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Physical activity, and not fat mass is a primary predictor of circadian parameters in young men

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Abstract

Circadian rhythms are ≈ 24 h oscillations in physiology and behavior, and disruptions have been shown to have negative effects on health. Wrist skin temperature has been used by several groups as a valid method of assessing circadian rhythms in humans. We tested the hypothesis that circadian temperature amplitude (TempAmp) and stability (TempStab) would significantly differ among groups of healthy young men of varying adiposities, and that we could identify physiological and behavioral measures that were significantly associated with these temperature parameters. Wrist skin temperatures taken at 10 min intervals for 7 consecutive days were determined in 18 optimal (OGroup), 20 fair (FGroup) and 21 poor (PGroup) %Fat grouped young men and subsequently analyzed using available validated software. Body composition, cardiorespiratory fitness, actigraphy, daily nutritional and sleep data, and fasting lipid, insulin and glucose concentration measures were also determined. Significant changes in TempAmp and TempStab parameters in subjects with a single metabolic syndrome (MetS) risk factor compared

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DECLARATION OF INTEREST

The authors have no conflicts of interest to report.

to those with no MetS factors was observed. In addition, stepwise multivariate regression analyses showed that 50% of the variance in TempAmp was explained by actigraphy (mean steps taken per day; MSTPD), cardiorespiratory fitness, and late night eating per week (#LNE); and 57% in TempStab by MSTPD, time spent in moderate-to-vigorous activity per day, fat mass, and #LNE. Overwhelmingly, physical activity was the most important measure associated with the differences in circadian rhythm parameters. Further research is warranted to determine the effects of increasing the amount and timing of physical activity on the status of the circadian system in a variety of populations.

Keywords

Circadian rhythm; temperature amplitude; temperature stability

INTRODUCTION

The circadian system of the human body produces approximate 24-hour oscillations of several physiological variables (Duguay & Cermakian, 2009; Refinetti & Menaker, 1992; Sarabia et al., 2008). Regulation of these circadian oscillations is organized by a hierarchical system with the central clock located within the suprachiasmatic nucleus (SCN) located in the hypothalamus. The SCN, known as the central pacemaker, is synchronized to light/dark cycles and functions to coordinate many physiological processes through neurohumoral signaling to peripheral tissues. The molecular clock mechanism, that underlies the 24 h timekeeping, has been identified in virtually all tissues and cell types throughout the body, including heart, lung, kidney, pancreas, muscle, and adipose tissue (Asher & Schibler, 2011; Barclay et al., 2012; Damiola et al., 2000; Duguay & Cermakian, 2009; Froy, 2010; Patton & Mistlberger, 2013).

In addition to regulation by neurohumoral signals from the SCN, the circadian oscillations of the molecular clock mechanism in peripheral tissues can also be influenced by behavioral signals. The light/dark cycle is the most dominant zeitgeber (time cue), but time of feeding and physical activity have also been shown to have the ability to alter peripheral clock gene expression (Ando et al., 2005; Damiola et al., 2000; Patton & Mistlberger, 2013). Many studies have shown that altering metabolic, endocrine, and homeostatic events can change the expression of circadian clock genes in the peripheral tissue with no accompanied change in the SCN (Ando et al., 2005; Froy, 2010; Kohsaka et al., 2007; Mendoza, 2007). This may result in a desynchronization or misalignment of the central and peripheral clocks with associated disruption in organ and system circadian rhythms. Studies have demonstrated that forced desynchrony in humans and rodents is associated with significant adverse changes in risk factors for cardiometabolic disease (Barclay et al., 2012; Carvas et al., 2012; Froy, 2010; Garaulet & Madrid, 2009; Marcheva et al., 2010; Muller et al., 1985).

A disruption in circadian rhythms has been shown to have a negative impact on health and may lead to some of the factors associated with the Metabolic Syndrome (MetS) (Garaulet & Madrid, 2009; Staels, 2006). The MetS is a cluster of health-risk factors associated with obesity that is a strong predictor of the development of cardiovascular disease and metabolic

derangements (Alberti et al., 2009). Previously, measures of circadian rhythm were labor intensive and intrusive (Hofstra & de Weerd, 2008; Sarabia et al., 2008). However, in the last 10 years, monitoring of wrist skin temperature has been successfully used as a non-invasive, easy to obtain candidate measure of circadian parameters in a variety of healthy and diseased populations. Early studies using this approach validated it against dim light melatonin measures and found that the rise in wrist temperature occurred in anticipation of the evening increase in melatonin. Decreases in activity, changes in body position and increases in declared sleep propensity were delayed with respect to increases in dim light melatonin (Bonmati-Carrion et al., 2014; Corbalán-Tutau et al., 2011; Gompfer et al., 2010; Kripke et al., 2005; Morgan et al., 1998; Nováková et al., 2013; Sarabia et al., 2008; van Marken Lichtenbelt et al., 2006). Furthermore, wrist skin temperature measures have been reported to be inversely correlated and phase-advanced with the oscillations of core body temperature, thus making this non-intrusive measurement method a reliable candidate to examine circadian rhythmicity (Marotte & Timbal, 1981; Sarabia et al., 2008). The presence of a circadian pattern in wrist skin temperature has also been shown in individuals who were awake for 24 h under laboratory conditions, demonstrating a contribution from the central clock to this measure of rhythms (Sarabia et al., 2008). The ability to non-invasively record over longer time frames suggests wrist skin temperature is a strong candidate for the examination of the status of the circadian system.

