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THE RELATIONSHIP BETWEEN DIET, PARENT'S FATNESS, AND OBESITY IN CHILDREN AND ADOLESCENTS

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Abstract

In this paper empirical evidence is presented on the determinants of obesity in youth in the U.S., with particular emphasis on isolating the effects of diet and parent's fatness on the obesity outcome. The results show that parent's fatness has statistically important impacts on skinfold growth among children and adolescents. Diets between obese and non-obese youth, however, do not differ substantially. Evidence that youth with "fatter parents" are able to produce more skinfold or adipose tissue from given calorie intakes includes the significant and relatively large parent's fatness (skinfold) effects in the youth skinfold equations, the larger calorie coefficients in the skinfold equation for 10-16 year old youths with "fat" mothers as compared to 10-16 year olds with around average mothers, and the significant and relatively large parent's fatness effects in the youth obesity probability equations. The probability models show that if either of the parents of a 10-16 year old is obese, the probability of the 10-16 year old being obese is .2, holding constant age, race, sex and calorie consumption. If both parents are obese the probability of the 10-16 year old being obese is .4. The data set is the Ten State Nutrition Survey, 1968-1970.

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1.0 Introduction

Obesity in youth is a widely recognized health problem. Since obese children tend to become obese adults, childhood obesity is a link to the high incidences of diabetes mellitus, high blood pressure, heart disease and gall bladder disease found among obese adults. Obesity in children and adolescents may also have more immediate impacts, particularly on mental health because of social rejection and low self-esteem.[1]

Previous research has established that obese parents tend to have obese children and that lean parents tend to have lean children.[2] There is disagreement, however, over the extent to which this clustering results from environmental influences--in particular, common values toward diet and exercise--or from genetic factors. There is substantial evidence in support of both the environmental[3-7] and the genetic explanations[8-10] but few attempts to quantify in some way the separate effects of each on the obesity outcome in children and adolescents.[11]

The purpose of this paper is to present empirical evidence on the determinants of obesity in children and adolescents in the United States with particular emphasis on isolating the effects of diet and the effects of genetic influences that correlate with parent's fatness. The data set utilized in this research is the Ten State Nutrition Survey, 1968-70.

2.0 The Model

The effects of diet and parent's fatness on obesity in children and adolescents are estimated in a multivariate statistical framework that

can be generalized as

$$O = O\ (D, F, A, R, S)$$

$$W = W\ (D, F, A, R, S)$$

$$D = D\ (W, O, I, E, X, S).$$

The model relates obesity (O) and weight growth (W) to diet (D), parent's fatness (F), age (A), race (R), and sex (S). The choice of diet (D) depends on weight (W), obesity status (O), family income (I), mother's education (E), family size (X) and sex (S). Obesity, weight growth and diet are endogenous or mutually determined within the model because causality is hypothesized to run from diet to weight growth and to the obesity outcome for obvious reasons and from weight growth and obesity status to choice of diet because weight is assumed to be highly correlated with children/adolescent demand for calories and nutrients (appetite) and because with weight held constant an obesity condition may alter diet choice (dieting). The rationales for the exogenous variables in the model are straightforward. Children's growth has been shown in a number of studies to differ by age, race, and sex and choice of diet for or by youth may be affected by family income, family size, and by education of the mother.[12]

Triceps skinfold measurements are used as the measure of adipose tissue or fat stores in this study. Skinfold measurements are generally considered more reliable indicators of obesity in children and youth than weight for height measures because there is rather large variation in adipose tissue among children of the same relative weight.[13] Daily calories is the primary diet measure used in the model for reasons more fully explored below.

