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Do Birth Cohorts Matter? Age-Period-Cohort Analyses of the Obesity Epidemic in the United States

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Abstract

Many studies have cited the importance of secular changes or "period effects" as causes of the U.S. obesity epidemic. Unfortunately, relatively little attention has been devoted to the possible influence of cohort-related mechanisms. To address this current gap in the scientific literature, this investigation utilized the responses from 1.7 million participants in the 1976-2002 National Health Interview Surveys to determine how birth cohorts may have contributed to the rapid increase in the prevalence of obesity. Results from hierarchical age-period-cohort (HAPC) models confirmed that period effects are principally responsible for the U.S. obesity epidemic. However, HAPC models also demonstrated that birth cohort membership is influential. Independent of age and period effects, the predicted probability of obesity at age 25 increased by 30% for cohorts born between 1955 and 1975. Our results also showed that age, period and cohort effects varied by race/gender and educational attainment. For instance, increases in the predicted probabilities of obesity were particularly sharp for recent cohorts of Black females. Our investigation successfully demonstrated that both secular change and birth cohort membership have independently contributed to elevated odds of obesity among recent generations of Americans, suggesting that cohort-specific strategies may be needed to combat disconcertingly high rates of obesity in the U.S.

Keywords

USA; obesity; obesity epidemic; body mass index (BMI); health disparities; birth cohorts; period effects, gender, ethnicity

Introduction

Studies of the U.S. population have consistently shown a disconcerting increase in obesity rates in recent decades (Flegal, Carroll, Ogden, & Johnson, 2002; Mokdad, Bowman, Ford, Vinicor, Marks, & Koplan, 2001; Ogden, Flegal, Carroll, & Johnson, 2002). Although recent data from the National Health and Nutrition Examination Surveys (NHANES) suggest that the rapid

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climb in obesity rates may be nearing a peak for adult women, these same data show that the obesity epidemic continues unabated among children and adult males (Ogden, Carroll, Curtin, McDowell, Tabak, & Flegal, 2006). Indeed, even if recent data offer some cause for optimism, it is sobering to consider that the prevalence of obesity among U.S. adults has more than doubled since 1980 (Flegal et al., 2002).

What has caused the obesity epidemic? Researchers and medical professionals have offered several different answers to this question, including technological innovations (Cutler, Glaeser, & Shapiro, 2003; Philipson & Posner, 2003), the aggressive marketing of high calorie foods (Brownell, 2002), and passive leisure activities (French, Story, & Jeffery, 2001). While specific explanations vary, nearly all share the common view that secular changes in U.S. society (i.e., period effects) lie at the root of the obesity epidemic. Many of these arguments are plausible and intuitively appealing. However, it is premature to conflate the obesity epidemic with secular trends, since there has been insufficient research to disentangle period effects from age and birth cohort effects in studies of the U.S. population.

Definitions of age, period, and cohort effects

Age, period, and cohort effects all refer to some type of time-related variation in the phenomena of interest, yet they carry distinct substantive meanings (Yang, 2008a). *Age effects* are defined as variations associated with different chronological age groups brought about by physiological changes, accumulation of social experience, and/or role or status changes. Age effects, therefore, reflect biological and social processes of aging internal to individuals and represent developmental changes across the life course. Extant research shows that weight gain is common from late adolescence through midlife, as people change physiologically (e.g., resting metabolic rate tends to decline) and reduce their levels of physical activity (Starling, 2001), perhaps in response to the demands of new roles, such as work and parenthood. Although changes in age structure could account for the increasing prevalence of obesity in the U.S., strong period trends persist after age-adjustment. With age removed from consideration, most scholars presume that the observed increase in obesity rates must be due to period effects (e.g., Lewis, Jacobs, McCreath, Kiefe, Schreiner, & Smith et al., 2000).

Period effects are defined as variation over time periods or calendar years that influence all age groups simultaneously (Yang, 2008a). Period effects subsume a complex set of historical events and environmental factors such as world wars, economic crises, famine, epidemics and pandemics of infectious diseases, public health interventions, and technologic breakthroughs. Shifts in social, cultural, economic, or physical environments may in turn induce similar changes in the lives of all people at a given point in time. Existing evidence shows that obesity rates have increased in recent decades among all sociodemographic and racial/ethnic groups in every geographic area of the U.S., lending credence to period-related speculation about the obesity epidemic. However, it is also important to recognize that obesity rates have not risen uniformly (Ogden et al., 2006). This raises the possibility that cohort-related mechanisms may have contributed to the historic rise in obesity rates.

Cohort effects are defined as changes across groups of people who experience an initial event such as birth or marriage in the same year or years (Yang, 2008a). Birth cohorts are the most commonly examined unit of analysis in demographic and aging research. A birth cohort moves through life together and encounters the same historical and social events at the same ages. Birth cohorts that experience different historical and social conditions at various stages of their life course, therefore, have diverse experiences. In a study of data from recent National Health Surveys in Australia, birth cohort membership was significantly associated with the prevalence of overweight, net of age and year of observation (Allman-Farinelli, Chey, Bauman, Gill, & James, 2008). The patterning of cohort effects in this study revealed a sharp increase in the prevalence of overweight for cohorts born after 1960—even after taking strong period effects

into consideration. The authors surmise that recent birth cohorts have spent more of their lives in environments conducive to obesity, predisposing them to rapid weight gain that begins relatively early in life.

Theoretical considerations

Consistent with several existing theories of the obesity epidemic, Ryder (1965) argues that technological innovation is the "principal motor of contemporary social change" (p. 851). Ryder also maintains that secular changes such as technological innovation generally influence multiple birth cohorts simultaneously. "To some extent all cohorts respond to any given period specific stimulus. Rarely are changes so localized in either age or time that their burden falls exclusively on the shoulders of one cohort" (p. 847). However, new cohorts are vitally important to the process of social change because they are more likely than older cohorts to embrace new technologies and their corresponding modes of work and leisure. Through receptivity to modern technologies, products and media, individuals in recent birth cohorts may be particularly likely to adopt sedentary lifestyles and high calorie diets. Conversely, older cohorts have significant histories of adaptation to past social norms, values and technological standards, which can make them resistant to social change. In light of these theoretical propositions, the finding that recent birth cohorts in Australia are more susceptible to overweight than their predecessors (Allman-Farinelli et al., 2008) does not seem surprising.

Extant research also suggests that recent birth cohorts are vulnerable to the obesogenic environment in the U.S. For instance, whereas the prevalence of obesity doubled among adults over the period 1976-80 to 1999-2000 (Flegal et al., 2002), the prevalence of overweight *tripled* among adolescents aged 12-19 over this same period (Ogden et al., 2002). These figures do not disentangle period and cohort effects, so it is impossible to determine why the prevalence of overweight has increased at such a fast pace among U.S. adolescents. However, one distinct possibility is that birth cohort membership contributes an added source of risk for young Americans, over and above period effects that influence the society at large. Exposure to innovative technologies and sophisticated marketing campaigns at impressionable ages may cause individuals in new cohorts to develop tastes and preferences that increase their risk of obesity, not just in childhood and adolescence, but throughout the life course. Consistent with this proposition, a recent study found that newer birth cohorts of Australian males engage in less leisure-time physical activity than their predecessors, net of age and period influences (Allman-Farinelli, Chey, Merom, Bowles, & Bauman, 2008).

