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Association of Leisure Physical Activity and Sleep with Cardiovascular Risk Factors in Postmenopausal Women

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Abstract

Objective—To examine the individual and combined associations of leisure-time physical activity and sleep with cardiovascular risk factors in postmenopausal women.

Methods—We analyzed cross-sectional 48-month follow-up data from 393 participants of the Woman on the Move through Activity and Nutrition (WOMAN) Study, a behavioral weight loss trial. Leisure-time physical activity data were collected with the past year Modifiable Activity Questionnaire; sleep data were collected with the Pittsburgh Sleep Quality Index. We compared physical activity and sleep categories using ANOVA, post hoc Scheffe tests, and multivariate analyses based on groups above/below median leisure-time physical activity level, above/below below sleep quality value = 5, and above/below sleep duration of 7 hrs/day.

Results—The average sleep quality and sleep duration did not significantly differ between women with high and low physical activity levels. When comparing women with good sleep quality, higher physical activity levels were associated lower BMI (2.0 kg/m^2; 0.3, 3.6), waist circumference (6.3 cm; 1.7, 10.9), and total body fat (2.1 %; 0.3, 4.0) ($p<0.05$). When comparing participants with poor sleep quality, highly active women had lower trunk fat, total body fat, and insulin levels than less active women ($p<0.05$). In multivariate analysis, physical activity was significantly associated with HDL, trunk fat, and total body fat after controlling for sleep quality, sleep duration, age, hormone therapy, smoking, and BMI.

Conclusions—The combined associations of leisure-time physical activity and sleep suggest that cardiovascular risk factors were more favorable in highly active women relative to less active women regardless of sleep.

Keywords

Physical activity; sleep; cardiovascular risk factors; menopause

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INTRODUCTION

While the deleterious effects of inadequate physical activity (both direct and indirect through obesity) on cardiovascular risk factors (CVRF) are well known, current research shows that sleep may also play an important role in cardiovascular health.[1,2,3,4,5] Women progressing through menopause are more likely to experience changes in weight and abdominal adiposity which increase their risk for developing cardiovascular disease and diabetes.[6,7] Inadequate physical activity levels and sleep disruptions are among the lifestyle factors contributing to obesity in this population.

In women, a U-shaped distribution between sleep duration and hypertension, dyslipidemia, and diabetes has been documented, such that women with very short and very long sleep durations have poorer CVRF profiles.[8,9,10,11,12,13,14] However, relatively less is known regarding the role of sleep quality and cardiovascular risk outcomes. There is some data to suggest that poor sleep quality is also associated with CVRF and incidence of diabetes in adults.[14,15,16,17] While it is unclear if menopausal status is a predictor of poor sleep in women, stress, sleep disordered breathing, hormone therapy status, vasomotor symptoms, and race have been associated with poor sleep in postmenopausal women [18,19,20] Therefore, it is critical to evaluate the role of poor sleep quality on CVRF in this population.

It is important for clinicians to understand the relative importance of leisure-time physical activity (LTPA) and sleep when targeting lifestyle modifications to improve cardiovascular health in postmenopausal women. The purpose of this analysis is to examine the independent and combined associations of LTPA and sleep on CVRF. We hypothesized that high levels of LTPA would have favorable associations with CVRF, but that these favorable effects might be attenuated by poor sleep quality. We also hypothesized that the most significant differences in CVRF would be between the high LTPA/good sleep categories and the low LTPA/poor sleep categories, with intermediate results in the high LTPA/poor sleep and low LTPA/good sleep categories.

METHODS

Study population

The Women on the Move through Activity and Nutrition (WOMAN) Study is a five year randomized clinical trial of 508 post menopausal women designed to test whether an intensive nonpharmacological lifestyle intervention will reduce measures of CVRF. The WOMAN Study design including description of groups, population, and eligibility criteria have been previously reported.[21,22] Briefly, subjects were recruited from April 2002 through October 2003 through direct mailing in Allegheny County, Pennsylvania. Participants were block randomized to a Health Education (HE) group or a Lifestyle Change (LC) group. The LC group aimed for a 10% loss of initial body weight through 150 minutes per week of moderate physical activity and a 1300–1500 kcal per day, low fatdiet. Sleep habits were not targeted in either the LC or HE group. Data were collected at baseline, 6, 18, 30, and 48 months; however, all measures relevant to these analyses were completed at the 48 month follow-up, as this was the only WOMAN assessment that included sleep measures. The LC and HE groups were pooled for this analysis due to the similarity in sleep and LTPA in these groups at 48 months after the formal intervention ended at 36 months.

