Annals of Internal Medicine | ARTICLE

The Natural History of the Development of Obesity in a Cohort of Young U.S. Adults between 1981 and 1998

Kathleen M. McTigue, MD, MS, MPH; Joanne M. Garrett, PhD; and Barry M. Popkin, PhD

Background: **Understanding the natural history of obesity in a population may be a critical step toward developing effective interventions.**

Objective: **To assess the development of body mass and examine the role of race or ethnicity, sex, and birth year in obesity onset in young U.S. adults.**

Design: **Prospective cohort study.**

Setting: **The National Longitudinal Survey of Youth 1979, a national sample with oversampling of minority ethnic groups.**

Participants: **9179 persons.**

Measurements: **Body mass index (BMI) calculated from 12 self-reported height and weight samples recorded between 1981 and 1998. Logistic regression identified predictors of obesity at age 35 to 37 years. Cox proportional hazards models compared the incidence of obesity by ethnicity and birth year.**

Results: **Overall, 26% of men and 28% of women were obese (BMI ≥ 30 kg/m^2) by age 35 to 37 years. Race or ethnicity and baseline BMI were significant predictors of obesity. Obesity onset was 2.1 (95% CI, 1.6 to 2.7) times faster for black women and 1.5 (CI, 1.1 to 2.0) times faster for Hispanic women than for white women. The pattern for men differed: Overall, obesity developed most rapidly in Hispanic men, but relative rates of obesity onset for white men compared with black men varied according to age. The rate of obesity onset increased 26% to 28% over an 8-year span in birth year.**

Conclusions: **Marked ethnic-based differences were found in rates of weight accumulation in young U.S. adults, with later birth cohorts experiencing earlier onset of obesity. To alter the course of obesity in the United States, interventions should target young adults, especially those of minority ethnic groups.**

Ann Intern Med. 2002;136:857-864. www.annals.org
For author affiliations, see end of text.
See editorial comment on pp 923-925.

The proportion of Americans who are overweight or obese is increasing at an alarming rate. From 1960 to 1999, the prevalence of excess weight (body mass index [BMI] ≥ 25 kg/m^2) increased from 44% to 61% of the adult population (persons aged 20 to 74 years) and the prevalence of obesity (BMI ≥ 30 kg/m^2) doubled from 13% to 27% (1, 2). Obesity has been linked to increased mortality and chronic morbidity from such diverse causes as hypertension, diabetes, sleep apnea, certain types of cancer, and depression. In the context of this rising prevalence, obesity is increasingly being recognized as an important medical and public health problem (3–6).

Treatment options are available for obesity. However, they can be expensive, often involve difficult lifestyle modifications, and pose adherence problems for many patients. In addition, weight loss, once achieved, is difficult to sustain. Because the health consequences are often long term, the cost–benefit ratio of treatment may be more favorable if people who have recently become obese are targeted. Similarly, prevention should focus on those at immediate risk for becoming obese or overweight. Such approaches require a clear understanding of who is at highest risk for obesity, when this risk is greatest, and how excess weight develops over time. Although inactivity and high caloric intake are the primary risk factors for obesity, other risk factors have been established. Three of the most influential are age, sex, and race or ethnicity (1, 7–9). To better identify people at risk for obesity and those who might benefit from early intervention, we assessed the relationship between weight development and these influential factors.

Despite the morbidity and mortality burden of obesity, its natural history has not been well characterized. Current knowledge is based largely on cross-sectional data. Although several large studies have examined weight development in adults, they have important limitations. Only a few tracked weight long enough to detect age-based trends. The first National Health and Nutrition Examination Survey (NHANES I) Epidemiologic Follow-up Study examined a representative group of U.S. residents but sampled only two time points, spaced 10 years apart, and had insufficient follow-up to track weight patterns in most minority groups (10). The Coronary Artery Risk Development in Young Adults study followed weight development in a large cohort of young black and white Americans but did not provide data for other populations and was geograph-

Context

Studies of the natural history of obesity have had limitations, such as cross-sectional design, short follow-up, or narrowly defined populations.

Contribution

Using data from a national study of Americans born between 1957 and 1964 and followed for nearly two decades, these investigators found that more than 25% were obese by age 35 years.

Obesity developed most quickly in black women, with moderate rapidity in Hispanic women, and most slowly in white women. Hispanic men developed obesity more quickly than other men.

