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Lifestyle Intervention for Sleep Disturbances among Overweight or Obese Individuals

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Abstract

Little is known about the effect of different lifestyle interventions on sleep disturbances among sedentary obese or overweight persons. We randomized 35–65 year-old men and women, to 6-months of a weight loss diet (D); or D combined with supervised exercise training (D+E). Measurements were self-reported sleep disturbances; the Profile of Mood States questionnaire; BMI; total, abdominal subcutaneous and visceral fat by magnetic resonance imaging; and aerobic fitness expressed as VO_2peak. The groups did not differ in changes for body weight, abdominal total fat, VO_2peak, and sleep disturbances. The novel finding herein is that reduced abdominal subcutaneous fat and depressive symptoms with either D or D+E, were associated with less sleep disturbances.

Keywords

sleep disturbance; lifestyle intervention; obesity; Type 2 diabetes

The increased rates of obesity and type 2 diabetes (T2D) have paralleled the rise in the number of reported sleep disturbances (Liu, Hay, & Faught, 2013), suggesting a strong relationship between sleep and chronic disease, including cardiovascular disease (CVD). Though mechanistic links have not been firmly established, sleep disturbances appear to induce weight gain by impairing glucose tolerance, and by increasing cortisol levels and sympathetic activity (Chaput, Klingenberg, Astrup, & Sjodin, 2011). Recently, it has been shown that poor sleep compromises dietary efforts to reduce adiposity, by altering the levels of appetite regulating hormones, thus reducing adherence to dietary regimens (Nedeltcheva, Kilkus, Imperial, Schoeller, & Penev, 2010). Specifically, lack of sleep increases ghrelin and decreases leptin resulting in increased appetite (Taheri et al., 2004). Moreover, it is possible that exercise has an effect on pineal function and thermoregulation that influence sleep

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(Atkinson & Davenne, 2007). Despite the growing body of evidence showing that diet, exercise, and sleep are interrelated (Schmid et al., 2009), lifestyle interventions targeting sleep improvement have been limited to young or relatively healthy volunteers (Driver & Taylor, 2000). Yet, the generalizability of these findings to high risk groups such as overweight and obese populations with impaired glucose metabolism is unknown.

The current study is a sub-study of a larger trial (NCT00928005, ClinicalTrials.gov) that was designed to examine the effects of lifestyle interventions on CVD outcomes among obese/overweight individuals with T2D or pre-diabetes. Using these data, the primary aim of the current analysis was to examine the effects of a weight loss diet combined with structured exercise (D+E) versus diet alone (D) on self-reported sleep disturbances. A secondary aim was to identify factors related to change in sleep disturbances. We hypothesized that D+E would be more effective than D alone for improving sleep disturbances by inducing greater reduction in body fat.

Subjects and Methods

The study was approved by the Johns Hopkins Institutional Review Board and written informed consent was obtained from all subjects. Seventy-seven subjects were randomized to a D intervention only group or D+E group. Subjects were 35–65 years of age with a body mass index (BMI) in the range of 26 and 42 kg/m^2, pre-diabetes or T2D as verified by medical records or blood test results. Study exclusions were a fasting glucose >400 mg/dl, glycosylated hemoglobin (HbA1c) >11%, or use of insulin. Other key exclusions were regular physical activity (< 90 minutes of moderate-intensity activity/week), history of CVD, cigarette smoking in the last 6 months, alcohol abuse, and any other major health conditions that could impair exercise ability.

Measures

All study outcomes were measured at baseline and following the 6-month intervention. Details about the following study measures were reported elsewhere (Nam, Dobrosielski, & Stewart, 2012): Body composition was determined using dual energy X-ray absorptiometry (DEXA; GE Lunar Prodigy; GE Medical Systems, Milwaukee WI, Software V. 13). Total abdominal, visceral and subcutaneous fat volumes were measured from images obtained by MRI (Siemens Vision 1.5T; Siemens Medical Systems, Iselin, NJ). Peak oxygen uptake was determined using a modified Bruce protocol on a treadmill with a Cardinal Health Vmax 229 Metabolic System (Cardinal Health, Inc. San Diego, CA), resting blood pressure, body weight, and height, were measured using standard procedures. Fasting plasma glucose, insulin, HbA1c levels, and lipids were determined using standard assays.

