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Automobile Traffic around the Home and Attained Body Mass Index: A Longitudinal Cohort Study of Children aged 10–18 Years

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Abstract

Objectives—The objective of this study is to examine the relationship between measured traffic density near the homes of children and attained body mass index (BMI) over an eight-year follow up.

Methods—Children aged 9–10 years were enrolled across multiple communities in Southern California in 1993 and 1996 (n = 3318). Children were followed until age 18 or high school graduation to collect longitudinal information, including annual height and weight measurements. Multilevel growth curve models were used to assess the association between BMI levels at age 18 and traffic around the home.

Results—For traffic within 150 m around the child's home, there were significant positive associations with attained BMI for both sexes at age 18. With the 300 m traffic buffer, associations for both male and female growth in BMI were positive, but significantly elevated only in females. These associations persisted even after controlling for numerous potential confounding variables.

Conclusions—This analysis yields the first evidence of significant effects from traffic density on BMI levels at age 18 in a large cohort of children. Traffic is a pervasive exposure in most cities, and our results identify traffic as a major risk factor for the development of obesity in children.

Keywords

Traffic; built environment; children; overweight and obesity; geographic information systems; multilevel models; cohort study

INTRODUCTION

Rates of overweight and obesity in children have nearly doubled over the past two decades. This doubling has increased the risks of obesity during adulthood and short- and long-term risks of cancer, type 2 diabetes, and cardiovascular disease (Dietz and Robinson, 2005). Metabolic and genetic research has deepened the understanding of the physiological aspects of body weight regulation (Stunkard, 1991). Little evidence, however, supports the notion that the current epidemic of obesity and related diseases is explained directly by acute metabolic or genetic defects. The more likely explanation relates to societal and environmental changes that promote the expression of an obese phenotype (i.e., fewer requirements for physical activity, more inducements to sedentary behavior, and greater abundance and availability of calorie-rich food) (Hill, et al., 2003). Because interventions to reduce obesity in children have been only modestly successful (Robinson and Sirard, 2005), prevention programs are needed to combat further increases in obesity and concomitant morbidity and mortality.

In the search for preventive solutions, researchers have focused on the influence of the built environment. Growing evidence now links the built environment to physical activity, dietary intake, and obesity (Papas, et al., 2007). Results from studies on adults, primarily based on cross-sectional data, have identified such variables as land use mixture, street connectivity (Frank, et al., 2005), food access (Morland, et al., 2006), and park access as modifiable factors significantly associated with physical activity and obesity (Dahmann, et al., 2009).

In the childhood and adolescent literature, results remain more mixed. The focus of most research has been on physical activity and the built environment. A review reported that the presence of sidewalks and controlled intersections, access to recreational facilities and schools, and access to desired destinations and public transportation were associated with physical activity in children and youth (Davison and Lawson, 2006). Another review suggested that home and school environments influenced physical activity levels in children, whereas low residential crime incidence was associated with more physical activity in adolescents (Ferreira, et al., 2007). Other work reported the number of cars (Frank, et al., 2007), proportion of green space (de Vries, et al., 2007), number of accessible destinations (Hume, et al., 2007), and safety (Alton, et al., 2007) contribute to children’s and adolescent’s physical activity. Overall, research suggests that the built environment may affect physical activity in children and adolescents. By extension, we might expect to see associations between obesity and the built environment.

Research investigating links between childhood obesity and the built environment remains formative. The majority of the studies are cross-sectional, and few have combined objective measures of weight status such as body mass index with objective measures of the built environment (Dunton, et al., 2009). Some of the results indicate null associations for the

same variables that appear protective in adults. For example, urban features such as walkability, retail floor area, land use mix and residential density were unrelated to obesity in adolescents (Kligerman, et al., 2007, Norman, et al., 2006).

Another possible influence on childhood obesity is traffic density and perceived safety. Although pedestrian injuries have declined over the past two decades, 1 in 5 deaths for children less than 14 years of age is from a traffic accident, and about one quarter of these involve pedestrians (Schieber and Vegega, 2002). In 2007, 647 children and young adults (ages 0–20) died in the US from pedestrian collisions with automobiles (FARS 2009). Perceptions of danger may impede mobility of children and contribute to the formation of obesity. Beyond the direct effects on perceptions, traffic-related air pollution may contribute to chronic disease formation (Gauderman, et al., 2007), which, in turn, may inhibit a child's capacity to engage in physical activity. Few studies have examined the role of traffic exposure as a risk factor for obesity in children. Researchers have investigated the relationship between vehicle miles traveled (VMT) in a county and adult obesity, with results suggesting that higher VMT is a risk factor (Lopez-Zetina, et al., 2006). Parent reports of heavy traffic were associated positively with obesity in older children, but had no association with obesity in younger children (Timperio, et al., 2005).

To our knowledge, there are no studies that link objectively measured traffic volumes near homes to the longitudinal development of childhood obesity. Traffic is a pervasive and involuntary exposure in many urban areas of the world, and by most accounts traffic is increasing at a rate greater than population growth (Frumkin, et al., 2004). The objective of this paper is to assess levels of Body Mass Index (BMI in kg/m^2) at the end of the childhood growth period in a large cohort of children living in Southern California in relation to residential traffic exposure around the home.

