

CHILDHOOD OBESITY

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## INTRODUCTION

Obesity is a common nutritional disorder in the United States; approximately 30% of our adult population is obese (1). Adult obesity is associated with increased risk of development of cardiovascular and renal diseases, hypertension, diabetes, gall bladder disease, gout and arthritis as well as increased risks in surgery and pregnancy (2). In determining the etiology of adult obesity, considerable attention is being focused on obesity which has its onset during infancy and early childhood. There is concern that early childhood obesity, if not successfully treated, may lead to a lifetime of obesity. Obesity in childhood also is associated with increased health risks such as hypertension, hyperlipidemia, and abnormalities of glucose tolerance (3). Obese infants have been found to be more prone to respiratory infections and other illnesses (4). Both socially and medically, the obese child is at a disadvantage when compared with peers of normal weight. The purpose of this paper is to discuss the nature of childhood obesity, its possible etiology, its tendency to progress from infantile to juvenile to adult forms, and intervention and treatment for childhood obesity.

## ASSESSMENT OF OBESITY

Obesity is defined as the presence of excessive body fat. Assessment of obesity involves measurement of body fat and the determination of the level at which it is excessive (5). The two methods of measurement most easily used for children in clinical settings are body weight and skinfold thickness.

### Body Weight

Body weight of a child is easily determined and therefore widely used to assess obesity. Growth charts such as those published by Tanner and Whitehouse (6) (fig. 1) and by the National Center for Health Statistics (7) are used as standards of comparison. Those charts have been derived from data obtained through measurement of large numbers of children at various ages. Age of the child is plotted along the abscissa and weight along the ordinate. A system of percentiles is used to plot the growth curves so that measurements taken at one particular age rank the child in comparison to other children of the same sex and age. Measurements plotted at several ages allow one to visualize the child's pattern of growth and to determine whether it is progressing within approximately the same percentile, as would be expected (8). Separate charts are constructed for children and infants of both sexes. While height and weight are basic measurements which are useful in defining normal growth patterns in children, these measurements are limited in the determination of actual body components such as protein, fat or inorganic constituents of the skeleton (9). Therefore, body weight alone is not a reliable indicator of the amount of fat in the body or of obesity in children.

### Skinfold Thickness

Skinfold thickness measurements can be made relatively easily and accurately with the use of calipers. Sites such as the triceps, subscapular and midaxillary have been used. These measurements are used to estimate total body fatness through measurement of the subcutaneous layer of fat (9). Standards for triceps skinfold measurements have been published by Seltzer et al. (10) and by Tanner and Whitehouse (11)

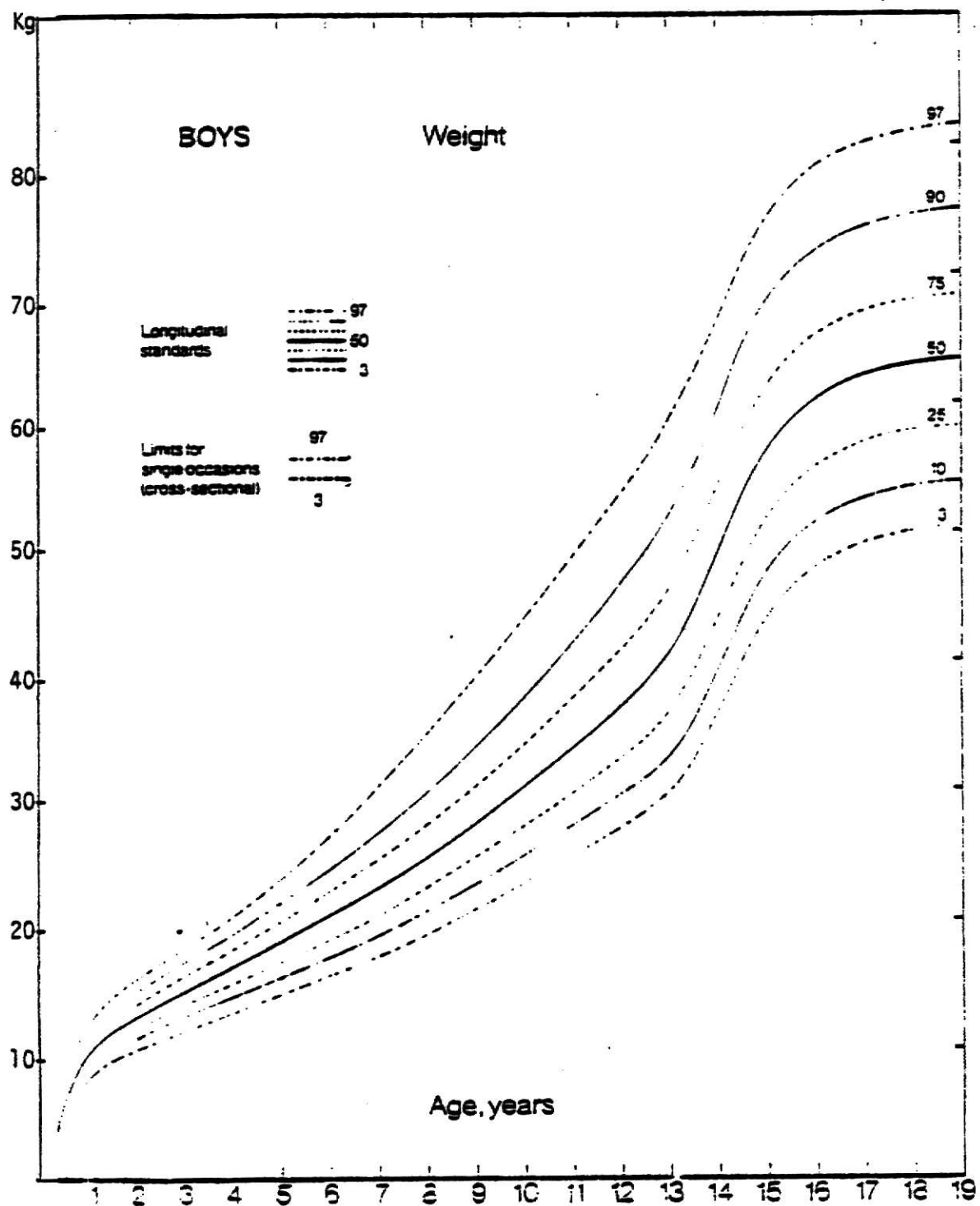


Fig 1- Longitudinal standards for weight attained at given age, boys. The shaded areas represent the 97th and 3rd centile limits of cross-sectionally derived standards (6).

