# Trust me, you will be in better health

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**Abstract**

Along the pathway traced by few recent contribution that attempt to identify the causal effect of social capital on health, this paper analyzes whether individual social capital reduces the probability of experiencing 11 long-lasting and chronic diseases. The empirical problems related to reverse causation and unobserved heterogeneity are addressed by means of a procedure that exploits the within-individual variation between the timings of first occurrence of the 11 diseases considered. Estimates indicate that the probability of occurrence is on average 14 to 18 percent lower among individuals reporting to "trust most of the other people". This result is robust to two alternative specifications as well as the inclusion or omission of individual controls.

## 1. Introduction

Among the socio-economic determinants of health, a growing attention is being devoted to the role of social capital. Many analysis have found a strong positive association between social capital and individual health (see Islam et al., 2006, for an extensive review) and the discussion about the pathways of this relationship is mounting (Folland et al., 2012).

Health economics and public health literature suggest several potential pathways for the influence of social capital on health. First, social capital may expand the informational resources available to individuals, allowing a faster and more intense circulation of health relevant information (Scheffler and Brown, 2008). Second, social capital favors the formation of informal networks and safety nets which provide mutual insurance to its members in case of health shocks (Murgai et al., 2002). Third, social capital may increase the political weight of a community making easier to obtain more and better public goods and social welfare programs (Kawachi and Kennedy, 1997). Fourth, social capital, by increasing the quality and the utility of future life, could discourage unhealthy behaviors, such as smoking, drinking or mis-nutrition (Folland, 2006). All pathways are justified and motivated by the fundamental characteristic of social capital of favoring cooperation within communities.

This paper investigates whether social capital benefits individual health, by exploiting rather detailed information about individual health condition and individual social capital included in the British Household Panel Survey between 1999 and 2008. For the first time, this paper investigates the influence of social capital on the likelihood of specific diseases rather than on self-reported general health (or analogous indicators) making possible to tell apart whether social capital does influence "true" health or just the way individuals assess and report their "true" health (Bailis et al. 2003; Jylhä 2009, 2010; Huisman and Deeg, 2010). Indeed, self-

reported health has been shown to be sensitive to changes in objective health conditions, such as the occurrence of a disease or the emergence of new symptoms, but to reflect also the individual prior (*self-concept*) on own health (Bailis et al., 2003). In particular, social factors could alter the way individuals assess and report their own health status (Rocco et al., 2013).

The empirical identification of the effect of social capital on health is problematic, because social capital is likely endogenous (Durlauf and Fafchampes, 2005). Building over few recent contribution that have attempted to identify the causal effect of social capital beyond simple associations, this paper innovates because it addresses two empirical problems, reverse causation and unobserved heterogeneity, by following an empirical strategy previously overlooked, inspired to duration analysis but quite specific. Reverse causation refers to the circular relationship likely to exist between social capital and health: indeed, not only social capital influences health but also the viceversa can be true. Unobserved heterogeneity, refers to the probable omission from the model of relevant and often unobservable characteristics, which can influence both social capital and health, such as individual preferences and attitudes. Both problems are responsible for unpredictable bias in the estimates obtained by simple regression models.

The identification strategy exploits within-individual variation in the timing of occurrence of 11 long-lasting and chronic diseases, both physical and mental[2]. The occurrence of each disease and the level of social capital reported before this occurrence form a *switching point.* Data are rearranged to obtain 11 switching points, one for each diseases, for all individuals. The empirical analysis is then conducted on the resulting dataset of switching points. As social capital is pre-determined at any switching point by construction, possible feedbacks from health conditions to social capital are ruled out. Moreover, since diseases occur at different times, the level of individual social capital varies within-individual across switching points. This variation allows to control for any time-invariant individual heterogeneity, such as preferences, family background and inherited culture by means of a standard individual fixed effect estimator. The effect of time-varying shocks is directly accounted for by a number of individual controls.

Results indicate that individuals rich of social capital, are on average 14 to 18 percent less likely to experience a disease, compared to individuals poor of social capital. This result is robust to two alternative specifications as well as the inclusion or omission of individual socio-economic controls.

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[2] In duration analysis this would be the timing of transition from one state to another.

The remaining of the paper is organized as follows: the definition of social capital adopted in this paper is justified in section 2; the relevant literature is reviewed in section 3; data are briefly described in section 4; the identification strategy is discussed in detail in section 5; results are reported in section 6 and finally section 7 concludes.

## 2. Definition of Social Capital

The concept of social capital has gained wide acceptance in social sciences and, more recently, in economics, where it has been used to explain economic growth (Knack and Keefer, 1997), size of firms (La Porta et al., 1997), institution's design and performance (Djankov et al., 2003), financial development (Guiso, Sapienza, and Zingales, 2004, 2008b), crime (Glaeser, Sacerdote, and Scheinkman, 1995), the power of the family (Alesina and Giuliano, 2007), innovation (Fountain, 1997), and the spread of secondary education (Goldin and Katz, 2001).

The term social capital is often traced back to the work of the sociologist Bourdieu (1977), but it gained popularity in the Nineties, mostly owing to Coleman (1990) and Putnam (1993). Though largely used, the concept is contested at both conceptual and measurement level. On the one hand, social capital has been conceptualized as a group attribute, i.e. as a property of the organization or the community, as opposed to a characteristic of the individual members[3]. On the other hand, the so called "network school" defines social capital as both an individual attribute and a property of the social network[4].

Social capital has generally been considered as a multi-faceted object and consequently the precise boundaries of the concept are still disputed. Quite differently from this tradition, Guiso et al. (2008a, 2011) have recently proposed a more clear-cut definition. They convincingly define social capital as an individual *belief* about others' willingness to cooperate. When defined in this way, social capital can be properly considered a form of capital, that can be accumulated, transferred and which returns accrue to its owner (in so doing answering to the well known Solow's critique – Solow, 1995). Indeed, beliefs are individual and vary across people, they can be updated as far as new information is acquired by means of social interactions (accumulation or de-cumulation of social capital) and can be transmitted from parents to children (transfer of social capital). Moreover, beliefs are probabilities and thus they have a well defined and undisputed metric. Last but not least, defining social capital as a belief avoids the often arising

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[3] Examples are social norms, sanctions, values and traditions that display their effects regardless of individuals' adhesion (Kawachi and Berkman, 2000).

[4] Examples are social support, information channels, social credentials, trust (Kawachi et al., 2008)

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confusion between social capital and some of its outcomes such as the quantity/quality of social relations, or involvement in social organizations, and makes social capital clearly distinct from human capital because its returns are contingent on the norms and beliefs of other community members. [5]

Rather than giving a proper account of the quite long list of social capital definitions appeared in the literature and pursuing an ecumenical approach trying to reconcile the multiple aspects of social capital, this paper grounds exclusively on Guiso et al. (2008a, 2011)'s definition and adopts the indicator (available in BHPS data) that most closely fits with this definition, i.e. generalized trust[6]. This approach has the advantage of simplifying the analysis and the interpretation of the results, since the object of interest is much more focused. Of course, the cost is that of losing results' richness and variety compared to an analysis dealing with a multi-faceted concept.

