American Journal of Preventive Cardiology 26 (2026) 101424

Contents lists available at ScienceDirect

American Journal of Preventive Cardiology

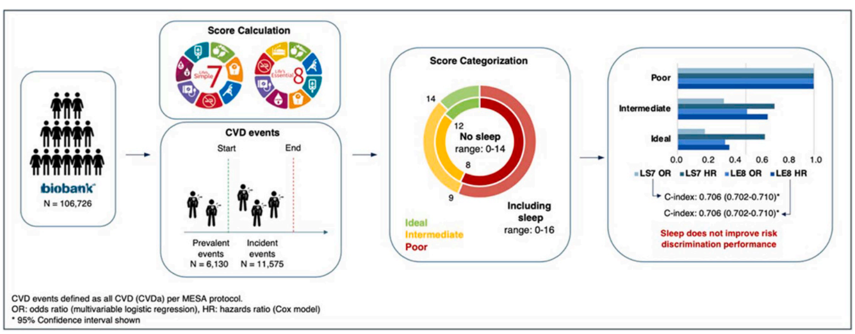
journal homepage: www.journals.elsevier.com/american-journal-of-preventive-cardiology

Understanding the impact of sleep on cardiovascular risk estimation: comparison of LS7 and LE8 performances in a European population

Sofia Mongardi [a], Martino F. Pengo [b,c,*], Carolina Lombardi [b,c], Patrizia Steca [d], Marco Masseroli [a]

[a] Department of Electronics, Information and Bioengineering (DEIB), Politecnico di Milano, Milan, Italy
[b] Department of Cardiovascular, Neural and Metabolic Sciences, Istituto Auxologico Italiano IRCCS, Milan, Italy
[c] Department of Medicine and Surgery, University of Milano-Bicocca, Milan, Italy
[d] Department of Psychology, University of Milano-Bicocca, Milan, Italy

GRAPHICAL ABSTRACT



ARTICLE INFO

Keywords:
Cardiovascular events
Sleep duration
Cardiovascular risk prediction
Cardiovascular health
CVD prevention

ABSTRACT

Background: Tools like Life's Simple 7 (LS7) can help estimate the risk of cardiovascular events in healthy subjects. Recently, the Life's Essential 8 (LE8) was developed, including sleep as an additional variable for a more precise cardiovascular risk estimation. However, it is unclear whether such an increase in complexity is associated with an improvement in the score's performance. We aimed to test the LS7 and LE8 in a European cohort in order to understand whether adding subjective sleep information could allow a better cardiovascular risk stratification.

Methods: UK Biobank data were used for computing the cardiovascular scores. Sleep duration was evaluated through questionnaires. The cardiovascular outcomes were fatal and non-fatal CVD events. Multivariable-adjusted logistic and Cox proportional hazards models were used to evaluate associations of the different metrics with CVD prevalence and incidence. The c-statistic was used to quantify differences in incident CVD discrimination.

Results: A cohort of 106,724 participants (mean age: 55.9 years, 55% males) included 6,130 prevalent and 11,575 incident CVD events (mean follow-up: 12.9 ± 2.7 years). CVH metrics were categorised into tertiles. LS7- and

* Corresponding author at: University of Milan-Bicocca, Department of Medicine and Surgery, IRCCS Istituto Auxologico Italiano, Department of Cardiology, Ospedale San Luca, Via Magnasco, 2, Milan, Italy.

E-mail addresses: m.pengo@auxologico.it, martino.pengo@unimib.it (M.F. Pengo).

https://doi.org/10.1016/j.ajpc.2026.101424
Received 2 September 2025; Received in revised form 22 December 2025; Accepted 11 January 2026
Available online 12 January 2026

LE8-based metrics effectively characterised prevalent and incident CVD events. LS7 models had similar C-statistics with (0.705, 95% CI: 0.701–0.709) and without (0.706, 95% CI: 0.702–0.710) sleep data. LE8 without sleep (0.708, 95% CI: 0.704–0.712) outperformed LS7 without sleep by 0.002 (95% CI: 0.001–0.003, $p < 0.05$). However, standard LE8 with sleep (0.706, 95% CI: 0.702–0.710) showed no significant difference from LS7.

Conclusions: In a European cohort, LS7 and LE8 are useful tools for risk stratification. However, despite the LE8 offering marginally better risk stratification than LS7, the inclusion of subjective sleep did not provide a tangible advantage.

