

Klasing, Mariko Jasmin; Klasing, Mariko J.; Milionis, Petros

Conference Paper

The International Epidemiological Transition and the Education Gender Gap

Beiträge zur Jahrestagung des Vereins für Socialpolitik 2016: Demographischer Wandel -
Session: Gender, No. E16-V3

Provided in Cooperation with:

Verein für Socialpolitik / German Economic Association

Suggested Citation: Klasing, Mariko Jasmin; Klasing, Mariko J.; Milionis, Petros (2016) : The International Epidemiological Transition and the Education Gender Gap, Beiträge zur Jahrestagung des Vereins für Socialpolitik 2016: Demographischer Wandel - Session: Gender, No. E16-V3, ZBW - Deutsche Zentralbibliothek für Wirtschaftswissenschaften, Leibniz-Informationszentrum Wirtschaft, Kiel und Hamburg

This Version is available at:

<https://hdl.handle.net/10419/145771>

Standard-Nutzungsbedingungen:

Die Dokumente auf EconStor dürfen zu eigenen wissenschaftlichen Zwecken und zum Privatgebrauch gespeichert und kopiert werden.

Sie dürfen die Dokumente nicht für öffentliche oder kommerzielle Zwecke vervielfältigen, öffentlich ausstellen, öffentlich zugänglich machen, vertreiben oder anderweitig nutzen.

Sofern die Verfasser die Dokumente unter Open-Content-Lizenzen (insbesondere CC-Lizenzen) zur Verfügung gestellt haben sollten, gelten abweichend von diesen Nutzungsbedingungen die in der dort genannten Lizenz gewährten Nutzungsrechte.

Terms of use:

Documents in EconStor may be saved and copied for your personal and scholarly purposes.

You are not to copy documents for public or commercial purposes, to exhibit the documents publicly, to make them publicly available on the internet, or to distribute or otherwise use the documents in public.

If the documents have been made available under an Open Content Licence (especially Creative Commons Licences), you may exercise further usage rights as specified in the indicated licence.

The International Epidemiological Transition and the Education Gender Gap

February 2016

Abstract

We explore the impact of the international epidemiological transition on educational outcomes of males and females over the second half of the 20th century. We provide strong evidence that the large resulting declines in mortality rates from infectious diseases gave rise to differential life expectancy gains across genders, with females benefiting mostly from them due to their greater responsiveness to vaccination. We also document that these gender differences in life expectancy gains are subsequently reflected in similar differential increases in educational outcomes for males and females. Using an instrumental variables strategy that exploits pre-intervention variation in mortality rates across different infectious diseases we confirm the causal nature of these effects and show that the magnitude of the effects account for a large share of the reduction in the education gender gap that emerged over this period.

Keywords: Epidemiological Transition, Vaccination, Life Expectancy Gains, Education, Gender Differences.

JEL Classification: I15, I25, J16, O11.

1 Introduction

Do improvements in health influence economic development and if so how? Simple as it may be, this question has been the subject of an extensive debate among economists, demographers and policy makers.¹ Closely related to this debate is the analysis of the impact of the international epidemiological transition that took place after World War II and led to unprecedented improvements in life expectancy across the globe (Becker et al., 2005; Acemoglu & Johnson, 2007). This paper contributes to this debate by exploring an important dimension of the international epidemiological transition that the literature has until now largely ignored, namely the differential it had on the health of men and women.

Over the second half of the 20th century, most countries of the world experienced large decreases in mortality rates from previously highly fatal infectious diseases. These decreases were brought forth by a sequence of important medical innovations related to the development of antibiotics, new and improved vaccines, and medical techniques. These developments, products of medical research in developed countries, quickly diffused across the globe as a result of the coordinated efforts of the United Nations and World Health Organization after the end of World War II. As a consequence of this diffusion, the prevalence of and mortality from various infectious diseases, which previously had affected large shares of the world's population, was eliminated or sharply reduced.

Following a line of research pioneered by Acemoglu and Johnson (2007) we explore the impact that the epidemiological transition had on life expectancy in a sample of 75 countries over the period 1940-1980. We provide strong evidence that the large resulting declines in mortality rates from infectious diseases not only reduced average life expectancy, but also led to differential life expectancy gains across genders. In particular, we show that women benefited more than men in terms of life expectancy as a result of the diffusion of the new medical innovations over this period. We further document that these gender differences in life expectancy gains were subsequently reflected in similar differential increases in educational outcomes. We show that over the period 1940-1980 female educational attainment rose faster compared to male educational attainment in places where women experienced relatively larger increases in life expectancy.

To demonstrate the causal nature of these effects, we use an empirical setup similar to Acemoglu and Johnson (2007) and Hansen (2013). Specifically, we exploit pre-intervention variation in mortality rates from different infectious diseases to construct an instrument for the resulting mortality decreases. This approach is rationalized by the fact that mortality reductions induced by the health interventions were naturally stronger in places where disease mortality was initially higher (Bleakley, 2007). Following this approach, we confirm the existence of systematic differences in the impact of the international epidemiological transition across genders in terms

¹See Cutler et al. (2006) and Weil (2014) for recent surveys of the broad literature on this question.

of both life expectancy and educational attainment.

We then proceed to explain the nature of this gender-specific impact of the epidemiological transition by exploring gender differences in the immune system responses to vaccines. Specifically, a recent medical literature has documented that females exhibit stronger immune responses to vaccines than men (Cook, 2008; Klein et al., 2010). This implies that vaccines are bound to be more effective in providing disease protection in women compared to men. In contrast, no such differences have been so far documented for antiviral and antibacterial drugs (Franconi et al., 2007).

In light of this evidence, we assess the extent to which mortality improvements across countries were due to vaccine- and non-vaccine related medical innovations. Separating mortality improvements along these lines, we document that women experienced larger increases in life expectancy and educational attainment than men in countries where the international epidemiological transition brought forth mortality reductions in vaccine-treatable infectious diseases. We also document the absence of such differential effects in cases of countries where mortality improvements were related to diseases for which vaccines played no role or for which effective vaccines were already available and widely used before 1940.

Taking into account both the gender and the disease-specific characteristics of the international epidemiological transition allows us to explain an important share of the reduction in the education gender gap that emerged across countries over this period. Based on the estimated magnitudes for the above described effects, we are able to explain 38% of the actual life expectancy increases of women and 35% of those of men. In terms of educational outcomes this effect accounts for 26% and 22% of the observed increases in female and male education. Moreover, given that life expectancy gains as a result of vaccine-treatable diseases were 27% higher for women than for men, this effect alone accounts for most of the differential gains in life expectancy and education of women relative to men.

Our results provide strong evidence supporting the link between life expectancy and education originally proposed by Ben-Porath (1967). This link has been recently analyzed in the macroeconomic and development literature by Boucekine et al. (2002), Cervellati and Sunde (2005, 2013), Soares (2005), and De la Croix and Licandro (2012). Our findings also relate to an extensive empirical microeconomic literature analyzing how health improvements influence individual education choices. This literature includes, among others, the contributions of Soares (2006), Bleakley and Lange (2009), Jayachandran and Lleras-Muney (2009), Lucas (2010), Bhalotra and Venkataramani (2015), and Oster et al. (2013). Most closely, our analysis relates to Hansen (2013), who exploits the exogenous nature of the post-war diffusion of new medical innovations to investigate the relationship between life expectancy and educational attainment across countries. Yet, none of the aforementioned papers has considered the gender-specific nature of mortality reductions related to the international epidemiological transition and its impact on educational

choices or economic development more broadly.

The rest of the paper is organized as follows. Section 2 reviews the evidence from the medical literature on gender differences in the context of infectious diseases. Section 3 discusses the data and 4 describes the empirical strategy. Section 5 documents the results and Section 6 concludes.

2 Infectious Diseases and their Differential Impact across Genders

Men and women are different, and this difference also extends to their response to infectious diseases and to the drugs used to treat and prevent these diseases. A very recent special issue in the *Journal of Infectious Diseases* has discussed in detail sex differences in the context of infectious diseases. The general evidence of the recent epidemiological literature is that men are more susceptible to contract infectious diseases and tend to have more severe disease outcomes; at the same time, women show better response to vaccines and higher levels of vaccine efficacy compared to men (van Lunzen and Altfeld, 2014).

In the context of sex differences in disease susceptibility and disease outcomes, Bernin and Lotter (2014) document that men are more frequently and more severely affected by parasitic diseases, such as Malaria, and this effect seems to be due to sex hormones, in particular testosterone. Sex differences in the risk of contracting parasitic diseases tend to peak during middle ages, following the increase in testosterone levels. Moreover, animal models have shown that treatment of female mice with testosterone reduces their resistance to parasites and increases their disease burden by, for example, increasing mortality, anemia and weight loss in the case of malaria (Cernetitch et al., 2006), whereas castration renders male mice more resistant (Wunderlich et al., 2002; Krucken et al., 2005).