To be eligible for the WOMAN Study, participants had to have a waist circumference of ≥80 cm, LDL-c between 100–160 mg/dL, BMI 25–40 kg/m^2, and blood pressure < 140/90 mmHg at randomization. Eligibility criteria also included no history of cancer in the past two years, no use of cholesterol lowering medications, no diagnosis of diabetes, a Beck

Depression Inventory (BDI) score of <20, no treatment for depression, and no physical limitations that would preclude walking.

All participants provided written informed consent and this study was approved by the Institutional Review Board (IRB) of the University of Pittsburgh.

Physical Activity Measures

Physical activity was collected through the Modifiable Activity Questionnaire (MAQ), an interviewer-administered questionnaire, which assess reported leisure and occupational activities over the past year.[23] Due to the limited reported occupational activity in the WOMAN study population, only the LTPA estimate is reported.[24] LTPA was calculated as the product of the duration and frequency of 39 common activities (in hours per week), weighted by a standardized estimate of the metabolic equivalent (MET) of that activity and summed for all activities performed.[25] Data were expressed as metabolic equivalent hours per week ($MET \cdot hr \cdot wk^{-1}$). The MAQ has been previously shown to be a reliable and valid estimate of self-reported physical activity.[23,26,27] High versus low LTPA was defined as above or below the median LTPA (11.8 $MET \cdot h \cdot wk^{-1}$) reported at 48 months follow-up. Assuming that brisk walking is a 4 MET level activity, 11.8 $MET \cdot h \cdot wk^{-1}$ is equivalent to 177 minutes per week of moderate LTPA. This division was used for univariate analyses and combined LTPA/sleep groups.

Sleep Measures

Sleep quality was assessed at 48 months only through the Pittsburgh Sleep Quality Index (PSQI).[28] This questionnaire addresses seven self reported components of sleep including sleep quality, sleep duration, sleep latency, sleep disturbance, use of sleep medication, daytime dysfunction, and habitual sleep efficiency. The sleep quality variable from the PSQI questionnaire is a global score ranging from 0 to 21 generated from a summation of the seven components. A higher score indicates worse sleep quality, with a score > 5 suggesting significant sleep disturbance.[28] In this analysis, poor sleep quality was defined as a PSQI score > 5 and good sleep quality as a PSQI score ≤ 5. Seven hrs/night is generally considered an average sleep duration in the literature and the National Sleep Foundation recommends that adults sleep 7–9 hours/night.[8,9,10,11,12,13,14,29,30,31,32] In this analysis, longer sleep duration was defined as ≥ 7 hours per night and shorter sleep duration was defined as < 7 hours per night. We could not further subdivide women into a very long sleep duration category (>8–9 hrs) because only 17 women in this study had sleep durations over 8 hours. The PSQI division of 5 and sleep duration division of 7 hours were used for both univariate analyses and combined LTPA/sleep groups.

HT status and Medications

HT status was obtained through self-report in addition to a medication inventory that was completed at the 48 month visit.

Body Composition and CVRF Measures

All WOMAN study data, including body composition measurements, weight, and blood samples were taken at the University of Pittsburgh's Health Studies Clinic. BMI was calculated from height and weight measured with a stadiometer and calibrated balance beam scale. Waist circumference was measured along the horizontal plane at the center of the naval using a fiberglass retractable tape measure. Blood samples were collected following a 12 hour fast and analyzed for cholesterol, triglycerides, HDL, insulin, glucose. Body composition data was collected through dual energy X-ray absorptiometry (DXA). Total body DXA scan was performed using the pencil beam technology (QDR 1500, Hologic,

Waltham, MA, USA; enhanced whole body, software version 5.71). A standard soft tissue examination includes total body and regional measurements of trunk, arms and legs to analyze body composition according to a three-compartment model (fat mass, lean tissue and bone mineral content). In these analyses, we focus on total and trunk fat mass.

