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What doesn't kill you makes you stronger? The Impact of the 1918 Spanish Flu Epidemic on Economic Performance in Sweden[‡]

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Abstract

We study the impact of the 1918 influenza pandemic on economic performance in Sweden. The pandemic was one of the severest and deadliest pandemics in human history, but it has hitherto received only scant attention in the economic literature – despite important implications for modern-day pandemics. In this paper, we exploit seemingly exogenous variation in incidence rates between Swedish regions to estimate the impact of the pandemic. Using difference-in-differences and high-quality administrative data from Sweden, we estimate the effects on earnings, capital returns and poverty. We find that the pandemic led to a significant increase in poverty rates. There is also relatively strong evidence that capital returns were negatively affected by the pandemic. On the other hand, we find robust evidence that the influenza had no discernible effect on earnings. This finding is surprising since it goes against most previous empirical studies as well as theoretical predictions.

Keywords: Spanish Flu; Difference-in-Differences.

JEL classification: I18; J31; O40.

1 Introduction

In 1918 the world is hit by a first wave of the very contagious Spanish flu. Estimates indicate that 500 million individuals worldwide were infected by the deadly virus, and that 50-100 million people died in the aftermath of an infection between 1918 and 1920 (Johnson and Mueller, 2002). Unlike when customary strains of influenza periodically circulate the world, the majority of the victims of the Spanish flu were healthy young people in the age interval 15-40 – not frail patients, nor children or elderly.

While much has been written and argued about the medical causes of the Spanish flu, the origins of the deadly virus and its connection to more recent pandemics, such as the 2006 bird flu (see e.g. Tumpey et al., 2005; Bos et al., 2011), limited attention has been given to the societal and economic effects of the epidemic. What are the economic consequences following from such a health shock where a large share of the population of working age falls dead within a very limited time period?

Gaining knowledge on this issue is relevant for several reasons. Studying the effects of the Spanish flu can give insights into the effects that future pandemics may have on various economic outcomes. An understanding of the link between such a health shock and subsequent economic performance may also be helpful in establishing appropriate policy responses. Moreover, by mainly affecting the young and healthy, the 1918 influenza shares striking similarities with the HIV/AIDS pandemic in developing countries. However, analysing the economic consequences using HIV/AIDS prevalence or related mortality rates is difficult, as the latter disease is a much slower process. Given the heightened awareness of economic issues associated with pandemics, it seems timely and relevant to acquire knowledge of any consequences of an event such as the Spanish flu. Not least since the influenza, although severe, appeared during a very short time, which facilitates the identification of the effects of interest and serves as a useful test of the effects of a health shock on economic outcomes.

However, the main interest of this work may not even lie in the domain of studying pandemics, past or present. The influenza pandemic also represents a vast labour supply shock, which usually do not occur with such force and randomness as the 1918 wave. Thus, our results will also be informative with respect to the empirical performance of macroeconomic models – since estimated outcomes can be compared to predictions of standard growth models in the literature. Depending on whether the economy is described by a neoclassical growth model or by

an endogenous growth model, different predictions concerning the impact of the shock emerge.

Using administrative data from Swedish regions (25 in total at the time), we employ an extension of the standard difference-in-differences (DiD) estimator using the differing mortality rates across Swedish regions for identification. Focusing on Swedish regions when studying the consequences of the Spanish flu has several advantages. A first advantage is the high variation in flu mortality across counties. Almost one percent of the Swedish population died from the Spanish flu, but influenza mortality varied widely and idiosyncratically between regions, with some areas experiencing almost three times the number of deaths per capita than areas where the pandemic was less severe (Åman, 1990). We use this variation to examine the impact of the 1918 influenza pandemic on economic performance. More specifically we study the effect on earnings, capital returns and poverty in Swedish counties. Besides high variation, using data on Sweden to study the subject matter is beneficial since the population is relatively homogeneous – in ethnic, cultural and socio-economic terms.

A second advantage of using Swedish data is that many key economic indicators are available from administrative datasets which have been consistently collected across regions and time, thus allowing for precise estimates.[1] Hence, the data allow us to estimate the effects of the influenza on a number of economic outcomes while carefully checking key methodological assumptions.

Third, Sweden did not take part in the Great War, during which the flu pandemic started. In this way we reduce the risk of confounding effects of the pandemic with disturbances related to the war. Obviously, Sweden was affected by the war in many ways – and below we try to allow for the possibility that these effects were asymmetrically distributed across regions. However, one main advantage of focusing on a non-belligerent country is the fact that there are no other major shocks to mortality coinciding with the disease. Previous empirical studies on the economic effects of the Spanish flu use data for countries participating in the war, which clearly introduces such a confounding factor, thus making the interpretation of results more difficult.

Finally, the pandemic took Sweden by surprise and the opportunities of evading the disease were limited if not non-existent. Obviously, there was little point in moving abroad or to another region – since most of Northern Europe was hit at roughly the same time. Besides, the

[1]It is well known in the literature on pandemics that a death caused by influenza was sometimes reported as pneumonia mortality in death records. However, the correlation between influenza and pneumonia mortality at the county level transpires to be quite weak. We interpret this as an indication of the quality of the data, which is in turn a consequence of the detailed instructions sent from national authorities to health personnel on how to verify the cause of death (see e.g. SCB 1911) and that the correct disease was, in fact, recorded.

possibilities of moving abroad were seriously restricted by the war in any case. As far as internal migration is concerned, it was further severely limited by the fact that labour arrangements in e.g. the agricultural sector generally comprised long-term contracts (see for instance Lundh and Olsson, 2008). For these reasons, it is unlikely that avoidance played a major role in Sweden.

In accordance with theoretical predictions, our results suggest that the pandemic had a strong negative impact on capital returns. However, we fail to find any evidence of an upward adjustment in wages – even though such an outcome would be expected given the capital deepening that took place after the pandemic. Instead, our estimates for this variable are very precise and robustly close to zero. This surprising result, however, may possibly be explained with reference to unexpected changes in inequality at the regional level. When we consider the proportion of poor people in the population, we find a surprisingly large positive effect of the epidemic. Thus, it appears that its disruption to economic activity goes beyond simply raising or lowering factor returns.

The next section outlines theory and empirical evidence on the Spanish flu. Section 2 also reviews the theoretical and empirical literature on economic consequences. Section 3 gives an overview of the economic situation in Sweden during the time period considered, and also provides important information on the course of the epidemic in Sweden. Section 4 describes the data used in this study, while section 5 presents the methodology. Section 6 visualises support of the common time trend hypothesis, and discusses the empirical results, namely the difference-in-difference estimates of the effect of regional exposure of the influenza on earnings, capital returns, and poverty. Finally, section 7 concludes.

2 The Spanish Flu Pandemic: Facts, Theory and Empirical Evidence

The first official reports on the 1918 flu came from Spain, which is how it got its popular name.[2] Recent research, however, suggests that the disease may have reached Europe due to the large movement of troops from the US taking part in battles on the Western front in the World War I. On the other hand there is also evidence suggesting that the Spanish flu originated in Asia and was transmitted to Europe by Chinese workers. In any case, upon reaching the European continent, the spread of the pandemic was accelerated by increased troop movement due to

[2]The reason why the first report came from Spain is likely related to the fact that the latter country did not take part in World War I and at the time had an uncensored media.

the war (Patterson and Pyle, 1991). Among researchers in medical history there was a basic consensus that the disease ran its course in three to four waves (even though three waves were experienced in most, but not all, world regions). The first wave was in the spring of 1918, with the disease returning in the fall of the same year and again in 1919. The last wave occurred mainly in Scandinavia and some isolated islands in the South Atlantic, but due to the relatively low spread experts do not agree on whether it qualifies as a "fourth wave". The second wave is estimated to have been the most severe one, and only areas that were exceptionally isolated escaped the influenza altogether.

One interesting feature of the second wave of the pandemic is that it took the world by complete surprise. It is important to understand that it may be very misleading to focus on reported incidence rates of the flu, since the pandemic changed character during the course of the year. The first documented eruption of the disease – i.e., the cases in Haskell County – is reported to have caused a severe death toll. However, as the influenza spread throughout the world during the spring of 1918, it actually had such a low mortality rate that experts started doubting whether it was influenza at all. For example, in the summer of 1918, Little et al. (1918) write in conclusion

> we wish to point out that although this epidemic has been called influenza for the want of a better name, yet in our opinion it cannot properly be considered such for the following reasons:
>
> 1. The clinical course, though similar to that of influenza, is of very short duration, and there is, so far as we have observed, an absence of relapses, recurrence, or complications [...]

This is but one example of how medical experts were confused by the *mildness* (!) of the influenza during the first wave, and consequently reluctant to accept it as such. In addition, as the spread of the virus halted in the late summer of 1918, many observers actually concluded that the epidemic had disappeared (Barry, 2005). Besides, given the strict censorship that was imposed by World War One participants on issues related to public health, it is very unlikely that the general public had any idea what was going on until they were reached by the more lethal wave that occurred during the autumn of 1918. As discussed in Section 3.4 and indicated by figure 2, the Swedish experience in this regard was no different from that of the rest of the world. For example, documentation between two doctors working in the southernmost region

Scania in Sweden at the time indicte that the first wave was very mild and further suggest that there were conflicting views between the two on whether there was a virus-caused influenza or a new typ of pneumonia (Petrn, 1918b) (Petrn, 1918a).

A prominent characteristic of the second wave of the Spanish flu were the exceptional mortality rates. During a normal influenza epidemic, approximately 0.1 per cent of all infected individuals perish. In comparison to this case fatality rate, the second and most severe wave of the epidemic in the fall of 1918 was 5 to 20 times more deadly. The main reason why the Spanish flu was so extraordinarily aggressive is that the virus not only attacked the bronchus, but also the lungs, leading to many people dying from severe pneumonia. According to Morens and Fauci (2007), most deaths resulted from "respiratory complications". Recent research indicates that it was the combination of three genes that made it possible for the virus to attack human lungs.[3] The incubation time of the Spanish flu and the time between infection outbreak and death was very short. According to Taubenberger and Morens (2006), most deaths occurred 6-11 days after the outbreak of infection, but there is also evidence that some deaths occurred as early as two days after infection (Åman, 1990). What furthermore characterizes the disease is the heavy toll among young adults. It is estimated that around half of the death toll was paid by individuals between 15 and 40 (Simonsen et al., 1998). This is very unusual and unlike other (influenza) diseases, which typically exhibit a U-shape in terms of the mortality distribution over age groups, the Spanish flu is known to have had a W-shaped distribution.

Due to lack of reliable world data, there is considerable variation in the number of deaths estimated to have been caused by the 1918-1919 influenza virus. Estimates range from 20-40 million (Patterson and Pyle, 1991) to 50 million, and the highest estimates are as high as 100 million (Johnson and Mueller, 2002). However, there is widespread agreement that the global consequences of the pandemic in terms of infections and death toll are unparalleled. Taubenberger and Morens (2006), for example, call it "the mother of all diseases" and estimate that as many as 500 million individuals were infected (out of a world population at the time of 1.8 billion).

[3]The combination of these three genes pioneered the creation of the protein RNA-polymerase. Due to the existence of this protein the virus could mass-produce in human lungs.

2.1 Theoretical Perspectives

As we have seen, the Spanish flu pandemic represented a massive health shock in Sweden and other countries. From a purely economic point of view, we may think of this as a shock to labour supply and human capital in the economy, which on the other hand leaves physical capital intact. Hence, contrary to war and other major shocks to the macroeconomy, which typically have an impact on physical as well as human capital, the Spanish flu pandemic is a useful 'natural experiment' for labour supply. Thus, in order to generate hypotheses for how our main outcome variables may react to the pandemic, we now briefly review what the macroeconomic literature on economic growth has to say on the topic. Also, considering that Swedish regions – which are our main unit of analysis – may be seen as small open economies in relation to each other, we cover some seminal contributions from trade theory.

In the context of growth theory, it is interesting to consider predictions concerning the immediate response to the pandemic as well as the medium term response during the transition back to a steady state. We now follow the example of Boucekkine et al. (2008) and contrast predictions from a standard neoclassical growth model with an endogenous growth model along the lines of Lucas et al. (1988). In the neoclassical one-sector model, deriving the short- and medium-term impact of a pandemic is relatively straightforward. The immediate impact of a negative human capital shock is that the ratio between physical and human capital is moved above its steady-state level. This leads to a corner solution in the immediate aftermath of the pandemic, in which investment in physical capital is equal to zero for a while. The relative abundance of capital also has consequences for factor returns: as long as the ratio between physical and human capital remains above its pre-epidemic level, returns to physical capital will be lower than returns to human capital, leading to more investment in human capital. Moreover, due to the smaller population size the economy will generally have been growing faster on a per capita basis than before the pandemic. However, the economy will return to an interior solution within a finite amount of time.

These predictions, which essentially suggest that survivors can enjoy higher wages and higher growth rates in the period following the pandemic, can be contrasted with model of endogenous growth. As with Boucekkine et al. (2008), we will focus on a Lucas-style model with a separate sector focusing on producing human capital. One alternative would be the AK model (see, for instance, Frankel, 1962), who considers a production function without decreasing returns, because in the typical AK model the accumulation of capital fosters endogenous technological

progress and growth. Within such a model the negative shock of labour supply might even increase capital returns. Another way to model endogenous economic growth is through the production of new ideas. Within this topic there are two main branches, the first focuses on innovation in the sense that with innovation product variety will increase (see, for instance, Rivera-Batiz and Romer, 1991). The second branch builds on the idea of Schumpeter's "creative destruction" and quality improving innovations (see, for instance, Philippe and Howitt, 1992). These models would predict a negative effect on overall growth and capital income because the negative shock to labour supply would also disrupt innovation and thus economic growth.

In a two-sector model in the spirit of Lucas et al. (1988), there is an educational sector which produces human capital. Growth in human capital is a function of the proportion of workers in the economy devoted to education. In this context, the immediate response is similar to that of the one-sector outlined above: human capital has become relatively scarce, and for this reason the remuneration of labour increases. However, the transition dynamics that follow are completely different from the one-sector case: the educational sector is more sensitive to increases in wages and, thus, there will be a shift of human capital from the educational sector to the final goods sector. As a consequence, the period after the epidemic will be characterised by slow growth and high wages.

The models discussed so far do not consider an effect on labour demand. However, this assumption might be too restrictive. Labour demand might be reduced because of the lower productivity of labour. Moreover, due to the high death toll the demand for goods might be decreased, also resulting in a negative shift of labour demand. If this is the case, wages might even decrease if the shift of labour demand is big enough.

Furthermore, the models above assume that factor returns are determined locally. This assumption might not give a very accurate representation of the actual situation of Swedish counties during the period of study. Thus, it is likely that additional insights may be gained if the possible consequences of the epidemic are analysed in a trade theory framework. As discussed by Hanson and Slaughter (2002), an endowment shock may alter output mixes in an economy and therefore mitigate the decrease of capital returns. This line of reasoning follows from Rybczynski (1955) – a main result in the Heckscher-Ohlin (HO) trade theory assuming a two sector economy with two factors. According to the theorem, a decrease in the relative endowment of one factor – for example labour – decreases the output of labour-intensive products and simultaneously increases the output of at least some other products. In other words, the

Spanish flu would affect the output mix of the economy. Such changes in the overall production decrease the relative demand for labour – thereby matching the changes in the relative supply of labour. This mechanism will only work if economies can specialize in their production and if output changes can be accommodated by corresponding changes in exports and imports.

