OBESITY IN WOMEN OF CHILDBEARING AGE: RISKS, PREVENTION, AND TREATMENT

Mary E. Cogswell, RN, DrPH, Geraldine S. Perry, RD, DrPH, Laura A. Schieve, PhD, and William H. Dietz, MD, PhD

This article reviews the health burden of obesity, its treatment and prevention, and potential barriers to care with special emphasis on adult women of childbearing age. From 1988 to 1994, 22% of nonpregnant women 18–49 years old in the United States were overweight (body mass index [BMI] ≥ 25–29.9), and 22% were obese (BMI ≥ 30). Both conditions increase the risk of chronic disease and mortality, and among women of childbearing age, overweight and obesity also increase the risk of infertility and adverse pregnancy outcomes.

The three main strategies for preventing obesity are weight maintenance, weight loss for overweight and obese persons, and physical activity for all. More than 44% of nonpregnant women of childbearing age are trying to lose weight, and more than 33% are trying to maintain weight, but less than 21% of women of childbearing age use the recommended combination of physical activity and caloric restriction to try to lose or maintain weight. Pregnant women should try to gain no more than the recommended weight gain range for their prepregnancy BMI, yet about one third gain more weight.

Although research has shown that advice from physicians can have an impact on their patients' eating habits and physical activity, many health professionals either provide no such advice or give inappropriate advice to women of childbearing age. Barriers may include inadequate reimbursement, time constraints, and lack of professional training. Frequent contact with women of childbearing age provides obstetricians and gynecologists and nurse specialists an opportunity to prevent and treat obesity successfully. (Prim Care Update Ob/Gyns 2001;8: 89–105. © 2001 Elsevier Science Inc. All rights reserved.)

From the Division of Nutrition and Physical Activity and the Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, Atlanta, Georgia

Twenty-two percent of male and female Americans aged ≥20 years are overweight (body mass index [BMI] ≥ 25.0–29.9), and an additional 23% are obese (BMI ≥ 30).[1] Both conditions increase the risk of chronic disease and premature mortality. In addition, among women of childbearing age, overweight and obesity increase the risk of infertility and adverse outcomes of pregnancy. Thus, the fact that in the last few decades, the prevalence of overweight and obesity rose more among women of childbearing age than among older women or men is cause for alarm. Findings such as these have increased interest in research on obesity, its treatment, and its prevention.

Primary care providers can do a great deal to prevent morbidity and mortality due to obesity by evaluating all patients and treating overweight and obese patients and by offering appropriate counseling on diet and physical activity to all their patients.[2] Studies suggest that people who report receiving advice from their physician about weight loss are more likely to try to lose weight than are those who do not report such advice.[3,4] In addition, those who are counseled to lose weight are more likely to report that they are eating fewer calories and less fat and that they are increasing their physical activity.[4] Randomized controlled trials demonstrate that brief (5–10 minutes) physician counseling can increase physical activity.[5]

Unfortunately, most physicians do not counsel their patients about weight loss and physical activity. In 1996, 44% of obese women (BMI > 30) in the United States who had had a routine checkup in the last 12 months recalled that their doctor, nurse, or another health professional advised them to lose weight.[3] In 1995, 33% of adult women who saw a physician in the previous year for a medical checkup recalled their doctor recommending that they begin or continue to engage in any type of exercise or physical activity.[6] The 1995 National Ambulatory Medical Care Survey indicated that obstetrician/gynecologists were less likely than cardiologists and general or family practitioners to

  1068-607X/01/$20.00 • PII S1068-607X(00)00087-1

Table 1. Classification of Overweight and Obesity by BMI, Waist Circumference and Associated Disease Risks (from NIH[9])*

Classification	BMI	Obesity Class	Waist Circumference† ≤88 cm (≤35 in)	Waist Circumference† >88 cm (>35 in)
Underweight	<18.5		—	—
Normal	18.5–24.9		—	—
Overweight	25.0–29.9		Increased	High
Obesity	30.0–34.9	I	High	Very high
	35.0–39.9	II	Very high	Very high
Extreme obesity	≥40	III	Extremely high	Extremely high

* Disease risk for type 2 diabetes, hypertension, and CVD.
† Increased waist circumference can also be a marker for increased risk even in persons of normal weight.
BMI, body mass index (weight in kilograms divided by square of height in meters).

counsel their patients about physical activity, diet, and losing weight.[7]

Evidence now suggests that significant health benefits can be obtained by performing a moderate amount of physical activity (eg, 30 minutes of brisk walking or raking leaves) on most if not all days of the week.[8] Clinicians seeking information about evaluating and treating overweight and obesity can consult guidelines published in 1998 by the National Institutes of Health (NIH).[9] In this paper, we discuss the definitions of *overweight* and *obesity*, the health burden of obesity, and the treatment and prevention of obesity (including potential barriers), with emphasis on adult women during childbearing years. We also used data from the Third National Health and Nutrition Examination Survey (1988–1994) to assess the prevalence of obesity among women aged ≥18 years and draw on data from the Pregnancy Nutrition Surveillance System to illustrate the risks of obesity and gestational weight gain on selected birth outcomes.

Overweight and Obesity: Definitions

An obese person has excess body fat. There are several phenotypes of obesity depending where the excess fat is located. Both the classification of these phenotypes of obesity and their implications remain unsettled,[10] but it is generally recognized that having excessive abdominal fat is independently associated with chronic disease.

The measurement of body fat overall (eg, by underwater weighing and deuterium oxide dilution) and by specific sites (eg, by computed tomography) is often not practical in clinical settings. Hence, BMI, which is the weight in kilograms divided by the square of the height in meters (kg/m^2), is commonly used to estimate a person's overall body fat, and waist circumference is used to estimate abdominal fat. Another way to calculate BMI is provided by the following formula: (weight in pounds/[height in inches]^2) × 704.5.

The 1998 NIH criteria for overweight and obesity are based on the association between BMI and mortality.[9] In nonpregnant women, a BMI of 18.5–24.9 represents a healthy weight; 25.0–29.9, overweight; ≥30, obesity. Epidemiologic data show an increase in mortality at BMI of ≥25, with the greatest increase occurring at ≥30.[11,12]

The BMI criteria used to define prepregnancy overweight and obesity differ from the criteria used for nonpregnant women.[9,13] BMI of <19.8 is low, BMI of 19.8–26 is average, BMI of >26.0–29.0 is high, and BMI of >29 is very high.[13] There is no statistical or scientific basis for using one set of values over the other when assessing prepregnancy weight for height.[13] Although the relationship between prepregnancy BMI and various fetal or maternal outcomes is generally considered linear, with no well-defined thresholds, two analyses suggest an increased risk of perinatal mortality with BMI ≥30.[14,15] More detail about these analyses is given in the section on perinatal mortality.

Up until the 1998 NIH report, there were no consensus criteria for assessing abdominal obesity. That report indicates that there is an increased relative risk for type 2 diabetes, dyslipidemia, hypertension, and cardiovascular disease in persons whose waist circumference is >88 cm (Table 1). In the present paper, the criteria in each cited study for overweight, obesity, and abdominal obesity will be specified when available and applicable.

Prevalence of Overweight and Obesity

In 1988–1994, 44% of nonpregnant women 18–49 years old in the United States were overweight (22%) or obese (22%; Table 2). Both overweight and obesity were highest among minority groups. Sixty percent of African-American and 62% of Mexican-American women were either overweight or obese. Both overweight and obesity were more prevalent in older age groups (Figure 1).

In all age groups, the prevalence of obesity has increased over time, with the most marked increases seen in the last two decades. Among women aged 20–29 years, for example, obesity increased from 7% in 1960–1962 to 11% in 1976–1980 to 17% in 1988–1994 (Figure 1). During the same time period, the prevalence of overweight among women aged 20–29 years almost doubled, from 11% in 1960–1962 to 16% in

Table 2. Prevalence of Overweight and Obesity Among U.S. Women Aged 18–49 Years, NHANES III, 1988–1994

Race/Ethnicity	*N*	Overweight (%)*	Obese (%)†	Total (%)‡
African-American	1,865	27.8	32.1	59.9
Mexican-American	1,673	31.3	30.4	61.7
White	1,691	20.0	19.0	39.0
Total	5,472	21.9	21.7	43.6

* Overweight defined as BMI 25–29.9.
† Obesity defined as BMI ≥30.
‡ Total includes women aged 18–49 years of ethnic designations other than African American, Mexican American, or white.
All prevalences are weighted to reflect the U.S. population; unweighted sample sizes are given.

1976–1980 to 19% in 1988–1994.[1] In contrast, there was little or no change seen in women aged 30–39 or 40–49 years.

