

Volland, Benjamin

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**The effects of income inequality on BMI and obesity:
Evidence from the BRFS**

by

Benjamin Volland

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Max Planck Institute of Economics
Evolutionary Economics Group
Kahlaische Str. 10
07745 Jena, Germany
Fax: ++49-3641-686868

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The effects of income inequality on BMI and obesity: Evidence from the BRFSSBenjamin Volland³Max Planck Institute of Economics⁴This version 21st May 2012**Abstract**

Despite increasing knowledge on its adverse consequences, obesity prevalence across the U.S. has been rising markedly over the past three decades. The private and economic costs of this development are substantial, and it has been estimated that its direct and indirect costs now sum to over 1% of annual GDP. While much progress has been achieved in recent years in understanding the economic changes that contribute to this development, a little researched factor that has also been argued to exacerbate the prevalence of obesity is the distribution of income. Augmenting data from 12 consecutive waves of the Behavioral Risk Factor Surveillance System (BRFSS), with a recently published data set on state-level income inequality based on tax payments, the present paper analyzes whether changes in income inequality can be considered a determinant of variations in body mass and obesity across the U.S. It finds that they have a significant positive effect on BMI and obesity. While the effect is small, it is in the range of other state-level determinants, suggesting that some form of redistributive policy may help containing the spread of unfavorable weight outcomes.

Keywords: BMI; obesity; income inequality, BRFSS

JEL classification: I14, I18

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⁴ Corresponding author: Benjamin Volland, Max Planck Institute of Economics, Evolutionary Economics Group, Kahlaische Strasse 10, 07745 Jena, volland@econ.mpg.de

1. INTRODUCTION

Obesity prevalence worldwide, but particularly in the U.S. has been rising markedly over the past three decades. In fact “obesity [...] is now so common that it is replacing the more traditional public health concerns, including undernutrition and infectious disease, as one of the most significant contributors to ill health.” (WHO, 2000: 1). This development poses a puzzle to medical sciences and economics, as obesity rates have been increasing despite ongoing public health efforts to disseminate information on its adverse consequences, and although a growing number of Americans are dieting and exercising (Philipson and Posner, 1999). Due to its association with various medical complications, like type-2 diabetes or hypertension, a growing proportion of expenditure on health care can be attributed to thwarting the effects of obesity. A recent survey suggests that an obese person causes roughly 25% higher health care expenditures than a an individual of normal weight (Withrow and Alter, 2010), such that in the U.S. between 5% and 10% of health care expenditure can be credited to obesity-related diseases (Tsai et al., 2010). Adding indirect costs, arising from productivity losses, early retirement, death, or disability, overall costs of obesity in the U.S. have been estimated to amount to just over 1% of annual GDP (cf. Sassi, 2010).

As a result, substantial effort has been devoted to identifying the social and economic changes that have contributed to the ongoing rise in obesity prevalence. While its main proximate cause has been identified as a surge in extra-meal snacking and secondary eating (Cutler et al., 2003; Bertrand and Whitmore Schanzenbach, 2009), ultimately an increasing labor force participation of women (Chou et al., 2002), a decline in physically demanding labor (Philipson and Posner, 1999), changes in food production technologies and prices (Cutler et al., 2003; Chou et al., 2004), and a decrease in smoking prevalence (Chou et al., 2004, Baum, 2009) have all been found to contribute to this development.

Another economic factor whose (potential) contribution to general health and mortality has attracted substantial attention over the past decade, and which has also been argued to exacerbate the prevalence of obesity is the distribution of incomes (Pickett et al., 2005; Wilkinson and Pickett, 2009). Proponents of this link argue that income inequality in a society acts as an ecological stressor impairing the (psychological) well-being of – at least a part of – its members (cf. Evans, 2002, Kawachi and Kennedy, 2002; Frank, 2007; Wilkinson and Pickett, 2009). Since the bio-mechanical pathways by which impaired emotional well-being and psychological distress compromise health are well-established (cf. Schneiderman et al., 2005; Sapolsky, 2007), this literature suggests that income inequality per se is detrimental to individual health. Moreover, as distressed individuals have repeatedly been found to alter their eating habits and behavior in favor of easily digestible, calorie-dense foods (Dallman, 2010; Berridge et al., 2010), rising levels of stress caused by growing income differentials may have

indeed contributed to the rise in obesity prevalence. Certainly income inequality across the U.S. has increased considerably over the past three decades (Frank, 2009), and the onset of this rise in the late 1970s roughly coincides with the starting point of what is now considered the obesity epidemic (cf. Sassi, 2010).

Yet, considerable skepticism has arisen whether the distribution of income can indeed be considered a relevant stressor in the context of health outcomes, particularly as a majority empirical studies relating individual health to the distribution of income yielded mixed or unsupportive results, notably when controlling for diminishing returns to absolute income (cf. Mellor and Milyo, 2002; Deaton and Lubotsky, 2003; Lorgelly and Lindley, 2008; Jen et al., 2009; Gravelle and Sutton, 2009; for a recent review in epidemiology see Zheng, 2012). However, a number of more recent contributions have suggested caution when interpreting these null-findings (Subramanian and Kawachi, 2004; Wilkinson and Pickett, 2006; Lorgelly and Lindley, 2008; Zheng, 2012). For instance, Lorgelly and Lindley (2008: 262) hold that the generally estimated static models relating current income inequality to current health outcomes, are not unlikely to report null-findings if the underlying process is inherently dynamic. In this case static models may identify such effects only partially. Yet, medical research indicates that it may take several years of exposure to risk factors before chronic diseases, like e.g. cardiovascular disorders, fully manifest (cf. Yusuf et al., 2001). Consequently, a substantial number of lags, or some sort of stock variable, would have to be considered in the analysis of the inequality-health/mortality nexus in order to obtain plausible results (cf. Blakely et al., 2000; Subramanian and Kawachi, 2004; Zheng, 2012). Because such data are hardly available, and correlation among the lags would present a considerable challenge for identification, an alternative is to analyze health outcomes, which are more likely to be sensitive to current fluctuations in income inequality (Zheng, 2012).

