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Social capital and rates of gonorrhea and syphilis in the United States: Spatial regression analyses of state-level associations

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Abstract

We conducted spatial regression analysis to account for spatial clustering of sexually transmitted diseases (STDs) and to examine the state-level association between social capital (using Putnam's public use data set) and rates of gonorrhea and syphilis. We conducted the analysis for the 48 contiguous states of the United States for 1990, 1995, and 2000 and controlled for the effects of regional variation in STD rates, and for state variation in poverty, income inequality, racial composition, and percentage aged 15–34 years. We compared the results of the spatial regression analysis with those of ordinary least squares (OLS) regression. Controlling for all population-level variables, the percentage of variation explained by the OLS regression and by the spatial regression were similar (mid-90s for gonorrhea and low-70s for syphilis), the standardized parameter estimates were similar, and the spatial lag parameter was not statistically significant. Social capital was not associated with STD rates when state variation in racial composition was included in the regression analysis. In this analysis, states with a higher proportion of residents who were African-American had higher STD rates. When we did not control for racial composition, regression analysis showed that states with higher social capital had lower STD rates. We conjecture that sexual networks and sexual mixing drive the association between social capital and STD rates and highlight important measurement and research questions that need elucidation to understand fully the relationship between social capital and STDs.

Keywords: Social capital; Sexually transmitted diseases; Spatial regression; Sexual mixing; Gonorrhea; Syphilis; USA

Introduction

The continued high geographic and racial disparities and state-level variations in sexually transmitted diseases (STD) rates necessitate new attempts to examine correlates of STD rates (Centers for Disease Control and Prevention, 2004; Institute of Medicine, 1996). Social capital offers a new concept that can be helpful in understanding the variations in STD rates. Social capital is built on the concept that the well-being of societies depends on the cohesiveness and cooperation of their citizens and on their social and civic engagement. Social capital is defined by many scholars as a population-level attribute that measures social relations and connections among people and social organization of communities (Bourdieu,

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1985; Coleman, 1990; Kawachi & Berkman, 2000; Lochner, Kawachi, & Kennedy, 1999; Nan, 2001; Putnam, 2002). Social capital is described as the glue that holds people together and enables them to build stronger communities. Higher measures of trust, cooperation, and reciprocity facilitate collective action, influence norms of behavior, and mobilization of resources for building the infrastructure of communities.

It is hypothesized that higher social capital is associated with better health outcomes, through positive social norms, social networks, social support, and the availability of strong organizational processes' influencing the availability and use of health care services (Kawachi & Berkman, 2000). Rapid diffusion of health information, adoption of healthy norms, and social control of risky behaviors are mechanisms through which social capital is thought to influence health outcomes (Kawachi, 1999). Controlling for population-level variables, higher social capital has been shown to be associated positively with self-rated health and negatively with mortality (Cohen et al., 1997; Kawachi, 1999; Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997; Kawachi, Kennedy, & Glass, 1999; Philadelphia Health Management Corporation, 2004; Subramanian, Kawachi, & Kennedy, 2001). Higher social capital is also associated with better social outcomes, including lower violence and better child welfare (Coleman, 1988; Furstenburg & Hughes, 1995; Kawachi, Kennedy, & Wilkinson, 1999; Parcel & Menaghan, 1993; Sampson, Raudenbush, & Earls, 1997), and with lower levels of risky behaviors associated with binge drinking (Weitzman & Kawachi, 2000), with infection with HIV and other STDs (Bhattacharya, 2005; Campbell, Williams, & Gilgen, 2002; Crosby et al., 2002; Crosby, Holtgrave, DiClemente, Wingood, & Gayle, 2003; Kawachi, Kennedy, & Wilkinson, 1999), and with drug use (Latkin, Williams, Wang, & Curry, 2005; Lindstrom, 2004; Lundborg, 2005). Recent data have shown that social capital is associated with people's self-rated health over and above the beneficial effects of personal social networks and support (Poortinga, 2006).

In terms of the association between social capital and STD rates, only two studies examined this association (Holtgrave & Crosby, 2003; Semaan, Zaidi, & Aral, 2003). Both studies conducted their analyses at the state level and controlled only for poverty and income inequality using forward stepwise ordinary least squares (OLS) regression. Both studies found that higher social capital was associated with lower STD rates, with social capital explaining a large proportion of the variation in STD rates (around 45% for gonorrhea and 35% for syphilis). This result was true for rates of gonorrhea and syphilis for 1999 (Holtgrave & Crosby, 2003) and for rates of other years (1990, 1995, 1998, 1999, and 2000) (Semaan et al., 2003). Neither study, however, controlled for population-level sociodemographic variables or for spatial clustering of STDs.

Three perspectives have been developed for understanding population-level variation in health rates, including STD rates (Aral & Holmes, 1999; Kaplan, 2004; Kawachi & Berkman, 2000; Poundstone, Strathdee, & Celentano, 2004). We refer to these perspectives as the compositional perspective, the clustering perspective, and the structural perspective. From a compositional perspective, variation in STD rates between states is a function of the nonrandom distribution of the population in these states (Kilmarx et al., 1997; Monteiro, Lacey, & Merrick, 2005; Thomas et al., 1999). States with higher STD rates have a larger number of persons who engage in risky behaviors associated with STD transmission (Bernstein et al., 2004; Jennings, Curriero, Celentano, & Ellen, 2005). From an infectious disease perspective and from a clustering perspective, STD rates in a given state are associated with similar STD rates in neighboring states and hence are not randomly distributed in space (Aral, Fullilove, Coutinho, & Van Den Hoek, 1992; Bernstein et al., 2004; Elliott et al., 2002; Fox et al., 1998; Jennings et al., 2005; Kerani, Handcock, Handsfield, & Holmes, 2005; Koumans et al., 2000; Webster, Rolfs, Nakashima, & Greenspan, 1991; Wylie, Cabral, & Jolly, 2005). Regional variation in rates of gonorrhea and syphilis, for example, continue to exist even when these rates are examined separately for Whites and for African-Americans (Farley, 2006). The regional variation in these rates for each racial group is different than that noted for the combined rates of the two racial groups (Farley, 2006). From a structural perspective, higher STD rates are affected by the social, medical, and economic environment (Adimora et al., 2001; Aral & Holmes, 1999; Bunnell et al., 1999; Cohen et al., 2000; Elliott et al., 2002; Gunn, Fitzgerald, & Aral, 2000; St.Louis, Farley, & Aral, 1996). Given these three perspectives, it seems important when analyzing the association between social capital and STD rates at the state level to control for compositional,

clustering, and structural determinants that affect exposure to and infection with STDs and that affect state variation in STD rates.

