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Recent origin and evolution of obesity-income correlation across the United States

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ABSTRACT From a gene-culture evolutionary perspective, the recent rise in obesity rates around the Developed world is unprecedented; perhaps the most rapid population-scale shift in human phenotype ever to occur. Focusing on the recent rise of obesity and diabetes in the United States, we consider the predictions of human behavioral ecology (HBE) versus the predictions of social learning (SL) of obesity through cultural traditions and/or peer-to-peer influence. To isolate differences that might discriminate these different models, we first explore temporal and geographic trends in the inverse correlation between household income and obesity and diabetes rates in the U.S. Whereas by 2015 these inverse correlations were strong, these correlations were non-existent as recently as 1990. The inverse correlations have evolved steadily over recent decades, and we present equations for their time evolution since 1990. We then explore evidence for a “social multiplier” effect at county scale over a ten-year period, as well as a social diffusion pattern at state scale over a 26-year period. We conclude that these patterns support HBE and SL as factors driving obesity, with HBE explaining ultimate causation. As a specific “ecological” driver for this human behavior, we speculate that refined sugar in processed foods may be a prime driver of increasing obesity and diabetes.

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Introduction

In the United States, where adult obesity prevalence rates have been rising since the 1970s (Kranjac and Wagmiller, 2016), about two-thirds of adults are now overweight and over 100,000 U.S. deaths per year are attributed to obesity (Ogden et al., 2014). With obesity rates having tripled in many U.S. states over the past 25 years, this rise in obesity prevalence has accelerated. In 1990, about 11% of a typical U.S. state population was obese and no state had more than 15% obesity in its adult population. By 2015, U.S. obesity rates had more than doubled, with several states above 35% adult obesity and no state below 20% obesity in the population (Centers for Disease Control and Prevention, 2017a). In one generation, the change has been so dramatic that the obesity rate in any U.S. state in 2015 would have been an extreme outlier in the U.S. in 1990.

From a gene-culture evolutionary perspective, the recent rise in obesity rates, occurring across the Developed world (Goryakin et al., 2017), is unprecedented. In the past, human niche construction evolved over a time scale of centuries or millennia (Creanza and Feldman, 2016; Milot et al., 2011). For example, the evolution of lactase persistence among Neolithic populations of central Europe was rapid in evolutionary terms but nevertheless took place over thousands of years, in coevolution with the intensification of dairying economies (Brock et al., 2015; Gerbault et al., 2013). In contrast, industrially-processed foods have transformed Western human diets in less than a century. Not only has this made calories and junk food abundant and inexpensive in high-income countries, but there appear to be other effects such as reduction of gut microbiome diversity (Smits et al., 2017; Muscogiuri et al., 2018).

In the simplest view, obesity in Developed economies is a result of over-abundance of inexpensive food calories combined with decreases in daily physical activity in the industrialized world and its built environment (Mattson et al., 2014; Mullan et al., 2017). Negative energy balance is not the only factor, however, and with heterogeneity across socioeconomic groups, the specific causes for the rapid and recent increase in U.S. obesity remain unclear (Cook et al., 2017; Dwyer-Lindgren et al., 2013; Flegal et al., 2016).

One thing that is clear in high-income countries is that, despite decades of economic growth, obesity disproportionately affects the poor—the “poverty-obesity paradox” (Hruschka and Han, 2017). The proportion of obese individuals in industrialized nations now correlates inversely with median household income. This phenomenon is called the “reverse gradient” because it is the reverse of the pattern in developing countries, where higher income correlates with higher body mass. In the United States and other developed countries, lower income households tend to have higher rates of obesity (Hruschka, 2012; Subramanian et al., 2011). In 2015, over 35% of the population was obese in U.S. states where median household incomes were below \$45,000 per year, whereas obesity was less than 25% of state populations where median incomes were above \$65,000 (Centers for Disease Control and Prevention, 2017c). Similarly in Europe today, poor individuals are 10% to 20% more likely to be obese (Salmasi and Celidon, 2017). This pattern is unique to Developed economies; within China, for example, an inverse correlation between income and obesity/diabetes is observed only in the most economically developed regions (Tafreschi, 2015).

Cultural evolution potentially offers a less proximate, more ultimate explanation for the recent rise in obesity. Evolutionary approaches to behavioral change include human behavioral ecology and cultural evolutionary theory; the former tends to prioritize optimality of adaptive behavior while the latter tends to prioritize social learning. Generally speaking, human behavioral ecology (HBE) emphasizes the plasticity of human physiology

and behavior, by which individuals minimize risk to survival and optimize their long-term reproductive payoffs (Higginson et al., 2017). As wealth mitigates survival risk, HBE predicts a positive correlation between BMI and wealth, as humans have evolved to store calories as insurance against future famine or food shortage (Shrewsbury and Wardle, 2012; Higginson et al., 2017; Tapper, 2017). In the poorest 80% of the world’s societies, body mass index (BMI) generally increases with household wealth (Subramanian et al., 2011)—except below about 400 USD per capita, when poverty is such that BMI is uniformly low (Hruschka et al., 2014). In high-income countries, HBE predicts greater obesity among the poor, partly because humans have evolved behavioral “rules” that lead to overeating in rich environments and partly because poorer people have more immediate risks and concerns than outweigh long-term mortality risk of being obese (Dittmann and Maner, 2017; Dohle and Hafmann, 2017; Higginson et al., 2017; Mani et al., 2013; Smith, 2017).

The HBE hypothesis predicts that obesity has recently evolved in strong correlation with both the food environment and with income/wealth. The “Insurance Hypothesis” (Nettle et al., 2017) uses HBE to explain why the reverse is true in Developed countries where extreme BMI (obesity) is more frequent among the poor. Under the Insurance Hypothesis (IH), “individuals should store more fat when they receive cues that access to food is uncertain” (Nettle et al., 2017). Poor people in high-income countries receive such cues, as they experience more stress and greater existential risk for multiple reasons. Prominent among these risks is malnutrition, yet empty calories are still inexpensively available as processed foods and sugar-sweetened beverages (Bray et al., 2004; Johnson et al., 2007; Jürgens et al., 2005; Bocarsly et al., 2010). The IH is consistent with observations of women in high-income countries, who are more likely to be obese when confronted by food insecurity (Nettle et al., 2017). An alternative explanation, however, occurs at the societal level in high-income countries, where heavier women tend to marry into poorer households due to through “anti-fat discrimination” in marriage (Hruschka, 2012; Hruschka and Han, 2017).

In contrast, social learning (SL) explanations emphasize learned behavior in groups: behaviors are inherited from parents and learned socially from contemporaries through the generations of family traditions or community cultures (Bentley et al., 2016; Colleran and Mace, 2015; Colleran, 2016). Dietary habits are often determined as much by cultural traditions as they are by nutritional needs and family economics (Anderson and Whitaker, 2010; Anderson, 2012; Hughes et al., 2010; Lhila, 2011; Mata et al., 2017; Redsell et al., 2010; Vizireanu and Hruschka, 2018). Cultural factors may therefore underlie local differences in obesity and diabetes rates, which exhibit effects of local neighborhood and its built environment (Alvarado, 2016; Carroll et al., 2016; Mullan et al., 2017; Kowaleski-Jones et al., 2017), family size (Datar, 2017), ethnic group and age group (Cook et al., 2017).

Under SL, obesity may also increase through social influence. A widely-discussed argument, first presented by Christakis and Fowler (2007), is that obesity “spreads” through social influence in networks of family and friends (Christakis and Fowler, 2013). Relatedly, recent modeling and experimental studies show how a minority group can initiate rapid change in social conventions, provided the minority reaches a ‘critical mass’ (Centola et al., 2018; Couzin et al., 2011). Under SL, therefore, a new behavior can become a new social norm relatively quickly, if obesity were indeed a new social norm.

The alternative to the social-learning explanation is homophily, if obesity clusters in social networks merely because those clusters are similar people in the same environments (Shalizi and Thomas, 2011). Homophily could be viewed either as similar behavior

derived from shared cultural ancestry or else as similar behavior that reflects adaptations to similar environments. Outside of carefully monitored conditions (Centola et al., 2018; Hobaier et al., 2014), however, it is difficult if not impossible to distinguish social influence from homophily, even if obesity is observed to cluster in social networks, without a fine-grained temporal dimension to the data (Christakis and Fowler, 2013; Shalizi and Thomas, 2011; Thomas, 2013).

Unlike the small-scale social network study of obesity versus specific friends and kin members (Christakis and Fowler, 2007), this study examines annual, population-scale obesity rates aggregated by U.S. county. If the aggregated data are time-stratified, however, we can still attempt to test the SL hypothesis. We will use multiple measures (obesity, leisure, income) and ten years of county-scale data to assess any “social multiplier” effects. The social multiplier effect is identified when the rate of behavior among a group is greater than what would be predicted based on individual-scale variables alone. Identification of groups is a problem in the empirical literature on social interactions (Blume et al., 2011), but useful proxies have based on Zip codes (Corcoran et al., 1992) and census tracts (Weinberg et al., 2004).

