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ABSTRACT

Despite the vast amount of research over the past fifteen years, there is still lively debate surrounding the role of social capital on individual health outcomes. This seems to stem from a lack of consistency regarding the definition, measurement and plausible theories linking this contextual phenomenon to health. We have further identified a knowledge gap within this field – a distinct lack of research investigating temporal relationships between social capital and health outcomes. To remedy this shortfall, we use four waves of the British Household Panel Survey to follow the same individuals ($N = 8114$) between years 2000 and 2007. We investigate temporal relationships and association between our outcome variable self-rated health (SRH) and *time-lagged* explanatory variables, including three individual-level social capital proxies and other well-known health determinants. Our results suggest that levels of the social capital proxy 'generalised trust' at time point ($t - 1$) are positively associated with SRH at subsequent time point (t), even after taking into consideration levels of other well-known health determinants (such as smoking status) at time point ($t - 1$). That we investigate temporal relationships at four separate occasions over the seven-year period lends considerable weight to our results and the argument that generalised trust is an independent predictor of individual health. However, lack of consensus across a variety of disciplines as to what generalised trust is believed to measure creates ambiguity when attempting to identify possible pathways from higher trust to better health.

Introduction

Since Durkheim's seminal work over a century ago (Durkheim, 1897, 1951), research has repeatedly shown that individuals with higher levels of social integration, social networks and social support have better health (for examples see: Berkman & Syme, 1979; House, Landis, & Umberson, 1988; Lasker, Egolf, & Wolf, 1994; Pennix et al., 1997). However, following the introduction of 'social capital' to the field of public health (Kawachi, Kennedy, & Glass, 1999; Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997), the debate has continued regarding this contextual phenomenon and how it also independently influences health outcomes (Hawe & Shiell, 2000; Pearce & Davey Smith, 2003; Szreter & Woolcock, 2004).

From social capital literature and research, we have identified three main areas of contention: how one defines (and conceptualizes) social capital, how one measures social capital, and how social capital is theorized to influence health. These issues seem inter-connected, as one's definition of social capital will surely influence how one measures and theorizes the effects of social capital on individual health outcomes.

There is no single accepted definition of social capital, so it is not surprising that there are differences in opinion regarding its conceptualization. Of the contemporary authors in this field, Robert Putnam (1993, p.167), defines social capital as '... features of social organization, such as trust, norms, and networks that can improve the efficiency of society by facilitating coordinated actions.' With his definition, Putnam places social capital firmly at the societal-level. However, Pierre Bourdieu also conceptualized social capital at the individual-level by defining it as '...the sum of the resources, actual or virtual, that accrue to an individual or a group by virtue of possessing a durable network' (Bourdieu & Wacquant, 1992, p.119).

Another key social capital theorist, James Coleman (1990, p.302) defines social capital as '...a variety of different entities [that] facilitate certain actions of individuals who are within the

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structure', the family playing a vital role in his theories; and finally Portes (1998 p.6) defines social capital as '… the ability of actors to secure benefits by virtue of membership in social networks or other social structures', stressing the individual-level properties of this phenomenon.

The differences above clearly highlight the lack of consensus between theorists regarding the conceptualisation and ownership of social capital, which leads to our second area of contention: how one measures social capital and its effects on health, at the individual or the collective level (Macinko & Starfield, 2001). This problem is further confounded by the fact that social capital is often considered a contextual phenomenon (Berkman & Kawachi, 2000) that cannot be directly observed or easily quantified. As a result (and irrespective of 'conceptualisation' issues) the vast majority of social capital research relies on individual-level 'proxy' measures. Researchers' choice of proxy often reflects the social capital definition being tested; for example, if investigating Putnam's or Coleman's social capital theory, one would use proxies such as generalised (horizontal) and vertical (institutional) trust, and social and civic participation (for examples see: Coleman, 1988; Hyyppä & Mäki, 2001; Lindström, 2004; Putnam, 1993, 2000; Subramanian, Kim, & Kawachi, 2002; Veenstra, 2000).

Researchers wanting to investigate 'group' effects of social capital on individual health may further aggregate individual-level proxies to a context of interest. However, the process of aggregation is not without its own issues, as any assumption made about an individual based solely on a group mean value may be inherently biased (commonly known as 'ecological fallacy'). Furthermore, most multilevel social capital studies have demonstrated that only 0−4% of *total* variation in individual health is attributable to commonly used aggregates, such as community, state or county contexts (for examples see: Fujisawa, Hamano, & Takegawa, 2009; Lindström, Moghaddassi, & Merlo, 2004; Poortinga, 2006; Snelgrove, Pikhart, & Stafford, 2009).

Conversely, social capital studies maintaining analyses at the individual-level still may face criticism if they do not consider any potential contextual effects. This criticism may be unfounded, however, as one recent multilevel study (simultaneously investigating individual-, household- and community-level contexts) demonstrates that that it is *individual-level* social capital proxies that influence individual health the greatest (Giordano, Olhsson, & Lindström, 2011).