1. Introduction

Cardiovascular disease (CVD) risk assessment is a cornerstone of preventive cardiology and depends on many different factors such as age, sex, and family history that cannot be changed. However, modifiable risk factors, including hypertension, high cholesterol, diabetes, smoking, obesity, sedentary lifestyle, and poor diet, can be addressed through medical interventions and lifestyle changes. Risk assessment tools, such as risk scores and algorithms, combine these risk factors to provide a quantitative estimate of an individual's 10-year risk of a cardiovascular event. These tools are invaluable in guiding clinical decision-making and in communicating the importance of lifestyle modifications to patients. In fact, by identifying individuals at higher cardiovascular risk, even before the onset of overt symptoms, healthcare providers can tailor preventive strategies to reduce the development of cardiovascular complications. In recent years, the role of sleep in cardiovascular health has received increasing attention. While traditional risk factors such as hypertension, high cholesterol, smoking habits, and unhealthy diet have long been recognised, emerging research has underscored the critical importance of sleep in maintaining cardiovascular well-being, as recently highlighted by the American Heart Association (AHA) [1]. Sleep deprivation and sleep disturbances have indeed been linked to metabolic imbalances, autonomic nervous system dysfunction, inflammation, endothelial dysfunction, increased blood pressure, all of which are known risk factors for cardiovascular disease [2,3]. By incorporating sleep into cardiovascular risk assessment, healthcare providers could gain a more comprehensive understanding of an individual's risk profile and tailor preventive strategies accordingly, promoting healthy sleep habits and addressing underlying factors that may be contributing to sleep disturbances. Among the various risk scores, the Life's Simple 7 (LS7) [4] and Life's Essential 8 (LE8) [1] metrics have emerged as valuable tools for assessing cardiovascular health. Proposed by the AHA, these metrics provide a comprehensive evaluation of various health domains, including smoking, diet, physical activity, body mass index, blood pressure, cholesterol, fasting glucose and, more recently, sleep in LE8. While both metrics have demonstrated strong predictive capabilities for incident cardiovascular events in American populations, the addition of sleep as a distinct domain in the LE8 raises the question of its potential impact on the overall predictive performance. In fact, a recent study on 30,239 community-dwelling American individuals did not show significant differences between the two scores in predicting incident cardiovascular events [5]. Thus, the aim of the present study was to test the performances of LS7 and LE8 in a large European cohort to understand their utility for cardiovascular risk prediction, and whether the addition of sleep metrics is associated with more accurate cardiovascular risk estimation in this population.

2. Materials and methods

For the present study, data from the UK Biobank (UKB), a large-scale prospective cohort study that recruited approximately 500,000 participants aged 40-69 years between 2006 and 2010 across the United Kingdom, were analysed [6]. The cohort comprises comprehensive phenotypic and genetic data, including detailed health records, lifestyle questionnaires, physical measurements, biological samples, and multi-modal imaging data from a subset of 100,000 participants.

2.1. Cardiovascular health metrics

The LS7 and LE8 scores consider different cardiovascular health (CVH) metrics. These metrics include body mass index (BMI), total cholesterol and blood glucose levels, blood pressure, diet, physical activity, and smoking. BMI, cholesterol, blood pressure, and blood glucose levels were measured during the baseline visit (UKB Data-Field 21001, "Body mass index"; UKB Data-Field 30690, "Cholesterol"; UKB Category 100011, "Blood pressure"; UKB Data-Field 30740, "Glucose"). Two measures of systolic and diastolic blood pressure readings were taken a few moments apart for each participant; the average of the 2 readings was used. Information on smoking, diet, and physical activity were obtained from the touchscreen questionnaire completed at the Assessment Centre during the baseline visit (UKB Data-Field 20116, "Smoking status"; UKB Category 100052, "Diet"; UKB Category 100054, "Physical activity"). We considered subjective sleep duration ("Sleep duration", touchscreen questionnaire, UKB Data-Field 1160) as sleep-derived metric. Sleep duration was assessed through the question: "About how many hours sleep do you get in every 24 hours? (including naps)". By design, the LE8 also considers additional CVH metrics in the score calculation, including High-Density Lipoprotein (HDL) cholesterol, Glycated hemoglobin (HbA1c), diabetes, and additional smoking information (UKB Data-Field 30760, "HDL cholesterol"; UKB Data-Field 30750, "Glycated hemoglobin"; UKB Data-Field 2443, "Diabetes diagnosed by doctor"; UKB Category 100058, "Smoking").

2.2. Computation of LS7 score

The Life's Simple 7, defined by the American Heart Association is computed based on adherence to recommendations for body mass index, cholesterol, blood pressure, blood glucose, diet, physical activity, and smoking, consistent with previous studies. For each of the considered parameters, a score of 2, 1, or 0 is assigned based on the level the recommendation met. These scores are summed to create the final LS7 score, where a higher score indicates a better CVH. Since 7 components are considered, the score ranges between 0 and 14. Following the work of Makarem et al. [7], we categorised the score as: 0 to 7 (poor), 8 to 11 (intermediate), and 12 to 14 (ideal). Additional information on the guidelines and score operalization is available in Table S1 in Supplementary Section S2.1. The diet score used in Makarem et al. [7] was computed considering habitual dietary intake from a validated 120-item food frequency questionnaire, modified from the Insulin Resistance Atherosclerosis Study instrument [8] (further details on the used recommendations can be found in Supplementary Section S2.2). Because such a questionnaire was not available for the UK Biobank cohort, we adapted the diet score by using the information extracted from the UK Biobank touchscreen questionnaire. A detailed description on the calculation of such a diet score is available in Supplementary Section S2.3. We also computed an alternative LS7 score that also includes sleep as an additional eighth metric. The subjective sleep duration (questionnaire) was used. By design, the new score is in the range [0, 16]; it was categorised as follows: 0 to 8 (poor), 9 to 13 (intermediate), and 14 to 16 (ideal).