A study of crime rates (Glaeser et al., 2003), for example, used statistics of individuals to predict crime rates and regressed those on crime rates in groups. This is how the social multiplier was defined at the county level, specifically by comparing the coefficient b in the regression,

$$\omega_{ig} = a + bx_i + \epsilon_i, \tag{1}$$

with the coefficient b' in its group counterpart

$$\bar{\omega}_g = a' + b'\bar{x}_g + \bar{\epsilon}_g, \tag{2}$$

where ω_{ig} denotes the choice of individual i in county g and x_i is a vector of observable individual-specific characteristics; the social multiplier is defined as the coefficient ratio, b'/b (Blume et al., 2011).

In this approach, estimating the social multiplier requires an estimate of individual-level rates, which do not exist in aggregated data. Faced with this problem, Glaeser et al., (2003) used nationwide arrest rates by age that, when combined with demographic data, provided a predicted level of crime in each neighborhood. These predicted rates were then regressed actual crime rates at the county level, yielding a coefficient of 1.7 at the county level, which was their estimate of the social multiplier at that scale of aggregation (Glaeser et al., 2003).

The data we use here are aggregated by U.S. county annually: over three thousand county-level time series of obesity, leisure and income rates over ten-year period (2004 to 2013). This amounts to ten sets of annual data, on several variables, for 3110 U.S. counties. If, controlling for the effects of household income, we find that lack of physical activity, or leisure rate, has a disproportionate effect on obesity rate in 2013 compared with 2004, then there may be support for the social multiplier effect. We have only aggregated statistics but we have the advantage of a time series. In principle there exists an individual-level, effectively physiological, connection between lack of exercise (leisure) and obesity rates, which we assume remains constant through time. The correlation between obesity and leisure rates should reflect this individual relationship as a baseline, plus any social multiplier effects over time.

In other words, change in the leisure-obesity correlation between 2004 and 2013 ought to reflect the social multiplier effect. As there are also unobservable connections between leisure and obesity, however, we follow the cautious approach of Glaeser

et al., (2003), who “take these results warily, as they may well overstate the true social multiplier.”

To investigate whether obesity increased in the classic S-shaped pattern consistent with social learning, we carried out regression analysis on the annual data for each state over the 1990–2016 period. A simple linear increase in obesity rate since 1990 serves as our null hypothesis, with the alternative hypothesis being a non-linear time trend. If the null hypothesis of non-linearity could not be rejected, by implication an S-curve would not be present in the data.

We applied two separate but complementary approaches. First, we tested the null of linearity against a general non-linear alternative, using the methodology of local linear regression (Cleveland and Devlin, 1988). We used the “loess” command in R. Local linear regression fits simple linear models to localized subsets of the data to describe the deterministic part of the variation in the data, point by point, without specifying a global functional form. An input parameter in the loess command (“span”) allows the “equivalent number of parameters” (ENP) to be varied. ENP serves as a measure of the non-linearity of the series. The approach enables ANOVA tests to be carried out of the null of linearity against a range of non-linear alternatives. Local linear regression is a powerful approach, but does not yield a specific functional form.

For the second approach, we tested the null of linearity against a specific non-linear functional form, namely that of a classic adoption curve (Bass, 1969; Bentley and Ormerod, 2010; Henrich, 2001). We have:

$$\frac{dF_{t,i}}{dt} = (\mu_i + q_i F_{t,i})(1 - F_{t,i}), \tag{3}$$

where $F_{t,i}$ is the obesity rate in year t of U.S. state i , μ_i is the chance in state i that a person becomes obese (i.e., BMI of 30 or above) through individual behavior and q_i is the probability within state i , where $F_{t,i}$ are already obese, that a person becomes obese through social influence. This ODE can be solved for $F_{t,i}$,

$$F_{t,i} = F_{0,i} + M \frac{1 - e^{-(\mu+q)t}}{1 + \frac{q}{\mu} e^{-(\mu+q)t}}, \tag{4}$$

where M is the magnitude of change and $F_{0,i}$ is set as the obesity rate of U.S. state i in year 1990. This equation can be fitted to the obesity data, with the goodness of fit reported as the adjusted R^2 statistic, defined as:

$$1 - \frac{(1 - R^2)(n - 1)}{n - \nu - 1}, \tag{5}$$

where ν is the total number of explanatory variables in the model (not including the constant term), and n is the sample size. The linear model has $\nu = 2$ parameters (slope and intercept), whereas the Bass model has $\nu = 3$ parameters (p , q , and M).

Here we focus on an under-studied, but revealing question: in the U.S., how has the correlation between household income and obesity changed in the past 25 years? In the U.S., obesity and diabetes rates currently have a strong negative correlation with household income (Hruschka, 2012). These correlations have been demonstrated cross-sectionally but not longitudinally, however, and therefore it is not possible to establish their causality (Boden and McLeod, 2017). In industrialized economies, the increase in obesity prevalence has been fastest among low income levels, as fast as tripling within a generation among certain subpopulations.

Data

Annual, age-adjusted data on obesity rates at the county level, for years 2004 to 2015, were obtained from the publicly accessible archive maintained by the Center for Disease Control and Prevention (www.cdc.gov/diabetes/data). For these county-level data, we also make use of the CDC age-adjusted estimates, (Klein and Schoenborn, 2001), in which rates are age adjusted to the 2000 U.S. standard population using age groups, 20–44, 45–64, and 65 or older (Centers for Disease Control and Prevention, 2017b). Older obesity data at the state level since 1990 were obtained from the annual reports of the Trust for America's Health and the Robert Wood Johnson Foundation (stateofobesity.org). For analyzing the time-series of state-level obesity rates, we cautiously added data from the years 1991 and 1998 from a different source (Mokdad et al., 1999) to examine more closely any potential non-linear change in the 1990s.

Due to missing data at the county level, we excluded Alaska from all analyses at county level, while including Alaska for state-level analysis. We also used five years of CDC data on age-adjusted annual CDC diabetes rates, 2009–2013, in U.S. counties (Centers for Disease Control and Prevention, 2017d). The estimates of diabetes rates are derived through telephone surveys, normalizes the data using population data from the US Census, and smooths the estimates such that three years of data are averaged in each annual estimate (Centers for Disease Control and Prevention, 2017c).

Importantly, we use age-adjusted rates for both obesity and diabetes in our analysis, and so we do not include the age profile of an area as an explanatory factor. This adjustment has already been carried out by the CDC in the data which we use. By using age-adjusted data, we minimize the effect of demographics in our results. To anticipate, we also note for reference that our results were essentially the same when we used data that were not age-adjusted.

Estimates of leisure-time physical inactivity come from the CDC Behavioral Risk Factor Surveillance System, a system of health-related telephone surveys, which began in 1984 with 15 U.S. states, and now collects data in all 50 states through over 400,000 adult interviews each year (Centers for Disease Control and Prevention, 2017c). The “leisure” statistic indicates the fraction of population who are designated as physically inactive, meaning they answered “no” to the question, “During the past month, other than your regular job, did you participate in any physical activities or exercises such as running, calisthenics, golf, gardening, or walking for exercise?”

Food desert data were recently made available through the Food Access Research Atlas (FARA) project (Rhone et al., 2017). The estimates are derived from the 2010 US Census and the 2010–2014 American Community Survey, in which census tracts are categorized by median income, vehicle availability, and Supplemental Nutrition Assistance Program (SNAP) participation (Rhone et al., 2017). To this geographic dataset are added two 2015 lists of supermarkets, supercenters, and large grocery stores to represent sources of affordable and nutritious food (Rhone et al., 2017). The FARA records for each U.S. Census tract the number and share of people more than a certain distance to a supermarket: in urban areas, that specified distance is half a mile or 1 mile, whereas in rural areas the distances are 10 miles or 20 miles (Rhone et al., 2017). Also recorded is whether the population in the census tract has overall low access to vehicles. Census tracts are also designated as rural, urbanized (over 50,000 people) or urban cluster (2500 to 50,000 people); for the purposes of estimating the urban/rural ratio of a county, we counted both urbanized and urban cluster tracts as being urban. Because food deserts are defined quite differently for urban (0.5 mile) versus

rural (10 miles) counties in the FARA, we consider just those counties whose populations we calculated as at least seven-eighths (87.5%) urban, totaling $n = 250$ counties across the U.S.

Results

We find the reverse gradient has only existed for less than thirty years. In the U.S. in 1990, when population-scale obesity rates were about a third of what they are today, there was no correlation between income and obesity or diabetes. The inverse correlations between income and diabetes/obesity rates have developed only within the past thirty years. By 2015, the correlation was stronger than ever: in states where median household incomes were below \$45,000 per year, like Alabama, Mississippi and West Virginia, over 35% of the population was obese, whereas obesity was less than 25% of state populations where median incomes were above \$65,000, such as in Colorado, Massachusetts or California.

