Social Epidemiology and Spatial Epidemiology: An Empirical Comparison of Perspectives

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Abstract

Social and spatial epidemiologists each bring a unique perspective to how they examine contextual or neighborhood-level determinants of health. Although both perspectives draw from epidemiology, social epidemiology is additionally grounded in sociology and causal counterfactual frameworks while spatial epidemiology is heavily influenced by medical geography and predictive models. No study to date has compared these two distinct perspectives, along with their corresponding analytical approaches and model results. Yet this comparison may advance contextual effects research in epidemiology by suggesting methodological enhancements, providing insights into the robustness of our conclusions to the perspective taken, and suggesting whether we can truly identify contextual effects from observational data.

To facilitate this comparison we used both perspectives to examine a research question: What is the estimated effect of increasing neighborhood education or income on overweight/obesity, type 2 diabetes, and current smoking, independent of individual-level differences? The social epidemiology approach employed propensity score matching while the spatial approach used approximated spatial multilevel models. Data for this study came from the California Health Interview Survey (2005, 2007, 2009) and the American Community Survey (2006-2010).

Results revealed minimal to no effect of neighborhood education and income on overweight/obesity, type 2 diabetes, or current smoking, but estimated effects did vary somewhat by approach.

This comparison highlighted fundamentally different goals in social and spatial epidemiology: identifying causal factors to intervene compared to predicting potential causal factors to describe reality. Attempts to improve causal inference in observational studies by integrating analytical techniques across subfields will likely be hampered by different objectives and model requirements. This incompatibility for integration, lack of strong evidence of effects, and the overall identification problem cast further doubt on our ability to identify causal contextual effects using observational data. However, this work may help in the design of experiments, which is where we should now focus.

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1. Introduction

The unique perspectives of social epidemiologists and spatial epidemiologists influence how each subfield of research approaches answering a similar contextual or neighborhood effects research question. The perspectives differ in terms of their theoretical and disciplinary foundations, foci, goals, and key challenges, which in turn influences not only their analytical approaches but also their model inferences. Although both perspectives draw from epidemiology, social epidemiology focuses on how social structures and phenomena influence health, while spatial epidemiology focuses on the spatial distribution of risk factors, disease outcomes, and their spatial intersection. Furthermore, social epidemiology frames causal research questions within the counterfactual causal framework, while spatial epidemiology works toward a strong predictive model.

No study has compared these two distinct perspectives, despite an increasing amount of work over the last few decades by both social and spatial epidemiologists examining contextual determinants of health and strong interest recently in bridging the divide between social epidemiology and spatial epidemiology or medical geography. Comparing these perspectives may advance contextual effects research in epidemiology by suggesting techniques one subfield can borrow from the other to make methodological enhancements, providing insight into whether our conclusions are robust to the chosen perspective and whether or not we can truly identify contextual effects. Previous methodological advances, such as multilevel models and geographic information systems

(GIS),[1-3] furthered contextual effects studies; however, many challenges remain to truly disentangling contextual effects from compositional effects.

To facilitate a comparison of perspectives, this study examined important public health problems hypothesized to be affected by contextual level factors. We selected neighborhood socioeconomic status (SES) as our exposure of interest as evidence suggests that individuals residing in lower-socioeconomic status (SES) neighborhoods[4-8] experience worse health. Specifically, we examined the relationship between neighborhood SES and several public health problems: overweight/obesity,[9-12] type 2 diabetes,[13, 14] and current smoking.[15, 16]

In particular, this study addressed the following research questions:

Research Question 1: What is the estimated effect of increasing neighborhood-level SES (defined as percent with at least a bachelor's degree or median household income) on individual-level overweight/obesity, type 2 diabetes, and current smoking, accounting for differences in individual-level characteristics?

Our objective was to address this question from both the social epidemiology and spatial epidemiology perspectives using cutting edge analytical techniques that address a key challenge to disentangling contextual effects. The social epidemiology perspective framed this question within a causal counterfactual framework and sought to mimic an experimental design as best as possible. This prompted a refined research question for the analytical approach: What is the estimated effect of moving to a neighborhood with one-quartile higher SES (education or income) on individual-level overweight/obesity, type 2 diabetes, and current smoking, accounting for differences in individual-level

characteristics? This study estimated this effect using propensity score matching in order to address challenges related to social stratification and structural confounding. Our hypothesis was that living in a neighborhood with higher SES would improve health, after accounting for individual-level differences.

By comparison, the spatial epidemiology perspective asked an etiologic question grounded in spatial econometric theory, but lacked any explicit causal framework. Furthermore, the spatial perspective sought to enhance the predictive ability of the model by incorporating spatial dependence and examining spatial patterns. The spatial epidemiology analytical approach followed from the basic research question, but also asked: Are neighborhood effects dependent on the characteristics of nearby neighborhoods? This approach approximated a spatial multilevel model and used spatial lag regression. Our hypothesis was that accounting for individual-level differences, living in a neighborhood with higher SES would improve health, and this effect would influence and be influenced by the effect in nearby neighborhoods.

Research Question 2: How are the social epidemiological and spatial epidemiological perspectives on contextual effects different, and what can we learn from comparing the two perspectives?

The overall goal for this question was to identify potential analytical improvements that may enhance causal inference from contextual effects studies using observational data. Our objectives specifically were to address the first research question again but with more traditional multilevel models, to compare the results from the social and spatial approaches to answering the first research question through the more

traditional multilevel models, and to broadly compare the perspectives and analytical approaches from the social and spatial models. Our hypothesis was that comparing the analytical approaches and results from these two perspectives would reveal important differences, some of which might be incorporated into the other approach to improve our causal conclusions and/or enhance our ability to disentangle contextual effects from individual-level characteristics.

We conducted a cross-sectional study using high-quality individual-level data pooled from the 2005, 2007, and 2009 California Health Interview Survey (CHIS) and contextual-level data from the 2006-2010 American Community Survey (ACS) 5-year Summary File from the US Census. The study sample was adults residing in the City and County of San Francisco, California.

2. Background

2.1 Contextual Effects

Also known as place, area, region, or neighborhood effects, contextual effects research examines characteristics of the social and/or physical environment hypothesized to affect an individual's health and health behaviors.[4, 17] Contextual effects arise from the context in which the population lives and are distinguished from compositional effects, which are due to differences in the characteristics of individuals across neighborhoods.[18] Interest in examining contextual factors has risen in recent decades with the acknowledgement that individual-level risk factors alone may fail to explain disease risk and health behaviors[4, 19-21] and the realization that identifying contextual-level health determinants offers the potential to intervene on a broader scale and thereby have a

greater impact on public health.[1, 19, 22, 23] However, despite increasing agreement on the importance of examining contextual effects on health,[4, 8, 17, 24] most studies suffer from methodological challenges that limit causal inference.[2]

The traditional approach to examining contextual determinants of health uses a multilevel model that regresses the health outcomes on contextual exposures, adjusts for differences in individual-level characteristics, and accounts for clustering within neighborhoods. To date the vast majority of contextual effects studies have been observational and have used this traditional multilevel model. (One rare exception is the Moving to Opportunity (MTO) study;[1] MTO was a randomized trial which relocated some participants to new neighborhoods while keeping others in their existing neighborhoods.[25]) Both social and spatial epidemiologists acknowledge a complex web of factors influencing health, where individuals influence the context in which they operate and the context influences the individuals.[26, 27] However, the traditional multilevel model approach fails to explicitly incorporate the complexity of social and spatial relationships, and thus has many challenges when attempting to disentangle contextual effects. Fortunately, social epidemiologists seek to address the complexity relating to social relations, while spatial epidemiologists work on challenges relating to place. Both approaches may enhance causal inference in contextual effects studies.

2.2 Social Epidemiology Perspective

Social epidemiologists typically examine social determinants of health, including social structures such as neighborhood SES.[2, 3, 26, 28] In an attempt to better disentangle contextual effects, social epidemiologists focus on the challenge of social stratification

and relatedly structural confounding. Social stratification is the process whereby different types of individuals sort into different types of neighborhoods. When individuals are sorted to the extent that similar people do not exist across different contextual exposure/treatment levels, we lack individuals who are the same but for the exposure. This lack of observations or data is called structural confounding and cannot be solved by simply adding more data.[29-31] Adding more data adds more observations to the strata with data and still leaves the other strata without data. Broadly speaking, confounding occurs given an imbalance of background characteristics. This imbalance provides an alternative explanation for the relationship identified and should be accounted for to rule out competing explanations and minimize bias. More specifically, structural confounding limits causal inference by violating positivity, as exposed individuals cannot be found in the unexposed neighborhoods and vice versa, and producing non-exchangeable exposure groups.[1, 2, 8, 29, 32, 33] Structural confounding is common in contextual effects studies, but studies using regression typically fail to address this challenge, which leads to off-support inference. Social epidemiologists increasingly seek to address the challenge of structural confounding by using propensity score matching together with a counterfactual causal framework.

The counterfactual theory of causation says that given an observable person or population exposed to treatment condition X with outcome Y, the counterfactual is the *un*observable outcome in the same person or population if they had *not* been exposed to treatment condition X at the very same time with all else being equal.[34-36] Clearly two mutually exclusive treatment conditions cannot exist in the same person at the same time,

so we attempt to approximate the counterfactual as best as possible. The ideal experiment is designed around the counterfactual, where one group is assigned the treatment and another group which is as similar as possible to the first group remains untreated.[2, 35] In observational contextual effects studies where the researcher cannot control the treatment assignment, the researcher typically adjusts for potential confounders in a multilevel regression model in an attempt to balance the covariates of those in the 'treated' group with those in the 'untreated' group as would be expected under randomization.[34] However, as previously mentioned, regression adjustment does not necessarily balance the covariates and may result in structural confounding.

Propensity score matching (PSM) offers a more sophisticated technique to balance covariates and better mimics an experimental design.[1, 30] In PSM exposed and unexposed individuals are matched based on a similar propensity to be exposed and then can be compared in an average effect of the treatment on the treatment (ATT) analysis. Despite providing more comparable or exchangeable populations, PSM does have some limitations including: potential confounding due to mismeasurement or missing variables,[1, 30] use of binary exposures only, potential loss of data when no match, no accounting for clustering within neighborhoods, and the assumption that treatment of one does not affect the others (i.e. stable unit treatment value assumption [SUTVA]).[30, 36]

2.3 Spatial Epidemiology Perspective

While social epidemiologists focus on the role of social structures, spatial epidemiologists primarily focus on the spatial distribution of diseases and their place-based determinants.[37, 38] In contextual effects studies spatial epidemiologists explicitly

address the challenge of the spatial dependence or spatial autocorrelation of nearby observations across neighborhoods or contexts. Although traditional multilevel models do account for the lack of independence of observations within a neighborhood, they ignore spatial dependence. This assumes that neighborhoods are independent units; however, many spatial researchers argue that neighborhoods (or other contextual units) should not be treated as independent.[39-43] Ignoring spatial dependence may bias effect estimates and affect estimate precision.[44-47]

Spatial contextual effects work has increasingly incorporated work from spatial econometrics to address spatial dependence.[43, 48-50] Spatial econometrics offers several spatial regression models, including one accounting for dependence in the error terms (spatial error model) and another accounting for dependence in the outcomes (spatial lag model).[44, 51, 52] Correlation in the error terms is considered a nuisance and may be due to model misspecification (e.g. missing variable), context misspecification (e.g. mismatch between neighborhood boundaries and scale of phenomena), or correlation in the exposure variables or covariates.[44, 53] As well as addressing spatial dependence, the spatial lag model explicitly examines spillover effects. Spillover effects theory posits that the effect of an exposure within one neighborhood spills over into surrounding neighborhoods.[49, 54] Research in sociology and public health supports spillover of the negative effect of neighborhood deprivation or crime on health.[54-56]

The primary limitation of the spatial error and lag models is the requirement of using only area-level (i.e. neighborhood-level) data, instead of both individual- and neighborhood-level data as in traditional multilevel models. However, a spatial multilevel

model can be approximated by first running ordinary least squares (OLS) regression using the individual-level covariates and outcome, and then using the predicted values from the OLS regression – which account for individual-level differences – in the spatial regression models.[49] These approximated spatial multilevel models incorporate spatial dependence into contextual effects studies, potentially enhancing causal inference over traditional multilevel models.

2.4 Neighborhood Socioeconomic Status and Health

Neighborhood SES may be an important contextual-level causal factor for many health outcomes, including overweight/obesity,[57-62] type 2 diabetes,[55, 63, 64] and smoking.[65-73] Although it has been established that individuals living in disadvantaged neighborhoods (e.g. low-income neighborhoods) experience worse health,[4-8] these relationships are worthy of continued attention as they may change over time[74] and especially as new analytical techniques are developed that may provide stronger evidence for causal inference.

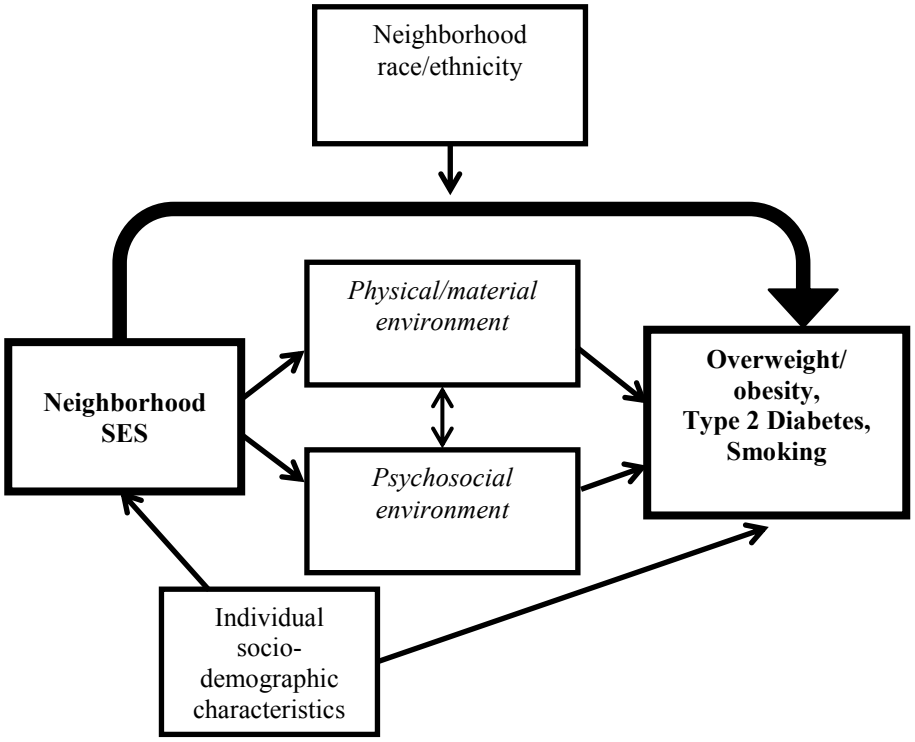
Neighborhood SES captures social sorting or stratification of individuals by social class. This stratification leads to differential access to social and material resources, where those in higher SES neighborhoods have more beneficial resources,[5, 6, 75] while those residing in lower SES neighborhoods have fewer resources and experience worse health.[4-8] SES can be measured is different ways; however, educational attainment and income are both strong, consistent single variable measures of SES,[76, 77] which also provide a clear point for intervention.[77] One limitation of using single variable measures is that they may not capture the complexity of SES as well as a composite measure or

index.[24, 77] Individuals are also sorted or segregated into neighborhoods by race/ethnicity; however, stratification by both neighborhood SES and neighborhood race/ethnicity together (i.e. examining race/ethnicity as an effect modifier of SES) may prohibit identification of the effects of one independent of the other.[55] This is in part due to the decreased probability of finding exchangeable populations when stratifying in the model by both neighborhood SES and neighborhood race/ethnicity.

Figure 1 provides a conceptual framework for this dissertation, which draws from several previous frameworks derived from the more general social-ecological framework.[78, 79] Neighborhood-level SES is hypothesized to affect health through access to physical and psychosocial resources, independent of individual-level socio-demographic characteristics, and possibly modified by neighborhood race/ethnicity. The individual-level characteristics are potential confounders, which are individual-level predictors of exposure to neighborhood SES, and include SES, demographic, and race/ethnicity variables.[32, 55] Based on the individual-level variables available from CHIS, this study accounted for: age, gender, household income, poverty (above/below 200% of federal poverty line), education (<high school; high school; some post-secondary; bachelor's degree or higher), African American, white, Hispanic, Asian, marital status (married or living with partner; separated, divorced, never married), employment (employed; unemployed), and home ownership (own; rent or other arrangement). This framework assumes that individual-level characteristics are not affected by neighborhood SES, which is a questionable assumption that we address later in this dissertation. As noted above this study does not include neighborhood race/ethnicity as an effect

modifier, due to the difficulty of identifying independent effects of neighborhood SES and neighborhood race/ethnicity.

Figure 1: Conceptual Framework



2.5 Comparing Social and Spatial Epidemiology Perspectives

Although social and spatial epidemiologists both ask contextual effects questions, they approach answering these questions with different perspectives, which results in differing theoretical foundations, analytical techniques, and potentially different conclusions. Comparing their perspectives may yield new insights that can strengthen causal inference in contextual effects studies. Indeed, Kawachi previously issued the call to make social epidemiology "the equivalent of an economic free zone[…] where enterprising investigators can shed their disciplinary baggage at the border, set up shop, and fruitfully exchange their ideas to enrich the field of the social determinants of health" (p.1741).[3] Likewise, geographers are also actively promoting interdisciplinary work between their field and public health. The recent 2013 Association of American Geographers Annual Meeting included a "Symposium on Geography, GIScience, and Health" with 38 sessions.

Directly comparing the social epidemiology and spatial epidemiology or medical geography sub-disciplines may be challenging. As Kuhn's theory of incommensurability suggests, we should not expect direct translations between the work of social epidemiologists and spatial epidemiologists.[80, 81] Referring to incommensurability Chen notes: "scientists from rival paradigms usually have different understandings of the goals of science and frequently have conflicting interests in the development of science" (p.264). Yet through comparisons we may still gain new insights into contextual effects and enhance effect identification – perhaps by blending cutting edge approaches from

each sub-discipline into the other. More generally, we may provide insight into how other social and spatial researchers can better cross the divide between disciplines.

Despite some work comparing social and spatial analytical techniques and results with each other or with a traditional multilevel model,[39, 41, 42, 48, 49, 82-84] to our knowledge no other study has explicitly compared the social epidemiology perspective with the spatial epidemiology perspective. Furthermore, we know of no study using cutting edge analytical techniques when comparing both approaches. Previous studies do suggest that taking a social or spatial approach compared with a more traditional multilevel regression approach may influence inference. Social epidemiology studies of neighborhood SES exposures have compared results using multilevel regression (or marginal maximal likelihood estimation) with those from propensity score matching analyses or regression analyses adjusting for propensity scores. These studies found little substantive differences in model results, suggesting that failure to address structural confounding in traditional multilevel regression may not necessarily produce unsupported model extrapolations.[55, 85, 86] However, they found some important differences when using propensity scores and propensity score matching, including wider confidence intervals due to the smaller sample sizes, considerably less overlap between comparison groups as the compared quartiles or tertiles grew further apart, and generally decreased estimated effects.[55, 85, 86] Studies comparing spatial analyses with multilevel regression analyses suggest that accounting for spatial dependence between neighborhoods aids model inference by providing more accurate estimates and standard errors.[39, 82]

These social and spatial studies provide examples of differences between traditional multilevel models and those used with a social or spatial approach. The present study moves beyond comparing analytical techniques and results to better understand the overarching social and spatial epidemiology perspectives. The ultimate goals of this comparison are to aid model interpretation and comparison, and hopefully improve identification of contextual effects in this and future studies.

2.6 Putting Contextual Effects in Context

This comparison of social and spatial epidemiology perspectives may help address the question of whether or not epidemiologists should even attempt to identify neighborhood effects from observational data. Oakes suggests this may be impossible due to a multitude of methodological challenges, which results in an identification problem.[2] The challenges of endogeneity and simultaneity are particularly troublesome and suggest that contextual effects are dependent on individual effects which are dependent on contextual effects and so on in a dynamic feedback loop.[2, 87] As part of this comparison, we separately attempt to address the challenges of structural confounding and spatial dependence in this study using social and spatial approaches, which may shed additional light on the surmountability of the identification problem.

Even if we can identify an effect the question remains whether or not we can actually intervene in a way that produces the intended effects. Contextual effects studies assume SUTVA; however, with dynamic feedback loops this is likely unrealistic. Thus, an intervention to move individuals to neighborhoods with a higher income or a higher percent of college graduates – as an intervention based on this study would suggest – may

not be effective. In the Moving to Opportunity (MTO) trial only some participants complied with their assigned treatment to move to a higher-SES neighborhood and others later moved backed to their original neighborhood.[88] The so-called white flight from neighborhoods after African Americans moved into the neighborhood is another example of the potential for unintended consequences. Specific to this study, the question is also can we expect to intervene on neighborhood SES on a broad scale, and given the great expense of MTO the answer would seem to be no. However, given our hypothesis that neighborhood SES affects access to salutogenic resources, identifying an effect of neighborhood SES could support community trials that increase neighborhood resources as a treatment.

Comparing the social and spatial epidemiology perspectives examining the effect of neighborhood SES on health outcomes may help us better address some challenges in observational contextual effects studies, while other challenges may remain insurmountable. Despite all of the challenges, work attempting to enhance identification of causal contextual determinants of health is meaningful but requires being mindful of the limitations inherent in these studies when interpreting and presenting results. Improved observational studies should provide a better starting point for community trials, where we can truly hope to get at causal contextual effects. Armed with experimental data from community trials, we will better inform public policy to intervene on major public health problems, such as obesity, type 2 diabetes, and smoking.

The following chapters lay out the social epidemiology perspective and analytical approach, the spatial epidemiology perspective and analytical approach, and a comparison of the two.

3. Social Epidemiology Perspective

Social stratification and structural confounding limit our ability to identify contextual effects on health in observational studies. Although observational neighborhood effects studies typically employ multilevel regression models to account for clustering by neighborhood, they fail to address the problems of stratification and structural confounding. Propensity score matching minimizes structural confounding and improves causal inference.

We employ propensity score matching to estimate the effect of moving from a lower to the next higher socioeconomic status (SES) neighborhood quartile on overweight/obesity, type 2 diabetes, and smoking. We approximate a real-world scenario by creating causal contrasts where those in a higher SES neighborhood quartile are matched with those in the next lower quartile. This approach yields more realistic policy estimates than most neighborhood effects studies.

Neighborhood SES is operationalized using census-tract level percent with a bachelor's degree of higher (neighborhood education) and median household income (neighborhood income) separately, which are classified into exposure quartiles. The sample includes San Francisco adults from the 2005, 2007, and 2009 California Health Interview Survey (CHIS) (n=2,515). We linked individual-level covariate and outcome data from CHIS with census-tract level SES data from the American Community Survey (ACS) 2006-2010 summary file.

Results suggest a significant effect of increasing neighborhood education on decreasing prevalence of overweight/obesity and smoking when moving from the

moderate-high to high neighborhood education quartile, using exchangeable exposure groups (i.e. independent of individual-level characteristics). Sensitivity analyses also suggest moving from a low to moderate-low neighborhood education quartile decreases overweight/obesity and moving from a moderate-low to moderate-high increases type 2 diabetes. However, results for overweight/obesity are quite sensitive to missing covariates and results for smoking are somewhat sensitive. We failed to find evidence of an effect for other comparisons using neighborhood education. All results using neighborhood median household income as the exposure were non-significant with estimates close to zero. The many challenges of observational neighborhood effects studies limit our ability to truly identify causal effects, but this study addresses one key challenge by using propensity score matching with policy-relevant causal contrasts.

3.1 Introduction

Identifying and intervening on contextual-level determinants of health may help combat major public health problems, such as overweight/obesity,[9-12] type 2 diabetes,[13, 14] and smoking.[15, 16, 89] Although the results of neighborhood effects studies (also known as contextual, place, or area effects studies) conducted over the past few decades suggest that neighborhood or contextual factors affect health,[4, 8, 17, 24] most studies have had a limited ability to estimate causal effects due to methodological challenges.[2] Critical among these challenges are social stratification and structural confounding, which may bias results.[2, 8, 29, 30, 33] Propensity score matching methods together with a counterfactual causal framework explicitly addresses social stratification and structural confounding and aims to mimic an experimental study using observational data.[30, 90] This offers a potential improvement for causal inference over regression analyses that adjust for individual-level differences in an attempt to identify contextual effects from observational data.[8, 31]

3.1.1 Social Stratification and Structural Confounding

Theory on social organization and structure argues that social processes segregate and sort individuals by social class and/or by race/ethnicity, so that different types of people live in different types of neighborhoods.[4, 40] This social stratification by neighborhood creates differential access to social and material resources, and a higher SES neighborhood has more beneficial resources for its inhabitants.[6, 75, 91] Evidence suggests that residents of neighborhoods with fewer social and/or material resources experience worse health.[4-8]

An important challenge when attempting to identify contextual causal factors occurs when individuals are stratified to the extent that similar people fail to exist across the different neighborhood exposure levels. This creates structural confounding, an absence of data to support inference.[29-31] Structural confounding cannot be solved by simply adding more data (i.e. observations), as this merely adds more of the same type of individuals to the same types of neighborhoods. When similar individuals do not reside in different neighborhoods, this violates the positivity assumption and fails to provide exchangeable groups for causal analyses, severely limiting our ability to make unbiased and supportable causal inferences.[1, 2, 8, 29, 32, 33] Given the complexity of the real world, this identification problem is a major challenge in observational studies when we want to tease apart contextual effects from underlying differences in individual-level characteristics across exposure groups.[33]

Structural confounding is common in neighborhood effects studies, which typically use regression analysis; however, regression masks this problem.[29, 30] When adjusting for covariates in regression analysis to make the populations of different neighborhoods comparable but for the exposure of interest, individuals are stratified into more and more specific strata (e.g. high-income, highly-educated, white, female). As the strata become more specific, it becomes increasingly unlikely that participants from the same strata could be found in both the exposed and unexposed groups.[2, 8] Regression does not address this structural confounding and inferences made in this situation are unsupported.

3.1.2 Propensity Score Matching

By comparison, propensity score matching offers a more robust approach by increasing exchangeability, thereby diminishing the identification problem and better approximating an experimental design.[30, 90, 92] Propensity score matching first calculates one's propensity to be exposed based on a set of covariates, each of which is associated with the exposure. Then in an attempt to mimic randomization to the treated and untreated groups (i.e. exposed and unexposed), the propensity scores are used to match observations across the exposure groups. This matches individuals that are similar but for their neighborhood exposure. After the matching process only "on-support" observations (i.e. those exposed individuals with an unexposed match) are retained for analysis, thereby limiting the potential for structural confounding.

3.1.3 Neighborhood Socioeconomic Status

Neighborhood SES may be an important contextual-level determinant of numerous health outcomes, including overweight/obesity,[57-62] type 2 diabetes,[55, 63, 64] and smoking.[65-73] Following from the Weberian tradition of SES studies which focus on the differences in accumulation of economic resources, knowledge, and skills,[76, 93] one's neighborhood SES is hypothesized to affect one's health by enabling or constraining access to social and material capital.[77] Despite a more recent shift toward examining specific hypothesized mechanisms on the causal pathway between neighborhood SES and health outcomes,[1, 4, 94] neighborhood SES itself remains of interest. Many stress the importance of examining "fundamental causes of disease" or "cause of causes" (e.g. neighborhood SES).[22, 95, 96] Intervening on these fundamental causes may have a greater effect on improving health outcomes than intervening on a specific downstream

mechanism. Furthermore, disentangling specific mechanisms on the causal pathway is complicated by the high correlation of social variables.[77]

Considerable debate exists over when to use a single variable versus a composite measure for SES. While some suggest that single measures should be used only when examining questions of a single variable (e.g. education),[97] others suggest that single variables may detect magnitudes of health comparable to indices.[98] Single variable measures of neighborhood SES provide a clear point for intervention, are easier to obtain,[77] and identify the contribution of the variable.[24] Drawbacks as compared to using composite measures include increased measurement error, as a single measure does not capture the full complexity of SES.[24, 77]

Educational attainment and income each provide strong, consistent proxy measures of SES.[76, 77] Higher education captures increased access to human, social, and environmental capital.[76, 77, 93] Educational attainment is easy to measure, relatively stable after age 25, and people are typically willing to provide it; all of which limit missing data and measurement bias.[77] Income measures access to material capital or conditions,[76, 77, 93] and with this typically comes better health.[93] Income is scalar and easy to measure, but is subject to participant nonresponse and misreport of actual income.[77] Household income is preferable to individual income as it may better capture the entire material resources available to an individual.[93] While poverty is often used as a univariate measure of SES, compared with income (from which it is derived) poverty is a coarser measure[77] that assumes a threshold and is based on the federal poverty level, which fails to account for cost of living and family type differences.[99]

Propensity scores are derived using predictors of the exposure only. Determinants of neighborhood SES include individual-level SES, demographic, and race/ethnicity variables.[32, 55]

3.1.4 Summary

In this study, we estimated the effect of increasing neighborhood SES (percent with bachelor's degree or higher and median household income) on individual-level overweight/obesity, type 2 diabetes, and smoking, accounting for differences in key individual-level characteristics. By using a counterfactual causal framework with propensity score matching methods we approximate an experimental design and increase the exchangeability of observations across the exposure levels. We hypothesized that moving from a lower to the next higher-SES quartile neighborhood improves one's health (decreases overweight/obesity, type 2 diabetes, and smoking), independent of one's individual-level characteristics.

3.2 Methods

This study uses a cross-sectional design, propensity score matching, and a counterfactual causal framework to ask whether an increase in neighborhood SES (either education level or median household income) decreased overweight and obesity, type 2 diabetes, or smoking, independent of individual-level differences, in San Francisco adults.

3.2.1 Data Sources

To address our study question we used cross-sectional data on San Francisco adults from two sources: 1) 2005, 2007, and 2009 California Health Interview Surveys

(CHIS)[100-102] (n=2,515), and 2) 2006-2010 5-year Summary File from the American Community Survey (ACS) for census tracts.[103] The CHIS data provided individual-level outcome and covariate data, which was linked to census tract (i.e. neighborhood-level) exposure data from ACS using latitude and longitude for each individual's residential address. A pooled dataset from the 2005, 2007, and 2009 CHIS surveys roughly overlaps with the ACS data which are averaged across 2006-2010.

CHIS surveys provide a representative sample of San Francisco County obtained using random digit dialing.[104] Given the challenges of conducting surveys by telephone, CHIS provides reasonable response rates that were similar to the California Behavioral Risk Factor Surveillance Survey (BRFSS) for 2005 and 2007, although somewhat lower for 2009.[105-107] Missing values were imputed by CHIS staff for most variables, although the amount of missing data was generally quite low (<2% of the sample), with some exceptions such as household income where missing data exceeded 20% in 2005 and 2007, and 25% in 2009.[108-110]

With the discontinuation of the US Census decennial long form, ACS 5-year summary files are the new standard for sociodemographic data for neighborhood studies. The ACS data are likely subject to the same limitations as other census data: undercounts of certain population segments (e.g. minorities)[97] and reduced accuracy of data at smaller geographical aggregation units to maintain confidentiality.[111] The benefits of ACS summary data include readily available, timely data collected by trained professionals at small geographic units (i.e. block groups and census tracts).

