Assessment of sleep and obesity in adults and children

Observational study

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Abstract
The sleep allows many psychological processes, such as immune system activity, body metabolism and hormonal balance, emotional and mental health, learning, mnemonic processes. The lack of sleep could undermine mental and physical purposes, causing an alteration in cognitive functions or metabolic disorders. In our study, we have examined the irregular sleep effects with the overweight and obesity risk in children and adults.

The sample was composed of 199 subjects, of which 71 adults, (29 males and 42 females), and 128 children (73 males and 55 females). We have measured the weight and height with standard techniques; we also have measured the body mass index dividing the weight in kg with the height square expressed in meters (kg/m²). Subjects were divided into underweight, normal weight, overweight, and obese. Were administered some questionnaires to measure the quantity and quality of sleep, and eating habits and individual consumption of food.

Analysis of demographic variables not showed significant differences between male and female groups but highlighted a significant trend differences in normal-weight score. The clinical condition has a substantial impact on body mass index score and sleep hours were significant predictor on this.

Quantity and quality sleep can also represent a risk factor of overweight and obesity, so sufficient sleep is a factor that influence a normal weight. Adults and children that sleep less, have an increase in obesity and overweight risk with dysfunctional eating behaviors, decreased physical activity, and metabolic changes.

Abbreviation: BMI = body mass index.

Keywords: adult, children, obesity, sleep

1. Introduction
Sleep is essential to support the functions and health of the entire body. Indeed, it guarantees the optimal functioning of a multiple physiological processes, such as immune system activity, body metabolism and hormonal balance, emotional and mental health, various forms of learning, the memorization processes. Numerous studies have highlighted negative effects of lack of sleep on mental and physical balance, indeed they could change cognitive functions, such as memory and emotions control, and hunger regulation. It has been shown that upset or insufficient sleep weakens immune system and favors metabolic disorders. Several studies have underlined that decreased duration and/or quality of sleep can affect glucose metabolism and interfere with the secretion of anabolic hormones like growth hormone (GH), prolactin, and testosterone; also it can alter amount and timing of catabolic hormones like glucocorticoids; and catabolic hormones,

Many epidemiological studies have shown decreased sleep duration and quality (6–7 hours per night) is associated with increased obesity incidence, diabetes, cardiovascular diseases, and mortality. In our study, we investigated the association between sleep duration and quality, and overweight risk and obesity in children and adults. The examined subjects diet and eating habits were also evaluated.

2. Method
The study was conducted on secondary school children, and on practice dental clinic patients. Were involved 199 subjects, of which 71 adults (29 males and 42 females) with age between 29 and 65 years, and 128 children (73 males and 55 females) with age between 10 and 13 years. Every subject was instructed about the goals and the procedures and signed an informed consent form. The study was approved by the local ethics committee. The data were picked in a period between October 2016 and November 2017. Were evaluated weight, height, and body mass
index (BMI) data. The weight and height were measured with standard techniques, the BMI was assessed dividing the weight in kg with the height square expressed in meters (kg/m²). The hormones involved in the hunger and satiety mechanisms can be measured during sleep, thereby, we not analyzed. Subjects were divided into underweight, normal weight, overweight, and obese. Were administrated several questionnaires to evaluate the quantity and quality of sleep, with which were highlighted hours of sleep overall, the mode of falling asleep, and the quality of sleep. The survey of eating habits and individual consumption of food was carried out through the administration of questionnaires and the use of a reasoned atlas of nutrition. [17]

2.1. Statistical analysis

We considered 2 groups: adults and children. The Shapiro normality test was carried out to analyze the distribution of the variables. Continuous variables were expressed as mean ± standard deviation, whereas as median, and first-third quartile in no normal distribution. The Student unpaired t test and the Mann–Whitney U test were used to compare 2 group (males and females groups), when appropriate. For intragroup analysis, correlations between variables were computed by Pearson correlation or Spearman coefficient. We performed a multiple regression analysis on the BMI score (dependent variables). At first, we focused on the influence of demographic and clinical variables, by using patient’s age and sleep hours scores as predictors. We applied a backward elimination stepwise procedure for the choice of the best predictive variables according to the Akaike information criterion. Analyses were performed using an open-source R3.0 software package. A 95% of confidence level was set with a 5% alpha error. Statistical significance was set at P < .05.

