NIH-PA Author Manuscript
NIH-PA Author Manuscript

NIH Public Access
Author Manuscript
Am J Hum Biol. Author manuscript; available in PMC 2012 May 1.

Published in final edited form as:
Am J Hum Biol. 2011 May ; 23(3): 346–352. doi:10.1002/ajhb.21140.

The Co-Occurrence of Obesity, Elevated Blood Pressure and Acanthosis Nigricans among American Indian School-children: Identifying Individual Heritage and Environment-level Correlates

Mary O. Hearst, PhD, MPH[1], **Melissa Nelson Laska, PhD, RD**[1], **John H. Himes, PhD, MPH**[1], **Mark Butterbrodt, MD**[2], **Alan Sinaiko, MD**[1,3], **Richard Iron Cloud, MA**[4], **Mary Tobacco**[5], and **Mary Story, PhD**[1,3]

[1] Division of Epidemiology and Community Health, University of Minnesota, Minneapolis, MN 55454

[2] Indian Health Service, Pine Ridge Indian Hospital, South Dakota

[3] Department of Pediatrics, University of Minnesota, Minneapolis, MN 55455

[4] Oglala Sioux Tribe, South Dakota

[5] Oglala Lakota College, Kyle, South Dakota 57752

Abstract

Objective—To estimate the prevalence and explore the social and cultural etiologic roots of weight status, blood pressure and acanthosis nigricans among American Indian children on a reservation in South Dakota.

Methods—This observational study was conducted in 26 schools from 1998–2002 and included 5,422 observations representing 3,841 children, ages 3–19. Trained staff measured height, weight, blood pressure and assessed the presence of acanthosis nigricans (AN). Percent Indian heritage (PIH) was abstracted from tribal records. Sociodemographic environment (SDE) was calculated using the 2000 Census at the city/town level. Descriptive analyses were conducted using one measurement time point, including tests for trend and co-occurrence of risk factors using the kappa statistic. Hierarchical, multivariate logistic regression estimated associations with overweight/obesity status, accounting for multiple measures on individuals and SDE.

Results—The overall prevalence of overweight/obesity was 46%, of hypertension 9%, and of AN 14%. The co-occurrence of risk factors was moderate to high. PIH and AN were positively associated in unadjusted analysis. Controlling for sex, age, and SDE, higher PIH was a significant correlate of overweight/obesity, although when hypertension (OR=5.92, CI=3.27–10.72), pre-hypertension (OR=3.80, CI=1.99–7.26) and AN (OR=16.20, CI=8.08–32.48) were included in the model PIH was no longer significant. SDE was not significantly associated with overweight/obesity.

Conclusion—PIH appeared to be an important correlate of overweight and obesity, except when adjusted for the co-occurrence of high blood pressure and AN. Overall, the prevalence and co-occurrence of various risk factors in this population was high. Obesity prevention initiatives targeting families and communities are needed, as well as access to screening and treatment services.

Correspondence To: Mary O. Hearst, Ph.D., M.P.H., University of Minnesota, 1300 South Second Street, Suite 300, Minneapolis, MN 55454, Phone Number: 612-624-5723, Fax Number: 612-624-3015, hearst@umn.edu.

Keywords

American Indian; overweight; obesity; acanthosis nigricans; blood pressure; children; type 2 diabetes

INTRODUCTION

Overweight and obesity among American Indian children and adolescents are 2–3 times more prevalent than among all U.S. children (Story, Evans et al. 1999; Zephier, Himes et al. 2006). The prevalence of related risk factors also has risen dramatically in past decades among American Indian children; particularly type 2 diabetes mellitus (DM) (Dabelea, Hanson et al. 1998; Fagot-Campagna 2000; Acton, Burrows et al. 2002). Because it is difficult to conduct routine diabetes screening within many American Indian communities, early screening for acanthosis nigricans (AN), as an indicator of insulin resistance, has been recommended as an inexpensive and noninvasive tool for identifying individuals at high risk for diabetes (Stuart, Smith et al. 1994; Stoddart, Blevins et al. 2002). AN presents as darkened patches of skin usually observed on the neck and/or the axilla. The prevalence of AN in American Indian youth has been estimated to be 30–40% (Stuart, Smith et al. 1994; Stoddart, Blevins et al. 2002), although current surveillance data are not available.

Less is known about other obesity-related cardiovascular disease risk factors in American Indian youth, such as pre-hypertension (pre-HTN) and hypertension (HTN). There is a strong association of elevated blood pressure with obesity and type 2 diabetes, an increased prevalence of hypertension among adults across various tribes, (MMWR 2000; Story, Stevens et al. 2003), and a strong link between blood pressure and body mass index (BMI) among adolescents (Gilbert, Percy et al. 1996).

