International Center for Research on Women
1717 Massachusetts Avenue, N.W., Suite 302
Washington, DC 20036

NUTRITION OF ADOLESCENT GIRLS RESEARCH PROGRAM

• *Research Report Series No. 3* •

# Nutrition, Health and Growth in Guatemalan Adolescents

December 1994

by

Reynaldo Martorell
Juan Rivera
Paul Melgar

INCAP Publication DCI/015

This publication was made possible through support provided by the Office of Nutrition, Bureau for Research and Development, U.S. Agency for International Development, under the terms of Cooperative Agreement No. DAN-5117-A-00-0087-00. The opinions expressed herein are those of the author(s) and do not necessarily reflect the views of ICRW or the U.S. Agency for International Development.

*Authors' Affiliations*

Reynaldo Martorell
Emory University School of Public Health
1599 Clifton Road, NE
Atlanta, Georgia 30329
(404) 727-9854

Juan Rivera
National Institute of Public Health
Cuernavaca, Morelos, 62508 Mexico
Tel: 52-73 11 01 11
Fax: 52-73 11 22 19

Paul Melgar
Institute of Nutrition
of Central America and Panama
Calzada Roosevelt Zona 11
Apartado Postal 1188
Guatemala City, Guatemala
tel: 502-72 37 62
fax: 502-73 65 29

For more information, please contact:

International Center for Research on Women
1717 Massachusetts Avenue NW, Suite 302
Washington, DC 20036
Tel: (202) 797-0007
Fax: (202) 797-0020
Email: icrw@igc.apc.org

# CONTENTS

## Executive Summary

In this project longitudinal data for 1 year on over 1000 girls and young women from four Guatemalan villages were collected on body size and composition, dietary patterns, physical activity, illness and health histories, fertility histories and education and occupation. In addition, data already available on some of the subjects were analyzed. The study was envisioned to achieve the following aims:

- Describe patterns of growth and development from birth to adulthood in Guatemalan females.

- Test specific hypotheses about the dependency of growth at various stages of life, including adolescence, on health, nutrition and socioeconomic status.

- Provide guidance useful for the formulation of policies and programs that seek to improve the nutritional status of adolescent girls.

The tasks proposed included a literature review of growth during adolescence and analyses and interpretation of the data collected in Guatemala. The key conclusions reached for these tasks are given below.

The purpose of the literature review was to assess the extent to which stunting, a phenomenon of early childhood, can be reversed in later childhood and adolescence. The focus was on data from developing countries. The conclusions of the review were as follows:

- Maturational delays in developing countries are usually less than two years, only enough to compensate for a small fraction of the growth retardation of early childhood.

- Follow-up studies find that subjects who remain in the setting in which they became stunted experience little or no catch-up in growth later in life.

- Improvements in living conditions, as through food supplementation or through adoption, trigger catch-up growth but do so more effectively in the very young.

- One study cautions that in older adopted subjects, accelerated growth may accelerate maturation, shorten the growth period and lead to short adult stature.

The key overall conclusion of the review was that the potential for catch-up growth was found to increase as maturation is delayed and the growth period is prolonged. Since delays in maturation in developing countries are often moderate (i.e. delays of 2 years or less), the actual amount of catch-up growth taking place through this mechanism is limited. In most settings, the growth failure of early childhood is reflected in reduced stature among adults.

The research results from the study lead to the following profile of health and nutrition in rural Latino Guatemalan adolescents:

- Pregnancy and the first 2-3 years of life appear to be the only periods in life when physical growth is retarded significantly. During adolescence, the timing of peak height velocity occurs a year or so later, but otherwise growth in height appears to be as in well-nourished children. Growth during adolescence can never be characterized as a period of significant growth failure.

- Menarche occurs about a year later, confirming a moderate delay in maturation. However, supplementation in early childhood does not impact on menarche.

- Adolescents are short (stunted) but this is a carry over from the retardation which occurred in early childhood and is not the result of poor growth during adolescence.

- Adolescents have adequate body mass indices (BMIs) and satisfactory skinfold values. Specifically, these are somewhat lower than observed in American subjects, judged by many to be fat.

- Dietary intakes are clearly adequate in terms of protein. In terms of energy, intakes are satisfactory if judged in terms of requirements per kilogram of body weight. Recognizing the deficiencies of dietary surveys, it is concluded that intakes of riboflavin and calcium may be low. Iron intakes appear to be adequate but bioavailability is poor (about 75% of the calories come from carbohydrates) and needs at the metabolic level may not be met. Still, anemia levels are low.

- Physical activity data indicate a moderate level of exertion for the typical adolescent.

- Most adolescents are healthy and only a few are occasionally sick enough that they need to stay in bed.

- Analyses reveal that diet, health and socioeconomic status (SES) are not predictive of growth in height during adolescence. This suggests unconstrained growth. On the other hand, energy intakes from the home diet did appear to be related to weight gain.

Thus, the picture that emerges is one of largely good health, adequate nutrition and satisfactory growth during adolescence. The effects of poor nutrition and health in early childhood are reflected however, in terms of short stature, a risk factor for delivery complications. It is not known whether nutrition interventions just prior or during adolescence would increase stature but our data would make this seem unlikely. Growth during adolescence, as noted above, is already as good as in well-nourished subjects. Neither diet, health or SES were related to growth in height during adolescence, suggesting unconstrained growth. On the other hand, supplementation during adolescence in at least one study of adopted Indian girls in Sweden indicates that maturation may be accelerated and that this in turn may affect final stature adversely. The other possibility is that weight and fatness levels may be increased, desirable only in thin adolescents.

It would be inappropriate to pursue programs aimed at redressing the effects of early childhood malnutrition through interventions in adolescence. These actions may not be effective (i.e. it may be impossible to reverse these effects) and may in fact be harmful in some settings (i.e. accelerate maturation); another drawback is that funds and resources may be diverted from more needy programs in early childhood. Clearly, more research is desirable on the issue of the value of nutrition interventions in adolescents.

This is not to say that nutrition programs should not be aimed at adolescents. Where thinness is documented, or where micronutrient deficiencies are known to exist, appropriate remedial measures need to be implemented. School age and adolescence are also opportune periods for nutrition and health education, both through the formal sector and outside of it.

# 1. Introduction

In this project, longitudinal data for 1 year on over a 1000 girls and young women from four Guatemalan villages (Table 1.1) were collected on body size and composition, dietary patterns, physical activity, illness and health histories, fertility histories and education and occupation. In addition, data already available on some of the subjects were analyzed. The study was envisioned to achieve the following aims:

- Describe patterns of growth and development from birth to adulthood in Guatemalan females.
- Test specific hypotheses about the dependency of growth at various stages of life, including adolescence, on health, nutrition and socioeconomic status.
- Provide guidance useful for the formulation of policies and programs that seek to improve the nutritional status of adolescent girls.

The villages studied are located in the Eastern part of Guatemala in an area inhabited by "Latinos" (mixed Spanish/Indian ancestry and Spanish speaking). The subjects studied were girls born between 1969 and 1983. In 1993 their ages ranged between 9 and 23 years (Table 1.1). Almost 80% of the subjects were single (Table 1.2) and about 85% had received at least some schooling (Table 1.3). Most of the subjects either worked at home in domestic chores (52.5%) or were still in school (38.0%); only a few were employed in factories (5.3%) or in agriculture (1%). About 13% of the sample had one child, 10% had two, and 6.5% had 3 or more children.

The expectation driving the hypotheses of the study is that adolescents in rural Latino Guatemala exhibit normal growth, consume adequate diets and are generally in good health. This expectation, as will be seen later, was indeed fulfilled. At the same time, these subjects exhibit the sequelae of failure to grow and of malnutrition in early childhood. The most important policy implication is that adolescence is not a time of nutritional stress as compared to early childhood. It remains to be demonstrated whether interventions aimed at adolescents can redress some of the effects produced by malnutrition in early childhood.

Information is presented in three chapters. Chapter 2 is a literature review of adolescent growth and Chapter 3 deals with the findings on body size and composition. The longitudinal data on growth, diet, physical activity and health are presented in chapter 4.

## 2. Review of Literature

The main purposes of the review[1] were (1) to assess the degree to which the severe, linear growth failure that commonly occurs in early childhood can be redressed through catch-up growth; and (2) to identify the epidemiological factors or ecological settings which predict the degree of catch-up growth. The review was exemplary and focused on epidemiological data from developing countries. Follow-up studies were examined in greater detail, in particular studies that measured the same subjects in early childhood and later in adolescence or adulthood. Three unique settings were examined: (1) continued residence in the environment that gave rise to stunting; (2) continued residence in the same environment but with improvements in nutrition; and (3) relocation to an improved environment.

The review was not exhaustive on the subject of catch-up growth. Important omissions were studies of the growth response following treatment of such illnesses as celiac disease or hormonal deficiencies (e.g., growth hormone, hypothyroidism). Though this omitted literature is vast, many are reports of selected cases, with statistical descriptions of defined populations being rare. It is likely that this had led to a bias towards the publication of cases with spectacular or more complete catch-up growth than commonly is the case. In commenting on this literature, Tanner (1981) observed that the potential for catch-up diminishes as a function of the severity, duration and timing (i.e., earlier in life limits potential) of the growth retarding influence.

The studies reviewed used a variety of approaches to assess the degree to which catch-up growth had taken place. Some used an internal comparison; for example, contrasting the stature of adults grouped according to their degree of stunting at 5 years of age. In other cases, heights during childhood, adolescence and young adulthood were directly compared to values from an external reference population, usually from the United

---

[1]This literature review was prepared under subagreement with ICRW (#IC-75/03) and has been published as Martorell R., Kettel Khan L., and Schroeder DG. Reversibility of Stunting: Epidemiological findings in children from developing countries. *European Journal of Clinical Nutrition*, 1994, 48 (suppl. 1): S45- S57.

States. This latter approach is of some concern. Whereas growth potential in early childhood is surprisingly similar across ethnic groups (Martorell, 1985), little is known about the importance of ethnic or genetic differences in growth during adolescence. Thus, inferences about catch-up growth from analyses which use an external reference population to assess growth during adolescence need to be made with caution.[2]

**The Timing of Stunting**

Stunting is a phenomenon of early childhood and a direct result of poor diets and infection (Martorell and Habicht, 1986). Though the prevalence of low birthweight is increased in poor societies, newborn lengths are often similar to well-to-do samples. The intense period of growth retardation is generally between 3 to 12 or 18 months. In some countries, growth retardation continues into the third year or longer but to a lesser extent. At the end of this process, marked departure from normality will have often occurred. Median regional prevalence of stunting, defined as children with lengths two or more standard deviations below the WHO/NCHS median, in children 12-23 months of age are 41% for Africa, 34% for Latin America, 47% for Asia and 39% for the Eastern Mediterranean (Victora, 1992).

There are multiple reasons why stunting occurs in early childhood and not later. In childhood, nutritional needs are greatest, in relation to weight, than at any time later. One of the reasons that nutritional requirements are high is that growth velocities are the greatest they will ever be, including peak height velocity in adolescence. Thus, the opportunity for growth retardation is great in early childhood partly because more growth is taking place. Because of high nutritional requirements and limited gastric capacity, energy and nutrient dense foods are required to complement breast milk during weaning, yet

---

2 Where appropriate, we specify which reference population is used. The "WHO/NCHS" reference population is used worldwide to assess the growth of pre-pubescent children (Hamill et al., 1979). Another, identified simply as "US reference values", is needed because curves for adolescents and adults are not included in the WHO/NCHS reference. It should be noted that these two reference populations are distinct despite the fact that both use NCHS data. The WHO/NCHS is a combination of Fels Research Institute data with various NCHS surveys, including NHANES I but not NHANES II. The US reference curves published by Frisancho (1990) include only NHANES I and II. Percentile values for prepubescent children are very similar in both references.

caretakers in developing countries often serve foods that are too bulky for young children. In addition, infections, particularly gastrointestinal ones, limit growth in young children because episodes are more frequent and more severe in the very young, especially the malnourished. Finally, young children are totally dependent on others for their care and are hence most vulnerable to poor caretaking. Older children can make their needs known more easily and are better able to take the initiative to procure more food.

**Age at Menarche**

Maturation can be assessed through a variety of approaches including skeletal age, appearance of secondary sexual characteristics and, for females, menarche. However, the majority of studies of adolescents, such as the ones reviewed here, only report menarche.

Tanner (1981) has summarized the available data for average age at menarche in 19th century Europe for several groupings of countries. Average ages at menarche in the United Kingdom, Scandinavia, Germany and Russia were between 15.0 and 16.8 years in working women and between 14.3 and 15.0 years in middle class women. For France, Spain and Italy, corresponding ranges were 14.5 to 15.4 years for working class samples and 13.5 to 14.3 years for middle class samples. The most complete overview of age at menarche for modern populations is given in Eveleth and Tanner (1990). The range given for European samples (all social classes) collected since 1970 is 12.1 to 13.5 years, indicating a decline of several years with respect to 19th century data. In Oslo, where serial data are most complete, the mean menarcheal age declined from 15.6 years in 1860 to 13.2 in 1960. Recent values for the US are 12.8 years for whites and 12.5 for Blacks.

Eveleth and Tanner (1990) also provide menarche data for various countries and regions of the world. For African samples since 1970, values range from 13.1 to 14.5 years. Values for India are 12.5 to 14.6 years while for Latin American countries they are 12.0 to 13.4 years, except in a sample of Oaxacan Indians from Mexico where mean age at menarche was 14.3. The data available are incomplete but they do suggest that environment has a greater effect on maturation than ancestry does.

For purposes of this review, samples with mean ages at menarche of 15 years or higher are assumed to have had marked maturational delays whereas those between 13.5

and 14.9 are assumed to have had mild to moderate delays.

**Continued Residence in the Environment That Gave Rise to Stunting**

The first situation to be considered is one in which subjects continue to live in the environment in which they became stunted. In this instance, two types of populations may be considered, those in more extreme settings, in which puberty and/or menarche are markedly delayed (i.e., age at menarche of 15 years or more) and those in populations more typical of agricultural societies in developing countries, where delays in menarche and/or maturation are less marked.

*Populations From Developing Countries Where Delays in Menarche/Maturation are Presumed to be Marked*

Substantial catch-up growth is possible in unusual circumstances where menarche and maturation are very delayed. A study of nineteenth century American slaves by Steckel (1987), and of Turkana pastoralists by Little, Galvin and Mugambi (1983) demonstrate this phenomenon. A possible exception is an account of the Bundi of Papua New Guinea by Malcolm (1970). In each of these populations, there is marked stunting in early childhood compared to US reference values as well as a pronounced maturation delay.

The American Black slaves described by Steckel (1987) were approximately 15 cm less than the NCHS reference mean prior to puberty. The initial differences of about 15 cm were reduced to about 5 cm at the end of the growth period. Nutritional status and prevalence of illness data are not available for this sample population, however, other reports note that poor prenatal care, early weaning, inadequate and contaminated food supplements, and a heavy disease burden hampered growth in early childhood (Swados, 1942; Postell, 1951; Kiple and King, 1977). However, the diets were deliberately improved when the slaves entered the labor force at 8 to 12 years of age. Still, age at menarche appears to have been markedly delayed; Trussel and Steckel (1978) report values of 15 years for some US slave populations. Another indication of delayed maturation is the observation that growth seems to have continued until about age 21 years in males and 19 in females.

In the American Black slaves, a prolonged growth period appeared to provide an opportunity for marked catch-up in growth. Differences in growth potential with respect to the reference population are not a likely explanation. American Blacks today attain adult heights that are similar to those of US Whites.

Little, Galvin and Mugambi (1983) examined Turkana pastoralists in Kenya who are tall, lean people. The basic diet of milk, meat and blood is derived from their cattle; however, hungry periods are common. The degree of pre-pubescent growth retardation is initially about 10 cm but increases to about 15 cm by age 13 years in both sexes. Final achieved stature in Turkana pastoralists is nearly the same as in the US reference population, suggesting nearly complete catch-up. An explanation for this is that age at menarche was 15 years, delayed compared to well-off populations, permitting a longer growth period. An alternative explanation for the apparent catch-up growth is that growth potential in the Turkana and other Nilotic peoples differs from that of the US population and what might be interpreted as catch-up growth during adolescence may, in fact, be an expression of genetic potential. If this were the case, a Turkana adult population which did not experience early childhood growth retardation would be significantly taller than the US population.

The Bundi subsist on sweet potatoes and the diet has a very low fat content; both energy and protein are probably limiting in young children and perhaps at older ages as well (Malcolm, 1970). Growth is extremely retarded, with the differences with respect to the NCHS reference mean increasing progressively with age, until reaching a high of 37.7 cm in girls at 12 years of age and of 40.8 cm in boys at 14 years of age. Differences with respect to US reference means diminish later in adolescence and in adults are 20.8 cm in males and 15.4 cm in females. Maturation is markedly delayed based on skeletal age and sexual maturation assessments. Age at menarche, for example, occurs at 18 to 19 years of age. Peak height velocity is delayed by about 2 years in comparison to British children and growth continues into the early twenties. The prolongation of growth provides an opportunity for catch-up growth but not as much seems to be achieved through this mechanism as in the two populations discussed above (i.e., the growth failure of early childhood is not made up by later growth). However, growth potential during adolescence may be less for the Bundi than for US populations (Malcolm, 1970); this would underestimate the extent of catch-up growth.

### *Populations From Developing Countries Where Delays in Menarche/Maturation are Presumed to be Moderate/Minor*

The circumstances described above are unusual and are not characteristic of what is generally observed in developing countries. Marked to moderate stunting in early childhood, with limited delays in menarche and other maturation indicators, is a more common pattern.

**Cross-sectional studies illustrating common patterns of growth retardation.** The diet of rural Embu Kenyans is based on cereals and tubers and energy and multiple micronutrient are probably deficient (Neumann, Bwibo & Sigman, 1992). Retardation in growth is pronounced at an early age. At 18 months of age, the average height for age *z* score (with reference to the WHO/NCHS reference data) was 2.0, decreasing to about -1.8 at 84 months (values similar for boys and girls). As adults, only a minor degree of catch-up growth seemed to have taken place; average *z* scores (with reference to the 1959 Metropolitan Life Insurance Data) were -1.5 in men and -1.4 in women. In other words, the retardation of early childhood growth appears to persist into adulthood with only modest attenuation.

In a rural population from Malawi, linear growth retardation increased throughout the first 60 months of age (Pelletier, Low and Msukwa, 1991). This is unusual; in most studies, differences are constant from about 24 months. In adulthood, there is a slight increase in the average differences in height with respect to the US age-specific mean compared to those seen at 60 months of age in males but a slight decrease in females. Menarche data were not reported in this or the Kenyan study. According to Eveleth and Tanner, age at menarche in African samples ranges from 13.1 to 14.5 years.

Cross sectional data from rural Guatemala, where menarche occurs approximately one year later (13.5 years) than in well-off populations (Khan et al., in press), show that prior growth retardation is not recuperated later. The difference in height at 11 years of age with respect to the US reference population is nearly the same as that observed at 3 years of age. Similarly, differences remain relatively constant from 11 years to adulthood;

the increasing differences in males at ages 12-15 years are probably caused by delayed timing of the adolescent growth spurt in the Guatemalan sample. It remains unclear whether growth potential during adolescence of Mexicans and Guatemalans is the same as that of the US population (Martorell *et al.*, 1989). If data from the Hispanic Health and Nutrition Examination Survey (NHANES) are used for comparison instead, the difference at the end of adolescence in Guatemalans becomes 8-9 cm instead of 13-14 cm. Since there are no differences between NHANES and the US reference population prior to 11 years, this would suggest modest catch-up in growth.

The use of cross-sectional studies to infer catch-up growth, as done above, assumes that all cohorts suffered the same degree of stunting in early childhood. This may not be the case in populations undergoing significant changes in the quality of life. For example, Mexican-Americans have increased in height dramatically in the last 20 years, probably as a result of better growth in early childhood. The smaller size of Mexican-American adolescents with respect to the reference population may reflect, not reduced growth during adolescence per se, but poorer growth in early childhood than experienced by younger cohorts. Follow-up studies of individuals initially measured in childhood would therefore appear to be better in design.

**Follow-up studies.** Satyanarayana and colleagues conducted a longitudinal study in children from 26 villages near Hyderabad, India. Heights on 197 girls at 5 years and at 18 years of age were analyzed (Satyanarayana *et al.* 1981). Subjects were divided into four groups according to the degree of stunting at 5 years of age as follows: I (> -2.0 SD), II (-2.0 to -3.0 SD), III (-3.0 to -4.0 SD), and IV (< -4.0 SD)) using the Harvard reference data (Reed and Stuart, 1959). The height differential of 14 cm seen at age five years between the most retarded group (90.1 cm) and the least retarded group (104.3 cm) was reduced to 7.7 cm by 18 years, 148.2 and 155.9 cm, respectively. In all groups, the differences with respect to the Harvard reference mean were less at 18 years than at 5 years, particularly in groups III and IV. The authors propose that the most plausible explanation for the apparent catch-up growth is delayed menarche (Satyanarayana *et al.*, 1981). The authors state that the pubertal growth spurt was delayed and that the growth period was prolonged about 1½ years, though no information on age at menarche or incremental growth is given.

In the same population, Satyanarayana, Nadamuni Naidu and Narasinga Rao (1980) examined the growth pattern of 667 Indian boys. The boys were classified in the same fashion as the study of girls. The height differential of 16.5 cm seen at age five years between the most retarded group (88.5 cm) and the least retarded group (105.0 cm) was only slightly improved (15.5 cm) by 18 years, 149.0 and 164.5 cm, respectively. The boys who were shortest (Group IV) at age 5 continued to be so at age 17 years. The most retarded group (< -4 SD) had a lower peak height velocity (6.9 cm/year) than British boys (7.3 cm/year) and its timing was delayed (14 versus 16 years). In spite of the maturation delay, the differences with respect to the Harvard reference data increased slightly from 5 to 18 years. In summary, the growth retardation of early childhood was slightly increased by adulthood in males but decreased in females.

