

# Level of irisin differs importantly between severe and mild Obstructive Sleep Apnea Syndrome cases

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## Research Article

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# Abstract

## Background

Sleep is vital for cognitive function and metabolic health. Obstructive Sleep Apnea Syndrome (OSAS) involves recurrent respiratory arrest and hypoxemia. This study investigates the relationship between OSAS severity and levels of irisin—a myokine that regulates energy metabolism by converting white adipose tissue to brown fat.

## Materials and Methods

161 patients were categorized into four groups based on their Apnea-Hypopnea Index (AHI): Control (n = 8), Mild (n = 39), Moderate (n = 47), and Severe OSAS (n = 67). Following full-night polysomnography, serum irisin levels were measured via the ELISA method. Statistical analyses were performed using Kruskal-Wallis H and post-hoc Tukey tests ( $P < 0.05$ ).

## Results

Irisin concentration was significantly higher in the Mild OSAS group ( $0.26 \pm 0.07$ ) compared to the Control group ( $0.01 \pm 0.00$ );  $P < 0.05$ . Conversely, irisin levels in the Severe OSAS group ( $0.09 \pm 0.06$ ) were significantly lower than in the Mild group ( $P < 0.05$ ). Increased OSAS severity correlated with prolonged apnea-hypopnea durations during both REM and Non-REM sleep.

## Conclusion

Serum irisin levels exhibit a biphasic relationship with OSAS severity, characterized by an initial compensatory increase in mild cases followed by a progressive decline in severe stages, reflecting the impact of chronic hypoxia and muscle fatigue.

## Introduction

Sleep is an important physiological event for the renewal of the human body. Adequate sleep provides restoration and renewal in the human body [1]. It has also been determined that sleep provides benefits in terms of memory, thinking and ratiocination. However, the relationship between chronic sleep deprivation and diabetes, hypertension and vascular diseases has also been determined [2].

Obstructive Sleep Apnea Syndrome (OSAS) is a sleep disorder that causes hypoxemia and often awakenings along with respiratory arrest during sleep [3]. It is known that there is a relationship between OSAS which negatively affects sleep quality and other diseases [4].

Irisin was discovered as a myokine in muscle tissue and was shown to convert white fat tissue into brown fat tissue [5], [6]. Irisin is found in the blood at basal levels; its release from skeletal muscle into the blood increases with acute and intense exercise and binds to an as yet unidentified receptor in subcutaneous adipose tissue. In this situation white fat tissue converts brown fat tissue [7]. Thus irisin increases thermogenesis and contributes to weight control [8]. It increases energy expenditure and produces effects similar to exercise, such as improving metabolism, cardiovascular health and cognitive functions [9]. In obesity patients, plasma irisin levels are positively associated with fat mass, fatless tissue and insulin [10].

OSAS cases are closely related to obesity, fat mass and different metabolic dysfunctions [11]. Especially obese and morbidly obese people are important risk groups for OSAS [12]. Therefore, determining the levels of irisin which converts white fat tissue to brown fat tissue in these groups and examining the effect factors for OSAS will contribute significantly to the development of OSAS treatment methods in the future.

## **Materials and Methods**

### **A Working Group and Research Format**

This study was conducted in the Sleep Laboratory of Chest Diseases Clinic of Isparta City Hospital, Ministry of Health. Ethics committee approval was obtained with the decision numbered 106 and dated 20/07/2016 of the Ethics Committee of Süleyman Demirel University Faculty of Medicine. Data collection was carried out between 30/05/2016 and 30/12/2017. The study protocol was conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study. During the study, blood samples were taken from patients who applied to Isparta City Hospital Sleep Laboratory for OSAS diagnosis, treatment and gave informed consent, and PSG reports were recorded. Serum irisin levels were quantified using a commercially available ELISA kit (SunRed, Shanghai, Catalog number: 201-12-5328) in accordance with the manufacturer's instructions. The assay utilized a quantitative sandwich enzyme immunoassay technique. A standard curve was generated by plotting the mean absorbance (450 nm) for each standard against the corresponding concentration, and sample concentrations were calculated using a four-parameter logistic (4-PL) curve-fit. The intra-assay and inter-assay coefficients of variation (CV) for the ELISA kit were maintained below 8% and 12%, respectively, ensuring analytical reliability and test quality control.

The study was conducted with groups formed according to the apnea-hypopnea index (AHI), defined as the number of events per hour of sleep. Patients with an AHI  $\geq 5$  events/hour were diagnosed with OSAS, whereas the Control Group consisted of individuals with an AHI  $< 5$  events/hour. The OSAS patients were further stratified in accordance with the American Academy of Sleep Medicine (AASM) guidelines into three severity subgroups: mild (AHI of 5–15 events/h), moderate (AHI of 15–30 events/h), and severe OSAS (AHI of  $> 30$  events/h). Patients with position-dependent OSAS, Upper Respiratory Tract

Resistance Syndrome, Complex Sleep Apnea Syndrome and concomitant lung disease those diagnosed with rapid-eyes-movement (REM)-dependent OSAS were excluded from the study.

