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Carbon Dioxide Emissions and Change in Prevalence of Obesity and Diabetes in the United States: An Ecological Study

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Abstract

Recent studies suggest that increasing levels of the greenhouse gas, carbon dioxide (CO_2), may influence weight gain and thus may play a role in rising trends in obesity and diabetes. We conducted an ecological study to examine the associations between CO_2 emissions from fossil fuel combustion and changes in the prevalence of obesity and diabetes in the United States. County-level data on CO_2 emissions, prevalence of obesity and diagnosed diabetes, other sociodemographic factors and neighborhood characteristics related to urbanicity, and fine particles ($PM_{2.5}$) between 2004 and 2008 were obtained from the Vulcan Project, Centers for Disease Control and Prevention, and American Community Survey. Linear mixed effect modeling of 3019 counties for the associations between average CO_2 emissions and changes in diabetes and obesity prevalence between 2004 and 2008 was performed. The average obesity and diabetes prevalence increased between 2004 and 2008 by 3.65% (SD: 1.88%) and 1.65% (SD: 1.70%), respectively. A marginally significant positive association between CO_2 emission and changes in obesity prevalence was found with adjustment for sociodemographic factors, indicators of urbanicity and spatial autocorrelation (p-trend=0.06). The association became weaker and nonsignificant with further adjustment for $PM_{2.5}$ (p-trend=0.17). There was a significant positive association between CO_2 emission and changes in diabetes prevalence before controlling for $PM_{2.5}$ (p-trend=0.05) but

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the association became null after controlling for $PM_{2.5}$ (p-trend=0.49), suggesting $PM_{2.5}$ is a critical confounder in the association between CO_2 emission and changes in diabetes prevalence. This study does not support the hypothesis that CO_2 emissions, a leading driver of climate change, may be linked to increasing trends in obesity and diabetes, though there was an indication of possible link between CO_2 and obesity.

Keywords

Carbon dioxide; Climate change; Diabetes; Greenhouse gas; Obesity; $PM_{2.5}$

1. Introduction

Obesity and type-2 diabetes have been increasing dramatically over the past several decades in the world and can be considered global epidemics (Chen et al., 2012; Malik et al., 2013). According to the 2011 National Diabetes Fact Sheet, 36% and 11% of the United States (U.S.) population aged 20 years and older were afflicted with obesity and type-2 diabetes in 2010, respectively (CDC 2011). Recently, the American Medical Association announced obesity as a disease (Hoven 2013). It has been suggested that such epidemics are attributable to poor lifestyle behavior, such as higher caloric input and lower physical energy output. However, poor diet and sedentary lifestyle do not account for all of the variations in obesity and type-2 diabetes (Thayer et al., 2012), suggesting that other factors may also play an important role.

A recent study by Hersoug et al. (2012) proposed that increases in obesity and type-2 diabetes may be due to increased atmospheric carbon dioxide (CO_2). This hypothesis was motivated by a finding that animal species living under controlled environments (various laboratory rodents and primates) or near human establishments (cats, dogs, feral urban and rural rats) had significant weight gain over the last 50 years, although those animals have had the same diets for decades (Klimentidis et al., 2011). The documentation of greater average weight in numerous species indicates that there may be an environmental component applicable to all species impacting current trends in body composition. Plausible biological mechanisms by which higher CO_2 exposure may lead to weight gain and other metabolic disorders are poorly understood, although a decrease in blood pH and subsequent hormonal reactions by elevated atmospheric CO_2 were proposed (Hersoug et al., 2012; Sunanaga et al., 2009). To our best knowledge, there is no study examining the association between atmospheric CO_2 and obesity and type-2 diabetes. Any such association could have important implications since the subtle accumulative effects of climate change on human health are relatively unknown. Furthermore it may hint at a previously unconsidered cost of increasing CO_2 in the atmosphere.

Using data from the Vulcan Project, Centers for Disease Control and Prevention (CDC), and American Community Survey we examined whether there were relationships between average fossil fuel CO_2 emissions and changes in diabetes and obesity prevalence at the county level in the U.S. between 2004 and 2008. We hypothesized that a positive change in the prevalence of obesity or diabetes would be associated with greater emissions of

atmospheric CO_2, independent of factors commonly associated with diabetes and obesity and neighborhood characteristics related to urbanicity, and fine particles (particulate matter less than 2.5 μm in diameter, $PM_{2.5}$). We also examined a secular trend of blood CO_2 as measured by serum bicarbonate levels in U.S. using data from the National Health and Nutrition Examination Survey (NHANES) from 1999 to 2012 to explore whether there is a temporal correlation between ambient CO_2 concentration and blood CO_2 in the general U.S. population.

