Violent Crime Exposure and Pediatric Cardiovascular Health: A Spatial and Hierarchical Analysis for Low Income Children Living in Boston.

Abstract

Exposure to violence in childhood is associated with negative health outcomes. We analyze the relationship between census tract level violent crime rate and rates of two cardiovascular outcomes, hypertension and obesity, among Boston children enrolled in subsidized insurance. We hypothesize that these relationships between violent crime and cardiovascular outcomes are structured by spatial and/or neighborhood level determinants (educational, social and economic, and health opportunity environment). Local Moran's I measurements show significant spatial autocorrelation in the exposure and the outcomes of interest. Ecological (census tract-level) as well as nested (individuals within census tracts) regression models were used for each outcome. Given the autocorrelation in the variables, spatial lag models were run. We also used neighborhood fixed effects models to assess whether the autocorrelation is induced by administrative district. This study underlines the health implications of violent crime exposure and the importance of considering spatial and neighborhood patterning in assessing its impact.

I. Introduction

Although an estimated 98% of Boston's children are covered by health insurance, rates of obesity are higher in Boston than those in the state of Massachusetts and in the nation. These rates are notably higher among children of low income households (1). Over 25% of Boston families with children live below the poverty line, and there are wide disparities in these percentages by neighborhood. Children of white race/ethnicity and those living in higher income families are more likely to have positive health outcomes and be insured through providers other than HealthNet/Medicaid (2). Children living in low income families in Boston are more likely to live in areas with higher incidence of violent crime (3).

The city of Boston has an established neighborhood structure, formed by the amalgamation of groups of census tracts and defined by administrative and community boundaries. There are 153 census tracts and 33 neighborhoods in Boston. The sense of community neighborhood membership is strong among citizens and is reflected in health and housing policy, which provides reason to believe that administrative neighborhood level characteristics could explain clustering (4). However, spatial dependence is also likely to initiate clustering in the exposure and outcome variables as health behaviors are known to vary across space, and violence has been shown to exhibit "infectious" diffusion qualities (5; 6). Further we recognize that neighborhood level measurements can introduce bias, as demonstrated by previous work on context and pediatric health in Boston (7). In order to better understand the ways in which violence and cardiovascular outcomes among disadvantaged youth in Boston relate and vary across space and community boundaries, this analysis employs administrative neighborhood fixed effects and spatial autoregressive Poisson models.

Previous work regarding urban stressors has identified neighborhood violence as an influential factor in cardiovascular health (8; 9; 10; 11). However, the relationship has not yet been explored spatially among low income youth in Boston. This paper examines the relationship between crime rate and distributions of both obesity and hypertension rates. This exploration is motivated and informed by the theoretical framework supporting *The Basic Science of Pediatrics*, which highlights the dynamic interactions between environmental, biological, and developmental aspects of child health (12) (Fig 1). We aim to better explain the structure of the spatial relationship between violence exposure and cardiovascular outcomes, understanding that all components of this framework vary by context. This analysis is conducted on measures by census tracts in Boston, examining outcomes among all children in the city receiving clinical care coverage through subsidized insurance (13).

II. Methods

Data Sources and Measurement

The outcomes of interest are BMI and blood pressure. De-identified, patient level data comes from the Massachusetts Health Disparities Repository (MHDR), which contains all clinical encounters and observations (recorded in the EHR) for all individuals receiving care coverage through the HealthNet insurance program (14; 13). Patients were considered eligible for inclusion in the study if they were aged 3 to 18 in 2010 and had at least one clinical encounter or observation from January 1, 2010-December 31, 2010 (N=10,873). Eligible patients in this system reside in 106 of Boston's 153 census tracts and all of the 33 neighborhoods (15; 16).

Every recorded measurement of height, weight, and blood pressure (diastolic and systolic) taken in 2010 was collected for eligible patients in the MHDR. Gender, race/ethnicity, and census tract was also collected for each patient. To avoid use of inaccurate and unrepresentative measures, blood pressures were only included if they were recorded during a "well patient" visit. Implausible

values were determined to be >15% higher than the 95th percentile for each growth and blood pressure measure. Measurements exceeding this limit were excluded as coding errors in the EHR. BMI was calculated from the growth measures using the formula: $BMI = \frac{weight(kg)}{meters^2}$.

Standardized z-scores were calculated for each observation of BMI and blood pressure based on the BMI and blood pressure distributions of the pediatric US population. BMI distributions are known to vary by age and gender, so BMI z-scores standardize based on these factors. Height also skews the distribution of blood pressure measurements, so diastolic and systolic blood pressure z-scores account for age, gender, and height (17; 18). Associated percentile values were calculated for the z-scores. Blood pressure measurements at or above the 95th percentile were considered to be cases of hypertension, and BMI measurements at or above this cutoff were considered to be cases of obesity.

The exposure of interest is violent crime rate. A list of violent incidents that occurred in Boston between January 1 and December 31, 2009 (including robbery, assault, and murder) was compiled from data provided by the Boston Police Department. The address for each incident was geocoded to latitude/longitude coordinates using ArcGIS 10.2 (19) and then aggregated to census tract-level rates as incidents per square mile (15). There is assumed to be some level of random error in the addresses assigned to each incident, but based on the distributions of even and odd address numbers, it is unlikely that this error will vary across any variables of interest for this study. Outcome variable data was collected from 2010 to ensure that obesity and hypertension cases occurred temporally subsequent to exposure to violence.

Other covariates included to address potentially influential contextual and compositional structure are population size and median household income by census tracts, as reported by the US Census Bureau (15), and the Child Opportunity Index, which includes indicators related to education, social and economic factors, and health environment, as reported by Diversity Data Kids (20). Concentrations of poverty and low child opportunity have been shown to be associated with concentrations of poor cardiovascular outcomes as well as violent crime (21; 20). Median household income is modeled continuously and centered on its population mean.

Spatial Analysis Methods

Tests for spatial autocorrelation in the variables were conducted, informed by Tobler's first law of geography (22). Global and Local Moran's I were estimated for the outcome and exposure variables using ArcMap 10.2 to measure any existing positive or negative spatial autocorrelation in rates of obesity, hypertension, and violent crime between census tracts (23). Univariate Local Moran's I's were estimated for each variable. A bivariate Local Moran's I was estimated to understand the ways in which rates of hypertension and obesity are clustered together spatially, as these cardiovascular outcomes are clinically related and likely to be correlated (24). Bivariate Local Moran's I's were also estimated for violent crime rates and each outcome variable.

A statistically significant Global Moran's I indicates clustering of the variable of interest in space. Local Moran's I identifies the locations of this clustering and the direction of the relationship (high values clustered with neighbors with high values, etc). Sensitivity to different weighting structures in clustering results was tested using inverse distance and contiguity (both queen's and rook's) weighting structures. In final tests, a first-order queen's contiguity method was used to define neighbors, i.e. census tracts sharing borders were considered neighboring (25). The False Discovery Rate (FDR) method was used to adjust for multiple testing in the local tests (26).

