SOCIAL CAPITAL AND HEALTH: A META-ANALYSIS

by

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5 January 2019

Abstract

This study investigates the extensive empirical literature on social capital and health using meta-analysis. Our final sample consists of 12,459 estimated effects taken from 450 studies. Our main result is that the overall mean size of the effect of social capital on health is very small, though it is statistically significant. This low association follows from a relatively large share of individually insignificant estimates, combined with the large sample sizes that characterize this literature. Furthermore, despite an extensive theoretical literature concerned with delineating different kinds of social capital, we find few systematic empirical differences. While cognitive social capital has a significantly stronger association with health than structural social capital, especially for mental health, the difference is empirically minor. There is no evidence of significant differences between bonding, bridging, and linking social capital.

JEL CODES: B49, C49, I10, I31

KEYWORDS: Social capital, Health, Meta-analysis, Meta-regression, Partial correlation coefficient, Research synthesis, Mental health, Physical health, Self-reported health

Acknowledgements: Xindong Xue acknowledges financial support from the National Social Science Fund of China (Grant No.:14BRK013). W. Robert Reed acknowledges financial support from the Czech Science Foundation, Grant 18-02513S. We are grateful to Xiaoping Ruan, Lina Wang, Weilun Wu, Yi Xia, Junyong Yuan, and Kunpeng Zhao for their excellent research assistance. We thank Andrea Menclova and Tom Coupè for insightful comments and suggestions. All remaining errors are our own.

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I. INTRODUCTION

This study examines the extensive literature on social capital and health. Social capital has many definitions – Claridge (2004) lists twenty[1] – but a commonly cited definition is "features of social organization, such as trust, norms and networks that can improve the efficiency of society by facilitating coordinated actions" (Putnam, 1993, page 167). Its roots span a number of disciplines, including sociology (Bourdieu, 1986; Coleman, 1990), economics (Loury, 1992), and political science (Putnam, 1993).

While academic interest in the relationship between social capital and health is relatively recent, the literature has burgeoned, with hundreds of empirical studies appearing in academic journals. A casual review of this literature reveals mixed results. Some studies show a significant association between social capital and health (Borgonovi, 2010; Berry and Welsh, 2010; Petrou and Kupek, 2008), whereas others do not (Meng and Chen, 2014; Hurtado et al., 2011; D'Hombres et al., 2010; Snelgrove et al., 2009; Yip et al., 2007; Norstrand and Xu, 2012). Furthermore, there is a suggestion that different types of social capital have different effects across alternative measures of health (Goryakin et al., 2014; Yamaoka, 2008).

In this study, we aggregate the literature on social capital and health and provide answers to three questions: (i) What is the overall effect of social capital on health? (ii) What are some factors that explain why estimates differ across studies? (iii) Are some kinds of social capital more salient for health than others? To do that, we employ meta-analysis (Egger et al., 2008; Borenstein et al., 2009; Stanley & Doucouliagos, 2012).

Meta-analysis is a quantitative method for aggregating estimated effects across multiple studies that estimate a similar relationship. At its core, it is nothing more than a method for averaging estimates, though there exist multiple, alternative procedures for doing this. Meta-analysis is particularly useful when there is a large number of estimated effects that come from

[1] See https://www.socialcapitalresearch.com/literature/definition/.

studies using different samples, estimation procedures, and variable specifications.

While the literature on social capital and health has attracted previous attention from meta-analysts, the scope of these meta-analyses has been relatively narrow. De Silva et al. (2005) is the first “meta-analysis” that we could find on social capital and health. They aggregate results from 21 studies that examine social capital (structural and cognitive social capital) and various measures of mental health. Meta-analysis is in quotes because DeSilva et al. (2005) do not attempt to average estimates across studies, but rather categorize them. They write, “Differences in the measurement of social capital and mental illness, and the varied nature of the statistical techniques used by the studies made formal meta-analysis impossible" (page 621). Accordingly, they place studies in four categories, Negative relationship/Significant; Negative relationship/Insignificant; Positive relationship/Insignificant; and Positive relationship/Significant. They find mixed evidence that social capital reduces mental illness.

The most extensive meta-analysis done to date is Holt-Lunstad et al. (2010). They study the literature on social relationships and mortality, collecting estimated effects from 148 studies. They combine studies that measure mortality from all causes with studies that focus on mortality from cardio-vascular disease and cancer. A wide variety of social capital variables are used in these studies, including perceptions of the availability of support, feelings of isolation, marital status, size of social network, living alone, and degree of social isolation. Holt-Lunstad et al. (2010) calculate a weighted average of the estimates and find a substantial relationship between stronger social relationships and decreased mortality.

Gilbert et al. (2013) examine the relationship between social capital and two measures of health: self-reported health and all-cause mortality. They collect a total of 288 estimates from 39 studies. Social capital variables include measures of control over one’s life; social participation; reciprocal relationships; sense of community; social capital indices; social support; trust; and bonding, bridging, and linking social capital. Like Holt-Lunstad et al. (2010), they calculate a

weighted average of estimates and find that a “one unit increase in social capital increases the odds of good health by 27%” (page 1388). Given the heterogeneity in both health and social capital variables, it is unclear how to interpret a “one unit increase”.

Nyqvist et al. (2014) also examine the relationship between social capital and mortality. Their study is distinctive because they restrict themselves to all-cause mortality, and only examine cohort studies (as opposed to cross-sectional studies). As a result, they only include 20 studies in their sample. Some of the social capital variables they include are numbers of friends and relatives, measures of number of social contacts, categories of social networks, measures of social isolation, membership in groups, and participation in activities and organizations. They stratify their estimated effects into four groups -- those that involve social participation, social networks, social support, and trust – and calculate weighted averages of the estimated effects for each group. They generally find that social capital is inversely related to mortality.

The only other meta-analysis that we are aware of is Kuiper et al. (2015). They study the relationship between social relationships and dementia, measured by a variety of tests. Their meta-analysis consists of 19 studies and, like Nyqvist et al. (2014), is restricted to cohort studies. A wide variety of social capital variables are included: social network size; social participation; frequency of social contacts, loneliness; and satisfaction with social networks. Weighted averages of the estimated effects are calculated for different subsets of estimated effects, depending on the category of the social capital variable. They find mixed results concerning the various types of social capital and the incidence of dementia.

Our study improves on previous meta-analyses of social capital and health in three main ways. First, we provide a better method of averaging estimates across studies. As highlighted above, De Silva et al. (2005) note the challenge (“impossibility”) of combining effects based on disparate social capital and health measures. Previous meta-analyses average estimated effects that not only use different types of social capital, but different approaches to measuring them.