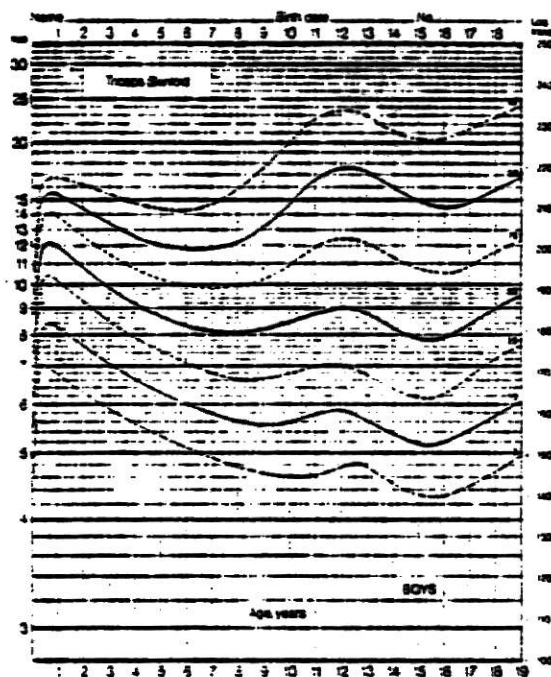
(fig. 2). Skinfold measurements were made in both the Ten-State Nutrition Survey (12) and the National Health Examination in the United States (9). Skinfold thickness measurements are considered to be the best available method for clinical assessment of obesity in children (5, 13).

## ETIOLOGY

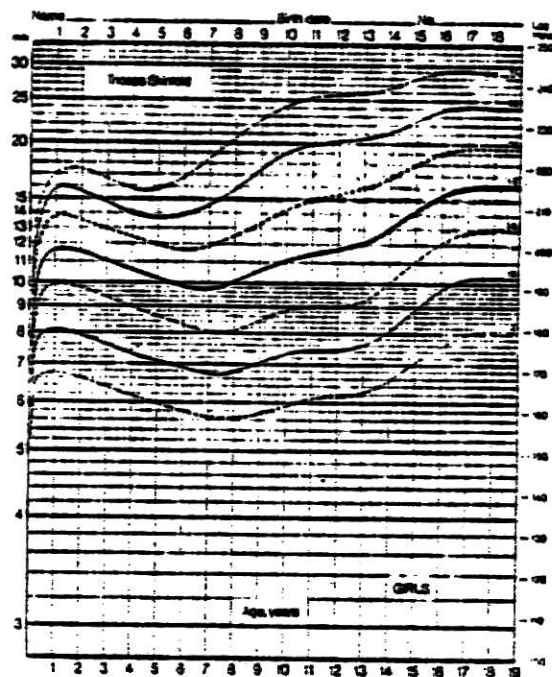
Etiology is an important aspect of childhood obesity, especially in regard to prevention of the disorder. The child is a product of both genes and environment; it is difficult to assess the importance of each factor separately. Is it possible that a child may have such a strong genetic tendency towards obesity that even in a "favorable" environment, excessive weight gain may occur? On the other hand, are infant feeding practices as well as eating habits and activity levels practiced within a family the most influential factors which determine whether a child will be obese?

### Genetics

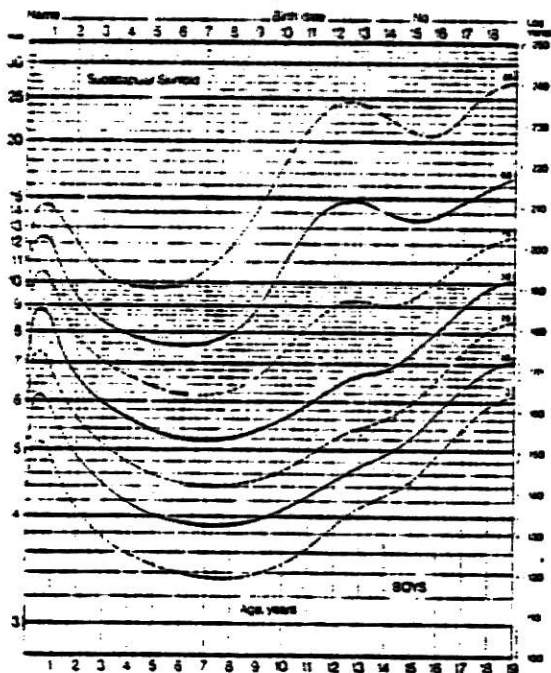
In animals, there is much evidence for genetic control of fatness or leanness as seen in selection for those carcass traits in pigs, sheep and poultry (14). Genetic control of level of fatness in human beings is not easily proven due to differences in environmental factors. Results of the Ten-State Nutrition Survey of 1968-1970 showed that fatness follows family lines (12). Triceps skinfold measurements were taken on 2,961 pairs of spouses, 20,554 parent-child pairs and 29,545 sibling pairs among the black and white subjects. Lean ~~fat~~fold values were defined as below the 15th percentile for age and sex, medium as from the 15th to the 85th percentile and obese above the 85th percentile. The level of fatness of the child was found to rise progressively with the level of



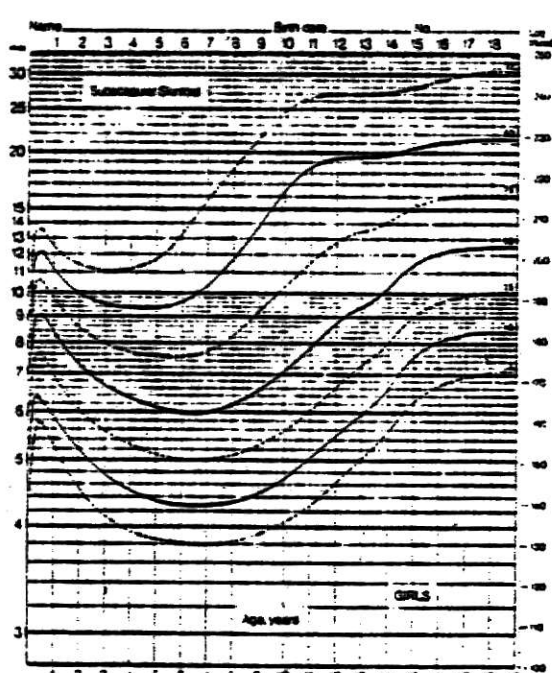
—Triceps skinfold standard, boys.



—Triceps skinfold standard, girls.



—Subscapular skinfold standard, boys.



—Subscapular skinfold standard, girls.

Fig 2- Skinfold thickness standards for boys and girls (11).

fatness of the parents. The children with two lean parents were the leanest and the children with two obese parents were the fattest while those with one obese parent, either their father or mother, were fatter than those with two lean parents. The fact that the sex of the obese parent made no difference would seem to support a genetic influence since the mother generally would be expected to have a greater environmental influence as the primary person involved in shopping for food and meal preparation.

Increased fatness of the children of two obese parents was found at all ages and tended to increase with age. By age 17, average triceps fatfold values indicated that children of two obese parents were three times as fat as children of two lean parents. A similarity in occurrence of obesity among siblings also was found. If one child in a family was obese, there was a 40% chance that a sibling would be obese and if there were three children in the family, there was an 80% chance that at least one of the siblings would be obese. These findings seem to indicate genetic influences on obesity. There also was considerable similarity in fatness of the two parents, with a correlation of 0.3 for parents, compared with 0.25 for parent-child and 0.40 for siblings. This finding seemed to indicate either homogamy, or more likely, common attitudes towards eating and exercise. Therefore, fatness does appear to run in families but genetics may not be the major determinant.

