Title: The effect of food environment on weight change: does residential mobility matter?

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Word count: 3,327
Pages: 20
Tables: 3

Financial disclosure: No financial disclosures were reported by the authors of this paper.

Abstract

Background: The evidence base on the relationship between food environment and body mass index (BMI) has not converged due to measurement and methodological limitations.

Purpose: This study assessed the within-individual association between changes in food environment and changes in BMI among adults with diabetes from 2006-2011.

Methods: The relationship between changes in food environment and 1-year lagged BMI was estimated using year and individual fixed effects. Food environment was measured using the kernel density of food venues hypothesized to have a positive influence on weight. Separate models were estimated for individuals who moved and those who stayed.

Results: There was a clinically negligible, inverse statistically significant effect of changes in food environment on BMI among individuals with no change of residence but not among those who moved.

Conclusions: Community-level policies to improve food environment are unlikely to have a clinically significant effect on BMI among adult patients with diabetes.

Keywords: body mass index; diabetes; residence characteristics; neighborhood-effects

Introduction

Over twenty-two million Americans, 7% of the U.S. population, had diabetes in 2012 at a total cost of $245 billion, including direct medical and lost productivity costs (Association 2013). Obesity is a major risk factor for development of type 2 diabetes; 60 to 90 percent of type 2 diabetes mellitus is associated with obesity or weight gain (Anderson, Kendall and Jenkins 2003). Weight management to manage blood glucose levels and reduce risk for cardiovascular disease based on a personalized diet and exercise plan is an important goal for diabetic patients (Inzucchi et al. 2012).

Recently, policymakers have focused on community-level in addition to individual-level approaches to reducing obesity (Brownell et al. 2010). A primary driver of the community-level approach is based on the notion that the "food environment" contributes to individual weight status and chronic conditions. Government interventions such as the Healthy Food Financing Initiative have been implemented to increase the number and types of stores carrying healthy food (Holzman 2010; Karpyn et al. 2010). Although some observational cross-sectional studies have supported this approach, the evidence for improved weight health outcomes is mixed (Feng et al. 2010; McKinnon et al. 2009).

First, it is unlikely that a supermarket has the same influence on weight for everyone. Jones-Smith et al. found that the relationship between healthy food environment and body mass index (BMI) among adults with diabetes differed by race and income in direction and magnitude (Jones-Smith et al. 2013). Additionally, the contemporaneous relationship between food environment and BMI may suffer from selection bias because people are not randomly assigned to their neighborhood (Oakes 2004). In other words, an individual's food environment may

reflect diet, health status, and exercise preferences and voluntary or involuntary neighborhood selection (Feng et al. 2010).

The Moving to Opportunity (MTO) study (Ludwig et al. 2011), which randomly assigned housing vouchers to low-income families to move to wealthier neighborhoods, found a significantly lower percentage of severe obesity ten-years later among the treatment group. Very few observational studies of the effect of the built environment on BMI have addressed the impact of endogeneity by using more sophisticated design or analysis techniques (Sturm and Datar 2005). One study found that the cross-sectional relationship between neighborhood food environment and youth body mass index disappeared when examined longitudinally (Shier, An and Sturm 2012).

The ambiguity surrounding the relationship between the food environment and obesity has also resulted from measurement issues. Despite the growing number of studies and the sophistication of spatial analysis techniques used, there has been little convergence in the instruments used to measure the food environment (Shier et al. 2012). A measure of good food environment was designed for this research to capture the popularly selected instruments of density and proximity (Burgoine, Alvanides and Lake 2013).

This study tests the longitudinal association of changes in the food environment on changes in BMI among diabetic adults over a five-year period. The hypothesis of this study as the food environment becomes more health promoting, as measured by the residential proximity to healthy food vendors, the BMI of adults with diabetes will decrease.

Specifically, this study contributes to advancing knowledge of the role of food environment on obesity by exposing two types of bias: 1) omitted variable bias, and 2) residential self-selection bias. The individual fixed-effects approach targets the within individual

change in BMI and by design controls for time-invariant person-specific variables (i.e., preferences for healthy food, tendency to cook, etc.). The variables that are left out in standard ordinary least squares (OLS) estimates are expected to induce positive bias on the coefficient on food environment. Specifically, the estimate of the effect of food environment on BMI will appear larger in OLS estimates if other variables that might be positively correlated with food environment are left out.

