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Pregnancy and Lactation in the Obese Rat: Effects on Maternal and Pup Weights

BARBARA J. ROLLS AND EDWARD A. ROWE

Department of Experimental Psychology, University of Oxford
South Parks Road, Oxford OX1 3UD, Great Britain

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ROLLS, B. J. AND E. A. ROWE. *Pregnancy and lactation in the obese rat: Effects on maternal and pup weights.* PHYSIOL. BEHAV. 28(3) 393–400, 1982.—Lister hooded female rats, fed palatable high energy foods and chow, weighed significantly more than chow-fed control rats before mating. A smaller proportion of the obese rats became pregnant, and they lost more litters in lactation. When litters survived (7±1 pups), maternal weight changes differed between groups during lactation. The controls gained 6.2±3.2 g, whereas the obese rats lost variable amounts of weight despite the continued availability of the palatable diet. The rats that were heaviest at mating and parturition and which showed the largest non-fetal weight gains in pregnancy (i.e., the "large weight loss group") lost 60.6±4.8 g, while less obese rats which showed similar non-fetal gains to controls (i.e., the "small weight loss group") lost 24.6±3.2 g. Thus the weights of all groups converged and were similar after three weeks of lactation, but diverged again after weaning. During lactation the total energy intakes and amounts of protein consumed by the obese rats were significantly below those of controls, and total fat intake was significantly elevated. Although litter size and pup weights did not differ significantly at birth, pups of obese mothers weighed significantly less than those of controls at weaning. Maternal obesity in lactation appears to influence both body weight regulation and lactational performance.

Body weight regulation Dietary obesity Diet selection Eating Lactation Obesity Pregnancy

PREGNANCY and lactation are examples of common, normal physiological states in which extreme changes in body weight and body fat occur. Despite this, little is known about the factors which determine food intake and fat deposition in these states. The present experiments examine the effects of established obesity on food intake and body weight in the pregnant and lactating rat. Effects of obesity on reproduction have previously had little experimental attention because the most frequently studied obese animals, those with genetic or hypothalamic obesity, are relatively infertile [2]. Obesity can also be induced in rats by offering free access to diets with high palatability and/or high fat content [27,29]. Rats with dietary obesity are also relatively infertile [9, 24, 33]. We found, for example, that only 67% became pregnant compared with 86% of normal weight control rats. Since some of the rats did become pregnant, it was possible to determine how obesity affects food intake and body weight during reproduction.

In rats of normal body weight, maternal body fat stores increase during pregnancy. The mobilization of these fat stores, and hyperphagia normally meet the energy demands of milk production in lactation. It is not clear whether the characteristic hyperphagia and changes in maternal body weight are regulated in pregnancy and lactation relative to the pre-existing nutritional status of the mother or the nutritional requirements of the offspring. The use of a variety of palatable, high energy foods to induce obesity has made it possible in the present study to determine whether the characteristic over-consumption of these foods seen in virgin rats also occurs in addition to the hyperphagia of pregnancy and lactation. The variable levels of obesity induced by palatable foods have also made it possible to study the effect of pre-existing variations in body fat stores on energy intake and body weight regulation in pregnancy and lactation.

In their studies of dietary self-selection using pure nutrients, Richter and Barelare [22] suggested that appetite can be used as a guide to assess nutritional needs in pregnant and lactating rats. More recently this suggestion has been supported by the finding that in pregnancy and lactation dietary self-selection follows the varying nutritional requirements of the organism so that protein intake increases [12]. In the present experiments we determined whether such "nutritional wisdom" was seen when rats were choosing between palatable diets (relatively high in fat and low in protein) and laboratory chow.

Preliminary reports of this work have been made to the Nutrition Society of Great Britain [25,26] and to a Symposium on the Body Weight Regulatory System [24].

METHOD

Subjects

Fifty-three female hooded Lister rats, 8–12 weeks of age were matched for body weight and allocated to an experimental group which received palatable high-energy foods (see Procedures) and chow (n=39), and a control group (n=14) which received only chow. An additional group (n=25) received a high energy liquid diet and chow.