3.0 The Data and Estimation Techniques

In the Ten State Nutrition Survey (TSNS) socioeconomic, dietary, and clinical data were collected from 24,000 families in ten states of the United States in the 1968-70 period. Selected subgroups, including children less than 36 months of age and individuals 10-16 years, received detailed dietary evaluations. Low income families are over represented in the TSNS sample.[14]

The diet data were collected by the 24 hour recall method. This technique will generally produce unbiased mean intakes for groups because the influences of subjects with unusually high and unusually low 24 hour intakes tend to cancel.[15] However, in an ordinary least squares regression context this random measurement error would bias downward the nutrient intake coefficients in the obesity or weight growth equations. This problem is alleviated in the estimates presented below because simultaneous equation estimation methods are required for the multi-equation model of obesity and diet choice that has been specified. The technique used in this paper is two stage least squares which removes bias due to mutual causality by using predicted values of the right-hand side endogenous variables in the structural equations. A by-product of this procedure is that random measurement error in endogenous variables, such as in the 24 hour recall diet variables will also be eliminated by the use of predicted values.

The empirical results are for children less than 36 months of age and for children/adolescents 10-16 years of age. These age groupings include all children and adolescents for which dietary data were obtained in TSNS. They correspond to the two most interesting periods

of children's growth in terms of potential impact on adult obesity. The first period is important because young children that acquire an excessive number of adipose cells may be destined to be obese adults. Some researchers argue that by the end of the first three years of life the development of the body's fat cells are complete and their number difficult or impossible to reduce in later life.[16] The 10 to 16 year age period is important because rapid growth occurs and the body contour changes significantly, approaching its eventual adult configuration. Obesity developed during this age period then may also be carried through adult life.

4.0 Empirical Results

4.1 Summary Statistics

In Tables 1-2 summary statistics are presented for the diet, anthropometric, and socioeconomic variables that are relevant to a model of obesity and diet choice such as specified in Section 2. The 10-16 years age grouping has been stratified into those in the upper 10 percent of the triceps skinfold distribution for their sex, those in the lower 25 percent, and those in the group between the 25th and 75th percentiles of the sex specific triceps skinfold distributions. The distributions are sex specific because of the differences in the levels of subcutaneous fat stores carried by males and females of similar ages; e.g., the mean triceps skinfold for males and for females 10-16 years old in TSNS is 11.7 and 15.4. The sample stratification are designed to enable comparisons between children and youth that are obese (upper 10 percent), of around average fatness (25 percent to 75 percent), and lean (lower 25 percent). Similar

stratifications following the same logic have been made for children in the younger age group.

The most striking result in Tables 1-2 is the similarity of diets across the subsamples within each age grouping. For 10-16 year olds, in fact, there is actually a slight inverse relationship between triceps skinfold measurement and caloric intake. Mean calorie consumption is 2,347 per day in the obese group (upper 10 percent of triceps skinfold distribution, mean triceps skinfold 28.1 mm) and 2,523 per day in the lean group (lower 25 percent of triceps skinfold distribution, mean triceps skinfold 7.2 mm). There is a slight positive relationship between triceps skinfold measurement and caloric intake in the 0 to 36 month age group but daily calories only increases from about 1,350 to 1,390 between the lean and the obese groups, while triceps skinfold increases from 7 to 15 mm.

These summary statistics imply then that obesity is not associated with above average calorie consumption. It could be argued that the average calorie consumption of obese 10-16 year olds is not representative of their lifetime diet patterns because dieting may begin for many in adolescence and when younger these individuals on average had diets higher in calories than their peers and became obese as a result. This argument loses force, however, when considering the results for the younger age groups which should be hardly affected by dieting and which show near identical diets across the subsamples. In any case, the regression results in the following section will enable a more formal examination of the effects of diet on obesity and will allow the presence of obesity to influence the choice of diet.

The summary statistics in Tables 1-2 do show generally large and statistically significant differences in parent's fatness between obese and non-obese children and adolescents. For 10-16 year olds, for example, mother's triceps skinfold for obese youth is 30.3 mm, for average youth is 22.5 mm, and for lean youth is 20.1 mm. The same figures for father's triceps skinfold are 18.2 mm, 13.4 mm, and 11.2 mm. This association between parent's fatness and children's fatness is also evident in the younger age group.