3.2.2 Outcome Variables

This study examines three dichotomous outcome variables obtained using self-report: overweight/obesity, type 2 diabetes, and smoking. Overweight/obesity was measured using body mass index (BMI)(kg/m^2) derived from height and weight measures, and includes those who are overweight (BMI=25.0-29.9) or obese (BMI ≥30.0).[9] Type 2 diabetes was measured using two questions: 1) doctor ever told that had diabetes, and 2) type 1 or 2 diabetes. Smoking was derived from several questions on current and past cigarette smoking, as well as frequency of use. Smoking behavior is dichotomized into current smokers compared with former or never smokers. Self-reported measures for type 2 diabetes and smoking behavior have been shown to have high validity.[112, 113] BMI from self-reported height and weight is generally significantly underestimated for overweight and obese adults; however, adjustment for age and other socio-demographic characteristics reduces the likelihood of bias due to measurement error.[114, 115]

3.2.3 Exposure Variables

The exposure of interest is neighborhood SES. This is examined using two separate measures: neighborhood median household income and percentage of the population aged 25 years or older with a bachelor's degree education or higher in the neighborhood. Each exposure was classified into quartiles based on the San Francisco distribution from the 2006-2010 ACS data (by 2010 census tract), with quartile 1 representing lowest neighborhood SES and quartile 4 highest. *A priori* cutpoints would provide a consistent measure for comparison with other studies, but both cutpoints and quartiles detect socioeconomic gradients.[98] Furthermore, San Francisco is more highly

educated and wealthier than the country as a whole, so *a priori* cutpoints may fail to provide sufficient samples in different exposure groups for this study area.

Census tracts are the neighborhood unit for this study. Although census tracts may create artificial neighborhood boundaries, they are commonly used in neighborhood studies. Tracts make a good proxy for a neighborhood given the relative sociodemographic homogeneity of the approximately 2,500-8,000 people in each tract.[116] Research suggests that census tracts capture neighborhood processes similarly to block groups and better than zip codes.[98] Given that our neighborhood-level data is aggregated to the tract, defining neighborhoods using a buffer of a certain distance around each individual's residence that cuts across tracts would require extraordinary assumptions, i.e. that the characteristics of the section of the tract included in the neighborhood are represented by those of the entire tract.

3.2.4 Covariates

Propensity score analyses account for predictors of the exposure of interest that occur before the outcome to increase the exchangeability of participants.[117] Based on social stratification into neighborhoods and data availability, the following individual-level covariates from CHIS were used to calculate the propensity score for all outcomes: age, gender, household income, poverty (above/below 200% of federal poverty line), education (<high school; high school; some post-secondary; bachelor's degree or higher), African American, white, Hispanic, Asian, marital status (married or living with partner; separated, divorced, never married), employment status (employed; unemployed), and home ownership (own; rent or other arrangement).

3.2.5 Analytical Models and Procedures

We used propensity score matching to estimate the average effect of higher neighborhood SES (income and education) on overweight/obesity, type 2 diabetes, and smoking in those who were actually exposed to higher neighborhood SES (i.e. average effect of the treatment on the treated [ATT]). For each neighborhood exposure we examined three separate higher vs. lower neighborhood SES causal contrasts: Quartile 2 vs. 1; Quartile 3 vs. 2; and Quartile 4 vs. 3. By limiting the causal contrasts to adjacent exposure quartiles, this better simulates a real-world policy intervention. Moving an individual to a one-higher neighborhood SES quartile is far more tenable than moving them from the first to the fourth quartile. Typically studies split a sample into one exposed group and one unexposed group, or compare all lower groups to a single high group (or vice versa).

A propensity score, a conditional probability of having been exposed, was calculated for each individual using a logistic regression model, where the covariates predictive of the neighborhood exposure are the exposures and the neighborhood exposure is the outcome.[30, 90, 118] We examined the propensity score overlap between those individuals exposed to higher neighborhood SES and those unexposed (i.e. exposed to lower neighborhood SES) to ensure exchangeability across exposure groups. Next, an exposed individual was matched with replacement to an unexposed individual with a similar propensity score using the nearest neighbor within a +/- 0.02 caliper approach. The chosen caliper of .02 was more precise than the suggested standard caliper (standard deviation*.25)[90, 119] for all causal contrasts. We examined standardized differences post-

match to ensure adequate balance (i.e. standardized difference <10)[120, 121] across exposure groups on the covariates.[32, 120] Finally, we estimated the ATT and used bootstrap with 1000 repetitions to estimate the corresponding standard errors and 95% confidence intervals.[30] ATT estimates the effect of the actual treatment or exposure,[92] or in other words ATT examines if the treatment is beneficial to those who are or would be treated.[36, 90] Propensity score calculations were conducted in Stata using the PSMATCH2 module.[122] Sensitivity analyses also examined differences due to caliper width and matching without replacement. The RBOUNDS module[123] in Stata was also used to determine the Rosenbaum bounds to evaluate the sensitivity of the estimates to unmeasured covariates. StataSE 12.1 was used for all analyses.

The University of Minnesota Institutional Review Board (IRB) determined that this study is exempt from full IRB review given that we could only access de-identified data. The Data Access Center (DAC) at the UCLA Center for Health Policy Research was approved by the UCLA South General Institutional Review Board to conduct analyses of confidential CHIS data (UCLA IRB #G09-05-103-01).

3.3 Results

Tables 1A and 1B provide characteristics of the study sample by neighborhood SES quartile (education and household income respectively) and for the entire sample. Differences exist in individual-level characteristics and health outcomes across neighborhood exposure quartiles for both exposures. As anticipated the lower neighborhood SES quartiles generally have higher percentages of minorities and unemployed, less-educated, and lower-income individuals. Although the health outcomes

in most cases appear to improve (i.e. decrease) with increasing neighborhood SES, this unadjusted relationship is inconsistent.

Histograms (Figures 2-7) show substantial overlap between the exposed and unexposed groups based on propensity scores. Tables 2A and 2B show the covariate imbalance across exposure groups pre- and post-matching for each causal contrast. An acceptable level of covariate balance (standardized difference <10) was achieved post-match for all covariates in all causal contrasts, with the exception of unemployment for the neighborhood education Quartile 2 vs. 1 contrast. Given our desire to have a consistent set of variables across all analyses and that the difference (12.2) does not vastly exceed an acceptable level, we retained the unemployment variable in this contrast. Both the histograms and the covariate balances suggest that the exposed and unexposed populations are relatively exchangeable and structural confounding is likely limited.

Tables 3A and 3B together with Figures 8-13 show the difference in ATT for neighborhood education and income for each causal contrast for overweight/obesity, type 2 diabetes, and smoking. The results suggest a significant negative effect only when moving from moderate-high (Q3) to highly educated (Q4) neighborhoods for overweight/obesity (-0.1042, 95% CI: -0.2022 to -0.0314) and smoking behavior (-0.0545, 95% CI: -0.1191 to -0.0266). The estimated ATT difference for overweight/obesity suggests that moving from Q3 to Q4 results in 10 fewer individuals who are overweight or obese per 100 individuals. The estimated ATT difference for smoking suggests a somewhat weaker effect for the same causal contrast with a decrease of 5 individuals who are current smokers per 100 individuals. No significant effects were

Table 1A: Sample Characteristics by Quartile of Census Tract Percentage with Bachelor's Degree or Higher and Total Sample, San Francisco Adults, Source: CHIS 2005, 2007, 2009.

	Neighborhood % Population with Bachelor's Degree or Higher				
	Quartile 1 (n=433)	Quartile 2 (n=700)	Quartile 3 (n=754)	Quartile 4 (n=628)	Total Sample (n= 2,515)
Mean age (SD)	51.7 (17.3)	53.5 (18.4)	53.4 (16.5)	53.4 (16.8)	53.1 (17.3)
Male gender	191 (44.1%)	300 (42.9%)	334 (44.3%)	295 (47.0%)	1120 (44.5%)
Race/Ethnicity					
African American	70 (16.2%)	40 (5.7%)	46 (6.1%)	16 (2.6%)	172 (6.8%)
Asian	187 (43.2%)	241 (34.4%)	138 (18.3%)	98 (15.6%)	664 (26.4%)
Hispanic/Latino	65 (15.0%)	75 (10.7%)	46 (6.1%)	30 (4.8%)	216 (8.6%)
White	142 (32.8%)	384 (54.9%)	565 (74.9%)	508 (80.9%)	1599 (63.6%)
Mean household income $ (SD)	53,744 (77,931)	66,744 (72,555)	105,902 (122,503)	134,042 (146,242)	93,050 (115,170)
Below 200% federal poverty rate	192 (44.3%)	226 (32.3%)	106 (14.1%)	54 (8.6%)	578 (23.0%)
Education					
< 12th grade	66 (15.2%)	82 (11.7%)	30 (4.0%)	15 (2.4%)	193 (7.7%)
12th grade/high school diploma	119 (27.5%)	116 (16.6%)	69 (9.2%)	34 (5.4%)	338 (13.4%)
Some post-secondary	113 (26.1%)	146 (20.9%)	141 (18.7%)	88 (14.0%)	488 (19.4%)
BA/BS or above	135 (31.2%)	356 (50.9%)	514 (68.2%)	491 (78.2%)	1496 (59.5%)
Unmarried, not living with partner	218 (50.4%)	393 (56.1%)	396 (52.5%)	324 (51.6%)	1331 (52.9%)
Unemployed	188 (43.4%)	294 (42.0%)	271 (36.0%)	192 (30.6%)	945 (37.6%)
Rent	237 (54.7%)	417 (59.6%)	357 (47.4%)	329 (52.4%)	1340 (53.3%)
Overweight or Obese	217 (50.1%)	282 (40.3%)	357 (47.4%)	240 (38.2%)	1096 (43.6%)
Type 2 diabetes	39 (9.0%)	48 (6.9%)	46 (6.1%)	31 (4.9%)	164 (6.5%)
Current smoker	50 (11.6%)	92 (13.1%)	90 (11.9%)	49 (7.8%)	281 (11.2%)

Quartile 1: 6%-34%; Quartile 2: >34%-54%; Quartile 3: >54%-69%; Quartile 4: >69%-100%.

Table 1B: Sample Characteristics by Quartile of Census Tract Median Household Income and Total Sample, San Francisco Adults, Source: CHIS 2005, 2007, 2009.

	Neighborhood Median Household Income				
	Quartile 1 (n=512)	Quartile 2 (n=594)	Quartile 3 (n=730)	Quartile 4 (n=679)	Total Sample (n= 2,515)
Mean age (SD)	53.0 (18.4)	51.6 (17.9)	52.8 (16.5)	55.0 (16.5)	53.1 (17.3)
Male gender	232 (45.3%)	252 (42.4%)	321 (44.0%)	315 (46.4%)	1120 (44.5%)
Race/Ethnicity					
African American	75 (14.7%)	51 (8.6%)	32 (4.4%)	14 (2.1%)	172 (6.8%)
Asian	165 (32.3%)	205 (34.5%)	196 (26.9%)	98 (14.4%)	664 (26.4%)
Hispanic/Latino	51 (10.0%)	63 (10.6%)	62 (8.5%)	40 (5.9%)	216 (8.6%)
White	252 (49.2%)	311 (52.4%)	477 (65.3%)	559 (82.3%)	1599 (63.6%)
Mean household income (SD)	51,887 (76,201)	79,647 (100,261)	95,269 (109,007)	133,429 (141,998)	93,050 (115,170)
Below 200% federal poverty rate	222 (43.4%)	162 (27.3%)	132 (18.1%)	62 (9.1%)	578 (23.0%)
Education					
< 12th grade	68 (13.3%)	58 (9.8%)	48 (6.6%)	19 (2.8%)	193 (7.7%)
12th grade/high school diploma	107 (20.9%)	107 (18.0%)	82 (11.2%)	42 (6.2%)	338 (13.4%)
Some post-secondary	112 (21.9%)	134 (22.6%)	129 (17.7%)	113 (16.6%)	488 (19.4%)
BA/BS or above	225 (44.0%)	295 (49.7%)	471 (64.5%)	505 (74.4%)	1496 (59.5%)
Unmarried, not living with partner	332 (64.8%)	299 (50.3%)	377 (51.6%)	323 (47.6%)	1331 (52.9%)
Unemployed	250 (48.8%)	203 (34.2%)	240 (32.9%)	252 (37.1%)	945 (37.6%)
Rent	379 (74.0%)	334 (56.2%)	365 (50.0%)	262 (38.6%)	1340 (53.3%)
Overweight or Obese	237 (46.3%)	260 (43.8%)	305 (41.8%)	294 (43.3%)	1096 (43.6%)
Type 2 diabetes	44 (8.6%)	39 (6.6%)	47 (6.4%)	34 (5.0%)	164 (6.5%)
Current smoker	83 (16.2%)	69 (11.6%)	74 (10.1%)	55 (8.1%)	281 (11.2%)

Quartile 1: $11,513-55,079; Quartile 2: >$55,079-76,597; Quartile 3: >$76,597-94,479; Quartile 4: >$94,479-155,099+.

Figure 2: Overlap of Exposed (i.e. Treated) and Unexposed (i.e. Untreated) Observations - Neighborhood Education Q2 vs. Q1

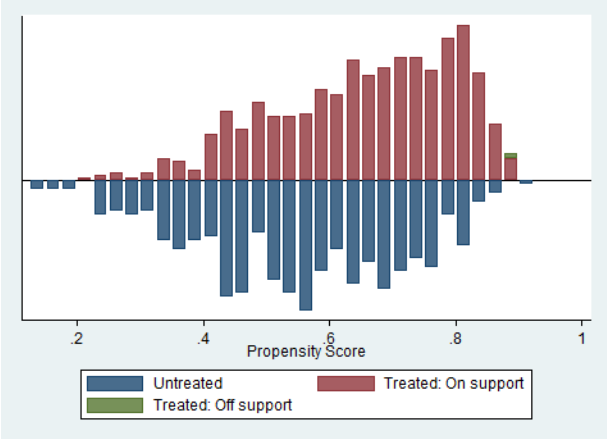


Figure 3: Overlap of Exposed (i.e. Treated) and Unexposed (i.e. Untreated) Observations - Neighborhood Education Q3 vs. Q2

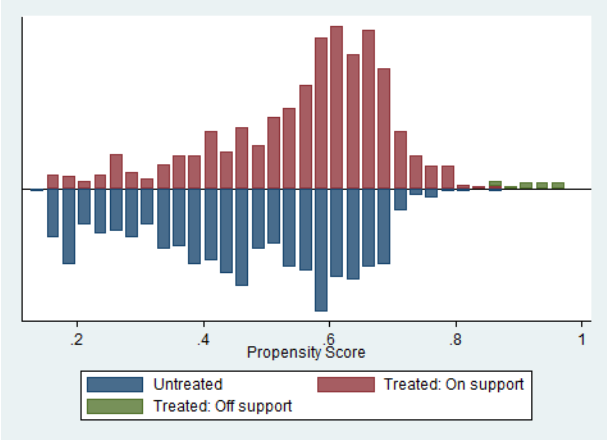


Figure 4: Overlap of Exposed (i.e. Treated) and Unexposed (i.e. Untreated) Observations - Neighborhood Education Q4 vs. Q3

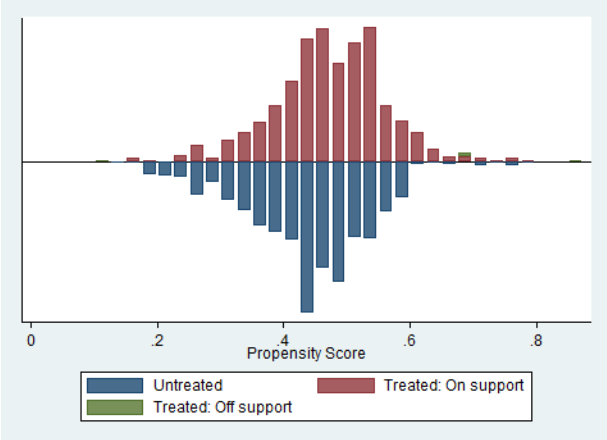


Figure 5: Overlap of Exposed (i.e. Treated) and Unexposed (i.e. Untreated) Observations - Neighborhood Income Q2 vs. Q1

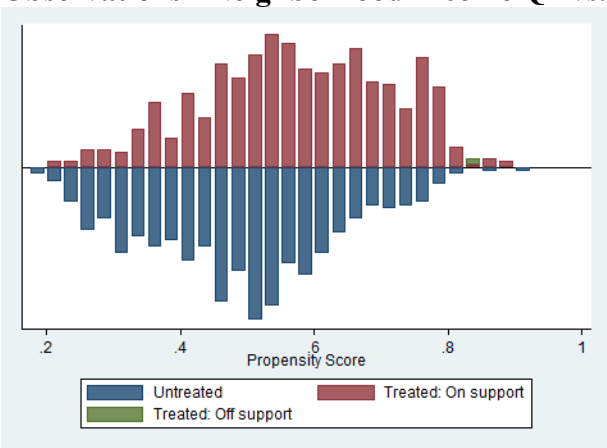


Figure 6: Overlap of Exposed (i.e. Treated) and Unexposed (i.e. Untreated) Observations - Neighborhood Income Q3 vs. Q2

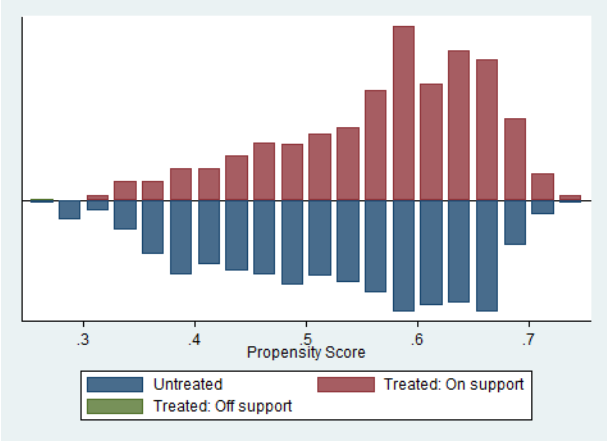


Figure 7: Overlap of Exposed (i.e. Treated) and Unexposed (i.e. Untreated) Observations - Neighborhood Income Q4 vs. Q3

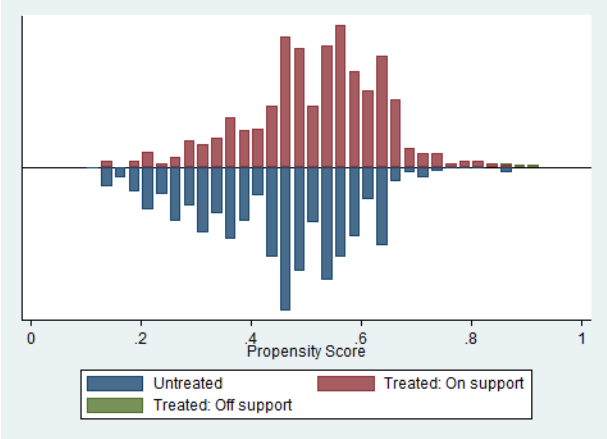


Table 2A: Covariate Imbalance Across Neighborhood Education Exposure Groups Pre- and Post-Matching for San Francisco Adults, 2005, 2007, and 2009.

	Moderate-low vs. Low Neighborhood % with Bachelor's Degree or Higher (Q2 vs Q1)					Moderate-high vs. Moderate-low Neighborhood % with Bachelor's Degree or Higher (Q3 vs Q2)					High vs. Moderate-high Neighborhood % with Bachelor's Degree or Higher (Q4 vs Q3)				
	Post-match mean		Standardized Difference			Post-match mean		Standardized Difference			Post-match mean		Standardized Difference		
	Q2 adults	Q1 adults	Pre-Match	Post-Match	% Bias Reduction	Q3 adults	Q2 adults	Pre-Match	Post-Match	% Bias Reduction	Q4 adults	Q3 adults	Pre-Match	Post-Match	% Bias Reduction
Mean age	53.480	52.805	10.0	3.8	62.3	53.556	52.574	-0.8	5.6	-563.2	53.394	53.176	0.0	1.3	-19298.7
Male gender	0.42980	0.39255	-2.5	7.5	-197.1	0.43725	0.47368	2.9	-7.3	-153.0	0.46955	0.47917	5.4	-1.9	64.1
African American	0.05731	0.06447	-33.9	-2.3	93.1	0.06073	0.05668	1.6	1.7	-4.7	0.02404	0.02244	-17.5	0.8	95.5
Asian	0.34527	0.33524	-18.0	2.1	88.5	0.18623	0.21727	-37.2	-7.2	80.8	0.15545	0.17147	-7.2	-4.3	40.6
Hispanic/Latino	0.10745	0.12751	-12.8	-6.0	53.3	0.06208	0.05263	-16.7	3.4	79.5	0.04808	0.04006	-5.8	3.5	39.5
White	0.54728	0.55731	45.6	-2.1	95.5	0.74494	0.71930	43.0	5.5	87.2	0.80769	0.80609	14.4	0.4	97.3
Mean household income $	66,742	73,860	17.3	-9.5	45.2	93,927	99,399	38.9	-5.4	86.0	131,488	128,466	20.9	2.2	89.3
Below 200% federal poverty rate	0.32378	0.34241	-25.0	-3.9	84.6	0.14305	0.12955	-44.2	3.3	92.6	0.08654	0.08013	-17.3	2.0	88.3
Education															
< 12th grade	0.11748	0.11891	-10.3	-0.4	95.9	0.04049	0.03104	-29.0	3.5	87.8	0.02404	0.03526	-9.1	-6.4	29.5
12th grade/high school diploma	0.16619	0.15330	-26.5	3.1	88.2	0.09312	0.08637	-22.3	2.0	90.9	0.05449	0.04167	-14.4	4.9	65.7
Some post-secondary	0.20917	0.20057	-12.4	2.0	83.6	0.18893	0.20513	-5.4	-4.1	24.9	0.13942	0.13942	-12.7	0.0	100.0
BA/BS or above*															
Unmarried, not living with partner	0.56017	0.52865	11.6	6.3	45.6	0.53036	0.49123	-7.3	7.9	-8.0	0.51282	0.51603	-1.9	-0.6	65.4
Unemployed	0.41834	0.47851	-2.9	**-12.2**	-324.3	0.36302	0.36707	-12.4	-0.8	93.3	0.30609	0.30449	-11.4	0.3	97.0
Rent	0.59456	0.60458	9.8	-2.0	79.3	0.47908	0.48718	-24.7	-1.6	93.4	0.52404	0.52724	10.1	-0.6	93.6

*Reference category

Table 2B: Covariate Imbalance Across Neighborhood Income Exposure Groups Pre- and Post-Matching for San Francisco Adults, 2005, 2007, and 2009.

	Moderate-low vs. Low Neighborhood Median Household Income (Q2 vs Q1)					Moderate-high vs. Moderate-low Neighborhood Median Household Income (Q3 vs Q2)					High vs. Moderate-high Neighborhood Median Household Income (Q4 vs Q3)				
	Post-match mean		Standardized Difference			Post-match mean		Standardized Difference			Post-match mean		Standardized Difference		
	Q2 adults	Q1 adults	Pre-Match	Post-Match	% Bias Reduction	Q3 adults	Q2 adults	Pre-Match	Post-Match	% Bias Reduction	Q4 adults	Q3 adults	Pre-Match	Post-Match	% Bias Reduction
Mean age	51.628	51.166	-7.5	2.6	65.8	52.829	53.136	7.1	-1.8	75.0	54.994	54.235	12.9	4.6	64.3
Male gender	0.42399	0.40203	-5.8	4.4	24.0	0.44033	0.39506	3.1	9.1	-192.4	0.46450	0.46893	4.9	-0.9	81.7
African American	0.08615	0.09122	-19.0	-1.6	91.6	0.04252	0.04527	-17.1	-1.1	93.5	0.02071	0.01036	-13.2	5.9	55.4
Asian	0.34628	0.33277	4.8	2.9	40.9	0.26886	0.27709	-16.7	-1.8	89.3	0.14497	0.15089	-31.0	-1.5	95.2
Hispanic/Latino	0.10642	0.12500	2.1	-6.1	-188.0	0.08505	0.09328	-7.2	-2.8	61.0	0.05917	0.07396	-10.1	-5.7	43.2
White	0.52196	0.52365	6.3	-0.3	94.6	0.65432	0.63786	26.6	3.4	87.3	0.82249	0.81953	39.4	0.7	98.3
Mean household income $	77,889	77,233	31.2	0.7	97.6	95,382	92,391	14.9	2.9	80.9	129,583	129,796	30.1	-0.2	99.4
Below 200% federal poverty rate	0.27365	0.29392	-34.1	-4.3	87.4	0.17970	0.15912	-22.1	4.9	77.6	0.09172	0.10059	-26.3	-2.6	90.1
Education															
< 12th grade	0.09797	0.09628	-11.0	0.5	95.2	0.06584	0.08230	-11.7	-6.0	48.4	0.02811	0.03254	-17.9	-2.1	88.3
12th grade/high school diploma	0.18074	0.15034	-7.3	7.7	-5.4	0.11111	0.10425	-19.3	1.9	89.9	0.06213	0.04734	-18.0	5.3	70.7
Some post-secondary	0.22466	0.22804	1.6	-0.8	50.6	0.17695	0.17147	-12.2	1.4	88.8	0.16716	0.17751	-2.7	-2.7	-0.6
BA/BS or above*															
Unmarried, not living with partner	0.50507	0.51182	-29.6	-1.4	95.3	0.51715	0.51440	2.6	0.5	79.0	0.47781	0.45562	-8.1	4.4	45.5
Unemployed	0.34122	0.31419	-30.0	5.5	81.6	0.32922	0.35254	-2.7	-4.9	-79.6	0.36834	0.38166	8.9	-2.8	68.6
Rent	0.56419	0.56926	-38.0	-1.1	97.2	0.49931	0.49383	-12.5	1.1	91.2	0.38609	0.40828	-23.1	-4.5	80.6

*Reference category

Table 3A: Effect Estimates of Neighborhood Tract Education on Overweight/Obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009.

Causal contrast	Number of matched pairs	Number exposed off-support	Rate of Outcome: Exposed to higher education neighborhood	Rate of Outcome: Unexposed to higher education neighborhood	ATT Difference	S.E.*	Lower 95% CI**	Upper 95% CI**
Overweight/Obesity								
Moderate-low vs. Low % with bachelor's degree or higher (Q2 vs. Q1)	698	2	0.40401	0.49785	-0.09384	0.04992	-0.19163	0.00294
Moderate-high vs. Moderate-low % with bachelor's degree or higher (Q3 vs Q2)	741	13	0.47368	0.42510	0.04858	0.04207	-0.03343	0.13182
High vs. Moderate-high % with bachelor's degree or higher (Q4 vs Q3)	624	4	0.38301	0.48718	-0.10417	0.04250	**-0.20218**	**-0.03142**
Type 2 Diabetes								
Moderate-low vs. Low % with bachelor's degree or higher (Q2 vs. Q1)	698	2	0.06877	0.08739	-0.01862	0.03011	-0.07525	0.03561
Moderate-high vs. Moderate-low % with bachelor's degree or higher (Q3 vs Q2)	741	13	0.06208	0.04723	0.01484	0.01848	-0.01776	0.04830
High vs. Moderate-high % with bachelor's degree or higher (Q4 vs Q3)	624	4	0.04968	0.04808	0.00160	0.01751	-0.03333	0.03509
Smoking								
Moderate-low vs. Low % with bachelor's degree or higher (Q2 vs. Q1)	698	2	0.13181	0.09169	0.04011	0.02651	-0.01158	0.08811
Moderate-high vs. Moderate-low % with bachelor's degree or higher (Q3 vs Q2)	741	13	0.11876	0.09717	0.02159	0.02642	-0.00687	0.08552
High vs. Moderate-high % with bachelor's degree or higher (Q4 vs Q3)	624	4	0.07853	0.13301	-0.05449	0.02519	**-0.11908**	**-0.02656**

*From bootstrap estimates.

**From bias-controlled bootstrap estimates.

Table 3B: Effect Estimates of Neighborhood Tract Income on Overweight/Obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009.

Causal contrast	Number of matched pairs	Number exposed off-support	Rate of Outcome: Exposed to higher income neighborhood	Rate of Outcome: Unexposed to higher income neighborhood	ATT Difference	S.E.*	Lower 95% CI**	Upper 95% CI**
Overweight/Obesity								
Moderate-low vs. Low neighborhood median household income (Q2 vs. Q1)	592	2	0.43750	0.45439	-0.01689	0.04876	-0.12034	0.06868
Moderate-high vs. Moderate-low neighborhood median household income (Q3 vs Q2)	729	1	0.41701	0.42181	-0.00480	0.04201	-0.06921	0.08078
High vs. Moderate-high neighborhood median household income (Q4 vs Q3)	676	3	0.43491	0.42751	0.00740	0.04115	-0.08533	0.07846
Type 2 Diabetes								
Moderate-low vs. Low neighborhood median household income (Q2 vs. Q1)	592	2	0.06588	0.08277	-0.01689	0.02230	-0.07521	0.00678
Moderate-high vs. Moderate-low neighborhood median household income (Q3 vs Q2)	729	1	0.06310	0.05350	0.00960	0.02063	-0.02260	0.05270
High vs. Moderate-high neighborhood median household income (Q4 vs Q3)	676	3	0.05030	0.06213	-0.01183	0.01993	-0.06107	0.02171
Smoking								
Moderate-low vs. Low neighborhood median household income (Q2 vs. Q1)	592	2	0.11655	0.11655	0.00000	0.03193	-0.04259	0.07770
Moderate-high vs. Moderate-low neighborhood median household income (Q3 vs Q2)	729	1	0.10014	0.10288	-0.00274	0.02567	-0.05328	0.04566
High vs. Moderate-high neighborhood median household income (Q4 vs Q3)	676	3	0.08136	0.08728	-0.00592	0.02320	-0.04977	0.03834

*From bootstrap estimates.