Statistical Analysis

In univariate analyses, CVRF were compared between categories of LTPA, sleep quality, and sleep duration based on groups above/below median LTPA level, above/below below PSQI value = 5, and above/below sleep duration of 7 hrs/day. We also examined Spearman correlations between CVRF and sleep quality. Normally distributed data is presented as mean with standard deviation and was analyzed using ANOVA and t-tests (continuous variables) and chi-square tests (categorical variables). Non-normally distributed variables are presented as medians with interquartile ranges and were analyzed using Mann-Whitney tests. For multivariate analyses, Scheffe post hoc tests, and linear trends tests, non-normally distributed data was transformed by taking the square root of these variables. CVRF were compared among four Leisure Physical Activity (LTPA) and sleep quality/duration categories (High LTPA/Good Sleep Quality (Longer Duration), High LTPA/Poor Sleep (Shorter Duration), Low LTPA/Good Sleep (Longer Duration), and Low LTPA/Poor Sleep (Shorter Duration)) using ANOVA with Scheffe post hoc tests and linear trend tests. For multivariate analysis, CVRF were the dependent variables and LTPA and either sleep duration or quality were the primary independent variables of interest (Model 1). Sleep quality and duration data were analyzed in separate models due to the significant correlation (r=0.6) between these two variables. Additional models controlled for age, hormone therapy (HT) status, and smoking status, either without (Model 2) and with (Model 3) BMI. Exploratory analyses examined the LC and HE groups separately (univariate analyses only). Statistical significance was considered if $p< 0.05$. All analyzes were conducted using Statistical Analysis Software, version 9.2 (SAS Institute, Inc., Cary, North Carolina).

RESULTS

Demographics/Medications

This analysis includes 393 participants from the WOMAN study at the 48 month follow-up visit. Although 454 participants remained active in the study at 48 months, we excluded 53 participants who were missing sleep data and 8 participants who were missing physical activity data. There were no significant differences in age, BMI, LTPA levels, race, sleep duration, sleep quality, HT use, hypertensive medications, diabetes medications, lipid lowering medications, caffeine, and smoking between included and excluded participants (data not shown). Women in the LC and HE groups were pooled for this analysis, and there were no significant differences in sleep or LTPA between these two groups (data not shown).

At 48 months, participants included in these analyses had an average age of 62. ± 3.0 years, BMI of 30. ± 3.0 kg/m^2, sleep duration of 6.7 ± 1.2 hours, sleep quality score of 6.3 ± 3.7, and median LTPA 12 (5.2, 21.) $MET{\cdot}hr{\cdot}wk^{-1}$. Participants were 10.% were black, 2.6% current smokers, and 9.4% consumed > 3 cups of coffee per day. Among the included women, 19% were on HT, 1.5% took diabetes medications, 14.% took lipid lowering medications, and 34.% took hypertensive medications. There were no significant differences in age, race, HT, diabetes medications, hypertensive medications, lipid lowering medications, smoking status, and coffee consumption across categories of sleep and LTPA, with the exception that there were more black women in the poor sleep quality/duration categories ($p < 0.05$; data not shown).

Univariate Analyses

Sleep, LTPA, and Hormone Replacement Therapy (HT)—The average PSQI score (6.2 ± 3.6) and sleep duration (6.7 ± 1.1 hours) of women with high LTPA did not differ significantly from the average PSQI score (6.4 ± 3.9) and sleep duration (6.8 ± 1.2 hours) of women with low LTPA. At baseline, the PSQI score (6.4 ± 3.8) and sleep duration (6.7 ± 1.2 hours) of women on HT did not significantly differ from the PSQI score (6.2 ± 3.7) and sleep duration (6.8 ±1.1 hours) of women not on HT. Similarly at 48 months, the PSQI score (5.7 ± 3.3) and sleep duration (6.8 ± 1.1 hours) of women on HT did not significantly differ from the PSQI score (6.4 ± 3.8) and sleep duration (6.7 ± 1.2 hours) of women not on HT.

LTPA and CVRF—Highly active women had significantly lower BMI, waist circumference, trunk fat, total body fat, insulin, triglycerides, glucose, and higher HDL than less active women (all $p<0.05$, data not shown).

Sleep and CVRF—CVRF did not differ significantly between women with poor and good sleep quality (data not shown), nor where there any significant correlations between CVRF and sleep quality expressed as a continuous variable (data not shown). Women with shorter sleep durations had significantly lower waist circumference (99 ± 12 cm), trunk fat (40 ± 5.5 %), and total body fat (41 ± 4.5 %) than women with longer sleep durations (waist circumference 100 ± 11 cm; trunk fat 42 ± 5.4 %; total body fat 42 ± 4.7 %) (all $p<0.05$).