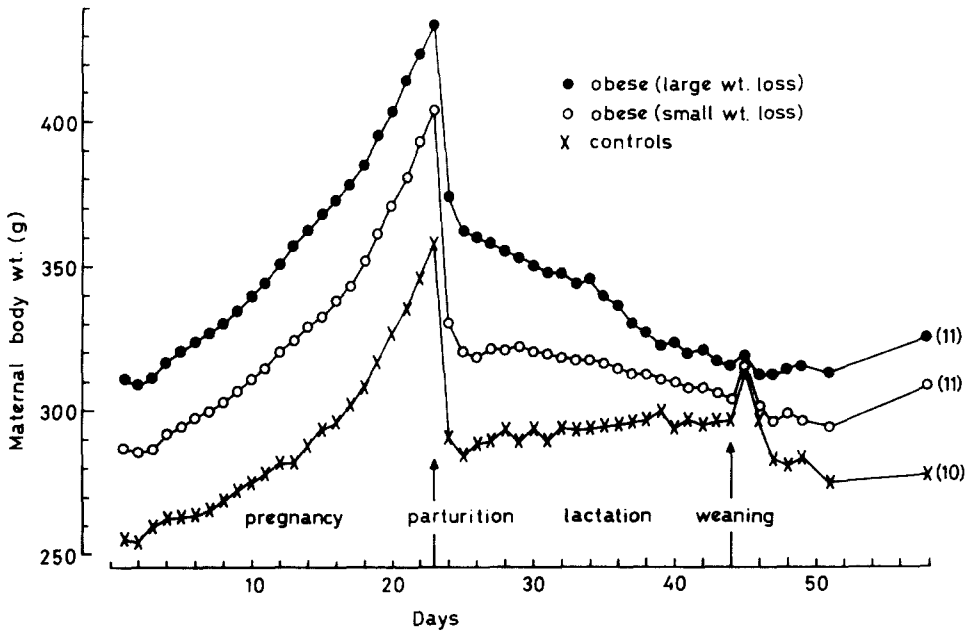

FIG. 1. Mean body weight changes during reproduction in normal weight rats eating chow or in obese rats eating chow plus palatable, high energy foods. Only dams that kept 6–8 pups throughout lactation are included. Obese rats were divided into a "large weight loss" group and a "small weight loss" group by a median split of the total weight loss during lactation. Numbers of rats per group are shown in parentheses.

Procedures

The rats were individually housed in breeding boxes (30×30×15 cm) provided with shredded paper bedding which was changed daily. Lighting (12 hr light, 12 hr dark) and temperature (23±2°C) were controlled. All animals received free access to water and a composite pelleted chow diet (Dixons FFG(M), energy value 15.1 kJ/g; 16% protein; 51% carbohydrate; 2% fat—percentages by weight from manufacturers' values) throughout the study. The experimental groups received free access to the palatable, high-energy foods and chow or the liquid diet and chow, and the control group received only chow. The palatable foods included plain salted potato chips (Golden Wonder), energy value 23.4 kJ/g, protein 5.9%, carbohydrate 50%, fat 38%; cheese crackers (Crawford's Cheddars), energy value 22.6 kJ/g, protein 10.9%, carbohydrate 50.6%, fat 32.6%; chocolate chip cookies (Lyon's Maryland Cookies) energy value 20.3 kJ/g, protein 4.8%, carbohydrate 63.6%, fat 25.2% (all percentages by weight from manufacturers' values). The liquid diet consumed by the additional group of rats was prepared from un-sweetened evaporated milk (Carnation) 45%, cooking oil 30%, and sucrose 25%, giving an energy value of 18.1 kJ/g, protein content 3.5%, carbohydrate 30.5%, fat 35%, all percentages by weight. Ten to 20 weeks after the palatable foods were introduced, experimental and control rats were housed with normal weight male rats. Impregnated females were returned to the home cage 24 hr after the appearance of sperm plugs. The experimental rats continued to receive the high energy foods and chow during pregnancy and lactation; the control rats continued to receive only chow. After parturition all litters were culled to eight pups each within 48 hr; litters of mothers which retained 7±1 pups throughout lactation were classified as surviving litters. Litters were weaned 21 days after parturition.

Measurements

Body weights of the adult females were recorded daily from one week before mating, during pregnancy and lactation, and for three weeks after the litters were weaned. Daily energy intakes were determined by measuring the amount eaten of each individual food, subtracting the spillage of each, and multiplying by the energy value of each food. The use of white shredded paper as bedding made it possible to locate and measure the spillage of each type of food. The energy intakes of protein, carbohydrate, and fat were determined by calculating the amounts of each nutrient eaten in each of the foods multiplied by the energy density of the nutrients (protein 17 kJ/g, carbohydrate 16 kJ/g, fat 37 kJ/g from [19]). Energy intakes in lactation included maternal intakes and during the last part offspring intakes.

Statistics

Separate analyses of body weight changes and food and nutrient intakes were made before mating, during pregnancy, during lactation, and after the litters were weaned, and included only animals which retained 6–8 pups per litter throughout lactation. Insufficient numbers of litters of obese mothers fed the liquid diet survived to allow statistical analyses.

