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Research article

Explaining adult obesity, severe obesity, and BMI: Five decades of change

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ABSTRACT

Obesity rates have increased across all segments of society since the late 1970s, but the reason behind population-level increases in body weight remains unclear. We used the 1971–2020 NHANES data to examine whether the observed trend in obesity prevalence is attributable to changing public health behaviors (i.e., intracohort change) *or* changing publics (i.e., cohort replacement). We partitioned total change in mean BMI, and rates of obesity and severe obesity, into its IC and CR components using linear and algebraic decomposition methods. We found that the IC mechanism (i.e., broad sectors of individuals changing) plays a dominant role in the overall increase in mean BMI, and obesity and severe obesity prevalence. Birth cohort membership (i.e., the CR mechanism) is also influencing mean BMI, and rates of obesity and severe obesity, but in differing ways. Specifically, the large positive IC and the small positive CR effects are amplifying one another, thus creating a steep increase in the observed rates of severe obesity. Conversely, the large positive IC effect is offset by a small negative CR effect, which created a more gradual rise in mean BMI and rates of obesity. Furthermore, we computed total change for models that entered separately sociodemographic, lifestyle, nutritional, and physical activity measures to estimate differences in mean BMI, and rates of obesity and severe obesity, among cohorts and time periods. Adjustment for all the compositional differences among the cohorts during the study period indicate that a combination of a more pronounced IC and a less pronounced CR drove the observed increase in mean BMI, and rates of obesity and severe obesity. Thus, "universal prevention" (i.e., entire community) strategies for healthy weight promotion may need to be combined with "selective prevention" (i.e., at-risk groups) and/or "targeted prevention" (i.e., at-risk individuals) approaches in order to reverse the obesity epidemic.

1. Introduction

The prevalence of obesity in the United States has tripled over the last five decades and the rise is forecast to continue despite population-based weight management efforts [1–4]. Obesity rates have increased across all segments of society since the late 1970s, and the upward trend is troubling because excess body weight is associated with adverse health outcomes and high health care expenditures [5–7]. Obesity is a multifactorial disease and efforts to address the obesity epidemic are complicated by many factors [8]. At the individual level, excessive adiposity results from a chronic positive energy balance through an interplay of genetic, biological,

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behavioral, socioeconomic, and environmental factors [9–14]. Still, at a population level, the reason behind the obesity epidemic remains unclear [15,16]. That said, understated is the notion that shifting rates of obesity among individuals over time might be a consequence of two proximate sources: broad sectors of individuals changing [(i.e., intracohort change (IC)] *or* populations changing [(i.e., cohort replacement (CR)] [7,17–21].

Across all segments of the population, the longstanding rise in obesity prevalence is associated with environmental factors such as technological advances and economic modernization (transportation, electronics, telecommunications), built environment (neighborhood deprivation, crime, walkability, access and proximity to grocery stores, parks, recreational facilities), food systems change (growth, processing, preservation of food), social hierarchy (inequality, perceived discrimination), and cultural predispositions (cultural standards, practices, beliefs) [22–29]. Important for our current study, the above-mentioned environmental determinants of obesity are associated with unhealthy nutrition practices (e.g., consumption of energy-dense diets with low nutritional value) and, possibly, sedentary lifestyle (e.g., screen time, car use), as well as tobacco and alcohol use [22–36]. Within populations, the prevalence of obesity is associated with individual characteristics, including age, sex, race/ethnicity, education, and socioeconomic status (SES) [37–39]. Notably, these individual-level factors also are associated with health behaviors such as overall levels of physical activity, dietary patterns, cigarette smoking, and alcohol consumption [40–43].

Given the wide range of factors impacting obesity and lack of consensus about the source of population-level increases in body weight, here we examined whether the observed trend in obesity prevalence is attributable to changing public health behaviors such as calorie intake, energy expenditure, alcohol drinking, and cigarette smoking (i.e., IC) *or* changing publics such as demographics and social status (i.e., CR). Partitioning total population-level change (TC) in obesity prevalence into its IC (i.e., individuals changing across sizeable segments of society) and CR (i.e., the change in population composition) components might help clarify the root causes of the obesity epidemic and guide us to clinically-meaningful population-level prevention strategies [44,45].

