



## The impact of education on dental health - ways to measure causal effects

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Abstract:	<p>To our knowledge, there are no studies in which a possible causal effect of education on dental health has been examined. Such studies are needed in order to predict whether more schooling for people with poor dental health, improves their dental health. Within social science, and in economics in particular, several methods have been developed to make causal inferences of the relationship between education and general health. These methods, which are based on observational data, are relevant to use for estimating a possible causal effect of education on dental health. This commentary provides an overview of the state-of-the-art of the following methods: the use of instrumental variables, twin studies and a regression discontinuity design. By using these methods, reversed causality and the omission of a third variable that influences both education and dental health can be controlled for. In that way, an unbiased estimate of the effect of education on dental health can be obtained. In order to implement each of the methods correctly, several criteria have to be fulfilled. These criteria are outlined and discussed below.</p>

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13 **The impact of education on dental health - ways to measure causal effects**  
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## Abstract

**To our knowledge**, there are no studies in which a possible causal effect of education on dental health has been examined. Such studies are needed in order to predict whether more schooling for people with poor dental health, improves their dental health. Within social science, and in economics in particular, several methods have been developed to make causal inferences of the relationship between education and general health. These methods, which are based on observational data, are relevant to use for estimating a possible causal effect of education on dental health. This commentary provides an overview of the state-of-the-art of the following methods: the use of instrumental variables, twin studies and a regression discontinuity design. By using these methods, reversed causality and the omission of a third variable that influences both education and dental health ~~are~~ **can be** controlled for. In that way, an unbiased estimate of the effect of education on dental health can be obtained. In order to implement each of the methods correctly, several criteria have to be fulfilled. These criteria are outlined and discussed below.

Key words: education, dental health, causal estimates, instrumental variables, twin studies, regression discontinuity

Running title: a possible causal effect of education on dental health

## Introduction

One of the most robust findings in the dental literature is the positive association between education and dental health. This relationship is found in many countries, at different educational levels and for various indicators of dental health. In a broader sense, the positive association between education and health is so ubiquitous that it is often referred to as “the gradient”<sup>1-4</sup>. However, this association may not reflect a causal relationship.

To my knowledge, **there is a lack of studies** in which a possible causal effect of education on dental health has been examined. Although correlation studies have a role to play, I believe that the most interesting research is about cause and effect. This is because causal estimates can be used to make valid predictions about the consequences of a change in circumstances or policies. For example, does more schooling for people with poor dental health, improve their dental health?

During the last 5-10 years, within the economic literature, there has been a lively debate about whether the relationship between education and health is causal or not (for example see:<sup>5-8</sup>). Alternatively, how much of it is causal? In this commentary, I will refer to this discussion, and relate it to the field of dentistry. Within the economic literature, a broad range of methods have been adopted for the estimation of causal effects. These methods are relevant to use for the estimation of causal effects of education on dental health. The strengths and weaknesses of these methods are discussed below. **I will discuss the effects of formal education only. Use of non-formal education, such as the use of Google and Wikipedia as an easy and useful source of general and specific knowledge to solve problems is not discussed.**

## Education – an important policy instrument

This discussion about causal effects of education on dental health is highly relevant for policy because, certainly in the long run, education is amenable to public policy interventions. For example, during the second half of the last century there has been a

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4 remarkable increase in the level of education in most western European countries<sup>9-14</sup>. In  
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6 1960 only 5 % of the population aged 25-64 years had tertiary education (Fig. 1). This  
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8 figure is the average for 14 western European countries. In 2005 the proportion had  
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10 increased to nearly 30 %. To a large extent, this increase is the result of public policies,  
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12 which have led to a substantial increase in the number of colleges and universities. Demand  
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14 side policies have also been effective, such as student grants and loans to stimulate young  
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16 adults to continue their education to a higher level.

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18 It is commonly believed that more schooling has given pecuniary returns, both to the  
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20 individual and to society as a whole. At the individual level more schooling leads to an  
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22 increase in labour participation and to higher earnings<sup>15</sup>. At the more aggregate level, an  
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24 increase in the proportion of young adults with tertiary education has made a substantial  
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26 contribution to the economic growth seen in most western countries during the second half  
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28 of the last century<sup>16</sup>. The issue is to what extent more schooling also provides non-  
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30 pecuniary returns, such as happier marriages, better parenting, more stable social networks,  
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32 more trustworthy individuals and better general health, dental health included. Better health  
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34 is often singled out as the key non-pecuniary benefit from additional schooling<sup>17</sup>.

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36 From a policy and efficiency point of view, it is a great advantage if education leads to an  
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38 increase in both pecuniary and non-pecuniary benefits. By using one policy instrument  
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40 only, i.e. raising the level of education, governments can achieve two aims simultaneously:  
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42 raise the wealth of the population and improve health. In the long run, this would be a more  
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44 successful policy than focusing on policies that only aim to improve health, such as  
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46 increasing public health care expenditure. In fact, such policies may not be that effective.  
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48 More resources spent on curative health care may have a minor effect on the health of the  
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50 population<sup>18-21</sup>.

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## What does schooling do?

Within a broader context, **and depending on the region/country**, schooling serves several aims, such as<sup>22-24</sup>:

- to develop personal autonomy and development of knowledge, skills and attitudes so that pupils can master their lives and as adults take part in working life and society
- to promote values related to democracy, equality and solidarity
- to teach pupils to act ethically and to respect human dignity and intellectual freedom.

With respect to the possible effect that schooling may have on health, the development of skills is particularly important. There are at least three types of skills<sup>25</sup>:

- Knowledge-based skills:  
Students learn to use information to develop skills that are needed to perform specific tasks.
- Critical thinking skills:  
Students learn logical reasoning so that they are able to assess information and draw inferences or conclusions that are supported by evidence.
- Social skills:  
These skills facilitate interaction and communication with others, **such as interaction with authorities and social structures/systems.**

The key question is then: Do the skills acquired through schooling lead to better dental health? A plausible answer is “yes”. In that case, the positive association between education and dental health should be interpreted as causal. This association could then be estimated using ordinary least squares regression analysis. However, there are other factors that can cause a positive association that have to be controlled for, i.e. eliminated from the picture. These factors are reversed causality and the omission of a third variable that influences both education and dental health<sup>6,26</sup>. Unless reversed causality and the third variable are

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4 controlled for, the regression coefficients measuring the effect of education on dental health  
5 are likely to be biased, i.e. the causal effect will be overestimated.  
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## 9 10 11 **Reversed causality** 12

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14 The direction of causality may run from better health to more education. In that case, health  
15 status contributes to the individual's social status. Within the literature in epidemiology and  
16 sociology, this is commonly termed a selection effect or a social drift effect<sup>27,28</sup>. These  
17 effects are most likely for diseases with an early onset that affect adult health. According to  
18 Grossman and Kaestner (1997) the observed relationship between adult health and  
19 schooling most likely suffers from an omitted variable bias unless infant/child health is  
20 controlled for in the analyses<sup>26</sup>. The omitted variable (infant/child health) is positively  
21 correlated with both adult health and schooling. Therefore, the omitted variable bias will be  
22 upwards<sup>26</sup>. Currie (2009) and Eide and Showalter (2011) have given excellent reviews of  
23 the literature that deals with the effects that infant/child health has on adult health and on  
24 education<sup>29,30</sup>. There are several pathways in which these effects may run – a few pathways  
25 are given below.  
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37 First, it is well established that infants with low birth weight have poorer school  
38 outcomes than infants with normal birthweight<sup>31-38</sup>. In addition, low birth weight is  
39 associated with poor health later in life; such as high blood pressure, cardiovascular  
40 disease, diabetes, asthma, kidney disease, mental disorders and caries<sup>39-45</sup>. Several of these  
41 health conditions are risk factors for reduced life expectancy.  
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47 Second, children's and adolescents' mental health has recently been recognized as  
48 being important for future life outcomes. For example, adolescents with behaviour  
49 problems such as hyperactivity and conduct disorders have significantly less schooling,  
50 lower earnings and less employment<sup>46-49</sup>. Teenagers diagnosed with ADHD have lower  
51 cognitive test scores than other children for mathematics and reading, much higher  
52 probabilities of being in special education or having repeated a year at school, and more  
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4 likely to suffer recurrent mental health problems as adults<sup>50-53</sup>. These effects are larger for  
5 ADHD than for other mental health problems, such as depression, among teenagers<sup>51</sup>.  
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7 Mental health symptoms that are not a diagnosis, such as mood changes and sleeping  
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9 problems, may also be important for teenagers' performance at school.  
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12 Third, older children who are sick or malnourished during childhood, independent  
13 of their birthweight, are more likely to miss school, learn less, and ultimately obtain fewer  
14 years of schooling<sup>26</sup>. Sick children are also more likely to become sick as adults. Specific  
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16 diseases among children and adolescents that have been identified as being important for  
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18 future educational attainment and adult health are: migraine, asthma and infectious diseases  
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20 such as typhoid, measles, influenza and diarrhea<sup>54-57</sup>.  
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### 27 **Omission of a third variable**