2.3. Computation of LE8 score

Life's Essential 8 is an updated version of the LS7, always defined by the AHA. This enhanced score incorporates sleep as an additional health metric and expands the scoring gradients for all 8 health components considered, transitioning from the previous three-level classification system for each metric to a more detailed and semi continuous scoring approach. We refer the readers to the work of Lloyd-Jones et al. [1] for a clear description of the CVH domains considered in the LE8 score. In the LE8 score, the diet, nicotine avoidance (expanded from smoking-only in LS7), body mass index, cholesterol, and blood pressure domains have 5 levels, the new sleep domain has 6 levels, blood glucose has 7 levels, and physical activity has 8 levels. Each domain is scored on a standardised scale ranging from 0 to 100, with the final LE8 score calculated as the mean across all eight domains. This refined scoring system provides greater granularity in health assessment compared to the original LS7 framework, potentially enabling more precise evaluation of CVD risk as subtle changes in each of the considered health domains can be better characterised, despite requiring increased effort in the collection of all the needed information. We computed the score following the guidelines and steps described in Lloyd-Jones et al. [1]. The diet score in the LE8 is computed considering self-reported daily intake of a DASH-style eating pattern (population) [9], or the Mediterranean Eating Pattern for Americans (MEPA) screener (individuals) [10]. As for the LS7, such a screener is not available for the UK Biobank cohort. We modified such diet score using information coming from the UK Biobank touchscreen questionnaire completed at baseline. Further details on the calculation of this diet score are reported in Supplementary Section S3.2. To categorize the LE8 score similar to the LS7 score for comparison, the LE8 score was normalised to a 0-16 scale. The normalised LE8 score was then categorised as follows: 0 to 8 (poor), 9 to 13 (intermediate), and 14 to 16 (ideal), as the LS7 score. Additionally, we constructed a LE8 alternative score that does not include sleep. This LE8-based metric was normalised to a 0-14 scale, and then categorised as the standard LS7 score.

2.4. Evaluation of cardiovascular outcomes

As CVD events, we considered fatal and non-fatal CVD events according to all CVD (CVDa) of the Ethnic Study of Atherosclerosis (MESA) protocol, focusing on both incident and prevalent events as described in the work of Makarem et al. [7]. Non-fatal CVD events include congestive heart failure (CHF), angina, myocardial infarction, resuscitated cardiac arrest, peripheral arterial disease, stroke, and transient ischaemic attack (TIA). Fatal cardiovascular endpoint categories generally include fatal coronary heart disease (CHD), fatal stroke, and other fatal CVD events. Additional information on the CVD events considered and corresponding codes of the 10^{th} revision of the International Classification of Diseases (ICD-10) are available in Supplementary Section S1. The follow-up period spanned from baseline to the earliest of three events: the occurrence of CVD events, loss to follow-up, or November 30, 2022.

2.5. Statistical analysis

We conducted two main analyses to compare the traditional LS7 and the modified LS7 score that includes sleep as the eighth metric. First, we used multivariable-adjusted logistic regression to examine how these scores related to existing cardiovascular diseases (prevalent cases). Second, we used Cox proportional hazards models to assess how these scores predicted new cases of CVD (incident cases). The prevalent and incident cases were calculated with respect to the date of the baseline visit. All models were adjusted for age (years), sex (male, female), race and ethnicity (White, Black, Hispanic, and Chinese-American), education (college or greater, less than college). We performed the same analyses also for the LE8-based scores, always adjusting for the same covariates. Additionally, to evaluate whether LE8 provides superior predictive value compared to LS7, we examined the difference between their respective C-statistics. The C-statistic measures a model's ability to discriminate between two randomly chosen subjects - one with and one without a cardiovascular event - by calculating the probability of assigning a higher risk score to the subject who experienced the event. The c-statistic ranges from 0.5 (indicating no predictive ability) to 1.0 (perfect prediction). To determine if the difference between the LS7 and LE8 C-statistics was statistically significant, we conducted a Wald test by dividing the difference in C-statistics by its standard error. We used bootstrap resampling with 1,000 iterations to calculate the standard error of this difference. Similarly, we applied the same bootstrap resampling procedure, using the Wald test to assess whether the model coefficients from the LS7- and LE8-based models including sleep in the score computation were significantly different from those obtained from models that did not include sleep. All statistical analyses were performed using the R software (version 4.2.3).

3. Results

3.1. Cohort definition and characteristic

We performed a complete case analysis, starting from the UK biobank cohort (N=502,233). We removed all participants with unknown event date (N=328), leaving us with a total of 501,905 participants. We further discarded all participants who had missing information for any of the variables considered in the analysis, including also all the metrics described in Section 2.1. All the self-reported questionnaire fields considered also included "Don't know" or "Prefer not to answer" as possible answers. We considered these answers as missing values and excluded all the corresponding participants from the analyses. Further details can be found in the Supplementary Section S4.1. The final cohort we used in our experiments had a total of 106,724 participants (mean age: 55.9 ± 8.1 years, 55% males). Such cohort included 6,130 prevalent CVD events and 11,575 incident events (mean follow-up: 12.9 ± 2.7 years). Table 1 provides descriptive statistics for the final cohort, giving a detailed overview of its characteristics. Given that the final cohort represents roughly one-fifth of the original 501,905 participants, we computed descriptive statistics for the full initial cohort to evaluate the representativeness of the final population. Descriptive statistics for the original cohort are also available in Table 1 and closely align with the statistics from the final derived cohort for the most of the considered metrics in the calculation of the LS7 and LE8 scores, as well as for the numbers of prevalent and incident CVD cases. The CVH metrics were categorised into tertiles, as described in Sections 2.2 and 2.3. Information on the number of participants and CVD events for each category are available in Tables 2 and 3, and Fig. 1.

