The Impact of the 1918 Spanish Flu Epidemic on Economic Performance in Sweden

An Investigation into the Consequences of an Extraordinary Mortality Shock

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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 representing an unparalleled labour supply shock. 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. However, 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.

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JEL classification: I18; J31; O40.

1 Introduction

In 1918 the world is hit by the Spanish flu. Estimates suggest that 500 million individuals worldwide were infected by the 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 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 about the medical causes of the Spanish flu, the origins of the 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 affecting mainly the population of working age within a very short time window?

Studying the effects of the Spanish flu can give insights into the effects that future pandemics may have on economic outcomes and be helpful in establishing appropriate policy responses. The influenza appeared during a very short time, which facilitates the identification of the economic effects and serves as a useful test of the effects of a health shock on economic outcomes. Thus, given the heightened awareness of economic issues associated with pandemics, it seems timely and relevant to acquire knowledge of consequences of an event such as the Spanish flu.

The influenza pandemic represents a large labour supply shock. Due to its force and randomness, the 1918 flu wave therefore also provide an interesting case for evaluating the empirical performance of macroeconomic models. Specifically, in line with Alvarez-Cuadrado (2008) who uses the World War II as a natural experiment to discriminate among competing growth specifications, we exploit the flu-generated exogenous labour shock to evaluate the predictions of endogenous growth models. [1]

Using administrative data from Swedish regions, we employ an extension of the standard difference-in-differences (DID) estimator to exploit the differing mortality rates across Swedish regions. Focusing on Swedish regions has several advantages. First, the variation in flu mortality is high across counties. Almost one percent of the Swedish population died from the Spanish flu, but there were important regional differences (Åman, 1990). For instance some counties experienced more than twice the flu mortality rate of others. We use this variation to examine the impact of the pandemic on earnings, capital returns and poverty.

[1] Alvarez-Cuadrado (2008) uses the war as a natural experiment in terms of the destruction of capital stocks and exploits the adjustment of economies after the war to tests various growth models.

Second, many key economic indicators are available from Swedish administrative datasets and they are consistently collected across regions and time, allowing for precise estimates.[2] Hence, the data allow us to estimate the effects of the influenza on a number of economic outcomes while carefully checking key methodological assumptions.

Thirdly, Sweden did not take part in the World War I, 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 (cf. Glick and Taylor, 2009; Kesternich et al., 2013). Obviously, Sweden was affected by the war in many ways. However, in a non-belligerent country there are no other major shocks to mortality coinciding with the disease. Finally, Sweden is a unitary state and a very homogeneous country and thus there is little need to worry about internal cultural differences or asymmetric responses in regional institutions (cf. Tabellini, 2010; Acemoglu et al., 2003).

Our empirical results support the prediction from endogenous growth theory that there will be slower growth in the economy during a transition period after the pandemic. However, in other parts, our empirical results are difficult to reconcile with standard theoretical models. Most importantly, we do not observe the immediate increase in GDP per capita which one would expect as a result of capital deepening. Moreover, there is an apparent redistribution between capital and labour taking place, which suggests that the impact of the pandemic goes beyond what standard growth theory would predict. In the discussion of this paper, we make an attempt at explaining these findings within the context of a growth model.

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

The first official reports on the 1918 flu came from Spain; hence its popular name.[3] Upon reaching the European continent, the spread of the pandemic was accelerated by increased troop movement due to the war (Patterson and Pyle, 1991). Among researchers in medical history there is consensus that the disease ran its course in three to four waves. The first wave was in the spring of 1918, with the disease returning in the fall of the same year and again in

[2]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 and that the detailed instructions sent from national authorities to health personnel on how to verify the cause of death (see e.g. Statistics Sweden 1911) served its purpose and that the correct disease was, in fact, recorded.

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

1919. The last wave occurred mainly in Scandinavia and some islands in the South Atlantic.

An interesting feature of the second wave of the pandemic is that it took the world by complete surprise. The first wave of the pandemic had such a low mortality rate that experts doubted whether it was influenza at all. For example, in the summer of 1918, Little et al. (1918) conclude

> *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 concluded that the epidemic had disappeared (Barry, 2005). Contemporary accounts by Swedish doctors also suggest that the first wave was very mild and that there were conflicting views of whether the disease was influenza or a new type of pneumonia (Petrén, 1918a,b).

This is in stark contrast to the second wave of the Spanish flu with exceptionally high 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 pneumonia (Morens and Fauci, 2007). The incubation time and the time between infection and death was very short. According to Taubenberger and Morens (2006), most deaths occurred 6-11 days after the outbreak, but there is 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 unusual and unlike other (influenza) diseases, which typically exhibit a U-shape in the mortality distribution over age groups, the Spanish flu had a W-shaped distribution over age.

2.1 Theoretical Perspectives

From a purely economic point of view, we may think of the Spanish flu pandemic as labour supply a shock to the economy, which on the other hand leaves physical capital intact. In order to generate hypotheses for how our outcome variables may react to the pandemic, we briefly review the macroeconomic literature on economic growth. Since we cannot observe GDP at a regional level, we look at the returns to capital and labour. Moreover, we are concerned with distributional effects and therefore look at poverty.

Our point of departure is a standard two-sector model (Lucas, 1988). Consider an economy with competitive markets. In each location, there is a large number of production units producing a homogeneous final good. Preferences over (per capita) consumption are given by

$$\int_0^\infty U\left(c\left(t\right)\right) e^{-\rho t} \mathrm{d}t \tag{1}$$

where ρ is the rate of time preference. Let $h\left(t\right)$ denote the skill level (human capital level) of a typical worker and $u\left(t\right)$ be the fraction of non-leisure time devoted to goods production. Then $1-u\left(t\right)$ is the effort devoted to the accumulation of human capital. It is assumed that the growth of human capital takes a simple form as

$$\dot{h} = \delta\left(1-u\right)h \tag{2}$$

where parameter δ is positive.

The output per capita $y(=Y/N)$ depends on the per capita capital stock, $k(=K/N)$, the effective work force uh, and the average level of human capital in the region $\bar{h}$:

$$y = Ak^{\beta}\left[uh\right]^{1-\beta}\bar{h}^{\gamma} \tag{3}$$

where parameter β is the income share of physical capital, and parameter γ is positive and captures external effects of human capital. The accumulation of physical capital is assumed to take the form

$$\dot{k} = y - c \tag{4}$$

In equilibrium, $h=\bar{h}$ because all production units within the region are treated as being

identical. Substituting this into the production function and solving the maximisation problem, one gets the social optimum. However, we want to solve for the competitive equilibrium here, which will be done by deriving first order conditions taking the whole path of $\left\{\bar{h}\left(t\right):t\geq 0\right\}$ as given. Thus, the current-value Hamiltonian may be written as

$$H\left(k,h,\theta_1,\theta_2,c,u;A,\sigma,\beta,\gamma,\delta,\left\{N\left(t\right),Y\left(t\right):t\geq 0\right\}\right)=\frac{1}{1-\sigma}\left[c^{1-\sigma}-1\right] \tag{5}$$
$$+\theta_1\left[Ak^{\beta}\left[uh\right]^{1-\beta}\bar{h}^{\gamma}-c\right]+\theta_2\delta\left(1-u\right)h$$

where θ_1 and θ_2 are the co-state variables for k and h respectively. Things taken as given are put after the semicolon in the Hamiltonian.

The first order conditions are thus given as follows:

$$\frac{\partial H}{\partial c}=c^{-\sigma}-\theta_1=0 \tag{6}$$
$$\frac{\partial H}{\partial u}=\theta_1\left(1-\beta\right)Ak^{\beta}u^{-\beta}h^{1-\beta}\bar{h}^{\gamma}-\theta_2\delta h=0 \tag{7}$$
$$-\dot{\theta}_1=-\rho\theta_1+\beta k^{\beta-1}\theta_1A\left(uh\right)^{1-\beta}\bar{h}^{\gamma} \tag{8}$$
$$-\dot{\theta}_2=-\rho\theta_2+\left(1-\beta\right)h^{-\beta}\theta_1Au^{1-\beta}k^{\beta}\bar{h}^{\gamma}+\theta_2\delta\left(1-u\right) \tag{9}$$
$$\dot{k}=y-c \tag{10}$$
$$\dot{h}=\delta\left(1-u\right)h \tag{11}$$

Boucekkine and Ruiz Tamarit (2005) present an analytical solution to this problem for the special case where $\sigma=\beta$. However, before proceeding we derive the factor returns which are crucial in our empirical analysis. Since the economy is competitive, we assume that input factors earn their private marginal products. Hence, we have

$$r=\frac{\partial Y}{\partial K}=\beta yk^{-1} \tag{12}$$
$$w=\frac{\partial Y}{\partial N}=\left(1-\beta\right)y \tag{13}$$

and thus capital returns per capita are given by $rk=\beta y$. A population shock has a direct impact on average physical capital $k=K/N$ and may also lead to adjustments in consumption c and the allocation of workers to the production of final goods, represented by u. Moreover,

since there is no socioeconomic gradient of the Spanish flu (we will provide evidence on this below), human capital h will not be affected by the population shock. The physical capital and consumption may be adjusted instantaneously, but only u has an instantaneous effect on production and factor returns. Thus, we may write:

$$\frac{\mathrm{d}r}{\mathrm{d}N} = \beta\left(1-\beta\right)AK^{\beta-1}\left(uh\right)^{1-\beta}N^{-\beta}h^{\gamma} + \beta\left(1-\beta\right)AK^{\beta-1}\left(hN\right)^{1-\beta}u^{-\beta}h^{\gamma}\frac{\mathrm{d}u}{\mathrm{d}N} \tag{14}$$

$$= \left(1-\beta\right)\left(\frac{1}{N} + \frac{1}{u}\frac{\mathrm{d}u}{\mathrm{d}N}\right)r \tag{15}$$

$$\frac{\mathrm{d}w}{\mathrm{d}N} = -\beta\left(1-\beta\right)AK^{\beta}\left(uh\right)^{1-\beta}N^{-1-\beta}h^{\gamma} + \left(1-\beta\right)^{2}AK^{\beta}\left(uN\right)^{-\beta}h^{1-\beta+\gamma}\frac{\mathrm{d}u}{\mathrm{d}N} \tag{16}$$

$$= \beta\left(-\frac{1}{N} + \frac{1-\beta}{\beta}\frac{1}{u}\frac{\mathrm{d}u}{\mathrm{d}N}\right)w \tag{17}$$

Now consider the responses of labour and capital returns expressed as elasticities:

$$\frac{\mathrm{d}\left(rk\right)}{\mathrm{d}N}\frac{N}{rk} = \frac{\mathrm{d}w}{\mathrm{d}N}\frac{N}{w} = \beta\left[-1 + \frac{1-\beta}{\beta}\frac{\mathrm{d}u}{\mathrm{d}N}\frac{N}{u}\right] \tag{18}$$

Hence, the immediate impact of the shock is equivalent in the two factor returns, and as long as there is limited accommodation on the part of time spent in education – i.e. $\mathrm{d}u^{*}/\mathrm{d}N$ is low – the immediate effect of the pandemic is an **increase in earnings** and also an **increase in capital returns**, even though the regional **interest rate** is predicted to fall in response to the population shock.

From (7), the optimal allocation of labour is given by

$$u^{*} = \left(\frac{\left(1-\beta\right)A}{\delta}\right)^{1/\beta}\left(\frac{\theta_1}{\theta_2}\right)^{1/\beta}h^{\frac{\gamma}{\beta}-1}k \tag{19}$$

Apart from k there are two variables in the equation that can potentially be affected by the labour supply shock: θ_1 and θ_2. If the co-state variables were unaffected, we might expect an increase in the number of hours worked in production, since capital deepening (the increase in k) has increased labour productivity in this sector. However, this direct effect is likely to be counteracted in a reduction in the shadow cost of capital accumulation – represented by θ_1 – and thus the net effect on workforce allocation may be limited. Indeed, Boucekkine and Ruiz Tamarit (2005) show that for parameter values $\sigma = \beta$, the allocation of labour between production and education is not only constant over time, it is also unaffected by a labour supply

shock. In this special case, the immediate elasticity of wages and capital returns with respect to the population shock simply equals β: for each per cent excess mortality, we expect a β percent increase in wages and capital returns.

Turning to the medium-term consequences, the imbalance effects after the population shock are analysed by Boucekkine and Ruiz Tamarit (2005), again for a particular combination of parameter values. Denoting by ω the ratio between physical and human capital, it can be shown that the growth rate of the economy in the aftermath of the pandemic is going to be proportional to

$$\frac{\bar{\omega}}{\omega} = \frac{\bar{k}/\bar{h}}{k/h} \tag{20}$$

where $\bar{k}$ and $\bar{h}$ are the values along the balanced growth path. Since the pandemic will affect the average capital stock $k = K/N$ (and will not affect human capital, since there is no socioeconomic gradient), ω is above the long-run equilibrium value after the epidemic, and thus the growth rate in the economy is lower than otherwise.

We may thus summarise our theoretical predictions regarding earnings and capital returns as follows:

Hypothesis 1 *If the regional economies behave according to the Lucas-Uzawa model, and the accommodation of labour allocation u^* is incomplete, the influenza pandemic can be expected to lead to*

1. *An immediate relative increase in earnings w and capital returns rk in heavily affected regions compared to less affected regions.*

2. *A slower growth rate (in production, earnings and capital returns) in the heavily affected regions during a transition phase after the pandemic.*

Next, we also make some predictions concerning poverty. The analysis of poverty is complicated for two reasons. First, the theoretical model does not incorporate any worker heterogeneity and thus it is not useful for making explicit predictions for this variable. Second, the pandemic may have two distinct effects on poverty. First, individuals who were dependent on family members for their living might lose this support in the aftermath of the pandemic. This effect is quite immediate and not directly related to the functioning of the economy – even though

Boucekkine and Laffargue (2010) show that an increase in the number of orphans may have important distributional consequences in the long term. According to the 1920 census, on average each worker in Sweden supported one additional inhabitant (Statistics Sweden, 1926). Hence, given the lack of a social gradient in flu mortality, and considering that not all dependants who lost their support became poor, we can think of this number as an upper bound to this effect. Second, changes in wages and capital returns – and their distribution within the population – may have given rise to changes in poverty rates. Given the predictions from *Hypothesis 1* above, we expect these changes to cause an immediate reduction in poverty rates, followed by convergence between more and less affected regions during the transition period. Hence, we may formulate the following proposition:

Hypothesis 2 *When comparing regions at different levels of exposure, the initial effects of the pandemic on poverty will be the net effect of two countervailing forces:*

1. *An increase in poverty due to dependants losing their breadwinners; an effect likely to be smaller than one.*
2. *A relative decrease in poverty due to rising wages and capital returns.*

In the medium term, both effects are likely to lose importance; i.e., we expect to see a closing of the gap in poverty between heavily and less heavily affected regions.

