Sleep and obesity

A preliminary study conducted at the sleep laboratory at Montfort Hospital

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Summary

Over several years, an increasing number of studies have shown a link between weight gain and sleep disorders. Furthermore, we have seen an increase in body weight accompanied by a parallel decrease in the duration of sleep in the population. In our study, we wished to demonstrate this relationship between obesity and sleep in the patient population at the sleep laboratory at Montfort Hospital. Our hypothesis was that the Body Mass Index (BMI) of the patients followed in the laboratory would show the following correlations:

– BMI is proportional to lack of sleep.
– BMI is directly related to the number of apnoeic episodes during sleep.
– BMI is directly related to the number of limb movements during sleep.
– BMI increases as sleep efficiency (SE) decreases.

This study was conducted at the Sleep Laboratory at Montfort Hospital. All data used in this study were drawn from patients who underwent sleep studies from 2008 to 2010. Our sample was randomly selected from the Sleep Laboratory's database. After the exclusion of patients not meeting participation criteria, our sample size was reduced from 350 to 314 patients.

We made the following observations: BMI was proportional to a decrease in SE (–0.13, $p = 0.018$), an increase in N1 (0.18, $p = 0.001$), and an increase in R1 (0.24, $p < 0.0001$). In addition, BMI was directly proportional to the number of apnoeic/hypopnoeic episodes (0.25, $p < 0.0001$) and partial airway obstructions (0.31, $p < 0.0001$). These results verified our research hypothesis by confirming the correlations between BMI and several polysomnographic parameters. Despite its small sample size, our study was able to confirm the results of other similar previous studies on this subject by Powered by Editorial Manager® and Preprint Manager® from Aries Systems Corporation demonstrating similar patterns.

We recommend that subsequent studies of this subject at Montfort Hospital be conducted with larger sample sizes, taking into account other sociodemographic, professional and ethnocultural factors.

Key words: sleep disorder; sleep apnoea; Body Mass Index; fat; screening; obesity

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Introduction

Obesity is one of the largest epidemics of our century, the causes and explanations of which have been more or less explored. The most widely discussed contributing factor is the changes found in modern diet and lifestyle. Lately, studies have demonstrated other factors which may explain the rise in obesity [1, 2]. Among them, one recognised factor is the lack of sleep related to the lifestyle of city-dwellers [3]. The impact of poor sleep on weight gain and obesity has long been suspected among researchers, although it has received little emphasis in the past. Generally, the usual recommendations for avoiding weight gain have been a healthy diet and physical exercise. Despite these efforts, however, we continue to witness a rise in body weight among all age groups in the general population, with serious consequences on public health and quality of life.

The problem of obesity is multifactorial. The challenge for researchers is in identifying the principal contributing factors despite the interactions that occur between them. In recent years, we have seen an increasing number of studies shedding light on the link between weight gains and sleep disorders. In 1960, a study led by the American Cancer Society found that the population had an average of 8.5 hours of sleep, between 8.0 and 8.9 hours [4]. The same study led 35 years later by The National Sleep Foundation (USA) found an average duration of 7 hours [5]. In 2008, over 30% of adults between the ages of 30 and 64 admitted to sleeping less than 6 hours per night [6]. This phenomenon is affecting younger and younger populations.

In fact, the risks of being overweight or obese increase when people fail to develop good lifestyle habits at a young age. Studies have demonstrated that newborns that sleep 1 hour less than normal (that is, 10 hours) have an elevated risk of becoming overweight in adolescence and of developing diabetes [7]. But what has changed? Why are we sleeping less? Studies show that we go to bed later now than we did in the past [8, 9]. At the same time, we are waking up earlier and earlier. This phenomenon has been attributed to a change in the pace of our lives in recent years. The advancement of media and communication, a change in our dietary habits, and a changing pattern of leisure and work-related activities are all factors, among many others, that influence our sleep and general quality of life [7].

There are a variety of problems linked to lack of sleep, including metabolic and endocrine changes that lead to decreased glucose tolerance and an increase in appetite. All of these factors influence weight gain.