Overweight also seems to have become more common among women immediately before pregnancy. Representative national data are not available, but data from the Pregnancy Nutrition Surveillance System for low-income women in 23 states and territories show that the prevalence of high and very high prepregnancy BMI combined (BMI > 26) increased from 30% in 1989 to 36% in 1996.[16]

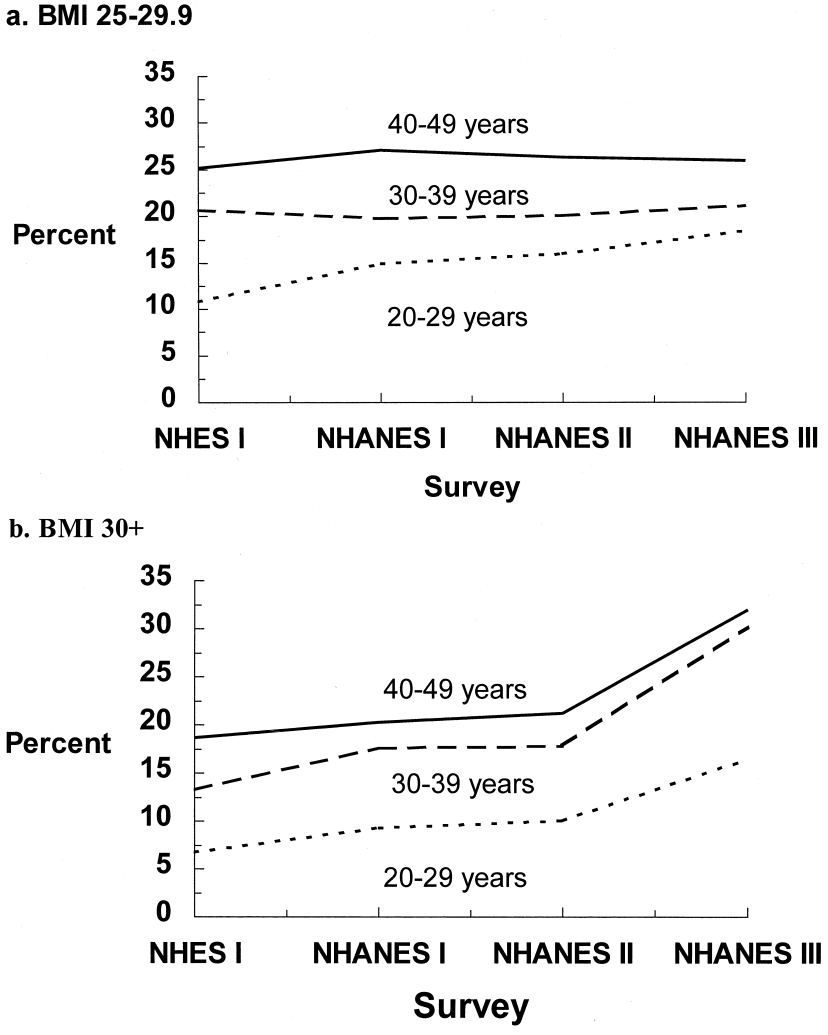

Figure 1. The prevalence of (a) overweight and (b) obesity among U.S. women of childbearing age across time, adapted from Flegal et al.[1] Data include the first National Health Examination Survey (NHES, 1960–1962) and the first, second, and third National Health and Nutrition Examination Surveys (NHANES I, 1971–1974, NHANES II, 1976–1980; and NHANES III, 1988–1994).

Incidence of Obesity and Major Weight Gain

Because national estimates of the current incidence (rate of new cases) of overweight and obesity are not available, one cannot determine whether earlier patterns have been maintained. A longitudinal study conducted by Williamson and colleagues from 1971–1975 to 1981–1985 found that 13.5% of women aged 35–44 years became overweight or obese (BMI > 27.3) over the 10-year period.[17] Among women aged 25–34 years, the incidence of overweight and obesity was 11.5%.

The incidence of obesity is related to the amount of weight gained over time. In the Williamson study, the incidence of major weight gain (an increase in BMI of ≥5) was greatest among overweight women aged 25–34 years (14.2%).[17] The lessons from this study are that obesity prevention should begin before the mid-20s and that physicians should pay particular attention to women already overweight.

The incidence of obesity can be increased by childbearing (through excessive weight gain during pregnancy) and rearing children (through lifestyle changes). Among U.S. white women aged 25–45 years in 1971–1974, the 10-year risk of obesity (BMI > 30) for women with one live birth was twice that of women without a live birth.[18] In addition, women who gave birth at least once in 10 years gained 1.7–2.2 kg more than women who did not. In another longitudinal study, this one among women aged

18–30 years at baseline, the mean difference in weight gain between primiparous and nulliparous women during a 5-year follow-up was 1.8 kg for white women and 3.0 kg for black women.[19] Although the greater weight gains associated with childbearing appear modest, they may still elevate the risk of obesity.

Health Risks of Overweight and Obesity

Overweight and obesity increase the risk of two of the leading causes of death, diabetes and cardiovascular disease.[9,20] During the childbearing years, overweight and obesity can complicate fertility, and among women who do become pregnant, it contributes to several mechanical, cardiovascular, and metabolic complications. Thus, overweight and obesity represent major challenges to the obstetrician/gynecologist. The risks associated with overweight and obesity are described in more detail below.

Mortality Risks

In women of childbearing age, the risk of mortality increases with both low and high BMI. In a 12-year follow-up study that included 154,474 white women aged 30–54 years who had never smoked, the relative risk of mortality from all causes and from cardiovascular disease was lowest for women with a BMI of 19.0–21.9.[11] At BMIs well above or below this range, risk was substantially higher. For example, compared with 30–44-year-old women with a BMI of 21, women with a BMI of 26.5 had a 50%-increased risk of mortality from cardiovascular disease. The relationship between BMI and mortality diminished with older age at baseline, however. A recent study indicated that the association between BMI and mortality is similar in black and white women.[21]

Effects of Weight Loss on Mortality

In 1996, a national study found that 44–51% of nonpregnant women aged 18–49 years were trying to lose weight and that 33–37% were trying to maintain their weight.[22] Seventy percent of overweight or obese women (BMI $>$ 25) were trying to lose weight.[22] In one of the few studies to examine the effects of intentional weight loss on mortality, in which the study group consisted of 15,069 nonsmoking white women aged 40–64 years of age with BMI of $>$27 and obesity-related health conditions, intentional weight loss protected against all-cause mortality in all but women with the slowest rate of weight loss ($<$20 lbs in more than 1 year[23]). By cause, the relationship was strongest for diabetes-associated mortality. This study also found that among women with no pre-existing illness (n = 28,387), intentional weight loss of $\geq$20 lbs in $\leq$1 year protected against all cause mortality (relative risk, 0.77; 95% confidence interval, 0.59–1.00). A subsequent study that used as a comparison group women who had never lost $\geq$20 lbs found that reporting one or more losses of $\geq$20 lbs was not associated with mortality among adult women aged 18–69 years.[24] This analysis adjusted for age, BMI, smoking status and estrogen use among other variables, but it and the other study were limited by potential biases from self-report and a lack of information on maintenance of weight loss over time.

Chronic Disease Risks

The risk of chronic disease increases with greater BMI, as demonstrated by the very large Nurses' Health Study. In the Nurses' Health Study, which included 121,700 female registered nurses (98% white) aged 30–55 years who were enrolled in 1976 from 11 states, information on height and weight was collected from mailed questionnaires at age 18 years, at the 1976 baseline, and on subsequent surveys. Medical diagnoses were confirmed through medical record review. In this study, the risks of cardiovascular disease, diabetes, and colon cancer all increased with obesity, and these increases were generally similar to those found in smaller studies and in other ethnic groups.[25–27] The prevalence of chronic disease overall has been found to increase with increased weight at similar rates in white, black, and Mexican-American women in the United States.[28]

Age-adjusted relative risks of chronic diseases found in various reports from the Nurses' Health Study are shown in Table 3. The strongest associations with overweight and obesity are with type 2 diabetes; compared with women with a BMI of $<$22, the incidence of diabetes was 5.5 times as high in women with a BMI of 25–26.9 and 61 times as high for those women with a BMI of $\geq$35. A BMI of $\geq$29 was also associated with increased risks of fatal and nonfatal coronary heart disease, ischemic stroke, pulmonary embolism, colon cancer, and gallstones but not with hemorrhagic stroke or breast cancer (Table 3). Obesity (BMI $>$ 31) protected slightly against risk of breast cancer in premenopausal women but was not associated with this risk in postmenopausal women. However, among postmenopausal women who never used hormone replacement, obesity increased risk of breast cancer by 59% (relative risk, 1.59 for BMI $>$ 31 versus BMI $\leq$ 20; 95% confidence interval, 1.09–2.32).[29]

In general, the associations between obesity and risk of chronic disease were not changed much by adjusting for additional factors,

such as cigarette smoking in the case of coronary heart disease, or the use of oral contraceptives or postmenopausal hormones in the case of stroke. However, the relative risks of coronary heart disease (both fatal and nonfatal) and stroke with higher BMI were greater among persons who smoked,[30,31] and the relative risk of coronary heart disease in obese persons was increased among those with hypertension, high cholesterol, or diabetes.[30]

With all the diseases in Table 3, weight gain between the ages of 18 and 30–55 generally increased the risks of chronic disease.[30–35] Weight loss between ages 18 and 30–55, on the other hand, reduced the risk of diabetes,[35] but not of ischemic stroke or gallstones.[31,34] Weight loss intent was not known. The effects of weight loss on risk of coronary heart disease, pulmonary embolism, and colon cancer were not assessed.[30,33,36]

Obesity is associated with osteoarthritis as well. Data from the Framingham Osteoarthritis Study on 796 women indicated that increased BMI was associated with onset of knee osteoarthritis;[37] in addition, a 2-unit reduction in BMI in 10 years protected against the onset of symptomatic knee osteoarthritis.[37] Among 58 women who had unilateral osteoarthritis, the incidence of contralateral knee arthritis was 47% among women with a BMI of ≥26 but just 10% among those with a BMI of 17–22.9.[38]

Independent of BMI, central adiposity is associated with increased risk of type 2 diabetes, the presence of cardiovascular risk factors, and coronary heart disease.[39–42] With control for age, BMI, and other factors, women in the Nurse's Health Study whose waist circumference was ≥96.4 cm were 22 times as likely to develop diabetes and three times as likely to develop coronary heart disease as women with a waist circumference of ≤71 cm.[40,41]

