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Propagation of Obesity across Generations: The Roles of Differential Realized Fertility and Assortative Mating by Body Mass Index

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Key Words
Obesity · Body mass index · Assortative mating · Realized fertility · Monte Carlo simulation

Abstract
Background/Aims: To quantify the extent to which the increase in obesity observed across recent generations of the American population is associated with the individual or combined effects of assortative mating (AM) for body mass index (BMI) and differential realized fertility by BMI. ***Methods:*** A Monte Carlo framework is formed and informed using data collected from the National Longitudinal Survey of Youth (NLSY). The model has 2 portions: one that generates childbirth events on an annual basis and another that produces a BMI for each child. Once the model is informed using the data, a reference distribution of offspring BMIs is simulated. We quantify the effects of our factors of interest by removing them from the model and comparing the resulting offspring BMI distributions with that of the baseline scenario. ***Results:*** An association between maternal BMI and number of offspring is evidenced in the NLSY data as well as the presence of AM. These 2 factors combined are associated with an increased mean BMI (+0.067, 95% CI: 0.056; 0.078), an increased BMI variance (+0.578, 95% CI: 0.418; 0.736) and an increased prevalence of obesity (RR 1.032, 95% CI: 1.023; 1.041) and BMIs >40 (RR 1.083, 95% CI: 1.053; 1.118) among offspring. ***Conclusion:*** Our investigation suggests that both differential realized fertility and AM by BMI appear to play a role in the increasing prevalence of obesity in America.

Introduction

The prevalence of obesity has increased substantially since 1980, with more than 60% of the US population now overweight or obese [1, 2]. The contribution of both environmental and genetic factors to the population's increase in obesity is clear [3]. Still, it has been argued that several decades is an insufficient amount of time for the gene pool to change significantly to influence obesity [4]. It is plausible, however, that genetic factors have influenced the prevalence of obesity in several ways. In a closed population and in the absence of disturbing forces, if a population is in Hardy-Weinberg equilibrium, then allele frequencies will remain constant across generations. The disturbing forces that may break the equilibrium include mutation, selection, nonrandom mating, genetic drift, and migration. Moreover, nonrandom mating can change genotype frequencies even without changing allele frequencies.

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Assortative Mating for BMI

Fairly consistent evidence suggests that human mate choice is phenotypically assortative for body mass index (BMI) [5–19]. Intermate correlation for BMI ranges from about 0.05 to 0.25 across many studies, and it averages around a correlation of 0.15 in industrialized societies. Intermate correlation of fat mass is higher at 0.41 [18]. The intermate correlation in adiposity does not increase during cohabitation, and it appears to be due to mate choice rather than factors such as selective divorce [5, 13, 15, 19]. Assortative mating (AM) increases genetic variance in offspring [20], and it can impact the distribution of phenotypes in subsequent generations. For example, Redden and Allison [21] determined that existing intermate BMI correlations alone could increase the prevalence of obesity by 2% over 11 generations if acting in isolation.

Differential Realized Fertility by BMI

Realized fertility is another factor that may define the extent to which genes contribute to obesity prevalence [22]. Fecundity is the *potential* to reproduce or an individual's or population's capacity to produce offspring. However, we are interested not in the capacity per se, but rather the realization of that capacity. Since the terminology used in the literature is inconsistent, we shall use 'realized fertility' to describe this factor of interest: the number of children born to a given woman over her lifetime.

Realized fertility is higher in individuals with higher levels of adiposity [23–25]. Epidemiological evidence consistently shows that women and couples with higher than average BMI produce more offspring. Because the BMI is heritable, increased realized fertility among those with higher BMIs may contribute to an increased prevalence of obesity by increasing the frequency of BMI-increasing alleles in subsequent generations.

Increased realized fertility in overweight and obese individuals as well as AM may have acted independently or in concert to contribute to the increasing prevalence of obesity over time. We aim to examine the extent to which these factors are associated with an increased mean BMI and the prevalence of obesity through an empirical Monte Carlo framework for childbirth events and offspring body composition.

Methods

Data Collection

Longitudinal data were collected from the National Longitudinal Survey of Youth (NLSY), 1979 and 1997 cohorts, conducted by the United States Bureau of Labor Statistics [26]. The 1979 cohort has followed individuals who were aged 14–22 in 1979 from that time forward to the present (we considered data collected up to 2010); the 1997 cohort similarly has followed people who were aged 12–17 in 1997. Some information is exceedingly limited in the 1979 survey cohort; in particular, neither height, weight, nor BMI were recorded for partners of the survey respondents. However, note that the mid-1980s was both the time frame in which the 1997 cohort youths were born as well as the prime child-bearing years for the individuals in the 1979 cohort. For this reason, we assumed that the matings in both cohorts were contemporaneous, and therefore may be considered generally comparable for our purposes. Thus this analysis primarily makes use of the 1979 cohort but will utilize data from the 1997 cohort to inform relationships among mothers, fathers and offspring.