To understand residential selection bias, separate models were run for the 83% of the sample who did not move during the observation period, and the 17% who relocated. Among those who remained in a single location (non-movers), the individual fixed effect model estimates the effect of changes in the residential neighborhood changes such as store openings and closings, which are presumably unexpected and thus can be assumed to be largely exogenous to residential decisions.

Estimating effects among movers is potentially more problematic due to possible endogenous migration related to either neighborhood change or other unobserved life changes with potential direct effects on BMI. To better understand the extent to which such processes may bias estimates, two approaches were taken that exploit different portions of neighborhood variation. First, we use an approach analogous to an "intent-to-treat (ITT)" model, examining only the variation over time in the movers' original neighborhood, and ignoring the characteristics of the new (endogenously chosen) neighborhood. We can think of moving to a new address as making an individual "non-compliant" with the treatment. While this ITT interpretation abstracts from the more endogenous portions of the neighborhood, it may attenuate estimates relative to models that examine characteristics of individuals' actual neighborhood at any given time.

Second, models were estimated for the movers that examine the association with the characteristics of the actual neighborhood in which the individual lived in the past year. This more accurately captures the actual neighborhood characteristics experienced, but introduces more chances of selection bias from unobserved determinants of neighborhood choice. If this latter model is significantly different from the model among non-movers, this would suggest selection bias in models that include movers, which would also raise the possibility of similar concerns in standard cross-sectional estimates that ignore neighborhood choice.

Methods

Study Design and Subjects

Individual-level data came from the Kaiser Permanente Northern California (KPNC) Diabetes Registry; a prospective cohort of insured patients established in 1993 to measure prevalence and incidence of diabetes and to understand factors associated with disease progression. KPNC is an integrated healthcare system with more than 3 million members. Clinical data used in this study was collected from 2007 to 2011 from all patient visits to KPNC medical centers. Patients with type 1 (3,616) or unknown type (11,085) diabetes, pregnancy within the study period and 1-year prior (2,327), cancer within the study period and 1-year prior (23,871), and histories of lower extremity amputation were excluded (3,922).

Patient residence was geocoded to the centroid of the census block according to the patient address data from February of each year. Patients with at least one valid geocodable address record from 2006 to 2010 were retained for analysis. Of patients with a census block identifier, 75% had at least one measure of BMI in all 5 years and 18% had at least one measure

in 4 of 5 years. Patients lived in 19 counties that contained or had KPNC facilities near county borders.

Main Outcome

The main outcome, body mass index (BMI, kg/m^2), was calculated at each patient visit and was grand mean centered.(Kromrey and Foster-Johnson 1998) Over the five-year period, the interquartile range (IQR) for number of BMI measurements was 10-27 (median of 17) and within a given year, the IQR was 2-8 (median of 4). We created two variables to account for BMI measurement timing and quality. First, to adjust for seasonal variation in BMI, 12 indicator variables captured the month of measurement. Second, an indicator for higher measurement precision (1= precise, 0 = midpoint of range-based BMI).

Measurement of the local food environment

A measure of the neighborhood food environment was constructed to capture both density and proximity dimensions. The software, ArcGIS (ArcGIS, version 10, Redlands, CA, Environmental Systems Research Institute), and the business database InfoUSA, as acquired from Environmental Systems Research Institute, were used to geocode food vendor addresses and identify initial categories (supermarkets, produce vendors,) for the measures of the food environment. Standardized industrial codes (SIC) were used to refine vendor classifications based on store name and sales volume ≥$2 million in sales, and ≥2,500 square feet for supermarkets.

