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ABSTRACT

The Effect of Early Life Health on Later Life Home Care Use: The Mediating Role of Household Composition*

In this paper we estimate the effect of early life health on home care use later in life, and we analyse whether this effect is mediated through household composition. We use Dutch administrative data on men born in 1944-1947 who were examined for military service between 1961-1965 and we link them to national data on non-residential care in the period 2004-2013 and data on house- hold status information for the years 1999-2014. We account for confounding factors that influence both early life health and later life home care use. We also account for selective attrition. Our empirical findings show that general health problems in youth is an important health predictor for later life home care use. A large portion of this effect is an indirect effect running through changes in partnership status.

JEL Classification: I10, C40, C30

Keywords: home care use, early life health, inverse propensity weighting, mediation

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1 Introduction

Population ageing is the most important demographic trend of this century. The rise in life expectancy has been coupled also with an increase in the number of years that people spend in poor health or disability. This demographic pressure is likely to have an impact on both the provision and financing of long-term care (LTC) around the world. Studies on the determinants of LTC use have focused mainly on current health. However, a large body of literature has shown that conditions early in life have an impact on health and mortality at older ages (Almond and Currie, 2011, Almond et al., 2018).

In this paper we study the impact of early life health (around age 18) on formal home care use[1] later in life (around age 60), and whether this effect is mediated through family structure. Many illnesses are inherently chronic and long lasting, leading to persistent health problems and need for home care. However, health problems may also be persistent because poor health in youth sets in motion lifelong trajectories of social and economic disadvantages, which in turn influence health outcomes later in life (Kuh and Shlomo, 2004). For example, early life health is likely to influence selection into and out of marriage and cohabitation (Guner et al., 2014, Van den Berg and Gupta, 2015). In turn, living with a partner has been shown to have a protective effect on health as married and cohabiting individuals are more likely to adopt healthier lifestyles, suffer from lower levels of psychological stress and engage in less risky behaviour (Gardner and Oswald, 2004, Lund et al., 2002, 2004, Murray, 2000).

We use Dutch administrative data on men born in 1944-1947 who were examined for military service between 1961-1965 and we link them to national data from the death register, data on persons who received non-residential care in the period 2004-2013 and data on household status information for the years 1999-2014. The military examination includes health measures at age 18, comprising height, weight and indicators of health problems (general health, hearing, vision and psychological problems), in addition to socio-economic information. For each individual we also observe whether he has used any home care later in life (in the years 2004-2014). In the Netherlands a large share of the long-term care is provided through formal home care, which has been increasing in recent years (Swinkels et al., 2016). The Netherlands has a separate mandatory public insurance system for legal entitlements to formal home care use which covers 100% of the population. Eligibility is assessed by independent government officials and is based on objective eligibility rules that are set centrally. However, the eligibility is contingent not only on medical needs but also on the availability of informal care. Having a partner increases the chance to use informal care when necessary.

From a methodological point of view, the association between early life health and later life home care use may be affected by confounding factors that influence both outcomes, such as parental background, education level and IQ. Often the mediator, a factor on the pathway from early life to later life home care use, household composition, is also affected by these confounding factors. Ignoring that such common factors exist may render the association spurious and therefore change the policy implications. Traditionally, causal mediation analysis has been formulated within the framework of linear structural models Baron and Kenny (1986). To study the role of household composition as a causal pathway through which poor health earlier in life translates into higher home care use at older ages, we build on recent advances in mediation analysis which allow us to distinguish between the

[1] LTC can be provided at home or in residential care facilities. Home care is non-residential care.

direct and indirect (through household composition) effect of early health on late life health (Huber, 2014, Imai et al., 2010a,b). We base our estimation of the mediation effects on the Inverse Propensity Weighting (IPW) method of Huber (2014). The advantage of the propensity score is that it enables us to summarize the many possible confounding covariates as a single score (Rosenbaum and Rubin, 1983).

Another issue that we need to take into account is that individuals who are observed on January 1, 2004, the first observation of home care use, may be a selective sample. It is likely that factors that influence home care use (including health at age 18) also influence observation (due to selective survival or migration) in 2004 (and subsequently in the years after). In this paper we propose an additional weighting, based on the attrition probability, to account for such selective observation.

As the estimator identifies causal mechanisms given that a sequential unconfoundedness condition holds, which is a strong and non–refutable assumption, we carry out a set of sensitivity analyses to quantify the robustness of our empirical findings to violations of the sequential ignorability assumption.

The paper proceeds as follows. Section 2 introduces the data and the descriptive statistics. Section 3 explains the econometric model we use for the mediation analysis. In Section 4 we discuss our results. Finally, Section 5 concludes.

2 Data and descriptive statistics

We draw data from a large sample of men born in 1944-1947 from the nationwide Dutch Military Service Conscription Register for the years 1961-1965. All men, except those living in psychiatric institutions or in nursing institutes for the blind or for the deaf-mute, were called to a military service induction examination at age 18. The data from the military examination include health measurements comprising height, weight and indicators of health problems. We use the General health status which was rated on a scale from 1 (fit) to 5 (unfit). We create indicators for poor general health which is equal to 1 if the score is above 2.

Our data also provide information on cognitive abilities at age 18. Cognitive abilities are measured through a standardized psychometric test battery including Raven Progressive Matrices, a nonverbal, untimed test that requires inductive reasoning about perceptual patterns, the Bennett Mechanical Comprehension test, and tests for Clerical Aptitude, Language Comprehension, Arithmetic and a Global comprehensive score, that combines all five tests. Scores for all tests were grouped in six levels from 1 (lowest) to 6 (highest), and we code missing values as 9. We only use the Global comprehensive score.

To control for conditions around birth, we also include birth order, family size, father's occupation, religion, region of birth and indicators for exposure to the Dutch famine during specific trimesters of the mother's pregnancy (see Ekamper et al., 2014). The Dutch famine took place in the winter 1944/1945 in the West Netherlands. The famine struck unexpectedly in a society without prior food shortages, the affected population had major problems obtaining food elsewhere. The famine was well defined in time (7 months) and space (cities in the West). Population was ethnically homogeneous without prior differences in dietary patterns. The details of this famine have been well documented (Lumey and van Poppel, 1994, Roseboom et al., 2001, Stein et al., 1975). After the German surrender in may

1945 the famine was soon over. Father's occupation was classified into five categories: professional and managerial workers; clerical, self-employed and skilled workers; farmers; semi-skilled workers including operators, process workers and shop assistants; and labourers and miners. Fathers with unknown occupations were classified separately. Religion is classified into five categories: roman catholic, Dutch reformed Calvinistic, other and none.

A sample of 45,037 men from the Dutch Military Service Conscription Register were linked by Statistics Netherlands to the Dutch residence and death register through the end of 2013, administrative data on non-residential care use for the years 2004-2013 and on household status information from 1999 to 2014 using unique personal identification numbers. The death register provides the age at death. After linking the data, our final sample includes 36,923 men. Of the 8,114 individuals missing from the initial sample, 3,442 are known to have died, 487 have emigrated and 4,185 are lost due to other reasons.

Recently, these data were also linked to the national home care use (2004-2014) and household status information (1999-2014) using unique personal identification numbers. Home care use is our main outcome of interest. Statistics Netherlands distinguishes four categories of formal home care use (non-residential care for which the expenditures are covered by the public insurance system): *household care*: an individual has received household assistance, such as cleaning and food preparation, which is partly paid through the 'Wet Maatschappelijke Ondersteuning (WMO)', the social support act; *personal care*: an individual has received personal care for which the expenditures are covered by 'Algemene Wet Bijzondere Ziektekosten (AWBZ)', General law on special sickness costs, such as aid with dressing, washing, eating and, drinking; *nursing care*: an individual has received nursing care for which the expenditures are covered by 'Algemene Wet Bijzondere Ziektekosten (AWBZ)', such as nursing, aid in medication use or injections and *total care*: an individual has received any of the three home care categories. For each individual we observe whether he has used any home care in a particular year and the number of hours of care used in that year. Of the original linked men 5,489 were lost from the sample between 1999, the start of the administrative registers and 2004, when the registration of home care use starts. Thus we have 36,923 men at the start of 2004 for whom we observe household care use 0.8%, personal care use 0.9%, nursing care 1.0% and total care use (which is either one of the three care categories) 1.7%. To check the possibility of selective observation of home care use in 2004 we report in Table 1 the demographic and socio-economic characteristics at the time of military examinations by observation status in 2004 and the significance of the difference. The table clearly shows that unhealthy men are more likely not to be observed in 2004, leading to potential attrition bias problems to which we return in Section 3.3.

The household register contains information on the type of household a person is living in. We distinguish 2 household types: either single or with a partner (with or without children). When looking at the relationship between household composition and home care use, we can clearly observe that men with a partner are less likely to need formal home care. While for this group the use of total care ranges between 0.2% and 1.5%, it is 5.4% for single men. As discussed earlier, this evidence can be explained both by the protective role of marriage and by a selection of healthy people into marriage and unhealthy people out of marriage (see Guner et al., 2014, Van den Berg and Gupta, 2015).