Nhamoyebonde and Leslie (2014) document that males are more frequently affected by tuberculosis than females. While a higher prevalence of certain risk factors such as more social contacts, alcoholism and smoking may explain some of the sex differences in the prevalence of tuberculosis, several studies, such as Guerra-Silveira and Abad-Franch (2013), demonstrate that these behavioral factors play a secondary role and hormonal differences seem to be the main driver. Animals models show that males are more susceptible to tuberculosis infections and develop more severe disease, while castration reduces disease severity. In addition, also studies among humans have shown that castrated males have a lower mortality rate from tuberculosis than intact males (Hamilton and Mestler, 1969) and women who underwent surgical removal of the ovaries had higher rates of tuberculosis mortality than the overall female population (Svanberg, 1981). Furthermore, there is evidence that genetic factors related to the X-chromosome may also play a role in explaining the sex bias in tuberculosis prevalence (Neyrolles and Quintana-Murci, 2009).

Muenchhoff and Gouldner (2014) review the evidence on sex differences in pediatric infectious diseases and document that males show typically higher disease susceptibility than females and have higher morbidity and mortality.² They document that for most pathogens, sex differences increase during the first year of life, coinciding with the surge of sex hormones during the so-called "mini-puberty", and then again during puberty when the variation in sex hormone levels between males and females is highest, suggesting that sex hormones play an important role. This is true for viral infections as well as bacterial and parasitic infections.³

In the context of response to vaccines, a key finding in the recent medical literature is that women respond more strongly to vaccines than men, as visible in a more robust immune response and higher antibody development, resulting in a higher level of vaccine efficacy (Poland et al., 2008a; Klein et al. 2010).⁴ This is true for both children and adults and is the result of genetic differences attributable to X-chromosome inactivation⁵ and sex hormones⁶, especially estrogens and testosterone.^{7,8} Note that this is not due to differences in vaccine dose administered to men in women. In fact, the current medical practice (still) is to universally administer the same set of vaccines to everyone on the population (Poland et al., 2008a), irrespective of sex .

In their reanalysis of data from existing vaccine studies, Klein et al. (2010) and Klein and Pekosz (2014) document that protection by yellow fever, influenza, measles, mumps, rubella, smallpox, hepatitis, and herpes vaccines is stronger for women than for men. For influenza, in fact, the authors document that women respond to half a dose of the vaccine with the antibody response equivalent to a full dose in men, while for herpes, studies have shown a vaccine efficacy level of 73% in women vs. 11% in men (Klein et al., 2010). Klein et al. (2015) also document

²See also the literature review in Giefing-Kroell, et al. (2015) that covers 24 diseases and focuses on the impact on adults. The studies reviewed by these authors report a male bias in incidences in 54% of the diseases and a male bias in mortality in 57% of the diseases. A female bias in incidences was found in 25% of the diseases and a female bias in mortality in 38% of the diseases. For the remaining 21% and 5% of the diseases respectively no significant difference between men and women were found.

³As documented by Gabriel and Arck (2014), the evidence for sex biases in influenza risk is largely unclear and likely driven by a generally very limited availability of sex-specific influenza surveillance data. For some seasonal influenza strains and some pandemics studies have reported a higher risk for males compared to females, while in other cases the opposite has been documented.

⁴Vaccine efficacy is the percentage reduction of disease in a vaccinated group of people compared to an unvaccinated group.

⁵Since females have two X-chromosomes, a maternal and a paternal one, there will be a random pattern of expression of either the maternal or the paternal chromosome in different cell populations. As a consequence, females have a more extensive repertoire of proteins and potential for greater diversity in many physiological processes (Spolarics, 2007; Fish, 2008; Libert et al., 2010).

⁶Estrogens have been shown to stimulate the activity of immune cells, while testosterone has been shown to suppress it (Fish, 2008; Klein et al., 2010; Giefing-Kroell, 2015).

⁷Given the crucial role of genes in influencing the immune response to vaccines (Poland et al., 2008b), the future of vaccinology may lie in pre-vaccination genetic screening and the introduction of personalized vaccines (Poland et al., 2008a)

⁸A stronger immune response to vaccines in women is in line with women being less susceptible to contract infectious diseases and having less severe disease outcomes. This is because sex hormones and genetic differences not only influence the functioning of immune cells in response to vaccines but also in response to naturally occurring pathogens.

such a female bias in vaccine response for tuberculosis. Furthermore, Cook (2008) in his review article documents that a female bias in the immune response to vaccines does not only exist for viral diseases but also for common bacterial diseases, such as diphtheria, tetanus and brucella.⁹ Since sex differences in immune response to vaccines are observed even prior to puberty, this suggests that genetic differences may also play a role in addition to hormonal differences (Klein and Pekosz, 2014).

As far as the response to antibiotics treatment is concerned, we have found no single study that has documented a clear sex difference in the effectiveness of antibiotics. Generally, the evidence on sex differences in drug response is very limited, due to the fact that most clinical trials either do not report sex-based information or suffer from a massive over-enrollment of men (Franconi et al., 2007). There seems to be some weak evidence that drugs acting in the central nervous system, such as antipsychotic drugs and antidepressants, are more effective in women than in men. Franconi et al. (2007) also document that beta-blockers seem to have a more beneficial effect on women compared to men in reducing the heart rate and systolic blood pressure. However, to this date, there is no evidence that drugs acting on the immune system, such as antivirals and antibiotics, have differential effectiveness in men and women. The only clear evidence from drug studies is that women generally are more prone to develop adverse reactions to drugs, such as allergic reactions (Tran et al., 1998; Franconi et al., 2007). Thus, we have no reasons to believe that the non-vaccine based health interventions, such as the introduction of antibiotics and DDT, had differential effects on female and male life expectancy. In other words, we do not expect any differential effects of initial mortality from the diseases in the 'non-vaccine' group on female and male life expectancy.

Given that, as documented above, men tend to face higher mortality from infectious diseases than women, we would generally expect that improvements in life expectancy brought about by the introduction of new drugs and other health measures should have been stronger for men than for women. On the other hand, the evidence regarding the female bias in vaccine efficacy suggests that, *ceteris paribus*, female life expectancy should have responded stronger to the vaccines introduced starting from the 1940s compared to male life expectancy. That is, for those diseases where the introduction of new or improved vaccines after 1940 was important for the control of the disease (vaccine group), we would expect to see a stronger increase in female life expectancy compared to male life expectancy. For those diseases, however, that are not vaccine-treatable or for which effective vaccines were already widely used before 1940 (non-vaccine group) we would not expect a stronger response in female life expectancy compared to male life expectancy. If at all, given the innate higher disease mortality rates for men, we would expect a stronger response in male life expectancy. Thus, provided that the enhanced vaccine response in women dominates

⁹For bacterial pneumonia, as one of the few exceptions, two of the 97 reviewed studies report a stronger immune response in men compared to women, but only among adults.

the effect coming from lower innate female disease mortality – which is plausible given that innate mortality differences documented in epidemiological studies are small compared to the differences in vaccine efficacy¹⁰ – we would expect that female life expectancy increased more compared to male life expectancy in places where initial mortality from diseases of the vaccine group was high, while in places where mortality from such diseases was low (and instead mortality from diseases in the non-vaccine group was high), we would not expect such a bias in favor of females. There, the differential effect for men and women should have been either be zero or (slightly) male-biased.

3 Data

Our sample includes 75 countries, which are listed in Appendix A, and covers the years 1940 and 1980.¹¹ That is, we focus on the era before the arrival of HIV/AIDS. To test for the differential effect of reductions in disease mortality (driven by medical and related technological innovations and other health policy measures) on female and male life expectancy and education, we need data on disease-specific mortality in the year 1940, and life expectancy data and data on education in 1940 and 1980, both differentiated by sex. Furthermore, we need data on additional control variables, which will be discussed later as they are introduced in the estimation.

For mortality from infectious diseases in 1940, we use the data provided by, Acemoglu and Johnson (2007), henceforth AJ, and group the diseases in their data set into a vaccine-group and a non-vaccine group. AJ report data for 13 infectious diseases. Seven of these fall into the vaccine group (diphtheria, influenza, measles, pneumonia, smallpox, tuberculosis, whooping cough) and six fall into the non-vaccine group (cholera, malaria, plague, scarlet fever, typhoid fever, typhus). See Appendix B for details on the characteristics and key methods of control of these diseases.

Unfortunately, the disease mortality data are not differentiated by sex, but we only have information on the average mortality rates in the population. We hence have to assume that initial (1940) mortality from these diseases was the same for men and women in a given country. This is not a problem, though, as this measurement error would simply bias the female-specific effect of disease eradication on life expectancy toward zero. To give an intuition for this, suppose for simplicity that female and male initial disease mortality differ by a factor of 20%. This would imply that the average level of disease mortality – the one we observe in the data – is 10% higher than actual female mortality. In other words, we overestimate female disease mortality in 1940 by 10%. If we now regressed 1940 disease mortality on changes in female life expectancy between

¹⁰For example, the large majority of studies on pneumonia reviewed by Falagas et al. (2007) document a ratio of male to female mortality rates ranging between 1.12 and 1.19. In contrast, Klein et al. (2010) document that protective antibody response after vaccination can be twice as high in females compared to males of all ages.

