Early risk factors for increased adiposity: a cohort study of African American subjects followed from birth to young adulthood[1–4]

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ABSTRACT

Background: Obesity is an increasing concern in the United States. Effective prevention of obesity requires the risk factors to be well defined. African Americans have a high risk of obesity.

Objective: The objective of this study was to identify risk factors, present at birth, for increased adiposity in adulthood in an African American population.

Design: In this retrospective analysis of a prospective cohort study, anthropometric and socioeconomic variables were collected at birth. A representative sample of 447 African American subjects was followed up until young adulthood, when skinfold thickness was measured. Associations between the independent variables and increased adiposity (skinfold thickness above the 85th percentile) were explored by using unadjusted and adjusted analyses.

Results: Three variables measured at birth were independently associated with adiposity in young adulthood, explaining 12% of the variance. The odds ratios (with 95% CIs) of these variables for increased adiposity were 2.7 (1.2, 6.2) for female sex, 4.0 (1.4, 11.2) for first-born status, and 1.15 (1.06, 1.25) for each unit increment in maternal prepregnancy body mass index (BMI; in kg/m^2). After adjustment for these variables, birth weight for gestational age and socioeconomic variables were not associated with adiposity.

Conclusions: This cohort study of African American subjects was the first to identify first-born status as an independent risk factor for increased adiposity in adulthood in a US population. The results of the study strengthen previous reports of the effect of female sex and maternal BMI on adulthood obesity. Identification of risk factors early in life may help target prevention toward high-risk children and allow healthy lifestyles to be established before the onset of obesity. *Am J Clin Nutr* 2000;72:378–83.

KEY WORDS Adipose tissue, African Americans, birth order, birth weight, body mass index, cohort studies, obesity, placenta, risk factors, skinfold thickness, socioeconomic factors

INTRODUCTION

Obesity is an increasingly important public health concern in the United States. One-third of adults and >10% of children are considered to be obese (1, 2). Several of the leading causes of morbidity and mortality are associated with obesity and produce a substantial financial burden for the health system (3–5). It is estimated that each year >300 000 Americans die from complications of obesity (6).

The treatment of obesity is often unsuccessful (7); for preventive strategies to be effective, the risk factors for obesity must be determined. Identification of modifiable risk factors can provide the rationale for public health and individual interventions, and identification of nonmodifiable risk factors can be used to target prevention toward individuals at high risk. Detection of individuals at high risk during childhood may help to establish healthy lifestyles and prevent the development of obesity before 2 critical periods for its onset: the adiposity rebound, which takes place between the ages of 4 and 6 y, and adolescence (8).

Several risk factors for overweight have been identified, including genetic and familial predisposition (9–15), birth weight (9, 13, 16–20), maternal diabetes (20, 21), and behavioral and socioeconomic factors (1, 2, 9, 12, 15, 22–25). However, most studies were limited by the use of self-reported or non–research-based anthropometric data to define obesity or by the use of body mass index (BMI; in kg/m^2) as an indirect measure of adiposity. Furthermore, many studies did not adjust for important potential confounders.

The prevalence of obesity is higher among Americans of African heritage than among Americans of European heritage (1, 2). However, the risk factors for obesity remain understudied in African Americans and might be different from those in other segments of the US population (24, 26, 27). The aim of this study was to identify independent risk factors, measurable at birth, for the eventual development of increased adiposity, as assessed by

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Received November 16, 1999.

Accepted for publication February 24, 2000.

skinfold thickness measurements in young African American adults. This prospective cohort study, initially designed to investigate risk factors for other diseases, offered a unique opportunity to assess simultaneously many important potential early risk factors for obesity.

SUBJECTS AND METHODS

Subjects

The National Collaborative Perinatal Project (CPP) was initiated to investigate the risk factors for cerebral palsy at 12 US sites (28). At the Philadelphia site, women who received prenatal care between 1959 and 1965 were registered in the study after informed consent was obtained. The study was restricted to African American subjects (87% of the original group in Philadelphia) and to women with a singleton gestation. Among the 9020 African American pregnant women who were registered, 872 were lost to follow-up or experienced fetal death, leaving 8148 live births in the study. These infants were followed up for 7 y and 6657 completed the CPP study, corresponding to 82% of the live births initially included in the study.

