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Emotional response to perceived racism and nocturnal heart rate variability in young adult African Americans

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Abstract

Background: Heightened autonomic nervous system (ANS) arousal is a well-established contributor to the effect of stress on adverse cardiovascular health outcomes which disproportionately affect African Americans. ANS arousal is normally attenuated during sleep and compromise of this shift is associated with multiple adverse cardiovascular outcomes. Parasympathetic nervous system (PNS) dominance during sleep can be altered by stress. Racism has been recognized to have many negative health consequences in African Americans. Perceived racism has been linked to ANS activity, however, we are not aware of prior research on racism and nocturnal ANS balance.

Objective: To examine relationships between perceived racism and nocturnal ANS activity indexed by heart rate variability (HRV) in healthy African American men and women age 18-35.

Methods: Fifty-four participants completed the Perceived Racism Scale and had 24-hour ambulatory electrocardiogram recordings in their homes. Power spectral analysis was used to derive normalized high frequency (nHF) to index PNS activity which was computed by 5-minute epochs during wake and sleep.

Results: Endorsement of racism and negative emotional reactions during the past year were inversely related to nHF during time in bed. Multiple regression analysis indicated that negative emotional reactions were a significant predictor of nHF during the sleep period $F(2,54) = 4.213$, $p = .020$, $R^2 = .135$ (adjusted $R^2 = .103$). Relationships during wake were not statistically significant.

Conclusion: Findings suggest that perseverative thoughts triggered by negative emotional reactions to racism influencing nocturnal ANS activity may be a pathway by which perceived racism affects health.

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Keywords

Heart Rate Variability; Sleep; Stress; Cardiovascular Disease; Racism; health disparities

Introduction

Compromised sleep is associated with increased risk for obesity (Cappuccio, et al., 2008), diabetes, cardiovascular disease (Ayas et al., 2003a, b), and early mortality (Kripke, et al. 2002). These conditions disproportionately affect African Americans (CDC, 2011). In a population-based study, short sleep duration (< 6 hrs) was more common among African American adults (Hale & Do 2007). Sleep in healthy subjects features a shift to parasympathetic dominance of the autonomic nervous system (ANS) especially during non-rapid eye movement sleep (NREM) (Burgess et al., 2004; Trinder et al., 2001). Parasympathetic nervous system (PNS) dominance during sleep may be an important mechanism for maintaining homeostasis and overall health; evidence includes associations of failure to shift to PNS dominance during sleep with blood pressure non-dipping (Kohara et al 1995) which is a well documented risk factor for adverse cardiovascular outcomes. Increased sympathetic nervous system (SNS) activity during sleep is associated with myocardial infarction (Guzzetti et al, 2002), and sleep apnea (Narkiwicz & Somers, 1997). Additionally, our recent work suggests a relationship between nocturnal ANS activity and immune system activity, with increased SNS activity during the first half of the night being associated with increased morning levels of the pro-inflammatory cytokine interleukin-6 (Bell, Kobayashi, et al, 2017).

Our laboratory has previously demonstrated differences in nocturnal ANS balance between African American research participants with posttraumatic stress disorder (PTSD) versus those with resilience to developing PTSD (Kobayashi, Lavela, Bell & Mellman, 2016). A subsequent analysis provided evidence that this difference may be attributable to characteristics of the resilient group (Mellman, Bell, & Abu-Bader, 2018). This paper also reported a relationship between nocturnal ANS balance and stressful neighborhood characteristics. While significant relationships were found with neighborhood disorder and sleep duration, a substantial amount of the varaince in predicting nocturnal ANS balance remained unaccounted for.

Brosschott, Pieper, & Thayer (2005) suggest that the initial response to acute stressors has only a transient impact on ANS activation, and mental representations of stressors need to be persistent to extend their physiological effects. Under the perseverative cognition hypothesis, Brosschott, Gerin & Thayer (2016), argue that persistent worry and anticipatory stress can prolong the stress response during wake periods, continue into sleep periods, and be associated with slow ANS recovery (Brosschott et al., 2007; Broschott, et al., 2005). Perseverative cognitions are therefore theorized to be a major effector of the impact of stress on ANS activity (Brosschott et al, 2006; Broschott et al., 2010). Previous research has indicated an effect of anticipatory stress on nocturnal ANS activity (Hall et al, 2004), however studies of the effects of psychosocial and emotional stress on nocturnal ANS activity are limited