The summary statistics then generally support the view that the diets of obese and non-obese children and adolescents do not differ greatly and, therefore, the obese are "more efficient producers" of adipose tissue from given levels of nutrient intakes.[17] This productive efficiency may well be inherited for the parents of obese children and adolescents carry significantly more adipose tissue than the parents of the non-obese.

4.2 Regression Results

In Table 3 regression results are presented for a model of triceps skinfold growth and choice of calories that follows the general structure presented in Section 2.[18] The results are for the 10-16 year and 0-36 month age groups of TSNS. Calories is the only diet measure examined at this stage of the research. By using calories as the diet measure we are emphasizing "quantity" and skirting issues of "quality" or diet composition. Models estimated with multiple diet indicators--for example, calories and protein--make it difficult to reach conclusions about the impact of diet on triceps skinfold growth because of multicolinearity.[19]

The first two regressions in Table 3 present results for triceps skinfold growth for 10-16 year olds. The parent's skinfold measures are highly significant and rather substantial in "elasticity" terms. The elasticity of triceps skinfold with respect to mother's tricep skinfold evaluated at the means using the regression parameter from equation 1 is .40. The elasticity figures from equation 2 for mother's tricep skinfold and for father's tricep skinfold are .34 and .22. To restate these results from equation 2, a youth with each parent having 10 percent larger triceps skinfold than the parent's of another youth, holding constant calorie consumption, race, age, and sex, would have a 5 to 6 percent larger skinfold. Note that because calories are held constant, these parent's fatness effects are not capturing the impact of a common household diet high in calories that results in similar skinfold growth within the family; an argument used by some to explain why fatness clusters in families.

The calorie coefficients in Table 3 for 10-16 year olds show a positive association between calories and skinfold growth although the coefficients are not statistically significant. This positive association is surprising in light of the summary statistics which showed the mean calorie intakes of the obese group to be slightly less than those of the non-obese groups. There is evidence in the calorie equation in Table 4 that the obese attempt to restrict their calorie consumption and thus their reported daily calorie intake may understate their daily average over the longer term. Theoretically, this reverse causality could be important and highlights the necessity of allowing causality to run from obesity to calorie intake as well as vice versa. It also brings out a potential weakness in the methodology of sample mean comparisons between the diets of the obese and non-obese such as

was performed in Section 4.1. To summarize this argument calorie intakes for a 24 hour period for an obese sample are more likely to understate longer term daily averages than for the non-obese because a higher proportion of the obese will be on calorie restricting diets and because most calorie restricting diets fail or are only temporary deviations from longer term average daily consumption. This could mean that, longer term, and in conflict with 24 hour recall data, the obese consume more than the non-obese and the additional calorie consumption results in their additional skinfold. This argument then does not rely on the notion that the obese utilize calories more efficiently and offers an explanation of why the obese have been reported in some studies to eat about the same amount, or even less than the non-obese.

Empirically, however, dieting effects do not appear to be particularly strong. The obese dummy variable in the first calorie equation in Table 4, which takes a value of one for 10-16 year olds in the upper 10 percent of the sex specific triceps skinfold distributions and a value of zero otherwise, has a coefficient of about -95. Since weight is also in the calorie equation, the result indicates that the obese in their 24 hour recall period consumed 95 calories less than the non-obese of their same weight. To the extent weight is a proxy for appetite the difference of 95 would represent the effects of dieting. This effect is rather weak given average diets of around 2,400 calories and given the low statistical significance of the dummy coefficient. It is difficult to argue, then, that the longer term average daily calorie consumption of the obese is much higher than would be revealed in a 24 hour recall period and that excessive calorie consumption determines obesity.

The diets of the obese appear to be very similar to those of the non-obese.

Differences in parent's fatness between the obese and non-obese are large and statistically significant, as previously noted. The parents of obese children and adolescents have triceps skinfold about one-third larger than parents of the non-obese. The regression results indicate that if children and adolescents in the obese category had parents with the same average fatness as the non-obese, the triceps skinfold of typical male and female youth in the obese category would be about 3 mm less, still leaving them with triceps skinfold measurements more than twice as great as for the typical 10-16 year old.