METHODS

Study design

A cohort of 3318 children aged 9–10 from 12 communities in Southern California within 200 miles of Los Angeles was enrolled in 1993 and 1996. These data formed the Children's Health Study cohort that focuses on assessing associations between respiratory health and environmental factors. (One of 12 original communities was excluded from the analysis because addresses could not be geo-coded accurately to estimate traffic exposure). Details on the design, site selection, subject recruitment and assessment of health effects are reported elsewhere (Navidi, et al., 1994). Briefly, children were recruited through their schools. At baseline, a parent or guardian of each participating child provided written informed consent and completed a written questionnaire that supplied detailed information on family demographic characteristics, history of respiratory illness and associated risk factors, indoor sources of exposure, physical activity patterns of the children, and household characteristics. In spring of each study year, an update questionnaire was completed by each child, and anthropometric measures such as height and weight were obtained.

Every child enrolled in the CHS had height and weight measured by a trained technician at baseline and annually through the entire 8-year follow-up. Technicians followed a

standardized procedure that included details on scale calibration and interaction with the children. These objective measures of height and weight allow for precise and accurate calculation of BMI (kg of weight/height in m^2). BMI and questionnaire-based individual information were then linked to built-environment measures around the children's homes and schools, along with physical features such as topography and social environment variables in the census tract of residence (e.g., poverty). Home locations were geocoded using the Teleatlas geocoding database to the corresponding road network. The research protocol was approved by the Institutional Review Boards of the University of Southern California and the University of California, Berkeley.

Description of Built Environment Measures

Figure 1 illustrates the conceptual path model that guided the GIS data compilation and subsequent statistical analysis. Two main pathways lead to obesity: diet and physical activity, either of which may contribute to positive energy balance. For the home neighborhood, the built environment may also have a direct effect on physical activity. We posit that aspects of the physical environment may create contextual effects that limit activity through perceived threats such as traffic or, for example in the case of air pollution, through acute health effects such as respiratory symptoms. We also indicate a feedback loop that follows the pathway of diseases where the environment leads to chronic health effects from risks such as air pollution, which may reduce physical activity. Food environments in the school, home, and areas surrounding these two important locales may also influence food intake. Finally, variables that represent urban sprawl, social context, and crime at the community or town level were specified in our multilevel models as potential confounders of local built environment influences such as traffic around the home. We were unable to operationalize all aspects of this path model, and the implications of these exclusions are underscored in our discussion section.

We organized our built environment variables in a geographic information system (GIS) through a classic framework identified by Lynch. This framework helps to classify how people perceive and navigate within cities through five essential features: districts, edges, paths, nodes, and landmarks (Lynch, 1960). Districts form distinct neighborhoods or easily recognized zones that are internally homogenous; edges are the boundaries and barriers between these districts; paths connect within and between districts; nodes are destinations and places where people gather; and landmarks serve to guide people through their neighborhoods. Landmarks were excluded from our analysis because their inclusion would have required detailed perceptual information that was unavailable to us in the study questionnaire. Table 1 summarizes the variables tested in this analysis for built environment influences. Unless otherwise noted, variables were tested with 500 m Euclidian buffers. Earlier screening of variables at smaller radii indicated that some features, such as food access, had limited data available below 500 m, while larger radii buffers beyond 500 m tended to diminish the effect sizes.

Traffic Density Exposure Estimates—Traffic exposure variables were based on the California Department of Transportation Functional Class (FC) data for the year 2000. Because the FC data are linked to a road network with lower positional accuracy, the annual

average daily traffic (AADT) volumes were conflated to the Teleatlas road network, which has excellent positional accuracy (Wu, et al., 2005). The link-based traffic volume data are available for freeways, highways, arterials, and some major collectors; they are not available for minor collectors that are common in suburban neighborhoods. The traffic data are based on continuous measurements on freeways, highways, and some major arterials, and intermittent measurements within the previous 3 years on other roads. The spatial pattern of traffic density changes slowly over time and the temporal period used here supplies a good representation of the longer-term traffic patterns around the subjects.

Maps of traffic densities, similar to those shown in Figure 2, were computed using a kernel estimate in the ArcGIS Spatial Analyst based on the line-link data with a 10 m grid cell size and a distance decay function of 90% within the first 150, 300, or 500 m. This method of estimation accounts for the likely diminishing effect of traffic around the home as a function of distance away from the residence (Bailey and Gatrell, 1995, Kan, et al., 2007). Compared to simple traffic metrics, such as the distance to the nearest big road, the traffic density metric has the advantage of accounting for multiple roads influencing a location of interest.

We hypothesize that more proximate exposures would have a larger effect on childhood BMI growth because the area most near the home probably influence perceptions of both the parent and the child with respect to traffic danger. Due to the lack of prior research investigating traffic effects, we had no prior knowledge of the distances around the child's home most likely to exert effects. We therefore explored point estimates of traffic density at each child's home using the 150, 300, and 500 m distance buffers. Each buffer used the same distance decay function specified above, which up-weights the local traffic influence. Therefore, the larger buffers examined the influence of more distant traffic, assuming a more gradual decline in the impact of traffic with distance from the home.

As a sensitivity analysis, we also used distance measures to major roads and highways. We assessed continuous distance to a limited access highway, which would be likely to have high traffic nearby. We also restricted this distance analysis to children living within 500 m of a major highway. This restriction was applied to examine distance decay gradients in areas of likely high traffic intensity.

