linear mixed models evaluated day-level associations between sleep metrics—wake-up time, sleep period time (i.e., time from sleep onset to wake-up) and sleep efficiency ([total sleep time/sleep period time]  $\times 100$ )—and diurnal glucose metrics, including mean glucose and its standard deviation (glycaemic variability).

**Results:** We analysed 3942 valid person-days. Each 1% increase in sleep efficiency was associated with lower next-day mean glucose (B [95% CI] =  $-0.05$  [ $-0.08$ ,  $-0.01$ ] mg/dL;  $p = 0.007$ ). Each 1 h delay in wake-up time was linked to reduced next-day glucose variability ( $-0.24$  [ $-0.38$ ,  $-0.10$ ] mg/dL;  $p = 0.001$ ). Conversely, each 1 mg/dL increase in daytime mean glucose was associated with later wake-up time (0.008 [0.002, 0.014] h;  $p = 0.008$ ), longer sleep period time (0.006 [0.000, 0.012] h;  $p = 0.039$ ) and lower sleep efficiency ( $-0.05\%$  [ $-0.08\%$ ,  $-0.01\%$ ] %;  $p = 0.005$ ) the subsequent night. Each 1 mg/dL increase in glucose variability was associated with earlier wake-up time ( $-0.02$  [ $-0.03$ ,  $-0.01$ ] h;  $p < 0.001$ ).

**Conclusions:** This study provides evidence that sleep and glucose dynamics are temporally associated in free-living adults at risk of type 2 diabetes. These findings underscore the potential of combining sleep and glucose metrics to inform cardiometabolic risk prevention strategies.

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## 1 | Introduction

Obesity is a major risk factor for developing insulin resistance and type 2 diabetes [1], with poor sleep increasingly recognised as a metabolic hallmark of these conditions [2]. Accumulating evidence suggests that inadequate sleep—characterised by irregular timing, insufficient duration and low efficiency—may act as a metabolic stressor, contributing to both obesity and impaired glucose homeostasis [2–6]. Sleep loss disrupts glucose metabolism by altering cellular responses to insulin signalling [2, 7], thereby promoting insulin resistance [8]. Conversely, dysregulated glycaemia may adversely affect sleep [9, 10], highlighting a potential bidirectional relationship between sleep and glycaemic control in individuals with overweight or obesity.

Several studies have linked both acute and chronic sleep restriction to impaired glucose tolerance and insulin resistance in healthy adults and those with overweight [11–14]. Most recently, self-reported later sleep timing and insufficient duration have been linked to increased glycaemic variability [15]. Additionally, impaired glucose homeostasis and incidence of type 2 diabetes have been associated with both short ( $\leq 5$ –6 h/day) and long ( $> 8$ –9 h/day) sleep duration in adults with obesity or prediabetes [16–18]. In the large-scale PREDICT study, poor sleep efficiency and later sleep timing were associated with greater postprandial glycaemic responses to breakfast the following morning in healthy adults under controlled dietary conditions [19]. However, it remains unclear whether these associations extend to overall diurnal glycaemic patterns in free-living adults with excess adiposity or whether daytime glucose fluctuations may also influence sleep. These day-to-day, bidirectional relationships remain largely unexplored in naturalistic settings, where behavioural factors like physical activity, diet and stress vary considerably within the same person [20].

Most previous research has relied on inpatient protocols or self-reported sleep, which may not accurately capture the complexity of behaviours under free-living conditions [13]. Actigraphy provides a reliable, day-to-day assessment of sleep timing, duration and efficiency using wrist-worn accelerometers [21], while continuous glucose monitoring (CGM) enables high-resolution tracking of 24 h glucose dynamics across both fasting and postprandial states [11, 22, 23]. Despite these methodological advances, to our knowledge, there are no studies that have integrated multidimensional, objectively measured sleep data with CGM under free-living conditions. Remarkably, the day-to-day interactions between sleep characteristics and glycaemic variability remain insufficiently characterised. Mapping these temporal dynamics in naturalistic settings may help identify modifiable behavioural targets and inform personalised strategies to improve glycaemic control through sleep optimisation.

We hypothesised that suboptimal sleep patterns would be linked to impaired glycaemic control the next day and that greater glucose levels and glycaemic variability during the day would be associated with impaired sleep timing, duration and efficiency the subsequent night. The aim of the present study was to investigate whether sleep features, including timing, duration and efficiency, were associated with glucose homeostasis the following day and the extent to which diurnal glucose levels and variability were associated with sleep timing,

duration and efficiency the subsequent night in adults at risk of type 2 diabetes.

## 2 | Materials and Methods

### 2.1 | Study Design and Setting

The present observational study used data from the baseline assessments of two randomised controlled trials (RCTs) that shared measurement protocols, ensuring methodological consistency: Study I (ClinicalTrials.gov identifier: NCT05310721) and Study II (NCT05897073). Study I was a multicenter RCT conducted in Granada (southern Spain) and Pamplona (northern Spain) [24], while Study II was single-centre RCT conducted in Granada [25]. Detailed descriptions of the trial rationale, design and protocols are available elsewhere [25, 26]. Both studies were approved by the appropriate regulatory authorities and ethics committees. All participants provided written informed consent. The study protocols followed the most recent revision of the Declaration of Helsinki and adhere to the STROBE reporting guidelines. During an identical 2 week lead-in period preceding the interventions, participants were instructed to simultaneously wear a wrist accelerometer and a CGM device for 14 consecutive days, 24 h per day, under free-living conditions. This standardised design enabled an observational repeated-measures, day-level analysis of temporally ordered associations between sleep and diurnal glucose homeostasis (Figure 1).