¹¹We focus on this long time horizon because mortality reductions will take a long time to affect life expectancy in the population and schooling outcomes.

1940 and 1980, the estimated coefficient, which is presumably positive, would be 10% lower than if we had regressed the life expectancy changes on actual 1940 female disease mortality. Since we are interested in whether there is a positive female bias in life expectancy improvements in response to the mortality reductions from the diseases of the vaccine-group vs. the non-vaccine group, the measurement error would go against us finding such an effect.

For life expectancy data in 1940, we started off with the information provided in Appendix C of AJ, where the authors document sex-specific information on life expectancy at birth for the 47 countries in their baseline sample of 47 countries. For the remaining 28 countries for which disease mortality data in 1940 are available in AJ’s data set we supplemented the life expectancy data with the information provided in the 1948, 1951, 1957, 1961 and 1967 editions of the UN Demographic Yearbook and Preston (1975), Table A.2. In many cases, data for exactly the year 1940 were not available. In these cases, we used the data from the closest year before and after 1940 and linearly interpolated between the two data points to produce an estimate for 1940. If for one of the years there was no sex-specific information but only information for both sexes combined, we calculated the ratio of male and female life expectancy relative to life expectancy in the total population in the next year with available data (this is typically the year 1952) and used this ratio to produce corresponding sex-specific estimates for the missing year. For 1980, we use the electronic World Population Prospects Revision 15 data of the UN Population Division.¹² Our main estimations are based on life expectancy at birth, but we also include life expectancy at age 20 in some robustness tests. The data for life expectancy at age 20 stem from the same sources as the life expectancy at birth data.

For education, we follow Hansen (2013) and use the average schooling years of the cohort of individuals that was 5-9 years old in the respective years (1940, 1980), as observed in the Barro and Lee (2013) data set 10 years later (1950, 1990).¹³ This is the group of individuals that entered the formal schooling system between 1937 and 1941 and 1977 and 1981, i.e. around the two sample years, respectively. Thus, in order to assess the impact of life expectancy improvements on human capital formation, we compare the level of educational attainment of an age cohort that started school around 1980 when life expectancy had already improved with that of an age cohort that started school before the onset of the international epidemiological transition and the resulting life expectancy increases.

Table 1 shows some descriptive statistics of the key variables.

[Table 1 around here]

¹²These data are available at <http://esa.un.org/unpd/wpp/>.

¹³Since tertiary education was extremely rare in 1940, most people had completed their education by the age of 15-19. This the cohort whose schooling data we observe in the Barro-Lee data set in 1950 and which corresponds to the population that was 5-9 years old in 1940. To be consistent, we therefore also measure the schooling years of people who entered the schooling system around the year 1980 based on the schooling data of the age group 15-19 in 1990.

4 Estimation Strategy

The first hypothesis we seek to test is whether countries in which initial disease mortality was high experienced larger increases in life expectancy over the following 40 years than countries where initial diseases mortality was low and whether these impacts on life expectancy differed for men and women. We are particularly interested in whether such differences exist for vaccine-treatable diseases, but not for diseases for whose cure vaccines played no role. Subsequently, we seek to test whether the predicted sex-specific changes in life expectancy can explain the observed changes in sex-specific years of schooling in the form of an IV regression.

The first stage of the IV takes the form:

$$LE_{sct} = \beta_1 \sum_{d \in V} (M_{cd} \times I_{1980}) + \beta_{1f} \cdot fem \cdot \sum_{d \in V} (M_{cd} \times I_{1980}) \\ + \beta_2 \sum_{d \in NV} (M_{cd} \times I_{1980}) + \beta_{2f} \cdot fem \cdot \sum_{d \in NV} (M_{cd} \times I_{1980}) + \beta_3 \cdot fem + \delta_c + \tau_t + \varepsilon_{sct},$$

with s, c and t indexing sex (male, female), country and time (1940, 1980) respectively and LE denoting life expectancy at birth. M_d is the initial (i.e. 1940) mortality rate from disease d and diseases are classified into a vaccine group V (diphtheria, influenza, measles, pneumonia, smallpox, tuberculosis, whooping cough) and a non-vaccine group NV (cholera, malaria, plague, scarlet fever, typhoid fever, typhus). I_{1980} is an indicator variable that equals one in 1980, fem is a dummy variable for females, and δ_c and τ_t indicate the country and time fixed effects respectively. ε_{sct} is the error term.

The main regression has the form:

$$School_{sct} = LE_{sct} + \gamma \cdot fem + \delta_c + \tau_t + \mu_{sct}.$$

In case where life expectancy is instrumented with 1940 disease mortality, LE_{sct} is replaced with the predicted level of life expectancy derived from the above first-stage regression.

Note that by construction $\sum_{d \in V} (M_{cd} \times I_{1980})$ and $\sum_{d \in NV} (M_{cd} \times I_{1980})$ are positive in 1980 and zero in 1940. In other words, larger changes in these terms between 1940 and 1980 indicate greater implied reductions in disease mortality. In light of this and the discussion above on sex biases in the immune response to vaccines, we expect $\beta_1, \beta_{1f}, \beta_2 > 0, \beta_{2f} = 0$ and $\gamma > 0$.

5 Regression Results

5.1 Mortality Reductions and Life Expectancy Gains

Table 2 shows the results of the first-stage regression with life expectancy at birth, separated by sex, as the dependent variable. With 75 countries, two sample periods, and two sexes, our total sample size is $75 \cdot 2 \cdot 2 = 300$.

[Table 2 around here]

In col. 1 we first look at the aggregate effects of mortality reductions on life expectancy by summing up the mortalities across all diseases ignoring possible differential effects for men and women. The regression results document that the health interventions increased life expectancy by about 7 years between 1940 and 1980, in line with Hansen (2013).

To separate the effect across sexes, in col. 2 we include the female dummy variable and an interaction term between overall disease mortality and the female dummy. The coefficient on the interaction term is positive and significant at the 5% level, indicating that, on average, female life expectancy rose faster between 1940 and 1980 than male life expectancy. Specifically, the coefficient of 2.8 implies that, compared to men, female life expectancy increased by an extra 1.2 years. In addition, we see that on average across both periods, life expectancy of females exceeded that of males by 3.6 years.

In col. 3 we assess the differential effects of mortality from the diseases in the vaccine group and the non-vaccine group. As we can see, the 1940 mortality rates in both groups were associated with a significant increase in life expectancy. However, while the interaction term between the female dummy and disease mortality in the vaccine-group is significantly positive, the interaction term between the female dummy and disease mortality in the non-vaccine group is insignificant. This indicates, as suggested by the recent medical literature, that female life expectancy responded stronger to the health interventions compared to male life expectancy, but only in those countries that in 1940 were primarily affected by diseases that became treatable with vaccines. For countries, though, in which the main cause of high mortality were diseases that became curable thanks to antibiotics or DDT (as in the case of malaria), we do not observe any sex differences in the responses of life expectancy. The estimated coefficients imply that, *ceteris paribus*, as a consequence of mortality reductions in vaccine-treatable diseases male life expectancy rose on average by 4.5 years and female life expectancy by 5.7 year, implying an added effect for females of 27%. For the non-vaccine group of diseases we find that the health interventions increased life expectancy for both sexes on average by 1.3 years.

In col. 4, 5, and 6 we analyze the three most important infectious diseases, malaria, pneumonia, and tuberculosis, separately, which together killed on average 38% of the population in 1940 and were responsible for 87% of the deaths from the 13 diseases. In all three cases, we control

for mortality in the residual group of diseases. That is, we control for overall disease mortality after having subtracted mortality from the respective disease of interest.

The patterns we find confirm our earlier results: For tuberculosis we see that the interaction term between disease mortality and the female dummy is positive and significant, while for malaria the interaction term is insignificant. Since tuberculosis is one of the diseases for which the key health intervention was the introduction of a vaccine, while malaria was primarily controlled by newly developed insecticides (DDT), the results are in line with our predictions.

For pneumonia, the coefficient on the female interaction term is also positive, but its significance level is just outside of the commonly acceptable range ($p = 0.113$). A plausible explanation for this relatively weaker female-specific effect is that the vaccine against *Streptococcus pneumoniae* – the most common cause of pneumonia infections – which became available in 1945, was hardly used as its introduction coincided with the discovery of penicillin. It was not until the late 1970s and early 1980s, i.e. after the end of our sample period, that the vaccine was substantially improved and became more widely used. Influenza vaccines introduced in the 1940s are effective against pneumonia caused by influenza virus strains, but these infections form only a small part of all pneumonia cases (Ruuskanen et al., 2011). Given this, we would expect some, but not very strong differences between the responses in male and female life expectancy.

The estimated coefficients in the case of the three big killers imply that the health interventions increased male and female life expectancy by 1 year in the case of malaria; for pneumonia, the associated life expectancy increases are 2.86 years for men and 3.7 years for women; for tuberculosis, the estimated coefficients imply a life expectancy increase of 1 year for women, while for men the effect is insignificant.