Body weights and energy and nutrient intakes were analysed by two factor analyses of variance. Because the weight losses in the lactating obese rats fed the palatable high energy foods appeared to fall into two groups, these rats were divided into two groups post hoc based on a median split of maternal weight loss in lactation, and are referred to as the "large weight loss group" and the "small weight loss group." Each analysis included these two experimental groups and the control group as one factor and time (days) as the other factor, and rates of change were examined by ex-

traction of the linear polynomials with time. At critical stages in the experiment comparisons of groups were made using the Student's t-test (2-tailed) using error terms from the analysis of variance. Other comparisons were made using the Student's t-test for correlated groups (2-tailed). The mean daily weights of litters before weaning were also compared between groups by an analysis of variance of the experimental groups and the control group with time, with extracted linear polynomials.

RESULTS

Maternal Body Weight

The mean body weights of control dams and of dams that ate the palatable high energy foods, are shown in Fig. 1. All the groups had similar body weights before the palatable foods were introduced. At mating both obese groups were significantly heavier than the controls ($t(19)=5.5$, $p<0.001$ for large weight loss group; $t(19)=3.2$, $p<0.01$ for small weight loss group). During pregnancy the large weight loss rats gained 120.7±7.3 g, the small weight loss rats gained 118.0±6.0 g, and the controls gained 103.8±6.2 g. These gains did not differ statistically, and there was no difference in the pattern of weight changes in pregnancy between the groups. However, the non-fetal weight gain (i.e., the difference between immediately post-partum and mating weights) did vary between the groups. In the controls this was 40.9±4.9 g, in the small weight loss group it was 42.5±5.5 g, and in the large weight loss group it was 61.8±7.0 g ($t(19)=3.2$, $p<0.01$ large weight loss group compared with controls). At parturition weight losses in the three groups were not significantly different (58.7±5.7 g for the large weight loss rats; 72.8±6.2 g for the small weight loss rats; and 67.7±5.4 g for the controls). There was a dramatic difference between the groups in the body weight changes during lactation (differences in the rate of weight change $F(2,577)=424.6$, $p<0.001$). The controls gained an average of 6.2±3.2 g, whereas the large weight loss group lost 60.6±4.8 g, and the small weight loss group lost 24.6±3.2 g so that body weights of the three groups converged and at weaning the large weight loss group was only slightly but significantly heavier than the controls ($t(19)=2.2$, $p<0.05$), and the small weight loss group and the controls had similar body weights. The weight losses of the obese groups correlated with body weights at mating ($r=0.43$, $p<0.02$), with non-fetal weight gains in pregnancy ($r=0.51$, $p<0.02$), and with body weights immediately after parturition ($r=0.78$, $p<0.001$). (Weight change in lactation in the control group was also correlated with body weights at mating $r=-0.85$, $p<0.01$; and post-partum weights, $r=-0.70$, $p<0.05$). The response to weaning differed between the groups. The controls showed a transient increase in weight which could have been due to accumulated milk or a delay in the reduction of food intake, and then body weight decreased. The obese groups showed a smaller increase in weight with weaning and then body weights increased so the groups again diverged.

Twenty nine percent of the rats giving birth had lost the entire litter by seven days after parturition. The body weights of these mothers were similar to those of the large weight loss group (weight at mating was 312.3±11.1 g, weight at parturition was 432.0±11.4 g, and weight after parturition was 366.6±11.3 g so the non-fetal gain was 54.2±9.6 g). Only 27% of obese mothers eating the liquid diet and chow retained 7±1 pups per litter throughout lactation. The body weight changes during pregnancy and lactation were similar in these rats to those of obese rats eating the palatable high energy foods (non-fetal weight gain in pregnancy 57.0±7.9 g, weight loss during lactation 71.7±6.1 g).

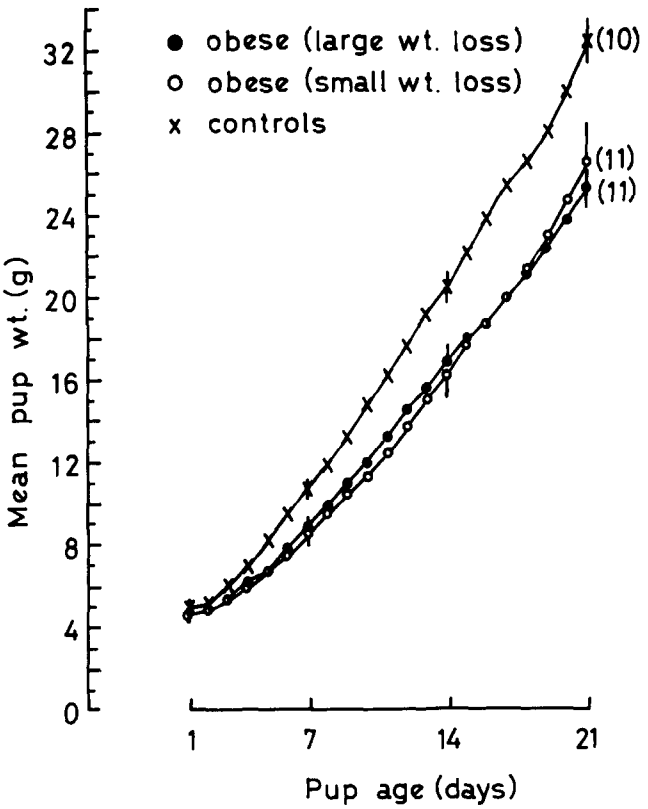

FIG. 2. Mean pup weights in litters of 6–8 pups suckled by mothers described in Fig. 1. Number of litters used in calculating mean weights are shown in parentheses and vertical bars indicate SEM.

