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Secular Changes in Physical Growth and Obesity among Southwestern American Indian Children over Four Decades

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Abstract

Background and Objectives—Most studies describing childhood obesity in the United States are based on cross-sectional surveys and do not include substantial numbers of American Indians (AI). Secular trends in height and weight reflect general health status. This study describes weight trends and transitions among AI children over a 43 year period.

Methods—Anthropometric data were obtained from a prospective study conducted in a southwestern US AI population (1965 through 2007). For cross-sectional analysis, 12,377 observations were available from 6,529 children across four birth cohorts (1955–64, 1965–74, 1975–84, 1985–94). Participants were stratified into three age groups: pre- (5–9 years), early (10–13) and late (14–17) adolescence. Longitudinal analyses included 1,737 children with one exam in each age group.

Results—In early and late adolescence, weight increased across birth cohorts. Prevalence of obesity among pre-adolescents was 17.5% (95% CI, 15.1%–19.9%) in the 1955–64 cohort, and 33.7% (95% CI, 30.1%–36.4%) in the 1985–94 cohort. 74% of children overweight in pre-adolescence in the 1985–94 cohort became obese by late adolescence; in the 1955–64 cohort, only 43% made this transition.

Conclusions—This study describes the rising prevalence of childhood obesity. Children obese in pre-adolescence remained obese in late adolescence, stressing the need for early intervention.

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CONFLICT OF INTEREST

The authors have no financial relationships relevant to this article to disclose. The authors have no potential conflicts of interest to disclose.

CONTRIBUTORS' STATEMENT

Ms. Vijayakumar designed the study and carried out the analyses, drafted the initial manuscript, and approved the final manuscript as submitted.

Mr. Wheelock and Ms. Kobes assisted in data analysis and critical review of the manuscript, and approved the final manuscript as submitted.

Drs. Nelson, Hanson, and Knowler obtained the study data, assisted in study design and analysis, critically reviewed the manuscript, and approved the final manuscript as submitted.

Dr. Sinha conceptualized the study, assisted in data analysis, drafted the initial manuscript, and approved the final manuscript as submitted.

Keywords

American Indian; Entrenched Obesity; Weight Transitions; Secular Trends; Preventions

INTRODUCTION

Secular changes in physical growth reflect intergenerational differences in both socioeconomic and environmental conditions within a population group.^{1,2} Trends in growth towards greater heights and weights occurred in most industrialized countries, including the United States (US), from the early nineteenth century until the mid-1960s, after which the secular trends became more variable. In the US, the mean age- and sex-specific heights were unchanged between the National Health Examination Survey (NHES) cycles II (1963 to 1965) and III (1966 to 1970) and the National Health and Nutrition Examination Surveys (NHANES) cycle II (1976–1980). While height increases in recent decades have stalled, a disproportionate increase in body weight has led to a rising prevalence of overweight and obesity among children and adolescents in all race and ethnic groups that include, non-Hispanic white, non-Hispanic black, Mexican-American and others.³ Childhood obesity carries significant risks for cardiovascular and metabolic disorders through adulthood, but the progression of obesity throughout childhood has not been studied in depth in a large population.

Data describing physical growth patterns among American Indian (AI) children and adolescents are particularly sparse, as they are not sufficiently sampled in the NHANES and are grouped into the “other” race/ethnicity category.⁴ The aim of this study is to describe the secular changes in growth parameters including weight, height and body mass index (BMI) over a 40-year period in a population of AI children in the southwestern US. The persistence of overweight and obesity in individuals who develop these conditions at a very young age was also assessed from longitudinal data.

METHODS

A longitudinal study of diabetes and related conditions was conducted in an AI community in the southwestern United States over a 43-year study period from 1965 through 2007; all participants, children and adults, were primarily of Pima Indian heritage.⁵ Residents of the community who were five years of age or older were invited to participate in biennial research examinations. The research study protocol included detailed medical history, anthropometric measures, and biochemical tests. Height and weight were measured by trained research personnel at each visit with subjects dressed in light clothing without shoes. Body mass index (BMI; kg/m²) was calculated from these measurements.

