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Original Research

GESDAV

Weekend-weeknight shifts in sleep duration predict risk for metabolic syndrome

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ABSTRACT

Background: Although sleep deprivation and shift-work are associated with risk for metabolic syndrome (MS), it is less evident whether small shifts in sleep duration (operationalized here as the difference between weekend (WE) and weeknight (WN) sleep duration) also predict risk for MS independently of average sleep duration. The purpose of the present study was to examine whether a difference in WE and WN sleep duration was found among adults across a wide age range, whether it predicted number of symptoms of MS after controlling for estimated sleep duration, whether the difference in sleep duration was moderated by age, and to test indirect effects via the specific diagnostic criteria for MS (blood pressure [BP], high-density lipoprotein, triglycerides, glucose, and waist circumference). **Methods:** The study reanalyzed archived data. Participants were business leaders attending workshops in 2007-2009 who self-reported WE and WN sleep duration, and provided physiological measures collected by health care professionals. **Results:** Shifts in sleep duration from WE to WN were reported throughout this sample. The difference predicted number of symptoms of MS independently of overall sleep duration and relevant covariates (age, gender, minority ethnicity, history of smoking, diet, exercise and body mass index). The association was not moderated by age, and included a significant indirect effect by way of changes in glucose level. **Conclusion:** Small inconsistencies in WE to WN sleep duration predicted risk for MS independently of average sleep duration and age. This association may be indirect via changes in glucose levels.

KEY WORDS: Business leader, circadian, glucose, metabolic syndrome, sleep duration inconsistency, weekend sleep duration

INTRODUCTION

Metabolic syndrome (MS) is present in approximately 24% of adults in the United States, and varies by ethnicity, body mass index (BMI), age, smoking, socio-economic status, diet, and level of physical activity [1,2]. MS is defined as the presence of >3 criteria that exceed established thresholds (blood pressure [BP], fasting blood sugar (glucose), waist, high-density lipoprotein (HDL), and triglycerides) [1]. It is a risk factor for type 2 diabetes and cardiovascular disease, among other disorders.

MS is associated with sleep duration, quality, apnea, and circadian disruption. Relative to individuals who reported routinely getting 7-8 h of sleep at night, middle-aged short sleepers' (<7 h) odds of having MS increased 45% [3]. Short sleepers had larger waists and higher glucose and triglyceride levels. Strong evidence links chronic circadian disruption (e.g. night shift-work) predicts increased rates of MS [4], regardless of age and physical activity [5]. Lifestyle choices may explain the link between circadian disruption and MS. Shift-workers may sleep less than day workers, exercise less, or consume a less healthy diet [6]. Other explanations include circadian disruptions of metabolism [7] or expression of clock genes [8].

While shift-work induces large disruptions in circadian patterns, a more common source of disruption is weekend to weeknight changes in sleep patterns (WE/WN change). The additional sleep on WEs may be insufficient to make up for sleep lost during the week. Assuming a need for 8 h of sleep per night [9], routinely sleeping 6 h/night during the week followed by two nights of 9 h may result in an accumulating net loss of sleep.

Cognitive, behavioral or health deficits resulting from WN sleep restriction may not be completely ameliorated by WE recovery sleep. Pejovic *et al*. found recovery sleep after restriction did not restore attentional decline to baseline level [10]. Other findings, however, suggest a benefit of catch-up sleep on WEs due to increased average sleep duration. For example, adults who reported an extra hour of sleep on WEs were less likely to be hypertensive [11-13].

Rationale and Hypotheses

It is unclear whether increased sleep duration provided by WE/WN change or the frequent circadian disruptions is the better predictor of health outcomes. WE to WN change may increase sleep debt [3] and associated daytime sleepiness [14], and

represent slight weekly disruption in circadian patterns [15]. The difference in WE to WN sleep appears to be related to, but different from, average duration [16]. The present study conceptualized this relatively small circadian change separately from average duration. Separate representation allowed examination of WE/WN change in sleep duration independently of average duration (i.e. a mild circadian disruption rather than lost sleep) [8]. WE/WN change was expected to predict worse health (operationalized as number of symptoms of MS >established thresholds) after controlling for average duration.