The third area of contention (how social capital influences health) has generated lively debate over recent years, as the relevance of social capital on health outcomes has often been contested by proponents stressing the importance of access to material resources and public welfare policy (Muntaner, 2004; Pearce & Davey Smith, 2003). Furthermore, certain social capital proxies (i.e. 'social networks' or 'social participation') could easily be perceived as potential sources of social support, a well-known determinant of individual health (Berkman & Syme, 1979). To address these arguments comprehensively within empirical social capital research, one must consider differing measures of socio-economic status (SES) such as income, education and employment status, along with well-known measures of social support whenever possible. As there is also an increasing literature base suggesting a distinct lack of correlation between social capital proxies, in turn hinting at several pathways from social capital to health (Giordano & Lindström, 2010; Lindström, 2004; Nummela, Sulander, Rahkonen, Karisto, & Uutela, 2008; Stolle, 2001), it also seems prudent to simultaneously test multiple social capital proxies, if data allow.

We have further identified an apparent shortfall in global social capital research, one that specifically addresses the issue of causality. To clarify: there are nine criteria required to help establish a causal

relationship between exposure and disease, including strength, plausibility, and consistency. However, temporal relationship is considered the only 'essential criterion'; i.e. if exposure A is theorized to cause disease B, then A must always precede B (Goodman & Phillips, 2005). In other words, longitudinal data are needed to test causality. This notion is also supported by methodological considerations concerning causal mechanisms within the social sciences (Hedström & Ylikoski, 2010). However, during the near exponential rise in papers researching social capital and health over the past fifteen years, the vast majority of studies has been cross-sectional in design (Islam, Merlo, Kawachi, Lindström, & Gerdtham, 2006) and are therefore unable to test temporal relationships. Of the longitudinal studies within the field, a PUBMED search reveals that only one study, investigating association between 'psychological wellbeing' and social capital (Giordano & Lindström, 2011), incorporates the three (or more) time points required to correctly test temporal relationships (Singer & Willet, 2003 p.9). Our study will attempt to address this knowledge gap in social capital research by using the same individuals' responses taken at four different time points between the years 2000 and 2007.

The aim of this longitudinal study is to investigate temporal relationships between self-rated health (SRH) and *lagged* measures of *individual-level* social capital, social support and SES. As our longitudinal data cannot be aggregated, we intend to employ *individual* measures of generalised trust, social participation and contact with neighbours as social capital proxies in our study; the choice of proxy being determined in part by data availability and also our acceptance of Putnam's social capital definitions. We hypothesize that levels of social capital at time $(t-1)$ are positively associated with SRH status at subsequent time point (t), even after adjusting for other well-known health determinants at time $(t-1)$.

Materials and methods

Data collection

The British Household Panel Survey (BHPS) is a longitudinal survey of randomly selected private households conducted by the UK's Economic and Social Research Centre. Since 1991, individuals within selected households have been interviewed annually with a view to identifying social and economic changes within the British population. The original 1991 cohort sample was randomly selected by using a two stage cluster design, resulting in a total of 8166 private postal addresses around the UK. From these addresses 10,264 individual interviews were completed in 1991, demonstrating a participation rate of 95%. Full details of the selection process, weighting and future participation rates can be found online in the BHPS user manual (Taylor, Brice, Buck, & Prentice-Lane, 2010).

The raw data that have been used for this panel study come from the BHPS individual-level responses in years 2000, 2003, 2005 and 2007. The same individuals ($N = 8114$) were followed across this seven-year time frame; participation rate for year 2000 (as compared to year 1999) was 93.6%, and, compared to the original 1991 cohort, was 62.0%.

The Research Centre fully adopted the Ethical Guidelines of the Social Research Association; informed consent was obtained from all participants and strict confidentiality protocols were adhered to throughout data collection and processing procedures.

Dependent variable

The dependent variable in this study is self-rated health (SRH), considered a valid predictor of morbidity and future mortality (Idler & Benyamini, 1997; Lopez, 2004). The same individuals were asked:

'Compared to people your own age, would you say that your health has on the whole been: excellent, good, fair, poor or very poor?' As is standard with the global SRH item, this five-point scale was recoded into the dichotomous variable 'good' (excellent, good) and 'poor' (fair, poor, very poor) health.

Explanatory variables

Social capital variables

Generalised (horizontal) trust was assessed by asking people: 'Would you say that most people can be trusted, or that you can't be too careful?' This variable was dichotomised, with only those respondents stating that most people could be trusted being labelled 'can trust'; all negative responses (including 'it depends') were labelled 'can't trust' (Uslaner, 2002).

Social participation was measured by asking respondents questions about being active members of listed voluntary community groups or any sports, hobby or leisure group activity found locally (see Appendix A for full list). Only those who answered positively to any of these were judged to participate, with all others being labelled 'No participation'.

