

RELATION OF EARLY INFANTILE WEIGHT, HEIGHT
AND FATFOLDS TO FATNESS AT ONE YEAR

by

Ramon Frank Caruso

A Thesis Submitted to the Faculty of the

COMMITTEE ON AGRICULTURAL BIOCHEMISTRY
AND NUTRITION (GRADUATE)

In Partial Fulfillment of the Requirements
For the Degree of

MASTER OF SCIENCE

In the Graduate College

THE UNIVERSITY OF ARIZONA

1 9 7 8

STATEMENT BY AUTHOR

This thesis has been submitted in partial fulfillment of requirements for an advanced degree at The University of Arizona and is deposited in the University Library to be made available to borrowers under rules of the Library.

Brief quotations from this thesis are allowable without special permission, provided that accurate acknowledgment of source is made. Requests for permission for extended quotation from or reproduction of this manuscript in whole or in part may be granted by the head of the major department or the Dean of the Graduate College when in his judgment the proposed use of the material is in the interests of scholarship. In all other instances, however, permission must be obtained from the author.

SIGNED: _____

APPROVAL BY THESIS DIRECTOR

This thesis has been approved on the date shown below:

Gail G. Harrison
GAIL G. HARRISON

Assistant Professor of Nutrition and
Food Science

July 13, 1978
Date

ACKNOWLEDGMENTS

The author wishes to express his gratitude to Dr. Gail G. Harrison for her assistance in the planning and execution of this study. The author would like to express thanks to Dr. Glenn M. Friedman, whose pediatric practice made this study possible and Mr. John A. Gaines for performing the statistical analysis.

The author wishes to thank Dr. James W. Berry and Dr. Charles W. Weber, and the professors, staff and secretaries of the Department of Nutrition and Food Sciences and the Department of Family and Community Medicine for their cooperation. Acknowledgment is also made to Ms. Linda S. Blocker and Ms. Geri Rizzo Caruso for assisting the author.

This study was supported by Biomedical Research Support Grant 3150-80 from the National Institutes of Health RR05675, to the College of Medicine, The University of Arizona, Tucson.

TABLE OF CONTENTS

	Page
LIST OF TABLES	v
LIST OF ILLUSTRATIONS	vi
ABSTRACT	vii
INTRODUCTION	1
LITERATURE REVIEW	2
Definition of Obesity	2
Prevalence	3
Adipose Tissue Cellularity	4
Family Influence on Childhood Obesity	6
Feeding Types	8
Familial Correlations	9
Relative Risk of Infant-Child-Adult-Progression of Obesity	11
SUBJECTS AND METHODS	15
Subjects	15
Recording Methods	15
Handling and Analysis of Data	17
RESULTS AND DISCUSSION	19
SUMMARY AND CONCLUSIONS	35
APPENDIX A: PRE-CODED DATA COLLECTION FORMS	37
LIST OF REFERENCES	40

LIST OF TABLES

Table	Page
1. Characteristics of infants in study population at encounters one, two and three	20
2. Subscapular fatfold measurements for the sixty one-year old infants at encounter three	22
3. Birthweight and sex of the thin and fat infants along with the variables recorded at encounters one and three .	23
4. Variables for the one-year old thin and fat infants along with their means, standard deviations, "t" and "p" values	25
5. Comparison of feeding types for thin and fat infants at birth through two months and at one year	27

LIST OF ILLUSTRATIONS

Figure	Page
1. Chi square and "p" values for feeding types at birth through two months	28
2. Path analysis for relationships among birthweight, sex, weight, height and subscapular fatfolds at each encounter	29

ABSTRACT

In a private pediatric practice, 184 normal White infants were enrolled and were measured at ages birth to two months. At six months, 127 of these infants were measured, and 60 at one year (\pm one month) were measured. Relationship of subscapular fatfolds at six months and one year to earlier anthropometric characteristics were explored using path analysis. Fatfolds at six months were related positively to fatfolds at zero to two months, but at one year there was no significant relationship of fatfold measurements with earlier fatfolds. At all ages, weight was a relatively poor predictor of adiposity, explaining less than 10% of the variability in fatfold measurement. Neither weight nor fatfold at one year was significantly associated with the same measurement at birth to two months. It is concluded that in this group of infants, fatness at one year is not necessarily an inevitable consequence of fatness in early infancy.

INTRODUCTION

Obesity is presenting a serious problem for Western civilization. Studies have indicated that obesity is associated with hypertension, diabetes and other related illnesses. Since treatment of the already obese person has a very low success rate, it is important to prevent the development of obesity.

Studies have indicated that some lifelong obesity has its origin in infancy. This is supported by adipose tissue cellularity studies and follow-up studies of infants who were obese, normal and thin during their early months. The evidence is still inconclusive concerning the risk for progression of obesity for the fat infant. Generally, the studies to date have been cross-sectional for only a short period of time and with limited populations.

The objective of this study was to determine in a group of basically healthy infants, if early infantile weight, height and subscapular fatfolds were related to fatness at one year of age.

LITERATURE REVIEW

Definition of Obesity

Obesity -- an excess of subcutaneous adipose tissue, caused by high caloric intake and/or under-activity. This working definition poses questions concerning the measurement of adipose tissue and what is excessive fat. Also, what should be the cut-off point for obese and normal subjects?

Body weight measurements are used to determine the weight of subjects and have been related to fatness. For body weight to be a reliable index of fatness, age, sex, weight and body build must also be considered as contributing factors for an adequate measure. If body weight is solely used it will underestimate fatness in children under seven years of age and overestimate fatness in adolescence.

Weight for height (W/H) indices have been used by some authors to define thin, normal or obese subjects. Height to the second power (H^2) is used to correct for the differences in height. Because there is no correction for age, as one gets older W/H² indices will show him getting fatter. W/H² also would exclude muscular subjects from the obese category and may also categorize subjects with increased body fat as non-obese.

A more direct measure of body fat is achieved with the use of fatfold calipers. Fatfold measurements are performed by "pinching" the skin with the calipers to determine the amount of subcutaneous fat.

present. Two commonly used body sites are triceps and subscapular. The assumption that subcutaneous fat constitutes a constant proportion of total body fat is made when using fatfold measurements as an index. Values have been charted from the data obtained from the Ten-State Nutrition Survey (Center for Disease Control 1972). Subjects above the eighty-fifth percentile are considered obese and thin subjects are below the fifteenth percentile. Fatfold measurements are a satisfactory index for community studies.

Because fatfold measurements are obtained by using an instrument that records in millimeters, there is a margin of error when different people perform the task. For this reason standardization among investigators within each study is needed. For greater accuracy in determining the obese child more information and data concerning fatfold measurements are needed. This is also true for people over the age of 70 years.

Prevalence

It is estimated that between 25 and 45% of the United States adult population is overweight (Bray 1972). For United States school children between six and fifteen years old, it is estimated there is a 15% obesity rate (Jelliffe and Jelliffe 1975). Approximately 59.6% of normal children attending a well-baby clinic in Sheffield, England, were overweight, some as early as six weeks of age (Taitz 1971). Also in England, in 1973, 40% of the babies weighed over 15½ pounds at three months of age, compared to 10% twenty years previously (Jelliffe and Jelliffe 1975). Furthermore, Neumann (Neumann and Alpaugh 1976) showed

the mean birthweight doubling time for both males and females from Los Angeles, was 3.8 months. This rate is much lower than a five to six months doubling time suggested by most pediatric textbooks. With the incidence of obesity increasing throughout all ages and the general long-run effectiveness of all existing methods of treatment, the best solution to the obesity problem would be to prevent its occurrence.