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29 Third variables influencing both education and health will lead to a biased estimate of the  
30 causal effect of education on health. In the case that the omitted variables are positively  
31 correlated with both education and health, the estimate will be upward biased. Third  
32 variables often mentioned in the literature are: parent's and sibling's level of education,  
33 cognitive ability, place of residence, and characteristics of the spouse<sup>6,17,26</sup> (Fig. 2).  
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35 Evidence from both the economic literature, and from the non-economic literature such as  
36 psychology, sociology and epidemiology, has shown that wealthy parents are likely to  
37 invest more in their children's health and in their education than less wealthy parents<sup>7, 58-63</sup>.  
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39 Therefore, unless family characteristics are controlled for, the regression coefficient  
40 measuring the effect of education on dental health is likely to be biased. Ability is another  
41 important confounding variable that should be controlled for. Smarter individuals may be  
42 more likely to obtain more schooling and to take better care of themselves, for example by  
43 having more favourable health behaviour<sup>64,65</sup>. Another potential confounder is place of  
44 residence<sup>66,67</sup>. For example, highly educated and wealthy individuals tend to live in affluent  
45 areas. These are also areas where the quality of both schooling and medical services is  
46 likely to be high. Finally, characteristics of the spouse may be an important confounder. A  
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4 highly educated person may marry a person who is also highly educated, and who has  
5 favourable health behaviour. Spillover effects with respect to adapting each other's health  
6 behaviour are likely<sup>68-72</sup>.  
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10 In the economic literature, the third variable most often mentioned is time preference<sup>73,74</sup>  
11 (Fig. 2). Decisions about education and health involve trade-offs of different outcomes over  
12 time. **How does the individual trade off current outcomes over future outcomes?**  
13 **People with a strong preference for the future relative to the present are more likely**  
14 **to invest in education, and at the same time they are more likely to engage in healthier**  
15 **activities and habits. Conversely, people who value the present highly will invest less**  
16 **in both education and healthy activities, such as looking after their teeth. It then**  
17 **follows that time preferences are likely to be positively correlated with both education**  
18 **and health. Unless these preferences are controlled for, the regression coefficients**  
19 **measuring the effect of education on health are likely to be upward biased, i.e. the**  
20 **causal effect will be overestimated<sup>75</sup>. Therefore, time preferences must be controlled**  
21 **for.**  
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32 As early as 1982, the award winning health economist Victor Fuchs rejected the hypothesis  
33 that education has a causal effect on smoking<sup>73,74</sup>. He showed that a negative relationship  
34 between education and smoking was due to time preferences. **Time preferences represent**  
35 **the individual's preference for current outcomes over future outcomes, i.e.**  
36 **preferences over the timing of outcomes<sup>75</sup>.** The time preference hypothesis has been  
37 tested by several researchers during the decades that followed after Fuchs's work. The main  
38 conclusion from that research is that the effect of education on health is markedly reduced,  
39 but does not disappear when time preferences are controlled for<sup>75,76</sup>.  
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## 51 **The instrumental variable approach**

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53 It is hard to distinguish a possible causal effect of education on dental health from potential  
54 confounders. One possibility is to make a list of all the confounders one can think of, and  
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4 include them in a regression model with education as the key explanatory variable and  
5 dental health as the outcome. This approach is difficult, mainly because several  
6 confounders are difficult to identify and measure. For example, how do you get a valid  
7 measure of ability? Even if the confounders could be measured, they may not be available  
8 in the set of data, or they may have been measured imprecisely.

14 Even if a possible confounder is available in the set of data, it should not necessarily be  
15 included in the analyses. This is supported by the evidence from research within the field of  
16 directed acyclic graphs<sup>77,78</sup>. The so-called colliders are of special interest. A variable is  
17 classified as a collider when it is the outcome of two variables of interest, for example an  
18 exposure and an outcome. Controlling for a collider will cause the regression coefficient  
19 between the exposure and the outcome to be biased. **This is because controlling for the  
20 collider opens the path between the exposure and the outcome, introducing a spurious  
21 (non-causal) association.** ~~the collider introduces a spurious (non-causal) association  
22 between the exposure and the outcome.~~ In technical terms, the collider “blocks” the  
23 association between the two variables of interest<sup>77,78</sup>.

32 Both unobserved variables and colliders are a challenge, because they easily introduce bias  
33 in the estimates of causal effects. To correct for these biases, several statistical techniques  
34 have been developed<sup>79</sup>. **In econometrics, probably** the most commonly used technique is  
35 the use of instrumental variables<sup>80,81</sup>. **Within dental public health and dentistry in  
36 particular, instrumental variables have hardly been used.** Instrumental variables began  
37 to be used in econometrics a long time ago. A substantial amount of work has subsequently  
38 been done to develop methods using these variables within epidemiology<sup>81-84</sup>. One can  
39 think of an instrumental variable as a device in which pseudo randomization is achieved  
40 using observational data. In an experiment, the assignment of individuals to a control group  
41 and a treatment group is done by randomization; i.e. they are not allocated to a group  
42 according to factors that are correlated with the outcome. **Instrumental variables are like  
43 a natural experiment. With the use of instrumental variables, the idea is to mimic the  
44 random assignment of controlled experiments by taking advantage of incidents where  
45 nature (for example floods, hurricane) or institutional rules and designs (for example  
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4 **introduction of any health care strategy, educational reforms) give rise to random**  
5 **variation**<sup>79</sup>. **It exploits the fact that nature sometimes makes random assignments.**  
6 **Therefore, such strategies for identification are often referred to as natural or quasi-**  
7 **experiments**<sup>85-87</sup>.  
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12 Within epidemiology and social sciences randomized controlled experiments are seldom  
13 feasible<sup>88</sup>. It would be unethical, practically difficult and very costly to randomly allocate  
14 preschool children into a “treatment group” and a “control group”. The treatment in this  
15 case is higher education, and the control group is compulsory schooling only.  
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20 Ideally, one would also want to observe the same child both as a control subject and as a  
21 test subject. This is not possible. The key issue is then to estimate what would have  
22 happened in the counterfactual situation; i.e. which outcome a treated person would have if  
23 he or she had not been treated. Differences in outcomes would then be fully explained by  
24 differences in length of schooling. As the counterfactual situation is not possible to observe,  
25 statistical techniques and research designs have been developed that have helped  
26 researchers to come as close as possible to this counterfactual comparison. Use of  
27 instrumental variables is one such technique, which if certain assumptions are fulfilled, is  
28 an effective tool to obtain causal estimates<sup>89</sup> (Fig. 3).  
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37 These assumptions are<sup>90</sup>:

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40 First, the instrumental variable must have a clear effect on the treatment variable; in  
41 our case on educational level. This is usually termed “instrument relevance”.  
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44 Second, the instrumental variable must not be correlated with the error term in the  
45 first stage regression, i.e. where the treatment variable is regressed on the instrumental  
46 variable. Alternatively, for a strong instrument this correlation can be small<sup>90</sup>.  
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50 Third, the instrumental variable must not be correlated with the error term in the  
51 regression equation. This is usually termed “the exclusion restriction”. This means that the  
52 instrumental variable has an effect on the outcome *only* through the treatment variable. In  
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4 that case, biases caused by reversed causality and omission of a third variable are  
5 eliminated.  
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## 10 11 **One type of instrument – school reforms**

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14 The trick is then to find a convincing instrumental variable. Valid instrumental variables are  
15 developed from a combination of in-depth institutional knowledge of the sector under  
16 study, and insight into the mechanism that determines the relationship between the  
17 instrumental variable and the treatment variable<sup>89</sup>. So far, within the social science  
18 literature, the most promising type of instrumental variable has been the introduction of  
19 compulsory schooling laws<sup>91</sup>. Such laws were introduced in several European and North  
20 American countries in different time periods during the last century.  
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28 Typically, **one of the most prominent** the effects of these laws was that the number of years  
29 of compulsory schooling was increased. In several countries, the number of years of  
30 compulsory schooling was increased from 7 to 9 years during the 1960s and the 1970s<sup>92</sup>.  
31 The laws were implemented at a national level and encompassed all preschool children; i.e.  
32 there was no selection of preschool children to the treatment group or control group  
33 according to their abilities, or their parents' level of education. The "treatment" group was  
34 then comprised of children with 9 years of compulsory education, and the "control" group  
35 was comprised of children with 7 years of compulsory education.  
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43 Several economists have used the random variation induced by the introduction of  
44 compulsory schooling to estimate causal effects of education on different types of health  
45 outcome measures (for example see:<sup>93-101</sup>). Such studies have been performed in the United  
46 States, Great Britain, the Netherlands and the Scandinavian countries. In several of the  
47 studies, but not all of them, a causal effect of education has been found. Typically, the  
48 regression coefficient from the instrumental variable estimation is smaller, often much  
49 smaller, than the ordinary least squares estimation (for a review see:<sup>91,92</sup>). This underscores  
50 the importance of using a statistical technique in which confounding variables have been  
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4 controlled for. These confounding variables are unobservable, hence their individual effects  
5 cannot be estimated.  
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9 **Several studies have found that the causal effects vary by gender<sup>95,100,102-104</sup>. For**  
10 **example, in a study from Germany, Kemptner et al. (2011) used the random variation**  
11 **induced by a school reform to estimate causal effects of education on different types of**  
12 **health outcome measures separately for men and women<sup>100</sup>. In Germany, the number**  
13 **of years of compulsory schooling increased from 8 to 9 years during the period 1949 to**  
14 **1970. Kemptner et al. (2011) showed in the first stage regression that the introduction**  
15 **of a compulsory 9<sup>th</sup> grade led to an increase in schooling of 0.6 years<sup>100</sup>. This is**  
16 **equivalent to the effect shown by the arrow from the *instrumental variable* to the**  
17 ***treatment variable* in Fig. 3. For men, the second stage estimate showed that one more**  
18 **year of schooling led to a reduction in the likelihood of work disability of 3.2**  
19 **percentage points, and of suffering from a long term illness of 4.1 percentage points.**  
20 **These figures are equivalent to the effect shown by the arrow from the *treatment***  
21 ***variable* to the *outcome variable* in Fig. 3. In contrast to men, women did not gain from**  
22 **more schooling in terms of improvements in health: the second stage estimate had an**  
23 **insignificant effect on health outcomes.**  
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37 Typically, ~~all~~ **most** studies in which a school reform has been used as an instrumental  
38 variable have been performed using panel data. Use of panel data is a great advantage, as it  
39 makes it possible to perform an anti-test (placebo test)<sup>105</sup>. Such a test can be used to assess  
40 the validity of the instrumental variable. An anti-test provides counter evidence by  
41 estimating a model in a context where no effect should be found. If an apparent effect is  
42 found, then the validity of the instrumental variable is questionable<sup>105</sup>.  
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48 With panel data we can **test the assumptions of instrumental variable estimation by**  
49 **pretending** ~~perform an experiment in which we pretend~~ that the increase in the number of  
50 years of compulsory schooling was introduced earlier than it actually was introduced. In  
51 such an experiment, we do not expect the reform to have any effect on health outcomes. If  
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4 there is an effect, the instrumental variable would be correlated with a third variable. Then  
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6 we have a poor instrument.  
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## 10 11 **Weaknesses of the instrumental variable approach**