Finally, we would like to emphasise that our analysis is carried out at the individual (per-capita) level so there is no need to be concerned with demand-side effects of the pandemic. It is of course true that elevated mortality rates will lead to a reduction in **aggregate** demand in most cases – even though this effect is likely to be of less importance in a small, open economy. However, if the shock is short and random, it represents a simple 'scaling down' of the economy and thus, no impact on the **individual-level** demand is to be expected.

2.2 Empirical Evidence

A growing literature tests the so-called Fetal Origins hypothesis, analysing the consequences of *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). These studies suggest long-term damage from 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.[4]

In this study, however, we are concerned with short- and medium-term aggregate effects of the pandemic. Up to now there very few empirical studies estimating this impact.[5] Besides, existing empirical studies face two serious problems. First, there is a lack of reliable data from the time period. Second, identification is difficult due, *inter alia*, to the fact that the flu occurred during and shortly after the World War I.

Brainerd and Siegler (2003) is one of 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 the analysis only focuses on the medium-term effects and does not distinguish whether the effect was due to recovery or economic growth. In any case, findings suggest significant positive effects: 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.

More recently Garrett (2009) analyses the effects of the pandemic on manufacturing wages. Using the same mortality data as Brainerd and Siegler (2003), but having access to wage growth between 1914 and 1919, the study can compare before and after the pandemic, but 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 results are attributable to the World War I.

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 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 methods

[4]There is a similar literature which documents the effects of childhood health and socio-economic background of parents on later outcomes (see for instance Smith, 2009; Banerjee et al., 2010; Buckles and Hungerman, 2012).

[5]In fact there is little empirical evidence on the economic effects of pandemics. Some scholars, see e.g.Johansson (2007), examine the short and medium-term economic implications of HIV/AIDS, but this on-going pandemic is very different compared to the Spanish flu and similar influenzas, as it is a much slower process.

typically used to conduct causal inference. The main reason appears to be a lack of reliable data. As shown below, Swedish data appear to offer a significant improvement in this regard.

2.3 Drivers of the Influenza

It has been argued 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) its *short duration*: 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.[6]

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 led 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 of surrounding cohorts.

Even though Brown raises valid concerns, it is unclear to what extent they apply to our study. The World War I also led to mobilisation and subsequent demobilisation in Sweden, but the disruption caused is of less importance when the short-term impact of the pandemic is concerned. Nevertheless, 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.[7] We find that this result also holds for Sweden (see Table 1).

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.

[6] See Levitt and List (2009) for a general discussion on inference from data generated by various types of (natural) experiments and necessary requirements.

[7] A related study from New Zealand (McSweeny et al., 2007) concludes that rural areas were less heavily affected by the 1918 influenza; however, the analysis fails to control for the age profile making this finding less informative.

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 also fail to find an association between influenza mortality and measures of population density or residential crowding.

3 Sweden in the early 20th century

To consider the particular economic environment which Sweden represented when the influenza pandemic struck in 1918, this section presents an overview of the general economic and political conditions in Sweden during and shortly after the First World War, and provides an overview of the spread of the influenza epidemic in Sweden.

3.1 General economic conditions

Following a surge in economic liberalisations in the second half of the 19th century, Sweden 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). The changes soon gave rise to rapid economic growth.

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 (Statistics Sweden, 1907), with a larger share working in the manufacturing sector in the more urbanized regions of the country. By 1930, 39.4 per cent of the population still earned their living from agriculture, compared to 35.7 per cent for manufacturing (Statistics Sweden, 1936). This structural change occurred at a relatively even pace during these three decades.

Sweden's transformation into a modern industrialised country was largely trade-driven. Figure 1 plots Swedish exports to key trading partners during the 1910-1930 period, expressed in 1917 crowns. Britain and Germany consistently accounted for a large share of Sweden's exports. Also, Scandinavian neighbours were important trading partners throughout the period, and their trade offered some stability in an otherwise fairly volatile environment. It should, however, be noted 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).

[Insert Figure 1 about here]

In terms of labour market regulations, the period considered falls before the labour movement's rise to power. Wages were relatively flexible and actually dropped in real terms in the 1913-17 period.

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 main disruption to Swedish trade was caused by external forces: the naval blockade imposed by the UK included the entire North Sea. 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, mainly imports were affected (Jörberg and Krantz, 1978).The war also led to increased regulation of the domestic economy. In 1916, new legislation authorised the government to regulate prices of groceries, fodder, fuel and clothing, leading to rationing of meat, eggs, butter and fish. However, a black market evolved and the regulations were of limited importance in practice (Schön, 2010).

Despite the disruption it brought to some parts of the economy, the war provided a favourable economic environment to Sweden. There was a massive surge in exports (iron ore, steel, engineering products) and a huge trade surplus evolved (Magnusson, 1996). Shortages in imported fuels led to the electrification of industry production all over the country – improving the competitiveness of Swedish industry. The agricultural sector also benefited from the shortfall in foreign competition (Schön, 2010). Following an increase in long term savings, the Swedish capital market was very liquid. Higher long term savings partly followed from that interest payments on accumulated debt remained within the country, but also from an increased scope for saving for low wage earners with new insurance companies and the establishment of new local banks (Larsson, 1998). Industrial investments of the period were thus to a large extent domestically funded and compared to the first phase of industrialization where companies mainly funded expansions with own capital returns, national and local banks now were their lenders (Gårdlund, 1947).

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 reversed in the post-war slump and thus might represent a confounding factor with respect to the regional exposure to the Spanish flu pandemic.

3.3 The Roaring Twenties

Sweden's period of economic growth was interrupted by a sharp downturn in 1920-21 in which GDP decreased by five per cent in a single year and dramatic increases in unemployment. 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). 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 (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 the sharp downturn of 1921 made them stop growing. Thus, the decade was also characterised by a gradual increase in returns to labour relative to capital returns (Schön, 2010). The fast growth in wages was partly due to the implementation of the shortening of the working day to eight hours in 1919 (Jörberg and Krantz, 1978).[8]

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. It killed almost 38,000 individuals, representing almost one per cent of the population. As in other parts of the world, flu prevalence rates were much higher, 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 south in late June 1918. 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 influenza deaths were reported, which is below the corresponding figure for 1917 (190 influenza deaths). Yet, once the situation changed in August and September, it did so with a terrifying speed.

[8] In 1910 the average work week corresponded to 57 hours of work. The working hour act of 1919 stated that a working week should not be longer than six days of work with no more than 48 working hours. Although the act in principle only covered workers in the industry, most workers legally not covered by the new legislation, e.g. employees in the service- and in the public sector, had corresponding working hour restrictions by collective agreements or regulations. For example, government officials had a 45-hour working week in 1920. Following seasonality, farm workers were covered by contracts regulating the maximum number of working hours on a yearly basis (Ryberg-Welander, 2000).

[Insert Figure 2 about here]

Figure 3 shows influenza mortality rates in Swedish counties 1918-1920 (per 100,000 inhabitants). Clearly flu mortality varied widely across counties, with some areas experiencing almost three times higher rates than others. In particular the counties *Jämtland* and *Västernorrland*, 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. However, 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 had been less exposed to earlier flu waves. Regarding immunity it has moreover been hypothesised that the W-shaped mortality distribution of the Spanish flu exhibited in Figure 4 may relate to exposure to the Russian flu in 1889-1890.

[Insert Figure 3 about here]

[Insert Figure 4 about here]

As discussed above, different industries fared differently during and after the war. Since different regions tend to be specialised in different industries, these fluctuations may become confounding factors. Table 1 tabulates all the counties, their influenza exposure and some key statistics from the 1910 census and the year just before the influenza pandemic, namely 1917, when available. Regions are ranked according to their 1918-20 influenza exposure. Interestingly, there is virtually no correlation between sectoral composition and Spanish flu mortality, suggesting that the spread of the influenza virus was largely unrelated to initial regional economic conditions.

[Insert Table 1 about here]

Normal flu waves affecting Sweden typically have their outbreak and peaks in February and March, but the Spanish flu peaked 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

public health measures, such as the banning of public gatherings (Influensakommittén, 1924). These actions however had limited effectiveness as the virus was transmitted through the air.[9]

Figure 5 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. 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, 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.[10]

[Insert Figure 5 about here]

3.5 Assessment

The purpose of section 3 was to give an overview of the environment in which the Spanish flu pandemic spread in 1918, with a particular focus on potential threats to the identification strategy employed. We have identified two main threats to the identification strategy which merit special attention. First, the downturn of the early 1920's 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, typically located in the countryside, were also particularly badly affected. However, as shown above there was no clear urban-rural divide in the influenza pandemic. A related issue is that the different regions may have specialised in different sectors of production, and these differences may not be fully captured by the urban/rural dichotomy. Although the spread of the virus does not correlate with initial regional economic conditions, the industries that benefited most from the war also had a less favourable evolution afterwards. Therefore it is essential to establish that the sectoral composition did not lead to regions already diverging during the war. This point calls for a careful investigation of the common time trend assumption for all outcome variables.

[9] There is also detailed documentation on the various treatments that were tested to prevent the spread of the flu in Sweden, see e.g. Influensakommittén (1924).

[10] 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 9.7. 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

Our analysis of the economic effects of the Spanish flu is conducted at the level of counties (Swedish: *län*). The data comes from high-quality administrative records. Sweden has a long tradition of collecting official statistics. Statistics Sweden was founded in 1858 and from 1911 onwards the bureau published the series *Sveriges officiella statistik*, divided into nine topics providing information on various issues, on a yearly basis. In addition, most public authorities have a convention of providing official statistics related to their activities.[11]

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 for national health services at the time. Medicinalstyrelsen's data tend to underestimate the number of cases and also report deaths by place of death and not place of residence (Hyrenius, 1914). Statistics Sweden, on the other hand, implemented more detailed and stricter reporting procedures in 1911, generating more complete death cause statistics and improving the reporting from rural areas (Hultkvist, 1940).[12] With respect to accuracy, reporting from urban areas were most likely, however, superior to reporting from rural areas, although it should be noted that special reporting procedures applied to deaths related to epidemics in both rural and urban areas (Hyrenius, 1914).[13]

We use data from Statistics Sweden on county-level influenza deaths reported on a yearly basis. As described below, we use this information together with monthly influenza incidence statistics from Medicinalstyrelsen (Kungliga Medicinalstyrelsen, 1930), to derive our treatment variable. Incidence data are of a lower quality than the mortality data due to the fact that the patient had to visit a physician to be recorded. However, doctors were obliged to report verified cases of the flu (Influensakommittén, 1924) and governmental historical records (see e.g. Influensabyrån, 1919) suggest that people did visit health care centres when they had the flu and that the pandemic clearly increased the demand for GPs.

[11] 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 their research assistants.

[12] Before 1911 there was no clear guidance on what could be defined as a death cause and how to record the main cause of death and often several death causes were reported in turn reducing data accuracy (Hyrenius, 1914). The new procedures likely also improved preciseness and the correctness of death cause statistics as the main death cause of a deceased hereinafter always was decided upon by a doctor. Clergymen had to make monthly reports on the likely cause of death of persons in cases where no doctor had been involved. These notes 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 Dödsorsaker 1911 (Statistics Sweden, 1915).

[13] Special reporting procedures also applied to violent deaths and suicide.

In baseline regressions we use yearly data for the time period 1911-1930 and focus on three economic outcomes. 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 and reported in the yearbook *Statistisk Årsbok* (e.g. Statistics Sweden, 1933).[14] We also use **earnings** per capita, referring to all taxed earnings from employment and pensions per capita collected from the same source.[15] From 1903 it was mandatory for all adults in Sweden to declare their incomes to the tax authorities. Everyone had to state their yearly earnings (including payment in kind and pensions), after deductions of pension contributions and for business expenses, and capital incomes to local tax boards that examined and controlled the declarations and those with an annual income of more than 600 crowns were taxed. Clearly there might be differences between the taxed income amount and actual incomes. However, as discussed by Roine and Waldenström (2009) the administrative routine in Sweden has been very thorough through the twentieth century and Swedish tax data are quite reliable. Moreover, contemporary sources report that the main difficulty was to get accurate information for property taxation rather than incorrectly reported incomes (Statistics Sweden, 1921).[16]

The third outcome variable is **poverty rates**, referring to the number of inhabitants in public poorhouses as a proportion of the total population in each region, collected from the yearly publication *Fattigvården*. People who were not able to support themselves or could not be supported by their family were eligible for the public poorhouses governed by the municipality (Statistics Sweden, 1911).[17] All applicants to poorhouses were carefully registered and exposed to a means test. An individual that was accepted to a poorhouse received housing, clothing, food, medical care and medicine, and the coverage of funeral costs, but was also declared legally incompetent (Rauhut, 2002).[18] Statistics Sweden provides information on the number of poor since 1871 when a new law demanding all municipalities to provide yearly statistical accounts is implemented. Information on the total number of poor, but also their sex, age and marital status was systematically reported to the authority using standardized forms (for an example,

[14] All monetary outcome variables are adjusted to real measures using 1917 as base year. The measure used for adjusting the variables is regional cost of living numbers provided by Statistics Sweden. All results are robust to using the Swedish national CPI, also available from Statistics Sweden.

[15] National pensions have basically always been regarded as taxable income in Sweden. As discussed by Elmér (1960) the amounts were small during the first decades and likely often not even declared.

[16] The standardized self declaration form had to be signed on word of honour. The punishment for submitting incorrect or improper information, which thereby led to that earnings were not taxed, was a fine between four to ten times the amount not reported (Riksskatteverket, 2003).