Interestingly, analyses in other areas of economic research focusing on more volatile outcomes like happiness and life satisfaction have reported that individuals tend to react averse to inequality, in the sense that inequality measures attract significant, negative coefficients (Alesina et al., 2004; Winkelmann and Winkelmann, 2010; for a recent review see Senik, 2005). As a number of controlled studies have provided evidence that eating preferences and behavior in humans are sensitive even to acute emotional and psychosocial distress (cf. Zellner et al. 2006; Rutters et al., 2008; for an extensive review see Dallman, 2010), we suggest that weight outcomes may also react more swiftly to short-term fluctuations in ecological stress patterns. Hence, static models may suffice to identify an effect of income inequality in these cases.¹

¹ Moreover, analyzing the relationship between weight status and income inequality, is also informative for the general relationship between somatic health and income inequality, as overweight and obesity are known to be causally linked to a wide range of chronic diseases (cf. Kopelman, 2007).

In this sense, the present paper aims to add to the ongoing debate on the relationship between income inequality and health, as well as to literature investigating the determinants of overweight and obesity. To do so, we apply a methodology, common in the literature estimating the effects of other macro-level determinants to individual behavior, like excess taxation (Carpenter and Cook, 2007; Fletcher et al., 2010) or mandatory seat belt laws (Carpenter and Stehr, 2008), to micro-level data from the 1994-2005 Behavioral Risk Factor Surveillance System (BRFSS). We augment these repeated cross-sectional data with state-level measures of income inequality from a recently published data set based on tax payments (Frank, 2008; 2009).

In what follows, we first discuss related literature on the relationship between social stress and weight outcomes, as well as prior studies investigating their sensitivity to income inequality. Then we describe data sets and empirical methodology, before presenting the empirical results, and discussing their implications for future research.

2. RELATED STUDIES

A vast number of medical and clinical studies have explored the relationship between individual stress and health (cf. Lovallo, 2005). Within this line of research it has also been demonstrated that a majority of individuals change their eating behaviors and habits as a reaction to chronic and acute distress, with significant increases in calorie consumption occurring in about 40% (Dallman, 2010). Moreover, distress has been shown to be linked to a shift in preferences towards foods high in fat and carbohydrate content, like sweet and salty snack foods (cf. Zellner et al., 2006), whose increased consumption has also been found to be the primary, proximate cause of rises in obesity prevalence (Cutler, et. al, 2003). These stress-induced changes have been demonstrated in laboratory settings in the absence of hunger or a homeostatic need for calories, and against deliberately taken decisions on dietary restrictions (Rutters et al., 2008). As people tend to justify their increased consumption of these foods by hedonic motivations, like wanting to feel better (Zellner et al., 2006), researchers have come to label such food items “comfort foods” (cf. Dallman et al., 2006).

Several psychological and neurobiological mechanisms have been suggested as an explanation to stress-induced overindulgence in food, usually emphasizing the stimulating effect of food consumption on the reward systems in the brain stem (cf. Dallman et al., 2003; 2006; Berridge et al., 2010). Animal studies also suggest that stress hormones directly influence the incentive salience of food cues, and thus contribute to compulsive intake (Peciña et al., 2006). Indeed, there is overwhelming evidence for a strong association between abdominal obesity and

(chronic) stress measured in various ways among human individuals (Björntorp, 2001; Drapeau et al., 2003; Dallman et al., 2004, Kyrou et al., 2007). For instance, high levels of stress in a baseline year significantly increase the odds ($OR = 2.72$) of a more than 10kg weight gain over the following 6 years (Korkeila et al., 1998). Therefore, rising levels of psychological ill-being have been suggested as a cause of the ongoing rise in obesity prevalence within wealthy societies (Dallman et al., 2004).

A number of studies have examined whether income inequality contributes to weight outcomes via this link. Using aggregate information Pickett et al. (2005) and Wilkinson and Pickett (2009) find strong support for an association between income inequality and weight outcomes across 21 developed countries and 50 U.S. states. Similar results have been reported by Diez-Roux et al. (2000) using individual data from the 1990 BRFSS, and by Subramanian et al. (2007) for a sample of Indian women. Surprisingly, Chang and Christakis (2005) using data from the 1996-98 BRFSS find no effects of inequality measured at the level of Metropolitan Statistical Areas. Yet, they measure income inequality using data from the 1990 U.S. Census, such that there is a gap of at least 6 years between their measure of income inequality and their measure of weight outcome.

Our approach differs from these studies in a number of ways. First, as aggregate studies have been strongly criticized on the grounds of non-comparability of data and data sources (cf. Judge, 1995; Beckfield, 2004), as well as for conflating the effects of income inequality with the effects of diminishing returns of absolute income on health (Gravelle, 1998; Deaton, 2003), we follow the example of Diez-Roux et al. (2000), Chang and Christakis (2005), and Subramanian et al. (2007) and apply multilevel models to individual level data. However, unlike these studies we do not rely on self-reported income to estimate inequality indices, as these have been found to lead to substantial underestimation of the extent of income inequality in the U.S. (Akhand and Liu, 2002). Instead we make use of a recently published data set, estimating state-level income inequality from tax data (Frank, 2008; 2009). Finally, we address recently raised empirical concerns on the validity of distributional assumption in multilevel modeling as applied by prior individual level studies. Ebbes et al. (2004) point out that the assumption of independence of explanatory variables and higher-level (random) effects are easily violated (e.g. by measurement errors in the regressors), and show that even modest violations of this assumption yield substantially biased regression estimates. They therefore suggest that an alternative approach using dummy sets to control for unobservable heterogeneity across states may overcome potential problems of this nature. By addressing these concerns we intend to provide a robust analysis of the relationship between income inequality and weight status across the U.S., and to shed some light on the contribution of rising income inequality to the rising prevalence of obesity.

