
Kröger H, Pakpahan E, Hoffmann R. [What causes health inequality? A systematic review on the relative importance of social causation and health selection](#). *European Journal of Public Health* 2015, 26(6), 951-960.

Copyright:

© The Author 2015. Published with open access by Oxford University Press on behalf of the European Public Health Association. All rights reserved.

DOI link to article:

<https://doi.org/10.1093/eurpub/ckv111>

Date deposited:

15/03/2018



This work is licensed under a [Creative Commons Attribution-NonCommercial 3.0 Unported License](#)

What causes health inequality? A systematic review on the relative importance of social causation and health selection

Hannes Kröger, Eduwin Pakpahan, Rasmus Hoffmann

Department of Political and Social Sciences, European University Institute, Via dei Roccettini, 9, 50014 San Domenico di Fiesole, Italy

Correspondence: Hannes Kröger, Department of Political and Social Sciences, Via dei Roccettini, 9, 50014, San Domenico di Fiesole, Italy, Tel: [+39] 055 4685 475, Fax: [+39] 055 4685 201, e-mail: hannes.kroger@eui.eu

Background: The social gradient in health is one of the most reliable findings in public health research. The two competing hypotheses that try to explain this gradient are known as the social causation and the health selection hypothesis. There is currently no synthesis of the results of studies that test both hypotheses. **Methods:** We provide a systematic review of the literature that has addressed both the health selection and social causation hypotheses between 1994 and 2013 using seven databases following PRISMA rules. **Results:** The search strategy resulted in 2952 studies, of which, we included 34 in the review. The synthesis of these studies suggests that there is no general preference for either of the hypotheses (12 studies for social causation, 10 for health selection). However, both a narrative synthesis as well as meta-regression results show that studies using indicators for socio-economic status (SES) that are closely related to the labor market find equal support for health selection and social causation, whereas indicators of SES like education and income yield results that are in favor of the social causation hypothesis. High standards in statistical modeling were associated with more support for health selection. **Conclusions:** The review highlights the fact that the causal mechanisms behind health inequalities are dependent on whether or not the dimension being analyzed closely reflects labor market success. Additionally, further research should strive to improve the statistical modeling of causality, as this might influence the conclusions drawn regarding the relative importance of health selection and social causation.

Introduction

The social gradient in health is one of the most reliable findings in public health research. There is a lot of evidence to support the hypothesis that health inequalities persist throughout time and across societies.¹ The implications of health inequalities are that people with a lower level of education, a lower occupational status or a lower level of income tend to live shorter lives and have a higher prevalence of disease. These inequalities in health have been found in all European countries with available data, and usually amount to between 5 and 10 years' difference in life expectancy, and between 10 and 20 years' difference in disability-free life expectancy.^{2,3} Several studies indicate that, in many countries in Europe and in the US, health inequalities are growing.^{4,5} Consequently, the question as to how these inequalities arise is more relevant than ever, and scholars continue to approach this problem using very diverse datasets, methods and research designs. Our intent is to provide a trans-disciplinary review of the literature of the last 20 years which will assess the relative importance of the health selection hypothesis vs. the social causation hypothesis.

The two competing hypotheses can be best described as follows⁶: 'health selection' is defined as the process by which differences in health status lead to differences in social position. Those who are in good health are able to achieve favorable positions in society; those in poor health have worse chances and will only achieve low-status positions. The 'social causation' hypothesis claims that circumstances in higher socio-economic positions are more beneficial to health than in lower socio-economic positions. The social gradient in health is therefore created by differences in resources, support, knowledge, behavior or other factors that are socially stratified.

Sometimes the idea of 'indirect selection' is put forward to explain the socio-economic status (SES)-health gradient. This concept implies that both health and social class are simultaneously affected by third

factor variables such as education.⁷ For our purposes, we would not treat such findings as support for the health selection hypothesis. They would be categorized either as spurious correlation or, in the example of higher education leading to higher status and better health, as support for social causation. We are therefore only interested in direct health selection effects that reflect a direct causal link between health and SES.

The reason for not counting indirect selection as support for health selection is also rooted in our notion of causality. It is clear that most of the studies investigating the reciprocal relationship between SES and health cannot claim to test causality in the sense of 'potential outcomes'.⁸ Health and SES are not randomly assigned, and the research question usually only arises because they are entangled in a complex pattern. Although it is rarely discussed explicitly in the studies examined for this review, we think that if causal inference is drawn it should be understood as what Heckman called econometric causality.⁹ According to this concept, the aim is to model the treatment so that, given control variables, SES and health are quasi-randomized.

Apart from the definition of causality, there is another important aspect in the evaluation of these two hypotheses that needs to be taken into account. 'Studies can draw their conclusions at either the individual or the population level: In the first case, the only question is whether a certain measure of health influences the individual's SES more strongly than their status influences the health measure'. However, one could also ask whether the gradient observed in the population can be better explained by health selection or social causation, which can yield different results. Therefore, the question as to the studies' level of conclusion is discussed separately in the results section.

Taking these considerations into account, the review follows two goals:

- First, we investigate the relative importance of two causal mechanisms for the emergence of health inequalities.

Identifying the contribution of health selection and social causation to health inequalities is fundamental to the effectiveness of measures directed at the prevention or reduction of health inequalities.

- Second, we point out caveats in this field of research, and make suggestions for improving comparability between studies and furthering the causal interpretation of research results.

Previous reviews

Early reviews of health and SES literature provide an overview of contemporary health inequality research, and deal predominantly with social causation, while scarcely mentioning health selection.^{10–12} One more recent review addresses the relationship between unemployment and mental health, and includes estimates for both the social causation and the health selection theory.¹³ However, these estimates are not based on studies which test both directions of causality.

In contrast to previous reviews of the association between SES and health, we put the matter of causal direction at the center of our review, instead of analyzing a particular dimension of health or SES.¹⁴ We review studies that try to assess whether health selection, social causation, or both are at work. This new perspective gives us the opportunity to look for common results in different fields like health economics, epidemiology, medical sociology and medical psychology. We can synthesize the results to give the best empirical basis for a public health discussion of the roots of health inequality. At the same time, we point out strengths and weaknesses across disciplines to improve future research.

Methods

For our review, we followed the PRISMA guidelines for systematic reviews.¹⁵ A review protocol was developed and updates were documented if necessary. The protocol is documented in the online Appendix A.

Search strategy

We searched for journal articles in seven scientific databases using the groups of keywords documented in the research protocol.