The primary exposure of interest, kernel density surface of "good" food (*goodfood*), was developed for each patient geocodable address from 2006-2010. As in previous studies(Jones-

Smith et al. 2013; Rundle et al. 2009; Spence et al. 2009) we included supermarkets and produce vendors in a single measure to avoid multicollinearity among multiple measures of stores selling nutritious items such fruits and vegetables. The kernel density score of good food can be theoretically considered a composite food environment measure of the density and proximity of vendors selling healthy foods. A fixed-bandwidth quartic kernel density estimation was used to create a smooth, continuous surface where each supermarket and produce vendor was assigned a density value without respect to administrative boundaries. The surface is highest above the vendor location and diminishes as distance increases, reaching zero one mile in any direction from the vendor location (Silverman 1998).

Covariates

Individual time-varying covariates were collected from 2007 to 2011. Demographic variables included age (age years, mean centered). An indicator for Medicaid (1= Medicaid, 0=not on Medicaid) was constructed. As all members of this cohort have health insurance, Medicaid was included to control for shocks in individual income that might influence an individual's interaction with the food environment and also body mass index. Health variables were included to control for variation in health status and healthcare. Covariates include a continuous Charlson comorbidity index score(Charlson et al. 1987), a weighted index taking into account the number and the seriousness of comorbid conditions. Medication indicators for use of insulin, oral diabetes medications and certain psychiatric medications that are clinically associated with weight change.

Time-varying census block group covariates from the American Community Survey (ACS) 2006-2010 were included as additional control variables. Three year pooled data provided

estimates at the block group level, which is the smallest geographic unit for which the Census Bureau publishes sample data. The ACS variables included continuous variables: population density (population per square mile), proportion of White (the number of white people divided by total population per block group), proportion of Black (the number of black people divided by total population per block group), and proportion of population under the federal poverty line (FPL, the number of people living under the federal poverty line divided by total population per block group).

The only individual time-invariant covariate was an indicator for *movers*. *Movers* (1=movers, 0=non-movers) are those who reported at least two valid different addresses between 2006-2010.

Statistical Methods

In order to understand how change in BMI is related to the change in food environment, we used modeled the within-person relationship between changes in food environment and 1-year lagged BMI using year and individual fixed effects as follows:

$$BMI_{it_s} = \beta_{oi} + \beta_1 Envir_{it-1} + \beta_2 M_{it_s} + \beta_3 X_{it} + \beta_4 Z_{it-1} + \beta_5 yearD_t + \varepsilon_{it}$$

where BMI_{it_s} is a measure of the BMI of individual *i* in occasion *s* nested in year *t*; β_{oi} is the individual intercept that is swept out in the fixed effects model; $Envir_{it-1}$ is a measure of the food environment (*goodfood*) for individual *i* in year *t-1*; M_{it_s} is a vector of measurement characteristics of *BMI* of individual *i* in occasion *s* nested in year *t* (*quality, month*); X_{it} is a vector of individual characteristics in year *t* (*age, medicaid, charlson, insulin, oral, gain, loss*); Z_{it-1} is a vector of lagged area level controls of individual *i* in year *t-1* (*population density, proportion white, proportion black, proportion poor*); *yearD* includes indicators for year *t*; and ε_{it} is the time and individual specific error term with a mean 0 and variance θ.

Several approaches were employed to better understand whether the association between changes in food environment and BMI was affected by selection bias. First, an intent-to-treat

(ITT) approach was estimated using the 2006 address for all cohort members to model the association between 1-year lagged BMI and the local food environment score as the neighborhood changed around members between 2006 and 2010. The same models were also fit using the actual address (non-ITT) at which the person lived each year prior to the BMI measurements.

For people who moved, the non-ITT approach captures the relationship between prior year's food environment and BMI along with residential self-selection bias, while the ITT approach estimates the effect of the food environment "offered" to individuals Those who moved can be considered to refuse the "offer". Individuals who moved into a neighborhood with a similarly dynamic food environment as their 2006 neighborhood are expected to have similar estimates for the ITT and non-ITT approach, in the absence of selection bias. Those who move into a better food environment are hypothesized to have a decrease in BMI. The process of moving is expected to offset this relationship through an increase in stress or a decline in time for self-care.