Many factors contribute to the onset of obesity. Less than 1% are obese due to endocrine abnormalities (Hassell 1974). The predominant factors associated with obesity include: social class (Charney et al. 1976), environment (Hartz, Giefer and Rimm 1977), lack of nutrition education (Charney et al. 1976; Jelliffe 1974), primary birth order (Huenemann 1974), excess caloric intake (Forbes 1977; Huenemann 1974), obesity among parents (Charney et al. 1976; Jelliffe 1974), and difference in adipose tissue morphology (Johnson and Hirsch 1972). One of the questions yet to be answered is what is the relative risk of an obese infant becoming an obese child and subsequently an obese adult?

Adipose Tissue Cellularity

In strong support of the hypothesis of the risk of infant-child-adult progression of obesity, researchers have been investigating adipose tissue cellularity. They have shown that the growth of adipose tissue begins with cell number increase and is then followed by cell hypertrophy. Hirsch (1975) stated, that in infant and child-onset obesity there will be an increase in cell number, whereas with adult-onset obesity, there will be an increase in cell size. He also

suggests that once the number of adipose tissue cells are formed they will not decrease in number. When an adult loses weight, only cell-size decreases.

Researchers differ over the critical period for cell proliferation. Brooke (1972) postulated that adipose tissue develops by cell proliferation from the age of 30 weeks in utero and continues for the first nine to twelve months after birth. He also noticed an increase in cell number in all obese people, while investigating those who were obese at one year and adults who dated their obesity to childhood (Brooke, Lloyd and Wolf 1972). In a later study with normal children, Knittle (1976) observed that cell proliferation did not reach the maximum number until the age of eight to twelve years. He also found a difference between obese and nonobese children. In the obese child, cell proliferation occurred throughout childhood until puberty. This contrasted with the nonobese child who showed cell proliferation during the first two years of life and again immediately before puberty.

Criticism surrounding adipose tissue cellularity is expressed not only by those who oppose the theory but also by its advocates. Much of the work with adipose tissue cellularity has been performed with non-human animals, such as pigs and rats. Questions usually arise from extrapolation of animal studies to humans (Hirsch and Han 1969). Hirsch, one of the primary workers in adipose tissue cellularity, admits there are dangers when attempting to extrapolate from animal studies. In utilizing electronic counting of osmium-fixed cells, Greenwood and Johnson (1977) realized that only cells having a fair quantity of lipid present could be counted. This method excludes the

cells with very little or no lipid content, very small cells, and the preadipocytes. Ashwell (Ashwell and Garrow 1973) expresses concern about extrapolating the data obtained from a few milligrams of adipose tissue to the kilograms of fat present in the human body. In a paper presented before the First International Congress on Obesity, Ashwell, Priest and Bondoux (1974) observed no significant difference in human adipose tissue cellularity when associated with the age of obesity-onset in overweight females.

Once the limitations of the procedures are perfected, this author feels the knowledge obtained from adipose tissue cellularity studies will be beneficial in the prevention of obesity. The questions now posed by this knowledge include:

- a. What causes an infant to acquire an excess number of adipose cells?
- b. How do the parents contribute to obesity of the infant?
- c. Is type of feeding important at an early age?
- d. Is the infant's birthweight a determining factor of later obesity?
- e. What is the risk for an obese infant eventually becoming an obese adult?

Family Influence on Childhood Obesity

The discussion concerning family influences upon infant obesity will be divided into two parts: what effect does the mother alone have on the infant, and secondly, how does the entire family affect the infant after birth?

Recent studies indicate a relationship between maternal pre-pregnant weight and weight gained during pregnancy to the birthweight of the child (Whitelaw 1976; Ademowore, Courey and Kime 1972). Ravelli (Ravelli, Stein and Susser 1976) conducted a retroactive study with Dutch males 19 years of age who were exposed to famine in utero and during their first few months of life. It was brought out that, when the mother was subjected to undernutrition during the first half of pregnancy, there was a higher obesity rate for the 19 year old boys. In contrast, the boys who were exposed to undernutrition during the last trimester of gestation and/or the first months of postnatal life, expressed lower obesity rates at age 19 years.

Currently a study (Udall et al. in press) is being conducted at The University of Arizona Health Sciences Center to determine whether maternal obesity prior to becoming pregnant and weight gain during pregnancy correlates with infant fatness and high birthweight. The relationship is being investigated by use of fatfold measurements of neonates and their mothers. Preliminary results from this study indicate that obese mothers are more likely to give birth to large for gestational age infants, and the thinner mothers to give birth to average for gestational age infants. It was also concluded that all large for gestational age (LGA) infants were not considered fat. The "fatter" infants in this LGA class all had increased fat deposition. Interestingly, the mothers of these "fatter" infants were not only more likely to be obese before pregnancy, but also to have gained in excess of 40 pounds during pregnancy. This preliminary study leads

one to believe that weight gain during pregnancy may affect the fatness of the infant at birth.

Feeding Types

The onset of obesity in infants has been attributed to breast-feeding, bottle-feeding, early introduction to solid foods, and over-feeding. Arguments surround the benefits and hazards of breast-feeding versus bottle-feeding. Thomson (1955) divided 40 pairs of British female infants into breast-fed and bottle-fed groups. The findings revealed identical weight gain by both groups at 16 weeks of age. Other follow-up studies from England also indicated bottle-fed infants were not significantly different from breast-fed infants by eight years of age in their incidence of obesity (Eid 1970; Mellbin and Vuille 1973). In contrast, Hooper (Hooper and Alexander 1971) indicated bottle-fed infants, from his practice in Newport, England, gained more weight than breast-fed infants. A British field study indicated that bottle-feeding and early weaning leads to an overweight infant (Shukla et al. 1972). Advocates for breast feeding agree that breast feeding may be a preventive measure against infant obesity. Another English study revealed that with 21 breast-fed and 240 bottle-fed infants, a significantly higher percentage of bottle-fed infants were above the nineteenth percentile for weight gain by six weeks of age (Taitz 1971). This work is further supported by studies from Australia and England that claim breast-fed infants will gain less weight than bottle-fed infants of approximately the same birthweight (Court 1977; Rayner et al. 1974).

Introduction of solid foods at an early age has also been implicated in the increase in infant obesity. Huenemann's (1974) work refutes the premise that early introduction to solid foods leads to a more obese infant. She found the age at which solid foods were introduced was approximately the same in the obese as in the lesser weight groups. Shukla and co-workers (Shukla et al. 1972) believe early introduction to solid foods may lead to infant obesity. If this is so, they feel the effect is only for the first six months of life. As expressed above, there are advocates and opponents for each theory concerning the development of obesity. There is no incontrovertible evidence that bottle feeding and early introduction to solid foods are potential factors leading to infantile obesity.