2. Methods

2.1. Data

We analyzed the National Health and Nutrition Examination Survey (NHANES) cross-sectional data from thirteen cycles, including: NHANES I (1971–75); NHANES II (1976–80); NHANES III (1988–94), and all 2-year "continuous" NHANES (1999–2020). The sample of ~5,000 persons each year is representative of the civilian, non-institutionalized U.S. population [46]. Samples were restricted to adults 20 years of age and older, consistent with the analytic and reporting guidelines issued by the National Center for Health Statistics [46]. We excluded those with an incomplete body measurement component (9.2%), as well as pregnant and recently pregnant females (6.8%). Missing data rates for other variables used in the study range from 1.7% for educational attainment to 9.7% for household income. The total sample size for all years is 76,360. In our analyses, we employed the NHANES cycle-specific sampling weights that account for differences in the unequal probabilities of selection and non-response. See the National Center for Health Statistics for more information on the NHANES data [46].

2.2. Measures

Three separate dependent variables were analyzed: body mass index (BMI), obesity, and severe obesity. BMI was calculated as weight (kg)/height (m) [2]. We logged BMI to adjust for non-linearity (positive skew). Obesity was defined as a BMI $\geq$30 and $\leq$ 39.9 kg m^{-2} and dichotomized to indicate whether or not the individual is obese [47]. Severe obesity was defined as a BMI at or above 40.0 kg m^{-2} and treated as a binary variable to indicate whether or not the individual is severely obese [47].

We added covariates to represent sociodemographic, nutritional, physical activity, and lifestyle factors that are associated with changes in body weight. Sociodemographic factors include survey year, birth year, sex (1 = female), race/ethnicity (1 = non-Latinx white), marital status (1 = not married), household income, and educational attainment (1 = less than high school). Nutritional factors include total intake of energy (1 = > 2,000 kcals), fat (1 = > 78 g), carbohydrates (1 = <100 g), protein (1 = > 56 g), and sodium (1 = >1,500 mg) during a 24-h period. We classified physical activity by the rate of energy expenditure (1 = < 3 metabolic equivalents (MET) defined as 3.5 ml $O_2 \cdot kg^{-1}$ min^{-1}), consistent with a particular activity. Lifestyle characteristics include current/former tobacco smoking behaviors (1 = smoked at least 100 cigarettes during entire life) and weekly alcohol usage (for women, 1 = 0 drinks; 2 = $\geq$1 and $\leq$ 7 drinks, 3 = >7 drinks; for men, 1 = 0 drinks; 2 = $\geq$1 and $\leq$ 14 drinks, 3 = >14 drinks).

2.3. Statistical analyses

We employed Glenn Firebaugh's (1989) linear decomposition technique to decompose TC in body weight into change driven by IC and change spurred by CR [48]. Differences among cohorts are estimated by regressing logged BMI, obesity, and severe obesity on birth year and survey year [48]. Because this method assumes linearity but change in body weight may not be linear, we also used Kitagawa's (1955) algebraic decomposition method, and collapsed birth year into cohort subgroups [48,49]. Both decomposition techniques partition TC in body weight into IC and CR, the two components of aggregate social change. To estimate IC, we multiplied the regression coefficient for the survey year variable by the study period length (i.e., last survey year - first survey year). To calculate CR, we multiplied the regression coefficient for birth year by the difference between the mean birth years for the final and initial survey years. IC tells us how much of the TC in body weight is attributable to individual change (i.e., period changes) and CR reveals how much of the TC in body weight is attributable to population turnover (i.e., the death of older cohorts with lower BMIs and the birth of

Table 1
Weighted means and standard errors for independent and dependent variables for adults aged 20+ years old, 1971–2020; N = 76,360.