Fertility

Obesity increases the risk of primary and secondary infertility as well as menstrual irregularities.[43–46] These associations generally stem from the relationship between obesity and polycystic ovary disease (PCOD), the most common cause of anovulatory infertility. However, an increased risk for infertility due to an ovulatory disorder has also been reported among overweight and obese women who lack a diagnosis of PCOD.[46] Additionally, menstrual disturbances and anovulation are more common in obese patients.[45,47,48] Evidence suggests PCOD infertility can usually be traced to excessive production of androgen. Multiple pathways may lead to this effect, several of which are related to obesity and its common consequence, hyperinsulinemia.[49,50]

In an early study, Rogers and Mitchell[43] reported that 43% of patients with menstrual disorders were obese (≥20% over ideal weight), versus 13% of patients of similar age but with normal menstrual function. Later studies confirmed this association. Zaadstra et al.[44] demonstrated that both an increased waist-hip ratio and an increased BMI were associated with reduced conception rates (after adjusting for smoking, parity, and age) among women undergoing artificial insemination. Balen et al,[45] who studied more than 1,700 women with PCOD, found that the risk for both primary and secondary infertility was increased by >70% among women with a BMI of >30 compared with women whose BMI was 20–30. Rich-Edwards et al,[46] who performed a nested case-control study within the Nurses' Health Study, reported that the risk of infertility was 2.7 times higher among women whose BMI at age 18 had been >30 than in those whose BMI at age 18 had been 20–21.9. These authors also reported that those risks were present among obese women both with and without a diagnosis of PCOD.

Losing weight decreases androgen concentrations and increases ovulation among obese women.[51,52] However, increased ovulation has also been associated with treatment with insulin-sensitizing agents, such as metformin, with little change in body weight reported.[53]

Finally, infertility treatment may be complicated by obesity. Ovulation induction therapy with either clomiphene or gonadotropins appears less effective among women with a high BMI.[54,55] Among treated patients with PCOD who conceive, obese women are at greater risk for spontaneous abortion than other women.[55]

Pregnancy Outcomes

Obesity increases the risk of maternal morbidity, complications of labor and delivery, neural tube defects, and perinatal mortality. These risks, which may be complicated by gaining excessive weight during pregnancy, are described in more detail below.

Maternal Morbidity. The Nurses' Health Study II provides information on the risk of maternal morbidity associated with prepregnancy obesity (Table 4). For example, in a comparison with women having a prepregnancy BMI of <20, the relative risk of gestational diabetes for women with prepregnancy BMI of 25–29.9 was 2.2 (95% confidence interval [CI], 1.7–2.8), and for those whose pre-pregnancy BMI was ≥30, it was 3.0 (95% CI, 2.2–4.0). Adjustment for age, family history of gestational diabetes, parity, ethnicity, and pregravid physical activity level did not change the association.[56] The risk for gestational diabetes increased with greater weight gains between ages 18 and 25–42,[56] which suggests that avoiding substantial weight gain in the early

Table 3. Age-Adjusted Relative Risks of Selected Chronic Diseases with Increased Body Mass Index (BMI), Nurses' Health Study Cohort, 121,700 Female Registered Nurses Aged 30–55 Years in 1976 (98% White) Living in 11 U.S. States and Returning a Mailed Questionnaire

Disease and Citation	Sample at Risk	Case Definition for Disease	Follow-Up Period	Cases of Disease	BMI-Referent Group	BMI Group(s)	Relative Risk
Type 2 diabetes[32]	113,861	At least one classic symptom plus plasma fasting glucose ≥140 mg/dL or random plasma glucose ≥200 mg/dL; or at least two elevated plasma glucose levels ≥200 mg/dL in absence of symptoms	1976–1984	873	<22	22–22.9	2.2
						23–23.9	3.6
						24–24.9	3.1
						25–26.9	5.5
						27–28.9	10.1
						29–30.9	20.0
						31–32.9	29.6
						33–34.9	40.2
						≥35	60.9
Fatal coronary heart disease[30]	115,886	Fatal myocardial infarction confirmed by hospital records or autopsy or the listing of coronary heart disease on the death certificate if it was the underlying and only plausible cause and evidence of previous coronary heart disease was available.	1976–1984	83	<21	21–22.9	1.4
						23–24.9	1.2
						25–28.9	2.0
						≤29	2.6
Nonfatal myocardial infarction[30]	115,886	World Health Organization criteria: Symptoms and either diagnostic electrocardiographic changes or elevated cardiac enzyme levels	1976–1984	238 confirmed, 68 probable	<21	21–22.9	1.1
						23–24.9	1.2
						25–28.9	1.4
						≥29	2.6
Angina pectoris[30]	115,886	Coronary angiography demonstrating more than 70% obstruction of any coronary artery, coronary artery bypass grafting or angioplasty, or ST-segment depression of at least 1 mm on exercise testing in conjunction with a positive response to a mailed Rose questionnaire.	1978–1984	216	<21	21–22.9	1.1
						23–24.9	1.5
						25–28.9	1.6
						≥29	1.5
Primary pulmonary embolism[33]	112,822	Diagnosis of pulmonary embolism in the absence of a predisposing factor. Confirmed by high probability ventilation perfusion lung scan, positive pulmonary angiogram, or autopsy.	1976–1992	125	<21	21–22.9	0.7
						23–24.9	1.1
						25–28.9	1.7
						≥29	3.2
All pulmonary embolism[33]	112,822	Diagnosis of pulmonary embolism. Confirmed by high probability ventilation perfusion lung scan, positive pulmonary angiogram, or autopsy.	1976–1992	125	<21	21–22.9	0.7
						23–24.9	1.2
						25–28.9	1.7
						≥29	3.4
Ischemic stroke[31]	116,759	National Survey of Stroke criteria: evidence on medical records of neurologic deficit with sudden or rapid onset that persisted for more than 24 hours or until death. Ischemic stroke, due to thrombotic or embolic occlusion of cerebral artery.	1976–1992	403	<21	21–22.9	0.9
						23–24.9	1.1
						25–26.9	1.0
						27–28.9	1.5
						29–31.9	1.5
						≥32	1.8

(Continued)

Table 3. (Continued).

Disease and Citation	Sample at Risk	Case Definition for Disease	Follow-Up Period	Cases of Disease	BMI-Referent Group	BMI Group(s)	Relative Risk
Hemorrhagic stroke[31]	116,759	National Survey of Stroke criteria: evidence on medical records of neurologic deficit with sudden or rapid onset that persisted for more than 24 hours or until death. Hemorrhagic stroke, due to either subarachnoid or intraparencyhmal hemorrhage	1976–1992	403	<21	21–22.9	0.8
						23–24.9	0.8
						25–26.9	0.6
						27–28.9	0.5
						29–31.9	0.7
						≤32	0.6
Gallstones[34]	88,837	Cholecystectomy or symptomatic gallstones confirmed by x-ray films or ultrasonographic examination	1980–1984	602	<20	20–20.9	0.8*
						21–21.9	0.9
						22–22.9	1.2
						23–23.9	1.1
						24–24.9	1.3
						25–26.9	2.2
						27–28.9	2.6
						29–31.9	3.0
						≥32	4.8
Colon cancer[36]	13,057	Women undergoing colonoscopy or sigmoidoscopy between 1986 and 1992; adenoma confirmed by histopathologic report (including carcinoma in situ)	1986–1992	330 adenoma of distal colon	<21	21–22.9	0.8†
						23–24.9	1.2
						25–28.9	1.0
						≥29	1.4
Breast cancer[29]	95,256	Self-report, 96% confirmed through hospital records	1976–1992	1,000 premenopausal	≤20	20.1–21	1.1
						21.1–22	0.9
						22.1–23	1.1
						23.1–24	1.1
						24.1–25	0.9
						25.1–26	1.1
						26.1–28	0.8
						28.1–31	0.8
						>31	0.6
Breast cancer[29]	95,256	Self report, 96% confirmed through hospital records	1976–1992	1,517 postmenopausal	≤20	20.1–21	1.1
						21.1–22	1.0
						22.1–23	1.2
						23.1–24	1.1
						24.1–25	1.1
						25.1–26	0.9
						26.1–28	1.2
						28.1–31	1.2
						>31	1.0

BMI = weight in kilograms divided by squared height in meters.
* Adjusted for age in 1980, BMI at age of 18, and BMI in 1976.
† Adjusted for age, family history of colorectal cancer, and previous endoscopy.

adult years is an important strategy for preventing gestational diabetes.

The risk of gestational hypertension and pre-eclampsia also increases with higher BMI (Table 4). Compared with women with prepregnancy BMI of 21–22.9, women with pre-pregnancy BMI of 25–29.9 were 1.7 times (95% CI, 1.1–2.5) as likely to develop hypertension during pregnancy, and women with a pre-pregnancy BMI of ≥29 were 2.2 times (95% CI, 1.3–3.6) as likely to do so. Adjustment for age, parity, family history of hypertension, smoking status, and history of elevated cholesterol slightly increased both associations.[57] The association between pre-pregnancy BMI and pre-eclampsia was not as strong: After multivariate adjustment, compared with women with prepregnancy BMI of 21–22.9, women with a prepregnancy BMI of ≥29 were 2.1 times (95% CI, 1.0–4.6) as likely to develop pre-eclampsia.