Appendix A also provides the code for a complete Medline search. The databases used are Medline, PsycINFO, Scopus, Web of Knowledge, Cochrane Library, Econlit and Proquest Social Science Journals. The search was conducted on 5 November 2013. Our aim was to give an overview of the research from the last 20 years. This timeframe enables a comprehensive evaluation of the current state of affairs in this field of research. Additionally, we excluded earlier time periods, because the kind of large scale longitudinal datasets usually required to answer the question have only really become available in the last two decades.

In addition to searching the literature databases, we also checked the references of the studies thus identified for additional studies relevant to the review. At the same time, the databases were used to screen for articles that cite the identified studies. Through these two steps, we found 11 additional potential studies. Appendix B provides the bibliographic information of all search results derived from the databases and of all the studies identified via references and citations.

Criteria for inclusion

The following criteria had to be fulfilled for a study to be included into the review. (i) The study appears in the English language in a peer-reviewed journal published after 1 January, 1994. (ii) The study population is from an industrialized country, as defined by OECD membership status, to restrict the analysis to comparable settings with regard to the standard of living, the quality and availability of health care and relevant welfare state arrangements. (iii) The

study is original, hypothesis-testing statistical analyses. Comments and reviews are excluded. (iv) The data is on the individual level and not, for example, on the regional level, e.g. communities or counties. (v) The study investigates SES via one of the following dimensions: education, occupation, income and wealth, or labor market participation and success. (Labor market indicators are not classical indicators of SES, but they constitute the foundation for broader measures like household income. Therefore we think it useful to include labor market indicators in the analysis.) (vi) Only studies that measure SES objectively—as opposed to subjectively—are included, because subjective measures are less common, less comparable and entail different causal associations with health than objective measures. All measures of mental and physical health are considered health outcomes, including both objective and subjective measures. Measures for quality of life, life satisfaction or happiness are not included, unless they are health-related quality-of-life measures. (vii) The most important and most restrictive criterion for inclusion is that the study conducts direct tests of the two competing hypotheses. Testing one hypothesis, and thus merely inferring the presence of the other causal mechanism by absence of the former, is not sufficient. Appendix C includes several examples of studies that we excluded due to one or more of the seven criteria.

One major concern that could be raised against a review on studies that test both directions of causality in the SES-health gradient is that it will mainly include so called model-based inference studies, and very few or no design-based studies.¹⁶ In model-based studies, quasi-randomization is generated through correct statistical modeling of all confounding factors, thus establishing causal inference. It is sometimes claimed, however, that design-based studies might provide stronger evidence of causal relationships than model-based studies.¹⁷ We include only studies that test both directions of causality (which consequently will be less likely to display strong exogenous variation for both health and SES) because our aim is to review studies that—in principle—allow a direct comparison of the health selection and social causation effect ‘for the same population’. A review focusing on design-based studies would make conclusions regarding the relative importance of HS and SC very difficult, because the reference populations would vary widely between studies. The relation of the populations’ characteristics to the relative strength of the estimated causal impact of health selection or social causation would remain unclear. Due to this important limitation of design-based studies, we think that model-based studies should be seen as complementary, and not alternative, to design-based studies.

Selection of studies

After combining all search results from the databases, and eliminating duplicates, the first reviewer screened the title and abstract of each study, and excluded all thematically irrelevant studies. The same reviewer assessed the full text of the remaining articles according to the inclusion criteria. The second reviewer double-checked the inclusion choices by reviewing the abstracts. Disagreements were settled in discussions with all three reviewers.

Data extraction

From the included articles, the first reviewer collected data about the study design and items, following the guidelines suggested by the STROBE Statement and similar reviews.^{18–20} In addition, the main conclusions regarding health selection and social causation mechanisms are reported. The information was stored in a spreadsheet (Appendix D) and checked by the second reviewer. A reduced form of the data can be found in table 2.

Quality of the studies

The reviewed studies raised five problems that arise in any attempt to model the relationship between SES and health in such a way that SES and health can be seen as quasi-randomized, fulfilling the

necessary condition for causality as defined above. As the review deals with causality, the question as to whether a single study addressed these problems is the essential indicator for quality. The five problems are listed in table 1.

First, measurement error in SES and health should be accounted for. Even if objective measures are available, they will still be subject to random measurement error. Second, the issue of missing values and panel drop-outs is important. Completely excluding such cases would rely on the unrealistic assumption that missing values are distributed randomly. Missing values can lead to a bias of unknown direction in the estimates. Third, the estimated coefficients should be comparable. This means that a comparison of the numerical values of the coefficients representing health selection and social causation should statistically correspond to the relative importance of the two directions of causality. This is important for the easy interpretation of the relative importance of health selection and social causation. Fourth, controlling for external factors is important. There is little benefit in putting a lot of effort into modeling the dualistic relationship between SES and health if the association is actually caused by a third variable. The standard approach is the introduction of control variables in the equations. One specific way of addressing omitted variables which is often used in health economics is the introduction of a random or a fixed effect in a panel model. This allows the researcher to control for time-constant factors, but not for time-varying unobserved factors. Finally, the reciprocal causal relationship is best estimated in a system of simultaneous equations. In this way, the statistical model very closely reflects the theoretical model, and comparability of estimates between the two hypotheses is also given.

Based on the five categories of problems discussed earlier, we constructed a quality score and quality categories for the statistical modeling of causality. If only one or none of the five problems was addressed, the study was categorized as low quality. Studies which addressed two or three problems were coded as middle quality, and those addressing four or five were coded as high quality (column 'quality' in table 2). These quality groups should be understood as rough indicators of the plausibility of a causal interpretation of the presented results, according to definition of causality.

Data analysis

The search strategy we applied allows for many different measures of SES and health. Consequently, a meta-analysis on the effect size cannot be conducted. An informal synthesis of the evidence was therefore chosen. This narrative synthesis was complemented by a meta-regression of the conclusion of the studies (HS vs. SC) on the study characteristics and a sign-test of for different dimensions of SES separately. We use the sign-test to whether the social causation is as strong as health selection.

$$H_0 : \theta_{SC} = \theta_{HS},$$

$$H_1 : \theta_{SC} \neq \theta_{HS}$$

Under H_0 the results from the studies that we reviewed should give preference to health selection or social causation following a

binomial distribution [$p(\theta_{SC} > \theta_{HS}) = 0.5$]. If the probability that the distribution of results favoring health selection or social causation is <5% (two-tailed test), this is treated as significant support that one of the two is stronger than the other.

