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Journal Title: American Journal of Medicine
Volume: Volume 130, Number 5
Publisher: Elsevier: 12 months | 2017-05-01, Pages 564-571
Type of Work: Article | Post-print: After Peer Review
Publisher DOI: 10.1016/j.amjmed.2016.12.007
Permanent URL: https://pid.emory.edu/ark:/25593/s9jdq

Final published version: http://dx.doi.org/10.1016/j.amjmed.2016.12.007

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Accessed February 4, 2020 7:01 PM EST
Habitual and Recent Sleep Durations: Graded and Interactive Risk for Impaired Glycemic Control in a Biracial Population

Donald L. Bliwise, Ph.D., Sophia A. Greer, M.P.H., Michael K. Scullin, Ph.D., and Lawrence S. Phillips, M.D.

Abstract

Background and Aims—We examined how habitual sleep duration interacts with recent sleep (two nights) to predict morning oral glucose tolerance test results. We hypothesized short habitual and recent sleep durations would be additive for poor glucose control.

Methods—A biracial population of adults (n = 1,559) without known diabetes and recruited from the workforce of two urban universities was assessed for glycated hemoglobin and underwent oral glucose tolerance testing. We used plasma two-hour post-loading (75 g) measurements. Participants answered sleep questions using 30-minute forced-choice formats. We employed multivariable logistic regression to derive Odds Ratios.

Results—Shorter habitual sleep duration was associated with greater Odds Ratios of glycated hemoglobin ≥6.0% increasing by 30-minute intervals beginning at < 7.0 hours and were more pronounced as durations shortened. Among participants with glycated hemoglobin < 6.0% and < 7.0 hours of habitual sleep (n = 636), abnormal glucose tolerance (two-hour oral glucose tolerance test ≥140 mg/dl) was significantly associated with a total sleep duration of ≤11 hours the two
nights preceding oral glucose tolerance testing, but was not associated with longer sleep durations. Results were independent of age, sex, race, body mass index, smoking, history of cardiovascular disease, or use of anti-hypertensive or cholesterol-lowering medication. Additional analyses implied that longer-than-usual recent sleep durations were protective for abnormal oral glucose tolerance testing.

Discussion—Short habitual and recent sleep durations interact in predicting abnormal glucose on oral glucose tolerance testing. Self-reported data are sufficiently sensitive to reflect 30-minute differences in sleep between individuals. Future studies examining other aspects of sleep, such as perceived sleep quality and objectively measured sleep duration and architecture, would be necessary to confirm these findings.

Conclusions—Short sleep duration for two nights prior to morning oral glucose tolerance testing may elevate glucose levels, this effect being detected among individuals habitually obtaining < 7 hours sleep and obtaining ≤ 11 hours of sleep for two nights preceding testing.

Keywords
Sleep duration; diabetes; glucose tolerance test; forced choice questions

INTRODUCTION
Getting less sleep is associated with poor glycemic control. In population studies, individuals who reported habitually short durations of sleep or habitually poor sleep quality have elevations in glycated hemoglobin, diagnosed diabetes or the metabolic syndrome [1–8]. In addition, small studies of generally healthy young adults have shown that shortening or interruption of sleep leads to increased insulin resistance and glucose intolerance [9–13]. However, the extent to which recent reductions in sleep are associated with higher glucose levels across a broad age range, in men and women, and among different racial groups remains unknown. The objective of this study was to examine how habitual sleep duration interacts with the amount of sleep obtained for two nights prior to morning oral glucose tolerance testing. We hypothesized that influence of short habitual and recent sleep durations would be additive for poor glucose control.

In this study we examined associations between habitual and recent sleep duration with glycemic control in a racially diverse population encompassing ambulatory women and men across a broad age range. We asked participants to judge their sleep durations using a novel, forced-choice, scaled format using 30-minute increments. Previous studies used broader categories based on one or two-hour integer bins for estimates of sleep durations.

