Assessing Spatial Relationships Between Rates of Crime and Rates of Gonorrhea and Chlamydia in Chicago, 2012

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Abstract Sexually transmitted infections (STIs) remain serious public health problems particularly in urban environments in the USA. Despite accumulating research into the role of aggregate rates of crime in shaping rates of STIs, few studies account for spatial dependence in the structure of geographical data. Using multiple spatial analysis methodologies, the following study investigated spatial patterns in community area rates of violent, drug, and property crimes and rates of infection of gonorrhea and chlamydia in 77 community areas in Chicago. Moran's *I* analyses confirmed global spatial dependence and statistically significant clusters of STI. Spatial lag regression analyses found that greater rates of drug crimes were associated with higher rates of chlamydia and gonorrhea after adjusting for percent in poverty and racial composition. Finally, a weighted geographic regression identified regions in the urban environment in which local regression coefficient values diverged from their global estimates. Spatial heterogeneity of STIs suggest that public health interventions must be targeted to specific areas of the urban environment with particular attention to substance use.

Keywords Urban health · Crime · Sexually transmitted infections · Spatial analysis

Introduction

Chlamydia and gonorrhea are two of the most common sexually transmitted infections (STIs) in the USA and for the first time since 2006, incidence rates of new infections are increasing at an alarming rate (Center for Disease Control [CDC]) [16]. Moreover, mounting empirical evidence suggests that rates of STIs are not homogeneously distributed across geographic spaces with concentrations of very high rates of infection in urban environments [23, 31, 32, 34, 38, 44, 63, 65]. This unequal burden in the geographic distribution of STI incidence extends to Chicago where overall rates of gonorrhea (308.10) and chlamydia (1013.50) were more than twice the national average (110.7 and 456.10, respectively) in 2014 (Centers for Disease Control [16]; Chicago Department of Public Health) [14]. Prior studies suggest that spatial patterns in STI morbidity exist locally within urban ecologies of neighborhoods and significantly intersect with high rates of poor socioeconomic conditions, racial inequities, and low social cohesion [25, 30, 40].

Research on individual and behavioral factors as drivers of morbidity corroborate findings from studies that examine local variation in spatial distribution of STIs. One example involves the impact of crime rates and criminal justice involvement on heightened risk factors for STI and sexual risk behaviors [41, 42]. Moreover, few studies have conducted spatial multivariable studies examining the association between aggregate rates of crime and rates of STIs. Insight into community and structural-level spatial factors that drive STI

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morbidity is a critical endeavor for public health research by virtue of illuminating where to direct resources, advocate for local communities and target interventions to achieve the greatest gains possible in attenuating the burden of STI morbidity in the USA. In order to address these gaps in extant literature, the following study elucidates the ecological association between community area rates of violent, drug, and property crimes and rates of infection of gonorrhea and chlamydia in Chicago.

Background

In recent years, an increasing number of studies have provided statistical evidence of relationships between aggregate rates of crime and incidence rates of STI [17, 51, 52, 64, 65]. For instance, Chesson et al. [17] conducted a state-level analysis and identified a significant correlation between rates of violent crime and incidence rates of gonorrhea for every year between 1981 and 2010. Additionally, Thomas and Sampson [66] used county-level jail admissions to identify a positive correlation between higher rates of incarceration and greater rates of chlamydia and gonorrhea in 100 counties in North Carolina. These studies suggest a potential spatial relationship that persists overtime in both county- and state-level studies.

Social control theory puts forth an explanation for the relationship between rates of crime and rates of STI that presumes crime erodes community cohesion and perceptions of social control, thus creating a local environment conducive to higher rates of sexual risk behaviors and STI infection [13, 65]. Additionally, the predominant policy response to growing crime trends in the USA involved incarcerating large portions of residents in urban neighborhoods further eroding informal avenues of social control, cohesion, capital, and collective efficacy [19, 20, 47, 56, 57]. Furthermore, research suggests that neighborhoods that meet these sociological profiles have greater rates of sexual risk behaviors and STI [12, 13, 25, 34, 40, 62].