3.2. Impact of LS7-based CVH metrics on prevalent and incident CVD prediction

The main results are reported in Table 4 for LS7-based metrics. Across all LS7-based CVH metrics, higher scores were linked to lower odds of prevalent CVD at the baseline visit. Individuals in the highest tertile (ideal) of the LS7 score exhibited 80% lower odds of developing CV compared to those in the lowest tertile (poor) (odds ratio [OR]: 0.20; 95% confidence interval [CI]: 0.14–0.27). Similar results were also obtained for the LS7-based score incorporating subjective sleep. Participants in the top tertiles of such a score had 84% reduction in CVD odds (OR: 0.16; 95% CI: 0.11-0.23). Those in the middle tertile (intermediate) also showed lower odds of having CVD at the baseline visit, with 66% and 65% reduction in CVD odds, respectively (OR: 0.34; 95% CI: 0.33-0.36, and OR: 0.35; 95% CI: 0.33-0.37, respectively).

Considering the incident CVD events, also the LS7-based scores in the highest tertile were significantly associated with CVD incidence (Table 4) according to the Cox proportional hazards models. Participants in this tertile had 36% and 36% lower risk, respectively (hazard ratio [HR]: 0.64; 95% CI: 0.55-0.75, and HR: 0.64; 95% CI: 0.54-0.76,

Table 1
Description of the final and original cohorts and components of the LS7 and LE8 CVH metrics.

		Final Cohort	Original Cohort
Age (years, mean ± SD)		55.9 ± 8.1	56.5 ± 8.1
Gender (N, %)	Female	47,524 (45%)	273,071 (56%)
	Male	59,200 (55%)	228,834 (44%)
Ethnicity (N, %)	White	104,408 (97.8%)	472,158 (94.6%)
	Asian	889 (0.8%)	11,433 (2.3%)
	Black	619 (0.6%)	8,042 (1.6%)
	Mixed	432 (0.4%)	4,549 (0.9%)
	Other	376 (0.4%)	2,951 (0.6%)
Smoking (N, %)	Never	70,533 (67%)	273,260 (54.8%)
	Previous	32,083 (29%)	172,829 (34.6%)
	Current	4,108 (4%)	52,871 (10.6%)
Body mass index (mean ± SD)		26.7 ± 4.0	27.4 ± 4.8
Cholesterol (mg/dL, mean ± SD)		103.2 ± 19.7	102.5 ± 20.6
Glucose (mg/dL, mean ± SD)		90.9 ± 18.0	92.2 ± 22.3
Diabetes (N, %)	Yes	3,329 (3%)	26,333 (5.3%)
	No	103,395 (97%)	137.8 ± 18.8
Systolic blood pressure (mmHg, mean ± SD)		138.0 ± 18.2	137.8 ± 18.8
Diastolic blood pressure (mmHg, mean ± SD)		82.4 ± 10.0	82.2 ± 10.2
Number of hours of sleep (hours, mean ± SD)		7.2 ± 1.0	7.2 ± 1.1
Follow-up (years, mean ± SD)		12.9 ± 2.7	12.7 ± 3.0
Prevalent CVD cases (N, %)		6,130 (7.8%)	38,960 (13.4%)
Incident CVD cases (N, %)		11,575 (5.7%)	67,060 (10.8%)
Participants		106,724	501,905

Descriptive statistics for the original cohort were calculated for each variable independently, excluding missing data.

Table 2
Number of participants (percentage%) in each score category for CVH metrics.

Type	LS7	LS7 sleep	LE8 no sleep	LE8
Poor	15,380 (14.4)	10,504 (9.8)	17,661 (16.5)	9,295 (8.7)
Intermediate	89,255 (83.6)	94,691 (88.7)	85,084 (79.7)	95,440 (89.4)
Ideal	2,089 (2.0)	1,529 (1.4)	3,979 (3.7)	1,989 (1.9)

LS7: Life's Simple 7; LE8: Life's Essential 8.

Table 3
Number of CVD events (prevalent/incident) in each score category for the considered CVH metrics.

Type	LS7	LS7 sleep	LE8 no sleep	LE8
Poor	2,281 (15)/ 2,737 (18)	1,696 (16)/ 2,012 (19)	1,979 (11)/ 4,054 (18)	1,203 (13)/ 4,882 (20)
Intermediate	3,798 (4)/ 8,663 (10)	4,402 (5)/ 9,429 (10)	4,054 (5)/ 8,202 (10)	4,882 (5)/ 9,640 (10)
Ideal	51 (2)/175 (8)	30 (2)/115 (9)	97 (2)/167 (4)	45 (2)/79 (4)

LS7: Life's Simple 7; LE8: Life's Essential 8.
The percentage (%) of corresponding CVD events within each considered category is also provided.

respectively). For the middle tertile, instead, individuals had 29%, and 32% reduced risk of incident CVD, respectively (HR: 0.71; 95% CI: 0.68-0.74, and HR: 0.68; 95% CI: 0.65-0.71, respectively). Assessment of the proportional hazards assumption indicated that it held for all covariates with significant associations. A deviation from the assumption was detected only for one non-significant covariate (Ethnicity), suggesting it does not affect the validity of the overall model inference.