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14 In the literature, different types of instrumental variables have been suggested. Most of  
15 them, but not all, have been dismissed (for a review see:<sup>6</sup>). The main reason is that the  
16 instrumental variable does not have a clear effect on the treatment variable. The correlation  
17 between the instrumental variable and the treatment variable might be small, i.e. the  
18 instrument is weak. This will occur with confounding<sup>90</sup>. Generally, the correlation between  
19 the instrumental variable and the treatment variable becomes weaker as the amount of  
20 confounding becomes larger. A weak instrumental variable leads to a biased estimate of the  
21 treatment effect. This bias will be in the direction of the estimate from the ordinary least  
22 squares regression of the outcome on the treatment variable<sup>90</sup>. Information about the size of  
23 the bias can be obtained from the F-statistic, where the treatment variable is regressed on  
24 the instrumental variable (i.e. the first stage regression). An F-value close to 1 indicates a  
25 large sample bias, whereas a value of 10 indicates that the bias is negligible<sup>106</sup>. A strength  
26 of most studies in which the introduction of a school reform has been used as an  
27 instrumental variable is that the F-values have been high<sup>95-97,99-101,107</sup>.  
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40 A potential weakness of using compulsory schooling laws for identification is that the  
41 consistency assumption for causal inference may be violated. This assumption “entails that  
42 the exposure is defined with enough specificity that different variants of the exposure do  
43 not have different effects on the outcome”<sup>108</sup>. Commonly, education is operationalized as  
44 the number of years of compulsory schooling. This measure does not capture different  
45 aspects of school quality; such as differences in school term duration. Another factor to  
46 consider is that compulsory schooling laws either introduce an extra year of schooling at an  
47 early age (for example the school entry age is reduced from 7 to 6 years of age) or an extra  
48 year of schooling in adolescence (for example the school leaving age is increased from 14  
49 to 15 or 16 years of age). Young and old children are at different stage of development.  
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4 This is not taken into account in the instrumental variable estimation, where the number of  
5 years of schooling is the treatment variable. Rehkopf et al. (2016) argue that “multiple  
6 attributes of education that are not typically specified may be differently associated with  
7 health outcomes, thus use of standard measures of level of education may violate the  
8 consistency assumption”<sup>108</sup>. In that case, the instrumental variable does not give a valid  
9 estimate of the causal effects of education on health outcomes. This has led researchers to  
10 look for alternative methods to estimate causal effects. Two such methods are twin studies,  
11 and studies with a regression discontinuity design.  
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### 23 **Twin studies**

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25 Identical (monozygotic) twins share common genes and have a common family  
26 background. Therefore, at the end of the last century, twin studies were considered to be a  
27 very promising way of estimating the causal effects of education on health outcome<sup>17,91</sup>. It  
28 was commonly believed that confounding variables related to intelligence and family  
29 characteristics were controlled for with samples of identical twins.  
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35 However, there are few identical twin studies in which the effect of education on health  
36 outcomes has been examined. In the few studies that exist, the effect of education is smaller  
37 in twin studies than in studies of the general population<sup>109-111</sup>. This indicates that  
38 intelligence and family characteristics are important confounding variables.  
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43 One challenge when studying identical twins is to get a large enough sample. A larger  
44 sample can be obtained by including non-identical (dizygotic) twins in the samples. That  
45 improves the statistical power of the analyses. Dizygotic twins are expected to share the  
46 same family environment, but their genes are different. **However, it can be questioned  
47 whether the family environment is similar for both twins. For example, twins that are  
48 not of same sex may be treated differently by parents, teachers and society in general.**  
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54 An interesting approach ~~is would be~~ to compare the similarity in health outcomes according  
55 to schooling of identical and non-identical twins. Such a comparison could provide insight  
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4 into the role of genes as opposed to family characteristics in determining education and  
5 health outcomes<sup>109,110,112</sup>. For example, we might find an effect of education on health  
6 outcomes for non-identical twins, but not for identical twins. That would indicate that  
7 genetics is the confounding variable that determines both level of education and health.  
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12 An extension of non-identical twin studies is comparison between siblings and between  
13 cousins. This is frequently done in epidemiology, public health and psychology<sup>78,113-116</sup>. In  
14 studies with a sibling-comparison design, the researcher exploits the fact that siblings share  
15 the same family environment as well as half the genome<sup>78,117</sup>. Outcomes are compared  
16 between siblings who are discordant on the intervention, and who are as similar as possible  
17 in family context and genetic predisposition. ~~To my knowledge,~~ There are ~~no~~ a few studies  
18 in which a sibling-comparison design has been used to estimate the causal effects of  
19 education on health outcome<sup>118,119</sup>. Such a design is not without its limitations. A concern  
20 is that the design is sensitive to measurement error and to confounding from factors that the  
21 siblings do not share<sup>116,117,120</sup>. Further, there should be no carryover effects; i.e. the effect of  
22 each participant's exposure to the risk factor should not influence the unexposed  
23 participants<sup>116,117,120</sup>. This assumption may be difficult to fulfill in the case where the  
24 exposed sibling and the unexposed sibling vary in years of schooling only. Skills and  
25 knowledge may well be transferred from the sibling with the most schooling to the sibling  
26 with the least schooling. The effect of schooling may then be underestimated.  
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40 Recently, several shortcomings of identical twin studies have also been suggested<sup>112,121</sup>.  
41 Are differences in schooling between identical twins random? Since monozygotic twins are  
42 meant to be genetically identical, it has been assumed that any variation in their schooling  
43 must be purely random. This may not be the case.  
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48 Monozygotic twins are different because they have different experiences. Different  
49 experiences begin as early as in the womb. For example, they compete for nourishment in  
50 the uterus. The "winner" grows and thrives at the expense of the "loser". The result is a  
51 difference in birthweight, which is correlated with a difference in educational attainment,  
52 ability and health later in life<sup>34,122-124</sup>. After birth, twins continue to experience different  
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4 environments. For example, they may be separated when they start school by being placed  
5 in different classes, then they are exposed to different teachers and classmates. Parents do  
6 not treat identical twins the same; a less able twin may be treated differently from a more  
7 able twin. Also, identical twins have different personalities, and this also causes parents to  
8 treat them differently. Finally, from an early age twins have a psychological need to be  
9 individuals. This increases their differences.  
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16 This indicates that differences in schooling between identical twins may be systematic  
17 rather than random. If these systematic differences are correlated with both education and  
18 health, then identical twin studies are not likely to provide causal estimates of education on  
19 health.  
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### 27 **Regression discontinuity design**

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30 Regression discontinuity uses precise rules that determine whether an individual ends up in  
31 a treatment group or a control group<sup>125</sup>. The designs are of two types: sharp design and  
32 fuzzy design. A graphical illustration of the sharp design is given in Fig. 4. All individuals  
33 who score above a predetermined cut-off value are assigned to the intervention group  
34 (treatment group), while those who score below are assigned to the control group. This is  
35 different from in a randomized controlled trial in which individuals are randomly assigned  
36 to a treatment group or a control group. In this simple illustration, the difference or  
37 “discontinuity” in the two regression lines provides an estimate of the intervention effect.  
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45 The idea of the regression discontinuity design is to compare individuals in a small range  
46 above and below the cut-off point<sup>126,127</sup>. These individuals only differ by being treated or  
47 not treated. They are meant to be identical regarding all observable and unobservable  
48 confounding variables that could influence the outcome. Any difference in outcome around  
49 the cut-off value can then be interpreted as a causal effect of the intervention. A limitation  
50 of the regression discontinuity design is that the external validity is often low. The results  
51 are difficult to generalize to populations within a broader range from the cut-off value.  
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4 An example of a cut-off value commonly used is date of birth, for example the 1st  
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6 January<sup>128,129</sup>. In several countries, school start is determined by the calendar year in which  
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8 children are born. Children who were born in December are the youngest, and those who  
9  
10 were born in January are the oldest in their class. Typically, researchers use this difference  
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12 in date of birth around the cut-off value to examine the effects of age at school entry on  
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14 outcomes such as test scores, educational attainment and labour market participation (for a  
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16 review see:<sup>130</sup>). The question is whether children who start school at a younger age do  
17  
18 worse than those who start at an older age.