2. Material & Methods

2.1. CO_2 Emissions

Data for annual CO_2 emissions were collected from the Vulcan Project for years from 2004 to 2008. The Vulcan Project is an initiative funded by the National Aeronautics and Space Administration/the U.S. Department of Energy that has quantified fossil fuel emissions with high resolution across North America (Gurney et al., 2009). Emissions were assessed based on million tons of carbon at the county level based on the U.S. Environmental Protection Agency's (EPA) National Emissions Inventory (NEI) data. The NEI provides carbon monoxide (CO) emissions by facility or unit, as well as nitrogen oxides (NO_x) and emission factors (EF). CO, NO_x, and EF are formulaically inserted to calculate a total fuel throughput, which is then used to find CO_2 emissions by accounting for emissions from pollutant materials (Gurney et al., 2009). CO_2 emission levels in U.S. did not change much between 2004 and 2006, then increased in 2007 and decreased in 2008 (EPA 2013a). Several factors influenced this year to year variability, such as energy (fuel) prices and weather conditions (EPA 2013a). For the exposure metric of our study we used a five-year average of county level CO_2 between 2004 and 2008.

2.2. Obesity and Diabetes Prevalence

County-level obesity and diabetes prevalence between 2004 and 2008 was obtained from the National Diabetes Surveillance System at the CDC (CDC 2012b). These data come from the Behavioral Risk Factor Surveillance System (BRFSS), which is an ongoing, monthly, state-based telephone survey of U.S. adults (CDC 2012a). Interviewees were considered obese if their body mass index (BMI) ≥30 kg/m^2, which was computed using self-report of height and weight. Interviewees who had been told by a physician they were diabetic were considered to have diagnosed diabetes. Women who indicated that they only had diabetes during pregnancy were not considered to have diabetes. The CDC used three-year averages of county-level estimates to improve the precision of data: for example, 2004 estimates were computed using data from 2003, 2004 and 2005 (CDC 2013a). County level prevalence was reported as age-adjusted percent of the population aged 20 years or older. The primary outcomes were determined to be change in prevalence of obesity or diagnosed diabetes between 2004 and 2008, which was computed based on the difference in prevalence levels provided for 2004 and 2008. There are no temporal correlation issues in the outcome measures in the present study because we examined the difference in prevalence as the outcome rather than absolute prevalences from multiple years.

2.3. County-Level Characteristics and Risk Factors

Five-year estimates from the U.S. Census American Community Survey (ACS) covering 2005-2009 were used for county-wide covariate data (Commerce 2012). The ACS five-year estimate was selected due to greater precision, and included more data points as opposed to one or three year estimates which included less counties. This included median age as well as percentages of males, persons aged 25 years or older with a high school degree or equivalent, and population with income below the poverty level. Poverty level is identical throughout the U.S. except Alaska and Hawaii, and is calculated based upon the household income, the household size, and the age of household members (ASPE 2014). Median age and percent male are based upon all age groups within each county, as there is no available data for median age or percent male based solely on adults. The ACS offers multiple race/ethnicity identification methods, and for this study individual identification as black or African American, Asian (including Asian Indian, Chinese, Filipino, Japanese, Korean, Vietnamese, and other), white, or Hispanic or Latino was used. Other race/ethnic groups such as American Indian/Alaska Native and Native Hawaiian/Other Pacific Islander were not included because proportions are too low and some regression models including those variables did not converge. Population sizes per county were divided by county land area (given in square miles) in order to estimate population density. Density of fast food restaurants (limited-service eating-places per 100,000-population count) was obtained from the 2006 BRFSS Supplement Survey. County-level leisure time physical inactivity which was also based upon the BRFSS survey was obtained from the CDC's National Diabetes Surveillance System along with diagnosed diabetes and obesity (CDC 2012b). Physical inactivity was defined as no participation in any physical activities or exercises other than regular job such as running, calisthenics, golf, gardening, or walking for exercise, during the past month (CDC 2013a). County-level $PM_{2.5}$, a combination of monitored and modeled data, was obtained from the CDC's National Environmental Public Health Tracking Network, a joint venture between the CDC and Environmental Protection Agency (CDC 2013c). Additionally, variables were added to each dataset for the latitude and longitude of each county centroid to explore spatial trends.