All models were weighted using the inverse of the number of BMI measurements over the entire period so as to generalize back to the individuals in this population. Due to the theoretically ambiguous prediction of appropriate lag length in which food environment can change BMI, alternate lag periods were considered. As results might be sensitive to timing, the models were re-estimated using a lag of 2-years (i.e. good food environment kernel density two years prior to BMI measures) and a contemporaneous specification.

Finally, standard models using ordinary least squares (OLS) and individual random-effects were estimated to compare with the *a priori* preferred fixed effects specifications. A Hausman test (Hausman 1978) was used to compare model specifications. It was hypothesized

that the fixed-effects models would attenuate the relationship between food environment and BMI observed in the OLS and random-effect models if omitted variable bias was reduced.

All analyses were conducted using STATA version 12.

Results

There were 194,652 individuals in this study that meet the inclusion criteria above and had at least one measure of BMI from 2007 to 2011. 3.7% (N = 7,218) had at least one missing time-varying parameter. 17.3% of the individuals moved at least once during the study period and on average, 7.2% of individuals moved each year.

Table 1 reports means and standard deviations for the time-varying variables. The mean BMI was 31.83, the within-person standard deviation was 1.69, and the between-person standard deviation was 7.04. The measure of food environment kernel density ranged from 0 to 5.70 with a mean of 0.34, a within-person standard deviation of 0.01, and between-person standard deviation of 0.45.

Results from the intent-to-treat (ITT) models 1-6 are shown in Table 2. These models assumed no relocation from the 2006 address and represent the effect on BMI of the food environment "offered" to individuals. In Model 1, the unadjusted association between kernel density of good food environment (*goodfood*) was inversely related to BMI. For each one standard deviation increase in *goodfood* (0.46) in the prior year, BMI decreased by 0.116 ($p<0.001$). Based on the average person in this sample with a BMI of 31, a height of 5'9" and weight of 210 lbs, this change is roughly equivalent to losing one pound. Model 2, adjusted for BMI measurement type, month, and year indicators, shows an attenuated association of good

food environment with BMI of -0.091. The addition of individual time-varying covariates in Model 3 and block-group covariates in Model 4 resulted in only small changes to the estimates.

Model 5 was restricted to non-movers and Model 6 restricted to movers, adjusting for the same covariates as Model 4. Among the non-movers, each one-unit increase in good food environment was associated with a decrease of 0.094 ($p<0.05$) BMI, translating into a loss of approximately one pound for an average cohort member, a small but statistically significant association in the anticipated direction. For those who moved at least once during the five-year study period, we found no statistically significant effect on BMI of change in the good food environment in the ITT model.

Results from the non-ITT models 7-12 using the actual observed food environment as the exposure of interest are shown in Table 3. Results of Model 7 show that, as in the equivalent ITT model, the unadjusted association of kernel density of good food environment was inversely related to BMI. Each one-unit increase in kernel density of good food environment in the prior year resulted in a decrease in BMI of 0.076 ($p<0.001$). In Model 8, which included BMI measurement type, and month and year indicators, the association between kernel density of good food environment and BMI became insignificant, and remained so in Models 9 and 10, when the individual time-varying covariates and the block-group controls were included, respectively.

Model 11 restricted the analysis to non-movers and Model 12 to movers only, and both included the same covariates as Model 10. Estimates from Model 11 can be thought of as representing the effect of the opening and closing of supermarkets and produce vendors in a residential area. For every one-unit increase in kernel density of good food environment there was a decrease of 0.084 ($p<0.05$) in BMI. These results were similar to the results of Model 5 in

a slightly smaller cohort. For movers, there was no relationship between kernel density of good food environment in year prior and change in BMI (Model 12).

We found no evidence for different effects of food environment on BMI using alternate lag structures (2 year lag or contemporaneous.

Results from the OLS and random-effect models with full sets of covariates had larger magnitudes of association; the coefficient on *goodfood* was -0.208 in the OLS model and -0.120 in the random-effects model (not shown), while there was no evidence of a statistically significant relationship of *goodfood* and BMI in the fully adjusted fixed effects model. The Hausman test indicated that the coefficient estimates from the random effects model are biased and inconsistent and thus that the fixed effects model is preferred.