**From bias-controlled bootstrap estimates.

Figure 8: Average Effect of Treatment on the Treated (ATT) for Overweight/obesity

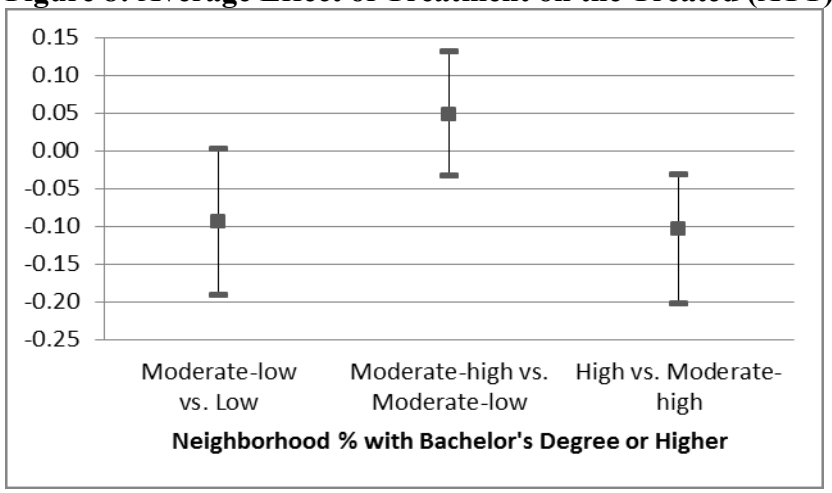


Figure 9: Average Effect of Treatment on the Treated (ATT) for Type 2 Diabetes

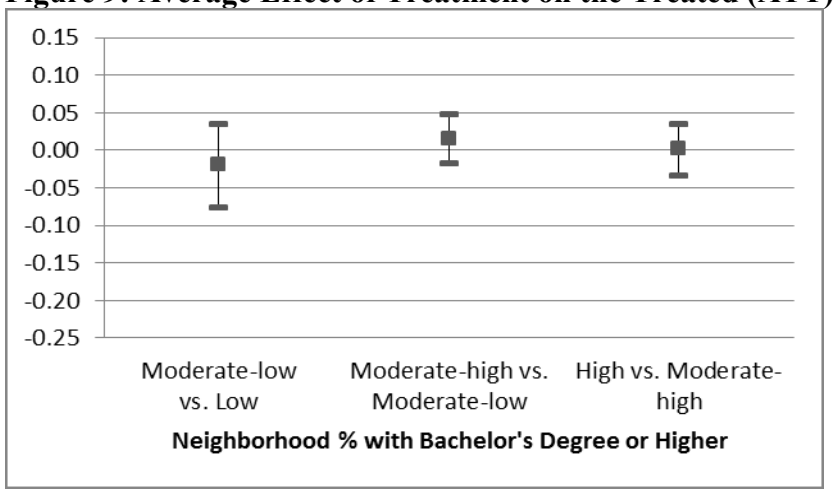


Figure 10: Average Effect of Treatment on the Treated (ATT) for Smoking

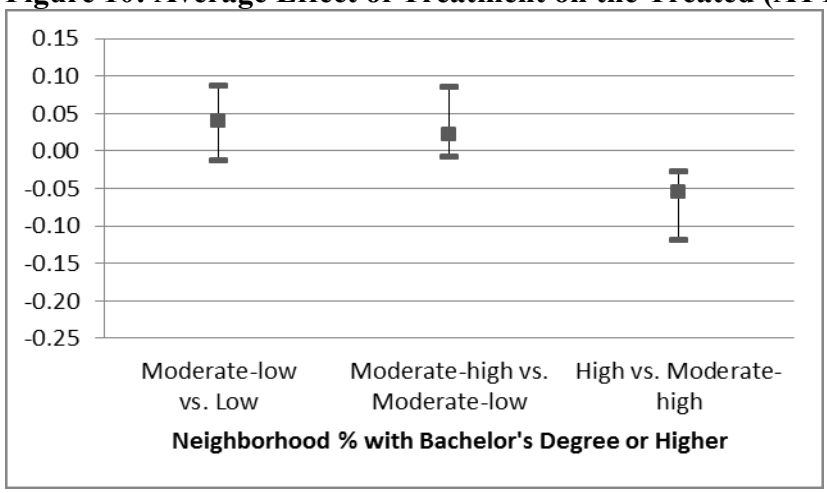


Figure 11: Average Effect of Treatment on the Treated (ATT) for Overweight/obesity

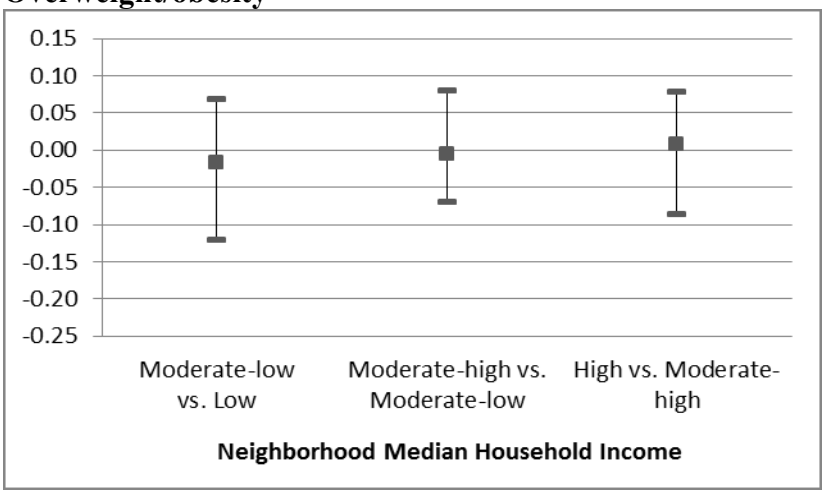


Figure 12: Average Effect of Treatment on the Treated (ATT) for Type 2 Diabetes

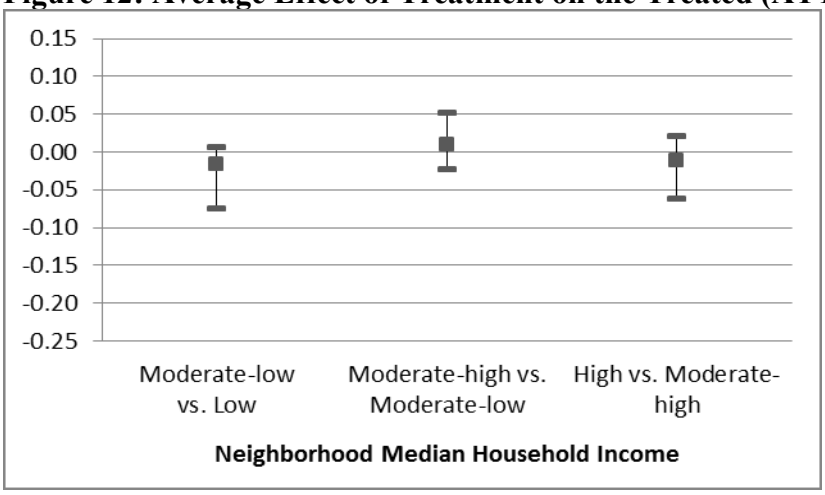
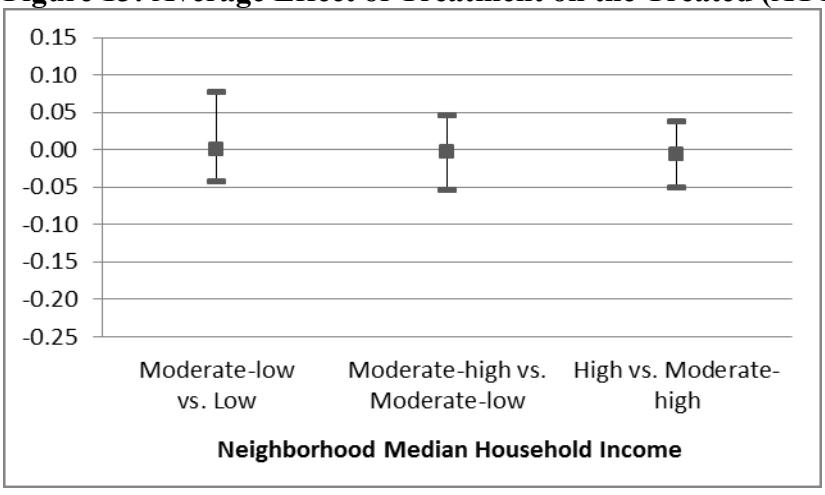


Figure 13: Average Effect of Treatment on the Treated (ATT) for Smoking



estimated for the neighborhood income exposure.

Sensitivity analyses conducted using more precise calipers of 0.01 and 0.015 resulted in few losses of on-support (i.e. exposed) observations (with the exception of a difference of 13 observations for the education Q4 vs. Q3 comparison), and thus unsurprisingly the results of the ATT analyses appear similar across calipers of 0.01, 0.015, and 0.02.

We also conducted sensitivity analyses using matching *without* replacement. Compared to matching *with* replacement, without replacement generated a substantially higher number of off-support treatment observations; however, this resulted in few substantive differences. The standardized differences in covariance balance between the exposed and unexposed groups were all under 10. Results using neighborhood education as the exposure were similar to matching with replacement; although two estimates achieved significance (overweight/obesity quartile 1 vs. 2 and diabetes quartile 2 vs. 3) and one was no longer significant (smoking quartile 3 vs. 4), each of these contrasts had either with or without replacement one bound of the confidence interval approaching zero. The estimate for overweight/obese for quartile 3 vs. 4 remained significant. Results using neighborhood income as the exposure found no meaningful differences between matching with and without replacement; all estimates failed to achieve significance.

The results of the Rosenbaum bounds procedure (not shown) to evaluate the sensitivity of estimates to missing covariates suggest the overweight/obesity estimates are highly sensitive, smoking estimates are somewhat sensitive, and the type 2 diabetes are relatively insensitive.[124]

3.4 Discussion

This study estimated a significant inverse effect of moving from a neighborhood with a moderate-high percent (Q3) to a neighborhood with a high percent (Q4) of residents with a bachelor's degree or higher on overweight/obesity and smoking behavior, but not on type 2 diabetes. For overweight/obesity and smoking, the significant effects may suggest that moving from a neighborhood with 55%-69% of adults with a bachelor's degree or higher to one with greater than 69% provides individuals with meaningful improvements in neighborhood resources which affect their health. Relatedly, a study of black women found that a negative association of obesity incidence over 10 years with neighborhood SES (based on an index) was strongest in college-educated women.[59] This may suggest that obtaining a college education (an individual-level characteristic) and moving to the most highly educated neighborhood (a neighborhood-level characteristic) both independently decrease obesity. In our study, for overweight/obesity the move from Q1 to Q2 approached significance (-0.0938, 95% CI: -0.1916 to 0.0029), and in a sensitivity analysis this contrast reached significance, which suggests a similar relationship.

The remaining results from the neighborhood education analyses were non-significant and some estimates were in the opposite direction of our hypothesis. The effect of neighborhood education on overweight/obesity and type 2 diabetes moving from Q1 to Q2 appears to be negative, but then is followed by a positive effect for moving from Q2 to Q3. Moving up from the lowest education quartile could decrease unhealthy food resources or crime in a neighborhood, leading to a decrease in overweight and

diabetes, while moving from the second to the third quartile might lead to a meaningful increase in access to resources such as supermarkets that also have unhealthy food. Relatedly, increases in smoking for Q2 vs. Q1 and Q3 vs. Q2 could reflect increased neighborhood resources that promote smoking, e.g. tobacco outlets or businesses which could encourage social smoking. However, all causal contrasts for the types 2 diabetes effect estimates were relatively close to zero and all effects appear non-significant except sensitivity analyses suggest that the Q3 to Q2 comparison may be marginally significant. The lack of significant results for diabetes may be because diabetes is a biological outcome as compared to overweight/obesity and smoking which are behavioral outcomes. Diabetes requires diagnosis by a physician, so that it may be more accurately measured. However, diabetes might also be less likely to be diagnosed in those residing in lower SES neighborhoods with less access to medical care.

The estimated effects of moving from one neighborhood median household income quartile to the next higher quartile on overweight/obesity, type 2 diabetes, and current smoking hover around zero and appear non-significant. Failing to detect any significant effect using neighborhood income may suggest that neighborhood education better captures the neighborhood SES construct. This could be due to increased measurement error in neighborhood income, as individuals are less likely to accurately report their income than their education, if they even report their income.

The majority of evidence from previous studies suggests that lower neighborhood SES is associated with worse health, including overweight/obesity,[57-60] type 2 diabetes,[55, 57, 63] and current smoking;[65-73] however, some differences exist across gender and

race/ethnicity. (We chose not stratify on individual-level gender or race/ethnicity primarily due to a limited sample size to detect an effect and reduced ability to find a counterfactual match.) Furthermore, these associations may not necessarily hold for all proxy measures of neighborhood SES, such as with the association between neighborhood education and smoking.[73] Some of our causal estimates here agree with these findings, but overall we found more non-significant than significant effects, and some of our significant results may be sensitive to unmeasured confounders. Our analysis techniques and design might explain estimating fewer significant effects than other studies. We examined the overlap of propensity scores in the exposed and unexposed groups for sufficient overlap to limit structural confounding, and then used propensity score matching to approximate an experimental design and counterfactual causal reasoning. Instead of using the highest quartile as the reference for all of the lower quartiles as is typically done, we chose to compare each quartile with the next lower quartile. This approach provides policy-relevant causal contrasts, because individuals are more likely to be able to move to the next higher neighborhood SES quartile. Using exchangeable populations for each causal contrast to minimize structural confounding and contrasting moving from one to the next highest quartile likely diminished the effects that we would have found using the approaches of the typical study, e.g. comparing quartiles 2, 3, and 4 to quartile 1.

The limitations of this study include several assumptions we made for propensity score matching. We assumed no confounding due to mismeasurement or missing variables (i.e. no hidden selection bias)[1, 30, 90] no clustering within neighborhoods, and

that the stable unit treatment assumption (SUTVA) holds (i.e. no spatial dependence between neighborhoods).[2, 30, 36, 125] Given these assumptions, the standard errors of the estimates could be too narrow because we did not account for clustering within neighborhoods, while any measurement error relating to using coarse dichotomous measures for outcomes likely weakened our estimates. Our sensitivity analyses for missing variables suggest that the estimated effects for obesity/overweight are highly sensitive to missing variables, thus the significant effect of moving from Q3 to Q4 education neighborhood may be confounded due to a missing variable. This may also be the case for smoking, although less likely, and quite unlikely for the diabetes outcome.

As with most other observational neighborhood effects studies, this study also fails to overcome the fundamental roadblock to identification of causal contextual effects: truly disentangling contextual effects from compositional effects. As Oakes points out, contextual effects suffer from an identification problem because "any emergent effects of neighborhoods on a person's health are *by definition* completely endogenous to the composition of neighborhoods; there is no exogenous intervention causing them." (p.1939).[2] A complex feedback loop exists between contextual and endogenous effects, where the neighborhood affects the individuals who affect the neighborhood, and so on. Manski describes this correlation as the "reflection problem" given that they appear to move in sync.[33, 87] Relatedly, we assume neighborhood-level SES is not on the causal pathway between individual-level SES and the health outcomes. Yet, we know that this is unlikely to be completely true given social interactions. In fact, most neighborhood studies make this assumption and many also examine mechanisms (e.g. social support) on

the causal pathway between neighborhood SES and health, which are arguably more enmeshed in the tangle of contextual and compositional (i.e. individual) factors. SUTVA is also inherently related to this part of the identification problem, in assuming that the effect of any treatment only affects the treated. For example, moving low-SES individuals into a higher-SES neighborhood affects the SES of the new neighborhood and thereby other individuals. Although some statistical techniques or conditions may help address the fundamental identification problem,[2, 33, 87] using these does not necessarily guarantee a firm causal foundation. Thus, the identification problems remains and we cannot know the true validity of our results.

Despite the limitations, this study has several key strengths which advance neighborhood effects research. First, we used robust analytical techniques for examining a neighborhood effects question. The use of propensity score matching explicitly examined the potential for structural confounding as well as more closely approximated an experiment by matching exposed individuals to unexposed individuals in order to calculate the average effect of the treatment on the treated. Second, although most studies would estimate the effects of moving from Q1 to Q2, Q1 to Q3, and Q1 to Q4, we chose an approach that is far more plausible in the real world for policy interventions. We examined an increase of just one quartile for each causal contrast, because someone in the lowest SES neighborhood could conceivably move to the next higher SES neighborhood but it is far less likely they could move to the highest SES neighborhood. We know of no other neighborhood effects study that has taken this approach across all quartiles. Third, we lost very few observations for each causal contrast as part of the

matching process, in large part because we compared adjacent neighborhood quartiles. Finally, we used high-quality data from CHIS and the US Census Bureau.

In conclusion, this study promotes a more technically advanced approach to estimating the effect of neighborhood SES on overweight/obesity, type 2 diabetes, and smoking than the traditional regression analysis. However, as with other neighborhood effects studies, the challenge of disentangling the individual from the neighborhood still exists. Future work with observational data should use a propensity score matching approach to address structural confounding and better approach an experimental design, but interventions and group randomized controlled trials may be required to truly advance this area of research.

4. Spatial Epidemiology Perspective

Neighborhood or contextual effects studies typically fail to account for spatial dependence across neighborhoods, which may bias estimates and/or affect their efficiency. Furthermore, these studies generally do not examine the ways in which a given neighborhood may be influenced by surrounding neighborhoods (i.e. spillover effects), data which could enhance interventions. Spatial regression addresses these shortcomings using spatial lag and/or spatial error models.

Overweight/obesity, type 2 diabetes, and smoking are major public health problems which may be caused in part by neighborhood-level characteristics, such as neighborhood socioeconomic status (SES). Structural forces stratify individuals into neighborhoods of differing SES, where they likely have differential access to resources which may positively or negatively affect their health. We hypothesize that individuals residing in higher-socioeconomic neighborhoods have a lower prevalence of overweight/obesity, type 2 diabetes, and current smoking, and that characteristics of the surrounding neighborhoods may have an effect on these health outcomes.

We estimated the effect of neighborhood SES (education and income) on overweight and obesity, type 2 diabetes, and current smoking. We approximated a spatial multilevel regression model by accounting for differences in individual-level covariates before aggregating the data up to the neighborhood and then running spatial lag models. The spatial lag models accounted for and explicitly examined the dependence of nearby neighborhoods.

We estimated a significant negative effect of increasing neighborhood education and income on type 2 diabetes and smoking. Accounting for spatial dependence slightly attenuated the effect estimates and tightened the confidence intervals but did not affect overall results. We also found evidence of significant spillover effects in these models. We failed to find evidence of an effect of neighborhood education or income on overweight/obesity.

Accounting for spatial dependence did reduce spatial autocorrelation of residuals, but the spatial multilevel models provided little improvement over the approximated multilevel models. These approaches were primarily useful as a diagnostic tool and as a means to examine potential spillover effects. The spatial lag model in particular suggests that the effect of neighborhood SES spills across nearby neighborhoods. Despite these analytical improvements, this study remains an observational study and likely suffers from an identification problem.

Keywords: Spatial dependence, spatial lag, spatial error, neighborhood effects, contextual effects

4.1 Introduction

Over the past few decades neighborhood effects studies have shown support for an association of neighborhood or contextual factors with health;[4, 8, 17, 24] however, most etiologic studies are limited by methodological challenges.[2] One key challenge is spatial dependence between neighborhoods. Failure to incorporate spatial dependence may bias estimates and affect precision. Incorporating spatial effects using spatial regression analysis may improve causal inference and thereby policy interventions aimed at reducing major public health problems, such as overweight/obesity,[9-12] type 2 diabetes,[13, 14] and smoking.[15, 16, 89] However, spatial models are markedly more complex than traditional multilevel models and still retain other potential identification problems inherent in observational neighborhood effects research.

4.1.1 Contextual Effects

Research into contextual effects – also called neighborhood, place, or area effects – on health has increased substantially in the last few decades with the recognition that health is affected by factors at many levels, not just at the individual level.[27, 95] Contextual effects arise from the context in which the population lives and are distinguished from compositional effects, which are due to differences in the population characteristics across neighborhoods.[18] In an attempt to isolate contextual from compositional effects, contextual effects studies account for differences in individual-level risk factors.[23] Multilevel regression is the standard approach in neighborhood effects studies of health outcomes, as it accounts for individual-level differences and clustering of individual observations within contexts.[126-128] Multilevel (i.e. hierarchical)

models assume that neighborhoods are independent of each other; however, many researchers argue that neighborhoods should not be treated as independent.[39-43, 55, 129]

4.1.2 Spatial Dependency

Tobler's informal 'First Law of Geography' states: "everything is related to everything else, but near things are more related than distant things" (p.236).[130] This dependence or clustering of observations across space is called spatial autocorrelation.[52, 53] Work on spatial dependency and spillover effects suggests that neighborhood effects may extend beyond the boundaries of each neighborhood, e.g. the health of a neighborhood's residents may depend not only on their own neighborhood's SES but also the SES of nearby neighborhoods.[39, 42, 49, 51, 54-56, 82, 83] Many contextual effects studies ignore this potential spillover effect and treat neighborhoods as independent contexts.[55, 129] Spatial autocorrelation may be present not only given true spatial effects but also in a misspecified model (e.g. missing covariates with a given spatial footprint) or context (e.g. neighborhood boundaries that do not match scale of proposed phenomena).[44, 45, 53] Ignoring spatial autocorrelation due to either may bias true contextual effects estimates and/or affect their precision.[44-47]

4.1.3 Addressing Spatial Dependence and Examining Spatial Effects with Spatial Models

In recent decades medical geographers and spatial epidemiologists have developed spatial regression techniques that incorporate spatial effects to account for spatial dependence *between* (instead of just within) neighboring areas and/or to specifically examine any spillover effects.[38, 45, 53] Most spatial regression techniques focus

on model building and prediction (e.g. geostatistical methods); however, models primarily from spatial econometrics – in particular the spatial lag model – often build from a theoretical base with a goal of identification of etiologic effects.[51, 131] Spatial error and spatial lag models are commonly used spatial econometric models, and both are types of spatial autoregressive models, which "regress the current observation on observed values[…] of other observations" (p. 362).[45]

The spatial error model anticipates that any spatial dependence is due to model misspecification which creates spatially correlated errors; this dependence is treated as a nuisance.[44, 51] The spatial error model builds from the standard linear regression equation

$$y = \mathrm{X}\beta + \varepsilon,$$

where X represents the exposure variables, β the corresponding coefficients, and ε the random error terms, but accounts for spatial dependence in the error term (ε) as follows:

$$\varepsilon = \lambda W\varepsilon + u,$$

where λ is the autoregressive parameter, W is the spatial weights matrix defining the spatial dependence, and u is the random error.[44, 51]

By contrast, the spatial lag model hypothesizes that the spatial autocorrelation represents a spatial process of interest such as spillover effects.[44, 51] The spatial lag model adds a spatially lagged dependent variable in the regression equation;[51] this is the autoregressive component of the spatial lag model.[132] The spatial lag regression equation is:

$$y = \rho Wy + \mathrm{X}\beta + \varepsilon,$$

where y on the left-hand side of the equation represents the outcome at the focal neighborhood, while y on the right-hand side of the equation represents the outcomes of other neighborhoods, W is the spatial weights matrix, Wy together is the spatial lag term, and ρ is the spatial lag term coefficient.[44, 51] The spatial lag term Wy represents the weighted average of the outcome of the neighbors, while its coefficient ρ represents the change in a neighborhood's outcome associated with a one-unit change in the outcome of its neighbors.[49] The lag coefficient ρ ranges from 0-1 and indicates the strength of the spatial lag.[49, 132] However, ρ does not necessarily identify a causal effect of the outcome in neighboring areas on the focal neighborhood, because *y* on the right-hand side of the equation represents not only the *y* of neighbors but also incorporates the spatial patterns in X and ε of the neighbors.[133] Furthermore, ρ captures the spatial simultaneity across the entire study area, where the outcome for each neighborhood is affected by its neighbors' outcomes, who are affected by their neighbors' outcomes and so on, introducing correlation between the outcome *y* for one location and the error terms of all other locations.[44, 51, 132] This spatial simultaneity or endogeneity problem is addressed using estimation methods (i.e. maximum likelihood estimation or method of moments estimation).[44, 51]

The challenges and limitations of using spatial lag and error models include specifying *a priori* the spatial weights matrix (i.e. quantifying how surrounding neighborhoods affect the focal neighborhood),[48, 134, 135] assuming spatial associations do not vary across space[43, 48] and no dependence between the outcomes in the individuals and the neighborhoods.[87, 134] Furthermore, spatial lag and error models typically use area-

level data and do not allow for a mix of point-level (i.e. individual-level) and area-level data,[51] as is often used in traditional multilevel regression of health data when attempting to disentangle the contextual effect from the compositional. However, by using census tract-level health outcomes adjusted for differences in individual-level characteristics, we can approximate spatial multilevel models.[49]

Despite these challenges, incorporating spatial effects into multilevel neighborhood effects studies may enhance etiologic inference in contextual effects studies, as well as function as a diagnostic tool to identify potential factors to consider in future studies. Furthermore, examining spatial effects may aid public health interventions, which can be designed at an appropriate scale and coordinated with neighboring administrative units given any spillover effects.[39, 42, 83]

4.1.4 Neighborhood Socioeconomic Status (SES), Structuralism and Health

Theory on socioeconomic status (SES) and structuralism supports the spatial study of neighborhood SES and health. The Weberian tradition of SES studies focuses on how differences in an individual's accumulation of economic resources, knowledge, and skills affect one's "life chances" and position in the social strata,[76, 93] which in turn may affect their health.[77] Moving from the individual to the neighborhood level, structuralism is "the stratification of neighborhoods by SES and their segregation by race/ethnicity" (p.295).[136] Foundational work on structuralism includes work from Wilson,[75] Massey,[5] Massey and Denton,[91] and Jargowsky.[137] Residents stratified or segregated into disadvantaged neighborhoods experience worse health.[4-8] This may be due in part to the limited health-promoting social and material resources in lower-SES neighborhoods,[6, 73,

[136, 138] which may constrain health-promoting behaviors of residents.[75] Evidence suggests that neighborhood SES may affect overweight/obesity,[57-62] type 2 diabetes,[55, 63, 64] and smoking behavior.[65-73] Furthermore, the effects of neighborhood SES may spill over into surrounding neighborhoods.[39, 42, 49, 51, 54-56, 82, 83]

Although most recent studies focus on a specific mechanism on the causal pathway between neighborhood SES and a health outcome,[1, 4, 94] neighborhood SES itself is still important to study, in large part given the high correlation of social variables which make disentangling any single mechanism especially challenging.[77] Neighborhood SES can be measured in many ways, but single variables (as compared to an index, for example) are easy to obtain and provide a clear focus for policy interventions.[77] However, use of a single measure (e.g. median household income) likely increases measurement error, given that a single measure cannot capture the full complexity of SES.[24, 77] Two strong single measures of neighborhood SES are educational attainment and income.[76, 77] Educational attainment assesses access to human, social, and environmental capital, while income evaluates access to material capital and conditions.[76, 77, 93] Although both are easy to measure, income suffers from much greater participant nonresponse and misreport.[77]

In this study we estimated the effect of increased neighborhood education and income on overweight/obesity, type 2 diabetes, and smoking, taking into consideration individual-level differences and spatial dependence. Our hypothesis was that neighborhood SES would inversely affect these health outcomes and spill over to surrounding neighborhoods.

4.2 Methods

This study uses a cross-sectional design with approximated multilevel and spatial multilevel models to estimate the effect of neighborhood SES (education and income) on overweight/obesity, type 2 diabetes, and smoking, accounting for individual-level differences. The spatial models also examine spatial dependence and spatial spillover.

4.2.1 Study Population and Study Area

The subject population is adults residing in the City and County of San Francisco, California. San Francisco is densely populated (17,246 people/sq. mile) and contains a diverse population: 48.5% white, 33.3% Asian, 6.1% African American, with 15.1% of any race identifying as Hispanic/Latino.[139] This density and diversity may increase the potential for comparable populations across neighborhood SES groups and thereby limit structural confounding,[29, 30] which this study does not address directly. San Franciscan adults are better educated and wealthier than US adults as a whole: 51% vs. 28% have at least a bachelor's degree and the median household income was $55,221 vs. $41,994.[103] Limiting the study to San Francisco has several benefits, including eliminating confounding due to higher-level macro factors and urban/rural differences, such as cost of living.

4.2.2 Neighborhood

We define neighborhoods as the 2010 census tracts. San Francisco contains 196 census tracts (per 2010 boundaries). Two census tracts were eliminated from the study area given their spatial segregation from the rest of San Francisco: 1) the Farallon Islands, which lie offshore in the Pacific Ocean, and 2) Treasure Island, which included

several small, uninhabited islands in the San Francisco Bay and one small sliver on the bay from the Bayview/Hunters Point area. The final sample contained 194 census tracts. Only four individuals were dropped from the analyses, all of whom were in the Treasure Island tract.

Census tracts are widely used in neighborhood effects studies, in part because data is readily available at the tract level. Tracts are relatively demographically homogeneous and thus arguably a good proxy for neighborhoods.[97] Although choice of scale and the ways in which regions are zoned for the geographic units may impact the results (i.e. the modifiable areal unit problem [MAUP][140]), research suggests tracts capture neighborhood processes better than zip codes and similar to block groups,[98] and might not affect inference at smaller scales.[141] Use of a spatial lag model softens the definitive tract boundaries by allowing exposures in nearby tracts to affect the health of residents in the focal neighborhood, instead of limiting the effect of an exposure to the focal neighborhood's exposure.

A common alternative to census-derived or fixed-neighborhood units are buffers around an individual's residence. The focus in these studies therefore is on changing each individual's context. By contrast, this study seeks to mimic a community trial, where the intervention is at the neighborhood level, so we use neighborhood-level exposure data. In addition, given that data for the exposure of interest (neighborhood SES) in this study comes from census tracts, using a buffer around an individual's residence to define the neighborhood would cut across tracts and require the extraordinary assumption that values for the tract are spread evenly across the entire tract.

4.2.3 Data Sources

Individual-level data for this study come from the 2005, 2007, and 2009 California Health Interview Survey (CHIS)[100-102] for San Francisco adults (n=2,511). Neighborhood-level data come from the 2006-2010 Summary File from the American Community Survey (ACS) for census tracts (n=194).[103] The latitude and longitude of each CHIS participant was used to identify their 2010 census tract, which was used to link to ACS data.

CHIS is the largest state health survey in the country, and provides a high-quality representative sample for San Francisco obtained using random digit dialing.[104] The statewide response rates for CHIS for 2005 (29%) and 2007 (24%) were comparable to the California Behavioral Risk Factor Surveillance Survey (BRFSS) and somewhat lower for 2009 (19.7% vs 27.1%).[105-107] Missing values were imputed by CHIS staff for most variables using a combination of hot deck and logical imputation; however, the amount of missing data was typically quite low (<2% of the sample), with some exceptions such as household income (approx. 20-25% missing).[108-110] One limitation of this pooled CHIS dataset is that a few tracts (6%) have only 1-3 observations. This may result in a "small numbers problem," where rates for a tract may be unstable due to the limited number of observations.[45, 142]

ACS 5-year Summary Files are replacing the decennial census long form as the standard for neighborhood socioeconomic data. These summary files provide high-quality data for smaller geographic units, which are readily available, timely, and collected by trained staff. As with long form data, ACS data likely undercount

minorities[97] and offer reduced accuracy at smaller aggregation units (in part to ensure confidentiality).[111]

4.2.4 Neighborhood SES Exposures

The two exposures of interest representing neighborhood SES are: percent of a tract's population aged 25 years or older with a bachelor's degree or higher (neighborhood education) and tract median household income (neighborhood income). Both exposures were classified into quartiles where quartile one (Q1) represents low, Q2 moderate-low, Q3 moderate-high, and Q4 high neighborhood SES (i.e. highest percent college educated or highest median household income).