2.4. Serum Bicarbonate and Ambient CO_2

We obtained serum bicarbonate data from the NHANES cycles between1999/2000 and 2011/2012 (CDC 2013b). Bicarbonate concentrations were determined with Hitachi Multichannel analyzer (Model 917) in the NHANES 1999/2000 and 2001/2002 cycles; Beckman Synchron LX 20 in the 2003/2004 and 2005/2006 cycles; and Beckman UniCel DxC 800 Synchron in the NHANES cycles from 2007/2008 to 2011/2012. We also collected complex sampling design factors such as primary sampling unit, strata and sampling weight from each cycle. We restricted only adults aged 20 years or older. A total of 33,546 subjects were included. Annual averages of atmospheric CO_2 concentrations at Mauna Loa, Hawaii were obtained from the Global Greenhouse Gas Reference Network available at the Earth System Research Laboratory, the U.S. National Oceanic & Atmospheric Administration (NOAA/ESRL 2014). CO_2 concentrations at the Mauna Loa Observatory represent the longest record of direct measurements of CO_2 in the atmosphere since 1958, which covers CO_2 data for the period of NHANES cycles between 1999 and 2012.

2.5. Data Analysis

Spatial maps were first created in order to present the distribution of CO_2 emissions, diabetes, and obesity across the continental U.S. between the years of 2004 and 2008 using Arc GIS v9.3.1 (ESRI, Redlands, CA). We then examined the distributions (mean, standard deviation (SD), minimum and maximum) of all variables. We assessed the Spearman's rank correlations between covariates considered and the outcomes (changes in the prevalence of obesity and diabetes from 2004 to 2008) and the exposure (5-year average of CO_2 emission). All data analyses were conducted using R version 2.15 (R Foundation for Statistical Computing, http://www.r-project.org).

Of the 3140 U.S. counties and county-equivalents, 3019 (96%) were included for statistical analysis. The 121 counties excluded in the study were withheld due to missing data on fast food density, or percent of population above 25 years of age with a high school degree or equivalent. Additional 25 counties in Alaska and Hawaii where $PM_{2.5}$ data are not available were excluded, leaving 2994 counties in the model with $PM_{2.5}$. Mixed effects models were fitted to examine the relationship between changes in prevalence of obesity or diabetes and quintiles of CO_2 emissions and to account for spatial autocorrelation. We used quintiles rather than continuous CO_2 as our primary form of exposure because the relationships did not appear linear (supplemental Figure 1). We considered three spatial correlation structures using information on the latitude and longitude of each county centroid; Gaussian, spherical, and exponential but selected exponential using the Akaike Information Criteria. Potential confounding factors considered include male prevalence, median age, race (percent black/African American, Asian, white, and Hispanic/Latino), poverty prevalence, high-school or equal educational attainment for those over 25 years of age, physical inactivity, fast food density, and population density. We additionally controlled for $PM_{2.5}$ because CO_2 emission was highly correlated with ambient $PM_{2.5}$ concentrations and a recent study reported a significant association between county-level $PM_{2.5}$ and diabetes prevalence in U.S. (Pearson et al., 2010). For diabetes, we further adjusted for change in obesity. Parameter estimates and 95% confidence intervals (CIs) were computed by comparing each of the upper 4 quintiles of CO_2 emission with the lowest quintile. Tests for linear trend were conducted using ordinal terms for the quintiles.

We plotted biannual averages of ambient CO_2 concentrations at the Mauna Loa Observatory (Hawaii) and serum bicarbonate concentrations against the continuous NHANES cycles from 1999/2000 to 2011/2012 to examine temporal trends and potential correlations. Survey linear regression was used to compute a biannual change in serum bicarbonate concentrations.

3. Results

Obesity and diabetes prevalence have varied over the five-year span of 2004-2008, increasing by an average of 3.67% (SD: 1.89%, range: −3.4% to 12.6%) for obesity and 1.64% (SD: 1.67%, range: −6.2% to 9.5%) for diabetes (Table 1). The average CO_2 emission level for counties across the U.S. was 0.55 million tons of carbon (SD=1.29 million tons, range: 0.002 to 21.7 million tons). The county-level annual $PM_{2.5}$ concentration averaged between 2004 and 2008 was 11.4 $\mu g/m^3$ (SD=2.47 $\mu g/m^3$, range:

4.48 to 20.1 μg/m^3). All sociodemographic variables, indicators of urbanicity, and $PM_{2.5}$ were significantly associated with CO_2 emission levels (p-values for trends across quintiles of CO_2 emission <0.0001). County-level estimates of the 5-year average CO_2 emission levels and changes in the prevalence of obesity and diagnosed diabetes between 2004 and 2008 are shown in Figure 1. High CO_2 emissions were found in southwest states, northeastern coasts, and Florida.