3. DATA

Individual level data

We obtain individual level data from the Behavioral Risk Factor Surveillance System (BRFSS), a repeated cross-sectional survey collected annually by the Centers of Disease Control and Prevention (CDC). The BRFSS is a nationally representative telephone survey collected by stratified random sampling on telephone digits within each state (CDC, 2006). Its main objective is to monitor the prevalence of a wide range of health behaviors and health outcomes among the noninstitutionalized adult population (aged 18 or older) across US states and territories. Aside from a rich set of information on risk factors and health behaviors it contains information on standard socio-demographic characteristics like the respondent's sex, age, or educational attainment. Reliability of BRFSS data is high, with correlations between self-reports and third-party measurements for most socio-economic and health-related information in excess of 0.8 (Nelson et al., 2001, and references therein). Another advantage of the BRFSS data is that it is used to estimate official obesity statistics across the U.S. such that obesity prevalence in our data matches official prevalence perfectly.

We pool data from 1994 to 2005. The time frame is limited by the availability of inequality data (see below), and the fact that 1994 was the first year in which the BRFSS was conducted in all U.S. states (cf. Gruber and Frakes, 2006). In line with previous work (cf. Chang and Christakis, 2005) we exclude from our estimations individuals who are residents of Guam, Puerto Rico, and the Virgin Islands, thus essentially restricting the analysis to the 50 states and the District of Columbia. To obtain plausible results for our weight estimations we furthermore remove all individuals with a BMI less than 13 or higher than 70 as these values imply severe pathological eating disorders or are likely to indicate misreporting (cf. Fletcher et al., 2010).² Next, we exclude all women who identify themselves as pregnant. We also drop individuals aged 70 or older, as a number of more recent publications suggest that among the elderly overweight and light obesity may have a protective effect on health (McAuley et al., 2007; Diehr et al., 2008).³ Finally, we have to take account of the potential endogeneity between obesity and individual

² Note that results are unchanged when including these individuals

³ Although these findings have been criticized on methodological grounds (Strandberg, et al., 2009), the issue is still disputed. It thus seems prudent to exclude an age group for which being overweight may have an opposite effect on health compared to the rest of the population. Moreover, the finding that weight and all-cause mortality become dissociated in later life has been substantiated even by studies finding no positive effect of overweight on health (cf. Corrada, et al., 2006). Note that, additional estimations applying different cut-off ages, as well as including the entire sample yielded very similar results. They are available from the author upon request.

income. While income may have an effect on obesity, overweight has also been reported to entail a wage penalty (e.g. Han et al., 2009), although this may be limited to workers with employer-sponsored health insurance (Bhattacharya and Bundorf, 2009). Others have found that overweight individuals suffer from serious discrimination in job application (cf. Rooth, 2009), and are more likely to retire early from the work force (Renna and Thakur, 2010). In the absence of suitable instruments for income in our data, we deal with this problem by adding a control for health insurance coverage, and by excluding all individuals for whom low income may indeed be a result of unfavorable weight outcomes. We therefore drop all individuals who report to be unable to work, are in early retirement, or have been unemployed for more than one year. After these adjustments we are left with full information from 1,450,350 individuals.

The main outcome variable of interest, i.e. the respondent's weight status, is derived from the body mass index (BMI) as supplied in the data set. Following the definition provided by the World Health Organization (WHO) we thereof construct a dichotomous variable measuring obesity ($BMI \geq 30kg/m^2$). Individual-level controls considered in this paper reflect the ones prominently employed in other empirical contributions on the inequality-health nexus (cf. Subramanian and Kawachi, 2004; Lorgelly and Lindley, 2008), in research relating inequality and subjective well-being (cf. Alesina et al., 2004), and on the determinants of the rise in obesity prevalence (cf. Cutler et al., 2003). More precisely they encompass per capita household income (deflated to prices of 1994), as well as its square term in order to control for diminishing returns of absolute income on health.⁴ They also contain the respondent's age (and its square term), race, marital and family status, employment status, and educational attainment. Finally, we control for the individual's smoking status as a number of more recent publication have suggested that the rise in average weight status may be linked with decreasing smoking prevalence (Chou et al., 2004; Baum, 2009). Descriptive statistics and coding details are given in Table A1. in the Appendix.

State level data

The main variable of interest at the state-level, i.e. income inequality, is taken from a new data set provided by Mark W. Frank (2008; 2009). It provides estimates of income inequality for all US states in the time between 1994 and 2005.⁵ In this data set inequality measures are estimated from tax data as published by the Internal Revenue Service's (IRS) *Statistics of*

⁴ In the BRFSS annual household income is coded on an ordinal scale ranging from 1 (< \$10,000) to 8 (\geq \$75,000). In order to establish comparability across households and waves, these values are corrected for household composition and national inflation. Results remain unchanged when using uncorrected values of income (available from the author upon request).

⁵ For further details see Frank (2008; 2009).

Income, and thus provide comprehensive information on the distribution of income across states on a year-to-year basis. Another advantage of using tax-based data is that misreporting to the IRS is penalized suggesting that reported measures of income inequality are substantially more reliable than measures based on self-reports (see also Akhand and Liu, 2002; Frank, 2009). Following the literature we chiefly rely on the Gini coefficient as a measure of income inequality. To facilitate interpretation it is recoded to a range between zero and 100, with rising values implying increasing inequality.

Other state level information includes logged GDP per capita measured in constant 2000 US \$, which was obtained from the OECD regional statistics, and state-level unemployment rates provided by the Bureau of Labor Statistics in the Local Area Unemployment Statistics. Both are likely to reflect the current state of the local economy, which has been reported to affect individual well-being and weight status, independent of the distribution of income (Di Tella et al., 2003). Altogether 612 state-year observations are available.

