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Child growth, nutrition and chronic disease risk in adults

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INTRODUCTION

First, I would like to give a quick introduction to some principles in child growth. Child growth consists of a predictable pattern of gains in weight, height, fat and fat-free tissue and of maturation of the various organ systems. Chronic undernutrition and disease will reduce body size, slow growth velocity and delay maturation. Growth charts like this show the course of growth. One must remember that there is considerable variability both within populations and among populations in body size and growth velocity but the growth pattern itself is universal.

Growth in height and weight is most rapid in the first year after birth and then again during adolescence but at a somewhat lower velocity—what we refer to as the «pubertal spurt». A great many changes occur during adolescence along with the spurt in height and weight. Boys usually gain in muscle mass and may lose some fat. This is accompanied by an increase in bone density and in cardio-pulmonary function. Girls gain less muscle mass but continuously add fat from 8 to 18 years. Within a population, girls mature earlier than boys. We use various markers of maturation—skeletal maturation, dental maturation and sexual maturation. Adult body size is the final result of the growth of the child. In all populations, adult men are taller than adult women.

Height and health risk

Mortality and Morbidity

We may ask whether tall individuals within a population have a lower disease or mortality risk than shorter ones. Waaler (1984) has done this for Norway. He has linked height and weight measurements taken between 1963 and 1975 of all Norwegians aged 15 years and above with the death registry. He found that all-cause mortality declined with increasing height and this held for all age groups. Taller individuals also had reduced mortality from obstructive lung disease and cardiovascular diseases. Other investigators also have observed lower risk for death from cardiovascular diseases among taller individuals. In one study of Harvard male alumni (Paffenbarger et al, 1966) there was an inverse relation between height and coronary heart disease risk. There are two British studies which reported similar findings. [Table 1] In a case-control study on women carried out in New England hospitals, taller women had lower mortality risk from myocardial infarction than women of average height (Palmer et al, 1990). The reasons for this are still to be determined. Nutrition early in life may play a role as it relates to hypertension and diabetes.

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TABLE 1
Height among cases of myocardial infarction and controls

	Women, New England 1985-88	
Age (YRS)	Height (CM)	Relative Risk
<50	<152	0.8
	160-163	1.0
	>170	0.6
50-59	<152	1.1
	160-163	1.0
	>170	0.6
60-64	<152	1.8
	160-163	1.0
	>170	0.8

Cancer

In striking contrast to the results showing that taller individuals have lower mortality risk for cardiovascular diseases, are opposite results for some cancers. Comparison among populations reveals positive correlation of height with some site-specific cancers. [Table 2]. The contribution of childhood size to later breast cancer and mortality was analyzed (Micozzi, 1987) using growth data from Worldwide Variation in Human Growth by Eveleth and Tanner (1976). Anthropometric measurements for ages 6 to 18 years from 32 populations were correlated with age-adjusted breast cancer mortality rates from the same countries. Significant positive correlations were found between height and weight and breast cancer incidence and mortality rates. Since growth reflects overall nutrient intake, these observations are believed to support the hypothesis of a dietary link for breast cancer risk.

TABLE 2
Correlations of Age-Specific Anthropometric Dimensions with Age-Adjusted Breast Cancer Mortality Rates Among 32 Populations [1]

Age (Years)	Stature	Weight (N)
6	0.49 (23)	0.48 (22)
7	0.38 (29)	0.39 (28)
8	0.56 (29)	0.40 (28)
9	0.56 (29)	0.42 (28)
10	0.53 (31)	0.37 (30)
11	0.45 (30)	0.37 (29)
12	0.57 (30)	0.42 (29)
13	0.65 (28)	0.43 (27)
14	0.62 (28)	0.50 (27)
15	0.75 (26)	0.58 (25)
16	0.77 (24)	0.69 (23)
17	0.74 (21)	0.68 (20)
18	0.72 (15)	0.75 (15)

N= number of paired observations at each age
1 (From Micozzi, 1987)

Blood Pressure

Blood pressure increases during growth and maturation. Both the rates of growth and maturation may influence blood pressure in the young adult. Children who are early maturers will be taller and perhaps heavier than their peers during the growth period (although they are most likely to be shorter adults). In the Philadelphia Blood Pressure Project we found that taller and heavier 7-year-old children had higher systolic and diastolic blood pressures at adolescence than smaller children in the sample (Katz et al 1980). However, blood pressure was more highly correlated with bone age than with chronological age so the association may have been due to maturity differences. In another study in Kentucky not only were similar results obtained but early maturing adolescent males continued to have higher blood pressure as young adults (Kotchen et al, 1989).