Both *a priori* cutpoints and quartiles can detect socioeconomic gradients;[98] however, compared with the US as a whole, San Francisco adults are more highly educated and have higher incomes. Therefore, in order to capture the neighborhood SES gradient and ensure sufficient samples in each exposure class, we chose to use quartiles instead of *a priori* cutpoints to classify neighborhood education and income. These quartiles were derived from census-based distributions of tract education and income in San Francisco.

4.2.5 Outcome Variables

Overweight or obesity, type 2 diabetes, and current smoking are the health outcomes of interest in this study. All outcomes are dichotomous and were obtained by self-report. The overweight/obesity measure is body mass index (BMI), obtained by dividing weight in kilograms by height in meters-squared (kg/m^2). This includes both those who were overweight (BMI=25.0-29.9) or obese (BMI≥30.0).[9] We included

overweight individuals with obese individuals because negative health consequences may begin already in the overweight and since San Francisco has comparatively fewer obese individuals than other areas. Type 2 diabetes comes from self-report of doctor diagnosed type 2 diabetes. Current smoking versus former or never smoking was classified from questions on current and past smoking behavior together with frequency of use. Self-report of Type 2 diabetes and smoking behavior are known to have high validity,[112, 113] and although self-report of height and weight for BMI is typically significantly underestimated for overweight adults, adjusting for socio-demographic characteristics minimizes the potential for measurement error bias.[114, 115]

4.2.6 Covariates

We minimized the possibility that an effect of neighborhood SES on the outcomes is confounded by differences at the individual-level by adjusting for a set of individual-level covariates hypothesized to be associated with both neighborhood SES and the outcomes. Given social stratification into neighborhoods and data availability from CHIS, the following individual-level covariates are accounted for in the analyses: age, gender, household income, poverty (above/below 200% of federal poverty level), education (<high school; high school; some post-secondary; bachelor's degree or higher), African American, white, Hispanic/Latino, Asian, marital status (married or living with partner; separated, divorced, never married), employment status (employed; unemployed), and home ownership (own; rent or other arrangement).

4.2.7 Analytical Models and Procedure

We used aspatial and spatial regression to estimate the effect of increasing neighborhood SES on overweight/obesity, type 2 diabetes, and current smoking. We accounted for individual-level differences before aggregating individual-level outcome data up to the neighborhood (i.e. tract) level. In the spatial models we also accounted for spatial dependence and estimated spillover effects to surrounding neighborhoods.

All spatial autocorrelation and spatial regression analyses used a queen's contiguity criterion to identify the neighbors of each tract. Queen's criterion captures all adjacent tracts including those that touch only at a corner. Compared with distance-based neighbors or more complicated criteria, queen's criterion is straightforward, simple to understand, and does not require on-going refinement, which could affect inference. We applied row-standardized weights to the neighbors, as is most common for spatial lag and error analyses.[51, 53] Row-standardization adjusts the weights by the total number of neighbors to provide an average value across all neighbors.[44, 45]

Descriptive statistics for the 2005/2007/2009 pooled sample were calculated by neighborhood exposure quartile using Stata/SE 12.1.[143] Exploratory spatial data analysis was conducted in ArcGIS 10.1[144] and included mapping each neighborhood exposure and the crude prevalence of each outcome by tract. We conducted global Moran's I tests to assess clustering of similar neighborhood-level observations across the study area (i.e. overall spatial dependence),[45] and local Moran's I (LISA) tests to identify any individual neighborhood clusters potentially driving an association (i.e. outliers).[45] Moran's I values typically range from -1 to 1, where values approaching 1 suggest clustering of similar values, 0 no clustering, and -1 clustering of dissimilar values.[45]

Answering our study question using spatial lag (and spatial error) models required aggregation of individual-level data to the tract level. However, given that we seek to disentangle the contextual effect from the individual, we also needed to limit confounding of the contextual effects due to individual-level differences. Following from others[42, 48, 49] we approximated a spatial multilevel model (i.e. spatial multilevel model) as follows: first, we ran an ordinary least squares (OLS) regression with only the individual-level covariates and outcomes to determine predicted outcomes for each individual. Next we averaged these individual predicted values by tract to determine a tract-level outcome value (i.e. adjusted prevalence of outcome in the tract). Finally, we used these tract-level values for the remaining analyses.

Leading up to the spatial analyses, we first performed an aspatial OLS regression of each adjusted, aggregated neighborhood-level outcome on neighborhood education and neighborhood income (separately). These OLS models are the approximated multilevel models, which estimate the independent effect of neighborhood SES on individual level outcomes. Next, we examined the OLS residuals using Moran's I tests of the residuals, maps of the residuals, Moran scatterplots, and LISA cluster and significance maps using ArcGIS 10.1 and GeoDa 1.0.1.[145] This exploratory residual analysis helped identify potential remaining spatial autocorrelation in these adjusted models, as well as provided information on model specification.

If the approximated aspatial multilevel model residuals exhibited spatial autocorrelation, we ran an approximated spatial multilevel regression using the spatial lag

model, as per our hypothesis that neighborhood effects would spill over to surrounding neighborhoods.

To examine model fit and the potential for model misspecification of the spatial lag models, we used Lagrange Multiplier (LM) tests, ran spatial error models, and examined model residuals from both the spatial lag and error models (as described above with multilevel models). LM tests are maximum likelihood based tests of the null hypothesis of no spatial autocorrelation, which attempt to differentiate the source of any remaining spatial autocorrelation in the residuals: spatial lag or spatial error (i.e. spatial spillover or nuisance factors).[44, 53] Using the LM test results and following from a decision rule process, whereby the spatial model with the largest significant test statistic is suggested to be the more appropriate model, and if both models are significant then the same rule is applied to the robust tests.[44, 53, 146] However, if the robust lag and error test statistics are close and/or non-significant, this may suggest model misspecification beyond any spatial element.[146]

More traditionally, medical geographers and spatial epidemiologists rely on the Lagrange Multiplier (LM) test together with model fit parameters to determine whether to use the lag model or the error model, as well as to identify ways to improve model prediction. However, we were interested in testing a specific hypothesis for a potential etiologic factor as is more common in epidemiology.

The approximated aspatial and spatial multilevel etiologic analyses were conducted in R using the spdep package.[147] The spdep package uses maximum likelihood estimation for both the spatial lag and error models.

4.3 Results

Descriptive statistics (Tables 1A and 1B – see above) show differences in individual-level characteristics and health outcomes by neighborhood education and income quartiles. As expected lower neighborhood SES quartiles contain higher percentages of minority, unemployed, less-educated, and lower-income individuals, and health outcomes generally improve as neighborhood SES improves; however, this association is not consistent between all quartiles. Visual comparison of the maps of crude neighborhood exposures (Figures 14-15) and crude aggregated outcomes in the study sample (Figures 16-18) indicated some overlap of neighborhood exposures with outcomes, which together with the descriptive statistics suggested possible relationships. Global Moran's I tests (Table 4) confirmed significant strong positive spatial autocorrelation of both neighborhood exposures. For the unadjusted disease outcomes Global Moran's I identified significant but weak positive spatial autocorrelation of the prevalence of overweight/obesity, but no significant correlation of the prevalence of type 2 diabetes or current smoking. Moran's I scatterplots and Local Moran's I (LISA) cluster maps (not shown) showed potential outliers for each variable, some of which appeared to be clustered across space. Although not all outcomes exhibited global spatial autocorrelation, we also examined spatial dependence in the adjusted models (see below), as individual-level differences could mask spatial clustering.

Our spatial analyses began with the aspatial OLS (i.e. approximated multilevel) regression model using adjusted data aggregated to the neighborhood level for each neighborhood exposure/outcome relationship. The statistically significant Moran's I tests

Figure 14: Neighborhood Percent with Bachelor's Degree or Higher, San Francisco 2006-2010

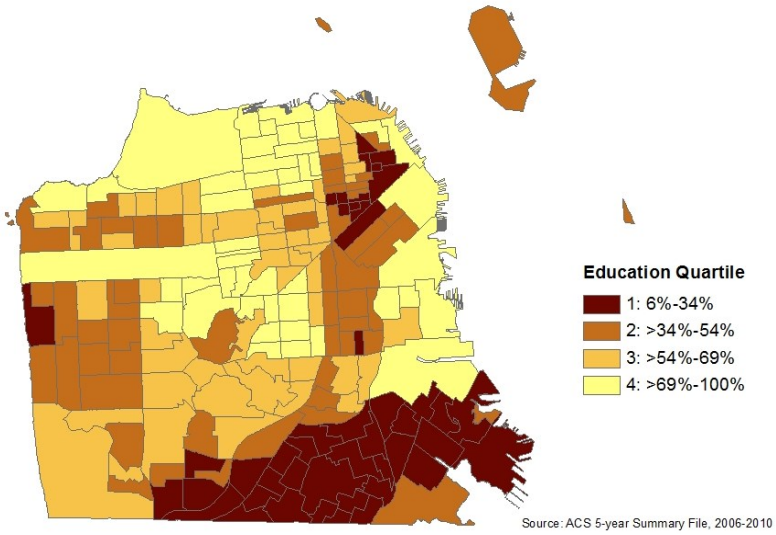


Figure 15: Neighborhood Median Household Income, San Francisco 2006-2010

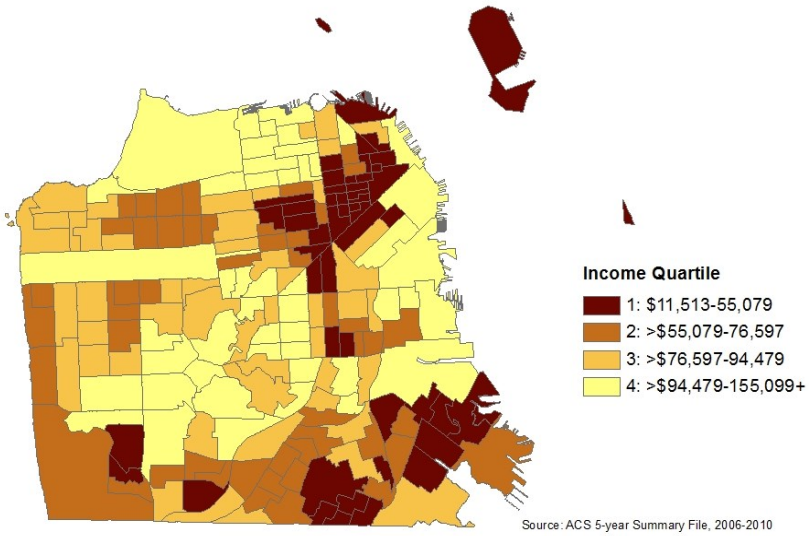


Figure 16: Overweight or Obesity in Adults by Census Tract, San Francisco 2005, 2007, 2009

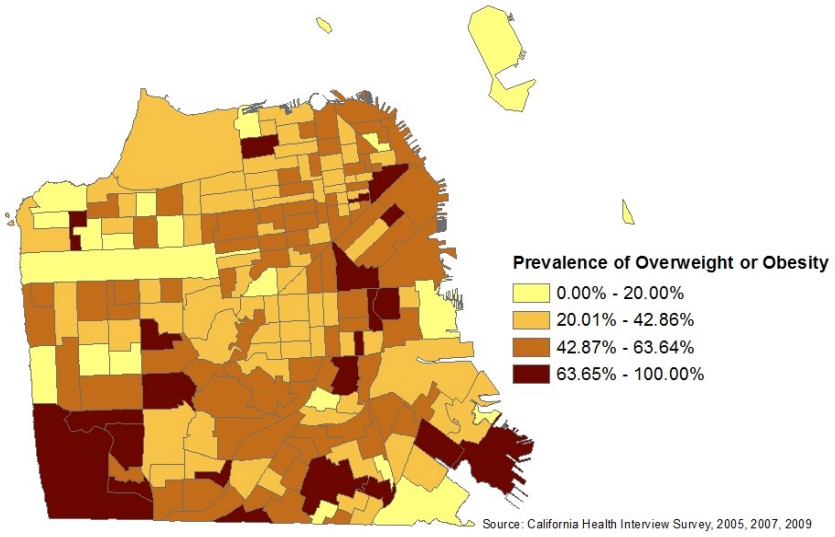


Figure 17: Type 2 Diabetes in Adults by Census Tract, San Francisco 2005, 2007, 2009

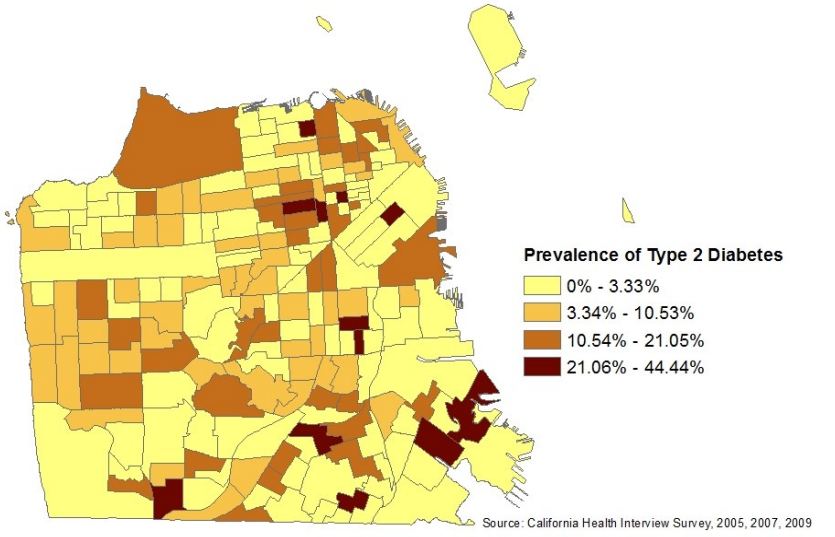


Figure 18: Current Smoking in Adults by Census Tract, San Francisco 2005, 2007, 2009

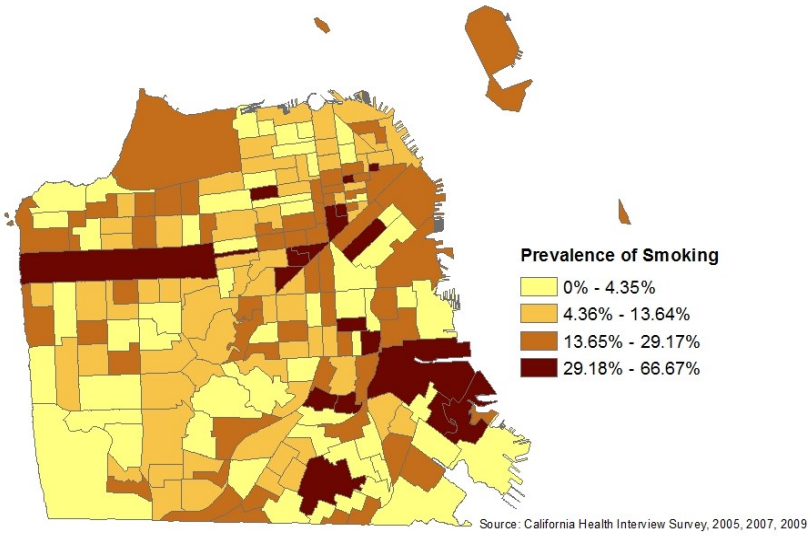


Table 4: Global Moran's I Test for Spatial Autocorrelation of Neighborhood Exposures and Health Outcomes, San Francisco Adults, 2005, 2007, and 2009 (Sources: ACS, CHIS).

	OLS Model	
	Moran's I	P-value
Neighborhood % with bachelor's degree or higher	0.6686	<0.0000
Neighborhood median household income	0.5074	<0.0000
Overweight or Obese	0.0855	0.0143
Type 2 Diabetes	-0.0459	0.8384
Current Smoking	0.0354	0.1623

Row-standardized weights, queen's criterion

of the OLS residuals (Table 5) together with other tools in the explanatory residuals analysis (Figures 19-24) suggested positive spatial dependence in the models for all outcomes. Given this spatial dependence we conducted spatial lag regression for all models, even for the overweight/obesity models which had non-significant estimates and particularly low R^2 values (see below). Estimated effects of neighborhood education and income from the approximated aspatial multilevel and spatial multilevel models are similar in magnitude and direction, and provide the same conclusions in terms of statistical significance (Tables 6A and 6B, Figures 25-26). Neighborhood education is not statistically significant for overweight/obesity, but it is significant for type 2 diabetes and smoking. The spatial lag model suggests that a one-quartile increase in neighborhood education causes a 0.008 (95% confidence interval [CI]: -0.012 to -0.005) decrease in prevalence of type 2 diabetes and a 0.008 (95% CI: -0.012 to -0.003) decrease in prevalent smoking, after accounting for spatial spillover. The spatial lag coefficient suggests significant spatial spillover across neighborhoods: a one-unit change in the outcome in nearby neighborhoods is associated with a 0.335 (p-value=0.001) change in diabetes and a 0.535 (p-value<0.000) change in smoking in the primary neighborhood. The spatial lag coefficient from the spatial lag model always falls between 0 and 1, and as such represents a rate of spillover or influence from nearby neighborhoods on the outcome in the primary neighborhood.[49] However, as discussed earlier, this should not be interpreted as a causal effect of diabetes or smoking in neighboring areas on the primary neighborhood, but rather as the potential effects from both observed and unobserved characteristics of nearby neighborhoods.[49]

Table 5: Moran's I Test for Spatial Autocorrelation of Model Residuals from OLS, Spatial Lag, and Spatial Error Regression of Health Outcomes Regressed on Neighborhood (Census Tract) Education and Median Income, San Francisco Adults, 2005, 2007, and 2009 (Sources: ACS, CHIS).

	OLS Model		Spatial Lag		Spatial Error	
	Moran's I	p-value	Moran's I	p-value	Moran's I	p-value
Neighborhood % with bachelor's degree or higher						
Overweight or Obese	0.2701	0.0000	-0.0311	0.5927	-0.0312	0.5852
Type 2 Diabetes	0.1542	0.0001	-0.0039	0.9084	0.0935	0.0190
Current Smoking	0.2837	0.0000	-0.0258	0.6834	-0.0378	0.4758
Neighborhood median household income						
Overweight or Obese	0.2733	0.0000	-0.0282	0.6276	-0.0319	0.5163
Type 2 Diabetes	0.1301	0.0010	-0.0303	0.5929	-0.0078	0.8445
Current Smoking	0.1570	0.0001	-0.0510	0.3038	-0.0280	0.4272

Row-standardized weights, queen's criterion

Figure 19: Residual Analysis from OLS Regression of Neighborhood Education on Prevalence of Overweight/obesity in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.

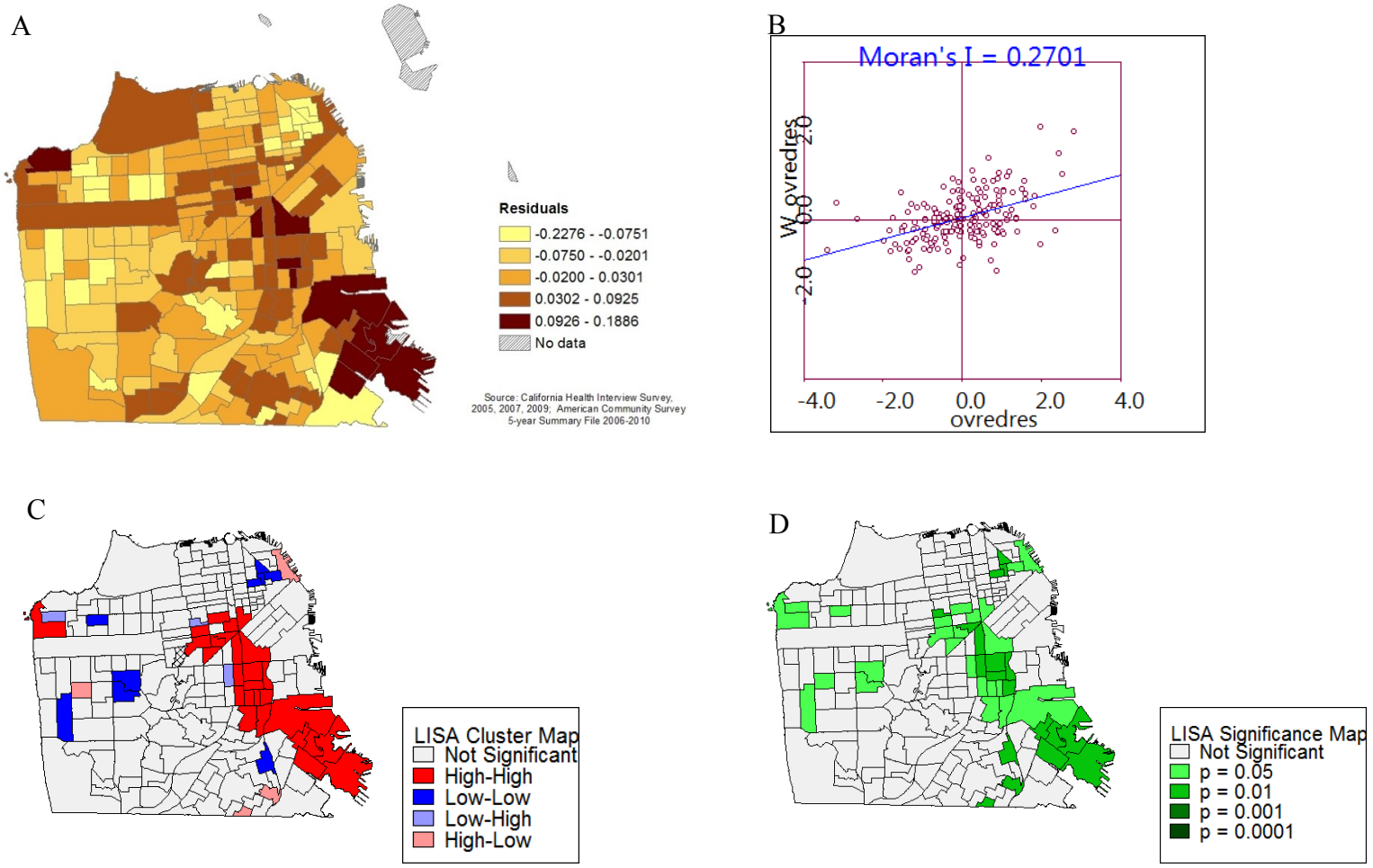


Figure 20: Residual Analysis from OLS regression of Neighborhood Education on Prevalence of Type 2 Diabetes in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.

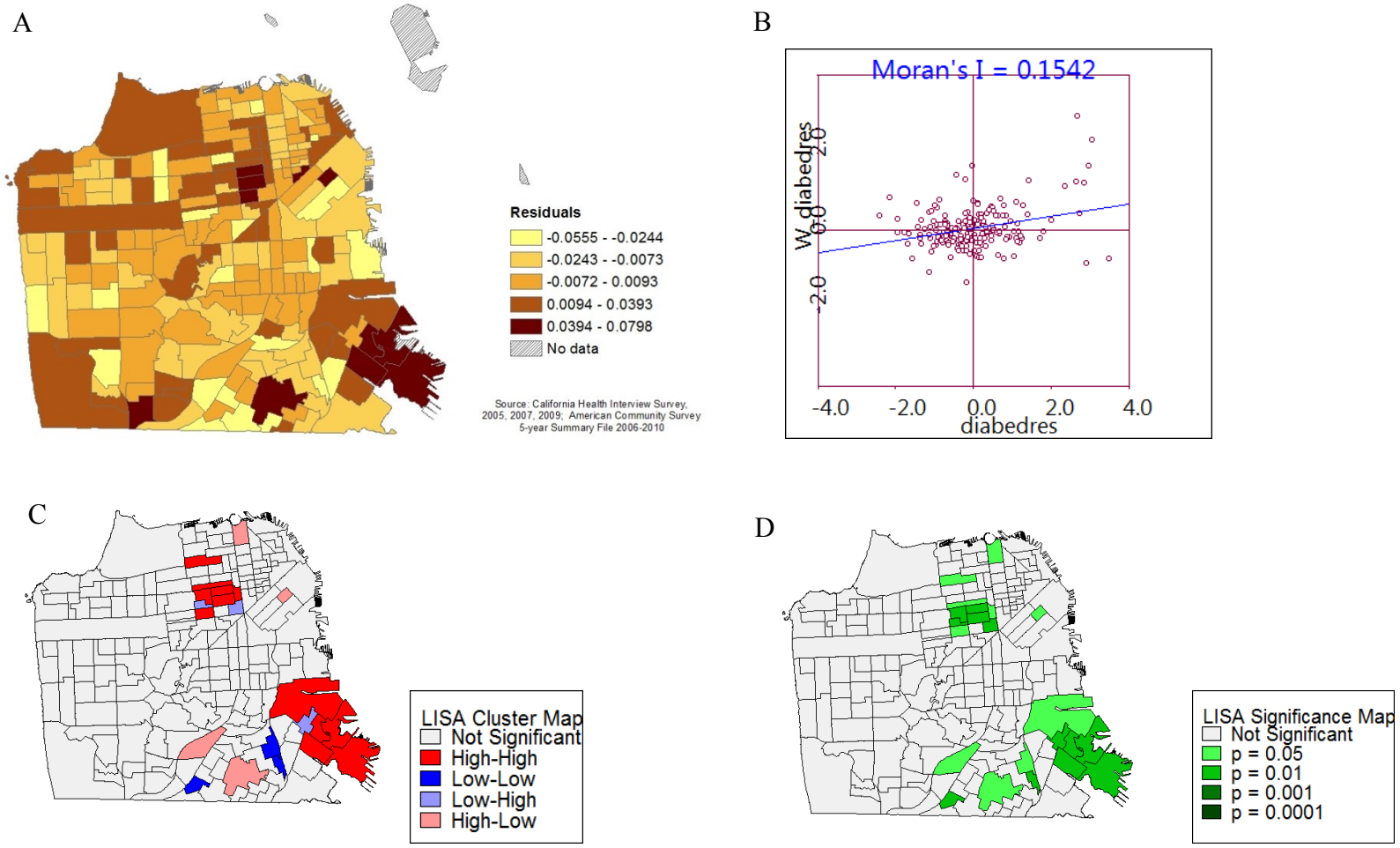


Figure 21: Residual Analysis from OLS Regression of Neighborhood Education on Prevalence of Current Smoking in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.

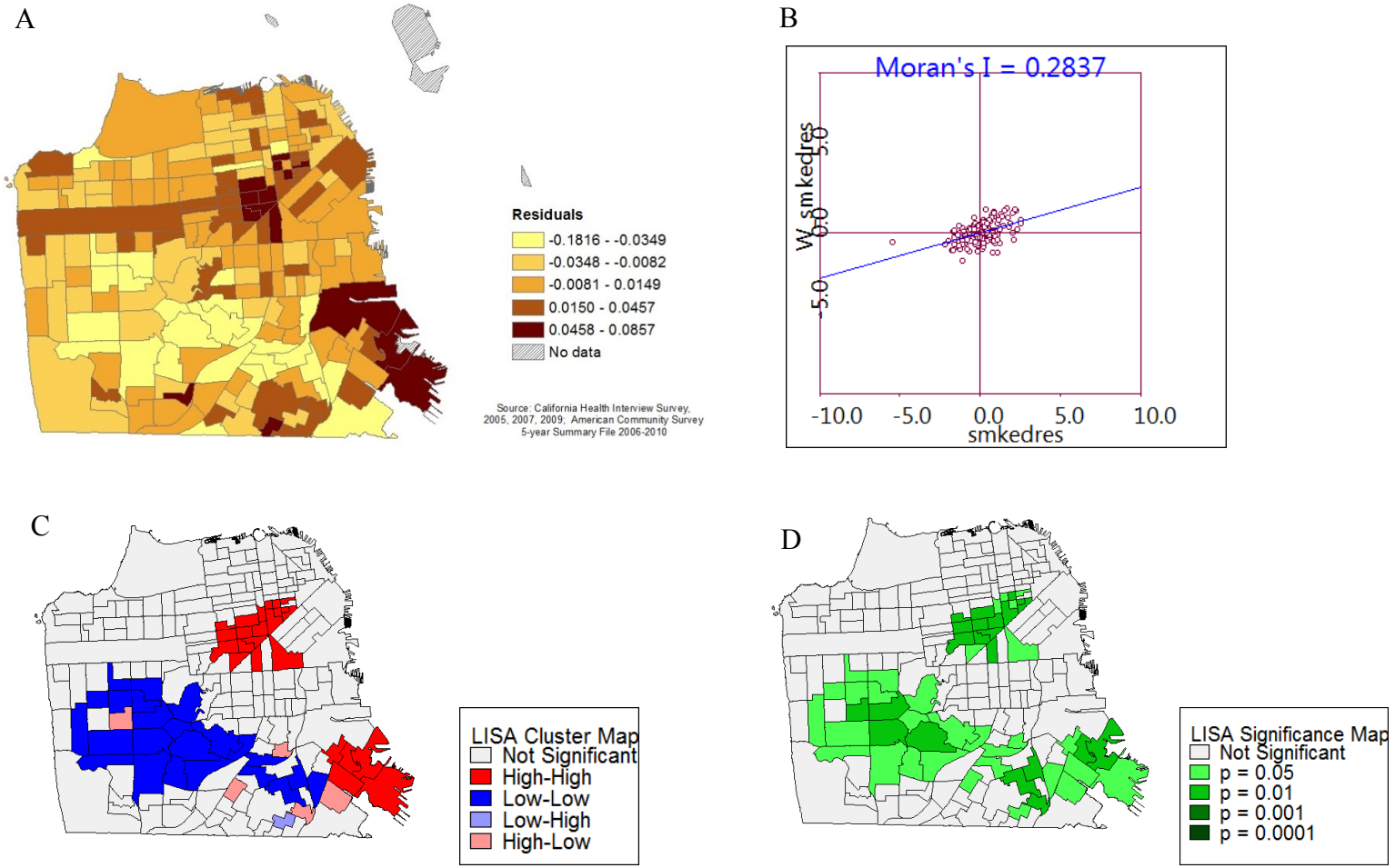


Figure 22: Residual Analysis from OLS Regression of Neighborhood Income on Prevalence of Overweight/obesity in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.