Variables collected from each of the 1979 cohort surveys included (parental) age, height, weight, gender, race/ethnicity, total number of children born to date, total years of education to date and total net family income. BMIs were calculated using self-reported height and weight. Variables collected from the 1997 cohort surveys included (offspring) age, height, weight, race/ethnicity and gender, as well as maternal height, maternal weight, paternal height, paternal weight and maternal gravid age. Data on age, height and weight were used to calculate maternal and paternal BMIs, as well as the BMI at age 18 for the offspring. Details related to data cleaning and missing data handling may be found in the Appendix.

Approach

Our overall goal was to simulate the effects of AM and differential realized fertility (DRF) on the distribution of offspring BMIs. In order to accomplish this, we built a simulation model that reflects AM and DRF and generates offspring BMIs; we will refer to this as the 'baseline model'. This framework must then be modified to produce offspring BMIs under the removal of AM (random mating; RM), the removal of DRF (no DRF), or both. We then simulated from the framework under each of 4 scenarios (RM and no DRF; RM and DRF; AM and no DRF; AM and DRF) and used the results to assess our factors of interest.

In order to make it as realistic as possible, we used data from the NLSY cohorts to inform all aspects of the baseline model. The process of generating a collection of offspring BMIs under the baseline model (an *instantiation* of this model) is as follows (see fig. 1 for an overview):

(1) A group of women is followed across their lifetimes from adolescence, and their children (if any) are simulated. Rather than simulating the complex and collinear covariates of these women in an imperfect manner, we used a representative, cross-sectional population of women from the 1979 NLSY cohort as the basis for our baseline model, since we have empirical covariate information across the lifetimes of these women.

(2) In a given year and for a particular woman, the number of children born in our simulation is based on a mapping m_1 of time-dependent maternal covariates to an expected number of children; we will refer to this quantity as λ_t. The number of children born is drawn from a Poisson with expectation λ_t. If any children are born in a year, this is called a *birth event*. The mapping m_1 was informed using the 1979 cohort.

(3) For each birth event, a paternal BMI is drawn under AM; recall that we do not have corresponding paternal BMIs for the women in the 1979 cohort. This link between maternal covariates

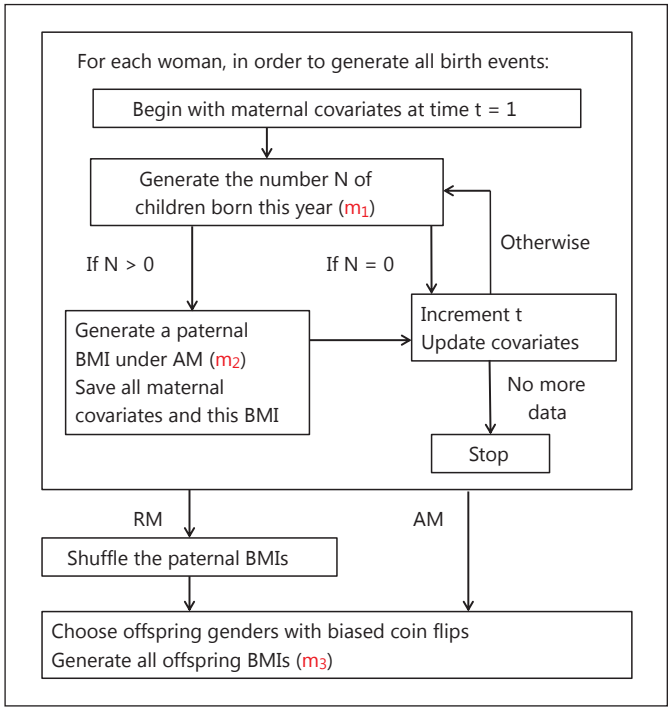

Fig. 1. A flow diagram for the operation of the Monte Carlo simulation framework. For a given woman, we generate some number N of offspring and, if any, a paternal BMI under AM, for each year under consideration. We then move forward in time by one year and repeat this until we have no more data for the woman under consideration. Once all birth events have been generated, offspring gender(s) and offspring BMI(s) are then simulated twice, once under these pairings (AM present), and once where the paternal BMIs have been shuffled across birth events to enforce RM.

