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Original Contribution

Food Environment and Weight Change: Does Residential Mobility Matter?

The Diabetes Study of Northern California (DISTANCE)

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Associations between neighborhood food environment and adult body mass index (BMI; weight (kg)/height $(m)^2$) derived using cross-sectional or longitudinal random-effects models may be biased due to unmeasured confounding and measurement and methodological limitations. In this study, we assessed the within-individual association between change in food environment from 2006 to 2011 and change in BMI among adults with type 2 diabetes using clinical data from the Kaiser Permanente Diabetes Registry collected from 2007 to 2011. Healthy food environment was measured using the kernel density of healthful food venues. Fixed-effects models with a 1-year-lagged BMI were estimated. Separate models were fitted for persons who moved and those who did not. Sensitivity analysis using different lag times and kernel density bandwidths were tested to establish the consistency of findings. On average, patients lost 1 pound (0.45 kg) for each standard-deviation improvement in their food environment. This relationship held for persons who remained in the same location throughout the 5-year study period but not among persons who moved. Proximity to food venues that promote nutritious foods alone may not translate into clinically meaningful diet-related health changes. Community-level policies for improving the food environment need multifaceted strategies to invoke clinically meaningful change in BMI among adult patients with diabetes.

built environment; diabetes; econometrics; food environment; obesity

Abbreviations: BMI, body mass index; ITT, intention to treat; OLS, ordinary least squares.

Over 22 million Americans—7% of the US population—were diagnosed with diabetes in 2012, at a total cost of $245 billion, including direct medical costs and lost productivity (1). Obesity is a major risk factor for development of type 2 diabetes; 60%–90% of cases of type 2 diabetes mellitus are associated with obesity or weight gain (2). Weight management for control of blood glucose levels and reduction of cardiovascular disease risk based on a personalized diet and exercise plan is an important goal for diabetic patients (3).

Recently, policy-makers have focused on community-level approaches to reducing obesity in addition to individual-level approaches (4). A primary driver of the community-level approach is the notion that the "food environment" contributes to individual weight status and chronic disease conditions. Government interventions such as the Healthy Food Financing Initiative have been implemented to increase the numbers and types of stores carrying healthy food (5, 6). Supermarkets and produce vendors have been identified as 2 types of food venues that may exert a positive influence on weight change (7).

Although there is some evidence that a healthy food environment and low body mass index (BMI) coexist, these associations may suffer from selection bias because people are not randomly assigned to their neighborhoods (8). In other words, a person's food environment may reflect diet, health status, and exercise preferences and voluntary or involuntary neighborhood selection (9). Because it is costly and ethically challenging to randomize individuals to different neighborhoods, few studies have been able to avoid selection bias by

design. Researchers from the Moving to Opportunity Study (10), in which housing vouchers were randomly assigned to low-income families to move to wealthier neighborhoods, found a significantly lower percentage of severe obesity in the intervention group 10 years later. Very few observational studies of the association between the built environment and BMI have addressed the endogeneity issue (in this case, the idea that neighborhoods are not randomly assigned) by using more sophisticated design or analysis techniques (11).

Although some observational cross-sectional studies have supported an association between the presence of supermarkets and lower BMI, including differential effects by race/ethnicity (12), the evidence for improved weight and health outcomes is mixed (9, 13–17). Shier et al. (18) found that the cross-sectional relationship between neighborhood food environment and youth body mass index disappeared when the association was examined longitudinally.

In this study, the Diabetes Study of Northern California (DISTANCE), we tested the longitudinal association between changes in the food environment and changes in BMI among diabetic adults over a 5-year period. A measure of good food environment was designed for this research in order to capture both density and proximity (19). We hypothesized that as the food environment became more health-promoting, as measured by residential proximity to healthy food vendors, the BMI of adults with diabetes would decrease.

Specifically, this study contributes to advancing knowledge on the role of the food environment in obesity by exposing 2 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.). This approach was taken because the variables that are omitted in standard ordinary least squares (OLS) estimates were expected to induce positive bias in the coefficient on food environment.