In the second part of the meta-analysis we run a multinomial logistic regression. The dependent variable took the categories from table 3 (study favored . . . k : 1 = SC, 2 = equal, 3 = HS). As predictors, we included the square root of the number of observations, three SES dimensions ($j = \{\text{Education, labor market, income}\}$) from table 3 as dummy variables (occupation had to be excluded from the model due to over determination of the results), the mean age of the sample, the quality score, the year of publication and a dummy for region (Europe vs. North America and Australia). We estimate marginal effects for the probability of being in one of the three categories:

$$P(y = k) = \alpha_k + \beta_{k1}\sqrt{Obs} + \beta_{k2j}SES_j + \beta_{k3}age + \beta_{k4}quality + \beta_{k5}year + \beta_{k6}Europe$$

Appendix E includes a short summary of every study under review. In our article, the studies are analyzed with reference to the following topics: the evidence for health selection and social causation, data and bias issues, different dimensions of health and SES indicators, and methodological considerations. Whether a study deems health selection or social causation more important was determined as follows. If standardized regression coefficients are reported, they are used for comparison. If they are not available, but one pathway has been found to have a (significant) effect and the other has not, then the first pathway is judged to be more important. If the authors instead conducted other model tests that supported one of the hypotheses over the other this result was noted. If this condition is not met, the authors' judgment is reported. Here, it should be noted again that some studies draw conclusions on the population level, while most draw conclusions on the individual level. In some cases, not enough information can be found to establish whether health selection or social causation is considered more important.

Results

Search results from the databases

The seven databases yielded 5485 search results. After duplicates were removed, this left 2952 unique search results. Of these unique search results, 2830 records were excluded via our title and abstract checks. From these 122 remaining articles, 94 were removed because they did not meet the eligibility criteria after a full text appraisal. From the 122 screened articles, eleven further references were identified as potential candidates for the review, of which six turned out suit the inclusion criteria. This left 34 articles in the actual review. The details of the search and exclusion process are presented in figure 1.

Table 1 Five problems for causal analysis and possible solutions found in the reviewed literature

Problem	Suggested solution
Random measurement error in SES and health	Measurement models for health or SES
Missing data in health, SES and control variables	Multiple imputation or full information maximum likelihood (FIML) estimation
Spurious correlation between SES and health	Controlling for third factor explanations through (a) control variables (beyond age and gender) (b) using methods to capture time-constant unobserved heterogeneity (random or fixed effects regression)
Comparability of estimates	Standardized regression coefficients
Modeling both directions of causality	Simultaneous equation models

Table 2 Summary of the main characteristics of studies under review

First author/ Year	Observations	Country	SES measures	Health measures	Age range	SEM	MIM	FERE	SDMV	CS	quality	Health selection	Social causation	Relative importance	Period	Response rate	Representative
Adams 2003 ⁶⁰	6489	US	Income, wealth, home owner	SRH, chronic illness, ADL, BMI, mental health, acute illnesses	70–90+	no	no	no	no	yes	Middle	yes	yes	ND	1993–1998	79.8	Nationally
Aittomaki 2012 ⁴²	211639	FI	Unemployment, wages, income, wealth	Sickness leave	17–66	yes	yes	no	yes	no	High	yes	yes	SC	1993–2006	100	Nationally
Cai 2006 ⁴⁷	9000	AUS	Employment	SRH	15–64	yes	no	no	no	yes	High	yes	yes	HS	2001	66	Nationally
Cai 2009 ⁴⁸	2242	AUS	Wages	SRH	25–64	yes	no	no	yes	yes	Middle	yes	yes	HS	2001	66	Nationally
Cai 2009 ²²	2891	AUS	Employment	SRH	45–64	yes	no	no	yes	yes	Middle	yes	no	HS	2001–2004	66	Nationally
Cai 2010 ⁴⁶	4669	AUS	Employment	SRH	25–64	yes	no	yes	yes	yes	High	yes	yes	HS	2001–2004	66	Nationally
Case 2005 ³⁹	5439	UK	Occupation, income, education	SRH, birth-weight, chronic illnesses, pre- natal influences	7–42	no	no	no	no	yes	Low	yes	yes	ND	1958–2000	98.7	Nationally
Chandola 2003 ²³	10308	UK	Employment grade, financial deprivation	Physical/Mental health	35–67	yes	yes	no	yes	yes	High	no	yes	SC	1985–1999	73	Civil servants
Eaton 2001 ²⁷	907	US	Labor income, HH- income, job percentiles, social benefits, other income	Depression	16–64	no	no	no	yes	yes	Middle	no	no	none	1980–1996	82	City
Elovaino 2011 ³⁴	8312	UK	Occupation, promotion	Biomarkers, cardio- metabolic problems	41–74	no	no	no	no	yes	Low	yes	yes	SC	1991–2004	73	Civil servants
Elovaino 2012 ⁴³	3596	FI	Occupation, income	Mental health	30–45	yes	yes	yes	no	no	High	yes	yes	HS	1980–2007	83	Nationally
Elstad 2003 ³⁷	9189	NO	Occupational group	SRH	25–59	no	no	no	no	no	Low	yes	yes	HS	1984–1997	88	County
Haan 2009 ⁴⁹	4420	DE	Employment	SRH	30–59	yes	no	yes	yes	yes	High	yes	yes	HS	1996–2007	60.4	Nationally
Haas 2006 ³²	2805	US	Education, occupation, wages, wealth	SRH, birth-weight	16–64	yes	no	yes	no	no	Middle	yes	yes	equal	1968–2001	76	Nationally
Hallerod 2011 ⁴¹	2976	SE	Occupational prestige, HH- income	Physical limita- tions, diseases	31–63	yes	yes	yes	yes	no	High	yes	yes	equal	1979–1998	60.4	Nationally
Hamilton 1997 ⁶¹	447	CA	Employment	Mental health	20–49	yes	no	no	no	yes	Middle	yes	yes	ND	1985–1987		City
Heponiemi 2007 ⁵⁰	78195	FI	Unemployment	Mental health, physical symptoms, digestive system	20–45	no	no	no	no	yes	Middle	yes	yes	HS	1990–2002	100	Health care professionals

(continued)