[17] As discussed by Elmér (1960) the 1913 pension reform reduced the number of old people in the poorhouses significantly wherefore demographic population structure should not explain regional variation in poverty.

[18] The inhabitants of poorhouses could consequently not vote in elections or referendums, nor get married or move away from the municipality.

see Jorner, 2008). 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. Notably, Statistics Sweden implement quality improving changes in their data collection routines from 1910.[19] Importantly, as discussed by Jorner (2008), Statistics Sweden's death causes are classified according to the 1911 nomenclature until 1930 and we have not noted any changes in any of the definitions of the other above indicators that could influence our results.[20]

Table 2 provides descriptive statistics for all variables. For the sake of comparability, all monetary variables have been expressed in 1917 crowns according to the average cost of living in the region. We provide averages of the variables for the period *before* (up until 1917), *during* (1918-20) and *after* (1920-30) the pandemic. Concerning the earnings variable – which together with the capital returns variable is based on official tax records – the numbers seem to be well in line with those available from other sources. As can be seen in the table, average taxable earnings per capita were 381 crowns during the 1918–20 period. The corresponding figure for 1920 only is 432 crowns (not shown). According to the 1920 census, male industrial workers earned 1,600 crowns per year on average (females 1,000), whereas agricultural workers typically earned less than 1,000 crowns and female workers in agriculture as little as 544 crowns.[21] Thus, if one considers the fact that only 49.9 per cent of the population was working, the average earnings of 432 crowns for 1920 seem to be of a similar order of magnitude.

[Insert Table 2 about here]

A concern is that the estimated effect of influenza mortality may actually be capturing long-lasting effects of influenza *infections*. 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). Moreover, both historical and medical records suggest that some Spanish flu survivors experienced a deterioration in health (e.g. chronic bronchitis,

[19] From 1910 data is more often collected directly from relevant informants rather than from administrative sources. The new routines follows from the investigation presented by the 1905 statistical committee on how Statistics Sweden should accomplish their mission (Jorner, 2008).

[20] As described by Elmér (1960) the Swedish pension system was basically unchanged during the period 1913-1936. Recent research also indicates that there have not been any systematic changes in the level of tax avoidance and evasion during the studied time period (Roine and Waldenström, 2009)

[21] Own calculations based on Statistics Sweden (1926) and a CPI deflator of 1.6524.

drowsiness, sleeping sickness) in later life (see e.g. Collier, 1996; Ravenholt and Foege, 1982), in line with recent findings in neuroscience on influenza effects unrelated to the viral infection per se Jang et al. (2009). Thus, we include influenza incidence in separate specifications as a robustness check. 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. Moreover, we also include further controls such as population density and the birth rate. Finally, internal and external migration might also influence our dependent variables (see for instance D' Amuri et al., 2010, on the effects of migration on the labour market).

[Insert Figure 6 about here]

Another concern regarding the internal validity is the volatility of the world economy during the time period studied. Section 3 suggests 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 disruption to the 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 biased.

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 some 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[22], where Swedish exports to other countries are explained with reference to their distance, their GDP and their GDP per capita:

$$\ln(PX_{st}) = \delta_0 + \delta_1 \ln(GDP_{st}) + \delta_2 \ln(GDP_{st}/Pop_{st}) + \delta_3 D_s + \upsilon_{st} \tag{21}$$

where PX_{st} are Swedish exports to country s in year t, GDP_{st} is the gross domestic product of country s in year t, Pop_{st} is the population size, and D_s is the distance from Stockholm to

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

the capital of country s. We estimate equation (21) 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 $\widehat{PX}_{it}$ for each county i. This variable refers to the total exports that would be expected in year t if Sweden were located at the centroid of county i:

$$\widehat{PX}_{it} = \sum_{s=1}^{27} e^{\hat{\delta}_0} GDP_{st}^{\hat{\delta}_1} \left(\frac{GDP_{st}}{Pop_{st}} \right)^{\hat{\delta}_2} D_{si}^{\hat{\delta}_3} \tag{22}$$

where D_{si} now represents the distance between county i and country s. Clearly, $\widehat{PX}_{it}$ has no obvious interpretation in economic terms, partly because equation (21) 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

We use an extension of the difference-in-differences method to estimate the effect of the epidemic. Thus, our inference concerning the effect will be based on a comparison of changes over time between regions experiencing different levels of exposure. By contrasting different regions, we are able to disregard confounding factors captured in nation-wide trends in the different outcome variables. In this section, we first present our 'treatment variable' capturing the degree of excess mortality in different regions. Then we proceed with a discussion of various threats to our identification strategy. Finally we present our estimator and discuss how to get appropriate standard errors.

5.1 Defining the Treatment Variable

Our analysis is conducted at the level of the 25 Swedish counties. As 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 total excess regional influenza mortality through the years 1918-20. In our baseline specifi-

cations, we furthermore assume that the effects of Spanish flu mortality is constant over time and a linear function of the excess mortality.

When calculating the excess mortality rates, two issues are of central importance: taking regional age structures into account, and adjusting the treatment indicator for the timing of the pandemic. We start out by explaining how the annual excess mortality rate is calculated, and then proceed to consider the timing issue.

Denote by n_{it}^d the relative population size of population group d in county i at time t. Likewise, denote by q_t^d the influenza mortality rate of demographic group d in year t at the national level. Then, the **predicted** influenza mortality rate of county i in year t is given by $\hat{m}_{it} = \sum_{d \in D} n_{it}^d q_t^d$ where D is the set of demographic groups into which the population has been partitioned. We have information on n_{it}^d and q_t^d for 80 distinct demographic groups, and may thus define the age-standardised excess mortality based on the difference $m_{it} - \hat{m}_{it}$, where m_{it} is the actual mortality rate. This fraction represents the proportion of the regional mortality rate in year t which cannot be attributed to demographic factors.[23]

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

Thus, we introduce some additional notation notation: monthly flu morbidity in county i denoted p_{it}^j – where t is the year and j is the month. We define both m_{it}^e and p_{it}^j as proportions of the county population at the end of year $t-1$. Using these variables, we can define the **effective excess mortality** m_{it}^e in year $t > 1917$ as

$$\begin{aligned} m_{it}^e &= (m_{it} - \hat{m}_{it}) \frac{\sum_{j=1}^{12} \left(0.5 \cdot p_{it}^j + \sum_{k=1}^{j-1} p_{it}^k\right)}{12 \sum_{k=1}^{12} p_{it}^k} \\ &= (m_{it} - \hat{m}_{it}) \frac{\sum_{j=1}^{12} (12.5 - j)\, p_{it}^j}{12 \sum_{k=1}^{12} p_{it}^k} \end{aligned} \tag{23}$$

[23] We also considered defining excess mortality in comparison to the regional-level average of three pre-influenza years. This alternative specification delivered very similar results, but since the increase in mortality could be confounded by age structures, we decided to use the approach presented here instead.

Considering the second line, the term $(m_{it} - \hat{m}_{it})$ represents the yearly excess mortality, defined as above. The fraction introduces weighting to account for the timing of the flu. Its denominator standardises the weights to give a number between zero and one – where the value zero corresponds to all deaths occurring at the end of the year, and the value one corresponds to all deaths occurring at the beginning of a year. Consider, for example, a situation where the flu appears in two waves in February and October with $p_{it}^{2} = 0.1$ and $p_{it}^{10} = 0.5$ but $p_{it}^{j} = 0$ for all other months. Then, the denominator would equal 7.2 and the numerator $1.05 + 1.25 = 2.3$. Thus, the annual excess mortality rate would get multiplied with the adjustment factor 0.32 in this case.

Having thus defined the excess flu mortality within a year, we can calculate the cumulative excess mortality at an annual basis. We define our treatment variable w_{it} as:

$$w_{it} = \begin{cases} 0 & \text{if } t < 1918 \\ \sum_{j=1918}^{t-1} (m_{ij} - \hat{m}_{ij}) + m_{it}^{e} & \text{if } t \in [1918, 1920] \\ \sum_{j=1918}^{1920} (m_{ij} - \hat{m}_{ij}) & \text{if } t > 1920 \end{cases} \tag{24}$$

where, notably, previous years are represented by m_{ij}, not m_{ij}^{e}: in any year t, we do not need to correct for the timing of period $t-1$ deaths as these individuals are now missing throughout the year.

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 (Lechner, 2010). Thus, the functional form imposed adds a further assumption to the standard set of assumptions, and it should clearly be formally tested.

5.2 Identification

Our empirical analysis crucially rests on the exogeneity assumption, i.e., that the regional exposure to the influenza pandemic was essentially random, and in particular, not correlated with potential outcomes. This assumption is not directly testable, but since it is essential for identification, we have exposed it to a battery of indirect tests, which are described in detail below.

Visual inspection of time trends. The common time trend assumption appears more plausible if one can show that regions with different exposure to the influenza have moved together in the past. Thus, we split the sample into two groups and plot time trends and

confidence intervals for all outcome variables considered. If the trends of the two groups diverge already before the 'treatment' in 1918, this is evidence suggesting that the common time trend assumption is not warranted. This graphical test will be performed before the main regressions.

Placebo regressions. By counterfactually assuming that the influenza pandemic hit Sweden between 1915-17 instead of 1918-20, we get an indirect test of not only the common time trend assumption: a placebo regression also tells us something about the statistical properties of the estimator. An insignificant but precisely estimated placebo coefficient suggest that we have acceptable size, whereas an estimate which is significantly different from zero either suggests that the common time trend assumption is violated, or that false positives is an issue. False positives may arise whenever the standard errors are downward biased – for example due to temporal or spatial autocorrelation – and the placebo regressions thus represent a useful test as to whether our dataset suffers from any of these problems. The specification applying a placebo approach is included as one of our regressions.

Relating influenza exposure to pre-influenza covariates. This test goes beyond what is actually necessary for the DID estimator to work, since it is not required that counties are at the same levels before the intervention – only that they follow common time trends. Nevertheless, given the geographical gradient in the influenza, there is the concern that our estimates are confounded by differences in the sectoral composition of the economy and other distinct traits of the pre-influenza regional economies. Thus, we calculate the correlation between our 'treatment' variable and various economic indicators. Doing so, we clearly face a multiple testing problem: random variation in these variables would sooner or later lead to us finding a strong correlation with some covariate. Nevertheless, for identification we require that the correlation between influenza exposure and these additional variables is negligible. Section 3.4 discusses and Table 1 presents the results for this test.

Region-specific time trends. In a separate set of specifications, we allow the regions to diverge over time by including region-specific time trends. Including these trends demands more of the data since more parameters need to be estimated and since there is a risk of multicollinearity with our treatment indicator. For this reason, it may be expected that results are somewhat weaker when these trends are included. Hence, we interpret point estimates which do not deviate significantly from our baseline specification as evidence that the estimated effect may indeed be interpreted as causal. The specification applying region-specific time trend is included among our other regressions.

Inclusion of covariates. The DID estimator does not allow inclusion of endogenous variables such as covariates that are possibly also affected by the influenza. However, there are some variables that are plausibly exogenous. It seems reasonable to assume that influenza morbidity (i.e. infections) was exogenous, and also the economic performance of vital trading partners can be assumed to be exogenous from the point of view of the regional economy. If the treatment indicator is truly exogenous, it should be robust to the inclusion of such covariates. However, we take this analysis one step further by also considering covariates which are potentially endogenous. This obviously gives rise to a 'bad control problem' (Angrist and Pischke, 2008), so the estimates need to be interpreted with caution, but it does seem reasonable to check whether results are robust to the inclusion of possible confounders such as birth rates, population density, degree of urbanisation, and internal migration. Specifications including additional covariates are included in our regressions.

Collapsing regions. There are several issues related to the spatial structure of the dataset. First, there is the already mentioned problem of spatial dependence of various kinds, which would lead to a violation of the *Stable unit treatment value* assumption (SUTVA). Second, we expect migration of workers and capital to even out some of the impact of the pandemic. Third, capital returns are typically registered in the county of the capital holder, which does not have to be the same region as the region where the capital is located. All of these issues can be addressed to some extent by collapsing the counties into larger geographical units. Thus, in a separate set of specifications we collapse the 25 counties into six 'super-regions' with approximately one million inhabitants each. Migration movements between these larger units are much smaller than between the original regions – and thus this alternative specification provides a useful test of whether our results are driven by these other factors.[24] The additional estimations using data for larger regions are presented in the Appendix.

5.3 Empirical Specification

For all outcome variables considered the main baseline specification is

$$y_{it} = \alpha_i + \beta w_{it} + \lambda_t + \epsilon_{it} \tag{25}$$

where y_{it} is the outcome variable (i.e. capital returns, earnings or poverty), α_i is a county fixed

[24] We also run a specification including a spatial lag of the treatment variable – an indication of the flu mortality in neighbour regions weighted by distance.

effect, w_{it} is our treatment indicator, 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 standard assumptions are fulfilled.

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 counties are at very different levels at the outset with respect to the outcome variables, a logarithmic specification seems preferable. However, as a robustness check the Appendix also provides estimates for the outcome variables specified in levels.

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

$$y_{it} = \alpha_i + \beta w_{it} + \gamma w_{it} \mathbf{1}\left(t > 1920\right) + \lambda_t + \epsilon_{it} \tag{26}$$

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 1920.

The placebo regression will take a very similar form:

$$y_{it} = \alpha_i + \delta w_{i,t+3} + \lambda_t + \epsilon_{it} \text{ if } t < 1918 \tag{27}$$

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. Since our estimates are based on relatively long panels, particular attention needs to be devoted to autocorrelation.[25]

In a seminal paper, Bertrand et al. (2004) discuss the problems associated with autocorrelation in difference-in-differences studies and compare different solutions. One solution which is

[25]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 major problem.

not discussed in their paper, but outlined by Wooldridge (2009), Stock and Watson (2008) and Arellano (2003), is to use robust standard errors in a fixed effects specification. This combination, which we use in our baseline specifications, is equivalent to clustering at the regional level and thus deals with the autocorrelation problem.

