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Temporal Associations between Nightly Sleep with Daytime Eating and Activity Levels in Free-Living Young Adults

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ABSTRACT

STUDY OBJECTIVES: This study aimed to quantify the temporal associations between nightly sleep quantity and timing with daytime eating behavior and activity levels in free-living (i.e., non-experimental) settings.

METHODS: Generally healthy young adults (N=63; 28.9±7.1 years) completed concurrent sleep (wrist actigraphy), eating (photo-assisted diet records), and activity (waist actigraphy) assessments over 14 days. Multilevel models quantified the associations between nightly sleep (total sleep time, timing of sleep and wake onset) with next-day eating behavior (diet quality, caloric intake, timing of eating onset/offset, eating window duration) and activity levels (total physical activity, sedentary time). Associations in the reverse direction (i.e., eating and activity predicting sleep) were explored. Models adjusted for demographic and behavioral confounders and accounted for multiple testing.

RESULTS: At within- and between-subject levels, nights with greater-than-average total sleep time predicted a shorter eating window the next day (all $p \leq 0.002$). Later-than-average sleep and wake timing predicted within- and between-subject delays in next-day eating onset and offset, and between-subject reductions in diet quality and caloric intake (all $p \leq 0.008$). At within- and between-subject levels, total sleep time was bidirectionally, inversely associated with sedentary time (all $p < 0.001$), while later-than-average sleep and wake timing predicted lower next-day physical activity (all $p \leq 0.008$).

CONCLUSIONS: These data underscore the complex interrelatedness between sleep, eating behavior, and activity levels in free-living settings. Findings also suggest that sleep exerts a greater influence on next-day behavior, rather than vice versa. While testing in more diverse samples is needed, these data have potential to enhance health behavior interventions and maximize health outcomes.

KEY WORDS: Actigraphy; Diet; Eating Timing; Physical Activity; Sedentary Behavior; Sleep Duration; Sleep Timing

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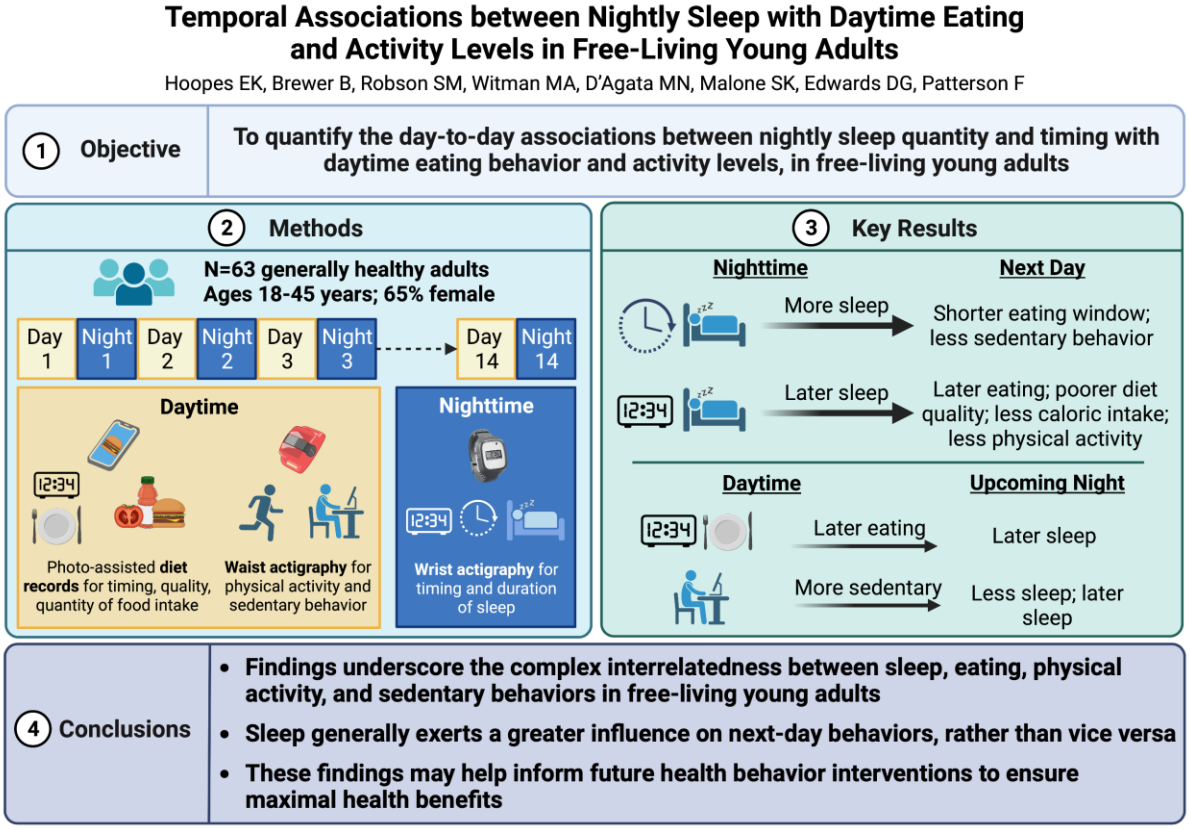
Graphical abstract



STATEMENT OF SIGNIFICANCE

In this micro-longitudinal study, we utilize 14 days of actigraphy and photo-assisted diet records to newly illustrate the temporal, day-to-day associations between nightly sleep (quantity, timing) with daytime eating behavior (eating timing, energy intake, diet quality) and activity levels (sedentary behavior, total physical activity) in free-living young adults. Our findings underscore the complex interrelatedness between sleep, eating, sedentary, and physical activity behaviors, while suggesting that sleep generally exerts a greater influence on next-day behaviors, rather than vice versa. These data converge with available experimental evidence investigating the relationships between these daily behaviors and extend this work into free-living settings. This information can help inform the design of health behavior interventions to ensure maximal health benefits.

INTRODUCTION

Sleep is a widely recognized determinant of chronic disease risk. Adults who do not obtain the recommended ≥7 hours of sleep per night experience a 22% higher risk of coronary artery disease [1], 18% higher risk of diabetes mellitus [2], and are 13% more likely to suffer premature mortality from all causes [1], when compared to those who obtain an adequate amount of sleep. Moreover, growing evidence shows that extending sleep duration in short sleepers can result in a variety of health benefits ranging from lowered blood pressure and weight loss [3,4] to improved cognition and mood [5-7]. Observations such as these, along with estimates that nearly 40% of U.S. adults do not meet sleep duration recommendations [8], have prompted an increased emphasis on optimizing sleep duration to prevent major chronic diseases of aging [9].

