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Development of Overweight Associated with Childbearing Depends on Smoking Habit: The Coronary Artery Risk Development in Young Adults (CARDIA) Study

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Abstract

Objective—To prospectively evaluate whether childbearing leads to development of overweight in women and to evaluate the role of other known risk factors.

Research Methods and Procedures—A prospective, multicenter observational study, the Coronary Artery Risk Development in Young Adults (CARDIA) Study from 1986 to 1996, examined subjects at baseline and in follow-up years 2, 5, 7, and 10. Included were 998 (328 black and 670 white) nulliparous women, age 18–30 years, who were not overweight at baseline. Relative odds for incident overweight (BMI ≥ 25 kg/m^2) associated with parity change (0, 1, or 2+) and risk factors were estimated using discrete-time survival models adjusted for baseline and time-dependent covariates.

Results—Parity change-association with development of overweight depended on smoking habit (interaction, $p < 0.001$). In multivariate adjusted models, 1 and 2+ births vs. 0, respectively, were associated with increased risk for development of overweight among never smokers [odds ratio (OR) = 2.66; 95% confidence interval (CI): 1.80, 3.93, and 2.10, 95% CI: 1.24, 3.56] and decreased risk among current smokers (OR = 0.41; 95% CI: 0.17, 0.96, and 0.36, 95% CI: 0.08, 1.65). Risk was increased for black vs. white race (OR = 3.49; 95% CI: 2.59, 4.69), frequent weight cycling (OR = 1.45; 95% CI: 1.03, 2.04), and high school education or less (OR = 2.21; 95% CI: 1.50, 3.26) and was decreased for highest physical activity quartile (OR = 0.62; 95% CI: 0.43, 0.90).

Discussion—Childbearing contributes to development of overweight in nonsmokers but not in smokers, where development of overweight is less likely in women who bear children. Race, education, and behaviors are important factors in development of overweight in young women.

Keywords

overweight; pregnancy; parity; smoking; African American

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Introduction

Overweight and obesity are associated with the development of hyperlipidemia, hypertension, cardiovascular disease (CVD),1 insulin resistance, type 2 diabetes, and shortened lifespan (1–3). Within the past three decades, the prevalence of overweight has doubled, and more than one-half of U.S. women between 20 and 45 years of age are considered overweight (BMI ≥ 25 kg/m^2), and about one-third are obese (BMI ≥ 30 kg/m^2) (1,4,5). The childbearing years have been identified as a critical life stage for excess weight gain and development of overweight. Both obesity and weight gain during young and middle adulthood are reported to elevate risk of CVD and early mortality in women (6–11). Some evidence has shown that high parity may elevate risk of CVD (12–17) and CVD mortality (15,16) and that excess weight gain putatively mediates the associations (14,18–20).

For most women, pregnancy-related weight gain is modest (averaging 0.5 to 1.5 kg for whites and 3 kg for blacks by 6 to 18 months postpartum), but individual variability is quite large (21). For example, ~15% to 25% of women are ≥5 kg heavier after delivery, and 7% to 10% become overweight within 1 or more years postpartum depending on race/ethnicity (21–23). High gestational gain (19,20,24–26), black race (20,26,27), pregravid overweight (28), primiparity (20), lower physical activity, and smoking cessation (19,20,24–26) have been cited as risk factors for excessive postpartum weight retention. Because these studies include only pregnant women without reference to nongravid or nonparous women, the upward weight shifts may partially reflect underlying secular trends as well as social and cultural influences on changes in employment, lifestyle, and behaviors in young and middle adulthood (21).

Three longitudinal studies, the NHANES I Epidemiological Follow-up Study (NHEFS) (18,28), the Coronary Artery Risk Development in Young Adults (CARDIA) Study, and the Black Women's Health Study (BWHS) (29) prospectively evaluated childbearing and weight changes controlling for secular trends and aging (30). Both CARDIA and Black Women's Health Study found greater weight gains for primiparous vs. nulliparous women. In CARDIA, primiparas vs. nulliparas had 2- to 3-kg higher 5-year gains and 1- to 6-kg higher 10-year gains that were not explained by behavioral changes, including lower physical activity and changes in smoking habits (28,30). In NHEFS, parous vs. nonparous white women gained 1.7 kg per birth and had a borderline significantly greater risk of overweight of 60% to 110% (18), independent of changes in physical activity, smoking habits, and other attributes. A second NHEFS analysis found that smokers who bore children tended to have lower 10-year weight gain than nonparous smokers, whereas the opposite was true for nonsmokers (31).

Although childbearing has a modest impact on weight gain, it has not been established whether pregnancy triggers excessive fat deposition and development of overweight for certain women. By prospectively examining change in parity and BMI assessed at 2- to 3-year intervals within a 10-year period in the CARDIA Study, the role of childbearing and other known risk factors in the development of overweight, apart from secular trends, may be elucidated in a large biracial cohort. The purposes of this study were to determine whether 1) initiation of childbearing is associated with increased risk of becoming overweight among those nulliparous and normal weight and 2) the association is confounded and/or modified by race (blacks vs. whites), age, dietary intake, education, weight cycling

[1]Nonstandard abbreviations: CVD, cardiovascular disease; NHEFS, NHANES I Epidemiological Follow-up Study; CARDIA, Coronary Artery Risk Development in Young Adults; OC, oral contraceptive; OR, odds ratio.

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history, employment, marital status, changes in smoking habits, and changes in physical activity.

Research Methods and Procedures

CARDIA Study Population

Details of the CARDIA Study design, methodology, and cohort characteristics have been reported elsewhere (32–34). Briefly, CARDIA is a multicenter, longitudinal observational study designed to track changes in risk factors for coronary heart disease in young black and white men and women within the U.S. Study Centers in Birmingham, AL, Chicago, IL, and Minneapolis, MN, recruited participants using community-based sampling, and Oakland, CA recruited participants by sampling the Kaiser Permanente Health Plan membership.