As an additional robustness check, we also reduce the time dimension into five time periods. The estimating equations remain the same as those above, but we now use a collapsed version of the outcome variable, defined as follows:

$$\tilde{y}_{it} = \begin{cases} \frac{1}{T_0} \sum_{s=t_0}^{1917} y_{is} & \text{if } t = 1917 \\ y_{it} & \text{if } t \in [1918, 1920] \\ \frac{1}{T_1} \sum_{s=1921}^{t_1} y_{is} & \text{if } t = 1921 \end{cases} \tag{28}$$

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. The treatment variable $\tilde{w}_{it}$ is defined analogously:

$$\tilde{w}_{it} = \begin{cases} 0 & \text{if } t = 1917 \\ w_{it} & \text{if } t \in [1918, 1921] \end{cases} \tag{29}$$

Thus, we require estimated effects to be robust to this change in specification.[26]

6 Results

6.1 Common Time Trend: Visual Evidence

Our case differs from the standard DID setting in the sense that we have more than two degrees of treatment intensity, and hence 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 -140 to 50 excess deaths per 100,000 population (see column 2 of Table 1). Above that, there

[26] A third alternative would be to rely on the GLS estimator originally suggested by Kiefer (1980). 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. 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.

is a smaller group of six counties which experienced between 82 and 398 excess deaths. To provide some visual evidence concerning the common time trend assumption we contrast these two groups.[27] In Figures 7 to 9, counties are weighted by their 1917 population size[28], and all monetary variables are expressed in 1917 crowns (adjusted according to the regional price level obtained from average regional cost of living). The solid curve in Figure 8 pictures growth in log capital incomes for counties which were hit particularly hard by the epidemic. The dotted curve plots the corresponding series for the less severely affected counties, while the grey curves show 95 % confidence intervals.

[Insert Figure 7 about here]

[Insert Figure 8 about here]

[Insert Figure 9 about here]

The graphs indicate that the common time trend is a reasonable assumption before the pandemic hit: the curves are quite close and their confidence intervals overlap. However, during and after the pandemic, the two groups diverge for most outcome variables.

Figure 8 suggests that more strongly affected counties experienced slightly slower earnings growth, and Figure 9 suggests that poverty increased by the pandemic. Both observations might be driven by a change in poverty rates. Thus, 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 10 provides visual evidence for the modified earnings variable. The common time trend assumption appears to be equally plausible as for the original variable, and no other important changes are discernible as well.

[Insert Figure 10 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 some of our outcome variables. Clearly, however, the above evidence is too crude and summaric to provide a reliable estimate of the effects. Hence, we now turn to more rigorous regression-based evidence.

[27] Allowing for more groups does not change the results, but makes the figures more difficult to read.
[28] The results not using any weights are very similar to the results presented in the text.

6.2 Regression Analysis

Table 3, Panel A presents the results for **capital income**.[29] The first column presents the overall effect of the pandemic. According to our estimate, each additional death per 100,000 inhabitants was associated with a reduction in capital income per capita by 0.082 per cent. To get an idea about the magnitude, one may compare the 25th and the 75th percentile, with an escess mortality of -0.096 and 0.035 respectively. The difference between these two counties would correspond to a reduction in capital incomes per capita by 11 per cent.

In the second column, we contrast the effects *during* (1918–20) and *after* (1921–30) the pandemic (as defined in equation 26). Parts of the effect are discernible during the pandemic itself, and there is possibly is an additional effect kicking in afterwards. In columns three and four we include *regional time trends*. As this variable exhibits a strong correlation with our treatment variable it is not surprising that the estimated effect weakens somewhat. Columns five and six control for cumulative morbidity and current morbidity respectively. The seventh column presents estimates controlling for export shocks (see the discussion before equation 21 for further details). Moreover, in column eight we include additional control variables (birth rates, internal and external migration, population density, percent of rural population).[30] Throughout these different specifications the variation in the estimated treatment effect is very limited. Finally, column nine allows for a 'placebo epidemic' (from equation 27). This estimate is nowhere near statistical significance and it is very precisely estimated. Thus, our observation from Figure 7 is confirmed and the common time trend assumption is maintained.

In columns ten and eleven, we collapse the time period into five periods in order to reduce problems related to autocorrelation (see equations 28 and 29 for details). The estimates clearly indicate that autocorrelation is of limited importance. Nevertheless, the estimated effects are still significant at the one per cent level. Thus, we may conclude that we have found very strong and robust evidence of a substantial immediate effect of the pandemic on capital returns, and some evidence of a further reduction in capital returns after the pandemic.

[Insert Table 3 about here]

Table 3, Panel B provides estimates for the **earnings** variable. For this outcome there

[29] In the baseline analysis we take the natural logarithm of all dependent variables. In the Appendix we provide estimates for the outcome variables in levels (Table A-1).

[30] We also perform a regression with migration as a dependent variable to see whether the Spanish flu affected migration behaviour. The treatment indicator turns out to be insignificant suggesting that people were not "fleeing" from the flu.

is much less evidence of a flu effect. The point estimate of the overall effect is −0.2, which, according to our previous comparison, would imply a relative decline of 2.9 per cent in the 75th percentile county compared with the 25th percentile. Importantly, the placebo estimate is smaller 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 per capita.[31] Next, Panel C presents the results when focusing on earnings of the non-poor population. Normalising earnings using the non-poor instead of the total population does not change our conclusions: the estimated effect is still insignificant and very similar to our previous estimates.

Table 3, Panel D 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 0.74. Comparing the 25th and the 75th percentile, the difference in flu mortality would give rise to an increase in poverty by 9.7 per cent. Again, the influenza effect is quite substantial – but it only appears after the pandemic receded. It is important to remember that there is a direct mechanism at work, which has little to do with the functioning of the economy, to the extent that deceased individuals leave dependants behind, who are unable to support themselves. Two pieces of evidence however suggest that this factor is not the main driver of the positive relationship between the influenza and poverty. First, we performed an additional analysis on disaggregated poverty statistics. This analysis shows that the baseline effect is neither driven by widows nor by orphans. Secondly, a close inspection of Panel C in the Appendix Table A-1 further reveals that dependants 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 implausible that all newly poor would were dependants of a deceased person.

Also for poverty the placebo estimate is insignificant, small and precisely estimated. Thus, in line with the visual evidence, the common time trend assumption seems to be confirmed also in this case. We conclude that the pandemic appears to lead to a large increase in poverty rates in the medium term.

The Appendix provides estimates for the outcome variables in levels (Table A-1). Clearly, as discerned from the reported R^2, our less preferred specification performs much worse in

[31] We also obtained data on agricultural wages. Data are not available for all counties and hence we do not include the results; however, the empirical evidence for this variable suggests that no flu-effect was observed on agricultural wages either.

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.[32]

As mentioned in Section 5.1, several concerns about confounders in the analysis may be addressed by collapsing the 25 counties into larger geographical units. The estimates from these 'super-regions', also presented in Table A-1 in the Appendix, show that baseline results are robust to this alternative regional division.

In order to further check the robustness of our findings, we perform additional regressions not included in this version of the paper. First, to handle potential spatial heterogeneity of regions, we run regressions including the spatial lag of the treatment variable – i.e. the flu mortality in neighbour regions weighted by distance. Our point estimates of the treatment effect on poverty is hardly affected and also the non-finding for earnings persists. However, for capital income the standard error increases, reducing statistical significance. Second, 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. Baseline results are completely unaffected by this modification. A third concern is whether the pandemic actually came as a surprise (especially the later waves). In order to address potential anticipation effects we estimate the effect including only the treatment of 1918. Baseline findings are not affected by these changes. Finally, to avoid the potential bias following from deaths caused by the flu being recorded as pneumonia cases – which, according to Figure 2, should not be a big problem – we also combine information on influenza and pneumonia from Statistics Sweden to derive a second version of our treatment variable. Results are not affected.

In conclusion, we find strong evidence for the pandemic having a positive impact on poverty in the medium term, and also strong evidence of an immediate and lasting negative effect on capital returns. However, there is no evidence whatsoever that earnings were affected by the pandemic. Placebo estimates are insignificant and close to zero suggesting that the common time trend assumption can be retained in all cases.

[32]Since all our dependent variables are weighted by total population and the number of inhabitants is directly affected by the flu, we also re-estimate our regressions dividing them by the population of 1917. This does not affect our results.

6.3 Discussion

Figure 11 summarises our main findings. We have calculated the impact of an increase in excess influenza mortality corresponding to the interquartile range between counties.[33] Clearly, most of the predictions delivered by the theoretical model in Section 2 failed to be confirmed by our empirical analysis. We do not observe the expected immediate increase in wages – instead, the immediate impact on earnings is negative throughout, but the point estimate is small and nowhere near statistical significance at conventional levels. Besides, we observe a rapid decline in capital returns, even though these are predicted to increase by the same proportion as wages. Moreover, we observe an effect of the pandemic on poverty which goes far beyond the direct effect coming from dependants losing their breadwinners: on average, each influenza death resulted in four individuals moving into poorhouses. This finding suggests that poverty rates would have increased even if these dependants could be disregarded.

[Insert Figure 11 about here]

On the other hand, our results clearly suggest that more heavily affected counties experienced slower growth than the less affected ones in the aftermath of the pandemic. This appears to be the only prediction of the theoretical model which is not rejected by our empirical analysis. Since regional GDP is made up of returns to labour and capital, it is quite clear that the regional economies suffered a setback in economic activity during the pandemic (capital returns dropped and wages remained constant) which was reinforced in the years following the pandemic (capital returns dipped further whereas no further change in wages was observed).

So how should our results be interpreted? In order to answer that question, it would surely be of interest to see whether there were adjustments in labour supply following the pandemic. Our results from the super-regions (Table A-1) clearly suggest that internal migration can be ruled out as a driver of our results. However, another possibility is that the employment rate was affected. Unfortunately, the data availability is very limited on this point. We only have some information on labour supply in factories, which we have used in additional regressions. Table 4 contains regressions using the proportion of child, adult female and adult male industrial workers in the entire population as outcome variable. The last four columns use the composition of the industrial sector workforce as outcome variable.

[Insert Table 4 about here]

[33] The interquartile range is 0.083 during the pandemic and 0.131 for the years after the pandemic.

According to these estimates, the pandemic led to a considerable effect on the labour supply of females and minors: each deceased person was replaced by 0.45 minors and 0.42 females – whereas the effect on male labour supply is insignificant. Once the timing of effects is taken into consideration, it becomes cear that minors were more responsive in the short term, whereas in particular males increased their labour supply in the aftermath of the pandemic – a finding which probably reflects differences in supply elasticities between these two groups.

Interestingly, the results suggest that the mortality shock might have been overcompensated by an expansion in labour supply. However, we would like to emphasise that the quality of these labour supply data is not as high as for the other outcome variables – and this is why we have not included them amongst our main results. Despite this caveat, however, the results give some hints as to why our findings are seemingly irreconcilable with theoretical predictions. But the story is far from straightforward. If the observed increase in industrial-sector labour supply is representative for the labour market as a whole, it would surely counteract the effects of capital deepening on wages and interest rates. However, per capita earnings would still increase as a result of an increase in the regional employment rate. On the other hand, the workers replacing deceased ones are likely to be less productive – in particular as far as minors are concerned – and there could well be an increased mismatch between labour and capital. This combined effect of capital deepening and a lowered average worker quality might then well lead to earnings remaining stable and a drop in capital returns.

Our preliminary conclusion would thus be that the assumption of human capital h being unaffected by the shock is faulty. If one allows for human capital h to include other aspects of productivity than education – such as age and experience – then it may of course be the case that the human capital per worker is affected by the shock, despite our inability to detect any signs of a socio-economic gradient in the pandemic. This observation seems to confirm recent advances in quantitative macroeconomics, which indicate that representative agent models are, by construction, ill-suited to answer many important policy questions (Heathcote et al., 2009) precisely due to the lack of an age dimension.

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. First, its provides an estimate of the actual economic consequences of the 1918 pandemic. Second, it gives us an idea of the possible effects of current and future pandemics on the performance of the economy. Third, 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.

We show that the Lucas (1988) model of endogenous growth delivers a set of clear predictions of how an economy can be expected to react to a epidemic of this kind. The immediate effect will be an increase in wages and capital returns, and a reduction in interest rates. This effect is a direct consequence of capital deepening and it will normally not be accommodated by workers moving from education into production. For the medium term, the model instead predicts negative imbalance effects on growth as long as the ratio of physical capital to human capital remains above the long-run equilibrium value.

Our study finds 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, 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 that our main results are robust to the inclusion of variables capturing different aspects of influenza incidence. Thus, it is our tentative conclusion that differences in excess mortality rates across regions are largely exogenous.

Our main findings are generally very robust. 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 medium-term responses. According to our estimates, the highest quartile (with respect to influenza mortality) experienced a drop of 5 per cent during the pandemic and an additional 6 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 had receded in 1920. For this variable, the top quartile suffered an increase in poverty by 11 per cent compared to the bottom quartile.

Strong as these results may seem, they do not fit very well with the most popular macroeconomic models. On the one hand, we do find that heavily affected counties had lower growth rates after the epidemic. On the other hand, our findings that earnings were unaffected and that capital incomes dropped is much more difficult to explain. Likewise, our finding that poverty

rates increased is also difficult to reconcile with the increased scarcity of labour. In order to shed some light on these surprising findings, we have presented some indirect evidence on the labour supply reaction to the pandemic. Our analysis shows that results are not driven by internal migration, but the pandemic appears to have increased regional employment rates in the industrial sector. This finding clearly suggests that the pandemic led to a significant reduction in average worker quality. This labour market adjustment, which we have only been able to study for one sector of the economy, may thus be an explanation to the unexpected results. In this way, our study provides evidence that heterogeneity of the labour force needs to be taken into account when analysing the effects of a pandemic.

References

Acemoglu, D., Johnson, S., and Robinson, J. (2003). Disease and development in historical perspective. *Journal of the European Economic Association*, 1(2-3):397–405.

Almond, D. (2006). Is the 1918 influenza pandemic over? Long-term effects of in utero influenza exposure in the post-1940 US population. *Journal of Political Economy*, 114(4):672–712.