In the U.S., there is considerable geographic heterogeneity in obesity prevalence. Figure 1 shows maps of obesity rates and diabetes rates by county in 2013 (Table 1). By 2013, the reverse gradient in the U.S. was pronounced; the simple correlation between obesity and $\ln(\text{income})$ across $n = 3110$ U.S. counties was $r = -0.486$ and between $\ln(\text{income})$ and diabetes was $r = -0.531$. All reverse gradients are better determined against the logged income data than against median income itself. Plots of $\ln(\text{income})$ against both diabetes and obesity (Fig. 2), aggregated at county level, reveal mild degrees of non-linearity in each of the relationships.

State-level data on annual obesity and diabetes rates, available for years 1990 through present, together with age-adjusted and inflation-adjusted income data from the U.S. Census, show how the reverse gradient in the U.S. changed over twenty-five years (Fig. 3). For the year 1990 (data from $n = 43$ states for this first year of data), the Pearson correlation between state-level obesity and the natural log of median household income was $r = -0.240$ [$-0.502, 0.061$], which is not significant ($p = 0.116$).

Twenty-five years later, a strong inverse correlation had developed between median household income and rates of obesity and diabetes (Figs. 3a, b). In 2015, the correlation between $\ln(\text{income})$ and obesity rate across all 50 states was $r = -0.697$ [$-0.816, -0.522$], which is highly significant ($p < 0.00001$); even limited to the 43 states also recorded in 1990, the correlation in 2015 still yields $r = -0.699$ [$-0.824, -0.508$] ($p < 0.00001$).

A similar change is evident for the reverse gradient involving diabetes and $\ln(\text{income})$; insignificant for 1990 ($r = -0.090$ [$-0.380, 0.216$], $p = 0.566$) and highly significant by 2015 with $r = -0.706$ [$-0.823, -0.532$] ($p < 0.00001$). Again, if we restrict the 2015 data to the 42 states available in 1990 (one state fewer than in the obesity data), the correlation between $\ln(\text{income})$ and diabetes rate yields $r = -0.684$ [$-0.816, -0.483$] ($p < 0.00001$).

Figures 3a, b show the actual and fitted values of the regressions of obesity and diabetes, respectively, on the log of income in both 1990 and 2015. In 1990, neither slope was significantly different than zero at the state level. In the regressions using 2015 state data, the slope coefficients for obesity are -0.163 ± 0.024 ($R^2 = 0.476$) and for diabetes -0.080 ± 0.011 ($R^2 = 0.491$). Figure 4 shows how the slope coefficient in these regressions has evolved, using data in 1990, 1995, 2000, 2003, and then annually from 2005 onwards.

Figure 5 shows how, at 5-year intervals from 1990 to 2015, the rate of growth in obesity or diabetes per year was inversely related to median household income. The temporal evolution of these reverse gradients, for the rates of obesity and diabetes,

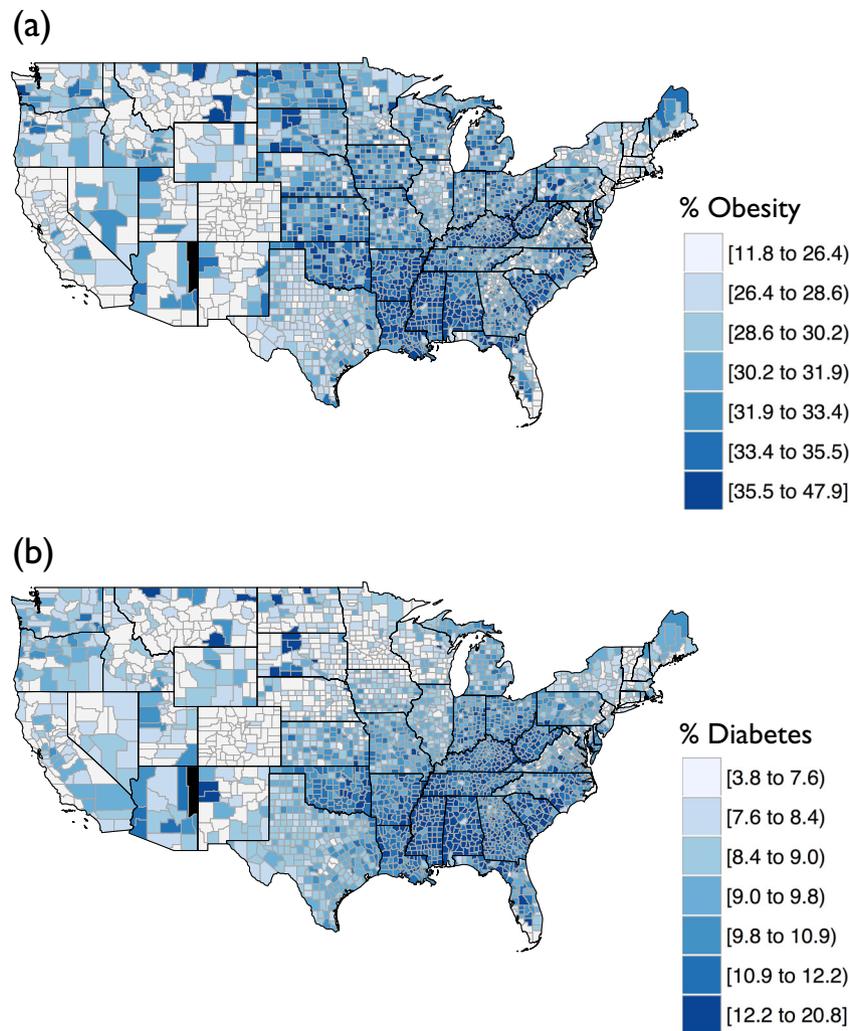


Fig. 1 Prevalence of adult **a** obesity and **b** diabetes in 2013, mapped at the scale of U.S. county for CDC age-adjusted figures

respectively, can be described by the following equations:

$$\text{Obesity rate} = (-0.0341 - 0.0049t)X_t + (0.4812 + 0.0609t), \tag{6a}$$

$$\text{Diabetes rate} = (-0.0022 - 0.0027t)X_t + (0.0639 + 0.032t). \tag{6b}$$

where X_t is the natural logarithm of median household income in year t . The colored lines in Fig. 5 show how well Eqs 6a and 6b represent the actual reverse gradients of $\ln(\text{income})$ versus obesity and diabetes rates, respectively, across 25 years of evolution of these negative gradients. The evolving slope coefficients imply that above an annual income level of \$250,000 for obesity and \$150,000 for diabetes, any further increases in income have negligible effects in term of further reducing obesity and diabetes (Fig. 5c).

Available data at the U.S. county level are available only from year 2004, so do not capture the start of this phenomenon, but these data include not only diabetes and obesity rates but also a “leisure” statistic derived from self-reported activity levels. Across all 3110 counties in any given year, the leisure statistic correlates best with both obesity but also with income, reflecting the feedback between income, health habits and obesity (Table 2). For the

leisure statistic in 2013, for example, the relationship with both obesity and diabetes is strongly positive and linear (Figs. 2c, d) with $r = 0.719$ [0.701, 0.735] for leisure versus obesity, and $r = 0.686$ [0.667, 0.704] for leisure versus diabetes.

We find that each year 2004 to 2013 the leisure and income statistics were a good predictor of ($r^2 > 0.5$) obesity rates at the county level (Table 2). Notably, the respective regression coefficient on leisure grew from about 0.40 to 0.63 (Table 2, Fig. 6). This means that if we applied the 2004 regression coefficient, the actual obesity rate would be 1.56 times our prediction based on 2013 leisure rate. Hence following Glaeser et al., (2003), we estimate the social multiplier as at least 1.56, since by 2004 there had already been a decade of sharp increase in obesity rates.

This steady increase change in coefficient (Table 2) is not due to change in the leisure rate, which rose and fell: the average county rate increased from 25.3% in 2004 to a peak of 26.9% in 2009 and then fell to 24.7% by 2013. We are not aware of an individual-level reason why the relationship between lack of exercise and obesity will have changed in ten years. We therefore posit 1.56 as a measure of social multiplier effect at county level.