Having discussed both income from capital and labour, we now consider effects on economic inequality and poverty. These additional variables are, of course, strongly related to factor returns. If wages increase and capital returns decrease, workers are likely to experience an increase in incomes while the income of capital holders decreases. At a first glance, these effects likely result in a reduction in absolute poverty and decreasing economic inequality as the income distribution becomes more compressed. On the other hand, the theoretical models predict an overall lower stock of labour and thus fewer people earning wages. Moreover, some survivors from the Spanish flu might be infirm and unable to work even after recovery. Such morbidity effects make the overall effect on poverty unclear. Besides, if the negative labour shock does not affect workers' wages, as suggested by the above-mentioned Rybczynski (1955), the existence of long lasting health effects may in fact increase poverty rates in the economy.

In a recent paper, Boucekkine and Laffargue (2010) consider distributional effects of epidemics. In an overlapping generations model with heterogeneity in worker skills, it is shown that income inequality may increase in the short and medium term – even though the epidemic leaves no permanent traces in the income distribution. Since we consider poverty as one outcome variable in this paper, Boucekkine and Laffargue (2010) provide a very useful theoretical backdrop. However, in their case the mechanism is a reduced investment in human capital due to orphanhood in low-skilled families. Since our analysis excludes orphans, our results must be driven by a different mechanism.

Also considering the fact that Sweden was a poor country in those days, additional insights may be gained concerning poverty from development economics. Lewis (1955) models a relatively undeveloped economy of two sectors: the traditional (agricultural) sector and the modern (manufacturing) sector. The model relies on two main assumptions: that wages in the latter sector are higher than in the former, and that there is a surplus of labour in the traditional sector which can be withdrawn from the sector without any loss of output. An employment expansion in the modern sector takes place and each time a worker moves from the traditional to the modern sector, that worker's economic well-being increases and poverty decreases. Assuming that the Spanish flu decreases or even eliminates the labour surplus in the traditional sector,

while also increasing the demand for labour in the modern one, absolute poverty will decrease. As the marginal product of labour in the traditional sector is no longer zero, additional workers in this sector can now only be withdrawn to the modern one at a higher cost of lost production, and workers economic well-being increases in both sectors.

In summary, we have found that the predictions concerning our three outcome variables – capital returns, earnings and poverty – may differ according to the theoretical perspective chosen. One result that seems fairly intuitive is that the immediate response should be an increase in earnings per capita and a reduction in capital returns. However, as we have seen, the transitional dynamics from that point might look very different depending on whether one considers a one-sector growth model, and endogenous growth model, or a trade model, in which adjustment is brought about by changing sectoral shares and not factor returns.

Finally, it should be noted that the outcome variables we are considering are capital returns per capita and earnings per capita. Most predictions above are concerned with wage rates, and other margins of adjustment (such as hours worked) are not considered at all. However, to the extent that wages are increased by the shock, one would expect earnings to increase as well since an increase in hours worked may be expected. However, one needs to keep in mind that the workers' health status could be worse after the shock, and this might reduce the impact of the pandemic on wages as well as the effect of wages on hours worked. Consequently, it is of great interest to find out whether survivors recovered fully or not – an issue that we are unfortunately unable to address satisfactorily with our data.

2.2 Empirical Evidence

A growing literature tests the so-called Fetal Origins hypothesis, analysing the consequences of environmental shocks and *in utero* exposure on later health and labour market outcomes, focusing in particular on the effects of the Spanish flu (cf. Almond and Mazumder, 2005; Maccini and Yang, 2009; Nelson, 2010). In general these studies suggest long term damage for prenatal exposure to pandemic influenza and that children of infected mothers are more likely to have health problems and experience lower wages as adults than non-affected children.

In this study, however, we are concerned with short- and medium-term effects of the pandemic. Up to now there have not been many empirical studies estimating this impact. Besides, most empirical studies face two serious problems. Firstly, there is often a lack of reliable data from the time period. The second problem is that identification is difficult due, *inter alia*, to

the fact that the flu occurred during and shortly after the First World War.

Brainerd and Siegler (2003) is one of the few papers that consider the effects of the influenza on economic growth. They study changes in real personal incomes between 1919/21 and 1930. Due to data restrictions they are only able to analyse medium-term effects and are not able to distinguish whether the effect was due to recovery or growth. In any case, the authors find a significant positive effect: those states that where hit harder by the flu experienced a higher income growth rate from 1919/1921 to 1930. From a theoretical point of view, this result might reflect either capital deepening or be driven by increased investment in human capital and higher population growth after the occurrence of the Spanish flu.

In a more recent paper, Garrett (2009) analyses the effects of the pandemic on manufacturing wages. Garrett (2009) uses the same mortality data as Brainerd and Siegler (2003), but he has access to wage growth between 1914 and 1919; thus, the study is able to compare before and after the pandemic, but it is only able to estimate effects in the very short term. The paper concludes that the epidemic appears to have had a positive impact on manufacturing wages. However, it is not always clear to what extent the above results are not attributable to the First World War.

Focusing on India, Bloom and Mahal (1997) analyse the effects of the Spanish flu using data on population changes and acre sown per capita in 13 provinces. India was severely hit by the pandemic, with very high death tolls (an estimated 17 to 18 millions people died in the flu) and the epidemic affected various regions of the country quite differently. Bloom and Mahal (1997) do not find that any relationship between the magnitude of population decline following from the influenza and the area sown per capita across Indian provinces.

In summary, there have been some attempts to estimate the economic effects of the Spanish flu pandemic in the US and India, but there is still no study which rigorously applies the methods typically used to conduct causal inference. The main reason appears to be a severe lack of reliable data. As we will show in the following sections, Swedish data appear to offer a significant improvement compared with previous studies.

In addition to the above three studies specifically examining the effects of the Spanish flu, there have been some attempts to analyse the economic consequences of other major influenza pandemics. The most relevant studies consider the effect of the Black Death (a combination of plagues) killing a large share of the West European population between 1348 and 1351. Robbins (1928) examines the economic consequences of the epidemic, using data from the fourteenth cen-

tury for France and England, and concludes that the epidemic affected the two economies quite differently. Although data limitations make it problematic to maintain that observed changes are directly attributable to the Black Death, the empirical analysis suggests that there were no permanent epidemic-related changes in commodity prices in any of the countries. Moreover, in many respects the economic organization of France was not affected by the Black Death. This is further confirmed by more recent work by Bloom and Mahal (1997), who do not find any relationship between real wages and population changes in France. For England, however, Robbins (1928) notes that the negative labour supply shock seems to have reduced the value of land, and dramatically increased wages. In addition, opening the door to many new kinds of employment, the Black Death re-organized labour and many people moved into new occupations. As concluded by Brainerd and Siegler (2003), the effect on income per capita is less clear.

Taking both a short- and a long-term perspective, Herlihy and Cohn (1997) analyses the effects of the Black Death in Europe. Along the lines of Robbins (1928), their results suggest that the drop in labour supply increased wages in the agricultural sector, while land rents dropped. Moreover, the study suggests that wages remained persistently high, in turn inducing a change in the economic structure in the long run, where labour was substituted by capital. Herlihy and Cohn (1997) also hypothesise that major pandemics will cause an increase in wealth inequality and claim that the Black Death skewed the distribution of wealth compared to an earlier situation when property was much more evenly distributed.

In a recent study, Alfani (2010) explores the consequences on economic structures of the severe mortality crisis caused by the 1630 plague using detailed documentation available for the city of Ivrea in Italy. Testing the Herlihy and Cohn (1997) hypothesis, the results indicate that the plague decreased inequality in the short term, but increased inequality in the long run. Cohn and Alfani (2007) suggest that it was primarily the unskilled immigration to the city to fill the gap in the urban population that fuelled the rise in economic inequality in the long run.

Finally, an alternative strategy for analysing the short- and medium term economic consequences of sharp health-related reductions in the workforce is to simulate the effects of potential future pandemics. Using micro-macro simulation models, Keogh-Brown et al. (2010) analyse the possible impact of an exhaustive influenza on the UK economy. A severe scenario like the Spanish flu, with more than one percent of the population dying, is estimated to yield an impact of more than 20 per cent loss of GDP in the first pandemic quarter, and more than 4 per cent

loss of GDP in the first year.

2.3 Drivers of the Influenza

It has been argued by several authors that the 1918 influenza pandemic represents a good 'natural experiment' – for estimating short term effects (Brainerd and Siegler, 2003) as well as for considering the long-run effects of *in utero* exposure (Almond, 2006). The facts that have been forwarded to support this claim are a) the unexpected onset of the pandemic in 1918 – which rules out behavioural changes in anticipation of exposure; b) the short duration of the epidemic: the majority of deaths occurred within a few months only; c) the large proportion of the population infected; and d) the random nature of influenza prevalence and influenza mortality.

The assumptions underlying Almond's (2006) analysis have recently been challenged by Brown (2010). The main problem, according to Brown, is that US participation in World War I lead to selection issues in childbearing in and around 1919: fathers in the "treatment group" are likely to be older, less educated and less healthy than fathers from surrounding cohorts. An empirical analysis using census data confirms that parents of the 1919 cohort were indeed different: not only were they older and less educated, they were also less likely to be of Caucasian origin. When taking these differences into account, Almond's estimates become smaller in magnitude and in many cases statistically insignificant.

Even though Brown raises valid concerns, it is unclear to what extent they also apply to the current study. Even though the First World War also led to mobilisation and subsequent demobilisation in Sweden, the disruption caused is of less importance when the short-term impact of the pandemic is concerned.

As mentioned above, it is unlikely that people had the information required to adapt their behaviour in anticipation of the pandemic. However, in order to conclude that the epidemic was an exogenous shock, we also need to consider the possibility that there were systematic differences in the precautions taken in response to the pandemic. Therefore, we now briefly discuss the literature on the determinants of the influenza during the pandemic.

Garrett (2008) analyses the determinants of influenza incidence in the U.S. and finds that even though densely populated areas in general have higher influenza mortality, there is no correlation between 1918 *excess* mortality and population density.[4] Interestingly, this finding

[4] A related study from New Zealand (McSweeny et al., 2007) concludes that rural areas were less heavily affected

goes against the evidence from Sweden, where many sparsely populated areas were among the most heavily affected, whereas the more densely populated regions tend to be in the middle in terms of influenza mortality. However, these differences are to some extent attributable to age structures in the population. Garrett also analyses differences in incidence by race and finds that non-whites and people on lower incomes were more heavily affected. Interestingly, this point again contrasts with findings from Nordic countries, where the mortality rates of ethnic minorities were found not to deviate a great deal from those of the majority population (Mamelund, 2003).

One particularly relevant study is Mamelund (2006) that considers socio-economic determinants of influenza mortality in the Norwegian capital Oslo (then *Kristiania*). Using register data on influenza mortality, Mamelund estimates the importance of variables such as age, marital status, socio-economic status and quality of housing. Although there are significant class differences in influenza mortality, these appear to be driven more by location than by class itself. Marital status also appears to be insignificant. In a related study, Chowell et al. (2008) consider socio-demographic and geographical patterns in the transmissibility and mortality impact of the epidemic in England. They fail to find an association between influenza mortality and measures of population density or residential crowding.

3 Sweden in the early 20th century

One hundred years ago, Sweden was a radically different society from today. Following a surge in economic liberalisations in the second half of the 19th century, the country had evolved into a modern capitalist state with strong institutions. These reforms included trade liberalisation, modern patent laws, and the introduction of joint-stock companies (Bergh, 2007). Administrative reforms, such as the modernisation of the public bureaucracy also played an important part (Rothstein, 2007). These changes soon gave rise to rapid economic growth and industrialisation from the 1870s onwards. In terms of GDP growth per hour worked, Sweden even outpaced Japan during the period 1870-1970. Thus, volumes have been written about the Swedish 'economic miracle', where explanations have moved from focusing on natural resources and neutrality in the World Wars, towards the previously mentioned institutional reforms.

Since it is necessary to consider the particular economic environment which Sweden repre-

by the 1918 influenza; however, since they fail to control for the age profile this finding is not very informative.

sented when the influenza pandemic struck in 1918, we have devoted this section to presenting an overview of the general economic and political conditions in Sweden during and shortly after the First World War. After that, we will provide an overview of the spread of the influenza epidemic in Sweden.

3.1 General economic conditions

The first half of the 20th century was characterised by rapid industrialisation. At the turn of the century, Swedish society was still largely agrarian: according to the 1900 census, 53 per cent of the population earned their living from agriculture and 29 per cent from manufacturing (SCB, 1907). By 1910 the corresponding figures were 49.4 to 32.3 (SCB, 1917), and in 1920 the proportions were 44 per cent for agriculture and 35 per cent for manufacturing (SCB, 1926). In 1930, finally, 39.4 per cent of the population earned their living from agriculture, compared to 35.7 per cent for manufacturing (SCB, 1936). Thus, structural change occurred at a relatively even pace during these three decades.

Sweden's transformation into a modern industrialised country was largely trade-driven: for most of the period starting in 1870, the growth in trade outpaced the growth in GDP. However, the period on which we are focusing in this paper actually witnessed a stagnation of trade as a proportion of GDP – mainly due to the First World War, crises and revolutions in neighbouring countries and then the protectionist policies that were becoming increasingly common from the 1920s onwards.

In Figure 1 we have plotted total Swedish exports alongside the exports to some key trading partners during the period 1910-1930, expressed in 1917 crowns. According to Statistics Sweden, the numbers are only comparable over time during periods with relatively stable prices, which is certainly not the case for the period under consideration. Thus, the graph needs to be taken with a pinch of salt. Nevertheless, it shows that Britain and Germany consistently accounted for a large share of Sweden's exports, even if their relative roles shifted back and forth over time. Also, the Scandinavian neighbours were important trading partners throughout the period, and their trade with Sweden obviously offered some stability in an otherwise fairly volatile environment. It should, however, be pointed out that the *relative* share of exports in GDP fluctuated much less than the absolute numbers in the figure: exports never went below 14.5 per cent of GDP (1918) or above 21.5 per cent (1913) (Krantz and Schön, 2007).

In terms of labour market regulations, the period we are considering falls in between the

deregulations that were implemented in the 19th century – such as the abolition of guilds in 1846 and the introduction of free enterprise in 1864 – and the increased regulation that followed the labour movements rise to power. Thus, wages were relatively flexible and actually dropped in real terms in the 1913-17 period. From 1924 onwards, collective bargaining agreements were introduced – i.e., the type of agreements that were later to become a cornerstone of the Swedish labour market model – but initially, they were of limited importance as their legal status was codified only in 1928.

Related to labour market regulations, Du Rietz et al. (2010) look at the marginal tax wedges from the middle of the 19th century up to modern times. Sweden has a progressive tax system dating back to 1903. However, the progressivity was very limited, leading to almost similar tax wedges among different income classes. Tax wedges were steadily increasing over time, and were around 10% up to 1918. Towards the end of the Great War tax wegdes increased at a slightly higher rate. Moreover, the goverment introduced a defense tax from 1915 to 1919 for the top income earners. Finally, social security contributions where introduced in 1913 in the form of a pension contribution. However, this contributions where quite small, amounting only to 1% of taxable income.