Figure 1 depicts the relation between home care prevalence and early life health, poor general

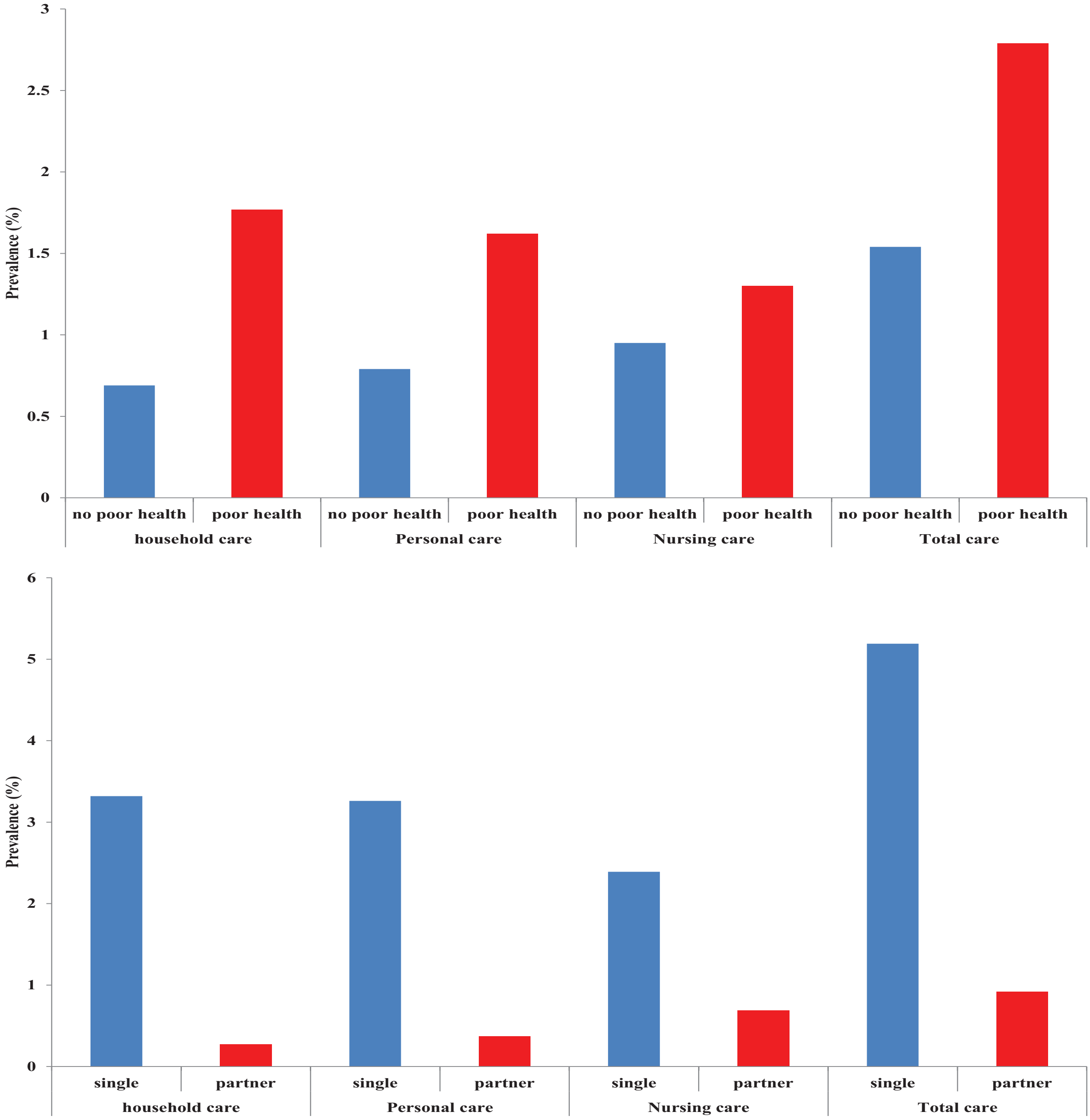
Table 1: Descriptive statistics - conditions at age 18

	Average		Difference
	Estimation sample	Missing in 2004	
height	177.60	177.47	0.13
Overweight	0.053	0.055	-0.002
poor general health	0.192	0.218	-0.027^{**}
poor sight	0.342	0.344	-0.003
poor psychological	0.254	0.284	-0.030^{**}
South	0.058	0.045	0.013^{**}
North	0.033	0.062	-0.029^{**}
East	0.047	0.041	0.006^{+}
birth rank	2.359	2.328	0.031
father's occupation			
professional	0.168	0.212	-0.045^{**}
white collar	0.283	0.285	-0.002
farm owner	0.065	0.061	0.004
skilled	0.269	0.241	0.028^{**}
unskilled	0.124	0.096	0.028^{**}
unknown	0.091	0.105	-0.014^{**}
religion			
Roman catholic	0.328	0.344	-0.016^{+}
Dutch reformed	0.302	0.305	-0.003
Calvinistic	0.076	0.065	0.010
Other	0.007	0.018	-0.011^{+}
None	0.287	0.267	0.020^{**}
IQ			
1 (lowest))	0.044	0.062	-0.018^{**}
2	0.223	0.221	0.002
3	0.196	0.184	0.012^{**}
4	0.320	0.315	0.005
5	0.113	0.107	0.006^{+}
6 (highest)	0.072	0.080	-0.008^{+}
9 (missing)	0.033	0.033	0.000
birth year			
1944	0.172	0.198	-0.026^{**}
1945	0.396	0.414	-0.018^{+}
1946	0.300	0.270	0.030^{**}
1947	0.132	0.118	0.014^{+}
$N =$	36,923	8,114	

$^{+}p < 0.05, ^{**}p < 0.01$.

health at age 18 and, between home care prevalence and household situation, partnership, in 2004. For three of the four home care categories (except for nursing care) we see a doubling of the home care prevalence if the individual was diagnosed with poor general health at the military examination. Having a partner also clearly influences the home care prevalences. All these differences are statistically significant on 1% level.

Figure 1: Home care use by health at age 18 and household situation in 2004



Notes. The first panel depicts the prevalence of home care use in 2004 by general health at the military examination at age 18. The second panel depicts the prevalence of home care use in 2004 by household situation in that year.

3 Methodology

We seek to find the impact of poor early health, measured by a binary health indicator at age 18, on the home care prevalence later in life (age 60-70) in our sample of male conscripts. We are especially interested in disentangling this effect of poor early life circumstances into a direct effect and an indirect effect (operating through household composition) on home care use.

3.1 Standard mediation analysis

Traditionally, causal mediation analysis has been formulated within the framework of linear structural equation models (LSEM) Baron and Kenny (1986). The LSEM assumes the following two equations: Let Y be the outcome, home care use, H the 'treatment', early life health , L the mediator, household composition (single or not), and X other included covariates.

$$L = \beta_L' X + \alpha_L H + \epsilon_L \tag{1}$$

$$Y = \beta_Y' X + \alpha_Y H + \gamma_L L + \epsilon_Y \tag{2}$$

where the error terms ϵ_L and ϵ_Y are assumed to be mutually independent. Then, the direct effect of early health, H, is α_Y and the indirect effect, running through the mediator, is $\gamma_L \cdot \alpha_L$. Note that this simple multiplication does not hold for a non-linear model.

3.2 Counterfactual approach

Recent papers have placed causal mediation analysis within the counterfactual/potential outcomes framework (Huber, 2014, Huber et al., 2016, Imai et al., 2010a,b) all assuming sequential unconfoundedness. Propensity score methods are increasingly used to take account of confounding in observational studies, e.g. see Caliendo and Kopeinig (2008) for a survey. We base our estimation of the mediation effects on the Inverse propensity weighting (IPW) method of Huber (2014).

Let H be the (early) health measure, a measurement of poor health. We denote by $Y(h)$ and $L(h)$ the potential outcome, home care use, and mediator, household type, under 'treatment', poor early life health, $h = 0, 1$. For each individual we only observe one of the potential outcomes and mediation states, with the observed states are ; $Y = Y(1) \cdot H + Y(0) \cdot (1 - H)$ and $L = L(1) \cdot H + L(0) \cdot (1 - H)$. The average treatment effect (of poor early health) is defined by $\mathrm{E}\big[Y(1) - Y(0)\big]$. To disentangle this into a direct and an indirect effect (operating through household composition L), rewrite the potential outcome as a function of both the treatment, early health H, and the mediator, household composition L: $Y(h) = Y\big(h, L(h)\big)$. This allows to formulate the direct effect $\theta(h)$, the fraction of the total effect that is not attributed to the mediator, and the indirect effect $\eta(h)$, the fraction of the total effect attributed to the mediator by:

$$\theta(h) = \mathrm{E}\Big[Y\big(1, L(h)\big) - Y\big(0, L(h)\big)\Big] \quad h = 0, 1 \tag{3}$$

$$\eta(h) = \mathrm{E}\Big[Y\big(h, L(1)\big) - Y\big(h, L(0)\big)\Big] \quad h = 0, 1 \tag{4}$$

Note that the average effect is the sum of the direct and indirect effects:

$$\begin{aligned} \mathrm{E}\big[Y(1)-Y(0)\big] &= \mathrm{E}\Big[Y\big(1,L(1)\big)-Y\big(0,L(0)\big)\Big] \\ &= \mathrm{E}\Big[Y\big(1,L(1)\big)-Y\big(0,L(1)\big)\Big]+\mathrm{E}\Big[Y\big(0,L(1)\big)-Y\big(0,L(0)\big)\Big]=\theta(1)+\eta(0) \quad (5)\\ &= \mathrm{E}\Big[Y\big(1,L(1)\big)-Y\big(1,L(0)\big)\Big]+\mathrm{E}\Big[Y\big(1,L(0)\big)-Y\big(0,L(0)\big)\Big]=\eta(1)+\theta(0) \end{aligned}$$

For identification of the causal mediation analysis we need to impose unconfoundedness and sequential ignorability:
Unconfoundedness: $Y(h)\perp H|X$ for $h=0,1$

where $\perp$ denotes independence. The unconfoundedness assumption (Rosenbaum and Rubin, 1983, Rubin, 1974) asserts that, conditional on (pre-treatment) covariates X, treatment assignment (early life health) is independent of the potential outcomes. This assumption requires that all variables that affect both home care use and early life health are observed. Note that this does not imply that we assume all relevant covariates are observed. Any missing factor is allowed to influence either the outcome or the early life health, not both. We check the robustness of our estimates to this, rather strong, unconfoundedness assumption by assessing to what extent the estimates are robust to violations of this assumption induced by including an additional simulated binary variable to capture unobservables (Ichino et al., 2008, Nannicini, 2007), see Section 4.1.

