Obesity, diabetes, and exercise associated with sleep-related complaints in the American population

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Abstract

Aim—Previous studies have demonstrated relationships between sleep and both obesity and diabetes. Additionally, exercise may improve sleep and daytime function, in addition to weight and metabolic function. The present study extends these findings by examining how general sleep-related complaints are associated with body mass index (BMI), diabetes diagnosis, and exercise in a large, nationally representative sample.

Subject and methods—Participants were respondents to the Behavioral Risk Factor Surveillance System (BRFSS). Sleep complaint (SC) was measured with "Over the last 2 weeks, how many days have you had trouble falling asleep or staying asleep or sleeping too much?" Daytime complaint (DC) was measured with "Over the last 2 weeks, how many days have you felt tired or had little energy?" Responses were dichotomized, with 6 days indicating complaint. Covariates included age, race/ethnicity, income, and education.

Results—Being overweight was associated with DC in women only. Obesity was significantly associated with SC and DC in women, and DC in men. Diabetes was associated with SC and DC in both genders. Any exercise in the past 30 days did not attenuate any BMI or diabetes relationships, but was independently associated with a decrease in SC and DC in both men and women.

Conclusion—These results suggest that for both men and women diabetes is a significant predictor of sleep and daytime complaints, and there is a relationship between obesity and sleep and complaints for women to a greater extent than men. Finally, exercise was associated with much fewer sleep and daytime complaints in both genders.

Introduction

Rates of obesity and diabetes are increasing and constitute a major public health issue. The rise of these conditions has been attributed to profound changes in societal and behavioral patterns, notably diet and exercise. Consonant with the growing obesity epidemic, there has been a growing concern about the relationships of sleep disturbances to a number of health outcomes (Grandner et al. 2010b; Grandner et al. 2010a; Colten et al. 2006). Sleep disturbances are significantly associated with obesity (Grandner et al. 2010b; Watanabe et al. 2010; Chaput et al. 2009a) as well as morbidity and mortality (Gallicchio and Kalesan 2009; Grandner et al. 2010d; Grandner et al. 2010b).

One aspect of sleep disturbance, sleep deprivation, has been explored in a number of ways with respect to obesity. Subjective habitual short sleep is associated with increased body mass index (BMI) (Watanabe et al. 2010; Thomas et al. 2009; Di Milia and Mummery 2009; Lauderdale et al. 2009; Cappuccio et al. 2008; Patel et al. 2008), less habitual sleep is

associated with a higher-fat diet (Grandner et al. 2010a), and experimental sleep deprivation is associated with physiological disruptions associated with obesity risk such as alterations in leptin and ghrelin (Mullington et al. 2009; Gangwisch 2009; Punjabi 2009; Hall et al. 2008; Van Cauter et al. 2008). In addition, decreased sleep duration is also a clinical feature of diabetes (Beihl et al. 2009; Chaput et al. 2009a; Tasali et al. 2009; Idris et al. 2009) and predicts its development (Beihl et al. 2009; Chaput et al. 2009a). While several explanations for the relationships between sleep and both obesity and diabetes have been postulated, the precise mechanisms of action are still under investigation.

Proposed mechanisms that have received the most attention include alterations in glucose tolerance, insulin resistance, and the metabolic hormones leptin and ghrelin. Current evidence from laboratory studies indicates that sleep deprivation is associated with impaired glucose tolerance and insulin resistance (Van Cauter et al. 2008; Van Cauter et al. 2007; Knutson et al. 2007). Additionally, sleep loss may alter the ability of leptin and ghrelin to accurately signal caloric need and may lead to increased food intake (Knutson et al. 2007). Epidemiological studies also demonstrate that short habitual sleep duration is associated with impaired glucose tolerance and insulin resistance (Gangwisch 2009; Spiegel et al. 2005; Schultes et al. 2005), as well as alterations of leptin and ghrelin secretion (Gangwisch 2009; Spiegel et al. 2005; Chaput et al. 2007). Together, these findings suggest that short sleep duration is an independent risk factor for the development of both obesity (Watanabe et al. 2010; Chaput et al. 2009b; Salihu et al. 2009) and diabetes (Tuomilehto et al. 2009; Knutson and Van Cauter 2008; Gangwisch et al. 2007), and that it may play a role in their development (Grandner and Patel 2009; Lévy et al. 2009; Schuster 2009; Gimble et al. 2009; Gangwisch 2009; Grandner et al. 2010b).

