In order to further evaluate the significance of this contrast, we used observations made by others on large representative samples of French schoolchildren. The results observed for the A children were compared to those expected on the basis either of their biological parents or of their adoptive parents.

For school failures, comparisons were made with a large and representative cohort of 14,000 children leaving primary school, whose failure rates were known, between grades 1 and 6 (*7*). For the WISC, no national norms are available concerning the various social groups, but for the collective test ECNI we have one of the best standardizations ever made, based on a representative sample of 120,000 schoolchildren aged 6 to 14 (*6*). This test may also be less culture-biased than the WISC: the children of highly skilled professional parents (the top 5 percent) only have a mean score of 111.5 compared with 94.8 for children of unskilled industrial workers.

The results of the comparison have been plotted in Fig. 1. For school failures, the percentages observed for the A and B groups (13 ± 6 and 56 ± 8 percent) are compared to the failure rates expected for the A children on the basis either of their adoptive parents (15 percent) or of their biological parents (55 percent). For the collective test ECNI, the percentages of scores below 95 (17 ± 5 and 49 ± 9 percent) are compared to the rates expected from the social class of the adoptive parents (15 percent) or of the biological parents (51 percent) and to the rates observed for five social groups.

We emphasize the absence of ascertainment bias and the fact that the two groups are biologically equivalent (*14*), so that the contrast observed between them is essentially of environmental origin. The details of our results and the possible sources of bias are being examined (*15*).

The contrast in intellectual status between the A and B children is close to that prevailing in the population at large between children of upper-middle-class parents and children of unskilled workers. Moreover, the failure rates observed for the A children are almost embarrassingly close to those expected solely on the basis of the social class of their adoptive parents. We think that the most economical interpretation of these observations is that there are no important genetic differences between social groups for factors relevant to school failures. An alternative interpretation would be that a certain genetic disadvantage of the A children was exactly compensated for by a special environmental advantage related to their adoptive status. In this case, however, one begins to wonder whether there exists any experimental design by which the hypothesis of a genetic origin for the "educational lag of disadvantaged children" (*1*) could be submitted to scientific test.

Table 1. Comparison of A and B groups. For IQ tests 1 and 2—usually WISC and ECNI (*11*)—percentages of failure were obtained by assuming a normal distribution of scores.

	Percentages	
	A	B
Serious failures		
Special class	2*	25†
Scoring below 85		
Test 1	1	20
Test 2	4	21
All failures		
Special class or repeat	13‡	56§
Scoring below 95		
Test 1	8	52
Test 2	17	49

Test	*N*	IQ	σ
1A	32	110.6	11.3
1B	20	94.5	11.3
2A	31	106.9	12.2
2B	20	95.4	12.9

*(½)/32 (*10*). †11/39 = 28 percent (*13*). ‡4/32. §24/39 = 62 percent (*13*).

MICHEL SCHIFF, MICHEL DUYME
ANNICK DUMARET, JOHN STEWART
STANISLAW TOMKIEWICZ
JOSUE FEINGOLD
Institut National de la Santé et la Recherche Médicale (INSERM), U 69, 1 rue du 11 Novembre, 92120 Montrouge, France

