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FROM SOCIAL CAPITAL TO HEALTH – AND BACK

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ABSTRACT

We assess the causal relationship between health and social capital, measured by generalized trust, both at the individual and the community level. The paper contributes to the literature in two ways: it tackles the problems of endogeneity and reverse causation between social capital and health by estimating a simultaneous equation model, and it explicitly accounts for mis-reporting in self-reported trust.

The inter-relationship is tested using data from the first four waves of the European Social Survey for 25 European countries, supplemented by regional data from Eurostat. Our estimates show that a causal and positive relationship between self-perceived health and social capital does exist and that it acts in both directions. In addition, the magnitude of the structural coefficients suggests that individual social capital is a strong determinant of health, whereas community level social capital plays a considerably smaller role in determining health.

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1. INTRODUCTION

Social capital, despite some ambiguity as to how it should best be defined, has increasingly been recognized among economists as an important concept that matters for a range of key economic outcomes, from financial development to the spread of secondary education (Knack and Keefer, 1997; Goldin and Katz, 2001; Zak and Knack, 2001; Guiso *et al*., 2004; Akçomak and ter Weel, 2009). Economists have developed theoretical frameworks analyzing the determinants of investment in social capital, concluding that social capital can be readily integrated into standard microeconomic models (Glaeser *et al*., 2002). Yet, the relationship between social capital and health has received some attention only very recently (Scheffler and Brown, 2008), in some contrast to a steadily growing public health literature on the subject that started in the mid-1990s.

The evidence of a link between social capital and health is strong (Cooper *et al*., 1999; Lochner *et al*., 1999; Machinco and Starfield, 2001; and Islam *et al*., 2006, for a review). Indeed, the large majority of the empirical studies does find a strong positive association (Petru and Kubek, 2008; Fujisawa *et al*., 2009; Hurtado *et al*., 2011), with only rare exceptions (e.g., Veenstra, 2000; Engstrom *et al*., 2008). However, both Durlauf (2002) and Durlauf and Fafchamps (2005) have demonstrated how the early literature on social capital generally failed to identify the *causal* effect, especially because most studies could not distinguish the effect of social capital from that of individual preferences or of other community characteristics. A small number of recent contributions have tried to address the causality problem by means of instrumental variables (Folland, 2007; D'Hombres *et al*., 2010; Ronconi *et al*., 2012). In addition, although the previous literature acknowledged that the relation between social capital and health might be circular (see e.g., Von dem Knesebeck *et al*., 2005; Islam

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et al., 2006), a more complete empirical evaluation of this complex, potentially bi-directional relationship has hitherto been missing.[1]

Besides, there is a need to specify the relevant dimension of social capital that may be responsible for the link between social capital and health: is it the social capital at the individual level or at some more aggregate community level or both that matters? This ambiguity is partly related to the way social capital is defined. The existing literature is divided into those papers that consider social capital primarily as an individualistic phenomenon (Mansyur *et al*., 2008), those that perceive it as a feature of the community (Poortinga, 2006a) and those that find a role for both levels (Engström *et al*., 2008; Iversen, 2008; Snelgrove *et al*., 2009). It is crucial to assess the relative importance of both dimensions, as this would suggest the appropriate entry point for policy interventions: should policy target social capital at the individual level (e.g., by providing social support through individualized 'befriending'[2]), or should policy rather promote social capital at the aggregate level (Thomson *et al*., 2006), or should one do both?

The purpose of the paper was twofold: first, we evaluate for the first time the simultaneous two-way causal link between social capital and health. Second, we analyze at which level the relationship operates. Our 'baseline' empirical modeling approach derives from an agnostic view about the direction of causality: we have specified a simultaneous equation model to allow for a general feedback relationship between social capital and health. In a further elaboration (and robustness check), we analyze whether and how possible mis-reporting in social capital and health might alter our baseline estimates. To the best of our knowledge, this is the first paper in this literature that explicitly deals with the issue of mis-reporting, generally considered as a major empirical challenge in the social science research on social capital (Guiso *et al*., 2010).

We find that the relationship between social capital and health is indeed circular and mutually beneficial, both in the baseline model and when we account for mis-reporting. Moreover, social capital measured at the individual level has both a stronger and a statistically more significant impact than community social capital, although the effects at either level remain positive and significant.

The paper is organized as follows. Section 2 reviews the concept of social capital and the plausible channels through which social capital influences health. Section 3 discusses the measurement of social capital in our paper and in the relevant literature. Section 4 describes the data, Section 5 introduces the model, Section 6 presents our results, Section 7 contains a discussion on the validity of the instruments and Section 8 concludes.

2. SOCIAL CAPITAL AND HEALTH

2.1. Definitions

A key limitation that has arguably stymied a larger research activity on social capital relates to the ambiguity of its definition (Durlauf and Fafchamps, 2005). One basic distinction is between those authors who perceive social capital as a quality of some aggregate (or 'community') level and those who consider social capital primarily as a characteristic of the individual. Sociological and political science definitions have tended – although by no means exclusively – to emphasize the aggregate level perspective. Putnam referred to social capital as those 'features of social organization, such as trust, norms, and networks that can improve the efficiency of society by facilitating coordinated actions' (Putnam, 1993:167).[3] By contrast, economists have commonly expressed a preference for a more individualistic notion. Glaeser *et al*. (2002) emphasize the

[1]An exception is Sirven and Debrand (2011).

[2]'Befriending' is defined in the public health literature as the forming of 'a relationship between two or more individuals, which is initiated, supported and monitored by an agency that has defined one or more parties as likely to benefit. Ideally, the relationship is non-judgmental, mutual and purposeful, and there is a commitment over time' (Mead *et al*., 2010, p. 96)

[3]Bourdieu (1985) defines social capital as the resources that can be derived from the possession of a 'durable network of more or less institutionalized relationships of mutual acquaintance or recognition' (p. 248). Loury's (1977) and Coleman's (1988) definitions refer to all those elements that facilitate certain action within social structures. They indicate social capital and in particular the interactions within the family as a crucial input for human capital. Putnam itself states 'the most fundamental form of social capital is the family' (Putnam, 1995, p.73).

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individuallevel perspective by defining social capital as the individual's social characteristics that enable private returns via interaction with others. They also point out that only if social capital is an individual concept can we hope to rationalize it, discuss its accumulation and its production by applying the economic toolbox. Guiso *et al*. (2008) define social capital as 'good' culture that is a set of beliefs and values that facilitates cooperation among the members of a community. Essentially, they consider social capital as a capital of civism and convincingly show that this definition answers to the critic of Solow (1995). According to this definition, social capital is an individual characteristic that can be accumulated and transmitted across generations and that is distinguished from human capital because its returns are contingent on the norms and beliefs of the other members of the community.