Although a major assumption of this work is that it is the sleep problems that are causally related to the health outcomes, it is likely that reciprocal relationships exist as well. Most of the studies in this domain are correlational and would reflect effects in both directions. Other studies have shown that intervening on risk factors for obesity and diabetes can have effects on sleep as well (Brand et al. 2010; Tuomilehto et al. 2009). Thus, although many previous studies target obesity and diabetes factors as outcomes with sleep as predictor, it is possible to explore relative influence of obesity and diabetes on sleep as an outcome, as it is likely that these relationships are, at least in part, bidirectional.

Adequate healthy sleep requires both sufficient sleep duration and good sleep quality; sleep disturbance may be due to habitual short sleep duration or chronic sleep deprivation, sleep fragmentation or sleep disruption, or even excessive sleep (Grandner et al. 2010c; Grandner and Patel 2009; Grandner et al. 2009; Lauderdale et al. 2008). Yet, as reviewed above, sleep duration has been the primary sleep variable in epidemiological surveys of obesity and diabetes and experimental sleep deprivation paradigms typically vary sleep duration rather than sleep quality.

Furthermore, physical activity is an important factor in not only obesity and diabetes, but it is also related to sleep (Brand et al. 2010; Reynolds et al. 2010; Ueno et al. 2009; Oliver et al. 2009). It is unclear, however, whether the degree of physical activity necessary for a significant impact on BMI or diabetes risk is necessary for a significant impact on sleep-related complaints (Gerber et al. 2010). It may be the case that a relatively small amount of physical activity may be insufficient to significantly impact obesity or diabetes risk directly, but may improve sleep and thus indirectly play a role.

Although it is well known that obesity is associated with diabetes and physical inactivity, and that all of these factors affect sleep, this study extends the existing literature in a few important ways: First, this study examines general sleep disturbance and fatigue, whereas

previous studies have examined the sleep disturbance resulting from sleep disorders (e.g., sleep apnea), sleep in artificial laboratory settings (e.g., sleep deprivation), or other aspects of sleep (e.g., sleep duration). Second, this study explores the unique contributions of obesity and diabetes to sleep disturbance and fatigue. Third, this study supports previous findings in a large, diverse sample.

The present study was designed to investigate whether obesity and diabetes diagnosis predict sleep disturbance and daytime fatigue in the American population and to what extent, if any, that exercise attenuates these relationships. Specifically, our hypotheses included the following:

1. Adjusting for all covariates (including diabetes), obesity is a significant predictor of increased sleep disturbance and daytime fatigue.

2. Adjusting for all covariates (including obesity), diabetes diagnosis is a significant predictor of increased sleep disturbance and daytime fatigue.

3. When exercise is added to the model, the relationship between both sleep disturbance and daytime fatigue will remain significant.

4. Exercise is independently associated with decreased sleep disturbance and daytime fatigue.

To address these hypotheses, we conducted an analysis using the Behavioral Risk Factor Surveillance System (BRFSS) for the year 2006.

Methods

Data source

Data were obtained from the 2006 BRFSS (Centers for Disease Control 2007). The BRFSS is a state-based, random-digit-dialed telephone interview survey of adults aged 18 years from all over the USA conducted every year. It is the world's largest telephone survey, designed to monitor health-related behaviors in the general population. For this study, participants were respondents who had answered a question on sleep complaint (SC): "Over the last 2 weeks, how many days have you had trouble falling asleep or staying asleep or sleeping too much?" To assess daytime complaint (DC), respondents answered the question: "Over the last 2 weeks, how many days have you felt tired or had little energy?" Answers for both questions ranged from 0 to 14. However, the distributions were bimodal, with highest peaks at 0 and 14 and few responses of 3–11 days; this non-normal distribution thus precludes analysis as a continuous variable. Thus, SC and DC were dichotomized into two categories: those who report complaints 6 days and those who report complaints <6 days. This is consistent with other classification approaches where a frequency of 3 or more events per week has been used [such as in insomnia where 3 nights per week or more of poor sleep is used (Perlis et al. 2010; American Academy of Sleep Medicine et al. 2006)].