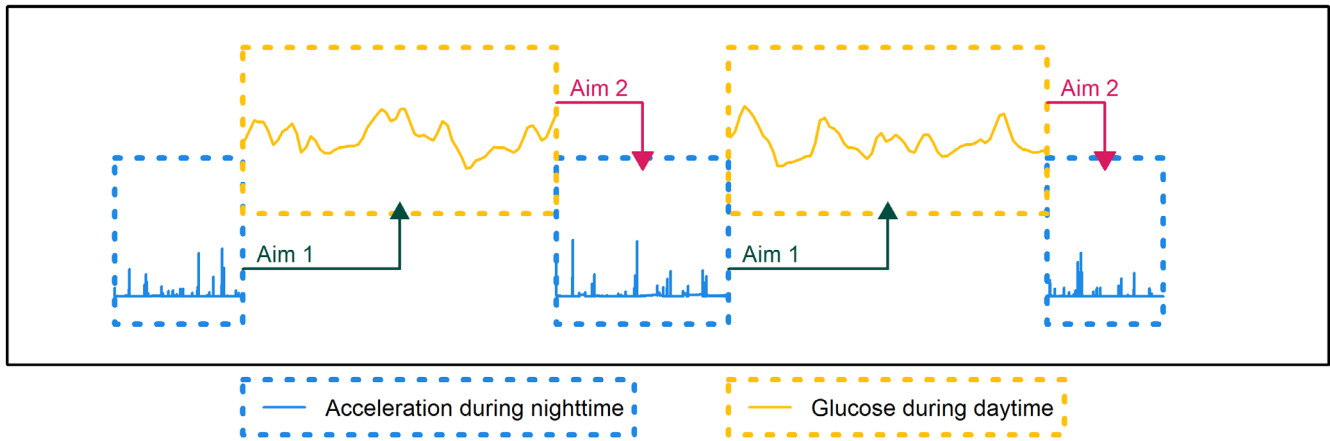
### 2.2 | Participants

For Study I, participants were recruited between 11 April 2022 and 5 December 2022, with a study completion date of 6 March 2023. For Study II, recruitment took place between 2 May 2023 and 6 April 2024, with a study completion date of September 5, 2025. Eligible participants were adult males and females aged 25–65 years with overweight or obesity, defined as a body mass index (BMI)  $\geq 25.0$ – $< 40.0$  kg/m<sup>2</sup>, who were physically inactive ( $< 150$  min/week of moderate-to-vigorous physical activity [MVPA]). Exclusion criteria were diagnosis of diabetes or cardiovascular disease, major sleep disorders, shift workers with nocturnal hours and taking any medications that could affect glucose metabolism. Full inclusion and exclusion criteria are available in the published protocols [25, 26].

### 2.3 | Measurements

#### 2.3.1 | Sleep Assessment

Participants were continuously monitored with a triaxial accelerometer (ActiGraph GT3X+, ActiGraph LLC, Pensacola, Florida, USA), initialised to record raw accelerations at a frequency of 100 Hz. Participants were instructed to wear the device on their non-dominant wrist continuously for 14 consecutive days. Additionally, participants were instructed to register the bedtime and the wake-up time every day in a study-specific mobile phone application (EXTREME:



Aim 1: association of sleep with diurnal glucose homeostasis the next day.

Aim 2: association of diurnal glucose homeostasis with sleep the next night.

**FIGURE 1** | Overview of the study design, including study aims and time windows used.

com.nnbi.app\_extreme\_granada; Tempus: com.nnbi.app\_extreme\_granada, NNBI2020 S.L., Navarra, Spain). When the 2-week lead-in period finished, raw accelerometer data were downloaded using ActiLife software (version 6.13.6; ActiGraph, Pensacola, Florida, USA) and processed with the open-source GGIR R package (version 3.1-8) [27]. Sleep and awake periods were identified using an automated algorithm based on the variability of the arm posture and guided by the sleep times reported by the participants [21, 28]. This provided information on sleep onset time, sleep period time (i.e., time from sleep onset to wake-up), total sleep time within the sleep period time, total awake time during the sleep period time (WASO), sleep efficiency after sleep onset (i.e., [total sleep time/sleep period time] × 100), wake-up time and sleep regularity index (SRI). The SRI ranges from 0 (i.e., completely random sleep/wake patterns) to 100 (i.e., perfectly consistent sleep/wake patterns) [29, 30]. Time in bed was not available, therefore, participant-reported bed and wake times were used only to guide the algorithm rather than to define sleep windows, given known discrepancies between self-reported and accelerometer-derived sleep timing in free-living settings [31]. Accordingly, sleep efficiency was calculated using sleep period time, consistent with large-scale accelerometry approaches [29], and may yield higher absolute values than conventional diary-based sleep efficiency estimates [21]. Additionally, we calculated the midsleep point as (sleep onset time + wake-up time)/2.

### 2.3.2 | Continuous Glucose Monitoring Measurements

Participants' daily glucose levels were measured using a CGM device (FreeStyle LibrePro in Granada and FreeStyle Libre 2 in Pamplona, Abbot Laboratories, Chicago, Illinois) for 14 days. These CGM models store averages of interstitial glucose every ~15 min, which provides approximately real-time glucose data [23]. The CGM device data were time-matched with the accelerometer data to allow for the calculation of the mean glucose over the diurnal and nocturnal windows, using the experimental tool that can be accessed at <https://github.com/PROFITH/GGIRmatcher>. We applied the

accelerometer-derived sleep onset and wake-up times to calculate the mean glucose over the full day (namely the 24 h mean glucose), the waking hours (diurnal mean glucose) and the sleeping hours (nocturnal mean glucose) for each participant's day. Within the diurnal window, we further computed additional glycaemic metrics, including median glucose, standard deviation (SD) of glucose, coefficient of variation (CV) and the area under the glucose curve (AUC; mg/dL\*min). The criteria to consider a valid day were as follows: (1) the CGM devices should have registered data for at least 70% of the time classified as awake in the day (i.e., considering a sampling frequency of 15 min) [23]; and (2) participants should have worn the accelerometers for at least 70% of the sleep period time in the day.