## 3. Literature

Beside a large and bourgeoning literature that indicates social capital as a key ingredient for economic growth, financial development and institutions' effectiveness, a number of empirical papers have estimated reduced form models to show that communities and individuals richer in social capital enjoy better health. Several authors (Cooper et al. (1999), Lochner et al. (1999), Macinko and Starfield (2001) and Muntaner et al. (2001)) offer reviews of the empirical literature on social capital and health. A very complete survey is that of Islam et al. (2006). Most studies adopt multilevel estimators and look only at the association between health and alternative measures of social capital, either at the individual or at the community level or both. Typically these studies estimate reduced form models. Among many others, Veenstra (2000), Petrou and Kupek (2008), Fujisawa et al. (2009) and Snelgrove et al. (2009)[7] focus on a single country, taking into account several possible measures of social capital. Giordano and Lindstrom (2010) uses BHPS to analyze how over-time variations in social capital, between 1999 and 2005, are associated with variations in self-reported health and find a positive relation between social capital and good general health. Giordano and Lindstrom (2011) again use BHPS and find a positive association between generalized trust and psychological health. Brunner (1997),

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[5] Taking one of the usual distinctions proposed in the literature, this definition considers social capital as a cognitive object (rather than structural).
[6] Generalized trust is defined over the answer that individuals provide to questions of the kind "Generally speaking, would you say that most people can be trusted?" alike to that originally included in the Value Social Survey. Thus generalized trust is an individual belief about the likelihood that other people are cooperative or instead adopt opportunistic behaviors.
[7] Snelgrove et al. (2009) look at the UK and use BHPS.

Brunner and Marmot (1999), Shkolnikova et al. (2009) find that lack of social support may increase the risk of heart diseases, susceptibility to infection, diabetes, high blood pressure and high levels of cortisol.

Poortinga (2006) and Mansyura et al. (2008) pool many countries and estimate the effect of individual and community level social capital, by means of multilevel methods. Both find that only the former is significant.

Most analysis report associations. Thus the estimated effect of social capital cannot be considered causal, since social capital is likely to be endogenous, as forcefully shown by Durlauf and Fafchamps (2005). The causal impact of social capital on individual health has been assessed in several recent papers (Rocco et al. 2013, d'Hombres et al., 2010; Sirven and Debrand, 2008; Folland, 2007; Scheffler, Brown and Fulton, 2008). The first four papers rely on instrumental variables to identify the effect of social capital while the last one exploits panel data and introduce individual fixed effects to control for unobservable heterogeneity.

## 4. Data

Data are from the British Household Panel Survey (BHPS) where information on both individual trust and health is included in the waves of 1999, 2001, 2004, 2006, 2008. About 12,000 people have been interviewed in each wave: more than 90 percent have been contacted in at least two waves, and 37% have been interviewed in all the waves between 1999 and 2008, i.e. about 4,900 individuals. The time span covered by the data is long enough to find some health and social capital variation, although especially trust is rather persistent over-time. For instance, among individuals observed in all waves, about 50 percent experienced at least one variation in their reported trust overtime, and 30 percent 2 or more variations.

Social capital is measured by the reported level of generalized trust, an indicator widely used in the literature on social capital and health[8]. The use of this indicator has been theoretically supported by Guiso et al. (2011) given that it can be readily assimilated to a belief about people willingness to cooperate. In the BHPS, respondents are asked: "Generally speaking, would you say that most people can be trusted, or that you can't be too careful in dealing with people?". They can choose between "Most people can be trusted" or "Can't be too careful". Our measure of trust is then a dummy variable taking on 1 when people answer "Most people can be trusted".[9]

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[8] Borgonovi, 2010; D'Hombres et al, 2010; Giordano and Lingstrom, 2010; Hurtado et al, 2011; Mansyur, 2008; Petrou and Kubek, 2008; Portinga, 2006b; Rostila, 2007; Snelgove et al, 2009; Giordano and Lindstrom (2010,2011) just to mention few contributions.

[9] BHPS includes other potential measures of social capital, such as (active) membership to formal and informal social organization, that I have chosen to not consider in this paper for a number of reasons: first,

In all waves, people are also asked to report the presence or absence of 13 specific health problems: 1) Problems or disability connected with: arms, legs, hands, feet, back, or neck (including arthritis and rheumatism); 2) Difficulty in seeing (other than needing glasses to read normal size print); 3) Difficulty in hearing; 4) Skin conditions/allergies; 5) Chest/breathing problems, asthma, bronchitis; 6) Heart/blood pressure or blood circulation problems; 7) Stomach/liver/kidneys or digestive problems; 8) Diabetes; 9) Anxiety, depression or bad nerves; 10) Alcohol or drug related problems; 11)  Epilepsy; 12) Migraine or frequent headaches; 13) Other health problems.

Moreover very detailed information about education, employment condition and type of occupation, marital status, region of residence, income, financial situation and number of accidents experienced are included.

Only residents in England  are retained, excluding Wales, Scotland and Northern Ireland, in order to increase the cultural homogeneity and limit problems of reporting heterogeneity in generalized trust. Moreover and more importantly, the analysis focuses on the initially healthy people, defined as those reporting no health problem when they first enter into the dataset. This is to eliminate a kind of problematic left censoring: the health history prior to the observed time window (between 1999 and 2008) might have influenced initial level of social capital  in a way that cannot be account for given that social capital is not reported prior to 1999. In other words, initial social capital could not be considered as pre-determined for people entering in the observation period with some ongoing disease. Conversely, initially healthy individuals are homogenous in the sense that their initial level of social capital differs only for exogenous reasons other than their past health history.

Furthermore, only individuals included in the last three to five waves are selected, in order to have enough time variation within individual. Eventually, the sample is composed of about 2100 individuals. Summary statistics of some key variables are reported in Table 1. In the final sample average age is 45 and for about 90 percent of the observations age is below 66. Thus, unsurprisingly, the largest majority of the individuals in our sample, 78 percent, is formally

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they are conceptually less well founded than generalized trust, since they are more an outcome of social capital than social capital itself; second, they are more prone to reverse causation since individual social life likely responds more to health, compared to a belief that is grounded on long years of experience and is typically quite persistent; third these information are available only for a subset of waves  so that estimates obtained by using generalized trust and those adopting alternative measures of social capital would not be fully comparable (active membership to formal and informal social organization would be available in 2004, 2006 and 2008).

employed. About 55 percent attained a upper secondary degree and about 28 percent a higher education degree[10].