Discussion

A clinically modest yet statistically significant association was found between improvements in the food environment and weight loss among adults with diabetes whose residential address remained unchanged over five years. Using data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, researchers (Boone-Heinonen et al. 2013) found similarly modest reductions in BMI with a large increase supermarkets density, however the differential association of residential mobility was not tested in that study. Our finding is arguably clinically insubstantial given that opening a supermarket is only expected to lead to a one-pound weight loss in a representative individual from the cohort. Given the scale of the obesity epidemic, policymakers should consider the cost-effectiveness of initiatives to incentivize the opening of supermarkets and produce vendors.

There was no evidence to support that there was an association between improvement in the food environment and weight status among adults who moved at least once over the five-year observation period. This could be explained by a selection bias due to healthier individuals moving to neighborhoods that reflect their tastes for healthier environments. Additionally, opening supermarkets and produce vendors might change proximate housing prices and inconsequentially lead to an attrition of lower socio-economic individuals. Pope found that opening a Wal-Mart store increased local housing prices (Pope and Pope 2012). More research is needed to understand the factors causing residential mobility and the downstream relationship with obesity.

Among the limitations of this study, the study findings may only be generalizable to insured adults with diabetes who live in Northern California. Further research should be conducted to determine if this relationship holds in other geographic areas. As this study only includes adults with Type 2 diabetes, the effect might be different for children or those without diabetes. Also, there was no data on those who moved outside the Kaiser Permanente Northern California catchment area. The physical distance of supermarkets and produce vendors was taken into account in this study, although other relational factors such as quality and prices, which are expected to play a role in a consumer's decision to switch stores, were not captured. More research is needed to understand how these influence purchasing decisions.

The results of this study call for the re-examination of food policies that encourage opening of supermarkets in "food deserts" as a means to reduce obesity in the neighboring communities. More thoughtful interventions that consider the cost per unit of effectiveness are needed in containing the obesity epidemic.

Acknowledgements

Research reported in this publication was supported by the Neighborhood Effects on Weight Change and Diabetes Risk Factors grant (R01 DK080744), Ethnic Disparities in Diabetes Complications grant (R01 DK065664), and the Educational Disparities in Diabetes Complications grant (R01 HD46113) from the National Institute for Health (NIH).

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Tables

Table 1: Mean and standard deviation of within and between individual

Variable		Mean	SD
Body Mass Index (BMI)	Overall	31.832	7.257
	Between		7.041
	Within		1.687
Good food environment, kernel density	Overall	0.341	0.460
	Between		0.450
	Within		0.010
Age, years	Overall	63.434	13.070
	Between		13.230
	Within		1.472
Medicaid, yes/no	Overall	0.021	0.145
	Between		0.123
	Within		0.044
Charlson Index,	Overall	2.170	1.918
	Between		1.454
	Within		0.993
Insulin, yes/no	Overall	0.201	0.401
	Between		0.318
	Within		0.183
Oral medication, yes/no	Overall	0.418	0.493
	Between		0.418
	Within		0.249
Medication causing weight gain (yes/no)	Overall	0.068	0.252
	Between		0.181
	Within		0.135
Medication causing weight loss (yes/no)	Overall	0.085	0.279
	Between		0.209
	Within		0.147
Quality of BMI measurement	Overall	0.968	0.176
	Between		0.114
	Within		0.160
Month of BMI measurement	Overall	6.411	3.403
	Between		1.540
	Within		3.230
Population density*	Overall	8118.376	8705.752
	Between		8588.290
	Within		1976.850
Proportion White*	Overall	0.585	0.232
	Between		0.229
	Within		0.046
Proportion Black*	Overall	0.075	0.112
	Between		0.109
	Within		0.025
Proportion below FPL*	Overall	0.108	0.106
	Between		0.102
	Within		0.027

