Role of area postrema in control of torpor in Siberian hamsters

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DAILY TORPOR HELPS small mammals cope with limited food availability and low ambient temperatures. Siberian hamsters undergo torpor during the short days of winter and in response to glucoprivation or food restriction. We tested whether the area postrema and the adjacent nucleus of the solitary tract (hereafter the AP), which monitor metabolic fuel availability, also control the onset of torpor. Siberian hamsters that had manifested torpor spontaneously or had entered torpor in response to 2-deoxy-D-glucose (2-DG) treatment were subjected to area postrema ablations (APx). Hamsters continued to display torpor postoperatively; most features of torpor were unaffected by APx. The AP is not necessary for expression of torpor elicited by short day lengths or metabolic challenge. In contrast, decreases in food intake manifested by hamsters treated with 2-DG were counteracted by APx. In Siberian hamsters, the AP appears to mediate effects of 2-DG on food intake but not torpor.

food intake; 2-deoxy-D-glucose
in Syrian hamsters treated with a high dose of 2-DG (1,750 mg/kg; Ref. 36), and diminishes the disruption of lordosis in food-restricted Syrian hamsters ovarioctomized and treated with estradiol benzoate and progesterone (19) and those injected with 2-DG (36). Collectively, these findings implicate the AP as a major component of the neural system that monitors glucose availability and thereby controls energy-dependent behaviors such as food consumption and estrus. The disruptive effect of 2-DG on estrous behavior could also reflect separate actions of the drug on secretion of gonadal hormones.

The neural pathways that mediate torpor in Siberian hamsters remain to be fully identified. The suprachiasmatic nucleus is necessary for the temporal organization of torpor bouts but not for their expression (29), the pineal is necessary for onset of spontaneous torpor in short day lengths but not that induced by food restriction (41, 42), and the PVN is necessary for spontaneous torpor in a majority of hamsters; its role in torpor elicited by food restriction is unknown (28).

Because torpor is an acutely energy-sensitive process, glucosensitive neurons of the AP (2) are well situated to participate in the control of this behavior. The integrity of the AP may determine whether hamsters can perceive or respond to the changes in metabolic fuels that trigger torpor onset. Individual hamsters adopt one of several tactics when energy demands are high; some utilize torpor bouts liberally, eat less, and reduce nocturnal locomotor activity, whereas others forego hypothermia and instead consume more food and increase nocturnal activity (31). To evaluate the role of the AP in regulation of torpor and food intake, we ablated this structure (APx) in Siberian hamsters and increase nocturnal activity (31). To evaluate the role of the AP in regulation of torpor and food intake, we ablated this structure (APx) in Siberian hamsters that were manifesting spontaneous bouts of torpor; other APx hamsters were challenged with 2-DG treatments that elicit torpor in neurologically intact animals. We anticipated that in the absence of the AP, Siberian hamsters would be less able to assess metabolic fuel availability and consequently less prone to display torpor when challenged with reduced fuel availability.

**Definition of Torpor**

Torpor was considered to occur when $T_b$ was $<31^\circ C$ for a minimum of 30 min. Depth of torpor refers to the absolute minimum temperature ($T_b \text{ min}$) reached during a bout of torpor and duration of torpor to the number of minutes $T_b$ was $<31^\circ C$. Latency to display torpor references the interval from the time of injection of 2-DG to the initial decrease in $T_b$ to $<31^\circ C$.

**Surgery**

APx designates ablation of the AP plus adjacent NTS unless otherwise noted. APx was performed under anesthesia as described above. Hamsters were placed in a stereotaxic instrument, and an incision was made from the occipital crest to the midcervical level. The top muscle layer was removed to expose the occipital bone. The foramen magnum was enlarged, the dura was removed, and the cerebellum was lifted to visualize the AP, which was removed by suction with an aspiration needle. Bleeding was controlled by inserting absorbable foam (Gel Foam, Upjohn, Kalamazoo, MI) into the foramen magnum when necessary. The muscles were then sutured and treated with 0.1% nitrofurazone ointment. Sham-operated animals underwent the same treatment except the AP was not removed. Postoperatively, acetaminophen and codeine were added to the drinking water for 3 days.