Using the queen's contiguity matrix that weights the influence of proximal census tracts more heavily than distant tracts, a spatially lagged Poisson regression model was used for each outcome against the covariates. The spatial lag model assumes that the clustering of each variable is

a direct result of the influence on that variable in one place on that variable in another place (27). All variables in this model are aggregated at the census tract level. The form of the spatial lag model is:

$$O \sim Poisson\ (\theta, \Sigma)$$
$$\log(\theta) = \alpha + \rho W y + X\beta + \epsilon$$

where θ is a vector of counts of cases of obesity or hypertension; *Wy* is the spatially lagged dependent variable by the queen's contiguity weights matrix *W*; *X* is a matrix of observations of the included covariates: opportunity index, violent crime rate, median household income, proportion black, proportion female, and average age; $\epsilon \sim N(0, \sigma_\epsilon^2)$ is a vector of error terms (random effects); ρ is the lag parameter; α is the fixed intercept parameter; and β is a vector of fixed effect parameters for the covariates. Incidence rate ratios (IRR) and robust standard errors were estimated for each covariate.

Administrative Neighborhood Fixed Effects Analysis Methods

Based on Boston's administrative neighborhood structure, a neighborhood fixed effects model is another reasonable approach to understanding the relationship between the outcomes and exposures as well as explaining clustering (4). A model was adopted specifying census tracts (106) nested within neighborhoods (33), which were defined a priori (16)(Fig 2). The fixed effects structure controls for all unmeasured, time invariant neighborhood characteristics, and it allows for within neighborhood estimates of the effects of interest. Variables in this model are aggregated at the census tract level. The form of the fixed effects model, *i* indexing census tracts and *j* indexing neighborhoods, is:

$$O_{ij} \sim Poisson\ (\theta_{ij}, \Sigma_{ij})$$
$$\log(\theta_{ij}) = \alpha + X\beta + \delta_j + \epsilon_{ij}$$

where θ_{ij} is a vector of counts of cases of obesity or hypertension; *X* is a matrix of observations of the included covariates: opportunity index, violent crime rate, median household income, proportion black, proportion female, and average age; δ_j represents a binary variable for each of the 33 neighborhoods; $\epsilon_{ij} \sim N(0, \sigma_\epsilon^2)$ is a vector of census tract level random effects; α is the fixed intercept parameter; and β is a vector of fixed effect parameters for the covariates. Incidence rate ratios (IRR) and robust standard errors were estimated for each covariate.

Hierarchical Analysis Methods

In order to assess the impact of crime exposure on individuals, a two-level, nested models were also used, specifying individuals nested within census tracts. The hierarchical structure allows conditionally independent estimation of individual and census tract-level effects. In the random intercepts model used, the intercept value was allowed to vary by census-level random effects. This corrects for clustering of variables within census tracts. The form of the multilevel model, *i* indexing individuals and *j* indexing census tracts, is:

$$O_{ij} \sim Binomial\ (\theta_{ij}, \pi_{ij})$$
$$\text{logit}(\pi_{ij}) = \alpha + X\beta + u_j + \epsilon_{ij}$$

where θ is a binary variable of observations of cases of obesity or hypertension, π_{ij} is a vector of individual probabilities of being a case of obesity or hypertension; *X* is a matrix of observations of

the included covariates at the census tract level: opportunity index, violent crime rate, median household income and individual level: sex, race, and age; $u_j \sim N(0, \sigma_u^2)$ is a vector of census tract level random effects; $\epsilon_{ij} \sim N(0, \sigma_\epsilon^2)$ is a vector of individual level random effects; α is the fixed intercept parameter; and β is a vector of fixed effect parameters for the covariates. Spatial and neighborhood fixed effects specifications were also applied to these hierarchical models. Odds ratios and robust standard errors were estimated for each covariate in each model.

Model Comparisons

Descriptive statistics were measured for each variable of interest. For exploratory purposes, crude distributions of the outcome variables were mapped. Spatial and non-spatial bivariate analyses were conducted between each covariate and both outcomes of interest. Additionally, two non-spatial, Poisson regression models were run—one for each outcome—against all of the covariates. Spatial autocorrelation in the residuals was assessed using the Global Moran's I test. A spatial dependence model was used to account for spatial autocorrelation in the variables (28). The residuals from the spatial lag and fixed effects models were assessed for autocorrelation using Global Moran's I in order to test for outstanding spatial clustering. For all the three models, the significance of the tests of autocorrelation in the residuals were compared to assess their relative adequacy in accounting for clustering in the variables. Additionally, model fit was compared across the three types for each outcome using the Akaike Information Criteria (AIC) method (29). All multivariate and non-spatial bivariate analyses were completed using Stata 13 (30).

III. Results

In 2010, there were 5,001 cases of obesity and 485 cases of hypertension among the 10,871 children, ages 3-18, who were part of the HealthNet system. The average rate of obesity was 47.7 cases/100 person-years, and the average untransformed rate of hypertension was 5.2 cases/100 person-years. Summary statistics for each variable and descriptive statistics are listed in Table 1. Crude rates of obesity and hypertension cases were mapped by census tract and displayed in Figure 3. Obesity and hypertension cases are standardized to sex and age, hypertension counts are also standardized by height.

In the display of crude case rates, there appears to be patterning in increased rates of both obesity and hypertension in South, central Boston. Local Moran's I test results are presented (after correction for multiple testing) for both outcomes of interest (Fig 4 and Fig 5) and violent crime rate (Fig 6). Clustering tests were not found to be sensitive to changes in weighting structure. The patterning of local clustering, based on univariate Local Moran's I estimates, appears to be distributed in a spatially similar way for obesity, hypertension, and violent crime rates. Complementing these exploratory assessments, the bivariate tests of local clustering between hypertension and obesity rates (Fig 7), obesity and violent crime rates (Fig 8), and hypertension and violent crime rates (Fig 9) show significant autocorrelation after adjusting for multiple testing. The corrected Local Moran's I estimate for violent crime rate identifies high-high clustering of crime rates in an area of South central Boston (Dorchester, Roxbury, and Mattapan) that appears to overlap the high-high clustered areas identified in the univariate Local Moran's I tests for both obesity and hypertension cases. It also identifies low-low clustered areas in West Roxbury and Brighton. Additionally, there were small pockets of significant local clustering in some of the covariates, but the significant areas do not overlap those in the exposure or outcomes of interest. The spatial bivariate results show high-high clustering in south central Boston between obesity rates and hypertension rates as well as each of the outcome rates and violent crime rates. Low-low clusters

were also found in each of these bivariate assessments in West Roxbury, Brighton, Allston, and East Boston.