4. EMPIRICAL STRATEGY

To relate income inequality and weight outcome we follow a methodology common in the literature assessing the effects of policy design and (excess) taxation on individual behavior (cf. Chou et al., 2004; Gruber and Frakes, 2006; Carpenter and Cook, 2007; Carpenter and Stehr, 2008; Baum, 2009; Fletcher et al., 2010), but which has also been applied in the inequality-life-satisfaction literature (cf. Alesina et al., 2004), and more recently in the analysis of other population level determinants in health outcomes (cf. Kravdal, 2010). Formally, the model can be depicted by:

$$w_{ist} = \beta_0 + \beta_1' X_{ist} + \beta_2' Z_{st} + \beta_3 \tilde{I}_{st} + \eta_s + \theta_t + \varepsilon_{ist}, \quad i = 1, \dots, N; s = 1, \dots, S; t = 1, \dots, T, \quad (1)$$

where w_{ist} denotes the weight outcome reported by individual i , interviewed in year t , and residing in state s . The coefficient of interest, β_3 , measures the correlation between this variable and the proxy of income inequality in her state of residence, \tilde{I}_s . In X_i , a set of variables is included characterizing individual i , and Z_s contains state-level characteristics other than the distribution of income. Both vary with the specification considered. Finally, η_s and θ_t specify full sets of dummies for the state of residence and the year in which the individual was surveyed, which is why this approach has also been labeled as two-way fixed-effects (cf. Fletcher et al., 2010). ξ_{ist} is the idiosyncratic error. Within this framework the effect of inequality on BMI is

estimated using ordinary least squares (OLS) estimators, while its effect on obesity is determined using logit models.⁶ Data in the BRFSS are weighted in order to account for the probability of selection, non-coverage, and non-response (CDC, 2006). In order to ensure the consistency of our estimates, we employ these sample weights in our estimations, however, adjusting them for the number of waves considered in our data (cf. Chou et al., 2004; Lu and Yang, 2012). Furthermore, we obtain robust Huber/White standard errors that allow for clustering within sample strata and year of observation.⁷

Note that by including η_s and θ_t we explicitly account for the nesting of observations in states and across years. In this sense our model is equivalent to other hierarchical models more widespread in epidemiological research (Gelman and Hill, 2007: Chap. 1), however, without sharing their strong distributional assumptions on the state-specific effects (i.e. $COV(\eta_s, \mathbf{Z}_{st}) = 0$). In this model within state changes in inequality relative to other states identify the effect of income inequality on weight outcomes (cf. Gruber and Frakes, 2006).

A drawback of the two-way fixed-effects approach is that it requires sufficient variation in state-level income inequality as otherwise the inequality variable will be highly collinear with the dummies. To check this requirement we bootstrap R-squared values from a number of regressions of inequality measures on state and year dummies, and test whether the explained variation in these estimations is significantly smaller than 90%, which is considered the critical cut-off value (cf. Carpenter and Cook, 2007, Fletcher et al., 2010). Results from these exercises using 1,000 bootstrap draws suggested sufficient variation in the Gini coefficient.

5. RESULTS

We start our analysis with a simple graphical illustration. As with the exception of Alaska obesity prevalence and inequality rates grew in all states in the time between 1994 and 2005, Figure 1. plots the deviations from average change in obesity prevalence and Gini coefficient for the 50 states and the District of Columbia over that time. Most states (65%) fall either into the lower left or into the upper right quadrant, suggesting that where income inequality grew disproportionately, so did obesity prevalence. The robustness of this first visual impression to econometric scrutiny is assessed in the following paragraphs.

Please insert Figure 1. about here

⁶ Other estimators assuming normal and complementary log-log distributions for the errors, instead of the logistic were additionally applied yielding similar results. So did linear probability models.

⁷ All estimations are performed applying the svy command using Stata Release 12 (StataCorp, 2011).

Table I. contains the results from a number of models for BMI and obesity, respectively. Coefficients from logit models are transformed into mean marginal effects in order to facilitate interpretation. We start our empirical analysis with a simple model containing no other controls but the Gini coefficient (Columns (1) and (4), respectively), and then gradually add more controls. Goodness-of-fit statistics (log-likelihood and the Akaike Information Criterion (AIC)) indicate that these add-ons improve model fit. However, their absolute size also indicates that this goodness-of-fit is limited. This is also reflected in the low explanatory power of our models, with R^2 values below 9%.⁸ These values are very similar to the ones from prior estimations (e.g. Chou et al., 2004; Baum, 2009), and are likely to be the effect of strong genetic influences on the expression of body mass. Moreover, recent research on populations of scheme-fed laboratory animals also revealed significant rises in body mass index, suggesting that other so far poorly understood factors, like pathogens or epigenetics may also play a significant role in determining body mass outcomes (Klimentidis, et al., 2010).

Please insert Table I. about here

With respect to individual characteristics, our results reproduce a number of well-known phenomena (cf. Chou et al., 2004; Chang and Christakis, 2005). They suggest that males have on average a higher BMI, and an increased probability of being obese. The same holds for respondents who declare themselves to be either black or of hispanic origin.⁹ We also reproduce the finding that higher levels of education are consistently associated with lower BMI, and decreased probability of being obese, and also find little indication of diminishing returns to schooling. In fact, the protective effect of a college degree compared to finishing high-school is roughly 3.5 times larger than the effect of a high-school degree compared to not having completed secondary education. Similarly, we find an inversely U-shaped relationship between age and weight outcomes, with BMI peaking at the age of 54, and the probability of being obese at the age of 41. Furthermore, our results are indicative of the link between smoking behavior and unfavorable weight outcomes. We find that having never smoked or being a former smoker translates into significantly higher BMI values and also increases the chances of reporting an unfavorable weight status compared to a current smoker. These results suggest that increases in average weight status across the U.S. may indeed be coupled with declining smoking prevalence (see also Baum, 2009).

⁸ Comparable R^2 -values from linear probability models on obesity (not reported) do not exceed 4%.

⁹ Interestingly, individuals of Asian-pacific or American Indian descent tend to have lower BMI and lower chances of being obese (see also Chou, et al., 2004: 578). The reason is that particularly among Asian populations mean and median BMI have been found to be lower than in comparable non-Asian populations, such that even population-specific cut-off points for overweight and obesity have been discussed (Barba, et al., 2004). As those population groups are of limited size we combine them in order to improve conversion of the estimations.