**Histological Assessment**

Hamsters were deeply anesthetized with pentobarbital sodium (80 mg/kg body wt) and perfused transcardially with a mixture of 15 ml of 0.9% NaCl and 0.75 ml Evans blue (100 mg/ml; Sigma Chemical, St. Louis, MO; dissolved in NaCl at pH 7.4), followed by buffered Formalin. Brains were postfixed for at least 2 h in Formalin and embedded in paraffin; frozen sections were cut at 15 μm throughout the AP region, stained with cresyl violet, and mounted onto slides for histological verification of extent of brain lesions.

**Statistics**

The Statview statistics package (Abacus Concepts, Berkeley, CA) was used for all statistical analyses. Mean differences between groups were analyzed by ANOVA with repeated measures where appropriate. Where significant $F$ ratios were obtained for one-way ANOVA, group means were compared with Fisher’s protected least significance difference test (Fisher’s PLSD). Comparisons involving frequencies were made using $x^2$ or Fisher’s exact test. Correlations between variables were evaluated with the squared Pearson correlation coefficient. Results were considered significant if $P < 0.05$ with two-tailed tests and are reported as such regardless of actual $P$ value. Number of animals represents individuals per group with working transmitters on the day of treatment, not total number of animals treated. Values are reported as means ± SE.

**Experiment 1: Role of AP in Torpor Induced by 2-DG**

**Animals.** Adult male hamsters (*Phodopus sungorus*), maintained from birth in a 16:8-h light-dark photoperiod (lights on 0300, Pacific standard time) at 23 ± 2°C, were fed Purina Rodent Chow 5015 and water ad libitum. Eight weeks before APx or sham surgery, animals were transferred to a cold room (15°C) with an identical photoperiod and individually housed in polypropylene cages (25 x 14 x 12 cm) with wood shavings for bedding and cotton nesting material. Two
weeks later, radiotransmitters were implanted intra-abdominally into hamsters whose body weights were recorded weekly thereafter. Animals that underwent either APx (n = 24) or sham surgery (n = 18) were allowed to recover at an ambient temperature of 23°C for 1–2 days postoperatively and were then returned to 15°C. 2-DG (Sigma; doses of 2,250 and 2,500 mg/kg, 625 mg/ml ddH2O ip) and saline were administered to each hamster in a counterbalanced design with 1 wk between injections, beginning 3–5 wk after surgery. All injections were given between 0800 and 0900. No pretest was administered, because 2-DG doses in the range that were used in the present study were previously demonstrated to reliably induce torpor in 50–80% of hamsters tested (6, 39), and propensity for torpor in one test was not highly predictive of another bout within individual hamsters.

**FI measurements.** After hamsters were acclimated for 7 days to the disturbance involved with measuring FI, 24-h food consumption was recorded daily, beginning 2 days before and ending 2 days after drug treatment. Food was stored in the cold room 1 wk before being dispensed to hamsters to compensate for changes in weight due to moisture absorption in the cold.

**Experiment 2: Role of the AP in Spontaneous Torpor**

*Animals.* A second cohort of adult male Siberian hamsters was transferred from the 16:8-h light-dark photocycle to a short photoperiod (8:16-h light-dark cycle, lights on at 0830) and low temperature (15°C) at 3–4 mo of age; 12 wk later they were monitored for a suite of winter responses including gonadal regression (assessed by palpation), decreased body weights (>10%), and onset of molt to a winter coat (pelage score >2) (10). Transmitters were implanted in animals that demonstrated these winter responses; those that manifested at least two torpor bouts over the next 4 wk were designated for APx or sham operations. Sixteen APx and six sham hamsters were monitored for 14–17 wk postoperatively.

**FI measurements.** Twenty-four-hour food consumption was recorded every day between 1400 and 1600 for 6 wk postoperatively.

**Activity monitoring.** Gross locomotor activity was measured using the same biotelemetry system as for Tb recordings. Activity data were recorded as the total number of locomotor movements that occurred in a 10-min interval. A minimum distance of 30 cm between adjacent boards was maintained to minimize interference between transmitters. Activity data were collected for 2 wk beginning 2 mo after neural surgery.

All procedures were approved by the University of California at Berkeley Animal Care and Use Committee.

