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Poor self-reported sleep is associated with risk factors for cardiovascular disease: A cross-sectional analysis in half a million adults

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Abstract

Background: Sleep is known to affect cardiovascular health, but some controversy exists on the independent association between different sleep characteristics (duration, restfulness, difficulties falling asleep) and specific risk factors for cardiovascular disease (CVD). We aimed to assess the association between self-reported sleep characteristics and the likelihood of major CVD risk factors.

Methods: Totally, 521,364 Spanish workers (32% female, 44 ± 9 years [18–64]) insured by an occupational risk prevention company participated in this nationwide cross-sectional study. Participants' sleep was considered 'poor' if they reported having ≥1 of the following conditions: excessively short (<6 h/d) or long (>9 h/d) sleep, unrestful sleep, or difficulties to fall asleep. We assessed the independent association between aforementioned sleep characteristics and the prevalence of hypertension, diabetes, hypercholesterolaemia, obesity and physical inactivity.

Results: Poor sleep (reported by 33% of participants) was associated with a higher likelihood of presenting all CVD risk factors individually, particularly physical inactivity (which prevalence was ~3-fold higher in the poor sleep group compared with participants reporting no sleep abnormality). In separate analyses, all the different sleep characteristics were associated with the likelihood of ≥2 CVD risk factors. Participants with optimal sleep, normal sleep duration, no difficulties falling sleep and restful sleep showed a lower total CVD risk score than their peers with poor sleep, short sleep duration, difficulties falling sleep and unrestful sleep, respectively (all $p < .001$).

Pedro L. Valenzuela and Alejandro Santos-Lozano contributed equally to this study.

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Conclusions: Poor sleep was associated with a higher likelihood of presenting major CVD risk factors. These findings might support the importance of monitoring and improving sleep patterns for primary CVD prevention.

KEYWORDS
diabetes, hypercholesterolaemia, hypertension, obesity, physical activity

1 | INTRODUCTION

Western societies are currently exposed to numerous sleep disruptors such as shift work, multiple jobs, 24 h access to shopping, internet, television, smartphones or artificial light. Sleep disturbances might have a negative impact on health status, particularly with regard to cardiovascular diseases (CVD).[1-3] There is indeed meta-analytical evidence that short sleep duration is associated with a higher risk of stroke and coronary heart disease (CHD).[4] Although controversy exists,[5] growing evidence suggests that excessive sleep duration might also be associated with an impaired cardiovascular health, with recent evidence for a J-shaped relationship between sleep duration and CVD events – with sleep durations above 8 h associated with a greater CVD and mortality risk.[6]

Cardiovascular health might also be affected by sleep quality – that is, the self-reported, retrospective appraisal of the sleep experience. The association between short sleep duration and incidence of CVD events is more marked in those individuals reporting poor sleep quality (*i.e.*, having a restless sleep).[7] A study with 60,586 participants found an inverse association between self-reported sleep quality (including difficulties to fall asleep) and CHD incidence.[8] A recent meta-analysis indicated that subjective sleep quality was inversely associated with CHD incidence; although as opposed to abnormal sleep duration, no significant associations were reported for mortality or other CVD outcomes (*e.g.*, stroke, overall mortality).[6]

Besides the documented link between sleep and CVD or CVD-related outcomes, an association has also been observed for some specific risk factors for CVD, notably a link between short and/or long sleep duration and diabetes,[9] hypertension[10] or obesity,[11,12] respectively, or between poor sleep quality and a worse lipid profile.[13] However, some questions remain open – for instance, the mediating role of age, with the deleterious association between short sleep duration and CVD risk factors such as obesity or hypertension potentially more marked in younger individuals.[14-16] There is also some controversy, with mixed results reported for the association between sleep duration and CVD risk factors – for instance, a reverse J-shaped relationship has been reported between sleep duration and risk of obesity, with the highest risk observed for short sleep duration, the lowest for 7–8 h and no differences for longer durations.[12] In turn, other authors have found long sleep to be associated with obesity and also with diabetes,[17] but not with hypertension.[5,17] On the other hand, the association between sleep and a given CVD risk factor might be confounded by the coexistence of other CVD risk factors since all these factors can interact with each other.[18-21]

The present study aimed to assess the independent association between different sleep characteristics (duration, quality and easiness to fall asleep), on the one hand, and the likelihood of presenting individual CVD risk factors (hypertension, diabetes, hypercholesterolaemia, obesity and physical inactivity) and/or a higher overall CVD risk (i.e., CVD risk score), on the other.

