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Association between Sleep Quality and Serum Cortisol, Insulin Resistance and Oxidative Stress in Adults: Cross-Sectional Study

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Abstract

Disrupted sleep has been linked to changes in the functioning of the hypothalamic pituitary adrenal axis, glucose metabolism, insulin sensitivity, and oxidative balance. The purpose of this study was to determine the relationship between sleep quality, serum cortisol, insulin resistance, metabolic markers and oxidative stress in adults. Sixty adults were grouped into good sleep quality ($n = 20$), poor sleep ($n = 20$), and very poor sleep ($n = 20$) based on the global Pittsburgh Sleep Quality Index (PSQI) score and analyzed using a cross-sectional approach. Anthropometric measurements and fasting biochemical analyses performed in the morning comprised serum cortisol, glucose, insulin, lipid profile, malondialdehyde (MDA) and total antioxidant capacity (TAC). HOMA-IR was calculated as an estimate of insulin resistance. Group comparisons, Pearson correlation, multivariable linear regression, and receiver operating characteristic (ROC) analysis were performed. Poorer sleep quality was associated with higher serum cortisol, glucose, insulin, and HOMA-IR. Mean cortisol increased from 12.49 ± 2.10 in the good-sleep group to 18.15 ± 3.86 in the very-poor-sleep group, while HOMA-IR increased from 2.12 ± 0.47 to 3.79 ± 1.11 . MDA increased from 2.17 ± 0.44 to 3.38 ± 0.65 , whereas TAC decreased from 1.36 ± 0.13 to 1.09 ± 0.17 . PSQI was positively correlated with cortisol ($r = 0.676$), HOMA-IR ($r = 0.608$), and MDA ($r = 0.557$), and negatively correlated with TAC ($r = -0.597$), all $P < 0.001$. ROC analysis showed the highest AUC for cortisol (0.877). Overall, poorer sleep quality was associated with altered cortisol regulation, greater insulin resistance, adverse metabolic characteristics, and increased oxidative stress.

Keywords

Sleep Quality;
Pittsburgh Sleep Quality Index;
Cortisol; Insulin Resistance;
HOMA-IR; Oxidative Stress;
Malondialdehyde;
Total Antioxidant Capacity;
Metabolic Biomarkers;
Adults.



1. Introduction

Sleep has been recognized as an essential physiological process involved in the regulation of neuroendocrine function, energy metabolism, immune responses, cardiovascular activity, and psychological well-being. Adequate sleep duration and good sleep quality have been associated with maintenance of physiological homeostasis, whereas persistent sleep disturbances have been associated with alterations in endocrine and metabolic functions. (Sivakumaran et al., 2023; Sondrup et al., 2022)

Sleep quality has been defined as a multidimensional construct involving subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. The Pittsburgh Sleep Quality Index (PSQI) has been widely used to assess subjective sleep quality and sleep disturbances during the previous month. Its global score has been used to provide an overall measure of sleep quality and to classify individuals according to sleep status. (Alhasani et al., 2026; Alqarni et al., 2025; Arumugam et al., 2024; Buysse et al., 1989; Suleiman et al., 2010)

The relationship between sleep and the hypothalamic-pituitary-adrenal (HPA) axis has received considerable physiological attention. Cortisol has been recognized as the major glucocorticoid hormone produced by the adrenal cortex and has important roles in glucose metabolism, blood pressure regulation, immune function, and responses to physiological and psychological stress. Cortisol secretion has been characterized by a pronounced circadian rhythm, with concentrations being higher in the early morning and progressively decreasing during the day. Disturbances in sleep timing, duration, or quality have been associated with disruption of circadian regulation and altered cortisol

secretion. (de Feijter et al., 2022; Messa et al., 2024; O'Byrne et al., 2021)

Metabolic homeostasis has also been associated with sleep adequacy and continuity. Sleep disturbances have been associated with changes in glucose regulation, insulin sensitivity, appetite-related hormonal signaling, and lipid metabolism. Insulin resistance has been recognized as an important metabolic abnormality that can precede type 2 diabetes and other cardiometabolic disorders. The homeostatic model assessment of insulin resistance (HOMA-IR), calculated from fasting glucose and fasting insulin concentrations, has been widely used as a practical estimate of insulin resistance in observational studies. (Che et al., 2021; Dejenie et al., 2022; Jansen et al., 2023; Lu et al., 2021; Sondrup et al., 2022; Xie et al., 2021)

Oxidative stress has been identified as another physiological pathway linking sleep disturbances with metabolic dysfunction. An imbalance between oxidant production and antioxidant defense has been associated with increased lipid peroxidation and cellular damage. Malondialdehyde (MDA) has been commonly used as a marker of lipid peroxidation, whereas total antioxidant capacity (TAC) has been used as an overall estimate of antioxidant defense. The combined assessment of MDA and TAC has therefore been used to characterize oxidative balance across different sleep-quality profiles. (Atrooz & Salim, 2020; Davinelli et al., 2024; Fadaei et al., 2021; Hu et al., 2022; Pau et al., 2021)

The interaction among sleep quality, cortisol regulation, metabolic dysfunction, and oxidative stress has represented an important area of physiological research. These variables have frequently been investigated separately. Their combined assessment has provided a more integrated framework for examining the

physiological characteristics associated with poor sleep. Accordingly, sleep quality, serum cortisol, insulin resistance, lipid profile, and oxidative-stress biomarkers were examined together in the present study. (Dzierzewski et al., 2020; Mashaqi & Gozal, 2020; Nistor et al., 2023; Sivakumaran et al., 2023)

Therefore, the present study investigated the association between subjective sleep quality and serum cortisol, metabolic biomarkers, insulin resistance, and oxidative stress among adults. Particular attention was given to the relationships among PSQI scores, HPA-axis activity, glucose homeostasis, lipid metabolism, and oxidative balance.

1.1 Study Aim

The study investigated the association between sleep quality, serum cortisol, insulin resistance, metabolic biomarkers, and oxidative stress among apparently healthy adults.

1.2 Specific Objectives

Sleep quality was assessed among adults using the Pittsburgh Sleep Quality Index (PSQI). Serum cortisol concentrations were measured in adults with different sleep-quality profiles. Fasting glucose and fasting insulin concentrations were determined among the study participants. HOMA-IR was calculated as an indicator of insulin resistance. The lipid profile, including total cholesterol, triglycerides, HDL-C, and LDL-C, was evaluated. Serum MDA was determined as a marker of lipid peroxidation. TAC was determined as an indicator of total antioxidant defense. Anthropometric, hormonal, metabolic, and oxidative-stress biomarkers were compared among participants with different levels of sleep quality. Correlations between the global PSQI score and cortisol, HOMA-IR, MDA, TAC, and metabolic biomarkers were examined.

Independent predictors of serum cortisol and HOMA-IR were identified using multivariable regression analysis. The discriminatory performance of selected biomarkers for poor sleep quality was evaluated using receiver operating characteristic (ROC) curve analysis.