Residential selection bias was the second source of bias addressed in this study. Separate models were fitted for persons who moved and persons who did not move during the observation period. Among those who remained in a single location, the individual fixed-effects model estimated the association between BMI and changes in the residential neighborhood, such as store openings or closings, which are presumably unexpected and thus can be assumed to be largely exogenous to residential decisions. Estimating associations 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.

METHODS

Study design and subjects

Individual-level data were obtained from the Kaiser Permanente Northern California Diabetes Registry, a prospectively assembled cohort of insured patients established in 1993 to measure the prevalence and incidence of diabetes and to understand factors associated with disease progression. Clinical data used in this study were collected during the period 2007–2011 from ambulatory patient visits to Kaiser Permanente Northern California clinics (n = 369,222). Patients with type 1 (n = 3,616) or unknown-type (n = 11,085) diabetes and patients who were pregnant at some point within the study period or 1 year prior (n = 2,327), had cancer within the study period or 1 year prior (n = 23,871), or had a history of lower-extremity amputation (n = 3,922) were excluded. All study protocols were approved by the Kaiser Permanente Division of Research and the Committee for Protection of Human Subjects of the University of California, Berkeley.

Due to patient privacy concerns, patient residence was geocoded to the census block centroid according to the patient address data from February of each year. Patients with at least 1 valid geocodable address during 2006–2010 in the 19 counties served by Kaiser Permanente Northern California were retained for analysis. Of patients with a census block identifier, 75% had at least 1 measurement of BMI in all 5 years, 18% had at least 1 BMI measurement in 4 of the 5 years, and fewer than 1% had only 1 BMI measurement during all 5 years.

Main outcome

The main outcome, BMI (weight (kg)/height (m)^2), was calculated at each patient visit and was grand-mean-centered (20). The interquartile range for number of BMI measurements was 10–27 (median, 17) over the 5-year study period and 2–8 (median, 4) per year. Two variables were created to account for the timing and quality of BMI measurement. First, indicator variables for each month of BMI measurement were generated to adjust for seasonal variation. Second, an indicator for measurement precision (1 = calculated from exact visit weight and height, 0 = midpoint of range-based BMI). One-year-lagged BMI was used as our main specification, since there is no evidence regarding the average length of time for which a new store could influence a change in shopping behaviors (21).

Measurement of the local food environment

A measure of food environment, kernel density surface of good food, was constructed because it captures both density and proximity dimensions (22). First, we used the software ArcGIS (ESRI, Redlands, California) and the business database InfoUSA (Infogroup, Papillion, Nebraska), as acquired from ESRI, to geocode the addresses of supermarkets and produce vendors from 2006 to 2010 in our study area. We identified 380 produce vendors using code 543101 of the North American Industry Classification System (23). We identified 1,096 supermarkets using codes 541105, 541101, 543102, and 543101 and included them if they had a sales volume of at least $2 million and a size of at least 2,500 square feet (232 m^2).

As in previous studies, 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 (16, 24). After identification of all supermarkets and produce vendors in 19 counties of Northern California, we used a quartic (biweight) kernel function to create a continuous kernel density surface with a 1-mile (1.6-km) radius, where the density was greatest at the

geocoded point for each vendor and decreased to zero at the edge of the radius. We selected the 1-mile fixed bandwidth as our primary measure because in this sample the mean distance to the closest supermarket was 1.3 miles (2.1 km), with a standard deviation of 1.2 miles (1.9 km). Two alternate measures with bandwidths of 2.1 miles (3.8 km) and 4.5 miles (7.2 km) were also created. A bandwidth of 2.1 miles was selected after applying Silverman's rule to determine the optimal smoothed surface (25). The bandwidth of 4.5 miles was selected because a majority of Americans travel no farther than 4.5 miles to reach their primary supermarket (26). Finally, we assigned the value of the surface of the patient's census block (smallest unit available) centroid for each year from 2006 to 2010.