### 2.3.3 | Covariates

Energy intake was assessed using three nonconsecutive 24 h dietary recalls (24HRs), including one from a nonworking day [24, 25]. Recalls were collected through face-to-face or online interviews conducted by trained research nutritionists.

Moderate-to-vigorous physical activity (MVPA) was classified when Euclidean Norm of the raw accelerations Minus One G (ENMO) during the awake time was  $\geq 100$  mg in bouts of at least 5 min (with an allowance of 20% of the time below the threshold) [32]. Further details on the calculation of MVPA are provided elsewhere [33]. As participants reported being inactive (<150 min/week of MVPA), the MVPA assessed was considered to be lifestyle physical activity.

Venous blood samples were collected and stored at  $-80^{\circ}\text{C}$  for the analysis of fasting glucose, insulin and glycated haemoglobin (HbA1c), as previously described [25, 26].

## 2.4 | Statistical Analyses

The sample size of the original trials was calculated based on their predefined primary outcomes [25, 26]. As the present study

is a secondary analysis of these trials, no additional sample size calculation was performed.

Prior to analysis, quantitative variables within the dataset were screened for normality through visual inspection of histograms. Outliers were handled by filtering out observations below the 1st percentile and above the 99th percentile (Appendices S1 and S2). Descriptive statistics were summarised using the mean and SD for continuous variables and frequency and percentage for categorical variables.

Pearson correlations were computed to assess potential collinearity among sleep and diurnal glucose homeostasis outcomes (Appendices S3 and S7). Variables exhibiting strong correlations were subjected to principal component analysis to extract meaningful, lower-dimensional representations of sleep and diurnal glucose homeostasis metrics while minimising redundancy (Appendices S4–S6 and S8–S10). We ultimately retained wake-up time (timing indicator), sleep period time (duration indicator) and sleep efficiency (continuity indicator) for sleep and diurnal mean glucose (overall levels indicator) and diurnal SD glucose (glycaemic variability indicator) for glucose homeostasis. Detailed analysis can be found in the [Supporting Information](#).

We applied linear mixed-effects models and spline-based mixed models to assess temporally the day-level associations between nightly sleep and next-day glucose outcomes and then, as secondary analyses, between daytime glucose metrics and subsequent sleep. Both sleep and CGM measures were alternately treated as dependent and predictor variables depending on the direction of association. Participant identifiers were included as participant-specific random intercepts to account for within-person clustering of repeated daily observations. We included the following covariates: age, sex, study location, study cohort, energy intake, MVPA and the SRI. Study location and cohort were included to account for possible systematic differences between sites and CGM devices. Model fit comparisons between linear and spline-based models were conducted using the Akaike Information Criterion, Bayesian Information Criterion and Likelihood Ratio Tests (Appendices S11 and S12).

We then explored whether age, sex, energy intake, MVPA, SRI and glucose regulation status (normal vs. impaired) modified the temporal associations between nightly sleep features and next-day glucose outcomes, as well as between daytime glucose measures and subsequent-night sleep outcomes. Effect modification was assessed by adding interaction terms to the linear mixed-effects models. Participants with impaired glucose homeostasis met at least one of the following criteria: (i) fasting plasma glucose levels between  $\geq 100$  and  $\leq 125$  mg/dL, (ii) HbA1c levels ranging from  $\geq 5.7\%$  to  $< 6.5\%$  or (iii) insulin resistance, defined as a HOMA-IR  $> 2.5$ . Participants with normal glucose homeostasis did not meet any of these criteria.

Given the observational nature of the study, all analyses were considered exploratory and hypothesis-generating and no adjustments for multiple testing were conducted. The level of statistical significance was set at  $p < 0.05$ . All statistical analyses were conducted using R version 4.4.2 (<https://cran.r-project.org/>, The R Project for Statistical Computing, Vienna, Austria).

### 3 | Results

A total of 388 adults (50% women; mean age [SD], 47.2 [8.7] years) with 3942 days valid paired sleep and glucose data during nocturnal and diurnal periods were included in the present study. Table 1 displays the descriptive characteristics of the participants, with sample sizes varying across variables due to incomplete or invalid device-derived measurements and missing clinical assessments in a subset of participants. These participants provided a mean of 10.4 (3.0) days of valid data from accelerometers and CGM devices and 84.0% provided 8 or more valid days.

#### 3.1 | Association Between Sleep and Next-Day Diurnal Glucose Homeostasis

We first examined whether wake-up time, sleep period time and sleep efficiency were associated with glucose homeostasis the following day. Figure 2G–I displays the distributions of valid observations for each sleep variable. Each 1% increase in sleep efficiency was associated with lower diurnal mean glucose the next day (B [95% confidence interval, CI] =  $-0.05$  [ $-0.08$ ,  $-0.01$ ] mg/dL;  $p = 0.007$ ; Figure 2C and Appendix S13). Each 1 h delay in wake-up time was associated with lower glucose variability the following day (B [95% CI] =  $-0.24$  [ $-0.38$ ,  $-0.10$ ] mg/dL;  $p = 0.001$ ; Figure 2D and Appendix S14). No significant associations were found between wake-up time or sleep period time and next-day diurnal mean glucose (both  $p \geq 0.381$ ; Figure 2A,B and Appendix S13), nor between sleep period time or sleep efficiency and next-day diurnal glucose variability (both  $p \geq 0.121$ ; Figure 2E,F and Appendix S14).