Demographic characteristics and disease information of all patients were recorded, and the reports of all patients' PSG records were examined; total sleep time, sleep onset time, sleep architecture (REM, Stage-1, Stage-2, Stage-3) and AHI polysomnography findings were evaluated.

## Polysomnography (PSG)

All patients underwent a comprehensive overnight polysomnographic (PSG) evaluation utilizing a 44-channel system (Compumedics, Abbotsford, VIC, Australia) configured in accordance with the international 10–20 system for electrode placement. The PSG setup incorporated electroencephalography (EEG), electrooculography (EOG) at a sampling rate of 256 mV/h, submental electromyography (EMG) filtered between 10–100 Hz, and electrocardiography (ECG) filtered between 0.3–30 Hz. Respiratory effort was monitored via piezoelectric chest and abdominal belts (filtered at 0.05–3 Hz), while airflow was assessed using a nasal cannula, and oxygen saturation alongside pulse rate was continuously tracked via a digital pulse oximeter, with motion sensors attached to both lower extremities to record leg movements with a 1–20 Hz filter. Polysomnograms were scored by two experienced chest physicians—each with a documented inter-rater concordance of 85–95% across 50 reference datasets and prior clinical experience of scoring at least 1,000 PSG studies—adopting the standard criteria established by Rechtschaffen and Kales. In these evaluations, apnea was defined as a complete cessation of airflow lasting ( $\geq 10$ ) seconds, desaturation was characterized by a ( $\geq 3\%$ ) drop in capillary oxygen saturation, and hypopnea was defined as a ( $\geq 50\%$ ) reduction in airflow lasting ( $\geq 10$ ) seconds accompanied by an arousal or desaturation. Furthermore, the oxygen desaturation index (ODI) was calculated by dividing the total number of scored desaturations by the estimated sleep duration (total time in bed minus wake time), and the diagnosis of obstructive sleep apnea syndrome (OSAS) was established when the apnea-hypopnea index (AHI) exceeded 5 events per hour, with disease severity stratified in accordance with the American Academy of Sleep Medicine (AASM) task force guidelines as mild OSAS (AHI of 5–15 events/h), moderate OSAS (AHI of 15–30 events/h), and severe OSAS (AHI of  $> 30$  events/h).

## Data Analysis

In the research process, data from 181 patients were obtained and 161 patients who met the research criteria were included in the study. Since the groups did not show a normal distribution, the groups were compared using the Kruskal-Wallis H test and post-hoc tukey using the SPSS statistical program.

Blood samples were measured using the irisin kit using the Enzyme-Linked Immunosorbent Assay (ELISA) method.

# Correlation Analysis

Spearman rank correlation analysis was performed to evaluate the degree and direction of monotonic relationships between serum irisin levels, polysomnographic parameters (including REM-AHI, Non-REM AHI, Supin-AHI, Hypopnea, Non-REM Apnea-Hypopnea, and Sleep Apnea-Hypopnea duration), and the Sleep-Wake Period. The strength of the correlation coefficient ( $r$ ) was defined as weak ( $0.10 \leq |r| < 0.30$ ), moderate ( $0.30 \leq |r| < 0.50$ ), or strong ( $r \geq 0.50$ ), with statistical significance set at ( $P < 0.05$ )

# Statistical Analysis

Data were analyzed using SPSS 21.0 for Windows (SPSS Inc., Chicago, IL, USA). The Kolmogorov-Smirnov test was used to assess the normal distribution of continuous variables. Continuous variables were expressed as mean (standard deviation), whereas categorical variables were reported as percentages. Categorical variables were compared using the Chi-squared test. The Kruskal-Wallis test followed by post-hoc Tukey tests was used for group comparisons. A P-value of  $< 0.05$  was considered statistically significant.

# Results

The mean age of the groups that underwent a single-night PSG at the Sleep Laboratory of the Chest Diseases Clinic of Isparta City Hospital was determined as ( $51.57 \pm 0.82$ ) years. In the same time 54.7% of the study population was male and 45.3% was female. The patients included in the study were divided into 4 groups according to disease severity (Table 1).

Table 1  
Experimental Groups

| Number of Groups | Name of Groups      | n  |
|------------------|---------------------|----|
| Group 1          | Control Group       | 8  |
| Group 2          | Mild OSAS Group     | 39 |
| Group 3          | Moderate OSAS Group | 47 |
| Group 4          | Severe OSAS Group   | 67 |

# Apnea Indexes

Among the OSAS groups included in the study, the apnea indexes and hypopnea index were obtained the highest in the Severe OSAS Group. Similarly, the other apnea indexes of the severe OSAS group were also at the highest level (Table 2).