**RESULTS**

**Lesion Verification**

The AP was completely ablated in 18 and 14 hamsters in experiments 1 and 2, respectively. Damage to the NTS varied between 0–25% and 75–100%. The hypoglossal nucleus also sustained minimal damage (<25%). Damage to the NTS and hypoglossal nucleus was determined at or near the level of the AP. As such, some residual NTS is likely to be present further rostrally and caudally. Representative photomicrographs are shown in Fig. 1. Extent of damage to the NTS or hypoglossal nucleus did not affect the propensity for animals to undergo torpor. For purposes of data analysis, animals were consequently assigned to either APx or sham groups. Visible damage to the AP was undetectable in all Sham hamsters.

**Experiment 1**

**APx and 2-DG-induced torpor.** Hamsters with complete ablation of the AP continued to manifest torpor when treated with 2,500 mg/kg 2-DG (Table 1). At the lower dose (2,250 mg/kg), only the sham animals responded with a torpor bout, but the sham and APx groups did not differ significantly (χ² = 3.29; P > 0.05).
Table 1. Torpor characteristics of hamsters injected with 2-DG

<table>
<thead>
<tr>
<th>Group</th>
<th>Dose of 2-DG, mg/kg</th>
<th>Sex</th>
<th>Proportion Torpid</th>
<th>( T_{b\min}, \degree C )</th>
<th>Duration of Torpor, min</th>
<th>Latency to Display Torpor, min</th>
</tr>
</thead>
<tbody>
<tr>
<td>APx</td>
<td>2,500</td>
<td>M</td>
<td>5/18</td>
<td>29.1 ± 0.3</td>
<td>100 ± 13</td>
<td>40 ± 5</td>
</tr>
<tr>
<td>Sham</td>
<td>2,500</td>
<td>M</td>
<td>7/18</td>
<td>27.9 ± 0.8</td>
<td>153 ± 21</td>
<td>36 ± 3</td>
</tr>
<tr>
<td>APx</td>
<td>2,250</td>
<td>M</td>
<td>0/17</td>
<td>27.6 ± 1.1</td>
<td>140 ± 45</td>
<td>40 ± 0</td>
</tr>
<tr>
<td>Sham</td>
<td>2,250</td>
<td>M</td>
<td>3/17</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SE. APx, area postrema ablation; 2-DG, 2-deoxy-D-glucose; \( T_{b\min} \), minimum body temperature.

As expected, no hamster entered torpor when injected with the saline vehicle. Neither the percentage of torpid animals (\( \chi^2 = 0.95; P > 0.05 \)), the duration of torpor \( [F(1,11) = 2.3; P > 0.05] \), nor the latency to undergo torpor \( [F(1,11) = 0.34; P > 0.05] \) differed between APx and sham animals treated with the higher dose of 2-DG (Table 1).

\( T_{b\min} \) did not differ between APx and sham hamsters on the day before 2-DG injections (Fisher’s PLSD; \( P > 0.05 \)). On the day of treatment with 2,500 mg/kg 2-DG, \( T_{b\min} \) was lower in both the APx and sham groups compared with their own values the previous day (\( t \)-test; \( P < 0.05 \)) and to \( T_{b\min} \) of hamsters treated with saline (Fisher’s PLSD; \( P < 0.05 \)). Mean \( T_{b\min} \) among hamsters that entered torpor did not differ among treatment groups (Fisher’s PLSD; \( P > 0.05 \)).

**Effects of surgery on body weight.** There was a significant effect of time and an interaction of time with lesion but not of lesion alone [time: \( F(1,12) = 16.2, P < 0.05 \); interaction: \( F(1,12) = 4.9, P < 0.05 \); lesion \( F(1,1) = 4.0, P > 0.05 \)]. Body weights did not differ preoperatively between APx and sham hamsters (\( t \)-test; \( P > 0.05 \)). Postoperatively, the body weights of APx hamsters were depressed compared with preoperative values until the time of the first treatment (2,250 and 2,500 mg/kg 2-DG or vehicle injection; \( t \)-test; \( P < 0.05 \)) but did not differ significantly from those of sham animals at the time of the second and third treatments (\( t \)-test; \( P > 0.05 \); Fig 2).