Survival and Growth of the Pups

Nine out of 31 litters of the obese mothers fed on the palatable high energy foods were lost (often, but not always, cannibalized) while 1 out of 11 litters of the controls did not survive past the first week of lactation. Six out of 11 litters of the obese mothers eating the liquid diet did not survive, and only three of the mothers retained 7±1 pups until weaning.

The number of pups born in each litter did not differ significantly between the groups (11.7±1.2 pups in large weight loss group; 12.8±1.0 pups in small weight loss group; 11.3±0.9 pups in control group; 11.0±2.1 pups in liquid diet group).

The mean birth weights per litter of pups in the obese and control groups (from litters which retained at least six pups until weaning) did not differ (Fig. 2). However, the pups of control mothers gained weight more rapidly than the offspring of obese mothers (difference in rates of weight gain $F(2,579)=127.2$, $p<0.001$) and by the first week of lactation the mean pup weights in the two obese groups were each significantly lighter than those of the control mothers, $t(19)=2.0$, $p<0.05$. Pups in both obese groups grew at the same rate and continued to diverge from the control pups throughout lactation so at weaning they were on average 6.4 g lighter than the controls, $t(19)>5.0$, $p<0.001$ in each case. The pups of obese mothers eating the liquid diet were similar

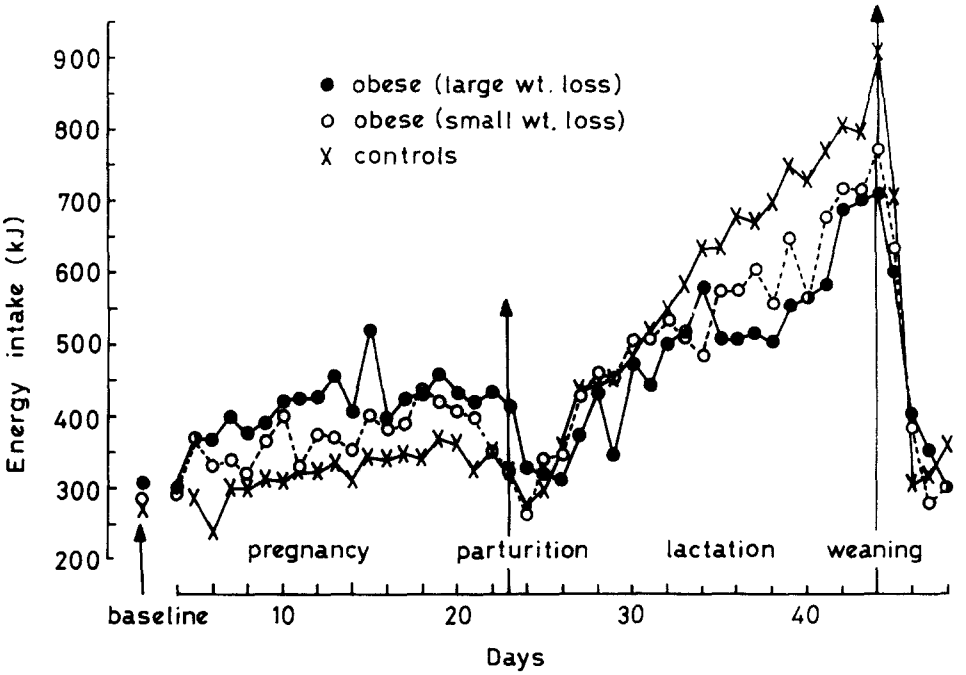

FIG. 3. Mean daily metabolizable energy intakes of the two obese groups and the control group of rats described in Fig. 1. In late lactation intakes reflect food consumed by pups as well as mothers.