The problem is implicitly highlighted by Gilbert et al, (2013) when they speak of a "one unit increase in social capital." In fact, there is no standardized "unit" of social capital. For example, in calculating an overall effect for frequency of social contact, Kuiper et al. (2015) combine coefficient estimates across the following social capital variables: "Visiting children or other relatives (Never vs. At least weekly-monthly)"; "Visits, phone calls or mail from family and friends (Less than weekly vs. Daily)"; "Visits to friends or family members (No vs. Yes)"; "Contact with relatives or friends (No vs. Daily)"; "Visiting friends (No vs. Yes); "Join in family activities (No vs. Yes)"; "Visiting friends or relatives (No vs. Yes)"; and "Social support activities (High (tertile 3) vs. Low (tertile 1))". Averaging coefficient estimates from estimated effects that measure such different things is highly hazardous.

What is required is that the respective estimates be converted to a unitless measure, such as an elasticity. As detailed below, our approach is to follow the economics meta-analysis literature and convert coefficient estimates to partial correlation coefficients (PCCs). While multiple health and social capital variables complicate our analysis, their existence also creates value for an approach that is able to combine them in order to obtain an overall picture of the literature. PCCs allow us to do that.

Our second value-added is that the scope of our analysis greatly exceeds previous meta-analyses on this subject. Our final sample consists of 12,459 estimated effects from 450 studies. With respect to health, we include studies that estimate the relationship between social capital and mental health, social capital and physical health, and social capital and general health. We also include a wide variety of social capital variables: cognitive and structural; bonding, bridging, and linking; and others. We understand that bigger is not always better when combining estimates across different studies. Of utmost importance is that the respective estimates measure similar effects. Our analysis addresses this concern while demonstrating the benefits of combining estimates across this extensive literature.

Lastly, unlike previous meta-analyses on social capital and health, we record an extensive list of study, data, and estimation characteristics for each estimated effect. We then perform meta-regression analyses to identify factors that are systematically related to estimated effects across studies. Other improvements include using a variety of weighting procedures to average estimates, accounting for correlations between estimates using clustered standard errors, and providing formal Funnel Asymmetry Tests (FATs) for publication bias. We note that all the data and code to reproduce the results in this paper are publicly posted at an accompanying Open Science Framework (OSF) storage site.[2]

II. METHODOLOGY

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 (*SC)*, along with a set of control variables (Z_k):

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

Let $\hat{\beta}_{1i}$ be the effect estimated by study i, and let there be a total of *M* estimates produced by multiple studies.[3]

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] We note that we will post the data once our manuscript is accepted for publication. In the meantime, the code, output, and other relevant files can be found here: https://osf.io/z7xqs/.

[3] 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 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 the social capital was associaated with an increase in good health.

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

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.[4]

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 the more precise estimates. If all estimates come from a population with the same true effect, so that the only source of variation in ε_i is due to sampling error -- i.e., $\text{var}(\varepsilon_i) = (SE_i)^2$ -- 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) $\frac{\hat{\beta}_{1i}}{SE_i} = \alpha_0 \cdot \left(\frac{1}{SE_i}\right) + \frac{\varepsilon_i}{SE_i}$ $i = 1,2,\ldots,M.$

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. 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.

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

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 $\text{var}(\varepsilon_i) = \sqrt{(SE_i)^2 + \tau^2} = \omega_i$. 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 69. 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.

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.

The last 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.

III. 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".

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 government reports. The search was ended in September 2017.

Our preliminary search produced 588 papers.[5] We then 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 drop studies employing path analysis that

[5] See the Appendix for the corresponding PRISMA diagram.
[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.

use social capital as a mediator variable. Lastly, we exclude studies measuring the intergenerational effect of social capital on health; e.g., the effect of parent's social capital on child's health.

TABLE 1 reports the most common journal outlets in our sample. By far the most frequently appearing journal outlet is *Social Science & Medicine*, an interdisciplinary journal that publishes social science research on health. Approximately one out of every five articles included in our sample come from this journal. The next three journals in terms of frequency are public health journals (*Health & Place, Journal of Epidemiology and Community,* and *BMC Public Health*). Following these is a sociology journal (*Journal of Health and Social Behavior*), two more interdisciplinary journals (*Journal of Gerontology* and *PLOS ONE*), and an economics journal (*Health Economics*). This certifies the broad, cross-discipline coverage of research on social capital and health.[7]

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.). 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).

For studies reporting odds ratios or confidence intervals in discrete choice models, we

[7] Greater detail about the journals where the individual studies in our meta-analysis were published is provided in a file entitled "Journals" that is posted at the OSF storage site that accompanies this manuscript: https://osf.io/z7xqs/.

used the conversion formulae described above to calculate standard errors. We also categorized the estimation procedures used to produce each estimate. Binary variables were used to indicate the following methods: OLS, FGLS, probit/logit, ordered logit or probit, 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 effect runs from social capital and health, the literature does recognize that causation can go in the opposite direction. For this reason, our analysis will pay particular attention to estimates that 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.

TABLE 2 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.[8]

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.[9]

TABLE 3 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.[10]

[8] See Footnote #3 for how we were able to combine estimates where an increase in the health measure indicated bad health, with estimates where increases in the health measure indicated good health.

[9] Greater detail about the measures of health used by the studies in our meta-analysis is provided in a file entitled "Measures of Health" that is posted at the OSF storage site that accompanies this manuscript: https://osf.io/z7xqs/.

[10] Greater detail about the measures of social capital used by the studies in our meta-analysis is provided in a file entitled "Measures of Social Capital" that is posted at the OSF storage site that accompanies this manuscript: https://osf.io/z7xqs/.

IV. DATA ANALYSIS: Part 1

Our initial dataset consisted of 12,715 observations gleaned from 451 studies.[11] Calculation of *PCC* uses *t*-values and *df*, so the first two columns of TABLE 4 focus on these variables. The full sample of *t*-values has mean and median values of 1.64 and 1.16, 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 29,573 and 3,293, 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 0.028 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 12,459 observations. 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.[12] 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 4 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 0.026 and 0.019. Based on Doucouliagos (2011), these do not even get close to the threshold value of 0.07 that

[11] Bibliographic information for the 451 studies included in this meta-analysis is provided in a document entitled "Studies" that is posted at the OSF storage site that accompanies this manuscript: https://osf.io/z7xqs/.

[12] 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. The output for this robustness check is posted at the OSF storage site that accompanies this manuscript: https://osf.io/z7xqs/.

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 56.8% 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 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 important 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 results.[13] 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

[13] 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.

unlikely.

