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Effect of Short-term Food Deprivation on Reproduction in Female Mice[1]

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ABSTRACT

CF-1 female mice were subjected to 24 or 48 h of food deprivation beginning when they were in estrus or diestrus, or when they were 2 or 12 days pregnant, or on Days 2 or 12 of lactation. Ovulation was delayed by a week or more when 48 h of food deprivation was initiated when the female was in diestrus; lesser delays occurred when food deprivation began in estrus. There was little effect of acute food deprivation on pregnancy. Most females deprived of food beginning on Day 2 of lactation ate their young, but females deprived on Day 12 of lactation rarely did so, These results are discussed in terms of the complexity of interacting factors that determine the degree to which each stage of the female's reproductive cycle is susceptible to disruption by acute food deprivation.

INTRODUCTION

Reproduction is an energy-consuming process. As such it is subject to inhibition both by food scarcity and by any condition that increases the body's other, competing, demands for energy. The female mammal of small size represents an interesting extreme in this regard. The mass of offspring produced by the small female is quite large relative to her own mass, yet her small size dictates high thermoregulatory costs, a paucity of fat reserves, and thus a continuing need to find and consume relatively large quantities of food (Millar, 1977; Peters, 1983). This combination of characteristics makes the reproductive effort of the small female exceptionally susceptible to food scarcity, particularly when it occurs in combination with low ambient temperature (Barnett, 1973; Marsteller and Lynch, 1983, 1985a,b,c; Bronson, 1985; Perrigo and Bronson, 1985).

The concern of this study was with the relative sensitivity of the various stages of a small female's reproductive cycle to disruption by acute food deprivation. Energy costs increase progressively throughout pregnancy and lactation as the mass that must be nourished increases (e.g., Millar, 1975; Randolph et al., 1977; Leon and Woodside, 1983). Intuitively, one might expect sensitivity to food shortage simply to increase directly with these costs. The present study identifies one and possibly two factors that ameliorate this expectation, at least in laboratory mice. First, the tendency to cannibalize young in response to food shortage decreases rather than increases as lactation progresses. Second, the degree to which a stage of reproduction requires immediate support by pituitary gonadotropins may be an important determinant of its sensitivity. Variation in fat reserves throughout the reproductive cycle is not an important consideration.

MATERIALS AND METHODS

A single mating of CF-1 mice was used to produce synchronized births of several hundred females. These females were weaned at 21–23 days of age and housed 6 per cage in a room containing no males. At 85 to 90 days of age the females were housed one per cage in the same room to initiate predictable estrous cycles (see Bronson, 1979). At 100 to 105 days of age the females were delegated at random to one of a series of experiments. These experiments were interrelated in that some of them shared common control groups and all were done simultaneously.

The details of each experiment are presented along with its results. In general, however, food intake, body weight, and some dimensions of body composition, including fat reserves, were monitored in some females throughout an entire reproductive cycle, from prior to insemination until the offspring were weaned. Other females were subjected to either 24 or 48 h of total food deprivation starting at one of six times: while the female was in estrus or diestrus, at 2 or 12 days of pregnancy, or at 2 or 12 days of lactation. The

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effects of these treatments on reproduction and fat reserves were assessed in relation to appropriately treated, ad libitum-fed controls.

Feeding and General Procedures

Unless otherwise noted, females were isolated in polyethylene shoe box cages and given ad libitum amounts of Purina Formulab No. 5008. Bedding was changed whenever an animal was scheduled for food deprivation in order to remove all food dust from the bottom of its cage. In situations where food intake was to be monitored, food was available in a container hanging from one end of the cage. This container measured 6 × 6 × 9 cm. It was constructed with three solid sides and a lipped bottom on the front to catch food dust. The remainder of the front side consisted of horizontal bars placed at 6-mm intervals through which the animal could reach the food. The change in combined weight of the container and its food between measurements was considered to be the amount of food consumed in the intervening time.

Two animal rooms were used in these experiments. Both were maintained at 22 ± 1°C, and each had separate ventilation that replaced the air 12 times an hour. Both rooms were maintained on a 14L:10D light cycle, with lights on at 0600 h. All manipulations (e.g., initiating or terminating food deprivation, weighing animals or food containers) were done between 1300 and 1400 h.