Cohort trends in key demographic groups

As noted, the obesity epidemic has been ubiquitous in its reach, affecting virtually all social and demographic groups in the U.S. While this is important, it is also essential to recognize that there are substantial inequalities in the prevalence of obesity in U.S. society. Two of the most widely documented sources of these inequalities are educational attainment and the intersection between race/ethnicity and gender. Unlike some measures of socioeconomic status (SES) such as income, which have shown inconsistent effects on obesity, educational attainment has consistently been found to have a strong, inverse association with body mass in more developed nations like the U.S. (McLaren, 2007; Mokdad et al., 2001). Also, although 2003-04 NHANES data show only minor differences in the prevalence of obesity between non-Hispanic Black men (34.0%) and non-Hispanic White men (31.1%), they reveal sharp differences between non-Hispanic Black women (53.9%) and non-Hispanic White women (30.2%) (Ogden et al., 2006).

Certainly it is important to understand the mechanisms responsible for these inequities, as a number of studies have sought to do. For instance, McLaren (2007) argues that well educated individuals may be particularly sensitive to cultural values regarding health and body images,

and sufficiently motivated to meet those standards. Also, Robert and Reither (2004) have found that both individual SES and community-level disadvantages help explain body mass differences between Black and non-Black women, although large differences persist after adjustment for these factors. However, in the context of the present investigation, it is also important to document how birth cohort trends in obesity have differed across these groups. Working from the theoretical framework established previously, it might seem reasonable to expect that recent birth cohorts from advantaged social groups would be at heightened risk for obesity due to superior access to modern goods and technologies. On the other hand, recent cohorts from advantaged social groups might be in a better position to utilize these technologies while minimizing their adverse side effects. Such a viewpoint would be consistent with the fundamental social cause perspective articulated by Link and Phelan (1996).

Research questions

To better understand the etiology of the obesity epidemic, this study will estimate age, period and cohort effects in cross-classified random-effects models (CCREMs) of obesity among U.S. adults in the National Health Interview Survey (NHIS), 1976-2002 (National Center for Health Statistics, 2003; U.S. Department of Health and Human Services, 2001). Three major research questions will be addressed through CCREMs of NHIS data:

1. Does birth cohort membership influence the odds of obesity in the U.S.?
2. Are period effects the predominant force behind the obesity epidemic?
3. Do age, period and cohort effects differ across groups defined by race/gender and educational attainment?

Methods

Study populations

The NHIS is a repeated cross-sectional household survey of the noninstitutionalized civilian population in the U.S. (National Center for Health Statistics, 2004). Its primary functions are to monitor the prevalence and distribution of disease and disability in the U.S. and assess patterns of health care utilization. Every week, interviewers from the U.S. Census Bureau gather information from “responsible family members” residing in randomly chosen households across the nation (Adams, Hendershot, & Marano, 1999, p. 2). Prior to 1997, demographic and health information was collected on every person in the household through self-responses, proxy responses, or some combination of the two. In 1997, NHIS underwent a major redesign that eliminated proxy reporting for adults (National Center for Health Statistics, 2000). On average, Census personnel complete interviews at about 94 percent of selected households (Adams et al., 1999).

Although the NHIS extends back to 1957, it did not begin collecting data on height and weight until 1976. This timing is fortunate since available estimates suggest that the onset of the obesity epidemic occurred sometime in the 1980s (Flegal, Carroll, Kuczmarski, & Johnson, 1998). In this study, we merged NHIS data from 1976-2002 into a single database consisting of approximately 1.7 million adults aged 18 and over. The NHIS also investigated the health of children and adolescents over the period 1976-2002, but it did not include estimates of their height or weight.

In addition to NHIS data, we incorporated data from NHANES II (1976-1980), NHANES III (1988-1994) and NHANES Continuous (1999-2002) for use in BMI corrections (details to follow). Like NHIS, NHANES utilizes probability sampling techniques to study the health of the noninstitutionalized civilian population in the U.S. (McDowell, Engel, Massey, & Maurer, 1981). Unlike NHIS, NHANES contains direct anthropometric measures of height and weight.

Despite this advantage, NHANES was only conducted periodically from 1976-2002 and, in recent waves, has censored information on the year of observation. By comparison, NHIS has superior statistical power and refined information on year of observation, which is vital to constructing birth cohorts and understanding the functional form of period effects.

Measures

We calculated body mass index (BMI = weight(kg)/height(m^2)) from values of height and weight reported to NHIS interviewers. Estimates of BMI from reported height and weight have known biases (Cawley, 2004; Kuczmarski, Kuczmarski, & Najjar, 2001; Villanueva, 2001). In addition, two features specific to NHIS warranted attention. First, prior to 1997, NHIS estimates of mean BMI were biased downward by the inclusion of proxy reports of weight. Following the lead of Reither and Utz (2009), we applied a series of regression equations to correct these biases. Second, comparing NHIS self-reports to NHANES examination data revealed that the downward bias in reported BMI increased over the period 1976-2002. We accounted for downward biases in NHIS estimates of BMI through a series of correction equations, developed with the aid of examination and survey data in the Second, Third and Continuous versions of NHANES. While this second step was rooted in a methodology developed by Cawley (2004), it also accounted for increasingly biased self-reports of weight over the period 1976-2002. As shown in Figure 1, this age-period-survey (APS) adjustment caused NHIS estimates of BMI to closely approximate estimates from NHANES examination data. Implementation of the APS adjustment did not alter the main substantive conclusions in this study but, without the adjustment, period effects were distorted (particularly in 1997) and biased downward. Readers interested in learning more about the APS adjustment should consult Reither (2005) for complete details.

For APC analyses, BMI was dichotomized into a categorical variable for obese (BMI $\geq$ 30) and not obese (BMI < 30). Age was subtracted from period (i.e., year of study) to identify birth cohorts, which ranged from 1877 to 1984. Cohorts were arranged into five-year groups, with the exception of the initial cohort (1877 to 1899), which covered a broader range of years to ensure a sufficient number of subjects. Age was measured in single year increments. Because each wave of NHIS was relatively large, periods were also measured in single year increments.

Measures of gender, race and education were also extracted from NHIS. In each year from 1976-2002, NHIS included a racial identification variable that categorized respondents as White, Black or other. Race was recoded as either Black or non-Black for use in stratified models. Due to substantial changes in the measurement of Hispanic ancestry during this period, ethnic classifications were not utilized. Education was recoded into less than high school (0-11 years of education), high school (12 years of education), some college (13-15 years of education) and college or more (16 or more years of education).

Statistical analyses

To this point, we have discussed the theoretical rationale for including cohort effects in analyses of the obesity epidemic. In addition, it is important to include age, period, *and* cohort effects in studies of health and mortality to avoid model misspecification and biased interpretations of age and period effects. For instance, studies of mortality trends in recent decades have tended to assume equal declines across birth cohorts and, consequently, have found evidence of strong period trends (Yang, 2008a). In contrast to these studies, Yang, Fu, and Land (2004) have shown that when age, period, and cohort are modeled simultaneously, mortality declines since 1960 are principally due to cohort effects.

Therefore, it is important to include birth cohorts in studies of the obesity epidemic for both theoretical and empirical reasons. With repeated cross-sectional survey data like the NHIS, it

is not possible to track the same individuals over time, as in a prospective cohort study. However, it is possible to construct synthetic birth cohorts from such data (Yang & Land, 2006). We illustrate the construction of the 1973 birth cohort with a small selection of NHIS data in Figure 2. Respondents who were 18 years of age in the 1991 NHIS belong to the 1973 birth cohort, as do respondents who were 19 years of age in the 1992 NHIS, and so on. Synthetic birth cohorts approximate actual birth cohort trajectories well, except in the case of large scale migration during survey years.