References and Notes

1. A. R. Jensen, *Harv. Educ. Rev.* **39**, 1 (1969).
2. H. J. Eysenck, *The Inequality of Man* (Temple Smith, London, 1973), p. 19.
3. R. J. Herrenstein, *IQ in the Meritocracy* (Little, Brown, Boston,1973), p. 214.
4. M. Skodak and H. M. Skeels, *J. Genet. Psychol.* **75**, 85 (1949); H. M. Skeels, *Child Dev. Monogr.* **31** (3) (1966); S. Scarr and R. A. Weinberg, *Am. Psychol.* **31**, 726 (1976).
5. L. F. Richardson, *Eugen. Rev.* **4**, 391 (1913).
6. Institut National d'Etudes Démographiques, *Travaux et Documents*, **54** (1969). The norms for the collective ECNI (Echelle Collective de Niveau Intellectuel) test are given in table II_1 of volume 64 (1973) of the same series. For the general population, the mean is 99.2, with a standard deviation of 15.
7. Ministère de l'Education, Service Central des Statistiques et Sondages, *Etudes et Documents* **28** (1974); the norms for school failures were obtained from the raw data of tables I_{21-24} and III_{21-25}.
8. Biological parents of the A children: all putative fathers were unskilled workers, and there were no skilled workers among the mothers. Class of adopted children: 20 belonged to the top 5 percent, 10 to the following 10 percent, and 2 to the next 10 percent, in terms of socio-professional status.
9. The 12 cases of B mothers without a B child were distributed as follows: Four mothers could not be followed up because their identity was not in the files, four had numerous other children but had abandoned them all, three had no other child, and one had left France.
10. The A group contained 35 children, including three twin pairs; each member of a twin pair was counted as half of one subject.
11. Usually, score No. 1 came from the Wechsler Intelligence Scale for Children (WISC) and score No. 2 from ECNI. For the collective test, one subject was absent and two were given the Cattell scale No. 2 because they were too old for the ECNI scale. The other exceptions (10 percent of scores) concern children who had been placed in special classes and for whom one or two IQ scores were available in school files.
12. A subjects: 6 in grade 3, 10 in grade 4, 3 in grade 5, and 13 in grade 6 or above.
13. B subjects: 6 in grade 2, 3 in grade 4, 3 in grade 5, and 27 in grade 6 or above. The probability of school failure increases with age; hence a small age correction was made to take account of the fact that, on the average, the B children were slightly older than the A children.
14. Briefly, the possible effect of selective placement of "bright" infants into "good" families is minimal because of the early age at which this placement occurred (mean age, 4 months; maximum age, 6 months). Another small systematic bias could have arisen from the fact that the fathers of the A and B children were different in most cases; however, this could only decrease the effect observed, because the fathers of the A subjects were purposely chosen to be of the lowest possible socio-professional group, whereas some of the fathers of the B children were in a somewhat higher group.
15. M. Schiff, M. Duyme, A. Dumaret, J. Stewart, S. Tomkiewicz, J. Feingold, in preparation.
16. Supported by the Grant Foundation, INSERM, Centre National de la Recherche Scientifique, and the Fondation pour la Recherche Médicale Française. In the initial stage, crucial support was given by R. Cohen, A. Gerbal, J. M. Goux, R. Havighurst, A. Jacquard, C. Meljak, S. Richardson, C. Ropartz, M. Vigier, and R. Zazzo.

22 November 1977; revised 15 March 1978

Role of Adipocyte Geometry in Eating Behavior

According to the lipostatic theory of body weight regulation (*1*), a humoral satiety signal, directly proportional in intensity to organismic adipose mass or some covariant, provides information to the nervous system which alters appetitive behavior. Meal size or frequency, or both, are thereby adjusted to maintain body mass relatively constant over the long term. The nature of this signal and the aspect of adipose mass to which it is sensitive are unknown, but insulin and some correlate of adipocyte size are, respectively, attractive candidates. Since the sensitivity of adipocytes to insulin-sensitive metabolic processes appears to vary inversely as their size, a closed-loop system relating prandial insulin levels to current adipose mass can be envisioned (*2*). A recent study of experimentally joined rats has provided strong evidence for an adipose mass–related humoral satiety factor (*3*).

The data of Faust *et al.* (*4, 5*), which demonstrate both a strong tendency toward constancy in organismic adipose mass and an apparent influence of adipocyte size on eating behavior, support this

0036-8075/78/0630-1504$00.50/0

theory and may be analyzed to suggest the nature of the adipose mass-related variable "reported" to the central nervous system.

On the basis of studies showing reduced food intake in lipectomized compared to nonlipectomized Osborne-Mendel rats at a point when both groups had similar adipocyte volumes but the lipectomized rats had a significantly lower total adipose mass and total adipocyte number, Faust *et al.* suggested that the achievement of a critical adipocyte volume is an important element in the reduction of food intake. However, since adipocyte volume in both groups was the same, it is difficult to see how this explains the divergent eating behavior. Classical lipostatic theory, which would predict reduced food intake in the animal with more adipose tissue, also fails to account for these observations.

Assuming that there is a humoral satiety factor (SF) whose per meal circulating concentration is

$$\frac{\text{mean adipocyte surface area}}{\text{total adipocyte number}}$$

and using adipocyte lipid content as a measure of cell volume (*6*), it can be shown that the Osborne-Mendel rats fed high fat for 9 weeks differ significantly with respect to total weight of fat depots ($t = 3.01$; $P = .006$), total adipocyte number ($t = 3.64$; $P = .001$), and SF ($t = 4.02$; $P = .0004$), while there is no significant difference in adipocyte volume or mean adipocyte surface area (*7*) between the two groups. The nonlipectomized animals have the larger values of total weight of fat depots and total adipocyte number, whereas SF is larger in the lipectomized animals. Thus, although the former two variables are apparently not those regulated by the lipostatic system, the SF values for the two groups differ in an appropriate and significant fashion.

Consistent with recent studies indicating a greater effect of adiopcyte volume than adipocyte number on eating behavior (*8*), the model outlined above predicts that the relative impact on SF of a multiplicative change (X) in adipocyte volume will be $X^{5/3}$ greater than an identical muliplicative change in adipocyte number. It also predicts that developmental increases in adipocyte number secondary to the normal hyperplasia of growth will reduce SF and thereby augment food intake. The relative hyperphagia of infancy and adolescence may then be a product rather than a cause of the adipose hyperplasia characterizing these periods. Likewise, excessive adipose hyperplasia induced by overfeeding during "critical periods" of adipocyte replication would permanently increase the denominator of the SF expression thereby requiring a permanently greater mean adipocyte surface area to return the ratio to "normal." This prediction is consistent with clinical observations indicating an increased incidence of obesity in children overfed as infants.