Studies have reported WE/WN change among adolescents and younger adults [16], but less is available about this pattern in middle- or older-adulthood. This study examined whether WN/WE change and its association with MS occurred throughout this sample. The ability of the circadian rhythm to adapt to frequent change diminishes across adulthood. Age-related changes in sleep duration and fragmentation may be exacerbated by shift-work [17,18]. Animal models indicate increased mortality among older animals exposed to phase advances or weekly reversal of light and dark [19]. Therefore, this study hypothesized that WE/WN change in sleep duration would increasingly predict symptoms of MS with age (i.e., a moderated effect).

Given that each symptom of MS may be sensitive to circadian changes, we expected small but significant indirect effects of WE/WN change when controlling for co-occurring symptoms. Circadian disruptions may contribute indirectly to MS via changes in fat distribution (operationalized as waist size) [20,21]. Shift-workers appear to be at risk for indices of worse metabolic processes, although findings are inconsistent, and may be moderated by age. Lower levels of HDL have been reported in some studies [4], but not all [22]. Likewise, associations with triglyceride and glucose levels have also been reported [4], but not in all cases [22]. Esquirol *et al*. [23] reported increased risk of high BP, high triglyceride levels, MS and possibly high BMI. Associations with glucose metabolism were less clear. While shift-workers may have higher BP [24-26], extra sleep on the WE may be protective [11]. Therefore, although an indirect effect was expected through BP, arguments could be made for either a positive or negative association.

METHODS

Participants

The present archived data included participants recruited in 2007-2009 from a development workshop for business leaders (Center for Creative Leadership) held in the Southeastern United States. Participants (N = 315) self-reported health and demographic information and provided objective physical measurements. They were primarily male, Caucasian, well-educated, and represented diverse businesses [public, for-profit, non-profit; Table 1]. Inclusion of business leaders represents both a strength and a weakness of the present study. The nature of the sample limited generalizability of findings, but bolstered number of reports of sleep and MS in this population. While other studies have included workers of similar socioeconomic status (e.g. sleep duration and safety among physicians), many studies, particularly in the shift-work literature, involve lower-status employees and may not generalize to other occupational statuses. Level of employment may be relevant to sleep and health [27,28]. Leadership often involves long work hours and high responsibility. Leaders may be older than other employees, and have more health care resources or education. Organizational hierarchy may predict hypertension [29] or MS [30], suggesting that workplace status should be considered when studying risk factors for MS, including sleep.

Table 1: Descriptive data

	Mean	SD	Range
Gender (81% male, n=255)			
Ever smoked (35% yes, n=111)			
Minority status (25%, n=79)			
Education (65% degree≥Master's/ professional degree, n=204)			
Age	48.65	6.51	32-67
Body mass index	26.53	3.98	18-44
Exercise[a]	4.60	1.81	2-8
Diet[b]	3.71	1.78	0-10
Average hours sleep[c]	6.90	0.69	4.57-8.57
Average discrepancy[d]	1.11	0.96	0-5
Systolic BP	123.19	13.98	90-173
Diastolic BP	80.57	9.57	54-120
Glucose	97.96	14.16	72-227
Triglycerides	123.36	84.41	27-697
HDL	57.73	15.46	27.20-149.60
Waist/cm	94.71	13.32	63-134
Metabolic Syndrome[#]	0.47	1.13	0-5

[a]Average number hours engage in resistance or aerobic exercise/week, [b]Average number servings of fruits or vegetables/day, [c]([Typical weekday sleep×5] + [typical WE sleep×2])/7, [d]Typical WN sleep-typical WN sleep, [#]Symptoms Metabolic Syndrome, N=315; includes data interpolated using linear trend-missing data for all variables≤4%, BP: Blood pressure, HDL: High density lipoprotein, SD: Standard deviation, BMI: Body mass index, SD: Standard deviation