Finally, we find strong evidence for the hypothesis that there are diminishing returns of income on favorable weight status, as has been suggested most prominently by Gravelle (1998) and Deaton (2003) for the more general relationship between individual income, health and mortality (see also Kawachi and Kennedy, 2002: 105). Table II. shows the income elasticities of weight status at increasing income deciles. They indicate that a one percent increase in income for the individuals at the 10% income decile decreases the likelihood to report a BMI in excess of 30 by roughly 3.8%, whereas an increase of similar magnitude decreases the probability of being obese only by 1.1% for an individual at the 90% income decile. In this sense, our results are indicative that a redistribution of income from rich to poor could improve overall weight status in the population.¹⁰

Please insert Table II. about here

More importantly, we find consistent support for an independent effect of the state-level Gini coefficient on being obese as well as on BMI, even when controlling for the nonlinear relationship between individual income and weight. Significance levels for BMI are higher than for obesity. This is little surprising considering that BMI as such is certainly more volatile than focusing on a single threshold. In fact, as becoming obese requires some time of severe overindulgence in food, our assumption on sufficient fluctuations in obesity may have been overly optimistic. Results suggest that a 1 percentage point increase in the state's Gini coefficient increases average BMI by roughly $.014 \text{ kg/m}^2$, and the probability of being obese by .09%. These results indicate that the overall contribution of income inequality in explaining the growth in obesity prevalence between 1994 and 2005 is limited. According to our data, the average Gini coefficient across the U.S. grew by roughly 4.1 percentage points over that time, suggesting that income inequality contributed 0.4 percentage points to the overall rise in obesity (9.9 percentage points).¹¹ While these effects are arguably small, they are comparable in size and significance to other state-level factors reported in the literature, particularly soft drink and tobacco taxes, that have been estimated using similar identification strategies (cf. Chou et al., 2004; Gruber and Frakes, 2006; Fletcher et al., 2010). Moreover, they are also comparable in size to the coefficients of similarly coded state-level variables like the unemployment rate. Unlike these variables, however, Gini coefficients are significant in almost

¹⁰ However, recall that the measure of income is based on the eight income groups available in the BRFSS, such that caution is advised when interpreting the size of the different coefficients. Moreover, we cannot control for potential confounding factors like working hours or work stress that are likely to be positively correlated with income, but negatively with weight status, (e.g. by limiting the time available for exercise), again suggesting caution concerning the absolute size of the coefficients.

¹¹ The increase in average BMI incurred from a 1% increase in the Gini coefficient is about .005% of the sample mean, while the increase in the probability of being obese corresponds to roughly .001% of the sample mean. Notably, coefficient size is also independent of the model, as estimations regressing weight outcome on the Gini coefficient alone yield similar coefficients.

all models. We therefore suggest that the ongoing rise in obesity prevalence is likely to be caused by a multitude of factors, each with limited impact on the development. However, we consider our results as strongly suggestive that income inequality is one of them.

Table III. presents the coefficients for the Gini measure from a number of exercises assessing the robustness of our results to potential confounders, subsamples, and changes in specification. We find that our results are robust to the inclusion of state-level racial composition (cf. Deaton and Lubotsky, 2003),¹² and the prices of food, well as the relative price of comfort foods (e.g. Finkelstein et al., 2005).¹³ Moreover, results from Table I. suggested that unemployed and retired had substantially higher BMI, and increased probabilities for being obese, questioning the validity of our results with respect to the endogeneity between weight and income. We therefore re-run estimations on sub-samples additionally excluding all retired and all unemployed (Column (3), Table III.), and focusing on individuals from households with a minimum annual income \$35,000, i.e. the top 75% of the income distribution (Column (4), Table III.). We also exclude individuals from Alaska, as Figure 1. suggested this state may be an outlier (Column (5), Table III.). Results, however, remain unchanged in all cases. Next, and we repeat estimations for whites and non-whites separately (Column (6) and (7), Table III.). While coefficients are similar between these sub-groups, only those for whites are significant. As racial distribution is highly concentrated across the U.S. and only few individuals are non-white in a substantial number of states, insufficient variation is likely to be the main cause for these insignificant results. We also substitute the Gini coefficient by the income share of the top 1% taxpayers (Column (8), Table III.). While the coefficient for BMI is robust to this substitution, the coefficient for obesity just fails conventional levels of significance. Note that both measures assess inequality to a different extent. Whereas, the Gini coefficient considers the entire available distribution, the top 1% measure assesses inequality only at the upper tail (cf. Frank, 2009). We therefore suggest that perceived income inequality may be better reflected by the entire distribution.

Please insert Table III. about here

Finally, previous results on the relationship between tobacco taxes and obesity prevalence suggested that the sign of macro-variable coefficients depended on the specification of time controls (cf. Gruber and Frakes, 2006; Baum, 2009). We therefore re-evaluated our results

¹² Information on state level racial composition was obtained from the US Census Bureau's Intercensal Estimates of the Resident Population of States.

¹³ Information on food prices, as well as the prices of two classical comfort foods, ice cream and potato chips, was taken from the Average Price Data of the Consumer Price Index, provided by the Bureau of Labor Statistics. These data are collected for urban consumers living in four distinct regions across the U.S. (Northeast, Midwest, West, and South). Ice cream and potato chips were chosen as they are the only comfort foods for which information was available for almost the entire time period. Missing values were imputed using linear approximation.

substituting time dummies by a quadratic time trend (Column (9), Table III.). While coefficients diminish slightly in size, no sign reversal can be observed, suggesting that the relationship between income inequality and body mass is not spurious. Note also that goodness-of-fit measures favor the dummy specification, such that we rely on these concerning the significance of the coefficients. Substituting time dummies by state-specific time trends also preserves size and significance of the estimates (Column (10), Table III.).

