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Decomposing Trends in Child Obesity

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Figures: 5

Abstract

We unravel the absolute level and relative prominence of two demographic processes that are relevant for childhood obesity, and that will ultimately determine the long-term course and pace of change in child obesity rates. We leverage data from the National Health and Nutrition Examination Survey (NHANES) to decompose change in child obesity from 1971 to 2012. We partition change into that attributable to (1) healthier, more nutritionally and economically advantaged cohorts in the population being replaced by cohorts of children who are less advantaged (between-cohort change), and (2) the health habits, nutrition, and social and economic circumstances of all cohorts of children worsening over time (within-cohort change). The rise in obesity among children aged 2 to 19 years old is solely due to intracohort change driven by variation in food security composition and in the diet of the population over time. Child obesity in the population rose largely because of individual increases in weight status that are broadly distributed across age and cohort groups. Smaller but significant cohort replacement effects slightly attenuated these intracohort change effects over the study period, leading to a more gradual increase in obesity. Our results provide some reasons for optimism. Given that population estimates of child obesity rose because the typical member of all cohorts became heavier over time at all stages of the early life course, successful policy and health interventions that focus on changing health habits across all ages and generations have the potential to quickly slow or reverse the upward trend in child obesity.

Key Words: Child Obesity; Child Health & Development; Population Health; Health Policy

Introduction

Child obesity is an important public health challenge. The most recent data indicate that approximately 18.5% of 2–19 year olds are obese in the Unites States alone (Hales *et al*. 2017). Although much research is devoted to describing the changing trends in child obesity (Fryar *et. al*. 2014; Ogden *et al*. 2016), our understanding of the underlying mechanisms leading to the rise in child obesity remain obscured (see Papoutsi *et al*. 2013, for review). For example, most population-level studies on child obesity, and this is true for official reports of obesity statistics, are perceived to be, measured as, and presented as a period phenomenon (Ogden *et al*. 2016; Han 2010. In other words, we tend to look at population averages for each year to identify how trends are growing over time without focusing on the proximate mechanisms actually driving the change (see Fu and Land's 2015 analysis on youth in China for an important exception). While scholars heavily point to an imbalance in energy consumed to that expended to explain the increases in obesity among children (Han 2010), emerging evidence suggests that changes in early childhood factors are particularly relevant for explaining the increases in child obesity (see Woo Baidal *et al*. 2016, for review).

Notably, many point to extremes in socioeconomic inequality as a potential explanation for the rise in child obesity (Frederick *et al*. 2014). Others propose that increases in food insecurity (Eisenmann *et al*. 2011) and changes to family structure (Miller and Chang 2015) are larger contributors to the growth. Still, the overwhelming majority of existing studies point to changes in consumption practices as the driving force behind the increasing prevalence and severity of obesity in children (Swinburn *et al*. 2011; Taubes 2016).

Missing from these studies, however, is recognition of the fact that changes in early life factors can affect the childhood obesity rate in two very different ways. On one hand, the obesity

rate can increase because more recent birth cohorts of children experience higher rates of socioeconomic inequality, food insecurity, and worse nutritional practices than did previous cohorts. That is, healthier, more nutritionally and economically advantaged cohorts are replaced by cohorts of children who are less advantaged (i.e., cohort replacement). If demographic turnover in the child population is responsible for the growth of child obesity, then the rise in obesity rates should be concentrated among more recent cohorts, and the increase in obesity over time should be gradual.

On the other hand, rates in child obesity may rise because the health habits, nutrition, and social and economic circumstances of all cohorts of children worsen over time. In this scenario, child obesity increases because all cohorts of children encounter a more obesogenic environment (i.e., intracohort change). If intracohort change is driving the rise in child obesity, then we should see change happen more quickly, with obesity distributed broadly among all cohorts and age groups of children. If intercohort and intracohort change reinforce one another, we should observe rapid changes in the child obesity rate over a relatively short period of time.

Unraveling the absolute level and relative prominence of these two demographic processes will help us to answer two important questions. First, why is it that the child and adolescent obesity rates continued to increase until 2003/04 then decreased among children 2 to 5 years old, increased among children 6 to 11 years old until 2007/08 and then plateaued, and increased among adolescents aged 12 to 19 years old (Ogden *et al*. 2016)? Second, which health, nutritional, and social and economic changes drove within- and between-cohort change in child obesity? To answer these questions, we pool 10 cross sectional surveys over 4 decades. We first generate our own estimates of the increase in child obesity over a 41-year period. Then, we track how different cohorts experience obesity differently over time by age and the year they were

born. Finally, we use algebraic decomposition techniques to separate and quantify how much of the change in obesity is due to cohort replacement and intracohort change processes.

Methods

Data Sources

We base our analyses on ten waves (1971/75, 1976/80, 1988/94, 1999/00, 2001/02, 2003/04, 2005/06, 2007/08, 2009/10, 2011/12) of the National Health and Nutrition Examination Survey (NHANES), a cross-sectional survey of the civilian, non-institutionalized U.S. population. NHANES uses a complex multistage probability sampling design, with oversampling of smaller racial/ethnic subgroups. Analyses were estimated using NHANES sampling weights to account for differences in the chances of selection and non-response.

