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Torpor and hypothermia: reversed hysteresis of metabolic rate and body temperature

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Geiser F, Currie SE, O'Shea KA, Hiebert SM. Torpor and hypothermia: reversed hysteresis of metabolic rate and body temperature. *Am J Physiol Regul Integr Comp Physiol* 307: R1324–R1329, 2014. First published September 24, 2014; doi:10.1152/ajpregu.00214.2014.—Regulated torpor and unregulated hypothermia are both characterized by substantially reduced body temperature (T_b) and metabolic rate (MR), but they differ physiologically. Although the remarkable, medically interesting adaptations accompanying torpor (e.g., tolerance for cold and ischemia, absence of reperfusion injury, and disuse atrophy) often do not apply to hypothermia in homeothermic species such as humans, the terms "torpor" and "hypothermia" are often used interchangeably in the literature. To determine how these states differ functionally and to provide a reliable diagnostic tool for differentiating between these two physiologically distinct states, we examined the interrelations between T_b and MR in a mammal (*Sminthopsis macroura*) undergoing a bout of torpor with those of the hypothermic response of a similar-sized juvenile rat (*Rattus norvegicus*). Our data show that under similar thermal conditions, *1*) cooling rates differ substantially (approximately fivefold) between the two states; *2*) minimum MR is approximately sevenfold higher during hypothermia than during torpor despite a similar T_b; *3*) rapid, endogenously fuelled rewarming occurs in torpor but not hypothermia; and *4*) the hysteresis between T_b and MR during warming and cooling proceeds in opposite directions in torpor and hypothermia. We thus demonstrate clear diagnostic physiological differences between these two states that can be used experimentally to confirm whether torpor or hypothermia has occurred. Furthermore, the data can clarify the results of studies investigating the ability of physiological or pharmacological agents to induce torpor. Consequently, we recommend using the terms "torpor" and "hypothermia" in ways that are consistent with the underlying regulatory differences between these two physiological states.

hypothermia; torpor; metabolic rate; hysteresis; body temperature

TORPOR (daily torpor and hibernation) and hypothermia in mammals and birds are both characterized by substantial reductions in body temperature (T_b), metabolic rate (MR), and other physiological processes (5, 11, 12, 16, 30). However, torpor is a highly controlled process, whereas pathogenic hypothermia is an uncontrolled state of low T_b induced by low ambient temperatures (T_a), depletion of metabolic fuels, or substances that interfere with heat production or thermoregulation. Nevertheless, scholarly works (e.g., 19, 38), textbooks (e.g., 1, 17, 20, 24), and nonspecialists (e.g., 27, 34) often use these terms in ways that can confuse readers.

Medical researchers have long recognized the benefits to biomedical science of understanding how the tissues of animals capable of torpor, unlike those of homeotherms such as humans, can tolerate low body temperatures and ischemia, potentially damaging conditions experienced during organ preservation, stroke, and cardiac arrest (5, 10). If they want to understand and apply these astonishing physiological adaptations, medical researchers must be able to recognize the differences between torpor and hypothermia.

Currently, there is no method allowing an unambiguous distinction between these two states apart from the ability to rewarm spontaneously from low T_b and to maintain a T_b-T_a differential during torpor at low T_a. We therefore aimed to compare T_b and MR during both torpor and hypothermia to provide a reliable diagnostic tool for clear differentiation. We compared adult dunnarts (*Sminthopsis macroura*), known to exhibit daily torpor (14), with similarly sized rat (*Rattus norvegicus*) pups at 10–16 days of age. Although these pups were homeothermic in the nest, hypothermia was easily induced by separating pups from littermates and reducing T_a from ~23 to ~16°C; hypothermia was later reversed by increasing T_a to ~29°C to induce passive warming.

METHODS

All animal procedures were performed in accordance with Australian National Health and Medical Research Council guidelines and were conducted under protocols submitted to and approved by the University of New England Animal Ethics Committee.

Adult stripe-faced dunnarts (*Sminthopsis macroura, n* = 12, body mass = 23.2 ± 1.5 g) of both sexes were obtained from a laboratory colony at the University of New England held under a light-dark 12:12 photoperiod at T_a of 20°C and maintained and fed as described (14). To quantify interrelations between T_b and rate of oxygen consumption, we measured both variables simultaneously. Because rate of oxygen consumption is directly proportional to MR, it is referred to as "index of MR" in the figures. To induce torpor, we placed animals in 0.75-liter respirometry chambers (T_a = 18.4 ± 0.7°C) situated within a temperature-controlled cabinet. Measurements began in the late afternoon and lasted for ~23 h. Metabolic rates and corresponding body temperatures were averaged over at least 36 min when both had reached a stable plateau. Dunnarts did not have access to food or water during this procedure.

