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The relationship between five different measures of structural social capital, medical examination outcomes, and mortality

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ABSTRACT

Higher social capital is associated with improved mental and physical health and reduced risk of premature mortality. We explored the relationship between five measures of structural social capital and 1) intermediate health outcomes (elevated C-reactive protein, cholesterol, blood pressure, and serum fibrinogen) and 2) distal outcomes (cardiovascular and all cause mortality). We did so using the National Health and Nutrition Examination Survey III 1988–1994 linked to the National Death Index with mortality follow-up through 2006. We employed ordinary least squares regression for the intermediate outcomes, seemingly unrelated regression (SUR) to consider combined effects, and Cox proportionate hazards models for mortality outcomes. We then performed extensive sensitivity analyses, exploring the contribution of various variables and reverse causality. We find that our measures of social capital did not predict statistically significant changes in the laboratory biomarkers we study. Nevertheless, belonging to organizations or attending church >12 times per year were associated with reduced all cause mortality (hazard ratio [HR] = 0.81, 95% confidence interval [CI] = 0.70–0.93 and HR = 0.72, 95% CI = 0.60–0.86, respectively). In SUR analyses, however, combined laboratory values were significant for all measures of social capital we study with the exception of visits to neighbors. This suggests that some forms of structural social capital improve survival through small changes in multiple measures of biological risk factors rather than moderate or large changes in any one measure.

Introduction

Social capital, or one's "networks, norms, and [degree of] trust" (Portes, 2000b; Putnam, 1995) is broadly believed to be an important determinant of population health (Kawachi, Subramanian, & Kim, 2010). Social capital is associated with improved mental health (McKenzie, Whitley, & Weich, 2002), improved cardiovascular health (Lomas, 1998), and lower overall mortality (Lochner, Kawachi, Brennan, & Buka, 2003). However, the biological mechanisms through which social capital works have not been extensively studied, and the few previous studies of biological mechanisms have suggested that the effect of social capital on mortality cannot be explained by improvements in conventional biological measures of health. We hypothesize that structural social capital affects distal outcomes (i.e., all cause mortality, cardiac-associated mortality) via small changes in many different intermediate health outcomes (e.g., elevated cholesterol or blood pressure), and that these many small changes ultimately translate into large reductions in mortality. We further hypothesize that not all forms of social capital impact health and longevity in the same way.

Definitions

Social capital is a multi-dimensional construct. Therefore, any study of social capital must clearly define the dimensions that are being measured (Portes, 2000a). At its core, social capital is a term meant to capture the social value of human interactions in society in the same way that that human capital captures the value inherent to the cognitive contributions to society or physical capital captures the value inherent to machines or buildings in a society (Coleman, 1988; Putnam, 2001).

Social capital is sometimes categorized into four general categories: collective efficacy, social trust and reciprocity, participation in voluntary organizations, and social integration (Harpham, Grant, & Thomas, 2002). It has also been conceptualized as "bonding" capital, which links similar individuals (e.g., members of a golf club), or "bridging" capital, which links dissimilar individuals (e.g., immigrant rights groups and their beneficiaries) (Kim, Subramanian, & Kawachi, 2006). Two useful broad conceptual

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categories are: 1) cognitive social capital, which involves internal states such as trust and 2) structural social capital (the form we evaluate in the present study), which involves the availability and use of social organizations or connections such as organizational membership or connections to others (Harpham, Grant, & Thomas, 2002). Another distinction is collective social capital and network social capital (Li, Pickles, & Savage, 2003; Portes, 2000a), with the former emphasizing the quantity of ties (e.g., the number of club memberships) and the latter the quality of ties (e.g., whether the participant is linked to powerful individuals). The central purpose of the present study, though, is not to dissect different forms of social capital, but rather explore the relationship between intermediate and distal health outcomes associated with various measures of structural social capital.

The present paper measures the frequency of interaction within forms of structural social capital. We first explore frequency of human interaction within what might be conceptualized as three levels of intensity of relationships. These include, in ascending order of intensity, frequency of contact with: 1) neighbors, 2) friends, and 3) relatives. Here, it is tempting to assume that, on average, each subsequent category will be indicative of more intensive and meaningful social connections. That is, each level may confer more meaningful benefits for health and economic well-being (e.g., if one had a connection to a desirable employer she would likely prefer to refer a relative over a friend, and a friend over a neighbor).

We also measure one's frequency of attending clubs and church, two measures of social participation. Of these, church attendance may, incidentally, be more tightly linked to normative behaviors that are, on average, associated with health and longevity. Finally, we measure meeting attendance, which may be conceptualized as a very broad measure of engagement within clubs, churches, etc. Together, these might be conceptualized as structural, rather than cognitive, forms of social capital (Grootaert & Van Bastelaer, 2002; Hitt, Lee, & Yucel, 2002; Uphoff & Wijayaratna, 2000).