3.2 Effects of the First World War

At the beginning of the war, Sweden, Norway and Denmark issued identically worded declarations of neutrality. The Swedish declaration had been approved uninamously by parliament. This demonstration of unity contrasts starkly with the political polarisation that was typical of the run-up to the war: in February 1914, the King had given a speech to a demonstration of 32,000 farmers who demanded increased military spending. This speech triggered a constitutional crisis and led to the resignation of the Liberal government. However, the general elections in the same year led to a new Liberal-Social Democrat coalition taking office; whereas the outbreak of the war clearly determined the issue of armament in favour of conservative forces.

[Insert Figure 1 about here]

The Swedish army was mobilised shortly after the outbreak of the war, and a ban on exports of arms, ammunition and military equipment was introduced. However, the main disruption to Swedish trade was of course caused by external forces: the naval blockade imposed by the UK

included the entire North Sea, which was declared a war zone which ships entered at their own risk. The blockade was very restrictive and, as its implementation was being stepped up, it lead to disruption in Sweden's trade with countries overseas. However, it was mainly imports that were affected. Thanks to an increased war-related demand for Swedish exports, a substantial current account surplus was built up despite an appreciation of the Swedish crown (Jörberg and Krantz, 1978). Indeed, the war years led to an almost uninterrupted boom for Swedish industry, and, in particular, production in import substitution markets flourished (Magnusson, 1996).

The war also led to increased regulation of the domestic economy. The government was given increasing powers to dispose of resources essential to the military. In 1916, new legislation authorised the government to regulate prices of groceries, fodder, fuel and clothing. A public commission was instituted with the task of rationing essential goods that were in short supply. This lead to rationing of meat, eggs, butter and fish. The consumption of sugar and some other necessities was also regulated. However, a black market evolved and thus the regulations were of limited importance in practice (Schön, 2010). The time around the First World War was also characterised by a surge in important social legislation. Even before the war, the first steps had been taken to separate child and elderly care from general poor relief. In 1914, a basic social security system had been introduced. In the same year, a public committee responsible for unemployment was formed, which was to play an important role in the shaping of active labour market policies. The intensity of reform activities of this period contrast sharply with the relative passivity of the 1920s (Jörberg and Krantz, 1978).

During the war, the gold standard was temporarily abandoned, and rapid inflation ensued. For essential goods, the price increase was 103 per cent from 1914 to 1918. In general, wages did not keep pace with these increases, but there were of course some industries that were prospering from the sudden shortfall of foreign competition. Whilst farmers enjoyed a steady increase in their incomes they also needed pay more for fertilisers and fodder – which could hardly be imported from 1916 onwards.

Despite the disruption brought by the war in some parts of the economy, the Great War provided a generally favourable economic environment to Sweden. There was a massive surge in exports (iron ore, steel, engineering products) and a huge trade surplus evolved. Shortages in imported fuels led to the electrification of industry production all over the country – which means that the competitiveness of Swedish industry improved. The agricultural sector also benefited from the shortfall in foreign competition. It was only in residential production that

investment plunged and remained low throughout the war (Schön, 2010).

However, the war gave rise to redistribution between different groups in society. Owners of capital benefited more than workers, and the gains and strains associated with the war were unevenly distributed between different sectors of the economy (Schön, 2010). It is important to keep this redistribution in mind, since it was typically reversed in the post-war slump and thus it might represent a confounding factor with respect to the regional exposure to the Spanish flu pandemic.

3.3 The Roaring Twenties

Having emerged from the Great War largely unscathed, the Swedish economy was subsequently to move on to another decade of rapid economic growth and structural transformation. However, this period of growth was interrupted by a sharp downturn in 1920-21 in which GDP decreased by five per cent in a single year. Interestingly, the industries that had benefited most from the war – such as sawmills and the iron and steel industry – were also the most hard hit by the crisis (Magnusson, 1996). There were dramatic increases in unemployment, which reached a level of 25 per cent at the peak of the crisis. However, the recovery was very quick: Swedish GDP increased by 8 per cent in 1922 and the country faced steady economic growth for the rest of the decade. During the first interwar years, the Swedish crown lost value to the dollar, but much less than other European countries. The gold standard was reinstated in 1924 (Jörberg and Krantz, 1978).

Moreover, the 1920s were characterised by fast growth in real wages: in 1930, they were at roughly twice their 1918 level, and not even in the sharp downturn of 1921 did they stop growing. Thus, the decade was also characterised by a gradual increase in returns to labour compared to capital returns (Schön, 2010). Even though unemployment remained relatively high throughout the decade it is generally believed that the fast growth in wages was partly due to the shortening of the working day to eight hours in 1919 (Jörberg and Krantz, 1978).

As previously mentioned, the iron and steel industry faced difficulties as excess capacity from the war had to be reduced and, in addition, demand for Swedish pig iron kept decreasing for other reasons (Jörberg and Krantz, 1978). This led to rationalisation and increased innovation within the industry. On the other hand, the pulp and paper industry (i.e. sawmills, pulp manufacturing and paper mills) was very expansive throughout the decade – particularly in the north of the country where it could benefit from hydroelectric power from the main rivers. In the

cities of the south, a growing shipbuilding industry started producing large vessels for the already very expansive Swedish and Norwegian shipping industries. Finally, the 1920s witnessed a rapid growth in services, in which employment growth was slightly higher than for manufacturing and construction (Schön, 2010).

3.4 The Spanish Flu Pandemic

With respect to the number of deaths caused, the Spanish flu is one of the most severe calamities ever to affect Sweden. Unequalled by any epidemic in modern times, the flu killed close to 38,000 individuals in total, representing almost one per cent of the population. As in other parts of the world, flu prevalence rates were much higher. According to official records more than ten per cent of the Swedish population got the flu in 1918 (Medicinalstyrelsen, 1920), but case studies suggest that incidence was as high as 75 per cent in some areas. There is consequently large variation in the estimates of the number of infected individuals, but generally it is believed that mortality rates amongst those infected approached 2 per cent.

The first case of the Spanish flu in Sweden was reported in the southern part of the country in late June 1918; in this case, the virus was carried from Oslo, Norway. At the beginning of July, further cases were reported to have been introduced from Germany, Austria, Britain and Denmark (Medicinalstyrelsen, 1920). Towards the end of the very same month not all regions were yet affected, but in early August an increasing number of cases are also reported to have died from the flu in the northern provinces. However, as shown by Figure 2, until the late summer months of 1918 there was no reason to be concerned about elevated influenza mortality in Sweden. During the first seven months of 1918, 148 deaths due to influenza were reported, which is clearly below the corresponding figure for 1917 (190). Besides, the number of pneumonia deaths added up to 3,602 during the same period, compared to 3,736 the year before. Yet, once the situation changed during August and September, it did so with a terrifying speed.

[Insert Figure 2 about here]

Figure 3 shows influenza mortality rates in Swedish counties in 1918 (per 100,000 inhabitants). Clearly influenza mortality varied widely across counties, with some areas experiencing almost three times higher mortality rates than others. In particular the regions *Jämtland* and *Västernorrland*, and villages along the main inland railway line *Inlandsbanan*, were severely hit. The high mortality rates in the remote northern areas have, in part, a demographic explanation

as these regions tended to have a young population at the time and the Spanish flu mainly affected people between 15-40 years. However, following the same line of reasoning as to why the young population was affected to a larger extent than the older, it has also been hypothesised that the high regional variation in mortality rates may be explained by remoteness, and that people living in these areas had less immunological protection against the virus as they generally had been less exposed to earlier flu waves. Regarding immunisation it has moreover been hypothesised that the W-shaped mortality distribution of the Spanish flu may relate to the exposure of the Russian flu in 1889-1890.

[Insert Figure 3 about here]

As discussed above, there was some variation in how different industries were faring during and after the First World War. Since different regions tend to be specialised in different industries, these fluctuations, which largely coincide with the influenza, may become confounding factors in our analysis. Thus, in Table 1, we have tabulated all the regions, their influenza exposures and some key statistics from the 1910 census. Regions are ranked according to their 1918-20 influenza exposure. Interestingly, there is almost no correlation between sectoral composition and Spanish flu mortality: the population-weighted correlation coefficient for flu mortality and agricultural share is 0.075, and the corresponding correlation coefficients for industry and commerce are -0.086 and 0.006, respectively. Thus, it seems safe to conclude that the spread of the influenza virus was largely unrelated to the economic conditions in the regions. Nevertheless, we will try below to deliver further evidence that the regions were following a common time trend prior to the outbreak of the pandemic.

[Insert Table 1 about here]

Normal flu waves affecting Sweden typically have their outbreak and peaks in February and March, but the Spanish flu reached its peak in October and November. During these two months only, the number of victims of the epidemic reached 20,000 individuals. Another, less severe, wave hit the country in March 1919 and new waves appeared until early 1920. Due to the fast spread of the disease in the North, the national government tried to mobilize medical resources to these areas. Moreover, local authorities took actions to limit the spread of the disease and implemented various public health measures, such as the banning of public gatherings, closure of theatres and of schools. These extraordinary actions however had limited effectiveness as the

virus was transmitted through the air. In fact, inhaling the virus coming from another person sneezing or coughing was actually enough to get infected.

Figure 4 provides an overview of the timing of the influenza in Sweden. The curves in the diagram show the ratio between 1918-20 monthly flu incidence and incidence in a 'normal' year (in this case, the averages of 1915-17). The three dashed curves show the progression of the epidemic among poor people in the three largest cities; and these figures are contrasted with the situation in the entire population in the rest of Sweden. Thus, the figure gives an indication of the socio-economic gradient of the influenza. Accordingly, the poor people in Malmö and Stockholm experienced a slightly lower increase in incidence rates compared to the rest of the country, whereas poor people in Gothenburg were more severely affected.[5]

[Insert Figure 4 about here]

3.5 Assessment

The purpose of this section has been to give a general overview of the environment in which the Spanish flu pandemic was spreading in 1918, with a particular focus on potential threats to the identification strategy employed in this paper. We have identified two main threats to the identification strategy which merit special attention. Firstly, even though Sweden soon rebounded from the crisis of the early 1920s, it is clear that it had asymmetric effects between urban and rural areas: in particular, agriculture suffered from a decline in prices when import markets opened after the war. Ironworks and sawmills, which were typically in the countryside, were also particularly badly affected. However, we have shown above that there was no clear urban-rural divide in the 1918 influenza pandemic. A related issue is that the different regions may have been specialised in different sectors of production, and these differences may not be fully captured by the urban/rural dichotomy. Since we have seen that the industries that benefited most from the war also had a less favourable evolution afterwards, it is essential to establish that the sectoral composition did not lead to counties already diverging during the First World War. This point calls for a careful investigation of the common time trend assumption for all outcome variables.

[5] Amongst poor people in Malmö, the average incidence rate was 9.1 times higher than in a normal year, in Stockholm it was 7.5 times higher, and in Gothenburg 14 times higher. The corresponding figure for the rest of Sweden was 9.73. However, the actual levels of rates are not comparable across locations, since better access to medical services automatically leads to higher recorded incidence and prevalence rates.

4 Data and Variables

The data used in our analysis comes from high-quality administrative records. Sweden has a long tradition of collecting and providing official statistics. Statistics Sweden was founded in 1858 and from 1911 and onwards the bureau has provided the printed series *Sveriges officiella statistik* with information on various issues on a yearly basis. In addition, most public authorities have a convention of providing official statistics related to their activities and assignments.[6]

There are two sources of county-level influenza statistics available for Sweden and they differ to some extent. We use data from Statistics Sweden, which are generally believed to be of high quality and more accurate compared to the influenza statistics provided by *Medicinalstyrelsen* – the authority responsible at the time for national health services. Medicinalstyrelsen's data tend to underestimate the number of cases and also report deaths by place of death and not place of residence. Statistics Sweden, on the other hand, implemented more detailed and stricter reporting procedures in 1911, which improved the reporting from rural areas. [7]With respect to accuracy, reporting from urban areas were most likely, however, superior to reporting from rural areas.

The number of reported deaths following from influenza in each county is reported on a yearly basis. As described in section 5.1 below, we use this information together with monthly statistics on people hit by the influenza, collected from Medicinalstyrelsen, to derive our treatment variable. Most likely incidence data are of a lower quality than the mortality data due to the fact that a visit to a physician was required to be recorded. However, governmental historical records (see e.g. Influensabyrn 1918) suggest that people did visit health care centres when they had the flu and that the pandemic clearly increased the demand for general practitioners.

In baseline regressions we use data for the time period 1912-1930 and focus on three economic outcomes available at a yearly basis. The first outcome variable is **capital incomes** per capita defined as incomes from e.g. asset yields, rents and dividends taken from official tax records (Statistisk Årsbok).[8]. We also use **earnings** per capita, referring to all earnings from employment and pensions per capita collected from the same source. The third outcome variable

[6] Official data for the time period covered in our analysis is available in hard copies and sometimes as scanned documents. The information used in this paper has been digitalized by the authors and research assistants.

[7]Clergymans had to make monthly reports to Statistics Sweden on the likely cause of death of persons in cases where no doctor had been involved. These notes and reports were then reviewed and confirmed by a GP who reported the final cause of death to the bureau, for details see the introductory chapter in Ddsorsaker 1911(SCB, 1911).

[8]All monetary outcome variables are adjusted to real measures using 1917 as base year. The measure used for adjusting the variables was the Swedish CPI obtained from Statistics Sweden

in our analysis is **poverty rates**, referring to the number of inhabitants in public poorhouses as a proportion of the total population.[9] People who were not able to support themselves or could not be supported by their (extended) family were eligible for the public poorhouses. Inhabitants were supported with housing, food, clothing and medical care. Data on poverty rates are collected from the yearly publication Fattigvården (for unknown reasons this report was not published in 1919 and poverty records for this particular year is missing in our data set). In order to avoid spurious effects of the influenza working through the denominators of the per capita variables, we use the average county population over the year throughout.

According to the yearly documentation and summary reports from our data sources, all variables seem to have been consistently collected across the time period of interest, and, reassuringly, we have not noted any change in any of the definitions of the above indicators that could influence our results. Descriptive statistics for all variables are provided in Table 2.

[Insert Table 2 about here]

A potential obstacle to the internal validity of our results relates to the course of disease and the fact that the flu virus attacked the lungs. To circumvent the potential bias following from deaths caused by the flu being recorded as caused by pneumonia – which, according to Figure 2, should not be a big problem – we also use information on influenza and pneumonia from Statistics Sweden to derive a second version of our treatment variable. This alternative treatment variable is based on the sum of excess deaths due to influenza and pneumonia.

Yet another concern is that the estimated effect of influenza mortality may actually be capturing long-lasting effects of influenza *prevalence*. The growing literature on effects of *in utero* exposure provides but one example of how the effects of the influenza might manifest themselves at the regional level (see e.g. Almond, 2006). Having access to regional data on the number of people hit by the influenza, we include this variable in some separate specifications as a robustness check. As noted above, measurement errors in this variable might be problematic. However, if we are willing to assume that the data were collected consistently over time in each region, the data will still be informative. Figure 6 shows the relationship between excess morbidity and excess mortality at the regional level. Even though the variables are clearly positively related, they are not as strongly correlated as one might expect: in the year 1918, the correlation coefficient for flu is 0.43, and for flu and pneumonia it is 0.46.

[9] It should be noted that although the variable used includes dependants of adult poorhouse residents, it does not include orphans, as these resided in special orphanages.

