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Birth cohort effects among U.S.-born adults born in the 1980s: Foreshadowing future trends in U.S. obesity prevalence

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Abstract

Background—Obesity prevalence stabilized in the U.S. in the first decade of the 2000s. However, obesity prevalence may resume increasing if younger generations are more sensitive to the obesogenic environment than older generations.

Methods—We estimated cohort effects for obesity prevalence among young adults born in the 1980s. Using data collected from the National Health and Nutrition Examination Survey between 1971 and 2008, we calculated obesity for respondents aged between 2 and 74 years. We used the median polish approach to estimate smoothed age and period trends; residual non-linear deviations

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CONFLICT OF INTEREST

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from age and period trends were regressed on cohort indicator variables to estimate birth cohort effects.

Results—After taking into account age effects and ubiquitous secular changes, cohorts born in the 1980s had increased propensity to obesity versus those born in the late 1960s. The cohort effects were 1.18 [95% CI: 1.01, 1.07] and 1.21 [95% CI: 1.02, 1.09] for the 1979–1983 and 1984–1988 birth cohorts, respectively. The effects were especially pronounced in Black males and females but appeared absent in White males.

Conclusions—Our results indicate a generational divergence of obesity prevalence. Even if age-specific obesity prevalence stabilizes in those born before the 1980s, age-specific prevalence may continue to rise in the 1980s cohorts, culminating in record high obesity prevalence as this generation enters its ages of peak obesity prevalence.

Keywords

Age factors; Obesity; Young adult; Developmental origins; Epidemiology; Models; statistical

INTRODUCTION

The 1980s are generally regarded as the start of a U.S. “obesity epidemic” [1]. During the 1980s, age-standardized obesity prevalence (body mass index [BMI] ≥ 30.0 kg/m^2) increased in U.S. adults by 55% from 14.5% to 22.5% obese. This increase occurred after two decades in which obesity prevalence was relatively stable [2].

The obesity increases of the 1980s continued in the 1990s then levelled off in the 2000s [2, 3]. In fact, between the early and late 2000s, in child and adult populations in the U.S., Europe, and Australia, obesity prevalence either stopped increasing, or increases decelerated [3]. It is unclear whether obesity prevalence in the U.S. and other countries has peaked or whether prevalence will resume increasing in the future [2, 3]. On the one hand, obesity prevalence may peak as anti-obesity efforts abate rising rates or as populations reach a prevalence ceiling. On the other hand, even in the U.S., the majority of adults are not obese and may remain susceptibility to increasing rates of obesity.

Because the causes of previous increases are not fully understood, it is difficult to predict future obesity trends[2]. Age-period-cohort analysis can improve forecasting by estimating one component of obesity trends: the relative susceptibility of birth cohorts to obesity. For example, Faeh et al. used a birth-cohort analysis to examine whether the stabilization of overweight prevalence in Switzerland is a temporary phenomenon [4]. They estimated that the birth cohorts of the 1960s and 1970s are more prone to overweight than those born between 1930 and 1959; therefore, Faeh et al. concluded that overweight prevalence may resume increasing as susceptible cohorts, adults currently in their 30s and 40s, age into their 50s and 60s, when risk of overweight is its highest [4]. This type of birth-cohort analysis is not definitive, however, because it failed to disentangle period effects from age-specific cohort trends [5].

To forecast future trends in the U.S., understanding the cohort-specific obesity susceptibility of those born in the 1980s may be key [5]. Individuals born during the 1980s experienced

gestation and early childhood – potentially key developmental life stages for obesity development -- during an era of rapidly increasing obesity prevalence. We hypothesize that the 1980s cohorts may be particularly sensitive to the obesogenic environment. For instance, hypotheses about the developmental origins of adult obesity posit that in utero and early-childhood exposures to obesogenic environments have latent biological or behavioral consequences the increase a person's susceptibility to excess weight gain into adulthood [6–9]. Two previous analyses of U.S. data found limited evidence of obesity risk being increased for cohorts born in the 1980s versus those born in the 1950s and 1960s but not the 1970s [10, 11]. However, both studies had limited data on these birth cohorts. The studies only assessed adult body size. Therefore, they observed the young adults born in the early 1980s for only the first few years of their adult lives in the 2000s, a narrow window of time to draw conclusions about lifelong cohort-specific risk. In addition, the studies only assessed trends in U.S. Whites and Blacks, failing to incorporate the experience of other U.S. racial and ethnic groups.

In this paper, we estimated cohort-specific propensity to obesity for those born in the U.S. in the 1980s. We used data on childhood obesity as well as adult obesity in order to produce estimates over the entire lifecourse of those born in the 1980s. Because the levelling off of obesity prevalence in the 2000s was more pronounced in women than men [2], we produced analyses stratified by sex. Further, because obesity prevalence varies significantly by racial/ethnic and sex subgroups, we also produced stratified estimates for the subgroups on which we had data extending back to the 1970s: non-Hispanic Black, non-Hispanic White, and U.S.-born Mexican-American men and women.

METHODS

Sample

We analyzed data from the National Health and Nutrition Examination Survey (NHANES), a nationally representative sample of the US civilian non-institutionalized population [12–14]. We included survey waves conducted between 1971 and 2008: NHANES I (1971–1975), NHANES II (1976–1980), NHANES III, phase 1 (1988–1991), NHANES III, phase 2 (1991–1994), and the continuous surveys (1999–2008), which are released in two-year increments. NHANES uses a complex, stratified, multi-stage probability cluster sampling design. We included survey weights in all analyses to correct for oversampling and non-response [2].

We limited the dataset to individuals aged 2–74 years who were born in the United States. We omitted foreign-born respondents because years spent in another country before immigration violate the model's assumption that individuals shared period and birth cohort exposures across their lifespans [15, 16]. We further excluded respondents who had missing measured height or weight (N=9 658, 8.8%) or were pregnant when weighed (N=2 035, 1.8%). In most waves of data collection, pregnancy was defined in females 12 years of age or older by self-report or positive urine test. Our final sample included 94 199 individuals.