In the third regression in Table 3, triceps skinfold results are presented for children 0 to 36 months in TSNS.[20] The results indicate that the elasticities of triceps skinfold with respect to calories and mother's tricep skinfold evaluated at the means are .7 and .2. The standard error of the calorie coefficient, however, is relatively large. Even if the calorie coefficient is assumed accurate and even if the effect of father's fatness is assumed equal to that estimated for the mother, the results do not explain much of the differences in tricep skinfold thickness between obese and non-obese children 0-36 months in TSNS. In this statistical decomposition there is almost no calorie effect because of the near identical diets of obese and non-obese children (see Table 2) and the differences in parent's fatness between the obese group and the around average group only account for about 10 percent of the difference in triceps skinfold, assuming equal effects for both parents.

An alternative way to consider the extent to which children and youth with "fatter" parents may be more efficient producers of fat tissue is to stratify the samples based on parent's fatness. Children and adolescents with "fatter" parents should have larger calorie coefficients in the triceps skinfold equations if they are more efficient producers of fat. Two stage least squares estimates for 10-16 year olds in TSNS whose mothers are in the top 20 percent of the skinfold distribution for all mothers of 10-16 year olds[21] and for 10 to 16 year olds whose mothers are between the 25th and 75th percentiles of this distribution yields calorie coefficients of .0037 and .0026, respectively.[22] The mean triceps skinfold of the youth in the fat mothers group is 17.4 mm and mean daily calories is 2,416. For the youth with mothers of around average stature mean triceps skinfold is 13.6 mm and daily calories is 2,466. The more efficient production of fat hypothesis can explain a good portion of the triceps difference of 3.8 mm (17.4-13.6) between the two groups. The difference in calorie coefficients of .001 implies the youths with "fat mothers" get 2.4 mm more skinfold from the 2,400 calorie diets common to both groups.[23]

The first regression of Table 3 can be used to produce a similar result. Mean triceps skinfold thicknesses for the group of fat mothers and for around average mothers are 38 and 23, respectively. Using the coefficient for mother's triceps skinfold of .24 from the regression, triceps skinfold for the youth in the "fat mothers" group should be 3.6 (15 x .24) greater than for the youth with near average mothers. This approximates the observed difference. In this case at

least parent's fatness effects are sufficient to explain differences in skinfold thickness between groups. The same methodology did not have much success in explaining differences in skinfold thickness between obese and non-obese youth because the difference in parent's fatness was much smaller and the difference in skinfold thickness to be explained was much larger.

Parent's fatness effects on the obesity outcome can also be investigated within the context of a probability model. In Table 5 results are presented for an obesity outcome equation for 10-16 year olds in TSNS where the dependent variable is the obesity dummy previously defined. Mothers and fathers have been similarly classified from their sex specific skinfold distributions and the parent's fatness variables are now dummies taking on a value of one for those in the upper ten percentiles. The parent's fatness coefficients are each about .2 and highly significant.[24] They can be interpreted as probabilities; that is if either of the parents of a 10-16 year old is obese, the probability of the 10 to 16 year old being obese is .2, holding constant age, race, sex, and calorie consumption. If both parents are obese the probability of the 10-16 year old being obese is .4. To say the same thing, there is a probability of .6 that a 10-16 year old with obese parents will not be obese. For children 0 to 3 years the probability of being obese given an obese mother is also .2 (see Table 5).[25]

Given these results it is not surprising to find that the distribution of obese parents across the obese, around average, and slim subsamples of 10-16 year olds of Table 1 is not overwhelmingly skewed

toward the obese youth group. Thirty-two percent of obese parents are in the obese youth category, 42 percent are in the around average youth category, and 10 percent are in the slim youth category. For children up to 36 months, 22 percent of obese mothers are in the obese children category, 44 percent are in the around average children category, and 24 percent are in the slim children category.

