The Climate Co-benefits of Obesity Reduction

Anthony Underwood*[a] and Sammy Zahran [b]

[a] Department of Economics
Dickinson College
Carlisle, PA 17013-2896
United States
underwoa@dickinson.edu

[b] Department of Economics
Colorado State University
Fort Collins, CO, 80523-1771
United States
szahran@colostate.edu

*corresponding author

Abstract

Rising obesity rates may contribute to greenhouse gas emissions both directly through increased food production and indirectly through higher passenger weights and increasingly sedentary lifestyles. Using panel data for the fifty US states over the period 1997 to 2011 we examine the relationship between the obesity rate and carbon dioxide emissions from energy use. Results indicate a positive and significant relationship, holding population, affluence, sectoral composition, and other factors constant. Specifically, we find that reversion to 1997 obesity rates (from 2013 levels) nationwide could reduce annual carbon dioxide emissions from energy use by 143 million metric tons, or approximately 2.8 percent of annual US emissions. This reduction yields annual climate benefits of between $5.7 and $8.9 billion using EPA estimates of the social cost of carbon. We suggest that policies directed toward obesity reduction have the potential to generate significant climate co-benefits alongside the well-known health benefits.

JEL Classifications: Q54, I12,

Keywords: climate change, obesity, co-benefits, health

1. Introduction

Global obesity prevalence has doubled since 1980. This has serious implications for public health, increasing the risk of cardiovascular disease, Type-2 diabetes, musculoskeletal disorders, and some cancers (WHO, 2015). While these risks are pronounced in OECD nations (OECD, 2010), the percentage of adults that are overweight or obese are on the rise in low- and middle-income nations, particularly in urban areas. In 2014, more than 1.9 billion adults (39%) were overweight worldwide, of which over 600 million (13%) were obese (WHO, 2015).

While obesity prevalence has stabilized or grown only modestly in many developed nations over the past decade the prevalence remains elevated and retrenchment of the epidemic is unlikely (Ogden et al., 2014; OECD, 2014). In the United States, 64% of adults were overweight in 2013, of which 28% were obese (CDC, 2014). This prevalence of overweight and obesity generates significant economic costs: both direct and indirect. Direct medical costs of overweight and obesity combined, including preventative, diagnostic, and treatment services, is an estimated $114 billion annually, comprising approximately 5% to 10% of healthcare spending the United States according to a meta-analysis of 33 U.S. studies (Tsai et al., 2011). Indirect costs associated with morbidity and mortality include the value of income lost from reduced productivity, restricted activity, absenteeism, bed days, and the value of future income lost due to premature death (CDC, 2014). While more difficult to assess, these indirect morbidity and mortality costs account for over 50% of the total economic costs of obesity (Dee et al., 2014).

The primary driver of obesity and overweight is an energy imbalance between calories consumed and calories expended. Globally, an increased intake of energy-dense foods that are high in fat and an increase in physical inactivity due to the increasingly sedentary nature of many forms of work, changing modes of transportation, and increasing urbanization are contributing to the prevalence of overweight and obesity (Ledikwe et al., 2006; Malik et al., 2013). These changes are likely the result of environmental and societal changes associated with development and need to be understood in that context (Wells, 2012). Alongside these changes in food production and consumption patterns, transportation, and urbanization the global atmospheric concentration of carbon dioxide (CO_2), the most prevalent greenhouse gas, has increased from 316 parts per million (ppm) in 1959 to over 400 ppm in 2014 (NOAA, 2015). These atmospheric concentrations are unprecedented in at least the last 800,000 years and anthropogenic emissions are the dominant driver of these changes and lead to global warming and climate change (IPCC, 2014). Since the 1950s, the atmosphere and ocean have warmed, the amounts of snow and ice have diminished, and sea level has risen. Continued greenhouse gas emissions will cause further warming and long-lasting changes in all components of the climate system, increasing the likelihood of severe, pervasive, and irreversible impacts for people and ecosystems (IPCC, 2014)

The implications of anthropogenic greenhouse gas emissions and climate change for global public health have long been recognized and may manifest themselves in varying ways in different parts of the world. In the United States, public health can be affected through disruptions of physical, biological, and ecological systems leading to increased cardiovascular and respiratory disease, injuries and premature deaths related to extreme weather events, changes in the prevalence and geographic distribution of food- and waterborne illnesses, and potential threats to mental health (Luber et al., 2014). An understanding of the immediate threats to human health resulting from ground-level air pollution, particularly ozone and particulate matter, coincidental with anthropogenic greenhouse gas emissions suggests that mitigation policies will lead to immediate improvements in health and generate significant co-benefits dramatically improving their cost-effectiveness (Buonocore, 2014; Shaw et al., 2014). Both the drivers and impacts of climate change play a significant role in population health through a variety of direct and indirect channels (McMichael, 2013). What if the reverse is also true?

