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Risk for obstructive sleep apnea in obese, non-diabetic adults varies with insulin resistance status

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Abstract

Purpose—Obstructive sleep apnea (OSA) is an increasingly common sleep disorder, especially among obese adults. Early identification of adults at risk for OSA would be of substantial benefit; however the magnitude of the obesity epidemic requires that screening be performed judiciously. The study's aim was to utilize questionnaires that assess OSA risk and symptoms to test the hypothesis that the most insulin-resistant subset of obese individuals is at highest risk for OSA.

Methods—Non-diabetic, overweight-to-obese volunteers underwent direct quantification of insulin sensitivity by measuring steady-state plasma glucose concentrations during the insulin suppression test. Insulin-sensitive and insulin-resistant individuals were administered the Berlin and STOP questionnaires to determine OSA risk status, and Epworth Sleepiness Scale (ESS) to evaluate daytime sleepiness. Fasting insulin and lipid/lipoprotein measurements were performed.

Results—Insulin-mediated glucose disposal differed 3-fold ($p<0.001$) between equally obese, insulin-resistant ($n=22$) and insulin-sensitive ($n=14$) individuals, associated with higher fasting insulin and triglyceride and lower high-density lipoprotein cholesterol (HDL-C) concentrations in insulin-resistant individuals. Fourteen (64%) insulin-resistant as compared with 2 (14%) insulin-sensitive individuals were found to be at high risk for OSA by both questionnaires ($p<0.01$). Whereas half of insulin-resistant individuals met ESS criteria for excessive daytime sleepiness, only 1 insulin-sensitive individual did ($p=0.011$).

Conclusions—High risk for OSA and excessive daytime sleepiness is prevalent amongst the insulin-resistant subgroup of obese individuals. Surrogate estimates of insulin resistance based on fasting insulin, triglycerides, and/or HDL-C can be used to help identify those obese adults who would benefit most from OSA screening and referral for polysomnography.

Keywords

obesity; sleep questionnaires; obstructive sleep apnea; daytime sleepiness; insulin-resistant

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Introduction

Approximately half of the United States adult population is reported to be overweight to moderately obese (Body Mass Index, BMI 25 – 35 kg/m²) [1]. As obesity rates continue to rise, obesity-related conditions are increasingly prevalent. Excess adiposity is a well-known risk factor for obstructive sleep apnea (OSA), the most common sleep-related breathing disorder. Among overweight to moderately obese Americans, prevalence estimates of OSA range from 1 in 5 to as high as 1 in 2 men and women [2,3]. Given the increased association of type 2 diabetes (T2DM) and cardiovascular disease (CVD) in patients with OSA [4,5], early identification and treatment of these individuals before onset of these serious conditions would be of substantial benefit. However, the breadth of the obesity epidemic as well as the cost and limited availability of polysomnography, the gold standard for diagnosis of OSA, require that clinicians be judicious in screening and subspecialty referral. In view of this situation, an approach to identifying those obese individuals at increased risk for OSA is warranted.

We have made efforts to respond to this need by focusing on the fact that obesity, *per se*, increases the risk of T2DM and CVD, a relationship that is particularly upheld in the tertile of obese individuals who are most insulin-resistant [6]. Since insulin resistance is also associated with OSA [7], this study was initiated to test the hypothesis that it is the most insulin-resistant subset of obese individuals who is at greatest risk for OSA. To test this formulation, we compared responses to validated screening questionnaires that assess risk [8,9] for and symptoms [10] of OSA in non-diabetic, overweight to obese individuals, divided into weight-matched insulin-resistant and insulin-sensitive subgroups based on a specific measure of insulin-mediated glucose disposal.