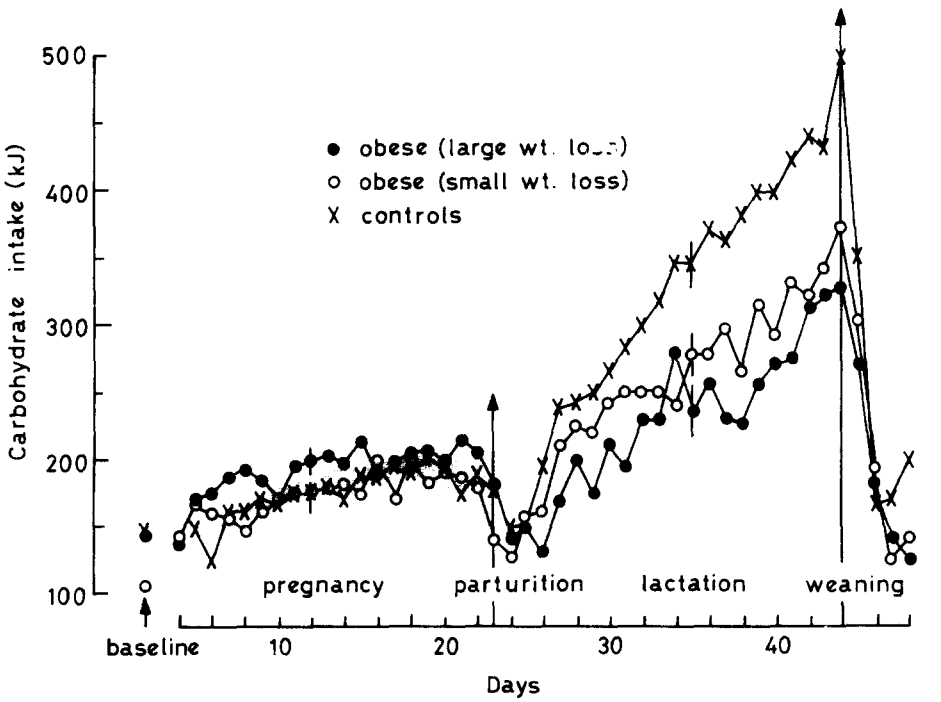

FIG. 4. Mean daily carbohydrate intakes of the two obese and the control groups described in Fig. 1.

in weight at birth to the other groups (4.8±0.1 g) and were substantially lighter than the control offspring at weaning (21.5±1.7 g).

Energy and Nutrient Intakes

The metabolizable energy intakes of the three groups of mothers differed significantly during pregnancy, $F(2,28) = 10.7$, $p < 0.01$ (Fig. 3), and these differences were consistent with the differences in the non-fetal weight gain shown by these groups. Thus the intakes of the large weight loss group, which also had the highest non-fetal weight gains in pregnancy were greater than those of the low weight loss

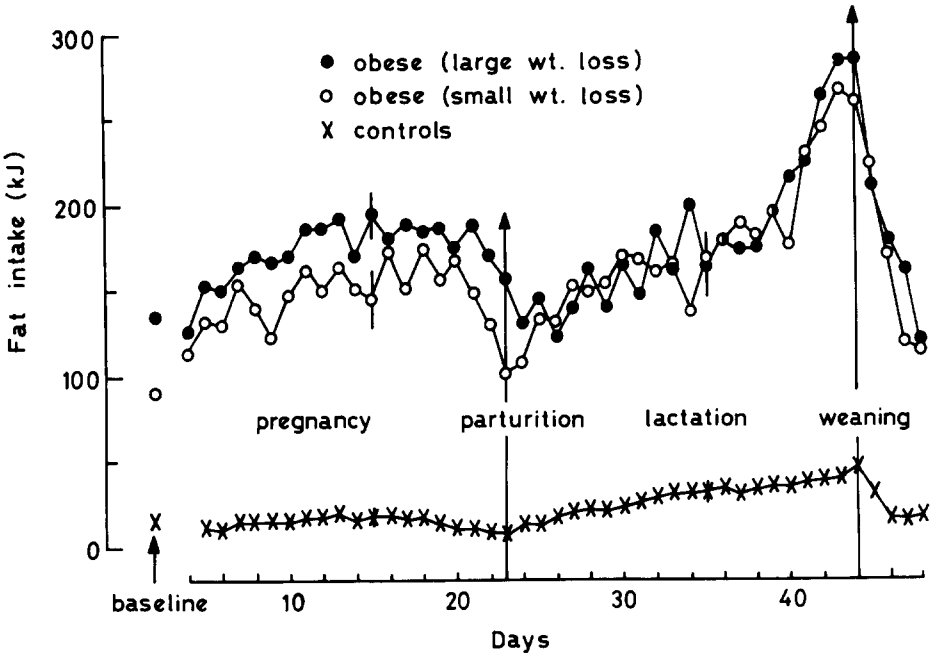

FIG. 5. Mean daily fat intakes of the two obese and the control groups described in Fig. 1.

group, $t(20)=1.9$, $p<0.1$, and the intakes of the latter group were significantly greater than the control intakes, $t(19)=2.8$, $p<0.01$.