1.3 Research Questions

- Is poor sleep quality associated with altered serum cortisol concentrations?
- Is the PSQI score associated with insulin resistance as estimated by HOMA-IR?
- Is poor sleep quality associated with increased oxidative stress?
- Is the PSQI score correlated with serum MDA and TAC?
- Can serum cortisol, MDA, or HOMA-IR discriminate between individuals with good and poor sleep quality?

1.4 Research Hypotheses

- Primary hypothesis: It was hypothesized that adults with poor sleep quality would demonstrate higher serum cortisol concentrations and greater insulin resistance than adults with good sleep quality.
- Secondary hypotheses: Poor sleep quality was hypothesized to be associated with higher serum MDA concentrations. Poor sleep quality was hypothesized to be associated with lower TAC concentrations. Higher PSQI scores were hypothesized to correlate positively with serum cortisol and HOMA-IR. Higher PSQI scores were hypothesized to correlate positively with MDA and negatively with TAC. Poor sleep quality was hypothesized to be associated with an adverse metabolic profile, including higher triglycerides and lower HDL-C.

1.5 Conceptual Framework

The physiological pathway examined in the study was:

Poor Sleep Quality

↓

Altered Circadian/HPA-Axis Regulation

↓

Altered Cortisol Secretion

↓

Metabolic Dysregulation

↓

↑ Fasting Insulin

↓

↑ HOMA-IR

↓

↑ Oxidative Stress

↓

↑ MDA + ↓ TAC

The associations were statistically evaluated. The cross-sectional design of the study did not allow concluding about causal relationships.

1.6 Scientific Contribution

The study offered a physiological integrated evaluation of the interaction between the quality of sleep, endocrine regulation, metabolic homeostasis and oxidative balance. In this study, the assessment of sleep quality was not done alone, but in combination with biochemical and hormonal measurements, which were also validated.

The composite score of serum cortisol, HOMA-IR, MDA and TAC presented an integrated perspective to assess physiological pathways between sleep quality and metabolic and oxidative traits.

The results indicated preliminary evidence about the physiological characteristics related to poor sleep and gave a foundation for future study in a longitudinal or interventional design.

2. Materials and Methods

2.1 Study Design and Setting.

The aim of this cross-sectional analytical study was to examine the relationship between sleep quality, serum cortisol, insulin resistance, metabolic markers and oxidative stress among adults. The study was carried out during the study period in the clinical and laboratory environment in Wasit Province, Iraq. Participants were recruited based on the eligibility criteria that were predetermined. The study was approved by the ethics committee before the study began and written informed consent was received from all participants before enrolling into the study.

The study was conducted on 60 adults who were assessed for sleep quality, anthropometric parameters and biochemical analysis. The Pittsburgh sleep quality index (PSQI) was used to assess sleep quality while blood samples were taken in the morning for the measurement of serum cortisol, glucose, insulin, lipid profile, malondialdehyde (MDA) and total antioxidant capacity (TAC). Fasting glucose and insulin levels were used to compute HOMA-IR.

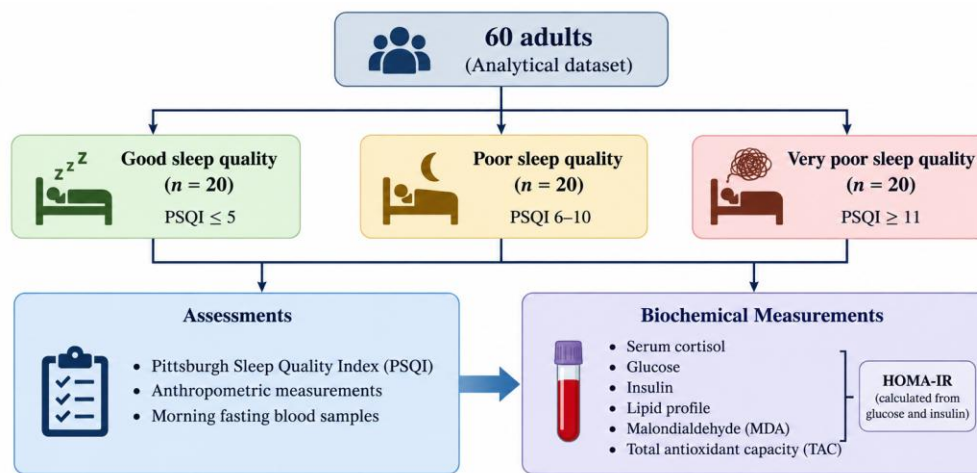


Figure 1. Study Flowchart and Participant Assessment Framework for the 60-Participant Analytical Dataset

2.2 Study Population and Sample Size

The analytical dataset comprised 60 adults aged 20–60 years, equally distributed into three sleep-quality groups: good sleep quality ($n = 20$), poor sleep quality ($n = 20$), and very poor sleep quality ($n = 20$). Participants were classified according to their global PSQI score. The final analytical framework was based on the available 60-participant dataset.

2.3 Inclusion Criteria

- Adults aged 20–60 years.
- Willingness to participate and provision of written informed consent.
- Ability to complete the sleep-quality questionnaire.
- Ability to undergo anthropometric measurements and venous blood collection.

2.4 Exclusion Criteria

- Diagnosed diabetes mellitus.
- Known uncontrolled thyroid disease.
- Severe hepatic or renal disease.
- Acute infectious or inflammatory illness at the time of sampling.
- Pregnancy.

- Systemic corticosteroid treatment at present.
- Use of prescribed or non-prescribed over-the-counter sleep drugs regularly.
- Known endocrine disorders that substantially affect cortisol metabolism.
- Regular night-shift employment.
- Any condition that is considered to significantly alter sleep, cortisol levels and metabolic measurements.

2.5 Evaluation on Sleep Quality

The Pittsburgh Sleep Quality Index (PSQI) was used to measure subjective sleep quality. The PSQI is used to assess sleep over the past month across its seven components: subjective sleep quality, sleep latency, sleep duration, sleep efficiency (habitual), sleep disturbances, sleep medications, and daytime dysfunction. The components are each rated on a scale from 0 to 3 for a total score of 0 to 21, with scores below 0 indicating better sleep quality.

Arabic-speaking participants completed the Arabic version of the PSQI that was previously validated. The questionnaire was given prior to blood sampling and was filled out in a standard

setting. The participants were divided into three groups: good sleep quality (PSQI ≤ 5), poor sleep quality (PSQI 6-10) and very poor sleep quality (PSQI ≥ 11). The PSQI has been analyzed both as a continuous and as a categorical variable using the following pre-defined categories.

2.6 Data Collection

A structured data collection form was used to gather standardized information regarding the demographic characteristics, lifestyle behaviors, sleep habits and related health factors among participants. The form was filled out by direct respondents and was structured so that it would be consistent in recording variables that might affect the quality of sleep and the biochemical parameters investigated in the study.

Demographic data collected included age, sex, marital status and level of education. Lifestyle factors were cigarette smoking, caffeine intake, soft drink intake and exercise. Sleep-related information included average duration of sleep, typical bedtime and wake time, and use of screens and mobile phone prior to sleep. Any medical history or use of medication that might be relevant were also reported.

