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No Effect of 8-Week Time-in-Bed Restriction on Glucose Tolerance in Older Long Sleepers

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SUMMARY

The aim of this study was to investigate the effects of 8 weeks of moderate restriction of time in bed (TIB) on glucose tolerance and insulin sensitivity in healthy older self-reported long sleepers. Forty-two older adults (ages 50–70 years) who reported average sleep durations of ≥ 8.5 h per night were assessed. Following a 2-week baseline, participants were randomly assigned to two 8-week treatments: either (1) TIB restriction (n = 22), which involved following a fixed sleep schedule in which time in bed was reduced by 90 min compared with baseline; (2) a control (n = 18), which involved following a fixed sleep schedule but no imposed change of TIB. Sleep was monitored continuously via wrist actigraphy recordings, supplemented with a daily diary. Glucose tolerance and insulin sensitivity were assessed before and following the treatments. Compared with the control treatment, TIB restriction resulted in a significantly greater reduction of nocturnal TIB (1.39 ± 0.40 h versus 0.14 ± 0.26 h), nocturnal total sleep time (TST) (1.03 ± 0.53 h versus 0.40 ± 0.42 h), and 24-hr TST (1.03 ± 0.53 h versus 0.33 ± 0.43 h) from baseline values. However, no significant effect of TIB restriction was found for glucose tolerance or insulin sensitivity. These results suggest that healthy older long sleepers can tolerate 8 weeks of moderate TIB restriction without impairments in glucose tolerance or insulin sensitivity.

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MRZ: No conflicts of interest to report.

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DFK: No conflicts of interest to report.

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Keywords

Long sleep; sleep restriction; glucose tolerance; insulin sensitivity; sleep deprivation; older adults

INTRODUCTION

Epidemiologic studies have consistently found a U-shaped association of self-reported sleep duration with increased mortality and with multiple morbidities, including cancer, cardiovascular disease, stroke (Kripke *et al*., 2002; Kronholm *et al*., 2006; Patel *et al*., 2006), type 2 diabetes, and impaired glucose tolerance (Ayas *et al*., 2003; Chaput *et al*., 2007; Gottlieb *et al*., 2006; Tuomilehto *et al*., 2007). The lowest risk has generally been associated with 7–8 hrs of reported sleep. Although far more attention has been paid to the risks of short sleep duration (< 7 h), generally the risks associated with long sleep (≥ 8 h per night) have been at least as great (Kripke *et al*., 2002; Kronholm *et al*., 2006; Lan *et al*., 2007; Patel *et al*., 2006). Moreover, some evidence has suggested that long sleep might be a greater public health risk than short sleep, since, in some studies, a greater percentage of the population have reported sleep durations of ≥ 8 h than < 7 h (Kripke *et al*., 2002; Patel *et al*., 2004; Tamakoshi *et al*., 2004).

Notwithstanding the epidemiologic evidence, the notion that long sleep is hazardous might seem counterintuitive. Of course, epidemiologic data cannot prove that long sleep causes mortality or morbidity. Experimental studies are needed to address causality. Moderate restriction of time in bed (TIB) in long sleepers is one such approach.

If long sleep reflects superfluous, unnecessary sleep, then moderate restriction of sleep or TIB might be well-tolerated, even beneficial, to those reporting sleep of more than 7.5 h (Youngstedt and Kripke, 2004). Benefits of moderate TIB restriction might be particularly important for older adults who often seem to spend more TIB than young adults (Carskadon *et al*., 1982), despite a steady decline in objective sleep duration with age (Ohayon *et al*., 2004).

One potential beneficial consequence of moderate TIB restriction is that it could lead to increased physical activity. Long self-reported sleep has been associated with lower levels of physical activity (Basner *et al*., 2007; Patel *et al*., 2006) and fitness (Morgan, 2007), and this could be explained by extra time spent in completely sedentary activity (bed-rest), feelings of lethargy associated with excessive sleep (Youngstedt and Kripke, 2004), and simply having less timed time to engage in physical activity (Basner *et al*., 2007).