Despite accumulating research into community factors, aggregate rates of crime, and STIs, few studies account for spatial dependence in the structure of geographical data. Stoltey et al. [60] employed a spatial independent random effect regression model and found a significantly positive relationship between rates of incarceration and census-tract-level incidence rates of chlamydia among young women in San Francisco after

adjusting for poverty and other socioeconomic factors. Using aggregate homicide crime rates and community perceptions of violence as proxy indicators of rates of incarceration, Thomas et al. [65] found an association between higher rates of violent crime and rates of STI at the census tract level using data from 1995. Spatial analysis included Moran's *I* tests to determine spatial dependence in rates of STIs and risk differences for predictor variables first with and then in the absence of spatial correlation weights [65]. Research into ecological correlates of rates of STI incidence suggest comorbidities of high rates of STI and crime in urban environments in the USA.

A growing body of empirical literature has mapped incidence rates of STI and examined neighborhood-level covariates other than crime to optimize population-level intervention and prevention strategies [5, 6, 45, 55, 72]. Other spatial drivers of aggregate rates of STIs include relative deprivation, poverty, racial composition, education, and deteriorated physical conditions of neighborhoods [22, 38, 43]. For instance, Owusu-Edusei and Chesson [50] identified spatial dependence in the relationship between greater proportion of African Americans and higher rates of chlamydia and gonorrhea in counties in Texas using spatial lag and other regression techniques. In Chicago, Kaplan et al. [39] identified a linear relationship between greater racial and ethnic homogeneity of Hispanic and African Americans and rates of chlamydia and gonorrhea but did not account for potential spatial dependence in the data. In addition to racial composition, Cohen et al. [22] operationalized characteristics of deteriorated physical conditions in a "broken windows" index and found an association between greater scores and higher rates of gonorrhea.

Research by Jennings et al. [37] at the census level used nonparametric multilevel models to identify a significant association between drug crimes and increased risk of high-risk sexual partnerships and incidence rates of STI. Additional research by Jennings et al. [36] employed novel and modern research methods using spatial longitudinal ecologic techniques to examine temporal and spatial associations between drug arrests and gonorrhea at the census bloc level from 2003 to 2005. The study did not find evidence of a longitudinal link but rather a correlational connection in which arrests for drug crimes predicted a 27 % rise in rates of gonorrhea. These studies emphasize the importance of understanding the role of drug crimes in shaping rates of STI at the population level. The heterogeneity in unit of analysis

ranging from state (i.e., [34]) to census block (i.e., [36]) introduces implications for inference of study estimates including loss of sensitivity to detect significant parameter estimates at increasingly macroscopic units of analysis. Studies at a higher unit of analysis may mask local clusters and significant relationships that are present at the local level.

The Social Ecological Model

Social ecological approaches to public health research provide a conceptual framework that acknowledges social and structural factors as important drivers of STI and HIV [24, 48, 67]. Within this framework, higher order risk factors including crime, poverty, and racial segregation are ecological predictors of STI at the population level of analysis [4]. Moreover, the social ecological model provides an epidemiologic framework that can shape the design of population-level STI intervention and prevention research. Although race is not biologically or behaviorally connected to higher incidence of STI, prior research has found that neighborhoods with greater residential segregation measured by concentrations of African Americans have higher rates of STI [3, 7, 8, 33, 53]. Based on prior literature, this study incorporates percent black in the social ecological framework as a proxy measure for residential segregation of black populations in Chicago. Identifying associations between ecological factors and rates of STI could shed insight into targeted interventions for populations that are disproportionately impacted by higher rates of STI.

Gaps in Extant Literature

The following study addresses several important gaps in the existing body of literature. First, it is unclear if rates of other types of crimes in addition to homicides including drug and property crimes are significantly related to STIs after accounting for spatial dependence in the data. Moreover, by accounting for spatial dependence, this study expands upon an understudied yet important methodological area of research into community-level analyses of drivers of rates of STI. Second, the data used in the study by Thomas et al. [65] is now over 20 years old and the literature would benefit from an analysis on more recent data that takes into account methodological improvements in the reporting systems of both rates of crime and STIs. Additionally, no studies to date have

assessed and visualized nonstationarity and local variation in coefficient values of predictors of incidence rates of STI using geographically weighted regression (GWR) analysis.