Data analysis

We used a multi-level growth curve model (Berhane and Molitor, 2008) that employed a flexible linear-spline based approach to characterize the nonlinear BMI trajectories during childhood. Similar models have been used extensively for assessing lung function growth in our cohort (Gauderman, et al., 2007) and were adapted for BMI growth. All BMI data were checked for outliers and internal consistency in the growth curves. We assessed separate gender-specific growth trajectories in models that combined data from both sexes and also modeled common adjustments for covariates. This modeling approach allowed for examination of the effects of covariates of interest at various levels: between times (within individual), between individuals, and between other levels of aggregation (e.g., school, neighborhood, community). The BMI trajectories were estimated using linear splines with breakpoints (known as knots) at ages 12, 14, and 16, essentially fitting four straight lines for <12, (12–14), (14–16) and >16 years of age and joining them smoothly at the knots to

provide nonlinear growth trajectories. Through reparameterization, attained BMI levels at age 18 were then extracted and their association was examined with explanatory variables.

Many individual risk factors for BMI growth, such as asthma and other respiratory conditions, individual and neighborhood/community level socio-economic indicators, smoking (both personal and second hand smoke), and numerous built environment variables around the home and school, were also tested as confounders. Table 2 shows the variables and the levels at which they were tested.

A final model was developed by including a set of basic design variables (race, ethnicity, gender, cohort of enrollment, and community) and any other additional confounders. Confounders were selected if they had a bivariate association with the outcome ($p < 0.2$) and they changed the effect of interest – traffic density – by at least 10% in either of the gender specific effects at each buffer distance (i.e., 150, 300, and 500 m). This allowed us to develop a consistent set of confounders to be used for each traffic exposure variable, which enabled comparison of the results between the different exposure metrics. Each traffic exposure model had different confounding variables based on our screening procedure. To compare across effects and maximally control for confounding, we selected the confounders from the 300 m model. Sensitivity analyses were also completed using confounding variables specific to each of the three traffic exposure variables, and the results were similar (not shown).

All analyses were conducted by using the SAS (SAS Institute Inc., Cary, NC) and/or R (R Foundation for Statistical Computing, Vienna, Austria) statistical software packages. Data were compiled using ArcGIS 9.2 (ESRI, Redlands, CA) and some distance measurements were computed using Matlab v.R2006a (The Mathworks, Natick, MA).

RESULTS

After exclusions for missing geocodes and other data, the analytic cohort used in this study included 2889 children. Table 3 gives the descriptive statistics on children, the BMI outcome variable, the exposure variables, and on the confounders selected through the testing procedure described in the Methods above. The majority of the cohort is White, Non-Hispanic, but a large proportion (30%) is of Hispanic ethnicity. African and Asian Americans comprised about 9% of the cohort. Some 35% of the children were exposed to second hand smoke and 14% had asthma. Annualized average traffic densities within the buffer distances averaged between 26 and 30 (dimensionless). The BMI z-scores based on the Centers for Disease Control classifications indicate 26.4% of the children were overweight or obese at enrollment (NCHS 2009). Table 4 gives details on the traffic exposures within each of the 11 communities. A wide range of exposure exist among the communities, with the highest traffic exposures in the urban communities of Long Beach and Riverside and the lowest in the more rural communities of Lake Elsinore and Lompoc.

Figure 3 presents the unadjusted average growth curves and the point estimates per year over the 8 years of follow up for males and females, respectively. As expected, average BMI

increased in both sexes and the gain over the 8 years of follow-up was approximately 5 BMI units for girls and 5.5 BMI units for boys.

Each model included parental education, personal weekly smoking, second hand smoke (current and past), ever asthma diagnosis, foreign born status, 500 m buffer population density, having no food stores within a 500 m road network buffer, gamma index of street connectivity within 500 m of the home (DeMers, 2004), proportion of people in poverty in the census tract of residence, average normalized difference vegetation index of greenness (NDVI) within 500 m of the home (Tilt, et al., 2007), and community level violent crime rate per 100,000. All models also included design variables for race/ethnicity (a composite variable), gender, cohort of enrollment, and community. In models that considered community level covariates, a random effect for community was used instead.

Results for the traffic exposure models at 3 distances are shown in Table 5 and Figure 4. Table 5 gives the exact coefficients based on a 1 unit change in traffic exposure, while Figure 4 plots the risks over the 10–90th percentile contrast in the traffic exposure (see Tables 3 and 4 for details of the traffic exposures). After inclusion of the confounding and design variables, we observed a significant positive association with attained BMI at age 18 and traffic density within 150 m. With the 300 m traffic exposure, attained BMI was associated with traffic, but only of borderline significance. Effects for the 500 m buffer were not related to BMI growth.

For the 150 and 300 m traffic exposures, results of the gender-specific models are shown in Figure 5. For the 150 m traffic exposure there is little difference between the estimates for males and females. With the 300 m traffic exposure, the effects appear larger in females than in males, but a test of interaction revealed no significant difference.

As a sensitivity analysis, we also examined distance from major highways with limited ramp access (FRC 01 Class), which would be expected to have substantial traffic nearby. The results were consistent with our traffic density findings, with decreased levels of attained BMI associated with increased distance from the highways. This association was not statistically significant. When we restricted the analysis to children within 500 m of a major highway, the effect became stronger and of borderline significance (results not shown). These results demonstrate a decay effect with closer distances to the roadways displaying larger effects and are consistent with our findings for traffic density.

DISCUSSION

Our analysis has detected a significant positive association between traffic density around the home and attained BMI at age 18 in a large cohort of children living in 11 Southern California communities. For increased traffic exposures within 150 m of the home, there was a significant increase in attained BMI for both sexes. For traffic density within 300 m of the home, the effects were about one third the size of those at 150 m and were of borderline significance. Other distance-based exposure metrics corroborated the findings from the traffic density models. Our results are consistent with the hypothesis that proximate exposures to traffic exert the largest effect on BMI growth. As the distance buffers increase

and the traffic exposure includes more distant effects, the size of effects consistently decreases.