Materials and Procedure

The original study was approved by an institutional review board, as was the present reanalysis of archived data. Prior to attending the workshop, participants provided health-related data (e.g. typical WN and WE sleep duration, exercise, diet) via electronic survey. Using Hall *et al*. [3] as a guideline, average sleep duration was estimated as (WN sleep*5 + WE sleep*2)/7. This formula weighted shorter WN duration more heavily than WE duration, and may indicate sleep debt [3]. WE minus WN duration indicated weekly change; higher scores represented greater discrepancy.

Participants received a morning physical examination in which qualified health professionals verified self-reported data (e.g. height, weight) and collected objective measures of health, including symptoms of MS (BP, fasting glucose, waist circumference, HDL, and triglycerides) [1]. These data were used

to calculate BMI and number of risk factors for MS [Table 1]. Risk factors for MS were defined as BP >130/85 mmHg, fasting blood sugar (glucose) >100 mg/dL, large waist circumference (Men: >102 cm women: >88 cm), low HDL (men: <40 mg/dL, women: <50 mg/dL), and triglycerides >150 mg/dL. Number of risk factors for MS was the number of symptoms that met or exceeded the thresholds (range 0-5) [31].

Hypotheses and Analyses

This study examined whether (1) WE/WN change predicted symptoms of MS after controlling for average sleep duration, (2) the moderating effect of age, and whether there were (3-7) indirect effects via each symptom of MS. A-priori power analysis indicated that a sample size of 293 provided power of 0.95, assuming a small effect size ($R^2 = 0.10$), eight covariates and seven predictors [32]. Multiple regression analysis tested the main effect of WE/WN change and its moderation by age. Age, gender [33], minority status [34], smoking [35], diet (number of servings fruits or vegetables/day) [36], exercise (hours aerobic or resistance exercise/week) [37] and BMI [16] were entered in an initial block as covariates, along with average sleep duration. WE/WN change was added in block 2, and the interaction term in block 3. To test the hypothesized moderation, WE/WN change and age were centered before multiplying, and the centered variables used as main effect predictors. Path analysis (confirmed by bootstrapping) tested indirect effects via systolic and diastolic BP, waist measurement, and fasting levels of HDL, and triglycerides, allowing these measures to co-vary. The outcome variable was number of symptoms of MS.

RESULTS

Preliminary Analyses

All participants' WE/WN change was >0, indicating equal or extended sleep duration on WEs. Risk factors for MS were higher among males, those who had smoked, those with higher BMI, and those with larger WE/WN change [Table 2]. Although correlations of symptoms of MS with age, minority ethnicity, exercise, and diet were not significant, they were left as covariates in the model because of their established association with MS and sleep. Sleep duration and WE/WN change were modestly correlated, suggesting they are related but not identical constructs. Increasing age predicted less sleep duration, but there appeared to be no correlation of age with WE/WN change. Although correlations of symptoms of MS with the two sleep variables were similar, only the correlation with WE/WN change reached significance. Individual predictors of MS were significantly correlated with each other as well as with the outcome, justifying their simultaneous inclusion in the model. Other work has reported a dichotomous outcome of MS (<3 symptoms vs. ≥3 symptoms) rather than number of symptoms. Logistic regression using this dichotomous outcome produced a similar pattern of results. Number of symptoms was reported because it had slightly more variability (0-5 vs. 0-1) and indicated increasing risk.

Planned Analyses

The first hypothesis predicted that WE/WN change would be significantly predict number of symptoms of MS [Table 3].