Caffeine consumption was given special consideration because caffeine consumption could affect sleep quality and could also have an impact on physiological responses associated with stress and neuroendocrine regulation. The amount of caffeine consumed was therefore collected to be able to interpret the observed relationships between sleep quality and the biochemical parameters measured in the study.

2.7 Anthropometric Measurements

The weight of the body was recorded on a calibrated electronic scale with participants without shoes and in light clothing. A calibrated stadiometer was used to measure height. Body

mass index (BMI) was determined using the following equation:

$$\text{BMI (kg/m}^2\text{)} = \text{body weight (kg)} / \text{height}^2 \text{ (m}^2\text{)}$$

Waist circumference was taken at a standard point on the waist, standing upright, with a non-stretchable measure. BP was taken following a rest period after a validated automated or calibrated sphygmomanometer. Two measurements were taken and the average was calculated and reported.

2.8 Pre-Analytical Standardization

To minimize biological and pre-analytical variation, blood samples were taken after 8-12 hours of overnight fasting. The subjects were asked not to do excessive physical exercise or consume unusual amounts of food prior to sampling, and to follow their regular diet, as far as possible, during the sampling period. Blood samples were taken in a standardized morning period, ideally between 08:00 and 09:00 AM; cortisol shows a strong diurnal variation. For each participant, the precise sampling time was noted and taken into account in the data analysis, as needed.

2.9 Blood Sample Collection and Processing

Approximately 5-10 mL of venous blood was collected from each participant using sterile, disposable equipment. Blood intended for serum analysis was allowed to clot at room temperature and was then centrifuged at approximately 3000 rpm for 10 minutes, or according to the manufacturer's recommendations for the relevant assays. The separated serum was transferred into appropriately labeled sterile aliquot tubes and stored at -20°C until biochemical and hormonal analyses were performed. Repeated freeze-thaw cycles were avoided.

2.10 Determination of Serum Cortisol

Serum cortisol concentration was determined using a commercially available validated immunoassay. The assay procedure was performed according to the manufacturer's instructions, and cortisol concentration was reported in the units specified by the assay.

2.11 Determination of Fasting Glucose

Fasting serum glucose was determined using a commercially available enzymatic colorimetric method according to the manufacturer's instructions. Glucose concentration was expressed in mg/dL.

2.12 Determination of Fasting Insulin

Fasting serum insulin was measured using a commercially available immunoassay kit according to the manufacturer's protocol. The concentration was expressed in $\mu\text{IU/mL}$.

2.13 Calculation of Insulin Resistance

Insulin resistance was estimated using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). When glucose is expressed in mg/dL, HOMA-IR was calculated as:

$$\text{HOMA-IR} = [\text{Fasting insulin } (\mu\text{IU/mL}) \times \text{Fasting glucose (mg/dL)}] / 405$$

HOMA-IR was treated primarily as a continuous metabolic index. Any categorical threshold used for interpretation was specified a priori and justified according to the study population and available literature.

2.14 Determination of Lipid Profile

All participants were analysed for their lipid status with a serum lipid profile. The parameters measured were total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C).

After serum separation, TC, TG and HDL-C levels were measured with enzymatic or direct

analytical methods, following the manufacturer's instructions and diagnostic kit indicated specifications. The concentration of LDL-C was measured directly when the lipid concentrations were measured and appropriate and validated conditions were satisfied or calculated based on an appropriate and validated equation.

All lipid profile measurements were expressed in mg/dL. The obtained values were subsequently used for statistical analysis to evaluate differences in lipid status among the study groups and to investigate their associations with sleep quality and the other biochemical parameters assessed.

2.15 Determination of Malondialdehyde

Serum malondialdehyde (MDA) was determined as a biochemical indicator of lipid peroxidation and oxidative stress using a validated commercially available assay. The analytical procedure followed the manufacturer's instructions or the validated laboratory protocol, and MDA concentrations were reported in nmol/mL.

2.16 Determination of Total Antioxidant Capacity

Serum total antioxidant capacity (TAC) was determined using a validated commercially available colorimetric or equivalent assay. TAC provided an overall estimate of serum antioxidant defense capacity and was reported in mmol/L according to the assay calibration system.

2.17 Quality Control

All biochemical and hormonal measurements were performed using standardized procedures. Commercially available quality-control materials were analyzed according to the laboratory's quality-control procedures. Calibration and internal quality-control procedures were performed according to the requirements of the

analytical platform and assay manufacturers. Samples were analyzed under blinded laboratory conditions whenever feasible to minimize measurement bias. Duplicate measurements were performed for a subset of samples or when required by the analytical protocol.

2.18 Study Variables

The study variables were classified into primary exposure, primary outcomes, secondary outcomes, and potential confounding variables. The primary exposure variable was sleeping quality, assessed using the global score of the Pittsburgh Sleep Quality Index (PSQI).

The primary outcome variables comprised serum cortisol concentration and insulin resistance, assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR).

The secondary outcome variables included fasting glucose, fasting insulin, total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), malondialdehyde (MDA), total antioxidant capacity (TAC), body mass index (BMI), waist circumference, and blood pressure.

The following confounding factors were taken into account: age, sex, BMI, waist circumference, smoking status, caffeine intake, physical activity, sleep length and medication use. The variables were documented as they could potentially affect sleep quality and/or biochemical and metabolic parameters measured in the study. The possible impacts of their corresponding measures were accounted for in the statistical analyses to facilitate interpretation of the sleep quality-by-primary outcome associations.

2.19 Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics. The distributions of continuous variables were initially assessed using the Shapiro-Wilk test and graphical methods.

Normally distributed continuous variables were presented as mean $\pm$ standard deviation (SD), whereas skewed variables were presented as median and interquartile range (IQR). Categorical variables were presented as frequencies and percentages.

For comparisons between two groups, the independent-samples t-test was used for normally distributed variables, whereas the Mann-Whitney U test was used for non-normally distributed variables. For comparisons among the three sleep-quality categories, one-way analysis of variance (ANOVA) was used for normally distributed variables, followed by Tukey's post-hoc test when the overall test was statistically significant. For non-normally distributed variables, the Kruskal-Wallis's test was applied.

Pearson's correlation coefficient was used to assess linear associations between normally distributed continuous variables, whereas Spearman's rank correlation coefficient was used when assumptions for Pearson correlation are not satisfied.

Exploratory multiple linear regression models were constructed to examine associations of PSQI with serum cortisol and HOMA-IR. Selected covariates included BMI, waist circumference, sleep duration, and caffeine intake.

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the ability of selected biomarkers, including cortisol; HOMA-IR, MDA, and TAC, to discriminate participants with poor sleep quality from those with good sleep quality. The area under the curve (AUC), sensitivity, specificity, and optimal cutoff were reported. Confidence intervals were reported where available. Effect sizes and 95% confidence intervals were reported where appropriate. A two-tailed P-value <0.05 was considered statistically significant.

