



## Out of Morpheus' embrace

“Poor sleep causes diabetes.” It almost reads like a grocery store tabloid headline. Yet Drs. Carol Touma and Silvana Pannain, in this issue of the *Journal* (page 549), review several studies and some underlying physiologic concepts that strongly link disturbed sleep with type 2 diabetes.

Much of the data are cross-sectional and epidemiologic, so the direction of causation (if causation exists) cannot be established with certainty. There is a host of interwoven confounders, and many of these intersect around the patient's weight and the presence of sleep apnea. Nevertheless, the authors explore some provocative associations.

Over the years, clinicians have increasingly recognized the myriad of comorbidities that accompany sleep apnea. We have discussed this in the *Journal* on several occasions since 2005. Naïvely, I have attributed many of these, particularly the cardiac complications, to downstream effects of repetitive hypoxic and hypercarbic insults, but there may be more fundamental physiologic principles in play, some linked to the affected sleep cycle and not to the apnea.

Drs. Touma and Pannain discuss some of the physiologic consequences of altered or decreased sleep cycles. Some of these are a result of disrupting the circadian release of hormones such as glucocorticoids and growth hormone, both of which can influence the body's sensitivity to insulin's hypoglycemic effects. The same can be said for disruption of normal sympathetic-parasympathetic nerve flow. In addition, sleep disruption affects appetite. Thinking back to residency, I recall the need to follow the admonition of one of my peers: in order to survive nights on call, never miss a meal. I still remember the (leptin-linked?) cravings after being up all night for a heavy carbohydrate-laden breakfast. Given these effects, coupled with the fatigue of sleep deprivation resulting in decreased exercise, it is easy to construct innumerable positive feedback loops contributing to the development of insulin resistance and type 2 diabetes.

So while it is a truism that sleep is good and that we all need to “recharge our batteries,” we still lack a full understanding of the complex physiology of sleep and the effects of sleep deprivation on a number of clinical conditions, from diabetes to fibromyalgia.

Recognizing the associations is a beginning. Knowing what to do about defective sleep in terms of preventing or ameliorating disease awaits appropriately controlled interventional trials—and the definition of appropriate interventions to evaluate.

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