FIGURE 1 and TABLE 4 aggregate 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 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 5 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 studies drawn upon for this analysis were published in the window from 1985 to 2017 (*PubYear*), and almost all of them relied on individual-level data in obtaining their estimates (*Individual*). Somewhat less than half of the estimates in our sample used panel data (*Panel*), with the remainder relying on cross-sectional data. The studies in our sample covered 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 (40.5% of estimates), closely followed by physical health (37.7%) and then mental health (22.5%). 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 (92.7%). Another perspective, which includes bonding, bridging, and linking social capital, was less commonly employed (27.9%). 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 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. 85.8% of estimates came from a regression specification including an age variable; 84.7% included a gender variable; 60.4% included an education variable; 39.1% included a marital status variable; and 40.0% 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 (56.0%). The next two most common procedures were hierarchical linear modelling (18.2%) and ordinary least squares (13.6%). 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 small percent of estimates employed instrumental variable procedures (1.7%).

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. 25.6% of the estimates calculated standard errors assuming some form of nonspherical

error behavior.

Finally, as noted above, three different methods were used to derive *t*-values. In 20.0% 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 17.9% 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 (62.1%), the *t*-statistic was calculated using the reported confidence interval or from the log of the estimated odds ratio (*tCalculatedByCI*).

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 6 identifies our 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%.[14]

If the 100% weight was divided equally across studies, given 450 studies, each study would receive a weight of 0.22%. 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 45.5%.[15] The top 3 studies account for 67.8% of the total weight, and the top 10 studies comprise almost 77.8%. Thus the "Fixed Effects" estimate is highly skewed towards a very small number of select studies that have large *PCC* values and/or use a large number of observations (*df*). In contrast, as noted above, the "Random Effects" estimator weights estimates more uniformly. The median value is 0.24%, compared to a mean value of 0.22%. The maximum

[14] 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).

[15] This ID for this study is 177. It has 72 estimated effects of social capital on health, and the number of observations in the respective samples range from 2,306,760 to 2,442,948.

weight any single study receives is 0.28%, and the top 10 studies account for 2.79%. While we report both "Fixed Effects" and "Random Effects", our preferred estimator is the latter.

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, informal tool for identifying publication bias is the "funnel plot". The funnel plot graphs estimated effect sizes (here, *PCC* values) against their respective standard errors (*SE*). A common representation is the inverted funnel shape of FIGURE 3. On the horizontal axis is the *PCC* value, and on the vertical axis is its standard error (*SE*). Note that the value at the top of the vertical axis is zero, so that *SE* values increase as one moves down the vertical axis. The vertical line extending upwards from the horizontal axis identifies the sample mean *PCC*, and the inverted V identifies the 95% confidence interval for *PCC* values that is estimated to arise from sampling error. For a given *SE* value, the associated confidence interval is given by a horizontal band extending from the left side of the 95% region to the right side. As the *SE* increases, the length of the band likewise increases, so that it is narrow at the top of the funnel and wide at the bottom.

If there was one true population effect of social capital on health, so that the only reason estimates differed across studies was due to sampling error, then one would expect a symmetric scatter of *PCC-SE* dots around the vertical line, with tight clustering towards the top of the graph (where the *SE* was small), and spreading out as one moved downwards on the graph (where *SE* values were larger). At least this is the pattern one would expect in the absence of publication bias.

Publication bias introduces asymmetry to the funnel plot. For samples characterized by

large error variance, sampling error will produce a wide range of effect estimates. This creates an opportunity for researchers to selectively report estimates that are larger in absolute size, and hence more likely to get published. This can be intentional on the part of the researcher. But it can also occur unintentionally, as sampling error causes some researchers to get large and significant estimates, while others get small and insignificant estimates. The former get published and the latter do not, generating publication bias in the literature, and introducing asymmetry to the funnel plot.

Possible evidence of publication bias is observable in FIGURE 3. The top panel plots the *PCC*-*SE* pairs for the truncated sample of 12,459 individual estimates, while the bottom panel calculates mean *PCC* and *SE* values for each study, so that each point represents a single study. In both cases, a slight positive tilt can be observed as the standard error increases.

A more formal test 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 it 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. The intuition underlying this can be gathered from FIGURE 3. Publication bias arises because sampling error creates opportunities for selective reporting of estimates. But when the standard error diminishes to zero, that opportunity vanishes. This situation is represented in FIGURE 3 by the tip of the funnel plot's inverted V. In the context of equation (7), this corresponds to *SE* taking on the value 0, so that the equation specification simplifies to the constant term. 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 7 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 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 0.031, which are close to the unweighted value of 0.026 reported in TABLE 4. These fall to 0.014 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.

V. 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 include 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 5), 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, 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.[16]

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: the publication bias variable, *SE*; variables to indicate the type of health outcome, *PhysicalHealth* and *MentalHealth,* with the omitted category being general health; and two variables that represent attempts to deal with endogeneity, *Panel* and *IV.*

The results are reported in TABLE 8. Across all four estimation procedures, the publication bias term is positive and statistically significant at the 1 percent level. The fact that *SE* continues to be statistically significant after controlling for other variables indicates that the FAT results from TABLE 7 are not a spurious outcome generated by omitted effects. It confirms the evidence that the social capital/health literature is influenced by publication bias.

Neither of the two health outcome variables are consistently significant. *PhysicalHealth* is significant at the 5-percent level in the *FixedEffects(Weight1)* and *RandomEffects(Weight1)* regressions. *MentalHealth* is only significant in the *RandomEffects(Weight1)* regression. 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

[16] 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 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.

the social capital variables will -- in addition to pooling the *PCC* values -- also divide the full sample into subsamples based on health outcomes.

We find weak evidence that correcting for endogeneity systematically affects estimates of social capital on health. While the estimated coefficients for *Panel* are consistently negative across the four regressions, indicating that correcting for endogeneity in this manner produces smaller estimated effects, the associated coefficients are significant at the 5-percent level in only two of the four regressions, and 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 8, 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 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.

Given the importance of the *t*-statistic in determining *PCC,* we also included a variable to see whether alternative methods for computing coefficient standard errors had an effect on *PCC* values. The negative coefficient for *SENonspherical* indicates that allowing for nonspherical behavior in the calculation of the standard error – for example, to adjust for heteroskedasticity or cluster effects – tends to lower *PCC* values. This is consistent with the associated standard error estimates being larger than those that assume spherical errors, which would serve to lower *t*-values, and hence *PCC* values.

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. As noted above, we assigned p-values for each number of stars. When the coefficient was insignificant and no stars were reported, we set the p-value equal to 0.50.

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 8, the coefficient sizes are relatively large in absolute value, ranging from -0.020 to -0.030. 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 even the 0.07 value that Doucouliagos identifies as "small".

Examination of the individual social capital variables. 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 9 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 12,459 estimates of social capital on health in our sample, 11,557 use social capital variables that can either be classified as cognitive or structural – approximately 93%. Of these, the great majority are structural: 69.4%, versus 30.6% that are cognitive.