Covariates

Data on individual time-varying covariates were collected from 2007 to 2011. Demographic variables included age (in years, mean-centered). An indicator for enrollment in the Medicaid program (1 = Medicaid, 0 = not on Medicaid) was constructed. Because all members of this cohort had health insurance, enrollment in Medicaid was included to control for shocks in individual income, which might affect a person's interaction with the food environment and also BMI. Health variables were included to control for variation in health status and receipt of health care. Covariates included a continuous score (possible range, 0–33) for the Charlson comorbidity index (27), a weighted index taking into account the number and seriousness of comorbid conditions. Medication indicators included use of insulin, oral diabetes medications, and certain psychiatric medications that are associated with weight change.

Time-varying census block group covariates from the American Community Survey (28) were included as additional control variables and linked to patient block group. In the absence of more precise measures, we used 5-year pooled estimates as a proxy for the middle year (i.e., 2005–2009 for 2007 and 2006–2010 for 2008). These measures included continuous variables: population density (population per square mile), proportion non-Hispanic white, proportion black, and proportion of households under the federal poverty line (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 residential moves. Movers (1 = movers, 0 = nonmovers) were defined as persons who reported at least 2 different valid addresses between 2006 and 2010.

Statistical methods

First, the mean values and standard deviations of time-varying variables were determined by decomposing the between-person and within-person components.

Standard models using OLS and individual random effects were fitted to compare with the a priori preferred fixed-effects specifications. A Hausman test (29) was used to compare model specifications for consistency. It was hypothesized that the fixed-effects models would attenuate the relationship between food environment and BMI observed in the OLS and random-effects models if omitted variable bias was reduced.

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

$$\mathrm{BMI}_{it_s} = \beta_{oi} + \beta_1 \mathrm{Envir}_{it-1} + \beta_2 \boldsymbol{M}_{it_s} + \beta_3 \boldsymbol{X}_{it} + \beta_4 \boldsymbol{Z}_{it-1} + \beta_5 \mathrm{Year}\, D_t + \varepsilon_{it},$$

where BMI_{it_s} is a measure of the BMI of individual i on 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 for individual i in year $t-1$; $\boldsymbol{M}_{it_s}$ is a vector of measurement characteristics of the BMI of individual i on occasion s nested in year t (quality, month); $\boldsymbol{X}_{it}$ is a vector of individual characteristics in year t (age, Medicaid enrollment, Charlson comorbidity index, and indicators of medication use (insulin, oral medication, weight gain medication, and weight loss medication)); $\boldsymbol{Z}_{it-1}$ is a vector of lagged area-level controls of individual i in year $t-1$ (population density, proportion white, proportion black, proportion poor); Year$D$ includes indicators for year t; and ε_{it} is the time- and individual-specific error term.

Several approaches were employed to better understand whether the association between changes in the food environment and BMI was affected by selection bias. To account for the extent to which residential moves may bias estimates, 2 approaches were taken that exploit different portions of neighborhood variation. First, an approach analogous to that of an "intention-to-treat" (ITT) model was used, examining only the variation over time in the movers' original neighborhood and ignoring the characteristics of the new (endogenously chosen) neighborhood. Moving was hypothesized to be analogous to a person becoming "noncompliant" with the treatment. The ITT approach was implemented 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.

Second, the same models were also fitted 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 the prior year's food environment and BMI along with residential self-selection bias, while the ITT approach estimates the association between BMI and the food environment "offered" to individuals. People who moved can be considered to have refused the "offer." People 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 approaches, in the absence of selection bias.

All models were weighted using the inverse of the number of BMI measurements taken over the entire period so as to generalize back to the individuals in this population. Due to the theoretically ambiguous prediction of the appropriate length of the lag period in which food environment can change BMI, alternate lag periods were considered, and

models were refitted using a lag of 2 years (i.e., good food environment kernel density 2 years prior to BMI measures) and a contemporaneous specification. To understand the change of transitioning from no food environment to any additional food, we recoded our primary exposure variable as a binary indicator that took the value of 0 when it was zero and 1 when it was greater than zero. To assess model sensitivity to kernel density bandwidth, we refitted our models with the 2 alternate measures with larger bandwidths. All analyses were conducted using Stata (StataCorp LP, College Station, Texas), version 12.