It has been suggested that obesity and adiposity are associated with higher emissions and thus climate change (Edwards and Roberts, 2009; Michaelowa and Dransfeld, 2008; Squalli, 2014) and that strategies to reduce obesity may also lead to greenhouse gas mitigation and more sustainable patterns of development (Gryka etal., 2012; Lowe, 2014; Reisch and Gwozdz, 2011; Webb et al., 2014). However, the empirical evidence of this association is suggestive but not convincing for two reasons: 1) questionable assumptions regarding the lifestyle and consumption patterns of obese and/or overweight individuals and 2) methodological decisions preventing effective control of confounding and unobservable factors. In section 2 we discuss these limitations and the existing theoretical explanations for and empirical evidence of this association. In section 3 we discuss our data and methodology. In section 4 we present results for the estimated obesity elasticity of emissions and discuss the potential for climate co-benefits of obesity reduction. Finally, in sections 6 and 7 we conclude with a discussion of the importance and relevance of the results.

2. Theoretical Explanations and Empirical Evidence

The hypothesized channels through which obesity leads to greenhouse gas emissions in excess of an otherwise ‘healthy’ population, as measured by BMI, are generally: 1) increased food production, especially animal-based products, and food waste generation due to higher caloric intake of obese and overweight individuals (Edwards and Roberts, 2009; Michaelowa and Dransfeld, 2008; Walpole et al., 2012); 2) higher fuel use from motorized transport due to increased passenger weight and the *assumption* that heavier individuals may use motorized travel more and choose larger fuel-inefficient vehicles (Edwards and Roberts, 2009; Michaelowa and Dransfeld, 2008; Goodman et al., 2012).

2.1 Increased food production

During the second half of the twentieth century, traditional plant-based diets have been replaced by high-fat energy dense diets consisting of substantial amounts of animal-based foods, such as meat and dairy products (WHO, 2003). Per-capita meat consumption has increased 43% in industrialized nations and 50% worldwide since 1964 and this likely explains some of the increased prevalence of obesity given the association between higher meat consumption and a variety of adiposity measures (Wang and Beydoun, 2009; WHO, 2003). The production of animal-based foods, especially ruminant meat, is associated with much higher greenhouse gas emissions than plant-based foods (Ripple et al., 2014; Scarborough et al., 2014; Stehfest et al., 2009), so if obesity is associated with higher meat consumption which leads to higher greenhouse gas emissions, then this link is quite plausible. Edwards and Roberts (2009) find that an overweight population (with mean BMI of 29 and 40% obese) would require 19% more food energy for its total energy expenditure compared to a 'normal' population (with mean BMI of 24.5 and 3.5% obese). However, the method through which they attribute this to higher GHG emissions is crude, at best. Michaelowa and Dransfeld (2008) also find that emissions from food production have increased, but do not establish a causal pathway to higher prevalence of obesity. Walpole et al. (2012) find that if all countries had the BMI distribution of the United States, it would be equivalent to having an 'extra' 473 million adults living on earth. Therefore, it appears the energy requirements of a heavier population are substantial and require increased food production which already comprises around one-fifth of global greenhouse emissions.

2.2 Higher transportation fuel use

In 2013, the transportation sector accounted for about 27% of total U.S. greenhouse gas emissions and about 15% worldwide (EIA, 2014). Vehicle weight is necessarily associated with the fuel efficiency of miles traveled, thus increased passenger weight can undermine improvements in fuel efficiency. Dannenberg et al. (2004) found that the increase in the average weight of U.S. citizens during the 1990s led to an increase in fuel use of 2.4% and annual emissions from US air traffic by 3.8 million metric tons. Using driving and passenger information in the US and historical anthropometric data, Tom et al. (2014) estimate that since 1970 over 205 billion additional liters of fuel were consumed to support the extra weight of the U.S. population, equivalent to 1.1% of total fuel use for transportation systems, resulting in an extra 502 million metric tons of CO_2 emissions. Edwards and Roberts (2009) also assume that heavier people may use motorized travel more (based on the assumption that walking requires more effort for the obese) and that heavier people may choose larger (more fuel-inefficient) vehicles and as a result find that an overweight population (defined above) would generate 12% more transport CO_2 emissions than a 'normal' population. Goodman et al. (2012) find that overweight and obese individuals generate approximately 14% more

emissions than ‘normal’ weight individuals and that this is partially explained by reduced active travel and larger car size. The bulk of the effect is attributable to increased motorized travel distance. It is plausible that obesity is the *result* of increased commuting requirements (due to family or work) and is not itself a cause of increased travel as Edwards and Roberts (2009) suggest (Goodman et al., 2012). Due to these complexities in the relationship between driving behavior and obesity it is likely that the association between transport CO_2 emissions and obesity is confounded by motorized travel distance making causal pathways difficult to establish (McCormack and Virk, 2014).