In order to address these gaps in the literature, the overarching aim of this study was to elucidate spatial relationships between community rates of crime and rates of STIs after adjusting for racial composition, poverty, education, and unemployment. Specifically, this study pursued three primary objectives: (1) to determine if spatial autocorrelation existed in rates of gonorrhea and chlamydia in an urban environment, (2) to assess the spatial and linear relationship between crime rates and rates of STIs after controlling for potential socioeconomic and demographic confounders, and (3) to pinpoint local patterns in nonstationarity where locally weighted regression parameter estimates of crime rates deviated from their global values, thus indicating local variation in the effects of rates of crime and rates of STIs.

Based upon contributions from prior literature on the effects of community and neighborhood factors, this study hypothesizes that communities with greater rates of drug and violent crime will exhibit greater rates of chlamydia and gonorrhea after accounting for socioeconomic factors. Moreover, it is expected that spatial nonstationarity exists in community rates of gonorrhea and chlamydia. Finally, it is expected that GWR will identify substantial local variation in the parameter estimates for rates of crime in which the coefficient values will be greater in specific community areas throughout the urban environment. In the pursuit of addressing the aims set forth in this study, several spatial and nonspatial statistical techniques will investigate the association between rates of crime and rates of gonorrhea and chlamydia in Chicago.

Methods

Data and Measures

Data for this study were extracted from several publicly available sources. The Chicago Department of Public Health collects annual data on reported cases of chlamydia and gonorrhea. Adjusted rates per 100,000 of the population are computed, and the data are provided in annual infectious disease surveillance reports. Reported cases of crimes are compiled by the Chicago Police

Department and reported according to the types of crimes set forth by the Uniform Crime Incident Reporting System through the Federal Bureau of Investigation (Federal Bureau of Investigation UCR) [26]. Incidence rates were calculated for violent, drug, and property crimes in 2012 by dividing the total number of reported crime events in each category by the total population for each community area and then multiplying the proportion by 100,000. Information on reported crimes and rates of STIs are disbursed through the City of Chicago Online Data Portal (Chicago Online Data Portal <https://data.cityofchicago.org/>) and are freely available for public use.

Variables measuring community-level socioeconomic and demographic characteristics were obtained through the 2007–2011 5-year American Community Survey conducted by the US Census Bureau and included proportion of households below 100 % of the federal poverty line and percent of the population who identify as African American [69]. Data was aggregated from census tracts to community levels using spatial joins and percentages were calculated using 2011 population data for each community area provided by the Social IMPACT research center at the University of Chicago [21]. The variable measuring percent black was dichotomized to reflect community areas that were predominantly African American. Prior to conducting spatial and linear regressions, all of the crime rate variables were converted to the logarithm scale.

Statistical Analyses

Univariate Analysis

In the pursuit of addressing the aims and objectives set forth in this study, descriptive and exploratory analyses first assessed the data for univariate spatial dependence in rates of gonorrhea and chlamydia (objective 1). Using QGIS, maps visualized crude rates of STIs using quintile breaks and provided an initial indication of spatial patterns in the data. Alongside crude rates, empirical Bayes adjusted Moran's I analyses examined the data for global spatial dependence and statistically significant local clusters of community rates of STIs. The Empirical Bayes Moran's I test for autocorrelation used predetermined weighting schemes to operationalize spatial dependence in the data [49, 68]. This study used a queen-based contiguity matrix to spatially weight the data and to identify spatial dependence. The queen

weighting contiguity matrix contained 77 regions with 394 links with 6.65 % nonzero weights, and an average number of 5.11 links. The number of randomized Monte Carlo simulations for empirical Bayes Moran's I calculations for both rates of chlamydia and gonorrhea were 99,999. Queen contiguity weight matrix and Moran's I computations were performed using the spatial analytic software, GeoDa, and subsequently imported into QGIS for visualization. The resulting value of the Moran's I analysis is a spatially weighted correlation coefficient measuring the slope of the regression line through the plotted values of a local unit's weighted value and the weighted average values of the unit's respective neighborhood (in this case community area) [10, 68, 70].