RESULTS

There were 194,652 individuals in this study who met the above inclusion criteria and had at least 2 BMI measurements between 2007 and 2011. Approximately 3.7% (n = 7,218) had at least 1 missing time-varying parameter, 17.3% of the participants moved at least once during the study period, and on average 7.2% of participants moved each year.

Table 1 shows mean values 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, interpreted as number of stores per square kilometer, ranged from 0 to 5.70, with a mean of 0.34 (or 0.13 stores per square mile), a within-person standard deviation of 0.01, and a between-person standard deviation of 0.45. Eighty-two percent of participants had a kernel density score greater than zero, and 14% had a baseline score of zero.

The results from the OLS and random-effects models with full sets of covariates had larger magnitudes of association than those from the a priori preferred fixed-effects models; the coefficient on good food environment was −0.208 in the OLS model and −0.120 in the random-effects model (both P's < 0.001; not shown). The null value of the Hausman test, which states that the random effect is not correlated with the other independent variables, was rejected. This means that the random-effects model is biased and inconsistent and the fixed-effects model is preferred.

Results from ITT models 1–6 are shown in Table 2. These models assumed no relocation from the 2006 address, and the results represent the association between BMI and the food environment "offered" to individuals. In model 1, there was a negative association between kernel density of good food environment and BMI. For each 1-unit increase in the prior year, BMI decreased by 0.25 units (P < 0.001). Based on the average person in this sample with a BMI of 31, a height of 5′9″ (175.3 cm), and a weight of 210 pounds (95.3 kg), this change is roughly equivalent to losing 2 pounds (0.9 kg).

Table 1. Time-Varying Characteristics of Participants in a Study of Neighborhood Food Environment and Adult Body Mass Index, Northern California, 2006–2011

Variable	Mean	Standard Deviation			Minimum	Maximum	No. of Individuals	No. of Observations
		Overall	Between-Person	Within-Person				
BMI[a]	31.83	7.26	7.04	1.69	15	134	214,830	2,815,549
Good food environment (kernel density)	0.34	0.46	0.45	0.01	0	5.7	198,030	2,810,220
Age, years	63.43	13.07	13.23	1.47	18	109	214,826	3,212,896
Medicaid enrollment (yes/no)	0.02	0.15	0.12	0.04	0	1	213,113	3,003,353
CCI score[b]	2.17	1.92	1.45	0.99	0	19	214,830	3,212,923
Insulin use (yes/no)	0.20	0.40	0.32	0.18	0	1	214,830	3,212,923
Oral medication use (yes/no)	0.42	0.49	0.42	0.25	0	1	214,830	3,212,923
Medication causing weight gain (yes/no)	0.07	0.25	0.18	0.14	0	1	214,830	3,212,923
Medication causing weight loss (yes/no)	0.09	0.28	0.21	0.15	0	1	214,830	3,212,923
Quality of BMI measurement	0.97	0.18	0.11	0.16	0	1	214,830	2,815,549
Month of BMI measurement	6.41	3.40	1.54	3.23	0	1	214,830	2,815,549
Census block group characteristics[c]								
Population density, persons/square mile[d]	8,118.38	8,705.75	8,588.29	1,976.85	0	227,475	198,206	2,295,612
Proportion white	0.59	0.23	0.23	0.05	0	1	198,206	2,295,612
Proportion black	0.08	0.11	0.11	0.03	0	1	198,206	2,295,612
Proportion below federal poverty line	0.11	0.11	0.10	0.03	0	1	198,206	2,295,612

Abbreviations: BMI, body mass index; CCI, Charlson comorbidity index.
[a] Weight (kg)/height (m)^2.
[b] The possible range of scores in the CCI (27) is 0–33. The range in this data set was 0–19.
[c] Measured at the US Census block group level.
[d] Number of persons per square mile (per 1.6 km^2).

