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Dietary induced weight gain and recovery of body weight in Mongolian gerbils

Maryann McCarthy

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DIETARILY INDUCED WEIGHT GAIN AND RECOVERY OF BODY WEIGHT IN MONGOLIAN GERBILS

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DIETARILY INDUCED WEIGHT GAIN AND RECOVERY OF BODY WEIGHT IN MONGOLIAN GERBILS

BY

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B.A., Nazareth College of Rochester, 1985

A Thesis

Submitted to the Graduate Faculty

of the University of Richmond

In Candidacy

for the Degree of

MASTER OF ARTS

in

Psychology

August, 1987 Richmond, Virginia

LIBRARY UNIVERSITY OF RICHMOND **VIRCINIA 23173**

I would like to take this opportunity to extend my appreciation to the chairman of this thesis committee, Dr. Frederick J. Kozub, for his invaluable assistance, generosity of spirit and for his interest which began long before the experiment did. I also would like to thank Dr. Kenneth A. Blick for always making time for me, and for having both a sense of equity and a sense of humor. I wish also to thank Dr. Edith Ott for giving me the tools necessary for professional growth and a good environment in which to use them. I want to express my gratitude to Kevin T. Williams for his constant support and encouragement, and for being a computer genius. Finally, I wish to thank my mother, Rita C. McCarthy, for without her advice and love none of this would have been possible.

Dietarily Induced Weight Gain and

Recovery of Body Weight in

Mongolian Gerbils

MaryAnn McCarthy

University of Richmond

Running Head: WEIGHT GAIN

Abstract

The purpose of this study was to explore the capacities of male and female gerbils for recovery from increased weight gain produced by ingestion of high fat food. Thirty-six gerbils, assigned to either 20% or 40% groups with nine males and nine females in each percent weight gain group, were fed powdered chow under baseline conditions, then switched to a high fat diet until they reached target weight increase, and then returned to powdered chow. The 40% group was eliminated from the comparisons due to the failure of the gerbils to reach criterion. No sex differences were found in the 20% gerbils weight gains to target weights. They drank significantly less water when eating the powdered chow, and there was a marginal interaction of diet condition and sex on calorie consumption, with the male and females consuming similar caloric amounts on the high fat diet.. There was no "voluntary diminution" of food intake during the early days of recovery. It was concluded that food contamination and the young age of the gerbils were confounding variables, and recommendations for future research were made.

Dietarily Induced Weight Gain and Recovery

of Body Weight in Mongolian Gerbils

The regulation of food intake and energy balance represents a complex physiological process, involving the recognition and integration of many different types of signals. The physiological system has a tendency to stabilize body weight by processes in which behavioral, metabolic, neural, and hormonal signals are united into a cohesive pattern. This study was intended to explore and investigate regulatory capacity in male and female gerbils, with respect to their recovery from excessive weight gain due to dietary manipulations. In order to establish the background on which this study was based, it was first necessary to look at in detail various aspects of regulatory mechanisms.

A salient feature of complex organisms is the ability to maintain a stable internal environment, in a changing external environment. Organisms maintain this homeostatic level by the constant monitoring of their internal state, and make fine adjustments to maintain their physiological system at its optimal values. Organisms defend a particular body weight. For example, rats who's weight was displaced by food deprivation or forced feeding, have shown compensatory changes, (increased or decreased ad-lib feeding), which allowed for a return to pre-manipulation weight (Hallonquist & Brandes,

1984). They regulate their food intake not only according to their daily needs, (short term regulation), but also according to their average body weight, (long term regulation). Short term satiety signals which influence food intake from day to day include blood glucose, body temperature, and osmotic factors; the long term satiety signals involve metabolites related to body fats (Hoebel & Teitelbaum, 1966).

All animals defend a weight below which they eat more and above which they eat less. In the majority of the research which manipulates body weight, the concept of a body weight "set point" is used to refer to situations where the organism's absolute body weight imposes constraints on its subsequent rate of body weight change. The process by which deviations from a given setpoint engage processes to reinstate the setpoint is called negative feedback. Deviations from a setpoint prompt reactions that decrease those deviations. As deviations lessen, so do the reactions to them, until the organism's stable state is reached. Most conceptual ideas of hunger are based on control systems which operate through negative feedback mechanisms. Mayer's (1952) glucostatic theory of hunger proposed that information on energy needs is conveyed via blood sugar levels, and acts as a signal to the brain about the amount of energy available, or needed. As the glucostatic mechanism was insufficient to account for long term regulation, Mayer added

a complementary lipostatic component. The lipostatic theory postulated that a circulating metabolite of the body's excess energy, stored as fat and known as free fatty acids (FFA), were responsible for long term regulation. When circulating levels of FFA were high, the organism increased its consumption of food. When FFA were low, the organism consumed less because the fat was being stored rather than utilized, therefore, fat stores were determinants of the weight at which the organism stabilized its body weight setpoint (Keesey, 1980). Researchers further postulated that the glucostatic and lipostatic mechanisms regulated the body's food intake on both a daily and a long term basis (LeMagnen, Devos, Gaudilliere, 1973).