	1971–2020		1971		2020		Δ2020–1971
Dependent Variable	Mean	Std.Err.	Mean	Std.Err.	Mean	Std.Err.	
BMI	28.3	0.2	25.7	0.2	30.0	0.1	4.3***
Logged BMI	3.3	0.0	3.2	0.0	3.4	0.0	0.1***
Obesity	0.3	0.0	0.2	0.0	0.3	0.0	0.2***
Severe Obesity	0.1	0.0	0.0	0.0	0.1	0.0	0.1***
Independent Variables							
Age							
Birth Year	1964	0.1	1927	0.6	1974	0.3	47.4***
Sociodemographics							
Sex							
Male	0.5	0.0	0.5	0.0	0.5	0.0	−0.0
Female^	0.5	0.0	0.5	0.0	0.5	0.0	0.0
Race							
Non-Hispanic White^	0.2	0.0	0.9	0.0	0.1	0.0	−0.8***
Non-Hispanic Black	0.1	0.0	0.1	0.0	0.1	0.0	0.0***
Other Race	0.8	0.0	0.0	0.0	0.8	0.0	0.8***
Marital Status							
Married^	0.6	0.0	0.8	0.0	0.6	0.0	−0.2***
Widowed/Divorced/Separated	0.2	0.0	0.1	0.0	0.3	0.0	0.1***
Not married	0.2	0.0	0.1	0.0	0.1	0.0	0.1***
Household Income	50.1	116.2	42.9	0.8	59.7	576.3	9.5***
Education							
Less than High School^	0.2	0.0	0.3	0.0	0.1	0.0	−0.3***
Highschool Graduate	0.2	0.0	0.4	0.0	0.3	0.0	−0.1***
Some College	0.3	0.0	0.2	0.0	0.3	0.0	0.1***
College Graduate	0.3	0.0	0.1	0.0	0.3	0.0	0.3***
Nutrition							
Energy (kcal)							
≤2,000	0.5	0.0	0.9	0.0	0.5	0.0	−0.4***
>2,000^	0.5	0.0	0.1	0.0	0.5	0.0	0.4***
Fat (gm)							
≤78	0.5	0.0	0.8	0.0	0.5	0.0	−0.3***
>78^	0.5	0.0	0.2	0.0	0.5	0.0	0.3***
Carbohydrates (gm)							
≤100	0.1	0.0	0.7	0.0	0.1	0.0	−0.5***
>100^	0.9	0.0	0.3	0.0	0.9	0.0	0.5***
Protein (gm)							
≤56	0.3	0.0	0.8	0.0	0.3	0.0	−0.5***
>56^	0.7	0.0	0.2	0.0	0.7	0.0	0.5***
Sodium (mg)							
≤1,500	0.1	0.0	0.8	0.0	0.1	0.0	−0.7***
>1,500^	0.9	0.0	0.0	0.0	0.9	0.0	0.8***
Activity							
<3 METS^	0.3	0.0	0.4	0.0	0.4	0.0	−0.0
≥3 and < 6 METS	0.4	0.0	0.4	0.0	0.3	0.0	−0.1***
≥6 METS	0.3	0.0	0.2	0.0	0.3	0.0	0.1***
Lifestyle							
Smoked at least 100 Cigarettes in Lifetime							
No^	0.5	0.0	0.4	0.0	0.6	0.0	−0.2***
Yes	0.5	0.0	0.6	0.0	0.4	0.0	0.2***
Average Weekly Alcohol Usage							
Male							
0 drinks^	0.2	0.0	0.4	0.0	0.2	0.0	0.2**
≥1 and ≤ 14 drinks	0.4	0.0	0.4	0.0	0.4	0.0	0.0
>14 drinks	0.4	0.0	0.2	0.0	0.2	0.0	0.0
Female							
0 drinks^	0.5	0.0	0.6	0.0	0.4	0.0	−0.2***
≥1 and ≤ 7 drinks	0.4	0.0	0.3	0.0	0.4	0.0	0.1**
>7 drinks	0.1	0.0	0.1	0.0	0.2	0.0	0.1**

Source: NHANES 1971-2020
Note: ^ Indicates Reference Group.
Note: Asterisks indicate significant change evaluated using two-tailed independent means *t*-test.

new cohorts with higher BMIs, for example). We used linear interpolation methods due to the multi-year NHANES data collection design, and assigned the midpoint mean for every survey wave (e.g., if 1971/73, we used 1972) [46].

3. Results

Between the early 1970s and early 2020s, BMI, obesity, and severe obesity increased significantly (Table 1). In 1971, the average population BMI for adults aged 20 years and older was 25.7 (logged = 3.2). By 2020, the mean BMI increased to 30.0 (logged = 3.4). During this period, obesity and severe obesity increased by 20% and 10%, respectively. In Figs. 1–3, we display trends in mean BMI, obesity, and severe obesity overall, and for men and women separately, during the past five decades. Average population BMI and obesity rates were relatively stable in the early-to-mid 1970s, followed by a sharp increase in the early 1980s. The increase in mean BMI and obesity did stabilize somewhat beginning in the late 1990s, but a steady incline did continue since the late 1970s. The obesity rate increased by 7% (15% to 22%) from the 1970s–1990s. During this time period, the severe obesity rate tripled, from 1% to 4%. Between 1971 and 2020, average BMI increased by more than 16% (25.7 to 29.8).