MATERIALS AND METHODS
Participants
Subjects (908 women; 651 men) were participants in a study [14] of screening for glucose intolerance in a population of adults at two urban United States universities (Emory University, Morehouse School of Medicine) and their associated health care facilities. Participants with a known diagnosis of diabetes were excluded from enrollment, as were
pregnant or nursing women or individuals taking glucocorticoids. Phase of menstrual cycle was not controlled. Among women, 198 had used hormone replacement therapy. No screening for diagnosed or undiagnosed sleep apnea was undertaken, and usage of sleep medication was not controlled. An unspecified number of subjects may have also had caregiving duties that interrupted their sleep. This study was approved by the Emory University Institutional Review Board. Demographics are shown in Table 1.

Study Procedures

Participants were examined both during an initial weekday screening visit and a follow up visit, generally within three weeks of the screening visit. The screening visit occurred at different times of day. Height, weight and a random blood glucose sample also was taken at that time. At the follow-up visit, after a prior overnight fast, glycated hemoglobin was measured with standard techniques [15] (see Methods Supplement) and a standard 75 g oral glucose tolerance test was begun prior to 11 AM, with glucose sampled at baseline (prior to loading), and at one and two hours after the glucose load. We confined our analyses to values derived from two-hours post-loading. Impaired glucose was defined as a two-hour glucose ≥140 mg/dl. There were no restrictions on exercise prior to testing. During the visits, sleep was assessed by questionnaires.

Medical History

We defined presence of cardiovascular disease as reported history of heart attack, angioplasty or stent, cardiac bypass surgery, peripheral arterial surgery of the legs, stroke, transient ischemic attack ["mini-stroke"], or cardiac endarterectomy. Smoking was characterized by being a smoker over the preceding three months. We also defined concurrent usage of two medication classes: anti-hypertensives and cholesterol-lowering agents.

Sleep Questions (Table 2)

At the time of their screening visit, all participants answered a question about their habitual sleep duration (“Over the last six months, how many hours of sleep did you usually get at night?”). At the time of the follow-up oral glucose tolerance testing visit, participants were asked two additional questions about their sleep reflecting their reported recent sleep duration (“How many hours of sleep did you obtain last night?” and “How many hours of sleep did you obtain the night before last night?”) (see Table 2). All three sleep questions (one habitual, two recent) employed an identical, forced-choice response format. That is, participants were asked to use 30-minute anchors (e.g., 5, 5 ½, 6, 6 ½, etc), ranging from < 4 to > 10 (i.e., 14 distinct response categories) to make their response. Subjects verbalizing a response of durations of less than 4 or greater than 10 hours were instructed to select the lowest and highest anchors, respectively. We summed the responses to the two questions referring to the two nights before the oral glucose tolerance test to provide a summed recent sleep duration. We used sums (rather than the mean of two nights) to maintain comparability with prior epidemiologic studies, which have used integer-based formats with which to inquire about sleep durations.
**Statistical Analyses**

Our general approach to these analyses was to employ logistic regression to examine abnormalities in glucose control in relation to sleep duration and to demonstrate an additive effect between habitual and recent durations. We relied upon maximum likelihood estimation to minimize the influence of potential predictor variables that were non-normally distributed. This approach minimizes the impact of outliers on the regression models that tend to be problematic when employing least squares linear regression [16]. We also examined the adequacy of the logistic regression by examining residuals from the model using regression diagnostic statistics (Pearson residual calculation, leverage \( h_i \), DFBETAS and chi-square deletion difference) following standard computational techniques [17, 18].

We initially present associations between habitual sleep durations and an integrated measure of glycemic control, by visually presenting associations between 30-minute decrements in habitual sleep durations and the odds of elevations in glycated hemoglobin (see Figure 1). Next, based on the observed statistically significant threshold for habitual sleep duration for elevated glycated hemoglobin risk (i.e., < 7 hours), we present stratification analyses involving recent sleep durations for the two nights immediately prior to oral glucose tolerance testing among participants with glycated hemoglobin < 6.0% (< 42 mmol/mol). These analyses controlled for age, sex, race and body mass index (BMI) \((\text{kg/m}^2)\). Finally, we modeled possible protective associations on oral glucose tolerance testing of longer-than-usual sleep durations by examining those individuals whose nightly recent sleep durations exceeded their habitual sleep durations. Results are expressed as Odds Ratios (ORs) and 95% Confidence Intervals (CIs). For purposes of statistical completeness and computational derivations, we also included beta coefficients in our primary logistic model.