Sleep disturbances were determined using the Johns Hopkins Sleep Survey that identifies symptoms of apnea, daytime fatigue, hypersomnolence, circadian rhythm sleep disorder, insomnia, restless sleep, parasomnia, and dream disorders. Sleep disturbances is expressed as a composite score of all these items. The Cronbach's α or our study was 0.82 and validity was tested with Epworth Sleepiness Scale ($r=0.457$ $p<0.001$) (Johns, 1991). The Medical Outcomes Study Short-Form 36-Item Health Survey (SF-36) (Cronbach's α for our study range:0.74–0.86) (Ware & Sherbourne, 1992) and the Profile of Mood States' depression

subscale (McNair, Lorr, & Droppleman, 1981), (Cronbach's α for our study: 0.91) were used to measure health-related quality of life and depressive mood states, respectively.

Diet intervention (D)—Each subject was seen by a dietitian initially and then weekly for the first 2 months and biweekly for 4 months. The recommended diet was designed to produce a 600 kcal deficit/day for each individual. The diet was structured to enhance glycemic control and to minimize CVD risk factors (ADA, 1997). Adherence to diet intervention and modifications made by the dietitian were guided by how much weight was lost and by examining 7-day food records.

Diet plus exercise intervention (D+E)—This group followed the same diet intervention as the D group. A 600 kcal deficit/day was prescribed on non-exercise days. Because there was an estimated energy expenditure of 250 kcal from the exercise program on exercise days, the recommended dietary deficit was 350 kcal. Subjects were scheduled to exercise on three nonconsecutive days per week for 6 months at the Johns Hopkins Clinical Research Unit's Exercise Facility. An exercise physiologist supervised all exercise sessions to ensure that each participant followed the American College of Sports Medicine Guidelines (American College of Sports Medicine, 2006).

Statistical analysis—Descriptive statistics and univariate analyses were conducted to compare differences in baseline characteristics and 6-month changes between the two interventions. A multivariate linear regression analysis including both groups was conducted to examine predictors of improvement in sleep disturbances. Correlations of ≥0.80 between independent variables, a tolerance of ≤0.1, and variance inflation factors (VIF) of >5 were considered indicative of multicollinearity. When a bivariate correlation of ≥0.8 was found, only 1 of the 2 independent variables was included in a multivariate regression model. In addition, a priori selection of potential predictors was made on the basis of factors that could affect sleep disturbances (Magee, Caputi, & Iverson, 2011). Sensitivity analysis was conducted to examine the impact of missing data on study outcomes by including the entire sample. Two approaches to handle missing data were as follows: (1) replace with the group mean, and 2) baseline-observation-carried-forward (BOCF). For all analysis, the level of significance was set at $p < 0.05$ (two-sided).

Results

Of 77 subjects randomized, 55 subjects (54.8±7.8 years; 24 [D], 31 [D+E]) completed the study. The dropouts were 14 of 38 (36.8%) in the D group and 8 of 39 (20.5 %) in the D+E. There were no significant differences in any baseline parameters between groups (Table 1).

Group comparison

At 6 months, both groups improved from baseline ($p < 0.05$ for all) but the groups did not differ in their changes for body weight (D: −6.04 ± 4.54 kg; D+E: −6.68 ± 4.48 kg, $p = 0.61$), abdominal total fat (D: −101.93 ± 68.67 cm^2; D+E:−104.16 ± 72.37 cm^2, $p = 0.92$), VO_2peak (D: 2.26 ± 3.92 ml/kg/min; D+E: 3.71 ± 2.65 ml/kg/min, $p=0.11$), and sleep disturbances (D: −3.53 ± 7.69; D+E: −0.37 ± 7.23, $p = 0.16$).

Bivariate analysis

The total sample of 55 was analyzed using bivariate analysis irrespective of the group assignment. A reduction in sleep disturbance score was associated with a reduction in BMI (r=0.47, p <0.01); abdominal subcutaneous fat (r =0.552, p <0.01); abdominal total fat (r=0.47, p <0.01); and depressive symptoms (r=−0.347, p <0.05). The reduction in abdominal subcutaneous fat was associated with a greater improvement in fitness (r= −0.360, p <0.05) but was not associated with improvement in all SF-36 subscales or depressive symptoms. The reduction in depressive symptoms was associated with improvement in sleep disturbances (r=−0.347, p <0.05) and the mental composite score of the SF-36 (SFMCS) (r=−0.375, p <0.05). The variables that were not significantly associated with improvement in sleep disturbances were age, sex, a change in fitness, HbA1c, lipids, status of T2D or pre-diabetes and the intervention group assignment.

The number of exercise session attended (adherence) was associated with a reduction in abdominal subcutaneous fat (r=−0.498, p <.05) and BMI (r=−0.455, p <.05) and improved fitness (VO_2peak, r=.367, p <.05). The baseline score of the sleep apnea subscale correlated with total abdominal fat (r=0.27, p <0.05), and abdominal visceral fat at baseline (r= 0.57, p <0.01). The baseline insomnia score was correlated with BMI (r=−0.26, p <0.05), waist circumference (r=−0.25, p <0.05), total abdominal fat (r=−0.28, p <0.05) and abdominal visceral fat (r=−0.36, p <0.01) at baseline, indicating that individuals with overweight and obesity tend to have relatively more self-reported insomnia than other sleep disturbance problems.