The flow rate (~400 ml/min) of dry air through the respirometry chamber was measured with a mass flowmeter (FMA-5606, Omega, Stamford, CT). Oxygen content of excurrent air was measured with an oxygen analyzer (FOX, Sable Systems, Las Vegas, NV). Solenoid valves were used to regulate airflow among respirometry channels (two or three for respirometers containing animals and one reference channel), so that oxygen content of outflowing air was determined sequentially for 3 min per channel every 9 min (if two animal channels) or 12 min (if three animal channels). Wax-coated (Paraffin/Elvax, Mini-Mitter, Bend, OR) temperature-sensitive transmitters (model X-M, ~1.2 g, Mini-Mitter) were calibrated between 10 and 40°C to the nearest 0.1°C against a precision mercury thermometer in a water bath. The transmitters were then implanted intraperitoneally

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under oxygen-isoflurane anesthesia using a previously described procedure (29). Animals were allowed to recover for at least 1 wk before measurements began.

Rat pups (Wistar, Animal Research Centre, Perth; $n = 10$, body mass = 25.6 ± 4.0 g) of both sexes, obtained from the breeding colony at University of New England, were studied at 10 to 16 days of age for comparison. Measurements of rate of oxygen consumption in individual rats were conducted as for *S. macroura*. T_b was measured rectally with a fine, calibrated thermocouple probe that was read to the nearest 0.1°C with a digital microprocessor (HH-71 T, Omega, Stamford, CT). The probe was inserted ~1.5 cm and secured to the tail with surgical tape, and T_b was recorded at 15-min intervals. We did not use transmitters for the rat pups because recovery from surgery would have required too much time and high growth rates would have prevented our measurements. For experiments rat pups were separated from littermates in the morning and individually marked; measurements lasted for up to 9 h, after which time pups were returned to the litter. Animals did not have access to food or water during this procedure. Rat pups were initially held at T_a of 23.3 ± 0.5°C. To induce hypothermia, T_a was lowered to and then maintained at 16.1 ± 1.1°C (see Fig. 1*B*) until MR had stabilized over at least 45 min. Because rat pups could not raise T_b and MR even after disturbance, T_a was then raised to 28.8 ± 1.0°C to allow passive rewarming. T_a in the respirometry chamber was measured to the nearest 0.1°C from a calibrated thermocouple in the respirometry chamber. Thermocouple output was amplified by a digital microprocessor (Omega DP116, Stamford, CT).

Student's *t*-tests (Minitab, v. 13.1) were used to compare measures between bouts of torpor in dunnarts and bouts of exogenously controlled hypothermia in rat pups. Differences were considered to be significant when $P < 0.05$. Data are presented as means ± SD for the number of individuals measured.

RESULTS

Body mass did not differ significantly (DF = 20, $t = 1.95$, $P = 0.065$) between dunnarts (23.2 ± 1.5 g) and rat pups (25.6 ± 4.0 g). Despite the similarity in body mass, rates of change in MR and T_b during entry into and arousal from torpor in dunnarts differed substantially from those during induction of hypothermia and passive rewarming in rat pups (Fig. 1). During torpor entry, which was observed in all dunnarts, the reduction of MR was rapid (90% reduction in 44 ± 16 min) and the minimum T_b and MR were reached after 142 ± 28 min. Induction of hypothermia in rat pups, however, was slower (DF = 20, $t = 11.6$, $P < 0.0001$), requiring 224 ± 17 min to reach minimum T_b and MR. Although minimum T_b during torpor (18.9 ± 1.1°C) was indistinguishable (DF = 20, $t = 0.38$, $P = 0.71$) from that during hypothermia (18.6 ± 2.8°C), minimum MR during torpor (0.23 ± 0.02 ml $O_2 \cdot g^{-1} \cdot h^{-1}$, ~5% of normothermic values) was only 12% (DF = 20, $t = 3.82$, $P = 0.001$) of that during hypothermia (1.99 ± 1.60 ml $O_2 \cdot g^{-1} \cdot h^{-1}$, ~35% of normothermic values). During spontaneous arousal from torpor (T_a maintained at ~18°C), dunnarts increased MR rapidly by 2500% over ~70 min to generate the heat required for active rewarming (Fig. 1*A*). In contrast, rat pups were unable to rewarm actively; during passive rewarming (induced largely by an increase in T_a), MR remained low and increased slowly with T_b (Fig. 1*B*).

