

Decker, Simon; Schmitz, Hendrik

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Health shocks and risk aversion

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801-2015

Health Shocks and Risk Aversion

Simon Decker and Hendrik Schmitz

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German Socio-Economic Panel (SOEP)
DIW Berlin
Mohrenstrasse 58
10117 Berlin, Germany

Contact: Uta Rahmann | soeppapers@diw.de



Health Shocks and Risk Aversion

Simon Decker^{a,c,d} and Hendrik Schmitz^{b,c,e}

^aUniversity of Duisburg-Essen

^bUniversity of Paderborn

^cCINCH Health Economics Research Centre

^dRuhr Graduate School in Economics (RGS Econ)

^eRWI Essen

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Abstract

Risk preferences are typically assumed to be constant for an individual across the life cycle. In this paper we empirically assess if they are time varying. Specifically, we analyse whether health shocks influence individual risk aversion. We follow an innovative approach and use grip strength data to obtain an objective health shock indicator. In order to account for the non-random nature of our data we employ regression-adjusted matching. Health shocks are found to increase individual risk aversion. The finding is robust to a series of sensitivity analyses.

JEL Classification: C81, D01, D81, I10, I12

Keywords: Risk Preferences, Health Shocks, Hand Grip Strength, Regression-Adjusted Matching

Correspondence to: Simon Decker, CINCH Health Economics Research Centre, Edmund-Körner-Platz 2, 45127 Essen, Phone: +49(201)-183-2926, Email: Simon.Decker@wiwinf.uni-due.de. Financial support by the Ruhr Graduate School (RGS) in Economics is gratefully acknowledged. Furthermore, we are grateful to Adam Pilny, Annika Herr, Gerrit Antonides, Helge Liebert, Jan Kleibrink, Johan Vikström, Markus Vomhof, Martin Fischer, Martin Karlsson, Matthias Westphal, Sarah Cattan, Stefanie Schurer and Vincenzo Carrieri for helpful and valuable comments. Moreover, we gratefully acknowledge comments and discussions at the CINCH health economics seminar (Essen), the RGS Jamboree (Duisburg) and the RGS Doctoral Conference (Essen). All errors are our own.

1 Introduction

Individuals face uncertainty in many essential economic decisions all through their life span. Accordingly, risk attitudes are a key determinant of individual utility. More risk-averse individuals are more likely to be unemployed (Diaz-Serrano and O'Neill, 2004), while other evidence suggests that individual risk aversion might positively influence wage growth (Shaw, 1996; Budria *et al.*, 2013) and also income (Hartog *et al.*, 2002). Moreover, a lower degree of individual risk aversion is correlated with higher cognitive abilities (Dohmen *et al.*, 2010) and higher education (Sahm, 2012). Important lifetime events such as marriage and childbirth are also linked to individual risk attitudes (Schmidt, 2008) as is the acquisition of health insurance (Barsky *et al.*, 1997; Schmitz, 2011; Vetter *et al.*, 2013).

Individual risk attitudes in economics are traditionally assumed to be constant over time (Stigler and Becker, 1977). However, recent empirical evidence suggests quite the contrary. For instance, childbirth (Görlitz and Tamm, 2015) or major economic downturns (Malmendier and Nagel, 2011; Cohn *et al.*, 2015) are found to be associated with higher individual risk aversion. Some find the same for increasing age (Dohmen *et al.*, 2011; Schurer, 2015) while other evidence suggests a U-shaped pattern of risk aversion across the life cycle (Cohen and Einav, 2007). Individuals who perceive their financial situation as having improved show a higher degree of risk willingness (Andersen *et al.*, 2008). Furthermore, there seems to be a socio-economic gradient in risk attitudes (Schurer, 2015). Those in more favourable positions tend to be more risk willing, while across the life cycle a specific pattern evolves. In all socio-economic groups alike there seems to be a decrease in individual risk willingness until middle age. Accordingly, the socio-economic gradient remains fairly constant until then. However, from this age onward those in favourable positions stabilise or even increase their level of risk willingness, while for the more disadvantaged it continues to decrease with age and the socio-economic gradient widens (Schurer, 2015).

Health is a central part of individual human capital with important direct and indirect implications for well-being and utility. After a health shock individuals are more likely to transit into labour market inactivity and into disability (Dano, 2005; García-Gómez, 2011), while

health shocks are also found to decrease individual earnings (Dano, 2005; Wagstaff, 2007). There exist good reasons to also expect an effect of health shocks on risk aversion. In this paper we empirically analyse this relationship. There might be two potential mechanisms linking these two economically and individually important variables. First of all, individual risk attitudes could potentially be state-contingent, i.e. dependent on the specific state of nature an individual is currently exposed to (Andersen *et al.*, 2008). We argue that different states of individual health might constitute different states of nature which imply different individual risk attitudes. Additionally, it might be that individuals are only imperfectly informed of their health status. In such a setting, health shocks could act as signals that reveal the true health status of individuals which is subsequently integrated in further behaviour (Clark and Etilé, 2002).¹ Such a setting would constitute a type of health state dependent utility that is related to what is analysed elsewhere (Finkelstein *et al.*, 2009, 2013).

A second possible mechanism could be that emotions such as fear cause changes in individual risk attitudes over time. Both, Guiso *et al.* (2013) and Cohn *et al.* (2015) provide evidence from laboratory experiments of this. In a study by Cohn *et al.* (2015), for instance, some participants are exposed to the threat of electric shocks and are significantly less risk-willing as a consequence. Similarly, health shocks might also create fear or other emotional reactions in individuals that influence their risk-taking behaviour.

As far as we are aware, only three other empirical studies so far analyse the relationship between health shocks and risk aversion directly. None of them focusses exclusively on this question. Moreover, the evidence is still inconclusive. Sahn (2012) studies a broad set of determinants for individual variation in risk attitudes in a sample of US-elderly from the Health and Retirement Study (HRS). Based on a correlated random effects model she finds no clear evidence with respect to individual health shocks, measured as the onset of a severe condition. Only one of the reported specifications suggests that there could be a health effect. Gloede *et al.* (2013) use interval regressions and analyse the effects of different kinds

¹Clark and Etilé (2002) propose a similar mechanism for the context of smoking. Current health is harmed by past smoking behaviour but the extent of this is not known with certainty. Health shocks serve as a signal of the true harm of smoking, which is subsequently integrated in future behaviour. A similar mechanism is proposed by Smith *et al.* (2001).

of self-rated shocks on the risk attitudes of individuals living in rural areas of Thailand and Vietnam. Among other shock measures they consider so-called *demographic* shocks, which are a combination of several events also containing individual health shocks. In Thailand no effects of such shocks are detected, while in Vietnam demographic shocks are associated with lower risk willingness. The authors argue that this could be driven by institutional differences between the health care systems of the two countries.

Schurer (2015) uses data from the German Socio-Economic Panel (SOEP) and detects a socio-economic gradient in individual risk attitudes as discussed above. In addition to that, Schurer (2015) differentiates the individuals in her sample according to the self-reported onset of health conditions. She documents that healthy individuals are more risk willing than sick individuals and that this gap seems to be constant over the life cycle of individuals. According to Schurer (2015, p.27) these results might not be interpreted as causal. Selection could be a potential source of bias. Those that suffer from a health shock might be a selective group, different from those without health shocks. Reverse causality might be an additional source of concern. Risk attitudes themselves might be determining the individual probability of health shocks.

Like Schurer (2015), in this paper we also use data from the SOEP. Nevertheless, there exist two differences between the previous studies and our analysis. Accordingly, we contribute to the literature as follows. First of all, we approach selection and reverse causality by a different method to any of the three previous papers. Specifically, we apply a regression-adjusted matching approach, control for a wide set of potentially confounding characteristics and exploit the panel structure of our data in order to justify unconfoundedness as our identifying assumption. Our second contribution to the literature is our measurement of health shocks. Several alternatives have been proposed in the literature so far, while the majority use self-reported data. In this paper we propose an innovative approach and use grip strength as an objective and reliable health shock measure. To the best of our knowledge we are the first to do so – not only in the literature on health and risk preferences but also on health shocks in general.