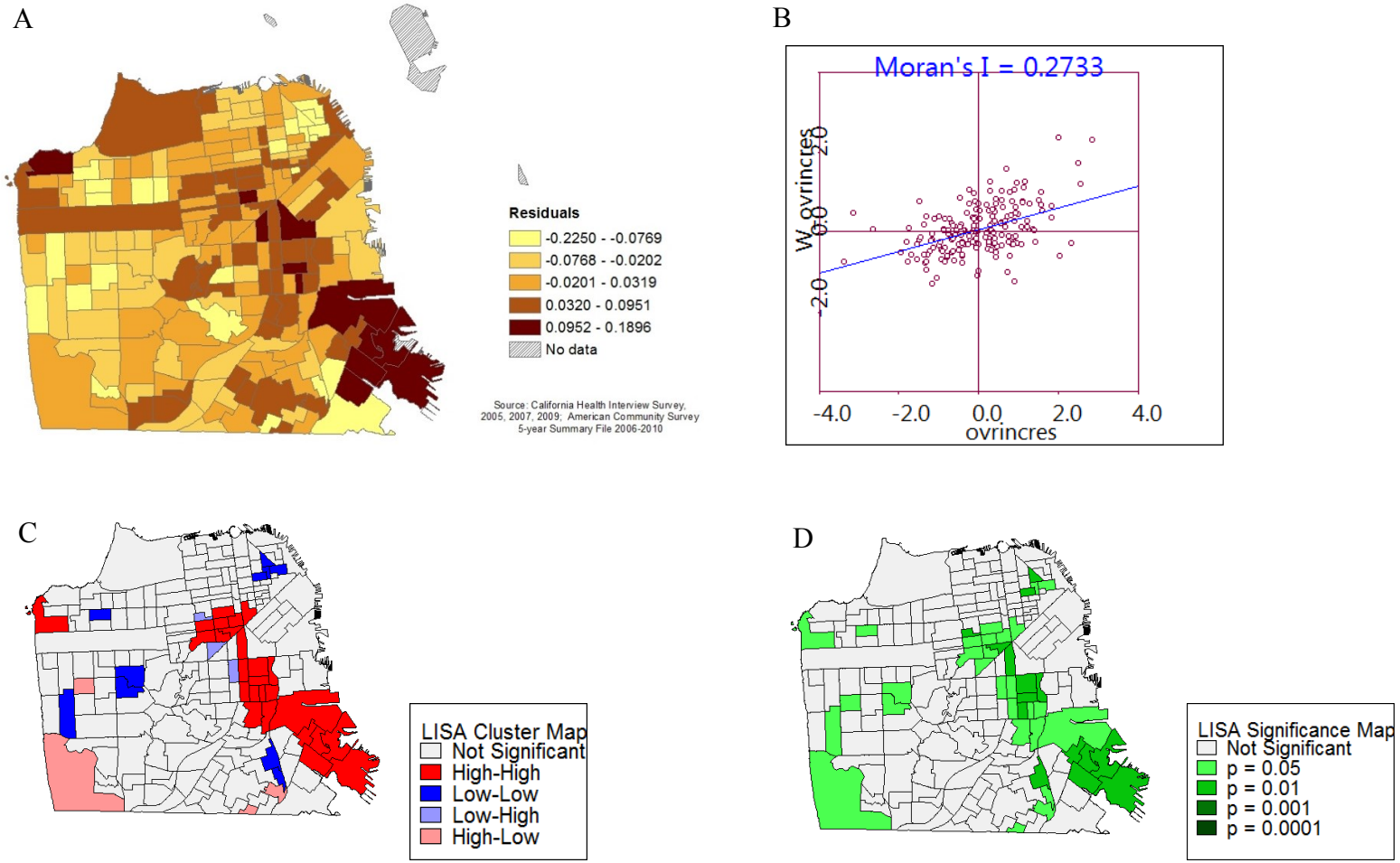
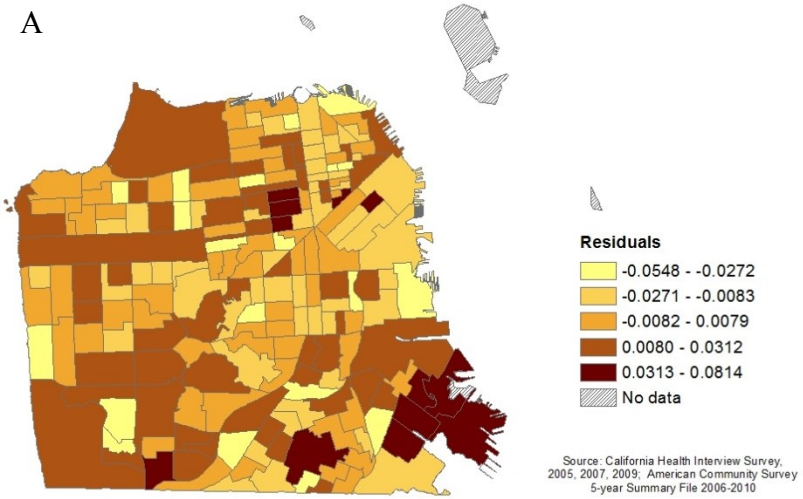


Figure 23: Residual Analysis from OLS Regression of Neighborhood Income on Prevalence of Type 2 Diabetes in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.

A



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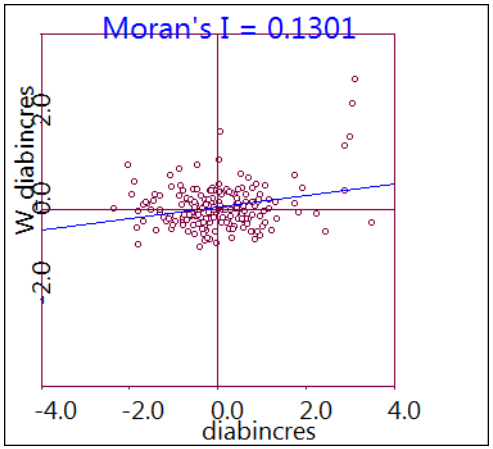

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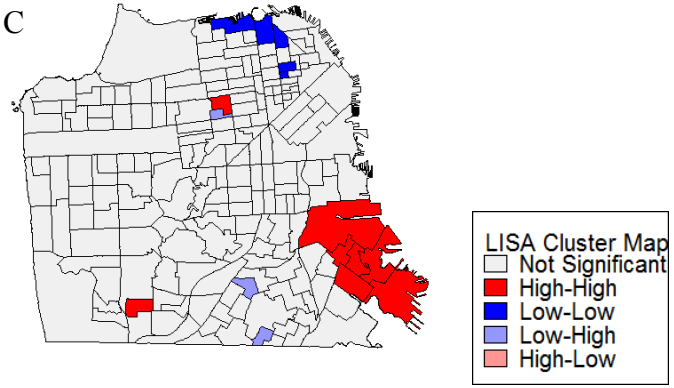

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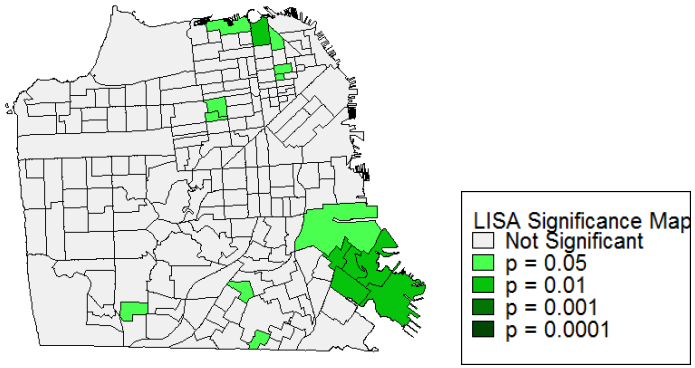
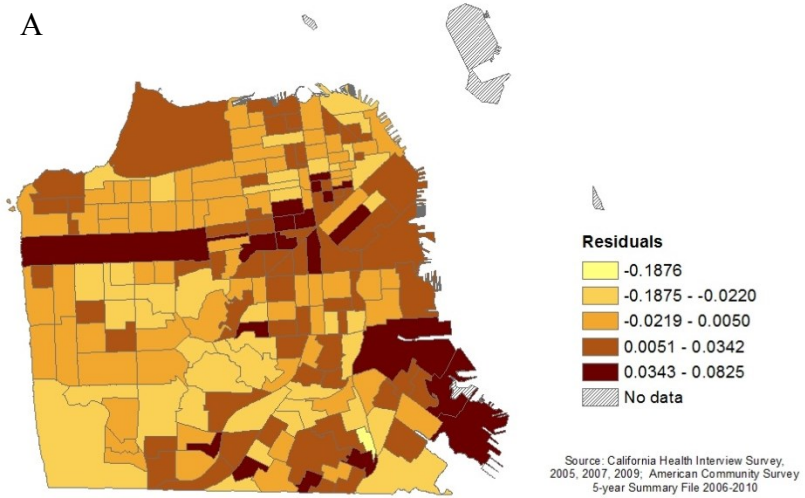


Figure 24: Residual Analysis from OLS Regression of Neighborhood Income on Prevalence of Current Smoking in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.

A



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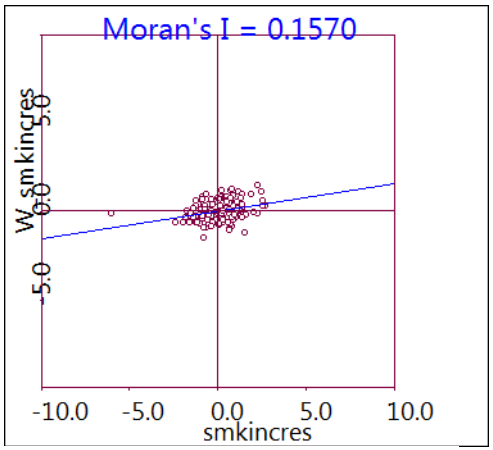

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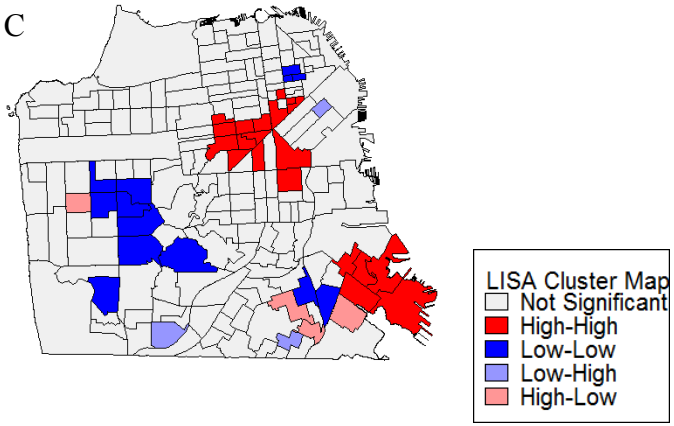

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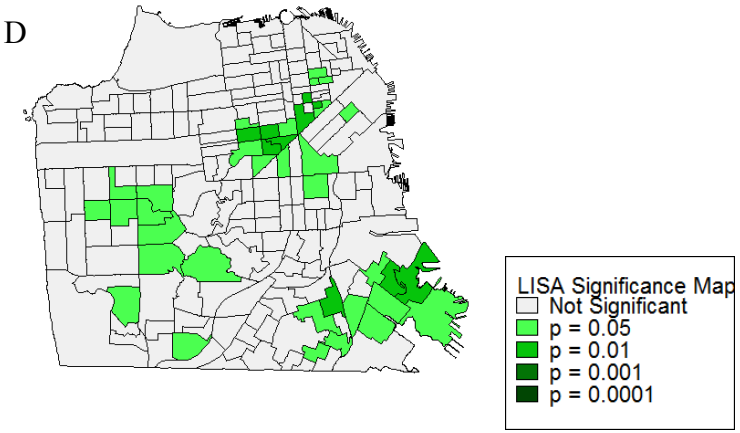


Table 6A: Effect Estimates of Neighborhood Education on Overweight/Obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009 (Sources: ACS, CHIS).

	OLS (approx. mixed model)					Spatial Lag				
			95% CI					95% CI		
	Estimate	SE	Lower	Upper	p-value	Estimate	SE	Lower	Upper	p-value
Overweight or Obese:										
Neighborhood education estimate	-0.0001	0.0044	-0.0087	0.0085	0.9810	0.0004	0.0039	-0.0073	0.0081	0.9250
Spatial lag (ρ)						0.5097				0.0000
R^2 (percent of variance explained)	-0.0052									
AIC	-495.6433					-523.9000				
Log likelihood	250.8216					265.9502				
Breusch-Pagan test	17.2559				0.0000	12.1237				0.0005
Type 2 Diabetes:										
Neighborhood education estimate	-0.0109	0.0015	-0.0139	-0.0080	0.0000	-0.0083	0.0016	-0.0115	-0.0051	0.0000
Spatial lag (ρ)						0.3354				0.0007
R^2 (percent of variance explained)	0.2119									
AIC	-910.1526					-919.5400				
Log likelihood	458.0763					463.7717				
Breusch-Pagan test	12.7730				0.0004	12.5459				0.0004
Current Smoking:										
Neighborhood education estimate	-0.0119	0.0022	-0.0162	-0.0076	0.0000	-0.0075	0.0021	-0.0116	-0.0034	0.0003
Spatial lag (ρ)						0.5346				0.0000
R^2 (percent of variance explained)	0.1295									
AIC	-764.7521					-797.9100				
Log likelihood	385.3761					402.9573				
Breusch-Pagan test	5.1689				0.0230	4.8952				0.0269

All models adjust for individual-level covariates.

Table 6B: Effect Estimates of Neighborhood Income on Overweight/Obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009 (Sources: ACS, CHIS).

	OLS (approx. mixed model)					Spatial Lag				
			95% CI					95% CI		
	Estimate	SE	Lower	Upper	p-value	Estimate	SE	Lower	Upper	p-value
Overweight or Obese:										
Neighborhood income estimate	0.0016	0.0043	-0.0068	0.0101	0.7050	0.0028	0.0039	-0.0047	0.0104	0.4635
Spatial lag (ρ)						0.5130				0.0000
R^2 (percent of variance explained)	-0.0045									
AIC	-495.7881					-524.4300				
Log likelihood	250.8941					266.2142				
Breusch-Pagan test	15.8654				0.0001	10.1897				0.0014
Type 2 Diabetes:										
Neighborhood income estimate	-0.0099	0.0015	-0.0129	-0.0070	0.0000	-0.0073	0.0015	-0.0102	-0.0043	0.0000
Spatial lag (ρ)						0.3669				0.0002
R^2 (percent of variance explained)	0.1801									
AIC	-902.4710					-914.2500				
Log likelihood	454.2355					461.1240				
Breusch-Pagan test	16.5360				0.0000	17.0894				0.0000
Current Smoking:										
Neighborhood income estimate	-0.0160	0.0020	-0.0199	-0.0120	0.0000	-0.0111	0.0020	-0.0151	-0.0072	0.0000
Spatial lag (ρ)						0.4424				0.0000
R^2 (percent of variance explained)	0.2445									
AIC	-792.2458					-812.8900				
Log likelihood	399.1229					410.4445				
Breusch-Pagan test	4.7418				0.0294	3.8187				0.0507

All models adjust for individual-level covariates.

Figure 25: Estimated Effect of Neighborhood Education on Overweight/obesity, Type 2 Diabetes, and Current Smoking, Accounting for Spatial Lag. San Francisco Adults 2005, 2007, 2009.

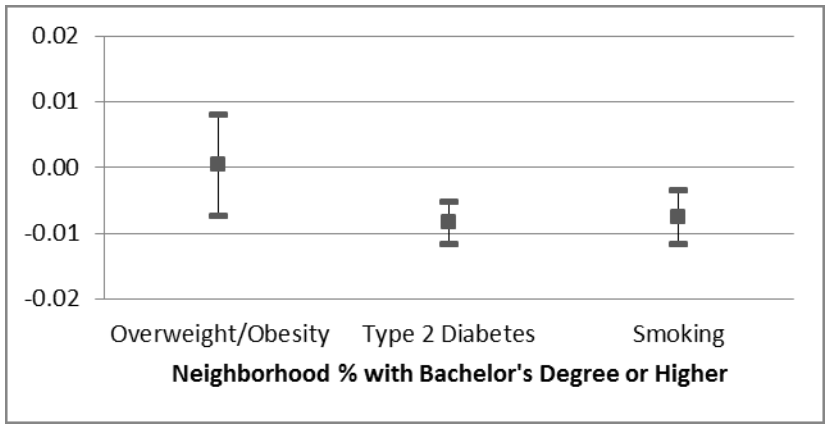
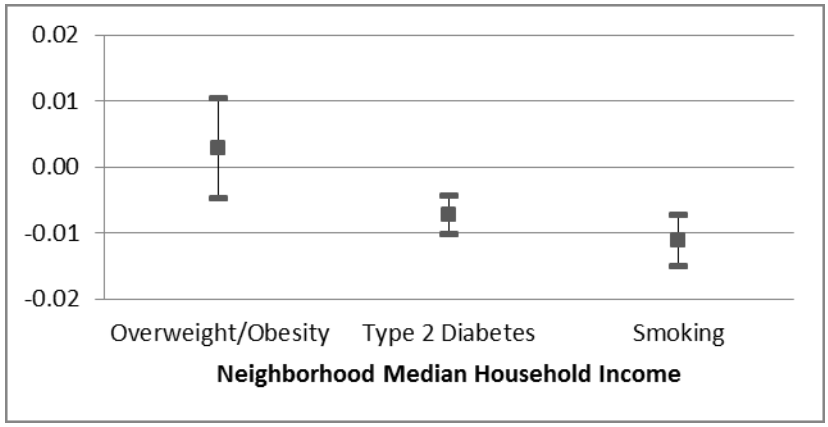


Figure 26: Estimated Effect of Neighborhood Income on Overweight/obesity, Type 2 Diabetes, and Current Smoking, Accounting for Spatial Lag. San Francisco Adults 2005, 2007, 2009.



Similarly, neighborhood income is not statically significant for overweight/obesity in the OLS and spatial lag models, but is significant for diabetes and smoking in both models. The spatial lag model suggests that a one-quartile increase in neighborhood median household income causes a 0.007 (95% CI: -0.010 to -0.004) decrease in prevalence of type 2 diabetes and a 0.011 (95% CI: -0.015 to -0.007) decrease in prevalent smoking, accounting for spatial spillover. The spatial lag coefficient suggests significant spatial spillover of outcomes across neighborhoods: a one-unit change in the outcome in nearby neighborhoods is associated with a 0.367 (p-value<0.000) change in diabetes and a 0.442 (p<0.000) change in smoking in the focal neighborhood.

The significant spatial lag term coefficient for overweight/obesity in the neighborhood education model (0.510, p-value<0.000) and the neighborhood income model (0.513, p-value<0.000) suggests spatial interaction between neighbors, while the non-significant neighborhood exposure terms imply that this spatial dependence is due to unobserved factors.[49]

Incorporating spatial dependence (i.e. moving from aspatial to spatial multilevel regression) in the diabetes and smoking models attenuated the neighborhood SES estimates and affected the standard errors; however, these changes were slight and did not affect statistical significance. In the overweight/obesity models the (non-significant) estimates increased modestly while the standard errors decreased slightly.

Based on Akaike information criterion (AIC) and log likelihood (LL) all spatial lag models provide a better fit (i.e. lower AIC and higher log likelihood values) to the data than the OLS model, despite the addition of one variable to the spatial models

(namely the spatial lag term). With the exception of the neighborhood income and diabetes model, Breusch-Pagan test estimates decreased in the spatial lag models, suggesting a reduction in heteroskedasticity; however, the significance of these tests implies that heteroskedasticity is still present that is due to non-spatial issues or spatial issues not accounted for by the spatial regression.

We further explored the potential for model misspecification in the spatial models starting with the LM test (which is derived from the OLS model results). All LM statistics were statistically significant for the error and lag models, suggesting potential model improvement with a spatial model, so we examined the robust LM statistics (Table 7). Given statistically significant estimates, the robust tests clearly suggested the lag model (over the OLS and spatial error models) for neighborhood income with the diabetes and smoking outcomes. Results for the other models are less clear. The robust spatial lag and spatial error estimates both failed to reach significance and in many cases were very similar, both of which suggested model misspecification.[146] However, this provides no information as to how the models might be misspecified (e.g. due to spatial weights or missing covariates or some other feature of the data). Compared to the original spatial lag models, the spatial error models (not shown) provided similar estimates and slightly wider confidence intervals although no change in statistical significance, as well as similar model fit based on AIC and LL estimates.

To better understand potential spatial processes and causes of misspecification, we examined residuals using residuals maps, Moran's I scatterplots, and LISA cluster and significance maps (Figures 27-32 for spatial lag models; spatial error models not shown).

Table 7: Lagrange Multiplier (LM) Test for Spatial Error and Spatial Lag of Health Outcomes Regressed on Neighborhood (Census Tract) Education and Median Household Income, San Francisco Adults, 2005, 2007, 2009 (Sources: ACS, CHIS).

	Overweight or Obese			Type 2 Diabetes			Current Smoking		
	Estimate	df	p-value	Estimate	df	p-value	Estimate	df	p-value
Neighborhood % with bachelor's degree or higher									
LMerr	42.3024	1	0.0000	13.4102	1	0.0003	48.6863	1	0.0000
LMlag	42.3080	1	0.0000	13.4324	1	0.0002	46.2764	1	0.0000
RLMerr	0.0984	1	0.7537	0.5020	1	0.4786	2.6030	1	0.1067
RLMlag	0.1040	1	0.7471	0.5242	1	0.4691	0.1931	1	0.6604
Neighborhood median household income									
LMerr	43.1954	1	0.0000	9.9747	1	0.0016	16.2595	1	0.0001
LMlag	42.6881	1	0.0000	16.1851	1	0.0001	26.7949	1	0.0000
RLMerr	3.0474	1	0.0809	2.3630	1	0.1242	2.0208	1	0.1552
RLMlag	2.5402	1	0.1110	8.5733	1	0.0034	12.5562	1	0.0004

Figure 27: Residual Analysis from Spatial Lag Regression of Neighborhood Education on Prevalence of Overweight/obesity in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.

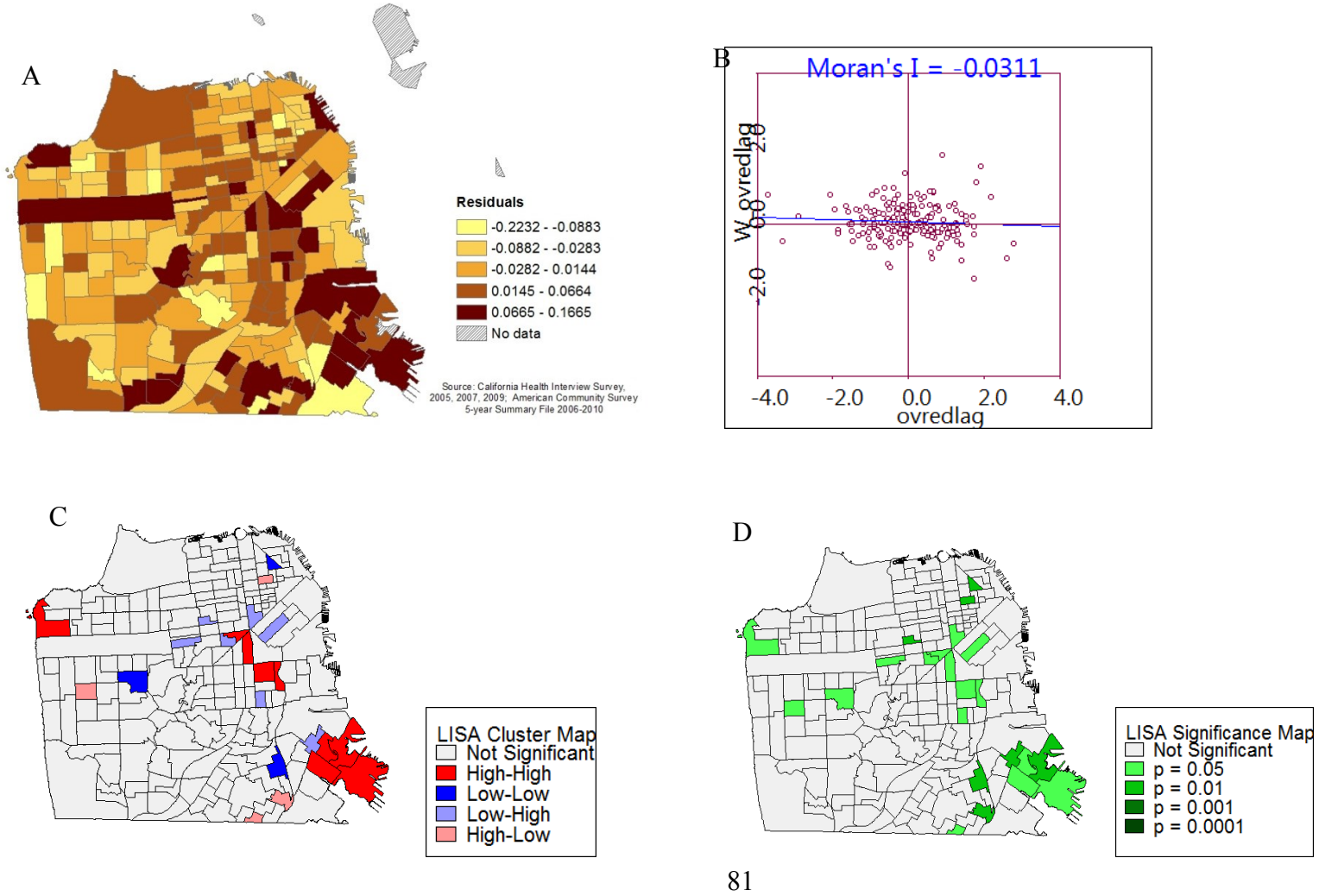


Figure 28: Residual Analysis from Spatial Lag Regression of Neighborhood Education on Prevalence of Type 2 Diabetes in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.

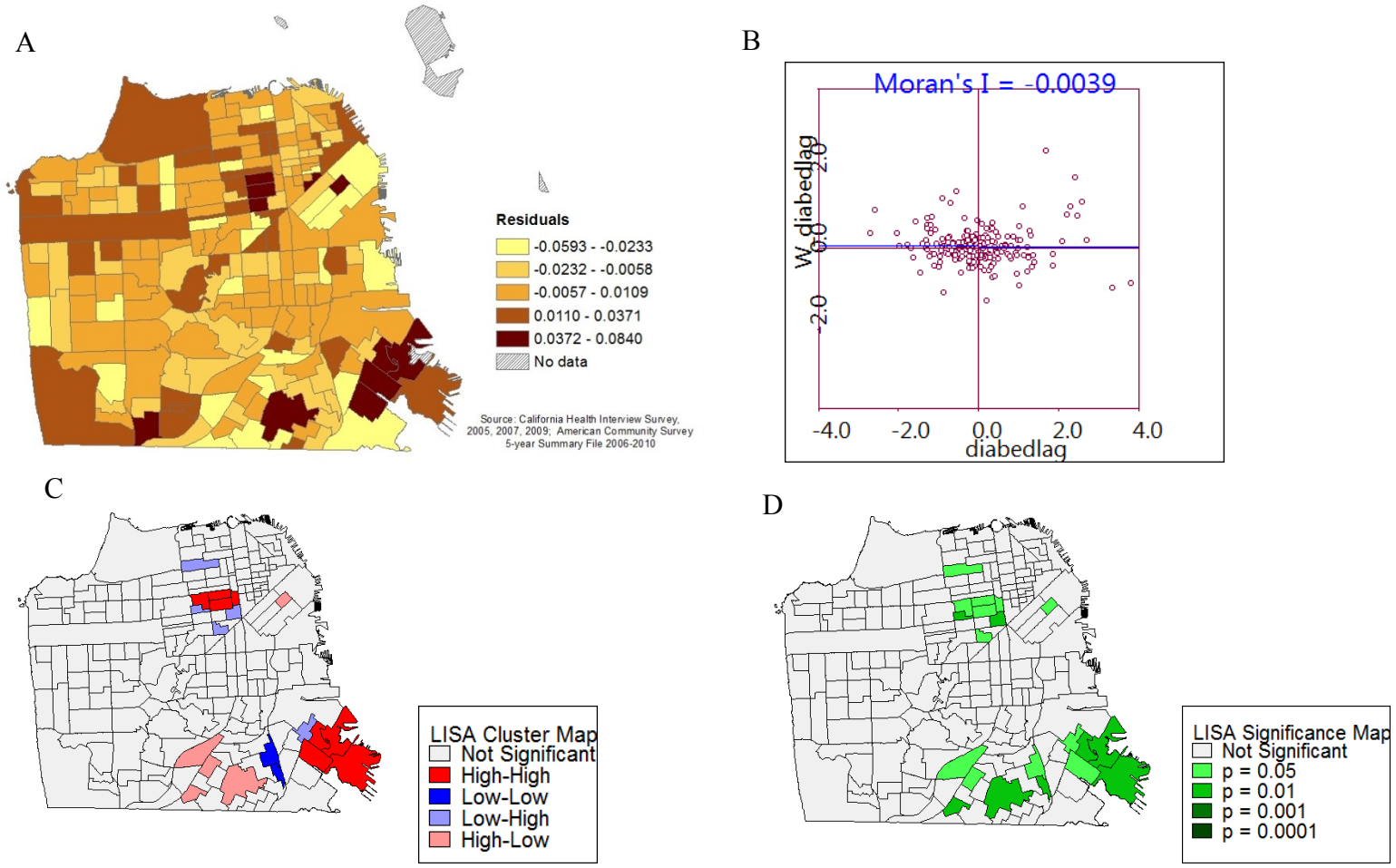
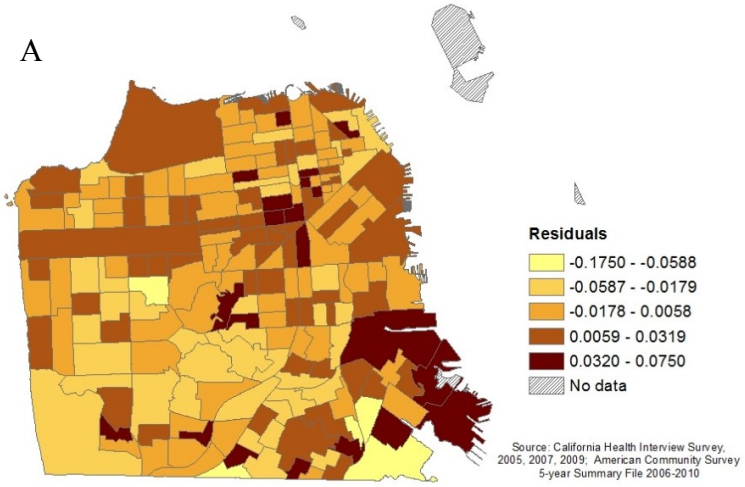
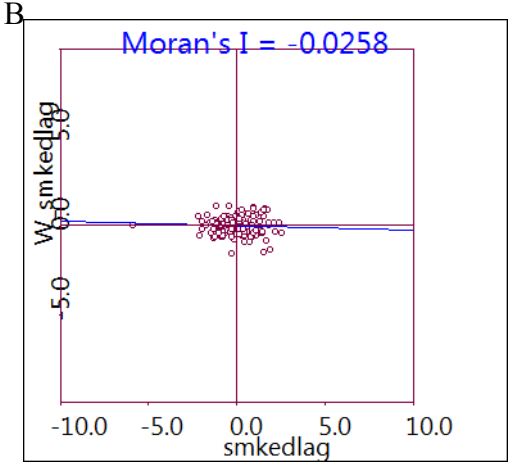


Figure 29: Residual Analysis from Spatial Lag Regression of Neighborhood Education on Prevalence of Current Smoking in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.