Combined Analyses

LTPA, Sleep quality, and CVRF—Table 1 shows comparisons of CVRF among LTPA and sleep quality categories for the pooled LC and HE groups. Sleep quality was not associated with significant differences in CVRF in women of the same LTPA category. Women in the high active group had more favorable BMI, waist circumference, blood pressure, LDL, HDL, triglycerides, total body fat, trunk fat, insulin, and glucose than women in the low active group regardless of sleep quality. When comparing women with good sleep quality, high vs. low LTPA was associated with lower BMI (2.0 kg/m^2 ; 0.3, 3.6), waist circumference (6.3 cm; 1.7, 11), and total body fat (2.1 %; 0.2, 4.0) ($p<0.05$). When comparing participants with poor sleep quality, women in the high active group had lower trunk fat, total body fat, and insulin levels than women in the low active group ($p<0.05$). In addition, women in the high active group with poor sleep quality had lower BMI, waist circumference, trunk fat, total body fat, and insulin and higher glucose than less active women with good sleep quality ($p <0.05$). Women in the high active group with good sleep quality also had lower total body fat than less active women with poor sleep quality ($p <0.05$).

When examining the categories in the order shown in Table 1 (high LTPA/good sleep quality, high LTPA/poor sleep quality, low LTPA/good sleep quality, low LTPA/poor sleep quality), there was a significant linear trend in the expected direction for BMI, waist circumference, trunk fat, total body fat, triglycerides, insulin, and HDL.

LTPA, Sleep Duration, and CVRF—The associations of LTPA and sleep duration with CVRF were similar to the associations of LTPA and sleep quality with CVRF (results not shown). A notable exception is that women in the high active group with shorter sleep duration did not have lower BMI than low active women with longer sleep duration (results not shown).

Multivariate Analyses

In multivariate analyses, LTPA was independently associated with BMI, waist circumference, HDL, systolic blood pressure, trunk fat, total body fat, insulin, and

triglycerides after controlling for sleep quality (Table 2, p <0.05). When controlling for sleep quality, age, smoking, and HT, the relationships between LTPA and CVRF all remained significant (Table 2, p<0.05). When controlling for BMI in addition to sleep quality, age, smoking, and HT, the relationships between LTPA, trunk fat, total body fat, and HDL remained significant (Table 2, p<0.05). The associations of LTPA with CVRF were identical to the above results in the multivariate analyses with LTPA and sleep duration (results not shown, p<0.05).

Sleep quality was significantly related to glucose after controlling for LTPA, age, smoking, HT, and BMI (Table 2, p<0.05). Sleep duration was significantly related to waist circumference, trunk fat, and total body fat when controlling for LTPA, age, smoking, and HT (results not shown, p<0.05). The relationships between sleep duration, trunk fat, and total body fat remained significant when controlling for LTPA, age, smoking, HT, and BMI (results not shown, p<0.05).

Results by Intervention Group

In the combined LTPA/sleep analyses of the divided HE and LC groups, high LTPA generally was associated with more favorable CVRF than low LTPA. Variations in CVRF with sleep quality/duration were similar to trends in the pooled population analysis, although the results generally were not statistically significant (data not shown).

DISCUSSION

Our analysis of the combined associations of LTPA and sleep on CVRF suggest that LTPA is more strongly associated with favorable CVRF profiles than sleep in this population of post menopausal women. Almost without exception, high active women had better body composition and CVRF profiles than low active women regardless of sleep quality or duration. There were no associations of sleep quality or duration with CVRF in women of the same LTPA category. With the exception of total body fat, we did not see significant differences between the high LTPA/good sleep and the low LTPA/poor sleep categories. We expected most to least favorable differences in CVRF to follow the categories in the following order: high LTPA/good sleep, high LTPA/poor sleep, low LTPA/good sleep, and low LTPA/poor sleep. Linear trend tests showed trends in the expected direction across LTPA/sleep groups for BMI, waist circumference, trunk fat, total body fat, insulin, and HDL.