Non spatial, bivariate tests of the covariates against each showed violent crime rate, opportunity index, proportion black, hypertension rate, and median household income to be significantly related with obesity rate. The same covariates had a significant relationship with hypertension rates. Incidence rate ratio estimates and associated standard errors for the multivariate analyses (standard Poisson, spatial, and fixed effects aggregate models) are presented in Table 2. It is notable that violence exposure is significantly, positively correlated with cases of obesity in all three models. However, the magnitude of association is very small, and the association does not persist in the models for hypertension. Median household income has a significant negative relationship with both outcomes in all three models for each. The proportion of black patients also has a significant positive relationship of great magnitude with both outcomes in all three models. Opportunity index has a significant negative association with obesity in all three models but not hypertension. Average age and proportion female are not significantly associated with either outcome in any model. This is to be expected, as the z-scores upon which cases are based are age and sex standardized.

Highly significant spatial autocorrelation was detected in the residuals of the standard Poisson models. Both Lagrange multiplier statistics were significant for both the obesity and hypertension models. In the obesity model, only the robust Lagrange multiplier for the lag model was significant. Both robust tests were significant for the hypertension model, but that for the lag model was more highly significant. Therefore, the lag model was chosen as the spatial regression technique for both outcomes. The lag parameters in both models was significant, and the Breusch-Pagan tests for heteroskedasticity were insignificant. Additionally, both spatial models resulted in improved AIC scores and elimination of significant autocorrelation in the residuals, based on Global Moran's I. The fixed effects models indicated significant neighborhood-level variance. These models showed improved further improved AIC scores and accounted for spatial autocorrelation (Table 2).

In the hierarchical models, the effects attenuate for most covariates. Being of black race remains a significant predictor of obesity and hypertension. Additionally, individuals with obesity are more likely to have hypertension. Consistent with the aggregated models, spatial autocorrelation was detected in the residuals of the standard logistic model. The spatial lagged and neighborhood fixed effects logistic models correct for this autocorrelation. The distribution of the variance in each of the nested models suggests that a large proportion of the variance lies at the individual level, which may explain the null findings.

I. Discussion

Significant global and local spatial clustering of cases of obesity and hypertension were detected among children enrolled in HealthNet insurance plans in the city of Boston in 2010. Distribution of violent crime in Boston in 2009 was also significantly clustered. In all bivariate and multivariable analyses aggregated at the census tract level, a significant relationship was detected between violent crime exposure and obesity. The spatial lag models accounted for detected autocorrelation in models including all covariates. The significance of the lag parameters in both models suggests that a substantial amount of the variance in the relationships described is spatial. However, the AIC scores indicate that the neighborhood fixed effects models are better fit for the data. The neighborhood-level variance parameter suggests that a significant amount of the distribution of the relationships modeled is explained by neighborhood level effects. While still insignificant, the p-values associated with the tests for spatial autocorrelation in the residuals are slightly lower than those evaluating the residuals of the spatial models. The covariates at the census tract level did not remain significant in hierarchical models predicting individual level obesity and hypertension odds. The high level of

individual level variation relative to that of the census tract level detected in these models suggests that the data was inadequate to detect exposure to violent crime, the variable of interest. Neither crime nor patient point location data was available, and both variables were aggregated at the census tract level. Point locations for both measures would enable more precise measurement of individual exposure through distance to violent crime episodes.

Policy Implications

These analyses show that disparities in cardiovascular outcomes among Boston's low income youth as well as distribution of violent crime rates throughout the city are spatially patterned. They also indicate that the relationship between exposure to violent crime and risk of poor cardiovascular outcomes varies across space and administrative district (neighborhood). Boston legislative officials should be aware of the spatial patterning of cardiovascular disease risk and violent crime and develop targeted policy to maximize impact. The significant spatial and neighborhood effects indicate that aiming efforts to diminish violent crime in clustered areas and/or neighborhoods with high incidence would be likely to effectively address the related, disparate increases in risk of negative cardiovascular outcomes. Addressing economic disadvantages in these areas is also likely to be impactful. The neighborhoods that appear to require immediate attention are Roxbury and Dorchester.

Limitations

While this study introduces an innovative methodological and theoretical approach to the life course framework of pediatric science, there were some limitations. This analysis was primarily limited by the aggregation of key covariates at the census tract level. Census tract borders are administrative districts which do not reflect population distributions well (7). More granular measurement of exposure to violent crime, for example point location data, would enable much more robust estimation of its impact on individual child health.

Additional limitations include data quality and generalizability. Because it is not collected for research purposes, data from the EHR and police department are likely subject to misclassification and measurement error (i.e. diagnoses and types of crime are often modified to fit available codes). Methods of accounting for measurement errors probably did not eliminate all bias introduced. The population of interest is relatively narrow, including only children in Boston enrolled in HealthNet, who make up about 30% of the Boston youth population. These results are certainly not generalizable to the entire pediatric population of Boston, but this probably led to more conservative estimates of variation than would have resulted had the entire population been studied (as substantiated evidence suggests children in higher income families in Boston have better health outcomes (2)). These results are also likely not generalizable to low income pediatric populations in other locations outside Boston. Despite these limitations, these analyses provide actionable evidence of spatial and hierarchical patterning of factors of health risk for low income children in Boston.

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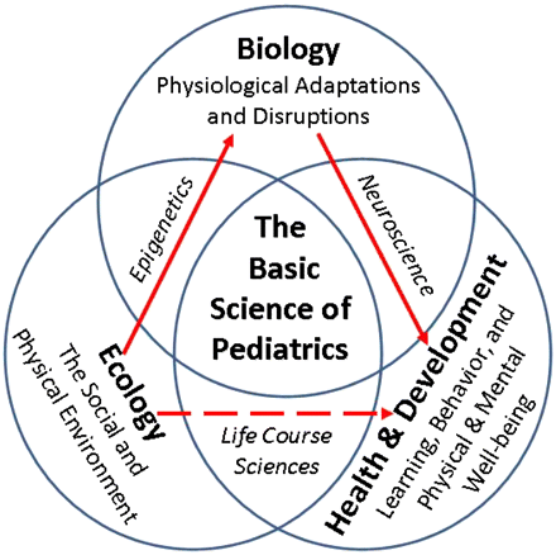

Fig 1. Shonkoff & Garner's proposed theoretical framework behind contemporary basic science of pediatrics (12)