Rosenbaum and Rubin (1983) show that if the potential outcomes are independent of treatment conditional on covariates X, they are also independent of treatment conditional on the propensity score, $p(x)=\Pr(H=1|X=x)$. Hence if unconfoundedness holds, all biases due to observable covariates can be removed by conditioning on the propensity score Imbens (2004). The average effects can be estimated by matching or weighting on the propensity score. Here we use weighting on the propensity score. Inverse probability weighting based on the propensity score creates a pseudo-population in which the early life health is independent of the measured covariates. The pseudo-population is the result of assigning to each individual a weight that is proportional to the inverse of their propensity score. Inverse probability weighting (IPW) estimation is usually based on normalized weights that add to unity.

$$W_i=\left[\frac{H_i}{\hat{p}(X_i)}\Big/\sum_{j=1}^{n}\frac{H_j}{\hat{p}(X_j)}\right]+\left[\frac{(1-H_i)}{1-\hat{p}(X_i)}\Big/\sum_{j=1}^{n}\frac{1-H_j}{1-\hat{p}(X_j)}\right] \tag{6}$$

where $\hat{p}(X_j)$ is the estimated propensity score.

The counterfactual framework enables us to disentangle the underlying causal pathway from early health to home care use into an effect of early health through a change in household composition (mediator or indirect effect) and an effect through other pathways (direct effect). We assume conditional independence (given X) of the treatment and the mediator:

Sequential Ignorability Assumption.

The following two statements of conditional independence hold:
$\{Y_i(h', j), L(h)\} \perp H | X$ and $Y_i(h', j) \perp L(h) | H = h, X$, $\forall h, h'$ and j in the support of L.

Huber (2014) shows that under the Sequential Ignorability Assumption the direct and indirect effects are identified using an inverse propensity score (IPW) method. The first condition of the Sequential Ignorability Assumption implies that, conditional on observed covariates X, no unobserved confounder exists that jointly affects early life health, the household type and the home care use. The second condition implies that, conditional on observed covariates X and early life health, no unobserved confounder exists that jointly affects the household type and the home care use. Sequential Ignorability Assumption is a strong assumption and non–refutable. We therefore carry out a set of sensitivity analyses to quantify the robustness of our empirical findings to violation of the sequential ignorability assumption in Section 4.1. We also have a common support restriction for the propensity score $\Pr(H = 1|X, L)$ including the mediator, i.e. the probability of poor early health given covariates X and mediator L, is bounded between zero and one.

Huber (2014) derives the weights needed to calculate the direct and indirect effects. The weights for the total effect are given in (6). The weights for the direct effect of poor early life on home care use $\theta(h)$, $(h = 0, 1)$ are

$$W_{Dir}(0) = \frac{1-\hat{p}(X,L)}{1-\hat{p}(X)}\left[\frac{H}{\hat{p}(X,L)} - \frac{(1-H)}{1-\hat{p}(X,L)}\right] \tag{7}$$

$$W_{Dir}(1) = \frac{1}{\hat{p}(X)}\left[H - (1-H)\frac{\hat{p}(X,L)}{1-\hat{p}(X,L)}\right] \tag{8}$$

where $\hat{p}(X, L)$ is the estimated propensity score including the household composition, L. The weights for the indirect effect of poor early life operating through household composition on home care use $\eta(h)$, $(h = 0, 1)$ are

$$W_I(0) = \frac{I(H=0)}{1-\hat{p}(X,L)}\left[\frac{\hat{p}(X,L)}{\hat{p}(X)} - \frac{1-\hat{p}(X,L)}{1-\hat{p}(X)}\right] \tag{9}$$

$$W_I(1) = \frac{I(H=1)}{\hat{p}(X,L)}\left[\frac{\hat{p}(X,L)}{\hat{p}(X)} - \frac{1-\hat{p}(X,L)}{1-\hat{p}(X)}\right] \tag{10}$$

with $I(H = h)$ is the indicator function. For estimation we use normalized versions of the sample implied by the weights, such that the weights in either treatment or control groups add up to unity (similar to (6)). We estimate the additional propensity scores conditional on the pre-treatment covariates and the mediator, $\hat{p}(X, L)$, by logit specifications. We assume a linear probability model for the home care prevalences and the total, direct and indirect effects can be estimated using a weighted (IPW) OLS for each observation year (2004–2013) separately. We estimate the standard errors by 1000 bootstrap draws.

3.3 Selective Attrition

Both poor health and home care use are related to mortality (and other attrition processes) as can be seen in Figure 2. Of the men diagnosed with poor general health at age 18 11% was not observed in

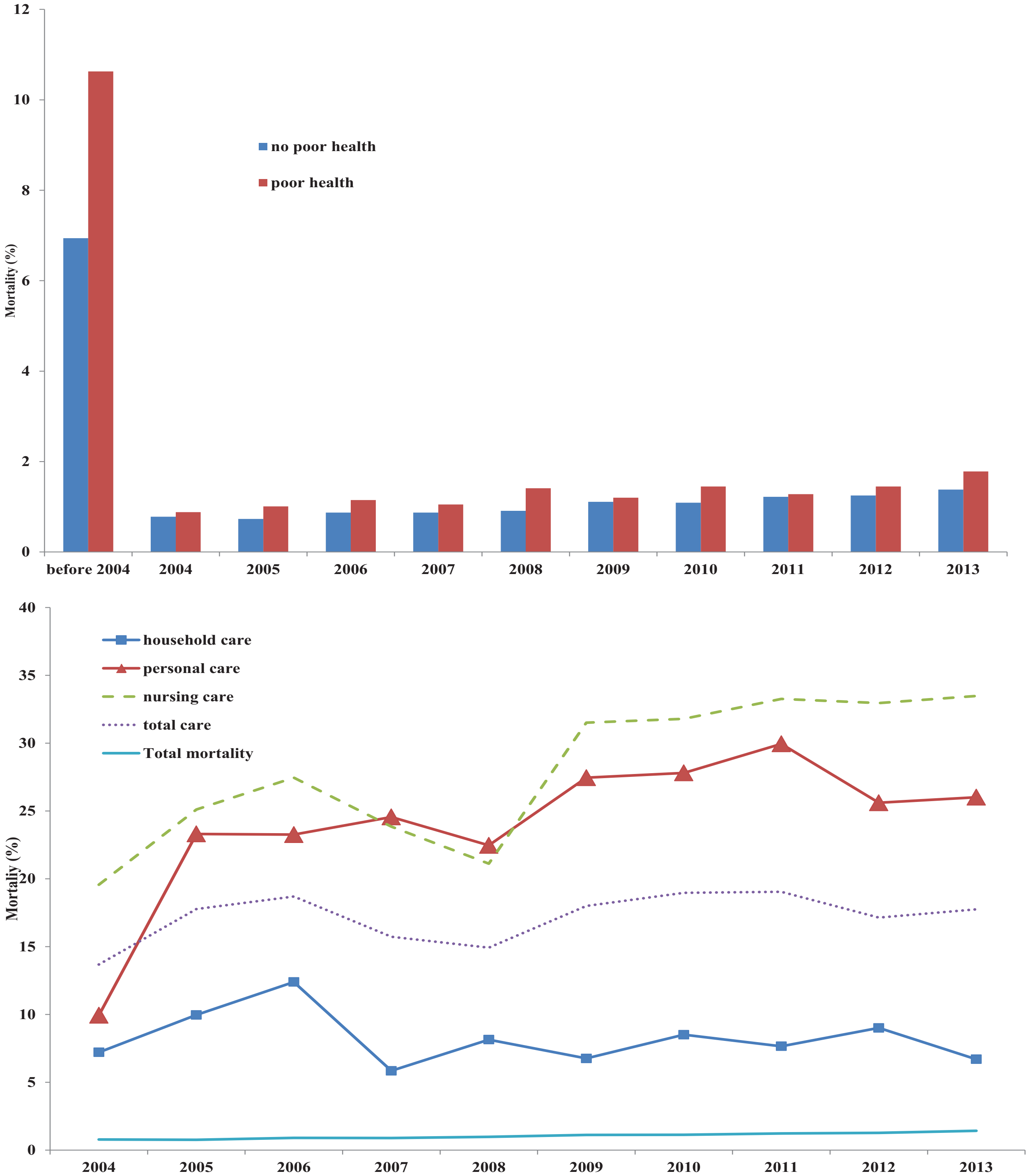
2004 (did not survive or emigrated), while only 7% of those without health problems are not observed (died) in 2004. In the years after 2004 the attrition is still higher for those with diagnosed health problems at age 18.[2] As we do not know whether any man used home care before 2004 (probably a negligible percentage) we can only relate home care use to attrition after 2004. For all home care categories mortality is elevated when men are observed users, especially when using personal and nursing care.

Thus, individuals who are observed on 1/1/2004, the first observation of home care use, may be a selective sample. It is likely that factors that influence the home care use (including health at age 18) also influence attrition till 2004 (and subsequently in the years after). We propose to use an additional weighting, based on the attrition probability, to account for selection on observables. Thus, we assume (again) unconfoundedness for the attrition. To this end we estimate a logit model for attrition (i.e. observation) till 2004 based on the whole sample of 45,037 men, including the same covariates as in the previous propensity score and the poor general health indicator. Each individual is weighted by the estimated probability to be observed in 2004.

Of course, even after 2004 attrition may be selective (related to home care use, household composition, early health and other individual characteristics). We therefore also estimate for each consecutive year (and each home care use separately) the (logit) attrition probability based on the same control variables and, additionally, the poor general health indicator, the home care use in the preceding year and the household composition. We calculated weights based on the inverse of these estimated attrition probabilities and re-estimate the total, direct and indirect effects of poor health on home care use using the weights in (6)-(10) together with these attrition weights in each the consecutive year. Again we calculate the standard errors by 1000 bootstrap draws.