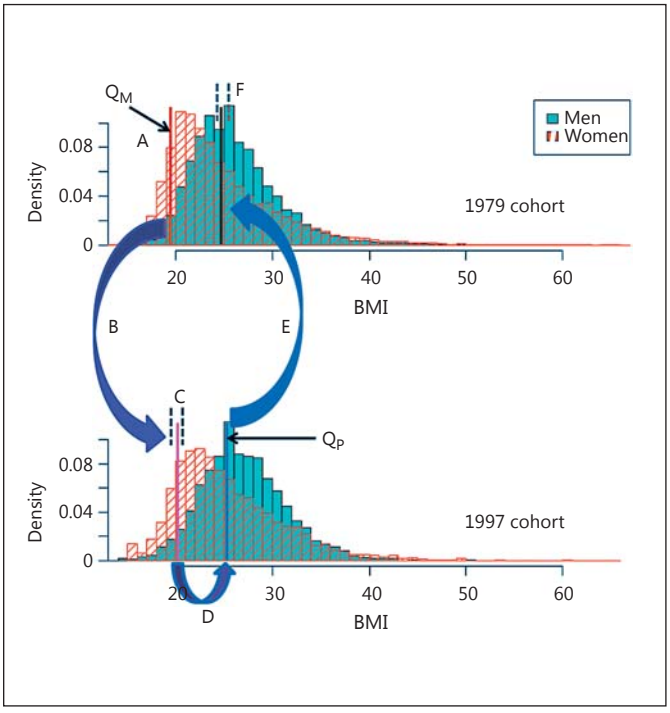

Fig. 2. An overview of the mapping m_2. Given a maternal BMI, we calculated the percentile that it corresponds to among all BMIs recorded in the 1979 cohort of women, calling it Q_M (A). We then gathered all mothers in the 1997 cohort that have a recorded BMI within a one-percentile window of Q_M (B) and chose one at random (C). The BMI of the selected woman's mate was then converted into a percentile Q_P (D), as calculated from the 1997 distribution of fathers. We then concluded the mapping by gathering all men in the 1979 cohort with BMIs within a one-percentile window of Q_P (E) and chose one at random (F) to be the mate for our original 1979 cohort mother.

and a paternal BMI was encoded in the mapping m_2 and was informed using both the 1979 and the 1997 cohorts.

(4) Given all birth events for all women, offspring genders are chosen via biased coin flips, where the probability of a male child is 0.5122 [27].

(5) Lastly, offspring BMIs at age 18 are sampled through the mapping m_3, which maps parental covariates and offspring gender to an offspring BMI. This mapping m_3 was informed by the 1997 cohort.

Once the baseline model has been established, our factors of interest can be removed from it. AM may be removed by shuffling paternal BMIs within race/ethnicity after step 3 has been completed for all women, producing RM with respect to BMI. DRF may be removed from the model by replacing m_1 with a new mapping m_1*, which does not use maternal BMI as an input.

Informing the Model Mappings

The details of the 3 mappings are as follows: m_1 models the number of children born in a given year (λ_t) as a function of maternal age, BMI, race/ethnicity, income and education as well as the number of children born to date and born in the previous year. This number can be (and usually is) zero and some women will never have any children over their simulated lifetimes. Since the model draws numbers of children born from a Poisson, we used Poisson regression to map a set of maternal covariates to a value of λ_t. We considered quadratic, cubic and quartic effects in addition to linear effects when modeling age, maternal BMI, children born to date and number of children born in the previous year, as linear effects alone are insufficient to capture the data. Years of education were reduced to whether or not more than a high school education has been achieved, as the more expansive measure of education was not a better predictor in the regression. Similarly, total net family income was trichotomized into USD <25,000, USD 25,000–75,000 and USD >75,000 in a given year. Covariate BMIs were centered at 25, the traditional boundary between normal and overweight [28], for ease of interpretation.

Mapping m_2 needs to map a mother's BMI and race/ethnicity, as recorded in the 1979 cohort, to a comparable paternal BMI under AM. Recall that mate BMI information is not available in the 1979 cohort, but BMIs for the parents of the 1997 cohort are avail-

able. While we have a suitable pool of male BMIs to draw upon in the 1979 cohort (from the men of the representative, cross-sectional sample), m_2 must match each mother with a mate in a manner that maintains the degree of AM observed in the 1997 cohort. As there are differences in BMI distributions across race/ethnicity, within this mapping we restricted all pairings to individuals of the same race/ethnicity.