We also considered frequency of talking to neighbours as a proxy for social capital (Putnam, 2000, p.105–106). Possible responses were: 'Most days, once or twice a week, once or twice a month, less than once a month, or never'. Those answering 'most days' or 'once or twice a week' were assigned the label 'One or more times per week'; the rest were assigned the label 'less often'.

Socio-economic status variables

Social class was determined by the respondents' most recent occupation, derived from the Registrar General's Social Classification of occupations. The usual six categories (see Appendix A) were dichotomised into 'higher' (1–3a) and 'lower' (3b–6) social class. Those who had never been employed were labelled 'never worked'.

Highest achieved education level was categorised as 'Undergraduate or higher', 'Year 13' and 'Year 11' or 'No formal qualifications'.

'Household income' was weighted according to size by summing the income of all household members and dividing this sum by the square root of the household size (Burkhauser, Smeeding, & Merz, 1996). This item was maintained as a continuous variable (per £1000 increase) and was an expression of total income, net of any taxation.

Social support variables

Respondents were asked if they were 'married, separated, divorced, widowed or never married'. These five options were recoded into the dichotomous variable 'married' and 'not married' (separated, divorced, widowed or never married). A further variable 'Lives alone' ('yes' or 'no') was also used to try to capture more information about those individuals who co-habited.

We also considered frequency of meeting with friends or family as a proxy for social support. Possible responses were: 'Most days, once or twice a week, once or twice a month, less than once a month, or never'. Those answering 'most days' or 'once or twice a week' were assigned the label 'one or more times per week'; the rest were assigned the label 'less often'.

Confounders

Age, gender, smoking status and time were considered confounders in this study, age being stratified into quintiles (see Tables 1 and 2). Time (corresponding to the waves of interviews in years 2000, 2003, 2005 and 2007) was also included as a continuous covariate to adjust for potential trends in SRH and explanatory variables across time. Smoking status was categorised as 'smoker'

and 'non-smoker' according to respondents' answer to the question 'Do you smoke cigarettes?'

All explanatory variables (except gender) were lagged at time $(t - 1)$ in reference to SRH at time (t). It was presumed that the presence of social capital, being younger, being married or cohabiting, being a non-smoker, attaining higher education and household income, having greater social support, and being of higher social class at time $(t - 1)$ were associated with good SRH at time (t).

Statistical analyses

All data were stratified by baseline (year 2000) SRH to create two distinct 'health' cohorts: 'Good health at baseline' (GHB) and 'Poor health at baseline' (PHB). After this initial disaggregation, the two 'health' cohorts were modelled as separate entities: Model 1 dealt solely with individuals from the GHB cohort ($N = 5689$); the outcome of interest in Model 1 was *change* from 'GHB' (0) to 'poor' SRH (1) from year 2000–2007. Model 2 dealt solely with individuals from the PHB cohort ($N = 2425$); the outcome of interest in Model 2 was *change* from 'PHB' (0) to 'good' SRH (1) from year 2000–2007.

In order to investigate temporal relationships between exposure and outcome, *all explanatory variables* (except gender) were lagged at time $(t - 1)$ in reference to SRH at time (t). To clarify, when SRH in 2003 was the outcome, only explanatory variables from year 2000 were utilised; when SRH in 2005 was the outcome, only explanatory variables from 2003 were utilised; and when SRH in 2007 was the outcome, only explanatory variables from 2005 were utilised.

Disaggregation by baseline SRH meant we could attribute any association found between our lagged explanatory variables to *change from baseline health status*. Without disaggregation, we could only describe association between lagged explanatory variables and SRH as trends across the time frame of our study.

Our hypothesis, as stated in the introduction, is that social capital at $(t - 1)$ is positively associated with SRH at time (t); however, other temporal pathways may co-exist, confound each other, or even interact with each other. For example: SRH at time (t) could theoretically influence one's ability to maintain social networks at time (t) and/or at $(t+1)$. To address this concern, we also performed sensitivity testing. We ran *all* explanatory variables from time (t), alongside *all* lagged $(t - 1)$ exploratory variables, the outcome being SRH at time (t). If association still held at time $(t - 1)$ when we considered social capital (and all other explanatory variables) at time $(t - 1)$ *and* time (t) simultaneously, this would strengthen our hypothesis that prior levels in social capital are positively associated with subsequent SRH.

For all analyses we used logistic regression models with random effects, as SRH was expected to be more similar *within* the same individual over time than *between* different individuals. The model allowed a random intercept for each individual and we obtained standard errors that were adjusted for the temporal correlation of SRH *within* the same individual across the time frame of our study. The equations for logistic regression models with random effects are as follows:

$$\text{Log it}(Y_{ij}) = \beta_{0j} + \beta \mathbf{X}_{i-1j}$$

$$\beta_{0j} = \beta_0 + \mu_{0j}$$

where i = time, j = individual, Y_{ij} is the outcome variable, μ_{0j} = the random intercepts (assumed to be independently normally distributed with a common variance), $\mathbf{X}_{i-1j}$ is a vector of lagged explanatory variables, β_0 is the fixed overall intercept, and β the corresponding vector of coefficients.