The definition of social capital in terms of values and beliefs resolves the debate as to whether social capital is an individual or community characteristic. A belief is by construction an individual measure of social capital formed on the basis of information and priors available to individuals. As the latter vary across individuals, so will the corresponding beliefs. At the same time, individual beliefs reflect at least imperfectly some latent fundamental traits of the society, namely the true community social capital.

Formally, we can write individual social capital (SC) of an individual *i* living in community *j* as the result of two components, the latent level of community social capital (CSC) in community *j* and an individual-specific deviation drawn from a zero-mean probability distribution

$$SC_{ij} = CSC_j + \eta_{ij} \qquad (*)$$

In this formalization, an estimate of the latent CSC can be recovered by averaging (*) at the community level. This would have the intuitive interpretation that each individual grounds her perception (i.e., belief) of the willingness to cooperate in the community on an imperfect (although not systematically biased) observation of its actual willingness to cooperate.[4]

2.2. Mechanisms

At least four mechanisms may account for the potential positive influence of SC on individual health.

(1) Social capital may provide easier access to health relevant information, as a result of more intense social interactions (Berkmann and Glass, 2000). The more an individual is involved in continuous social interaction, the easier and cheaper her access to information.
(2) Social capital may facilitate the provision of informal health care and/or psychological support (Murgai *et al*., 2002). Financial support may be required to cover the out-of-pocket costs of health care. The market or the public health care systems are usually unable to provide such services. Therefore, people agree on mechanisms of informal assistance between neighbors or friends. Such support tends to arise only in a context of reciprocal trust, as there is no enforceable contract guaranteeing obligations.[5]
(3) Social capital may facilitate people's lobbying efforts and coordination to obtain health-enhancing goods and services (Kawachi *et al*., 1997; Mellor and Milyo, 2005).
(4) Social capital, by increasing expected value-of-life, may induce rational people to reduce the amount of risky behaviors (Folland, 2006).

However, more intense social relationships may also help spread poor health (e.g., increased susceptibility to infectious diseases or favor the adoption of unhealthy behaviors), by means of stronger peer effects (Kawachi and Berkman, 2001).[6]

[4]Note that the individual perception of community social capital, that is, individual social capital, constitutes perhaps the most important dimension of social capital to consider, as it only drives individual actions, regardless of the level of latent community social capital.
[5]Cooperation allows reducing transaction costs and establishing efficient transactions in the presence of incomplete contracts (Alesina and La Ferrara, 2002).
[6]However, Brown *et al*. (2006) find a negative association between community social capital and smoking.

3. MEASURING SOCIAL CAPITAL

Measuring SC is a particular challenge to applied researchers because SC has many complementary dimensions. The theory suggests considering indicators related to individual beliefs and values, as Guiso *et al.* (2008, 2010) convincingly argued.

In line with extensive practice in the SC literature (Poortinga, 2006b; Rostila, 2007; Mansyur *et al.*, 2008; Petrou and Kupek, 2008; Snelgrove *et al.*, 2009; Borgonovi, 2010; D'Hombres *et al.*, 2010; Giordano and Lindstrom, 2010; Hurtado *et al.*, 2011), we focus on the level of generalized trust, an idiosyncratic individual belief at the core of SC. Generalized trust has been shown to have predictive validity for a number of other objective SC indicators, for example, the share of wallets returned in wallet drop experiments from capitals around the world (Zak and Knack, 2001), and with indicators of corruption and violent crime (Uslaner, 2002). In addition, studies find a close correlation between generalized trust in surveys and actual behavior as observed in experiments (e.g., Glaeser *et al.*, 2000; Fehr and Fischbacher, 2003). Our choice is also supported by Guiso *et al.* (2010), who conclude that trust is the best measurable indicator of civic capital. Other measures of norms contained in existing surveys are less reliable, because individuals tend to modify the answer toward a more socially acceptable one.[7] Last but not the least, the reported degree of trust is a quantity, which can be easily converted into a probability, the usual way economic theory defines beliefs.

Having adopted generalized trust as a measure of individual SC, we follow the literature in using as a measure of community SC the average level of generalized trust in the reference group of an individual (Mellor and Milyo, 2005; Portinga, 2006b; Olsen and Dahl, 2007; Rostila, 2007; Yip *et al.*, 2007; Mansyur *et al.*, 2008; Fujisawa *et al.*, 2009; Snelgrove *et al.*, 2009).[8] In this paper, reference groups are defined according to individual age and place of residence to better approximate the boundaries of true but unobservable individual social networks. This definition also allows to make reference groups only partially overlap across individuals (De Giorgi *et al.*, 2010; Carrieri, 2012).

4. DATA AND DESCRIPTIVE STATISTICS

The empirical analysis is based on a sample size of more than 130,000 people interviewed in the first four waves (2002/03, 2004/05, 2006/7 and 2008/9) of the European Social Survey (ESS), a repeated cross-section survey, which covers many European countries (see Tables 1 and 2 for summary statistics).[9] The ESS provides detailed information on individual social behavior and perceptions as well as about respondents' socio-economic characteristics and parental background. Unfortunately, health has not been a major focus in the survey design: respondents are only asked to self-report their current general health status and whether they are hampered in daily activities by illness or disability.

Information on the region of residence, at NUTS2-level (Nomenclature of Territorial Units for Statistics) in most cases, is also available. This feature has allowed us to supplement additional data about regional characteristics from the Eurostat REGIO dataset.

Regarding health, people are asked to rate their current health on a five-step scale ranging from very bad (1) to very good (5). Although self-reported health is a noisy measure of true health, it has proven a remarkably reliable proxy. In particular, it has been shown that self-reported health is highly correlated with subsequent mortality at the individual level (Ferraro and Farmer, 1999).

[7]We refer to indicators deriving from the fairness of behaviors such as claiming government benefits to which you are not entitled, avoiding a fare on public transport, cheating on taxes if you have a chance, accepting a bribe in the course of their duties, lying in your own interest, throwing away litter in a public space and speeding over the limit in built up areas.
[8]Some authors use generalized trust (and other similar items) to construct a trust index query (Veenstra, 2000; Yip *et al.*, 2007; Baron-Epel *et al.*, 2008; Engstrom *et al.*, 2008).
[9]See Table 1 for a list of the countries covered.