Before the introduction of model specifications for this analysis, it is necessary to address the identification problem posed by APC analysis using conventional linear regressions applied to the aggregate tabular age by period data. As implied by Figure 2, regression coefficient estimates are not unique due to the exact linear dependency, Period – Age = Birth Cohort, when age and period are measured in the same interval length (Mason & Fienberg, 1985). Conventional solutions to the identification problem include: 1) setting equality constraints on at least one set of parameter estimates for age, period or cohort; and 2) linear nonparametric transformations, such as polynomial terms of age effects in repeated cross-sectional APC analysis (Yang, 2008b). For more information on conventional solutions to the identification problem as well as the limitations of these solutions, please consult Mason and Fienberg (1985), and Robertson, Gandini, and Boyle (1999).

In this investigation, we group birth cohorts into 5-year intervals, which are conventional in demography (Yang, 2008b). These 5-year intervals also function as equality constraints, since each birth cohort within the interval is constrained to have the same effect. We also estimate obesity as a quadratic function of age based on exploratory data analysis (as shown in Figure 3). These two steps suffice to identify fixed-effects models, but because such models do not account for the multilevel structure of the data, the estimation of fixed-effects models may result in collinearity, downwardly biased standard errors, and Type I errors that far exceed specified values for alpha. Therefore, we adopted a hierarchical APC (HAPC) approach and specified cross-classified random-effects models (CCREMs) that estimate fixed effects for age and its quadratic term, but that estimate random effects for periods and birth cohorts by treating these variables as level-2 factors (Yang & Land 2006; Yang & Land, 2008). Such a model may be expressed as follows:

$$Y_{ijk}=\alpha_{ijk}+\beta_{1jk}A+\beta_{2jk}A^2+e_{ijk}, \quad (1)$$

where Y_{ijk} represents the presence or absence of obesity for the ith participant for i = 1, ..., n_{jk} individuals in the jth period of observation for j=1,..., J period and the kth birth cohort for k = 1, . . ., K cohort. The terms A and A^2 represent age and age-squared, respectively. Also, we "centered" at 25 years of age to facilitate interpretation of the random effects, and also to reduce the association between age and age-squared.

Random intercepts embedded in equation (1) may be expressed as follows:

$$\alpha_{jk}=\pi_0+t_{0j}+c_{0k}, \quad (2)$$

where α_{jk} indicates that the overall mean varies for each period of observation j and each birth cohort k, π_0 is the expected mean when level-1 variables (i.e., the centered values for age and age-squared) are zero, t_{0j} is the overall period effect in terms of residual random coefficients for period j averaged over all birth cohorts with variance σ_{t0}, and c_{0k} is the overall cohort effect in terms of residual random coefficients of cohort k averaged over all time periods with variance σ_{k0} (notation and terminology adapted from Yang (2008b)). To ensure that the CCREM is

appropriate for our data, we conducted a Hausman specification test to compare results from fixed- and random-effects models. Following the guidelines for large samples developed by Yang and Land (2008), we failed to reject the null hypothesis of no difference in coefficient vectors ($\chi^2(2) = 11.856$; $p = 0.003$), indicating that the CCREM specification is preferable.

With the models specified in Equations 1 and 2, we estimated a logit CCREM of obesity for the U.S. population using SAS PROC GLIMMIX (Littell, Milliken, Stroup, Wolfinger, & Schabenberger, 2006). In addition, we stratified CCREMs of obesity by race/gender and educational attainment to estimate the degree to which age, period and cohort effects have varied across these key demographic groups. In the stratified models for educational attainment, we retained only NHIS participants aged 25 or older to allow sufficient time for completion of formal education. Survey weights provided by NHIS were used to adjust for response probabilities and sampling design.

Results

Descriptive statistics are shown in Figure 3. Application of NHIS weights produced estimates of population and obesity prevalence for all age groups and birth cohorts that are nationally representative over the period of observation in this study (1976-2002). The prevalence of obesity increased sharply with age, peaking at nearly 26 percent in the early to mid-50s, before declining in later life. The prevalence of obesity also increased sharply by period of observation. In 1976, the prevalence of obesity was about 12 percent among adults in the U.S. By 2002, it had climbed to over 30 percent. According to descriptive data, rates of obesity rates rose steadily among U.S. cohorts born during the first three decades of the twentieth century, from around 10 percent to nearly 24 percent. Obesity rates then declined steadily for a series of birth cohorts, before rising once again for cohorts born in the late 1960s and early 1970s. The rest of the results are organized by the three main research questions in our investigation.

Cohort membership influences the odds of obesity

Coefficients from CCREMs demonstrate that the odds of obesity vary significantly by birth cohort membership in the U.S., net of age and period effects ($\sigma_{k0} = 0.0096$; $p < 0.01$). Moreover, twelve out of eighteen random cohort effects were statistically significant among the sample of U.S. adults in the NHIS (see Table 1). But more importantly, the pattern of cohort effects suggests that recent birth cohorts are at elevated risk for obesity in adulthood. To facilitate interpretation of the coefficients shown in Table 1, we transformed them into predicted probabilities of obesity (see Yang 2008b for details). As shown in Figure 4, the predicted probability of obesity for a 25 year-old adult (recall that age was centered at 25) in the U.S. population declined steadily from 0.133 for the 1890 birth cohort to 0.108 in the 1955 cohort, then rose to a peak of 0.141 in the 1975 cohort. This indicates that the probability of obesity at age 25 increased by 30% for cohorts born between 1955 and 1975. While the predicted probability of obesity dropped slightly for the 1980 birth cohort, the overall pattern of random effects shows a distinct upward trend after 1955.

Period effects are principally responsible for the obesity epidemic

Results from the CCREM for U.S. adults (see Table 1) show that period of observation had a significant influence on the odds of obesity between 1976 and 2002 ($\sigma_{t0} = 0.136$; $p < 0.001$). Independent of birth cohort effects, the predicted probability of obesity for a 25 year-old adult was 0.075 in 1976 (see Figure 4). By 2002, the predicted probability of obesity for a 25 year-old adult had risen to 0.206, representing an increase of 175%. In addition, the pattern of predicted probabilities for U.S. adults shows a monotonic increase over time, with no sign of abatement in recent periods of observation. Although we have shown that birth cohort membership influences the predicted probabilities of obesity, these results clearly indicate that

ubiquitous secular changes (i.e., period effects) are principally responsible for the obesity epidemic in the U.S. population.

APC effects differ by race/gender and educational attainment

The importance of birth cohort membership varies substantially by race/gender and educational attainment. For instance, there are hardly any discernable trends in cohort effects for non-Black men or college graduates (see Figure 4 and Figure 5).[1] Conversely, cohort trends for non-Black women and those with some college closely follow the overall trend among U.S. adults. Even more striking, however, are the very strong birth cohort effects observed for Black participants in the NHIS, especially Black women (see Figure 4). Like the rest of U.S. population, Black adults who were born during the first half of the twentieth century experienced a gradual decline in rates of obesity. However, after reaching a nadir in 1955, the predicted probabilities of obesity rose sharply for subsequent cohorts of Black men and women. For instance, from a low of 0.190 for a 25 year-old adult from the 1955 cohort of Black women, the predicted probability of obesity increased to 0.307 for the 1975 cohort of Black women—an increase of 62%.

Like cohort effects, both age and period effects differ across race/gender groups and varying levels of educational attainment. Among all groups studied, the predicted probability of obesity increased rapidly through young adulthood, peaked in middle age and then declined in later life (see Figure 4 and Figure 5). However, the exact patterning of age effects was substantially different among these groups. For instance, the predicted probability of obesity rose more rapidly with age and peaked at much higher levels among Black females than other race/gender groups. Although not as dramatic, a similar pattern was observed for NHIS respondents with less than a high school education. Also, it is interesting to note that predicted probabilities peaked at younger ages for men than women, regardless of race.