In our current study, we included children aged 5–17 years who had a complete set of anthropometric measures available for at least one study visit. This criterion yielded 6,529 individual participants available for analysis across four birth cohorts: 1955–1964, 1965–1974, 1975–1984, and 1985–1994.

We divided participants into three age categories that correspond to the following developmental stages: pre-adolescence (5–9 years), early adolescence (10–13 years) and late adolescence (14–17 years).⁶ Data from the first examination within each age category per individual were analyzed, yielding 12,377 observations across the 6,529 individuals. A sub-analysis of 1,737 children who had one exam in each age category (5,211 observations) was performed to examine intra-individual changes in height, weight and BMI. Age-sex-specific z-scores and percentiles were determined using the Centers for Disease Control and Prevention (CDC) growth charts for weight, height, and BMI.⁷ Weight categories were defined by the following BMI percentiles: normal weight (< 85th percentile), overweight (85th to 95th percentile), and obese (> 95th percentile).

The Institutional Review Board of the National Institute of Diabetes and Digestive and Kidney Diseases approved the longitudinal study. Written informed consent was obtained from parents at each examination and assent was obtained from the children.

Statistical analysis

Statistical analyses were conducted using the Statistical Analysis System (SAS 9.3; SAS Institute, Cary, NC). Population-specific percentiles for weight and height were calculated at the 1st, 5th, 50th, 95th, and 99th levels. The 1st and 99th percentiles for anthropometric measures have been included in our analysis, however we acknowledge that estimates of the extreme of centiles are imprecise because of small sample size and non-normal distribution. We examined temporal changes in BMI by plotting frequency distributions of BMI z-scores by age group and birth cohort. BMI z-scores were computed using the computer program and 2000 CDC growth charts for children between 24 and 239 months of age (cdc.gov/growthcharts/computer_programs.htm accessed October 4, 2016). We used the “modified z-score” that is similar to the usual z-score method (distance from the mean in standard deviation units). We did not use the unmodified z-score provided by the CDC program, because it severely compresses the frequency distribution of high z-scores such that very few have values >3. This resulted in a highly and artificially skewed distribution that does not describe the high BMI distribution in this population. Regression coefficients were estimated after weight, height, and age were standardized to a mean of 0 and a standard deviation of 1. Model fit for regression analysis of weight as a function of age, sex, height, and birth cohort was assessed by examining the ‘studentized’ residuals, and a test for linear trend was used to assess the effect of the four birth cohorts on weight group tracking. Weight gain per year was calculated in the longitudinal cohort by subtracting the weight in kilograms at the earlier exam from the weight in kilograms at the later exam, and dividing by the time in years between the two exams.

Incidence rates of obesity were calculated for each birth cohort as the number of new cases per 1,000 person-years at risk. The clinical outcome of obesity was defined as a BMI percentile greater than or equal to 95. Person-time was accumulated from the first non-obese examination after age 5 to the first examination at which obesity was observed or to the last examination before age 18, whichever came first. Out of the 6,529 individuals included in our study, 3,190 had at least two exams between the ages of 5 and 18 and were therefore included in incidence analysis. Incidence rates were standardized by the direct method of

age-sex adjustment as previously described⁸ using the age-sex distribution of the full study cohort of 12,377 observations as the standard. The Mantel “extension” chi-square test was used to test the incidence rates for linear trend with birth cohort.⁹

To compare obesity trend among the birth cohorts in our AI population with the national trend in childhood obesity, publicly available data corresponding to the same birth cohorts and age range from 6–17 years were extracted from multiple national cross sectional surveys: NHANES I (1971–74), NHANES II (1976–80), NHANES III (1980–1994) and NHANES: 1999–2000, 2001–2002, 2003–2004, and 2005–2006 (https://www.cdc.gov/nchs/nhanes/nhanes_questionnaires.htm).¹⁰ The anthropometric data including BMI were combined from these surveys, and restricted to 11,993 children of Caucasian race. Date of birth was available only for subjects in NHANES I and II; an approximate birth cohort was assigned for the rest based on age at exam and the midpoint of the study period.