Methods

Participants

The study population consisted of adult volunteers from the San Francisco Bay area recruited from 2009 through 2010, in response to study advertisements asking for healthy individuals to volunteer for our research studies on the role of insulin resistance in human diseases. All subjects gave informed consent, and the Stanford Human Subjects Committee approved the study protocols. Participants were in general good health, without anemia, kidney, liver, or cardiovascular disease. Individuals for the present study were selected to be overweight to moderately obese, non-diabetic (as defined by the American Diabetes Association[11]), and without a previous diagnosis of OSA. Values of BMI (kilogram/meter²) and waist circumference (WC, measured midway between iliac crest and rib cage at end-expiration) were obtained.

Insulin suppression test

Insulin-mediated glucose uptake was quantified by a modification [12] of the original insulin suppression test [13], as originally described and validated [13,14]. After an overnight fast, subjects were infused for 180 minutes with octreotide (0.27 µg/m²/min), insulin (32 mU/m²/min), and glucose (267 mg/m²/min). Plasma glucose and insulin were measured every 10 minutes during the 150- to 180- minute period and averaged to determine steady-state plasma glucose (SSPG) and insulin (SSPI) concentrations. Because SSPI values are similar for all individuals, SSPG concentrations provide a direct measure of insulin-mediated glucose uptake; the higher the SSPG, the more insulin-resistant the individual. Insulin-mediated glucose uptake measurements by the insulin suppression test were shown to be highly correlated ($r > 0.9$) with those obtained using the hyperinsulinemic euglycemic clamp [13]. Volunteers were classified as insulin-resistant or insulin-sensitive, based on their

SSPG concentration being in the highest or lowest tertile of an apparently healthy population studied previously [15]. Subsequent prospective studies have demonstrated significant increases in the development of adverse clinical outcomes, including cardiovascular disease, in the most insulin-resistant third of a population without known disease at baseline [16,17]. Individuals classified as insulin-resistant or insulin-sensitive proceeded to complete the sleep questionnaires described below. Those who did not fulfill the definition for either of these 2 groups were excluded from the study. Fasting plasma glucose, insulin, and lipid/lipoprotein measurements were performed as described previously [18,15].

Sleep questionnaires

Fourteen insulin-sensitive (SSPG = 6.66 mmol/L) and 22 insulin-resistant (SSPG = 9.99 mmol/L) individuals completed the Berlin and STOP questionnaires [8,9]. The Berlin questionnaire has been validated against polysomnography in the community setting and has been shown to be a reliable screening tool for OSA, with a positive predictive value of 89% [9]. This questionnaire consists of 10 questions grouped into 3 symptom categories. One point is given per category for a maximum total score of 3, based on a specified number of positive responses within each category. The STOP (snoring, tiredness, observed apneas, high blood pressure) questionnaire was developed and validated for use in pre-surgical patients [8]. One point is given per positive response for a maximum score of 4. Individuals are classified as having 'high risk' for OSA for scores ≥ 2 by either questionnaire. Scores 0 – 1 by either questionnaire classify individuals as 'low risk' for OSA. The Epworth Sleepiness Scale (ESS) was developed to assess degree of daytime sleepiness [10], which is considered a hallmark symptom of OSA [19]. ESS scores ≥ 10 identify individuals with excessive or pathologic daytime sleepiness.

Statistical analysis

Group differences were assessed using Student's unpaired *t* tests for continuous variables, and χ^2 or Fisher's exact tests for categorical variables. Insulin, triglyceride (TG), high- and low-density lipoprotein cholesterol (HDL-C and LDL-C) values were log-transformed prior to analysis to improve normality for statistical analyses. *P* value < 0.05 was taken to indicate statistical significance. All data analyses were performed using SPSS 17.0 (Chicago, IL, USA).

Results

Participant characteristics

Participant characteristics are shown in Table 1. By definition, mean SSPG concentrations were significantly higher in insulin-resistant than insulin-sensitive individuals (13.1 vs 4.82 mmol/L, $p < 0.001$). While the two groups did not differ in BMI (31.6 vs 30.9 kg/m², $p = 0.42$) or WC (106 vs 105 cm, $p = 0.95$), fasting plasma insulin (FPI) levels were two-fold ($p < 0.001$) and TG concentrations 1.6-fold ($p < 0.05$) higher in insulin-resistant individuals. The ratio of TG to HDL-C was also 2-fold higher ($p < 0.01$).