Familial Correlations

The outcome of the infant is affected not only by the condition of the mother during pregnancy and feeding but also by the influence of the family after the birth of the child (Garn 1976; Rayner et al. 1974; Sveger et al. 1975). The effects of relationships between family members on trends in obesity are clearly represented by the data collected during the Ten-State Nutrition Survey.

The Ten-State Nutrition Survey (TSNS) (Center for Disease Control 1972) conducted from 1968 through 1970, addressed the problem of over-nutrition as well as under-nutrition. Weight for height relationships were not an adequate measure of fatness, for a child may be overweight but not necessarily fat. For this reason, fatfold measurements (triceps and subscapular) were used to establish body categories --

"lean," "medium," and obese. The TSNS was conducted with more than 40,000 people, including parents and their children. With this wealth of data available, comparisons not only of parents and children but also of siblings could be made.

Garn and Clark (1976) were co-authors for the Ad Hoc Committee to Review the Ten-State Nutrition Survey and published a paper concerning the occurrence of obesity along family lines. They noticed that, as the mating types (lean, medium, obese) progressed towards obesity, the children also increased in fatness. This trend in fatness, TSNS noted, increased to the end of the child's teens, and, at age 17, children of obese parents are approximately three times fatter than children of lean parents. There was no indication that one parent contributes more to the obesity of the child than another. Sons and daughters were equally affected by their parents.

There was a higher correlation of obesity between siblings than between parent and child; $r = 0.40$ versus $r = 0.25$, respectively. Sex had no influence on risk of obesity; brothers as well as sisters were equally affected. In sibling relationships, the fatness of the sibling is in direct proportion to the fatness of the older brother or sister, even though not all obese children have obese brothers and/or sisters. If one child in a family is identified as obese, there is a 40% chance that the second child in the family will be obese. In a three child family, there was an 80% chance that at least one of the two siblings of an obese child will also be obese (Garn and Clark 1976).

Obesity therefore, manifests itself along family lines. This fact may be attributed to one or all of the following reasons:

- a. The tendency of familiar body types to marry each other.
- b. The tendency for mates to become more alike after marriage.
- c. Similarities in eating habits and attitudes towards food.
- d. Younger child imitating older child and vice versa.
- e. Genetic inheritance.

To reinforce further the family-line tendencies towards obesity, it was shown that adopted American children became similar to their parents and/or new siblings in body fat composition. This degree towards likeness is a function on the length of time living with their new family (Food and Nutrition Board 1978). Also, infants placed with obese foster mothers tended to become more obese than infants placed with non-obese foster mothers (Shenker, Fisichelli and Lang 1974).

Relative Risk of Infant-Child-Adult- Progression of Obesity

Is an obese infant destined to a life of obesity? Is a child with an increased number of adipocytes "programmed" to be an obese adult? These questions and others concerning the hypothesized infant-child-adult progression of obesity have been studied by many researchers. As with the previous concerns, the results are inconclusive.

In the early 1970's, Shukla (Shukla et al. 1972) and co-workers studied 300 infants from Dudley, Worcestershire, England and discovered a relatively high incidence of obese (17%) and overweight (25%) infants during their first year of life. In the population, they found a positive correlation between birthweight and weight at one year. Infants who had a high nutrient intake during their first 13 weeks, were noticed to show an increase in weight throughout their first year.

Five years after Shukla conducted his study, Poskitt (1976) studied the children again to determine who were presently obese or overweight. His findings revealed,

- a. one in four obese infants became an overweight child,
- b. 65% of the obese infants and 75% of the overweight infants were normal children at age five, and
- c. 1 1/2% of the normal infants became obese by age five.

He concluded that obese infants usually return to normal weight by childhood, and that infant obesity may lead to childhood obesity but that it is only one of many factors contributing to it.

The relative risk of infant-child-adult progression of obesity offers a wide area for research. Certain populations may express this relationship, although others may not. Because of this, researchers do not agree whether a positive prediction can be made. Asher (1966), in a study with overweight British children, noticed that an excessive weight gain within the child's first few months of life, led to an obese child. Four years later, Eid (1970), in a follow-up study, also related childhood obesity to rapid weight gain among British infants. The mean height of children who gained weight rapidly in infancy was significantly higher than of children who gained weight slower. The number of obese children in the rapid weight gain group was significantly higher than in the average and slower weight groups combined. He also states that rapid weight gain in infancy is a better indicator for risk of a child's being overweight in childhood than weight of the parents. A study of a population in Northern Labrador showed 70% of the infants less than one year old were overweight or obese. It also

indicated about 49% of children ten years old were also overweight or obese (Creery 1972). Among the subjects, there was a high incidence of fatness at one year old and at ten years old; therefore, it may be safe to assume that the majority of the one year olds will also be overweight at ten years of age.

Separate studies in Australia (Court 1977) and Sweden (Sveger et al. 1975) indicated similar results concerning relative risk of infant-child-adult progression of obesity. Both studies indicated approximately 80% of the overweight or obese young children became overweight older children and adults. The Swedish study related infantile obesity with high birthweight. In the Australian study, rapid weight gain and high birthweight were not accurate indicators that the infant would be an obese adult. Charney and co-workers (1976) compared records of 366 American infants born between 1945 and 1955. Those infants whose weights were above the ninetieth percentile during their first six months had approximately a 2½ times greater possibility of being overweight as adults.

As stated previously, researchers disagree over the extent of correlation between obesity in an infant leading to adult obesity. Lloyd (1977) found no convincing connection between obesity at one year and adult obesity. She indicated that between 60% and 80% of overweight infants will be of normal weight by seven years of age. Of the 20% that remains obese, 80% of these will become obese adults. Mellbin and Vuille (1973) also indicated that weight gain during the first year is not a strong indicator for obesity at seven years of age. Studies concerning birthweight as an indicator of infant obesity have

also offered conflicting reports as to whether obesity will be present throughout life (Lloyd 1977; Neumann and Alpaugh 1976).

Whitelaw (1977), in a follow-up study of British infants found no relation between subcutaneous fat at birth and at one year. His study consisted of a population of obese, normal, and thin infants at birth with a randomly chosen sample of ten thin, 20 obese, and 60 normal infants at one year.

In summary, the possibility that infant obesity will progress to adult obesity is still questionable. With many factors contributing to the obese infant, child, and adult, it would presently be difficult to attribute adult obesity to infant obesity.

SUBJECTS AND METHODS

Subjects

Two hundred infants were used as the study population, all of whom were healthy at birth and did not experience any difficulties during birth. The population was Caucasian, and included both males and females from birth to two years old. Siblings were also included. The majority of the subjects resided in Scottsdale, Arizona, an upper-middle class community. All the subjects were patients of Glenn M. Friedman, M.D., whose practice also is in Scottsdale. This author obtained the raw data from Dr. Friedman's files. All measurements were performed by a nurse.

The data reviewed and recorded span the years 1971 through 1977; the author used no measurements taken after the subjects were two years old. The author reviewed the data during the summer of 1977 and transferred the appropriate information to pre-coded forms (Appendix A).

Recording Methods

The author established criteria for selecting the study population prior to arriving in Scottsdale. Each infant had to meet basic requirements: all infants used had to be healthy at birth and to suffer from no birth defects; infants of diabetic mother were eliminated; and infants had to have at least two encounters beyond their

births but within two years after their births. Approximately 300 infants fit the criteria above.