Mapping m_2 is as follows (we provide fig. 2 as a visual aid and will reference its labels here): given a maternal BMI, we calculated the percentile that it corresponds to among all BMIs recorded in the 1979 cohort of women – calling it Q_M. We then gathered all mothers in the 1997 cohort that have a recorded BMI within a one-percentile window of Q_M and chose one at random. For example, if an African-American mother's BMI corresponded to the 80th percentile in the 1979 cohort, we gathered all African-American mothers from the 1997 cohort whose BMIs fell between the 79th and 81st percentile and chose one at random. The BMI of the selected woman's mate was then converted into a percentile Q_P, as calculated from the 1997 distribution of fathers. We then concluded the mapping by gathering all men in the 1979 cohort with BMIs within a one-percentile window of Q_P and chose one at random, to be the mate for our original 1979 cohort mother. While laborious, this approach allowed us to generate pairings within the 1979 cohort under AM.

For m_3, drawing an offspring BMI given the parental BMIs, gravid age and offspring gender/race/ethnicity was performed by gathering all 1997 parent-offspring triads that match the given set of covariates and randomly selecting one of them. We allowed the parental BMIs and gravid ages in our selection pool to lie within a window around the actual covariate values. These windows were set to be small (±0.5 BMI units or years as appropriate) but were allowed to grow if an insufficient number (less than 50) of matching 1997 records was available.

For both m_2 and m_3, we determined the covariates to be used in the mappings by variable selection via linear regression applied to the 1997 cohort data; recall m_1 was informed through Poisson regression. In all regressions, a full model containing all pairwise interactions was initially considered and then pared down using backwards selection until only terms that had p values <0.1 remained; terms required by the hierarchy principle (e.g. the constituent main effects of an interaction) were also retained. As aforementioned, in addition to m_1, a mapping m_1* was derived for scenarios without DRF by removing the main effect for maternal BMI as well as all of its interactions from the backwards selection (table 1). All regressions, model selection procedures and random draws using the empirical mappings were performed in R 2.14.2 [29].

Comparing Scenarios through Simulation

We wish to compare 4 different scenarios through simulation to quantify generational effects of AM and DRF. These scenarios are: AM on a background of no DRF, RM on a background of no DRF, AM on a background of DRF, and RM on a background of DRF. DRF may be included or excluded from the simulation through the use of m_1 or m_1*, respectively, and paternal BMIs may be shuffled to induce RM. We utilized observed covariate information from a representative sample of women aged 14–22 in 1979 in order to make our simulations realistic; this ensured that covariates remained collinear and changed over time, without having to build these complex dynamics into our simulation. However, since we were simulating birth events, all empirical data detailing those events were stripped out, so that no women entered into our simulation with prior birth events.

Because the simulation framework is not a deterministic process under all 4 scenarios, the distribution of offspring BMIs at age 18 varies across realizations of the simulation process. Therefore, the Monte Carlo simulation model was instantiated 300 times per scenario, in order to quantify and minimize this uncertainty. The resulting distributions of observed BMIs are compared based on differences in the mean BMI, differences in the spread about that mean (BMI variance), and differences in the prevalence of obesity and morbid obesity. Significance and 95% confidence intervals (CI) for these comparisons across scenarios (for example, between the baseline scenario and one without AM by BMI) were obtained via Welch's t tests unless otherwise noted; note that with a common DRF/no DRF background, instantiations of RM and AM outcomes are paired and hence paired t tests were used in these cases. As these tests are assessing stochastic variation arising from the nondeterministic nature of our model (see Discussion) and significant results can hence be associated with smaller and smaller p values by increasing the number of simulation instantiations, we do not report p values; these are all <0.05 whenever significance is indicated and usually on the order of 10^{-5}–10^{-29}.

For the prevalence of obesity, we present the relative risk (RR) of obesity where it is relative to the scenario without one or both factors of interest. In all cases, inference is based on the log RR and then back-transformed. In comparisons where instantiations were paired, we had an RR estimate for each instantiation and mad statistical comparisons via t tests on the log RR. In cases where instantiations were not paired, we pooled subjects within scenarios, across instantiations, and treated these as 2 big populations. A point estimate of the RR and the asymptotic standard error of the log RR were then based on the 4 counts of obese and nonobese over the 2 scenarios under consideration using a Z test.