Between 1985 and 1986, baseline data collection was completed for 5115 subjects (2787 women) 18 to 30 years of age; 52% were black, and 48% were white. Follow-up examinations conducted at 2, 5, 7, and 10 years after baseline had high overall retention rates of ~91%, 86%, 81%, and 79% of surviving participants. The five examinations included a variety of physiological and self-report measures: anthropometry, demographics, medical and reproductive history [e.g., pregnancies, oral contraceptives (OCs)], and behavioral parameters (smoking habits, weight cycling, dietary intake, physical activity). These are described elsewhere (32–34).

Sample Selection Criteria

Of 2787 women, 2192 had information on pregnancies and births for one or more follow-up examinations. We also excluded those who had had a hysterectomy or removal of both ovaries ($n = 17$), had been pregnant within the past 3 months ($n = 46$), were currently pregnant ($n = 3$), currently breastfeeding ($n = 33$), or missing other data ($n = 23$) at baseline. For both races, women who were lost to follow-up or excluded did not differ in baseline demographic and anthropometric characteristics from the 2070, except that the whites who were excluded were more likely to be current or past smokers, have 12 or fewer years of education, and have higher parity at baseline. From the remaining 2070 women, the analysis included 998 (328 black and 670 white) women who were premenopausal, nulliparous, and normal weight (BMI < 25 kg/m^2) at baseline and had attended one or more follow-up examinations. Between baseline and year 10, measured nonpregnant weights were available for all five examinations in 799 (80%) and for four of five examinations in 147 (15%) women. A total of 976 women (97.8%) had measured nonpregnant weights obtained at both baseline and year 10. Institutional Review Boards at each participating study center approved the study.

Data Collection

At each examination, anthropometric measurements were obtained, and sociodemographic, reproductive history, and behavioral characteristics were assessed by self- and interviewer-administered questionnaires according to standardized protocol.

Anthropometric data were obtained at the baseline examination and years 2, 5, 7, and 10. Body weight was measured in street clothing to the nearest 0.2 kg on a calibrated balance beam scale, and height was measured without shoes to the nearest 0.5 cm using a vertically mounted metal centimeter ruler and a metal carpenter’s square. BMI was computed as weight in kilograms divided by squared height in meters. Weight change was calculated by subtraction of baseline weight from weight at the end of follow-up.

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Definition of Incident Overweight During Follow-up

At each follow-up examination (years 2, 5, 7, and 10), we classified each woman as overweight (BMI ≥ 25 kg/m^2) or normal weight based on her BMI. For time-points where BMI was missing (because of nonattendance or current pregnancy), the outcome classification for the previous time-point with available data was assigned to a single interim time-point (for 130 women) and to a maximum of two interim time-points (for 31 women with missing weights at examinations before year 10). Subjects were excluded from subsequent follow-up time-points if they became overweight or if they were missing the follow-up examination weight in year 10 (n = 22).

Time-Dependent Parity Groups

At each examination, participants were asked by the interviewer whether they were currently pregnant and the number of times they had been pregnant since the previous examination, including the number of abortions, miscarriages, stillbirths, live births, and delivery dates. Gestations of 20 weeks or longer were counted as births, and those <20 weeks were coded as "short" pregnancies (miscarriages/abortions). Based on the cumulative number of births reported after baseline, women were categorized into one of three time-dependent parity groups, zero births, one birth, and two or more births, for each of the four time-points. Parity groups were maintained for the subsequent examination unless new births were reported. Short pregnancies were counted for each interval and modeled as a time-dependent continuous variable.

Time-Dependent Covariates

At each examination, subjects were asked about sociodemographic and behavioral characteristics including cigarette smoking habit, OC use, marital status, and employment. Questionnaires asked whether the subject had ever smoked cigarettes regularly for at least 3 months. Regularly was defined as at least five cigarettes per week almost every week (33). Subjects who said yes were asked whether they still smoked cigarettes regularly. Based on their responses, women were classified as never, current, or former smokers. Current smokers were defined as those who reported at the examination that they regularly smoked cigarettes. Current OC use was defined as use of OCs at the time of the examination.

Each characteristic was constructed as a time-dependent covariate based on classification of women at each examination into the following categories: smoking habit—never, former, or current smoker; OC use—never, former, or current user; marital status—never married, widowed, separated or divorced, or married; employment status—full-time outside home, part-time outside home, or none. At each examination, assessments of physical activity were obtained using the interviewer-administered CARDIA Physical Activity History described previously (35). Race-specific quartiles for physical activity at each examination were formed because of the skewedness of the data (data not shown).

Baseline Covariates

Dietary intake and history of weight cycling were measured at baseline. Dietary intake during the previous month was assessed using the CARDIA Dietary History administered by a trained interviewer (36). Daily dietary nutrient measures of alcohol (milliliters per day), total fat, total protein, total carbohydrate (grams per day), and energy intake (kilojoules per day) were used to obtain the percentages of kilocalories from fat, protein, and carbohydrate. Subjects were asked "How many times would you say that you have lost and gained back 10 lb or more?" The responses were categorized into one of three groups, 0, 1–2, or 2 or more times, to construct the history of weight cycling variable.

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Statistical Methods

χ^2 tests were used to assess associations between overweight outcome groups and baseline demographic and behavioral characteristics (smoking, education, OC use, weight cycling, marital status, employment) within each race group. Student's *t* test statistics were used to assess differences in baseline height, weight, BMI, dietary intake, and age between outcome groups; *p* values were obtained from two-sided tests (significance: $p < 0.05$). A Kruskal-Wallis one-way test was used to assess differences in alcohol intake and physical activity caused by skewedness in the distributions.

We calculated the incidence of overweight (%) within each interval (0 to 2, 2 to 5, 5 to 7, and 7 to 10 years) by dividing the number of women who became overweight during each interval by the number of women at risk of becoming overweight. New cases of overweight for each interval were counted and categorized by parity group (0, 1, or 2 or more births) defined as total number of births delivered during the follow-up interval.