The birth records (sex, weight, length, whether adopted, whether premature) were obtained either from the various hospitals or from the parents themselves. Measurements of the subjects were taken during their regular office visits for well-child care and consisted of weight (beam balance), length, and subscapular fatfold (Lange calipers). In addition, the author sought further information which was not regularly recorded during each visit. The information consisted of:

1. feeding type
2. race or ethnic group
3. mother a smoker (yes, no)
4. history of family hypertension
5. history of coronary or stroke in family members under age 60
6. mother diabetic while pregnant
7. was baby adopted
8. was baby premature

It should be noted that most of the records did not include all the questions above.

The raw data for weight and length were recorded in pounds and ounces and inches, respectively. These data were recorded by one of several different nurses during the six year span. The author converted the measurements to metric numbers with conversion factors of ounces x 28.35 and inches x 2.54. Because several nurses recorded the data, the author assumes a margin of error in the data; however,

because the author cannot determine such a margin, he must also assume that measurement error was random.

Handling and Analysis of Data

The data were arranged according to "encounters," descriptive of the time periods at which the infants were measured:

1. birth
2. encounter one (birth through two months)
3. encounter two (five through seven months)
4. encounter three (eleven through thirteen months)
5. encounter four (seventeen through nineteen months)
6. encounter five (twenty-two through twenty-four months)

Once the arrangement of the data was established, it was determined that only 200 infants had a first encounter within two months of birth.

At first, the author included in the regression analysis all the variables listed on the previous page. Because of missing data, only the following variables were used: birthweight, sex, weight, length, and subscapular fatfold. The preliminary analysis also revealed that the scope of the study had to be further limited because data spanning encounters four to five (17 through 24 months) were insufficient. This analysis showed:

encounter one:	184 infants
encounter two:	127 infants
encounter three:	60 infants
encounter four:	27 infants
encounter five:	insufficient data

Regression analysis was performed with the variables stated above and encounters one through three. Regressions were performed as follows:

- a. encounter one (weight, height, fatfold) with birthweight and sex;
- b. encounter two (weight, height, fatfold) with encounter one and birthweight and sex;
- c. encounter three (weight, height, fatfold) with encounter one and birthweight and sex.

Once all regressions were completed, path analysis was used as another statistical aid to interpret the data.

Path analysis is a convenient representation of data utilizing multiple regression with structured ordering. Consideration is given to the correlation of the variables. Sewall Wright (1921) developed path analysis and states,

a method of measuring the direct influence along each separate path in such a system and thus finding the degree to which variation of a given effect is determined by each particular course. The method depends on the combination of knowledge of the degree of correlation among the variables in a system with such knowledge as may be possessed of the casual relations. The purpose of path analysis is to determine whether a proposed set of interpretations is consistent throughout (Wright 1921, p. 557).

Because the data were arranged in a time sequence and because there was logical ordering of the variables, it was possible to utilize path analysis.

RESULTS AND DISCUSSION

The initial population consisted of 128 (63%) female and 74 (37%) males, all of whom were normal White infants. The data were arranged according to the following time sequence:

- a. encounter one (zero through two months)
- b. encounter two (seven through five months)
- c. encounter three (eleven through thirteen months).

One hundred eighteen (64%) female and 61 (36%) males were included in encounter one, along with 83 (65%) females and 44 (35%) males for encounter two. Encounter three consisted of 38 (63%) females, and 22 (37%) males.

With the decrease in cases from 184 to 60, encounter one through encounter three respectively, the data were reviewed to determine if birthweight, sex, weight, height and fatfolds at the initial encounter were different from the groups remaining in the study versus those who accounted for the attrition. Table 1 illustrates the mean and standard deviation for the five variables and indicates no appreciable difference between the cases at encounter one, encounter two and encounter three when related at zero through two months. The figures for sex indicated that females were dominant throughout the encounters. Since the 60 infants from encounter three were a representative sample of the population relative to the variables of interest, a comparison of these infants was explored.

Table 1. Characteristics of infants in study population at encounters one, two and three. -- Means and standard deviations are presented.

Variable	Encounter One n = 184	Encounter Two n = 127	Encounter Three n = 60
Birthweight (gm)	3289.1 \pm 489.9	3287.4 \pm 461.6	3256.6 \pm 486.7
Sex (0 = M) (1 = F)	0.636	0.622	0.617
Weight (gm) at Encounter One	4069.8 \pm 898.9	4060.7 \pm 945.8	3930.5 \pm 664.4
Height (cm) at Encounter One	54.6 \pm 3.6	54.8 \pm 4.0	54.1 \pm 2.8
Subscapular fatfold (mm) at Encounter One	5.7 \pm 1.7	5.7 \pm 1.7	5.2 \pm 1.5

The 60 one-year old infants were compared to determine if a distinction could be made between characteristics of "fatter" and "thinner" infants at one year. The categories fat and thin do not correspond with standard values for fatfold measurements, but were used for this population exclusively. Infants with fatfold measurements of $4\frac{1}{2}$ millimeters and below were arbitrarily designated as thin, while those of 9 millimeters and above were termed fat. Five females and three males were included in the thin population, with the fatter infants being three females and five males (Table 2).

Table 3 lists the characteristics of the thin and fat infants at encounter one and three along with birthweight and sex. From these data Student's t-tests were performed to compare the two groups with regard to:

- a. birthweight,
- b. weight at one year,
- c. weight gained by one year,
- d. percent weight gained by one year, and
- e. fatfold measurements at one year.

The feeding types of the two groups at the age of birth through two months were also studied utilizing the chi square.

A list of the five variables mentioned above with their means, standard deviations, "t" and "p" values are presented in Table 4. As was expected, birthweight, weight at one year and weight gain by one year had larger mean and standard deviations for the fatter infants than the thinner ones. The "p" values for the three categories showed no significant difference at any level. This also held true for

Table 2. Subscapular fatfold measurements for the sixty one-year old infants at encounter three

Subscapular Fatfold (mm)	n (N = 60)
4.0 ^a	6
4.5 ^a	2
5.0	8
6.0	12
6.5	1
7.0	15
8.0	8
9.0 ^b	2
10.0 ^b	4
11.0 ^b	2

^aArbitrarily designated as thin.

^bArbitrarily designated as fat.

Table 3. Birthweight and sex of the thin and fat infants along with the variables recorded at encounters one and three. -- Means and standard deviations are given for the appropriate variables.