Next, we investigated whether individual characteristics moderated the association between sleep and next-day glucose regulation. Sex significantly moderated the associations of sleep period time and sleep efficiency with next-day diurnal mean glucose (interaction terms: sleep period time  $\times$  sex, B [95% CI] =  $0.64$  [ $0.19$ ,  $1.10$ ] mg/dL;  $p = 0.005$ ; sleep efficiency  $\times$  sex, B [95% CI] =  $0.11$  [ $0.04$ ,  $0.18$ ] mg/dL;  $p = 0.002$ ), indicating that these associations differed between males and females (Appendix S18). SRI also moderated the associations of wake-up time and sleep period time with next-day diurnal mean glucose (wake-up time  $\times$  SRI, B [95% CI] =  $0.03$  [ $0.00$ ,  $0.05$ ] mg/dL;  $p = 0.019$ ; sleep period time  $\times$  SRI, B [95% CI] =  $-0.021$  [ $-0.041$ ,  $-0.001$ ] mg/dL;  $p = 0.044$ ; Appendix S18). Additionally, we observed that energy intake modified the relationship between sleep efficiency and diurnal glucose variability (interaction term: energy intake  $\times$  sleep efficiency, B [95% CI] =  $-0.00005$  [ $-0.00009$ ,  $-0.000003$ ] mg/dL;  $p = 0.035$ ; Appendix S19), with a stronger inverse association between sleep efficiency and next-day glucose variability at higher energy intake. No significant moderating effects by age, MVPA or glucose regulation status were observed (all  $p \geq 0.052$ ; Appendices S18 and S19).

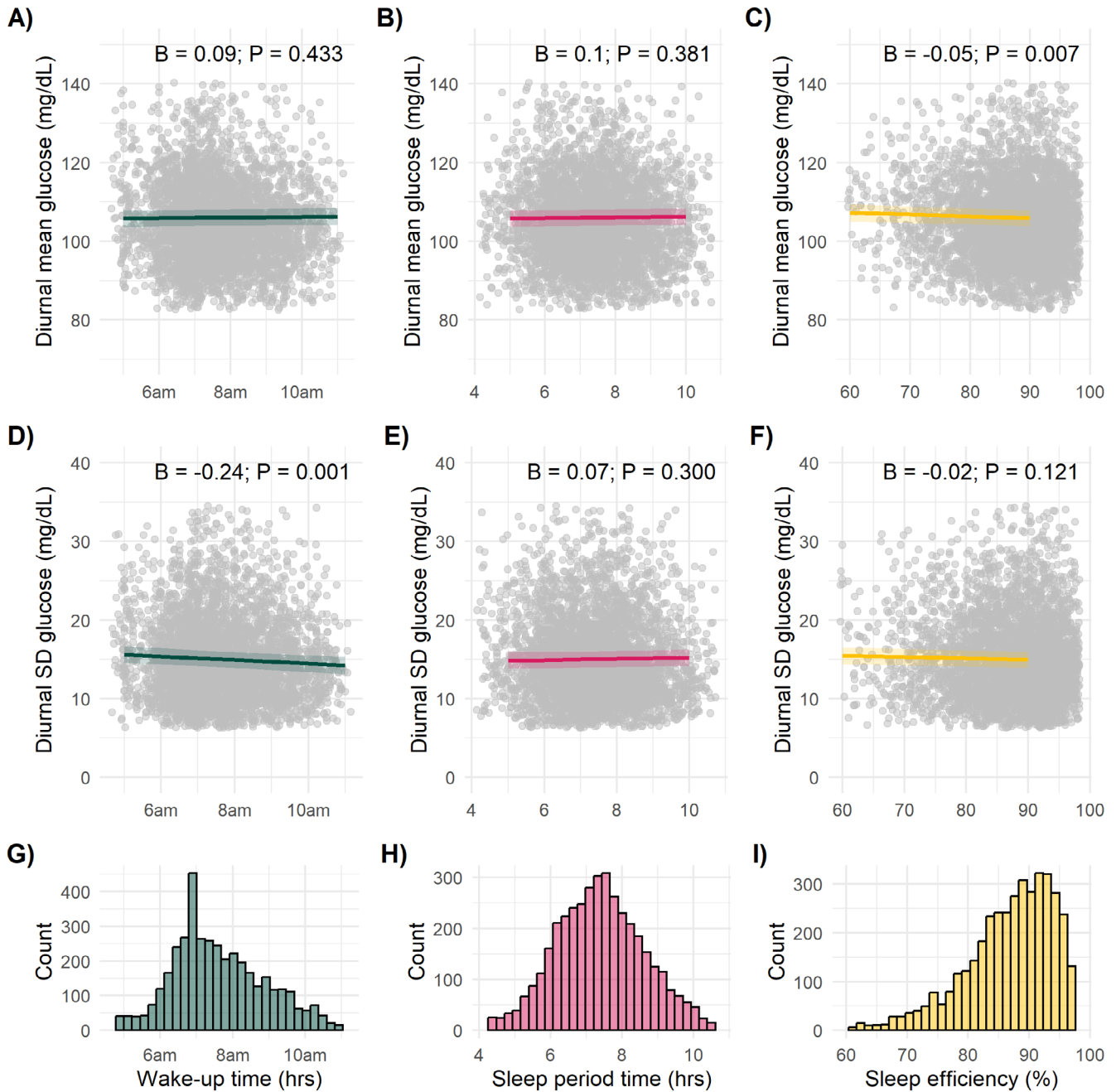
#### 3.2 | Association of Diurnal Glucose Homeostasis With Sleep the Next Night

Then, we investigated whether diurnal glucose levels and variability were associated with sleep characteristics the