In a later analysis of boys from the same population, Satyanarayana *et al.* (1989) described the dynamics of growth during puberty through 19 to 24 years using a Preece and Baines model. Subjects who were previously measured biannually throughout their pre-school years during 1965-1969 where measured again annually from 1976 to 1984 (n=323). The boys were classified according to the degree of stunting in childhood. Three groups (not four as in earlier studies) were formed: I (> -2.0 SD), II (-2.0 to -4.0 SD), and III (< -4.0 SD). Adult height was defined as height at 18 years or older (range was 18 to 24 years). The gain in height from 5 to 18 years exceeded that of British children only in the case of the most stunted group. Growth after 5 years was related to the degree of stunting such that the differences between groups I and III was reduced from 14.4 cm at 5 years to 9.7 cm at 18 years or older, suggesting a moderate degree of catch-up growth.

The parameters of the Preece-Baines model were examined. Age at take off is only slightly later in the most stunted subjects compared to the rest of the Indian sample and to British children (11.3 years vs. 10.6/10.7 years). The primary factor leading to the short adult stature in this population is the growth retardation already present at take-off. During adolescence, all Indian groups grew more during adolescence compared to British children, with the most stunted groups growing slightly more than the least stunted Indian group. The additional growth is apparently achieved, not by greater peak height velocity, but by a lengthening of the puberty growth period which is 1.3 years longer for the most stunted

Indian children with respect to British children.

Data from Guatemala indicate that there is no catch-up growth during later childhood and adolescence (Martorell, Rivera and Kaplowitz, 1990). As in the Indian studies, subjects were grouped by the degree of stunting at 5 years of age: I (>-2.0 SD), II (-2 SD to -3 SD) and III (<-3 SD) using US reference values. Growth from 5 years to adulthood was similar in all three groups such that the differences among groups were similar in adulthood as at 5 years of age. The relative ranking of groups was maintained (5 years vs. adulthood) and the differences with respect to the reference data were increased by about the same amount in all three groups.

**Continued Residence With Improvements in Nutrition**

If nutritional deficits and infections are the primary causes of stunting in early childhood as stated earlier, improvements in nutrition to avert or reverse stunting should be most efficacious at the ages that these conditions are most extreme. Using data from a longitudinal nutritional supplementation trial conducted in Guatemala, an analysis of the age differences in the impact of supplementation on length gain strongly supports this hypothesis.

In the original study, nearly 2400 children between birth and seven years of age (and their mothers) received either a high energy, high protein supplement (Atole) or low energy, no protein supplement (Fresco) between 1969 and 1977 (Martorell *et al.* 1982). For this analysis, annual growth, supplement, illness, and home diet data were summarized for the first seven years of life. A series of multiple linear regression models were then used to examine the impact of supplement intake on length change while controlling for body size, percent of time with diarrhea, home diet, socioeconomic status, and gender (Schroeder *et al.*, in press).

During the first year of life, each 100 kcals/day of supplement was associated with approximately 9 mm in additional length gain. The impact of this amount of supplement decreased to 5 mm and 4 mm in years two and three, respectively; nutritional supplementation had no significant impact on linear growth after age three.

These findings are consistent with other studies that have examined the impact of supplement by age. In India, 1-2 year old children who were fed approximately 170 kcal/day of supplement in the form of a sweet cake grew 2.8 cm more over 14 months compared to unsupplemented children of the same age (Gopalan *et al.* 1973). This difference was decreased to 1.7 cm in 2-3 and 3-4 years olds and only 1.1 cm for 4-5 year olds. An effect was likely seen in the 4-5 year olds in India because this population was more severely malnourished compared to the Guatemalan population described above, where no effect was found at these older ages.

The hypothesis that dietary and illness patterns determine whether nutritional improvements can avert or reverse stunting is corroborated by work from Colombia. Comparing supplemented and unsupplemented children, Lutter *et al.* (1990) found that responsiveness of length to supplementary feed was greatest during the weaning period (3-6 months of age) and the peak prevalence of diarrhea (9-12 months of age).

**Relocation From the Environment That Gave Rise to Stunting**

The third group of studies to be considered involve an abrupt improvement in living conditions through migration or adoption.

*Children Who Migrated to Developed Countries*

Changes from 1986 to 1989 in the prevalence of low length or height for age, defined as values below the 5th percentile of the WHO/NCHS curves, were investigated using data collected by the Pediatric Nutrition Surveillance System of the Centers for Disease Control from 28,725 Southeast Asian refugees and 2,173,644 poor children under 5 years of age who enrolled in public health clinics from 12 US states (Yip, Scanlon and Trowbridge, 1992). There was a progressive and significant decline in the prevalence of low length-for-age in Southeast Asian but not in poor US children. In Asian children 1 to 23 months of age, the prevalence declined from 24.8% in 1982 to 13.5% in 1989, a relative reduction of more than 46%. A similar relative decline (50%) was observed in low height-for-age older children (24 to 60 months). Standardized height-for-age $z$ scores were then used to determine whether the reductions reflected improvements in a sub-population of short children or in the general population. The distribution of height $z$ scores for three periods over ten years had similar variance and shape implying the later. Each successive cohort became taller suggesting that the steady improvement in the quality of life of the Southeast Asian population was the most likely explanatory factor.

Schumacher, Pawson and Kretchmer (1987) assessed the impact of migration and change in nutrition on the growth of 835 children 6 to 12 years of age of Hispanic, Chinese, Southeast Asian, and Filipino descent enrolled in three 'newcomer schools' in the San Francisco Bay area between December 1982 and June 1985. All birth cohorts were short upon arrival; most ethnic groups were between the fifth and twenty-fifth percentile of WHO/NCHS reference values. The authors note that most cohorts exhibited a median growth velocity that was similar to or exceeded the US median. However, a sense of the magnitude of this accelerated growth is not given, limiting the potential value of the study.

*Children Adopted into Developed Countries*

Winick, Meyer and Harris (1975) and Lien, Meyer and Winick (1977) examined whether adoption of previously malnourished Korean children into US families results in improved development. Subjects were classified according to height on admission to the Holt Adoption Service using Korean growth standards (Hong, 1970). The first study included initial measurements prior to 2 years of age (early adoption) and follow-up at 6 to 8 years; the second study used measurements from 2 to 5 years of age (late adoption) compared to measurements when the children were 7 to 9 years. The ranking of the groups remained at follow-up. However, mean percentiles for height-for-age were greater for all groups at follow-up, suggesting catch-up growth relative to their status prior to adoption and to the Korean reference data. Groups who were younger at adoption improved the most (i.e., study 1 vs. study 2). The authors note that all three groups were below the 50th percentile of the Harvard curves. This suggests incomplete catch-up growth.

In two separate studies, one longitudinal and the other retrospective, Proos and colleagues examined the growth of Indian children from underprivileged circumstances who were adopted by Swedish families.

The longitudinal study by Proos *et al.* (1992), which followed 46 boys and 68 girls for 2 years after arrival, illustrates the remarkable potential for catch-up growth if children are moved from a deprived to an adequate environment at a very young age. Children were 3 to 72 months of age at arrival in Sweden (mean = 15.2 months); with 62% younger than 12 months and more than 90% less than 36 months of age. The prevalence of stunting (< -2 SD below the WHO/NCHS median) decreased from 54% to 5% after 2 years of follow-up. The mean length-for-age *z* scores were -2.2 upon adoption but rose to -0.7 SD 18 months after adoption. In those classified as stunted at adoption, the mean *z* score declined from -3.2 SD to -1.0 SD and in those who were not stunted on adoption, the decline was from -1.1 SD to -0.31 SD after 2 years.

The retrospective study examined 107 Indian girls from the Adoption Centre, Stockholm (Proos *et al*, 1991). The mean age of adoption was 3.7 years (range: 1 month to 11 years). Sixty two percent of the girls were classified as stunted (< -2 SD) at arrival with a mean -2.2 SD. After two years, the mean height *z* score increased to -0.8 SD and only 20% of the girls were stunted. Oddly, mean final height, defined as the height attained three years after menarche, was 154 cm, only slightly greater than the height of the general Indian population. The mean final height *z* score decreased to -1.4 SD with 31% of the sample classified as stunted relative to WHO/NCHS reference values.[3] The mean age-at onset of menarche was 11.6 years which is 2 to 3 years earlier than found in rural Indian populations and lower in fact than observed in Swedish girls.[4] Analyses indicated that later adoption was associated with faster catch-up growth and that faster catch-up growth in turn, predicted earlier menarche ($r = .31, p < .001$). The explanation for understanding why final stature was less than expected may lie in the timing of menarche. It is possible that catch-up growth in older pre-pubescent girls may trigger endocrinological responses which would lead to an earlier onset of menarche. By accelerating maturation, the duration of growth would be shortened and in this way, some of the earlier growth compensated through catch-up growth would be negated.

---

[3] The WHO/NCHS reference curves used (Hamill et al, 1979) do not contain values for adults. Proos et al (1991) appear to have used NCHS original data to estimate z-scores for final height.

[4] Eveleth and Tanner (1990) give values of 13.0 years for Swedish girls.

**Discussion and Conclusions Regarding the Literature Review**

Linear growth retardation, or stunting, occurs primarily in the first 2 to 3 years of life and is a reflection of the interactive effects of poor energy and nutrient intakes and infection. In this review, data were examined to investigate how much, if any, catch-up growth occurs during later childhood and adolescence in various settings in developing countries.

Some of the findings presented may be questioned because of the use of external reference data, commonly from the United States. If ethnic differences among populations are important determinants of adolescent growth patterns (e.g., variations in age at take off, peak height velocity and duration of growth), then our ability to detect catch-up growth will be obfuscated by the use of an inappropriate reference population. These concerns do not apply to the analyses of Guatemalan and Indian data which relate stunting in early childhood to growth from 5 years of age till adulthood (i.e., these analyses do not depend on an external reference). The Korean study (Lien, Meyer and Winick, 1977; Winich, Meyer and Harris, 1975) also is less problematic since it used a Korean standard. Most analyses presented, however, relied on US reference data to make inferences about catch-up growth from cross-sectional data. For many of the populations studied, the issue of ethnic differences in adolescent growth with respect to the US reference population remains unresolved. With regards to the American slaves, there should be less concern about appropriateness of the reference population since US Blacks and whites have similar growth patterns during adolescence and nearly identical mean adult heights (Frisancho, 1990).

One conclusion of this review is that prolongation of the growth period can make up for some of the earlier growth retardation. How much catch-up growth is achieved depends on the degree to which biological maturation is delayed. In settings where maturation is grossly retarded, the potential for catch-up in growth will be marked. However, in developing countries today, maturation is measurably delayed but not dramatically retarded. Median ages at menarche, for example, are almost always less than 15 years and sometimes not very different from what is observed in well-nourished populations (Eveleth and Tanner, 1990). Along with less maturational retardation, there is also less stunting compared to a few decades ago (Eveleth and Tanner, 1990). These two conditions mean that while there is less growth failure to make up there is less opportunity to do so through the simple mechanism of a prolonged growth period.

A second mechanism through which catch-up growth can be achieved is through accelerated growth rates. This is clearly evident in the studies of children adopted by Swedish families. The change in the prevalence of stunting and in the average *z* scores two years after adoption were of such magnitude that growth rates must have greatly exceeded normality. Is this an unusual occurrence? Are children prior to adoption endocrinologically compromised, perhaps through psycho-social mechanisms, and hence likely to show catch-up growth once adopted? There are no clear answers to these questions.

A strong conclusion is that in most populations in developing countries, such as exemplified in this review by samples from rural Kenya, Malawi, Indian and Guatemala, the marked degree of retardation incurred in early childhood generally remains into adulthood. In other words, catch-up growth during later childhood and adolescence seems to be minimal in populations which continue to reside in the same environment which gave rise to stunting in early childhood. On the other hand, stunting can be reduced in early childhood if the environment is improved. This is demonstrated by the beneficial impact of food supplementation on the growth of Guatemalan children less than 3 years of age. The adoption studies of Korean children by US families and of Indian children by Swedish families show that adoption within the first few years of life leads to substantial catch-up in growth (though initial inter-group differences may remain) and increased height at follow-up. Unfortunately, these studies have not provided us with good information about growth during adolescence nor do they always report adult stature. Lack of this information does not allow us to make more definitive statements about the degree of catch-up growth which can be achieved through adoption.

Stunting in school age children and in adolescents may be less reversible. Earlier it was noted that if subjects remain in their places of origin with conditions unchanged, little or no catch-up in growth usually occurs. The Guatemalan study shows that a food supplement that is highly effective in improving growth during the period of growth failure does not improve growth in children from 3 to 7 years of age, when growth is normal. Confirmation of this differential effect of supplementation by age in other developing country settings is desirable. Also needed are studies that examine the effects on growth of nutrition and health interventions aimed at adolescents in developing countries; currently, there is no information of this type. There are also the intriguing findings from Sweden that suggest that although older children grow faster after adoption, the response hastens maturation, shortens the growth period, and in effect limits the possibility of catch-up growth. Confirmation of these findings is desirable as well.

Finally, what is the effect of catch-up growth in school age or adolescence on the functional correlates of stunting? While this is not a question addressed in this review, we would like to caution that remedial actions later in life may not redress all of the problems for which stunting is a marker. The effects on the functional correlates of stunting may vary greatly, some aspects improving along with stature and others not, perhaps as a function of whether they are direct or indirect consequences of growth retardation. Possible examples of direct and hence alterable correlates of growth are increased lean body mass and decreased obstetric risk in women due to small maternal size. On the other hand, effects of early childhood malnutrition on learning and behavior may not be redressed fully by the conditions which can bring on catch-up growth. If these views hold, addressing the problems of malnutrition and infection of early childhood is the preferred strategy in a resource poor environment because it would prevent stunting as well as its functional correlates. Intervening later, on the other hand, may have only a limited effect.

## 3. Growth During Adolescence in Rural Guatemala

### Patterns of Growth in Height

For analyses presented in this section, two sets of data were used: (a) length and weight data from the INCAP longitudinal study (1969-77) collected in subjects between birth and 7 years of age; and (b) height and weight data collected through a cross-sectional study of the same subjects when they were 11 to 24.9 years old (INCAP follow-up study). These data were then compared to reference data for the USA general population and for Mexican-Americans (Martorell, Schroeder & Kapolowitz, in press). The data for males are shown in Figure 3.1 for children less than 3 years and in Figure 3.2 for adolescents. The results for girls are similar and are not shown. The key findings shown in Figures 3.1 and 3.2 are as follows:

- At birth, the median length of Guatemalan children is at the 50th percentile of the NCHS/WHO reference.
- By six months of age, Guatemalan children are shorter, on average, than the 5th percentile of the reference curves and are about 5 centimeters below the NCHS/WHO median.
- By three years, children are about 10 centimeters shorter than the NCHS/WHO median.
- As adults, Guatemalans are at about the same absolute level of deficit (about 13 cm) as they were at age 3 years.
- Mexican-Americans are at the 50th percentile as young children and as young adolescents but are near the 25th percentile as young adults.

Interpretation of these descriptive findings suggest that if the U.S.A. population is used for comparison, Guatemalans can be said to grow as expected during adolescence, neither recuperating the growth retardation of early childhood nor falling further behind in size. If the Mexican-American sample is selected instead, it would appear that some catch-up in growth occurs in Guatemalan adolescents (also see Figure 3.3 below). Regardless, of the choice of reference population, growth is markedly retarded only in early childhood; adolescence is not a period when growth is significantly constrained.

**Age at Menarche**

For analyses presented in this section, retrospective data on age at menarche collected for 832 Guatemalans 15 to 30 years in age were used (Khan et al., in press). The analyses were designed to test whether exposure to a high energy and high protein supplement (Atole with 163 kcals energy and 11.5 g protein in 180 ml) during childhood led to earlier menarche than did exposure to a low energy, no protein supplement (Fresco with 59 kcals in 180 ml). It was found that mean age at menarche was similar in Atole (13.75 ± 1.22 yrs.) and Fresco (13.74 ± 1.36 yrs.) groups. The corresponding values for immigrants (n=144), subjects not exposed to the supplements, were 13.55 ± 1.20 yrs. Year of birth as well as socioeconomic status (SES) were associated with age at menarche. Age at menarche declined by 0.5 years per decade and menarche occurred earlier in higher SES households. Finally, significant, positive interactions between nutritional supplement type and SES as well as between supplement type and year of birth were found, but plausible explanations for them could not be advanced. The results indicate that there was little or no effect of the supplementation program on age at menarche. Comparison on age at menarche in the study population to values from well-nourished populations indicate that maturational delays amount to between 1 and 1.5 years.

**Size and Body Composition**

For this section, cross-sectional data from the Guatemalan adolescents were compared to the distribution observed in Mexican-Americans using data from HHANES (1982-84). The group of Mexican-Americans has a similar Spanish/Indian ancestry to that of the study population. Differences between Mexican-Americans and Guatemalans may interpreted without the usual concern about genetic biases with a view towards assessing the role of poor nutrition. Key findings are as follows:

- Guatemalans are at about the 10th percentile of the Mexican-American distribution for both height (Figure 3.3), and weight (Figure 3.4).

- Values for Body Mass Index (Figure 3.5) are somewhat greater in females than in males relative to the sex-specific distribution in Mexican-Americans. As adults, Guatemalan women are at the 25th percentile and men between the 25th and 10th percentiles.

- Guatemalans are leaner than Mexican-Americans, according to their subscapular skinfold thickness (Figure 3.6), particularly the men in whom values are often around the 10th percentile.

- Frame size is also smaller in Guatemalans. Biiliac crest breadths are between the 25th and 50th percentiles in adults (Figure 3.7). The possible significance of these values for dimensions of the pelvic inlet is unknown. Men appear to be somewhat smaller than women.

## 4. Longitudinal Study of Adolescence

Longitudinal data were collected for a year during adolescence on growth, diet, morbidity and physical activity in order to test descriptive as well as analytic hypotheses. The methods of the longitudinal study are reviewed first and then the results are presented by area. The presentation of results is divided by area: growth, diet, health and physical activity.

### Hypotheses and Methods

The descriptive hypotheses in the area of growth in height were:

- Adolescents growing up in a rural environment which produced marked stunting in early childhood are able to grow as well as adolescents in well-nourished circumstances.

- The duration of the growth period will be prolonged by a year or less but the contribution of this delay to final height will be minor.

The analytic hypotheses were:

- Growth during adolescence will be independent of growth in early childhood, i.e. growth retardation in early childhood will not be recuperated in adolescence.

- Growth during adolescence will be unconstrained, i.e. not dependent upon the socioeconomic, dietary, and infectious disease factors which cause growth retardation in early childhood.

- The effects of the INCAP nutrition intervention during early childhood will not affect growth during adolescence i.e. there will be no long-term, residual effects on adolescent growth.

Data collection in the adolescent longitudinal component is described in Table 4.1. The census used methods previously used by INCAP. The existing information for the household was printed out. The interviewer used the printout in asking the informant whether changes had occurred. Changes were recorded in an "update" form and integrated with the previous data. Anthropometry was collected using commonly accepted techniques. The anthropomorphists were carefully trained and standardized. Quality control included inspection of forms in the field and examination of the data for outliers. A 24-hour recall survey was carried out in the home using methods used by INCAP in previous studies (Habicht & Martorell, 1992). The food composition table for Central America and Panama was used to convert amounts to nutrients. Software especially developed at INCAP was used to achieve this. A morbidity survey was carried out using a pre-coded form. All signs and symptoms reported in the previous two weeks were recorded, including when they began and ended. Activity was assessed by means of an interview in which the subject reported her activities, including hours in bed, for a 24-hour period. Age at menarche was asked of all girls who had previously not reported it.

**Results on Growth**

*Attained growth*

Table 4.2 and Figure 4.1 present the mean attained stature by age of females participating in the study and the 50th, 25th, 10th, and 5th percentile of a representative sample of the U.S. population of the same age groups, based on NHANES I and NHANES II (Frisancho 1990). The pattern of attained stature of Guatemalan females follows, in general, that of the U.S., but the mean stature is far below the U.S. mean, falling at or below the 5th percentile at most ages. On the basis of mean attained stature, it seems that no growth or very little growth occurs after 14 years of age. Table 4.3 and Figure 4.2 present mean attained weight of Guatemalan females as compared to the 50th, 25th, 10th, and 5th percentiles of the NHANES curves. Mean attained weights fall between the 10th and the 25th percentiles of the U.S. distribution from 9 to 18 years.

*Incremental growth and the descriptive hypotheses*

Weight and height velocities were estimated as the regression coefficient of weight (kg) or height (cm) on age (month) within individuals. The average age at which the repeated measurements occurred was obtained. Regression coefficients were grouped in yearly age intervals according to the mean age corresponding to each coefficient. Repeated anthropometric measures (at least two) were available for females between 9 and 23 years of age.

Tables 4.4 and 4.5 and Figures 4.3 and 4.4 present mean and standard deviations of the regression coefficients of height on age and weight on age, respectively, by age. The units are cm or kg per month. Tables 4.4 and 4.5 also present increments per year. After 18 years of age growth in stature is very small (less than 0.5 cm/year). Weight gain after 17 years of age tends to be minimal, except at 18 and 20 years of age, probably due to random variability.

There are not comparable data from a developed country for use as a reference. However, we have estimated height velocities from differences in attained growth between age groups using the NHANES data. Since differences between age groups may contain cohort effects, comparisons with our data should be made with caution.

Table 4.6 presents mean regression coefficients of height on age, by age, and the differences in attained height between age groups in the NHANES sample. Age grouping for the Guatemalan and NHANES data are different. While our data are grouped on the basis of the mean age for which regression coefficients were estimated and are presented by age in years (for example 14 years is the interval between 14.0 and 14.9 years), data for the US population are differences between mean attained growth of age intervals (for example, the difference 13-14 years is the differences in mean attained height between 14.0-14.9 and 13.0-13.9 years). This is reflected in Table 4.6 by the use of different rows for the Guatemalan and the U.S. samples. The mean height differences between adjacent intervals from U.S. data are presented in parenthesis in the rows corresponding to the age groups for which Guatemalan data are presented. Increments in the Guatemalan sample seem to be similar or larger than the height differences between ages in the U.S. sample, except at 9 years of age, probably due to delayed maturation.