Spatial Regression Techniques

After determining if spatial dependence existed in the outcome data, regression techniques and model-fit diagnostics scrutinized the data for the most appropriate regression model (objective 2). In order to adequately measure spatial heterogeneity in the effects of predictor variables on rates of STI, this study first conducted ordinary least squares regressions to provide initial relationships in the data without consideration for the presence of spatial dependence. LaGrange spatial diagnostics, the Akaike information criterion, and rho estimates were used to select the most appropriate models to analyze the data [70]. The Lagrange spatial diagnostics provides three fit statistics to select between spatial lag, error, and a combination of both lag and error adjustments (spatial Durbin model) [46]. Selection between the spatial error and lag models is determined by significance on either the error or lag maximum likelihood fit statistics [1, 9]. Statistical significance in both the error and lag or the SARMA fit statistics calls for application of the spatial Durbin model. The spatial Durbin model combines error term constraints on parameter estimates (spatial error model) with the introduction of spatially lagged independent variables (spatial lag model) [1]. Spatial autoregressive parameters (rho for spatial lag and spatial error and lambda for the spatial Durbin model) captured the magnitude and statistical significance of the spatial processes that are controlled for in each model. All regression analyses and evaluations of model fit were performed using the statistical computing software R version 5.

Next, GWR identified community areas and regions where parameter estimates of locally weighted

regression coefficients diverged from their respective global estimates (objective 3) [29]. Moreover, this study used GWR to identify the presence of heterogeneity in the distribution of coefficient values throughout the urban study environment. Geographically weighted regression requires selection of either a fixed or adaptive bandwidth with a local spatial weights kernel to relax the assumption of stationarity that is imposed upon data by linear regression techniques [11, 15, 27, 28]. This study utilized a fixed bandwidth function with a Gaussian spatial weight kernel [71]. Analyses were performed in R, and geographically weighted parameter estimates were imported into QGIS to visualize specific locations of heterogeneity in coefficient values. Moreover, the fixed bandwidth function with a Gaussian spatial weight specification accounts for spatial autocorrelation when identifying local variation in parameter estimates.

Results

Exploratory Data Analysis

Table 1 presents overall summary statistics for each of the variables in the study that with ranges of values for the sample of 77 community areas in Chicago. The mean rates of crime for the sample were 1077.91 (47.81–4765.34), 1353.70 (62.62–8748.15), and 4449.28 (876.64–19156.92) for violent, drug, and property crimes, respectively. Overall, the average racial composition was 40.00 % black (0–99.00) and 21.77 % in poverty (3.30–56.50) for the entire study space.

Rates of Gonorrhea

The spatial distribution of rates of gonorrhea and results from Bayes empirically adjusted Moran's *I* tests for spatial dependence provide preliminary evidence of significant spatial clustering in the data (Fig. 1). Rates of gonorrhea ranged between 0 and 1383.30 per 100,000 of the population with a mean of 377.03 (0–1383.30). The empirically adjusted Bayes Moran's *I* global test statistic (0.58, $p < 0.001$) suggests statistically significant spatial clustering of community rates of gonorrhea. Empirical Bayes Local Moran's *I* analysis identified 13 adjacent communities with significantly high rates of

Table 1 Overall descriptive statistics and ranges of values

	Overall	Range
Gonorrhea		
Cases	9181	0–832
Incidence ^{a,b}	377.03	0–1383.30
Chlamydia		
Cases	26,144	10–2247
Incidence ^{a,b}	1049.45	89.40–3160.90
Crime ^{a,c}		
Violent	1077.91	47.81–4765.34
Drug	1353.7	62.62–8748.15
Property	4449.28	876.64–19,156.92
Total	6878.44	1082.39–21,913.36
Population	2,700,493	2770–98,321
Neighborhood controls ^d		
Poverty (%)	21.77	3.30–56.50
Black (%)	40.00	0–99.00

^a Per 100,000

^b Calculated by the Chicago Department of Health

^c Population-adjusted using 2011 population data from Project IMPACT for community areas in Chicago

^d American Community Survey, Census Bureau

gonorrhea adjacent to other community areas with high rates of gonorrhea (high-high). There were a total of 18 communities with significantly low rates of gonorrhea adjacent to other community areas with low rates of gonorrhea (low-low). Two high-high clusters were identified with 3 and 10 community areas, respectively. There were two low-low clusters comprised of 5 and 13 community areas, respectively.