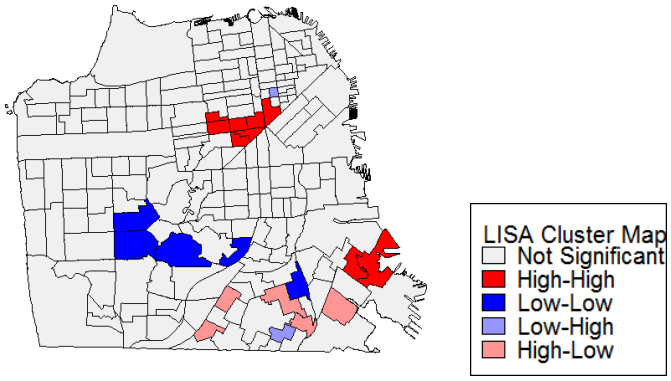
A



B



C



D

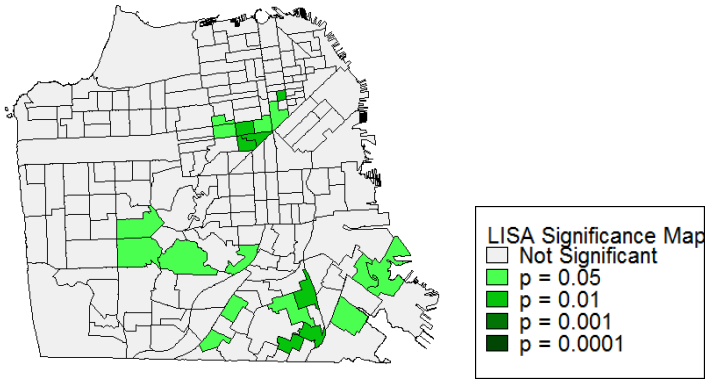


Figure 30: Residual Analysis from Spatial Lag Regression of Neighborhood Income on Prevalence of Overweight/obesity in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.

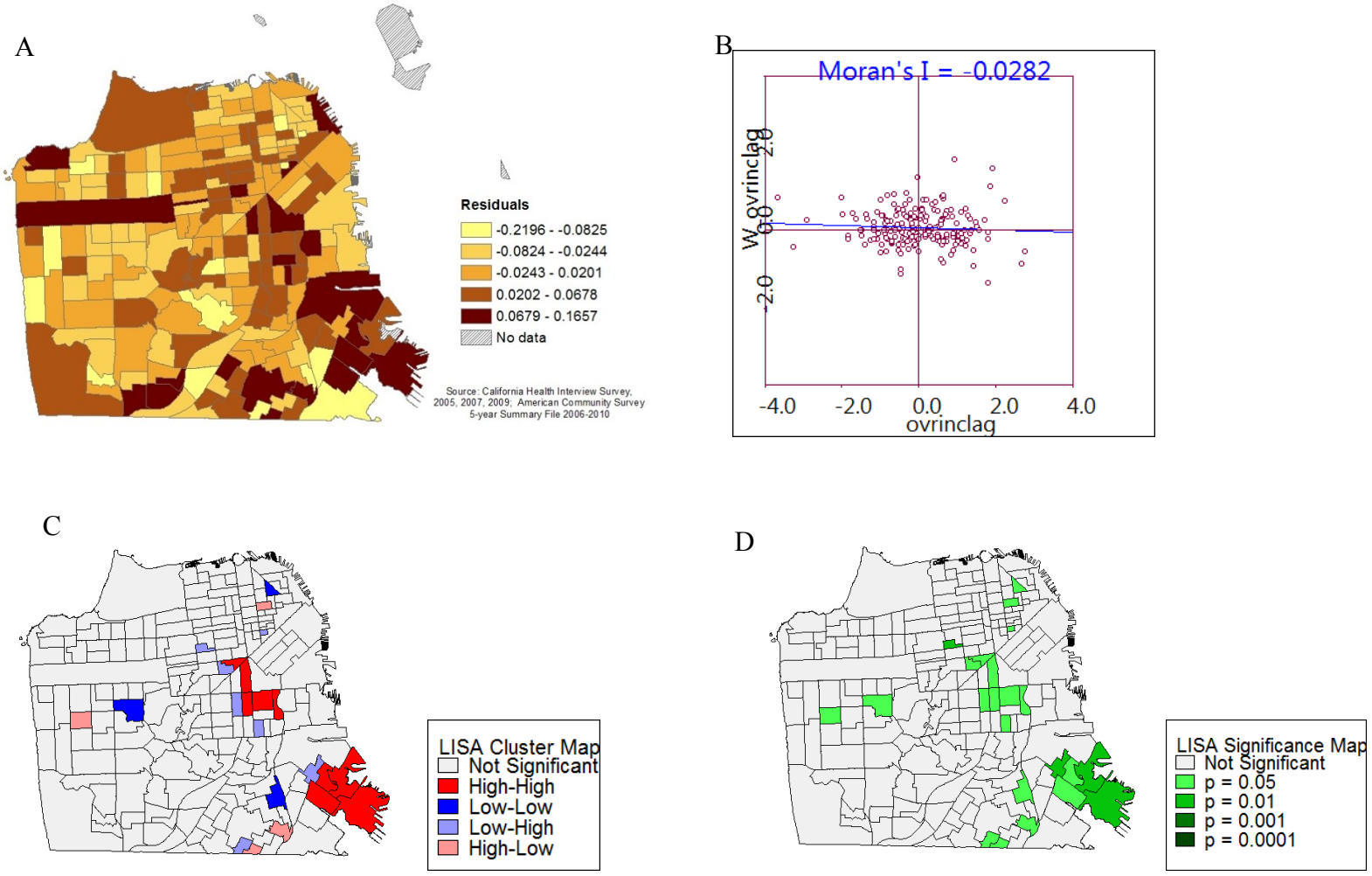


Figure 31: Residual Analysis from Spatial Lag Regression of Neighborhood Income on Prevalence of Type 2 Diabetes in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.

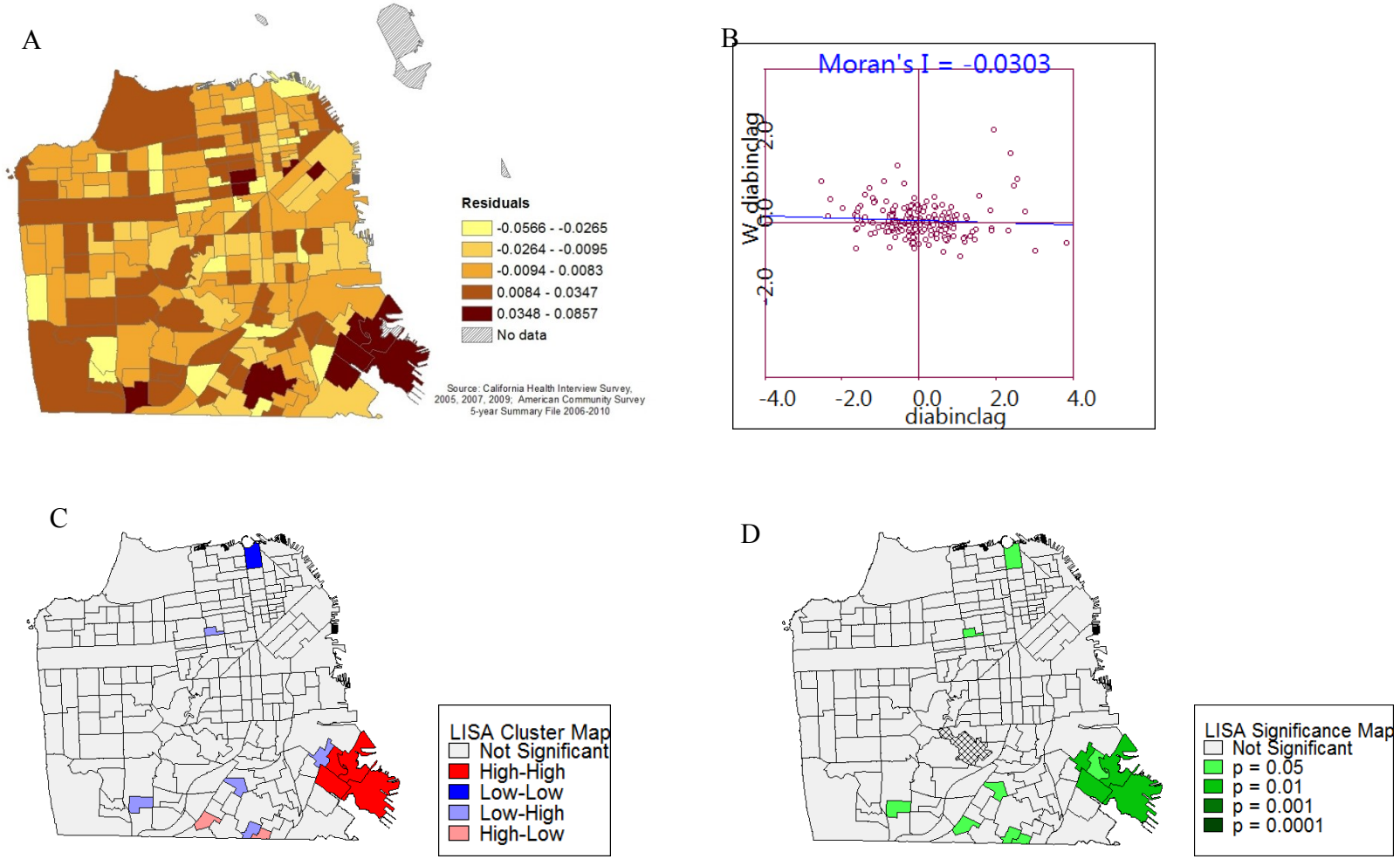
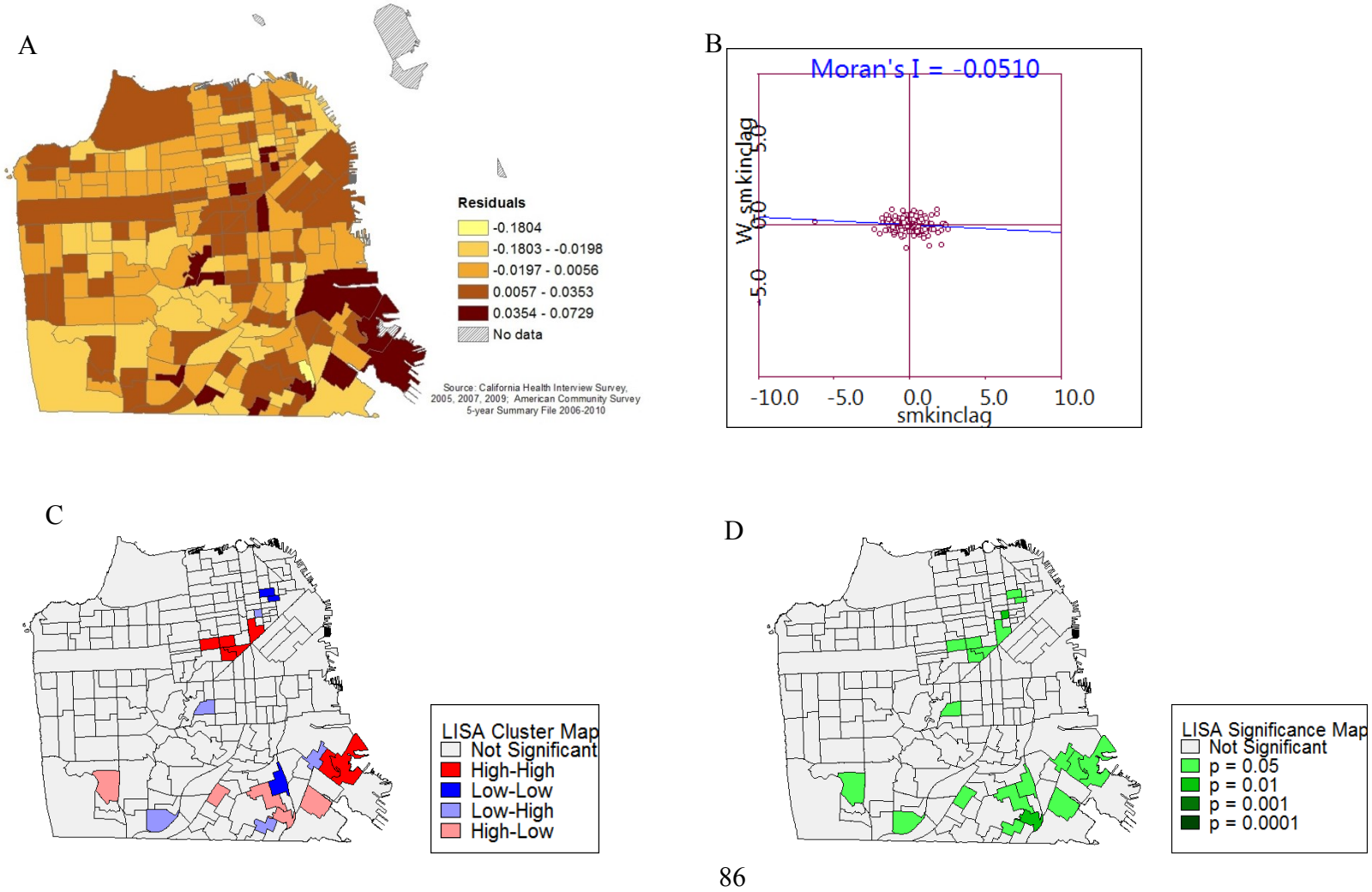


Figure 32: Residual Analysis from Spatial Lag Regression of Neighborhood Income on Prevalence of Current Smoking in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS): A)Residuals, B)Moran's I Scatterplot, C)LISA Clusters, D)LISA Significance.



These show less spatial clustering in both the spatial lag and error models (compared with the OLS models). Moran's I tests of the residuals suggested no significant clustering in the residuals except for the neighborhood education/diabetes spatial error model (Table 5). LISA maps suggest significant clustering of the residuals in some portion of the Bayview/Hunters Point neighborhood in all spatial lag and error models, except for the neighborhood education and diabetes spatial error model. (Although clustering of residuals may exist in other areas, this clustering was not consistent across the models.) Care should be taken not to attach too much weight to the LISA significance estimates, as they are subject to the multiple comparisons problem.[148] Although no clear solution exists to deal with this problem in LISA, the Bonferroni correction provides a very conservative approach,[148] which for this study suggests a significance cutpoint of 0.0003 (alpha of 0.05/194 neighborhoods). Even if we use a less conservative approach by a factor of about 10, the LISA maps suggest no remaining significant residual clusters at 0.005.

Given the potential for a 'small numbers problem,' we also conducted a weighted aspatial (OLS) multilevel regression to account for the different number of observations in each census tract. Weighted regression revealed estimates very similar to the standard OLS estimates, suggesting that the variable number of observations and potential instability in rates are unlikely to significantly influence the results of the spatial models.

4.4 Discussion

This study supports findings from previous studies, which generally suggest that lower neighborhood SES is associated with higher prevalence of type 2 diabetes[55, 57, 63]

and smoking.[65-73] We estimated a significant inverse effect of increasing neighborhood education and income on type 2 diabetes and smoking, adjusting for differences in individual characteristics, in both the approximated aspatial and spatial multilevel models. However, these significant estimates suggest only a minor improvement in health outcomes with a move to the next higher neighborhood SES quartile.

Conversely, we failed to find evidence of an effect of neighborhood SES (education or income) on overweight/obesity, although several previous studies found evidence of this association.[57-60] Many different factors might explain the lack of significance for overweight/obesity as compared to diabetes and smoking (and other obesity studies), such as higher measurement error for self-reported BMI, our inclusion of overweight with obesity, or the potential inability of neighborhood education or income to capture a more complex set of influences on overweight/obesity.

We found evidence of spatial autocorrelation of residuals in all approximated aspatial (i.e. OLS) multilevel models, suggesting spatial dependence in the relationships of neighborhood education and income with overweight/obesity, type 2 diabetes, and current smoking, accounting for individual-level differences. This implies that ignoring the spatial autocorrelation could bias effect estimates and affect estimate precision. Compared to the aspatial models, the spatial lag models with significant estimates provided weaker exposure estimates and similar standard errors, while the models with non-significant estimates (i.e. overweight/obesity) had stronger exposure estimates and smaller standard errors. However, the differences were small and statistical significance did not change with incorporation of a spatial lag. Spatial spillover of neighborhood SES

could plausibly reduce the effect estimate – which represents the estimate for the focal neighborhood – and redistribute some of the total effect to nearby neighborhoods.[146] For example, as those residing in lower-SES neighborhoods travel across neighborhood lines to access health-promoting resources in a higher-SES neighborhood, the effect of their own neighborhood SES lessens.

The significant spillover effects together with the significant coefficients for the corresponding neighborhood exposure variables suggest that the education and income of neighboring areas affect diabetes and smoking prevalences in the focal neighborhoods. We also found evidence of significant spatial spillover in the neighborhood education and income models for overweight/obesity. However, since the effect estimates were non-significant, this suggests that this spatial dependence may be due to unobserved variables.

Despite any statistically significant estimates, close examination of model fit parameters, LM estimates, and model residuals provided evidence of misspecification in all models, although less so for neighborhood income models for diabetes and smoking. Model misspecification may bias the results, so estimates should be interpreted with extra caution. Furthermore, the model estimates and fit for the spatial lag and error models were quite similar. Thus, it is unclear whether a lag process is actually occurring or if the spatial dependence occurs due to nuisance factors (e.g. due to a missing variable).

Our exploratory analyses suggest potential model improvements for future work. As noted earlier nearly all spatial models exhibited spatial clustering of the residuals in the Bayview/Hunters Point neighborhood. Bayview/Hunters Point abutts an old naval shipyard in the southeastern corner of San Francisco and is historically African

American. This neighborhood includes the three census tracts with the highest concentration of African Americans in the city (65-71% African American).[103] This suggests the models might be refined by adding a neighborhood percent African American variable; however, this could complicate our ability to extract policy-relevant recommendations given the entanglement of race/ethnicity with SES. Including both neighborhood education and income in the same models might also improve the models; however, the same limitation applies as with including African American race. Clustering of the spatial model residuals in other areas was not as consistent across models; however, elevation may be another factor that could enhance the models. San Francisco experiences rapid elevation changes throughout the city that might explain spatial health patterns over and above individual and neighborhood SES factors accounted for in this study.

In general, estimating etiologic effects in spatial models is challenging, so this study has several additional limitations. A fundamental challenge with using spatial lag and error models for neighborhood effects studies is the (current) necessity of choosing between point data and area (i.e. polygon) data, when one has both types of data.

We chose the spatial lag approach using neighborhood area data for several key reasons. First, we were interested in estimating an etiologic effect, which spatial econometrics models attempt to approach. By contrast, the more commonly-used geostatistical techniques create continuous surfaces from point data but generally provide predictive models and interpolation surfaces. Second, given our interest in how neighborhood SES affects individual-level health, the ideal experiment (if we could run

it) would be a community trial where we change neighborhood-level SES. Third, our neighborhood exposure data came from area-level census data and disaggregating the data to points involves the perhaps unsupportable assumption of a homogeneous population spread equally across the area. Finally, we were interested in quantifying any spatial spillover, which the spatial lag model allows. Given the necessity of having only area-level data for the spatial lag (and error) models, we approximated a multilevel model by adjusting tract-level outcomes for differences in individual covariates before aggregating to the tract. This approximated multilevel model masks variability within the tracts and thus likely decreases the true precision of the estimates. However, this crude approach allowed us to incorporate individual level data into an otherwise area-level model and minimize selection bias, while also eliminating the need to account for correlation of individuals within each neighborhood as in standard multilevel models. Importantly, this limits the potential for the ecological fallacy, so we can speak to how neighborhood education or income affects individuals.

This study is also subject to a 'small numbers problem' due to aggregated data and small numbers of observations per area for some neighborhoods. However, comparison of OLS regression results with those from a weighted OLS model revealed little change when accounting for the number of observations in each tract. Although various smoothing techniques are often used to reduce instability of estimates due to small numbers, this requires using the existing data to interpolate data based in part on spatial relationships. This may be especially helpful for predictive models, but could introduce spatial autocorrelation artifacts which might muddle our ability to identify true

causal effects.[45] Another limitation is that we did not incorporate information from neighboring areas outside of San Francisco, which could create edge or boundary effects.[38] However, San Francisco is bordered by major water bodies on three sides, limiting potential problems to the southern side of the city.

Geographers and other social scientists have been debating for decades how to best define a neighborhood.[149] By defining a neighborhood using administrative units we create neighborhood boundaries where they might not exist. However, using the spatial lag model allows an effect in the focal neighborhood to spill over into nearby neighborhoods, thus capturing effects at a broader scale and minimizing the potential for MAUP. Furthermore, the neighborhood exposure data was recorded by tract, and use of a buffer around an individual's residence cutting across different tracts would require an unsubstantiated assumption that the population is evenly spread and homogeneous across the entire tract.

One key challenge to both spatial and aspatial observational neighborhood effects studies is the assumption that contextual effects can be disentangled from individual effects. We chose a study area with a densely populated but relatively small urban area where individuals could potentially live in most neighborhood SES quartiles in order to improve exchangeability across exposure groups and lessen this identification problem (or "reflection problem").[87] (For example, San Francisco has middle-class homes with in-law apartments that may be rented to those in a lower SES.) However, identifying whether the neighborhood affects the individual, the individual affects the neighborhood, or a combination of the two, and the effect of any one part may not be possible with

observational data.[87] The spatial spontaneity problem of spatial lag models is very similar, which – although statistically addressed here by using maximum likelihood estimation – does also seem to limit our ability to make causal inferences from these spatial regression models.

Finally, a limitation specific to using CHIS data is that outside researchers are not allowed direct access to the individual-level CHIS data. All code for analyses involving individual-level CHIS data were submitted to CHIS staff to run. This restriction necessitated meticulous planning by the authors to minimize back and forth and allow for adequate turnaround times.

Despite these limitations, using the approximated spatial multilevel model provided this neighborhood effects study with several key strengths. First, addressing spatial dependence likely reduced bias of the effect estimates and in some models improved precision as compared to aspatial models. Second, we were able to explicitly examine the presence and strength of spatial spillover across neighborhoods while also accounting for differences in individual-level characteristics. Understanding spatial spillover not only provides useful information for interventions but also reduces the potential for MAUP. Third, most neighborhood effects studies fail to account for spatial dependence or examine spatial spillover. Of those studies that do address spatial dependence, most choose to convert any area-level data to points for use in geostatistical models, which have their own assumptions and limitations. We provide a relatively uncommon example of using spatial lag models in neighborhood effects studies of health outcomes to account for spatial dependence and examine spillover effects. Although

work by Morenoff[49] a decade ago highlighted the potential benefits of spatial lag models for neighborhood effects research, very little work in public health has followed his lead to examine spillover effects (a recent exception is work by Chen and Wen[42]). The use of a spatial error model to treat the spatial dependence as a nuisance to be accounted for is perhaps slightly more common than spatial lag models (see e.g. Duncan et al[150]). Part of this resistance to incorporate spatial econometric models into this area of health research may be the substantial increase in complexity that they bring with them compared to traditional OLS and multilevel models, such as by requiring modeling of the spatial relationships via a spatial weights matrix and data aggregation to approximate multilevel models.

Additional strengths of this study are the use of high-quality data from CHIS and ACS, and the similar timeframe of these datasets. Single-variable measures of neighborhood SES provide policy makers with information that is easy to understand, instead of complex neighborhood indices that are challenging to translate into interventions. Our study design limits the potential for confounding at supra-county level, as the study area is one complete county.

In summary, this study approximated spatial multilevel models to estimate the effects of neighborhood SES on overweight/obesity, type 2 diabetes, and current smoking. Results from the spatial lag models estimated a significant effect of neighborhood SES on type 2 diabetes and smoking (but not overweight and obesity), and suggest that nearby neighborhoods influence the effect of neighborhood SES on health outcomes in the focal neighborhood. Although reaching statistical significance, our

results suggest only a minor improvement when moving to a higher SES neighborhood: a decrease in disease prevalence of around 1 in 100. Compared to the aspatial multilevel models, the spatial multilevel models decreased bias, provided better model fit, and found evidence suggesting spatial dependence and perhaps even spillover. Despite their differences the conclusions drawn from both models are fundamentally the same. Perhaps given the potential problems with model misspecification, the biggest benefit of using the spatial multilevel models in this study is as a diagnostic tool to help point to potential sources of model misspecification, especially missing variables. We may be able to then improve future models by adding specific variables or by examining a larger study area. Future research should also use more powerful cutting edge spatial models that attempt to incorporate both spatial effects and spatial heteroskedasticity.[135] However, our ability to draw etiologic conclusions from neighborhood effects studies continues to be limited by our inability to truly disentangle contextual effects from compositional effects.

5. Comparing Social and Spatial Epidemiology Perspectives

Social and spatial epidemiologists each bring a unique perspective to contextual effects studies, leading to different analytical approaches and potentially different conclusions. No study has compared these perspectives and their approaches; however, this comparison may advance contextual effects research in epidemiology by suggesting methodological improvements or providing insights into our ability to identify contextual effects. Successfully identifying contextual determinants of health may improve contextual-level interventions, which may curb major public health problems at a broader level than individual-level interventions.

We compared the social and spatial epidemiology perspectives by examining their grounding theory, foci, goals, and key challenges in contextual effects research. Our main research question was: How are the social and spatial epidemiological perspectives on contextual effects questions different, and what can we learn from comparing the two perspectives? We also addressed the following contextual effects question: What is the estimated effect of increasing neighborhood education or income on overweight/obesity, type 2 diabetes, and current smoking, independent of individual-level differences? In this study we ran a traditional multilevel model (generalized estimating equations) to address this question. We then compared these results with the results from previous work asking the same research question but using a social epidemiology analytical approach with a propensity score matching (PSM) model and a spatial approach with approximated spatial multilevel models.

Spatial epidemiology is perhaps best differentiated from social epidemiology by its strong connection to medical geography, which develops models to predict the reality of a specific place. By contrast, social epidemiologists tend to ask causal questions placed within a causal counterfactual framework to identify modifiable factors generalizable to a broader population.

Across models from all approaches (social, spatial, traditional) we see little to no evidence of an effect of neighborhood education and income on overweight/obesity, type 2 diabetes, and current smoking. Effect estimates and confidence intervals changed somewhat when the sample or analytical technique changed.

Given the fundamental differences in the two perspectives, we found few possibilities for methodological advancements through integration of analytical techniques. Furthermore, comparing the social and spatial epidemiology perspectives highlighted the unlikelihood of identifying causal contextual effects using observational data. However, strong social and spatial observational studies can still indirectly improve our ability to identify causal contextual factors by aiding in the design of more efficient and effective community trials.

Keywords: social epidemiology, spatial epidemiology, neighborhood effects, contextual effects

5.1 Introduction

Social epidemiologists and spatial epidemiologists both ask causal or etiologic contextual effects questions, but each brings a unique perspective to answering these questions. Contextual effects – or neighborhood, place, or area effects – research examines characteristics of the physical and/or social environment hypothesized to affect an individual's health.[4, 17] The social epidemiology perspective focuses on the effect of social contexts or structures on health and places questions within a causal framework. The spatial perspective focuses on the spatial distribution of health outcomes and contextual risk factors and typically develops predictive models. These different perspectives influence the analytical approaches and potentially the conclusions drawn from contextual effects research.

Although social and spatial epidemiologists often still use a traditional multilevel model (i.e. hierarchical or multilevel model) when examining contextual effects questions, both are increasingly employing cutting-edge analytical approaches in their subfield to address some of the challenges of effect identification at the contextual level. In recent years social and spatial epidemiologists have acknowledged challenges to contextual effects research identified by the other subfield, including spatial scale and dependence[1, 4, 8, 23, 39, 82-84, 125] and social processes,[41, 42] but most work remains focused on addressing the key challenges recognized within one's subfield.

Despite some work comparing social and spatial analytical techniques or results with a traditional multilevel model (and far less often with each other),[39, 41, 42, 48, 49, 82-84] to our knowledge no other study has explicitly compared the social epidemiology

perspective with the spatial epidemiology perspective. Furthermore, we know of no study using cutting-edge analytical techniques for both analytical approaches. Previous studies do suggest that taking a social or spatial approach compared with a more traditional multilevel regression approach may influence results and provide a more comprehensive understanding of the relationships under study, but may not necessarily change conclusions.[39, 48, 55, 82-86]

The present study moves beyond a simple comparison of analytical techniques and results from a social approach using propensity score matching (PSM) or a spatial approach using an approximated spatial multilevel model with those from a traditional multilevel model. Our research question was: How are the social and spatial epidemiological perspectives on contextual effects questions different, and what can we learn from comparing the two perspectives? We seek to better understand the overarching social and spatial epidemiology perspectives and how these perspectives influence analytical approach, model inference, and our ability to identify causal contextual effects. Comparing these perspectives may yield new insights that can strengthen causal inference in contextual effects studies. Indeed, Kawachi previously issued the call to make social epidemiology "the equivalent of an economic free zone[…] where enterprising investigators can shed their disciplinary baggage at the border, set up shop, and fruitfully exchange their ideas to enrich the field of the social determinants of health" (p.1741).[3] Directly comparing these sub-disciplines may be challenging. As Kuhn's theory of incommensurability suggests, we should not expect direct translations between the work of social epidemiologists and spatial epidemiologists.[80, 81] Referring to

incommensurability Chen notes: “scientists from rival paradigms usually have different understandings of the goals of science and frequently have conflicting interests in the development of science” (p.264). However, we hypothesized that critically examining these perspectives and their analytical approaches may help us identify methodological enhancements, which could advance identification of contextual effects in future research, and thereby improve our ability to effectively intervene on major public health problems at a broader scale.

We start by identifying key theoretical elements from the social and spatial perspectives. In the Methods section we outline the social and spatial analytical approaches used in previous work and the need to compare them through traditional multilevel models, which thus serve as intermediary models. Next we present results from the intermediary models and our previous analyses using social and spatial approaches in the Results section, and finally compare perspectives, model results, and approaches in the Discussion.

5.2 Social Epidemiology Perspective

Social epidemiology examines the emergence and distribution of social phenomena and their effect on human health. Central to social epidemiology is the notion that social conditions, structures, and interactions may create differential exposures leading to differential health outcomes in the population.[26] The goal is to identify social determinants of health and promote interventions on these factors to improve population health.[3, 26]

Social determinants occur at many different levels, including at the neighborhood level (i.e. contextual level). When social epidemiologists examine contextual effects questions, they build from a social ecological framework. This framework pulls from theory in many fields and identifies multiple, interrelated levels (e.g. individual, contextual) of influence on health, with contextual influences including features from the social and physical environment.[4, 20, 151] Social epidemiologists also refer to a multilevel conceptual framework based on the work of Coleman (i.e. the "Coleman bathtub"), which focuses on the influence of macro-level factors on micro-level factors and micro on macro over time.[26]

Much of the theoretical support for studying the effect of contextual factors on health comes from sociology. The theory behind neighborhoods as distinct, spatially-defined units worthy of study developed from the Chicago school of urban sociologists.[4, 152] The Chicago school proposed that a neighborhood has emergent properties, distinguishable from the sum of the properties of individuals living within the neighborhood.[129] Furthermore, theory on social organization and structuralism argues that social processes segregate and sort individuals by social class and race/ethnicity, so that different types of people reside in different types of neighborhoods.[4, 40] This social stratification results in differential exposure to contextual-level factors, including social and material resources,[6] which may affect health. In fact, evidence suggests that those living in disadvantaged neighborhoods have the poorest health outcomes.[4-8] Thus, social structures are a fundamental focus in social epidemiological studies of neighborhood effects; however, social stratification may lead to the problem of structural confounding.