In 1977, a secondary project, the Philadelphia Blood Pressure Project (PBPP), a representative sample of 688 African American children born between 1961 and 1965 and who completed the CPP study was recruited by random selection (29). The 447 subjects successfully followed up to young adulthood (65% of the recruited sample) constituted the sample reported in this analysis. This study was approved by the Institutional Review Board of The Children's Hospital of Philadelphia.

Measurements

The following variables were collected by interview at enrollment of the pregnant women into the CPP: maternal prepregnancy weight, maternal education, presence of the father in the household, number of adults and siblings living in the household, and number of previous pregnancies. Because the questionnaire was slightly changed in 1962, not all variables were available for every subject. The offspring was considered to be first-born if the mother reported no previous delivery or no other children living in the household at the time of delivery, depending on which question was asked at that time. Maternal weight and height were measured at admission for delivery, and maternal prepregnancy BMI was calculated. Maternal obesity was defined as a prepregnancy BMI ≥ 30 (30). At birth, the sex, birth weight, and length of the child and the placental weight were recorded. Gestational age was determined by the senior physician who assumed responsibility for the delivery using all clinical and historical information.

Anthropometric measurements were made at the end of the follow-up period, when the offspring were aged between 18.0 and 22.9 y. The measurement closest to the subject's 20th birthday was used for the analysis when more than one assessment was available. Each subject's weight was measured by a research-trained anthropometrist using a beam balance scale, and height was measured with a stadiometer (Holtain Ltd, Crymych, United Kingdom). Triceps and subscapular skinfold thickness was measured in duplicate by using a caliper (Holtain Ltd), and the mean was recorded (31). The correlation coefficients for inter- and intraobserver reliabilities for all anthropometric measurements were >0.94.

Analysis

The baseline differences between the subjects who were not followed up and the subjects with complete data were assessed by using a *t* test, a Wilcoxon rank-sum test, and a chi-square test, as appropriate. The primary continuous outcome variable was a summary measurement of truncal and peripheral adiposity in young adulthood. This sum of triceps and subscapular skinfold thicknesses between ages 18.0 and 22.9 y was expressed as age- and sex-specific SDs (SF *z* score) by using a nationally representative sample (32, 33). This procedure allows a sex comparison of the prevalence of increased adiposity, accounting for physiologic differences between the sexes. Additional analyses were performed by using the absolute sum of 2 skinfold-thickness measurements (in mm) as a more direct way of expressing adiposity. The main dichotomous outcome was increased adiposity in young adulthood, defined as a sum of triceps and subscapular skinfold thicknesses above the 85th percentile for age and sex (33).

Among all potential independent variables assessed at birth, 9 were selected for this analysis on the basis of their association with adult overweight in previous studies (1, 2, 9, 11–20, 22, 24, 27, 34–37): sex, birth weight for gestational age, placental weight, first-born status, maternal prepregnancy BMI, maternal pregnancy weight gain, maternal education, presence of the father in the household, and the number of adults living in the household at enrollment. Birth weight for gestational age was expressed as a *z* score based on a sex- and race-specific reference population (32, 38).

Univariate analyses were performed between each independent variable and the primary continuous outcome variable (SF *z* score) by using simple linear regression, a *t* test, or the Wilcoxon rank-sum test, as appropriate. Continuous independent variables were also divided into quintiles and tested for trend by using a nonparametric test across ordered groups (39). The unadjusted relative risk (RR) and 95% CIs for increased adiposity were calculated for each dichotomous independent variable, and its statistical significance was assessed with a chi-square test.

Stratified analyses were performed to test possible interactions between independent variables. There were 9 possible predictive variables. Therefore, in the initial model, a stepwise linear regression was conducted for the subjects with complete data (initial model $n = 163$) with a P value of 0.05 for inclusion and 0.20 for exclusion (40), including all 9 independent variables and the primary continuous outcome variable, SF *z* score. An additional analysis using a robust variance estimate was performed to account for the skewness of the SF *z* score. Significant contributing variables were then introduced in a logistic regression model with the main dichotomous outcome (adulthood increased adiposity defined by skinfold thickness above the 85th percentile) to assess odds ratio (ORs) and 95% CIs. To evaluate the effect of missing data, an additional model was built that used an imputation of missing data by best-subset regression (41, 42). STATA 5.0 software was used for the statistical analysis (42).