The obesity and severe obesity rates vary for men and women, with somewhat higher rates for women than for men from the late 2000s into the early 2020s. For example, the obesity rate for women and men increased by about 20% and 14%, respectively, from the 2011/12 to 2017/20 cycle, and the severe obesity rate for women and men increased by about 35% and 43%, respectively. While trends in mean BMI and obesity for men and women appear to parallel one another, obesity rates grew more sharply for men. For both men and women, mean BMI and obesity rates were largely unchanged between 1971/74–1976/80. Between 1976/80–1999/2000, the obesity and severe obesity rates for men increased by 83% and 326%, respectively, whereas these rates grew by 58% and 161% for women. Since 1990/2000, the obesity rates for men and women rose by 14% and 20%, respectively, whereas the severe obesity rate for both men and women increased by about 34%.

The sharp rise in average population BMI, and prevalence of obesity and severe obesity, over the last five decades may reflect two fundamentally different processes of change. *If* IC is the principal driver of the increase, then all cohorts will increase after the rise began in the early 1980s. The increase will be greater for more recent, compared with earlier, cohorts, thus leading to a faster rise. However, *if* CR is central to this increase, then the rates for more recent cohorts will be higher than for those of previous cohorts throughout adulthood, thus creating a more gradual rise.

As a preliminary step, in order to uncover the relative importance of IC and CR for mean BMI, as well as obesity and severe obesity rates, we constructed ten-year age group intervals for the cohorts in our study with sufficient sample sizes ($N \geq 50$; Figs. 4–6). In Figs. 4–6, we show that both IC and CR played significant roles in mean BMI, obesity, and severe obesity increase over the last 50 years. Consistent with the CR explanation, more recent cohorts have higher mean BMI, as well as obesity and severe obesity rates, than do earlier cohorts over the course of adulthood. For example, 8% of 20–29-year-olds born between 1940 and 1949 were obese (1% were severely obese), whereas 20% of 20–29-year-olds born between 1980 and 1989 were obese (4% were severely obese). At the other end of the age continuum, the obesity rate for 70–79-year-olds increased from 17% to 35% for the 1900–1909 and 1940–1949 cohorts, respectively. The severe obesity rate for this age group increased from 1% to 8%. Consistent with the IC explanation, mean BMI and obesity rates increased over the life course for almost all cohorts. For example, the obesity and severe obesity rates rose from 7% to 1% for the 1950–1959 cohort at ages 20–29, respectively, to 40% (obesity) and 8% (severe obesity) at ages 70–79. With the exception of one of the earliest cohorts (1910–1919), mean BMI, and rates of obesity and severe obesity, increased significantly over time as cohort members aged.

Regarding the compositional differences between cohorts, in Fig. 7, we display the percentages of TC in mean BMI, obesity, and severe obesity due to IC and CR for unadjusted models (no control variables), as well as for conditional models that adjust for socio-demographic, nutritional, physical activity, and lifestyle differences among cohorts and across time periods. The top bar in each panel depicts the percentage of TC in mean BMI, obesity, and severe obesity attributable to IC (blue) and CR (red), *if* we ignore the compositional differences among the cohorts.

In Fig. 7, we show that the increase in average population BMI, and obesity and severe obesity rates over the last five decades largely reflect IC (i.e., within-cohort or individual change). Regarding the mean BMI and obesity prevalence, in the unadjusted model,