**Role of the Funding Source**

This work was sponsored by the United States National Institutes of Health, which had no role in the study design, the collection, analysis or interpretation of data, the writing of the current report, or the decision to submit for publication.

**RESULTS**

Demographics of the study population with complete sleep question data are shown in Table 1. We have reported elsewhere that, despite diabetes exclusion by history, approximately 10% of this population had glycated hemoglobin values \( \geq 6.0\% \) (\( \geq 42 \text{ mmol/mol} \)), including a number with frank diabetes \( (\geq 6.5\%) \) (\( \geq 48 \text{ mmol/mol} \)) \((n=33)\) and those at high risk \((6.0\% - 6.4\%)\) (42 – 46 mmol/mol) \((n=120)\). Patients with glycated hemoglobin values \( \geq 6.0\% \) (\( \geq 42 \text{ mmol/mol} \)) were older, more obese and more likely to be Black and female than those with values < 6.0% (< 42 mmol/mol). As expected, they also showed higher fasting glucose and higher glucose levels on oral glucose tolerance testing and were also more likely to have a history of cardiovascular disease, and use anti-hypertensive medication and cholesterol-lowering agents (Table 1). A small number of cases \((n=16)\) had incomplete data on selected demographics, but they did not differ from participants with complete \((n=1,559)\) data on age, BMI, gender composition or race. Mean (SD) levels for random and baseline (i.e., morning prior to oral glucose tolerance testing) glucose were 97.3 (18.0)

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*Am J Med. Author manuscript; available in PMC 2018 May 01.*
mg/dl and 93.8 (11.0) mg/dl, respectively, and these were correlated significantly ($r = 0.37$, $p < .0001$).

Table 3 shows results from the multivariate logistic regression model examining the association between habitual and recent sleep durations and the increased odds of an abnormal two-hour response on the oral glucose tolerance test. Both older age and higher BMI, as well as use of anti-hypertensive medication, were more likely to be associated with abnormal glucose control. Regression diagnostics, including examination of residuals and influence statistics [17, 18] indicated that the regression assumptions were not violated by inclusion of the relatively small number of outliers in the model. This analysis also demonstrated a statistically significant interaction between habitual and recent sleep durations. Based on the statistically significant interaction observed, we then employed a stratification approach to further understand the nature of this interaction.

Because habitual (c.f., recent) sleep duration should be more strongly associated with a more stable marker of glycemic control (glycated hemoglobin), we first examined the relationship between habitual sleep duration and glycated hemoglobin values $\geq 6.0\%$ ($\geq 42$ mmol/mol) (Figure 1). Associations between short habitual sleep duration and the odds for elevated glycated hemoglobin indicated that a threshold of less than 7 hours of habitual sleep was associated with values $\geq 6.0\%$ ($\geq 42$ mmol/mol) (Figure 1). This association became stronger as sleep durations shortened, even by 30-minute decrements. This relationship was observed down to durations of 4 or fewer hours of sleep (Figure 1). We next determined to what extent such habitual effects might be moderated by recent sleep durations occurring immediately prior to oral glucose tolerance testing.

Table 4 shows relationships between varying levels of recent sleep duration and elevations in post-ingestion glucose for those 636 individuals with a habitual sleep duration of less than 7 hours. For these individuals (all of whom demonstrated glycated hemoglobin $< 6.0\%$; $< 42$ mmol/mol), heightened “risk” for potentially recent adverse effects on glucose regulation were most apparent for those participants who report a sum of less than or equal to 11 hours of sleep for the two nights prior to oral glucose tolerance testing. A significant difference ($p < .05$) in 2-hour glucose value also occurred when this group of subjects was divided simply into those with a sum of less than 11 hours (mean [SD] glucose = 112.8 [34.4]) versus those with a sum of greater than 11 hours (mean [SD] glucose = 103.8 [31.9]) for the two nights prior to oral glucose tolerance testing. Sums of less than 9 hours of sleep did not reach statistical significance, probably because of lower statistical power inherent in the small numbers of participants reporting such very short, acute sleep durations. Also owing to small sample size, results among individuals with glycated hemoglobin $\geq 6.0\%$ ($\geq 42$ mmol/mol) were not significant. Finally, among those individuals obtaining 7 or greater habitual hours of sleep, OR’s were generally non-significant for all two-night sums of recent sleep durations.