6. DISCUSSION

In this paper we examined whether the distribution of income can be considered a determinant of variations in body mass and obesity across the U.S., and to which extent changes in income distribution have contributed to the rising obesity prevalence. While in general the relationship between income inequality and health outcomes has been questioned by a recent surge in non-supportive results, others have suggested that ignoring the temporal structure (accumulation and latency effects) of the inequality-health nexus may explain a majority of these null-findings (Lorgelly and Lindley, 2008; Zheng, 2012). We therefore focus on individual weight outcome arguing that weight may be more susceptible to short-term fluctuations in inequality.

Employing a two-way fixed-effects (state and year) approach to 12 consecutive waves from the BRFSS we indeed find an independent effect of income inequality on weight outcomes. While this effect is small, it is comparable in size other state-level determinants like the unemployment rate, and also matches closely to the size of other macro-level determinants like tobacco and soft drink taxes, reported in the literature. In this sense, redistributive policies may help to contain the current obesity epidemic, particularly, as its effect will also make use of the diminishing returns to individual income, for which we find evidence.

Further analysis revealed that these initial findings are robust to a number of changes in variable and model specification. Moreover, results also remain unchanged when restricting the estimation sample in various ways, indicating that problems of endogeneity between income and weight outcomes are unlikely to drive our results. On the other hand the use of repeated cross-sections naturally limits the scope of our analysis, as it becomes impossible to control for unobserved heterogeneity across individuals. However, as weight and height, constituting the central elements of the BMI, are comparatively straightforward measures inter-individual differences in assessment can be assumed to play a minor role. While such an analysis would be desirable nevertheless, questions of effect latency and accumulation become even more pronounced in this case. We therefore suggest that future research should put a strong emphasis on the dynamics in the inequality-health nexus, particularly considering the potential

accumulative effect of inequality on individual health, whose correct specifications are likely to be crucial in order to obtain plausible results (see also Zheng, 2012).

We would also suggest that further studies explore the mechanisms by which income inequality in a society becomes observable for its members. As “objective” information on income distribution (e.g. the Gini coefficient) is hardly present in every-day life, individuals are likely to make inferences based on other indicators. Among these indicators (visible) consumption can be expected to play an important role (cf. Dressler et al., 1999; Kawachi and Kennedy, 2002; Frank, 2007). Winkelmann and Winkelmann (2010), for instance, find that increased conspicuous consumption (measured by the prevalence of luxury sports cars in a region) adversely affects well-being across Swiss municipalities. A result of this form of lay-measurement could be that the prevalence of consumption races and the social acceptability of wealth displays may contribute to explaining the prevailing differences in cross-national findings. Moreover, this would have considerable consequences for policy implications, as it would suggest that progressive consumption taxes may be preferable to a simple taxation of incomes (cf. Frank, 2007). It may also indicate that certain psychological factors, like a materialistic value orientation, could determine individual differences in the vulnerability to perceived inequality, as they influence the importance attached to the (relative) living standard (cf. Richins and Rudmin, 1994).

To summarize, our results indicate that careful theoretical and empirical considerations are needed when aiming to assess the relationship between income inequality and individual health. This is particularly true, as so far our understanding of its dynamics is very limited, and much more research is needed in this respect. While arguably, our results suggest that the impact of income inequality per se is limited in size, they nevertheless indicate the existence of such a link, independent of individual income. We therefore argue that further research in this domain is warranted.

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TABLES

Table I. Results from reduced form two-way fixed-effects estimations (OLS and logit), regressing weight outcomes (BMI, obesity, and overweight) on income inequality, individual and state-level controls.

Dependent Estimator Column number	BMI				Obesity (BMI≥30)			
	OLS (1)	OLS (2)	OLS (3)	OLS (4)	Logit ^a (5)	Logit ^a (6)	Logit ^a (7)	Logit ^a (8)
	<i>Variable of interest</i>							
State Gini coefficient	.0088*** (.0024)	.0117** (.0053)	.0143*** (.0051)	.0145*** (.0051)	.0003* (.0002)	.0006 (.0004)	.0009** (.0004)	.0009** (.0004)
	<i>Individual level predictors</i>							
Per capita income			-.5778*** (.0218)	-.5780*** (.0218)			-.0385*** (.0017)	-.0385*** (.0017)
Per capita income squared			.0590*** (.0030)	.0590*** (.0030)			.0038*** (.0002)	.0038*** (.0002)
Age			.3024*** (.0044)	.3024*** (.0044)			.0163*** (.0004)	.0163*** (.0004)
Age squared			-.0028*** (.0001)	-.0028*** (.0001)			-.0002*** (.0000)	-.0002*** (.0000)
Male			1.2941*** (.0150)	1.2941*** (.0150)			.0163*** (.0012)	.0163*** (.0012)
Health insurance			.0126 (.0245)	.0132 (.0244)			-.0027 (.0019)	-.0027 (.0019)
High school graduate			-.2144*** (.0329)	-.2147*** (.0329)			-.0159*** (.0023)	-.0159*** (.0023)
College graduate			-.8260*** (.0323)	-.8263*** (.0323)			-.0540*** (.0024)	-.0540*** (.0024)
Black			1.6712*** (.0284)	1.6715*** (.0284)			.0829*** (.0019)	.0829*** (.0019)
Hispanic			.6555*** (.0320)	.6567*** (.0319)			.0226*** (.0025)	.0227*** (.0025)
Asian-Pacific/ American Indian			-.8541*** (.0343)	-.8538*** (.0343)			-.0526*** (.0032)	-.0526*** (.0032)
Working for wages			.4104*** (.0255)	.4104*** (.0255)			.0216*** (.0021)	.0216*** (.0021)
Self-employed			-.0352 (.0324)	-.0350 (.0324)			-.0075*** (.0027)	-.0075*** (.0027)