LTPA and CVRF

Our results are consistent with literature showing beneficial associations of high LTPA levels with CVRF. The overall results of the WOMAN study showed an increase in physical activity levels in women in the LC group when compared to the HE group at both 18- and 30-month follow-up visits, and corresponding improvements in several CVRF over the same time frame.[33] We expected physical activity to be associated with lower BMI, body fat, blood pressure, lipids, glucose, and insulin in postmenopausal women.[3,4,5,6,7] The beneficial associations of LTPA with CVRF are likely related to lower abdominal adiposity and BMI. In univariate analyses, women in the high active group had significantly lower BMI, waist circumference, trunk fat, total body fat, insulin, triglycerides, glucose, and higher HDL than less active women. In multivariate analysis, we found that LTPA was significantly related to BMI, waist circumference, HDL, systolic blood pressure, trunk fat, total body fat, and insulin after controlling for sleep quality or duration. However, only the relationships between LTPA and HDL, trunk fat, and total body fat were significant after controlling for sleep quality, sleep duration, age, smoking, HT, and BMI. Physical activity may have the greatest associations with changes in blood pressure and lipids in women with high blood

pressure or lipid levels.[4,34,35] It is possible that fewer differences were seen in CVRF with LTPA than expected because our analysis included a population of relatively healthy women. It has also been hypothesized that frequent and higher volume exercise decrease pro-inflammatory markers (IL-1, IL-6, CRP) which contribute to increased risk of cardiovascular disease, and this relationship has been shown in postmenopausal women.[36,37] Inflammatory marker data were not available for this analysis.

Sleep and CVRF

We expected both long and short sleep duration and poor sleep quality to be associated with hypertension, dyslipidemia, and diabetes in women.[8,9,10,11,12,13,14,15,16,17] We examined both sleep quality and sleep duration in this analysis for a more complete picture of the impact of sleep on CVRF. Our average sleep duration and PSQI values are comparable to other sleep studies in postmenopausal women.[38] In univariate analyses, CVRF did not differ significantly between women with poor and good sleep quality. We expected women with short sleep duration to have less favorable CVRF than women with longer sleep duration, but found that women with shorter sleep durations had significantly lower waist circumference, trunk fat, and total body fat than women with longer sleep duration. In multivariate analyses, we did not see any significant relationships between sleep quality and CVRF, with the exception that sleep quality was significantly related to glucose after controlling for age, smoking status, hormone replacement status, and BMI. Sleep duration was significantly related to waist circumference, trunk fat, and total body fat, and the relationships between sleep duration, trunk fat, and total body fat remained significant when controlling for age, smoking, HT, and BMI. Again, it is possible that more significant differences in CVRF were not seen in this analysis because the participants were relatively healthy.

Poor sleep may contribute to increased CVRF through numerous mechanisms. Similar to LTPA, it is likely that sleep mediates CVRF through BMI. The relationship between sleep duration and BMI has been shown to be curvilinear in adults, with the longest and shortest sleep durations associated with the greatest BMI.[39] Sleep deprivation may increase blood pressure through sympathetic activation and changes in baroreflex sensitivity.[40] Obstructive sleep apnea (OSA) is well established as a link between sleep, obesity, and CVRF. Sleep disordered breathing and sleep deprivation may lead to increases in pro-inflammatory cytokines (IL-1, IL-6, TNF-α, CRP), slow glucose metabolism, impair insulin sensitivity, and cause elevations in stress hormones such as cortisol which increase the risk for cardiovascular disease.[40,41,42] In this analysis, PSQI did not include polysomnography data needed to make a diagnosis of OSA.

Combined LTPA/Sleep and CVRF

It does not seem from our analysis that either poor sleep duration or quality attenuates the beneficial associations of LTPA. The beneficial associations of LTPA with BMI and waist circumference were only significant in women with good sleep duration or quality. The beneficial associations of LTPA with trunk fat and insulin were only significant in women with poor sleep duration or quality. The results do suggest, however, that good sleep quality or duration does not compensate for low levels of LTPA. We found that women in the high LTPA/poor sleep categories had more favorable waist circumference, trunk fat, and total body fat than women in the low LTPA/good sleep categories.

Many hypotheses have been made on the physiological links between sleep and LTPA and their effects on CVRF. Poor sleep and exercise are associated with increases and decreases in pro-inflammatory markers, respectively.[16,29,36,40,41] Both poor sleep and exercise may lead to hunger through increases in ghrelin and decreases in leptin.[30,40] Physical activity

may affect sleep through changes in thermoregulation and circadian rhythms.[43] Moderate intensity exercise may promote better sleep quality and duration in adults.[44,45] Strenuous activity, however, can disrupt sleep through hormones that induce wakefulness (IL-6) and stress response (cortisol) which can take several hours to return to pre-exercise levels.[45] In turn, disturbed sleep can lead to fatigue and less energy to exercise during the day.