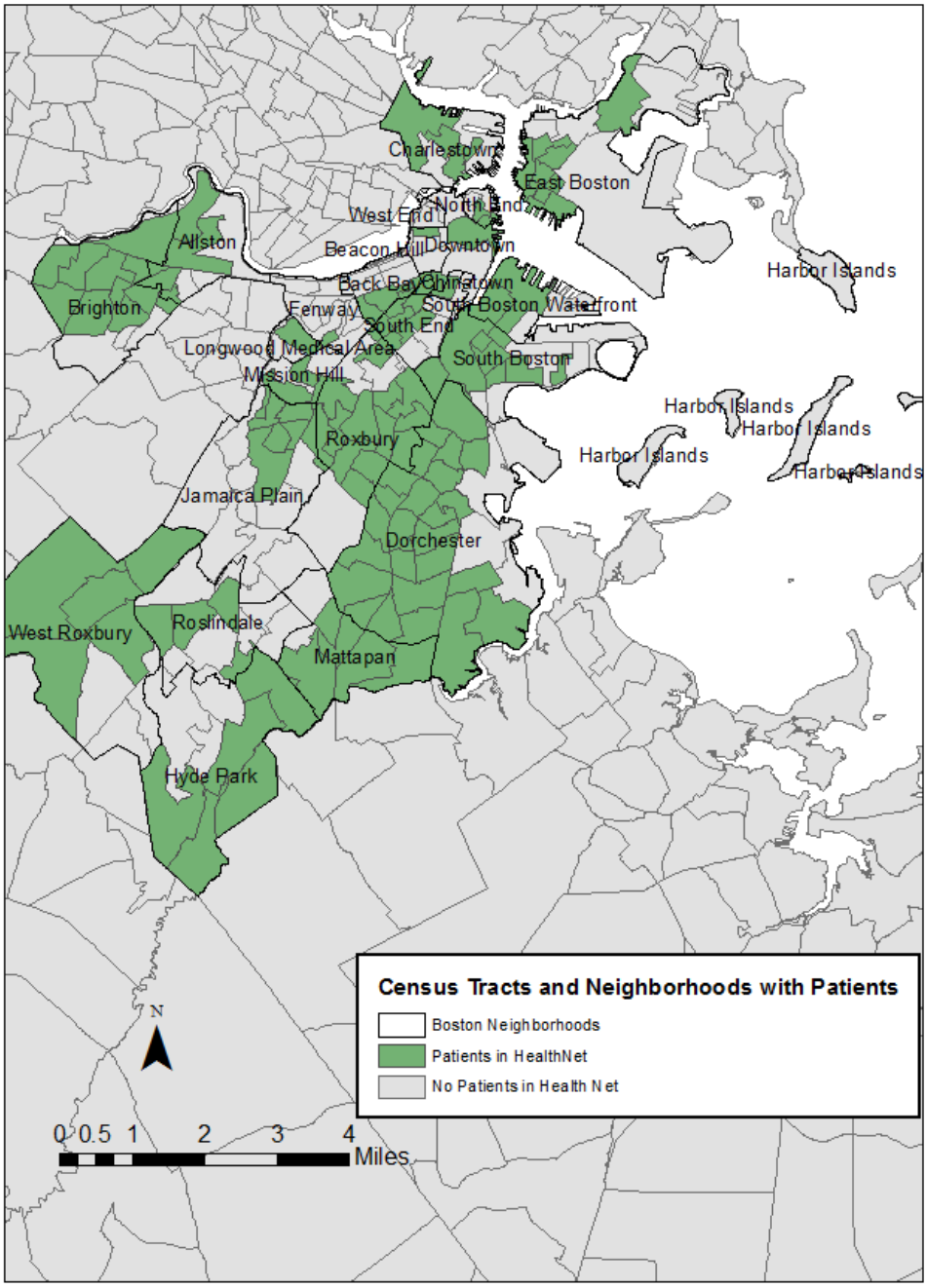

Fig 2. Administrative levels in Boston.

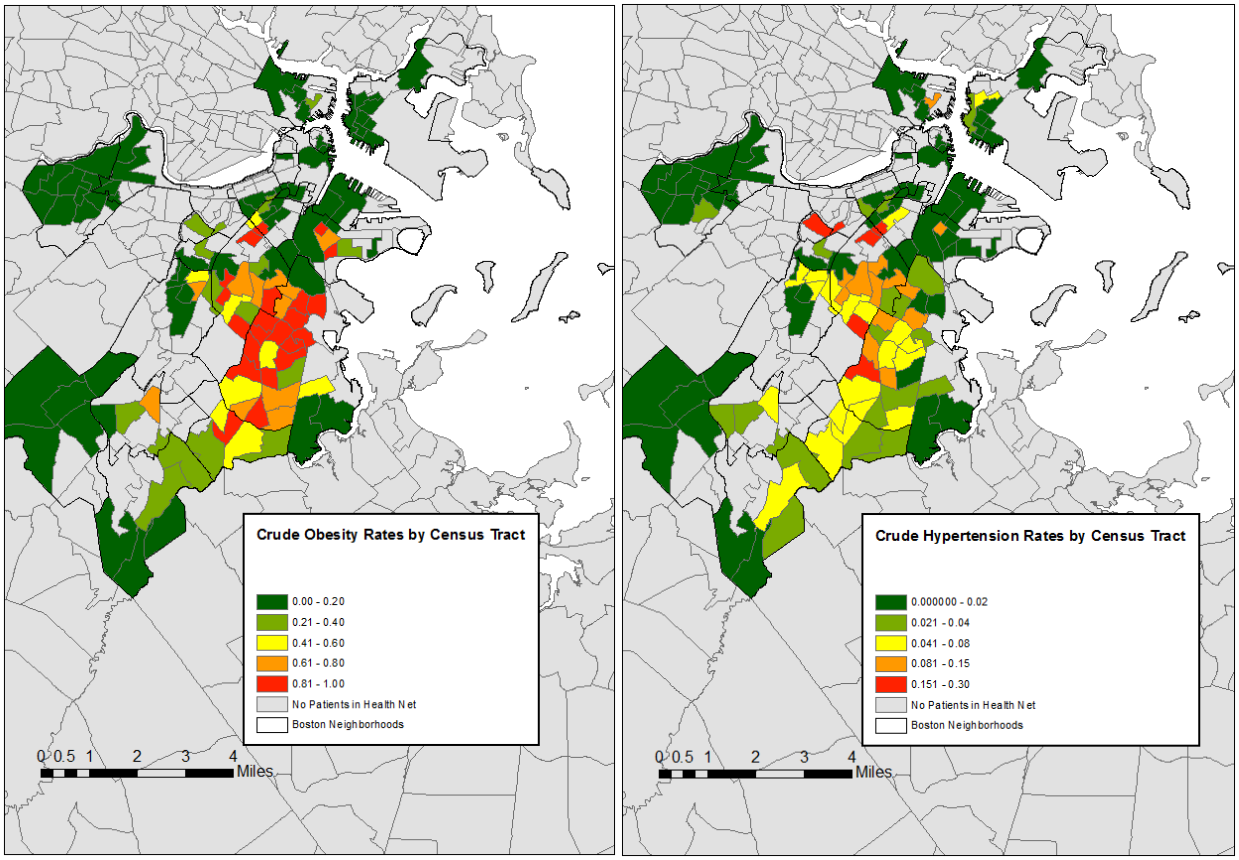

Fig 3. Categories of crude rates of obesity (left) and hypertension (right) by census tract.