Table 2 Continued

First author/ year	Observations	Country	SES measures	Health measures	Age range	SEM	MM	FERE	SDMV	CS	quality	Health selection	Social causation	Relative importance	Period	Response rate	Representative
Huurre 2005 ³³	1262	FI	Occupation, education	Psychosomatic distress	16–32	yes	yes	no	yes	no	Middle	yes	yes	HS	1983–1999	96.7	City
Michaud 2008 ²¹	3386	US	Wealth	SRH, chronic illness, limita- tions (ADL), BMI, mental health	51–61	yes	no	yes	no	yes	Middle	yes	no	HS	1992–2002	81.6	Nationally
Miech 1999 ³⁰	939	US	Occupation, education	Mental health	15–21	no	no	no	no	yes	Middle	yes	yes	equal	1987–1994	73.8	City
Mirowsky 1995 ⁴⁴	2436	US	Employment	SRH, physical functioning	20–64	yes	yes	no	yes	yes	Low	yes	yes	SC	1977–1982		Nationally
Mulatu 2002 ²⁸	705	US	HH-income, education, occupation	Physical limita- tions, accident or injury, distress, sleep	41–88	yes	no	no	no	yes	High	yes	yes	SC	1974–1995	76	Nationally
Olesen 2013 ⁵⁷	7176	AUS	Unemployment	Mental health	20–55	no	no	no	no	yes	Middle	yes	yes	ND	2001–2009	66	Nationally
Palloni 2009 ²⁹	1858	UK	Occupation, education	SRH, birth-weight	7–42	no	no	no	no	no	Low	yes	yes	SC	1958–2000	98.7	Nationally
Power 1996 ³⁵	17414	GB	Occupational group	SRH	7–33	no	no	no	no	yes	Low	yes	yes	SC	1974–1991	98.7	Nationally
power2002 ³⁶	5340	UK	Unemployment, financial hardship, redundancies, occupational class	Psychological distress	23–33	no	no	no	no	no	Middle	yes	yes	SC	1965–1991	98.7	Nationally
Ribet 2003 ³⁸	4715	FR	Occupation	Drinking, smoking, arterial hyper- tension, overweight	43–53	no	no	no	no	no	Low	yes	yes	equal	1985–1999	44.6	Company
Ritsher 2001 ²⁵	175	US	Occupation, education	Mental health	18–49	no	no	no	no	yes	Low	no	yes	SC	1977–1994	80	Community
Smith 2004 ⁵⁸		US	Wealth, income, education, employment tenure	Chronic illnesses	51–61	yes	no	no	no	yes	Middle	yes	yes	ND	1992–1997	81.6	Nationally
Stansfeld 2011 ⁴⁰	9377	GB	Occ. Classes, tenure	Depression, psy- chological distress	7–42	no	no	no	no	no	Low	yes	yes	equal	1958–2003	98.7	Nationally
Steele 2013 ⁴⁵	8784	UK	Employment, unemployment	Mental health (GHQ)	16–64	yes	no	yes	no	yes	Middle	yes	yes	SC	1991–2009	74	Nationally
Van de Mheen 1998 ²⁴	2800	NL	Education, occupation, EGP	SRH, mortality, physical symptoms, chronic illnesses	15–59	no	no	no	yes	no	Low	no	yes	SC	1991–1995	70.1	County

(continued)

Table 2 Continued

First author/ year	Observations	Country	SES measures	Health measures	Age range	SEM	MM	FERE	SDMV	CS	quality	Health selection	Social causation	Relative importance	Period	Response rate	Representative
Van der Lucht 1995 ³¹	502	NL	Occupation, education	SRH, school absence, doctors diagnoses	10–11	no	no	no	no	yes	Middle	yes	yes	equal	1989–1990	44	City
Warren 2009 ²⁶	2394	US	Education, occu- pational education	SRH, symptoms, sickness absence	18–65	yes	yes	no	yes	no	High	no	yes	SC	1957–2004	87.1	State

Note: SRH, self-rated health; HS, health Selection; SC, Social Causation; Relative Importance favours; ND, not determined; A, acknowledged; SEM, simultaneous equation modeling; MM, measurement model; FERE, fixed or random-effects; FERE, fixed or random-effects; SD, standardized coefficients; MV, missing values; CS, controls for spurious correlation.

The reciprocal causal relationship between health and SES

For each study, we assessed whether the authors found support for health selection, social causation, both, or neither, and whether either of the two was found to be more important. There are two studies^{21,22} that find support for health selection, but not social causation, and four that find the opposite pattern.^{23–26} One study does not find support for either of the hypotheses.²⁷ The other 27 studies found support for both directions of causality. Twelve studies conclude that social causation is more important than health selection, 10 studies come to the opposite conclusion, and six studies conclude that both hypotheses were equally strong.

From this broad overview, it is impossible to conclude that one causal direction has been established more frequently than the other. Therefore, we will first assess whether the findings differ by the dimensions of SES, and then determine whether they differ by the dimension of health.

Different dimensions of SES and health indicators

There are 11 studies that use education as an indicator of SES. Of these 11 studies, five find stronger support for social causation,^{24–26,28,29} three find equal support,^{30–32} and only one finds stronger support for health selection.³³ Two studies do not assess which of the two is more important.

Occupational status or class are the most frequently used indicators for SES. Eight studies claim that social causation is stronger.^{24–26,28,29,34–36} Two studies give preference to the health selection explanation,^{33,37} and seven studies attribute equal weight to social causation and health selection.^{30–32,38–41}

For household income (and related measures), we found three studies that thought social causation more important,^{23,28,42} one study which gave preference to health selection,⁴³ and three studies which attributed equal importance.^{27,32,41}

For each of these three classical dimensions of SES, the majority of studies judged social causation to be more important than health selection, although several studies reached a conclusion of equal importance.

However, there were several studies that did not use these classical, broad indicators of SES, but instead applied more specific, labor market-related measures like wages, employment status or employment grade. Of these studies, five indicate that social causation is more important.^{23,34,44,45} Six studies find stronger support for health selection^{22,46–50} and two studies find equal support.^{27,32}

When we compare these findings, we see that health selection and social causation are found to be equally important in studies relying on labor market-related indicators of SES. In contrast, studies using

Table 3 Studies favoring health selection or social causation—results by dimension of SES

SES-dimension	Study finds more support for			p($\theta_{SC}=\theta_{HS}$)
	Social causation	Health selection	Equal support	
All	12	10	6	0.851
Education	11	1	3	0.013
Occupation/occupational class	8	2	7	0.210
Household income	3	1	3	0.688
Labor market indicators	5	6	2	1.000

Note: The P-value is calculated under the hypothesis $H_0: \theta_{SC} = \theta_{HS}$ with $p(\theta_{SC} > \theta_{HS}) = 0.5$. Studies reporting equal support are equally divided among SC and HS. For unequal numbers of equal support studies, one study is not assigned to either SC or HS.