Experimental studies have demonstrated that alterations in sleep duration can impact other daily health behaviors, including diet and physical activity. For instance, sleep-restricted adults exhibit obesogenic behaviors including increased energy intake [10], poorer dietary choices [11-13], later timing of caloric consumption [11], and reduced overall activity levels [14-16]. Meanwhile, early evidence in adults suggests that sleep extension may reduce energy intake [17] and improve diet quality [18,19], while adolescent studies have observed favorable reductions in sedentary behavior [20,21]. Evaluating the impact of sleep duration on subsequent eating behavior and activity level in free-living (i.e., non-experimental) contexts could enhance the generalizability of this experimental evidence, given that field research may capture individuals in a greater variety of situations or environments that are more representative of habitual experiences [22]. Ecologically valid evidence of these associations is also important given that current behavioral interventions tend to fixate on isolated targets (e.g., extending sleep duration), with little consideration to

the impact of these behavioral changes on other health behaviors, and potential cumulative health effects.

Observational micro-longitudinal studies evaluate behaviors in natural, real-world settings, over several consecutive days, providing a unique opportunity to examine the temporal interrelationships among daily behaviors in ecologically valid contexts [23]. Micro-longitudinal designs have been increasingly leveraged to quantify the day-to-day associations between sleep and activity patterns in a range of populations. However, a recent meta-analysis of such studies emphasized that studies utilizing longer monitoring protocols (i.e., ≥12 days) under naturalistic conditions are needed to disentangle the bidirectional associations between sleep and physical activity [24]. Moreover, very little is known about the temporal, day-to-day associations between sleep and next-day dietary intake in adults within free-living settings. Another drawback of prior research is the limited attention to timing of behavior, despite evidence that the time at which an individual engages in sleep or eating may play a meaningful role in chronic disease development. Indeed, bedtimes later than 11pm are seen in 40-80% of adults [25-27], are independently associated with cardiometabolic disease risk factors [28,29], and have been shown to predict overt cardiovascular disease development [25,27], when compared to those with earlier bedtimes. Similarly, late-night eating is associated with risk factors such as obesity [30] and hypertension [31], while eliminating food intake at night has been shown to improve these risk factors [32-35]. Given the pervasiveness of poorly timed behaviors in today's 24-hour society [36], identifying ecologically valid targets that promote more optimal timing of sleeping and eating could have a considerable impact on improving the public's health.

To begin addressing these gaps, we utilized a 14-day micro-longitudinal design to comprehensively characterize multiple dimensions of sleep (i.e., quantity, timing), eating

behavior (i.e., diet quantity, energy intake, eating timing), and activity level (i.e., time spent sedentary and physically active) in free-living, non-shift working adults. Our primary aim was to quantify the temporal associations between nightly sleep with next-day eating behaviors and activity levels. Secondarily, we explored these associations in the reverse direction, to determine the extent to which daytime eating behavior and activity level predicted sleep metrics for the upcoming night.

METHODS

Study Participants

This study was approved by the Institutional Review Board at the University of Delaware and was conducted in accordance with the ethical standards of the Declaration of Helsinki. Participants were recruited using various strategies including printed flyers and mass-media advertisements targeting Newark, Delaware and surrounding regions. All participants provided written informed consent prior to participation. Participants included men and women between the ages of 18 and 45 years. As these data were collected as part of a larger study focused on cardiometabolic health outcomes, individuals were excluded for the following: a history of any major chronic diseases or conditions, including cardiovascular, renal, metabolic, autoimmune, or cancerous conditions; a recent history of COVID-19 infection (<60 days) or vaccination (<14 days); diagnosis of a sleep disorder (e.g., insomnia, restless leg syndrome, sleep apnea) or at high-risk for a sleep disorder (Insomnia Severity Index score >14 [37]; STOP-bang score ≥3 [38]); seated resting blood pressure >140/90 mmHg; body mass index <18.5 or >35 kg/m^2; use of any supplements or medications for sleep; diagnosis of depression, or at risk for depression (CES-D score ≥16 [39]); current night-shift work; use of any medications that significantly alter cardiovascular physiology; women who were currently pregnant or breastfeeding; women who reported being perimenopausal or post-menopausal; suspected alcohol use disorder (AUDIT score ≥15 [40]); current tobacco use (≥1 cigarette in the last month); engagement in weight-loss (e.g.,

calorie-restricted diet) or an extended fasting regimen; unstable body weight (>4.5 kg/~10 lbs. change) over last 3-months; or lack of regular access to a smart phone.

Sleep Metrics

Sleep metrics were estimated using a wrist accelerometer (Actiwatch Spectrum Plus; Philips-Respironics, Inc.), worn continuously on the non-dominant wrist, in conjunction with a standardized sleep diary [41]. As part of the larger study protocol, participants were asked to wear wrist accelerometers until they completed all study procedures, which could range from 14-21 days, depending on participant scheduling. Data were collected in 30-second epochs and processed using Philips Actiware software (version 6.1.0). Nights with >1 hour of missing data were considered invalid [42]. Rest intervals were identified following a standardized protocol that incorporates sleep diaries, activity level, and "lights out" [42,43]. Sleep-wake scoring was based on the medium threshold setting for sleep/wake detection using the algorithm provided by the manufacturer. Actiwatch accelerometers have been validated against polysomnography, the gold standard method of sleep assessment (sensitivity>0.90, accuracy>0.80) [44,45]. Metrics of nightly *total sleep time* (time scored as "sleep" between sleep onset and wake onset, in minutes), *sleep onset* (clock time at sleep onset), and *wake onset* (clock time at awakening), were generated for each night of wear.