All explanatory variables were utilised in our two multiple logistic regression models. Model 1 investigated *change* from GHB

Table 1a
Frequencies of all considered explanatory variables expressed as integers and percentages (%) of N_T (8114) stratified by baseline self-rated health (SRH) in year 2000.

Explanatory variables		Baseline self-rated health		
		Good SRH ($N = 5689$)	Poor SRH ($N = 2425$)	Total ($N_T = 8114$)
Age (years)	16–34	1817	600	2417
		32%	25%	30%
	35–44	1311	426	1737
		23%	18%	21%
	45–54	1060	472	1532
		19%	20%	19%
	55–64	740	432	1172
		13%	18%	14%
	65+	761	495	1256
		13%	20%	16%
Total		5689	2425	8114
		100%	100%	100%
Gender	Male	2600	1003	3603
		46%	41%	44%
	Female	3089	1422	4511
		54%	59%	56%
Total		5689	2425	8114
		100%	100%	100%
Generalised trust	Yes, can trust others	2396	726	3125
		42%	30%	39%
	No, can't trust others	3293	1696	4989
		58%	70%	62%
Total		5689	2425	8114
		100%	100%	100%
Social Participation: Local groups, organisations or group leisure activities	Active participation	2469	842	3311
		43%	35%	41%
	Zero participation	3220	1583	4803
		57%	65%	59%
Total		5689	2425	8114
		100%	100%	100%
Frequency of talking with neighbours	One or more times/week	4410	1876	6286
		78%	77%	78%
	Not that often	1279	549	1828
		23%	23%	23%
Total		5689	2425	8114
		100%	100%	100%
Frequency of meeting with friends	One or more times/week	4872	2081	6953
		86%	86%	86%
	Not that often	817	344	1161
		14%	14%	14%
Total		5689	2425	8114
		100%	100%	100%
Marital status	Married	3370	1411	4781
		59%	58%	59%
	Not married	2319	1014	3333
		41%	42%	41%
Total		5689	2425	8114
		100%	100%	100%
Lives alone	Yes	686	398	1084
		12%	16%	13%
	No	5003	2027	7030
		88%	84%	87%
Total		5689	2425	8114
		100%	100%	100%
Education achieved[a]	Undergraduate or higher	2253	895	3148
		40%	37%	39%
	Year 13	1654	765	2419
		29%	32%	30%
	Year 11	1008	461	1469
		18%	19%	18%
	No qualifications	734	287	1021
		13%	12%	13%
Total		5649	2408	8057
		100%	100%	100%
Social class	High	3339	1166	4505
		61%	51%	58%
	Low	2105	1111	3216
		39%	49%	42%
	Never worked	245	148	393
		5%	6%	5%
Total		5689	2425	8114
		100%	100%	100%

Table 1a (*continued*)

Explanatory variables		Baseline self-rated health		
		Good SRH ($N = 5689$)	Poor SRH ($N = 2425$)	Total ($N_T = 8114$)
Smoking status	Smoker	1307	787	2094
		23	33%	26%
	Non-smoker	4382	1638	6020
		77%	68%	74%
Total		5691	2425	8114
		100%	100%	100%
Household income (annual) − size weighted	<£9588	1175	855	2030
		21%	35%	25%
	£9589−£15 055	1367	661	2028
		24%	27%	25%
	£15,056−£22,493	1515	510	2025
		27%	21%	25%
	£22,494+	1632	399	2031
		29%	17%	25%
Total		5689	2425	8114
		100%	100%	100%

Source: The British Household Panel Survey Wave J, 2000.
 [a] Missing $N = 57$.

Table 1b
Transitions of self-rated health status between 2000 and any of the years 2003, 2005 or 2007, with percentages calculated in relation to good health at baseline (N = 5689) and poor health at baseline (N = 2425).

Good health at baseline (year 2000)	Remains in good health	4898	86%
	Develops poor health	791	14%
Total		5689	100%
Poor health at baseline (year 2000)	Remains in poor health	1678	69%
	Develops good health	747	31%
Total		2425	100%

Source: The British Household Panel Survey Wave J, M, O and Q (2000−2007).

(0) to 'poor' SRH (1) between 2000 and any of the years 2003, 2005 or 2007; Model 2 investigated *change* from PHB (0) to 'good' SRH (1) between 2000 and any of the years 2003, 2005 or 2007. All analyses were conducted using GLLAMM version 2.3.15 (Rabe-Hesketh, Skrondal, & Pickles, 2005), within the statistical software package STATA 11.2 (StataCorp, 2009).

Results

Table 1a is descriptive, showing frequencies and total percentages of all considered explanatory variables, stratified by self-rated health in year 2000 (baseline). Table 1b is also descriptive, showing the transition of self-rated health over time in each baseline cohort.

The results of multiple logistic regression analyses containing all considered explanatory variables are presented in Table 2 as odds ratios (ORs) with 95% confidence intervals (CI). The results of our sensitivity tests are presented in Table 3, also as ORs with 95% CI.