Conceptual framework

Many of the measured dimensions of social capital—particularly those we study here—have been linked to sizable impacts on mortality (Kawachi et al., 2010). For some forms of social capital (e.g., church attendance), we might expect that strong norms surrounding health behaviors could produce large impacts on mortality. However, for other forms of social capital it is less clear from where these sizable impacts arise. For instance, a politically active group might advocate for social benefits that have a health and survival advantage, such as health insurance in the US (Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997). However, the impact of social capital on health insurance rates may not be large, and, regardless, those who possess health insurance live only weeks to months longer than those who do not (Muennig, Franks, & Gold, 2005). Thus, the attributable risk of structural social capital on mortality should be much smaller than has been observed in the literature (Knack & Keefer, 1997). However, in practice, social capital could plausibly produce many different health effects that add up to larger impacts on mortality. For instance, belonging to a group such as Neighborhood Watch may improve health by reducing rates of neighborhood crime, it might also provide access to financial resources in times of need (e.g., providing financial support from other members), or it might induce or reduce physiologically damaging states (e.g., fear or psychological stress) (Islam et al., 2006; Kubzansky & Kawachi, 2000; Song, 2011). Social support conferred by friends and family can buffer psychological stress, thereby improving both physical and mental health (Berkman, 2009; Berkman, Blumenthal, Burg, Carney, Catellier, Cowan, Schneiderman, et al., 2003; Berkman & Syme, 1979; Kawachi & Berkman, 2001; McEwen, 1998).

Since structural social capital can produce small impacts through many different mechanisms that add up to large impacts on mortality, we might expect small impacts on an array of biological systems. Each of these impacts may seem unimpressive on its own, but the net effect can be large. For instance, spending time with friends or family—or even with neighbors—may confer stress buffering social support with its associated improvements in cholesterol levels, hypertension, C-reactive protein (CRP), and fibrinogen (Glazier, Elgar, Goel, & Holzapfel, 2004; Kessler, Price, & Wortman, 1985; McEwen, 1998; Turner, 1981; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). It may also confer beneficial social connections leading to a quality job that offers health insurance. The quality job may also improve socio-economic status, a risk factor that is also associated with improved biomarker profiles (Alley et al., 2006; Berkman, 2009; Cohen et al., 1997; Davey Smith, Harbord, & Ebrahim, 2004; Ernst & Resch, 1993; Kawachi & Berkman, 2001; Markowe et al., 1985; Muennig, Sohler, & Mahato, 2007; Pradhan, Manson, Rifai, Buring, & Ridker, 2001; Ridker, Hennekens, Buring, & Rifai, 2000). Together, these small impacts may produce add up to large changes in some biomarker profiles, or may add up to small changes in many biomarker profiles.

We consider multiple health outcomes simultaneously (Folland, 2007). We explore these effects using medical examination data linked to mortality data. Hypertension, hypercholesterolemia, elevated levels of CRP, and elevated levels of serum fibrinogen have each been linked to both behavioral risk factors and psychosocial stressors, all of which increase one's risk of premature death later in life (Gruenewald, Seeman, Ryff, Karlamangla, & Singer, 2006).

It is possible to further tease apart the independent effects of psychosocial stressors and behavioral risk factors via blood cotinine levels—a breakdown product of nicotine use; this helps us understand first- or second-hand cigarette smoke exposure as a potential mediator. Finally, it is plausible that many small reductions in laboratory values synergize to add up to larger reductions in mortality. We therefore use seemingly unrelated regression to test weather small, clinically non-significant changes in cholesterol, blood pressure, CRP, and serum fibrinogen, induced by higher social capital add up to larger reductions in mortality (Zellner, 1962). We also conduct sensitivity analyses to test whether there are cohort effects (e.g., older generations have different types of social capital than younger ones) and whether there is evidence of reverse causality (e.g., sick people cannot participate in groups or rely more on friends and family relative to well people).

Methods

Data

We examined the NHANES-III, a nationally representative cross-sectional survey of 33,994 adults aged $\geq$18 in the United States conducted between 1988 and 1994 (NCHS, 2010). This survey was linked to up to 18 years of prospective mortality follow up data (through 2006) via the National Death Index (NDI) to form the NHANES-III–NDI (NCHS, 2011b, 2012). A subsample of NHANES-III participants had medical exams (30,818 in the mobile examination center, 493 in their homes). In addition to a physical examination and food consumption interview, laboratory tests were obtained on sub-samples of the cohort. The population size tested for blood values of CRP, hypertension and cholesterol varies depending on the test. Table 1 lists the sample size by variable after excluding missing values. We restricted our sample to participants aged 18–65.

The present study includes only those subjects who were examined medically and who had valid laboratory test values. Those without complete medical examination data do not differ from those with complete medical examination data with respect

Table 1
Descriptive statistics of our analytical sample by each measure of social capital. All measures are dichotomized into <12 or ≥12 times per year except club membership, which is dichotomized into yes/no. 1978–2002 General Social Survey-National Death Index dataset with follow up through 2008.