While previous research has indicated that some American Indian tribes are genetically susceptible to developing type 2 diabetes and related sequelae (Baier and Hanson 2004), it is likely that the increases in the prevalence of these adverse health outcomes over the last three decades are due to a complex array of genetic and environmental factors (Story, Stevens et al. 2003). Environmental attributes -- including sociodemographic, physical and cultural characteristics -- have been shown to have a strong influence on a wide range of health outcomes and behaviors (Smedley and Syme 2001). Little research to date, however, has sought to describe and understand the environments of American Indian reservations and their impacts on residents (Story, Stevens et al. 2003). Reservations currently represent some of the most economically impoverished and underserved areas in the nation, and it is important to explore the impacts of broad sociodemographic characteristics, as well as specific attributes of the physical and cultural environment, that may have an influential role in weight-related health behaviors and outcomes among American Indian communities (Story, Stevens et al. 2003).

The purpose of this study was to examine individual Indian heritage and environment-level correlates of weight status, blood pressure and type 2 diabetes risk using school screening data, collected in 1998–2002 from school children attending Head Start through Grade 12 on the Pine Ridge Reservation in South Dakota. Specifically, this study estimated the clustering of overweight, HTN, and AN in American Indian children and explored the correlates of these risk factors by comparing the associations with Indian heritage (genetic/individual traits) and the contextual characteristic of environmental sociodemographic composition.

METHODS

Study sample

The study sample comprised 5,422 school-based screenings that were conducted on American Indian children aged 3 to 19 years attending 26 schools on a reservation in South Dakota. There were 3,841 individuals in the sample and many individuals were screened multiple times. There were 2,621 participants measured at only one time point, 1,220 with measures at two time points, 332 with measures at three time points, 24 at four points, and 5 at five time points. Sample sizes are provided in the tables to account for variations in sample size due to missing data, measurement at first time point only, and inclusion of multiple measures.

Measurement

Data were collected by the Indian Health Service (IHS) from 1998–2002. Measurements were conducted in schools by trained staff. Percent Indian heritage (PIH) was collected from tribal records by a tribal staff member. Using addresses of the schools, we integrated U.S. Census data of the city/town where the school was located to characterize sociodemographic attributes of the environment and to examine the relative environmental contributions to weight-related health outcomes.

BMI—Height and weight were measured by staff trained by a public health nurse and Indian Health Service physician. Height was measured to the nearest 0.1 centimeter using a portable stadiometer and weight was measured to the nearest 0.1 pound using a balance scale. Weight in pounds was converted to kilograms. BMI was calculated as weight (kg)/height (m)^2. Height, weight and BMI were converted to age- and sex-specific z-scores as recommended by the US Centers for Disease Control and Prevention (Centers for Disease Control and Prevention 2000). Participants were categorized as overweight if age and sex specific BMI ≥ 85th and < 95th percentile and categorized as obese if BMI ≥ 95th percentile.

Data cleaning procedures—BMI z-scores were calculated for 5,307 observations with available data. A two-step data cleaning procedure was undertaken to remove outliers. First, the height-for-age z-score (HAZ) was truncated to only include values between −3.0 and 3.0. Biologically, it was deemed implausible for HAZ to be outside this range. Based on this criterion, 175 observations were excluded. The second phase of data cleaning used a nonparametric statistical approach to identify BMI outliers that were probable recording errors (Iglewica and Hoaglin 1993). Within age groups, BMI z-scores less than the 25th percentile minus 1.5 times the interquartile range, and those greater than the 75th percentile plus 1.5 times the interquartile range, were excluded. Based on this two-step process 143 observations were excluded from the analysis, yielding 5,164 observations available for analysis.

Blood Pressure—Blood pressure measurement was performed in the 26 schools using auscultation by staff trained by experts in blood pressure measurement with the children in the seated position and after five minutes of rest in a quiet room. A single measurement was used for systolic and 5th phase Korotkoff diastolic blood pressure. Blood pressure data were available for 2,501 observations. Both systolic blood pressure (SBP) and diastolic blood pressure (DBP) were considered errors and excluded if DBP was equal to or greater than the recorded SBP (n=6).

HTN and pre-HTN were defined according to the guidelines recommended by the 2004 National High Blood Pressure Education Program Working Group (2004). Participants were considered hypertensive if the average SBP and/or DBP was greater than or equal to the 95th

NIH-PA Author Manuscript
NIH-PA Author Manuscript

percentile for gender, age and height. Pre-hypertension was defined as average SBP and/or DBP level greater than or equal to the 90th percentile but less than the 95th percentile for gender, age and height.

Acanthosis nigricans (AN), a skin lesion marked by thick, darkened skin at the back of the neck (Sedano and Gorlin 1987), was assessed by health personnel trained by a local Indian Health Service physician. This location is reported to allow for more reliable detection (kappa=0.62) than other locations on the body (Burke, Hale et al. 1999). Results were categorized as present (‘1’) or absent (‘0’). AN data were available for 3,621 observations.

The tribe provided official tribal records that included Indian heritage expressed as a fraction, from which the investigators calculated the percentage of Indian heritage (PIH). The fraction of Indian heritage is generated by the tribe upon enrollment of individuals into the tribe. We have no documentation of the specific criteria used to estimate the fractions of Indian heritage by the tribe but have only assumed that it should fairly correspond to an ordinal ranking of individuals for this variable. PIH was available for 3,603 individuals.