Other studies in primary care populations have shown similar associations between pregnancy morbidity and increased pre-pregnancy BMI.[15,58–63] Two of the studies indicated that the association between pre-pregnancy BMI and mor-

Table 4. Age-Adjusted Relative Risks Adverse Reproductive Outcomes with Increased Prepregnancy Body Mass Index (BMI), Nurses' Health Study II Cohort, 116,670 Female Registered Nurses Aged 25–42 Years in 1989 Living in 14 U.S. States

Disease	Sample at Risk	Case Definition for Disease	Follow-Up Period	Cases of Disease	BMI Referent Group	BMI Group(s)	Relative Risk
Gestational diabetes mellitus[56]	14,613 with a singleton pregnancy and no history of diabetes	Self-reported gestational diabetes mellitus with validation by medical record review in a subset	1990–1994	722	<20	20–21.9	1.0
						22–24.9	1.2
						25–29.9	2.2
						≥30	3.0
Gestational hypertension[57]	15,262 with a singleton pregnancy and no history of hypertension	Baseline systolic BP <140 mmHg and a systolic increase of >30 mmHg on two occasions; a baseline diastolic BP <90 mmHg and an increase of >15 mmHg on two occasions; and urinalysis data demonstrating ≤1+proteinuria.	1991–1995	216	21–22.9	<21	0.7
						23–24.9	1.4
						25–28.9	1.7
						≥29	2.2
Preeclampsia[57]	15,262 with a singleton pregnancy and no history of hypertension	Baseline systolic BP <140 mmHg and a systolic increase of >30 mmHg on two occasions; a baseline diastolic BP <90 mmHg and an increase of >15 mmHg on two occasions; urinalysis data demonstrating ≥2+proteinuria or at least 300 mg of urine protein in 24 h, and absolute systolic BP >140 mmHg or diastolic BP >90 mmHG.	1991–1995	86	21–22.9	<21	0.7
						23–24.9	1.1
						25–28.9	0.8
						≥29	1.9

BMI = weight in kilograms divided by squared height in meters in 1989.

bidity was not affected by gestational weight gain.[60,64]

Operative Delivery. In a retrospective cohort study, the risk of cesarean delivery was more than 60% higher among obese women (BMI ≥ 29) than nonobese women (BMI < 29) after age, multiparity, high birthweight, and pregnancy complications were controlled.[65] The association may be even stronger than indicated by this study, because overweight women (BMI 25– <29) were included in the nonobese comparison group. The authors hypothesized that dystocia due to an increased deposition of soft tissue in the maternal pelvis may lead to the observed increase in cesarean delivery. In addition, women may find it more difficult to push during delivery because of increased visceral fat.

Infant Birthweight and Gestational Age at Delivery. Among overweight and obese women, the risk of adverse infant outcomes varies by the gestational weight gain.[13,66–69] Unadjusted data from nine states in the Pregnancy Nutrition Surveillance System on 176,928 white, black, or Hispanic women delivering liveborn, singleton infants at 39–41 weeks of gestation in 1990–1993 are shown unadjusted in Figures 2a and 2b. Data from the same source on 266,172 white, black, or Hispanic women delivering a liveborn, singleton infant at 29–42 weeks of gestation are shown in Figure 2c. Adjustment for age, parity, and smoking status did not change the results. As in other studies, the risks of low birthweight (at term) and preterm delivery are generally lower among women with higher pre-pregnancy BMI (Figure 2a and 2c). On average, heavy women need to gain less weight during pregnancy than other women to prevent low birthweight and preterm delivery. At the same time, the risk of high birth weight may increase with greater weight gain among women of all BMI categories (Figure 2b). As in other studies,[67,68] these findings from the Pregnancy Nutrition Surveillance System were consistent across racial or ethnic groups.

Congenital Malformations. Independently of other risk factors, maternal obesity appears to increase the risk of delivering an infant with major congenital malformations, neural tube defects in particular (Table 5). Several recent case-control and cohort studies have addressed this issue. Case-control studies, however, may inflate the associations because of potential biases in maternal recall.[70–73] In two large cohort studies, which is a type generally considered superior to case-control studies, overweight and obese women were 20–40% more likely than average weight

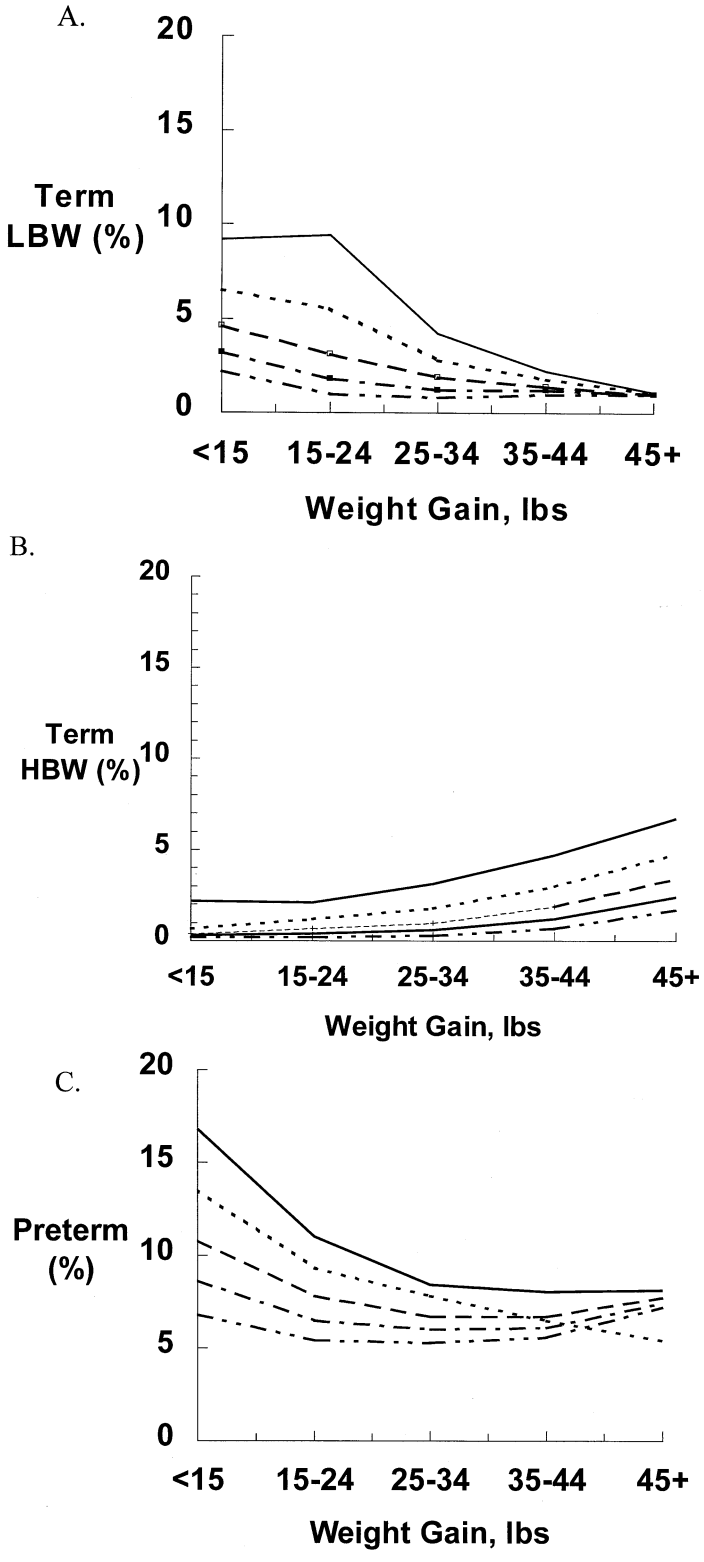

Figure 2. The rate of (A) term low-birthweight (birthweight <2500 g), (B) term high birthweight (birthweight >4,000 g) and (C) preterm delivery (delivery, 26–37 weeks gestation), by prepregnancy BMI and weight gain during pregnancy. Data from the Pregnancy Nutrition Surveillance System were used for all figures. Total weight gain was estimated from the weight gain rate per week times 40 weeks of gestation. Pre-pregnancy BMI of <15 is represented by ——, BMI 15–24 by ▪▪▪▪▪▪, BMI 25–29 by —— —— BMI 30–34 by —— ▪ ——BMI 35+ by —— ▪▪.

women to deliver an infant with a major congenital malformation.[15,74] It is also possible that folic acid may lose its protective effect against neural tube defects among overweight and obese mothers.[73]

Perinatal Mortality. Data from the Swedish Medical Birth Register[14] and the Collaborative Perinatal Study[15] indicates that infants born to women with a prepregnancy BMI of ≥30 were 2.0–4.3 times as likely to die in the perinatal period as were infants born to women with a prepregnancy BMI of <20. Versus the same comparison group, infants born to women with a prepregnancy BMI of 25–30 were 50% more likely to die in the perinatal period.[14,15] The association between prepregnancy obesity and perinatal mortality was robust in that it occurred after accounting for parity, diabetes status, presence of hypertensive disorders, smoking, age, or socioeconomic status. Among women with low weight gain during pregnancy, however, Naeye found that thin women (BMI < 20) were at greater risk of perinatal mortality than average-weight, overweight, or obese women.[15] Naeye's results suggest that the higher risk of perinatal mortality among infants born to obese women does not hold when women gain <0.8 kg/mo, but this finding requires confirmation.

Obesity Prevention Among Women Who Are Not Pregnant

Because obesity and central adiposity are independently related to the onset of chronic disease and because obesity is related to adverse reproductive outcomes, obstetricians/gynecologists should include obesity screening and prevention as part of routine clinic visits for nonpregnant women. Strategies to prevent obesity include weight maintenance for all, weight loss for obese, and physical activity for all.