In contrast, only 3,480 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 61.5% bonding, 34.7%

bridging, and only 3.8% linking.

The bottom panel of TABLE 9 reports that there are 3,532 estimated effects that are based on a cognitive social capital variable; 8,025 effects are based on a structural social capital variable; 2,141 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 0.021, 0.023, and 0.025, respectively -- very close to the mean value of 0.026 for the entire sample. In contrast, the *Cognitive* = 1 and *Linking* = 1 subsamples are different, with the *Cognitive* subsample showing a higher (0.039), and the *Linking* subsample a lower (0.009) mean *PCC* value.

To investigate these differences further, we employ the same MRA procedure we used in TABLE 8, only this time we add social capital variables.[17] 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[18] We then implement the backwards stepwise regression procedure described above.

TABLE 10 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 11,557 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.

[17] 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.

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

The next three panels break out the sample by the three health outcomes: physical health, mental health, and general health. The *Cognitive* coefficent is significant at the 5-percent level only once in the physical and general health subsamples. In contrast, it is significant at the 1-percent level in all four of the regressions for the mental health subsample. However, the estimated values are still small by Doucouliagos' (2011) standards.

The last panel of TABLE 10 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 10, 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 11 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. In none of the regression are either of these coefficient significant at the 5-percent level. Further, when we test the joint hypothesis that both coefficients equal zero, we fail to reject it every time.

These results are consistent with there being no 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 confirms the finding from the top panel in TABLE 11, that there is no systematic difference in how bonding, bridging and linking social capital affect health.

VI. CONCLUSION

The last twenty years have seen an explosion of research on social capital and health. Hundreds of articles have been written on the subject. It continues to be a very active research field (Folland & Nauenberg, 2018) and the subject regularly appears in policy documents by international organizations such as the WHO and the OECD (Keeley, 2007; Rocco & Suhrcke, 2012). However, because the concept spans heterogeneous fields such as sociology, public health, economics, political science, and epidemiology; and because there is no generally agreed definition of what social capital is, let alone how to measure it; it is difficult to quantitatively summarize this literature.

This study addresses this challenge. It investigates the extensive empirical literature on social capital and health using meta-analysis. Our final sample consists of 12,459 estimated effects taken from 450 studies. Our main finding is that while we find that social capital is significantly related to health, the size of the effect is very small. This result follows directly from

the relatively large percentage of insignificant estimates in the literature, combined with the large samples typical in studies of social capital and health.

Furthermore, despite an extensive theoretical literature concerned with delineating different kinds of social capital, we find few systematic differences in the effects that different kinds of social capital have on health. We generally estimate insignificant differences between the effects of bonding, bridging, and linking social capital on health. And while we see some evidence of statistically significant differences between cognitive and structural social capital, these are small in terms of economic significance.

This study represents the most comprehensive attempt to date to analyze the extensive and disparate literature on social capital and health. It reveals that the norm is for social capital to have a relatively minor effect on health. Therefore, a potentially fruitful line of future research is to better understand why a subset of studies are able to estimate large effects while most studies do not. Despite the substantial efforts expended on researching the relationship between social capital and health, and the extensive interest in this subject from policymakers and international health organizations, there remains much uncertainty about what "works" and what does not when it comes to social capital and health. It is hoped that this study will stimulate further efforts in this direction.

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TABLE 1
Common Journal Outlets

Journal	*Percent*
Social Science & Medicine	20.2
Health & Place	3.9
Journal of Epidemiology and Community	3.5
BMC Public Health	3.3
Journal of Health and Social Behavior	2.8
Journal of Gerontology	2.0
PLOS ONE	2.0
Health Economics	1.7
Research on Aging	1.7
Social Indicators Research	1.7
International Journal of Epidemiology	1.5
Journal of Gerontology: Social Sciences	1.5
Ageing & Society	1.3
Journal of Aging and Health	1.3
Psychosomatic Medicine	1.3
Quality of Life Research	1.3

NOTE: Authors' calculations. Further information is provided in the file "Journals" posted at the OSF storage site accompanying this paper (https://osf.io/z7xqs/).

TABLE 2
Common Measures of Health

Measure	Percent
Physical Health	
Mortality	55.0
Disease/Illness	16.6
General	8.0
Self-Reported	21.9
Mental Health	
Depression	29.3
General	22.2
Self-Reported	89.4

NOTE: Authors' calculations. Further details are provided in the file "Measures of Health" posted at the OSF storage site accompanying this paper (https://osf.io/z7xqs/).

TABLE 3
Common Types of Social Capital

Measure	Percent
COGNITIVE / STRUCTURAL	
Cognitive (what people *feel*)	
Social trust	12.2
Perceived social support	7.1
Perceived social cohesion	2.0
Perceived reciprocity	1.4
Sense of belonging	1.4
Loneliness	0.9
Perceived social isolation	0.5
Structural (What people *do*)	
Social participation	15.6
Social networks/ties	14.7
Social support	6.2
Social engagement/contacts	6.1
Volunteering	4.4
Organization (association/group) membership	4.2
Social integration/connections	3.6
Social relationships	3.1
Voting	0.9
Mixed (Cognitive+Structural)	5.7
BONDING / BRIDGING / LINKING	
Bonding (Horizontal ties between similar people)	17.1
Bridging (Horizontal ties between dissimilar people)	9.6
Linking (Vertical ties between different people)	1.1

NOTE: Authors' calculations. Further details are provided in the file "Measures of Social Capital" posted at the OSF storage site accompanying this paper (https://osf.io/z7xqs/).

TABLE 4
Descriptive Statistics for Estimated Effects and t-statistics

	t-Statistics		*df*		*PCC Values*	
	Full	***Truncated***	***Full***	***Truncated***	***Full***	***Truncated***
Mean	1.64	1.56	29,573	30,159	0.028	0.026
Median	1.16	1.16	3,293	3,451	0.019	0.019
Minimum	-59.67	-17.44	5	5	-0.747	-0.158
Maximum	850.00	48.86	2,442,948	2,442,948	0.998	0.334
Std. Dev.	8.49	3.50	204,176	206,220	0.081	0.057
1%	-4.96	-4.47	39	82	-0.159	-0.113
5%	-2.68	-2.58	208	254	-0.062	-0.055
10%	-1.82	-1.65	434	468	-0.034	-0.031
90%	4.68	4.63	23,153	23,327	0.099	0.095
95%	7.17	6.98	44,986	44,986	0.138	0.128
99%	15.48	14.45	1,358,932	1,358,932	0.336	0.217
Obs	12,715	12,459	12,715	12,459	12,715	12,459