Eating Behavior Metrics

Time-stamped, photo-assisted 24-hour diet records were collected over 14 consecutive days that overlapped with sleep monitoring. Diet records were obtained via a smartphone application ('MealLogger,' Wellness Foundry) and were immediately available to research staff via the internet. Diet records were reviewed by research staff for

completeness and accuracy with questions answered by messaging with participants directly within the smartphone application, direct text messages to participants (as needed), and an additional thorough review with participants via scheduled phone calls at the end of days 1 and 8, as well as an in-person review immediately upon completion of the monitoring period on the morning of day 15. Diet records were analyzed using the Nutrition Data System for Research software (University of Minnesota, Minneapolis, MN). All diet records were quality checked after entry into NDSR by a Registered Dietitian or by research staff under the direction of the Registered Dietitian. Diet data were used to determine *total caloric intake* (kcals/day) and *diet quality* (Healthy Eating Index-2015 score [46]; scores range from 0 to 100, with a score of 100 reflecting those whose diet aligns with key dietary recommendations from the *Dietary Guidelines for Americans*) for each day. Additionally, daily metrics of eating timing, including *eating onset* (timing of first caloric intake after awakening), *eating offset* (timing of last caloric intake before sleep), and *eating window duration* (time elapsed between eating onset and offset), were computed.

Activity Level Metrics

Metrics of physical activity and sedentary behavior were estimated via waist-worn accelerometry [47] (ActiGraph wGT3X+, ActiGraph, LLC) during all waking hours for 14 consecutive days that overlapped with sleep and dietary monitoring. Data were collected in 10-s epochs and processed using ActiLife software (version 6.13.4). Wear time was first validated using the Troiano algorithm [48], and periods of non-wear were removed from analyses. Activity intensity was estimated using activity counts per minute (CPM) according to the Freedson Combination 1998 algorithm, such that sedentary behavior was categorized as 0-99 CPM, light physical activity (LPA) as 100-1951 CPM, and moderate-vigorous physical activity (MVPA) as >1951 CPM [49]. A minimum of 10 hours of wear-time was required to be considered a valid day [47]. Daily metrics of *sedentary time* (0-99 counts per

minute) and *total physical activity* (>99 counts per minute) were expressed in two ways: (1) the total number of minutes per day, and (2) the percentage of total time spent awake that day.

Covariates

Age (in years), sex, and body mass index (BMI) were obtained at screening. For BMI, height was assessed using a wall-mounted stadiometer, and weight using a calibrated scale, then BMI was calculated (weight in kilograms divided by height in m^2). These covariates were selected on a theoretical basis, as each of these variables have an established association with lifestyle behaviors and may also influence the interrelatedness among these behaviors [50-52]. Daily data were also coded based on whether they reflected a weekday or weekend day (binary variable; Saturdays/Sundays were coded as weekend days, while Monday through Friday were coded as weekdays), which was also included as a covariate in all models.

Statistical Analyses

Time-of-day variables were converted to a military time decimal representation and midnight-centered when the construct of interest spanned the overnight hours (e.g., sleep onset) such that a numerical average would accurately reflect the true point of centrality. Diet and activity data were temporally aligned such that they corresponded to a given wake period rather than a given calendar day. For instance, an individual who had a light snack at 12:30 AM before going to bed at 1:00 AM had their recorded calories “moved” to the wake period associated with the previous calendar day. Similar action was taken for physical activity recorded after midnight but before sleep onset. Following data cleaning and reparameterization, summary statistics were generated for all variables of interest – means

and standard deviations for continuous variables, and frequencies and percentages for categorical variables. Time-of-day variables are presented as clock time to aid in interpretation.

To assess the relationship between within-subject nightly sleep metrics and next-day behaviors (i.e., eating and activity), all modeled variables were first mean-centered relative to each individual's average value. Mean-centered data were then used to fit multiple generalized linear models (GLMs) utilizing generalized estimating equations (GEEs). This modeling strategy served as a natural fit for our nuanced analysis given that the research question aimed to determine the population-averaged effect of within-subject deviations on the stated outcomes. Both the m-dependent and autoregressive working correlation structures were used with the data acting as a guide to determine which was more appropriate for each model. Given that beta coefficients estimated using GEEs possess many desirable statistical properties even in the event of the working correlation matrix's misspecification, the data-driven strategy used is sufficient to produce robust conclusions regarding the population under study. Between-subject analyses made use of the same modeling process and working correlation matrices; however, all variables under study were mean-centered relative to the full-sample mean before modeling. All models adjusted for age, sex, BMI, and weekday/weekend status. Additionally, in models where timing of sleep onset or wake onset were the independent variable, deviation from average total sleep time was adjusted for, to identify the role of sleep or wake timing independent of possible concomitant changes in sleep quantity.

Data cleaning and summary statistic generation were performed in R v. 4.1.3. All within- and between-subject models were fit using SAS v. 9.4. The Benjamini-Hochberg procedure was used to account for multiple testing and control the false discovery rate to

α=0.05. For our set of model p-values, this corresponded to an "individual model" significance threshold of α=0.017.

RESULTS

Participant Characteristics

The analytic sample consisted of N=63 adults who were aged 28.9 ± 7.1 years, 65.1% female, with a mean BMI of 24.3 ± 3.1 kg/m^2. In terms of race/ethnicity, the sample was predominantly non-Hispanic White (66.7%), but also consisted of participants who identified as Asian (11.1%), Black (7.9%), Hispanic (9.5%), and other races (4.8%). All participant characteristics, including average sleep, eating behavior, physical activity, and sedentary behavior metrics, are provided in **Table 1**.

Data Availability and Compliance

Across all participants, the dataset comprised 1,045 nights of sleep, 874 days of diet, and 863 days of activity data. An average of 16.6 ± 2.3 nights of sleep data, 13.9 ± 0.5 days of dietary data, and 13.7 ± 0.9 days of activity data were obtained per participant. Multilevel models examining the temporal associations between nighttime sleep with next-day diet and activity patterns were generated from a mean of 9.6 ± 2.6 days of paired data per participant, while models exploring temporal associations between daytime diet and activity patterns with same-night sleep were generated from a mean of 11.3 ± 1.8 days of paired data per participant.

Associations Between Total Sleep Time with Eating Behavior and Activity Level

Results for models testing the associations between total sleep time with eating behavior and activity level are summarized in **Figure 1** (complete results can be found in **Supplemental Table 1**). At both within- and between-subject levels, nights with greater-

than-average total sleep time predicted a shorter eating window the following day (within: B=0.23 ± 0.08 hours, p=0.002; between: B=-0.50 ± 0.09 hours, p<0.001). At the between-subject level, greater-than-average total sleep time also predicted a later next-day eating onset (B=0.41 ± 0.07 hours, p<0.001) and an earlier next-day eating offset (B=-0.12 ± 0.05 hours, p=0.011). At both within- and between-subject levels, total sleep time was inversely associated with next-day sedentary time (within: B=-20.8 ± 2.6 minutes, p<0.001; between: B=-32.8 ± 3.6 minutes, p<0.001) and time spent in physical activity (within: B=-5.6 ± 2.3 minutes, p=0.016; between: B=-6.1 ± 2.4 minutes, p=0.011). In exploratory post hoc models, further characterizing physical activity according to intensity dampened the magnitude of these associations, as total sleep time did not significantly predict next-day LPA or MVPA at within- or between-subject levels.

