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Xindong Xue
W. Robert Reed
Andrea Menclova

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Department of Economics and Finance
School of Business
University of Canterbury
Private Bag 4800, Christchurch
New Zealand

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Social Capital and Health: A Meta-Analysis

Xindong Xue[1]
W. Robert Reed[2†]
Andrea Menclova[2]

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Abstract: The relationship between social capital and health has received extensive attention in fields such as public health, medicine, epidemiology, gerontology and other health-related disciplines. In contrast, the economics literature on this subject is relatively small. To address this research gap, we investigate the cross-disciplinary empirical literature using meta-analysis. We analyze 12,778 estimates from 470 studies. Our analysis finds that social capital is significantly related to a variety of positive health outcomes. However, the effect sizes are consistently very small. This finding is robust across different types of social capital (e.g., cognitive, structural, bonding, bridging, linking), and for many different measures of health outcomes (e.g., mortality, disease/illnesses, depression). The small effects that we estimate cast doubt on recent initiatives to promote health through social capital such as those by the WHO, the OECD, and US Healthy People 2020.

Keywords: Social capital, Health, Meta-analysis, Mental health, Physical health, Self-reported health

JEL Classifications: B49, C49, I10, I31

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[1] School of Public Administration, Zhongnan University of Economics and Law, and Collaborative Innovation Center for Social Governance of Hubei Province, CHINA

[2] Department of Economics and Finance, University of Canterbury, NEW ZEALAND

†Corresponding author: W. Robert Reed, email: bob.reed@canterbury.ac.nz

I. INTRODUCTION

This study examines the literature on social capital and health. It has long been recognized that social capital plays an important role in economic affairs. A recent search on Web of Science identified over 1,000 studies in economics journals in which social capital appears in the title. Research on social capital has been published in the *American Economic Review* (Guiso, Sapienza, & Zingales, 2004; Karlan, 2005), the *Quarterly Journal of Economics* (Knack & Keefer, 1997, Glaeser, et al., 2000; Bowles & Gintis, 2002), the *Economic Journal* (Glaeser, Laibson, & Sacerdote, 2002; Durlauf, 2002; Helliwell, 2006), and other top economics journals. The topic continues to be of interest to economists (McCoy, McDonough, & Roychowdhury, 2019; Hoi, Wu, & Zhang, 2019; de Vaan, Frenke, & Boschma, 2019; Andini & Andini, 2018; and Wang, 2019).

However, most of the economic research on social capital has focused on areas such as economic growth (Knack & Keefer, 1997; Algan & Cahuc, 2010), financial development (Guiso, Sapeienza & Zingales, 2004), political governance (Putnam, 1993; Nannicini et al., 2013) and formation of large firms and organizations (La Porta et al., 1997). While some economic research has focused on health, the literature is relatively small. This is surprising, not only because social capital has been found to be an important determinant of economic outcomes in other areas, but also because there is a voluminous literature on social capital and health outside of economics, particularly in public health, medicine, epidemiology, gerontology and other health-related disciplines.

This study uses meta-analysis to aggregate empirical findings in the literature on social capital and health.[1] Our goal is to assess the overall economic impact of social capital on health

[1] Previous meta-analyses of social capital and health have been published outside of economics (De Silva et al., 2005; Holt-Lunstad et al., 2010; Gilbert et al., 2013; Nyqvist et al., 2014). A working paper version of this manuscript describes the value-added of our analysis. Among other things, our study far exceeds these previous efforts in scope and detail. Further, previous studies have directly combined disparate estimates. In contrast, we translate estimates into partial correlation coefficients, which is a more appropriate method for comparing and combining diverse estimates.

and determine whether it is "large" or "small". Given the broad scope of both "social capital" and "health", we also want to dig deeper into the overall relationship to explore whether certain types of social capital are particularly impactful for certain types of health outcomes.

Our primary motivation is to determine whether this is a subject that should attract more attention from economists. If studies from other disciplines, and the extant economics literature on this topic, have found that social capital is an important determinant of health, then the dearth of economic research in this area represents a gap that should be addressed. On the other hand, if the conclusion from existing studies is that social capital is not an important determinant of health, then this justifies the relative lack of attention this subject has received in the economics discipline. Further, it would call into question recent initiatives to promote health through social capital by the WHO, the OECD, and US Healthy People 2020 (Rocco & Suhrcke, 2012; Centers for Disease Control and Prevention, 2010; Keeley, 2007).

This study proceeds as follows. Section II reviews the economics literature on social capital. Section III explains our use of meta-analysis and our strategy for combining diverse estimates from different studies. Section IV presents and discusses the data that we use in our analysis. Section V presents initial estimates of the "overall" effect of social capital on health, along with the results of a commonly used test for publication bias. Section VI uses meta-regression to identify the factors that affect the size of the estimated effects of social capital on health. It also takes a closer look at finer categories of social capital and health to see if there are some kinds of social capital that may affect some kinds of health outcomes, even if the overall effect is small. Section VII concludes by summarizing our results and drawing out implications for public policy and further study.

II. LITERATURE ON THE ECONOMIC EFFECTS OF SOCIAL CAPITAL

As noted above, there is a large literature that investigates the relationship between social capital and economic activity. A number of channels have been proposed for this relationship (Paldam

& Svendsen, 2000). Social capital may enter the production function directly, alongside physical capital and human capital; e.g., in the form of consumer networks, reputation, and goodwill. Social capital may reduce free-riding and thus lessen the need for regulation in settings that require cooperation, reducing monitoring costs. Further, social capital in the form of trust can reduce transaction costs, facilitating economic exchange and contributing to economic growth.

The last mechanism has received the most empirical attention. LaPorta et al. (1997) show that trust improves the performance of large organizations. Zak and Knack (2001) present empirical evidence that trust is significantly related to economic growth. Their findings are corroborated by Beugelsdijk, De Groot, & Van Schaik (2004). Algan & Cahuc (2010) further substantiate the relationship between trust and economic growth by using "inherited trust" of descendants of US immigrants to establish a causal link between trust and growth.

Another area that has received much attention is the relationship between trust and economic institutions. Berggren & Jordahl (2006) find that legal structure and security of property rights increase trust. Alesina, & Giuliano (2015) provide a recent summary of how culture, trust, and institutions interact to affect economic outcomes. Relatedly, Guiso, Sapienza, & Zingales (2004) and Tabellini (2010) find a strong link between culture, of which trust is a major component, and trade and economic development, respectively. Overall, there is broad consensus in the literature that the economic benefits of social capital in general – and trust in particular – are large.

In contrast, the role of social capital in health is more opaque. Folland (2008) identifies a number of avenues by which social capital can affect health. Social capital may reduce stress through supportive relationships, trust, and the benefits of socializing. Stress reduction, in turn, positively affects health. Social capital in the form of socializing may make it easier to obtain (health-related) information. Further, having strong social ties may encourage individuals to make more "responsible" choices as their health and wellbeing are of importance to others

whom they care about.

In Folland's (2008) formal model, social capital affects an individual's utility U both directly, and indirectly via its effects on health:

(1.a) $U = U(S, H, X)$, where

(1.b) $H = H(S, C(S))$,

and S is social capital, H is health, X are other consumption goods, and C are all other health inputs.

Social capital in the form of socializing may improve health directly. For example, as noted above, supportive relationships can improve health by reducing stress, so that $\frac{\partial H}{\partial S} > 0$. Social capital can also affect health indirectly. For example, strong family relationships may encourage an individual to obtain regular medical checkups, C, so that $\frac{\partial H}{\partial C}\frac{\partial C}{\partial S} > 0$. Alternatively, a desire to belong to a peer group which shares cigarettes may encourage an individual to smoke, C, $\frac{\partial H}{\partial C}\frac{\partial C}{\partial S} < 0$. As a result, the overall effect of social capital on health is theoretically ambiguous.

Other formal models of the relationship between health and social capital are presented by Costa-Font & Mladovsky (2008) and Laporte (2014). Costa-Font and Mladovsky (2008) model the effects of social capital on health via the effects of peers on health-related preferences. This corresponds to the effect of S on C, and subsequently H, in Folland's model above. However, Costa-Font & Mladovsky (2008) extend this logic by modelling the propagation of preferences within a group through a social multiplier effect. Their approach allows for the dissemination of both health-improving (e.g., physical activity, medical checkups, vaccinations) and health-retarding (e.g., social smoking, drinking, and the obesity epidemic) behaviors within a social group.

Laporte (2014) extends the classic Grossman (1972) model of investment in health to include social capital. She allows for two, complementary types of social capital, private social capital, in which the individual invests over time, and public social capital, which is exogenously determined. One consequence of the model is that increased geographical mobility on the part of health consumers can induce them to invest less in private social capital. Communities may respond by investing more in public social capital. This highlights the role of public policy in social capital. Folland, Kaarbøe, & Islam K. (2014) also explore the role of public policy in the provision of social capital.

The above provides ample theoretical reasons for believing that social capital can be an important input to health. It also points out that social capital's influence need not always be positive. Either way, the size of the economic impact of social capital on health is an empirical question. The next section explains the methodology we will use to answer this question.

III. METHODOLOGY

We use meta-analysis to aggregate empirical findings in the cross-disciplinary literature on social capital and health. To be included in our meta-analysis, a study must estimate the "effect" that social capital has on health. As will be discussed below, there are a variety of estimation procedures and model specifications that studies have used to do this. Conceptually, we can represent these efforts with a linear model that regresses a measure of health (*H*) on a measure of social capital (*S),* along with a set of control variables (Z_k):

(1) $$H = \beta_0 + \beta_1 S + \sum_{k=2}^{K} \beta_k Z_k + error.$$

Let $\hat{\beta}_{1i}$ be the effect estimated by study *i*, and let there be a total of *M* estimates produced by multiple studies.[2]

[2] A complication arises because increases in the health variable can mean an improvement or a decline in health, depending on how it is measured. For example, a positive estimate of β_1 in equation (1) when health is measured by mortality means something different than when health is measured by a categorical variable increasing in good health. Likewise, sometimes a measure of social capital is defined so that a larger number means an increase in social capital, but sometimes it is measured so that a larger number means a decrease in social capital. In order to

In meta-analysis, the estimated effects, $\hat{\beta}_{1i}$, become the dependent variable. OLS estimation of α_0 in the equation below produces a value equivalent to the arithmetic average of the *M* estimates.