5.0 Summary and Conclusions

To summarize, the statistical results of the preceding section have indicated that parent's fatness has statistically important impacts on skinfold growth among children and adolescents. Diets between obese and non-obese youth, however, do not differ substantially. Evidence that youth with "fatter parents" are able to produce more skinfold or adipose tissue from given calorie intakes includes the significant and relatively large parent's fatness (skinfold) effects in the youth skinfold equations, the larger calorie coefficients in the skinfold equation for 10-16 year old youths with "fat" mothers as compared to 10-16 year olds with around average mothers, and the significant and relatively large parent's fatness effects in the youth obesity probability equations. Although the statistical results could explain a large portion of the difference in skinfold growth between youth with "fat" mothers and youth with around average mothers, the models were not nearly as successful in explaining differences in skinfold between obese and non-obese youth. The results show that calories, parent's fatness, age and race differences between obese and non-obese youth explain less than one-half of the skinfold growth

differential, with nearly all of this explanatory power coming from parent's stature effects.[26]

The regression results for choice of calories for or by children and adolescents show no significant family income, mother's education, or family size effects for children less than three years, while there is stronger evidence for such effects among 10-16 year olds, although the implied elasticities are small.[27] This same pattern holds when the demand for nutrients is considered, such as protein. It is interesting that socioeconomic effects on diet may be more important for older children than for preschool children, despite the presence of school lunch programs.

Footnotes

[1]For reviews of the health effects of obesity in youth, see Coates, T.J. and Thoresen, C.E., "Treating Obesity in Children and Adolescents: A Review." American Journal of Public Health, 68: 103, 1979 and Mallick, M. Joan, "Health Hazards of Obesity and Weight Control in Children: A Review of the Literature." American Journal of Public Health, 73: 78, 1983.

[2]See, for example, Garn, S.M. and Clark, D.G. "Trends in Fatness and the Origins of Obesity." Pediatrics, Vol. 57, No. 4, April 1976.

[3]Jacoby, A.; Altman, D.; Cook, J.; Holland, W.; and Elliott, A. "Influence of Some Social and Environmental Factors on the Nutrient Intake and Nutritional Status of School Children." Br. J. Prov. Soc. Med., Vol. 29, 116-120, 1975.

[4]Kohns, M.B.; Eklund, D.; and O'Neal, R. "The Association of Obesity with Socioeconomic Factors in Missouri." Am. J. Clin. Nutr., Vol. 32, 2120-2128, 1979.

[5]Rimm, I.J. and Rimm, A.A. "Association Between Socioeconomic Status and Obesity in 59,556 Women." Preventive Med., Vol. 3, 543-572, 1974.

[6]Shenker, I.R.; Risichelli, V.; and Lange, J. "Weight Differences between Foster Infants and Overweight and Nonoverweight Foster Mothers, Brief Clinical Observations." J. Pediat., Vol. 84, 715-719, 1974.

[7]Stunkard. A. "Obesity and Social Environment." Ann. N.Y. Acad. Sci., Vol. 300, 298, 1977.

[8]Biron, P.; Mongeau, J.; and Bertrand, D. "Familial Resemblance of Body Weight and Weight/Height in 374 Homes with Adopted Children." Journal of Pediatrics, 91: 555, 1977.

[9]Bennett, W., and Gurin, J. "Do Diets Really Work?." Science 82, 3:42, 1982.

[10]Stunkard, A. Obesity. W.B. Saunders Company, Philadelphia, 1980.

[11]Hartz, A.; Giefer, E.; and Rimm, A.A. "Relative Importance of the Effect of Family Environment and Heredity on Obesity." Ann. Hu. Genet., Vol. 41, No. 2, 1 5-193, October 1977.

[12]Two papers that investigate differences in children's growth by diet, age, race, and sex and that consider the extent to which family income and education of the mother are obstacles to the provision of adequate diets in poor American families are:

Chernichovsky, D. and Coate, D. "The Choice of Diet for Young Children and Its Relation to Children's Growth," Journal of Human Resources, XV, Spring 1980.