The sociodemographic environment (SDE) was determined from a published standardized neighborhood deprivation index (Messer, Laraia et al. 2006) using the 2000 U.S. census data for each city or town in which the schools were located (n=12). Using previously described methods (Messer, Laraia et al. 2006) the census variables included: 1) % in crowded conditions (>1 person per room); 2) % living in poverty; 3) % with income <$30,000; 4)% female headed households; 5) % on public assistance; 6) % civilians unemployed; 7) % males in management; and 8) % with less than high school education. Principal component analyses were used to derive an SDE score for each city/town. For these analyses SDE scores then were standardized to mean = 0, standard deviation=1. Towns where schools were located were rank ordered from lowest score to highest score, using the index with the highest score indicating the lowest sociodemographic environment. As a check of face validity, cities/towns were ordered according to the SDE score and staff familiar with the reservation confirmed that those cities/towns with low scores had more resources than those cities/towns with high scores. The SDE was subsequently assigned to each respective city/town and consequently to each child affiliated with that school, creating a hierarchical data set.

ANALYSIS PLAN

Given the missing data for each measure, we tested whether or not “missingness” of our primary analytic variables differed by characteristics of the study sample and the study implementation. In general, the absence of blood pressure, height and weight were significantly associated with date of the screening, school, and age at screening. As time progressed, blood pressure, height, and weight were less likely to be collected. The youngest age group (3–5 years) was also the least likely to have blood pressure, height, and weight measured. Missing data on PIH was also significantly associated with age, with younger participants less likely to have tribal enrollment information (including PIH) available. There were only 26 observations without AN documented. No difference in missingness was noted by sex. Given this pattern of missing data, we cautiously interpret findings, in particular among the youngest in the sample.

Baseline sample frequencies were calculated for categorical variables by age groupings, sex, PIH and SDE. A Wald chi-square test was used to assess the linear trend by age grouping and PIH. Kappa statistics were calculated to examine chance-adjusted concordance of more than one risk factor. Finally, multilevel models, accounting for clustering at the school and multiple measures within individuals were constructed, beginning with demographic factors and area-level SDE and subsequently adding PIH, blood pressure categories, and presence of AN. A Wald test was used for assess the linear trend of PIH for each model. All data management and analyses were conducting using Stata v. 10 [Stata Statistical Software: Release 10.0, College Station, TX]. Collection and analyses of these data were approved by the tribe, as well as the University of Minnesota Institutional Review Board.

RESULTS

Table 1 presents the prevalence of overweight, obesity, pre-HTN, HTN, and AN by participant age category, sex, PIH and SDE for all participants with complete data at their first measurement time point. Overall, 45.9% of the sample was overweight or obese; however, the prevalence of overweight/obesity did not significantly increase by age. Overall, 24.3% of the sample was obese, with the proportion ranging from 21.3% among 3–5 year olds to 27.1% among those over 12 years old, and then dropping to 22.7% for those over 15 years, although the trend was statistically significant (p=0.003). Males had higher a prevalence of obesity (26.2%) compared to females (22.3%). For both overweight/obese and obese participants, there was a significant trend for PIH. Of note, the prevalence of obesity among those participants with less than 25 PIH was 15.0% compared to 28.5% for participants with over 75 PIH. The mean level of SDE differed for the overweight/obese and obese children compared with healthy weight participants. In both cases, the mean SDE was higher for overweight or obese children, suggesting relatively more challenged areas, compared to residential areas for healthy weight participants.

The prevalence of pre-HTN was 6.7% overall, and 9.0% for HTN overall. The prevalence of pre-HTN did not increase significantly as age increased; however, the prevalence of HTN did significantly increase with increasing age category. Among those with HTN, the prevalence increased from 5.6% in 3–5 year olds to 14.0% in those 12 years and older, decreasing somewhat (10.9%) for those older than 15 years.

Using the first measurement point, the overall prevalence of AN was 13.5%. There was a significant linear trend by age category, with a prevalence of 2.0% in 3–5 year olds to 20.6% in participants 12 –15 year olds, and 12.3% among 15 years of age or older. Females had a higher prevalence of AN (15.1%) compared to males (12.1%) (p=0.01). There was a significant trend in the prevalence of AN by PIH, from 7% in those with less than 25 PIH to 18.6% in those with greater than 75 PIH.

In examining the co-occurrence of risk factors using the sample of participants with valid data at their first examination, we found statistically significant kappa statistics for all combinations of risk factors. The kappa's ranged from a low of 0.12, with 58% exact agreement between overweight or obese and HTN, to a high of kappa=0.41 with an 80% exact agreement between obesity and presence of AN (Table 2).

The relationship between SDE and PIH was evaluated using Pearson's correlation coefficient, and the linear trend between categories of PIH and SDE was assessed. The correlation between the continuous measures of PIH and SDE was positive and statistically significant (r=0.08, p<0.001) (data not shown). The test for linear trend using a 1-degree-of-freedom Wald chi-square test showed a positive and significant linear trend between categories of PIH and SDE (overall p<0.001). The children in the study with higher categories of PIH were also living in the more challenged communities.