Study Population

Models were restricted to 2–19 year old children[1] with a completed body measurement component. We excluded 5,403 cases (15.98%) due to missing height or weight measures. Pregnant females were also excluded. The total sample size across all years is 33,806 participants. Sample sizes by survey wave are as follows: 1971/75, n=2,117; 1976/80, n=2,073; 1988/94, n=5,816; 1999/00,, n=2,755; 2001/02 n=3,722; 2003/04, n=3,451; 2005/06, n=3,650; 2007/08, n=3,309; 2009/10, n=3,607; 2011/12, n=3,306.

Outcome Variable

The outcome of interest is an age- and sex-specific dichotomous variable indicating whether or not the child is obese. We calculated body mass index (BMI) from height and weight measures using the standard formula (weight [kg/height [m]^2) based on the BMI-for-age-growth

[1] We follow the standard age cut offs provided by the Centers for Disease Control (Ogden *et al*. 2016).

charts for boys and girls (Kuczmarski 2002). Children were coded as being obese if they had a BMI ≥95th percentile.

Covariates

We included covariates to represent child demographic, nutrition, family, and socioeconomic factors known to be associated with children's weight-related outcomes. Demographic characteristics include continuous measures of survey year and birth year, and categorical measures of gender (1 = boy) and race (1= non-Hispanic white). Nutritional components are child- and/or proxy-reported depending on the age of the study participant, and include dichotomized age- and sex-adjusted total intake of energy consumption (1 = normal kcals), fat (1 = ≤ 65gm), carbohydrates (1 = ≤ 24gm), protein (1 = ≤ 45 gm), and sodium (1 = ≤ 1,500 mg) based on daily caloric recommendations for a moderately active child in a 24-hour period (U.S. Department of Health and Human Services & U.S. Department of Agriculture 2015), see Moshfegh et al. (2008) for more details on the NHANES dietary data. Family and socioeconomic characteristics include a categorical measure of parental marital status (1 = married), a continuous measure of household income measured in thousands of dollars, a categorical measure of educational attainment (1 = college graduate), and a dichotomized measure of household food security (1 = food secure).

Statistical Analysis

We began our analysis by describing the overall trends in obesity rates for boys and girls across the age distribution. We then examine age-specific trends in child obesity rates by birth cohort (1950-1959, 1960-1969, 1970-1979, 1980-1989, 1990-1999, and 2000-2009). These trend analyses allow us to visually evaluate the roles of intra- and inter-cohort change on the rise in child obesity.

To better understand intra- and inter-cohort change and the roles of changes in child demographic, nutrition, family, socioeconomic, and food security factors over time on the child obesity rate, we regressed obesity on birth year, survey year, and the covariates using logistic regression. We estimated both an unconditional model that only included cohort (birth year) and period (survey year) and a series of conditional models that included the covariates to assess the impacts of child demographic, nutrition, family, socioeconomic, and food security factors on the estimated coefficients for cohort and period. We present estimates for the final model that includes all covariates because results from the full model paralleled the domain specific model. Prior to estimating the final models, we tested for non-linearity in the effects of the period and cohort variables by including second- and third-order polynomial terms for survey and birth year. These tests indicated that the effect of birth year is linear and the effect of survey year is quadratic.

Finally, we decomposed the sources of aggregate change in the obesity rate from 1971 through 2012 using Kitagawa's (1955) algebraic decomposition technique. With this approach, birth year is collapsed into cohort subgroup categories (Firebaugh 1989), and change in the obesity rate between survey wave $k+l$ and k is partitioned into those parts attributable to cohort replacement, individual change, and the joint effects of inter- and intra-cohort change. We used this decomposition approach because it does not assume linearity, which our preliminary investigations found is not a valid assumption in this case. We decomposed change for the latter period that included as many cohorts as possible within narrowly spaced time points (2001/02-2011/12).

In order to evaluate the role that changes in child demographic, nutrition, family, socioeconomic, and food security factors over time have on changes in child obesity rates and for

the relative effects of inter- and intra-cohort change, we applied two different sets of analysis weights in the algebraic decomposition. In the unstandardized algebraic decomposition, we used wave-specific 2-year full sample NHANES sampling weights for both interviewed and Mobile Examination Center (MEC) examined participants in the descriptive and logistic regression analyses. In the standardized algebraic decomposition, we applied post-stratification weights that reweighted the NHANES sample so that the weighted distributions on the covariates for each wave were identical to those for the 1971 NHANES sample. Analyses were performed using SAS 9.4 software (2018; SAS Institute Inc., Cary, NC, USA).

Results

Cross-Cohort Trends in Obesity

Child obesity increased nearly 14 percentage points between 1971 and 2012 (Table 1). Figure 1 displays historical trends in obesity for this period, both overall and separately for boys and girls. Obesity rates have varied considerably across the 41-year period. A brief rise in 2003/04 was followed by a sudden drop in 2005/06 before a plateau occurred in 2007/08. Although obesity rates have moderated considerably since the early 2000s, between the early 1970s and the early 2010s the obesity rate nearly tripled, rising from 8% to 22%. While trends in obesity for boys and girls parallel one another, rates are generally higher for boys than for girls in the early waves. By the early 2010s, the obesity rates for boys and girls converged – with the rate for girls increasing between 1971/75 and 2011/12 from 7% to 22% and the rate for boys rising from 9% to 22%.