To date and to our knowledge, only Squalli (2014) has investigated whether obesity prevalence (as measured by BMI) is associated with higher greenhouse gas emissions at the national or regional level. In other words, there is little empirical evidence to support the hypothesis that policies designed to reduce obesity will generate substantial climate co-benefits in form of reduced greenhouse gas emissions at the national or global level. Sqaulli (2014), using data for the fifty US states in 2010, estimates that a 10% reduction in the obesity rate reduces CO_2 emissions by 0.7% and concludes that reversion to year 2000 obesity rates in the US can reduce greenhouse gas emissions by at least 136 million metric tons. However, given the cross-sectional nature of the sample and the inability to account for likely unobserved heterogeneity and spatial dependence among the 50 states, these estimates are likely biased and inconsistent.

Hersoug et al. (2012) suggest that increases in obesity and type-2 diabetes are due to increased atmospheric CO_2 concentrations. In other words, the causality is reversed. This hypothesis is based on the notion that increased environmental CO_2 reduces blood pH leading to an increased firing rate in the orexin neurons in the hypothalamus, wherein several key processes in the brain could be affected, leading to greater appetite and increased energy storage (Hersoug et al., 2012). Zheutlin et al. (2014) examine this possibility and determine that while the association between obesity and emissions is positive, it becomes insignificant when controlling for ambient particulate matter. This suggests that ground-level air pollution (such as ozone and particulate matter) may lead to higher prevalence of obesity but that the causal pathway suggested by Herzoug et al. (2012) (higher CO_2 concentrations leading to greater prevalence of obesity) is unlikely. Therefore, the goal of this paper is to provide evidence for a possible causal relationship between obesity and emissions from energy use and generate empirical evidence to support the hypothesis that policies designed to reduce obesity may generate substantial climate co-benefits in the form of reduced greenhouse gas emissions.

3. Data and Methods

We construct a longitudinal data set of the fifty US states over the period 1997-2011 using data from the US Energy Information Administration (EIA), the US Census Bureau, the US Bureau of Economic Analysis (BEA), and the US Centers for Disease Control and Prevention (CDC) in order to estimate the relationship between obesity and CO_2emissions from energy use.

3.1 Emissions

Energy-related CO_2 constitutes over 80% of total emissions, thus the state energy-related CO_2 emission levels provide a good indicator of the relative contribution of individual states to total greenhouse gas emissions in the United States (EIA, 2015). The EIA emissions estimates at the state level for energy-related CO_2 are based on data contained in the State Energy Data System (SEDS) on energy consumption for several fuel types: coal (residential/commercial, industrial, and electric power sector), natural gas, and ten petroleum products[1] (EIA, 2015).

3.2 Overweight/Obesity

Obesity and overweight prevalence data by state over the sample period were retrieved from the CDC Behavioral Risk Factor Surveillance System Survey (CDC, 2015).[2] Body mass index (BMI), on which this study and the majority of prior studies (Edwards and Roberts, 2009; Michaelowa and Dransfeld, 2008; Walpole et al., 2012) are based does not directly measure fatness (adiposity). Individuals with BMI ≥ 30 are considered obese, individuals with BMI ≥ 25 are considered overweight, and individuals with BMI < 18.5 are considered underweight. The only information needed to calculate BMI is the individual's height and weight, where $BMI = weight\ (kg)/height(m)^2$, therefore its use is widespread due to the routine collection of this information in medical screenings. While BMI has been shown to be moderately correlated with other measures of adiposity (Wohlfahrt-Veje et al., 2014) it does not adequately capture the heterogeneity of obesity across groups and individuals (Green et al., 2015), especially with respect to body composition and gender, leading to somewhat arbitrary cut-points for classification (Shah et al., 2012). Despite these limitations of BMI as a measure of adiposity, it remains the mostly widely available metric, and to our knowledge, the only statistic with valid state-level estimates over the entire sample period. Using this CDC data on the percent of the state *overweight* (BMI > 30) and *obese* (25 ≤ BMI ≤ 29.9), we generated our variable of interest, $obesity = overweight + obese$, measuring the percent of the state overweight or

[1] These include: asphalt and road oil, aviation gasoline, distillate fuel, jet fuel, kerosene, hydrocarbon gas liquids (HGL), lubricants, motor gasoline, residual fuel, and other petroleum products.

[2] Centers for Disease Control and Prevention (CDC). *Behavioral Risk Factor Surveillance System Survey Data.* Atlanta, Georgia: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention (2015).

obese (BMI ≥ 25). Only Idaho in 2004 did not conduct surveillance leading to missing obesity values, therefore to obtain a balanced panel values for overweight and obese were imputed using a Gaussian normal regression imputation method (Schenker and Taylor, 1996).

3.3 Control Variables

Based on well-established literature regarding the human drivers of greenhouse gas emissions we control for urbanization (as measured by population density), affluence (as measured by real GDP per capita, and sectoral composition (as measured by the share of GDP devoted to the most carbon intensive industries) (Rosa and Dietz, 2012; Jorgenson and Clark, 2012; Jorgenson et al., 2014; Liddle, 2014). Due to a lack of data availability we are unable to control for household size to account for the potential of household scale economies, yet given the relationship between household size, urbanization, and affluence the bias introduced by this omission is likely small (Underwood and Zahran, 2015).