Table 3 presents the results from the staged hierarchical models for all observations with valid data, including multiple measures for individuals. Model 1 included age, sex and SDE associations with overweight and obesity. None of the adjusted odds ratios (OR) were statistically significant. Model 2 added PIH to the previous model and resulted in a stepped increase in the odds of overweight or obesity as PIH categories increased. The test for linear trend across these quartiles was statistically significant (chisq=6.15, p=0.01). Compared to those with the lowest category of PIH, those with 25–50 PIH had 1.93 (CI=1.02–3.65) times the risk of overweight/obesity; those with 50–75 PIH had 2.02 (CI=1.08–3.75) times the risk of overweight/obesity and those with the highest PIH (≥ 75%) had 2.47 (1.34–4.55) times the risk of overweight/obesity. No other demographic or SDE characteristics were statistically significant.

Adding HTN to the hierarchal model (Model 3) attenuated the association between PIH and overweight/obesity, leaving only those with the highest PIH compared to lowest PIH with greater odds of overweight/obesity (OR=2.47, CI=1.34–4.55). Nevertheless the trend across categories of PIH remained significant (chisq = 4.99, p=0.03). In Model 3 the adjusted odds of overweight/obesity were 6.49 (CI=3.91–10.78) times as high for those with HTN compared to those without HTN. Note that the available sample for Model 3 is reduced to 1,629 due to the fewer numbers of observations including blood pressure.

The final model (Model 4), which includes AN, attenuates the association between PIH and overweight/obesity and diminishes the effect of HTN on overweight/obesity, although still substantial (OR=4.98, CI=2.86–8.64). AN was highly significant (OR=8.50, CI=8.50–29.87), yet a wide CI likely reflects lack of precision. Demographic and SDE characteristics remain non-significant. Models 1–3 were re-estimated using the 1,615 participants available for Model 4 (data not shown) and yielded similar conclusions, suggesting that the differences in associations among the four models in Table 3 did not result from using different sample sizes or the patterns of missing data.

The same procedures were used in a staged approach to modeling pre-HTN and HTN as outcome variables and overweight/obesity as an independent variable in the model (data not shown). There were no significant associations detected when pre-HTN was examined as the outcome, although the number of cases of pre-HTN was quite low. The adjusted OR of HTN was 11.74 among overweight/obese compared to healthy weight, although the confidence limits were quite large (95% C.I. = 2.28–60.38). When adding AN to the final model, OR of HTN was attenuated for overweight/obese compared to healthy weight participants (adjusted OR=10.07, 95% C.I. = 1.83–55.35), but with wide confidence limits. Presence of AN was not significantly associated with HTN (adjusted OR=1.93, 95% C.I. = 0.41–9.04).

DISCUSSION

Limited information is available about obesity and cardiovascular disease risk factors and their interrelationships with individual heritage and community-level sociodemographic environmental factors in American Indian children and adolescents. While studies have explored some of the psychosocial and behavioral factors related to higher risk (Neumark-Sztainer, Story et al. 1997; Story, Evans et al. 1999; Story, Snyder et al. 2003), there are few studies examining individual heritage or contextual factors, particularly among children as young as 3 years old. This study offers one of the first investigations of the relative importance of SDE, individual Indian heritage and the co-occurrence of multiple risk factors with overweight and obesity. This secondary data analysis was not designed for causal analysis, but emphasized the complex interrelationships among a traditionally underserved population.

We found that the overweight prevalence (44–49%) was not significantly different by age, but obesity prevalence (21–27%) showed an increasing trend by age. Age-adjusted prevalence estimates from a similar American Indian childhood population estimated overweight at 39.1% for males and 38% for females, obesity at 22.0% for males and 18.0%

for females (Zephier, Himes et al. 1999). These estimates are lower but more comparable with our results than are national estimates not including American Indian children. Comparatively, the National Health and Nutrition Examination Survey (NHANES) from 1999–2004 reported an increase over time of overweight children aged 2–19 from all racial/ethnic groups of 28.2 to 33.6% and an obesity prevalence increase from 13.9–17.1% (Ogden, Carroll et al. 2006). While non-Hispanic black and Hispanic children had higher levels of overweight and obesity than non-Hispanic white children in the NHANES data, the prevalence of overweight and obesity in the American Indian children in our study were substantially higher than both the non-Hispanic black and Hispanic children in the national data from NHANES.

Using simple bivariate comparisons, we found that mean SDE was higher among overweight and obese children and adolescents compared to healthy weight youth, suggesting that overweight and obese children live in an area with more challenging social and environmental factors, such as more area-level poverty, more unemployment and lower educational attainment, which have been shown to be related to lack of access to health supporting resources (Oliver and Hayes 2005; Fleischer, Diez Roux et al. 2008). However, in using multilevel modeling to examine different levels of influence on weight status and account for correlations between multiple observations of the same individual, we found that there was no association between weight status and area SDE when modeled with age and sex, or other modeled variables.