One could argue that the insignificance of the female interaction effect for the non-vaccine group of diseases could be due to the very small level of mortality from these diseases compared to the diseases in the vaccine group: Since the two biggest killers are part of the vaccine group, mortality from this group of diseases was much higher in 1940 than mortality from the non-vaccine group of diseases (see Table 1). To rule out that this is what is causing the insignificance of the interaction term, in col. 7 we focus instead on the smaller diseases, excluding the above three big killers. For these diseases, the mortality rates are much smaller and the differences in mortality rates between the two groups of diseases are also very small. The results demonstrate that the previously found effects are robust to this modification: Also for the small diseases we find that for those falling into the vaccine group, the health interventions had a stronger impact on female life expectancy than on male life expectancy, while for the non-vaccine group we find no significant differences between the impact on men and women. The estimated coefficients imply that the health interventions led to an increase of 0.7 years of life for men and women for diseases in the non-vaccine group, while for the diseases in the vaccine group they led to 2 years of extra life for men and 3.2 years of extra life for women.

To show the relationship between the implied mortality reductions and life expectancy increases also visually, we present in Figures 1a and 1b sex-specific conditional scatter plots of the relationship between changes in mortality and changes in life expectancy over the period 1940-1980. That is, we visualize the relationships underlying the estimations in col. 2 (Fig. 1a) and col. 3 (Fig. 1b), omitting in the latter the non-vaccine case for which we did not find any significant differences between men and women. In both cases, the relationship for men is plotted in panel A and that for women in panel B. As the figures demonstrate, the estimated relationships hold well over the whole range of data points and are not driven by few observations. Furthermore, the figures clearly demonstrate, just like the above regression results, that the same underlying reductions in disease mortality – larger values in the graph indicate larger implied falls in mortality – are associated with stronger life expectancy gains for women compared to men.

5.2 Life Expectancy Gains and Educational Attainment

Turning now to the impact the life expectancy improvements over the period 1940-1980 had on human capital formation over the same period, we first present in col. 1 of Table 3 a simple OLS regression of years of schooling of the age group 5-9 in the respective years on life expectancy. In line with Hansen, we find that improvements in life expectancy are positively related to gains in schooling.

[Table 3 around here]

In col. 2, we allow the effect of life expectancy to differ for men and women. We find no evidence that such a differential effect is present. In other words, given increases in life expectancy had the same effects on female and male education. In addition, we find, not surprisingly, that women have on average less education than men, the difference being about 0.6 years of schooling.

In col. 3, 4 and 5 we present the corresponding results based on IV regressions. In col. 2 the first stage is based on the results displayed in col. 2 of Table 2 (overall diseases mortality), while col. 3 and 4 use the first stage results shown col. 3 of Table 2. In other words, in these columns actual life expectancy is replaced with predicted life expectancy and the interaction term $Female * LE$ is replaced with the predicted level of life expectancy multiplied with the female dummy, with the standard errors corrected accordingly.

Both regressions confirm the previous OLS results: Life expectancy improvements led to an increase in schooling, but given increases in life expectancy had the same effect on male and female education. However, since the underlying predicted increases in life expectancy differ for men and women, our estimates imply, in correspondence with the above first-stage regression results, that in countries that were initially primarily affected by diseases of the vaccine-group, female education rose, holding everything else constant, faster than male education. Specifically, given

that the coefficient estimates in col. 2 of Table 2 implied that between 1940 and 1980 male life expectancy rose on average by 6.1 years and female life expectancy by 7.6 as a consequence of the variation in initial disease mortality alone, these life expectancy gains together with the estimated coefficient of 0.117 in col. 3 imply that the health interventions increased male schooling by 0.7 years and female schooling by 0.9 years.

Focusing on mortality from the diseases in the vaccine group, the life expectancy improvements resulting from the health interventions together with the estimated coefficient of 0.114 in col. 3 imply that the development of new and improved vaccines alone raised male schooling by 0.5 years and female schooling by 0.65 years. This accounts for 16% of the actual male schooling increases and for 19% of the actual female schooling increases over this period.

5.3 Robustness Tests

In the following, we assess in Table 4 the robustness of our key estimation shown in col. 4 of Table 3, which we replicate for ease of comparison in col. 1 of Table 4. We first modify the set of countries included in the sample, focusing only on the 47 countries that were included in AJ's baseline sample. Second, we include other variables that may be potentially correlated with either life expectancy or education. Third, we check whether the estimated positive relationship between schooling and life expectancy is driven by common underlying time-trends in the schooling and life expectancy data.

[Table 4 around here]

Col. 2 of Table 4 shows that focusing on the 47 countries used by AJ in their main estimations does not affect our previous results. Both the first stage of the estimation and the second stage are very similar to our original results.

In col. 3, we include the log of GDP per capita, taken from the 2009 version of Maddison's data set.¹⁴ In col. 4, we control for the quality of institutions, measured by the constraints on the executive reported in the Polity IV database, averaged over the period 1950-1970 and interacted with a 1980 year dummy.¹⁵ In col. 5, we simultaneously include both these variables. We find that the inclusion of these variables does not affect our previous results. The estimated effect of life expectancy on years of schooling, when instrumented with the initial disease mortality rates and their interactions with the female dummy, remain significant at the 1% level. In fact, the point estimate increases compared to what we documented in Table 2, which is partially driven by the reduction in sample size.

¹⁴See <http://www.ggdnet.net/maddison/oriindex.htm>.

¹⁵Using a mid-point estimate for the period 1950-1970 rather than year-specific data is driven by the fact that in 1940, in the mids of World War II, many countries were in a state of state failure with an absent political system (Polity IV codes -66, -77, and -88).

In col. 6, we control for life expectancy at age 20. This is motivated by the idea that the health interventions and the resulting disease mortality reductions are likely also correlated with the life expectancy gains of adults and it may be that this is the primary driver behind the rise in education. As the results clearly show, this is not the case. Improvements in life expectancy at age 20 are not significantly related to the observed increases in schooling.¹⁶

It is possible that there are pre-existing trends in the schooling data that drive their above estimated relationship with life expectancy, but which are in fact unrelated to the life expectancy improvements triggered by the post-1940 health interventions. To assess this, we employ two tests. First, we include in col. 7 a 40-year lag of the schooling data.¹⁷ While one should be cautious with interpreting the estimated coefficients because the lagged dependent variable may be correlated with the error term of the regression (Nickell, 1981), the clear insignificance of lagged schooling and the fact that the estimated coefficient on life expectancy is almost identical to the one found in col. 1 makes us confident that trends in the schooling data are not driving the results.

As a second test, we replace in col. 8 our dependent variable – years of schooling of the age group 5-9 in the respective sample years – with lagged years of schooling (i.e. years of schooling of the same age groups in 1900 and 1940). If health improvements driven by the post-1940 health interventions can explain the rise in schooling between 1900 and 1940, this would indicate that the previously estimated relationship is spurious and the post-1940 rise in human capital could have occurred even in the absence of the medical advances. We find that the rise in life expectancy brought forward by the post-1940 medical advances are uncorrelated with the rise in schooling observed over the previous 40-year period. This also makes us confident that pre-existing trends in the schooling data are not driving the effects.

As a last test, we conduct in col. 9 a falsification exercise and examine the impact of improvements in life expectancy between 1940 and 1980 on education levels of the cohort of individuals that were of age 60-64 in the respective years.¹⁸ Since most people who were 60-64 years old in 1980 completed their formal education before 1940, life expectancy improvements that occurred after 1940 (which may or may not have influenced the survival rates of this age group as well) should not have influenced their education choices. In other words, the changes in education lev-

¹⁶Here, we instrument, as before, for life expectancy at birth with the disease mortality data and their interaction terms with the female dummy and simply include life expectancy at age 20 as an (exogenous) control variable. Alternatively, we could instrument for life expectancy at age 20 with the proposed mortality instruments and include life expectancy at birth as a simple control variable. The results are qualitatively the same, with life expectancy at age 20 being unrelated to years of schooling and life expectancy at birth having a significant positive relationship with the latter. In this set-up, though, the first stage regression fails in the sense that the correlation between the disease mortality instruments and life expectancy at age 20 is not strong enough to pass the commonly applied threshold values for the F-statistic.

¹⁷People who were 5-9 years old in 1900 were 55-59 years old in 1950. Their schooling can hence be observed in the Barro-Lee dataset.

¹⁸As before, we observe their schooling data in the Barro-Lee dataset 10 years later, i.e. in 1950 and 1990 respectively.

els between 1940 and 1980 for this group of older people should be unrelated to life expectancy improvements over this period and hence unrelated to the medical advances made after 1940. As we can see, when replacing our original dependent variable with the years of schooling of the age group 60-64, the estimated relationship between life expectancy, instrumented by the disease mortality data, and schooling is no longer positive. In fact, we find it to be negative, indicating that the factors that positively affected the schooling decisions of older generations correlate positively with disease mortality in 1940 and hence negatively with the subsequent life expectancy improvements.