In studying 265 mothers and their babies, Whitelaw (15) found that maternal obesity was associated with increased subcutaneous fat in the newborn. Skinfold thicknesses of the infants were obtained at four sites on both sides of the body: biceps, triceps, subscapular and suprailiac, within 48 hours of birth. Maternal left triceps skinfolds were measured.

At every site, the babies of obese mothers had larger skinfolds than the infants of normal mothers. The babies of normal mothers were also fatter than those of thin mothers.

Brook and his coworkers (16) measured triceps and subscapular skinfold thicknesses of 222 pairs of like-sex twins (78 monozygotic and 144 dizygotic) aged 3 to 15 years, to determine the contribution of genetic and environmental influence on skinfold thickness in children.

Heritability, defined as the proportion of total variation in a population due to genetic causes, was calculated from the correlation coefficients of skinfold thicknesses. Heritability of skinfold thickness was high for all children over age 10, whereas environmental factors were found to be more important in younger children.

To help resolve the nature versus nurture argument in childhood obesity, Poskitt and Cole (17) reviewed the feeding practices and weight of 203 5-year-old children that they had studied previously as infants. Measurements made during infancy and at age 5 included height and weight and estimations of a 24-hour food intake. Height, weight and 24-hour food intake of the mother also were obtained. The child's weight was compared to his expected weight which was calculated as follows:  $\text{actual weight of child} / 50\text{th percentile weight at that age when the child's height was on the 50th percentile of reference standards} \times 100$ . Children and mothers were classified as underweight if their weight was less than 90% of the expected or desirable, normal weight if it was 90-110% and overweight if it was over 110%. Although there was not a significant correlation between the weights of the mothers and their infants, there was a very significant correlation between weights of the mothers and their 5-year-old children. Overweight 5-year-olds were more likely to



have overweight mothers than were those children who were not overweight. This was not due to a higher caloric intake at the time of evaluation; the children of overweight mothers had a mean 24-hour energy intake almost exactly the same as the children of underweight mothers. Therefore, the conclusion was that the genetic aspect was more important than the dietary environment in determining the children's weight at 5 years.

### Environmental Influences

The extent of environmental influences on the development of obesity in children has not been established. However such factors as infant feeding practices, common family dietary and activity patterns and general parental attitudes have been examined in regard to their roles in childhood obesity.

Infant Feeding Practices Culturally, a fat baby has been regarded as a healthy baby for many years (18). Plump infants have been portrayed on infant food containers and in advertisements. Overfeeding in infancy may occur for many reasons such as maternal concerns about giving the baby enough food, inability of the mother to interpret signs of hunger and satiety on the baby's part, concern about food waste, mistakes in formula preparation or use of food as a pacifier (19). Another problem over the years has been disagreement among experts as to the best way to feed a baby. As infantile obesity has become a concern, infant feeding practices have been examined in regard to their contribution to overfeeding. Bottle feeding, which is chosen more often than breast feeding in the United States (8), and early introduction of solid foods are the practices which have drawn the most attention, with some concern for the frequency of feedings.



In a study of 261 normal, full-term infants, Taitz (20) found that at six weeks of age, the 240 artificially fed infants weighted substantially more than was expected on the basis of the Tanner charts. Only 16 (6.7%) of the artificially fed infants were above the 90th percentile at birth but 93 (38.8%) of them were over this mark at six weeks. Of the 21 breast-fed infants, 1 (4.8%) was over the 90th percentile at birth and 4 (19.1%) were over this mark at six weeks. A dietary history taken on 40 of the artificially fed infants indicated that all of them were receiving non-milk supplements which, for most, had been introduced during the first week of life. The mean caloric intake was 135 kcal/kg of body weight per day, which is above the recommended 115 kcal/kg/day.

Shukla and his coworkers (21) studied the feeding patterns and nutrient intake of 300 infants up to one year of age. They found that 50 infants (16.7%) were obese, 83 (27.7%) were overweight and 167 (55.6%) were of normal weight. Overweight was considered to be between 110 and 120% of expected weight using Tanner's graphs and obesity was classified as 120 to 140% of expected weight. Mean triceps skinfold measurements were 11.7 mm for the obese group, 10.7 mm for the overweight group and 9.3 mm for the normal group. There was a low incidence of breast feeding; 84 mothers had begun breast feeding but by 12 weeks of age, only 19 infants were receiving breast milk and all of these also were receiving solid food. Solids were introduced to breast-fed babies as early as four weeks. Solids were offered to some bottle-fed babies as early as their first week and initially the total consumption of milk was unchanged even with the addition of solids, boosting the daily caloric intake from an average of 590 calories for the exclusively bottle-fed infants to 775 calories for those receiving solids in addition to bottle feeding. The

daily caloric intake was highest for those infants aged 3 months and under; 136 kcal/kg for males and 149 kcal/kg for females, well over the recommended 115 kcal/kg/day. The incidence of obesity increased from the first age group of 3 months and younger to the 3 to 6 month old age group and declined thereafter to one year of age.

Hall (22) suggested that infants have an appetite-control mechanism which operates in response to changes in the composition of breast milk. He found that milk samples from nursing mothers contained 4 to 5 times as much lipid and 1.5 times as much protein at the end of the feeding as in the beginning. This occurred at all stages of lactation, ranging from 3 to 35 weeks postpartum. These changes in composition occurred in both breasts, so Hall hypothesized that after the infant finishes milk from one breast which is rich in fat and protein, that the change to the more watery initial milk from the other breast may satisfy his thirst. Hall suggested that a change in taste or texture of the milk may be the reason for termination of feeding or willingness to accept the other breast. If this were the case, bottle feeding would not supply the necessary changes in taste or texture as cues to the infant to terminate the feeding.

Ounsted and Sleight (23) observed weight gain and energy intake of 103 male and 88 female infants at birth and again at the age of 2 months. They classified 45 of them as small-for-date (S.F.D. -- birth weights 2 standard deviations or more below the mean for their length of gestation) and 38 as large-for-date (L.F.D. -- birth weights 2 standard deviations or more above the mean). The S.F.D. infants consumed considerably more milk per kg body weight and had larger weight gains per day than did the L.F.D. Thirty-five percent of the S.F.D. infants were

being breast fed shortly after birth compared with 32% of the L.F.D. infants. By 2 months, only 9% of the S.F.D. infants continued to be breast fed compared with 21% of the L.F.D. infants. This difference was significant. The S.F.D. infants who were breast fed had the largest weight gains.

Oakley (24) measured triceps and subscapular skinfold thicknesses of 315 infants within 24 hours after birth and at six weeks of age to determine differences in subcutaneous fat between breast- and formula-fed infants. At six weeks of age, despite similar weights, the breast-fed group had a greater increase in triceps and subscapular skinfold measurements than the formula-fed group. In a third group of infants which was bottle and cereal fed, males showed an intermediate increase in subcutaneous fat while females showed a greater increase than even breast-fed infants.

