# Prolactin and testosterone inhibit torpor in Siberian hamsters

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**Ruby, Norman F., Randy J. Nelson, Paul Licht, and Irving Zucker.** Prolactin and testosterone inhibit torpor in Siberian hamsters. *Am. J. Physiol.* 264 (*Regulatory Integrative Comp. Physiol.* 33): R123–R128, 1993.—Female Siberian hamsters maintained in a winter photoperiod (8 h light/day) ceased to undergo daily torpor during infusion of prolactin (PRL) from osmotic minipumps; winter torpor was reinstated within 3 days of discontinuation of treatment. By contrast, PRL infusion was ineffective in suppressing daily torpor elicited by restricting food intake in female hamsters housed in a summer photoperiod (16 h light/day). Summer daily torpor was, however, completely inhibited in long-day gonadectomized male hamsters treated with testosterone (T). We suggest that the hyperprolactinemia, which in previous studies is characteristic of hamsters that sustain ablation of the suprachiasmatic nucleus, is incompatible with winter torpor. Summer torpor may be controlled by a mechanism less responsive to variations in plasma PRL concentration. Both winter and summer torpor are inhibited by exogenous T; it remains uncertain, however, whether sustained decreases in endogenous T secretion are as essential for the expression of summer as they are for winter torpor.

photoperiod; body temperature; suprachiasmatic nucleus; food restriction

DAILY TORPOR in Siberian hamsters is characterized by a reduction in body temperature ($T_b$) from a euthermic value of 37°C to 18–25°C and is accompanied by diminished $O_2$ consumption (12). Torpor bouts begin at dawn, typically last 4–8 h, and occur almost daily at circadian intervals (24, 25, 28). Prolonged exposure to short days (i.e., winter daylengths) elicits daily torpor, and ablation of the suprachiasmatic nucleus (SCN) terminates its expression (23). This winter torpor can, however, be reinstated in SCN-lesioned hamsters by a brief period of restricted feeding (24). Torpor bouts reinstated in this manner differ in their temporal structure from normal daily torpor; they occur any time of day or night and 2 to 3 bouts may occur in a single day (24).

In long days (i.e., summer daylengths), daily torpor can be elicited in Siberian hamsters by restricting their food intake (24, 28). Summer daily torpor bouts are similar to ones elicited by short days. In both cases, the minimum $T_b$ reached during a bout is rarely maintained for more than 20 min; lower minimum $T_b$ values are associated with longer torpor bouts (24). However, bouts are generally less frequent and shallower in long days (24). In contrast to winter torpor, bouts of summer torpor are less tightly entrained to the light-dark cycle and not restricted to the daylight hours (24, 28). Both types of torpor conserve energy in small animals facing challenges posed by low ambient temperature ($T_a$) and food scarcity (14, 19).

The hormonal and photoperiodic conditions under which summer and winter torpor occur are substantially different. During the short days of winter, plasma gonadal (10, 29) and pituitary gonadotropin (5, 7) hormone concentrations are low; winter torpor is reversibly inhibited by administration of testosterone (T) but does not occur in gonadectomized hamsters housed in long days (28, 29). Exposure to short days over the course of several weeks and a decline in T secretion are both necessary for the appearance of winter torpor (21, 29). Onset of winter torpor also is correlated with a decline in plasma prolactin (PRL) concentrations (6, 30). In contrast, summer torpor is manifested during the long days of spring and summer when plasma concentrations of T and PRL reach their annual maximums. Although restricted feeding lowers endogenous concentrations of T and PRL, the influence of these hormones on summer torpor remains to be assessed.

In the present study we sought to determine whether PRL inhibits winter and summer torpor. The relation between PRL and torpor is relevant to an evaluation of the role of the SCN in winter torpor (4, 23) because ablation of the SCN eliminates this behavior and produces hyperprolactinemia (2, 23). We also assessed whether T concentrations that inhibit winter torpor also suppress summer torpor, and whether a decrease in T secretion must precede summer torpor.

## MATERIALS AND METHODS

### *Housing Conditions*

Hamsters (*Phodopus sungorus sungorus*) were obtained from our breeding colony, derived from stock donated by Dr. Bruce Goldman of the University of Connecticut. Hamsters were caged from birth in rooms illuminated by overhead fluorescent white lights (16:8-h light-dark cycle, lights on 0400 h, PST). Light intensity at cage level ranged from 50 to 300 lx. Room temperature ($T_a$) was maintained at 23 ± 2°C, and food (Purina Chow no. 5015) and tap water were available ad libitum unless otherwise specified. Hamsters were 2 mo old at the beginning of each experiment.