Table 2  
Apnea Index Values

| Control Group       | Apnea             | Hypopnea            | Central Apnea      | Obstructive Apnea  | Total Apnea        |
|---------------------|-------------------|---------------------|--------------------|--------------------|--------------------|
|                     | <b>0,1 ± 0,08</b> | <b>16,37 ± 4,28</b> | <b>0,62 ± 0,49</b> | <b>0,12 ± 0,12</b> | <b>0,75 ± 0,62</b> |
| Mild OSAS Group     | 0,78 ± 0,16       | 70,23 ± 3,16        | 2,23 ± 0,46        | 2,35 ± 0,88        | 5,36 ± 1,17        |
| Moderate OSAS Group | 1,26 ± 0,24       | 127,63 ± 4,78       | 3,8 ± 1,14         | 4,21 ± 1,11        | 8,49 ± 1,7         |
| Severe OSAS Group   | 12,44 ± 1,82      | 246,92 ± 12,39      | 17,28 ± 5,14       | 56 ± 10,59         | 78,76 ± 12,06      |

## Relationship Between OSAS and Irisin

The study revealed significant differences in Non-REM apnea-hypopnea duration between the Mild OSAS Group ( $26.5 \pm 1.54$ ) minutes and the Control Group ( $5.03 \pm 1.34$ ) minutes, ( $P < 0.05$ ). Post-hoc Tukey analysis confirmed that the duration was significantly longer in the Mild OSAS Group. Similarly, a significant difference was observed regarding total sleep apnea-hypopnea duration when comparing the Mild OSAS Group ( $32.09 \pm 1.34$ ) minutes to the Control Group ( $8.13 \pm 2.11$ ) minutes, with the Mild OSAS Group exhibiting longer durations ( $P < 0.05$ ). Furthermore, a comparison between the Severe OSAS Group ( $117.86 \pm 6.6$ ) minutes and the Mild OSAS Group ( $26.5 \pm 1.54$ ) minutes showed a significant difference in Non-REM apnea-hypopnea times, where post-hoc analysis indicated substantially longer durations in the Severe OSAS Group ( $P < 0.05$ ). Finally, the total sleep apnea-hypopnea duration in the Severe OSAS Group ( $128.06 \pm 5.60$ ) minutes was significantly higher than that of the Mild OSAS Group ( $32.90 \pm 1.34$ ) minutes, ( $P < 0.05$ ). Post-hoc Tukey tests further validated that the durations were significantly more prolonged in the Severe OSAS Group (Fig. 1).

In examining the relationship between OSAS and irisin, the AHI was identified as a critical PSG parameter. When AHI values during REM sleep were compared, the Mild OSAS Group ( $33.42 \pm 2.49$ ) exhibited a significantly higher index than the Control Group ( $7.65 \pm 2.74$ );  $P < 0.05$ , a finding further supported by post-hoc Tukey analysis. Similarly, the Severe OSAS Group ( $39.06 \pm 3.45$ ) showed a significantly higher AHI during REM sleep compared to the Mild OSAS Group ( $P < 0.05$ ), with post-hoc testing confirming the higher index value in the severe cases. Regarding the HI, the Mild OSAS Group ( $70.23 \pm 3.16$ ) demonstrated a significant increase over the Control Group  $16.37(4.28)$ ;  $P < 0.05$ , with post-hoc results validating this elevation. Furthermore, the HI difference between the Severe OSAS Group ( $246.92 \pm 12.49$ ) and the Mild OSAS Group ( $70.23 \pm 3.16$ ) was statistically significant ( $P < 0.05$ ), and post-hoc Tukey analysis indicated that the Severe OSAS Group maintained the highest index values (Fig. 2).

## ELISA Analysis

OSAS groups were compared according to serum irisin concentrations as well as PSG parameters. When the serum irisin concentration in the mild OSAS group ( $0.26 \pm 0.07$  ng/mL) was compared with that of the control group ( $0.01 \pm 0.00$  ng/mL), the concentration in the mild OSAS group was significantly higher ( $P < 0.05$ ). Furthermore, when the severe OSAS group ( $0.09 \pm 0.06$  ng/mL) was compared with the mild OSAS group ( $0.26 \pm 0.07$  ng/mL), the serum irisin concentration in the severe OSAS group was significantly lower ( $P < 0.05$ ) (Fig. 3).