On the other hand, if reported long sleep reflects normal variation in sleep need (de Castro, 2002), then detrimental effects of sleep restriction may be evident in long sleepers (i.e., those who sleep ≥ 8 h per night) as well as normal sleepers. One of the most commonly discussed risks of sleep restriction has been impairment in glucose/insulin regulation. Older adults, who are more prone to diabetes than young adults, might be particularly susceptible to this risk. Although this risk has been suggested by epidemiologic studies (Ayas *et al*., 2003; Gottlieb *et al*., 2005; Knutson, *et al*., 2006; Kohatsu *et al*., 2006; Mallon *et al*., 2005; Taheri *et al*., 2004; Tuomilehto et al., 2007; Yaggi *et al*., 2006) – insofar as short sleep can be attributed to sleep restriction –, experimental support for this assumption has been somewhat limited (Gonzalez-Ortiz *et al*., 2000; Kuhn, *et al*., 1969; Spiegel *et al*., 1999; Spiegel *et al*., 2004; Tasali *et al*., 2008; VanHelder *et al*., 1993; Vondra *et al*., 1981).

An influential study of this topic found significant impairment of glucose tolerance in 11 young males following 6 nights during which they spent 4 h in bed compared with a

subsequent 6 day period during which they spent 12 h in bed per day (Spiegel *et al*., 1999). However, limitations of the study included a small sample size; failure to include a control condition or to counterbalance order of assessment; and administration of an influenza vaccine (Spiegel *et al*., 2002) during the TIB restriction period, a stimulus that can impair glucose tolerance (Munoz *et al*., 2005). Finally, the relatively severe and short-term TIB/sleep restriction raised questions about the generalizability of the findings. Other experimental studies on this topic have involved even more dramatic total sleep deprivation for periods ranging from 24–120 h (Gonzalez-Ortiz *et al*., 2000; VanHelder *et al*., 1993; Vondra *et al*., 1981), which may have little relevance to the situation of a person who is chronically sleep-restricted for only an hour or two.

The aim of the present experiment was to examine the influence of 8 weeks of moderate TIB restriction on glucose and insulin regulation in healthy older self-reported long sleepers. The study was part of a larger investigation regarding the potential risks or benefits of moderate sleep restriction in older self-reported long sleepers.

METHODS

Forty-two participants participated in this 10-week study (Table 1). Initial exclusion criteria were based on several screening questionnaires and included reported average sleep duration of < 8.5 h; average daily napping of ≥ 60 minutes; recent shift-work experience or travel across multiple time zones; signs and symptoms of severe sleep apnea or idiopathic hypersomnia; severe daytime sleepiness; diabetes mellitus; and medical, neurological, or psychiatric illness which might cause long sleep.

Prospective participants who passed the initial questionnaire screening were invited to the laboratory to further discuss the study and their ability/willingness to participate. Thereafter, participants were invited to sign a written informed consent, which had been approved by the University of South Carolina Institutional Review Board.

Pre-Treatment Oral Glucose Tolerance Test

An oral glucose tolerance and insulin sensitivity test was used to further exclude potential participants who had diabetes, as well as to provide pre-treatment data. Participants reported to a hospital laboratory at 7:00–7:30 h following a 12-h fast and 24-h abstention from exercise. Glucose tolerance and insulin sensitivity were assessed with standard laboratory procedures. First, fasting levels of glucose and insulin were assessed via a 4-cc venous blood draw. Volunteers then consumed 7.5 oz of a highly concentrated glucose drink (equivalent to 75 g of dextrose) and sat in a comfortable chair in a quiet room. Two hrs following glucose ingestion, a blood draw was repeated for glucose and insulin assessment. According to convention, glucose tolerance was defined by the 2-h glucose level. Measurements of glucose and insulin are described below. Exclusion criteria for diabetes mellitus were fasting glucose levels ≥ 126 mg/dL and/or 2-h glucose levels ≥ 200 mg/dL.

Immediately following the laboratory visit, the participants were given a physical exam and interview by a sleep physician (RKB) to exclude participants for whom the protocol might be unsafe and to exclude for other sleep disorders or medical causes of long sleep.

Experimental Procedures

Following pre-treatment screening, participants began the 10-week study, which included a 2-week baseline period and an 8-week experimental treatment. Throughout the 10 weeks, participants refrained from transmeridian travel and shift-work. Weekly data collection was conducted in the homes of most of the participants, or in the laboratory for a few of the participants.