Cox's extension of the proportional hazard model for discrete time was used to model the logit of the hazard of overweight in relation to parity group at each follow-up examination (years 2, 5, 7, and 10) and other covariates (37). Relative odds ratios (ORs) of becoming overweight were calculated for one birth and two or more births groups separately using zero births as the reference group. Through the use of appropriate cross-product terms, race, age, height, study center, and baseline (BMI, education, weight cycling, dietary intake) as well as time-dependent covariables (smoking, physical activity, number of short pregnancies, alcohol intake, OC use, marital status, employment) were examined as potential confounders and/or effect modifiers in the association between parity groups and the risk of becoming overweight. Based on these analyses, multivariate models estimated smoking group-specific relative hazard ratios for parity groups and the risk of becoming overweight adjusted for study center, race, age, baseline covariates (BMI, education, dietary intake, weight cycling) and time-dependent covariates (physical activity, number of short pregnancies).

Results

Overall, 175 (53%) black and 183 (27%) white women became overweight during follow-up. Among blacks and whites, respectively, 169 (52%) and 373 (56%) remained nulliparous, 98 (30%) and 130 (19%) gave birth once (primiparas), and 61 (19%) and 167 (25%) gave birth two or more times (multiparas) during follow-up.

In both races, women who became overweight were heavier ($p < 0.001$) at baseline (Table 1). Baseline BMI level was consistent across follow-up parity groups (data not shown). White women who became overweight had lower education and dietary carbohydrate intake and more weight cycling episodes than those who remained normal weight, as well as borderline higher dietary fat and protein intakes. Black women who became overweight did not differ in other baseline characteristics from those who did not become overweight.

The crude incidence of overweight (%) within each time interval (0 to 2, 2 to 5, 5 to 7, and 7 to 10 years) was higher in both blacks and whites who had either one birth or two or more births during follow-up compared with women who never gave birth during each interval (Table 2). The incidence rates according to parity did not differ by race (interaction, $p = 0.62$) but did vary by cigarette smoking habit (interaction, $p < 0.001$). Among never smokers, the incidence of overweight for each time interval (Table 3) was higher for women who had one birth and two or more births during follow-up than for those who had none. In contrast, the risk of becoming overweight was lower or similar for parous compared with

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nulliparous women among current smokers and moderately increased with parity among former smokers.

Median weight gains during follow-up were much higher for those who became overweight than for those who did not become overweight (range, 8 to 14 vs. 2 to 5 kg) across all smoking categories (Table 4). Among both never and former smokers, primi- and multiparas who became overweight tended to have higher median weight gains (by 2 to 5 kg more) compared with nulliparas who became overweight. Among current smokers who became overweight, primiparas or multiparas had median weight gains similar to nulliparas (~9 kg). Differences in mean weight gain had a similar pattern.

In discrete time multivariate proportional hazard models, smoking status was a strong effect modifier of the association of parity group with risk of becoming overweight ($p < 0.001$). Other two-way interactions for parity by race, age, education, weight cycling, baseline BMI, and time-dependent physical activity were not significant. In both unadjusted and adjusted models (Table 5), risk of becoming overweight among never smokers was doubled for both one birth and two or more birth groups compared with no births. Among former smokers, risk was 1.6 to 1.7 times greater for one or more births, although this was not significant. Last, among current smokers, risk of becoming overweight was reduced by more than one-half for one or more births vs. none. These associations were not explained by race, baseline BMI, education, weight cycling, time-dependent physical activity, or other covariates.

Among parous women, the risk of developing overweight during follow-up did not vary by time from delivery to the next examination. Time interval categories of ≤3, >3 to 6, >6 to 12, and >12 months showed similar percentages of women who developed overweight, with a trend for a higher proportion of women with longer time since delivery to become overweight (data not Shown). Weight change between CARDIA examinations was also similar across time intervals from delivery to next examination among parous women within each separate outcome group (overweight and not overweight).

In multivariate models (Table 5), black women were 3.5 times more likely than whites to become overweight. Adjusted risk of becoming overweight was increased by 65% to 120% with high school education or less, decreased by one-third for the highest physical activity quartile vs. the lowest, and increased by 45% for two or more weight cycling episodes vs. none at baseline.

Discussion

Only one prospective study of white women (25 to 45 years of age at baseline), of whom <50 delivered a first birth during follow-up, has previously estimated risk of overweight associated with parity change based on two weight measurements (at baseline and 10 years later), and this study had inconclusive results (18). Our study examined development of overweight associated with childbearing in a large population-based biracial cohort of black and white nulliparous women (18 to 30 years of age at baseline), of whom >456 delivered a first birth during 10 years of follow-up. We found that smoking habit was a strong effect modifier of the association between childbearing and development of overweight. Incidence of overweight during follow-up for parous compared with nulliparous women was doubled among never smokers, reduced by more than one-half among current smokers, and somewhat greater among former smokers.

Prospective studies controlling for change in smoking status and other covariates have reported relatively modest average weight gains associated with childbearing (18,28,30). In CARDIA, primiparas gained 1 kg more than nulliparas during 10 years among those who were normal weight at baseline (blacks and whites) (30). In NHEFS, parous vs. nonparous

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white women gained 1.7 kg per additional birth (18). However, effect modification by smoking status in the association between parity change and 10-year weight gain had been previously reported in NHEFS, although the results were equivocal (31). Among nonsmokers, one or more births vs. none was associated with higher weight gain, but childbearing had the opposite effect on weight gain in smokers, although smoking status was assessed only at baseline (31).

In studies of pregnant women, smoking status has been associated with postpartum weight. Women who continue to smoke after delivery are reported to retain 1.1 kg less weight than nonsmokers at 6 months or more postpartum (23,38). Smoking cessation during pregnancy has been associated with 2- to 3-kg higher weight retention at 1 year postpartum compared with nonsmokers and those who continued to smoke after pregnancy in one study (39) and with higher weight gain a decade or more later in one study (25) but not in another study (19). Cross-sectional studies of nonpregnant women have shown an association between cigarette smoking and lower body weight in women (40–42), and longitudinal studies have shown similar or reduced weight gain in smokers compared with nonsmokers among nonpregnant subjects (43,44). However, our findings are novel, showing that women who continue to smoke after pregnancy have lower long-term weight gain than smokers who do not bear children (nulliparas).

