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Body fat
dietary composition
puberty

Body Fat at Puberty in Rats: Alteration by Changes in Diet

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Summary

The body composition (percent body fat) at vaginal opening of groups of rats fed different diets was determined. Serial changes in body composition prior to vaginal opening were also measured to assess the temporal relationship between first achievement of the body composition subsequently found at puberty and the actual onset of puberty (vaginal opening). The body weight at vaginal opening and the total carcass fat at vaginal opening were significantly different among the three dietary groups ($F = 43.06$ and 44.60, respectively; $P < 0.001$). In addition, the percent body fat at vaginal opening was also significantly different among the three dietary groups ($F = 30.30$; $P < 0.001$). In one group, the percent body fat was the same at the age of 31 days as it was at vaginal opening (mean age 41 days). In a group where food intake was restricted, the percent body fat prior to and at vaginal opening remained lower than that seen at vaginal opening in a group fed the same diet *ad libitum*. Thus, attainment of a critical level of body weight, total body fat stores, or percent body fat does not trigger puberty in rats.

Speculation

Sexual maturation and body fat accumulation may be parallel but asynchronous processes which reflect completion of an animal's early period of rapid growth.

The nature of the factors controlling the onset of puberty and the relationship between the processes of somatic growth and sexual maturation are areas of importance not only to researchers in several fields, but also to those who deal with clinical disorders of growth and development. The hypothesis that the onset of puberty in animals was more closely related to body size than to age was first suggested by Kennedy and Mitra (12). Frisch and colleagues have extended this concept to humans and suggested that attainment of a critical body weight (2, 6–8) or percent body fat (5, 9) may trigger menarche. To test this theory in animals, the body composition (percent body fat) at vaginal opening of groups of rats fed different diets was determined. Serial changes in body composition prior to vaginal opening were also measured to assess the temporal relationship between first achievement of the body composition subsequently found at puberty and the actual onset of puberty (vaginal opening).

MATERIALS AND METHODS

Female Wistar rats (17) were housed in individual cages after weaning at the age of 21 days. They were exposed to a 14/10 light-dark cycle and given free access to water. No male rats were present. Beginning at the age of 21 days, groups of rats were placed on one of three dietary regimens: standard laboratory rat chow (18) *ad libitum*, a complete synthetic amino acid (AA) diet *ad libitum* (19), or the AA diet in restricted amounts (5–7 g/day). The synthetic AA diet, the composition of which has been published (11), contained 12.5% protein and 15% fat and was very similar to the standard complete synthetic AA diet described by Rogers and Harper (13). Weanling female rats fed the AA diet *ad libitum* displayed normal growth. Laboratory chow contains approximately 5% fat and 23% protein.

After sacrifice by decapitation at or prior to the time of vaginal opening, the animal carcass was weighed and hydrolyzed in 20% KOH. Lipids were extracted with heptane and weighed after solvent evaporation. Total carcass fat was calculated after assuming that all lipid in the heptane extract was fatty acid derived from triglyceride. The percent body fat was calculated as total carcass fat divided by carcass weight. In the group fed the AA diet *ad libitum*, determinations of body weight, total carcass fat, and percent body fat were made at the ages of 21, 24, 28, and 31 days and at vaginal opening (mean age 41 days). Similar determinations were made in the group fed the AA diet in restricted amounts at the ages of 35 and 49 days and at vaginal opening (mean age 100 days) and in the group fed laboratory chow *ad libitum* at vaginal opening (mean age 37 days). In each group, seven or eight animals were used at each time point except for the AA-restricted group at vaginal opening ($n = 5$ for all parameters) and the AA *ad libitum* group at the age of 28 days and at vaginal opening ($n = 4$ for percent body fat and total carcass fat).

Statistical analysis was done by analysis of variance.