Baseline Assessment—During a 2-week baseline period, participants were asked to follow their usual nocturnal sleep and daytime napping habits. Sleep and light exposure were assessed continuously via wrist actigraphic recording (Octagonal Basic Motionlogger, Ambulatory Monitoring, Inc., Ardsley, NY, USA), supplemented with a daily sleep diary. The actigraphic recording also provided a crude index of physical activity. At the end of each week, actigraphic data were downloaded into a laptop computer, and questionnaire data were collected.

Experimental Treatment—Following baseline, participants were assigned to one of two 8-week treatments: (1) TIB restriction or (2) control. Since the goal of this study was to examine $n = 30$ and $n = 20$ volunteers, respectively, in these treatments, every 5^{th} participant was assigned to the sleep restriction treatment, whereas other participants were randomly assigned to the treatments. As during baseline, sleep/wake and wrist activity were assessed continuously via wrist actigraphy and sleep diaries, and research staff visited with the volunteers at the end of each week to download the actigraphic data.

The **TIB restriction treatment** required following a fixed sleep-wake schedule in which TIB was 90 minutes less than the median TIB established during the baseline period. Consulting with the PI, each participant decided before the 8-week experimental manipulation began how he/she would restrict TIB, i.e., by delaying bedtime, advancing wake-time, or some combination of both. Participants were asked to maintain their usual napping habits. However, to facilitate maintenance of wakefulness, they were allowed to increase their usual caffeine intake by up to 200 mg per day.

The **control treatment** involved no restriction of TIB. However, to control for potential beneficial effects of maintaining a fixed sleep-wake cycle, the control treatment also involved a fixed sleep-wake cycle, with timing of TIB consistent with the median established for each individual during the baseline period. As in the TIB restriction treatment, participants were asked to maintain their usual napping habits.

Post-Treatment Oral Glucose Tolerance Test

The post-treatment OGTT was within 1 day of completing the experiment for most of the participants ($n = 30$, 75%), but 2, 3, 4, and 5 days after the experiment in $n = 2$, 3, 4, and 1 participants, respectively. Participants were instructed to maintain their assigned experimental treatment TIB schedule until completion of the OGTT. The procedures described for baseline glucose tolerance were repeated.

Data Processing

Glucose, Glucose Tolerance, and Insulin Sensitivity Assays and Quantification—The primary variables of interest were: (1) fasting serum glucose concentration; (2) glucose tolerance, defined as serum glucose concentration at 2 hours following glucose ingestion; and (3) insulin sensitivity (defined below). Serum glucose concentration (mg/dL) was determined with a spectrophotometer (Bayer Advia 1650) using a glucose hexokinase liquid solution (COBA INTEGRA Glucose HK Liquid, ADVIA Centaur, Japan).

Sera were assessed for insulin levels using direct chemiluminescent technology on a Bayer Advia Centaur with liquid insulin reagent (ADVIA Centaur, Japan). Insulin sensitivity was measured by the quantitative insulin-sensitivity check index (QUICKI). QUICKI was calculated as $Q = 1/(\log FPI + \log FPG)$, where FPI is the fasting plasma insulin (mU/L) and FPG is the fasting plasma glucose (mg/dL) (Trout *et al*., 2007). In this calculation, lower levels are associated with lower insulin sensitivity (i.e., increased insulin resistance).

Sleep Assessment—Actigraphic data were scored with a validated algorithm associating wrist movement with polysomnographically-assessed sleep using Action 4 software (Ambulatory Monitoring, Inc., Ardsley, NY, USA), with some editing (e.g., for time in which the actigraph was removed). The variables of interest for the present study were the nocturnal TIB and TST, the duration of daytime napping, and 24-h TST (including napping).

Wrist Activity Assessment—The proportional integral mode (PIM) channel (Ambulatory Monitoring, Inc., Ardsley, NY, USA) was used to assess acticraphic wrist movement. Mean wrist activity during baseline and the experimental treatment were determined.

Statistical Analyses

Data are presented as mean ± standard deviation (SD). Statistical significance was set at $P < 0.05$. Demographic data and pre-treatment levels of sleep, glucose, and insulin regulation were compared between treatment groups with independent samples *t*-tests. Mean baseline and mean 8-week treatment levels of TIB, nocturnal TST, 24-h TST, and total napping time were calculated. Changes were compared between treatments with repeated measures treatment by time ANOVA.