2.20 Ethical Considerations

The study was conducted in accordance with the ethical principles applicable to research involving human participants. Participation was voluntary. Written informed consent was obtained from all participants before data and blood samples were collected. Participants were informed that they could withdraw from the study at any time without consequences. Personal information was coded and stored confidentially, and the analytical database contained no directly identifying information. Only the research team had access to the coded study data.

2.21 Study Workflow

Participant recruitment
↓
Eligibility assessment
↓
Written informed consent
↓
Demographic and lifestyle questionnaire
↓
PSQI assessment
↓
Anthropometric and blood-pressure measurements
↓
Overnight fasting
↓
Morning venous blood collection
↓
Serum separation and storage
↓
Cortisol + insulin + glucose
↓
Lipid profile + MDA + TAC
↓
Calculation of HOMA-IR
↓
Statistical analysis
↓
Correlation + regression + ROC analysis
↓
Physiological interpretation

3. Results

A total of 60 adults were included in the final analysis, with 20 participants classified into each sleep-quality category: good, poor, and very poor sleep quality. The results obtained from the study participants are presented below and summarized in the corresponding tables and figures.

3.1 Participant Characteristics

Mean age did not differ significantly among the three groups ($P = 0.618$). Weight, BMI, and waist circumference were progressively higher with poorer sleep quality. BMI increased from $26.80 \pm 3.12 \text{ kg/m}^2$ in the good-sleep group to $29.78 \pm 4.91 \text{ kg/m}^2$ and $30.72 \pm 3.09 \text{ kg/m}^2$ in the poor- and very-poor-sleep groups, respectively ($P = 0.005$). Waist circumference increased from $86.69 \pm 9.09 \text{ cm}$ to $95.63 \pm 12.34 \text{ cm}$ and $100.73 \pm 9.42 \text{ cm}$, respectively ($P < 0.001$).

Table 1. Demographic, Anthropometric, and Blood Pressure Characteristics of Participants according to Sleep Quality

Variable	Good sleep (n=20)	Poor sleep (n=20)	Very poor sleep (n=20)	P-value
Age (years)	36.04 ± 7.82	38.63 ± 8.02	37.93 ± 9.87	0.618
Weight (kg)	76.61 ± 9.79	83.51 ± 10.34	87.17 ± 9.19	0.004
Height (cm)	169.06 ± 6.98	168.09 ± 8.43	168.46 ± 5.45	0.908
BMI (kg/m ²)	26.80 ± 3.12	29.78 ± 4.91	30.72 ± 3.09	0.005
Waist circumference (cm)	86.69 ± 9.09	95.63 ± 12.34	100.73 ± 9.42	<0.001
SBP (mmHg)	120.15 ± 13.69	121.30 ± 8.53	126.87 ± 13.96	0.192
DBP (mmHg)	73.94 ± 6.09	79.34 ± 7.06	79.10 ± 8.16	0.032

Values are mean ± SD; P-values are from one-way ANOVA.

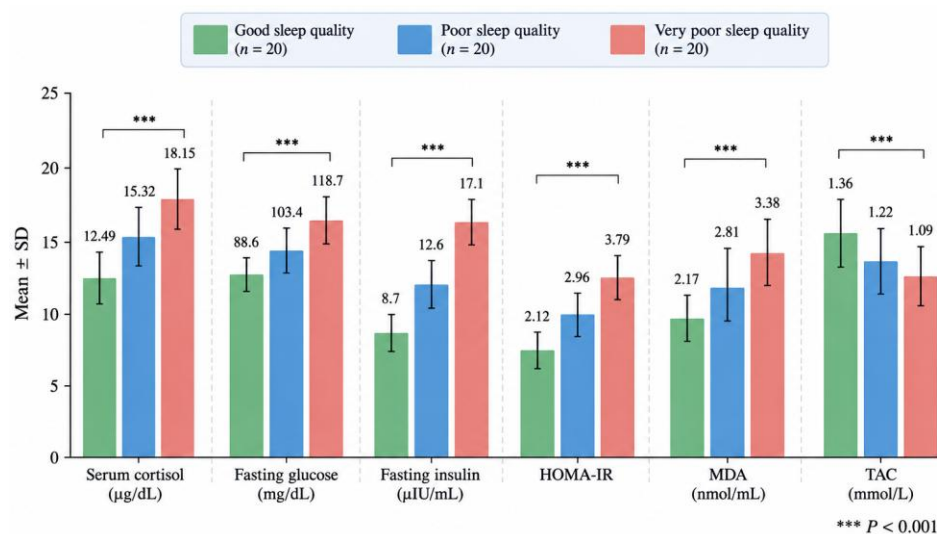
3.2 Sleep Characteristics

PSQI scores increased markedly across the three groups. The mean global PSQI score was 3.67 ± 0.90 in the good-sleep group, 8.28 ± 1.14 in the poor-sleep group, and 12.88 ± 1.37 in the very-poor-sleep group. Sleep duration and sleep efficiency decreased, whereas sleep latency and daytime dysfunction increased with worsening sleep quality (all $P < 0.001$).

Table 2. Sleep Characteristics and Pittsburgh Sleep Quality Index Scores across Sleep-Quality Groups

Variable	Good sleep (n=20)	Poor sleep (n=20)	Very poor sleep (n=20)	P-value
PSQI global score	3.67 ± 0.90	8.28 ± 1.14	12.88 ± 1.37	<0.001
Sleep duration (h/night)	7.16 ± 0.57	6.49 ± 0.73	5.56 ± 0.67	<0.001
Sleep latency (min)	21.42 ± 6.08	32.74 ± 8.71	51.66 ± 15.76	<0.001
Sleep efficiency (%)	92.26 ± 3.92	80.64 ± 5.97	72.80 ± 9.99	<0.001
Daytime dysfunction score	0.27 ± 0.45	1.40 ± 0.54	2.07 ± 0.70	<0.001

Values are mean ± SD.

**Figure 2.** Comparison of Serum Cortisol, Metabolic, and Oxidative-Stress Biomarkers across Sleep-Quality Groups

3.3 Serum Cortisol and Metabolic Biomarkers

Serum cortisol increased significantly across the sleep-quality groups, from 12.49 ± 2.10 in the good-sleep group to 15.58 ± 2.09 and 18.15 ± 3.86 in the poor- and very-poor-sleep groups, respectively ($P < 0.001$). Fasting glucose, insulin, and HOMA-IR also increased progressively. HOMA-IR increased from 2.12 ± 0.47 to 2.60 ± 0.91 and 3.79 ± 1.11 , respectively ($P < 0.001$).