Measurements

The number of young born, the number weaned, and the weight of the litter mass were recorded routinely. All litters were culled at the time of birth to a standard number of 8 young; 4 males and 4 females. Estrous cycles were followed routinely by daily examination of vaginal smears. Where important for calculating delay due to food deprivation, however, ovulation always was verified directly by killing the female while in estrus or metestrus and examing her oviducts for eggs.

Some animals were autopsied and subjected to fat extraction. Reproductive organs and the gastrointestinal tract were removed, the carcass was oven-dried at 75°C, and fat extraction was done with ether in a Soxhlet apparatus. Two measures of body composition were of concern here: total fat content and residual dry weight. The latter was defined as the difference between the dried carcass weight (gastrointestinal and reproductive tracts excluded) and the fat content.

Statistical Assessment

Unless otherwise noted, statistical probabilities recorded in this paper reflect appropriate analyses of variance (ANOVAs), followed by individual treatment comparisons only when justified by the results of the ANOVA.

RESULTS

Change in Food Intake and Body Weight throughout a Reproductive Cycle

Sixteen isolated females were weighed every 3 days for 9 days and food intake was assessed over the same 3-day intervals to establish baselines. Stage of the female's estrous cycle was ignored. Proved stud males then were placed in each female's cage, and all females were inseminated within 4 days. Food intake and body weight were measured every 3 days throughout pregnancy, starting on the day a vaginal plug was observed. All females gave birth on Days 19 or 20 of pregnancy; this difference was ignored, and the last measurements were made on the 18th day after mating. After culling the young to 8 per litter on the day of birth, food intake and body weight again were assessed every 3 days, beginning on Day 2 of lactation and ending on Day 17 when the young were weaned and the experiment was concluded (the day of birth was considered to be Day 0).

The results of this study are shown in the top panel of Fig. 1. As expected, food intake increases with the mass of the female and her young.

Effect of Acute Food Deprivation on Total Body Weight, Fat Content and Residue Weight

Six females were killed either before or after 24 or 48 h of food deprivation, which was initiated at one of five times: before pregnancy, on Days 2 or 12 of pregnancy, or on Days 2 or 12 of lactation. Litters of lactating females remained with them during deprivation. All females were subjected to fat extraction as described earlier. A separate control experiment verified that the handling associated with our food deprivation and weighing procedures had no effect on litter weight or mortality. In this regard it should be noted also that the repeated handling done in the previously described experiment likewise yielded no mortality.

As shown in the top panel of Fig. 1, the amount of body weight lost by a female because of food deprivation generally increases as the mass that is being nourished by the female increases. Prior to pregnancy 48 h of food deprivation caused a loss of only about 6 g, or about 19% of a female's body weight. The comparable figures for late lactation were 15 g and 35%. A notable divergence from this trend occurred on Day 2 of lactation. As is discussed later, this was a reflection of the propensity of food-deprived females to kill and eat their young at that time.

Fat reserves decline during late lactation ($P<0.01$), averaging 60% lower than in the non-