The size of the traffic density effect is about 0.27 BMI attained units over the 10–90 percentile of the 150 m traffic distribution. This translates into about a 5% increase in attained BMI at age 18. Although this effect may appear small, the ubiquity of exposure to traffic implies that potentially large populations may be affected, such that even small changes in the BMI in response to traffic may be associated with impacts on overweight and obese status in the population. This risk may combine with other factors such as poor diet or sedentary behavior, putting more children at greater risk of becoming overweight as young adults. Empirical evidence suggests that once children are overweight or obese at age 18, there is a high probability (about 80%) of them remaining in overweight status into young adulthood (Whitaker, et al., 1997).

Possible reasons for this positive association between traffic density and the longitudinal progression of BMI are twofold. First and most directly, traffic around the home may create a sense of danger among parents and children that inhibits mobility on foot or bicycle (Timperio, et al., 2006). Second, in related research on the same cohort we have shown that air pollution from traffic relates to decrements in lung function growth (Gauderman, et al., 2007), prevalent asthma (McConnell, et al., 2006) and incident asthma (Jerrett, et al., 2008). Reduced lung function or asthma may reduce a child's capacity for exercise and other factors being equal reduce energy expenditure and create a positive energy balance that leads to higher BMI growth over time. As illustrated in Figure 1, both explanations may operate simultaneously.

A few limitations deserve mention. On the outcome data, we used BMI, which is not technically a measure of body fatness. Several studies have, however, demonstrated high correlations between BMI and body fatness in children and adolescents (Hu 2008; Johnson-Taylor and Everhart 2006). On the exposure variable, some of the areas around where the children in this study live had few direct traffic counts available. This probably has not affected the relative ranking of places by traffic density because many of the areas given zero values in our traffic density metrics most likely had very low traffic volumes. Thus the relative rank ordering of the exposures was a fairly good approximation of the traffic density, although we cannot rule out the possibility of some misclassification of exposure.

Returning to our conceptual model in Figure 1, on the intake of food, we controlled for food access, but we were not able to evaluate dietary factors directly. Given what is known about the association between lower socioeconomic position and higher traffic exposures in California (Green, et al., 2004), some of the effects observed here may be confounded by dietary variables that are also associated with lower socioeconomic status, such as sugar and fats (Drewnowski, 2007). Our models did control extensively for socioeconomic status in the home and neighborhood, so although possible, it is unlikely that residual confounding relating to social status is present in the models. Finally, indicators of physical activity available to us for this cohort (participation in team sports and time spent outside) did not explain the effect of traffic, but these variables may not evaluate the relevant physical

activity near the home that would be likely to mediate the effect of traffic exposure on the increase in BMI over the follow up period.

CONCLUSION

Although future research will be needed to replicate the findings here, assessing the role of traffic in the development of obesity in children is important for guiding possible interventions. Traffic is a modifiable risk factor, which can be calmed or diverted to other areas to avoid exposure in sensitive residential neighborhoods (Bunn, et al., 2003, Zein, et al., 2001). Other measures such as congestion charging may actually reduce traffic in sensitive areas. For example the London congestion charge reduced traffic by 18% in the central city (Santos 2004). Moreover, solutions posited in existing literature on built environments suggest that connected, mixed land use neighborhoods with good "walkability" to destinations in proximity to the home may provide some protection against obesity and physical inactivity. If traffic is an independent risk factor, however, improving the physical structure of the city may be ineffective if high levels of traffic persist in residential neighborhoods.