Table 2: Bivariate correlations

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Gender (80% male)	-														
Ever smoked (35% yes)	0.10	-													
Minority status	0.04	0.002	-												
Age	−0.03	0.11*	−0.10	-											
Body mass index	0.39**	0.14*	0.03	0.02	-										
Exercise[a]	−0.08	−0.06	−0.03	0.03	−0.06	-									
Diet[b]	−0.15**	−0.09	−0.01	−0.004	−0.06	0.004	-								
Average hours sleep[c]	−0.09	−0.09	0.03	−0.22**	−0.11*	0.07	0.07	-							
Average discrepancy[d]	−0.10	0.07	0.02	0.04	−0.03	−0.10	−0.001	−0.22**	-						
Systolic BP	0.31**	0.09	0.02	0.23**	0.43**	−0.13*	−0.04	−0.09	0.05	-					
Diastolic BP	0.31**	0.11*	0.09	0.10	0.37**	−0.01	−0.09	−0.08	−0.04	0.61**	-				
Glucose	0.13*	0.03	0.04	0.25**	0.25**	−0.06	−0.01	−0.06	0.12*	0.24**	0.18**	-			
Triglycerides	0.20**	0.08	−0.06	0.05	0.32**	−0.04	0.05	−0.14*	0.08	0.13*	0.16**	0.33**	-		
HDL	−0.45**	06	−0.06	0.10	−0.47**	0.03	−0.02	0.03	0.04	−0.16**	−0.24**	−0.23**	−0.46**	-	
Waist/cm	0.63**	0.20**	−0.01	0.05	0.85**	−0.08	−0.14*	−0.09	−0.05	0.47**	0.42**	0.26**	0.34**	−0.54**	-
Metabolic Syndrome[#]	0.19**	0.14*	−0.07	0.02	0.38**	−0.06	−0.01	−0.10	0.11*	0.19**	0.26**	0.40**	0.68**	−0.42**	0.40**

**$P<0.01$, *$P<0.05$, #$P\leq0.07$ correlations of dichotomous variables (gender, ever smoked, minority status) with continuous variables are point-biserial correlations, [a]Average number hours engage in resistance or aerobic exercise/week, [b]Average number servings of fruits or vegetables/day, [c]([Typical weekday sleep×5] + [typical WE sleep×2])/7, [d]Typical WE sleep-typical WN sleep, [#]Symptoms Metabolic Syndrome BP: Blood pressure, HDL: High density lipoprotein, WE: Weekend, WN: Weeknight

Regression modeling indicated that the covariates explained 17% of the variability in number of symptoms of MS. Adding WE/WN change to the model explained an additional 1% of total variability. After controlling for sleep duration, 1 h of difference between WE and WN sleep duration predicted a significant 14% increase in number of symptoms of MS. Average sleep duration was not a significant predictor, suggesting that WE/WN change better predicted risk for symptoms of MS.

Table 3: Tests of moderation and indirect effects

	b	SE	*B*	95% CI
Block 1 R^2=0.17**				
Constant	−1.67*	0.78		−3.20-−0.14
Gender	0.12	0.17	0.04	−0.20-0.45
Age[e]	−0.002	0.01	−0.01	−0.02-0.02
Minority	−0.22	0.14	−0.08	−0.49-0.05
BMI	0.10**	0.02	0.35**	0.07-0.13
Ever smoke	0.21	0.13	−0.09	−0.04-0.46
Exercise[a]	−0.02	0.03	−0.03	−0.08-0.05
Diet[b]	0.02	0.03	0.03	−0.05-0.08
Average daily sleep[c]	−0.09	0.09	−0.05	−0.26-0.09
Block 2 (hypothesis 1) R^2=0.18** $R^2\Delta$=0.01				
Constant	−2.06*	0.79		−3.61-−0.50
Gender	0.16	0.16	0.06	0.16-0.49
Age[e]	−0.001	0.01	−0.01	−0.02-0.02
Minority	−0.23	0.14	−0.09	−0.50-0.04
BMI	0.10**	0.02	0.35**	0.07-0.13
Ever smoke	0.19	0.12	0.08	−0.06-0.44
Exercisea	−0.01	0.03	−0.02	−0.08-0.05
Diet[b]	0.02	0.03	0.03	−0.05-0.08
Average daily sleep[c]	−0.04	0.09	−0.03	−0.22-0.14
WE/WN change[d,e]	0.14*	0.06	0.12*	0.02-0.26
Block 3 (hypothesis 2) R^2=0.18** $R^2\Delta$=0.001				
Constant	−2.04*	0.79		−3.61-−0.49
Gender	0.16	0.17	0.06	−0.16-0.49
Age[5]	−0.001	0.01	−0.01	−0.02-0.02
Minority	−0.22	0.14	−0.09	−0.49-0.04
BMI	0.10**	0.02	0.35**	0.07-0.13
Ever smoke	0.19	0.13	0.08	−0.06-0.44
Exercise[a]	−0.01	0.03	−0.02	−0.08-0.05
Diet[b]	0.02	0.03	0.03	−0.05-0.08
Average daily sleep	−0.04	0.09	−0.02	−0.22-0.14
WE/WN change[d,e]	0.14*	0.06	0.12*	0.02-0.27
WE/WN change×Age	0.01	0.01	0.03	−0.01-0.02