We also repeated these analyses excluding 187 individuals who reported that they worked an “odd hours shift,” such as 3 to 11 PM or 11 PM to 7 AM, and the results were virtually identical, suggesting that shiftworkers did not differentially contribute to the reported results.
Similar analyses excluding women with hormone replacement therapy exposure showed comparable results to the population of women as a whole.

Because individuals with habitual short sleep durations evidenced higher odds for abnormal oral glucose tolerance testing for those nights in which also they reported short durations of sleep for the two nights prior to oral glucose tolerance testing, we also modeled a potential protective influence (as proof-of-concept) of longer-than-usual sleep durations in such individuals. For individuals who habitually slept 6 hours or less and had glycated hemoglobin values < 6.0% (< 42 mmol/mol), two-night recent sleep durations of greater than 13 hours total were protective (i.e., two-hour values < 140 mg/dl) (OR = 0.25, 95% CI 0.09–0.67) on morning oral glucose tolerance testing, relative to two-hour values for individuals who slept less than 11 hours total prior to their morning oral glucose tolerance testing.

CONCLUSIONS

Our results indicate that habitual and recent sleep durations interact with regards to glucose metabolism. More specifically, habitual sleep durations of less than 7 hours appear to lower the threshold for the glucose intolerance associated with shortened recent sleep durations. Longer-than-usual recent sleep durations may also be protective on oral glucose tolerance testing for individuals with short habitual sleep durations.

This study is broadly confirmatory of many others that have found short sleep durations to be associated with elevated glucose levels [1–8]. Perhaps somewhat surprising in our data was the robustness and the graded nature of the relationships observed, since subjective estimations of participants’ sleep durations maintained such relationships with a forced-choice temporal resolution of 30-minutes. The majority of epidemiologic studies that have demonstrated associations between short sleep durations and glucose intolerance have categorized sleep by one-hour decrements (e.g., [1–3; 5]). The fact that participants’ answers reflect subtle gradations suggests that, although estimates of sleep durations are prone to error, individuals’ ability to make discriminations as specific as 30 minutes apparently hold considerable meaning. Our data indicated that compromised glucose control also could be detected in association with short sleep in individuals who fall within the normal range of glycated hemoglobin but who were unable to obtain at least a modest amount of sleep the nights before their oral glucose tolerance test (Table 4). This finding is consistent with many experimental studies showing that restriction of sleep or interruption of certain stages of sleep interferes with insulin response to a glucose load in healthy young adults [9–13]. Our data imply that such immediate effects of sleep restriction on glucose dysregulation are also observed in a relatively large, diverse population and are likely not to be confounded by age, race, BMI or sex.

Some epidemiologic studies [1–5] have shown elevated risk for poor glucose control with longer, as well as shorter, sleep durations. We did not observe this association in our data, possibly owing to the small cell sizes for individuals reporting longer sleep intervals. However, we were able to model longer-than-usual recent sleep durations as conferring potential protection for an abnormal glucose response. Whether such an influence could be
demonstrated in a laboratory situation remains to be seen, although a return to normal insulin resistance was noted in studies when sleep-restricted subjects were allowed recovery or normal duration sleep [10,11]. Recent data have suggested that in young, healthy subjects, extending sleep duration by an average of about 45 minutes per night was associated with improvement in insulin sensitivity [19], and as little as two nights of extended sleep following severe acute sleep loss were associated with recovery of the disposition index, reflecting both insulin sensitivity and the acute response of insulin to glucose infusion [20]. Lengthening the initial period of sleep (prior to first voiding episode) was also shown to have a beneficial effect on lowering blood glucose in a retrospective analysis [21]. We must consider our apparent protective effect of recent longer sleep as hypothesis-generating rather than conclusive, as no large-scale clinical trials have yet shown that improving sleep duration pharmacologically or behaviorally exerts a salutary influence on glucose control.