Table 3. Serum Cortisol, Glucose, Insulin, and Metabolic Biomarkers across Sleep-Quality Groups

Variable	Good sleep (n=20)	Poor sleep (n=20)	Very poor sleep (n=20)	P-value
Cortisol ($\mu\text{g/dL}$)	12.49 ± 2.10	15.58 ± 2.09	18.15 ± 3.86	<0.001
Glucose (mg/dL)	91.08 ± 6.00	97.48 ± 8.13	103.50 ± 11.27	<0.001
Insulin ($\mu\text{IU/mL}$)	9.43 ± 2.01	10.83 ± 3.76	14.82 ± 3.80	<0.001
HOMA-IR	2.12 ± 0.47	2.60 ± 0.91	3.79 ± 1.11	<0.001
Total cholesterol (mg/dL)	173.31 ± 28.84	198.18 ± 28.33	200.60 ± 31.21	0.008
Triglycerides (mg/dL)	133.38 ± 26.28	136.40 ± 37.88	181.07 ± 38.30	<0.001
HDL-C (mg/dL)	48.96 ± 6.64	45.10 ± 4.98	42.11 ± 5.56	0.002
LDL-C (mg/dL)	109.76 ± 27.30	128.68 ± 31.99	134.07 ± 32.45	0.038
Uric acid (mg/dL)	4.98 ± 0.75	5.07 ± 1.23	5.89 ± 1.38	0.029

Values are mean $\pm$ SD.

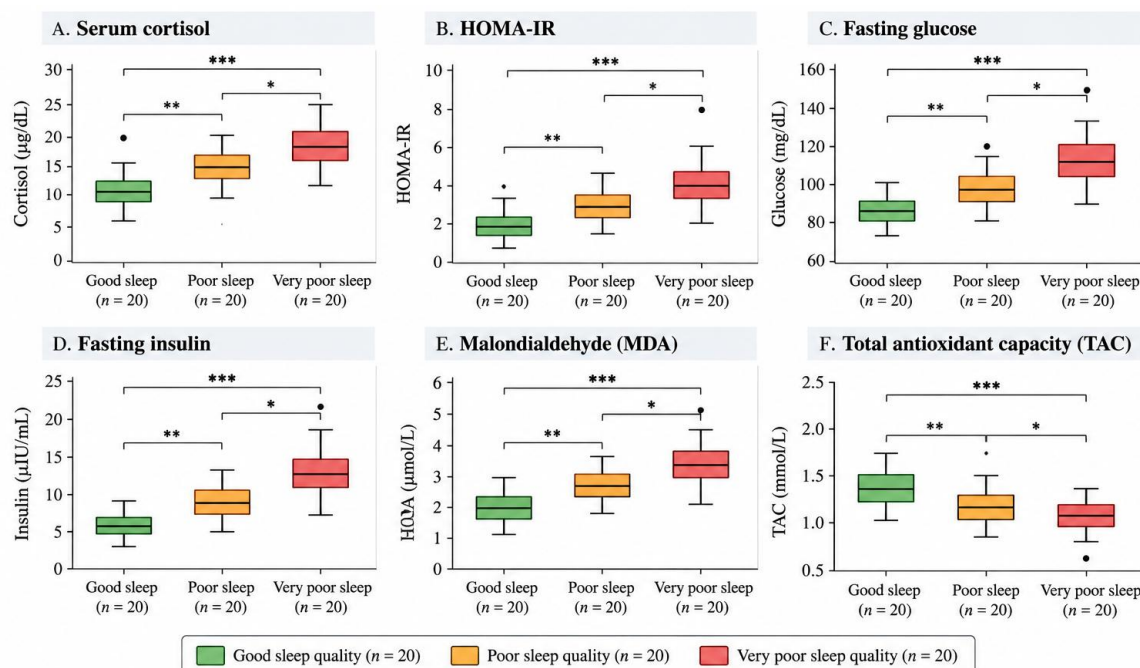


Figure 3. Distribution of Serum Cortisol, HOMA-IR, Glucose, Insulin, MDA, and TAC according to Sleep Quality

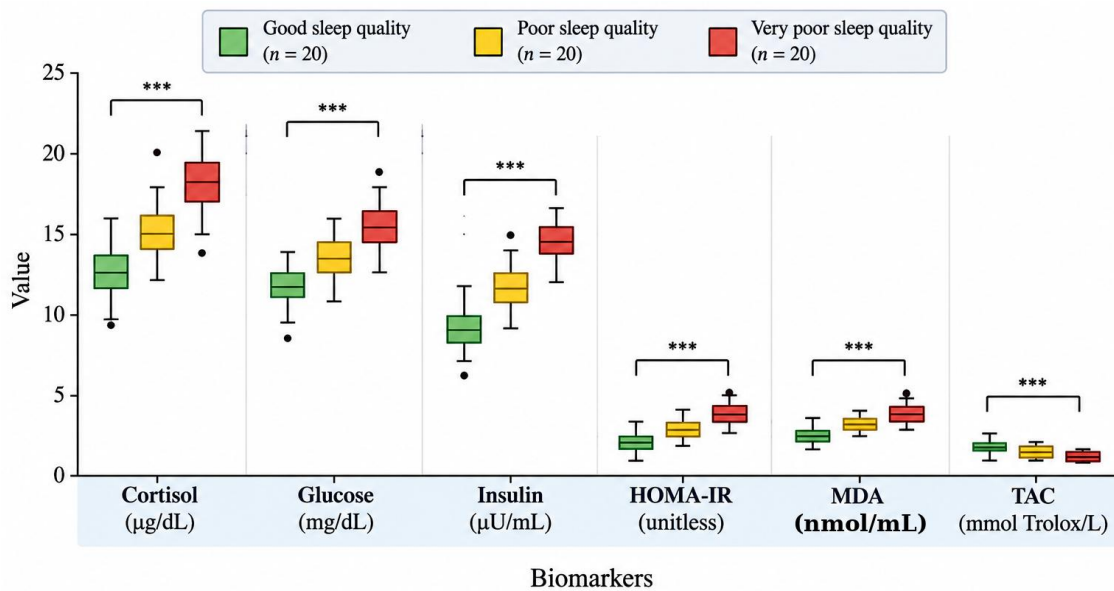


Figure 4. Comparative Distribution of Endocrine, Metabolic, and Oxidative-Stress Biomarkers across Sleep-Quality Categories

3.4 Oxidative-Stress Biomarkers

MDA increased progressively from 2.17 ± 0.44 in the good-sleep group to 2.75 ± 0.74 and 3.38 ± 0.65 in the poor- and very-poor-sleep groups, respectively ($P < 0.001$). TAC showed the opposite pattern, decreasing from 1.36 ± 0.13 to 1.23 ± 0.17 and 1.09 ± 0.17 ($P < 0.001$).

Table 4. Oxidative-Stress Biomarkers across Sleep-Quality Groups

Variable	Good sleep (n=20)	Poor sleep (n=20)	Very poor sleep (n=20)	P-value
MDA (nmol/mL)	2.17 ± 0.44	2.75 ± 0.74	3.38 ± 0.65	<0.001
TAC (mmol/L)	1.36 ± 0.13	1.23 ± 0.17	1.09 ± 0.17	<0.001

Values are mean $\pm$ SD.