Table 2 provides the Spearman's rank correlation coefficients (rho) between each covariate and the two outcomes (change in diabetes and obesity prevalence). The Spearman's rank correlation demonstrated evidence that counties with higher changes in obesity generally had higher changes in diabetes though the two were not strongly correlated (rho=0.12). Counties that had higher change in diabetes also had higher mean CO_2 emissions (rho=0.16). Change in obesity prevalence was weakly correlated with CO_2 emission (rho=0.03). Counties with increase in physical inactivity, population density, and percent black or African American all showed increasing change in prevalence of diabetes and obesity, while education attainment, percent white, and median age showed a decrease in prevalence of both diseases.

In models adjusted for sociodemographic variables, indicators of urbanicity, and spatial autocorrelation (exponential), higher CO_2 emission levels were marginally associated with larger changes in obesity prevalence from 2004 to 2008 (p for trend=0.06) (Table 3). As compared to counties within the first quintile, the upper four quintiles had 0.093% (95% CI, −0.115% to 0.300%), 0.226% (0.007% to 0.445%), 0.163% (−0.073% to 0.399%), and 0.241% (−0.014% to 0.497%) larger changes in prevalence than the first quintile. The strength of the association slightly decreased and became nonsignificant with further adjustment for $PM_{2.5}$ (changes in obesity prevalence in the upper four quintiles: 0.068% (95% CI, −0.140% to 0.276%), 0.171% (−0.051% to 0.392%), 0.111% (−0.133% to 0.354%), and 0.196% (−0.071% to 0.463%); p for trend=0.17). For diabetes, we found a significant positive association between CO_2 emission and changes in diabetes prevalence (changes in diabetes prevalence in the upper four quintiles: −0.086% (95% CI, −0.255% to 0.083%), 0.119% (−0.159% to 0.197%), 0.156% (−0.036% to 0.348%), and 0.121% (−0.087% to 0.328%); p for trend<0.05) but the association became null after controlling for $PM_{2.5}$ (changes in diabetes prevalence in the upper four quintiles: −0.136% (95% CI, −0.309% to 0.037%), −0.044% (−0.225% to 0.138%), 0.087% (−0.109% to 0.283%), and −0.027% (−0.242% to 0.187%); p-trend=0.49).

Figure 2 shows the temporal trends in biannual averages of atmospheric CO_2 concentrations at Mauna Loa, Hawaii, and serum bicarbonate concentrations in U.S. adults. There was an increasing trend in serum bicarbonate concentrations. On average, a 0.27 mmol/L (95% CI, 0.20 to 0.33) increased every cycle (every two years) between 1999/2000 and 2011/2012 (data not shown).

4. Discussion

This study does not support the hypothesis that CO_2, a leading driver of climate change, may be linked to increasing trends in obesity and diabetes. Although statistical significance

was not achieved, the association between CO_2 emission and the change in obesity prevalence showed the expected positive direction even after controlling for ambient $PM_{2.5}$. On the other hand, the significant association between CO_2 emission and the change in diabetes prevalence completely disappeared after controlling for ambient $PM_{2.5}$, suggesting that fine particles, not CO_2, may play a role in the increasing trend of diabetes prevalence and are an important confounder of the association between CO_2 and the change in diabetes prevalence. The present study also showed that serum bicarbonate, a marker of body CO_2 levels, increased significantly in the general U.S. population over the past 14 years. Potential factors that may have driven this trend including atmospheric CO_2 were not determined. Our findings suggest that increases in sources of CO_2 from fossil fuel combustion in the U.S. may not only have implications for global warming but could also be a contributing factor to the rising rates of obesity prevalence. This is consistent with a biological model proposed by Hersoug et al. (2012), who suggest that serum CO_2 can be a catalyst for weight gain, although our study findings cannot be interpreted as causal.