Weight and health risk

Overweight and Obesity

The prevalence of adult overweight and obesity has been increasing worldwide, including in some segments of Latin American populations. Certainly some adult obesity reflects environmental conditions during childhood. Childhood obesity also is increasing although I want to make clear that obese children do not necessarily go on to become obese adults.

Nevertheless, an obese child probably should not be considered a healthy child. The association of excess morbidity and mortality with overweight and obesity is well established. The diseases we are concerned with are diabetes, cardiovascular disease, some site-specific cancers, gallbladder disease, gout, and some osteoarthritis. Obesity itself may be considered as a risk factor for many diseases although it may not act independently from the standard risk factors.

The commonly used means to define overweight and obesity are (1) weight-for-height or body mass index (BMI) which is weight/height squared, and (2) skinfold measurements of subcutaneous fat. Even though weight-for-height is better correlated than BMI with total adipose tissue (r=0.967 for men and 0.984 for women) (Sjostrom, 1989), BMI is currently used to predict chronic disease risk.

Blood Pressure

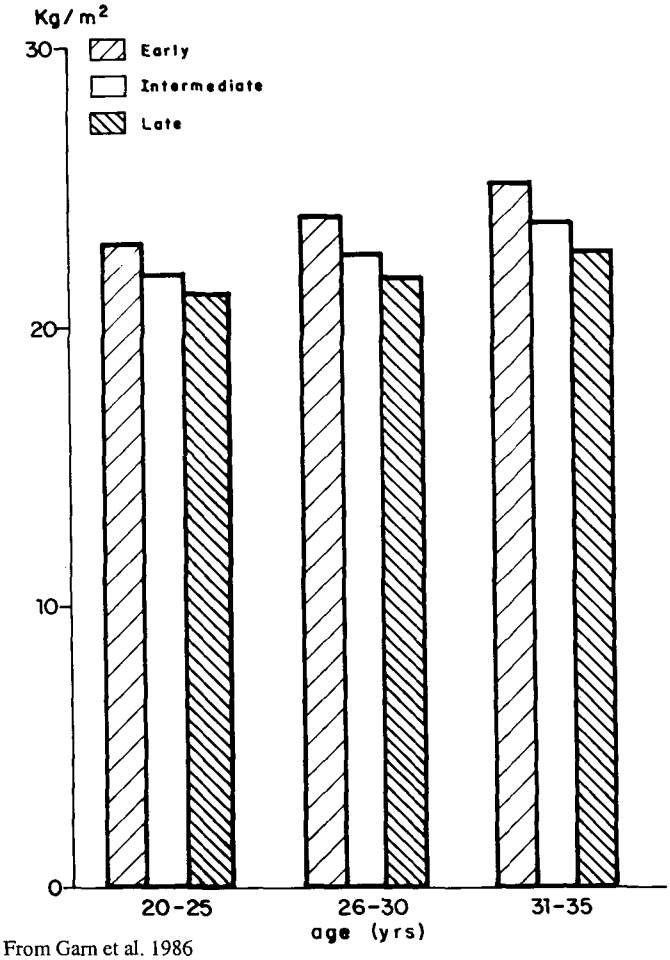
Both BMI and some skinfolds have been reported from numerous studies to be related to elevated blood pressure in adults. In the United States NHANES, adolescents at the highest percentile for subscapular skinfolds had systolic blood pressure elevated over those with less fat (Comoni-Huntley et al, 1979). Moreover, weight loss has been seen to lower blood pressure while weight gain is related to changes in the standard cardiovascular risk factors: serum cholesterol, blood glucose, blood pressure, serum uric acid.

Atherosclerotic disease is now recognized to begin in childhood and increased levels of risk factors continue from childhood to adulthood. Adolescents who have higher blood pressure may be at a greater risk of being hypertensive as young adults since it has been shown that blood pressure tracks throughout growth (Martin-Bothig et al, 1989; Katz et al, 1980; Kochen et al, 1989). Many lifestyle changes occur in young adults and can contribute to future risk in middle age.

Cancer

Overweight and obesity have been associated with some types of cancer, especially breast, endometrial, prostate and colon. Maturational timing is a factor both for fatness and for breast cancer risk. Girls who have early menarche are at a greater risk for breast cancer. In fact, it has been estimated that breast cancer risk is reduced 10-20% for each year menarche is delayed (Henderson et al, 1991). We see an interaction between maturation and weight. Children and adolescents who are overweight and obese tend to be more advanced in maturity (Beunen et al, 1994; Garn et al, 1986)). Even young adults who were early maturers are fatter than those who were late maturers. This along with earlier rise in estrogens may explain why advanced maturers have an increased risk for breast cancer. Again the early influence on disease later in life is demonstrated.