[Insert Figure 6 about here]

Another threat to the internal validity of our estimates is arguably the volatility of the world economy during the period of our study. We have seen in Section 3 above that the Swedish economy appears to have weathered crises in the surrounding world relatively well. Nevertheless, the Spanish flu pandemic was preceded by the First World War and the Russian Revolution, and largely coincided with the 1918-19 revolution in Germany (one of Sweden's main trading partners) and the civil war in Russia (including Finland, bordering Sweden). If these and other external events caused serious disruption to the Swedish economy, and if these influences were spatially heterogeneous in a way that coincides with the exposure to the epidemic, then our estimates of the effect of the epidemic may be confounded by these influences.

In order to check the robustness of our findings we take the volatility of the economic environment into account by also including information on GDP in other countries in our main specifications. Information on GDP and population size is available for the 27 countries which together represent virtually all of the Swedish exports of the time. Our trade variable is derived in two steps. First, we estimate a partial gravity function[10], where Swedish exports to other countries were explained with reference to their distance, their GDP and their GDP per capita:

$$\ln\left(PX_{it}\right) = \delta_0 + \delta_1 \ln\left(GDP_{it}\right) + \delta_2 \ln\left(GDP_{it}/Pop_{it}\right) + \delta_3 D_i + \upsilon_{it} \tag{1}$$

where PX_{it} are Swedish exports to country i in year t, GDP_{it} is the gross domestic product of country i in year t, Pop_{it} is the population size, and D_i is the distance from Stockholm to the capital of country i. We estimate equation (1) using the random effects estimator. The results indicate that distance and total GDP are strongly significant, whereas GDP per capita is marginally significant.

In the next step we generate the variable $\widehat{PX}_{jt}$ for each county j. This variable refers to the total exports that would be expected in year t if Sweden were located at the centroid of county j:

$$\widehat{PX}_{jt} = \sum_{i=1}^{27} e^{\hat{\delta}_0} GDP_{it}^{\hat{\delta}_1} \left(\frac{GDP_{it}}{Pop_{it}}\right)^{\hat{\delta}_2} D_{ij}^{\hat{\delta}_3} \tag{2}$$

where D_{ij} now represents the distance between county j and country i. Clearly, $\widehat{PX}_{jt}$ has no

[10] Anderson (1979) provides the first theoretical foundation of a gravity trade model; cf. Rose (2000) for an overview of the literature.

obvious interpretation in economic terms, partly because equation (1) is only half a gravity equation. Nevertheless, we believe that this variable goes a long way towards controlling for asymmetric shocks related to the business cycle and major events in neighbouring countries.

5 Econometric Approach

In this section, we first present our main treatment indicator, then discuss the assumptions required for identification. Next, we present our regression equations and then we discuss how to estimate standard errors in the presence of non-spherical disturbances. Finally, we present the methods we use to support our identification strategy.

5.1 Defining the Treatment Indicator

Our analysis is conducted at the level of Swedish counties (Swedish: *län*) of which there were 25 at the time of the Spanish flu. As already mentioned, the incidence and mortality of the pandemic exhibit considerable variation across regions. The main assumption underlying our analysis is that the regional exposure to the Spanish influenza represents an exogenous shock and that regions that were affected particularly hard would have followed the same time trend as other regions in the absence of the pandemic. Thus, we define treatment as the degree of exposure to the pandemic measured by total excess regional influenza mortality through the years 1918-20. In our baseline specifications, we furthermore assume that the effects of Spanish flu mortality is constant over time and a linear function of the excess mortality.

However, since the outcome variables are measured annually, we need to correct for the timing of the flu in some cases. Most importantly, since the 1918 wave of the epidemic reached its peak only in October and November, it is clear that it would not have a full effect on the economy in that year. Unfortunately, we do not have monthly mortality data at the regional level. However, given that the time period between infection and death was so short (typically 6 – 11 days), we can actually approximate the timing of the fatalities using the timing of influenza incidence.

Thus, we introduce the following notation: yearly flu mortality in county i is denoted m_{it}; and monthly flu morbidity denoted p_{it}^{j} – where t is the year and j is the month. We have defined both variables as proportions of the county population at the end of year $t-1$. Furthermore, we define the corresponding variables in a 'normal' year (inferred from the averages from the

period 1915-17) as m_{i0} and p^j_{i0}.

Using these variables, we can define the **effective excess mortality** m^e_{it} in year $t > 1917$ as

$$m^e_{it} = (m_{it} - m_{i0}) \frac{\sum_{j=1}^{12} \left(\frac{p^j_{it} - p^j_{i0}}{2} + \sum_{k=1}^{j-1} \left(p^k_{it} - p^k_{i0} \right) \right)}{12 \sum_{k=1}^{12} \left(p^k_{it} - p^k_{i0} \right)} = (m_{it} - m_{i0}) \frac{\sum_{j=1}^{12} (12.5 - j) \left(p^k_{it} - p^k_{i0} \right)}{12 \sum_{k=1}^{12} \left(p^k_{it} - p^k_{i0} \right)} \tag{3}$$

In words, $m_{it} - m_{i0}$ is the excess mortality rate on an annual basis. The denominator of the next term normalises weights such that we adjust for the fact that p^j_{it} captures morbidity and not mortality. In the numerator, we first have the 'excess morbidity rate' of the current month: we divide it by two to correct for the fact that not all cases appear at the beginning of the month. The second term in the numerator captures the cumulative effect of influenza exposure in previous months.

Having thus defined the excess flu mortality within a certain year, we can calculate the cumulative excess mortality at an annual basis. Denoting this variable w_{it}, it will be defined as

$$w_{it} = \begin{cases} 0 & \text{if } t < 1918 \\ \sum_{j=1918}^{t-1} (m_{ij} - m_{i0}) + m^e_{it} & \text{if } t \in [1918, 1920] \\ \sum_{j=1918}^{1920} (m_{ij} - m_{i0}) & \text{if } t > 1920 \end{cases} \tag{4}$$

where, notably, previous years are represented by m_{ij}, not m^e_{ij}: in the following year, we obviously do not need to correct for the timing of period $t-1$ deaths. The treatment indicator has been adjusted for the proportion of individuals aged 0-40 according to the 1920 census.

These equations might not seem very intuitive but, in fact, we are simply integrating the number of cumulative deaths over time. This aspect is emphasised in Figure 7, where we have plotted the treatment variable together with the cumulative number of excess deaths. Clearly, the treatment variable adds the integral of new deaths occurring during the current year to those deaths that occurred in previous years. This way, the probable impact of these deaths on annual data are captured more convincingly.

[Insert Figure 7 about here]

5.2 Identifying Assumptions

The method we use is an extension of the standard difference-in-differences estimator; our extension is simply that we need to allow for varying treatment intensity. Thus, the functional form imposed adds a further assumption to the standard set of assumptions, and it should clearly be formally tested. In addition, it is important to spell out the standard DID assumptions since it is possible to assess their credibility (Lechner, 2010).

In order to gauge the plausibility of the assumptions, it is useful to introduce some additional notation. As is standard in the programme evaluation literature, we refer to the concept of *potential outcomes*, although in this setting the potential outcomes have a support along the entire range of the treatment variable. Also, we denote the vector of treatment intensities for all time periods for a single individual i by $\mathbf{w}_i$. Moreover, we denote possible conditioning variables, that may possibly be included in the regression equations below, by X_{it}. Finally, we denote the **potential outcome function** by $Y_{it}(\cdot)$. Thus, we may state our first assumption as:

Assumption 1 (SUTVA)

$$y_{it} = Y_{it}(\mathbf{w}_i) \tag{5}$$

The SUTVA assumption states that a) only one amongst the potential outcomes is observed, and b) there are no relevant interactions between units (i.e. only $\mathbf{w}_i$ matters in the determination of unit i's outcome). For most variables that we consider in this study, the assumption seems plausible. It is unlikely that the influenza exposure in one county had a strong impact on economic outcomes in other regions, especially if they are far away from each other. It should furthermore be noted that the assumption is reconcilable with *spatial autocorrelation*, i.e., that the residual variation in the outcomes is spatially correlated.

Next, we introduce the *Common Trend* assumption, which is absolutely essential for the DID method:

Assumption 2 (CT)

$$Y_{it}(\overline{w}_t) \equiv Y_{i0}(0) + \overline{Y}_t - \overline{Y}_0$$

This assumption, which is somewhat stronger than in the standard DID setting, states that differences in time trends between counties are only attributable to differences in treatment exposure. The assumption does not require that counties are at similar levels, but only that the

dynamics of the outcomes would have been similar if the exposure to treatment had also been similar. This assumption may be sensitive to functional form assumptions, and for this reason we always consider outcomes in levels as well as in natural logarithms as a robustness check. Intuitively, the logarithmic specification appears to be the most appealing one. Since most of our outcome variables are defined in Swedish crowns (SEK) per capita – and since furthermore there were disparities in terms of living standards between individual counties – it seems reasonable to assume that a random shock in the mortality rate has the same proportional impact in all regions in the country.

In our view, the CT assumption is the most problematic of them all, since it is possible that diverging time trends among regions confound the estimated effects of the pandemic. The assumption is not directly testable, but we can assess the extent to which it is plausible by looking at periods during which there were no significant differences in influenza mortality between counties. We will provide this analysis in two steps below: firstly, we will provide visual evidence of how the outcomes evolved in different regions, sorted by degree of affectedness, before the pandemic. Secondly, we will run regressions using a 'placebo' treatment, in which we counterfactually assume that Sweden was hit by the pandemic in a year prior to 1918.

Next, we consider the *No Effect Prior to Treatment* assumption:

Assumption 3 (NEPT)

$$Y_{it}\left(\mathbf{w}_i\right) = Y_{it}\left(w_{it}\right) \tag{6}$$

This assumption rules out the possibility that the excess influenza mortality in one year has an impact on (potential and observed) outcomes in previous years. It should be noted, however, that due to the way w_{it} has been constructed, the converse does not hold: the excess mortality in one year may very well have an impact after that year – indeed, this is one of the main issues analysed in this paper. The NEPT assumption rules out economic effects based on an *anticipation* of the pandemic. This assumption seems very reasonable for the years preceding 1918, since the first report worldwide on the pandemic appeared only in January of that year. Once the influenza had started spreading, an anticipation of further deaths in subsequent years may of course in theory have had an impact on the behaviours of economic agents; however, this aspect seems very unlikely to matter in practice. Nevertheless, we allow for this possibility in separate regressions where we use only the flu deaths occurring in 1918 as our treatment variable.

Since a rich set of possible conditioning variables are available, we could also have included covariates, denoted X_{it}. However, these variables would need to satisfy an exogeneity assumption which might not be warranted:

Assumption 4 (EXOG)

$$X_{it}\left(w_{it}\right) = X_{it} \tag{7}$$

In short, the exogeneity assumption states that conditioning variables are unaffected by the treatment. This seriously limits the number of conditioning variables that we may consider. The assumption is obviously going to hold for variables which do not vary over time, but these are washed out by the fixed county effects in any case. As far as time-varying covariates are concerned, it is difficult to think of any economically relevant variable which is not also potentially affected by the pandemic. Thus, we only condition on time and county fixed effects in what follows. In robustness checks, we also include some variables that are plausibly exogenous (influenza morbidity, GDP of neighbouring countries).

Finally, one additional assumption is needed due to the variable treatment intensity in our case. This is the **mean independence** assumption:

Assumption 5 (MI)

$$\mathbb{E}\left[\left(Y_{it}\left(w_{it}\right) - Y_{it}\left(\bar{w}_t\right)\right)\left(w_{it} - \bar{w}_t\right)\right] = 0 \tag{8}$$

where $\bar{w}_t$ denotes the population mean of the treatment variable in year t. The mean independence assumption states that the exposure to treatment $(w_{it} - \bar{w}_t)$ is uncorrelated to the effects of treatment $(Y_{it}\left(w_{it}\right) - Y_{it}\left(\bar{w}_t\right))$. It is important to note that this assumption allows the treatment variable to be related to the outcomes in a certain county: it only requires that the excess influenza mortality is unrelated to the *effect* of that mortality. Thus, our assumption would probably be untenable if we had a situation where mortality was correlated with individuals' (and thus regions') future economic prospects. On the other hand, considering the limited possibilities individuals had of influencing their survival prospects, such a scenario seems highly unlikely.

Next, we also define the particular treatment effect we are seeking to identify. Since we have no particular interest in the functional form of the relationship, we simply define the treatment effect as the average change in the outcome variable per unit change in the treatment variable:

Definition 1 (Average Treatment Effect)

$$ATE_t \equiv \mathbb{E}\left(\frac{Y_{it}\left(w_{it}\right) - Y_{it}\left(\bar{w}_t\right)}{w_{it} - \bar{w}_t}\right) \tag{9}$$

In what follows, we will also make use of the notation $TE_{it} \equiv \left(Y_{it}\left(w_{it}\right) - Y_{it}\left(\bar{w}_t\right)\right) / \left(w_{it} - \bar{w}_t\right)$ for individual treatment effects.

5.3 A Linear Specification

For all the various outcome variables we consider, our main baseline specification is

$$y_{it} = \alpha_i + \beta w_{it} + \lambda_t + \epsilon_{it} \tag{10}$$

where y_{it} is the outcome variable (i.e. capital returns, earnings or poverty), α_i is a county fixed effect, λ_t is a year fixed effect, and ϵ_{it} is a residual disturbance. It is straightforward to show that an OLS estimate of β captures the treatment effect if the assumptions above are fulfilled (a proof is provided in the Appendix).

Lemma 1 *Suppose assumptions SUTVA, CT, NEPT and MI are fulfilled. Then, the OLS estimate of β identifies the average treatment effect.*

In an alternative set of specifications, we also allow the impact of the influenza pandemic to vary over time:

$$y_{it} = \alpha_i + \beta w_{it} + \gamma w_{it} \mathbf{1}\left(t > \tau\right) + \lambda_t + \epsilon_{it} \tag{11}$$

where γ captures treatment effect heterogeneity over time, and $\mathbf{1}\left(t > \tau\right)$ is a dummy variable indicating that the year is after some cutoff year denoted τ.

The placebo regression will take a very similar form:

$$y_{it} = \alpha_i + \delta w_{i,t+3} + \lambda_t + \epsilon_{it} \text{ if } t < 1918 \tag{12}$$

In words, we estimate the 'effect' of a counterfactual placebo epidemic, which is assumed to have occurred in the years 1915-17 with the incidence rates of 1918-20. If the placebo parameter δ is precisely estimated and close to zero, it can be seen as evidence for the common time trend.

Moreover, it will give us an indication of whether spatial autocorrelation is a problem in the dataset.

5.4 Estimating Standard Errors

Inference in DID models has attracted considerable attention in the literature over the past decade. Particular attention is devoted to two issues related to the estimation of standard errors: autocorrelation and common group effects. Since we use data aggregated at the regional level throughout, common group errors as discussed by Donald and Lang (2007) are unlikely to represent a problem. On the other hand, our estimates are based on relatively long panels – we have as many as 20 time periods in some specifications below – so the possibility of autocorrelation needs to be taken into account.