The effect of maternal smoking on adiposity, fat distribution and deposition during pregnancy, and postpartum fat loss has not been studied extensively. Therefore, the mechanism by which smoking may reduce the risk of becoming overweight with childbearing among current smokers is not well understood. However, it is well established that components of cigarette smoke suppress appetite and increase metabolic rate (45–47). Inadequate maternal fat deposition, especially among women who are not overweight before childbearing, may restrict fetal growth and deplete maternal fat stores. Among smokers, lower body fat stores, higher metabolic rate, and appetite suppression, combined with increased nutritional needs and anabolic demands of pregnancy, may lead to postpartum weight deficits that result in lower long-term weight gain among parous smokers relative to nulliparous smokers.

Given the adverse health risks associated with smoking to both the mother and her fetus, smoking is clearly not a viable public health strategy to avoid overweight. Smoking during pregnancy is associated with fetal growth retardation, placenta previa and abruptio, preterm delivery, and higher perinatal mortality (48,49). Women are advised to quit smoking or reduce the amount smoked during pregnancy to improve infant outcomes (49–51). Women who smoke during pregnancy, particularly heavier smokers, are more likely to have insufficient gestational weight gain (49,52–54), but adequate maternal gestational weight gain does not overcome the adverse effects of smoking on fetal growth and health (50,55,56).

Our study found no dose-response relationship between number of births and risk of overweight. Risk of overweight, reflected in overlapping confidence intervals, was similar for primiparous and multiparous women compared with nulliparous women. CARDIA has previously reported excess weight gain after a first birth but no additional increment with higher order births (28,30). In addition, compared with whites, blacks were three times more likely to become overweight in multivariate models including age, parity, smoking, and other risk factors. A higher prevalence of overweight and obesity for black women has been reported in numerous population-based epidemiological studies (1,4,5). In the 1988 National Maternal and Infant Health Survey, 22% of black women vs. 8% of white women were ≥9 kg heavier than preconception levels at 18 months postpartum (22). Our findings

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conclusively show that the higher risk among black women is not caused by childbearing or other behaviors measured in this study.

An inverse association between physical activity and body weight has been shown in both cross-sectional and intervention studies (57), and change in physical activity has been shown in prospective population-based studies in U.S. men and women. However, few population-based studies have examined whether low levels of physical activity are a risk factor for the development of obesity, or conversely, whether high levels are protective. In NHEFS, risk of 10-year weight gain >13 kg quadrupled among women with low recreational physical activity (58). Both increased physical activity in the CARDIA Study cohort (59) and increased physical fitness in the Aerobics Center Longitudinal cohort have been associated with attenuated weight gain and reduced risk of significant weight gain (60) up to a decade later. Our finding of an inverse association between risk of overweight and physical activity is consistent with these reports and supports the hypothesis that higher physical activity prevents weight gain in young women, independent of childbearing status, and that changes in physical activity do not explain the parity-associated weight gains. Overall, employment and marital status did not confound the associations.

This prospective 10-year analysis in the CARDIA cohort, based on several weight measurements and covariates over 2- to 3-year intervals, permitted estimation of the relative odds of becoming overweight associated with childbearing, after controlling for secular trends as well as known demographic and behavioral risk factors. By restricting our analysis to women who were normal weight before pregnancy, we avoided confounding from the differential effects of smoking on energy metabolism in overweight and normal weight women (61) to assess effect modification by smoking in the association of childbearing and long-term weight gain. In our study sample at baseline, 22% were current smokers, which is similar to the 1990 U.S. national statistics reporting that 25% of women smoked in the year before pregnancy (62). In 1990, only 23% of smokers in a national survey reported that they stopped smoking completely on learning of their pregnancy (62). Smoking habit in our study was modeled as a time-dependent covariate based on self-report at each examination to account for smoking cessation reported at follow-up examinations in parous vs. nulliparous groups.

Ours is the largest prospective sample of parous and nonparous women of reproductive age followed at repeated intervals during 10 years. However, 50% of black women compared with 20% of white women were parous before the baseline CARDIA examination, resulting in fewer blacks than whites in the sample. Thus, our study findings may be most informative about black women who begin childbearing at later ages, and studies that include younger black women are necessary to confirm these associations.

Limitations of the study include potential confounding from unmeasured covariates such as change in dietary habits during follow-up or other factors, lack of information on whether women continued to smoke during pregnancy, and reliance on self-reported smoking habit. Risk estimates for parous current smokers are somewhat imprecise because of the small number who became overweight. Other limitations include lack of gestational gain measurements, no information on smoking behaviors during pregnancy in CARDIA, and the variable time interval from pregnancy delivery until the next follow-up CARDIA examination. However, the risk of overweight and weight gain did not vary by time from delivery to next examination among parous women. By continuing to classify parous women who were missing interim weights due to being pregnant as not overweight at an interim time-point (based on a previous weight measurement), our results are conservative and potentially underestimate the risk associated with parity. Last, few women in our sample reached a BMI of ≥30 kg/m^2, which precluded examination of obesity.

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Our findings show that, for the majority of women, initiation of childbearing is associated with development of overweight. Incidence of overweight associated with childbearing was more than doubled among never smokers, who comprise up to 70% of pregnant women. This evidence suggests that the reproductive years are a critical time to implement public health screening to evaluate excess postpartum weight retention and to implement interventions to prevent overweight and obesity in women. Excess weight gain after pregnancy not only increases maternal risk for chronic disease in the future, but it adversely affects maternal and fetal health during subsequent pregnancies. Maternal pregravid overweight is the most common high-risk obstetric condition and is associated with increased maternal and infant morbidity, including gestational diabetes and hypertension disorders in the woman, as well as neural tube defects, macrosomia, and perinatal mortality in the newborn (63).