Our findings should be interpreted with caution. The nature of this study does not allow for conclusion that increased CO_2 exposure shows a causal relationship with diabetes or obesity. This is an ecological study and therefore may be the result of the ecological fallacy (Piantadosi et al., 1988). For example, areas of greater CO_2 emissions may have greater average income and/or greater use of vehicles. Bassetti *et al.* has previously demonstrated a "strong, long-run, positive relationship between income and CO_2 emissions" (Bassetti et al., 2013). Health care access may be greater in those areas, and have higher diagnosis rates because of this. Also, CO_2 emissions from motor vehicles contribute to approximately 31% of total U.S. CO_2 releases and 26% of total U.S. greenhouse gas emissions in 2011 (EPA 2013b). Although we controlled for neighborhood indicators of socioeconomic position (e.g., race/ethnicity, educational attainment, and percent below poverty), population density, physical inactivity, access to fast food restaurants, and ambient fine particles, it remains possible that there is residual confounding by other factors unknown to this study.

We also cannot rule out a possibility of reverse causality, in that, counties with increased obesity prevalence emit more CO_2. Some argue that obese individuals may contribute to CO_2 emission through increased food consumption and greater requirements for transportation energy (Edwards and Roberts 2009). Edwards and Roberts (2009) reported that obese individuals lead to an increase of 0.173 gigatons and 2.038 gigatons in greenhouse gas emissions in the U.S. per year due to greater energy requirements for car and air travel. They also suggested that greater availability of cheap marketable food is connected to higher rates of purchase without complete consumption, which increases food waste and leads to more greenhouse gas emissions when it decomposes (Edwards and Roberts 2009). In 2003 the energy burden of wasted food amounted to 4% of the total oil consumption of the United States (Hall et al., 2009). While many have studied obesity as a cause for CO_2, evidence to support this is not convincing. Obesity is much more common in low socioeconomic areas, and the availability for personal automobile utilization is significantly lower in these areas (Gallar 2010). Additionally, no study of CO_2 emissions has been able to identify purchasing groups, and to assume it is obese individuals consuming goods that contribute to greenhouse gases is not based upon data (Gallar 2010).

Nevertheless, further investigation is needed to identify temporality of the association between CO_2 emission and obesity.

We examined CO_2 emissions rather than atmospheric CO_2 concentrations as the primary exposure variable because data for atmospheric CO_2 are restricted to few testing stations that do not provide an adequate representation of county-level concentrations. Although atmospheric CO_2 concentrations are known to be correlated with CO_2 emissions (IPCC 2013), it is unclear to what extent spatial variations in atmospheric CO_2 concentrations exist at the county-level. Our statistical approaches to control for spatial autocorrelations and categorization of exposure could account for this potential issue though this study is still subject to exposure misclassification.

Despite many limitations and non-significant findings, this study showed an indication of possible link between CO_2 from fossil fuel combustion and the prevalence of obesity. However, underlying biological mechanisms are poorly understood. It is hypothesized that increased ambient CO_2 exposure can lower blood pH which in turn initiates a cascade response in the hypothalamus by activating orexin neurons (Hersoug et al., 2012). Recent studies have shown possible mechanisms by which orexin can impact weight disposition of organisms. Under hypercapnic conditions of 10% CO_2, Sunanaga et al. (2009) measured increased stimulation of orexin neurons by CO_2 and H^+ demonstrated by elevated c-Fos expression. Both CO_2 and extracellular acid are physiochemical signals for wakefulness, and excess presence in serum can lead to higher excitability (Williams et al., 2007). Longer exposures to elevated CO_2 can result in decreased pH of the blood, even after exposure to air with lower concentration of CO_2 (King et al., 1955). This increased acidity of the blood can restrict movement of ions through post-synaptic K^+ leak-like channels, which can increase the firing of orexin neurons, and thus up the concentration of orexin neurotransmitters in the body (Williams et al., 2007). By stimulating the body's desire for increased caloric intake, individuals are disposed to weight gain. The alteration of homeostatic regulation by the hypothalamus, leading to increase orexin neuron activity, induced by CO_2, is a plausible path for excess weight gain (Hersoug et al., 2012). Further studies are necessary to elucidate potential biological mechanisms of the link between CO_2 exposure and weight gain and obesity. In particular, the increasing trend in serum bicarbonate found in our study is related to elevated ambient CO_2 and climate change needs to be determined.

Our study suggests that fine particles, not CO_2 emission, may play a role in the change in diabetes prevalence. In the fully adjusted model including both CO_2 emission and ambient $PM_{2.5}$, a 10 μg/m^3 increase in $PM_{2.5}$ was associated with a 0.90% (95% CI, 0.58% to 1.21%) increase in the diabetes prevalence ($p<0.0001$, data not shown). $PM_{2.5}$ was not associated with changes in obesity prevalence ($p=0.26$, data not shown). This is consistent with the previous finding that counties with high levels of $PM_{2.5}$ had significantly greater prevalence of diagnosed diabetes in U.S. (Pearson et al., 2010). Numerous observational studies also supported the link between long-term air pollution exposure to the risk of type-2 diabetes (Brook et al., 2013; Coogan et al., 2012; Kramer et al., 2010). $PM_{2.5}$ exposure to mice, with diet-induced insulin resistance, promoted greater insulin resistance and visceral inflammation and influenced insulin signaling pathway and apoptosis, key biological mechanisms in type-2 diabetes (Sun et al., 2009; Xu et al., 2011a; Xu et al., 2011b).