## Correlation Analysis

Spearman rank correlation analysis revealed key monotonic relationships between serum irisin levels and polysomnographic parameters across the study population. Serum irisin levels demonstrated positive correlations with the Sleep-Wake Period ( $r = 0.4$ ) and REM-AHI ( $r = 0.4$ ). Additionally, irisin showed weak positive correlations ( $r = 0.2$ ) with Non-REM Apnea-Hypopnea duration, total Sleep Apnea-Hypopnea duration, Supin-AHI, Hypopnea index, and NON-REM AHI. Conversely, the Sleep-Wake Period showed strong negative correlations ( $r = -0.8$ ) with respiratory disturbance metrics, including Non-REM Apnea-Hypopnea, total Sleep Apnea-Hypopnea, Supin-AHI, Hypopnea, and NON-REM AHI. Strong positive inter-correlations ( $r = 0.8$  to  $1.0$ ) were observed among the primary respiratory disturbance and severity indices (Fig. 4).

## Discussion

The most significant finding of the study is the correlation between irisin levels and the severity of Obstructive Sleep Apnea Syndrome (OSAS). Although correlation analysis and specific subgroup trends reveal a biphasic dynamic—wherein irisin levels initially rise as a compensatory metabolic or muscular response in mild OSAS cases but subsequently fall during severe stages due to chronic hypoxia and tissue depletion—this non-linear pattern accounts for the statistical variance. While the overall linear inverse correlation reflects the general decline observed in advanced-stage disease, localized positive correlations correspond to the early compensatory phase. Analyses of PSG and ELISA data indicate that irisin levels decrease as OSAS severity increases; this phenomenon may explain the quantity of irisin released in conjunction with heightened muscle activity, particularly in mild to moderate OSAS cases. Conversely, it can be posited that irisin levels drop in patients with severe OSAS due to chronic and recurrent apneas associated with reduced muscle activity [13], [14]. Furthermore, it was determined that in mild OSAS cases, irisin levels initially rise compared to the control group before gradually declining. This trend is attributed to increased oxidative stress resulting from hypoxia associated with the clinical progression of OSAS. Another contributing factor is the decline in irisin levels linked to reduced muscle activity as the body attempts to overcome the hypoxia inherent in the chronic course of severe OSAS.

In their study investigating the use of serum irisin levels as a diagnostic marker for gestational diabetes, Kulhan et al. found that irisin levels were significantly lower compared to the control group [15]. Yano et al. reported that the hormone irisin improves pancreatic beta-cell function by converting white adipose tissue into brown adipose tissue; furthermore, it ameliorates insulin resistance in patients with type 2

diabetes by enhancing hepatic glucose and lipid metabolism, as well as increasing insulin receptor sensitivity in cardiac and skeletal muscles [16]. Due to irisin's ability to convert white adipocytes into brown adipocytes, its impact on obesity has been investigated, and the hormone is recognized as aiding in weight control [17], [18].

Findings from the variance analyses in our study demonstrated an inverse relationship between irisin levels and the severity of OSAS. Therefore, these findings are of great importance for understanding the relationship between irisin and metabolism. A negative correlation exists between irisin levels and OSAS in patients with severe OSAS; irisin levels decrease as the severity of OSAS increases [19]. Additionally, it has been established that obesity has a significant impact on OSAS and that OSAS occurs twice as frequently in obese patients. The findings of our study support this view.

An examination of irisin values reveals a significant decrease in patients with severe OSAS [20]. Our study also demonstrated that irisin levels decrease as the severity of OSAS increases. The findings regarding the relationship between irisin and OSAS can be further reinforced by an additional observation from our study: while irisin levels rise in patients with mild OSAS—driven by respiratory muscle activity—they decrease in cases of severe OSAS due to reduced muscle activity.

Regarding the biphasic effect observed in the correlation analysis, it is important to note that because the Spearman rank correlation coefficient is designed to measure monotonic relationships across the entire dataset, statistical limitations arise when attempting to express the biphasic pattern of serum irisin levels—characterized by a compensatory increase in mild cases and a decline in severe cases—via a single global correlation coefficient; consequently, this complex distribution exhibits non-linear dynamics resulting from the combination of localized positive correlations in the early stages and an overall inverse correlation in the advanced stages.

## Conclusion

In the study, serum irisin levels exhibit a non-linear (biphasic) relationship with OSAS severity, showing an initial compensatory rise in mild stages followed by a significant decrease in severe stages. When the literature was examined, in addition to being consistent with this relationship, a new result was obtained; while irisin levels increase in mild OSAS patients, this level decrease in moderate and severe OSAS patients. What actually occurs here is the chronic hypoxic situation that develops as OSAS progresses and the change in irisin levels that occurs in relation to this.

As a result, in the diagnosis and treatment of OSAS cases at the non-clinical stage, irisin hormone analysis should be performed and evaluated together with the patients' anamnesis. In this way, irisin level can be recognized as a hormone at the center of metabolism in clinical routine. It is possible to explain many physiopathologies based on the relationship between OSAS, which is a metabolic disease and the decreasing irisin level. In addition, developing alternative methods in the treatment of OSAS and different respiratory diseases can be achieved by monitoring irisin levels. Further studies are needed to develop and traditionalize diagnosis and treatment protocols regarding the OSAS-irisin relationship.