### *Body Temperature and Locomotor Activity*

Body temperature ($T_b$) and gross locomotor activity were measured telemetrically using intraperitoneal radiofrequency transmitters (model VM-FH, Mini-Mitter). Data were collected at 10-min intervals for the duration of the experiments by computer (software by Dataquest). Calibration and implantation of transmitters have been described elsewhere (23).

### *Torpor Criteria*

Torpor bouts were identified by visual inspection of printed $T_b$ data. The trough, defined as the single lowest $T_b$ reached during the bout, was readily determined in all bouts examined. To qualify as a torpor bout, $T_b$ had to remain below 30°C for at least 4 h. In euthermic hamsters $T_b$ occasionally approached but

never fell below 34°C (Ruby, unpublished observations). In every instance where $T_b$ fell below 34°C a torpor bout was initiated. The duration of torpor was measured as the interval during which $T_b$ remained below 34°C; onset and offset of torpor were considered to occur when $T_b$ first declined below 34°C and the last time point during the arousal process at which $T_b$ was <34°C, respectively.

*Food Restriction*

Average daily food intake was calculated during ad libitum feeding by measuring food intake at 24-h intervals for 7 consecutive days. During food restriction, each animal was given a preweighed amount of food daily at 1500 h, equivalent to 70% of its ad libitum food intake. These food rations were gradually reduced over 2–3 wk to adjust body mass to 65–70% of values during ad libitum feeding. If no torpor bouts occurred, food intake was reduced an additional 10% every week until at least one bout was observed or when body mass had been maintained at 65-70% of the baseline value for 2 wk. Because further reductions in body mass compromised the health of the animals, hamsters that failed to enter torpor after 2 wk at 65–70% of normal body mass were allowed to feed ad libitum and removed from the experiment. In *experiments 2* and *3*, body mass was clamped at the level that elicited torpor until experiments were terminated.

*Blood Samples*

Blood samples (200–300 μl) were collected between 1100 and 1300 h from the retroorbital sinus of euthermic hamsters anesthetized with methoxyflurane vapors (Metofane, Pitman-Moore) or pentobarbital sodium (Nembutal, 80 mg/kg). Blood was centrifuged for 30 min at 4°C and plasma aliquots were stored at −70°C until assayed.

*Hormone Administration*

*PRL.* Ovine PRL [oPRL; National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)-oPRL-19] was dissolved in 10 mM phosphate buffer solution containing 1.2% glycerol, 0.02% sodium azide, and 1.0 U/ml sodium heparin. Osmotic mini-pumps (Alza, model no. 2002) containing either oPRL or vehicle solution (VEH) were implanted under the dorsal skin while the animal was under pentobarbital sodium anesthesia. The concentration of oPRL in the pumps was adjusted to mimic endogenous PRL concentrations of hyperprolactinemic SCN-lesioned Siberian hamsters (2); the pumps were specified by the manufacturer to produce a continuous flow rate of 0.55 μl/h for 14 days.

*Testosterone.* Capsules were made by packing crystalline T (Sigma) into 4-mm lengths of Silastic tubing (Dow Corning; 1.95 mm ID and 3.13 mm OD) and sealing the ends with silicon sealant (Dow Corning). Dry capsules were soaked in saline at room temperature for 24 h and rinsed in 70% alcohol before implantation. Empty (blank) capsules were prepared in the same manner; capsules were implanted subcutaneously between the scapulae while animals were under pentobarbital sodium anesthesia.