[2] Note that only the first attrition difference and attrition differences for the years 2008 and 2010 between those with poor and with no poor health is statistically significant on a 5%-level.

Figure 2: Attrition by health at age 18 and home care use



Notes. The first panel depicts the percentage that was not observed by general health at the military examination at age 18. The second panel depicts the percentage that was not observed by home care use in each year.

4 Results

First we estimate a 'standard' Linear Structural equations Model (LSEM), defined in (1) and (2), for each of the four possible home care use types for each year of observation. In all models we include poor general health (the health assessment was fairly or worse) as early health indicator measured at age 18 and having a partner as the mediator. We include other controls that may also influence the home care use: family size, birth rank, region of birth, father's occupation (in six categories), IQ measurement at age 18, religion, a trend in birth month (and trend-squared). We also included another measure of poor health, the famine exposure in utero indicators : Post-natal, born in January 1944 - October 1944; 3rd trimester, born November 1944- January 1945; 2nd trimester, born February 1945 - April 1945; 1st trimester, born may 1945 - July 1945; Pre-conception, born August 1945 - March 1946, the famine cities dummy and the interaction between the time dummies and the famine cities dummy (which give the DiD results).

After fitting each linear equation via ordinary least squares the LSEM results are reported in Table 2. We find large (about 70% of the observed prevalence) effect of poor early health on home care use. The first column in Table 2 presents the raw difference in home care use by general health indication (poor vs not poor). For both household and total care the LSEM results indicate significant total and direct poor health effects on the use of these formal care for the whole observation period 2004–2013. For personal and nursing care the LSEM results are only significant for a few years. For none of the home care categories the LSEM finds a significant indirect effect of poor early life health running through household composition.

The LSEM imposes linear equations. This is relaxed in when using an IPW method. Next we estimate the poor health effects on home care use using the IPW method. We use the same control variables to estimate the propensity score of poor health indicator using a logit model.[3] For an IPW method to hold we need to check if the propensity score is able to balance the distribution of all included variables in both the control (no poor general health) and treated (poor general health) group. One suitable way to check whether there are still differences is by calculating the standardized bias, or normalised difference in means. The standardized bias for each included control variable reveals substantial imbalances between those who have good and those who have poor general health before weighting. These imbalances disappear when we use the inverse propensity weights.[4]

As we mentioned in Section 3.3 the individuals for which we observe their home care home in 2004 (and beyond) may be a selective sample of the original linkage sample. To account for this selectivity we therefore estimate a logit model for attrition (observation in 2004) and additionally a logit model for attrition in each consecutive year.[5] Based on the implied attrition probability we impose an additional weight on the estimation to account for selective attrition. Table 3 presents the total estimated effect and the decomposition estimated effects of the early poor general health health indicator on home care use probability for 2004-2013 accounting for selective attrition.

[3]The Table A.1 in the appendix presents the estimated coefficients.

[4]Table A.2 in the appendix reports the standardized bias for each included control variable before and after adjusting the data using inverse propensity weighting.

[5]The Table A.1 in the appendix presents the estimated coefficients for attrition in 2004. The coefficients of the estimated logit models for consecutive years, accounting for home care use and household composition in the preceding years, are available upon request.

Table 2: Impact of early poor general health on home care probability and how it is mediated using LSEM, 2004-2013

	raw difference	OLS[a]		
		total	direct	indirect
	Household care			
2004	1.08%**	0.68%**	0.56%**	0.12%
2005	1.12%**	0.71%**	0.57%**	0.14%
2006	1.27%**	0.82%**	0.66%**	0.16%
2007	1.13%**	0.71%**	0.55%**	0.16%
2008	1.05%**	0.58%**	0.40%*	0.18%
2009	1.13%**	0.73%**	0.54%**	0.26%
2010	1.39%**	0.93%**	0.70%**	0.24%
2011	1.54%**	0.97%**	0.73%**	0.24%
2012	1.54%**	0.95%**	0.70%**	0.25%
2013	1.59%**	1.09%**	0.84%**	0.24%
	Personal care			
2004	0.83%**	0.40%**	0.29%*	0.12%
2005	0.70%**	0.37%*	0.29%*	0.08%
2006	0.37%**	0.10%	0.02%	0.07%
2007	0.51%**	0.29%+	0.21%	0.08%
2008	0.82%**	0.44%*	0.34%*	0.10%
2009	0.46%**	−0.00%	−0.10%	0.09%
2010	0.86%**	0.48%*	0.37%*	0.11%
2011	1.13%**	0.75%**	0.65%**	0.10%
2012	0.63%**	0.30%	0.18%	0.12%
2013	0.65%**	0.26%	0.13%	0.12%
	Nursing care			
2004	0.35%+	0.13%	0.06%	0.07%
2005	0.65%**	0.24%	0.18%	0.06%
2006	0.66%**	0.30%	0.22%	0.08%
2007	0.50%**	0.26%	0.18%	0.08%
2008	0.48%**	0.14%	0.06%	0.08%
2009	0.42%+	0.13%	0.06%	0.07%
2010	0.55%**	0.29%	0.24%	0.05%
2011	0.65%**	0.37%+	0.33%	0.04%
2012	0.61%**	0.54%**	0.48%**	0.06%
2013	0.52%**	0.34%	0.28%	0.06%
	Total care			
2004	1.25%**	0.63%**	0.46%*	0.17%
2005	1.48%**	0.77%**	0.62%**	0.16%
2006	1.71%**	0.99%**	0.81%**	0.18%
2007	1.65%**	0.99%**	0.79%**	0.20%
2008	1.75%**	0.88%**	0.64%*	0.24%
2009	1.58%**	0.82%**	0.58%*	0.24%
2010	1.79%**	1.05%**	0.78%**	0.27%
2011	2.17%**	1.34%**	1.08%**	0.26%
2012	1.94%**	1.33%**	1.05%**	0.20%
2013	1.86%**	1.20%**	0.92%**	0.28%

[a] Included exogenous covariates: birth order, family size, father's occupation, IQ scores, religion, quadratic trend of the birth date, famine exposure and region of birth. $^{+}p < 0.10, ^{*}p < 0.05, ^{**}p < 0.01$.

In the first column of Table 3 presents total effect of poor general health on each of the four home care probability for 2004-2013 using the inverse propensity weighted method with the weights defined in (6). Next we decompose the total effect into an effect running through household composition (indirect effect) and running through other pathways (direct effect). We distinguish two different decompositions, depending on the whether we look at individuals with poor health or not.

Comparing the IPW results with the LSEM results reveals that not controlling for selectivity lead to overestimate the impact of early health on later life home care use in early years, 2004-2006, and underestimates the impact in later years, 2009-2013.[6] Poor health at age 18 increased the prevalence of household care in 2004 with 0.5% and the prevalence of total care with 0.4%. In 2013 the impact is 1.8% on household care and 2.8% on total care (and 1.5% on personal care and 1.1% on nursing care). For those without poor health at age 18 a large proportion of this effect is a direct impact of poor health, $\theta(0)$. For those with poor health at age 18 we only find a significant direct effect, $\theta(1)$, for a few observation years. For nursing care we do not find any significant direct effect of poor early life health. For household and total care use we find that the direct effect of poor health for those with poor health, $\theta(1)$, is larger than the direct effect of poor health for those without poor health, $\theta(0)$. For personal care we only find a significant direct effect of poor health in 2011. The effect of poor health on home care use running through household composition for those with poor health, $\eta(1)$, is significant for all home care categories. This indirect effect is increasing over most of the observation years. In 2013 poor health running through household composition is larger for those with poor health, $\eta(1)$, than for those without poor health, $\eta(0)$. Note that, although the direct effect for those with poor and without poor early health seems to differ a lot, this difference is not significant (for none of the years for none of the home care categories). The same hold for the indirect effects.

[6]Comparing these results with the results from an IPW without account for selective attrition, see Table A.3 in the appendix, we find that accounting for selective attrition increases the estimated impact of early life poor health on home care use, especially in later years.

Table 3: Total, direct and indirect effect of early poor general health on home care probability, 2004-2013, linear probability model with IPW, accounting for selective attrition