	Visit friends or relatives		Visit neighbors		Attend church		Attend meetings		Belong to clubs	
	<12	≥12	<12	≥12	<12	≥12	<12	≥12	No	Yes
Number of individuals	1080	11,546	6637	5988	5618	7008	953	2527	9145	3485
Average age***	42	38***	38	38	37	39*	41	41	37	41*
Gender (%)										
Female	49.0	52.0	52.1	51.4	45.9	57.9***	36.89	54.34***	53.56	48.6***
Male	51.0	48.0	47.9	48.6	54.1	42.1	63.11	45.66	46.44	51.4
Race (%)***										
White	76.4	83.3***	82.1	83.6	85.9	79.7***	93.69	85.48***	79.82	88.16***
Black	16.8	12.2	12.8	12.1	9.6	15.4	4.52	11.17	14.41	9
Other	6.8	4.6	5.1	4.3	4.5	4.9	1.79	3.35	5.77	2.84
Census region (%)										
Northeast	16.3	20.1	21.8	17.9	22.4	17.2***	23.35	19.9	19.25	21.04
Midwest	18.3	24.6	24.2	24.2	23.8	24.6	27.39	25.36	23.17	26.02
South	38.5	34.8	32.6	37.5	31.0	39.2	29.51	33.42	36.61	32.15
West	26.9	20.5	21.4	20.5	22.8	19.0	19.76	21.32	20.97	20.8
Self-rated health (%)***										
Excellent	14.1	22.3***	21.4	22.1	19.8	23.9**	26.72	27.63	18.57	27.3**
Very good	25.0	32.8	31.1	33.6	32.1	32.5	36.62	37.13	29.7	36.96
Good	34.7	32.6	33.2	32.3	34.4	31.0	28.14	26.86	35.82	27.32
Fair	21.2	10.4	12.1	10.2	11.8	10.5	7.28	7.15	13.39	7.19
Poor	5.1	1.8	2.2	1.9	2.0	2.1	1.23	1.23	2.51	1.23
Education (%)***										
< High school	37.4	21.0***	22.3	21.9	25.3	18.8**	13.82	9.32***	28.57	10.8***
High school	32.0	35.5	34.8	35.8	36.8	33.7	32.8	28.13	38.48	29.63
Some college+	30.6	43.4	42.9	42.3	38.0	47.5	53.39	62.55	32.95	59.57
Cholesterol (mg/dl)	203.26	199.31	200.4	198.76	198.8	200.35	202.6	200.98	198.5	201.56*
Systolic blood pressure (mm/hg)**	121.15	118.23**	118.56	118.29	118.6	118.23	120.1	118.76*	118.0	119.21*
Diastolic blood pressure (mm/hg)***	76.19	73.74***	73.92	73.88	73.97	73.81	75.41	74.36*	73.44	74.71*

* $p < 0.05$.
** $p < 0.01$.
*** $p < 0.001$.

to their sociodemographic profiles. The study employs a complex sampling design consisting of sampling at the county, household, and individual levels, and appropriate weights were applied to all analyses. The NHANES-III protocol was approved by a governmental Institutional Review Board. All subjects were financially compensated for participation in the study.

Laboratory specimens

The complete methodology of variable measurement for NHANES-III is recorded elsewhere (Third National Health and Nutrition Examination Survey (NHANES III), 1996). Blood pressure readings were taken over four readings, and the average of the last two blood pressure recordings was used to calculate participant blood pressure. CRP was measured by high-sensitivity CRP assay using a BN II nephelometer (Dade Behring, Deerfield, IL) at the University of Washington Medical Center. Serum total cholesterol was measured at Johns Hopkins University Lipoprotein Analytical Laboratory using a Hitachi 704 Analyzer (Boehringer Mannheim Diagnostics, Indianapolis, Indiana). Data on low density lipoprotein levels and statin use were not available for a sufficient number of participants to analyze.

Measures

The dependent variables of interest are all-cause mortality, cardiovascular mortality, and the following laboratory measures: CRP (mg/L), blood pressure (systolic and diastolic mmHg), serum fibrinogen (mg/dL), and total cholesterol (mg/L). We chose cardiovascular mortality as a separate outcome measure because there is an extensive literature examining the relationship between cardiovascular mortality and both low perceived social support and social capital, the most rigorous of which has produced equivocal results (Berkman, 2009; Berkman, Blumenthal, Burg, Carney, Catellier, Cowan, Schneiderman, et al., 2003; Ellaway & Macintyre, 2007). The independent variables of interest are five measures of social capital, each dichotomized to address non-linear associations with the outcome measures of interest and to estimate hazards: the number of visits per year with friends or relatives, the number of visits per year with neighbors, number of times per year attending church or religious services, membership in any clubs or organizations, and how often the participant attends meetings outside of work. We assumed that meaningful measures of social capital constituted at least 1 visit per month on average (i.e., <12 visits per year or ≥12 visits per year) to friends or family, neighbors, church, or meetings. We chose 12 visits *a priori* as a cutoff point because a monthly visit was felt to represent the minimal meaningful exposure in order to produce a health outcome. These measures were tested at different cut-off points, but different cut-off points did not substantively influence outcomes. Belonging to any club was dichotomized as yes or no.

For demographic measures, we adjusted for the following covariates in our analyses: age, race (white, black, and other), gender, educational attainment (<high school, high school diploma, some college or more), and geographic region (northeast, Midwest, south, and west). We included age, race, and gender to

adjust for non-modifiable characteristics of the cohort that plausibly co-vary with social capital. We included geographic region because regional differences in social capital may be confounded by regional differences in health practices (e.g., those in the southern United States may have both different levels social capital and a higher risk of cardiovascular disease than those in the northern United States.) We restricted our sample to those aged between 18 and 65 because there is evidence of survival effects beginning around age 60–70, and because the NHANES-III–NDI has unusually long follow-up. We included educational attainment, because education is correlated both with social capital and with health and may be a confounder since educational attainment likely concluded before social capital and health were measured. We chose not to include income in addition to education in the main model because social capital can improve both employment and income, and these may subsequently improve health, such that income may mediate the relationship between social capital and health. Finally, we included survey year in the model because levels of social capital may change over time and the relationship between social capital and mortality could plausibly change over time.