Covariates used to estimate socioeconomic factors included education (less than high school, high school graduate, some college, college graduate) and income level (<US $10,000 pretax income per year, US $10,000–$15,000, US $15,000–$20,000, US $20,000–$25,000, US $25,000–$35,000, US $35,000–$50,000, US $50,000–$75,000, and > US $75,000). Previous analyses have found that education and income are significant predictors of SC in this sample (Grandner et al. 2010c; 2009).

Obesity was estimated by calculating BMI based on self-reported estimates of weight and height and categorizing volunteers as normal weight (BMI<25), over-weight (BMI=25−29.9), or obese (BMI 30)—categories widely used in research and practice.

Although calculation of BMI based on self-report may be problematic, BMI data from the BRFSS are accepted estimates in other studies (Fairley et al. 2010; Heo et al. 2010; Jia and Lubetkin 2010). Diabetes diagnosis history was obtained via self-report.

The presence of exercise was assessed by asking participants the following question: "During the past month, other than your regular job, did you participate in any physical activities or exercises such as running, calisthenics, golf, gardening, or walking for exercise?" (coded "yes" or "no"). This item, in particular, was chosen for a number of reasons. Numerous studies have shown that increased exercise is associated with decreased risk for diabetes and decreased BMI. However, the amount of moderate to vigorous activity (generally required for significant effects on BMI and diabetes risk) in the general population is probably very low (Hubácek 2009; Macfarlane and Thomas 2010). Additionally, subjective estimates of moderate and vigorous exercise (especially those requiring precision) are known to be unreliable. The chosen item reflects these limitations by assessing exercise with minimal precision (with no need to estimate intensity or duration, which would likely be inaccurate) and allowing for an amount of activity (minimal) that would more likely be attainable by the majority of the population. In addition to these benefits, there are a number of problems with this item. For example, interpretation of results may be difficult. If no significant effect is found, it would be unclear whether a significant effect could have been found with a measure that was related to the higher levels of exercise previously reported to be associated with reductions in BMI and diabetes risk. If a significant effect is found, the measure is so broad that it is unclear what level of exercise would be required to show the effect. Despite these limitations, this item was chosen because of its novelty and ability to reflect minimal exercise in a way that may be more reliable than other estimates.

Statistical analyses

Complete-case analysis was implemented for both SC and DC; thus, only participants who provided complete data were included for each analysis. Percentages of respondents indicating SC and DC across variables were calculated and differences in reported SC and DC among groups were compared using Rao-Scott chi-square tests using SAS software (SAS Institute 2008) with the PROC SURVEYFREQ procedure. Data were also reviewed graphically to assess distribution and relationship to sleep disturbance.

The logit of odds of the categorical SC and DC was modeled using generalized linear models for each individual variable and all combined variables both for all and separated by gender. Age was considered as a continuous variable while others were considered as categorical variables, to best model how the data were originally collected and to maximize resolution. All sampling was weighted appropriately for representativeness, using weighting scores specifically developed for BRFSS 2006 (Centers for Disease Control 2007). The odds ratios (ORs) and 95% confidence intervals (CIs) were estimated among groups relative to a preselected reference. Analyses were performed using SAS software (SAS Institute 2008) with the PROC SURVEYLOGISTIC procedure. All statistical tests were two-tailed. Statistical significance was set at the $p<0.05$ level unless otherwise indicated. Interactions for all variables with race/ethnicity were computed separately for men and women.

For men and women, for both SC and DC, three models were computed: (1) BMI + diabetes, (2) BMI + diabetes + covariates (age, education, income), and (3) BMI + diabetes + covariates + exercise. The first model explores unadjusted effects, and the second model explores the contribution of age, education, and income to the unadjusted findings. The final model includes exercise, to determine its value as an independent predictor and to evaluate the impact of exercise on the degree of the relationship between SC/DC and both BMI and diabetes.

Results

Subject characteristics

A total of n=156,252 participants provided complete data for the SC analyses and n=155,761 participants provided complete data for the DC analyses. Characteristics of the samples are reported in Table 1 (SC) and Table 2 (DC). Based on this classification, 19.37% of the sample (21.76% of women and 15.87% of men) were classified as reporting SC (SC+) and 22.81% of the sample (25.78% of women and 18.47% of men) were classified as reporting DC.