3.4 Model Specification

Despite the inclusion of the control variables mentioned above we expect some time-constant unobserved heterogeneity, such as weather, climate, access to fossil fuels, access to waterways, and other geographic features, to exist among the fifty states that may explain some of the differences in both obesity and emissions. Likewise we expect that states will respond similarly to nationwide factors such as technological change, policy change, and demographic change, so we also account for time-variant unobserved effects. Therefore, we begin generally with a two-way unobserved effects model for the 50 US states over the 15 year period 1997-2011, a fully balanced short-panel with N > T where the dependent variable is CO_2 emissions (millions of metric tons), CO_2 emissions per capita (metric tons of CO_2), or the CO_2 intensity of GDP (metric tons of CO_2 per dollar of real GDP). We include state fixed effects, year fixed effects (as year dummies with 1997 as the reference year), several control variables: population density (measured in millions of people per 100 square kilometers), real GDP per capita (measured in chained 2009 US dollars), several variables denoting the share (as a percent) of state GDP devoted to the most carbon intensive sectors (agriculture, mining, utilities, construction, and manufacturing), and our variable of interest: the obesity rate. The data are summarized in Table 1.

Based on the nature of the data, several estimation strategies are plausible, most of which control out between state variation in favor of estimating within state effects, here we focus on two: 1) a static fixed effects model with clustered standard errors which are robust to serial correlation and heteroskedasticity but assume cross-sectional independence (observations across states are uncorrelated); or 2) a dynamic Prais-Winsten (PW) regression including a lagged dependent variable with panel-corrected standard errors

Table 1. Summary Statistics

VARIABLES	State-Year Observations	Mean	Std. Dev.	Min	Max
total CO_2 emissions	750	114.769	114.584	5.699	724.097
CO_2 emissions per capita	750	25.316	19.868	8.113	132.268
CO_2 intensity of GDP	750	584.197	430.846	132.142	2746.661
population density	750	0.00722	0.00975	0.00004	0.04639
real GDP per capita	750	44682.21	8315.24	28919.48	71475.69
obesity rate	750	59.8	4.5	46.4	70.3
%agriculture	750	1.32	1.44	0.11	8.89
%mining	750	3.02	6.05	0.00	40.31
%utilities	750	2.14	0.63	0.60	4.43
%construction	750	5.52	1.47	2.89	14.80
%manufacturing	750	12.44	5.52	1.54	31.37

(including state dummies to account for unobserved heterogeneity and year dummies) which are robust to cross-sectional dependence, AR(1) serial correlation within each state, and heteroskedasticity. If cross-sectional dependence is present the fixed effects estimator is still consistent (but inefficient), if the unobservables generating this (spatial) dependence are uncorrelated with the other explanatory variables in the model (Hoechle, 2007). If however, these unobservables are correlated with the explanatory variables, then the fixed effects estimator is both biased and inconsistent (De Hoyos and Sarafidis, 2006). In this case given the likely strong spatial dependence among states in similar regions with respect to geography and commerce the fixed effects estimator is likely inappropriate. A Pesaran test for cross-sectional dependence (De Hoyos and Sarafidis, 2006) is implemented for total CO_2 emissions, per capita CO_2 emissions, and CO_2 intensity and the null of cross-sectional independence is rejected in each case ($p = 0.015$) so there is significant evidence of cross-sectional dependence, a result confirmed by Frees' test (Frees, 1995). Therefore, the FE estimates are likely bias and inconsistent. Additionally, a certain degree of path dependence is expected in state-level emissions so we consider a dynamic PW model with panel-corrected standard errors which assumes idiosyncratic errors which are first-order serially correlated within panels (states) and heteroskedastic and contemporaneously correlated across panels. This model would be an improvement over the static fixed effects model. However, the inclusion of a lagged value of the dependent variable introduces the possibility of a unit-root in some panels and the potential inconsistency resulting from the likely correlation between the unobserved effects and the lagged dependent variables, often referred to as Nickell bias (Nickell, 1981). Typically, the inclusion of this lagged dependent variable would warrant the use of dynamic panel estimators, such as Arellano and Bond (1991) or Blundell and Bond (1998). However, these estimators assume cross-sectional independence so we would be trading one source of bias and inconsistency for another.

4. Results

Given the potential for substantial bias generated by spatial dependence in the fixed effect model, we continue with a dynamic specification of the Prais-Winsten model, following Beck and Katz (1996). In order to test for a unit-root we implement the robust Hadri Lagrange multiplier test designed for serially correlated heteroskedastic short panels (with N > T) and confirm that some panels may contain a unit-root in (logged) total CO_2 emissions, per capita CO_2 emissions, and the CO_2 intensity of real GDP.[3] To account for the presence of this unit-root in some panels we assume that the AR(1) parameters are unique to each panel. The results of this dynamic PW model are summarized in Table 2.