*Hormone Assays*

*PRL.* oPRL was measured in duplicate 20- or 40-μl aliquots of plasma with double-antibody ovine radioimmunoassay (RIA) with a kit provided by NIDDK. The first antibody was diluted 1/500,000 in 0.25% concentration of normal rabbit serum-EDTA to 100 μl with phosphate-buffered saline (PBS). The second antibody, goat anti-rabbit gamma-globulin obtained from Antibodies (Davis, CA), was diluted to 1/150 concentration in cold 0.001 M PBS (100 μl total volume). Purified oPRL, iodinated by Hazelton Labs (Washington, DC), was diluted in 0.1% bovine serum albumin in PBS to a final radioactivity of ~12,000 counts·min$^{-1}$·tube$^{-1}$ of $^{125}$I-labeled oPRL. Final hormone concentrations were expressed as nanogram equivalents of oPRL per milliliter of hamster blood plasma. The standard curve was based on 10 values ranging from 25 to 1,500 pg/tube or 9 values ranging from 250 to 7,500 pg/tube. The lower and upper limits of the assay were 5 and 375 ng/ml per blood sample, respectively; concentrations >375 ng/ml represent extrapolated values. All plasma samples were measured in a single assay. The intra-assay coefficient of variation (CV) for eight samples from a pool of plasma was 9.6%. Samples of Siberian hamster serum showed no detectable cross-reaction with this assay.

*Testosterone.* Plasma T was measured as described by Licht et al. (18) in ether-extracted samples by RIA with an antiserum cospecific for testosterone and dihydrotestosterone supplied by Dr. Gordon Niswender. All samples were run in a single assay with a CV of 9%.

*Body Mass*

Animals were weighed (±0.1 g) weekly. Experimental and control groups were matched for body mass before food restriction and hormone treatments.

*Data Analysis*

Comparisons between groups were made using Student's $t$ tests, or $\chi^2$-tests where appropriate. Differences were deemed statistically significant if $P < 0.05$ (two-tailed tests) and are reported as $P < 0.05$ regardless of actual $P$ value.

*Experiment 1: Prolactin and Winter Torpor*

Ninety adult female Siberian hamsters were housed in a short photoperiod (8:16-h light-dark cycle, lights on 0800 h PST, $T_a$ = 23°C) for 12 wk. Forty-eight of these animals that had molted to a winter pelage (white fur) and weighed <30 g were ovariectomized and implanted with radiofrequency transmitters while under pentobarbital sodium anesthesia. Twenty-four hours later they were transferred to a cold chamber and maintained at 15°C in the same short-day photoperiod. Fourteen days later blood samples were taken from euthermic hamsters that had expressed at least four bouts of torpor. Pumps containing either oPRL or VEH were implanted 1 wk later. Blood was sampled again 10 days after pump implantation and pumps were removed 4 days later. $T_b$ and activity were monitored for 14 more days after pump removal.

*Experiment 2: Prolactin and Summer Torpor*

Twenty-four adult ovariectomized hamsters, previously housed in the 16:8-h light-dark photoperiod, were moved to a cold chamber on *day 0* (16:8-h light-dark cycle, lights on at 0400 h PST; $T_a$ = 15°C). On *day 14* blood samples were taken, and restricted feeding began on *day 28*. Within 3 days of the first torpor bout, a second blood sample was obtained and a pump containing either oPRL or VEH was implanted. Ten days after implantation a third blood sample was taken and the experiment was terminated.

*Experiment 3: Testosterone and Summer Torpor*

Thirty-two adult male hamsters, previously housed in the 16:8-h light-dark photoperiod, were moved to a cold chamber on *day 0* (16:8-h light-dark cycle, lights on at 0400 h PST; $T_a$ = 15°C). On *day 14* blood samples were taken and restricted feeding began on *day 28*. Within 3 days of the first torpor bout a second blood sample was obtained, hamsters were gonadectomized, and capsules filled with T or left empty (blank) were implanted. All three procedures were performed in one operation. After capsules had been in place for 10 days, a third blood sample was taken and capsules were removed. A third group of

hamsters (designated NI) never expressed torpor and were neither gonadectomized nor implanted with capsules. Blood samples were obtained from NI hamsters before food restriction and again when their percent body mass loss was equivalent to that of animals implanted with capsules.

## RESULTS

### *Experiment 1: Prolactin and Winter Torpor*

*Torpor.* PRL administration markedly decreased the incidence of torpor. Torpor was observed in 19 and 91% of hamsters during administration of PRL ($n = 16$) or VEH ($n = 11$), respectively ($\chi^2 = 23.2$, $P < 0.05$). During the first 10 days after treatment was discontinued, 91 and 88% of hamsters treated previously with PRL or VEH, respectively, displayed torpor one or more times. Although torpor was completely suppressed in 13 of the 16 hamsters during PRL infusion, it resumed within 2.8 ± 0.4 (±SE) days of pump withdrawal (e.g., Fig. 1*A*).