One possible explanation is that this effect is driven by the sharp reductions in life expectancy that occurred around 1918-1920 during the 'Spanish flu' influenza pandemic, which affected about 35% and killed about 3% to 6% of the world's population (Taufenberger and Morens, 2006). Life expectancy data around this period, which are available in the Human Mortality Database for selected countries¹⁹, show that on average life expectancy at birth declined by 9.8 years between 1917 and 1918 and rose again by 13.5 years between 1918 and 1921.²⁰ Since people who were 60-64 years old in 1980 were born around the time of the Spanish flu, it is likely that their upbringing and their education were influenced by this decline in life expectancy. Therefore, if mortality rates during the 1918 influenza pandemic were positively correlated with influenza mortality in 1940 – which is plausible – this would create a negative correlation between the changes in life expectancy of those born around 1880 and 1920 (the 60-64 year-olds in 1940 and 1980) and changes in life expectancy between 1940 and 1980.

6 Conclusion

This paper explores the gender-specific nature of the medical advances related to the international epidemiological transition in order to analyze the link between life expectancy and educational attainment. Specifically, we demonstrate that the large mortality reductions from infectious diseases that took place across the globe during the second half of 20th century had a differential impact on female relative to male life expectancy, and that this in turn resulted in differential increases in female and male educational attainment. We argue that the origin behind these gender-specific effects lies in the fact that females exhibit stronger immune responses to vaccines than men. As a result of that, females benefited more from the international diffusion of new vaccines against infectious diseases such as tuberculosis and influenza, they gained more in terms of life expectancy compared to men and these gains allowed them to invest more in education.

Overall, these results provide a strong confirmation for the positive association between life ex-

¹⁹The data are available at <http://www.mortality.org>. The countries with available data for this period are Denmark, Finland, France, Iceland, Italy, Netherlands, Norway, Spain, Switzerland, and Sweden.

²⁰Data from Denmark are excluded in this calculation as it is the only country among the ten that seemingly was spared by the Spanish flu.

pectancy and educational attainment, proposed first by Ben-Porath (1967) and analyzed further in recent contributions in the health and development economics literature. Our contribution, though, is unique in this context as it exploits exogenous variation across genders and diseases due to the international epidemiological transition in order to estimate the causal effect of life expectancy on educational attainment. The gender-specificity of the effect can also shed light on other important questions related to the international epidemiological transition, such as why the resulting increases in educational attainment have not clearly been reflected in aggregate economic outcomes, as shown by Acemoglu and Johnson (2007). This is something that we plan to explore in subsequent research.

References

- ACEMOGLU, D., AND S. JOHNSON (2007): “Disease and Development: The Effect of Life Expectancy on Economic Growth,” *Journal of Political Economy*, 115(6), 925–985.
- BAKER, J., AND S. KATZ (2004): “Childhood Vaccine Development: An Overview,” *Pediatric Research*, 55(2), 347–356.
- BARRO, R., AND J. LEE (2013): “A New Data Set of Educational Attainment in the World, 1950–2010,” *Journal of Development Economics*, 103, 184–198.
- BECKER, G., T. PHILIPSON, AND R. SOARES (2005): “The Quantity and Quality of Life and the Evolution of World Inequality,” *American Economic Review*, 95, 277–291.
- BEN-PORATH, Y. (1967): “The Production of Human Capital in the Life Cycle of Earnings,” *Journal of Political Economy*, 75, 352–365.
- BERNIN, H., AND H. LOTTER (2014): “Sex Bias in the Outcome of Human Tropical Infectious Diseases: Influence of Steroid Hormones,” *Journal of Infectious Diseases*, 209(Suppl 3), S107–S113.
- BHALOTRA, S., AND A. VENKATARAMANI (2015): “Shadows of the Captain of the Men of Death: Health Innovation, Human Capital Investment, and Institutions,” Working Paper, University of Essex.
- BLEAKLEY, H. (2007): “Disease and Development: Evidence from Hookworm Eradication in the American South,” *Quarterly Journal of Economics*, 122(1), 71–117.
- BLEAKLEY, H., AND F. LANGE (2009): “Chronic Disease Burden and the Interaction of Education, Fertility, and Growth,” *Review of Economics and Statistics*, 91(1), 52–55.
- BONAH, C. (2005): “The Experimental stability of the BCG vaccine: safety, efficacy, proof, and standards, 1921–1933,” *Studies in History and Philosophy of Biological and Biomedical Sciences*, 36, 696–721.
- BOUCEKKINE, R., D. DE LA CROIX, AND O. LICANDRO (2002): “Vintage Human Capital, Demographic Trends and Endogenous Growth,” *Journal of Economic Theory*, 104, 340–375.
- CERNETICH, A., L. GARVER, AND A. JEDLIČKA (2006): “Involvement of gonadal steroids and gamma interferon in sex differences in response to bloodstage malaria infection,” *Infection and Immunity*, 74(3), 190–203.
- CERVELLATI, M., AND U. SUNDE (2005): “Human Capital, Life Expectancy and the Process of Development,” *American Economic Review*, 95(5), 1653–1672.
- (2013): “Life Expectancy, Schooling, and Lifetime Labor Supply: Theory and Evidence Revisited,” *Econometrica*, 81(5), 2055–2086.
- COMSTOCK, G. (1994): “The International Tuberculosis Campaign: A Pioneering Venture in Mass Vaccination and Research,” *Clinical Infectious Diseases*, 19(3), 528–540.

- COOK, F. (2008): “Sexual dimorphism of humoral immunity with human vaccines,” *Vaccine*, 26, 3551–3555.
- CUTLER, D., A. DEATON, AND A. LLERAS-MUNIEY (2006): “The Determinants of Mortality,” *Journal of Economic Perspectives*, 20, 97–120.
- DE LA CROIX, D., AND O. LICANDRO (2012): “The Child is the Father of the Man: Implications for the Demographic Transition,” *Economic Journal*, 123(567), 236–261.
- FALAGAS, M., E. MOURTZOUKOU, AND K. VARDAKAS (2007): “Sex differences in the incidence and severity of respiratory tract infections,” *Respiratory Medicine*, 101, 1845–1863.
- FENNER, F. (1996): “Smallpox eradication: the vindication of Jenner’s prophesy,” in *Vaccinia, Vaccination, Vaccinology: Jenner, Pasteur and their successors*, ed. by S. Plotkin, and B. Fantini. Elsevier, Paris.
- FISH, E. (2008): “The X-files in immunity: sex-based differences predispose immune responses,” *Nature Reviews Immunology*, 8, 737–744.
- FRANCONI, F., S. BRUNELLESCHI, L. STEARDO, AND V. CUOMO (2007): “Gender differences in drug responses,” *Pharmacological Research*, 55, 81–95.
- GABRIEL, G., AND P. ARCK (2014): “Sex, Immunity and Influenza,” *Journal of Infectious Diseases*, 209(Suppl 3), S93–S99.
- GIEFING-KROELL, C., P. BERGER, G. LEPPERDINGER, AND B. GRUBECK-LOEWENSTEIN (2015): “How sex and age affect immune responses, susceptibility to infections, and response to vaccination,” *Aging Cell*, 14, 309–321.
- GRANSTROEM, M. (1996): “The history of pertussis vaccination: from whole-cell to subunit vaccines,” in *Vaccinia, Vaccination, Vaccinology: Jenner, Pasteur and their successors*, ed. by S. Plotkin, and B. Fantini. Elsevier, Paris.
- GUERRA-SILVEIRA, F., AND F. ABAD-FRANCH (2013): “Sexual Inequality in Tuberculosis,” *PLoS ONE*, 8(4), 1–13.
- HAMILTON, J., AND G. MESTLER (1969): “Mortality and survival: comparison of eunuchs with intact men and women in a mentally retarded population,” *Journal of Gerontology*, 24, 395–411.
- HANSEN, C. (2013): “Disease and Development: The Effect of Life Expectancy on Economic Growth,” *Journal of Health Economics*, 32, 1142–1152.
- HOYT, K. (2006): “Vaccine Innovation: Lessons from World War II,” *Journal of Public Health Policy*, 27(1), 38–57.
- JAYACHANDRAN, S., AND A. LLERAS-MUNIEY (2009): “Life Expectancy and Human Capital Investments: Evidence from Maternal Mortality Declines,” *Quarterly Journal of Economics*, 124(1), 349–397.
- KLEIN, J., AND S. PLOTKIN (2007): “Robert Austrian: 1917–2007,” *Clinical Infectious Diseases*, 45, 2–3.