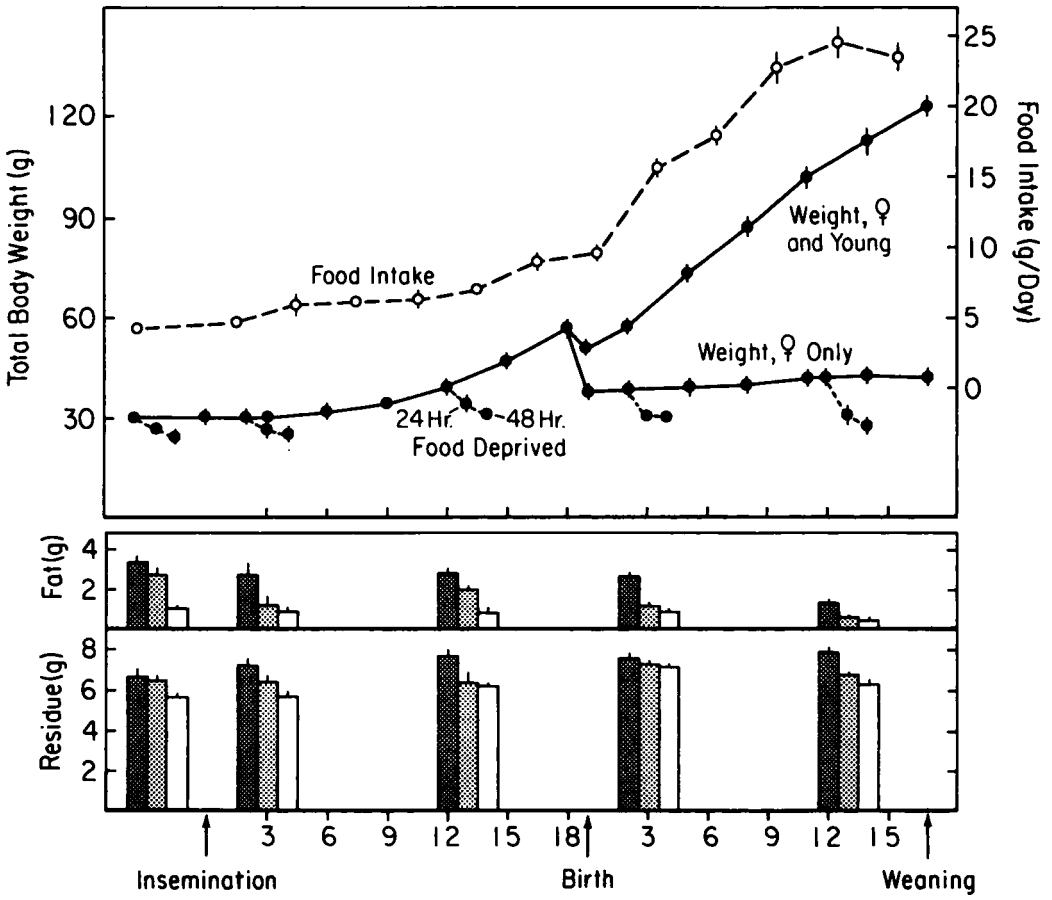

FIG. 1. Body weight, food intake, fat content, and residue weight (mean ± SEM) during the reproductive cycle of the CF-1 female, and the effects of 24 or 48 h of food deprivation initiated at various times during this cycle. In the *top panel* some standard errors are too small to depict graphically, being less than the diameter of the circle indicating the mean. In the bottom panel the *heavily stippled bars* represent ad libitum-fed controls, the *lighter stippled bars* represent females subjected to 24 h of food restriction, and the *white bars* females food-restricted for 48 h.

pregnant condition (middle panel of Fig. 1). The potent effect of food deprivation ($P<0.001$) on fat reserves is obvious in these data. Residual dry weight (total body weight minus fat, water, gastrointestinal tract, and reproductive organs) did not vary greatly throughout the cycle in ad libitum-fed females ($P>0.10$; lower panel of Fig. 1). Food deprivation had profound effects on residue weight except on Day 2 of lactation, again because these females tended to eat their young when deprived of food.

Recovery from Food Deprivation

Recovery from 24 or 48 h of food deprivation was examined only prior to pregnancy. In this experiment food intake and body weight were assessed twice over a 6-day period in 10 isolated females. Then these females were subjected to either 24 or 48 h of food deprivation (5 each). Body weight and food intake were monitored daily thereafter for several days. The results, shown in Fig. 2, suggest that the weight loss caused by food restriction is regained quickly when a female again is allowed unlimited access to food. Recovery is slightly ($P<0.05$) faster among females deprived for 24 h, and is dependent upon the amount of food eaten ($P<0.05$) based upon a repeated-measures ANOVA of weight gain with food consumption. Even females subjected to 48 h of deprivation recovered most of their lost weight within 24 h simply by doubling their food intake. Food intake remained above normal for several days

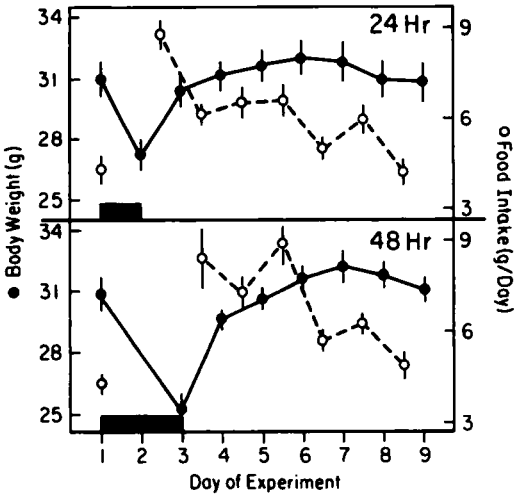

FIG 2. The effect of 24 or 48 h of food deprivation on body weight and food intake in nonpregnant, nonlactating adult female mice. *Horizontal black bars* indicate period of food deprivation.

thereafter, with a slight but nonsignificant tendency to overshoot weight recovery.