Table 4.7 presents similar data for increments in weight. Results show that the pattern of weight increments of Guatemalans are different than the corresponding differences observed in the U.S. From 9 to 11 years of age, increments in Guatemalan girls are smaller than weight differences between ages in the U.S. After 11 years, increments in Guatemalans tend to be larger (from 12-16 years) or equal (18 years) than corresponding figures in the U.S. population, except at 17 years. However, although the pattern seems to be different, the cumulative weight gain from 9 to 18 years is almost identical (about 30 kg) in U.S and Guatemala. The differences in weight gain patterns may be the result of differences in patterns of maturation.

For reasons that are unclear, the velocity estimates appear to overestimate the amount of growth in height which takes place between 16 and 23 years of age (Table 4.6) compared to estimates obtained by comparing attained values at the ages of concern (Table 4.2).

**Dietary intakes**

The information below is based on repeated dietary surveys and is generally presented as mean dietary intakes by age. The data analyses were restricted to subjects who consumed all three customary meals at home. A total of 92.4% of dietary survey forms reported the consumption of three meals. The cut-off points to eliminate outliers were based on the RDA values for energy per kg of weight. For the highest value, it was defined as the level at two standard deviations above the mean for people 25 years of age (81.3 kg x 56 Kcals/kg = 2140 + 2 s.d. = 3107 kcals). For the lowest extreme, the cut-off was defined initially as less than three standard deviations below the mean for the youngest age group (for 9 years old, 15.4 weight in kg x 62 Kcals/kg- 3 s.d. = 955 Kcals). This was later modified to select the same number of cases in the lower extreme as in the upper extreme. The final cut-off point selected for the lower limit was 836 kilocalories (-3.5 s.d.). The number of outlier did not differ by age. The number of cases deleted was about 3.6% each at high and low ends of the distribution (or 85 subjects at each and). The final number of dietary surveys was 757. The age groups 24 and 25 years were not included because they had too few subjects. In some analyses, the age groups were collapsed into the age ranges used by the National Research Council: 9-10, 11-14, 15-18 and 19-25 years (1989).

Table 4.8 presents mean (S.D.) energy intakes by age group. The number of cases is above 30 for all ages except for 23 years of age and older. There is surprisingly little variation in intakes among ages.

Figure 4.5 present the percent of energy provided by each micronutrient by age. Little variation is observed among ages. In general, carbohydrates provide about 75% of total energy, protein about 11% to 12%, and fat between 15% and 19%. The proportion provided by fat tends to be slightly increased at older ages. The proportions approximate the ideal from the point of view of some health advocates in the United States.

Table 4.9 presents energy intake expressed as percent of recommended intakes. Two criteria are used: a) age-based recommended dietary allowances (RDAs) for U.S. populations and b) similar recommendations based on weight. The latter were based on recommended intakes per body weight, provided by the U.S. National Research Council, and on mean observed weights at each age in the study population. Intakes of the Guatemalan sample lag behind recommendations for age by about 15%. However, since the mean weights of Guatemalan women are between the 10th and 25th percentile of the U.S. distribution, the population's intake is near that recommended when values are expressed per kg of body weight.

Protein is not limiting in the diets of the Guatemalan adolescents studied. Although dietary protein is primarily of vegetable origin, nearly 85% at all ages according to Figure 4.6, substantial amounts of protein are consumed as shown in Figures 4.7 (vegetable origin, adjusting for body weight) and Figure 4.8 (animal origin, adjusting for body weight). The median vegetable portion consumed (Figure 4.7) equals or exceeds the recommended values and the amount of animal protein consumed makes an important additional contribution (Figure 4.8). Protein quality is not an limited among adolescents as it is among young children (FAO/WHO/UNU, 1985) and the amounts ingested from vegetable and animal sources exceed protein requirements by a wide margin.

Table 4.10 presents energy intake as a percent of recommended intakes for age and for body weight. As mentioned earlier, intakes are below recommendations for age by about 14-16%. However, relative to body weight, deficits are smaller and range between 0 and 7%. The only age group with a possible deficit is the youngest (i.e. 9-10 years). After 10 years of age, intakes are very close to recommendations.

This information suggests that, except for girls between 9 and 10 years of age, energy intakes in this population may be adequate, given the small body size of the population as a result of stunting during childhood. Later, information on physical activity levels will be presented and it will be made clear that energy expenditures in this population are not unusually high.

Table 4.11 presents the median and the 90th percentile of the distribution of intakes of micronutrients and fiber[5]. The 90th percentile is presented since dietary recommendations, except for energy, are based on intakes that satisfy the requirements of practically all the population of a given age (i.e. the RDA is generally set at 2 S.D.'s above the mean of the distribution). Therefore, values at the 90th percentile that are below the RDA may be viewed as indicating a poor diet.

In general, the 90th percentile values are above the recommendations, except possibly in the case of riboflavin and calcium. For iron, intakes at the 90th percentile are above the RDA; however, given the low availability of iron in diets such as the ones consumed in rural Guatemala (with high content of phytate and fiber), it could be suggested that iron status is not optimum. However, the prevalence of anemia in this population is low (Murdoch et al. forthcoming).

**Health**

Morbidity information was obtained through interviews of the adolescents; it includes information about the presence and duration of a number of signs of disease during the two weeks prior to the interview. Morbidity was classified as mild, moderate or severe based on the criteria given in Table 4.12. For example, for upper respiratory infections, mild was indicated by the presence of any symptoms (011, 014, 310, 311, 042, 312, or 313), moderate if fever was present and severe if the subject was forced to stay in bed.

The morbidity findings are presented in Table 4.13, which presents information using subject-day as the unit of analyses. The findings show that 61.4% of subject-days were free of symptoms and that 38.6% involved some illness. However, over half of the subject-days with illness were mild. Only 4.7% of subject-days were characterized as severely ill, that is, ill enough to be in bed.

---

[5] The fiber data are difficult to interpret. Intakes at the median and at the 90th percentile are about 6 and 10g respectively. These amounts seem too low suggesting that the food composition data used are inadequate.

**Physical Activity**

Physical activity data were collected and summarized in terms of BMR equivalents. Information about the time dedicated by each individual to various physical activities was obtained by recall for a 24-hour period. The expenditure cost per activity was obtained by considering the time spent and the cost of the activity, using information provided by FAO/WHO/UNU (1985). The average physical activity level (PAL) was obtained for each individual. FAO/WHO/UNU considers light activity in women to be equivalent to a PAL of 1.56, 1.64 to indicate moderate activity and 1.82 to be heavy activity. These values are in reference to the basal metabolic rate, the reference base of "1". Thus, a moderately active woman expends energy during a 24-hour period at an average rate which is 64% higher (i.e. PAL = 1.64) than if she spent the entire 24 hours sleeping (PAL = 1.0).

Commonly carried out activities included fetching water, collecting firewood, cooking, making tortillas, sports and games, delivering lunch to field workers and many others. The percentile distribution by age of the PAL is given in Figure 4.9. One may look across age, for example, at the 50th percentile. This shows that the 50th percentile is between 1.6 and 1.5 between 8 and 14 years of age and that it rises to about 1.7 thereafter. Thus, the average girl had a moderate level of physical activity. The data can also be examined up and down within age to look at the distribution of the PAL index. At ages older than 15 years, some 25% of subjects are engaged in heavy physical activity whereas an equal percentage is characterized by light activity.

The percentage of girls with moderate or heavy physical activity is presented by age group in Figure 4.10. Clearly, physical activity is lowest at ages 8-14; at 15 years of age or older, about 60 of cases have moderate or heavy patterns of physical activity. Figure 4.11 present the information by village.

The pattern of physical activity is that of a moderately active population. While there are some who engage in heavy physical activity, an equal number are not very physically active.

## Analytic Hypotheses

*Lack of catch-up growth during adolescence*

The first analytic hypothesis that growth during adolescence will be independent of growth in early childhood (i.e. growth retardation in early childhood will not be recuperated in adolescence) was confirmed using several approaches, based on results presented above. In summary, analyses of our data show that the level of stunting at 3 years of age is independent of growth during adolescence; comparison of growth rates during adolescence to those implied in the NHANES data show them to be similar; and comparison to NHANES data indicate that Guatemalans track along a similar percentile throughout adolescence. The above results lead us to affirm that growth retardation in the Guatemalan population is not recuperated in adolescence.

*Environmental factors and adolescent growth*

The second analytic hypothesis is that growth during adolescence will be unconstrained, i.e., not dependent upon the socio-economic dietary and infectious disease factors which cause growth retardation in early childhood. That is, the same variables that were determinants of growth during early childhood in this population are not determinants of growth during puberty and adolescence. To test this hypothesis, we assessed the relationship between growth rates and several potential determinants of growth. Only females under 17 years of age were included in this part of the analysis because growth was almost negligible after this age.

The variables used in the analysis are described below:

- Weight and height velocities. As described above (kg/year and cm/year, respectively).

- Age. In years, obtained from the girls and/or their mothers during this study and corroborated by the records of the INCAP longitudinal study for those who were participants 1969-1977.

- Socioeconomic index. A socioeconomic score was generated from factor analysis using information about living conditions of the family in 1987. After initial testing, the model was restricted to one factor. Only factors with loadings at or above 0.5 were retained. The variables composing the factor were house characteristics (type of floor, an overall assessment of the quality of house construction, type of excreta disposal, the location of the kitchen, and facilities for cooking) and possession of household items. Standardized factor scores were used in the analysis.

- Dietary intake. Obtained by 24-hour recall dietary intake. Food intake was transformed into energy and nutrient consumption using the INCAP food composition tables. Variables used in the analysis include: energy intake (Kcal/day and % RDA), protein intake (% RDA), and iron (mg/day) and vitamin A (mcg R.E./day) intake.

- Physical Activity Level (% of BMR). As described above (PAL) expressed as % Basic Metabolic Rate.

- Morbidity. The variables used in the analysis are dichotomous variables with a value of 0 for subjects who were healthy or mildly ill during the previous two weeks and a value of 1 for subjects who were moderately or severely ill or mildly ill (see Table 4.12 for the definitions used for the various items).

Table 4.14 presents correlation coefficients between adolescent growth rates and several variables that were shown in the past to be determinants of growth in children under three years of age in this population and were therefore considered as potential determinants of growth during puberty and adolescence. Protein and energy intake, as well as level of physical activity, are associated with either weight or height velocity from 9-17 years of age. However, age was highly associated with growth and with dietary intakes and energy expenditure. Therefore, age needs to be controlled for in the multivariate analysis that follows.

Table 4.15 presents univariate and multivariate regression models of the potential determinants of height velocity during puberty and adolescence. Independent variables are age (years), energy intake (% RDA), protein intake (% RDA), socioeconomic index, level of physical activity, and overall morbidity.

Age, protein intake, and energy expenditure had significant correlation with height velocity in the univariate models and in multivariate model 2, which does not include age. However, when age is included in the model (Models 1 and 3), protein intake and energy expenditure are no longer significant predictors. The only variable that explains height velocity in Guatemalan adolescent females is age; from 9 to 17 years, height velocity decreases as age increases.

Table 4.16 presents univariate and multivariate regression models for weight velocity. The same independent variables used in the models to explain variability in height velocity were used. Only age and energy intake had significant correlation in the univariate models. Protein intake and morbidity were borderline significant. In the multivariate models, energy intake was the only variable that remained significant even after controlling for age. We conclude that energy intake is a determinant of weight gain but not of height gain in Guatemalan female adolescents.

Stunting is the principal nutritional problem in Guatemala as well as in most Latin American countries. Moreover, the adolescent girls studied have a high prevalence of stunting while the prevalence of underweight is low. Our results suggest that improving dietary intake during puberty and adolescence will not improve linear growth and, therefore, stunting will not be reduced through interventions during puberty and adolescence. In contrast, weight gain may increase as a result of improvements in dietary intakes. Given that BMI is adequate in this age, improvements in weight gain without parallel improvements in height velocity has either no public health importance or may lead to overweight.

*Supplementation in early childhood and growth during adolescence*

The third analytic hypothesis is that the effects of the INCAP nutrition intervention during early childhood will not affect growth during adolescence, i.e. there will be no long-term, residual effects on adolescent growth. In the study design, two villages received a high protein, high energy supplement (Atole: 163 kcal energy and 11.5g protein per cup, that is 180 mL) and two others a low energy, no protein supplement (Fresco: 59 kcal per 180 ml). The supplements were available from 1969-77. The follow-up was carried out ten years after the end of the supplementation study.

The results lead to acceptance of this hypothesis. In a simple analysis, growth rates during adolescence were shown to be similar in Atole and Fresco villages. This suggests that there are no long-term effects of the intervention on adolescent growth. A more formal test which takes account of several potentially confounding factors is given in Table 4.17. The potentially confounding variables are the height of the adolescents' mothers (a proxy for genetic potential), SES in early childhood, diarrhea and diet in early childhood, skeletal age at adolescence (also includes a quadratic term). Three models are presented. The first tests effects of supplement (Atole = 1, Fresco = 0) on length at 3 years. The second model tests effects on adolescent height, adding maturation as a confounding variable. The last model, by including length at 3 years of age, in effect tests the relationship between supplement and growth from 3 years to adolescence.

The results indicate that supplement is significantly related to length at 3 years of age (model 1) that it is much less related to height at adolescence (model 2, significant only for females). The results for model 3 indicate that supplement is not related to growth between 3 years and adolescence. Thus, the relationship between supplement and height at adolescence is due to the effects of supplement on length at 3 years. The fact that the coefficient for supplement (model 3) is slightly negative (but non-significant) indicates a tendency for growth in Fresco villages (non-supplemented group) to be slightly greater than in Atole villages from 3 years to adolescence.

## 5. Discussion and Conclusions

Much is known in developing countries about growth and development in early childhood and the factors which shape its course. Considerably less is known about growth in later childhood and adolescence; these are the *dark ages*, literally.

A consensus is emerging in the international nutrition literature about the timing of greatest growth failure and nutritional stress among the poor in developing countries. The emerging view is that the first two to three years of life demarcate the period in life where massive growth retardation occurs, the "age of growth failure," brought on by the complex interplay of poor diets and infection, forces imbedded in a setting of poverty and its educational, environmental and social correlates. A cogent articulation of these views is contained in the monograph "The Uses of Anthropometry" recently released by the Subcommittee on Nutrition of the United Nations (Beaton et al., 1990).

That the term "age of growth failure" appropriately describes the first few years of life in most settings in developing countries is rarely disputed. What is a subject of considerable speculation, however, is whether the effects produced in this stormy early period endure undiminished throughout life or whether children are able to make up, at least some of the losses, later in life when the burden of poor diets and infection is less. In particular, adolescence, a phase of marked and accelerated growth and rapid maturation, looms as the most promising opportunity for recuperation in the minds of some.

While most of the studies to date have failed to find catch-up growth during adolescence (Martorell, Khan and Schroeder, 1994), there are, however, some studies that suggest some potential for recuperation during adolescence (Satyanarayana et al., 1981). In none of these studies were environmental conditions altered to test whether patterns of development could be improved. There are several limitations in the studies which should be emphasized. For example, few have examined longitudinal data during adolescence as a function of earlier growth. Also, none prior to our study have directly investigated the effects of diet, health, and other factors on growth during adolescence to establish whether there are factors affecting growth at this period that are amenable to intervention.

The paucity of data about the "dark ages" of later childhood and adolescence allow for potentially conflicting views about the most appropriate policies and programs to pursue and implement. At the extremes, there are two views. One sees the effects on growth and body size in early childhood as largely irreversible as most of the studies above would suggest. Under this view, to adequately prepare females for future work and reproductive demands, it is essential that adequate growth and development in early childhood take place. Growth is perceived as a dynamic process under the influence of metabolic forces controlled by maturation; in effect, by a biological clock. Once the biological clock moves beyond the period of fast but decelerating growth in the first two years of life, no amount of refeeding, it is felt, would permit older children to grow as fast as younger children. Under this model, the potential for catch-up growth would be limited largely to delays in the biological clock (see Martorell et al., 1979 for a presentation of this model). In situations of scarce resources, the highest priority would be given to early childhood programs; programs aimed at older children and adolescents would be difficult to justify unless one could show that significant impairment of growth and development occurs at these ages.

At the other extreme is a view which ascribes greater resilience and plasticity to human growth, much in the same way as many developmental psychologists view development. The notable auxologist, James Tanner, has described growth as a target seeking phenomenon (Tanner, 1986). Once the influences of factors which deflected growth from its pre-determined trajectory are withdrawn, restoring forces emerge to place the child back on its course. This second view offers more flexible policy options. A dual strategy would seem to emerge: continued support to actions which will bring about healthy growth and development in early childhood but coupled with attention to the adolescence period to ensure adequate nutrition in this final phase and to make up for earlier losses. Which of these perspectives of human growth holds truer in poor societies is a matter for serious consideration and much in need for further research.

Thus, the present study fills an important gap in the literature. It examines health, diet and growth interrelationships from birth to adulthood using a mixed-longitudinal design which combines information obtained from 1969 to 1988 in 4 rural Guatemalan villages with complementary data collected in 1992. First, from 1969 to 1977, INCAP carried out a longitudinal study from birth to seven years of age in which home diets, supplement ingestion, morbidity and growth and development were recorded. In 1988-89, a comprehensive, cross-sectional follow-up was carried out which provided current-status information for all subjects previously studied, then 10 to 26 years of age. The assessment included body size and composition, maturation, fertility history, intelligence, numeracy, literature and schooling information among others. These two sources of data allow an in-depth characterization of growth and development in early childhood, make possible the study of changes between early childhood and adolescence, but permit only a *cross-sectional analysis* of growth during adolescence because each subject was measured only once during adolescence.

The additional component reflected here included the collection of *longitudinal* data on growth, diet, morbidity and physical activity in adolescents. These new data allow for a semi-longitudinal assessment of growth during adolescence and immensely enrich the existing data set's potential for studying the process of growth and development from birth to adulthood.

The data collected with ICRW support has enabled us to achieve the following aims:

- describe patterns of growth and development from birth to adulthood in Guatemalan females.
- test specific hypotheses about the dependency of growth at various stages of life, including adolescence, on health, nutrition and socioeconomic status.
- provide guidance useful for formulation of policies and programs that seek to improve the nutritional status of adolescent girls.

The expectation driving the formal hypotheses of this study was that adolescents in the study population are in good health. By this it is meant that they do not exhibit wasting or clinical signs of nutritional deficiencies. Also, adolescents are expected to consume diets that meet estimated protein-energy needs and to grow adequately when compared to reference populations. However, it was anticipated that milestones such as menarche, peak height velocity, and achievement of adult height would be delayed moderately (i.e. less than 1 year). Prior to the study, we knew that growth *in early childhood* was constrained as indicated by strong relationships between growth rates and socioeconomic status, diarrheal diseases, home dietary and food supplement intakes. *We did not expect to find such relationships during adolescence* because we hypothesized that growth during adolescence was unconstrained by environmental factors. Furthermore, we expected to show that growth during adolescence was independent of growth in early childhood (i.e. that there is no catch-up in growth; if there were, the relationship between growth in early childhood and in adolescence would be strongly negative). Finally, we knew of no mechanisms which would lead us to anticipate that supplementation during early childhood would affect longitudinal growth during adolescence.

These expectations were fully confirmed by the literature review and the findings of the study. The purpose of the literature review was to assess the extent to which stunting, a phenomenon of early childhood, can be reversed in later childhood and adolescence. The focus was on data from developing countries. The specific conclusions of the review were as follows:

- Maturational delays in developing countries are usually less than two years, only enough to compensate for a small fraction of the growth retardation of early childhood.

- Follow-up studies find that subjects who remain in the setting in which they became stunted experience little or no catch-up in growth later in life.

- Improvements in living conditions, as through food supplementation or through adoption, trigger catch-up growth but do so more effectively in the very young.

- One study cautions that in older adopted subjects, accelerated growth may accelerate maturation, shorten the growth period and lead to short adult stature.

The key overall conclusion of the review was that the potential for catch-up growth was found to increase as maturation is delayed and the growth period is prolonged. Since delays in maturation in developing countries are often moderate (i.e. delays of 2 years or less), the actual amount of catch-up growth taking place through this mechanism is limited. In most settings, the growth failure of early childhood is reflected in reduced stature among adults.

The research findings of the study lead to the following profile of health and nutrition in rural Latino Guatemalan adolescents:

- Pregnancy and the first 2-3 years of life appear to be the only periods in life when physical growth is retarded significantly. During adolescence, the timing of peak height velocity occurs a year or so later, but otherwise growth in height appears to be as in well-nourished children. Growth during adolescence could never be characterized as a period of significant growth failure.

- Menarche occurs about a year later, confirming a moderate delay in maturation. However, supplementation in early childhood did not impact on menarche.

- Adolescents are short (stunted) but this is a carry over from the retardation which occurred in early childhood and not the result of poor adolescent growth.

- Adolescents have an adequate BMI and satisfactory skinfold values. Specifically, these are somewhat lower than in American subjects who are judged by many to be fat.

- Dietary intakes are clearly adequate in terms of protein. In terms of energy, intakes are satisfactory if judged in terms of requirements per kg. Recognizing the deficiencies of dietary surveys, it would appear that intakes of riboflavin and calcium may be low. Iron appears to be adequate but bioavailability is poor and the needs may not be met. Still, anemia levels are low.

- Physical activity data indicate a moderate level of exertion for the typical adolescent.

- Most adolescents are healthy and only a few are typically sick enough that they need to stay in bed.

- Analyses reveal that diet, health and socioeconomic status are not predictive of growth in height during adolescence. This suggests unconstrained growth. On the other hand, energy intake from the diet did appear to be related to weight gain.