When social stratification produces exposure groups with fundamentally different (i.e. non-comparable) individuals, this creates a data problem called structural confounding. Without similar individuals in different neighborhoods we lack the data to support inference, and adding more data (i.e. individuals or observations) fails to solve the problem as this merely adds more data where data already exists.

Structural confounding presents a critical challenge to the identification of causal contextual effects. This is perhaps best understood when contextual effects are framed within the counterfactual theory of causation, which social epidemiologists often use. Given an observable person or population exposed to treatment condition X with outcome Y, the counterfactual is the *un*observable outcome in the same person or population if they had *not* been exposed to treatment condition X at the very same time with all else being equal.[34-36] Clearly two mutually exclusive treatment conditions cannot exist in the same person at the same time, so epidemiologists attempt to approximate the counterfactual as best as possible. The ideal experiment is designed around the counterfactual, where one group is assigned the treatment and another group which is as similar as possible to the first group remains untreated.[2, 35] In observational contextual effects studies where the researcher cannot control the treatment assignment, the researcher typically adjusts for potential confounders in a multilevel regression model in an attempt to balance the covariates of those in the 'treated' group with those in the 'untreated' group as would be expected under randomization.[34] Although regression attempts to make individuals more comparable by creating more strata (i.e. adjusting for more potential confounders), with more covariates it becomes increasingly unlikely that

individuals from the same strata will exist in the exposed and unexposed groups.[2, 8] This inhibits positivity as well as the exchangeability of individuals across neighborhood exposures, and thereby limits effect identification.[1, 2, 8, 29, 32, 33]

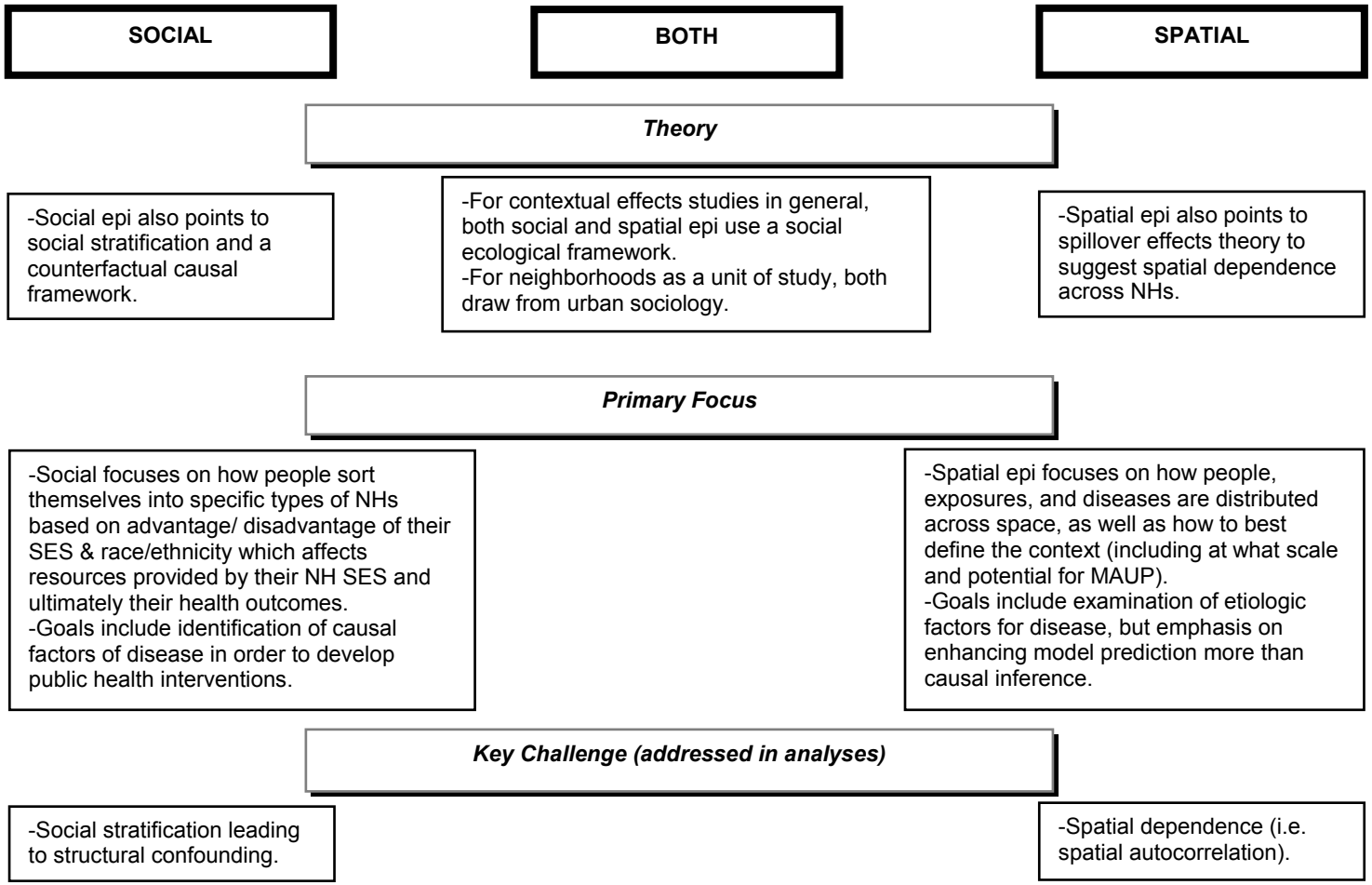
Unfortunately, structural confounding is common in neighborhood effects studies as is the use of regression analysis,[29-31] which does not solve structural confounding and leads to off-support inference. For social epidemiologists structural confounding is a key challenge that needs to be overcome to better approximate the ideal experiment and achieve the general goal of epidemiologists to identify causal factors affecting health. Social epidemiologists increasingly use propensity score matching to pre-process observational data and explicitly address the challenge of structural confounding.

Figure 33 summarizes key elements of the social epidemiology perspective, as well as the spatial epidemiology perspective.

5.3 Spatial Epidemiology Perspective

Spatial epidemiology examines and describes the spatial distribution of disease, risk factors for disease, and the intersection of the two both visually and statistically using geographically-referenced data.[142] Spatial epidemiologists are interested in a wide range of risk factors including: "demographic, environmental, behavioral, socioeconomic, genetic, and infectious risk factors" (p.998).[142] Spatial epidemiology combines theory and work primarily from epidemiology and medical geography. Although research in medical geography and spatial epidemiology clearly overlaps, spatial epidemiology appears to extend the analytical techniques of medical geography by explicitly incorporating more epidemiologic techniques, while maintaining a clear interest in space and place.

Figure 33: Social and Spatial Perspectives on Contextual Effects Studies



Much work in medical geography follows the triangular human ecology population-habitat-behavior model,[153] which highlights the importance of contextual factors to medical geographers and spatial epidemiologists. As with social epidemiology, spatial epidemiology also places contextual effects research within a social-ecological framework and uses urban sociological theory to justify the study of neighborhoods as spatially distinct units with emergent properties.

Given their distinct focus on how place affects health, spatial epidemiologists are addressing increasingly complex spatial challenges and attempting to better incorporate spatial complexity into contextual effects studies. One key challenge for spatial epidemiologists is spatial dependence, which relates to the unofficial first rule of geography from Tobler: "everything is related to everything else, but near things are more related than distant things" (p.236).[130] This dependence or clustering of observations across space is called spatial autocorrelation.[52, 53] Spatial researchers argue that a neighborhood is unlikely to be truly independent of its neighbors,[39-43] and that the effects from one neighborhood will spill over into surrounding neighborhoods and affect the health of those residents.[49, 51, 54] Spillover effects theory comes in part from spatial econometrics, which examines spatial autocorrelation and incorporates spatial effects into models.[51] Previous studies support the potential for spillover of the health effects of low neighborhood SES to surrounding neighborhoods.[54-56] Traditional multilevel models in contextual effects studies account for dependence of observations within a neighborhood but not between neighborhoods. Spatial epidemiologists address the challenge of spatial

dependence across neighborhoods in spatial models and often specifically address spillover effects.

Similar to social epidemiology the broad goal of spatial epidemiology contextual effects research is to examine contextual risk factors for disease; however, spatial epidemiology places less emphasis on causal inference and more on enhancing model prediction. This emphasis originates from geography, which promotes model fitting and prediction improvement (i.e. a less firm *a priori* hypothesis) throughout the analytical process in order to better understand what is occurring at a certain place. When attempting to improve model prediction, spatial researchers also consider the additional characteristics of the place under study when examining spatial patterns. For many spatial researchers context cannot or should not necessarily be distilled down to single variables. With spatial econometrics analytical techniques (such as spatial lag and error regression) medical geographers and spatial epidemiologists move in the direction of etiologic models by using an etiologic hypothesis; however, this work continues to be influenced by predictive models.

Although the social and spatial epidemiology perspectives share many similarities in their theoretical foundations of contextual effects studies, their differences lead each down different analytical paths when answering contextual effects questions.

5.4 Methods

This paper builds on the work of two earlier studies, one using a social epidemiology perspective and the other a spatial epidemiology perspective to answer the same basic contextual effects research question. Both studies use a cross-sectional design

with census tract (i.e. neighborhood) level exposure data from the 2006-2010 American Community Survey (ACS)[103] and individual-level outcome and covariate data pooled from the 2005, 2007, and 2009 California Health Interview Surveys (CHIS).[100-102] Pooling the CHIS surveys for the subject population – adults residing in the City and County of San Francisco – provides 2,515 individuals residing within 195 census tracts.

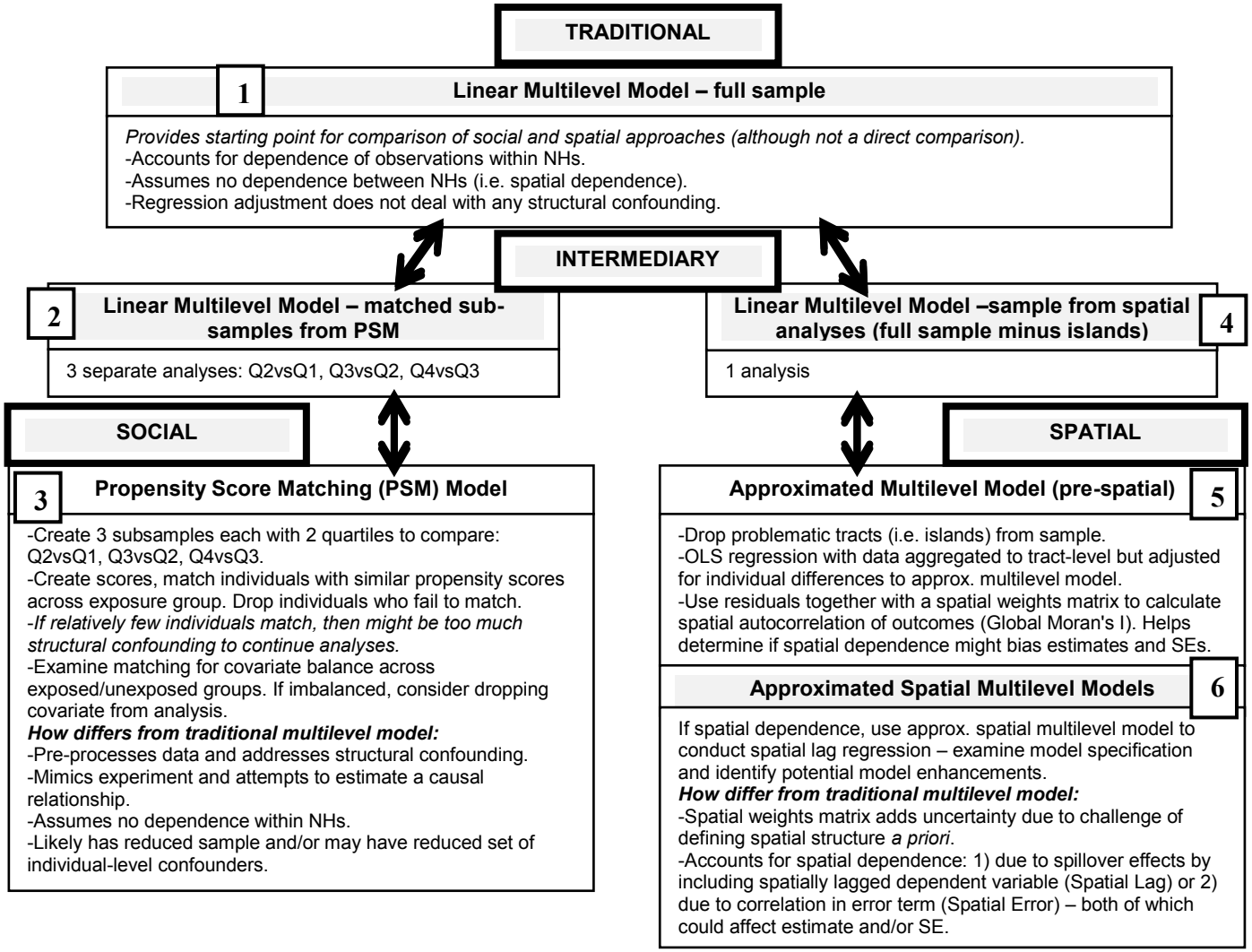
The exposure of interest was neighborhood socioeconomic status (SES), which was modeled separately using quartiles of 1) the percent of the neighborhood (census tract) with a bachelor's degree or higher (i.e. neighborhood education) and 2) neighborhood median household income (i.e. neighborhood income). The dichotomous outcomes were overweight/obesity, type 2 diabetes, and current smoking. All analyses accounted for a set of individual-level potential confounders associated with individual SES. Additional study design details can be found in the previous studies.

The University of Minnesota Institutional Review Board (IRB) determined that this study is exempt from full IRB review given that we could only access de-identified data. The Data Access Center (DAC) at the UCLA Center for Health Policy Research was approved by the UCLA South General Institutional Review Board to conduct analyses of confidential CHIS data (UCLA IRB #G09-05-103-01).

5.4.1 Analytical Models and Procedure

Although the social and spatial analyses started with the same basic research question, the specific questions these analyses addressed became increasingly different as the analyses diverged, so that the results are not directly comparable. Given this we proposed to compare the social and spatial analyses back to a central traditional

Figure 34: Comparing Analytical Techniques for Contextual Effects Studies



multilevel model (i.e. hierarchical model) instead of directly to each other. However, we had to compare each through additional intermediary models given different final samples and analytical approaches (see Figure 34).

With the social analyses, the study samples changed when the full sample was apportioned into three subsamples (neighborhood SES quartile 2 vs. 1, 3 vs. 2, and 4 vs. 3), and during pre-processing for the PSM model when any unmatched exposed or unexposed individuals were dropped from the subsample analyses. As for the analytical approach, the social epidemiology analyses used a PSM model to match exposed and unexposed individuals with similar propensities of being exposed and then calculated the average treatment effect on the treated (ATT). These analyses attempted to mimic an experiment by using a causal counterfactual framework.

In the spatial analyses the overall sample changed very little. Examination of the study area identified two census tracts with islands – one with no individual-level observations, the other with four – which were dropped given limitations of the selected spatial analytical techniques to deal with non-contiguous areas (i.e. neighborhoods). However, in order to conduct spatial lag (and error) regression for our analyses, we aggregated adjusted individual-level predicted outcomes up to the neighborhood level. This fundamentally changed our sample to a sample of neighborhoods and also masked some variability within each neighborhood. The approximated spatial multilevel models accounted for spatial dependence using a spatially lagged dependent variable (and as comparison an analysis accounting for spatial correlation in the error term). These spatial models required specifying a spatial weights matrix *a priori* to establish spatial

relationships. We specified queen's contiguity neighbors and a row-standardized weights matrix. Following from spatial econometrics this approach framed an etiologic question with the hypothesis that the effect of neighborhood SES on the health outcomes would spillover to surrounding neighborhoods; however, effort was also spent on examining model specification and prediction to aid the development of future models.

To facilitate comparisons between the social and spatial approaches, we conducted three new intermediary analyses. First, we estimated the effect of neighborhood education and income on overweight/obesity, type 2 diabetes, and current smoking, accounting for potential individual-level confounders, using a traditional multilevel model with the full study sample (Figure 34: Model 1). More specifically, we used a generalized estimating equations (GEE) model to account for clustering within neighborhoods (i.e. tracts) and provide robust standard errors.[154] Next, we conducted separate intermediary traditional multilevel model analyses modeled as above but with the sample used in the social analyses (three matched subsamples) (Figure 34: Model 2) and the sample used in the spatial analyses (full sample minus two tracts with islands) (Figure 2: Model 4). Finally, we compared the results from these intermediary models with those from the social approach (Figure 34: Model 3) and the spatial approach (Figure 34: Model 5 [pre-spatial] and Model 6 [spatial]) from our earlier work.

We hypothesized that compared to their traditional multilevel models counterparts, the social analyses (i.e. the PSM models) would provide: 1) narrower confidence intervals (i.e. increased precision) due to the preprocessing of observations, which eliminates off-support observations and provides a subsample that requires less

interpolation, and 2) differing effect estimates given a subsample (vs. the using the entire sample). However, we note that the social analyses fail to explicitly account for clustering within neighborhood, which could falsely inflate significance. For the spatial analyses (i.e. the approximated spatial multilevel models), we hypothesized that compared to the appropriate traditional multilevel model, the estimates would be stronger as they incorporate the influence of nearby neighborhoods but still in the same direction.

5.5 Results

Tables 8A and 8B show the results from the multilevel models using the full study sample. We estimated a significant inverse effect only for neighborhood education on overweight/obesity, although even this confidence interval approached zero. The multilevel models using the pre-processed sample from the PSM models (i.e. the social analyses)estimated a significant decrease in overweight/obesity when moving from neighborhood education quartile (Q)1 to Q2 and from Q3 to Q4 and a significant decrease in smoking moving from neighborhood education Q3 to Q4 (Tables 9A and 9B). Results from the actual PSM models (from previous work) were similar. In the neighborhood education models the standard errors increased slightly while magnitude, direction, and statistical significance of estimates were very similar (Table 9A). The only exception is neighborhood education Q2 vs. Q1 in the overweight/obesity model, which lost significance in the PSM model with the wider confidence interval. The results for the neighborhood income models were also similar between the multilevel and PSM models, and although some estimates changed direction, none of the estimates were significant.

Table 8A: Multilevel Model Effect Estimates of Neighborhood Tract Education on Overweight/obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009, using Full Sample.

	N	Estimate	Robust SE	Lower 95% CI	Upper 95% CI
Overweight/Obesity					
Neighborhood Education Quartile	2515	**-0.0319**	0.0113	**-0.0541**	**-0.0098**
Type 2 Diabetes					
Neighborhood Education Quartile	2515	-0.0027	0.0055	-0.0135	0.0082
Current Smoking					
Neighborhood Education Quartile	2515	-0.0017	0.0066	-0.0147	0.0113

Models adjusted for individual-level covariates

Linear model, Gaussian distribution, exchangeable correlation structure

Table 8B: Multilevel Model Effect Estimates of Neighborhood Tract Median Household Income on Overweight/obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009, using Full Sample.

	N	Estimate	Robust SE	Lower 95% CI	Upper 95% CI
Overweight/Obesity					
Neighborhood Income Quartile	2515	-0.0132	0.0098	-0.0323	0.0060
Type 2 Diabetes					
Neighborhood Income Quartile	2515	-0.0023	0.0049	-0.0118	0.0073
Current Smoking					
Neighborhood Income Quartile	2515	-0.0096	0.0058	-0.0209	0.0018

Models adjusted for individual-level covariates

Linear model, Gaussian distribution, exchangeable correlation structure

Table 9A: Comparison of Results from Multilevel Models Using PSM Samples with PSM Models: Effect Estimates of Neighborhood Tract Education on Overweight/Obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009.

	Multilevel Models					PSM Models				
Causal contrast	**N**	**Estimate**	**Robust SE**	**Lower 95% CI**	**Upper 95% CI**	**N - matched pairs**	**ATT Difference**	**SE**	**Lower 95% CI**	**Upper 95% CI**
Overweight/Obesity										
Moderate-low vs. Low % with bachelor's degree or higher (Q2 vs. Q1)	972	**-0.09377**	0.03637	**-0.16506**	**-0.02248**	698	-0.09384	0.04992	-0.19163	0.00294
Moderate-high vs. Moderate-low % with bachelor's degree or higher (Q3 vs Q2)	1104	0.05015	0.03190	-0.01238	0.11268	741	0.04858	0.04207	-0.03343	0.13182
High vs. Moderate-high % with bachelor's degree or higher (Q4 vs Q3)	979	**-0.09250**	0.03689	**-0.16480**	**-0.02020**	624	**-0.10417**	0.04250	**-0.20218**	**-0.03142**
Type 2 Diabetes										
Moderate-low vs. Low % with bachelor's degree or higher (Q2 vs. Q1)	972	-0.02367	0.02060	-0.06404	0.01669	698	-0.01862	0.03011	-0.07525	0.03561
Moderate-high vs. Moderate-low % with bachelor's degree or higher (Q3 vs Q2)	1104	0.00699	0.01376	-0.01998	0.03397	741	0.01484	0.01848	-0.01776	0.04830
High vs. Moderate-high % with bachelor's degree or higher (Q4 vs Q3)	979	0.00368	0.01496	-0.02564	0.03299	624	0.00160	0.01751	-0.03333	0.03509
Current Smoking										
Moderate-low vs. Low % with bachelor's degree or higher (Q2 vs. Q1)	972	0.03210	0.02078	-0.00862	0.07282	698	0.04011	0.02651	-0.01158	0.08811
Moderate-high vs. Moderate-low % with bachelor's degree or higher (Q3 vs Q2)	1104	0.01652	0.01746	-0.01770	0.05075	741	0.02159	0.02642	-0.00687	0.08552
High vs. Moderate-high % with bachelor's degree or higher (Q4 vs Q3)	979	**-0.04554**	0.01981	**-0.08436**	**-0.00671**	624	**-0.05449**	0.02519	**-0.11908**	**-0.02656**

Models adjusted for individual-level covariates.

Table 9B: Comparison of Results from Multilevel Models Using PSM Samples with PSM Models: Effect Estimates of Neighborhood Tract Income on Overweight/Obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009.

	Multilevel Models					PSM Models				
Causal contrast	**N**	**Estimate**	**Robust SE**	**Lower 95% CI**	**Upper 95% CI**	**N - matched pairs**	**ATT Difference**	**SE**	**Lower 95% CI**	**Upper 95% CI**
Overweight/Obesity										
Moderate-low vs. Low neighborhood median household income (Q2 vs. Q1)	972	0.01507	0.03779	-0.05901	0.08914	592	-0.01689	0.04876	-0.12034	0.06868
Moderate-high vs. Moderate-low neighborhood median household income (Q3 vs Q2)	1104	-0.05331	0.03310	-0.11819	0.01157	729	-0.00480	0.04201	-0.06921	0.08078
High vs. Moderate-high neighborhood median household income (Q4 vs Q3)	979	-0.01481	0.03606	-0.08548	0.05585	676	0.00740	0.04115	-0.08533	0.07846
Type 2 Diabetes										
Moderate-low vs. Low neighborhood median household income (Q2 vs. Q1)	972	-0.00516	0.01719	-0.03884	0.02853	592	-0.01689	0.02230	-0.07521	0.00678
Moderate-high vs. Moderate-low neighborhood median household income (Q3 vs Q2)	1104	-0.02039	0.01385	-0.04755	0.00676	729	0.00960	0.02063	-0.02260	0.05270
High vs. Moderate-high neighborhood median household income (Q4 vs Q3)	979	-0.01389	0.01224	-0.03788	0.01010	676	-0.01183	0.01993	-0.06107	0.02171
Current Smoking										
Moderate-low vs. Low neighborhood median household income (Q2 vs. Q1)	972	-0.01352	0.02097	-0.05462	0.02757	592	0.00000	0.03193	-0.04259	0.07770
Moderate-high vs. Moderate-low neighborhood median household income (Q3 vs Q2)	1104	0.00002	0.01928	-0.03777	0.03782	729	-0.00274	0.02567	-0.05328	0.04566
High vs. Moderate-high neighborhood median household income (Q4 vs Q3)	979	0.00675	0.01771	-0.02797	0.04146	676	-0.00592	0.02320	-0.04977	0.03834

Models adjusted for individual-level covariates.

Tables 10A and 10B show results from the multilevel models using the spatial samples. These analyses dropped only four observations, and as expected the results are almost identical to those from the multilevel models with the full sample (shown in Tables 8A and 8B). Tables 11A and 11B show the results from the approximated aspatial and spatial multilevel models. Results from the approximated aspatial multilevel model suggest a small but significant effect of increasing neighborhood SES (education and income) quartile on diabetes and smoking. Compared to the approximated aspatial multilevel model, the estimates in the approximated spatial multilevel models (spatial lag and spatial error regression) are very similar in magnitude, direction, and significance. The spatial multilevel models accounting for spatial lag generally have the narrowest confidence intervals, followed by the aspatial multilevel models and then the spatial multilevel models accounting for correlation in the error terms. Although some models are significant, the estimated effects are quite small ranging from -0.0075 to -0.0160, and the confidence intervals are unrealistically narrow given the removal of individual variance. The spatial lag (and spatial error) terms were significant for all neighborhood exposure/outcome analyses, which together with previous work examining spatial autocorrelation suggested spatial dependence in the aspatial multilevel model.

Figures 35-40 provide an overview across all models for each neighborhood exposure-health outcome set. The multilevel model with full sample is in the center (demarcated with an empty square). Moving away to the left are first the multilevel models with PSM sub-samples, then PSM models (i.e. the social approach). Moving to the right are first the multilevel model with the spatial sample, next the approximated

Table 10A: Multilevel Model Effect Estimates of Neighborhood Tract Education on Overweight/Obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009, Using Spatial Sample.

	Estimate	Robust SE	95% CI Lower	95% CI Upper	p-value
Overweight or Obese:					
Neighborhood education	**-0.0319**	0.0113	**-0.0540**	**-0.0097**	0.005
QIC	591.498				
Type 2 Diabetes:					
Neighborhood education	-0.0027	0.0055	-0.0135	0.0082	0.632
QIC	181.024				
Current Smoking:					
Neighborhood education	-0.0017	0.0066	-0.0148	0.0113	0.794
QIC	269.825				
Number of individuals	2511				
Number of neighborhoods	194				

Models adjusted for individual-level covariates.

Neighborhood % with bachelor's degree or higher

Table 10B: Multilevel Model Effect Estimates of Neighborhood Tract Income on Overweight/Obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009, Using Spatial Sample.

	Estimate	Robust SE	95% CI Lower	95% CI Upper	p-value
Overweight or Obese:					
Neighborhood income	-0.0122	0.0097	-0.0313	0.0069	0.209
QIC	593.143				
Type 2 Diabetes:					
Neighborhood income	-0.0024	0.0049	-0.0120	0.0072	0.625
QIC	180.829				
Current Smoking:					
Neighborhood income	-0.0095	0.0058	-0.0210	0.0019	0.101
QIC	269.302				
Number of individuals	2511				
Number of neighborhoods	194				

Models adjusted for individual-level covariates.

Neighborhood median household income

Table 11A: Comparison of Results from Approximated Multilevel Models Using Spatial Samples with Spatial Multilevel Models: Effect Estimates of Neighborhood Education on Overweight/Obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009.

	Approx. Multilevel Model				Spatial Lag				Spatial Error			
			95% CI				95% CI				95% CI	
	Estimate	SE	Lower	Upper	Estimate	SE	Lower	Upper	Estimate	SE	Lower	Upper
Overweight or Obese:												
Neighborhood education	-0.0001	0.0044	-0.0087	0.0085	0.0004	0.0039	-0.0073	0.0081	0.0014	0.0056	-0.0097	0.0124
Spatial lag (ρ)					0.5097		<0.0000*					
Spatial error (λ)									0.5107		<0.0000*	
Type 2 Diabetes:												
Neighborhood education	**-0.0109**	0.0015	**-0.0139**	**-0.0080**	**-0.0083**	0.0016	**-0.0115**	**-0.0051**	**-0.0104**	0.0018	**-0.0140**	**-0.0068**
Spatial lag (ρ)					0.3354		0.0007*					
Spatial error (λ)									0.3408		0.0011*	
Current Smoking:												
Neighborhood education	**-0.0119**	0.0022	**-0.0162**	**-0.0076**	**-0.0075**	0.0021	**-0.0116**	**-0.0034**	**-0.0110**	0.0028	**-0.0165**	**-0.0054**
Spatial lag (ρ)					0.5346		<0.0000*					
Spatial error (λ)									0.5469		<0.0000*	
Number of individuals	2511				2511				2511			
Number of neighborhoods	194				194				194			
** p-value for spatial terms*												

Models adjusted for individual-level covariates.

Neighborhood % with bachelor's degree or higher

Table 11B: Comparison of Results from Approximated Multilevel Models Using Spatial Samples with Spatial Multilevel Models: Effect Estimates of Neighborhood Income on Overweight/Obesity, Type 2 Diabetes, and Smoking, San Francisco Adults, 2005, 2007, 2009.

	Approx. Multilevel Model				Spatial Lag				Spatial Error			
			95% CI				95% CI				95% CI	
	Estimate	SE	Lower	Upper	Estimate	SE	Lower	Upper	Estimate	SE	Lower	Upper
Overweight or Obese:												
Neighborhood income	0.0016	0.0043	-0.0068	0.0101	0.0028	0.0039	-0.0047	0.0104	0.0060	0.0049	-0.0037	0.0156
Spatial lag (ρ)					0.5130		<0.0000*					
Spatial error (λ)									0.5222		<0.0000*	
Type 2 Diabetes:												
Neighborhood income	**-0.0099**	0.0015	**-0.0129**	**-0.0070**	**-0.0073**	0.0015	**-0.0102**	**-0.0043**	**-0.0080**	0.0017	**-0.0114**	**-0.0047**
Spatial lag (ρ)					0.3669		0.0002*					
Spatial error (λ)									0.3503		0.0020*	
Current Smoking:												
Neighborhood income	**-0.0160**	0.0020	**-0.0199**	**-0.0120**	**-0.0111**	0.0020	**-0.0151**	**-0.0072**	**-0.0124**	0.0023	**-0.0170**	**-0.0079**
Spatial lag (ρ)					0.4424		<0.0000*					
Spatial error (λ)									0.4301		0.0001*	
Number of individuals	2511				2511				2511			
Number of neighborhoods	194				194				194			

** p-value for spatial error term*

Models adjusted for individual-level covariates.