Table 5. Relative Risks of Congenital Anomalies with Increased Body Mass Index (BMI) from Cohort and Case-Control Studies

Country	*N*	Study Characteristics	Type of Congenital Anomalies	Cases *(n)*	BMI Referent Group	BMI Risk Group	Relative Risk	Control for Associated Factors
United States[15]	56,857 (598 twins)	Cohort study, births in 12 medical school-affiliated hospitals in different regions between 1959–1966	Major congenital malformations	2,504	<20	20–24 25–30 >30	1.1 1.4 1.4	None, comment that results not changed by advanced maternal age, cigarette smoking, or twins.
Sweden[74]	665,706	Cohort study, women followed prenatally and infants registered at birth, 1983–1993	Anencephaly, spina bifida, and encephalocele	393	≤26 ≤29	>26 >29	1.4 1.3	Adjusted for year of birth, maternal age, parity, education level, smoking, and immigrant status.
Sweden[74]	665,706	Cohort study, women followed prenatally and infants registered by birth, 1983–1993	Spina bifida alone	313	≤26 ≤29	>26 >29	1.5 1.5	Adjusted for year of birth, maternal age, parity, education level, smoking, and immigrant status.
United States[70]	534 controls	Case-control study, women and infants diagnosed prenatally and infants born 1985–1987	Neural tube defects	499	19–27	<16 16–17 18 28–30 31–37 ≥38	0.8 1.2 0.9 0.9 1.8 3.0	Controls matched by diagnostic procedure, race or ethnic group, gestational age at diagnosis, date of diagnosis, and geographic area.
United States[70]	534 controls	Case-control study, women and infants diagnosed prenatally and infants born 1985–1987	Congenital malformations other than neural tube defects (excluding cleft lip and palate)	337	19–27	<16 16–17 18 28–30 31–37 ≥38	0 1.6 0.9 0.8 1.7 1.6	Controls matched by diagnostic procedure, race or ethnic group, gestational age at diagnosis, date of diagnosis, and geographic area.
United States[71]	2,755 controls	Case-control study, live or stillborn infants identified through a population-based registry	Anencephaly or spina bifida	307	>19.8–26	<16.5 16.5–19.8 >26–29 >29	0.6 1.0 1.6 1.9	Controls frequency matched by race, period of birth, and hospital of birth; mothers with self-reported diabetes were excluded.
United States[72]	517 controls	Case-control study, singleton liveborn infants with neural tube defects identified through a population-based registry	Anencephaly, spina bifida, cystica, chranioshisis, or iniencephaly	500	19–27	<16 16–17 18 28–30 31–37 ≥38	0 0.7 1.1 1.9 1.5 2.6	Frequency matched by time and hospital of birth; mothers with previous neural tube defects excluded
United States[73]	Controls: 93 nonmalformed, 317 malformed	Case-control study, infants or fetus identified through hospital based surveillance. Infants with known chromosomal anomaly excluded.	Anencephaly, spina bifida, or encephalocele	88	19–23.9	<19.0 24–27.9 28–31.9 ≥32	1.1 1.7 1.9 2.8	Adjusted for age, education, family income, liveborn/stillborn vs therapeutic abortion, average folate intake, and average energy intake.

BMI = weight in kilograms divided by square of height in meters.

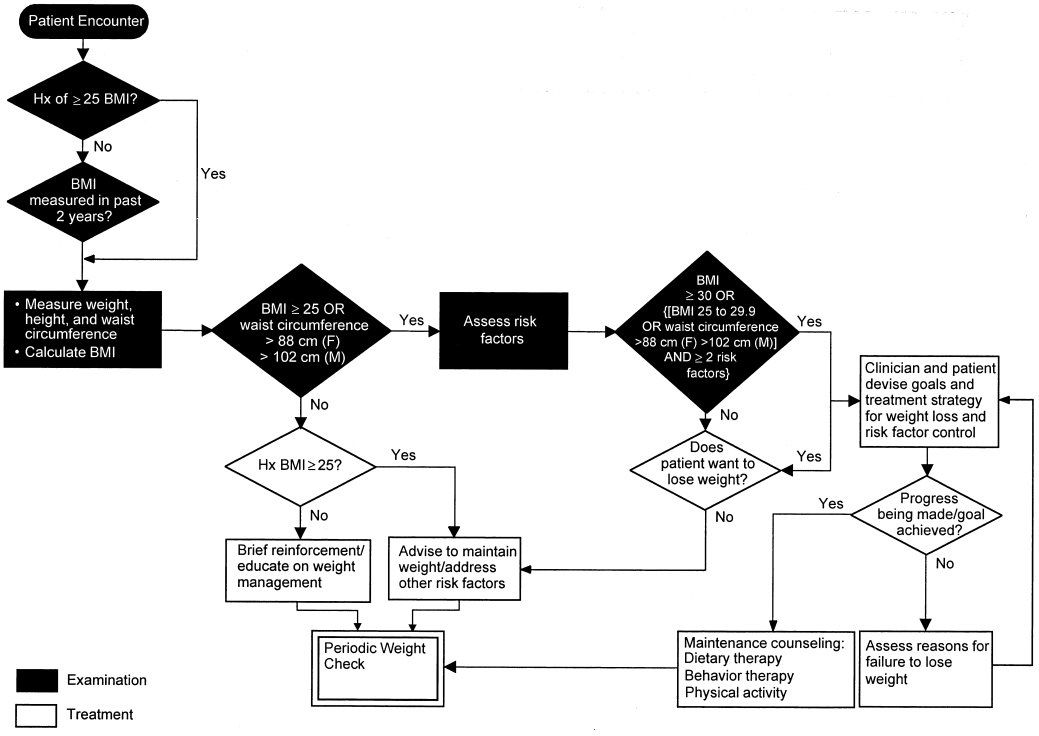

Figure 3. Treatment algorithm from the clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults.[9]

The NIH algorithm, *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults*, is presented in Figure 3; the guidelines may be briefly summarized as follows:[9]

To assess obesity and gauge ongoing disease risk, physicians should regularly measure the patient's weight, height, and waist circumference and calculate her BMI. There are two basic goals for treatment: weight reduction and weight maintenance. Weight reduction is for all patients with a BMI ≥ 30, as well as for those with a BMI 25–29.9 or a waist circumference of >88 cm and two or more risk factors. Among the risk factors discussed in the NIH report are the following: established coronary heart disease, risk factors for coronary heart disease, and other obesity-related disorders.[9] Weight maintenance is for all other persons and is also the first step necessary for obese persons to lose weight.

The decision to lose weight should be made after considering other risk factors (eg, quitting smoking is more important than losing weight) and patient preferences.[9] Willingness to lose weight can be assessed by asking, "Are you concerned about your weight?" Patients who are not concerned about their weight can be asked, "What would make you decide to do something about your weight?", and appropriate counseling can be given that fits their response.

A goal of losing 10% of baseline weight over 6 months is recommended for weight reduction. Weight maintenance is defined as a weight regain of <3 kg (6.6 lbs) over 2 years and a sustained reduction in waist circumference of at least 4 cm. The recommended strategy to reduce and maintain weight has two parts: restricting calories and increasing physical activity. Yet in 1996, only about 20% of women trying to lose weight and 10% of women trying to maintain weight reported using this combination of strategies.[22]

For dietary restriction, the goal is a deficit of 500–1000 kcal/d, which should achieve a weight loss of 1–2 lb per week. The current recommended therapy for weight loss is a low-calorie diet of 800–1,500 kcal/d. For losing weight, reducing fat intake is not effective unless calories are also reduced. Yet in 1996, 43% of women trying to lose weight reported consuming less fat, but only a little more than one fourth reported eating fewer calories (with or without less fat).[22]

For physical activity, the goal is 30–45 minutes of moderate activity on ≥5 days per week. With this regimen, 100–200 calories per day can be expended depending on body weight and intensity of activity. Physical activity alone generally produces an average 2–3% total decrease in body weight, and thus it must be combined with caloric restriction if goals for weight loss are to be met. Although those who restrict

Table 6. Behavioral Therapy Strategies Used in Weight Loss and Weight Maintenance Programs

Strategy	Examples
Self-monitoring	Recording amounts and types of food eaten and frequency, intensity, and type of physical activity
Stress management	Coping strategies, meditation, and relaxation techniques
Stimulus control	Keeping high-calorie foods out of the house; limiting times and places of eating
Problem solving	Identifying weight-related problems, generating or brainstorming solutions and choosing one, planning and implementing healthier alternatives, and evaluating the outcome of possible changes in behavior
Contingency management	Verbal or tangible rewards for specific actions such as increasing time spent walking or reducing consumption of specific foods. Rewards can come from the professional team or the patient and can be monetary or social
Cognitive restructuring	Rationale responses designed to replace negative thoughts. For example, the thought, "I blew my diet this morning by eating that doughnut; I may as well eat what I like for the rest of the day," could be replaced with "Well, I ate the doughnut this morning, but I can still eat in a healthy manner at lunch and dinner"
Social support	Family, friends, or colleagues can assist a patient in maintaining motivation and providing positive reinforcement; some patients may benefit by entering a weight reduction support group

Adapted from Wadden.[80]

calories and increase physical activity are more successful at long-term weight loss maintenance,[75–78] these behaviors are difficult to maintain.

