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Sleep well, but be active. Effect of sleep and sedentariness on incidence of diabetes

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ABSTRACT

Aims: We aimed to determine the individual effect of long/short sleep and of inactivity on diabetes risk using data from a population-based prospective study in Switzerland.
Methods: Prospective study with a median (min-max) follow-up of 9 (2.4–11.5) years. Incident diabetes was defined based on 1) fasting plasma glucose (FPG), 2) glycated hemoglobin (HbA1c), or 3) any diagnostic criterion (FPG, HbA_1c or medical diagnosis). Sleep and sedentary levels were assessed by questionnaire. Sleep was categorized into short (<7 h/day), adequate (7–9 h/day) and long (>9 h/day).
Results: Data from 3355 participants (57.6% women, mean age years 56.6 ± 10.3) was analyzed. There were 136, 110 and 142 incident cases of diabetes defined by FPG, HbA_1c or any criterion, respectively. Participants who developed diabetes had a higher sedentariness but no differences were found regarding sleep duration. Similar results were obtained after adjusting for age, gender, education, smoking and body mass index: hazard ratio (95% confidence interval) for sedentariness 1.61 (1.11–2.35), 1.40 (0.93–2.12) and 1.39 (1.04–1.87) for diabetes defined by FPG, HbA_1c or any diagnostic criterion, respectively.
Conclusion: Being sedentary, but not being a long or a short sleeper, increases the risk of developing diabetes.

1. Introduction

Diabetes affects one in 10 people worldwide and is a major health challenge given the continued increase in its prevalence[1]. Hence, it is crucial to understand its modifiable risk factors.

Lack of sleep has become a modern way of life. Numerous studies have shown that both long and short sleepers have a higher incidence of type 2 diabetes (T2DM) [2]. Insufficient sleep can lead to increased insulin resistance and increased risk of diabetes through a variety of pathways, including alterations in glucose metabolism. A J-shaped relationship between sleep duration and HbA_1c levels has also been reported [3]. Conversely, adequate sleep duration and improved sleep quality may help reduce diabetes risk [4].

Adequate physical activity (PA) levels have also been shown to prevent the development of diabetes. Data from 20 cohort studies showed that regular PA can significantly reduce the risk of diabetes [5]. A high level of PA improves insulin sensitivity [6] and is associated with a 20–30% reduction in diabetes risk [5]. Conversely, sedentary behavior may play an important role in the development of T2DM. Among adults with 9 h of sedentary behavior per day, an increase in 1 h of sedentary time per day was associated with a 22% increased risk of diabetes [7].

Whether sleep and sedentary behavior exert the same effect on the risk of diabetes has rarely been assessed jointly. Thus, we aimed to determine the individual effects of long/short sleep and sedentary behavior on diabetes risk using data from a population-based prospective study in Switzerland.

2. Participants and methods

2.1. Participants

The study used data from the CoLaus|PsyCoLaus study (www.colaus-psycolaus.ch), a prospective study conducted in the population of Lausanne, Switzerland. Recruitment began in 2003 and ended in 2006 and included 6733 participants. The first follow-up (FU) was performed between 2009 and 2012 and included 5064 of the initial participants (75.2%). The second FU was performed between 2014 and 2017 and included 4881 of the initial participants (72.5%). The third FU was

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performed between 2018 and 2021 and included 3751 of the initial participants (55.7%). Supplementary figure 1 provides the flowchart of the entire study. Median follow-up time was 5.4 (range 4.5–8.8) years for the first, 10.7 (range 8.8–13.6) years for the second, and 14.5 (range 13.2–17.3) for the third FU. As no sleep or PA data was assessed at baseline, for this study, data from the first FU were used.

2.2. Assessment of T2DM

Within each follow-up, participants attended a clinical examination that included self-reported and administered questionnaires, blood drawing, and anthropometric measurements. Biological assays were performed by the CHUV Clinical Laboratory on fresh blood samples within 2 h of blood collection. Fasting plasma glucose was assessed by glucose hexokinase and HbA_1c levels were measured by high performance liquid chromatography. Diabetes status was defined as a presence of antidiabetic treatment and/or 1) a fasting blood glucose $\geq$ 7 mmol/L (definition 1); 2) a HbA_1c level $\geq$ 48 mmol/mol (6.5%, definition 2) or 3) definition 1 or definition 2 or a positive answer to the question "did a doctor ever told you that you had diabetes?".