3.3. Predictive value of LE8-based CVH scores on prevalent and incident CVD

We report the results from the statistical analyses for LE8-based CVH metrics in Table 5. Across all LE8-based CVH metrics, higher scores were consistently associated with lower odds of prevalent CVD events at the baseline visit. Individuals in the highest tertile (ideal) of the modified LE8 score not including sleep exhibited a 59% reduction in CVD odds compared to those in the lowest tertile (poor) (OR: 0.41; 95% CI: 0.34–0.51). Similar findings were observed for the standard LE8-based score, incorporating subjective sleep. Indeed, those in the highest tertiles of these scores demonstrated 65% lower odds of CVD (OR: 0.35; 95% CI: 0.26–0.47). Those in the middle tertile (intermediate) also exhibited reduced odds of CVD at the baseline visit, showing a 45% (no sleep) and 49% (subjective sleep) reduction in CVD odds, respectively (OR: 0.55; 95% CI: 0.52–0.58, and OR: 0.51; 95% CI: 0.47–0.55, respectively).

Significant associations were also observed for the Cox proportional hazards models. Indeed, participants in the highest tertile of LE8-based CVH metrics showed significantly reduced incident CVD risks by 58% (no sleep) and 62% (subjective), respectively (HR: 0.42; 95% CI: 0.36–0.49, and HR: 0.38; 95% CI: 0.30-0.48, respectively). Instead, individuals in the middle tertile (intermediate) demonstrated 32% and 34% reductions in the risk of incident CVD, respectively (HR: 0.68; 95% CI: 0.65–0.71, and HR: 0.66; 95% CI: 0.62–0.69, respectively). As for the LS7-based models, the proportional hazards assumption was satisfied for all covariates that were significantly associated with the outcome.

3.4. Comparative analysis of LS7- and LE8-based scores

We further examined whether the potential informational advantage of the LE8 score would demonstrate statistical significance compared to the LS7 score. As outlined in Section 2.3, calculating the LE8 score involves greater complexity, as it requires additional and more detailed data. Additionally, we aimed to evaluate whether incorporating sleep-derived information could enhance the predictive performance of the developed models. Differences in the ability to discriminate incident CVD were assessed using changes in the C-statistic as evaluation metric. Recognising that both LS7 and LE8 are primarily designed for primary prevention, we conducted additional analyses by excluding participants with prevalent events prior to the baseline visit, resulting in a refined cohort of 100,594 subjects. Details and information on this additional cohort are provided in Supplementary Section S5 along with the results from its statistical analysis. The C-statistics values for the different adjusted Cox proportional hazards models on the original ("whole cohort") and refined ("Incident cases") cohorts are presented in Table 6. Considering the original cohort ("whole cohort"), the LS7-based models, both without sleep data (0.706, 95% CI: 0.702–0.710) and with subjective sleep (0.705, 95% CI: 0.701–0.709), show comparable C-statistics. The LE8 model, when not incorporating sleep (0.708, 95% CI: 0.704–0.712), demonstrates a significantly better discrimination performance than the baseline LS7 (no sleep) model, with an increase of 0.002 (95% CI: 0.001–0.003, p-value < 0.05). However, for the standard LE8, which includes subjective sleep information, the performance (0.706, 95% CI: 0.702–0.710) is identical to that of the baseline LS7 (no sleep) model, with no significant difference between them. In the refined ("Incident cases") cohort, for the LS7-based metrics, the inclusion of subjective sleep did not alter the discrimination performance, as both models show equal results (0.698, 95% CI: 0.792–0.704). For the LE8 metrics, the subjective sleep model (0.700, 95% CI: 0.694–0.706) has a slight decreased discrimination performance with respect to the no sleep

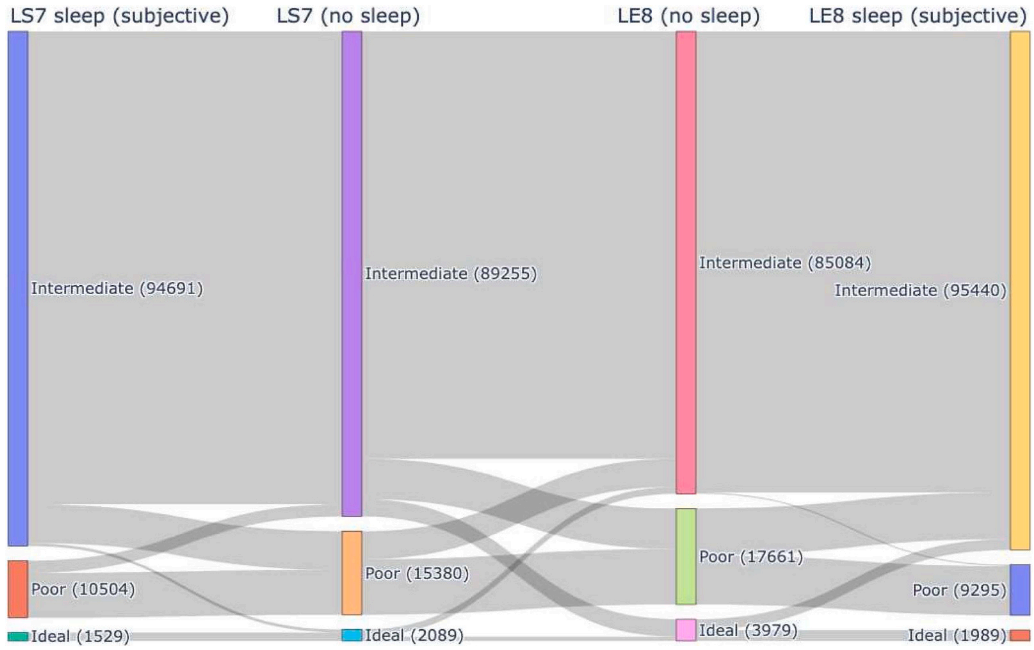


Fig. 1. Visual representation of flow transitions between CVH categories for the Life's Simple 7 (LS7)- and Life's Essential 8 (LE8)-based metrics, displaying the relative contributions and connections among categories. For additional information of transition counts see Supplementary Table S6.