Though the majority of research on perseverative cognition and ANS activity has focused on the general population, racism, and emotional response to acute and chronic exposure to racism is an insidious form of stress that affects African Americans and other minority groups. Research has suggested that racial discrimination contributes to health disparities and poor cardiovascular health outcomes in African Americans (Davis, Liu, et al., 2005; Harrell, Burford, et al., 2011; Drevdal, Taylor & Phillips). In a comprehensive review on the effects of racism in African American populations, Lockwood, Marsland, et al. (2018), confirm relationships between hypothalamic-pituitary-adrenal axis activity including ANS and endocrinological dysregulation as a result of exposure to chronic exposure to racist events. Consistent with anticipatory mehcanisms being related to responses to racism, Feagin & Sykes (1994) suggest that individuals can mentally prepare for the possibility of discrimination on a daily basis which Clark, Benkert, et al, (2006) refer to as “racism related vigilance.” Anticipation and chronic experience of racial discrimination may therefore increase ANS arousal during sleep and thereby contribute to adverse health outcomes.

Negative affectivity and negative emotional responses, including: hostility and anger (Suarez et al, 1997), anxiety (Thayer, Friedman & Borkovec, 1996), and depression (Thayer & Lane, 2000) have also been linked to cardiovascular disease and other adverse health outcomes (Steptoe & Kivimaki, 2012; Chida, Steptoe, 2009; Suarez et al 1998; Thayer & Lane, 2000) in the general population. Assari found that the relationship of hostility and anger to cardiovascular mortality was stronger among African Americans than among European Americans (Assari, 2017).

ANS arousal is determined by the balance between activity of the SNS and PNS. Heart rate variability (HRV) is a non-invasive method that is used to indirectly assess ANS activity from sets of consecutive R-peaks in the cardiac cycle. Malik et al. (1996) established guidelines for time and frequency domain methods of HRV using different algorithms to convert R-peak series into signals of parasympathetic and sympathetic control. In using frequency domain analysis, electrocardiogram signals are filtered into different frequencies including low frequency (LF) and high frequency (HF) bands where each band reflects either circadian patterns or sympathetic and parasympathetic (vagal) influences. In a study examining HRV during sleep, results confirmed consistent reproducibility for the HF band indexing PNS activity, whereas reproducibility was low for the low frequency band and the LF/HF ratio often used to index SNS activity (Herzig et al. 2018).

Due to the uncertain reliability of the LF/HF ratio during sleep (Herzig et al. 2018), we focus here on nocturnal vagal tone. Previous studies have focused on blood pressure and HRV parameters during periods of wakefulness in relation to racism but have not examined nocturnal HRV as a function of perceived racism and all focused on acute effects. The objectives of the present study are to: (1) examine whether perceived racism, and emotional response to racist events influence nocturnal ANS activity via analysis of heart rate variability (HRV).

Methodology

Participants:

Participants were young adult (18 – 35) African Americans (self-identified) recruited between 2008 and 2012 from the Washington, DC metropolitan area via posting flyers and referrals from prior participants. The protocol was approved by the Howard University (HU) Institutional Review Board and participants reviewed a detailed description of the study and provided written informed consent prior to participating. Pre-screening evaluations were conducted at the Clinical Research Unit (CRU) of Howard University Hospital.

Initial phone and in-person screenings excluded participants taking medications or engaging in behaviors that would affect sleep or heart rate variability.These exclusions included a body mass index >=40, excessive use of caffeine (>5 cups of coffee or its equivalent of other caffeinated drinks per day), heavy smoking (>20 cigarettes per day) or hazardous drinking (>14 drinks/week in men, >7 drinks/week in women), chronic medical conditions or severe psychiatric illness (e.g. schizophrenia, bipolar disorder, chronic depression that was not secondary in onset and severity to PTSD, psychotic disorders), and any other condition that required consistent use of medications, and having regular night shift work or unusual habitual sleep or rise times (i.e. after 3AM, after 11AM, respectively). Current alcohol or substance abuse/dependence/misuse reported during the clinical interview, and/or positive urine toxicology screening for illicit drugs were also exclusionary.