More recent birth cohorts became obese faster than earlier cohorts.

Implications

Understanding ethnic differences in the age of onset and rate of progression of obesity may help efforts to prevent obesity.

–The Editors

ically restricted (8). Other studies have been performed outside of the United States, but the results may not apply here (10, 11).

We designed our study to address the limitations described earlier. We examined data from the National Longitudinal Survey of Youth 1979 (NLSY79). This large, prospective cohort study allowed longitudinal examination of weight trends over a time frame sufficient to capture the dynamics of such a slowly changing problem and of an age range during which obesity becomes prevalent. Because not all weight gain has been documented to be harmful, our analysis focused on obesity onset (defined as the time at which a person reaches obesity) rather than weight gain. We used the clinical definition of obesity (BMI $\geq$ 30 kg/m^2) as our end point because it reflects a point at which mortality risk increases substantially (12). Understanding how obesity develops over time, who is at greatest risk, and when that risk is highest may be essential in reversing the population trend toward excess weight.

METHODS

Study Group and Obesity Measurement

We analyzed data from the NLSY79, which was initiated by the U.S. Department of Labor, Bureau of Labor Statistics. This survey was designed to gather information about the labor market activities and other life events of young U.S. citizens. It has been used extensively in economic and sociologic analyses, and the sample design is described elsewhere (13–16). The cohort of men and women, born between 1957 and 1964, has been followed for more than two decades. It consists of a nationally representative group, with supplemental samples overrepresenting minority racial and ethnic groups and poor persons. We excluded a sample of military personnel because this population was removed from the study in 1985 and because physical activity requirements for these individuals could lead to atypical weight patterns. We included only persons who reported that the ethnicity with which they most closely identified was Hispanic, black, or white (or European origin). Height and weight data were self-reported; height was reported in 1981 and 1985, and weight was reported 12 times between 1981 and 1998.

Examination of Age-Specific Body Mass Index Patterns

Age-specific BMI was calculated for all participants. Height was assumed to remain constant after age 21 years. Standard clinical definitions were used for overweight (BMI 25 to 29.9 kg/m^2) and obesity (BMI $\geq$ 30 kg/m^2). Trends in age-specific BMI were examined by race or ethnicity and birth year.

Cohort Differences

We examined whether rates of obesity onset differed for people maturing at different times in our sample. Birth cohorts were grouped by 2-year increments. To standardize relative rates to age rather than calendar year, time was expressed as the number of years until a person reached obesity, starting from a standard age. In the cohort analysis, obesity onset was evaluated from age 24 years (the baseline age of the oldest participants). The outcome of interest was obesity, and the time variable was years from age 24 years to obesity. People who were already obese at age 24 years were excluded. Kaplan–Meier plots visually demonstrated differences in rates of obesity onset. Cox proportional hazards modeling estimated the ratio of rates (hazard ratio) between groups. For this method to be correct, the ratio of rates must be constant over time (proportional). To assess this assumption, we used two methods: a statistical test of the

Table. **Sample Characteristics in 1981**

Variable	All Patients	Men	Women
Race or ethnicity, *n (%)*			
Hispanic		863 (19)	905 (19)
Black		1379 (31)	1417 (30)
White		2246 (50)	2369 (51)
Total		4488 (100)	4691 (100)
Mean age ± SD (range), *y**	20.4 ± 2.2 (17–24)		
Mean body mass index ± SD (range), *kg/m²*†	22.7 ± 3.6 (11.3–51.5)		
Mean weight ± SD, *kg*‡	65.6 ± 13.3 (31.7–136.1)		

* 9179 patients.
† 8789 patients.
‡ 8794 patients.

scaled Schoenfeld residuals, with a *P* value greater than 0.05 implying that the rates were proportional and a visual method with parallel lines implying proportional rates. In all our analyses, results of proportional hazards testing were reported whenever the assumption was violated.

Timing of Obesity Onset by Race or Ethnicity and Sex

We evaluated relative rates of obesity onset in 17- and 18-year-old persons of different race or ethnicity who were not obese at the start of the study; different models were used for men and women. Time was expressed as the number of years from age 17 years until reaching obesity. To control for birth cohort differences, models were adjusted for birth year. As before, Kaplan–Meier plots were examined and hazard ratios were estimated by using Cox proportional hazards modeling. To determine relative importance of weight gain during childhood compared with adulthood in terms of the timing of obesity onset, we adjusted the models for baseline BMI at 17 or 18 years of age and examined the effect on hazard ratio estimation.