Regression results

Results of unadjusted analyses are reported in Table 3 for SC and Table 4 for DC when considering BMI and diabetes (model 1). For women, overweight, obesity, and diabetes were significantly associated with an increased odds of both SC and DC. For men, the relationships varied: diabetes diagnosis was significantly associated with an increased OR for both SC and DC; obesity status was significantly associated with an increased OR for DC only (not SC); and overweight status was associated with a reduced odds risk for both SC and DC.

Results of adjusted analyses (model 2: BMI + diabetes + covariates of age, education, and income) are presented in Table 3 for SC and Table 4 for DC. After adjusting for age, income, and education, overweight status no longer is significantly associated with an increased OR for SC in women and a decreased OR for SC and DC in men. All other relationships are generally maintained: diabetes diagnosis predicting both SC and DC in men and women and obesity was associated with both SC and DC in women, and only DC in men.

Results of adjusted analyses including exercise (model 3: BMI + diabetes + exercise + covariates of age, education, and income) are presented Table 3 for SC and Table 4 for DC. Though slightly attenuated, all of the significant relationships found in the previous models were maintained after adjusting for exercise. This is despite the finding that exercise itself was significantly associated with SC and DC in both men and women, such that individuals who reported any exercise were about half as likely to report DC and about a third less likely to report SC. Figure 1 represents the findings of this model for SC, and Fig. 2 represents the findings for DC.

Because the pattern of findings for SC and DC were so similar, we explored whether these two variables were measuring the same construct. However, although there was a significant correlation between SC and DC (r=0.32, p<0.0005), each of these two variables only explain approximately 10% of the variance in the other (r^2=0.1024). Thus, they represent two very different constructs that, though related, are not collinear.

Discussion

The present study evaluated BMI and diabetes diagnosis as predictors of sleep disturbance in a large representative sample of the American population. This was explored using 3 models: model 1 (BMI + diabetes), model 2 (BMI + diabetes + covariates), and model 3 (BMI + diabetes + covariates + exercise). The findings suggest a significant relationship between DC and both overweight and obesity status in women and obese (but not overweight) men. In contrast, only in women was obesity significantly associated with SC. Diabetes was significantly associated with both SC and DC in men and women. Any exercise in the past 30 days was associated with reduced levels of both SC and DC, though

the ORs for the other relationships were not notably affected, suggesting that the variance explained by exercise is different than that explained by obesity or diabetes.

It is important to note that previous studies have tended to view sleep disturbance (however measured) as an independent variable, whereas the analyses in the present study view sleep as a dependent variable influenced by obesity, diabetes, and exercise. This is not meant to imply a unidirectional relationship where sleep problems are caused by obesity/diabetes/ exercise. Rather, we designed our statistical analyses around the notion that obesity, diabetes, and exercise are related constructs, each linked to sleep. Using this approach, the relative variance contributions to sleep-related complaints of these related constructs can be evaluated simultaneously.

Sleep disturbance is associated with diabetes risk independent of obesity

Several previous studies have shown a relationship between diabetes and sleep deprivation (Gangwisch 2009; Knutson and Van Cauter 2008). Fewer studies have investigated the association with sleep disruption, daytime sleepiness, and fatigue. Nonetheless, significant relationships have been reported (Knutson et al. 2006). Several studies of sleep apnea patients (who experience sleep disruption) have demonstrated that this condition is a significant predictor of diabetes diagnosis (and vice versa) (Ronksley et al. 2009). This study extends those previous studies to a much larger and more representative sample. That the relationship of diabetes to SC and DC was independent of covariates, BMI, and exercise, and nearly identical in men and women, suggests that there is something specific to the pathophysiology of diabetes itself that confers risk. The specific nature of this risk still needs to be explored, but it may have to do with alterations in insulin sensitivity and glucose metabolism (Barf et al. 2010; Lui and Ip 2010; Buxton et al. 2010) associated with sleep disruption. Additionally, other factors associated with diabetes that have been independently associated with sleep disturbance over and above effects of BMI may be implicated, such as atherogenic lipoprotein profile (Grandner et al. 2010a; Gangwisch et al. 2010; Kaneita et al. 2008; Mackiewicz et al. 2008; Mackiewicz et al. 2007; Williams et al. 2007), inflammatory factors (Jain and Mills 2007; Opp et al. 2007; Irwin 2002; Meier-Ewert et al. 2004; Okun et al. 2009), and the hormones leptin and ghrelin (Knutson and Van Cauter 2008; Van Cauter et al. 2008; Chaput et al. 2007; Littman et al. 2007; Knutson et al. 2007; Gangwisch 2009; Copinschi 2005; Spiegel et al. 1999; Spiegel et al. 2004a, b).