Chernichovsky, D. and Coate, D. "An Economic Analysis of the Diet, Growth, and Health of Young Children in the United States." Annual Series of Research in Human Capital and Development, edited by Ismail Sirageldin and David Salkever. Greenwich, Connecticut: JAI Press (forthcoming).

[13]Dean, M.S. et al. "Where Do the Heaviest Children Come From? A Prospective Study of White Children from Birth to 5 Years of Age." Pediatrics, 63: 1, 1979.

[14]TSNS is described in detail in Health Services and Mental Health Administration DHEW. Ten State Nutrition Survey, 1968-1970. DHEW (HSM) 72-8130-8134, 1972. The sample design of TSNS was expected to produce "essentially self weighted estimates" (Health Services and Mental Health Administration, I-13) for low income groups based on characteristics of the population from the 1960 Census. However, because the survey was carried out in the 1968 to 1970 period and because this research uses sub-samples from TSNS, standard errors of estimates reported in this paper, which are not adjusted for sample design, may understate the "true" standard errors.

[15]The fact that obese respondents are more likely to be on diets than non-obese respondents presents special problems in the interpretation of 24 hour recall data that are discussed below.

[16]See Hirsh, J. "Can We Modify the Number of Adipose Cells?" Post-graduate Medicine, 51 (May 1972), and Coates and Thoresen, op. cit., p. 147.

[17]It is possible that differences in exercise or activity levels rather than in ability to produce adipose tissue better explain obesity in children and adolescents, although data from Cycle I of the Health and Nutrition Examination Survey, 1971-1974 (HANES) do not support this view. In HANES, two questions were asked of 12 to 16 year olds concerning activity levels. These related to the amount of activity in a usual day aside from recreation and to the amount of exercise during recreation.

There are virtually no differences in the responses to these questions across samples stratified on triceps skinfold measurements using the criteria of Table 1.

[18]The weight equation of the model specified in Section 2 is necessary to complete the system of three endogenous variables but the results for the weight equation itself are not of vital interest and are not presented in this paper.

[19]The correlation between calories and protein intake is .85 among 10-16 year olds.

[20]Regression results are not presented for children 0 to 36 months with the father's tricep skinfold as a right-hand side variable because of the large number of missing observations for this variable.

[21]A similar condition on father's stature was not imposed to preserve observations, a shortage of which would have developed for a jointly conditioned fat parent's group because skinfold measurements are available for only one-half as many fathers as mothers.

[22]Race, age, and sex were held constant as in previous triceps skinfold specifications. The first stage instruments included these exogenous variables and family income, mother's education, and family size.

[23]The calorie coefficients are not significantly different from zero (t values about 1.0 for each) nor are they significantly different from each other. Thus, this analysis stretches the limits of statistical inference. Furthermore, the same methodology applied to the 0 to 3 years of age group yielded a slightly smaller coefficient for the children in the obese mother's group than for children in the around average mother's group.

[24]The equations in Table 5 are estimated by using two stage least squares. The first stage instruments are age, race, sex, family income, family size, and mother's education; that is the equations can be considered part of a general model of obesity and diet choice. With a dummy endogenous variable in the system two stage least squares yields consistent estimates although they may not be as efficient as estimates derived from more sophisticated methods. For a complete discussion of estimation procedures for a dummy endogenous variable in a simultaneous system, see J. Heckman, "Dummy Endogenous Variables in a Simultaneous Equation System." Econometrica, July 1978.

[25]To preserve observations, father's stature is again allowed to vary, that is, it is not included in the regression.

[26]Race, age, and sex effects on triceps skinfold have not been discussed to this point. For 10-16 year olds, the race variable in Table 3 indicates blacks may have slightly less skinfold than whites, other things the same, while females have 5 mm more skinfold than males. For 0-3 year olds the race effect is evident, but there is no sex effect. Age effects are not strong for either age group.

[27]The elasticities at the means for calories with respect to family income and mother's education are .004 and .14.