We find that health shocks significantly increase individual risk aversion. Those that experience a health shock are significantly more risk averse afterwards. The results are robust to various sensitivity analyses. This finding is relevant for our understanding of individual behaviour in uncertain economic decisions. Apparently, the risk attitudes of an individual are not a constant personality trait. We discuss further implications in the concluding section. The remainder of this paper is organised as follows. The empirical approach is laid out in the next section, while section 3 introduces the data. The results are discussed in section 4 and section 5 concludes.

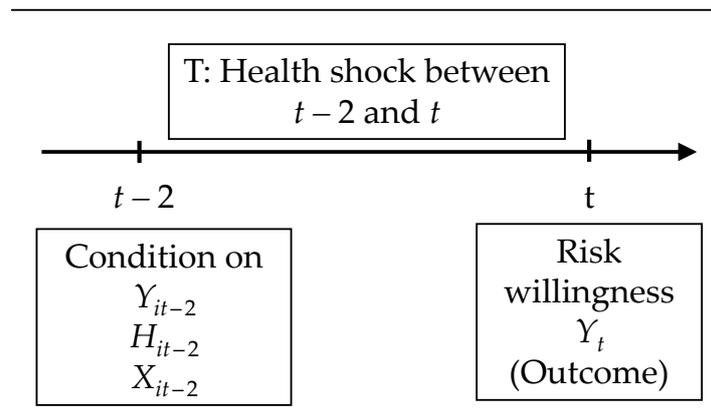
2 Empirical Approach

In laying out the empirical approach to estimate the effects of health shocks on risk aversion, we follow the notation of the treatment effects literature. Building on the potential outcome model (Rubin, 1974) we aim at estimating the effect of a treatment T (a health shock) on an observed outcome Y (individual risk willingness). Obviously, those in the treatment group might be a selective group, unlike the untreated with respect to several characteristics. If these characteristics are also correlated with Y a comparison of those with and without health shocks yields misleading results. In order to account for this, we need to control for all potentially confounding characteristics. In doing so, our empirical approach relies on the unconfoundedness assumption: Conditional on a specific set of covariates, treatment T is independent of potential outcomes. In other words, given controls, assignment to T is as good as random and the average outcome Y may be compared between the treatment and the control group.

Figure 1 shows the time structure and our approach to lend credibility to the unconfoundedness assumption. Treatment occurs between periods $t - 2$ and t (see section 3 for a detailed definition of the treatment in our data), while the outcome variable Y_{it} is measured in t . We are concerned with three parts of selection that might be a threat to the unconfoundedness assumption. A first one is that there is an effect of a baseline level of risk aversion on the

experience of a health shock. It could be, for instance, that more risk-willing individuals exhibit an unhealthier lifestyle, smoke more or are less cautious car drivers and are therefore more likely to suffer from health shocks. We approach this by matching on pre-treatment values of risk willingness Y_{it-2} . This also accounts for a lot of individual unobserved heterogeneity up to period $t - 2$ (e.g. including reporting styles to the risk-willingness question, see section 3).

Figure 1: Time Structure



Source: Own illustration.

A second issue is baseline health, which is probably correlated with both risk aversion and the probability of undergoing a health shock. Thus, we include pre-treatment values of individual health H_{it-2} as well. Third, we include many other covariates (X_{it-2}) that account for individual differences and are explained below. All of them are taken from the pre-treatment period $t - 2$ in order to mitigate the potential problem of covariates themselves being affected by the treatment status. All in all, there is potential selection into the treatment or, put differently, the experience of a health shock is not a random event as such. However, we account for this by exploiting the richness of the data in terms of control variables and the panel structure in particular, enabling us to include important pre-treatment variables.

In choosing the covariates X_{it-2} and their functional form we apply a data-driven approach suggested by Imbens (2014) in order to enhance the flexibility of the specification. We start with a set of basic covariates and then add further variables in first- and second-order terms

as determined in a stepwise procedure. Specifically, we control for age, gender, income, education, migration, family structure, occupation and characteristics of the place of residence.²

We apply regression-adjusted matching (Rubin, 1979, also used by, e.g., Marcus, 2014; Schmitz and Westphal, 2015). First, we perform propensity score matching (with a probit for the propensity score and an Epanechnikov kernel) and obtain weights to make the treatment and control group comparable.³ The weights are henceforth used to weight the observations in the following regression:

$$Y_i = \alpha + \beta \cdot T_i + X_i' \gamma + \epsilon_i. \quad (1)$$

The advantage of this joint approach of matching and regression is the *double robustness property* (Bang and Robins, 2005). If either the regression function or the propensity score function are correctly specified the resulting estimates are consistent. Assuming unconfoundedness the coefficient $\hat{\beta}$ is a consistent estimate of the average treatment effect on the treated (ATT). It is an estimate of the effect of health shocks on risk willingness for those suffering from a health shock.

We provide two kinds of robustness checks in order to assess the flexibility of the functional form in which pre-treatment risk willingness and individual health are connected to the outcome. First, we require an *exact match* of the treatment and control group according to the pre-treatment health status H_{it-2} . That means, unlike in our baseline specification, we stratify our sample according to health status in the pre-treatment period and analyse each stratum separately. An illustration of this is given in Figure 2 for the exemplary case if one had two strata of pre-treatment health status indexed by j .⁴ One stratum (or subsample)

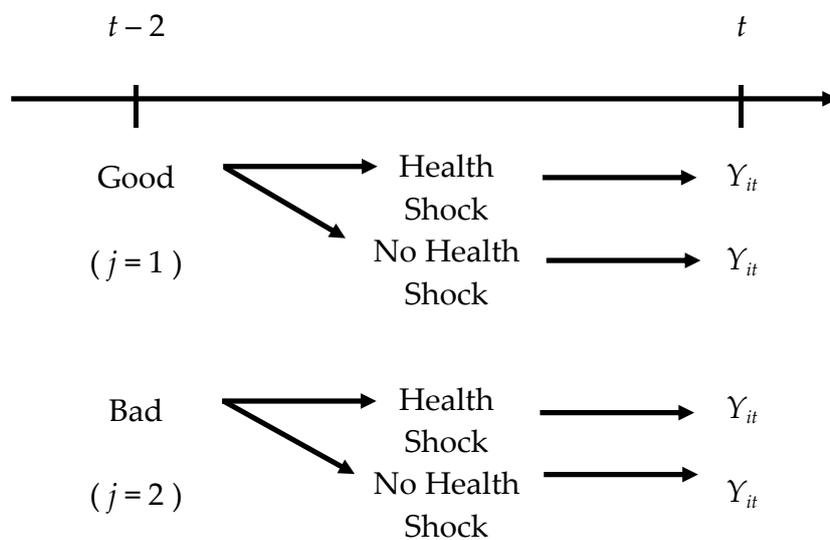
²Details of the procedure of covariate selection may be found in Appendix A.1. More information on these variables are given in section 3.3.

³We use the ado-file *psmatch2* (Leuven and Sianesi, 2003) in Stata 12.1 to compute the matching weights. According to the literature the specific kernel function seems to be of practical unimportance for the results while the choice of the bandwidth seems to be more crucial (Caliendo and Kopeinig, 2008). We therefore assess the sensitivity of all our results provided in this paper with respect to the bandwidth k . It makes practically no difference to the results if we try out 0.02, 0.03, 0.06 or 0.09 as alternatives. The effect sizes and signs remain unchanged, while in some cases significance levels vary slightly. The results with varying bandwidth are not shown but are available upon request.

⁴In order to group observations according to pre-treatment health status we use a self-assessed health measure based on a 5-point Likert scale. In some cases we collapse the outer categories of this variable to ensure

contains only those in good health in $t - 2$, while those in bad health are in the other stratum. The regression-adjusted matching as outlined above is now conducted separately for each stratum and the results are subsequently combined.⁵ Our second sensitivity analysis works analogously. Here, instead of conditioning on pre-treatment health, we require an exact match of the observations with respect to pre-treatment outcome Y_{it-2} .

Figure 2: Exact Matching on Pre-Treatment Health — Example with Two Strata ($j = 2$)



Source: Own illustration.

3 Data

The data come from waves 2006–2012 of the German Socio-Economic Panel (SOEP, version 29). It was retrieved using PanelWhiz, a user-written Stata Add-On (Hahn and Haisken-DeNew, 2013).⁶ The SOEP is a representative survey of German households that started

sufficient sample sizes. However, we always keep the mental and the physical SF-12 index as regressors when using equation (1) in each stratum.