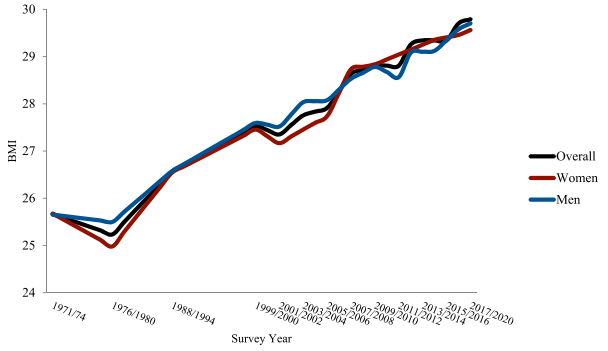

Fig. 1. Historical trends in BMI for adults age 20+ years, 1971–2020: NHANES

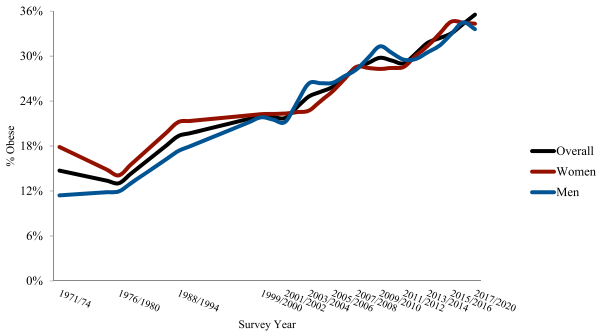

Fig. 2. Historical trends in obesity for adults age 20+ years, 1971–2020: NHANES

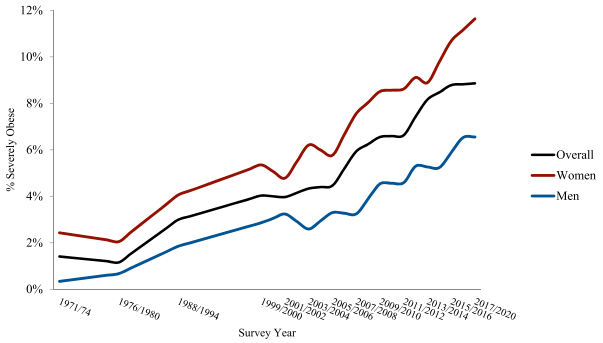

Fig. 3. Historical trends in severe obesity for adults age 20+ years, 1971–2020: NHANES

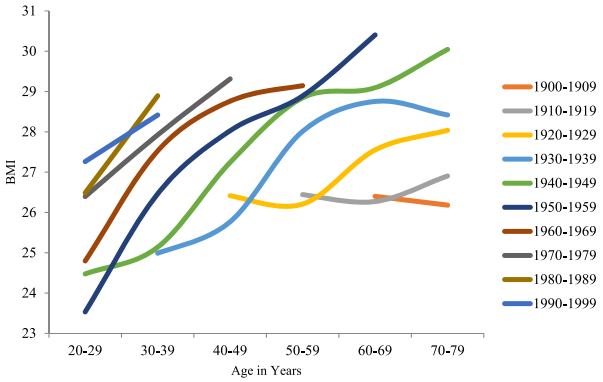

Fig. 4. Within cohort change in BMI, 1971–2020: NHANES

negative CR partially offsets the increase across cohorts. This partial offset is evident because IC accounts for nearly 78% and 82% of TC in mean BMI and obesity, respectively, whereas CR offsets 22% and 18% of the increase for mean BMI and obesity, respectively. Concerning the severe obesity prevalence, in the unadjusted model, the observed increase in prevalence is due to a large positive IC and CR effects, with their effect amplifying rather than offsetting one another, thus creating a steep rise in severe obesity prevalence.

Adjusting for changes in the population composition may affect the relative contributions of IC (i.e., individual change) and CR (i.e., population turnover) to TC (i.e., aggregate change). For example, *if* more recent cohorts had higher mean BMI and obesity rates than did previous cohorts because they had worse health behaviors, controlling for changes in lifestyle habits would attenuate or eliminate the influence of CR on TC. The 2nd through 5th bars in each panel in Fig. 7 indicated how accounting for these compositional differences influenced the relative contributions of IC and CR. We see that the increase in mean BMI and obesity is by-and-large attributable to IC once we compare the top bars in each panel (i.e., no controls) and bottom bars in each panel (i.e., all controls) in Fig. 7. In fact, CR has slightly attenuated the increase in average population BMI and obesity, thus creating a more gradual rise.

Sociodemographic compositional changes of the population led to a sharper rise in obesity. In fact, adjusting for sociodemographics eliminates the negative, and actually leads to a strong positive, CR effect. Conversely, the changing sociodemographics contributed to a