The transition from adolescence to young adulthood is an important time to evaluate cardiovascular health in young adult males. This transition period has been shown to be a time of increased risk of developing obesity due to changes in behavioral, nutritional, and physical activity habits (Gordon-Larsen et al., 2004; Morrell et al., 2011). The purpose of this study was to determine if there were significant differences in circadian rhythms measured by noninvasive wrist skin temperature rhythm monitoring among optimal, fair, and poor percentage fat (%Fat) categorized healthy (non-diabetic, normotensive) young men. The second purpose of the study was to determine if the circadian rhythm parameters were associated with factors characterizing the MetS [including abdominal circumference, resting systolic and diastolic blood pressure, lipid profile measures [total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), triglyceride concentrations, and total cholesterol to HDL ratio], and insulin and glucose concentrations, cardiorespiratory fitness, actigraphy measures, and/or nutritional intake (frequency and timing) in a group of young men of varying adiposities.

METHODS

Subjects

A total of 59 young, healthy male subjects aged 20–34 years old were recruited for this study and studied during a 60 day time period (20 January 2014 to 20 March 2014). For recruitment purposes, the subjects were categorized by body mass index (BMI) status (22 healthy weight: BMI <25.0 kg/m²; 26 overweight: 25.0 BMI 29.9 kg/m²; 11 obese: BMI 30.0 kg/m²). All volunteers were free from any known cardiovascular disease, diabetes mellitus, pulmonary disease, and/or orthopedic limitations. None of the subjects were taking medications affecting cardiovascular function or insulin sensitivity. Prior to study initiation,

each subject provided written informed consent approved by University of Kentucky Medical Institutional Review Board which is registered Department of Health and Human Services and in compliance with the ethical standards of the journal (Portaluppi, 2010). Each subject filled out a Physical Activity Readiness-Questionnaire and Health History Form to identify any existing contraindications to the maximal graded exercise testing as specified in the American College of Sports Medicine Guidelines for Exercise Testing and Prescription (Pescatello, 2014). All resting heart rate and blood pressure, body composition, and maximal graded exercise testing were completed in a single testing session, followed by 7 consecutive days of circadian rhythm, daily questionnaires, and objective physical activity recording. Subjects were then scheduled for a second study session in the early morning following an overnight fast (minimum 8 h) to provide a resting venous blood sample.

Resting heart rate and blood pressure measurements

Resting heart rate and blood pressure was determined for each subject following a minimum of 5 min of seated rest in a thermo neutral room. Heart rate was determined using a heart rate monitoring device (Polar A3, Lake Success, NY) and blood pressure was determined by manual auscultation using an appropriate sized cuff and hand aneroid (Welch Allyn Model DS58-MCCB, Bangor, ME) and stethoscope (3M Health Care Littmann Cardiology III, St. Paul, MN) by a trained investigator.

Body composition measurements

Standing height and body weight were determined for each subject using a wall-fixed stadiometer (Seca Stadiometer Pat. No. 4694581) and a calibrated scale (Teraoka D1-10; Singapore), respectively. A total body dual energy X-ray absorptiometry (DXA) scan was performed on each subject using a Lunar iDXA (Lunar Inc., Madison, WI) densitometer. All scans were analyzed by a single-trained investigator using Lunar software version 14.10. DXA fat mass (g), fat-free mass (FFM; g), mineral-free lean mass (g), and percentage fat (%Fat) were assessed. For analyses purposes, %Fat measures were used to re-categorize the subjects into optimal (%Fat 17.9%), fair (%Fat 18.0–24.9%), and poor (%Fat 25.0) groups using previously reported cut points (Pescatello, 2014). Circumference measurements (waist, abdominal, and hip) were taken in triplicate and the mean measurement was used for analyses using a fiberglass anthropometric tape (Creative Health Products BMS-8) in accordance with the guidelines established by the Airlie Conference Proceedings for each participant (Lohman et al., 1988).

Maximal graded exercise test

Each subject performed a maximal graded exercise test (GXT; 2 min progressive speed and grade) on a treadmill using an indirect calorimetry testing system with integrated electrocardiogram (ECG) (SensorMedics Vmax Encore, CareFusion Corporation, San Diego, CA). During the tests, continuous measurements of oxygen consumption were recorded and cardiovascular movement was monitored. At the final 30 s of each stage, heart rate, blood pressure, and rating of perceived exertion (RPE) were taken and recorded. The test was terminated upon volitional fatigue. After completing the maximal GXT, five minutes of passive recovery data (heart rate and blood pressure) were taken. Each subject reached VO_2 peak ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) determined by the participant's ability to obtain a

minimum of two of the following criteria: respiratory exchange ratio >1.10, maximum heart rate $\pm$ 1SD, and/or RPE $\geq$ 17.