Measures

Age, sex, Black-White race, and Hispanic ethnicity were self-reported by the respondent or a respondent's parent for children under age 16 years. Data on Black-White race were available for all survey waves. Therefore, stratified analyses of Blacks and Whites covered the periods from 1971 to 2008. Data on Hispanic ethnicity were not available until 1988. Data on Mexican ethnicity, a subset of Hispanic ethnicity, were available earlier, in 1976. Therefore, we produced analyses stratified on Mexican ethnicity for the survey years between 1976 and 2008 rather than Hispanic ethnicity, which would only cover the survey years between 1988 and 2008. In order to define Blacks and Whites as non-Hispanic, we excluded those with any known Hispanic ethnicity (including Mexican ethnicity) from the Black and White categories. The final stratified samples included 53 809 non-Hispanic Whites, 23 989 non-Hispanic Blacks, and 12 Mexican-Americans.

Obesity status was assessed using body mass index (BMI) calculated from measured height and weight. For respondents aged 20–74 years, obesity was defined as BMI ≥30.0 kg/m^2. For respondents aged 2–19 years, obesity was defined as BMI ≥ the 95th BMI percentile of the sex- and age-specific CDC 2000 standards or BMI ≥ 30 kg/m^2, whichever obesity standard used a lower BMI value for the respondent's sex and age.

Analysis

Age-period-cohort analysis is a family of models that uses information on respondent age, time period of observation, and birth cohort to track the prevalence of health outcomes over time. These models identify unique age effects (the distribution of an outcome across the lifecourse), period effects (secular trends in the prevalence of an outcome that are evident among all age groups in the population), and cohort effects (the aggregation of the outcome among individuals who were born in or around the same year). Cohort effects often reflect differential variation in the effects of an environmental exposure across different ages of the lifecourse [17]. Therefore, cohort effects can sometimes be conceptualized as an interaction of period and age effects [18–20].

Age, period, and cohort effects can be estimated using a variety of statistical techniques [21]. For the present analysis we utilized the median polish technique [5]. The median polish approach explicitly defines cohort effects as interactions of age and period effects. That is, this model assumes that effects of environmental influences vary by age and can be meaningfully estimated as a cohort effects.

To implement the median polish technique, we first created a 15 × 8 contingency table of obesity prevalence. The 15 rows represent 15 five-year age groups, while the eight columns denote eight five-year blocks of calendar time (Figure 1). The diaganols represent 22 birth cohorts. NHANES assessed obesity prevalence during periods of variable timing and duration: 1971–1975, 1976–1980, 1988–1991, 1991–1994, then in continuous 2-year blocks from 1999–2008. Therefore, we approximated seven synthetic 5-year period categories using the NHANES data (Figure 1). Because no NHANES data were available between 1981 and 1988, we interpolated age-specific obesity prevalences for the synthetic period 1981–1985 by averaging age-specific prevalence for the previous (1976–1980) and

subsequent (1986–1990) periods. For stratified analyses of subgroups with relatively small sample sizes and low obesity prevalence, e.g., Mexican-American males, some cells in the contingency tables had obesity prevalence of 0%. For identification purposes, we replaced values of 0% with 1.0% in the initial contingency tables.

Once the contingency table was complete (Table 1), we performed the median polish method by iteratively subtracting the median prevalence value of each row or column from all cells in its respective row or column. This process was repeated until the median values of all rows and columns equaled 0. This process removes the additive period and age effects. The values that remain in the table are non-additive residuals of the period and age effects. The median polish technique interprets these residuals as the sum of cohort effects and random error. Further statistical and conceptual details of the median polish method are given elsewhere [18, 20, 22].

Using the residuals from the contingency table, we used generalized linear regression to estimate cohort effect ratios for each birth cohort. To define mutually exclusive birth cohort categories, we assigned each cell in the contingency table to a synthetic five-year birth cohort category. However, birth year could vary by as much as 9 years for each intersection of a 5-year age and 5-year period category. Therefore, we assigned synthetic five-year cohort categories centered on the median year of the possible 9-year birth range (see Figure 1). We regressed the residuals from the contingency table on these nominal categories of 5-year birth cohort, using 1964–1968 as the reference category.

This analysis compares the birth cohorts of the 1980s with those born before 1980. In particular, we focus on cohort effects estimated for the 1979–1983 and 1984–1988 birth cohorts. We also estimated effects for birth cohorts born after 1988. However, because, these estimates were based on a paucity of data, we do not discuss these more recently born cohorts.

RESULTS

Removing the effects of age and birth cohort through the median polish iterative subtraction, we estimated period, or secular, increases in obesity. Based on visual inspection, we observed similar secular trends to those previously observed using NHANES data for obesity surveillance [2]: secular increases in obesity prevalence were larger in the 1980s and 1990s than the 2000s (Figure 2, Part A), and, in the 2000s, there was little secular change in prevalence among women but continuing secular increases among men (Figure 2, Parts B and C).

Removing the effects of age and period, we isolated non-additive residuals. Based on the regression analyses of the residuals, we found evidence of positive cohort effects for the 1979–1983 and 1984–1988 birth cohorts versus the 1964–1968 cohort: prevalence ratio (PR) = 1.15 (1.04, 1.26) and PR=1.17 (1.06, 1.30), respectively (Figure 3, Part A).

To investigate whether specific demographic groups were driving the cohort effects, we conducted stratified analyses (Figure 3, Parts B and C). In the sex-stratified analysis, cohort

effects were larger for females than males: 1.22 (1.05, 1.41) versus 1.05 (0.93, 1.19) for the 1979–1983 cohort and 1.23 (1.06, 1.44) versus 1.05 (0.92, 1.19) for the 1984–1988 cohort.