Dubois et al. (25) observed 4 to 9 month-old obese and normal infants for differences in feeding practices such as breast or bottle feeding, age at which solids were introduced as well as maternal response to infant cues of hunger and satiety. Forty-seven infants were chosen whose weights were above the 90th percentile of weight for age according to the charts of Tanner and Whitehouse. Those infants were matched in terms of family characteristics, age and sex with a group of 42 control infants with weights between the 25th and 75th percentiles. Mothers completed a 3-day food record for their infants and were subsequently interviewed regarding the food record as well as past feeding practices and concepts and attitudes about infant feeding. Dubois et al. found no significant differences between the two groups in regard to incidence of breast feeding, which was moderately high in both groups.

In those infants no longer being breast-fed, the obese group had a slightly higher average energy intake than the control group but it was not statistically significant. On a per kilogram body weight basis however, the obese group consumed less energy than the control group. Neither group had caloric intakes above the recommended 115 kcal/kg/day. Age of introduction of solids was a little later for the obese group of infants for most foods; strained desserts was the one food item in which the difference achieved statistical significance. Meats were added to the obese infants' diets slightly earlier than to the diets of control group. There were no significant differences found between the two groups of mothers in the frequencies with which they used cues external to the baby in preference to the baby's signals indicating hunger or satiety. Nor were there differences in frequencies with which mothers used food to stop the baby's crying or attempted to feed the infant more or less than the baby wanted.

Huenemann (26) studied 448 six month old infants and found no significant differences in incidence of breast feeding or in age of introducing solid food between the 10% most obese and the 10% least obese infants. Obesity index scores were calculated by a formula using a combination of three variables: weight gain since birth, waist circumference and suprailiac skinfold. Caloric intake was a little higher in the 10% most obese group than in the 10% least obese group but it was significantly different for only the infants with the highest and lowest 5% of obesity index scores. There was no significant difference found in frequency of feeding between the two groups.

DeSwiet et al. (27) observed 758 infants at 6 weeks and 6 months of age and found that there were no significant differences between the

weights of the breast- and bottle-fed infants, whether or not the infants were fed solids. At 6 weeks, the weights of the 251 babies who had been only bottle fed were related to the volume of feed given.

A concern that has been voiced in regard to bottle feeding has been that of over concentration of the formula (18). This could occur accidentally in mixing or could be due to lack of understanding on the mother's part as to how to properly prepare the formula or the belief that "more is better." Not only the caloric density, but also the renal solute load of the feed is increased. This has been found to increase the baby's thirst which may be quenched with another bottle, compounding the problem.

One concern regarding early introduction of solids as it relates to infantile obesity has been the higher caloric density of solid foods. A comparison of the caloric density of human milk and some solid foods showed that meats, fruits, desserts and egg yolks had higher caloric density while juices, creamed and plain vegetables, soups and dinners, as well as most formulas, had lower caloric density than human milk (28).

Ravelli et al. (29) examined prenatal and postnatal levels of nutrition during the Dutch famine of 1944-45 in relation to incidence of obesity in 307,700 male offspring at age 19. Caloric levels of rations had varied from a low of 580 kcal/day to 1800 kcal/day in 1944-45. In the group of men who had been exposed to the famine both during the third trimester in utero and the first 3 to 5 months of life, the obesity rate was significantly lower than in the men who had not been exposed to famine. Postnatal famine exposure alone did not result in differences in the incidence of obesity. The incidence of obesity was significantly higher in the group of men who had been exposed to famine

entirely prenatally and mainly in the first two trimesters. It has been postulated that this is the time in which the differentiation of the hypothalamic regulatory center takes place and that the caloric level of the diet during this time could determine its future function in regulation of appetite and growth.

Common Family Dietary Practices As shown by data from the Ten-State Nutrition Survey, fatness tends to follow family lines (12). It becomes difficult then to separate the genetic contribution from a common family environment. In order to more clearly separate these factors, Garn et al. (30) studied 7,230 parent-child pairs, 7,726 biological and 504 adoptive, for resemblances in height, weight and triceps skinfold. Biological parents and their children were found to have correlations of 0.35 for stature, 0.26 for weight and 0.21 for triceps fatfold. Adoptive parent-child correlations were 0.29 for stature, 0.16 for weight and 0.19 for triceps fatfold. Though the correlations were lower for the adoptive parents and their children, they were not close to zero as would be expected for unrelated parents and children if genetics was the only factor in determination of body size. These findings indicate that genetics does play a part in determination of height and weight but that living together and eating together also brings about similarities, especially in the level of fatness.

Hawk and Brook (31) recorded height, weight and skinfold measurements for 330 families of parents and their children, aged 2 to 15 years, to determine amount of family resemblance in these measurements. Two hundred and fifty-six (78%) of the families were remeasured after offspring had reached at least age 20. Adult height was found to be largely genetically determined but skinfold thicknesses showed very

little resemblance between offspring and their parents or their siblings. The parents' skinfold measurements were not taken the first time and by the second time, most of the parents and offspring were no longer sharing a common environment. The results of this study suggest little genetic contribution to body fatness but the possibility that a common family environment could be a factor in determination of body fatness.

Shenker et al. (32) studied weight gain of 66 foster infants after observing that those placed in homes of overweight foster mothers tended to be heavier than those placed in homes of foster mothers who were not overweight. Foster mothers were divided into two groups, one of overweight and one of normal weight women. The 66 infants entered foster care at an average age of 3.5 weeks. Twenty-one boys and 17 girls were in homes of overweight foster mothers and 17 boys and 11 girls were placed in homes of normal weight foster mothers. Weights of infants were noted up to one year of age. Mean weights of both boys and girls with overweight foster mothers were higher than those with foster mothers of normal weight. The same trend was noted in obesity ratios, defined as weight in ounces divided by length in inches squared. Weights of all infants, however, were within normal range. Conclusions were that the overweight foster mothers tended to feed their infants more than the normal weight foster mothers fed their foster infants.

Common Activity Levels It is likely that living together as a family not only results in similar eating habits but also in similar patterns of activity and exercise. Activity is difficult to measure. Also, in less active obese children, it is difficult to determine whether they are less active because of their obesity or were less active prior to the development of obesity.



Wilkinson et al. (33) matched 10 obese boys and 10 obese girls at age 12 with a similar number of normal weight children in order to study energy intake and measured activity. The obese children were above the 90th percentile of weight for height and the control subjects had weights between the 25th and 75th percentiles. Dietary records were kept by the children's mothers and by the children themselves between meals for a weekend. To measure activity, the children wore a pedometer on a belt on their waist during waking hours for a 24-hour period on a school day. There was no significant difference between the energy intake or measured activity of the obese group and the control group, although the obese boys tended to be somewhat less active than their controls. It was concluded that the obese children had reached an equilibrium and were at the time maintaining their weight.