Multivariate analysis

The multiple regression model that included all the significant predictor variables from bivariate analyses and priori selected variables based on our conceptual model–i.e., age; baseline sleep composite score; changes in depression, SFMCS, and fitness; and a reduction in abdominal subcutaneous and visceral fat–explained 62% of the variance in the sleep disturbance improvement (R^2=0.618, p <0 .0001). In the multivariate model, adjusting for baseline sleep disturbances, a reduction in subcutaneous fat (β = 0.39, p <0.01) and improvements in depressive symptoms (β = −0.56, p <0.01) were associated with improved sleep disturbances. In addition, we used BOCF to handle missing data to provide a more conservative reflection of the study outcomes; that is, the overall regression model including missing data (both “replacing with group mean” and “BOCF” methods) remained significant, which showed the robustness of the study results. Reduction in subcutaneous fat and improvements in depressive symptoms were still significantly associated with sleep disturbance improvements (supplement 1: sensitivity analysis).

Discussion

The key finding was that a 6 month- lifestyle intervention (D or D+E) improved self-reported sleep disturbances among sedentary obese or overweight individuals with T2D or pre-diabetes. Although we hypothesized that the D+E intervention would show more significant abdominal fat loss and less sleep disturbances, both intervention groups lost a similar amount of body weight over the 6 months and there was no difference in changes in

self-reported sleep disturbances, body weight, abdominal total fat, and VO2 peak between the two groups.

Our data extend previous findings by demonstrating that abdominal subcutaneous fat is an important factor in improving sleep disturbances regardless of the type of lifestyle intervention used to induce weight loss. For example, increased exercise improved sleep disturbances in some studies, while other studies found no correlation between exercise and sleep disturbances in healthy young or older subjects (Kline et al., 2012; Oudegeest-Sander et al., 2013). Our study suggests that the loss of body fat may be a potential mediator in the effect of lifestyle intervention on sleep disturbance. Thus, when explaining the relationship between sleep and exercise, the extent of body fat loss or weight loss from interventions should be taken into account, — whether the body fat loss from the intervention was substantial enough to lead to a significant, positive effect of exercise on sleep.

Though visceral fat is strongly linked to sleep apnea and, in turn, to CVD risk (Giusti et al., 2004), in our sample of sedentary, obese or overweight individuals who participated in diet alone or diet plus exercise, reduction in subcutaneous fat was the strongest body composition predictor of sleep disturbance improvement. One possible explanation for this finding is that when people lose a substantial amount of body weight, they lose relatively more subcutaneous fat than visceral fat. The volume of subcutaneous fat– which is much larger than visceral fat– may play a more important role in sleep disturbance improvement in the current study (i.e., mechanical effects of a large volume of subcutaneous fat on pharyngeal soft tissues) (Schwartz et al., 2008). However, one important caveat is that our study could not address specific sleep disorders (e.g. apnea) due to our use of the broad sleep measure.

Sleep disturbances are also independently affected by improvement in the level of depression; however, these results should be interpreted with caution because sleep disturbances are one of the core symptoms in depression. There may be an overlapped construct between sleep and depression from the self-reported measures and thus the association may be overestimated. Future studies including objective measures of sleep disturbances are needed to better understand the association between depression and sleep, given the high prevalence of depression in persons with diabetes and other chronic disease.

There are some limitations in our study. Because the present study was a sub-study of a trial of weight loss on CVD health, the subjects were not recruited specifically for sleep disturbances. Thus, the effects of our intervention may have been different if we had recruited subjects based on having sleep disturbances at baseline. Also, without a non-intervention control group, the potential changes and variability in our study variables– including sleep disturbances over time– may not be captured. We also acknowledge that with use of the broad sleep measure and the preliminary nature of our study related to specific sleep disorders, depression and abdominal subcutaneous fat, differentiating between sleep disorders that might have distinctively different mechanisms through which they impact body weight and specific lifestyle behaviors may not be possible. Future studies including objective and specific measures of sleep would be also helpful to better understand the relationship between sleep and various self-reported psychosocial factors. Another

limitation is our relatively high dropout rate, which often happens during diet and exercise interventions (Schmidt, Gruman, King, & Wolfson, 2000). One possible reason for more dropouts in the diet group than in the combined group could be that the subjects might have expected to participate in a structured exercise when they signed up for our study. If assigned to diet alone group, however, some subjects might have lost their interest in continuing to participate in the study. For the diet plus exercise group (D+E), the intensity of exercise and commitment could be challenges for consistent and continued participation. Therefore, the study results may be affected by the more adherent and motivated subjects who completed the study.