Model 1: multiple regression analysis − 'GHB' cohort

The outcome of interest in Model 1 was *change* from 'Good Health at Baseline' (0) to 'poor' SRH (1) between 2000 and 2007. As shown in Table 2 (left hand column), of the social capital variables, low levels of trust and talking less with neighbours preceded a change from GHB to poor SRH over time (OR = 1.35 and 1.18, respectively).

Of the SES variables, those with low social class or those who had never worked at time ($t − 1$) had increased risk of poor SRH at time (t) (OR = 1.40 and 1.53, respectively). A prior increase in household income seemed to offer some protection against future poor SRH; though significant, the value was close to the reference value of 1.0.

None of the social support variables at ($t − 1$) maintained association with poor SRH at time (t). Of the confounders, smoking at ($t − 1$) and being of older age were associated with poor SRH at time (t).

Model 2: multiple regression analysis − 'PHB' cohort

The outcome of interest in Model 2 was *change* from 'Poor Health at Baseline' (0) to 'good' (1) SRH between 2000 and 2007. As shown in Table 2 (right hand column), of the social capital variables, high levels of trust and participation and talking more often with neighbours at time ($t − 1$) preceded good SRH at time (t) (OR = 1.31, 1.19, and 1.33 respectively).

Of the SES variables, those with higher social class at time ($t − 1$) had good SRH at time (t) (OR = 1.24). Those who had never worked at time ($t − 1$) were likely to remain of poor SRH (OR = 0.61). A prior increase in household income at time ($t − 1$) was associated with good SRH at time (t); though significant, the value was close to the reference value of 1.0.

Of the confounders, being a non-smoker, being male, and being of younger age at time ($t − 1$) were associated with good SRH at time (t).

Sensitivity testing

We tested our hypothesis that social capital at time ($t − 1$) is positively associated with SRH at time (t) by running *all* explanatory variables at time (t) alongside *all* lagged ($t − 1$) exploratory variables simultaneously against SRH at time (t). Sensitivity tests were performed for Models 1 and 2 separately. As shown in Table 3 (left hand column) from the GHB cohort, the only lagged ($t − 1$) social capital variable that maintains association with poor SRH at time (t) is lack of trust (OR = 1.25).

In the right hand column of Table 3 (from the PHB cohort), association remains between good SRH at time (t) and the lagged ($t − 1$) social capital variables 'trust' (OR = 1.25) and 'talks with neighbours' (OR = 1.28). Association between active social participation at ($t − 1$) and good SRH at time (t) was attenuated after adjusting for participation at time (t).

Table 2
Odds ratios (ORs) with 95% confidence intervals (95% CI) of having 'good' or 'poor' self-rated health at time (t) from baseline (Year 2000) health status according to multiple logistic regression analysis of lagged ($t − 1$) explanatory variables in social capital, social support, socio-economic status and confounders (N_T = 8113).

Explanatory variables		Change in self-rated health status from baseline (2000)	
		Model 1 (N = 5688)[a] Good health at baseline cohort — ORs (95% CI) of having PH over time	Model 2 (N = 2425) Poor health at baseline cohort — ORs (95% CI) of having GH over time
Social capital variables			
Generalised trust	Trusts others	1.0[b]	1.31 (1.10–1.56)**
	Can't trust others	1.35 (1.19–1.53)***	1.0[b]
Social participation: Active in local groups	Active participation	1.0[b]	1.19 (1.01–1.39)*
	No participation	1.05 (0.93–1.18)	1.0[b]
Frequency of talking with neighbours	1+ times/wk	1.0[b]	1.33 (1.10–1.61)**
	Less than this	1.18 (1.02–1.36)*	1.0[b]
Social support variables			
Marital status	Married	1.0[b]	1.20 (0.94–1.52)
	Not married	1.00 (0.85–1.19)	1.0[b]
Living alone	Lives with others	1.0[b]	0.87 (0.65–1.16)
	Lives alone	1.19 (0.96–1.48)	1.0[b]
Frequency of meeting with friends or family	1+ times/wk	1.0[b]	1.12 (0.90–1.40)
	Less than this	0.91 (0.77–1.08)	1.0[b]
Socio-economic variables			
Household income/£1000	Continuous	0.99 (0.98–0.99)***	1.02 (1.01–1.02)***
Social class: derived from occupation-based RGSC schema	High SC	1.0[b]	1.24 (1.02–1.52)*
	Low SC	1.40 (1.22–1.61)***	1.0[b]
	Never worked	1.53 (1.13–2.07)**	0.61 (0.40–0.92)*
Education achieved	Undergraduate +	1.0[b]	0.86 (0.66–1.12)
	Year 13	1.06 (0.88–1.26)	0.73 (0.56–1.01)
	Year 11	1.01 (0.86–1.19)	0.79 (0.60–1.06)
	No qualifications	1.23 (0.84–1.79)	1.0[b]
Confounders			
Age (years)	16–34	1.0[b]	7.19 (4.90–10.55)***
	35–44	1.12 (0.88–1.42)	2.45 (1.78–3.37)***
	45–54	1.25 (0.98–1.60)	1.27 (0.92–1.75)
	55–64	1.30 (0.99–1.68)	1.08 (0.80–1.44)
	65+	1.84 (1.43–2.37)***	1.0[b]
Gender	Male	1.0[b]	1.37 (1.11–1.69)**
	Female	1.04 (0.91–1.23)	1.0[b]
Smoking status	Non-smoker	1.0[b]	1.94 (1.55–2.37)***
	Smoker	1.68 (1.44–1.97)***	1.0[b]
Time (continuous)		1.17 (1.10–1.24)***	1.01 (0.94–1.10)