Prevalence and Incidence of Obesity in Early Adulthood

For this analysis, we defined "early adulthood" as the period from 20 to 22 years of age to 35 to 37 years of age. Persons born between 1959 and 1963 were queried about their weight one to two times during each of these age intervals. We calculated the prevalence and incidence of obesity across the early adulthood range. Logistic regression modeling predicted obesity status at age 35 to 37 years from an individual's race or ethnicity, sex, and BMI at age 20 to 22 years. The model was adjusted in 2-year increments for year of birth. Its predictive ability was quantified by using receiver-operating characteristic (ROC) curves.

This study was evaluated and exempted from review by the Institutional Review Board of the University of North Carolina at Chapel Hill.

Role of the Funding Sources

Primary funding was through the Robert Wood Johnson Clinical Scholars Program, with additional support from the National Institute of Child Health and Human Development. Neither entity had a role in the design, conduct, or reporting of this study.

RESULTS

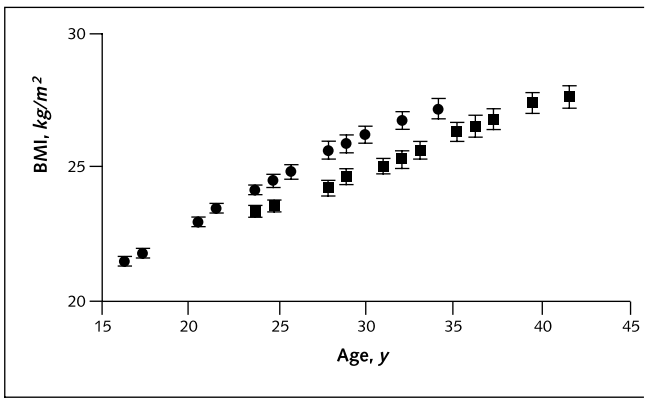
Description of the Study Sample

The total sample included 9179 people: 19% Hispanic, 31% black, and 50% white (**Table**). People who did not report ethnicity ($n = 106$) were not included. Age (sample year minus birth year) ranged from 17 to 24 years in 1981 to 34 to 41 years in 1998, and participants were equally distributed across 8 years of birth. Baseline weight was 31.7 to 136.1 kg, and baseline BMI was 11.3 to 51.5 kg/m^2.

Relationship of Body Mass Index to Time

Mean age-specific BMI increased with time. This relationship between weight and time has two components, illustrated with data from the oldest and youngest birth cohorts of our sample (**Figure 1**). The first is the natural history of body weight: Average BMI increased with age throughout early adulthood. The second is a secular weight trend: Persons born in later calendar years tended to have larger age-specific BMI values. In every

Figure 1. **Mean age-specific body mass index (*BMI*), based on age and birth cohort.**



Circles represent mean BMI for people born in 1964 (n = 1228), and squares represent mean BMI for people born in 1957 (n = 1196). Error bars represent 95% CIs.

case in which the same age was sampled for both birth cohorts, the group born in 1957 had a lower mean BMI than the group born in 1964. Intermediate birth cohorts had intermediate-range mean BMI values.

In the complete sample, there were striking differences in the development of BMI by ethnicity (**Figure 2**). Black women had the highest average BMI values at each age sampled, and white women had the lowest. Mean BMI values for Hispanic women were between those for the other two groups. Average BMI reached overweight status (BMI $\geq$ 25 kg/m^2) in black women by age 26 years, in Hispanic women by age 28 years, and in white women by age 35 years. By age 39 years, the average BMI of black women was in the obese range (BMI $\geq$ 30 kg/m^2), whereas that of white women was in the mildly overweight range.

Ethnic-based differences in average BMI were also seen in men. However, Hispanic men consistently had the highest mean age-specific BMI values. Average BMI values for black and white men were similar until age 30 years, at which time they diverged, with the mean BMI in black men increasing more rapidly. Average BMI reached overweight status by age 25 years in Hispanic men and by age 27 years in all other men.