Period effects for Black men, non-Black men and non-Black women closely followed trends for the population of U.S. adults (see Figure 4). Although period trends for Black women were not dissimilar, their predicted probability of obesity was clearly much higher across the entire period of observation. In 1976, educational attainment showed a distinct gradient effect, where the predicted probability of obesity decreased with each step up in education. However, by 2002 this gradient had essentially disappeared due to a relatively rapid increase in the odds of obesity among NHIS participants with a high school degree or some college. By the end of this period, only a college degree appeared to confer any meaningful protection against obesity.

Discussion

At the outset of this investigation, we identified three main research questions. First, does birth cohort membership influence the odds of obesity in the U.S.? Second, are period effects the predominant force behind the obesity epidemic? Third, do age, period and cohort effects differ across groups defined by race/gender and educational attainment? Results of CCREMs for 1.7 million NHIS participants over the period 1976-2002 answered each of these questions with an unambiguous “Yes.”

The answer to the first of these questions is most vital to our study—and it is worth reiterating. Independent of age and period effects, birth cohort membership has significantly influenced the odds of obesity in the U.S. Relative to cohorts born toward the end of the nineteenth century, cohorts born during the first half of the twentieth century tended to experience declining odds of obesity. However, the predicted probabilities of obesity for more recent birth cohorts have

[1]Regression estimates for the HAPC models represented in Figure 5 are available upon request.

tended to increase since the late 1950s and early 1960s. This indicates that newer birth cohorts have suffered a form of double jeopardy in which ubiquitous secular changes and birth cohort membership have independently contributed to increased odds of obesity. In the context of secular changes that have caused obesity rates to increase sharply across nearly all social and demographic groups in the U.S., the trends we observed among recent birth cohorts are deeply troubling. As noted recently in a series of life table analyses, life expectancy in the U.S. could decline in the near future if obesity trends persist (Olshansky, Passaro, Hershow, Layden, Carnes, & Brody et al., 2005). Although this rather dire prediction has been the subject of much criticism, our finding that newer birth cohorts are increasingly susceptible to obesity—even as period trends continue upward—suggests that there is some cause to heed Olshanksy's warning. Furthermore, because our findings were broadly consistent with those reported by Allman-Farinelli et al. (2008) for the Australian population, we have some concern that the health and longevity of populations in other developed nations could also be in jeopardy.

The cohort trends we observed are consistent with assertions that newer birth cohorts permit social change through rapid cultural and technological adaptation (Ryder, 1965). Although the mechanisms that have caused obesity to increase among more recent birth cohorts are not well understood, one possibility is that the amount of time that children and adolescents spend in front of computers and video games has increased rapidly in recent years (Subrahmanyam, Kraut, Greenfield, & Gross, 2000). While some studies suggest that computers and video games do not substantially contribute to sedentary behaviors among children and adolescents (e.g., Sturm, 2005), others indicate that these technologies do not substitute for television viewing but rather increase the total amount of “screen time” (Coffee & Stipp, 1997; Nielsen Media Research, 1999). Computer use is also positively associated with the consumption of soft drinks and snacks among middle- and high-school students, perhaps due to advertisements that target youths through internet media (Utter, Neumark-Sztainer, Jeffery, & Story, 2003). Other plausible mechanisms include changing dietary patterns, sleep debt, pharmaceutical drug use (e.g., the rising use of antidepressants and antihistamines), and epigenetic factors, to name just a few (Keith et al., 2006). The latter is an alarming possibility, as it suggests that recent birth cohorts may be predisposed to gain weight—and that they could pass on this predisposition to their own children. Given the potential threat to public health caused by the rising odds of obesity among newer birth cohorts, it is vitally important to begin the process of identifying these mechanisms—and determining their relative importance—so that appropriate interventions can be implemented.

Recent cohorts could also manifest higher rates of obesity than their predecessors through improved survivorship among obese persons. Analyses of NHANES data have found that, relative to persons of normal weight, the risk of mortality among obese persons has declined in the U.S. since the 1970s (Flegal, Graubard, Williamson, & Gail, 2005). Reductions in mortality among obese persons are likely due to a combination of improving cardiovascular risk profiles among the obese (Gregg, Cheng, Cadwell, Imperatore, Williams, & Flegal et al., 2005) and declining rates of coronary heart disease mortality (Gu, Cowie, & Harris, 1999), which is the chief cause of death among obese persons in the U.S. (Flegal et al., 2005). Improving mortality profiles among obese persons may seem at odds with bleak predictions about the future health and longevity of the U.S. population and, indeed, there are some who argue that medical progress could more than compensate for increasing obesity rates (e.g., Preston 2005). However, the promise of continued medical progress should be balanced against compelling evidence that obesity at young ages can have serious long-term health consequences (see, for example, Freedman, Patel, Srinivasan, Chen, Tang, & Bond et al., 2008). In any event, through the twin mechanisms of increasing probabilities of obesity and declining rates of cardiovascular mortality, new cohorts may contribute to the obesity epidemic through “demographic metabolism” in which older, leaner cohorts are replaced with newer, heavier cohorts (Ryder, 1965, p. 843).

In addition to confirming the existence of birth cohort effects, our study affirms that period effects were principally responsible for the obesity epidemic. Independent of age andcohort effects, the predicted probability of obesity increased substantially over the period 1976-2002. Predicted probabilities of obesity increased for all groups studied, confirming the ubiquitous reach of the obesity epidemic. Secular changes responsible for strong period effects may include an increasing reliance on technology for work (French et al., 2001; Lakdawalla & Philipson, 2002), food preparation (Cutler et al., 2003) and leisure activities (French et al., 2001). Also, technological innovations have reduced food costs and nearly removed economic incentives to engage in physical activity (Lakdawalla & Philipson, 2002; Philipson & Posner, 2003). Moreover, modern technologies have been used to develop and transmit aggressive marketing campaigns designed to increase the consumption of high calorie foods (Brownell, 2002; French et al., 2001). While there is no shortage of speculation about the underlying causes of period effects, research has yet to disentangle the relative importance of these factors.

Results from this study support previous research showing that body mass increases rapidly through young adulthood but subsequently increases at a slower pace (Lewis et al., 2000; Rissanen, Heliovaara, & Aromaa, 1988). Since physical activity tends to decline during young adulthood (Sternfeld, Sidney, Jacobs Jr., Sadler, Haskell, & Schreiner, 1999) and dietary behaviors may include frequent fast food consumption (French, Harnack, & Jeffery, 2000), it is not surprising to observe increasing odds of overweight and obesity among young adults. The plateau observed during middle age may result from more careful monitoring of calorie intake (Lewis et al., 2000), since available evidence suggests that basal energy expenditure and physical activity continue to decline at these ages (Crespo, Keteyian, Heath, & Sempos, 1996; Starling, 2001). These results are also consistent with findings that weight loss is common at older ages (Rissanen et al., 1988). Ironically, this is not encouraging since weight loss in later life may result from muscular atrophy and is associated with increased risk of illness, hospitalization and mortality (Dey, Rothenberg, Sundh, Bosaeus, & Steen, 2001; Roubenoff, 1999).

Although age effects are parabolic for each demographic group that we studied, considerable differences in the predicted probabilities of obesity were nevertheless detected. For instance, the predicted probability of obesity increased and then declined more rapidly with age among Black women relative to other race/gender groups. We made a similar, though less pronounced finding for NHIS respondents with less than a high school education. Consistent with existing evidence, these findings indicate that Black women and persons without a high school diploma are at particularly high risk of developing obesity at some point during the life course. The sharp decline in obesity among undereducated persons and Black women as they aged may also indicate that individuals in these groups are particularly likely to suffer the adverse consequences of obesity, such as weight loss due to illness and premature mortality.