The mean number of torpor bouts observed during treatment did not differ between hamsters infused with VEH and the three that expressed torpor during infusion with PRL (4.5 ± 0.8 vs. 6.0 ± 2.0 bouts, respectively; $P > 0.05$). In two of the latter three hamsters, mean minimum $T_b$ during torpor was similar during and after termination of PRL treatment (23.9 ± 1.1 vs. 23.8 ± 1.1°C; $P > 0.05$), whereas the third animal had shallower torpor bouts during PRL treatment (Fig. 1*B*; minimum $T_b = 27.1 \pm 1.2$°C during and 18.3 ± 0.2°C before PRL infusion; $P < 0.05$).

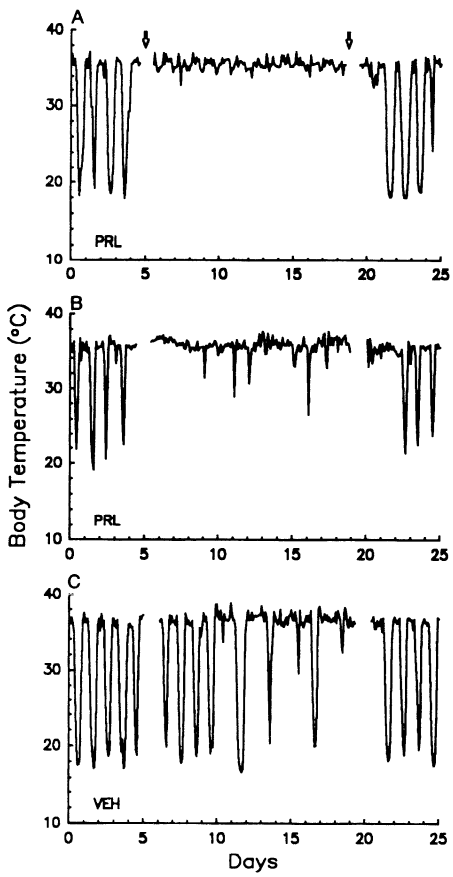


Fig. 1. Body temperature ($T_b$) of hamsters in which ovine prolactin (oPRL) completely inhibited (*A*) or was compatible with limited torpor (*B*). $T_b$ of a hamster administered vehicle solution is shown in *C*. Arrows in *A* indicate when pumps were implanted (*day 5*) and removed (*day 19*); blood samples were taken on *day 15*. Data were averaged over 60-min intervals.

*Prolactin concentrations.* Hamsters that continued to express torpor during PRL treatment ($n = 3$) had PRL concentrations similar to those of hamsters in which PRL inhibited torpor ($n = 13$; $P > 0.05$; Table 1); plasma concentrations of PRL ranged from 255 to 338 ng/ml and from 75 to 549 ng/ml in these two groups, respectively. Plasma PRL was not assayed in animals during VEH infusion because endogenous PRL does not cross-react in the radioimmunoassay.

*Body mass.* Body mass did not differ between groups before or during treatment with PRL or VEH ($P > 0.05$).

### *Experiment 2: Prolactin and Summer Torpor*

*Torpor.* Before treatment the latency to the initial torpor bout and number of bouts displayed did not differ between PRL- and VEH-treated hamsters ($P > 0.05$; combined values for both groups were 14.6 ± 2.8 days and 2.0 ± 0.3 bouts; $n = 12$ animals). At least one torpor bout was observed in 75 or 100% of hamsters during 10 days of infusion of PRL or VEH, respectively (Fig. 2*A*). During infusion neither the mean number of torpor bouts (3.0 ± 1.0 vs. 1.8 ± 0.5) nor the minimum $T_b$ attained during torpor (25.3 ± 1.5 vs. 22.3 ± 1.1°C) differed between hamsters treated with PRL ($n = 6$) or VEH ($n = 4$), respectively ($P > 0.05$; e.g., Fig. 3). The latency to the first torpor bout after onset of infusions was not different for hamsters treated with PRL or VEH (6.2 ± 1.1 vs. 6.3 ± 0.9 days, respectively; $P > 0.05$).

*Prolactin concentrations.* During PRL administration, mean plasma concentrations of PRL were high and did not differ between animals that did or did not express torpor ($P > 0.05$; Table 2).