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

TABLE 5
Description of Variables

Variable	*Description*	*Mean*	*Min*	*Max*
	STUDY TYPE			
Journal	=1, if study is a journal	0.977	0	1
PubYear	Year study was published/appeared	2009.4	1985	2017
	DATA CHARACTERISTICS			
Individual	=1, if estimate based on individual-level data	0.980	0	1
Panel	=1, if estimate based on panel data	0.444	0	1
*Cross-sectional**	=1, if estimate based on cross-sectional data	0.554	0	1
	COUNTRIES			
EastAsia	=1, if country studied is located in East Asia	0.203	0	1
*USA**	=1, if country studied is USA	0.211	0	1
WestNorthEurope	=1, if country studied is located in Western or Northern Europe	0.337	0	1
HighIncome	=1, if country studied is high income country not included above	0.092	0	1
OtherCountry	=1, if country studied is none of the above	0.157	0	1
	HEALTH MEASURE			
PhysicalHealth	=1, if health variable measures physical health	0.377	0	1
MentalHealth	=1, if health variable measures mental health	0.225	0	1
*GeneralHealth**	=1, if health variable measures overall health	0.405	0	1
SelfReported	=1, if health variable is self-reported	0.676	0	1
	SOCIAL CAPITAL MEASURE			
CognitiveStructural	=1, if social capital is cognitive or structural	0.927	0	1
BondBridgeLink	=1, if social capital is bonding, bridging, or linking	0.279	0	1
NumberSCVariables	Number of SC variables included in the regression	6.52	1	28

Variable	*Description*	*Mean*	*Min*	*Max*
	CONTROL VARIABLES			
Age	=1, if an age variable is included in the regression	0.858	0	1
Gender	=1, if a gender variable is included in the regression	0.847	0	1
Education	=1, if an education variable is included in the regression	0.604	0	1
MaritalStatus	=1, if a marital status variable is included in the regression	0.391	0	1
Income	=1, if an income variable is included in the regression	0.400	0	1
	ESTIMATION METHOD			
OLS	=1, if estimation method is OLS	0.136	0	1
ORHazard	=1, if estimation method is Odds Ratio or Hazards Ratio	0.560	0	1
HLM	=1, if estimation method is Hierarchical Linear Modelling	0.182	0	1
*FGLS**	=1, if estimation method is FGLS	0.017	0	1
*ProbitLogit**	=1, if estimation method is Probit or Logit	0.048	0	1
*OrderedProbitLogit**	=1, if estimation method is Ordered Probit or Logit	0.031	0	1
*IV**	=1, if estimation method is Instrumental Variables	0.017	0	1
*OtherEstimation**	=1, if estimation method is none of the above	0.009	0	1
SENonspherical	=1, if standard error estimation assumes nonspherical errors	0.256	0	1
	CALCULATION OF t –STATISTIC			
tNormal	=1, if t-statistic is calculated as ratio of coefficient to standard error	0.200	0	1
tCalculatedBypValue	=1, if t-statistic is calculated from p-value	0.179	0	1
*tCalculatedByCI**	=1, if t-statistic is calculated from confidence interval	0.621	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 6
Study Weights

	Fixed Effects	***Random Effects***
Mean	0.22%	0.22%
Median	0.01%	0.24%
1%	0.0001%	0.0628%
5%	0.0003%	0.0961%
10%	0.0008%	0.1383%
90%	0.1633%	0.2713%
95%	0.3350%	0.2752%
99%	1.9149%	0.2787%
Maximum	45.5%	0.28%
Top 3	67.8%	0.84%
Top 10	77.8%	2.79%
Studies	450	450

NOTE: The methodology for calculating "study weights" is described in Footnote #14 in the text.

TABLE 7
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	1.227*** (5.71)	1.425*** (6.76)	0.549*** (4.79)	0.473*** (3.86)	----	----
PET	0.004 (1.45)	0.007*** (3.08)	0.014*** (5.11)	0.022*** (7.88)	0.024*** (12.44)	0.031*** (15.94)
Observations	12,459	12,459	12,459	12,459	12,459	12,459

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 8
Meta-Regression Analysis

Variables	*Fixed Effects (Weight1) (1)*	*Fixed Effects (Weight2) (2)*	*Random Effects (Weight1) (3)*	*Random Effects (Weight2) (4)*
SE	0.559*** (3.80)	0.726*** (4.90)	0.522*** (4.66)	0.482*** (3.99)
PhysicalHealth	-0.011*** (-3.11)	-0.008* (-1.73)	-0.012*** (-3.73)	-0.007 (-1.26)
MentalHealth	-0.006 (-1.60)	-0.003 (-0.61)	-0.006** (-2.00)	-0.002 (-0.45)
Panel	-0.005** (-2.02)	-0.005* (-1.71)	-0.012*** (-3.67)	-0.007* (-1.80)
IV	-0.000 (-0.07)	0.001 (0.14)	-0.003 (-0.51)	0.001 (0.07)
PubYear	----	-0.000 (-0.96)	----	----
EastAsia	0.005* (1.73)	0.009** (2.29)	----	----
WestNorthEurope	0.006*** (2.79)	0.009*** (2.79)	0.011*** (2.93)	0.007* (1.77)
HighIncome	----	0.004 (0.88)	----	----
OtherCountry	----	----	-0.006 (-1.54)	----
SelfReported	0.008** (2.38)	0.015*** (3.28)	----	0.010* (1.84)
NumberSCVariables	-0.001*** (-2.99)	-0.002*** (-3.68)	-0.001** (-2.10)	-0.002*** (-3.92)
Age	-0.003* (-1.71)	----	----	0.007 (0.99)
Gender	----	0.003 (1.15)	----	----
Education	----	-0.003 (-1.34)	-0.006** (-2.03)	-0.011*** (-2.59)

Variables	*Fixed Effects (Weight1) (1)*	*Fixed Effects (Weight2) (2)*	*Random Effects (Weight1) (3)*	*Random Effects (Weight2) (4)*
MaritalStatus	-0.004* (-1.70)	-0.006** (-2.48)	----	----
Income	-0.002 (-1.11)	----	----	----
OLS	0.004 (0.67)	0.004 (0.59)	----	----
SENonspherical	-0.006** (-2.19)	-0.008*** (-2.93)	-0.010*** (-3.05)	-0.011*** (-2.76)
tNormal	-0.007** (-2.13)	-0.008** (-2.10)	0.004 (0.97)	----
tCalculatedBypValue	-0.020*** (-7.14)	-0.023*** (-6.84)	-0.022*** (-5.89)	-0.030*** (-6.81)
Adjusted R-squared	0.191	0.198	0.274	0.299
Hypothesis Test: ***Physical = Mental = 0***	*F = 4.86* *(p = 0.008)*	*F = 1.50* *(p = 0.22)*	*F = 7.53* *(p = 0.001)*	*F = 0.81* *(p = 0.447)*
Observations	12,459	12,459	12,459	12,459