- KLEIN, S., A. JEDLICKA, AND A. PEKOSZ (2010): “The Xs and Y of immune responses to viral vaccines,” *The Lancet*, 10, 338–349.
- KLEIN, S., I. MARRIOTT, AND E. FISH (2015): “Sex-based differences in immune function and responses to vaccination,” *Transactions of the Royal Society of Tropical Medicine & Hygiene*, 109(1), 9–15.
- KLEIN, S., AND A. PEKOSZ (2014): “Sex-based Biology and the Rational Design of Influenza Vaccination Strategies,” *Journal of Infectious Diseases*, 209(Suppl 3), S114–S119.
- KRUCKEN, J., M. DKHIL, J. BRAUN, R. SCHROETEL, M. EL-KHADRAGY, P. CARMELIET, H. MOSSMANN, AND F. WUNDERLICH (2005): “Testosterone suppresses protective responses of the liver to blood-stage malaria.,” *Infection and Immunity*, 73(1), 436–443.
- LIBERT, C., L. DEJAGER, AND I. PINHEIRO (2010): “The X chromosome in immune functions: when a chromosome makes the difference,” *Nature Reviews Immunology*, 10, 594–604.
- LINDENMANN, J. (2002): “Typhus Vaccine Developments from the First to the Second World War (On Paul Weindling’s ‘Between Bacteriology and Virology...’),” *History and Philosophy of the Life Sciences*, 24(3/4), 467–485.
- LUCAS, A. (2010): “Maladia Eradication and Educational Attainment: Evidence from Paraguay and Sri Lanka,” *American Economic Journal: Applied Economics*, 2(2), 46–71.
- MUENCHHOFF, M., AND P. GOULDNER (2014): “Sex Differences in Pediatric Infectious Diseases,” *Journal of Infectious Diseases*, 209(Suppl 3), S120–S126.
- NEYROLLES, O., AND L. QUINTANA-MURCI (2009): “Sexual Inequality in Tuberculosis,” *PLoS Medicine*, 6(12), 1–6.
- NHAMOYEBONDE, S., AND A. LESLIE (2014): “Biological Differences Between the Sexes and Susceptibility to Tuberculosis,” *Journal of Infectious Diseases*, 209(Suppl 3), S100–S106.
- NICKELL, S. (1981): “Biases in Dynamic Models with Fixed Effects,” *Econometrica*, 49(6), 1417–1426.
- OSTER, E., I. SHOULSON, AND R. DORSEY (2013): “Limited Life Expectancy, Human Capital and Health Investments: Evidence from Huntington Disease,” *American Economic Review*, 103(5), 1977–2002.
- PARISH, H. (1965): *A History of Immunization*. E. & S. Livingstone Ltd., London.
- POLAND, G., I. OVSYANNIKOVA, AND R. JACOBSON (2008): “Personalized vaccines: the emerging field of vaccinomics,” *Expert Opinion on Biological Therapy*, 8(11), 1659–1667.
- PRESTON, S. (1975): “The Changing Relation between Mortality and Level of Economic Development,” *Population Studies*, 29(2), 231–248.
- RUUSKANEN, O., E. LAHTI, L. JENNINGS, AND D. MURDOCH (2011): “Viral pneumonia,” *The Lancet*, 377, 1264–1275.

- SOARES, R. (2005): “Mortality Reductions, Educational Attainment, and Fertility Choice,” *American Economic Review*, 95(3), 580–601.
- (2006): “The Effect of Longevity on Schooling and Fertility: Evidence from the Brazilian Demographic and Health Survey,” *Journal of Population Economics*, 19(1), 71–97.
- SPOLARICS, Z. (2007): “The X-files of inflammation: cellular mosaicism of X-linked polymorphic genes and the female advantage in the host response to injury and infection,” *Shock*, 27, 597–604.
- SVANBERG, L. (1981): “Effects of estrogen deficiency in women castrated when young,” *Acta Obstetricia et Gynecologica Scandinavica*, 106, 11–15.
- TAUBENBERGER, J., AND D. MORENS (2006): “1918 Influenza: The Mother of all Pandemics,” *Emerging Infectious Diseases*, 12(1), 15–22.
- TRAN, C., S. KNOWLES, B. LIU, AND N. SHEAR (1998): “Gender differences in adverse drug reactions,” *Journal of Clinical Pharmacy and Therapeutics*, 38, 1003–1009.
- VAN LUNZEN, J., AND M. ALTFELD (2014): “Sex Differences in Infectious Diseases—Common but Neglected,” *Journal of Infectious Diseases*, 209(Suppl 3), S29–S80.
- WEIL, D. (2014): “Health and Economic Growth,” in *Handbook of Economic Growth*, ed. by P. Aghion, and S. Durlauf. North Holland.
- WORLD HEALTH ORGANIZATION (2010): “Cholera vaccines: WHO position paper,” *Weekly epidemiological record*, 85(13), 117–128.
- WUNDERLICH, F., W. BENTEN, AND M. LIEBERHERR (2002): “Testosterone signaling in T cells and macrophages,” *Steroids*, 67, 535–538.

Appendix A: List of countries in our sample

Algeria, Argentina, Australia, Austria, Bangladesh, Barbados, Belgium, Belize, Bolivia, Brazil, Bulgaria, Canada, Chile, China, Colombia, Costa Rica, Czech Republic, Denmark, Dominican Republic, Ecuador, Egypt, El Salvador, Finland, France, Germany, Greece, Guatemala, Guyana, Haiti, Honduras, Hungary, Iceland, India, Indonesia, Iran, Iraq, Ireland, Italy, Jamaica, Japan, Korea, Malaysia, Mauritius, Mexico, Morocco, Myanmar, Netherlands, New Zealand, Nicaragua, Norway, Pakistan, Panama, Paraguay, Peru, Philippines, Portugal, Poland, Romania, Russian Federation, Slovakia, South Africa, Spain, Sri Lanka, Sweden, Switzerland, Taiwan, Thailand, Trinidad and Tobago, Tunisia, U.S.A., United Kingdom, Uruguay, Venezuela, Vietnam, Yugoslavia.

Appendix B: Disease Classification Information

As part of our analysis we classify the different infectious diseases into two groups: a group of vaccine-treatable diseases and a group of non-vaccine-treatable diseases. In order to perform this classification we rely on specific information regarding the nature of each disease and the medical breakthroughs related to its treatment that took place as part of the international epidemiological transition.

Cholera: Cholera is an acute diarrheal disease caused by the bacterium *Vibrio cholerae* and is spread by drinking contaminated water or eating contaminated food. The first effective vaccine against cholera was developed in 1896 by Willhelm Kolle. However, the vaccines available since then including modern ones developed in the 1990s provide only limited immunity, typically for no more than one year. (Parish, 1965; WHO, 2010) The most important mechanism for controlling cholera is the provision of clean drinking water and improved sanitation. Since cholera-induced diarrhea results in severe dehydration within 3-4 hours, the development of more effective oral rehydration therapy in the 1950s provided a major breakthrough in the treatment of cholera. Furthermore, antibiotics developed in the 1940s shorten the course of the disease and reduce the symptoms. We code cholera as a non-vaccine case given the ineffectiveness of the available vaccines.

Diphtheria: Diphtheria is caused by the *Corynebacterium diphtheriae* and is spread by air or touch. Kitasano and Behring developed in 1890 the first antitoxin from the serum of animals that had been infected with the toxins secreted by the bacilli and showed that this antitoxin could be used to treating diphtheria patients. In 1913, Behring reported first trials of successful immunization with toxin-antitoxin mixtures (Baker and Katz, 2004). However, even though toxin-antitoxin mixtures had been successfully used for vaccination in some U.S. cities in the 1920s, it was generally difficult to employ these mixtures as it was unclear how toxins and

antitoxins should be balanced to induce an effective at the same time safe immunization. The key event in the development of safe vaccines was the development of a chemically modified toxin, called toxoid, by Leon Ramon of the Pasteur Institute. By using heat and formalin, the toxin's toxicity was reduced without affecting its immunizing power. This vaccine was approved for children in France in 1927 and made compulsory for children in France in 1938 (Parish 1965), but other countries only adopted the vaccine later. Mass immunization at the global scale did not start until after World War II and after the licensing of the combined diphtheria-tetanus (DT) vaccine in 1947 (Baker and Katz, 2004). Large-scale vaccinations were further improved and facilitated with the development of the jet injector in the 1960s and the invention of freeze drying during WWII, which greatly increased the storability of vaccines and facilitated vaccine transportation. Treatment of diphtheria became more effective with the introduction of antibiotics, in particular penicillin, in the 1940s. Given that global large-scale immunization campaigns did not start before the mid 1940s, we code diphtheria as a vaccine-case.

Influenza: Influenza is caused by three strands of the influenza virus, types A, B, and C, and is passed by breath-born droplets, such as sneezing and coughing. The type 1 A virus was first isolated and identified in 1934 and type B in 1940. The first vaccine was developed during World War II to protect soldiers and production was expanded to civilian markets in 1946 (Hoyt, 2006). Vaccination is the primary control mechanism, but the development of antibiotics in the 1940s also reduced mortality from secondary bacterial infections. Hence, we place influenza in the vaccine group.

Malaria: Malaria is caused by parasites transmitted by the female mosquito of the Anopheles type. The causal parasites were identified and the role of the Anopheles mosquito established in the 1890s. Since then, control of mosquitoes has been generally applied strategy to control malaria, but became much more effective with the discovery of DDT residual insecticides in the late 1940s. Effective vaccines still do not exist to this date. Given this, we use 1940 as the intervention date and assign malaria to the non-vaccine group.