Our study has limitations. The validity of self-reported data about sleep durations have been questioned, though some studies suggest modest validity [22–25], and the latter is also supported by our findings of graded associations. We also did not examine data reflecting sleep quality, sleep fragmentation or sleep depth, all of which represent separate components of reported sleep, apart from sleep duration [26]. Future studies could expand these findings by including such additional measures of the subjective components of sleep. Elsewhere we have argued that reported sleep duration may be somewhat of a proxy for overall health and general well-being [27], rather than a true quantitative marker. Laboratory studies have clearly shown that above and beyond sleep duration, fragmentation of sleep can be associated with glucose dysregulation. For example, interruption of slow wave sleep in the beginning of the night is associated with a 25% decrease in insulin sensitivity [9]. Rapid eye movement sleep, which is otherwise associated with a decrease in interstitial glucose, may be impaired by sleep apnea occurring in that stage of sleep [28], and in one study, decreased levels of both slow wave sleep and rapid eye movement sleep were both associated with reduced glucose control [29]. Our study did not include measures such as these.

Another limitation in our data is the absence of direct measurement of sleep apnea as a potential confounder in our results. In a subject population of this average age and BMI, sleep apnea, known to be associated with abnormalities in glucose control [30], is likely to be highly prevalent. To what extent the very specific graded associations between habitual and acute sleep durations that we have demonstrated here might be influenced by participants also having sleep apnea is uncertain. However, to the extent that our analyses were multivariate and accounted for age and BMI (as well as other sleep apnea risk factors, such as sex and Black race), we may have minimized the impact of such confounders (as partial proxies for sleep apnea) in our results.

It is somewhat surprising how, in the context of our data, relatively small differences in response on a questionnaire (i.e., 30-minute intervals) can impact the strength of associations so substantially. Psychometric theory suggests that when people are asked to make fine discriminations they are able to do so [31], and it may well be that detailed responses to a question about sleep duration can be more meaningful than may otherwise appear. There are indeed precedents for within subjects’ changes in reported sleep durations holding clinical significance. In the Whitehall II study, Ferrie and colleagues [32], for
example, have shown that changing intervals in reported sleep durations of one or two hours
over five years were significant predictors of adverse outcomes, which argues for validity of
self-reported differences in such measures. Our data imply that, on a population-wide basis,
individuals’ knowledge of their own sleep durations represents an important aspect of
modifiable health behavior.

Perhaps the most far-reaching and speculative implication of our data involves glucose
tolerance testing. If oral glucose tolerance test results can be skewed by failure of subjects to
be adequately rested before the test is performed (defined here as at least 11 hours of
summed sleep on the two nights prior to the test), then results could be subject to false
positive error. Parallels to overnight fasting are obvious. Just as a non-fasting oral glucose
tolerance testing can generate falsely elevated glucose levels, short sleep may similarly
invalidate test results. Other studies, relying on both on self-report in the field and/or
involving laboratory-based polysomnographic measurements, should attempt to replicate
this aspect of our findings in other population-based samples. Until then, at the very least,
we suggest that patients being evaluated with a traditional oral glucose tolerance test be
questioned, not only about whether they have fasted, but also about the number of hours that
they have slept in the two nights before their morning test.

METHODS SUPPLEMENT

Details of Measurements

Height was measured with stadiometer after shoes were removed. Weight was measured
using a digital scale with participants in light clothing. Measurements of glycated
hemoglobin were performed with a turbidometric, immunoassay (Beckman Coulter
Synchron LX Hemoglobin A1c assay; Beckman-Coulter, Brea, CA). Glucose was analyzed
using sodium-fluoride/oxalate preservative and plasma frozen at − 80°C within 30 minutes
and also were analyzed on the Synchron LX. Impaired glucose tolerance was defined as a
two-hour oral glucose tolerance testing ≥ 140 mg/dl. Elevated glycated hemoglobin was
defined by ≥6.0% (≥42 mmol/mol).