It is possible that sleep apnea may also play a role in the link between diabetes and SC or DC. The fact that diabetes remained a risk factor even after controlling for obesity/ overweight status suggests that there are additional factors associated with diabetes. It is possible that the presence of diabetes increases the risk of sleep apnea beyond that associated with a specific BMI category, or that diabetes itself causes SC or DC through other pathology.

It is unclear whether the relationship between sleep and diabetes is unidirectional or bidirectional. Increasing evidence suggests that this relationship is complex (Eastwood et al. 2010; Bopparaju and Surani 2010; Celen et al. 2010; Leow 2010; Lam et al. 2010; Zizi et al. 2010; Barone and Menna-Barreto 2010). These findings support the proposition that maintaining healthy sleep can be included in strategies for diabetes prevention as well as the suggestion that diabetes preventive activities should incorporate an aim of reducing sleep disturbance. For example, the recent IMAGE guidelines for preventing type 2 diabetes includes a discussion of sleep (Lindström et al. 2010).

Sleep complaint and daytime tiredness are associated with obesity

Numerous studies have found that short sleep duration is a significant predictor of BMI (Grandner et al. 2010b; Chaput et al. 2009b; Must and Parisi 2009; Grandner and Patel 2009), and the findings from laboratory studies have also supported this (Nedeltcheva et al. 2009). Studies in the sleep apnea literature have found significant relationships between sleep fragmentation (due to respiratory events) and daytime sleepiness and fatigue (Yue et al. 2009). However, previous studies have not examined this issue more broadly by looking for a more general link between obesity and sleep quality. Of note, being overweight was significantly related to both SC and DC in men and women in unadjusted analyses, but the inclusion of age and socioeconomic factors attenuate this relationship significantly, suggesting that this relationship may have been related to a general unhealthy lifestyle, to which overweight is related as well as age and socioeconomic factors, rather than BMI. It should be noted that the findings were more robust in women than men, replicating previous findings regarding sleep duration and BMI (Kripke et al. 2002) and cardiovascular risk (Cappuccio et al. 2007). Not all studies have found this pattern, though (Watanabe et al. 2010)

Presence of physical activity is important

Although a significant relationship among obesity, diabetes, and poor sleep has been reported frequently in the literature, few studies have examined the role of exercise. Self-report measures of exercise are difficult because responses depend on social desirability and demand characteristics of the individual study (Youngstedt and Kline 2006). Thus, we chose a variable that presumably would have high reliability, despite low resolution. Including a simple measure of any exercise in the past 30 days into the model resulted in two notable findings: (1) the ORs for obesity and diabetes were attenuated only very slightly, even though (2) this simple measure of exercise was strongly, inversely related to both SC and DC.

That there was little attenuation of the obesity and diabetes relationships in the presence of exercise suggests that exercise does not directly alter the relationship of sleep, diabetes, and obesity. The mechanism by which diabetes and obesity are associated with SC or DC may not be impacted by exercise as measured in this study. Previous studies have found that health outcomes of obesity and diabetes are affected by exercise (Macfarlane and Thomas 2010), and this large study raises some questions about how this is involved in sleep and tiredness.

The finding that exercise had beneficial effects on SC and DC in the larger adjusted model is encouraging. It suggests that even in the presence of obesity or diabetes, and irrespective of age or socioeconomic factors, exercise can have a beneficial effect on SC and DC. Previous studies have found beneficial effects of exercise on sleep (Brand et al. 2010). However, these studies often found relatively modest effects, with careful measurement of physical activity. That such a general measure was so highly predictive, given its imprecise measurement, is surprising. Perhaps, for general sleep quality, it is not the intensity or duration of exercise that is important—merely its presence (or the subjective judgment of its presence) is sufficient to categorically improve sleep quality and daytime tiredness.