<Table 1 & Figure 1>

The Correlates of the Rise in Child Obesity

Figure 2 displays odds ratios for the fully specified regression model. The odds of child obesity increased by 15% for each one-year increase in the survey period, and each one-year increase in birth year is linked to a 12% decrease in the odds of obesity (both $p < 0.001$). As we move from top to bottom of Figure 2, we see that child demographics characteristics have little influence on the odds of child obesity (all p-values > 0.05). By contrast, childhood obesity is associated with caloric consumption. In line with existing studies (Wang et al. 2006), high-energy consumption is estimated to increase the odds of obesity by 22% ($p = 0.02$), relative to children who consume the recommended number of calories per day. Continuing to move down the figure, we see that parental marital status and education are also linked to an increase in the odds of obesity. The odds of obesity for children whose parents are widowed, divorced, or separated are 9% higher than children whose parents are married. Compared to children whose parents are college graduates, the odds of obesity are 37% higher among children whose parents did not complete high school ($p < 0.001$), 28% higher for children whose parents graduated from high school ($p < 0.001$), and 24% higher for children whose parents attended some college ($p < 0.001$). We see that food security does not play a prominent role in the odds of obesity. Results of the logistic regression model indicate that calories consumed ($p = 0.02$), parent's marital status ($p <0.01$) and educational attainment (all p-values < 0.001) matter most for child obesity across this 41 year period, and that odds of obesity have risen over time (i.e., survey waves) but declined across birth cohorts.

<Figure 2>

The Role of Inter- and Intra-Cohort Change in the Rise in Child Obesity

Two very different processes of change may drive the significant rise in child obesity. It could be that obesity rates increased because more recent birth cohorts are exposed to poorer social, economic, and health conditions than previous generations of children including higher rates of food insecurity or worsening dietary practices (i.e., CR). Conversely, it is also possible that obesity rates rose because the health habits and social and economic circumstances of all cohorts of children worsened over time (i.e., IC).

In Figure 3 we present obesity rates by age for the cohorts in our study with sufficient sample sizes ($n \geq 50$). We do this as a first step to determine the relative importance of IC and CR on the rise in obesity. For example, if IC is the principal driver of the increase in obesity then age-specific rates for all cohorts will increase after childhood obesity rates began to increase in the early 1980s and these age-specific rate increases will be greater for more recent than earlier cohorts (i.e. age slopes will be greater for later cohorts). If, on the other hand, CR is central to this increase, obesity rates for more recent cohorts will be higher than for previous cohorts throughout childhood (i.e., age intercepts will be higher for more recent cohorts). If both IC and CR are important, not only will obesity rates be higher for more recent cohorts but obesity will rise more sharply as cohort members age (i.e., age intercepts and slopes will be higher for later cohorts).

< Figure 3>

Figure 3 indicates that both IC and CR played significant roles in the rise in obesity from the early 1970s to late 2010s. Consistent with the CR explanation, more recent cohorts have higher rates of obesity than earlier cohorts throughout childhood. For example, 4% of 2–4 year olds born between 1970 and 1979 were obese, whereas 10% of 2–4 year olds born between 1990

and 1999 were obese. At the other end of the age continuum, the obesity rate for 17–19 year olds rose from 7% for the 1960-1969 cohort to 20% for the 1980-1989 cohort.

Consistent with the IC explanation, obesity rates also increase over the course of childhood for nearly all cohorts until early adolescence, and the rate of increase is greater for more recent cohorts. The obesity rate for the 1960-1969 cohort increased by 2.7 points (from 2.7% at ages 2–4 to 5.4% at ages 17) while the rate for the 1970-1979 cohort rose by 9.1 points (from 3.5% to 12.6%), the rate for the 1980 to 1989 cohort rose by 13.5 points (from 7.1% to 20.7%), and the rate for the 1990 to 1999 cohort increased by 14.2 points (from 9.4% to 33.9%).

To identify which compositional differences between cohorts are most important for child obesity, we computed total change (TC = IC + CR) for models that entered demographic, nutritional, family, socioeconomic, and food security measures separately. Accounting for changes in the population composition may affect the relative contribution of TC. For example, if more recent cohorts have higher rates of obesity than did previous cohorts because they had poorer diets, controlling for changes in nutrition would attenuate or eliminate the influence of CR on TC. Figure 4 compares estimates of total change in rates of obesity for the unconditional rate that does not adjust for compositional differences to a series of rates that adjusted for demographic, nutritional, family, socioeconomic, and food security differences among the time periods. If changes in the population composition matter, adjusting for these differences will attenuate total change. Our interest here is how much each bar shrinks when adding different sets of measures.

< Figure 4>

As shown in Figure 4, compositional changes by-and-large do not explain the increase in obesity over the study period. Adjustment for all factors (bottom bar) increased TC in obesity

from 6.7 to 8.0. Changes in the demographic and socioeconomic composition of the population are not responsible for higher obesity rates, nor are between-cohort differences in obesity attributable to changes in family structures and food security. However, changes in diet and nutrition played a more prominent role, accounting for 18.2% of the uptick in obesity.