Although obesity is an important risk factor of type-2 diabetes (Schulze and Hu 2005) and thus we hypothesized that CO_2 may influence two related metabolic outcomes, our study does not support this hypothesis probably due to different biological mechanisms between CO_2 and $PM_{2.5}$. The present study examined data between 2004 and 2008 because CO_2 emission data in the U.S. at the county level are available only in this period and it allowed for maximum county inclusion from all of the providing data sources. It may be that five years does not provide a long enough time frame to fully examine how CO_2 levels play into obesity or diabetes prevalence at the population level. Measuring CO_2, especially as ambient concentrations, over an extended period of time has been restricted to specific sites globally, and is not available on a county level in U.S. However, data from the Mauna Loa observatory demonstrates the increase in ambient CO_2. Specifically, from 2004 to 2008 atmospheric CO_2 from this location increased from 377.49 to 385.85 ppm and continued to increase to 396.48 ppm in 2013 (NOAA/ESRL 2014).

In summary, our study does not support a link between CO_2 emissions from fossil fuel combustion and increasing trends of obesity and diabetes, though there was an indication of possible link between CO_2 and obesity. Potential health impacts of climate change have received huge attentions recently, especially following acute natural disasters, such as Hurricane Sandy of 2012 and the Oklahoma tornadoes of 2013. Increased CO_2 is a key component of global climate change, which poses numerous threats such as food security by altering the preferred growing microclimate, human safety by heightened intensity and frequency of natural disasters (Fischer et al., 1994; Mills 2009). However, the subtle accumulative effects of increased CO_2 from climate change on human health are relatively unknown. It is an urgent need to explore potential cumulative effects of climate change, such as increasing atmospheric CO_2, especially on homeostatic regulation given widespread exposure to CO_2 in large populations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- We examined CO_2 emission and changes in prevalence of obesity and diabetes from 2004 to 2008 in US
- Higher CO_2 emission was not associated with larger changes in diabetes prevalence
- There was an indication of possible link between CO_2 emission and obesity prevalence changes
- This finding should be interpreted with caution as it is an ecological study

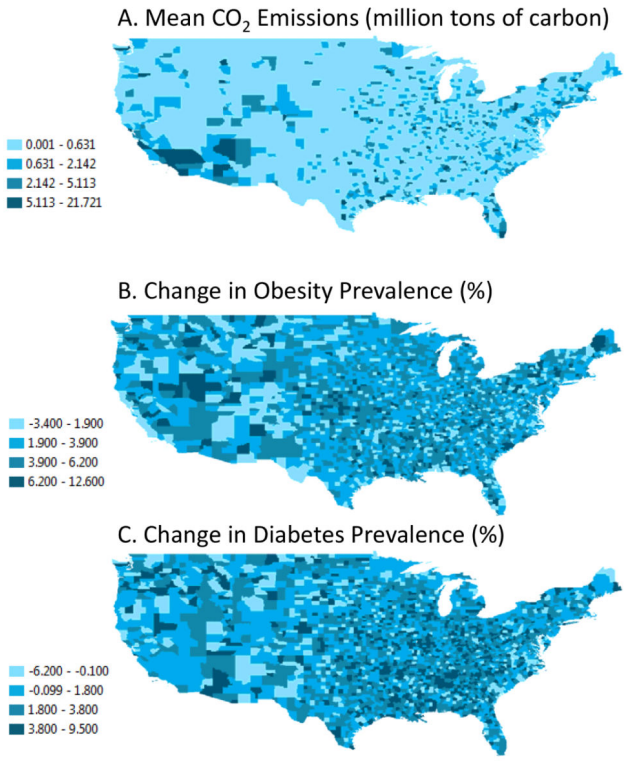

Figure 1.

Maps of the United States demonstrating county-levels of CO_2 emissions (average of annual levels between 2004 and 2008; million tons of carbon) (A), change in the prevalence (%) of obesity (B) and diagnosed diabetes (C) from 2004 to 2008.