We considered two measures of mortality: death due to all causes, and death due to cardiovascular disease in particular. Follow up of participants for survival extended through December 15, 2006. The *International Classification of Diseases, Ninth Revision* (ICD-9) codes were used to classify deaths from cardiovascular disease between 1988 and 1998, and 10th revision codes (ICD-10) were used for deaths occurring from 1999 to 2006. ICD-9 and ICD-10 codes were recoded as underlying classification of death (UCOD) in NHANES III-NDI. Deaths from cardiovascular-related diseases included deaths from ischemic heart disease (I20–I25), heart failure (I50), essential hypertensive heart disease (I11–I13), cerebrovascular disease (I60–I69) and atherosclerosis (I70–I71). Non-cardiovascular-related mortality included all deaths except the aforementioned.

Statistical approach

First, we built ordinary least squares models to explore the relationship between the intermediate outcomes and our measures of structural social capital adjusting for the above-mentioned covariates. Next, we employed Cox proportional hazards models to further examine the association between mortality and the social capital variables, adjusting for demographics as well as self-rated health, education, blood pressure and cholesterol in addition to the covariates included in the ordinary least squares models. These additional covariates are included to get a sense of the effect of social capital on mortality independent of the intermediate mediators. Given the complex sampling design mentioned above, we weighted the dataset before conducting our analyses. Replicate weights using Fay's method were used to correct the standard errors of estimates. We also sorted the input data prior to analysis (NCHS, 2011a).

We used two methods to test the proportional hazard assumption. First, we examined the interaction of survival time with social capital, and the interaction was not significant. We also checked the log–log survival curves for each of the social capital variables, and no violations of the proportional assumption were observed. However, for the number of visits with neighbors variable, the two curves overlapped.

We then did a series of sensitivity analyses. We explored the effect of smoking in a sensitivity analysis, adding cotinine (a breakdown product of nicotine) into our regression and Cox models to investigate whether and how the coefficient changes and in which direction. This was done to explore the impact of social capital on smoking, both as primary smoking and second hand smoke, on the outcomes of interest. (Exposure to others may plausibly reduce smoking behavior, increase it, or indirectly impact health because others around the participant smoke.) In another sensitivity analysis, we controlled for health insurance to investigate whether there was an indirect effect of nepotistic connections on access to medical care broadly defined (e.g., some may have garnered a better job via nepotistic connections, and health insurance effects on our intermediate and distal outcome measures could remain even after controlling for income effects.) We also ran the models before and after restricting the sample to healthy participants. We did so to test the effect of reverse causality on the association between social capital and health (e.g., healthy people may be more likely to attend church or to join clubs). Finally, we conducted sensitivity analyses on the effect of sample weighting and varying the cutoff points used in our measures of social capital.

To address multiple comparisons, we both conducted Bonferroni corrections and seemingly unrelated regression (SUR) across all laboratory and mortality outcomes (Kling & Liebman, 2004; Zellner, 1962). SUR explores correlations across error terms within each regression equation. SUR allows us to estimate whether any given measure of social capital was significantly associated with a change in all outcomes combined.

$$y_{it} = \beta_i X_{it} + \varepsilon_{it},\ i = 1,\ldots m,\ t = 1,\ldots T \quad \left(E\left[\varepsilon_{it}\varepsilon_{js}|X\right] = 0, t \neq s;\ E\left[\varepsilon_{it}\varepsilon_{jt}|X\right] = \sigma_{ij}\right)$$

However, it provides only a *P*-value rather than an overall effect size, and is not useful for determining whether the results were clinically meaningful. We defined clinically meaningful on a population level as a coefficient equal to a 10% change in laboratory values (based on a change in cholesterol likely to effect a detectable change on mortality) or a 5% change in mortality relative to the control group (Nissen et al., 2005; Ridker et al., 2005). All analyses were adequately powered to detect a clinically meaningful change in a coefficient. However, power estimates were based upon unweighted regressions. Because weighting can artificially increase confidence intervals around parameter estimates and have been the subject of controversy (DuMouchel & Duncan, 1983), we also ran analyses without weighting. Finally, we examined cohort effects by stratifying the analysis by age (18–40 years and ≥40 years). This cutoff was decided *a priori* as a rough division of the mid-point of our analytical age range and as a rough division of life preferences.

Finally, we conducted mediation analysis following the work of MacKinnon, Lockwood, Hoffman, West, and Sheets (2002). Mediation tests explore the extent to which a third variable explains the relationship between a dependent and independent variable. Using the MacKinnon et al. (2002) paper as a guide, we examined two approaches with high statistical power for detecting an intervening variable effect while minimizing the Type I error rate (Bobko & Rieck, 1980; Olkin & Finn, 1995). The first approach tests the extent to which the relationship between the dependent and independent variable is reduced by the mediator (Olkin & Finn, 1995). The second test examines the product the correlation between the independent variable and the mediating variable, and the partial regression coefficient relating the mediator and the dependent variable, controlling for the independent variable (Bobko & Rieck, 1980).