Birth Cohort Differences

After adjustment for ethnicity, birth cohort variables were statistically significant in Cox proportional

Figure 2. **Mean age-specific body mass index (*BMI*), based on sex and race or ethnicity.**

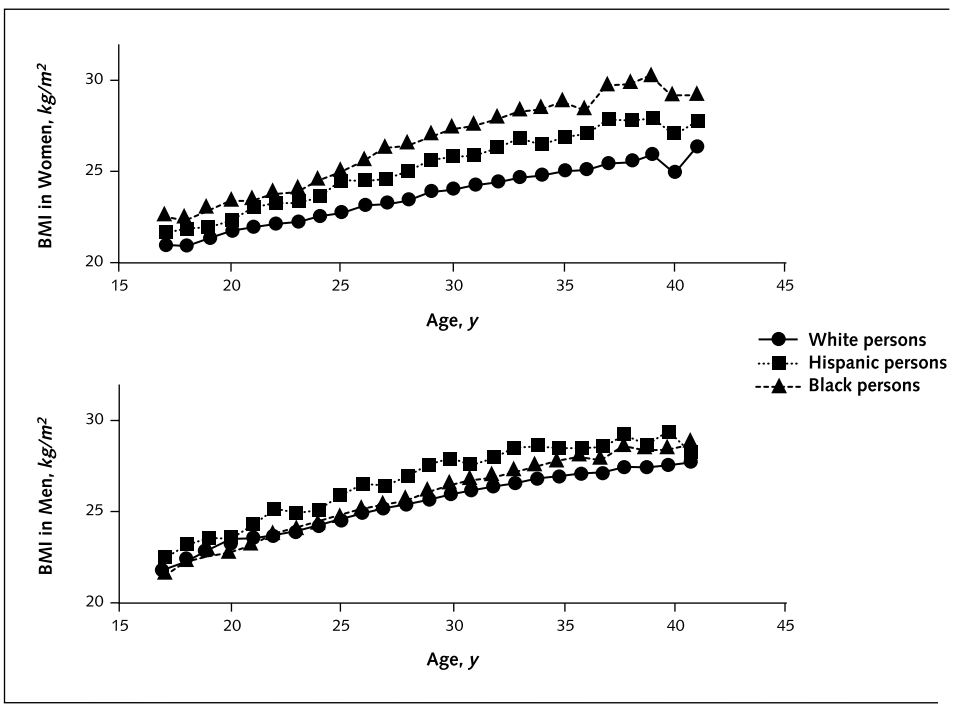
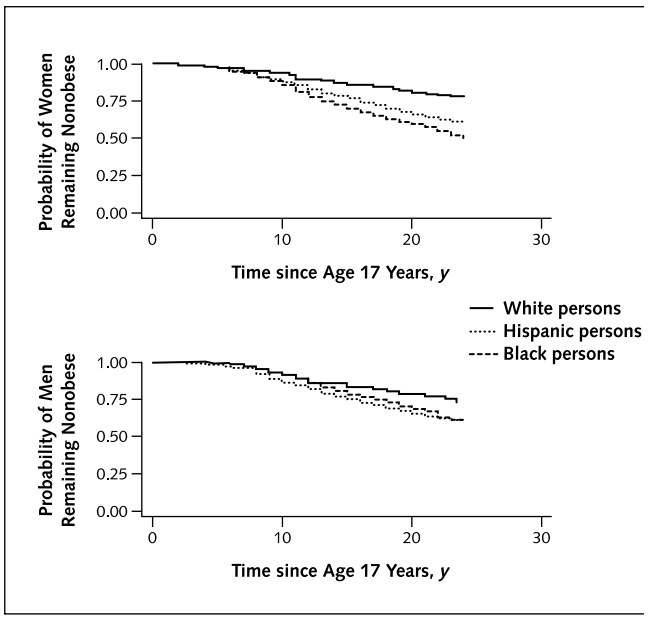


Figure 3. **Kaplan–Meier survivor curves: time to obesity for 17- and 18-year-old men and women who were not obese.**



hazards models of time to obesity from age 24 years for men (n = 3946) and women (n = 4043), quantifying the trend seen in **Figure 1**. The hazard ratio for birth year, in 2-year increments, was 1.06 (95% CI, 1.03 to 1.10); for each 2-year increment in birth year, obesity onset occurred 6% faster. Hazard ratios were nearly identical for men and women. Overall, across 8 birth years sampled, the latest-born men became obese 26% more rapidly and the latest-born women 28% more rapidly than those of the earliest birth cohort.