(2) $\hat{\beta}_{1i} = \alpha_0 + \varepsilon_i$, *i = 1,2,...,M,*

If the individual estimates constitute a representative sample from the population of estimated effects, then OLS will produce an unbiased estimate of the mean true effect of social capital on health.[3]

However, the OLS estimate will not be efficient. OLS gives equal weight to the individual estimated effects. But some of the $\hat{\beta}_{1i}'$s are estimated more precisely than others, as indicated by their different standard errors, $s.e.(\hat{\beta}_{1i}) \equiv SE_i$. An efficient estimator would assign greater weight to more precise estimates. If all estimates come from a population with the same true effect, so that the variation in ε_i is proportionate to the sampling error -- i.e., $\text{var}(\varepsilon_i) = (SE_i)^2\sigma^2$ -- then Weighted Least Squares (WLS) will be efficient, with the appropriate weight being the inverse of $(SE_i)^2$.

This model of effect size heterogeneity is known in the meta-analysis literature as "Fixed Effects", and is not to be confused with the panel data estimator of the same name. "Fixed Effects" WLS estimation of equation (2) is equivalent to dividing each observation by SE_i and then estimating with OLS:

(3) $\frac{\hat{\beta}_{1i}}{SE_i} = \alpha_0 \cdot \left(\frac{1}{SE_i}\right) + \frac{\varepsilon_i}{SE_i}$ *i* = 1,2,…,*M*.

Assuming representative sampling, "Fixed Effects" WLS estimation of α_0 will produce an unbiased and efficient estimate of the mean true effect of social capital on health.

Many researchers find the "Fixed Effects" model of effect heterogeneity too restrictive.

get the sign of the effect consistent across studies, we standardized the signs of the estimates so that a positive estimate implied that an increase in social capital was associated with an increase in good health.

[3] We address issues of publication bias and endogeneity below.

More likely, there is not a single, true effect of social capital on health, but a distribution of true effects. This model of effect heterogeneity is known in the meta-analysis literature as "Random Effects", which again should not be confused with the panel data estimator of the same name.

Let τ^2 represent the variation in ε_i due to the fact that estimated effects are drawn from populations with differing true effects. Assuming the two sources of variation in ε_i are independent, then $\text{var}(\varepsilon_i) = [(SE_i)^2 + \tau^2]\sigma^2 = \omega_i\sigma^2$. The corresponding "Random Effects" WLS estimator is equivalent to estimating the following equation by OLS:

$$(4) \qquad \frac{\widehat{\beta}_{1i}}{\omega_i} = \alpha_0 \cdot \left(\frac{1}{\omega_i}\right) + \frac{\varepsilon_i}{\omega_i}, \; i = 1,2,\ldots,M.$$

Note that the "Random Effects" estimator produces a more uniform distribution of weights than "Fixed Effects", since the weighting terms include a common constant, τ^2. While researchers generally agree that the "Random Effects" model most closely matches reality, there is some debate about which works best in practice (Doucouliagos & Paldam, 2013; Reed, 2015). Accordingly, our analysis uses both.

A related issue concerns the weighting of estimates versus studies. The number of estimates per study can vary widely. In our sample, partly because many specifications contain multiple measures of social capital, the number of estimates per study ranges from 1 to 240, with a mean of 27. The preceding WLS estimators implicitly give greater weight, sometimes dramatically greater weight, to studies with more estimates. Accordingly, we employ an alternative weighting system that, ceteris paribus, gives equal weight to studies rather than individual estimates. In the subsequent discussion, *Weight1* and *Weight2* denote the weighting systems that give equal weights to individual estimates and studies, respectively.

Two more issues need to be addressed. The preceding assumes that it is meaningful to average estimated effects across studies. However, in the empirical literature on social capital

and health, different measures are used for both health and social capital. Further, different estimation procedures are employed – linear regression, ordered probit models, hazard models, odds ratio models and more – so that the interpretation of coefficients varies greatly, despite the fact that the respective studies are all concerned with estimating the effect of social capital on health.

This is a common situation in meta-analysis, and there is a common solution: to convert the respective estimates to partial correlation coefficients (PCCs):

$$(5) \qquad PCC_i = \frac{t_i}{\sqrt{t_i^2 + df_i}},$$

where t_i and df_i are the *t*-statistic and degrees of freedom associated with the respective estimated effect. The associated standard error is given by:

$$(6) \qquad SE(PCC_i) = \sqrt{\frac{1 - PCC_i^2}{df_i}}.$$

The preceding analysis still holds, except that $\hat{\beta}_{1i}$ is replaced by PCC_i, and SE_i now stands for the standard error of PCC_i, so that equations (3) and (4) become

$$(3') \qquad \frac{PCC_i}{SE_i} = \alpha_0 \cdot \left(\frac{1}{SE_i}\right) + \frac{\varepsilon_i}{SE_i} \quad i = 1,2,\ldots,M;$$

and

$$(4') \qquad \frac{PCC_i}{\omega_i} = \alpha_0 \cdot \left(\frac{1}{\omega_i}\right) + \frac{\varepsilon_i}{\omega_i}, \quad i = 1,2,\ldots,M.$$

Accordingly, the parameter α_0 represents the mean true effect of social capital on health measured as a correlation.

How should one assess the estimates of α_0? Like any correlation, *PCC* takes values between -1 and 1. Cohen (1988) suggested that correlation values of 0.10, 0.30, and 0.50 in absolute value should be interpreted as "small", "medium" and "large" effects, and his interpretation is widely accepted. However, as Doucouliagos (2011) points out, Cohen's

taxonomy refers to simple, not partial, correlations. To investigate partial correlation sizes, Doucouliagos collected over 22,000 estimates in empirical economics and converted them to *PCCs*. He then ranked them from smallest to largest in absolute value. He defined the 25th, 50th, and 75th percentile values as "small", "medium", and "large". While there was some difference across subfields of economics, overall *PCC* values of 0.07, 0.17, and 0.33 corresponded to "small", "medium" and "large" effect sizes, respectively. This establishes a scale for comparing *PCC* values to other *PCC* values in the literature, and it is the standard we will employ in interpreting our empirical work.[4]

IV. DESCRIPTION OF DATA

Selection of studies. We followed the MAER-Net protocols outlined by Stanley et al. (2013) in our search for studies. To account for the multi-dimensional nature of both social capital and health, the following combination of key words was used: "social capital", "social trust", "social networks", "social participation", "social support", "social engagement", "social integration", "social relationships", "social ties", "reciprocity", "social cohesion", "social connections", "social connectedness", "volunteering", "health", "mortality", "depression" and "disease".[5]

[4] A remaining issue concerns the determination of the *t*-statistic used in calculating *PCC* in equation (5). In many cases, studies either directly report the *t*-statistic corresponding to the estimated coefficient, or they report the standard error, from which the *t*-statistic is easily calculated. However, in the social capital and health literature, the most common estimation procedure is some variant of an odds ratio model, where the estimated coefficient is the odds ratio associated with a binary outcome. In addition to the estimate, studies either report the standard error of the estimated odds ratio, or the lower and upper bounds of a 95% confidence interval. In the former case, the *t* (or better, *z*) statistic is calculated by $t_i = \ln(\hat{\beta}_{1i}) \cdot \hat{\beta}_{1i}/s.e.(\hat{\beta}_{1i})$. In the latter case, one first calculates $SE_{ORi} = (\ln(UpperBound_i) - \ln(LowerBound_i))/(2 \cdot 1.96)$, and then calculates $t_i = \ln(\hat{\beta}_{1i})/SE_{ORi}$. Calculation of *PCC* proceeds accordingly. A related complication arises when studies only report the coefficient and a set of stars to indicate the level of statistical significance: e.g., *** = significant at the 1 percent level, ** = significant at the 5 percent level, * = significant at the 10 percent level, and no stars = insignificant at the 10 percent level. In these cases, we set the *p*-value = 0.005, 0.025, 0.075, and 0.50, respectively, and work backwards from the inverse of the *t*-distribution to calculate a *t*-value. We record how we calculate our *t*-values to see if the respective methods affect our results.

[5] The search was conducted using the search engines EconLit, JSTOR, EBSCO, Google Scholar, RePEc, SSRN, Social Science Citation Index (SSCI), Science Citation Index (SCI) and Scopus. Backward and forward citation searching was employed to leverage articles identified through the search engine process. We also manually searched academic journals that were found to have published studies on social capital and health. To be as comprehensive as possible, we also searched working papers, books, doctoral dissertations, master theses, and

We followed a stepwise procedure to finalize a set of studies from which to draw our estimated effects. First, we excluded theoretical studies that did not report estimated effects. Second, we excluded studies that did not report sufficient information to calculate *PCC* values and their standard errors. Specifically, a study had to report a numerical estimate for the effect of social capital on health, and a corresponding standard error, t-statistic, confidence interval, or p-value.

Third, we excluded studies that estimated the effect of social capital on well-being, welfare, quality of life, and life satisfaction. While indirectly related to health, these outcomes are not comparable to direct health effects. Fourth, we eliminated studies using interaction terms and quadratic specifications of the social capital variable because of the difficulty of combining multiple coefficients to calculate a single estimated effect with standard error (cf. Gunby, Jin & Reed, 2017).[6] For similar reasons, we dropped studies employing path analysis that use social capital as a mediator variable. Lastly, we excluded studies measuring the intergenerational effect of social capital on health; e.g., the effect of parent's social capital on child's health.