Regarding the policy implications of the study, the picture which emerges is one of largely good health, adequate nutrition and satisfactory growth during adolescence. The effects of poor nutrition and health in early childhood are reflected however, in terms of short stature, a risk factor for delivery complications. It is not known whether nutrition interventions just prior or during adolescence would increase stature, but our data would make this seem unlikely. Growth during adolescence, as noted above, is already as good as in well-nourished subjects. Diet, health or SES were unrelated to growth in height during adolescence, suggesting unconstrained growth. On the other hand, supplementation during adolescence in at least one study of adopted Indian girls in Sweden indicates that maturation may be accelerated and that this in turn may affect final stature adversely. The other possibility is that weight and fatness levels may be increased, desirable only in thin adolescents.

Clearly, the issue of the value of nutrition interventions in adolescents needs to be pursued from the research angle. It would be inappropriate at the present to pursue programs designed with the expectation of fully redressing the effects of early childhood malnutrition through interventions in adolescence. These actions may not be effective (i.e. it may be impossible to reverse these effects) and may in fact be harmful in some settings (i.e. accelerate maturation); another drawback is that funds and resources may be diverted from more needy programs in early childhood.

This is not to say that nutrition programs should not be aimed at adolescents. Where thinness is documented, or where micronutrient deficiencies are known to exist, appropriate remedial measures need to be implemented. School age and adolescence are also opportune periods for nutrition and health education, both through the formal sector and outside of it.

In conclusion, this study has addressed an important gap in the literature. Its two strongest and most important findings are as follows:

- Guatemalan adolescents are healthy; they exhibit adequate growth, eat diets that satisfy most of their nutritional needs, are rarely ill and have moderate levels of physical activity.

- Guatemalan adolescents carry the sequelae of prior malnutrition, reflected in short stature. This places women at risk of delivery complications and death and impacts negatively on birth weight.

Finally, the type of study undertaken leaves an important question unanswered. We do not know if a nutritional intervention during adolescence can improve growth in height and thus make up for the growth lost during early childhood. While we hypothesize that these effects are unlikely, we believe that appropriate studies in this area need to be carried out because of the important policy questions that would be enlightened by such research.

## References

Beaton G, Kelly A, Kevany J, Martorell R and Mason J. Appropriate uses of anthropometric indices in children. ACC/SCN State-Of-The-Art-Series Nutrition Policy Discussion Paper No. 7, December 1990.

Eveleth PB & Tanner JM. *Worldwide variation in human growth*, 2nd edn. Cambridge: Cambridge University Press, 1990.

FAO/WHO/UNU: Energy and protein requirements. WHO Tech. Rep. Ser. No. 724. WHO, Geneva, 1985.

Frisancho AR. *Anthropometric standards for the assessment of growth and nutritional status.* Ann Arbor, MI: The University of Michigan Press, 1990.

Gopalan G, Swaminathan VK, Krishna Kumari VK, Hanamantha Rao D & Vijayaraghavan K. Effect of calorie supplementation on growth of undernourished children. *Am. J. Clin. Nutr.*, 1973, 26, 563-6.

Habicht J-P & Martorell R. Objectives, research design, and implementation of the INCAP longitudinal study. *Food Nutr Bull.*, 1992, 14(3):176-190.

Hammill PVV, Drizd TA, Johnson CL, Reed RB, Roche AF & Moore WM. Physical growth: National Center for Health Statistics percentiles. *Am. J. Clin. Nutr.,* 1979, 32, 607-629.

Hong CY. *Pediatric Diagnosis and Treatment.* Yongrin, 1970, Korea.

Khan AD, Schroeder DG, Martorell R & Rivera J. Age at menarche and nutritional supplementation. *J. Nutr.*, in press.

Kiple KF & King VH. Slave mortality: some nutritional answers to a perennial puzzle. *J. Soc. Hist.,* 1977, 10, 284-309.

Lien NM, Meyer KK & Winick M. Early malnutrition and "late" adoption: a study of their effects on the development of Korean orphans adopted into American families. *Am. J. Clin. Nutr.*, 1977, 30, 1734-1739.

Little MA, Galvin K & Mugambi M. Cross-sectional growth of nomadic Turkana Pastoralists. *Hum. Bio.*, 1983, 55, No. 4, 811-830.

Lutter CK, Mora JO, Habicht J-P, Rasmussen KM, Robson DS & Herrera MG. Age-specific responsiveness of weight and length to nutritional supplementation. *Am. J. Clin. Nutr.*, 1990, 51, 359-64.

Malcolm LA. 1970 *Growth and development in New Guinea: A study of the Bundi people of the Madang District*. Institute of Human Biology, Madang, 1970.

Martorell R, Yarbrough C, Klein RE, & Lechtig A. Malnutrition, body size and skeletal maturation: Interrelationships and implications for catch-up growth. *Human Biology,* 1979, 51:371-389.

Martorell R. Child growth retardation: A discussion of its causes and its relationship to health. International Symposium on Nutritional Adaptation in Man, Royal Windsor, England. In *Nutritional adaptation in man*, eds. K Blaxter and JC Waterlow, 1985, 13-30. London and Paris: John Libbey.

Martorell R & Habicht JP. Growth in early childhood in developing countries. In *Human growth: A comprehensive treatise Volume 3. Methodology: Ecological, genetic, and nutritional effects on growth*, 2nd edn, eds. F Falkner & JM Tanner, 1986, 241-262. New York and London: Plenum Press.

Martorell R, Habicht J-P & Klein RE. Anthropometric indicators of changes in nutritional status in malnourished populations. In *Methodologies for human population studies in nutrition related to health*, ed. BA Underwood, 1982, 96-110. NIH Publication No. 82-2462. US Government Printing Office, Washington, DC.

Martorell R, Kettel Khan L & Schroeder DG. Reversibility of stunting: Epidemiological findings in children from developing countries. *Europ. J. Clin. Nutr.*, 1994, 48(suppl. 1):S45-S57.

Martorell R, Mendoza FS & Castillo RÓ. Genetic and environmental determinants of growth of Mexican-Americans. *Pediatrics* 84(5), 1989:864-871.

Martorell R, Rivera J & Kaplowitz H. Consequences of stunting in early childhood for adult body size in rural Guatemala. *Annales Nestlé* , 1990, 48, No. 2, 85-92.

Martorell R, Rivera J, Kaplowitz H & Pollitt E. Long-term consequences of growth retardation during early childhood. In *Human growth: Basic and clinical aspects*, ed. M Hernandez, J Argente, 1992, 143-149. Amsterdam, The Netherlands: Elsevier Science Publishers B.V.

Martorell R, Schroeder DG & Kaplowitz H. Patterns of linear growth in rural Guatemalan Adolescents and Children. *J Nutr.*, in press.

Murdoch S., J.D. Haas, J. Rivera, and R. Martorell. Prevalence of impaired iron status of adolescents and young adults from rural Guatemala. Forthcoming.

National Research Council, *Recommended Dietary Allowances*. Washington, DC: National Academy of Sciences, 1989.

Neumann C, Bwibo NO & Sigman M. *Human nutrition collaborative research support program. Functional implications of malnutrition: Kenya Project*. Los Angeles, CA: UCLA School of Public Health, 1992.

Pelletier DL, Low JW & Msukwa LAH. Malawi maternal and child nutrition study: study design and anthropometric characteristics of children and adults. *Am. J. Hum. Biol.*, 1991, 3, No. 4, 347-361.

Postell WD. *The Health of Slaves on Southern Plantations*. Baton Rouge: Louisiana State University Press, 1951.

Proos LA, Hofvander Y & Tuvemo T. Menarcheal age and growth pattern of Indian girls adopted in Sweden. *Acta Paediatr. Scand.*, 1991, 80, 852-858.

Proos LA, Hofvander Y, Wennqvist K & Tuvemo T. A longitudinal study on anthropometric and clinical development of Indian children adopted in Sweden. II. Growth, morbidity and development during two years after arrival in Sweden. In *Growth and development of Indian children adopted in Sweden*, LA Proos, 1992, V:1-18. Lindbergs Grafiska HB, Uppsala.

Reed RB & Stuart HC. Patterns of growth in height and weight from birth to eighteen years of age. *Pediatrics*, 1959, 24, 904-21.

Satyanarayana K, Nadamuni Naidu A & Narasinga Rao BS. Adolescent growth spurt among rural Indian boys in relation to their nutritional status in early childhood. *Ann. Hum. Biol.*, 1980, 7, No. 4, 359-365.

Satyanarayana K, Nadamuni Naidu A, Swaminathan MC & Narasinga Rao BS. Effect of nutritional deprivation in early childhood on later growth -community study without intervention. *Am. J. Clin. Nutr.*, 1981, 34, 1636-1637.

Satyanarayana K, Radhaiah G, Murali Mohan R, Thimmayamma BVS, Pralhad Rao N & Narasinga Rao BS. The adolescent growth spurt of height among rural Indian boys in relation to childhood nutritional background: an 18 year longitudinal study. *Ann. Hum. Biol.*, 1989, 16 (4), 289-300.

Schroeder DG, Martorell R, Rivera J, Ruel M & Habicht J-P. Age differences in the impact of nutritional supplementation on growth. *J. Nutr.*, in press.

Schumacher LB, Pawson IG & Kretchmer N. Growth of immigrant children in the Newcomer schools of San Francisco. *Pediatrics*, 1987, 80, 861-868.

Steckel RH. Growth depression and recovery: the remarkable case of American slaves. *Ann. Hum. Biol.* 1987, 14, No. 2, 111-132.

Swados F. Negro health on the Ante Bellum Plantations. *Bull. Hist. Med.*, 1942, 10, 460-472.

Tanner JM. Growth as a target-seeking function: catch-up and catch-down growth in man. In *Human growth: a comprehensive treatise, Volume I. Developmental biology: prenatal growth*, 2nd edn, eds. F Falkner & JM Tanner, 1986, 167-179. New York: Plenum Press.

Tanner JM. *A History of the Study of Human Growth*. Cambridge: Cambridge University, 1981.

Trussel J & Steckel RN. The age of slaves at menarche and their first birth. *J. Interdis. History*, 1978, 8(3), 477-505.

Victora CG. The association between wasting and stunting: an international perspective. *J. Nutr.*, 1992, 122(5), 1105-1110.

Waterlow JC. *Protein energy malnutrition*. London: Edward Arnold, 1992.

Winick M, Meyer KK & Harris RC. Malnutrition and environmental enrichment by early adoption. *Science*, 1975, 190, 1173-1175.

Yip R, Scanlon K & Trowbridge F. Improving growth status of Asian refugee children in the United States. *JAMA*, 1992, 267, 937-940.

## List of Tables

## List of Figures

**Table 1.1**

**Resident Females Born 1969-1983**
**by Year of Birth**

| YEAR OF BIRTH | AGE AT 1993 | NUMBER OF SUBJECTS | |
|---|---|---|---|
| | | TOTAL | % |
| 83 | 9 | 63 | 5.93 |
| 82 | 10 | 89 | 8.38 |
| 81 | 11 | 67 | 6.31 |
| 80 | 12 | 73 | 6.88 |
| 79 | 13 | 71 | 6.70 |
| 78 | 14 | 54 | 5.08 |
| 77 | 15 | 68 | 6.40 |
| 76 | 16 | 72 | 6.78 |
| 75 | 17 | 96 | 9.04 |
| 74 | 19 | 94 | 8.85 |
| 73 | 20 | 89 | 8.39 |
| 72 | 21 | 78 | 7.35 |
| 71 | 22 | 77 | 7.26 |
| 70 | 23 | 70 | 6.60 |
| TOTAL | | 1061 | 100.00 |

Table 1.2

Marital Status of Women Born 1969 - 1983

| MARITAL STATUS | T O T A L | % |
|---|---|---|
| Single | 838 | 79.0 |
| Single Mother | 37 | 3.5 |
| Common Law Wife | 157 | 14.8 |
| Married | 102 | 9.6 |
| Divorced | 3 | 0.3 |
| T O T A L | 1061 | 100.0 |

Table 1.3

Schooling Among Women Born 1969-1983

| CATEGORY | GRADES (YEARS) | NUMBER | % |
|---|---|---|---|
| No Schooling | 0 | 153 | 14.5 |
| Elementary School | 1-3 | 419 | 39.5 |
| | 4-6 | 416 | 39.3 |
| Junior High School | 7-9 | 48 | 4.5 |
| High School | 10+ | 25 | 2.3 |
| T O T A L | | 1061 | 100.00 |

Table 4.1

**Data Collection in the Adolescent Longitudinal Component+**

| Area | Measures | Periodicity |
|---|---|---|
| Census | Update on household structure, marital status, migration, etc. | Every 3 months for 1 year. |
| Anthropometry | Height, sitting height, weight; head, arm, waist, hip, thigh and calf circumferences; biacromial, bicristal knee, elbow and wrist breadths; biceps, triceps, subscapular, midaxillary, suprailiac, anterior thigh, and medial calf skinfolds. | Height and weight every 3 months for 1 year (5 occasions). The rest of the variables only at the start and end of the year of data collection. |
| Dietary Intakes | 24-hour recall surveys of individual intakes using INCAP's standard methods. | Every 3 months for 1 year. |
| Health and Morbidity | Morbidity recall over the past month using INCAP methods and questionnaire. | Once |
| Physical Activity Pattern | 24-hour activity questionnaire. | Once |
| Maturation | Age at menarche | Appropriate questions every 3 months for those not yet menstruating. |

+ Extensive socioeconomic data on the households are available for the period 1969-77. This information was collected again in 1988. The 1988 information was updated in 1992.

Table 4.2

**Stature (cm) of Guatemalan Females and NHANES Percentile Values**

| AGE YEARS | GUATEMALAN | | | NHANES PERCENTILES+ | | | |
|---|---|---|---|---|---|---|---|
| | n* | MEAN | SD | 50 | 25 | 10 | 5 |
| 9 | 193 | 125.6 | 5.9 | 135.6 | 130.5 | 127.5 | 125.7 |
| 10 | 302 | 130.6 | 6.9 | 141.6 | 136.3 | 132.2 | 129.5 |
| 11 | 254 | 135.7 | 7.3 | 148.4 | 142.3 | 138.1 | 134.7 |
| 12 | 244 | 141.7 | 7.2 | 154.6 | 149.6 | 145.2 | 143.0 |
| 13 | 215 | 145.5 | 6.7 | 158.8 | 155.1 | 151.1 | 149.1 |
| 14 | 165 | 148.2 | 6.1 | 160.8 | 156.8 | 153.0 | 151.0 |
| 15 | 176 | 150.0 | 4.9 | 162.7 | 158.8 | 155.2 | 152.8 |
| 16 | 142 | 150.4 | 4.6 | 162.3 | 157.7 | 153.6 | 151.4 |
| 17 | 155 | 150.1 | 4.7 | 162.3 | 159.2 | 155.5 | 153.2 |
| 18 | 132 | 149.8 | 5.0 | 163.1 | 158.8 | 154.8 | 152.3 |
| 19 | 115 | 150.6 | 5.2 | | | | |
| 20 | 102 | 150.4 | 5.2 | | | | |
| 21 | 90 | 149.5 | 4.8 | | | | |
| 22 | 101 | 150.4 | 5.2 | | | | |
| 23 | 66 | 149.6 | 5.7 | | | | |

* Number of anthropometric measurements

+ From Frisancho (1990); percentile values for 18 years are actually for the age range 18-24 years.

Table 4.3

**Weight (kg) of Guatemalan Females and NHANES Percentiles for Weight**

| AGE YEARS | GUATEMALAN | | | NHANES PERCENTILES+ | | | |
|---|---|---|---|---|---|---|---|
| | n* | MEAN | SD | 50 | 25 | 10 | 5 |
| 9 | 192 | 25.0 | 3.8 | 30.7 | 26.8 | 24.8 | 23.7 |
| 10 | 300 | 28.5 | 5.5 | 33.9 | 29.6 | 27.0 | 25.6 |
| 11 | 254 | 31.0 | 5.9 | 39.8 | 34.3 | 30.5 | 29.1 |
| 12 | 236 | 35.5 | 6.7 | 45.9 | 39.1 | 34.3 | 32.5 |
| 13 | 205 | 39.3 | 7.1 | 49.6 | 44.3 | 39.3 | 37.2 |
| 14 | 154 | 43.6 | 7.2 | 52.7 | 47.3 | 42.9 | 40.3 |
| 15 | 170 | 47.1 | 7.2 | 54.2 | 48.6 | 45.3 | 43.4 |
| 16 | 156 | 48.0 | 6.6 | 55.7 | 50.8 | 46.1 | 43.4 |
| 17 | 168 | 48.4 | 6.7 | 57.4 | 51.9 | 46.4 | 43.2 |
| 18 | 130 | 48.6 | 8.3 | 58.3 | 52.6 | 48.4 | 45.6 |
| 19 | 119 | 48.5 | 7.0 | | | | |
| 20 | 100 | 50.5 | 8.4 | | | | |
| 21 | 99 | 50.0 | 8.2 | | | | |
| 22 | 120 | 51.4 | 7.7 | | | | |
| 23 | 75 | 50.2 | 8.7 | | | | |

* Number of anthropometric measurements.

+ From Frisancho (1990); percentile values for 18 years are actually for the age range 18-24 years.

Table 4.4

Height Velocity* by Age in Guatemalan Females

| AGE (years) | N | HEIGHT VELOCITY PER MONTH* (cm/month) | HEIGHT VELOCITY PER YEAR (cm/year) |
|---|---|---|---|
| 9 | 56 | 0.477 (0.129) | 5.72 |
| 10 | 87 | 0.550 (0.159) | 6.60 |
| 11 | 66 | 0.512 (0.141) | 6.14 |
| 12 | 73 | 0.456 (0.187) | 5.47 |
| 13 | 62 | 0.403 (0.212) | 4.84 |
| 14 | 49 | 0.189 (0.176) | 2.27 |
| 15 | 53 | 0.122 (0.137) | 1.46 |
| 16 | 40 | 0.068 (0.084) | 0.82 |
| 17 | 51 | 0.045 (0.079) | 0.54 |
| 18 | 43 | 0.045 (0.129) | 0.54 |
| 19 | 35 | 0.039 (0.113) | 0.46 |
| 20 | 34 | 0.019 (0.059) | 0.23 |
| 21 | 32 | 0.014 (0.085) | 0.17 |
| 22 | 34 | 0.016 (0.092) | 0.19 |
| 23 | 21 | 0.039 (0.062) | 0.47 |
| TOTAL | 736 | 0.22 (0.27) | 3.12 |

* Mean and standard deviation of regression coefficients of height (cm) on age (months) within individuals.

Table 4.5

**Weight Velocity* By Age in Guatemalan Females**

| AGE (years) | N | WEIGHT VELOCITY PER MONTH* (cm/month) | PER YEAR (cm/year) |
|---|---|---|---|
| 9 | 56 | 0.26 (0.13) | 3.12 |
| 10 | 87 | 0.33 (0.19) | 3.96 |
| 11 | 67 | 0.36 (0.20) | 4.32 |
| 12 | 70 | 0.44 (0.25) | 5.28 |
| 13 | 60 | 0.40 (0.20) | 4.80 |
| 14 | 45 | 0.30 (0.21) | 3.60 |
| 15 | 51 | 0.20 (0.25) | 2.40 |
| 16 | 41 | 0.17 (0.28) | 2.04 |
| 17 | 52 | 0.01 (0.24) | 0.12 |
| 18 | 42 | 0.08 (0.25) | 0.96 |
| 19 | 32 | 0.04 (0.25) | 0.48 |
| 20 | 35 | 0.09 (0.26) | 1.08 |
| 21 | 32 | -0.02 (0.26) | -0.24 |
| 22 | 39 | -0.02 (0.27) | -0.24 |
| 23 | 20 | 0.00 (0.22) | 0.00 |
| TOTAL | 729 | 0.22 (0.27) | 2.64 |

* Mean and standard deviation of regression coefficients of height (cm) on age (months) within individual.