Changes in fasting serum glucose and insulin, in glucose and insulin levels at 2 h following glucose ingestion, and in insulin sensitivity (QUICKI) were compared between treatments by assessing post-treatment levels with ANCOVA, with covariate control for baseline levels and body mass index (BMI). Differences in mean wrist activity were assessed between treatments with post-treatment ANCOVA, with covariate control for baseline levels.

Associations of changes in the glucose and insulin measures with changes in TIB, nocturnal TST, and 24-h TST were explored with Spearman rank-order correlations of these data, both for the entire sample and separately for the TIB restriction group.

RESULTS

Due to laboratory error, post-treatment glucose and insulin data were not obtained for 2 of 42 participants. Due to equipment technical failures, actigraphic data were not obtained for 7 of 40 participants for whom there was complete glucose/insulin data. However, actigraphic data confirmed the success of the experimental TIB manipulation in the other 33 participants, and sleep diary data indicated that all the participants followed the experimental schedule. Therefore, these present results include $n = 40$ for demographic data and glucose/insulin data, but $n = 33$ for the sleep data and for the correlations of sleep and glucose/insulin data.

Demographic Data

There were no significant differences between groups in age, height, weight, or BMI (Table 1). The sleep restriction group was comprised of 16 women (73%) and 6 men (27%), whereas the control group was comprised of 12 women (67%) and 6 men (33%).

Sleep Data

Mean actigraphic sleep data are displayed in Table 2. No significant pre-treatment differences in TIB or nocturnal TST were observed between the treatment groups. Compared with pre-treatment levels, the TIB restriction group had a mean TIB reduction of 1.39 ± 0.40 h (range: −1.96 to −0.60 h), a reduction that was significantly greater than experienced by the control group (0.14 ± 0.26 h) (range: −0.53 to 0.26 h) (treatment by time: $F = 131.51$, $P < 0.001$). Likewise, the reduction of nocturnal TST during the TIB restriction

treatment (1.03 ± 0.53 h) was significantly greater than that observed during the control treatment (0.40 ± 0.42 h) (treatment by time: $F = 14.45$, $P = 0.001$). In addition, the change in TST over 24 h (including napping time) from baseline values was significantly greater following the TIB restriction treatment (1.03 ± 0.53 h) than the control treatment (0.33 ± 0.43 h) ($F = 17.65$, $P < 0.001$). No significant treatment, time, or treatment by time effects for daytime napping were observed.

Wrist Activity

Mean wrist activity from the actigraphic PIM channel are shown in Table 2. A borderline-significant effect of increased physical activity with TIB restriction vs. control treatment was found ($F = 4.176$, $P = 0.050$).

Glucose, Glucose Tolerance, and Insulin Sensitivity

Mean glucose and insulin data are displayed in Table 3. No significant pre-treatment differences were found between treatment groups in fasting glucose or 2-h post-glucose ingestion.

ANCOVA, controlling for baseline levels and BMI, revealed a significant ($F = 4.38$, $P = 0.043$), albeit small, post-treatment effect for fasting glucose, which was mediated by a 1% increase in fasting glucose following TIB restriction (from 99.27 ± 9.26 to 100.41 ± 9.03 mg/dL), and a 4% decrease following the control treatment (from 103.22 ± 8.32 to 99.94 ± 8.21 mg/dL). However, there were no significant post-treatment effects for glucose tolerance (i.e., glucose level at 2-h post-ingestion).

Compared with the control group, pre-treatment fasting insulin was significantly lower in the TIB restriction group [t (38) = 7.129, $P = 0.011$]. However, ANCOVA revealed no significant post-treatment effect for fasting insulin. Pre-treatment insulin at 2-h post-glucose ingestion did not differ significantly between groups, nor was there a significant post-treatment effect for 2-hr post-ingestion levels of insulin (Table 3).

Pre-treatment insulin sensitivity (QUICKI) was significantly higher for the TIB restriction group vs. the control group [t (38) = 7.194, $P = 0.011$]. However, ANCOVA controlling for pre-treatment QUICKI and BMI, revealed no significant post-treatment effect for insulin sensitivity.

No significant correlations were found for changes in TIB, nocturnal TST, or 24-h TST with fasting glucose, glucose tolerance, or insulin sensitivity. This held true for the entire sample, as well as for analyses limited to the sleep restriction group.