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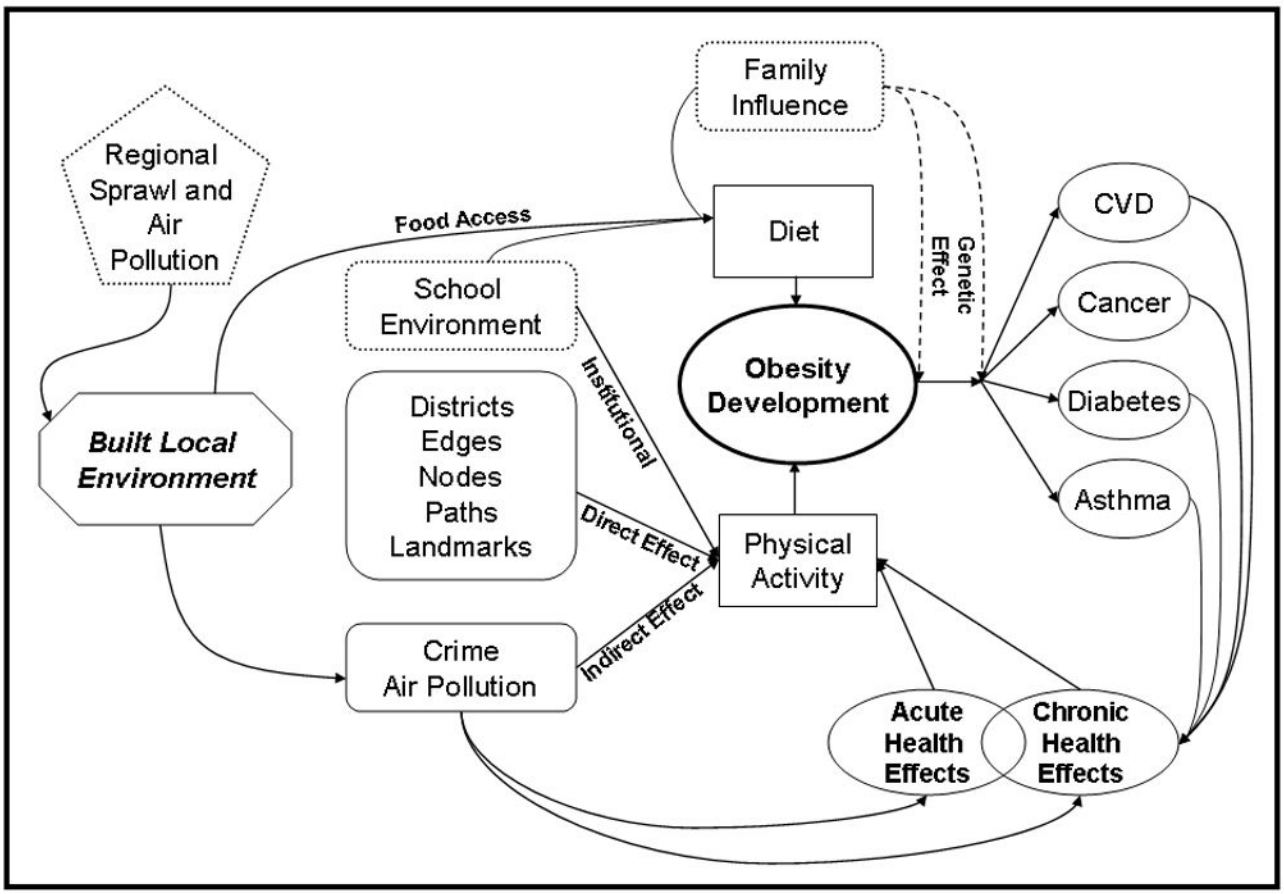


Figure 1.

Conceptual Path Model Showing Possible Influence of the Built Environment in the Context of other Variables that May Affect the attained BMI in Children

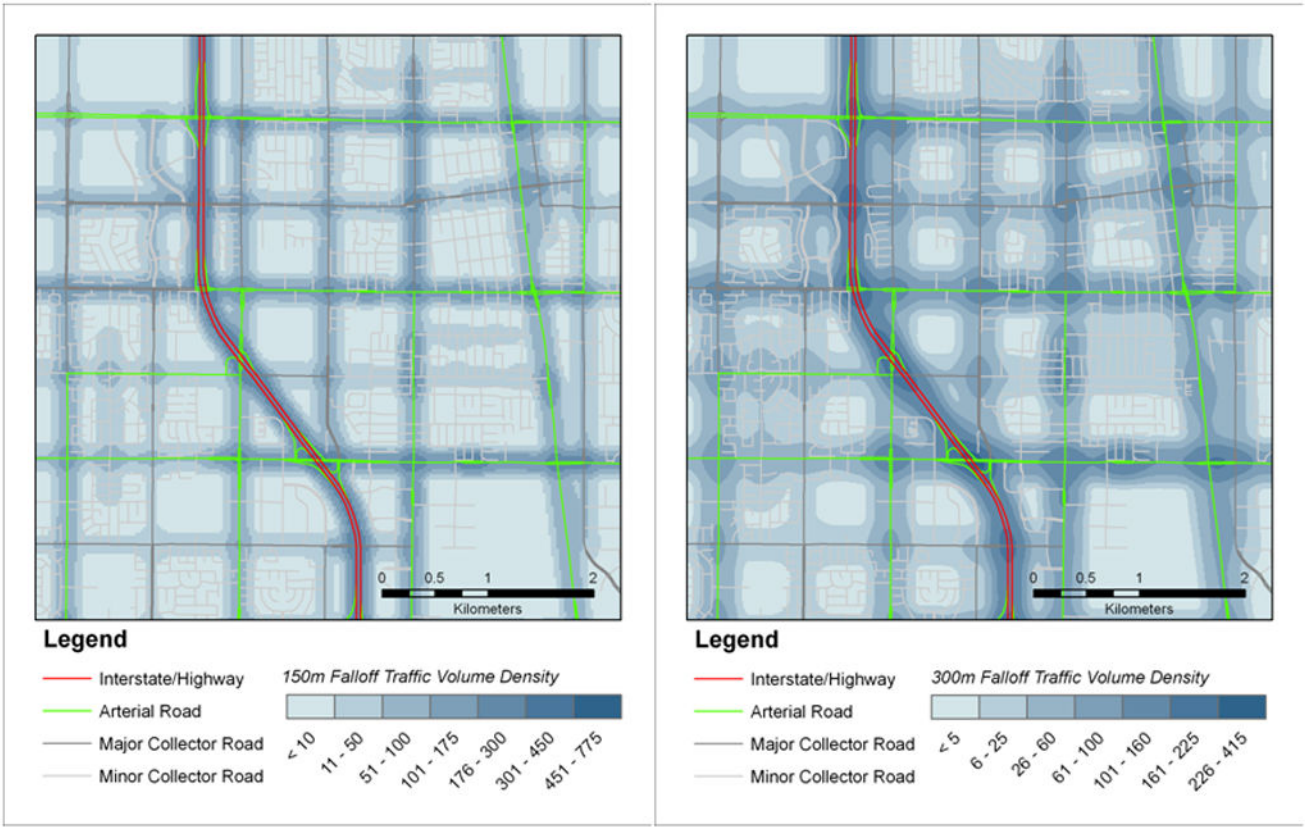


Figure 2.