2 | METHODS

2.1 | Experimental design and participants

The present study followed a cross-sectional observational design and conforms to broad Enhancing the QUAlity and Transparency Of health Research guidelines.[22] We followed the 'Strengthening the Reporting of Observational Studies in Epidemiology' guidelines.

A cohort of Spanish workers (18–64 years) from all over the country insured by a large occupational risk prevention company (*Quirónprevención*) participated in the study. From 2012 to 2016, participants underwent routine (~1 per year) medical examinations as part of their health insurance coverage. Participants provided their oral consent, and the local ethics committee approved the protocol (Universidad Europea Miguel de Cervantes, reference #CEIC_2019_001). All procedures were performed in accordance with the Declaration of Helsinki and its amendments.

2.2 | Variables

We collected data from the last available medical examination for each participant during the aforementioned

period. All variables were assessed and registered by a physician. Demographic/descriptive variables included date of examination, participant's age, sex and socioeconomic status. The latter – a combination of occupation variables, such as activity and professional status of the household – was obtained from the Spanish census. Thus, a territorial indicator was assigned to each record by merging census street map and territorial indicators information (Spanish census and individuals' code postal) as detailed elsewhere.[23]

During examinations, participants filled a questionnaire where they reported sleep duration ('normal' [6-9 h per day], excessively short [<6 h per day] or excessively long [>9 h per day]) and quality ('restful' or 'nonrestful') and also indicated whether they had difficulties falling asleep. Participants reporting one or more sleep abnormalities – that is, short or long sleep duration, nonrestful sleep or difficulties falling asleep – were considered to have 'poor' sleep. Those not reporting any of these sleep abnormalities – that is, those with normal sleep duration, restful sleep and no difficulties falling sleep – were considered to have an 'optimal' sleep.

The clinician in charge also assessed the presence of diabetes (*i.e.*, medicated against diabetes or presenting a fasting glycaemia ≥125 mg/dL),[24] hypercholesterolaemia (*i.e.*, medicated against hypertension or presenting a total blood cholesterol concentration ≥240 mg/dL)[25] and hypertension (*i.e.*, medicated against hypertension or presenting a systolic/diastolic office blood pressure ≥140/90 mmHg).[26] Physical inactivity was determined from self-reported leisure-time physical activity levels, assessed as explained elsewhere.[27] Briefly, participants filled a questionnaire about the frequency and intensity of physical activity during a typical week. They were considered 'inactive' if they did not meet minimum World Health Organization recommendations for aerobic physical activity (*i.e.*, <150 and <75 min per week in moderate and vigorous aerobic activities, respectively).[28] The relevant clinician also measured participants' body mass and height for the computation of body mass index (BMI), and participants were categorised as having normal weight (BMI <25.0 kg/m^2), overweight (BMI 25.0–29.9 kg/m^2) or obesity (BMI ≥30.0 kg/m^2).

2.3 | Statistical analyses

Data are shown as mean ± standard deviation for continuous data and as percentages for categorical ones. Between-group comparisons were performed with the Student's unpaired *t*-test (for continuous data) or the χ^2 test (for categorical data). We used logistic regression analyses to determine the crude and adjusted association between the different sleep characteristics, one the one hand, and the presence of each CVD risk factor, on the other, with individuals showing 'optimal' sleep as the reference group. Regression analyses for each specific CVD risk factor were adjusted by all those variables that significantly differed between individuals with 'poor' sleep and those with 'optimal' sleep (see Results section). In addition, because we aimed to analyse the *independent* association between sleep characteristics and each individual CVD risk factor, analyses for each CVD risk factor were adjusted for the presence of all the other CVD risk factors (i.e., the analyses of hypertension were adjusted for obesity, physical inactivity, diabetes and hypercholesterolaemia; the analyses of obesity were adjusted for hypertension, physical inactivity, diabetes and hypercholesterolaemia). Sub-analyses were performed attending to different age ranges (≤45 and >45 years).