⁵In each stratum j we use equation (1) and a combination of resulting estimates according to: $\hat{\beta} = \frac{1}{n} \sum_j n_j \hat{\beta}_j$, $\hat{se} = \sqrt{\frac{1}{n^2} \sum_j n_j^2 \hat{se}_j^2}$.

⁶PanelWhiz was written by Dr. John P. Haisken-DeNew. See Hahn and Haisken-DeNew (2013) and Haisken-DeNew and Hahn (2010) for details. The PanelWhiz generated DO file to retrieve the data used here is available upon request. Any data or computational errors in this paper are our own.

in 1984 and has been conducted yearly since. The total annual sample contains more than 20,000 individuals from nearly 11,000 households. The survey covers a broad range of topics by providing socio-economic and demographic characteristics as well as information on the labour market and the family status of the respondents. Most importantly with respect to our research question, the SOEP also provides also data on individual attitudes as well as the health status of the respondents.⁷

3.1 Health Shock Measurement with Grip Strength Data

In the literature several alternatives exist to measure the experience of a health shock. A large share of these are typically confined to self-reported information of some kind. A prominent approach is to work with changes in the self-assessed health status of individuals (Clark and Etilé, 2002; García-Gómez, 2011; Sundmacher, 2012). Several other studies also refer to the onset of severe health conditions such as cancer (Smith *et al.*, 2001; Clark and Etilé, 2002; Sahm, 2012). In some studies individuals are also asked directly whether they have experienced a health shock (Bünnings, 2013; Gloede *et al.*, 2013). However, individual reporting styles need to be considered when working with self-reported data. It could be, for instance, that variation in some health measure due to reporting styles is mistaken as a health shock. In this paper we follow an innovative approach and exploit an objective medical indicator that is measured on a clearly defined scale. Specifically, we use data on hand grip strength measured in kilograms. This gives us the advantage of being able to define and interpret differences between individuals or between measurements at different points in time.

Grip strength contains more information than just the muscle strength of the hands. Based on broad empirical evidence from the medical literature, grip strength is known as a valid indicator of the overall health status of an individual. Several medical studies document the association between a low level of grip strength and certain negative health outcomes, such as decreased overall muscular strength, the onset of chronic diseases, nutritional de-

⁷Cf. Wagner *et al.* (2007) for more details.

pletion, physical inactivity and mortality; cf. Rantanen *et al.* (2003), Bohannon (2008) or Ambrasat *et al.* (2011) and the references therein. The underlying mechanisms are not yet completely understood but it is suggested that ‘poor muscle strength could be a marker of disease severity, which in turn is associated with mortality’ (Rantanen *et al.*, 2003, p. 637).

Analogous evidence exists for extreme losses of grip strength over time (Rantanen *et al.*, 1998; Ling *et al.*, 2010; Xue Q *et al.*, 2011; Stenholm *et al.*, 2012). For example, Ling *et al.* (2010) compare 89-year-old Dutch males with different developments of grip strength over four years. They find that those with a decline in grip strength of 25% or more have a significantly higher mortality risk than those with a lower decrease or even an increase. It is our aim to incorporate this medical evidence when defining our health shock measure. A general cut-off point that identifies those with extreme losses in grip strength would be ideal but despite intense literature research we are unaware of such information. We therefore take the aforementioned value of a loss of 25% or more from Ling *et al.* (2010) as a starting-point for our analysis.

Grip strength in the SOEP is measured by the regular interviewers as it is also common in other well-known surveys, such as the Survey of Health, Ageing and Retirement in Europe (SHARE) or the US Health and Retirement Study (HRS). The SOEP interviewers are equipped with so-called dynamometers, receive instruction on the usage of these small, non-invasive devices and are then able to assess the grip strength of the survey respondents. Due to a limited number of dynamometers this is only done within a subsample of the total SOEP survey population. Every other year from 2006 onwards grip strength is measured within a subsample of roughly 5,000 individuals. This subsample is similar in terms of important characteristics such as age, gender and region to the total annual sample of the SOEP, which is representative of the current residential population of Germany.⁸ In our analysis, we lose one wave of observations as our health shock measure is based on changes in grip strength over time. Further observations are also lost as not all respondents of the grip strength sub-

⁸Schupp (2007) documents this for the 2006 subsample, Ambrasat and Schupp (2011) do so for the 2008 subsample.

sample can be contacted repeatedly in subsequent waves.⁹ Our resulting sample consists of 10,034 person-year observations equally balanced between the years 2008, 2010 and 2012.

Grip strength measurement takes place as part of the regular SOEP interview and in individuals homes rather than, for example, in a laboratory, which might have potentially shielded away survey respondents from participating. Schupp (2007) analyses the first wave of grip strength data of the SOEP and reports that many respondents even enjoyed participating in the measurements as a nice break from the survey questionnaires. Almost everybody of the targeted respondents participated (96%).¹⁰ The actual measurement procedure is as follows: The interviewer illustrates the use of the dynamometer first and then asks the respondents to press it twice with each hand as hard as they can, starting with the right hand and alternating afterwards. Test trials are not allowed. There exist several alternatives in the medical literature on how to summarize this information (Roberts *et al.*, 2011). Ambrasat and Schupp (2011) and Ambrasat *et al.* (2011) have analysed the case of the SOEP rigorously. They suggest using the maximal value from all available measurements as a measure for the grip strength of an individual as, due to the absence of test trials, some individuals might not exert their full grip strength in the first measurements. We follow this suggestion. Furthermore, as Ambrasat *et al.* (2011) also document a considerable degree of individual heterogeneity in grip strength, we define our health shock measure based on *relative* changes in *individual* grip strength (GS) over time.¹¹ Specifically we calculate $\Theta_t = (GS_t - GS_{t-2}) / GS_{t-2}$ for $t = 2008, 2010, 2012$.

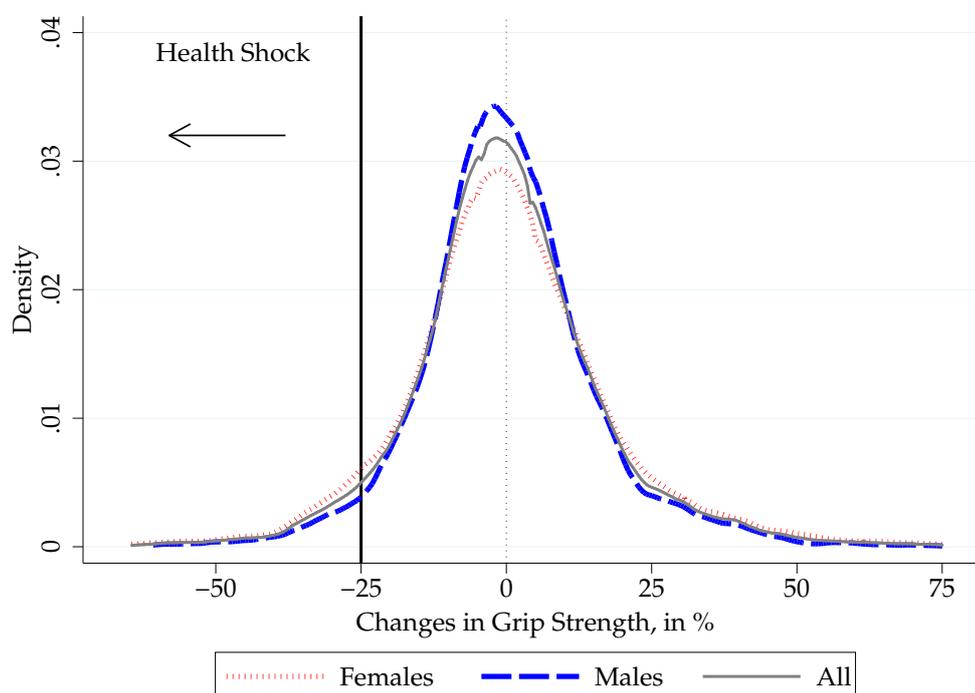
The distribution of the resulting variable is depicted as the grey thick line in Figure 3. There exists considerable heterogeneity in changes of individual grip strength over time. At the lower end of the distribution there are individuals that experience a loss of more than 60% in their individual hand grip strength over the course of two years. At the upper end there are also individuals whose grip strength increases by more than 50%. While such extreme

⁹This happens mainly because the dynamometers are distributed amongst interviewers and not every interviewer might successfully visit the exact same respondents each year.