A great deal of research has been generated regarding the nature of regulatory mechanisms in mammals. Adult animals normally regulate their body weight at relatively stable levels by controlling food consumption and energy expenditures, thus defending against weight excesses and deficits. While many studies have concentrated on the effects of deprivation and subsequent recovery from deprivation, investigations of overfeeding have focused on obesity produced in hypothalamically damaged animals. Other forms of producing obesity have received less attention (Sclafani & Springer, 1974). The various experimental procedures that have produced overeating and obesity have included: surgical isolation (via knife cuts), or lesioning the

ventromedial hypothalamus; chronic injections of insulin; electrical brain stimulation; and a very few studies have used dietary manipulations (Sclafani & Springer, 1976).

Although research has been done using animal models other than rats, [Fenton & Carr, 1951, and Lemmonier, 1972 (mice); and Romas, Hornshuh, & Leveille, 1978 (dogs)], the bulk of the research has concentrated on rats. While there is a paucity of data on gerbils with respect to recovery from dietary induction of obesity, data on overfeeding in the rat, a species inhabiting a nearby ecological niche, was considered. The findings from the rat data were necessary to provide procedural information and results from which relevant considerations were taken into account.

There have been several nutritional experiments that have demonstrated the capability of rats to become obese when fed highly palatable and caloric diets for long periods (Ingle, 1949; Mickelson, Takahashi, & Craig, 1955; Miller, Mirsky, Caul, & Sakata, 1969; Nisbett, 1972). The most commonly used dietary means of inducing obesity in rats has been to feed them a high fat diet (Geliebter, Liang, & Van ltallie, 1984; Scalfani, 1980). Unpublished research at the University of Richmond showed that when gerbils were fed a diet of two parts purina lab chow powder and one part vegetable fat one third of them increased their body weight by 40%. High fat diets promote obesity in

two ways: increased caloric intake and improved efficiency of food utilization. Intake of food was increased by stimulating the appetite, fat increased the efficiency by which dietary energy was converted into stored energy, and the rats stored a greater proportion of their intake in the form of body fat (Schemmel, 1976; Schemmel & Mickelson, 1974; Wood & Reid, 1975). Although rats fed a high fat diet consumed less food than when they were fed a low fat diet or a standard lab chow diet, this difference was typically not sufficient to compensate for the increased caloric density of the high fat ration, and caloric intake rose by 10% to 20% more (Carlisle & Stellar, 1969; Corbit & Stellar, 1964; Schemmel, Mickelson & Gill, 1970). The failure of rats to compensate fully for the caloric density of high fat diets did not appear to represent an inadequacy in their caloric monitoring ability, but rather reflected their preference for the taste and texture of the diets (Carlisle & Stellar, 1969; Hamilton, 1964). Corbit and Stellar (1964) demonstrated that the rats excessive intake of the high fat diet was not due soley to the diet's caloric density but that the stimulus properties of the high fat diet were sufficient to produce a high intake.

The magnitude of the reaction to the stimulus properties of food has been shown to be a function of body weight, and experiments have indicated that stored energy has inhibitory effects on feeding as a function of the

amount stored (Cohn & Joseph, 1962; Corbit & Stellar, 1964; LeMagnen, Devos, & Gaudilliere, 1973; Steffens, 1975). Sclafani (1980) has stressed the relationship between the animal's body weight and its setpoint. Rats on a chow diet have defended their body weight when subjected to a variety of homeostatic challenges and have gained the reputation of being precise regulators of body weight. But, when given a choice, rats rejected chow diets for those high in fat, and, became moderately to extremely obese (Adolph, 1947; Faust, Johnson, & Stern, 1978). Evidence for the inhibitory influence of obesity upon food intake is shown by Cohn & Joseph (1962) and Hoebel &Teitelbaum (1966). In their studies, intact rats were made obese by force feeding, and/or by the chronic administration of potamine-zinc insulin. When the forced feeding and the insulin administration ceased, the animals ate very little, lost weight, and reached their pre-treatment levels. As their weights decreased, food intake increased. On the other hand, Faust et al. (1978) have shown that an important feature of dietarily induced obesity is that while it is largely reversible by returning the animals to a low fat diet, it could lead to permanent elevations in fat cells and thus, elevate the animal's setpoint and subsequent body weight. Some researchers have found that rats fed a high fat diet for twenty five weeks, initially lost weight after being returned to a chow diet, but their weight reached a plateau at a level significantly higher

than the controls fed only the chow diet (Peckham, Entenman, & Carrol, 1962). This apparently permanent elevation in body weight was attributed to the adipocyte hyperplasia induced by the high fat diet.

The degree of obesity induced by diets high in fat is also influenced by the strain and sex of the animal (Sclafani, 1980). Female rats normally gain less weight and body fat following weaning than do males, and the differences are potentiated by the availability of high fat food (Schemmel et al., 1970). Sex related differences have also been reported in animals fed high fat rations with regard to their body fat distribution. However, when body fat is expressed as a percentage of body weight, female rats fed high fat diets accumulated as much body fat as did males (Lemmonier, 1972; Schemmel & Mickelson, 1974).