Finally, analysis of covariance (with age, sex, socioeconomic status and smoking as covariables) was used to compare the total CVD risk score (*i.e.*, the number of CVD risk factors shown by a given individual, therefore ranging from 0 to 5) between the following groups: 'poor' vs. 'good sleep', abnormal (long or short) vs. normal sleep duration, unrestful vs. restful sleep and difficulties vs. no difficulties to fall sleep, respectively. Missing data were not imputed. Statistical analyses were performed with Stata 14.0 (StataCorp, Texas, USA) and the level of significance was set at .05.

3 | RESULTS

Participants' descriptive characteristics are shown in Table 1. Data from 527,662 participants (with 0% missing data for the different sleep characteristics and CVD risk factors expect for physical inactivity [7.1%]) were analysed. Thirty-three percent had poor sleep, that is, 14%, 20% and 7% reported abnormal sleep duration, unrestful sleep and difficulties falling asleep, respectively. The proportion of women as well as the socioeconomic status was higher in the 'poor' sleep group compared to their peers with 'optimal' sleep, but there was a lower proportion of smokers in the former. As such, age, sex, socioeconomic status and smoking were used as covariates in regression analyses. On the other hand, the poor sleep group also showed a slightly greater prevalence of obesity, hypercholesterolaemia, hypertension, diabetes and physical inactivity.

'Poor sleep' was associated with a greater likelihood of each of the different individual CVD risk factors in adjusted analyses, with the strongest association observed for physical inactivity (*i.e.*, threefold higher likelihood in participants with poor sleep; Figure 1, see File S1 for both unadjusted and adjusted results). Analyses pertaining to

TABLE 1 Descriptive characteristics of study participants

	'Optimal' sleep (n = 353,534)	'Poor' sleep (n = 174,128)	p-value
Age (years, mean ± SD)	42 ± 9	44 ± 9	<.001
Sex (female; %)	29	38	<.001
Average socioeconomic status (arbitrary units)*	0.978 ± .122	0.983 ± .120	<.001
BMI (kg/m^2, mean ± SD)	26 ± 4	26 ± 3	.140
BMI category (%)			<.001
Normal weight	44	43	
Overweight	39	40	
Obesity	17	17	
Hypercholesterolaemia (%)	28	29	<.001
Total cholesterol (mg/dL, mean ± SD)	195 ± 37	197 ± 36	<.001
HDL (mg/dL, mean ± SD)	56 ± 75	57 ± 101	.187
LDL (mg/dL, mean ± SD)	123 ± 32	124 ± 31	<.001
Hypertension (%)	14	16	<.001
SBP (mmHg, mean ± SD)	116 ± 15	116 ± 15	<.001
DBP (mmHg, mean ± SD)	71 ± 10	71 ± 10	<.001
Diabetes (%)	3.2	3.4	<.001
Fasting glycaemia (mg/dL, mean ± SD)	91 ± 17	92 ± 17	<.001
Smoker (%)	30	29	<.001
Regular alcohol drinker (%)	10	10	.312
Physical inactivity (%)	74	79	<.001

Note: Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; HDL, high density lipoprotein; LDL, low density lipoprotein; SBP, systolic blood pressure. All variables were assessed in all participants (n = 527,662) except for physical inactivity (n = 490,020). * 'Average socioeconomic status' is an indicator from the Spanish Census (*Instituto Nacional de Estadística*, INE) calculated from a combination of occupation variables, such as activity and professional status of the household, where a higher value represents a higher socioeconomic level.[23]

each sleep variable separately revealed that all of them were associated with a greater likelihood of at least two CVD risk factors, expect for a lack of association between unrestful sleep and physical inactivity and also between difficulties to fall asleep and both physical inactivity or diabetes (Figure 2). Moreover, whereas short sleep duration was associated with a greater likelihood (by 1%–33%) of all CVD risk factors except for diabetes, long sleep duration was associated only with physical inactivity and diabetes, especially the latter – albeit with wide confidence intervals (Figure 2).

Sub-analyses attending to the age of the participants indicated that poor sleep was associated with a greater likelihood of all individual CVD risk factors in both adults aged ≤45 or >45 years, except for a lack of association between poor sleep and diabetes in the former (File S2).