Griffiths and Payne (34) measured energy intake and expenditure of 4- and 5-year-old children of obese (Group O) and non-obese (Group N) parents. The 8 children in Group O had one parent who at some time in their adult life had been 20% above desirable weight for height. The 12 children in Group N had parents who never had been obese. Height, weight and skinfold measurements indicated that the two groups did not differ in body size. Daily energy expenditure was estimated from the integrated pulse rate for the day, which was based on the relationship between oxygen consumption and pulse rate. Initially, these two measurements were made on the child while lying down and then while he was riding a stationary tricycle. Then for 4 to 7 days, the child's integrated heart rate was measured on a SAMI (Socially Acceptable Monitoring Instrument). Bomb calorimetry was used to measure the energy content of duplicate portions of everything the child ate for 5 to 7



days. The estimates of energy intake and expenditure, including expenditure at rest, were found to be significantly lower in Group O than in Group N. There was a larger difference between expenditure at rest and the total expenditure in Group N than in Group O, which may indicate a smaller amount of voluntary physical activity in Group O. Whether these differences were due to genetic or environmental factors or both was unclear.

General Characteristics of Parents Attitudes of parents towards food, eating and infant feeding may influence the development of obesity in children. Parental expectations that a child finish everything on the plate or that an infant finish a jar of baby food or the formula in a bottle encourage a child to eat past the point of satiety (5). Eating also may be regarded as a means to relieve tension or as a way of showing affection. Some foods also may be used as rewards. Due to advertisement and social pressure, food and eating have come to have significance far beyond a nutritional value (5).

Huenemann (26) noted in her study of 448 infants that an obese infant's mother was frequently fat herself and not knowledgeable about good nutrition. She did not spend as much time cooking or enjoy cooking as much as mothers of the lean infants. She was less likely to withhold dessert if her child did not finish everything on the plate. She also was more likely to be dissatisfied with her child's eating and wish that he or she were thinner. Huenemann concluded that this mother was possibly less sure of herself and less accepting of her child's weight.

## ADIPOSE TISSUE CELLULARITY

Onset of obesity during infancy or childhood has been associated with an increase in the number of adipose cells. Therefore there may be critical periods in which overfeeding leads to an abnormal increase in number of adipocytes which remain throughout the life of the person.

### Infants

Hager et al. (35) observed normal development of adipose tissue cellularity of 16 non-obese infants at regular intervals from birth to 18 months of age. Total body fat was calculated from whole-body potassium measurements and samples of subcutaneous tissue were taken by needle aspiration. Weight and recumbent height were measured and all infants were found to be within normal limits for age and sex. Body fat increased from about 0.7 to 2.8 kg from birth to one year. Another 0.5 kg of body fat was accumulated by 18 months. No sex differences were found in body fat nor fat cell size or number. From 1 to 12 months there was only an increase in fat cell size, not in number. From 12 to 18 months, only fat cell number increased, not cell size. From other studies, fat cell size was noted to be the same at 18 months as in 8-year-olds and 22-year-olds, whereas the cell number increased with age.

### Children

Brook (36) examined 52 obese children and 64 control children to determine whether or not there is a sensitive period in childhood for adipose cell number increase. All children were weighed and measured for height and skinfold thickness at four sites (biceps, triceps, subscapular and suprailiac) on the left side. Total body fat was

calculated from skinfold measurements. The control children were undergoing elective surgery and had a sample of subcutaneous adipose tissue taken at that time. Adipose tissue was obtained by aspiration from subcutaneous tissue in the obese children. The obese children who had gained weight excessively in the first year were found to have a greater number of adipose cells than either those children who became obese after their first year or the controls. There was no difference in the total body fat of the children with early or late onset of obesity. Brook found that the number of adipose cells increased up to puberty in the control population and that these values were similar to those found in adults. There were no differences in number of adipose cells between males and females, which indicated differences must be in cell size. Brook also found that those children who were obese in the first year of life had an increase in linear growth and advanced skeletal maturation as well as an increased number of adipose cells. He suggested that the basic complement of adipose cells is established during the period from 30 weeks gestation through the first 9 to 12 months of life.

Hager et al. (37) also observed adipose tissue cellularity in 18 grossly obese girls before and after dietary treatment as compared with 17 non-obese control girls. Past height and weight data were obtained to determine age of onset of obesity. Current weight and height measurements were taken as well as skinfold measurements at 4 sites (biceps, triceps, subscapular and suprailiac). Samples of subcutaneous tissue were taken by needle aspiration and mean fat cell size determined. Lean body mass was determined by whole body potassium counts from which body fat was estimated. Fat cell number also was approximated. Five girls had become obese before 2 years of age and 13 after age 2. Lean

body mass was greater in the obese group of girls and was the greatest in the cases of early onset of obesity. There was a positive correlation between amount of body fat and fat cell size in both groups. Fat cell size was largest in those with early onset of obesity. There was also a positive correlation between amount of body fat and fat cell number in both groups but this was not related to the age of onset of obesity. After the obese girls had reduced,  $43\% \pm 5\%$  of their body weight as a group, fat cell size decreased. Fat cell number, however, increased in this group while it remained unchanged in the control group. Those girls who were least successful in reducing had the highest increase in cell number.

#### Adult

Hirsch and Knittle (38) studied obese adults, some of whom had reduced, with different ages of onset of obesity and compared them with subjects of normal weight as to the cellularity of their adipose tissue. Adipose tissue was obtained by needle aspiration and cell number and cell size were estimated. Total adipose cell number for the whole body was then estimated. Adipose cells in the obese subjects were 40% larger in size and 190% greater in number than those of normal weight subjects. There was a good correlation between the degree of obesity and the degree of adipose cell hyperplasia (number of cells). The degree of fatness, however, could not be correlated with cell size. Age of onset of obesity was determined through interviewing the subject and by viewing family photographs. In general, the group with the highest number of cells contained individuals with the earliest onset of obesity. In the subjects who had reduced (each had lost approximately 50 kg of weight), the cell number stayed elevated while cell size was greatly reduced.

Jung et al. (39) studied 83 women and 24 men aged 16 to 80 years whose weights ranged from 13% underweight to 225% overweight. Obesity was defined as 20% or more over the midpoint of the weight range for a medium frame size according to the tables of the Metropolitan Life Insurance Company. In 46 cases, samples of subcutaneous fat were removed during elective surgery, in 44 of these patients intraabdominal fat samples also were obtained. In the remaining cases, subcutaneous tissue was aspirated from the epigastric, suprailiac and femoral areas. Total body fat was derived from skinfold measurements; these values were almost identical to those obtained from whole body potassium measurements. Lipid content of the adipocytes increased with more severe obesity until the patient was 80% overweight. There was considerable difference from site to site, depending on the location of maximum deposition of fat. Women of equivalent relative weight to men, obese or not, had larger adipose cells. There was a positive correlation between the number of adipocytes and the degree of obesity. The correlation between the number of fat cells and the age of onset of obesity was not significant. There was however, a positive correlation between the degree and duration of obesity. Omental fat cells, though larger in obese patients than in normal weight patients, were much smaller than subcutaneous fat cells, from which most estimates of fat cell number are derived. Fat cell number then, often may be underestimated. The number of fat cells in persons in the control group varied so much that all of the obese patients had fat cell numbers that fell within the 95% confidence limits for normal values. For these reasons, as well as the lack of relationship between number of fat cells and age of onset of

obesity, the suggestion that childhood obesity is hyperplastic in nature is unwarranted.