Unemployed			.5865*** (.0530)	.5860*** (.0530)			.0384*** (.0039)	.0384*** (.0039)
Retired			.3283*** (.0471)	.3282*** (.0471)			.0182*** (.0038)	.0182*** (.0038)
Cohabiting couple			-.0153 (.0248)	-.0154 (.0248)			-.0082*** (.0020)	-.0082*** (.0020)
Widowed/divorced/separated			-.0406 (.0287)	-.0406 (.0287)			-.0077*** (.0022)	-.0077*** (.0022)
Number of children (log)			-.1826*** (.0236)	-.1827*** (.0236)			-.0144*** (.0018)	-.0144*** (.0018)
Single parent			.1192*** (.0290)	.1191*** (.0290)			.0040* (.0021)	.0040* (.0021)
Former smoker			1.0448*** (.0214)	1.0447*** (.0214)			.0633*** (.0017)	.0633*** (.0017)
Never smoked			.8030*** (.0184)	.8030*** (.0184)			.0482*** (.0016)	.0482*** (.0016)
<i>State level predictors</i>								
GDP per capita				-.1845 (.2522)				-.0015 (.0205)
Unemployment rate				.0140 (.0131)				-.0007 (.0011)
Population (in 10 ⁶)				-.0053 (.0167)				-.0006 (.0011)
Time & state dummies	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Observations	1,470,989	1,470,989	1,450,350	1,450,350	1,470,989	1,470,989	1,450,350	1,450,350
F-Statistic	13.22	139.34	588.85	582.14	3.27	94.22	214.09	215.61
Loglikelihood	-4,496,640	-4,487,102	-4,365,977	-4,365,974	-68,123,858	-67,503,743	-64,124,086	-64,123,818
AIC	8,993,285	8,974,331	8,732,123	8732121	136,247,718	135,007,608	128,248,334	128,247,804
R-squared	.0000	.0129	.0898	.0898				

Notes: All equations include a constant. Heteroskedasticity-consistent Huber/White standard errors allowing for clustering at the strata-level and year of observation in parentheses. ^a All coefficients from logit estimations are presented as mean marginal effects.; * significantly different from zero at the 10% level; ** significantly different from zero at the 5% level; *** significantly different from zero at the 1% level

Table II. Income elasticity of body mass index and probability of being obese at different income deciles.

Income decile	BMI	Obesity (BMI≥30)
10%	-.5014*** (.0181)	-.0376*** (.0017)
20%	-.4634*** (.0163)	-.0337*** (.0015)
30%	-.4337*** (.0149)	-.0309*** (.0013)
40%	-.4080*** (.0138)	-.0285*** (.0011)
50%	-.3835*** (.0127)	-.0264*** (.0010)
60%	-.3488*** (.0112)	-.0235*** (.0008)
70%	-.3057*** (.0094)	-.0202*** (.0006)
80%	-.2424*** (.0072)	-.0157*** (.0004)
90%	-.1650*** (.0060)	-.0108*** (.0004)

Notes: estimated from the models presented in column (4), Table I. for BMI, and column (8), Table I. for being obese.

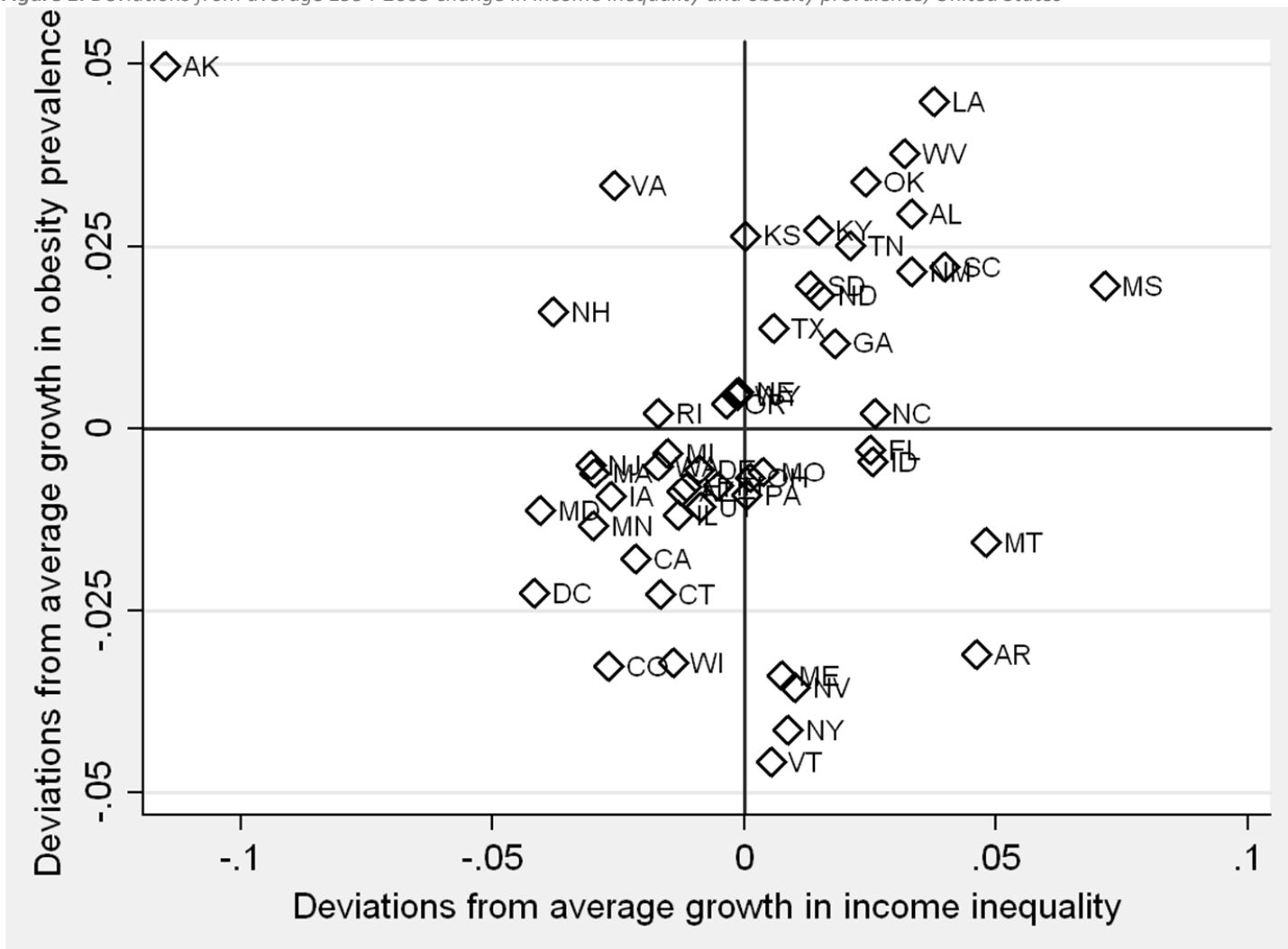
Table III. Robustness checks regressing weight outcomes (BMI, and obesity) on income inequality, individual and state-level controls.