Finally, participants with optimal sleep, normal sleep duration, no difficulties to fall sleep and restful sleep showed a significantly lower total CVD risk score than their peers with poor sleep, short sleep duration, difficulties to fall sleep and unrestful sleep, respectively (all $p < .001$, Table 2).

4 | DISCUSSION

The present nationwide cross-sectional study suggests that poor self-reported sleep is potentially associated with a worse CVD risk profile, as different sleep characteristics were associated with the likelihood of major CVD risk factors individually (Figure 2). Particularly, having one or more of the characteristics indicative of poor sleep (*i.e.*, abnormal sleep duration, unrestful sleep or difficulties to fall asleep) was individually associated with a higher likelihood of presenting each of the major CVD risk factors we studied (hypertension, diabetes, hypercholesterolaemia, obesity and physical inactivity). On the other hand, although the association was weak for most risk factors,

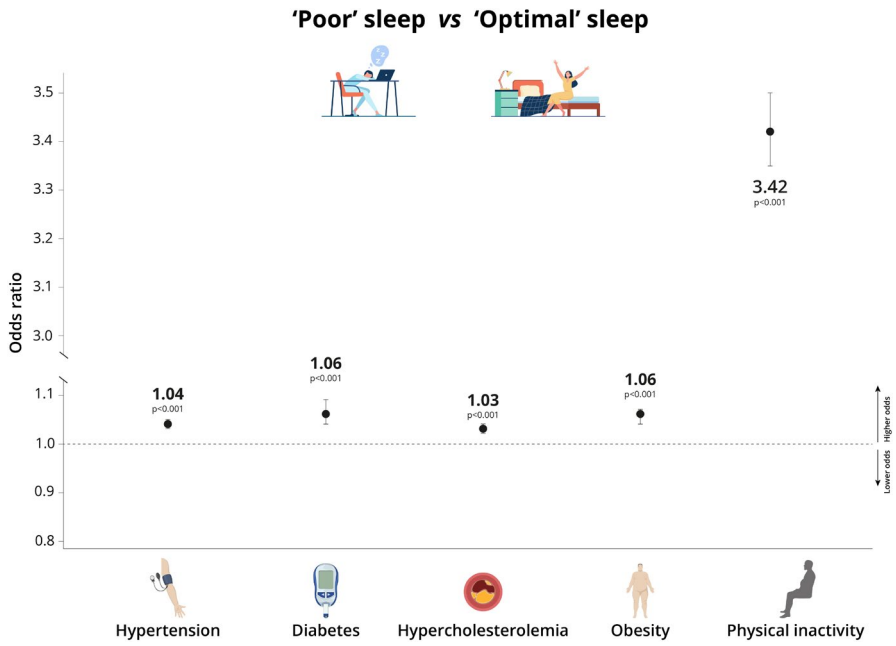

FIGURE 1 Association between poor self-reported sleep and the likelihood of main cardiovascular risk factors. Data are odds ratio (OR) and 95% confidence interval, with participants showing 'optimal' sleep characteristics considered as the reference group. Analyses were adjusted for age, sex, socioeconomic status and smoking. The analyses for each specific cardiovascular risk factor were also adjusted by all the other risk factors. All variables were assessed in all participants (n = 527,662) except for physical inactivity (n = 490,020)

a strong association was found for physical inactivity. Moreover, most characteristics indicative of poor sleep were individually associated with the likelihood of presenting at least three CVD risk factors, with the strongest association for abnormal sleep duration and particularly short sleep duration.