Study coding. For each estimate in our sample, we coded data to enable construction of effect sizes and their standard errors, and to record corresponding study, data and estimation characteristics. Information included the study's author(s), type of publication (e.g., journal), journal name, publication year, countries included in the study sample, number of observations, and data type (cross-sectional or panel). We recorded the estimated coefficient and associated statistics (such as standard errors, confidence intervals, etc.).[7]

government reports. The search was ended in September 2017.

[6] Note that we do not encounter problems with calculating marginal effects for nonlinear models because all we require for *PCC* is to be able to calculate a *t*-statistic. The problem arises when more than one coefficient is involved, such as when there is an interaction or quadratic term.

[7] We note that due to poor reporting practices and the generally large number of observations used in the respective studies – the median number of observations for a given estimate in our study was approximately 3,500 – we substituted the number of observations for degrees of freedom in calculating *PCC* and *SE(PCC)* in equations (5) and (6).

We categorized the estimation procedures used to produce each estimate. Binary variables were used to indicate the following methods: OLS, FGLS, probit/logit, ordered probit/logit, Hierarchical Linear Model (HLM), and instrumental variables (IV). In addition, a set of dummy variables were used to indicate whether common control variables were included in the regression specifications (e.g., age, gender, income, etc.).

Endogeneity is certainly a concern in the social capital and health literature. While most studies assume that the effect runs from social capital to health, the literature does recognize that causation can go in the opposite direction. For this reason, our analysis will pay particular attention to estimates derived from panel data or IV estimation procedures. Holding other factors constant, we will want to investigate whether these estimates differ systematically from estimates that do not address endogeneity.

For each estimate, we coded the associated health outcome, type of social capital, and whether the social capital variable was individual-based or was aggregated at the community level. TABLE 1 provides some detail about the different measures of health employed by the studies in our sample. Measures of health consisted of measures of physical health, mental health, and measures of overall/general health. Among measures of physical health, the most common measure was mortality, usually "all-cause" mortality, but sometimes mortality due to a particular illness or disease, such as cancer or cardio-vascular disease. Studies measured mortality over given sample periods, and as the sample periods differed in length, the interpretation of mortality rates differed accordingly.

The second most common type of physical health measure was the presence or onset of a particular illness or disease (e.g., hypertension, heart disease, diabetes). Various measures of overall health were also common. Sometimes these were indices constructed from multiple questions about a person's health, and sometimes they were categorical measures in which a respondent's health was characterized as good, fair, poor, etc. A substantial number of the

physical health measures relied on respondents' own assessments.

Among mental health measures, depression was the most common category of mental health. Other categories included dementia, mental distress or anxiety, and measures of cognitive ability. As with physical health, many of the studies used an overall measure of mental well-being. Perhaps not surprisingly, a very large number of mental health measures relied on self-reported assessments.

TABLE 2 gives a sense of the wide variety of social capital variables used by the studies in our sample. The most common framework employed by studies was cognitive/structural, where cognitive refers to what people feel, and structural to what people do. Social trust was the most common type of cognitive social capital, followed by the perception of social support, and then the perception of social cohesion. The key element here is the respondent's perception of these constructs. The most common measures of structural social capital was participation in some form of social activity, followed by measures of one's network of personal relationships. It is noteworthy that some measures of social capital mixed the two types of social capital, often by composing an index of social capital that relies on both.

An alternative framework for categorizing social capital is bonding/bridging/linking. While less common than cognitive/structural, it is still widely used. Bonding refers to horizontal ties between similar people, while bridging refers to horizontal ties between dissimilar people. Linking refers to hierarchical relationships. Of these, bonding was the most commonly used social capital variable in the studies included in our sample.

V. DATA ANALYSIS: Part 1

Our initial dataset consisted of 13,040 observations gleaned from 471 studies. Calculation of *PCC* uses *t*-values and *df*, so the first two columns of TABLE 3 focus on these variables. The full sample of *t*-values has mean and median values of 1.67 and 1.20, respectively. We will comment on these relatively low values later. For now, we wish to note the minimum and

maximum values of -59.67 and 850. This raises concern with outliers. A similar concern applies to the *df* variable. It has mean and median values of 29,023 and 3,300, with minimum and maximum values of 5 and 2,442,948.

The corresponding distribution of *PCC* values ranges from -0.747 to 0.998, with mean and median values of 0.030 and 0.019. Large (absolute) values of *PCC* are potentially a problem because of the key role that *PCC* plays in determining the standard error, and hence, the weights used in the empirical analysis: $s.e.(PCC_i) = \sqrt{\frac{1-PCC_i^2}{df_i}}$, with weights increasing in the absolute value of *PCC*.

As a result, we proceed by truncating the top and bottom 1% of *PCC* values, leaving 12,778 observations from 470 studies. The truncated distributions of *t*-statistic, *df*, and *PCC* values are also reported in TABLE 4, immediately to the right of the full sample statistics.[8] Corresponding histograms for the *t*-statistics and *PCC* values are reported in FIGURE 1. The two histograms in FIGURE 1 and corresponding columns in TABLE 3 go far in answering our first question about the size of the effect of social capital on health.

The mean and median *PCC* values for the truncated sample are 0.028 and 0.019. Based on Doucouliagos (2011), these do not even get close to the threshold value of 0.07 that Doucouliagos sets for "small". If social capital has an effect on health, these values suggest that the effect is very small. The reasons for the small *PCC* values are not hard to identify. First, a large number of estimates in the literature are statistically insignificant. The table immediately below the histogram in the top panel of FIGURE 1 reports that 56.4% of all *t*-values lie between -2 and 2. Compounding these relatively low *t*-values are relatively large sample sizes. The distribution of *df* values for the truncated sample ranges from 5 to 2,442,948, with a median value

[8] As a robustness check, we also truncated the observations based on the lowest and highest one-percent of t-values and df's. We redid all the analyses with this alternative truncation strategy. The results were very similar to those reported in this paper, and the qualitative conclusions were identical.

of 3,451. If we calculate the *PCC* value that corresponds to the median *t* and *df* values using equation (5), we obtain a value for *PCC* equal to 0.020.

However, there are two caveats. First, the numbers in TABLE 4, and the values represented in FIGURE 1, are unweighted. So we need to re-compute our estimate of the mean true effect, α_0, using the different weighting schemes described above. Second, the analysis ignores publication bias.

Publication bias arises when the results reported by researchers, and/or the studies accepted for publication by journals, comprise a biased sample of the population of all estimates. Note that "publication bias" can occur even in working papers that are not published in journals. This can happen if researchers choose not to write up results because the initial analyses did not produce interesting/promising findings.[9] In that case, even unpublished working papers can be characterized by publication bias.

Publication bias is widely recognized as a problem, with selection typically favoring estimates that are statistically significant, and/or consistent with researchers' and journals' preconceived beliefs (Christensen & Miguel, 2018). That being said, we note that in order for publication bias to explain the low *PCC* values we see in our sample, it would have to discriminate against statistically significant estimates. Most researchers would view this as unlikely.

FIGURE 1 and TABLE 3 present "overall" estimates, aggregating estimates of the effects of social capital across different kinds of health outcomes. To address concerns about the validity of combining these estimates, FIGURE 2 breaks the full sample of *PCC* values into three subsamples, depending on whether the health outcome is physical health, mental health, or general health. A table at the bottom of the figure reports means and standard deviations for

[9] Franco, Malhotra, and Simonvits (2014) report that the main source of publication bias is failure of researchers to write up results that are not significant or interesting.

each of the three subsamples. Mean *PCC* values are similar, with most values lying within the range characterized by Doucouliagos (2011) as small. The similar distributions provide casual support for the legitimacy of combining estimates for the different health outcomes. We will explore this subject in greater detail below.

TABLE 4 describes some of the other variables in our data. Approximately 98% of the estimates in our sample are drawn from peer-reviewed journals (*Journal*). It is common in meta-analyses to include a mix of published and unpublished studies, mostly to address publication bias. While our initial search produced a larger set of working papers, most of these were subsequently published, and were eliminated as duplicates. As it turns out, publication bias ends up not being a major concern in our study. While we find evidence of publication bias in our subsequent analysis, its estimated influence is very small, a result foreshadowed by the small *PCC* values reported above. The variable *PubYear* reports that the studies in our sample were published in the window from 1985 to 2017.

Social capital can be constructed on an individual-level (Glaeser et al., 2002) or group-level (Coleman, 1990; Putnam, 1995) basis. 86.5% of the estimates in our sample are associated with individual-level measures (*IndividualSC*). Our analysis will address whether individual- and group-level measures of social capital have different effects on health.

Our sample covers a diverse set of countries, with the largest number of estimates using data from Western or Northern Europe (33.7%). East Asia (20.3%) and the USA (21.1%) were also common areas to study, but a substantial number of estimates came from countries other than those above.

Of the three categories of health outcomes, most studies examined the effect of social capital on a general measure of health (40.4% of estimates), closely followed by physical health (37.7%) and then mental health (23.0%). Two thirds of the estimated effects were based on self-reported assessments of health.

Almost all of the estimates were derived using social capital variables categorized as cognitive and structural social capital (92.2%). Another perspective, which includes bonding, bridging, and linking social capital, was less commonly employed (28.0%). It is quite common for studies to include an array of social capital variables in a single regression equation (*NumberSCVariables*). In our sample, these ranged from 1 to 28 separate social capital variables in a single specification, with a mean of 6.5 social capital variables per equation.

We also tracked the control variables that were commonly included in studies of social capital and health. 85.2% of estimates came from a regression specification including an age variable; 84.5% included a gender variable; 59.9% included an education variable; 38.9% included a marital status variable; and 40.3% included an income variable.

A large number of different estimation procedures were used to produce the estimated effects in our sample. The most common procedure involved the odds ratio model, such as logistic regression or hazard model estimation (55.2%). The next two most common procedures were hierarchical linear modelling (17.9%) and ordinary least squares (13.9%). Other procedures included probit and logit models, ordered probit and logit models, feasible generalized least squares, and instrumental variable estimation. We note that only a very low percentage of estimates employed instrumental variable procedures (1.9%).