**TABLE 1** | Descriptive characteristics of the participants.

	<i>n</i>	All	<i>n</i>	Males	<i>n</i>	Females
<b>Demographics and study cohorts</b>						
Age (years)	388	47.2 (8.7)	194	47.3 (7.3)	194	47.1 (10.0)
Cohort study I, no. (%)		201 (51.8)		100 (49.7)		101 (50.3)
Cohort study II, no. (%)		187 (48.2)		94 (50.3)		93 (49.7)
<b>Anthropometry and body composition</b>						
Body weight (kg)	385	96.9 (14.7)	194	104.0 (13.9)	191	89.7 (11.6)
Height (cm)	385	169.0 (9.4)	194	175.7 (6.9)	191	162.1 (6.0)
Body mass index (kg/m <sup>2</sup> )	385	33.9 (3.7)	194	33.6 (3.5)	191	34.1 (3.9)
Fat-free mass (kg)	385	57.6 (12)	194	66.7 (8.8)	191	48.4 (6.6)
Fat mass (kg)	385	38.8 (8.4)	194	36.7 (8.5)	191	40.9 (7.7)
Fat mass (%)	385	40.4 (7.2)	194	35.3 (5.2)	191	45.6 (4.9)
<b>Sleep</b>						
Sleep onset time (h:min)	361	00:14 (00:51)	179	00:16 (00:52)	182	00:12 (00:50)
Wake-up time (h:min)	361	07:37 (00:50)	179	07:33 (00:49)	182	07:41 (00:52)
Midsleep point (h:min)	361	03:56 (00:46)	179	03:55 (00:47)	182	03:57 (00:46)
Sleep period time (h)	361	7.4 (0.7)	179	7.3 (0.7)	182	7.5 (0.8)
Total sleep time (h)	361	6.4 (0.8)	179	6.2 (0.8)	182	6.6 (0.8)
Wake after sleep onset time (h)	361	1.0 (0.4)	179	1.1 (0.5)	182	0.9 (0.4)
Sleep efficiency (%)	361	87 (6)	179	86 (7)	182	88 (5)
Sleep regularity index (%)	371	52 (10)	182	52 (10)	189	52 (11)
<b>Glucose homeostasis</b>						
Fasting glucose (mg/dL)	385	92 (11)	194	94 (12)	191	91 (11)
Glycated haemoglobin (%)	385	5.4 (0.4)	194	5.4 (0.4)	191	5.4 (0.3)
HOMA-IR	383	2.61 (1.49)	193	2.85 (1.65)	190	2.38 (1.27)
Diurnal mean glucose (mg/dL)	361	105 (10)	179	106 (11)	182	103 (9)
Diurnal median glucose (mg/dL)	361	102 (10)	179	103 (11)	182	100 (9)
Diurnal SD glucose (mg/dL)	361	15 (5)	179	15 (5)	182	15 (5)
Diurnal CV glucose (%)	361	14 (4)	179	14 (4)	182	14 (4)
Diurnal AUC glucose (mg/dL*min)	361	19 900 (9784)	179	21 300 (10 700)	182	18 600 (8598)
Normal glucose homeostasis, no. (%)		163 (42.6)		75 (46.0)		88 (54.0)
Impaired glucose homeostasis, no. (%)		220 (57.4)		118 (53.6)		102 (46.4)
<b>Dietary intake</b>						
Energy intake (kcal/day)	384	1941 (473)	193	2054 (468)	191	1828 (451)
<b>Physical activity</b>						
MVPA (min/day)	368	23.9 (20.9)	174	25.3 (22.5)	174	22.4 (19.1)

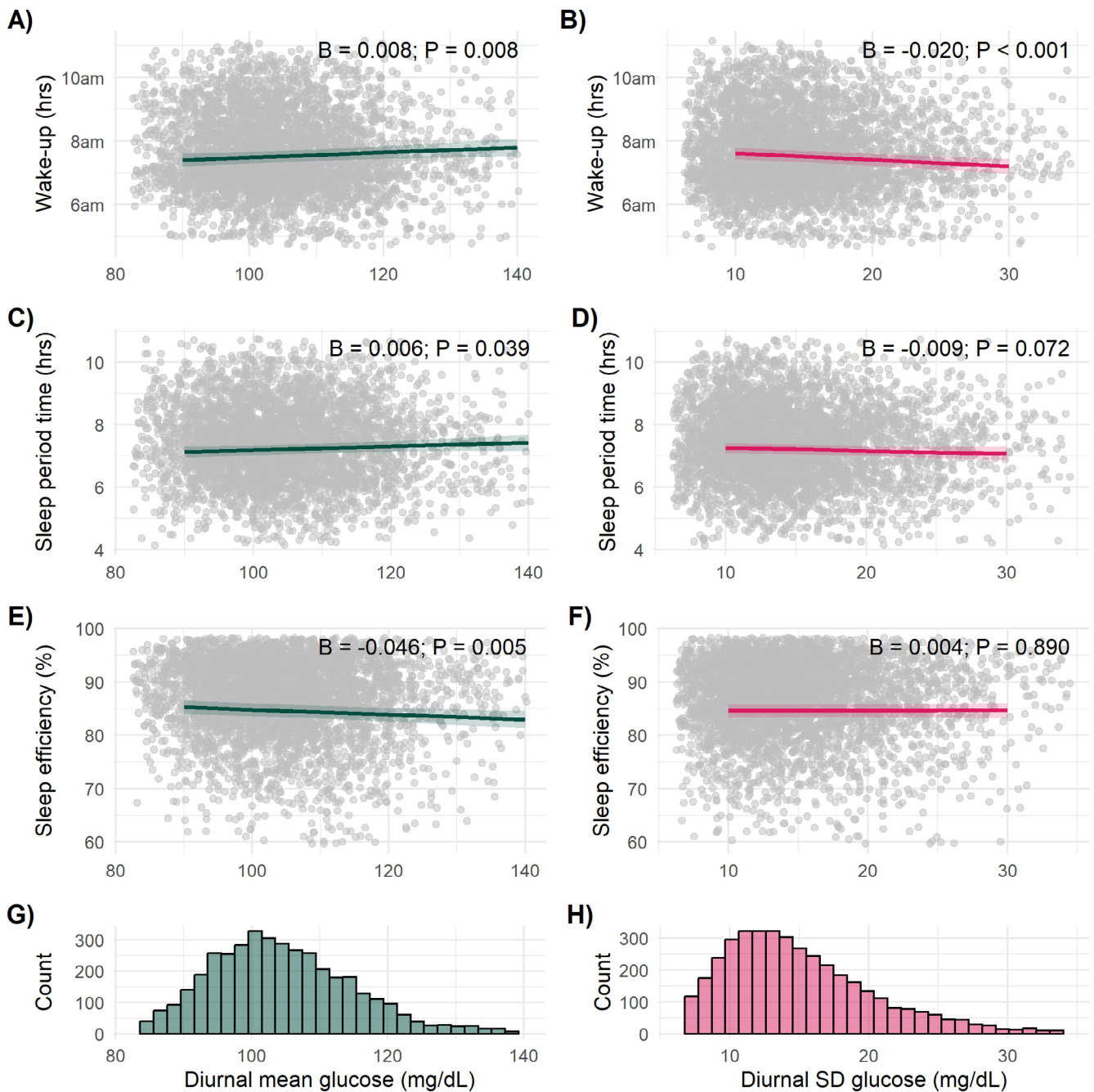
*Note:* Data are presented as mean SD or as number (%) of participants. Sample sizes vary across variables due to differences in data availability (e.g., missing or invalid accelerometry, continuous glucose monitoring, physical activity or clinical measurements). Participants with impaired glucose homeostasis met at least one of the following criteria: fasting plasma glucose levels between  $\geq 100$  and  $\leq 125$  mg/dL, glycated haemoglobin levels ranging from  $\geq 5.7\%$  to  $< 6.5\%$  or insulin resistance, defined as a homeostasis model assessment of insulin resistance (HOMA-IR)  $> 2.5$ . Participants with normal glucose homeostasis did not meet any of these criteria. Abbreviations: AUC, area under the curve; CV, coefficient of variation; MVPA, moderate-to-vigorous physical activity.