Table 2. Dynamic Prais-Winsten

VARIABLES	(1) ln(total CO_2)	(2) ln(per capita CO_2)	(3) ln(CO_2 intensity)
ln(obesity)	0.127**	0.122**	0.080
	(0.056)	(0.057)	(0.063)
ln(population density)	0.098	-0.233***	-0.240***
	(0.065)	(0.052)	(0.062)
ln(real GDP per capita)	0.094**	0.075*	-0.440***
	(0.042)	(0.044)	(0.056)
ln(%agriculture)	0.028***	0.026***	0.030***
	(0.008)	(0.008)	(0.009)
ln(%mining)	0.014*	0.014*	0.016*
	(0.007)	(0.007)	(0.009)
ln(%utilities)	0.0003	0.008	0.024
	(0.023)	(0.022)	(0.024)
ln(%construction)	0.077***	0.070***	0.084***
	(0.018)	(0.019)	(0.022)
ln(%manufacturing)	0.012	0.015	0.008
	(0.012)	(0.012)	(0.013)
lagged dependent variable	0.669***	0.663***	0.551***
	(0.062)	(0.062)	(0.063)
Constant	-0.500	0.716	1.932***
	(0.550)	(0.564)	(0.662)
Observations	699	699	699
R-squared	0.9997	0.9993	0.9998
Number of States	50	50	50
State FE	YES	YES	YES
Year FE	YES	YES	YES

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

The obesity elasticity is equal to 0.127 or 0.122 and significant for both total CO_2 emissions and per capita CO_2 emissions ($p = 0.02$ and $p = 0.03$) respectively, while the obesity elasticity is equal to 0.08 and

[3] For logged total CO_2, $LM = 12.795, p = 0.00$; for logged per capita CO_2, $LM = 21.724, p = 0.00$; and for logged CO_2 intensity, $LM = 28.290, p = 0.00$.

insignificant ($p = 0.20$) for CO_2 intensity of real GDP. Therefore, there is significant evidence that obesity and CO_2 emissions are correlated which provides support for the hypothesized causal relationship.

However, if higher obesity actually increases the CO_2 intensity of GDP (and thus total CO_2 emissions), then total CO_2 emissions should be more responsive to changes in real GDP when obesity rates are higher. In other words, higher obesity last period should increase the income (real GDP per capita) elasticity of emissions. To test this hypothesis, we estimate the following Prais-Winsten regression with panel-corrected standard errors with an AR(1) parameter common to all panels:

$$\Delta \ln C_t = \alpha + \beta \ln(obesity)_{t-1} + \gamma \Delta \ln(realGDPpc)_t + \delta(\ln(obesity)_{t-1} * \Delta \ln(realGDPpc)_t) + u_t$$

where C_t is total CO_2 emissions. The dependent variable is therefore the growth rate of total CO_2 emissions. The results of this regression are presented in Table 3. Here we are primarily interested in δ, the coefficient on the interaction term between the obesity rate last period and the growth rate of real GDP per capita. If this coefficient is positive it provides support for our hypothesis that obesity is acting to amplify the total

Table 3. PW growth rate model

	(1)	(2)
VARIABLES	Δln(total CO_2)	
$\ln(obesity)_{t-1}$	-0.129***	-0.143**
	(0.049)	(0.068)
$\ln(obesity)_{t-1}$ * Δln(real GDP per capita)	2.897**	2.922**
	(1.319)	(1.265)
Δln(real GDP per capita)	-11.36**	-11.50**
	(5.399)	(5.170)
ln(population density)		-0.085
		(0.057)
Constant	0.523***	0.973***
	(0.202)	(0.309)
Observations	700	700
R-squared	0.170	0.270
Number of States	50	50
State FE	NO	YES

Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

CO_2 emissions response to economic growth (or decline). This coefficient is positive and significant ($p = 0.052$) confirming our hypothesis. This result is relatively robust to the inclusion of other control variables and state fixed-effects, as presented in column 2 of Table 3. The coefficient remains positive and significant ($p = 0.066$). Thus it appears that obesity does indeed have an effect on total CO_2 emissions.