The standard error of the estimated effect plays an important role in weighting observations. As a result, we recorded the procedure used to estimate the standard error, categorizing these as either assuming spherical errors (homoskedasticity, error independence) or not. 25.5% of the estimates calculated standard errors assuming some form of nonspherical error behavior.

Finally, three different methods were used to derive *t*-values. In 21.5% of the cases, *t*-statistics were either directly reported, or could be calculated by dividing the estimated coefficient by its reported standard error (*tNormal*). In 17.8% of the cases, all we had was a p-

value, and we worked backwards from the inverse *t*-distribution function to obtain a *t*-value (*tCalculatedBypValue*). However, in most cases (60.6%), the *t*-statistic was calculated using the reported confidence interval or from the log of the estimated odds ratio (*tCalculatedByCI*).[10]

While we are aware of no study that compares the frequency of "Fixed Effects" and "Random Effects" estimators in the meta-analysis literature, our sense is that "Fixed Effects" is generally preferred by researchers. TABLE 5 identifies a concern with "Fixed Effects". It calculates a "study weight" for each study in our sample, weighting the individual estimates of that study by the respective weighting scheme ("Fixed Effects"/"Random Effects") and then aggregating the weights at the study level. In this way, each study receives a weight, the sum of which equals 100%.[11]

If the 100% weight was divided equally across studies, given 470 studies, each study would receive a weight of 0.21%. Against this benchmark, "Fixed Effects" produces a highly skewed weighting distribution. The median weight is 0.01%, and the maximum weight for a single study is 45.2%.[12] The top 3 studies (out of 470!) account for 67.4% of the total weight, and the top 10 studies comprise almost 77.3%. Thus the "Fixed Effects" estimate will disproportionately weight a small number of select studies that have large *PCC* values and/or use a large number of observations (*df*).

In contrast, the large size of τ^2 in the "Random Effects" estimator swamps the size of the individual $(SE_i)^2$ terms, so that the "Random Effects" estimator weights the individual estimates much more uniformly.[13] The median value is 0.23%, compared to a mean value of

[10] See footnote #4 for more details.

[11] Study weights were calculated by $w_i/\sum w_i$, where $w_i = 1/(SE_i)^2$ or $w_i = 1/[(SE_i)^2 + \tau^2]$ depending on whether Fixed Effects or Random Effects were being used (cf. Rinquist, 2013, page 128).

[12] Our ID for this study is 177 (Blakely et al., 2006). It has 72 estimated effects of social capital on health, and the number of observations in the respective samples ranges from 2,306,760 to 2,442,948.

[13] The corresponding I^2 value is 89.9%, which indicates that τ^2 is approximately nine times as large as the average SE^2 value.

0.21%. The maximum weight any single study receives is 0.26%, and the top 10 studies account for 2.63%. This arguably overcompensates the extreme skewness of the "Fixed Effects" estimator. In what follows, we report both "Fixed Effects" and "Random Effects", with a mild preference for the latter because it is not so heavily dependent on a small number of studies. As a practical matter, all of our key findings are robust across estimators.

Publication bias. Publication bias represents a serious challenge to the validity of meta-analysis. If the estimates in the literature are disproportionately large and significant, then averaging them will preserve this bias, producing a distorted estimate of the mean true effect. Methods to identify and correct publication bias remains an active research area in the meta-analysis literature (Andrews & Kasy, 2017; Stanley et al., 2017; Alinaghi & Reed, 2018).

A common test for publication bias is given by the Funnel Asymmetry Test (FAT). The FAT is carried out by adding the standard error variable, *SE*, to the constant-only regressions above. It is designed to capture the idea that publication bias introduces a systematic relationship between the effect size (*PCC*) and its standard error (*SE*):

(7) $PCC_i = \alpha_0 + \alpha_1 SE_i + \varepsilon_i.$

The FAT consists of testing the significance of *SE*. Rejection of $H_0: \alpha_1 = 0$ is taken as evidence that publication bias exists. Note that if we add *SE* to equations (3') and (4'), we obtain:

(3'') $\frac{PCC_i}{SE_i} = \alpha_0 \cdot \left(\frac{1}{SE_i}\right) + \alpha_1 + \frac{\varepsilon_i}{SE_i}$.

and

(4'') $\frac{PCC_i}{\omega_i} = \alpha_0 \cdot \left(\frac{1}{\omega_i}\right) + \alpha_1 \cdot \left(\frac{SE_i}{\omega_i}\right) + \frac{\varepsilon_i}{\omega_i}.$

It is standard practice when the meta-analysis sample consists of multiple estimates from the same study to correct for non-independence of the error terms by using cluster robust standard errors.

Inclusion of the *SE* variable also serves to control for the influence of publication bias on the estimate of mean true effect, α_0. Thus, the estimate of α_0 in the specification of equation (7) represents the bias-adjusted estimate of the mean true effect of social capital on health. A test of the significance of $\hat{\alpha}_0$ is known as the Precision Effect Test (PET). Rejection of $H_0: \alpha_0 = 0$ is taken as evidence that the mean true effect of social capital on health is nonzero.

TABLE 6 reports the FAT/PET results, with the FAT results reported on the first row and the PET results on the second. The first four columns report the various combinations of "Fixed Effects"/"Random Effects" and weighting by individual estimate("Weight1")/weighting by study("Weight2"). Across all four columns, we reject $H_0: \alpha_1 = 0$ at the 1 percent level of significance, indicating the existence of publication bias. The positive coefficient indicates positive publication bias, suggesting sample selection that favors the publication of positive estimates of the effect of social capital on health.

In three of the four cases we also reject $H_0: \alpha_0 = 0$, with the corresponding estimates of α_0 significant at the 1 percent level. The exception is the "Fixed Effects(Weight1)" regression. Thus, the PET results generally support the finding that social capital is positively and significantly related to health. However, the sizes of the coefficient estimates indicate that this effect is very small. Bias-adjusted estimates of the mean true effect of social capital on health range from 0.004 to 0.022, substantially below the value that Doucouliagos (2011) identifies as being "small".

Columns (5) and (6) in TABLE 7 report the weighted average estimates of mean true effect, uncorrected for publication bias, using the "Random Effects(Weight1)" and "Random Effects(Weight2)" estimators. The associated estimates are 0.024 and 0.032, which are close to the unweighted value of 0.028 reported in TABLE 3. These fall to 0.013 and 0.022, respectively, when *SE* is added to the specification to control for publication bias. Thus, while publication bias positively inflates estimates of the effect of social capital on health, it does not

inflate them very much.

VI. DATA ANALYSIS: Part 2

Meta-regression analysis. The preceding section has been concerned with estimating the mean true effect of social capital on health. In this section, we investigate factors that affect the size of that effect. To do that, we add potential moderator variables $X_k, k = 1,2, \dots K$, into the specification of equation (7):

(8) $$PCC_i = \alpha_0 + \alpha_1 SE_i + \sum_{k=1}^{K} \alpha_{k+1} X_{ki} + \varepsilon_i.$$

The coefficient α_{k+1} measures the change in the effect of social capital on health due to X_k, where a positive coefficient indicates that studies/regressions that have characteristic X_k estimate a larger effect of social capital on health. The specification of equation (8) is known as a meta-regression.

Given the large number of study, data, and estimation characteristics included in our dataset (cf. TABLE 4), we are concerned that multicollinearity may disguise significant relationships. Accordingly, we adopt a model selection algorithm to select a "best" specification. We use a backwards stepwise procedure that is designed to select the model specification with the smallest Bayesian Information Criterion value (Lindsey & Sheather, 2010). In the first round, all the variables are included in the regression equation: *SE*, *PubYear, IndividualSC, Panel, EastAsia, WestNorthEurope, HighIncome, OtherCountry, PhysicalHealth, MentalHealth, SelfReported, NumberSCVariables, Age, Gender, Education, MaritalStatus, Income, OLS, ORHazard, HLM, IV, SENonspherical, tNormal, and tCalculatedbypValue.* At each subsequent round, the algorithm drops the variable that causes the largest decrease in BIC. It continues to do that, one variable at a time, until the BIC can no longer be reduced. We then re-estimate the final, best model in order to obtain cluster robust standard errors.[14]

[14] We encountered a problem when using the stepwise regression algorithm with the "Random Effects" estimators. Note that there is no constant term in the weighted specification of equation (4''), as the constant term in the

We do this for each of the four estimation procedures ("Fixed Effects(Weight1)", "Fixed Effects(Weight2)", "RandomEffects(Weight1)", and "RandomEffects(Weight2)". We forced five variables to be retained in each step of the selection process: two variables to indicate the type of health outcome, *PhysicalHealth* and *MentalHealth,* with the omitted category being general health; a variable indicating that the respective social capital variable was individual-level as opposed to a community average (*IndividualSC*); and two variables that represent attempts to deal with endogeneity, *Panel* and *IV*.

The results are reported in TABLE 7. The publication bias term *SE* is positive and statistically significant at the 1 percent level in two of the four estimation procedures.[15] However, the estimated coefficients suggest that the size of the bias is small.[16] Thus, while the social capital and health literature may be affected by publication bias, it is of little practical consequence.

Neither of the two health outcome variables are consistently significant. *PhysicalHealth* is significant at the 5-percent level in two of the regressions (*FixedEffects-Weight1, RandomEffects-Weight1)*. *MentalHealth* never achieves significance. This confirms the casual observation from FIGURE 2 regarding the similarity of the *PCC* distributions for the three different health outcomes. Nevertheless, to be cautious, our subsequent analysis of the social capital variables will -- in addition to pooling the *PCC* values -- also divide the full sample into subsamples based on the nature of the health outcomes.

In contrast, we find strong evidence that social capital variables based on individual-level measures are more effectual for positive health outcomes than community average measures.

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original equation is divided by ω_i. The Stata program that we used, *vselect*, does not allow one to drop the constant term. Our workaround was to estimate the "best" model with a constant term, and then estimate that same variable specification, but without the constant term. Note that was not a problem for the "Fixed Effects" estimators, because the publication bias variable, *SE*, reduces to the constant term when divided through by *SE* (see equation 3''). In this case, the "constant" term is actually the coefficient on *SE*, α_1.