Acknowledgments

Funding

This work was supported in part by NIH awards DK066204 (LSP), DK099716 (LSP), NS050595 (DLB),
AG041543 (MKS), and UL1 RR025008 (LSP); VA award HSR&D IIR 07-138 (LSP), Cystic Fibrosis Foundation
award PHILLI12A0 (LSP); Emory Neuroscience Initiative Award (DLB); and the United States Department of
Veterans Affairs (LSP). The funding organizations had no role in the design and conduct of the study; collection,
management, analysis, and interpretation of the data; and preparation, review, or approval of the manuscript.

Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>Body Mass Index</td>
</tr>
<tr>
<td>OR</td>
<td>Odds Ratio</td>
</tr>
<tr>
<td>95% CI</td>
<td>95% Confidence Intervals</td>
</tr>
</tbody>
</table>
References


Clinical Significance

- Experimental studies show that sleep curtailment disrupts glycemic control
- These studies used small, selected samples with potentially low generalizability
- In a population of 1,559 adults, short sleep 2 nights before OGTT may bias results
- This untoward effect on glucose control is moderated by habitual sleep duration
- Forced choice estimation of sleep durations made with 30-minute increments may have meaning for control of glucose
Figure 1. Habitual Sleep Duration in Relation to Elevated Glycated Hemoglobin
Odds ratios for Glycated Hemoglobin ≥6.0% (≥42 mmol/mol) for habitual reported sleep duration by 30-minute forced choice increments (n = 1,559). Asterisks refer to statistically significant odd ratios for the habitual sleep duration thresholds shown on the x-axis. Error bars represent 95% confidence intervals for the odds ratios.
## TABLE 1

Demographic and Clinical Measures on Study Population (n = 1,559)

<table>
<thead>
<tr>
<th>Demographic/Clinical Measure</th>
<th>Total (n=1,559)</th>
<th>Glycated Hemoglobin &lt; 6% (&lt; 42 mmol/mol) (n=1,406)</th>
<th>Glycated Hemoglobin ≥ 6% (≥ 42 mmol/mol) (n=153)</th>
<th>t or χ²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>47.9 ± 12.1</td>
<td>47.4 ± 12.2</td>
<td>52.7 ± 9.5</td>
<td>6.3</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Body Mass Index (kg/m²)</td>
<td>30.3 ± 6.8</td>
<td>29.7 ± 6.4</td>
<td>35.6 ± 7.8</td>
<td>8.9</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Race (% Black)</td>
<td>58.1 ± ---</td>
<td>54.8 ± ---</td>
<td>88.2 ± ---</td>
<td>63.5</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Sex (% Female)</td>
<td>58.2 ± ---</td>
<td>57.1 ± ---</td>
<td>68.6 ± ---</td>
<td>7.5</td>
<td>.006</td>
</tr>
<tr>
<td>Cardiovascular Disease History (% positive)</td>
<td>3.9 ± ---</td>
<td>3.6 ± ---</td>
<td>6.6 ± ---</td>
<td>3.44</td>
<td>.064</td>
</tr>
<tr>
<td>Current Smoker (% positive)</td>
<td>13.9 ± ---</td>
<td>13.1 ± ---</td>
<td>21.7 ± ---</td>
<td>8.49</td>
<td>.511</td>
</tr>
<tr>
<td>Anti-hypertensive medication (% using)</td>
<td>28.7 ± ---</td>
<td>26.2 ± ---</td>
<td>51.6 ± ---</td>
<td>43.5</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Cholesterol lowering medication (% using)</td>
<td>8.4 ± ---</td>
<td>8.3 ± ---</td>
<td>9.8 ± ---</td>
<td>0.43</td>
<td>.004</td>
</tr>
<tr>
<td>Glycated Hemoglobin (%)</td>
<td>5.43 ± 0.46</td>
<td>5.3 ± 0.3</td>
<td>6.3 ± 0.6</td>
<td>20.2</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Fasting glucose (mg/dl)</td>
<td>95.4 ± 13.6</td>
<td>93.8 ± 11.1</td>
<td>110.3 ± 22.5</td>
<td>8.9</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>2-hr post-loading glucose (mg/dl)</td>
<td>112.6 ± 40.2</td>
<td>107.8 ± 34.5</td>
<td>156.7 ± 58.5</td>
<td>10.2</td>
<td>&lt;.0001</td>
</tr>
</tbody>
</table>
TABLE 2