Circadian rhythm measures

Circadian rhythm parameters (wrist temperature amplitude and stability) were measured by noninvasive wrist temperature rhythm monitoring and recording devices (Thermochron iButton; Embedded Data Systems, Lawrenceburg, KY). Temperature amplitude and stability were determined for each subject. The stability of an individual's circadian rhythm is reflected by the variation of the oscillatory patterns of circadian data over several days and the amplitude is defined by the difference between the peak midline values (Hughes et al., 2010). The iButton was programmed to record temperatures every 10 min with a sensitivity of 0.0625 °C. Each subject wore an iButton for 7 consecutive days inside the wrist of the non-dominant hand. The subjects were given strips of latex-free dressing tape (Hypafix; Smith & Nephew Inc; Memphis, TN) and a double-sided cotton sports wrist band to maintain the iButton securely against the wrist, and allow for infrequent removal during times when the subject had direct contact with water (i.e. bathing or swimming) followed by replacement. Following the 7 days of wearing the iButton, it was returned to the research team and the information was transferred through an adapter (SK-IB-R-iButton Connectivity Kit; Embedded Data Systems, Lawrenceburg, KY) and was analyzed using a modified version of the software program (JTK_CYCLE (Hughes et al., 2010)). While wearing the iButton, the subjects were asked to record answers to daily questions concerning sleep, frequency and timing of nutritional intake, alcohol use, smoking, and removal times of the wrist skin temperature monitor.

Actigraphy measures (physical activity assessment)

All subjects were instructed to wear a New Lifestyles-1000 (NL; LeesTown, MO) medical grade physical activity monitor for 7 consecutive days coinciding with the days the wrist temperature-monitoring device was worn. The number of daily steps, miles, and time spent in moderate to vigorous activity were recorded. To avoid reactivity, all physical activity monitors were sealed using stickers after stride length, time of day, and an intensity threshold of 4 were internally programmed.

Blood sampling and analyses procedures

The subjects were asked to give a fasted venous blood sample to evaluate lipid profiles, and insulin and glucose concentrations. The subjects had their blood samples drawn in the early morning following a minimum of 8 h of fasting. The blood samples were analyzed for total cholesterol, HDL, LDL, triglyceride concentrations, total cholesterol to HDL ratio, insulin and glucose concentration, and the insulin-resistance index. The homeostasis model assessment (HOMA-IR) formula was used to determine the insulin resistance index (Ferrannini & Mari, 1998). The lipid profiles were determined using enzymatic-colorimetric methods (Roche Diagnostics Corp, Indianapolis, IN). The insulin concentrations were determined using an ELISA immunoassay (ALPCO, Salem, NH) technique with resulting intraassay, inter-assay and sample duplicate coefficient of variations (CV) of 1.4%, 1.7% and 2.6%, respectively. The glucose concentrations were determined using an automated glucose analyzer (YSI 2300 STAT Plus, YSI Life Sciences, Yellow Springs, OH) with

quality control linearity within the manufacture's specified 2% and with a sample duplication CV of 0.3%.

Statistical analysis

Data was analyzed using Statistical Package for Social Sciences (Version 22, Armonk, NY). Descriptive data are presented as means and standard deviations for all study variables. Comparisons between percent fat categories (optimal, fair, and poor) were made using one-way ANOVA; *post-hoc* two group comparisons were made to compare optimal and fair to poor when appropriate. Comparisons were made on circadian rhythm parameters (temperature amplitude and stability) measured by the noninvasive wrist temperature rhythm monitors as well as demographic and behavioral characteristics. Group means of temperature amplitude and stability were also compared based on the presence of at least one MetS or not using one-way analysis of variance. Pearson's correlation coefficients were estimated to quantify bivariate relationships between continuous variables and correction for multiple testing was not performed to preferentially favor a low probability of a Type II error for this exploratory study. To examine multiple variable relationships of circadian rhythm temperature parameters with MetS factors, physical activity, physical fitness, sleep, nutritional intake frequency and timing, multiple linear regression was used. Stepwise selection was used to aid in model selection. Results from the linear regression are presented as explained variance, unstandardized β estimates, and standardized β estimates. A two-sided significance level of 0.05 was used for all statistical tests.

RESULTS

The subject physical characteristics and behavioral factors are shown in Table 1. Fifty-nine healthy young men participated in this study. For analysis purposes, the subjects were organized into body composition groups according to %Fat cut-points defined by American College of Sports Medicine (Reiter et al., 2012). The optimal, fair, and poor %Fat Groups included 18, 20, and 21 subjects, respectively. Of the 59 subjects that participated in this study, 17 optimal %Fat; 17 fair %Fat; 18 poor %Fat subjects were willing to participate in the fasting blood sampling procedure. In addition, objective physical activity measures were not obtained from 4 (1 optimal and 3 fair %Fat grouped) subjects due to physical activity monitor malfunction or noncompliance with study procedures.