*P<0.05,**P<0.001. [a]Average number hours engage in resistance or aerobic exercise/week, [b]Average number servings of fruits or vegetables/day, [c]([Typical weekday sleep×5] + [Typical WE sleep×2])/7, [d]WE/WN change=Typical WE sleep−typical WN sleep, [e]Centered values used for test of moderation, N=315, CI: Confidence interval, BMI: Body mass index, WE: Weekend, WN: Weeknight

The second hypothesis was that variance accounted for by WE/WN change would be moderated by age. This hypothesis was not supported. The interaction explained an additional .1% of the variability; but the association of risk for MS with WE/WN change was consistent across age within this sample.

Hypotheses 3-7 predicted indirect effects of WE/WN change through each diagnostic criterion of MS when all potential indirect pathways were entered simultaneously [Table 4, Figure 1]. Although diastolic BP, glucose, and triglycerides were significant main effect predictors in the full model, a path model indicated an indirect effect of WE/WN change through glucose level only. Approximately half of variability explained by WE/WN change was direct, and half indirect.

DISCUSSION

Small, frequent changes in sleep duration predicted number of symptoms of MS independently of average sleep duration, and the association did not vary with age. The difference in sleep duration appeared to have an indirect effect through glucose when all predictors of MS were simultaneously considered as potential indirect paths. These findings made several contributions to the literature. WE make-up sleep appeared to be commonly practiced among these business leaders across age. The finding that over half of this adult sample had a discrepancy >1 h was similar to Roenneberg *et al.*[16]. However, Roenneberg *et al.* found that the difference decreased across adulthood, whereas no age-related decline was evident here. Similarly, the association of WE/WN change with symptoms of MS did not change with age; greater discrepancy was a risk factor regardless of age. Age may not be the most meaningful indicator of change