For the time series of state-level obesity rates from 1990 to 2016, regression analyses serve to rule out any strong social-shaped pattern in obesity rise for any of the U.S. states. Figure 7 shows typical examples. Using the local linear regression approach (see Methods), the null hypothesis of linearity could not

Table 1 Values and correlations (adjusted R^2) across U.S. states, 2013

State (# counties)	Income ('09\$)	Diabetes, %	Obesity, %	Leisure, %	Inc-Obes: Adj. R^2 (p-value)	Inc-Diab: Adj. R^2 (p-value)	Leis-Obes: Adj. R^2 (p-value)
AL (67)	35,356	14.1	32.4	29.1	0.447 (0.000)	0.512 (0.000)	0.219 (0.000)
AZ (14)	40,148	10.6	26.8	21.5	-0.082 (0.918)	-0.080 (0.85)	0.712 (0.000)
AR (75)	33,960	12.0	34.6	32.1	0.177 (0.000)	0.067 (0.014)	0.118 (0.001)
CA (58)	50,436	8.6	24.1	17.3	0.315 (0.000)	0.023 (0.128)	0.564 (0.000)
CO (64)	47,473	6.1	21.3	16.0	0.259 (0.000)	0.136 (0.001)	0.754 (0.000)
CT (8)	63,014	8.6	25.0	19.5	0.853 (0.000)	0.494 (0.031)	0.602 (0.014)
DE (3)	51,783	10.8	31.1	24.4	0.325 (0.394)	0.067 (0.478)	0.793 (0.209)
FL (67)	39,892	11.1	26.4	25.6	0.451 (0.000)	0.368 (0.000)	0.726 (0.000)
GA (159)	37,751	11.2	30.3	26.1	0.145 (0.000)	0.175 (0.000)	0.262 (0.000)
HI (4)	55,366	7.3	21.8	18.0	-0.481 (0.887)	0.558 (0.159)	-0.001 (0.424)
ID (44)	41,307	8.5	29.6	20.3	0.127 (0.010)	0.037 (0.110)	0.339 (0.000)
IL (102)	46,024	9.7	29.4	22.9	-0.002 (0.405)	0.162 (0.000)	0.233 (0.000)
IN (92)	44,779	11.1	31.8	27.0	0.125 (0.000)	0.124 (0.000)	0.137 (0.000)
IA (99)	47,269	9.1	31.3	24.0	0.032 (0.040)	0.057 (0.009)	0.169 (0.000)
KS (105)	43,381	9.5	30.0	25.5	0.067 (0.004)	0.075 (0.002)	0.122 (0.000)
KY (120)	36,538	11.8	33.2	29.2	0.268 (0.000)	0.251 (0.000)	0.406 (0.000)
LA (64)	38,065	11.8	33.1	30.3	0.322 (0.000)	0.139 (0.001)	0.357 (0.000)
ME (16)	41,151	8.9	28.9	20.1	0.492 (0.001)	0.354 (0.008)	0.692 (0.000)
MD (24)	61,764	10.2	28.3	22.7	0.458 (0.000)	0.497 (0.000)	0.683 (0.000)
MA (14)	58,617	9.0	23.6	18.6	-0.036 (0.476)	-0.063 (0.637)	0.872 (0.000)
MI (83)	40,494	10.1	31.5	22.4	0.089 (0.003)	0.073 (0.007)	0.318 (0.000)
MN (87)	49,675	7.8	25.5	20.6	0.114 (0.000)	0.025 (0.075)	0.130 (0.000)
MS (82)	32,391	13.1	35.1	32.6	0.549 (0.000)	0.457 (0.000)	0.246 (0.000)
MO (115)	37,633	10.0	30.4	26.5	0.045 (0.012)	-0.001 (0.335)	0.118 (0.000)
MT (56)	40,409	8.1	24.6	22.8	0.023 (0.131)	0.127 (0.003)	0.389 (0.000)
NE (93)	44,380	8.5	29.6	23.9	-0.010 (0.895)	0.013 (0.139)	0.008 (0.180)
NV (17)	49,898	8.2	26.2	20.8	-0.06 (0.901)	0.060 (0.174)	0.651 (0.000)
NH (10)	53,368	9.0	26.7	19.8	-0.07 (0.554)	0.020 (0.306)	0.390 (0.031)
NJ (21)	65,487	9.4	26.3	22.2	0.514 (0.000)	0.520 (0.000)	0.537 (0.000)
NM (33)	37,244	8.5	26.4	20.6	-0.00 (0.345)	0.080 (0.059)	0.666 (0.000)
NY (62)	48,904	9.2	25.4	22.5	0.384 (0.000)	0.132 (0.002)	0.030 (0.093)
NC (100)	38,732	11.2	29.4	25.4	0.254 (0.000)	0.243 (0.000)	0.576 (0.000)
ND (53)	51,489	9.2	31.0	24.9	0.069 (0.031)	0.033 (0.100)	0.007 (0.247)
OH (88)	44,130	11.2	30.4	26.3	0.183 (0.000)	0.383 (0.000)	0.318 (0.000)
OK (77)	39,806	11.9	32.5	30.4	0.010 (0.184)	0.219 (0.000)	0.212 (0.000)
OR (36)	42,097	9.3	26.5	16.3	0.021 (0.192)	-0.017 (0.525)	0.198 (0.003)
PA (67)	45,441	10.1	30.0	23.0	0.401 (0.000)	0.355 (0.000)	0.358 (0.000)
RI (5)	58,745	8.9	27.3	19.2	0.361 (0.168)	0.742 (0.038)	0.565 (0.088)
SC (46)	36,602	12.3	31.7	26.7	0.670 (0.000)	0.643 (0.000)	0.573 (0.000)
SD (66)	42,982	8.9	29.9	22.2	0.454 (0.000)	0.559 (0.000)	0.256 (0.000)
TN (95)	37,262	12.4	33.7	31.5	0.061 (0.008)	0.119 (0.000)	0.103 (0.000)
TX (254)	42,401	9.8	30.9	24.0	0.026 (0.005)	0.014 (0.030)	0.290 (0.000)
UT (29)	50,072	8.2	24.1	18.9	0.007 (0.278)	-0.026 (0.593)	0.328 (0.000)
VT (14)	46,037	7.6	24.7	18.9	0.401 (0.008)	0.324 (0.019)	0.692 (0.000)
VA (134)	48,040	NA	27.2	23.3	0.266 (0.000)	0.242 (0.000)	0.382 (0.000)
WA (39)	45,786	9.1	27.2	18.9	0.106 (0.024)	0.087 (0.038)	0.668 (0.000)
WV (55)	36,041	11.9	35.1	28.9	0.166 (0.000)	0.210 (0.000)	0.274 (0.000)
WI (72)	45,836	8.6	29.8	20.8	0.096 (0.012)	0.272 (0.000)	0.270 (0.000)
WY (23)	52,873	8.6	27.8	21.8	-0.04 (0.982)	-0.028 (0.533)	0.685 (0.000)

The three columns on the right show correlations between income, obesity and leisure across counties of each state

be rejected for seven of the 46 states examined (the few we excluded lacked data points for the early 1990s): Connecticut; Delaware; Iowa; Louisiana; Maine; Vermont; Wisconsin; Wyoming. For a further seven states, linearity could be rejected at the standard 5 per cent level, but only when the alternative exhibits a mild degree of non-linearity, with the Equivalent Number of Parameters being just 2.33: Idaho, Illinois; Kentucky; North Dakota; Oregon; Virginia; West Virginia. In the adoption-curve analysis, this favors “r-curves” of individual learning (Fig. 7). Adjusted r -squared values (Table 3) are strong where the individual parameter p is the same magnitude as the social parameter q , and fit almost as well with the “social” parameter, q , set to zero (Table 3) to represent pure individual learning.

Lastly, the effect of food deserts is also evident, among a subset of U.S. counties (Fig. 8, Table 4). As described in our Methods, from the FARA data we consider just those counties whose populations we calculated as at least seven eighths (87.5%) urban, totaling $n = 250$ counties across the U.S. In these urban counties, the regression of obesity rates versus the share of population without access to supermarkets within half a mile yields Pearson’s $r = 0.292$ [0.175, 0.401], which is significant ($p < 0.00001$). For diabetes, however, the correlation with this food desert variable in 2013 is not significantly different from zero ($p = 0.223$). For these same urban counties, food deserts among low income census tracts has stronger correlation with obesity (Fig. 8b), with $r = 0.563$ [0.472, 0.642], and now a significant correlation with