Previous research supports a link between sleep and cardiovascular health. There is meta-analytical evidence that short sleep duration is associated with a higher likelihood of some of the CVD risk factors assessed here, notably diabetes,[9] hypertension[10] or obesity[11,12] and previous studies have reported an association between short sleep duration and sedentary behaviour (*e.g.*, screen time).[29-31] More controversy exists regarding the influence of long sleep duration on CVD risk factors. Although some evidence suggests that long sleeping might be even more detrimental for cardiovascular health than short sleeping,[6] mixed results have been reported for the association between long sleep duration and CVD risk factors such as obesity, hypertension or dyslipidaemia.[12,17] Moreover, a recent Mendelian randomisation analysis concluded that, contrary to genetically predicted short sleep duration, genetically predicted long sleep was not associated with a major CVD risk factor, hypertension or the incidence of CVD per se (including CHD).[5] Our findings suggest that, although a short sleep duration is associated with the likelihood of all the main CVD risk factors except for diabetes, excessive sleep is only associated with physical inactivity and diabetes – which is in line with previous studies.[9,32] Notwithstanding, given that physical inactivity can increase the likelihood of all the major CVD risk factors,[33] it is possible to hypothesise that excessive sleep time might also be detrimental for cardiovascular health in the long term. Moreover, our results suggest that other less commonly analysed sleep-related variables such as difficulties to fall asleep and particularly how restful an individual perceives the sleep are associated with some CVD risk factors. Thus, it might be useful to promote interventions aiming to achieve an optimal sleep duration (approximately 7–8) and enhance sleep quality for potential primary CVD prevention.

Several mechanisms could explain, at least partly, the association between sleep and cardiovascular health. On the one hand, sleep restriction has been associated with deregulated levels of appetite-related hormones such as leptin and ghrelin,[34] and a meta-analysis of randomised controlled trials found that sleep restriction (to a total of 3.5–5.5 h per day) increases subjective hunger, calorie intake and consequently weight gain.[35] Nonetheless, the association between obesity and sleep can be bidirectional, as obesity can impair sleep quality by increasing the risk of sleep-disrupting conditions such as obstructive sleep apnoea (OSA).[36] On the other hand, poor sleep might promote a pro-inflammatory status,[37] decrease insulin sensitivity,[38] induce stress-related responses (*e.g.*, leading to chronically increased activity of the sympathetic nervous system)[39] and impair vascular endothelial function,[40] all of which might contribute to the likelihood of CVD risk factors, particularly diabetes and hypertension. Poor sleep might also result in a misalignment of circadian rhythms, which in turn is a potent risk factor for hypertension,[41] and could reduce energy to engage in regular physical activity.[33]

Despite epidemiological and physiological evidence suggesting an association between sleep and CVD, there is scarce evidence from randomised controlled trials to