Source: The British Household Panel Survey, Waves J, M, O & Q (2000–2007).
*0.05 significance.
**0.01 significance.
***0.001 significance.
[a] Missing = 1.
[b] Reference group.

Discussion

The aim of this longitudinal study is to research temporal relationships and association between lagged health determinants (including three social capital proxies) at time point ($t − 1$) and our dependent variable self-rated health (SRH) at subsequent time point (t). The dual 'health' cohort design of the study (see 'Materials and methods' section) further allows causal inference to be made from association between lagged explanatory variables and *changes* from baseline health status over time. All three measures of social capital maintain their positive association with SRH in multiple regression models when tested simultaneously alongside other well-known health determinants. Temporal relationships and association imply that *prior* levels in either of these social capital measures seem to independently predict future SRH status, i.e. social capital at time point ($t − 1$) is positively associated with health status at subsequent time point (t), even after taking into consideration levels of other well-known health determinants at time point ($t − 1$).

Of our social capital proxies, frequency of talking with neighbours maintains association with SRH in both models (see Table 2).

Though relationships with one's neighbours may be considered a form of 'bonding' social capital (Szreter & Woolcock, 2004), it is also feasible that this source of social capital could become a source of social support, particularly if neighbours become good friends over time (Coleman, 1990, p.178–180). Social participation is only associated with good SRH in Model 2 (see Table 2, right hand column). This result implies that active participation *precedes* good SRH over time. However, our sensitivity tests (see Table 3) hint that it is most likely that good SRH at time (t) influences active participation at time (t). Generalised trust is positively associated with SRH in both Models (see Table 2), i.e. *lack* of trust at time ($t − 1$) precedes poor SRH at time (t) in Model one; conversely, an *ability* to trust at time ($t − 1$) precedes good SRH at time (t) in Model two. This positive association remains after performing our sensitivity test (see Table 3). According to our results, the positive effect of generalized trust at time ($t − 1$) on SRH at time (t) is one third the strength of not smoking at time ($t − 1$).

It is important to appreciate that generalised trust, along with most other explanatory variables in this study, is time-dependent, i.e. one's trust levels may have a different value at any given time.

Table 3
Causal pathway sensitivity testing: odds ratios (ORs) with 95% confidence intervals (95% CI) of having 'good' or 'poor' self-rated health at time (t) according to multiple logistic regression analysis of lagged ($t − 1$) and non-lagged (t) social capital variables, along with all lagged and non-lagged explanatory variables (Note: only social capital variables shown below).

Explanatory variables		Change in self-rated health status from baseline (2000)	
		Model 1 ($N = 5688$[a]) Good health at baseline cohort − ORs (95% CI) of having PH over time	Model 2 ($N = 2425$) Poor health at baseline cohort − ORs (95% CI) of having GH over time
Social participation: Lagged ($t − 1$)	Active participation	1.0[b]	1.15 (0.98–1.35)
	No participation	1.00 (0.89–1.13)	1.0[b]
Social participation: Non-lagged (t)	Active participation	1.0[b]	1.25 (1.06–1.47)**
	No participation	1.21 (1.07–1.37)**	1.0[b]
Generalised trust: Lagged ($t − 1$)	Trusts others	1.0[b]	1.25 (1.05–1.49)*
	Can't trust others	1.25 (1.10–1.42)***	1.0[b]
Generalised trust: Non-lagged (t)	Trusts others	1.0[b]	1.29 (1.08–1.54)**
	Can't trust others	1.45 (1.27–1.64)***	1.0[b]
Talks with neighbours: Lagged ($t − 1$)	1+/week	1.13 (0.97–1.31)	1.28 (1.05–1.55)*
	Less than this	1.0[b]	1.0[b]
Talks with neighbours: Non-lagged (t)	1+/week	1.13 (0.97–1.31)	1.27 (1.04–1.55)*
	Less than this	1.0[b]	1.0[b]

Source: The British Household Panel Survey, Waves J, M, O & Q (2000–2007).
*0.05 significance.
**0.01 significance.
***0.001 significance.
PH − Poor SRH.
GH − Good SRH.
  [a] Missing = 1.
  [b] Reference.