Table 4
Association of Life's Simple 7 (LS7) score and an alternative LS7 modification that includes subjective sleep.

Type	LS7 CVD OR no sleep	LS7 CVD HR no sleep	LS7 CVD OR subjective	LS7 CVD HR subjective
Poor	1.00	1.00	1.00	1.00
Intermediate	0.34 (0.33-0.36)	0.71 (0.68-0.74)	0.35* (0.33-0.37)	0.68* (0.65-0.71)
Ideal	0.20 (0.14-0.27)	0.64 (0.55-0.75)	0.16 (0.11-0.23)	0.64 (0.54-0.76)

prevalence: odds ratio (95% confidence interval). incidence: hazard ratio (95% confidence interval).

* significant coefficients with respect to the no-sleep model (p-value < 0.05), using bootstrap resampling with 1,000 iterations to calculate the standard error of this difference (Wald test)

Table 5
Association of Life's Essential 8 (LE8) score (including subjective sleep) and an alternative LE8 score that does not include sleep.

Type	LE8 CVD OR no sleep	LE8 CVD HR no sleep	LE8 CVD OR subjective	LE8 CVD HR subjective
Poor	1.00	1.00	1.00	1.00
Intermediate	0.55 (0.52-0.58)	0.68 (0.65-0.71)	0.51* (0.47-0.55)	0.66 (0.62-0.69)
Ideal	0.41 (0.34-0.51)	0.42 (0.36-0.49)	0.35* (0.26-0.47)	0.38 (0.30-0.48)

prevalence: odds ratio (OR) (95% confidence interval). incidence: hazard ratio (HR) (95% confidence interval).

* significant coefficients with respect to the no-sleep model (p-value < 0.05), using bootstrap resampling with 1,000 iterations to calculate the standard error of this difference (Wald test)

model (0.701, 95% CI: 0.696–0.704), but both are significantly better than the LS7 models (p-value < 0.05). These findings show that, despite occasional statistical significance, the actual gain in predictive performance is minimal, suggesting that LE8 offers little meaningful improvement over LS7 in practice. Furthermore, the most significant

Table 6
Discrimination performance of the LS7- and LE8-based CVH metrics (95% CI).

Cohort	LS7 (reference) (no sleep)	LS7 (subjective)	LE8 (no sleep)	LE8 (subjective)
Whole cohort (n=106,724)	0.706 (0.702-0.710)	0.705 (0.701-0.709)	0.708* (0.704-0.712)	0.706 (0.702-0.710)
Incident cases (n=100,594)	0.698 (0.792-0.704)	0.698 (0.792-0.704)	0.701* (0.696-0.704)	0.700* (0.694-0.706)

Whole cohort: cohort including subjects with both prevalent and incident cases. Incident: cohort with only incident case subjects.

* C-statistics significant with respect to the baseline LS7 CVH metric (p-value < 0.05), using bootstrap resampling with 1,000 iterations to calculate the standard error of this difference (Wald test).

LS7: Life's Simple 7; LE8: Life's Essential 8.

differences in terms of discriminative power were mainly found for the LE8-based models without subjective sleep duration, indicating the observed improvements are mainly due to the more fine-grained score calculation rather the inclusion of sleep information.

4. Discussion

Cardiovascular risk estimation still represents a challenge, particularly in patients in primary prevention. Refining risk scores can allow a more precise risk estimation however on the other side leads to increased complexity making such scores to difficult to be implemented in clinical practice. In the present study, we confirmed the usefulness of LS7 and LE8 metrics for risk stratification in a European cohort. However, the addition of sleep metrics, in particular subjective sleep duration, does not seem to confer additional improvement in risk stratification. Our findings are consistent with previous research that demonstrated the overall efficacy of LS7 and LE8 scores in diverse populations [11,12].

Certain insights into the role of sleep and the comparative utility of LS7 and LE8 warrant further exploration. While the inclusion of sleep as an eighth metric in LE8 reflects growing recognition of its role in cardiovascular health, our results suggest that subjective sleep measurements do not significantly enhance risk prediction in a European cohort. Several factors can explain these findings with the main one being the inherent subjectivity of self-reported sleep assessments that could have potentially diluted the predictive power of this metric within the LE8 framework. Furthermore, relying on sleep duration alone could have missed some important information about sleep quality and the presence of sleep disorders such as obstructive sleep apnea (OSA), a known risk factor for cardiovascular diseases. Nevertheless, sleep duration can still hold important prognostic information as shown in a systematic review and meta-analysis of 16 prospective studies (1,382,999 participants) supporting the presence of a relationship between self-reported sleep duration and all-cause mortality [13]. The findings of the present work pose the question whether objective sleep assessments, such as actigraphy or polysomnography, could provide a more accurate and robust evaluation of sleep-related cardiovascular risk. A study on an American cohort of the Sleep Heart Health Study (SHHS) [14] showed that although objective and self-reported sleep duration were both associated with all-cause and CVD mortality they showed different patterns suggesting different relationship with cardiovascular outcomes. Another important aspect, which is not fully captured by self-reported sleep duration, is sleep regularity that has been shown to be a stronger predictor of mortality risk than sleep duration [15]. This is relevant as irregular sleep patterns often hide the presence sleep disorders such as insomnia, which has been itself associated with increased risk of developing cardiovascular consequence [16].