Measles: Measles are a viral infection, principally occurring among children. The disease is transmitted by air and through touch. Measles vaccines were first produced in 1958 and licensed in 1963. A single dose of the measles virus vaccine provides life-long immunity in over 95% of cases. First steps in the development of measles vaccines was the discovery that the disease was caused by a virus in 1911 and the isolation of the measles virus in 1954. We code measles as a vaccine-case.

Plague: Plague is caused by the bacterium *Yersinia pestis* and is transmitted to humans through the bite of infected fleas. First vaccines were developed in the late 1890s, but the exact nature of the disease and the role of the flea in transmitting the disease from infected rats and other wild rodents were not fully studied until 1914 (Parish, 1965). When this was established, the elimination of rats and fleas became a key step in the prevention of plague. While the

vaccines developed in the 1890s were effective in reducing plague incidences and plague mortality, protection only lasted for few months (Parish, 1965). With the development of antibiotics, especially streptomycin, in the 1940s, vaccination became of secondary importance. Given the limited protection vaccines afforded and the fact that they had been available since the 1890s, we assign plague to the non-vaccine group.

Pneumonia: Pneumonia is caused by a variety of infectious agents, including viruses and bacteria. The most common cause is infection with the bacterium *Streptococcus pneumoniae*. The bacterium was first isolated by Louis Pasteur in 1880, the identification of other causative agents followed gradually. Vaccines against pneumococcal pneumonia - pneumonia caused by the *Streptococcus pneumoniae* bacterium - became first available 1945. But they were not widely used, not very effective, and not further developed since their introduction coincided with the discovery of penicillin and other antibiotics, which proved very effective in treating and reducing mortality from bacterial pneumonia. It was only in the late 1970s and early 1980s that these vaccines were further developed, which resulted in the licensing of effective vaccines in 1977 and 1983, the final breakthrough being the development of pneumococcal conjugate vaccines in 2000.(Klein and Plotkin, 2007) As far as viral pneumonia is concerned, viral vaccines, in particular influenza vaccines developed in the 1940s, are effective in preventing influenza infections, which are a common causative agent, but much less frequent than *Streptococcus pneumoniae* (Ruuskanen et al., 2011). We assign pneumonia to the vaccine group given that key vaccines became available in the 1940, albeit only partially effective.

Scarlet fever: Scarlet fever is caused by certain strains of group A *Streptococcus* bacteria. The bacteria were first isolated in 1887. Efforts to produce a streptococcus vaccine followed but the results were not convincing. To this date no effective vaccine exists. However, the disease is effectively treated with antibiotics, including penicillin, developed in the 1940s. Given this, we assign scarlet fever to the non-vaccine group.

Smallpox: Smallpox was an infectious disease caused by the viruses *Variola major* and *Variola minor*. It was eradicated by the late 1970s. An effective vaccine was developed in 1798 by Edward Jenner, which was derived from cowpox, a related disease found among animals. However, large-scale immunization was not possible until the 1950s when freeze-drying, invented in the 1940s, was adopted for mass production of vaccines (Fenner, 1996) Until then, the storability of the vaccine was very limited and temperature fluctuations frequently caused the vaccine to lose its potency. The invention of the jet injector in the 1960s was another important development that significantly facilitated the administration of the vaccine. We assign smallpox to the vaccine group.

Tuberculosis: Tuberculosis is caused by the bacterium *Mycobacterium tuberculosis* and is transmitted through air. The bacterium was discovered in 1882 by Robert Koch. The first effective vaccine, the BCG (*Bacille Calmette Guérin*) vaccine, developed by the French Pasteur

Institute and still in use today, became available for use in humans (Parish, 1965) in 1921. However, mass immunization did not start until after the late 1940s when UNICEF in cooperation with the Danish and Swedish Red Cross teams started mass immunization campaigns. Until then, the vaccine had been primarily used in France where by 1933 about 20% of all children were vaccinated within the first week of life, but outside of France only 500,000 children had been vaccinated at that time (Parish, 1965). During the mass immunization campaigns in 1948-1951 alone 30 million people were tuberculin tested and 14 million were vaccinated. The campaign was extended further under a joint UNICEF/WHO initiative which led to the vaccination of an additional 60 million people by 1956 (Comstock, 1994). Given the sensitivity of the BCG vaccine to sunlight and temperature fluctuations and the fact that it could only be stored for 10 days after initial preparation (Bonah, 2005), the invention of freeze-drying in the 1950s provided another major step forward in the fight against tuberculosis (Parish, 1965). In addition, drugs developed in the 1940s and 1950s, in particular streptomycin in 1944, provide effective treatment and made most cases of the disease curable. Given that mass immunization campaigns did not start before the late 1940s, we treat tuberculosis as a vaccine-case.

Typhoid fever: Typhoid fever is caused by the bacterium *Salmonella typhi* and is spread through contaminated water or food. Vaccines were developed before World War I, but they were not be very effective. With the introduction of antibiotics in the 1940s, which provided effective treatment, the use of vaccines has become rather of secondary importance in the control of the disease. We assign typhoid fever to the non-vaccine group given that vaccines, albeit possibly not very effective, had been already available long before 1940.

Typhus: Typhus is caused by *Rickettsia* bacteria transmitted by the human body louse *Pediculus humanus corporis*. The role of the louse was established in 1909 and the bacterium first demonstrated in 1916 (Parish, 1965). Public health measures such as good hygiene and sanitation played an important role in the control of the disease. First attempts in developing a vaccine to be used in humans were made in the 1930s in the form of the Weigl vaccine, using killed bacteria, and the Cox vaccine, using live bacteria (Lindenmann, 2002). They were albeit difficult to manufacture and turned out to not be very effective. With the introduction of antibiotics, which provide effective treatment for typhus, and the development of DDT, which allowed for very efficient delousing, vaccines fell into disuse (Lindenmann, 2002). We assign typhus to the non-vaccine group given that vaccines were already available before 1940 and not much used after the introduction of broad-spectrum antibiotics.

Whooping cough: Whooping cough, or pertussis, is caused by the bacterium *Bordetella pertussis* and occurs primarily among young children. The causative agent was first isolated in 1900 and the first effective vaccine was developed in the 1930s.²¹ First large-scale clinical trials were

²¹There had been earlier attempts already in the 1920s to develop a pertussis vaccine. However, these early vaccines had very limited efficacy and became obsolete with the introduction of the vaccines developed by Kendrick and Eldering (Parish, 1965; Granstroem, 1996)

conducted in the U.S. in the late 1930s and early 1940s, which demonstrated the effectiveness and safety of the vaccine (Parish, 1965), and the American Academy of Pediatrics approved the vaccine for routine use in 1943. However, it was not until the later 1940s when the DTP combination vaccine was licensed that pertussis vaccines were widely used. Britain in fact, was not convinced by the American studies and did not recommend the use of pertussis vaccines until the late 1950s (Parish, 1965).

Table 1: Descriptive statistics**Panel A: Men**

Variable	Mean	S.D.	Min	Max
Mortality, overall, 1940	0.436	0.274	0.0326	1.126
Mortality, vaccine group, 1940	0.363	0.241	0.03	0.909
Mortality non-vaccine group, 1940	0.0635	0.14	0	0.853
Small diseases, vaccine group, 1940	0.0383	0.0325	0.0024	0.154
Small diseases, non-vaccine group, 1940	0.0105	0.0105	0	0.0861
Malaria, 1940	0.051	0.14	0	0.853
Pneumonia, 1940	0.176	0.151	0	0.621
Tuberculosis, 1940	0.149	0.123	0	0.609
Life expectancy at birth, 1940	46.8	11.19	25.46	66.97
Life expectancy at birth, 1980	64.07	6.52	48.64	73.56
Years of schooling, 1940	4.54	2.51	0.144	11.16
Years of schooling, 1980	7.69	1.94	3.21	11.44

Panel B: Women

Variable	Mean	S.D.	Min	Max
Mortality, overall, 1940	0.436	0.274	0.0326	1.126
Mortality, vaccine group, 1940	0.363	0.241	0.03	0.909
Mortality non-vaccine group, 1940	0.0635	0.14	0	0.853
Small diseases, vaccine group, 1940	0.0383	0.0325	0.0024	0.154
Small diseases, non-vaccine group, 1940	0.0105	0.0105	0	0.0861
Malaria, 1940	0.051	0.14	0	0.853
Pneumonia, 1940	0.176	0.151	0	0.621
Tuberculosis, 1940	0.149	0.123	0	0.609
Life expectancy at birth, 1940	49.61	12.17	28.44	70.72
Life expectancy at birth, 1980	69.64	7.5	51.51	79.6
Years of schooling, 1940	4.26	2.54	0.146	9.17
Years of schooling, 1980	7.71	2.17	3.13	11.61