RESULTS

Differences in BMI across birth cohorts

Anthropometric data from 3,584 pre-adolescent, 4,742 early adolescent and 4,008 late adolescent examinations were analyzed. Among pre-adolescent children, the median BMI percentile was 75 (IQR, 53–90) in the 1955–64 birth cohort, and increased to 86 (IQR, 60–98) in the 1985–94 cohort (Table 1). The magnitude of this cohort effect increased with age; by late adolescence, the median BMI percentile was 87 (IQR, 66–96) for the 1955–64 birth cohort and 97 (IQR, 85–99) for the 1985–94 birth cohort. Among late adolescents in the 1985–94 birth cohort, the IQR of BMI percentile lies entirely above the overweight BMI percentile threshold of 85, as defined by the CDC.

In each age group, the frequency distributions of BMI z-scores widened and shifted to the right, while remaining approximately normal, with increasing birth cohorts (Figure 1, top panel). The right shift resulted in much higher prevalence rates of obesity (bottom panel), especially in the 10–13 and 14–17 year age groups. The prevalence of obesity among 5–9 year olds increased from 17.5% (95% CI, 15.1%–19.9%) in the 1955–1964 birth cohort, to 33.7% (95% CI, 31.0%–36.5%) in the 1985–94 cohort; among 14–17 year olds, the prevalence increased from 30.1% (95% CI, 27.3%–33.0%) to 58.1% (95% CI, 54.9%–61.3%).

Secular changes in weight and height

Figure S1 illustrates the age and sex stratified weight and height changes over the four decades by plotting the weight and height at the 1st, 5th, 50th, 95th, and 99th percentile of each birth cohort. At the 1st and 5th percentiles in both sexes, weight and height remained stable across all birth cohorts. At the higher percentiles, a trend toward weight increase in later birth cohorts was observed, while there was only a slight increase in height across cohorts. The change in weight over time was highest at the 95th and 99th percentiles (Table S1), with an increase of about 20 kilograms across all birth cohorts among late adolescent males. A similar trend is noted among females, suggesting that in both sexes children became heavier, and the difference between the median and highest weight increased. The

upward shift in weight percentiles paralleled the shifts in the BMI z-score distributions (Figure 1). In a standardized linear regression model, weight ($\beta=0.15$ SD increase in weight/decade of birth year, $p<0.0001$), was more strongly associated with birth cohort than was height, ($\beta=0.08$ SD increase in height/decade of birth year, $p<0.0001$), after controlling for age and sex (Table S2).

Incidence of obesity in birth cohorts

To compare the development of obesity across birth cohorts the incidence rate of obesity within each cohort was calculated for 3190 children with at least one follow-up exam (Table S3). Mean age at baseline was 8.6 years, with a mean follow-up time of 5.7 years. The age-sex adjusted incidence rate rose from 32 cases per 1,000 person-years in the 1955–64 cohort to 72 cases per 1,000 person-years in the 1985–94 cohort, a significant linear trend ($p<0.0001$).

Longitudinal changes in weight group

Of the 1,737 children with research exams in all three age categories, 462 (27%) were obese and 300 (17%) were overweight at the first exam. Of the obese children, 433 (94%) were still obese at the second exam, and 420 (91%) were obese at the last exam. Only 42 (9.1%) children who were obese at ages 5–9 decreased their weight status to overweight or normal weight by ages 14–17 (Figure 2). Of those children who were overweight at the first exam, 161 (54%) were obese by the second exam, and 181 (60%) were obese by the last exam, compared with only 54 (18%) who were normal weight by the last exam. Thus, being overweight at age 5–9 years increases the likelihood of persistent overweight and obesity in adolescence.