	Total effect	Direct effect		Indirect effect	
		$\theta(1)$	$\theta(0)$	$\eta(0)$	$\eta(1)$
	Household care				
2004	0.49%**	0.41%*	0.35%*	0.08%	0.15%**
2005	0.51%**	0.37%$^{+}$	0.35%*	0.15%	0.17%**
2006	0.65%**	0.46%$^{+}$	0.46%*	0.19%$^{+}$	0.19%**
2007	0.67%**	0.41%	0.44%*	0.26%*	0.23%**
2008	0.57%*	0.19%	0.33%	0.38%**	0.24%**
2009	0.88%**	0.54%	0.56%*	0.34%*	0.31%**
2010	1.11%**	0.67%$^{+}$	0.71%*	0.43%**	0.39%**
2011	1.37%**	0.69%	0.90%*	0.69%**	0.47%**
2012	1.41%**	0.52%	0.92%*	0.88%**	0.48%**
2013	1.80%**	0.85%	1.29%**	0.95%**	0.52%**
	Personal care				
2004	0.22%	0.06%	0.09%	0.16%	0.13%**
2005	0.25%	0.14%	0.15%	0.11%	0.10%**
2006	−0.03%	−0.06%	−0.13%	0.05%	0.10%**
2007	0.20%	0.13%	0.09%	0.07%	0.11%**
2008	0.47%	−0.01%	0.29%	0.48%*	0.18%**
2009	0.14%	−0.50%	−0.02%	0.64%*	0.17%**
2010	0.58%	0.37%	0.40%	0.21%	0.19%**
2011	1.17%*	1.05%*	0.91%*	0.12%	0.26%**
2012	0.80%	0.35%	0.47%	0.45%	0.24%**
2013	1.51%*	0.72%	1.08%	0.79%**	0.46%**
	Nursing care				
2004	0.06%	0.00%	−0.02%	0.06%	0.08%**
2005	0.12%	−0.00%	0.04%	0.12%	0.08%**
2006	0.12%	0.09%	0.04%	0.03%	0.08%*
2007	0.24%	−0.06%	0.16%	0.30%	0.08%*
2008	0.07%	−0.15%	−0.06%	0.22%	0.13%**
2009	−0.04%	−0.30%	−0.10%	0.25%	0.06%*
2010	0.24%	0.02%	0.18%	0.22%	0.06%$^{+}$
2011	0.37%	0.34%	0.27%	0.03%	0.10%$^{+}$
2012	0.73%$^{+}$	0.72%	0.60%	0.02%	0.13%*
2013	1.14%$^{+}$	0.80%	0.93%	0.34%	0.21%*
	Total care				
2004	0.38%$^{+}$	0.24%	0.19%	0.14%	0.19%**
2005	0.60%*	0.29%	0.39%	0.30%*	0.21%**
2006	0.75%**	0.56%$^{+}$	0.52%$^{+}$	0.19%	0.23%**
2007	0.96%**	0.45%	0.68%*	0.51%*	0.28%**
2008	0.86%*	0.12%	0.49%	0.74%**	0.37%**
2009	1.17%*	0.34%	0.72%$^{+}$	0.83%**	0.44%**
2010	1.42%**	0.79%	0.92%*	0.63%**	0.50%**
2011	1.95%**	1.22%	1.34%*	0.72%**	0.61%**
2012	2.24%**	1.06%	1.56%*	1.18%**	0.68%**
2013	2.76%**	1.38%	2.00%**	1.39%**	0.76%**

Included exogenous covariates in the estimation of the propensity scores: birth order, family size, father's occupation, IQ scores, religion, quadratic trend of the birth date, famine exposure and region of birth. $^{+}p < 0.05$, $^{**}p < 0.01$.

4.1 Sensitivity analysis

The critical assumption in propensity score weighting is that of no selection on unobservables. To test the sensitivity of matching estimators to the unconfoundedness assumption we build on the sensitivity analyses of Nannicini (2007) and Ichino et al. (2008). The Ichino et al. (2008) sensitivity analysis assumes that the possible unobserved confounding factors can be summarised in a binary variable, U, and that the unconfoundedness assumption holds conditional on X and U,$Y(h) \perp H|X,U$ and we also impose sequential ignorability conditional on U: (i) $\{y(h',q), L(h)\} \perp H|X,U$ and (ii) $y(h',q) \perp L|H=h,X,U$ for $\forall h,h'=0,1$ and q in the support of L. Given the values of the probabilities that characterize the distribution of U we can simulate a value of the unobserved confounding factor for each individual and re-estimate the model. Then, the distribution of the unobserved binary confounding factor U can be characterised by specifying the probabilities in each of the four groups.

$$p_{ij} = \Pr(U=1|H=i,Y=j,X) = \Pr(U=1|H=i,Y=j) \qquad i,j=0,1 \tag{11}$$

A measure of how the different configurations of p_{ij}, chosen to simulate U, translate into associations of U with the outcome is ω, the coefficient of U in a linear regression for the control group ($H=0$) using U and X as covariates. Nannicini (2007) call this coefficient the 'outcome effect'. A measure of the effect of U on the relative probability to be assigned to the treatment is ξ, with ξ the coefficient of U in a logit model on the treatment assignment ($H=1$) using U and X as covariates. Nannicini (2007) call this (exponentiated) coefficient the 'selection effect'. A new measure we introduce is, the 'mediator-effect', ψ, the coefficient of U in a logit model on the household composition for the control group using U and X as covariates.

We search for the existence of 'killer'-confounders, i.e. the existence of a set of probabilities p_{ij} such that if U were observed the estimated effects would be driven to zero. The reason for doing this is to assess the plausibility of the resulting configuration of U and how comparable this is to the distribution of observed confounders. In order to reduce the dimensionality of the characterisation of the 'killer'-confounders we follow the suggestion of Nannicini (2007) and fix the probability of $\Pr(U=1)$ to 0.4 and the difference $p_{11}-p_{10}$ to zero. Now the simulated confounders U can be fully described by two differences $d = p_{01}-p_{00}$ and $s = p_{1.}-p_{0.}$, with $p_{i.} = \Pr(U=1|D=i) = p_{i1} \cdot \Pr(\delta_1 = 1|D=i) + p_{i0} \cdot \Pr(\delta_1 = 0|D=i)$ for $i=0,1$, the fraction of individuals with $U=1$ by education level. Nannicini (2007) argues that d is an (inconsistent) measure of the effect of U on the outcome (home care use) for the untreated (lower education level), while s is an (inconsistent) measure of the selection into treatment (higher education level). Both d and s are inconsistent measures because they do not account for the association between U and W, while our outcome, Ω, selection effects, ξ and mediation effects ψ, account for this.

For each probability configuration of U we repeat the simulation of U, the estimation of the outcome effect, the selection effect and the estimation of the treatment effects $M=100$ times and obtain the average of these 100 simulations. The total variance of these averages can be estimated from (see Nannicini (2007)):

$$\text{Var}_f = \frac{1}{M}\sum_{m=1}^{M} s_m^2 + \frac{M-1}{M(M-1)}\sum_{m=1}^{M}(\hat{f}_m - \bar{f})^2 \tag{12}$$

with $f \in \{\omega, \xi\}$ of each pairwise education comparison, $\hat{f}_m$ is the estimated f in each simulation sample m and s_m^2 is its estimated variance.

Next we re-estimate the total effect of early life poor general health on on home care use including U in the propensity score and the decomposition of the effect into a direct and an indirect effect. We only present the results for total home care use. Table 4 reports the simulated difference in total effect and the difference of its decomposition compared to the original estimates when the distribution of U is defined by d, s with $d, s = 0.1, \ldots, 0.5$ for observation year 2013.[7] Although for high s the differences with the original estimates of the total and direct effects seems large these differences are not significant.

[7]Table A.5 to Table A.7 in the appendix reports the sensitivity of the estimation for the three home care use categories. The sensitivity results for other observation years are available upon request. The simulated outcome-, selection and mediation effects can be found in Table A.4 in Appendix A.

Table 4: Sensitivity analysis characterizing 'killer' confounders (mediator), difference with original estimates 2013, *Total care*

	Total effect	Direct effect		Indirect effect	
		$\theta(1)$	$\theta(0)$	$\eta(0)$	$\eta(1)$
original	2.76%**	1.38%	2.00%**	1.39%**	0.76%**
$d = 0.1$ & $s = 0.1$	−0.10%	−0.11%	−0.07%	0.00%	−0.02%
$d = 0.1$ & $s = 0.2$	0.03%	−0.06%	−0.01%	0.03%	−0.01%
$d = 0.1$ & $s = 0.3$	0.06%	−0.02%	0.04%	0.07%	0.00%
$d = 0.1$ & $s = 0.4$	0.12%	0.00%	0.11%	0.11%	0.01%
$d = 0.1$ & $s = 0.5$	0.18%	0.02%	0.16%	0.15%	0.02%
$d = 0.2$ & $s = 0.1$	−0.07%	−0.09%	−0.05%	0.01%	−0.02%
$d = 0.2$ & $s = 0.2$	0.03%	−0.02%	0.04%	0.04%	−0.01%
$d = 0.2$ & $s = 0.3$	0.14%	0.04%	0.13%	0.09%	0.01%
$d = 0.2$ & $s = 0.4$	0.23%	0.09%	0.21%	0.13%	0.02%
$d = 0.2$ & $s = 0.5$	0.26%	0.08%	0.24%	0.17%	0.03%
$d = 0.3$ & $s = 0.1$	−0.05%	−0.07%	−0.03%	0.01%	−0.02%
$d = 0.3$ & $s = 0.2$	0.07%	0.02%	0.09%	0.05%	−0.01%
$d = 0.3$ & $s = 0.3$	0.21%	0.10%	0.20%	0.10%	0.01%
$d = 0.3$ & $s = 0.4$	0.29%	0.14%	0.27%	0.14%	0.02%
$d = 0.3$ & $s = 0.5$	0.27%	0.08%	0.24%	0.17%	0.03%
$d = 0.4$ & $s = 0.1$	−0.04%	−0.07%	−0.02%	0.02%	−0.02%
$d = 0.4$ & $s = 0.2$	0.12%	0.06%	0.13%	0.06%	0.00%
$d = 0.4$ & $s = 0.3$	0.25%	0.13%	0.23%	0.11%	0.01%
$d = 0.4$ & $s = 0.4$	0.29%	0.14%	0.27%	0.14%	0.02%
$d = 0.4$ & $s = 0.5$	3.27%	0.09%	0.24%	0.17%	0.03%
$d = 0.5$ & $s = 0.1$	−0.04%	−0.07%	−0.02%	0.02%	−0.02%
$d = 0.5$ & $s = 0.2$	0.14%	0.07%	0.14%	0.06%	0.00%
$d = 0.5$ & $s = 0.3$	0.24%	0.13%	0.23%	0.11%	0.01%
$d = 0.5$ & $s = 0.4$	0.29%	0.14%	0.27%	0.14%	0.02%
$d = 0.5$ & $s = 0.5$	0.27%	0.09%	0.24%	0.17%	0.03%

Based on adding U to propensity score under the assumption that $\Pr(U = 1) = 0.4$ and $p_{01} - p_{00} = 0$, the differences $d = p_{11} - p_{10}$ and $s = p_{1.} - p_{0.}$. $^{+}p < 0.05$ and $^{**}p < 0.01$.