2.3. Sedentariness and sleep levels

PA and sleep levels were subjectively assessed in the first FU using the Physical Activity Frequency Questionnaire (PAFQ). The PAFQ has been validated in the population of Geneva [8] and assesses the type and duration of 70 kinds of (non)professional activities and sports during the previous week. Sedentary status was defined as spending more than 90% of the daily energy in activities below moderate- and high-intensity (defined as requiring at least 4 times the basal metabolic rate, BMR [9]) BMR multiples are close to Metabolic Equivalent of Task (MET) multiples, although MET multiples do not take into account participant sex, age or height.

In a separate questionnaire, participants indicated the hour of going to bed and the hour of waking up, which enabled the calculation of sleep time. Sleep levels were categorized into short (<7 h/night), adequate (7–9 h/night) and long (>9 h/night). A second definition of short (<6 h/night), adequate (6–8 h/night) and long (>8 h/night) was applied.

2.4. Other covariates

Socio-economic and lifestyle data was collected by questionnaire. Education was categorized as mandatory education, apprenticeship, high school, and university education. Smoking was categorized as never, former and current. Medications were collected and classified according to the WHO ATC criteria. Alcohol consumption was categorized as drinker / nondrinker.

Body weight and height were measured with participants barefoot and in light indoor clothes. Body weight was measured in kilograms to the nearest 100 g using a Seca® scale (Hamburg, Germany). Height was measured to the nearest 5 mm using a Seca® (Hamburg, Germany) height gauge. Body mass index (BMI) was calculated and categorized into normal (BMI<25 kg/m^2), overweight (25–29.9 kg/m^2) and obesity (BMI$\geq$30 kg/m^2).

Blood pressure (BP) was measured using an Omron® HEM-907 automated oscillometric sphygmomanometer after at least a 10-minute rest in a seated position, and the average of the last two measurements was used. Hypertension was defined by a SBP $\geq$ 140 mm Hg or a DBP $\geq$ 90 mm Hg or presence of antihypertensive drug treatment.

2.5. Exclusion criteria

Participants were excluded if: 1) they presented with diabetes at baseline; 2) they had no follow-up data and 3) they missed the covariates needed for adjustment.

2.6. Statistical analyses

Statistical analysis was conducted using Stata version 17.0 (Stata Corp, College Station, TX, USA). Summary statistics are reported as mean $\pm$ standard deviation for continuous variables and as number of participants (percentage) for categorical variables. Bivariate between-group comparisons were performed using t-test for continuous variables and chi-square for categorical variables.

As it was not possible to precisely assess the date of T2DM occurrence, interval-censored survival-time analysis with a Weibull survival distribution was used to determine the association between incidence of T2DM with PA and sleep levels. We used the last available date for participants who did not develop diabetes, and an interval where the lower bound was the last date where no diabetes was diagnosed, and the upper bound the date where the diagnosis of diabetes was established. For example, for a participant developing diabetes between the baseline and the first follow-up, the lower bound was set to zero and the upper bound was defined as the date of examination at the first follow-up; for a participant developing diabetes between the first and the second follow-up, the lower and upper bounds were defined as the dates of the first and the second follow-ups, respectively. Results were expressed as hazard ratio (HR) and 95% confidence intervals (CIs). Three models were applied: using the two categorizations of sleep and using sleep duration as a continuous variable (in hours). Covariates adjusted for multivariate models included baseline age, sex (male or female), smoking status (never, former, current), educational level (four categories), alcohol consumption (yes, no), and BMI categories (normal, overweight, obese).

A first sensitivity analysis was carried using inverse probability weighting to take into account excluded participants. Briefly, the probability of inclusion was modelled using a logistic regression based on age, sex, smoking status, educational level, alcohol consumption and BMI categories, and the inverse of the probability was used as weight. As BMI could lie in the causal pathway between sleep and T2DM, we also performed analyses stratifying on BMI, using both unweighted and weighted models. Statistical significance was considered for a two-sided test with $p < 0.05$.