Birthweight (gm)	Sex	Weight ^a (gm)	Height ^b (gm)	Fatfold ^c (mm)	Weight ^d (gm)	Height ^e (gm)	Fatfold ^f (mm)	Feeding Types g h	
Thin Infants (n=8)									
2268	F	3232	50	4.5	9979	79	4.0	3	9
2410	F	3515	52	5.0	9752	80	4.0	6	7
3033	F	3545	51	5.0	8335	75	4.5	1	7
3189	M	4082	53	4.0	9667	77	4.0	6	7
3204	F	3487	53	4.5	9497	77	4.5	1	7
3232	F	3912	50	5.0	8732	77	4.0	6	7
3246	M	4309	53	5.0	9299	76	4.0	5	7
3317	M	3941	58	5.0	9185	77	4.0	2	7
2987 [±] 410		3752 [±] 363	53 [±] 2.6	4.8 [±] 0.4	9306 [±] 548	77 [±] 1.6	4.1 [±] 0.2		
Fat Infants (n=8)									
2140	F	2977	48	2.0	8505	72	9.0	6	7
2339	M	3119	50	4.0	9157	74	10.0	2	7
3204	M	4479	55	3.0	8335	80	9.0	2	7
3274	F	3175	51	5.0	9696	74	10.0	1	7
3374	F	3062	55	7.0	11964	77	10.0	6	7
3445	M	4026	53	8.0	8789	76	11.0	6	7
3473	M	4536	59	5.0	9979	77	11.0	1	7
4139	M	4876	61	6.0	12332	81	10.0	2	7
3174 [±] 645		3782 [±] 782	54 [±] 4.4	5 [±] 2	9845 [±] 1530	76 [±] 3.1	10 [±] 0.8		

Table 3 Continued.

^aWeight at encounter one

^bHeight at encounter one

^cSubscapular fatfold at encounter one

^dWeight at encounter three

^eHeight at encounter three

^fSubscapular fatfold at encounter three

^gFeeding types at encounter one

1 = breast

2 = formula

3 = combination

5 = solids and breast

6 = solids and formula

^hFeeding types at encounter three

7 = table food

9 = no data

Table 4. Variables for the one-year old thin and fat infants along with their means, standard deviations, "t" and "p" values.

Variables	Thin Infants	Fat Infants	"t"	"p"
Birthweight (gm)	2987 \pm 410	3174 \pm 645	0.693	N.S.
Weight at one year (gm)	9306 \pm 548	9845 \pm 1530	0.937	N.S.
Weight gain by one year (gm)	6318 \pm 844	6671 \pm 1215	0.675	N.S.
Percent of birth-weight gained by one year	219 \pm 65	217 \pm 56	0.064	N.S.
Subscapular fatfold at one year (mm)	4.1 \pm 0.2	10.0 \pm 0.8	20.98	<0.001

percentage of weight gained by one year, even though the fatter infants had a lower (2%) percentage rate. The fatfold measurements for the two groups did show a significant difference at the $p < 0.001$ level.

Feeding types were compared to establish if a difference in eating patterns between the two groups were present at the age of birth through two months. The feeding types were divided into two categories: solid versus non-solid and bottle versus breast versus bottle and breast (both) (Table 5). A feeding type was considered solid if any part of the meal contained solid food and the bottle and breast-fed categories were used even if solid foods were present. Feeding types at one year were not compared because all infants were fed table food by that time.

The data (Fig. 1) indicated that at $p < 0.05$ ($\chi^2 = 5.334$) there was a difference between solid and non-solid feedings, with fatter infants at one year being less likely to have been fed solid foods prior to age two months than thinner infants. This is opposite of what was expected, for it seems that the fatter infants should have been fed solids earlier than the thinner infants. The second category did not show any significant difference ($\chi^2 = 1.667$) for the fat and thin infants between the three groups of feedings.

The relationships among birthweight, sex, weight, height and subscapular fatfolds at each encounter were explored using path analysis (Fig. 2). Fatfolds at encounter two were positively correlated ($r = 0.24$) with fatfolds at encounter one. Birthweight and weight at encounter two also contributed to predicting the fatfold measurement at encounter two (multiple $R = 0.39$). No significant correlation was

Table 5. Comparison of feeding types for thin and fat infants at birth through two months and at one year.

Thin Infants			Fat Infants	
Feeding Types		n	Feeding Types	
				n
Birth through two months	Bottle	1	Breast	2
	Bottle and breast	1	Bottle	3
	Solids and breast	1	Bottle and breast	3
	Breast	2		
	Solids and bottle	3		
One Year	Table food	8	No data	1
			Table food	7

A

	Solids	No Solids
Thin Infants	4	4
Fat Infants	0	8

$$\chi^2 = 5.334$$

$$p = 0.05$$

B

	Bottle	Breast	Both
Thin Infants	4	3	1
Fat Infants	2	3	3

$$\chi^2 = 1.667$$

$$p = \text{N.S.}$$

Figure 1. Chi square and "p" values for feeding types at birth through two months. -- A, solid feeding versus no solid feeding for the thin and fat infants. B, bottle feeding versus breast feeding versus bottle and breast feeding for the thin and fat infants.