Figure 5 displays the percentages of total change in child obesity due to IC and CR for unadjusted and adjusted models. Figure 5 shows that the sharp increase in rates of child obesity in the U.S. over the last decade is attributable to within-cohort change (i.e., individual change). In fact, population turnover has slightly attenuated this increase in childhood obesity. That is, more recent cohorts – net of the overall rise in obesity over the study period – are slightly less obese than earlier cohorts of children.

< Figure 5>

The top bar in Figure 5 shows the percentages of overall change in obesity attributable to IC (black) and CR (gray), if compositional differences among the time periods are ignored. In the unadjusted model, IC accounts for 105% of the overall change in the obesity rate. This means that all of the increase is due to changes happening within (and not between cohorts). In fact, CR is actually lowering the obesity rate a bit (5%), leading to a more gradual increase. Rates in obesity rose solely because the typical member of all cohorts became heavier over time. The strong IC effects may be due to a number of things including changing nutritional habits. In the next set of rate decompositions, we adjust for temporal changes in the population in terms of demographic, nutritional, family, and food security characteristics. The intention of including the adjustments is not to analyze the extent of these associations with obesity but to see whether these change the relative influence and magnitude of CR or IC effects.

Adjustment for sociodemographic and family characteristics has little impact on estimates of either CR or IC. Changes in the sociodemographic and family characteristics of the population of children over time are neither responsible for the overall increase in childhood obesity nor the strong IC effects. Adjusting for food insecurity and nutrition, respectively, strengthens the large, positive IC effect, while simultaneously boosting the moderating effect of CR on total change in the obesity rate. If levels of food insecurity and nutrition remained as they were in the early 1970s, IC over the study period would be even larger.

Discussion

Currently, one sixth of children are obese in the United States (Hales *et al*. 2017). Often, researchers narrowly focus on the fluctuating trends in child obesity (Fryar et al. 2014; Ogden et al., 2016) at the expense of the underlying sources of change. While these analyses provide critical information on the changing patterns of prevalence, they do little to draw out the proximate mechanisms that led to the rise in child obesity and that will ultimately determine whether high rates persist. If, for example, the increases are largely a consequence of younger, less obese cohorts replacing older, more obese cohorts (i.e., cohort replacement), gradual increases in obesity will be concentrated among more recent cohorts and younger children. Conversely, if intracohort change is the primary driver of the uptick in child obesity, change will happen more quickly and be distributed more broadly among all birth cohorts and age groups.

Disentangling these two processes of change helps us to get at two important things. First, identification of the underlying mechanisms producing the steep rise in child obesity is the initial step to locating the underlying causes of the increase. Second, uncovering which process of change is driving the growth will aid policy makers in their development of strategies and educational campaigns to reduce obesity. If the rising rates are primarily a consequence of

population turnover, efforts should focus on younger children and youth since changes are happening between and not within cohorts. However, if the increase is primarily a result of intracohort change than policy efforts should focus on all age groups since all children are becoming more obese over time. We find that the rise in obesity among children aged 2 to 19 years old is solely due to intracohort change. Consequently, our results provide some reasons for optimism.

Given that population estimates of child obesity rose due to individual increases in weight status across all ages and cohort groups (i.e., intracohort change), successful social, dietary, medical, or policy interventions have the potential to quickly slow or reverse the upward trend— but only if efforts are approached in the right way. Policy and health interventions should focus on changing health habits across all children and target all ages and generations since the increases in obesity are happening at all stages of the early life course.

Although we are the first to show that increases in child obesity are primarily a consequence of intracohort change, our study is not without limitations. The present analysis is constrained by the rudimentary measures available in the NHANES data across a 41-year period, thereby limiting the scope of our analysis. For example, we know that Latino children have higher odds of obesity (Chen et al. 2016), and that this effect varies by level of acculturation (Frisco et al. 2016), but we lack access to this information in the early years of the NHANES data. Relatedly, we dichotomized measures of nutritional intake to normal/ high ranges due to a lack of variation across children, likely masking more nuanced patterns. Finally, we do not link individual- and family-level data to neighborhood measures despite a growing body of evidence that explicitly links neighborhood characteristics to child obesity outcomes (Kranjac et al. 2019).

Despite these limitations, we take a much-needed first step in the identification of underlying mechanisms driving the rise in child obesity.

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Conflict of Interest: The authors report no conflicts of interest.