From Table 2 we obtain an obesity elasticity of 0.13 for total CO_2 emissions, a small but not irrelevant impact. The prevalence of overweight and obese adults in the United States increased from 51.4% in 1995 to 64.3% in 2013 (CDC, 2014), an increase of 25.1% and total annual CO_2 emissions from energy use have

increased by 73 million metric tons (EIA, 2014); our results imply that over this time period 3.3% of this increase in total CO_2 emissions from energy use (around 2.4 million metric tons per year) is attributable to the increased prevalence of obesity. To put this into perspective, about one metric ton of CO_2 is produced to the meet the average monthly energy demand of the typical American household for heating, cooling, cooking, electricity use, and other energy needs; therefore increased prevalence of obesity is similar to the effect of having an additional 160,833 households, or nearly half a million additional people, in United States every year.[4]

5. Climate Co-benefits of Obesity Reduction

These results suggest that policies designed to reduce obesity may produce direct health benefits and climate co-benefits in the form of reduced CO_2 emissions. Using the results from the dynamic PW model just discussed we calculate that reversion to 1997 obesity rates in every state nationwide in 2014 would reduce annual U.S. CO_2 emissions from energy use by 143 million metric tons, or 2.7% below 2013 emission levels. Using estimates of the social cost of carbon (SCC) from the U.S. Environmental Protection Agency (EPA) of $40 or $62 per metric ton, for discount rates of 3% and 2.5% respectively, this reduction yields annual climate benefits of between $5.7 and $8.9 billion (in 2014 dollars). These benefits are not inconsequential. The EPA estimates that direct climate benefits from implementation of the Clean Power Plan (using the same SCC estimates and discount rates just discussed) are between $2.9 and $4.3 billion in 2020 and between $10.5 and $15.8 billion in 2025 (in 2014 dollars) for annual emission reductions of 63 and 211 million metric tons, respectively.[5] Therefore, the potential for climate co-benefits of obesity reduction in the United States is substantial and can provide additional incentive for obesity reduction at the individual, community, and national level.

6. Discussion

The various channels through which this association occurs were not investigated here and warrant additional research, but our results are suggestive of a mutually-reinforcing relationship between policies designed to improve public health (via reduced obesity) and climate change mitigation. Single-use zoning

[4] The percent change in the obesity rate from 1995-2013 is 25.1, our estimated elasticity (in the total CO_2 emissions model) is equal to 0.127, so an increase in the obesity rate of this amount yields an increase in emissions of 2.65%. Therefore, 2.65% of the total increase in annual emissions of 73 million metric tons (from 5323 to 5396 million metric tons) is 1.93 million metric tons. According to the EPA, the average American household generates 12 metric tons of CO_2 per year, therefore these additional emissions are the equivalent to the effect of having (=1,930,000/12) 160,833 additional households, or approximately (=160,833*2.6) 418,167 additional people, assuming the average US household size of 2.6 persons in 2010, according to the US Census Bureau.

[5] Using EPA estimates derived using rate-based approach, as described in the Regulatory Impact Analysis for the Clean Power Plan Final Rule (EPA, 2015). Estimates, reported in 2011 US dollars, were converted to 2014 dollars using a CPI conversion factor of 1.05.

and the resulting transportation emissions are some of the leading contributors to unsustainable suburban sprawl (Ewing and Hamidi, 2015) and the sedentary nature of automobile commuting is often cited as a contributing factor in rising obesity prevalence (Malik et al., 2013). Policies designed to reduce sprawl via mixed-use zoning in suburban areas can reduce miles traveled and time spent commuting, reducing both fuel use and obesity rates. This has the potential for direct and indirect climate benefits. Policies designed to promote active transport, such as improving walkability in cities, can reduce fuel use and emissions alongside improvements in public health leading to direct and indirect climate benefits. Policies designed to improve the geographic availability of supermarkets and eliminate food deserts can both reduce fuel use from transportation and reduce obesity through improved access to fresh food. While carbon pricing certainly remains the most effective climate change mitigation strategy, the indirect climate co-benefits resulting from obesity reduction have the potential to further improve the cost-effectiveness of potential carbon pricing policies. For example, if higher meat (especially ruminant) consumption contributes to obesity prevalence and meat consumption is carbon intensive, then potentially higher meat prices resulting from a carbon price would generate reductions in meat consumption (providing direct climate benefits) and reductions in obesity (providing indirect climate benefits). These co-benefits have the potential to provide needed cost-effectiveness as the goals of sustainable development, along its many dimensions, are pursued.

7. Conclusion

Our results demonstrate the importance of the built environment for *both* public health and sustainable development. “Where people live, how they get around, how much they eat and are physically active—all contribute to the epidemics of obesity and chronic disease” (Jackson et al., 2013). The same could be said for anthropogenic climate change. The potential for mutually-reinforcing policy outcomes is significant, while carbon pricing remains a more effective mitigation strategy, obesity reduction has its place in a movement to a low-carbon future.

8. References

Arellano, M., & Bond, S. (1991). Some Tests of Specification for Panel Data: Monte Carlo Evidence and an Application to Employment Equations. *The Review of Economic Studies, 58*(2), 277-297.

Beck, N., & Katz, J. (1996). Nuisance vs. Substance: Specifying and Estimating Time-Series-Cross-Section Models. *Political Analysis, 6*(1), 1-36.

Blundell, R., & Bond, S. (1998). Initial conditions and moment restrictions in dynamic panel data models. *Journal of Econometrics, 87*(1), 115-143.

Buonocore, J. (2014). Climate policy not so costly. *Nature Climate Change, 4*, 861-862.