When MR is plotted as a function of T_b, the differences between torpor and hypothermia become even more apparent. During the cooling phase of torpor entry, the MR of dunnarts initially fell substantially, even while T_b remained at ~35°C (Fig. 1*C*). As T_b fell from ~35 to ~20°C, there was a further, smaller reduction in MR. During the initial phase of rewarming from torpor, MR increased steeply with respect to T_b but then declined once active T_b had been reached. Throughout rewarming, MR exceeded that during entry into torpor for any given T_b.

In contrast, rat pups showed a reversed "hysteresis" of T_b and MR as they became hypothermic (Fig. 1*D*). Despite declining T_b, MR remained high during much of the cooling phase but ultimately decreased as T_b fell below ~20°C. At all body temperatures during rewarming, MR remained well below that during the cooling phase, increasing comparatively slowly as T_b increased.

DISCUSSION

Our study shows that the interrelations between MR and T_b during the cooling and rewarming phases of a torpor bout are opposite to those observed during the cooling and rewarming of hypothermia. While both relations show hysteresis (a difference in the relation between the two variables that depends on the direction of change), the hysteresis observed in hypothermia is the reverse of that in torpor. At the onset of cooling during entry into torpor, MR and T_b decrease precipitously at first, then slowly reach a plateau; warming is likewise characterized by a rapid rise in MR and T_b followed by a plateau at normothermic levels. Entry into cold-induced hypothermia, by contrast, proceeds slowly at first and becomes more rapid after the body begins to cool; warming likewise begins slowly and increases in rate only after T_b has started to rise. Therefore, low T_b alone is insufficient to establish unambiguously which has been induced: torpor or hypothermia. These findings demonstrate that torpor and hypothermia differ fundamentally, and they provide a new diagnostic tool for differentiating between these two states.

Underlying the different relations between T_b and MR in torpor and hypothermia are the relative values of T_b and the hypothalamic T_b set point. A low T_b or MR could result either from a reduction in the T_b set point (regulated hypometabolic state) or from an inability to produce heat sufficient to offset heat loss (failure to thermoregulate). At the onset of descent into a torpor bout, rapid cooling is initiated by a rapid decline in the T_b set point (15), which causes a reduction in MR (12, 16), and, as tissues cool, by the direct effects of lowered tissue temperature on MR (12). The ensuing precipitous reduction in both MR and T_b allows the animal to realize almost immediate energy savings. At the onset of arousal from torpor, the T_b set point is reset to the normothermic value, promoting a rapid increase in heat production (MR) that briefly exceeds even normothermic active levels and quickly warms the animal to its normothermic, behaviorally responsive state. Furthermore, it is important to note that all stages of torpor are subject to endogenous regulation. Even at the minimum T_b reached during steady-state torpor, MR can be regulated; T_b is maintained by proportional thermoregulation at the temporarily lowered hypothalamic set-point value even when T_a is substantially lower than the T_b set point (reviewed in Ref. 16). To achieve this control, the capacity for metabolic thermogenesis must be present throughout a bout of torpor.

During descent into hypothermia, by contrast, the T_b set point remains at normothermic levels, stimulating increased