# Study Limitations

Although this study provides significant insights into the relationship between irisin levels and OSAS severity, several limitations must be acknowledged. First, the sample size of the control group ( $n = 8$ ) is notably smaller than the OSAS patient groups, which may limit the statistical power of some comparisons. Second, as a single-center study conducted at Isparta City Hospital, the results may not be fully generalizable to broader or more diverse populations. Third, while irisin is known to be influenced by acute physical activity and metabolic factors, the participants' daily exercise routines and dietary habits were not strictly controlled or recorded prior to blood sampling. Additionally, the cross-sectional design of the research allows for the identification of correlations but prevents the establishment of a definitive causal link between irisin fluctuations and the progression of OSAS. Finally, since the specific receptor for irisin remains unidentified, the precise molecular pathways through which OSAS-induced hypoxia influences irisin secretion require further investigation.

Furthermore, several limitations regarding metabolic and anthropometric variables should be noted. Although body mass index and general demographic data were recorded, detailed body composition parameters (such as visceral fat mass and precise fat-to-muscle ratios) and comprehensive metabolic profiles (including insulin resistance markers and lipid panels) were not systematically adjusted for as covariates in our primary group models. Consequently, the potential confounding role of obesity and metabolic derangements on irisin variance cannot be entirely ruled out, and longitudinal studies incorporating rigorous body composition analyses are warranted to validate these mechanisms.

Furthermore, certain methodological limitations regarding the correlation analyses must be addressed. Spearman's rank correlation coefficient is inherently designed to measure monotonic relationships across an entire dataset. Because serum irisin levels exhibit a non-linear, biphasic pattern across OSAS severity groups—showing an initial compensatory elevation in mild cases followed by a progressive decline in moderate to severe stages—fitting this complex distribution into a single global monotonic correlation coefficient presents inherent statistical constraints. While localized subgroup trends and clinical physiological markers reflect distinct phases of the disease response, future investigations should employ segmented regression models or polynomial curve-fitting techniques to capture these non-linear trajectories more precisely.

## Declarations

This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethics committee approval was granted by the Clinical Research Ethics Committee of Süleyman Demirel University Faculty of Medicine (Date: 20/07/2016, Decision No: 106). Written informed consent was obtained from all individual participants included in the study.

## Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Ethics Approval

Approval was obtained from the Clinical Research Ethics Committee of Süleyman Demirel University Faculty of Medicine (Date: July 20, 2016; Decision No: 106).

## The data availability statement

The data sets used and/or analysed during the current study available from the corresponding author on reasonable request.

All data supporting the findings of this study are included within the article.

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## Author Contribution

F.O. and M.S. conceived and designed the study. F.O., O.O., T.G., and A.Y. participated in data collection and patient evaluation. F.O. and M.S. performed the laboratory experiments and statistical analyses. F.O., M.S., and O.O. drafted the initial manuscript. M.S., O.O., T.G., and A.Y. critically revised and edited the manuscript for intellectual content. M.S. supervised the project. All authors read and approved the final manuscript.