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Individual SC is captured by the individual degree of generalized trust. The question is 'Generally speaking, would you say that most people can be trusted, or that you can't be too careful in dealing with people? Please tell me on a score of 0 to 10, where 0 means you can't be too careful and 10 means that most people can be trusted'. Hence, respondents rate their trust on a Likert-type scale ranging from 0 to 10. We have recoded this variable to allow it to take values between −5 and 5. The community SC associated with individual *i* is measured as the mean trust of his or her reference group. The reference group is composed of the pool of people who the individual is more likely to relate to, and it is defined within the region of residence (coded as NUTS 2), which is the most disaggregated geographical level at which data are available in the ESS for most countries.

Given the precise definition of a reference group is ultimately arbitrary, we examine the robustness of our results against a range of three alternative definitions. Members of the reference group of individual *i* are the following:

Definition 0 (baseline): all people living in the same region of *i* and who are at most 10 years older or 10 years younger than *i*.
Definition 1: the residents of the same region as individual *i*, whose age belongs to the interval $[age_i - (2+0.2*age_i),\ age_i+(2+0.2*age_i)]$.
Definition 1 adopts the plausible notion that younger people tend to interact more with people of similar age, compared with older people. Both Definition 0 and Definition 1 imply a sharp discontinuity at the age boundary. Definition 2 eliminates such a discontinuity:
Definition 2: the residents of the same region as individual *i*. A value of 1 is assigned to people whose age differs from *i*'s age by less than 3 years, and a smaller weight, decreasing at increasing rates with age difference, is assigned to those aged 3 years older or younger than *i*.

In each definition, the reference groups are assumed to partially overlap, as in the work of De Giorgi *et al*. (2010).[10]

5. EMPIRICAL MODELING STRATEGY

5.1. Baseline model

We describe the circular relationship between SC and health by defining a simultaneous equation model. Following Stern (1989),

$$h^{**} = \alpha_0 + \alpha_1 SC^{**} + \alpha_2 CSC^{**} + \alpha_3 Z_h + X\alpha_4 + \varepsilon_h \quad (1)$$

$$SC^{**} = \beta_0 + \beta_1 h^{**} + \beta_2 CSC^{**} + \beta_3 Z_{SC} + X\beta_4 + \varepsilon_S \quad (2)$$

where h^{**} is individual health, SC^{**} is individual social capital and CSC^{**} is community social capital, defined as the average individual SC of her reference group. Z_h is a set of controls specific to equation (1), Z_{SC} a set of controls specific to equation (2) and X a set of controls common to both equations. Finally, ε_h and ε_S are i.i.d. error terms.Variables h^{**} and SC^{**} are jointly and endogenously determined in the system, whereas CSC^{**} is considered endogenous because it is likely correlated with (unobservable) community historical characteristics. The double asterisks indicate that these variables are continuous latent variables, not directly observed by the researcher, but truly reported and correctly measured. The relationship between latent (h, S and) and observed categorical variables (h^{**}, SC^{**} and $CSC**$) is described by the following equations:

[10]This specification alleviates some of the identification problems raised by Durlauf (2002) and Acemoglu and Angrist (2001), whose concerns and results, developed for fully overlapping reference groups, do not apply in our context. Note that we have defined reference groups on the basis only of age, an exogenous individual characteristic. This is admittedly a rather crude approach, but it is motivated by the need to avoid the problem of self-sorting of individuals into reference groups.

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$$h = i \text{ if } \Xi_{i-1} < h^{**} \leq \Xi_i, i = 1, 2.., 5 \quad (3)$$

$$S = i \text{ if } \Sigma_{i-1} < SC^{**} \leq \Sigma_i, i = 0, 1.., 10 \quad (4)$$

$$\bar{S} = i \text{ if } \bar{\Sigma}_{i-1} < CSC^{**} \leq \bar{\Sigma}_i, i = 0, 1.., 10 \quad (5)$$

where Ξ_i, Σ_i and $\bar{\Sigma}_i$ are threshold parameters with Ξ_0, Σ_{-1} and $\bar{\Sigma}_{-1}$ equal to $-\infty$ and Ξ_5, Σ_{10} and $\bar{\Sigma}_{10}$ equal to $+\infty$.

Following Maddala (1983) and Stern (1989), we estimate the model using a two-stage procedure. We first derive the reduced form of the system (1)–(2) as

$$h^{**} = W\Pi_h + \mu_h \quad (6)$$

$$SC^{**} = W\Pi_{SC} + \mu_{SC} \quad (7)$$

$$CSC^{**} = W\Pi_{CSC} + \mu_{CSC} \quad (8)$$

where $W = [Z_h, Z_{SC}, Z_{CSC}\ X]$ includes all the exogenous variables in equations (1) and (2) and variable Z_{CSC} is an external-to-the-model exogenous variable required to identify the effect of CSC^{**}.[11] Finally, Π_j, $j = h, SC, CSC$ are the reduced form parameters and μs are error terms.

In equations (6)–(8) the three endogenous variables depend on all the exogenous variables. They express the equilibrium levels of health, individual and community SC. Parameters Π_j indicate the contribution of each exogenous variable to such equilibrium levels. Equations (6)–(8) are separately estimated by means of ordered probit. The predicted values h^{**}, SC^{**} and CSC^{**} of the latent variables obtained in this first stage are replaced in equations (1)–(2), which are as well estimated in the second stage by ordered probit. Standard errors are estimated by bootstrapping the entire procedure, given that the usual 2SLS standard error correction does not apply to nonlinear models.

Our identification of the structural parameters of equations (1)–(2) is not based only on the nonlinear functional form, but mainly on the exclusion restrictions (Stern, 1989). The crucial non-testable assumptions are that excluded variable vector Z_h has no autonomous effect on SC^{**}, Z_{SC} has no autonomous effect on h^{**} and Z_{CSC} has no autonomous effect on both SC^{**} and h^{**}. Conversely, Z_h must be relevant to equation (1), and symmetrically, Z_{SC} must be relevant in equation (2). These conditions are equivalent to the IV conditions of excludability and relevance of the instruments in the single-equation IV models (see Wooldridge, 2002, chapter 9).

5.2. Endogenous variables, inclusions and exclusions

We condition the estimates of both equations (1)–(2) on a long battery of controls, the vector X that includes[12] the following:

(1) *Parental background indicators*: respondent father's and mother's educational attainment, father's and mother's employment status when respondent was 14 years old, whether either father or mother died before respondent was 14 years old and whether either father or mother was born in the country of the respondent's residence.
(2) *Individual characteristics*: gender, age, age squared, years of education, type of occupation, religion, respondent's main household income type, whether the respondent was born in the country of current residence, respondent's marital status and place of residence (urban/rural).
(3) *Reference group controls*: average years of education, percentage of peers in each type of income and proportion of men.