Sample Traffic Density Maps with 150 m (left) and 300 m (right) Distance Decay Kernel Functions for Lancaster, CA.

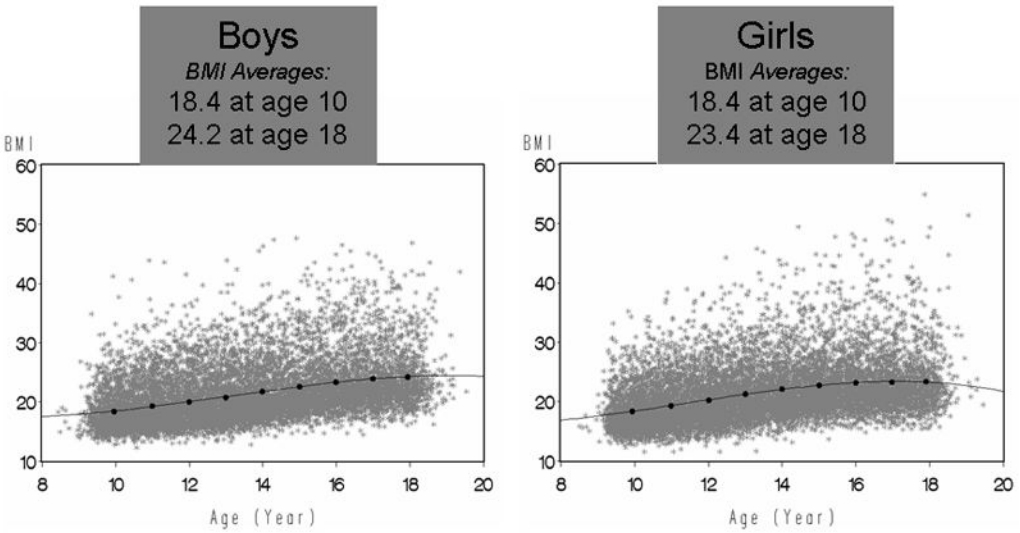
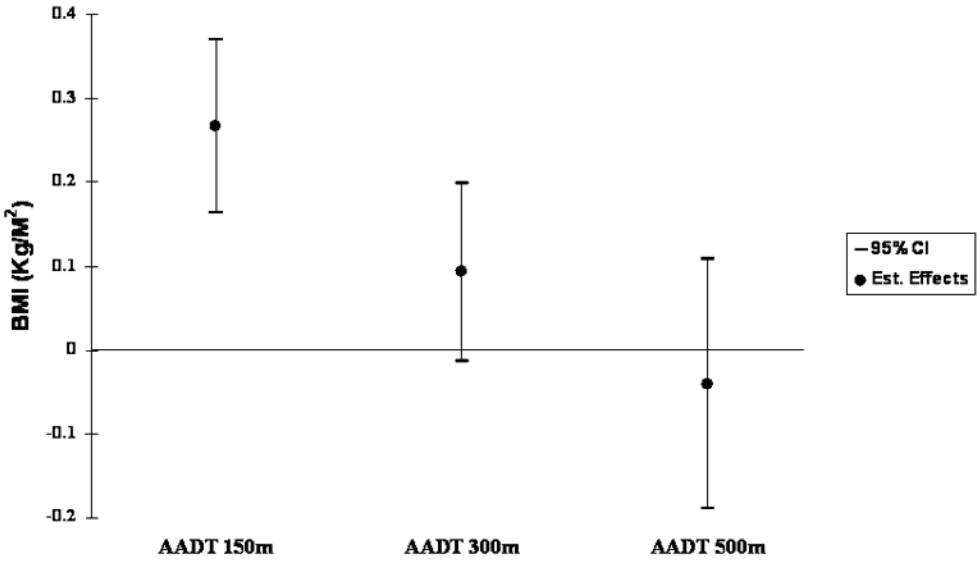


Figure 3.

BMI Growth Over 8 Years within the Analytic Cohort of Children Aged 10–18 in 11 Southern California Communities with Individual Measurements and the Yearly Average of all Measurements Plotted



Confounders: Ethnicity/Race, Gender, Cohorts variables, in addition, adjusted for Parental Education, Personal Weekly Smoking, Second Hand Smoke (Current + Past), Ever Asthma, Buffer Population, Gamma Index, Proportion of Below Poverty People within Census Block, NDVI, Foreign Born, Town Level Violent Crime Rate, and Having No Food Stores within 500m Road Network Buffer with Random Community Effects

Figure 4.

Effects of Annual Average Daily Traffic Exposures at 150 m, 300 m, and 500 m on Attained Body Mass Index at Age 18 in Children Aged 10–18 in 11 Southern California Communities with Adjustment for Confounders