A, Encounter One

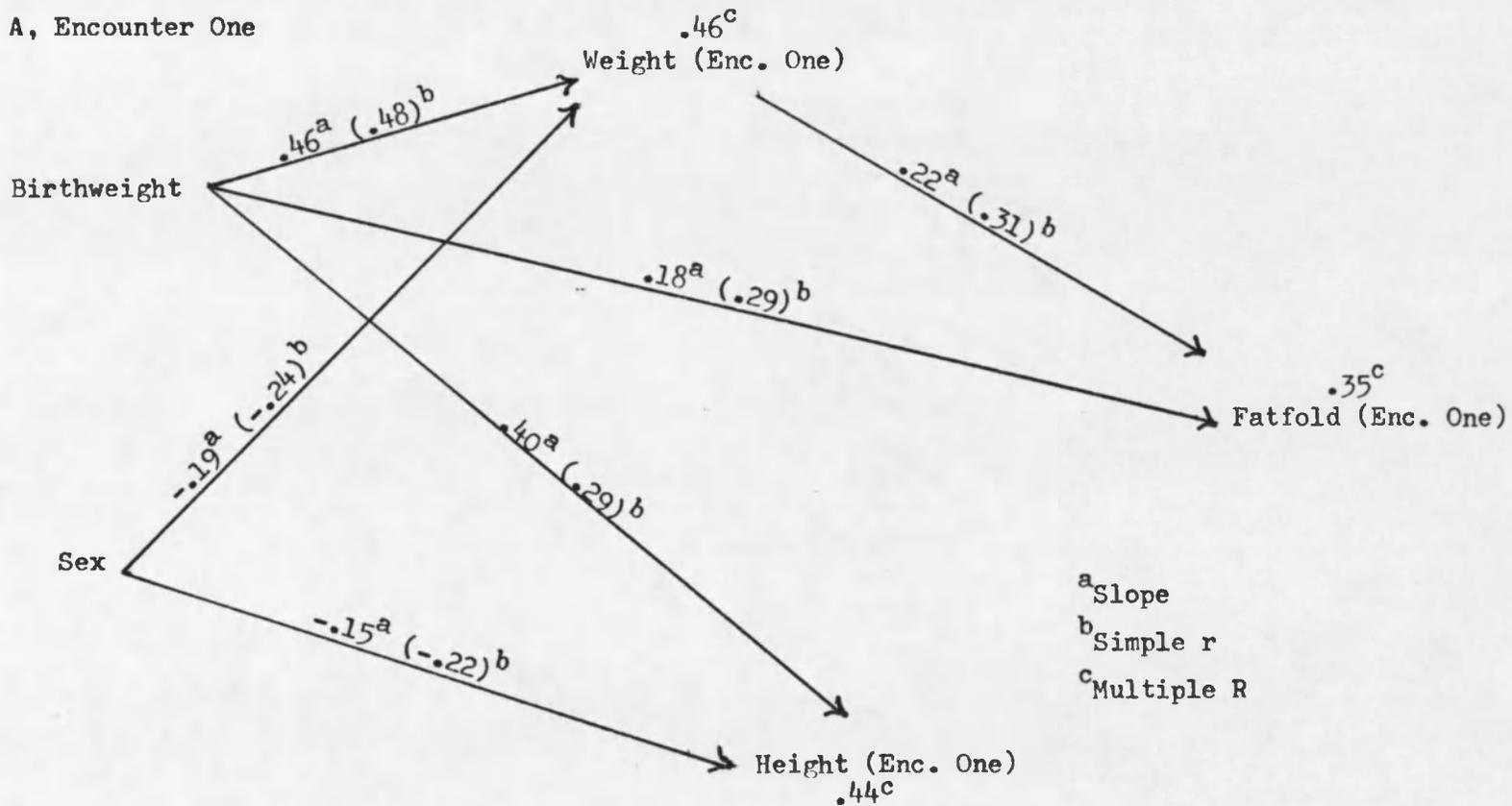


Figure 2. Path analysis for relationships among birthweight, sex, weight, height and subscapular fatfolds at each encounter. -- A, Encounter One.

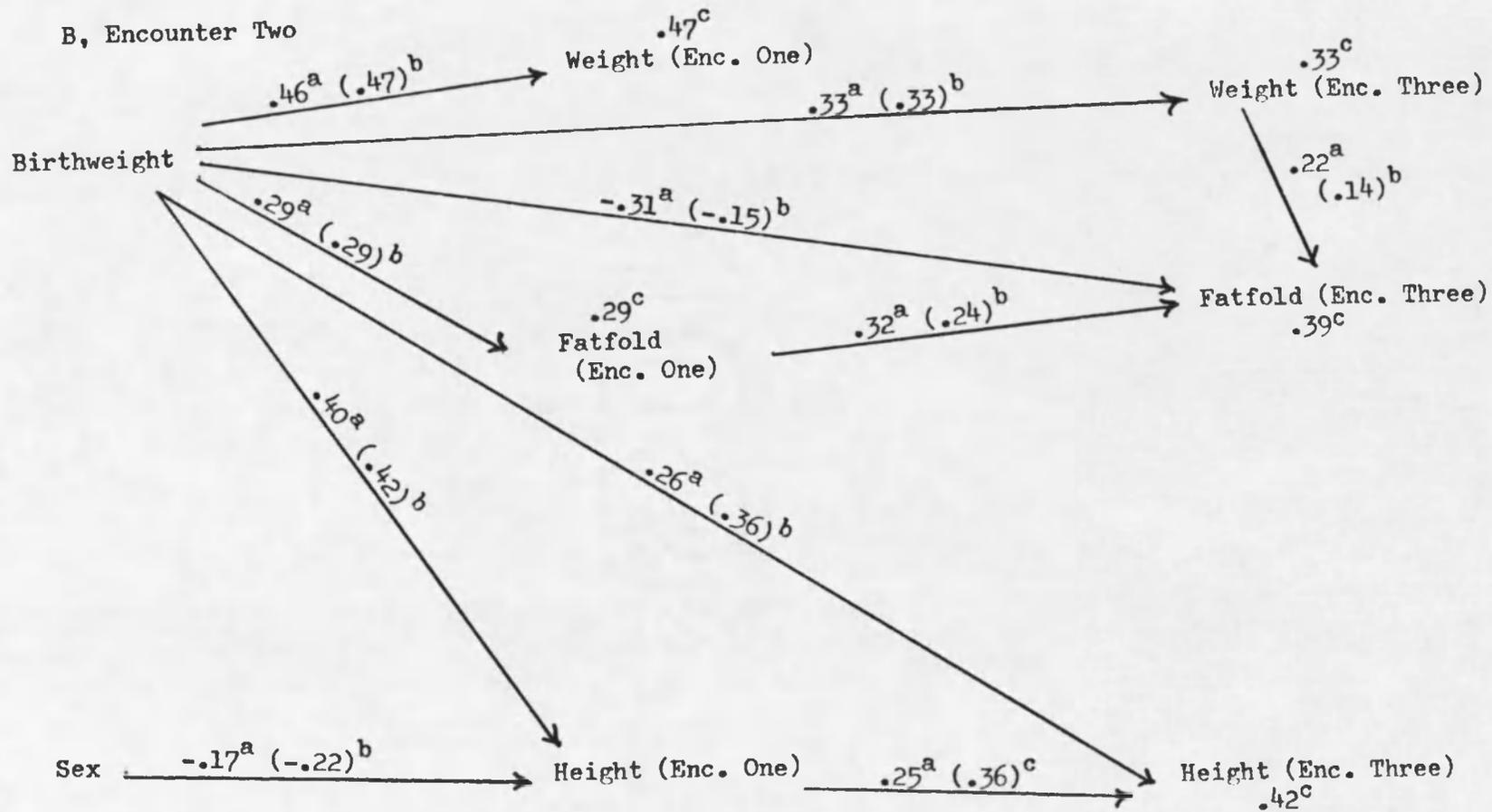


Figure 2 Continued. -- B, Encounter Two.