[11]The use of external information is necessary because CSC^{**} is included in both equations.
[12]See Table 2A for the complete list.

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(4) *Regional controls*: working age (15–64 years) activity rate and employment rate, young adults (25–34 years) activity rate and employment rate, youth (15–24 years) unemployment rate, proportion of college graduates, GDP per capita, GDP growth rate, age structure of the population, population density, kilometers of motorways, proportion of households without internet access, proportion of immigrant residents, proportion of citizens out of total residents and, finally, a measure of the level of crime.[13]

Parental background has long been recognized as being closely correlated with individual unobserved ability and preferences, which in turn influence individual perception and behaviors. Furthermore, education and type of occupation have an obvious influence on both SC and health. In addition, both reference group characteristics and regional ones are included to account for possible confounders of CSC. For instance, CSC could be lower in more sparsely populated regions, because of higher mobility costs of meeting friends and fellows. At the same time, health could be better in sparsely populated areas because they are typically rural, less industrialized and less polluted.

Variable Z_h included in equation (1) is the number of doctors per 1000 inhabitants, a measure of health care supply at the regional level (NUTS 2). The relevance of this variable rests on the fact that health care services are inputs into individual health. Symmetrically, conditional on the common set of controls, X, and specifically on the population age structure, which captures residents' average health, the supply of doctors is unlikely to have a direct impact on individual SC. Further, we neutralize the possibility that the quality of the health care system is indirectly correlated with individual SC via its correlation with more general good public governance, by conditioning on individual, peer and regional characteristics. These considerations motivate the exclusion of Z_h from equation (2).

Variable Z_{SC} in equation (2) is a measure of crime victimization. Respondents are asked whether they have been victim (in the past 5 years) of a burglary or an assault, that is, petty crimes, which hardly have any lasting medium or longer-term direct physical or mental health consequences, as they do not involve physical injuries. Conditional on regional characteristics, regional crime level, individual wealth, education, gender and age, having being victim of a burglary can be considered a *random event* outside individual control. In addition, Z_{SC} is not an individual perception, possibly correlated with individual characteristics, but an actual experience, which leads individuals to be more skeptical and less trusting vis-à-vis the rest of the society, at least with respect to those outside the inner circle of close relatives and friends. We shall discuss more in depth the validity of this instrument in Section 7.

Finally, variable Z_{CSC} is the average of Z_{SC} at the reference group level. Given that we are controlling for the overall crime rate in the region of residence (at the NUTS 2 level), Z_{CSC} can be excluded in equations (1) and (2).

5.3. Extended model

A potential threat to the validity of our estimates is mis-reporting in self-reported variables. To account for this problem, we add equations (11) and (12) to the baseline model:

$$h^{**} = \alpha_0 + \alpha_1 SC^{**} + \alpha_2 CSC^{**} + \alpha_3 Z_h + X\alpha_4 + \varepsilon_h \tag{9}$$

$$SC^{**} = \beta_0 + \beta_1 h^{**} + \beta_2 CSC^{**} + \beta_3 Z_S + X\beta_4 + \varepsilon_S \tag{10}$$

$$SC^{*} = SC^{**} + (1-\lambda)(CSC^{*} - SC^{**}) + \gamma h^{**} + \theta_S \tag{11}$$

$$h^{*} = h^{**} + \theta_h \tag{12}$$

Reported individual SC^{*} is a latent variable equal to truthfully reported individual SC^{**} plus a bias depending on (i) the average level of SC *reported* by the reference group,[14] (ii) the true individual health status, even if the

[13]We use the proportion of residents who report having been victim of a burglary or an assault in the past 5 years. This choice of crime measure is important for our identification strategy, as we shall explain further in the paragraphs that follow.

[14]Given λ is less than one, if the individual latent real social capital is lower than the average social capital reported in her community, she will adjust her reported social capital upward by a factor equal to this difference multiplied by $(1-\lambda)$.

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sign is difficult to predict, and (iii) a random error possibly correlated across members of the same reference group. In other words, we allow reports of individuals belonging to a given reference group to be strategic complements. This specification of the mis-reporting accounts for the possibility that individuals 'anchor' their situation[15] to the average value reported by their peers (Winkelmann and Winkelmann, 1998; Senik, 2004).

By rewriting (11) as

$$SC^{*} = \lambda SC^{**} + (1-\lambda)CSC^{*} + \gamma h^{**} + \theta_S \tag{13}$$

SC^{*} is a weighted average of SC^{**} and CSC^{*}. This offers a complementary interpretation for mis-reporting, which is the desire of the respondent to appear more society-oriented than she actually is, or to conform to the answers of her reference group. Conformism could be the outcome of the so-called 'social desirability bias', the tendency of individuals to deny socially undesirable traits and behaviors and to profess socially desirable ones (Randall and Fernandes, 1992).

Finally, equation (12) simply assumes that reported health is equal to true health plus a random independently distributed error term. Individual variables X could easily be included in (12) without affecting the estimate and the identification of the structural parameters, provided that they are orthogonal to θ_h.[16]

By averaging (11) over the members of individual's reference group, we (approximately) obtain

$$CSC^{*} = CSC^{**} + (1-\lambda)(CSC^{*} - CSC^{**}) + \gamma\overline{h}^{**} + \bar{\theta}_S \tag{14}$$

where $\overline{h}^{**}$ is the average health status of the reference group and $\bar{\theta}_S$ is the average error term, which can interpreted as an unobservable group effect (for further details about this approximation, see Appendix A in Rocco *et al.*, 2011). By solving (14) for CSC^{**}, we obtain

$$CSC^{**} = CSC^{*} - \frac{\gamma}{\lambda}\overline{h}^{**} - \frac{\bar{\theta}_S}{\lambda} \tag{15}$$

From (11), SC^{**} can be written as

$$SC^{**} = \frac{1}{\lambda}SC^{*} - \frac{1-\lambda}{\lambda}CSC^{*} - \frac{\gamma}{\lambda}h^{**} - \frac{\theta_S}{\lambda} \tag{16}$$

Finally, by substituting first (15) and (16) and next (12) in both (9) and (10), we obtain a specification of the extended model expressed exclusively in terms of the observed variables.