Risk of OSA and daytime sleepiness

Table 2 compares OSA risk and degree of daytime sleepiness in the two groups. High risk for OSA was significantly more prevalent among insulin-resistant individuals by either the STOP or Berlin questionnaire ($p < 0.05$). When the two questionnaires were combined, 14 (64%) insulin-resistant individuals were found to be at high risk for OSA, compared with only 2 (14%) insulin-sensitive individuals ($p < 0.01$). All individuals classified as high risk for OSA by the STOP also met criteria for high risk by the Berlin questionnaire. When the frequency distribution of STOP and Berlin scores were compared side-by-side between

insulin-resistant and insulin-sensitive individuals, no (0%) insulin-sensitive individuals scored > 2 by either STOP or Berlin questionnaires, as compared with 9 (41%) insulin-resistant individuals who did ($p < 0.01$).

Notably, mean ESS scores were almost 2-fold higher in insulin-resistant individuals ($p = 0.013$). Moreover, whereas half of all insulin-resistant individuals met criteria for excessive daytime sleepiness, only 1 insulin-sensitive person did ($p = 0.011$). Of the 11 insulin-resistant subjects with ESS scores ≥ 10 , 8 (73%) met STOP or Berlin criteria for high risk for OSA.

Discussion

Our findings reveal that apparently healthy, obese adults of comparable adiposity, but who represented extremes of insulin resistance and sensitivity, differ in that presence of insulin resistance is associated with significantly increased risk for OSA. These results extend previous findings in OSA and altered glucose metabolism by using rigorous methods to assess insulin action, as well as underscoring the validity of this independent relationship by selecting obese individuals of a specific BMI range to eliminate BMI *per se* as a potential confounder. Prior studies have used surrogate estimates of insulin resistance (e.g. impaired fasting glucose or glucose intolerance in response to oral glucose challenge) to demonstrate an association with OSA after adjustments were made to account for variable adiposity in these population-based studies [20,21]. The present data provide persuasive evidence of an association between insulin resistance and risk for OSA. Traditionally it has been suggested that insulin resistance occurs as a consequence of OSA via obesity as a shared causal link; recently, alternate pathways underlying OSA physiology have been postulated to trigger disturbances in glucose regulation [7]. These mechanisms may include sleep fragmentation and/or intermittent hypoxia in potentiating insulin resistance, possibilities supported by epidemiologic evidence as well as small studies conducted under short-term, experimentally-induced conditions [7]. The contribution to altered glucose metabolism of other processes that may be activated by OSA, e.g. inflammation and/or adipocytokines, are less defined and require further investigation [22]. Given the dearth of experimental evidence, it would also be remiss not to consider the less conventional hypothesis that insulin resistance could increase one's risk for OSA. Indeed, this cross-sectional analysis leaves open for interpretation the possibility that being insulin-sensitive might, in fact, render it less likely for an overweight/ obese individual to have OSA. Although there is less evidence in the literature to support this line of reasoning, it should be noted that a prospective study demonstrated an association between high baseline insulin levels and incident observed apnea [23]. Ultimately, as cross-sectional studies can only provide evidence of association, further prospective or interventional studies are necessary to ascertain direction of causality.

The other main result of the present study is that insulin-resistant individuals reported increased daytime sleepiness and were more likely to exhibit excessive daytime sleepiness as compared to equally obese, insulin-sensitive individuals. These results are especially provocative in light of a recent study demonstrating that an independent relationship between OSA and type 2 diabetes was confined to patients with severe OSA who reported excessive sleepiness [24]; on the contrary, severe OSA in non-sleepy patients was not associated with prevalent diabetes mellitus. These findings are also consistent with previous results demonstrating that excessive daytime sleepiness is associated with the Homeostasis Model Assessment index (a surrogate index of insulin sensitivity) among patients with OSA [25,26]. Taken together, these data suggest that sleepiness may be a necessary component in the pathogenic link between insulin resistance and OSA. Indeed, it has been postulated that sleepiness in patients with OSA may confer a different metabolic risk profile from non-