[15] The *SE* variable is dropped by the selection algorithm for the *Random Effects(Weight2)* regression.

[16] Doucouliagos & Stanley (2013) determine that "the literature suffers from substantial selectivity" if the estimated publication bias coefficient has an absolute value greater than 1. Note that the *SE* coefficient is less than 1 in all three of the regressions in which it appears.

However, while the *IndividualSC* estimates are significant at the 1-percent level in all four regressions, the sizes of the estimated differences in *PCC* are small, ranging between 0.013 and 0.020.

We find weak evidence that correcting for endogeneity diminishes the estimated effect of social capital on health. The coefficients for *Panel* are consistently negative across the four regressions. While significant at the 5-percent level in every case, the sizes of the estimated coefficients are small. The *IV* estimates are even weaker -- very small in size and never attaining significance at even the 10-percent level.

As we proceed to the other variables included in the meta-regressions of TABLE 7, we limit our discussion to those that are significant at the 5 percent level in at least three of the four regressions. The coefficient for *WestNorthEurope* is positive, indicating that social capital is estimated to have a larger effect on health for residents of Western and Northern Europe compared to other countries. The negative coefficient on *NumberSCVariables* indicates that the estimated effect of a given social capital variable tends to be smaller when more social capital variables are included in the regression. This is to be expected because the estimated effect variable, *PCC,* is a function of the *t*-statistic in the original study. One would expect that the more social capital variables there are in the equation, the more likely collinearity will reduce the significance of any given social capital variable.

The last variable in our meta-regression specification is *tCalculatedBypValue*. For approximately one-fifth of the estimated effects in our sample, the only information related to coefficient standard errors reported by the original study was stars; e.g. three stars to indicate significance at the 1 percent level, two stars to indicate significance at the 5 percent level, and so on. In our original coding, we assigned p-values for each number of stars.[17] When the coefficient was insignificant and no stars were reported, we set the p-value equal to 0.50.

[17] See Footnote #4.

The negative coefficient estimates for *tCalculatedBypValue* suggest that this was not a good approximation. Estimated effects calculated from the resulting *t*-values were significantly lower than those calculated following standard procedures. Further, compared to the other effects for binary variables reported in TABLE 7, the coefficient sizes are relatively large in absolute value, ranging from -0.023 to -0.034. The takeaway from this is that our estimates of the unconditional mean true effect are downwardly biased by the inclusion of estimated effects using these *t*-values. However, even after compensating for this, the estimated mean true effect fails to reach the 0.07 value that Doucouliagos identifies as "small".

Examination by type of social capital. The last part of our analysis carries out a closer examination of the social capital variables included in our sample. The top panel of TABLE 8 provides some statistical detail about the different kinds of social capital variables that studies have employed. Most studies in our sample use social capital variables that fit within the cognitive-structural framework. Of the 12,778 estimates of the effects of social capital on health in our sample, 11,776 use social capital variables that can either be classified as cognitive or structural – approximately 92%. Of these, the great majority are structural: 69.4%, versus 30.6% that are cognitive.

In contrast, only 3,572 of the estimated effects fit within the bonding–bridging–linking framework. Indeed, many of these can be cross-classified as either cognitive or structural. The breakdown for the bonding-bridging-linking social capital variables are 62.0% bonding, 34.2% bridging, and only 3.9% linking.

The bottom panel of TABLE 8 reports that there are 3,603 estimated effects that are based on a cognitive social capital variable; 8,173 effects are based on a structural social capital variable; 2,213 are based on a bonding social capital variable; and so on. For each subsample, we calculate the mean, unweighted *PCC* value unadjusted for publication bias. Mean *PCC* values for the *Structural* = 1, *Bonding* = 1, and *Bridging* = 1 subsamples are 0.022, 0.023, and 0.025,

respectively -- very close to the mean value of 0.028 for the entire sample. In contrast, the *Cognitive* = 1 and *Linking* = 1 subsamples are different, with the *Cognitive* subsample showing a higher (0.041), and the *Linking* subsample a lower (0.010) mean *PCC* value.

To investigate these differences further, we employ the same meta-regression analysis we used in TABLE 7, only this time we add social capital variables.[18] In the first set of exercises, we add *Cognitive*, so that the omitted social capital variable is *Structural*. In the next set of exercises, we add *Bonding* and *Bridging* for the full sample analysis, with *Linking* being the omitted variable; and *Bonding* for the subsample analysis, with *Bridging* and *Linking* being the omitted variables[19] We then implement the backwards stepwise regression procedure described above.

TABLE 9 displays the results for the *Cognitive/Structural* framework. In the interests of brevity and to focus attention, we only report estimated coefficients for the social capital variables. The top panel uses the full sample of 11,776 estimates that are derived from either cognitive or structural social capital variables. Across all four regressions, the coefficient for *Cognitive* is positive and statistically significant at the 1 percent level. This indicates that, in general, estimates of social capital on health that rely on cognitive social capital variables will find larger effects than those that rely on structural social capital variables.

The next three panels stratify the sample by the three health outcomes: physical health, mental health, and general health. The *Cognitive* coefficient is significant twice (at at least the 5-percent level) in the general health subsample, three times in the physical health subsample, and in all four of the regressions for the mental health subsample. However, the estimated values are still small by Doucouliagos' (2011) standards.

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[18] Another difference is that we do not force any variables into the equation other than a constant term and the respective social capital variables. In the weighted regressions of equations (3') and (4'), the "constant term" is actually the slope coefficient on the $\left(\frac{1}{SE_i}\right)$ and $\left(\frac{1}{\omega_i}\right)$ terms, respectively. We forced these variables into the equation so that there would always be a constant term in the final regression.

[19] We could not add both *Bonding* and *Bridging* in the subsample analysis because there were too few observations of *Linking.*

The last panel of TABLE 9 again uses the full sample, but includes interaction terms for cognitive social capital and physical health (*Cognitive*Physical*), and cognitive social capital and mental health (*Cognitive*Mental*). The omitted health category is *GeneralHealth*. The coefficients should be interpreted as follows: The coefficient on *Cognitive* represents the difference between the mean estimated effect of cognitive social capital on general health, and the mean estimated effect of structural social capital on any kind of health. The coefficient on *Cognitive*Physical* (or *Cognitive*Mental*) represents the difference between the mean estimated effect of cognitive social capital on physical health (or mental health) compared to the mean estimated effect of structural social capital on any kind of health.

Across the four regressions in TABLE 9, only the coefficient for *Cognitive*Mental* is consistently significant at the 5 percent level. The mean partial correlation of cognitive social capital and health is estimated to be approximately 0.02 to 0.03 larger than the mean partial correlation of structural social capital on any kind of health. Together with the previous results, these estimates indicate that cognitive social capital is particularly salient for mental health, as opposed to physical or general health.

TABLE 10 performs a similar set of exercises for the *Bonding/Bridging/Linking* social capital variables. The top panel pools all the estimates that are based on these social capital variables. We include dummy variables for *Bonding* and *Bridging* social capital variables. With two exceptions (*Bonding/Random Effects-Weight2; Bridging/Random Effects-Weight2)*, the associated coefficients are all insignificant at the 5-percent level. Further, when we test the joint hypothesis that both coefficients equal zero, we fail to reject it in three of the four regressions. These results suggest that there is little difference between bonding, bridging, and linking social capital variables with respect to their effect on health.

The next three panels break the full sample into three subsamples based on health outcomes. Due to the small number of estimates that use linking social capital variables in each

of the subsamples, we are forced to combine linking and bridging social capital as a single omitted category, so that the only included social capital variable is *Bonding.* Across the three subsamples, *Bonding* is significant only once, in column (4) for the physical health subsample. When the three subsamples are combined again in the bottom panel, with interaction terms for *Bonding* and *Physical Health*, and *Bonding* and *Mental Health*, the respective interaction terms are everywhere small in size and statistically insignificant. This supports the finding from the top panel in TABLE 10 that there is no systematic difference in how bonding, bridging and linking social capital affect health.

TABLE 11 provides one last deep dive into the finer categories of the social capital and health variables. Reported are the number of estimates associated with the respective subcategory (Column 2), the mean *PCC* value for those estimates (Column 3), and the percent of associated *t*-statistics that are (i) less than -2.00, (ii) between -2 and 2, and (iii) greater than 2.00 (Columns 3-5). Not a single subcategory of social capital or health rises to the level of "small" using Doucouliagos' *PCC* (2011) guidelines. The social capital sub-categories with the largest mean *PCC* values are *Social trust* (0.045) and *Perceived reciprocity* (0.053). With only two exceptions (*Social trust* and *Loneliness*), over half of all estimated coefficients are statistically insignificant ($-2.00 \leq t \leq 2.00$). This is all the more noteworthy given the large sizes of the samples underlying these estimates (cf. TABLE 3).

VII. CONCLUSION

The relationship between social capital and health has received extensive attention in fields such as public health, medicine, epidemiology, gerontology and other health-related disciplines. Hundreds of studies have reported that social capital is significantly associated with a variety of positive health outcomes. In contrast, the economics literature on this subject is relatively small.

The primary aim of this study has been to determine whether the effect of social capital on health is "large" or "small". If "large", then the dearth of economic research in this area

represents a gap that should be addressed. If "small", then this justifies the relative lack of attention this subject has received in the health economics discipline.

We use meta-analysis to analyze the effects of 12,778 estimates from 470 studies. To place estimated effects on a common scale, we transform the individual estimates into partial correlation coefficients and compare these against the "size" guidelines established by Doucouliagos (2011). Our analysis finds that social capital is significantly related to a variety of positive health outcomes. However, the effects are consistently very small. This result is robust across a wide variety of different types of social capital (e.g., cognitive, structural, bonding, bridging, linking), and for many different measures of health outcomes (e.g., mortality, disease/illnesses, depression).