Table 1: Weighted Means and Standard Errors for Independent and Dependent Variables for Children Aged 2-19 years old, 1971-2012 NHANES; N = 33,806									
	1971-2012		1971		2012		Δ2012-1971	
Dependent Variable	Mean	Std.Err.	Mean	Std.Err.	Mean	Std.Err.		
Obesity	0.19	0.00	0.08	0.01	0.22	0.01	0.14***	0.14
Independent Variables								
Child Demographic								
Age								
Birth Year	1992	0.24	1960	0.20	2002	0.25	42.00***	42.00
Survey Year	2003	0.19	1972	0.13	2011	0.18	39.00***	39.00
Sex								
Male	0.51	0.00	0.51	0.01	0.51	0.01	0.00	0.00
Female*	0.49	0.00	0.49	0.01	0.49	0.01	0.00	0.00
Race								
Non-Hispanic white*	0.28	0.01	0.86	0.01	0.15	0.02	-0.71***	-0.71
Non-Hispanic black	0.08	0.00	0.14	0.01	0.08	0.02	-0.06***	-0.06
Other Race	0.64	0.01	0.01	0.00	0.77	0.02	0.76***	0.76
Nutrition								
Energy (kcal)								
Normal*	0.53	0.01	0.50	0.01	0.53	0.01	0.03	0.03
High	0.47	0.01	0.50	0.01	0.47	0.01	-0.03	-0.03
Fat (gm)								
≤65*	0.49	0.01	0.64	0.01	0.54	0.01	-0.10***	-0.10
>65	0.51	0.01	0.36	0.01	0.46	0.01	0.10***	0.10
Carbohydrates (gm)								
≤130*	0.88	0.01	0.90	0.01	0.82	0.01	-0.08***	-0.08
>130	0.12	0.01	0.10	0.02	0.18	0.02	0.08***	0.08
Protein (gm)								
≤45*	0.73	0.01	0.82	0.00	0.68	0.01	-0.14***	-0.14
>45	0.27	0.01	0.18	0.00	0.32	0.01	0.14***	0.14
Sodium (mg)								
≤1,500*	0.83	0.01	0.71	0.00	0.79	0.01	-0.08***	0.08
>1,500	0.17	0.01	0.29	0.00	0.21	0.01	0.08***	-0.08
Family Characteristics								
Parent Marital Status								
Married*	0.73	0.01	0.84	0.01	0.74	0.02	-0.10***	-0.10
Widowed/Divorced/Separated	0.16	0.00	0.01	0.01	0.14	0.01	0.13***	0.13
Not married	0.11	0.01	0.15	0.03	0.12	0.02	-0.03**	-0.03
Socioeconomic Status								
Household Income	70.52	1.45	32.16	1.05	78.18	4.83	46.02***	46.02
Parent Education								
Less than High School	0.22	0.01	0.37	0.02	0.22	0.02	-0.15***	-0.15
Highschool Graduate	0.26	0.01	0.38	0.01	0.19	0.02	-0.19***	-0.19
Some College	0.29	0.01	0.20	0.01	0.30	0.02	0.10***	0.10
College Graduate *	0.23	0.01	0.05	0.01	0.29	0.03	0.24***	0.24
Household Food Security								
Food Secure*	0.84	0.01	0.75	0.01	0.79	0.02	0.04**	0.04
Non-Food Secure	0.16	0.01	0.25	0.01	0.21	0.02	-0.04**	-0.04
Source: NHANES 1971-2012								
*Note: * Indicates Reference Group*								
Note: Astericks indicate significant change evaluated using two-tailed independent means t-test Δ2012-1971								