It is possible that SDE does not play a role in American Indian children's weight status. Although there was variability in SDE for this area, this is an economically challenged area overall; thus heterogeneity in this sample-standardized SDE is limited. In fact, the county that covers most of the reservation was the 4^{th} poorest county in the U.S in 2007, with a poverty rate of 47.4% (STATS Indiana 2007). Three of the four poorest counties in the US are located in south and central South Dakota (STATS Indiana 2007). It is possible that the majority of the participants attend schools in relatively impoverished areas, so no independent effect of poverty could be observed. In addition, the use of area-level measures does not take into account heterogeneity among the people that reside within a given area (Ionnides 2004); as such it may not accurately reflect an individual's economic or educational status. This is referred to as the 'ecologic fallacy,' and has been discussed in depth in previous literature (Rothman and Greenland 1998).

The prevalence of overweight/obesity and risk factors in American Indian children and adolescents is alarming, and is perhaps reflective of a genetic/individual heritage component given the prevalence of obesity and obesity-related risk factors in this population was so much higher than in other racial/ethnic groups. We found that as PIH increased, the odds of overweight/obesity increased in the hierarchical models. Because overweight and obesity are polygenic phenotypes, one would expect that categories of increasing PIH would be associated with approximately monotonically changing risks, if there is differential risk between Indian heritage and other heritage relative to overweight and obesity. In models 2 and 3 (Table 3) the post-hoc testing for trend was statistically significant for PIH categories. Although the steps in point estimates of OR with increasing quartile of PIH are not exactly equal, the 95% C.I. around the point estimates were sufficiently wide that the OR estimates satisfy such a theoretical model. For model 4, it may be that including AN in predicting overweight and obesity over-controls for PIH associations because of considerable covariance with PIH (see Table 1).

Often, researchers use referent groups to compare outcomes of one population group to a different population group. The unique feature of this study was that all participants were of

NIH-PA Author Manuscript

NIH-PA Author Manuscript

American Indian heritage with the referent group reflecting the lowest PIH (<25%). This offers a comparison within the American Indian culture itself.

The finding that as PIH categories increase, overweight and obesity status increases may reflect other unmeasured factors related to environmental or social exposures, such as discrimination. We found a positive significant correlation and test for trend between PIH and SDE, suggesting that those children with the higher percentage of Indian heritage live in more socially and economically challenged communities, which subsequently may have less health promoting resources, such as easy access to healthy food choices and opportunities for safe physical activity. In addition, other social factors, such as discrimination and/or segregation, may also play a role in these relationships. There is considerable documentation in the literature on patterns of discrimination and segregation based on race/ethnicity and skin tones for African Americans (Massey 1990; Turner 1995) and toward American Indians in general (Berger 2009). The patterning of discrimination and segregation is correlated and may be causally related to worse health outcomes due to limited positive life opportunities and resources (Story, Stevens et al. 2003; Acevedo-Garcia, Osypuk et al. 2008; Hearst, Oakes et al. 2008). Together, these factors suggest the need for a socioecologic approach to obesity prevention.

There is a complex synergy of health consequences associated with overweight and obesity. DM is strongly associated with obesity and the prevalence of DM is increasing among American Indians (Lee, Welty et al. 2002). In the present study we used AN as a marker for risk of DM. The prevalence of AN in our sample was 13.5%, with a low of 2% among pre-school aged children and a high of 20.6% among those 12 years and older. Similarly, prevalence estimates from a diverse sample of students from the southwest found AN present in 18.9% of students screened (Mukhtar, Cleverley et al. 2001), although only a small proportion (19%) of that sample were American Indian. Other studies found prevalence estimates ranging from 19–38% depending on tribal affiliation, with the lowest estimates among the youngest children (Stuart, Smith et al. 1994; Stoddart, Blevins et al. 2002). We found that AN was highly concordant with overweight, obesity and hypertension. A previous study has shown that American Indian children from multiple Indian Health Service Clinics with severe AN had higher BMI compared to those with milder forms of AN (Copeland, Pankratz et al. 2006).

Consistent with our findings, pre-HTN and HTN have also been shown to be associated with overweight and obesity, particularly among American Indian populations (Story, Snyder et al. 2003). Our findings indicate that 6.7% of the sample had pre-HTN, although the prevalence estimate among 3–5 year olds (9.6%) may be partially reflective of sampling error. Nine percent of our sample had HTN, increasing to 14% among those 12 years and older. Age and sex adjusted pre-HTN and HTN were highly concordant with overweight and AN. Hypertension if left unchecked can lead to cardiovascular disease and a higher risk of stroke (Hayes, Denny et al. 2006), increasing the chance of early death or disability.

Regardless of individual and community level demographics, and family heritage (PIH), AN had the strongest association with overweight/obesity followed by HTN, although AN, HTN and overweight/obesity are strongly clustered. Particularly given the prevalence of risk factors at early ages, this finding speaks to the need to begin preventive services early in life. By the time the children are school-aged and progressing toward middle and high school, there is extensive clustering of risk factors, and the intervention needs likely revolve around secondary prevention or screening/treatment services.