¹⁰Reasons for non-participation are mainly health related (Schupp, 2007). Accordingly, we expect our results to be lower bound for the effect of a health shock.

¹¹We can verify this, cf. Figure A1 in the Appendix.

Figure 3: Relative Changes in Grip Strength across Genders



Source: SOEP, v29, 2008-2012 (n=10,034).

Relative changes in grip strength (GS): $\Theta_t = (GS_t - GS_{t-2}) / GS_{t-2}$ for $t = 2008, 2010, 2012$.

changes are quite rare, 80% of our sample experience grip strength changes of between a -20% decrease and +20% increase over time.

We define our health shock measure as a loss of 25% or more in individual grip strength over two years. This definition is illustrated by the black vertical line in Figure 3. Those to the left of this vertical line experience a loss of their individual grip strength of 25% or more and are accordingly defined as those that experienced a health shock during the last two years. Conversely, those to the right are defined as not having experienced a health shock. Applying the definition results in the following health shock variable: 514 (5.1%) of the individuals experience a health shock while 9,520 (94.9%) do not, cf. line 3 in Table 1.

Figure 3, also provides a differentiation of grip strength by gender. Extreme changes in grip strength seem to be slightly more common for women than for men. Accordingly, 330 females (6.3%) in our sample experienced a health shock (a loss of 25% in grip strength), while the same is true for only 184 males (3.9%). Figure 4 explores age heterogeneity in grip strength. Those in their eighties or older experience extreme decreases in grip strength more

often than younger individuals. Conversely, the youngest in our data experience extreme decreases least often. However, across all age groups and both genders, extreme decreases and increases appear less often than moderate changes.

Table 1: Sample Sizes According to Health Shock Measure

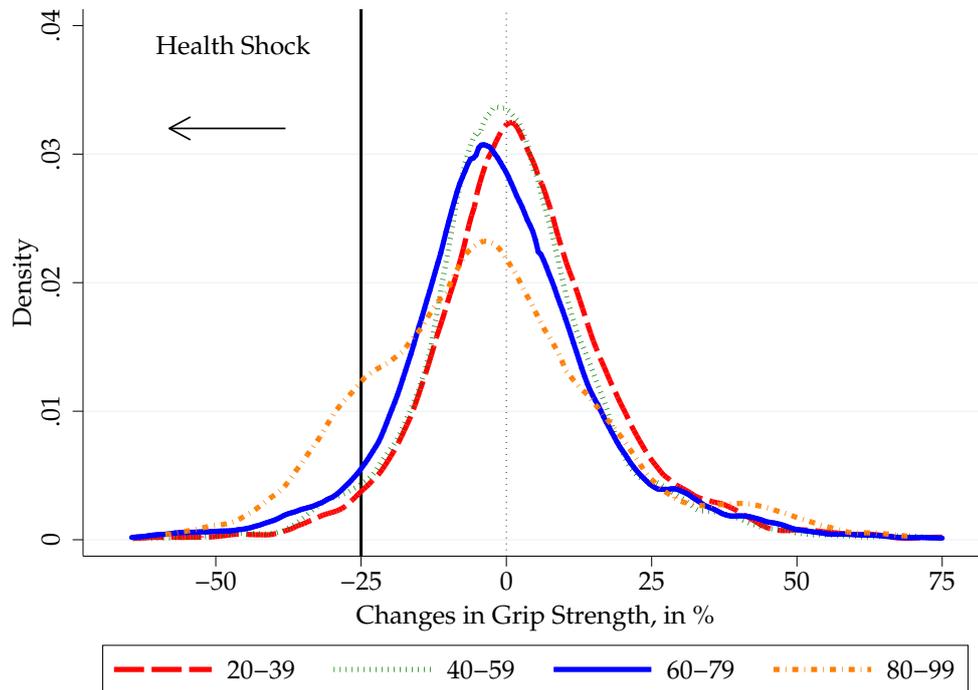
	Health Shock	No Health Shock	Total	Years
(1) Grip Strength Loss, $\geq 30\%$	315 (3.1%)	9,719 (96.9%)	10,034 (100%)	2008, 2010, 2012
(2) Grip Strength Loss, $\geq 27.5\%$	411 (4.1%)	9,623 (95.9%)		
(3) Grip Strength Loss, $\geq 25\%$	514 (5.1%)	9,520 (94.9%)		
(4) Grip Strength Loss, $\geq 22.5\%$	646 (6.4%)	9,388 (93.6%)		
(5) Grip Strength Loss, $\geq 20\%$	841 (8.4%)	9,193 (91.6%)		
(6) Onset Condition ^a	2,494 (19.4%)	10,392 (80.6%)	12,886 (100%)	2011
(7) Drop in SAH ^b	4,422 (23.9%)	14,087 (76.1%)	18,509 (100%)	2006, 2008, 2010, 2011, 2012

Source: *Source*: SOEP, v29, 2006-2012. ^a Onset of condition between 2009 and 2011 (one or more of the following): stroke, cardiovascular diseases, high blood pressure, cancer, diabetes, asthma, migraine, dementia and depression. Respondents are asked if a physician has ever diagnosed one of the aforementioned conditions. ^b Self-assessed health. Exactly as in García-Gómez (2011). Scale: 1=excellent, 2=good, 3=fair, 4=bad, 5=very bad. Health shock: $t : 1, 2 \rightarrow t + 1 : 3, 4, 5 \rightarrow t + 2 : 3, 4, 5$. No health shock: $t = t + 1 = t + 2 = 1$ or $t = t + 1 = t + 2 = 2$.

We vary the cut-off point in our health shock definition in order to assess the sensitivity of our results in this respect. Graphically, that means we move the black vertical lines in figures 3 and 4 to the left or to the right. Specifically, we use a loss of 30% (27.5%, 22.5%, 20%) as additional cut-off points (cf. Table 1 for the respective sample sizes). Furthermore, we include two alternative health shock indicators in our analysis that rely on established definitions from the literature. These additional measures are not restricted to the grip strength subsample of the SOEP. Accordingly, the sample sizes are larger for those measures, cf. Table 1. Specifically, we use the onset of a health condition over time, such as a stroke, diabetes or cancer. The questionnaire asks respondents to state whether a physician has ever diagnosed a specific health condition. We use a change over time in this variable to define a health shock.¹² Almost 20% of the respective sample experienced such a health shock. As a further indicator we use a drop in self-assessed health over time. We apply exactly the same definition as in García-Gómez (2011): If *excellent* or *good* health in an initial period are followed

¹²The specific conditions contained in this health shock indicator are stroke, cardiovascular diseases, high blood pressure, cancer, diabetes, asthma, migraine, dementia and depression.

Figure 4: Relative Changes in Grip Strength across Age Groups



Source: SOEP, v29, 2008-2012 (n=10,034).

Relative changes in grip strength (GS): $\Theta_t = (GS_t - GS_{t-2}) / GS_{t-2}$ for $t = 2008, 2010, 2012$.

by a permanent drop to a *satisfactory* or worse health status in the two subsequent periods, then this individual is defined as having experienced a health shock.¹³

3.2 Measuring Risk Willingness

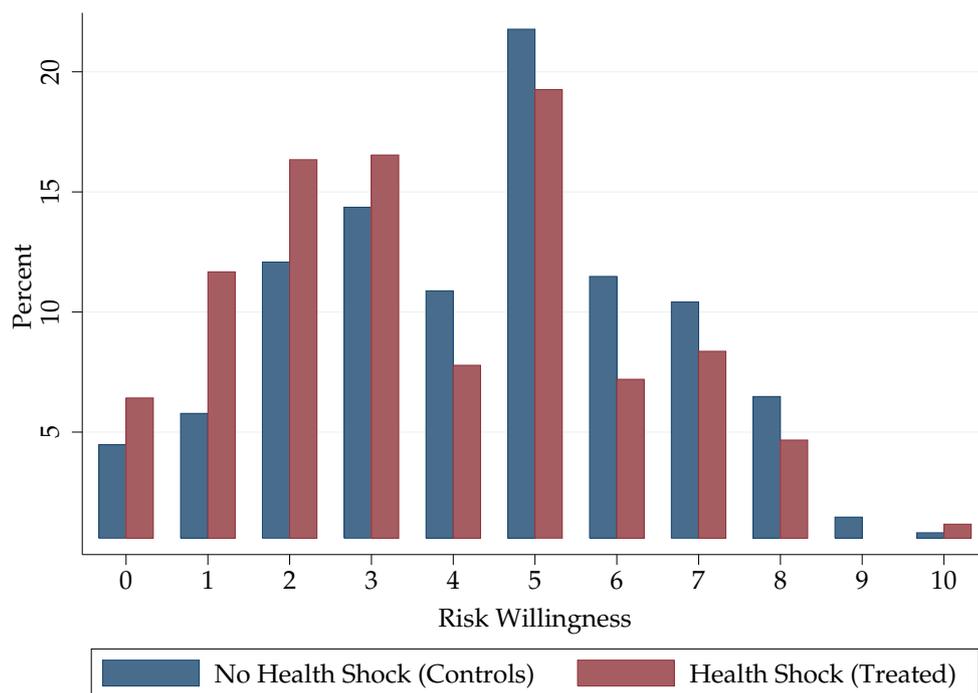
In the SOEP, individuals evaluate their own risk willingness on an 11-point Likert scale ranging from 0 (not at all willing to take risks) to 10 (very willing to take risks).¹⁴ We use this as the outcome variable of our study. Figure 5 displays how it is distributed in our sample, differentiated by treatment status. For both the treated and control group it holds that the