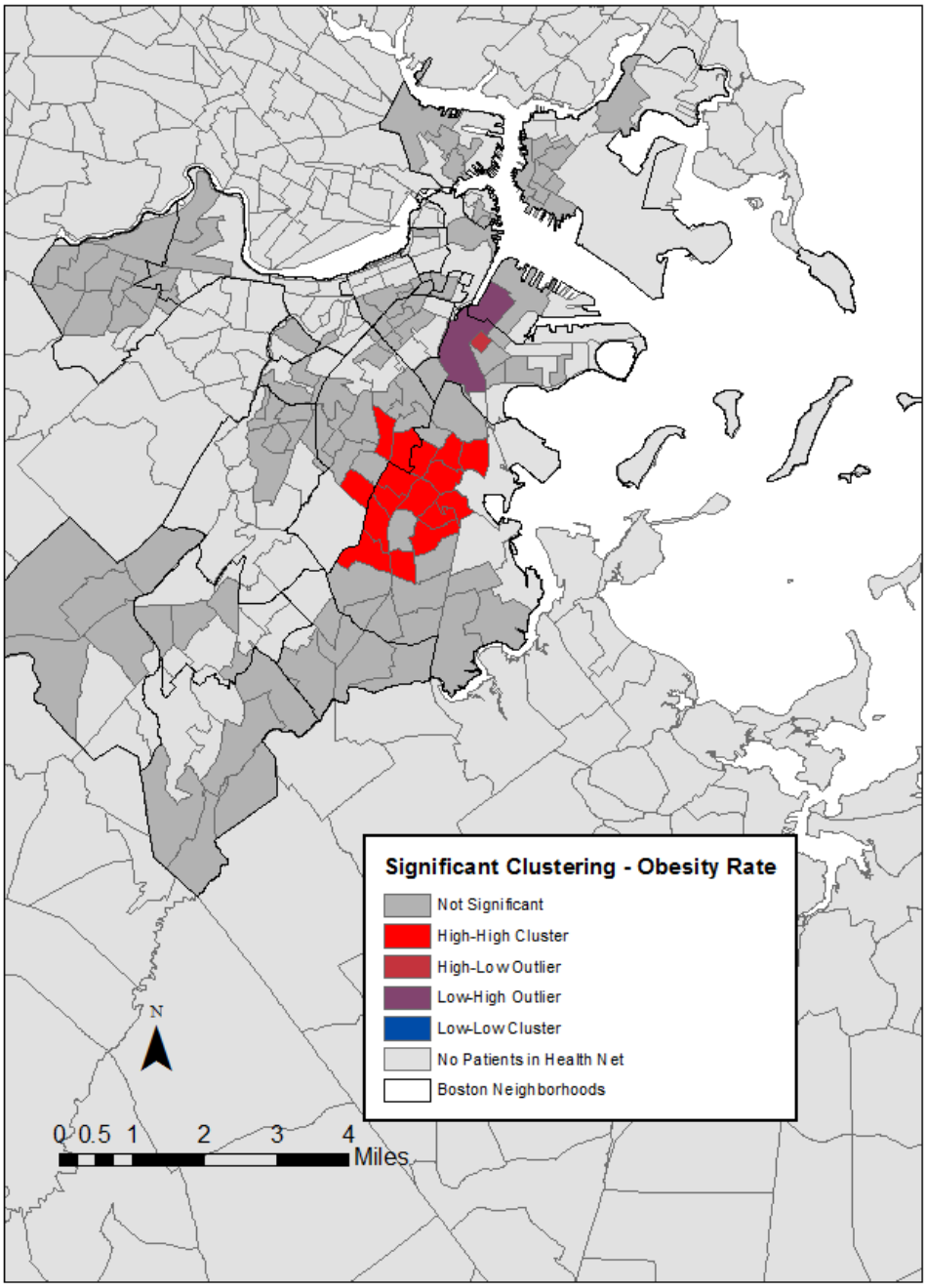

Fig 4. Significant Local Moran's I results for obesity rate, at the census tract level, after correcting using False Discovery Rate method.