Results

Those in excellent or very good health, those with higher educational attainment, and those who are white are more likely to get together with friends and relatives more than 12 times per year (i.e., at least monthly, on average) (Table 1). However, whites and

those with higher educational attainment are less likely to attend church at 12 times per year than are blacks or those with lower educational attainment. No one group was more or less likely than any other group to visit neighbors more than 12 times per year. Males, whites, those in good health, and those with less education were more likely to belong to clubs than were females, blacks, those in poor health, or those with more education. While systolic and diastolic blood pressure tends to be lower among those who spend time with friends or relatives more than 12 times per year and those who attend meetings, the opposite was true of those who attend meetings more than 12 times per year.

After adjusting for age, race, gender, census region, and educational attainment, the laboratory markers of health (CRP, serum cholesterol, serum fibrinogen, systolic blood pressure, and diastolic blood pressure) varied slightly across measures of social capital (Table 2), but mortality (Table 3) rates were quite different between social capital measures. Additional outcomes from our sensitivity analyses are available as an online Supplement to this article.

Table 3
Results from Cox proportionate hazards analyses of associations between social capital measures and measures of mortality. All measures are dichotomized into <12 or ≥12 times per year except club membership, which is dichotomized into yes/no. 1978–2002 General Social Survey-National Death Index dataset with follow up through 2008. (95% confidence interval in parentheses.)

	All causes hazard ratio[a]	Cardiovascular causes hazard ratio[a]	Sample size *n*
Visit friends or relatives	0.85 (0.66–1.11)	0.88 (0.45–1.72)	12,626
Visit neighbors	1.06 (0.91–1.24)	1.35 (0.87–2.11)	12,625
Attend church	0.72* (0.60–0.86)	0.83 (0.57–1.21)	12,626
Attend meetings	0.88 (0.63–1.24)	1.04 (0.49–2.21)	3480
Belong to clubs?	0.81* (0.70–0.93)	0.71 (0.49–1.02)	12,630

*$p < 0.001$.
[a] Controls for age, gender, race/ethnicity, geographic region, educational attainment, blood pressure, cholesterol, and self-rated health.

Visiting friends and family

Visiting friends or relatives 12 or more times per year (as compared to <12 times/year) was associated with a statistically significant 1.1 mmHg decrease in diastolic blood pressure (−1.14 mmHg, 95% confidence interval [95% CI] −2.09, −0.19). When the sample was limited only to healthy participants, diastolic blood pressure was a 2 mmHg lower among those visiting friends and relatives ≥12 times per year ($P < 0.01$, see Supplementary materials), a value that still does not reach clinical significance. While no other intermediate outcome measures were statistically or clinically significant, together they reached borderline statistical significance in SUR analyses ($P = 0.06$), but not a statistically significant decrease in all cause (HR = 0.85; 95% CI = 0.66–1.11) or cardiovascular mortality (HR = 0.88; 95% CI = 0.45–1.72). Accordingly, none of the laboratory measures were found to mediate the relationship (see Table 4). This is true both before and after including weighting. When the sample was limited to healthy participants, the mortality coefficient decreased by 17% but remained non-significant. (Since removing weighting skews confidence intervals, we do not report them here. See Supplementary materials.) Including smoking produced mixed results in the intermediate outcome coefficients and a negligible increase (0.01) in the mortality coefficients (see Supplementary material). Controlling for health insurance did not impact the observed associations, nor did stratifying by age.

Visiting neighbors

The frequency with which one visits neighbors was associated with a small increase in CRP (0.03 mg/L, 95% CI: 0.00, 0.06), but was not statistically significantly associated with any other outcome measure. Moreover, laboratory values were not jointly statistically significant in SUR analyses. Interestingly, for this measure of social capital, the coefficient on all cause and cardiovascular mortality was positive, with a non-significant 10% increase for all cause mortality and 31% increase in cardiovascular mortality (HR = 1.06; 95% CI = 0.91–1.24). Accordingly, none of the laboratory measures were found to mediate the relationship (see Table 4). When weighting is removed, visiting neighbors is associated with a statistically significant 43% increase in cardiovascular mortality ($p < 0.001$, see Supplemental materials). However, when the sample was limited to healthy participants, the coefficient dropped to roughly 1.0 (HR = 0.96; 95% CI = 0.77–1.14) regardless of whether weighting was applied. It also dropped below 1.0 for those under the age of 40 (HR = 0.88; 95% CI = 0.64–1.21). Including smoking or health insurance did not alter coefficients in any consistent way.