3. Results

3.1. Characteristics of the participants

Of the initial 5064 participants, 3355 (66%) were included in the analysis. The reasons for exclusion are indicated in Fig. 1 and the characteristics of the included and excluded participants are provided in supplementary table 1). Excluded participants were older, with a higher BMI, more frequently men, obese, with mandatory education, and current smokers.

3.2. Association between sleep, sedentariness and incidence of diabetes

During a median (min-max) follow-up of 9 (2.4–11.5) years, 136, 110 and 214 participants developed diabetes as defined by FPG, HbA_1c or any criterion, respectively. The characteristics of the participants who developed diabetes according to the definition criterion are provided in Table 1. Irrespective of the criterion applied, participants who developed diabetes were older, more frequently men, with a higher BMI, more frequently obese and less well educated than participants who did not develop diabetes.

Participants who developed diabetes had a higher prevalence of sedentariness, while no difference was found regarding sleep duration categories or duration (Table 2).

The multivariable analysis of the associations between sleep time, sedentariness and incidence of T2DM are indicated in Table 3. Irrespective of the definition of T2DM or the categorization of sleep duration, being sedentary was associated with a higher likelihood of developing diabetes, while no association was found with sleep

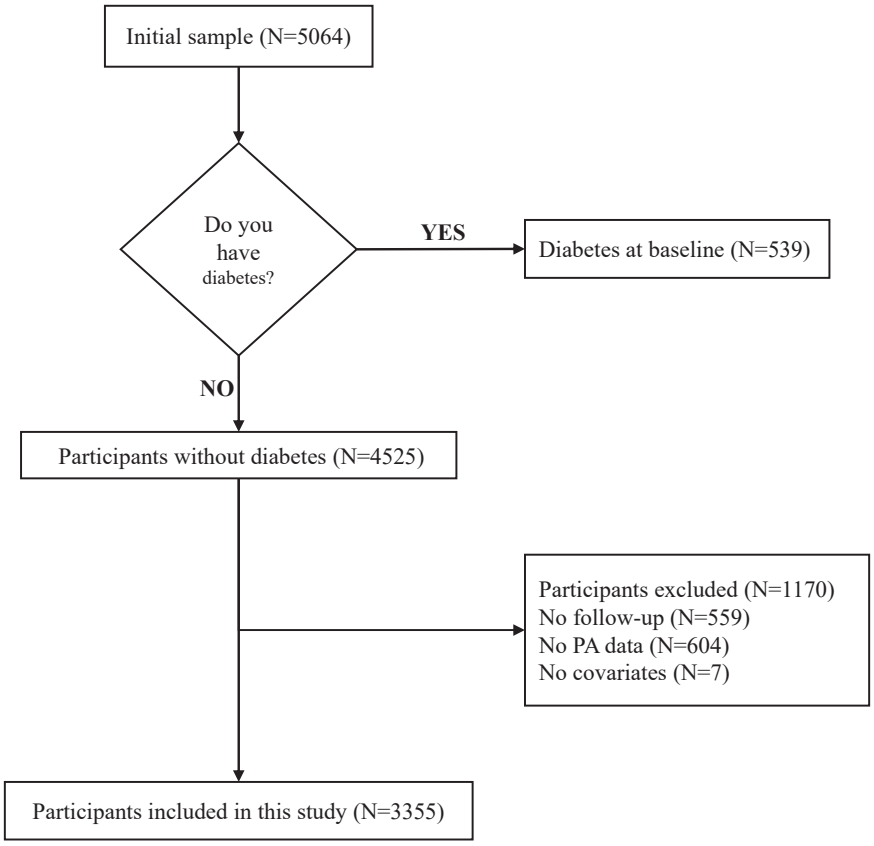

Fig. 1. selection procedure, CoLaus|PsyCoLaus study, Lausanne, Switzerland.