There has been a controversy in the literature pertaining to sex differences in the efficacy of ventromedial hypothalamic (VMH) lesioning. Various studies have indicated that overeating and obesity are much more pronounced in the female (Cox, Kakolewski & Valenstein, 1969; Valenstein, Cox, & Kakolewski, 1969). However, other studies have indicated that VMH lesioned males obtain the same degree of obesity when fed highly palatable diets (Gold, 1970). In particular, Grossman & Hennessy (1976) established that female rats with knife cuts to the posterior hypothalamus, doubled food

intake and quadrupled rate of weight gain, whereas males with similar cuts, showed no significant increases in either food intake or weight gain. They felt this was indicative of a sex difference that suggested a possible fundamental difference in the organization of neural mechanisms related to satiety and body weight setpoint.

Nance, Gorski, and Panksepp (1976) posited that male and female rats regulate their food intake differently and have proposed a model to account for the differences. The model states that there is an initial sexually bipotential neural base for body weight and food intake regulation that is analogous to the regulation of gonadotropins and sex behavior. During a critical period, exposure or nonexposare to androgens results in sexual differentiation of the brain, in either a masculine or feminine pattern of energy regulation. Again, analogous to gonadotropin and sex behavior regulation, sex specific hormones secondarily reinforce and activate the expresion of this sexual dimorphism in energy regulation. Nance et al., (1976) further conceptualized sexual differentiation of body weight and food intake regulation as occurring along a continuum, the two ends of which are bounded by animals with VMH lesions and lateral hypothalamic (LH) lesions. They believed males to be relegated toward the VMH lesion end, and showed greater dependence upon short term factors for controlling feeding behavior. They

placed females towards the LH end, relying more on long term factors to regulate feeding. Therefore, they postulated that the same basic regulation of energy balance which resulted in increased food intake and body weight in VMH lesioned animals was to a lesser degree, the equivalent regulatory pattern in males having a higher body weight and food intake than females. The foundation for the model was based on hypothalamic lesion regulation research which showed that female rats, after VMH lesions, exhibited a more dramatic hyperphagia than males. Males already demonstrated a regulatory pattern similar to VMH lesioned animals and, therefore, showed a less dramatic effect (Valenstein et al., 1969).

As mentioned earlier, there exists little data concerning food intake and body weight regulation in the gerbil. In recent years, researchers have added substantially to the body of work on regulatory food behaviors in rats and also have begun to explore animals in the same genus, specifically hamsters (Silverman & Zucker, 1976; Borer, 1974; Rowland, 1984; DiBattista, 1986). However, little attention has been aimed at a comparative analysis in the gerbil. One of the few studies found the gerbil to differ from the rat in the manner in which both animals experienced weight loss as a function of water, and food and water deprivation conditions (Dunstone, Krupski, & Weiss, 1971). Another investigation found the gerbil to be similar to the rat in its

adaptability to intermittent feeding (Silverman & Zucker, 1976). The remainder of the studies have had as their main concern gerbil metabolism and have focused on physiological as opposed to behavioral mechanisms. The effect of diet on cholesterol metabolism has been of primary interest. In contrast to the rat, the gerbil has been found to be a lipemic species and therefore useful in the study of lipid metabolism and the mechanisms thought to inhibit atherosclerosis (Hegsted & Gallagher, 1967). The gerbil has been reported to show similarities to humans in its responses to dietary cholesterol manipulation (Robinson, 1986). For this reason it was felt that any information regarding obesity in the gerbil might also contribute to the understanding of mechanisms involved in human obesity. None of these studies nowever, provided information about the behavior of an animal when maintained on. a calorie dense diet, or on recovery of setpoint when taken off the diet.

Rothwell and Stock (1979) have stated:

In the search for a better understanding of energy balance, much attention has been focused on the development of obesity in humans and experimental animals, and we would like to suggest that investigations into the recovery from temporary obesity provide an alternative approach that could be equally instructive.. (p. 1 034)

As previously stated, this investigation was intended to explore the male and female gerbil's regulatory capacities and recovery functions from

excessive, high fat caloric intake and increased body weight, due to dietary manipulations. Sclafani and Springer (1976) found considerable variability in the weight gains displayed by rats fed a highly palatable diet, although all did outgain the animals fed only chow.

The major focus of this study was to explore the capacities of gerbils for recovery from increased weight gain produced by dietary manipulations. The dietary manipulation was used because as Van ltallie, Gale & Kisselef (1978) noted, a dietary manipulation was minimally invasive, (as compared with a repeated stomach loading procedure, brain lesions, or serial insulin injections), and was likely to give more "naturally produced results". The major questions asked were: 1.) did the induced weight gain alter the setpoint, i.e. did the animals return to baseline weights after they reached target weights; 2.) were there sex differences in recovery time to the setpoint, and secondarily, were there sex differences in weight gain to target weights; 3.) was recovery time to setpoint dependent on the initial amount of weight gain and sex; 4.) would there be a "voluntary diminution" in food intake during the early days of recovery, [as noted by Corbit & Stellar (1964) and Hoebel & Teitelbaum (1966)].

Method

Subjects

Thirty six adult gerbils, 18 males and 18 females, were obtained from Tumblebrook Farms, West Brookfield, Massachusetts. The animals were maintained under constant illumination, in a temperature controlled room (72 \degree ± 2 \degree F). So had ad-lib access to Purina chow powder and water for ten days prior to the experiment to insure stable eating.