5 Conclusion

Most studies on the determinants of LTC use have focused on the impact of current health status on the prevalence of formal home care. In this paper we estimate the causal effect of early life health on home care use later in life, and we analyse whether this effect is mediated through household composition.

We use conscription data of Dutch men born between 1944-1947 who were examined for military service between 1961-1965, and linked to national death records, data on persons who received non-residential care in the period 2004-2013 and data on household status information. A limitation of our datais that we only observe men and no information on women is available.

Using an IPW method we estimate the impact of poor health at age 18, the military examination, on home care use later in life. We decompose the impact of poor health into an effect running through household composition (indirect effect) and an effect running through other pathways (direct effect). We account for possible selective attrition till 2004 (and consecutive years) by additionally weighting by the estimated attrition probability.

We find a large impact of poor early life health on home care use, especially in later years. Poor health at age 18 increases the prevalence of household care in 2013 with 1.80% on household care, 1.51% on personal care, 1.14% on nursing care and, 2.76% on total care. For those without poor health at age 18 a large proportion of this effect is a direct impact of poor health. For personal care we only find a significant direct effect of poor health in 2011. The effect of poor health on home care use running through household composition for those with poor health is significant for all home care categories. This indirect effect is increasing over most of the observation years.

From both an academic and a policy perspective, knowing the social mechanisms by which early life health disadvantages translate into poor health outcomes later in life is crucial for the understanding of how to reduce health inequalities and long-term care costs.

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Appendix A Additional tables

Table A.1: Parameters logit propensity scores

	Attrition 2004	Poor general health	Mediation
poor general health	-0.332^{**}	–	–
	(0.051)		
partner	–	–	-0.251^{**}
			(0.041)
famine exposure in West			
Pre-conception	−0.011	0.117	0.125
	(0.134)	(0.119)	(0.119)
1st trimester	0.353^{+}	−0.001	−0.005
	(0.179)	(0.185)	(0.185)
2nd trimester	0.014	−0.006	−0.009
	(0.175)	(0.179)	(0.179)
3rd trimester	−0.358	−0.055	−0.048
	0.213)	(0.180)	(0.180)
Post-natal	(0.181	0.085	0.086
	0.131)	(0.130)	(0.130)
famine cities	−0.098	0.059	0.049
	(0.099)	(0.084)	(0.084)
famine exposure period			
Pre-conception	−0.015	−0.211	−0.219
	(0.140)	(0.124)	(0.124)
1st trimester	−0.317	−0.249	−0.243
	(0.196)	(0.200)	(0.200)
2nd trimester	−0.114	−0.355	−0.349
	(0.209)	(0.208)	(0.208)
3rd trimester	0.311	−0.082	−0.078
	(0.260)	(0.229)	(0.230)
Post-natal	−0.107	−0.144	−0.136
	(0.264)	(0.258)	(0.258)

First column gives the coefficients in a logit model of attrition in 2004. The second column gives the coefficients of a logit model of poor general health (excluding the mediator household composition). The third column gives the coefficients of a logit model of poor general health including the mediator. Reference category: region West, father's occupation professional, religion Catholic, born outside famine exposure period, height 170–185 cm, IQ-level 5. $^{+}p < 0.05$, $^{**}p < 0.01$.

Table A.1: Parameters logit propensity score (continued)

	Attrition 2004	Poor general health	
		Med	
family size	−0.014	0.102**	0.099**
	(0.012)	(0.011)	(0.011)
birth rank	0.023	−0.100**	−0.096**
	(0.013)	(0.012)	(0.012)
South	−0.075	−0.038	−0.035
	(0.110)	(0.101)	(0.101)
North	−0.053	0.120	0.126
	(0.119)	(0.112)	(0.112)
East	−0.211	0.166	0.173
	(0.109)	(0.099)	(0.099)
father's occupation			
white collar	−0.013	−0.108+	−0.102+
	(0.058)	(0.051)	(0.051)
farm owner	−0.080	−0.281**	−0.266**
	(0.112)	(0.107)	(0.107)
skilled	−0.116	−0.091	−0.081
	(0.059)	(0.053)	(0.053)
unskilled	−0.184**	−0.099	−0.094
	(0.069)	(0.063)	(0.063)
unknown	−0.275**	0.493**	0.480**
	(0.079)	(0.068)	(0.068)
IQ			
1 (highest)	−0.157**	−0.039	−0.039
	(0.054)	(0.049)	(0.049)
2	−0.183**	0.066	0.062
	(0.061)	(0.054)	(0.054)
4	−0.312**	0.146+	0.139+
	(0.069)	(0.063)	(0.063)
5	−0.611**	0.188**	0.174**
	(0.072)	(0.072)	(0.072)
6 (lowest)	−0.562**	0.762**	0.723**
	(0.098)	(0.084)	(0.085)
9 (missing)	−0.663**	2.166**	2.134**
	(0.093)	(0.070)	(0.070)
religion			
Dutch reformed	0.021	0.146**	0.149**
	(0.048)	(0.044)	(0.044)
Calvinistic	0.014	−0.011	−0.002
	(0.079)	(0.071)	(0.071)
Other	0.660**	0.880**	0.906**
	(0.197)	(0.140)	(0.139)
None	−0.100	−0.005	−0.009
	(0.049)	(0.047)	(0.047)
trend	0.245	0.177	0.185
	(0.208)	(0.206)	(0.206)
trend-squared	−0.032	−0.051	−0.052
	(0.038)	(0.037)	(0.037)
constant	2.752**	−2.504**	−2.301**
	(0.332)	(0.322)	(0.324)

First column gives the coefficients in a logit model of attrition in 2004. The second column gives the coefficients of a logit model of poor general health (excluding the mediator household composition). The third column gives the coefficients of a logit model of poor general health including the mediator. Reference category: region West, father's occupation professional, religion Catholic, born outside famine exposure period, height 170–185 cm, IQ-level 5. $^{+}p < 0.05$, $^{**}p < 0.01$.

Table A.2: Standardized bias before and after matching

	Before	After	After IPW survival
family size	31.82	−4.04	−3.43
birth rank	−13.25	1.91	1.76
region			
South	−2.68	0.54	0.55
North	0.58	−0.41	−0.42
East	3.91	−0.25	−0.15
father's occupation			
white collar	−13.64	−0.07	−0.31
farm owner	−4.57	0.45	0.46
skilled	−8.29	−0.21	−0.22
unskilled	−3.96	0.38	0.45
unknown	40.66	−1.89	−1.31
IQ			
1 (highest)	−20.78	−0.22	−0.42
2	−10.41	−0.01	−0.08
4	−4.64	0.00	0.11
5	−2.28	−0.55	−0.32
6 (lowest)	9.58	−0.78	−0.68
9 (missing)	62.80	2.78	3.38
religion			
Dutch reformed	5.27	−0.63	−0.61
Calvinistic	−5.23	0.41	0.29
Other	22.02	1.72	1.76
None	−5.62	−0.82	−0.69
famine exposure in West			
Pre-conception	3.95	−0.05	−0.09
1^{st} trimester	−4.70	−0.80	−0.82
2^{nd} trimester	−8.95	0.29	0.39
3^{rd} trimester	−3.95	−0.37	−0.31
Post-natal	−2.26	−0.33	−0.23
famine cities	−1.27	−0.05	−0.07
famine exposure periods			
Pre-conception	3.21	0.06	0.02
1^{st} trimester	−4.90	−0.58	−0.58
2^{nd} trimester	−9.62	0.18	0.28
3^{rd} trimester	−3.85	−0.61	−0.55
Post-natal	−4.30	−0.26	−0.10
trend	12.99	1.04	0.80
trend-squared	12.18	1.15	0.94
partner	−20.20	−8.10	−8.33

Table A.3: Impact of early poor general health on home care probability, 2004-2013, linear probability model with IPW, without accounting for selective attrition

	Total effect	Direct effect		Indirect effect	
		$\theta(1)$	$\theta(0)$	$\eta(0)$	$\eta(1)$
	Household care				
2004	0.48%**	0.40%*	0.33%*	0.08%	0.15%**
2005	0.47%**	0.33%	0.31%*	0.14%	0.16%**
2006	0.57%**	0.40%+	0.40%*	0.17%*	0.17%**
2007	0.56%**	0.36%	0.37%*	0.20%*	0.19%**
2008	0.44%*	0.11%	0.25%	0.33%*	0.19%**
2009	0.59%**	0.32%	0.36%+	0.27%*	0.23%**
2010	0.75%**	0.43%	0.48%*	0.32%**	0.27%**
2011	0.85%**	0.37%	0.55%*	0.48%**	0.30%**
2012	0.80%**	0.24%	0.52%*	0.56%**	0.28%**
2013	0.97%**	0.39%	0.71%**	0.58%**	0.26%**
	Personal care				
2004	0.20%	0.05%	0.05%	0.15%	0.15%**
2005	0.22%	0.11%	0.10%	0.11%	0.12%**
2006	−0.07%	−0.13%	−0.13%	0.06%	0.06%**
2007	0.17%	0.11%	0.11%	0.06%	0.06%**
2008	0.32%	−0.09%	−0.09%	0.41%*	0.41%**
2009	−0.05%	−0.52%+	−0.58%	0.47%*	0.53%**
2010	0.32%	0.18%	0.17%	0.14%	0.15%**
2011	0.57%*	0.49%+	0.45%*	0.08%	0.12%**
2012	0.18%	−0.11%	−0.12%	0.29%	0.30%**
2013	0.33%	−0.21%	−0.22%	0.54%**	0.55%**
	Nursing care				
2004	0.05%	−0.01%	−0.03%	0.06%	0.08%**
2005	0.10%	−0.02%	0.04%	0.12%	0.06%**
2006	0.08%	0.05%	0.03%	0.03%	0.05%*
2007	0.15%	−0.10%	0.10%	0.25%	0.05%+
2008	0.01%	−0.09%	−0.07%	0.10%	0.08%**
2009	−0.04%	−0.16%	−0.09%	0.12%	0.05%*
2010	0.24%	0.09%	0.20%	0.15%	0.04%
2011	0.18%	0.20%	0.13%	−0.02%	0.05%+
2012	0.46%*	0.49%*	0.39%+	−0.03%	0.07%+
2013	0.30%	0.11%	0.24%	0.19%	0.06%**
	Total care				
2004	0.37%+	0.21%	0.17%	0.16%	0.20%**
2005	0.50%*	0.21%	0.31%	0.29%*	0.19%**
2006	0.60%*	0.41%	0.39%+	0.21%	0.21%**
2007	0.74%**	0.32%	0.49%+	0.42%+	0.25%**
2008	0.60%*	0.00%	0.32%	0.60%**	0.28%**
2009	0.59%*	0.02%	0.30%	0.57%**	0.29%**
2010	0.84%**	0.38%	0.53%+	0.46%*	0.31%**
2011	1.01%**	0.56%	0.67%*	0.45%*	0.34%**
2012	1.13%**	0.50%	0.77%*	0.63%*	0.36%**
2013	1.10%**	0.26%	0.74%*	0.84%**	0.36%**