Format of 30-minute Forced Choice Sleep Duration Questions Assessing both Habitual and Recent Sleep Durations.

<table>
<thead>
<tr>
<th>For Habitual Sleep Duration:</th>
<th>Over the last six months, how many hours of sleep do you usually get at night?</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 or less</td>
<td>4.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>For Recent Sleep Duration One Night Prior to Oral Glucose Tolerance Testing:</th>
<th>How many hours of sleep did you get last night?</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 or less</td>
<td>4.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>For Recent Sleep Duration Two Nights Prior to Oral Glucose Tolerance Testing:</th>
<th>How many hours of sleep did you get the previous night?</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 or less</td>
<td>4.5</td>
</tr>
</tbody>
</table>
Table 3
Multivariate Logistic Regression Predicting Elevated Two-hour Plasma Glucose (≥140 mg/dl) during Oral Glucose Tolerance Testing among Entire Study Population Including Both Habitual and Recent Sleep Durations

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds Ratio</th>
<th>Lower Bound 95% Confidence Interval</th>
<th>Upper Bound 95% Confidence Interval</th>
<th>Beta coefficient</th>
<th>Std error</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2.4771</td>
<td>.3098</td>
</tr>
<tr>
<td>Habitual hrs of sleep (hrs)</td>
<td>0.57</td>
<td>0.28</td>
<td>1.17</td>
<td>—</td>
<td>.3666</td>
<td>.1279</td>
</tr>
<tr>
<td>Recent hrs of sleep (hrs)</td>
<td>0.63</td>
<td>0.43</td>
<td>0.92</td>
<td>—</td>
<td>.1924</td>
<td>.0162</td>
</tr>
<tr>
<td>Age (years)</td>
<td>1.04</td>
<td>1.02</td>
<td>1.05</td>
<td>—</td>
<td>.0075</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Race (ref = white)</td>
<td>1.04</td>
<td>0.75</td>
<td>1.45</td>
<td>—</td>
<td>.0835</td>
<td>.7994</td>
</tr>
<tr>
<td>Sex (ref = male)</td>
<td>0.78</td>
<td>0.57</td>
<td>1.07</td>
<td>—</td>
<td>.0811</td>
<td>.1231</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>1.06</td>
<td>1.03</td>
<td>1.08</td>
<td>—</td>
<td>.0123</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Cardiovascular Disease (ref = no cardiovascular disease)</td>
<td>1.04</td>
<td>0.50</td>
<td>2.18</td>
<td>.0434</td>
<td>.3759</td>
<td>.9081</td>
</tr>
<tr>
<td>Smoking (ref = not smoking)</td>
<td>0.99</td>
<td>0.63</td>
<td>1.56</td>
<td>—</td>
<td>.2328</td>
<td>.9585</td>
</tr>
<tr>
<td>Anti-hypertensive medication (ref = not using)</td>
<td>1.64</td>
<td>1.17</td>
<td>2.31</td>
<td>.9467</td>
<td>1.726</td>
<td>.0040</td>
</tr>
<tr>
<td>Cholesterol-lowering medication (ref = not using)</td>
<td>0.78</td>
<td>0.43</td>
<td>1.40</td>
<td>.2508</td>
<td>.3009</td>
<td>.4047</td>
</tr>
<tr>
<td>Interaction term (Recent x Habitual)</td>
<td>1.06</td>
<td>1.01</td>
<td>1.12</td>
<td>.0608</td>
<td>.0275</td>
<td>.0277</td>
</tr>
</tbody>
</table>
### Table 4