#### Adults and Children

Brook et al. (40) compared 54 obese children and 25 obese adults with early and late onset of obesity to determine differences in size and number of adipose cells. Skinfold thickness was used to determine obesity in adults whereas both weight and skinfold thickness measurements were used for the children. Age of onset of obesity in children was determined from the history given by the parents and objective weight records. The child was considered to have been obese the first year of life if weight at one year exceeded the 97th percentile on the Tanner charts and if percentile lines had been crossed the first year. History alone had to be used for determination of age of onset of obesity in adults. Children's ages ranged from 0.3 years to 17.7 years. Adults ranged from 22 to 71 years of age. Samples of adipose tissue were obtained and cell size and number were estimated. Cell size of individuals with early onset of obesity was smaller than that of individuals with late onset of obesity, but all obese subjects had adipose cells considerably larger than the controls. The number of adipose cells was greater in children with early onset of obesity but children who had become obese after age one had a normal number. The same pattern held for adults.

#### PROGRESSION OF INFANTILE OBESITY INTO CHILDHOOD AND ADULTHOOD

The treatment or prevention of infantile obesity may prevent childhood obesity and, in turn, much of adult obesity. If obesity which has

begun in the early months of life results in a lifetime of obesity, prevention is very important. The question of whether a fat baby becomes a fat child who in turn becomes a fat adult then becomes crucial.

Eid (41) compared 138 children who had gained weight above the 90th percentile on Tanner charts in their first six months of life (R group) with 33 who were around the 50th percentile (M group) and 53 whose weights fell below the 10th percentile (S group). These children were remeasured at the ages of 6, 7, or 8 years. Standing height, weight and triceps and subscapular skinfold measurements were taken. The children's mothers also were measured and height and weight data for the fathers were obtained from their wives. A child was considered obese if weight was more than 20% over the expected weight for height, age and sex and overweight if 10% over expected weight. Thirteen of the 138 children in the R group were obese compared with 125 who were not obese. In groups M and S only 2 had become obese as children while 84 had not. This difference between group R and groups M and S was significant. The number of children who were overweight in the R group also was significantly higher when compared with the S group. Even excessive weight gain at 6 weeks of age led to a somewhat higher incidence of obesity in childhood. Weight gain in infancy was a better predictor of obesity than parents' weight. Thickness of skinfold measurements of the children was significantly greater in the R group than the S group. Infants with low birth weights tended to gain weight most rapidly and were therefore at a greater risk of subsequent obesity.

Charney et al. (42) obtained heights and weights of 366 subjects between 20 and 30 years of age who had weights and recumbent heights recorded at a physician's office at the ages of six weeks, 3 months and 6



months. Stuart's growth charts were used as a reference and infants who had weights above the 90th percentile at one of the three ages were placed in one group. Those who had weights between the 25th and 75th percentiles all three times were in another group and ones who had fallen below the 10th percentile at least once were in a third group. Data from the National Health Survey was used as a reference for adult weight and 10% above the median weight was classified as overweight and 20% or more as obese. Those infants who had weights above the 90th percentile at least once were found to be 2.6 times as likely to be overweight or obese adults as those of average or light weight. Underweight as an infant also correlated with adult underweight. The risk of adult overweight rose progressively from the 75th percentile. Birth weight did not correlate significantly with adult weight. Crossing percentiles did not increase the risk of adult obesity unless it was over the 75th or, particularly, 90th percentile. Rather, the absolute weight attained was the important factor.

Fisch et al. (43) studied 1,786 subjects whose heights and weights were measured at birth and at either age 4 or 7 years or both. A ratio index was computed for each by dividing weight in kg by height in cm. Subjects in the highest and lowest fifth percentiles were designated as "very obese" and "very slender," respectively. Generally, infants with a very high ratio index tended towards normal weight but a somewhat high percentage of them (77%) were still above the 70th percentile at age 7. Only 26% of the very slender infants were above the 70th percentile at age 7. There was a very high correlation between obesity at 4 and 7 years of age. Only a few obese 4 year olds had slimmed down by age 7 and most obese 7 year olds had been obese 4 year olds.



Poskitt and Cole (44) took height, weight and skinfold measurements of 203 children, who also had been measured as infants 4 to 5 years earlier, to determine if fat infants stay fat. Percentage expected weight was calculated by two methods. The first method was percentage weight for height ratio and was calculated as follows:  $(\text{actual weight} / \text{actual height}) \times (50\text{th centile height for age} / 50\text{th centile weight for age}) \times 100\%$ . The second method was percentage weight at age when height is on 50th centile and was calculated as follows:  $(\text{actual weight} / 50\text{th centile weight at age when child's height is on 50th centile}) \times 100\%$ . Tanner's charts were used as a standard and values over 120% indicated obesity, 110 to 120% overweight, 90 to 110% normal and under 90% underweight. In infancy, the children were significantly heavier but not longer than expected. In childhood they were both heavier and longer than expected. In infancy, both triceps and subscapular skinfolds were thicker than reference standards but in childhood, only the subscapular skinfold was thicker. By the first index, 35 infants were found to be obese and 6 of these were obese and 10 overweight in childhood. By the other index, only 28 infants were classified as obese and 3 were obese and 7 overweight as children. Some of the obese infants returned to normal weight in childhood, but many tended to remain at least overweight. The authors concluded that there was some relationship between infantile and childhood obesity but there were many factors which determine later weight.

Hawk and Brook (45) conducted a longitudinal study to determine the relevance of body fatness in childhood to body fatness in adulthood as assessed by skinfold thickness measurements. Children of 330 families had been measured when they were aged 2 to 15 years. A total of 318 male

and 303 female subjects were traced and remeasured as adults, ages 17 to 30 years. Skinfold measurements were taken at triceps, subscapular, suprailiac and biceps sites. Individual predictability of adult skinfold thicknesses from childhood values varied widely from age to age and site to site. In larger groups, a moderate degree of prediction could be achieved but this still varied from group to group. No differences were found in this predictability when childhood values over the 75th percentile were analyzed separately.

### TREATMENT

Treatment of obesity in children should lead to a higher rate of success than in adults since dietary and activity patterns have not been practiced for as many years as they have been by adults. High dropout rates, however, exist in programs for both children and adults (46). Recently, efforts to treat the problem of increasing numbers of obese children have been focused on both prevention programs and treatment of existing obesity. Which type of program might be more effective depends on whether genetic or environmental factors are most important in determination of obesity. Rate of success of treatment also is dependent on whether early onset obesity results in higher numbers of adipose cells which make weight reduction more difficult. If this is the case, focus on prevention is more desirable.

#### Prevention and Education

According to the Ten-State Nutrition Survey, fatness runs along family lines (12). The Ad Hoc Committee to review the Ten-State Nutrition Survey recommended that identification, therapy and control of obesity be done on a family-line basis. Approaches that might be

considered in prevention of obesity in infants of obese parents have been suggested by Jelliffe and Jelliffe (18). Those include monitoring of growth, dietary guidance and increased opportunities for exercise. Weight and height should be recorded monthly; triceps skinfold thickness and arm circumference also should be measured periodically. Dietary guidance should focus on prevention of overfeeding the infant and should include information on breast and bottle feeding, introduction of solids and caloric value of various solid foods. Exercise should be encouraged by insuring that the baby's movement is not restricted and that he or she is not overheated by unnecessary clothing or blankets. Toys that encourage movement also should be provided.