Included exogenous covariates in the estimation of the propensity score: birth order, family size, father's occupation, IQ scores, religion, quadratic trend of the birth date, famine exposure and region of birth. $^{+}p < 0.10, ^{*}p < 0.05, ^{**}p < 0.01$.

Table A.4: Sensitivity analysis characterizing 'killer' confounders: outcome, selection and mediator effects, 2013

	Household care			Personal care		
	ω	ξ	ψ	ω	ξ	ψ
$d = 0.1$ & $s = 0.1$	0.406**	0.020	−0.026	0.407**	0.015	−0.014
$d = 0.1$ & $s = 0.2$	0.805**	0.026	−0.352	0.807**	0.018	−0.028
$d = 0.1$ & $s = 0.3$	1.235**	0.031	−0.077	1.229**	0.020	−0.039
$d = 0.1$ & $s = 0.4$	1.711**	0.037	−0.103+	1.707**	0.021	−0.053
$d = 0.1$ & $s = 0.5$	2.305**	0.040	−0.129**	2.301**	0.021	−0.066
$d = 0.2$ & $s = 0.1$	0.400**	0.032	−0.026	0.401**	0.025	−0.014
$d = 0.2$ & $s = 0.2$	0.799**	0.037	−0.052	0.802**	0.028	−0.028
$d = 0.2$ & $s = 0.3$	1.226**	0.043	−0.077	1.222**	0.030	−0.039
$d = 0.2$ & $s = 0.4$	1.702**	0.048	−0.102+	1.698**	0.031	−0.052
$d = 0.2$ & $s = 0.5$	2.292**	0.048	−0.129**	2.289**	0.027	−0.066
$d = 0.3$ & $s = 0.1$	0.393**	0.043	−0.025	0.395**	0.035	−0.014
$d = 0.3$ & $s = 0.2$	0.791**	0.049	−0.051	0.796**	0.038	−0.027
$d = 0.3$ & $s = 0.3$	1.218**	0.054	−0.076	1.216**	0.040	−0.039
$d = 0.3$ & $s = 0.4$	1.692**	0.054	−0.102+	1.690**	0.037	−0.052
$d = 0.3$ & $s = 0.5$	2.279**	0.048	−0.129**	2.276**	0.028	−0.066
$d = 0.4$ & $s = 0.1$	0.387**	0.054	−0.025	0.389**	0.045	−0.013
$d = 0.4$ & $s = 0.2$	0.783**	0.060	−0.051	0.790**	0.048	−0.027
$d = 0.4$ & $s = 0.3$	1.210**	0.060	−0.076	1.208**	0.045	−0.039
$d = 0.4$ & $s = 0.4$	1.682**	0.055	−0.101+	1.680**	0.037	−0.052
$d = 0.4$ & $s = 0.5$	2.265**	0.048	−0.128**	2.266**	0.028	−0.066
$d = 0.5$ & $s = 0.1$	0.379**	0.065	−0.024	0.381**	0.055	−0.013
$d = 0.5$ & $s = 0.2$	0.777**	0.064	−0.050	0.783**	0.051	−0.027
$d = 0.5$ & $s = 0.3$	1.204**	0.060	−0.075	1.201**	0.045	−0.039
$d = 0.5$ & $s = 0.4$	1.673**	0.055	−0.101+	1.673**	0.037	−0.052
$d = 0.5$ & $s = 0.5$	2.553**	0.048	−0.128**	2.257**	0.028	−0.065

Based on adding U to propensity score under the assumption that $\Pr(U = 1) = 0.4$ and $p_{01} - p_{00} = 0$, the differences $d = p_{11} - p_{10}$ and $s = p_{1.} - p_{0.}$. No effect would give $\omega = 0, \xi = 0$ and $\psi = 0$. $^{+}p < 0.05$ and $^{**}p < 0.01$

Table A.4: Sensitivity analysis characterizing 'killer' confounders: outcome, selection and mediator effects, 2013 (continued)

	Nursing care			Total care		
	ω	ξ	ψ	ω	ξ	ψ
$d = 0.1$ & $s = 0.1$	0.400^{**}	0.013	-0.007	0.402^{**}	0.029	-0.030
$d = 0.1$ & $s = 0.2$	0.804^{**}	0.016	-0.014	0.802^{**}	0.036	-0.059
$d = 0.1$ & $s = 0.3$	1.227^{**}	0.018	-0.018	1.225^{**}	0.041	-0.087
$d = 0.1$ & $s = 0.4$	1.694^{**}	0.020	-0.024	1.700^{**}	0.047	-0.117^{+}
$d = 0.1$ & $s = 0.5$	2.292^{**}	0.020	-0.031	2.289^{**}	0.051	-0.147^{**}
$d = 0.2$ & $s = 0.1$	0.394^{**}	0.020	-0.007	0.391^{**}	0.049	-0.029
$d = 0.2$ & $s = 0.2$	0.799^{**}	0.023	-0.014	0.791^{**}	0.055	-0.059
$d = 0.2$ & $s = 0.3$	1.221^{**}	0.026	-0.018	1.213^{**}	0.062	-0.087
$d = 0.2$ & $s = 0.4$	1.688^{**}	0.027	-0.024	1.684^{**}	0.067	-0.116^{+}
$d = 0.2$ & $s = 0.5$	2.281^{**}	0.025	-0.031	2.269^{**}	0.065	-0.146^{**}
$d = 0.3$ & $s = 0.1$	0.390^{**}	0.027	-0.006	0.380^{**}	0.068	-0.028
$d = 0.3$ & $s = 0.2$	0.794^{**}	0.031	-0.014	0.780^{**}	0.075	-0.058
$d = 0.3$ & $s = 0.3$	1.215^{**}	0.033	-0.018	1.201^{**}	0.081	-0.086
$d = 0.3$ & $s = 0.4$	1.682^{**}	0.031	-0.024	1.671^{**}	0.079	-0.115^{+}
$d = 0.3$ & $s = 0.5$	2.272^{**}	0.025	-0.031	2.249^{**}	0.065	-0.145^{**}
$d = 0.4$ & $s = 0.1$	0.386^{**}	0.035	-0.006	0.369^{**}	0.088	-0.027
$d = 0.4$ & $s = 0.2$	0.789^{**}	0.038	-0.014	0.770^{**}	0.095	-0.057
$d = 0.4$ & $s = 0.3$	1.210^{**}	0.036	-0.018	1.189^{**}	0.091	-0.085
$d = 0.4$ & $s = 0.4$	1.676^{**}	0.031	-0.024	1.656^{**}	0.079	-0.114^{+}
$d = 0.4$ & $s = 0.5$	2.263^{**}	0.025	-0.031	2.228^{**}	0.066	-0.145^{**}
$d = 0.5$ & $s = 0.1$	0.380^{**}	0.042	-0.006	0.358^{**}	0.108^{+}	-0.026
$d = 0.5$ & $s = 0.2$	0.784^{**}	0.040	-0.014	0.758^{**}	0.103	-0.056
$d = 0.5$ & $s = 0.3$	1.203^{**}	0.036	-0.018	1.176^{**}	0.092	-0.084
$d = 0.5$ & $s = 0.4$	1.668^{**}	0.031	-0.024	1.640^{**}	0.080	-0.113^{+}
$d = 0.5$ & $s = 0.5$	2.255^{**}	0.025	-0.030	2.209^{**}	0.066	-0.144^{**}

Based on adding U to propensity score under the assumption that $\Pr(U = 1) = 0.4$ and $p_{01} - p_{00} = 0$, the differences $d = p_{11} - p_{10}$ and $s = p_{1.} - p_{0.}$. No effect would give $\omega = 0, \xi = 0$ and $\psi = 0$. $^{+}p < 0.05$ and $^{**}p < 0.01$

Table A.5: Sensitivity analysis characterizing 'killer' confounders (mediator), difference with original estimates 2013, *Household care*