Effect of Food Restriction on Ovulation

In this study 45 females were housed alone on one side of a split cage measuring 30 x 30 x 15 cm. The cage was divided into two parts by a wire-mesh barrier. An adult male was housed across the barrier from each female. This procedure produces the most rapid and predictable estrous cycles in house mice (Bronson, 1979). Vaginal smears were obtained from each female for the duration of at least one cycle, as measured from estrus to estrus. All but 3 of these animals experienced normal cycles of either 4 or 5 days in length. Ten of the normally cycling females then were delegated at random to each of four experimental treatments. They were subjected to either 24 or 48 h of food deprivation that was initiated either at estrus or diestrus (diestrus 2 in the case of 5-day cycling females). All 40 females were smeared throughout food deprivation, and continuing on until each had achieved estrus again. They were killed then to verify ovulation.

Each female in this experiment served as its own control. Based on the length of its first cycle, one could predict the number of days expected until its second ovulation. Any delay caused by food deprivation then could be calculated as the difference between the number of days expected and that which was actually observed. The average delays caused by food deprivation are presented in Table 1. Initiating 24 h of food deprivation while a female was in estrus resulted in only 1 day's delay in ovulation (nonsignificant); 48 h of food deprivation initiated at this time yielded an average of 2.4 days' delay ($P<0.05$).

Our females reacted in one of two ways when food deprivation was initiated in diestrus. The vaginal cycles of some of these females simply proceeded on schedule into proestrus and estrus. When these females were killed, however, it was determined that none had ovulated. Two females subjected to 24 h of deprivation and four females subjected to 48 h of deprivation reacted this way. The other 14 females exhibited prolonged diestrus and achieved ovulation only after several days' delay. Only the latter females were used in making the calculations presented in Table 1. Thus it must be emphasized that the 3.2- and 6.8-day average delays noted in this table actually underestimate the effect of initiating food restriction in diestrus. Nevertheless, the effect of initiating food deprivation at this time obviously is potent.

TABLE 1. Mean (± SEM) number of days that ovulation was delayed when females were subjected to 24 or 48 h of food deprivation, starting when they were in either estrus or diestrus (each mean represents 6 to 10 females; see text).

Duration of deprivation	Days of delay	
	Estrus	Diestrus
24 hours	1.0 ± 0.2^{a}	3.2 ± 0.2^{b}
48 hours	2.4 ± 0.4^{ab}	6.8 ± 0.7^{c}

[a-c]Means in this table with different superscripts differ significantly (Scheffe test, $P>0.05$).

TABLE 2. Effect of 24 or 48 h of food deprivation on number and weight of young produced, when food deprivation was initiated on Days 2 or 12 of pregnancy (each value represents 12 pregnant females).

Duration of deprivation	% Females giving birth	Litter size at birth	Mean pup weight (g)	
			Birth	Weaning
Ad lib controls	100	12.3 ± 0.8[a]	1.6 ± 0.1[a]	10.0 ± 0.3[a]
Day 2: 24 h	100	13.5 ± 0.4[a]	1.5 ± 0.1[a]	10.2 ± 0.3[a]
48 h	77	13.2 ± 0.7[a]	1.5 ± 0.1[a]	10.1 ± 0.4[a]
Day 12: 24 h	83	13.0 ± 0.8[a]	1.6 ± 0.1[a]	10.4 ± 0.3[a]
48 h	83	9.7 ± 0.9[b]	1.5 ± 0.1[a]	10.1 ± 0.5[a]

[a,b]Means in the same column with different superscripts do not differ (Scheffe test, $P<0.05$).

Effect of Food Deprivation in the Pregnant Female

A group of inseminated females were subjected to either 24 or 48 h of food deprivation, starting on either Day 2 or Day 12 of pregnancy (12 females per group). Litter size and pup weight were recorded at birth, after which all litters were culled to 8, reweighed, and then reweighed again at weaning (17 days). As shown in Table 2, there was little effect of food deprivation on the reproductive success of these females. A few failed to give birth (nonsignificant), and litter size was reduced significantly in females subjected to 48 h of food deprivation on Day 12 of pregnancy. There were no effects of food deprivation on weight of offspring as assessed either at birth or at weaning.