*Body mass.* Body masses were similar at the start of PRL and VEH infusions (24.3 ± 1.1 vs. 24.2 ± 0.8 g, respectively; $P > 0.05$), and the body mass loss that preceded the first torpor bout did not differ between these groups ($P > 0.05$; Fig. 2*B*). Body mass remained constant throughout the 10-day infusion interval for both groups (not illustrated, $P > 0.05$).

### *Experiment 3: Testosterone and Summer Torpor*

*Torpor.* The latency from the onset of food restriction to the initial torpor bout was 11.3 ± 1.7 days; animals displayed 1.8 ± 0.3 bouts before hormone treatments began ($n = 13$). None of the seven hamsters administered T displayed even a single torpor bout (e.g., Fig. 4). In contrast, all six hamsters implanted with blank capsules had at least one torpor bout during the 10-day treatment phase ($\chi^2 = 14.0$, $P < 0.05$; e.g., Fig. 4). The mean minimum $T_b$ attained during torpor was 25.1 ± 0.9°C, and

Table 1. *Short days: plasma prolactin concentrations*

| Treatment | *n* | Torpor | oPRL, ng/ml |
|---|---|---|---|
| oPRL | 13 | No | 305.9±38.9 |
| oPRL | 3 | Yes | 304.0±25.6 |
| VEH | 11 | Yes | Not measured |

Values are means ± SE; *n*, no. of animals. oPRL, ovine prolactin; VEH, vehicle. "Yes" in Torpor column indicates that animals expressed at least 1 torpor bout during the 14-day interval of treatment.

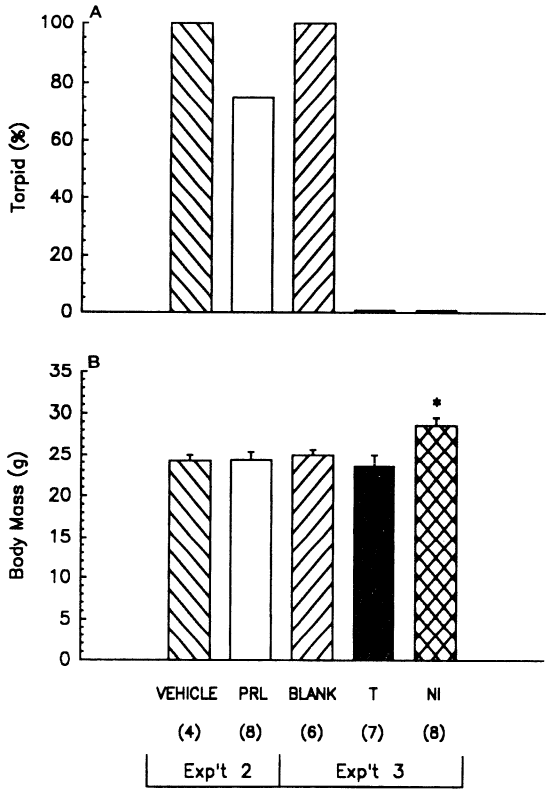


Fig. 2. Percent of food-restricted hamsters that expressed at least one torpor bout during 10 days of hormone treatment (*A*). Body mass (means ± SE) is given for the day of capsule implantation for all groups except hamsters that were never implanted or gonadectomized (NI); body mass for NI hamsters is given for the first week when their % body mass loss was equivalent to that of testosterone-treated (T) and blank groups (*B*). Numbers in parentheses, sample size. Dark horizontal bars in *A*, 0% torpor for T and NI groups. * Body mass greater than that of other groups ($P < 0.05$).