Behavioral therapy is useful for helping obese patients to alter their eating and activity habits and to maintain healthy habits over time. On the basis of the concept that many behaviors, including maladaptive ones, are learned, behavior therapy may be used to control either the environmental antecedents or the consequences of the behavior. The antecedents are the conditions under which the behavior occurs. The consequences are the positive and negative reinforcers of the behavior.[79] Behavioral strategies are listed in Table 6.[80] Self-monitoring, for example, allows patients to become aware of the conditions under which overeating occurs and helps them to control such situations. Patients who self-monitor are more likely to sustain long-term weight losses.[81,82]

Pharmacologic agents may be indicated for patients who do not lose 10% of their weight in 6 months after following a low-calorie diet and engaging in physical activity. Drugs are indicated only for patients with an initial BMI of $\geq$30, or $\geq$27 if other risk factors are present (eg, hypertension, diabetes, dyslipidemia), as an adjunct to calorie reduction and participating in regular physical activity. Continual assessment of safety and efficacy by the clinician is necessary. The popular drugs dexfenfluramine and fenfluramine were removed from the U.S. markets in 1997 because of their association with cardiac valvular dysfunction.[83]

Recently published randomized controlled trials evaluated the safety and efficacy of the relatively new weight loss drugs sibutramine and orlistat.[84–86] Sibutramine inhibits the uptake of both serotonin and norepinephrine and decreases food intake through β_1 and $5HT_{2A/2C}$ receptor agonist activity.[87] In some people, the drug may mildly increase heart rate and blood pressure or prevent their expected decline with weight loss. The clinical importance of these adverse effects is unclear. Orlistat inhibits activity of pancreatic and gastic lipases and blocks gastrointestinal uptake of approximately 30% of ingested fat.[88] Gastrointestinal symptoms are the most commonly observed adverse events. In randomized controlled trials, 57% of patients treated with Orlistat lost 5% of baseline body weight, versus 31% of patients treated with placebo.[88] The safety of the drug beyond 2 years of treatment has not been determined.[89]

Aside from screening for and treating obesity, U.S. guidelines emphasize moderate physical activity (eg, 30 minutes of brisk walking) on most or preferably all days of the week or more vigorous activity three times per week.[8] Physical activity need not be vigorous or continuous to produce health benefits. Multiple short bouts of activity of 10 minutes are as effective as long bouts (40 minutes) at reducing weight and improving cardiorespiratory fitness.[90] Increasing lifestyle activity (eg, walking instead of driving, taking stairs instead of the elevator) provides results comparable to those from increasing structured exercise in terms of losing body fat and improving cardiovascular risk factors.[91–93] Improvements in cardiorespiratory fitness attenuate age-related weight gain in healthy women.[94] Physician-based counseling can promote adoption of these guidelines.[5,95]

Obesity Prevention Among Pregnant Women

Four percent of all women and 7% of women with a BMI of >29 try to lose weight during pregnancy,[96] but such attempts are not recommended. Instead, prepregnancy BMI should be calculated at the first pregnancy visit and weight gain monitored at each prenatal care visit.[9,13,97] Because excess weight gain during pregnancy may be related to

postpartum weight retention,[98–100] physicians should reinforce the need for proper weight gain during pregnancy. The American College of Obstetricians and Gynecologists[97] recommends that women with low BMI (<19.8) gain 28–40 lbs; women with average BMI (19.8 to 26.0), 25–35 lbs; women with high BMI (>26.0–29.0), 15–25 lbs; and women with very high BMI (>29.0), at least 15 lbs.

Approximately one third of all women and two thirds of overweight women (BMI, 26 to <29) gain more than the recommended amount of weight during pregnancy.[101,102] In addition, one third of obese women (BMI > 29) gain more than 25 lbs.[102] Epidemiologic studies suggest that pregnant women are more likely to gain within the recommended weight gain guidelines if their physicians instruct them to do so.[101–103] However, in two studies, one quarter of pregnant women reported that their health care providers gave them no advice about weight gain.[102,103] Among those who received advice, in one study, 22% were reportedly advised to gain more than the recommended amount.[102] Most of these women (80.2%) were overweight or obese (BMI > 26).

Most women should be encouraged to remain physically active during pregnancy.[97] Current evidence suggests that participation in safe and appropriate exercise of moderate to high intensity by healthy and well-conditioned women through pregnancy does not compromise fetal growth and development.[104,105] In addition, for obese women, exercise may help reduce the risk of gestational diabetes mellitus.[106] The effects of physical activity in the prenatal period on postpartum weight retention require further study.[107,108]

Another approach to reducing retention of pregnancy-related weight gain is obesity treatment during the postpartum period. In a recent study, treatment by mail correspondence was used for women who had retained at least 15 pounds more than their prepregnancy weight 3–12 months postpartum. Thirty-three percent of the women in the treatment group returned to their prepregnancy weight, compared with only 11% in the no-treatment control group.[109] Gradual weight loss (≤2 kg/mo) seems to have no adverse effect on milk volume or composition, provided the mother is not undernourished and is breast-feeding her infant on demand.[110] A recent study suggests that weight loss of about 0.5 kg/wk between 4 and 14 weeks postpartum in overweight women does not affect the growth of exclusively breast-fed infants.[111] Randomized controlled studies of the effects of physician counseling on achieving appropriate weight gain during pregnancy and weight losses after pregnancy have not been pulished.

Environmental Changes

Despite good intentions on the part of the physician and their patients, the environment may work against them.[112] Over the last 20 years, the availability and promotion of energy dense foods has increased.[113] During the same period, the availability of labor-saving devices has decreased the daily need for physical activity. Lack of sidewalks and public transportation may decrease the desire to walk, and community safety may also be an issue.[114] Finally, sedentary activities, such as watching television, have become more prevalent.[113] The physician needs to be aware of these issues, but at the same time, more research is needed to examine which changes to the environment promote healthy diet and physical activity behaviors.[115]

Conclusions

Obesity-associated diseases represent a major source of morbidity and mortality in the United States. As we have indicated, nearly half of women in the United States are overweight or obese (BMI > 25), and >20% of women of childbearing age are obese (BMI > 30, or approximately 30 lbs overweight). Furthermore, the prevalence of obesity doubled between 1980 and 1994 in some of the childbearing-age groups. Obese women of childbearing age are at risk not only for the comorbidities that apply equally to men, such as hyperlipidemia, hypertension, and diabetes, but also for complications related to reproduction, such as infertility, as well as for maternal and fetal complications during pregnancy, delivery, and in the postpartum period. Diabetes is increasing in the United States, and cardiovascular disease, for which obesity is a major risk factor, remains the leading cause of death for women in the United States.

Many obstetrician/gynecologists, nurse practitioners, and midwives provide primary care for women, but three major barriers may make their care for overweight and obese women less than it should be: knowledge, attitudes, and time. The recommendations in the 1998 NIH Guidelines, available on the World Wide Web at www.nhlbi.nih.gov, may be of great value in overcoming the knowledge barrier.

The evidence reviewed to develop the guidelines demonstrates conclusively that modest weight loss induced by diet, exercise, behavior modification, or pharmacotherapy significantly reduces obesity-associated comorbidities.[9] The recommendations are clear, but how they are best implemented in primary care settings remains less certain, and thus there are important opportunities for office-based research.

Attitudes of primary care providers are equally important. Little is known about the attitudes of obstetricians and gynecologists, but surveys of other physicians suggest at least two obstacles to implementing recommendations: 1) a perception that the recommendations are not important and 2) pessimism about the ability to change patients' behavior to meet recommendations.[116–118] The high prevalence and substantial associated morbidity of obesity suggest that counseling about physical activity and diet should receive as much attention as efforts to prevent pregnancy, human immunodeficiency virus, or osteoporosis or to detect breast cancer early. The pessimism about changing behavior through counseling may be driven in part by inadequate knowledge or use of ineffective counseling tools.

Time is a perennial problem, but office-based computer assessments may reduce the time needed to assess or counsel patients. Medical care providers remain the most credible source of advice for patients, yet counseling about diet and physical activity may be provided by obstetricians and gynecologists at <20% of office visits.[16] Physicians need to document counseling during patient visits, and insurance companies need to reimburse physicians for this type of care. Still, both of these practices may be difficult in the current health care environment, and thus, other approaches may be needed. The most successful interventions have included fairly frequent contact with persons in the initial stages of weight loss, with less frequent contact over time. With chronic diseases such as diabetes and hypertension, more frequent and intensive attention from interested and knowledgeable nurse case managers working with physicians, dieticians, and other health professionals and following of a specified protocol has proven more successful than routine care.[119] The same approach may be useful for overweight and obese women of childbearing age. Frequent contact with women of childbearing age provides obstetricians and gynecologists an opportunity to prevent and treat obesity successfully.