In a seminal paper, Bertrand et al. (2004) discuss the problems associated with autocorrelation in difference-in-differences studies and also compare different solutions. Whenever variables are (positively) serially correlated, DID estimates not correcting for autocorrelation are likely to lead to over-rejection, i.e., the null hypothesis of no treatment effect is rejected too often. Several solutions are available to handle this problem, each one of them with their specific drawbacks. Given the low number of counties available, only two alternatives seem appealing for this study: to rely on the GLS estimator originally suggested by Kiefer (1980), or to reduce the time dimension by collapsing the data. The GLS approach is based on estimating the full covariance matrix, allowing for any pattern of correlation between time periods. In a recent paper, Hausman and Kuersteiner (2008) analyse the properties of this GLS estimator. Their main conclusions are that a FGLS procedure generally outperforms procedures where the time dimension is reduced by aggregating observations. They do, however, find that there might still be size distortions when the number of observations is small – and they suggest a size correction that is shown to have better properties. Even though their size correction is promising, we decided not to follow that route here. The estimated correlation matrix exhibited positive autocorrelation in the short term but negative autocorrelation in the long term – and, thus, standard errors often turned out smaller than in the original OLS specification.

Thus, we decided to reduce the number of time periods instead, by collapsing the data. Hence, in a set of robustness checks, we shrank the time dimension into five or less time periods, and checked whether results were robust to this modification. The estimating equations remain the same as those above, but we now used a collapsed version of the outcome variable, defined

as follows:

$$\tilde{y}_{it} = \begin{cases} \frac{1}{T_0} \sum_{s=t_0}^{1917} y_{is} & \text{if } t = 1917 \\ y_{it} & \text{if } t \in [1918, 1920] \\ \frac{1}{T_1} \sum_{s=1921}^{t_1} y_{is} & \text{if } t = 1921 \end{cases} \tag{13}$$

where T_0 is the number of time periods before 1918; t_0 is the first year covered by the panel; T_1 is the number of time periods after 1920, and t_1 is the last year covered by the panel. Obviously, the treatment variable $\tilde{w}_{it}$ is defined analogously:

$$\tilde{w}_{it} = \begin{cases} 0 & \text{if } t = 1917 \\ w_{it} & \text{if } t \in [1918, 1921] \end{cases} \tag{14}$$

Thus, we require estimated effects to be robust to this change in specification.

6 Results

In this section, we present the main results from our analysis. First, we present visual evidence supporting the common time trend hypothesis for the main outcome variables. We then turn to estimates of the effects of the pandemic.

6.1 Common Time Trend: Visual Evidence

As mentioned above, our case differs from the standard DID setting in the sense that we have more than two degrees of treatment intensity, and hence the counties included in the analysis do not form two distinct groups. However, in terms of the total excess influenza mortality experienced over the entire 1918-20 period, we may distinguish two different strata of exposure. Most counties fall within the range of 440-635 additional deaths per 100,000 population. Above that, there is a smaller group of seven counties which experienced between 655 and 1017 additional deaths. To provide some visual evidence concerning the common time trend assumption we present visual evidence contrasting these two groups.[11] In the graphs that follow, counties are weighted by their 1917 population size, and all monetary variables are expressed in 1917 crowns (adjusted according to the country-wide CPI).

[11] Allowing for more groups did not make much difference to our results, but made the figures more difficult to read.

Figure 8 presents evidence for capital income per capita. The brown curve pictures the growth in capital incomes (compared with the 1912 level) for counties which were particularly hard hit by the epidemic. The blue curve plots the corresponding series for the less severely affected counties, while the dotted curves show the 95 % confidence intervals. The figure indicates that the common time trend is a reasonable assumption before the pandemic hit: the curves are very close and their confidence intervals have a significant overlap. During and after the pandemic, the two groups diverge. The group of counties more severely affected by the pandemic appears to suffer a quite significant relative loss in capital incomes due to the pandemic compared to the less affected group. At the same time there is a rapid increase in capital income per capita, so it is unclear whether this observation holds for closer scrutiny.

[Insert Figure 8 about here]

Figure 9 presents the corresponding information for earnings per capita. For this outcome variable, the common time trend assumption seems slightly harder to maintain: the two curves are somewhat further apart but, in fact, after 1913 they are fairly parallel. Also in this case, the figure suggests that counties more hard hit by the Spanish flu experienced slower earnings growth during and after the pandemic.

[Insert Figure 9 about here]

Next, we provide visual evidence for the poverty variable. As manifested in Figure 10 the common time trend assumption appears to be satisfied. The proportion of the population that was living in public poorhouses appears to follow a reasonably parallel trend before the pandemic and then starts diverging from 1921 onwards. The more severely affected counties experienced faster growth in poverty rates: on average, there is an increase from 4.6 per cent in 1921 to 7.1 per cent in 1930, whereas the corresponding increase for the rest of the counties is 4.2 per cent and 5.5 per cent respectively.

[Insert Figure 10 about here]

Figure 9 suggests that more strongly affected counties experienced slower earnings growth, and Figure 10 suggests that poverty rates were increased by the pandemic. Obviously, both these observations might be driven by a change in poverty rates. To overcome this potential obstacle, we define an alternative earnings variable, where total annual earnings at the county

level are divided by the number of inhabitants who are not poor. Figure 11 provides visual evidence for the modified outcome variable.[12] The common time trend assumption now appears to be even more plausible than for the original earnings variable, but otherwise no important changes are discernible.

[Insert Figure 11 about here]

In conclusion, there are no blatant violations of the common time trend in our data, and the pandemic appears to have had an impact on all outcome variables of interest. Clearly, however, the above evidence is too crude and summaric to provide a reliable estimate of the effects. This applies in particular for capital income and earnings per capita, which seem to have increased rapidly throughout the country during the epidemic. Hence, we now turn to more rigorous regression-based evidence.

6.2 Regression Analysis

In the regression analysis each outcome variable is analysed in eight different specifications: first, we estimate the overall impact of the pandemic using the entire sample. Second we allow the effect to be different *during* and *after* the pandemic. Third, we test if results are sensitive to the inclusion of regional morbidity in two different ways. Next, we examine the role of the economic and political turmoil of trading partners. Finally, we check the validity of our results by running placebo estimations. Restricting the sample to the years before 1918, we assume counterfactually that the pandemic struck Sweden between 1915-17 and estimate the effects of this 'placebo' intervention, using county-specific incidence rates of 1918-20, on economic outcomes. Clearly, if the common trend assumption is true, the placebo pandemic should not have any effect on outcome variables. Finally, we address the issue with autocorrelation by presenting estimates where the dataset has been collapsed into five time periods: before, 1918, 1919, 1920 and after. [13]

It is well known that the DID estimator is sensitive to functional form assumptions. In our case, the natural alternatives are to use either levels or logarithms of the outcome variables. Since the different counties are at very different levels at the outset with respect to the outcome variables, a logarithmic specification seems preferable. However, as a test of robustness we also

[12] Since the poverty variable is missing for 1919, we used linear intrapolation for the denominator in 1919.

[13] One concern already mentioned is whether or not the pandemic came as a surprise. In order to address this we estimated the effect including only the treatment of 1918. However, our results were not affected by this change.

provide estimates for the outcome variables in levels in the Appendix. Similar results from the different estimations will add credibility to our evidence on the economic effects of the Spanish flu, while diverging results will suggest that the functional form deserves particular attention. [14]

Table 3 presents the results for capital income per capita. The first column presents the overall effect of the pandemic, averaging over all years from 1918 onwards. According to our estimate, each additional death per 100 inhabitants was associated with a reduction in capital income per capita by 0.074 per cent. Since the median total mortality rate between 1918-20 was 0.541, this coefficient indicates that the median county suffered a reduction in capital incomes by 40 per cent due to the pandemic. In order to avoid extrapolation, it is reasonable to compare the 25th and the 75th percentile, with an incidence of 0.291 and 0.616 respectively. The difference between these two counties would correspond to a reduction in capital incomes per capita by 24 per cent, which is still a fairly large amount.

In the second column, we contrast the effects during (1918-20) and after (1921-30) the pandemic. Clearly, parts of the effect are discernible during the pandemic itself, whereas there is an additional effect kicking in afterwards. Columns three and four control for morbidity and cumulative morbidity respectively. Throughout these different specifications the variation of our estimate for the treatment effect is very limited. The fifth column presents estimates controlling for the export shocks (see the discussion before Figure 1 for further details). Finally, column six allows for a 'placebo epidemic'. This estimate is nowhere near statistical significance and it is very precisely estimated. Thus, our observation from Figure 8 is confirmed and the common time trend assumption is maintained.

In columns seven and eight, we collapse the time period into five in order to reduce problems related to autocorrelation. The estimates in these column clearly indicate that autocorrelation is an issue. Nevertheless, the effects observed after the epidemic are still significant at the five per cent level.

[Insert Table 3 about here]

Table 4 provides estimates for the earnings variable. For this variable, there is much less

[14] A second concern is the classification of excess deaths. Deaths from the Spanish flu could be registered either as flu deaths or pneumonia deaths, since pneumonia is also a symptom of the Spanish flu. In terms of cases the flu cases increased substantially during the Spanish flu (around 0.5% of the population), while the increase in pneumonia deaths was much smaller (around 0.01% of the population). As a robustness check we also included pneumonia deaths in the treatment variable, which however did not change our results.

evidence of an effect of the pandemic. The point estimate of the overall effect is −0.140, which, according to our previous comparison, would imply a reduction in wages by 7.5 per cent in the median county; or a relative decline of 4.55 per cent in the 75th percentile county compared with the 25th percentile. Importantly, it should be noted that the placebo estimate is smaller still and estimated with a similar degree of precision. Hence, the common time trend assumption cannot be rejected, and we may thus conclude that the epidemic appears to have had no effect at all on earnings er capita.

[Insert Table 4 about here]

Table 5 reports results for poverty rates. The pandemic appears to have had a strong and lasting positive effect on poverty. The overall effect is estimated at 0.66, implying that the pandemic increased the poverty rate in the median county by 33.5 per cent. Comparing again the 25th and the 75th percentile, the difference in flu mortality would give rise to an increase in poverty by 20 per cent. Again, the influenza effect is quite substantial – and if we consider long-term effects, it is even higher. However, when this variable is concerned, it is important to keep in mind that there is probably a direct mechanism at work, which has little to do with the functioning of the economy, to the extent that deceased individuals leave behind dependants, who are unable to support themselves. As mentioned above, orphans will not give rise to such a pattern, since they are not included in the variable. On the other hand, it is possible and probable that some of the victims of the pandemic left behind family members of the older generation who might end up in a poorhouse as a result. But a close inspection of Table A-3 in the Appendix reveals that these cannot be responsible for the entire effect: according to our estimates from the specification in levels, each death caused by the epidemic led to **four** additional poorhouse residents – and, considering the age pyramid in those days, it is very implausible that all of them would have been dependants of the deceased person.

For the poverty variable, the placebo estimate is also reassuringly insignificant, small and, in relative terms, precisely estimated. Thus, in line with the visual evidence the common time trend assumption seems to be confirmed in this case also.

[Insert Table 5 about here]

Next, we turn to the earnings of the non-poor population. Results are presented in Table 6. However, normalising earnings using the non-poor instead of the total population does not change much: the estimated effect is still insignificant, albeit somewhat higher than before.

[Insert Table 6 about here]

In the Appendix we provide the estimates for the outcome variables in levels as a robustness check. Clearly, as can be discerned from the reported R^2, our less preferred specification performs much worse in terms of explanatory power, and the statistical significance of estimated effects is lost in some cases. Nevertheless, these results appear to be generally reconcilable with estimates based on the logarithmic specifications. In terms of capital income the estimated effect of -0.026 together with the income level of 22 before the disease would point to a decrease of 0.11, while in the logarithmic equation we estimate a somewhat larger effect of 0.73. For earnings, the results confirm that the flu seems to have no effect. For poverty we find an increase of 0.004. Together with the average baseline poverty level of 4.2 per cent, this would suggest an increase of 0.90, which is not too far from the 0.66 estimated in the log specification.

In order to check the robustness of our findings, we performed additional regressions not included in the current version of the paper. Spatial heterogeneity of regions might be an issue. In order to check whether this is the case, we performed a regression including the spatial lag of the treatment variable in the regression. Our point estimate of the treatment effect on the poverty share was hardly affected and also the non-finding for earning persists. However, for capital income the standard error increases, thus reducing the statistical significance of our findings. Furthermore, since we find that earnings are unaffected by the flu we included earnings as a further control variable in the poverty and capital income regressions. Our results were left completely unaffected by this modification. Thus we conclude that our estimates are rather robust.

In conclusion, we find very strong evidence for the pandemic having a long-term poverty-increasing impact and relatively strong evidence that capital incomes were negatively affected by the Spanish flu. However, there is no evidence whatsoever that earnings or productivity were affected by the pandemic. Moreover, using placebo regressions we find that the common time trend assumption can be retained in all cases – placebo estimates are generally insignificant and close to zero.

7 Conclusion

It has been argued that regional differences in exposure to the 1918 influenza pandemic were largely random. If this holds to be true, these regional patterns in mortality rates can be

exploited to estimate the effects of a substantial health shock to the economy. Such an exercise has the potential to shed light on at least three important issues. Firstly, we would get an estimate of the actual economic consequences of the 1918 pandemic. Secondly, it would give us an idea of the possible effects of current and future pandemics on the performance of the economy. Thirdly, we might be able to say something in general about the functioning of the economy, and how labour supply shocks are transmitted through the system.

Economic theory has come up with at least three different views of how the economy reacts to a shock of this kind. Firstly, there are two different modelling approaches devised to explain economic growth: neoclassical growth models and endogenous growth models. Both classes of models agree on the immediate consequences of a negative labour supply shock: due to capital deepening, wages increase whereas capital returns are depressed. In the medium term, however, the two modelling approaches diverge: in a simple one-sector model, the differential in factor returns will trigger a very rapid accumulation of human capital, which in turn accelerates growth in the post-epidemic phase. In a two-sector framework, on the other hand, increasing wages increase the cost of producing human capital and thus resources are shifted to the production of final goods - and thereby the growth rate of the economy is reduced. On the other hand, taking a perspective from trade theory would suggest that wages are left unaffected by the pandemic, and instead the shares of different sectors in the economy are affected.

In our study, we find no evidence against the assumption that the epidemic was a largely random shock to Swedish regions. The common time trend assumption appears to be satisfied for all variables that we consider, and we also fail to identify a socioeconomic gradient in the incidence of the epidemic. Besides, since influenza incidence and mortality tend to follow the same spatial patterns in general, it is reassuring to see that our main results are robust to the inclusion of variables capturing different aspects of influenza incidence. Thus, it is our tentative conclusion that the differences in excess mortality rates that we observe across regions are largely exogenous.

The results from our main regressions are remarkably robust to various changes in specification. For capital incomes, we find that the pandemic had a strong negative impact, and this impact appears to have been a combination of immediate and post-epidemic responses. According to our estimates, the highest quartile (with respect to influenza mortality) would have experienced a drop of 14 per cent during the pandemic and an additional 12 per cent afterwards. For earnings, on the other hand, we are unable to detect any effect either during

or after the pandemic. For poverty, finally, we find a strong and positive effect, which seems to have appeared only once the epidemic was unequivocally over in 1920. For this variable, the top quartile suffered an increase in poverty by 23 per cent compared to the bottom quartile.

Strong as these results may seem, they do not appear to sit very well with the most popular macroeconomic models. On the one hand, a relative reduction of capital returns is exactly what one would expect after the ratio between physical and human capital has increased. On the other hand, our very robust finding that earnings were unaffected is much more difficult to explain – and it is also in direct contradiction to our result for capital income. Likewise, our finding that poverty rates increased is also difficult to reconcile with the increased scarcity of labour. On the other hand, it could possibly be explained with reference to the Rybczynski theorem, according to which adjustment to a shock of this kind is transmitted through the sectoral shares in the economy and not the factor returns.