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 *SE*, *PhysicalHealth*, *MentalHealth*, *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 9
Social Capital Variables

A. Sample Statistics for Social Capital Variables

Type of Social Capital	*Obs*	*Mean*	*Min*	*Max*
Cognitive	11,557	0.306	0	1
Structural	11,557	0.694	0	1
Bonding	3,480	0.615	0	1
Bridging	3,480	0.347	0	1
Linking	3,480	0.038	0	1

NOTE: Values in the table report sample statistics of the social capital variables for two sets of observations. The first set of 11,557 observations consist of estimates on health of social capital variables using the cognitive/structural framework. The second set of 3,480 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	3,532	0.039	-0.158	0.328
Structural = 1	8,025	0.021	-0.158	0.331
Bonding = 1	2,141	0.023	-0.158	0.327
Bridging = 1	1,206	0.025	-0.150	0.234
Linking = 1	133	0.009	-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 10
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***	0.011***	0.011***	0.014***
	(4.03)	(3.43)	(3.65)	(3.66)
Observations	11,557	11,557	11,557	11,557
		PHYSICAL HEALTH == 1		
Cognitive	0.010*	0.010*	0.010**	0.011*
	(1.85)	(1.94)	(2.27)	(1.71)
Observations	4,387	4,387	4,387	4,387
		MENTAL HEALTH = 1		
Cognitive	0.025***	0.029***	0.028***	0.029***
	(4.49)	(4.01)	(5.19)	(3.53)
Observations	2,555	2,555	2,555	2,555
		GENERAL HEALTH = 1		
Cognitive	0.012***	0.005	0.003	0.006
	(3.53)	(1.60)	(0.74)	(1.17)
Observations	4,710	4,710	4,710	4,710

Variables	*Fixed Effects (Weight1) (1)*	*Fixed Effects (Weight2) (2)*	*Random Effects (Weight1) (3)*	*Random Effects (Weight2) (4)*
		FULL SAMPLE with Interaction Terms		
Cognitive	0.008*** (2.74)	0.004 (1.18)	0.004 (0.97)	0.006 (1.14)
Cognitive*Physical	0.003 (0.44)	0.009 (1.48)	0.006 (0.97)	0.005 (0.54)
Cognitive*Mental	0.019*** (2.63)	0.031*** (2.91)	0.023*** (3.43)	0.029*** (3.05)
Observations	11,557	11,557	11,557	11,557

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 11
Meta-Regression Analysis: Bonding/Bridging/Linking

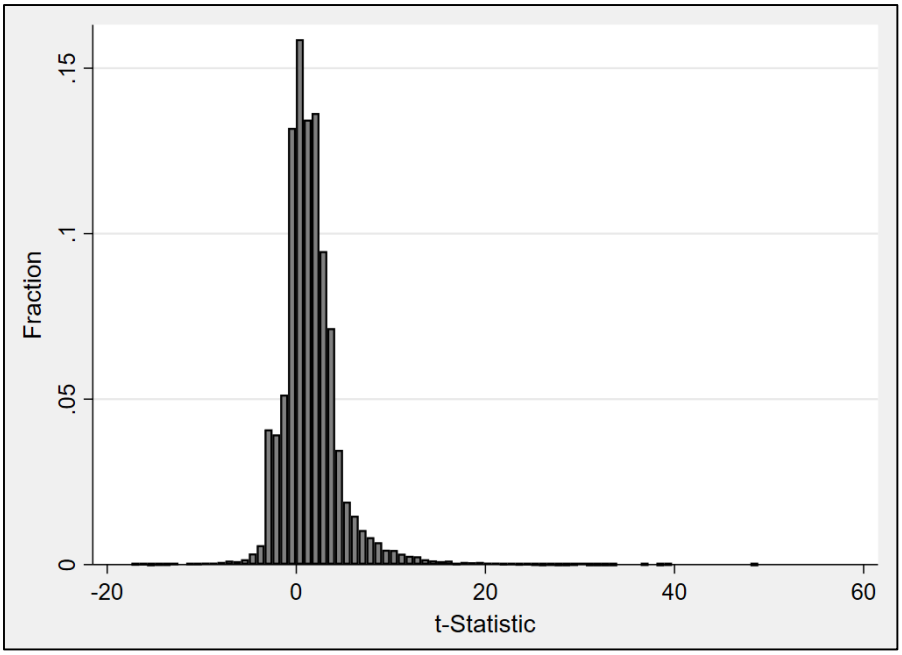
Variables	*Fixed Effects (Weight1) (1)*	*Fixed Effects (Weight2) (2)*	*Random Effects (Weight1) (3)*	*Random Effects (Weight2) (4)*
		FULL SAMPLE		
Bonding	0.003 (1.23)	0.007 (1.71)	0.011 (1.87)	0.013 (1.59)
Bridging	0.002 (0.39)	0.004 (0.83)	0.012 (1.82)	0.012* (1.79)
H_0: Bonding = Bridging = 0	*F = 0.99* *p-value = 0.372*	*F = 1.79* *p-value = 0.171*	*F = 1.93* *p-value = 0.148*	*F = 1.64* *p-value = 0.200*
Observations	3,480	3,480	3,480	3,480
		PHYSICAL HEALTH == 1		
Bonding	-0.002 (-0.54)	0.003 (1.09)	0.007 (1.05)	0.015** (2.22)
Observations	1,017	1,017	1,017	1,017
		MENTAL HEALTH = 1		
Bonding	0.008 (0.94)	-0.001 (-0.10)	0.007 (0.87)	0.001 (0.13)
Observations	934	934	934	934

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)	0.004 (0.44)	-0.005 (-1.05)	-0.001 (-0.14)
Observations	1,582	1,582	1,582	1,582
		FULL SAMPLE with Interaction Terms		
Bonding	-0.001 (-0.07)	0.010 (1.10)	-0.006 (-1.23)	0.003 (0.40)
Bonding*Physical	0.003 (0.46)	-0.012 (-0.92)	0.012 (1.54)	0.003 (0.25)
Bonding*Mental	0.001 (0.24)	-0.014 (-1.28)	0.014 (1.58)	-0.005 (-0.46)
Observations	3,480	3,480	3,480	3,480

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.