Table 2. Coefficients (β (SE[a])) from Ordinary Least Squares Models of the Influence of Food Environment on Body Mass Index[b] With Individual Fixed Effects (ITT Approach[c]), Northern California, 2006–2011

Variable	Model[d]					
	1	2	3	4	5 (Nonmovers)	6 (Movers)
Intercept	31.59 (0.01)[e]	31.37 (0.02)[e]	36.58 (0.10)[e]	36.73 (0.18)[e]	36.73 (0.2)[e]	36.75 (0.42)[e]
Food environment in prior year[f]	−0.25 (0.03)[e]	−0.09 (0.03)[e]	−0.097 (0.03)[e]	−0.09 (0.03)[e]	−0.09 (0.04)[g]	−0.06 (0.07)
Age, years (centered)			−0.12 (0.00)[e]	−0.12 (0.00)[e]	−0.12 (0.00)[e]	−0.13 (0.01)[e]
Medicaid enrollment			0.16 (0.08)[g]	0.08 (0.12)	0.01 (0.12)	0.16 (0.23)
Charlson comorbidity index			−0.03 (0.00)[e]	−0.04 (0.00)[e]	−0.04 (0.00)[e]	−0.04 (0.01)[e]
On insulin[h]			0.47 (0.02)[e]	0.28 (0.02)[e]	0.27 (0.03)[e]	0.30 (0.05)[e]
On oral medication[h]			0.32 (0.01)[e]	0.25 (0.02)[e]	0.26 (0.02)[e]	0.22 (0.04)[e]
On weight gain[h]			−0.04 (0.03)	−0.04 (0.03)	−0.03 (0.04)	−0.09 (0.08)
On weight loss[h]			−0.1 (0.02)[e]	−0.09 (0.03)[e]	−0.09 (0.03)[e]	−0.11 (0.07)[i]
Population density, persons/square mile[j]				0.01 (0.00)	0.00 (0.01)[i]	0.00 (0.01)
Proportion white				−0.01 (0.07)	0.08 (0.08)	−0.15 (0.11)
Proportion black				0.03 (0.04)	0.05 (0.05)	−0.01 (0.06)
Proportion below federal poverty line				0.08 (0.08)	0.09 (0.11)	0.07 (0.12)
No. of individuals	179,378	179,378	177,244	171,120	139,784	31,336
No. of observations	2,380,290	2,380,290	2,344,061	1,438,136	1,172,446	265,690

Abbreviations: ITT, intention to treat; SE, standard error.
[a] Robust SE clustered on the individual.
[b] Weight (kg)/height (m)^2.
[c] With the ITT approach, participants' residences were modeled using their 2006 address, regardless of whether they moved.
[d] Models 2–6 adjusted for month and year indicators and measurement method (not shown).
[e] $P < 0.01$.
[f] Kernel density surface of good food ($t - 1$).
[g] $P < 0.05$.
[h] Indicators for use of medication (insulin, oral medication, and medications that cause weight gain or weight loss).
[i] $P < 0.1$.
[j] Coefficient was multiplied by 1,000 for display.

Model 2, which adjusted for BMI measurement type, month, and year indicators, showed an attenuated association of good food environment with a BMI loss of 0.09 units ($P < 0.001$). 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 nonmovers and model 6 was restricted to movers, adjusting for the same covariates as in model 4. Among the nonmovers, each 1-unit increase in good food environment was associated with a BMI decrease of 0.094 units ($P < 0.05$), translating into a loss of approximately 1 pound (0.45 kg), a small but statistically significant association in the anticipated direction. For persons who moved at least once during the 5-year study period, there was no statistically significant association between change in good food environment and BMI in the ITT model.