A conceptual model is helpful for understanding how social capital is associated with STD rates. Farley (2006) proposed a model of 13 predictor variables to explain elevated rates of STDs in African-Americans: historical and current racism, racial segregation, poor education, loss of low-skill jobs, joblessness, long-term concentrated joblessness, drug and alcohol marketing, social disorganization (referred to interchangeably as social capital), male incarceration, unstable relationships, drug and alcohol use, low access to health care, and casual sex. An important contribution of this model is its inclusion of social capital and its proposed association with STDs. The empirical testing of the model requires measurement, collection, and analysis of data on all model variables.

For the purpose of our analysis of the association between social capital and gonorrhea and syphilis rates, we assumed that state variation in racial composition and in economic variables (measured by poverty and income inequality) are associated with STD rates directly and indirectly through social capital and that social capital has a direct and an indirect association with STD rates. Accordingly, we examined the state-level association between social capital and two bacterial STDs—gonorrhea and syphilis (primary and secondary syphilis)—for the 48 contiguous states and controlled for several variables.

We selected gonorrhea and syphilis for our analysis because their case definition has been applied consistently throughout the 1990s (Centers for Disease Control and Prevention, 2004). Although gonorrhea and syphilis are less prevalent than other reportable bacterial STDs, they are still important, show regional and racial disparities, and have been consistently reported across states and time during the 10 years covered in this study. While chlamydia trachomatis is the most commonly reported bacterial STD, trends in chlamydia reporting have been influenced by changes in screening, diagnostic, and reporting practices (Centers for Disease Control and Prevention, 2004). Although the rates of reported primary and secondary syphilis decreased during the 1990s, they remained high among African-Americans and in the South and reflected consistent racial and regional variations, highlighting the importance of conducting our analysis for the 1990s.

Methods

Study hypotheses and years of analyses

We hypothesized that states with higher social capital have lower gonorrhea and syphilis rates. For each STD, we hypothesized that (1) a significant proportion of the variation in STD rates would be explained by social capital or by other population-level variables and (2) that modeling the spatial effects would improve the inference of the association between social capital and STD rates than would OLS regression. Because the Putnam data for social capital used in this study was considered to reflect social capital for the 1990s (Putnam, 1995, 2002, 2004), we examined the association between social capital and STD rates for the 3 years 1990, 1995, and 2000. Conducting the analysis for 1990, 1995, and 2000 was important for understanding the nature and consistency of the relationship between social capital and STD rates. Because the overall results for 1990, 1995, and 2000 were similar and because of space limitations, we report in this paper the results for 2000 (other tables available upon request).

Variables and data sources

Dependent variables

The two dependent variables were gonorrhea and syphilis rates per 100,000 population. The numerator was the number of the new cases for 2000, as reported by the states' health departments to the Centers for Disease Control and Prevention, and was obtained from the STD federal surveillance system (Centers for Disease Control and Prevention, 2001). To facilitate the distinction between states with different population sizes that had zero cases, we added one case to these states. Four states (Montana, South Dakota, North Dakota, and Vermont) reported zero cases for primary and secondary syphilis in 2000. No states reported zero gonorrhea cases in 2000. The denominator represented the Bureau of the Census estimates for the population of each state for 2000 (Census of Population and Housing, 2000). Gonorrhea and syphilis rates were computed as the number of cases in the state divided by the state population, a standard practice when calculating STD rates (Centers for Disease Control and Prevention, 2004). Given the distribution of STD rates and the diagnostic tests for the residuals from the OLS

regression, we used in the regression analyses a natural logarithmic transformation of the rates to accommodate the assumption of normally distributed errors.

Predictor variable-social capital

The measure of social capital, obtained from Putnam's public use data set (Putnam, 1995, 2002), assessed how people connected to family, friends, neighbors, and civic institutions, and was considered to provide a comprehensive snapshot of social capital at the state level for the 1990s (Putnam, 2002). The data on the social capital measure was available only at the state level, for the 48 contiguous states (i.e. excluding Alaska and Hawaii) (Putnam, 2004). Putnam constructed this measure using existing databases and data collected through national surveys, including the general social surveys, the Roper social and political trends archive, and the DDB Needham life style archive. Individual responses to 14 indicators of social capital were aggregated to the state level to form the social capital measure. The correlations between each of the 14 indicators and the social capital measure ranged from the mid-60s to the low-90s (Appendix A), indicating that the 14 indicators measured related, but distinct, aspects of social capital, and tapped one underlying dimension of social capital (Putnam, 2002). The measure (index) of social capital assessed five dimensions: informal sociability, social trust, community volunteerism, community organizational life, and engagement in public affairs (Appendix A). The measure was constructed as a mean of the standardized 14 indicators, each normalized to have equal weight (email communication with Thomas Sander of the Saguaro Seminar, www.ksg.harvard.edu/saguaro, 9/26/2006). A state with a positive value on the social capital index meant that its social capital exceeded the overall average. A state with a negative value on the social capital index meant that its social capital was lower than the overall average.

Control variables

Percentage of African-American residents

The percentage of African-American residents for each state was measured by using the census data for residents who identified themselves as African-American or Black, and by using the 1990–2000 intercensal state and county characteristics population database. We used a natural logarithmic transformation of this variable in the regression analyses to satisfy the assumption of linearity between this variable and the dependent variables.

Percentage aged 15–34

Data on the proportion of people for each state aged 15–34 were obtained from the Bureau of the Census. We used the 1990–2000 intercensal state and county characteristics population database.

Region

The contiguous states of the four regions of the United States were based on the census definition. We included region as a categorical variable in the regression analyses, with the Western region as the reference region. Including the region as a control variable served to control for the regional variation in STD rates and for the different patterns of regional variation in STD rates observed for the White and for the African-American population (Farley, 2006).