Nevertheless, in our study, LS7 and LE8 scores, whether or not they included sleep, were consistently associated with both prevalent and incident CVD. This contrasts with previous findings from an American cohort [7], where neither LS7 nor a modified LS7 incorporating sleep duration was associated with incident CVD risk prediction. Notably, LS7-based metrics in our study showed stronger risk categorisation for prevalent CVD, while LE8-based scores more effectively characterised incident events. Additionally, the LE8 score offered as slight improvement over LS7 in predicting incident cases, consistent with its more detailed and comprehensive approach. However, these improvements do not appear to be primarily driven by the inclusion of sleep duration data, as the LE8-based model without sleep outperformed the standard LE8 model in risk prediction accuracy. Furthermore, the enhancement is modest and comes at the cost of increased data collection and computational complexity. In particular, the additional metrics in LE8, including refined measures of lifestyle and clinical variables, require substantial resources that can limit their applicability in routine clinical practice. Indeed, despite a significant proportion of participants lacking complete data for either score (61% for LS7 and 80% for LE8), the proportion of incomplete data was notably higher for the LE8 score. It is also important to note that, despite the substantial reduction in cohort size, the final sample remained representative of the original population, as confirmed by the consistency in descriptive statistics between the two cohorts (see Table 1). Our results are consistent with a similar analysis proposed by Howard et al. [5], who tested LS7 and LE8 performance in 11,609 Black and White American participants from the REGARDS study (Reasons for Geographic and Racial Differences in Stroke) and concluded that both were associated with incident CVD with no statistically significant difference between the scores. As mentioned before, sleep duration is one of the domains of sleep health, which does not include the presence of sleep disorders such as insomnia and OSA that are recognised CV risk factors. Indeed, You et al. [17] demonstrated that adding additional sleep metrics such as chronotype, duration, insomnia, snoring, and daytime dozing could further help in defining the most favorable sleep pattern associated with the lowest risk of CVD complications. This is relevant as the treatment of sleep disorders and restoring a more regular sleeping pattern could have some relevant implications in improving cardiovascular health [18]. Nevertheless, our data support the LS7 as a valuable tool to identify cardiovascular health; however, adding information about sleep duration does not appear to improve its capability of reclassifying patients based on their CVD risk. This might suggest that adding more variables and more detail to a score does not necessarily improve key aspects of its performance [19].

Some limitations of this study warrant consideration. First, the reliance on self-reported sleep data introduces potential bias due to inaccuracies in participants' recollection and perception of their sleep habits. This subjectivity may have contributed to the marginally lower predictive value of LE8 compared to LS7. Although the UK Biobank provides objective sleep data, it is available only for a limited subsample of participants and was collected during follow-up rather than at the baseline visit (mean temporal distance: 5.2 ± 1.1 years). Furthermore, because it was not collected in conjunction with a study visit, no clinical information is available for that time point, making it unsuitable for inclusion or direct comparison in the present analysis. Second, sleep data were collected only at baseline, preventing an assessment of temporal changes in sleep patterns over the follow-up period. Considering the dynamic nature of sleep and its potential interactions with other risk factors, this limitation could affect the comprehensiveness of the analysis. Another limitation is the generalizability of findings beyond the predominantly White, European-based UK Biobank cohort. Although the results provide valuable insights for European populations, the applicability of these findings to more diverse populations remains uncertain. Additionally, while LE8 incorporates more metrics for cardiovascular risk assessment, the increased data requirements may pose challenges for resource-limited healthcare settings, potentially limiting its feasibility for widespread implementation. Furthermore, an additional limitation of our study, shared with prior LS7 and LE8 investigations in European cohorts [20,17], is the need to adapt the diet component based on available dietary data; however, as detailed in Supplementary Sections S2.3 and S3.2, we reconstructed the LS7 and LE8 diet scores as faithfully as possible, following the same principles and using all equivalent information available.

5. Conclusion

In conclusion, while LE8 offers marginally better risk stratification than LS7, the inclusion of subjective sleep data in its current form may not provide a tangible advantage. Future advancements in sleep assessment hold promise for refining cardiovascular risk prediction and broadening the utility of these scoring systems.

Funding

This work was supported by the MUSA – Multilayered Urban Sustainability Action – project, funded by the European Union – NextGenerationEU, under the National Recovery and Resilience Plan (NRRP) Mission 4 Component 2 Investment Line 1.5: Strengthening of research structures and creation of R&D “innovation ecosystems”, set up of “territorial leaders in R&D”.

CRediT authorship contribution statement

Sofia Mongardi: Writing – original draft, Software, Methodology, Formal analysis, Data curation, Conceptualization. **Martino F. Pengo:** Writing – review & editing, Validation, Supervision, Methodology, Conceptualization. **Carolina Lombardi:** Writing – review & editing, Visualization, Validation. **Patrizia Steca:** Writing – review & editing, Visualization, Supervision, Funding acquisition. **Marco Masseroli:** Writing – review & editing, Visualization, Validation, Supervision, Methodology, Funding acquisition, Conceptualization.

Declaration of competing interest

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us.

We confirm that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property.