Results from the non-ITT models (models 7–12) using the actual observed food environment as the exposure of interest are shown in Table 3. Results from model 7 show that, as in the equivalent ITT model, there was a negative association between kernel density of good food environment and BMI. Each 1-unit increase in kernel density of good food environment in the prior year resulted in a decrease in BMI of 0.076 units ($P < 0.001$). 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 it 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 nonmovers and model 12 to movers only; both included the same covariates as model 10. Estimates from model 11 can be thought of as representing the change in the density of supermarkets and produce vendors in a residential area. For every 1-unit increase in kernel density of good food environment, there was a decrease in BMI of 0.084 units ($P < 0.05$). These results were similar to the results from model 5 in a slightly smaller cohort. For movers, there was no relationship between kernel density of good food environment in the prior year and change in BMI (model 12).

There was no evidence for different associations between the food environment and BMI using alternate lag structures. The results from substituting the binary exposure variable were nearly identical to the main results (not shown). Our estimates were sensitive to the bandwidth

Table 3. Coefficients (β (SE[a])) from Ordinary Least Squares Models of the Influence of Food Environment on Body Mass Index[b] With Individual Fixed Effects (Non-ITT Approach[c]), Northern California, 2006–2011

Variable	Model[d]					
	7	8	9	10	11 (Nonmovers)	12 (Movers)
Intercept	31.53 (0.02)[e]	31.37 (0.012)[e]	36.58 (0.10)[e]	36.34 (0.13)[e]	36.34 (0.31)[e]	35.81 (0.29)[e]
Food environment in prior year[f]	−0.08 (0.02)[e]	−0.01 (0.02)	−0.02 (0.02)	−0.02 (0.02)	−0.08 (0.03)[g]	0.01 (0.03)
Age, years (centered)			−0.12 (0.00)[e]	−0.12 (0.00)[e]	−0.12 (0.00)[e]	−0.11 (0.01)[e]
Medicaid enrollment			0.22 (0.08)[e]	0.17 (0.09)[h]	0.12 (0.10)	0.25 (0.18)
Charlson comorbidity index			−0.03 (0.00)[e]	−0.04 (0.00)[e]	−0.04 (0.00)[e]	−0.033 (0.008)[e]
On insulin[i]			0.46 (0.02)[e]	0.35 (0.21)[e]	0.35 (0.02)[e]	0.37 (0.04)[e]
On oral medication[i]			0.32 (0.01)[e]	0.28 (0.01)[e]	0.3 (0.02)[e]	0.23 (0.03)[e]
On weight gain[i]			−0.05 (0.02)[g]	−0.06 (0.03)[g]	−0.05 (0.03)	−0.10 (0.07)
On weight loss[i]			−0.09 (0.02)[e]	−0.09 (0.02)[e]	−0.09 (0.03)[e]	−0.09 (0.06)
Population density, persons/square mile[j]				0.01 (0.00)	0.01 (0.00)	0.01 (0.00)
Proportion white				0.09 (0.06)	0.28 (0.42)	0.08 (0.07)
Proportion black				0.01 (0.12)	0.86 (0.70)	−0.02 (0.13)
Proportion below federal poverty line				0.05 (0.11)	−0.06 (0.62)	0.05 (0.11)
No. of individuals	194,652	194,652	191,621	187,144	152,036	35,108
No. of observations	2,474,790	2,474,790	2,429,800	1,909,969	1,555,353	354,616

Abbreviations: ITT, intention to treat; SE, standard error.
[a] Robust SE clustered on the individual.
[b] Weight (kg)/height (m)^2.
[c] With the non-ITT approach, participants' residences were modeled using their actual address.
[d] Models 8–12 adjusted for month and year indicators and measurement method (not shown).
[e] $P < 0.01$.
[f] Kernel density surface of good food ($t - 1$).
[g] $P < 0.05$.
[h] $P < 0.1$.
[i] Indicators for use of medication (insulin, oral medication, and medications that cause weight gain or weight loss).
[j] Coefficient was multiplied by 1,000 for display.

selected for the data; there was no association between kernel density of good food environment and BMI for any model with a 2.1-mile (3.4-km) or 4.5-mile (7.2-km) bandwidth (Table 4).