Table 1
Clinical characteristics of participants who developed diabetes according to the definition applied, CoLaus|PsyCoLaus study, Lausanne, Switzerland.

	Incident diabetes by FPG			Incident diabetes by HbA_1c			Incident diabetes, any criterion		
	No	Yes	P value	No	Yes	P value	No	Yes	P value
Sample size	3209	136		3235	110		3141	214	
Woman (%)	1870 (58.3)	58 (42.7)	< 0.001	1878 (58.1)	50 (45.5)	0.009	1832 (58.3)	100 (46.7)	0.001
Age (years)	56.6 ± 10.3	58.5 ± 9.4	< 0.001	56.6 ± 10.3	59.1 ± 9.5	0.010	56.5 ± 10.3	58.7 ± 9.7	0.003
BMI (kg/m^2)	25.4 ± 4.1	29.2 ± 4.7	< 0.001	25.4 ± 4.1	29.4 ± 4.8	< 0.001	25.3 ± 4.1	28.4 ± 4.8	< 0.001
BMI categories (%)									
Normal	1632 (50.9)	20 (14.7)	< 0.001	1638 (50.6)	14 (12.7)	< 0.001	1611 (51.3)	46 (21.5)	0.036
Overweight	1193 (37.2)	69 (50.7)		1260 (37.3)	56 (50.9)		1161 (37.0)	104 (48.6)	
Obese	384 (12.0)	47 (34.6)		391 (12.1)	40 (36.4)		369 (11.8)	64 (29.9)	
Educational level (%)									
University education.	771 (24.0)	20 (14.7)	0.002	773 (23.9)	18 (16.4)	0.004	754 (24.0)	40 (18.7)	0.046
High school	877 (27.3)	28 (20.6)		886 (27.4)	19 (17.3)		857 (27.3)	49 (22.9)	
Apprenticeship	1123 (35.0)	59 (43.4)		1130 (34.9)	52 (47.3)		1099 (35.0)	87 (40.7)	
Mandatory education	437 (13.6)	29 (21.3)		445 (13.8)	21 (19.1)		430 (13.7)	38 (17.8)	
Smoking status (%)									
Never	1393 (43.4)	47 (34.6)	0.120	1406 (43.5)	34 (30.9)	0.030	1369 (43.6)	74 (34.6)	0.036
Former	1187 (37.0)	57 (41.9)		1196 (37.0)	48 (43.6)		1156 (36.8)	92 (43.0)	
Current	629 (19.6)	32 (23.5)		633 (19.6)	28 (25.5)		616 (19.6)	48 (22.4)	
Family history (%)	653 (20.4)	39 (28.7)	0.019	662 (20.5)	30 (27.3)	0.083	639 (20.3)	56 (26.2)	0.042
Alcohol drinker (%)	2502 (78.0)	101 (74.3)	0.309	2524 (78.0)	79 (71.8)	0.124	2450 (78.0)	160 (74.8)	0.271

Results are expressed as number of participants (column percentage) for categorical variables and as average ± standard deviation for continuous variables. Between group comparisons performed using chi-square for categorical variables and student's t-test for continuous variables.

duration. Similar findings were obtained after inverse probability weighting (supplementary table 2), although the association between sedentary status and incident T2DM was no longer significant.

The results of the analysis after stratification on BMI are provided in supplementary tables 3 to 5 for normal, overweight, and obese categories, respectively, for the unweighted model and in supplementary tables 6 to 8 for normal, overweight, and obese categories, respectively, for the inverse probability weighted model. In all cases, the hazard ratios for sedentary behavior were higher than unity, while the hazard ratios for sleep duration were close to or below unity. No statistically significant result was found, due to the reduction in sample size by the stratification.

4. Discussion

In this population-based, prospective study, being sedentary was

Table 2
Sedentariness and sleep levels of participants who developed diabetes according to the definition applied, CoLaus|PsyCoLaus study, Lausanne, Switzerland.