$$\begin{aligned} h^{*} = {} & \frac{\alpha_0}{\left(1+\alpha_1\frac{\gamma}{\lambda}\right)} + \frac{\frac{\alpha_1}{\lambda}}{\left(1+\alpha_1\frac{\gamma}{\lambda}\right)}SC^{*} + \frac{\left(\alpha_2 - \alpha_1\frac{1-\lambda}{\lambda}\right)}{\left(1+\alpha_1\frac{\gamma}{\lambda}\right)}CSC^{*} + \frac{-\alpha_2\frac{\gamma}{\lambda}}{\left(1+\alpha_1\frac{\gamma}{\lambda}\right)}\overline{h}^{*} + \\ & + Z_h\frac{\alpha_3}{\left(1+\alpha_1\frac{\gamma}{\lambda}\right)} + X\frac{\alpha_4}{\left(1+\alpha_1\frac{\gamma}{\lambda}\right)} + \left[\theta_h + \frac{\varepsilon_h - \alpha_1\frac{\theta_S}{\lambda} - \alpha_2\frac{\bar{\theta}_S}{\lambda}}{\left(1+\alpha_1\frac{\gamma}{\lambda}\right)}\right] \end{aligned} \tag{17}$$

$$\begin{aligned} SC^{*} = {} & \lambda\beta_0 + \lambda\left(\beta_1 + \frac{\gamma}{\lambda}\right)h^{*} + (1-\lambda+\lambda\beta_2)\,CSC^{*} - \gamma\beta_2\overline{h}^{*} + \\ & + Z_S\lambda\beta_3 + X\lambda\beta_4 + [\theta_S - \theta_h + \beta_2\bar{\theta}_S + \lambda\varepsilon_S] \end{aligned} \tag{18}$$

where we used the fact that $\bar{\theta}_h = 0$ because θ_h is i.i.d.

Compared with equations (1)–(2), equations (17) and (18) need to include the average health of the reference group. As $\theta_S \perp h^{**}$ and each individual contribution to the reference group averages is negligible, $\overline{h}^{*}$ can be considered exogenous.

[15]Often people tend to conform to others' declarations and dislike to take stands too far from the average.

[16]A possible concern is related to reporting heterogeneity in self-reported health both within country and between countries (see for instance Lindeboom and van Doorslaer, 2004 on this topic).

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We estimate the system (17)–(18) by means of the procedure described for the baseline model. Importantly, all the structural parameters are identified (see Appendix B in Rocco *et al*., 2011).

6. RESULTS

The baseline model estimates based on reference group Definition 0 are displayed in Table 3. Only the coefficients of the endogenous variables and exclusions Z_{SC}, Z_{CSC} and Z_h have been reported.[17] First-stage estimates, that is, those corresponding to the reduced form equations (6)–(8), are reported in columns 1 to 3. Second-stage estimates of the SC equation (2) and the health equation (1) are reported in columns 4 and 5, respectively.

The reduced form equations (6)–(8) are the roots of the simultaneous system (1)–(2); hence, they represent the equilibrium levels of individual health, SC and CSC. Thus, their parameters capture the total effect of the exogenous on the endogenous variables in equilibrium.

Instead, if taken alone, structural equation (1) describes how SC, CSC and the exogenous variables influence health, by treating SC and CSC as if they were not jointly determined with health. The same is true for equation (2). Therefore, their parameters capture partial effects.

The estimates of the structural equations (columns 4 and 5) indicate that there exists a circular relationship between individual SC and health. Moreover, CSC has a positive influence on both SC and health, although only marginally significant in the second case. The beneficial impact of individual SC on health turns out to be at least an order of magnitude larger than the one of CSC.

Exclusion restrictions are always highly statistically significant and have the expected sign. Increasing the number of doctors is beneficial to individual health (column 5). Importantly, its impact on health is positive and statistically significant also at equilibrium (column 1), and given the reinforcing two-way relationship between SC and health, it is positively correlated with the equilibrium value of individual SC (column 2). The sign of the coefficient on Z_h is negative as it clearly results from the estimate of the number of doctors reported in column (3). Having been victim of a crime (variable Z_{SC}) significantly reduces individual SC (column 4), and the same effect prevails at equilibrium for both individual SC and health (columns 1 and 2). Finally, the proportion of crime victims in the reference group significantly reduces CSC (column 3), as expected.

The stability of the point estimates across alternative definitions of the reference groups is remarkable, as it emerges from Tables 4 and 5, where we have adopted Definitions 1 and 2, respectively.

We have tested for the presence of unobserved regional factors by estimating one side of the relationship (i.e., the effect of SC on health) by including regional fixed effects (Table 6). Estimates are very similar to those in Table 3, indicating that our controls already capture most of the relevant heterogeneity across regions.

Table 7 reports estimates of the extended model that explicitly accounts for mis-reporting and serves as a robustness check of the findings from the baseline model. Unsurprisingly, because the model estimated in Table 7 is very close to that of Table 3, point estimates are similar.

However, now the estimated coefficients have to be understood as combinations of the structural parameters and cannot be directly compared with the results of the baseline model. Structural parameters have been derived by using the formulas reported in Appendix B by Rocco *et al*. (2011) and have been included in Table 8.

There is evidence of mis-reporting, as the weight of reported CSC on reported individual SC ranges between 0.194 and 0.352. Besides, the circular relation between SC and health is confirmed, as well as the finding that individual SC contributes far more to individual health than does CSC. However, the size of the individual SC effect is considerably lower (i.e., by about one-third) in the extended model, whereas the effect of CSC is broadly unchanged and it is now very precisely estimated. Finally, health contributes considerably more to individual SC according to the extended model than according to the baseline one, thus reinforcing the indication that SC and health are part of a feedback relationship that works strongly in both directions.

[17]Complete estimate tables are available upon request.

The results also suggest that people in worse health conditions tend to report a higher SC, and unlike in the baseline model, the link between true CSC and true individual SC is insignificant in two out of three specifications.

7. VALIDITY OF THE INSTRUMENTS

In this section, we discuss more in depth the validity of the instrumental variables 'crime victimization in the past 5 years' and 'physician density'. The first-stage estimates indicate that both instruments are strong. As for the exogeneity of crime victimization, we can exclude the possibility of a direct effect of the crime on physical health, because we are considering only petty crimes. However, the experience of being victimized could increase the probability of developing depression or other mental disorders. Sorenson and Golding (1990) find that people mugged or sexually assaulted in the 6 months prior to the interview are more likely to be depressed or suicidal, although no significant effect emerges in case of burglary. Norris and Kaniasty (1994) follow a sample of crime victims for 15 months after the event and study short and long run effects of crime victimization on a number of mental health outcomes including symptoms of depression and anxiety. They find that violent crimes have a strong and long-lasting effects on mental health, whereas the effect of property crimes is smaller and mainly short run. In any case, crime will have to exceed some threshold level of severity for it to influence victims' mental health.[18] Overall, this evidence implies that small crimes that occurred up to 5 years earlier (i.e., our instrumental variable) can hardly have direct consequences on mental health, supporting the validity of our instrument.