Limitations

This study has a number of limitations. First, the SC and DC items are not specific for particular symptoms. This limits our ability to use responses to this item to describe symptoms or syndromes (e.g., insufficient sleep, long sleep, insomnia, sleep apnea, daytime sleepiness). The broad nature of the SC question in particular captures a gamut of etiologies for suboptimal sleep including acute and chronic partial sleep deprivation, insomnia, poor

sleep quality, and excessive sleep. SC is not a typical metric that has been employed in epidemiological analyses of sleep in the population. However, it provides a valuable and sensitive estimate of suboptimal sleep, for whatever reason, in the general population. We purport that the item is sensitive because almost any problem associated with sleep—especially the most common (sleep insufficiency, sleep fragmentation, insomnia, sleep apnea, restless legs, etc.)—could be captured by this question. However, this question has not been validated against standard measures of subjective and objective sleep. This is a major limitation of these items. However, we still believe that they are useful, as they provide an overall population-level indicator of the presence of sleep complaints, with almost none of the limitations that would be applied if specific symptoms had been targeted. This general level of complaint demonstrates a great deal of face validity and, more importantly, external validity, as it represents the vague complaints presented when people discuss sleep problems (Cole et al. 2007; Grandner et al. 2006). This is pertinent as recognition for the importance of sleep and sleep disorders in public health continues to rise (Colten et al. 2006).

Second, cross-sectional analyses limit our ability to comment on causality. Thus, SC and DC ORs may reflect other causes as well.

A third limitation is the self-report nature of the survey. Diabetes may be underreported. Also, self-reports of BMI are known to have limited reliability and validity, though they have been useful in documenting population trends (Fairley et al. 2010; Heo et al. 2010; Strine et al. 2005; Richardson et al. 2008). Regarding the exercise question, subjective self-reports of exercise frequency and severity are known to be very unreliable and may not accurately reflect actual activity. This question avoids this problem by not asking details—any exercise in the past 30 days is all that is asked. As would be expected, most individuals endorsed this item, and it is probably not related to much, if any, moderate or strenuous activity. This may explain why the addition of this item to the model did not affect the ORs for diabetes and obesity, although physical activity is a well-known factor in these conditions; the sort of physical activity captured by this question is probably insufficient to have an impact on these. However, that there was such a strong relationship with SC and DC is rendered even more surprising then, as these findings suggest that even very small amounts of activity might have profound impacts on sleep and tiredness.

Conclusions

The large representative sample size and broad sleep measures allow us to posit important population-level observations about sleep in the population. Based on these data, we conclude that obesity and diabetes are significant independent predictors of sleep disturbance and daytime complaints, though the relationship with obesity is notably stronger in women than men. A broad measure of exercise (any at all in the past 30 days) was a significant predictor of lack of both sleep disturbance and daytime complaints, with those reporting exercise one third less likely to report sleep problems and half as likely to report daytime tiredness. Analyses of smaller sample sizes reporting diabetes and obesity effects on sleep are supported by this analysis.

Regarding future directions, the biological pathways linking diabetes, sleep disturbances, and daytime sequelae of poor sleep remain to be fully elucidated. From a public health perspective, this study highlights the importance of sleep in public health campaigns targeting obesity (and diabetes) and the potential benefits of exercise.