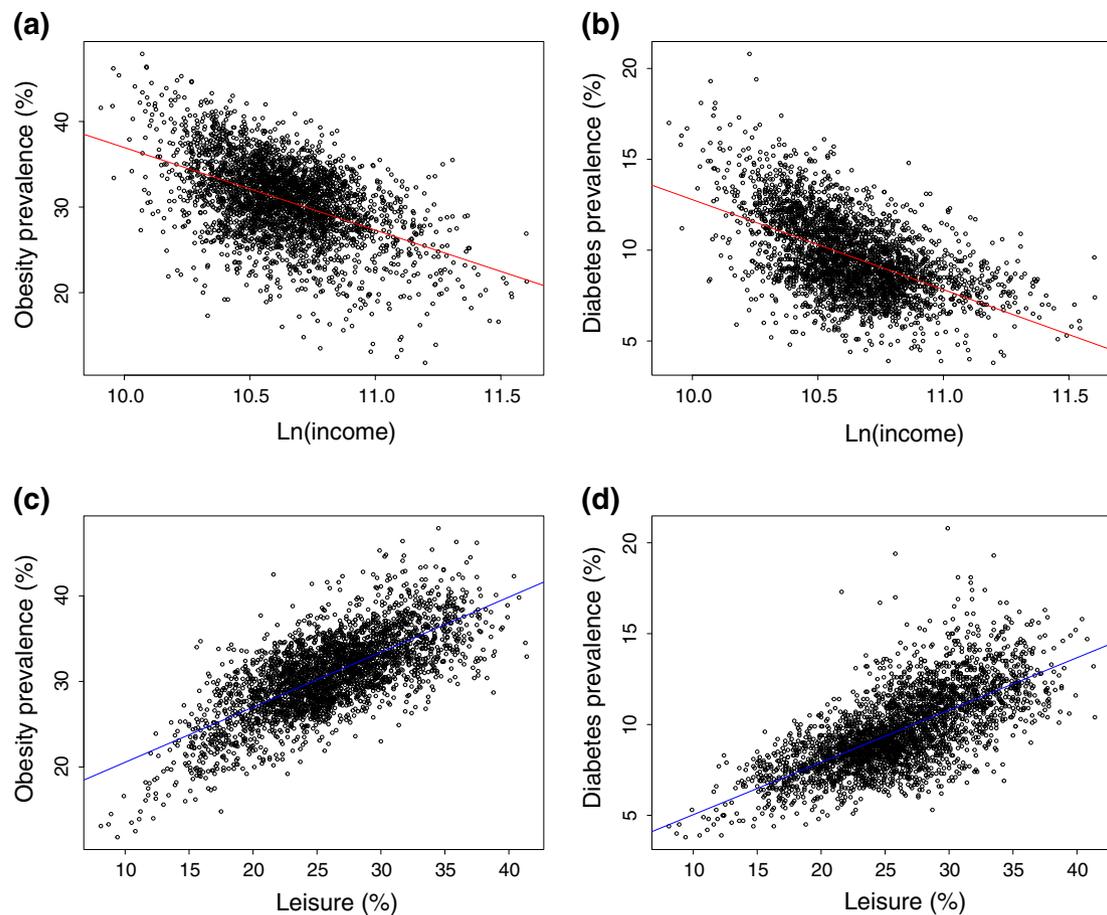


Fig. 2 Reverse gradients across U.S. counties in 2013 (in all states except Alaska) between the natural logarithm of household income and rates of **a** obesity (regression slope = -9.66) and **b** diabetes (slope = -4.93). Also shown are correlations between prevalence of physical inactivity (“leisure”) versus **c** obesity (slope = 0.643) and **d** diabetes (slope = 0.288). Correlation values within each state are listed in Table 1

diabetes rate $r = 0.462$ [0.359, 0.544]; both correlations are highly significant ($p < 0.000001$).

Food deserts are, of course, closely related to income. We confirm the validity of the individual correlations between obesity and diabetes and income and leisure in Table 2 by running simple multiple regressions of obesity and diabetes on income and leisure. The results are set in Table 5. Although each overall fit could be slightly improved with mild non-linearities, the simple regressions show that both $\ln(\text{income})$ and leisure have significant effects on obesity and diabetes.

Our analyses here use data aggregated from both sexes, as the same correlations for each sex were quite similar to the results for both sexes aggregated together. In 2013 for example, among the 43 states with sufficient data points for men and women (excluding AK, CT, DE, HI, NH, and RI), the reverse gradient between income and diabetes is significant for both sexes at $p < 0.01$ in 30 states. In some states, however, there are small differences. In eight states the reverse gradient in 2013 is significant at $p < 0.01$ for one sex and $p < 0.05$ for the other. In Arizona, New Mexico, New Jersey and Utah, the reverse gradient is significant for men but not for women, whereas in Massachusetts, it is significant for women but not for men.

Discussion

Here we have explored the origins and development of the inverse correlation between household income and obesity/diabetes rates in the U.S. We used data on mean household incomes and rates

of obesity and diabetes at the level of U.S. state, which date back to 1990, as well as county level statistics that offer larger sample sizes and higher spatial resolution but only extend back the early 2000s.

Using age-adjusted U.S. data on mean household incomes and rates of obesity and diabetes—at state level since 1990 and county level since the mid 2000s—we found that the reverse gradient originated and evolved over a period of about 25 years. Here, we report that this reverse gradient did not exist in the U.S. in 1990 but has increased markedly since then.

Specifically, across the U.S. states by 2015 there were highly significant correlations between $\ln(\text{income})$ and state-level rates of obesity ($r = -0.697$, $p < 0.00001$) and diabetes ($r = -0.706$, $p < 0.00001$), whereas in 1990 neither correlation was yet evident. By 2013, the age-adjusted prevalence of obesity in the U.S. was 35% among men and 40% among women (Flegal et al., 2016)—across all age adult groups, obesity rates among U.S. women have been 4% to 5% higher than among men (Arroyo-Johnson and Mincey, 2016). Since 1990, this change was continual, such that we determine equations for the linear development of these reverse gradients since 1990.

In the U.S., an inverse correlation between obesity and median household income (logarithm) developed from nonexistent in 1990 into a steep inverse correlation, known as the “reverse gradient”, 25 years later. The reverse gradient involving diabetes prevalence also developed in the U.S. over the same period; this lagged the reverse gradient with obesity in 1990 but had fully caught up with it after 2010 (Fig. 3). Both facets of the reverse

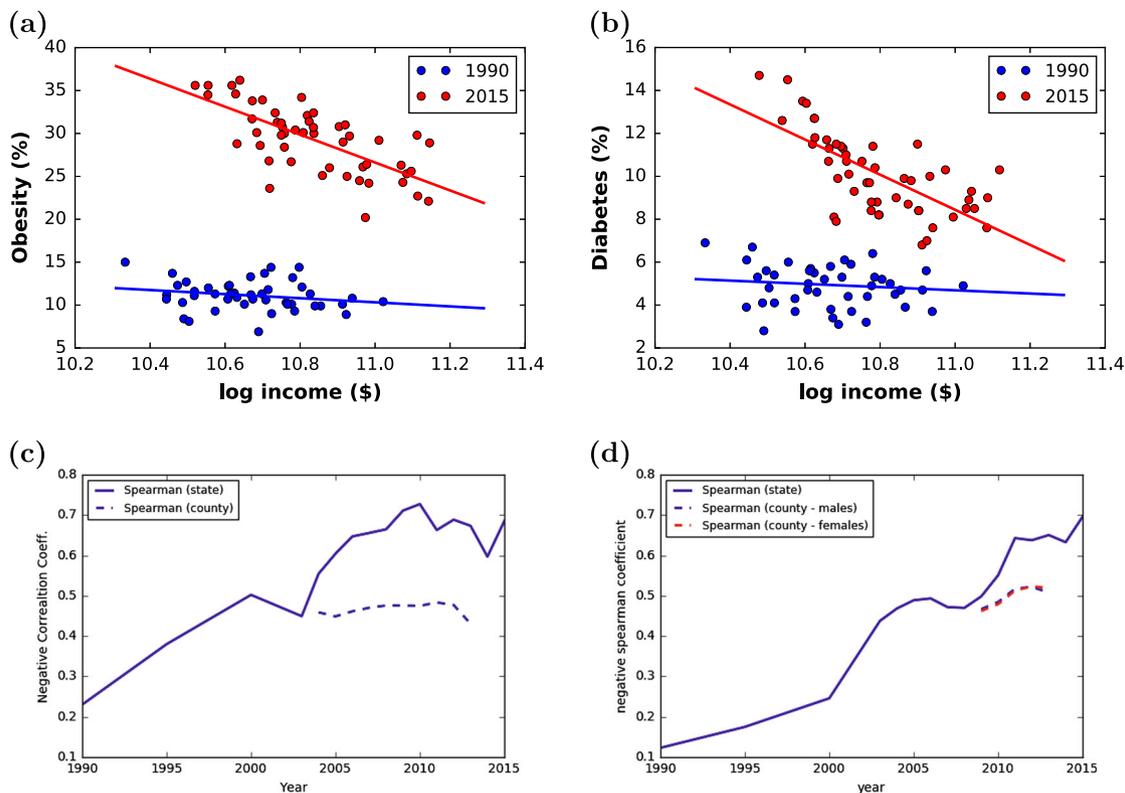


Fig. 3 Negative gradient between household income and obesity and diabetes rates. Scatterplots showing **a** Obesity vs. \ln (income) by state, 1990 and 2015; **b** Diabetes vs. \ln (income) by state, 1990 and 2015. For 1990 (blue), the slopes are -2.39 for obesity and -0.75 for diabetes; for 2015, the slopes -16.26 for obesity and -8.18 for diabetes. Panels **c**, **d** show the change in these correlations over time, between \ln (income) and **c** Obesity and **d** Diabetes, at both state (solid) and county (dashed) levels

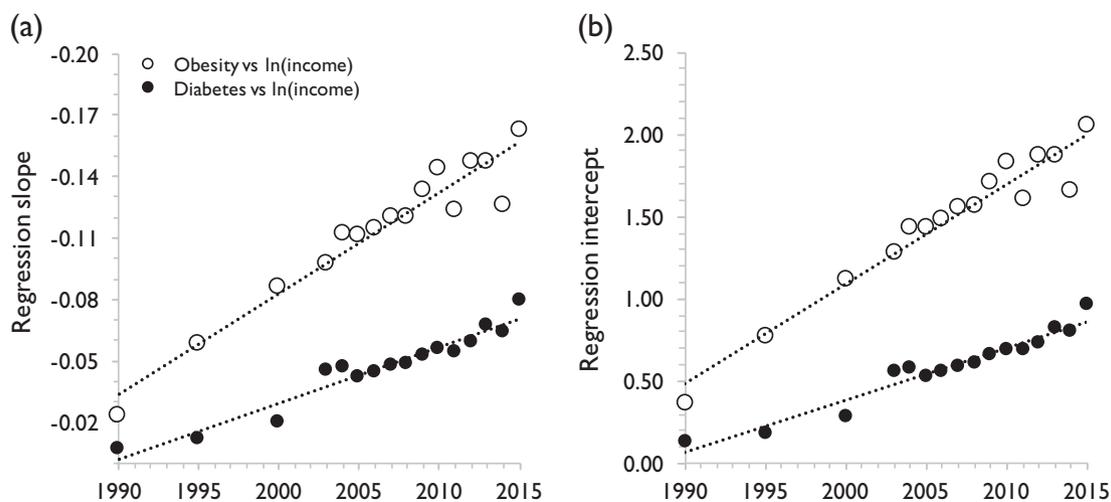


Fig. 4 **a** Slopes and **b** intercepts of the reverse gradients between household income and obesity and diabetes rates, 1990-2015

gradient developed remarkably quickly in the U.S., in about one generation.