**FIGURE 2** | Associations of wake-up time (hours; A and D), sleep period time (hours; B and E) and sleep efficiency (%; C and F) with diurnal mean glucose (mg/dL; A, B and C) and SD of diurnal mean glucose (mg/dL; D, E and F) the following day. Panels G–I show the distributions of valid observations for each sleep variable. Wake-up time reflects sleep timing, sleep period time reflects sleep duration and sleep efficiency reflects sleep continuity. Regression coefficients (B) represent non-standardised fixed-effect estimates from adjusted linear mixed-effects models, indicating the change in diurnal mean glucose or diurnal SD glucose (mg/dL) per 1 h increase in wake-up time or sleep period time and per 1% increase in sleep efficiency. Models were adjusted for age, sex, study location, study cohort, energy intake, moderate-to-vigorous physical activity and sleep regularity index.

subsequent night. Figure 3G,H displays the distributions of valid observations for each diurnal glucose homeostasis variable. Each 1 mg/dL increase in daytime mean glucose was associated with later wake-up time (B [95% CI]=0.008 [0.002, 0.014] h;  $p=0.008$ ), longer sleep period time (B [95% CI]=0.006 [0.000, 0.012] h;  $p=0.039$ ) and lower sleep efficiency (B [95% CI]=−0.05 [−0.08, −0.01] %;  $p=0.005$ ) the

next night (Figure 3A,C,E and Appendix S15–S17). In contrast, each 1 mg/dL increase in glucose variability was associated with earlier wake-up time (B [95% CI]=−0.02 [−0.03, −0.01] h;  $p<0.001$ ; Figure 3B and Appendix S15), while associations with sleep period time and sleep efficiency were not statistically significant (both  $p\geq 0.072$ ; Figure 3D,F and Appendices S16 and S17).



**FIGURE 3** | Association of diurnal mean glucose (mg/dL; A, C and E) and SD of diurnal mean glucose (mg/dL; B, D and F) with wake-up time (hours; A and B), sleep period time (hours; C and D) and sleep efficiency (%) (E and F) the next night. Panels G and H show the distributions of valid observations for each diurnal glucose homeostasis variable. Diurnal mean glucose reflects levels and diurnal SD glucose reflects glycaemic variability. Regression coefficients (B) represent non-standardised fixed-effect estimates from adjusted linear mixed-effects models, indicating the change in wake-up time (hours), sleep period time (hours) or sleep efficiency (%) per 1 mg/dL increase in diurnal mean glucose or diurnal SD glucose. Models were adjusted for age, sex, study location, study cohort, energy intake, moderate-to-vigorous physical activity and sleep regularity index.

We also examined whether the association between daytime glucose levels and sleep outcomes the following night was modified by individual factors. SRI significantly moderated the association between diurnal mean glucose and sleep period time (B [95% CI] =  $-0.00058$  [ $-0.00113$ ,  $-0.00003$ ] h;  $p = 0.038$ ), indicating a more negative association at higher SRI (Appendix S21). No significant moderating effects were observed for sex, age, energy intake, MVPA or glucose regulation status (all  $p \geq 0.059$ ; Appendices S20, S21 and S22).

#### 4 | Discussion

In this day-level analysis conducted under free-living conditions including 3942 person-days, higher sleep efficiency was associated with lower next-day glucose levels in adults at risk of type 2 diabetes. Later wake-up times were linked to lower glucose variability the following day. Conversely, higher daytime glucose levels were temporally associated with later wake-up times, longer sleep period time and reduced sleep efficiency that night,

whereas greater daytime glycaemic variability preceded earlier wake-up times. These findings suggest that specific aspects of sleep quality and timing are temporally associated with short-term fluctuations in glucose levels and variability, suggesting that both sleep characteristics and daytime glycaemia may provide complementary information in the early detection of metabolic dysregulation.