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## Figures

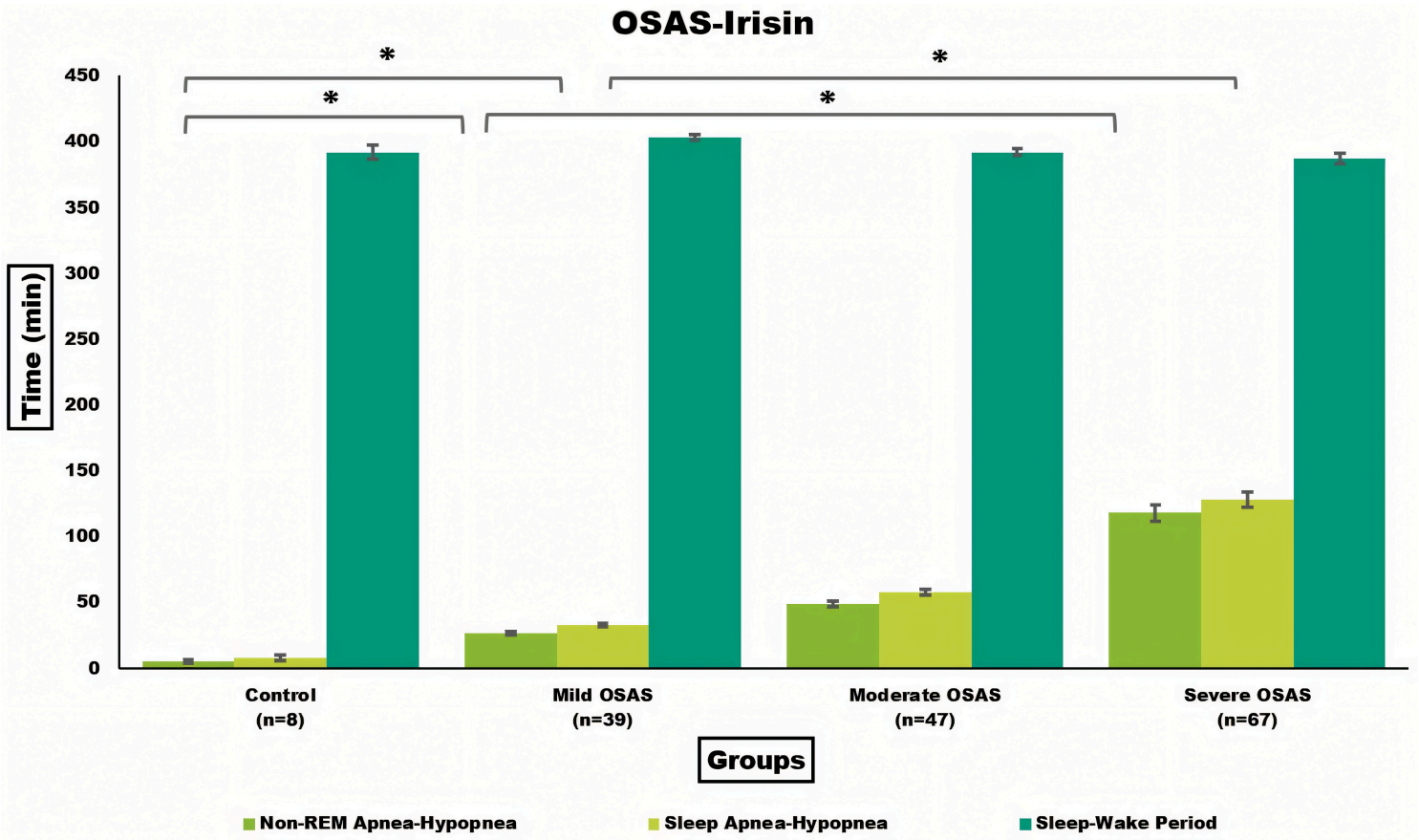


Figure 1

OSAS Times. Each point is expressed as Time (minutes) and SD. \* $P\leq 0,05$ ; compared according to control groups and mild OSAS groups.