RESULTS

To assess the accuracy of self-reported maternal prepregnancy weight, the agreement with the weight measured at delivery, adjusted for gestational age, was assessed by linear regression ($R^2 = 0.87$, $P < 0.0001$). Potential differences in baseline variables were evaluated between the subjects recruited at birth but

TABLE 1
General characteristics of the subjects, number of observations available, and median or proportion (10th and 90th percentiles)[1]

Characteristic	Observations available	Median or proportion (10th and 90th percentiles)
Subject at birth		
Sex (% male)	447	50.6 (NA)
Birth weight (g)	447	3119 (2495, 3657)
Gestational age (wk)	354	40 (37, 40)
Birth weight for gestational age (*z* score)	354	−0.63 (−1.64, 0.60)
Placental weight (g)	431	430 (330, 570)
Family		
First-born status (%)	365	16.4 (NA)
Maternal prepregnancy BMI (kg/m^2)	332	22.5 (19.1, 29.1)
Maternal obesity (% obese)	332	8.1 (NA)
Maternal pregnancy weight gain (kg)	221	9.5 (3.6, 16.8)
Maternal education (y)	259	11 (9, 12)
Presence of the father in the household (% present)	262	63.4 (NA)
Number of adults in the household	262	2 (1, 4)
Subject as adult		
Weight (kg)	445	64.2 (50.6, 88.0)
Height (m)	447	1.70 (1.58, 1.82)
BMI (kg/m^2)	445	22.2 (18.9, 30.0)
Sum of 2 skinfold thickness measurements (mm)	447	22 (13, 58)
Sum of 2 skinfold thickness measurements (*z* score)	447	−0.38 (−1.17, 1.50)
Sum of 2 skinfold thickness measurements above the 85th percentile	447	13.9 (NA)

[1] NA, not available.

not followed up and the subjects who completed the study. Sex distribution, birth weight for gestational age, maternal BMI, presence of the father in the household, and the number of adults in the household were not significantly different between these 2 groups. Placental weight was slightly higher (438 ± 94 compared with 420 ± 101 g, rank-sum test; $z = 3.3$, $P = 0.001$) and first-born status less frequent (16% compared with 22%; $\chi^2 = 5.4$, $P = 0.02$) in the group that completed follow-up.

The subjects' characteristics and the number of observations for each variable are presented in **Table 1**. Continuous variables are reported as medians (with 10th and 90th percentiles) to account for the skewness of the distribution. The primary outcome, SF *z* score in adulthood (±SD), was smaller in men than in women (−0.25 ± 1.02 and – 0.03 ± 1.07, respectively; rank-sum test: $z = -2.4$, $P = 0.02$). SF *z* score increased with increasing quintiles of birth weight for gestational age (test for trend: $z = 2.84$, $P = 0.004$) and maternal prepregnancy BMI (**Figure 1**). SF *z* score was higher in the subjects who were first-born than in those who had older siblings (0.30 ± 1.21 and – 0.21 ± 1.05, respectively; rank-sum test: $z = -3.3$, $P = 0.001$). The unadjusted RR for increased adiposity, defined as a skinfold thickness above the 85th percentile, for a first-born subject compared with a subject with older siblings was 2.0 (95% CI: 1.2, 3.3; $P = 0.01$). No significant association was observed among the following variables and adiposity in young adulthood: placental weight, maternal pregnancy weight gain, maternal education, presence of the father in the household, or the number of adults in the household. No significant interactions were observed.