¹³Cf. Table 1 for detailed definition.

¹⁴A translation of the original German question reads: How do you see yourself: Are you generally a person who is very willing to take risks or do you try to avoid taking risks? The original German questionnaire may be found here: <http://panel.gsoep.de/> In addition to this general question there exists a health specific measure of risk willingness in the SOEP. However, we focus on the general risk willingness measure here, as the health-specific information is only collected twice (2004, 2009). This would leave us with too few observations for an analysis. Moreover, Dohmen *et al.* (2011) report that the general and the health-specific, risk-willingness measure are well correlated (pairwise correlation coefficient = 0.4768). In our sample we find a similar correlation (0.4619) of these two measures. Dohmen *et al.* (2011) furthermore suggest, that the 'question about risk taking in general generates the best all-round predictor of risky behaviour', p. 522.

largest fraction of individuals rate their risk-willingness with 5, while there clearly exists heterogeneity across the whole range. The second and third most prominent outcomes are 3 and 2, respectively. The overall mean of our risk-willingness measure is 4.37 with a standard deviation of 2.25.¹⁵ The overall median is 5 in the sample. Comparing the respective risk willingness categories by treatment status the following pattern becomes apparent: More individuals from the treatment group than from the control group belong to the classes of relatively low risk willingness (0 to 3), while the opposite holds for the categories of a higher degree of risk willingness (4 to 9), except for those with the highest degree of risk willingness (10). This descriptive evidence suggests that there could be a negative effect of health shocks on the risk willingness of individuals. The following analysis needs to ascertain whether this is really an effect or whether this is simply driven by other covariates.

Figure 5: The Distribution of Risk Willingness by Treatment Status



Source: SOEP, v29, 2008-2012.

Distribution based on grip strength subsample, i.e. only those individuals that participate in grip strength measurement (n=10,034). Overall sample mean: 4.37 (2.25 sd). Original question: Are you generally a person who is very willing to take risks or do you try to avoid taking risks? (0 = not at all willing, 10 = very willing). The distribution of the risk willingness measure looks similar for the two other samples. Cf. Table 1 for an overview.

¹⁵ Assuming cardinality across the different categories.

One may question the reliability of our outcome variable. One major concern is incentive-compatibility. Would individuals behave in accordance to their stated preferences when they face real economic decisions with monetary incentives at stake? In an extensive study Dohmen *et al.* (2011) document the reliability of the SOEP survey item with the use of an incentivised laboratory experiment. In addition, they explore the determinants of actual risky behaviour of the SOEP survey respondents, such as smoking, stock holding and self-employment and find evidence of the explanatory power of the risk willingness survey item in this respect. Furthermore, the same measure of risk-willingness is used by Dohmen *et al.* (2010) who study the relationship between cognitive ability and risk preferences, and by Dohmen *et al.* (2012) who analyse the intergenerational transmission of risk and trust preferences.

The aforementioned empirical approaches in section 2 assume cardinality across the distinct categories of risk willingness as our outcome measure. In section 4.2 we therefore assess this assumption and document the robustness of our finding in this respect.

3.3 Further Covariates

We exploit a broad set of covariates. Specifically, we include age (linear and quadratic term), gender, income and education. Income is deflated, logarithmised and equivalised according to the OECD scale, while education is measured in years. Furthermore, we control for migration and the family structure (marital status, presence of children). With respect to occupation we distinguish between retired, unemployed, self-employed, blue- and white-collar workers, and civil servants from those in education and others. We take the degree of urbanisation of the place of residence into account and differentiate between East and West Germany. We also enclose measures of individual health status by a mental and a physical health index, based on the well-known SF-12 questionnaire.¹⁶ Summary statistics on these covariates are given in the next section.

¹⁶For details of these health indices cf. Andersen *et al.* (2007).

4 Results

We start with an illustration of how matching makes the treatment and control group comparable. The different columns in Table 2 report descriptive statistics of the subgroups by treatment status, based on the grip strength sample ($n = 10,034$). As a standardised measure of the difference between the subgroups we calculate the Standardised Bias (SB). The literature suggests that the SB for each variable should be less than 5% after matching (Caliendo and Kopeinig, 2008). This high degree of similarity between the treatment and control group is ensured for the results provided in this section.

Table 2: Descriptive Statistics by Treatment Status

	Treated		Unmatched Controls		Matched Controls		SB ^a , in %	
	mean (1)	sd (2)	mean (3)	sd (4)	mean (5)	sd (6)	Unmatched (7)	Matched (8)
Risk will. _{<i>t-2</i>}	4.04	2.28	4.48	2.26	4.09	2.30	-19.16	-1.87
Phys. health _{<i>t-2</i>}	45.97	10.58	49.51	9.71	46.18	10.60	-36.28	-1.94
Mental health _{<i>t-2</i>}	51.07	10.33	51.72	9.38	51.43	9.75	-6.91	-3.58
Age _{<i>t-2</i>}	58.63	16.64	51.68	16.04	58.13	16.86	43.23	2.96
Age sq. _{<i>t-2</i>}	3714.03	1918.26	2928.38	1677.30	3663.84	1913.38	46.47	2.62
Female _{<i>t-2</i>}	0.64	0.48	0.52	0.50	0.63	0.48	25.06	1.76
Children _{<i>t-2</i>}	0.20	0.40	0.28	0.45	0.21	0.41	-18.55	-2.88
Married _{<i>t-2</i>}	0.66	0.47	0.65	0.48	0.66	0.47	2.17	0.28
Single _{<i>t-2</i>}	0.09	0.29	0.18	0.38	0.10	0.30	-23.53	-3.31
Migrated _{<i>t-2</i>}	0.16	0.36	0.14	0.35	0.16	0.36	4.84	0.12
Income _{<i>t-2</i>}	0.51	0.36	0.55	0.35	0.52	0.35	-12.42	-2.95
Education _{<i>t-2</i>}	11.79	2.59	12.19	2.69	11.80	2.62	-14.89	-0.51
Urban _{<i>t-2</i>}	0.48	0.50	0.46	0.50	0.46	0.50	2.87	3.84
Rural _{<i>t-2</i>}	0.25	0.44	0.25	0.44	0.25	0.43	-0.29	0.24
West-Germany _{<i>t-2</i>}	0.72	0.45	0.72	0.45	0.71	0.45	-0.25	1.36
Retired _{<i>t-2</i>}	0.45	0.50	0.29	0.45	0.45	0.50	33.51	-0.53
In Education _{<i>t-2</i>}	0.02	0.14	0.04	0.19	0.02	0.14	-9.44	-1.14
Unemployed _{<i>t-2</i>}	0.05	0.22	0.06	0.23	0.05	0.21	-3.18	1.46
Self-Empl. _{<i>t-2</i>}	0.05	0.21	0.07	0.25	0.05	0.22	-8.33	-1.34
Blue-collar _{<i>t-2</i>}	0.11	0.32	0.15	0.36	0.12	0.32	-10.61	-0.35
White-Collar _{<i>t-2</i>}	0.21	0.40	0.29	0.45	0.22	0.42	-17.81	-3.66
Civil _{<i>t-2</i>}	0.03	0.17	0.04	0.19	0.03	0.16	-4.85	1.40
Year 2010	0.38	0.49	0.35	0.48	0.38	0.49	6.61	0.94
Year 2012	0.31	0.46	0.30	0.46	0.31	0.46	2.39	0.05
Observations	514		9,520		514 ^b		1,028 ^b	

Source: SOEP, v29, 2006-2012. ^a Standardized Bias: $SB(X_j) = 100 \cdot (\bar{X}_{j,T=1} - \bar{X}_{j,T=0}) / \sqrt{0.5 \cdot (\sigma_{j,T=1}^2 + \sigma_{j,T=0}^2)}$ for each covariate X_j . $T = 0, 1$ distinguishes control and treatment group, respectively. ^b This is the weighted number of observations (matching weights applied). The raw numbers of observations are 9,520 (matched controls) and 10,034 (whole sample), respectively. Risk will._{*t-2*} : Indicates risk willingness of individual in pre-treatment period. Cf. section 3 for summary statistics on risk willingness as the outcome variable. Children_{*t-2*} : Indicator of children living in household. Self-Empl._{*t-2*} : Indicator of self-employment. Year 2010/2012: Indicators for calendar years.