Apparatus

Ss were individually housed in 18- x 18- x 24-cm. cages. Water was available in inverted graduated cylinders with metal drinking spouts attached to the front of the cage. Food was available from Whamann food cups with lids.

Diet

Ad libitum feeding consisted of Purina lab chow powder. The calorie content of this powder was 4.16 k/calories per gram. The treatment diet consisted of.2 parts powdered Purina lab chow to 1 part vegetable shortening, and its calorie content was 5.75 k/calories per gram. This was a 38% increase over the powdered food.

Procedure

The male and female gerbils were randomly assigned to two groups

with nine males and nine females in each group. After a ten day baseline access period to an ad-lib diet of powder, the animals were switched to the high fat diet. Ss were maintained on the high fat diet until they reach a target weight increase over baseline weight of 20%, for group #1, and 40% increase, for group #2. At this point, each animal was maintained on the diet for an additional day, to insure that there was not a random flux in target weight. The animals were then switched back to powder until they returned back to baseline, or for a maximum of fourteen days. Daily food intake and body weight measures were taken and used as an index of the regulatory capacities of the animal.

Results

The number of subjects in the female 40% group was reduced by one due to the death of one subject. This in no way effected the following comparisons as they were made on the 20% groups only.

Because of the exploratory nature of this thesis, the probability levels for ANOVA and MANOVA F values were expanded to report anything up to .10 significance.

Although the expectation was that all subjects would have achieved

criteria weight gain targets, only 6 out of nine males and 7 out of nine females achieved target weights. In the 40% group 0 out of 9 males and 1 out of 8 females achieved target weights. Therefore as subjects in the 40% group did not reach criterion, analyses on the comparisons of the 20% and the 40% groups were not made. Data for the 40% groups with respect to percent weight gains achieved have been presented in tabular form after presentation of 20% groups results.

A single factor independent groups ANOVA was used to determine if there were weight gains to target weights. None of the skewness and kurtosis comparisons exceeded 1.96 and therefore were not significant. The assumption of homogeneity of variance was satisfied as F max {2,5) was 1.059 and not significant. The mean number of days to reach criterion, (and standard deviations), for the male and female groups were 15.17 {3.31) and 16.57 (3.41) respectively. This comparison was not significant, F (1.11) $=$.56, p. > .1 0, and indicated that there were no differences in group means to reach target weight.

 A 2 x 3 MANOVA repeated on the dimension of dietary conditions was used to compare male and female body weights under baseline, high fat and return to powder diet conditions. Skewness and kurtosis comparisons were made with 43% found to be significant. The assumption of homogeneity of

variance was satisfied since F max (2,5) for baseline conditions was 5.26 and not significant. Since no standard deviation in each of the three conditions was 3 or 4 times larger than another, no significant variance within conditions was indicated. The means and standard deviations are presented in Table 1.

Insert Table 1 about here

There was no significant interaction of sex and conditions $[F (2,22) =$ 2.18, p. > .1 0] on body weight. There was a significant main effect of sex on the means $[F 1,11] = 48.33$ p. < .05]. There was also a significant main effect of conditions $[F (2,22) = 110.49, p. < .05]$. The Student Newman-Keuls multimean test revealed that of the three differences in means, all three were significant. Significance of mean body weights is shown in Table 2. Insert Table 2 about here

These results indicate that there were significant differences in the mean body weights in each of the three conditions. The mean body weights in the baseline condition were different from those in the return condition; the mean body weights in the high fat condition were different from those in the baseline condition; and the mean high fat body weights were different from the mean return body weights. The mean body weights for both males and

females in the high fat condition were significantly higher than in the baseline and return conditions. The mean body weights in the return condition were significantly higher than in the baseline condition.

 A 2 x 3 MANOVA repeated on the dimension of dietary conditions was also used to compare water intake (corrected for 100 grams body weight) for males and females under baseline, high fat and return to powder diet conditions. No evidence of skewness and kurtosis was found . F max $(2,5)$ = .20 for baseline conditions was found to be not significant thus there was no significant deviation among the groups. No significant variance within conditions was indicated since standard deviations in each of the three conditions were less than 3 or 4 times larger than each other. The means and standard deviations are presented in Table 3.

Insert Table 3 about here

No significant interaction of sex and conditions was found on fluid intake $[F (2,22) = .03, p. > .10]$, nor was there a significant main effect of sex. Results however did show a significant main effect of conditions on mean fluid intake $[F (2,22) = 4.52, p. < .05]$. A Student Newman-Keuls indicated that only two of the three differences were significant. Significance of mean fluid intakes are shown in Table 4.

Insert Table 4 about here

Specifically, there was no significant difference between baseline mean fluid intake and return mean fluid intake. There was however a difference between mean fluid intake in the return and high fat conditions, and in the baseline and high fat conditions. These results indicate that when eating powdered food, the gerbils drank equivalent amounts of water, yet while eating high fat food, they drank significantly less water than when eating powdered chow.