TABLE 1

Summary Statistics, TSNS, 10-16 Years of Age

Variable	Top 10% by Triceps Skinfold[a]		25% to 75% by Triceps Skinfold[a]		Lower 25% by Triceps Skinfold[a]	
	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation
Daily calories	2,347	1,110	2,461	1,064	2,523	1,133
Daily protein (gm)	95.4	46.8	94.8	48.5	96.0	50.0
Daily vitamin C (mg)	77.2	76.6	71.2	74.3	73.2	78.4
Daily iron (mg)	12.7	6.8	13.2	7.4	13.7	8.6
Daily calcium (mg)	1,039	687	1,123	680	1,153	798
Daily vitamin A (IU)	4,497	5,899	4,434	5,615	4,705	5,736
Daily thiamine (mg)	1.2	.7	1.3	.8	1.4	.9
Daily riboflavin (mg)	2.0	1.2	2.2	1.9	2.4	2.7
Daily niacin (mg)	16.8	9.2	17.8	12.9	18.7	16.7
Weight (kg)	62.1	14.9	44.0	10.9	41.4	10.9
Weight (kg)/height (mm)	.039	.008	.029	.005	.027	.005
Triceps skinfold (mm)	28.1	5.9	12.0	3.2	7.2	1.9
Mother's weight/height	.046	.011	.041	.009	.041	.008
Father's weight/height	.046	.007	.044	.007	.043	.006
Mother's triceps skinfold (mm)	30.3	10.7	22.5	8.8	20.1	7.9
Father's triceps skinfold (mm)	18.2	8.5	13.4	6.9	11.2	6.1

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TABLE 1 (concluded)

Variable	Top 10% by Triceps Skinfold[a]		25% to 75% by Triceps Skinfold[a]		Lower 25% by Triceps Skinfold[a]	
	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation
Age (months)	159.8	20.4	154.1	20.9	154.0	20.9
Sex (1 = female)	.51	.50	.53	.50	.51	.50
Mother's education (years)	9.91	3.6	9.87	3.4	9.76	3.3
Family income	7,616	3,996	8,060	4,560	7,822	4,318
Family size	5.93	2.15	6.4	2.4	6.8	2.5
Race (1 = black)	.28	.45	.24	.43	.30	.46
N[b]	300		1,200		700	

[a]Sample stratifications are children and adolescents 10-16 years in the top 10 percent of the triceps skinfold distribution for their sex, between the 25th and 75th percentiles of the triceps skinfold distribution for their sex, and in the lower 25 percent of the triceps skinfold distribution for their sex.

[b]Summay statistics for father's characteristics are based on observations totaling about one-half of the listed N.

TABLE 2

Summary Statistics, TSNS, 0-36 Months of Age

Variable	Top 10% by Triceps Skinfold[a]		25% to 75% by Triceps Skinfold[a]		Lower 25% by Triceps Skinfold[a]	
	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation
Daily calories	1,388	632	1,367	575	1,354	695
Daily protein (gm)	56.9	26.5	55.1	24.7	56.0	33.0
Daily vitamin C (mg)	56.3	52.3	56.4	65.8	54.7	55.0
Daily iron (mg)	11.1	11.2	10.7	12.0	10.1	10.7
Daily calcium (mg)	1,095	515	1,049	551	1,062	604
Daily vitamin A (IU)	4,428	6,522	4,087	3,726	4,150	3,822
Daily thiamine (mg)	1.0	.64	1.0	.7	1.0	.9
Daily riboflavin (mg)	1.9	1.4	1.8	.8	1.8	.9
Daily niacin (mg)	9.7	6.8	9.3	6.1	8.8	7.1
Weight (kg)	12.0	3.0	11.1	4.2	10.6	4.4
Weight (kg)/height (mm)	.015	.002	.014	.005	.013	.004
Triceps skinfold (mm)	14.5	4.0	9.1	1.3	6.5	1.2
Mother's weight/height	.040	.008	.039	.008	.040	.008
Father's weight/height	.044	.010	.042	.007	.043	.008
Mother's triceps skinfold (mm)	22.9	9.1	20.0	8.6	19.2	8.2
Father's triceps skinfold (mm)	14.4	8.6	12.7	7.3	12.9	7.9