CDC. (2014). *Adult Obesity Facts*. Retrieved from Division of Nutrition, Physical Activity, and Obesity, National Center for Chronic Disease Prevention and Health Promotion: http://www.cdc.gov/obesity/data/adult.html

Dannenberg, A. L., Burton, D. C., & Jackson, R. J. (2004). Economic and environmental costs of obesity: the impact on airlines. *American Journal of Preventive Medicine, 27*(3), 264.

De Hoyos, R. E., & Sarafidis, V. (2006). Testing for cross-sectional dependence in panel-data models. *The Stata Journal, 6*(4), 482-496.

Dee, A., Kearns, K., O'Neill, C., Sharp, L., Staines, A., O'Dwyer, . . . Perry, I. (2014). The direct and indirect costs of both overweight and obesity: a systematic review. *BMC Research Notes, 7*(242).

Edwards, P., & Roberts, I. (2009). Population adiposity and climate change. *International Journal of Epidemiology, 38*(4), 1137-1140.

EIA. (2014). *U.S. Energy -Related Carbon Dioxide Emissions, 2013.* Washington D.C.: U.S. Energy Information Administration, Department of Energy.

EPA. (2015). *Regulatory Impact Analysis for the Clean Power Plan Final Rule.* Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Air Quality Planning and Standards.

Evans, B. (2012). Climate change and the politics of fatness. *Environmental Politics, 21*(2), 334-336.

Ewing, R., & Hamidi, S. (2015). Compactness versus Sprawl: A Review of Recent Evidence from the United States. *Journal of Planning Literature, 30*(4), 413-432.

Frees, E. W. (1995). Assessing cross-sectional correlation in panel data. *Journal of Econometrics, 69*(2), 393-414.

Gallar, M. (2010). Obesity and climate change. *International Journal of Epidemiology, 39*(5), 1398-1399.

Goodman, A., Brand, C., & Ogilvie, D. (2012). Associations of health, physical activity and weight status with motorised travel and transport carbon dioxide emissions: a cross-sectional, observational study. *Environmental Health, 11*(52).

Green, M., Strong, M., Razak, F., Subramanian, S., Relton, C., & Bissell, P. (2015). Who are the obese? A cluster analysis exploring subgroups of the obese. *Journal of Public Health*, doi: 10.1093/pubmed/fdv040.

Gryka, A., Broom, J., & Rolland, C. (2012). Global warming: is weight loss a solution? *International Journal of Obesity, 36*, 474-476.

Herzoug, L., Sjodin, A., & Astrup, A. (2012). A proposed potential role for increasing atmospheric CO2 as a promoter or weight gain and obesity. *Nutrition and Diabetes, 2*, e31; doi:10.1038/nutd.2012.2.

Hoechle, D. (2007). Robust standard errors for panel regressions with cross-sectional dependence. *The Stata Journal, 7*(3), 281-312.

IPCC. (2014). Climate Change 2014: Synthesis Report. Contribution of Working Groups I, II and III to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. In R. Pachauri, & L. Meyer (Eds.). Geneva: Intergovernmental Panel on Climate Change.

Jackson, R., Dannenberg, A., & Frumkin, H. (2013). Health and the Built Environment: 10 Years After. *American Journal of Public Health, 103*(9), 1542-1544.

Jorgenson, A. K., Auerbach, D., & Clark, B. (2014). The (De-) carbonization of urbanization, 1960-2010. *Climatic Change, 127*(3-4), 561-575.

Ledikwe, J. H., Blanck, H. M., Kettel Khan, L., Serdula, M. K., Seymour, J. D., Tohill, B. C., & Rolls, B. J. (2006). Dietary energy intensity is associated with energy intake and weight status in US adults. *The American Journal of Clinical Nutrition, 83*, 1362-1368.

Liddle, B. (2014). Impact of population, age structure, and urbanization on carbon emissions/energy consumption: evidence from macro-level, cross-country analyses. *Population and Environment, 35*(3), 286-304.

Lowe, M. (2014). Obesity and climate change mitigation in Australia: overview and analysis of policies with co-benefits. *Australian and New Zealand Journal of Public Health, 38*(1), 19-24.

Luber, G., Knowlton, K., Balbus, J., Frumkin, H., Hayden, M., Hess, J., . . . Ziska, L. (2014). Ch. 9: Human Health. Climate Change Impacts in the United States: The Third National Climate Assessment. In J. Melillo, T. Richmond, & G. Yohe (Eds.). US Global Change Research Program.

Malik, V. S., Willet, W. C., & Hu, F. B. (2013). Global obesity: trends, risk factors, and policy implications. *Nature Reviews Endocrinology, 9*(1), 13-27.

McCormack, G. R., & Virk, J. S. (2014). Driving towards obesity: A systematized literature review on the association between motor vehicle travel time and distance and weight status in adults. *Preventive Medicine, 66*, 49-55.

McMichael, A. J. (2013). Globalization, Climate Change, and Human Health. *The New England Journal of Medicine, 368*(14), 1335-1343.