Of interest was the caloric intake of subjects during the three phases of the manipulation. In order to determine if any significant differences in caloric intake (corrected for body weight) existed between males and females under the three diet conditions, another 2×3 MANOVA, repeated on the dimension of dietary conditions, was used. Skewness and kurtosis comparisons were made with 6.6% found to be significant. F max $(2,5) = 1.01$, p. > .05, was not significant, thus satisfying the assumption of homogeneity of variance. The standard deviations in each of the 3 conditions were not 3 or 4 times larger than each other, indicating no significant variance over repeated measure conditions. Calorie means corrected for body weight, and their respective standard deviations are presented in Table 5.

Insert Table 5 about here

To avoid making a beta error while doing this exploratory research, the probability levels were expanded up to .1 0. The interaction of sex and conditions for calorie intake was found to be $F(2,22) = 2.50$ with a probability of .105. Because this is $>$ than p. .10, it does not warrant a simple effects analysis. However, to avoid making a beta error, the interaction of sex and condition was found to be marginally significant. Figure 1 presents the marginal interaction.

Insert Figure 1 about here

There were no significant main effects of sex or conditions. However the results present the possibility that sex and baseline, high fat, and return conditions might have had and effect on caloric intake. It appears that the males consumed the least amount of calories at baseline, more in high fat and then dropped towards baseline caloric levels in the return to powder condition. In contrast the females consumed the most calories at baseline, and decreased consumption through high fat and return conditions. The males and females consumed approximately the same numbers of calories in the high fat condition.

To determine if there was a "voluntary diminution" in food and calorie intake represented by a decrease in in food intake and calorie intake (corrected per 1 00 grams of body weight) from the last day on high fat food to the first day of return to powdered food, $a \, 2 \times 2$ MANOVA repeated on the dimension of food and calorie intake under last day and first day conditions was performed. No evidence of significant skewness or kurtosis was found since none of the indices for either calorie or food intake exceeded 1.96. The assumption of homogeneity of variance was satisfied since F max (2,5) for last day and first day was 0 and not significant for either calorie of food intake. The means and standard deviations over the last day and first day conditions for food intake and calorie intake are presented in Tables 6 and 7 respectively.

Insert Tables 6 & 7 about here

No significant interaction was found for food intake $[F (1,11) = 1.23, p. >$ 10], nor for calorie intake $[F(1,11) = 1.81, p. > .10]$. No significant main effect on food intake was found for sex $[F (1,11) = 1.63, p. > .10]$, nor on calorie intake $[F (1,11) = .023, p.$ > .10]. There was also no significant main effect on food intake for conditions $[F(1,11)]$, = 2.02, p. > .10], nor on calorie intake $[F (1,11) = .29, p. > .10]$. The results indicated that there were no

significant differences in the gerbils' mean food or calorie intake on the last day of high fat and the first day of return to powder.

For each subject in the 40% groups, the baseline weight, highest weight over baseline and the percent of weight reached over baseline weight are presented in Table 8 for males and Table 9 for females. Also included in the tables is the mean percent weight reached over baseline and the standard deviations, along with the range of weights.

Insert Tables 8 & 9 about here

Discussion

The failure of all but one gerbil to reach 40% criteria was especially disappointing in this exploratory study since this ruled out addressing any proposed comparisons between 20% and 40% groups. In the male 40% group, the mean highest percent over body weight actually achieved was 13.46%. In the female group the mean highest percent over body weight achieved was 28. 71. It was not possible ·to add to the 20% group those female gerbils from the 40% group that achieved at least 20% over their baseline weights because they had not been switched to powdered food when they reached their target weights. Rather than speculate at this point about the reasons for the failure

of the 40% groups to reach criteria, a discussion of the findings in the 20% groups might provide clarification since both groups sustained the same experimental problems.

It was not possible to look for differences in recovery time to setpoint based on weight gain group and sex. since after 4 weeks on high fat food only one female gerbil had reached 40% criteria. Likewise, it was not possible to look for sex differences in recovery time to setpoint due to the fact that the 13 gerbils who reached criteria in the 20% group had stabilized at the end of the fourteen day cutoff at weights closer to their respective high fat weights than baseline weights, thus they never returned to setpoint. Even though there was one less male than female gerbil reaching target weight, there were no sex differences in weight gain time to target weights.

Given that males are larger than females it was not surprising to find that the males weighed significantly more than the females. What was interesting was the significant differences in body weights over the three conditions. The mean body weights of males and females were significantly higher in high fat than in baseline or return conditions, and significantly higher in the return than in the baseline conditions. However, this is not necessarily attributable to alterations in setpoint, but more likely due to the fact that the gerbils in the study were still growing and increasing in weight.

Sclafani and Springer (1976) have noted that along with strain and sex, age is an important factor that might influence the development of obesity. The target weights reached by the 20% over baseline group were within the range of normal weights for young mature gerbils. The mean weights in the return condition were closer to high fat weights than baseline weights and, again, were representative of normal weights for young mature gerbils. The experimental manipulation seemed to have coincided with the gerbils' normal growth. It is likely then that the results were influenced by this normal growth pattern. The high fat food seemed to have slightly elevated the gerbils normal developmental weights, and after return to powder, their body weights stabilized at values within normal ranges for gerbils in that age group.