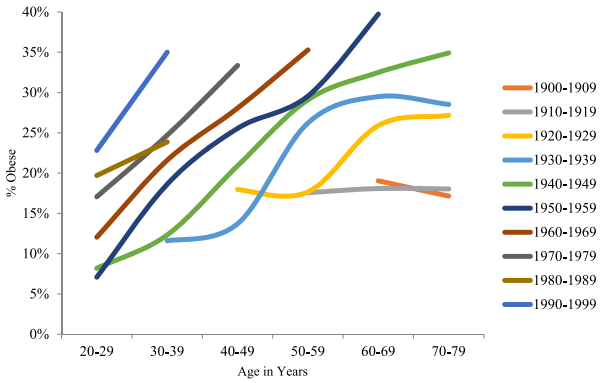

Fig. 5. Within cohort change in obesity, 1971–2020: NHANES

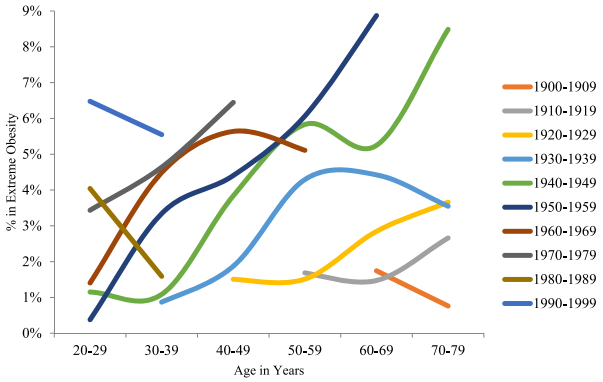

Fig. 6. Within cohort change in severe obesity, 1971–2020: NHANES

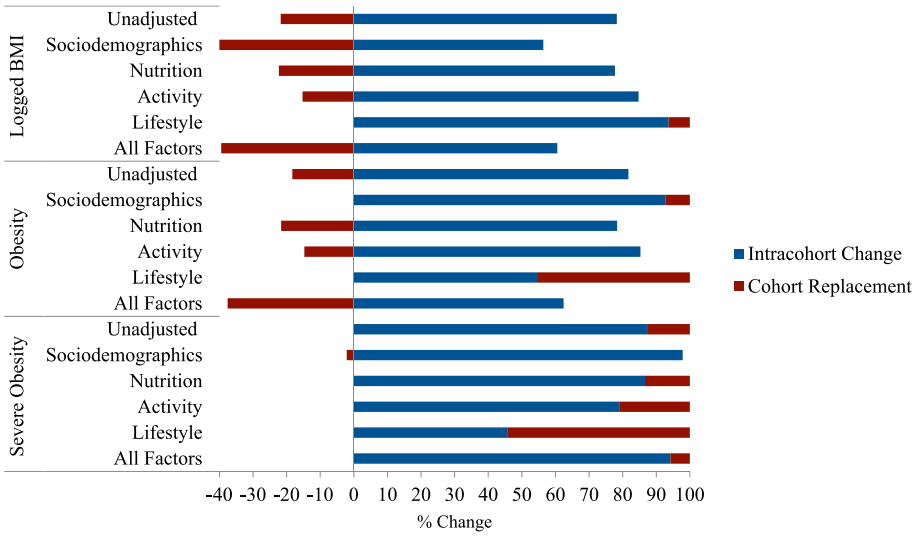

Fig. 7. Intracohort change and cohort replacement, unadjusted and adjusted compositional effects, 1971–2020: NHANES

slower rise in BMI and severe obesity, evidenced by the negative CR effect. Adjusting for physical activity weakens the large, positive IC effect, while simultaneously boosting the moderating effect of CR on TC in the severe obesity rate. Ultimately, more recent cohorts have higher rates of severe obesity than do earlier cohorts because, in part, their physical activity habits have changed. Changing lifestyle habits led to a sharper rise in BMI, obesity, and severe obesity. Actually, adjusting for lifestyle factors eliminates the negative, and leads to a strong positive, CR effect. Results from the algebraic decomposition method, which does not assume linearity, reveal

similar findings to estimates reported here.

4. Discussion

Obesity is a chronic, relapsing morbus with a multifactorial etiology and pathogenesis that increases the risk of several non-communicable diseases [50–52]. Unintentional, excessive weight gain at the individual level is an unintended consequence of multiple layers of influence (e.g., obesogenic environment) at a population level [15,16,53]. The prevalence of obesity in the United States has increased dramatically over the last five decades, but it has proven difficult to identify the reasons behind excessive adiposity at a population level [2,8,17]. It is troubling that the rates of obesity continue to rise while the effectiveness of existing population-based initiatives to combat this deadly epidemic is yet unclear [3,54–57]. In line with previous studies of U.S. adults, our current results indicate that average population BMI, as well as obesity and severe obesity rates, are at historically high levels (Figs. 1–3) [1–3]. In fact, the U.S. has already moved away from the *Healthy People 2030* goal of 36% prevalence of obesity in adults [58]. Notably, the steadily increasing obesity prevalence is forecast to continue, with steepest increases in higher income nations [59]. Certainly, the modeled trends are a cautionary tale for all Americans with important implications for how the obesity epidemic will evolve in the United States [60,61]. As shown in Fig. 3, of great concern is the rapid increase in severe obesity rates, from 1.5% in 1971/74 to 9% in 2017/20, especially among women (2.5% in 1971/74 and 12% in 2017/20). Indeed, severe obesity is linked to a higher risk of future morbidity and mortality compared to that of overweight or obesity, and is remarkably taxing on the individual person and the health care system [62–64].