Percentage in poverty

This variable measured the percentage of people in a state living below the federal poverty level. For 2000, we used the publicly available data, from the Bureau of the Census, representing the 3-year moving average for 1999–2001 (US Census Bureau, 2005).

Income inequality

By definition, this variable measures the distribution of income between different categories making up a population, such as quintiles. For our analysis, income inequality for each state represented the ratio of the family average income for the top-earning one-fifth of families to the bottom one-fifth of families. For 2000, we used the publicly available data representing the 3-year moving average for 1998–2000, which was calculated by the Center on Budget and Policy Priorities/Economic Policy Institute, using data from the US Census Bureau's Current Population Surveys (Center on Budget and Policy Priorities/Economic Policy Institute, 2000).

Unit of analysis

The state was our unit of analysis. We limited our interpretations to state-level associations and did not make inferences about individual-level associations to avoid cross-level bias or ecologic fallacy (Schwartz, 1994; Susser, 1994). We used an alpha level of 0.01 to guard against type 1 error.

Software

We used SAS for windows to conduct the univariate, bivariate, and the OLS multiple linear regression analyses (2004). We used the free software by Luc Anselin called the GeoDa version 0.9.5-i (Anselin, 2003) and the R version 1.8.1 (www.r-project.org) to conduct the spatial analyses.

Analytic methods

Spatial regression analyses

Spatial clustering of STD infections has received limited attention in multivariate analyses of STD rates (Greenberg, 2004; Koumans et al., 2000). However, it is important, when appropriate, to use spatial regression techniques to account for spatial clustering of STD rates. Spatial clustering or dependence is also known as spatial autocorrelation (Tobler, 1970). In contrast to OLS regression, a spatial lag regression model allows the incorporation of spatial dependence through a spatial lag parameter. OLS regression is built on the assumption that observations are statistically independent. But in the presence of substantial spatial autocorrelation estimates of the predictor variables from OLS regression may be biased and inferences may be inaccurate. Substantively, spatial clustering is important because it influences the transmission dynamics of STD infections.

Analytic strategy

Following univariate analyses, through which we examined distribution of the data, we conducted exploratory spatial data analyses, followed by bivariate and multivariate analyses. At the bivariate level, we examined the correlations between the dependent, predictor, and control variables, using Spearman correlations. We first used forward stepwise OLS regression. Consistent with previous findings (Holtgrave & Crosby, 2003; Semaan et al., 2003), income inequality and poverty, dropped off from this analysis, leaving social capital to be the only variable associated with gonorrhea rates and syphilis rates. When the analysis was repeated using backward elimination, the results were the same. All subsequent regression analyses, both OLS and spatial analyses, examined the association between social capital and STD rates, controlling for regional variation in STD rates, and for state variation in the percentage of African-American residents and the percentage aged 15–34. We conducted the OLS multiple linear regression analysis to examine the residuals and the need for spatial regression analysis, and to compare the results of the OLS and the spatial regression analyses for any differences in the magnitude and significance of the regression coefficients (Appendix B provides details of spatial regression analysis).

While spatial Poisson models are available in the literature, there is no current readily available software package to fit such models, like there is for Gaussian spatial models. Therefore, we applied a common transformation for Poisson type data to approximate a Gaussian distribution, which is not uncommon in practice so that we could apply Gaussian spatial regression methods to our data.

Results

Univariate analyses

The mean values of the control variables were modest and they had small standard deviations (S.D.) (Table 1). For the poverty rate, the mean value was 11% and the S.D. was 3. For income inequality, the mean value was 9% and the S.D. was 1. For the percentage of African-Americans, the mean value was 10% and the S.D. was 10. For the percentage aged 15–34, the mean value was 28% and the S.D. was 2. For the predictor variable, social capital, the mean (0.02) and median (−0.07) values were low, showing positive skew, with a large S.D. of the mean (0.78). For the 48 states, the values for social capital ranged from −1.43 to 1.71, with a higher value indicating higher social capital. With social capital varying widely across the states, the values for the upper quartile and lower quartile were

Table 1
Mean and standard deviation (S.D.) for control, predictor, and dependent variables, ($N = 48$), 2000

Variable	Mean	S.D.
Poverty	11.22	3.03
Income inequality	9.10	1.17
% African-American	10.23	9.72
% aged 15–34	27.94	1.76
Social capital	0.02	0.78
Gonorrhea rate[a]	109.82	82.87
Syphilis rate[a]	1.89	2.17
Log gonorrhea rate[a]	4.28	1.10
Log syphilis rate[a]	−0.10	1.31

[a]Cases per 100,000 persons.

0.55 and −0.49, respectively. The mean value for the gonorrhea rate (110 cases per 100,000 persons) was much higher than that for the syphilis rate (two cases per 100,000 persons). The S.D. for gonorrhea was 83 and for syphilis 2 (Table 1).

The lowest rates for gonorrhea were reported in the West, followed by higher rates in the Northeast and the Midwest. The lowest rates for syphilis were reported in the Northeast, followed by higher rates in the West and Midwest. For gonorrhea and syphilis, the highest rates were reported in the South (Table 2).

Bivariate correlations

The correlations between the control variables and social capital were substantial and statistically significant (Table 3). States with higher poverty, higher rates of income inequality, higher percentage of African-American residents, and higher percentage of residents in the age group 15–34 had lower social capital. The correlations between social capital and gonorrhea rates and syphilis rates were also substantial and statistically significant. States with higher social capital had lower gonorrhea ($r = -0.68$) and lower syphilis ($r = -0.68$) rates. States with a higher percentage of African-American residents had significantly higher gonorrhea ($r = 0.95$) and higher syphilis ($r = 0.81$) rates (Table 3).

Table 2
Regional variation in rates of dependent variables, 2000

STD[a]	Northeast		Midwest		South		West	
	Mean	S.D.	Mean	S.D	Mean	S.D.	Mean	S.D.
Gonorrhea	57.95	41.86	105.85	57.37	188.42	77.45	42.28	30.24
Syphilis	0.52	0.30	1.33	1.82	3.83	2.29	0.77	1.04

Midwest (12 states): Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, and Wisconsin.
South (16 states and the District of Columubia): Alabama, Arkansas, Delaware, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and West Virginia.
West (11 states): Arizona, California, Colorado, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, and Wyoming.
Northeast (nine states): Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont.
[a]Cases per 100,000 persons.