We also detected important differences in period and cohort effects for various demographic groups. For instance, while Black women did not exhibit substantially different period trends than other race/gender groups, their predicted probabilities of obesity were substantially higher than other race/gender groups across the entire period of observation. The predicted probabilities of obesity for cohorts of Black females were also consistently elevated. Also troubling was the very sharp increase in cohort effects that we observed for Black females who were born after 1955. Although the mechanisms responsible for this increase are not clear, they are broadly consistent with a fundamental social cause interpretation (Link & Phelan, 1996). That is, relative to more advantaged demographic groups, young Black females may not be well equipped to mitigate the risks introduced by recent technological changes in U.S. society.

Another important finding in our study was the convergence of period trends for all groups with less than a college education. From 1976 through the mid-1990s, the predicted

probabilities of obesity declined at each step up the educational ladder. But from the late 1990s onward, only a college education appeared to confer any protection against obesity. This finding may reflect improved high school graduation rates and increasing exposure to college over this period of observation (Newburger & Curry, 1999). Persons without a college degree are increasingly disadvantaged in U.S. society, and part of this disadvantage appears to be reflected in the period trends that we observed. Race/gender and educational differences in age, period and cohort effects pose a significant challenge to laudable public health goals to reduce health disparities, such as those articulated in *Healthy People 2010* (U.S. Department of Health and Human Services, 2000).

Strengths of our study include a large nationally representative sample, micro-level data and refined measures of age, period and cohort. Also, the initial period of observation (1976) in our study preceded the sharp increase in obesity rates (Flegal et al., 1998), suggesting that estimates for period effects were not attenuated by the inability to include earlier time periods. In addition, our investigation utilized a state-of-the-art methodological approach (CCREM) to help overcome problems that have historically plagued age-period-cohort analyses, such as collinearity and biased standard errors. This methodological approach enabled us to demonstrate that secular changes are principally responsible for the obesity epidemic, while simultaneously showing that the odds of obesity have increased for recent birth cohorts. The adoption of CCREMs also provides a framework for future analyses to incorporate covariates (e.g., food prices) that may account for rising rates of obesity. Presuming that the inclusion of such variables leads to substantial attenuation in period and/or cohort effects, it will contribute much to our understanding of the etiological factors responsible for the obesity epidemic.

The unavailability of direct, anthropometric measures of height and weight is a limitation of NHIS data, particularly since NHIS used a peculiar mix of proxy, partial-self and self-reports between 1976 and 1996. However, a unique quality of our investigation is that it accounted for reporting status and other biases through the development of a new adjustment procedure that caused BMI estimates in NHIS to approximate those based on NHANES examination data. Another limitation of this study is its inability to identify Hispanics. Between 1970 and 2000, the Hispanic population increased from 4.7 to 12.5 percent of the total U.S. population (U.S. Census Bureau, 2008). Although obesity rates do not differ for Hispanic and non-Hispanic White men, the prevalence of obesity tends to be higher among Hispanic women than non-Hispanic White women (Ogden et al., 2002). As a consequence, the period and cohort effects that we observed for non-Black females may be due in part to the changing ethnic composition of this group. Although similarities in our estimates for non-Black females and non-Black males suggests that the influence of Hispanics is relatively small, it is worth noting that the divergence in cohort effects for these two groups after 1965 may reflect the increasing influence of Hispanics on recent cohorts of non-Black females. Finally, this study is unable to assess whether the odds of obesity have continued to increase among cohorts born after 1984. Clearly, it is imperative to bring data to bear on this important question.

Future research should investigate obesity trends among more recent birth cohorts by conducting APC investigations of adolescents and young adults with existing resources (e.g., the National Longitudinal Study of Adolescent Health) and, if necessary, primary data collection. Emerging public health issues among children and adolescents such as type 2 diabetes and fatty liver—health problems that used to be true medical oddities in this age range—strongly suggest that the birth cohort trends we observed in this study have persisted for more recent birth cohorts (Ludwig, 2007). If additional research confirms our findings, it could prove useful to develop cohort-specific strategies that limit weight increases early in the life course. Two possible strategies include targeted mass media campaigns (Bauman, 2004) and the removal of obstacles to routine physical activity that did not exist for older birth cohorts (Dellinger, 2002). As U.S. society continues to evolve, it may also be advisable to craft

interventions that are sensitive to the formative social and cultural milieu that influence the values and preferences of individual birth cohorts, in much the same way that some health-related policies are sensitive to issues involving gender or race/ethnicity. Developing successful cohort-specific interventions is likely to be challenging, in part because the social changes responsible for the obesity epidemic are unlikely to reverse course, and also because the causes of and solutions to this problem are likely to be complex, multifactorial, and somewhat different for various birth cohorts. Nevertheless, because the obesity epidemic has serious implications for the health (Must, Spadano, Coakley, Field, Colditz, & Dietz, 1999; Visscher & Seidell, 2001), longevity (Olshansky et al., 2005), economy (Wolf & Colditz, 1998) and quality of life (Han, Tijhuis, Lean, & Seidell, 1998; Hassan, Joshi, Madhavan, & Amonkar, 2003) of the U.S. population, every effort should be undertaken to improve our understanding of its causes and potential solutions.

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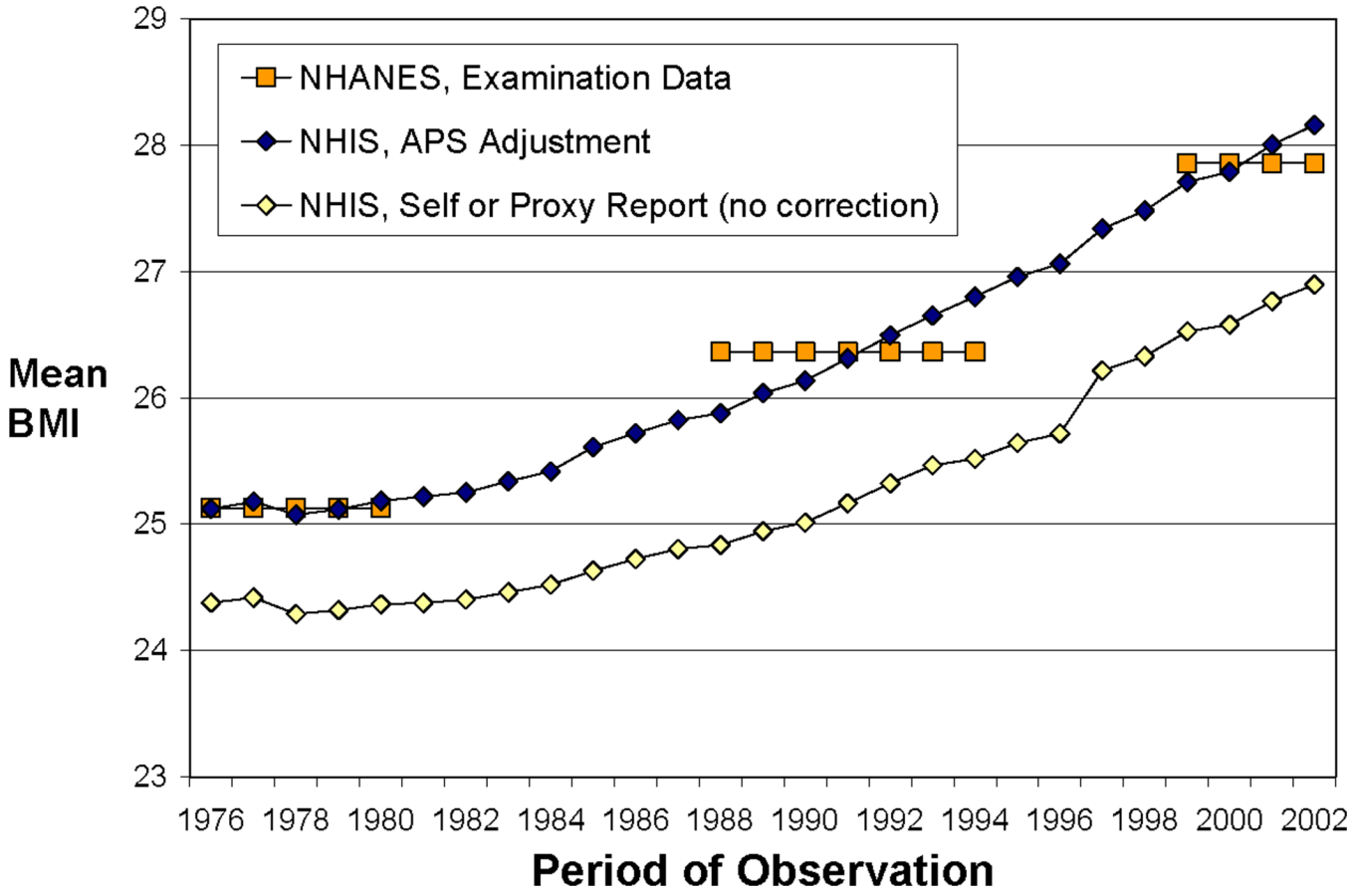