The significant effect of diet conditions on water intake are less well understood. The male and female gerbils drank equivalent amounts of water in the powdered food conditions and just slightly less in the high fat condition. Researchers have found in previous food and water intake restriction studies that water restriction reduced eating dry food (Reynierse, Scavio, & Spanier, 1970). Perhaps the sensory "moistness" of the high fat food was sufficient enough to lower water .intake from dry powdered baseline and return conditions.

Because of the exploratory nature of this study, the confidence intervals were lowered so as to avoid overlooking any finding of scientific merit. With this framework in mind, it is possible to look at the marginal interaction of sex and diet conditions on the gerbils' calorie intake. Researchers have stated that diets high in fat increase calorie intake and stimulate fat deposition (Carlisle & Stellar, 1969; Corbit & Stellar, 1964; Schemmel, Mickelson & Gill, 1970). In the present study neither sex nor diet conditions alone had a significant effect on calorie intake. However, there was some evidence that their interaction did have ah effect on the amount of calories consumed. The males seemed to have increased caloric intake from baseline to high fat and then decreased it in the return condition, whereas the females had a steady decline in intake from baseline through return. Both males and females ate similar amounts of food in the high fat condition. These findings need to be more fully explored in a future study, because they could have been effected by food contamination. Unfortunately, the gerbils urinated and defecated in their food cups. This may have had an effect on their subsequent food consumption, and effected the weight of the food consumed since caloric intake was computed using food intake measures. Therefore, caloric intake may also have been distorted. Furthermore, Tanimoto (1942) has described the gerbil as actively burrowing day and night to make underground nesting

sites. This behavior was evidenced in the present study in the gerbils continuous "burrowing" in their food cups which resulted in scattering both powdered and high fat food, and may have caused a decrease in the accuracy of the intake measures.

Taken together these variables could have been responsible for the lack of any differences between the last day high fat food/calorie intake and the first day return to powder food/ calorie intake. The findings were in contrast to those of Corbit & Stellar (1964) and Hoebel & Teitelbaum (1966), who noted a "voluntary diminution" of food intake during the early days of recovery.

Various researchers have speculated on the components that influence intake and occurrence of obesity in animals. They are in agreement that those components include: availability of food; type of diet; percent of caloric change; animal's taste preference to the diet;health of animals;age; strain; and sex (Kanarek, 1975; Geliebter et al., 1984; Panksepp, 1976). It has already been pointed out that the gerbils were not mature, and that they severely contaminated their food. This contamination along with their tendency to throw their food seriously limited the availability of the food. In addition, under the high fat diet, many of the gerbils lost a substantial amount of their fur, (this was not termed a "serious problem" after

conversation with Dr. P. Coleman, D.V.M., 1987). However, although the room temperature was maintained at a constant of 72° \pm 2° F, it was observed that many of the gerbils had some respiratory distress; they wheezed, had runny eyes and crusty noses, and in contrast to the continual gnawing on their cages or "playing" with their food cups that was found in the baseline conditions, ffle gerbils slept or moved about slowly when on the high fat diet. (The likely cause of the hairless was due to rubbing the high fat diet on their coats and the subsequent grooming, apparently to clean it off). After removal . from the high fat diet, the animals made substantial recoveries, regained the lost hair, and appeared to be in good health. Also, again through casual observation, the animals who reached criteria were those who appeared to have had the least amount of hairloss.

The type of diet used and the gerbils' preference for this diet may also have contributed to the failure to achieve 40% criteria, as well as ill health. Sclafani and Springer (1976) found considerable variability in the weight gains of rats fed high fat diets, resulting in only modest weight gains. Yet when fed diets containing high fat, high sugar and high calcium, increased gains were noted. In a very recent study by Nairn, Brand and Kare (1987) rats offered a choice between high fat and powdered diets preferred the unadulterated diet. This led the researchers to conclude that although under

no-choice conditions, the rats increased calorie intake, the high fat diets were either unpalatable to the rats or a preference for them was transient. They felt the reason these results were in contrast to previous literature was due to the fact that mixing fat with powder led to a dilution of micronutrients, in addition to changes in calorie density. They stated that even though the sensory properties of the high fat diet were appealing, in the long run the absence of important nutrients led to a preference for the unadulterated foods. This might have accounted for the poor health of the animals since this nutritional imbalance came during a critical time in their growth and development.

Other investigators (Hegsted & Gallagher, 1987; Robinson, 1986) have used vegetable oil adulterated diets rather than hard vegetable fat diets. For future studies it might be necessary to experiment with different types of diets that satisfy the gerbils' taste preferences, meet their nutritional requirements, and provide additional calories to produce weight gain.

In an unpublished preliminary investigation done at the University of Richmond as a mini-group project with multiple experimentors, there were the attendant problems of non-uniformity of data collection. In this study there were strict controls over data collection procedures, with the same method of collection used by all three persons weighing intake and body

weights. Also, one scale was used soley for body weights and the other for weighing the food cups. This was done to ensure consistency and uniformity in data collection by eliminating calibration errors. This might have accounted for why 2 out of 6 gerbils reached 40% over body weight in that study and only 1 out of 17 reached 40% in the present study.