Based on our findings, premenopausal women most susceptible to developing overweight are primiparas who are nonsmokers. African-American women, as well as women with less education, more frequent weight loss episodes, or lower physical activity levels, are also at greater risk. Development of overweight after a first birth may be related to either a biological susceptibility to excess fat tissue deposition during pregnancy, significant changes in lifestyle accompanying childrearing responsibilities, or other factors, including hormonal changes that are primarily influenced by a first birth. Further investigation is needed to determine potential mechanisms influencing adaptations to pregnancy and the demands of childbearing and childrearing that result in large weight gains for certain women.

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References

1. National Institutes of Health, National Heart, Lung, and Blood Institute. The Evidence Report. Washington, DC: National Institutes of Health; 1998. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults.
2. Peeters A, Barendregt JJ, Willekens F, Mackenbach JP, AlMamun A, Bonneux L. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. Ann Intern Med. 2003; 138:24–32. [PubMed: 12513041]
3. Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath CW. Body-mass index and mortality in a prospective cohort of U.S. adults. N Engl J Med. 1999; 341:1097–1105. [PubMed: 10511607]
4. Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960–1994. Int J Obes Relat Metab Disord. 1998; 22:39–47. [PubMed: 9481598]
5. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. JAMA. 2002; 288:1723–1727. [PubMed: 12365955]
6. Manson JE, Colditz GA, Stampfer MJ, et al. A prospective study of obesity and coronary heart disease in women. N Engl J Med. 1990; 322:882–889. [PubMed: 2314422]
7. Willett WC, Manson JE, Stampfer MJ, et al. Weight, weight change, and coronary heart disease in women. Risk within the ‘normal’ weight range. JAMA. 1995; 273:461–465. [PubMed: 7654270]
8. Kannel WB. Metabolic risk factors for coronary heart disease in women: perspective from the Framingham Study. Am Heart J. 1987; 114:413–419. [PubMed: 3604900]
9. Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. Circulation. 1983; 67:968–977. [PubMed: 6219830]

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10. Williamson DF, Pamuk E, Thun M, et al. Prospective study of intentional weight loss and mortality in never-smoking overweight US white women aged 40–64 years. Am J Epidemiol. 1995; 141:1128–1141. [PubMed: 7771451]
11. Manson JE, Willett WC, Stampfer MJ, et al. Body weight and mortality among women. N Engl J Med. 1995; 333:677–685. [PubMed: 7637744]
12. Ness RB, Harris T, Cobb J, et al. Number of pregnancies and the subsequent risk of cardiovascular disease. N Engl J Med. 1993; 328:1528–1533. [PubMed: 8267704]
13. de Kleijn MJJ, van der Schouw YT, van der Graaf Y. Reproductive history and cardiovascular disease risk in postmenopausal women. A review of the literature. Maturitas. 1999; 33:7–36. [PubMed: 10585171]
14. Ness RB, Schotland HM, Flegal KM, Shofer FS. Reproductive history and coronary heart disease risk in women. Epidemiol Rev. 1994; 16:298–314. [PubMed: 7713181]
15. Dekker JM, Schouten EG. Number of pregnancies and the risk of cardiovascular disease. N Engl J Med. 1993; 329:1893–1894. [PubMed: 8247048]
16. Green A, Beral V, Moser K. Mortality in women in relation to their childbearing history. BMJ. 1988; 297:391–395. [PubMed: 3408979]
17. Rosenberg L, Palmer JR, Rao RS, Adams-Campbell LL. Risk factors for coronary heart disease in African American women. Am J Epidemiol. 1999; 150:904–909. [PubMed: 10547135]
18. Williamson DF, Madams J, Pamuk E, Flegal KM, Kendrick JS, Serdula MK. A prospective study of childbearing and 10-year weight gain in US white women 25 to 45 years of age. Int J Obes Relat Metab Disord. 1994; 18:561–569. [PubMed: 7951478]
19. Rooney BL, Schauberger CW. Excess pregnancy weight gain and long-term obesity: one decade later. Obstet Gynecol. 2002; 100:245–252. [PubMed: 12151145]
20. Gunderson EP, Abrams B, Selvin S. The relative importance of gestational gain and maternal characteristics associated with the risk of becoming overweight after pregnancy. Int J Obes Relat Metab Disord. 2000; 24:1660–1668. [PubMed: 11126221]
21. Gunderson EP, Abrams B. The epidemiology of gestational weight gain and body weight change after pregnancy. Epidemiol Rev. 1999; 21:261–275. [PubMed: 10682262]
22. Keppel K, Taffel S. Pregnancy-related weight gain and retention: implications of the 1990 Institute of Medicine Guidelines. Am J Pub Health. 1993; 83:1100–1103. [PubMed: 8342716]
23. Greene GW, Smiciklas-Wright H, Scholl TO, Karp RJ. Postpartum weight change: how much of the weight gained in pregnancy will be lost after delivery? Obstet Gynecol. 1988; 71:701–707. [PubMed: 3357658]
24. Ohlin A, Rossner S. Trends in eating patterns, physical activity and socio-demographic factors in relation to postpartum body weight development. Br J Nutr. 1994; 71:457–470. [PubMed: 8011603]
25. Linne Y, Dye L, Barkeling B, Rossner S. Weight development over time in parous women—the SPAWN study—15 years follow-up. Int J Obes Relat Metab Disord. 2003; 12:1516–1522. [PubMed: 14634683]
26. Boardley DJ, Sargent RG, Coker AL, et al. The relationship between diet, activity, and other factors, and postpartum weight change by race. Obstet Gynecol. 1995; 86:834–838. [PubMed: 7566859]
27. Parker JD, Abrams B. Differences in postpartum weight retention between black and white mothers. Obstet Gynecol. 1993; 81:768–774. [PubMed: 8469470]
28. Gunderson EP, Murtaugh M, Lewis CE, Quesenberry C, Smith West D, Sidney S. Excess gains in weight and waist circumference associated with childbearing: the Coronary Artery Risk Development in Young Adults Study (CARDIA). Int J Obes Relat Metab Disord. 2004; 28:525–535. [PubMed: 14770188]
29. Rosenberg L, Palmer JR, Wise LA, Horton NJ, Kumanyika SK, Adams-Campbell LL. A prospective study of the effect of childbearing on weight gain in African-American women. Obes Res. 2003; 12:1526–1535. [PubMed: 14694218]
30. Smith DE, Lewis CE, Caveny JL, Perkins LL, Burke GL, Bild DE. Longitudinal changes in adiposity associated with pregnancy. JAMA. 1994; 271:1747–1751. [PubMed: 8196117]