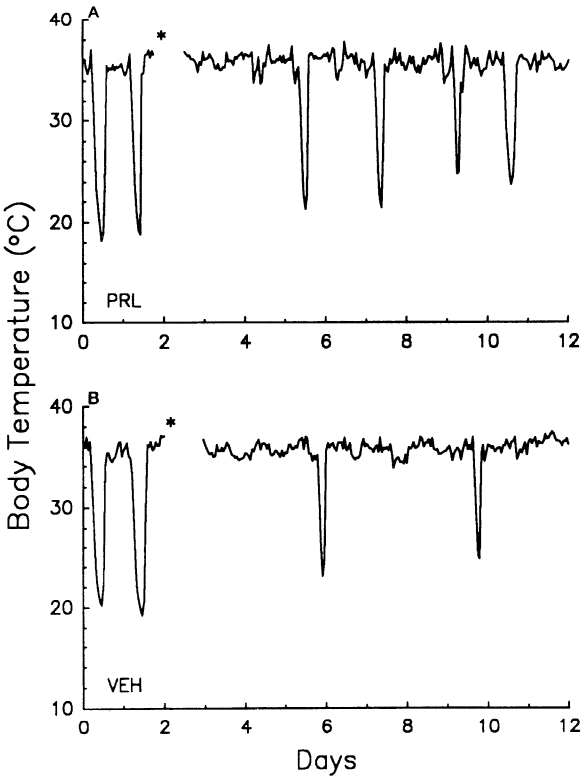


Fig. 3. Representative $T_b$ values of food-restricted hamsters administered oPRL (*A*) or vehicle solution (*B*). * Day when pump was implanted; blood samples were taken on *days 2* and *12* and pumps removed on *day 12*. Data were averaged over 60-min intervals.

Table 2. *Long days: plasma prolactin concentrations*

| Treatment | *n* | Torpor | oPRL, ng/ml |
|---|---|---|---|
| oPRL | 2 | No | 353.3±110.7 |
| oPRL | 6 | Yes | 303.0±32.0 |
| VEH | 4 | Yes | <5.0 |

Values are means ± SE; *n*, no. of animals. "Yes" in Torpor column indicates that animals expressed at least 1 torpor bout during the 10-day interval of treatment.

the latency to the initial bout after pump implantation was 3.5 ± 0.3 days for animals treated with blank capsules.

*T concentrations.* Endogenous T concentrations (baseline values) during ad libitum feeding were 1.1 ± 0.2 ng/ml for T, blank, and NI groups combined ($n = 21$). At the time of the first torpor bout (i.e., before differential hormone treatment began) T concentrations were reduced below baseline values, but the decrease was not statistically significant (0.6 ± 0.2 vs. 1.0 ± 0.3 ng/ml for animals subsequently implanted with capsules; $n = 13$; $P > 0.05$). Four hamsters had T concentrations of ≥1.0 ng/ml at the time of their first torpor bout. NI intact males ($n = 7$) had significantly lower T concentrations compared with baseline values (0.2 ± 0.0 vs. 0.9 ± 0.4 ng/ml; $P < 0.05$) after 37 days of restricted feeding. T concentrations generated by T capsules (3.7 ± 1.1 ng/ml) were higher than baseline T concentrations of intact animals ($P < 0.05$) and in every case suppressed torpor (e.g., Fig. 4).

*Body mass.* Body mass reductions were similar among T-treated, blank-control, and NI hamsters (63.4, 65.6, and 66.2% of baseline body mass, respectively). Hamsters that failed to express torpor during restricted feeding (NI group) were heavier both before and during food restriction than animals that became torpid ($P < 0.05$; Fig. 2*B*). Body mass at the time of capsule implantation did not differ between hamsters treated with T or blank capsules ($P > 0.05$; Fig. 2*B*) and remained constant during the 10-day treatment interval for both groups (not illustrated, $P > 0.05$).

## DISCUSSION

Exogenous PRL reversibly inhibited daily torpor in female Siberian hamsters maintained in short days (winter torpor). Most hamsters that failed to express torpor had plasma PRL concentrations >300 ng/ml. Ablation of the SCN in Siberian hamsters produces similarly elevated endogenous PRL concentrations (2) and eliminates the expression of torpor (23). Therefore, elimination of winter torpor after ablation of the SCN may be due to hyperprolactinemia rather than to interruption of neural pathways essential for torpor. The present results also raise the possibility that PRL concentrations, which are greatly elevated in Siberian hamsters during lactation (22), may be incompatible with torpor during the latter part of the breeding season when daylengths are decreasing or already short. We know of no reports of lactating