Although the results were unanticipated and beset with problems, the study was informative, (given the exploratory nature of this thesis), and instructive for future researches. Important to future studies are: 1.) the age of animal, with stable weight as a crucial factor; 2.) strict control over data collection procedures; 3.) nutritional balance of the adulterated diet with maintenance of increased caloric content, but tailored to the gerbils' taste preferences; 4.) initial target criteria set at 20-25% weight gain and then, if warranted, an increase in the amount of targeted weight gain; 5.) changes in the tooa cup position and angle so that the food intake would not be restricted but contamination would be minimized or eliminated. This could be accomplished by placing the cup on an incline, by using a triangular block of wood. This would prevent the gerbils from sitting on the food cups and defecating and urinating into them.

The original comparisons proposed and the questions presented remain valid areas of exploration and more studies need to be conducted, (using the

appropriate experimental modifications suggested here), to yield information on dietarily induced weight gain and subsequent body weight recovery in adult gerbils.

References

- Adolph, E. F. (1947). Urges to eat and drink in rats. American Journal of Physiology, 151, 110-125.
- Borer, K. T. (1974). Absence of weight regulation in exercising hamsters. Physiology & Behavior, 12, 589-597.
- Carlisle, H. J., & Stellar, E. (1969). Caloric regulation and food preference in normal, hyperphagic, and aphagic rats. Journal of Comparative Physiological Psychology, 69, 107-114.
- Cohn, C., & Joseph, D. (1962). Influence of body weight and body fat on appetite of "normal" lean and obese rats. Yale Journal of Biological Medicine, 34, 598-607.

Coleman, P. (1987). Personal communication to F.J. Kozub, March 1987.

- Corbit, J. D., & Stellar, E. (1964). Palatability, food intake, and obesity in normal and hyperphagic rats. Journal of Comparative Physiological Psychology, 58, 63-67.
- Cox, V. C., Kakolewski, J. W., & Valenstein, E. S. (1969). Ventromedial . hypothalamic lesions and changes in body weight and food consumption in male and female rats. Journal of Comparative Physiological Psychology, 67, 320-326.

DiBattista, D. (1986). Voluntary ethanol consumption and obesity in golden

hamsters. Physiology & Behavior, 36, 41-45.

- Dunstone, J. L, Krupski, G. M., & Weiss, C. S. (1971). Weight loss in gerbils (meriones unguiculatus) continously deprived of food, water, and both food and water. Psychological Reports, 29, 931-936.
- Faust, I. M., Johnson, P. R., & Stern, J. S. (1978). Diet-induced adipocyte number increase in adult rats: a new model of obesity. American Journal of Physiology, 58, 63-67.
- Fenton, P. F., & Carr, C. (1951). The nutrition of the mouse: responses of four strains to diets differing in fat content. Journal of Nutrition, 45, 225-234.
- Geliebter, A., Liang, J. T., & Van ltallie, T. B. (1984). Effects of repeated isocaloric macronutrient loads on daily food intake of rats. American Journal of Physiology, 247, R387-R392.
- Gold, R. M. (1970). Hypothalamic hyperphagia: males get just as fat as females. Journal of Comparative Psychology, 71 , 347-356.

Grossman, S. P., & Hennessey, J. W. (1976). Differential effects of cuts through the posterior hypothalamus on food intake and body weight in male and female rats. Physiology & Behavior, 17, 89-102.

Hallonquist, J. D., & Brandes, J.S. Ventromedial hypothalamic lesion in rats: gradual elevation of body weight set-point. Physiology & Behavior (33),

1984.

Hamilton, C. L. (1964). Rat's preference for high fat diets. Journal of Comparative Physiological Psychology, 58, 459-460.

- Hegsted, D. M., & Gallagher, A. (1967). Dietary fat and cholesterol and serum cholesterol in the gerbil. Journal of Lipid Res, 8, 210-214.
- Hoebel, B. G., & Teitelbaum, P. (1966). Weight regulation in normal and hypothalamic hyperphagic rats. Journal of Comparative and Physiological Psychology, 61, 189-193.

Ingle, D. J. (1949). A simple means of producing obesity in the rat.

Procedures in Social, Experimental, Biological Medicine, 72, 604-605.

- Kanarek, R. B. (1975). Energetics of meal patterns in rats. Physiology & Behavior, 17, 395-399.
- Keesey, R. E. (1980). "A set-point analysis of the regulation of body weight," in Obesity, ed. A. J. Stunkard, Philadelphia: W. B. Saunders, 1980.
- LeMagnen, J., Devos, M., Gaudilliere, J. P. (1973). Role of a lipostatic mechanism in regulation by feeding of energy balance in rats. Journal of Comparative Physiological Psychology, 84, 1-23.
- Lemmonier, D. (1972). Effect of age, sex, and site on the cellularity of the adipose tissue in mice and rats rendered obese by a high fat diet. Journal of Clinical Nutrition, 51, 2907-2915.
- Mayer, J. (1952). The glucostatic theory of regulation of food intake and the problem of obesity. Bulletin of the New England Medical Center, 14, 43-49.
- Mickelson, 0., Takahashi, S., & Craig, C. (1955). Experimental obesity: production of obesity in rats by feeding high fat diets. Journal of Nutrition, 57, 544-548.
- Miller, R. E., Mirsky, I. A., Caul, W. F., & Sakata, T. (1969). Hyperphagia and polydipsia in socially isolated monkeys. Science, 165, 1027-1028.
- Nance, D. M., Gorski R. A., & Panksepp J. (1976). Neural and hormonal determinants of sex differences in food intake and body weight. in Hunger: Basic Mechanisms and Clinical Implications, eds. D. Novin, W. Wyrwicka and G. A. Bray, New York: Raven Press, pp. 257-271.
- Nisbett, R. E. {1972}. Hunger, obesity, and the ventromedial hypothalamus. Psychological Review, 79, 433-453.
- Panksepp, J. (1976). On the nature of feeding patterns- primarily in rats. in Hunger: Basic Mechanisms and Clinical Implications, eds. D. Navin, W. Wyrwicka and G. A. Bray, New York: Raven Press, pp. 257-271.
- Peckham, S. C., Entenman, C., & Carrol, H. W. (1962). The influence of a hypercaloric diet on gross body and adipose tissue composition in the rat. Journal of Nutrition, 77, 187-197.