sleepy patients with OSA [27]. It should be cautioned that while daytime sleepiness is a hallmark feature of OSA, high ESS scores are not necessarily specific for OSA. Nonetheless, that ESS scores were concordant with results of the Berlin and STOP questionnaires in our study affirms that obese, insulin-resistant individuals are at high risk for OSA, characterized by greater degree of daytime sleepiness.

To the best of our knowledge, this is the first study to provide evidence of the efficacy of the Berlin, STOP, and ESS questionnaires in identifying an association between OSA risk and/or daytime sleepiness with insulin resistance. These findings thereby lend support to use of these questionnaires in areas where cost and/or limited resources preclude offering polysomnography to all patients, as an efficacious screening tool to help identify obese, insulin-resistant individuals at highest risk for OSA. While confirmation of these results with polysomnography would have been preferable, it is reasonable to expect that these questionnaires are reliable indicators of OSA risk. The Berlin questionnaire has been widely used in large epidemiologic studies [2,3,9] and validated against polysomnography [9] in primary care populations/ communities similar to ours. While the STOP questionnaire was developed for use in pre-surgical patients, it was incorporated in the study because unlike Berlin, the STOP does not include BMI as one of its criteria. As anticipated in our overweight/ obese study population, the Berlin questionnaire identified a greater number of individuals at high risk for OSA as compared with the STOP. Nevertheless, the two questionnaires were concordant in identifying a preponderance of insulin-resistant individuals at high risk for OSA. Use of ESS in addition to the STOP and Berlin questionnaires provided further evidence in support of our hypothesis. These results also support use of the STOP questionnaire as an alternative to Berlin in a primary care setting, particularly when rapid screening is desirable due to its simplicity and ease of administration. Finally, that these screening tools were able to detect disparate responses between the two groups of equally obese, but metabolically very different individuals suggests that insulin resistance modulates OSA risk, independent of adiposity, in a substantial manner.

These findings raise the question how best to apply this information in a primary care practice. Given the rising prevalence of obesity, including these questionnaires in routine evaluation of all overweight subjects may not be feasible. However, our results suggest that considerable clinical benefit might be gained by administering these questionnaires to overweight/obese individuals who are insulin-resistant, and therefore at highest risk for OSA. What then is the best way to identify those overweight/obese individuals who are insulin-resistant? While there is no simple way to accomplish this task in a clinical setting, there are at least two surrogate estimates of insulin resistance to consider. The data in Table 1 indicate that both FPI and TG/HDL-C ratio were 2-fold higher in insulin-resistant individuals. Indeed, FPI is a significant predictor of insulin resistance, and individuals whose FPI fall in the upper quartile of an apparently healthy population are likely to be insulin-resistant [15]. Another approach is to use the plasma TG/HDL-C concentration ratio. When this method was applied to a group of overweight/obese individuals [18], a ratio 3.0 (in traditional units, 1.3 in SI units) was relatively successful in identifying individuals who were insulin-resistant. There is evidence that this value is a useful indicator for hyperinsulinemia regardless of race/ethnicity [28]. The higher the FPI concentration and/or the TG/HDL-C ratio, the more likely the overweight/obese individual is to be insulin-resistant. Overweight/ obese individuals considered to be insulin-resistant by either or both of these approaches can be further evaluated with the screening questionnaires. Based on their scores, those deemed at highest risk of OSA could be referred for polysomnography. This approach provides clinicians with a reasonably cost-effective way to identify individuals at enhanced risk of OSA, thereby providing a means for early treatment and/or life-style intervention to prevent complications associated with OSA.