In analyses stratified jointly by sex and race-ethnicity, effects were smallest for White males (PRs=1.06 and 0.99) and Mexican-American males (PRs=1.03 and 1.02). Mexican-American females had evidence of modestly increased cohort risk (PRs=1.13 and 1.09) as did White females (PRs=1.17 and 1.15). Black males showed stronger evidence of positive cohort effects (PRs=1.22 and 1.21). Young Black females showed the largest cohort effects: PRs=1.43 and 1.57.

DISCUSSION

We found evidence of positive cohort effects among young adults born in the 1980s. We interpret these cohort effects to mean that these young adults have increased age-specific propensity to be obese than previous generations, even given exposure to a similarly obesogenic environment. These cohort effects have implications for obesity trends over the next 30 years. In the first decade of the 2000s, obesity prevalence stabilized in the U.S. [2]. However, obesity prevalence may resume increasing if contemporary young adults have increased cohort-specific obesity propensity compared to previous generations. In the U.S., obesity prevalence tends to increase with age until age 60. If contemporary young adults are more susceptible, then the peak prevalence of obesity in this group when they reach their 50s and 60s will exceeed that of contemporary 50- and 60-year olds even if the environmental and behavioral influences on obesity do not change over the next three decades.

In the past decade, obesity prevalence stabilized more in U.S. women than men [2]. However, we found that women born in the 1980s may be sensitive to the obesogenic environment than their older female counterparts. On the other hand, we estimated generally small cohort effects in men born in the 1980s, indicating that contemporary young men should be no more sensitive or resistant to the obesogenic environment than their older male counterparts. However, period effects were pronounced for men, indicating that secular environmental influences are driving obesity prevalence higher among all U.S. men, just not differentially by birth cohort.

Ethnic-specific results for U.S.-born Mexican-American men and women were encouraging. In U.S.-born Mexican-Americans, we found stable secular trends and little suggestion of positive cohort effects. In other words, Mexican-American men and women born in the U.S. in the 1980s do not necessarily have greater sensitivity than U.S.-born Mexican-Americans of earlier birth cohorts. However, these findings should be interpreted with caution: because of small sample sizes, estimates for U.S.-born Mexican-Americans are more statistically unreliable than estimates in White and Black Americans.

Estimated cohort effects were greatest for young Black men and women. The stratified results for Black women are especially discouraging. Black women suffer extremely high obesity prevalence, but age-adjusted secular increases in obesity prevalence stopped during the past decade [2]. However, our analysis indicates that Black women born in the 1980s

experience even greater sensitivity to the obesogenic environment to obesity than their older counterparts. Our results also indicate that Black men born in the 1980s are at great risk of rapidly increasing levels of obesity prevalence. Not only does obesity prevalence in Black men born in the 1980s display secular increases similar to that of same-cohort White men, but the contemporary young Black men also show positive cohort effects compared to previous generations of Black men.

Various mechanisms could underlie cohort effects observed in the 1980s birth cohorts. Excess risk for the 1980s birth cohorts may be attributable to longer exposure to the obesogenic environment or more intense exposure at periods critical for physiological and behavioral development. One hypothesis related to critical developmental periods is the developmental overnutrition hypothesis. The hypothesis posits mothers who are obese during gestation produce offspring who are physiologically predisposed to obesity [6, 23]. Because obesity prevalence among women aged 20–40 years, prime childbearing ages, increased 50% between the late 1970s and early 1990s [1], the fetal overnutrtion hypothesis could be a factor in increasing susceptibility for the 1980s birth cohorts. In general, little research has empirically tested the fetal overnutrition hypothesis in adult populations. While our finding of positive cohort effects may support the hypothesis, future research is needed to investigate this hypothesis directly.

In addition, there is evidence that developmental effects on obesity risk could act in a sex-specific manner. For instance, an alternative to the overnutrition hypothesis is the mismatch hypothesis, under which restricted nutrition in utero or during infancy could increase risk of obesity as an adult via a mismatch between an offspring's early-life predictive adaptive response and a later calorically rich environment [9]. There is experimental, epidemiologic, and quasi-experimental evidence that a nutritional mismatch could especially increase obesity risk in females [9, 24]. The fact that U.S. childhood poverty, which is often characterized by perinatal nutritional deprivation and later caloric abundance, increased in the late 1970s and early 1980s [25] could be one explanation for the increase in generational divergence of obesity risk for females versus males in our 1980s cohorts. Further, the experimental literature has identified numerous biological mechanisms by which maternal stress and malnutrition may influence obesity risk in a sex-specific manner: interference with sex hormone signaling; alteration of methylation patterns of genes, including insulin-like growth factor-2; sex-dependent perturbations to the functioning of the placenta; greater "catch-up" growth in infant girls versus boyds; and long-term, sex-specific effects on appetite and weight regulation [26,27, 28].

This the most comprehensive analysis to date of cohort effects among birth cohorts from the 1980s. Because our secular coverage extended to 2008 and we examined obesity across the life course, we have more data on the 1980s cohorts than any previous analysis. Another strength of our analysis is the use of measured height and weight data, which avoids bias from differential self-reporting by sex, race, ethnicity, age, and body size [29–31]. Finally, to our knowledge, this is the first age-period-cohort analysis of any U.S. Hispanic subgroup. These strengths should be considered with the caveat that data on early childhood obesity for the 1980s birth cohorts was interpolated because the NHANES survey was not conducted in the years 1981–1988.

We further note that the results of age-period-cohort analyses are sensitive to the assumptions of the model chosen. We have previously demonstrated in similar data that the conclusion regarding the presence of cohort effects in obesity in the U.S. differs depending on whether researchers use a model that assesses age, period, and cohort as additively associated versus multiplicatively [22]. Recent methodological studies suggest that nonlinear age-period-cohort models offer special advantages in the estimation of age, period, and cohort effects by not restricting the effects to be additive and linearly related to the outcome variable, hence avoing the identification problem [32]. In the present analyses, we conceptualized cohort effects as estimates of generational divergence from the obesity prevalence expected assuming additive age and period effects. Therefore, we employed a model which operationalized that definition by explicitly defining the cohort effect as the interaction between additive age and period effects. However, other models may render different conclusions, an issue common to all age-period-cohort analyses and a topic of ongoing research. Further, previous analyses of NHANES data suggested that there was no cohort effect for obesity prevalence using the median polish approach [22]. The present study used a wider range of data and specifically examined cohort effects within previously uncharacterized subgroups, which illuminated the presence of striking cohort effects for the most recently born cohort of adults, especially young women and Black Americans.