Another exclusion criterion was sleep apnea quantified as an apnea/hypopnea index (AHI ≥ 5 on a screening sleep recording). As in Mellman et al. (2018) a more conservative approach to apnea exclusion was used compared to previous reports from the study (Mellman et al, 2015) due to the effect apneic events have on ANS activity (Narkiwicz & Somers, 1997). All of the current sample were participants in the study recently reported by Mellman et al. (2018), however the PRS was only administered to the first cohort of this study resulting in N=57. For a more complete description of the study population see Mellman (2018).

Measures

Self-report measures—Self-report measures included a demographic questionnaire assessing age, gender, race/ethnicity, highest education level, and the Perceived Racism Scale (PRS) (McNeilly, MD, et al., 1996). The Perceived Racism Scale consists of 51 items assessing the perceived experience of racial discrimination by African American adults. The PRS consists of three sections totaling 51 items, each section is scored separately and can serve as stand alone measures. The scale provides an overall score for perceived racism throughout one's lifetime, and for the year, in academic settings, on the job, in public, and exposure to racist statements and/or microaggressions and attitudinal and behavioral manifestations of racism. Scales have been shown through factor analysis to factor separately with respect to the different scales, (Atkins, 2014).

The first section consists of 43 items on a 5-point Likert type scale that measures frequency of exposure to such events and/or experiences ranging from "Not applicable to "Several times a day" . Individual items for this section include questions assessing an individual's perception of racist events and includes items such as "I've been made to feel uncomfortable

in a classroom of white students" and "I've been called insulting names related to my skin color". Participants answer these questions in sets of two, where the first assesses racist encounters for the year and the second during the lifetime. For the purpose of this study, which was to related racism exposure to a current physiological measure, we analyzed relationships for encounters during the past year.

Section two of the PRS, the negative emotional response segment, consists of four items assessing to what extent they feel a particular emotion in response to racist interactions and/or perception of microaggressions (anger, hurt, frustrated, sad, powerless, hopeless, ashamed, empowered/strengthened) directed towards one's race. Participants rate items on a 5-point Likert scale ranging from "Not at all" to "Extremely" amongst individual items such as "When I hear racist statements I generally feel", "When I experience racism in the public realm I generally feel". The item feeling "empowered/strengthened" is reverse coded.

The PRS was administered to the first cohort of participants that included 57 of those who went on to have ambulatory monitoring. All participants endorsed having perceived racism and some degree of negative emotional response to those events. Cronbach's Alpha was run to test the internal consistency of the PRS Cronbach's Alpha = .951 for exposures during the past year and .521 for the emotional responses scale.

Physiological Measures

Polysomnography and Heart Rate Variability (HRV)—Overnight polysomnography (PSG) recordings were conducted 1-2 weeks before ambulatory cardiac monitoring to exclude apnea. Recordings were obtained using an Embla (Denver, Colorado) Titanium portable unit. Electrode placement included bilateral frontal, central, and occipital leads, two electrooculograms, and chin electromyograms. Participants were instructed to go to bed and get up at near their normal times. PSG results are reported elsewhere (Mellman, et al. 2014). Two 24-hour ambulatory electrocardiogram (EKG) recordings were collected 1-week apart. Actigraphy was used to relate cardiac measures to sleep duration and continuity. Ambulatory blood pressure (BP) monitors utilizing an oscillometric method (Spacelabs 90207) were programmed to inflate and record BP every hour. BP results are reported elsewhere (Mellman et al. 2015). Participants then left the CRU and assumed normal daytime activities and returned the next morning.

Continuous measurement of the EKG was obtained using the Nasiff Cardio™ system (Brewerton, NY; www.nasiff.com). Skin was prepared using alcohol swabs over designated spots for electrode placement, then 5 electrodes were placed over the sternum and lower ribs to enable 3 channel recording to a small ambulatory recorder). Participants were instructed to avoid getting the recording device and electrodes wet and avoid contact with water and pushing or pulling on electrodes. After set-up and baseline monitoring for 15-minutes, electrodes were adjusted to minimize artifact, and following this, EKG recording began. The participants left the CRU and returned 24-hours later to end the EKG recording. Signal quality was checked before the participants' departures. The EKG ambulatory monitoring system was set to digitize recordings at a sampling frequency of 256 Hz.