Part 1: Literature search January 1994 - November 2013

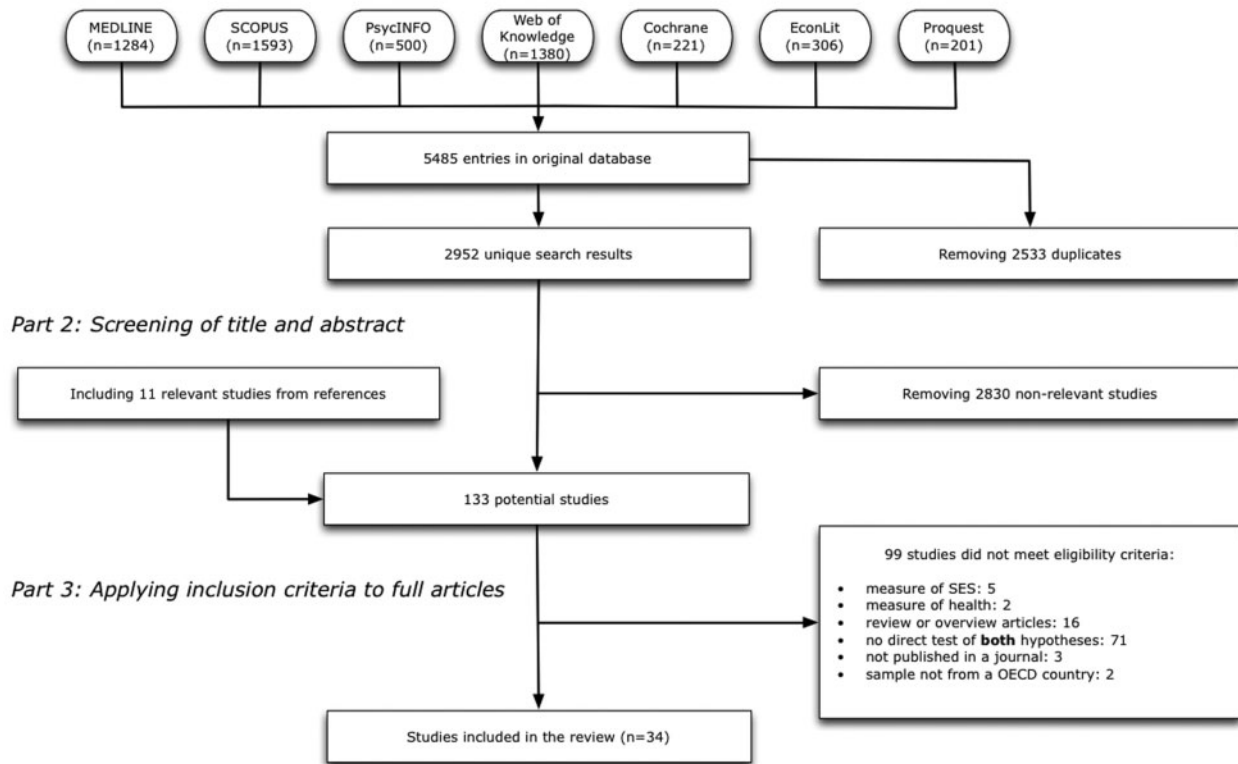


Figure 1 Caption: flow diagram of the process of including reviewed studies

broad indicators of social status tend to favor social causation. Table 3 gives an overview of the findings stratified by dimension of SES.

Similar differences for different measures of health could not be found in the reviewed studies. The choice of health measure (categorized into: chronic illnesses, mental health, functional limitations or self-rated health) is not associated with conclusions in favor of one of the two hypotheses. The category of functional limitations covers standard indicators like activities of daily living (ADL), self-reports of handicaps and weaknesses, and other physical conditions that limit activities. Self-rated health is, in all studies, a variant of the commonly used scale to report subjective health (e.g. poor to very good health). There are too few studies, and too many different health measures, to make a statement about a potential influence of the health measure on the decision between the two causal hypotheses.

Level of conclusion

One interesting finding from our review shows that there is no agreement in the literature regarding the level at which to draw conclusions about the relative importance of causation vs. selection. One type of study (26 studies in total) focuses exclusively on the individual level. In this case, researchers only compare the relative impact of health and SES on the individual's life. They try to determine whether health is more important for SES, or whether SES has a stronger impact on health. The second approach that we identified in five of the studies we reviewed^{29,35,37,51,52} looks at the explanatory power of health selection and social causation for health inequalities on the population level. Three other studies explicitly acknowledge this level of conclusion, but do not provide their own contribution based on their analyses.^{32,33,44}

The interpretation of the results with regard to population-level health inequalities is neither superior nor inferior to the individual-level interpretation. It can nevertheless yield very different results, which should be seen as complementing the conclusion on the

individual level. Both ways of drawing conclusions answer different research questions under a common thematic framework.

It is interesting to note that, of the five studies providing conclusions on the population level, one gives some slight preference to health selection,³⁷ while the other four studies give strong preference for social causation.^{29,35,51,52} At this level of conclusion, the overall results seem to be strongly in favor of social causation. However, this should not be over-interpreted, as it represents a minority of studies, and we have no way of establishing similar conclusions for the other studies.

Meta-analysis

In addition to the narrative synthesis, we conducted two types of meta-analysis. For the results grouped by the different dimensions of SES, we conducted a sign test. The null hypothesis here is that health selection and social causation are equally strong. Those studies that reported equal support were divided equally into the SC and HS groups for the calculation of the *P* values in table 3. If the number of these studies was uneven, one study was not assigned. The *P*-values show that only for education can we say that the number of studies supporting social causation is significantly higher ($P=0.013$). The overall difference in the number of studies giving more support to social causation than health selection, or vice versa, is not statistically significant.

The results of the meta-regression are reported in table 4, presented as marginal effects. The numbers can be read as the change in probability of being in any of the three categories, reported in percentage points. We did not report odds-ratios, because the prevalence of health selection and social causation group are so high that they lose their interpretative power as relative risks.

The sample size and the average age in the sample are not related to the results presented here. A regional difference between Europe and North America and Australia could not be established.