One of the most compelling findings from our analysis is that the majority of estimates in the social capital/health literature are statistically insignificant. This is striking given the typically large sample sizes of the studies included in our analysis: Median sample size for the studies in our sample was 3,451. Over 75% of the estimates came from regressions with more than 1,000 observations. Large sample sizes with high rates of statistical insignificance are indicative of small effect sizes.

How could such a large literature grow from such small effects? We suspect several factors are responsible. First, estimated coefficients are difficult to interpret. Social capital variables are typically measured using Likert scale type questions. As a result, small effect sizes are not recognized as such. Second, and perhaps as a consequence of the first factor, researchers place undue emphasis on statistical significance. This is compounded by the fact that studies typically include multiple social capital variables in the same regression. The mean number of social capital variables in a regression in our sample was 6.5. The combination of large sample sizes with multiple social capital variables makes it relatively easy to find at least one social capital variable that is statistically significant. Studies rarely apply Bonferroni-type corrections

when assessing the statistical significance of multiple estimates. This explains how most studies can conclude that social capital has a significant effect on health, even while the majority of estimates are insignificant.

Our study is the most extensive analysis of the extant empirical literature on social capital and health ever undertaken. As such, it can be viewed as establishing a Bayesian prior belief that the effect of social capital on health is very small. The results suggest that economics is justified in not following the leads of other disciplines in devoting substantial research resources to the study of social capital and health.

Our study also calls into question initiatives to promote health through social capital such as those by the WHO, the OECD, and US Healthy People 2020 (Rocco & Suhrcke, 2012; Centers for Disease Control and Prevention, 2010; Keeley, 2007). In a recent systematic review, Villalonga-Olives, Wind, & Kawachi (2018) note the "enthusiasm of policy makers to implement social capital interventions to manipulate health outcomes for the better" (p. 217). Our results suggest that this enthusiasm may not translate into meaningful improvements in public health.

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TABLE 1
Common Measures of Health

Measure	Percent
PHYSICAL HEALTH (4,770 observations)	
Mortality	51.2
Disease/Illness	17.4
Self-Reported	20.9
MENTAL HEALTH (2,934 observations)	
Depression	28.2
Self-Reported	90.9

NOTE: Authors' calculations. Percent values are either percent of total Physical Health observations or percent of total Mental Health observations.

TABLE 2
Common Types of Social Capital

Measure	Percent
COGNITIVE / STRUCTURAL (11,776 observations)	
Cognitive (What people *feel*)	
Social trust	13.2
Perceived social support	7.3
Perceived social cohension	2.0
Perceived reciprocity	1.5
Sense of belonging	1.5
Loneliness	1.0
Structural (What people *do*)	
Social participation	16.7
Social networks	16.4
Social support	6.7
Social engagement	6.5
Volunteering	4.8
Group membership	4.6
Social integration	3.9
Social relationship	3.5
BONDING / BRIDGING / LINKING (3,572 observations)	
Bonding (Horizontal ties between similar people)	62.0
Bridging (Horizontal ties between dissimilar people)	34.2
Linking (Vertical ties between different people)	3.9

NOTE: Authors' calculations. Percent values are either percent of total Cognitive/Structural observations or percent of total Bonding/Bridging/Linking observations.

TABLE 3
Descriptive Statistics for Estimated Effects and t-statistics

	t-Statistics		*df*		*PCC Values*	
	Full	***Truncated***	***Full***	***Truncated***	***Full***	***Truncated***
Mean	1.67	1.60	29,023	29,600	0.030	0.028
Median	1.20	1.20	3,300	3,451	0.019	0.019
Minimum	-59.67	-17.44	5	5	-0.747	-0.156
Maximum	850.00	48.86	2,442,948	2,442,948	0.998	0.360
Std. Dev.	8.40	3.53	201,648	203,663	0.084	0.059
1%	-4.91	-4.37	39	49	-0.157	-0.113
5%	-2.66	-2.58	190	244	-0.061	-0.055
10%	-1.75	-1.63	412	462	-0.033	-0.031
90%	4.70	4.67	23,153	23,153	0.102	0.097
95%	7.16	7.02	44,986	44,986	0.143	0.132
99%	15.34	14.60	271,642	1,358,932	0.361	0.239
Obs	13,040	12,778	13,040	12,778	13,040	12,778

NOTE: The truncated sample is obtained from the Full Sample by deleting observations having the top and bottom 1% of *PCC* values.

TABLE 4
Description of Variables

Variable	*Description*	*Mean*	*Min*	*Max*
	STUDY TYPE			
Journal	=1, if study is a journal article	0.977	0	1
PubYear	Year study was published/appeared	2009.3	1985	2017
	DATA CHARACTERISTICS			
IndividualSC	=1, if estimate based on individual-level social capital variable	0.865	0	1
Panel	=1, if estimate based on panel data	0.444	0	1
*Cross-sectional**	=1, if estimate based on cross-sectional data	0.556	0	1
	COUNTRIES			
EastAsia	=1, if country studied is located in East Asia	0.204	0	1
*USA**	=1, if country studied is USA	0.209	0	1
WestNorthEurope	=1, if country studied is located in Western or Northern Europe	0.339	0	1
HighIncome	=1, if country studied is a high income country not included above	0.094	0	1
OtherCountry	=1, if country studied is none of the above	0.155	0	1
	HEALTH MEASURE			
PhysicalHealth	=1, if health variable measures physical health	0.373	0	1
MentalHealth	=1, if health variable measures mental health	0.230	0	1
*GeneralHealth**	=1, if health variable measures overall health	0.404	0	1
SelfReported	=1, if health variable is self-reported	0.679	0	1
	SOCIAL CAPITAL MEASURE			
CognitiveStructural	=1, if social capital is cognitive or structural	0.922	0	1
BondBridgeLink	=1, if social capital is bonding, bridging, or linking	0.280	0	1
NumberSCVariables	Number of social capital variables included in the regression	6.49	1	28

Variable	*Description*	*Mean*	*Min*	*Max*
	CONTROL VARIABLES			
Age	=1, if an age variable is included in the regression	0.852	0	1
Gender	=1, if a gender variable is included in the regression	0.845	0	1
Education	=1, if an education variable is included in the regression	0.599	0	1
MaritalStatus	=1, if a marital status variable is included in the regression	0.389	0	1
Income	=1, if an income variable is included in the regression	0.403	0	1
	ESTIMATION METHOD			
OLS	=1, if estimation method is OLS	0.139	0	1
ORHazard	=1, if estimation method is Odds Ratio or Hazards Ratio	0.552	0	1
HLM	=1, if estimation method is Hierarchical Linear Modelling	0.179	0	1
*FGLS**	=1, if estimation method is FGLS	0.017	0	1
*ProbitLogit**	=1, if estimation method is Probit or Logit	0.053	0	1
*OrderedProbitLogit**	=1, if estimation method is Ordered Probit or Logit	0.031	0	1
*IV**	=1, if estimation method is Instrumental Variables	0.019	0	1
*OtherEstimation**	=1, if estimation method is none of the above	0.010	0	1
SENonspherical	=1, if standard error estimation assumes nonspherical errors	0.255	0	1
	CALCULATION OF t –STATISTIC			
tNormal	=1, if t-statistic is calculated as ratio of coefficient to standard error	0.215	0	1
tCalculatedBypValue	=1, if t-statistic is calculated from p-value	0.178	0	1
*tCalculatedByCI**	=1, if t-statistic is calculated from confidence interval	0.606	0	1

NOTE*:* When the grouped variables include all possible categories, the categories omitted in the subsequent analysis (the benchmark categories) are indicated by an asterisk.

TABLE 5
Study Weights

	Fixed Effects	***Random Effects***
Mean	0.21%	0.21%
Median	0.01%	0.23%
1%	0.0001%	0.0467%
5%	0.0003%	0.0959%
10%	0.0007%	0.1399%
90%	0.1622%	0.2576%
95%	0.3251%	0.2607%
99%	1.9023%	0.2636%
Maximum	45.2%	0.26%
Top 3	67.4%	0.79%
Top 10	77.3%	2.63%
Studies	470	470

NOTE: The methodology for calculating "study weights" is described in Footnote #11 in the text.

TABLE 6
The Funnel Asymmetry Test (FAT) and Precision Effect Test (PET)

	Including Publication Bias Term				***Excluding Publication Bias Term***	
	Fixed Effects (Weight1) (1)	***Fixed Effects (Weight2) (2)***	***Random Effects (Weight1) (3)***	***Random Effects (Weight2) (4)***	***Random Effects (Weight1) (5)***	***Random Effects (Weight2) (6)***
FAT	1.269*** (5.93)	1.491*** (6.98)	0.599*** (5.33)	0.551*** (4.34)	----	----
PET	0.004 (1.45)	0.007*** (2.87)	0.013*** (5.15)	0.022*** (7.49)	0.024*** (12.79)	0.032*** (16.17)
Observations	12,778	12,778	12,778	12,778	12,778	12,778

NOTE: The FAT and PET results in Columns (1) through (4) come from estimating α_1 and α_0, respectively, in equation (7) in the text using Weighted Least Squares (WLS). The four WLS estimators (*Fixed Effects-Weight1, Fixed Effects-Weight2, Random Effects-Weight1,* and *Random Effects-Weight2*) are described in Section II of the text. The PET results in Columns (5) and (6) are taken from estimates of equation (4'). All of the estimation procedures calculate cluster robust standard errors. *, **, and *** indicate statistical significance at the 10-, 5-, and 1-percent level, respectively.