Nevertheless, there are limitations to this analysis. We assessed obesity trends using BMI. Other measures of obesity, such as waist circumference or percent body fat, may yield different results [33]. Second, the unit of analysis in this paper was the prevalence estimate, which was estimated with error from survey data; this error was not incorporated into our final confidence intervals. Therefore, the confidence intervals may overestimate the precision of the cohort effect estimates. Finally, because there were gaps in the data, we extrapolated data for the 1981–1985 period. We believe that this does not bias our results because other data collected during this period show linearly increasing BMI prevalence between the late 1970s and the late 1980s, the years between which we interpolated [7]. Finally, our results only apply to the U.S.-born. Obesity prevalence in immigrants tends to be lower than that in the U.S.-born [34].

Although some investigators argue that obesity did not suddenly begin increasing in the 1980s [33, 35], that decade is generally regarded as the start of a U.S. “obesity epidemic” [1]. The recent deceleration of increases in obesity prevalence is encouraging but could be temporary. The birth cohorts of the 1980s may be more prone to obesity than previous generations, even holding constant age effects and secular influences on obesity. If these cohorts do experience greater age-specific sensitivity to the obesogenic environment, then we may observe a generational divergence of obesity trends: even as age-adjusted obesity prevalence stabilizes in those born before the 1980s, it may continue to rise in the 1980s cohorts. The obesity epidemic of the 1980s could ripple through the population again as this generation of young adults ages, culminating in an new high in obesity prevalence in the 2030s as this generation reaches their 50s.

These findings have implications for obesity research and public health practice and policy. Research into the causes of deceleration in obesity increases should investigate the 1980s birth cohorts separate from older adults. There is evidence that obesity rates have continued

to rise in the 1980s birth cohorts even in countries in which age-adjusted adult obesity prevalence has stabilized [3]. Public health policy and practice must avoid becoming complacent in efforts to prevent excess weight gain. In fact, it may be necessary to create new initiatives targetted at preventing excess weight gain among young adults in their 20s and 30s. Policy, cultural, or behavioral changes that are effective enough to stablize age-adjusted obesity prevalence in generations born before 1980 may not be effective in stabilizing obesity prevalence in those born after 1980. To merely stabilize age-adjusted obesity prevalence in those born in the 1980s may require targeted cohort-specific interventions or widespread interventions powerful enough to substantially decrease obesity in older birth cohorts. Much like initiatives targetted to prevent childhood obesity, public health initiatives and policies may be warranted to prevent incident obesity and further weight gain among contemporary young adults of the 1980s birth cohort.

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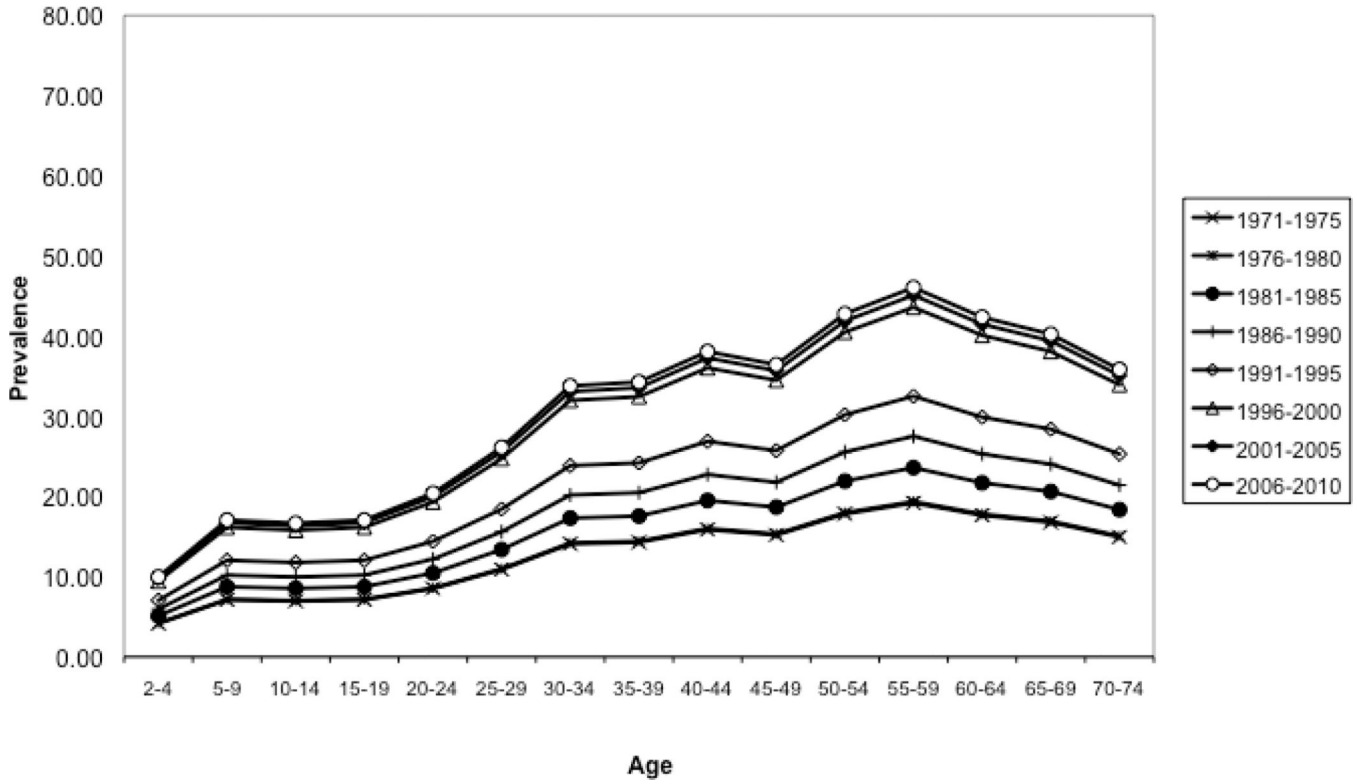
	NHANES periods	1971-1975	1976-1980		1989-1991	1991-1994	1999-2000	2001-2004	2005-2008
NHANES ages (years)	Synthetic periods & ages	1971-1975	1976-1980	1981-1985	1986-1990	1991-1994	1996-2000	2001-2005	2006-2010
2-4	0-4	1969-1973	1974-1978		1984-1988	1989-1993	1994-1998	1999-2003	2004-2008
5-9	5-9	1964-1968	1969-1973		1979-1983	1984-1988	1989-1993	1994-1998	1999-2003
10-14	10-14	1959-1963	1964-1968		1974-1978	1979-1983	1984-1988	1989-1993	1994-1998
15-19	15-19	1954-1958	1959-1963		1969-1973	1974-1978	1979-1983	1984-1988	1989-1993
20-24	20-24	1949-1953	1954-1958		1964-1968	1969-1973	1974-1978	1979-1983	1984-1988
25-29	25-29	1944-1948	1949-1953		1959-1963	1964-1968	1969-1973	1974-1978	1979-1983
30-34	30-34	1939-1943	1944-1948		1954-1958	1959-1963	1964-1968	1969-1973	1974-1978
35-39	35-39	1934-1938	1939-1943		1949-1953	1954-1958	1959-1963	1964-1968	1969-1973
40-44	40-44	1929-1933	1934-1938		1944-1948	1949-1953	1954-1958	1959-1963	1964-1968
45-49	45-49	1924-1928	1929-1933		1939-1943	1944-1948	1949-1953	1954-1958	1959-1963
50-54	50-54	1919-1923	1924-1928		1934-1938	1939-1943	1944-1948	1949-1953	1954-1958
55-59	55-59	1914-1918	1919-1923		1929-1933	1934-1938	1939-1943	1944-1948	1949-1953
60-64	60-64	1909-1913	1914-1918		1924-1928	1929-1933	1934-1938	1939-1943	1944-1948
65-69	65-69	1904-1908	1909-1913		1919-1923	1924-1928	1929-1933	1934-1938	1939-1943
70-74	70-74	1899-1903	1904-1908		1914-1918	1919-1923	1924-1928	1929-1933	1934-1938