Any hypotheses for the recent rise in obesity must account for the obesity epidemic having emerged only in the last few decades in the U.S. We observed that over ten years, the regression coefficient on leisure in predicting obesity increased from 0.40 to 0.63. We interpret this to be a social multiplier effect, as a physiological effect ought to have had that same coefficient through time. Our social multiplier estimate of 1.56 is close to that of crime and U.S. wages, both about 1.7,

estimated by Glaeser et al., (2003) at similar scales of aggregation. While this is some evidence for social influence, these estimates might overstate the true social multiplier, due to correlation between demographics and unobservable elements (Glaeser et al., 2003).

Our simple S-curve approach was more illuminating than we had expected (see Kandler and Powell, 2018), because they were unexpectedly linear or perhaps slightly r-shaped (sensu Henrich 2001), i.e., the adoption patterns do not show much evidence for social (S-shaped) diffusion. A plausible explanation for the steady,

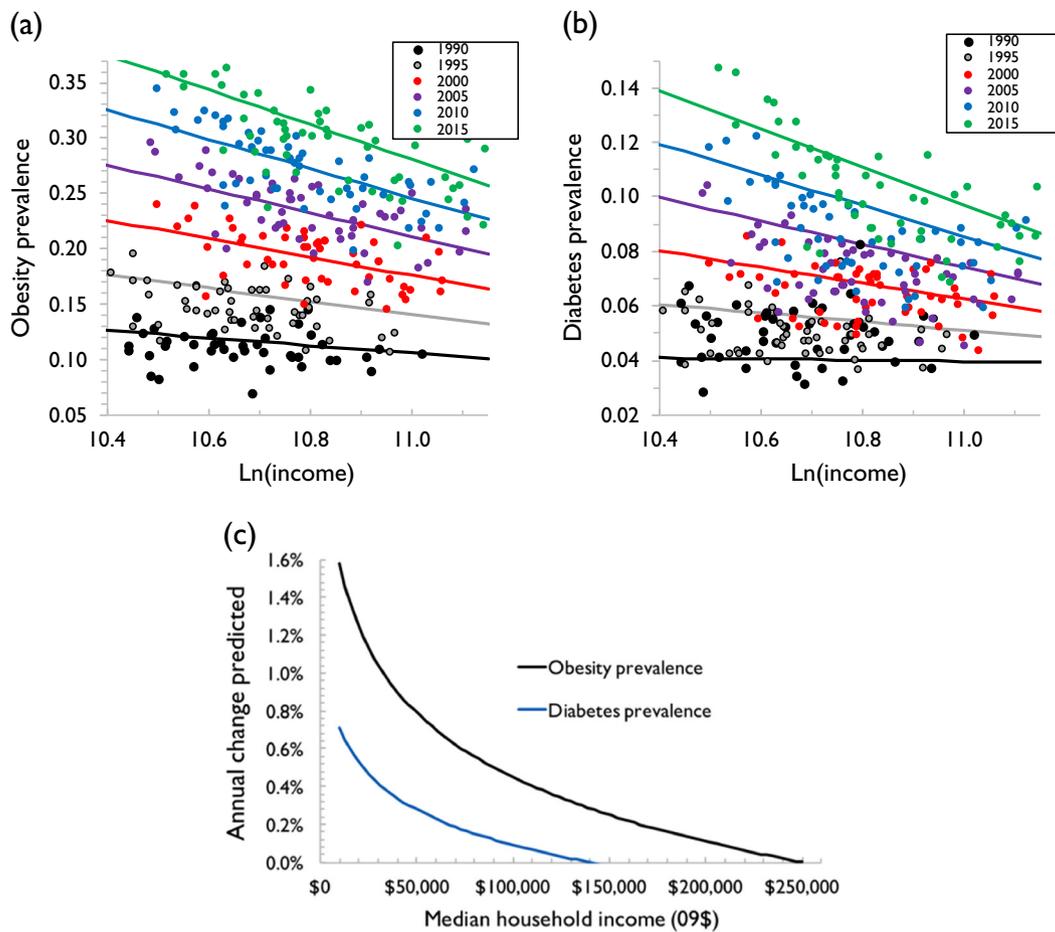


Fig. 5 Evolution of the reverse gradients for **a** obesity and **b** diabetes at 5-year intervals from 1990 to 2015. Colored lines show how the time-evolution of these gradients can be described by the equations in Eqs 6a and 6b, which yields **c** an approximated annual change as a function of household income

Table 2 Multiple regressions predicting obesity rates, U.S. counties, 2004–2013

Year	Coefficient, leisure	s.e	Coefficient, income	s.e	R ² (adjusted)
2004	0.397	0.009	-2.16	0.21	0.507
2005	0.430	0.010	-1.97	0.21	0.520
2006	0.424	0.010	-2.48	0.23	0.518
2007	0.475	0.011	-2.20	0.23	0.531
2008	0.521	0.011	-1.85	0.23	0.567
2009	0.552	0.013	-2.58	0.26	0.545
2010	0.549	0.012	-3.14	0.27	0.551
2011	0.563	0.013	-3.12	0.27	0.557
2012	0.568	0.013	-3.34	0.28	0.556
2013	0.627	0.014	-2.25	0.29	0.543

All regressions significant with $p < 10^{-6}$ or better

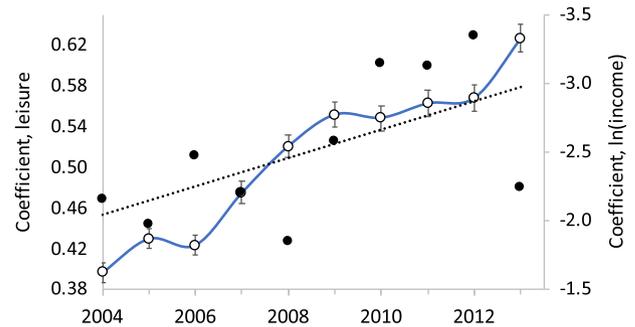


Fig. 6 Change in multiple regressions, U.S. counties, 2004–2013. For regressions predicting obesity rates, the open circles (with blue curve) show the coefficient on the leisure statistic; black filled circles show coefficient on $\ln(\text{income})$. The dashed line shows the trend through the black filled circles ($R^2 = 0.36$). Data are shown in Table 2

25-year increase in state-level obesity rates is a higher-obesity younger generation progressively entering the adult cohort.

The recent origin of the reverse gradient appears to favor the Insurance Hypothesis of HBE. The time series reveal only weak evidence of social learning in most states, insufficient to falsify the HBE hypothesis that obesity increased due to individual responses to a changing nutritional/economic environment. Additionally, there is a lack of evidence for a deep cultural history to obesity. While economic development may be a prerequisite for the reverse gradient (Tafreschi, 2015), the U.S. and Europe possessed developed economies for a century before the reverse

gradient materialized. In Western Europe, there was still no reverse gradient as of 2008 (García Villar and Quintana-Domeque, 2009). The second is the fact that the reverse gradient developed smoothly over time, as described by Eq 6, which indicates the close relationship between income levels and propensity toward obesity. These observations are consistent with the Insurance Hypothesis, which is predicated on an evolved tendency for lower-income people to perceive risks in their local environment and over-compensate through excessive calorie intake.