Results

The regression results that inform the m_1 and m_1* mappings may be found in table 1. Figure 3 illustrates some results under m_1, specifically the probabilities of having at least one child as a function of maternal age for Caucasian women with a BMI of 25 and an education at the high school level or less under different conditions. Figure 3a shows curves for poorer households (total net family income USD <25,000). Figure 3b is for wealthier households (total net family income USD >75,000). The plotted curves are given for childless women, women who had their first child last year, women with 1 child not born last year, women with 2 children and 1 of them born last year and women with 2 children and neither of them born last year.

We note some characteristics of our baseline model, which reflect the NLSY data sets as closely as possible. First, existing offspring are more associated with new births in poor families than in wealthy households. Thus,

Table 1. Model fit for log number of offspring (mappings m_1 and m_1*)

Model term	Coefficient (SE) for m_1	Coefficient (SE) for m_1* with maternal BMI removed
Intercept	–3.451 (0.369)	–3.710 (0.340)
Age	0.562 (0.222)	0.611 (0.213)
Age^2	–0.074 (0.041)	–0.079 (0.040)
Age^3	0.004 (0.003)	0.004 (0.003)
Age^4	–0.00008 (0.00007)	–0.00009 (0.00007)
BMI	0.066 (0.030)	0[a]
BMI^2	–0.003 (0.0004)	0[a]
BMI^3	0.00006 (0.00001)	0[a]
Children BTD	1.501 (0.071)	1.523 (0.071)
BTD^2	–1.183 (0.067)	–1.194 (0.067)
BTD^3	0.292 (0.020)	0.295 (0.020)
BTD^4	–0.022 (0.002)	–0.022 (0.002)
Children PRV	–0.732 (0.156)	–0.731 (0.155)
PRV^2	0.338 (0.140)	0.339 (0.139)
Income USD 25,000–75,000	–1.228 (0.717)	–1.216 (0.740)
Income USD >75,000	0.625 (1.882)	0.727 (1.727)
R/Eth (AA)	0.191 (0.041)	0.233 (0.040)
R/Eth (Hisp)	0.150 (0.046)	0.177 (0.046)
Education	–0.659 (0.043)	–0.702 (0.042)
Age × BMI	–0.019 (0.012)	0[a]
Age^2 × BMI	0.002 (0.001)	0[a]
Age^3 × BMI	–0.0001 (0.00007)	0[a]
Age^4 × BMI	0.000002 (0.000001)	0[a]
Age × income 25,000–75,000	–0.255 (0.372)	–0.273 (0.388)
Age × income >75,000	–1.062 (0.519)	–1.122 (0.479)
Age^2 × income 25,000–75,000	0.088 (0.063)	0.092 (0.066)
Age^2 × income >75,000	0.181 (0.059)	0.189 (0.056)
Age^3 × income 25,000–75,000	–0.006 (0.004)	–0.006 (0.004)
Age^3 × income >75,000	–0.010 (0.003)	–0.010 (0.003)
Age^4 × income 25,000–75,000	0.0001 (0.00009)	0.0001 (0.0001)
Age^4 × income >75,000	0.0002 (0.00007)	0.0002 (0.00007)
BMI × education	0.026 (0.005)	0[a]
BTD × income 25,000–75,000	–0.201 (0.031)	–0.213 (0.031)
BTD × income >75,000	–0.303 (0.081)	–0.317 (0.081)
BTD × R/Eth (AA)	–0.048 (0.026)	–0.060 (0.026)
BTD × R/Eth (Hisp)	0.027 (0.028)	0.024 (0.028)
BTD × education	0.226 (0.027)	0.246 (0.027)
R/Eth (AA) × income 25,000–75,000	–0.474 (0.067)	–0.464 (0.066)
R/Eth (AA) × income >75,000	–0.259 (0.192)	–0.226 (0.190)
R/Eth (Hisp) × income 25,000–75,000	–0.209 (0.068)	–0.206 (0.068)
R/Eth (Hisp) × income >75,000	–0.229 (0.182)	–0.229 (0.182)
Education × income 25,000–75,000	0.502 (0.056)	0.500 (0.056)
Education × income >75,000	0.611 (0.136)	0.588 (0.136)

[a] This term was explicitly set to zero. BTD = Born to date; PRV = born in the previous year; R/Eth = race/ethnicity; AA = African American; Hisp = Hispanic.