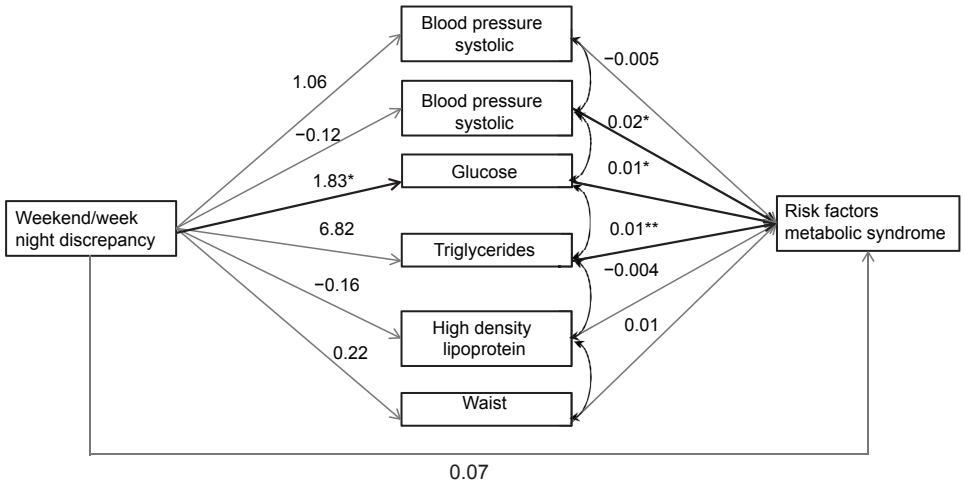

Figure 1: Hypotheses 3-7: Path analysis showing unstandardized coefficients

Table 4: Hypotheses 3-7: Unstandardized direct and indirect effects of WE/WN change

Weekend/weeknight discrepancy	Metabolic syndrome						Number symptoms
	Systolic BP	Diastolic BP	Glucose	Triglycerides	High density lipoprotein	Waist	
Direct	1.06	−0.12	1.83*	6.82	−0.16	0.22	0.07
Indirect	−0.01	−0.002	0.02*	0.05	0.001	0.002	0.07
Total	1.05	−0.122	1.85	6.87	−0.161	0.222	0.14*

Predictor variables were controlled for age, minority status, BMI, smoking, exercise and diet, indirect effects confirmed by bootstrapping; $N=315$, $R^2=0.55$**, *$P<.05$, **$P<.01$, BMI: Body mass index, BP: Blood pressure, WE: Weekend, WN: Weeknight

over time. Previously reported age-related associations between shift-work and symptoms of MS may reflect extended exposure to shift-work rather than aging, per se [38]. It is unknown whether there is a cumulative effect of WE/WN change on health. The present data did not indicate how long the sleep pattern had been maintained, suggesting the need to distinguish the roles of age and length of exposure.

The present data cannot yield causal explanations for why such a small shift in sleep duration should predict measurable differences in health risk, but possible mechanisms are suggested in the literature. Extended WE sleep may alter eating patterns (e.g. delaying or eliminating some meals), thereby desynchronizing metabolic processes. In rats, being forced to eat during a normal rest time interfered with the functioning of clock genes in the liver and genes associated with metabolism [39]. In humans, forced desynchronization by about 12 h resulted in decreased leptin (which signals satiety) and increased glucose despite increased insulin, as well as changes in cortisol levels, arterial BP, and sleep efficiency [7]. The slight WE/WN change examined here may have altered perception of hunger, or type or amount of food consumed. Shift-workers have reported different dietary patterns on work days and rest days [40]. The present study controlled for number of servings of fruits and vegetables typically consumed/day, but did not address the type, amount or timing of food.

When controlling for overlap of the individual symptoms of MS, WE/WN change had an indirect effect through glucose but not through other symptoms. The finding that chronic desynchronization of sleep duration has an indirect effect through glucose is not, by itself, surprising. Most other studies, however, have looked at large circadian shifts, whereas the present study looked at small, chronic, commonly-practiced changes. Even small changes may be enough to disrupt glucose metabolism. The other indices of MS did not provide indirect routes. The simplest possibility is that glucose may be the most responsive of these mechanisms to small but chronic WE/WN change in sleep duration. Because the present data don't allow for such a strong conclusion, other explanations must be considered. Short sleep duration can predict total cholesterol, HDL cholesterol, triglycerides and BP [41]. Because average sleep was entered as a covariate, associations via HDL, triclycerides, or BP may have been obscured. The absence of an indirect effect via HDL may have been due to controlling for age. Karlsson *et al*. [4] found lowered HDL cholesterol only in younger shift-workers. The lack of an indirect effect via triglycerides may be related to recent alcohol consumption, especially in combination with diet. Alcohol alters the synthesis of triglycerides in the liver [42]. Recent alcohol consumption was not reported, so this could have been a confound. Although short sleep duration predicts hypertension [43], shift-work may [44] or may not [22] predict BP levels or patterns. Likewise, one study reported BP surged on Monday mornings (after a WE away from work), but it was not clear whether this reflected the change in sleep, increased stress upon returning to work, or some other factor [45]. The present study recorded BP levels in the morning, whereas changes in the circadian-driven rise and fall of BP over 24-h may be a better predictor [46]. However, a diagnosis of MS utilizes a static measure of BP, making this an appropriate measure in this context. Use of BMI as a covariate likely accounted for much of the variability due to waist size.