At the within-subject level, every 1-hour increase in the duration of the eating window predicted 3.0 ± 1.2 minutes less sleep that night (p=0.015). Additionally, at the within-subject level, every 60-minute increase in time spent sedentary was associated with 11.4 ± 2.2 less total sleep time that night (p<0.001). At the between-subject level, each 1-hour delay in eating offset was associated with 5.0 ± 1.6 less minutes of sleep that night (p=0.002), while each 60-minute increase in time spent sedentary was associated with 12.3 ± 2.5 less minutes of total sleep time that night (p<0.001).

Associations Between Sleep Onset Timing with Eating Behavior and Activity Level

Results for models testing the associations between sleep onset timing with eating behavior and activity level are summarized in **Figure 2** (complete results can be found in **Supplemental Table 2**). At the within-subject level, each 1-hour delay in sleep onset timing predicted a 0.77 ± 0.11-hour later timing of eating onset (p<0.001) and a 0.62 ± 0.10-hour

shorter eating window (p<0.001) the following day. These associations were similarly observed at the between-subject level (eating onset: B=1.00 ± 0.09 hours, p<0.001; eating window: B=-0.72 ± 0.11 hours, p<0.001). In addition, at the between-subject level, later-than-average timing of sleep onset predicted a significant delay in next-day eating offset timing (B=0.34 ± 0.08 hours, p<0.001), a significant reduction in HEI score (B=-1.9 ± 0.7, p=0.007), and a significant reduction in total caloric intake (B=-98.7 ± 37.3 kcals, p=0.008). At both within- and between-subject levels, later-than-average timing of sleep onset predicted a significantly higher percentage of sedentary behavior (within: B=0.8 ± 0.3, p=0.003; between: B=0.9 ± 0.3, p=0.001), lower percentage of physical activity (within: B=-0.8 ± 0.3, p=0.003; between: B=-0.9 ± 0.3, p=0.001), and less minutes of physical activity the next day (within: B=-11.2 ± 2.8, p<0.008; between: B=-13.8 ± 2.7, p<0.001). Exploratory post hoc analyses revealed that associations between later sleep onset and less time spent in physical activity the following day were due to reductions in next-day LPA when expressed as minutes (within: B=-8.0 ± 1.9, p<0.001; between: B=-10.9 ± 2.1, p<0.001) and as a percentage of wake time (within: B=-0.6 ± 0.2, p<0.001; between: B=-0.8 ± 0.2, p=0.001).

At the within-subject level, a delay in the timing of eating onset and eating offset predicted significantly later sleep onset that night, of comparable magnitude (eating onset: B=0.15 ± 0.04 hours, p<0.001; eating offset: B=0.12 ± 0.03 hours, p<0.001). Additionally, every 60-minute increase in sedentary time was associated with a 0.18 ± 0.04-hour delay in sleep onset timing that night (p<0.001). When expressed as a percentage of total daily activity, each 10% increase in sedentary time was associated with a 0.26 ± 0.07-hour delay in sleep onset (p<0.001), while a 10% increase in total physical activity was associated with a comparable advance in sleep onset that night (B=0.26 ± 0.07, p<0.001). Each of these associations were also observed at the between-subject level. Exploratory post hoc analyses revealed that the association between higher-than-average physical activity and earlier sleep

onset that night was driven by increases in the proportion of time spent in LPA (within: B=-0.24 ± 0.07 hours per 10% increase in LPA, p=0.002; between: B=-0.28 ± 0.07 hours per 10% increase in LPA, p<0.001).

Associations Between Wake Onset Timing with Eating Behavior and Activity Level

Results for models testing the associations between wake onset timing with eating behavior and activity level are summarized in **Figure 3** (complete results can be found in **Supplemental Table 3**). At both within- and between-subject levels, later-than-average wake onset timing predicted significantly later eating onset (within: B=0.79 ± 0.11 hours, p<0.001; between: B=1.05 ± 0.08 hours, p<0.001) and a shorter eating window (within: B=-0.66 ± 0.10 hours, p<0.001; between: B=-0.77 ± 0.11 hours, p<0.001). Additionally, at the between-subject level only, each 1-hour delay in wake onset predicted a 0.31 ± 0.07-hour delay in eating offset that day (p<0.001), as well as a 2.0 ± 0.7 reduction in HEI score (p=0.002) and 121.1 ± 39.6 reduction in total caloric intake that day (p=0.002). At both within- and between-subject levels, later-than-average wake onset timing predicted a significantly higher percentage of sedentary behavior (within: B=0.7 ± 0.3 %, p=0.008; between: B=0.7 ± 0.3 %, p=0.007), lower percentage of physical activity (within: B=-0.7 ± 0.3 %, p=0.008; between: B=-0.8 ± 0.3 %, p=0.007), and less minutes of physical activity that day (within: B=-10.6 ± 2.8 minutes, p<0.001; between: B=-12.7 ± 2.8 minutes, p<0.001). Exploratory post hoc analyses revealed that the associations between later-than-average wake onset and less physical activity that day were due to reductions in daytime LPA when expressed as minutes (within: B=-7.9 ± 2.0, p<0.001; between: B=-10.0 ± 2.1, p<0.001) and as a percentage of wake time (within: B=-0.6 ± 0.2, p=0.003; between: B=-0.6 ± 0.2, p=0.008).

At both within- and between-subject levels, later-than-average eating onset timing predicted significantly later wake onset the next day (within: B=0.19 ± 0.05 hours, p<0.001; between: B=0.27 ± 0.05 hours, p<0.001). Additionally, a longer-than-average eating window predicted significantly earlier wake onset timing the next day (within: B=-0.08 ± 0.03 hours, p=0.004; between: B=-0.10 ± 0.03 hours, p<0.001). At the between-subject level only, each 1-hour delay in eating offset predicted a 0.08 ± 0.03-hour delay in wake onset the next day (p=0.006), while each 60-minute increase in total physical activity predicted a 0.13 ± 0.05-hour advance in wake onset the next day (p=0.009). Exploratory post hoc analyses revealed that an increase in the proportion of time spent in LPA was associated with a significant advance in wake onset the next day (within: B=-0.23 ± 0.08 hours per 10% increase in LPA, p=0.005; between: B=-0.26 ± 0.07 hours per 10% increase in LPA, p<0.001).