**APx and FI.** Both doses of 2-DG decreased FI of sham hamsters (Fig 3); on the day of and the day after 2-DG injection (\( \text{days 0 and 1} \)), FI was depressed to <50% of pretreatment values (\( \text{day -1} \)) (\( t \)-test; \( P < 0.05 \)). Recovery to baseline FI values was complete on \( \text{day 2} \). This relation held regardless of whether or not 2-DG induced torpor. In contrast, 2-DG treatment was completely ineffective in suppressing FI in APx hamsters on either the day of treatment or the subsequent day (\( t \)-test; \( P > 0.05 \)). APx and sham hamsters injected with saline did not change their FI (\( t \)-test; \( P > 0.05 \); Fig 3).

**Experiment 2**

**APx and torpor induced by short days.** All hamsters manifested torpor preoperatively; APx and sham groups had comparable numbers of such bouts over 4 wk [3 ± 0.6 and 4 ± 0.8; \( F(1,16) = 1.1, P > 0.05 \)]. Postoperatively, all animals continued to undergo torpor; the mean number of such bouts displayed over the course of 14–17 wk was comparable for both groups [\( F(1,16) = 1.1, P > 0.05 \); Table 2].

The duration of torpor did not differ pre- or postoperatively among APx and sham groups (Fisher’s PLSD; \( P > 0.05 \)). Latency to undergo spontaneous bouts of torpor did not differ preoperatively between the two groups (Fisher’s PLSD; \( P > 0.05 \)) (Table 2). The first torpor bouts postoperatively occurred 6 days earlier in sham than APx hamsters, but the difference was not significant (Fisher’s PLSD; \( P > 0.05 \)). The depth of torpor (\( T_{b\min} \)) was slightly lower in APx than in sham hamsters (Fisher’s PLSD; \( P < 0.05 \); Table 2).

**Circadian timing of torpor.** Circadian distribution of torpor bouts did not differ between sham and APx hamsters. Torpor was initiated a few hours before or during the light phase, except for one APx animal that initiated four bouts at the onset of the dark phase (1 pre- and 3 postoperatively). Except for one APx hamster, no animal displayed more than one torpor bout per day.

There was a positive correlation (\( r^2 = 0.55; P < 0.05 \)) between the length of the torpor season (beginning of torpor onset preoperatively to last torpor bout postoperatively) and total number of bouts, but there was no relation between onset of torpor season and total number of torpor bouts (\( r^2 = 0.02; P > 0.05 \)) for both groups of animals.

Mean activity counts for individual hamsters during the first 4 h after a torpor bout were reduced compared with the number of counts for the same interval on

![Fig. 2. Mean (±SE) body weight of male sham and area postrema ablation (APx) hamsters injected with 2-deoxy-D-glucose (2-DG) or saline. Surgery occurred during weeks 1–2. Time of 1st, 2nd, and 3rd injections is indicated above respective week of treatment in long day lengths. * \( P < 0.05 \) compared with APx.](http://ajpregu.physiology.org/ by 10.22033.3 on November 7, 2016)
normothermic days (16.4 ± 2.4 vs. 32.0 ± 4.4; t-test; $P < 0.05$). Food consumption decreased as the number
of torpor bouts increased ($r^2 = 0.41; P < 0.05$).

**FI.** APx hamsters consumed slightly less food (4.0 g)
on the day of spontaneous torpor (day 0) than during
the preceding 24 h (4.6 g; t-test; $P < 0.05$) and recovered
to baseline values the day after torpor. FI of sham
hamsters did not differ significantly on the day of
torpor from values the day before or the day after torpor (t-test; $P > 0.05$).

**Effects of surgery on body weight.** There was a sig-
nificant effect of time and an interaction of time with
lesion but not of lesion alone (time: $F(1,15) = 6.1, P <
0.05$; interaction: $F(1,15) = 2.7, P < 0.05$; lesion:
$F(1,1) = 0.034, P > 0.05$). Body weight did not differ
significantly between groups at any point during the
experiment.

**DISCUSSION**

Even though the AP is involved in monitoring energy
availability and energy availability influences torpor,
ablations of the AP did not prevent the occurrence of
torpor in response to treatment with high doses of
2-DG or prolonged exposure to winter day lengths in
Siberian hamsters. Torpor survived APx with little
change from preoperative baseline values in frequency,
duration, and depth of bouts. APx appeared marginally
effective in decreasing the incidence of torpor in ham-
sters treated with a lower dose of 2-DG; perhaps the AP
mediates metabolic challenges that more closely simu-
late those within the physiological range. The monitor-
ing of energy availability by AP neurons is not neces-
sary for expression of torpor in short day lengths;
structures distinct from those in the AP are sufficient to induce
the hypometabolic state. These findings do not, how-
ever, preclude AP participation in monitoring fuel
shortages that induce torpor in neurologically intact
hamsters.