FIGURE 1
Distribution of t-and PCC Values

A. *t*-Statistics



Distribution of t-statistics	*Percent*
t < -2.00	7.4
-2.00 ≤ t ≤ 2.00	56.8
t > 2.00	35.7

B. PCC Values

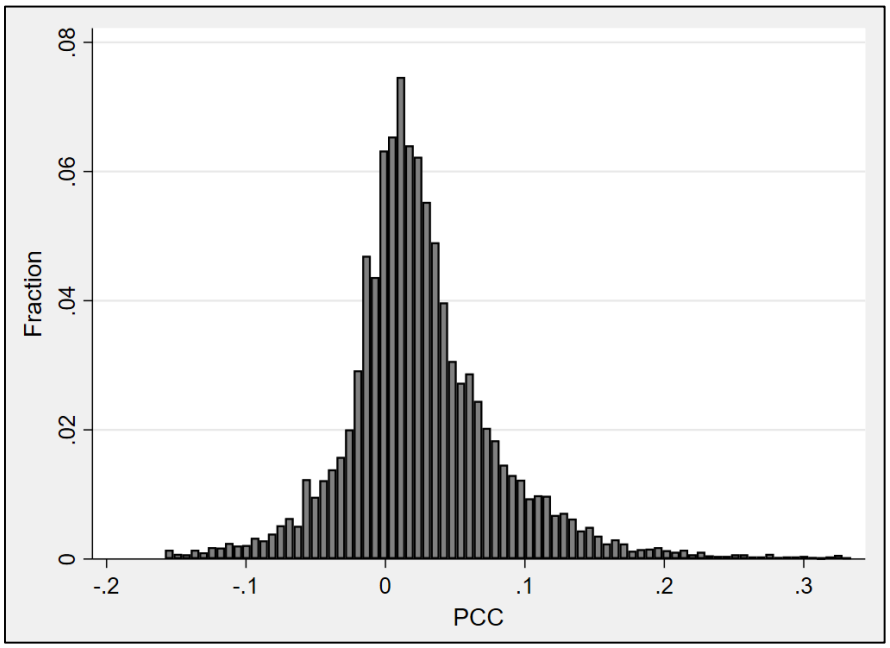
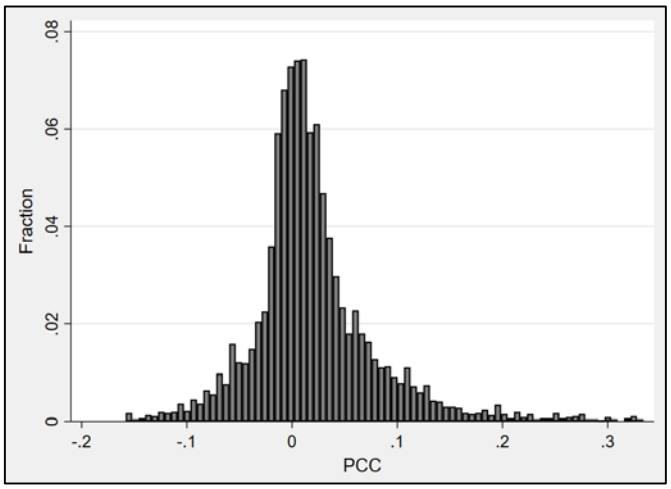
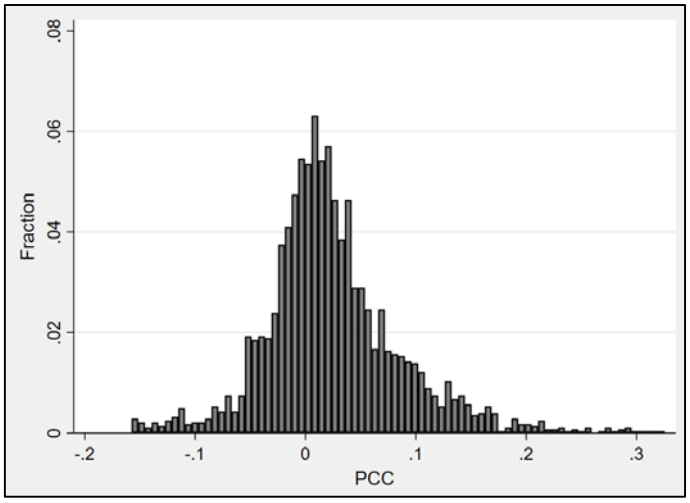


FIGURE 2
Distribution of PCC Values by Measure of Health

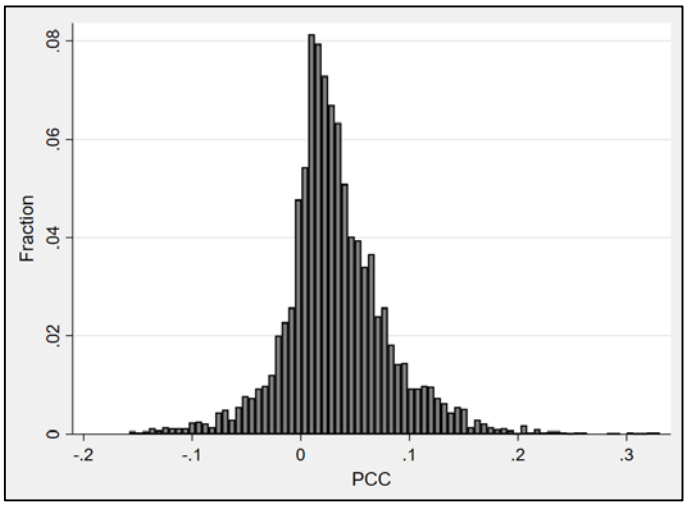
A. Physical Health



B. Mental Health



C. General Health

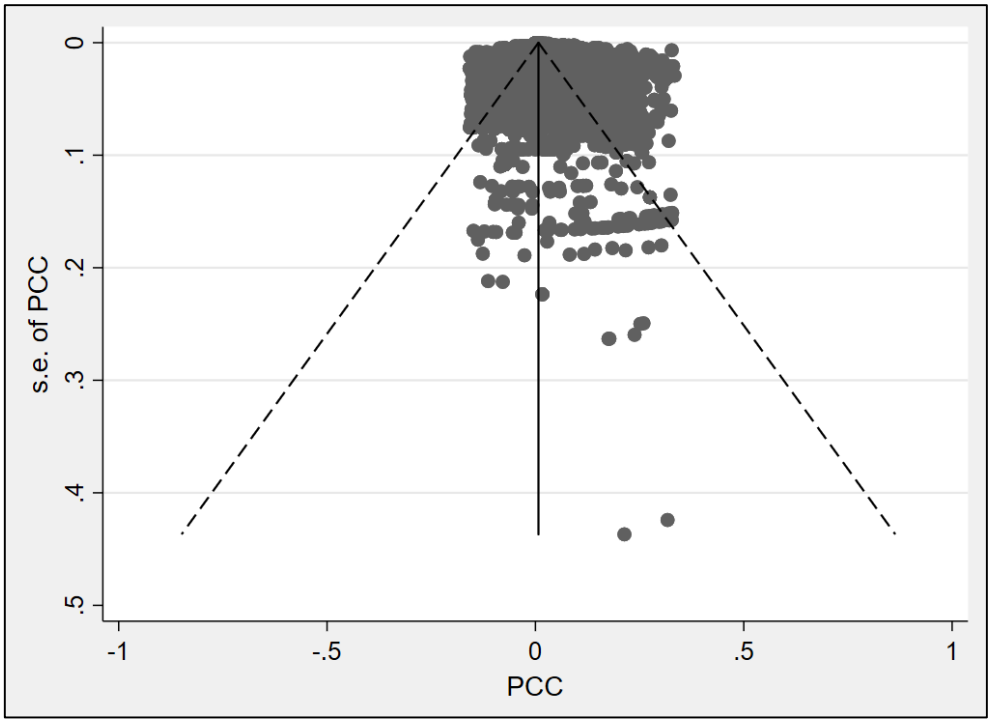


Subsample	Mean	Std. Dev.
Physical Health == 1	0.020	0.060
Mental Health == 1	0.025	0.063
General Health == 1	0.032	0.051