Almond, D. and Mazumder, B. (2005). The 1918 influenza pandemic and subsequent health outcomes: An analysis of SIPP data. *The American Economic Review*, 95(2):258–262.

Alvarez-Cuadrado, F. (2008). Growth outside the stable path: Lessons from the European reconstruction. *European Economic Review*, 52(3):568–588.

Åman, M. (1990). *Spanska sjukan: Den svenska epidemin 1918-1920 och dess internationella bakgrund*. Ubsaliensis Academiae.

Anderson, J. (1979). A theoretical foundation for the gravity equation. *The American Economic Review*, 69(1):106–116.

Angrist, J. and Pischke, J. (2008). *Mostly harmless econometrics: An empiricist's companion*. Princeton University Press.

Arellano, M. (2003). *Panel Data Econometrics*. Oxford University Press.

Banerjee, A., Duflo, E., Postel-Vinay, G., and Watts, T. (2010). Long-run health impacts of income shocks: Wine and phylloxera in nineteenth-century France. *The Review of Economics and Statistics*, 92(4):714–728.

Barry, J. (2005). *The great influenza: The epic story of the deadliest plague in history*. Penguin Group USA.

Bergh, A. (2007). *Den kapitalistiska välfärdsstaten*. Norstedts akademiska förlag.

Bertrand, M., Duflo, E., and Mullainathan, S. (2004). How much should we trust differences-in-differences estimates? *Quarterly Journal of Economics*, 119(1):249–275.

Bloom, D. and Mahal, A. (1997). AIDS, flu, and the Black Death: Impacts on economic growth and well-being. In *The Economics of HIV and AIDS: The Case of South and South East Asia*, pages 22–52. Delhi: Oxford University Press.

Bos, K., Schuenemann, V., Golding, G., Burbano, H., Waglechner, N., Coombes, B., McPhee, J., DeWitte, S., Meyer, M., Schmedes, S., et al. (2011). A draft genome of yersinia pestis from victims of the black death. *Nature*, 478(7370):506–510.

Boucekkine, R. and Laffargue, J. (2010). On the distributional consequences of epidemics. *Journal of Economic Dynamics and Control*, 34(2):231–245.

Boucekkine, R. and Ruiz Tamarit, R. (2005). Imbalance effects in the Lucas model: An analytical exploration. *Topics in Macroeconomics*, 4(1).

Brainerd, E. and Siegler, M. (2003). The economic effects of the 1918 influenza epidemic. Working Paper 3791, Centre for Economic Policy Research.

Brown, R. (2010). The 1918 US influenza pandemic as a natural experiment, revisited. *mimeo*.

Buckles, K. S. and Hungerman, D. M. (2012). Season of birth and later outcomes: Old questions, new answers. *Review of Economics and Statistics*.

Chowell, G., Bettencourt, L., Johnson, N., Alonso, W., and Viboud, C. (2008). The 1918–1919 influenza pandemic in England and Wales: Spatial patterns in transmissibility and mortality impact. *Proceedings of the Royal Society B: Biological Sciences*, 275(1634):501.

Collier, R. (1996). *The plague of the Spanish lady: the influenza pandemic of 1918-1919*. Allison & Busby.

D' Amuri, F., Ottaviano, G. I., and Peri, G. (2010). The labor market impact of immigration in Western Germany in the 1990s. *European Economic Review*, 54(4):550–570.

Donald, S. and Lang, K. (2007). Inference with difference-in-differences and other panel data. *The Review of Economics and Statistics*, 89(2):221–233.

Elmér, Å. (1960). *Folkpensioneringen i Sverige, med särskild hänsyn till ålderspensioneringen: Old age pensions in Sweden*, volume 17. CWK Gleerup, Lund.

Gårdlund, T. (1947). *Svensk industrifinansiering under genombrottsskedet, 1830-1913*. Svenska bankföreningen.

Garrett, T. (2008). Pandemic economics: The 1918 influenza and its modern-day implications. *Federal Reserve Bank of St. Louis Review*, 90(2):75–93.

Garrett, T. (2009). War and pestilence as labor market shocks: US manufacturing wage growth 1914–1919. *Economic Inquiry*, 47(4):711–725.

Glick, R. and Taylor, A. M. (2009). Collateral damage: Trade disruption and the economic impact of war. *The Review of Economics and Statistics*, 92(1):102–127.

Hausman, J. and Kuersteiner, G. (2008). Difference in difference meets generalized least squares: Higher order properties of hypotheses tests. *Journal of Econometrics*, 144(2):371–391.

Heathcote, J., Storesletten, K., and Violante, G. L. (2009). Quantitative macroeconomics with heterogeneous households. *Annual Review of Economics*, 1(1):319–354.

Hultkvist, G. (1940). Några anmärkningar till vår nya dödsorsaksstatistik. *Allmänna Svenska Läkartidningen*, 17:58–60.

Hyrenius, H. (1914). Den svenska officiella dödsorsaksstatistiken. *Social-medicinsk tidskrift*, 11:1179–1188.

Influensabyrån (1919). *Koncept. Registratur 1918–1919*. Stockholm: Kungl. Medicinalstyrelsen.

Influensakommittén (1924). Influensakommitténs rapport. *Svenska Läkartidningen*, 10:1–32.

Jang, H., Boltz, D., Sturm-Ramirez, K., Shepherd, K. R., Jiao, Y., Webster, R., and Smeyne, R. J. (2009). Highly pathogenic H5N1 influenza virus can enter the central nervous system and induce neuroinflammation and neurodegeneration. *Proceedings of the National Academy of Sciences*, 106(33):14063–14068.

Johansson, L. M. (2007). Fiscal implications of AIDS in South Africa. *European Economic Review*, 51(7):1614–1640.

Johnson, N. and Mueller, J. (2002). Updating the accounts: Global mortality of the 1918-1920 "Spanish" influenza pandemic. *Bulletin of the History of Medicine*, 76(1):105–115.

Jörberg, L. and Krantz, O. (1978). *Ekonomisk och social politik i Sverige, 1850-1939*. Ekonomisk-historiska institutionen, Lunds universitet.

Jorner, U. (2008). *Summa summarum – SCB:s första 150 år*. Statistiska Centralbyrån (SCB)-Tryck, Örebro.

Kesternich, I., Siflinger, B., Smith, J., and Winter, J. (2013). The effects of World War II on economic and health outcomes across Europe. *Review of Economics and Statistics*.

Kiefer, N. (1980). Estimation of fixed effect models for time series of cross-sections with arbitrary intertemporal covariance. *Journal of econometrics*, 14(2):195–202.

Krantz, O. and Schön, L. (2007). *Swedish historical national accounts 1800-2000*. Almqvist & Wiksell International.

Kungliga Medicinalstyrelsen (1912-1930). *Allmän hälso- och sjukvård*. Stockholm: P.A. Norstedt & söner.

Larsson, M. (1998). *Staten och kapitalet-det svenska finansiella systemet under 1900-talet (The state and the capital-Sweden's financial system during the 20th century)*. Stockholm: SNS förlag.

Lechner, M. (2010). The estimation of causal effects by difference-in-difference methods. *University of St. Gallen Department of Economics working paper series 2010*.

Levitt, S. D. and List, J. A. (2009). Field experiments in economics: the past, the present, and the future. *European Economic Review*, 53(1):1–18.

Little, T., Garofalo, C., and Williams, P. (1918). The absence of the bacillus influenzæ in the exudate from the upper air-passages in the present epidemic. *The Lancet*, 192(4950):34–34.

Lucas, R. (1988). On the mechanics of economic development. *Journal of Monetary Economics*, 22(1):3–42.

Maccini, S. and Yang, D. (2009). Under the weather: Health, schooling, and economic consequences of early-life rainfall. *American Economic Review*, 99(3):1006–1026.

Magnusson, L. (1996). *Sveriges ekonomiska historia*. Rabén Prisma.

Mamelund, S. (2006). A socially neutral disease? Individual social class, household wealth and mortality from Spanish influenza in two socially contrasting parishes in Kristiania 1918-19. *Social Science & Medicine*, 62(4):923–940.

McSweeny, K., Colman, A., Fancourt, N., Parnell, M., Stantiall, S., Rice, G., Baker, M., and Wilson, N. (2007). Was rurality protective in the 1918 influenza pandemic in New Zealand? *Journal of the New Zealand Medical Association*, 120(1256).

Morens, D. and Fauci, A. (2007). The 1918 influenza pandemic: Insights for the 21st century. *Journal of Infectious Diseases*, 195(7):1018.

Nelson, R. (2010). Testing the fetal origins hypothesis in a developing country: Evidence from the 1918 influenza pandemic. *Health economics*, 19(10):1181–1192.

Patterson, K. and Pyle, G. (1991). The geography and mortality of the 1918 influenza pandemic. *Bulletin of the History of Medicine*, 65(1):4.

Petrén, E. (1918a). Om influensans epidemiologi och profylax. *Allmänna Svenska Läkartidningen*, 15:1630–1643.

Petrén, K. (1918b). Till influensans, särskilt influensapneumoniernas nosografi och behandling. *Allmänna Svenska Läkartidningen*, 15:1577–1600.

Rauhut, D. (2002). *Fattigvård, socialbidrag och synen på fattigdom i Sverige*. Lund Studies in Economic History 18, Lund.

Ravenholt, R. and Foege, W. (1982). 1918 influenza, encephalitis lethargica, parkinsonism. *The Lancet*, 320(8303):860–864.

Riksskatteverket (2003). *Deklarationen 100 år och andra tillbakablickar: En jubileumsbok*. Norstedts juridik, Stockholm.

Roine, J. and Waldenström, D. (2009). Top incomes in Sweden over the twentieth century. *Top Incomes: A Global Perspective*, 2.

Rose, A. (2000). One money, one market: The effect of common currencies on trade. *Economic Policy*, 15(30):7–46.

Ryberg-Welander, L. (2000). *Arbetstidsregleringens utveckling: En studie av arbetstidsreglering i fyra länder*. Sociologiska institutionen, Stockholms Universitet.

Schön, L. (2010). *Swedens's Road to Modernity: An Economic History*. SNS förlag.

Simonsen, L., Clarke, M., Schonberger, L., Arden, N., Cox, N., and Fukuda, K. (1998). Pandemic versus epidemic influenza mortality: A pattern of changing age distribution. *Journal of Infectious Diseases*, 178(1):53.

Smith, J. P. (2009). The impact of childhood health on adult labor market outcomes. *The Review of Economics and Statistics*, 91(3):478–489.

Statistics Sweden (1907). *Bidrag till Sveriges Officiella Statistik. A) Befolkningsstatistik*. Stockholm: Kungl. Statistiska Centralbyrån.

Statistics Sweden (1911). *Sveriges officiella statistik. Kommunernas Fattigvård och Finanser*. Stockholm: Kungl. Statistiska Centralbyrån.

Statistics Sweden (1915). *Dödsorsaker år 1911*. Stockholm: Kungl. Statistiska Centralbyrån.

Statistics Sweden (1917). *Folkräkningen den 31 December 1910*. Stockholm: Kungl. Statistiska Centralbyrån.

Statistics Sweden (1921). *Skattetaxeringarna samt fördelningen av inkomst och förmögenhet*, volume 1917. Stockholm: Kungl. Statistiska Centralbyrån.

Statistics Sweden (1926). *Folkräkningen den 31 December 1920*. Stockholm: Kungl. Statistiska Centralbyrån.

Statistics Sweden (1933). *Statistisk årsbok för Sverige*. Stockholm: Kungl. Statistiska Centralbyrån.

Statistics Sweden (1936). *Folkräkningen den 31 December 1930*. Stockholm: Kungl. Statistiska Centralbyrån.

Stock, J. and Watson, M. (2008). Heteroskedasticity-robust standard errors for fixed effects panel data regression. *Econometrica*, 76(1):155–174.

Tabellini, G. (2010). Culture and institutions: economic development in the regions of europe. *Journal of the European Economic Association*, 8(4):677–716.

Taubenberger, J. and Morens, D. (2006). 1918 influenza: The mother of all pandemics. *Rev Biomed*, 17:69–79.

Tumpey, T., Basler, C., Aguilar, P., Zeng, H., Solórzano, A., Swayne, D., Cox, N., Katz, J., Taubenberger, J., Palese, P., et al. (2005). Characterization of the reconstructed 1918 Spanish influenza pandemic virus. *Science*, 310(5745):77.

Wooldridge, J. (2009). *Introductory econometrics: A modern approach*. South-Western Pub.

Figure 1. Swedish Exports 1910–1930.

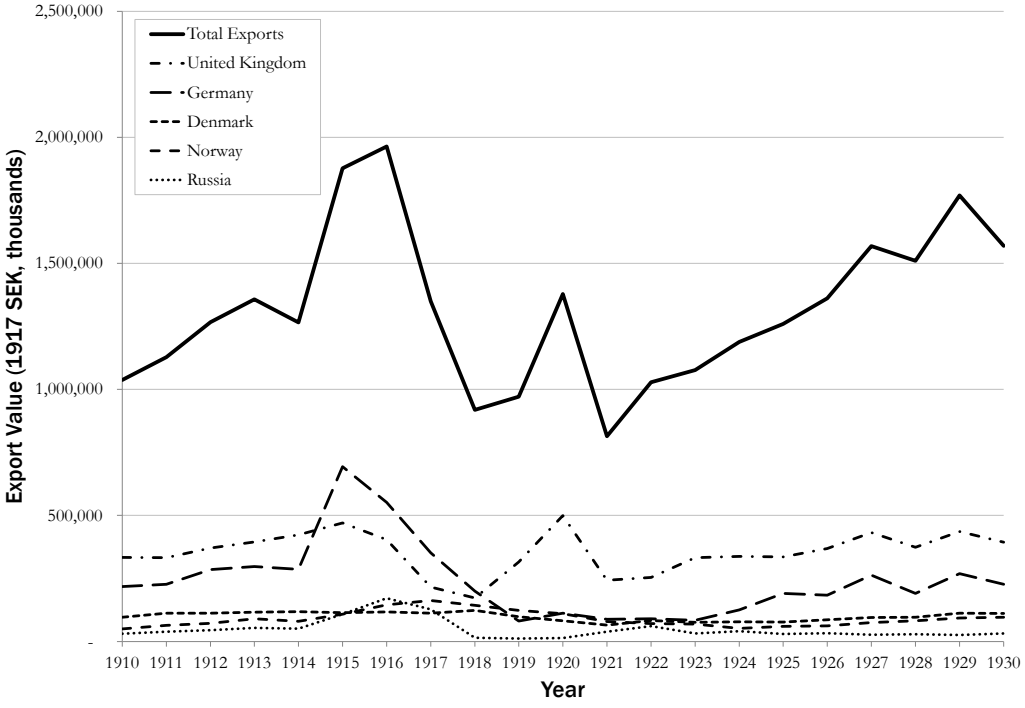

Figure 2. Monthly Influenza and Pneumonia Deaths. Sweden 1917–20.