The 9 potential independent variables chosen for this study were then introduced into a stepwise multiple linear regression model, with SF *z* score as the continuous outcome. The following 3 variables remained in the final model ($P < 0.0001$) and explained 12% of the variance: sex ($P = 0.03$), first-born status ($P = 0.0003$), and maternal prepregnancy BMI ($P < 0.0001$). The birth weight for gestational age, which was associated with adiposity in adulthood in the unadjusted analysis, did not contribute ($P = 0.39$) to the final model adjusted for maternal BMI, first-born status, and sex. Similar results were found when we used the sum of 2 skinfold-thickness measurements (in mm), unadjusted for sex and age, as an alternative outcome variable and used a robust variance estimate to account for the skewness of the SF *z* score. The 3 significant predictive variables were then introduced into a multiple logistic regression in which a skinfold thickness above the 85th percentile was used to define increased adiposity (**Table 2**). To explore the potential effect of the missing data, an additional model with imputed values for missing data was evaluated. The same 3 independent variables were identified by using stepwise linear regression and the adjusted ORs were similar to those of the initial model.

Because first pregnancies are more frequent in younger women than in older women, maternal age was introduced in a post hoc analysis. First-born status remained independently associated with the development of increased adiposity (OR = 3.4; 95% CI: 1.2, 9.6; $P = 0.02$), and maternal age did not significantly contribute to the model (OR = 0.94; 95% CI: 0.87, 1.02; $P = 0.13$).

DISCUSSION

There were 3 major findings of this prospective study of the early risk factors for increased adiposity in a cohort of young African American adults followed from birth: *1*) being a first-born child was significantly associated with increased adiposity in young adulthood; *2*) 3 risk factors that were present at birth accounted for 12% of the variability in adulthood adiposity: first-born status, female sex, and high maternal BMI; and *3*) after

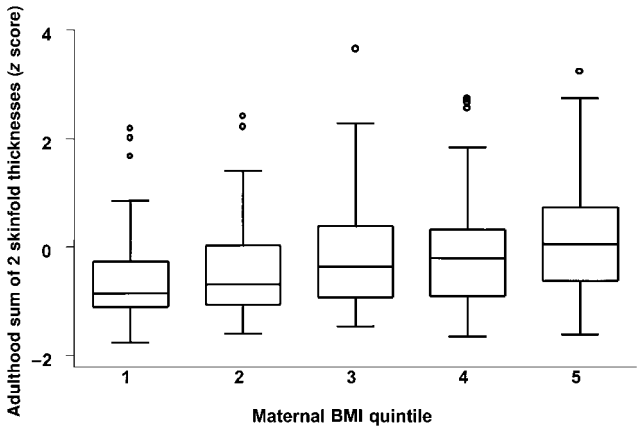

FIGURE 1. Sum of 2 skinfold-thickness measurements in young adulthood (*z* score) by maternal body mass index (BMI) quintiles. First quintile: 16.8, 20.0; second quintile: 20.0, 21.8; third quintile: 21.8, 24.0; fourth quintile: 24.0, 26.8; fifth quintile: 26.8, 40.7. Box plots represent median and interquartile ranges. Test for trend: $z = 5.09$, $P < 0.0001$.

TABLE 2
Multiple logistic regression (n = 253, P = 0.0004) of 3 significant risk factors present at birth for increased adiposity in young adulthood, defined by the sum of 2 skinfold thicknesses above the 85th percentile[1]

Risk factor	Odds ratio	95% CI	P
Female sex	2.7	(1.2, 6.1)	0.02
First-born status	4.0	(1.4, 11.2)	0.009
Maternal prepregnancy BMI (for each kg/m^2 increment)	1.15	(1.06, 1.25)	0.001

[1] Reference 33.

these 3 factors were accounted for, birth weight was unrelated to adulthood adiposity.

The association of first-born status and adulthood overweight was studied previously in European and African populations, with contradictory results (22, 34–37). Patterson et al (27) observed that the risk of obesity in 10-y-old US girls decreased as the number of siblings increased, but the effect of first-born status was not assessed. To our knowledge, ours was the first study of the effect of first-born status on the development of adiposity in young adulthood in an African American or other US sample. This large effect (adjusted OR = 4.0) could be due to gestational factors, postnatal factors, or both. In the present study, the group of first-born children was composed of children who were the first-born of several children and children without siblings; these 2 groups that might be exposed to different postnatal environments. Further studies are necessary to confirm this observation in other US samples and to distinguish between the effect of being first-born and that of being an only child.