References

1. Flegal KM, Carroll MD, Kuczmarski RJ, et al. Overweight and obesity in the United States: prevalence and trends, 1960–1994. Int J Obes Relat Metab Disord 1998;22: 39–47.
2. U.S. Preventive Services Task Force. Guide to clinical preventive services. 2nd ed. Alexandria, VA: International Medical Publishing, 1996:611–42.
3. Galuska DA, Will JC, Serdula MK, et al. Are health care professionals advising obese patients to lose weight? JAMA 1999;282:1576–8.
4. Nawaz H, Adams ML, Katz DL. Weight loss counseling by health care providers. Am J Public Health 1999;89:764–7.
5. Simons-Morton DG, Calfas KJ, Oldenburg B, Burton NW. Effects of interventions in health care settings on physical activity or cardiorespiratory fitness. Am J Prev Med 1998;15:413–32.
6. Wee CC, McCarthy EP, Davis RB, et al. Physician counseling about exercise. JAMA 1999;282:1583–8.
7. Centers for Disease Control and Prevention. Missed opportunities in preventive counseling for cardiovascular disease—United States, 1995. MMWR 1998;47:91–5.
8. U.S. Department of Health and Human Services. Physical activity and health: a report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, 1996.
9. National Institutes of Health, National Heart, Lung, and Blood Institute. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity, the evidence report. Rockville, MD: U.S. Department of Health and Human Services, 1998.
10. Bouchard C, Perusse L. Genetic aspects of obesity. Ann NY Acad Sci 1993;699:26–35.
11. Stevens J, Cai J, Pamuk ER, et al. The effect of age on the association between body mass index and mortality. N Engl J Med 1998;338: 1–7.
12. Troiano RP, Frongillo EA Jr, Sobal J, et al. The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. Int J Obes Relat Metab Disord 1996; 20:63–75.
13. National Academy of Sciences, Institute of Medicine, Food and Nutrition Board, Committee on Nutritional Status During Pregnancy and Lactation, Subcommittee on Nutritional Status and Weight Gain During Pregnancy. Nutrition during pregnancy. Part 1, weight gain. Washington, DC: National Academy Press, 1990.
14. Cnattingius S, Berstrom R, Lipworth L, et al. Pre-pregnancy weight and the risk of adverse pregnancy outcomes. N Engl J Med 1998;338:147–52.
15. Naeye RL. Maternal body weight and pregnancy outcome. Am J Clin Nutr 1990;52:273–9.
16. Centers for Disease Control and Prevention. Pregnancy nutrition surveillance, 1996 full report. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, 1998.
17. Williamson DF, Kahn JS, Remington PL, et al. The 10-year incidence of overweight and major weight gain in US adults. Arch Intern Med 1990;150:666–72.
18. Williamson DF, Madans J, Pamuk E, et al. A prospective study of childbearing and 10-year weight gain in US white women 25–45 years of age. Int J Obes Relat Metab Disord 1994;18:561–9.
19. Smith DE, Lewis CE, Caveny JL, et al. Longitudinal changes in adiposity associated with pregnancy. The Cardia Study. Coronary Artery Risk Development in Young Adults Study. JAMA 1994;271: 1747–51.
20. Pi-Sunyer FX. Medical hazards of obesity. Ann Intern Med 1993;119: 655–60.
21. Durazo-Arvizu R, Cooper RS, Luke A, et al. Relative weight and mortality in US blacks and whites: findings from representative national population samples. Ann Epidemiol 1997;7:383–95.
22. Serdula MK, Mokdad AH, Williamson DF, et al. Prevalence of attempting weight loss and strate-

gies for controlling weight. JAMA 1999;282:1353–8.
23. 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;141:1128–41.
24. French SA, Folsom AR, Jeffery RW, et al. Prospective study of intentionality of weight loss and mortality in older women: the Iowa women's health study. Am J Epidemiol 1999;149:504–14.
25. Cooper RS, Ford E. Comparability of risk factors for coronary heart disease among blacks and whites in the NHANES I Epidemiologic Follow-Up Study. Ann Epidemiol 1992;2:637–45.
26. Folsom AR, Stevens J, Schreiner PJ, et al. Body mass index, waist/hip ratio, and coronary heart disease incidence in African Americans and whites. Atherosclerosis Risk in Communities Study Investigators. Am J Epidemiol 1998;48:1187–94.
27. Ford ES. Body mass index and colon cancer in a national sample of adult US men and women. Am J Epidemiol 1999;150:390–8.
28. Must A, Spadano J, Coakley EH, et al. The disease burden associated with overweight and obesity. JAMA 1999;282:1523–9.
29. Huang Z, Hankinson SE, Colditz GA, et al. Dual effects of weight and weight gain on breast cancer risk. JAMA 1997;278:1407–11.
30. Manson JE, Colditz GA, Stampfer MJ, et al. A prospective study of obesity and risk of coronary heart disease in women. N Engl J Med 1990;322:882–9.
31. Rexrode KM, Hennekens CH, Willett WC, et al. A prospective study of body mass index, weight change, and risk of stroke in women. JAMA 1997;277:1539–45.
32. Colditz GA, Willett WC, Stampfer MJ, et al. Weight as a risk factor for clinical diabetes in women. Am J Epidemiol 1990;132:501–13.
33. Goldhaber SZ, Grodstein F, Stampfer MJ, et al. A prospective study of body mass index, weight change, and risk of stroke in women. JAMA 1997;277:1539–45.
34. Maclure KM, Hayes KC, Colditz GA, et al. Weight, diet, and the risk of symptomatic gallstones in middle-aged women. N Engl J Med 1989;321:563–9.
35. Colditz GA, Willett WC, Rotnitzky A, et al. Weight gain as a risk factor for clinical diabetes mellitus in women. Ann Intern Med 1995;122:481–6.
36. Giovannucci E, Colditz GA, Stampfer MJ, et al. Physical activity, obesity, and risk of colorectal adenoma in women (United States). Cancer Causes Control 1996;7:253–63.
37. Felson DT, Zhang Y, Anthony JM, Naimark A, et al. Weight loss reduces the risk for symptomatic knee osteoarthritis in women. Ann Intern Med 1992;116:535–9.
38. Spector TD, Hart DJ, Doyle DV. Incidence and progression of osteoarthritis in women with unilateral knee disease in the general population: the effect of obesity. Ann Rheum Dis 1994;53:565–8.
39. Perry AC, Applegate EB, Allison ML, et al. Relationship between anthropometric measures of fat distribution and cardiovascular risk factors in overweight pre- and post-menopausal women. Am J Clin Nutr 1997;66:829–36.
40. Rexrode KM, Carey VJ, Hennekens CH, et al. Abdominal obesity and coronary heart disease in women. JAMA 1998;280:1843–8.
41. Carey VJ, Walters EE, Colditz GA, et al. Body fat distribution and risk of non-insulin dependent diabetes mellitus in women. The Nurse's Health Study. Am J Epidemiol 1997;145:614–9.
42. Han TS, van Leer EM, Seidell JC, et al. Waist circumference as a screening tool for cardiovascular risk factors: evaluation of receiver operating characteristics (ROC). Obes Res 1996;4:533–47.
43. Rogers J, Mitchell GW. The relation of obesity to menstrual disturbances. N Engl J Med 1952;247:53–5.
44. Zaadstra BM, Seidell JC, Van Noord PAH, et al. Fat and female fecundity: prospective study of effect of body fat distribution on conception rates. BMJ 1993;306:484–7.
45. Balen AH, Conway GS, Kaltsas G, et al. Polycystic ovary syndrome: the spectrum of the disorder in 1741 patients. Hum Reprod 1995;10:2107–11.
46. Rich-Edwards JW, Goldman MB, Willett WC, et al. Adolescent body mass index and infertility caused by ovulatory disorder. Am J Obstet Gynecol 1994;171:171–7.
47. Conway GS, Honour JW, Jacobs HS. Heterogeneity of the polycystic ovary syndrome: clinical, endocrine and ultrasound features in 556 patients. Clin Endocrinol 1989;30:459–70.
48. Homburg R. Polycystic ovary syndrome—from gynaecological curiosity to multisystem endocrinopathy. Hum Reprod 1996;11:29–39.
49. McKenna TJ. Pathogenesis and treatment of polycystic ovary syndrome. N Engl J Med 1988;318:558–62.
50. Franks S. Polycystic ovary syndrome. N Engl J Med 1995;333:853–61.
51. Hollmann M, Runnebaum B, Gerhard I. Effects of weight loss on the hormonal profile in obese infertile women. Hum Reprod 1996;11:1884–91.
52. Clark AM, Ledger W, Galletly C, et al. Weight loss results in significant improvement in pregnancy and ovulation rates in anovulatory obese women. Hum Reprod 1995;10:2705–12.
53. Velazquez E, Acosta A, Mendoza SG. Menstrual cyclicity after metformin therapy in polycystic ovary syndrome. Obstet Gynecol 1997;90:392–5.
54. Shepard MK, Balmaceda JP, Leija CG. Relationship of weight to successful induction of ovulation with clomiphene citrate. Fertil Steril 1979;32:641–5.
55. Hamilton-Fairley D, Kiddy D, Watson H, et al. Association of moderate obesity with a poor pregnancy outcome in women with polycystic ovary syndrome treated with low dose gonadotrophin. Br J Obstet Gynaecol 1992;99:128–31.
56. Solomon CG, Willett WC, Carey VJ, et al. A prospective study of pregravid determinants of gestational diabetes mellitus. JAMA 1997;278:1078–83.
57. Thadhani R, Stampfer MH, Hunter DJ, et al. High body mass index and hypercholesterolemia: risk of hypertensive disorders of pregnancy. Obstet Gynecol 1999;94:543–50.
58. Harris SB, Caulfield LE, Sugamori ME, et al. The epidemiology of diabetes in pregnant Native Canadians. Diabetes Care 1997;20:1422–5.
59. Abrams B, Parker J. Overweight and pregnancy complications. Int J Obes 1988;12:293–303.
60. Edwards LE, Hellerstedt W, Alton IR, et al. Pregnancy complications