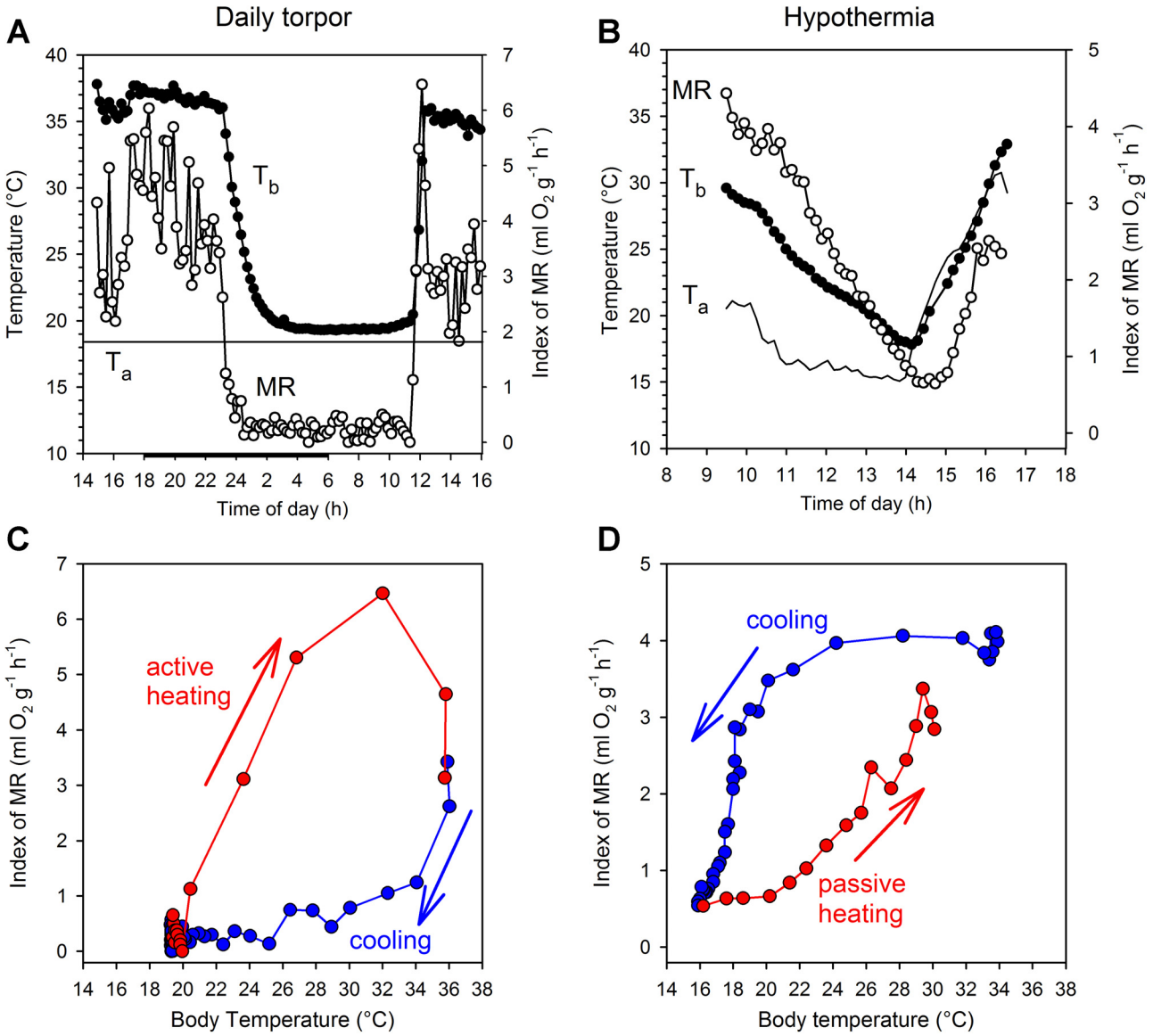

Fig. 1. Temporal changes in metabolic rate (MR) (proportional to O_2 consumption, quantified via respirometry) and body temperature (T_b) [quantified via radiotelemetry (dunnarts) or rectal thermocouples (rats)] during torpor in dunnarts (*A* and *C*) and hypothermia in rat pups (*B* and *D*). Data shown are from a representative dunnart and rat pup.

heat production to return T_b to the set-point value. When energy reserves are depleted or when heat loss exceeds heat production in the attempt to achieve the T_b set point, gradually declining tissue temperature directly reduces maximum heat production and MR, causing T_b to drop more precipitously and hypothermia to ensue. In contrast to torpid animals, hypothermic animals cannot defend a lowered T_b set point, can be cooled to any T_a, and ultimately die if they are not exogenously warmed (21). Because hypothermia is usually caused by an inability to generate sufficient heat, hypothermic individuals are unable to rewarm themselves actively; instead, they must rely on exogenous sources of heat to rewarm passively. A second diagnostic feature of torpor, then, is that animals are capable of rewarming actively at any time during a bout of torpor (37), whereas hypothermic animals are unable to do so without an external source of heat. Although some torpid animals may use passive warming to reduce the energy cost of active heating during arousal (8, 13, 40), which is the most energetically expensive phase of a torpor bout, they are physiologically capable of spontaneous, endogenously regulated arousal in the absence of an external heat source, thus distinguishing themselves physiologically from hypothermic animals.