Eleven diseases (out of 13) are retained: "Epilepsy" and "Alcohol or drug related problems" have been pooled to the residual category "Other health problems" because very few people in the sample report of suffering from them. The prevalence of each disease is indicated in Table 2. Note that for most diseases the prevalence is rather low. This is unsurprising in a sample of initially healthy people. Nevertheless, on average, each respondent reports the occurrence of about one disease within the ten years between 1999 and 2008 and about one quarter report 2 or more diseases.

**[Table 1 ABOUT HERE]**

**[Table 2 ABOUT HERE]**

## 5. Identification Strategy

Two problems, unobserved heterogeneity and reverse causation, make the identification of the influence of social capital on health difficult to achieve. Unobserved heterogeneity refers to the fact that unobservable variables such as individual preferences or family background might determine both social capital and health. Recently, Guiso and al. (2008a) have argued that individual beliefs about the trustworthiness of other person are transmitted from generation to generation in the household. At the same time the recently growing literature on endogenous preferences suggests that other traits of individual preferences, such as patience and (likely) risk aversion are taught by parents (Doepke and Zilibotti, 2005). Indeed family background contributes to both individual social capital and, by influencing sons' preferences, alters sons' decisions about their health.

Reverse causation arises because not only social capital could influence health, but also health could influence social capital. For instance, sick or disabled people might have less intense social interactions and then have less opportunities to update their believes about others' trustworthiness (Guiso et al. 2008a). Also social capital reporting could be altered by health, as health is likely to influence the mood of the respondents.

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[10] While education is predetermined for individuals in our sample and does not vary between 1999 and 2008, the occupational status may change along ten years considered for a non-negligible proportion of individuals.

Both phenomena tend to produce positive spurious correlation between social capital and health. Therefore simple regression analysis that estimate associations would yield too optimistic results.

This paper proposes an identification strategy that inspires to duration/survival analysis but that aims to bypass the difficulties that duration models face to properly deal with unobserved heterogeneity and time-varying explanatory variables (see Cameron and Trivedi, 2005, chapters 17 and 18 ; Hosmer and Lemeshow, 1999, p.248 )[11].

Summarizing, the proposed empirical strategy, alike duration models, exploits transitions from a status to another, i.e. from the healthy status to a disease status. However, different from duration models, which are based on the probability that transition occurs at any given time, here only the occurrence or absence of a transition matters, while the timing of the transition matters only to the extent that it induces variation in the explanatory variables. For each individual, many transitions are pooled together by simultaneously looking at multiple diseases.[12] Identification depends on the fact that the level of individual social capital prevailing before each transition varies across transitions. This within-individual variation is thus exploited to control for unobservable and time-invariant characteristics (such as the individual generic propensity to fall ill, family background and preferences). The effect of time-varying shocks influencing both social capital and health conditions is directly accounted for by a number of individual controls.

To avoid terminological confusion with the duration model framework, that despite similarities in the intuition is quite different technically-wise, I have preferred to refer to transitions as switching points. Precisely, a switching point is defined as the pair composed by the (possible) occurrence of a disease and the level of social capital reported immediately before this occurrence.

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[11] In fact standard duration model might accommodate for unobserved heterogeneity only if the individual effect is orthogonal to all the included controls. This is at odds with the literature and the evidence suggesting that unobservable individual characteristics such as time-preferences and risk aversion are related to social capital (see for instance Rocco and Fumagalli, 2013, on this point).
Furthermore, duration models are inherently cross-sectional. Controls should be pre-determined at the beginning of the observation interval and vary only across individuals to guarantee the assumption of strict exogeneity. In principle it is possible to extend the analysis to time-varying controls and panel structures and exploit the additional information provided by the over-time variation. However, this is quite limited in practice precisely because reverse causation will produce a mechanic violation of the strict exogeneity assumption underlying duration analysis (see Cameron and Trivedi, 2005, p.598).
[12] Precisely, I focus exclusively on the first transitions for each considered disease. The possible selection concerns arising from using only the first transition are limited by the fact that I am considering long lasting and chronic diseases. Particularly, the latter may occur only once and never heal, so that there will exist at most one transition from healthy to sick state.

The initial panel of individual-year observations is re-arranged into a different panel of individual-disease observations. In the latter the outer dimension corresponds to the individual identifier ($i$) and the inner dimension corresponds to the disease identifier ($d$). Eleven long-lasting and chronic diseases are considered[13] and therefore eleven records, one for each possible disease, are associated to each individual. This implies that all observations (the statistical units) of the dataset shall be indexed by the pair *(i,d)*. Each observation corresponds to a switching point that is codified in two variables, the event variable $P_{id}$ and the social capital indicator $S_{id}$. If disease $d$ ever occurred between 1999 and 2008 to individual $i$, the variable $P_{id}$ takes the value 1. Otherwise it takes zero. Attention is restricted to those individuals who are in good health (i.e. that do not report any diseases at the onset of the observation period) to avoid that their health history prior to 1999 influence their level of social capital reported in 1999.[14] The second component of the switching point, the variable $S_{id}$, collects the level of social capital declared by individual $i$ (in the wave of data) before the occurrence of disease $d$. By construction, social capital is then pre-determined with respect to disease $d$, a feature that reduces concerns of endogeneity.

Eventually, two vectors $P_{id}$ and $S_{id}$ , collecting all possible switching points, are obtained after data reshaping. Note that the time dimension is left implicit by this representation, but it crucially determines within-individual variation in $S_{id}$.[15]

Figure 1 helps to clarify how switching points are defined. The Figure represents the situation of a hypothetical individual, initially healthy, observed five times, denoted from t0 to t4, at regular intervals, along a period of given and fixed length. Suppose for simplicity that there exist information about five diseases, from D1 to D5, where D5 is a residual category encompassing all other diseases excepting D1, D2, D3 and D4. The timing of the first occurrence of a disease is indicated by a triangle. If a disease never occurs between t0 and t4 a star is marked at t0. Next, the occurrence of each disease is associated with the level of individual social capital at the time just preceding the disease onset. In the Figure circles indicate the timing at which these information is collected.[16] If a disease does not occur, the considered level of social capital is that reported at time t0. For any disease, the combination of one triangle (or star) and one circle

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[13] Short-term and recurrent diseases are excluded because it is unlikely they have permanent effects on social capital. Recall that one disease is residual and it is defined as "other diseases".

[14] Concerns relative to possible sample-selection resulting from the decision of looking only at the initially-healthy people are largely mitigated by the inclusion of individual fixed effects, which account for the time-invariant characteristics which are associated with selection into the sample.

[15] In the present context fixed effect models that rest only on overtime variation for identification, are of little utility since social capital is very persistent overtime for any given individual.

[16] Also other relevant time-varying information, such as his/her income, kind of occupation and marital status are collected at this timing.

graphically identify the timing of a switching point, i.e. the timing at which an individual condition changes (a disease before absent has now occurred) and the timing at which the information about individual social capital is collected.