C, Encounter Three

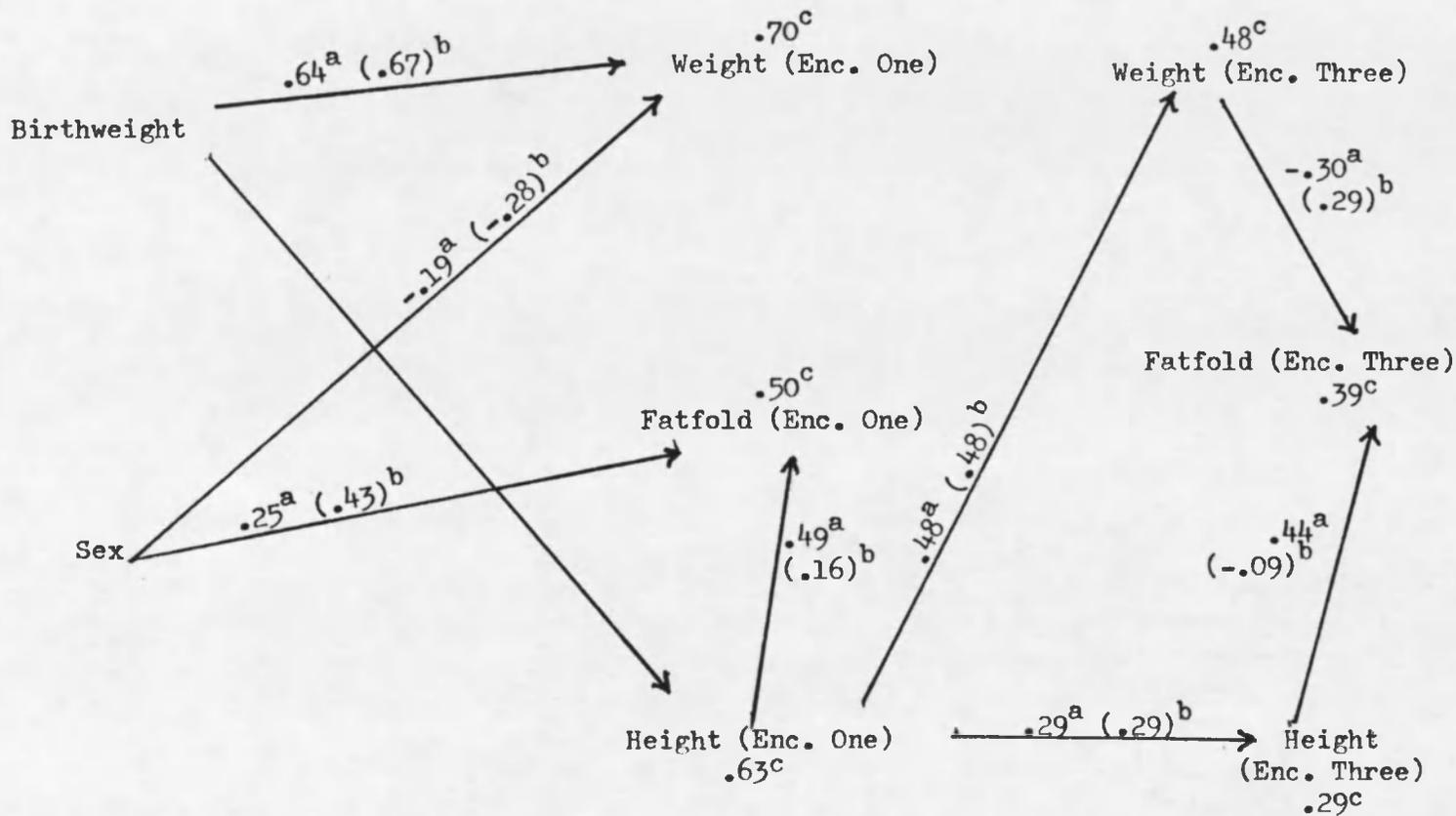


Figure 2 Continued. -- C, Encounter Three.

established between fatfold measurements at encounter three with fatfold measurements at encounter one.

Weight and fatfold measurements at encounter three were not significantly correlated with the same measurements at encounter one. This is consistent with Whitelaw's (1977) finding of fatfold thickness at birth not being correlated to fatfolds at one year in British infants. Weight and height at encounter three were positively correlated (multiple $R=0.39$) with fatfolds at the same encounter. This relationship was expected because by one year, fatter infants tend to also be taller than their thinner counterparts. Even though weight at encounter three had some predictive value for fatfolds at encounter three, it was a poor indicator of fatfold measurements at all three encounters, accounting for approximately 10% of variability in the measurements.

Prediction equations for weight, height and subscapular fatfolds at each encounter were generated from the data.

Birth through two months:

$$\text{Weight} = 1507.4 + 0.8464 (\text{birthweight}) - 348.4 (\text{sex}), R=0.548$$

$$\text{Height} = 45.57 + 0.00296 (\text{birthweight}) - 1.161 (\text{sex}), R=0.444$$

$$\begin{aligned} \text{Subscapular fatfold} &= 1.918 + 0.00415 (\text{weight, enc. one}) \\ &+ 0.00640 (\text{birthweight}), R=0.346 \end{aligned}$$

Five through seven months:

$$\text{Weight} = 5070.3 + 0.6539 (\text{birthweight}), R=0.328$$

$$\begin{aligned} \text{Height} &= 50.08 + 0.1860 (\text{birthweight}) + 0.2076 (\text{height, enc. one}), \\ &R=0.424 \end{aligned}$$

$$\begin{aligned} \text{Subscapular fatfold} &= 5.769 + 0.3144 (\text{fatfold, enc. one}) \\ &- 0.1100 (\text{birthweight}) + 0.3919 (\text{weight, enc. two}), R=0.392 \end{aligned}$$

Eleven through thirteen months:

$$\text{Weight} = 1249.44 + 196.35 (\text{height, enc. one}), R=0.479$$

$$\text{Height} = 54.36 + 0.3986 (\text{height, enc. one}), R=0.293$$

$$\begin{aligned} \text{Subscapular fatfold} &= 11.087 + 0.6910 (\text{weight, enc. three}) \\ &- 1.4301 (\text{height, enc. three}), R=0.390 \end{aligned}$$

From the equations one can predict what the weight at encounter one will be approximately 27% more accurately than random if both birthweight is the only variable affecting weight at the same encounter and by one year, height at encounter one is the sole factor. Birthweight must be known for predicting heights at encounter one and two, with sex contributing negatively to height at encounter one. By encounter three only height at encounter one is significant.

Fatfold measurements at encounter one was only affecting fatfolds at encounter two, birthweight and weight at encounter two was needed for this prediction. By one year weight and height at encounter three were needed to predict fatfolds at the same encounter. Birthweight and weight at encounter one aids in the prediction for encounter one's fatfold.

Dividing the sixty one-year old infants into thin, normal and fat categories and subsequently using the thin and fat infants for analysis proved beneficial. The only significant relationship was between fatfolds at one year. It was interesting to note that bottle feeding compared with breast feeding showed no significant correlation. The bottle-fed babies at one year were no different than the breast-fed babies at the same time.

The study had its limitations for the population was not a typical cross-section of the American population, for the majority of

the subjects resided in Scottsdale, Arizona, an upper-middle class community. The study also utilized only healthy, White, infants who attended a private pediatric clinic.

It was the belief of this author that Dr. Glenn Friedman has had a positive effect on the outcome of the infants. It may be possible that infants who were obese or infants of obese parents may have left his practice, for he incorporates nutrition and exercise education into his practice. This education is not only for his pediatric patients but also their parents.

SUMMARY AND CONCLUSIONS

The success rate for treatment of obesity has not been totally effective, therefore a means of predicting the cases at risk is much needed. This study was designed to determine if in a given population of infants (from birth through one year of age) there was a way to screen the infants that show a potential risk of being obese. This was achieved by weight, height, and subscapular fatfold measurements taken at birth through two months, five through seven months and eleven through thirteen months. The study was homogeneous in the sense that it dealt with only healthy, white, upper-middle class infants.

Path and regression analysis was performed on the following variables: birthweight, sex, weight, height and subscapular fatfold measurements for the three encounters. Sixty-one-year old infants were arbitrarily divided into categories of fat, normal and thin based on subscapular fatfold measurements. Eight fat and eight thin infants were compared using Student's t-test for the following variables:

- a. birthweight
- b. weight at one year
- c. weight gained by one year
- d. percent weight gained by one year
- e. fatfold measurements at one year.

The relationships of feeding types at two months to fatness at one year was shown by the use of chi square.

Several conclusions were drawn from this particular study:

1. There was no predictability of fatfold measurements at one year from that at two months.

2. Fatfolds and weight were significant but not highly correlated at each age; therefore weight was a poor index of adiposity in infants.

3. There was no significant difference between the "fat" and "thin" infants at one year of age with regard to birthweight, weight at one year, weight gain in first year, percent of birth weight gained in the first year, or bottle versus breast feeding.

It was finally concluded that fatness at one year was not necessarily an inevitable consequence of fatness in early infancy.