The mechanism by which 2-DG triggers torpor ex-
pression in Siberian hamsters has been questioned
recently. Whereas plasma glucose concentrations de-
cline during entry and increase during arousal from
torpor, injections of insulin that produce similar levels
of hypoglycemia as occur during spontaneous torpor
did not trigger a decline in $T_b$ (6). It remains possible
that the extreme glucoprivation produced by 2-DG
affects brain sites distinct from those engaged by insulin
or exposure to short day lengths.

Contrary to its lack of effect on torpor, APx either
eliminated or substantially attenuated changes in food
intake induced by 2-DG in hamsters. The counteractive
effect of APx on FI was evident regardless of whether
2-DG treatment also induced torpor. High doses of
2-DG may act on the AP to suppress food intake but
induce torpor via a different mechanism. It can be
argued that the extreme glucoprivation used in this
study made the hamsters ill. The AP has been impli-
cated as a chemoreceptor trigger zone for emesis; the
vomiting response to most but not all emetic drugs is
abolished by APx (reviewed in Ref. 17). It is unknown
whether or not 2-DG at any dose induces vomiting in
Siberian hamsters. We have not observed any such
response with the current doses of 2-DG (2,250 and
2,500 mg/kg) or with lower doses (800 and 1,500 mg/kg;
Ref. 7). Interestingly, a 1,000-mg/kg dose of 2-DG ad-
ministered to neurologically intact deermice (Peromy-
ascus maniculatus) slightly suppressed food intake dur-
ing the first 3 h after treatment, but the same stimulus
elicited hyperphagia in neurologically intact house
mice; no general debilitation was noted in either spe-
cies (27). We cannot, however, completely discount the
possibility that the decrease in FI observed in AP-

**Table 2. Postoperative torpor characteristics of hamsters undergoing spontaneous bouts of torpor**

<table>
<thead>
<tr>
<th>Group</th>
<th>Proportion Torpid</th>
<th>Number of Torpor Bouts</th>
<th>$T_{b_{min}}, ^\circ C$</th>
<th>Duration of Torpor, min</th>
<th>Latency to Display Torpor, days</th>
</tr>
</thead>
<tbody>
<tr>
<td>APx</td>
<td>14/14</td>
<td>26.8 ± 4.3</td>
<td>22.6 ± 0.2*</td>
<td>243 ± 7</td>
<td>113 ± 4</td>
</tr>
<tr>
<td>Sham</td>
<td>6/6</td>
<td>19.8 ± 5.9</td>
<td>24.3 ± 0.3</td>
<td>221 ± 10</td>
<td>11 ± 4</td>
</tr>
</tbody>
</table>

Values are means ± SE. APx, area postrema ablation; $T_{b_{min}}$, minimum body temperature. *Significantly different from values for sham group ($P < 0.05$).
intact hamsters administered 2-DG in the present study were due to nausea (without vomiting) or other ill effects associated with high doses of 2-DG. Similarly, 2-DG may depress food consumption in intact hamsters because the drug produces physiological manifestations of stress. Consequently, elimination of hypophagia in APx hamsters treated with 2-DG may reflect the absence or amelioration of illness or stress after drug treatment, rather than a direct effect on mechanisms that control food intake.

Siberian hamsters decrease, laboratory rats increase, and Syrian hamsters do not change FI in response to 2-DG (22, 37). These apparent species differences may in part reflect differences in methodology rather than interspecific variation; the typical dose of 2-DG administered to rats is usually <400 mg/kg, compared with >1,500 mg/kg in Syrian and Siberian hamsters; also, posttreatment measures of FI were typically short term (<6 h) in rats compared with 24 h for Syrian and Siberian hamsters (20, 22, 39). The response to 2-DG may be dose related; a low dose (125 mg/kg 2-DG) insufficient to increase FI in rats increased FI in Siberian hamsters tested 2–24 h posttreatment (3), whereas higher doses (2,250 and 2,500 mg/kg 2-DG) in the present study decreased FI in neurologically intact Siberian hamsters.