Exogeneity of the instruments could further be questioned if the instrument influenced health via channels other than SC. For instance, Kuroki (2012) finds a negative association between crime victimization and individual happiness (although this seems to be the case for burglaries but not for robberies). If happiness were an input into individual health distinct from SC, then crime victimization would not be a valid instrument anymore, as it could not be excluded from the health equation. What happens if we pretend that happiness was indeed an input into health and we include it into the model?[19] Its inclusion restores the validity of crime victimization as instrument for SC, given that it controls for the possible influence of crime victimization on health mediated by happiness.[20] Estimates from the so-augmented baseline model show small changes in the effects of individual SC and health. The only noticeable result of the inclusion of happiness is on the effect of community SC that becomes smaller and insignificant.[21]

As a further check to the validity of crime victimization, we have adapted the approach proposed by Altonji *et al*. (2005a, 2005b) to analyze the bias of our IV estimates from the health equation. The idea behind this approach is that one can measure the bias of the IV estimates under the hypothesis that the instrument is correlated in the same manner with included and omitted variables. This approach is very general, because it provides an indication about the reliability of the IV estimates regardless of what the threat to instrument exogeneity can be. Results indicate that – under the assumption of Altonji *et al*. (2005a, 2005b) – the bias is small and, remarkably, negative. This means that the effect of individual SC on health found in our analysis is likely to be a lower bound of the true effect.[22]

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[18] Sorensen and Goldwing (1990) use data collected in the Los Angeles area, whereas Norris and Kaniasty (1994) use data from the state of Kentucky.

[19] The evidence of a causal impact from happiness to health is scarce, although an association between the two variables has been found in several analyses (Scheier and Carver, 1992; Heliwell 2002; Levy *et al*., 2002; Lyubomirsky *et al*., 2005; Bjørnskov, 2008; Veenhoven, 2008). Recently, Sabatini (2011) attempted to identify a causal effect by means of an instrumental variables approach. On the other hand, Carrieri (2012) quite convincingly suggests that there exists a reverse causal influence, running from health to happiness.

[20] We make the strong assumption that, conditional on our battery of controls, happiness is exogenous. In reality, happiness, as social capital, is likely to be endogenous, and to properly account for this problem, we would need an additional instrument.

[21] Results are available from the authors upon request.

[22] Results are available from the authors upon request.

Finally, let us briefly discuss the validity of physician density. Suppose that a higher physician density is correlated with a higher health care quality, for instance because doctors have more time for each visit and each patient. This might reinforce the link between patients and doctors and might induce patients to augment their trust vis-à-vis doctors and the health personnel. If so, a higher physician density could influence individual SC directly, invalidating the use of physician density as an instrument. However, a considerable body of research has failed to find robust empirical support for the role of physician density on health care quality (Perrin and Valvona, 1986; Jürges and Pohl, 2012, and the references therein). Although a higher number of doctors increase the quantity of health services provided and facilitate access to healthcare, it does not significantly influence the time spent per patient, the appropriateness of the treatments or the degree of adherence to evidence-based medical guidelines.

8. CONCLUSIONS

In this paper, we have examined the relationship between SC, measured by self-reported generalized trust, and individual health in a large sample of European countries, using four cross-sectional rounds of the ESS. To the best of our knowledge, this is the first paper that explicitly explores and models the potential simultaneous relationship between SC and health and that explicitly takes into account the potential bias resulting from mis-reporting in SC.

We find strong evidence for a circular, mutually reinforcing relationship between SC and health even when mis-reporting is taken into account. In addition, individual SC is far more important than community SC as a determinant of health.

However, it is important to acknowledge the limitations of our analysis. First, the influence of SC on health (and vice versa) may in principle differ between countries. However, given the large sample size needed to achieve the required precision of the estimates, it was not possible to run the regressions by country. Second, the way residents in different countries report their health conditions and their SC could differ even if their true latent variables were the same. Although the richness of the set of controls included in our model presumably captures reporting heterogeneity of the type ‘index-shift’, reporting heterogeneity of the type ‘cut-point shift’ (Lindeboom and van Doorslaer, 2004) could not be adequately accounted for. More fundamentally, reporting heterogeneity can be taken into account only if some alternative and possibly more objective measures of health and SC were available in the data. Unfortunately, this is not the case in ESS. Third, a more adequate specification of the model would include also an interaction between individual and community SC, to explicitly test the hypothesis of complementarity between the two. Unfortunately, in light of the already complex model structure, and especially given that SC at both levels is endogenous, it would not be feasible to control for such interaction effect and achieve identification. Fourth, the health-reporting model has been kept very parsimonious to make the overall system tractable and achieve identification of the structural parameters. In this regard, a possible concern is that SC might influence not only the latent health conditions but also health reporting. As self-reported health is generally understood as a holistic measure of health/well-being, it might then be directly influenced by individual SC. Although we cannot exclude this possibility, our rich set of individual controls should at least partly address this concern.

Although our results suggest that SC is good for health (and vice versa), by themselves they provide only limited concrete policy implications, beyond the recommendation that policymakers should at least consider SC, alongside several other factors, when developing health policy. There remains significant scope for improving our understanding of how SC can be promoted. Szreter (2004) has studied which factors were important in building SC and, as a consequence, improving health. The first is a rejection of policies that concentrate wealth in the hands of a few. The second is a mechanism by which civic society can articulate its concerns and the political structures can respond to them. The third is political participation.

There is also some evidence that much can be achieved more locally through programs designed to increase social interaction (Semenza and Krishnasamy, 2007). A recent systematic review found some evidence of gains in mental health linked to interventions giving employees' greater control in organizations (Egan *et al*., 2007).

If it is true that promoting individual level SC has potentially more significant health effects than can be reaped from targeting community level SC, then interventions may want to target the former rather than the latter. Examples of individual level SC interventions include the provision of support for families and parenting (Halpern, 2005). Befriending approaches have been applied to some extent and with some success in the context of education (see e.g., Johnson, 1999; Kahne and Bailey, 1999). Alternative approaches may include the promotion of volunteering (Halpern *et al*., 2002).