3. Results

3.1. Adult group

Analysis of demographic variables not showed significant differences between male and female groups but highlighted a significant trend differences in normal-weight score (P = .07) (Table 1). In male group found a significative positive correlation between age and BMI score (r = 0.48; P = .009) and a significative negative correlation between sleep hours and BMI score (r = −0.64; P < .001) (Fig. 1A and B). In female group, we found a significative negative correlation between sleep hours and BMI score (r = −0.78; P < .001) (Fig. 1C). Results in Table 2 showed that the clinical condition of the patients has a significant impact on BMI score. Sleep hours was significant predictor on BMI score for male and female group.

| Table 1 | Socio-demographic and clinical characteristics of adults group. |
|---------|---------------------------------------------------------------|
| Sample  | Male | Female | P-value |
| N. Subjects | 71 | 29 | 42 |
| Age (mean ± SD) | 43.65 ± 11.39 | 42.21 ± 11.55 | 44.64 ± 11.31 | 0.38 |
| BMI (mean ± SD) | 25.25 ± 3.87 | 25.97 ± 3.41 | 24.76 ± 4.12 | 2.2 |
| Sleep hours (mean ± SD) | 7.5 ± 0.91 | 7.42 ± 0.88 | 7.56 ± 0.93 | 0.52 |
| BMI | Underweight (median [I–III]) | 17 (17.0–17.0) | – | 17 (17.0–17.0) | – |
| Normal-weight (median [I–III]) | 23 (21.0–24.0) | 23 (22.0–24.0) | 22 (20.0–23.0) | 0.07 |
| Overweight (median [I–III]) | 27 (26.0–28.0) | 27 (27.0–28.0) | 27 (26.0–27.5) | 0.2 |
| Obese (median [I–III]) | 32 (31.0–33.0) | 32 (31.0–34.0) | 32 (31.25–32.75) | 0.9 |
| Sleep hours | Underweight (median [I–III]) | 8 (8.0–8.0) | – | 8 (8.0–8.0) |
| Normal-weight (median [I–III]) | 8.3 (7.8–8.5) | 8 (7.5–8.5) | 8.3 (8.0–8.5) | 0.29 |
| Overweight (median [I–III]) | 7 (6.5–7.3) | 7 (6.5–7.3) | 7.05 (6.02–7.3) | 0.98 |
| Obese (median [I–III]) | 6.5 (6.0–7.0) | 6.5 (6.25–6.75) | 6.4 (5.7–6.87) | 0.7 |

$I$ = first quartile, $II$ = third quartile, BMI = body mass index, SD = standard deviation.

Figure 1. Correlation between BMI score and demographic and clinical variables. (A) Scatter plot of BMI score and age in male adult group. (B) Scatter plot of BMI score and sleep hours in male adult group. (C) Scatter plot of BMI score and sleep hours in female adult group. BMI = body mass index.
3.2. Children group

Analysis of demographic variables not showed significant differences between male and female groups but highlighted a significant differences in hours of sleep in underweight group ($P = .03$) (Table 3). A significative negative correlation between sleep hours and BMI score ($r = -0.6; P < .001$) and ($r = -0.56; P < .001$) in male and female group respectively were found (Fig. 2A and B). Results in Table 4 showed that the clinical condition of the patients has a significant impact on BMI score. Sleep hours were significant predictor of BMI score for male and female group.

| Table 2 | Backward linear regression: predictors on BMI score in adult group. |
|---------|---------------------------------------------------------------|
| Dependent variables | Predictors | $\beta$ | Std $\beta$ | P-value | Adjusted $R^2$ |
|---------------------|-------------|--------|-------------|---------|----------------|
| BMI male group      | Age         | 0.08   | 0.04        | .09     | 0.43           |
|                     | Sleep hours | -2.07  | 0.6         | .002    |                |
| BMI female group    | Age         | 0.04   | 0.04        | .28     | 0.59           |
|                     | Sleep hours | -3.49  | 0.44        | <.001   |                |