Ethnic Differences in Time to Onset of Obesity

We analyzed relative rates of obesity onset in 1089 men and 1053 women who were 17 and 18 years of age and not obese. In women, Kaplan–Meier curves showed the same ethnic-based patterns seen in the BMI plots (**Figure 3**). Cox proportional hazards modeling quantified relative rates of obesity onset: Black women reached obesity 2.1 (CI, 1.6 to 2.7) times faster and Hispanic women reached obesity 1.5 (CI, 1.1 to 2.0) times faster than did white women.

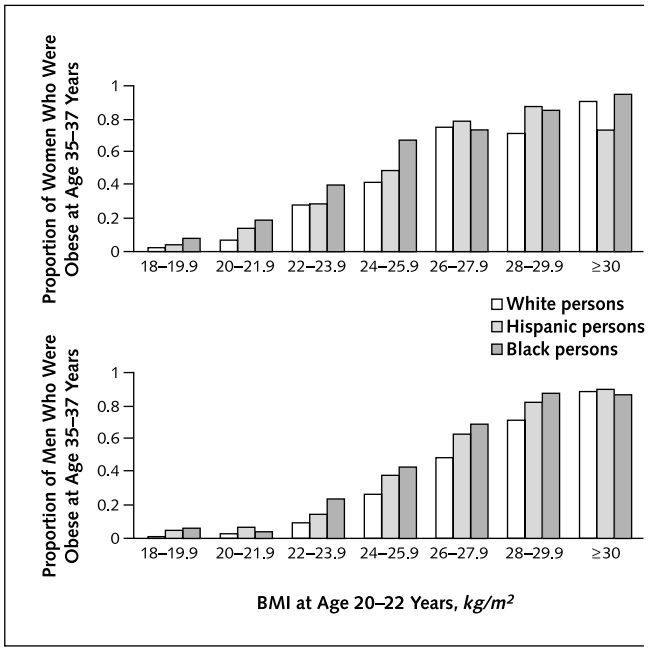
Hazard was proportional between Hispanic and white men, and the hazard ratio quantified obesity onset as 2.5 (CI, 1.9 to 3.3) times faster among Hispanic men. When obesity onset in black men was compared with that in white men, the proportional hazards assumption was violated. This can be seen from the Kaplan–Meier curves: Hispanic ethnicity was associated with the most rapid onset of obesity at all ages, but differences between black and white men in time to onset of obesity were age-dependent (**Figure 3**). Both black men and white men showed similar rates of obesity onset at the transition into adulthood, but obesity developed more rapidly in black men after approximately age 28 years. Because of the proportional hazards violation, black and white men were compared across two periods. From age 17 to 28 years, there was no significant difference between the time to onset of obesity between these two groups, whereas after age 28 years, obesity developed significantly more rapidly in black men (hazard ratio, 2.2 [CI, 1.5 to 3.4]). The proportional hazards assumption for the latter model was met by both graphical and Schoenfeld criteria.

When the Cox proportional hazards models were repeated and baseline BMI at age 17 to 18 years was adjusted, results were similar, although ethnic differences were slightly less pronounced. Black women reached obesity 1.5 (CI, 1.2 to 2.0) times faster than did white women, and Hispanic women showed a nonsignificant trend (hazard ratio, 1.1 [CI, 0.8 to 1.5]) toward more rapid onset of obesity than did white women. Hispanic men reached obesity 2.1 (CI, 1.5 to 2.7) times faster than did white men. No significant association was found between rates of obesity onset in black and white men between ages 17 and 28 years, but after age 28 years, black men became obese 2.4 (CI, 1.6 to 3.6) times more rapidly than did white men.