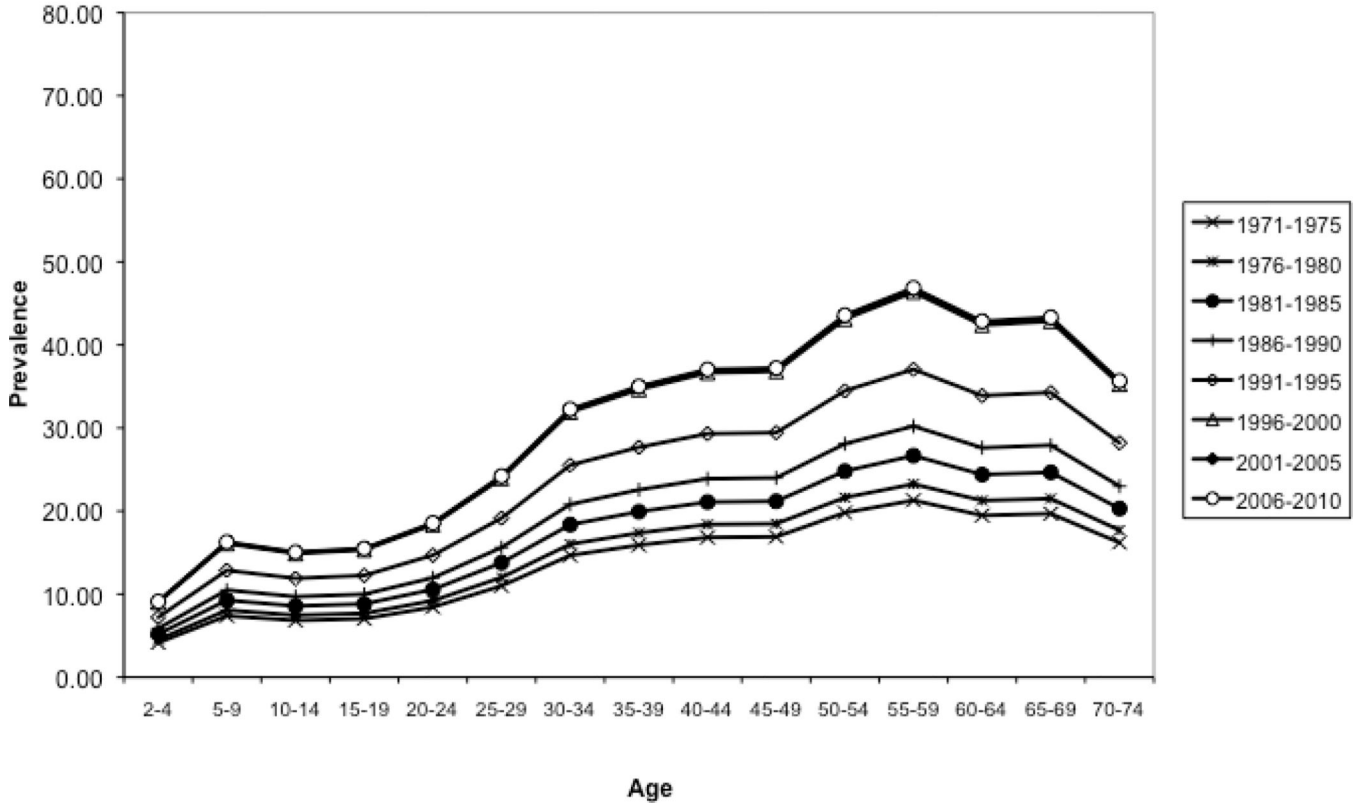
Figure 1.