Figure 1 Population distribution according to gender and group ages. Men are represented in black and women in white and the values in the histogram represent the number of persons from each sex. In the group age 75–90, the gender distribution is two women and one man.

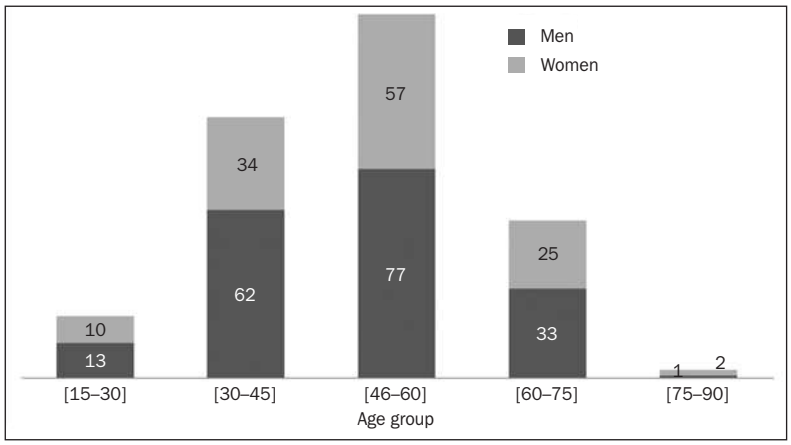


Table 1 Sample characteristic*.

Characteristic	Study sample (n = 314)
Age	48.2 ± 12.6 (17–79)
% women	41.1
BMI	31.9 ± 7.2 (17.2–61.4)
SP	138.9 ± 21.3
DP	85.9 ± 14.1 (11–192)
ESS	8.1 ± 4.9 (0–24)
Sleep Architecture	
TDL (min)	431.9 ± 66.6 (107.5–535.9)
TST (min)	336.4 ± 83.3 (18.5–480)
Stage N1 (min)	39.2 ± 22 (2–133.5)
Stage N2 (min)	241.8 ± 67.8 (0–369)
Stage N3 (min)	8.5 ± 18.9 (0–207)
Stage R (min)	46.9 ± 28.9 (0–137.5)
SE %	77.5 ± 16.1 (13.9–98.3)
Summary of position during sleep	
Left (min)	110.8 ± 85.4 (0–374.9)
Right (min)	114.1 ± 94.5 (0–433.5)
Spine (min)	95.9 ± 94.1 (0–458.5)
Supine (min)	14.4 ± 45.6 (0–366.9)
Summary of the respiratory event	
Apnoeas + hypopnoeas	34.8 ± 63.6 (0–446)
Partial obstruction	37.1 ± 43.3 (0–297)
Summary of the saturation of O_2	
Saturation min. O_2%	81.8 ± 8.6 (50–95)
Saturation max. O_2%	98.6 ± 1.1 (93–100)
Summary index of leg movements	
PLMs	10.8 ± 19.8 (0–157.4)
RLS	0.5 ± 1.8 (0–14)
Summary of heart rate	
Mean during night	65.8 ± 10.5 (0–100)

BMI = Body Mass Index (calculated with the weight in kilograms divided by height in meters squared); SP = systolic pressure; DP = diastolic pressure; ESS = Epworth Sleepiness Scale; TDL = time in bed; TST = total sleep time; SE = sleep efficiency (calculated with TST / TDL × 100); PLMs = periodic leg movements during sleep; RLS = the restless legs syndrome.

* The given values are represented as mean ± standard deviation (range) unless otherwise indicated.