Our findings align with previous cross-sectional studies indicating that good sleep quality is associated with improved glucose homeostasis in adults with overweight or obesity at risk of type 2 diabetes [19, 34]. One cross-sectional study reported that poor sleep quality, assessed subjectively using the Pittsburgh Sleep Quality Index (PSQI), was associated with increased insulin resistance (i.e., higher HOMA-IR) in adults with overweight or obesity [34]. Similarly, low sleep efficiency has been linked to poorer postprandial glycaemic control the following day in healthy adults [19]. However, other studies did not find significant associations between PSQI-derived sleep quality and fasting glucose levels or between day-level accelerometer-derived sleep efficiency and next-day diurnal mean glucose, in adults with overweight or obesity, with or without prediabetes or type 2 diabetes [35–37]. These discrepancies across studies may reflect differences in sample characteristics, reliance on subjective sleep assessments, differences in the operational definition of sleep efficiency (e.g., sleep period time vs. time in bed) which may limit comparability with diary-based studies and may yield higher absolute sleep efficiency estimates and the use of fasting glucose measures rather than CGM-derived metrics, which provide detailed insights into glycaemic variability and glucose levels throughout the day. By leveraging objective day-level sleep and CGM data under free-living conditions, our study helps clarify these mixed findings by showing that higher sleep efficiency is consistently associated with lower next-day glucose levels in adults at elevated cardiometabolic risk. We also found that later wake-up times were associated with lower next-day diurnal glucose variability, an association not previously reported in this population. Although later wake-up times and evening chronotypes have been linked to poorer long-term glycaemic control, our day-level findings suggest that short-term variability in glucose may respond differently, potentially influenced by behavioural factors such as later meal timing, differences in fasting duration, light exposure patterns, physical activity distribution or social schedules that shape daily metabolic response [38]. Additionally, later wake-up times on a given day may reflect recovery from prior sleep restriction or closer alignment with individual circadian preference. These possibilities remain speculative and require further investigation.

We then observed that some of these associations were modified by individual characteristics. Specifically, the relationship of sleep period time and sleep efficiency with next-day mean glucose was more pronounced in males than in females, suggesting that males may be more susceptible to the impact of poor sleep efficiency and duration on glucose homeostasis. This is consistent with prior evidence indicating sex-specific differences in sleep physiology, hormonal regulation and glucose metabolism, which may influence the metabolic response to variations in sleep patterns [39]. Additionally, we observed that the associations between wake-up time and sleep period time with next-day glucose levels were moderated by the SRI, such

that later wake-up times and longer sleep period time were associated with higher daytime glucose levels among individuals with higher sleep regularity. These findings align with evidence suggesting that while greater sleep regularity generally supports metabolic health [40], later wake-up times and extended sleep period time may reflect circadian phase delays, which can impair diurnal glucose regulation despite overall sleep regularity. Furthermore, we observed that higher energy intake appeared to strengthen the association between greater sleep efficiency and lower next-day glucose variability in free-living adults with overweight or obesity. This suggests that maintaining good sleep efficiency may be particularly important for glycaemic stability under higher caloric intake. However, this observation may partly reflect differences in body size or total energy requirements, which should be considered when interpreting the result, as the role of energy intake in moderating the sleep–glycaemic variability relationship remains largely unexplored.

Although previous studies have suggested a potential bidirectional relationship between daily sleep and glucose homeostasis [9], the current evidence is inconclusive. In our study, the reciprocal associations between later wake-up times and greater glucose variability support the notion of a short-term bidirectional interplay between sleep timing and glycaemic stability. Although these temporally structured associations suggest a dynamic interplay, the observational design does not allow conclusions regarding causality and the findings should be interpreted as hypothesis-generating. Moreover, our associations between daytime glucose measures and subsequent sleep characteristics revealed distinct patterns. Higher daytime mean glucose levels were associated with later wake-up times, longer sleep period time and poorer sleep efficiency that night, whereas greater daytime glycaemic variability was associated with earlier wake-up times. These findings align with recent evidence suggesting complex interactions between glycaemic control and sleep. A large meta-analysis and Mendelian randomisation study in >840000 adults with impaired glucose regulation or type 2 diabetes showed that participants with abnormal glucose metabolism exhibited both short and long sleep duration [16]. Moreover, given the associations detected between diurnal glucose homeostasis and subsequent sleep, our findings may suggest that daytime behaviours could influence this relationship, indicating that habits during waking hours might be particularly relevant for improving sleep hygiene. These observations may have implications for future digital health strategies integrating sleep and CGM-derived metrics for individualised monitoring.

From a physiological perspective, reduced sleep efficiency may impair next-day glycaemic control through several mechanisms. Sleep disruption has been associated with alterations in autonomic sympathovagal balance, characterised by increased sympathetic activity and reduced parasympathetic tone, which may impair peripheral glucose uptake and decrease insulin sensitivity [41]. These alterations may also disrupt 24h cortisol rhythms, promoting insulin resistance and impaired glucose tolerance [38, 42]. Conversely, recent evidence suggests that higher glucose levels in the evening may impair sleep quality and nighttime glucose spikes can contribute to sleep fragmentation and disrupt glucose regulation [9]. In addition, misalignment between sleep timing and the endogenous melatonin rhythm may influence nocturnal insulin secretion and

glucose tolerance, as melatonin modulates  $\beta$ -cell function and insulin sensitivity [43]. These mechanisms highlight how elevated glucose levels during the day and specially evening may affect sleep patterns, warranting further investigation under free-living conditions.