DISCUSSION

In this 14-day micro-longitudinal study, we comprehensively characterize the temporal, day-to-day associations between sleep, eating behaviors, and activity levels in free-living young adults. Two key and novel findings related to sleep and eating behaviors identified by this study are: (1) nights with more sleep predicted a significantly shortened eating window the next day; and (2) nights with later sleep and wake timing predicted notable delays in next-day eating onset and offset, as well as between-subject reductions in next-day diet quality and quantity. Our rigorous study design also enabled us to shed new light on the temporal associations between nightly sleep and daytime activity levels, highlighting that: (1) nightly total sleep time was bidirectionally and inversely associated with the amount of time spent sedentary during the day; and (2) nights with later sleep or wake timing predicted significant reductions in next-day physical activity. Collectively, these data underscore the complex interrelationships between nightly sleep with daytime eating and activity as they are experienced in naturalistic settings.

Our findings suggest that longer-than-average sleep duration may elicit a condensed daytime eating window, which has been increasingly marketed as a plausible strategy to improve cardiometabolic health in adulthood [53]. We found that each 60-minute increase in total sleep time predicted a ~15-30-minute shorter eating window the following day at both within- and between-subject levels. This association was also apparent in the reverse direction, although only at the within-subject level, and at a smaller magnitude. In a meta-analysis of interventions that reduced participants' eating windows to 4-12-hour periods, it was confirmed that this behavioral strategy can result in small but significant improvements in several cardiometabolic parameters, including weight loss, reduced fasting glucose, and lowered blood pressure [32]. Interestingly, results of observational studies are less consistent. For instance, in cross-sectional epidemiologic studies of midlife women, longer nightly fasting durations (i.e., shorter eating windows) have been independently associated with lower hemoglobin A_{1c} values [54,55]. Additionally, a prospective analysis in a large sample of mostly healthy men and women found those with prolonged overnight fasting (≥18 hours) exhibited significantly reduced BMI when compared to those with shorter fasts (12-17 hours) [56]. By contrast, others have reported higher circulating insulin and C-reactive protein concentrations [57], a higher risk of obesity [58], and poorer overall cardiovascular health [59] in those with shorter eating windows. Given these mixed findings, our results emphasize the importance of considering the impact of sleep extension on subsequent eating windows, and the effect that this concomitant behavior change might have on anticipated health benefits of sleep promotion.

One of the most striking results of our analysis was the effect that later sleep timing has on delaying next-day eating timing, in addition to the similar impact that eating timing has on subsequent sleep timing. While this finding appears intuitive, empirical evidence to

support the impact that sleep timing and eating timing impose on each other is surprisingly limited, and the magnitude of these associations are not well characterized. We found that each 60-minute delay in sleep or wake onset predicted a ~40-60-minute delay in next-day eating onset at both within- and between-subject levels, and a ~20-minute delay in next-day eating offset at the between-subject level. A later timing of eating onset and offset also tended to predict a later timing of sleep and wake onset that night, but to a smaller degree. Later timing of sleep (e.g., late bedtimes) and eating (e.g., large meals in the evening/at night) have been increasingly linked with various adverse cardiometabolic health outcomes [25,27,29,31], while an accumulating number of interventions have modified eating timing to mitigate these cardiometabolic consequences [32]. However, most interventions have not fully considered the concurrent influence of sleep timing, despite evidence that poorly-timed sleep episodes and late-day caloric consumption have been linked with similar cardiometabolic risks [60]. Given that the impact of sleep timing on eating timing appears greater than vice versa, our data suggest that intervening on sleep timing *concomitantly* with eating timing may enhance sustainability and optimize the efficacy of behavior change strategies that have traditionally targeted eating timing alone (e.g., time-restricted eating).

At the between-subject level, caloric intake was significantly reduced on days following nights with later-than-average sleep or wake onset. It is possible that avoidance of healthier foods may have contributed to the observed caloric deficit, as significantly poorer diet quality (i.e., lower HEI score) was also observed on days following nights with later-than-average sleep and wake timing. Our finding might be viewed as divergent with experimental research, as studies in both children and adults have shown that daily caloric intake significantly increases when bedtimes are delayed, likely due to greater caloric consumption during evening hours [11,61]. However, prior experimental studies are difficult to compare to the current analysis, as they have simultaneously induced sleep loss ranging from ~1.5-4.5 hours, whereas the current analysis adjusted for any deviation from average sleep quantity.

Of note is that our findings do align with cross-sectional observations that adults with a preference for later sleep/wake timing ('evening chronotypes') exhibit poorer diet quality than those with an earlier preference 'morning chronotypes'), independent of sleep duration [62,63]. Those who report later bedtimes have similarly been shown to consume poorer quality diets than those with earlier bedtimes, irrespective of sleep quantity [64,65]. Our data add to this literature by suggesting that staying awake or waking up later than usual may increase the likelihood of making modestly poorer dietary choices the following day. To our knowledge, this is the first observational study in adults to report on the temporal associations between sleep and wake timing with quantity and quality of dietary intake in non-experimental settings, warranting further investigation in larger and more diverse samples.

Our finding that each 60-minute increase in total sleep time predicted ~20-30 minutes of less sedentary time the next day is similar to results from several micro-longitudinal studies in mostly middle-aged and older adult samples [66-69]. This association also converges with results of experimental studies that have examined sedentary behavior following manipulation of sleep duration. For instance, a randomized crossover trial in young adults of comparable age to the current study found that restricting sleep by ~2 hours per night for 14 nights resulted in significantly increased sedentary time (+21 min/day) [15]. Additionally, sleep extension interventions that have increased sleep quantity by an average of ~1.5-3 hours have been found to reduce sedentary time in adolescent and adult samples [20,70]. We also found that higher-than-average total sleep time predicted a small but significant reduction in total physical activity the following day; however, exploratory post hoc analyses rendered this association non-significant when next-day activity was characterized according to intensity. While results of prior micro-longitudinal studies are mixed [24], many have reported inverse associations between sleep quantity and next-day physical activity [71-73]. On the other hand, experimentally increasing the sleep opportunity by ~1-3 hours

has not produced meaningful changes in subsequent physical activity levels in adults or adolescents [18,20,70]. Meanwhile, the impact of experimental sleep loss has been less clear, with some studies reporting reduced activity levels [15,74], while others have found increases [16], likely as a function of greater time spent awake. Collectively, this body of literature underscores the complex relationship between sleep quantity and physical activity, which varies widely according to study design. Nevertheless, the current findings suggest that health professionals should make a concerted effort to ensure that promotion of increased sleep quantity preferentially displaces sedentary time rather than physical activity.