Results of OLS regression analysis

When controlling only for poverty and income inequality, results of the forward stepwise OLS regression analysis showed that social capital was the only significant predictor variable associated with gonorrhea rates and syphilis rates (Table 4). Higher social capital was associated with lower STD rates. The standardized coefficients for social capital were substantial and significant (for gonorrhea: −0.66 and for syphilis: −0.69). The percentage of variation explained by social capital was high (44% for gonorrhea and 48% for syphilis).

Controlling only for regional variation in STD rates and for state variation in those aged 15–34, the OLS regression analysis showed that social capital was negatively associated with gonorrhea rates and with syphilis rates (Table 5). States with higher social capital had lower gonorrhea rates and lower syphilis rates. The standardized coefficients for social capital were substantial and statistically significant (−0.69 for gonorrhea and −0.53 for syphilis). While region was significant, it did not control for all the spatial variation in the data, confirming the rationale for using spatial regression analysis. Age was not significant. The percentage of variation explained, after controlling for the two population-level variables, was higher (69% for gonorrhea and 59% for syphilis).

Controlling for all three population-level variables, regional variation in STD rates, state variation in those aged 15–34, and state variation in racial composition, OLS results showed that social capital was no longer associated with gonorrhea rates or with syphilis rates (Table 6). States with a higher percentage of African-American residents had higher gonorrhea rates and higher syphilis rates. The standardized coefficients for racial composition were substantial and statistically significant (0.98 for gonorrhea and 0.66 for syphilis). Region and age were not significant. The percentage of variation explained after including the three population-level variables in the regression analysis was even higher (94% for gonorrhea and 71% for syphilis).

Exploratory analysis of spatial clustering

The exploratory spatial data analyses, (as shown by the global Moran I statistic, which was positive and statistically significant), showed that gonorrhea

Table 3
Spearman correlations between variables ($N = 48$), 2000

Variable	Variable				
	Poverty	Income inequality	% African-American (logged)	% aged 15–34	Social capital
Poverty	–	0.59***	0.18	0.33	−0.48**
Income inequality	–	–	0.42*	0.16	−0.59***
% African-American (logged)	–	–	–	0.22	−0.77***
% aged 15–34	–	–	–	–	−0.27
Gonorrhea rate (logged)	0.20	0.36*	0.95***	0.26	−0.68***
Syphilis rate (logged)	0.29	0.45**	0.81***	0.30	−0.68***

*$p<0.01$; **$p\leqslant0.001$; ***$p\leqslant0.0001$.

Table 4
Results of regression analysis of social capital and gonorrhea and syphilis rates, 2000

STD and variables	Forward step-wise regression			Spatial lag regression		
	Unstandardized coefficient	Standardized coefficient	Standard error[a]	Unstandardized coefficient	Standardized coefficient	Standard error[a]
Gonorrhea						
Social capital	−0.93***	−0.66***	0.16	−0.40*	−0.28*	0.13
Spatial parameter[b]	N/A	N/A	N/A	0.71***	0.71***	0.10
R^2	0.44	–	–	0.73	–	–
Syphilis						
Social capital	−1.15***	−0.69***	0.18	−0.67***	−0.40***	0.19
Spatial parameter	N/A	N/A	N/A	0.48***	0.48***	0.14
R^2	0.48	–	–	0.59	–	–

Note: Simple regression model. Only social capital was included in this model. Income inequality and poverty dropped off from forward stepwise OLS regression.
NA and (–) indicate not applicable.
*$p\leqslant0.01$, **$p\leqslant0.001$, *** $p\leqslant0.0001$.
[a]Standard error for the unstandardized coefficient.
[b]The spatial parameter is a unitless measure of spatial clustering. It is not affected by standardization of the variables in the analysis.

and syphilis rates clustered significantly in space. This result confirmed the need to conduct spatial regression analysis.

Fig. 1 shows the clustering of high–high gonorrhea rates was mostly in the South (as indicated by the lighter grey color (Red online) and by the label "high–high"). The clustering of low–low gonorrhea rates was found in the West, mid-West, and in two states in the Northeast, as indicated by the darker grey color (Blue online) and by the label "low–low". More specifically, in the South, states with higher-than-average national STD rates formed statistically significant clusters. In the mid-West and West, states with lower-than-average national STD rates formed statistically significant clusters. As expected, there was no significant clustering between states with opposing rates ("high–low" or "low–high"); they appear "whited" out in Fig. 1.

A similar and significant spatial clustering effect was found for syphilis rates (figure available upon request). When our exploratory spatial analysis showed that STD rates exhibited positive and significant spatial autocorrelation because they were not randomly distributed in space, we needed to use spatial regression analysis (Anselin, 1988; Anselin & Bera, 1998).

Spatial regression analyses for gonorrhea and syphilis rates

When controlling only for poverty and income inequality, results of the spatial regression analysis

Table 5
Results of regression analysis of social capital and gonorrhea and syphilis rates controlling for two population-level variables (regional variation in STD rates, and state variation in age composition), 2000

STD and variables	OLS regression			Spatial lag regression		
	Unstandardized coefficient	Standardized coefficient	Standard error[a]	Unstandardized coefficient	Standardized coefficient	Standard error[a]
Gonorrhea						
Social capital	−0.97***	−0.69***	0.16	−0.65***	−0.46***	0.15
Midwest region[b]	1.48***	1.35***	0.28	0.89**	0.81**	0.26
South region[b]	0.90*	0.82*	0.30	0.31	0.29	0.27
Northeast region[b]	0.33	0.30	0.33	0.12	0.11	0.27
% aged 15–34	0.01	0.02	0.07	0.001	0.002	0.06
Spatial parameter[c]	NA	—	—	0.52***	0.52***	0.12
R^{2d}	0.69	—	—	0.77	–	–
Syphilis						
Social capital	−0.88**	−0.53**	0.22	−0.67*	−0.40*	0.22
Midwest region[b]	0.71	0.55	0.39	0.48	0.36	0.36
South region[b]	1.15*	0.88	0.41	0.82	0.63	0.41
Northeast region[b]	0.07	0.05	0.46	−0.04	−0.031	0.42
% aged 15–34	0.02	0.02	0.09	0.003	0.004	0.085
Spatial parameter[c]	NA	—	—	0.27	0.27	0.16
R^{2d}	0.59	—	—	0.62	—	—