Limited information in the literature is available on the combined effects of sleep and physical activity. Our conclusion that LTPA is more strongly associated with CVRF than sleep is consistent with one lifestyle intervention study of middle aged adults where the diet and exercise intervention resulted in similar improvements in body weight and insulin sensitivity regardless of sleep duration.[29] In another study of an exercise intervention program in postmenopausal women, exercise induced weight loss was greatest among participants with the lowest sleep duration or poor sleep quality.[30] Similar to our analysis, the favorable associations of LTPA were greater in participants with poor sleep duration or quality for certain CVRF. Further research is needed to clarify if sleep influences the beneficial associations of physical activity, across a continuum of intensity levels, with CVRF.

Few studies exist that have examined the longitudinal relationships between sleep, LTPA, and CVRF. In one longitudinal study of postmenopausal women over 32 years, sleep disturbance was associated with obesity at midlife but did not affect future diabetes incidence.[46] Weight gain did not differ over time between women with and without sleep disturbance. The Nurse's Health Study followed 68,183 women of ages 39–65 years over 16 years.[47] The study found a weight gain of 1.14 kg in women sleeping less than 5 hours relative to women sleeping 7 hours that was independent from physical activity. In another study of adults, short sleep duration but not physical inactivity were associated with significant gains in weight over 6 years.[48] It is unclear if there is a relationship between sleep duration or quality and future weight gain or CVRF in postmenopausal women. More longitudinal research is needed to explain these relationships.

Strengths/Limitations

The main strength of our study is the variety and quality of data analyzed. We were able to compare many CVRF and measures of body composition. Body composition data was available from basic measurements (waist circumference) and DXA, giving us an accurate assessment of fat distribution in the study population. Comparisons of sleep quality and leisure physical activity data are often complicated by differing questionnaires of sleep quality. We used well established and widely utilized questionnaires (MAQ and PSQI) to gather information about physical activity and sleep that can be compared to analyses from existing and future studies.

Several limitations of our analysis should be acknowledged. We did not have longitudinal sleep data, and we cannot address causality or the longitudinal consequences of low LTPA and poor sleep in this cross-sectional analysis. There is also the potential in cross-sectional studies such as ours to introduce bias; for example, if women with poor sleep started exercising in an attempt to improve their sleep, then our study could underestimate benefits of exercise on sleep. Sleep quality and duration data in this analysis were self reported, and OSA in our population is unknown. Additionally, there were significantly more black women in the poor sleep groups in our analysis, and postmenopausal black women may have shorter sleep durations and more sleep disturbance due to sleep disordered breathing than postmenopausal white women.[31] The overall number of black women in this analysis was too low to examine this group separately. It is possible that unmeasured variables, such as dietary measures, may be confounders and explain some of the relationships between LTPA, sleep, and CVRF. Lastly, although our overall sample size (n=393) is sizable, the

number of women in the cross-categories is smaller, which could also possibly explain some of our null findings.

Conclusion

In conclusion, this combined analysis of LTPA and sleep may suggest that LTPA is a more important lifestyle modification than sleep to improve CVRF in postmenopausal women. CVRF were more favorable in women with high LTPA relative to low LTPA regardless of sleep quality or duration almost without exception. Further research of combined LTPA and sleep data is needed to clarify if sleep influences the beneficial effects of LTPA in postmenopausal women.

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Table 1

CVRF by LTPA/Sleep Quality Categories (n=393)