	Incident diabetes by FPG			Incident diabetes by HbA_1c			Incident diabetes, any criterion		
	No	Yes	P value	No	Yes	P value	No	Yes	P value
Sample size	3209	136		3235	110		3141	214	
Sleep duration (%)									
< 7 h	465 (14.5)	18 (13.2)	0.863	465 (14.4)	18 (16.4)	0.845	454 (14.5)	29 (13.6)	0.890
7–9 h	2074 (64.7)	91 (66.9)		2095 (64.9)	70 (63.6)		2032 (64.8)	142 (66.4)	
> 9 h	665 (20.8)	27 (19.9)		670 (20.7)	22 (20.0)		650 (20.7)	43 (20.1)	
Sleep duration (%)			0.513			0.549			0.767
< 6 h	89 (2.8)	6 (4.4)		90 (2.8)	5 (4.6)		89 (2.8)	6 (2.8)	
6–8 h	1380 (43.1)	56 (41.2)		1390 (43.0)	46 (41.8)		1342 (42.8)	97 (45.3)	
> 8 h	1735 (54.2)	74 (54.4)		1750 (54.2)	59 (53.6)		1705 (54.4)	111 (51.9)	
Sleep duration (min)	480 [430–510]	480 [420–510]	0.871	480 [430–510]	480 [420–510]	1.000	480 [430–510]	480 [420–510]	0.626
Sedentary (%)	1766 (55.0)	94 (69.1)	0.001	1786 (55.2)	74 (67.3)	0.012	1728 (55.0)	141 (65.9)	0.002

Results are expressed as number of participants (column percentage) for categorical variables and as median [interquartile range] for continuous variables. Between group comparisons performed using chi-square for categorical variables and Kruskal-Wallis t-test for continuous variables

Table 3
Association between sleep duration and sedentary behavior on incidence of diabetes, CoLaus|PsyCoLaus study, Lausanne, Switzerland.

	Definition[a]	P value	Definition[b]	P value	Definition[c]	P value
Model 1						
Sleep duration (hours)						
< 7 h	0.72 (0.43–1.21)	0.218	0.99 (0.58–1.70)	0.980	0.79 (0.52–1.20)	0.269
7–9 h	reference		reference		reference	
> 9 h	0.79 (0.51–1.24)	0.309	0.79 (0.48–1.30)	0.351	0.80 (0.56–1.14)	0.220
Sedentary (yes vs. no)	1.61 (1.11–2.35)	0.013	1.40 (0.93–2.12)	0.109	1.39 (1.04–1.87)	0.027
Model 2						
Sleep duration (hours)						
< 6 h	1.32 (0.56–3.07)	0.525	1.31 (0.52–3.31)	0.568	0.79 (0.34–1.80)	0.570
6–8 h	reference		reference		reference	
> 8 h	1.05 (0.72–1.54)	0.791	0.95 (0.62–1.45)	0.809	0.86 (0.63–1.16)	0.311
Sedentary (yes vs. no)	1.60 (1.10–2.34)	0.014	1.39 (0.92–2.11)	0.116	1.40 (1.04–1.87)	0.026
Model 3						
Sleep (per 20-min)	1.00 (0.95–1.05)	0.882	1.00 (0.94–1.05)	0.885	1.00 (0.96–1.04)	0.819
Sedentary (yes vs. no)	1.61 (1.10–2.35)	0.013	1.39 (0.92–2.11)	0.115	1.39 (1.04–1.86)	0.028

Results expressed as hazard ratio and (95% confidence interval). For diabetes defined by [a]: fasting plasma glucose; [b]: HbA_1c, [c] any criterion. All models adjusted for age, gender, education, BMI, alcohol consumption and family history of diabetes.

significantly related to the incidence of diabetes, but no obvious association was found between sleep duration and incidence of diabetes.

4.1. Association between sleep, sedentariness and incidence of diabetes

So far, many studies have shown that adequate physical activity can prevent diabetes, namely by improving insulin sensitivity [10]. On the opposite, sedentary behavior is strongly associated with diabetes risk, and those who are sedentary have more than double the risk of developing diabetes compared with those who are not sedentary [11]. Increasing the amount of activity from 11.3 to 22.5 MET hours/week reduces the risk of type 2 diabetes by 10%, and reduce the total risk by 36% compared with inactivity [12]. Adequate physical activity increases the expression of brain-derived neurotrophic factor (BDNF), which is beneficial in the management of T2DM. Interrupting sedentary behavior, especially sedentary behavior for more than 15 min, can increase the plasma abundance of BDNF, which has a non-negligible impact on metabolic and cognitive functions in patients with T2DM [13].