Pisacano et al. (47) compared incidence of overweight in a group of 80 infants fed a special diet with that in a group of 50 infants fed an average American infant diet in 1964. In the test group, formula was discontinued at 3 months and skimmed milk was used. Excessive salt and sugar were not used and the number of eggs was limited. In the comparison group, whole milk was used after the formula was discontinued at 3 months and commercial baby foods, an egg a day, custards and puddings were given. Weight and height measurements were taken at 3 years of age. The diet group had an incidence of overweight of 25% at the age of 3 months when the study began compared to an incidence of 34.1% in the comparison group. At the age of 3 years, only 1.28% of the diet group were overweight compared to 25.5% of the comparison group.

Smicklas-Wright and D'Augelli (48) began a Preschool Eating Patterns (PEP) Program in 1977 at Pennsylvania State University for families with preschool children to confront the problem of overweight. Any interested family could attend, not just the overweight. Four families

constituted a group and a nutritionist and a behavioral science specialist led each group. The groups met weekly for 5 weeks to examine their families' eating and activity patterns, plan and implement behavioral changes and evaluate the effectiveness of those changes. Three areas were emphasized: food selection, eating habits and activity habits. Food selection concerned development of preferences for foods of high nutritional value. Eating habits emphasized slow eating, moderate sized portions, slow chewing and eating as a function of hunger vs. situational cues. Activity habits focused on development of activities that children could enjoy for a lifetime. Since a family-system approach was used, parents as well as their children could benefit from the program. The researchers suggested that this would help families to develop "thin" eating and activity styles which would be followed throughout a lifetime.

Cosper et al. (49) evaluated the effectiveness of a nutrition education program emphasizing weight control in 45 fifth-grade students with cultural endemic obesity. The control group of 22 students received no nutrition instruction while a group of 23 students were taught eight 30-minute units over 4 months time. Students in both groups were 87% Mexican-American and 13% Anglo. A test was administered before and after to test nutritional knowledge and a 24-hour recall was obtained before and after instruction. The experimental group knew a greater number of nutrition concepts after the instruction. Dietary intakes, however, did not show significant improvement. Predietary intakes were generally below the RDA in calories, calcium, iron, thiamine and vitamin A in the experimental group. There was little change in this after instruction. The authors concluded that without parental involvement, it was difficult to change the children's dietary habits.

### Treatment Methods

Early identification of obesity in children is clearly of value. Weil (5) suggested that dietary restriction is not advisable under one year of age however. Work with parents in helping them understand how to meet their infant's needs other than with food would be warranted. Between the ages of one and five years, use of skimmed or partially skimmed milk, elimination of carbohydrate snack foods, trimming of fat from meats, removing skin from fowl and use of broiling, roasting or boiling for cooking are approaches which may be useful. Significant caloric restriction (20 to 25%) should be used only in children over 5 years of age and then the goal should be either stable weight as height increases or no more than one-half pound weight loss per week in order to insure nutritional adequacy of the diet. In addition to dietary considerations, helping the child to increase physical activity is also beneficial.

Behavior modification has shown some promise in successful treatment of childhood obesity. Levitz and Jordan (46) reviewed unpublished research of Rivinus and coworkers at the Children's Hospital of Philadelphia of ten 10-year-old black children who were under treatment or awaiting treatment at the outpatient clinic. Mean percentage above ideal weight was 70%. The children kept records of foods eaten and other situational and environmental variables as is done for adults. A treatment team which included a pediatrician, a psychiatrist and a psychologist met and had dinner with the children weekly for 10 weeks. Mothers also attended the meetings and rewards were given the children when weight was lost. These rewards were intended to increase activity such as tickets to roller skating rinks or bowling alleys. Nine of the

children continued for 10 weeks and the average weight lost was 6.2 pounds. Those children had gained an average of 3.5 pounds in the 10 weeks prior to treatment. The drop-out rate was lower than the usual 33% in the outpatient clinic. Results also showed that children of normal-weight mothers lost more than children of obese mothers.

#### SUMMARY

Obesity is associated with increased risks of development of many diseases and is a common nutritional disorder in the United States. Prevention or treatment of obesity at an early age may help reduce the number of obese adults.

Assessment of obesity in children is usually done clinically by measurement of body weight, height and skinfold thickness. Those measurements may then be compared with standard charts.

Genetics and environment are both determinants of obesity in children, but the extent to which each affects its development is not clear. Fatness tends to run in families but whether this is due more to heredity or common family eating and activity patterns is not well established. Some studies have shown that bottle-feeding of infants and early introduction of solid foods lead to an increased incidence of obesity while other studies have shown no difference between infants who were artificially fed and those who were breast fed with later introduction of solids.

For the most part, studies in the early 1970's indicated that childhood obesity with onset during the first year of life was hyperplastic in nature. More studies in the latter half of the decade indicated an increase in fat cell number was not correlated with early onset

obesity. Most investigators agreed that there is some relationship between obesity in infancy and obesity in childhood and adulthood, but that there are many factors which determine a person's weight.

Prevention through nutrition education, influencing changes in activity and monitoring of weight gain are recommended. In treatment of childhood obesity, dietary restriction is not recommended under the age of one year. Even in older children, severe dietary restriction is generally unadvisable and the goal of treatment should be maintenance of a stable weight as height increases or no more than a loss of one-half pound per week. Behavior modification programs have been used successfully in treatment of some cases of childhood obesity.

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## LITERATURE CITED

1. Berkowitz, D. (1974) Obesity: etiologic mechanisms. In: Obesity: Causes, Consequences and Treatment (Lasagna, L., ed.), p. 3, Medcom Press, New York.
2. Robinson, C.H. (1972) Normal and Therapeutic Nutrition, p. 416, Macmillan Co., New York.
3. Court, J.M. & Dunlop, M. (1975) Obesity from infancy: a clinical entity. In: Recent Advances in Obesity Research: I (Howard, A., ed.), p. 34, Newman Publishing Ltd., London.
4. Garn, S.M., Clark, D.C. & Guire, J. (1975) Growth, body composition, and development of obese and lean children. In: Childhood Obesity (Winick, M., ed.), p. 23, Wiley, New York.
5. Weil, W.B. (1977) Current controversies in childhood obesity. J. Pediatr. 91, 175-187.
6. Tanner, S.M. & Whitehouse, R.N. (1976) Clinical longitudinal standards for height, weight, height velocity and stages of puberty. Arch. Dis. Child. 51, 170-175.
7. National Center for Health Statistics Growth Charts, Monthly Vital Statistics Report (1976) Vol. 25, no. 3, Suppl 6-22-76, DHEW Publ. No. (HRA) 76-1120.
8. Pipes, P.L. (1977) Nutrition in Infancy and Childhood, pp. 6-8, C.V. Mosby Co., St. Louis.
9. U.S. Department of Health, Education and Welfare (1972) Skinfold thickness of children 6-11 years. Data from the National Health Survey. Series 11, No. 20, DHEW Publication No. (HSM) 73-1602.
10. Seltzer, C.C., Goldman, R.F. & Mayer, J. (1965) The triceps skinfold as a predictive measurement of body density and body fat in obese adolescent girls. Pediatr. 36, 212-217.
11. Tanner, J.M. & Whitehouse, R.H. (1975) Revised standards for triceps and subscapular skinfolds in British children. Arch. Dis. Child. 50, 142-145.
12. Garn, S.M. & Clark, D.C. (1976) Trends in fatness and the origins of obesity. Pediatr. 57, 443-455.
13. Brook, C.G.D. (1979) Fat in the newborn. Arch. Dis. Child. 54, 845-847.
14. Garn, S.M. & Clark, D.C. (1975) Does obesity have a genetic basis in man? Ecol. Food Nutr. 4, 57-60.