Column	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Change	Including racial composition of state	Including food prices)	Excluding all retired and unemployed	Minimum annual household income \$ 35, 000	Excluding Alaska	Whites only	Non-whites only	Income share of top 1% tax payers instead of Gini	Quadratic time trend instead of time dummies	State-specific time trends instead of time dummies
BMI	.0143*** (.0052)	.0130*** (.0049)	.0154*** (.0053)	.0175*** (.0055)	.0152*** (.0052)	.0161*** (.0052)	.0119 (.0117)	.0145** (.0056)	.0084* (.0046)	.0101* (.0053)
Obesity (BMI≥30)	.0007* (.0004)	.0006* (.0004)	.0009** (.0004)	.0010** (.0004)	.0009** (.0004)	.0007* (.0004)	.0012 (.0009)	.0007 (.0005)	.0004 (.0004)	.0007* (.0004)
Observations	1,450,350	1,450,350	1,335,436	1,086,813	1,431,268	1,170,358	279,992	1,450,350	1,450,350	1,450,350

Notes: Only the coefficients for the Gini measure are reported. All equations correspond to the ones in Columns (4) and (8) of Table I. for BMI and obesity, respectively. They include state dummies, year dummies, and a constant. Further controls: Individual level predictors include per capita household income (corrected for inflation), its square term, the respondent's age, and its square term, and sets dummy sets controlling for race (white/other (base); black; Hispanic; Asian-pacific/American Indian), educational attainment (some high school or less (base); high school graduate; some college or college graduate), and smoking status (current smoker (base); former smoker; never smoked), marital status (never married (base); cohabitating couple; widowed/divorced/separated), and employment status (homemaker/student (base); working for wages; self-employed; unemployed; retired/unable to work);^c State level predictors include GDP per capita in constant 2000 US\$ (log), state level unemployment rate, state population in millions. Heteroskedasticity-consistent Huber/White standard errors allowing for clustering at the strata-level in parentheses. All coefficients for BMI stem from logit estimations and are presented as mean marginal effects. * significantly different from zero at the 10% level; ** significantly different from zero at the 5% level; *** significantly different from zero at the 1% level

FIGURES

Figure 1. Deviations from average 1994-2005 change in income inequality and obesity prevalence, United States



Source: own estimations, based on 1994-2005, BRFSS

APPENDIX

Table A1. Descriptive statistics for individual level data. Source: BRFSS, 1994-2005

Variable	Description	N	Mean	SD	Min	Max
	<i>Dependent variables</i>					
BMI	Body mass index in kg/m ²	1,470,989	26.35	.0075	13.0	69.9
Obesity	1 = BMI ≥ 30 0 = otherwise	1,470,989	.1968	.0006	0	1
	<i>Independent variables</i>					
Per capita income	Household income corrected for inflation and household size	1,467,003	1.9695	.0017	.039	8
Age	Age in years	1,470,989	40.05	.0205	18	69
Male	1 = male 0 = female	1,470,989	.5197	.0007	0	1
Health insurance	1 = has health insurance 0 = otherwise	1,468,829	.8464	.0006	0	1
Cohabiting couple	1 = cohabiting couple 0 = otherwise	1,469,205	.6491	.0007	0	1
Widowed/divorced/separated	1 = widowed, divorced, or separated 0 = otherwise	1,469,205	.1438	.0005	0	1
Working for wages	1 = gainfully employed 0 = otherwise	1,467,427	.6985	.0007	0	1
Self-employed	1 = self-employed 0 = otherwise	1,467,427	.1022	.0004	0	1
Unemployed	1 = unemployed 0 = otherwise	1,467,427	.0332	.0003	0	1
Retired	1 = retired or unable to work 0 = otherwise	1,467,427	.0429	.0003	0	1
High school graduate	1 = high school graduate 0 = otherwise	1,470,271	.3024	.0007	0	1
Some college or college graduate	1 = some college or graduation 0 = otherwise	1,470,271	.6019	.0006	0	1
Black	1 = black 0 = otherwise	1,464,828	.0939	.0004	0	1
Hispanic	1 = Hispanic origin 0 = otherwise	1,464,828	.1166	.0006	0	1
Asian-pacific/American Indian	1 = Asian-pacific or American Indian 0 = otherwise	1,464,828	.0541	.0004	0	1
Former smoker	1 = former smoker 0 = otherwise	1,468,122	.2194	.0006	0	1
Never smoked	1 = never smoked 0 = otherwise	1,468,122	.5385	.0007	0	1
Single parent	1 = single parent 0 = otherwise	1,467,005	.0431	.0002	0	1
Number of children (log)	Number of children in natural logarithm	1,467,005	.2574	.0007	0	3.04

Table A2. Descriptive statistics for state level data. United States, 1994-2005

Variable	Description	Source	State- years	Mean	SD	Min	Max
Gini coefficient	Gini coefficient	Frank (2009)	612	57.71	3.02	52.13	67.47
Income share of top 1%	Income share of top 1% taxpayers	Frank (2009)	612	15.59	3.43	9.57	27.52
GDP per capita	Logged GDP per capita in 2000 US \$	OECD regional data	612	10.39	0.25	9.96	11.75
Unemployment Rate	Percent unemployed	Local Area Unemployment Statistics (BLS)	612	4.88	1.21	2.3	8.7
Population in (10 ⁶)	State population	Intercensal Estimates of the Resident Population of States (U.S. Census Bureau)	612	5.44	6.05	.47	35.83