RESULTS

The body weight at vaginal opening (Fig. 1, bottom) and the total carcass fat at vaginal opening (Fig. 1, top) were significantly different among the three dietary groups ($F = 43.06$ and 44.60, respectively; $P < 0.001$). In addition, the percent body fat at vaginal opening (Fig. 1, middle) was also significantly different among the three dietary groups ($F = 30.30$, $p < 0.001$). The group fed the AA diet *ad libitum* had the same mean percent body fat at the age of 31 days as it had at vaginal opening (mean age 41 days), despite 100% increases in body weight and total carcass fat during this 10-day interval. Furthermore, the group fed the AA diet in restricted amounts had low percent, body fat through the age of 49 days, and vaginal opening occurred without this group ever having attained the percent body fat present at vaginal opening in the group fed the same diet *ad libitum*.

DISCUSSION

The exact nature of the mechanisms regulating the development of puberty is not known. Frisch and coworkers (2, 6–8) suggested that menarche in humans might be triggered by attainment of a critical body weight. Our studies show that the body weight at vaginal opening in rats differed significantly among groups on three different dietary regimens. This finding confirms previous studies by ourselves (10) and others (3) which showed that dietary alteration could affect the body weight of rodents at puberty. Thus, attainment of a critical body weight does not trigger puberty in rats.

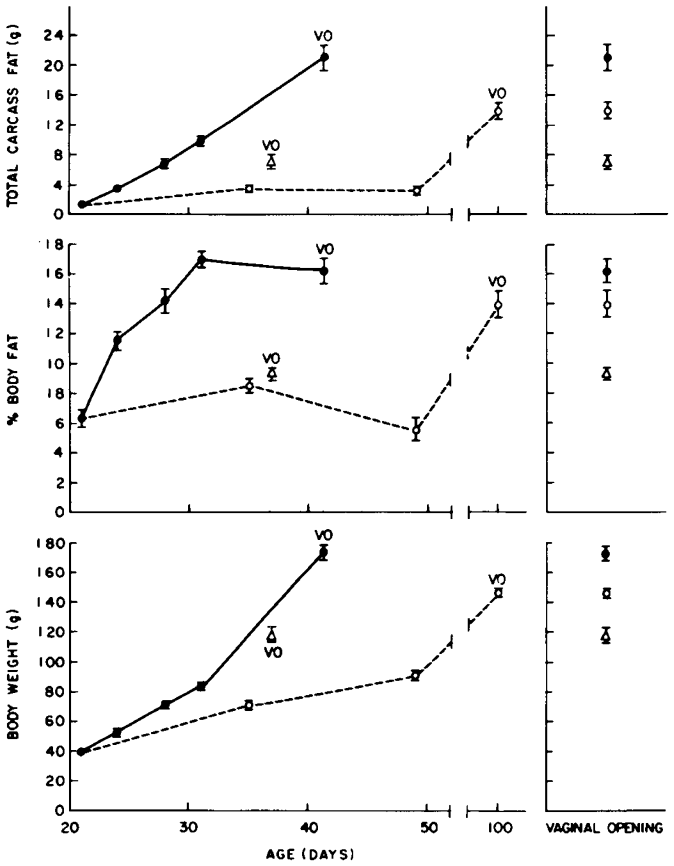

Fig. 1. Body weight (*bottom*), percent body fat (*middle*), and total carcass fat (*top*) in groups of rats fed either laboratory chow *ad libitum* (△), a synthetic AA diet *ad libitum* (●), or the AA diet in restricted amounts (○). Data obtained at the time of vaginal opening (VO) are displayed at the mean age of vaginal opening for each dietary group (*left*) and repeated in a single column for rapid comparison (*right*). The SE for each point is shown by error bars, unless it is smaller than the size of the symbol. At vaginal opening, all three variables (body weight, percent body fat, and total carcass fat) were significantly different among the three dietary groups ($P < 0.001$).

Frisch and colleagues found that total body fat at puberty was similar in groups of rats given diets differing in fat content (4), although they and others (16) showed that total body fat at puberty in rodents increases with increasing age at puberty. In our study, the total carcass fat at puberty was significantly different among groups of rats on three different dietary regimens. Therefore, attainment of a critical level of total body fat stores does not trigger puberty in rats.