Table 2: Disease mortality and life expectancy

	Dependent Variable: Life expectancy at birth						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mortality, overall	15.39*** [1.805]	13.98*** [1.631]					
Female		3.578*** [0.504]	3.531* [0.504]	4.118* [0.417]	3.782* [0.479]	3.598*** [0.459]	3.758** [0.545]
Female x Mortality, overall		2.816** [1.385]					
Mortality, vaccine group			12.33* [1.873]				
Female x Mortality, vaccine group			3.384* [1.638]				31.25* [17.62]
Mortality, non-vaccine group			20.60* [3.495]				
Female x Mortality, non-vaccine group			1.485 [3.923]				-31.19 [36.98]
Malaria				19.12* [3.528]			
Female x Malaria				2.836 [3.956]			
Pneumonia					16.23* [3.119]		
Female x Pneumonia					4.657^ [2.923]		
TB						-1.900 [3.608]	
Female x TB						7.997** [3.366]	
Overall mortal. excl. Malar./Pneum./TB				14.11* [1.606]	13.32* [2.118]	20.38*** [1.773]	
Mortality small diseases, vaccine group							53.27** [17.52]
Mortality small diseases, non-vaccine							70.21** [32.09]
Observations	300	300	300	300	300	300	300
Within R-squared	0.87	0.91	0.92	0.88	0.9	0.86	0.89

Standard errors in brackets

All regressions include country and year fixed effects

*** p<0.01, ** p<0.05, * p<0.1, ^ p<0.15

Table 3: Life expectancy and schooling

	Dependent Variable: Years of schooling of age group 5-9				
Method	(1) OLS	(2) OLS	(3) IV	(4) IV	(5) IV
Measure of diseases mortality in 1st stage			overall	vacc./non-vacc.	vacc./non-vacc.
Life expectancy	0.101*** [0.0190]	0.0977*** [0.0214]	0.117*** [0.0287]	0.114*** [0.0277]	0.112*** [0.0309]
Female	-0.553*** [0.161]	-0.772 [0.622]	-0.620*** [0.170]	-0.606*** [0.167]	-0.737 [0.553]
Female x LE		0.00391 [0.0108]			0.00233 [0.00998]
Observations	300	300	300	300	300
1st stage F-statistic	-	-	107.012	29.29	57.57

Standard errors in brackets

All regressions include country and year fixed effects

*** p<0.01, ** p<0.05, * p<0.1, ^ p<0.15

Table 4: Robustness tests

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Panel A: 2nd stage results	Dependent Variable: Years of schooling of the age group:								
Age group	5-9	5-9	5-9	5-9	5-9	5-9	5-9	5-9, lagged	60-64
Life expectancy at birth	0.114*** [0.0277]	0.171*** [0.0265]	0.160*** [0.0320]	0.126*** [0.0311]	0.141*** [0.0360]	0.176*** [0.0566]	0.116*** [0.0282]	0.0269 [0.0195]	-0.0634*** [0.0201]
Female	-0.606*** [0.167]	-0.811*** [0.169]	-0.830*** [0.185]	-0.665*** [0.181]	-0.744*** [0.199]	-0.818*** [0.157]	-0.648*** [0.179]	-0.542*** [0.118]	-0.405*** [0.122]
Log GDP per capita			1.196*** [0.349]		1.203*** [0.363]				
Institutions x 1980				-0.116* [0.0685]	-0.141* [0.0756]				
Life expectancy at age 20						-0.0324 [0.0639]			
Lagged years of schooling							-0.0771 [0.0842]		
Panel B: 1st stage results	Dependent Variable: Life expectancy at birth								
Mortality, vaccine group	12.33*** [1.873]	12.1*** [2.641]	14.52*** [2.145]	10.166*** [1.870]	13.3*** [2.039]	9.465*** [1.591]	12.185*** [1.856]	12.33*** [1.873]	12.33*** [1.873]
Female x Mortality, vaccine group	3.384** [1.638]	4.027* [2.318]	3.302** [1.673]	3.32** [1.587]	3.255** [1.563]	1.393* [1.138]	3.189* [1.624]	3.384** [1.638]	3.384** [1.638]
Mortality, non-vaccine group	20.60*** [3.495]	48.15*** [13.41]	11.045^ [7.448]	20.549*** [3.750]	6.407 [7.01]	4.268 [4.749]	20.333*** [3.463]	20.60*** [3.495]	20.60*** [3.495]
Female x Mortality, non-vaccine	1.485 [3.923]	-12.74 [15.45]	1.121 [4.823]	2.261 [4.260]	1.592 [4.523]	-2.167 [2.264]	1.496 [3.885]	1.485 [3.923]	1.485 [3.923]
Female	3.531*** [0.504]		3.58*** [0.521]	3.573*** [0.492]	3.641*** [0.488]	[0.455]	3.831*** [0.516]	3.531*** [0.504]	3.531*** [0.504]
Log GDP per capita			-0.455 [1.190]		0.225 [1.173]				
Institutions x 1980				-1.024*** [0.183]	-1.02*** [0.184]				
Life expectancy at age 20						0.88*** [0.0734]			
Lagged years of schooling							0.616** [0.269]		
Observations	300	188	278	284	274	256	300	300	300
1st stage F-test	29.29	23.72	22.77	25.55	20.32	12.17	28.71	29.29	29.29

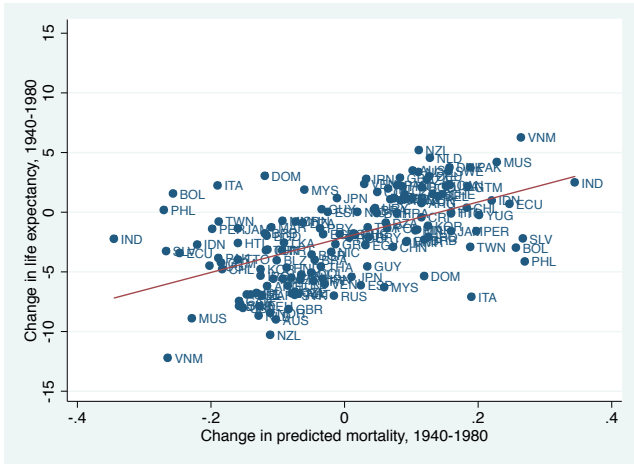
Standard errors in brackets

All regressions include country and year fixed effects

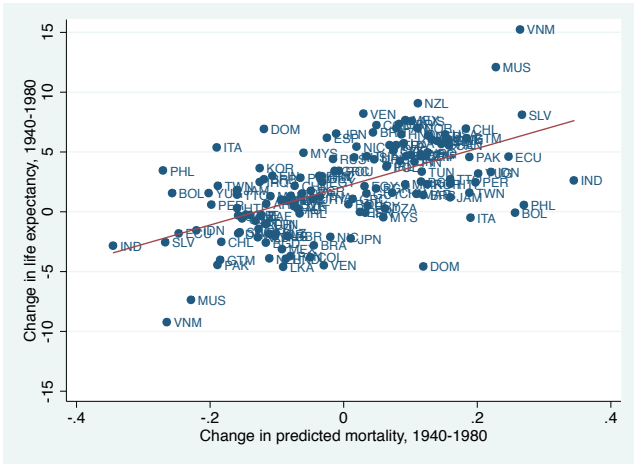
*** p<0.01, ** p<0.05, * p<0.1, ^ p<0.15

Figure 1: Changes in life expectancy and disease mortality, 1940-1980

1a): All diseases

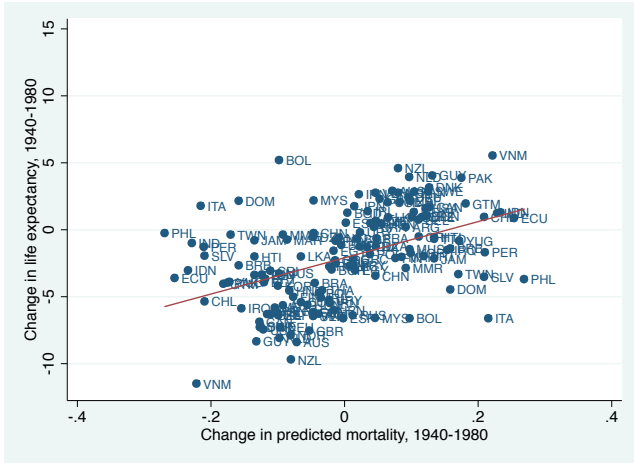


Panel A: Men

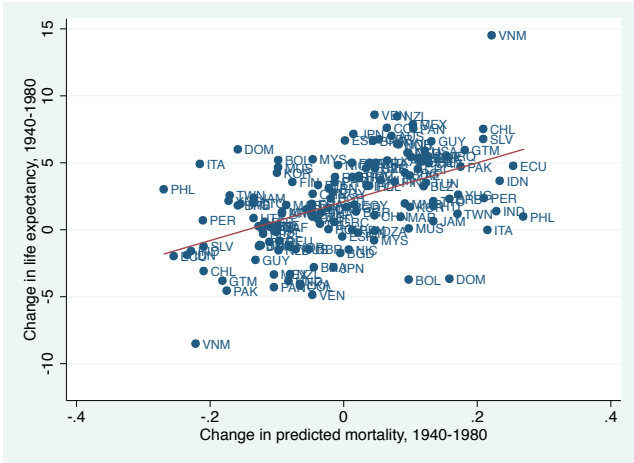


Panel B: Women

1b): Vaccine-treatable diseases



Pane A: Men



Panel B: Women