Post-hoc analysis assessed the ten participants with the greatest reduction in night-time TST (80.86 ± 16.29 min; i.e., 7.84 ± 0.58 to 6.50 ± 0.63 h) and in 24-h TST (81.42 ± 17.88 min; i.e., 8.14 ± 1.09 to 6.78 ± 1.04 h). Even in this group, there were no significant changes in fasting glucose, 2-h glucose, or insulin sensitivity.

The present study had 80% power to detect a significant increase in fasting glucose of 6.49 mg/dL, an increase in 2-h post-glucose levels of 28 mg/dL, and a decrease in QUICKI of 0.0191 following TIB restriction vs. control treatment (Sample Power, SPSS Inc., Chicago, IL, USA).

DISCUSSION

This study found no significant changes in glucose tolerance or insulin sensitivity following 8 weeks of moderate TIB restriction in healthy older self-reported long sleepers. It was

unfortunate that random assignment yielded some differences between groups before treatment. Although a significant post-treatment effect for fasting glucose appeared, post hoc analysis revealed that this effect was due to a significant decrease in fasting glucose following the control treatment ($t = 2.199$, $P = 0.042$), and a minimal non-significant increase in fasting glucose following sleep restriction (1.14 mg/dL). No significant correlation was found between the degree of sleep restriction and changes in glucose or insulin regulation. These data do not coincide with those from other experimental studies that have found impairment of glucose/insulin regulation following more extreme sleep loss (Gonzalez-Ortiz *et al*., 2000; Kuhn *et al*., 1969; Spiegel *et al*., 1999; Spiegel *et al*., 2004; VanHelder *et al*., 1993; Vondra *et al*., 1981).

Apparent discrepancies in findings might be attributed to several methodological differences between the present study and previous studies. First, whereas previous studies assessed young individuals with average sleep durations, the present study assessed older self-reported long sleepers. Considering epidemiologic associations of long sleep with impaired glucose tolerance (Ayas *et al*., 2003; Gottlieb *et al*., 2005; Yaggi *et al*., 2006), an improvement in glucose tolerance following moderate TIB restriction and/or sleep restriction might have been posited, though perhaps difficult to demonstrate in light of the high baseline levels of glucose tolerance and insulin sensitivity (i.e., a ceiling effect) (Youngstedt, 2003).

However, a noteworthy finding of this study was that while participants spent a mean of > 9 h in bed, their mean baseline nocturnal TST was only 7.4 h. The nocturnal baseline TST recorded in our participants was about 60 min longer than age-matched normative data (Ohayon *et al*., 2004), though not at a level that is typically considered long sleep. Nonetheless, prolonged TIB has been associated with significant sleep fragmentation, which has been linked independently with impaired glucose tolerance (Chasens, 2007; Coughlin *et al*., 2004; De La Eva *et al*., 2002; Manzella *et al*., 2002; Meslier *et al*., 2003; Scheen *et al*., 1996; Tassone *et al*., 2003).

Conversely, improved sleep consolidation following TIB restriction could conceivably improve glucose tolerance, or at least prevent impairment in glucose tolerance. Our data do not strongly support this scenario, as sleep efficiency improved by only a modest amount (about 1%) following TIB restriction (Youngstedt *et al*., 2005). Nonetheless, it is plausible that these individuals had different metabolic responses to TIB restriction than young adults with average sleep duration.

The high degree of sleep fragmentation might be partly explained by the age of the participants. Our analyses suggest that for these older self-reported long sleepers (≥8.5 h), reported sleep was largely a reflection of TIB (Youngstedt *et al*., 2005). Meta-analytical data further indicate that objective sleep of ≥ 8.5 h is quite rare for this age group (Ohayon *et al.*, 2004).

Another main methodological difference is that previous studies have involved profound TIB restriction or total sleep deprivation for no longer than 6 consecutive nights (Gonzalez-Ortiz *et al*., 2000; Kuhn, *et al*., 1969; Spiegel *et al*., 1999; Spiegel *et al*., 2004; VanHelder *et al*., 1993; Vondra *et al*., 1981), whereas the present study involved relatively modest TIB restriction over 8 weeks. Thus, the differences in results could be due to dose-response or threshold effects of sleep and/or TIB restriction on glucose/insulin regulation. On the other hand, the results could reflect adaptation to more chronic TIB restriction, and the intervention of the present study seems far more generalizeable to common experience.