These findings could reflect interconnections between sleep and emotional regulation, stress, anxiety, depression [47] or feelings of general well-being [48]. The WE/WN change may have played less of a role in health outcomes than underlying psychosocial factors [49]. For example, longer WE sleep may have been a symptom of depression or a method of coping with stress. Frequent shifts in sleep duration may increase allostatic load (cumulative physiological changes in response to stress [50] such as change in salivary cortisol response upon awakening [51]). Change in WE/WN duration may improve the ability to cope with stress (with downstream benefits to health) by decreasing overall sleep debt, or it may be counterproductive, or at least insufficient, to overcome lowered ability to respond to stress.

Although associations of symptoms of MS with sleep duration or exercise did not emerge in this sample, this does not suggest they are unimportant. Sleep duration reported here fell into the mid-range of typical patterns in this age group [52], with few participants indicating extremely short or long sleep hours. With a greater range of sleep duration, a larger association might have emerged. Furthermore, some work has suggested that total sleep per 24-h periods should be measured instead of typical nighttime sleep, as napping may be protective [53]. Changing sleep patterns may be part of a larger set of health behaviors [54]. Reports of younger samples indicate that adolescents with an evening chronotype (who may increase WE sleep) [55] were less likely to eat breakfast [56], and children who consistently slept more had a higher level of physical activity [57]. The present study controlled for frequency (but not intensity) of exercise, consumption of fruits and vegetables (but not other dietary choices) and smoking, but not all potentially harmful behaviors were modeled. A combination of unhealthy lifestyle factors may have been more predictive than WE/WN shifts in sleep patterns alone. Although the present data did not address this question,

the findings suggest that interventions to minimize risk for MS should include both sleep duration and consistency, along with other health behaviors.

Physical measures utilized here were objective, collected by qualified health professionals, and therefore credible. Several limitations should be kept in mind when interpreting these data. Significant effect sizes were small. Sleep duration but not quality was reported. A number of studies of other populations have suggested that sleep quality may be an important consideration [58]. Health behaviors were self-reported, with all the limitations of such data. However, the sleep times reported here are similar to those reported elsewhere [59]. Self-reported sleep duration may be moderately correlated with objective measures [60]. The data did not include typical bed or awakening times, preventing identification of changes in the timing of sleep. Furthermore, some of the participants might have had a sleep disorder. Sleep apnea is recognized as a risk factor for obesity and MS [61]. Use of prescription drugs could have masked a potential risk factor; therefore symptoms of MS may have been underestimated [1]. While inclusion of only high-level executives in this sample limited the generalizability of the findings, it also expanded the study of sleep in a population under-reported in the literature. Participants represented diverse types of businesses, but were predominantly male and Caucasian. Sleep patterns, values, and views about sleep/work trade-off may be culture-specific [62]. Therefore, the findings may not apply to business leaders in other cultures.

The present work was exploratory, and findings need to be substantiated by examining multiple sleep parameters using objective measures. Although these findings suggest weekly change in sleep duration is more of a health liability (or at least an inadequate solution) than boon in this population, additional research is needed to confirm this, identify underlying mechanisms, and determine whether the findings generalize to other occupational roles. If the findings are robust, then inquiry is needed to determine whether reducing WE/WN change improves symptoms of MS. While not conclusive, these findings suggest that this dimension of sleep may be relevant to health risks and should be considered separately from average duration.

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