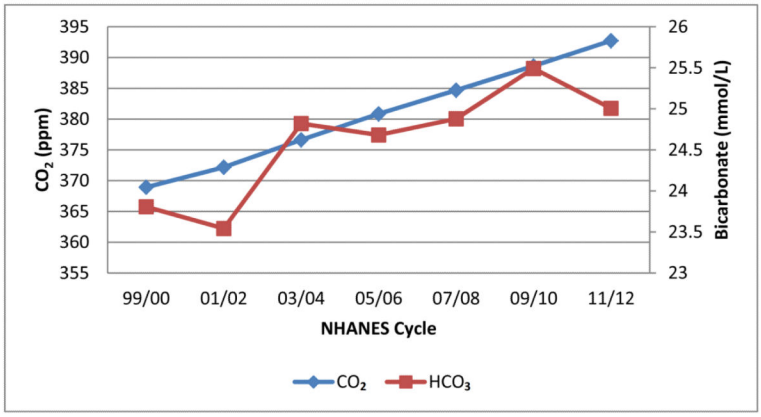

Figure 2.

Temporal trends in biannual averages of atmospheric CO_2 concentrations at Mauna Loa, Hawaii, and serum bicarbonate concentrations in U.S. adults.

Table 1

Distributions of the study variables for the 3019 counties analyzed.

	Mean (SD)	Range	Quintile of CO_2 emission [mean (SD)]				
			Q1	Q2	Q3	Q4	Q5
Average CO_2 emission (million tons emitted)	0.55 (1.29)	0.002, 21.7	0.03 (0.01)	0.06 (0.01)	0.13 (0.03)	0.31 (0.10)	2.23 (2.19)
Prevalence of obesity in 2004 (%)	25.3 (3.25)	12.3, 38.0	25.4 (3.28)	25.6 (3.13)	25.5 (3.15)	25.2 (3.36)	24.8 (3.32)
Prevalence of obesity in 2008 (%)	28.9 (3.71)	11.7, 43.7	29.1 (3.77)	29.1 (3.53)	29.2 (3.52)	28.9 (3.91)	28.4 (3.89)
Change in obesity (%)	3.67 (1.89)	−3.40, 12.6	3.50 (1.81)	3.66 (1.75)	3.83 (1.94)	3.72 (1.98)	3.61 (1.95)
Prevalence of diabetes in 2004 (%)	8.29 (1.59)	3.00, 14.6	8.51 (1.68)	8.33 (1.59)	8.31 (1.60)	8.21 (1.52)	8.05 (1.51)
Prevalence of diabetes in 2008 (%)	9.93 (2.05)	3.00, 18.2	10.1 (2.03)	9.96 (2.07)	10.1 (2.07)	9.93 (1.98)	9.70 (2.09)
Change in diabetes (%)	1.64 (1.67)	−6.20, 9.50	1.20 (1.66)	1.41 (1.62)	1.68 (1.64)	1.99 (1.62)	1.92 (1.68)
Percent Male*	49.8 (2.22)	42.7, 74.9	50.1 (2.87)	50.0 (2.46)	49.7 (2.00)	49.7 (2.06)	49.3 (1.34)
Median Age*	39.4 (4.82)	21.7, 55.0	42.3 (4.96)	40.5 (4.29)	39.0 (4.41)	37.8 (4.57)	37.2 (4.01)
Percent Hispanic or Latino	7.45 (12.6)	0.00, 98.6	5.90 (12.5)	7.04 (13.9)	6.61 (11.7)	7.16 (10.9)	10.5 (13.5)
Percent White	84.2 (16.1)	5.75, 100	87.3 (17.2)	86.0 (15.9)	84.2 (16.0)	84.7 (14.1)	78.8 (15.9)
Percent Black or African American	8.76 (14.2)	0.00, 86.8	6.77 (15.0)	8.26 (15.1)	9.71 (15.7)	7.94 (11.3)	11.1 (13.2)
Percent Asian	1.09 (2.46)	0.00, 47.5	0.30 (0.45)	0.45 (0.56)	0.63 (0.69)	1.30 (2.83)	2.73 (4.14)
Percent Below Poverty	15.4 (6.38)	1.40, 51.0	16.9 (7.53)	16.4 (6.48)	15.9 (6.30)	14.1 (5.38)	13.7 (5.29)
Percent of population 25 years and older with a high school degree or equivalency	82.5 (7.54)	46.5, 98.7	80.9 (8.52)	80.6 (8.02)	81.7 (7.42)	84.3 (6.35)	84.8 (6.06)
Population Density (population per square mile)	232 (1745)	0.08, 71000	17.2 (17.5)	37.3 (87.6)	64.5 (85.6)	137 (138)	908 (3828)
Percent physical inactivity in County	26.4 (5.10)	9.52, 43.5	28.0 (4.33)	27.5 (5.07)	26.9 (5.36)	25.1 (4.87)	24.5 (4.91)
Density for fast food restaurant (number of restaurants per 100,000 residents)	68.8 (36.1)	0.00, 867	59.0 (57.9)	63.3 (31.5)	69.4 (26.6)	75.9 (24.2)	81.4 (23.3)
Average $PM_{2.5}$ concentration†	11.0 (2.47)	4.48, 20.1	9.63 (2.34)	10.7 (2.36)	11.1 (2.22)	11.5 (2.31)	12.1 (2.39)