	Total effect	Direct effect		Indirect effect	
		$\theta(1)$	$\theta(0)$	$\eta(0)$	$\eta(1)$
original	1.80%**	0.85%	1.29%**	0.95%**	0.52%**
$d = 0.1$ & $s = 0.1$	−0.04%	−0.05%	−0.04%	0.00%	−0.01%
$d = 0.1$ & $s = 0.2$	−0.05%	−0.06%	−0.04%	0.01%	−0.01%
$d = 0.1$ & $s = 0.3$	−0.06%	−0.07%	−0.05%	0.02%	−0.02%
$d = 0.1$ & $s = 0.4$	−0.06%	−0.09%	−0.06%	0.03%	−0.02%
$d = 0.1$ & $s = 0.5$	−0.07%	−0.12%	−0.06%	0.04%	−0.02%
$d = 0.2$ & $s = 0.1$	−0.05%	−0.06%	−0.05%	0.00%	−0.01%
$d = 0.2$ & $s = 0.2$	−0.06%	−0.07%	−0.05%	0.01%	−0.02%
$d = 0.2$ & $s = 0.3$	−0.07%	−0.09%	−0.06%	0.02%	−0.02%
$d = 0.2$ & $s = 0.4$	−0.07%	−0.11%	−0.06%	0.04%	−0.02%
$d = 0.2$ & $s = 0.5$	−0.08%	−0.13%	−0.07%	0.05%	−0.01%
$d = 0.3$ & $s = 0.1$	−0.07%	−0.07%	−0.06%	0.01%	−0.02%
$d = 0.3$ & $s = 0.2$	−0.07%	−0.09%	−0.06%	0.02%	−0.02%
$d = 0.3$ & $s = 0.3$	−0.08%	−0.11%	−0.07%	0.03%	−0.02%
$d = 0.3$ & $s = 0.4$	−0.08%	−0.12%	−0.07%	0.04%	−0.02%
$d = 0.3$ & $s = 0.5$	−0.08%	−0.13%	−0.07%	0.05%	−0.01%
$d = 0.4$ & $s = 0.1$	−0.09%	−0.09%	−0.08%	0.01%	−0.02%
$d = 0.4$ & $s = 0.2$	−0.09%	−0.11%	−0.08%	0.02%	−0.02%
$d = 0.4$ & $s = 0.3$	−0.09%	−0.12%	−0.08%	0.03%	−0.02%
$d = 0.4$ & $s = 0.4$	−0.08%	−0.12%	−0.07%	0.04%	−0.02%
$d = 0.4$ & $s = 0.5$	−0.08%	−0.13%	−0.07%	0.05%	−0.02%
$d = 0.5$ & $s = 0.1$	−0.11%	−0.12%	−0.09%	0.01%	−0.02%
$d = 0.5$ & $s = 0.2$	−0.10%	−0.12%	−0.09%	0.02%	−0.02%
$d = 0.5$ & $s = 0.3$	−0.09%	−0.12%	−0.08%	0.03%	−0.02%
$d = 0.5$ & $s = 0.4$	−0.08%	−0.12%	−0.07%	0.04%	−0.02%
$d = 0.5$ & $s = 0.5$	−0.08%	−0.13%	−0.07%	0.05%	−0.02%

Based on adding U to propensity score under the assumption that $\Pr(U = 1) = 0.4$ and $p_{01} - p_{00} = 0$, the differences $d = p_{11} - p_{10}$ and $s = p_{1.} - p_{0.}$. $^{+}p < 0.05$ and $^{**}p < 0.01$.

Table A.6: Sensitivity analysis characterizing 'killer' confounders (mediator), difference with original estimates 2013, *Personal care*

	Total effect	Direct effect		Indirect effect	
		$\theta(1)$	$\theta(0)$	$\eta(0)$	$\eta(1)$
original	1.51%*	0.72%	1.08%	0.79%**	0.46%**
$d = 0.1$ & $s = 0.1$	−0.05%	−0.03%	−0.07%	−0.02%	−0.01%
$d = 0.1$ & $s = 0.2$	0.01%	0.01%	−0.02%	−0.01%	0.00%
$d = 0.1$ & $s = 0.3$	0.07%	0.05%	0.03%	0.01%	0.01%
$d = 0.1$ & $s = 0.4$	0.14%	0.11%	0.09%	0.04%	0.03%
$d = 0.1$ & $s = 0.5$	0.22%	0.16%	0.15%	0.06%	0.04%
$d = 0.2$ & $s = 0.1$	−0.02%	0.00%	−0.04%	−0.02%	−0.01%
$d = 0.2$ & $s = 0.2$	0.06%	0.06%	0.02%	0.00%	0.00%
$d = 0.2$ & $s = 0.3$	0.14%	0.13%	0.10%	0.02%	0.02%
$d = 0.2$ & $s = 0.4$	0.24%	0.19%	0.17%	0.04%	0.04%
$d = 0.2$ & $s = 0.5$	0.29%	0.22%	0.21%	0.07%	0.05%
$d = 0.3$ & $s = 0.1$	0.01%	0.02%	−0.02%	−0.01%	−0.01%
$d = 0.3$ & $s = 0.2$	0.11%	0.11%	0.07%	0.00%	0.01%
$d = 0.3$ & $s = 0.3$	0.21%	0.19%	0.16%	0.03%	0.03%
$d = 0.3$ & $s = 0.4$	0.29%	0.24%	0.22%	0.05%	0.04%
$d = 0.3$ & $s = 0.5$	0.29%	0.22%	0.21%	0.07%	0.05%
$d = 0.4$ & $s = 0.1$	0.03%	0.05%	0.01%	−0.01%	0.00%
$d = 0.4$ & $s = 0.2$	0.16%	0.15%	0.11%	0.00%	0.01%
$d = 0.4$ & $s = 0.3$	0.25%	0.22%	0.19%	0.03%	0.03%
$d = 0.4$ & $s = 0.4$	0.29%	0.24%	0.22%	0.05%	0.04%
$d = 0.4$ & $s = 0.5$	0.29%	0.22%	0.21%	0.07%	0.05%
$d = 0.5$ & $s = 0.1$	0.06%	0.07%	0.03%	−0.01%	0.00%
$d = 0.5$ & $s = 0.2$	0.17%	0.17%	0.13%	0.01%	0.02%
$d = 0.5$ & $s = 0.3$	0.25%	0.22%	0.19%	0.03%	0.03%
$d = 0.5$ & $s = 0.4$	0.29%	0.24%	0.22%	0.05%	0.04%
$d = 0.5$ & $s = 0.5$	0.29%	0.22%	0.21%	0.07%	0.05%

Based on adding U to propensity score under the assumption that $\Pr(U = 1) = 0.4$ and $p_{01} - p_{00} = 0$, the differences $d = p_{11} - p_{10}$ and $s = p_{1.} - p_{0.}$. $^{+}p < 0.05$ and $^{**}p < 0.01$.

Table A.7: Sensitivity analysis characterizing 'killer' confounders (mediator), difference with original estimates 2013 , *Nursing care*

	Total effect	Direct effect		Indirect effect	
		$\theta(1)$	$\theta(0)$	$\eta(0)$	$\eta(1)$
original	1.14%$^{+}$	0.80%	0.93%	0.34%	0.21%*
$d = 0.1$ & $s = 0.1$	−0.02%	0.02%	−0.01%	−0.04%	−0.01%
$d = 0.1$ & $s = 0.2$	0.05%	0.08%	0.05%	−0.03%	0.00%
$d = 0.1$ & $s = 0.3$	0.13%	0.15%	0.12%	−0.02%	0.01%
$d = 0.1$ & $s = 0.4$	0.23%	0.24%	0.20%	0.00%	0.03%
$d = 0.1$ & $s = 0.5$	0.34%	0.32%	0.29%	0.02%	0.05%
$d = 0.2$ & $s = 0.1$	0.02%	0.05%	0.02%	−0.03%	−0.01%
$d = 0.2$ & $s = 0.2$	0.10%	0.13%	0.10%	−0.03%	0.01%
$d = 0.2$ & $s = 0.3$	0.21%	0.22%	0.19%	−0.01%	0.02%
$d = 0.2$ & $s = 0.4$	0.34%	0.33%	0.30%	0.00%	0.04%
$d = 0.2$ & $s = 0.5$	0.42%	0.40%	0.36%	0.02%	0.05%
$d = 0.3$ & $s = 0.1$	0.05%	0.08%	0.05%	−0.03%	0.00%
$d = 0.3$ & $s = 0.2$	0.16%	0.18%	0.15%	−0.02%	0.01%
$d = 0.3$ & $s = 0.3$	0.30%	0.31%	0.27%	−0.01%	0.03%
$d = 0.3$ & $s = 0.4$	0.39%	0.39%	0.35%	0.00%	0.04%
$d = 0.3$ & $s = 0.5$	0.42%	0.40%	0.36%	0.02%	0.05%
$d = 0.4$ & $s = 0.1$	0.08%	0.12%	0.08%	−0.03%	0.00%
$d = 0.4$ & $s = 0.2$	0.22%	0.25%	0.21%	−0.02%	0.02%
$d = 0.4$ & $s = 0.3$	0.33%	0.34%	0.30%	−0.01%	0.03%
$d = 0.4$ & $s = 0.4$	0.39%	0.39%	0.35%	0.00%	0.04%
$d = 0.4$ & $s = 0.5$	0.42%	0.40%	0.36%	0.02%	0.05%
$d = 0.5$ & $s = 0.1$	0.12%	0.16%	0.12%	−0.03%	0.00%
$d = 0.5$ & $s = 0.2$	0.24%	0.26%	0.23%	−0.02%	0.02%
$d = 0.5$ & $s = 0.3$	0.33%	0.34%	0.30%	−0.01%	0.03%
$d = 0.5$ & $s = 0.4$	0.39%	0.39%	0.35%	0.00%	0.04%
$d = 0.5$ & $s = 0.5$	0.42%	0.40%	0.36%	0.02%	0.05%

Based on adding U to propensity score under the assumption that $\Pr(U = 1) = 0.4$ and $p_{01} - p_{00} = 0$, the differences $d = p_{11} - p_{10}$ and $s = p_{1.} - p_{0.}$. $^{+}p < 0.05$ and $^{**}p < 0.01$.