Impairments in glucose tolerance following TIB restriction might have been partly offset by an increase in physical activity, which was found in the present study, albeit with the crude

physical activity index of wrist actigraphic data. A previous study by VanHelder et al. (1993) found that exercise significantly attenuated impairment in insulin regulation following sleep deprivation. Further investigation of the effects of TIB restriction on physical activity is warranted (Morgan, 2007).

The present study had several limitations that might have made impairment of glucose/insulin regulation somewhat difficult to detect. First, the randomization outcome was not ideal, since baseline insulin sensitivity was significantly higher in the TIB restriction vs. control group.

Second, the screening procedures, deemed necessary for the safety of the study, probably resulted in a sample that was not representative of the population of older self-reported long sleepers. However, it should be noted that the participants were not superbly fit seniors. According to conventional BMI standards, 47.5% and 10% of the subjects were classified as overweight or obese, respectively. Moreover, based on our estimates, only about 10% of the participants were physically active.

Third, the routine techniques that were used for assessing glucose tolerance and insulin sensitivity had less sensitivity than techniques used in other studies, including hyperglycemic clamps and other refined methods available in clinical research center settings (Spiegel *et al*., 1999), which limits their comparability. Nonetheless, the techniques employed have well-established validity and are quite clearly adequate for detecting clinically significant changes in glucose tolerance (Cheng *et al*., 2004; Huggett *et al*., 2004; Maslovitz *et al.*, 2006; Van Helder *et al*., 1993). Moreover, the study had 80% power to detect an increase in 2-h post-ingestion glucose of 28 mg/dl and a final level (128 mg/dl) that would still be below the common criterion for glucose intolerance (140 mg/dl).

A fourth limitation of the present home-based study was that it allowed less experimental control (e.g., of glucose intake) than in other studies which involved in-patient control of diet or constant intravenous feeding (e.g., Spiegel *et al*., 1999). However, such exact control of glucose intake would not be practical for prolonged TIB restriction studies, for which this study was setting a basis, and the present results might have more ecological validity. Indeed, greater impairments in glucose tolerance might be expected under field conditions because of hypothesized increases in appetite and sugar craving (Knutson *et al*., 2007).

A fifth limitation was that although the TIB restriction intervention of the present study was much longer than in previous studies, a more chronic duration of TIB restriction might exacerbate impaired glucose tolerance. It has been claimed that cumulative impairment in glucose tolerance associated with sleep restriction may be analogous to cumulative effects posited for neurobehavioral performance, such as the psychomotor vigilance test (Spiegel *et al*., 2002; Van Dongen *et al*., 2003).

A sixth limitation was that our control group experienced significant reductions in nocturnal sleep duration (0.40 h), 24-hr sleep duration (0.33 h), and TIB (0.14 h) ($t = 3.749$, $P = 0.002$; $t = 2.232$, $P = 0.039$; and $t = 3.749$, $P = 0.002$, respectively). Although a rigid sleep schedule is helpful for insomniacs with erratic sleep (Joshi, 2008), it might be slightly restrictive for individuals without sleep complaints, who would otherwise go to sleep and arise when needed.

A seventh limitation was that scheduling of post-treatment glucose tolerance more than 1 day after the experiment could have potentially resulted in attenuated effects of sleep restriction, particularly if the participants had slept exceedingly long durations after completing the study. However, beside the fact that 75% of the participants were given the OGTT the day after completing the experiment, there are several reasons why we do not

think the delay in OGTT for the other participants significantly impacted the results. First, participants were asked to maintain their experimental sleep schedule until the time of the OGTT. Second, the OGTT always occurred at 7:00 or 7:30 a.m., a time which required getting up even earlier than scheduled during the sleep restriction protocol for approximately 75% of the subjects. Third, as we will report elsewhere, the sleep restriction protocol elicited no apparent increase in sleepiness or impairment in performance, which might have promoted a need to recover lost sleep. Indeed, in 12-month follow-up assessments, we found that participants continued to voluntarily restrict their self-reported sleep by an average of approximately 1 h below baseline levels. Fourth, post-hoc analysis revealed no difference in OGTT changes between participants tested 1 vs. more than 1 day after completing the study.