In our study, sedentary behavior was associated with an increased incidence of diabetes. This finding is also consistent with that of Proper et al. who found modest evidence of an association between sedentary behaviour and diabetes in a systematic review of prospective studies [14]. In fact, current guidelines recommend moving as often as possible, or interrupting sedentary periods with bouts of vigorous activity at least every 30 min, as this plays a key role in blood sugar management in diabetics [15].

Some scholars have found that people who sleep for 7–8 h at night have the lowest incidence of T2DM [2]. Both long sleep and short sleep increase the risk of T2DM, the association between sleep time and the incidence of T2DM presenting a U-shaped relationship [16,17]. This may be related to the fact that sleep restriction increases the activity of the sympathetic nervous system, leading to decreased insulin sensitivity [18]. No significant association was found between sleep duration and diabetes in our study. Our findings do not replicate those of previous studies [16,17]. The reason for the inconsistency of the results may be caused by the different classifications of sleep duration, or the difference in living habits caused by different groups of people. However, the mechanism of the increased risk of T2DM caused by long sleep is still not completely clear which still needs further discussion and analysis.

4.2. Implications for clinical practice

The number of adults with diabetes is predicted to increase to 783 million worldwide in 2045 [19]. In Switzerland, it has been estimated that over 1.5 million people present with diabetes or impaired glucose tolerance. Both the European guidelines [19,20] and the Swiss clinical recommendations [21] advise reducing the amount of sedentary time among people with (pre)diabetes, but no information regarding sleep duration have been issued.

Our study suggests that being sedentary is related to the incidence of diabetes, but no association was found between sleep duration and incidence of diabetes. Thus, patients at risk of diabetes should be urged to exercise, while allowed to sleep adequately, as inadequate sleep duration has been associated with cardiovascular disease [22–24].

4.3. Strengths and limitations

Our study combined sedentary time and sleep time. It is also the first study of the relationship between diabetes, physical activity and sleep duration in the Swiss general population. The follow-up time was 9 years, thus allowing a medium-term estimation of the risk of diabetes.

However, our research still has some limitations. For example, although our questionnaire has been validated, we are unsure whether the participants recorded their sleep time accurately. Still, most studies assessing the association between sleep duration and incidence of T2DM used questionnaires to assess sleep duration [16,25]. Secondly, our sample size may be too small to detect the associations between sleep and incidence of T2DM; indeed, most studies that found significant associations included over 15,000 participants [16,25]. Thirdly, the reporting of diagnosed T2DM by the participants might be subjected to a reporting bias; still, the results were similar to those using objective measurements such as fasting blood glucose or HbA_1c. Fourthly, compared to the initial cohort, the studied sample was more educated, with a lower prevalence of current smokers, with a higher mean BMI, and with lower obesity levels (supplementary table 9). Still, this finding is in agreement with the literature, participants with lower educational level, smokers or with comorbidities presenting higher drop-out rates [26,27]and it has been suggested that differential loss to follow-up rarely affects estimates of association[28,29]. Finally, we could not adjust for all potential risk factors of T2DM; hence, it is possible that some residual confounding might persist.

5. Conclusion

Being sedentary, but not being a long or a short sleeper, is related to a higher risk of developing diabetes.

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Ethical statement

The institutional Ethics Committee of the University of Lausanne, which afterwards became the Ethics Commission of Canton Vaud (www.cer-vd.ch) approved the baseline CoLaus study (reference 16/03). The approval was renewed for the first (reference 33/09), the second (reference 26/14) and the third (reference PB_2018–00040) follow-ups. The approval for the entire CoLaus|PsyCoLaus study was confirmed in 2021 (reference PB_2018–00038, 239/09). The full decisions of the CER-VD can be obtained from the authors upon request. The study was performed in agreement with the Helsinki declaration and its former amendments, and in accordance with the applicable Swiss legislation. All participants gave their signed informed consent before entering the study.