Graphical representation of the construction of 22 synthetic birth cohorts using data from the National Health and Nutrition Examination Survey (NHANES), 1971–2008. The diagonals represent distinct birth cohorts.

2A



2B



2C

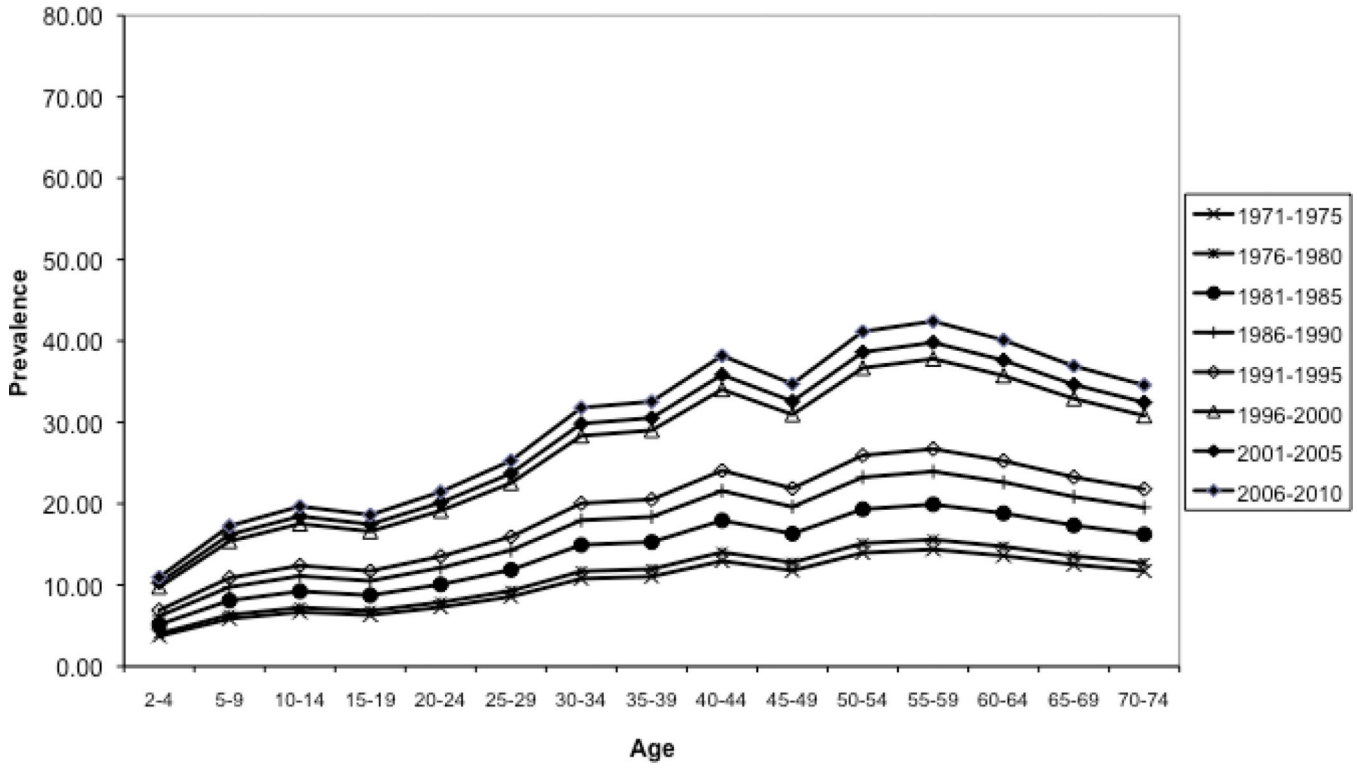


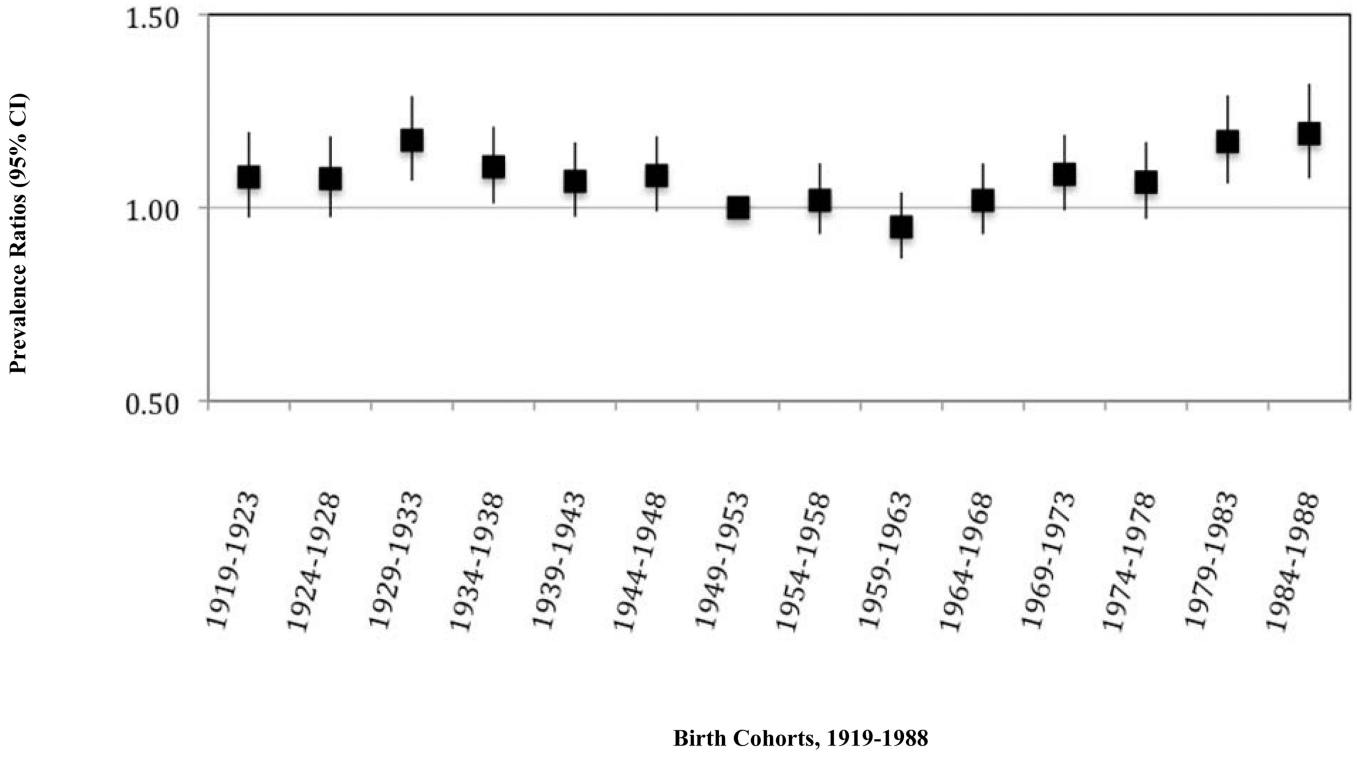
Figure 2.