On the reshaped data, the following empirical model is estimated

$$P_{id} = \alpha_0 + \alpha_1 S_{id} + \alpha_2 S_{id} * \delta_d + \alpha_3 X_{id} + \mu_i + \delta_d + \varepsilon_{id} \quad i = 1, \ldots, N; \quad d = 1, \ldots, 11$$

where $P_{id}$ is the indicator variable which captures the possible occurrence of disease $d$ to individual $i$, and $S_{id}$ and $X_{id}$ are the levels of individual $i$'s social capital and other relevant time-varying characteristics declared before the occurrence of disease $d$. Specificities in the effect of social capital across diseases are captured by a full set of interactions between social capital and disease-specific dummies $S_{id} * \delta_d$. Therefore the marginal effect of social capital on the probability that disease $d$ occurs is given by $\alpha_1 + \alpha_2 * \delta_d$. Individual fixed effects $\mu_i$ are included to control for all time-invariant and individual-specific features, which can be correlated with both social capital and health, and, finally, a full set of disease dummies $\delta_d$ are included to control for disease-specific prevalence rates. The error term $\varepsilon_{id}$ is allowed to be clustered at individual level.

Individual controls included in $X_{id}$ are time-varying and, particularly, they are evaluated at the time of the switching point. They are: the number of accidents occurred since 1997, employment condition and type of occupation, log of household annual income, marital status, and region of residence[17]. These controls account for possible time-varying shocks which might simultaneously influence social capital and health. For instance variation in the marital status accounts for partner's death or separation. Variation is the region of residence dummies, in employment condition and occupation account for other shocks important to individual social life and health.

A complication that we have not discussed yet is the possible interdependence between switching points. The first insurgence of a particular disease, especially if hampering social relations (such as sight and hearing problems), likely slows down the subsequent process of social capital accumulation and in turn influences the likelihood that other diseases occur in the future[18]. For instance in Figure 1, the occurrence of D1 at time t2 might alter the contemporaneous level of social capital that is associated to the occurrence of D3. Treating

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[17] Of course, individual fixed effect account for all individual time-invariant characteristics, such as gender, the age when they entered our dataset and predetermined variables, such as schooling that does not vary among adult people.

[18] Moreover, some diseases might be complications of previously developed chronic conditions. For instance the probability of eyesight, heart or kidney problems is larger among the diabetics.

observations D1 and D3 as independent rather than part of unique process could then be misleading.[19] To avoid this problem, an alternative procedure, illustrated in Figure 2, is worth considering. If two diseases occur, say, at time t2 and t3, the level of individual social capital (and other factors) to be recorded is that reported at time t=min(t2,t3)-1=t1. More generally the relevant timing to consider is that of the onset of the fist disease, whatever the number of diseases occurred in the period of time considered.

In what follows, *Specification 1* will refer to the procedure corresponding to Figure 1 and *Specification 2* to that corresponding to Figure 2.

Compared to *Specification 1*, *Specification 2* modifies only the way information reported by individuals experiencing two or more diseases is codified in the switching points: these people count for less than one quarter of the sample.

**[Figure 1 ABOUT HERE]**

**[Figure 2 ABOUT HERE]**

## 6. Results

Model (1) is estimated by means of an individual fixed-effect linear probability model. Estimates of the parameters associated to $S_{id}$ and $S_{id}*\delta_d$ are reported in Table 3 (to save space all remaining controls have been omitted from the table). Columns 1-3 refer to *Specification 1* and columns 4-6 refer to *Specification 2*. Columns 1 and 4 report the estimates of the basic model omitting controls $X_{id}$, while in columns 2 and 5 controls are included. Finally in columns 3 and 6 the model has been restricted to the subset of chronic diseases (breathing, heart, diabetes, sight and hearing problems). Although focusing on chronic diseases is interesting per se, the main purpose of this restriction is that of shedding further light on the role played by *Specification 2*. By definition, chronic diseases are never-ending. Hence, it is likely that the level of social capital reported after their occurrence changes permanently. This is the situation where results produced by *Specification 2* should differ the most from those of *Specification 1*.

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[19] Competing risk models would not be suitable in this context. Indeed they assume "exclusive spells" (see Cameron and Trivedi, p.642), that is only one of the risks can actually materialize. After this event, all other risks are censored. The typical application is that analyzing the role of different causes of death, where people die only once and for only one cause, even if multiple risks are competing to achieve this (unfortunate) outcome. In our context instead multiple diseases might coexist. On top of this the problem of unobserved heterogeneity would remain unsolved.

Estimates indicate that individual trust significantly reduces the probability of occurrence of all diseases. The size of the reduction is quite large, around 14-18 percent, varies little across specifications and is scarcely influenced by the inclusion or omission of socio-economic controls. Although these effects appear quite large, it should be kept in mind that the available measure of social capital is dichotomous and implies a comparison between rather extreme level of trustfulness ("most people can be trusted" vs "you can't be too careful").

The impact of each disease is similar, as the estimates corresponding to the interactions indicate. Only for "hearing problems" and "other problems", trust has a systematically smaller impact on their likelihood. When only chronic diseases are considered, estimates are larger, but only the estimates corresponding to Specification 2 (column 6) are significantly larger. This fact indicates that the influence of a disease on subsequent social capital level (that is neutralized in Specification 2) is sizeable and tends to reduce the magnitude of the effect of trust on the probability of experiencing a disease.

Next, Table 4 shows how results change by considering different population subsets separately. The potentially more reliable *Specification 2* is adopted and the full set of diseases and controls are included. Thus, the benchmark against which to compare estimates of Table 4 is column 5 of Table 3. Of course the reduced sample size makes more difficult to get precise estimates. However looking at the point estimates is informative enough to draw few broad conclusions. In column 1 and 2 the model is estimated separately on females and males respectively. Although the impact of trust is larger for males, the difference between genders is modest. Next, in columns 3 and 4 people aged less than 50 are compared with the over-50. The choice of this threshold is motivated by the fact that the fifties are the age at risk for cardiovascular diseases and other chronic diseases. Surprisingly, the effect of trust is stronger among the younger, although the younger tend to have less social capital than the elderly, as Guiso et al. (2008a) have shown, and as it is the case also in our sample (37 percent of the of the younger declare to trust others compared to 51 percent of the aged 50+).

Moving to the level of education, columns 5 and 6, people with a university degree enjoy higher returns of social capital than the less educated. Typically, they have larger social networks and so more opportunities for social capital to display its beneficial influences. Indeed the more educated people tend to report much more trust than the less educated (59 percent compared to 39 percent).

Finally in columns 7 and 8 the residents in regions with average levels of trust below the median (0.44) are contrasted with those residing in regions above the median. The impact of trust is larger for people residing in areas richer of social capital. This result indicates that individual and

community social capital are indeed complements, consistently with Guiso et al.'s theory of social capital. Social capital is not an asset which can be used in isolation, as it could be for instance human capital. Individual social capital produces positive consequences only in a favorable social environment.