Michaelowa, A., & Dransfeld, B. (2008). Greenhouse gas benefits of fighting obesity. *Ecological Economics, 66*, 298-308.

Nickell, S. (1981). Biases in Dynamic Models with Fixed Effects. *Econometrica, 49*(6), 1417-1426.

NOAA. (2015). *Trends in Atmospheric Carbon Dioxide*. Retrieved from National Oceanic and Atmospheric Administration, Earth System Research Laboratory, Global Monitoring Division: http://www.esrl.noaa.gov/gmd/ccgg/trends/

OECD. (2010). *Obesity and the Economics of Prevention: Fit not Fat.* Paris: OECD Publishing.

Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2014). Prevalence of Childhood and Adult Obesity in the United States, 2011-2012. *The Journal of the American Medical Association, 311*(8), 806-814.

Reisch, L. A., & Gwozdz, W. (2011). Chubby cheeks and climate change: childhood obesity as a sustainable development issue. *International Journal of Consumer Studies, 35*, 3-9.

Ripple, W. J., Smith, P., Haberl, H., Montzka, S. A., McAlpine, C., & Boucher, D. H. (2014). Ruminants, climate change and climate policy. *Nature Climate Change, 4*, 2-5.

Rosa, E. A., & Dietz, T. (2012). Human drivers of national greenhouse-gas emissions. *Nature Climate Change, 2*, 581-586.

Russell, C., Cameron, E., Socha, T., & McNinch, H. (2013). "Fatties Cause Global Warming": Fat Pedagogy and Environmental Education. *Canadian Journal of Environmental Education, 18*, 27-45.

Scarborough, P., Appleby, P. N., Mizdrak, A., Briggs, A. D., Travis, R. C., Bradbury, K. E., & Key, T. J. (2014). Dietary greenhouse gas emissions of meat-eaters, fish-eaters, vegetarians and vegans in the UK. *Climatic Change, 125*, 179-192.

Schneker, N., & Taylor, J. M. (1996). Partially parametric techniques for multiple imputation. *Computational Statistics and Data Analysis, 22*, 425-446.

Shah, N. R., & Braverman, E. R. (n.d.). Measuring Adiposity in Patients: The Utility of Body Mass Index (BMI), Percent Body Fat, and Leptin. *PLoS ONE, 7*(4), e33308. doi:10.1371/journal.pone.0033308.

Shaw, C., Hales, S., Howden-Chapman, P., & Edwards, R. (2014). Health co-benefits of climate change mitigation policies in the transport sector. *Nature Climate Change, 4*, 427-433.

Squalli, J. (2014). Is obesity associated with global warming? *Public Health, 128*, 1087-1093.

Stehfest, E., Bouwman, L., van Vuuren, D. P., den Elzen, M. G., Eickhout, B., & Kabat, P. (2009). Climate benefits of changing diet. *Climatic Change, 95*, 83-102.

Tom, M., Fischbeck, P., & Hendrickson, C. (2014). Excess passenger weight impacts on US transportation systems fuel use (1970-2010). *Journal of Transport and Health, 1*, 153-164.

Tsai, A., Williamson, A., & Glick, H. (2011). Direct medical cost of overweight and obesity in the USA: a qualitative systematic reveiw. *Obesity Reviews, 12*, 50-61.

Underwood, A., & Zahran, S. (2015). The carbon implications of declining household scale economies. *Ecological Economics, 116*, 182-190.

Walpole, S. C., Prieto-Merino, D., Edwards, P., Cleland, J., & Stevens, G. (2012). The weight of nations: an estimation of adult human biomass. *BMC Public Health, 12*(439).

Wang, Y., & Beydoun, M. (2009). Meat consumption is associated with obesity and central obesity among US adults. *International Journal of Obesity, 33*, 621-628.

Webb, G. J., & Egger, G. (2014). Obesity and Climate Change: Can We Link the Two and Can We Deal With Both Together? *American Journal of Lifestyle Medicine, 8*(3), 200-204.

Wells, J. C. (2012). The evolultion of human adiposity and obesity: where did it all go wrong? *Disease Models and Mechanisms, 5*, 595-607.

WHO. (2003). *Diet, Nutrition and the Prevention of Chronic Diseases.* Geneva: World Health Organization.

WHO. (2015). *Obesity and Overweight Fact Sheet*. Retrieved from http://www.who.int/mediacentre/factsheets/fs311/en/

Wohlfahrt-Veje, C., Tinggaard, J., Winther, K., Mouritsen, A., Hagen, C., Mieritz, M., . . . Main, K. (2014). Body composition, energy expenditure, and physical activity. *European Journal of Clinical Nutrition, 68*, 664-670.

Zheutlin, A. R., Adar, S. D., & Park, S. K. (2014). Carbon dioxide emissions and change in prevalence of obesity and diabetes in the United States: An ecological study. *Environment International, 73*, 111-116.