Table 4.6

**Height Velocity of Guatemalan Females Compared to Differences in Attained Height Between Age Groups in the NHANES Population (Frisancho, 1990).**

| Guatemalan Females | | NHANES | |
|---|---|---|---|
| Age (years) | Height Increment/year (cm) | Age interval (years) | Height difference between ages (cm) |
| 9 | 5.72 | 8 - 9 | 6.0<br>(6.4) |
| 10 | 6.60 | 9 - 10 | 6.8<br>(6.5) |
| 11 | 6.14 | 10 - 11 | 6.2<br>(6.0) |
| 12 | 5.47 | 11 - 12 | 5.8<br>(5.0) |
| 13 | 4.84 | 12 - 13 | 4.2<br>(3.1) |
| 14 | 2.27 | 13 - 14 | 2.0<br>(1.95) |
| 15 | 1.46 | 14 - 15 | 1.9<br>(0.75) |
| 16 | 0.82 | 15 - 16 | -0.4<br>(-0.2) |
| 17 | 0.54 | 16 - 17 | 0<br>(0.45) |
| 18 | 0.54 | 17 - 18 | 0.9<br>(0.30) |
| 19 | 0.46 | 18 - 30 | 0.3 |
| 20 | 0.23 | | |
| 21 | 0.17 | | |
| 22 | 0.19 | | |
| 23 | 0.47 | | |

Table 4.7

**Weight Velocity of Guatemalan Females Compared to Differences in Attained Weight Between Age Groups in the NHANES Population (Frisancho, 1990).**

| Guatemalan Females | | NHANES | |
|---|---|---|---|
| Age (years) | Weight Increment/year (kg) | Age interval (years) | Weight difference between ages (kg) |
| 9 | 3.12 | 8 - 9 | 3.8 (3.5) |
| 10 | 3.96 | 9 - 10 | 3.2 (4.6) |
| 11 | 4.32 | 10 - 11 | 5.9 (6.0) |
| 12 | 5.28 | 11 - 12 | 6.1 (4.9) |
| 13 | 4.80 | 12 - 13 | 3.7 (3.4) |
| 14 | 3.60 | 13 - 14 | 3.1 (2.3) |
| 15 | 2.40 | 14 - 15 | 1.5 (1.5) |
| 16 | 2.04 | 15 - 16 | 1.5 (1.6) |
| 17 | 0.12 | 16 - 17 | 1.7 (1.3) |
| 18 | 0.96 | 17 - 18 | 0.9 (1.0) |
| 19 | 0.48 | 18 - 30 | 1.1 |
| 20 | 1.08 | | |
| 21 | -0.24 | | |
| 22 | -0.24 | | |
| 23 | 0.00 | | |

Table 4.8

**Mean and Standard Deviation of Dietary Energy Intake by Age in Guatemalan Females**

| AGE (years) | n | ENERGY (Kcal) | |
|---|---|---|---|
| | | MEAN | S.D. |
| 9 | 55 | 1764 | 451 |
| 10 | 78 | 1710 | 353 |
| 11 | 72 | 1760 | 414 |
| 12 | 76 | 1894 | 456 |
| 13 | 68 | 1858 | 514 |
| 14 | 47 | 1878 | 507 |
| 15 | 49 | 1997 | 453 |
| 16 | 55 | 1787 | 437 |
| 17 | 56 | 1869 | 483 |
| 18 | 39 | 1928 | 434 |
| 19 | 44 | 1915 | 440 |
| 20 | 36 | 1921 | 437 |
| 21 | 32 | 1867 | 375 |
| 22 | 32 | 1950 | 343 |
| 23 | 16 | 1882 | 478 |

Table 4.9

**Percentage of Energy Intake Adequacy, According to Age and Weight Recommendations**

| AGE (years) | N | % OF RECOMMENDATIONS | |
|---|---|---|---|
| | | BY AGE* | BY WEIGHT |
| 9 | 55 | 88.0 | 101. |
| 10 | 78 | 84.5 | 87.1 |
| 11 | 72 | 80.0 | 116.1 |
| 12 | 76 | 85.9 | 113.5 |
| 13 | 68 | 84.5 | 100.2 |
| 14 | 47 | 85.4 | 89.0 |
| 15 | 49 | 90.8 | 106.2 |
| 16 | 55 | 81.2 | 93.2 |
| 17 | 56 | 85.0 | 96.2 |
| 18 | 39 | 87.6 | 95.3 |
| 19 | 44 | 87.0 | 100.6 |
| 20 | 36 | 87.3 | 100.8 |
| 21 | 32 | 84.9 | 98.9 |
| 22 | 32 | 88.6 | 99.7 |
| 23 | 16 | 96.3 | 96.3 |

* Source: Recommended Dietary Allowances, National Research Council 1990.

Table 4.10

**Percentage of Energy Intake Adequacy According to Age and Weight Recommendations by Age Group**

| AGE (years) | n | % RECOMMENDATIONS | |
|---|---|---|---|
| | | BY AGE | BY WEIGHT |
| 9-10 | 133 | 86.6 | 93.3 |
| 11-14 | 263 | 83.9 | 103.2 |
| 15-18 | 199 | 85.9 | 97.3 |
| 19-25 | 162 | 86.7 | 99.9 |

**Table 4.11**

**Median and 90th Percentile of Selected Nutrients in Guatemalan Females, Compared to Recommended Dietary Allowances**

| AGE (years) | n | VITAMIN A (Mcg RE) | | | n | VITAMIN C (mg) | | | n | THIAMIN (mg) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Median | 90th percentile | RDA | | Median | 90th percentile | RDA | | Median | 90th percentile | RDA |
| 9-10 | 133 | 605 | 1073 | 700 | 133 | 24 | 83 | 45 | 133 | 0.9 | 1.3 | 1.0 |
| 11-14 | 263 | 572 | 1113 | 800 | 263 | 32 | 102 | 50 | 263 | 1.0 | 1.5 | 1.1 |
| 15-18 | 199 | 521 | 1037 | 800 | 199 | 30 | 108 | 60 | 199 | 1.0 | 1.5 | 1.1 |
| 19-25 | 162 | 635 | 1242 | 800 | 162 | 29 | 137 | 60 | 162 | 1.0 | 1.5 | 1.1 |
| RIBOFLAVIN (mg) | | | | | NIACIN (mg) | | | | PHOSPHORUS (mg) | | | |
| 9-10 | 133 | 0.5 | 0.9 | 1.2 | 133 | 8 | 13 | 13 | 133 | 952 | 1531 | 800 |
| 11-14 | 263 | 0.6 | 1.0 | 1.3 | 263 | 9 | 14 | 15 | 263 | 1035 | 1513 | 1200 |
| 15-18 | 199 | 0.6 | 0.9 | 1.3 | 199 | 10 | 15 | 15 | 199 | 1072 | 1471 | 1200 |
| 19-25 | 162 | 0.6 | 1.0 | 1.3 | 162 | 10 | 15 | 15 | 162 | 1073 | 1455 | 1200 |
| IRON (mg) | | | | | CALCIUM (mg) | | | | DIETARY FIBER (g) | | | |
| 9-10 | 133 | 10 | 15 | 10 | 133 | 615 | 998 | 800 | 133 | 6 | 10 | |
| 11-14 | 263 | 10 | 17 | 15 | 263 | 704 | 1135 | 1200 | 263 | 7 | 11 | |
| 15-18 | 199 | 10 | 16 | 15 | 199 | 711 | 1097 | 1200 | 199 | 7 | 10 | |
| 19-25 | 162 | 11 | 17 | 15 | 162 | 714 | 1067 | 1200 | 162 | 7 | 11 | |

## Table 4.12

**Classification Criteria of Morbidity by Severity (Mild/Moderate/Severe) Reported in 15 Day Recall Questionnaire**

| A. ACUTE RESPIRATORY INFECTIONS | | |
|---|---|---|
| UPPER AIRWAYS | CODE | |
| Nasal Discharge<br>Productive Cough<br>Non-Productive Cough<br>Nasal Bleed<br>Ear Pain<br>Sore Throat<br>Herpetic Stomatitis | 011<br>014<br>310<br>311<br>042<br>312<br>313 | Any of the above symptoms alone or combined<br>+ Nothing = Mild<br>+ (94) Fever = Moderate<br>+ (93) Bed = Severe |
| LOWER AIRWAYS | | |
| Wheezing/Rales | 012 | + Nothing = Mild<br>+ (94) Fever = Moderate<br>+ (93) Bed = Severe |
| B. GASTROINTESTINAL DISORDERS | | |
| Abdominal Pain<br>Nausea<br>Simple Gastritis<br>Flatulence<br>Abdominal Bowel Sounds<br>Helminthic Infections | 029<br>306<br>307<br>308<br>309<br>314 | Any of the above symptoms alone or combined<br>+ Nothing = Mild<br>+ (94) Fever = Moderate<br>+ (93) Bed = Severe |
| Diarrhea<br>Diarrhea Containing Flecks and Blood Stained Mucus<br>Vomiting | 029<br>023<br>026 | Any of these alone or combined<br>+ Nothing = Mild<br>+ (94) Fever = Moderate<br>+ (93) Bed = Severe |
| C. SKIN DISORDERS AND TRAUMA | | |
| Minor Trauma and Dermatitis<br>Fractures-Burns-Major Trauma | 121<br>122 | Mild<br>Severe |
| D. NEUROLOGIC DISORDERS | | |
| Dizziness<br>Seizures<br>Other Neuropathies | 141<br>142<br>143 | Any of these alone or combined<br>+ Nothing = Mild<br>+ (94) Fever = Moderate<br>+ (93) Bed = Severe |

Table 4.13

Number of Subject-Days According to Health Status

| CATEGORY | NUMBER OF DAYS REPORTED | PERCENT |
|---|---|---|
| Healthy | 5025 | 61.4 |
| Mild | 1812 | 22.1 |
| Moderate | 965 | 11.8 |
| Severe | 388 | 4.7 |

Table 4.14

**Correlation Coefficients with Growth Rates for Females Under 17 Years of Age**

| Variables | Height Velocity | Weight Velocity | Age |
|---|---|---|---|
| Age (years) | -0.67 *** | -0.15 *** | ----- |
| Socioeconomic index | 0.03 | -0.01 | -0.03 |
| Energy intake (Kcal) | -0.08 * | -0.05 | 0.10 * |
| Energy Intake (%RDA) | 0.08 | 0.14 ** | -0.11 * |
| Protein Intake (% RDA) | 0.14 ** | 0.09 | -0.22 *** |
| Iron intake | 0.02 | 0.07 | 0.05 |
| Retinol Intake | 0.00 | 0.08 | -0.05 |
| Energy Expenditure (% relative to BMR=100) | -0.11 * | -0.05 | 0.10 * |
| Overall Morbidity | -0.04 | -0.10 | 0.02 |
| ARI (lower respiratory tract) | 0.00 | 0.00 | 0.00 |
| ARI (upper respiratory tract) | 0.03 | -0.01 | -0.09 |
| Gastrointestinal Infections | 0.01 | 0.02 | -0.06 |

* $P < 0.05$
** $p < 0.01$
*** $p < 0.001$

Table 4.15

Regression Coefficients on Height Velocity

| INDEPENDENT VARIABLES | UNIVARIATE MODELS | | MULTIVARIATE MODELS | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | MODEL 1 | | MODEL 2 | | MODEL 3 | |
| | Coef. (S.E.) | P | Coef. (S.E.) | P | Coef. (S.E.) | P | Coef. (S.E.) | P |
| Age | -0.86 (0.04) | 0.0001 | -0.81 (0.06) | 0.0001 | | | 0.87 (0.05) | 0.0001 |
| Energy Int (%RDA) | 0.01 (0.00) | 0.1058 | 0.001 (0.008) | 0.9365 | -0.02 (0.011) | 0.0693 | | |
| Protein Int (%RDA) | 0.01 (0.00) | 0.0032 | 0.009 (0.006) | 0.9726 | 0.02 (0.007) | 0.0035 | 0.00 (0.00) | 0.914 |
| Socioec Index | 0.09 (0.13) | 0.5138 | 0.005 (0.127) | 0.9676 | 0.12 (0.164) | 0.4554 | | |
| Energy Exp | -0.49 (0.18) | 0.0063 | -0.199 (0.166) | 0.2317 | -0.66 (0.210) | 0.0019 | -0.09 (0.15) | 0.5421 |
| Overall Morbidity | -0.20 (0.28) | 0.4897 | 0.087 (0.248) | 0.7259 | 0.08 (0.320) | 0.8051 | | |
| Adj R. Square | | | 0.4355 | | 0.0562 | | 0.4775 | |

## Table 4.16

### Regression Analysis on Weight Velocity

| INDEPENDENT VARIABLES | UNIVARIATE MODELS | | MULTIVARIATE MODELS | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | MODEL 1 | | MODEL 2 | | MODEL 3 | |
| | Coef. (S.E.) | P | Coef. (S.E.) | P | Coef. (S.E.) | P | Coef. (S.E.) | P |
| Age | -0.19 (0.06) | 0.0007 | -0.09 (0.08) | 0.2456 | | | | |
| Energy Int (%RDA) | 0.01 (0.005) | 0.0060 | 0.024 (0.01) | 0.0253 | 0.022 (0.01) | 0.0377 | 0.016 (0.006) | 0.0111 |
| Protein Int (%RDA) | 0.007 (0.004) | 0.0529 | -0.009 (0.007) | 0.1851 | .0.008 (0.007) | 0.2886 | | |
| Socioec Index | -0.037 (0.131) | 0.7764 | -0.146 (0.164) | 0.3769 | -0.131 (0.164) | 0.4244 | | |
| Energy Exp | -0.122 (0.181) | 0.4489 | -0.035 (0.215) | 0.8690 | -0.083 (0.210) | 0.6944 | | |
| Overall Morbidity | -0.518 (0.268) | 0.0539 | 0.517 (0.318) | 0.1058 | -0.528 (0.318) | 0.991 | -0.239 (0.3031) | 0.4293 |
| Adj R. Square | | | 0.01 | | 0.015 | | 0.02 | |

## Table 4.17

### Multiple Regression Models for Length at 3 Years and Height at Adolescence

| | MALES | | | | | | FEMALES | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Length at 3 years (cm) | | Height at adolescence (cm) | | Height at adolescence controlling for length at 3 years | | Length at 3 years (cm) | | Height at adolescence (cm) | | Height at adolescence controlling for length at 3 years | |
| Model Number | 1 | | 2 | | 3 | | 4 | | 5 | | 6 | |
| INDEPENDENT VARIABLES | β | p | β | p | β | p | β | p | β | p | β | p |
| Intercept | 48.77 | .000 | -76.48 | .000 | - | .000 | 53.15 | .000 | 71.17 | .000 | 30.53 | .001 |
| Supplement[a] | 1.97 | .000 | 1.15 | .111 | 109.40 | .111 | 2.83 | .000 | 2.03 | .001 | -0.27 | .631 |
| Maternal Height | 0.24 | .000 | 0.53 | .000 | -0.94 | .000 | 0.20 | .000 | 0.47 | .000 | 0.31 | .000 |
| SES Status (1975) | 0.60 | .025 | 0.54 | .202 | 0.29 | .965 | 0.24 | .320 | 0.74 | .030 | 0.55 | .050 |
| Diarrhea (%) | -0.07 | .015 | -0.04 | .416 | 0.01 | .454 | -0.06 | .063 | -0.05 | .280 | -0.01 | .893 |
| Dietary Intake[b] | 1.66 | .000 | 1.76 | .016 | 0.03 | .677 | 0.00 | .029 | 0.51 | .425 | -0.27 | .611 |
| Maturation (y) | ---- | ---- | 16.66 | .000 | 0.24 | .000 | ---- | ---- | 0.44 | .011 | 0.41 | .004 |
| Maturation Sq. | ---- | ---- | -0.44 | .000 | 15.16 | .000 | ---- | ---- | ---- | ---- | ---- | ---- |
| Length 3 years (cm) | ---- | ---- | ---- | ---- | 0.40 | .000 | ---- | ---- | ---- | ---- | 0.77 | .000 |
| | | | | | 0.98 | | | | | | | |
| Adjusted R Sq. | 0.25 | | 0.61 | | 0.76 | | 0.27 | | 0.31 | | 0.53 | |

[a] Supplement type: Atole = 1, Fresco = 0

[b] Dietary Intake: At or above gender specific median = 1, below gender specific Median = 0

Figure 3.1: Median Length in Males 0-36 Months Old: Rural Guatemalans and Mexican-Americans Compared to Reference Population (NCHS/WHO, 1983)

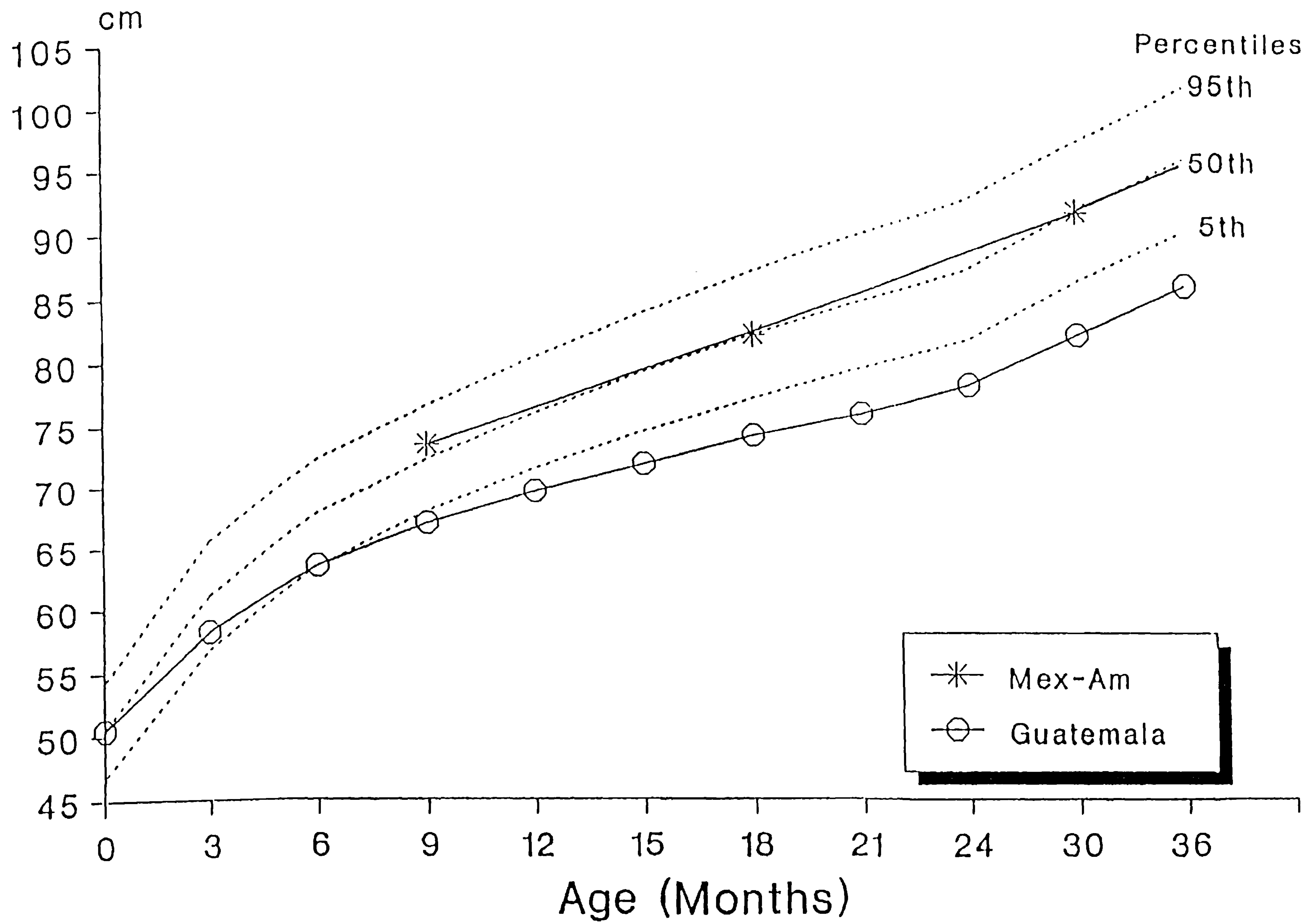
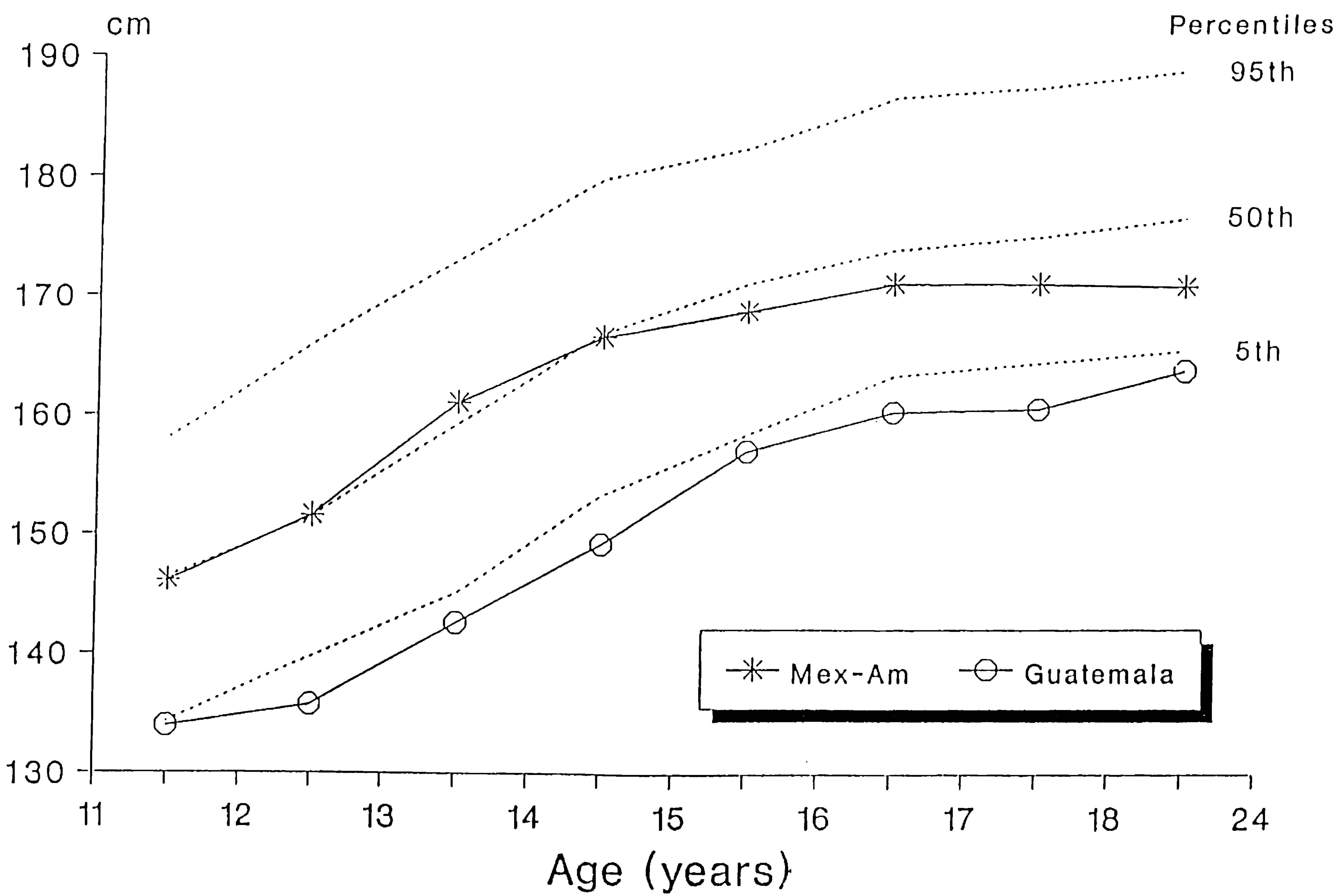
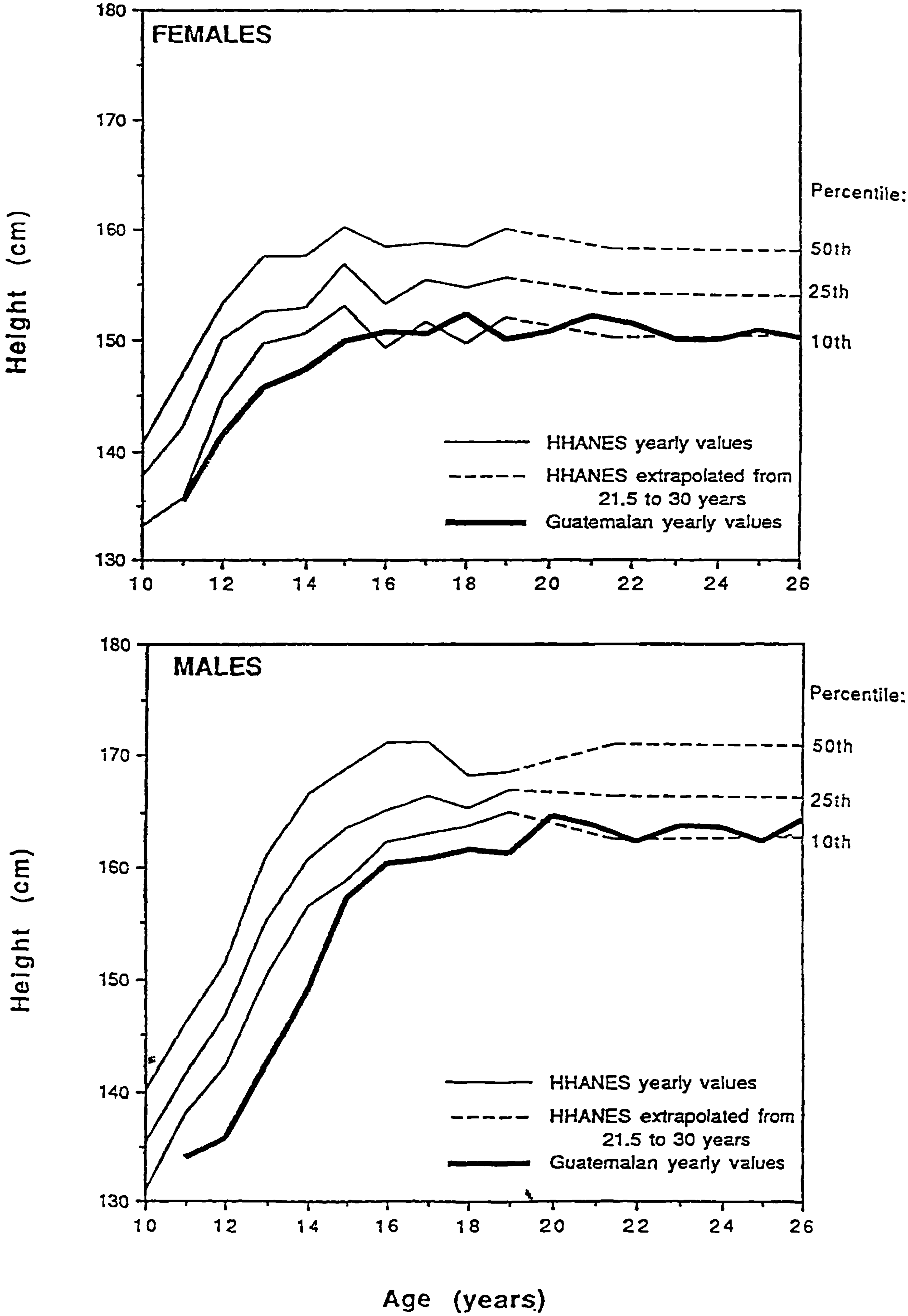