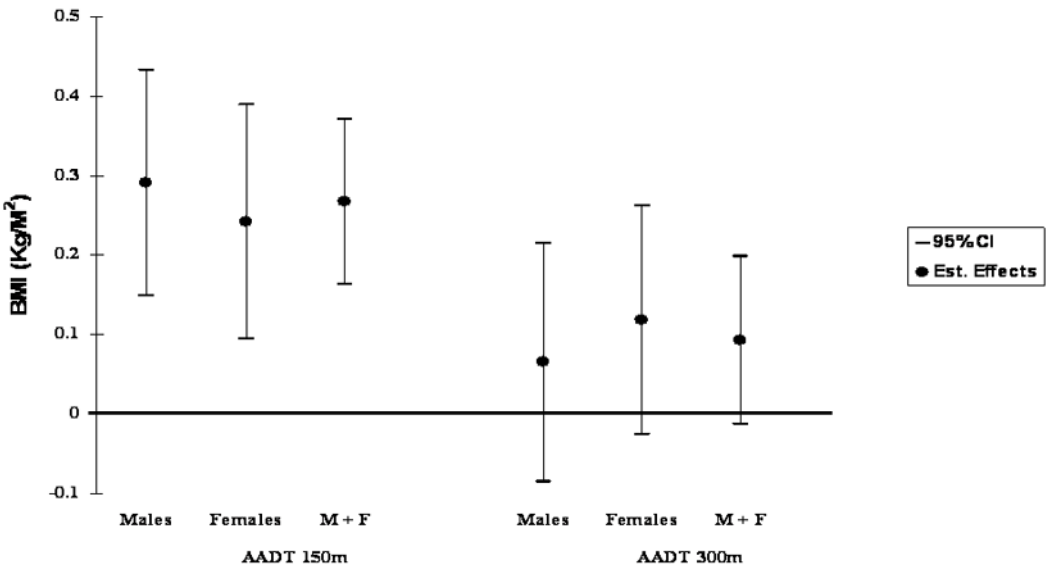


Figure 5.

Comparison of 150 m and 300m Traffic in Females and Males in Children Aged 10–18 in 11 Southern California Communities

Table 1

Built Environment Variables Used in the Data Analysis Organized Under the Lynch Framework[1]

Lynch Framework	Variable	Operationalized[2]
Paths	Connectivity	Measure of Block Size and Intersection Type (T vs. X); Gamma and Beta Indices
	Road Type	Major versus Minor/local Roads
Nodes	Destinations	InfoUSA Detailed Business Data on Food Retail Sources
	Unhealthy food access	InfoUSA Detailed Business Data on Food (over the counter food vendors)
	Healthy food nodes	InfoUSA Detailed Business Data on Grocery Stores
	Recreation	Location of Parks and Recreation Programs Assembled from Web Audits of Public Data Sources
	Commercial	Land Use Data from Municipal Provider Organizations
Edges	Major Roads	TeleAtlas and Proximity to Highways
	Major Industrial	Land Use Data from Municipal Provider Organizations
	Physical Barriers	Land Use Data from Municipal Provider Organizations
	Topography	United States Geological Service Digital Elevation Model Data Processed to Assess Hilliness in Terrain (i.e., variance in elevation)
Districts	Crime	Crime per 100,000 for Various Crimes at the Community Scale from the California Department of Justice
	Green Cover	Total Green Space Based on Land Use, and on Normalized Difference Vegetation Indices Derived from Landsat Images Taken During the Wet Season
	Air Pollution	Estimated by CALINE Dispersion Models
	Traffic Density	Annualized Average Daily Traffic from the California Functional Class Data (conflated to the Teleatlas road network)
	Population Density	Pop/sq. Mile
	Land Use Mix	Homogeneity Index Similar to Frank et al. 1994
	Block Size	Average Block Size

[1] Subjects were from 11 Communities in Southern California with Aged 10–18 years

[2] Variables operationalized in 500 m Euclidean buffers around the subjects homes. For food access, 500 m network buffers were used as well around homes and schools. Other spatial scales tested but not reported. See text for details.

Table 2

Variables Included in the Multilevel Growth Curve Analysis[1]

Variable Group	Variable	Data Source	Spatial Scale
Individual Outcome (BMI kg/m2) and Confounding Variables	Height	CHS Questionnaire	Individuals Living in the 11 Study Communities
	Weight		
	Sex		
	Race		
	Age		
	Diagnosed Asthma Ever		
	Current Wheeze		
	Smoking		
Family Environment Confounding Control	Parental Education	CHS Questionnaire	In Homes of Study Subjects
	Household income		
	Family socioeconomic status (parent education)		
	Environmental Tobacco Smoke		
	Health Insurance Availability		
	Parental Respiratory Disease		
Neighborhood Home Context	**Refer to** Table 1 for Details of Built Environment Variables		
Neighborhood Social Context	Unemployment in Census Tract of Home	Census 1990	Census Tract
	Poverty in Census Tract of Home		
	Low Education in Census Tract of Home		
Community Scale	Density variables for Sprawl	Census 1990	Over 11 Communities
	Unemployment		
	Low Education		
	Poverty rate		
	Crime Rate	California Dept. of Justice 1998	

[1] Subjects were from 11 Communities in Southern California with Aged 10–18 years

Table 3

Subject Characteristics, Exposures and Confounding Variables Used in the Analysis from 11 Communities in Southern California with Children Aged 10–18 years

