Two Nights of Recovery Sleep Reverses the Effects of Short-term Sleep Restriction on Diabetes Risk

Josiane L. Broussard,[1] Kristen Wroblewski,[2] Jennifer M. Kilkus,[3] and Esra Tasali[3]

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Sleep restriction is associated with insulin resistance and an increased risk for type 2 diabetes (1–3). Here, we investigated whether only 2 nights of recovery sleep, as may occur on weekends, reverses the negative effects of short-term sleep restriction on glucose homeostasis.

Nineteen healthy young lean men were studied under controlled laboratory conditions during normal sleep and sleep restriction in a randomized order, as previously reported (1,2,4). The institutional review board of The University of Chicago approved the protocol, and all participants gave written informed consent. During normal sleep, participants were allowed 8.5 h in bed (2300–0700) for 4 consecutive nights. During sleep restriction, participants were allowed 4.5 h in bed (0100–0530) for 4 consecutive nights, immediately followed by recovery sleep for 2 consecutive nights with 12 h in bed on the first night (2200–1000) and 10 h in bed on the second night (2200–0800). A frequently sampled intravenous glucose tolerance test (ivGTT) was performed at 1000 after 4 nights of normal sleep, 4 nights of sleep restriction, and 2 nights of recovery sleep to assess insulin sensitivity, acute insulin response to glucose, and disposition index (i.e., insulin sensitivity $\times$ acute insulin response to glucose). Participants received standardized meals during the 24 h prior to each ivGTT. We previously reported the effects of sleep restriction versus normal sleep on sleep stages, insulin sensitivity, and insulin response from this cohort (1,2,4). The effects of sleep condition on glucose homeostasis were assessed using a mixed-effects regression model.

On average, participants slept 7.8 $\pm$ 0.1 h during normal sleep, 4.3 $\pm$ 0.02 h during sleep restriction, and 9.7 $\pm$ 0.2 h during sleep recovery ($P < 0.001$; all data are mean $\pm$ SEM). Weight measured prior to each ivGTT was similar between sleep conditions ($P = 0.21$). Insulin sensitivity was reduced by 23% after sleep restriction relative to normal sleep, which improved after recovery sleep (Fig. 1*A*). Acute insulin response to glucose did not differ between conditions (Fig. 1*B*). Disposition index was reduced by 16% following sleep restriction relative to normal sleep, consistent with increased diabetes risk, which reverted back to normal sleep levels after recovery sleep (Fig. 1*C*).

A common question is whether, and how quickly, an individual can recover from the adverse effects of sleep loss on glucose homeostasis. We have demonstrated that 2 nights of recovery sleep averaging nearly 10 h per night following 4 nights of sleep restriction in healthy young lean men is sufficient to improve insulin sensitivity and restore disposition index (a marker of diabetes risk) to the levels observed after normal sleep. Our findings suggest that catching up on sleep can reverse the negative metabolic effects of short-term sleep restriction. These data are clinically relevant because such sleep patterns (i.e., short-term sleep restriction on workdays and recovery sleep on weekends) are quite common in modern society (5). Future studies in real-world settings are needed to investigate whether catching up on sleep could be an effective behavioral intervention in the prevention and management of type 2 diabetes.

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[1]*Department of Integrative Physiology, University of Colorado Boulder, Boulder, CO*
[2]*Department of Public Health Sciences, The University of Chicago, Chicago, IL*
[3]*Department of Medicine, The University of Chicago, Chicago, IL*

Corresponding author: Josiane L. Broussard, josiane@uchicago.edu.

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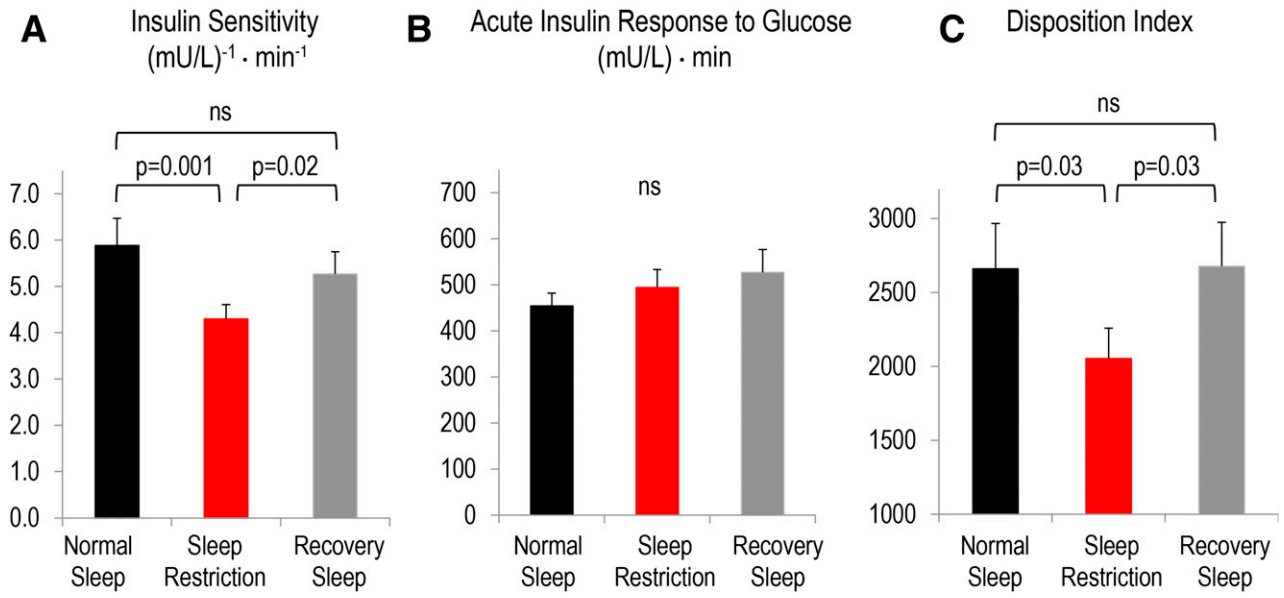

Figure 1—Effects of normal sleep (black bars), sleep restriction (red bars), and recovery sleep (gray bars) on insulin sensitivity (*A*), acute insulin response to glucose (*B*), and disposition index (*C*). The disposition index is insulin sensitivity × acute insulin response and is a marker of diabetes risk. Data are mean ± SEM. Overall *P* values for sleep condition were $P = 0.003$ for insulin sensitivity, $P = 0.19$ for acute insulin response to glucose, and $P = 0.047$ for disposition index. The effects of sleep condition insulin sensitivity, acute insulin response to glucose, and disposition index were assessed using a mixed-effects linear regression model using restricted maximum likelihood with a small-sample adjustment to hypothesis tests using the Kenward and Roger method.

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