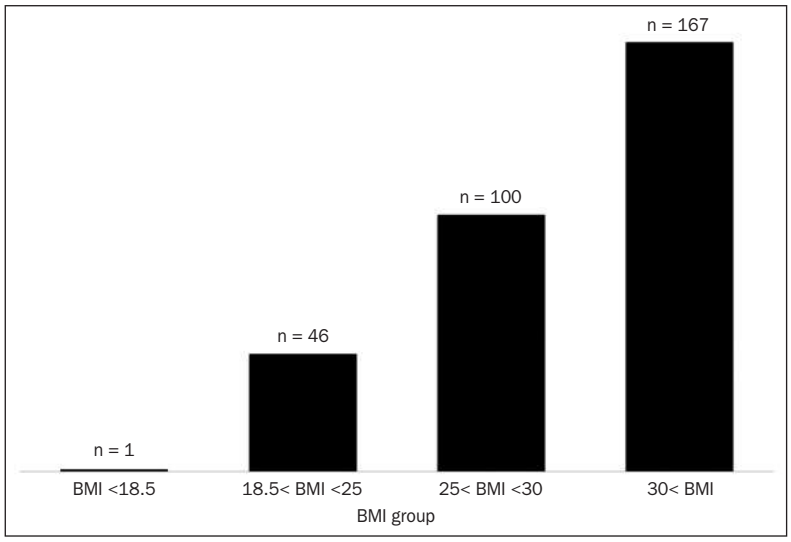
We know that two distinct processes regulate sleep: a homeostatic process that depends on the quantity of sleep and wakefulness, and a circadian process that is controlled by an endogenous rhythm. Among the different stages of sleep, slow-wave sleep (SWS) is the deepest and is comprised of the highest rates of arousals [10]. Among patients who previously suffered from loss of sleep, the recovery of SWS seems to be favoured over rapid eye movement (REM) sleep [11]. Studies have shown that during a night of recovering after a period of sleep deprivation, a rebound is first seen in SWS, and then in other stages of sleep [12–15].

Sleep disorders also encompass problems such as breathing disorders during sleep, restless legs syndrome (RLS) and periodic limb movements (PLM). Studies show that breathing disorders are directly related to obesity [16], and that they affect the structure of sleep, reducing both the duration and quality of sleep [17]. Breathing disorders generally comprise of apnoeic and hypopnoeic episodes, as well as partial airway obstructions during sleep. They result in momentary arrests of respiration during sleep that cause the individual to awaken for a brief moment (a microarousal) in order to compensate for the resulting oxygen deficit. Generally, patients are not conscious of this pathology as it happens, but they experience consequences such as fatigue, diurnal somnolence, memory and concentration problems, irritability, anxiety, mood disorders and sexual dysfunction (low libido and erectile dysfunction). Restless legs syndrome (RLS) is defined by the almost irresistible urge to move the legs. Periodic limb movement disorder (PLMD) is defined by repeated flexions of the hips, knees and ankles [18]. This pathology is present in 90% of patients with RLS [19–21], but can also occur in patients without RLS. We observed that in 25% of patients undergoing a polysomnographic study [22], and it is conventionally interpreted as an accidental result associated with breathing disorders of sleep [23], narcolepsy or disorders related to paradoxical sleep [24]. Although RLS and PLMD can occur independently of each other, their frequent association suggests a common aetiology. This was confirmed recently by the discovery of a common genetic variation (BTBD9) that confers a susceptibility to both RLS and PLMD [25, 26]. Recently, Xiang Gao et al. demonstrated through their research that both abdominal and overall adiposity are associated with a higher risk of RLS. A similar association was found between obesity in young adults (18–21 years) and the prevalence of RLS later in life (40 years and older), which suggests that obesity is a risk factor for the development of RLS.

In the setting of the research conducted at the Montfort Hospital's Sleep Laboratory, we wished to explore the relationship between sleep and obesity by comparing biometric parameters with the polysomnographic data collected in the laboratory. These parameters were blood pressure, weight, Body Mass Index, sex and age. This preliminary research allows us to paint a general picture of the patients seen at the sleep laboratory. Of course, individuals admitted for a study at the laboratory generally suffer from disorders that disturb their sleep, such as breathing disorders of sleep (BDS), RLS, primary or secondary insomnias, for example.

The current study focused on shedding light on the correlations between Body Mass Index (BMI) and poly-

Figure 2 Distribution of the Body Mass Index (BMI, kg/m²) according to the classification scheme of Health Canada: underweight with BMI <18.5, normal weight with BMI between 18.5 and 25, overweight with BMI between 25 and 30, and obese with BMI greater than 30. The values above the histogram represent the size of each group.



somnographic data. The main relationships that we wanted to access through this study were whether BMI is directly proportional to lack of sleep, directly related to number of apnoeic episodes during sleep, directly related to limb movements during sleep, and if it increases as sleep efficiency decreases.