TABLE 7
Meta-Regression Analysis

Variables	*Fixed Effects (Weight1) (1)*	*Fixed Effects (Weight2) (2)*	*Random Effects (Weight1) (3)*	*Random Effects (Weight2) (4)*
SE	0.468*** (3.22)	0.641*** (4.77)	---	---
PhysicalHealth	-0.008** (-2.37)	-0.003* (-0.59)	-0.012*** (-3.61)	-0.006 (-0.90)
MentalHealth	-0.006* (-1.95)	-0.004 (-0.99)	-0.006* (-1.93)	-0.003 (-0.72)
IndividualSC	0.013*** (4.84)	0.020*** (5.50)	0.015*** (3.62)	0.018*** (4.52)
Panel	-0.006** (-2.41)	-0.006** (-2.23)	-0.014*** (-4.52)	-0.012*** (-2.85)
IV	-0.000 (-0.06)	0.002 (0.50)	-0.007 (-1.38)	-0.000 (-0.04)
PubYear	----	-0.000 (-1.15)	----	-0.000 (-1.37)
EastAsia	0.006 (1.60)	0.006* (1.69)	----	0.006 (1.12)
WestNorthEurope	0.009*** (2.91)	0.010*** (3.07)	0.009*** (2.66)	0.007 (1.59)
HighIncome	0.003 (0.78)	0.006 (1.44)	----	0.006 (0.81)
OtherCountry	----	----	-0.005 (-1.24)	----
SelfReported	0.007** (2.18)	0.015*** (2.97)	----	0.010 (1.61)
NumberSCVariables	-0.001*** (-3.92)	-0.002*** (-4.73)	-0.001*** (-3.93)	-0.002*** (-5.38)
Age	-0.003* (-1.96)	----	-0.004 (-1.06)	----
Education	-0.002* (-1.74)	-0.004* (-1.92)	-0.006** (-2.09)	-0.010** (-2.44)

Variables	*Fixed Effects (Weight1) (1)*	*Fixed Effects (Weight2) (2)*	*Random Effects (Weight1) (3)*	*Random Effects (Weight2) (4)*
MaritalStatus	-0.003 (-1.36)	-0.005** (-2.08)	----	-0.004 (-1.11)
OLS	0.006 (1.02)	0.006 (0.86)	----	0.007 (0.98)
HLM	----	0.006 (1.51)	---	---
SENonspherical	-0.003 (-1.10)	-0.007 (-1.66)	-0.007** (-2.11)	-0.006 (-1.46)
tNormal	-0.010*** (-3.47)	-0.011** (-3.61)	----	-0.007 (-1.61)
tCalculatedBypValue	-0.023*** (-7.72)	-0.027*** (-6.93)	-0.023*** (-6.39)	-0.034*** (-6.58)
Adjusted R-squared	0.206	0.226	0.275	0.302
Hypothesis Test: ***Physical = Mental = 0***	*F = 3.15* *(p = 0.044)*	*F = 0.53* *(p = 0.59)*	*F = 7.13* *(p = 0.001)*	*F = 0.48* *(p = 0.621)*
Observations	12,778	12,778	12,778	12,778

NOTE: The table reports the results of estimating equation (8) in the text. The top value in each cell is the coefficient estimate, and the bottom value in parentheses is the associated *t*-statistic. The variables *PhysicalHealth, MentalHealth, IndividualSC, Panel,* and *IV* were locked into each regression specification. Remaining control variables were selected using a backwards stepwise regression procedure that chooses variables to minimize the Bayes Information Criterion. The four WLS estimators (*Fixed Effects-Weight1, Fixed Effects-Weight2, Random Effects-Weight1,* and *Random Effects-Weight2*) are described in Section II. All four estimation procedures calculate cluster robust standard errors. *, **, and *** indicate statistical significance at the 10-, 5-, and 1-percent level, respectively. The hypothesis test reports the results of testing whether there is no difference in mean *PCC* values for the three different health outcomes after controlling for the effects of other variables.

TABLE 8
Social Capital Variables

A. Sample Statistics for Social Capital Variables

Type of Social Capital	*Obs*	*Mean*	*Min*	*Max*
Cognitive	11,776	0.306	0	1
Structural	11,776	0.694	0	1
Bonding	3,572	0.620	0	1
Bridging	3,572	0.342	0	1
Linking	3,572	0.039	0	1

NOTE: Values in the table report sample statistics of the social capital variables for two sets of observations. The first set of 11,776 observations consist of estimates on health of social capital variables using the cognitive/structural framework. The second set of 3,572 observations consist of estimates based on the bonding/bridging/linking framework.

B. Distribution of PCC Values by Type of Social Capital

Type of Social Capital	*Obs*	*Mean*	*Min*	*Max*
Cognitive = 1	3,603	0.041	-0.155	0.360
Structural = 1	8,173	0.022	-0.157	0.360
Bonding = 1	2,213	0.023	-0.157	0.347
Bridging = 1	1,220	0.025	-0.150	0.234
Linking = 1	139	0.010	-0.073	0.308

NOTE: Values in the table report conditional mean, minimum, and maximum values of the *PCC* variable for observations satisfying the condition in the leftmost column.

TABLE 9
Meta-Regression Analysis: Cognitive/Structural

Variables	*Fixed Effects (Weight1) (1)*	*Fixed Effects (Weight2) (2)*	*Random Effects (Weight1) (3)*	*Random Effects (Weight2) (4)*
		FULL SAMPLE		
Cognitive	0.012***	0.013***	0.011***	0.017***
	(4.54)	(4.03)	(3.65)	(4.00)
Observations	11,776	11,776	11,776	11,776
		PHYSICAL HEALTH = 1		
Cognitive	0.012**	0.014***	0.011**	0.016*
	(2.02)	(3.91)	(2.41)	(1.96)
Observations	4,421	4,421	4,421	4,421
		MENTAL HEALTH = 1		
Cognitive	0.024***	0.028***	0.026***	0.028***
	(4.14)	(4.01)	(4.41)	(3.40)
Observations	2,638	2,638	2,638	2,638
		GENERAL HEALTH = 1		
Cognitive	0.011***	0.011***	0.005	0.007
	(3.72)	(3.42)	(1.15)	(1.60)
Observations	4,810	4,810	4,810	4,810

Variables	*Fixed Effects (Weight1) (1)*	*Fixed Effects (Weight2) (2)*	*Random Effects (Weight1) (3)*	*Random Effects (Weight2) (4)*
		FULL SAMPLE with Interaction Terms		
Cognitive	0.010*** (3.55)	0.008** (2.30)	0.005 (1.18)	0.008* (1.69)
Cognitive*Physical	-0.001 (-0.17)	0.004 (0.45)	0.005 (0.85)	0.009 (0.85)
Cognitive*Mental	0.015** (2.22)	0.024** (2.26)	0.022*** (3.27)	0.028*** (2.91)
Observations	11,776	11,776	11,776	11,776

NOTE: The table reports the results of estimating equation (8) in the text for different samples of estimates, using different estimation procedures. Only the coefficient estimates for the social capital variables are reported. The top value in each cell is the coefficient estimate, and the bottom value in parentheses is the associated *t*-statistic. In addition to a constant term, the variable *Cognitive* was locked into the regressions in the first four sets of regressions (*Full Sample, Physical Health = 1, Mental Health = 1, General Health* = 1). The last set of regressions (*Full Sample with Interaction Terms)* also locked in the interaction terms *Cognitive*Physical* and *Cognitive*Mental.* The remaining control variables were selected using a backwards stepwise regression procedure that chooses variables to minimize the Bayes Information Criterion. The four WLS estimators (*Fixed Effects-Weight1, Fixed Effects-Weight2, Random Effects-Weight1,* and *Random Effects-Weight2*) are described in Section II. All four calculate cluster robust standard errors. *, **, and *** indicate statistical significance at the 10-, 5-, and 1-percent level, respectively.

TABLE 10
Meta-Regression Analysis: Bonding/Bridging/Linking

Variables	*Fixed Effects (Weight1) (1)*	*Fixed Effects (Weight2) (2)*	*Random Effects (Weight1) (3)*	*Random Effects (Weight2) (4)*
		FULL SAMPLE		
Bonding	0.002 (0.80)	0.002 (1.42)	0.010* (1.74)	0.017** (2.38)
Bridging	0.000 (0.04)	-0.000 (-0.07)	0.011* (1.84)	0.016** (2.45)
H_o: Bonding = Bridging = 0	*F = 0.32* *p-value = 0.724*	*F = 1.02* *p-value = 0.362*	*F = 1.81* *p-value = 0.166*	*F = 3.33* *p-value = 0.038*
Observations	3,572	3,572	3,572	3,572
		PHYSICAL HEALTH = 1		
Bonding	-0.001 (-0.50)	0.004 (1.43)	0.009 (1.50)	0.015** (2.31)
Observations	1,023	1,023	1,023	1,023
		MENTAL HEALTH = 1		
Bonding	0.008 (0.96)	0.003 (0.24)	0.005 (0.81)	-0.002 (-0.37)
Observations	1,004	1,004	1,004	1,004

Variables	*Fixed Effects (Weight1) (1)*	*Fixed Effects (Weight2) (2)*	*Random Effects (Weight1) (3)*	*Random Effects (Weight2) (4)*
		GENERAL HEALTH = 1		
Bonding	0.001 (0.10)	0.003 (0.37)	-0.005 (-1.13)	0.000 (0.08)
Observations	1,597	1,597	1,597	1,597
		FULL SAMPLE with Interaction Terms		
Bonding	-0.001 (-0.15)	0.005 (0.64)	-0.006 (-1.50)	0.004 (0.56)
Bonding*Physical	0.004 (0.47)	-0.003 (-0.38)	0.014* (1.79)	0.003 (0.28)
Bonding*Mental	0.003 (0.56)	-0.009 (-0.83)	0.010 (1.35)	-0.007 (-0.68)
Observations	3,572	3,572	3,572	3,572