**[Table 3 ABOUT HERE]**

**[Table 4  ABOUT HERE]**

## 7. Conclusions

This paper provides additional evidence in favor of a positive relationship between social capital and health. So far the largest part of the literature has looked at self-reported indicators of general health and only recently it has paid attention to the issues of unobserved heterogeneity and reverse causation. The contribution of this paper is twofold. First, it adopts as health outcomes the more objective occurrence of specific diseases within a time window of about ten years. This strategy allows to isolate the effect of social capital on "true" health from the effect on the individual assessment and reporting of health. Second, the paper suggests a previously overlooked strategy to avoid reverse causation and to account for unobserved individual heterogeneity. Evidence of a strong causal beneficial effect of social capital has emerged: individuals who declare that "most people can be trusted" are about 14 to 18 percent less likely to incur in any disease, establishing that social capital does influence "true" health and not only how health is assessed and reported by the individuals. Results are robust to two alternative specifications and to the inclusion or omission of individual socio-economic controls. Furthermore, the effect of social capital appears to be larger among the younger, the more educated and among those living in regions richer in social capital.

Estimates also indicate that trust affects the likelihood of all diseases to a similar extent. The latter result suggests that social capital does not enter into the medical etiology specific to each disease, but rather that it modifies the generic propensity of an individual to fall ill. This interpretation fits well with the mechanisms, mentioned in the introduction, that explain the link between social capital and general health. Indeed, all these mechanisms influence the socio-economic context where people live, creating conditions favorable to health protection. For instance, social capital favors the diffusion of health-relevant information and the creation of social safety nets, discourages all risky behaviors by increasing their opportunity costs, favors

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political cohesion in a community allowing for a larger influence in the repartition of the national resources devoted to public health.

Several limitations of the current study can be mentioned. The most important is that there are no other suitable measures of social capital in the dataset at use. This prevents to check the robustness of our estimates by using alternative indicators equally consistent with Guiso et al. definition of social capital. The second limitation is that, given the timing of data collection of BHPS, social capital is observed two years before the occurrence of the corresponding disease, while the optimal timing would be immediately before the occurrence of the disease. Hence the social capital indicator could be affected by an error-in-variable. The third limitation is that the time span covered by data is at most 10 years and in some cases just 8 or even 6 years. Thus, given the high persistence of social capital, the number of available switching points for each individual is rather small, a feature that reduces estimates precision (as it is particularly apparent in the analysis carried over smaller samples). Another limitation is the fact that the presence or absence of a disease is declared by the respondents and might be influenced by individual education and socioeconomic status. Indeed, typically, the more educated and the better off are more aware of their health conditions. Also the trust variable is not without potential drawbacks. First, generalized trust is dichotomous, implying smaller variation and imprecise correspondence with the latent degree of trustfulness which is likely to be continuous. Second, generalized trust is self-reported and subject to mis-reporting. Deviations in reporting might depend, for instance, on respondents' willingness to please the interviewer.

Undoubtedly, further research is necessary in this field and, particularly, better data are necessary to get more reliable results, although the evidence published in the recent years is almost unanimous in pointing to the existence of a positive causal influence of social capital on health.

This paper contributes to the view that social factors do play an important role in shaping individual health conditions, a role perhaps overlooked by health policy so far.

It is widely acknowledged that education and income cannot be the only cause for differences in mortality and morbidity and that other social factors contribute to individual health and health inequality. Already Marmot and Wilkinson in their seminal book (Marmot and Wilkinson, 2005) included social support, social cohesion and social exclusion among the social determinants of health. Conceptually, social capital is at the root of many social determinants, and particularly social cohesion and social support.

However, social capital, as far as it is defined as an individual attitude to cooperate or an individual belief about others' willingness to cooperate, is largely outside the reach of governments, which can do little to increase the stock of social capital in the society, at least in the short run, because beliefs are highly persistent and, according to Guiso et al. (2007, 2008a), they are updated only after a long period of experienced cooperation.

Governments have a role in promoting the rule-of-law, a condition that certainly induce individuals to expect cooperation from their partners in the domain of formalized relationships, such as contracts and economic transactions. It is much less obvious that rule-of-law might play a role in the domain of informal relationships, which is crucial for the pathways through which social capital benefits health. Take for instance information diffusion, or informal support in case of need: these mechanisms are typically informal and do not respond to government legislation.

Nevertheless, governments can certainly favor the deployment of social capital's beneficial effects. For instance, they could support the operations of informal safety nets by promoting more flexible working time, by supporting voluntary activities, or by permitting that someone outside the circle of strict relatives might go to see a hospitalized person beyond visiting hours and obtain reserved information from the hospital and the doctors if properly authorized.

**References.**

Alesina, A., and P. Giuliano (2007). "The Power of the Family." Working paper, Harvard University.

Bailis, D.S.,  Segall, A. and  Chipperfield, J.G.  (2003). "Two views of self-rated general health status", *Social Science & Medicine* 56:203–17.

Bourdieu P. (1977). Outline of a Theory of Practice. Cambridge University Press: Cambridge.

Brunner, E. and Marmot, M. (1999) Social organization, stress, and health. In: Marmot. M. and Wilkinson, R. G. (eds) Social Determinants of Health pp17-43. Oxford: Oxford University Press.

Brunner, E. J. (1997). Stress and the biology of inequality. British Medical Journal 314: 1472-6.

Cameron, A.C. and Trivedi, P.K. (2005) *Microeconometrics*. Cambridge University Press

Coleman, J.S. (1990). Foundations of Social Theory Cambridge, MA: Harvard University Press;.

Cooper, H., Arber, S., Fee, L. and Ginn, J. (1999). The Influence of Social Support and Social Capital on Health: a review and analysis of British data. London: Health Education Authority.

D'Hombres B., Rocco L., Suhrcke M., Mckee M. (2010), Does Social Capital Determine Health? Evidence from Eight Transition Countries, Health Economics 19(1): 56–74

Djankov, S., Glaeser, E., La Porta, R., Lopez-de-Silanes, F., and A. Shleifer (2003). "The New Compartive Economics." Journal of Comparative Economics, 31, 595–619

Doepke, M. and F Zilibotti (2005) "Social class and the spirit of capitalism", Journal of the European Economic Association, 3(2-3): 516-524

Durlauf, S.,and Fafchamps, M. (2005). Social capital. In Handbook of Economic Growth, vol. 1B, Durlauf S, Aghion P. (eds). North Holland: Amsterdam, 1639–1699

Folland, S. (2006), "Value of life and behavior toward health risks: an interpretation of social capital" Health Economics. 15: 159–171

Folland, S. (2007). Does "community social capital" contribute to population health? Social Science and Medicine 64, 2342-2354.

Folland, S., Islam, M.K. and Kaarbøe, O.M. (2012) "Social Capital and Types of Illness: A Look at How Its Results Are Achieved", mimeo, presented at the IV workshop of the Global Network on Social Capital and Health, Padova (Italy) October 4-5, 2012.