Analysis of wrist temperature has been used in the circadian assessment of human subjects in studies measuring sleep quality, blood pressure, aging, cancer therapeutics, and weight loss (Bandin et al., 2014; Blazquez et al., 2012; Corbalán-Tutau et al., 2011; Gompfer et al., 2010; Roche et al., 2014; Romeijn & Van Someren, 2011; Sarabia et al., 2008; Yoon et al., 2003). These temperature rhythms have been shown to correlate with changes in dim light melatonin onset, a standard for determining circadian phase in people (Nováková et al., 2013). Despite the aforementioned studies, assessment of wrist temperature rhythms in healthy college age men with variable sleep/wake/activity schedules has not been performed. Figure 1(A) shows a representative 7 day wrist temperature rhythm trace from a single subject. A rhythmic pattern can be observed with elevated night time temperatures (inverse to core body temperature) and lower temperature values during waking hours. Wrist

temperature continues to oscillate with a consistent pattern despite reported altered sleep/wake behavior. This is especially present on the third night when reported bed time occurs several hours after the rise in wrist temperature begins. These data demonstrate the efficacy of wrist temperature as a measure of the output of the circadian system. In addition, our study population consisted of a range of body compositions among study subjects. We did not observe any association between body fat and peak ($r = -0.05$; $p = 0.79$) or mean $r = 0.23$; $p = 0.08$) wrist temperature during night time hours (Figure 1B and C). These data demonstrate that despite varying adiposities heat was able to dissipate similarly, suggesting that fat is not serving as an insulator and thus measures of wrist temperature amplitude and stability accurately reflect status of the circadian system.

The ANOVA resulted in significant differences in temperature amplitude (Figure 2A) and stability (Figure 2B) among the %Fat groups. The poor %Fat group (0.96 ± 0.11) had significantly lower temperature amplitude than the optimal (1.46 ± 0.16) or fair (1.38 ± 0.13) %Fat groups. Similarly, the poor %Fat group (109.10 ± 14.12) had significantly lower circadian temperature stability than the optimal %Fat (166.52 ± 17.84) or fair %Fat group (175.21 ± 23.96).

Mean differences in temperature amplitude and stability were assessed between groups of subjects with at least one risk factor of the MetS (waist circumference >94 cm, triglycerides 150 mg/dL, systolic blood pressure 130 mmHg, diastolic blood pressure 85 mmHg, HDL 40 mg/dL, glucose 100 mg/dL), as defined by Alberti et al. (2009) compared to those with no risk factors of the MetS. Figure 2(C) and (D) shows that subjects with a minimum of one factor of the MetS had significantly lower temperature amplitudes and stability compared to those without any risk factors of the MetS. Figure 3(A) and (B) shows representative 7-day averaged temperature profiles for a subject from the fair adiposity and the poor adiposity groups, respectively.

Temperature amplitude was significantly correlated with several study measures. For the total group of subjects, temperature amplitude was significantly correlated with waist ($r = -0.41$), abdomen ($r = -0.41$), and hip circumferences ($r = -0.30$); mean steps ($r = 0.41$), miles ($r = 0.39$), and time spent in MVPA ($r = 0.27$) per day; VO_2 peak ($r = 0.49$); % fat ($r = -0.38$); fat mass ($r = -0.39$); fasting HDL ($r = 0.38$), ratio of total cholesterol to HDL ($r = -0.39$), triglycerides ($r = -0.24$), insulin concentration ($r = -0.29$), and insulin resistance ($r = -0.28$); and the number of MetS risk factors ($r = -0.41$). Temperature amplitude in the optimal %Fat group was significantly correlated with number of late night snacks in a week (eating after 8 pm; $r = -0.64$), and the number of the MetS risk factors ($r = -0.48$). In the fair %Fat group, temperature amplitude was significantly correlated with VO_2 peak ($r = 0.49$) and number of times skipped break-fast per week ($r = -0.47$). Temperature amplitude was significantly correlated with the mean steps ($r = 0.66$), mean miles ($r = 0.60$), and the mean time spent in MVPA ($r = 0.48$); VO_2 peak ($r = 0.46$); and fasting HDL ($r = 0.61$), total cholesterol to HDL ratio ($r = -0.60$), and LDL ($r = -0.60$) in the poor %Fat group.