An association between birth weight and adult weight was observed previously (9, 13, 16, 18, 19). The unadjusted analysis of the present data showed a similar association. However, after adjustment for first-born status, sex, and maternal BMI, the association between birth weight for gestational age and adult adiposity was not significant. Prior studies of this association did not adjust for all these confounders. Furthermore, most other studies used self-reported birth weight or birth weight reported by the mother or did not adjust for gestational age. The results of the present study suggest that an observed association of birth weight and adult weight might be confounded by other important factors in the absence of adjustment for these factors.

The results of this study strengthen previous reports of a higher prevalence of overweight in African American women than in African American men (2) and the association between maternal and offspring overweight, probably reflecting genetic and environmental influences (9, 11–15, 22, 43). The association of a lower socioeconomic status with overweight has been shown consistently in Americans of European descent but not in Americans of African descent (24, 27, 43, 44). The present analysis did not detect an association between parental socioeconomic status, assessed by maternal education and household composition, and the development of increased adiposity. This lack of association could be due to the relatively homogeneous low socioeconomic status of our subjects, who were recruited from an inner-city hospital between 1961 and 1965 (45).

The aim of the present analysis was to identify independent risk factors for increased adiposity rather than to predict the variability in adulthood adiposity associated with these factors. However, sex, first-born status, and maternal BMI accounted for 12% of the variability in adulthood adiposity. Therefore, >85% of this variability was associated with unmeasured factors present at birth, such as paternal BMI and gestational diabetes, or with the many factors operating between birth and young adulthood, such as diet and physical activity. Although sex and first-born status are nonmodifiable risk factors, their identification may help target prevention toward individuals with a high risk of increased adiposity. Maternal obesity can potentially be modified, but it is unknown whether this type of intervention has any effect on the risk of increased adiposity in the offspring. In our study population, 54% of the male and 57% of the female subjects with increased adiposity either were first-born or had an obese mother.

This study had certain limitations. Environmental and behavioral factors present between birth and young adulthood, such as food intake, physical activity, and family interactions (23, 25, 46, 47), were not measured and could have confounded the observed associations. Screening for gestational diabetes was not performed routinely at the time of this study and could not be included in this analysis. Maternal prepregnancy weight was collected by interview at the first prenatal visit, but the high agreement with the maternal weight measured at delivery, adjusted for gestational age, suggests acceptable accuracy of this value. The random selection of the representative sample at the beginning of the PBPP (29) would account for most of the decrease in sample size. However, 18% of the subjects were lost to follow-up during the CPP and 35% were lost during the PBPP. Most characteristics were similar between the subjects who were followed up and those who were not followed up, but placental weight was slightly higher, and first-born status less frequent, in the group that completed follow-up. Although these differences might have introduced a bias in the observed associations, this would have been the case only if the association of first-born status with adult increased adiposity had been different between subjects who were followed up and those who were not. The median birth weight for gestational age of this sample was lower than that in the more recent African American reference population (38). This difference could be explained by secular trends or other differences between the 2 samples.

The present study also had unique strengths. Unlike in several similar studies, the present data were collected as part of a prospective research protocol, thus reducing selection bias and recall bias and improving the reliability of the anthropometric measurements. Furthermore, skinfold thickness is a more direct indicator of adiposity than is BMI. To our knowledge, this was the first prospective cohort study of the early risk factors for increased adiposity that followed subjects from birth to young adulthood. Adjustment of birth weight for gestational age allowed the differentiation of premature infants from term infants with growth retardation. The results of the various analyses (univariate analysis, multivariate analysis, and use imputed values for missing data) led to similar conclusions.

In summary, first-born status, high maternal prepregnancy BMI, and female sex were all independently associated with increased adiposity in these young African American adults. Further studies are needed to identify other modifiable and nonmodifiable risk factors for obesity in children and young adults. Identification of these factors might help target evidence-based preventive strategies toward establishing healthy lifestyles for high-risk children before the onset of obesity.

We thank Jim Coleman for managing the data files.

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