During lactation the large weight loss group ingested significantly less energy than the small weight loss group, $t(20)=2.0, p<0.05$, and the control group, $t(19)=4.6, p<0.01$; and the small weight loss group ingested significantly less energy than the control group, $t(19)=2.6, p<0.05$ (difference between all the groups $F(2,29)=10.5$, $p<0.01$). Differences between the groups in early lactation (days 0–8) and late lactation (days 9–12) were also examined separately. In early lactation the control group and the small weight loss group consumed similar amounts (mean daily intake: control group 430±20 kJ, small weight loss group 430±11 kJ) and both consumed significantly more, $t(19)=2.3$, $p<0.05$, than the large weight loss group (395±10 kJ). The obese mothers eating the liquid diet ingested 423±24 kJ per day. In late lactation the intakes (which included intake by the pups) of the obese groups were not significantly different (mean daily intakes: large weight loss group 575±47 kJ, small weight loss group 618±21 kJ) but were significantly less than those of the controls (740±17 kJ, comparison with large weight loss group, $t(19)=6.2$, comparison with small weight loss group, $t(19)=4.8$, $p<0.001$). The dams fed the liquid diet ingested only 503±24 kJ per day. After weaning there was a delay of one day before intakes returned to pre-mating levels [18]. During the day when intake was still elevated, that of the controls was higher than that of both obese groups, which could partly explain the greater rise in body weight at that time.

The carbohydrate intake did not differ between groups during pregnancy (see Fig. 4). During lactation the carbohydrate intake of the control rats diverged from that of the obese groups (difference in rate of change of intake, $F(2,563)=40.1, p<0.001$) and the control rats ingested significantly more carbohydrate than the obese groups ($F(2,29)=42.8$, $p<0.001$, controls versus large weight loss group $t(19)=9.1, p<0.001$, controls versus small weight loss group $t(19)=6.4, p<0.001$). The small weight loss group also ingested significantly more carbohydrate than the large weight loss group, $t(20)=2.8$, $p<0.01$.

The total fat intake of the obese groups was very much higher than that of the controls (pregnancy $F(2,28)=181.6$, $p<0.001$; lactation $F(2,29)=175.9, p<0.001$; see Fig. 5). During pregnancy the large weight loss group ate significantly more fat than the small weight loss group, $t(20)=2.8, p<0.01$, but the fat intakes of the two obese groups did not differ during lactation so total fat intake does not provide the explanation for the differences in weight loss of these groups.

The total protein intakes of the obese groups were significantly lower (pregnancy $F(2,28)=96.7$, $p<0.001$; lactation $F(2,29)=208.6, p<0.001$) than those of the controls (see Fig. 6). Despite the relatively lower protein intakes in pregnancy, the pups of the obese groups were similar in weight to those of the controls at birth. During pregnancy the protein intakes of the two obese groups were similar but during lactation the protein intakes of the large weight loss group were significantly less than those of the small loss group, $t(20)=3.3$, $p<0.01$.

DISCUSSION

Obesity and the ingestion of palatable high energy foods caused diminished growth and survival of suckling rat pups. There were substantial changes in energy balance and the nutritional status of the dams which may have been associated with the impairment of reproductive performance.

Although pregnancy appeared to proceed normally in the obese dams, and the mean number of offspring produced at parturition was the same as in the lean controls, the mean birth weight of the pups was slightly reduced, and they may have been predisposed by gestational events to a lower rate of growth. These obese dams had increased energy and fat

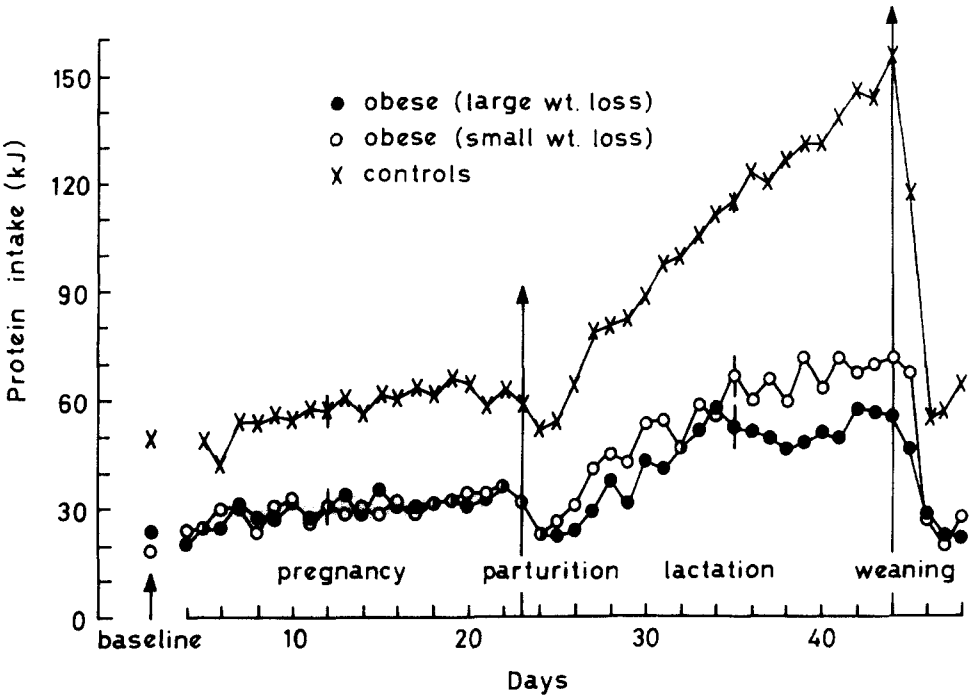

FIG. 6. Mean daily protein intakes of the two obese and the control groups described in Fig. 1.