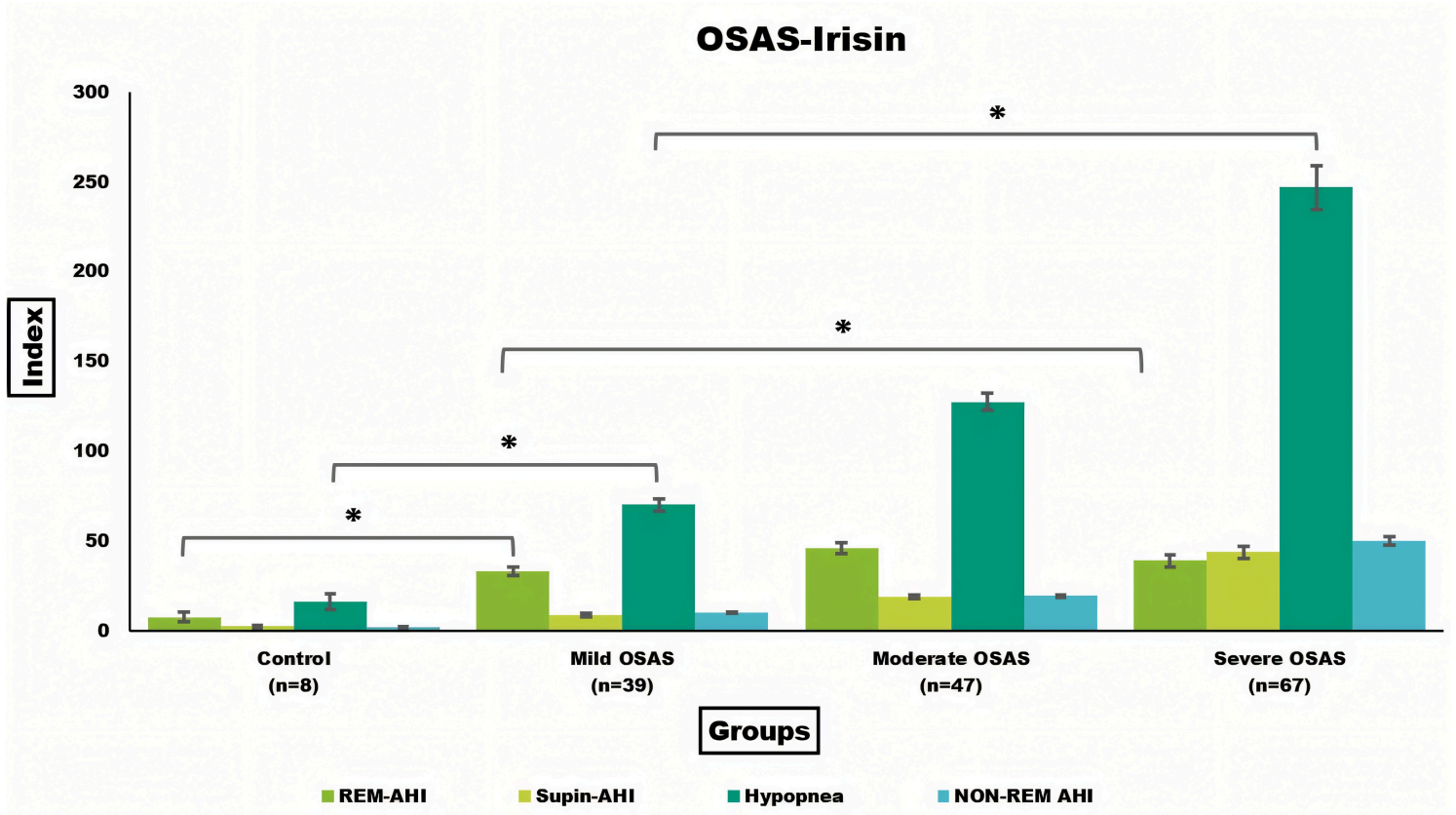


**Figure 2**

Apnea Hypopnea Indexes. Each point is expressed as Index and SD. \*  $P < 0.05$ ; compared according to control groups and mild OSAS groups.