Attending church

Attending church more than 12 times per year was negatively associated with decreased serum fibrinogen (−8.05 mg/dL, 95% CI: −14.8, −1.3) and was strongly statistically significant in joint SUR analyses, predicting improved laboratory markers of health. All cause mortality (HR = 0.72, 95% CI: 0.60, 0.86) was significantly lower, and remained statistically significant when the sample was limited to healthy participants (HR = 0.75, $p < 0.01$). Systolic blood pressure mediated this relationship in both of our tests, and C-reactive protein was a strong mediator in one test and a marginally statistically significant mediator in another test (see Table 4). Cardiovascular mortality did not reach statistical significance (HR = 0.83, 95% CI: 0.57, 1.21). Removing weighing did not influence this value. Including smoking did not alter intermediate outcome coefficients in any consistent way, however it tended to increase the coefficients for both measures of mortality. For instance, the HR for all cause mortality increased slightly to 0.78 (95% CI 0.64, 0.95) and

Table 2
Ordinary least squares regression analyses considering the association between each social capital variable and laboratory measures of health. All measures are dichotomized into <12 or ≥12 times per year except club membership, which is dichotomized into yes/no. 1978–2002 General Social Survey-National Death Index dataset with follow up through 2008. (95% confidence interval in parentheses.)

	C-reactive protein	Systolic blood pressure	Diastolic blood pressure	Serum fibrinogen	Total cholesterol	Joint value[a]
Visit friends or relatives?	−0.01 (−0.08 to 0.05)	0.25 (−1.14 to 1.63)	−1.14* (−2.09 to −0.19)	4.68 (−8.18 to 17.53)	2.05 (−1.41 to 5.5)	$p = 0.06$
Visit neighbors	0.03* (0.00–0.06)	−0.20 (−0.86 to 0.46)	−0.06 (−0.60 to 0.48)	−1.78 (−8.23 to 4.67)	−1.26 (−3.22 to 0.69)	$p = 0.56$
Attend church	−0.03 (−0.06 to 0.00)	−0.60 (−1.19 to 0.00)	−0.31 (−0.77 to 0.15)	−8.05* (−14.79 to −1.30)	−0.29 (−2.23 to 1.64)	$p = 0.00$
Attend meetings	0.023 (−0.01 to 0.06)	0.05 (−1.21 to 1.30)	−0.24 (−1.09 to 0.60)	−10.5 (−22.00 to 0.94)	0.54 (−3.27 to 4.05)	$p = 0.04$[b]
Belong to clubs	−0.03 (−0.06 to 0.01)	−0.22 (−0.85 to 0.42)	0.06 (−0.50 to 0.62)	−0.21 (−8.11 to 7.70)	−1.96 (−4.24 to 0.33)	$p = 0.01$

* $p < 0.05$ (not statistically significant after Bonferroni correction).
[a] Derived from seemingly unrelated regression analyses.
[b] While SUR does not require correction for multiple comparisons across the laboratory values, it does require correction across measures of social capital. Therefore, this test is non-significant after Bonferroni correction.

Table 4
P-values associated with two approaches (Bobko & Rieck, 1980; Olkin & Finn, 1995) to exploring the mediating effects of laboratory measures of health that might explain the association between 5 measures of structural social capital and mortality. Values less than 0.05 represent statistically significant mediation effects.

	Friends	Neighbors	Church	Clubs	Meetings
Olkin and Finn (1995)					
Systolic blood pressure	0.901	0.41	0.046[a]	0.038	0.719
Diastolic blood pressure	0.66	0.532	0.229	0.068[b]	0.585
C-reactive protein	0.95	0.568	0.00[a]	0.99	0.002[a]
Total cholesterol	0.452	0.497	0.275	0.306	0.412
Fibrinogen	0.43	0.428	0.47	0.461	0.521
Bobko and Rieck (1980)					
Systolic blood pressure	0.866	0.388	0.042[a]	0.024	0.697
Diastolic blood pressure	0.553	0.523	0.221	0.013[a]	0.527
C-reactive protein	0.99	0.368	0.09[b]	0.99	0.079[b]
Total cholesterol	0.415	0.502	0.123	0.154	0.353
Fibrinogen	0.43	0.431	0.367	0.338	0.507

[a] Statistically significant value.
[b] Borderline statistical significance.

the statistical significance declined from 0.001 to 0.05. Including health insurance in the analyses did not alter the coefficients in any meaningful or consistent way nor did stratifying by age.

Belonging to clubs

Interestingly, belonging to clubs was not significantly associated with any intermediate outcome measure, but did show joint statistical significance under SUR ($P = 0.01$) and was associated with a reduction in all-cause mortality (HR = 0.78, 95% CI: 0.64, 0.95). Systolic blood pressure mediated this relationship in both of our tests, and diastolic blood pressure was a strong mediator in one test and a marginally statistically significant mediator in another test (see Table 4). When mortality analyses were restricted to cardiovascular mortality, none of the measures were statistically significant. This was not influenced by removing weighting or by restricting the sample to healthy participants. Including smoking or health insurance did not alter coefficients in any consistent way, nor did stratifying by age.

Attending meetings

Attending meetings did not produce any statistically significant changes in intermediate outcomes or in either measure of mortality. However, it did reach joint statistical significance for reductions in intermediate outcome measures ($P = 0.04$, not significant after Bonferroni correction). These values were not influenced by excluding weighting or by limiting the sample to healthy participants. Including smoking or health insurance did not alter coefficients in any consistent way, nor did stratifying by age.