Prevalence, Incidence, and Predictors of Weight Problems in Early Adulthood

Obesity prevalence at age 35 to 37 years was substantial. Of 2998 men and 3134 women sampled at these ages, 26% (CI, 24.5% to 27.7%) of men and 28% (CI, 26.3% to 29.5%) of women were obese. More than 80% of men and women who were obese in their mid-30s became obese during early adulthood. When obesity status at age 35 to 37 years was compared with BMI values at age 20 to 22 years, the observed prevalence of obesity at age 35 to 37 years increased with early adult-

Figure 4. **Observed obesity at age 35 to 37 years in relationship to baseline body mass index (*BMI*) at age 20 to 22 years.**



Within each ethnic group and 2-point range of baseline BMI at age 20 to 22 years, bar height represents the proportion of persons who reached obesity at age 35 to 37 years.

hood BMI (**Figure 4**). This relationship differed by race and ethnicity. For example, 41% of white, 47% of Hispanic, and 66% of black women who had a BMI of 24 to 25 kg/m^2 became obese by age 35 to 37 years. A similar pattern was seen for men.

In the logistic regression model that quantified risk for obesity at age 35 to 37 years, baseline BMI, race or ethnicity, and sex were statistically significant predictors when adjusted for birth year. The model had good predictive ability, with an area under the ROC curve of 0.86. Predicted probability of obesity increased with increasing baseline BMI. Each additional 1 kg/m^2 at age 20 to 22 years was associated with a 1.67-fold (CI, 1.6-fold to 1.7-fold) increase in the odds of being obese at age 35 to 37 years. Black persons had the highest odds for obesity onset (odds ratio, 2.3 [CI, 1.9 to 2.8]), followed by Hispanic persons (odds ratio, 1.6 [CI, 1.3 to 1.9]), and finally white persons. Men were less likely than women to develop obesity (odds ratio, 0.5 [CI, 0.4 to 0.6]). If a 60% risk for obesity by age 35 to 37 years is considered substantial, then all women, regardless of race or ethnicity, were at substantial risk even if BMI values were only mildly elevated (25 to 27 kg/m^2) at age 20 to 22 years. Men reach this level of risk with slightly higher baseline BMI values (26 to 28 kg/m^2).

DISCUSSION

Much attention has been focused on the role of weight loss in treating chronic health conditions, especially diabetes, hypertension, and heart disease. Yet, the development of weight problems in adults has not been well described. This study uses unique longitudinal data from a large random sample of Hispanic, black, and white young adults to show substantial obesity incidence in early adulthood. We found that differences based on race or ethnicity were due to differential pediatric and young adult weight gain. Individuals at high risk for obesity could be identified at entry to adulthood. In addition, a 2-year increment in birth year was linked with more rapid onset of obesity.

Because we analyzed subgroups of the NLSY79 and examined data longitudinally, we cannot extrapolate our findings to national prevalence. However, more than one quarter of our sample was obese by the time they reached their mid-30s, an age much younger than the age at which most of the long-term harmful health consequences of obesity begin. Early treatment may prevent a significant health burden and, thus, should be a priority. Of note, fewer than 15% of those who were obese at age 20 to 22 years were nonobese at age 35 to 37 years, suggesting that although obesity can be reversed, such reversal is uncommon. This may underscore the difficulty in treating obesity or reflect patterns of medical practice that overlook obesity intervention (17).

It is critical to note that not only is obesity potentially preventable, but persons at high risk for obesity could be identified at a young age. In all racial and ethnic groups, being mildly or moderately overweight at age 20 to 22 years was linked with substantial incidence of obesity by age 35 to 37. Prevention efforts should focus particularly on persons just transitioning into adulthood who exhibit any level of being overweight. In this sample, the risk for obesity reflects weight accumulation in childhood and early adulthood, suggesting that interventions should incorporate both pediatric and adult health care concerns and providers. Overweight children are at increased risk for becoming obese adults,

and obese adults are, in turn, at risk for raising obese children. Effective public health intervention must address the links between these two populations (18, 19). In addition, women were twice as likely as men to become obese by age 35 to 37 years, adjusted for race or ethnicity, pediatric weight development, and birth year. Women should therefore receive particular attention.

We confirmed ethnic differences in weight development, which may contribute to the high prevalence of obesity in black and Hispanic populations. In our sample, black women and Hispanic men were at highest risk. Efforts to prevent or treat obesity in black men should take into consideration the delay in elevated risk in this group until late in the third decade of life. Although we cannot assess the cause of such differences in our study, it is important to note that race or ethnicity and sex are likely surrogates, totally or in part, for other factors, such as dietary and exercise standards, income, education, and parity. Here, we merely identify groups in which further examination is essential if we are to understand underlying culture-specific contributors to obesity.