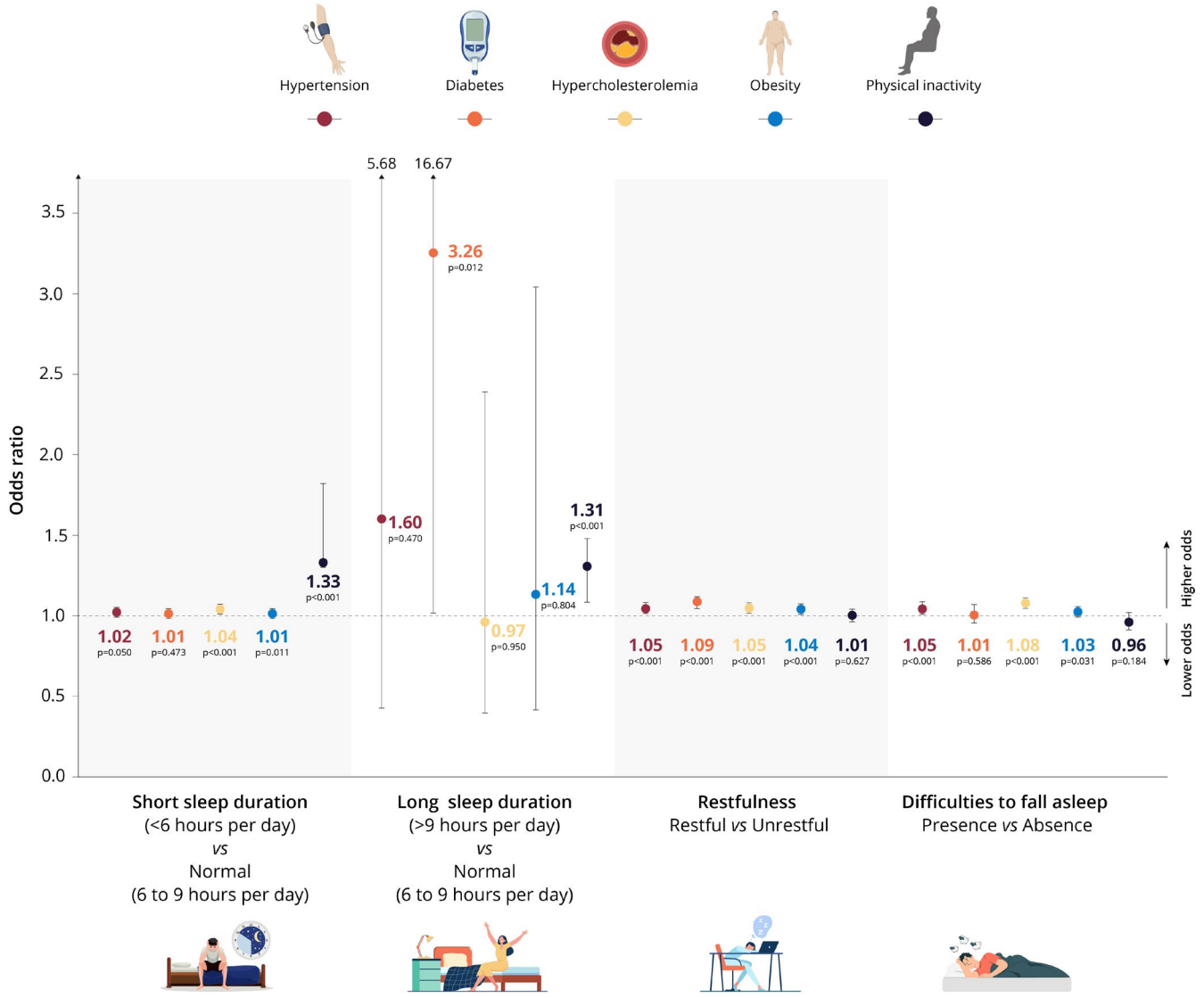

FIGURE 2 Association between specific self-reported sleep characteristics and the likelihood of main cardiovascular risk factors. Data are odds ratio (OR) and 95% confidence interval. Analyses were adjusted for age, sex, socioeconomic status and smoking. The analyses for each specific cardiovascular risk factor were also adjusted by all the other risk factors. All variables were assessed in all participants (n = 527,662) except for physical inactivity (n = 490,020)

support the effectiveness of sleep interventions to enhance cardiovascular health. Nonetheless, some promising findings have been reported with OSA treatment interventions,[42,43] sleeping pills[44] or sleep extension,[42,43] which can lower blood pressure in pre-hypertensive or hypertensive individuals. In addition, the use of continuous positive airway pressure for the treatment of OSA might improve glucose metabolism among diabetic or obese patients.[45-47] Similarly, an extension of sleep duration among short-duration sleepers might attenuate gains in body weight and fat mass over a 6-year follow-up,[48] and epidemiological evidence suggests that substituting 30 min of sedentary time for the equivalent time of sleep can promote weight loss.[49] In turn, other authors have failed to report a beneficial effect of sleep interventions on blood pressure or physical activity levels in individuals with sleep impairments despite improvements in sleep quality.[50,51] Further research is therefore needed to confirm the effectiveness of sleep interventions for enhancing cardiovascular health.

Some limitations of the present study should be acknowledged. The cross-sectional design we used precludes inferring a cause–effect relationship, and thus future longitudinal research is needed to confirm whether the associations reported in our study translate into a higher risk of CVD-related events or mortality. Moreover, even if we attempted to isolate the influence that a given CVD risk factor can have on the presence of each of the other CVD risk factors with adjusted analyses, some overlapping between factors cannot be discarded, with limits the clinical

relevance of our findings for each factor. The self-reported nature of sleep characteristics can also be considered a limitation. In this regard however, previous research has shown a significant association between self-reported and objective (*e.g.*, obtained through polysomnography) sleep measures.[52,53] Moreover, despite potential discrepancies between subjective and objective sleep assessments, both have clinical relevance.[54] Future research should thus corroborate the association between objective sleep measures and CVD risk. The lack of information on other significant variables affecting CVD risk and sleep characteristics, such as dietary factors or the presence of mental disorders (*e.g.*, depression) should also be considered a study limitation. In turn, the included sample size (with data from participants across a whole country and representing one of the largest studies to date on the topic), the absence of missing data except for physical inactivity, the variety of CVD risk factors we assessed and having adjusted the analyses of each CVD risk factor for all the remaining risk factors should be considered major strengths.