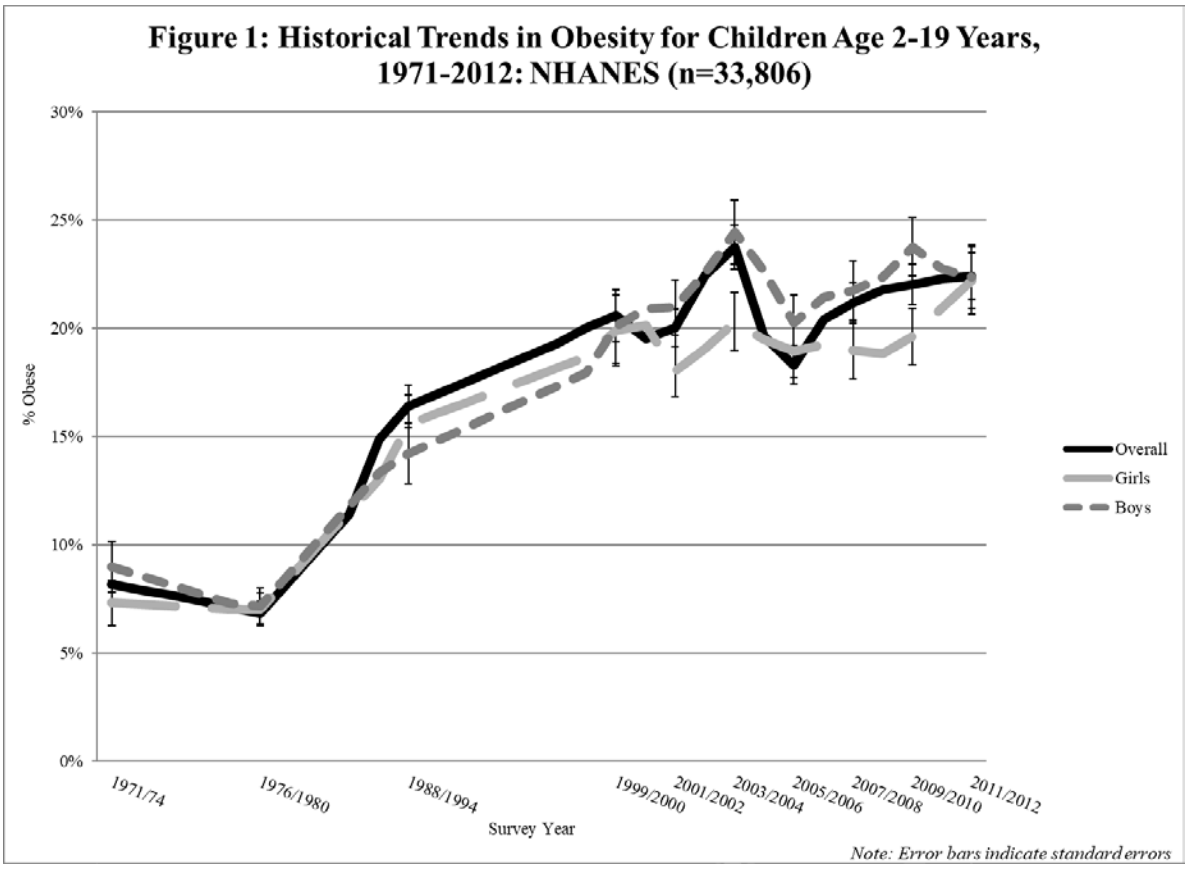
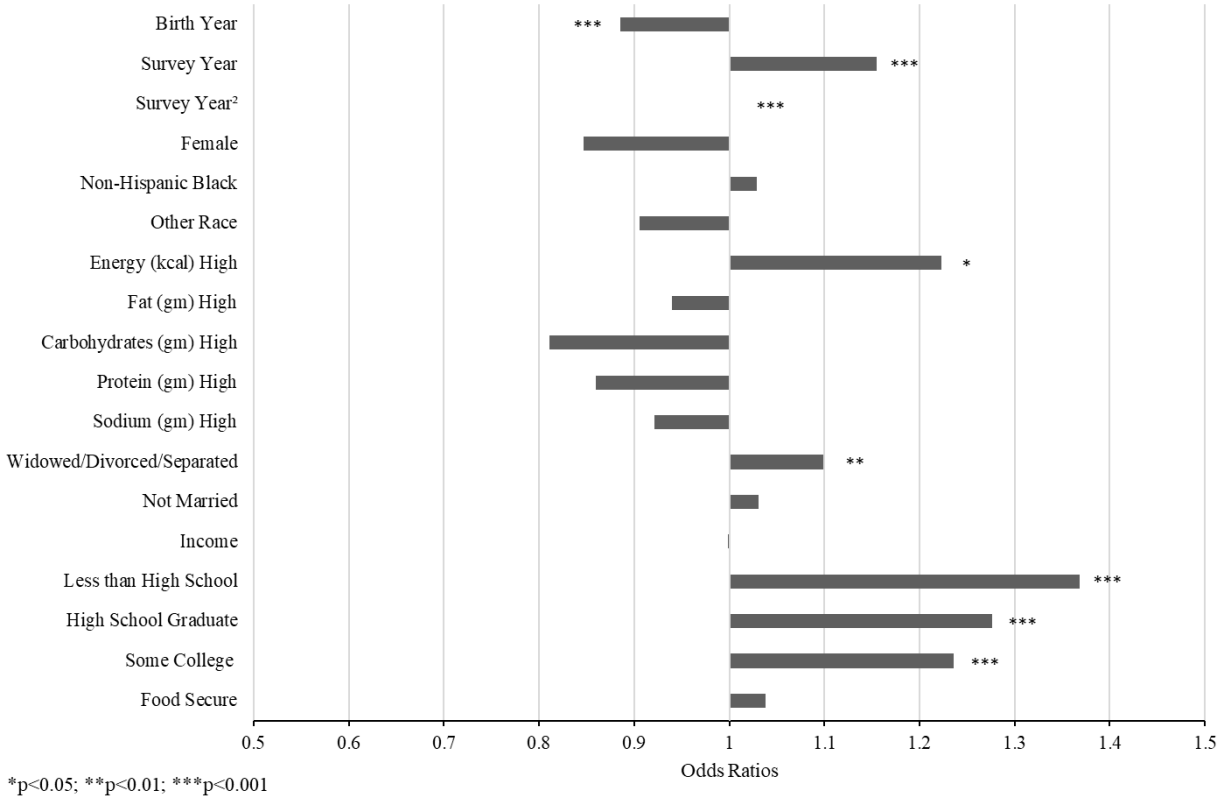


Figure 1: Historical Trends in Obesity for Children Age 2-19 Years, 1971-2012: NHANES (n=33,806)
30%
25%
20%
15%
10%
5%
0%
% Obese
1971/74
1976/1980
1988/1994
1999/2000
2001/2002
2003/2004
2005/2006
2007/2008
2009/2010
2011/2012
Survey Year
Overall
Girls
Boys
Note: Error bars indicate standard errors

Figure 2: Weighted Logistic Regression Models Regressing Child Obesity on Birth Year, Survey Year, and Key Covariates, NHANES 1971-2012 (n = 33,806)