and birth outcomes in obese and normal weight women: effects of gestational weight change. Obstet Gynecol 1996;87:389–94.
61. Eskenazi B, Fenster L, Sidney S. A multivariate analysis of risk factors for preeclampsia. JAMA 1991; 266:237–41.
62. Stone JL, Lockwood CJ, Berkowitz GS, et al. Risk factors for severe preeclampsia. Obstet Gynecol 1994;83:357–61.
63. Sibai BM, Gordon T, Thom E, et al. Risk factors for pre-eclampsia in healthy nulliparous women: a prospective multicenter study. Am J Obstet Gynecol 1995;172:642–8.
64. Bianco AT, Smilen SW, Davis Y, et al. Pregnancy outcome and weight gain recommendations for the morbidly obese woman. Obstet Gynecol 1998;91:97–102.
65. Crane SS, Wojtowycz, Dye TD, Aubry RH, et al. Association between prepregnancy obesity and the risk of cesarean delivery. Obstet Gynecol 1997;89:213–6.
66. Schieve LA, Cogswell ME, Scanlon KS. An empiric evaluation of the Institute of Medicine's pregnancy weight gain guidelines by race. Obstet Gynecol 1998;91: 878–84.
67. Schieve LA, Cogswell ME, Scanlon KS. Maternal weight gain and preterm delivery: differential effects by body mass index. Epidemiology 1999;10:141–7.
68. Caulfield LE, Stoltzfus RJ, Witter FR. Implications of the Institute of Medicine weight gain recommendations for preventing adverse pregnancy outcomes in black and white women. Am J Public Health 1998;88:1168–74.
69. Parker JD, Abrams B. Prenatal weight gain advice: an examination of the recent prenatal weight gain recommendations of the Institute of Medicine. Obstet Gynecol 1992;79:664–9.
70. Waller DK, Mills JL, Simpson JL, et al. Are obese women at higher risk for producing malformed offspring? Am J Obstet Gynecol 1994; 170:541–8.
71. Watkins ML, Scanlon KS, Mulinare J, et al. Is maternal obesity a risk factor for anencephaly and spina bifida? Epidemiology 1996;7:506–12.
72. Shaw GM, Velie EM, Schaffer D. Risk of neural tube defect-affected pregnancies among obese women. JAMA 1996;275:1093–6.
73. Werler MM, Louik C, Shapiro S, et al. Prepregnant weight in relation to risk of neural tube defects. JAMA 1996;275:1089–92.
74. Kallen K. Maternal smoking, body mass index, and neural tube defects. Am J Epidemiol 1998;147: 1103–11.
75. Perri MG, McAdoo WG, McAllister DA, et al. Enhancing the efficacy of behavior therapy for obesity: effects of aerobic exercise and a multicomponent maintenance program. J Consult Clin Psychol 1986;54:670–5.
76. van Dale D, Saris WH, ten Hoor F. Weight maintenance and resting metabolic rate 18–40 months after a diet/exercise treatment. Int J Obes 1990;14:347–59.
77. Klem ML, Wing RR, McGuire MT, et al. A descriptive study of individuals successful at long term maintenance of substantial weight loss. Am J Clin Nutr 1997;66:239–46.
78. Kayman S, Bruvold W, Stern JS. Maintenance and relapse after weight loss in women: behavioral aspects. Am J Clin Nutr 1990;52: 800–7.
79. Masters JC, Burish TG, Hollon SD, et al. The nature of behavioral therapy. In: Masters JC, ed. Behavior therapy: techniques and empirical finding. San Diego, CA: Harcourt Brace Jovanovich, 1987:1–35.
80. Wadden TA. The treatment of obesity. In: Stunkard AJ, Wadden TA, eds. Obesity: theory and therapy. New York: Raven Press, 1993:197–217.
81. Holden JH, Darga LL, Olson SM, et al. Long-term follow-up of patients attending a combination very-low calorie diet and behaviour therapy weight loss programme. Int J Obes Relat Metab Disord 1992;16:605–13.
82. Boutelle KN, Kirschenbaum DS. Further support for consistent self-monitoring as a vital component of successful weight control. Obes Res 1998;6:219–24.
83. Centers for Disease Control and Prevention. Cardiac valvulopathy associated with exposure to fenfluramine and dexfenfluramine: US Department of Health and Human Services Interim Public Health Recommendations, November 1997. MMWR 1997;46: 1061–5.
84. Bray GA, Blackburn GL, Ferguson JM, et al. Sibutramine produces dose-related weight loss. Obes Res 1999;7:189–98.
85. Davidson MH, Hauptman J, Digirolamo M, et al. Weight control and risk factor reduction in obese subjects treated for 2 years with Orlistat: a randomized control trial. JAMA 1999;281:235–42.
86. Hollander PA, Elbein SC, Hirsch IB, et al. Role of Orlistat in the treatment of obese patients with type 2 diabetes: a 1-year randomized double-blind study. Diabetes Care 1998;21:1288–94.
87. Lean ME. Sibutramine—a review of clinical efficacy. Int J Obes Relat Metab Disord 1997;21:S30–6.
88. Drent ML, van der Veen EA. First clinical studies with orlistat: a short review. Obes Res 1995;3: 623S–5S.
89. Nightingale SL. From the Food and Drug Administration. JAMA 1999; 281:19.
90. Jakicic JM, Winters C, Lang W, et al. Effects of intermittent exercise and use of home exercise equipment on adherence, weight loss, and fitness in overweight women. JAMA 1999;282:1554–60.
91. Dunn AL, Marcus BH, Kampert JB, et al. Comparison of lifestyle and structured interventions to increase physical activity and cardiorespiratory fitness. JAMA 1999; 281:327–34.
92. Dunn AL, Andersen RE, Jakicic JM. Lifestyle physical activity interventions: history, short- and long-term effects, and recommendations. Am J Prev Med 1998;15: 398–412.
93. Manson JE, Hu FB, Rich-Edwards JW, et al. A study of walking as compared with vigorous exercise in the prevention of coronary heart disease in women. N Engl J Med 1999;341:650–8.
94. DiPietro L, Kohl HW III, Barlow CE, et al. 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.
95. Calfas KJ, Long BJ, Sallis JF, et al. A controlled trial of physician counseling to promote adoption of physical activity. Prev Med 1996; 25:225–33.
96. Cogswell ME, Serdula MK, Mokdad AH, et al. Attempted weight loss during pregnancy. Int J Obes Relat Metab Disord 1996;20:373–5.

97. American Academy of Pediatrics and American College of Obstetricians and Gynecologists. Guidelines for perinatal care. 4th ed. Elk Grove, IL, and Washington, DC: American Academy of Pediatrics and American College of Obstetricians and Gynecologists, 1997.
98. Parker JD, Abrams B. Differences in postpartum weight retention between black and white mothers. Obstet Gynecol 1993;81:768–74.
99. Keppel KG, Taffel SM. Pregnancy related weight gain and retention: implications of the 1990 Institute of Medicine guidelines. Am J Public Health 1993;83:1100–3.
100. Scholl TO, Hediger ML, Schall JI, et al. Gestational weight gain, pregnancy outcome, and postpartum weight retention. Obstet Gynecol 1995;86:423–7.
101. Taffel SM, Keppel KG, Jones GK. Medical advice on maternal weight gain and actual weight gain: results from the 1988 Maternal and Infant Health Survey. Ann NY Acad Sci 1993;678:293–305.
102. Cogswell ME, Scanlon KS, Fein SB, et al. Medically advised, mother's personal target, and actual weight gain during pregnancy. Obstet Gynecol 1999;94:616–22.
103. Taffel SM, Keppel KG. Advice about weight gain during pregnancy and actual weight gain. Am J Public Health 1986;76: 1396–9.
104. Pivarnik JM. Potential effects of maternal activity on birth weight: brief review. Med Sci Sports Exer 1998;30:400–6.
105. Kardel KR, Kase T. Training in pregnant women: effects on fetal development and birth. Am J Obstet Gynecol 1998;178:280–6.
106. Dye TD, Knox KL, Artal R, et al. Physical activity, obesity, and diabetes in pregnancy. Am J Epidemiol 1997;146:961–5.
107. 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;86:834–8.
108. Ohlin A, Rossner S. Trends in eating patterns, physical activity, and sociodemographic factors in relation to postpartum body weight development. Br J Nutr 1994;71:457–70.
109. Leermakers EA, Anglin K, Wing RR. Reducing postpartum weight retention through a correspondence intervention. Int J Obes Relat Metab Disord 1998;22:1103–9.
110. Dewey KG. Effects of maternal caloric restriction and exercise during lactation. J Nutr 1998;128: 386S–89S.
111. Lovelady CA, Garner KE, Moreno KL, et al. The effect of weight loss in overweight, lactating women on the growth of their infants. N Engl J Med 2000;342:449–52.
112. Hill JO, Peters JC. Environmental contributions to the obesity epidemic. Science 1998;280:1371–4.
113. Jeffery RW, French SA. Epidemic obesity in the United States: are fast foods and television viewing contributing? Am J Public Health 1988;88:277–80.
114. Centers for Disease Control and Prevention. Neighborhood safety and the prevalence of physical inactivity—selected states, 1996. MMWR 1999;48:143–6.
115. Sallis JF, Bauman A, Pratt M. Environmental and policy interventions to promote physical activity. Am J Prev Med 1998;15:379–97.
116. Orleans CT, George LK, Houpt JL, et al. Health promotion in primary care: a survey of U.S. family practitioners. Prev Med 1985;14:636–47.
117. Lewis CE, Clancy C, Leake B, et al. The counseling practices of internists. Ann Intern Med 1991;114: 54–8.
118. Weschler H, Levine S, Idelson RK, et al. The physician's role in health promotion—a survey of primary care practitioners. N Engl J Med 1983;308:97–100.
119. Aubert RE, Herman WH, Waters J, et al. Nurse case management to improve glycemic control in diabetic patients in a health maintenance organization. A randomized, controlled trial. Ann Intern Med 1998;129:605–12.

Address correspondence and reprint requests to Mary E. Cogswell, Centers for Disease Control and Prevention, Mail Stop K-25, Division of Nutrition and Physical Activity, 4770 Buford Highway, Atlanta, GA 30341-3724.