A: Period effects on obesity prevalence in overall samplestratified by 5-year synthetic age groups, U.S.-born respondents to the National Health and Nutrition Examination Survey (NHANES), 1971–2008

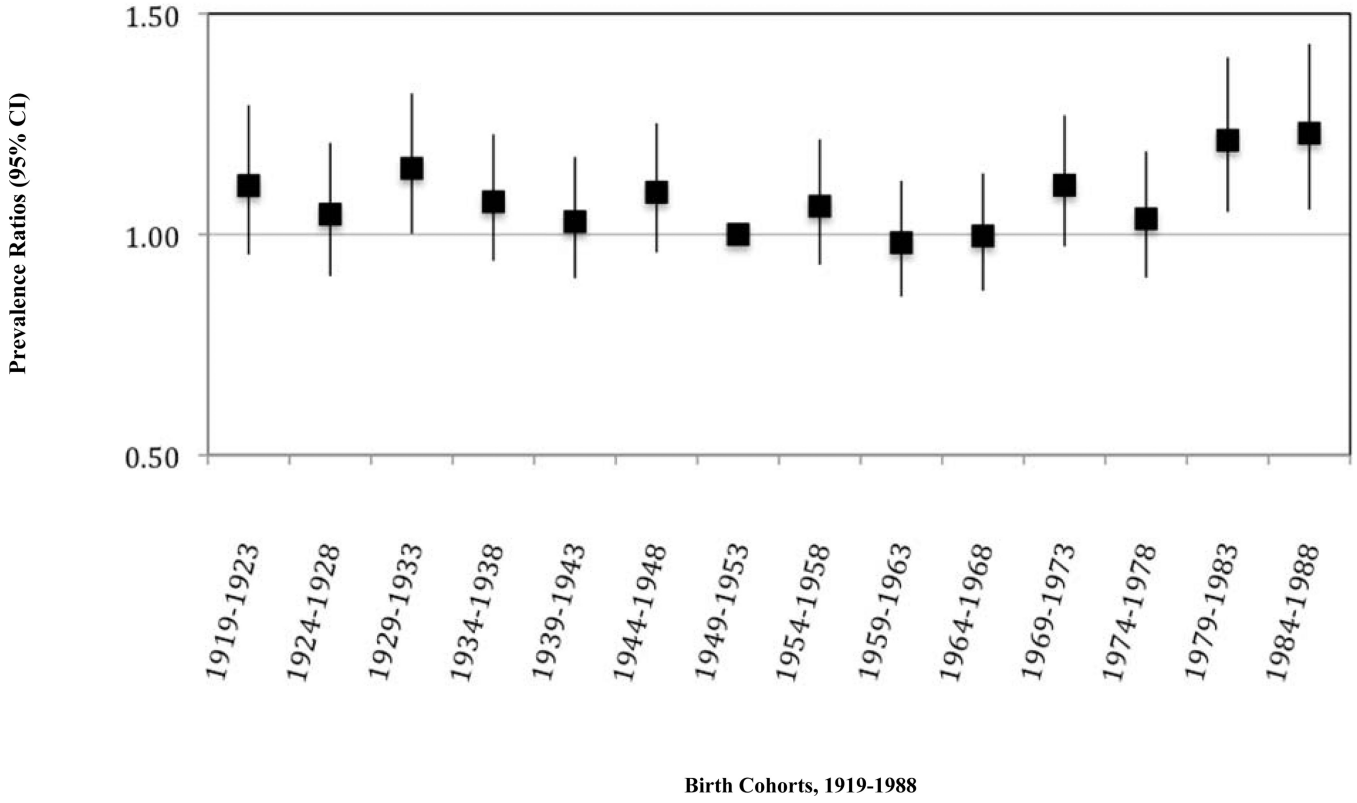
B: Period effects on obesity prevalence in females stratified by 5-year synthetic age groups, U.S.-born respondents to the National Health and Nutrition Examination Survey (NHANES), 1971–2008

C: Period effects on obesity prevalence in males stratified by 5-year synthetic age groups, U.S.-born respondents to the National Health and Nutrition Examination Survey (NHANES), 1971–2008