19  
20 **In a few studies, a fuzzy regression discontinuity design has been used to estimate the**  
21 **causal effects of education on health outcomes<sup>94,95,131</sup>. In such a design, treatment and**  
22 **control individuals are observed both below and above the cut-off point. The design**  
23 **exploits discontinuities in the probability of treatment. Thus, it can be interpreted as**  
24 **an instrumental variable approach where the discontinuity is the instrument for**  
25 **treatment<sup>79</sup>. Van Kippersluis et al. (2011) used the discontinuity in schooling caused**  
26 **by a school reform in the Netherlands in the 1920s, to estimate causal effects of**  
27 **education on mortality in old age<sup>95</sup>. The first stage regression showed that the years of**  
28 **schooling increased by 0.8 years around the cut-off point. Their second stage estimate**  
29 **showed that one more year of schooling reduced the probability of dying before the**  
30 **age of 89 by 3 percentage points. Van Kippersluis (2011) performed several**  
31 **robustness tests, which showed that the treatment was as good as randomly assigned**  
32 **around the cut-off point<sup>95</sup>.**  
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## 46 **Issues related to statistical inference**

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49 All the three methods described above rely on the use of regression analyses for estimation.  
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51 In order to obtain reliable estimates from the analyses, there are at least three statistical  
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53 issues to consider. These are:  
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5 First, clustering: Typically, the data will have a multilevel structure, for example  
6 preschool children living in different municipalities, and twins growing up in different  
7 families. In statistical terms, individuals are clustered within a higher order unit.  
8 Observations within a cluster are correlated – there is dependence between them. This is  
9 because preschool children or twins within the same cluster have some of the same  
10 environmental and family background characteristics. Failure to control for within-cluster  
11 dependence can lead to misleadingly small standard errors, and consequently low p-values,  
12 i.e. the null hypothesis will incorrectly be rejected<sup>132</sup>. Controlling for clustering within data  
13 by using clustered/robust standard errors is fairly straightforward with cross-sectional data,  
14 but a bit trickier with panel data. With panel data, there is correlation between the same  
15 observations over time as well as correlation between different observations at one point in  
16 time. The correlation over time is due to the fact that, for example, once a school reform  
17 has been implemented it remains in place in all the subsequent years. With panel data,  
18 controlling for clustering within data by using clustered/robust standard errors can be  
19 particularly challenging in the case of “few” clusters, usually defined as less than 20<sup>132</sup>.  
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32 Second, trends: With panel data, it is common to include different trend  
33 specifications in the regression analyses. This is particularly important if unobserved  
34 variables influence the treatment group and the control group differently<sup>133</sup>. Whether this is  
35 the case or not, can be tested. For example, in the analyses using the introduction of  
36 compulsory schooling laws as the instrumental variable, municipality specific trends could  
37 be included as control variables. The estimate of schooling on mortality is biased if it is  
38 influenced by the inclusion of these trend variables. In that case, the instrumental variables  
39 are correlated with some unobservable variables in the error term in the regression  
40 equation. Then the exclusion criterion for the instrumental variable is violated and the  
41 instrument is most likely not appropriate for examination of the effect, which may still be  
42 causal.  
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52 Third, external validity: For several identification strategies, only part of the  
53 population is exposed to the intervention. For example, the introduction of compulsory  
54 schooling laws only affects those at the bottom tail of the educational distribution. The  
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4 question is whether we can generalize the findings to people in the middle and upper range  
5 of the educational distribution<sup>89,134</sup>. For twin samples, the question is whether the findings  
6 can be generalized to the population of people who are not twins. Under certain  
7 assumptions such a generalization is possible. These assumptions are outlined and  
8 discussed in the broader and more recent literature related to local and average treatment  
9 effects<sup>89,134</sup>.

### 18 **Issues related to interpretation of causal estimates**

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21 A relevant question is whether the causal estimate obtained through the use of compulsory  
22 schooling laws, should be interpreted as an absolute or a relative effect of education. In the  
23 case of an absolute effect interpretation, the focus is on each individual's level of education,  
24 and whether more schooling lowers the risk of mortality or having poor health. An  
25 alternative view is that an individual's health is also a function of the level of education in  
26 his or her reference group. Usually, the relevant reference group is defined as individuals  
27 who live in the same area, for example in the same municipality<sup>135-139</sup>. Relative education  
28 can then be defined as the level of education of an individual relative to the mean  
29 educational level of the reference group. The causal estimates of education on health  
30 identified through compulsory schooling laws may represent a relative rather than an  
31 absolute effect. This is because a school reform functions as a way of stratifying individuals  
32 relative to each other. Those who were exposed to the reform increase their level of  
33 education relative to those who were not exposed. **This implies that those who were**  
34 **exposed benefit, in terms of better health, while those who were not exposed may lose**  
35 **out in terms of worse health.**

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49 In the literature in public health and epidemiology, there are numerous studies in which the  
50 effects of relative income on health have been estimated. In several studies, it is found that  
51 relative income matters (for a review see: <sup>140-143</sup>). Wilkinson has argued that this is because  
52 people are concerned about their place in the social hierarchy, based on their relative  
53 position according to income<sup>144</sup>. Low relative income may cause psychosocial stress and  
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4 depression, conditions that are associated with poor health and risky health behaviour such  
5 as smoking and drinking. Lynch et al. (2000) further argue that a low place in the hierarchy  
6 can easily be translated into “antisocial behaviour, reduced civic participation, and less  
7 social capital and cohesion within the community”<sup>145</sup>. These are all factors that raise the  
8 probability of contracting an illness. These effects of relative income are most likely caused  
9 by education<sup>146,147</sup>. In real life, education predates income, and those with more education  
10 have higher income. Thus income may be the mediating variable in which education exerts  
11 its effect. Most likely, in the case of identification through compulsory schooling laws, this  
12 effect is due to a relative effect of education.  
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## 24 **Conclusion**

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26 There are different strategies that can be used to estimate causal effects of education on  
27 dental health. They each have their strengths and weaknesses. Identification through  
28 compulsory schooling laws is a promising strategy. This is because in most studies, the  
29 instrument has been shown to be valid; as indicated by high F-values. A potential weakness  
30 using compulsory schooling laws for identification is that the consistency assumption for  
31 causal inference may be violated. In particular, years of compulsory schooling does not  
32 capture different aspects of school quality. Identical twin studies have the potential to  
33 identify causal effects. The challenge is to get large enough samples, which is necessary in  
34 order to obtain sufficient precision of the regression coefficients. Larger samples can be  
35 obtained by using non-identical twins, siblings or cousins. However, such a design is not  
36 without its limitations. A particular concern is confounding from factors that the siblings do  
37 not share. The use of a regression discontinuity design is an alternative. However, the  
38 challenge is to find a precise rule to determine whether a child ends up in the group with  
39 much schooling (treatment group) or the group with little schooling (control group). It is  
40 not obvious what the rule should be.  
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54 Whatever the weaknesses are, the use of one or more of the methods outlined above is an  
55 improvement on the methodologies that have been used so far to estimate the relationship  
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4 between education and dental health. The prevailing methods, which have been correlation  
5 studies, have not been able to provide causal estimates. The methods described above have  
6 the potential to provide such estimates.  
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### 10 11 12 13 **Acknowledgements** 14

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60

## References

1. Watt RG, Listl S, Peres M, Heilman A, editors. Social inequalities in oral health: from evidence to action. London: International Centre for Oral Health Inequalities Research & Policy, 2015.
2. Sabbah W, Tsakos G, Chandola T, Sheiham A, Watt RG. Social gradients in oral and general health. *J Dent Res* 2007; 86: 992-6.
3. Geyer S, Schneller T, Micheelis W. Social gradients and cumulative effects of income and education on dental health in the Fourth German Oral Health Study. *Community Dent Oral Epidemiol* 2010; 38: 120-8.
4. Sanders A, Slade GD, Turrell G, Spencer AJ, Marcenes W. The shape of the socioeconomic-oral health gradient: implications for theoretical explanations. *Community Dent Oral Epidemiol* 2006; 34: 310-9.
5. Cutler DM, Lleras-Muney A. Education and health: evaluating theories and evidence. NBER Working Paper 12352. Cambridge, MA: National Bureau of Economic Research, 2006. Available at: <http://www.nber.org/papers/w12352> (February 16, 2017).
6. Grossman M. Education and nonmarket outcomes. In: Hanushek EA, Welch F, editors: *Handbook of the economics of education*, Volume 1. North-Holland: Elsevier, 2006; 577-633.
7. Desai S, Alva S. Maternal education and child health: is there a strong causal relationship? *Demography* 1998; 35: 71-81.
8. Cutler DM, Lleras-Muney A, Vogl T. Socioeconomic status and health: dimensions and mechanisms. In: Glied S, Smith PC, editors: *The Oxford handbook of health economics*. Oxford: Oxford University Press, 2011; 124-63.