Figure 1.
Comparison of uncorrected NHIS estimates of mean BMI to corrected (APS) NHIS estimates and NHANES examination data, 1976-2002

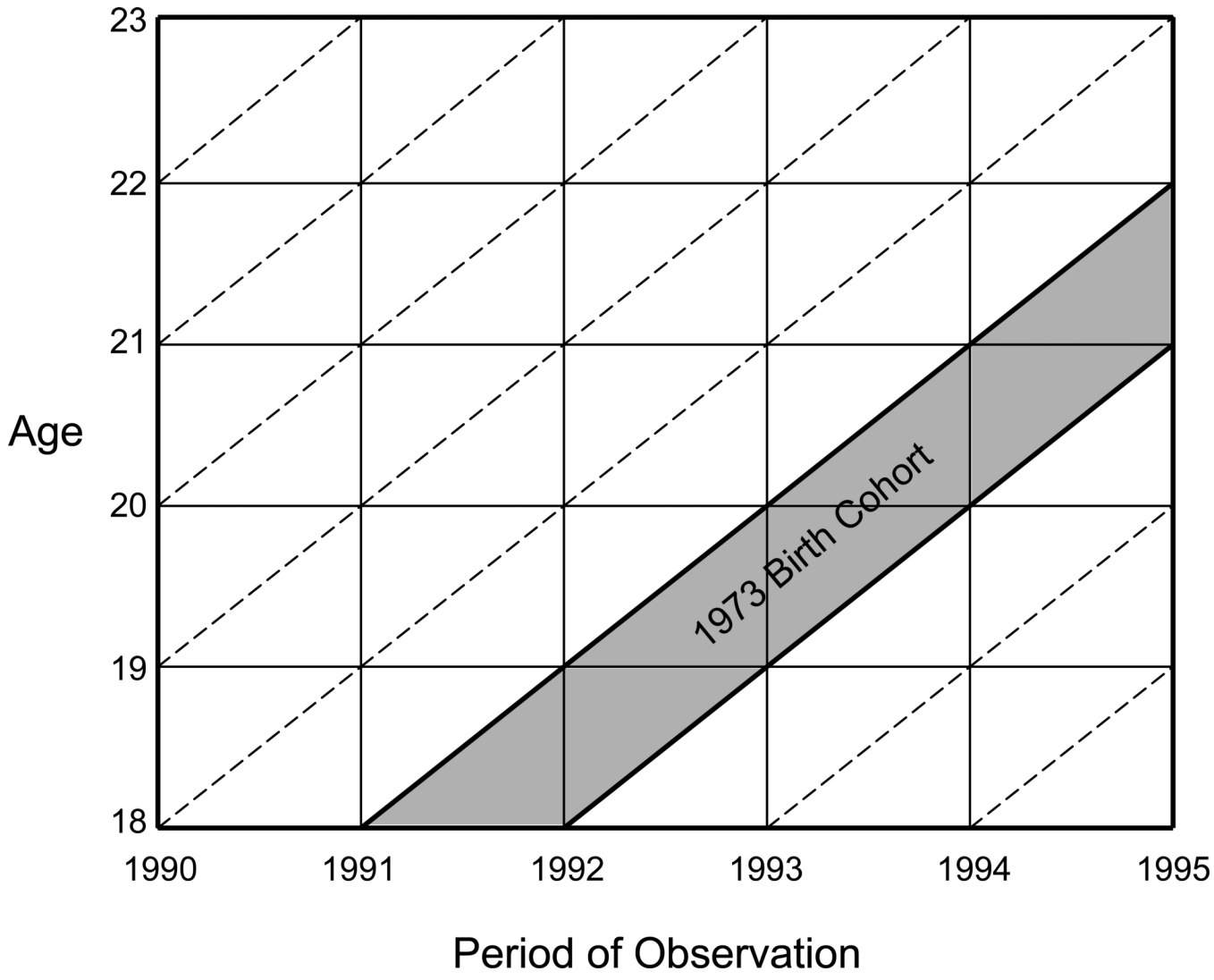

Figure 2.
Illustration of the construction of a synthetic birth cohort with repeated cross-sectional data