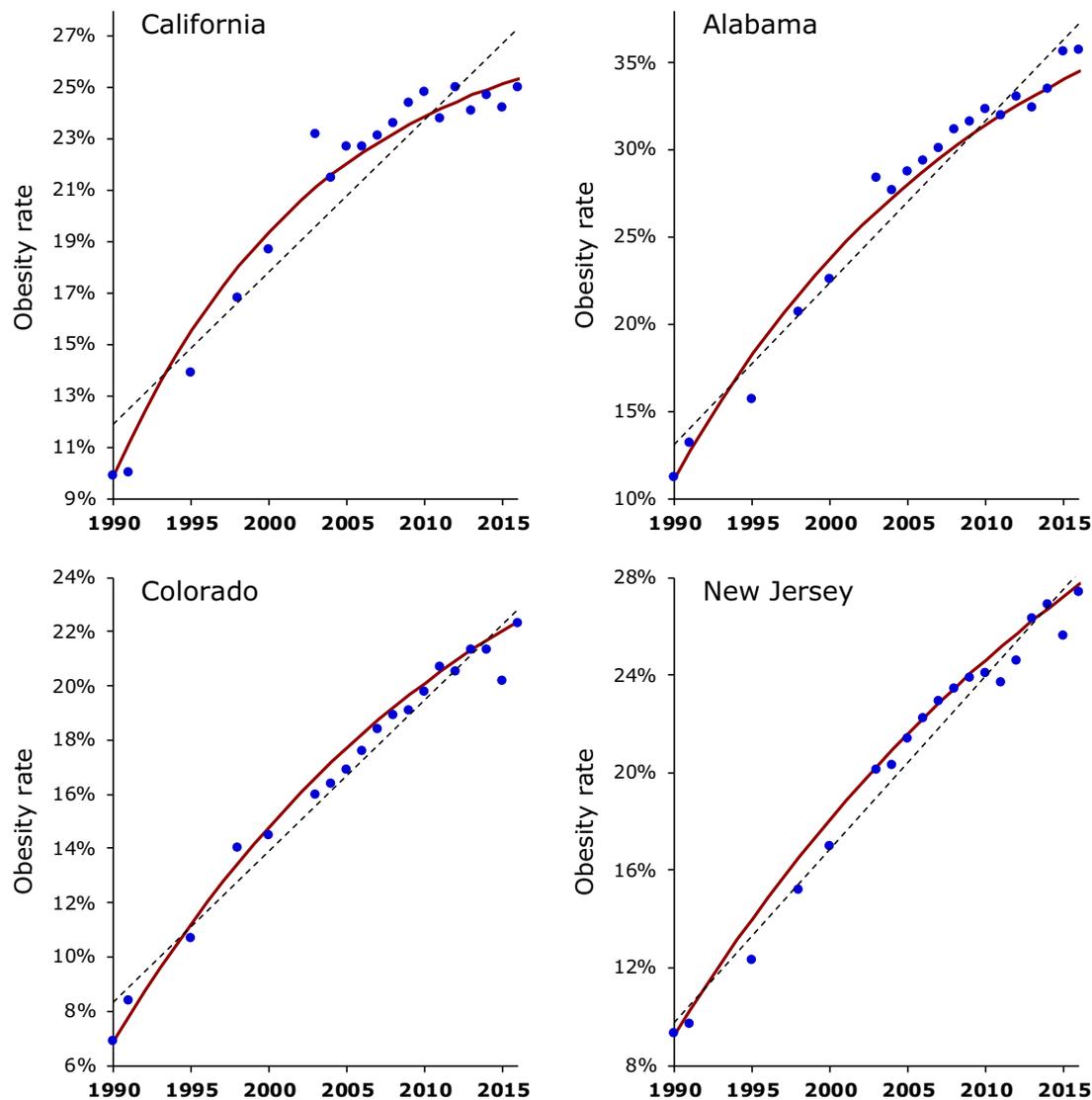


Fig. 7 Rise in state-level obesity rates in four different states, showing the fit of the adoption curve (Eq. 4) with the social parameter, q , set to zero (solid red), as well as a linear fit (dashed black). See Table 3

There are alternatives to the Insurance Hypothesis, as an explanation based upon the behavioral responses of individuals. At the scale of economic geography, a significant factor is food deserts, where “easy geographic access to fast-food outlets and convenience stores encourages individuals to consume foods that are high in energy and saturated fats” (Mullan et al., 2017). Over 50 million people, almost 18% of the U.S. population, live in low-income areas without convenient access to a supermarket (Rhone et al., 2017). In high-income, highly urbanized countries, diabetes correlates positively with the percentage living in urban areas (Goryakin et al., 2017).

Some research has focused on the effect of highly processed foods, which typically contain much more added sugar than unprocessed foods (Lhila, 2011; Bocarsly et al., 2010; Jürgens et al., 2005; Martínez Steele et al., 2016; Stanhope et al., 2009). Excessive sugar intake, which may be addictive (Avena et al., 2008) is a causal factor in diabetes (Hu and Malik, 2010; Shang et al., 2012; Cornelsen et al., 2016) and may also be a causal factor in high obesity rates (Basu et al., 2013; Hu and Malik, 2010; Shang et al., 2012).

Americans have consumed refined sugar since the nineteenth century, however, so the question remains why the obesity

increase, and the reverse gradient happened only in the past three decades. One possible explanation is the recent introduction of high fructose corn syrup (HFCS) into the food economy. Fructose, which decreases insulin sensitivity in obese people (Stanhope et al., 2009), has been used in commercial sugar-sweetened beverages since about 1970. That said, the trend might be due to unobserved individual-level effects, such as the more leisure, the more HFCS drinks consumed. The timing is suggestive; Fig. 9 shows a timeline of the increase in contribution of refined sugar and HFCS to U.S. diet, together with the increase in U.S. obesity rate. While overall sugar consumption rose gradually in the 20th century, from 12% of U.S. food energy in 1909 to 19% by the year 2000, the use of high fructose corn syrup in the U.S. increased from virtually zero per capita in 1970 to over 60 pounds per capita annually in the U.S. in 2000 (Gerrior et al., 2004), about half of total sugar consumption. HFCS became the main sweetener in soft drinks. By 2016 in the U.S., sweetened beverages constituted over 7% of household food expenditures and over 9% of expenditures for low-income households in the SNAP program (Garasky et al., 2016).

The metabolic effects of HFCS include complications of glucose metabolism, lipid profile and insulin resistance (Pereira et al.,

Table 3 Regression fits to the rise in obesity in U.S. states, 1990-2016, including a linear fit, the fit of the adoption curve (Eq. 4), and the adoption curve with the social parameter, q set to zero

State	R^2 (linear)	R^2 (Eq. 4)	p (Eq. 4)	q (Eq. 4)	R^2 (Eq. 4, $q = 0$)	q (Eq. 4, $q = 0$)
Alabama	0.955	0.983	0.050	0.050	0.979	0.050
Arizona	0.963	0.961	0.015	0.050	0.949	0.010
California	0.872	0.970	0.070	0.040	0.965	0.075
Colorado	0.967	0.985	0.040	0.040	0.985	0.040
Connecticut	0.978	0.976	0.018	0.040	0.970	0.010
Delaware	0.934	0.929	0.012	0.040	0.917	0.007
Florida	0.965	0.979	0.040	0.040	0.977	0.030
Georgia	0.945	0.981	0.040	0.040	0.982	0.045
Hawaii	0.929	0.943	0.030	0.090	0.932	0.030
Idaho	0.969	0.986	0.025	0.040	0.985	0.020
Illinois	0.991	0.992	0.030	0.030	0.989	0.030
Indiana	0.976	0.979	0.025	0.020	0.978	0.020
Iowa	0.992	0.992	0.020	0.030	0.990	0.015
Kentucky	0.980	0.987	0.030	0.020	0.986	0.020
Louisiana	0.985	0.985	0.015	0.015	0.984	0.013
Maine	0.991	0.992	0.015	0.025	0.988	0.010
Maryland	0.973	0.986	0.030	0.010	0.987	0.030
Massachusetts	0.974	0.981	0.015	0.025	0.981	0.012
Michigan	0.971	0.984	0.034	0.020	0.983	0.030
Minnesota	0.924	0.960	0.050	0.080	0.950	0.050
Mississippi	0.977	0.988	0.032	0.040	0.984	0.030
Missouri	0.969	0.988	0.040	0.020	0.988	0.035
Montana	0.963	0.977	0.033	0.030	0.975	0.030
Nebraska	0.986	0.991	0.022	0.010	0.991	0.020
New Hampshire	0.961	0.977	0.026	0.040	0.972	0.020
New Jersey	0.966	0.990	0.036	0.095	0.984	0.030
New Mexico	0.982	0.992	0.029	0.022	0.991	0.020
New York	0.926	0.958	0.047	0.060	0.953	0.040
North Carolina	0.966	0.989	0.022	0.180	0.980	0.030
North Dakota	0.987	0.990	0.026	0.030	0.989	0.020
Ohio	0.960	0.990	0.027	0.035	0.989	0.02
Oklahoma	0.972	0.987	0.032	0.07	0.982	0.02
Oregon	0.974	0.979	0.028	0.02	0.979	0.02
Pennsylvania	0.982	0.987	0.017	0.02	0.987	0.015
South Carolina	0.964	0.988	0.032	0.07	0.984	0.03
South Dakota	0.960	0.979	0.03	0.095	0.968	0.015
Tennessee	0.961	0.977	0.036	0.09	0.97	0.036
Texas	0.978	0.991	0.03	0.02	0.993	0.03
Utah	0.956	0.988	0.04	0.09	0.982	0.04
Vermont	0.978	0.980	0.025	0.10	0.975	0.015
Virginia	0.963	0.980	0.04	0.06	0.977	0.03
Washington	0.956	0.984	0.05	0.03	0.982	0.04
West Virginia	0.985	0.992	0.035	0.02	0.991	0.03
Wisconsin	0.989	0.990	0.02	0.09	0.987	0.01