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31. Wolfe WS, Sobal J, Olson C, Frongillo EA, Williamson DF. Parity-associated weight gain and its modification by sociodemographic and behavioral factors: a prospective analysis in US women. Int J Obes Relat Metab Disord. 1997; 21:802–810. [PubMed: 9376894]
32. Cutter GR, Burke GL, Dyer AR, et al. Cardiovscular risk factors in young adults. The CARDIA Baseline Monograph. Control Clin Trials. 1991; 2:1S–77S. [PubMed: 1851696]
33. Friedman GD, Cutter GR, Donahue RP, et al. CARDIA: study design, recruitment, and some characteristics of the examined subjects. J Clin Epidemiol. 1988; 41:1105–1116. [PubMed: 3204420]
34. Hughes GH, Cutter G, Donahue R, et al. Recruitment in the Coronary Artery Disease Risk Development in Young Adults (Cardia) Study. Control Clin Trials. 1987; 8:68S–73S. [PubMed: 3440391]
35. Andersen N, Jacobs DR, Sidney S, et al. Change and secular trends in physical activity patterns in young adults: a seven-year longitudinal follow-up in the Coronary Artery Risk Development in Young Adults Study (CARDIA). Am J Epidemiol. 1996; 143:351–362. [PubMed: 8633619]
36. MacDonald A, Van Horn L, Slattery M, et al. The CARDIA dietary history. J Am Diet Assoc. 1991; 91:1104–1112. [PubMed: 1918764]
37. Cox DR. Regression models and life-tables (with discussion). J Roy Stat Soc B. 1972; 43:187–220.
38. Schauberger CW, Rooney BL, Brimer LM. Factors that influence weight loss in the puerperium. Obstet Gynecol. 1992; 79:424–429. [PubMed: 1738527]
39. Ohlin A, Rossner S. Maternal body weight development after pregnancy. Int J Obes Relat Metab Disord. 1990; 14:159–173.
40. Barrett-Connor E, Khaw KT. Cigarette smoking and increased central adiposity. Ann Intern Med. 1989; 111:783–787. [PubMed: 2817625]
41. Rasky E, Stronegger WJ, Freidl W. The relationship between body weight and patterns of smoking in women and men. Int J Epidemiol. 1996; 25:1208–1212. [PubMed: 9027526]
42. Istvan JA, Cunningham TW, Garfinkel. Cigarette smoking and body weight in the Cancer Prevention Study I. Int J Epidemiol. 1992; 21:849–853. [PubMed: 1468844]
43. Wagenknecht LE, Craven TE, Preisser JS, Manolio TA, Winders S, Hulley SB. Ten-year trends in cigarette smoking among young adults, 1986–1996: the CARDIA Study. Ann Epidemiol. 1998; 8:301–307. [PubMed: 9669612]
44. Williamson DF. Descriptive epidemiology of body weight and weight change in U.S. adults. Ann Intern Med. 1993; 119:646–649. [PubMed: 8363190]
45. Perkins KA, Epstein HL, Marks BL, Stiller RL, Jacob RG. The effect of nicotine on energy expenditure during light physical activity. N Engl J Med. 1989; 320:898–903. [PubMed: 2927460]
46. Li MD, Kane JK, Konu O. Nicotine, body weight and potential implications in the treatment of obesity. Curr Top Med Chem. 2003; 3:899–919. [PubMed: 12678839]
47. Jo YH, Talmage DA, Role LW. Nicotinic receptor-mediated effects on appetite and food intake. J Neurobiol. 2002; 53:618–632. [PubMed: 12436425]
48. English PB, Eskenazi B. Reinterpreting the effects of maternal smoking on infant birth weight and perinatal mortality: a multivariate approach to birthweight standardization. Int J Epidemiol. 1992; 21:1097–1105. [PubMed: 1483814]
49. Institute of Medicine. Nutrition During Pregnancy. Part I: Weight Gain. Washington, DC: Food and Nutrition Board, National Academy of Sciences; 1990.
50. Sexton M, Hebel JR. A clinical trial of change in maternal smoking and its effect on birth weight. JAMA. 1984; 251:911–915. [PubMed: 6363731]
51. Muscati SK, Gray-Donald K, Newson EE. Interaction of smoking and maternal weight status in influencing infant size. Can J Public Health. 1994; 85:407–412. [PubMed: 7895216]
52. Strychar IM, Chabot C, Champagne F, et al. Psychosocial and lifestyle factors associated with insufficient and excessive maternal weight gain during pregnancy. J Am Diet Assoc. 2000; 100:353–356. [PubMed: 10719411]
53. Olson CM, Strawderman MS. Modifiable behavioral factors in a biopsychosocial model predict inadequate and excessive gestational weight gain. J Am Diet Assoc. 2003; 103:48–54. [PubMed: 12525793]

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54. Picone TA, Allen LH, Schramm MM, Olsen PN. Pregnancy outcome in North American women. I. Effects of diet, cigarette smoking, and psychological stress on maternal weight gain. Am J Clin Nutr. 1982; 36:1205–1213. [PubMed: 7148739]

55. Davies DP, Abernethy M. Cigarette smoking in pregnancy: associations with maternal weight gain and fetal growth. Lancet. 1976; 1:385–387. [PubMed: 55649]

56. Groff JY, Mullen PD, Mongoven M, Burau K. Prenatal weight, gain patterns and infant birthweight associated with maternal smoking. Birth. 1997; 24:234–239. [PubMed: 9460314]

57. DiPietro, L. Physical Activity, Body Weight, and Adiposity: An Epidemiologic Perspective. Baltimore, MD: Williams & Wilkins; 1995.