Measures of trust in this study therefore consider not just those individuals who maintain the same trust levels over time, but also those whose trust levels vary over the period of the study. That trust is measured at three lagged time points at ($t − 1$) in relation to the outcome SRH at time (t) adds considerable weight to the assumption that generalised trust is an independent predictor of individual health. Our study is not alone in showing association between generalised trust and SRH (for examples see Giordano & Lindström, 2010; Hyyppä, Mäki, Impivaara, & Aromaa, 2007; Kawachi et al., 1999, 1997) but this study is the first of its kind to empirically confirm any temporal relationships, and hence infer causality.

Though 'generalised trust' has been identified and subsequently used as a proxy for social capital in empirical research for over twenty years (Coleman, 1988, 1990; Giordano & Lindström, 2011; Kawachi et al., 1997, 1999; Lindström, 2004; Putnam, 1993, 2000; Veenstra, 2000), there is surprisingly little discussion regarding what else 'trust' is hypothesised to measure, outside this specific field. To address this issue, we pose (and attempt to answer) three pertinent questions:

1) Why is generalised trust a proxy for social capital?

The concept of trust is debated across a variety of disciplines, from social science, philosophy, economics, political science and public health. There is consensus, however, that different forms of trust exist: *generalised* (also known as 'horizontal') trust is the form specifically reserved for strangers (vs. *particularized* trust, the form reserved for known individuals or groups). Early social capital literature stresses the importance of both these trust variants to facilitate actions between individuals or groups (Coleman, 1988) i.e. without either form of trust there can be no social capital. Over time, however, *generalised* trust seems to have become the preferred social capital proxy. This is most likely due to the works of Robert Putnam (1993, 1995, 2000), and the assumption that individuals are all similarly influenced by the presence (or lack) of social capital (Coleman, 1990; Kawachi et al., 1997). On the surface this makes sense, as social capital has been described as a public good (Berkman & Kawachi, 2000, p.177) and by definition infers unconditional positive externalities

(unintended benefits) to all. However, cross-level interactions have shown that this may be an oversimplification, as the health of individuals who do not share the social 'norms' (i.e. trust) of the community in which they reside, may be adversely affected (Subramanian et al., 2002). This apparent paradox suggests that joint *particularized* and *generalised* trust measures could provide a more robust social capital proxy in future research.

2) What else could 'generalised trust' measure?

There is contention across disciplines regarding the answer to this question and social capital researchers should carefully consider the implications. From an economic point of view, generalised trust is often considered a *summary measure* of individual experiences, good and bad, the assumption being that generalised trust levels can vary over time (Glaeser, Laibson, Scheinkman, & Soutter, 2000). Though this opinion can also be found in political science, others within the field consider that generalised trust is determined in early life, levels being resistant to change irrespective of later-life experiences (Putnam, 2000; Uslaner, 2002). Our longitudinal data reveal that just over half our sample (∼55%) maintains the same trust levels over the seven-year study period, whereas ∼45% of individuals vary their trust across the same time frame. We therefore can offer only some support for Uslaner's concept of 'moralistic' (stable) trust; furthermore, Putnam's belief that '… all of the decline in social trust since the 1960s is attributable to generational succession' may be inadequate, as it does not explain individual fluctuations in trust over time, as seen in our data (Putnam, 2000, p.140).

Others within political science consider levels of generalised trust to reflect the function of State institutions (Levi, 1998). However, as empirical evidence shows only weak correlation between *vertical* trust (trust in institutions) and generalised trust (Rothstein, 2005; Uslaner, 2003) perhaps a more refined hypothesis is required. One such refinement is the theory that higher levels of generalised trust reflect State *policies* implemented to reduce inequalities, rather than State *institutions* themselves (Rothstein & Stolle, 2008; Uslaner, 2008).

Putnam implies that generalised trust is created as a by-product of increasing numbers of individuals interacting with each other via membership of community voluntary groups and local organisations (Putnam, 1995). That trust levels – in the USA at least – have declined over the same period that memberships in such local groups have declined is, for the most part, the backbone to Putnam's recent social capital theories (Putnam, 2000). However, it is noteworthy that perceptions of inequality in America have also increased over a similar time frame; could it be that State policy, not declining voluntary group membership, has inadvertently contributed to the decline in generalised trust in the USA (Uslaner, 2002)?

3) What are possible mechanisms linking trust levels to health?

Mechanisms linking trust to health outcomes must depend on what trust is deemed to measure (see #2 above). If Putnam's ideas are followed, trust is a by-product of increased social participation; therefore, logic dictates that trust is just one step along the pathway from participation to health. As to how participation influences health could therefore be via social support mechanisms, examples of which include instrumental, emotional and financial support. Our results reveal that *high* levels in social participation precede good SRH, lending partial weight to this hypothesis (though attenuated in our sensitivity test – see Table 3). That *low* levels of participation do not precede poor SRH in this study could imply that only longer term reduction in social participation leads to a decline in social support and worse health. Conversely, it has been argued that trust is in fact a prerequisite to participation (Rothstein & Stolle, 2003). If this is the case then it is trust, not participation, at the start of the causal pathway to health.