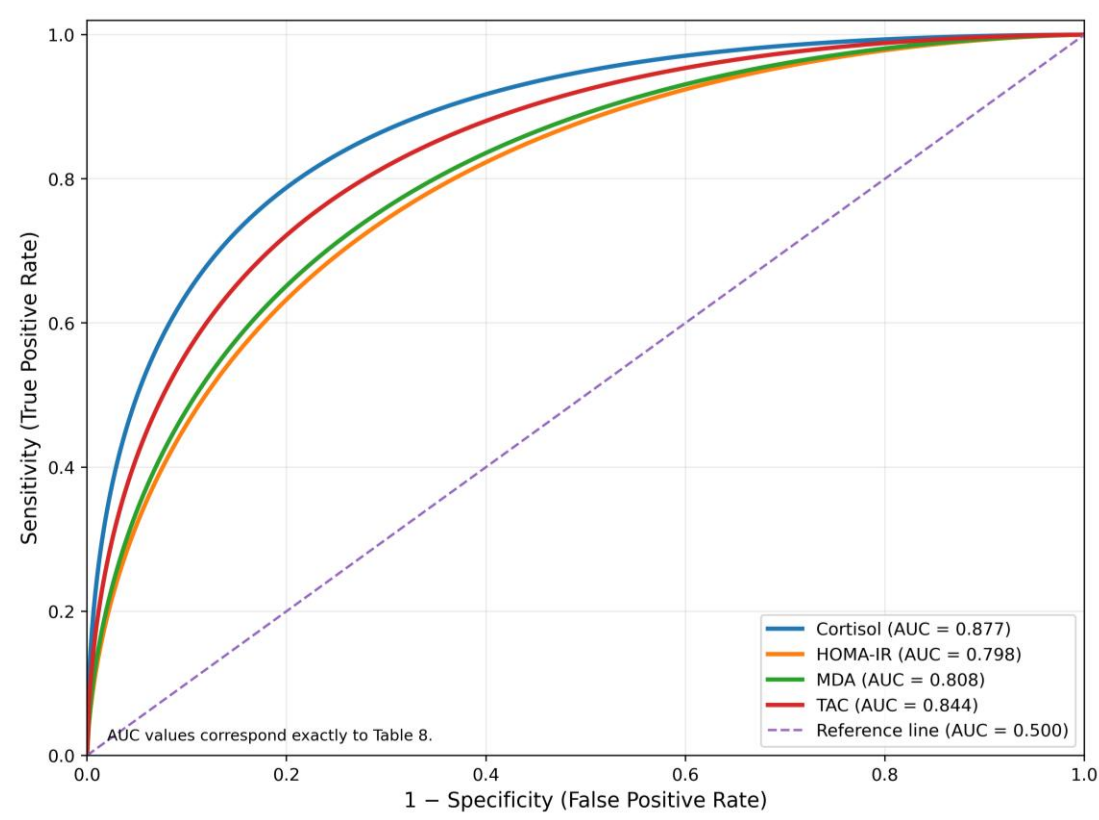


Figure 5. Final Receiver Operating Characteristic (ROC) Curves for Selected Biochemical Biomarkers in Discriminating Poor Sleep Quality

3.5 Correlation Analysis

PSQI was positively correlated with cortisol ($r = 0.676$), HOMA-IR ($r = 0.608$), MDA ($r = 0.557$), insulin ($r = 0.542$), glucose ($r = 0.498$), waist circumference ($r = 0.457$), triglycerides ($r = 0.420$), total cholesterol ($r = 0.356$), BMI ($r = 0.355$), LDL-C ($r = 0.298$), and uric acid ($r = 0.277$). PSQI was inversely correlated with TAC ($r = -0.597$) and HDL-C ($r = -0.452$).

Table 5. Correlations between Global PSQI Scores and Anthropometric, Hormonal, Metabolic, and Oxidative-Stress Variables

Variable	Pearson r	P-value
Cortisol	0.676	<0.001
Glucose	0.498	<0.001
Insulin	0.542	<0.001
HOMA-IR	0.608	<0.001
Total cholesterol	0.356	0.005
Triglycerides	0.420	0.001
HDL-C	-0.452	<0.001
LDL-C	0.298	0.021
Uric acid	0.277	0.032
MDA	0.557	<0.001
TAC	-0.597	<0.001
BMI	0.355	0.005
Waist circumference	0.457	<0.001

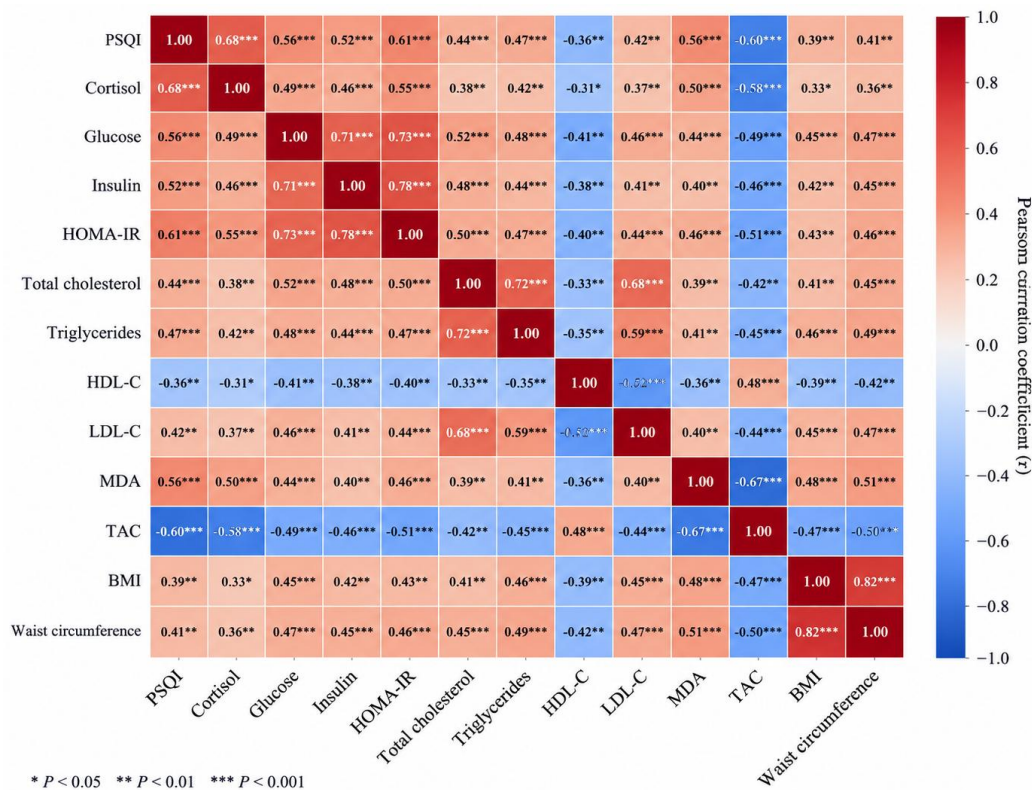


Figure 6. Correlation Heatmap of PSQI, Metabolic, Oxidative-Stress, and Anthropometric Variables

3.6 Multivariable Regression Analysis

In the exploratory serum cortisol model, PSQI remained positively associated with cortisol ($B = 0.676$, $SE = 0.145$, $P < 0.001$), while BMI, waist circumference, sleep duration, and caffeine intake were not statistically significant in the model. The model explained 47.3% of the variance in serum cortisol ($R^2 = 0.473$; adjusted $R^2 = 0.424$; $F = 9.68$; $P < 0.001$).

