OUTSIDE OF THE TROPICS, winter can pose a significant threat to survival because food availability wanes as the ambient temperatures decrease and energy requirements for thermoregulation increase. Many animals cope with this seasonal energetic shortfall by engaging multiple winter adaptations to conserve energy, including cessation of reproduction and territorial defense, changes in body mass, increased food hoarding and nest building, reduced locomotor activities, increased fur development, and increased digestive efficiency (8). Some animals also enter a period of deep torpor (hibernation) to conserve sufficient energy to increase the odds of survival (7). Individuals of other species, such as Siberian hamsters (*Phodopus sungorus*), display daily bouts of shallow torpor, during which body temperature may be reduced from 37°C to ~15–20°C for 4–8 h during the rest phase of the daily locomotor activity cycle (6). The phenomenon of torpor has been known for some time. For example, writing in *Goldsmith’s Natural History* published in 1845, Mrs. Mary Pilkington notes, “Strictly speaking, . . . these animals cannot be said to sleep during the winter; it may be called rather a torpor, a stagnation of all the faculties” (p. 443). The energetic savings associated with torpor are substantial; torpid Siberian hamsters can save ~20% of their daily energy expenditures (10).

Despite its adaptive value, the physiological cues that initiate torpor remain unspecified. In some rodent species, such as Turkish hamsters (*Mesocricetus auratus*) and European hamsters (*Cricetus cricetus*), reduced ambient temperature is sufficient to induce hibernation (4, 5). In other species, exposure to short day lengths (and the associated increase in the duration of nocturnal melatonin) predisposes animals to enter hibernation or to display daily torpor (8). Indeed, regression of the reproductive system and subsequent decrease in testosterone appears to be a prerequisite for the display of torpor for male mammals (4, 9), but reduced testosterone is not the exclusive trigger because females also display torpor.

In this issue of the *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, Freeman and colleagues (3) attempted to address the question of what physiological factors initiate torpor. Specifically, these researchers tested the hypothesis that low leptin concentrations are necessary to permit Siberian hamsters to enter torpor. In addition to reproductive retrogression, short photoperiods diminish food consumption, which provokes a progressive loss of body mass (primarily body fat) in this species. Because torpor only occurs when body fat and thus leptin concentrations are at their nadir, the authors proposed a role for leptin in the regulation of torpor. In support of this notion, all hamsters in their study displaying nightly torpor had low blood leptin concentrations. To examine the causal link between low leptin and torpor, osmotic minipumps, which secreted either a constant release of leptin or vehicle, were implanted into Siberian hamsters displaying torpor. After 14 days, leptin treatment eliminated torpor in most, but not all, treated hamsters. Freeman and colleagues concluded that low leptin concentrations were necessary, but not sufficient, for evoking torpor.

Evidence that reduced leptin concentrations did not directly trigger torpor includes 1) leptin concentrations did not significantly differ between torpid hamsters and hamsters that never entered torpor in the cold, 2) leptin concentrations did not differ significantly within the same hamsters during torpid and nontorpid days, 3) leptin concentrations remained unchanged during the transitions between torpor and euthermia, and 4) the lowest three leptin values in the study were in food-deprived hamsters that never entered torpor. In addition to failing to trigger torpor, reduced leptin concentrations also failed to affect the duration or depth of torpor. On the basis of their findings, the authors propose that low circulating leptin concentrations provide a permissive, “starvation signal” to allow the onset of torpor.

This well-crafted study by Freeman and coworkers (3) rules out leptin as the physiological trigger for torpor; however, it does not point to the factor(s) that initiates torpor. It also raises the interesting question of why some individuals appear to be uncoupled from the permissive effects of leptin on torpor. Although this variation may simply reflect the choice of leptin dose (e.g., a “peri-threshold” dose), torpor may reflect an exaggerated drop in the sleep-time reduction in body temperatures (2), and the circadian minima in body temperatures can be blocked by leptin administration (1, 11). Thus it remains possible that the mechanism triggering torpor may comprise two separate systems, one that is signaled by leptin and another system mediated by, as of yet, unidentified mechanisms. If true, then this study could point the way to understanding the permissive effects of low leptin on the initiation of torpor. Although additional studies are required to investigate the individual differences in the effects of leptin on torpor, these results provide an important first step in elucidating the precise role of leptin on torpor.

**REFERENCES**