There were also several study variables significantly correlating with temperature stability. Temperature stability for the total group significantly correlated with waist ($r = -0.34$), abdomen ($r = -0.34$) and hip circumferences ($r = -0.06$); mean steps ($r = 0.50$), miles ($r =$

0.50) per day; VO_2 peak ($r = 0.40$); percent fat ($r = -0.30$) and fat mass ($r = -0.33$); number of times skipped breakfast per week ($r = -0.031$); fasting HDL ($r = 0.34$), total cholesterol to HDL ratio ($r = -0.33$) and insulin concentrations ($r = -0.028$); and the number of MetS risk factors ($r = -0.36$). The stability of temperature in the optimal %Fat group was significantly correlated with mean steps ($r = 0.52$) and miles ($r = 0.52$) per day; and the number of times eating a late night snack per week ($r = -0.51$). In the fair %Fat group, temperature stability was significantly correlated with VO_2 peak ($r = 0.56$) and number of times skipped breakfast per week ($r = -0.48$). Temperature stability was significantly correlated with mean steps ($r = 0.65$), miles ($r = 0.63$), and time spent in MVPA ($r = 0.44$); and fasting HDL ($r = 0.60$), total cholesterol to HDL ratio ($r = -0.62$), and LDL ($r = -0.54$) in the poor %Fat group.

The stepwise multivariate regression analysis was statistically significant and accounted for 50% of the variance of temperature amplitude (Figure 4). The mean steps taken per day alone positively influenced temperature amplitude and resulted in an explained variance of 31%. The addition of VO_2 peak to the equation also positively influenced temperature amplitude and increased the explained variance to 41%. While the number of times late night eating occurred per week negatively impacted the temperature amplitude, the addition of this adverse behavior increased the explained variance to 50%. The stepwise multivariate regression was statistically significant and accounted for 57% of the variance in temperature stability (Figure 4). The mean steps per day alone positively influenced temperature stability and explained 42% of the variance in temperature stability. The addition of mean MVPA per day, fat mass, and the number of times late night eating occurred per week all negatively influenced temperature stability and increased the explained variance to 47%, 52%, and 57%, respectively. Table 2 further demonstrates the unstandardized and standardized collective estimate contributions of the significant variables to the models of both temperature amplitude and temperature stability. In both models, mean steps per day were positively associated with amplitude and stability even after accounting for additional variables. The standardized estimates were also observed to be the largest among the variables included in the model which suggests that changes in mean steps per day would have a larger impact on temperature and amplitude when compared to other variables in the model.

DISCUSSION

Evidence for circadian disruption persists across a variety of conditions including shift workers but is also seen across many chronic diseases (Logan & Sarkar, 2012; Sack et al., 1992; Wulff et al., 2010). In addition, there have been an increasing number of reports demonstrating associations between circadian rhythm disruptions and the development of metabolic dysfunction, disease state progression, and other deleterious health effects (Colwell & Matveyenko, 2014; Garaulet & Madrid, 2009; Knutsson & Bøggild, 2000; Reiter et al., 2012; Zelinski et al., 2014). The initial purpose of our study was to determine if circadian rhythm parameters (wrist temperature amplitude and stability) significantly differed among groups of healthy young men of varying adiposities. Similar to previous reports, we demonstrated significantly lower temperature amplitude and stability in our obese group of subjects (Corbalán-Tutau et al., 2011). We also concurred with previous

findings (Atkinson et al., 2007; Carvas et al., 2012; Colwell & Matveyenko, 2014; Garaulet & Gomez-Abellan, 2014) that several physiological and behavioral measures were significantly associated with circadian rhythm parameters when the total group, and adiposity groups were examined separately. It was interesting to note that we detected changes in circadian amplitude and stability parameters in subjects with only one MetS risk factor compared to those with no MetS factors. Results from the study of Corbalán-Tutau et al. (Corbalán-Tutau et al., 2011) showed similar findings, reporting decreased temperature amplitudes in women who had greater risk of the MetS demonstrated by higher insulin concentrations and insulin resistance indexes. Scheer et al. (2009) found many adverse metabolic changes in humans, including increased circulating glucose levels and decreased insulin sensitivity when individual circadian rhythms were disrupted. These findings suggest that disturbed circadian rhythms may contribute to, or at a minimum, precede the overt development of health derangements including diabetes and cardiovascular disease (Corbalán-Tutau et al., 2011; Marcheva et al., 2010; Scheer et al., 2009).

The results of the multivariate regression analyses of our study revealed that both physiological factors and behavioral measures explained a significant amount of the explained variance in temperature amplitude and stability. A measure of cardiorespiratory fitness (VO₂ peak) significantly contributed to the variance in temperature amplitude. To date, few studies have examined the contribution of physical fitness to circadian parameters, however there have been numerous suggestions that disruption in circadian parameters may contribute to the development of cardiovascular disease. Atkinson et al. (1993), Carvas et al. (2012), Hofstra & de Weerd (2008), Refinetti & Menaker (1992) found that individuals with higher fitness levels have higher amplitudes and lower night minimums in body temperature when compared to individuals with lower fitness levels. Interestingly, no additional measure of total body composition, estimate of regional adiposity, or fasting glucose or insulin concentration or indexes contributed to our models of temperature amplitude, and the addition of fat mass only minimally contributed to our model of temperature stability, despite their previously demonstrated relationship to the development of diabetes (Carvas et al., 2012; Colwell & Matveyenko, 2014; Ferrannini & Mari, 1998; Morrell et al., 2011). We speculate that the lack of or minimal contribution of these measures to our temperature parameter regressions models may be due to our focus on healthy adults and the exclusion of known diabetics or those taking glucose altering medications.