A secular trend toward increasing overweight and obesity prevalence as well as weight gain in a particular group over time has been previously noted (1, 8). However, earlier studies have not quantified a shift in the age of obesity onset. We found a 26% to 28% more rapid onset of obesity in birth years from 1957 to 1964. This alteration in human weight development is too rapid to reflect a plausible shift in population genetics and more likely reflects a change in societal dietary and exercise patterns. We cannot determine whether this societal shift has concluded or is still in progress. It has disturbing implications for the incidence of obesity-related morbidity as the U.S. population ages.

This study has limitations. One is the use of self-reported weight and height. Our study probably provides a conservative estimate of the sample's obesity incidence and prevalence. Self-reporting bias tends to underestimate obesity that is based on BMI, especially for people at height or weight extremes (11, 20). This bias varies with age and is minimized in young adults (21, 22). In the NHANES III population, the sensitivity of self-reported BMI values for correctly classifying people as overweight (BMI $\geq$ 25 kg/m^2) was 86% to 94% in the age range examined here (22). Most important, for the NHANES III participants, self-reported compared with measured BMI values revealed no appreciable, consistent difference across non-Hispanic white, non-Hispanic black, and Mexican-American groups (22). Therefore, we expect that our ethnic-based differences are not due to systematic reporting error. A second limitation is attrition. Weight reporting decreased over time, with only 70% of the sample reporting weight in 1998. About one third of the loss was due to exclusion of a white, impoverished subsample in 1992, which altered the socioeconomic structure of the white group and potentially introduced bias. The baseline BMI values of those excluded did not differ from the general sample, and the response rate of eligible participants did not vary by race or ethnicity. A third limitation is the difficulty of accurate adjustment for clustering by region. Even with a conservative adjustment for such clustering, conclusions were unchanged: All results that were initially statistically significant remained so.

As we looked for insights into how to combat the slow-moving epidemic of obesity in our society, we reviewed the experience of more traditional outbreak investigations, which are based on evaluation of disease in terms of place, person, and time. We have addressed the "place" domain with a geographically diverse sample of the U.S. population. The identification of people at highest risk for obesity helps target further investigation, both in terms of clarifying the mechanisms of increased risk and by exploring how this risk can best be combated. The "time" domain of the obesity problem is complex because weight develops on such a slow time scale. However, this aspect may provide the most insight to the underlying problem. Time of highest risk refers not just to an age range at which an individual is at highest risk for weight gain but also to the era in which the person matures. Both must be accounted for in understanding an individual's risk, and both can be targeted for intervention. This can be done by recognizing that treatment of obesity in young adults should be a health priority and that society must work to alter dietary and exercise standards.

It is disquieting to note that obesity is a risk factor for four of the six most frequent causes of death in 1999 (5, 6, 12, 23). This observation underscores the importance of reversing the trend of obesity and the fact that chronic disease is the dominant cause of death in the United States. As a slowly developing health problem that is difficult to treat, frustrating for patients and pro-

viders, and subject to a behavioral component, obesity may be a good model for planning clinical guidelines and public health interventions for chronic disease. Examining the development of a chronic disease on a time scale specific to that disease allows us to effectively focus on factors that structure the population dynamics and potential opportunities for intervention. This study provides important evidence that for obesity, more emphasis needs to be placed on the young adult period and particularly selected subpopulations.

From the University of North Carolina at Chapel Hill, Chapel Hill, North Carolina.

Acknowledgments: Dr. McTigue thanks the Robert Wood Johnson Clinical Scholars Program for its support. The authors thank Tom Swasey for graphical assistance.

Grant Support: By the Robert Wood Johnson Clinical Scholars Program and National Institute of Child Health and Human Development (R01-HD39183).

Requests for Single Reprints: Kathleen McTigue, MD, MS, MPH, 5034 Old Clinic Building, CB#7105, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599-7105; e-mail, kathleen_mctigue@unc.edu.

Current author addresses and contributions are available at www.annals.org.

References

1. Health, United States, 2000. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. DHHS Publication No. (PHS) 2000-1232-1. Hyattsville, MD: National Center for Health Statistics; 2000.

2. Prevalence of Overweight and Obesity among Adults: United States, 1999. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. Hyattsville, MD: National Center for Health Statistics; 2000.