Figure 3.2: Median Height in Males 11-25 Years Old: Rural Guatemalans and Mexican-Americans Compared to Reference Population (Frisancho, 1990)




FEMALES
MALES
Height (cm)
Percentile:
50th
25th
10th
HHANES yearly values
HHANES extrapolated from 21.5 to 30 years
Guatemalan yearly values
Age (years)

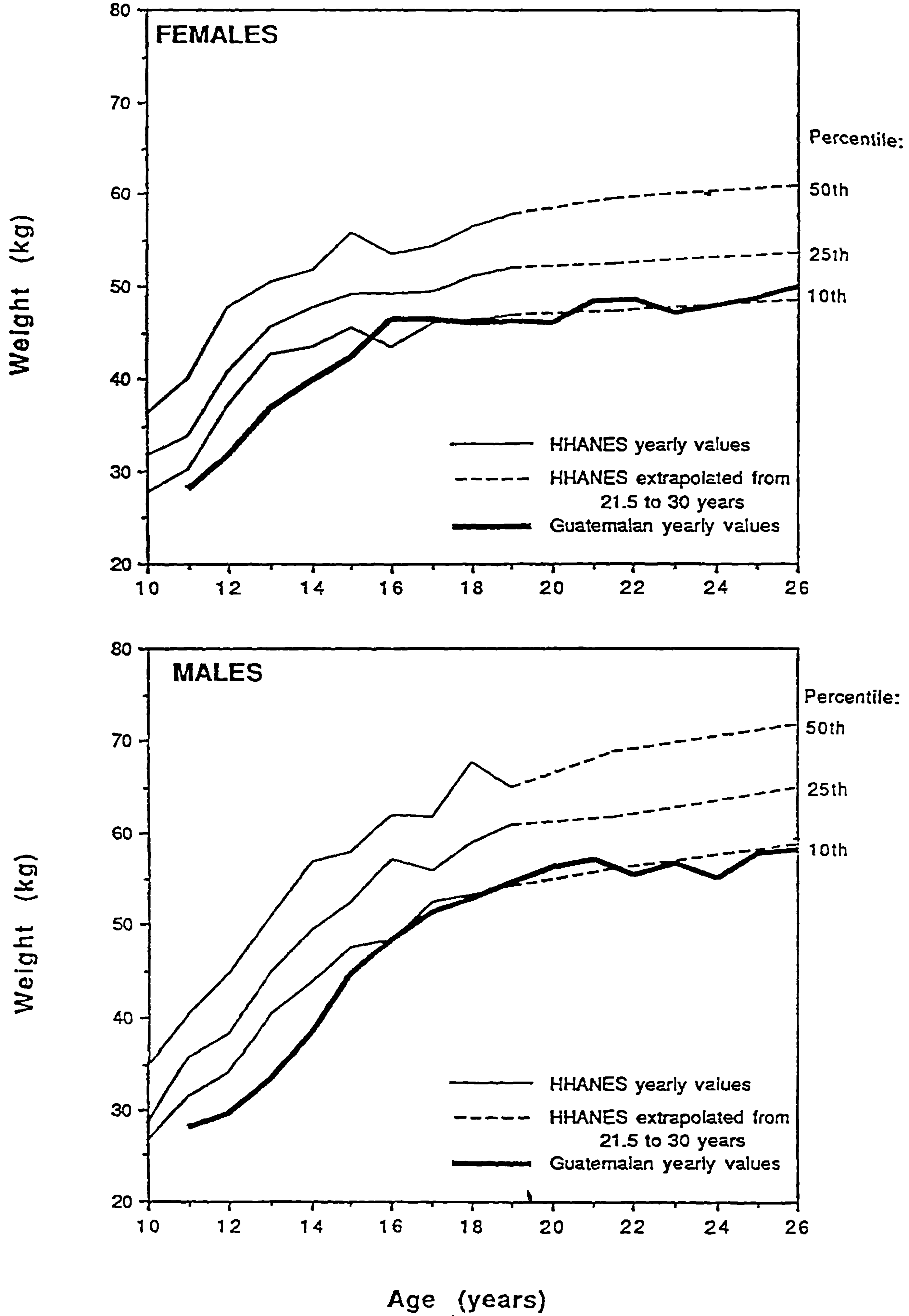

FEMALES
Weight (kg)
Percentile:
50th
25th
10th
HHANES yearly values
HHANES extrapolated from 21.5 to 30 years
Guatemalan yearly values
MALES
Weight (kg)
Percentile:
50th
25th
10th
HHANES yearly values
HHANES extrapolated from 21.5 to 30 years
Guatemalan yearly values
Age (years)

Figure 3.5: Body Mass Index of Rural Guatemalans Compared to HHANES Percentiles

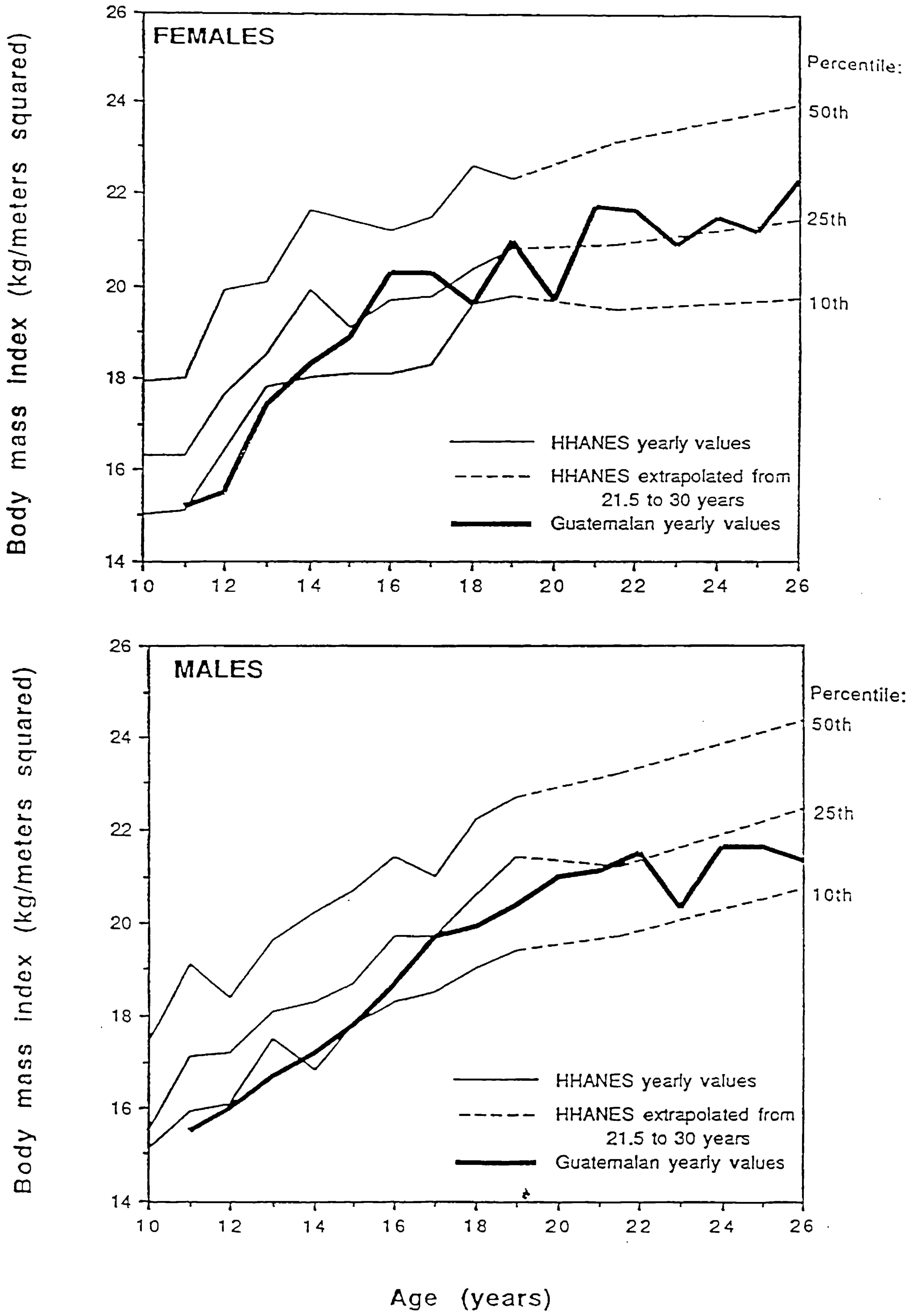


Figure 3.6: Subscapular Skinfold in Rural Guatemalans Compared to HHANES Percentiles

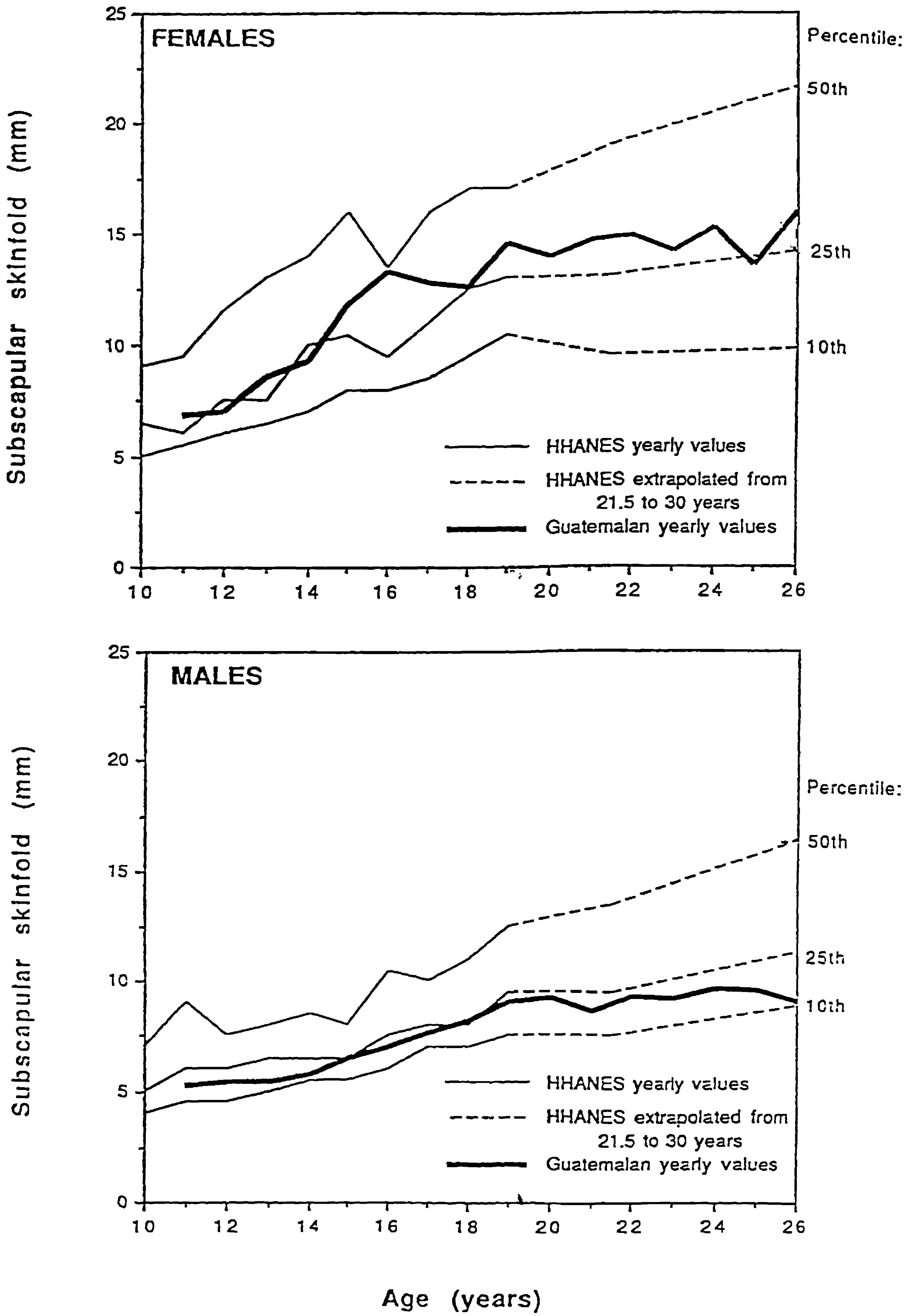


Figure 3.7: Biiliac Crest Breadth in Rural Guatemalans Compared to HHANES Percentiles