Methods

Overview

This research was conducted in the sleep clinic of Montfort Hospital. All the data were taken from patients seen in the clinic from 2008 and 2010. We randomly selected a sample of 350 patients from the database of Montfort Hospital.

Sample characteristic

Patients missing certain criteria were excluded from the study, and the size of our population was subsequently reduced from 350 to 314 patients. In our sample population, we found that during their sleep 231 patients had apnoeic and/or hypopnoeic episodes, 286 patients had partial obstructions, 181 patients had periodic limb movements (PLMs), and 50 patients had restless legs syndrome (RLS). Figure 1 shows the distribution of the population by age and sex. Slightly less than half of our population were between 45 and 60 years of age. We can say that the curve follows a normal distribution, despite the fact that the sizes of the groups at the extremes of age (15–30 and 75–90) are small. Table 1 shows the characteristics of the study population. The sample was composed of 59% men and 41% women. The mean age of the sample was 48.2 years and the mean BMI was 31.9 kg/m².

Figure 2 shows the distribution of the population according to BMI groups. More than half of the population had a BMI above 25 kg/m². The graph does not follow a normal distribution. Furthermore, we had to remove the first group (BMI <18.5) because it was too small for further analysis.

Data collection

The data collected in the sleep clinic are based on sleep studies done during the night in the rooms of the sleep laboratory. The moment of onset of sleep and wakening depends on each patient. After consent, the patients complete a questionnaire about the quality of their sleep (Epworth) and their medications. Their height, weight and blood pressure are measured. For this preliminary study, we did not take into account medications and diagnosis from medical and psychiatric conditions because of the small population sample, and because we were only interested to investigate the correlation between sleep and BMI.

Subjective measures of sleep

We used the Epworth Sleepiness Scale to measure quality of sleep and to determine degree of somnolence. A score of higher than 10 indicated an abnormally high level of sleepiness, pointing to a lack of sleep secondary to a sleep disorder that would be investigated by polysomnography at the sleep laboratory [42].

Polysomnography

A polysomnographic system was used to assess the stages of sleep, breathing disorders and cardiac variables (Grass Instruments, Quincy, Massachusetts, United States). Sleep was studied with the use of electroencephalography (EEG), electro-oculography (EOG) and electromyography (EMG). Continuous monitoring of arterial oxyhaemoglobin saturation was achieved by pulse oximetry. Oral and nasal air flow, nasal air pressure and thoracic respiratory movements were used to assess for breathing disorders. A period of 30 seconds of the recording time was used to define the stages of sleep and breathing disorders using standard criteria (Grass Instruments, Quincy, Massachusetts, United States).

Polysomnographic measurements of deep sleep

The polysomnographic measurements of sleep duration were used to evaluate the degree of restriction of deep sleep. Total sleep time (TST) defined by polysomnography is the total number of hours of sleep. Sleep efficiency (SE) is the total sleep time divided by the time spent in bed.

Statistical analysis

All of our analyses were cross-sectional studies obtained with the use of XLSTAT software. We used ANCOVA, Pearson tests and chi-square tests for categorical data to evaluate the relationships between BMI as an independent variable and the parameters collected in the sleep laboratory. All of our tests were adjusted with respect to age and sex. We grouped the patients into five age cohorts: (15–30), (30–45), (45–60), (60–75) (75–90). There were no individuals

Table 2 Univariate association between BMI and confounders.