58. Williamson DF, Madams J, Anda RF, Kleinman JC, Kahn HS, Byers T. Recreational physical activity and ten-year weight change in a US national cohort. Int J Obes Relat Metab Disord. 1993; 17:279–286. [PubMed: 8389337]

59. Schmitz KH, Jacobs DR Jr, Leon AS, Schreiner PJ, Sternfeld B. Physical activity and body weight: associations over ten years in the CARDIA study. Coronary Artery Risk Development in Young Adults. Int J Obes Relat Metab Disord. 2000; 24:1475–1487. [PubMed: 11126345]

60. DiPietro L, Kohl HW III, Barlow CE, Blair SN. Improvements in cardiorespiratory fitness attenuate age-related weight gain in healthy men and women: the Aerobics Center Longitudinal Study. Int J Obes Relat Metab Disord. 1998; 22:55–62. [PubMed: 9481600]

61. Audrain JE, Klesges RC, Klesges LM. Relationship between obesity and the metabolic effects of smoking in women. Health Psychol. 1995; 14:116–123. [PubMed: 7789346]

62. LeClere FB, Wilson JB. Smoking behavior of recent mothers, 18–44 years of age, before and after pregnancy: United States, 1990. Adv Data. 1997; 288:1–11. [PubMed: 10182807]

63. Galtier-Dereure F, Boegner C, Bringer J. Obesity and pregnancy: complications and cost. Am J Clin Nutr. 2000; 71:1242S–1248S. [PubMed: 10799397]

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Table 1

Baseline characteristics and development of overweight at the end of follow-up by race

	Black			White		
Baseline characteristics	**Became overweight (BMI ≥ 25 kg/m^2; *n* = 175)**	**Not overweight (BMI < 25 kg/m^2; *n* = 153)**	***p***	**Became overweight (BMI ≥ 25 kg/m^2; *n* = 183)**	**Not overweight (BMI < 25 kg/m^2; *n* = 487)**	***p***
Education [*n* (%)]			0.37			0.02
High school of less	59 (48.4)	63 (51.6)		39 (34.8)	73 (65.2)	
Some college	77 (55.8)	61 (44.2)		62 (31.0)	138 (69.0)	
4+ years of college	39 (57.3)	29 (42.7)		82 (22.9)	276 (77.1)	
Employment [*n* (%)]			0.22			0.92
Full-time	83 (54.3)	70 (45.8)		121 (27.4)	321 (72.6)	
Part-time	47 (47.0)	53 (53.0)		37 (26.2)	104 (73.8)	
Not employed	45 (60.0)	30 (40.0)		25 (28.7)	62 (71.3)	
Marital status [*n* (%)]			0.74			0.42
Never married	161 (54.0)	137 (46.0)		131 (26.0)	372 (74.0)	
Widow/divorced/separated	2 (50.0)	2 (50.0)		13 (29.6)	31 (70.4)	
Married	12 (46.2)	14 (53.9)		39 (31.7)	84 (68.3)	
Smoking habit [*n* (%)]			0.31			0.34
Never smoker	127 (53.6)	110 (46.4)		111 (28.1)	284 (71.9)	
Former smoker	10 (71.4)	4 (28.6)		29 (22.3)	101 (77.7)	
Current smoker	38 (49.3)	39 (50.7)		43 (29.7)	102 (70.3)	
OCs [*n* (%)]			0.19			0.60
Never	45 (47.9)	49 (52.1)		47 (24.7)	143 (75.3)	
Past	70 (59.8)	47 (40.2)		76 (29.0)	186 (71.0)	
Current	60 (51.3)	57 (48.7)		60 (27.5)	158 (72.5)	
Weight cycling [*n* (%)]			0.11			<0.001
None	58 (46.0)	68 (54.0)		31 (17.6)	145 (82.4)	
1 to 2 times	73 (57.9)	53 (42.1)		48 (19.0)	204 (81.0)	
>2 times	44 (57.9)	32 (42.1)		104 (43.0)	138 (57.0)	
Center [*n* (%)]			0.16			0.14
Alabama	40 (54.1)	34 (46.0)		32 (27.6)	84 (72.4)	

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Baseline characteristics	Black			White		
	Became overweight (BMI ≥ 25 kg/m^2; *n* = 175)	**Not overweight (BMI < 25 kg/m^2; *n* = 153)**	***p***	**Became overweight (BMI ≥ 25 kg/m^2; *n* = 183)**	**Not overweight (BMI < 25 kg/m^2; *n* = 487)**	***p***
Chicago	40 (65.6)	21 (34.4)		37 (21.8)	133 (78.2)	
Minneapolis	27 (46.6)	31 (53.5)		62 (32.8)	127 (67.2)	
Oakland	68 (50.4)	67 (49.6)		52 (26.7)	143 (73.3)	
Age (years) [mean (SD)]	22.8 (3.4)	22.5 (3.5)	0.42	25.1 (3.5)	25.2 (3.4)	0.78
Weight (kg) [mean (SD)]	60.3 (7.1)	54.5 (6.9)	<0.001	61.9 (6.5)	56.6 (6.3)	<0.001
BMI (kg/m^2) [mean (SD)]	22.6 (1.6)	20.2 (1.8)	<0.001	22.8 (1.5)	20.8 (1.7)	<0.001
Height (cm) [mean (SD)]	163.3 (7.4)	164.4 (6.8)	0.20	164.9 (6.4)	165.3 (6.3)	0.44
Dietary intake [mean (SD)]						
Percent kilocalories fat	37.4 (5.9)	37.6 (5.0)†	0.73	36.9 (6.9)	35.8 (6.2)	0.05
Percent kilocalories carbohydrates	48.0 (7.7)	48.9 (6.4)†	0.25	46.1 (7.4)	47.7 (7.2)	0.01
Percent kilocalories protein	14.1 (2.6)	13.7 (2.2)†	0.19	15.5 (2.9)	15.1 (2.6)	0.09
Physical activity score [median, (interquartile range)]*	280 (153 to 455)	252.0 (116 to 420)	0.25	390 (252 to 570)	410 (249 to 624)	0.29

*Kruskal-wallis test.