DISCUSSION

A clinically modest yet statistically significant association was found between improvements in the food environment proximate to one's residence and weight loss among adults with diabetes whose residential addresses remained unchanged over a 5-year period. Using data from the Coronary Artery Risk Development in Young Adults (CARDIA) Study, Boone-Heinonen et al. (30) found similarly modest reductions in BMI with a large increase in supermarket density; however, differences by residential mobility were not tested.

Like prior studies that have shown that the relationship between weight change and the food environment varies based on measurement (18), our results were not robust to the size of the bandwidth of the kernel of our exposure. The null observed association of weight change with bandwidths wider than 1 mile (1.6 km) could be due to the food environment's having little influence beyond a distance of 1 mile, or it could be an artifact of oversmoothing.

There was no evidence to support the possibility of an association between improvement in the food environment and weight status among adults who moved at least once during the 5-year observation period. This could be explained by a selection bias due to healthier people's moving to neighborhoods that reflect their tastes for healthier environments. Additionally, openings of supermarkets and produce vendors might change proximate housing prices and inadvertently lead to attrition of persons of lower socioeconomic status. Pope and Pope (31) found that the opening of a Walmart store (Wal-Mart Stores, Inc., Bentonville, Arkansas) increased local housing prices. More research is needed to understand the relationship between residential mobility and obesity.

Among the limitations of this study is the fact that the study's findings may only be generalizable to insured adults with diabetes who live in Northern California. Because this study included (mostly older) adults with type 2 diabetes, the results might be different for young adults, children, or persons without diabetes. In addition, there were no data on participants who moved outside of the Kaiser Permanente Northern California catchment area. Changes in food environment might

Table 4. Coefficients (β (SE[a]) from Ordinary Least Squares Models of the Influence of Food Environment on Body Mass Index[b] With Individual Fixed Effects (Alternate Kernel Density Measures), Northern California, 2006–2011[c]

Exposure	ITT Approach			Non-ITT Approach[d]		
	All Participants	Nonmovers	Movers	All Participants	Nonmovers	Movers
1-mile (1.6-km) bandwidth	−0.09 (0.03)[e]	−0.09 (0.04)[f]	−0.06 (0.07)	−0.02 (0.02)	−0.08 (0.03)[f]	0.01 (0.03)
2.1-mile (3.4-km) bandwidth	0.10 (0.12)	0.12 (0.13)	0.06 (0.29)	0.03 (0.05)	0.05 (0.08)	0.02 (0.06)
4.5-mile (7.2-km) bandwidth	0.39 (0.27)	0.45 (0.29)	0.20 (0.67)	0.07 (0.09)	0.40 (0.18)	−0.01 (0.10)
No. of individuals	194,652	194,652	191,621	187,144	152,036	35,108
No. of observations	2,474,790	2,474,790	2,429,800	1,909,969	1,555,353	354,616

Abbreviations: ITT, intention to treat; SE, standard error.
[a] Robust SE clustered on the individual.
[b] Weight (kg)/height (m)^2.
[c] All models adjusted for age, Charlson comorbidity index, medication use, Medicaid enrollment, month, year, measurement method, and neighborhood variables.
[d] With the non-ITT approach, participants' residences were modeled using their actual address.
[e] $P < 0.01$.
[f] $P < 0.05$.

be accompanied by other changes in the built environment (new parks or restaurants), which might not have been controlled for by the community-level demographic variables. In addition, there might have been measurement error caused by the quality of the data obtained from InfoUSA. Finally, our study did not take into account the food environment of the workplace, which might have differed from that of the residential environment.

Cummins et al. (21) found that the opening of a new store in Philadelphia, Pennsylvania, increased awareness of access but had no relationship with obesity or diet. The results of that study, along with the clinically insubstantial findings of this analysis, call for the reexamination of food policies that encourage the opening of supermarkets in "food deserts" as a means of reducing obesity in the neighboring communities. Future studies should move beyond the geographical conception of food environment and take into account variation in food acquisition, food quality, and purchase price and purchase behaviors. Ghosh-Dastidar et al. (32) suggested that store-level prices might be more important than distance in the relationship with obesity. More research is needed to identify the factors that will be most salient in slowing the obesity epidemic.

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