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# Obesity as an independent predictor of subjective excessive daytime sleepiness☆

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**Summary**
*Introduction:* Sleepiness is one of the cardinal complaints of patients in tertiary referral centres for sleep disorders. It is commonly associated with sleep disorders, but the additional influence of obesity remains unclear. We hypothesised that obesity may be an independent predictor of sleepiness in this population.
*Patients and methods:* We reviewed 335 patients undergoing overnight polysomnography over a 3 month period (May–July 2011), recording selected sleep parameters in addition to subjective sleepiness as measured by the Epworth Sleepiness Scale (ESS, 0–24 points).
*Results:* 173 patients (120 male, age 52 (13) years, BMI 33.9 (7.4) kg/m$^2$) had obstructive sleep apnoea and 59 of those were established on continuous positive airway pressure (CPAP). 55 patients (38 male, age 56 (14) years, BMI 31.7 (6.7) kg/m$^2$) had PLM disorder, with 3 on medical treatment. 155 patients were obese (93 male, age 50 (14) years, BMI 37.3 (6.1) kg/m$^2$) and this group was significantly sleepier than the 180 non-obese subjects (107 male, age 42 (15) years, BMI 25.3 (3.0) kg/m$^2$). The mean ESS of the obese group was 12.9 (5.5) points, compared to 10.4 (5.7) points for the non-obese patients ($p < 0.001$). Those with a diagnosis of OSA had an ESS of 12.3 (5.8) versus 10.7 (5.7) points for those without ($p = 0.007$). Females had an ESS of 12.8 (5.9) compared to 10.7 (5.5) points for male patients ($p < 0.001$). Regression analysis revealed that obesity ($p = 0.007$), PLM disorder ($p = 0.010$) and hypertension ($p = 0.032$) were independently associated with subjective sleepiness (adjusted $R^2 = 0.384$, $p = 0.02$), obesity accounting for 15.7% of the variability in sleepiness.
*Conclusion:* Independent of underlying sleep disorders, obesity contributes significantly to daytime sleepiness. Measures to promote weight loss and close links to a dietician assisted service will be helpful in the specialist setting of a sleep disorders service to improve long-term health outcomes.


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## Background

Excessive daytime sleepiness (EDS) is a common complaint, with as many as 40% of the general population estimated to suffer from some form of abnormal sleepiness at some stage in their lives.[1] It is the most common presenting complaint in the Sleep Disorders Centre, with obstructive sleep apnoea (OSA) and periodic limb movement disorder (PLMD) frequently cited as the underlying diagnoses. However, for many patients seen in tertiary referral centres, treatment of the underlying condition may not entirely treat their symptoms,[2] suggesting that there are other important factors contributing to hypersomnolence.

Additionally, it remains uncertain what factors contribute to the severity of sleepiness symptoms in patients with OSA. The apnoea—hypopnoea index (AHI) used to describe sleep-disordered breathing and measure the severity of OSA does not correlate well with the presence or degree of hypersomnolence. However, the relative success of continuous positive airway pressure (CPAP) treatment in relieving EDS suggests that the correction of intermittent nocturnal hypoxaemia can improve symptoms. Despite this, studies examining the significance of intermittent nocturnal hypoxia have produced conflicting results.[2–4]

Obesity has been linked to EDS in the general population, even in the absence of sleep-disordered breathing.[5–7] Similarly, diet and food intake may contribute.[8] It has been suggested that EDS in obesity is a result of systemic metabolic changes, which disrupt normal sleep regulation.[9]

However, data on the significance of obesity in the setting of the sleep disorder clinic with multiple different diagnoses is currently lacking. We hypothesised that obesity is a significant contributing factor to excess daytime sleepiness in this patient population, independent of underlying co-existing sleep disorder.

## Methods and patients

This study reviewed patients undergoing overnight polysomnography during a three month period (May—July 2011) in the Sleep Disorders Centre at Guy's & St Thomas' NHS Foundation Trust, London, UK. It was approved by the local service board (project number 2011/2402). Age, gender and body mass index (BMI) of each patient were recorded, along with significant co-morbidities and medications. Daytime sleepiness, as measured by the Epworth Sleepiness Scale (ESS), was assessed. In the sleep study, we recorded sleep latency (from a single night polysomnography), apnoea—hypopnoea index (AHI) and periodic limb movement (PLM) index. Technicians in our sleep laboratory follow AASM guidelines to ensure standardisation of sleep latency measurement during polysomnography (lights out to first epoch of any sleep in minutes).[10] In patients who had undergone the multiple sleep latency test we included a formal mean sleep latency (MSL) time. Data on co-morbidities was obtained from the patients' notes (ischaemic heart disease, diabetes mellitus, heart failure and depression). Patients with a diagnosis of narcolepsy were excluded, as this contributes to daytime sleepiness independently of BMI.

## Epworth sleepiness score

The ESS is used as a subjective measure of daytime sleepiness. Patients answer a self-administered questionnaire, in which they rate how likely they are to fall asleep in eight everyday situations. Each item is scored from 0 (not at all) to 3 (very likely), allowing for a range of 0—24 points for a total score (increasing scores indicate more severe sleepiness). Total scores of more than 10 points are considered excessively sleepy and commonly used as a marker of pathological hypersomnolence,[11] with a high accuracy of identifying pathological sleepiness.[12] The test has a good internal and re-test reliability over 5 months.[13]

## Polysomnography

Records from overnight polysomnography studies at St Thomas' Hospital were used to identify the sleep latency, AHI and PLM index for each subject, as well as the MSL where available. In this study, "sleep latency" refers to the sleep latency time available from a single polysomnography study for each patient, and MSL time refers to the result from a formalised mean sleep latency test.[10] The AHI measures the number of apnoeas and hypopnoeas per hour of sleep. Apnoeas greater than 10 s in duration are considered clinically significant and were recorded by measuring nasal and oral airflow and pressure. Obstructive apnoeas are associated with ongoing respiratory muscle effort, whilst central apnoeas are not. Hypopnoeas are caused by respiratory obstructions, where there is a greater than 50% reduction in airflow associated with a drop in blood oxygen saturation.[14] An AHI of 5/hour or more was used as the cut off for classifying patients as having OSA. Where available, the MSL score was recorded. During polysomnography, electrodes on the limbs were used to record electromyographic activity of the muscles and identify periodic limb movements.

## Multiple sleep latency test

The mean sleep latency is derived from the multiple sleep latency test, in which subjects take successive naps at 2 h intervals. Polysomnographical recording is used to assess the mean time to sleep onset for each nap, with an mean sleep latency of less than 10 min considered abnormal, an mean sleep latency of less than 5 min qualifies as severe sleepiness.[14] The mean sleep latency recorded from an MSLT is an objective measure of sleepiness, whereas the ESS provides a subjective assessment of symptoms. In this study, a formal mean sleep latency from an MSLT was available for 68 patients.

## Statistical analysis

Initial analysis revealed that ESS and sleep latency scores were not normally distributed and, thus, characteristics between groups were compared using the Mann—Whitney *U* test. Spearman's rank correlations were used to assess the significance of age, BMI, AHI, oxygen desaturation index (ODI) and PLM index on daytime sleepiness. Finally, the