Persistent overweight or obesity was even more pronounced in later birth cohorts (Figure 2). In the 1955–64 birth cohort, 43% of overweight children in pre-adolescence were obese by late adolescence; by 1985–94, that figure increased to 74%. Figure 2a illustrates that the first transition from pre- to early adolescence was associated with a greater trend towards obesity across birth cohorts than the second transition in Figure 2b, suggesting that this first transition plays an important role in the increased prevalence of obesity. Rate of weight gain varied with birth cohort (Table S4); from pre- to late adolescence, girls in the 1955–64 birth cohort gained a median of 4.99 kg/year, while girls in the 1985–94 cohort gained a median of 6.26 kg/year. For boys the corresponding median rates were 5.50 kg/year and 7.14 kg/year, respectively.

Obesity trend compared with NHANES: Figure S2 shows the median BMI across the different birth cohorts in males (S2a) and females (S2b) among our AI cohort and the Caucasians in the NHANES national surveys. Both groups demonstrate a rising trend in obesity over the decades, while the BMI medians were higher in the AIs in all age, sex, and birth year groups.

DISCUSSION

This study describes the secular changes in growth among AI children and adolescents in a southwestern community over four decades. Within each age and sex category, the

distribution of height remained fairly stable across birth cohorts; in contrast, the distribution of weight widened with each birth cohort, as the 95th and 99th percentile of weight rose faster than the lower percentiles. For boys ages 14–17, the median height rose by only two centimeters from 1955–64 to 1985–94, but the median weight rose by almost twenty kilograms. These secular trends are reflected by an increase in the prevalence of obesity in all age categories, and an overall shift in BMI towards higher CDC-standardized z-scores, indicating that not only is the prevalence of obesity rising, but the heaviest children are getting heavier. Similar secular trends in obesity in recent decades have been observed among Navajo youth, in whom obesity begins early in childhood.¹¹ Prevalence of obesity among native population groups such as Canadian Aboriginal youth and adults has been shown to be higher compared to the rest of the population.¹² In a subset of children for whom we had follow-up data, those who were normal or overweight at ages 5–9 in the later birth cohorts were more likely to become obese than their counterparts in the earlier cohorts. Even within the normal weight category, children in the 1985–94 cohort gained more weight than their counterparts in 1955–64 between ages 5–9 and 10–13. This increase in weight gain is also reflected in the incidence rate of obesity, which is over twice as high in the latest birth cohort compared with the earliest birth cohort. When compared with Caucasian children and adolescents in the US stratified by age and birth groups, a similar trend in obesity was observed in both groups although the magnitude was higher among our AI population.

In many developed countries, increases in adult height that were attributed to improved nutrition and health practices plateaued after World War II.¹³ We similarly found little increase in height among southwestern AI study participants in recent years. This parallel may suggest that the social and environmental obesogenic factors observed nationally are also at play in this study population. “Toxic food environments,” characterized by cheap calorie-dense fast food, sugar-sweetened beverages, and processed foods, may have contributed to the increasing risk of obesity, which is further compounded by lack of access to safe play areas, and increased screen time.¹⁴ These factors have promoted rapid weight gain, especially among minority children.¹⁴

Our study extends past findings by showing that obesity is present in children as young as 5–9 years of age, and that the majority of these children remain obese throughout adolescence. Children who were overweight or obese at ages 5–9 gained more weight each year than those who were normal weight at ages 5–9, further studies are needed to assess if interventions to treat early childhood obesity is effective in helping overweight or obese children transition back to a normal weight. Weight group tracking is stronger in the later birth cohorts, suggesting that the more recent sociocultural environment is more obesogenic. Similar findings of positive weight tracking are observed in other studies, which also report an inverse relationship between socioeconomic prosperity and weight gain.^{15,16} Entrenched obesity that emerges at a very young age and persists into adolescence and even adulthood is more common among minority and disadvantaged children.¹⁷