Recently, Frisch and coworkers (5, 9) have suggested that attainment of a critical level of percent body fat may trigger menarche in girls. We found that the percent body fat at puberty differed significantly among groups of rats on three different dietary regimens. Moreover, a group of rats fed the AA diet *ad libitum* had the same percent body fat at the age of 31 days as it had at vaginal opening, which occurred a mean of 10 days later. In this case, the first attainment of the body composition subsequently seen at vaginal opening was not closely related temporally to the actual onset of puberty. In addition, the group of rats fed the AA diet in restricted amounts had vaginal opening without ever having attained the percent body fat seen at vaginal opening in the group fed the same diet *ad libitum*. Rats fed a low valine diet *ad libitum* had delayed puberty compared with growth-matched controls (10), despite having a higher percent body fat than control at the ages of 35 and 49 days (data not shown). Previous studies have indicated that percent body fat at puberty in rats showed a significant positive correlation with age at puberty (16) and can be altered by changes in dietary fat (4). Taken together, these findings suggest that attainment of a critical level of percent body fat does not trigger puberty in rats.

Our study was not designed to determine whether achievement of a particular growth milestone will initiate puberty at some critical interval after attainment of this milestone. Moreover, the use of vaginal opening as a marker of puberty may not be ideal, since, like menarche in humans, it is not always associated with ovulation. However, since puberty is a continuous process from a hormonal point of view, selection of any particular event as a marker of puberty is somewhat arbitrary. Our data and that of others cited above supports the idea that the timing of pubertal events does not depend on the attainment of a particular landmark in growth, if one ignores the pattern of somatic and hormonal development prior to arrival at such a landmark.

Although attainment of milestones in pubertal development may not be directly related to achievement of milestone in somatic development or body composition, accumulation of body fat does accompany sexual maturation. All groups we studied, including those subject to severe reductions in caloric intake, were relatively and absolutely fatter at vaginal opening than at the time of weaning. Body fat accumulation during the maturation of the animal may reflect the fact that the entire caloric intake is no longer totally committed to vital developmental processes during the early period of relatively rapid growth. These excess calories may then be deposited as fat. Similarly, establishment of adult sexual function should also be associated with availability of uncommitted excess calories for gestation and lactation. Thus, accumulation of body fat and sexual development may be parallel but asynchronous processes which both reflect the "maturation" of the animal and completion of vital developmental processes during an early period of rapid growth. Such a dependence of sexual maturation on completion of early rapid growth is supported by our previous finding of a significant negative correlation between the timing of pubertal events and the growth rate in rats (10). In fact, since estrogen may affect regulation of food intake and body weight (15), the hormonal changes of sexual maturation may be responsible for some of the changes in body composition in developing animals, rather than vice versa.

Extrapolation of these findings to humans must be done cautiously, since the pattern of growth of rats is qualitatively different from that of humans. However, rapidly growing girls, whose period of early growth is completed sooner, have both earlier menarche (1) and more rapid accumulation of body fat (9, 14) than their slower growing counterparts. Further investigations are needed to determine the nature of the signal conveying the information that early growth is completed and adult development processes may begin.

CONCLUSION

Body weight, total carcass fat, and percent body fat were determined at and prior to vaginal opening in groups of rats on three different dietary regimens. At vaginal opening, all three variables (body weight, total carcass fat, and percent body fat) were significantly different among the three dietary groups ($P < 0.001$). In one group, the percent body fat was the same at the age of 31 days as it was at vaginal opening (mean age 41 days). In a group where food intake was restricted, the percent body fat prior to and at vaginal opening remained lower than that seen at vaginal opening in a group fed the same diet *ad libitum*. Thus, attainment of a critical level of body weight, total body fat stores, or percent body fat does not trigger puberty in rats.

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