The strengths of our study include the use of a supervised exercise program and a dietitian monitored diet intervention, and by investigating comprehensive physiological measures such as MRI and DEXA and psychosocial measures. To our knowledge, no intervention studies have been reported regarding individuals with T2D and pre-diabetes in which the effect of diet and exercise on sleep– including abdominal fat, fitness, psychosocial and CVD risk factors such as age, sex, a change in fitness, HbA1c, and lipid, status of T2D or pre-diabetes– were examined together. Our subjects were also monitored by an exercise physiologist and worked with experienced dieticians, which increased adherence to the intervention among those subjects that completed the study. To date, the current study is the first to investigate the effect of lifestyle intervention on sleep disturbances targeting a unique population with high risk of sleep disturbances and CVD.

In conclusion, we demonstrated that a 6- month lifestyle intervention targeting weight loss with diet alone or diet plus exercise resulted in sleep improvement among obese/overweight individuals with T2D or pre-diabetes. In particular, abdominal subcutaneous fat reduction and improvement in depressive symptoms were the strongest correlates of reduced sleep disturbances. Future randomized controlled trials should continue to target this group and both physiological and psychological changes should be considered to enhance our understanding of lifestyle modification for sleep and, in turn to reduce the burden of CVD and diabetes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Baseline sample characteristics

Characteristics	Mean (SD)		p-value	Total (n=77)
	Diet alone (n=38),	Combined (n=39)		
Age, years	56.37 (7.17)	53.26 (8.17)	.08	54.79 (7.80)
Female	30 (78.9 %)	30 (76.9 %)	.83	60 (77.9%)
Race			.60	
White	24 (63.2%)	26 (66.7%)		50 (64.9%)
African American	13 (34.2 %)	13 (33.3 %)		26 (33.8 %)
Asian	1 (2.6 %)	0 (0 %)		1 (1.3 %)
Type 2 diabetes	15 (39.5%)	22 (56.4%)	.14	37 (48.1%)
Pre-diabetes	23 (60.5%)	17 (43.6%)		40 (51.9%)
BMI, kg/m^2	34.11 (4.49)	34.77 (5.02)	.54	34.44 (4.75)
HbA1c, %	5.76 (0.68)	5.89 (0.89)	.47	5.83 (0.79)
LDL, mg/dl	108.61 (28.24)	99.86 (32.07)	.23	104.3 (30.29)
HDL, mg/dl	52.92 (5.96)	52.26 (19.17)	.87	52.59 (17.52)
VLDL, mg/dl	24.72 (11.37)	27.60 (15.54)	.38	26.14 (13.57)
Triglyceride, mg/dl	119.53 (57.76)	141.57 (103.73)	.26	130.4 (83.82)
Total cholesterol, mg/dl	185.08 (32.69)	179.21 (41.71)	.50	182.14 (37.34)
Waist circumference, cm	104.87 (10.69)	105.41 (11.14)	.83	105.14 (10.85)
Total abdominal fat (MRI), cm^2	714.38 (141.34)	709.74 (147.25)	.90	711.87 (143.41)
Abdominal subcutaneous fat (MRI), cm^2	503.41 (131.69)	499.62 (131.49)	.91	501.37 (130.53)
Abdominal visceral fat (MRI), cm^2	169.31 (62.89)	175.15 (79.61)	.75	172.46 (71.89)
Peak oxygen uptake, ml O_2/kg/min	23.38 (4.08)	24.26 (5.09)		23.83 (4.61)
Systolic blood pressure, mmHg	125.95 (13.68)	127.51 (15.07)	.64	126.74 (14.33)
Diastolic blood pressure, mmHg	72.58 (8.38)	73.38 (8.35)	.67	72.99 (8.32)
Duration of sleep during work days, hours	7.24 (.93)	6.92 (1.04)	.16	7.08 (.99)
Sleep composite scores	15.84 (9.49)	18.47 (9.12)	.25	17.25 (9.32)
Depression	4.92 (5.74)	5.46 (8.78)	.75	5.19 (7.40)
SF-36, mental component summary	54.11 (9.36)	52.87 (10.11)	.58	53.48 (9.70)
SF-36, physical component summary	46.58 (8.88)	46.90 (8.68)	.87	46.74 (8.72)

Abbreviations: SD, standard deviation; BMI, body mass index; MRI, magnetic resonance image; LDL, low-density lipoprotein; HDL, high-density lipoprotein; HbA1c, glycosylated hemoglobin