The first two columns of Table 2 contain the summary statistics of the treated subgroup (those that suffer from a health shock). They are on average 58.6 years old, 64% of them are females and 9% are single. The third and fourth column give an overview of the unmatched controls. Striking differences between the treated and unweighted control group are apparent, which is also mirrored by the SB being well above the suggested value of 5% in many cases (column 7). After applying the matching weights the treatment and control group are quite similar. The SB for these two groups is less than 5% for each covariate in the sample (column 8).

4.1 Main Specification

Table 3 reports the regression results based on our main specification. The estimated effect of health shocks on risk willingness is -0.254 and statistically significant, see column A. The mean value of our risk-willingness measure is 4.37 with a standard deviation of 2.25. Thus, health shocks lead to a decrease in risk willingness of about 11% of a standard deviation. This direct and negative effect of health shocks on the risk willingness of individuals is the main finding of our paper. In the following we check the sensitivity of this finding in various ways. While there seems to be some variation in effect size, the main finding remains robust.

Table 3: The Effect of Health Shocks on Individual Risk Willingness

Outcome: Risk Willingness	(A) Baseline	(B) Exact Match Pre-Treatment Health	(C) Exact Match Pre-Treatment Risk Willingness
Health Shock (Loss of Grip Strength, $\geq 25\%$)	-0.254^{***} (0.095)	-0.273^{***} (0.096)	-0.192^{**} (0.085)
Observations	10,034	10,032 ^a	10,006 ^a

Source: SOEP, v29, 2006-2012. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Columns refer to different matching procedures. (A): No exact matching on any covariate. All covariates enter analysis in the standard way. (B): Exact matching of individuals on pre-treatment health status required. (C): Exact matching of individuals on pre-treatment outcome (risk willingness) required. Kernel bandwidth: $k = 0.02$. Standardised Bias (SB) $< 5\%$ for all matching covariates. Standard errors in parentheses, clustered on household level. ^a Number of observations is slightly smaller than with matching procedure (A) due to restrictions of common support.

As a first robustness check we partition our sample and compare only those individuals that have the same value of pre-treatment health. The results prove to be robust in this respect.

The estimated effect is negative, slightly larger in absolute size (−0.273) and statistically significant (column B, Table 3). Similarly, specification C ensures that individuals are equal in their pre-treatment risk willingness. The respective estimated effect is again negative and statistically significant. Though it is a smaller decrease than previously estimated (−0.192), it still corresponds to a drop of 9% of a standard deviation in risk willingness.

Table 4: Robustness of the Health Shock Effect

Outcome: Risk Willingness	(A) Baseline	(B) Exact, Health	(C) Exact, Outcome
(1) Grip Strength Loss, $\geq 30\%$	−0.289** (0.124)	−0.298** (0.121)	−0.187* (0.107)
(2) Grip Strength Loss, $\geq 27.5\%$	−0.287*** (0.108)	−0.249** (0.105)	−0.238** (0.094)
(3) Grip Strength Loss, $\geq 25\%$	−0.254*** (0.095)	−0.273*** (0.096)	−0.192** (0.085)
(4) Grip Strength Loss, $\geq 22.5\%$	−0.220** (0.088)	−0.213** (0.087)	−0.162* (0.079)
(5) Grip Strength Loss, $\geq 20\%$	−0.147* (0.076)	−0.131* (0.076)	−0.080 (0.072)
(6) Onset Condition	−0.094** (0.045)	−0.083* (0.046)	−0.110** (0.044)
(7) Drop in SAH ^a	−0.162*** (0.039)	−0.164*** (0.039)	−0.162*** (0.038)

Source: SOEP, v29, 2006-2012. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Seven different health shock measures (rows) are used in three different matching procedures (columns). (A): No exact matching on any covariate. All covariates enter analysis in the standard way. (B): Exact matching of individuals on pre-treatment health status required. (C): Exact matching of individuals on pre-treatment outcome (risk willingness) required. Kernel bandwidths between matching procedures may vary: The broadest bandwidth is chosen that ensures a Standardised Bias (SB) $< 5\%$ for all matching covariates; there is exactly one exception to this rule (cell B2). Standard errors in parentheses, clustered on household level. ^a SAH = self-assessed health.

We furthermore assess the sensitivity of our results with respect to the cut-off point in our health shock definition. So far, those with a loss of grip strength of 25% or more are defined as having a health shock. We now gradually change this cut-off point in both directions and alternatively define as treated those that lost 30% (27.5%, 22.5%, 20%) or more of their grip strength. The results are reported in the upper five lines of Table 4. Each line refers to a different definition of our health shock measure. Defining those that lost 27.5% or more of their grip strength as treated changes the effect sizes slightly (not significantly with respect

to the 25% measure, however, line 2). This is also found for those who lost 30% or more of their grip strength (line 1). Moving the cut-off point to the other direction also results in a negative effect, though that is sometimes no longer statistically significant (lines 4 and 5).

Table 4 provides further evidence of the robustness of our main finding. We try out two established health shock measures from the literature in our estimations. Line 6 contains the estimation results if the onset of a severe health condition like cancer is used as a health shock indicator. As before, a negative and significant effect is found across all specifications. When we use a drop in self-assessed health the same is found. Furthermore, our results are robust to different sized kernel bandwidths in the matching procedure.¹⁷

4.2 Distributional Regressions

So far we have assumed cardinality across the different categories of our outcome measure and have implicitly treated the differences between two neighbouring categories of risk willingness as comparable across all possible outcomes. That means we have treated the difference between 2 and 3 as the same as the one between 8 and 9, for example. This might be seen as an overly strong assumption given the ordinal nature of this variable. In the following we document the robustness of our finding in this respect. Specifically, we employ distributional regressions to develop an understanding of the effects of health shocks on the entire distribution of risk willingness.¹⁸

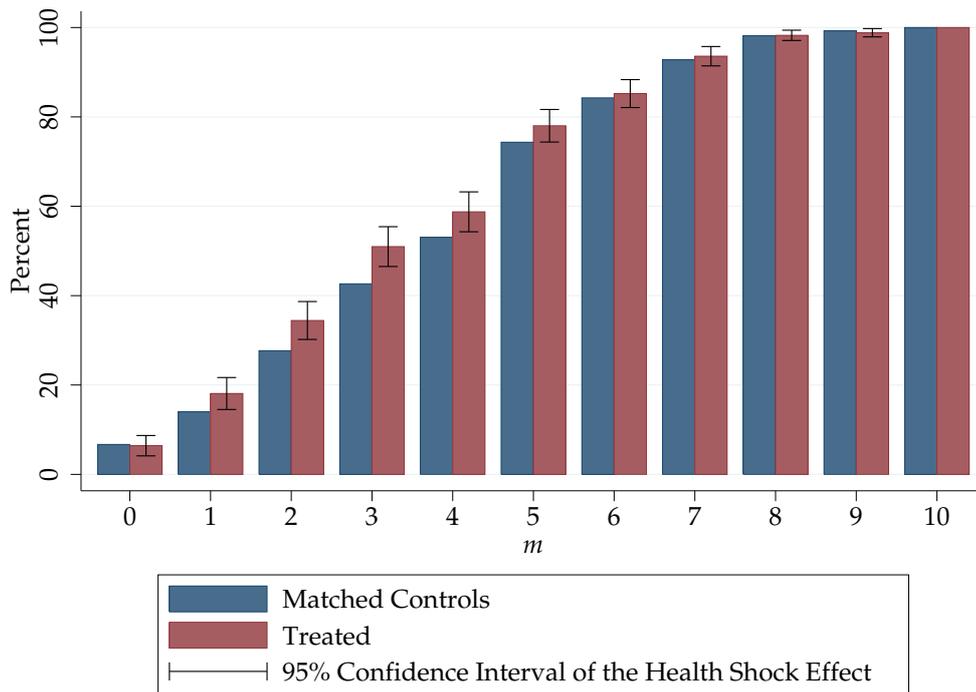
Distributional regression goes back to Foresi and Peracchi (1995) and was also used by de Meijer *et al.* (2013). Kolodziej and García-Gómez (2015) employ it also on an ordered outcome variable as we do here. It is comparable to quantile regression but has the advantage that the conditional distribution does not need to be smooth. Our outcome has 11 different categories increasing in the degree of risk willingness. We transform it into 10 dummy variables indicating whether risk willingness is below or equal to a specific value