3. **Allison DB, Saunders SE.** Obesity in North America. An overview. Med Clin North Am. 2000;84:305-32. [PMID: 10793644]

4. **Thompson D, Edelsberg J, Colditz GA, Bird AP, Oster G.** Lifetime health and economic consequences of obesity. Arch Intern Med. 1999;159:2177-83. [PMID: 10527295]

5. **Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH.** The disease burden associated with overweight and obesity. JAMA. 1999;282:1523-9. [PMID: 10546691]

6. **Pi-Sunyer FX.** Health implications of obesity. Am J Clin Nutr. 1991;53: 1595S-1603S. [PMID: 2031492]

7. **Najjar MF, Kuczmarski RJ.** Anthropometric data and prevalence of overweight for Hispanics: 1982–1984. Series 11: Data from the National Health Survey No. 239. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control. DHHS Publication No. (PHS) 89-1689. Hyattsville, MD: Centers for Disease Control; 1989.

8. **Lewis CE, Jacobs DR Jr, McCreath H, Kiefe CI, Schreiner PJ, Smith DE, et al.** Weight gain continues in the 1990s: 10-year trends in weight and overweight from the CARDIA study. Coronary Artery Risk Development in Young Adults. Am J Epidemiol. 2000;151:1172-81. [PMID: 10905529]

9. **Rissanen A, Heliövaara M, Aromaa A.** Overweight and anthropometric changes in adulthood: a prospective study of 17,000 Finns. Int J Obes. 1988;12: 391-401. [PMID: 3235260]

10. **Williamson DF, Kahn HS, Remington PL, Anda RF.** The 10-year incidence of overweight and major weight gain in US adults. Arch Intern Med. 1990;150:665-72. [PMID: 2310286]

11. **Rowland ML.** Self-reported weight and height. Am J Clin Nutr. 1990;52: 1125-33. [PMID: 2239790]

12. National Institute of Diabetes and Digestive and Kidney Diseases (U.S.) Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: the Evidence Report. National Heart, Lung, and Blood Institute. NIH publication no. 98-4083. Bethesda, MD: National Heart, Lung, and Blood Institute; 1998.

13. **Johnson TP, Mott JA.** The reliability of self-reported age of onset of tobacco, alcohol and illicit drug use. Addiction. 2001;96:1187-98. [PMID: 11487424]

14. **Cawley J.** An instrumental variables approach to measuring the effect of body weight on employment disability. Health Serv Res. 2000;35:1159-79. [PMID: 11130815]

15. **Guo G, Harris KM.** The mechanisms mediating the effects of poverty on children's intellectual development. Demography. 2000;37:431-47. [PMID: 11086569]

16. User's Guide: A Guide to the 1979–1998 National Longitudinal Survey of Youth 1979 Data. Sponsored by the U.S. Bureau of Labor Statistics. Columbus, OH: Center for Human Resource Research, Ohio State Univ; 1999.

17. **Galuska DA, Will JC, Serdula MK, Ford ES.** Are health care professionals advising obese patients to lose weight? JAMA. 1999;282:1576-8. [PMID: 10546698]

18. **Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T.** Do obese children become obese adults? A review of the literature. Prev Med. 1993;22:167-77. [PMID: 8483856]

19. **Parsons TJ, Power C, Logan S, Summerbell CD.** Childhood predictors of adult obesity: a systematic review. Int J Obes Relat Metab Disord. 1999;23 Suppl 8:S1-107. [PMID: 10641588]

20. **Kuskowska-Wolk A, Karlsson P, Stolt M, Rössner S.** The predictive validity of body mass index based on self-reported weight and height. Int J Obes. 1989; 13:441-53. [PMID: 2793299]

21. **Nieto-García FJ, Bush TL, Keyl PM.** Body mass definitions of obesity: sensitivity and specificity using self-reported weight and height. Epidemiology. 1990;1:146-52. [PMID: 2073502]

22. **Kuczmarski MF, Kuczmarski RJ, Najjar M.** Effects of age on validity of self-reported height, weight, and body mass index: findings from the Third National Health and Nutrition Examination Survey, 1988-1994. J Am Diet Assoc. 2001;101:28-34. [PMID: 11209581]

23. Deaths: Final Data for 1999. Department of Health and Human Services, Centers for Disease Control and Prevention. National Vital Statistics Reports Vol. 49, No. 8. Hyattsville, MD: National Center for Health Statistics; 2001.