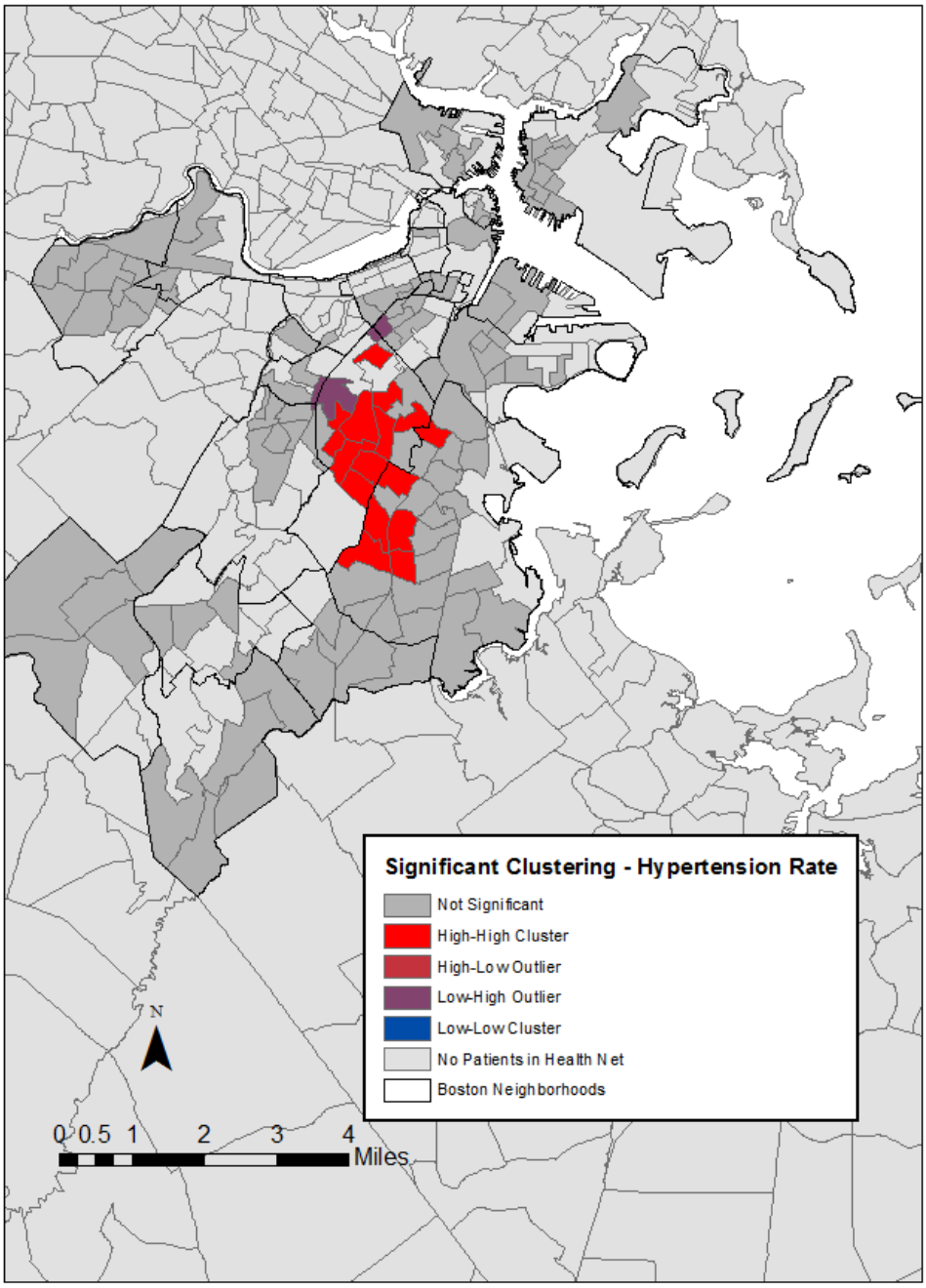

Fig 5. Significant Local Moran's I results for hypertension rate, at the census tract level, after correcting using False Discovery Rate method.

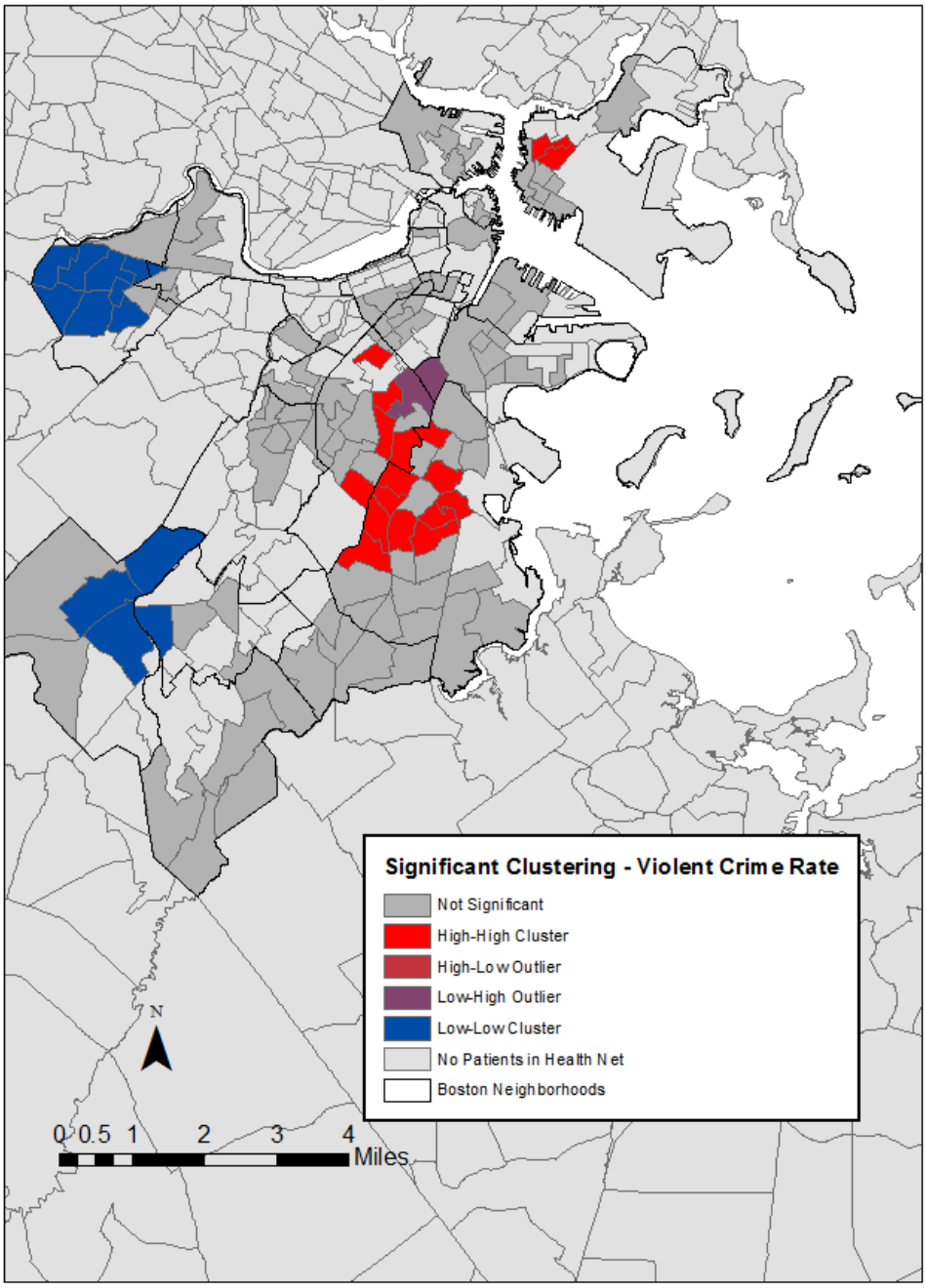

Fig 6. Significant Local Moran's I results for violent crime rate, at the census tract level, after correcting using False Discovery Rate method.