Robinson D. G. (1986). Lipid metabolism studies. The Gerbil Digest, 12.

- Romsos, D. R., Hornshuh, M. J., & Leveille, G. A. (1978). Influence of dietary fat and carbohydrate on food intake, body weight and body fat of adult dogs. Experimental Biological Medicine, 157, 278-281.
- Rothwell, N. J. & Stock, M. J. (1979). Regulation of energy balance in two models of reversible obesity in the rat. Journal of Comparative and Physiological Psychology, 93, 1024-1034.
- Rowland, N. (1984). Metabolic fuel homeostasis in golden hamsters: fasting, refeeding, glucose and insulin. American Journal of Physiology, 147, R57-R64.
- Schemmel, R. (1976). Physiological considerations of lipid storage and utilization. American Zoology, 16, 661-670.
- Schemmel, R., & Mickelson, 0. (1974). Influence of diet, strain, age and sex on fat depot mass and body composition of the nutritionally obese rat. In Vague, J., and Boyer, J., eds.: The Regulation of Adipose Tissue Mass. New York, American Elsevier Publishing Co., pp. 238-253.
- Schemmel, R., Mickelson, 0., & Gill, J. L. (1970). Dietary obesity in rats: body weight and body fat accretion in seven strains of rat. Journal of Nutrition, 100, 1941-1948.
- Sclafani, A. (1980). "Dietary Obesity," in Obesity, ed. A. J. Stunkard,

Philadelphia: W. B. Saunders, pp. 166-181.

- Sclafani, A., & Springer, D. (1976). Dietary obesity in adult rats: similarities to hypothalamic and human obesity syndromes. Physiology & Behavior, 17, 461-471.
- Silverman, H. J., & Zucker, I. (1976). Absence of post-fast food compensation in the golden hamster (mesocricetus auratus). Physiology & Behavior, 17,271-285.
- Steffens, A. B. (1975). Influence of reversible obesity on eating behavior, blood glucose and insulin in the rat. American Journal of Physiology, 208, 1-5.
- Tanimoto, K. (1942). Studies on mammals in relation to bubonic plague in manchuria, part I. Zoology Magazine, Tokyo, 54.
- Valenstein, E. S., Cox ,V. C., & Kakolewski, J. W. (1969). Sex differences in hyperphagia and body weight following hypothalamic damage. Annuals of the New York Academy of Sciences, 157, 1030-1048.
- Van ltallie, T. B., Gale, S. K., & Kissileff, H. R. (1978). Control of food intake in the regulation of depot fat: an overview. In Katzen, H. M., and Mahler, R.J., eds.: Diabetes. Obesity and Vascular Disease, Vol. 2.
- Wood, J. D. & Reid, J. T. (1975). The influence of dietary fat on fat metabolism and body fat deposition in meal-feeding and nibbling rats.

British Journal of Nutrition, 34, 15-24.

Means and Standard Deviations for Body Weight of Male and Female Gerbils in Baseline. High Fat. and Return Conditions

Newman-Keuls: Ordered Differences for Baseline. High Fat. and Return **Conditions**

*indicates significance at .05 level

Means and Standard Deviations for Fluid Intake (corrected for body weight) of. Male and Female Gerbils in Baseline. High Fat. and Return Conditions

Newman-Keuls: Ordered Differences for Baseline. High Fat. and Return **Conditions**

*indicates significance at .05

Means and Standard Deviations for Caloric Intake (corrected for body weight) of Male and Female Gerbils in Baseline. High Fat. and Return Conditions

Figure 1

Mean Calorie Intake (corrected for body weight) for Male and Female Gerbils in Baseline. High Fat. and Return Conditions

Means and Standard Deviations for Food Intake (corrected for body weight) of Male and Female Gerbils on Last Day of High Fat. and First Day Return **Conditions**

Means and Standard Deviations for Caloric Intake (corrected for body weight) of Male and Female Gerbils on Last Day of High Fat. and First Day Return **Conditions**

Baseline Weight. Highest Weight Achieved. Percent Over Baseline Weight. and Means and Standard Deviations for Male 40% Group Gerbils

Baseline Weight. Highest Weight Achieved. Percent Over Baseline Weight. and Means and Standard Deviations for Female 40% Group Gerbils