All variables had statistically significant linear trends across the quintiles of CO2 emission (p for trend <0.0001) except obesity variables.

*These variables were based on all age groups within each county, as there is no available data solely on adults in the U.S. American Community Survey.

†$PM_{2.5}$ available in 2994 counties (no $PM_{2.5}$ data available in Alaska and Hawaii).

Table 2

Spearman correlation coefficients between covariates considered and the outcomes (changes in the prevalence of obesity and diabetes from 2004 to 2008) (N=3019).

	Change in Obesity	Change in Diabetes
Change in Obesity	1	0.12
Change in Diabetes	0.12	1
Average CO_2 emission	0.03	0.16
Percent Male	−0.02	0.05
Median Age	−0.11	−0.34
Percent Hispanic or Latino	−0.06	0.07
Percent White	−0.03	−0.06
Percent Black or African American	0.08	0.16
Percent Asian	−0.02	0.14
Percent Below Poverty	0.09	−0.02
Percent of population 25 years and older with a high school degree or equivalency	−0.08	−0.05
Population Density	0.05	0.28
Percent physical inactivity	0.15	0.05
Density of fast food restaurant	−0.07	0.06
Average $PM_{2.5}$ concentration	0.11	0.26

Statistically non-significant correlations (p-value > 0.05) were between change in obesity and CO_2 emission; percent male; percent white; and percent Asian; and between change in diabetes and percent below poverty. P-values for all other correlation coefficients were < 0.01.

Table 3

Mean difference in change (95% CI) in obesity and diabetes prevalence between 2004 and 2008 for each quintile of CO_2 emissions.

	Quintile of CO_2 emission (range, million tons)						
	Q1 <0.0414	**Q2 0.0414 < 0.0881**	**Q3 0.0881 < 0.1810**	**Q4 0.1810 < 0.5540**	**Q5 >0.5540**	**P for trend**	**AIC**
Obesity							
Crude*	0	0.099 (−0.108,0.306)	0.242 (0.031,0.453)	0.130 (−0.088,0.349)	0.145 (−0.080,0.370)	0.21	12298.6
Model 1†	0	0.093 (−0.115,0.300)	0.226 (0.007,0.445)	0.163 (−0.073,0.399)	0.241 (−0.014,0.497)	0.06	12302.3
Model 2‡	0	0.068 (−0.140, 0.276)	0.171 (−0.051, 0.392)	0.111 (−0.133, 0.354)	0.196 (−0.071, 0.463)	0.17	12158.0
Diabetes							
Crude*	0	0.126 (−0.052,0.304)	0.390 (0.208,0.571)	0.658 (0.470,0.845)	0.600 (0.407,0.793)	<0.0001	11386.5
Model 1†	0	−0.086 (−0.255,0.083)	0.019 (−0.159,0.197)	0.156 (−0.036,0.348)	0.121 (−0.087,0.328)	0.05	11066.6
Model 2‡	0	−0.136 (−0.309, 0.037)	−0.044 (−0.225, 0.138)	0.087 (−0.109, 0.283)	−0.027 (−0.242, 0.187)	0.49	11054.5

AIC, Akaike Information Criteria.

*Crude: considering only spatial correlation (exponential correlation structure).

†Model 1: adjusted for change in obesity prevalence (diabetes only), percent male, median age, percent Hispanic or Latino, percent white, percent black or African American, percent Asian, percent below poverty, percent of population 25 years and older with a high school degree or equivalency, population density, percent physical inactivity, density for fast food restaurant, as well as exponential spatial correlation.

‡Model 2: Model 1 + $PM_{2.5}$.