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56  
57  
58  
59  
60
9. Dolton P, Asplund R, Barth E. Education, wage inequality and the labour market. In: Dolton P, Asplund R, Barth E, editors: Education and inequality across Europe. Cheltenham: Edward Elgar, 2009; 1-23.
10. De la Fuente A, Doménech R. Educational attainment in the OECD, 1960-95. CEPR Discussion Paper No. 3390. London: Centre for Economic Policy Research, 2002.
11. OECD. Education at a glance 2005. Paris: OECD, 2005.
12. OECD. Education at a glance 2006. Paris: OECD, 2006.
13. OECD. Education at a glance 2007. Paris: OECD, 2007.
14. OECD. Employment outlook 2007. Paris: OECD, 2007.
15. Harmon C, Oosterbeek H, Walker I. The returns to education: microeconomics. J Econ Surv 2003; 17: 115-55.
16. Sianesi B, Van Reenen J. The returns to education: macroeconomics. J Econ Surv 2003; 17: 157-200.
17. Oreopoulos P, Salvanes KG. Priceless: the nonpecuniary benefits of schooling. J Econ Perspect 2011; 25: 159-84.
18. World Health Organization. The World Health Report 2000. Health systems: improving performance. Geneva: World Health Organization, 2000.
19. Evans RG, Barer ML, Marmor TR, editors. Why are some people healthy and others not? The determinants of health of populations. New York: Aldine de Gruyter, 1994.
20. Watt RG. From victim blaming to upstream action: tackling the social determinants of oral health inequalities. Community Dent Oral Epidemiol 2007; 35: 1-11.



- 1  
2  
3  
4 21. Petersen PE. The World Oral Health Report 2003: continuous improvement of oral  
5 health in the 21<sup>st</sup> century – the approach of the WHO Global Oral Health  
6 Programme. Community Dent Oral Epidemiol 2003; 31 (Suppl 1): 3-24.  
7
- 8  
9  
10  
11 22. Brighouse H. Moral and political aims of education. In: Siegel H, editor: The  
12 Oxford handbook of philosophy of education. Oxford: Oxford University Press,  
13 2009.  
14
- 15  
16  
17 23. Child Rights International Network. Convention on the rights of the child. Article  
18 29: aims of education. Available at:  
19 <https://www.crin.org/en/home/rights/convention/articles/article-29-aims-education> .  
20 (February 16, 2017).  
21  
22
- 23  
24  
25 24. Ministry of Education and Research. Education act. 2007. Available at:  
26 <https://www.regjeringen.no/en/dokumenter/education-act/id213315/> (February 16,  
27 2017).  
28  
29
- 30  
31 25. Embrey D. Understanding human behaviour and error. Human Reliability  
32 Associates, 2005. Available at: [http://www.humanreliability.com/articles/](http://www.humanreliability.com/articles/Understanding%20Human%20Behaviour%20and%20Error.pdf)  
33 [Understanding%20Human%20Behaviour%20and%20Error.pdf](http://www.humanreliability.com/articles/Understanding%20Human%20Behaviour%20and%20Error.pdf) (February 16,  
34 2017).  
35  
36
- 37  
38  
39 26. Grossman M, Kaestner R. Effects of education on health. In: Behrman JR, Stacey  
40 N, editors: The social benefits of education. Ann Arbor: The University of  
41 Michigan Press, 1997; 69-123.  
42  
43
- 44  
45 27. Adler NE, Ostrove JM. Socioeconomic status and health: what we know and what  
46 we don't. Ann NY Acad Sci 1999; 896: 3-15.  
47  
48
- 49  
50 28. Fox AJ, Goldblatt PO, Jones DR. Social class mortality differentials: artefact,  
51 selection or life circumstances? J Epidemiol Community Health 1985; 39: 1-8.  
52  
53
- 54  
55 29. Currie J. Healthy, wealthy, and wise: socioeconomics status, poor health in  
56 childhood, and human capital development. J Econ Lit 2009; 47: 87-122.  
57  
58  
59  
60

- 1
- 2
- 3
- 4
- 5 30. Eide ER, Showalter MH. Estimating the relation between health and education:  
6 what do we know and what do we need to know? *Econ Educ Rev* 2011; 30: 778-91.  
7
- 8
- 9 31. Bhutta AT, Cleves MA, Casey PH, Cradock MM, Anand KJS. Cognitive and  
10 behavioral outcomes of school-aged children who were born preterm. *JAMA* 2002;  
11 288: 728-37.  
12
- 13
- 14
- 15 32. Anderson P, Doyle LW, the Victorian Infant Collaborative Study Group.  
16 Neurobehavioral outcomes of school-age children born extremely low birth weight  
17 or very preterm in the 1990s. *JAMA* 2003; 289: 3264-72.  
18
- 19
- 20
- 21 33. Hack M, Flannery DJ, Schluchter M, Cartar L, Borawski E, Klein N. Outcomes in  
22 young adulthood for very-low-birth-weight infants. *N Engl J Med* 2002; 346: 149-  
23 57.  
24
- 25
- 26
- 27 34. Black SE, Devereux PJ, Salvanes KG. From the cradle to the labor market? The  
28 effect of birth weight on adult outcomes. *Q J Econ* 2007; 122: 409-39.  
29
- 30
- 31
- 32 35. Currie J, Hyson R. Is the impact of health shocks cushioned by socioeconomic  
33 status? The case of low birthweight. *Am Econ Rev* 1999; 89: 245-50.  
34
- 35
- 36 36. Behrman JR, Rosenzweig MR. Returns to birthweight. *Rev Econ Stat* 2004; 86:  
37 586-601.  
38
- 39
- 40 37. Royer H. Separated at girth: estimating the long-run and intergenerational effects of  
41 birthweight using twins. Ann Arbor MI: Ford School of Public Policy, University of  
42 Michigan, 2005.  
43
- 44
- 45
- 46 38. Conley D, Bennett NG. Is biology destiny? Birth weight and life chances. *Am*  
47 *Sociol Rev* 2000; 65: 458-67.  
48
- 49
- 50
- 51 39. Hardy R, Wadsworth MEJ, Langenberg C, Kuh D. Birthweight, childhood growth,  
52 and blood pressure at 43 years in a British birth cohort. *Int J Epidemiol* 2004; 33:  
53 121-9.  
54
- 55
- 56
- 57
- 58
- 59
- 60

- 1  
2  
3  
4  
5 40. Holland FJ, Stark O, Ades AE, Peckham CS. Birth weight and body mass index in  
6 childhood, adolescence, and adulthood as predictors of blood pressure at age 36. *J*  
7 *Epidemiol Community Health* 1993; 47: 432-5.
- 8  
9  
10  
11 41. Hoy WE, Rees M, Kile E, Mathews JD, Wang Z. A new dimension to the Barker  
12 hypothesis: low birthweight and susceptibility to renal disease. *Kidney Int* 1999; 56:  
13 1072-7.
- 14  
15  
16  
17 42. Iliadou A, Cnattingius S, Lichtenstein P. Low birth weight and Type 2 diabetes: a  
18 study on 11 162 Swedish twins. *Int J Epidemiol* 2004; 33: 948-53.
- 19  
20  
21  
22 43. Shaheen SO, Sterne JAC, Montgomery SM, Azima H. Birth weight, body mass  
23 index and asthma in young adults. *Thorax* 1999; 54: 396-402.
- 24  
25  
26  
27 44. Wahlbeck K, Forsén T, Osmond C, Barker DJP, Eriksson JG. Association of  
28 schizophrenia with low maternal body mass index, small size at birth, and thinness  
29 during childhood. *Arch Gen Psychiatry* 2001; 58: 48-52.
- 30  
31  
32  
33 45. Nicolau B, Marcenes W, Bartley M, Sheiham A. A life course approach to assessing  
34 causes of dental caries experience: the relationship between biological, behavioural,  
35 socio-economic and psychological conditions and caries in adolescents. *Caries Res*  
36 2003; 37: 319-26.
- 37  
38  
39  
40 46. Miech RA, Caspi A, Moffitt TE, Entner Wright BR, Silva PA. Low socioeconomic  
41 status and mental disorders: a longitudinal study of selection and causation during  
42 young adulthood. *Am J Sociol* 1999; 104: 1096-131.
- 43  
44  
45  
46 47. Farmer EMZ. Extremity of externalizing behavior and young adult outcomes. *J*  
47 *Child Psychol Psyc* 1995; 36: 617-32.
- 48  
49  
50  
51 48. Kessler RC, Foster CL, Saunders WB, Stang PE. Social consequences of psychiatric  
52 disorders, I: educational attainment. *Am J Psychiat* 1995; 152: 1026-32.
- 53  
54  
55  
56 49. McLeod JD, Shanahan MJ. Trajectories of poverty and children's mental health. *J*  
57 *Health Soc Behav* 1996; 37: 207-20.
- 58  
59  
60