Considering the growing burden and challenge of managing the societal impact of excessive weight gain in terms of population health and economic outcomes, here we expand upon our previous research and partition total social change in average population BMI, obesity, and severe obesity into its IC and CR components [17]. Crucially, this method may help elucidate the root causes of the obesity epidemic and provide the empirical evidence base for the use of interventions likely to be most effective at reversing the disconcertingly high obesity rates [7,17,65,66]. More specifically, *if* population turnover (i.e., CR) is driving the nationwide increases in obesity prevalence, policymakers should target younger generations because the change is happening between, rather than within, cohorts. However, *if* period effects (i.e., IC) are influencing the overall prevalence of obesity, we need population-wide policies because high obesity rates are distributed widely across all ages and generations. Our present findings indicate that the IC mechanism (i.e., broad sectors of individuals changing) plays a dominant role in the overall increase in mean BMI, and obesity and severe obesity prevalence (Fig. 7). In other words, we are seeing a significant upward trend because the members of all cohorts are getting heavier simultaneously. These results are comparable to our previous findings where we used NHANES data from 1971 to 2012 [17]. Furthermore, birth cohort membership (i.e., the CR mechanism) is also influencing mean BMI, as well as rates of obesity and severe obesity, but in differing ways. Specifically, the large positive IC and the small positive CR effects are amplifying one another, thus creating the steep increase in the observed rates of severe obesity (Fig. 3). Thus, similar to our earlier report, this means that fewer people with severe obesity in earlier-born cohorts are dying and being replaced by a greater number of people with severe obesity in later-born cohorts [17]. Conversely, for average population BMI and rates of obesity, the large positive IC effect is offset by the small negative CR effect, which created a more gradual rise (Figs. 1 and 2). These findings regarding mean BMI and rates of obesity are in contrast to our previously published results [17]. Indeed, between 1971 and 2012, the positive CR effect reinforced and amplified the positive IC effect, which led to a rapid increase in average population BMI and rates of obesity [17]. Hence, our present results signify that people with a higher mean BMI in earlier-born cohorts are dying and being replaced by people with a lower mean BMI in later-born cohorts, as well as that a greater number of people with obesity in earlier-born cohorts are dying and being replaced by fewer people with obesity who belong to later-born cohorts.

Discourse around the dynamics of the obesity epidemic requires consideration of empirical evidence that crosses levels of analysis, from individual to structural. Here, we used linear and algebraic decomposition techniques to identify the sources of the changing trends in the distribution of mean BMI, obesity, and severe obesity in the adult U.S. population from 1971 to 2020. We computed TC for models that entered separately sociodemographic, nutritional, physical activity, and lifestyle measures to estimate differences in mean BMI, and rates of obesity and severe obesity among cohorts and across time periods. Adjusting for differences in the sociodemographic composition of the population influenced the estimates of IC and CR for the mean BMI, obesity, and severe obesity measures (2nd bar in Fig. 7). Specifically, once we account for cross-cohort change in sociodemographics, later-born cohorts have much higher rates of obesity than do those earlier-born cohorts that they are replacing. This finding parallels our previous report and indicates that the obesity prevalence increased because the size of demographic and socioeconomic groups with higher rates of obesity got larger and the size of groups with lower obesity prevalence got smaller [17]. On the other hand, for mean BMI and rates of severe obesity, the changing sociodemographics muted what would have otherwise been an even larger IC effect. Further, the changes in nutrition over time had a negligible effect on mean BMI, and obesity and severe obesity prevalence (3rd bar in Fig. 7). Changes in physical activity account for the overall shift and increase in severe obesity, but not in mean BMI or rates of obesity (4th bar in Fig. 7). This means that the rates of severe obesity increased, in part, because physical activity habits changed across earlier-born and later-born cohorts [67,68]. In our previous study, we did not include lifestyle factors (i.e., alcohol use, cigarette smoking) in the analysis [17]. Here, we show that the changing lifestyle habits over time influenced the estimates of IC and CR for the mean BMI, obesity, and severe obesity measures (5th bar in Fig. 7). Once we account for cross-cohort change in lifestyle characteristics, later-born cohorts have higher mean BMI and rates of obesity than do those earlier-born cohorts that they are replacing. Indeed, the decrease in the prevalence of cigarette smoking between 1971/75–1999/2002 has been associated with a small increase in the prevalence of obesity during this time period [35]. Also, as shown in Table 1, the prevalence of females who average more than 7 drinks per week has increased significantly from 1971 to 2020. This is noteworthy given that heavy alcohol intake may be a risk factor for obesity [36]. The rates of severe obesity