Since there is some disagreement between our results and theoretical predictions, it is useful to consider whether our estimates could be driven by selective mortality. As mentioned above, we find it reassuring that we are unable to link the excess influenza mortality at the regional level to any observable characteristic in the pre-influenza period. Nevertheless, it remains an open issue whether influenza mortality was selective, and also whether the health shock went beyond elevated mortality rates (although one could argue that we have addressed this second point by taking incidence into account). However, it is difficult to explain our findings in terms of selective mortality as well. If there were *positive* selective mortality, so that the most healthy died, we might expect increases in poverty and also no effect on wages – but then there would also be less capital deepening and, as a consequence, a smaller reduction in capital returns. If, on the other hand, there is *negative* selective mortality, i.e. 'survival of the fittest', one would not expect to observe an increase in poverty, and the wages should increase even more than predicted by standard theory. In conclusion, neither of these two types of selection appears to sit well with our results.

Given that our results are so difficult to interpret, there seems to be a need to corroborate them further using data from other countries. If they are confirmed, they suggest that theoretical models might need to be refined to accommodate the inconsistencies identified in this study.

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Figure 1: Swedish Exports 1910–1930

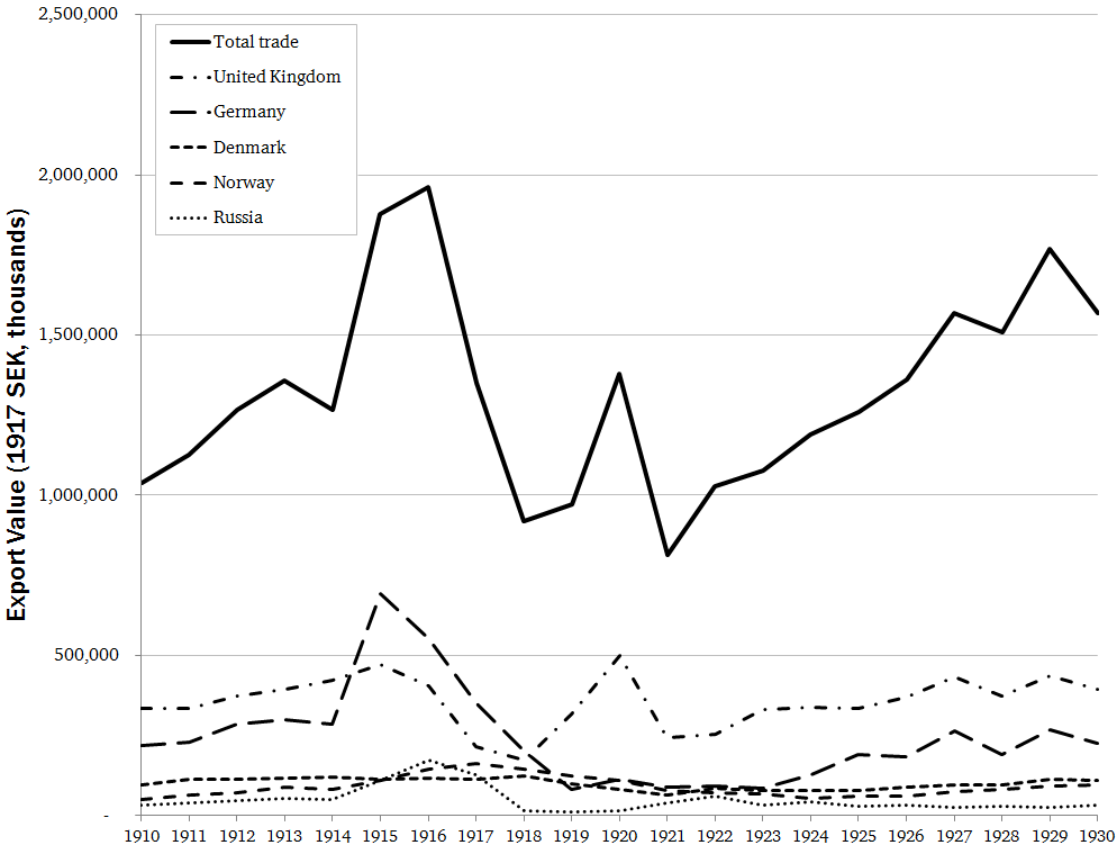

Figure 2: 1917-20 Monthly influenza and pneumonia deaths, Sweden

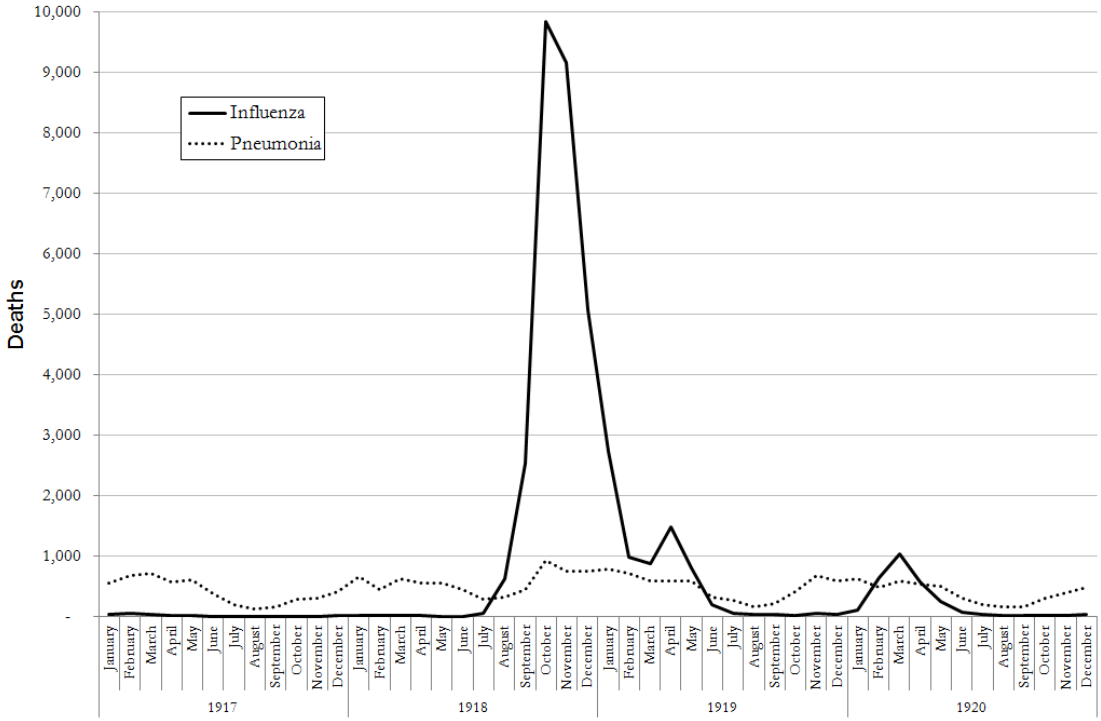

Figure 3: 1918 Influenza mortality in different counties

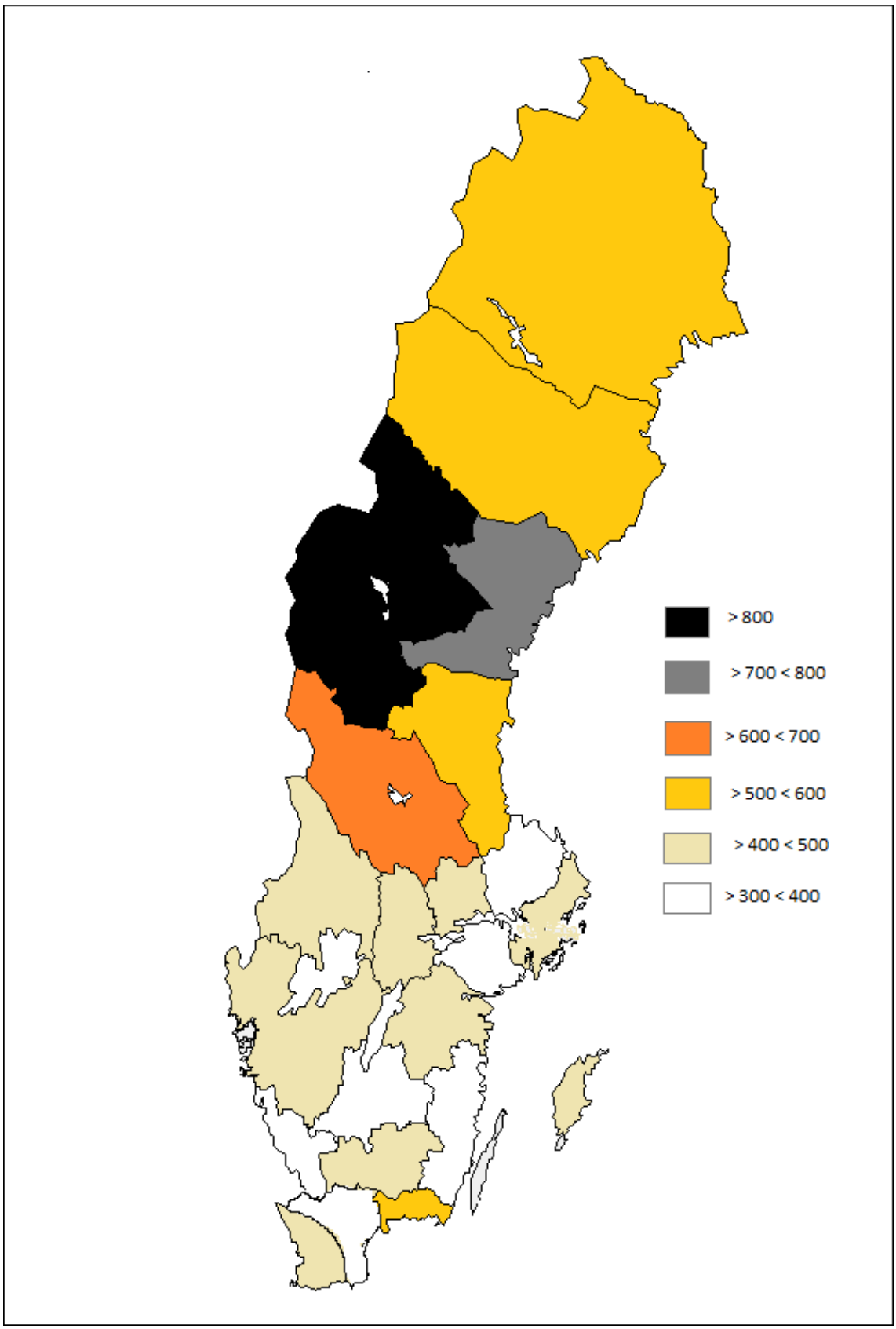

Figure 4: 1918-20 Incidence of influenza in different locations and socioeconomic groups