FIGURE 1
BMI Differences
NCPP Survey



From Garn et al. 1986

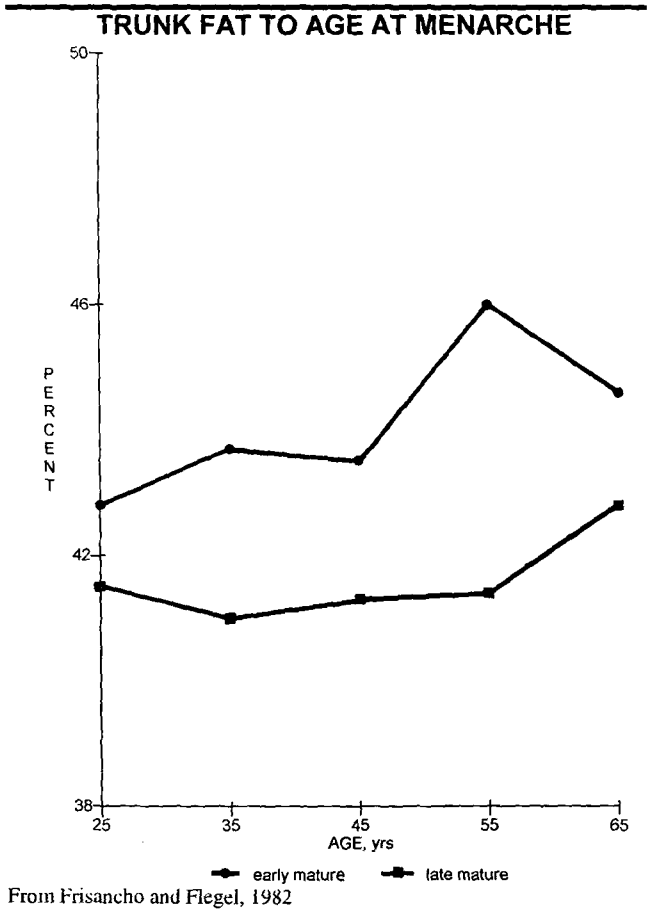
Fat patterning

Overweight and obesity determined by BMI are well-established risk factors for noninsulin dependent diabetes. Of greater predictive value, however, is how fat is distributed in the body. Epidemiological studies have shown that fat deposited on the trunk or upper body (centralized) is a strong risk factor (Kissebah et al, 1988; Mueller et al 1984), not only for diabetes but for coronary heart disease (Larsson, 1988). There is a wealth of evidence that upper body fatness is causally associated with altered glucose metabolism.

It has been suggested that the effect of fat distribution on diabetes is greater in women than men (Haffner, 1989). As I have pointed out, within populations females have more fat than males. Girls and boys are quite similar in the amount of subcutaneous fat they have until just before puberty. At puberty fat in girls increases and in boys may either decrease or remain level throughout puberty. Fat patterns appear to change in adolescence, moving from a more peripheral (fat on extremities) to a centralized pattern (fat on trunk). Thus, in contrast to some other growth parameters, fat patterns in adolescence cannot be reliably predicted from childhood patterns (Baumgartner & Roche, 1988).

Advanced maturation seems to be related to a more centralized fat pattern, implying more risk. The NHANES I data showed that adolescents who were advanced maturers (estimated by bone age and menarche) were likely to have more fat on the trunk than on the limbs at adult ages (Frisancho & Flegel, 1982). In adolescent-onset obesity the fat distribution appears to be truncal or centralized.

FIGURE 2
Trunk fat to age at menarche



From Frisancho and Flegel, 1982

Abdominal fat may be a more important risk factor for some chronic diseases than subcutaneous fat. The waist to hip circumference ratio is used to estimate abdominal fat. It is significantly correlated with the ratio of intra-abdominal to extra-abdominal fat measured by CAT scans (Ashwell et al, 1985). However, the waist and hip circumferences are not usually measured in growth studies because they are not especially meaningful for growth. In children they appear to be less exact than skinfolds to describe fat distribution (Mueller et al,1989).

CONCLUSIONS

Early child environment and growth are important factors in determining health in the adult years. Questions remain as to whether tallness (which we assume reflects good nutrition during growth) will lead to fewer chronic diseases in adults. Although it is associated with lower all-cause mortality and lower cardiovascular mortality, it may be acting disadvantageously for some adult cancers. Overweight and obesity which may reflect excess nutrition during childhood and adolescence is strongly linked with many chronic diseases in adults.

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