NA and (–) indicate not applicable.
*$p \leqslant 0.01$, **$p \leqslant 0.001$, ***$p \leqslant 0.0001$.
[a]Standard error for the unstandardized coefficient.
[b]The Western region served as the reference region.
[c]The spatial parameter is a unitless measure of spatial clustering. It is not affected by standardization of the variables in the analysis.
[d]For OLS, we reported unadjusted R^2.

showed that social capital was the only significant variable associated with gonorrhea and syphilis rates (Table 4). Higher social capital was associated with lower STD rates. The standardized coefficients for social capital were significant (for gonorrhea: −0.28 and for syphilis: −0.40). The spatial lag parameter was significant. The percentage of variation explained by social capital was high (73% for gonorrhea and 59% for syphilis).

Controlling for regional variation in STD rates and for state variation in those aged 15–34, the spatial regression analyses showed that social capital was negatively associated with gonorrhea rates and with syphilis rates (Table 5). States with a higher level of social capital had lower gonorrhea and lower syphilis rates. The standardized coefficients for social capital were substantial and statistically significant (−0.46 for gonorrhea and −0.40 for syphilis). Region was significant only in the regression analysis of gonorrhea. Age was not significant in either regression analysis. The spatial lag parameter was significant only in the regression analysis of gonorrhea. The percentage of variation explained by including two population-level variables in the spatial regression analysis was high (77% for gonorrhea and 62% for syphilis).

Controlling for all three population-level variables, regional variation in STD rates, state variation in those aged 15–34, and state variation in racial composition, the spatial results showed that social capital was no longer associated with gonorrhea rates or with syphilis rates (Table 6). States with a higher percentage of African-American residents had higher gonorrhea rates and higher syphilis rates. The standardized coefficients for racial composition were substantial and statistically significant (0.91 for gonorrhea and 0.64 for syphilis). Region, age, and the spatial lag parameter were no longer significant. The percentage of variation explained after including the three population-level variables in the regression analysis was the highest (95% for gonorrhea and 72% for syphilis).

Comparison of the OLS and spatial regression analyses

When social capital was the only variable included in the regression analysis or when we included only

Table 6
Results of regression analysis of social capital and gonorrhea and syphilis rates controlling for three population-level variables (regional variation in STD rates, state variation in age composition, and state variation in racial composition), 2000

STD and variables	OLS regression			Spatial lag regression		
	Unstandardized coefficient	Standardized coefficient	Standard error[a]	Unstandardized coefficient	Standardized coefficient	Standard error[a]
Gonorrhea						
Social capital	0.04	0.03	0.10	0.08	0.06	0.10
Midwest region[b]	0.24	0.22	0.16	0.12	0.11	0.15
South region[b]	−0.05	−0.05	0.15	−0.19	−0.17	0.15
Northeast region[b]	−0.35	−0.32	0.16	−0.38	−0.35	0.14
% African-American (logged)	0.79***	0.98***	0.06	0.74***	0.91***	0.06
% aged 15–34	−0.02	−0.03	0.03	−0.02	−0.03	0.03
Spatial parameter[c]	NA	—	—	0.18	0.18	0.09
R^{2d}	0.94	—	—	0.95	–	–
Syphilis						
Social capital	−0.07	−0.04	0.28	0.02	0.01	0.26
Midwest region[b]	−0.29	−0.22	0.41	−0.39	−0.30	0.38
South region[b]	0.38	0.29	0.40	0.21	0.16	0.39
Northeast region[b]	−0.48	−0.37	0.42	−0.53	−0.40	0.38
% African-American (logged)	0.64***	0.66***	0.16	0.61***	0.64***	0.15
% aged 15–34	−0.01	−0.01	0.08	−0.02	−0.02	0.07
Spatial parameter[c]	NA	—	—	0.16	0.16	0.16
R^{2d}	0.71	—	—	0.72	—	—

NA and (–) indicate not applicable
*$p \leqslant 0.01$, **$p \leqslant 0.001$, ***$p \leqslant 0.0001$.
[a]Standard error for the unstandardized coefficient.
[b]The Western region served as the reference region.
[c]The spatial parameter is a unitless measure of spatial clustering. It is not affected by standardization of the variables in the analysis.
[d]For OLS, we reported unadjusted R^2.

two population-level variables in the regression analysis (regional variation in STD rates, and state variation in percentage aged 15–34), there were substantial differences in the regression coefficients obtained in the spatial regression analysis as compared with those obtained in the OLS regression analysis. The standard errors of the spatial regression coefficients were marginally smaller and the spatial lag parameter was statistically significant for some of the regression analyses (Tables 4 and 5). After controlling for three population-level variables, the conclusions of the spatial lag regression analyses were similar to those of the OLS analyses for both STDs, and showed that social capital was not associated with STD rates (Table 6). When we did not include percentage of African-American residents in the regression analysis, the OLS residuals for gonorrhea consistently revealed a spatial dependence favoring the selection of a spatial regression model. However, when we included the variable measuring percentage of African-American residents in the regression analyses, we no longer found any significant residual spatial autocorrelation at $p = 0.01$ and social capital lost its significant effect. The spatial lag parameter was not statistically significant after we controlled for three population-level variables and the fit of the spatial regression analyses was better as evidenced, by the scatter plots of the residuals (graph not shown) which were more randomly scattered than the residuals of the OLS analysis. While our analysis showed that STD rates varied by region at the bivariate level, region was not associated with STD rates at the multivariate level.