Odds Ratios for Abnormally Elevated Two-hour Plasma Glucose (≥ 140 mg/dl) in Relation to Summed Recent Sleep Durations for the Two Nights Prior to Oral Glucose Tolerance Testing in Participants with Glycated Hemoglobin < 6.0% (< 42 mmol/mol) and Less than Seven Hours of Habitual Sleep (n = 636)

<table>
<thead>
<tr>
<th>Two-night Sum: Reported Recent Duration (hours)</th>
<th>Number of Cases Exceeding Reported Recent Sleep Duration</th>
<th>Odds Ratio</th>
<th>Lower Bound 95% CI</th>
<th>Upper Bound 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 17.5</td>
<td>632</td>
<td>0.13</td>
<td>0.02</td>
<td>1.02</td>
</tr>
<tr>
<td>&lt; 17.0</td>
<td>630</td>
<td>0.32</td>
<td>0.05</td>
<td>1.91</td>
</tr>
<tr>
<td>&lt; 16.5</td>
<td>628</td>
<td>0.27</td>
<td>0.06</td>
<td>1.24</td>
</tr>
<tr>
<td>&lt; 16.0</td>
<td>619</td>
<td>0.68</td>
<td>0.19</td>
<td>2.50</td>
</tr>
<tr>
<td>&lt; 15.5</td>
<td>614</td>
<td>0.70</td>
<td>0.23</td>
<td>2.17</td>
</tr>
<tr>
<td>&lt; 15.0</td>
<td>600</td>
<td>0.80</td>
<td>0.32</td>
<td>2.04</td>
</tr>
<tr>
<td>&lt; 14.5</td>
<td>585</td>
<td>0.90</td>
<td>0.40</td>
<td>2.03</td>
</tr>
<tr>
<td>&lt; 14.0</td>
<td>539</td>
<td>1.26</td>
<td>0.65</td>
<td>2.45</td>
</tr>
<tr>
<td>&lt; 13.5</td>
<td>480</td>
<td>1.33</td>
<td>0.77</td>
<td>2.31</td>
</tr>
<tr>
<td>&lt; 13.0</td>
<td>408</td>
<td>1.12</td>
<td>0.70</td>
<td>1.80</td>
</tr>
<tr>
<td>&lt; 12.5</td>
<td>363</td>
<td>1.39</td>
<td>0.87</td>
<td>2.21</td>
</tr>
<tr>
<td>&lt; 12.0</td>
<td>269</td>
<td>1.37</td>
<td>0.87</td>
<td>2.15</td>
</tr>
<tr>
<td>&lt; 11.5</td>
<td>217</td>
<td>1.82</td>
<td>1.15</td>
<td>2.87</td>
</tr>
<tr>
<td>&lt; 11.0</td>
<td>162</td>
<td>1.83</td>
<td>1.14</td>
<td>2.96</td>
</tr>
<tr>
<td>&lt; 10.5</td>
<td>118</td>
<td>1.97</td>
<td>1.18</td>
<td>3.30</td>
</tr>
<tr>
<td>&lt; 10.0</td>
<td>64</td>
<td>2.12</td>
<td>1.11</td>
<td>4.03</td>
</tr>
<tr>
<td>&lt; 9.5</td>
<td>44</td>
<td>3.11</td>
<td>1.52</td>
<td>6.35</td>
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<tr>
<td>&lt; 9.0</td>
<td>22</td>
<td>2.46</td>
<td>0.89</td>
<td>6.77</td>
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<tr>
<td>&lt; 8.5</td>
<td>16</td>
<td>2.26</td>
<td>0.68</td>
<td>7.54</td>
</tr>
</tbody>
</table>

Data shown for individuals reporting habitual sleep durations < 7.0 hours; Odds Ratios adjusted for age, sex, body mass index and race; no participants reported a two-night sum of recent sleep durations less than 8.0 hours.