*p<0.05; **p<0.01; ***p<0.001

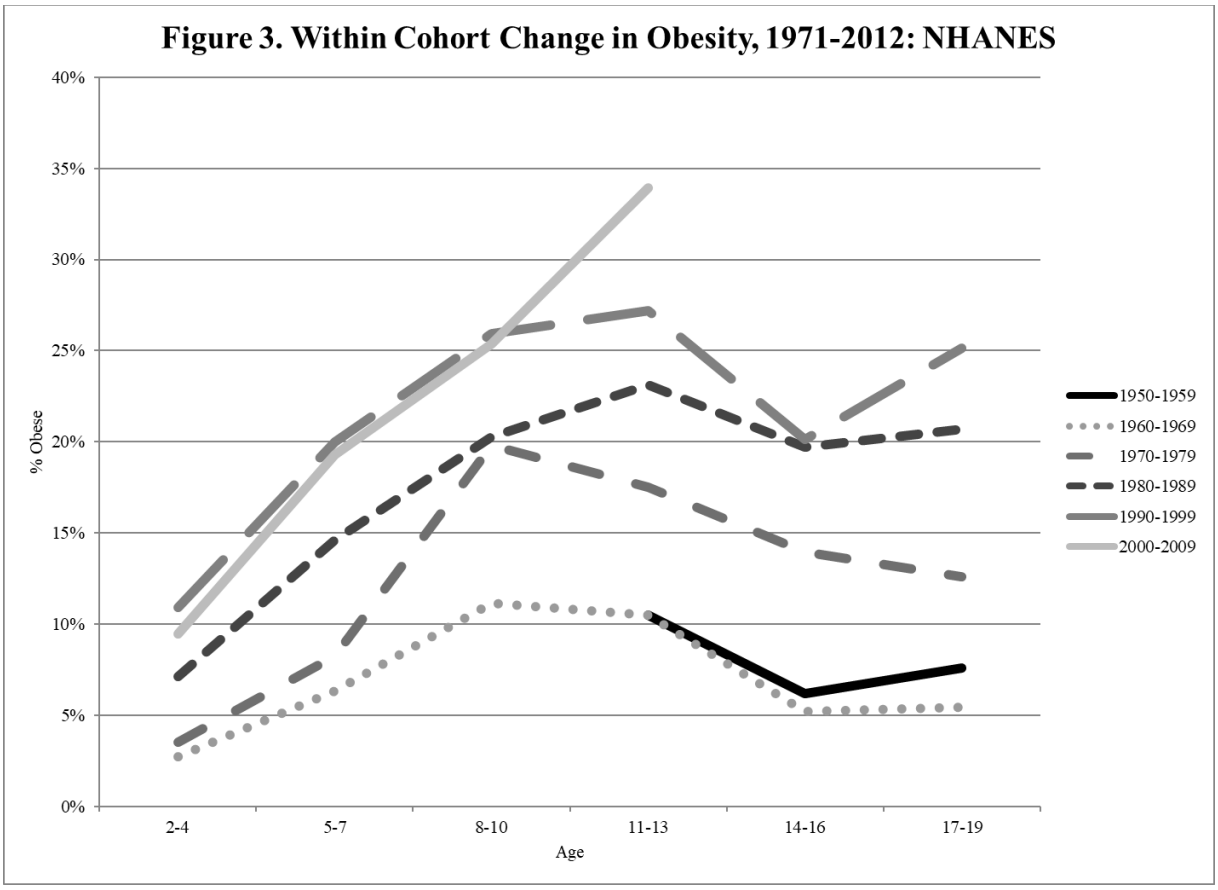


Figure 3. Within Cohort Change in Obesity, 1971-2012: NHANES
40%
35%
30%
25%
20%
15%
10%
5%
0%
% Obese
2-4
5-7
8-10
11-13
14-16
17-19
Age
1950-1959
1960-1969
1970-1979
1980-1989
1990-1999
2000-2009