CRediT authorship contribution statement

Keyuan Liu: investigation, visualization, formal analysis, writing – original draft. Pedro Marques-Vidal: conceptualization, methodology, data curation; validation, writing – review & editing, supervision. All authors have approved the final article.

Declaration of Competing Interest

The authors declare no conflict of interest.

Data Avaiability

The data of CoLaus|PsyCoLaus study used in this article cannot be fully shared as they contain potentially sensitive personal information on participants. According to the Ethics Committee for Research of the Canton of Vaud, sharing these data would be a violation of the Swiss legislation with respect to privacy protection. However, coded individual-level data that do not allow researchers to identify participants are available upon request to researchers who meet the criteria for data sharing of the CoLaus|PsyCoLaus Datacenter (CHUV, Lausanne, Switzerland). Any researcher affiliated to a public or private research institution who complies with the CoLaus|PsyCoLaus standards can submit *a* research application to research.colaus@chuv.ch or research.psycolaus@chuv.ch. Proposals requiring baseline data only, will be evaluated by the baseline (local) Scientific Committee (SC) of the CoLaus and PsyCoLaus studies. Proposals requiring follow-up data will be evaluated by the follow-up (multicentric) SC of the CoLaus|PsyCoLaus cohort study. Detailed instructions for gaining access to the CoLaus|PsyCoLaus data used in this study are available at www.colaus-psycolaus.ch/professionals/how-to-collaborate/.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.pcd.2023.08.002.

References

[1] W H Organization, World Health Organization Global Report on Diabetes, World Health Organization, Geneva, 2016.
[2] J. Wang, M.K. Kwok, S.L. Au Yeung, A.M. Li, et al., Sleep duration and risk of diabetes: observational and Mendelian randomization studies, Prev. Med 119 (2019) 24–30.
[3] T. Ohkuma, H. Fujii, M. Iwase, Y. Kikuchi, et al., Impact of sleep duration on obesity and the glycemic level in patients with type 2 diabetes: the Fukuoka Diabetes Registry, Diabetes Care 36 (3) (2013) 611–617.
[4] S. Reutrakul, E. Van Cauter, Sleep influences on obesity, insulin resistance, and risk of type 2 diabetes, Metabolism 84 (2018) 56–66.
[5] J.M. Gill, A.R. Cooper, Physical activity and prevention of type 2 diabetes mellitus, Sports Med 38 (10) (2008) 807–824.
[6] K. Karstoft, B.K. Pedersen, Exercise and type 2 diabetes: focus on metabolism and inflammation, Immunol. Cell Biol. 94 (2) (2016) 146–150.
[7] J.D. Van Der Berg, C.D. Stehouwer, H. Bosma, J.H. Van Der Velde, et al., Associations of total amount and patterns of sedentary behaviour with type 2 diabetes and the metabolic syndrome: the Maastricht study, Diabetologia 59 (4) (2016) 709–718.
[8] M. Bernstein, D. Sloutskis, S. Kumanyika, A. Sparti, et al., Data-based approach for developing a physical activity frequency questionnaire, Am. J. Epidemiol. 147 (2) (1998) 147–154.
[9] M.S. Bernstein, A. Morabia, D. Sloutskis, Definition and prevalence of sedentarism in an urban population, Am. J. Public Health 89 (6) (1999) 862–867.
[10] J.F. Burr, C.P. Rowan, V.K. Jamnik, M.C. Riddell, The role of physical activity in type 2 diabetes prevention: physiological and practical perspectives, Phys. Sport. 38 (1) (2010) 72–82.
[11] E.G. Wilmot, C.L. Edwardson, F.A. Achana, M.J. Davies, et al., Sedentary time in adults and the association with diabetes, cardiovascular disease and death: systematic review and meta-analysis, Diabetologia 55 (11) (2012) 2895–2905.
[12] J.P. Kirwan, J. Sacks, S. Nieuwoudt, The essential role of exercise in the management of type 2 diabetes, Cleve Clin. J. Med 84 (7 Suppl 1) (2017) S15–s21.
[13] P.B. Júdice, J.P. Magalhães, M. Hetherington-Rauth, I.R. Correia, et al., Sedentary patterns are associated with BDNF in patients with type 2 diabetes mellitus, Eur. J. Appl. Physiol. 121 (3) (2021) 871–879.
[14] K.I. Proper, A.S. Singh, W. Van Mechelen, M.J. Chinapaw, Sedentary behaviors and health outcomes among adults: a systematic review of prospective studies, Am. J. Prev. Med 40 (2) (2011) 174–182.
[15] P. Sgrò, G.P. Emerenziani, C. Antinozzi, M. Sacchetti, et al., Exercise as a drug for glucose management and prevention in type 2 diabetes mellitus, Curr. Opin. Pharm. 59 (2021) 95–102.
[16] H. Liu, G. Chen, J. Wen, A. Wang, et al., Association between sleep duration and incidence of type 2 diabetes in China: the REACTION study, Chin. Med J. (Engl.) 135 (10) (2022) 1242–1248.