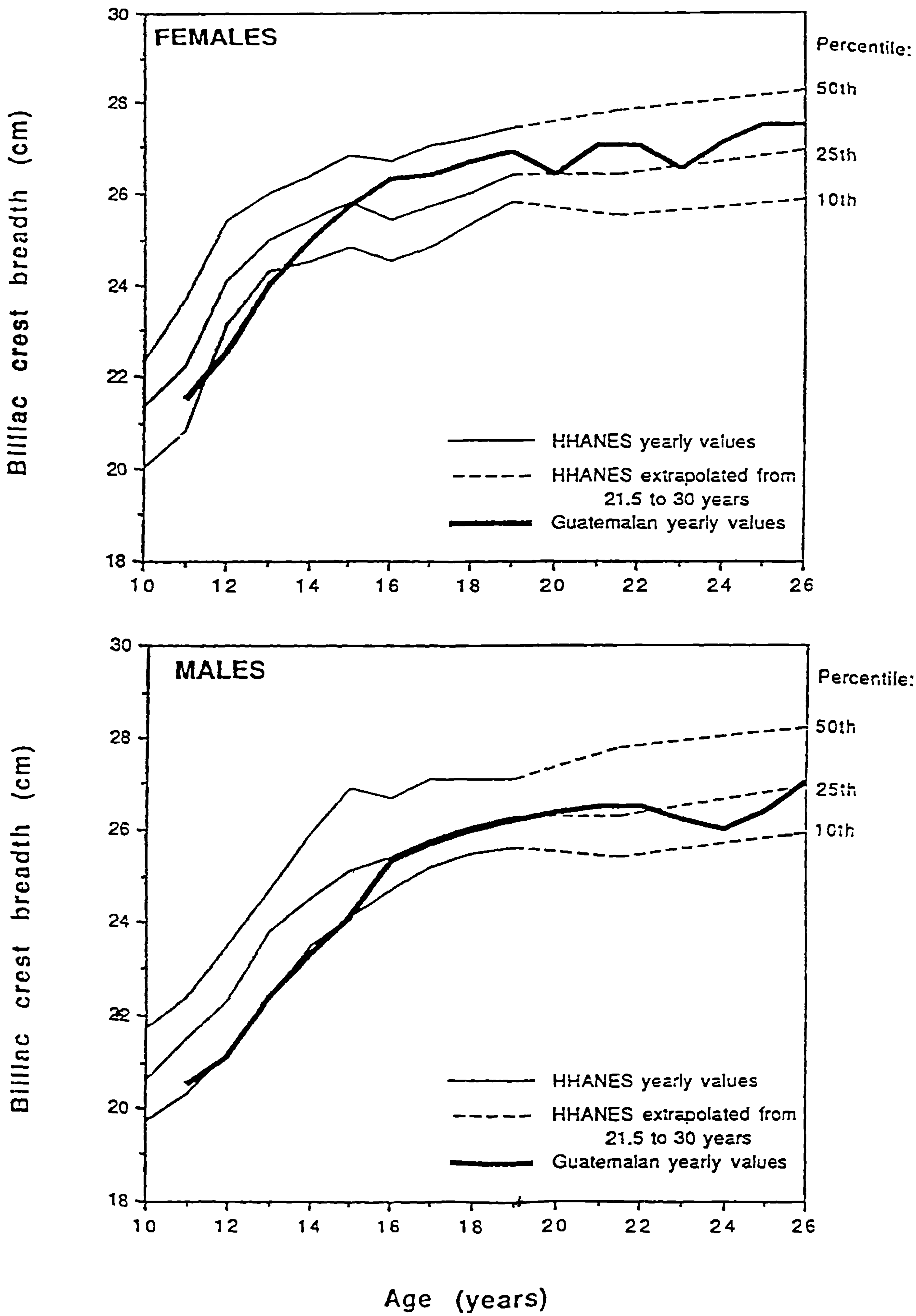


Figure 4.1: Mean Height by Age of Guatemalan Females Compared to NHANES' Data

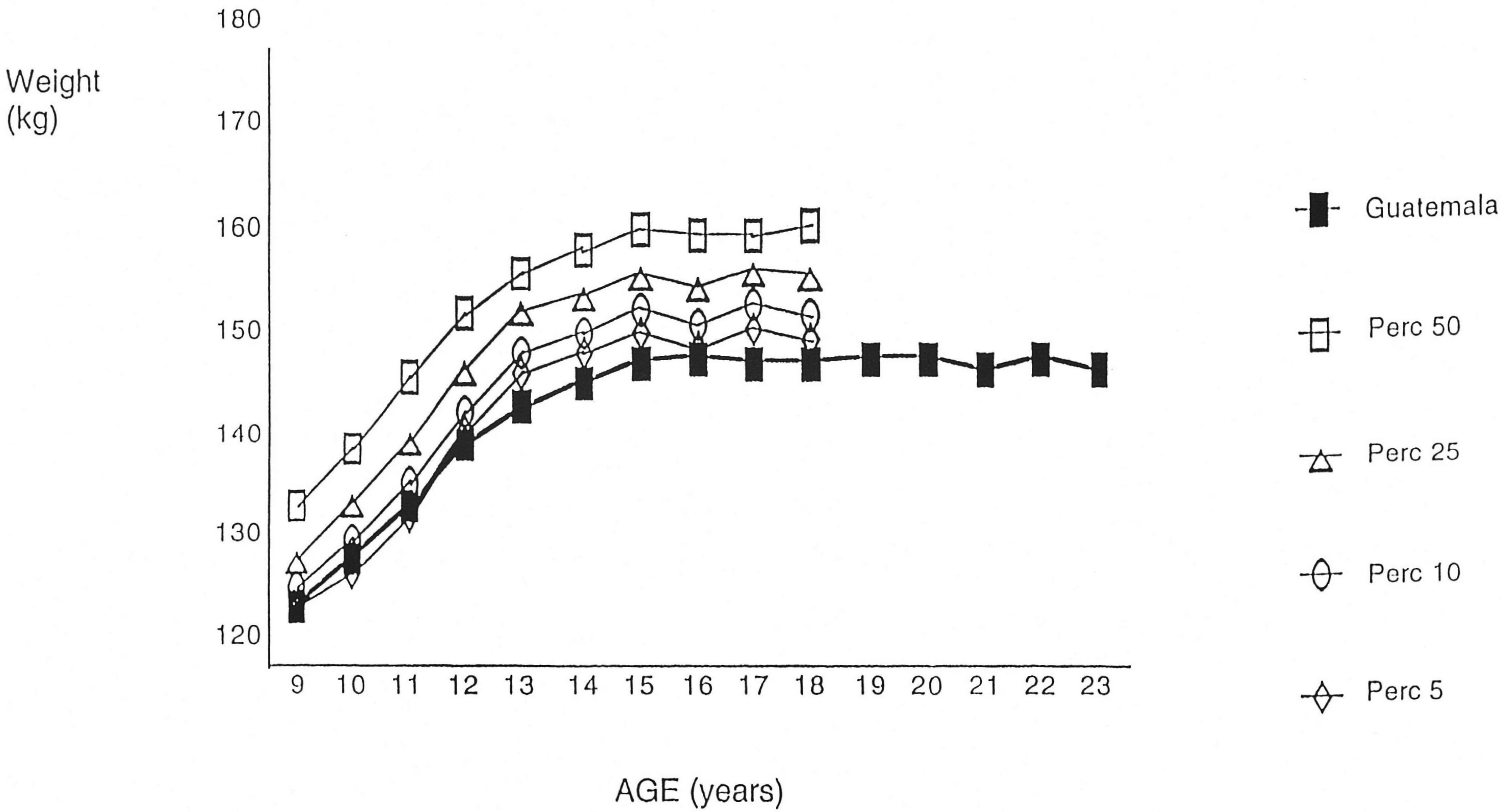


Figure 4.2: Mean Weight by Age of Guatemalan Females Compared to NHANES' Data

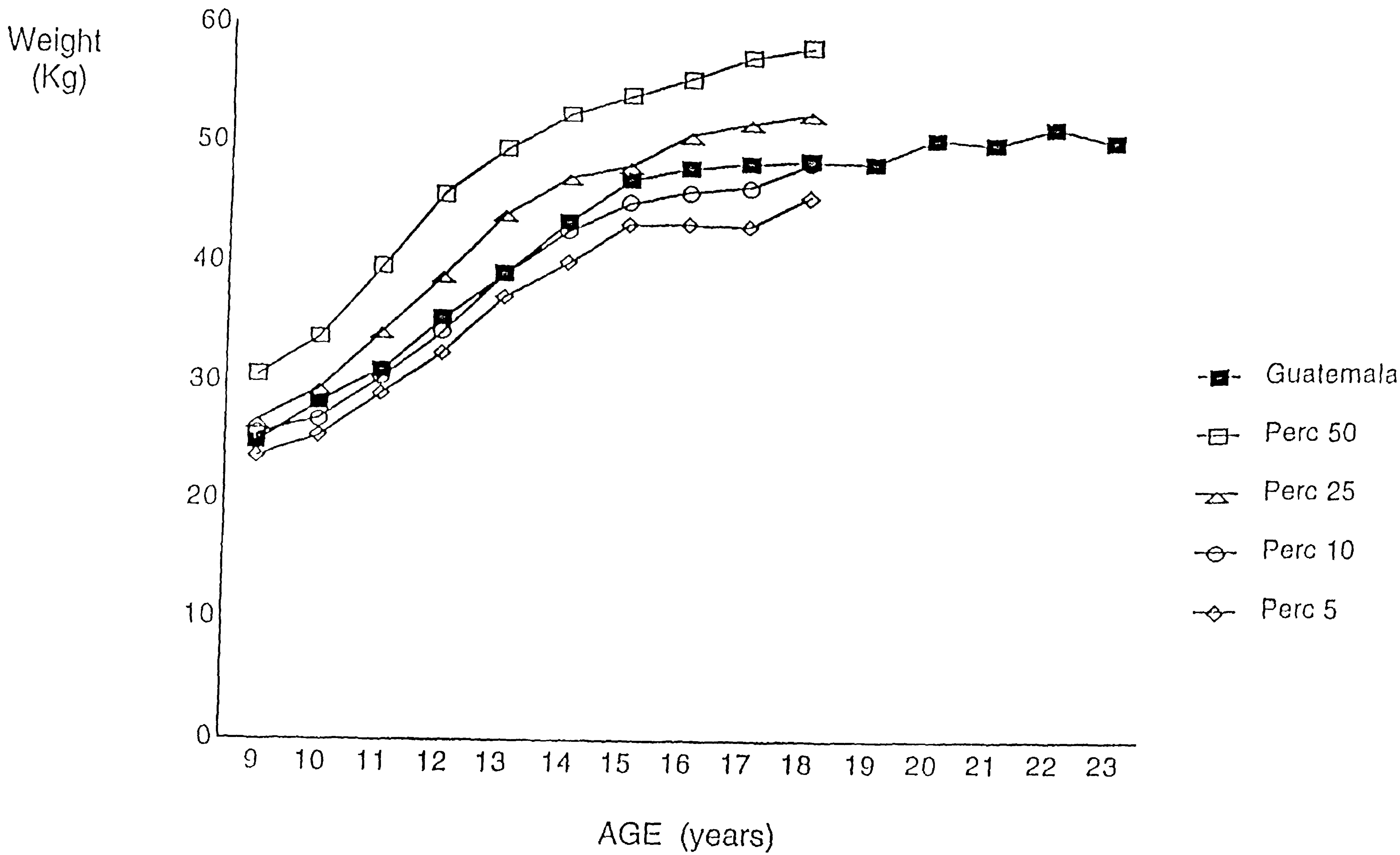


Figure 4.3: Height Velocity per Month

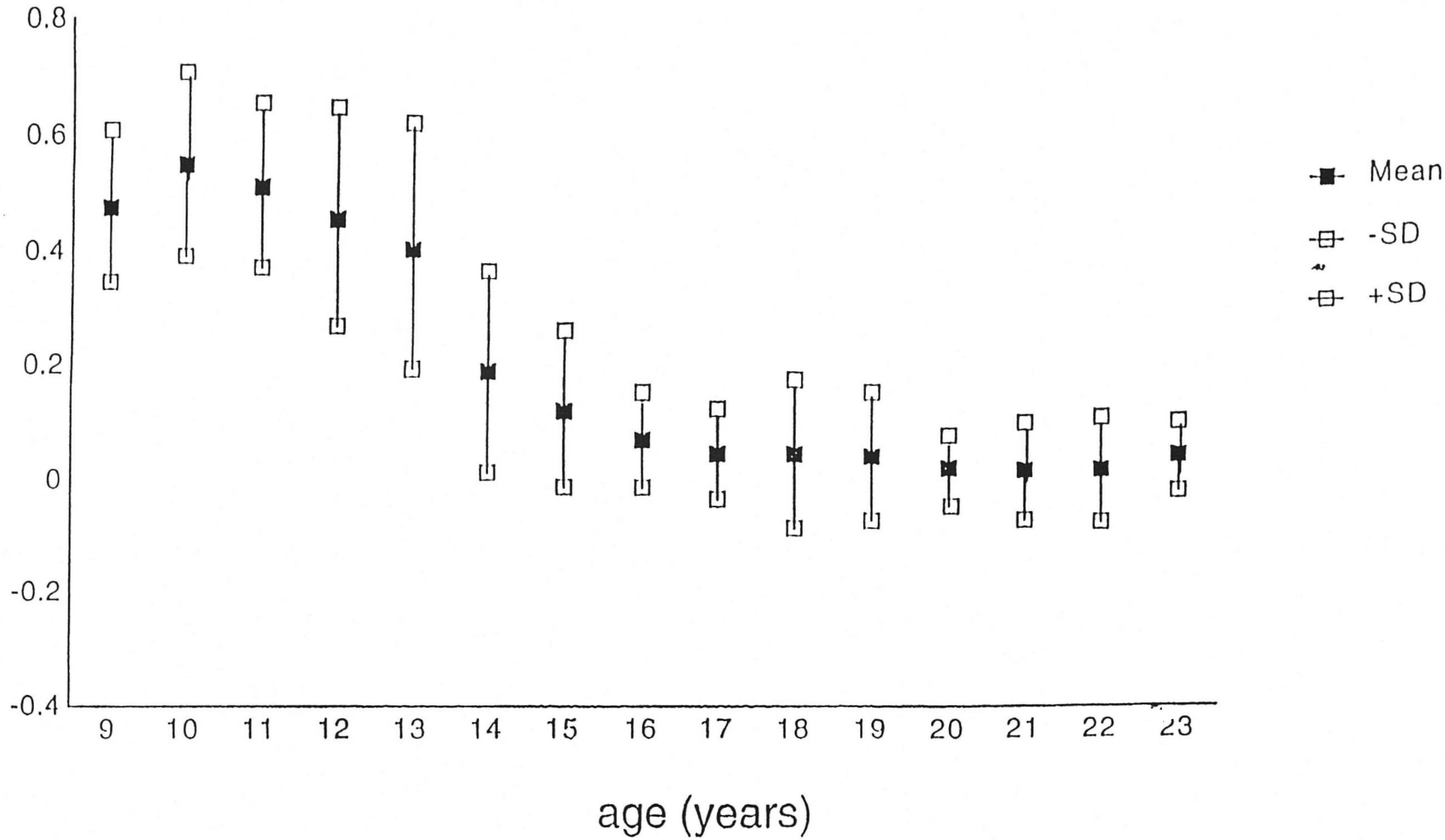


Figure 4.4: Weight Velocity per Month

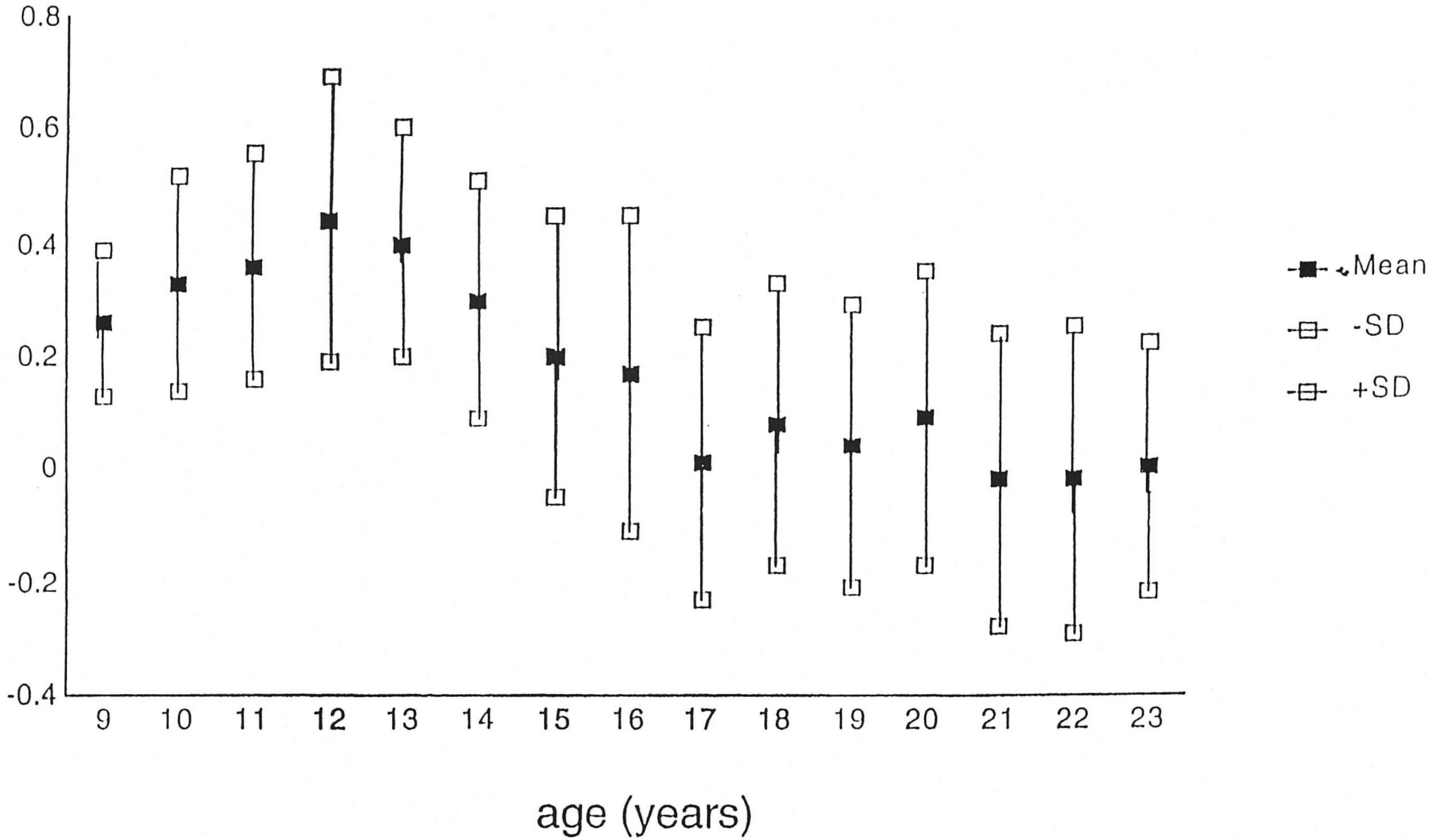


Figure 4.5: Percent of Energy Intake in Guatemalan Females Provided by Macronutrients

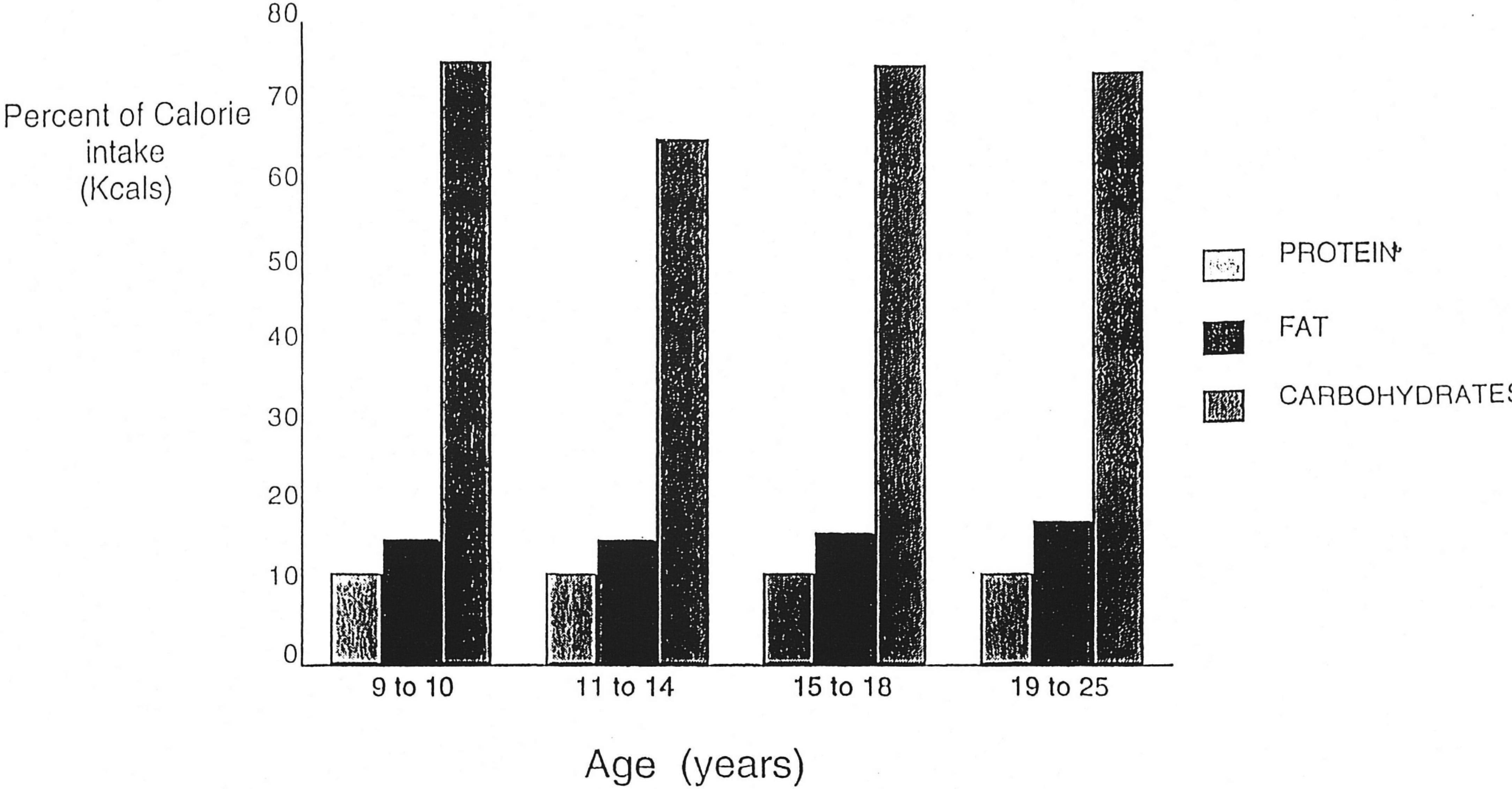


Figure 4.6: Percent of Animal and Vegetal Protein in Guatemalan Females

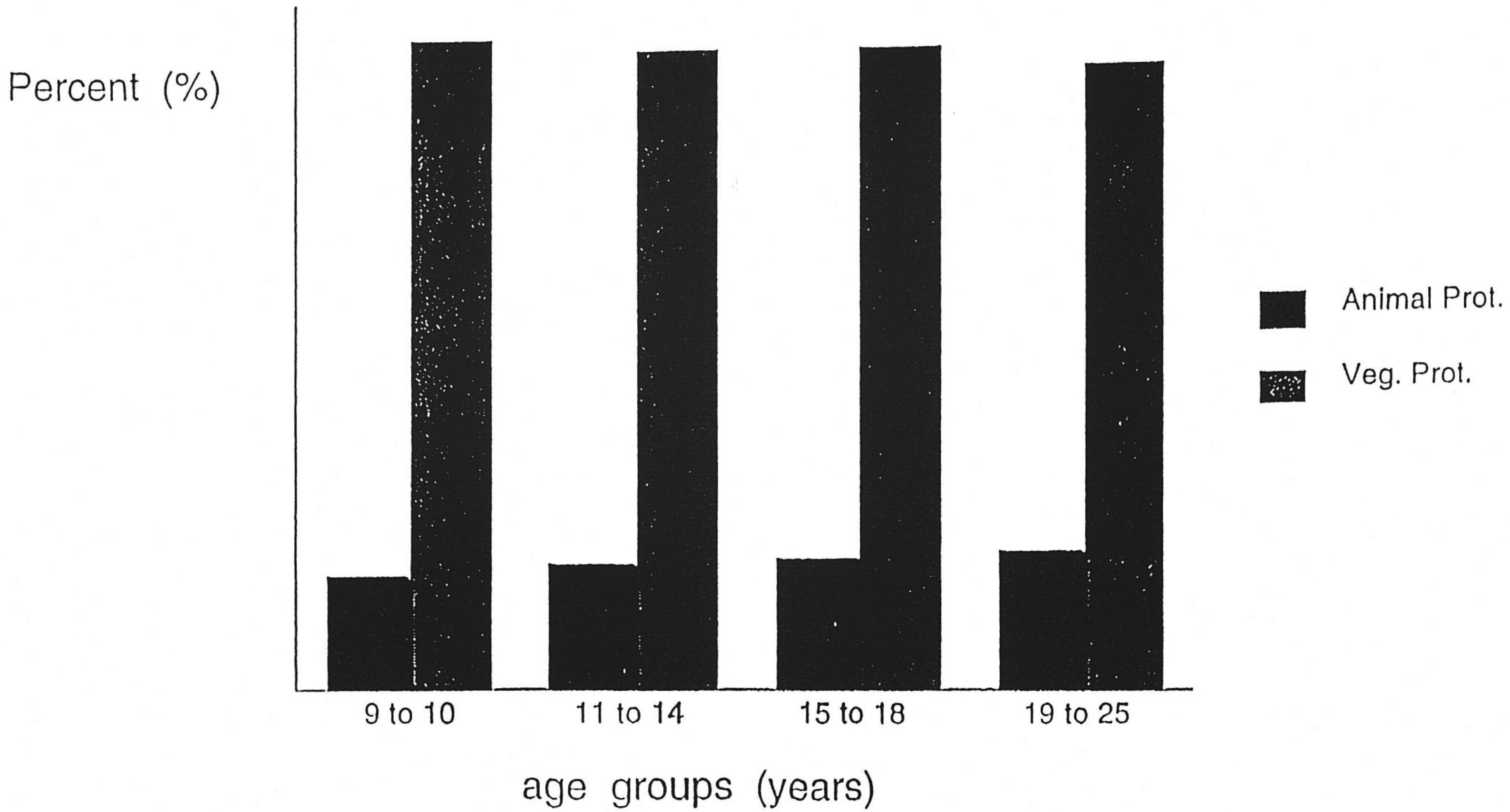


Figure 4.7: Median, 10th and 90th Percentiles of Vegetable Protein Intake Compared to Recommended Dietary Allowances Adjusted by Body Weight