This study has several limitations. First, the observational nature of this repeated-measures study precludes causal inference. Second, findings may not be generalizable to older adults, individuals of diverse ethnic backgrounds or those with diagnosed type 2 diabetes. Third, although we accounted for estimated average energy intake, we lacked day-level data on total energy intake and dietary composition, both of which could influence daily sleep-glucose interplay [9]. Fourth, while accelerometers provided objective day-to-day sleep assessments, they do not capture sleep architecture (i.e., time spent in different sleep stages). Further studies using polysomnography can provide additional insights into our results. Fifth, the day-level analytical approach may overrepresent some participants, which could lead to biased estimates of the associations. Moreover, two CGM device models were used in this study, acknowledging that accuracy may vary between systems [44]. However, our analyses focused on individuals with overweight or obesity at risk of type 2 diabetes and relatively stable glucose profiles, a setting in which FreeStyle Libre devices have demonstrated accuracy comparable to other real-time CGM systems [45]. We did not assess day-to-day napping behaviour, which is common in the Spanish population and may affect both glycaemic control and sleep quality. Although napping is often assessed via self-report and may be prone to recall bias and inconsistencies, we cannot exclude that unmeasured daytime sleep may have influenced the observed associations. Although we adjusted for several relevant covariates and modelled within-person day-level variability, residual confounding due to unmeasured time-varying factors (e.g., meal timing, caffeine or alcohol intake, psychological stress or weekday-weekend differences) cannot be excluded. This should be considered when interpreting the modest effect sizes. Moreover, given the relatively large number of statistical tests performed, the risk of type I error should also be considered when interpreting these findings. Finally, several associations were modest in magnitude and should therefore be interpreted with caution. We frame these day-level estimates primarily as indicators of short-term physiological coupling rather than as prescriptive goals based on a single night's change. While the absolute short-term effect may appear negligible, sustained improvements over longer periods could still yield meaningful cumulative benefits and may be relevant for future long-term intervention studies.

## 5 | Conclusions

In adults with overweight or obesity at risk of type 2 diabetes under free-living conditions, higher sleep efficiency was consistently associated with lower next-day glucose levels, while later wake-up times were linked to lower glucose variability. Conversely, higher daytime glucose levels were temporally associated with poorer sleep efficiency and altered sleep timing that night. These findings suggest that daily sleep quality and glycaemic patterns are temporally interconnected in real-world settings. Importantly, the magnitude of these associations differed

according to sex and energy intake, suggesting that individual characteristics may influence sleep-glucose dynamics. Although exploratory and modest in magnitude, these findings reinforce the concept that sleep and daily glycaemic dynamics are interrelated components of metabolic regulation in free-living adults at elevated cardiometabolic risk.

### Author Contributions

Study concept and design: A.C.-J., J.H.M. and J.R.R. Acquisition of data: A.C.-J., J.J.M.-O., A.C.-C., M.M.-F. and M.D.-M. Analysis and interpretation of data: A.C.-J., J.H.M. and J.R.R. Drafting of the manuscript: A.C.-J. Critical revision of the manuscript: A.C.-J., J.J.M.-O., J.H.M., A.C.-C., M.M.-F., M.D.-M., J.M., M.M.-T., I.L. and J.R.R. Statistical analysis: A.C.-J., J.H.M. and J.R.R. Obtained funding: M.M.-T., I.L. and J.R.R. Administrative, technical or material support: M.M.-T., I.L. and J.R.R. and, Study supervision: J.R.R.

### Acknowledgements

Funding for open access charge: Universidad de Granada/CBUA.

### Funding

This study (Project ref. PID2022.141506OB.I00) is funded by the MCIU/AEI/10.13039/501100011033 and by ERDF, EU a way of making Europe to J.R.R.; Junta de Andalucía, Consejería de Transformación económica, Industria, Conocimiento y Universidades (A-CTS-516-UGR20) to J.R.R.; the University of Granada Plan Propio de Investigación-Excellence actions: Unit of Excellence on Exercise Nutrition and Health (UCEENS) to J.R.R.; the Consejo Superior de Deportes, Ministerio de Cultura y Deporte, Plan de Recuperación, Transformación y Resiliencia, Funded by the European Union – NextGenerationEU (EXP\_75091) to I.L.; the Government of Navarra, Departamento de Desarrollo Economico y Empresarial (0011-1365-2021-00070), Plan de Promoción de Grupos de Investigación de la Universidad Pública de Navarra to I.L.; Spanish Ministry of Universities (FPU21/01161 to A.C.-J., FPU22/01631 to J.J.M.-O., FPU23/02158 to M.M.-F., and FPU18/03357 to M.D.M). J.H.M. is supported by grant RYC2024-050453-I funded by MCIN/AEI/10.13039/501100011033. A.C.-C. is supported by the Junta de Andalucía, Consejería de Salud y Consumo (RHJ-0098-2024). J.M. is supported by the Novo Nordisk Foundation grant NNF23SA0084103, the EFSD/Novo Nordisk Foundation Future Leaders Award (no. 0094134) and the European Union (HORIZONEIC-2023-PATHFINDERCHALLENGES-01-101161509). In addition, funding was provided from the EXERNET Research Network on Exercise and Health (DEP2005-00046/ACTI; 09/UPB/19; 45/UPB/20; 27/UPB/21 to I.L. and J.R.R.). Instituto de Salud Carlos III, MCIU/AES and by ERDF, EU a way of making Europe (PI24/01360). This work is part of a doctorate thesis conducted in the Official Doctoral Program in Biomedicine of the University of Granada, Spain. The funders had no role in the design, conduct or interpretation of the study.

### Conflicts of Interest

The authors declare no conflicts of interest.

### Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

### Peer Review

The peer review history for this article is available at <https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/dom.70675>.

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### Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Data S1:** dom70675-sup-0001-Supinfo.docx.