¹⁷See footnote 3 in section 2 for further details.

¹⁸A natural alternative to deal with the ordered nature of the outcome variable is to apply an ordered probit or logit or an interval regression. Given that one needs stronger assumptions for these approaches, we do not outline this strategy here but report results in the Appendix for completeness. The results are in line with what we find with the distributional regressions.

m with $m = 0, \dots, 9$. We then use each of these dummies as an outcome in the regression-adjusted matching model as before. That is, we use the same 25% health shock indicator, the same control variables and the same matching weights as in our basic specification (column A, Table 3). Accordingly, as the transformed outcome variables are now binary, we employ 10 different linear probability models.

Figure 6: Health Shock Effects on $P(\text{Risk Willingness} \leq m)$



Source: SOEP, v29, 2008-2012.

Figure is based on estimated statistics from 10 linear probability models, scaled up by a factor of 100 in order to facilitate interpretation. Each outcome is a dummy variable indicating whether the risk willingness measure is below or equal to a specific value m with $m = 0, \dots, 9$ (increasing in risk willingness). The regressors in each model are the same health shock indicator (loss of grip strength, 25% or more) and the same control variables as in the basic specification of the regression-adjusted matching approach (column A, Table 3). The same matching weights are also applied.

From each of the 10 linear probability models we estimate three different statistics: The conditional probability for the treatment group that risk willingness will fall within or below some specific category, the respective probability for the group of matched controls and the health shock effect of that specific category. Detailed regression results are reported in Table A1 in the Appendix. We graphically summarise all the results in Figure 6, which accordingly depicts the cumulative distribution of risk willingness differentiated by treatment status.

The left blue bars at each of the m categories depict the conditional probabilities for the sample of matched controls, i.e. the sample of untreated observations after the application of matching weights. For instance, the bar at value 3 indicates that the probability to report a risk willingness of that value or lower is 43% for the group of matched controls. Likewise, the red bars on the right belong to the treated observations. The difference between the two bars depicts the effect of a health shock. Accordingly, the probability of 3 or lower is significantly increased by 8 percentage points to 51%.¹⁹

Looking at the entire distribution, Figure 6 documents that more probability mass is allocated to lower risk-willingness categories as an effect of a health shock. Specifically, for categories 1 to 3 (all below the sample median of 5) a significant increase is detected.²⁰ Accordingly, the probability of reporting these low values of risk willingness is increased significantly by a health shock. To the contrary, no such effects are found for the upper categories of the distribution. Thus, we conclude that the results detected with our main specification in section 4.1 seem to be driven by a shift of the risk-willingness distribution toward the very low categories of 1 to 3.

5 Conclusion

From the previous literature it is not clear whether health shocks affect the risk attitudes of individuals. We apply regression-adjusted matching as a different approach to previous papers concerned with this question. Based on our sample of German adults we find that health shocks lead to a decrease of about 9 to 11% of a standard deviation in the risk willingness of individuals. The finding is robust to a series of sensitivity analyses. Given our identifying assumption, our result might be interpreted as evidence of a causal effect

¹⁹Cf. Table A1 for the exact values.

²⁰The effects for the categories 4 and 5 are also positive and significant, cf. Table A1 for exact values. However, the respective treatment effects are smaller in absolute size to those of category 3. As the effects on the cumulative distribution are analysed here, the increases in category 3 have to be subtracted from the increases in categories 4 and 5 in order to isolate the respective effects. Doing this reveals that the actual effects are negative for these categories, considered individually.

that could also be driving the association between bad health and lower risk willingness as reported by Schurer (2015).

Our paper illustrates the instability of individual preferences over time and contributes to an emerging literature on this topic. Apparently, risk attitudes as a part of individual preferences are not a constant personality trait, fixed over the life cycle. Important events such as health shocks are shown to influence the degree of risk willingness of an individual. Moreover, given the importance of risk willingness as a central variable in individual decision making, it is possible that health shocks via risk attitudes also exert indirect effects on certain important economic outcomes, such as, for instance, education or labour market success. Such mechanisms could give rise to vicious circles, in which a health shock marks the starting point for a series of negative consequences in several domains of the life of an individual.

Similarly, as Finkelstein *et al.* (2013) also outline when analysing health dependent utility, we argue that our results also relate to the topics of insurance or individual optimisation across the life cycle. For instance, the individual decision for a specific insurance plan is determined by risk attitudes amongst other factors and, as such, might be influenced by the health effects documented in this paper. The same might be true for the optimal reimbursement by a health insurance or the individual decision to save for retirement across the life cycle. Furthermore, there exists empirical evidence that health shocks affect the probability that individuals smoke (Smith *et al.*, 2001; Sundmacher, 2012; Bünnings, 2013). One possible mechanism for such a behavioural change could be a shift in individual risk aversion as a result of a health shock. However, it should be kept in mind that we only identify short-term effects and it might well be that in the long run, risk willingness reverts back to the baseline level. This should be analysed in future work.

Our finding also has implications for applied empirical work. Risk attitudes are often assumed to be constant over time, which implies that they can be taken into account by fixed-effect or first-difference methods. However, according to the finding of this paper such attempts should be done carefully. This seems to be especially important when (a change

in) the health status of an individual is also part of an analysis. Similarly, we argue that risk attitudes might be potentially *bad control* variables which are themselves affected by health and other influences.

We also see implications of our finding for evaluation methods that are used to inform policy decisions such as, for instance, cost-benefit analyses. Risk attitudes play an important role here as the respective costs and benefits are typically not known with certainty. According to our finding we argue that such evaluations should differentiate between individuals, depending on the experience of a health shock. For instance, as those that suffered from a health shock exhibit higher risk aversion such individuals might c.p. benefit more from a reduction in uncertainty as an outcome of a policy. We suspect this to be of particular importance for health-related decisions, for instance, in a cost-benefit analysis concerned with the introduction of a screening device or a therapy.