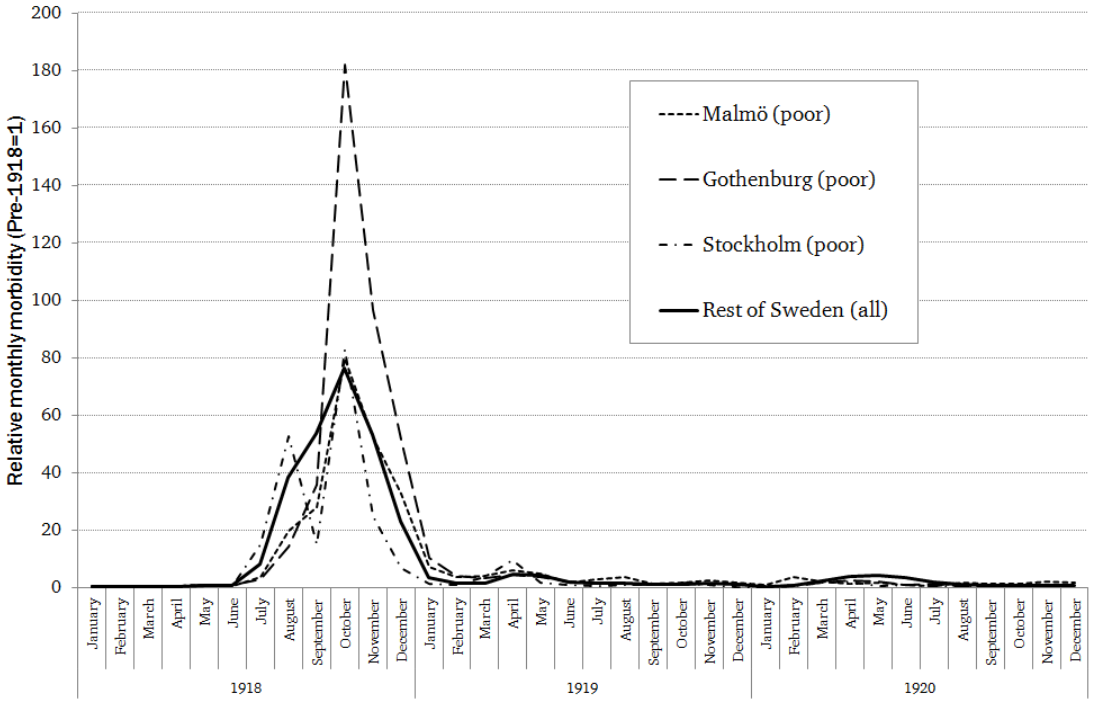

Figure 5: 1918-20 Influenza mortality in different counties

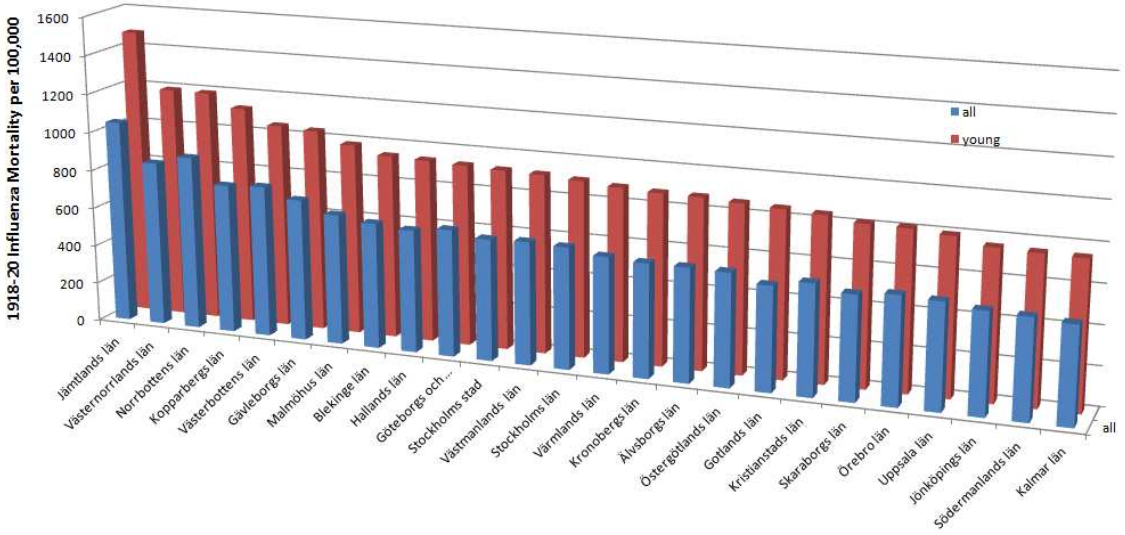

Figure 6: Excess morbidity and mortality at the regional level

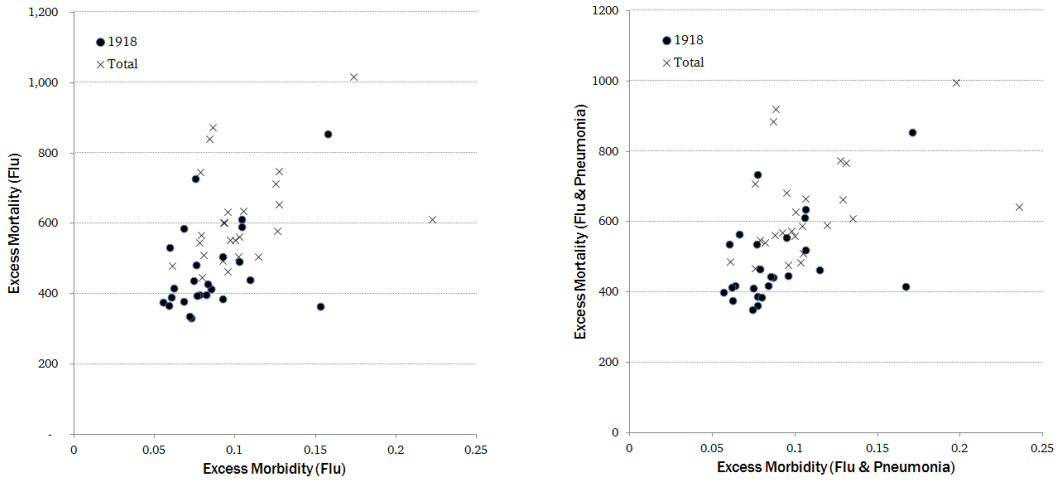

Figure 7: Derivation of the treatment variable

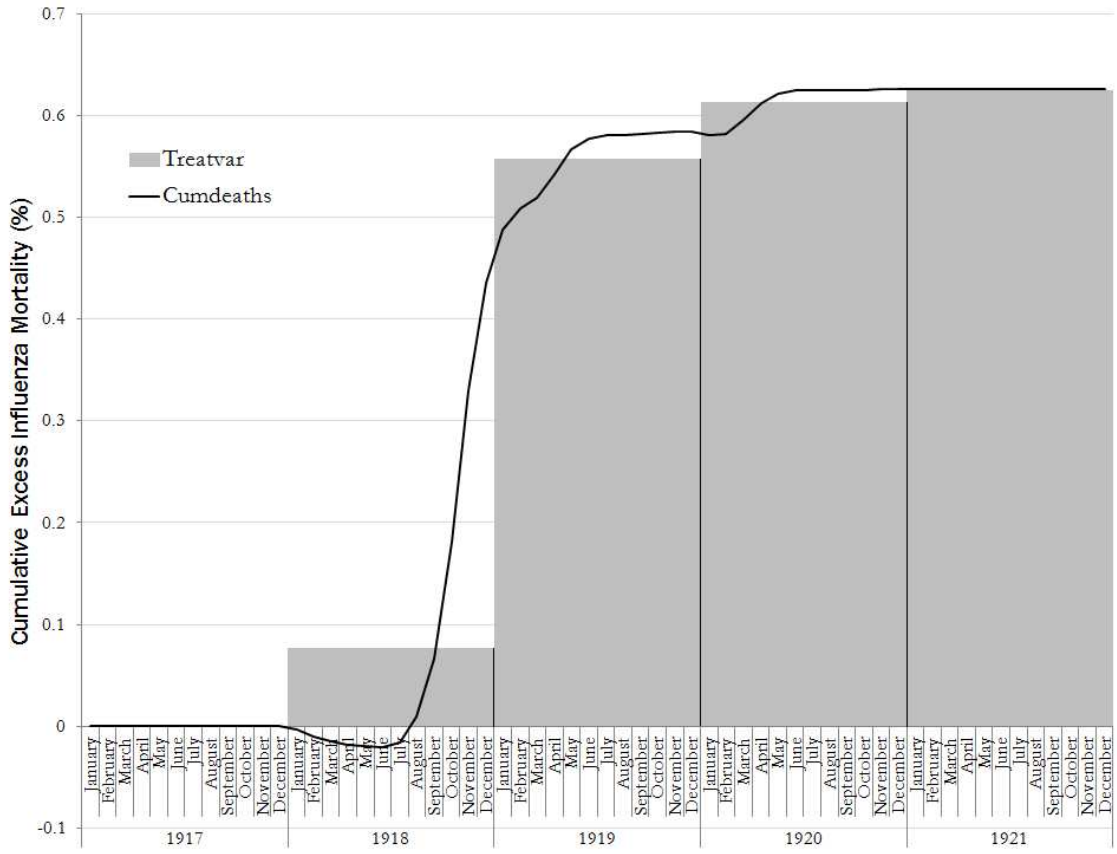

Figure 8: Common Time Trend for Capital Income

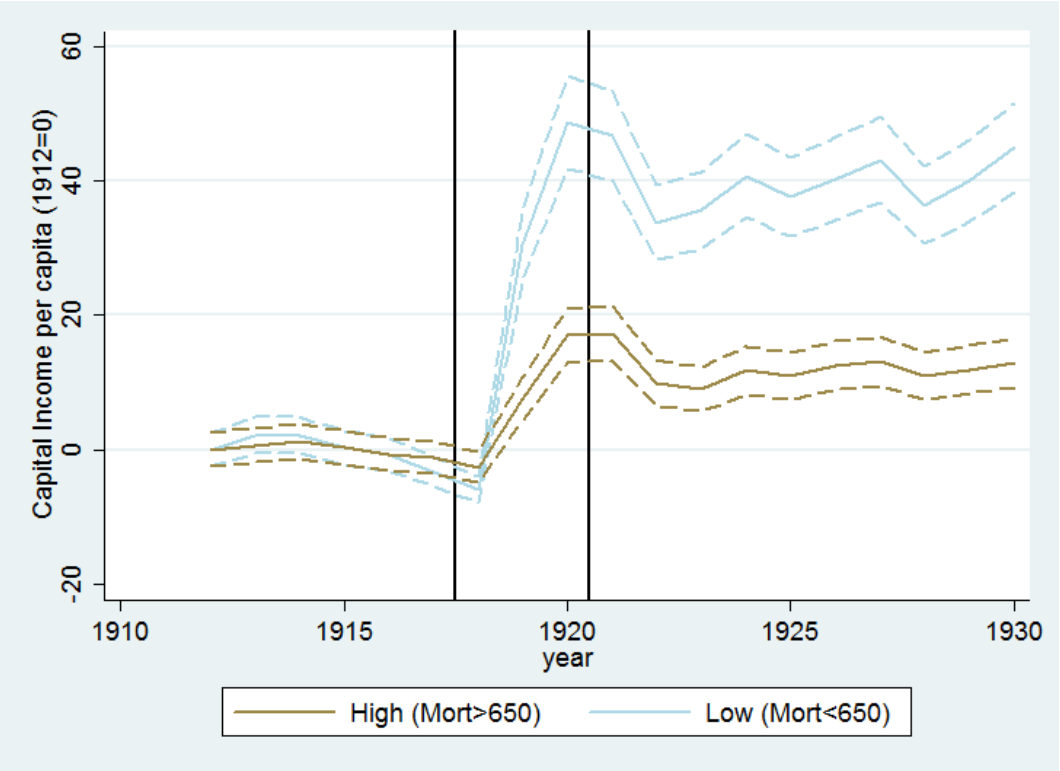

Figure 9: Common Time Trend for Earnings

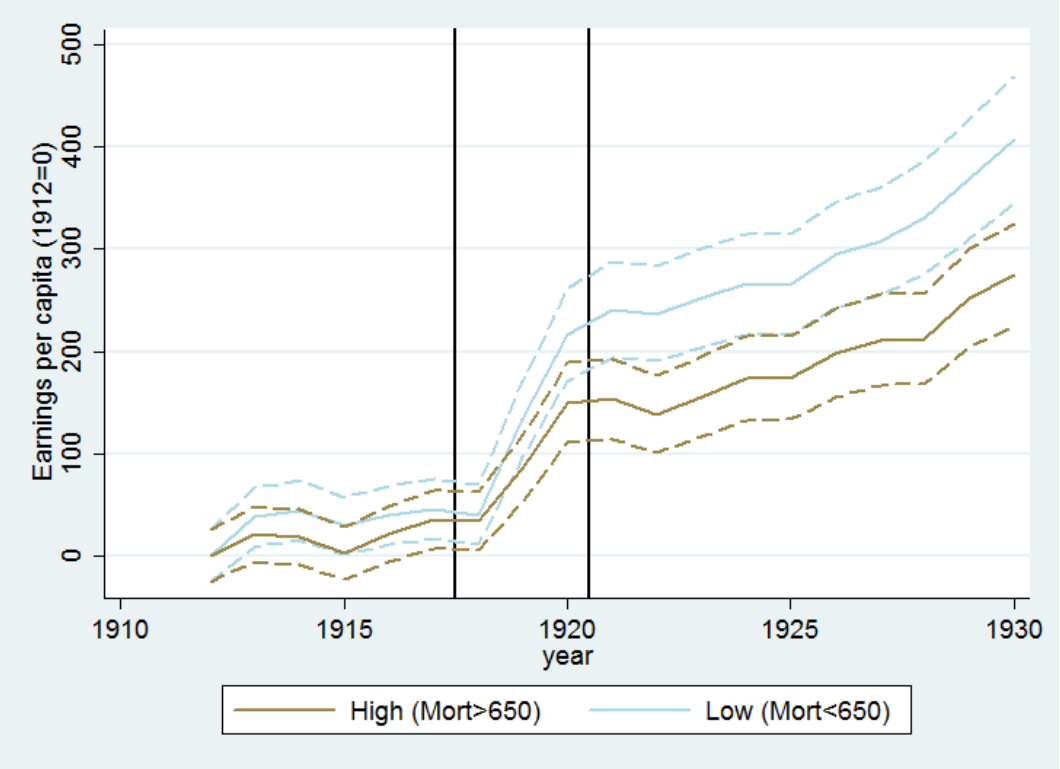

Figure 10: Common Time Trend for Poverty

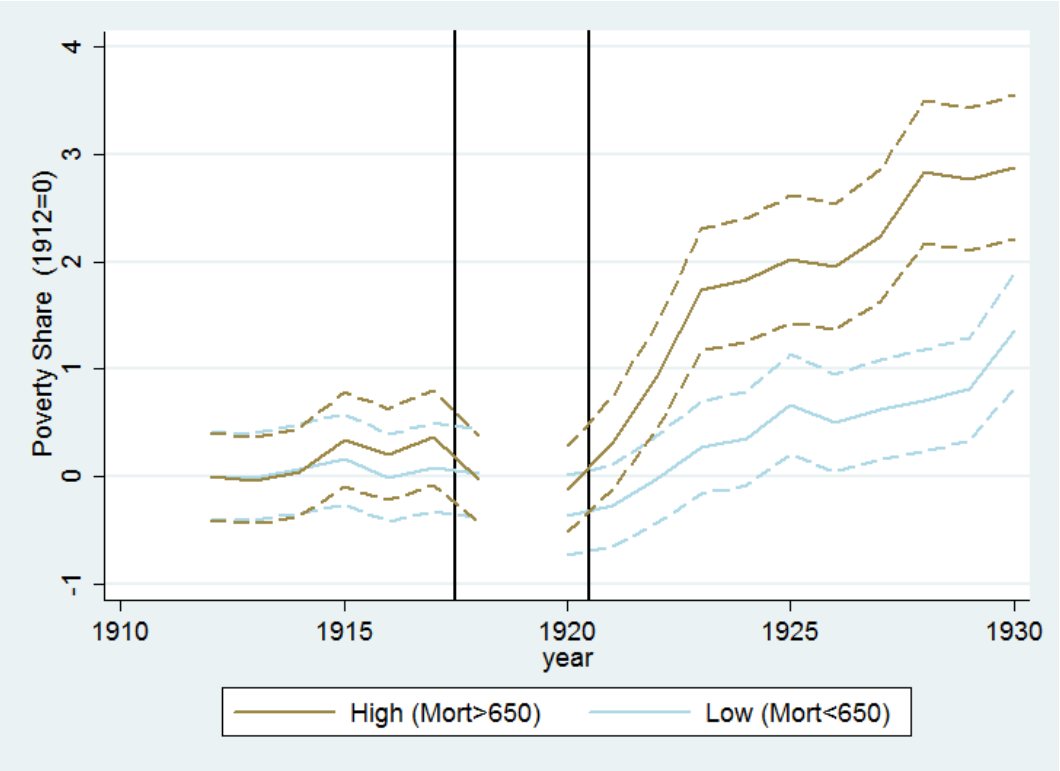

Figure 11: Common Time Trend for Non-Poor Earnings

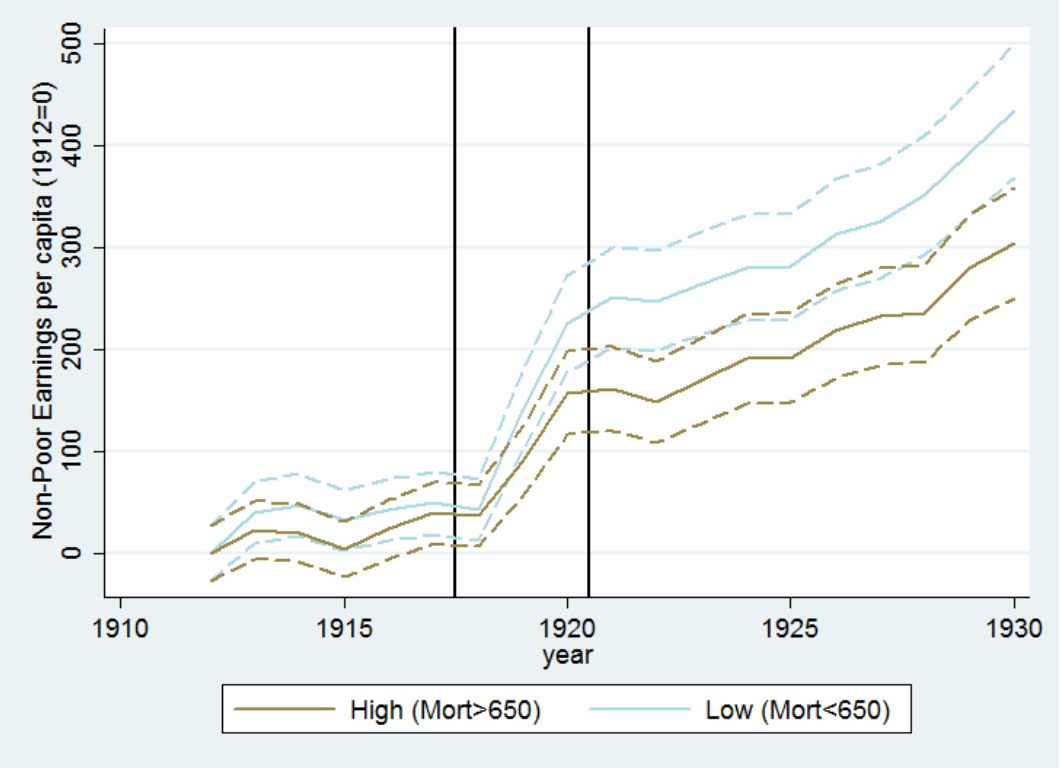

Table 1: Sectoral Composition and Influenza Exposure

County	1910 Population	Flu Mortality	Farming	Industry	Commerce
Gotlands län	55,217	445.9	0.571	0.166	0.080
Södermanlands län	178,568	463.5	0.464	0.272	0.094
Kalmars län	228,129	480.3	0.481	0.238	0.097
Örebro län	207,021	494.5	0.419	0.341	0.086
Jönköpings län	214,454	504.3	0.470	0.283	0.073
Uppsala län	128,171	506.1	0.449	0.257	0.077
Skaraborgs län	241,284	510.6	0.589	0.186	0.065
Kristianstads län	228,307	545.6	0.538	0.216	0.084
Östergötlands län	294,179	552.0	0.420	0.299	0.098
Älvsborgs län	287,692	553.3	0.533	0.250	0.065
Värmlands län	260,135	562.9	0.533	0.251	0.068
Kronobergs län	149,654	567.0	0.592	0.190	0.058
Västmanlands län	155,920	577.6	0.435	0.316	0.078
Hallands län	147,224	601.4	0.521	0.214	0.099
Stockholms län	229,181	602.0	0.358	0.318	0.115
Stockholms stad	342,323	610.7	0.005	0.381	0.247
Göteborgs o Bohus län	381,270	631.5	0.252	0.331	0.180
Blekinge län	149,359	634.7	0.403	0.258	0.088
Malmöhus län	457,214	654.6	0.304	0.343	0.148
Gävleborgs län	253,792	712.9	0.381	0.328	0.111
Västerbottens län	161,366	746.3	0.693	0.141	0.045
Kopparbergs län	233,873	748.7	0.487	0.316	0.067
Västernorrlands län	250,512	840.3	0.473	0.269	0.088
Norrbottens län	161,132	873.7	0.541	0.224	0.088
Jämtlands län	109,851	1017.4	0.694	0.124	0.052
Sweden	5,505,828	616.2	0.430	0.277	0.104