	High LTPA/Good Sleep Quality (N=99)	High LTPA/Poor Sleep Quality (N=98)	Low LTPA/Good Sleep Quality (N=90)	Low LTPA/Poor Sleep Quality (N=106)	P value
BMI (kg/m^2)	29.3 ± 3.6	29.5 ± 4.0	31.3 ± 4.7	30.9 ± 4.5	0.001^{ab*}
Waist Circumference (cm)	97.8 ± 10.3	97.5 ± 11.0	103.8 ± 12.2	101.3 ± 11.6	0.0002^{ab*}
Systolic Blood Pressure (mmHg)	122.7 ± 15.9	122.4 ± 14.1	123.9 ± 12.9	125.7 ± 15.1	0.3
Diastolic Blood Pressure (mmHg)	76.3 ± 7.0	76.0 ± 8.0	76.4 ± 8.4	77.1 ± 8.6	0.8
LDL (mg/dl)	125.2 ± 27.7	126.6 ± 28.2	126.9 ± 31.4	130.5 ± 31.20	0.6
Cholesterol (mg/dl)	213.1 ± 28.6	217.0 ± 32.1	213.2 ± 37.5	219.0 ± 34.0	0.5
HDL (mg/dl)	63.0 ± 14.5	66.6 ± 16.9	60.3 ± 16.0	60.6 ± 14.4	0.02*
Trunk Fat (%)	40.4 ± 5.8	39.7 ± 6.0	42.6 ± 4.9	42.2 ± 4.6	0.0005^{bc*}
Total Body Fat (%)	40.7 ± 4.7	40.5 ± 5.2	42.8 ± 4.4	42.8 ± 3.9	0.0001^{abcd*}
Insulin (mg/dl)	12.4 (8.6, 16.4)	11.5 (9.2, 14.10)	13.0 (10.7, 17.5)	13.5 (11.0, 18.3)	0.0009^{bc}
Glucose (mg/dl)	103.0 (94.0, 108.0)	104.0 (96.0, 114.0)	101.0 (94.0, 106.0)	102.0 (96.0, 109.0)	0.02^{b}
Triglycerides (mg/dl)	110.0 (90.0, 156.0)	112.0 (78.0, 145.0)	124.0 (91.0, 155.0)	142.0 (89.0, 173.0)	0.09*

Note: Data are means (standard deviations), with the exception of insulin, glucose and triglycerides which are presented as median (25, 75 quartiles). P-value reflects ANOVA for the difference in means, with the exception insulin, glucose and triglycerides of which are determined by Wilcoxan rank sum test.

*Significant linear trend.

For Scheffe post hoc tests:

^{a}Significant difference between High LTPA/Good Sleep Quality and Low LTPA/Good Sleep Quality

^{b}Significant difference between High LTPA/Poor Sleep Quality and Low LTPA/Good Sleep Quality

^{c}Significant difference between High LTPA/Poor Sleep Quality and Low LTPA/Poor Sleep Quality

^{d}Significant difference between High LTPA/Good Sleep Quality and Low LTPA/Poor Sleep Quality

Table 2

Multivariate Analysis of CVRF, LTPA and Sleep Quality

	Model 1: LTPA and sleep quality		Model 2: LTPA, sleep quality, HT, age, and smoking		Model 3: LTPA, sleep quality, HT, age, smoking, and BMI	
	LTPA	Sleep Quality	LTPA	Sleep Quality	LTPA	Sleep Quality
BMI (kg/m^2)	−0.58[f]	0.0097	−0.58[f]	0.0044	---	---
Waist Circumference (cm)	−1.3[f]	−0.06	−1.4[f]	−0.037	−0.15	−0.046
Systolic Blood Pressure (mmHg)	−0.83[h]	0.14	−0.9[h]	0.18	−0.51	0.18
Diastolic Blood Pressure (mmHg)	−0.39	0.034	−0.39	0.026	−0.18	0.024
LDL (mg/dl)	−0.61	0.53	−0.51	0.52	−0.43	0.52
Cholesterol (mg/dl)	0.047	0.86	0.12	0.85	0.14	0.85
HDL (mg/dl)	1.4[g]	0.19	1.4[g]	0.16	0.95[h]	0.17
Trunk Fat (%)	−0.77[f]	−0.0028	−0.78[f]	0.0096	−0.34[g]	0.0063
Total Body Fat (%)	−0.68[f]	0.026	−0.69[f]	0.032	−0.35[g]	0.03
Insulin (mg/dl)	−0.083[f]	−0.00053	−0.084[f]	−0.00064	−0.034	−0.001
Glucose (mg/dl)	−0.032	−0.018	−0.031	−0.018	−0.006	−0.019[h]
Triglycerides (mg/dl)	−0.15[h]	0.038	−0.16[h]	0.045	−0.08	0.044

Values in columns represent beta coefficients from linear regression models.

1: LTPA and sleep quality; 2: LTPA, sleep quality, HT, age, and smoking; 3: LTPA, sleep quality, HT, age, smoking, and BMI

[f] $p < 0.001$;

[g] $p < 0.01$;

[h] $p < 0.05$