We understand that the Corresponding Author is the sole contact for the Editorial process (including Editorial Manager and direct communications with the office). He is responsible for communicating with the other authors about progress, submissions of revisions and final approval of proofs.

We confirm that we have provided a current, correct email address which is accessible by the Corresponding Author.

Acknowledgments

This research has been conducted using the UK Biobank Resource under Application Number 102297 in accordance with the guidelines provided at http://www.ukbiobank.ac.uk/using-the-resource/. The authors extend their sincere gratitude to the investigators, the dedicated staff, and the participants in the UK Biobank study for their invaluable contributions.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ajpc.2026.101424.

References

[1] Lloyd-Jones D M, Allen N B, Anderson C A, Black T, Brewer L C, Foraker R E, Grandner M A, Lavretsky H, Perak A M, Sharma G, et al. Life's Essential 8: updating and enhancing the American Heart Association's construct of cardiovascular health: a presidential advisory from the American Heart Association. Circulation 2022;146(5):e18–43.

[2] Covassin N, Singh P, McCrady-Spitzer S K, St Louis E K, Calvin A D, Levine J A, Somers V K. Effects of experimental sleep restriction on energy intake, energy expenditure, and visceral obesity. J Am Coll Cardiol 2022;79(13):1254–65.

[3] Lombardi C, Pengo M, Parati G. Obstructive sleep apnea syndrome and autonomic dysfunction. Auton Neurosci 2019;221:102563.

[4] Lloyd-Jones D M, Hong Y, Labarthe D, Mozaffarian D, Appel L J, Van Horn L, Greenlund K, Daniels S, Nichol G, Tomaselli G F, et al. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic impact goal through 2020 and beyond. Circulation 2010;121(4):586–613.

[5] Howard G, Cushman M, Blair J, Wilson N R, Yuan Y, Safford M M, Levitan E B, Judd S E, Howard V J. Comparative discrimination of Life's Simple 7 and Life's Essential 8 to stratify cardiovascular risk: is the added complexity worth it? Circulation 2024;149(12):905–13.

[6] UK Biobank (2006). Protocol for a large-scale prospective epidemiological resource. www.ukbiobank.ac.uk/resources/.

[7] Makarem N, Castro-Diehl C, St-Onge M P, Redline S, Shea S, Lloyd-Jones D, Ning H, Aggarwal B. Redefining cardiovascular health to include sleep: prospective associations with cardiovascular disease in the MESA sleep study. J Am Heart Assoc 2022;11(21):e025252.

[8] Mayer-Davis E J, Vitolins M Z, Carmichael S L, Hemphill S, Tsaroucha G, Rushing J, Levin S. Validity and reproducibility of a food frequency interview in a multi-cultural epidemiologic study. Ann Epidemiol 1999;9(5):314–24.

[9] Mellen P B, Gao S K, Vitolins M Z, Goff D C. Deteriorating dietary habits among adults with hypertension: DASH dietary accordance, NHANES 1988-1994 and 1999-2004. Arch Intern Med 2008;168(3):308–14.

[10] Cerwinske L, Rasmussen H, Lipson S, Volgman A, Tangney C C. Evaluation of a dietary screener: the Mediterranean eating pattern for Americans tool. J Hum Nutr Diet 2017;30(5):596–603.

[11] Sebastian S A, Shah Y, Paul H, Arsene C. Life's essential 8 and the risk of cardiovascular disease: a systematic review and meta-analysis. Eur J Prev Cardiol 2024:zwae280.

[12] Shetty N S, Gaonkar M, Patel N, Li P, Arora G, Arora P. Association of life's essential 8 and simple 7 scores with mortality: comparison with pooled cohort equation. JACC 2024;3(6):100945.

[13] Cappuccio F P, D'Elia L, Strazzullo P, Miller M A. Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. Sleep 2010;33(5):585–92.

[14] Zhao B, Meng Y, Jin X, Xi W, Ma Q, Yang J, Ma X, Yan B. Association of objective and self-reported sleep duration with all-cause and cardiovascular disease mortality: a community-based study. J Am Heart Assoc 2023;12(6):e027832.

[15] Windred D P, Burns A C, Lane J M, Saxena R, Rutter M K, Cain S W, Phillips A J. Sleep regularity is a stronger predictor of mortality risk than sleep duration: a prospective cohort study. Sleep 2024;47(1):zsad253.

[16] Javaheri S, Redline S. Insomnia and risk of cardiovascular disease. Chest 2017;152(2):435–44.

[17] You S, Zhang H F, Zhang S L, Gao Q Y, Cai Y W, Huang Z G, Wu Y B, Chen Y X, Wang J F, Gao J W, et al. Sleep patterns and traditional cardiovascular health metrics: joint impact on major adverse cardiovascular events in a prospective cohort study. J Am Heart Assoc 2024;13(9):e033043.

[18] Maiolino G, Bisogni V, Silvani A, Pengo MF, Lombardi C, Parati G. Treating sleep disorders to improve blood pressure control and cardiovascular prevention: a dream come true? a narrative review. J Thorac Dis 2020;12(2):S225–34.

[19] Everett B M. The confusing landscape of cardiovascular health assessment, promotion, and prediction. Circulation 2024;149(12):914–6.

[20] van Loon I G, van der Schouw Y T, Handoko M L, Verschuren W M, Uijl A. Comparing Life's Simple 7 and Life's Essential 8 with risk of heart failure. JACC 2025;4(10_Part_2):102127.