History of coronary or stroke under age 60

1 = yes, parent
2 = yes, grandparent
3 = no
9 = no data

Diabetes Mother
(IDM)

(51)

1 = yes

LIST OF REFERENCES

- Ademowore, A. S., N. G. Courey and J. S. Kime. (1972) Relationships of Maternal Nutrition and Weight Gain to Newborn Birthweight. *J. Obstet. Gynec.* 39:460.
- Asher, P. (1966) Fat Babies and Fat Children; the Prognosis of Obesity in the Very Young. *Arch. Dis. Child* 41:672.
- Ashwell, M. and J. S. Garrow. (1973) Full and Empty Fat Cells. *Lancet* 2:1036.
- Ashwell, M., P. Priest and M. Bondoux. (1974) Adipose Tissue Cellularity in Obese Women. In *Recent Advances in Obesity Research*: 1, A. Howard, Ed. Technomic Publishing Co., Inc., Westport, Conn., 74 pp.
- Bray, G. A. (1972) The Management of Obesity. *Am. J. Clin. Nutr.*, 23(9):1141.
- Brooke, C. G. D. (1972) Evidence for a Sensitive Period in Adipose Cell Replication in Man. *Lancet* 2:624.
- Brooke, C. G. D., J. K. Lloyd and O. H. Wolf. (1972) Relation Between Age of Onset of Obesity and Size and Number of Adipose Cells. *Brit. Med. J.* 2:25.
- Center for Disease Control. (1972) Ten-State-Nutrition Survey 1968-1970 DHEW Publication No. (HSM) 72-8130, Vol. 1-6.
- Charney, M., H. C. Goodman, M. McBride, B. Lyon and R. Pratt. (1976) Childhood Antecedents of Adult Obesity: Do Chubby Infants Become Obese Adults? *N. Eng. J. Med.* 295:6.
- Court, J. M. (1977) Obesity in Childhood. *Med. J. Aust.* 1:888.
- Creery, R. D. G. (1972) Correspondence: Infantile Overnutrition. *Brit. Med. J.* 4:727.
- Eid, E. E. (1970) Follow-up Study of Physical Growth of Children Who Had Excessive Weight Gain in First Six Months of Life. *Brit. Med. J.* 2:74.
- Food and Nutrition Board. (1978) National Research Council, National Academy of Sciences. Fetal and Infant Nutrition and Susceptibility To Obesity. *Nut. Rev.* 36:122.

- Forbes, G. B. (1977) Nutrition and Growth. *J. Pediatr.* 91:40.
- Garn, S. M. (1976) The Origins of Obesity: Marginal Comments. *Am. J. Dis. Child* 130:465.
- Garn, S. M. and D. C. Clark. (1976) Trends in Fatness and the Origins of Obesity. *Pediatrics* 57:443.
- Greenwood, M. R. C. and P. R. Johnson. (1977) Adipose Tissue Cellularity and Its Relationship to the Development of Obesity in Females. In *Nutritional Disorders of American Women*, M. Winich, ed. John Wiley and Sons, N. Y., 119 pp.
- Hartz, A., E. Giefer and A. A. Rimm. (1977) Relative Importance of the Effect of Family Environment and Heredity on Obesity. *Ann. Hum. Genet.* 41:185.
- Hassell, T. (1974) Health Hazards Pertaining to Obesity. *Cajanus* 7(3):94.
- Hirsch, J. (1975) Cell Number and Size as a Determinant of Subsequent Obesity. In *Childhood Obesity*, Vol. 3, M. Winick, ed. John Wiley and Sons, N. Y. 15 pp.
- Hirsch, J. and P. W. Han. (1969) Cellularity of Rat Adipose Tissue: Effects of Growth, Starvation and Obesity. *J. Lipd. Res.* 10:77.
- Hooper, P. D. and E. L. Alexander. (1971) Infant Morbidity and Obesity. *Practitioner* 207:221.
- Huenemann, R. L. (1974) Environmental Factors Associated with Pre-school Obesity, Part 1 and 2. *J. Am. Diet Assoc.* 64:480.
- Jelliffe, D. B. and E. F. P. Jelliffe. (1975) Fat Babies, Prevalence, Perils and Prevention. *Environ. Child. Health*, June, 124 pp.
- Jelliffe, E. F. P. (1974) Why Are Some Babies Fat? *Cajanus* 7(2):58.
- Johnson, P. R. and J. Hirsch. (1972) Cellularity of Adipose Depots in Six Strains of Genetically Obese Mice. *J. Lipd. Res.* 13:2.
- Knittle, J. L. (1976) Nutrient Requirements in Adolescence. In *Adolescence*, McKigny and Munroe, eds. MIT Press, Cambridge, Mass. 75 pp.
- Lloyd, J. K. (1977) Prognosis of Obesity in Infancy and Childhood. *Postgrad. Med. J. suppl.* (27), 53:11.

- Mellbin, T. and J. Vuille. (1973). Physical Development at Seven Years of Age in Relation to Velocity of Weight Gain in Infancy with Special Reference to Incidence of Overweight. *Brit. J. Prev. Soc. Med.* 27:225.
- Neumann, C. G. and M. Alpaugh. (1976) Birthweight Doubling Time: A Fresh Look. *Pediatrics* 57:469.
- Poskitt, E. M. E. (1976) Dudley Five Years Later: Follow-up Study of Well-nourished Infants. *Arch. Dis. Child* 51:811.
- Ravelli, G., Z. A. Stein and M. W. Susser. (1976) Obesity in Young Men After Famine Exposure in Utero and Early Infancy. *New Eng. J. Med.* 295:349.
- Rayner, P. H. W., A. Shikla, H. A. Forsyth, C. M. Anderson and S. M. Marwah. (1974) Is Childhood Obesity Preventable? In *Recent Advances in Obesity Research: 1*, A. Howard, ed. Technomic Publishing Co., Inc., Westport, Conn. 46 pp.
- Shenker, R. I., V. Fisichelli and J. Lang. (1974) Weight Differences Between Foster Infants of Overweight and Nonoverweight Foster Mothers. *J. Pediatr.* 91:40.
- Shukla, A., H. A. Forsyth, C. M. Anderson and S. M. Marwah. (1972) Infantile Overnutrition in the First Year of Life: A Field Study in Dudley, Worcestershire. *Brit. Med. J.* 4:507.
- Sveger, T., T. Lindberg, B. Weibull and U. L. Olsson. (1975) Nutrition, Overnutrition, and Obesity in the First Year of Life in Malmo, Sweden. *Acta Pediatr. Scand.* 64:635.
- Taitz, L. S. (1971) Infantile Overnutrition Among Artificially Fed Infants in the Sheffield Region. *Brit. Med. J.* 1:315.
- Thomson, J. (1955) Observations on Weight Gain in Infants. *Arch. Dis. Child* 30:322.
- Udall, J. N., G. G. Harrison, G. Morrow III, P. D. Walson and Y. Vaucher. (In press) The Interaction of Maternal and Neonatal Obesity. *Pediatrics*.
- Whitelaw, A. G. L. (1976) Influence of Maternal Obesity in Subcutaneous Fat in the Newborn. *Brit. Med. J.* 1:985.
- _____. (1977) Infant Feeding and Subcutaneous Fat at Birth and at One Year. *Lancet* 2:1098.
- Wright, S. (1921) Correlation and Causation. *J. Ag. Res.* 20:557.