When examining the impact of activity level on subsequent sleep quantity, we found that higher sedentary time, but not physical activity, predicted less sleep the upcoming night. However, this association was smaller in magnitude than what was observed in the reverse direction. Importantly, prior evidence does support the notion that sedentary behavior may have a negative impact on sleep health. A meta-analysis of 16 studies demonstrated that higher amounts of sedentary behavior are associated with 1.18 and 1.38 higher odds of insomnia and sleep disturbances, respectively [75]. An important consideration is that this meta-analysis only consisted of cross-sectional and longitudinal studies due to a lack of randomized controlled trials on the subject. Still, this finding has important implications for future interventions, given the considerable focus on increasing physical activity [76], and a less pronounced emphasis on reducing sedentariness [75], to extend sleep duration.

Later-than-average timing of sleep and wake onset predicted a small but significant reduction in the percentage of time spent physically active, and inadvertently, a higher percentage of time spent sedentary the next day, which was observed at both within- and between-subject levels. Each 60-minute delay in the timing of sleep or wake onset also predicted ~10-14 minutes less total physical activity the next day, which appeared to be

predominantly driven by reductions in next-day LPA. Of note is that these models adjusted for any deviation from average total sleep time, indicating that the observed associations are independently reflective of changes in the placement of sleep episodes within the 24-hour day. These data converge with evidence that evening chronotypes tend to be less physically active, and engage in more sedentary behavior, when compared to morning-types [77]. An experimental study in adolescents recently demonstrated that increasing sleep duration via advancing bedtimes by nearly two hours reduced sedentary time by ~1.7 hours while having no adverse impact on MVPA [20]. To our knowledge, no experimental studies have systematically tested this paradigm in adults. Additionally, the impact of experimentally shifted sleep timing on subsequent activity levels, independent of changes in sleep quantity, also remains uncertain. In the reverse direction, each 60-minute increase in sedentary time predicted a ~10-minute delay in sleep onset that night at both within- and between-subject levels. Results were similar when sedentary time was expressed as a percentage of time awake, such that each 10% increase in sedentary time predicted a ~15-minute delay in sleep onset that night. Interestingly, a 10% reduction in daytime LPA was associated with comparable delays in the timing of subsequent sleep episodes (i.e., ~12-18 minutes), suggesting that replacing sedentary behavior with LPA may represent a relatively simple strategy to bolster interventions that seek to advance sleep timing. Altogether, these findings reiterate the need to consider sedentary behavior as a viable target to improve sleep health – not only in terms of sleep quantity, but also in terms of optimizing sleep timing.

There are numerous mechanisms that may help explain the observed associations in this study. For instance, sleeping at the wrong circadian time (e.g., later-than-average sleep onset), or obtaining less-than-average sleep quantity, can increase fatigue and have been previously linked with a reduction in activity levels [78,79]. Additionally, sedentary behavior may result in reduced exposure to bright sunlight during the daytime as a function of less time spent outside,[80] while evening sedentariness likely involves exposure to artificial

ambient light and/or electronic screens (e.g., reading, TV watching, cell phone or computer use). These unfavorable light exposure patterns can interfere with sleep and circadian rhythms [81], which could plausibly manifest as later and less sleep following days with more sedentariness. Associations between later-than-average sleep onset and poorer next-day dietary intake might also be a consequence of circadian misalignment, defined as wakefulness, activity, or eating occurring during the biological night [82]. Circadian misalignment has been shown to disrupt hunger hormones in such a way that has been associated with poorer food choices [83-85]. Finally, several of these observations are likely explained, at least in part, by implicit time-constraints within a fixed 24-hour period [86]. There is some extent of mutual exclusivity between sleep and daytime behaviors, such that when one is asleep, they cannot be physically active, sedentary, or eating, and vice versa. This inherently yields some degree of interrelatedness among aspects of these behaviors, such as the amount of time available to dedicate to them, and their placement within the 24-hour day. Empirical evidence that quantifies the natural interrelatedness among these daily behaviors is of public health importance, as this knowledge can be leveraged to ensure that health behavior recommendations are designed to yield maximal health benefits.

The current results should be viewed in the context of several important considerations. First, our sample consisted of young, active, non-shift working adults that were well-screened for chronic diseases or sleep disorders. Therefore, our findings may not be generalizable to other populations such as older adults, those with chronic health conditions, or those at high risk for sleep disturbances or clinical sleep disorders. Given these considerations, the daily behaviors examined in this study were guided by the selection of objective metrics that were most applicable to the current sample while also addressing the most novel research questions. However, it is important to acknowledge that sleep, eating, physical activity, and sedentary behaviors are multidimensional; accordingly, there are a rapidly growing number of ways to characterize these behaviors. Thus, the

temporal associations among these daily behaviors warrant continued examination while also encompassing other dimensions not covered here (e.g., sleep quality), particularly in samples that exhibit greater heterogeneity in these measures. Additionally, several of the observed associations were relatively small in magnitude, which is important to consider in the context of broader clinical relevance. However, recognizing that chronic conditions such as cardiovascular disease and type 2 diabetes begin developing early in life and progress with age, it could be argued that even minor modifications in these daily behaviors may have cumulative, meaningful effects if initiated in early adulthood and sustained over time. Finally, a strength of this study is the rigorous micro-longitudinal design that consisted of 14 consecutive days of sleep, diet, and activity assessments, obtained via mostly objective methods and with minimal missing data, yielding a notably robust dataset.