$\beta$ = regression coefficient, BMI = body mass index, Std $\beta$ = standardized regression coefficient.

| Table 3 | Socio-demographic and clinical characteristics of children group. |
|---------|---------------------------------------------------------------|
| Sample  | Male | Female | P-value |
| N. Subjects | 128   | 73    | 55   |
| Age (mean±SD) | 11.32±1.14 | 11.33±1.16 | 11.31±1.14 | .92 |
| BMI (mean±SD) | 19.49±3.12 | 19.63±3.28 | 19.30±2.92 | .56 |
| Sleep hours (mean±SD) | 9.03±0.91 | 9.07±0.92 | 8.98±0.89 | .55 |
| BMI Underweight (median (I–III)) | 16.2 (16.0–16.6) | 16.3 (16.0–16.6) | 16.1 (16.0–16.6) | .68 |
| Normal-weight (median (I–III)) | 18 (17.7–18.6) | 28.15 (17.8–18.7) | 18 (17.7–18.4) | .45 |
| Overweight (median (I–III)) | 21.4 (20.6–22.0) | 21.2 (20.2–21.8) | 21.5 (21.0–22.0) | .47 |
| Obese (median (I–III)) | 26.5 (25.2–27.0) | 26.5 (25.6–27.0) | 26.5 (25.2–27.0) | .91 |
| Sleep hours Underweight (median (I–III)) | 9.5 (9.0–10.1) | 10 (9.4–10.5) | 9.3 (9.0–9.6) | .03 |
| Normal-weight (median (I–III)) | 9.3 (9.0–10.0) | 9.3 (9.0–10.0) | 9.5 (9.3–10.0) | .15 |
| Overweight (median (I–III)) | 8.1 (8.0–8.3) | 8 (8.0–8.3) | 8.2 (8.0–8.3) | .92 |
| Obese (median (I–III)) | 8.3 (8.0–8.6) | 8.3 (8.0–8.7) | 8.3 (8.0–8.5) | .62 |

$I$ = first quartile, $II$ = third quartile, BMI = body mass index, SD = standard deviation.

*B* $< .05$. 

Figure 2. Correlation between BMI score and demographic and clinical variables. (A) Scatter plot of BMI score and sleep hours in male children group. (B) Scatter plot of BMI score and sleep hours in female children group. BMI = body mass index.
of foods containing carbohydrates with high caloric density over controls.\textsuperscript{[6]}

In addition, numerous epidemiological studies have shown that short chronic duration of sleep, even in school children, was associated with low levels of leptin.\textsuperscript{[33]}

Observational studies by Peter Jones and his team, at the University of Manitoba, also have shown that during the morning breakfast, subjects undergoing 4 hours of sleep compared to a control group with a rest of 9 hours, took an average of 300 Kcalories and about 30\% more saturated fat.

Moreover to eating habits, physical activity also affects body weight. Sleep deprivation contributes to weight gain by reducing energy expenditure and physical activity.\textsuperscript{[30,34]} Indeed, people who sleep less are less active.

The lateral hypothalamus plays an important role in many motivated behaviors, in sleep/wake cycle, in food introduction, in energy balance. It is also home-based to a heterogeneous population of neurons that expresses and co-expresses many neuropeptides, including hypocretin.\textsuperscript{[7]}

Hypocretin 1 (Orexin A) is a key signal in the regulation of the wake/sleep cycle, regulating the introduction of food by stimulating appetite and energy metabolism.\textsuperscript{[37,38]} Neurons that produce hypocretin are sensitive to glucose and leptin concentrations; indeed, when glucose concentration decreases, hypocretin neurons are activated, which induce an increase in food intake. On the contrary, when leptin levels increase, hypocretin neurons are inhibited and the amount of food ingested decreases.

In case of sleep deprivation, decrease of leptin may contribute to hypocretin increase which prolongs wakefulness, stimulates appetite and allows the search for food.