Table 4 Results from the meta-regression of preference of HS, SC or equal support on study characteristics

Study characteristic	Dependent variable: study finds more support for		
	Social causation	Equal support	Health selection
Number of observations (square root)	0.1 [−0.2;0.4]	0.1 [−0.1;0.3]	−0.2 [−0.6;0.2]
Year of publication	2.9 [−0.1;6.0]	−2.2 [−4.9;0.5]	−0.7 [−3.5;2.0]
Education	39.3 [−13.8;92.4]	−58.2 [−123.5;0.7]	18.9 [−32.2;70.0]
Income	40.5 [−19.1;100.1]	−90.7 [−162.1;−19.3]	50.2 [−5.3;105.8]
Labor market indicator	14.9 [−37.1;66.9]	−32.8 [−87.1;21.5]	18.0 [−31.1;67.0]
Europe (ref. North America and Australia)	−7.9 [−50.6;34.8]	3.1 [−32.9;39.2]	4.8 [−33.3;43.0]
Mean age of the sample	0.8 [−1.0;2.7]	0.2 [−2.3;1.9]	0.6 [−2.0;0.7]

Note: Marginal effects reported in percentage points. 95% Confidence interval reported in brackets.

The publication year shows some support for a preference for social causation in more recent publications (significant on the 10%-level). For the dimensions of SES, we can see that income studies display a preference for health selection significantly less frequently. The same result is found for education (significant on the 10%-level). Surprisingly, labor market indicators also predict a reduced probability of reporting health selection as the dominant mechanism. The coefficient of the quality score indicates that higher quality studies more often find stronger support for health selection.

In sum, we can see that the different dimensions of SES seem to be the strongest predictor of the studies' conclusions. The effect of the quality-score could mean that, unless researchers consistently apply more advanced modeling techniques, there could be a tendency to underestimate the relative importance of health selection.

Discussion

After reviewing the literature, we can say that both health selection and social causation seem to play a role in generating health inequalities. There is no general consensus regarding the relative importance of health selection and social causation among studies that test both hypotheses. Most studies find support for both hypotheses, regardless of the type of health or SES measure used. In areas that are directly related to labor market activities—like wages, employment or promotions—health selection and social causation seem to play an equal role in explaining health inequalities, while social causation seems to be more significant for health inequalities measured by more general status differences like education, occupational group or household income. It should be mentioned that studies investigating education leaned most strongly toward social causation. One explanation from a life-course perspective would be that, in contrast to income or occupation, educational attainment usually concludes after a set period of time, and cannot be further influenced by health. This point is reached at a stage in the life course at which overall variation in health status is naturally lower than later in life, potentially leaving less room for health selection effects. It would therefore be wise to look at the different dimensions of SES separately if the relative importance of health selection and social causation is to be established.⁵³ This conclusion is strongly corroborated by the results from the meta-regression, which highlighted the importance of the SES dimensions.

One explanation for the link between health selection and labor market indicators is the influence of health on productivity or absence through sickness, which will lead employers to favor workers who are healthy and dismiss those who are least healthy.^{54,55} Complementary to the two competing hypotheses, the reviewed literature highlights the necessity of taking third factor explanations into account. Certain personality traits could be responsible for both a healthy lifestyle and high educational attainment or economic success. The relationship between certain SES dimensions and health could therefore be partly spurious.

In terms of methodology, further research should aim to harmonize not only measures of SES and health, but also statistical approaches, in order to guarantee the highest standards of causal inference. It is important that a harmonization of methodological approaches is combined with a diversification of data sources, which will reduce the relative number of studies using the same data source. For replication studies,⁵⁶ underutilized datasets should be employed which fulfill the requirements for answering the complex question of health selection vs. social causation: without making any claims to a complete or systematic list, we suggest the Survey of Health, Ageing, and Retirement in Europe (SHARE), The Heinz Nixdorf Recall Study, The Swiss Household Panel Study, Understanding Society, or the Japanese Study of Aging and Retirement (JSTAR) as possible candidates. These surveys represent countries for which only limited evidence exists in our review, and provide a longitudinal structure with a relatively large sample and high quality data. It becomes apparent from our review that scholars have put a stronger emphasis on careful causal modeling in recent years. This is an important, positive development, one that should be refined even further in future research. Some studies leave serious doubts as to whether their results should be given a causal interpretation. It is crucial to address problems of third factor explanations, measurement error, comparability of coefficients, missing values and simultaneous estimation of reciprocal causal paths. The fact that the quality score was a significant predictor of the conclusion of the studies (higher quality studies were more likely to be in favor of health selection) shows that high methodological standards are not merely a means to itself, but can also genuinely affect conclusions in this field.

Strengths and limitations of the study

When considering possible sources of bias, it should be noted that several datasets are used repeatedly for the analysis of the causal relationship between SES and health. HILDA^{22,46–48,57} and NCDS^{29,35,36,39,40} provide the data for five papers each. The Whitehall II study is the basis for two papers.^{23,34} The HRS and AHEAD study are used in three studies.^{21,58,59} This might give the false impression of valid results from different studies, although the data are actually the same and the results are not independent.

Regarding the limits for generalization of the reviewed studies due to their sampling design, there does not seem to be any systematic association between representativeness, response rate and results with regard to the question of causality. However, it should be noted that the countries and regions in which the studies were conducted are not randomly chosen (e.g. strong US focus) due to the availability of datasets. Therefore, further research needs to consider carefully whether the results from our study can be generalized to different contexts (other welfare state system, labor market regulation or health care provision).

We grouped the studies according to the degree to which they used statistical methods to make causal interpretation more feasible, specifically by addressing the five problems that were identified in the reviewed studies (tables 1 and 2, column 'quality'). Ten studies

fall into the low quality group, 15 into the middle category, and nine into high quality category. Most of the high quality studies were published within the last five years of the reviewed period (2009–2013). This shows a clear trend toward higher standards for causal modeling. Although we could not find any overall association between the quality of the study and its conclusion, the meta-regression showed that there is an association between quality and a higher preference for health selection when adjusting for other study characteristics. This emphasizes the importance of careful statistical modeling.

Modeling reciprocal causality through different variations of simultaneous regression equations has become a useful tool. Almost all of the most recent studies we reviewed applied this technique. A similar positive trend can be detected for the practice of accounting for measurement error through measurement models of health or SES. Only a minority of studies account for missing values in their data, and no positive trend can be identified. Standardization of the coefficients is less common; only seven studies use explicitly standardized coefficients. This is surprising, as we consider this an important tool for valid comparisons of the relative importance of health selection and social causation.