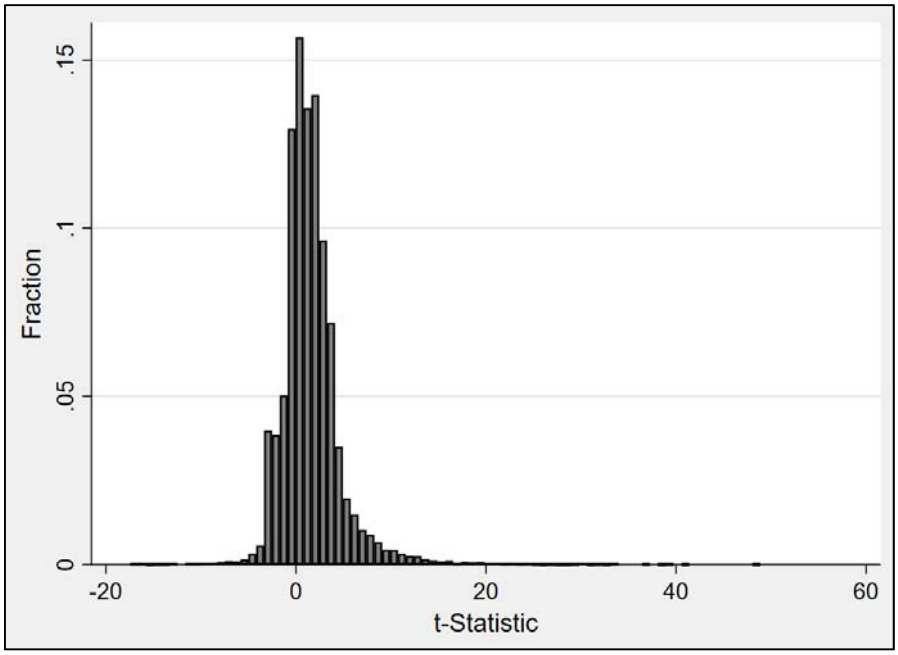
NOTE: The table reports the results of estimating equation (8) in the text for different samples of estimates, using different estimation procedures. Only the coefficient estimates for the social capital variables are reported. The top value in each cell is the coefficient estimate, and the bottom value in parentheses is the associated *t*-statistic. In addition to a constant term, the following variables were locked into the regressions: *Bonding* and *Bridging* for the set of *Full Sample* regressions; *Bonding* for the set of *Physical Health = 1, Mental Health = 1, General Health* = 1 regressions; and *Bonding, Bonding*Physical* and *Bonding*Mental* for the *Full Sample with Interaction Terms* set of regressions. The remaining control variables were selected using a backwards stepwise regression procedure that chooses variables to minimize the Bayes Information Criterion. The four WLS estimators (*Fixed Effects-Weight1, Fixed Effects-Weight2, Random Effects-Weight1,* and *Random Effects-Weight2*) are described in Section II. All four calculate cluster robust standard errors. *, **, and *** indicate statistical significance at the 10-, 5-, and 1-percent level, respectively. The hypothesis test in the *Full Sample* set of regressions reports the results of testing whether there is any difference in the estimated effects on health for the *Bonding, Bridging,* and *Linking* social capital variables.

TABLE 11
Mean PCC Values and the Distribution of t-values for Finer Categories of Social Capital and Health

	Obs	*Mean PCC*	*Distribution of t-values*		
			t < -2.00	*-2.00 ≤ t ≤ 2.00*	*t > 2.00*
	(1)	*(2)*	*(3)*	*(4)*	*(5)*
Sub-categories of Cognitive Social Capital					
Social trust	1,553	0.045	3.0	40.9	56.1
Perceived social support	863	0.038	5.0	62.1	32.9
Perceived social cohesion	237	0.043	3.8	61.2	35.0
Perceived reciprocity	179	0.053	0.6	60.9	38.5
Sense of belonging	173	0.038	4.6	53.8	41.6
Loneliness	114	-0.010	28.9	48.2	22.8
Sub-categories of Structural Social Capital					
Social participation	1,965	0.034	5.3	51.3	43.3
Social networks	1,931	0.015	9.7	68.8	21.5
Social support	785	0.020	8.1	69.8	22.0
Social engagement	769	0.019	5.7	57.5	36.8
Volunteering	567	0.024	4.4	51.8	43.7
Group membership	537	0.022	9.7	61.4	28.9
Social integration	456	0.007	15.6	57.9	26.5
Social relationship	411	0.020	17.0	49.1	33.8
Sub-categories of Health					
Mortality	2,441	0.020	9.5	62.3	28.2
Disease/Illness	830	0.014	5.5	75.9	18.5
Depression	826	0.028	8.6	59.8	31.6

FIGURE 1
Distribution of t-and PCC Values

A. *t*-Statistics



Distribution of t-statistics	*Percent*
t < -2.00	7.2
-2.00 ≤ t ≤ 2.00	56.4
t > 2.00	36.3

B. PCC Values

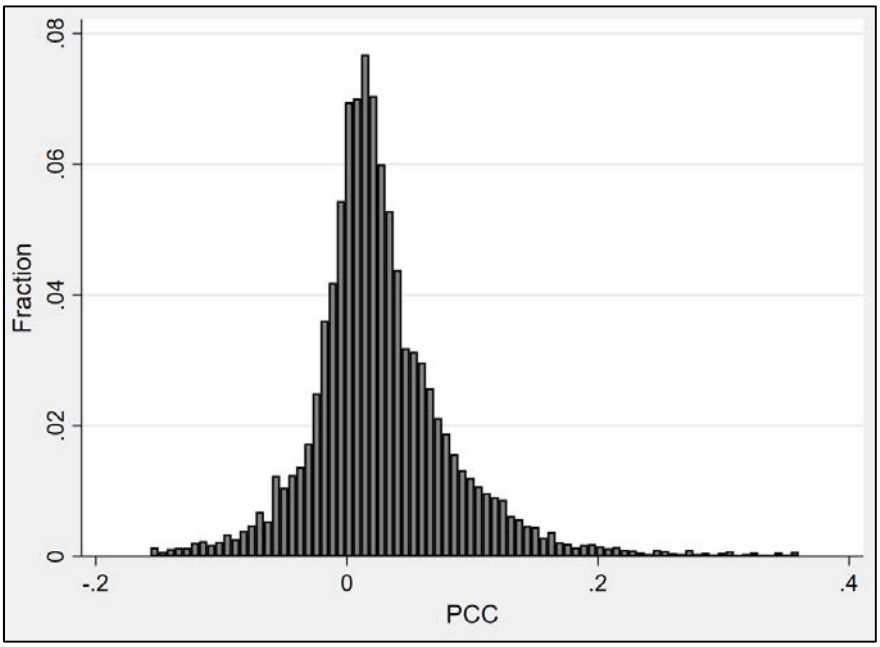
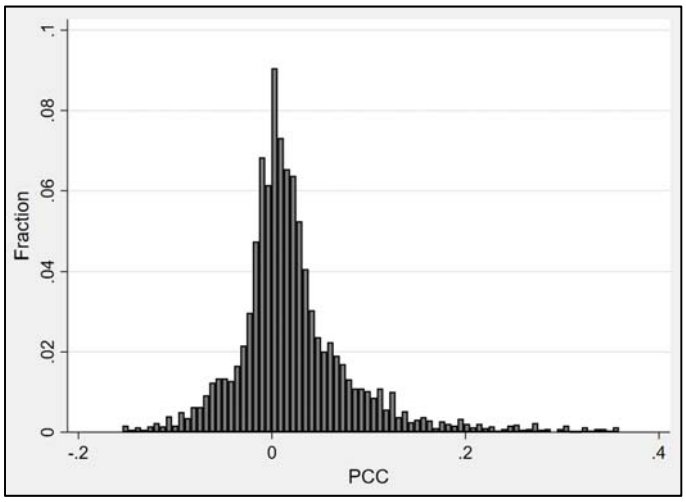
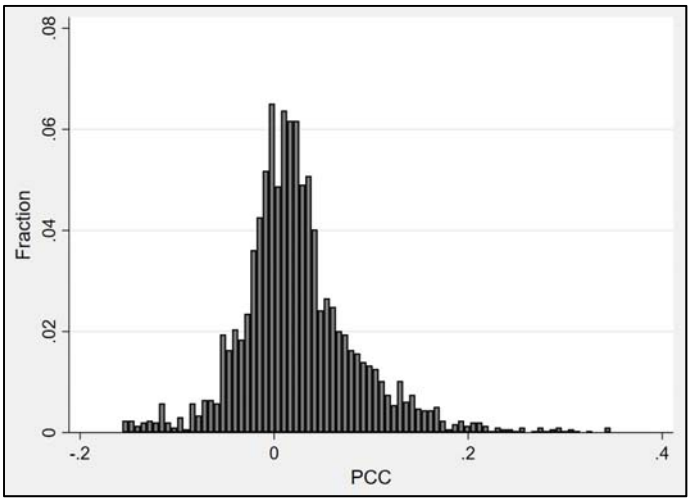


FIGURE 2
Distribution of PCC Values by Measure of Health

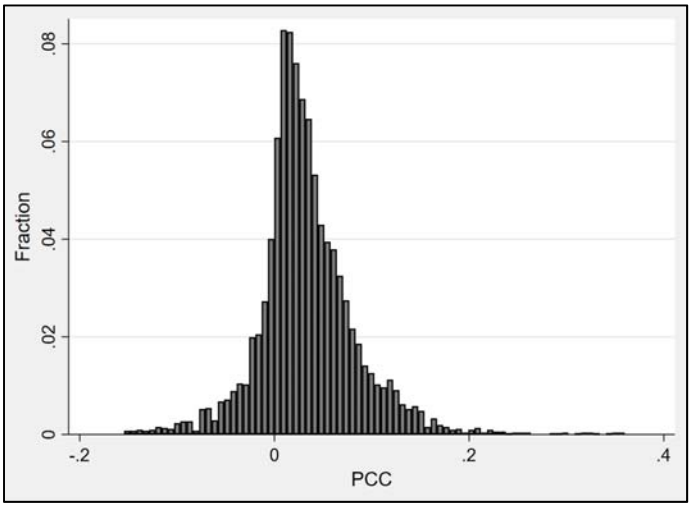
A. Physical Health



B. Mental Health



C. General Health



Subsample	Obs	Mean	Std. Dev.
Physical Health = 1	4,770	0.023	0.065
Mental Health = 1	2,934	0.025	0.062
General Health = 1	5,167	0.033	0.051