Because of the remarkable abilities of torpid animals to avoid the potentially fatal consequences of hypothermia, there is great interest in determining whether a similar state could be induced in humans for medical purposes (4). The value of some forms of medically induced hypothermia is widely accepted, as it is already used successfully in cardiovascular surgery. Closely managing the duration and depth of tissue cooling, however, is critical to avoiding dangerous medical sequelae such as neurocognitive impairment and stroke, conditions that do not develop in torpid animals (4, 23). The search for methods of inducing a torpor-like state without these risks

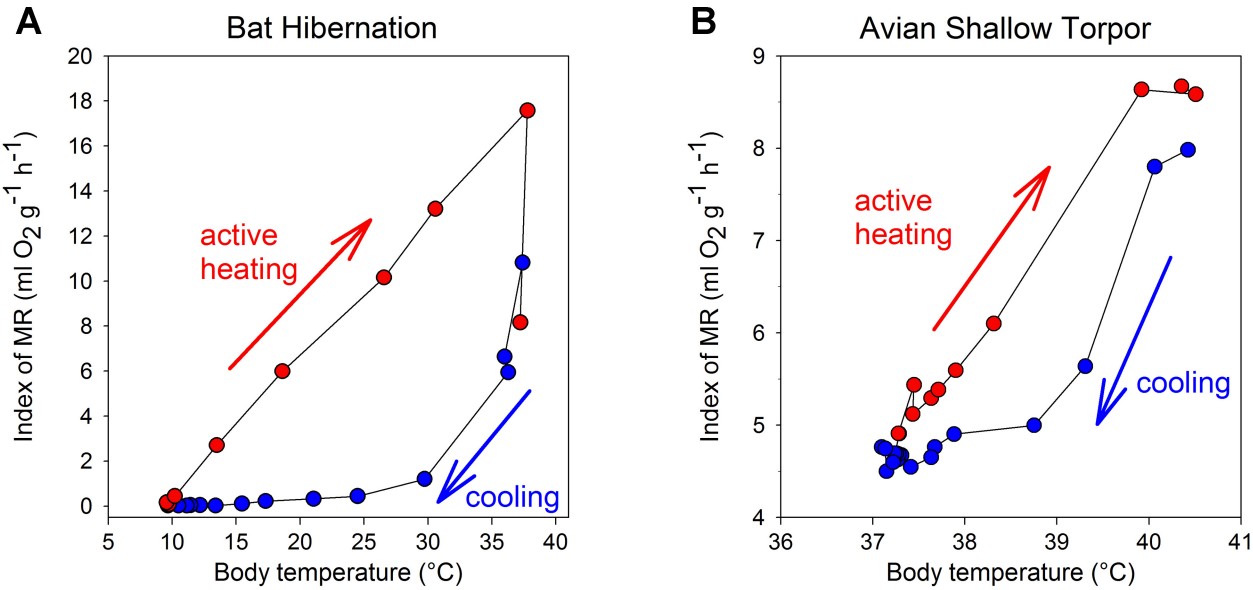

Fig. 2. MR as a function of T_b in a hibernating bat (*Nyctophilus gouldi*, 9 g, data from Ref. 6) (*A*) and a heterothermic bird (*B*) showing nocturnal shallow torpor often referred to as "nocturnal or facultative hypothermia" (*Zosterops lateralis*, 11 g, data from Ref. 22).