Chlamydia

Figure 2 presents overall rates of chlamydia and results from the Moran's *I* test of global and local spatial dependence. Rates of chlamydia ranged from 89.40 to 3160.90 per 100,000 of the population with a mean of 1049.45 (89.40–3160.90) per 100,000 population. Empirical Bayes Global Moran's *I* tests identified significant spatial clustering (0.56, $p < 0.001$) with 13 community areas in high-high clusters and 17 in low-low clusters. Two clusters of high values were identified, one containing 10 and a second with 3 community areas. One large cluster contained 16 out of the 17 low rate community areas. A total of two community areas with low rates were adjacent clusters of high rates.

Fig. 1 Spatial distribution and empirical Bayes adjusted clusters of gonorrhea in Chicago [18]

Spatial Regression Results

Exploratory data analysis identified significant spatial dependence in the data thus warranting spatial regression model selection and specification. All multivariable spatial and linear regressions were performed on rates of STIs and logged crime rates (drug, violent, and property). All models adjusted for percent in poverty and if the community area was predominantly African American.

Gonorrhea

Table 2 presents regression models estimating the effects of logged rates of drug, violent, and property crimes on rates of gonorrhea. Without adjusting for potential confounders and spatial dependence, a 1-unit increase in logged rates of drug and violent crimes was associated with an increase in the rates of gonorrhea of 146.49 (SD=42.12; $p < 0.001$) and 196.30 (SD=56.61; $p < 0.001$), respectively. However, after adjusting for percent black and poverty, logged rates of violent crime were not significantly associated with rates of gonorrhea in any of the adjusted regression models. The parameter estimates of all the predictor variables were similar

across all of the multivariate regression models. Interpretation of the SARMA fit statistic (6.19, $p < 0.05$) indicated that the spatial Durbin model was a significant improvement over the regression model that did not control for spatial dependence in the data. The Durbin model identified a significant relationship in which a 1-unit increase in drug rates resulted in an increase of 102.51 (SD=33.29; $p < 0.01$) in community rates of gonorrhea. In terms of sociodemographic characteristics, a 1 % increase in poverty resulted in an increase in community rates of gonorrhea of 607.93 (SD=245.99; $p < 0.001$). After adjusting for other covariates and spatial dependence, rates of gonorrhea in community areas that were predominantly black were greater than other community areas by 312.38 cases per 100,000 of the population (SD=62.16; $p < 0.001$).

Chlamydia

Linear and spatial regression results for associations between community area crime rates, predominantly black, percent in poverty, and rates of chlamydia are presented in Table 3. In the unadjusted model, a 1-unit increase in the logged drug and violent crime rates

Fig. 2 Spatial distribution and empirical Bayes adjusted clusters of chlamydia in Chicago [18]

Table 2 Results examining linear and spatial relationships between crime and rates of gonorrhea

	Unadjusted OLS β	OLS β	Error term β	Spatial lag β	Durbin model β
Crime rates					
Drug (log)	146.49*** (42.12)	103.49** (34.28)	86.50** (33.56)	115.01*** (31.45)	102.51** (33.29)
Violent (log)	196.30*** (56.61)	13.49 (52.55)	36.70 (51.99)	-37.38 (51.18)	-6.97 (52.18)
Property (log)	-30.92 (72.49)	12.52 (57.40)	-8.21	13.92 (51.97)	-16.90 (63.59)
Sociodemographic					
Poverty (%)		665.90*** (56.32)	683.55** (267.23)	615.74* (251.09)	607.93*** (245.99)
Black (%)		383.49* (56.32)	363.69*** (59.38)	307.29*** (55.29)	312.38*** (62.16)
Overall model fit					
Adjusted r^2	0.74	0.84			
F test	68.65***	79.36***			
Wald test			5.13*	11.55***	
AIC		1003.5	1002.3	997.51	996.78
Rho/lambda			0.33	0.31***	0.28
Robust Lagrange multiplier diagnostics for spatial dependence					
Error term		0.02			
Spatial lag effects		3.70			
SARMA		6.19*			

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ **Table 3** Results examining linear and spatial relationships between crime and rates of chlamydia