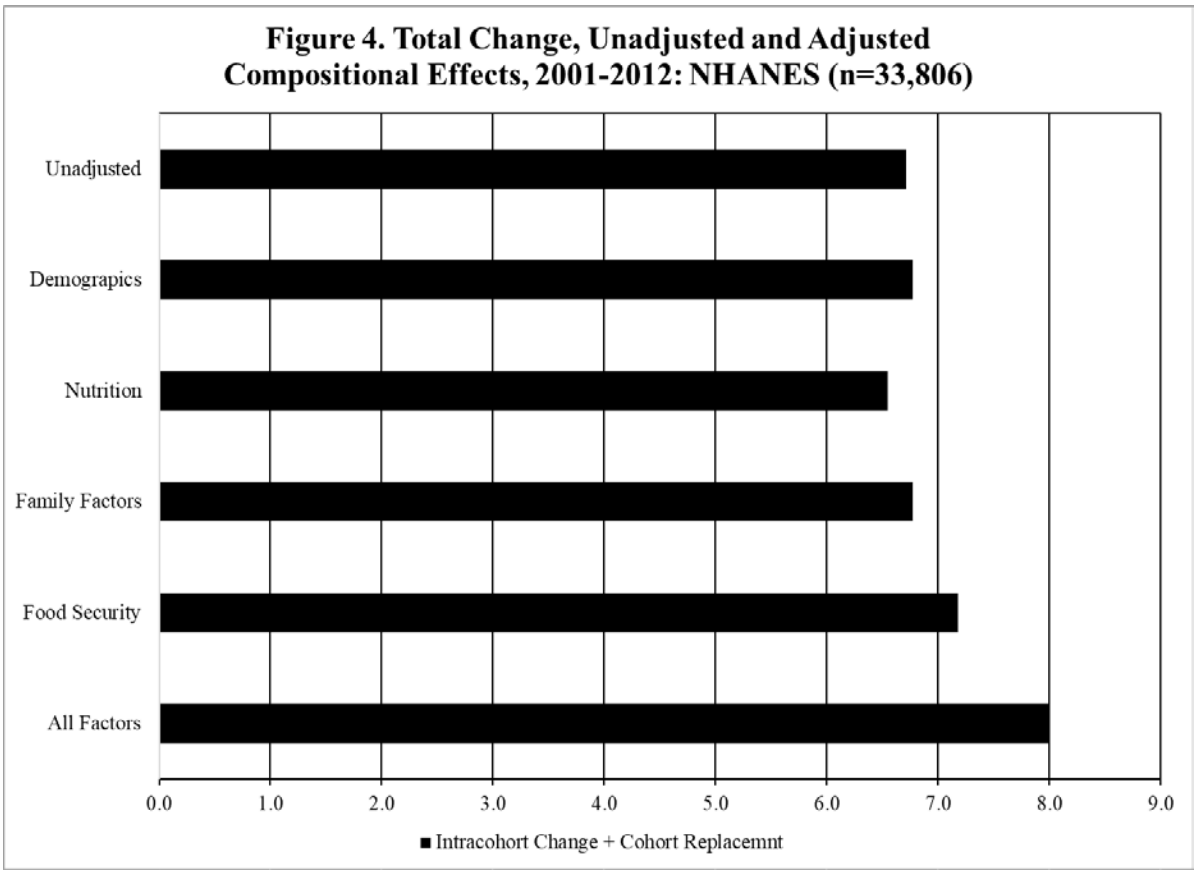
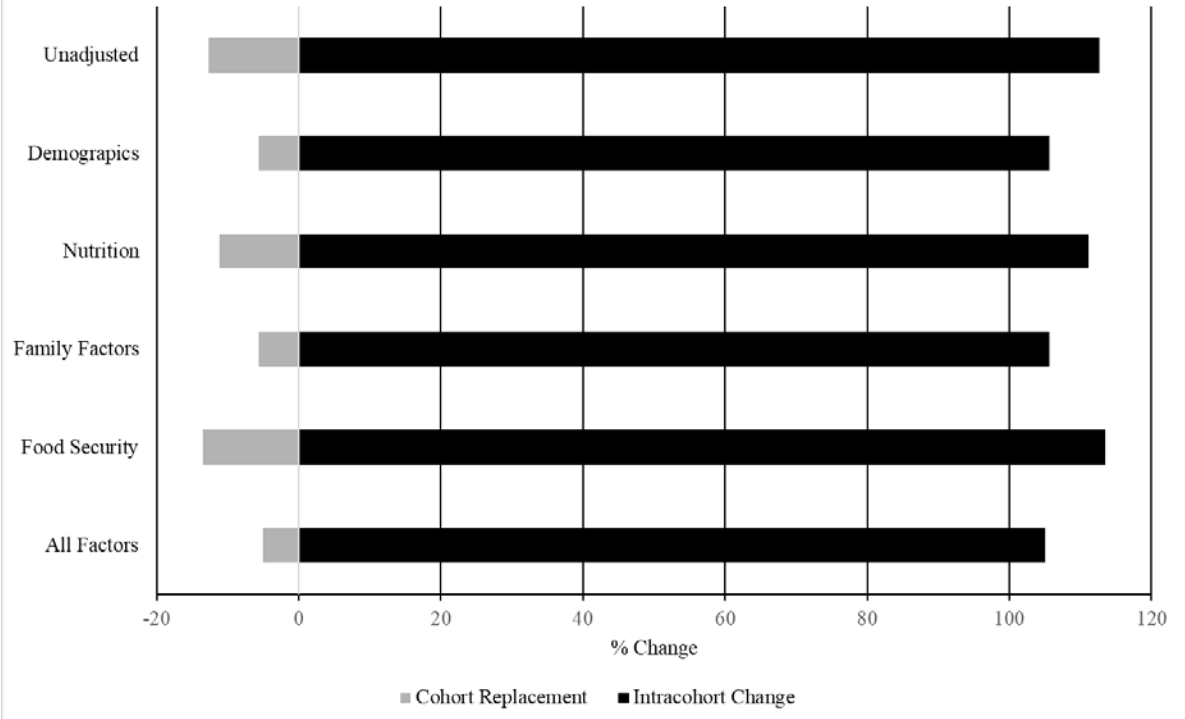


Figure 4. Total Change, Unadjusted and Adjusted Compositional Effects, 2001-2012: NHANES (n=33,806)
Unadjusted
Demograpics
Nutrition
Family Factors
Food Security
All Factors
0.0
1.0
2.0
3.0
4.0
5.0
6.0
7.0
8.0
9.0
Intracohort Change + Cohort Replacemnt

Figure 5. Intracohort Change (IC) and Cohort Replacement (CR), Unadjusted and Adjusted Compositional Effects, 2001-2012: NHANES (n=33,806)



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