	Mean ± SD	*p*-value
SP	138.943 ± 21.285	<0.0001
DP	85.869 ± 14.124	0.896
ESS	8.051 ± 4.929	0.636
TIB	431.912 ± 66.601	0.959
TST	336.397 ± 83.286	0.042
N1	39.184 ± 22.009	<0.0001
N2	241.750 ± 67.780	0.001
N3	8.511 ± 18.939	0.160
R	46.929 ± 28.940	0.396
Left	110.765 ± 85.412	0.033
Right	114.086 ± 94.542	0.016
Supine	95.918 ± 94.122	0.919
Prone	14.436 ± 45.648	0.401
Apnoeas + hypopnoeas	34.764 ± 63.643	<0.0001
Partial Obstructions	37.134 ± 43.275	<0.0001
Min. O_2	0.818 ± 0.086	<0.0001
Max. O_2	0.986 ± 0.011	<0.0001
PLMs	10.807 ± 19.811	0.635
RLS	0.551 ± 1.814	0.718
Age group	48.2 ± 12.6	0.280
SE	77.5 ± 16.1	0.345

SP = systolic pressure; DP = diastolic pressure; ESS = Epworth Sleepiness Scale; TIB = time in bed; TST = total sleep time; SE = sleep efficiency (calculated with TST / TDL × 100); PLMs = periodic leg movements during sleep; RLS = the restless legs syndrome.

The *p*-value is from comparison BMI and descriptive variables ANCOVA (continuous variables with normal distribution) or chi-square test (categorical variables).

younger than 15 years of age. We grouped BMI into five groups according to the classification scheme of Health Canada: underweight with BMI <18.5, normal weight with BMI between 18.5 and 25, overweight with BMI between 25 and 30, and obese with BMI greater than 30. The patients were also categorised into two groups based on sleep efficiency: patients with sleep efficiency less 80% and patients with sleep efficiency greater than or equal to 80% [27]. Patients missing certain criteria were excluded from the study, and the size of our population was subsequently reduced from 350 to 314 patients.

Stepwise regression was used to assess the relationship of BMI with the parameters collected in the sleep laboratory. All models were first adjusted for max. O_2, and then further adjusted for multiple confounding variables. Potential confounding variables were identified, and their association with the predictor was assessed. The confounding variables examined included systolic pressure (SP), supine position, partial obstructions, min. O_2, max. O_2, age group and interactions between diastolic pressure (DP) and age group, right and sex, left and sex, prone position and age group, apnoeas/hypopnoeas and SE, and RLS and age group.

In the first model, the covariates were SP, supine position, partial obstructions, O_2 min., O_2 max., age group, and interactions between DP and age group, right and sex, left and sex, prone position and age group, apnoeas/hypopnoeas and SE, and RLS and age group.

In the second model, interactions between prone and age group, apnoeas/hypopnoeas and SE, RLS and age group were removed.

Table 3 Pearson correlations* between the distribution of BMI and sleep efficiency, sleep architecture**.

		Sleep architecture			
	SE	**Stage N1**	**Stage N2**	**Stage N3**	**Stage R**
	r(p-value)	*r(p-value)*	*r(p-value)*	*r(p-value)*	*r(p-value)*
BMI *(n = 314)*	–0.13 (0.018)	0.18 (0.001)	–0.06 (0.279)	–0.10 (0.080)	–0.24 (<0.0001)
≤18.5 *(n = 1)*	–	–	–	–	–
>18.5 to ≤25 *(n = 46)*	–0.21 (0.155)	0.24 (0.103)	–0.20 (0.187)	–0.44 (0.003)	0.09 (0.551)
>25 to ≤30 *(n = 100)*	0.33 (0.001)	0.11 (0.281)	0.24 (0.018)	–0.08 (0.418)	0.25 (0.014)
>30 *(n = 167)*	–0.21 (0.007)	0.10 (0.196)	–0.16 (0.043)	–0.04 (0.617)	–0.29 (0.000)

BMI = Body Mass Index (calculated with the weight in kilograms divided by height in meters squared); SE = sleep efficiency (calculated with TST / TDL × 100).

* Data are represented in the given correlation coefficient (*p*-value).

** BMI was compared with the duration of each of the stages of sleep.

Table 4 Pearson correlations* between the distribution of BMI apnoea and hypoapnoea and limb movement.