	Unadjusted OLS β	OLS β	Error term β	Spatial lag β	Durbin model β
Crime rates					
Drug (log)	321.10*** (84.68)	197.57** (59.11)	144.92* (57.98)	203.89*** (55.90)	135.76* (58.65)
Violent (log)	586.70*** (113.82)	125.19 (90.61)	157.96 (89.74)	69.99 (92.06)	116.97 (90.86)
Property (log)	-247.87 (145.75)	-117.69 (98.97)	-98.27 (102.20)	-95.05 (94.42)	-49.41 (108.11)
Sociodemographic					
Poverty (%)		2234.72*** (478.61)	2359.29*** (462.57)	2191.95*** (452.21)	2195.05*** (433.34)
Black (%)		843.25*** (97.10)	839.28*** (102.04)	777.61*** (101.29)	814.61*** (109.38)
Overall model fit					
Adjusted r^2	0.80	0.92			
F test	99.04***	154.60***			
Wald test			1.26	2.32	1.61
AIC		1102.8	1104.00	1087.10	1099.80
Rho/lambda			0.18	0.13	0.21
Robust Lagrange multiplier diagnostics for spatial dependence					
Error term		0.73			
Spatial lag effects		0.73			
SARMA		2.72			

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

resulted in an increase in community area rates of chlamydia of 321.10 (SD=84.68; $p<0.001$) and 586.70 (SD=113.82; $p<0.001$), respectively. Violent crime rates were not significant after adding sociodemographic characteristics of predominantly black and percent in poverty. Lagrange multiplier diagnostics for spatial dependence did not find significant fit statistics for selecting the appropriate spatial regression model. However, a lower AIC statistic (1087.10) for the spatial lag model indicated superior fit to the data compared to the rest of the models. In terms of crime rates, each unit increase in community area drug crime rates was associated with an increase in of 203.89 (SD=55.90; $p<0.001$) in rates of chlamydia after controlling for spatial lag and other covariates. In terms of sociodemographic characteristics, a 1 % increase in the proportion of people in community areas in poverty was associated with an increase in rates of chlamydia of 2191.95 (SD=452.21; $p<0.001$). Regarding racial composition, in neighborhoods that were predominantly black, rates of chlamydia were greater than other community areas by 777.61 cases per 100,000 of the population (SD=101.29, $p<0.001$).

Geographically Weighted Regression

Gonorrhea

GWR analyses were performed to examine the data for locations where coefficient values of the regression analysis predicting gonorrhea diverged significantly from global parameter estimates (Table 4). The local coefficient values of drug crimes (−49.74–188.70), percent poverty (200.50–1715.00), and predominantly black (55.41–730.40) diverged substantially from their respective global parameter estimates when allowed to vary by location in the GWR model.

Figure 3 visualizes local variation in coefficient values for logged rates of drug crimes and r^2 values for the community areas within the urban environment. The coefficient values of logged drug crimes were not randomly distributed in space with the greatest impact of coefficient values distributed in the central and southern areas of the study environment. Additionally, variation in local r^2 values suggested that the model performed best in the northern area of the study space with progressively less of the overall variance of the model explained in the southern region. In regions where the effects of logged drug crimes were the greatest, a 1-unit increase was associated with an increase in rates of gonorrhea of 188.73.

Chlamydia

Table 5 presents results from GWR analyses examining local variation in predictors of chlamydia with a Gaussian kernel function and a fixed bandwidth of 5.84. The range in coefficient values for the effects of log drug crime, percent in poverty, and predominantly black ranged from −55.26 to 400.80, 435.10 to 3232.00, and 1018.00 to 1673.00, respectively.

Figure 4 visualizes local variation in coefficient values of logged drug crimes and local r^2 values for explained variance of the overall model across the urban environment. The local r^2 value ranged from 0.887 to 0.986 indicating local variation in the variance explained by the overall model throughout the urban environment. The variables included in the model explained the greatest variance in the northern region with progressively less variance explained in more southern areas. In terms of logged drug rates, coefficient values ranged from −55.26 to 400.75. Specifically, in regions of the study environment where the effects were greatest, a 1-unit increase in logged drug crimes was associated with an increase in the rates of chlamydia of 400.75.