Actigraphy and Sleep Diaries—A night of actigraphy alone was obtained after the first full ambulatory monitoring to determine whether sleep was disrupted by blood pressure cup inflation. Participants were fitted with actigraphs (MicroMini-Motionlogger, AMI, Ardsley, NY) they then resumed normal daily activities to examine relationships between sleep duration and continuity with cardiac measures. Sleep diaries required participants to report bed times and wake times during weekdays and weekends, number of awakenings, and length of time to fall asleep. This information was used in calculating total sleep time, wake times, and wake after sleep onset. Wake time, and time in bed (tib) for HRV parameters were determined by actigraphy and sleep diary reports. Time in bed was operationalized as periods spent in bed after lights out.

Data Cleaning and Analysis—When equipment was returned after recording, flash cards were downloaded to CardioCard for Windows on a PC or laptop where the data were stored. Recordings from the second full day of ambulatory recording (week after the first) were analyzed for HRV. The entire recording was reviewed by 5-minute epochs to confirm accurate automatic marking of R-peaks in the waveform. Epochs with artifact that obscured and prevented accurate identification of successive R-peaks were excluded from analysis. Any record that contained >= 30% of unusable epochs due to artifacts or unidentifiable wave forms was excluded.

CardioCard software (Nasiff) calculated power spectral density for LF (0.04-0.15 Hz) and HF(0.15-0.40 Hz) for each nonoverlapping 5-min epoch using fast Fourier Transformation. HF was normalized by dividing HF by the sum of FF and HF. Normalized HF (nHF) was used to represent PNS activity. After nHF was computed, data were imported to SPSS 25.0 (IBM) for analysis and inspected for normality. Wake time, and time in bed (tib) for HRV parameters were determined by actigraphy and sleep diary reports. Time in bed was operationalized as periods spent in bed after lights out. Bivariate correlations using Pearson's r were run between perceived racism, negative emotional response to racist events and nHF. PROCESS Version 3 (Hayes, 2017) was used to conduct mediation analysis with negative emotion as a mediator between perceived racism and nocturnal nHF.

Results

Participants of this study was N=85, however the PRS was administered to the first cohort of participants resulting in N=57 (54.4% female). Demographics including gender, age, body mass index, total sleep time and wake after sleep onset (WASO) are presented in Table 1.

Correlations are presented in Table 2. Perceived racism with nHF during time in bed (nHF_{tib}) was marginally significant, r = −.256, p =.052. Standard multiple regression was performed for nHF_{tib} as the criterion, and perceived racism and negative emotional responses to racist events as predictors. The regression model for nHF_{tib} was significant, $F(2,56) = 4.213$, $p = .020$, $R^2 = .135$ (adjusted $R^2 = .103$) with the two predictors accounting for 10.3% of the variance for nHF_{tib}. This effect was driven by negative emotional response to racist encounters with $\beta = -.308$, $p = .027$. Beta values for the PRS was not statistically significant in the model even though it had a marginal first order correlation with nHF.

In order to examine whether these relationships were independent of the previously reported associations with Neighborhood Disorder (Mellman et al., 2018) we ran a stepwise regression predicting nHF_{tib} with Neighborhood disorder in the first step and perceived racism and negative emotional response in the second. After controlling for neighborhood disorder, perceived racism and negative emotional response explained a similar significant proportion of the variance in nHF_{tib} with $\Delta R^2 = .129$. $p = .022$. Because other covariates such as smoking may influence the outcome, we performed the multiple regression without these participants and results remained statistically significant, $F(2.54) = 3.944$, $R^2 = .132$ (Adjusted $R^2 = .098$), $p = .025$. Because the possibility of covariates influencing the outcome, the model was run with body mass index, age, education and gender as covariates. In this model, negative emotional response to racist encounters was the sole significant predictor.