[17] Z. Shan, H. Ma, M. Xie, P. Yan, et al., Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies, Diabetes Care 38 (3) (2015) 529–537.
[18] S. Reutrakul, B. Mokhlesi, Obstructive sleep apnea and diabetes: a state of the art review, Chest 152 (5) (2017) 1070–1086.
[19] H. Sun, P. Saeedi, S. Karuranga, M. Pinkepank, et al., IDF diabetes atlas: global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045, Diabetes Res Clin. Pr. 183 (2022), 109119.
[20] F. Cosentino, P.J. Grant, V. Aboyans, C.J. Bailey, et al., 2019 ESC guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD, Eur. Heart J. 41 (2) (2020) 255–323.
[21] G. Gastaldi, B. Lucchini, S. Thalmann, S. Alder, et al., Swiss recommendations of the Society for Endocrinology and Diabetes (SGED/SSED) for the treatment of type 2 diabetes mellitus (2023), Swiss Med Wkly 153 (2023) 40060.
[22] C. Krittanawong, A. Tunhasiriwet, Z. Wang, H. Zhang, et al., Association between short and long sleep durations and cardiovascular outcomes: a systematic review and meta-analysis, Eur. Heart J. Acute Cardiovasc Care 8 (8) (2019) 762–770.
[23] P.R. Pienaar, T.L. Kolbe-Alexander, W. Van Mechelen, C.R.L. Boot, et al., Associations between self-reported sleep duration and mortality in employed individuals: systematic review and meta-analysis, Am. J. Health Promot 35 (6) (2021) 853–865.
[24] C.S. Kwok, E. Kontopantelis, G. Kuligowski, M. Gray, et al., Self-reported sleep duration and quality and cardiovascular disease and mortality: a dose-response meta-analysis, J. Am. Heart Assoc. 7 (15) (2018), e008552.
[25] I.H. Wu, N. Heredia, Q. Dong, L.H. Mcneill, et al., Sleep duration and type 2 diabetes risk: a prospective study in a population-based Mexican American cohort, Sleep. Health 7 (2) (2021) 168–176.
[26] C. Canivet, A. Nilsson, J. Björk, M. Moghaddassi, et al., Assessment of selection bias due to dropouts in the follow-up of the Scania Public Health Cohort, Scand. J. Public Health 49 (4) (2021) 457–464.
[27] N. Saiepour, R. Ware, J. Najman, P. Baker, et al., Do participants with different patterns of loss to follow-up have different characteristics? A multi-wave longitudinal study, J. Epidemiol. 26 (1) (2016) 45–49.
[28] N. Saiepour, J.M. Najman, R. Ware, P. Baker, et al., Does attrition affect estimates of association: a longitudinal study, J. Psychiatr. Res 110 (2019) 127–142.
[29] J. Powers, M. Tavener, A. Graves, D. Loxton, Loss to follow-up was used to estimate bias in a longitudinal study: a new approach, J. Clin. Epidemiol. 68 (8) (2015) 870–876.