- 1
- 2
- 3
- 4
- 5 50. Currie J, Stabile M. Child mental health and human capital accumulation: the case
- 6 of ADHD. *J Health Econ* 2006; 25: 1094-1118.
- 7
- 8
- 9 51. Currie J, Stabile M. Mental health in childhood and human capital. In: Gruber J,
- 10 editor: *The problems of disadvantaged youth: an economic perspective*. Chicago:
- 11 University of Chicago Press, 2009; 115-48.
- 12
- 13
- 14
- 15 52. Mannuzza S, Klein R. Long-term prognosis in attention-deficit/hyperactivity
- 16 disorder. *Child Adol Psych Cl* 2000; 9: 711-26.
- 17
- 18
- 19 53. Webbink D, Vujic S, Koning P, Martin NG. The effect of childhood conduct
- 20 disorder on human capital. *Health Econ* 2012; 21: 928-45.
- 21
- 22
- 23
- 24 54. Rees DI, Sabia JJ, The effect of migraine headache on educational attainment. *J*
- 25 *Hum Resour* 2011; 46: 317-32.
- 26
- 27
- 28 55. Fowler MG, Davenport MG, Garg R. School functioning of US children with
- 29 asthma. *Pediatrics* 1992; 90: 939-44.
- 30
- 31
- 32
- 33 56. Halterman JS, Montes G, Aligne CA, Kaczorowski JM, Hightower D, Szilagyi PG.
- 34 School readiness among urban children with asthma. *Ambul Pediatr* 2001; 14: 201-
- 35 5.
- 36
- 37
- 38
- 39 57. Case A, Paxson SC. Early life health and cognitive function in old age. *Am Econ*
- 40 *Rev* 2009; 99: 104-9.
- 41
- 42
- 43 58. Davis-Kean PE. The influence of parent education and family income on child
- 44 achievement: the indirect role of parental expectations and the home environment. *J*
- 45 *Fam Psychol* 2005; 19: 294-304.
- 46
- 47
- 48
- 49 59. Sewell W, Shah VP. Parents' education and children's educational aspirations and
- 50 achievements. *Am Sociol Rev* 1968; 33: 191-209.
- 51
- 52
- 53
- 54 60. Ermisch J, Francesconi M. Family matters: impacts of family background on
- 55 educational attainments. *Economica* 2001; 68: 137-56.
- 56
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60
61. Newacheck PW, Strickland B, Shonkoff JP, Perrin JM, McPherson M, McManus M, Lauver C, Fox H, Arango P. An epidemiologic profile of children with special health care needs. *Pediatrics* 1998; 102: 117-23.
62. Lau RR, Quadrel MJ, Hartman KA. Development and change of young adults' preventive health beliefs and behavior: influence for parents and peers. *J Health Soc Behav* 1990; 31: 240-59.
63. Case A, Fertig A, Paxson C. The lasting impact of childhood health and circumstance. *J Health Econ* 2005; 24: 365-89.
64. Baker DW, Parker RM, Williams MV, Clark WS, Nurss J. The relationship of patient reading ability self-reported health and use of health services. *Am J Public Health* 1997; 87: 1027-30.
65. Auld MC, Sidhu N. Schooling, cognitive ability and health. *Health Econ* 2005; 14: 1019-34.
66. Eberhardt MS, Pamuk ER. The importance of place of residence: examining health in rural and nonrural areas. *Am J Public Health* 2004; 94: 1682-6.
67. Hartley D. Rural health disparities, population health, and rural culture. *Am J Public Health* 2004; 94: 1675-8.
68. Falba TA, Sindelar JL. Spousal concordance in health behavior change. *Health Serv Res* 2008; 43: 96-116.
69. Sutton GC. Assortative marriage for smoking habits. *Ann Hum Biol* 1980; 7: 449-56.
70. Venters MH, Jacobs R Jr, Luepker RV. Spouse concordance of smoking patterns: the Minnesota Heart Survey. *Am J Epidemiol* 1984; 120: 608-16.

- 1  
2  
3  
4 71. Leonard KE, Das Eiden R. Husband's and wife's drinking: unilateral or bilateral  
5 influences among newlyweds in a general population sample. *J Stud Alcohol* 1999;  
6 60: 130-8.  
7  
8
- 9  
10 72. Macken LC, Yates B, Blancher S. Concordance of risk factors in female spouses of  
11 male patients with coronary heart disease. *J Cardiopulm Rehabil* 2000; 20: 361-8.  
12  
13
- 14 73. Fuchs VR. Time preference and health: an exploratory study. In: Fuchs, editor:  
15 *Economic aspects of health*. Chicago: University of Chicago Press for the National  
16 Bureau of Economic Research, 1982; 93-120.  
17  
18
- 19 74. Farrell P, Fuchs VR. Schooling and health: the cigarette connection. *J Health Econ*  
20 1982; 1: 217-30.  
21  
22
- 23 75. Van der Pol M. Health, education and time preference. *Health Econ* 2011; 20: 917-  
24 29.  
25  
26
- 27 76. Ippolito R. Education versus savings as explanations for better health: evidence  
28 from the health and retirement survey. *George Mason Law & Economics Research*  
29 *Paper No. 03-04*. Arlington: George Mason University School of Law, 2003.  
30  
31
- 32 77. Shrier I, Platt RW. Reducing bias through directed acyclic graphs. *BMC Med Res*  
33 *Methodol* 2008; 8: 70.  
34  
35
- 36 78. D'Onofrio BM, Class QA, Rickert ME, Sujun AC, Larsson H, Kuja-Halkola R,  
37 Sjölander A, Almqvist C, Lichtenstein P, Oberg AS. Translational epidemiologic  
38 approaches to understanding the consequences of early-life exposures. *Behav Genet*  
39 2016; 46: 315-28.  
40  
41
- 42 79. Schlotter M, Schwerdt G, Woessmann L. Econometric methods for causal  
43 evaluation of education policies and practices: a non-technical guide. *Educ Econ*  
44 2011; 19: 109-37.  
45  
46
- 47 80. Newhouse JP, McClellan M. Econometrics in outcomes research: the use of  
48 instrumental variables. *Annu Rev Public Health* 1998; 19: 17-34.  
49  
50  
51  
52  
53  
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2  
3  
4 81. Greenland S. An introduction to instrumental variables for epidemiologists. *Int J Epidemiol* 2000; 29: 722-9.
- 5  
6  
7  
8  
9 82. Rassen JA, Brookhart MA, Glynn RJ, Mittleman MA, Schneeweiss S. Instrumental variables I: instrumental variables exploit natural variation in nonexperimental data to estimate causal relationships. *J Clin Epidemiol* 2009; 62: 1226-32.
- 10  
11  
12  
13  
14  
15 83. Lawlor DA, Harbord RM, Sterne JAC, Timpson N, Smith GD. Mendelian randomization: using genes as instruments for making causal inferences in epidemiology. *Stat Med* 2008; 27: 1133-63.
- 16  
17  
18  
19  
20  
21 84. Angrist JD, Imbens GW, Rubin DB. Identification of causal effects using instrumental variables. *J Am Stat Assoc* 1996; 91: 444-55.
- 22  
23  
24  
25  
26 85. Angrist JD, Pischke JS. The credibility revolution in empirical economics: how better research design is taking the con out of econometrics. *J Econ Perspect* 2010; 24: 3-30.
- 27  
28  
29  
30  
31  
32 86. Deaton A. Instruments, randomization, and learning about development. *J Econ Lit* 2010; 48: 424-55.
- 33  
34  
35  
36  
37 87. Oreopoulos P, Salvanes KG. Priceless: the nonpecuniary benefits of schooling. *J Econ Perspect* 2011; 25: 159-84.
- 38  
39  
40  
41 88. Listl S, Jürges H, Watt RG. Causal inference from observational data. *Community Dent Oral Epidemiol* 2016; 44: 409-15.
- 42  
43  
44  
45 89. Angrist JD, Pischke JS. Mostly harmless econometrics. An empiricist's companion. Princeton and Oxford: Princeton University Press, 2009; 113-218.
- 46  
47  
48  
49 90. Martens EP, Pestman WR, de Boer A, Belitser S, Klungel OH. Instrumental variables. Application and limitations. *Epidemiology* 2006; 17: 260-7.
- 50  
51  
52  
53  
54 91. Mazumder B. The effects of education on health and mortality. *Nord Econ Policy Rev* 2012; 1: 261-303.
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92. Gathmann C, Jürges H, Reinhold S. Compulsory schooling reforms, education and mortality in twentieth century Europe. *Soc Sci Med* 2015; 127: 74-82.
  93. Lleras-Muney A. The relationship between education and adult mortality in the United States. *Rev Econ Stud* 2005; 72: 189-221.
  94. Clark D, Royer H. The effect of education on adult mortality and health: evidence from Britain. *Am Econ Rev* 2013; 103: 2087-120.
  95. Van Kippersluis H, O'Donnell O, Van Doorslaer E. Long-run returns to education. Does schooling lead to an extended old age? *J Hum Resour* 2011; 46: 695-721.
  96. Fletcher JM. New evidence of the effects of education on health in the US: compulsory schooling laws revisited. *Soc Sci Med* 2015; 127: 101-7.
  97. Braakmann N. The causal relationship between education, health and health related behaviour: evidence from a natural experiment in England. *J Health Econ* 2011; 30: 753-63.
  98. Lager ACJ, Torssander J. Causal effect of education on mortality in a quasi-experiment on 1.2 million Swedes. *Proc Natl Acad Sci USA* 2013; 109: 8461-66.
  99. Arendt JN. Does education cause better health? A panel analysis using school reforms for identification. *Econ Educ Rev* 2005; 24: 149-60.
  100. Kemptner D, Jürges H, Reinhold S. Changes in compulsory schooling and the causal effect of education on health: evidence from Germany. *J Health Econ* 2011; 30: 340-54.
  101. Grytten J, Skau I, Sørensen RJ. Educated mothers, healthy infants. The impact of a school reform on the birth weight of Norwegian infants 1967-2005. *Soc Sci Med* 2014; 105: 84-92.
  102. Meghir C, Palme M. Educational reform, ability, and family background. *Am Econ Rev* 2005; 95: 414-24.