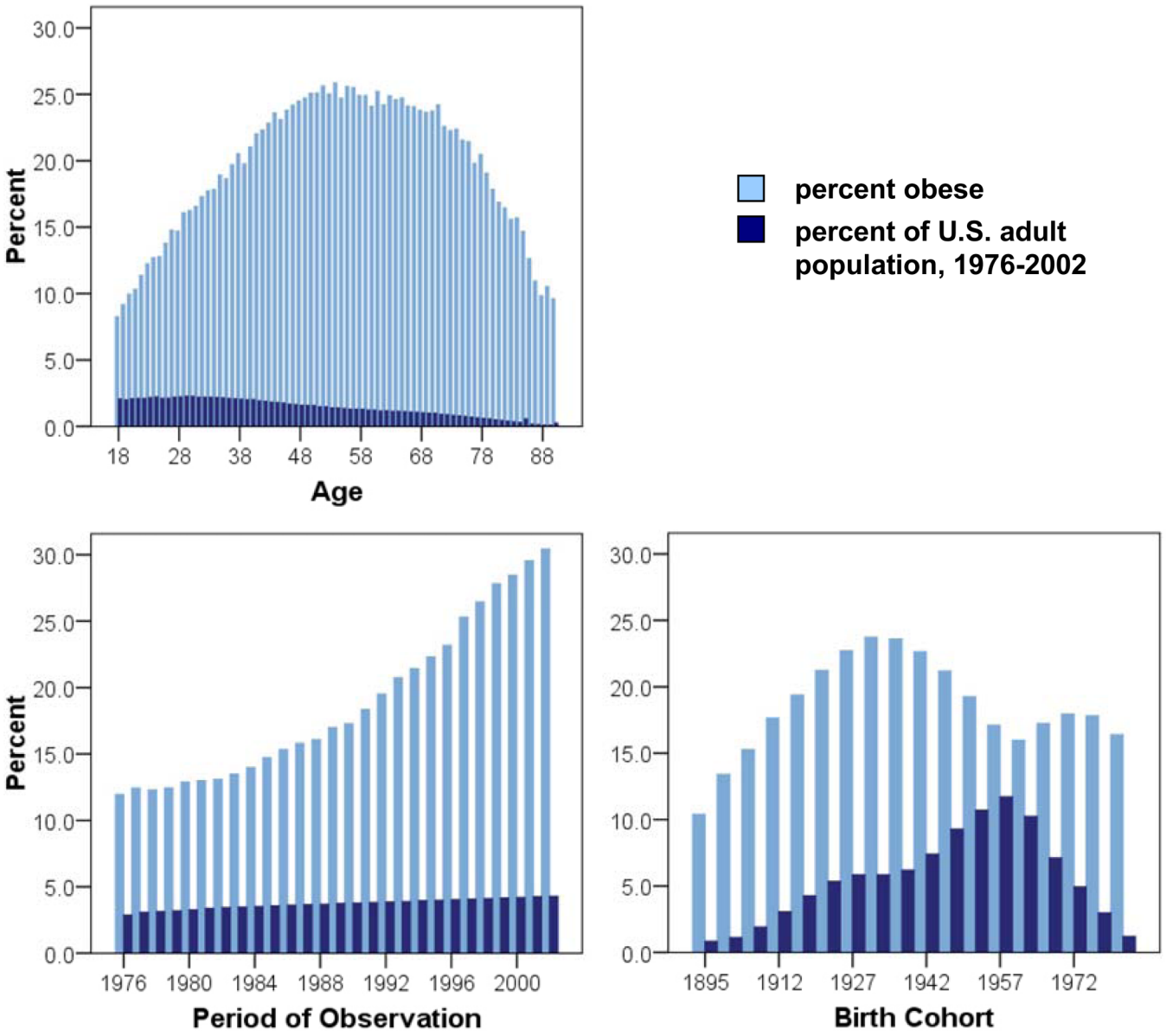

Figure 3.
Descriptive statistics for age, periods of observation and birth cohorts in the U.S. adult population, NHIS 1976-2002

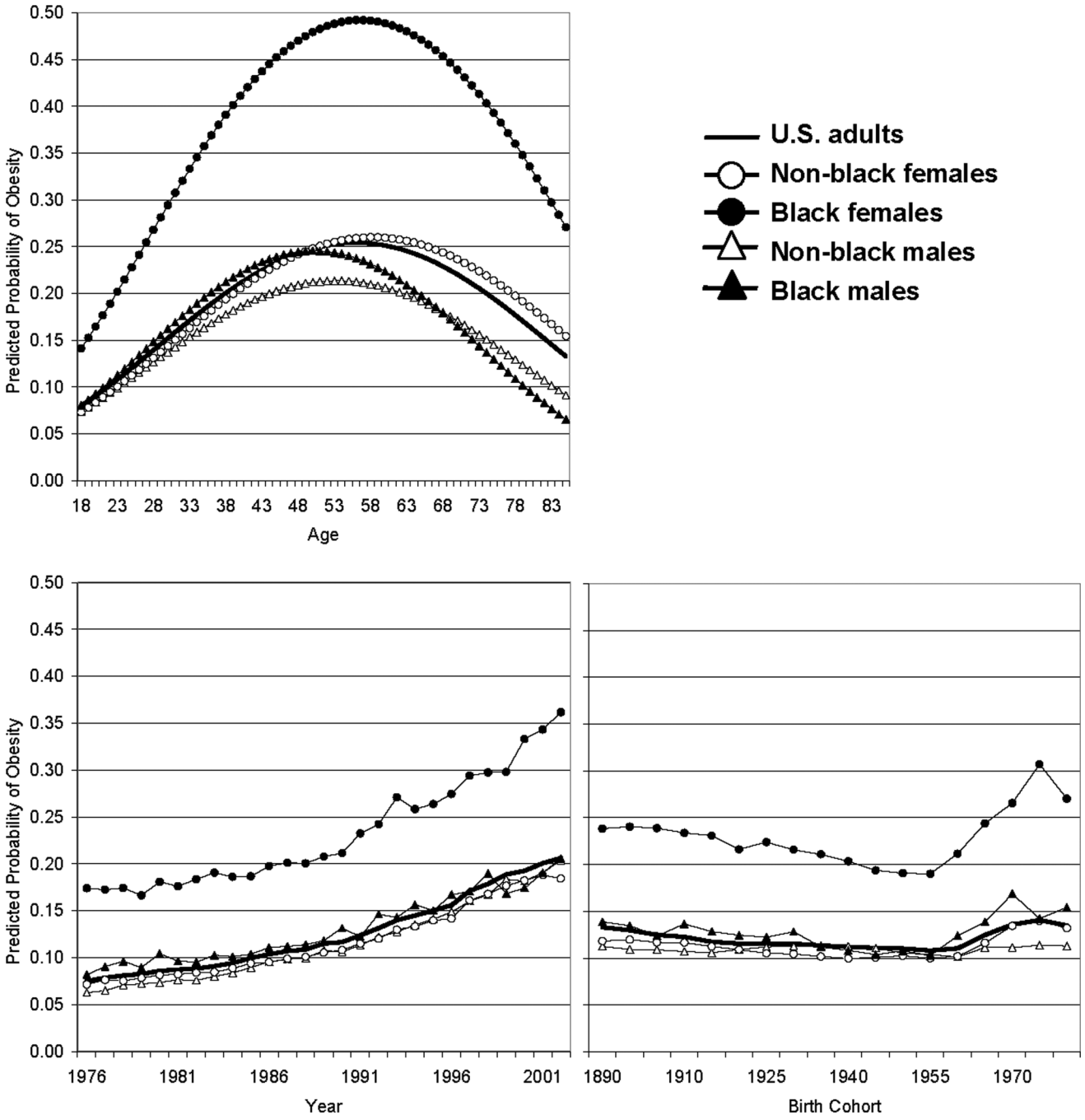

Figure 4.
Estimated predicted probabilities from hierarchical age-period-cohort models of obesity for U.S. adults and groups defined by race and gender, NHIS 1976-2002