Discussion

The five measures of social capital contained within the NHANES III–NDI were not consistently predictive of intermediate measures of health or mortality. Specifically, while some measures showed reasonably strong predictive value for mortality hazards, visiting one's friends or family, visiting one's neighbors, or attending meetings more than 12 times per year were not associated with either the intermediate measures (elevated cholesterol, blood pressure, serum fibrinogen, and CRP) or the distal health outcomes (cardiovascular or all-cause mortality).

Unlike spending time with neighbors, being involved with clubs or attending church more than 12 times per year was broadly protective against premature mortality, a finding consistent with the literature (Kawachi et al., 2010). In our analysis, serum fibrinogen was statistically and clinically significantly lower among those attending church more than 12 times per year before correcting for multiple comparisons. While none of the remaining intermediate measures (elevated cholesterol, blood pressure, and CRP) were either statistically or clinically significant, together they were strongly statistically significant. This suggests that attending church or clubs produces small improvements in laboratory measures that add up to large changes in mortality. While attending meetings more than 12 times per year was also statistically significant prior to Bonferroni correction, given the low *P* value, it is unlikely that the joint effect size was very large.

From our sensitivity analyses, restricting the sample only to healthy participants generally improved laboratory measures and in some cases reduced the mortality hazards. Smoking did not seem to explain the variation in health outcomes for any measure of social capital on health either, suggesting that social capital works through other mechanisms. While smoking rates are declining over time and that smoking behaviors spread through social networks (Christakis & Fowler, 2008), the intensity of contact with one's social network may not be important for smoking behaviors. Additional sensitivity analyses controlling for marital status and income (data available upon request) also did not affect the associations observed between measures of structural social capital and health.

When the analysis is unweighted, visiting one's neighbors is associated with a statistically significant 43% increase in mortality. Including weighting is controversial as it artificially increases type II error and may produce misleading results, but excluding it also impacts the representativeness of the sample (DuMouchel & Duncan, 1983). However, this effect disappears entirely when the sample is restricted to healthy participants only. This suggests that illness confounds the association; it is possible that less healthy people rely on their neighbors more for daily tasks and therefore visit them often. The finding that those under the age of 40 also have an HR that is less than 1.0 suggests that either cohort or survivor effects (Flegal, Graubard, & Williamson, 2004) are relevant for this measure of social capital. However, in a separate unpublished analysis using the General Social Survey linked to the National Death Index (Muennig, Johnson, Kim, Smith, & Rosen, 2011), we find that frequently visiting neighbors is associated with a statistically significant 16% higher morality hazards (Nau & Muennig, 2012). That analysis is not sensitive to stratification by health status or age. In that analysis, however, we find that spending time with neighbors is protective for married couples and harmful for single couples. We speculate that spending time with neighbors competes with other, potentially more meaningful forms of social capital for some, and for others it may increase exposure to behavioral risks; people may smoke or drink with their neighbors. They may also not fully benefit from stress buffers, as the relationships might not be as meaningful as deeper, more supportive relationships in which the participant defines the other as a friend.

One surprise was that visiting friends and family more than 12 times per year or belonging to clubs did not reach statistical significance in this adequately powered study. There is a large body of literature suggesting that social ties do reduce mortality, and most of this literature is based on studies with a smaller sample size (Berkman & Syme, 1979; Schoenbach, Kaplan, Fredman, & Kleinbaum, 1986). However, to our knowledge, this literature does not include large nationally-representative surveys with medical examination data. For example, one large and rigorous randomized trial found no improvement in cardiovascular mortality associated with an intervention to reduce depression and improve social support (Berkman, Blumenthal, Burg, Carney, Catellier, Cowan, Schneiderman, et al.,

2003). It is also worth noting that our joint laboratory (which reached borderline significance) and mortality analyses were insensitive to changes in model assumptions.

We contribute to the literature struggling to disentangle the mechanisms through which social capital may (or may not) affect health, while acknowledging that our findings are correlational and not necessarily causal. In our study, we hypothesize that psychological stress may elevate the laboratory tests we examined. Serum fibrinogen was the one laboratory measure that reached statistically significantly lower levels among those attending church, and this is one measure of the independent effect of psychological stress on physiological processes (Davey Smith et al., 2004; Markowe et al., 1985; Muennig et al., 2007; Steptoe et al., 2003). On the other hand, both cholesterol and CRP are lowered by commonly used cholesterol-lowering medications (Ridker, Rifai, Pfeffer, Sacks, & Braunwald, 1999), and blood pressure can also be controlled with medications. This raises the possibility that medical care played a complementary role in the joint significance value. While adding health insurance coverage to the model did not alter our results in meaningful ways, it is possible that insured participants with higher social capital have access to higher quality medical care (a factor that we could not measure).

Others have previously found that bonding versus bridging have different associations with health outcomes (Beaudoin, 2009; Hanibuchi et al., 2012; Iwase et al., 2010). Using examination and mortality outcomes, we find that the relationship between different types of bonding social capital and health also differ. Debates remain regarding ways to measure social capital; researchers use both cognitive dimension (e.g., level of agreement with 'people in this neighborhood can be trusted') and structural dimension such (e.g., membership in clubs) (Kawachi & Berkman, 2003; Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997; Lochner et al., 2003). Different types of social capital may be interrelated. For example, multiple, strong friendships provide socioemotional support, decrease feelings of stress and animosity, and provide social connections that may benefit them in finding a job or seeking medical care (Link & Phelan, 1995). These friendships may be fostered through secular organizations, attendance at church functions and meetings, and participation in neighborhood activities (Wilkinson, 1999). Finally, it is possible that the cumulative advantage of different forms of social capital can have larger impacts on health (Willson, Shuey, & Elder Jr, 2007).