Discussion

As reported previously, the 48 contiguous states had, on average, modest rates of social capital (Putnam, 2002) and higher gonorrhea rates than syphilis rates (Centers for Disease Control and Prevention, 2001). For this paper, and using spatial

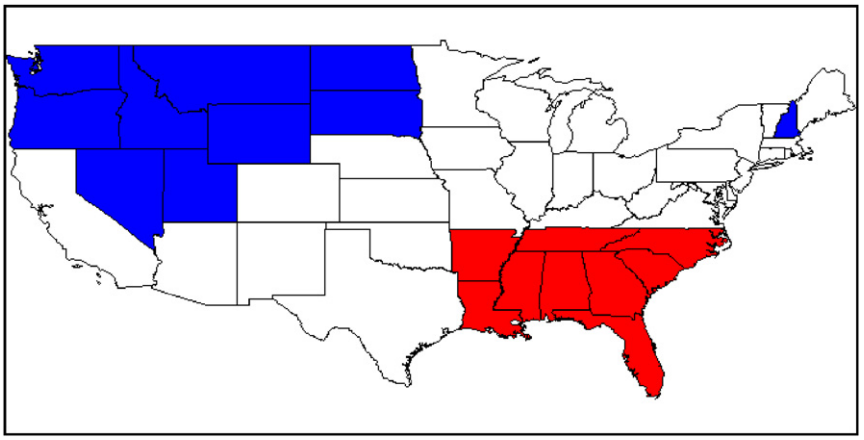

Fig. 1. (Color online) Cluster map for gonorrhea rates, 2000. (Cases per 100,000 persons). (Red, online) The light grey color indicates clustering of high–high rates. (Blue, online) The dark grey color indicates clustering of low–low rates.

regression analysis to account for spatial clustering of STDs, we examined the association, at the state level, between social capital and rates of gonorrhea and syphilis. The control variables were regional variation in STD rates, state variation in poverty, income inequality, racial composition, and percentage aged 15–34 years. Our bivariate analyses showed stronger correlations between racial composition and STD rates than between social capital and racial composition and between social capital and STD rates. Our multivariate analyses showed that social capital was not associated with gonorrhea or with syphilis rates when we controlled for state variation in racial composition and that states with a higher percentage of African-American residents had higher STD rates. When state variation in racial composition was not included in the regression analysis, social capital was negatively associated with STD rates; with higher social capital associated with lower STD rates; a finding reported earlier (Holtgrave & Crosby, 2003; Semaan et al., 2003). The spatial regression analyses also showed that controlling for state variation in racial composition rendered the spatial autocorrelation in STD rates insignificant. The variables included in the regression analyses explained a high proportion of the variation in the STD rates and explained the spatial dependence in the STD rates.

Taken together, the results indicate that the association between social capital and STD rates is influenced by the association between social capital and state variation in racial composition and by the association between state variation in racial composition and STD rates.

The variable measuring percentage of African-American residents serves as a proxy variable accounting for the similarity of gonorrhea and syphilis rates in neighboring states. In other words, the spatial behavior of gonorrhea and syphilis rates is explained by the spatial behavior of the variable measuring percentage of residents who are African-American. Four considerations are relevant to the triangular relationships between social capital, racial composition, and STD rates. These considerations are important because of the cross-sectional and aggregate nature of the data we analyzed and because the empirical evidence is still in its infancy regarding the association between social capital and STD rates. Thus, it is relevant to examine (1) the meaning for controlling for state variation in racial composition, (2) the meaning of the association between social capital and state variation in racial composition, (3) the construct and measurement of social capital, and (4) the implications of our results for developing interventions to control STDs.

Some investigators argue that controlling for racial composition should be abandoned in favor of controlling for social and health variables (Fullilove, 1998) to avoid stereotyping and stigmatizing minority populations and making incorrect inferences (Jones, 2001). Others call for explaining adequately the reasons for controlling for racial composition (Anderson, Moscou, Fulchon, & Neuspiel, 2001; Kaplan & Bennett, 2003), especially that this variable does not capture the heterogeneity in behaviors in the African-American population (Jones, 2001). Still, others argue that abandoning

race as a control variable would perpetuate racial disparities and decrease efforts aimed at ameliorating economic and political conditions that produce social and health disparities (Bonilla-Silva, 2003; Krieger, 2001; Krieger, Williams, & Zierler, 1999).

The variable measuring racial composition seems to behave largely as a proxy marker capturing the effect of social forces that influence sexual behaviors and STD rates. Racial disparities in health rates, including STD rates, are influenced by economic and social processes, socialization patterns, and adequacy and use of health care services (Adimora & Schoenbach, 2002; Krieger, 2000; Thomas & Gaffield, 2003). Contextual and structural factors, such as residential segregation, sexual mixing patterns, and male-to-female sex ratios, play key roles in shaping the socialization patterns that contribute to racial and ethnic disparities in STD rates (Adimora & Schoenbach, 2005; Aral, 2002; Kilmarx et al., 1997; Thomas et al., 1999).

The literature indicates that social capital reflects historical and contemporary social, economic, educational, cultural, and political circumstances (Hean, Cowley, Forbes, Griffiths, & Maben, 2003; Macinko & Starfield, 2001; Pilkington, 2002; Shortt, 2004). Accordingly, it is critical to assess empirically why social capital is highly associated with state variation in racial composition and to understand if there are better predictors of social capital than state variation in racial composition. If better predictors are found, including those predictors in future regression analysis might explain away the association between state variation in racial composition and STD rates.

Social epidemiologists got interested in social capital to explain how income inequality is associated with health disparities and to explain the relationship between community-level variables and health outcomes (Lomas, 1998; Subramanian & Kawachi, 2004; Whitehead, 2001; Wilkinson, 1996). However, the concept of social capital and its association with public health outcomes remains under debate. The criticisms of social capital include that (1) its definition, conceptualization, and dimensions are not clear; (2) it incorporates elements, such as trust and civic engagement, that generate social capital and that are also produced by social capital; (3) it is not uniquely different from other concepts such as collective efficacy, and community capacity; (4) it is not measured by instruments with reliable and valid psychometric properties; (5) the mechanisms by which it influences health, including STD rates, are not empirically evaluated (Hean et al., 2003; Macinko & Starfield, 2001; Pilkington, 2002; Shortt, 2004); and (6), its positive association with public health outcomes, while supported by many (Kawachi & Berkman, 2000; Poundstone et al., 2004; Putnam, 2002), is debated by others (Caughy, O'Campo, & Muntaner, 2003; Lynch, Due, Muntaner, & Smith, 2000; Muntaner & Smith, 2001; Muntaner et al., 2002; Navarro, 2002).