The dramatic increase in weight and sharp rise in obesity observed in this study population may reflect rapid lifestyle changes. The post-World War II era saw a shift from an indigenous to a Western diet, which was associated with increased risk for metabolic

abnormalities, including type 2 diabetes mellitus, especially among the youth in this southwest AI population.^{18,19} Earlier studies have attributed the post-World War II surge in the prevalence of obesity among adults in this population to increasingly Westernized lifestyles.²⁰ Our study shows a continued pattern of increased risk for childhood obesity in more recent birth cohorts, extending previous observations through 1998 of increasing age-sex-specific percentiles of weight relative to height and age.²¹ Several studies have also shown that the risks for obesity in childhood begin with maternal diabetes, suggesting a potential link between the concurrent rise in adult diabetes and childhood obesity.^{22–25}

A strength of this study lies in the large number of observations over four decades, including many participants with longitudinal follow up from early childhood through late adolescence. Study limitations include a lack of data on children below five years of age. Generalizability of the findings in this AI community in the southwestern US is uncertain, but an increasing prevalence of obesity has been observed in virtually all racial and ethnic groups worldwide, suggesting that our findings may be relevant to other populations.

Childhood obesity is associated with an increased risk for many comorbid conditions, and has long-term adverse health outcomes. A recent study in the same AI population showed that BMI in 5–19 year olds is a significant predictor of type 2 diabetes, independent of 2-hour post-load plasma glucose levels, and rates of premature mortality (before age 55 years) from endogenous causes were more than double among those in the highest quartile of BMI than among those in the lowest quartile.^{26,27} Previous studies have demonstrated an alteration in cardio-metabolic risk measures in preschool and middle school children in parallel with shifts in BMI category, confirming that biochemical changes including hyperglycemia and dyslipidemia as well as fatty liver buildup occur acutely in the very young.^{28,29} Further, analyses from the Bogalusa Heart Study demonstrated a direct positive relationship between childhood obesity and increased risk factors for coronary heart disease in adulthood.³⁰ These findings underline the importance of understanding the patterns of obesity and weight gain in children, and developing preventive and therapeutic strategies.

In a southwestern AI population, the prevalence of obesity was higher in more recent birth cohorts; children in these cohorts also had a higher likelihood of becoming obese at an earlier age and remaining obese throughout adolescence. Increasing incidence rates of obesity indicate that childhood obesity preventive measures in this population need to be strengthened, and should be targeted at pre- and early adolescence.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

AI	American Indian
BMI	Body Mass Index
CDC	Centers for Disease Control
NHANES	National Health and Nutrition Examination Surveys
US	United States
IQR	Interquartile Range
SD	Standard Deviation

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WHAT'S KNOWN ON THIS SUBJECT

Little is known about trends in obesity among American Indian children; the majority of studies describing changes in growth patterns among children in the United States are based on national surveys that are cross-sectional and do not target this population group.

WHAT THIS STUDY ADDS

This is the first longitudinal study describing obesity trends among American Indian children over such a long continuous study period (43-years) and shows a rising trend in obesity across birth cohorts; it also describes transitions between weight categories over time based on birth cohorts in this population that may reflect national trends.

Figure 1. Distributions of BMI Measures by Birth Cohort

Frequency distributions of BMI z-scores in pre- (A), early (B), and late (C) adolescence (ages 5–9, 10–13, and 14–17 years); frequency of weight groups defined by BMI percentiles in pre- (D), early (E), and late (F) adolescence. BMI z-scores and percentiles are referenced to the 2000 CDC age-sex specific BMI standards.

Figure 2. Weight Status Transitions across Age Groups, by Birth Cohort

Weight status transitions from pre-adolescence (ages 5–9) to early adolescence (ages 10–13) (A), from early to late adolescence (ages 14–17) (B), and from pre- to late adolescence (C). p values reflect a test for trend over birth cohorts within each baseline weight status.

