

been demonstrated to be associated with increased circulating levels of soluble endothelial adhesion molecules (4), indicating the presence of an early proinflammatory activation of vascular endothelium (5), probably because of augmented pressure overload throughout 24 h. In addition, blunted nocturnal BP decline is combined with increased generation of reactive oxygen species (6), which are powerful mediators of atherogenesis (5). These data demonstrate that an impaired BP drop has the potential to affect vascular biology, uncovering a proinflammatory phenotype.

Thus, we propose that changes in circadian BP profile due to sleep deprivation should also be taken into account to explain the increased circulating levels of hs-CRP observed after sleep deprivation in healthy subjects (1).

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REPLY

The letter by Dr. Desideri and colleagues in the *Journal* raises some interesting issues. Whereas our study (1) was performed on men and did not record 24-h blood pressure (BP), studies are currently being conducted by our group to investigate BP and individual differences with respect to adiposity in the inflammatory response to sleep loss in men and women.

The issue of nocturnal dipping is important, and as the investigators state, several studies have reported that hypertensive patients with nondipping BP profiles have a worse cardiovascular prognosis compared with dippers. The fact

that there is considerable night-to-night variability in this non-dipping pattern, and that it is associated on a given night with self-perceived sleep quality (2), is intriguing. Desideri et al. (3) found elevated cellular adhesion molecules in hypertensive nondippers, and it would certainly be of interest to know about sleep quality and quantity in their subjects. Lusardi et al. (4) found that a single night of sleep reduced to 4 h increased BP in hypertensive patients.

The effects of chronic sleep disruption or inadequate sleep on BP and development of hypertension remain to be studied. We do not know how acutely the inflammatory (and clotting) systems can be activated by sleep loss, nor do we know how long they take to return to normal after being activated. The cumulative effects and clinical consequences of inadequate sleep over months or years on the inflammatory system, as likely occurs in the “real world,” are not known. In patients with sleep-disordered breathing, successful treatment with continuous positive airway pressure (CPAP) has been shown not only to reduce C-reactive protein (CRP) levels (5) but also to restore the dipper profile (6,7) to nondipper subjects.

In our study of acute total sleep deprivation in healthy men, we had BP data for only 8 of the 10 subjects, and it is not surprising, owing to the variability of this measure, that we did not see statistically significant increases in BP after one night. In contrast, CRP was significantly increased after a single night, in part because of its lower variability as a measure. This difference in variability of measures by no means diminishes the possibility that the autonomic system, related to and/or via its effects on BP, leads to the activation of the inflammation, including the coagulation system.

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