Fountain, J. E. (1997). "Social Capital: A Key Enabler of Innovation in Science and Technology." In Investing Innovation: Toward a Consensus Strategy for Federal Technology Policy, edited by L. M. Branscomb and J. Keller. MIT Press

Fujisawa, Y., Hamano T, and S. Takegawa (2009) "Social capital and perceived health in Japan: An ecological and multilevel analysis", Social Science & Medicine 69:500–505

Glaeser, E., Sacerdote, B.,  and J.A. Scheinkman (1995). "Crime and Social Interactions." Quarterly Journal of Economics, 111, 507–548

Glaeser E, Laibson D and Sacerdote B. 2002. An economic approach to social capital. The Economic Journal 112(483):437–458.

Goldin, C., and L. Katz (2001). "Human Capital and Social Capital: The Rise of Secondary Schooling in America, 1910–1940." In Patterns of Social Capital, edited by R. Rotberg. Cambridge University Press.

Giordano, G.N., and Lindstrom, M. (2010). The impact of changes in different aspects of social capital and material conditions on self-rated health over time: A longitudinal cohort study. Social Science & Medicine 70 :700-710

Giordano, G.N., and Lindstrom, M. (2011). Social capital and change in psychological health over time. Social Science & Medicine 72 : 1219-1227.

Guiso, L., Sapienza, P., and L. Zingales (2004). "The Role of Social Capital in Financial Development." American Economic Review, 94(3), 526–556

Guiso, L., Sapienza, P., and L. Zingales (2007). "Long-Term Persistence." Working paper, University of Chicago

Guiso, L., Sapienza, P., and L. Zingales (2008a). "Alfred Marshall Lecture Social Capital As Good Culture", Journal of the European Economic Association, 6(2–3):295–320

Guiso, L., Sapienza, P., and L. Zingales (2008b). "Trusting the Stock Market." Journal of Finance, 63(6):2557 - 2600.

Guiso, L., Sapienza P.,  and L. Zingales (2011) *Civic Capital As The Missing Link*, in Benhabib, J., Bisin, A. and Matthew Jackson (eds. ) the Handbook of Social Economics,  North Holland.

Hosmer, D.W. and Lemeshow S. (1999) *Applied Survival Analysis*. John Wiley & Sons Inc

Huisman, M. and Deeg, D.J.H. (2010) "A commentary on Marja Jylhä 's `What is self-rated health and why does it predict mortality? Towards a unified conceptual model'(69:3, 2009, 307–316)", Social Science & Medicine 70:652–54.

Islam, M. K., J. Merlo, I. Kawachi, M. Lindstrom, and U. G. Gerdtham (2006). Social capital and health: Does egalitariamism matter? A literature review. International Journal for Equity in Health 5, 1-28.

Jylhä, M. (2009) "What is self-rated health and why does it predict mortality? Towards a unified conceptual model", Social Science & Medicine 69 : 307–16

Jylhä, M. (2010) "Self-rated health between psychology and biology. A response to Huisman and Deeg", Social Science & Medicine 70:655–57.

Kawachi I., Kennedy B. (1997). The relationship of income inequality to mortality: does the choice of indicator matter? Social Science & Medicine 45: 1121–1127.

Kawachi, I., & Berkman, L. F. (2000). Social cohesion, social capital, and health. In L. F. Berkman, & I. Kawachi (Eds.), Social Epidemiology (pp. 174–190). New York: Oxford University Press.

Kawachi, I., Subramanian, S.V., and D. Kim (2008). Social Capital and Health, Springer

Knack S. and Keefer P. (1997). "Does Social Capital have an Economic Impact? A Cross-Country Investigation." Quarterly Journal of Economics, 112(4), 1252–1288

La Porta, R., Lopez de Silanes F., Shleifer A., and R. Vishny (1997). "Trust in Large Organizations." American Economic Review, 87(2), 333–338

Lochner, K., Kawachi, I. and B. P., Kennedy  (1999). Social capital: a guide to its measurement. Health and Place 5: 259-70.

Macinko, J. and Starfield, B. (2001). The utility of social capital in research on health determinants. The Milbank Quarterly 79: 387-427.

Mansyura, C.,  Amick, B.C., Harrist, R.B., Franzini, L. (2008). "Social capital, income inequality, and self-rated health in 45 countries", Social Science & Medicine 66: 43–56

Marmot, M. and Wilkinson, R. (2005). *Social determinants of health*, Oxford: Oxford University Press.

Muntaner, C., Lynch, J. and Davey Smith, G. (2001). Social capital, disorganized communities, and the third way: understanding the retreat from structural inequalities in epidemiology and public health. International Journal of Health Services 31: 213-37.

Murgai R, Winters P, Sadoulet E, de Janvry A. (2002). Localized and incomplete mutual insurance. Journal of Development Economics 67: 245–274.

Petrou, S. and E. Kupek (2008). Social capital and its relationship with measures of health status: Evidence from the health survey for England 2003. Health Economics 17, 127-143.

Poortinga, W. (2006). Social capital: An individual or collective resource for health? Social Science and Medicine 62, 292-302.

Putnam R.D., Leonardi, R., Nanenetti R. (1993). *Making Democracy Work: Civic Traditions in Modern Italy*. Princeton University Press: Princeton, NJ.

Rocco, L., Fumagalli, E. and Suhrcke, M. (2013). From social capital to health – and back, Health Economics, forthcoming

Rocco, L. and Fumagalli, E. (2013). The empirics of social capital and health, in Folland, S. and Rocco, L. (eds.) The Economics of Social Capital and Health, World Scientific

Scheffler, R.M., Brown, T.T. (2008). Social capital, economics, and health: new evidence. Health Econ Policy Law, 3(4):321-31

Scheffler, R., Brown, T.T. and Fulton B. (2008) "Child health outcomes and their relationship with social capital or social capital and education. The relationship between Social Capital and the Health and Educational Outcomes of Children: the Role of Parental Education", mimeo

Shkolnikova M. et al. (2009) "Biological mechanisms of disease and death in Moscow: rationale and design of the survey on Stress Aging and Health in Russia (SAHR)" BMC Public Health 9(293):1-16

Sirven, N. and T. Debrand (2008). Promoting Social Participation for Healthy Ageing. A counterfactual analysis from the survey on health, ageing, and retirement in Europe. IRDES Working Paper (7).

Solow R. 1995. Trust: the social virtues and the creation of prosperity (book review). The New Republic 213: 36–40.

Snelgrove, J.W., Pikhart, H. and M. Stafford (2009) "A multilevel analysis of social capital and self-rated health: Evidence from the British Household Panel Survey" Social Science & Medicine 68:1993–2001

Veenstra, G. (2000). Social capital, SES and health: an individual-level analysis. Social Science and Medicine 50: 619-29.