We note that much of the research literature has been associational, but there are increasing numbers of studies considering the causal impact of social capital on health outcomes around the world. Among older adults, good health and high social capital may affect each other in a positive feedback loop such that health and social capital inequalities are exacerbated (Sirven & Debrand, 2012), and an instrumental variables analysis concluded that people with higher social capital are healthier (Ronconi, Brown, & Scheffler, 2012), a trend also observed in instrumental variable analyses of populations across the age spectrum (d'Hombres, Rocco, Suhrcke, Haerpfer, & McKee, 2011).

Limitations

This is a prospective cohort study. As such, it is subject to confounding by unobserved variables. For example, others have discussed the potential role of smoking and drinking behaviors regarding the relationship between social capital and health outcomes (Ahern, Galea, Hubbard, Midanik, & Syme, 2008; Giordano, Björk, & Lindström, 2012; Lê, Ahern, & Galea, 2010). While we were able to include smoking in our models, most data about alcohol consumption were missing, precluding us from including it in our analyses. Given that there have been few experimental analyses of social capital, it is also possible that the observed associations between social capital and health are not, in fact, causal. For instance, a positive outlook is associated with improved health outcomes (Yan et al., 2003). Those with a positive outlook on life may also be more likely to form close friendships, participate in politics, or belong to organizations that effect change. Likewise, sick people are probably less likely to socialize, participate in civil society, and so forth. Our sensitivity analysis of only those with high self-rated health attempted to address potential issues of reverse causality. We found no evidence of reverse causality in this sample. In the case of those visiting neighbors more than 12 times per year, we do observe evidence of confounding in the opposite direction (i.e., those who visit neighbors frequently may be less healthy than those who do not). Also of note is that this study did not examine the effects of cognitive social capital.

In addition, while the sample size of NHANES-III is very large and mortality follow-up is quite long, we still did not have the statistical power to conduct mortality analyses for less common causes of death. We encourage future researchers to examine whether different specific causes of death may be attributable to different types of social capital. Finally, while SUR is a powerful tool for examining the joint significance of multiple covariates of interest, it does not provide an interpretable coefficient. Therefore, it is difficult to estimate the magnitude of the effect of overall improvements or decrements in laboratory tests.

Finally, while the NHANES-III has been extensively tested for non-response bias and other forms of exogenous and endogenous selection, measures of structural social capital produce unique selection issues. Those who are more social may both be more likely to attend meetings and to participate in research.

Summary and implications for research and policy

We find that not all forms of social capital are predictive of health or mortality, and that those measures of social capital that do matter for mortality exert relatively little influence on laboratory measures of health. However, belonging to clubs or attending church more than 12 times per year does produce small improvements in multiple intermediate measures of health, and that, collectively, these add up to large reductions in mortality hazards. This latter finding potentially explains how social phenomena might produce weak effects on objective measures of health but larger impacts on mortality. Given publication bias, it is difficult to know the extent to which previous studies have failed to find associations between measures of social capital and self-rated health independent of mortality, however. We highlight the importance of multiple measures of single constructs as a measurement strategy, and seemingly unrelated regression as a useful analytic tool.

We encourage contextualizing social capital especially when considering the long hours that people work and the time that they spend in front of devices, such as televisions, computers, tablets, and phones. The amount of time spent in front of such devices is plausibly related to one's ability to attend to social affairs, and has itself been linked to mortality (Dunstan et al., 2011; Parsons, Manor, & Power, 2008; Uslaner, 1998; Wijndaele et al., 2011). With this in mind, we also note that all of the measures of social capital we employed rely on face-to-face interaction; future researchers could consider if social capital developed in-person versus online may have differential health implications.

Social capital analyses also have important policy implications. For example, as public health practitioners take increasingly upstream approaches to health promotion, one approach that has taken hold is to partner public health researchers with urban planners to promote health (Coburn, 2009). Cities are now developing metrics to track progress toward improved health outcomes (Corburn & Cohen, 2012). With this in mind, it becomes critically

important to understand which forms of social capital matter for health, for whom they matter, and in which context they matter, so that they can be included as indicators of a healthy city. For example, improvements to sidewalks, increasing storefront space, and increasing the numbers of parks have each been shown to improve contacts with one's neighbors. Some cities have undertaken these initiatives under the assumption that such enhancements are good for social capital and for everyone's health (Coburn, 2009). While it is quite possible that this is the case for reasons other than social capital, our findings suggest that this assumption requires more nuance. With good health data, policymakers will be better able to evaluate the health implications of trends toward building dense, urban cores with active living designs and public transit—designs that force a more active, energy efficient lifestyle, but also force large numbers of people into close proximity to one another. By better understanding the influence of social capital on health, we can better understand how to better optimize urban design.

Appendix. Supplementary material

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.socscimed.2013.02.007.

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