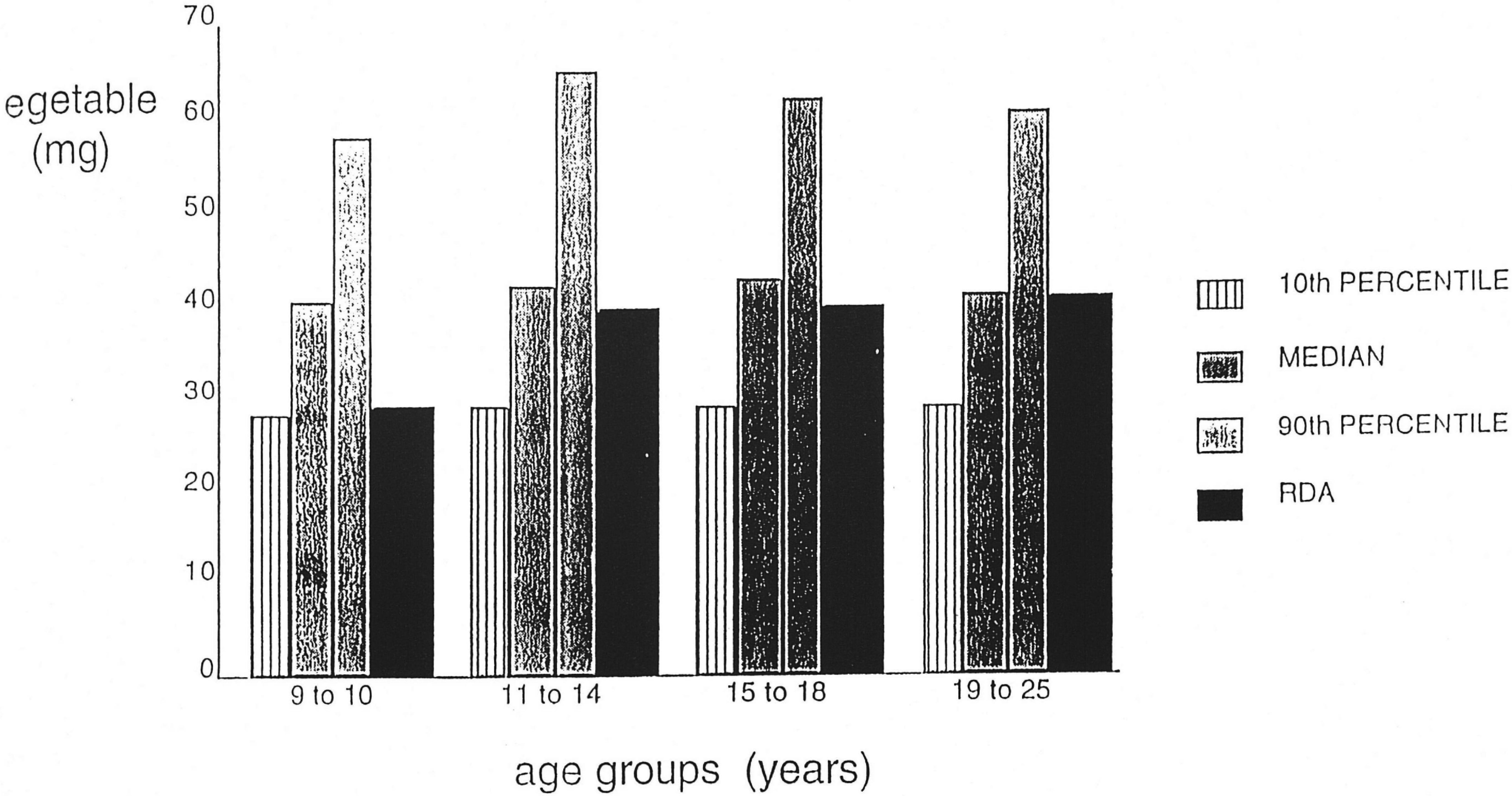


Figure 4.8 Median, 10th and 90th Percentiles of Animal Protein Intake Compared to Recommended Dietary Allowances Adjusted by Body Weight

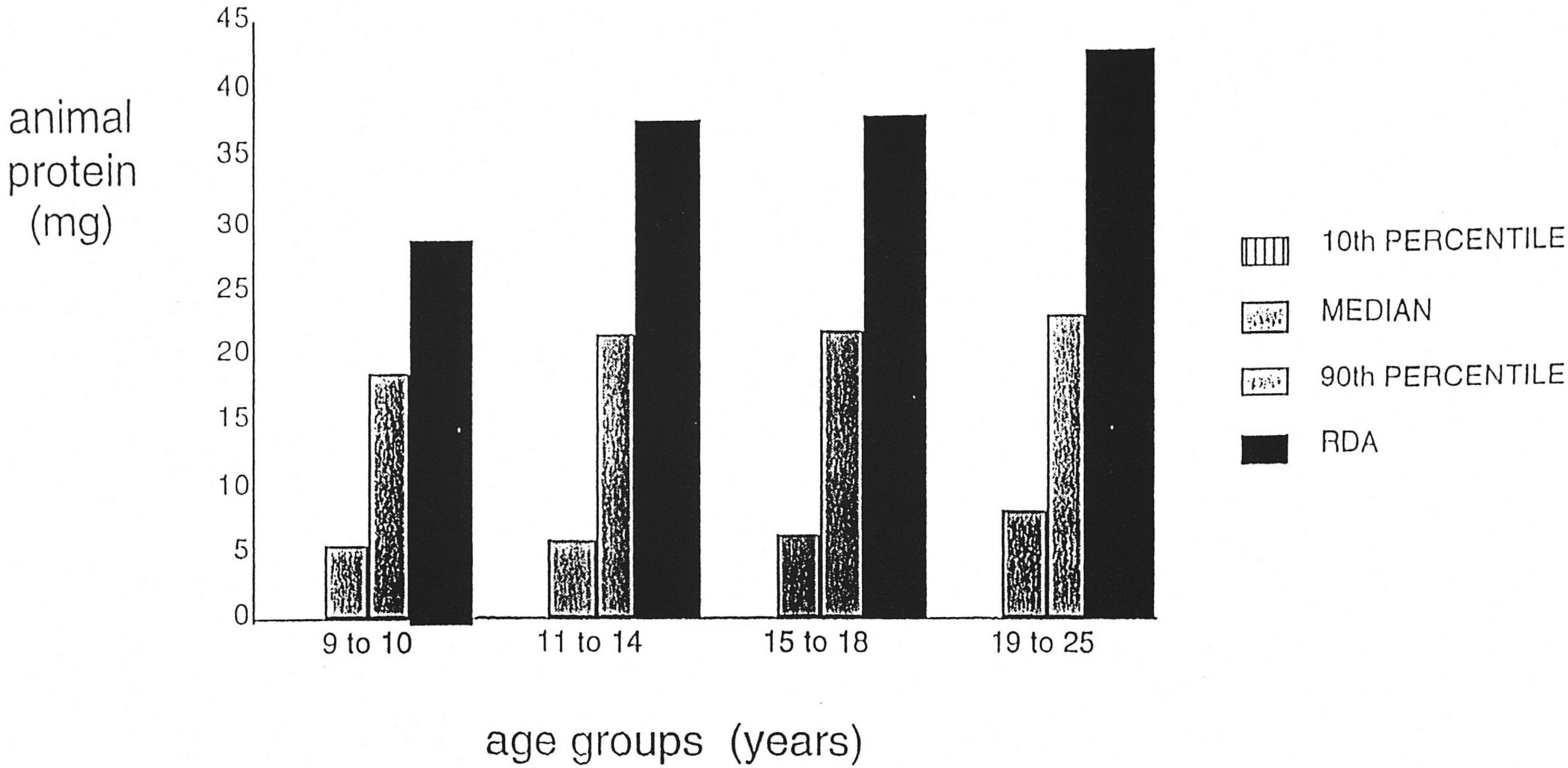


Figure 4.9 Percentile Distribution of % of Adequacy of Energy Expenditure Above BMR (100%) By Age Group

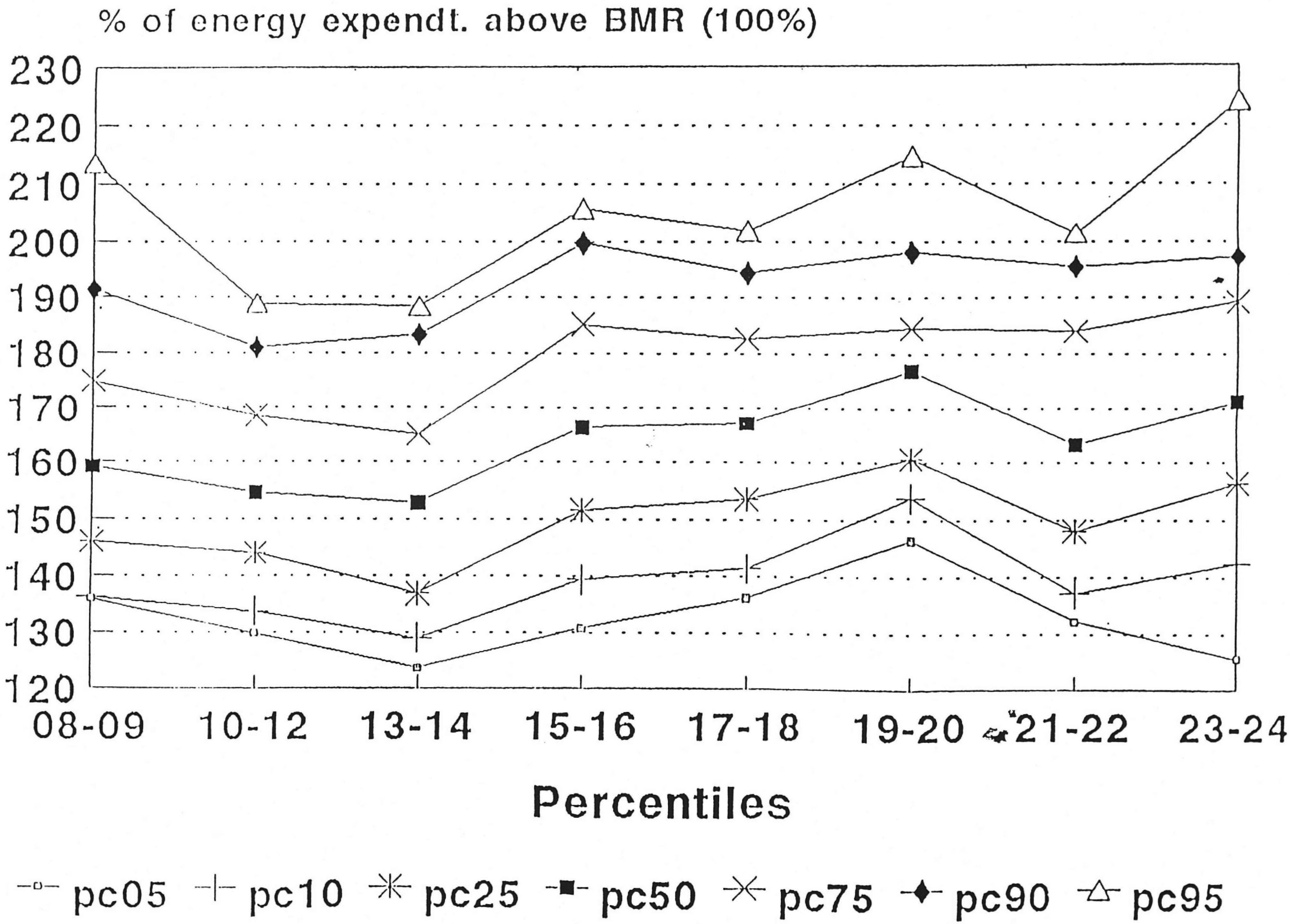


Figure 4.10 Percentage of Girls With Moderate Activity By Age Group

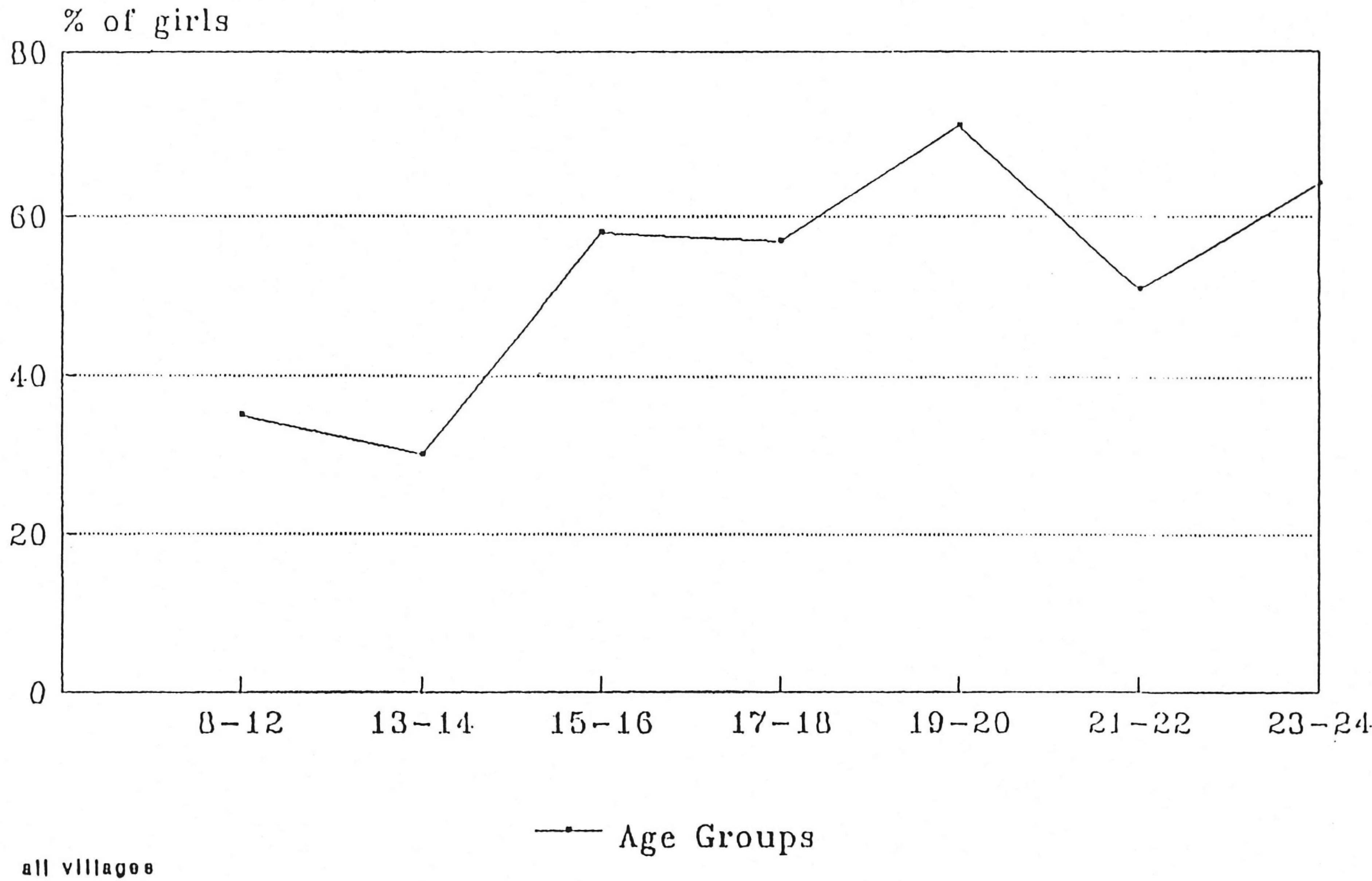
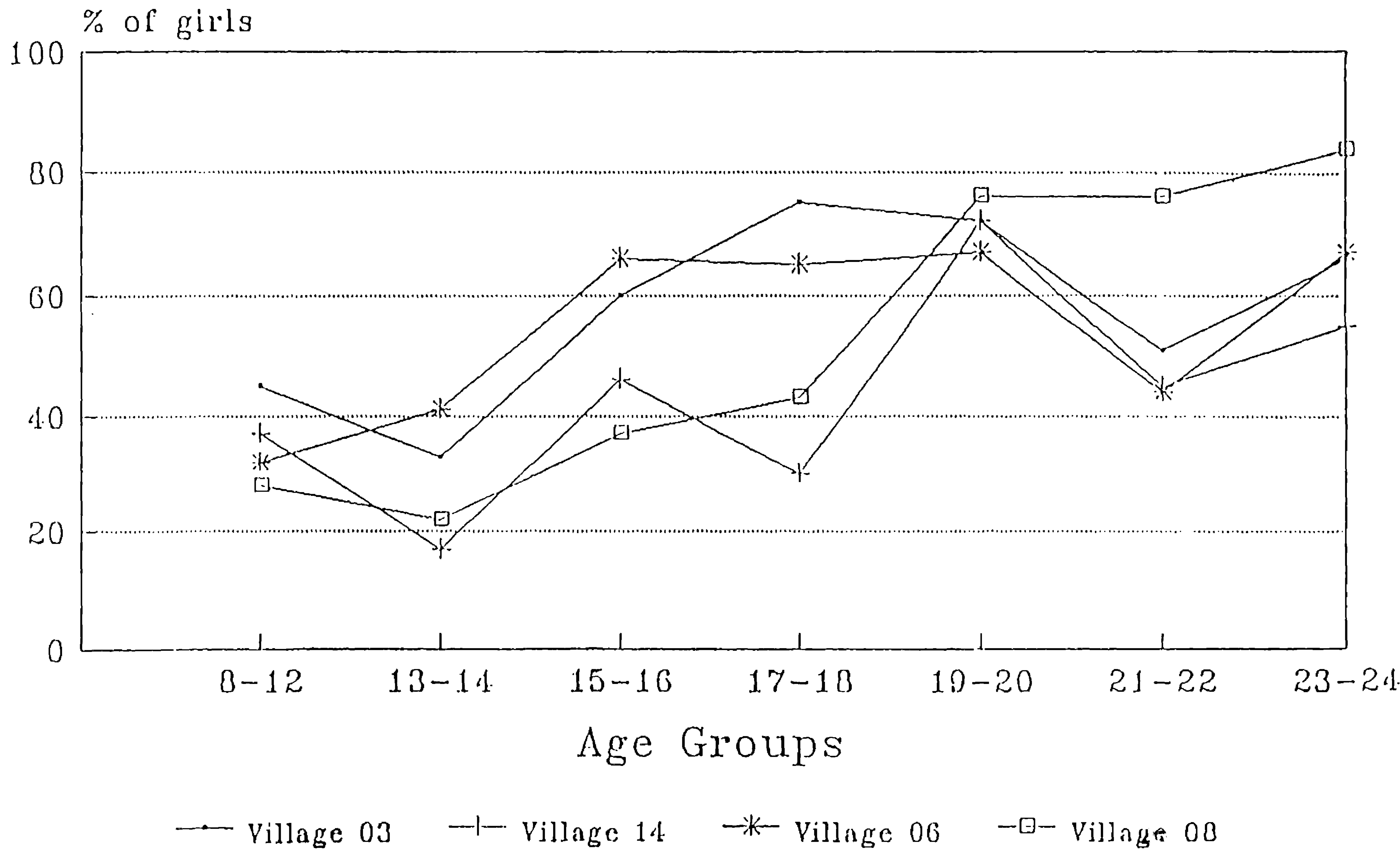


Figure 4.11 Percentage of Girls With Moderate Activity By Age Group and Village



## About the Nutrition of Adolescent Girls Research Program

The Nutrition of Adolescent Girls Research Program was established at ICRW in 1990 through a cooperative agreement with the Office of Nutrition, U.S. Agency for International Development. The premise for the Research Program was that the period of adolescence should be viewed as a window of opportunity for enhancing the lives of adolescents in developing countries, both for their present and future adult roles. Investments during adolescence can help girls and boys realize social and educational opportunities, manage their home and market responsibilities, and improve their nutritional and health status. However, little information is available to guide the formulation of policies and programs to address adolescent nutrition. The objective of the Research Program was to provide information on the many factors that are related to nutritional status, including physical growth, morbidity, food intake, energy expenditure, education level, family structure, intrahousehold food distribution, social and economic status, and self-perceptions. The program includes 11 research projects: five in Latin America and the Caribbean, four in Asia, and two in Africa.

**Publications from the Nutrition of Adolescent Girls Research Program**

The Nutrition and Lives of Adolescents in Developing Countries: Findings from the ICRW Nutrition of Adolescent Girls Research Program *by* Kathleen M. Kurz and Charlotte Johnson-Welch.

Investing in the Future: Six Principles for Promoting the Nutritional Status of Adolescent Girls in Developing Countries *by* Kathleen M. Kurz, Nancy L. Peplinsky, and Charlotte Johnson-Welch.

Addressing Needs and Opportunities: A Survey of Programs for Adolescents *by* Nancy L. Peplinsky.

Report Series:

No. 1. Nutritional and Health Determinants of School Failure and Dropout in Adolescent Girls in Kingston, Jamaica *by* S.P. Walker, S. Grantham-McGregor, J.H. Himes, S. Williams and F. Bennett. (Available also in Spanish.)

No. 2. Response of Endogenous Growth Factors to Exercise and Food Supplementation in Stunted Pubertal Girls in Guatemala *by* B. Torún, F. Viteri, M. Ramírez-Zea, M. Rodríguez, and K. Guptill.

No. 3. Nutrition, Health and Growth in Guatemalan Adolescents *by* R. Martorell, J. Rivera, and P. Melgar.

No. 4. Early Nutrition and Physical and Mental Development in Mexican Rural Adolescent Women *by* A. Chávez, C. Martínez, B. Soberanes, L. Domínguez, and A. Avila. (Available also in Spanish.)

No. 5. A Multidimensional Study of Nutritional Status of Adolescent Filipinas *by* A.T. Roldan, V. Bautista, and R. Manalo.

No. 6. A Study on the Factors Influencing Nutritional Status of Adolescent Girls in Nepal *by* S. Regmi and R. Adhikari.

No. 7. Understanding Gender-Differentiated Constraints to Philippine Farm Household Investments in Adolescents: Implications for their Nutritional Status *by* H.E. Bouis, M. Palabrica-Costello, O. Solon, and A.B. Limbo.

No. 8. Influence of Women's Social Status on the Nutritional Status of Adolescent Girls in Bénin *by* E. Alihonou, S. Inoussa, S. Vissoh, V. Capo-Chichi, C. Quenum, and A. Sagbohan. (Available also in French.)

No. 10 Study of the Factors that Influence the Nutritional Status of Adolescent Girls in Cameroon *by* K.M. Kurz and J. Ngo Som.

No. 11. Improving Nutritional Practices of Ecuadorean Adolescents *by* Y. de Grijalva and I. Grijalva. (Available also in Spanish.)

---