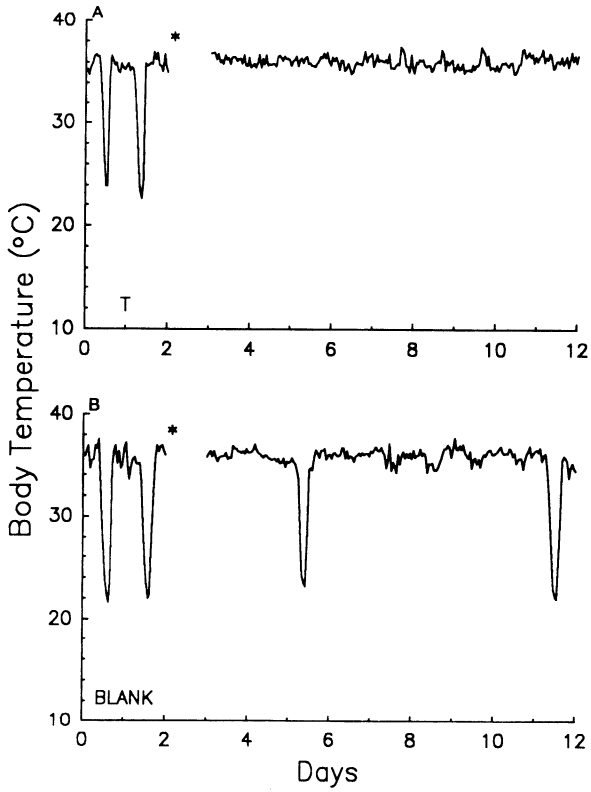


Fig. 4. Representative $T_b$ values of hamsters implanted with testosterone (T) capsules (*A*) or blank capsules (*B*). * Day when capsule was implanted; blood samples were taken on *days 2* and *12* and capsules were removed on *day 12*. Data were averaged over 60-min intervals.

mammals other than bats undergoing torpor (16). Three hyperprolactinemic hamsters continued to express torpor; in only one of these animals was the depth of torpor reduced during PRL treatment. Factors that permitted torpor in these animals cannot be specified presently.

The rapidity (1–2 days or less) with which winter torpor was affected by variations in plasma PRL concentrations is notable. Several seasonal, behavioral, morphological, and physiological adaptations are first evident only several weeks after the transition from long to short days produces changes in hormone secretion (1, 9, 11, 17). In contrast, winter torpor was affected almost immediately by variations in plasma PRL concentrations, perhaps indicating a different mechanism of action.

PRL was not effective in terminating summer torpor. This form of nutritionally induced torpor is usually elicited by food shortages during the spring and summer breeding seasons (14, 19) when annual plasma PRL concentrations are high (30). Siberian hamsters may have evolved mechanisms that permit summer but not winter torpor in the presence of high PRL concentrations. Alternatively, food restriction may decrease target tissue sensitivity to PRL, thereby facilitating torpor during the summer breeding season. Restricted feeding regimens decrease PRL secretion in rats (3, 27) but the relevant measurements have not been made in Siberian hamsters.

The decrease in plasma T associated with testicular regression must occur before winter torpor can be expressed in Siberian hamsters (8, 15); torpor is inhibited in short-day gonadectomized hamsters treated with T (29). The present study extends this relation to summer torpor; all long-day hamsters failed to enter torpor during T treatment. Generally, T concentrations generated by the capsules were three times higher than endogenous concentrations. It is curious, however, that four intact hamsters had high endogenous T concentrations (>1.0 ng/ml) at the time of their first torpor bout. T secretion is episodic and pulsatile, rather than continuous, in Siberian hamsters (13) as in other rodents. Thus it is unclear whether the high T values of the four hamsters are representative of modal T concentrations during the interval of restricted feeding, or reflect transitory high values. In either case it remains to be established that a sustained decrease in T secretion is a prerequisite for summer torpor.

One group of long-day hamsters (NI group) never displayed summer torpor despite sustaining a percent body mass loss similar to hamsters that did express this behavior. Our restricted feeding paradigm for eliciting torpor is based on percent body mass loss; absolute body mass may, however, be more important in determining whether summer torpor will occur. The mean body mass was <25 g for hamsters that expressed summer torpor and >25 g for those that did not display summer torpor (Fig. 2); the latter group failed to become torpid despite quite low T concentrations (0.2 ng/ml) and an interval of food restriction that was over twice as long as that of animals that did express summer torpor. Low T concentrations and body masses of <25 g may act synergistically to promote torpor because neither condition alone was sufficient to elicit torpor in gonadally intact long-day hamsters.

The rapidity with which T affects torpor is remarkable compared with other behavioral responses (e.g., mating behavior; Ref. 20). This may be advantageous for a male hamster facing food scarcity; males in reproductive condition may temporarily decrease androgen production and become torpid in response to food scarcity without compromising subsequent reproductive opportunities. Severe food shortages far in excess of those that elicit torpor do not eliminate reproduction in male rats (26).

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