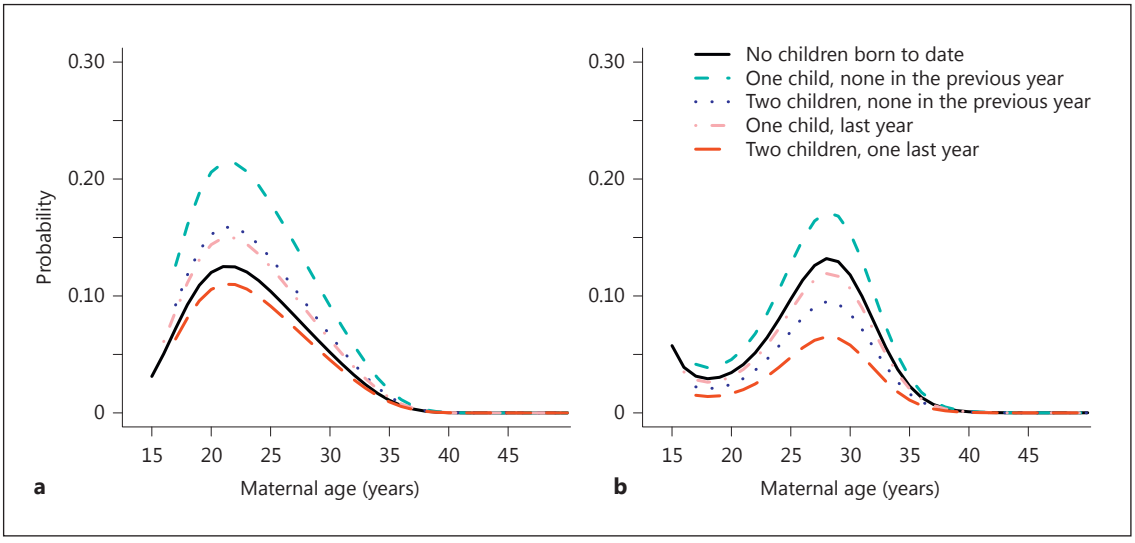

Fig. 3. Selected curves denoting the probability of having any children for Caucasian women with BMIs of 25 and a high school education or less, as a function of maternal age and previous history of childbirth events. **a** Poorer households (total net family income: USD <25,000). **b** Wealthier households (total net family income: USD >75,000).

a mother with 2 children is more likely to have another child than a childless woman if she is poor, but the reverse holds if she is wealthy. Second, higher BMIs are associated with higher overall realized fertility, but in a nonlinear fashion: the highest realized fertilities are among overweight women (BMI roughly between 25 and 30), the second highest among 'normal' and obese women (BMIs between 20 and 25 and BMIs between 30 and 35), the third highest for the morbidly obese (BMI >35) and the least among the low end of 'normal' and underweight women (BMI <20). Lastly, a correlation of 0.2 is observed between maternal and paternal BMIs in the 1997 cohort data. Thus our model, as informed by the NLSY data sets, reflects aforementioned relationships that agree with the literature [12, 23].

Comparisons of Scenarios

Monte Carlo computation quantified the model effects of these relationships on the next generation. Recall that the resulting distributions of observed offspring BMIs were compared based on differences in mean BMI, differences in the spread about that mean (BMI variance), and differences in the prevalence of obesity and morbid obesity. Visual summaries of the point estimates and their corresponding 95% CIs are presented in figure 4.

First, we compared the effect of DRF or no DRF on the distributions of offspring BMI. Under RM, DRF was associated with an increased mean offspring BMI (+0.046, 95% CI: 0.035; 0.057) and an increased prevalence of obesity (RR 1.020, 95% CI: 1.011; 1.028) compared to no DRF. The variances of the 2 distributions were not significantly different (+0.116, 95% CI: –0.038; 0.270). Under AM, these associations were strengthened: DRF was associated with an increased mean offspring BMI (+0.056, 95% CI: 0.045; 0.067) and an increased prevalence of obesity (RR 1.019, 95% CI: 1.011; 1.028) compared to no DRF; the difference in variances remained nonsignificant (+0.102, 95% CI: –0.068; 0.271).