This study has some important limitations. First, the data were analyzed cross-sectionally, so causality cannot be ascertained. Although longitudinal data were present, traditional longitudinal analysis was not possible because of the lack of patterned data collection and lack of consistent intervals by age. As the repeated visits increase, the number of participants drops substantially as described in the methods section. We observed lower prevalence of overweight, obesity, HTN and AN in those over 15 years compared to younger age categories. Given the nature of cross-sectional studies, that pattern may be related to sampling, secular change or biology. Furthermore, blood pressure measurements were based on single determinations on each measurement occasion so we did not have the advantages of replicate measurements to overcome the known variability within each child (Rosner, Cook et al. 1987). Nevertheless, including the multiple measures over time for those participants who had them did help improve the within–person precision. Finally, environmental attributes were based on school-level location and not the home address of the child. In addition, the U.S. Census data may not accurately represent the population characteristics of American Indians living on the reservations.

These are historical data; hence it is certainly possible that the prevalence estimates will have changed, most likely increased, in the time that has passed since the data were collected. However, we have little reason to believe that the observed associations among the variables would have changed during that time. In addition, not all measures were collected on all individuals and the number of data collection points varied by participant. For example, timing of enrollment in the tribe is a family decision and varies considerably, so there were incomplete data on PIH. It is possible that those who were already enrolled in the tribe were somehow different from those that were not enrolled. Finally, the Pine Ridge Indian Reservation has many individuals with the same name. While we verified the name, birth date and tribal enrollment certificate number, if available, it is possible that we did not accurately identify all multiple observations.

Despite these limitations, this study is an important contribution to the sparse literature available about obesity among American Indian children and the co-occurrence of related risk factors. To our knowledge, this study is the first to examine multi-level factors associated with weight status, including physiological, genetic/individual heritage and environmental factors, among American Indian children and adolescents. By approaching the analysis as we did, we were able to maximize the use of a large sample to improve our understanding of these complex relationships. By including and accounting for clustering of individuals with repeated measures, it was possible to take advantage of all the data available and to maximize within-person precision. Given the observed heterogeneity between American Indian tribes and limited obesity prevention interventions, it is possible that subsequent interventions need to be tailored to unique tribes or perhaps by regional location. What is apparent is that prevention interventions need to begin at very young ages, beginning at least in preschool. Already by ages 3–5, the prevalence of overweight/obesity and co-occurring risk factors leads us to look at secondary prevention and treatment needs in addition to primary prevention.

Further investigation is needed to understand the sociodemographic environments of American Indian reservations and their impact on residents. This may include alternative measurement approaches such as qualitative assessment or household-level factors to better capture the range of experiences and exposures facing American Indian children and families in largely economically challenged areas. Additionally, further exploration is needed to explore the role of PIH and its possible role in health exposures and outcomes. American Indian children, particularly those in underserved areas, need to have regular screening of weight status and other risk factors, such as the non-invasive use of assessing AN. However, more than just screening, children need to have access to healthy nutrition and physical activity environments, to health services as part of wellness promotion, and healthcare access to address treatment of HTN, obesity, and the potentially advancing Type 2 DM.

Acknowledgments

Funding for analysis of this study was provided by pilot studies from the Minnesota Obesity Center and the University of Minnesota Center for Transdisciplinary Research on Energetics and Cancer (NCI Grant 1 U54 CA116849-01). Additional support for the analysis of these data was provided by Award Number K07CA126837 from the National Cancer Institute.

The authors would like to acknowledge all staff who participated in data collection and data entry, the schools, the children and adolescents, the Indian Health Service, Oglala Sioux Tribe (Oglala Lakota), the Office of Minority Health who funded the original data collection and University of Minnesota students who assisted with data entry.

References

Acevedo-Garcia D, Osypuk TL, et al. Toward a policy-relevant analysis of geographic and racial/ ethnic disparities in child health. Health Aff (Millwood). 2008; 27(2):321–33. [PubMed: 18332486]

Acton KJ, Burrows NR, et al. Trends in diabetes prevalence among American Indian and Alaska native children, adolescents, and young adults. Am J Public Health. 2002; 92(9):1485–90. [PubMed: 12197981]

Baier L, Hanson R. Genetic studies of the etiology of type 2 diabetes in Pima Indians: hunting for pieces to a complicated puzzle. Diabetes. 2004; 53:1181–1186. [PubMed: 15111484]

Berger, B. UCLA Law Review 56 UCLA L Rev. 2009. Red: Racism and the American Indian; p. 591

Burke JP, Hale DE, et al. A quantitative scale of acanthosis nigricans. Diabetes Care. 1999; 22(10): 1655–9. [PubMed: 10526730]

Centers for Disease Control and Prevention. Growth Charts 2000: United States. 2000. Retrieved January 28, 2008, from http://www.cdc.gov/growthcharts

Copeland K, Pankratz K, et al. Acanthosis Nigricans, insulin resistance (HOMA) and dyslipidemia among Native American children. J Okla State Med Assoc. 2006; 99(1):19–24. [PubMed: 16499154]

Dabelea D, Hanson RL, et al. Increasing prevalence of Type II diabetes in American Indian children. Diabetologia. 1998; 41(8):904–10. [PubMed: 9726592]