increased, in part, because lifestyle habits changed across earlier-born and later-born cohorts. Adjustment for all the compositional differences among the cohorts during the study period indicate that CR contributed moderately to the increase in severe obesity (6%; 5th bar in Fig. 7). Thus, overall, a combination of a more pronounced IC and a less pronounced CR drove the observed increase in average population BMI, and rates of obesity and severe obesity. Accordingly, per the World Health Organization framework, it is likely that "universal prevention" (i.e., entire community) strategies for healthy weight promotion may need to be combined with "selective prevention" (i.e., at-risk groups) and/or "targeted prevention" (i.e., at-risk individuals) approaches in order to reverse the obesity epidemic [69].

Our study, though informative, is not without limitations. The cross-sectional data limit our ability to make causal inferences [46]. Similarly, the sometimes-crude nature of measures available in the NHANES across a 49-year period for several key concepts may influence our estimates [46]. For example, Latinx Americans are more likely to present with obesity than non-Latinx whites, but the early years of the NHANES do not have these data [46,70]. Related, we dichotomized nutritional intake measures to normal/high ranges due to a lack of variation across cohorts, likely masking more nuanced patterns [46]. Additionally, genetic makeup contributes to individual variation in obesity and BMI, and early-life development of obesity is a risk factor for adult obesity, but NHANES does not have this information [46,71–73]. Also, we did not link individual- and family-level data to neighborhood measures despite that the neighborhood socioeconomic environment is a strong determinant of body weight status [13]. Moreover, although BMI is a simple measure, it is reasonably good for the classification of excess adiposity, especially when height and weight measures are collected objectively, but more so for adults than the elderly [74]. Also, BMI is a useful measure for estimating risk of obesity-related health outcomes in large, representative samples [75,76]. Finally, linear and algebraic decomposition techniques are but two approaches to estimate age, period, and cohort effects [77]. Despite these limitations, we took a much-needed step closer to the identification of underlying mechanisms that are driving the obesity epidemic.

Here, we sought to uncover the putative sources of population-level increases in the prevalence of obesity and severe obesity among American adults during the last five decades. The upward shift in prevalence seems to be driven by IC, wherein individuals of all ages and across multiple birth cohorts are gaining body weight simultaneously. Thus, the rates of obesity, and especially severe obesity, continue to rise rapidly in the United States. Similarly, we found a long-term upward trend with average population BMI, wherein large segments of the society are presenting with higher BMIs. Accordingly, the case for immediate action is strong, and the cost of inaction is too great [65]. Decision-makers and policy-makers must translate evidence-based research, and practice-based evidence, into practice [78]. At present, systemic biases in discourse, policies, and practices cultivate socioeconomic inequities that, in turn, create health disparities [79]. Policy solutions should tackle the sociodemographic patterning of obesity, if we are to overcome the epidemic [80, 81]. It also is evident that one tool alone, be it a junk food tax, subsidies to encourage physical activity, or restriction of alcohol and tobacco marketing, is not enough to achieve population-level benefits [82–85]. Crucially, large-scale changes to the food environment are required to help sustain the individual consumers' transition towards healthy diets [86,87]. In terms of biological and psychosocial influences, the relationship between alcohol or tobacco and obesity undoubtedly is complex [88,89]. For example, problematic alcohol use and cigarette smoking share common underlying genetic architecture [90]. Moreover, heavy smokers or heavy drinkers tend to present with greater body weight than do light smokers or light drinkers and nonsmokers or nondrinkers, which may be due to clustering of health compromising behaviors such as physical inactivity and poor diet [91]. Finally, it is necessary to evaluate contradictory data and claims that arise due to different research methods and levels of analysis when investigating the relationship between alcohol consumption or tobacco use and the risk of weight gain and obesity [92]. The obesity epidemic has no simple solutions, and broader social changes are needed to move beyond the current impasse.

Author contribution statement

AW Kranjac, D Kranjac: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data; Wrote the paper.

Data availability statement

Data associated with this study has been deposited at https://wwwn.cdc.gov/nchs/nhanes/Default.aspx.

Additional information

No additional information is available for this paper.

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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