Next, we compared RM and AM; recall that these comparisons were paired because they arise from the same set of birth events in every instantiation. When the birth events were generated with DRF excluded from the model (under m_1*), the distributions of offspring BMI under RM versus under AM slightly differed in the mean BMI (+0.011, 95% CI: 0.0003; 0.0217). The spread about that mean, however, was significantly higher under AM (+0.475, 95% CI: 0.322; 0.629), due to a higher prevalence of very low and very high BMIs, relative to the mean. This was therefore associated with an increased prevalence of BMIs >30 (RR 1.012, 95% CI: 1.004; 1.021) and >40 (RR 1.074, 95% CI: 1.039; 1.109). When DRF influenced birth events, AM was associated with an increase in the mean BMI (+0.021, 95% CI: 0.010; 0.032) as well as spread about that mean (+0.461, 95% CI: 0.300; 0.622), and the preva-

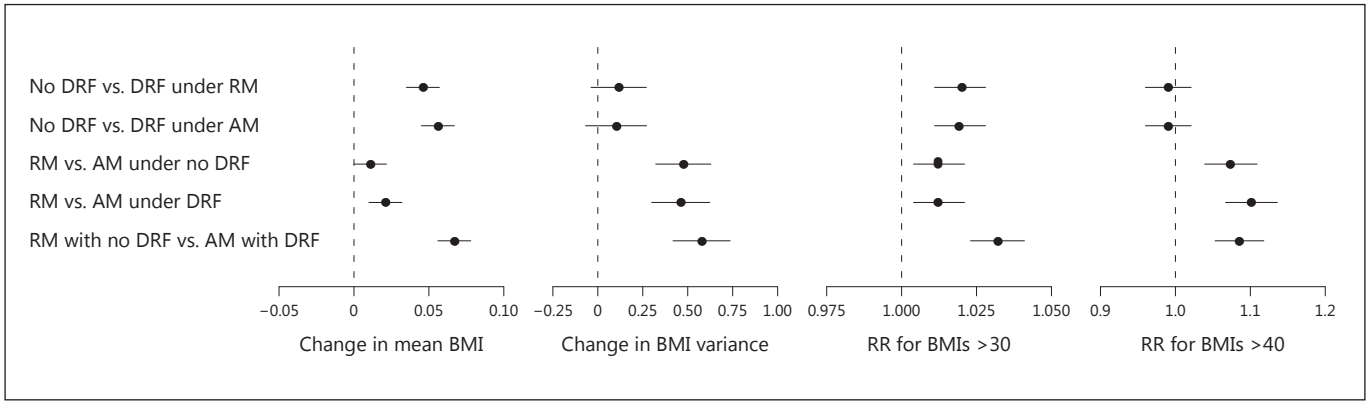

Fig. 4. Visual summary of change in mean BMI, in BMI variance and in RR for BMIs >30 and >40 in each of 5 scenarios: AM on a background of no DRF, RM on a background of no DRF, AM on a background of DRF, RM on a background of DRF, and a comparison of RM combined with no DRF against AM with DRF.

lence of BMIs >30 (RR 1.012, 95% CI: 1.004; 1.021) and >40 increased by an even greater degree (RR 1.101, 95% CI: 1.067; 1.136).

Lastly, we compared the scenarios of no DRF under RM versus DRF under AM, in order to examine them in concert. We found that both factors together were associated with an increased mean BMI (+0.067, 95% CI: 0.056; 0.078), an increased BMI variance (+0.578, 95% CI: 0.418; 0.736), and an increased prevalence of BMIs >30 (RR 1.032, 95% CI: 1.023; 1.041) and >40 (RR 1.085, 95% CI: 1.053; 1.118).

Discussion

We found significant associations between realized fertility by BMI and AM by BMI concerning the distribution of BMIs in the next generation. Both the mean BMI and the prevalence of obesity were evidenced as being shifted to higher levels by these factors.

Our approach has several strengths. Our framework is empirical and informed by large, nationally representative samples of longitudinal data collected over several decades and our framework closely emulates the empirical data so that, for instance, realized fertility declines to zero with advancing maternal age. In addition, we are not restricted to married couples or families where there are children: we follow teenaged women forward in time, they may get married or not, and they may have children or not. We empirically modeled offspring BMI rather than making parametric model assumptions that might have invalidated our inference. Lastly, simulation of this nature is one of the only ethical ways to examine factors that may influence obesity over generations, as randomized controlled trials that examine these factors are not feasible in humans.

On the other hand, our approach has some limitations that should be noted. As aforementioned, we used the 1997 NLSY cohort to inform some portions of our model since mate BMI information was not available in the 1979 cohort, and we further assumed that the 2 NLSY data sets were comparable for these purposes. Parental BMIs were only recorded once in the 1997 cohort, and so we assumed that their BMIs at a given youth's birth were comparable to that pair of measurements; our use of percentiles when moving across cohorts aimed to address this concern.