Acknowledgments

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Fig. 1.
Graphical representation of ORs for SC from multivariate analysis that included BMI categories, diabetes diagnosis, and report of exercise in the past 30 days, adjusted for age, income, and education (model 3). Reference categories were normal weight (BMI), no diagnosis (diabetes), and no exercise (exercise)
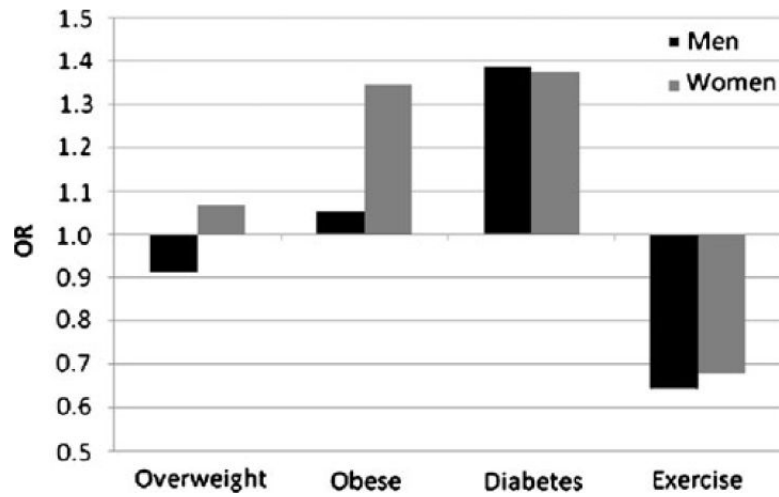
Fig. 2.
Graphical representation of ORs for DC from multivariate analysis that included BMI categories, diabetes diagnosis, and report of exercise in the past 30 days, adjusted for age, income, and education (model 3). Reference categories were normal weight (BMI), no diagnosis (diabetes), and no exercise (exercise)
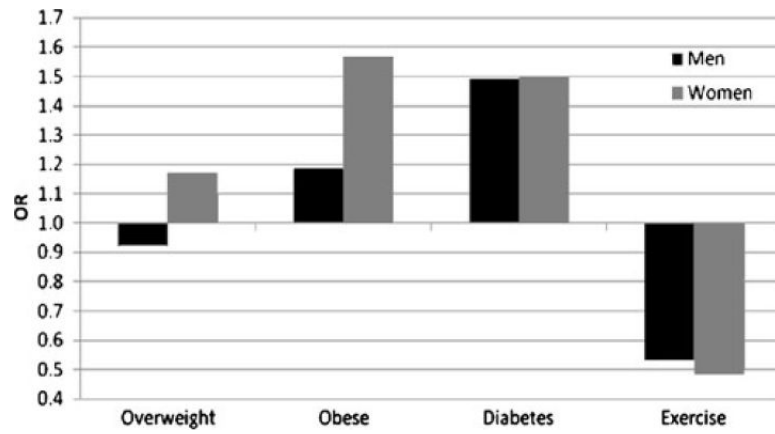
Table 1

Characteristics of participants who responded to the SC question, separated by group[a]

| | Women (*n*=92,924) | | | Men (*n*=63,328) | | |
Category	SC− (%)	SC+ (%)	Total (%)	SC− (%)	SC+ (%)	Total (%)
Age						
Mean	53.06	51.43	53.18	51.71	51.12	52.15
Education						
Less than high school	6.62	2.93	9.55	7.58	2.34	9.92
High school	22.73	7.29	30.02	23.81	5.21	29.02
Some college	21.82	6.56	28.38	20.76	4.21	24.97
College graduate	27.07	4.98	32.04	31.98	4.11	36.09
Income						
< US $10,000	4.67	2.64	7.31	2.71	1.40	4.10
US $10,000–$15,000	5.13	2.36	7.49	3.32	1.44	4.76
US $15,000–$20,000	6.51	2.45	8.95	4.82	1.44	4.76
US $20,000–$25,000	7.66	2.54	10.20	6.66	1.74	8.40
US $25,000–$35,000	10.51	2.90	13.41	10.71	2.22	12.92
US $35,000–$50,000	12.99	3.13	16.12	14.74	2.48	17.22
US $50,000–$75,000	13.27	2.72	15.99	16.58	2.31	18.89
US $75,000+	17.50	3.03	20.53	24.61	2.84	27.45
BMI						
Normal	34.46	7.86	42.31	23.38	4.34	27.72
Overweight	24.55	6.55	31.10	38.78	6.33	45.11
Obese	19.24	7.35	26.58	21.98	5.19	27.17
Diabetes						
No	69.70	18.24	87.94	74.71	13.15	87.87
Yes	8.55	3.51	12.06	9.42	2.71	12.13
Exercise						
No	18.27	7.99	26.25	17.14	5.35	22.49
Yes	59.98	13.77	73.75	67.00	10.52	77.51

[a]SC+ indicates that difficulties were reported 6 days across 2 weeks; SC- indicates that difficulties were reported 6 days across 2 weeks

Table 2

Characteristics of participants who responded to the DC question, separated by group[a]