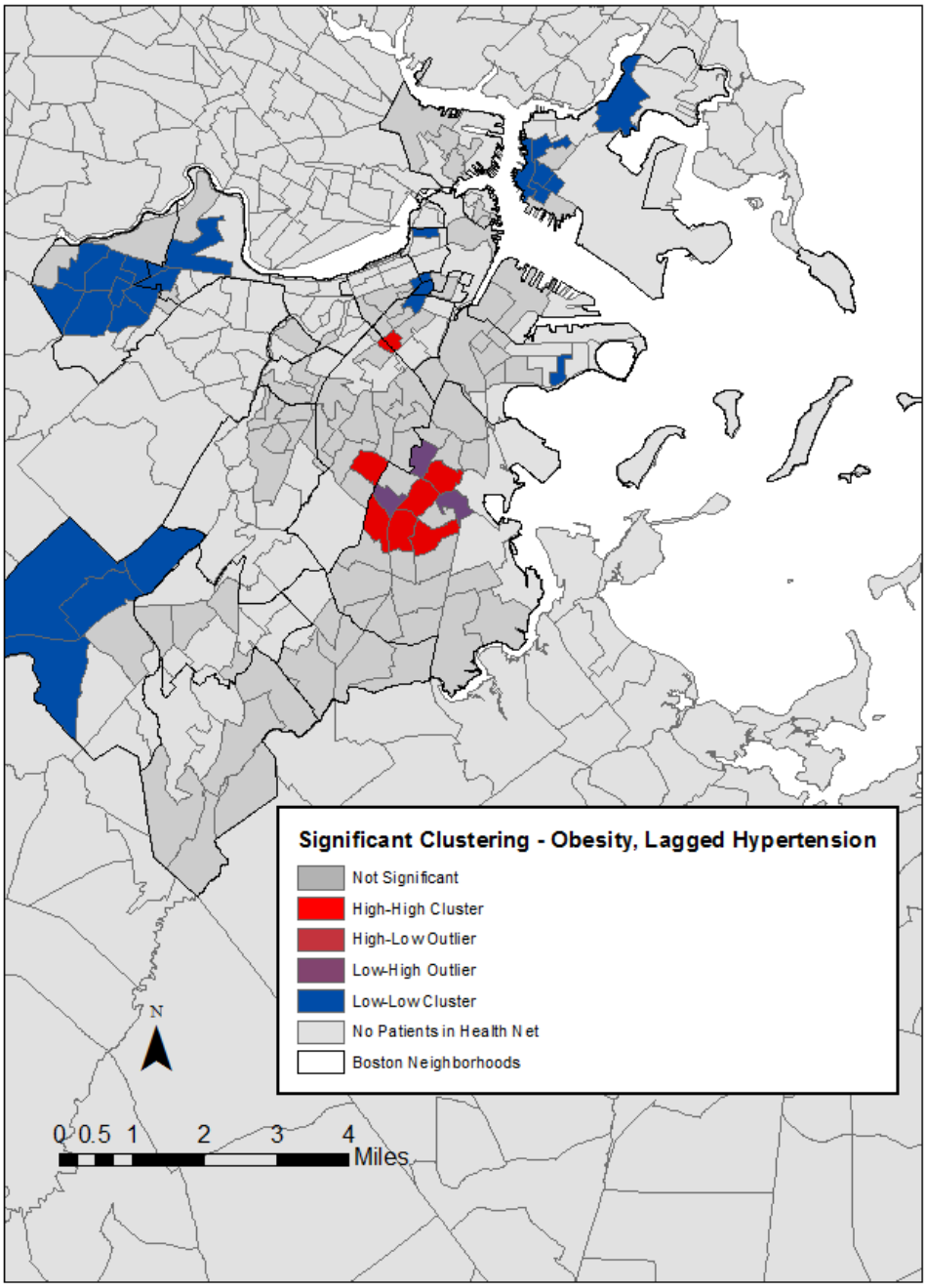

Fig 7. Significant bivariate Local Moran's I results for obesity and hypertension rate, at the census tract level, after correcting using False Discovery Rate method.

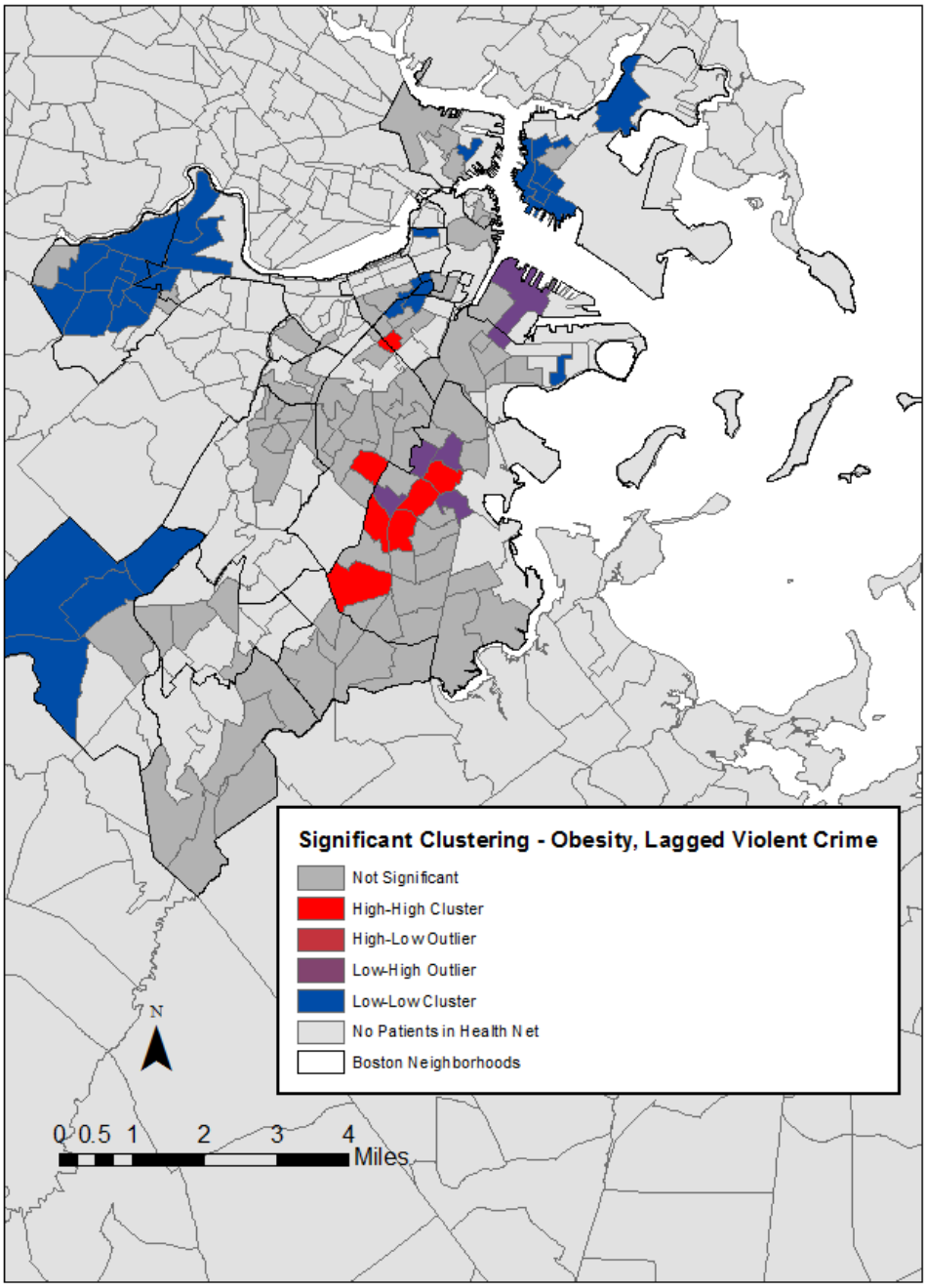

Fig 8. Significant bivariate Local Moran's I results for obesity and violent crime rate, at the census tract level, after correcting using False Discovery Rate method.