Two behavioral factors significantly contributed to the temperature amplitude and stability regression models of our study. These factors included the amount of physical activity (the mean steps taken per day) and the number of time late night eating occurred during the 7-consecutive day data collection period. Late night eating has long been recognized as a contributing factor in the development of obesity and elimination of this behavior has been encouraged as a weight reduction strategy. It has been previously recognized that the timing of nutritional intake can strongly influence the function of peripheral clocks independent of the central circadian clock found in the SCN, and if not properly timed can lead to circadian dysynchrony. Support for the influence of inappropriate timing of nutritional intake on the status of the circadian system has been demonstrated in both humans and mice. Corbalán-Tutau et al. (2011), Damiola et al. (2000), Froy (2010), Marcheva et al. (2010) demonstrated that restricting the feeding mice (nocturnal animals) to daylight hours resulted in an

inversion of circadian gene expression in liver, kidney, pancreas, and heart, but not in the SCN, leading to circadian dysynchrony between central and peripheral clocks. Interestingly, we found no relationship between hours of sleep per night and temperature amplitude or stability; however, circadian dysynchrony independent of sleep loss has been shown to have detrimental effects on health (Colwell & Matveyenko, 2014).

Perhaps the most striking finding of our study was that stepwise regression analyses revealed overwhelmingly that physical activity (mean steps taken per day) was the most significant contributor to both circadian temperature amplitude and stability in a group of non-clinically diseased (non-diabetic, normotensive, and non-medicated) young men (Figure 4). A previous study (Colwell & Matveyenko, 2014) found similar results demonstrating that obese women displayed flattened circadian rhythm temperature amplitude when compared to healthy weight women. While the subjectively assessed amount of physical activity and the time spent in MVPA was significantly greater in the healthy weight compared to the obese group of women, the associations between physical activity and circadian rhythm parameters were not reported. While physical activity has been implicated as a time cue for peripheral tissues (Atkinson et al., 2007; Callard et al., 2001), examination of its influence in concert with other environmental cues in humans under free-living conditions has not been fully elucidated. It has been previously suggested that exercising routinely may stabilize an individual's day-to-day habits, thus creating more consistent (stable) circadian rhythm (Atkinson et al., 2007). Recently, Rubio-Sastre et al. (2014) reported the contribution of 7 days of scheduled physical activity (45 min of running) occurring in morning (09:00 h) and night (21:00 h) to that of a control (no scheduled physical activity) to temperature rhythmicity in a small group of healthy-weight young women. The results demonstrated that physical activity performed at night had deleterious effects on several circadian rhythm parameters compared to both the control and morning sessions. Unfortunately, the objective physical activity monitors used in our study did not provide information concerning what time of day the activity occurred in the men we studied.

Additional analyses of our data demonstrated that just 9% of the variance in %Fat was attributed to physical activity (mean steps per day), further suggesting that increasing physical activity alone may contribute to improvements in the circadian rhythms of young men. While it has been well documented that significant contributors to obesity include lack of physical activity, physical fitness, and poor nutritional habits (excess caloric intake, skipped meals and late night eating) (Gallant et al., 2012), our findings suggest that optimizing physical activity and nutritional behavioral strategies alone may favorably influence circadian rhythm parameters independent of the current level of adiposity.

We and others have shown wrist skin temperature to be a valid method of assessing peripheral body temperature reflective of circadian rhythm (Corbalán-Tutau et al., 2011). Mounting evidence has demonstrated a link between circadian disruption and an increased risk of obesity, metabolic disorders, and cardiovascular diseases (Barclay et al., 2012; Froy, 2010). Our study demonstrates that physical activity, physical fitness, and late night eating are primary contributors to the status of the circadian system assessed as wrist temperature amplitude independent of body composition or sleep, and that the addition of fat mass only

modestly contributed to temperature instability. We suggest that increasing physical activity and fitness and reducing late night eating can improve an individual's circadian rhythm. Further research is warranted to provide additional information concerning the effects of the timing, intensity and mode of physical activity and the interactions with other circadian cues on circadian rhythm parameters in a variety of populations.

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FIGURE 1.

(A) The half hour moving average of a representative wrist temperature trace recorded over six consecutive days is shown. Black and white bars at the top of the graph show the time of sun up and sun down at the time of recording. Gray shaded bars on the graph reveal the reported time asleep. Peak (B; $r = -0.05$; $p = 0.79$) and mean (C; $r = 0.23$; $p = 0.08$) wrist temperature measured at night does not vary with percent fat. Shown are individual, peak temperature measurements with corresponding individual percent body fat showing no correlation indicating that despite increased adiposity similar heat dissipation is measured at the wrist.