The experimental tool developed by Faust *et al.* (*4*, *5*), which allows independent manipulation of adipocyte number, size, and total adipose mass, could be combined with the parabiotic system described by Parameswaran *et al.* (*3*) to test this hypothesis.

RUDOLPH L. LEIBEL
Department of Pediatrics, Harvard Medical School and Cambridge Hospital, Cambridge, Massachusetts 02139

References and Notes

1. G. C. Kennedy, *Proc. R. Soc. London Ser. B* **140**, 587 (1953).
2. R. L. Leibel, *J. Theor. Biol.* **66**, 297 (1977).
3. S. V. Parameswaran, A. B. Steffens, G. R. Hervey, L. De Ruiter, *Am. J. Physiol.* **232**, R150 (1977).
4. I. M. Faust, P. R. Johnson, J. Hirsch, *Science* **197**, 391 (1977).
5. ———, *ibid.*, p. 393.
6. Surface area = $3V(V/4.19)^{-1/3}$ where V is adipocyte lipid content.
7. The insulin resistance of large adipocytes may depend on cell volume rather than cell surface area. Substituting V for mean adipocyte surface area (MASA) in the SF expression only increases the differences between animals because when $V_2/V_1 = X$, $\text{MASA}_2/\text{MASA}_1 = X^{2/3}$.
8. J. G. Kral, *Am. J. Physiol.* **231**, 1090 (1976); I. M. Faust, P. R. Johnson, J. Hirsch, *ibid.*, p. 538.
9. I thank A. Walker for help with statistical calculations and J. D. Crawford for reviewing the manuscript. A more extensive treatment of the material presented here will be provided by the author on request.

29 September 1977; revised 18 October 1977

The most straightforward, but not necessarily the best, interpretation of our findings is that when fat cells approach a particular large size they exert a restraining influence on food intake. Various existing models of adipose tissue behavior offer somewhat different explanations. Leibel offers one such alternative interpretation based on his view of the way in which cell size and number interact. This view, which he describes above, predicts that not only will enlargement of adipocytes cause food intake to be restrained, but that a decrease in adipocyte number will do so as well, while an increase in adipocyte number will cause a sufficient enhancement of food intake that adipocyte size will increase. Unfortunately, such predictions do not appear to be correct.

First, decreased adipocyte number does not necessarily cause a decline in food intake. Lipectomized Osborne-Mendel rats eat as much chow and accumulate as much body fat as nonlipectomized control rats. They continue to do so even though their adipocytes become larger than those of their controls and SF becomes even larger than it was at the time of surgery. When they are given a much more palatable diet, lipectomized rats increase food intake just as much as controls, making SF relative to controls increase even further. It appears that lipectomized rats finally begin to eat less than their controls only when their adipocytes approach an extremely large size (*1*).

The prediction that increased adipocyte number will cause an increase in adipocyte size and thus an increase in food intake also does not seem to be supported empirically. When adult rats are fed a very palatable diet for a sufficient period of time, both adipocyte size and number are increased substantially. However, when chow feeding is reinstituted, food intake declines and adipocyte size returns completely to normal, but adipocyte number does not decline at all (*2*). Thus, chow consumption by such rats is depressed even though the depression results in a decrease of SF to a subnormal level.

Finally, it is perhaps incorrect to conclude that by the end of our experiment the mean adipocyte size in lipectomized rats was the same as that in control rats in all depots. As reported, we found that differences between the two groups in retroperitoneal and mesenteric depots with regard to total lipid or mean adipocyte size were quickly obliterated by high-fat feeding. However, as shown in figure 1 of our report (*1*), total lipid in noninguinal, subcutaneous depots of lipectomized rats was still somewhat greater than in controls. Consequently, it is likely that the mean subcutaneous adipocyte size in the lipectomized rats was also still greater than that in the controls.

The important matter of how fat storage status is communicated to the central nervous system continues to concern us, and we appreciate Leibel's efforts to formulate a new unifying hypothesis to guide future work in this area. We hope our few comments will encourage, rather than discourage, further developments of that hypothesis.

IRVING M. FAUST
PATRICIA R. JOHNSON
JULES HIRSCH
Rockefeller University, New York 10021

References and Notes

1. I. M. Faust, P. R. Johnson, J. Hirsch, *Science* **197**, 393 (1977).
2. I. M. Faust, P. R. Johnson, J. S. Stern, J. Hirsch, *Am. J. Physiol.*, in press.

12 April 1978