Table 1
Demographic characteristics and BMI (raw, percentile and z-scores) stratified by age categories and birth cohort

	Birth cohorts	Pre-Adolescence (Ages 5 – 9)		Early Adolescence (Ages 10 – 13)		Late Adolescence (Ages 14 – 17)	
		Total	Sub group*	Total	Sub group*	Total	Sub group*
N (Male/Female)	1955–1964	959 (481/478)	517 (226/291)	1122 (527/595)	517 (226/291)	1005 (462/543)	517 (226/291)
	1965–1974	769 (384/385)	466 (224/241)	1166 (577/589)	466 (224/241)	1036 (500/536)	466 (224/241)
	1975–1984	714 (336/378)	352 (145/207)	1081 (533/548)	352 (145/207)	1051 (488/563)	352 (145/207)
	1985–1994	1142 (543/599)	402 (177/225)	1416 (660/756)	402 (177/225)	916 (401/515)	402 (177/225)
Mean Age (SD)	1955–1964	7.4 (1.4)	7.4 (1.4)	11.6 (1.0)	11.5 (0.9)	15.5 (1.0)	15.6 (1.03)
	1965–1974	7.5 (1.4)	7.5 (1.4)	11.9 (1.0)	11.8 (1.0)	15.5 (1.0)	15.4 (0.98)
	1975–1984	7.9 (1.2)	7.8 (1.2)	11.7 (1.1)	11.6 (0.9)	15.6 (1.1)	15.6 (1.02)
	1985–1994	7.3 (1.4)	7.1 (1.4)	11.6 (1.0)	11.4 (0.9)	15.3 (1.0)	15.3 (0.90)
Mean BMI, raw (SD)	1955–1964	17.8 (3.3)	17.7 (3.1)	21.8 (5.2)	21.9 (5.0)	25.8 (6.1)	25.9 (6.0)
	1965–1974	18.5 (4.3)	18.5 (4.3)	23.1 (5.9)	23.0 (5.9)	26.6 (7.1)	26.7 (6.7)
	1975–1984	19.3 (4.6)	19.1 (4.1)	24.1 (5.9)	24.3 (5.7)	28.7 (7.1)	29.5 (7.1)
	1985–1994	19.3 (4.6)	19.2 (4.2)	26.0 (6.6)	25.9 (6.6)	30.2 (7.8)	30.6 (7.9)
Mean BMI, Age-Sex Specific CDC Z-score (SD)	Pearson's $r^{\dagger}$	0.157 < 0.0001	0.164 < 0.0001	0.267 < 0.0001	0.271 < 0.0001	0.242 < 0.0001	0.277 < 0.0001
	1955–1964	0.70 (0.98)	0.68 (0.98)	0.86 (1.03)	0.89 (1.03)	1.03 (0.98)	1.04 (0.98)
	1965–1974	0.78 (1.13)	0.76 (1.16)	1.04 (1.05)	1.03 (1.07)	1.10 (1.05)	1.13 (1.03)
	1975–1984	0.94 (1.11)	0.95 (1.07)	1.26 (1.01)	1.33 (0.96)	1.41 (0.97)	1.52 (0.92)
Median BMI, Age-Sex Specific CDC %tile (IQR)	1985–1994	1.04 (1.16)	1.07 (1.18)	1.52 (0.97)	1.53 (0.98)	1.59 (0.97)	1.63 (0.98)
	Pearson's $r^{\dagger}$	0.131 < 0.0001	0.139 < 0.0001	0.246 < 0.0001	0.248 < 0.0001	0.223 < 0.0001	0.241 < 0.0001
	1955–1964	75 (53–90)	75 (51–90)	82 (54–95)	85 (57–97)	87 (66–96)	87 (66–97)
	1965–1974	79 (51–95)	78 (50–95)	88 (61–97)	87 (61–97)	88 (66–97)	89 (67–97)
Median BMI, Age-Sex Specific CDC %tile (IQR)	1975–1984	84 (54–97)	82 (55–97)	93 (73–98)	94 (77–98)	95 (79–98)	96 (84–99)
	1985–1994	86 (60–98)	87 (59–98)	96 (84–99)	97 (83–99)	97 (85–99)	97 (86–99)
	Pearson's $r^{\dagger}$	0.099 < 0.0001	0.104 < 0.0001	0.213 < 0.0001	0.206 < 0.0001	0.177 < 0.0001	0.183 < 0.0001

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Children who had research exams in each of the three age categories
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Pearson Correlation Coefficient of BMI measure with birth year, partial for sex and age