Although the aforementioned programs may well have resulted in positive health outcomes as well, this has typically not been evaluated. Moreover, even if some examples do exist as to how to promote SC, a full economic evaluation of SC interventions would still require the assessment of the associated costs and exact health (and possibly non-health) benefits.

APPENDIX A

Table I. Number of Observations

Country	Number of observations	Percentage of the sample
Austria	6227	4.72
Belgium	6764	5.12
Bulgaria	2043	1.55
Czech Republic	5425	4.11
Germany	10,321	7.82
Denmark	2146	1.63
Estonia	4553	3.45
Spain	5581	4.23
Finland	4789	3.63
France	4685	3.55
Greece	6367	4.82
Croatia	1136	0.86
Hungary	4999	3.79
Ireland	4169	3.16
Italy	2063	1.56
Latvia	1358	1.03
Netherland	7224	5.47
Norway	6968	5.28
Poland	6643	5.03
Portugal	7423	5.62
Romania	1456	1.10
Sweden	5398	4.09
Slovenia	4819	3.65
Slovakia	4341	3.29
United Kingdom	7880	5.97
Total	132,031	

Note: Data, ESS (first 4 waves). Sample size: 132,031.

Table IIA. Control variables included in the models. Descriptive Statistics. Source: ESS waves 1–4.

Panel 1

Individual variables	Mean	Standard deviation
Self-reported health	3.785	0.913
Male	0.465	0.499

(*Continues*)

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Table IIA. (Continued)

Individual variables	Mean	Standard deviation
Age	47.223	18.248
Age squared	2563.028	1816.175
Born in the country	0.924	0.264
Urban	0.621	0.485
Married	0.533	0.499
Years of education	11.975	4.024
Religious	4.787	2.994
Victim of a crime	0.190	0.393

Note: Data, ESS (waves 1–4). Sample size: 132,031.

Panel 2

Individual variables (dummy)	Mean	Standard deviation
Religion. Excluded dummy: no religion		
Roman catholic	0.347	0.476
Protestant	0.151	0.358
Eastern orthodox	0.073	0.260
Other Christian denomination	0.016	0.127
Jewish	0.001	0.031
Islam	0.013	0.115
Eastern religions	0.003	0.055
Other non-Christian religions	0.002	0.048
Occupation. Excluded dummy: unemployed		
Legislators, senior officials and managers and armed force	0.495	0.500
Professionals	0.051	0.220
Technicians and associate professionals	0.082	0.274
Clerks	0.089	0.285
Service workers and shop and market sales workers	0.052	0.223
Skilled agricultural and fishery workers	0.069	0.253
Craft and related trades workers	0.015	0.120
Plant and machine operators and assemblers	0.065	0.246
Elementary occupations	0.038	0.191

Note: Data, ESS (waves 1–4). Sample size: 132,031.

Panel 3

Parental characteristics	Mean	Standard deviation
Father born in the country	0.894	0.308
Mother born in the country	0.896	0.306
Number of household members	2.778	1.427
Employed father	0.663	0.473
Employed mother	0.425	0.494
Self-employed father	0.224	0.417
Self-employed mother	0.097	0.295
Father died	0.058	0.235
Mother died	0.020	0.141
Missing father's education	0.074	0.261
Missing mother's education	0.059	0.235
Education (parents). Excluded dummy: father/mother not completed primary education		
Father in primary school or first stage of basic education	0.243	0.429
Father in lower secondary or second stage of basic education	0.218	0.413
Father in upper secondary	0.221	0.415
Father in post secondary, non-tertiary	0.035	0.184
Father in first stage of tertiary	0.091	0.288

(*Continues*)

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Panel 3. (Continued)

Parental characteristics	Mean	Standard deviation
Mother in primary school or first stage of basic education	0.273	0.445
Mother in lower secondary or second stage of basic education	0.246	0.431
Mother in upper secondary	0.202	0.401
Mother in the post secondary, non-tertiary	0.027	0.161
Mother in first stage of tertiary	0.064	0.245
Principal source of income. Excluded dummy: wages or salaries		
Self-employed or farming	0.077	0.266
Pensions	0.255	0.436
Unemployment/redundancy	0.019	0.136
Any other social benefit	0.026	0.160
I ncome from investment and saving	0.005	0.071
Income from other sources	0.014	0.118

Note: Data, ESS (waves 1–4). Sample size: 132,031.

Panel 4

Peers characteristics (reference group characteristic 0)	Mean	Standard deviation
Percentage of males (peers)	0.465	0.081
Years of education (peers)	12.034	2.178
Percentage of people whose main income comes from self-employed or farming activities (peers)	0.607	0.287
Percentage of people whose main income comes from pensions (peers)	0.077	0.068
Percentage of people whose main income comes from unemployment/redundancy (peers)	0.251	0.320
Percentage of people whose main income comes from any other social benefit (peers)	0.019	0.027
Percentage of people whose main income comes from investment and saving (peers)	0.026	0.042
Percentage of people whose main income comes from other sources (peers)	0.005	0.012
Years of education (peers)	12.034	2.178
Percentage of people victims of a crime	0.191	0.102

Note: Data, ESS (waves 1–4). Sample size: 132,031.

Panel 5

Regional characteristics (Nuts 2 level)	Mean	Standard deviation
Activity rate 15–64	71.910	6.013
Activity rate 25–34	85.804	4.006
Employment rate 15–64	66.672	7.532
Employment rate 25–34	79.255	6.117
Unemployment rate 15–24	16.091	8.696
Unemployment rate 15–64	7.393	4.318
Density	336.807	710.632
Number of graduates	0.240	0.084
Length of motorways	30.084	39.440
GDP	23858.130	13338.500
GDP growth	4.683	4.818
Percentage of young	0.165	0.023
Percentage of adults	0.676	0.022
Percentage of old	0.151	0.041
Percentage of people victims of a crime	0.190	0.080
Number of doctors	3.272	1.001
percentage of citizens	0.961	0.057
No internet	0.328	0.163
Percentage of immigrants	0.097	0.088

Note: Data, ESS (waves 1–4). Sample size: 132,031.

Panel 6

Other controls	Mean	Standard deviation
Round. Excluded dummy: Round 1		
Round 2	0.247	0.431
Round 3	0.233	0.423
Round 4	0.270	0.444
Geographic region: excluded dummy: south		
North	0.237	0.426
Central-west	0.286	0.452
Central-east	0.148	0.355
East	0.130	0.337

Note: Data, ESS (waves 1–4). Sample size: 132,031.