3A



3B



3C

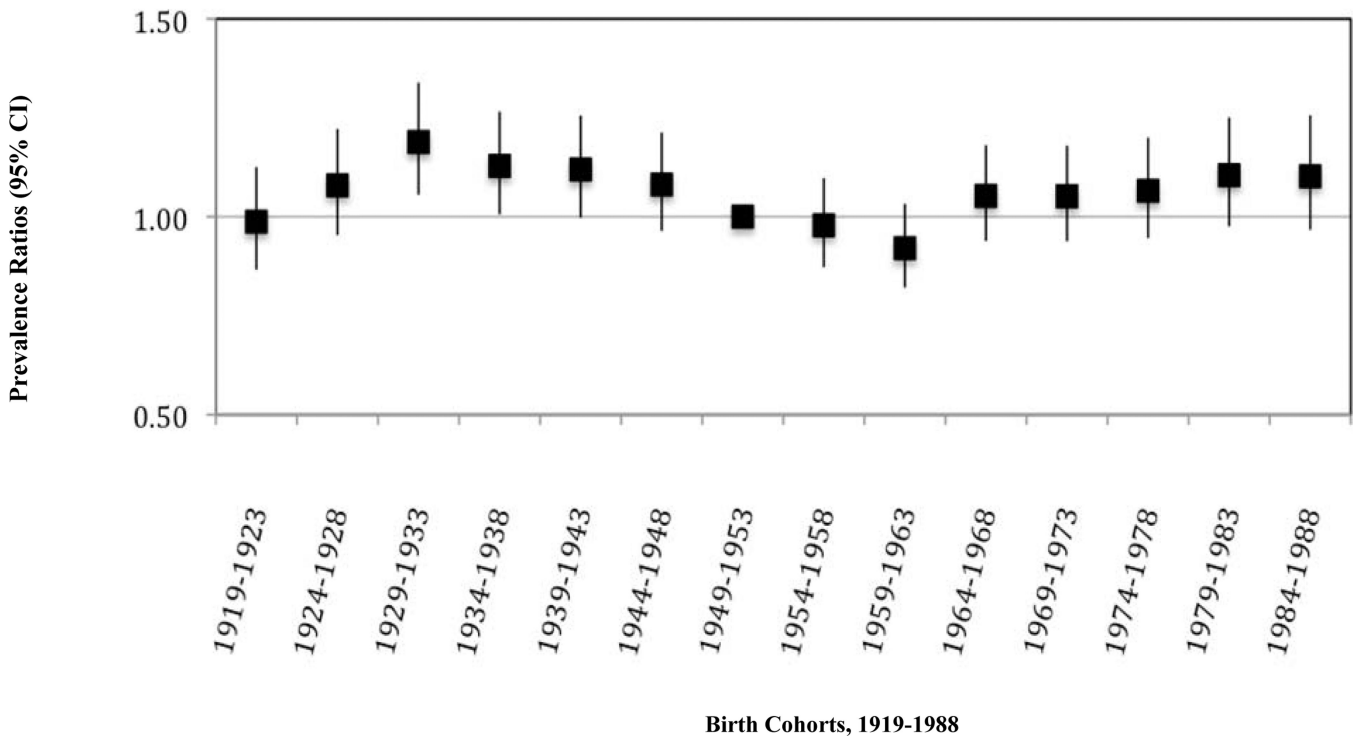


Figure 3.

A: Birth cohort effects on obesity prevalence in overall sample, U.S.-born respondents to the National Health and Nutrition Examination Survey (NHANES), 1971–2008

B: Birth cohort effects on obesity prevalence in females, U.S.-born respondents to the National Health and Nutrition Examination Survey (NHANES), 1971–2008

C: Birth cohort effects on obesity prevalence in males, U.S.-born respondents to the National Health and Nutrition Examination Survey (NHANES), 1971–2008

Table 1

Contingency table of obesity prevalence arrayed by age and period, U.S.-born respondents to the National Health and Nutrition Examination Survey (NHANES), 1971–2008[1]

	NHANES periods	1971–1975	1976–1980		1989–1991	1991–1994	1999–2000	2001–2004	2005–2008
NHANES ages (years)	**Synthetic ages and periods**	**1971–1975**	**1976–1980**	**1981–1985**	**1986–1990**	**1991–1995**	**1996–2000**	**2001–2005**	**2006–2010**
2–4	0–4	4.54	3.37	4.83	6.29	6.12	9.95	10.15	9.59
5–9	5–9	4.24	7.51	8.85	10.19	13.27	14.78	16.71	15.59
10–14	10–14	5.57	6.89	8.23	9.56	13.08	16.06	18.74	19.92
15–19	15–19	6.23	5.12	8.07	11.02	11.70	17.14	17.61	17.57
20–24	20–24	6.46	7.00	9.92	12.84	12.63	20.77	26.77	25.79
25–29	25–29	10.66	10.46	11.94	13.42	18.82	27.89	26.73	27.20
30–34	30–34	14.17	14.12	18.49	22.86	20.34	31.73	27.68	34.33
35–39	35–39	14.66	15.18	17.50	19.82	23.09	29.28	33.88	34.39
40–44	40–44	16.98	16.25	19.50	22.75	25.66	32.96	36.20	38.68
45–49	45–49	14.99	18.45	19.24	20.02	29.66	32.78	36.16	35.34
50–54	50–54	17.69	17.88	22.58	27.28	36.91	40.75	35.28	39.71
55–59	55–59	19.16	19.40	26.52	33.64	33.21	35.90	38.58	41.06
60–64	60–64	18.67	17.77	20.90	24.03	30.39	41.69	39.81	42.48
65–69	65–69	16.78	19.55	21.79	24.02	28.32	41.21	37.06	36.97
70–74	70–74	17.42	16.27	18.68	21.08	25.43	30.28	33.62	35.67

[1] Obesity was calculated using body mass index (BMI) from measured height and weight. Respondents were classified as obese if they (1) were aged 20–74 years and had BMI ≥30.0 kg/m^2 or (2) were aged 2–19 years and had BMI ≥ the 95th percentile of the sex- and age-specific CDC 2000 standards or BMI ≥30.0 kg/m^2.