**Figure 1 – Specification 1**
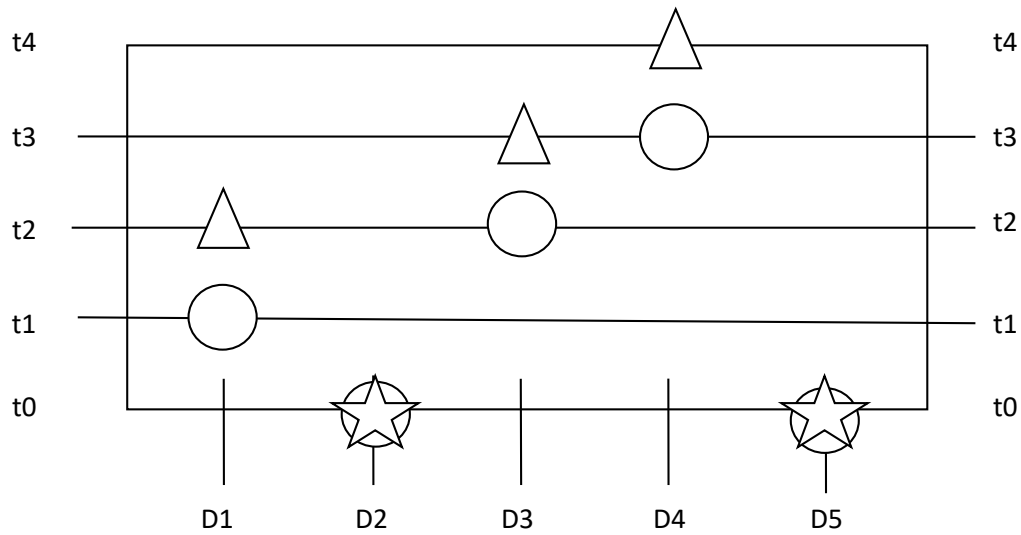


Legend: D1-D5 indicate diseases; t0-t1 indicate points in time; triangles indicate the first occurrence of the disease reported on the horizontal axis; stars indicate that the corresponding disease on the horizontal axis has never occurred in the period between t0 and t4; finally, circles indicate the time at which information on individual social capital and other controls is collected for each possible disease.

**Figure 2 – Specification 2**
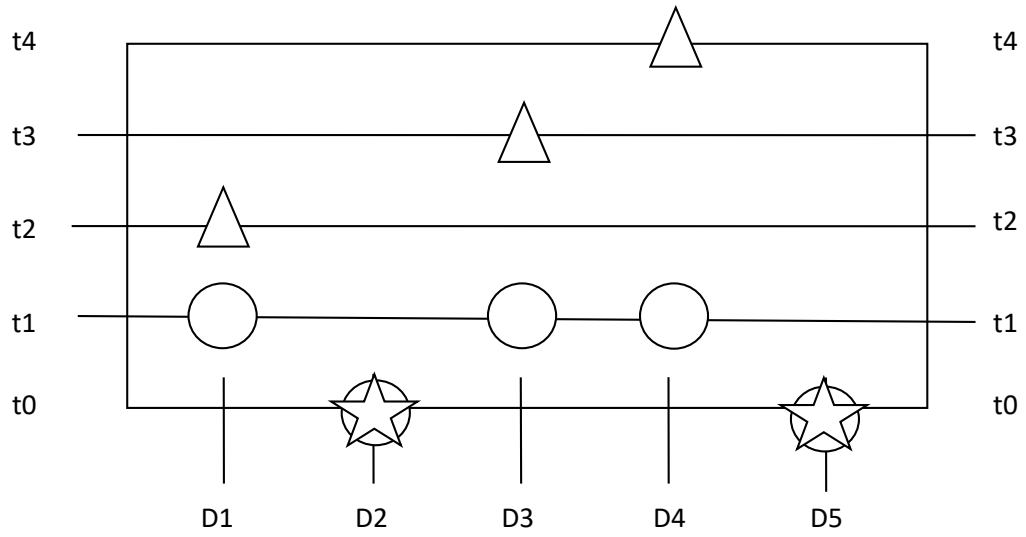


Legend: see Figure 1

**Table 1 – Summary statistics**

| variable | mean | std. dev. | min | max |
|---|---|---|---|---|
| number of diseases reported between 1999 and 2008 | 1.000 | 1.191 | 0 | 9 |
| trust* | 0.424 | 0.487 | 0 | 1 |
| age | 45.480 | 15.107 | 21 | 90 |
| log annual income | 8.645 | 2.119 | 1.098 | 12.291 |
| number accidents since 1997 | 0.112 | 0.355 | 0 | 3 |
| married* | 0.512 | 0.497 | 0 | 1 |
| employer, manager, or self-employed* | 0.148 | 0.350 | 0 | 1 |
| non manual worker* | 0.382 | 0.478 | 0 | 1 |
| manual worker* | 0.205 | 0.398 | 0 | 1 |
| own account worker* | 0.039 | 0.191 | 0 | 1 |
| agricultural worker* | 0.006 | 0.078 | 0 | 1 |
| non employed* | 0.220 | 0.407 | 0 | 1 |
| female* | 0.485 | 0.500 | 0 | 1 |
| higher education* | 0.278 | 0.448 | 0 | 1 |
| upper secondary education* | 0.556 | 0.497 | 0 | 1 |

Note: (*) dummy variable

**Table 2 – Disease Occurrence**

| diseases | never occurred | occurred | total |
|---|---|---|---|
| anxiety | 1,953 | 179 | 2,132 |
| arms-legs | 1,558 | 580 | 2,138 |
| breathing | 1,959 | 174 | 2,133 |
| diabetes | 2,085 | 49 | 2,134 |
| hearing | 2,020 | 114 | 2,134 |
| heart | 1,859 | 277 | 2,136 |
| migraine | 1,975 | 164 | 2,139 |
| others | 1,978 | 156 | 2,134 |
| sight | 2,035 | 99 | 2,134 |
| skin | 1,887 | 249 | 2,136 |
| stomach | 1,990 | 146 | 2,136 |