All R^2 values are adjusted R^2 . See Fig. 7 for examples. Each state listed has $N = 19$ years of data; five states (AR, KS, NV, RI, WY) not included due to lack of data points in the 1990s

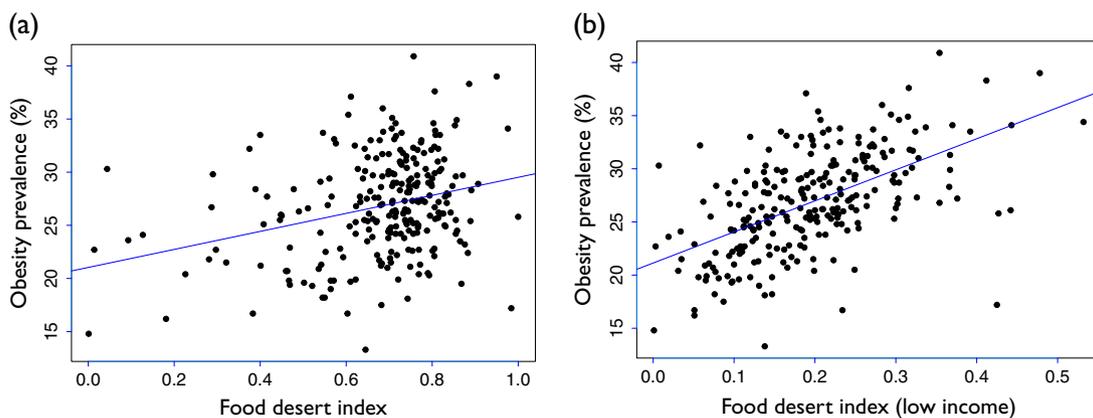


Fig. 8 Food desert index versus obesity rate for 250 urban counties in the U.S. in 2013. In **a** the food desert index is the share of the urban population living a half mile or more from a supermarket. The blue line shows the regression (Pearson's $r = 0.292 \pm 0.11$). In **b** the food desert index is the share of low-income population living a half mile from supermarket. Blue line shows the regression (Pearson's $r = 0.563 \pm 0.08$)

Table 4 Correlations (Pearson’s *r*) between food desert measures (Rhone et al., 2017) and obesity/diabetes rates, across 3108 U.S. counties, 2013

Food desert measure	Correlation with obesity rates	Correlation with diabetes rates
low access within ½ mile	0.470	0.533
low access within 1 mile	0.407	0.430

The food desert measures used are for share of the population with both low income and low access to food within ½ mile and 1 mile

Table 5 Multiple regressions, U.S. counties, 2013, showing outcome variable versus different predictor variables, the estimate of each regression slope and its standard error (s.e.), t-statistic, residual standard error and adjusted R²

Outcome	# counties	Predictor	Estimate	s.e.	t statistic	Res. s.e.	Adj. R ²
Obesity	3109	Intercept	39.23	3.29	11.9	3.14	0.543
		ln (income)	−2.25	0.29	−7.7		
		leisure	0.627	0.014	45.7		
Diabetes	3109	Intercept	23.7	1.64	14.4	1.55	0.498
		ln (income)	−1.91	0.15	−13.2		
		leisure	0.239	0.0065	36.6		
Diabetes	250 (urban)	Intercept	47.5	8.56	5.6	2.60	0.695
		ln (income)	−3.76	0.724	−5.2		
		leisure	0.721	0.049	14.6		
		Food deserts	7.3	1.02	7.2		

ln (income) uses 2009 U.S. dollars. All regressions significant with $p < 10^{-6}$ or better

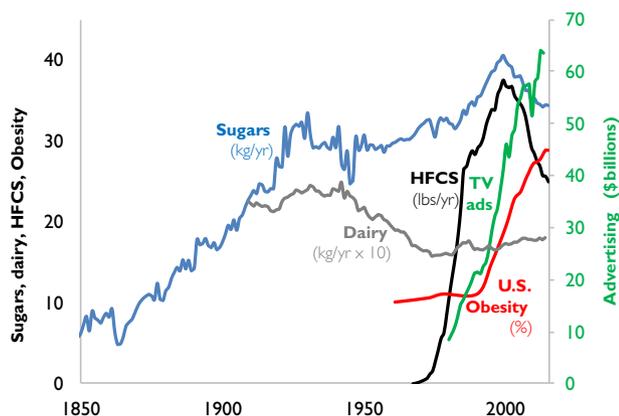


Fig. 9 A timeline of the increase in contribution of refined sugar and high fructose corn syrup (HFCS) to U.S. diet, together with the increase in U.S. obesity rate. The data for sugar, dairy and HFCS consumption per capita are from USDA Economic Research Service (Johnson et al., 2009) except for sugar consumption before 1967, which are historical estimates (Guyenet et al., 2017). Obesity data (% of U.S. adult population) are from the Robert Wood Johnson Foundation’s Trust for America’s Health (stateofobesity.org). Total U.S. television advertising data are from the World Advertising Research Center (www.warc.com). The y-axis on the left covers all data series except advertising expenditures, which uses the y-axis on the right

2017; Johnson et al., 2016; Bocarsly et al., 2010; Bray et al., 2004; Jürgens et al., 2005). HFCS as the driver of obesity and diabetes epidemics would be consistent with HBE in the general sense of human physiology having evolved around a diet containing little sugar and no refined carbohydrates. Hunter-gatherers generally do not exhibit obesity, diabetes, or cardiovascular disease (Kaplan et al., 2017). The HFCS explanation is also consistent with the Insurance Hypothesis, in that poor families are most subject to food scarcity (Hernandez, 2015) and HFCS-sweetened beverages predominate the food economy of poor regions of the U.S.

Conclusions

In conclusion, we find a steady increase, since 1990, in the “reverse gradient” or negative correlation between median household income and both obesity and diabetes rates. In 1990, there was no correlation across the US between either obesity and income or diabetes and income, yet by 2015 strong negative correlations existed across and within U.S. States. We have determined equations for the continual development of these reverse gradients over the past 25 years.

To explain this change, we find evidence in support for both HBE and “social multiplier” effect, a balance similar to empirical studies of other human behavior (Aral et al., 2009). We ascribe more weight to the HBE explanation, in that evolved mechanisms that increase fat storage in response to resource scarcity should promote obesity in high-income countries, where the poor have greater exposure to junk food and other cheap calories including processed sugars (Hill et al., 2017; Wells, 2017).

We speculate that the rapid increase in consumption of high fructose corn syrup (HFCS) may have been a key driver. The obesity and diabetes epidemics could be driven by the commercial oversupply and widespread marketing of inexpensive high-sugar foods, especially HFCS-sweetened beverages (Johnson et al., 2007; Song et al., 2012; Basu et al., 2013).

A fuller explanation of the timing and geography of the obesity epidemic will require the specific history of societal-level factors. Besides the suggestive temporal concurrence between obesity, food deserts and HFCS-sweetened beverages, additional clues lie in the considerable variation in the strength and evolution of the reverse gradient within different states of the U.S. This marked geographic variation in the slope of the reverse gradient indicates that government health policies can mitigate the effect of socio-economic disparities. To explore the scale of these drivers, future work would review and compare state level health policies versus how the negative gradient evolved in those states.

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Data availability

All data we used for this study are publicly-accessible, aggregated data. The datasets analyzed during the current study are available in the Dataverse repository: <https://doi.org/10.7910/DVN/SMTX3X>. These datasets were derived from the following public domain resources: Age-adjusted data on obesity rates at the county and state level for years 2004 to 2015 (Centers for Disease Control and Prevention, 2017d), as well as diabetes rates for 2009–2013 (Centers for Disease Control and Prevention, 2017d), are available from CDC. Available at: www.cdc.gov/diabetes/data/countydata/countydataindic State-level obesity rates since 1990 were obtained from the annual reports of the Trust for America's Health (Robert Wood Johnson Foundation). Available at: stateofobesity.org/adult-obesity. Estimates of "leisure" (physical inactivity) are available from the CDC Behavioral Risk Factor Surveillance System (Centers for Disease Control and Prevention, 2017a). These CDC data come from health-related telephone surveys, which began in 1984 with 15 U.S. states, and now collects data in all 50 states through over 400,000 adult interviews each year. Available at: www.cdc.gov/diabetes/data/countydata/countydataindic. Food desert designations for the U.S. were recently made available through the Food Access Research Atlas (FARA) project (Rhone et al., 2017). The estimates are derived from the 2010 US Census and the 2010–2014 American Community Survey, in which census tracts are categorized by median income, vehicle availability, and SNAP participation. Available at: www.ers.usda.gov/webdocs/publications/82101/eib-165.pdf?v=42752.

Additional information

Competing interests: The authors declare no competing interests.

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