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103. Lager ACJ, Torssander J. Causal effect of education on mortality in a quasi-experiment on 1.2 million Swedes. *P Natl Acad Sci USA* 2012; 109: 8461-66.
104. Arendt JN. In sickness and in health – till education do us part: education effects on hospitalization. *Econ Educ Rev* 2007; 27: 161-72.
105. Jones AM. Identification of treatment effects in health economics. *Health Econ* 2007; 16: 1127-31.
106. Staiger D, Stock JH. Instrumental variables regression with weak instruments. *Econometrica* 1997; 65: 557-86.
107. Lindeboom M, Llena-Nozal A, van der Klaauw. Parental education and child health: evidence from a schooling reform. *J Health Econ* 2009; 28: 109-31.
108. Rehkopf DH, Glymour MM, Osypuk TL. The consistency assumption for causal inference in social epidemiology: when a rose is not a rose. *Curr Epidemiol Rep* 2016; 3: 63-71.
109. Fujiwara T, Kawachi I. Is education causally related to better health? A twin fixed-effect study in the USA. *Int J Epidemiol* 2009; 38: 1310-22.
110. Madsen M, Andersen AMN, Christensen K, Andersen PK, Osler M. Does educational status impact adult mortality in Denmark? A twin approach. *Am J Epidemiol* 2010; 172: 225-34.
111. Lundborg P. The health returns to education – what can we learn from twins? Tinbergen Institute Discussion Paper No. TI 08-027/3. Rotterdam: Tinbergen Institute, 2008. Available at: [http://papers.ssrn.com/sol3/papers.cfm?abstract\\_id=1113685](http://papers.ssrn.com/sol3/papers.cfm?abstract_id=1113685) (February 16, 2017).
112. Madsen M, Osler M. Commentary: Strengths and limitations of the discordant twin-pair design in social epidemiology. Where do we go from here? *Int J Epidemiol* 2009; 38: 1322-3.

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113. D'Onofrio BM, Lahey BB, Turkheimer E, Lichtenstein P. Critical need for family-based, quasi-experimental designs in integrating genetic and social science research. *Am J Public Health* 2013; 103 (suppl 1): S46-S55.
114. Lahey BB, D'Onofrio BM. All in the family: comparing siblings to test causal hypotheses regarding environmental influences on behavior. *Curr Dir Psychol Sci* 2010; 19: 319-23.
115. Rutter M. Proceedings from observed correlation to causal inference. *Perspect Psychol Sci* 2007; 2: 377-95.
116. Frisell T, Öberg S, Kuja-Halkola R, Sjölander A. Sibling comparison designs. Bias from non-shared confounders and measurement error. *Epidemiology* 2012; 23: 713-20.
117. Donovan SJ, Susser E. Commentary: advent for siblings designs. *Int J Epidemiol* 2011; 40: 345-9.
118. Søndergaard G, Mortensen LH, Andersen AMN, Andersen PK, Dalton SO, Madsen M, Osler M. Does shared family background influence the impact of educational differences on early mortality? *Am J Epidemiol* 2012; 176: 675-83.
119. Cook CJ, Fletcher JM. Can education rescue genetic liability for cognitive decline? *Soc Sci Med* 2015; 127: 159-70.
120. Keys KM, Smith GD, Susser E. On siblings designs. *Epidemiology* 2013; 24: 473-4.
121. Bound J, Solon G. Double trouble: on the value of twins-based estimation of the return to schooling. *Econ Educ Rev* 1999; 18: 169-82.
122. Behrman JR, Rosenzweig MR, Taubman P. Endowments and the allocation of schooling in the family and in the marriage market: the twins experiment. *J Polit Econ* 1994; 102: 1131-74.

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123. Kamin LJ. Transfusion syndrome and the heritability of IQ. *Ann Hum Genet* 1978; 42: 161-71.
  124. James WH. The IQ advantage of the heavier twin. *Br J Psychol* 1982; 73: 513-7.
  125. Lee DS, Lemieux T. Regression discontinuity designs in economics. *J Econ Lit* 2010; 48: 281-355.
  126. Zuckerman IH, Lee E, Wutoh AK, Xue Z, Stuart B. Application of regression-discontinuity analysis in pharmaceutical health services research. *Health Serv Res* 2006; 41: 550-63.
  127. Linden A, Adams JL, Roberts N. Evaluating disease management programme effectiveness: an introduction to the regression discontinuity design. *J Eval Clin Pract* 2006; 12: 124-31.
  128. McCrary J, Royer H. The effect of female education on fertility and infant health: evidence from school entry policies using exact date of birth. *Am Econ Rev* 2011; 101: 158-95.
  129. Albouy V, Lequien L. Does compulsory education lower mortality? *J Health Econ* 2009; 28: 155-68.
  130. Angrist JD, Pischke JS. *Mostly harmless econometrics. An empiricist's companion.* Princeton: Princeton University Press, 2009; 251-67.
  131. Zhong H. Does a college education cause better health and health behaviours? *Appl Econ* 2015; 47: 639-53.
  132. Cameron AC, Miller DL. A practitioner's guide to cluster-robust inference. *J Hum Resour* 2015; 50: 317-72.
  133. Angrist JD, Pischke JS. *Mostly harmless econometrics. An empiricist's companion.* Princeton: Princeton University Press, 2009; 227-33.

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60
134. Oreopoulos P. Estimating average and local average treatment effects of education when compulsory schooling laws really matter. *Am Econ Rev* 2006; 96: 152-75.
135. Helliwell JF, Putnam RD. Education and social capital. NBER Working Paper 7121. Cambridge, MA: National Bureau of Economic Research, 1999. Available at: <http://www.nber.org/papers/w7121> (February 16, 2017).
136. Eibner C, Evans WN. Relative deprivation, poor health habits and mortality. *J Hum Resour* 2005; 40: 591-620.
137. Gerdtham U-G, Johannesson M. Absolute income, relative income, income inequality, and mortality. *J Hum Resour* 2004; 39: 228-47.
138. Cattell V. Poor people, poor places, and poor health: the mediating role of social networks and social capital. *Soc Sci Med* 2001; 52: 1501-16.
139. Kaplan BH, Cassel JC, Gore S. Social support and health. *Med Care* 1977; 15: 47-58.
140. Wilkinson RG, Pickett KE. Income inequality and population health: a review and explanation of the evidence. *Soc Sci Med* 2006; 62: 1768-84.
141. Kawachi I, Subramanian AV, Almeida-Filho N. A glossary for health inequalities. *J Epidemiol Community Health* 2002; 56: 647-52.
142. Singh-Manoux A, Adler NE, Marmot MG. Subjective social status: its determinants and its association with measures of ill-health in the Whitehall II study. *Soc Sci Med* 2003; 56: 1321-33.
143. Wilkinson RG. Socioeconomic determinants of health. *Health inequalities: relative or absolute material*. *BMJ* 1997; 314: 591-9.
144. Wilkinson RG. *Unhealthy societies. The afflictions of inequality*. London: Routledge, 1996.

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60
145. Lynch JW, Smith GD, Kaplan GA, House JS. Income inequality and mortality: importance to health of individual income, psychosocial environment, or material conditions. *BMJ* 2000; 320: 1200-4.
146. Muller A. Education, income inequality, and mortality: a multiple regression analysis. *BMJ* 2002; 324: 1-4.
147. Marmot M. The influence of income on health: views of an epidemiologist. *Health Affairs* 2002; 21: 31-46.
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Captions:

Fig. 1. Percentage of the population with tertiary education among European countries during the period 1960-2005

Fig. 2. The relationship between different types of third variables and education and dental health outcome