intakes, but reduced protein intakes in pregnancy. As in the rat, human pregnancy is characterized by a substantial, though variable, fat deposition [20]. There are several indications that there is an optimal level of fat deposition as assessed by numbers of fetal and neonatal deaths. The optimal weight gain varied according to the pre-pregnancy weight of the mother, i.e., the heavier the woman the lower the optimal gain. There are also indications that maternal overeating affects the fetus, in that excessive weight gain led to greater mortality [14].

Despite the importance of weight changes in pregnancy [8], the factors controlling food intake and fat deposition are not well understood. In the rat there is an increase in food intake which results in part from the elevation of plasma progesterone level which is known to lead to hyperphagia [6] and the suppression of estrogen which normally inhibits food intake [32]. These hormonal changes in pregnancy also favor the deposition of fat. It is not clear whether the level of body fat around the time of conception affects food intake and fat deposition in pregnancy. If food intake were to be geared to obtaining optimal levels of body fat at parturition, it should have been lower in the obese than in the control rats. Food intake was, however, higher in the obese rats eating the palatable foods and these rats, particularly those that were very heavy showed significantly higher non-fetal weight gains in pregnancy than the controls. Thus if food intake is normally regulated in pregnancy to provide an optimal level of body fat, it is readily overridden by dietary factors such as palatability and energy density, and this is supported by other studies in rats showing that feeding a high fat diet significantly enhanced fat deposition in rats that were of normal weight at mating [10,30].

The factors which control feeding and body weight in lactation are also not well understood. Lactation is particularly interesting because it is characterized by marked hyperphagia while there is generally a loss of body fat. In this study the rat dams with the highest weight gains in pregnancy showed a greater than average loss of weight during lactation and had a smaller than average food intake, whereas the mothers with the lowest weight gains in pregnancy lost the least weight during lactation and ate more food. Naismith has found that loss of weight during the first three months after birth in lactating women was similarly related to weight gain in pregnancy and energy intake after birth [16,17].

That the loss of weight in the obese lactating rat is due to enhanced mobilization of fat is indicated by the accumulation of fat found in the livers of obese rats during lactation (Agius, Williamson, Rolls, and Rowe, unpublished observations) which may result from the flux of non-esterified fatty acids, mobilized from adipose tissue, exceeding the capacity of the liver to secrete triglycerides formed from the fatty acids. It has been reported that rats of normal weight at mating which gain excess weight and fat during pregnancy through over-consumption of a high fat diet also lose more fat during lactation than chow-fed controls, despite the continued availability of the high fat diet [10,30]. In those studies the rats eating the high fat diet had levels of body fat at the end of three weeks of lactation which were similar to those of non-pregnant chow-fed controls. In the present study the rats which were heaviest after parturition had the greatest weight loss in lactation, and although the body weights of the two obese sub-groups and the controls differed markedly at the beginning of lactation, there was convergence of the body weights so that they were similar at the end of lactation. This may imply that the fat loss in lactation is regulated, but more experimental work is needed to clarify this point. A further point which requires clarification is whether the fat

gain in pregnancy or the total amount of carcass fat has the more important influence on weight loss during lactation. In the present study weight loss was correlated with the non-fetal weight gain in pregnancy, body weight at mating, and body weight after parturition.

In humans there is an indication that weight gain in pregnancy should achieve an optimal level not only for infant survival [14], but also for successful lactation. Naismith [16,17] has reported that women who have low weight gains in pregnancy may fail to lactate adequately. There is also an indication that obesity may affect lactation adversely [7]. Significantly more women greater than 20% above ideal weight have problems with lactation than do women of normal weight at conception. We have found in the rat that obesity was associated with high pup mortality and poor growth. The reasons for this poor lactational performance may be complex and are not yet understood. In another study where pup mortality was very high it was suggested that maternal behavior was abnormal. A high level of pup licking was observed which could have been due to an abnormal response to the taste of the pups, and which could have triggered a high level of cannibalism [33].