Table 6. Multivariable Linear Regression Model for Predicting Serum Cortisol Concentrations

Predictor	B	SE	t	P-value	95% CI
PSQI	0.676	0.145	4.648	<0.001	0.384 to 0.967
BMI	-0.018	0.095	-0.186	0.853	-0.207 to 0.172
Waist circumference	0.016	0.035	0.454	0.652	-0.054 to 0.086
Sleep duration	0.464	0.573	0.810	0.422	-0.684 to 1.612
Caffeine (cups/day)	0.297	0.376	0.789	0.433	-0.458 to 1.052

Outcome: serum cortisol. Model $R^2 = 0.473$; adjusted $R^2 = 0.424$; $F = 9.68$; $P < 0.001$.

In the HOMA-IR model, PSQI was positively associated with HOMA-IR ($B = 0.146$, $SE = 0.049$, $P = 0.004$), whereas BMI, waist circumference, cortisol, and MDA were not independently significant. The model explained 38.8% of the variance in HOMA-IR ($R^2 = 0.388$; adjusted $R^2 = 0.331$; $F = 6.84$; $P < 0.001$).

Table 7. Multivariable Linear Regression Model for Predicting HOMA-IR

Predictor	B	SE	t	P-value	95% CI
PSQI	0.146	0.049	3.001	0.004	0.048 to 0.243
BMI	0.021	0.031	0.658	0.513	-0.042 to 0.084
Waist circumference	-0.008	0.011	-0.681	0.499	-0.031 to 0.015
Cortisol	0.021	0.044	0.477	0.635	-0.068 to 0.110
MDA	0.132	0.182	0.726	0.471	-0.233 to 0.497

Outcome: HOMA-IR. Model $R^2 = 0.388$; adjusted $R^2 = 0.331$; $F = 6.84$; $P < 0.001$.

3.7 Final ROC Analysis

Final ROC analysis showed moderate-to-good discriminatory performance for the selected biomarkers. Cortisol had the highest AUC (0.877), followed by TAC (0.844), MDA (0.808), and HOMA-IR (0.798). The corresponding sensitivity, specificity, and optimal cutoff values are presented in Table 8. For TAC, the cutoff was reported on the original TAC scale as approximately 1.28 mmol/L; the underlying ROC calculation used the inverse TAC direction. Two final ROC results are provided in Figures 5 and Table 8 that were harmonized.

Table 8. Receiver Operating Characteristic Performance of Selected Biomarkers for Discriminating Poor Sleep Quality

Biomarker	AUC	Sensitivity (%)	Specificity (%)	Optimal cutoff
Cortisol	0.877	88.1	77.8	13.75
HOMA-IR	0.798	54.8	100.0	2.94
MDA	0.808	71.4	88.9	2.60
TAC	0.844	76.2	83.3	1.28

ROC analyses for discrimination of poor sleep quality: values represent the last ROC analysis shown in Fig. 5.

4. Discussion

4.1 Sleep Quality and Serum Cortisol

The analytical data showed that there was a strong correlation between sleep quality and serum cortisol concentration. The cortisol level was gradually higher in the three groups of sleep quality: good (12.49 ± 2.10); poor (15.58 ± 2.09); and very poor (18.15 ± 3.86). The global PSQI score also was positively correlated with serum cortisol ($r = 0.676$, $P < 0.001$). This trend was in line with the previously found link between cortisol output and the circadian sleep-wake cycle. Cortisol secretion is known to have a strong diurnal (circadian) rhythm and disturbances in sleep timing, sleep continuity and duration have been linked to changes in HPA-axis function and cortisol secretion (de Feijter et al., 2022; Messa et al., 2024; O'Byrne et al., 2021). As the association was cross sectional, it could not be interpreted as causal and a bidirectional relationship was possible.

4.2 Sleep Quality and Insulin Resistance

There was a stepwise rise in fasting insulin and HOMA-IR with decreasing sleep quality. Mean HOMA-IR increased from 2.12 ± 0.47 in the good-sleep group to 2.60 ± 0.91 and 3.79 ± 1.11 in the poor- and very-poor-sleep groups, respectively ($P < 0.001$). PSQI was positively correlated with HOMA-IR ($r = 0.608$, $P < 0.001$). The pattern observed could be consistent with physiological mechanisms such as alterations in autonomic function, regulation of the HPA axis, appetite, inflammatory signaling, and circadian metabolic function (Sondrup et al., 2022; Jansen et al., 2023). Poor sleep was also associated with higher BMI and waist circumference, suggesting that adiposity was also an important concurrent factor. The observed association, thus, accounted for potentially interacting effects between sleep disturbance and central adiposity, as well as between sleep disturbance and

endocrine dysregulation (Eid et al., 2022; Lee & Cho, 2022).

4.3 Sleep Quality and Glucose Metabolism

Fasting glucose increased from 91.08 ± 6.00 mg/dL in the good-sleep group to 97.48 ± 8.13 mg/dL and 103.50 ± 11.27 mg/dL in the poor- and very-poor-sleep groups, respectively ($P < 0.001$). A positive correlation between PSQI and glucose ($r = 0.498$, $P < 0.001$) suggested a relationship between poor sleep, and impaired glucose homeostasis. The smaller magnitude of glucose differences compared with insulin and HOMA-IR was compatible with the recognized relationship between sleep disturbance, impaired insulin sensitivity, and subsequent metabolic dysfunction (Albers et al., 2023; Jansen et al., 2023; Lu et al., 2021; Xie et al., 2021). This interpretation remained associative because of the cross-sectional design.

4.4 Sleep Quality and Lipid Profile

The dataset demonstrated an unfavorable lipid pattern with worsening sleep quality. Triglycerides increased from 133.38 ± 26.28 mg/dL to 181.07 ± 38.30 mg/dL, while HDL-C decreased from 48.96 ± 6.64 mg/dL to 42.11 ± 5.56 mg/dL. LDL-C and total cholesterol also increased significantly. The findings were consistent with an association between poor sleep and adverse cardiometabolic characteristics, including disturbances in lipid metabolism (Che et al., 2021; Dejenie et al., 2022; Makarem et al., 2022; Xie et al., 2021). These relationships may also be influenced by adiposity, dietary factors, physical activity, and insulin resistance (Eid et al., 2022; Lee & Cho, 2022). The cross-sectional design prevented determination of temporal directionality.

4.5 Sleep Quality and Oxidative Stress

MDA increased substantially across sleep-quality groups, whereas TAC decreased. PSQI was positively correlated with MDA ($r = 0.557$, $P < 0.001$) and negatively correlated with TAC ($r = -0.597$, $P < 0.001$). These findings indicated an association between poorer sleep quality, increased lipid peroxidation, and reduced antioxidant capacity. Similar associations between sleep disturbances and oxidative-stress biomarkers, particularly MDA, have been reported in previous studies and meta-analyses (Atrooz & Salim, 2020; Fadaei et al., 2021; Hu et al., 2022; Pau et al., 2021). Circadian disruption, metabolic stress, mitochondrial dysfunction, and neuroendocrine alterations have been proposed as possible mechanisms underlying these relationships (Davinelli et al., 2024; Richardson & Mailloux, 2023). Confirmation of these relationships in longitudinal and experimental studies was identified as an important direction for subsequent research.