Hypocretin-producing neurons project into the ventrolateral preoptic area and induce the transition from sleep to wakefulness, significantly increase awakening, physical activity and energy expenditure, and reduce sleep. In addition, partial sleep deprivation reduces the effectiveness of hypocretin in stimulating physical activity and energy expenditure.\textsuperscript{[139]}

Finally, observational and experimental studies have shown an association between decreased duration and quality of sleep with reduced insulin sensitivity, insulin resistance, and decreased glucose tolerance,\textsuperscript{[40,41]} with an increased risk of type II diabetes. The risk of falling ill with type II diabetes increases by 50\% in subjects with less than 5 hours of sleep, compared to those who sleep 8 hours. Restricting sleep by 4 hours for 6 nights causes a decrease in glucose tolerance as measured by the glucose tolerance test (19). A long period of sleep restriction (<6.5 hours per night) can lead to a 40\% decrease in glucose tolerance. Studies conducted by Bergman and Yaggi have shown a significant reduction in insulin sensitivity during night-time sleep\textsuperscript{[42,43]} and the insulin sensitivity index in subjects with short sleep duration is lower than in subjects with normal sleep duration.\textsuperscript{[142]}

### 4. Discussion

We have highlighted, in previous surveys on a Sicilian student population with overweight/obese subjects compared to normal-weight subjects, a lower adherence to the Mediterranean diet. Observational studies and clinical experiences have shown that adherence to the Mediterranean diet is inversely associated with obesity. Furthermore, we have found a lower consumption of breakfast and a higher consumption of snacks and out-course meals.\textsuperscript{[17,18]}

Our study has highlighted that the duration and quality of sleep can also represent a risk factor of overweight and obesity in examined subjects. Therefore, sufficient sleep is required to maintain a normal weight.

One of the first indications of the link between reduced sleep duration and increased body weight came from the observation that the increase in the number of overweight or obese subjects, in industrial societies, was accompanied by an equal decrease in the average sleep duration of about 1 or 2 hours. Several studies\textsuperscript{[4,19–25]} have shown that children aged 5 to 9 years who sleep less than 10 hours per night run a 1.5 to 2-fold risk of becoming obese compared to those who sleep properly; while adults that sleep less than 8 hours per night run a 1.5 to 2-fold risk of becoming obese.

Increased incidences of obesity and type II diabetes has been highlighted by epidemiological and experimental studies.\textsuperscript{[4,19–25]} In addition, people who sleep less than 8 hours have an increase in BMI proportionate to the decrease in sleep.\textsuperscript{[26]} The association between sleep duration and weight gain in women is supported by backward linear regression: predictors on BMI score in children group. Therefore, sufficient sleep is required to maintain a normal weight.

### Table 4

| Dependent variables | Predictors   | β    | Std β | P-value | Adjusted R² |
|---------------------|--------------|------|-------|---------|-------------|
| BMI male group      | Age          | −0.2 | 0.27  | .45     | 0.35        |
|                     | Sleep hours  | −2.12| −0.34 | <.001   |             |
| BMI female group    | Age          | −0.17| 0.3   | .57     | 0.29        |
|                     | Sleep hours  | −1.81| 0.38  | <.001   |             |

β = regression coefficient, BMI = body mass index, Std β = standardized regression coefficient.
Sleep deprivation causes an increase in cortisol (4) and GH (1), hormones that contribute to determining resistance to the action of insulin. High cortisol secretion and a shift in the circadian rhythm of cortisol; therefore, contribute to significant alterations in glucose tolerance, insulin-sensitivity, and insulin secretion. Adipocytes, through the production of hormones called adipokines or adipocytokines, can also directly modulate insulin sensitivity. Circulating adipocytokine patterns are profoundly altered in obese patients. An increase in fat mass is associated with a general increase in adipocytokines that can reduce insulin sensitivity and have a direct effect on the genesis of insulin resistance (eg, tumor necrosis factor-alpha, interleukin 6, resistin). On the other hand, adiponectin levels are inversely related to obesity and insulin resistance.

Indeed, adiponectin increases tissue insulin sensitivity and circulating levels of adiponectin are commonly decreased in obese subjects and are associated with insulin resistance, inadequate insulin response, and type 2 diabetes. Levels of Visfatin, an Adipoqunein with insulin-like action, are also considered a link between obesity and insulin resistance. In fact, sleep deficiency is associated with high BMI, high Visfatin levels and high insulin resistance.

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