Table 4 Summary of GWR coefficient estimates at data points for regression model predicting rates of gonorrhea

	Min	First quarter	Median	Third quarter	Max	Global
Drug	−49.79	96.55	137.70	155.80	188.70	103.49**
Violent	−212.30	−105.20	−38.16	−15.02	37.08	13.48
Property	−7.51	50.43	86.86	163.80	580.10	12.52
Poverty	200.50	509.20	755.30	882.40	1715.00	665.90***
Majority black	55.41	349.30	446.20	566.10	730.40	383.49***

* $p<0.05$; ** $p<0.01$; *** $p<0.001$

Fig. 3 Geographically weighted local variation in logged drug crime coefficient estimates predicting community rates of gonorrhea and local r^2 values in Chicago [18]

Discussion

Findings from this study identified significant global spatial dependence and variation in the association between crime and rates of STDs (objective 1). Global models of regression analyses presume a single model adequately encompasses all the spatial variation of a study region; however, the data put forth in this study suggest otherwise. This study provides preliminary statistical evidence that the spatial distribution of high and low rates of gonorrhea and chlamydia are more spatially clustered than would be expected if underlying spatial processes were entirely random. Moreover, rates of gonorrhea and chlamydia were extremely locally concentrated and were characterized by clusters of communities with high rates (high-high) and clusters of communities with low rates (low-low). Additionally, very few significant clusters consisting of communities with divergent rates (high-low, low-high) were identified in the Moran's I analyses. These findings suggest extreme local segregation and distinct polarity in the geographic

distribution of rates of gonorrhea and chlamydia in Chicago.

Findings from the multivariate spatial regression analyses identified that logged drug crime rates predicted increased rates of gonorrhea and chlamydia (objective 2). Parameter estimates were similar across all of the models in which the magnitude of the effects of percent in poverty and predominantly black were robust and significantly associated with higher rates of gonorrhea and chlamydia. The GWR analysis identified a wide degree of variation in model fit and parameter coefficient values for logged drug crimes, percent in poverty, and predominantly black predicting rates of chlamydia and gonorrhea (objective 3). This study expands upon prior research suggesting local clustering of STIs and crime in urban environments [34, 37, 54] Moreover, identifying comorbidities with crime and STI is imperative to forging synergistic opportunities for collaboration between criminal justice and public health agencies within local governments and to identify potentially causal relationships for researchers.

Table 5 Summary of GWR coefficient estimates at data points for regression model predicting rates of chlamydia

	Min	First quarter	Median	Third quarter	Max	Global
Drug	-55.26	131.00	212.30	299.20	400.80	197.57***
Violent	-126.40	41.50	117.60	171.10	332.30	125.19
Property	-182.50	-49.45	-23.68	30.27	192.80	-117.69
Poverty	435.10	1544.00	1992.00	2483.00	3232.00	2234.72***
Majority black	1018.00	603.10	889.00	1307.00	1673.00	843.25***

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

Fig. 4 Geographically weighted local variation in logged crime coefficient estimates predicting community rates of chlamydia and local r^2 values in Chicago [18]

These findings generate several important implications for the design of community-level public health and criminal justice interventions. Although the extreme concentrated spatial dependence of rates of chlamydia and gonorrhea identified by Moran's I analyses in this study is alarming, it also emphasizes the importance of future research incorporating geographical considerations into targeted public health interventions and allocation of resources. Moreover, structural- and policy-level interventions that address comorbid criminal justice and public health problems are essential to attenuating high rates of STI in urban environments. Findings from this study present a compelling justification for expanding research into STI testing and treatment services that take into account the profound importance of geographic context in shaping how epidemics of infectious diseases unfold in urban environments. Finally, given the significant effects of drug crimes on rates of STI, increasing community support for substance abuse and mental health treatment as well as reducing the disproportionate arrest of individuals for drug crimes show promise in reducing rates of STIs. Moreover, high rates of STIs are just one of the collateral health impacts of burgeoning rates and drug abuse. Findings from this study emphasize the importance of community interventions to address the impact of violence on the health and well being of communities in Chicago as well as other urban environments.