The high doses of 2-DG used in the present study are potentially toxic, at least in some rodent species. The 1,000-, 1,250-, 1,500-, 1,750-, and 2,000-mg/kg doses of 2-DG decrease FI and produce ataxia within seconds that ends several minutes later in some Syrian hamsters (22, 35). The latency between 2-DG injection and torpor was ~40 min for Siberian hamsters, and hypothermia was sustained for 2 h in the present study. Injections of 1,000 mg/kg 2-DG in gerbils resulted in lethargy and depression of FI (26), whereas 500 mg/kg 2-DG (administered iv) produced lethargy in rats (no FI measures were taken) (4). Common features of treatment with high doses of 2-DG in rats, gerbils, and Syrian hamsters include varying degrees of lethargy, stupor, and hypothermia. In contrast, treatment with 2,500 mg/kg 2-DG did not elicit torpor or ataxia in deermice, another photoperiodic species capable of undergoing seasonal torpor (38). In addition, house mice eat more and do not become lethargic when administered 1,000 mg/kg 2-DG (27).

2-DG at 2,500 or 2,000 mg/kg (a lower dose sometimes effective in inducing torpor) elicits a significant and sustained reduction in $T_b$ and subsequent spontaneous recovery to euthermia that resembles naturally occurring seasonal torpor in Siberian hamsters (7). 2-DG has been a convenient tool in studying torpor, because the response is elicited in Siberian hamsters within an hour of injection; spontaneous torpor often is not manifested until Siberian hamsters have been held in short day lengths for up to 16 wk. The use of this drug also has advantages over food deprivation in that hamsters are spared prolonged periods of food restriction that must produce substantial loss of body weight before torpor is displayed. The two forms of torpor (spontaneous and drug induced) yielded comparable results regarding the role of the AP; the nonpharmacological approach is, nevertheless, likely of greater relevance to an understanding of seasonal torpor.

Ablation of the AP had little impact on ad libitum FI of Siberian hamsters that were undergoing seasonal torpor during extended exposure to short day lengths. Short-day adaptations may render further adjustments in feeding unnecessary, whether torpor is utilized or not; the AP may not be involved in the mechanisms that mediate the gradual reductions in FI that precede bouts of spontaneous torpor (32).

The reduction in locomotor activity after spontaneous bouts of torpor was most evident 4 h after torpor onset. The 48% reduction in mean activity recorded in the first 4 h after torpor (corresponding to 3 h before the onset of the dark phase) compares with a 71% reduction reported previously (30). Reduced activity after bouts of spontaneous torpor may represent sleep, a homologous process that, in common with torpor, functions to conserve energy; electroencephalogram slow-wave activity is enhanced in Siberian hamsters after a bout of spontaneous torpor in a manner similar to that found after sleep deprivation (8). Unlike Ruf and Heldmaier (30), however, we did not observe decreased activity over the course of the entire dark phase after a torpor bout. This decrease may represent reduced foraging efforts in the field and reflect reduced energy needs due to savings accrued during torpor. Any of several procedural differences may account for the discrepant findings. Amount of food consumed was negatively correlated with the number of torpor bouts for hamsters at 18 and 23°C (31) and was confirmed in this study for animals kept at 15°C. This suggests that, on a given night for a given individual, different tactics may be used in response to the same challenge; in some instances, hamsters may display torpor, eat little, and move little, whereas others will forego torpor, eat, and move about, presumably in search of food (31).

The role of AP in torpor induced by food restriction was not addressed in the present study; the milder energetic challenges presented by food restriction could be mediated through the AP, but we consider this unlikely. Delays in puberty associated with food restriction were not reversed by ablating the AP in mice (1), and the absence of the AP did not affect the ability of 2-DG to elicit arousal from hibernation in ground squirrels (J. Dark, D. R. Miller, H. H. Bae, and D. A. Lewis, unpublished data), although the suppression of estrous behavior induced by food restriction in Syrian hamsters was reversed by APx (19). Thus many but not all energy-sensitive processes are dependent on the AP.

Collectively, the present results demonstrate that the AP is not necessary for the expression of torpor in male Siberian hamsters but is critical for modifications in FI induced by 2-DG. The AP is not implicated in modulation of FI of Siberian hamsters undergoing spontaneous torpor in winter day lengths. The integ-
rity of the AP is evidently more important for the control of FI than for torpor.

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