Risk Factor	No. (%)[a]	Mean (SD)
Subject Characteristics		
Race/Ethnicity		
African American	155 (4.7)	
Asian	151 (4.6)	
Hispanic	1000 (30.1)	
White, Non-Hispanic	1825 (55.0)	
Other	187 (5.6)	
Gender		
Male	1647 (49.6)	
Female	1671 (50.4)	
Individual and Household Characteristics		
Parental Education (by Ketan)		
High School or Less	1055 (33.2)	
Some College	1417 (44.6)	
College or Greater	705 (22.2)	
Personal Weekly Smoking		
Not Smoked in the Past One Week	3311 (99.8)	
Ever Smoked in the Past One Week	7 (0.2)	
Second Hand Smoke (Current + Past)		
No One Ever Smoked in the House	2070 (65.4)	
Any One Ever Smoked in the House	1094 (34.6)	
Second Hand Smoke (Current)		
No One Currently Smokes in the House	2590 (80.5)	
Any One Currently Smokes in the House	553 (17.2)	
Currently Smokes in the House only when Child Away	73 (2.3)	
Ever Asthma		
No	2775 (85.8)	
Yes	459 (14.2)	
Foreign Born		
No	3059 (93.4)	
Yes	216 (6.6)	
Local Home or School Environment		
Having No Food Stores within 500m Road Network Buffer		
No	826 (26.2)	
Yes	2333 (73.8)	
Buffer Population (Total Population within 500 m buffer)	3162	1256.8 (1121.0)
Street Connectivity (Gamma Index 500 m buffer)	3162	0.4 (0.1)
Parks and Recreation (unit: Acre in 500 m buffer)	3162	5.6 (13.9)

Risk Factor	No. (%)[a]	Mean (SD)
NDVI v3 (0–0.42 in 500 m buffer)	3162	0.1 (0.1)
Neighborhood Social Context		
Proportion of Unemployment Males and Females within Census Tract	3161	0.1 (0.0)
Proportion of Below Poverty People within Census Tract	3058	0.1 (0.1)
Community Scale		
Community Level Violent Crime Rate (Crime per 100,000 population) (347.83 – 1670.58)	3318	885.5 (432.8)
Exposure Variable of Interests		
AADT 150m	2943	26.7 (74.8)
AADT 300m	2943	30.1 (69.0)
AADT 500m	2943	36.6 (56.0)
Major Outcome		
BMI at Baseline [b]	*3173*	*18.3 (3.4)*
Males	1587	18.3 (3.4)
Females	1586	18.3 (3.4)
BMI CDC percentile at Baseline		
BMIp < 85	2334 (73.6)	
85 ≤ BMIp < 95	460 (14.5)	
95 ≤ BMIp	379 (11.9)	
BMI at Age 18[c]	*1262*	*23.8 (4.8)*
Males	620	24.1 (4.7)
Females	642	23.5 (5.0)
BMI CDC percentile at Age 18		
BMIp < 85	952 (75.4)	
85 ≤ BMIp < 95	164 (13.0)	
95 ≤ BMIp	146 (11.6)	

[a] Total subject number=3318 at baseline; numbers of subjects in table vary due to missing covariates values.

[b] The first observation of the subject in the first year of the study.

[c] 17.5 ≤ Age < 18.5.

NOTES:
(1) Age 18 means 17.5 –18.5.

(2) 10–90%ile Range of AADT 150m is 69.2.

(3) 10–90%ile Range of AADT 300m is 70.3.

(4) 10–90%ile Range of AADT 500m is 79.9.

Table 4

Descriptive Statistics for the Traffic Exposure Variables based on Assigned Exposures of Annual Average Daily Traffic Kernel Estimates at 150, 300 and 500 m[1]

Community	Sample size (n)	AADT 150m		AADT 300m		AADT 500m	
		Mean (SD)	Range	Mean (SD)	Range	Mean (SD)	Range
Alpine	273	15.7 (29.2)	196.2	15.6 (26.5)	149.9	16.5 (20.7)	79.3
Lake Elsinore	262	8.1 (18.4)	97.3	8.8 (14.7)	65.5	10.8 (11.9)	59.9
Lancaster	284	16.9 (29.0)	165.8	17.3 (23.6)	132.2	22.3 (14.6)	93.6
Lompoc	245	10.8 (14.2)	77.0	12.0 (13.4)	58.2	13.4 (10.4)	42.9
Long Beach	286	79.3 (163.9)	1338.9	89.1 (143.8)	960.3	99.3 (103.7)	480.1
Mira Loma	275	9.7 (17.0)	133.9	10.4 (16.4)	148.4	12.5 (16.6)	145.9
Riverside	277	57.5 (135.4)	789.3	65.1 (125.5)	574.5	72.2 (86.3)	473
San Dimas	251	29.9 (59.4)	423.3	34.5 (55.4)	384.8	43.8 (47.5)	232
Atascadero	258	15.2 (27.1)	193.5	18.2 (30.4)	195.4	20.0 (24.0)	115.5
Santa Maria	273	26.2 (38.5)	211.8	33.4 (31.5)	162.2	60.3 (40.2)	147.7
Upland	259	19.2 (27.4)	151.1	20.9 (24.9)	165.7	24.7 (30.1)	322

[1] Subjects were from 11 Communities in Southern California with Aged 10–18 years

Table 5

Results from the Multivariate Growth Curve Models for Traffic at 150, 300 and 500 m Based on a 1 Unit Change in Traffic Exposures[1]

Variables of Interest	Combined Effect Modeling[2] Male + Female	
	β (SE)	P-value
Traffic		
AADT 150m	0.0039 (0.0008)	<0.0001
AADT 300m	0.0013 (0.0008)	0.09
AADT 500m	−0.0005 (0.0009)	0.59

[1] Subjects were from 11 Communities in Southern California with Aged 10–18 years

[2] Confounders: Ethnicity, Gender, Cohorts variables, in addition, adjusted for Parental Education, Personal Weekly Smoking, Second Hand Smoke (Current + Past), Ever Asthma, Buffer Population, Gamma Index, Proportion of Below Poverty People within Census Block, NDVI, Foreign Born, Community level Violent Crime Rate, and Having No Food Stores within 500m Road Network Buffer with Random Community Effects.