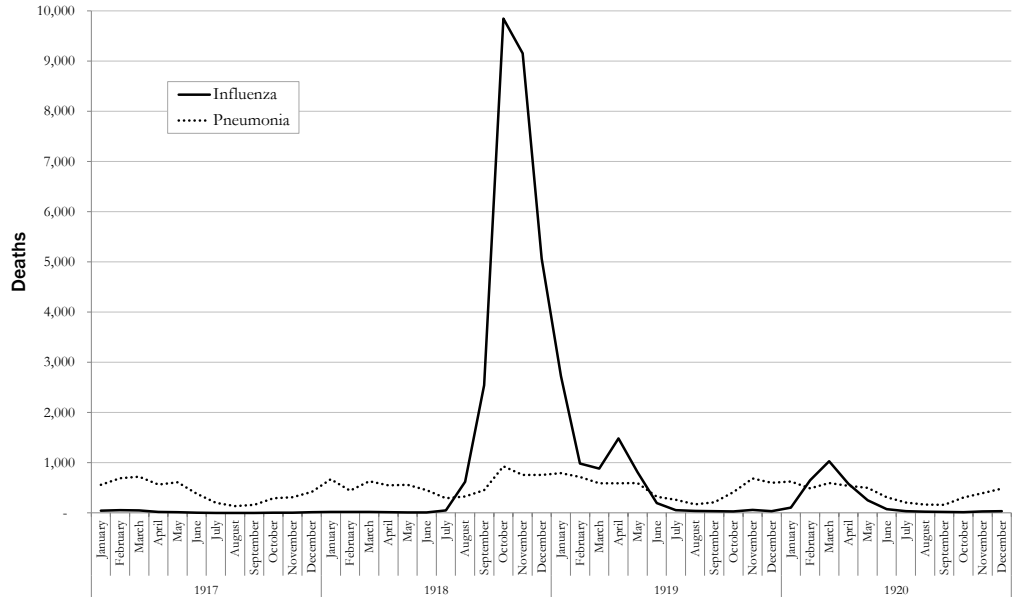

Figure 3. 1918 Influenza Mortality in Swedish Counties.

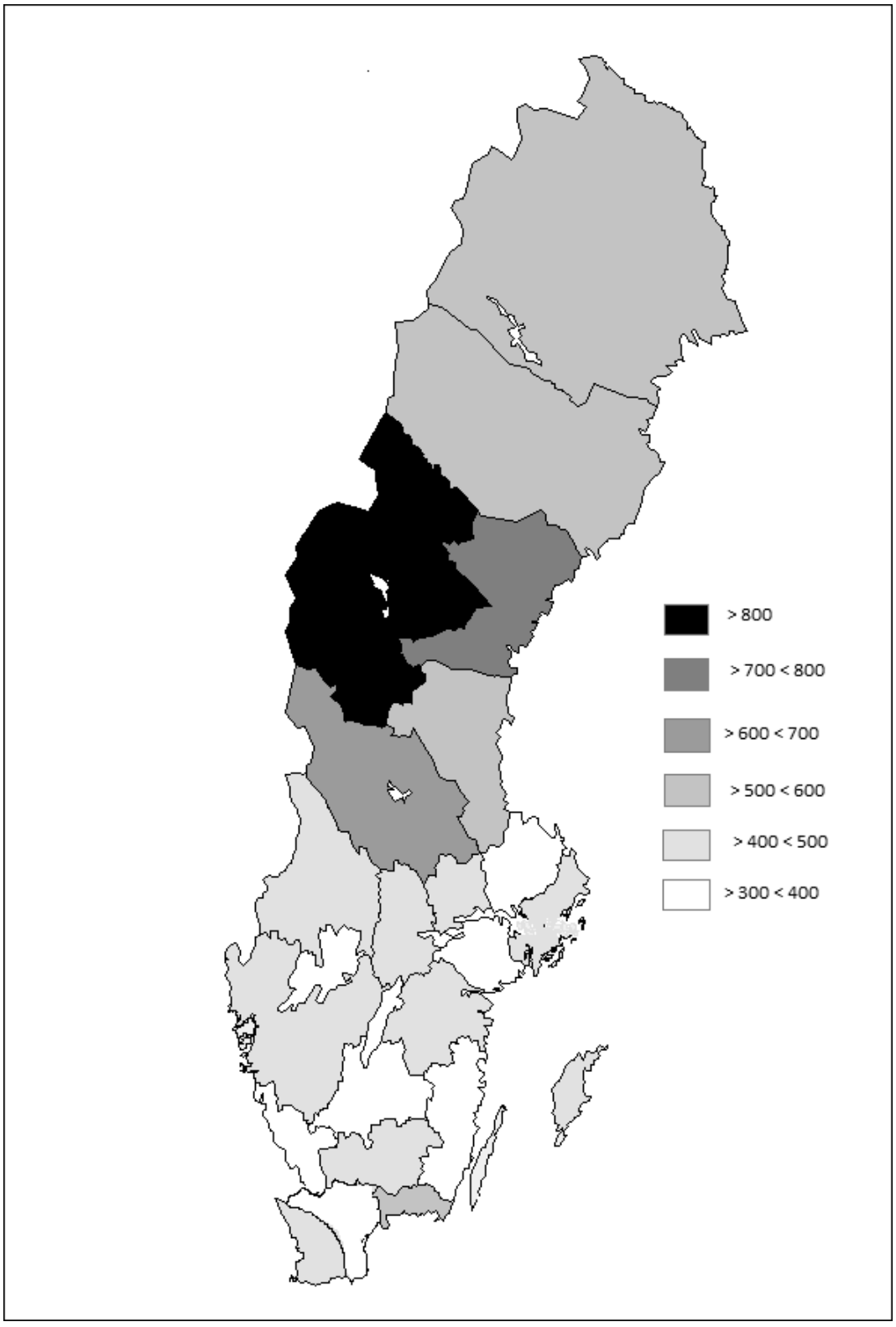

Figure 4. Age Distribution of Influenza Mortality.

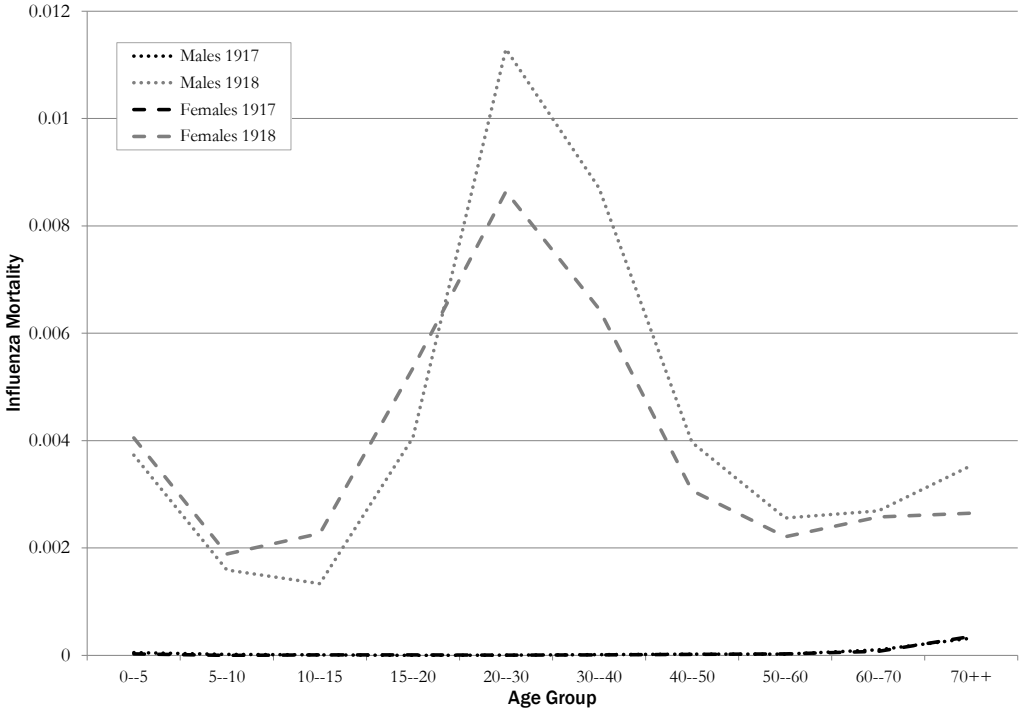

Figure 5. Incidence of Influenza in Different Locations and Socioeconomic Groups.

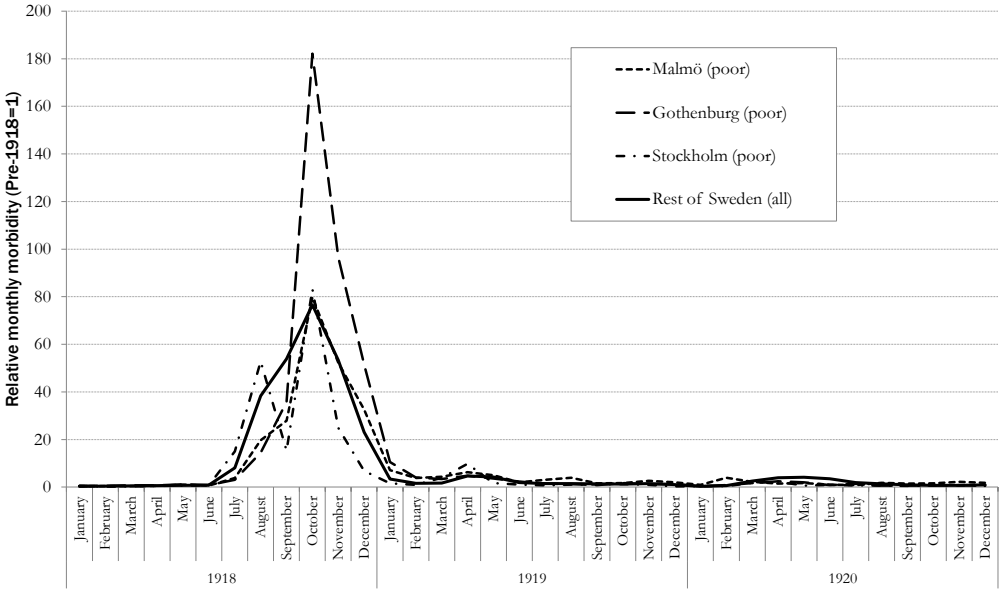

Figure 6. Excess Morbidity and Mortality at the Regional Level.

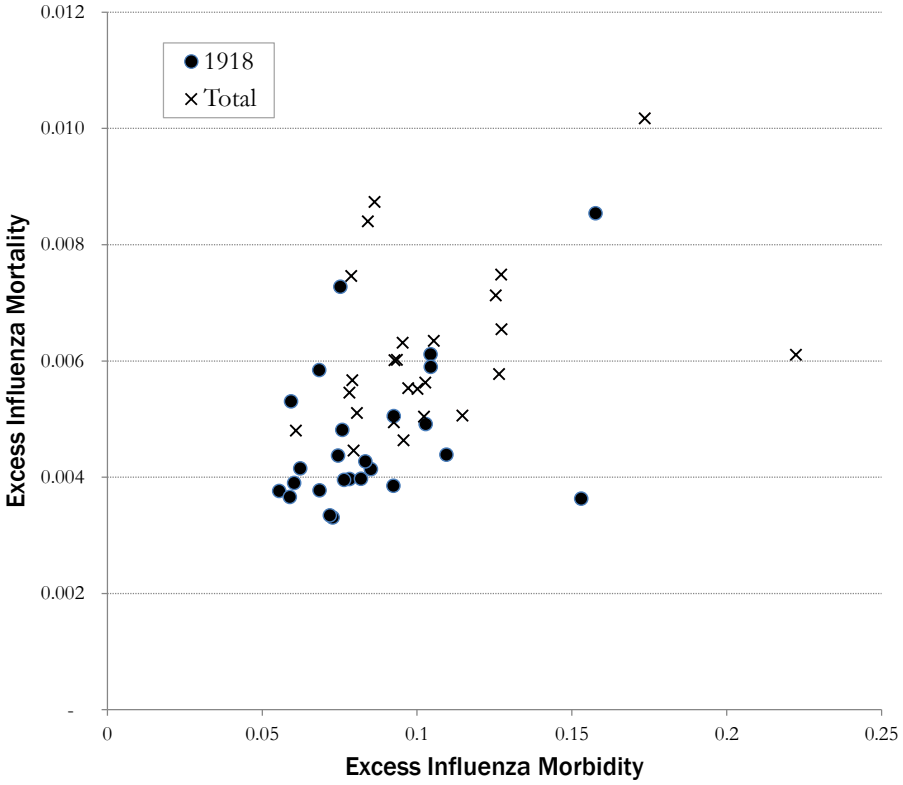

Figure 7. Common Time Trend for Log Capital Income.