(continued on next page)

TABLE 2 (concluded)

Variable	Top 10% by Triceps Skinfold[a]		25% to 75% by Triceps Skinfold[a]		Lower 25% by Triceps Skinfold[a]	
	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation
Age (months)	18.2	8.8	19.6	9.8	18.5	10.0
Sex (1 = female)	.49	.50	.54	.50	.45	.50
Mother's education (years)	10.9	3.2	10.7	3.0	10.5	3.0
Family income	6,693	3,797	6,971	5,975	6,149	3,496
Family size	5.0	2.0	5.1	2.1	5.1	2.0
Race (1 = black)	.19	.40	.18	.38	.31	.46
N[b]	100		400		250	

[a]Sample stratifications are for children 0 to 36 months in the top 10 percent of the triceps skinfold distribution for their sex, between the 25th and 75th percentiles of the triceps skinfold distribution for their sex, and in the lower 25 percent of the triceps skinfold distribution for their sex.

[b]Summary statistics for father's characteristics are based on observations totaling about one-half of the listed N.

TABLE 3

Structural Equation Estimates for Skinfold Growth for Children and Adolescents, TSNS, Two Stage Least Squares

Independent Variables	Dependent Variables		
	Triceps Skinfold 10-16 Years	Triceps Skinfold 10-16 Years	Triceps Skinfold 0-3 Years
Calories	.0026	.0023	.005
	(1.8)	(1.3)	(.9)
Race	-.42	-.98	-1.4
	(-1.0)	(-1.4)	(-1.8)
Age	.016	.02	-.10
	(1.0)	(1.0)	(-.8)
Sex	5.02	4.95	.20
	(6.3)	(4.7)	(.4)
Mother's triceps skinfold	.24	.20	.08
	(13.5)	(7.4)	(2.8)
Father's triceps skinfold		.22	
		(6.2)	
Intercept	-3.27	-4.97	3.79
	(-1.4)	(-1.7)	(.8)
N	1,823	829	488

Indicates predicted value, t statistics in parentheses.

TABLE 4

Structural Equation Estimates for Calories for Children and Adolescents, TSNS, Two Stage Least Squares

Independent Variable	Dependent Variables: Calories 10-16 Years	Calories 0-3 Years
$\hat{\text{Weight}}$	22.8	106.2
	(5.2)	(9.2)
$\hat{\text{Obese}}$ dummy	-95.5	
	(-.3)	
Sex	-543	50.0
	(-7.8)	(.9)
Family income	.0013	-.44
	(1.6)	(-.8)
Mother's education	33.5	9.4
	(2.5)	(.8)
Family size	-.63	-12.1
	(-.1)	(-.8)
Intercept	1,172	155
	(3.8)	(.7)
N	1,837	488

$\hat{}$ Indicates predicted value, t statistics in parentheses.

TABLE 5

Structural Equation Estimates for Obesity Probability Functions for Children and Adolescents, TSNS, Two Stage Least Squares

Independent Variables	Dependent Variables		
	Obese Dummy 10-16 Years	Obese Dummy 10-16 Years	Obese Dummy 0-3 Years
Calories	.00005	.00003	0.00062
	(1.9)	(.9)	(1.4)
Race	.009	.005	-0.089
	(.5)	(.2)	(-1.1)
Age	.0006	.001	-0.017
	(1.3)	(1.8)	(-1.5)
Mother obesity status	.24	.23	0.24
	(10.4)	(6.6)	(2.2)
Father obesity status		.19	
		(5.5)	
Intercept	-.14	-.19	-0.39
	(-2.2)	(-2.1)	(-1.0)
N	1,833	829	493

^ Indicates predicted value, t statistics in parentheses.