Another possible explanation for the poor performance of the obese mothers is that milk production may be abnormal. If rats were fed a high fat diet from 30 days of age, but had the diet withdrawn before mating, histological analysis indicated that these obese animals had mammary glands which were not fully developed. As in the present study pup survival and growth were impaired [31]. Mammary gland metabolism and milk composition are also altered in obese rats eating the cafeteria diet. We ([26] and Agius, Williamson, Rolls, and Rowe, unpublished observations) assessed the metabolic function of the mammary gland by measuring lipogenesis in vivo with ^3H_2O in lactating rats with litters retaining eight pups. Lipogenesis was 86% lower in the obese rats than in the controls. The depressed rate of lipogenesis could have been due to a decreased rate of milk production or to an increased contribution of dietary or adipose tissue lipid to milk lipid. An indication that the obesity and not just the diet may affect mammary gland function comes from the finding that normal weight rats eating the same cafeteria diet from parturition showed a reduction in mammary gland lipogenesis compared to controls [1] but this reduction was 72% less than that of the obese rats. Furthermore, the pups of these mothers showed improved survival and growth over that of pups of the obese mothers. The effects of obesity on total milk output are currently under investigation. The composition of the milk of cafeteria-fed obese rats differs from that of controls. The proportion of fat in the milk of the obese rats was approximately doubled and it had a lower proportion of the characterisitic medium chain fatty acids and a higher proportion of long chain fatty acids. Lactose content was reduced by 32%, but the protein was little affected in the obese group [23]. To clarify further the etiology of the poor lactational performance more work is currently in progress to separate nutritional effects from those metabolic and endocrine changes accompanying obesity.

The hormonal changes in lactation influence both energy intake and metabolism. There may be a hormonal basis for the weight loss, low food intake and poor lactational performance of obese mothers. Prolactin and insulin are implicated in the integration of mammary gland metabolism with that of other tissues (see [34]). For example, prolactin may be responsible for suppressing the activity of lipoprotein lipase in adipose tissue during lactation thus potentially increasing the supply of nutrients for the mammary gland. Insulin is important in the short term regulation of glucose metabolism and lipogenesis in the lactating gland, and it is probable that prolactin opposes the action of insulin in tissues other than mammary gland and thus may act as an integrator of lipid metabolism during lactation. Also, both these hormones may be involved in the control of food intake. Insulin, for example, is elevated in rats with dietary obesity [27] and administration of insulin can increase food intake. Prolactin may stimulate the hyperphagia of lactation [11] although this is a controversial point [4].

The lower food intakes of the obese dams during lactation could be caused by a reduction in the neurogenic influence of suckling on food intake [3] if the pups, perhaps weakened by gestational or other factors associated with obesity, suckle less frequently. Our preliminary observations of suckling times suggest that there is a reduction in total nursing time in obese dams.

The nutritional status of the mothers may also be involved in the poor lactational performance associated with obesity. Although obese rats consume less energy than controls during lactation, total maternal energy intake cannot provide the entire explanation of the poor pup growth since it was found that by day 8 of lactation the pups of the small weight loss mothers were significantly lighter than those of controls yet total food intake of the groups did not differ in that period. Possibly it is an imbalance of nutrient intakes by the obese rats which is more important. In our study the proportion of carbohydrate consumed is unlikely to be the explanation for the poor lactational performance since the large weight loss rats were eating significantly less carbohydrate than the small weight loss group yet the pups were growing at the same rate. However, the proportions of either protein or fat eaten could have affected lactation since these were similar in both groups of obese rats and were significantly different from the intakes of controls. Naismith [15] has reported that the gain in weight of rat pups is related directly to the protein value of the maternal diet. However, protein intake is unlikely to have been the only influence on the performance of the obese mothers because in another study we have found that increasing the protein intake of the obese, cafeteria-fed mothers to that of the controls did not re-establish normal pup growth and survival. The high fat content of the diet could be responsible for the poor lactational performance of the obese groups since high fat consumption affects mammary gland metabolism and milk composition (see [34]). However, several studies have shown that normal weight mothers eating a high fat, protein-supplemented diet are able to maintain the growth of the pups at control levels [5, 13, 21, 28].

The poor lactational performance and negative energy balance of the obese dams is unlikely to be attributable to components of the diet such as toxic flavoring agents or colorants since similar effects were seen with the liquid diet which was simply evaporated milk with pure fat and sugar added. It is possible that the rats failed to consume enough of some vital trace element, but it should be remembered that the nutritionally balanced chow was always available to the rats. It is clear from these studies that when palatable high energy foods are readily available appetite alone may not be an adequate guide to nutrient intake in pregnancy and lactation as has been suggested [12,22].

Thus obesity is associated with alterations in maternal energy balance and poor pup growth and survival in lactation. Many interesting questions arise from this work and

future experiments will determine whether there is an optimal level of body weight and body fat for successful lactation and what mechanisms underlie body weight changes in lactation.

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