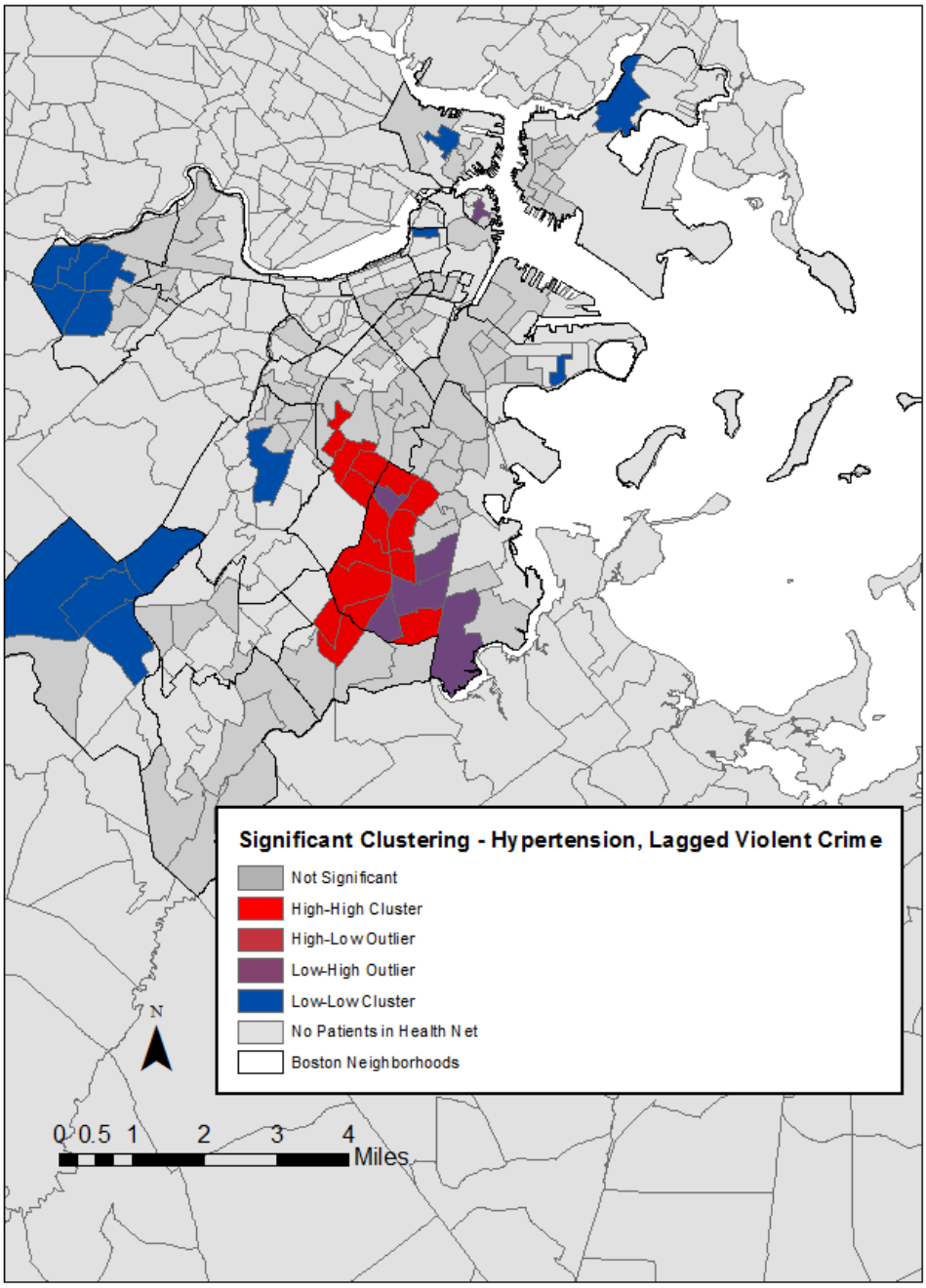

Fig 9. Significant bivariate Local Moran's I results for hypertension and violent crime rate, at the census tract level, after correcting using False Discovery Rate method.

Table 1.

Measure	Mean (SD)
Subjects per census tract	102.6 (47.9)
Age (years)	9.82 (5.14)
Gender *(% female)*	0.49 (0.35)
Cases of obesity per 500 person-years	47.7 (4.37)
Cases of hypertension per 500 person-years	5.21 (0.73)
Violent crimes per square mile	42.5 (13.2)
Median Household Income (US $)	40811.42 (14477.65)
Racial Composition *(% black)*	0.26 (0.12)

Table 2.

Multivariable Poisson models for rates of obesity, then hypertension by Boston census tract

Obesity	Standard Poisson		Lagged Spatial		Neighborhood FE	
	IRR	Robust SE	IRR	Robust SE	IRR	Robust SE
Constant	14.81 **	1.79	14.34 **	1.77	15.49 **	3.52
Hypertension	1.04 **	0.01	1.04 **	0.01	1.04 **	0.04
Proportion black	2.20 **	0.19	1.99 **	0.18	1.32 *	0.15
Proportion female	0.96	0.16	0.94	0.16	1.01	0.26
Average age	1.00	0.86	1.00	0.19	1.00	0.19
Median income	0.98 **	0.00	0.99 **	0.00	0.99 **	0.00
Opportunity Index	0.34 **	0.02	0.35 **	0.02	0.73 **	0.05
Violent Incidents/mi^2	1.01 **	0.00	1.01 **	0.00	1.01 **	0.00
Spatial Lag (Rho)			1.00 **	0.00		
AIC	627.905		555.448		551.238	
Global Moran's I p-value	<0.0001		0.531		0.394	
Hypertension	**Standard Poisson**		**Lagged Spatial**		**Neighborhood FE**	
	IRR	Robust SE	IRR	Robust SE	IRR	Robust SE
Constant	1.70	0.65	1.61	0.62	11.80 **	5.30
Obesity	1.01 **	0.00	1.01 **	0.00	1.01 **	0.00
Proportion black	6.31 **	1.86	5.92 **	1.78	2.35 *	0.84
Proportion female	2.97	1.71	3.04 *	1.67	1.67	1.12
Average age	1.99	1.25	2.14	1.78	1.53	1.07
Median income	0.99 **	0.00	0.99 **	0.00	0.99 **	0.00
Opportunity Index	1.30	0.21	1.32	0.22	1.31	0.28
Violent Incidents/mi^2	1.01 **	0.00	1.00	0.01	1.00	0.01
Spatial Lag (Rho)			1.01 **	0.00		
AIC	242.673		200.589		173.433	
Global Moran's I p-value	<0.0001		0.307		0.267	

**p<0.05 **p<0.001*

IRR: Incidence Rate Ratio

FE: Fixed Effects