The model reflects the data well with a few caveats. We had to put in an explicit stopping rule for the rare times when λ_t was calculated to be greater than 3 (this occurred in less than 1 out of every 13 million calculations of λ_t) to prevent unduly large (e.g. triple digit) multiple birth events in a single year. Our modeling of offspring BMIs in the third mapping m_3 was limited by the covariate information that was available in both cohorts. We took into account differential birth rates for the 2 genders [27] but did not take into account higher mortality before age 18 in male youths [30].

Other factors inherent to our model should be taken into consideration when interpreting our results. First, the BMI is an imperfect indicator of adiposity. Extension of these results outside of the populations and generations sampled by the NLSY must be done with caution, as

we only had 1 generation of matings to consider. Lastly, we cannot make causal claims, as longitudinal surveys are observational in nature.

There are 2 sources of variation in our model for the point estimates of mean BMI shifts and increases in the prevalence of obesity. One of these is the stochastic nature of the simulation model, and our p values reflect an uncertainty due to this source; because of our large number of simulations per scenario, this variation is fairly minimal and well accounted for. In addition, however, there is an uncertainty associated with the fact that our inference is based on a single representative sample, and the simulation framework described previously does not take that into account. Properly accounting for this source of variation, e.g. via bootstrapping, is a topic for future research.

Conclusion

In summary, a small portion of the prevalence of obesity from one generation to the next is associated with the presence of DRF and AM by BMI. Further research using multigenerational data sets is required to fully ascertain the degree of association of these factors on population-level obesity.

Appendix

Data Cleaning and Missing Data

Like all longitudinal surveys, the NLSY data sets contain errors, omissions and values that are implausible or logically inconsistent or both. These issues needed to be addressed and we briefly outline our methods for doing so below. When we say that a value was 'rejected', we mean that it was replaced by a missing value indicator to be addressed through multiple imputation.

Implausible Values. Heights shorter than 75 cm (2′6″) and taller than 244 cm (8′) were rejected. Any weight recorded as 996 was rejected; while not explicitly decoded in the NLSY codebooks, a value of 996 appears to be an 'out-of-range' indicator.

Logically Inconsistent Values. Some of the measurements under consideration, such as years of education or total number of offspring born to date, are naturally monotone: they should not decrease as one moves forward in time. This property was used to fill in values that were nominally missing but could be deductively inferred. For example, if a mother had 2 children in total in 1981 and also in 1983 but a missing value is recorded for 1982, we replaced that omission with the value '2'. Similarly, there are data entries that are not missing but exhibit non-monotone behaviors, such as offspring vanishing from the longitudinal record. Roughly half of these violations reflect discrepancies in the 1979–1981 offspring totals compared to those taken after 1981; in these cases, we used the 1982 values carried backward. In the remaining cases where the offenses were not as systematic, invalid values were rejected.

Nonsurvey Years. Our framework models birth events on an annual basis but the NLSY surveys were not undertaken in every year and, furthermore, some variables were not collected in more than a handful of years (e.g. in the 1979 cohort, height was only recorded in 6 calendar years). To ameliorate these issues, height was carried forward and all other values were interpolated when possible and considered to be missing values when not.

1997 Parental BMIs. The measurements used to calculate parental BMIs in the 1997 cohort were not recorded as 'maternal height' or the like. Rather, information on sex, height and weight were recorded for the respondent adult and possibly for nonrespondent biological parents (NRBPs) No. 1 and No. 2 (sometimes the adult queried at the household was not a biological parent of the youth in question). In cases where only NRBP No. 1 was indicated but that parent's sex matched that of the primary respondent, only the information for NRBP No. 1 was used to ascribe either maternal or paternal BMI information (i.e. we did not consider the respondent to be a biological parent). When there were 2 NRBPs and both were listed as the same sex, both parental BMIs were treated as missing. Fortunately, the gravid age of the mother was recorded separately and did not need to be inferred in this manner.

Youth BMI at Age 18. The BMI was missing at age 18 for some youths. If the BMI could be calculated for either age 17, 19 or both, an average of the available BMIs was used. If not, an average of the BMIs available for ages 15–21 was used, if possible. If all of those were missing, a missing value was recorded and was later addressed during the multiple imputation.

Missing Data. In order to address the presence of missing data, we performed multiple imputation in SAS v.9.3 [31] on the 1979 and 1997 data sets separately after cleaning. Specifically, the Markov Chain Monte Carlo incarnation of PROC MI was used, with the number of imputations chosen in order to obtain 95% efficiency, based on the highest fraction of missing information by expectation-maximization [32]. This resulted in 4 and 13 imputations for the 1979 and 1997 cohort data sets, respectively. Additionally, predictive mean matching was employed so that the imputed values would be plausible.

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