Given these concerns about social capital, it is obvious that more empirical work is needed to measure social capital and to examine its association with STDs. It is our hypothesis that different aspects of social capital affect different health outcomes. For health outcomes, such as self-rated health and mortality, the social support aspect of social capital may be the most important factor, as this factor may explain the positive association with self-rated health and the negative association with mortality (Kawachi, 1999; Kawachi & Berkman, 2000; Kawachi et al., 1999a). It is also our hypothesis that the network aspect of social capital is the mechanism by which social capital affects STD rates. The strong correlation between social capital and STD rates indicates that similar factors, such as shared social networks which are also associated with sexual networks, influence the association between social capital and STD rates. The concept of sexual mixing can explain both of our results: the significant association between social capital and STD rates when we did not control for racial composition and the significant association between racial composition and STD rates in the regression analysis that rendered social capital and spatial autocorrelation insignificant.

The concepts of sexual mixing and characteristics of sexual networks are important in explaining the racial disparities in STD rates (Adimora & Schoenbach, 2005; Aral, 2002). Demographic and environmental factors create social and sexual networks that affect population-level variations in STD rates. Laumann and Youm (1999) showed the critical role of sexual networks in explaining the racial differences in STD rates. They found that higher rates of sexual contact between members of the core group (those who had four or more partners in the past year) and the periphery (those who had only one partner in the past year) among African-Americans facilitated the spread of STD infections in this population. The authors suggested that the sexual segregation of African-Americans from other racial

or ethnic groups resulted in STDs remaining inside the African-American population.

Paradoxically, it is possible that higher levels of social capital or of social networking may result in increased rates of STDs, especially if sexual mixing occurs in populations that have high STD rates, low rates for screening and testing for STDs, low rates of protective measures, and low rates of disclosure of infectiousness. Although a tight-knit network may have positive psychosocial outcomes, the STD consequences of such a network may not necessarily be positive (Kissinger, Niccolai, & Mangus, 2003; Semaan, Klovdahl, & Aral, 2004; Simoni & Pantalone, 2004). Thus, in developing network-based interventions it is important to emphasize the importance of communication about sexual health, negotiation of protective health measures, and health-seeking behaviors. Given the positive and negative role of social capital, future research efforts need to focus on examining the mechanisms by which social capital affects health outcomes, including the positive and negative aspects of the association between social networks, sexual mixing, social capital, sexual behaviors, and STD rates.

The Putnam data base did not offer data for social capital by race to allow us to examine the association between social capital and STD rates by race. Although the criticisms about social capital and its measurement are documented in the literature, we did not find other publicly available national data sets for different measures of social capital, more recent data on social capital, or race-specific measures of social capital. It is relevant to examine the association between social capital and STD rates in other populations, including, for example, the Hispanic population, given the differential growth of this population across the contiguous states.

Because of the limitations in the available data on social capital, we used a uni-dimensional concept of social capital and relied on one aggregate measure of social capital. Because a single measure of social capital may not reflect all relevant domains may cause too much variation in its measurement, as reflected in our data, it is possible that more than one variable should be used for social capital. Additionally, it is important to determine whether individual or contextual levels of social capital are associated with STDs. Separating the individual and contextual effects would require data collected at multiple levels and the use of different analytical techniques. Such a future analysis is important because conceptualization of social capital as a unitary or as a multi-dimensional variable and its different levels of measurement may influence the association between social capital and STD rates. As presented in a recent paper (Poortinga, 2006), we would like to acknowledge the on-going debate about the construct of social capital as the property of individuals (social networks and support) or of the social structure (contextual). We approached our analysis, defining social capital as a population-level attribute. Even in a recent analysis (Poortinga, 2006), social capital contributed to people's self-rated health over and above the beneficial effects of personal networks and support.

Thus, it is important to collect national and more recent data on social capital, to develop race-specific measures of social capital, and to identify how different aspects of social capital relate to different outcome measures. It would be also useful to conduct an analysis to explain why states with a high proportion of African-American residents have low social capital and high STD rates. Such an analysis can help in finding a particular aspect of social capital that affects variation in STD rates. Equally important is the need to collect data for units of analysis that are smaller than a state, including county-level data, and individual-level data. Because the measure we used depended on the social survey approach, where responses from individuals were aggregated to represent the state-level measure of social capital, there is a need to examine the correlation between direct and aggregate measures of social capital. This is important because state residents may perceive the strengths of their states differently or may access different services (Lochner et al., 1999). It is also important to assess the extent to which direct measures of social capital are associated with STDs.

The directionality of the relationship between social capital and health outcomes requires further examination. Given the cross-sectional nature of the data, reverse association could not be dismissed: increased STD rates may result in higher levels of social networking or social capital. A study with repeated waves of data collection is needed to establish the causal association between social capital and STDs. It may also be useful to introduce an explicit temporal dimension in the regression

analysis and to examine the extent to which changes over time in social capital or in other contextual variables influence STD rates. By collecting data over time on social capital, it will be possible to examine how social capital changes over time and to examine the effect that social capital has over time with STDs. Social capital seems to be an important concept that can potentially present new ways to reduce the racial and regional disparities in STD rates. However, in the absence of longitudinal and experimental studies, it is difficult to establish the causal relations that link social capital with STD rates.

Given the differences in the epidemiology and transmission dynamics of different STDs, it is worthwhile to examine the association between social capital and other STDs, including latent syphilis (early latent and late latent), and viral STDs. One previous study showed that higher social capital was associated with lower chlamydia rates and with lower AIDS cases for 1999 (Holtgrave & Crosby, 2003). Given the many lessons that can be learned from improved data collection and from additional analysis, further analyses of the relationship between social capital and other bacterial and viral STDs and between social capital and risk behaviors for STDs and HIV will be helpful.