Effect of Food Deprivation in the Lactating Female

A group of females were allowed to give birth. After culling their litters to 8 young, these females were subjected to either 24 or 48 h of food deprivation, starting either on Day 2 or on Day 12 of lactation (12 lactating females per group). Young were weighed and counted before and after food deprivation and again at 17 days of age when the experiment was concluded. The number of females killing pups in each treatment was analyzed using a 2-way, log-linear contingency analysis (Sokal and Rohlf, 1981).

The most striking result of this experiment was the fact that females subjected to food deprivation on Day 2 of lactation tended to kill and eat their young, whereas those deprived on Day 12 rarely did so ($G = 6.5$, $P<0.05$). All females subjected to 48 h of food restriction on Day 2 ate some of their young, as did 8 of 12 females deprived for 24 h. The average number eaten in each litter was over 6 with 48 h of deprivation, but only about 1 with 24 h of deprivation (Table 3). As recorded earlier in Fig. 1, one result of this behavior was that these females lost much less weight than expected, and they suffered no loss at all in their residual dry weight. In contrast, only 4 of 12 females food deprived for 48 h on Day 12 of lactation ate their young, and only 2 of 12 ate young

TABLE 3. Effect of 24 or 48 h of food deprivation on behavior of the lactating female, and on the weight of their young, when deprivation was initiated on Days 2 or 12 of lactation (each value represents 12 lactating females).

Duration of restriction	% Females killing young	% Young killed	Change in weight during test period*		Weight of pups at weaning
			Controls	Deprived	
Ad lib controls	0	0	–	–	10.0 ± 0.3
Day 2: 24 h	33	8	+0.6 ± 0.1	+0.1 ± 0.1	10.0 ± 0.5
48 h	100	77	+1.2 ± 0.1	−0.5 ± 0.1	11.3 ± 0.9
Day 12: 24 h	17	4	+1.2 ± 0.1	+0.3 ± 0.1	9.3 ± 0.2
48 h	33	12	+2.4 ± 0.1	−0.9 ± 0.1	7.4 ± 0.3

*Mean (± SEM) change in grams experienced by the litter during the 24- or 48-h period of food deprivation.

when deprived for 24 h. Those females eating young ate only 1 or 2 of their offspring.

Food deprivation of the mother also had an obvious effect on the weight of the surviving young (Table 3). Recovery of lost growth was not completed at weaning in those young whose mothers had been deprived of food for 48 h starting on Day 12 of lactation ($P<0.001$).

DISCUSSION

The objective of this research was to determine the relative susceptibility of each stage of a female mouse's reproductive cycle to disruption by food deprivation. The effect of *long-term* food restriction on reproduction has been examined several times in female rats and mice (e.g., Berg, 1965; Zamini, 1978; Glass and Swerdloff, 1980; Lederman and Rosso, 1980; McClure, 1981; Marsteller and Lynch, 1983, 1985a,b,c). While long-term food restriction is a good tool for exploring the relationship between available food and ovulation, it is not a good tool for comparing the relative sensitivity of each of the later stages of a female's reproductive cycle. The onset of each stage depends upon the successful completion of the preceding stage, and the confounding action of chronic food restriction limits the utility of this approach. Stage-by-stage probing with *short-term* food deprivation provides an acceptable alternative (see McClure, 1962, 1966).

Fundamental to understanding the small female mammal's reproductive response to acute food deprivation is the recognition that these animals have a paucity of energy reserves (see also Millar, 1975; Merson and Kirkpatrick, 1981; Peters, 1983). Prior to pregnancy our CF-1 females had about 3 g of ether-extractable fat. Assuming that all of this could be mobilized when confronted with food deprivation (which it could not), these females store a maximum of only about 30 kcal in their fat. This is the equivalent of only about 2 days' normal food intake. Twenty-four hours of food deprivation did not reduce greatly the fat reserves of these animals, probably due to the relatively large amount of food that is being processed at any one time in the stomach and intestines. Food deprivation for 48 h largely eliminated fat stores, however, and it necessitated the catabolic mobilization of additional energy from sources other than fat (i.e., residual dry weight dropped by 15%; see Fig. 1).