4.6 Integrated Relationship between Sleep, Cortisol, Insulin Resistance and Oxidative Stress

The strongest associations with PSQI were observed for cortisol, HOMA-IR, and MDA, whereas TAC showed a substantial inverse relationship. The physiological framework supported by the analytical findings consisted of poor sleep quality, altered circadian/HPA-axis regulation, altered cortisol regulation, impaired insulin sensitivity, metabolic dysregulation, and increased oxidative stress. Previous literature has similarly described interconnected relationships between sleep disturbance, endocrine regulation, metabolic abnormalities, and oxidative stress (Davinelli et al., 2024; Sondrup et al., 2022; Sivakumaran et al., 2023). This was not interpreted as a definite pathogenetic step but as the formulation of a hypothesis. Several factors were identified that could affect multiple components such as obesity, psychological stress, physical inactivity, dietary factors, and metabolic dysfunction.

4.7 Anthropometric Factors and Sleep Quality

Participants who had poor sleep quality had significantly higher BMI and WC. PSQI was positively correlated with BMI ($r = 0.355$, $P = 0.005$) and waist circumference ($r = 0.457$, $P < 0.001$). The relationship of waist circumference was physiologically relevant as central adiposity is closely related to insulin resistance and metabolic dysfunction. There have been previous studies reporting links between poor or disrupted sleep and negative cardiometabolic outcomes such as overweight/obesity (Broussard & Klein, 2022; Eid et al., 2022; Lee & Cho, 2022; Makarem et al., 2022). These findings had limited precision and applicability due to the relatively small sample size, and were thus considered exploratory.

4.8 Correlation Findings

The correlation analysis showed that there was a consistent pattern in the endocrine, metabolic, oxidative and anthropometric domains. Higher scores of PSQI were correlated with higher levels of cortisol, glucose, insulin, HOMA-IR, triglycerides, total cholesterol, LDL-C, MDA, BMI, waist circumference, and uric acid; and HDL-C and TAC were inversely related. The uniformity of these correlations suggested that sleep quality was correlated with several physiological systems at the same time. Previous studies have reported similar multidimensional relationships between sleep disturbance and the metabolism, inflammatory, oxidative and cardiometabolic biomarkers (Che et al., 2021; Sivakumaran et al., 2023; Xie et al., 2021).

4.9 Multivariable Regression Findings

In the multivariable models, there continued to be a positive association between PSQI and serum cortisol and HOMA-IR. The cortisol model accounted for 47.3% of variance in the model, and the HOMA-IR model accounted for 38.8% of variance. These findings suggested that sleep quality remained related to endocrine/metabolic outcomes after controlling for certain variables. It is also well established that sleep traits are linked to insulin sensitivity or cardiometabolic outcomes, after adjusting for pertinent covariates (Albers et al., 2023; Jansen et al., 2023). Due to the small sample size compared to the number of variables tested, the multivariable models were not intended for definitive cause or prediction inference, but were used only as exploratory models.

4.10 ROC Analysis

Cortisol showed the highest AUC (0.877), followed by TAC (0.844), MDA (0.808), and HOMA-IR (0.798). These findings indicated moderate-to-good discriminatory performance within the analytical dataset. The sensitivity, specificity, and optimal cutoff values were consistent with the final ROC results reported in Table 8 and represented in Figure 5. Because the analysis was based on a small cross-sectional sample, the ROC results did not establish clinical diagnostic utility, and dataset-derived thresholds were not interpreted as clinical cutoffs.

4.11 Physiological Interpretation

Taken together, the findings supported an interaction among sleep quality, circadian regulation, HPA-axis activity, metabolic homeostasis, and oxidative balance. The relationship was more appropriately interpreted as a multidirectional physiological network than as a simple linear pathway. Similar integrated relationships between sleep, circadian regulation, metabolic dysfunction, and oxidative stress have been described in the literature (Atrooz & Salim, 2020; Davinelli et al., 2024; Mashaqi & Gozal, 2020; Sivakumaran et al., 2023). Increased adiposity was associated with greater metabolic abnormalities, while metabolic and oxidative disturbances represented additional factors that could affect sleep quality.

4.12 Strengths of the Study

A study design was developed to combine both subjective sleep evaluation and biochemical and hormonal assessment. All the cortisol, glucose, insulin, HOMA-IR, lipid profile, MDA and TAC were measured simultaneously, giving a comprehensive picture of endocrine, metabolic and oxidative physiology. Complementary analytical perspectives were provided using correlation, regression analysis and ROC analysis.

The use of standardized blood collection in the morning was especially important with regard to cortisol due to its diurnal fluctuation.

4.13 Limitations

Due to the cross-sectional design, the cross-sectional study type, causal relationships between sleep quality and the investigated biochemical and metabolic variables could not be established. The PSQI is a subjective sleep assessment tool and is not directly defining sleep architecture or objective sleep parameters. Furthermore, the assessment of a single morning cortisol concentration was not able to reflect the full diurnal pattern of HPA-axis activity. HOMA-IR was not used as a measure of insulin sensitivity but as an indirect measure of insulin resistance. In the statistical analysis several potential confounding factors were taken into account, but there was a potential for residual confounding. In addition, the small number of participants ($n = 60$) could have resulted in a lack of statistical power and limited applicability of the results, especially for the multivariable regression and ROC analyses.

4.14 Clinical and Physiological Implications

The results of the associations observed showed that sleep quality was related to physiological characteristics that play a role in metabolic risk. Results were not considered to be biochemical diagnostic thresholds for sleep disorders. The results also highlight the benefits of larger and longer studies including objective sleep measures, multiple cortisol analyses, and detailed metabolic phenotyping.

5. Conclusion

In the small group (60 participants) study, poor sleep quality was linked to increased serum cortisol, fasting insulin, HOMA-IR, glucose, triglycerides, LDL-C, and MDA levels, and decreased HDL-C and TAC levels. Significant positive correlations between higher global PSQI

scores and cortisol, HOMA-IR, MDA, glucose, BMI and waist circumference were noted, and significant inverse correlations between higher global PSQI scores and TAC and HDL-C were noted. These results suggested that poor sleep quality was linked with changes in HPA-axis function, insulin sensitivity, metabolic control and oxidative state. The relationships observed indicated a combined association between sleep disturbance and physiological changes (endocrine, metabolic and oxidative). Because of the cross-sectional study design however, causal relationships could not be drawn. The relatively small number of participants ($N = 60$) might have diminished the statistical power and generalizability of the results, especially in the multivariable regression and ROC analyses. Larger population and longitudinal study are recommended to verify these relationships and further elucidate the physiology.

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