**Table 3. Baseline Estimates. The effect of trust on the probability that a disease occurs.**

| VARIABLES | Specification 1 | | | Specification 2 | | |
|---|---|---|---|---|---|---|
| | w/o controls | with controls | chronic diseases | w/o controls | with controls | chronic diseases |
| | baseline: anxiety | baseline: anxiety | baseline: breathing | baseline: anxiety | baseline: anxiety | baseline: breathing |
| trust | -0.143** | -0.165*** | -0.190** | -0.166* | -0.178** | -0.247** |
| | (0.0596) | (0.0574) | (0.0809) | (0.0859) | (0.0823) | (0.118) |
| trust*armslesgs | -0.0116 | -0.0182 | | -0.00196 | -0.00498 | |
| | (0.0214) | (0.0211) | | (0.0210) | (0.0205) | |
| trust*breathing | 0.00813 | 0.00349 | | 0.0204 | 0.0185 | |
| | (0.0158) | (0.0154) | | (0.0160) | (0.0154) | |
| trust*diabetes | 0.0246* | 0.0181 | 0.0128 | 0.0238* | 0.0194 | -0.000568 |
| | (0.0129) | (0.0128) | (0.0119) | (0.0131) | (0.0127) | (0.0123) |
| trust*hearing | 0.0396*** | 0.0322** | 0.0279** | 0.0436*** | 0.0407*** | 0.0223 |
| | (0.0148) | (0.0146) | (0.0137) | (0.0153) | (0.0149) | (0.0141) |
| trust*heart | 0.0266 | 0.0228 | 0.0192 | 0.0342* | 0.0322* | 0.0109 |
| | (0.0176) | (0.0171) | (0.0162) | (0.0178) | (0.0173) | (0.0165) |
| trust*migraine | 0.00861 | 0.00507 | | 0.00800 | 0.00617 | |
| | (0.0148) | (0.0147) | | (0.0149) | (0.0145) | |
| trust*others | 0.0434*** | 0.0348** | 0.0298** | 0.0384** | 0.0346** | 0.0146 |
| | (0.0157) | (0.0153) | (0.0145) | (0.0154) | (0.0148) | (0.0148) |
| trust*sight | 0.0210 | 0.0158 | | 0.0291** | 0.0231 | |
| | (0.0140) | (0.0138) | | (0.0145) | (0.0142) | |
| trust*skin | 0.0326* | 0.0252 | | 0.0320* | 0.0300* | |
| | (0.0170) | (0.0164) | | (0.0173) | (0.0166) | |
| trust*stomach | 0.0173 | 0.0143 | | 0.0269* | 0.0252* | |
| | (0.0154) | (0.0151) | | (0.0155) | (0.0150) | |
| Observations | 23490 | 23490 | 10672 | 23486 | 23486 | 10671 |
| R-squared | 0.058 | 0.107 | 0.080 | 0.057 | 0.100 | 0.077 |
| Number of individuals | 2160 | 2160 | 2141 | 2153 | 2153 | 2139 |

Note: Robust standard errors in parentheses, clustered by individual *** p<0.01, ** p<0.05, * p<0.1
Individual fixed effect linear probability model estimates. The dependent variable is occurrence of a disease (1 occurred / 0 never occurred between 1999 and 2008). Regressions include the social capital variable (trust) and its interactions with each disease (reported in the table) and control for the number of accidents occurred since 1997, employment condition and type of occupation dummies, log of household annual income, marital status, region of residence dummies and diseases-specific dummies (non reported). Columns 1-3 refer to Specification 1 and columns 4-6 to Specification 2. In columns 1 and 4 controls are omitted. In columns 3 and 6 only chronic diseases are retained.

**Table 4. Estimates separately by socio-economic conditions. The effect of trust on the probability that a disease occurs.**

| VARIABLES | By gender | | By age | | By education | | By place of residence | |
|---|---|---|---|---|---|---|---|---|
| | female | male | younger than 50 | older than 50 | higher education | lower education | below median gen. trust | above median gen. trust |
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| trust | -0.145 | -0.187 | -0.247** | -0.0214 | -0.377** | -0.124 | -0.180 | -0.250* |
| | (0.114) | (0.118) | (0.101) | (0.122) | (0.169) | (0.110) | (0.139) | (0.130) |
| trust*armslesgs | -0.0271 | 0.0108 | 0.00583 | -0.0897** | 0.0493 | 0.0263 | -0.0211 | 0.0344 |
| | (0.0307) | (0.0272) | (0.0224) | (0.0382) | (0.0340) | (0.0269) | (0.0299) | (0.0261) |
| trust*breathing | 0.0124 | 0.0187 | 0.0187 | 0.00306 | 0.0253 | 0.0632*** | 0.0122 | 0.0242 |
| | (0.0244) | (0.0190) | (0.0168) | (0.0295) | (0.0305) | (0.0195) | (0.0213) | (0.0203) |
| trust*diabetes | 0.0192 | 0.0130 | 0.0307** | -0.00271 | 0.0536** | 0.0307* | 0.0257 | 0.0135 |
| | (0.0203) | (0.0152) | (0.0137) | (0.0248) | (0.0266) | (0.0158) | (0.0162) | (0.0175) |
| trust*hearing | 0.0430* | 0.0321* | 0.0330** | 0.0321 | 0.0735*** | 0.0557*** | 0.0548*** | 0.0309 |
| | (0.0233) | (0.0185) | (0.0144) | (0.0304) | (0.0273) | (0.0187) | (0.0204) | (0.0197) |
| trust*heart | 0.0268 | 0.0319 | 0.00894 | -0.000564 | 0.0546* | 0.0764*** | 0.0261 | 0.0252 |
| | (0.0271) | (0.0216) | (0.0157) | (0.0355) | (0.0309) | (0.0225) | (0.0237) | (0.0226) |
| trust*migraine | -0.00202 | 0.0109 | 0.0121 | 0.00193 | 0.0489* | 0.00365 | 0.0267 | 0.00201 |
| | (0.0248) | (0.0158) | (0.0173) | (0.0253) | (0.0267) | (0.0187) | (0.0203) | (0.0186) |
| trust*others | 0.0490* | 0.0209 | 0.0256 | 0.0382 | 0.0259 | 0.0409** | 0.0383** | 0.0329* |
| | (0.0251) | (0.0163) | (0.0163) | (0.0281) | (0.0317) | (0.0183) | (0.0190) | (0.0199) |
| trust*sight | 0.00584 | 0.0334* | 0.0345** | 0.000746 | 0.0449* | 0.0395** | 0.0289 | 0.0157 |
| | (0.0225) | (0.0172) | (0.0157) | (0.0265) | (0.0264) | (0.0176) | (0.0199) | (0.0186) |
| trust*skin | 0.0147 | 0.0398* | 0.0378* | 0.0332 | 0.0838** | 0.0200 | 0.0650*** | -0.00600 |
| | (0.0265) | (0.0203) | (0.0201) | (0.0282) | (0.0346) | (0.0210) | (0.0246) | (0.0207) |
| trust*stomach | 0.0222 | 0.0231 | 0.0284 | 0.0154 | 0.0402 | 0.0489** | 0.0414* | 0.0109 |
| | (0.0239) | (0.0180) | (0.0173) | (0.0274) | (0.0283) | (0.0198) | (0.0214) | (0.0193) |
| Observations | 11353 | 12133 | 14966 | 8520 | 5338 | 13826 | 11332 | 12154 |
| R-squared | 0.098 | 0.121 | 0.147 | 0.138 | 0.198 | 0.117 | 0.107 | 0.092 |
| Number of individuals | 1043 | 1110 | 1372 | 781 | 491 | 1265 | 1142 | 1185 |

Note: Robust standard errors in parentheses, clustered by individual *** p<0.01, ** p<0.05, * p<0.1

Individual fixed effect linear probability models estimated separately on sub-samples indicated in each column head. All estimates are obtained by adopting Specification 2 with controls included (see note Table 3). Baseline disease is anxiety.