One particular concern we have with several studies is their neglect of third factor explanations. Control variables other than age and gender are lacking in many studies.^{23–27,33,34,37,38,40,41} If health has common correlates with, for example, cognitive skills, qualifications, family situation or personal characteristics like locus of control, any association might be spurious. More recent studies address third factor explanations more carefully.^{32,41,43,45,46,49}

With regard to publication bias, we think that our approach to choose only studies that test both directions of causality greatly reduces the risk of biased results. Usually, non-significant results are published less often. However, if two hypotheses are tested against each other, the chances of both of them being insignificant are smaller.

Implications for policy and research

The general question as to whether health selection or social causation is more important is of great significance for social policy, because different answers would call for different policy reactions: If SES causes societal health differences, policy actions should focus on income redistribution, education, employment and lifestyles. If health differences are causally prior to social differences, policy makers should keep in mind that this process of health-related selection is not a natural (that is, Darwinistic) law. For example, regulations that allow more flexibility in working hours during periods of illnesses, or that protect persons with chronic illnesses, can alter health selection processes on the labor market and contribute to an overall reduction in health inequalities.

This review shows a slight preference for social causation as the explanation for health inequalities where education and income are the social variables, but health selection is also important, especially when selecting into labor market outcomes. The investigation of health inequalities in developed countries should therefore remain an open topic for research. To improve our knowledge of the causal origins of health inequalities, and to inform the policies aimed at reducing these inequalities, future research should focus especially on a harmonization of the measurement of health and SES indicators, on careful causal modeling, and on the replication of previous studies using a greater variety of data sources.

Acknowledgements

We would like to thank three anonymous reviewers for their helpful comments and suggestions to an earlier version of the article. The research was funded by the European Research Council (ERC): [ERC: 313532].

Conflicts of interest: None declared.

Key points

- For SES dimensions pertaining to labor market success, social causation and health selection are equally important for the explanation of health inequalities.
- For SES dimensions related to education, income and occupational class social causation was found to be more important than health selection.
- Future research should incorporate advanced statistical modeling approaches and longitudinal data structures to make inferences more robust.

References

- 1 Mackenbach JP. The persistence of health inequalities in modern welfare states: the explanation of a paradox. *Soc Sci Med* 2012;75:761–9.
- 2 Mackenbach J. Health inequalities: Europe in profile: an independent, expert report commissioned by the UK Presidency of the EU. Rotterdam Dept Public Health Erasmus MC 2006;41:3–46.
- 3 Mackenbach JP, Stirbu I, Roskam A-JR, et al. Socioeconomic inequalities in health in 22 European countries. *N Engl J Med* 2008;358:2468–81.
- 4 Singh GK, Siahpush M. Widening socioeconomic inequalities in US life expectancy, 1980–2000. *Int J Epidemiol* 2006;35:969–79.
- 5 Mackenbach JP, Kulhánová I, Menvielle G, et al. Trends in inequalities in premature mortality: a study of 3.2 million deaths in 13 European countries. *J Epidemiol Community Health*. 2015;69:207–17.
- 6 Goldman N. Social factors and health: the causation-selection issue revisited. *Proc Natl Acad Sci* 1994;91:1251–5.
- 7 Blane D, Smith GD, Bartley M. Social selection: what does it contribute to social class differences in health? *Sociol Health Illn* 1993;15:1–15.
- 8 Rubin DB. Causal inference using potential outcomes. *J Am Stat Assoc* 2005;100:322–31.
- 9 Heckman JJ. *Econometric Causality*. Cambridge, MA: National Bureau of Economic Research, 2008.
- 10 Williams DR. Socioeconomic differentials in health: a review and redirection. *Soc Psychol Q* 1990;53:81–99.
- 11 Feinstein JS. The relationship between socioeconomic status and health: a review of the literature. *Milbank Q* 1993;71:279–322.
- 12 Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation* 1993;88:1973–98.
- 13 Paul KI, Moser K. Unemployment impairs mental health: meta-analyses. *J Vocat Behav* 2009;74:264–82.
- 14 Sellström E, Bremberg S. Review article: the significance of neighbourhood context to child and adolescent health and well-being: a systematic review of multilevel studies. *Scand J Public Health* 2006;34:544–54.
- 15 Liberati A, Altman DG, Tetzlaff J, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanation and elaboration. *PLoS Med* 2009;6:1–28.
- 16 Rubin DB. The design versus the analysis of observational studies for causal effects: parallels with the design of randomized trials. *Stat Med* 2007;26:20–36.
- 17 Angrist JD, Pischke J-S. The credibility revolution in empirical economics: how better research design is taking the con out of econometrics. *J Econ Perspect* 2010;24:3–30.
- 18 Von Elm E, Altman DG, Egger M, et al. The strengthening of reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. *Prev Med* 2007;45:247–51.
- 19 Sanderson S, Tatt ID, Higgins JP. Tools for assessing quality and susceptibility to bias in observational studies in epidemiology: a systematic review and annotated bibliography. *Int J Epidemiol* 2007;36:666–76.
- 20 Gunasekara FI, Carter K, Blakely T. Change in income and change in self-rated health: systematic review of studies using repeated measures to control for confounding bias. *Soc Sci Med* 1982 2011;72:193–201.