has uncovered hydrogen sulfide (H_2S), a metabolic inhibitor that achieves metabolic depression by inhibiting cytochrome *c* oxidase. While H_2S is claimed to initiate a state similar to hibernation and daily torpor in house mice (3), the time course of the effect and the relation between MR and T_b during this "suspended animation-like state" fit the general profile of unregulated hypothermia rather than of torpor. Furthermore, the naturally occurring torpor in house mice (18, 26) differs from the bouts of lowered T_b reported in Ref. 3: exposure to H_2S resulted in a stepwise reduction of MR by about 60% within 3 min, likely brought about not by a reduction of the T_b set point, but by inhibition of thermogenesis. This lowered heat production (coupled with a lowering of T_a from 24°C to 13°C) then resulted in a slow reduction of T_b by 20°C over 6 h, in sharp contrast to the rapid torpor entry times of 2–4 h in mice (18). T_b during continued H_2S exposure decreased further to 15°C or even 10°C (3, supporting online material), whereas mice in natural torpor regulated T_b at about 18°C when exposed to cold (18). Furthermore, most arousals from natural torpor by mice, even during cold exposure, were completed in less than 2 h as in *Sminthopsis* (Fig. 1*A*), whereas rewarming from the H_2S-induced state, exogenously assisted by restoring T_a to 24°C, took 3–4 h or approximately twice as long. While discovering additional means of inducing medically controlled hypothermia will certainly be useful, H_2S has not been shown to reliably induce a hypometabolic state in larger animals (4), and it is not surprising that the H_2S-induced state does not reliably confer the known benefits of torpor in other animals (9), particularly in those that do not normally use torpor. The tolerance of mice to H_2S-induced metabolic depression, on the other hand, may result from the natural ability of mice, unlike humans, to enter torpor.

Because Blackstone et al. (3) measured both T_b and MR in their study, it is easy to distinguish the H_2S-induced state from true physiological torpor. In other studies, measurement of only one of these variables has made interpretation more difficult. To elucidate the energy cues used to initiate torpor, a series of studies measured T_b of naturally heterothermic rodents when they were treated with various inhibitors of glucose and fatty acid metabolism (e.g., 32, 33) or by activating intracellular sensors of energy availability (42). Concurrent measurements of MR (e.g., 41) would have been useful in determining whether the ensuing bouts of reduced T_b showed the MR-T_b relationship of torpor (4), so that initiation of torpor could be distinguished from nonspecific metabolic inhibition that might mimic some aspects of torpor simply by interfering with the capacity to produce heat (35, 36).

To maintain the important distinction between torpor and hypothermia, we believe that these terms need to be used judiciously to refer to regulated (adaptive) and unregulated (nonadaptive) states of lowered T_b and MR, respectively. In the avian literature, for example, the terms "hypothermia," "adaptive hypothermia," and "facultative hypothermia" have been used to describe relatively small but energetically important reductions in nocturnal T_b in a variety of species (e.g., 2, 25, 28). One reason for using this terminology may be that some authors define torpor by the depth of T_b reduction rather than on the basis of whether or not it is regulated through alterations in the T_b set point (e.g., 7, 33). Accumulating evidence, however, points to a range of hypometabolic states with reductions in temperature ranging from only a few degrees Celsius below normothermic T_b to T_b values close to 0°C (1, 25, 30, 31, 39). Because the state referred to as "adaptive" or "facultative" hypothermia is a regulated hypometabolic state like torpor and indeed deep hibernation showing the same hysteresis between T_b and MR (Fig. 2), we suggest "shallow torpor" as a term that better captures the important underlying physiological differences between torpor and hypothermia. Our further hope is that using the terminology in this way, as originally suggested by Lyman et al. (21) and reiterated more recently (30), will unify the field by emphasizing both physiological homologies and physiological differences between regulated and unregulated hypometabolic states.

Perspectives and Significance

Our study demonstrates a fundamental and relatively easily measured physiological difference between regulated torpor and unregulated hypothermia, providing a robust diagnostic tool for distinguishing between these two superficially similar states. While both states are characterized by depressed T_b and MR, the relation between these two variables as an animal first cools and later rewarms shows hysteresis. Importantly, the direction of this hysteresis during a bout of torpor, as demonstrated here in a variety of vertebrate heterothermic endotherms, is opposite to that during an episode of cold-induced hypothermia. The difference in the direction of hysteresis results because torpor is initiated and terminated by a change in the T_b set point, whereas hypothermia results not from a change in T_b set point but from an inability to generate enough heat to maintain T_b at the set point. Our study suggests the importance of simultaneously measuring T_b and MR to distinguish between torpor and hypothermia, of using the appropriate terminology when referring to these two physiological states, and of translating conclusions from one of these states to the other with caution.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: F.G. and S.M.H. conception and design of research; F.G., S.E.C., K.A.O., and S.M.H. performed experiments; F.G., K.A.O., and S.M.H. analyzed data; F.G., S.E.C., and S.M.H. interpreted results of experiments; F.G. and S.M.H. prepared figures; F.G. and S.M.H. drafted manuscript; F.G., S.E.C., and S.M.H. edited and revised manuscript; F.G., S.E.C., and S.M.H. approved final version of manuscript.

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