Fagot-Campagna A. Emergence of type 2 diabetes mellitus in children: epidemiological evidence. J Pediatr Endocrinol Metab. 2000; 13(Suppl 6):1395–402. [PubMed: 11202215]

Fleischer NL, Diez Roux AV, et al. Social patterning of chronic disease risk factors in a Latin American city. J Urban Health. 2008; 85(6):923–37. [PubMed: 18830819]

Gilbert TJ, Percy CA, et al. Blood pressure and body measurements among Navajo adolescents. Public Health Rep. 1996; 111(Suppl 2):44–6. [PubMed: 8898772]

Hayes D, Denny C, et al. Racial/Ethnic and socioeconomic differences in multiple risk factors for heart disease and stroke in women: behavioral risk factor surveillance system, 2003. J Womens Health (Larchmt). 2006; 15(9):1000–8. [PubMed: 17125418]

Hearst MO, Oakes JM, et al. The Effect of Racial Residential Segregation on Black Infant Mortality. Am J Epidemiol. 2008

Iglewica, B.; Hoaglin, D. How to detect and handle outliers. In: Mykytka, E., editor. The ASQ Basic References in Quality Control: Statistical Techniques. Milwaukee, WI: American Society for Auality, Statistics Division; 1993. p. 16

Ionnides Y. Neighborhood Income Distributions. Journal of Urban Economics. 2004; 56:435–457.

Lee ET, Welty TK, et al. Incidence of diabetes in American Indians of three geographic areas: the Strong Heart Study. Diabetes Care. 2002; 25(1):49–54. [PubMed: 11772900]

Massey DS. American Apartheid: Segregation and the Making of the Underclass. American Journal of Sociology. 1990; 96(2):329–357.

Messer LC, Laraia BA, et al. The development of a standardized neighborhood deprivation index. J Urban Health. 2006; 83(6):1041–62. [PubMed: 17031568]

MMWR. Prevalence of selected cardiovascular disease risk factors among American Indians and Alaska Natives--Unites States, 1997. MMWR. 2000; 49:461–465. [PubMed: 10882292]

Mukhtar Q, Cleverley G, et al. Prevalence of acanthosis nigricans and its association with hyperinsulinemia in New Mexico adolescents. J Adolesc Health. 2001; 28(5):372–6. [PubMed: 11336866]

National High Blood Pressure Education Program Working Group. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics. 2004; 114(2 Suppl):555–76. 4th Report. [PubMed: 15286277]

Neumark-Sztainer D, Story M, et al. Psychosocial concerns and weight control behaviors among overweight and nonoverweight Native American adolescents. J Am Diet Assoc. 1997; 97(6):598–604. [PubMed: 9183319]

Ogden CL, Carroll MD, et al. Prevalence of Overweight and Obesity in the United States, 1999–2004. JAMA. 2006; 295(13):1549–1555. [PubMed: 16595758]

Oliver LN, Hayes MV. Neighbourhood socio-economic status and the prevalence of overweight Canadian children and youth. Can J Public Health. 2005; 96(6):415–20. [PubMed: 16350864]

Rosner B, Cook NR, et al. Reproducibility and predictive values of routine blood pressure measurements in children. Comparison with adult values and implications for screening children for elevated blood pressure. Am J Epidemiol. 1987; 126(6):1115–25. [PubMed: 3687921]

Rothman, KJ.; Greenland, S. Modern Epidemiology. 2. Philadelphia, PA: Lippincott-Raven Publishers; 1998.

Sedano HO, Gorlin RJ. Acanthosis nigricans. Oral Surg Oral Med Oral Pathol. 1987; 63(4):462–7. [PubMed: 3554094]

Smedley B, Syme S. Promoting health: intervention strategies from social and behavioral research. Am J Health Promot. 2001; 15:149–166. [PubMed: 11265579]

STATS Indiana. USA Counties in Profile. 2007.

Stoddart ML, Blevins KS, et al. Association of acanthosis nigricans with hyperinsulinemia compared with other selected risk factors for type 2 diabetes in Cherokee Indians: the Cherokee Diabetes Study. Diabetes Care. 2002; 25(6):1009–14. [PubMed: 12032107]

Story M, Evans M, et al. The epidemic of obesity in American Indian communities and the need for childhood obesity-prevention programs. Am J Clin Nutr. 1999; 69(4 Suppl):747S–754S. [PubMed: 10195597]

Story M, Snyder MP, et al. Changes in the nutrient content of school lunches: results from the Pathways study. Prev Med. 2003; 37(6 Pt 2):S35–45. [PubMed: 14636807]

Story M, Stevens J, et al. Obesity in American-Indian children: prevalence, consequences, and prevention. Prev Med. 2003; 37(6 Pt 2):S3–12. [PubMed: 14636804]

Stuart CA, Smith MM, et al. Acanthosis Nigricans among Native Americans: an indicator of high diabetes risk. Am J Public Health. 1994; 84(11):1839–42. [PubMed: 7977931]

Turner R. The color complex: Intraracial discrimination in the workplace. Labor Law Journal. 1995; 46(11):678–684.