Discussion

The purpose of the present study was to evaluate relationships of perceived racism and negative emotional responses to racist events with nocturnal PNS activity indexed by HRV. This study is the first to our knowledge to examine the effects of perceived racism and nocturnal HRV. As hypothesized, results show that exposure to racist events is negatively related to the indicator of PNS activity during participants' time in bed with marginal significance in an unadjusted model, however negative emotional reactions to racist events remains a significant predictor in an adjusted model. This effect may result from negative emotional reactions to chronic experiences with racism triggering perseverative cognitions preceding sleep that affect physiological activity during sleep. This formulation is consistent with Clark & Benkert's (2006) hypothesis on race related vigilance, and Brosschott et al.'s hypothesis of unconscious and anticipatory worry potentiating adverse physiological effects (Brosschott et al 2006; Brosschott, 2007), with concerns regarding racism serving as a specific perseverative cognition that disrupts the normal shift to PNS dominance seen in healthy sleep. While these relationships were found during sleep, they were not found during wake times, possibly due to other stressors and factors affecting ANS activity and obscuring the effects of perceived racism. An analysis further indicated that the variance accounted for by perceived racism and negative emotional reactions is independent of the variance accounted for by Neighborhood Disorder. This may be due to the relative segregation of minorities living in poorer neighborhoods, whereas those less exposed to neighborhood violence and decay may encounter individuals of other ethnicities more often and therefore be more exposed to actions perceived to be racist (South & Crowder, 2018).

Our study findings suggest that negative emotional responses to perceived racism affect PNS activity during sleep in a manner that could have adverse health consequences through diminished vagal tone which in turn could influence immune (Bell et al., 2018) and neurovisceral systems (Thayer & Lane, 2000). This is consistent with other studies that relate discrimination and racism to changes in HPA axis activity depending on whether exposure is acute or chronic, (Busse, et al. 2017). Slopen et al. (2016) in a review also confirm endorsement of discrimination to be related to poor sleep, however no objective measures were used. Additionally, though perceived racism showed a trend with decreased vagal tone and models were significant, the effects were primarily driven by negative

emotional responses towards racist encounters rather than the actual encounter itself. Thus effects on nocturnal heart rate variability appear related to feelings of sadness, hopelessness, frustration and powerless in response to racism. Such relationships are consistent with previous research examining negative affectivity and cardiac function, where neuroticism and rumination have been associated with lower levels of heart rate variability persisting into sleep periods, (Brosschott, Gerin & Thayer, 2016). Rumination on, racist encounters may serve as a present stressor thereby influencing heart rate variability and other physiological markers, (Gerin et al, 2006). Collectively these findings provide a route to cardiovascular disease and immune system activity, (Thayer & Lane, 2000). It is also important to note the possibility of trait rumination serving as a mediator between heart rate variability, and anxiety/depression resulting from chronic exposure to racism. Trait rumination, a facet of both depression and anxiety have been shown to affect blood pressure and vagally mediated heart rate variability, (Gerin et al. 2006). Supporting previous research on negative affectivity, trait rumination may influence emotional regulation to adverse events such as exposure to racism, (Williams. Pandya, Hill, Kemp, Way, Thayer, & Koenig, 2017).

Limitations of the current study include that racism was assessed retrospectively and did not include a racism vigilance measure. A further limitation is not having controlled for daily life stressors (Brosschott et al., 2007). Additionally, personality variables, including trait rumination may serve as antecedents to an individuals' emotional response to racist events and other coping mechanisms were not evaluated in this study. The cross-sectional nature of this study presents a further limitation and there is a possibility of other pathways and other variables influencing nocturnal heart rate variability.

Future studies should include race related vigilance assessments (Clark & Benkert, 2006) and address the role of sympathetic activity and the dynamic interplay between branches of the ANS possibly by including the pre-ejection period (PEP) and monitoring autonomic balance and shifts during wake periods with respect to daily stressors and sleep. In addition, ecological momentary assessment (EMA) could serve as a useful tool for quantifying wake time stressors that may not be evident due to environmental noise and subconsciously stressors including perceived racism and/or perseverative cognitions that may occur during wake periods, (Zawadazki et al. 2019).

In conclusion, we found effects of perceived racism and negative emotional responses on vagal activity during sleep which could potentiate CV risks in a population already at risk for adverse cardiovascular events. Clinical implications of this study include interventions that may potentiate parasympathetic function prior to sleep to decrease negative emotional responses to and perseverative cognitions resulting from racist encounters to improve sleep and maintain the normal shift to parasympathetic dominance during sleep.