In summary, our results show that race and spatial clustering either replace or mitigate the relationship between social capital and gonorrhea and syphilis rates. Although neither race nor region is amenable to public health action, it is important to implement interventions that reduce geographic and population disparities in STD rates. Social capital may provide a new avenue for influencing STD rates and network-based interventions that empower populations in regions with high STD rates which can be important in reducing or eliminating regional and racial disparities in STD rates. Network-based interventions can focus on individual-networks as well as on networks within a certain group (Wohlfeiler and Potterat, 2005). The reasons for the high rates of STDs and the related geographic and population disparities are multifactorial and elimination of these disparities continue to require a coordinated interdisciplinary effort, a strong program alliance, and empirical analysis. On the basis of our results, as well as on the basis of the accumulated knowledge in STD prevention and control, we conjecture the importance of network-related, provider-based, and community-level interventions to reduce STD rates and related disparities. The STD prevention and control goals of Healthy People 2010 are to promote responsible sexual behaviors, to strengthen community capacity, and to increase access to high quality services (US Department of Health and Human Services, 2000). Both gonorrhea and syphilis are preventable and curable, highlighting the importance of continually reassessing and refining surveillance, prevention, and control strategies to reduce or eliminate these diseases.

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Note: The findings and conclusions in this article are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention.

Appendix A. : Social capital index—14 indicators (Putnam, 2002)

Dimension and variables	Correlation with social capital index
Informal sociability	
1. Agree that "I spend a lot of time visiting friends"	0.73
2. Average number of times entertained at home in last year	0.67
Social trust	
3. Agree that "most people can be trusted"	0.92
4. Agree that "most people are honest"	0.84
Community volunteerism	
5. Number of civic and social organizations per 1000 population	0.82
6. Average number of times volunteered in last year	0.65
7. Average number of times worked on community project in last year	0.66

Community organizational life	
8. Average number of club meetings attended in last year	0.78
9. Average number of group memberships	0.74
10. Number of non-profit (501[c]3) organizations per 1000 population	0.82
11. Served as officer of some club or organization in last year (%)	0.83
12. Served on committee of some local organization in last year (%)	0.88
Engagement in public affairs	
13. Turnout in presidential elections, 1988 and 1992	0.84
14. Attendance at any public or school meeting in town last year (%)	0.77

Appendix B. : Details of the spatial regression analysis

Exploratory spatial data analysis

Using Moran's I statistic, we tested the null hypotheses of spatial randomness against the alternative hypothesis of positive and significant spatial autocorrelation. A positive and significant statistic would indicate that similar STD rates clustered in space, where states with high STD rates would have neighboring states that also have high STD rates (high–high) and states with low STD rates would have neighboring states that also have low STD rates. Visually, we explored spatial dependence using the Moran Scatter map.

Spatial weights

In order to apply the methods of spatial regression analysis, we needed to express the nearness of geographic units, in this case states, using a spatial weights matrix whose elements represented the binary spatial weights assigned to pairs of states. (Anselin, 1992) We used a first-order contiguity weighting matrix; where we considered states that shared boundaries and vertices as contiguous. By selecting the states that yield a ring around each state, the spatial weights matrix represented an assumption about the potential reach of sexual relations and networks across states. We made a binary assignment such that all states that were considered contiguous were assigned a value of 1 and the remaining pairs of states were assigned a value of zero. To facilitate the interpretation of the estimated spatial dependence parameters based on this matrix, we followed the common procedure of row standardizing the weights, by dividing each element of the matrix by its corresponding row sum, to be between 0 and 1. The product of the spatial weights matrix and the STD rate of the neighboring states yielded, for each state, a spatially lagged variable, called the spatial lag parameter. This parameter was included as a covariate in the spatial lag regression analysis and represented a weighted average of the STD rates of the neighboring states.

Ordinary least squares regression

After we observed spatial dependence in the exploratory spatial analysis, we estimated the usual OLS multiple regression model, applied tests for normally distributed and homoskadistic errors, and used the regression residuals to test for spatial lag and for spatial error. Either form of spatial dependence, lag or error, would result in spatial autocorrelation among the regression residuals, an indication that the explanatory variables in the OLS model did not account for the spatial dependence in the outcome variable. We examined clustering of the residuals of the OLS models using an adjusted Moran's I statistic designed for OLS residuals. Moran's I can be interpreted as summarizing the strength of the association between model residuals and neighboring residuals (Baller, Anselin, Messner, Deane, & Hawkins, 2001). Under the assumption of independence, or no spatial autocorrelation, one would not expect to see a significant relationship summarized by Moran's I.

Choice of the spatial regression model

When spatial dependence was detected, we needed to incorporate it in the regression analysis. Along with theoretical reasoning, we used two common tests, the robust Lagrange Multiplier (LM) test for spatial lag dependence and the robust LM test for spatial error dependence, for deciding, in part, whether the spatial lag model or the spatial error model was the correct alternative to the OLS model (Anselin & Bera, 1998). The tests assessed, respectively, the relative sizes of two covariances. The first covariance involved model

residuals and neighboring values of the dependent variable. The second involved model residuals and neighboring values of model residuals. Theoretically, the larger covariance would determine, in part, the spatial regression model that would be used. If the former covariance was larger, the lag model was preferred. If the latter covariance was larger, the error model would be used. In addition to these statistical results, our knowledge of the literature on sexual mixing patterns helped in determining the choice of the model that we used. By definition, the spatial lag model would allow for an influence of the dependent variable (STD rates) of neighboring states above and beyond the influence reflected in error terms. The spatial lag model would imply that the values of STD rates of neighboring states were associated with each other—true conceptually for sexual risk variables and for sexual networks. The spatial error model would imply the presence of unmeasured covariates that would account for any remaining clustering of STD rates, indicating that STD rates clustered in space only to the extent that covariates of STD rates also clustered in space. Theoretically, the spatial lag model was more in sync with STD transmission dynamics and was our model of choice for the spatial analysis. This choice was also substantiated by the results of the LM tests. Once the spatial model was chosen and estimated, residuals were tested again for any remaining spatial dependence. Both spatial models, the spatial lag and the spatial error, incorporate spatial dependence and are estimated by the method of maximum likelihood (Anselin & Hudak, 1992; Haining, 1990).

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