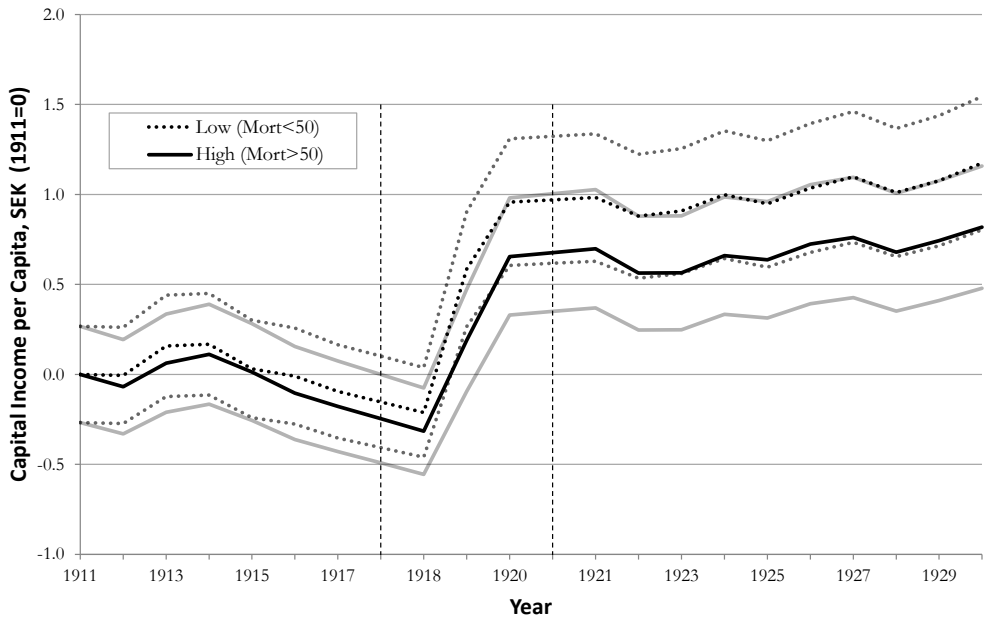

Figure 8. Common Time Trend for Log Earnings.

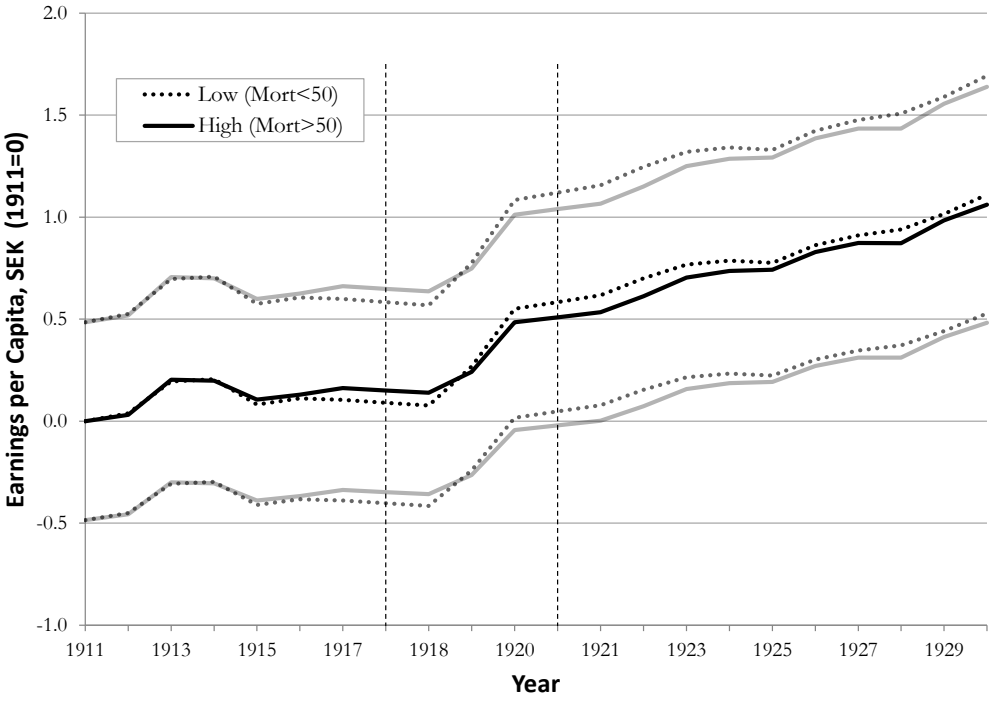

Figure 9. Common Time Trend for Log Poverty Rates.

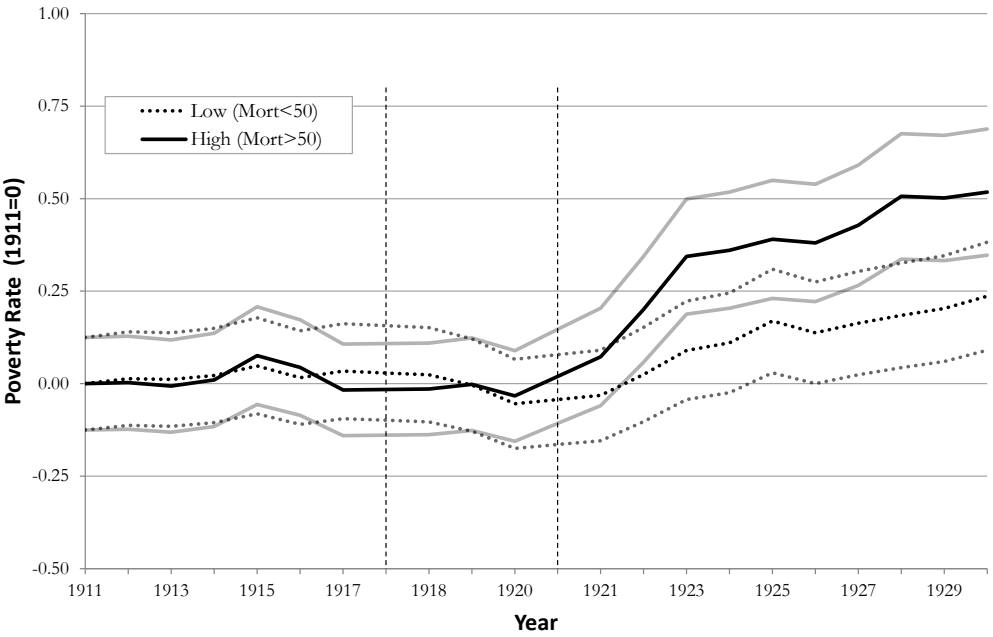

Figure 10. Common Time Trend for Log Earnings in the Non-Poor Population.

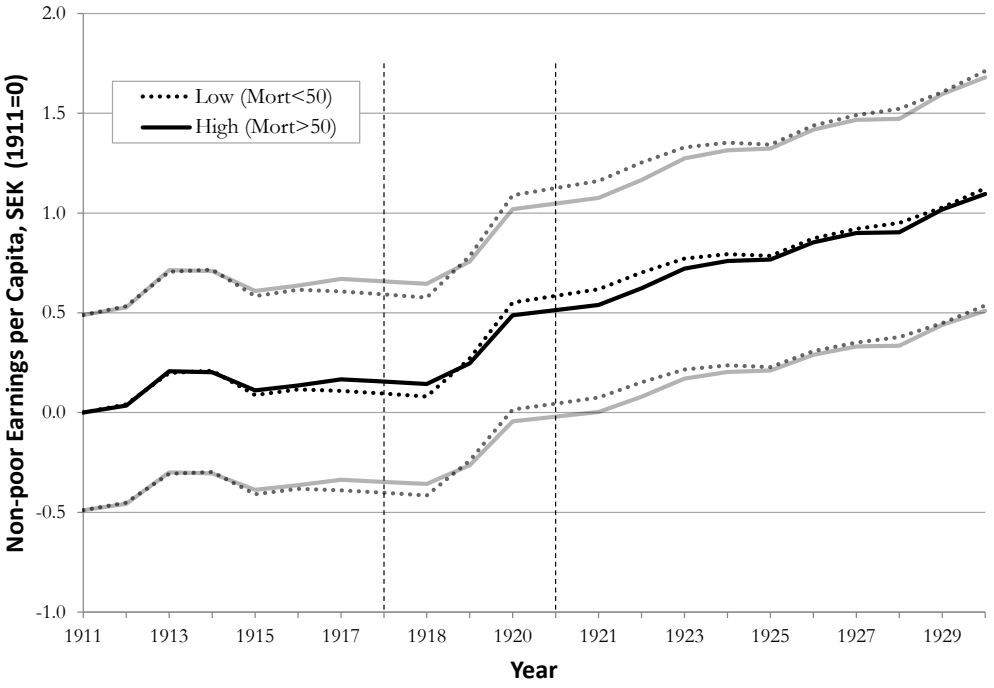

Figure 11. Summary of Results.

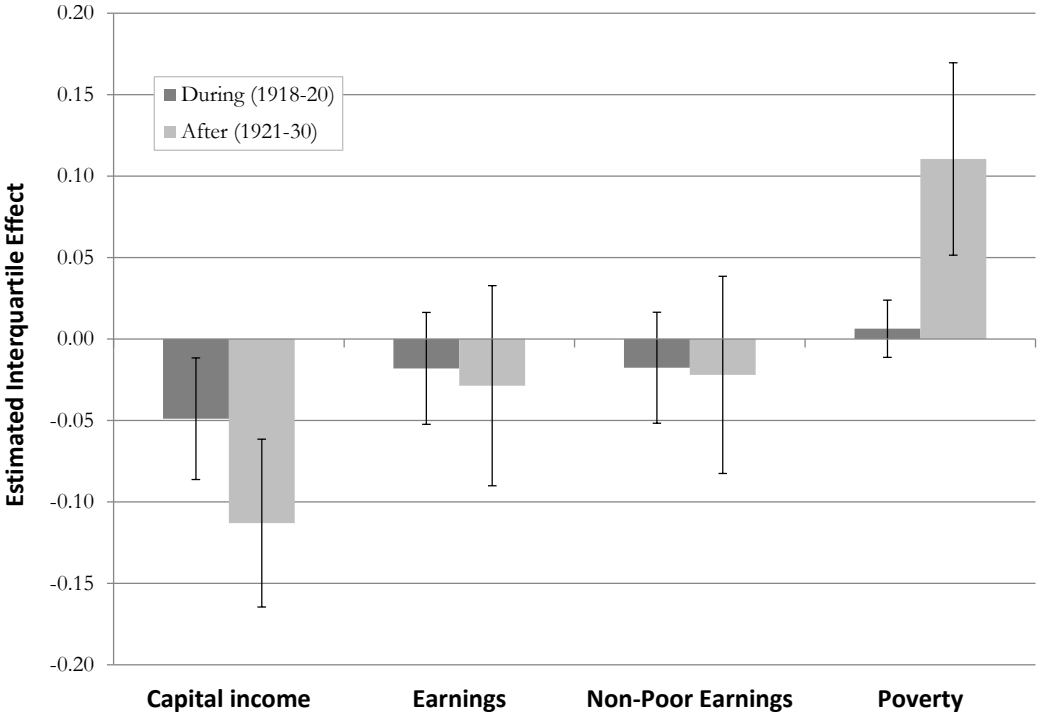

Table 1. Treatment Correlations.

Year	**1918-20**	**1910**				**1917**		
County	Excess Mortality	Population	Agriculture	Manufact.	Commerce	Population	Pop. Density	Earnings per Cap.
Kalmar	-137.7	228,129	48.1	23.8	9.7	228,998	19.8	183.9
Södermanlands	-124.7	178,568	46.4	27.2	9.4	187,891	27.6	333.1
Jönköpings	-124.6	214,454	47.0	28.3	7.3	222,607	19.3	246.7
Skaraborgs	-110.5	241,284	58.9	18.6	6.5	242,081	28.5	166.1
Uppsala	-102.7	128,171	44.9	25.7	7.7	133,506	25.1	301.2
Gotlands	-96.9	55,217	57.1	16.6	8.0	55,873	17.7	146.4
Örebro	-95.9	207,021	41.9	34.1	8.6	214,437	23.5	322.0
Kristianstads	-74.3	228,307	53.8	21.6	8.4	237,576	36.8	175.2
Kronobergs	-62.2	149,654	59.2	19.0	5.8	157,270	15.9	171.2
Östergötlands	-58.9	294,179	42.0	29.9	9.8	302,175	27.4	272.5
Älvsborgs	-56.3	287,692	53.3	25.0	6.5	297,629	23.4	201.4
Stockholm city	-45.6	342,323	0.5	38.1	24.7	413,163	3,642.4	735.2
Värmlands	-40.1	260,135	53.3	25.1	6.8	262,525	13.6	250.0
Stockholm county	-28.0	229,181	35.8	31.8	11.5	230,212	29.7	448.2
Västmanlands	-10.1	155,920	43.5	31.6	7.8	165,238	24.6	355.8
Hallands	-2.4	147,224	52.1	21.4	9.9	147,762	30.0	163.1
Blekinge	11.0	149,359	40.3	25.8	8.8	148,866	49.4	223.2
Göteborgs och Bohus	11.1	381,270	25.2	33.1	18.0	416,843	82.6	406.6
Malmöhus	35.4	457,214	30.4	34.3	14.8	481,657	99.7	372.3
Gävleborgs	82.3	253,792	38.1	32.8	11.1	263,989	13.4	328.8
Kopparbergs	113.4	233,873	48.7	31.6	6.7	248,019	8.3	322.4
Västerbottens	132.9	161,366	69.3	14.1	4.5	175,031	3.0	171.8
Västernorrlands	200.0	250,512	47.3	26.9	8.8	262,005	10.3	269.7
Norrbottens	255.6	161,132	54.1	22.4	8.8	177,285	1.7	236.4
Jämtlands	397.7	109,851	69.4	12.4	5.2	128,209	2.5	166.8
ρ Flu	1	-0.045	0.153	-0.140	-0.067	-0.041	-0.114	-0.065

The table shows the standardized excess influenza mortality 1918–20 (Section 5.1 describes how we calculate this variable); population size and sectoral shares (public and home sector omitted) according to the 1910 census (Statistics Sweden, 1917); as well population, population density (measured in inhabitants per square kilometre) and earnings per capita in 1917. In the last row we present the correlations of the various variables with excess influenza mortality, which are weighted by respective populations in 1910 and 1917.

Table 2. Summary Statistics.