Zephier E, Himes JH, et al. Prevalence of overweight and obesity in American Indian School children and adolescents in the Aberdeen area: a population study. Int J Obes Relat Metab Disord. 1999; 23(Suppl 2):S28–30. [PubMed: 10340802]

Zephier E, Himes JH, et al. Increasing prevalences of overweight and obesity in Northern Plains American Indian children. Arch Pediatr Adolesc Med. 2006; 160(1):34–9. [PubMed: 16389208]

Table 1

Descriptive characteristics of risk factors in the study sample at first measurement point by age, gender, percent Indian heritage and sociodemographic environment.

% (n)	Overweight/obese ≥85%ile BMI-for-age and gender n=3,640	Obese ≥95%ile BMI-for- age and gender n=3,640	Pre- Hypertension n=2,010	Hypertension n=2,010	Acanthosis Nigricans n=3,621
Overall	45.9 (1,669)	24.3 (884)	6.7 (135)	9.0 (180)	13.5 (488)
Age groups					
3–5 years	48.9 (251)	21.3 (109)	9.6 (34)	5.6 (21)	2.0 (10)
6–8 years	44.3 (439)	22.5 (223)	3.9 (16)	4.9 (21)	7.7 (76)
9–11 years	45.8 (458)	24.5 (245)	6.6 (39)	9.2 (60)	17.0 (169)
12–14 years	48.4 (387)	28.9 (231)	8.9 (39)	14.3 (73)	24.2 (192)
15–19 years	40.0 (134)	22.7 (76)	17.1 (7)	10.9 (5)	12.3 (41)
Overall p-value	0.34	0.01	0.28	<0.001	<0.001
Gender					
Male	45.9 (855)	26.2 (487)	7.2 (67)	5.6 (87)	12.1 (223)
Female	45.8 (814)	22.3 (397)	7.6 (68)	9.4 (93)	15.0 (265)
Overall p-value	0.91	0.01	0.76	0.52	0.01
% Indian Heritage					
<25%	40.6 (65)	15.0 (24)	5.8 (5)	4.4 (4)	7.0 (11)
25–50%	45.3 (253)	23.1 (129)	5.8 (15)	11.4 (33)	9.5 (53)
50–75%	46.3 (329)	27.3 (194)	8.1 (29)	9.4 (37)	15.9 (112)
>75%	49.7(459)	28.5 (263)	8.1 (41)	9.2 (51)	18.6 (170)
Overall p-value	0.02	0.0002	0.22	0.83	<0.001
Sociodemographic environment (mean(SD))[1]	0.16 (0.12)*	0.18 (0.12)**	0.14 (0.05)	0.14 (0.03)	0.18 (0.03)

[1] Standardized with mean of zero and sd = 1.

* Significantly different from healthy weight youth.

** Significantly different from non-obese youth.

Note: Overall p-value assessed using chi-square statistic

Table 2

Percentage agreement, kappa statistics and p-values for the concordance of risk factors using first measurement point data on those with complete data.

% agreement Kappa P-value	Pre-HTN	HTN	AN
≥85%ile BMI-for-age	58.6% 0.077 <0.001	57.8% 0.121 <0.001	64.1% 0.251 <0.001
≥95%ile BMI-for-age	78.0% 0.148 0.02	76.4% 0.195 <0.001	81.9% 0.411 <0.001
Pre-HTN			81.9% 0.061 0.003
HTN			82.5% 0.180 <0.001

Table 3

Examining associations with overweight/obesity using mixed models accounting for intra-individual and city/town level clustering.

	Model 1 N=3,487		Model 2 N=3,487		Model 3 N=1,629		Model 4 N=1,615	
	OR	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Age	0.99	0.96–1.51	0.99	0.91–1.00	0.96	0.91–1.00	0.92	0.86–0.96
Sex	1.17	0.89–1.54	1.16	0.78–1.51	1.11	0.84–1.47	1.02	0.76–1.40
Area-level deprivation	1.20	0.96–1.51	1.15	0.91–1.45	1.11	0.88–1.40	1.17	0.90–1.51
% Indian Heritage								
<25%			Ref		Ref		Ref	
25–50%			1.93	1.02–3.65	1.50	0.80–2.81	1.58	0.80–3.12
50–75%			2.02	1.08–3.75	1.63	0.88–3.00	1.31	0.68–2.54
>75%			2.47	1.34–4.55	1.94	1.06–3.55	1.62	0.85–3.11
Test for trend			X^2= 6.15, p=0.01		X^2= 4.99, p= 0.03		X^2= 1.06, p= 0.30	
Blood pressure								
Normal					Ref		Ref	
Pre-HTN					4.21	2.39–7.43	3.80	1.99–7.26
HTN					7.45	4.42–12.55	5.92	3.27–10.72
AN							16.20	8.08–32.48

Note: Staged multilevel mixed models were constructed, beginning with demographic factors and area-level SDE and subsequently adding PIH, hypertension and presence of AN. A Wald test was used for assess the linear trend of PIH for each model.