In summary, this comprehensive analysis underscores the interrelatedness between nightly sleep with daytime eating behaviors and activity levels as they are experienced by young adults in free-living, non-experimental contexts. Collectively, these data suggest that sleep generally exerts a greater influence on next-day eating and activity levels, rather than vice versa. Nevertheless, there is an observable impact of daytime behaviors, namely the timing of eating and amount of time spent sedentary, on subsequent sleep health. While these complex interdependencies warrant additional investigation in more diverse samples, our findings support the examination of interventions that target sleep health concomitantly with eating behavior or activity levels, to optimize responses and associated health outcomes. Ultimately, this line of inquiry has the capacity to innovate current paradigms of health behavior change.

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FIGURE CAPTIONS

Figure 1. Summary of temporal associations between total sleep time with eating behavior and activity level. Green and red lines illustrate significant positive and negative associations, respectively. Dashed lines illustrate within-subject associations, dotted lines illustrate between-subject associations, and solid lines illustrate associations at both within- and between-subject levels. Data are unstandardized beta estimates ± standard error. For models where total sleep time is the independent variable, estimates are presented per 60-minute increase in total sleep time. In models where eating behavior and activity level metrics are the independent variable, estimates are presented as follows: HEI score, per 10-point increase; total caloric intake, per 100-kcal increase; timing of eating onset and eating offset, per 1-hour delay; duration of eating window, per 1-hour increase; sedentary time and total physical activity (minutes), per 60-minute increase; sedentary time and total physical activity (%), per 10% increase. W, within-subject; B, between-subject. Associations observed at both within- and between-subject levels, and in both directions, were considered bidirectional. *p<0.017, **p<0.001.

Figure 2. Summary of temporal associations between timing of sleep onset with eating behavior and activity level. Green and red lines illustrate significant positive and negative associations, respectively. Dashed lines illustrate within-subject associations, dotted lines illustrate between-subject associations, and solid lines illustrate associations at both within- and between-subject levels. Data are unstandardized beta estimates ± standard error. For models where sleep onset is the independent variable, estimates are presented per 1-hour delay in sleep onset timing. In models where eating behavior and activity level metrics are the independent variable, estimates are presented as follows: HEI score, per 10-point increase; total caloric intake, per 100-kcal increase; timing of eating onset and eating offset, per 1-hour delay; duration of eating window, per 1-hour increase; sedentary time and total physical activity (minutes), per 60-minute increase; sedentary time and total physical activity (%), per 10% increase. W, within-subject; B, between-subject. Associations observed at both within- and between-subject levels, and in both directions, were considered bidirectional. *p<0.017, **p<0.001.

Figure 3. Summary of temporal associations between timing of wake onset with eating behavior and activity level. Green and red lines illustrate significant positive and negative associations, respectively. Dashed lines illustrate within-subject associations, dotted lines illustrate between-subject associations, and solid lines illustrate associations at both within- and between-subject levels. Data are unstandardized beta estimates ± standard error. For models where wake onset is the independent variable, estimates are presented per 1-hour delay in wake onset timing. In models where eating behavior and activity level metrics are the independent variable, estimates are presented as follows: HEI score, per 10-point increase; total caloric intake, per 100-kcal increase; timing of eating onset and eating offset, per 1-hour delay; duration of eating window, per 1-hour increase; sedentary time and total physical activity (minutes), per 60-minute increase; sedentary time and total physical activity (%), per 10% increase. W, within-subject; B, between-subject. Associations observed at both within- and between-subject levels, and in both directions, were considered bidirectional. *p<0.017, **p<0.001.

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Table 1. Participant Characteristics (N=63)

	Mean (SD) or n (%)
General Characteristics	
Age, years	28.9 (7.1)
Sex	
Male	22 (34.9)
Female	41 (65.1)
Race	
White, non-Hispanic	42 (66.7)
Black, non-Hispanic	5 (7.9)
Asian, non-Hispanic	7 (11.1)
Hispanic	6 (9.5)
Other	3 (4.8)
Body Mass Index, kg/m^2	24.3 (3.1)
Sleep Characteristics	
Total Sleep Time, min/night	417.2 (38.1)
Sleep Onset, clock time	23:31 (1hr 04m)
Wake Onset, clock time	07:03 (1hr 01m)
Eating Behavior Characteristics	
Diet Quality, HEI score	59.4 (10.6)
Total Caloric Intake, kcal/day	2230.2 (757.5)
Eating Onset, clock time	08:50 (1hr 28m)
Eating Offset, clock time	20:19 (1hr 00m)
Duration of Eating Window, h/day	11.49 (1.6)
Activity Characteristics	
Sedentary Behavior, min/day	670.8 (69.0)
Sedentary Behavior, %/wake	75.4 (6.2)
Total Physical Activity, min/day	218.5 (57.0)
Total Physical Activity, %/wake	24.6 (6.2)
LPA, min/day	163.1 (46.3)
LPA, %/wake	18.3 (5.3)
MVPA, min/day	55.3 (29.2)
MVPA, %/wake	6.2 (3.1)

HEI, Healthy Eating Index; LPA, light physical activity; MVPA, moderate-vigorous physical activity

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Figure 1.