There are several limitations of this study that warrant explication and provide fruitful avenues of future research. First, the small sample size of community areas (77) may have suppressed the power of models to detect statistically significant relationships between predictor and outcome variables in both the linear and

spatial regression models. Future research is in order that focuses on smaller aerial units to increase the sample size and provide more reliable parameter estimates for STI rates. Second, several community areas reported fewer than 20 cases of either gonorrhea or chlamydia which introduces some degree of instability in the estimation of population adjusted incidence rates and the Chicago Department of Public Health suggests some measure of caution be taken when interpreting parameter estimates. Third, none of the robust Lagrange multiplier diagnostics for spatial dependence or spatial autoregressive parameters (ρ and λ) were significant in the models predicting rates of chlamydia, thus suggesting that the models potentially do not capture the underlying spatial processes in the data. However, the smallest AIC statistics was for the spatial lag model indicating a significant improvement in model fit over the baseline OLS model. A study with a greater number of potential confounders could increase the power to detect significant associations and capture more unexplained variance in the underlying spatial processes in the data. Constraints on the number of predictor variables were imposed by the sample size of 77 community areas. Adding an excessive number of covariates to the models could result in instability and power to detect statistical significance by virtue of decreasing the number of degrees of freedom. Future research at a more local aerial unit would enable the inclusion of more potential confounders, which could improve the power to capture more unexplained variance in the underlying spatial processes in the data.

Similarly, the small sample size precluded the design of scales mirroring prior research by Sampson and

Groves [58]. Sampson et al. [59] and Shaw and McKay [61]. Although there is a lack of consensus regarding appropriate item to sample ratio [2], it would not be statistically prudent to include complex aggregate scales or multiple measures of neighborhood controls in the multivariable spatial regression models used in this study. Future research with a greater number of units of analysis should incorporate and validate scales using multiple neighborhood controls to provide a more nuanced and complex narrative of the influence of social ecologies on rates of STI.

While racial composition at the neighborhood and individual level is a powerful predictor of STIs, there is substantial literature that suggests the effects are largely due to how race interacts with other social determinants and not biologic or behavioral factors [35]. However, in this study, percent black provided a proxy indicator of racial segregation and sheds insight into a structural factor that may play an important role in shaping population-level STI risk. Racial segregation and poverty coupled with high rates of crime converge with existing literature, suggesting that ecological factors may work together to influence rates of STI [35]. Specifically, stiffer surveillance and policing in predominantly African American neighborhoods and disproportionate arrests for drug and violent crimes suggests the possibility of an interaction between race and crime. Future research would benefit from examining interactions between racial segregation and rates of crime to elucidate heterogeneity in the effects of crime on rates of STI based upon community-level measures of racial segregation. Finally, the close relationship between crime and race introduces the possibility of collinearity between the two variables. In this circumstance, the race variable could pull some of the model variance away from other more upstream variables such as rates of crime and poverty. Given these limitations, it is possible that the study underestimates the true magnitude of effects of crime rates and poverty detected in the spatial regression models tested in this study.

Limitations notwithstanding, this study provides findings that are fruitful for public health practice. Although overall rates of gonorrhea are gradually declining locally in Chicago, this study suggests that clusters of communities may have reservoirs of extremely high rates of STI. Reducing rates within the clusters identified in this study would make the largest impact on overall STI morbidity. Targeted resources are essential to tackling HIV and STI epidemics in urban

communities. This study lends supports to future research into interventions to reduce drug use and arrests for drug crimes as a population-level STI prevention intervention. This study provides useful information for public health practitioners and policy makers who are concerned with identifying locations for public health campaigns, health communication, and free testing services. Additionally, future research should investigate the impact of situating substance abuse treatment and grassroots antiviolence campaigns within clusters of elevated STI rates. Findings from this study could empower local communities that are disproportionately impacted by STI, violence, and drug use to advocate for additional resources to expand services and testing programs.

Conclusion

Findings from this study have important public health implications. Community members, policy makers, legislators, as well as public health and criminal justice practitioners must collaborate to generate interventions that operate at levels beyond the individual. Research that goes beyond the individual and includes communities is a powerful tool for public health and social welfare advocacy. Findings from this research must inform the work of local organizations that are working to improve the health and wellbeing of structurally disadvantaged and marginalized communities. Moreover, community-level research opens avenues to address the health of large populations of individuals who are disproportionately impacted by STI morbidity and also extreme socioeconomic, and racial inequities.

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