*Measured at the block group level

Table 2 -ITT: Change in body mass index (BMI), individual fixed effects models

Variables	(1)	(2)[i]	(3)	(4)	(5) nonmover	(6) mover
Intercept	31.586 (0.008)***	31.369 (0.015)***	36.583 (0.101)***	36.734 (0.178)***	36.729 (0.198)***	36.754 (0.422)***
goodfood (prior year)	-0.253 (0.031)***	-0.091 (0.032)***	-0.097 (0.032)***	-0.087 (0.031)***	-0.094 (0.035)**	-0.062 (0.067)
Age (centered)			-0.124 (0.002)***	-0.124 (0.004)***	-0.124 (0.004)***	-0.126 (0.011)***
Medicaid			0.164 (0.080)**	0.075 (0.120)	0.013 (0.124)	0.159 (0.227)
Charlson index			-0.032 (0.003)***	-0.040 (0.004)***	-0.041 (0.004) ***	-0.037 (0.010)***
On insulin			0.468 (0.018)***	0.278 (0.024)***	0.272 (0.027)***	0.301 (0.052)***
On oral			0.321 (0.012)***	0.250 (0.016)***	0.260 (0.018)***	0.218 (0.035)***
On weight gain			-0.038 (0.025)	-0.044 (0.033)	-0.028 (0.036)	-0.090 (0.078)
On weight loss			-0.096 (0.022)***	-0.092 (0.027)***	-0.087 (0.030)***	-0.111 (0.067)*
Population density				0.00001 (0.0001)	0.00001 (0.0001)*	0.00001 (0.0001)
Proportion white				-0.010 (0.066)	0.083 (0.084)	-0.146 (0.107)
Proportion black				0.026 (0.040)	0.052 (0.053)	-0.013 (0.061)
Proportion under FPL				0.076 (0.081)	0.090 (0.106)	0.065 (0.124)
Individuals	179,378	179,378	177,244	171,120	139,784	31,336
Observations	2,380,290	2,380,290	2,344,061	1,438,136	1,172,446	265,690

***p<0.01, **p<0.05, *p < 0.1

¥Robust Standard errors are in parenthesis

(i) Models 2-6 adjusted for month and year indicators and measurement method (not shown)

Table 3 - Non-ITT: Change in body mass index (BMI), individual fixed effects models

	(7)	(8)[i]	(9)	(10)	(11)	(12)
Variables					nonmover	mover
Intercept	31.530 (0.021)***	31.369 (0.015)***	36.583 (0.101)***	36.344 (0.132)***	36.342 (0.305)***	35.811 (0.292)***
*goodfood** (prior year)	-0.076 (0.021)***	-0.014 (0.021)	-0.016 (0.022)	-0.015 (0.016)	-0.084 (0.034)**	0.008 (0.028)
Age (centered)			-0.122 (0.002)***	-0.117 (0.003)***	-0.120 (0.002)***	-0.105 (0.007)***
Medicad			0.223 (0.076)***	0.174 (0.094)*	0.120 (0.104)	0.252 (0.175)
Charlson index			-0.031 (0.003)***	-0.037 (0.003)***	-0.039 (0.004) ***	-0.033 (0.008)***
On insulin			0.455 (0.018)***	0.354 (0.206)***	0.350 (0.024)***	0.365 (0.043)***
On oral			0.323 (0.012)***	0.284 (0.013)***	0.299 (0.015)***	0.233 (0.028)***
On weight gain			-0.050 (0.024)**	-0.060 (0.028)**	-0.049 (0.031)	-0.097 (0.065)
On weight loss			-0.086 (0.021)***	-0.091 (0.020)***	-0.092 (0.025)***	-0.087 (0.055)
Population density				0.00001 (0.0001)	0.00001 (0.0001)	0.00001 (0.0001)
Proportion white				0.093 (0.064)	0.281 (0.417)	0.084 (0.065)
Proportion black				0.014 (0.124)	0.858 (0.695)	-0.018 (0.125)
Proportion under FPL				0.046 (0.108)	-0.060 (0.617)	0.050 (0.110)
Individuals	194,652	194,652	191,621	187,144	152,036	35,108
Observations	2,474,790	2,474,790	2,429,800	1,909,969	1,555,353	354,616

***p<0.01, **p<0.05, *p < 0.1

¥Robust Standard errors are in parenthesis

(i) Not shown: Models 8-12 adjusted for month and year dummies, and measurement method.