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A Appendix

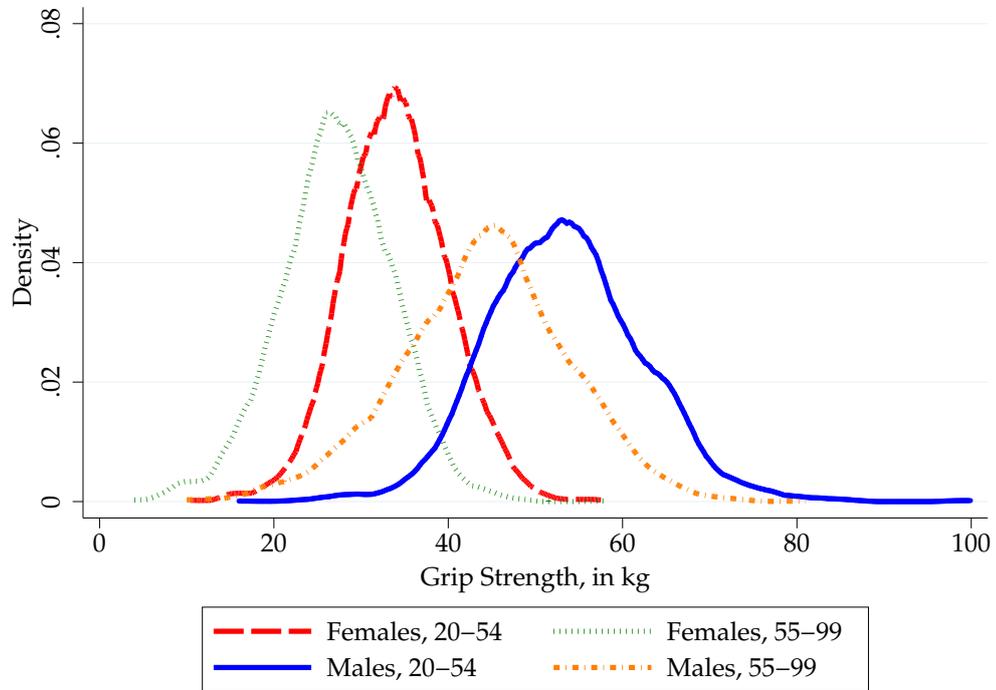
A.1 Estimation of the Propensity Score

We apply a data-driven approach suggested by Imbens (2014) in order to choose covariates to estimate the propensity score (the probability of a health shock).²¹ We start with a *basic set* of covariates that we include upfront because of their suggested relevance. This set consists of pre-treatment risk willingness, gender, age (linear and quadratic term), physical health, dietary concerns, smoking, the frequency of exercise and indicators of calendar years. We then add the remaining covariates of our sample sequentially (mental health, marital status, presence of children, income, education, migration, occupation, degree of urbanisation of place of residence, East/West Germany) and decide if we should include them based on likelihood ratio tests. We first include first-order terms until every covariate has been tried out. With the resulting set of covariates we then try out every possible combination of second-order terms (i.e. quadratic terms, interactions). As a threshold value for the likelihood ratio tests for linear (second order) terms we use $C_{lin} = 1$ ($C_{qua} = 2.71$). We apply the procedure separately for every health shock measure. Using a loss of grip strength of 25% or more as our health shock indicator we include the following covariates to estimate the propensity score, in addition to our basic set: $Single_{t-2}$, $Migrated_{t-2}$, $Migrated_{t-2} \times Diet_{t-2}$, $Migrated_{t-2} \times Female_{t-2}$. Accordingly, it is suggested that all other variables that are tried as previously described are left out of the procedure. For the other health shock measures this information is available upon request.

²¹We are grateful to Matthias Westphal for providing an ado-file with this procedure.

A.2 Additional Figures and Tables

Figure A1: Distribution of Maximum Grip Strength across the Sample



Source: SOEP, v29, 2008-2012 (n=10,034).

Mean values: 34.1 (Females, 20-54), 27.2 (Females, 55-99), 53.4 (Males, 20-54), 44.1 (Males, 55-99)

Table A1: Detailed Results Distributional Regressions

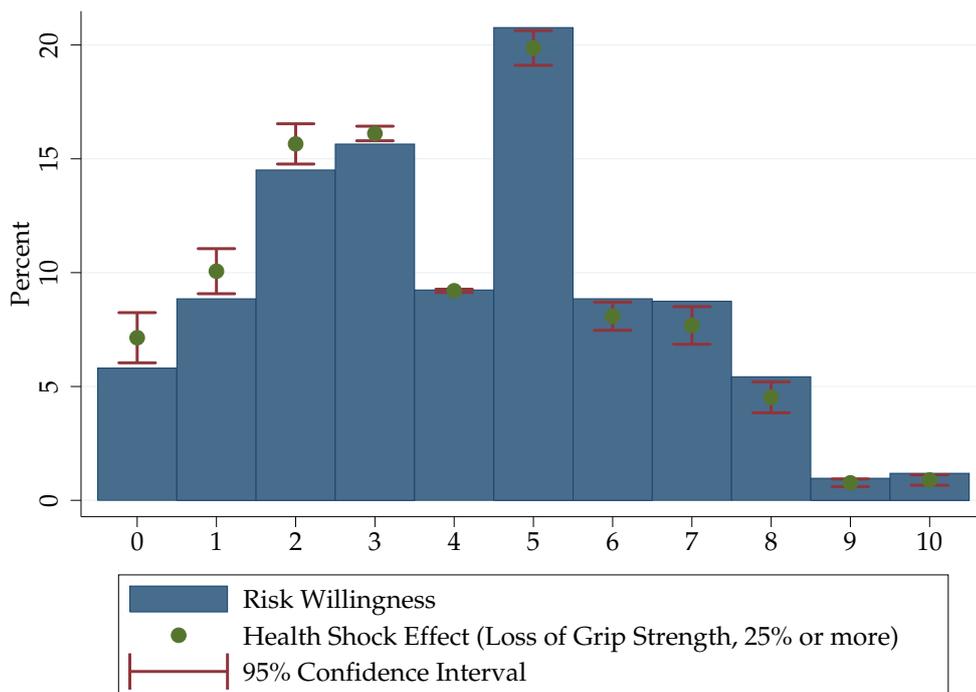
m	P(Risk Willingness $\leq m$), in %		Effect of a Health Shock (Grip Strength Loss, $\geq 25\%$), in percentage points
	Matched Controls (2)	Treated (3)	(4)
0	6.70	6.42	-0.43 (1.08)
1	14.01	18.09	3.62** (1.65)
2	27.68	34.44	6.14*** (1.99)
3	42.64	50.97	7.52*** (2.08)
4	53.06	58.75	4.92** (2.08)
5	74.33	78.02	3.06* (1.76)
6	84.26	85.21	0.46 (1.55)
7	92.79	93.58	0.58 (1.10)
8	98.16	98.25	0.00 (0.60)
9	99.27	98.83	-0.50 (0.48)

Source: SOEP, v29, 2006-2012. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors in parentheses, clustered on household level. Each line represents results from a different linear probability model, scaled up by a factor of 100 in order to facilitate interpretation. Each outcome is a dummy variable indicating if the risk willingness measure is below or equal to a specific value m with $m = 0, \dots, 9$ (increasing in risk willingness). The regressors in each model are the same health shock indicator (loss of grip strength, 25% or more) and the same control variables as in the basic specification of the regression adjusted matching approach (column A, Table 3). The same matching weights are also applied.

A.3 Ordered Probit and Interval Regression

We also employ an ordered probit model and estimate the probability to report specific values of our outcome, plotted in Figure A2. The different bars now depict the probability of choosing a specific value of risk willingness and the dots refer to the estimated marginal effects on each of these categories. Figure A2 documents significant increases in the probability to report lower risk willingness (0 to 3). For values of moderate and high risk willingness (5 to 10) significant decreases as a result of a health shock are found. We get similar results if we estimate an interval regression model: The estimated effect is -0.253 with a standard error of 0.099 ($n = 10,034$).

Figure A2: Effects on the Responses to the Risk Willingness Question



Source: SOEP, v29, 2008-2012.

Figure is based on estimated statistics from an ordered probit model, scaled up a factor of 100 in order to facilitate interpretation. Outcome variable: Risk willingness measure, 11 different categories, increasing in risk willingness, s. Section 3.2. Regressors: The same health shock indicator (loss of grip strength, 25% or more) and the same control variables as in the basic specification of the regression adjusted matching approach (column A, Table 3). The same matching weights are also applied.

Table A2: Regression Results from Ordered Probit

n	Probability (Risk Willingness = n), in % (1)	Effect of a Health Shock (Grip Strength Loss, $\geq 25\%$), in percentage points (2)
0	5.81	1.33** (0.56)
1	8.85	1.21** (0.51)
2	14.51	1.14** (0.45)
3	15.65	0.46*** (0.16)
4	9.24	-0.04 (0.04)
5	20.76	-0.90** (0.39)
6	8.85	-0.77** (0.32)
7	8.74	-1.06** (0.42)
8	5.43	-0.90** (0.35)
9	0.97	-0.20** (0.09)
10	1.19	-0.29** (0.12)

Source: SOEP, v29, 2006-2012.

Figures in table are estimated statistics of an ordered probit model, scaled up by a factor of 100 in order to facilitate interpretation. Outcome variable: Risk willingness measure, 11 different categories, increasing in risk willingness. Regressors: health shock indicator (loss of grip strength, 25% or more), same control variables as in the basic specification of the regression adjusted matching approach (column A, Table 3). The same matching weights are also applied.