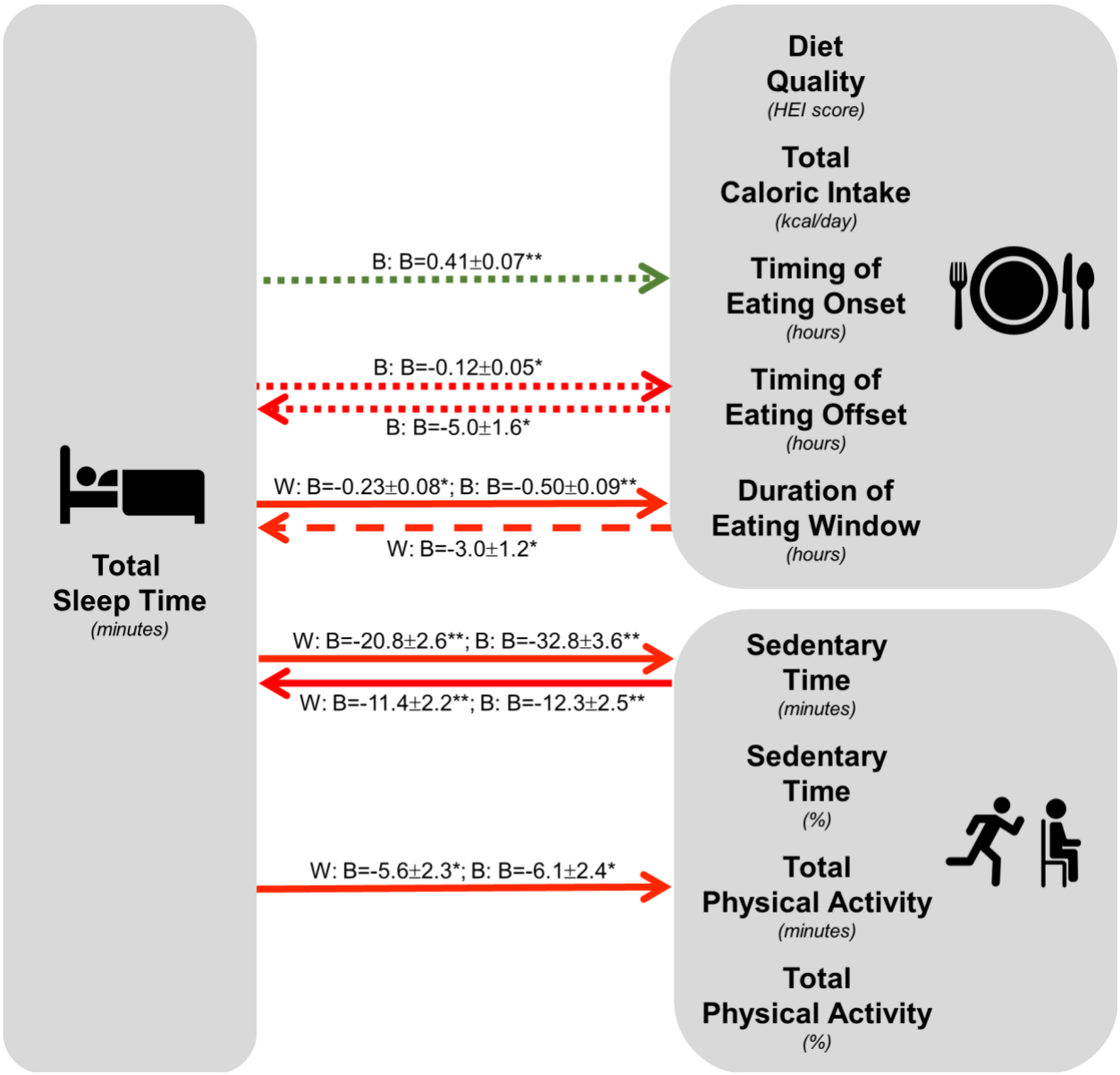

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Figure 2.

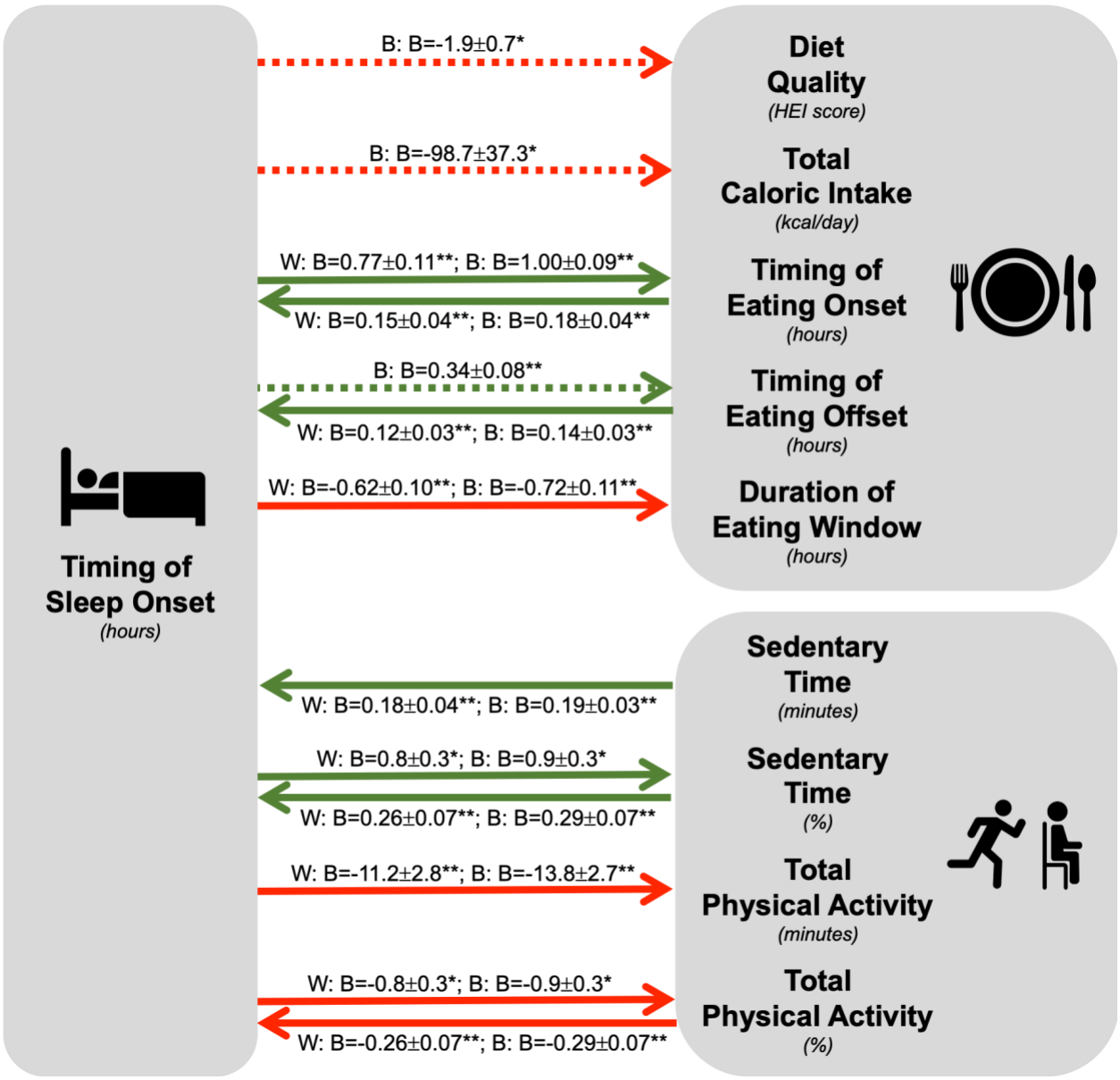

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Figure 3.