15. Whitelaw, A.G.L. (1976) Influence of maternal obesity on subcutaneous fat in the newborn. *Brit. Med. J.* 1, 985-986.
16. Brook, C.G.D., Huntley, R.M.C. & Slack, J. (1975) Influence of heredity and environment in determination of skinfold thickness in children. *Br. Med. J.* 2, 719-721.
17. Poskitt, E.M. & Cole, T.J. (1978) Nature, nurture and childhood overweight. *Br. Med. J.* 1, 603-605.
18. Jelliffe, D.B. & Jelliffe, E.F. (1975) Fat babies: prevalence, perils, and prevention. *J. Trop. Pediat. Environ. Child. Health* 21, 123-159.
19. Dwyer, J.T. & Mayer, J. (1973) Overfeeding and obesity in infants and children. *Bibl. Nutr. Dieta.* 18, 123-134.
20. Taitz, L.S. (1971) Infantile overnutrition among artificially fed infants in the Sheffield region. *Brit. Med. J.* 1, 315-316.
21. Shukla, A., Forsyth, H.A., Anderson, C.M. & Marwah, S.M. (1972) Infantile overnutrition in the first year of life: A field study in Dudley, Worcestershire. *Brit. Med. J.* 4, 507-514.
22. Hall, B. (1975) Changing composition of human milk and early development of an appetite control. *Lancet* 1, 779-781.
23. Ounsted, M. & Sleight, G. (1975) The infant's self-regulation of food intake and weight gain. *Lancet* 1, 1393-1397.
24. Oakley, J.R. (1977) Differences in subcutaneous fat in breast- and formula-fed infants. *Arch. Dis. Child.* 52, 79-80.
25. Dubois, S., Hill, D.E. & Beaton, G.H. (1979) An examination of factors believed to be associated with infantile obesity. *Am. J. Clin. Nutr.* 32, 1997-2003.
26. Huenemann, R.L. (1974) Environmental factors associated with preschool obesity. I. Obesity in 6 month old children. *J. Am. Dietet. Assoc.* 64, 480-491.
27. DeSwiet, M., Fayers, P. & Cooper, L. (1977) Effect of feeding habit on weight in infancy. *Lancet* 1, 892-894.
28. Himes, J.H. (1979) Infant feeding practices and obesity. *J. Am. Dietet. Assoc.* 75, 122-125.
29. Ravelli, G.P., Stein, Z.A. & Susser, M.V. (1976) Obesity in young men after famine exposure in utero and early infancy. *N. Engl. J. Med.* 295, 349-353.
30. Garn, S.M., Bailey, S.M. & Cole, P.E. (1976) Similarities between parents and their adopted children. *Am. J. Phys. Anthropol.* 45, 539-543.

31. Hawk, L.J. & Brook, C.G.D. (1979) Family resemblances of height, weight and body fatness. *Arch. Dis. Child.* 54, 877-879.
32. Shenker, I.R., Fisichelli, V. & Lange, J. (1974) Weight differences between foster infants of overweight and non-overweight foster mothers, brief clinical and laboratory observations. *J. Pediatr.* 84, 715-719.
33. Wilkinson, P.W., Parkin, J.M., Pearlson, G., Strong, H. & Sykes, P. (1977) Energy intake and physical activity in obese children. *Br. Med. J.* 1, 756-757.
34. Griffiths, M. & Payne, P.A. (1976) Energy expenditure in small children of obese and non-obese parents. *Nature* 260, 698-700.
35. Hager, A., Sjostrom, L., Arvidsson, B., Bjorntorp, P. & Smith, U. (1977) Body fat and adipose tissue cellularity in infants. A longitudinal study. *Metabolism* 26, 607-613.
36. Brook, C.G.D. (1972) Evidence for a sensitive period in adipose cell replication in man. *Lancet* 2, 624-627.
37. Hager, A., Sjostrom, L., Arvidsson, B., Bjorntorp, P. & Smith, U. (1978) Adipose tissue cellularity in obese school girls before and after dietary treatment. *Am. J. Clin. Nutr.* 31, 68-75.
38. Hirsch, J. & Knittle, J.L. (1970) Cellularity of obese and non-obese human adipose tissue. *Fed. Proc.* 29, 1516-1521.
39. Jung, R.T., Gurr, M.I., Robinson, M.P. & James, W.P.T. (1978) Does adipocyte hypercellularity in obesity exist? *Br. Med. J.* 2, 319-321.
40. Brook, C.G.D., Lloyd, J.K. & Wolff, O.H. (1972) Relation between age of onset of obesity and size and number of adipose cells. *Brit. Med. J.* 2, 25-27.
41. Eid, E.E. (1970) Follow-up study of physical growth of children who had excessive weight gain in the first 6 months of life. *Br. Med. J.* 2, 74-76.
42. Charney, E., Goodman, H.C., McBride, M., Lyon, B. & Pratt, R. (1976) Childhood antecedents of adult obesity. *N. Engl. J. Med.* 295, 6-9.
43. Fisch, R.O., Bilek, M.K. & Ulstrom, R. (1975) Obesity and leanness at birth and their relationship to body habitus in later childhood. *Pediatr.* 56, 521-527.
44. Poskit, E.M.E. & Cole, T.J. (1977) Do fat babies stay fat? *Br. Med. J.* 1, 7-9.
45. Hawk, L.J. & Brook, C.G.D. (1979) Influence of body fatness in childhood on fatness in adult life. *Br. Med. J.* 1, 151-152.

46. Jordan, H.A. & Levitz, L.S. (1975) Behavior modification in the treatment of childhood obesity. In: Childhood Obesity (Winick, M., ed.), pp. 141-150, John Wiley & Sons, New York.
47. Pisacano, J.C., Lichter, H., Ritter, J. & Siegal, A.P. (1978) An attempt at prevention of obesity in infancy. *Pediatr.* 61, 360-364.
48. Smicklas-Wright, H. & D'Augelli, A.R. (1978) Primary prevention for overweight: Preschool Eating Patterns (PEP) program. *J. Am. Dietet. Assoc.* 72, 626-629.
49. Cosper, B.A., Hayslip, D.E. & Foree, S.B. (1977) The effect of nutrition education on dietary habits of fifth-graders. *J. Sch. Health* 47, 475-477.

CHILDHOOD OBESITY

by

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AN ABSTRACT OF A MASTER'S REPORT

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