FIGURE 2.

(A) Circadian amplitude data displayed as mean $\pm$ SE for optimal group (1.46 ± 0.16 ; percent fat 18.0; $n = 18$), fair group (1.38 ± 0.13 ; percent fat 18.1–23.9; $n = 20$), poor group (0.96 ± 0.11 ; percent fat 24.0; $n = 21$) (*significance at $p < 0.05$ versus optimal group, #significance at $p < 0.05$ versus fair group). (B) Circadian stability data expressed as the negative natural log of the JTK_CYCLE circadian p value mean $\pm$ SE for optimal group (1.46 ± 0.16 ; percent fat 18.0; $n = 18$), fair group (1.38 ± 0.13 ; percent fat 18.1–23.9; $n = 20$), poor group (0.96 ± 0.11 ; percent fat 24.0; $n = 21$) (*significance at $p < 0.05$ versus optimal group, #significance at $p < 0.05$ versus fair group). (C) Circadian amplitude data for subjects with a minimum of one factor of metabolic syndrome ($n = 22$) versus subjects without any metabolic factors ($n = 30$). The group with at least one metabolic syndrome factor had one of the following criteria: waist circumference > 94 cm, triglycerides > 150 mg/dL, systolic blood pressure > 130 mmHg, diastolic blood pressure > 85 mmHg, HDL < 40 mg/dL, glucose > 100 mg/dL. Data displayed as mean $\pm$ SE (* $p < 0.05$ versus group with no metabolic syndrome factors). (D) Circadian stability data for subjects with a minimum of one factor of metabolic syndrome ($n = 22$) versus subjects without any metabolic factors ($n = 30$). The group with at least one metabolic syndrome factor had one of the following criteria: waist circumference > 94 cm, triglycerides > 150 mg/dL, systolic blood pressure > 130 mmHg, diastolic blood pressure > 85 mmHg, HDL < 40 mg/dL, glucose > 100 mg/dL. Data displayed as mean $\pm$ SE. (* $p < 0.05$ versus group with no metabolic syndrome factors).

FIGURE 3.

Representative data from subjects with (A) fair adiposity and (B) poor adiposity are shown. The data is presented as the half hour, 7 day mean $\pm$ SE. Increased variability (decreased stability) is shown by the larger error in the individual with poor adiposity. In addition, a damped amplitude is evident.

FIGURE 4.

Shown is a graphical representation of stepwise multivariate regression analysis demonstrating the overwhelming contribution of physical activity to the variance of temperature amplitude and stability.

TABLE 1

Physical characteristics and behavioral factors of subjects.