Variable	N	Mean	St. Dev.	Before	During	After
Capital Income (SEK/capita)	500	38.825	41.796	22.646	42.843	49.622
Earnings (SEK/capita)	500	396.616	234.357	273.647	381.107	499.151
Poverty (%)	500	4.588	1.400	4.169	4.043	5.157
Trade Demand (SEK)	500	140.262	43.717	129.491	116.579	159.164
Population (,000)	500	235.775	97.761	227.173	235.197	242.723
Flu Incidence, Per Capita	500	0.005	0.020	0	0.026	0
Cum. Flu Incidence, Per Capita	500	0.063	0.056	0	0.079	0.105
Cum. Flu Prevalence (w_{it};%)	500	0.002	0.099	0	0.002	0.003
Population Density (Inhabitants per km^2)	500	174.885	730.179	159.980	172.428	187.570
Rural Population (%)	500	75.806	19.546	77.638	75.790	74.389
Birth Rate (‰)	500	20.073	3.921	22.617	21.542	17.440
Internal Migration (‰)	500	-0.266	8.020	-0.212	0.852	-0.805
Imigration (‰)	500	1.064	0.669	1.202	1.231	0.881
Emigration (‰)	500	2.051	1.506	2.370	1.226	2.169

The table shows descriptive statistics for the variables, and shows means of all variables before (t<1918), during (t $\geq$ 1918 & t $\leq$ 1920) and after (t>1920) the Spanish flu pandemic. Incidence (infections) have been calculated as excess rates. Moreover, prevalence (mortality) has been calculated as a deviation from predicted mortality, based on the national mortality level and the various demographic groups (see Section 5.1 for more details).

Table 3. Regression results in Logs

Panel A: Capital income

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
w_{it}	-0.824***	-0.589**	-0.529**	-0.399*	-0.536**	-0.593**	-0.653***	-0.613**		-0.612***	-0.521**
	0.187	0.223	0.211	0.228	0.253	0.219	0.199	0.222		0.194	0.224
$w_{it} \times \mathbf{1}(t > 1920)$		-0.271		-0.198	-0.279	-0.271	-0.226	-0.215			-0.223
		0.208		0.221	0.211	0.207	0.201	0.231			0.240
Placebo (w_{it+3})									-0.0208		
									0.120		
Cumulative Incidence					-0.856						
					0.650						
Incidence						-0.627					
						0.719					
Trade Demand							2.636**				
							0.962				
Constant	3.053***	3.053***	3.053***	3.053***	3.053***	3.053***	-9.750**	3.457***	3.053***	3.076***	3.076***
	0.0324	0.0324	0.0192	0.0191	0.0292	0.0322	4.664	0.559	0.0214	0.0305	0.0306
Further Controls	No	No	No	No	No	No	No	Yes	No	No	No
Regional Time Trends	No	No	Yes	Yes	No	No	No	No	No	No	No
p value for Treatment	0.000	0.001	0.019	0.058	0.002	0.001	0.000	0.001	0.864	0.004	0.009
R^2	0.958	0.958	0.972	0.973	0.959	0.958	0.960	0.962	0.686	0.954	0.955
Observations	500	500	500	500	500	500	500	500	175	125	125

Panel B: Earnings

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
w_{it}	-0.218	-0.217	-0.193	-0.197	-0.144	-0.222	-0.222	-0.276		-0.228	-0.230
	0.228	0.206	0.165	0.146	0.221	0.201	0.203	0.166		0.188	0.179
$w_{it} \times \mathbf{1}(t > 1920)$		-0.00130		0.00656	-0.0134	-0.00150	0.00175	0.144*			0.00533
		0.0758		0.0911	0.0791	0.0753	0.0719	0.0736			0.0794
Placebo (w_{it+3})									-0.0193		
									0.125		
Cumulative Incidence					-1.182***						
					0.356						
Incidence						-0.856					
						0.589					
Trade Demand							0.181				
							0.765				
Constant	5.534***	5.534***	5.534***	5.534***	5.534***	5.534***	4.653	4.695***	5.534***	5.642***	5.642***
	0.0283	0.0283	0.0105	0.0106	0.0237	0.0280	3.702	0.299	0.0174	0.0187	0.0188
Further Controls	No	No	No	No	No	No	No	Yes	No	No	No
Regional Time Trends	No	No	Yes	Yes	No	No	No	No	No	No	No
p value for Treatment	0.347	0.570	0.254	0.404	0.810	0.536	0.532	0.008	0.879	0.237	0.415
R^2	0.967	0.967	0.987	0.987	0.970	0.967	0.967	0.976	0.651	0.963	0.963
Observations	500	500	500	500	500	500	500	500	175	125	125

Panel C: Non Poor Earnings

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
w_{it}	-0.174	-0.212	-0.178	-0.208	-0.129	-0.217	-0.216	-0.270		-0.205	-0.226
	0.224	0.204	0.164	0.147	0.223	0.200	0.201	0.165		0.186	0.178
$w_{it} \times \mathbf{1}(t > 1920)$		0.0444		0.0462	0.0308	0.0443	0.0472	0.177**			0.0503
		0.0702		0.0846	0.0740	0.0699	0.0667	0.0714			0.0741
Placebo (w_{it+3})									-0.0135		
									0.123		
Cumulative Incidence					-1.337***						
					0.341						
Incidence						-0.757					
						0.547					
Trade Demand							0.161				
							0.780				
Constant	5.575***	5.575***	5.575***	5.575***	5.575***	5.575***	4.791	4.784***	5.575***	5.687***	5.687***
	0.0284	0.0284	0.00988	0.00989	0.0230	0.0282	3.776	0.293	0.0168	0.0191	0.0192
Further Controls	No	No	No	No	No	No	No	Yes	No	No	No
Regional Time Trends	No	No	Yes	Yes	No	No	No	No	No	No	No
p value for Treatment	0.446	0.412	0.288	0.299	0.718	0.374	0.336	0.003	0.914	0.282	0.282
R^2	0.968	0.968	0.988	0.988	0.972	0.969	0.968	0.978	0.665	0.964	0.964
Observations	500	500	500	500	500	500	500	500	175	125	125

Panel D: Poverty Share

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
w_{it}	0.740***	0.0759	0.359**	-0.101*	0.208	0.0833	0.0924	0.0997		0.393**	0.0889
	0.204	0.105	0.164	0.0576	0.136	0.110	0.102	0.104		0.149	0.0946
$w_{it} \times \mathbf{1}(t > 1920)$		0.765***		0.698***	0.744***	0.766***	0.754***	0.534***			0.744***
		0.200		0.216	0.196	0.201	0.202	0.151			0.214
Placebo (w_{it+3})									0.0284		
									0.0958		
Cumulative Incidence					-2.137**						
					0.933						
Incidence						1.234*					
						0.690					
Trade Demand							-0.687				
							0.649				
Constant	1.429***	1.429***	1.429***	1.429***	1.429***	1.429***	4.764	2.072***	1.429***	1.448***	1.448***
	0.0249	0.0249	0.0262	0.0253	0.0217	0.0252	3.134	0.354	0.0117	0.0196	0.0197
Further Controls	No	No	No	No	No	No	No	Yes	No	No	No
Regional Time Trends	No	No	Yes	Yes	No	No	No	No	No	No	No
p value for Treatment	0.001	0.003	0.039	0.002	0.004	0.003	0.003	0.005	0.770	0.014	0.007
R^2	0.576	0.601	0.780	0.799	0.663	0.603	0.602	0.765	0.146	0.547	0.622
Observations	500	500	500	500	500	500	500	500	175	125	125

The table shows four panels with the results from fixed effects regressions. In the first specification we regress the natural logarithm of the dependent variable, which varies with each panel, on our treatment variable w_{it}. The second column includes an additional dummy which equals one for points in time after the Spanish flu interacted with the treatment variable. Columns 3 and 4 include regional time trends. Column 5 and 6 control for (cumulative) flu infection rates. Column 7 includes trade demand as an additional control. We explain at the end of section 4 how this variable is formed. Specification 8 includes further controls, including birth rates, migration within Sweden and abroad, population density, percent of rural population and birth rates. Model 9 performs the placebo regression which is formed only for the years before 1918 by moving the treatment 3 years in the past. The final specifications 10 and 11 collapse the data in order to control for autocorrelation. All regressions include year dummies, which are not displayed. The second row presents robust standard errors. As discussed by Wooldridge (2009), Stock and Watson (2008) and Arellano (2003), robust standard errors are equivalent to regionally clustered standard errors in the case of fixed effects estimates. The stars represent significance at the following p-values: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. In the first row of the additional statistics we display the p value for the hypothesis that the treatment effect (the combined treatment effect) is equal to zero.

Table 4. Labor Supply Effect.

	Minors		Females		Males		Females Share of Total		Minors Share of Total	
w_it	0.448***	0.335**	0.423**	0.310	1.143	0.577	3.575***	3.002***	-0.183	0.853
	0.153	0.135	0.195	0.200	0.855	0.675	0.859	0.958	2.120	2.273
$w\_it \times \mathbf{1}(t > 1920)$		0.349*		0.351**		1.739**		1.777		-3.207
		0.182		0.132		0.697		1.568		2.057
p value for Treatment	0.007	0.017	0.040	0.009	0.194	0.042	0.000	0.002	0.932	0.297
R^2	0.826	0.830	0.761	0.764	0.749	0.756	0.915	0.916	0.674	0.678
Observations	450	450	450	450	450	450	450	450	450	450

The table shows the results from fixed effects regressions. In the first two models the dependent variable is the labor supply of minors (as share of total population). Models (3-4) and (5-6) measure the treatment effects on the labour supply of females and males respectively (as share of total population). Finally, in the last four models we change the denominator to the total workforce. All regressions include year dummies, regional time trends, and the relative population share of minors, females and males respectively which are not displayed. The second row presents robust standard errors. As discussed by Wooldridge (2009), Stock and Watson (2008) and Arellano (2003), robust standard errors are equivalent to regionally clustered standard errors in the case of fixed effects estimates. The stars represent significance at the following p-values: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. In the first row of the additional statistics we display the p-value for the hypothesis that the treatment effect (the combined treatment effect) is equal to zero.

Appendix

Table A-1. Regression Results. Alternative Specifications.

	Outcome Variable in Levels					Results for Super-Regions				
Panel A: Capital income	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
w_{it}	-63.96*	-26.36		-44.16	-27.29	-1.081**	-0.936		-1.003*	-0.946
	35.55	25.55		36.76	32.82	0.273	0.549		0.391	0.544
$w_{it} \times \mathbf{1}(t > 1920)$		-43.36*			-41.26*		-0.162			-0.127
		22.05			22.57		0.311			0.336
Placebo (w_{it+3})			8.616					-0.0859		
			9.866					0.214		
Constant	28.43***	28.43***	28.43***	27.90***	27.90***	3.155***	3.155***	3.155***	3.175***	3.175***
	3.551	3.554	1.501	3.575	3.589	0.0525	0.0528	0.0298	0.0496	0.0510
Further Controls	No	No	No	No	No	No	No	No	No	No
Regional Time Trends	No	No	No	No	No	No	No	No	No	No
p value for Treatment	0.085	0.144	0.391	0.241	0.196	0.011	0.000	0.705	0.050	0.001
R^2	0.622	0.625	0.268	0.604	0.608	0.983	0.983	0.835	0.979	0.979
Observations	500	500	175	125	125	120	120	42	30	30
Panel B: Earnings	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
w_{it}	-158.2	-68.34		-113.3	-70.36	-0.290	-0.266		-0.343	-0.337
	132.0	48.00		94.67	51.43	0.183	0.312		0.198	0.271
$w_{it} \times \mathbf{1}(t > 1920)$		-103.6			-104.9		-0.0265			-0.0121
		152.3			159.4		0.164			0.168
Placebo (w_{it+3})			18.16					0.0153		
			45.87					0.175		
Constant	290.0***	290.0***	290.0***	314.1***	314.1***	5.595***	5.595***	5.595***	5.697***	5.697***
	12.30	12.31	6.455	5.762	5.770	0.0405	0.0407	0.0290	0.0245	0.0251
Further Controls	No	No	No	No	No	No	No	No	No	No
Regional Time Trends	No	No	No	No	No	No	No	No	No	No
p value for Treatment	0.242	0.245	0.696	0.243	0.387	0.174	0.101	0.934	0.143	0.055
R^2	0.845	0.845	0.397	0.830	0.831	0.988	0.988	0.756	0.985	0.985
Observations	500	500	175	125	125	120	120	42	30	30
Panel C: Poverty	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
w_{it}	4.385***	0.531		2.311**	0.522	0.841***	0.117		0.522**	0.164
	1.135	0.589		0.886	0.515	0.147	0.159		0.133	0.136
$w_{it} \times \mathbf{1}(t > 1920)$		4.443***			4.375***		0.813***			0.791***
		0.754			0.801		0.0307			0.0356
Placebo (w_{it+3})			0.204					0.00739		
			0.413					0.0831		
Constant	4.336***	4.336***	4.336***	4.436***	4.436***	0.762***	0.762***	0.762***	0.789***	0.789***
	0.129	0.129	0.0524	0.122	0.123	0.0264	0.0264	0.0168	0.0216	0.0217
Further Controls	No	No	No	No	No	No	No	No	No	No
Regional Time Trends	No	No	No	No	No	No	No	No	No	No
p value for Treatment	0.001	0.000	0.626	0.015	0.000	0.002	0.000	0.933	0.011	0.000
R^2	0.521	0.552	0.161	0.490	0.591	0.811	0.839	0.298	0.785	0.874
Observations	500	500	175	125	125	120	120	42	30	30

The table shows three panels with results from fixed effects regressions. In the first specification we regress the dependent variable (which varies with each panel) on our treatment variable w_{it}. The second column additionally interacts the treatment variable with a dummy which equals one for data points after the Spanish flu. The third column presents the placebo regression which is estimated using years before 1918 and the third lead of the treatment variable ($w_{i,t+3}$). The final specifications 4 and 5 collapse the data in order to control for autocorrelation. Columns 6–10 present exactly the same specifications but for the logarithmic outcome variables. In these additional specifications we have collapsed the 25 counties into six 'super-regions' with approximately one million inhabitants each. All regressions include year dummies which are not displayed. The second row presents robust standard errors. As discussed by Wooldridge (2009), Stock and Watson (2008) and Arellano (2003), in a fixed effects model, robust standard errors are equivalent to regionally clustered standard errors. The asterisks represent significance at the following p values: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.