The relationship between energy demand and energy stores becomes more critical as the reproductive cycle progresses. Pregnancy is not a particularly costly process, energy-wise, but lactation is (Fig. 1). Indeed, by late lactation the caloric value of a small female's fat reserves is the equivalent of only about one-eighth of her daily food intake (3 h), and now even 24 h of food deprivation necessitates mobilization of energy from nonfat sources (see Vernon and Flint, 1984). This progressive change in energy demand, when placed against a background of minimal fat reserves, must be acknowledged as the most fundamental force determining susceptibility of each stage of the small female's reproductive cycle to disruption by food deprivation.

Another factor of importance here, however, is the energy modulation of reproductive effort by cannibalism. Infanticide is not an unusual phenomenon in small mammals, and often it seems related to the need to cull litter size for one reason or another (Hausfater and Blaffer-Hrdy, 1984; McClure, 1981; Marsteller and Lynch, 1983, 1985b,c). Lactating CF-1 females obviously use their pups as an emergency food source early in lactation, but not later. Cannibalism was a common response to food deprivation on Day 2 of lactation, but not on Day 12. This was in spite of the fact that the energy reserves of the latter females were totally exhausted after 48 h without food; they were cold to the touch and in obvious thermoregulatory distress despite the relatively warm ambient temperature. Still, only a few killed and ate any of their young.

The adaptive significance of the transition during lactation from cannibalism to noncannibalism is not immediately clear, but two potential explanations may be offered here. First, these results may suggest an adaptive strategy wherein the female balances three factors: the time and energy already invested in her pups, the time and energy required yet until the pups can be weaned, and her own risk of starving. Selection may have acted to promote survival of the young while increasing the lactating female's risk if food deprivation is encountered when the young are approaching the time when they can disperse and fend for themselves. In and of itself this explanation is not adequate, however, because 12-day-old pups are still several days away from independence.

A second, but not mutually exclusive, explanation involves the thermoregulatory development of the pups as it determines the amount of time a female living in the wild can

devote to foraging. Early in lactation, pups are heterothermic and this must limit greatly the amount of time a female can forage (see Harland and Millar, 1980). As the pups grow and develop mature thermoregulation, the female will be allowed progressively longer and longer foraging bouts. At some point, her drive to forage may simply replace the tendency to use pups as a food source. Certainly recovery from food deprivation could be rapid if the female could find a new food source (see Fig. 2). Either or both of these explanations may underly the present results, but at this time their meaning remains somewhat unclear.

The role of pituitary hormones in modulating the female's response to food restriction throughout her reproductive cycle likewise is not totally clear. Food restriction inhibits the secretion of all gonadotropic hormones, at least in the rat (e.g., Campbell et al., 1977). Thus one should expect all stages of a female's reproductive cycle to be readily susceptible to acute food deprivation, except late in pregnancy when the placenta takes over some of the functions of the pituitary. Thus it is not surprising that acute food restriction interferes greatly with ovulation in mice (see also McClure, 1966). This effect is particularly obvious when food deprivation is initiated in diestrus, when the next ovulating release of luteinizing hormone (LH) is being programmed by estradiol (Bronson and vom Saal, 1979); the slowly elevating titers of estradiol characteristic of late diestrus require chronic LH stimulation. Initiating acute food restriction at estrus, just after ovulation has occurred, yields much less delay in achieving the next ovulation.

Likewise, as should be expected, acute food deprivation yielded only minor effects on late pregnancy. Some embryo mortality resulted from 48 h of food deprivation in 12-day-pregnant females, probably due to the secondary effects of energy insufficiency, but all in all these effects were relatively minor. Quite surprising, however, was the almost total lack of effect of food deprivation beginning on Day 2 of pregnancy. Direct support by LH is required for the ovarian secretion of progesterone and estradiol necessary to prepare the uterus for implantation (reviewed by Yoshinaga, 1982). The lack of effect seen here is even more surprising since two previous studies have documented a dramatic inhibition of implantation in other strains of mice (McClure, 1962; Bruce, 1963). The reason for the difference between these reports is not known, but it may have to do with the rate at which LH secretion recovers from acute energy insult in the various strains.

In summary, the degree to which a particular stage of a female house mouse's reproductive cycle is susceptible to disruption by acute food deprivation seems to depend upon a complexity of factors. These include the energy jeopardy generally inherent in small size, the energy costs of the stage in question, the amount of direct support by pituitary gonadotropins required at that stage, and some complex and poorly understood strategies involving cannibalism.

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