Notwithstanding its unique sample and limitations, the present study circumscribes the widely-accepted theory that restricting the available amount of time to sleep necessarily impairs glucose tolerance or insulin sensitivity. Previous studies leading to this theory have had their own limitations. The findings of our study should be expanded to studies of different age groups, participants with a range of sleep durations, more prolonged TIB restriction, and more precise measures of glucose tolerance and insulin sensitivity. A long-term goal of this research is to provide groundwork for clinical trials of TIB restriction as a method of limiting the mortality and morbidity associated with long sleep.

In conclusion, these data suggest that healthy older self-reported long sleepers can tolerate 8 weeks of moderate TIB restriction without significant impairments in glucose tolerance or insulin sensitivity. Although no improvement in glucose/insulin regulation was observed, the lack of impairment was consistent with the hypothesis that some older self-reported long sleepers might be spending too much time in bed.

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Table 1

Demographic Pre-treatment Characteristics

Treatment Group	Age (yr)	Height (cm)	Weight (kg)	BMI (kg⋅−2)
Control (*n* = 18)	60.89 ± 5.07	166.79 ± 6.89	74.34 ± 13.67	26.58 ± 3.43
TIB Restriction (*n* = 22)	59.95 ± 5.49	170.67 ± 8.53	71.01 ± 10.13	24.65 ± 3.00

Values are Mean ± SD. TIB: time in bed; BMI: body mass index. There were no significant differences in demographic characteristics between the treatment groups.

Table 2

Pre-treatment and Post-treatment Daily TIB, Nocturnal and 24-h TST, and Wrist Activity

Treatment Group	Pre-treatment	Post-treatment
	TIB (h)	
Control	9.22 ± 0.67	9.08 ± 0.72**
TIB Restriction	9.11 ± 0.63	7.71 ± 0.53**
	Nocturnal TST (h)	
Control	7.22 ± 1.12	6.82 ± 1.05**
TIB Restriction	7.58 ± 0.67	6.55 ± 0.65**
	Napping (min)	
Control	17.17 ± 13.07	21.14 ± 19.21
TIB Restriction	16.40 ± 28.72	16.17 ± 25.05
	24-hr TST (h)	
Control	7.50 ± 1.21	7.17 ± 1.18**
TIB Restriction	7.85 ± 1.00	6.81 ± 0.89**
	Wrist Activity	
Control	3779.8 ± 595.9	3772.7 ± 756.9*
TIB Restriction	4365.1 ± 1155.8	4699.3 ± 1114.3*

Values are Mean ± SD. TIB: time in bed; TST: total sleep time.

* Significant post-treatment difference between treatment groups (following control for baseline values) ($P < 0.05$).

** Significant treatment-by-time interaction ($P < 0.05$).

Table 3

Pre-treatment and Post-treatment Glucose and Insulin Results

Treatment Group	Pre-treatment		Post-treatment	
	Serum Glucose (mg/dL)			
	Fasting	2-h Post	Fasting	2-h Post
Control	103.22 ± 8.32	108.17 ± 36.56	99.94 ± 8.21**	103.94 ± 34.05
TIB Restriction	99.27 ± 9.26	97.23 ± 29.63	100.41 ± 9.03**	90.82 ± 20.70
	Serum Insulin (mU/L)			
	Fasting	2-h Post	Fasting	2-h Post
Control	9.93 ± 5.59*	68.12 ± 70.83	8.93 ± 5.43	61.81 ± 59.71
TIB Restriction	5.51 ± 1.92*	37.98 ± 29.95	6.27 ± 3.39	32.57 ± 28.30

Values are Mean ± SD. 2-h Post: assessment 2 h following initial glucose ingestion (75 g).

* Significant pre-treatment difference between treatment groups ($P < 0.05$).

** Significant post-treatment difference between treatment groups (following control for baseline BMI and baseline values) ($P < 0.05$).

Table 4

Pre-treatment and Post-treatment Insulin Sensitivity using QUICKI

Treatment Group	Pre-treatment	Post-treatment
	Insulin Sensitivity	
Control	0.341 ± 0.029*	0.349 ± 0.029
TIB Restriction	0.370 ± 0.023*	0.367 ± 0.032

Values are Mean ± SD.

*Significant pre-treatment difference between treatment groups ($P < 0.05$).