	Respiratory disorders		Limb movement	
	Apnoea + hypopnoea	**Partial obstruction**	**PLM**	**RLS**
	r(p-value)	*r(p-value)*	*r(p-value)*	*r(p-value)*
BMI *(n = 314)*	0.25 (<0.0001)	0.31 (<0.0001)	0.05 (0.431)	0.05 (0.365)
≤18.5 *(n = 1)*	–	–	–	–
>18.5 to ≤25 *(n = 46)*	0.13 (0.407)	0.10 (0.502)	0.17 (0.269)	0.15 (0.326)
>25 to ≤30 *(n = 100)*	0.35 (0.000)	0.30 (0.003)	0.04 (0.665)	–0.14 (0.178)
>30 *(n = 167)*	0.04 (0.641)	0.09 (0.641)	–0.06 (0.464)	–0.07 (0.400)

PLMs = periodic leg movements during sleep; RLS = restless legs syndrome.

* Data are represented in the given correlation coefficient (*p*-value).

Results

Table 2 shows the univariate association between BMI and confounders. Higher BMI was associated with higher systolic pressure, lower TST, lower time in the stage N2, more time in the stage N2, longer time in the left and right position, more apnoeas/hypopnoeas and partial obstruction episodes (table 2).

Tables 3 and 4 show the correlations between the distribution of BMI and sleep efficiency, duration of sleep, duration of stages of sleep, apnoeic/hypopnoeic episodes, and limb movements during sleep. In our sample, 0.3% had a BMI <18.5, 15% had a BMI between 18.5 and 25, 31% had a BMI between 25 and 30, and 53% had a BMI >30. In the table, we did not include patients with BMI <18.5 as we only had one such patient.

Figure 3 Population distribution according to sleep efficiency (SE) and BMI group. SE <80% are represented in grey and SE >80% in white, and the values in the histogram represent the number of people from each category.

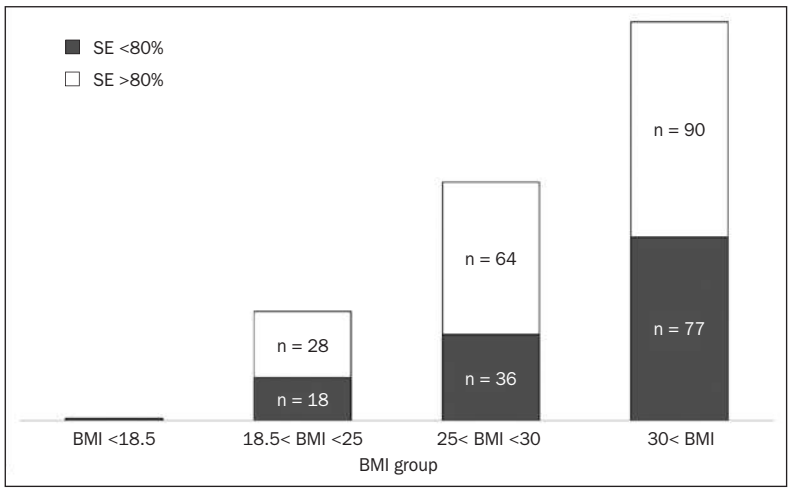
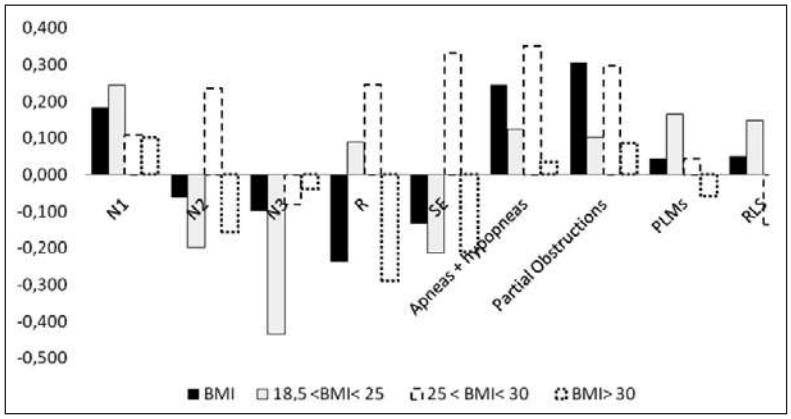


Figure 4 Pearson correlations between the distribution of Body Mass Index (BMI) and sleep efficiency, duration of sleep stages, apnoea and hypoapnoea and limb movement. It is observed in the figure that the parameters which are positively correlated are represented by histogram bars above 0. The parameters that are negatively correlated are represented by histogram bars below 0. Individuals whose BMI was below 18.5 were not shown because this class contained only one individual.