	Optimal % Fat (n = 18)	Fair % Fat (n = 20)	Poor % Fat (n = 21)	Total group (n = 59)
Age (yrs)	23.8 ± 3.2	24.1 ± 2.8	26.4 ± 3.9 ^{*#}	24.8 ± 3.5
Height (cm)	177.5 ± 7.7	178.4 ± 7.4	178.0 ± 7.5	177.8 ± 7.5
Weight (kg)	77.6 ± 12.5	83.1 ± 12.7	95.9 ± 22.3 ^{*#}	85.8 ± 18.1
BMI (kg/m ²)	24.5 ± 2.9	26.0 ± 2.8	30.0 ± 5.7 ^{*#}	27.0 ± 4.6
Syst BP (mmHg)	120.4 ± 1.1	121.4 ± 1.2	125.5 ± 1.54 ^{*#}	122.6 ± 0.8
Diast BP(mmHg)	77.8 ± 0.7	78.4 ± 0.6	80.2 ± 0.8 [*]	78.9 ± 0.4
Fat mass (kg)	10.9 ± 0.7	17.6 ± 0.7 [*]	29.8 ± 0.3 ^{*#}	19.9 ± 1.5
Fat free mass (kg)	66.9 ± 2.5	65.0 ± 2.2	65.5 ± 2.4	65.8 ± 1.3
MFL mass (kg)	63.6 ± 2.4	61.6 ± 2.1	62.2 ± 2.3	62.4 ± 1.3
% Fat	13.9 ± 2.8	21.4 ± 1.5 [*]	30.4 ± 6.1 ^{*#}	22.3 ± 7.9
Waist C (cm)	81.1 ± 1.1	85.3 ± 1.2 [*]	95.7 ± 2.9 ^{*#}	87.7 ± 1.4
Abdomen C (cm)	81.5 ± 1.3	88.1 ± 1.3 [*]	99.8 ± 3.2 ^{*#}	90.3 ± 1.6
Hip C (cm)	97.5 ± 1.5	102.2 ± 1.0 [*]	109.4 ± 2.2 ^{*#}	103.4 ± 1.2
VO2 peak (ml/kg/min)	61.6 ± 1.8	56.0 ± 1.3 [*]	45.8 ± 92.0 ^{*#}	54.1 ± 1.3
Mean sleep (hrs/night)	8.0 ± 0.2	7.9 ± 0.2	7.5 ± 0.2	7.8 ± 0.1
Skip breakfast (#/wk)	0.5 ± 0.3	2.0 ± 0.5 [*]	1.4 ± 0.4	1.3 ± 0.2
Skip lunch (#/wk)	0.8 ± 0.3	0.8 ± 0.2	0.7 ± 0.2	0.8 ± 0.1
Skip dinner (#/wk)	0.5 ± 0.2	0.4 ± 0.1	0.5 ± 0.2	0.5 ± 0.1
Late night eating (#/wk)	3.06 ± 0.48	3.40 ± 0.494	2.84 ± 0.40	3.11 ± 0.26
Mean steps (steps/day) [§]	8911.5 ± 500.3	9055.9 ± 966.6	6823.5 ± 677.4 [*]	8158.9 ± 442.3
Mean mileage (miles/day) [§]	4.2 ± 0.3	4.1 ± 0.4	3.3 ± 0.3	3.8 ± 0.2
MVPA (mins/day) [§]	32.3 ± 3.5	31.3 ± 4.2	24.8 ± 3.5	29.1 ± 2.2
Total cholesterol (mg/dL)	147.71 ± 5.78	157.65 ± 4.43	161.22 ± 6.81	155.63 ± 3.38
HDL (mg/dL)	62.00 ± 2.83	54.88 ± 3.11	46.28 ± 2.90 [*]	54.23 ± 1.90
Total cholesterol/HDL	2.44 ± 0.10	3.05 ± 0.21 [*]	3.80 ± 0.40 [*]	3.11 ± 0.17
LDL (mg/dL)	73.00 ± 4.18	84.88 ± 4.90	92.50 ± 5.09 [*]	83.63 ± 2.92
Triglycerides (mg/dL)	63.76 ± 5.30	89.06 ± 11.48	112.17 ± 18.77 [*]	88.79 ± 8.05
Insulin (μIU/mL)	3.88 ± 0.37	6.89 ± 1.49	9.73 ± 3.21	6.89 ± 1.23
Glucose (mg/dL)	87.61 ± 1.66	96.25 ± 2.50 [*]	96.04 ± 2.21 [*]	93.41 ± 1.35
HOMA-IR	0.84 ± 0.08	1.78 ± 0.48	2.50 ± 0.93	1.72 ± 0.36
Metabolic syndrome factors	0.18 ± 0.10	0.29 ± 0.11	1.33 ± 0.37 ^{*#}	0.63 ± 0.15

Data is displayed as Mean ± SE.

Resting heart rate (HR), resting systolic blood pressure (Syst BP), Resting Diastolic Blood Pressure (Diast BP), Mineral Free Lean Mass (MFL), Waist Circumference (Waist C), Abdominal Circumference (Abdominal C), Hip Circumference (Hip C), Moderate to Vigorous Physical Activity (MVPA), low density lipoproteins (LDL), homeostasis model assessment insulin-resistance (HOMA-IR).

*
 $p < 0.05$ versus optimal %Fat group.

 $p < 0.05$ versus fair %Fat group.

\$
Indicates $n = 55$ (17 Optimal %Fat; 17 fair %Fat; 21 poor %Fat group) mean steps, mileage, MVPA (time spent in moderate to vigorous activity) computed with a minimum of four days of data.

Indicates $n = 52$ (17 Optimal %Fat; 17 fair %Fat; 18 poor %Fat group).
Mean sleep, skipped breakfast, lunch and dinner and late night eating computed from 7 days of data.

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TABLE 2

Unstandardized and standardized estimates of the contributing factors to the models of temperature amplitude and temperature stability.

	Temperature amplitude			Temperature stability		
	<i>r</i>	β Estimate $\pm$ SE	Standardized β estimate	<i>r</i>	β Estimate $\pm$ SE	Standardized β estimate
Mean steps (steps/day) ^{\$}	0.41 *	9.03 ¹⁰⁻⁵ $\pm$ 0.000	0.457	0.50 *	0.028 $\pm$ 0.005	1.000
VO2 peak (ml/kg/min)	0.49 *	0.021 $\pm$ 0.006	0.372			
Late night eating (#/wk)	-0.26	-0.092 $\pm$ 0.035	-0.283	-0.15	-10.589 $\pm$ 4.735	-0.230
MVPA (mins/day) ^{\$}				0.24	-2.706 $\pm$ 1.007	-0.468
Fat mass (kg)				-0.33 *	-0.002 $\pm$ 0.001	-0.226

All β estimates were significant ($p < 0.05$) contributors to the models.

* $p < 0.05$.

^{\$} Indicates $n = 55$ (17 Optimal %Fat; 17 fair %Fat; 21 poor %Fat group) mean steps per day and MVPA (time spent in moderate to vigorous activity) computed with a minimum of four days of data.