- 21 Michaud P-C, van Soest A. Health and wealth of elderly couples: causality tests using dynamic panel data models. *J Health Econ* 2008;27:1312–25.
- 22 Cai L, Cong C. Effects of health and chronic diseases on labour force participation of older working-age Australians. *Aust Econ Pap* 2009;48:166–82.
- 23 Chandola T, Bartley M, Sacker A, et al. Health selection in the Whitehall II study, UK. *Soc Sci Med* 2003;56:2059–72.
- 24 Van de Mheen H, Stronks K, Looman CWN, Mackenbach PJ. Does childhood socioeconomic status influence adult health through behavioural factors? *Int J Epidemiol* 1998;27:431–7.
- 25 Ritsher JEB, Warner V, Johnson JG, Dohrenwend BP. Inter-generational longitudinal study of social class and depression: a test of social causation and social selection models. *Br J Psychiatry* 2001;178:s84–90.
- 26 Warren JR. Socioeconomic status and health across the life course: a test of the social causation and health selection hypotheses. *Soc Forces* 2009;87:2125–53.
- 27 Eaton WW, Muntaner C, Bovasso G, Smith C. Socioeconomic status and depressive syndrome: the role of inter- and intra-generational mobility, government assistance, and work environment. *J Health Soc Behav* 2001;42:277–94.
- 28 Mulatu MS, Schooler C. Causal connections between socio-economic status and health: reciprocal effects and mediating mechanisms. *J Health Soc Behav* 2002;43:22–41.
- 29 Palloni A, Milesi C, White RG, Turner A. Early childhood health, reproduction of economic inequalities and the persistence of health and mortality differentials. *Soc Sci Med* 2009;68:1574–82.
- 30 Miech RA, Caspi A, Moffitt TE, et al. Low socioeconomic status and mental disorders: a longitudinal study of selection and causation during young adulthood. *Am J Sociol* 1999;104:1096–131.
- 31 Van Der Lucht F, Groothoff J. Social inequalities and health among children aged 10–11 in the Netherlands: causes and consequences. *Soc Sci Med* 1995;40:1305–11.
- 32 Haas SA. Health selection and the process of social stratification: the effect of childhood health on socioeconomic attainment. *J Health Soc Behav* 2006;47:339–54.
- 33 Huurre T, Rahkonen O, Komulainen E, Aro H. Socioeconomic status as a cause and consequence of psychosomatic symptoms from adolescence to adulthood. *Soc Psychiatry Psychiatr Epidemiol* 2005;40:580–7.
- 34 Elovainio M, Ferrie JE, Singh-Manoux A, et al. Socioeconomic differences in cardiometabolic factors: social causation or health-related selection? Evidence From the Whitehall II cohort study, 1991–2004. *Am J Epidemiol* 2011;174:779–89.
- 35 Power C, Matthews S, Manor O. Inequalities in self rated health in the 1958 birth cohort: lifetime social circumstances or social mobility? *BMJ* 1996;313:449–53.
- 36 Power C, Manor O, Li L. Are inequalities in height underestimated by adult social position? Effects of changing social structure and height selection in a cohort study. *BMJ* 2002;325:131–4.
- 37 Elstad JJ, Krokstad S. Social causation, health-selective mobility, and the reproduction of socioeconomic health inequalities over time: panel study of adult men. *Soc Sci Med* 2003;57:1475–89.
- 38 Ribet C, Zins M, Gueguen A, et al. Occupational mobility and risk factors in working men: selection, causality or both? Results from the GAZEL study. *J Epidemiol Community Health* 2003;57:901–6.
- 39 Case A, Fertig A, Paxson C. The lasting impact of childhood health and circumstance. *J Health Econ* 2005;24:365–89.
- 40 Stansfeld SA, Clark C, Rodgers B, et al. Repeated exposure to socioeconomic disadvantage and health selection as life course pathways to mid-life depressive and anxiety disorders. *Soc Psychiatry Psychiatr Epidemiol* 2011;46:549–58.
- 41 Halleröd B, Gustafsson J-E. A longitudinal analysis of the relationship between changes in socio-economic status and changes in health. *Soc Sci Med* 2011;72:116–23.
- 42 Aittomaki A, Martikainen P, Laaksonen M, et al. Household economic resources, labour-market advantage and health problems—a study on causal relationships using prospective register data. *Soc Sci Med* 2012;75:1303–10.
- 43 Elovainio M, Pulkki-Raback L, Jokela M, et al. Socioeconomic status and the development of depressive symptoms from childhood to adulthood: a longitudinal analysis across 27 years of follow-up in the Young Finns study. *Soc Sci Med* 2012;74:923–9.
- 44 Ross CE, Mirowsky J. Does employment affect health? *J Health Soc Behav* 1995;36:230–43.
- 45 Steele F, French R, Bartley M. Adjusting for selection bias in longitudinal analyses using simultaneous equations modeling: the relationship between employment transitions and mental health. *Epidemiology* 2013;24:703–11.
- 46 Cai L. The relationship between health and labour force participation: evidence from a panel data simultaneous equation model. *Labour Econ* 2010;17:77–90.
- 47 Cai L, Kalb G. Health status and labour force participation: evidence from Australia. *Health Econ* 2006;15:241–61.
- 48 Cai L. Effects of health on wages of Australian men. *Econ Rec* 2009;85:290–306.
- 49 Haan P, Myck M. Dynamics of health and labor market risks. *J Health Econ* 2009;28:1116–25.
- 50 Heponiemi T, Elovainio M, Manderbacka K, et al. Relationship between unemployment and health among health care professionals: health selection or health effect? *J Psychosom Res* 2007;63:425–31.
- 51 Power C, Stansfeld SA, Matthews S, et al. Childhood and adulthood risk factors for socio-economic differentials in psychological distress: evidence from the 1958 British birth cohort. *Soc Sci Med* 2002;55:1989–2004.
- 52 Van De Mheen HD, Stronks K, Mackenbach JP. A Lifecourse perspective on socio-economic inequalities in health: the influence of childhood socio-economic conditions and selection processes. *Sociol Health Illn* 1998;20:754–77.
- 53 Braveman PA, Cubbin C, Egerter S, et al. Socioeconomic status in health research: one size does not fit all. *JAMA* 2005;294:2879–88.
- 54 Mastekaasa A. Unemployment and health: selection effects. *J Community Appl Psychol* 1996;6:189–205.
- 55 Haahr JPL, Frost P, Andersen JH. Predictors of health related job loss: a two-year follow-up study in a general working population. *J Occup Rehabil* 2007;17:581–92.
- 56 Schmidt S. Shall we really do it again? The powerful concept of replication is neglected in the social sciences. *Rev Gen Psychol* 2009;13:90–100.
- 57 Olesen SC, Butterworth P, Leach LS, et al. Mental health affects future employment as job loss affects mental health: findings from a longitudinal population study. *BMC Psychiatry* 2013;13:144.
- 58 Smith JP. Unraveling the SES: health connection. *Popul Dev Rev* 2004;30:108–32.
- 59 Adams J, White M, Pearce MS, Parker L. Life course measures of socioeconomic position and self reported health at age 50: prospective cohort study. *J Epidemiol Community Health* 2004;58:1028–9.
- 60 Adams P, Hurd MD, McFadden D, et al. Healthy, wealthy, and wise? Tests for direct causal paths between health and socioeconomic status. *J Econom* 2003;112:3–56.
- 61 Hamilton VH, Merrigan P, Dufresne É. Down and out: estimating the relationship between mental health and unemployment. *Health Econ* 1997;6:397–406.

Appendix

- A. Research protocol including complete list of keywords and Medline search terms.
- B. Bibliographic information on all studies from the literature search.
- C. Examples of excluded studies.
- D. Full table of all studies with all extracted data.
- E. Short summaries of all included studies.