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Highlights

- The association of perceived racism and vagal activity during sleep was marginally significant
- A model that included perceived racism and negative emotional reactions to racism was significant in predicting vagal tone during sleep and accounted for 10.3% of the variance
- Negative emotional response was a significant mediator between perceived racism and nocturnal heart rate variability
- The impact of perceived racism and negative emotional response on vagal tone during sleep could be a pathway by which racism impacts health

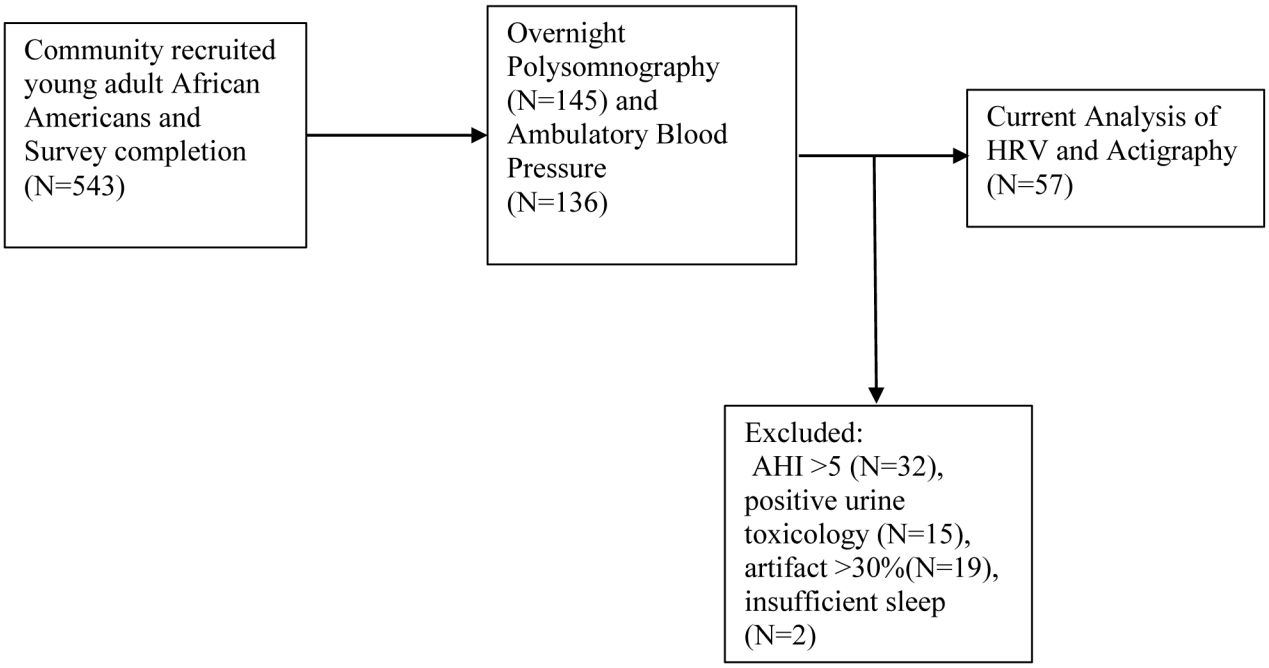

Figure 1.
Flowchart of study population and procedure. Exclusion did not receive Perceived Racism Scale (PRS).

Table 1.

Participant demographics. WASO = Wake after sleep onset.

	N	Mean	SD	Range
Female	31(54.4%)			
Male	26(45.6%)			
Age		22.95	4.63	18-35
Body Mass Index		25.38	4.31	17-40
Total Sleep Time (min)		334.77	113.59	74-655
WASO		68.82	66.12	1-335

Table 2.

Correlations between perceived racism, negative emotional response, and cardiac metrics.

	Age	Gender	Perceived Racism	Negative Emotional Response	nHF_{tib}	nHF_{wake}	TST	WASO
Age	-							
Gender	.035	-						
Perceived Racism	.130	−.024	-					
Negative Emotional Response	.117	−.059	.396**	-				
nHF_{tib}	−.056	.151	−.256*	−.350**	-			
nHF_{wake}	−.072	.150	−.089	.248	.635**	-		
TST	.109	−.036	−.032	.131	.230	.054	-	
WASO	.069	.097	.038	−.266*	.041	.028	.100	-

** p<.001,

* p=052. nHF_{tib} = normalized high frequency time in bed. nHF_{wake} = normalized high frequency during wake period. TST = Total sleep time. WASO = Wake after sleep onset.