This study was limited by its small size. Extremely obese individuals were also excluded from this study. Prevalence estimates for OSA among Americans with BMI > 35 kg/m² are even higher [2,3] than that of the present BMI distribution, and a case could be made to screen most individuals within this category of obesity especially since these individuals as of yet comprise a minority of Americans. We felt it important rather to focus on overweight to moderately obese individuals which make up nearly half of the U.S. population. Regardless, these study findings may not be applicable to adults who are extremely obese. While use of screening questionnaires to assess OSA risk has its advantages as discussed above, it would have been optimal to confirm our study findings with polysomnography. Nonetheless, the robustness of our results lends strength to our conclusions.

In summary, overweight to moderately obese, non-diabetic adults vary remarkably in OSA risk detectable by conventional questionnaires, and is associated with insulin resistance status. High risk for OSA and excessive daytime sleepiness is prevalent among insulin-resistant individuals, features that are relatively spared in their weight-matched insulin-sensitive counterparts. It is suggested that administration of screening questionnaires to apparently healthy, obese individuals, classified as being insulin-resistant, provides a clinically effective way to identify individuals who are likely to have undiagnosed OSA, or who are at risk for developing the syndrome.

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Table 1

Characteristics of study participants, stratified as insulin-sensitive and insulin-resistant by the insulin suppression test

Variable	Insulin-sensitive individuals ^a (n=14)	Insulin-resistant individuals (n=22)	p value
SSPG (mmol/L)	4.82 ± 0.80	13.1 ± 1.66	<0.001
Number of Men/Women	5/ 9	10/ 12	0.73
Race (Caucasian/Asian/Hispanic/Black)	13/ 1/ 0	16/ 4/ 1/ 1	0.48
Age (years)	58 ± 8	55 ± 7	0.32
BMI (kg/m ²)	30.9 ± 2.6	31.6 ± 2.5	0.42
Waist circumference (cm)	105 ± 10	106 ± 11	0.95
Systolic blood pressure (mm Hg)	121 ± 12	130 ± 16	0.10
Diastolic blood pressure (mm Hg)	70 ± 8	79 ± 9	0.006
Fasting glucose (mmol/L)	5.50 ± 0.59	5.81 ± 0.40	0.11
Fasting plasma insulin (pmol/L)	102 ± 32	207 ± 67	<0.001
Total cholesterol (mmol/L)	4.80 ± 0.84	4.66 ± 0.84	0.64
LDL-cholesterol (mmol/L)	2.90 ± 0.58	2.75 ± 0.74	0.54
HDL-cholesterol (mmol/L)	1.45 ± 0.26	1.13 ± 0.25	0.001
Triglycerides (mmol/L)	1.08 ± 0.52	1.72 ± 0.85	0.03
Triglyceride/HDL-cholesterol ratio	0.77 ± 0.4	1.66 ± 1.1	0.002

Data are number (percentage) of subjects or mean ± SD.

BMI, Body Mass Index; *HDL*, High-Density Lipoprotein; *LDL*, Low-Density Lipoprotein; *SSPG*, Steady-state plasma glucose

^aLipid data available in 12 of the 14 insulin-sensitive individuals.

Table 2

Risk of OSA and daytime sleepiness in insulin-sensitive and insulin-resistant individuals

Variable	Insulin-sensitive individuals (n=14)	Insulin-resistant individuals (n=22)	<i>p</i> value
Individuals at high risk for OSA by STOP questionnaire ^a	2 (14%)	14 (64%)	0.006
Individuals at high risk for OSA by Berlin questionnaire ^a	4 (29%)	16 (73%)	0.016
Individuals at high risk for OSA by STOP and Berlin questionnaire ^a	2 (14%)	14 (64%)	0.006
ESS score (points)	5.5 ± 2.3	8.9 ± 5.2	0.013
Individuals with ESS score ≥ 10	1 (7%)	11 (50%)	0.011

Data are number (percentage) of subjects or mean ± SD.

ESS, Epworth sleepiness scale; OSA, obstructive sleep apnea

^aScores ≥ 2 by STOP or Berlin questionnaires identified individuals at high risk for OSA.