Fig. 3. Estimation of causal effect by the use of instrumental variable

Fig. 4. Regression discontinuity design

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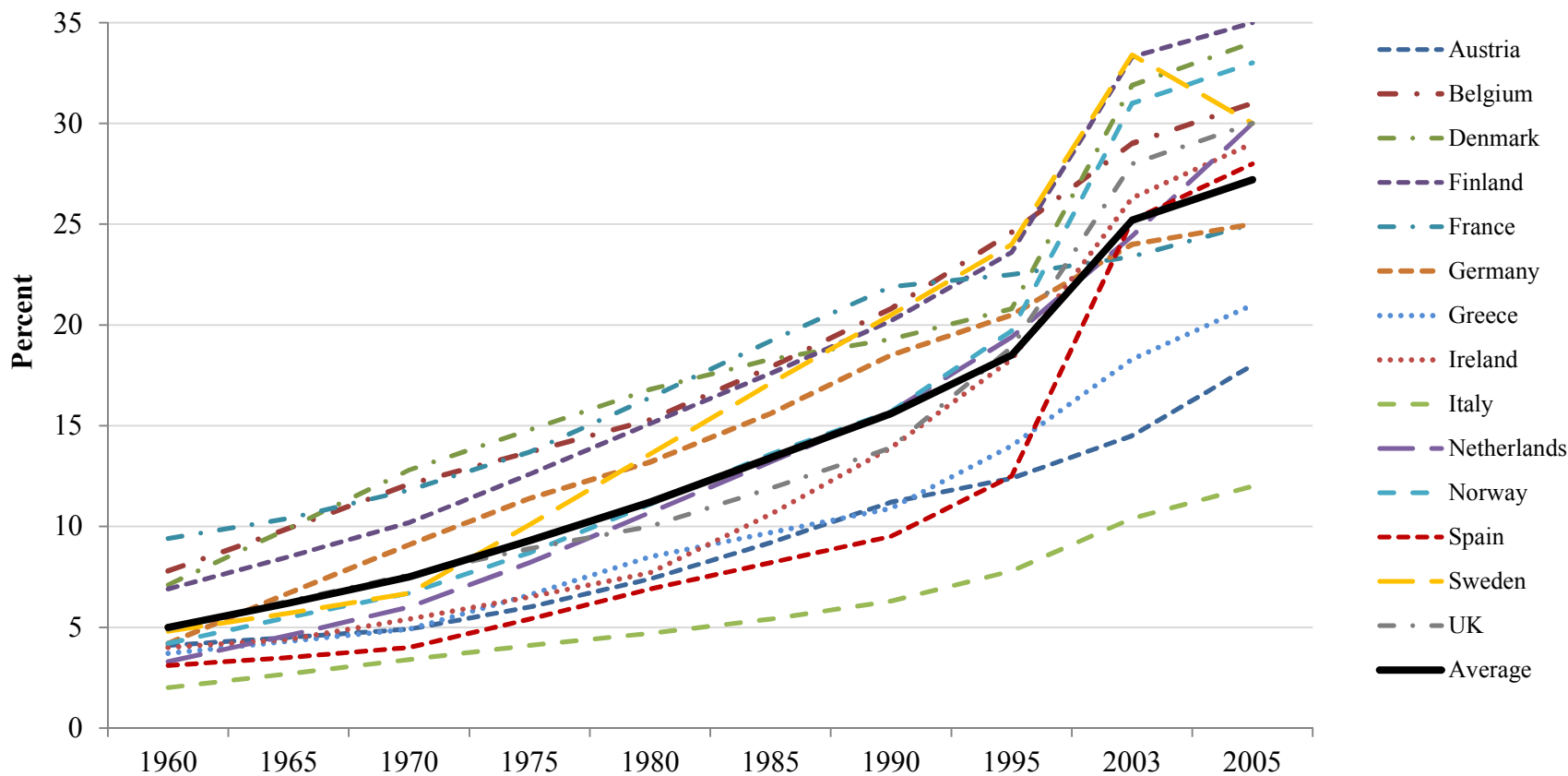


Fig. 1. Percentage of the population with tertiary education among European countries during the period 1960-2005.

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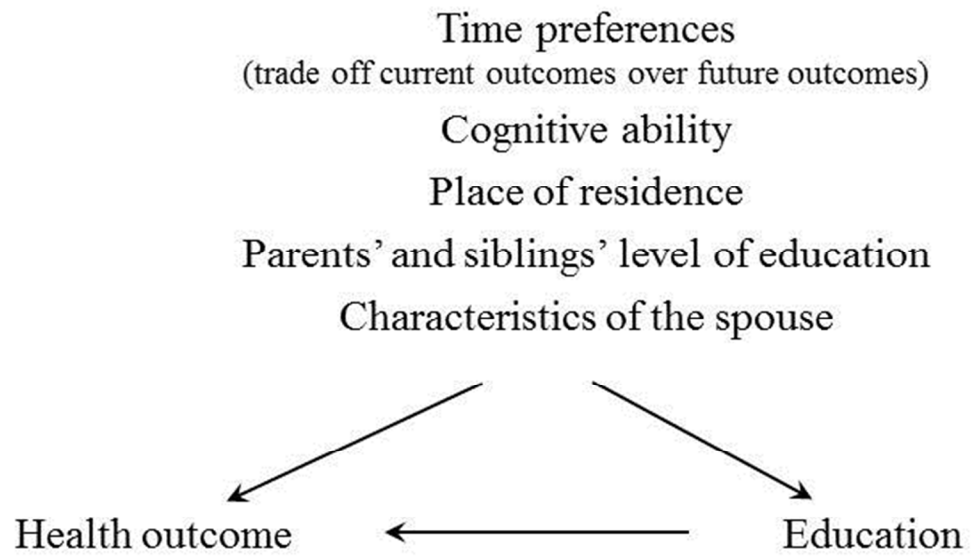


Fig. 2. The relationship between different types of third variables and education and dental health outcome



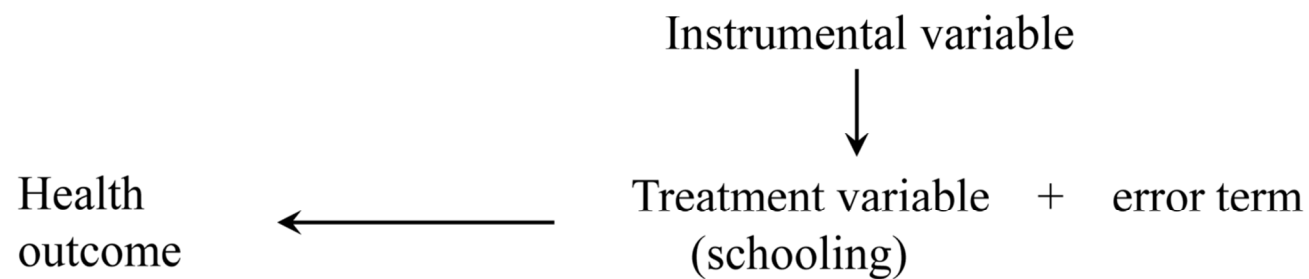


Fig. 3. Estimation of causal effect by the use of instrumental variable

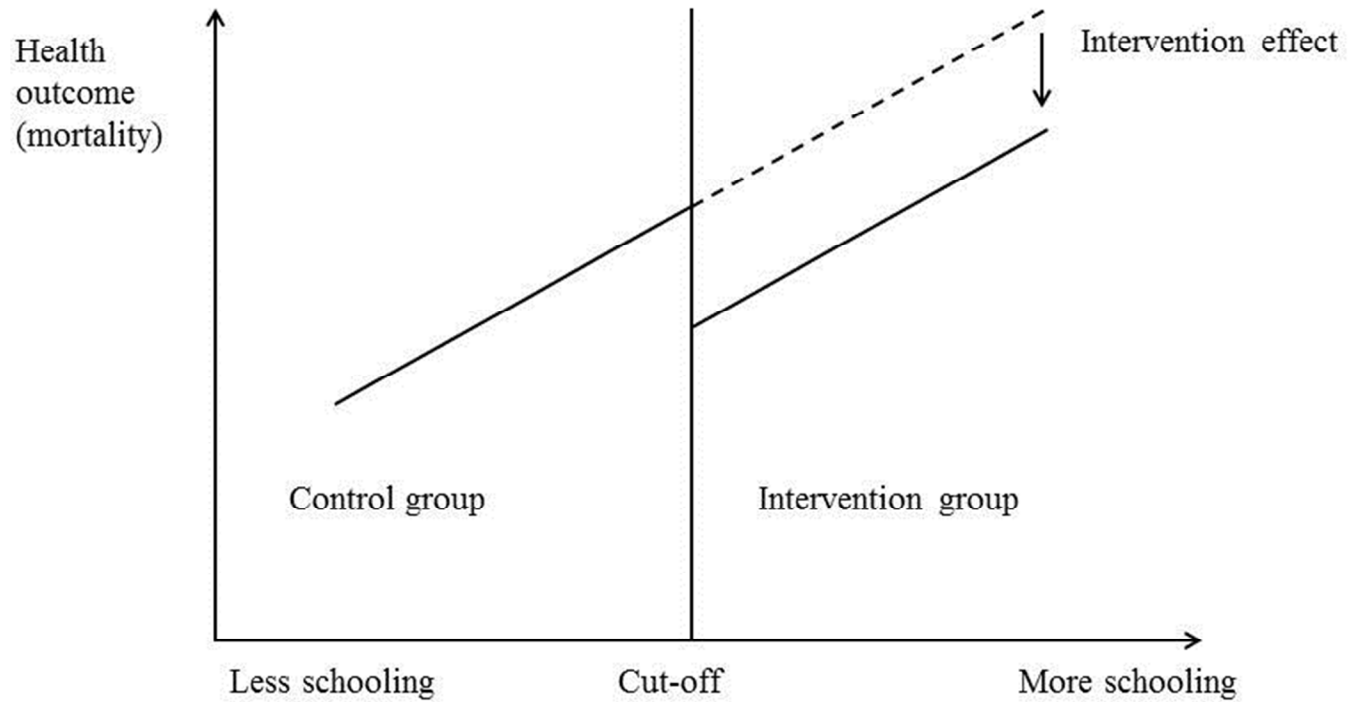


Fig. 4. Regression discontinuity design