Table IIB. Size of the groups of peers

Definition	Average number of peers in the group	Standard deviation
Definition 0	116.723	108.769
Definition 1	135.289	130.574
Definition 2	116.856	115.004

Note: Data, ESS (waves 1–4). Sample size: 132,031.

Table III. Baseline model – reference group definition 0

	First stage			Second stage	
	Health	SC	CSC	SC	Health
# doctors	0.051***	0.005	−0.025***		0.047***
	(0.005)	(0.005)	(0.006)		(0.008)
Victim	−0.128***	−0.095***	0.005	−0.074***	
	(0.008)	(0.007)	(0.008)	(0.012)	
% victims	−0.826***	−0.460***	−2.231***		
	(0.065)	(0.055)	(0.082)		
health				0.164**	
				(0.073)	
CSC				0.146***	0.090*
				(0.035)	(0.051)
SC					1.356***
					(0.127)
N	132031				

Note: Data, ESS (waves 1–4). Sample size: 132,031. *# doctors* is the number of doctors in the region of residence (NUTS 2); *Victim* is a dummy taking 1 in the respondent have been victim of a burglary or an assault in the past 5 years; *% victims* is the proportion of victims of a burglary or assault in the respondent's reference group; *health* is self-reported health status; *CSC* and *SC* are community and individual social capital, respectively. The first 3 columns report first-stage estimated parameters of the excluded exogenous variables. The last two columns report second-stage estimates of exclusions and endogenous variables (baseline model with reference group definition 0). Asterisks indicate level of statistical significance: $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$. Boostrapped standard errors in parentheses.

Table IV. Baseline model – reference group definition 1

	First stage			Second stage	
	Health	SC	CSC	SC	Health
# doctors	0.051*** (0.005)	0.005 (0.005)	−0.021*** (0.006)		0.047*** (0.008)
Victim	−0.129*** (0.008)	−0.096*** (0.008)	0.006 (0.009)	−0.077*** (0.013)	
% victims	−0.864*** (0.067)	−0.399*** (0.056)	−1.946*** (0.089)		
health				0.154** (0.078)	
CSC				0.137*** (0.041)	0.166*** (0.062)
SC					1.355*** (0.135)
N	132031				

Note: See Table 3 (baseline model with reference group definition 1).

Table V. Baseline model – reference group definition 2

	First stage			Second stage	
	Health	SC	CSC	SC	Health
# doctors	0.050*** (0.005)	0.006 (0.005)	−0.022*** (0.006)		0.045*** (0.009)
Victim	−0.123*** (0.008)	−0.092*** (0.008)	0.010 (0.009)	−0.068*** (0.013)	
% victims	−1.530*** (0.103)	−0.828*** (0.098)	−2.616*** (0.133)		
health				0.212*** (0.075)	
CSC				0.193*** (0.053)	0.156* (0.084)
SC					1.354*** (0.138)
N	132031				

Note: See Table 3 (Baseline model with reference group definition 2).

Table VI. Baseline model – reference group definition 0 with regional dummies

	First stage		Second stage
	SC	CSC	Health
Victim	−0.096*** (0.007)	0.002 (0.009)	
% victims	−0.447*** (0.059)	−2.746*** (0.093)	
CSC			0.082* (0.045)
SC			1.350*** (0.135)
N	132031		

Note: Data, ESS (waves 1–4). Sample size: 132,031. *Victim* is a dummy taking 1 in the respondent have been victim of a burglary or an assault in the past 5 years; *% victims* is the proportion of victims of a burglary or assault in the respondent's reference group; *health* is self-reported health status; *CSC* and *SC* are community and individual social capital, respectively. The first two columns report first-stage estimates of the excluded exogenous variables of the health equation. The last column reports second-stage estimates of the endogenous variables (baseline model with reference group definition 0 with regional dummies). Asterisks indicate level of statistical significance: $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$. Boostrapped standard errors in parentheses.

Table VII. Extended model – reference group definition 0

	First stage			Second stage	
	health	SC	CSC	SC	Health
Mhealthtot	0.505***	0.046***	0.207***	−0.090*	0.452***
	(0.009)	(0.010)	(0.012)	(0.048)	(0.018)
# doctors	0.041***	0.004	−0.029***		0.034***
	(0.005)	(0.005)	(0.006)		(0.008)
Victim	−0.129***	−0.095***	0.005	−0.069***	
	(0.008)	(0.007)	(0.008)	(0.015)	
% victims	−0.497***	−0.428***	−2.086***		
	(0.061)	(0.053)	(0.080)		
Health				0.204**	
				(0.099)	
CSC				0.156***	−0.041
				(0.031)	(0.054)
SC					1.361***
					(0.133)
N	132031				

Note: See Table 3. (Extended model with reference group definition 0).

Table VIII. Structural Parameters

	Reference gr. definition 0	Reference gr. definition 1	Reference gr. definition 2
Extended model			
Effect of true SC on health α_1	0.353***	0.426***	0.499***
	(0.037)	(0.042)	(0.041)
Effect of true CSC on health α_2	0.077***	0.113***	0.272***
	(0.020)	(0.027)	(0.062)
Effect of health on true SC β_1	2.170***	1.650***	1.255***
	(0.318)	(0.236)	(0.181)
Effect of true CSC on true SC β_2	−0.059	−0.058	−0.211**
	(0.033)	(0.039)	(0.093)
Effect of health on reported SC γ	−1.524***	−1.151***	−0.567***
	(0.243)	(0.186)	(0.111)
Effect of reported CSC on reported SC 1-λ	0.203***	0.194***	0.352***
	(0.028)	(0.037)	(0.055)
Baseline model			
Effect of true SC on health α_1	1.356***	1.355***	1.354***
	(0.127)	(0.135)	(0.138)
Effect of true CSC on health α_2	0.090*	0.166***	0.156*
	(0.051)	(0.062)	(0.084)
Effect of health on true SC β_1	0.164**	0.154**	0.212***
	(0.073)	(0.078)	(0.075)
Effect of true CSC on true SC β_2	0.146***	0.137***	0.193***
	(0.035)	(0.041)	(0.053)

Note: Data, ESS (waves 1–4). Sample size: 132,031. Upper panel: structural parameters of the extended model, according to definitions 0, 1, 2. Lower panel: structural parameters of the baseline model (Table 3). Asterisks indicate level of statistical significance; $^{***}p<0.01$, $^{**}p<0.05$, $^{*}p<0.1$. Boostrapped standard errors in parentheses.

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