†*n* = 150.

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Table 2

Number of incident overweight women for each time interval divided by the number of women at risk, for each follow-up examination (%) stratified by race and parity groups during follow-up

Parity groups by race	Year 0 to 2 (overweight/no. at risk)		Year 2 to 5 (overweight/no. at risk)		Year 5 to 7 (overweight/no. at risk)		Year 7 to 10 (overweight/no. at risk)	
	n	%	*n*	%	*n*	%	*n*	%
Black (total)	50/328	15.2	56/277	20.2	23/219	10.5	46/193	23.8
0 births	45/292	15.4	31/201	15.4	13/155	8.4	15/105	14.3
1 birth	5/36	13.9	24/66	36.4	5/44	11.4	19/53	35.9
2 or more births	0	0	1/10	10.0	5/20	25.0	12/35	34.3
White (total)	43/670	6.4	50/625	8.0	42/572	7.3	48/520	9.2
0 births	40/624	6.4	31/490	6.3	28/400	7.0	24/292	8.2
1 birth	2/43	4.7	14/91	15.4	8/98	8.2	14/100	14.0
2 or more births	1/3	33.3	5/44	11.4	6/74	8.1	10/128	7.8

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Table 3

Number of incident overweight women for each time interval divided by the number of women at risk, for each follow-up examination (%) stratified by smoking group and parity groups during follow-up

Parity groups by smoking group	Year 2 (overweight/no. at risk)		Year 5 (overweight/no. at risk)		Year 7 (overweight/no. at risk)		Year 10 (overweight/no. at risk)	
	n	%	*n*	%	*n*	%	*n*	%
Never (total)	60/575	10.4	63/513	12.3	37/448	8.3	58/406	14.3
0 births	53/525	10.1	33/394	8.4	19/317	6.0	21/233	9.0
1 birth	6/48	12.5	24/86	27.9	10/76	13.2	25/84	29.8
2 or more births	1/2	50.0	6/33	18.2	8/55	14.6	12/89	13.5
Former (total)	18/255	7.1	18/236	7.6	18/216	8.3	21/193	10.9
0 births	18/234	7.7	10/179	5.6	12/145	8.3	7/95	7.4
1 birth	0/20	0	8/42	19.0	3/42	7.1	6/48	12.5
2 or more births	0/1	0	0/15	0	3/29	10.3	8/50	16.0
Current (total)	15/165	9.1	25/150	16.7	10/124	8.1	15/111	13.5
0 births	14/154	9.1	19/116	16.4	10/91	11.0	11/67	16.4
1 birth	1/11	9.1	6/28	21.4	0/23	0	2/20	10.0
2 or more births	0/0		0/6	0	0/10	0	2/24	8.3

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Table 4

Weight gain (kg) from baseline to end of follow-up according to whether women did or did not become overweight during follow-up stratified by smoking habit at end of follow-up and parity groups

		Not overweight					Became overweight					Difference in median weight gain for became overweight vs. not overweight
Smoking groups	**Parity groups**	***n***	**Mean**	**SD**	**Median**	**Interquartile range**	***n***	**Mean**	**SD**	**Median**	**Interquartile range**	
Never												
	0 births	219	2.9	4.6	2.6	6.8	126	9.1	6.0	8.0	6.6	+5.4
	1 birth	60	2.4	4.9	2.3	6.1	65	12.7	6.1	12.0	8.0	+9.7
	2+ births	78	3.6	4.5	3.6	6.1	27	9.8	3.8	10.0	5.9	+6.4
Former												
	0 births	94	2.5	4.7	2.5	6.4	47	8.9	5.8	8.0	7.0	+5.5
	1 birth	43	4.4	4.4	4.6	5.0	17	14.5	6.4	13.6	5.7	+9.0
	2+ births	43	3.3	4.8	3.4	6.3	11	11.9	5.4	12.4	7.6	+9.0
Current												
	0 births	59	3.4	3.8	3.9	4.8	54	9.9	5.7	9.1	6.7	+5.2
	1 birth	19	3.3	7.0	1.7	12.3	9	13.1	9.5	9.4	5.6	+7.7
	2+ births	22	4.6	3.8	4.1	4.2	2	8.5	3.7	8.5	5.2	+4.4

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Table 5

Unadjusted and multivariate adjusted relative odds of becoming overweight and 95% confidence intervals (CIs) stratified by smoking status and parity groups

Time-dependent smoking groups*	Unadjusted relative odds	95% CI	Multivariate adjusted relative odds†	95% CI
Never smokers				
0 birth	1.00	Reference	1.00	Reference
1 birth	2.90	2.07, 4.05	2.66	1.80, 3.93
2+ births	1.80	1.13, 2.87	2.10	1.24, 3.56
Former smokers				
0 birth	1.00	Reference	1.00	Reference
1 birth	1.38	0.76, 2.49	1.70	0.86, 3.36
2+ births	1.38	0.68, 2.81	1.59	0.69, 3.67
Current smokers				
0 birth	1.00	Reference	1.00	Reference
1 birth	0.86	0.41, 1.81	0.41	0.17, 0.96
2+ births	0.35	0.08, 1.50	0.36	0.08, 1.65
Race				
Whites			1.00	Reference
Blacks			3.49	2.59, 4.69
Education (at baseline)				
High school or less			2.21	1.50, 3.26
Some college			1.65	1.18, 2.29
4 years college			1.00	Reference
Weight cycling (at baseline)				
None			1.00	Reference
1 to 2 times			0.98	0.71, 1.37
>2 times			1.45	1.03, 2.04
Physical activity (time dependent)				
First quartile			1.00	Reference
Second quartile			1.13	0.81, 1.59
Third quartile			0.78	0.54, 1.13
Fourth quartile			0.62	0.43, 0.90

*Interaction term for time-dependent smoking groups by parity groups in association with risk of becoming overweight ($p < 0.001$).

†Estimates adjusted for race, age, study center, baseline BMI, dietary intake (percent kilocalories from carbohydrate and protein), number of short pregnancies (time dependent), and other covariates shown.