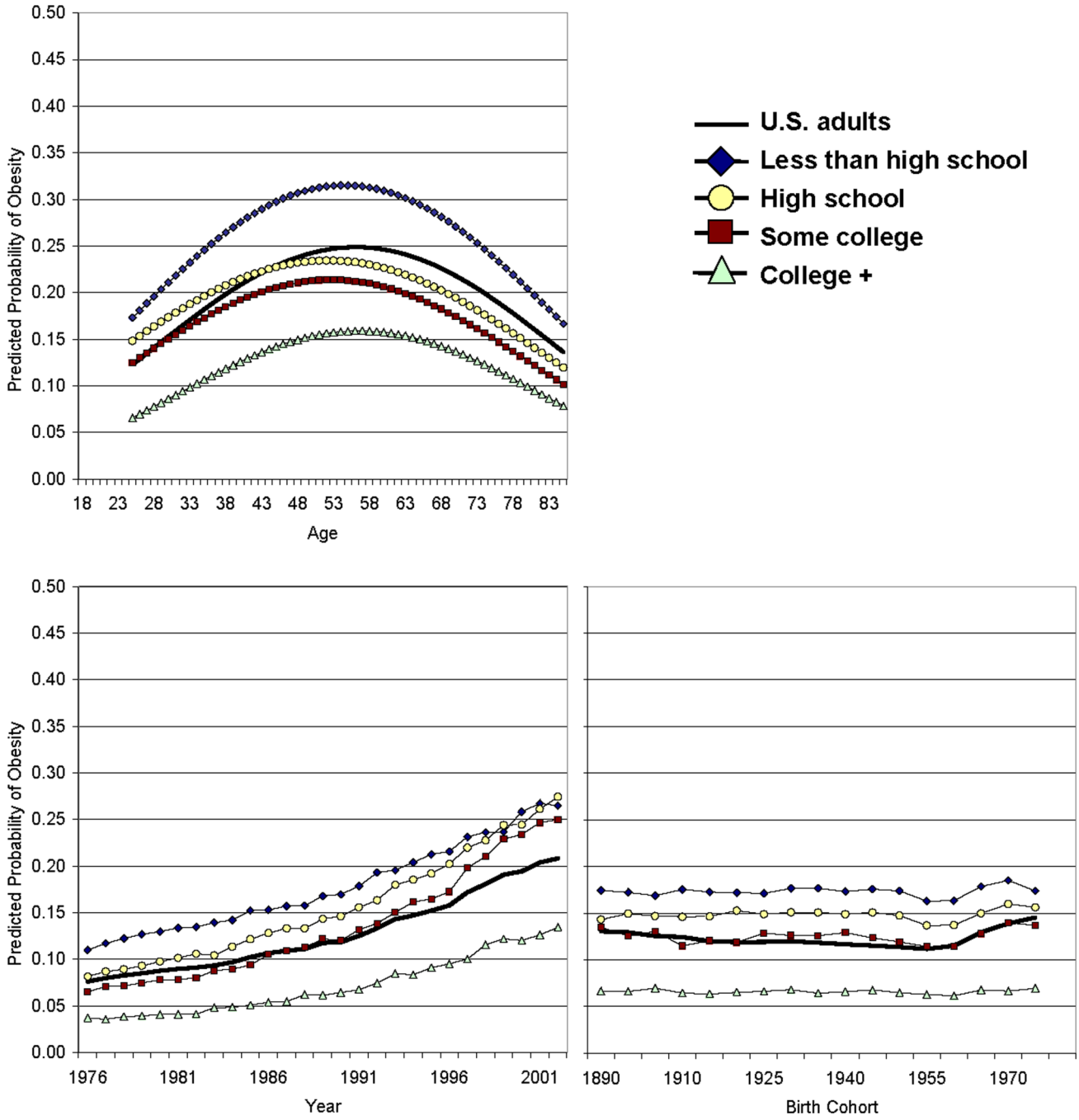

Figure 5.
Estimated predicted probabilities from hierarchical age-period-cohort models of obesity for U.S. adults and groups defined by educational attainment, NHIS 1976-2002

Table 1

Results from hierarchical age-period-cohort models of obesity for U.S. adults and groups defined by race and gender, NHIS 1976-2002

Fixed Effects	U.S. Adults	Non-Black Females	Black Females	Non-Black Males	Black Males
Intercept, π_0	−1.9876 ***	−2.0675 ***	−1.2201 ***	−2.0942 ***	−1.9294 ***
Age, π_1	0.0588 ***	0.0613 ***	0.0752 ***	0.0560 ***	0.0640 ***
Age^2, π_2	−0.0010 ***	−0.0009 ***	−0.0012 ***	−0.0010 ***	−0.0013 ***
Random Effects					
Period Effects, σ_{t0}	0.1363 ***	0.1280 ***	0.1034 ***	0.1687 ***	0.1007 ***
1976	−0.5243 ***	−0.4960 ***	−0.3386 ***	−0.6108 ***	−0.4900 ***
1977	−0.4740 ***	−0.4269 ***	−0.3479 ***	−0.5751 ***	−0.3784 **
1978	−0.4430 ***	−0.4435 ***	−0.3360 ***	−0.4834 ***	−0.3135 ***
1979	−0.4228 ***	−0.3996 ***	−0.3919 ***	−0.4595 ***	−0.3913 ***
1980	−0.3767 ***	−0.3557 ***	−0.2912 ***	−0.4408 ***	−0.2226 **
1981	−0.3618 ***	−0.3455 ***	−0.3228 ***	−0.3982 ***	−0.3096 ***
1982	−0.3492 ***	−0.3242 ***	−0.2732 ***	−0.4070 ***	−0.3233 ***
1983	−0.3143 ***	−0.3150 ***	−0.2260 **	−0.3489 ***	−0.2394 **
1984	−0.2747 ***	−0.2639 ***	−0.2557 ***	−0.2989 ***	−0.2579 **
1985	−0.2143 **	−0.2008 **	−0.2514 ***	−0.2266 **	−0.2273 **
1986	−0.1685 *	−0.1856 **	−0.1817 *	−0.1521	−0.1545
1987	−0.1346	−0.1440 *	−0.1591 *	−0.1232	−0.1410
1988	−0.1174	−0.1213	−0.1636 *	−0.1086	−0.1227
1989	−0.0544	−0.0715	−0.1189	−0.0215	−0.0797
1990	−0.0390	−0.0431	−0.0967	−0.0369	0.0424
1991	0.0292	0.0262	0.0253	0.0367	−0.0418
1992	0.0992	0.0750	0.0792	0.1199	0.1652
1993	0.1720 *	0.1652 *	0.2298 **	0.1698 *	0.1370
1994	0.2118 **	0.1945 **	0.1657 *	0.2357 **	0.2445 **
1995	0.2559 ***	0.2493 ***	0.1929 **	0.2877 ***	0.1964 **
1996	0.2989 ***	0.2651 ***	0.2472 ***	0.3485 ***	0.3217 ***
1997	0.4103 ***	0.4154 ***	0.3437 ***	0.4361 ***	0.3476 ***
1998	0.4625 ***	0.4672 ***	0.3600 ***	0.4883 ***	0.4752 ***
1999	0.5291 ***	0.5282 ***	0.3636 ***	0.6015 ***	0.3316 ***
2000	0.5549 ***	0.5659 ***	0.5256 ***	0.5884 ***	0.3728 ***
2001	0.6053 ***	0.6044 ***	0.5703 ***	0.6481 ***	0.4793 ***
2002	0.6400 ***	0.5803 ***	0.6513 ***	0.7307 ***	0.5791 ***
Cohort Effects, σ_{k0}	0.0096 **	0.0157 **	0.0311 *	0.0019 *	0.0295 *
1890	0.1127 *	0.0579	0.0581	0.0284	0.1038
1900	0.0855 *	0.0723	0.0691	−0.0018	0.0691
1905	0.0401	0.0453	0.0609	−0.0064	−0.0278
1910	0.0203	0.0426	0.0329	−0.0271	0.0855
1915	−0.0284	0.0003	0.0165	−0.0402	0.0142
1920	−0.0470	−0.0326	−0.0679	−0.0044	−0.0206
1925	−0.0523 *	−0.0737 *	−0.0228	0.0242	−0.0404
1930	−0.0489	−0.0804 *	−0.0707	0.0342 *	0.0140
1935	−0.0665 **	−0.1106 ***	−0.0998	0.0397 *	−0.1422 *
1940	−0.0833 ***	−0.1339 ***	−0.1455 **	0.0301	−0.1790 **
1945	−0.0934 ***	−0.1235 ***	−0.2056 ***	0.0086	−0.2256 ***
1950	−0.1013 ***	−0.1024 **	−0.2250 ***	−0.0437 **	−0.1837 ***
1955	−0.1222 ***	−0.1310 ***	−0.2297 ***	−0.0617 ***	−0.2382 ***

Fixed Effects	U.S. Adults	Non-Black Females	Black Females	Non-Black Males	Black Males
1960	−0.0992 ***	−0.1087 **	−0.0953	−0.0895 ***	−0.0250
1965	0.0386	0.0380	0.0888	0.0170	0.1029
1970	0.1381 ***	0.2055 ***	0.2030 **	0.0164	0.3332 ***
1975	0.1784 ***	0.2496 ***	0.4064 ***	0.0431	0.1331
1980	0.1288 **	0.1851 **	0.2267 *	0.0329	0.2267 *

Notes: σ_{t0} and σ_{k0} refer to the variance of random effects for period and cohort, respectively.

Standard errors for parameter estimates are omitted in the interest of space.

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$