Neighborhood median household income

Figure 35: Comparison of All Models for Effect of Neighborhood Education on Prevalence of Overweight/obesity in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS).

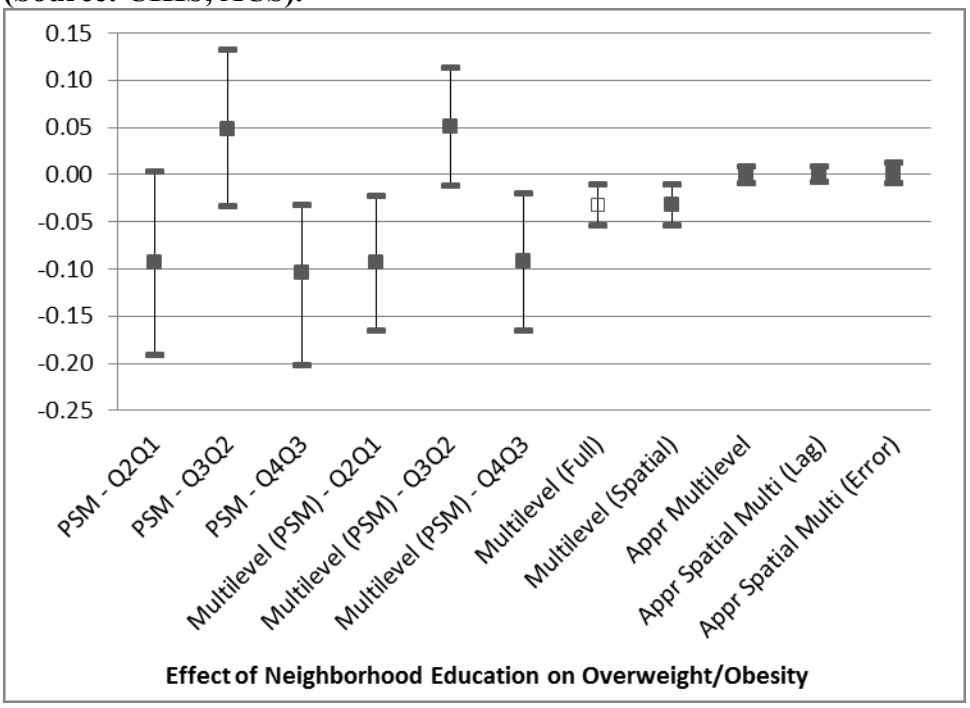


Figure 36: Comparison of All Models for Effect of Neighborhood Education on Prevalence of Type 2 Diabetes in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS).

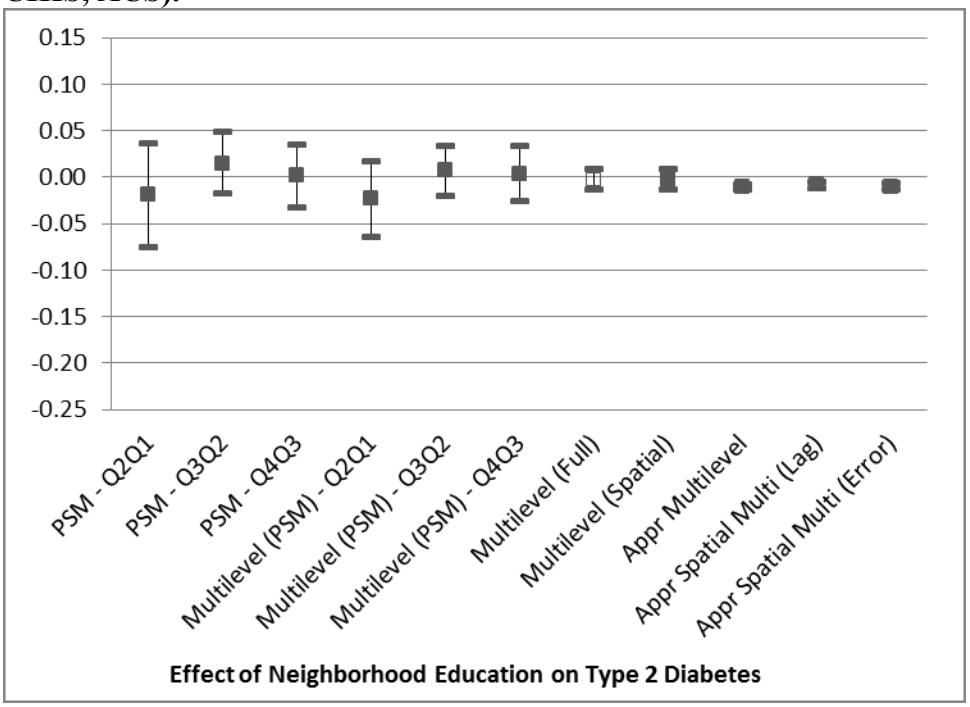


Figure 37: Comparison of All Models for Effect of Neighborhood Education on Prevalence of Current Smoking in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS).

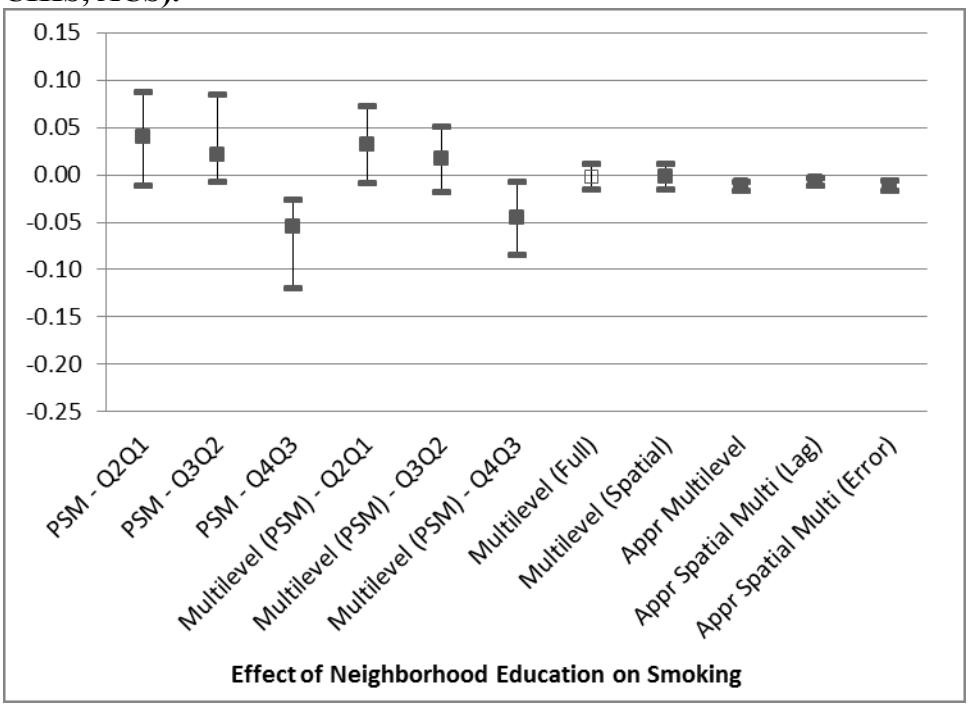


Figure 38: Comparison of All Models for Effect of Neighborhood Income on Prevalence of Overweight/obesity in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS).

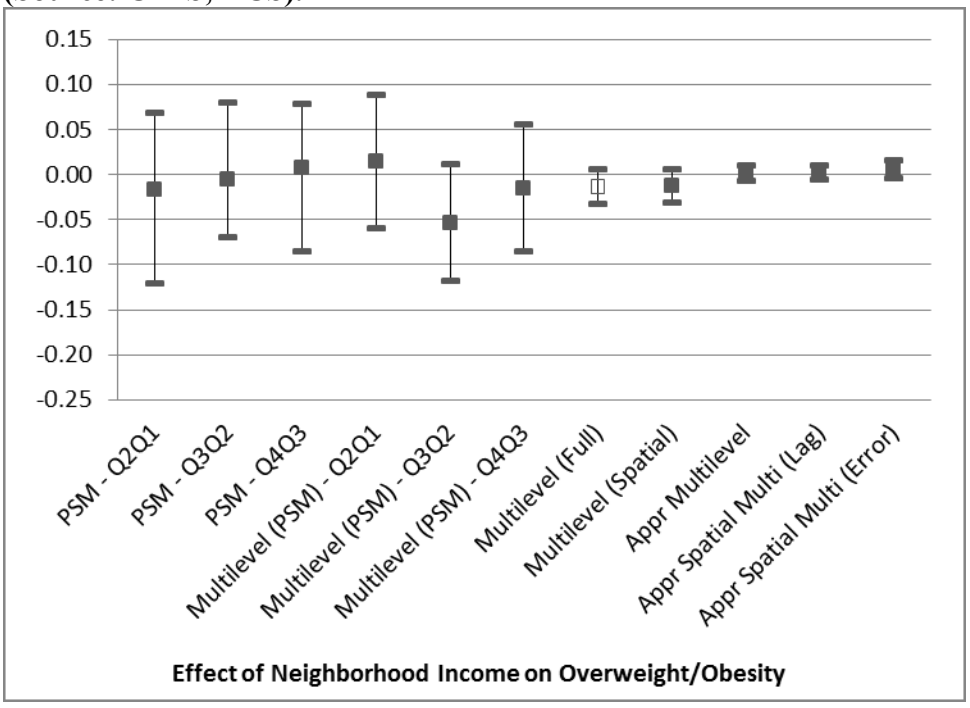


Figure 39: Comparison of All Models for Effect of Neighborhood Income on Prevalence of Type 2 Diabetes in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS).

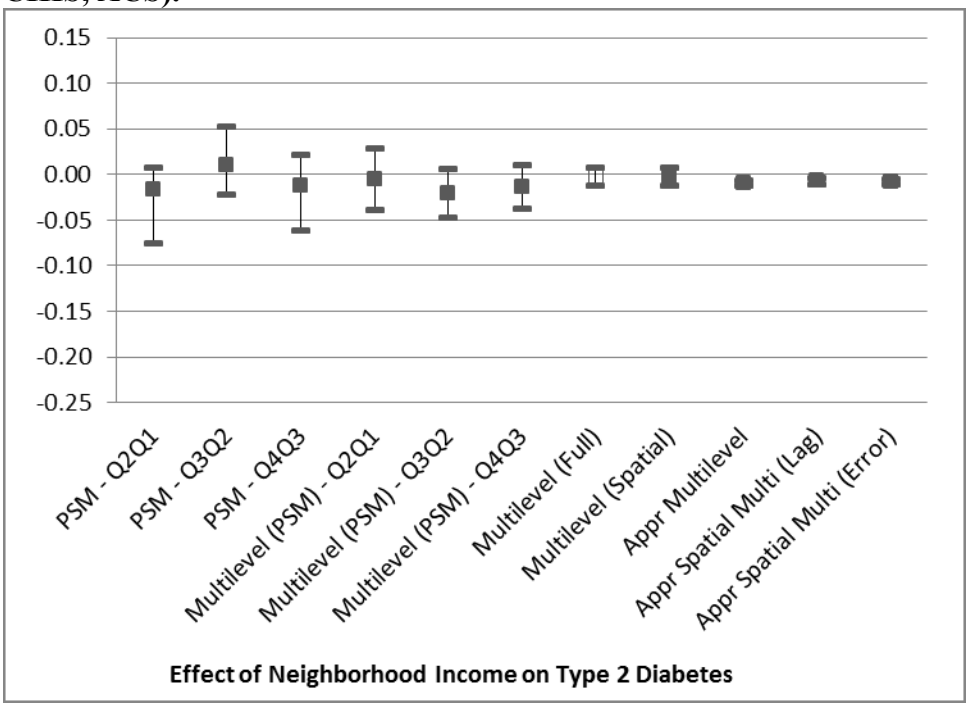
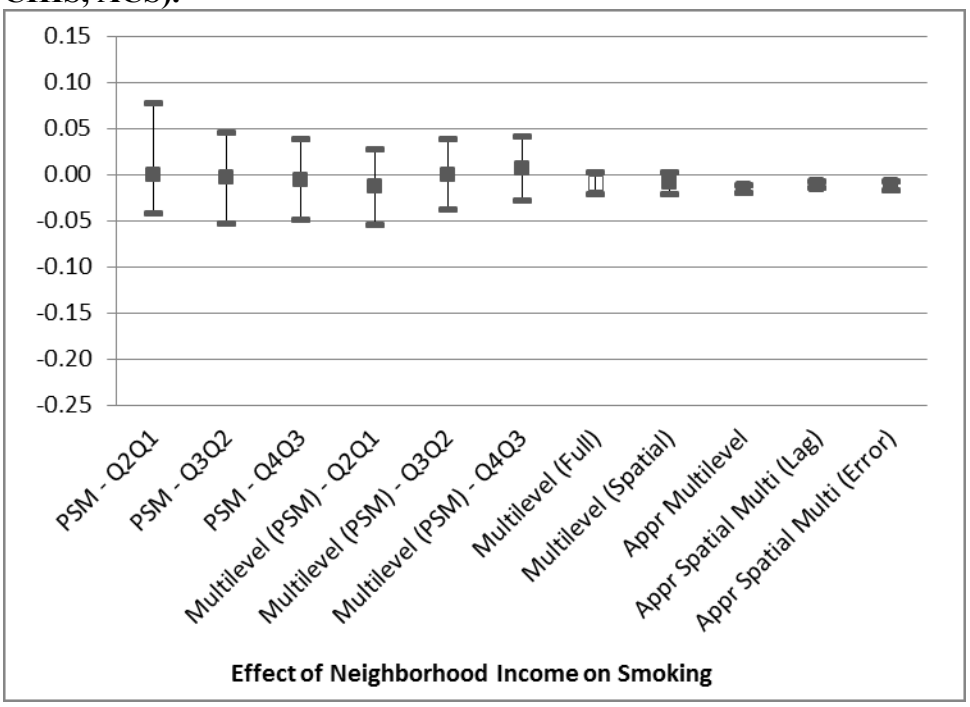


Figure 40: Comparison of All Models for Effect of Neighborhood Income on Prevalence of Current Smoking in Adults, San Francisco 2005, 2007, 2009 (Source: CHIS, ACS).



multilevel model (sample aggregated to the neighborhood level), then the spatial multilevel models accounting for spatial dependence due to spatial lag and then correlation of error terms. Several patterns are readily apparent. First, the confidence intervals appear widest with the smallest samples (i.e. subsamples for PSM) and narrowest with the samples aggregated to the neighborhood level (approximated aspatial and spatial multilevel models). Second, using a scale that captures all effect estimates with their confidence intervals, the majority of estimates fall near zero suggesting little to no effect of neighborhood education and income on the health outcomes. However, examining the move from the multilevel models with the full sample to the PSM models, we see more differentiation in the estimates by breaking the sample into sub-samples by different neighborhood exposure levels and including only matched exposed/unexposed pairs, although the confidence limits reach close to zero even when they are significant. In the transition from the multilevel models to the approximated spatial multilevel models, we see more statistically significant estimates; however, the confidence intervals overlap (or nearly so with neighborhood education and overweight/obesity). Furthermore, the significance of many if not all estimates could be due to misleadingly narrow confidence intervals resulting from aggregation to the neighborhood level. The only spatial multilevel model to lose significance compared with the multilevel models is the neighborhood education and overweight/obesity model. This is also likely due to data aggregation, which although accounting for individual-level differences within tracts averages those differences, potentially hiding extremes. These models also do not account for different sample sizes across neighborhoods. In a sensitivity analysis

comparing the approximated multilevel model with a similar model weighted by neighborhood sample sizes, the estimate for neighborhood education in the overweight/obesity model moved slightly closer to the estimate for the multilevel model with the spatial sample. Similar reasons might explain why the estimated effect of neighborhood income on overweight/obesity changes direction, although neither estimate is significant.

Broadly comparing all of the models by neighborhood exposure, we see that the neighborhood income models have more consistent estimates than neighborhood education models. Given that people are less willing to report their income (than their educational attainment) and thus income is more likely to be imputed, this may average out differences and make it harder to identify an effect.

5.6 Discussion

Our main research question was: How are the social and spatial epidemiological perspectives on contextual effects questions different, and what can we learn from comparing the two perspectives? As part of this comparison we originally wanted to directly compare results from the social and spatial analyses asking the same basic question: What is the estimated effect of increasing neighborhood education or income on overweight/obesity, type 2 diabetes, and current smoking, independent of individual-level differences? However, social and spatial epidemiologists have different perspectives on how to approach contextual effects questions, which generated different samples, models, and analytical techniques, as well as modified the basic research question into something more complex for each approach. In effect the final question we asked in the social

analyses was: What is the estimated causal effect of moving to a neighborhood with a one-quartile higher SES (education or income) on overweight/obesity, type 2 diabetes, and current smoking, accounting for individual-level confounders, matching each individual exposed to higher neighborhood SES with an individual exposed to lower neighborhood SES based on similar propensities to be exposed, but assuming no spatial dependence? In the spatial analyses the final questions we asked were in effect: What is the estimated effect of increasing neighborhood SES (education or income) quartile on overweight/obesity, type 2 diabetes, and current smoking, accounting for individual-level confounders, assuming no structural confounding, and incorporating spatial dependence where neighbors are those who touch any boundary of a focal neighborhood and have the same weight as other neighbors? And, are neighborhoods influenced by surrounding neighborhoods? Clearly, the questions that we answered in practice for each approach were quite different than our original research question and thus the results are not directly comparable across social and spatial approaches. Nevertheless, examining the results of the social and spatial approaches through the intermediary models provided valuable insights.

Results from the social epidemiology approach, which explicitly addresses structural confounding, suggest some significant inverse effects of the next higher neighborhood education quartile on overweight/obesity and smoking depending upon the causal contrast (i.e. the specific neighborhood quartiles compared). Results from the spatial approach, which incorporates significant spatial dependence, suggest a significant inverse effect of increasing neighborhood education and income quartiles on type 2

diabetes and smoking; however, all estimates are relatively close to zero and may be significant due to falsely narrow confidence intervals.

In agreement with one hypothesis relating to the social epidemiology approach, we found that effect estimates did vary with the use of subsamples compared with the full sample, with subsamples providing more variability in the estimates, but estimates for both samples were often close to zero. Breaking the full sample into the PSM samples fundamentally changed the comparisons and highlights the potential for different relationships between different neighborhood SES levels. We hypothesized that compared to their traditional multilevel models counterparts the social approach (i.e. PSM models) would provide narrower confidence intervals due to the preprocessing of observations, which eliminates off-support observations and provides a sub-sample that requires less interpolation. However, we found wider confidence intervals in the multilevel models using the PSM samples. The wider confidence intervals are mostly due to the smaller sample sizes as there was substantial overlap between the exposed and unexposed observations, so that very few observations were dropped. Furthermore, after taking into account these changes due to splitting the samples into the PSM subsamples, we found somewhat larger confidence intervals in the PSM models compared with the multilevel models with the PSM samples, suggesting that moving closer to an experimental model accounted for additional uncertainty. However, the PSM models fail to explicitly account for clustering within neighborhood which could also falsely inflate the significance and hide greater standard errors.

Previous work in social epidemiology using neighborhood SES exposures which compared results from multilevel regression (or marginal maximal likelihood estimation) with those from propensity score matching analyses or regression analyses adjusting for propensity scores found results similar to ours. When using propensity scores and propensity score matching, they found wider confidence intervals due to the smaller sample sizes, and also considerably less overlap between comparison groups as the compared quartiles or tertiles grew further apart, and generally decreased estimated effects.[55, 85, 86] (In this study by comparing only neighboring quartiles we not only maximized overlap of individuals between comparison groups but also better simulated a real-world policy change.) These studies also found little substantive differences in model results, suggesting that failure to address structural confounding in traditional multilevel regression may not necessarily produce unsupported model extrapolations.[55, 85, 86]

For the spatial approach we expected similar results from the multilevel model with the spatial sample and the approximated multilevel model, given both attempt to model the same multilevel relationship. However, when the structure of the sample changed from individuals within neighborhoods in the multilevel model to individuals aggregated into neighborhoods in the approximated multilevel model, the variance decreased suggesting overly precise standard errors in the approximated multilevel and spatial multilevel models. This likely hampers our ability to make meaningful inferences based on the statistical significance of the approximated multilevel models. For the spatial multilevel models, we hypothesized that compared to the appropriate traditional multilevel model, the effect estimates would be stronger – as they incorporate the

influence of nearby neighborhoods – but still in the same direction. Instead we found somewhat weaker statistically significant effect estimates in the spatial models accounting for spatial dependence, as the effect was redistributed across neighborhoods. Thus, changing the statistical technique from the approximated multilevel model to spatial multilevel model likely decreased bias due to spatial dependence, although this change appears modest.

Previous studies comparing spatial regression analyses with multilevel regression analyses suggest that accounting for spatial dependence between neighborhoods aids model inference by providing more accurate estimates and standard errors.[39, 82] We also found more precise standard errors in the spatial lag models; however, whether the accuracy of the estimates and standard errors improved in our models is difficult to ascertain given the aggregation problem discussed above.

Overall, despite all of the differences between the social and spatial analytical approaches – including different samples, analytical techniques, challenges addressed – most of the effect estimates are either non-significant or have confidence intervals reaching close to zero. Taken together this study fails to find broad evidence of an effect of neighborhood education or income on overweight/obesity, type 2 diabetes, or current smoking, accounting for individual-level covariates.

Regardless, these analyses using social and spatial approaches did provide valuable information. Results from the social approach suggest two key benefits to dividing the sample into quartiles and comparing only the adjacent quartiles with each other. First, different effects may occur between different neighborhood SES levels.

Second, this approach may limit structural confounding given the low numbers that were dropped in pre-processing for PSM (see previous work). Both suggest this as a more advanced and policy-relevant approach to future contextual effects work using observational data. Results from the spatial approach suggest that effects spill across neighborhoods boundaries, although the source of this spillover is a tangle of the neighborhood exposure, unmeasured variables, and the outcome of surrounding neighborhoods. This suggests that interventions should take into account what is happening in nearby neighborhoods instead of just the focal neighborhood.

The second part of answering our main research question – How are the social and spatial epidemiological perspectives on contextual effects questions different? – was to compare the broader perspectives (as opposed to the analytical approaches and statistical results). Kuhn's theory of incommensurability suggests that comparisons across disciplines are challenging and results not directly translatable,[80, 81] so drawing any firm conclusions by directly comparing statistical values or ranking one analytical approach over the other may be a fool's errand. Therefore, we took a step back to examine the broader perspectives that social and spatial epidemiologists bring to a contextual effects question and to put the analytical approaches, statistical results, and conclusions in context.

Although both social and spatial epidemiology share roots in epidemiology, the distinction between the two perspectives is perhaps best defined by the strong connection of spatial epidemiology to medical geography. Above all epidemiology seeks to improve

public health, while medical geography seeks to describe and predict spatial patterns of health phenomena.[37, 153]

In social epidemiology the goal to improve the health of the population drives researchers to attempt to identify clear, modifiable causal factors on which they can intervene to improve health. This drives relatively straightforward questions of does X cause Y, which may be estimated using predictive regression techniques but are placed within a causal framework. Many social epidemiologists enhance causal inference by establishing their work within a counterfactual causal framework, which seeks to approach the ideal experiment as closely as possible.

In contrast, spatial epidemiologists may attempt to answer etiologic (i.e. causal) questions, but their focus remains on developing models which strongly predict spatial health patterns. The standard first step in a spatial study is to conduct exploratory spatial data analysis, which examines actual (as opposed to hypothesized) spatial patterns and can lead to modifications in the model.[45] As analysis proceeds spatial researchers continue to attempt to improve their predictive models by examining spatial patterns of model residuals, often using local knowledge to identify additional factors which could explain the pattern, and possibly adding variables or modifying spatial relationships. This results in increasingly complex models which may diverge from the original hypothesis that promoted the study. This limits the ability to make causal inferences, because the ideal experiment under the causal counterfactual framework is either continually changing or too complex to model. However, knowing your study area is very important to spatial researchers, and it is common for a geographer to describe the study area in

some detail and place the results within the context of that specific place. By contrast, epidemiology studies typically fail to mention characteristics of the study area unless they are directly related to the exposure and outcome, likely because epidemiologists typically develop studies whose results can be generalizable to larger populations.

The implied public health lever also differs between the perspectives. The social epidemiology perspective attempts to identify the X that causes Y, in order to intervene on that element (X) and achieve a certain change in Y in a broad population. The spatial epidemiology perspective implies that reality is more complex and intervening on the one X might not achieve the expected result, especially if taken out of context. By developing models specific to a defined study area, the spatial perspective incorporates local context and may enhance interventions in specific locations, which a study generalizable to the larger population cannot do.

Furthermore, the specifically spatial elements of the spatial models – such as the spatial weights matrix, neighborhood definition, neighborhood scale, and geostatistical or spatial regression techniques – create a model with infinitely more knobs to turn than in a social model. Thus, spatial models are more complex and appear to require more assumptions. Spatial researchers can't possibly conduct a sensitivity analysis for every variation, and it seems to become much harder to pull a clear answer to a causal question out of this complex web as compared with analyses from the social perspective.

However, the complexity of spatial models reminds us: "All of the fruits of scientific work, in epidemiology or other disciplines, are at best only tentative formulations of a description of nature, even when the work itself is carried out without

mistakes"(p.24).[155] Some from spatial epidemiology or medical geography would argue that spatial analysis attempts to incorporate a more realistic view of the situation, which is less prone to unrealistic or meaningless conclusions. Perhaps the social epidemiology perspective creates a bigger illusion of identifying causal effects by attempting to answer a contextual effects question using just one or even a few contextual variables and ignoring the remaining real world 'context.' Perhaps the complexity of the spatial approach does a fine job highlighting the absurd challenge of attempting to truly disentangle a contextual effect from compositional effects using observational data.

This identification problem may be insurmountable using either approach, because in many cases contextual and compositional variables influence each other (i.e. endogeneity[153] or the reflection problem[87]), and with observational data we cannot say with certainty which causes which. Spatial lag regression includes an endogenous term on the right-hand side of the equation, which directly acknowledges the influence of surrounding neighborhoods on the central neighborhood that in turn influences the surrounding neighborhoods. (However, it is important to note that although statistical techniques resolve the statistical problem of endogeneity in spatial lag regression, they still do not truly allow us to identify causal effects but rather to enhance prediction.) Likewise, models that include compositional variables as confounders ignore their potential to act as mediators,[156] and thereby may 'over-control' for these variables[27] and "remove part of the contextual effect" (p.119).[24] Thus, examining a contextual effects question using the spatial approach helped us uncover even more obstacles to identifying

causal contextual effects, a problem frequently recognized by some social epidemiologists.

5.7 Conclusions

Comparing the social epidemiology and spatial epidemiology perspectives and their analytical approaches provided several valuable insights into contextual effects research using observational data. First, the potential for integration of social and spatial perspectives to generate methodological advancements in contextual effects research appears very limited. Perhaps the most notable difference between the perspectives is the focus in the social epidemiology perspective on causal inference and identifying modifiable factors on which to intervene and improve public health, while in the spatial perspective the focus is on prediction and accurately modeling the real world. Spatial analyses rarely operate within the bounds of a causal framework. Relatedly, although starting with the same basic question, each perspective generated different goals and answered a different question using a different analytical technique to address a different critical challenge to contextual effect studies. This divergence severely limits the possibilities for directly integrating elements from the social or spatial approach into the other in order to advance identification of causal contextual factors. One possibility is to use propensity scores to identify a sample that limits structural confounding and incorporate this sample into spatial analyses. Indeed some socio-spatial work in this area has already begun to use propensity scores as sensitivity analyses.[55, 86, 157] However, if the spatial analyses use area-level data then dropping individuals reduces the sample size in some neighborhoods, which could lead to a small numbers problem and thus unstable

estimates. Furthermore, if a neighborhood no longer has any observations this creates a hole in the study area which hampers the ability to use spatial lag and error models.

Second, comparing the approaches further emphasized the fundamental identification problem inherent to contextual effects research. It is unclear how meaningful any contextual effects research using observational data can be given the complexity of contextual interactions. Given this identification problem researchers clearly need to push for more experimental studies.

Third, despite limited potential for direct integration across perspectives and the identification problem, social and spatial epidemiology studies using observational data may still provide valuable information to aid in the design of contextual-level experiments (i.e. community trials). For example, social epidemiology can suggest modifiable social factors, whereas spatial epidemiology may predict how an intervention might affect a certain place. If we can recognize potential exposures and confounders before implementing a community trial, we may enhance the effectiveness of the intervention and/or limit problems, thereby saving vast amounts of resources while furthering this area of research.

Finally, perhaps the most provocative question suggested from this study is should even continue pursuing identification of contextual determinants of health, whether using observational or experimental studies. Certainly the evidence from this study plays just a small part in answering that question; however, generally we found little to no effect of neighborhood education or income on overweight/obesity, type 2 diabetes, or current smoking. These results are limited by our observational data,

relatively small sample size, focus on one geographic area, and the technicalities and assumptions of our chosen social and spatial analytical approaches, and of course the identification problem. However, this study has many strengths including the use of high quality data, multiple outcomes, and cutting-edge analytical approaches from two different epidemiological perspectives.

Even if we had found stronger evidence to suggest causal contextual effects as some studies have, other studies have noted the relatively small or even insignificant influence of contextual factors compared with those at the familial or individual levels. Studies that partition total variance in individual-level outcomes to examine the total neighborhood variance generally find a very low percent of total variance due to the neighborhood-level exposure (typically less than 10% but as low as 0.2%).[1, 125, 158-160] Debate exists over whether or not the small percent of variance attributable to context indicates that contextual effects are meaningful even when statistically significant.[1, 125, 160]

On top of the potentially non-significant and/or meaningless effects, we need to identify modifiable contextual factors that when intervened on produce the desired effects. Contextual effects studies assume a stable unit treatment value (SUTVA); however, with dynamic feedback loops this is likely unrealistic. Thus, an intervention to move individuals to neighborhoods with a higher income or a higher percent of college graduates – as an intervention based on this study would suggest – may not be effective. In the Moving to Opportunity (MTO) trial only some participants complied with their assigned treatment to move to a higher-SES neighborhood and others later moved backed to their original neighborhood.[88] Specific to this study, the question is also can we expect

to intervene on neighborhood SES on a broad scale, and given the great expense of MTO the answer would seem to be no. However, given our hypothesis that neighborhood SES affects access to salutogenic resources, identifying an effect of neighborhood SES could support community trials that increase neighborhood resources as a treatment.

As suggested by several of the insights gained from this study, the overwhelming majority of contextual effects studies to date suffer from an identification problem in large part due to the use of observational data, so we do not have enough information yet to determine whether pursuing identification of contextual effects is worthwhile. The next steps seem clear: use data from the most advanced social and spatial epidemiology studies to design and implement community trials. Data from these trials will meaningfully inform future public policy interventions for public health problems.

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