TABLE 2 Total cardiovascular (CVD) risk score by sleep characteristics

Overall ('combined') sleep characteristics		
'Optimal' sleep (n = 327,655)	**'Poor' sleep (n = 162,365)**	**p-value**
.55 ± .88	.60 ± .92	<.001
Sleep duration		
Normal (6–9 h/d) (n = 357,715)	Long (>9 h/d) (n = 63,703)	
.55 ± .88	.74 ± 1.02	.310
Normal (6–9 h/d) (n = 357,715)	Short (<6h/d) (n = 68,603)	
.55 ± .88	.60 ± .92	<.001
Sleep quality		
No difficulties to fall sleep (n = 456,846)	Difficulties to fall sleep (n = 33,174)	
.56 ± .89	.64 ± 0.93	<.001
Restful sleep (n = 393,976)	Unrestful sleep (n = 96,044)	
.56 ± .88	.64 ± .93	<.001

Note: Data are mean ± SD. Total CVD risk score was computed as the number of CVD risk factors shown by each an individual, therefore ranging from 0 to 5.

5 | CONCLUSIONS

The present nationwide population-based study suggests an independent association between poor self-reported sleep and the likelihood of presenting major CVD risk factors, particularly physical inactivity (see Figure 3for a graphical summary). This association was separately confirmed for different sleep characteristics including unrestful sleeping, difficulties to fall asleep, as well as short or long sleep duration. Although no causal inferences can be made due to the cross-sectional design we used, these results might support the importance of monitoring a 'nontraditional' risk factor such as sleep for primary prevention of CVD.

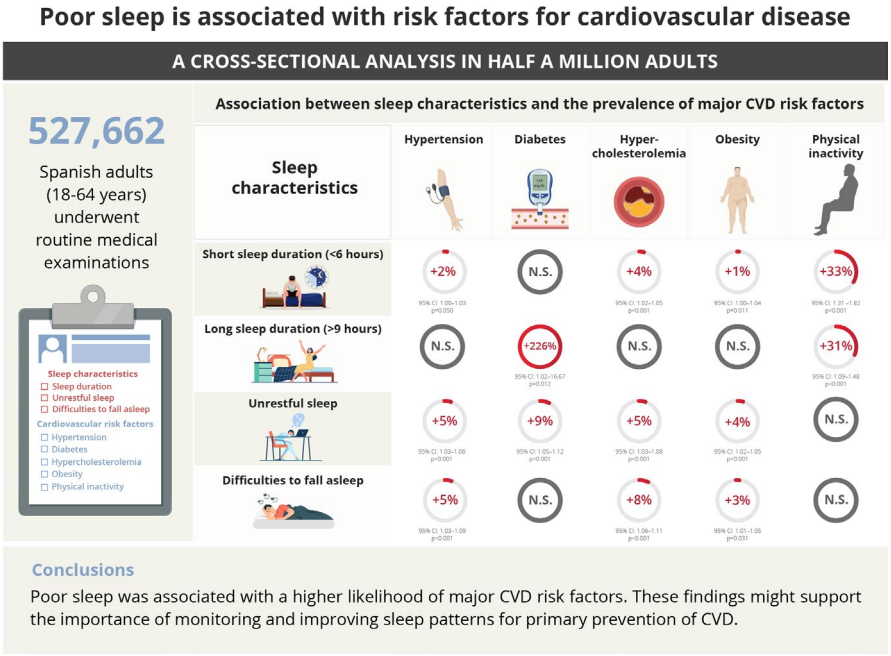

FIGURE 3 Graphical abstract. Abbreviations: CI, confidence interval; CVD, cardiovascular disease; N.S., nonsignificant

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CONFLICT OF INTEREST

None.

AUTHOR CONTRIBUTIONS

Conceptualisation: PLV, ASL and AL; data curation: ASL, ATB, DRI; formal analysis: ASL and ATB; investigation: all authors; methodology: PLV, ASL and AL; supervision: PLV, ASL, LMR, JMO and AL; visualisation: ACG; writing original draft: PLV; writing – review and editing: all authors.

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SUPPORTING INFORMATION

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