Sleep efficiency (SE) (fig. 3)

We performed a chi-square test to assess the independence of sleep efficiency and BMI (table 2). We determined that SE and BMI were dependent ($p = 0.345$). From the Pearson correlation, we observed that BMI was inversely proportional with sleep efficiency (–0.13, $p = 0.018$). We observed that not all correlations were significant or consistent across groups. In the group with BMI between 15 and 30, BMI was proportional with an increase in sleep efficiency (0.33, $p = 0.001$). On the contrary, in the group with BMI >30, BMI was proportional with a decrease in SE (–0.21, $p = 0.007$).

Duration of stages of sleep

In the sample, we observed that the durations of the N1 and REM stages of sleep had a significant correlation ($p < 0.05$) with BMI in our population. BMI was directly proportional with an increase in N1 (0.18, $p = 0.001$) and R (0.24, $p < 0.0001$). Unfortunately, not all of the correlations were significant and consistent across the groups. In the case of N1, the correlation was not significant in the groups. In the case of R among patients with BMI between 25 and 30, BMI was proportional to an increase in R (0.25, $p = 0.014$). By contrast, in the group with BMI >30, BMI was proportional to a decrease in R (–0.29, $p = 0.000$). In the case of N2, we did not observe a significant correlation with BMI, while in the group with BMI between 25 and 30, BMI was proportional to an increase in N2 (0.24, $p = 0.018$), and in the group with BMI >30, BMI was proportional to a decrease in N2 (–0.16, $p = 0.043$). With regards to N3, we did not observe a significant correlation with BMI in our sample while for the group with BMI between 18.5 and 25, BMI was proportional to a decrease in N3 (–0.44, $p = 0.003$).

Breathing disorders during sleep

In our sample, BMI was directly proportional to the number of apnoeic/hypopnoeic episodes (0.25, $p < 0.0001$) and partial airway obstructions (0.31, $p < 0.0001$). Unfortunately the correlations were not all significant. In the group with BMI between 25 and 30, the correlations were consistent, and BMI was proportional to an increase in the number of apnoeic/hypopnoeic episodes (0.35, $p = 0.000$) and partial airway obstructions (0.30, $p = 0.000$).

Limb movements

With regards to restless legs syndrome (RLS) and periodic limb movements (PLMs), we did not obtain significant correlations with BMI in the population or with specific BMI groups in our sample.

Summary of results (fig. 4)

Looking at the results of the study, we note that N1 stage, N3 stage, the number of apnoeic and hypopnoeic episodes, the number of partial airway obstructions, and limb movements all progressed in the same direction but with different ampli-

tudes. For the N1 stage, the correlation with BMI is stronger for the group from 18.5 to 25 than for the sample as a whole. For apnoeic/hypopnoeic episodes, the group from 25 to 30 was the predominate BMI of the sample. The number of partial airway obstructions was the only variable with which BMI was much more correlated with regards to the groups. Overall, we observed that BMI had a positive correlation with the N1 stage, apnoeic/hypopnoeic episodes and number of partial airways obstructions, and a negative correlation with the N3 stage. The other parameters studied did not show a consistent correlation.

Discussion

The overall results of our study investigating the correlation between poor quality of sleep and increased BMI are consistent with the results of previous studies on the subject [28–30]. Our results contribute to a body of research that has established a link between sleep disorders and BMI [31], whether these sleep disorders be secondary to breathing disorders [32], restless leg syndrome (RLS), or periodic limb movements (PLMs) [16].

BMI and lack of sleep

It was observed that lower sleep efficiency in the sample was associated with patients who had a BMI over 30 (fig. 4), but care needs to be taken with these observations because there were discrepancies within the BMI group. One possible explanation is that individuals with a poor quality of sleep spend more time in bed and thereby have lower overall energy expenditures [33].