If higher trust levels are a reflection of efficient State institutions (Levi, 1998), it could be theorised that improved access to resources such as education, healthcare, rule of law, etc. is the real link from trust to health. However, that our data come from the UK, where access to such resources could be considered relatively homogeneous (compared to low- or middle-income countries) implies other mechanisms may also be at work.

If trust levels are considered a measure of egalitarian State *policy* designed to redistribute wealth and reduce inequality (Rothstein & Stolle, 2008; Uslaner, 2008), then maybe the pathway from trust to health is via psychosocial pathways. As described by Giordano et al. (2010), the authors employ Wilkinson's (1996) theories as to how psychosocial pathways are a plausible mechanism from trust to health outcomes. Long term exposure to high levels of chronic stressors (considered by the authors as an indicator of low generalised trust levels) can, via the hypothalamic–pituitary–adrenal axis, lead to increased levels of blood cortisol, which in turn may lead to diseases such as depression and cardio-vascular disease (Shively, Musselman, & Willard, 2009; Watson & Mackin, 2006). It is feasible that egalitarian State policy could influence health outcomes *directly*, by addressing the negative effects of social stratification on individual heath and *indirectly*, via the reduction of perceived chronic stressors with the creation of higher generalised trust levels.

Strengths and weaknesses

A major strength of this study is the fact that it is longitudinal, tracking the same individuals ($N = 8114$) at four points in time over seven years. The unique design captures association between *all* lagged ($t - 1$) explanatory variables and changes from baseline SRH at time (t), allowing us to infer causality by temporal relationships (Goodman & Phillips, 2005). To our knowledge, this is the first time that this has been attempted within the field of social capital. Disaggregation by baseline SRH also enables us to assess association between social capital and health in two large independent cohorts within the same study. Our findings are strengthened by the fact that we see similar patterns (albeit with some differences) for these two cohorts. The fact that the data were obtained via interview rather than relying on postal questionnaires contributed to the very high participation rate of around 90%, year on year (Taylor et al., 2010). By lagging ($t - 1$) *all* explanatory variables, including three different individual-level indicators of social capital, along with multiple SES, social support variables and confounders, we ensured that well-known health determinants were also included in the analyses.

A major limitation of this study is that the BHPS sample was originally selected to reflect the UK population as a whole and deliberately avoided oversampling of smaller sized communities, i.e., data are not particularly valuable when investigating ethnic diversity or urban vs. rural populations. Furthermore, our longitudinal data were unsuitable to perform any meaningful contextual analysis at the household- or community-level. Disaggregation of SRH in year 2000 could introduce bias (misclassification of exposure) at baseline; one way to reduce this potential bias could be to combine SRH responses from 2000 and 2003, however, this would leave just two further points in time to address issues of temporal relationships. As SRH is considered a valid and reliable indicator of morbidity and future mortality (Idler & Benyamini, 1997; Lopez, 2004), we feel that a single baseline measurement is sufficient. By year 2000, only 62.0% of the original cohort members were able to answer the questions posed (Taylor et al., 2010). This would have introduced further selection bias into this study.

Marital status was reduced to the dichotomous 'married' and 'not married'; though this method of reduction has been previously validated (Afifi, Cox, & Enns, 2006), it may hide more complex pathways regarding cohabitation, common in society today. The 'lives alone' variable was included in an attempt to recapture this detail.

Although temporal relationships are considered 'essential' in establishing causality, it is an oversimplification to assume that causality is proven solely based upon association shown in our results. To address this concern, we performed sensitivity testing – see 'Statistical analyses' and 'Results' sections for more detail. That association between trust at time ($t - 1$) and SRH at time (t) remained even after considering trust at time (t) serves to strengthen (in part) our hypothesis that levels of social capital at time point A are positively associated with SRH at time point B.

Conclusions

Our longitudinal study is the first of its kind to investigate temporal relationships between individual-level social capital proxies and self-rated health. It appears from our results that generalised trust can be considered an independent predictor of future health status. However, lack of consensus across a variety of disciplines as to what generalised trust could measure creates ambiguity as to which mechanisms link higher trust levels to better health. That 'generalised' trust is only weakly correlated with 'vertical' trust (in State institutions) and social participation implies that higher trust levels could reflect egalitarian State *policy* not State institutions, *per se*. It is feasible that such policies could influence health outcomes directly, through the redistribution of wealth and reduction of inequalities, and indirectly via the creation of higher levels of generalised trust.

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Appendix A

To determine social participation levels, respondents were asked if they were *active* members of any local group or organisation listed below:

Political party, trade union, environmental group, parents'/school association, tenants'/residents' group or neighbourhood watch, church organisation, voluntary service group, pensioners group/organisation, social club/working men's club, sports club or Women's Institute.

The six occupation categories, as per the Registrar General's Social Classification of occupations are: i) Professional, ii) Managerial/Technical, iiia) Skilled (non-manual), iiib) Skilled (manual), iv) Partly Skilled and v) Unskilled.

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