Category	Women (n=92,645)			Men (n=63,116)		
	DC− (%)	DC+ (%)	Total (%)	DC− (%)	DC+ (%)	Total (%)
Age						
Mean	52.96	51.54	52.61	51.43	52.08	51.54
Education						
Less than high school	5.96	3.55	9.51	7.12	2.74	9.87
High school	21.46	8.53	29.99	22.66	6.38	29.04
Some college	20.76	7.66	28.42	20.15	4.85	25.00
College graduate	26.03	6.04	32.08	31.59	4.50	36.09
Income						
< US $10,000	4.15	3.16	7.30	2.56	1.52	4.08
US $10,000–$15,000	4.54	2.90	7.44	3.08	1.70	4.77
US $15,000–$20,000	6.06	2.90	8.96	4.47	1.81	6.29
US $20,000–$25,000	7.14	3.05	10.19	6.34	2.04	8.38
US $25,000–$35,000	9.92	3.48	13.40	10.26	2.64	12.90
US $35,000–$50,000	12.43	3.70	16.13	14.27	2.95	17.22
US $50,000–$75,000	12.79	3.23	16.01	16.17	2.73	18.91
$75,000+	17.19	3.38	20.57	24.37	3.08	27.45
BMI						
Normal	33.42	8.88	42.29	22.78	4.95	27.73
Overweight	23.43	7.65	31.08	37.91	7.21	45.17
Obese	17.36	9.27	26.63	20.83	6.32	27.15
Diabetes						
No	66.65	21.30	87.96	72.82	15.05	87.87
Yes	7.56	4.49	12.04	8.71	3.42	12.13
Exercise						
No	15.53	10.72	26.26	15.46	6.96	22.42
Yes	58.67	15.07	73.74	66.07	11.51	77.58

[a]DC+ indicates that difficulties were reported   6 days across 2 weeks; DC- indicates that difficulties were reported   6 days across 2 weeks

Table 3

OR and 95% CI for SC

Variable	Women			Men		
	OR	CI		p	OR	CI		p
Model 1: BMI + diabetes						
BMI (reference = normal)						
Overweight	1.117	1.029 1.212	0.0082	0.833	0.734 0.946	0.0049
Obese	1.556	1.430 1.694	<0.0001	1.039	0.916 1.178	0.2035
Diabetes (reference = no diagnosis)						
Yes	1.488	1.348 1.641	<0.0001	1.459	1.295 1.643	<0.0001
Model 2: BMI + diabetes + age + education + income						
BMI (reference = normal)						
Overweight	1.079	0.993 1.173	0.0718	0.919	0.807 1.047	0.2035
Obese	1.408	1.293 1.534	<0.0001	1.090	0.959 1.238	0.1873
Diabetes (reference = no diagnosis)						
Yes	1.396	1.265 1.541	<0.0001	1.409	1.238 1.603	<0.0001
Model 3: BMI + diabetes + age + education + income + exercise						
BMI (reference = normal)						
Overweight	1.068	0.982 1.162	0.122	0.913	0.802 1.040	0.1723
Obese	1.346	1.235 1.467	<0.0001	1.053	0.927 1.197	0.4297
Diabetes (reference = no diagnosis)						
Yes	1.375	1.244 1.519	<0.0001	1.388	1.218 1.583	<0.0001
Exercise (reference = no exercise last 30 days)						
Yes	0.678	0.627 0.734	<0.0001	0.643	0.573 0.720	<0.0001

Table 4

OR and 95% CI for DC

Variable	Women				Men			
	OR	CI		*p*	OR	CI		*p*
Model 1: BMI + diabetes								
BMI (reference = normal)								
Overweight	1.209	1.117	1.310	<0.0001	0.863	0.772	0.966	0.0101
Obese	1.846	1.703	2.000	<0.0001	1.208	1.077	1.355	0.0013
Diabetes (reference = no diagnosis)								
Yes	1.580	1.439	1.736	<0.0001	1.653	1.476	1.850	<0.0001
Model 2: BMI + diabetes + age + education + income								
BMI (reference = normal)								
Overweight	1.191	1.097	1.293	<0.0001	0.931	0.828	1.046	0.2262
Obese	1.696	1.562	1.841	<0.0001	1.247	1.108	1.404	0.0003
Diabetes (reference = no diagnosis)								
Yes	1.534	1.393	1.688	<0.0001	1.516	1.346	1.708	<0.0001
Model 3: BMI + diabetes + age + education + income + exercise								
BMI (reference = normal)								
Overweight	1.171	1.077	1.273	0.0002	0.922	0.820	1.037	0.1754
Obese	1.568	1.441	1.706	<0.0001	1.187	1.054	1.338	0.0048
Diabetes (reference = no diagnosis)								
Yes	1.496	1.358	1.648	<0.0001	1.490	1.318	1.685	<0.0001
Exercise (reference = no exercise last 30 days)								
Yes	0.486	0.450	0.524	<0.0001	0.533	0.482	0.589	<0.0001