In fact, a lack of sleep favours a positive energy balance, which may contribute to weight gain and obesity. Fewer hours spent asleep results in more hours awake which can allow increased food intake. Another effect created by a lack of sleep is a reduction in physical activity in the daytime due to increased daytime somnolence [33]. The behavioural aspects of snacking may be explained by a physiological level by recent studies on hormonal changes, notably involving ghrelin, leptin and orexin [34–36].

These studies show that dysregulation in the secretion and plasma levels of these hormones may have an effect on appetite, raising the risk of excessive food consumption and weight gain.

BMI and breathing disorders

We found a significant relationship between respiratory disorders during sleep and BMI. Our sample shows a strong correlation between BMI, apnoea and hypopnoea and partial airway obstructions (fig. 4). This tendency was more marked in the group with BMI from 25 to 30. These data are consistent with observations that have been made in the literature [37]. Breathing disorders of sleep are generally associated with increased BMI. In one study, it was estimated that 58% of patients suffering from obstructive sleep apnoea were obese [37]. Another recent study found an increase in body weight in patients newly diagnosed with obstructive sleep apnoea [38]. These studies show a bi-directional interaction between breathing disorders and obesity. Most studies view respiratory disorders such as obstructive sleep apnoea as an effect of weight gain rather than a factor that contributes to weight gain. The incidence of sleep apnoea in obese individuals is linked to the deposition of fat around the oropharynx, which obstructs the airway during sleep. Generally, obese individuals have shorter necks with increased fatty tissue, which raises their risk of obstructive sleep apnoea.

BMI and limb movements

Our results unfortunately do not show a significant relationship between increased BMI and RLS or PLMs in our sample (table 3), perhaps explained by the small size of our sample. In a more general sense, our results confirm the observations of other studies [39]. In one horizontal study of 1803 men and women over 18 years of age in the United States, each 5 kg/m^2 increase in BMI was associated with a 31% increase in the probability of having RLS [40].

BMI and sleep architecture

We observed that sleep architecture had an effect on BMI. Among patients who were obese (BMI >30) and overweight (BMI between 25 and 30), this relationship was found to be significant, compared to those with a normal BMI (between 18.5 and 25) (table 3). In our sample, individuals with a BMI above 30 spent less time in deep sleep (N3), and more time in the N1 stage. The mechanisms underlying the relationship between sleep architecture and obesity are still poorly understood [41]. Researchers have yet to define the exact role of each stage of sleep in general [41], and in particular with regards to increased BMI.

Conclusion

This preliminary study confirmed our research hypothesis by demonstrating a correlation between the BMI of overweight and obese individuals and several polysomnographic parameters. Despite its weaknesses related to sample size, our study was essentially able to confirm the findings of previous reference studies of this type conducted on an international level by yielding similar patterns as these studies.

The limitation of this preliminary study was the small sample size compared to reference studies. A further limitation is the absence of detailed information regarding socio-demographic and professional backgrounds of our study population, (employment, social class, lifestyle, etc.), which may have a significant influence on sleep. The absence of consideration of medical and psychiatric co-morbidities that may lead to or complicate sleep disorders is also a limitation, as is the absence of a control group for comparison, due to the small sample size as well as the fact that patients at the sleep laboratory usually have problems arising from sleep disorders. We encountered a lack of financial and human resources to collect the necessary data and complete the study in the given time of three months.

In order to address the weaknesses noted in our study on sleep and obesity, we suggest the following recommendations for future research in this domain:

A prospective study should be conducted on sleep and obesity in the Franco-Ontarian setting, with a larger sample size similar to those found in the reference studies.

Detailed information should be taken into account regarding sociodemographic and professional backgrounds of the study population, such as employment, social class, lifestyle, ethnocultural group and race. These factors may have a significant influence on sleep in a given population. The prospective study should take into account medical and psychiatric conditions which may result in or complicate sleep disorders. A prospective study should compare patient groups to control groups with larger sample sizes in order to draw conclusions characterising the relationship between sleep and obesity in the general Franco-Ontarian population.

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