The table shows 1910 population size and sectoral shares according to the 1910 census (SCB, 1917) as well as standardised excess influenza mortality 1918-20.

Table 2: Descriptive statistics

	N	Mean	St. Dev.	Before	During	After
Capital Income (SEK/capita)	475	39.655	42.353	22.596	42.843	49.611
Earnings (SEK/capita)	475	402.986	236.416	273.581	381.107	498.979
Poverty, Per Cent	450	4.655	1.461	4.196	4.037	5.167
Trade Demand (SEK)	475	140.922	44.101	129.786	116.579	159.164
Population in Thousands	475	236.448	97.963	227.795	235.197	242.772
Flu Incidence, Per Capita	475	0.006	0.020	0	0.026	0
Cum. Flu Incidence Per Capita	475	0.066	0.056	0	0.080	0.105
Cum. Flu Mortality, Per Cent	475	0.395	0.309	0	0.469	0.626

The table shows descriptive statistics for the variables, and shows means of all variables *before* ($t<1918$), *during* ($t\geq1918$ and $t\leq1920$) and *after* ($t>1920$) the Spanish flu pandemic. Data for poverty is missing in 1919. Incidence (Infections) and and Mortality have been calculated as excess rates.

Table 3: Capital Income

	(1) baseline	(2) dynamic	(3) morbidity	(4) cum_morb	(5) trade	(6) placebo	(7) collapsed	(8) collapsed
w_{it}	-0.735***	-0.420**	-0.382*	-0.420**	-0.465**		-0.488**	-0.370*
	0.202	0.178	0.195	0.179	0.169		0.191	0.183
$w_{it} \times \mathbf{1}(t > 1920)$		-0.368**	-0.370**	-0.368**	-0.338**			-0.311
		0.165	0.165	0.165	0.161			0.196
Placebo						0.0353		
						0.109		
Cum. Incidence			-0.362					
			0.51					
Incidence				0.0536				
				0.669				
Trade Demand					1.905**			
					0.91			
Constant	3.035***	3.035***	3.035***	3.035***	-6.292	3.035***	3.050***	3.050***
	0.0251	0.0251	0.0244	0.0251	4.456	0.0116	0.0297	0.0298
Observations	475	475	475	475	475	150	125	125
R^2	0.946	0.947	0.947	0.947	0.948	0.566	0.95	0.951

Fixed effects regression, year dummies not shown in the tables, regressions are weighted by population in 1917. Robust standard errors in second row. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 4: Earnings

	(1) baseline	(2) dynamic	(3) morbidity	(4) cum_morb	(5) trade	(6) placebo	(7) collapsed	(8) collapsed
w_{it}	-0.14	-0.105	0.0064	-0.105	-0.104		-0.127	-0.118
	0.226	0.162	0.185	0.162	0.159		0.183	0.156
$w_{it} \times \mathbf{1}(t > 1920)$		-0.0414	-0.0462	-0.0431	-0.0417			-0.0256
		0.105	0.105	0.105	0.103			0.113
Placebo						0.0732		
						0.145		
Cum. Incidence			-1.078**					
			0.441					
Incidence				-0.182				
				0.465				
Trade Demand					-0.0184			
					1.119			
Constant	5.481***	5.481***	5.481***	5.481***	5.572	5.481***	5.609***	5.609***
	0.0913	0.0914	0.0898	0.0914	5.518	0.0843	0.0213	0.0214
Observations	475	475	475	475	475	150	125	125
R^2	0.892	0.892	0.896	0.893	0.892	0.164	0.945	0.946

Fixed effects regression, year dummies not shown in the tables, regressions are weighted by population in 1917. Robust standard errors in second row. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 5: Poverty Share

	(1) baseline	(2) dynamic	(3) morbidity	(4) cum_morb	(5) trade	(6) placebo	(7) collapsed	(8) collapsed
w_{it}	0.662**	-0.0129	0.205	-0.027	0.00453		0.402	0.0147
	0.279	0.127	0.225	0.119	0.132		0.244	0.141
$w_{it} \times \mathbf{1}(t > 1920)$		0.728***	0.714***	0.754***	0.720***			0.716***
		0.212	0.216	0.213	0.214			0.237
Placebo						0.151		
						0.192		
Cum. Incidence			-2.075**					
			1.002					
Incidence				1.084				
				0.852				
Trade Demand					-1.076*			
					0.523			
Constant	1.442***	1.442***	1.442***	1.442***	6.709**	1.442***	1.455***	1.455***
	0.0251	0.0252	0.0211	0.0254	2.545	0.0122	0.019	0.019
Observations	450	450	450	450	450	150	100	100
R^2	0.539	0.551	0.604	0.553	0.555	0.128	0.575	0.627

Fixed effects regression, year dummies not shown in the tables, regressions are weighted by population in 1917. Robust standard errors in second row. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 6: Non-Poor Earnings

	(1) baseline	(2) dynamic	(3) morbidity	(4) cum_morb	(5) trade	(6) placebo	(7) collapsed	(8) collapsed
w_{it}	-0.12	-0.132	-0.0302	-0.131	-0.124		-0.136	-0.146
	0.223	0.164	0.175	0.165	0.162		0.185	0.16
$w_{it} \times \mathbf{1}(t > 1920)$		0.0138	0.00931	0.0149	0.00788			0.0254
		0.1	0.0996	0.0995	0.0989			0.107
Placebo						0.0081		
						0.122		
Cum. Incidence			-0.980**					
			0.376					
Incidence				0.123				
				0.373				
Trade Demand					-0.381			
					0.564			
Constant	4.972***	5.542***	5.589***	5.541***	7.362**	4.981***	4.987***	4.987***
	0.0327	0.146	0.162	0.147	2.699	0.0796	0.0157	0.0158
Observations	450	450	450	450	450	125	125	125
R^2	0.945	0.945	0.948	0.945	0.945	0.127	0.953	0.953

Fixed effects regression, year dummies not shown in the tables, regressions are weighted by population in 1917. Robust standard errors in second row. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Appendix

Proof 1 (of Lemma 1) *For simplicity, we provide the proof for a two-period model were the treatment occurs between the two periods. According to the Frisch-Waugh-Lovell theorem, the OLS estimate of β is given by*

$$\begin{aligned}\hat{\beta}_{OLS} =& \frac{\sum_{t=1}^{2}\sum_{i=1}^{N}\tilde{y}_{it}\tilde{w}_{it}}{\sum_{t=1}^{2}\sum_{i=1}^{N}\tilde{w}_{it}^{2}} = \frac{\sum_{t=1}^{2}\sum_{i=1}^{N}\left(y_{it}-\bar{y}_{i.}-\bar{y}_{.t}+\bar{y}\right)\left(w_{it}-\bar{w}_{i.}-\bar{w}_{.t}+\bar{w}\right)}{\sum_{t=1}^{2}\sum_{i=1}^{N}\left(w_{it}-\bar{w}_{i.}-\bar{w}_{.t}+\bar{w}\right)^{2}} \\ =& \frac{\sum_{i=1}^{N}\left(y_{i2}-y_{i1}-\bar{y}_{.2}+\bar{y}_{.1}\right)\left(w_{i2}-\bar{w}_{.2}\right)}{\sum_{i=1}^{N}\left(w_{i2}-\bar{w}_{.2}\right)^{2}}.\end{aligned}$$

Hence,

$$\begin{aligned}\mathbb{E}\left(\hat{\beta}_{OLS}\right) &\overset{SUTVA}{=} \mathbb{E}\left(\frac{\sum_{i=1}^{N}\left(Y_{i2}\left(w_{i2}\right)-y_{i1}-\bar{y}_{.2}+\bar{y}_{.1}\right)\left(w_{i2}-\bar{w}_{.2}\right)}{\sum_{i=1}^{N}\left(w_{i2}-\bar{w}_{.2}\right)^{2}}\right) \\ &\overset{CT,MI}{=} \frac{\mathbb{E}\left[\left(Y_{i2}\left(w_{i2}\right)-Y_{i2}\left(\bar{w}_{.2}\right)\right)\left(w_{i2}-\bar{w}_{.2}\right)\right]}{\mathbb{E}\left[\left(w_{i2}-\bar{w}_{.2}\right)^{2}\right]} \\ &= \frac{\mathbb{E}\left[TE_{i2}\left(w_{i2}-\bar{w}_{.2}\right)^{2}\right]}{\mathbb{E}\left[\left(w_{i2}-\bar{w}_{.2}\right)^{2}\right]} \overset{MI}{=} ATE_{2}\end{aligned}$$

□

Table A-1: Level-Level Estimation: Capital Income

	(1) baseline	(2) dynamic	(3) morbidity	(4) cum_morb	(5) trade	(6) placebo	(7) collapsed	(8) collapsed
w_{it}	-25.79	-7.362	-82.90*	-8.325	-9.468		-11.55	-4.121
	31.17	34.11	40.57	35.42	34.51		40.24	42.16
$w_{it} \times \mathbf{1}(t > 1920)$		-21.56**	-18.27*	-26.76**	-17.54			-19.55**
		8.461	8.956	11.43	11.53			8.746
Placebo						0.643		
						3.35		
Cum. Incidence			731.7***					
			131.9					
Incidence				-545.9**				
				227.9				
Trade Demand					0.169			
					0.164			
Constant	27.92***	27.92***	27.92***	27.92***	4.573	27.92***	27.98***	27.98***
	4.733	4.738	2.636	4.981	19.24	0.525	4.861	4.887
Observations	475	475	475	475	475	150	125	125
R^2	0.54	0.541	0.822	0.565	0.544	0.231	0.534	0.535

Fixed effects regression, year dummies not shown in the tables, regressions are weighted by population in 1917. Robust standard errors in second row. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A-2: Level-Level Estimation: Earnings

	(1) baseline	(2) dynamic	(3) morbidity	(4) cum_morb	(5) trade	(6) placebo	(7) collapsed	(8) collapsed
w_{it}	-2.86	-2.091	-260.8*	-6.079	-5.618		3.931	1.966
	112.2	58.33	134.3	62.23	64.26		97.46	72.06
$w_{it} \times \mathbf{1}(t > 1920)$		-0.9	10.36	-22.44	5.832			5.170
		74.78	74.98	69.62	76.8			80.30
Placebo						16.75		
						37.03		
Cum. Incidence			2505.8***					
			567.2					
Incidence				-2261.1**				
				993				
Trade Demand					0.282			
					0.926			
Constant	281.7***	281.7***	281.7***	281.7***	242.6*	281.7***	310.9***	310.9***
	29.19	29.22	28.46	29.63	120	20.51	12.96	13.03
Observations	475	475	475	475	475	150	125	125
R^2	0.767	0.767	0.862	0.779	0.767	0.155	0.782	0.782

Fixed effects regression, year dummies not shown in the tables, regressions are weighted by population in 1917. Robust standard errors in second row. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A-3: Level-Level Estimation: Poverty Share

	(1) baseline	(2) dynamic	(3) morbidity	(4) cum_morb	(5) trade	(6) placebo	(7) collapsed	(8) collapsed
w_{it}	3.799***	-0.151	1.296	-0.225	0.0701		2.259*	-0.0268
	1.33	0.705	1.33	0.648	0.714		1.186	0.755
$w_{it} \times \mathbf{1}(t > 1920)$		4.263***	4.171***	4.402***	3.843***			4.228***
		0.828	0.845	0.845	0.7			0.951
Placebo						0.99		
						1.074		
Cum. Incidence			-13.75**					
			5.134					
Incidence				5.741				
				3.635				
Trade Demand					-0.0180***			
					0.00567			
Constant	4.399***	4.399***	4.399***	4.399***	6.899***	4.399***	4.471***	4.471***
	0.132	0.133	0.0976	0.134	0.724	0.0549	0.119	0.119
Observations	450	450	450	450	450	150	100	100
R^2	0.465	0.478	0.555	0.48	0.506	0.14	0.526	0.596

Fixed effects regression, year dummies not shown in the tables, regressions are weighted by population in 1917. Robust standard errors in second row. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A-4: Level-Level Estimation: Non-Poor Earnings

	(1) baseline	(2) dynamic	(3) morbidity	(4) cum_morb	(5) trade	(6) placebo	(7) collapsed	(8) collapsed
w_{it}	5.945	-3.43	-141.0*	-8.151	-4.47		2.96	-2.230
	56.37	28.7	71.94	30.16	31.58		48.69	35.53
$w_{it} \times \mathbf{1}(t > 1920)$		10.92	16.97	-0.536	13.06			13.66
		39.33	39.58	36.2	41.06			41.78
Placebo						0.62		
						16.46		
Cum. Incidence			1329.7***					
			294.2					
Incidence				-1221.0**				
				541.5				
Trade Demand					0.0898			
					0.482			
Constant	166.3***	259.1***	196.4***	269.3***	160.1***	166.9***	167.0***	167.0***
	9.365	38.37	52.41	36.91	53.4	10.16	6.146	6.178
Observations	450	450	450	450	450	125	125	125
R^2	0.784	0.784	0.882	0.798	0.784	0.146	0.789	0.789

Fixed effects regression, year dummies not shown in the tables, regressions are weighted by population in 1917. Robust standard errors in second row. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$