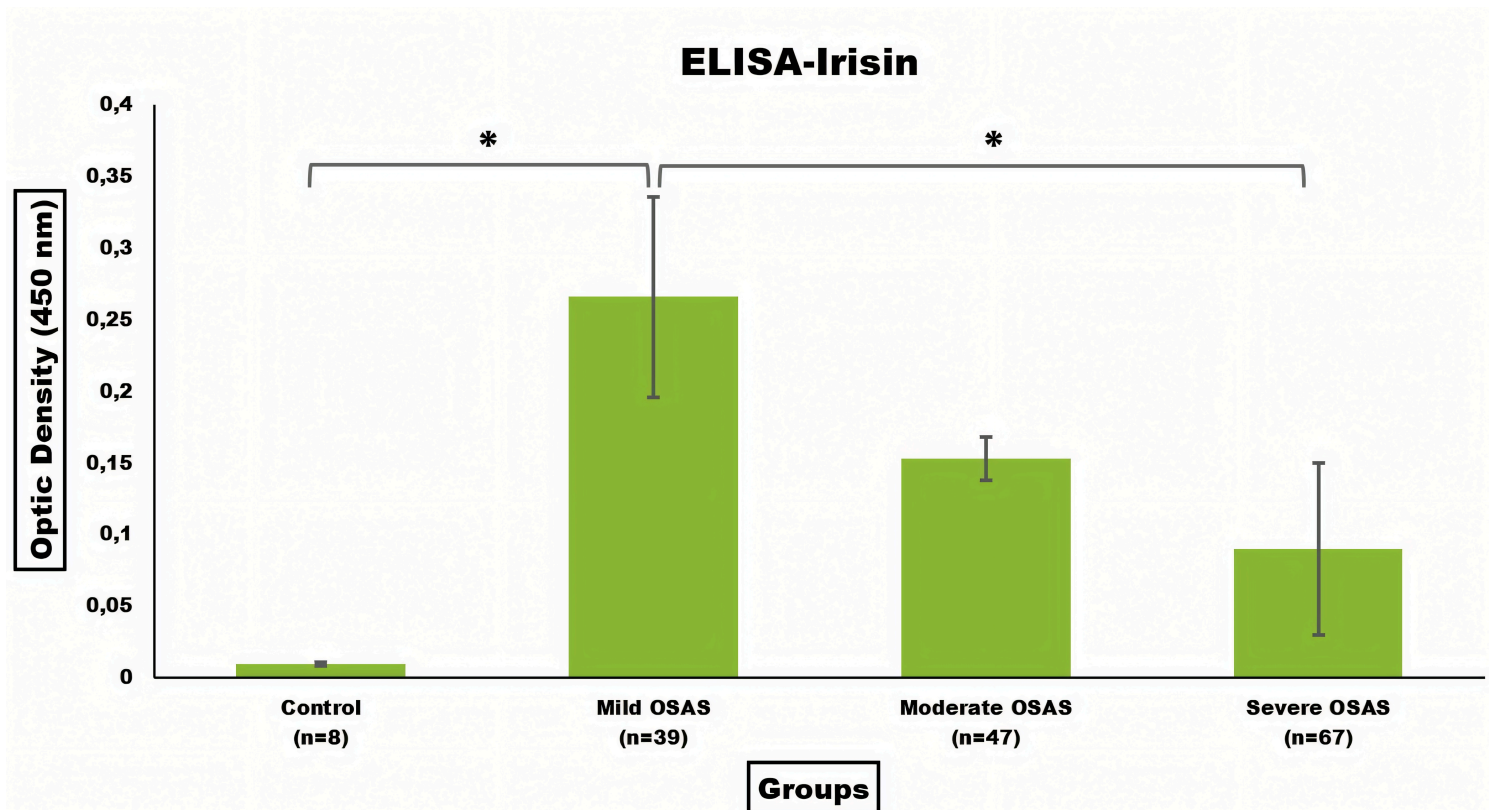


Figure 3

Irisin ELISA test. Each point is expressed as concentration values and SD. \* $P\leq 0.05$ ; compared according to control groups and mild OSAS groups.

Spearman Correlation Matrix of OSAS Parameters and Irisin

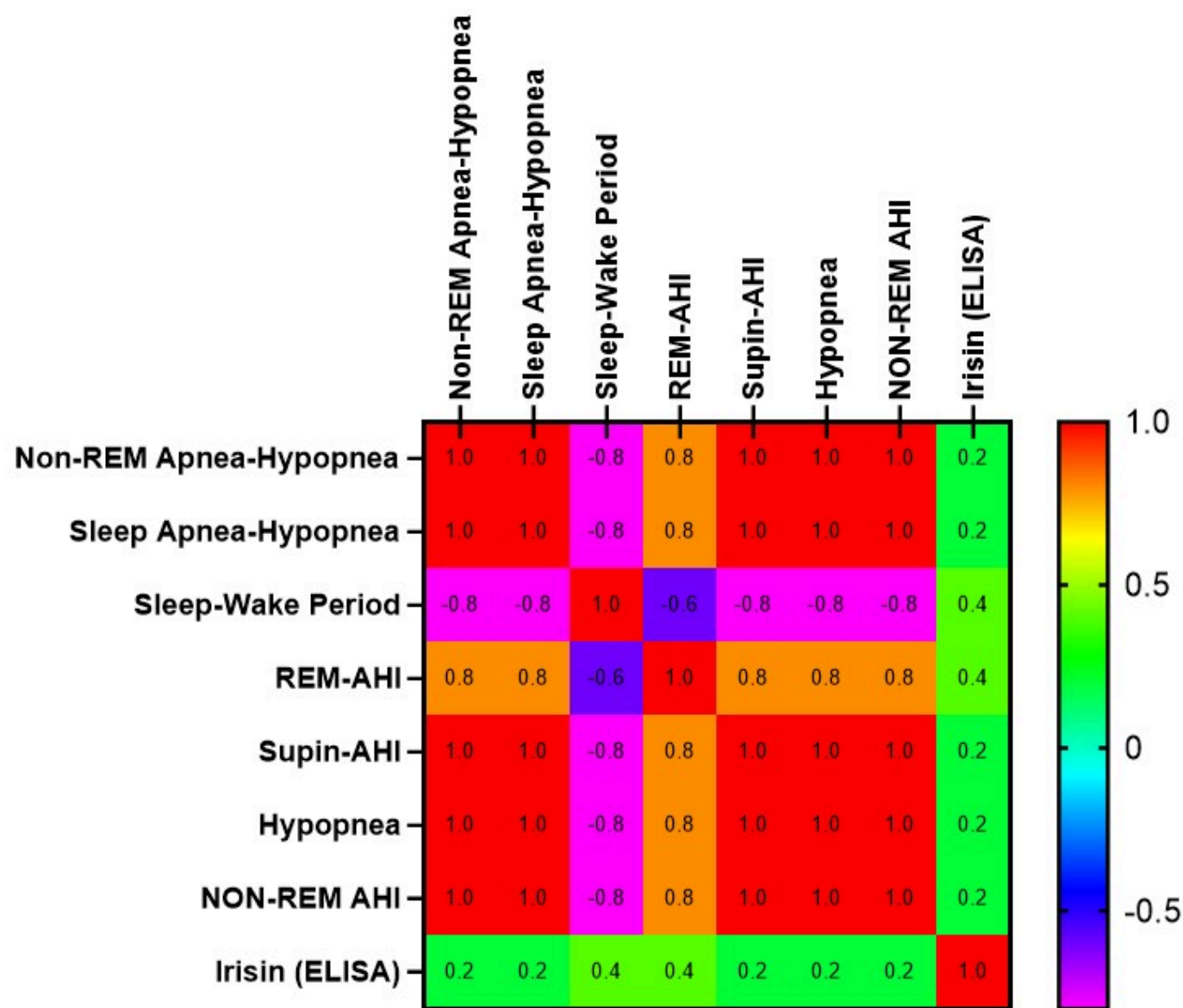


Figure 4

Heatmap of Spearman rank correlation analysis showing the relationships between serum irisin levels and polysomnographic parameters in OSAS patients.