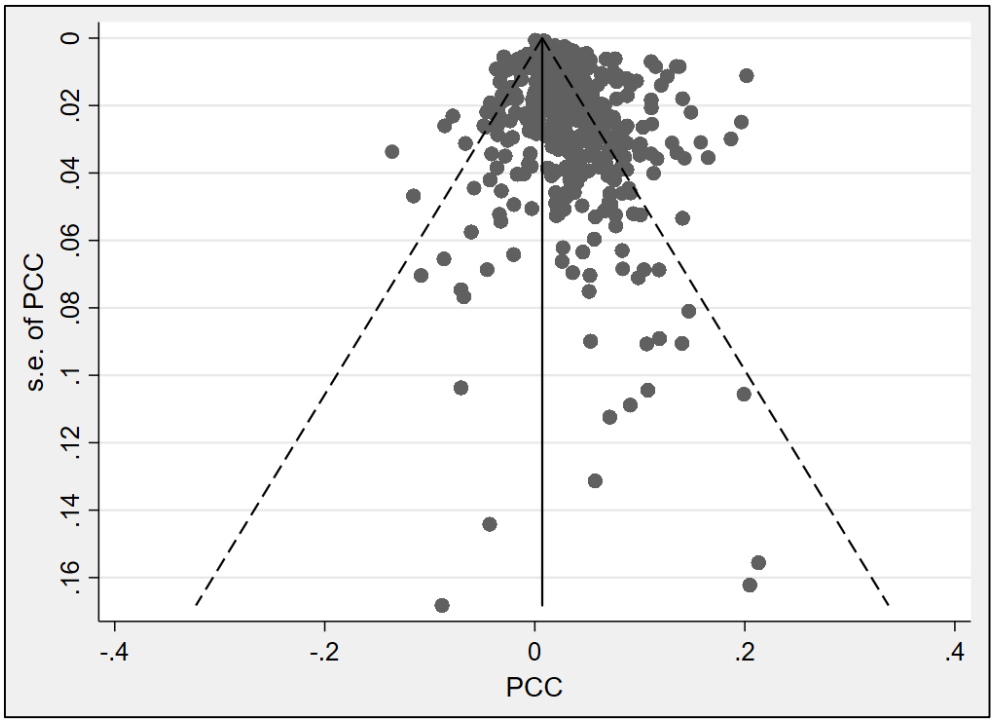
FIGURE 3

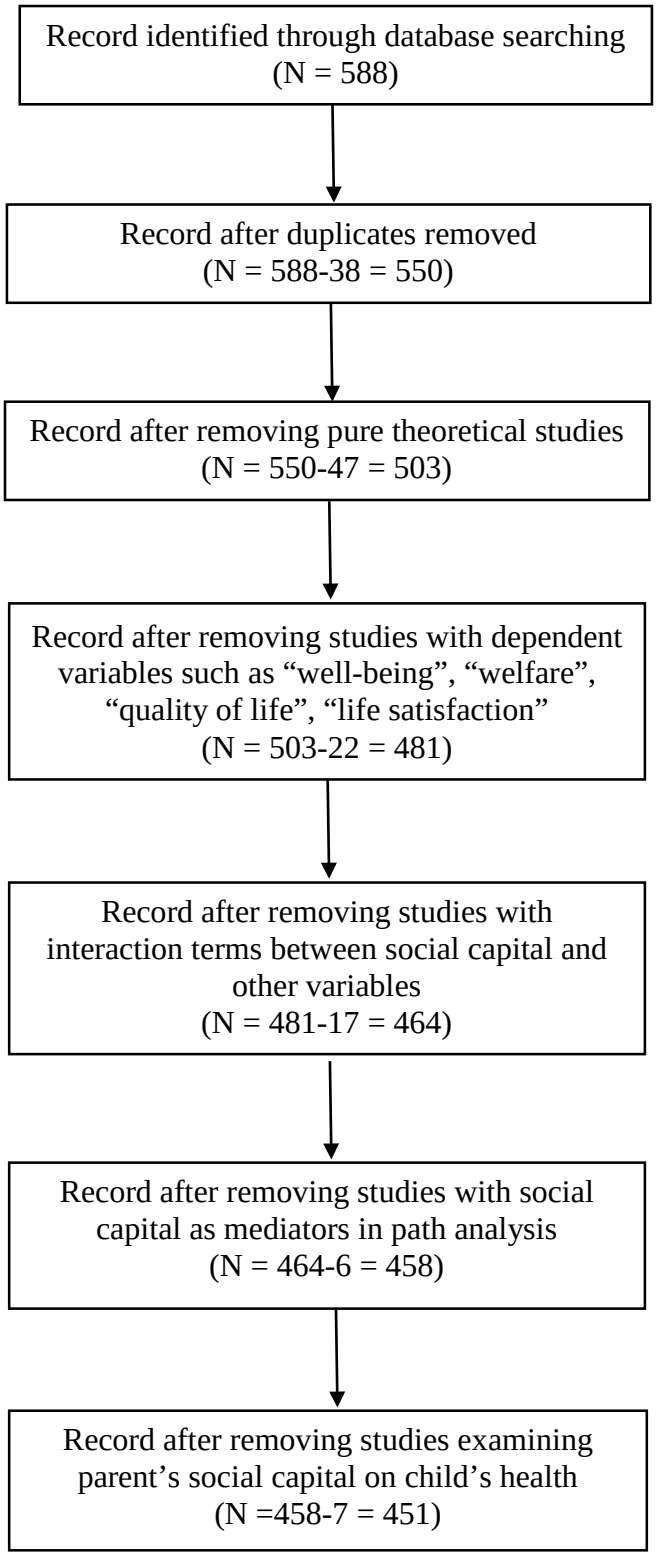
Funnel Plots

A. Individual Estimates



B. Mean Study Estimates



APPENDIX

Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) Diagram