

A Service of

ZBW

Leibniz-Informationszentrum Wirtschaft Leibniz Information Centre for Economics

Floris, Joël; Kaiser, Laurent; Mayr, Harald; Staub, Kaspar; Woitek, Ulrich

# Working Paper Survival of the weakest? Culling evidence from the 1918 flu pandemic

Working Paper, No. 316

# Provided in Cooperation with:

Department of Economics, University of Zurich

*Suggested Citation:* Floris, Joël; Kaiser, Laurent; Mayr, Harald; Staub, Kaspar; Woitek, Ulrich (2019) : Survival of the weakest? Culling evidence from the 1918 flu pandemic, Working Paper, No. 316, University of Zurich, Department of Economics, Zurich, https://doi.org/10.5167/uzh-166014

This Version is available at: https://hdl.handle.net/10419/195156

#### Standard-Nutzungsbedingungen:

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

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

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

#### Terms of use:

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

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

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



# WWW.ECONSTOR.EU



University of Zurich

Department of Economics

Working Paper Series

ISSN 1664-7041 (print) ISSN 1664-705X (online)

Working Paper No. 316

# Survival of the weakest? Culling evidence from the 1918 flu pandemic

Joël Floris, Laurent Kaiser, Harald Mayr, Kaspar Staub and Ulrich Woitek

January 2019

# Survival of the weakest? Culling evidence from the 1918 flu pandemic<sup>\*</sup>

Joël Floris<sup>a</sup>, Laurent Kaiser<sup>b,c</sup>, Harald Mayr<sup>d,e</sup>, Kaspar Staub<sup>a,f</sup>, Ulrich Woitek<sup>d,g</sup>

<sup>a</sup>Institute of Evolutionary Medicine, University of Zurich, Winterthurerstrasse 190, 8057 Zurich, Switzerland

<sup>b</sup>Department of Medicine, Infectious Diseases Service, University Hospital of Geneva and Faculty of Medicine, University of Geneva, Rue Gabrielle-Perret-Gentil 4, 1205 Geneva, Switzerland

<sup>c</sup>Department of Genetical and Laboratory Medicine, Virology Laboratory, Laboratory Medicine, University

Hospital of Geneva, Rue Gabrielle-Perret-Gentil 4, 1205 Geneva, Switzerland

<sup>d</sup>Department of Economics, University of Zurich, Schönberggasse 1, 8001 Zurich, Switzerland

<sup>e</sup>Department of Economics, Harvard University, 1805 Cambridge Street, Cambridge, MA 02138, USA

<sup>f</sup>Institute of History, University of Bern, Länggassstrasse 49, 3012 Bern, Switzerland

<sup>f</sup>CESifo, Poschingerstr. 5, 81679 Munich, Germany

January 30, 2019

#### Abstract

When a negative shock affects a cohort in utero, two things may happen: first, the population suffers detrimental consequences in later life; and second, some will die as a consequence of the shock, either in utero or early in life. The latter effect, often referred to as culling, may induce a bias in estimates of later life outcomes. When the health shock disproportionately affects a positively selected subpopulation, the long-term effects are overestimated. The 1918 flu pandemic was plausibly more harmful to mothers of high socioeconomic status, as a suppressed immune system in mothers of low socioeconomic status may have been protective against the most severe consequences of infection. Using historical birth records from the city of Bern, Switzerland, we assess this concern empirically and document that a careful consideration of culling is paramount for the evaluation of the 1918 flu pandemic and other fetal health shocks.

JEL Classification: I10, I15, I18, N34, J24.

Keywords: Fetal origins hypothesis, 1918 flu pandemic, culling, survivorship bias.

\*Corresponding author: Harald Mayr, University of Zurich, Department of Economics, Zürichbergstrasse 14, 8032 Zurich, Switzerland; email: harald.mayr@econ.uzh.ch

## 1 Introduction

Selective mortality threatens the internal validity of many studies that estimate the effect of fetal health shocks. Nevertheless, empirical evidence on the importance and nature of culling (i.e. selective fetal mortality) is surprisingly scarce. The fetal origins literature seems to agree that culling is innocuous in the sense that it induces a bias toward zero. We challenge this assumption and argue that 'survival of the weakest' can imply a bias *away from zero*. Specifically, for the 1918 flu pandemic, it is plausible that women with a suppressed immune system were partly protected against the most severe consequences of the virus. We evaluate this possibility using birth records from the maternity hospital in Bern, Switzerland. Our results indeed suggest 'survival of the weakest'.

When a negative health shock affects a cohort in utero, two things may happen: firstly, the population suffers detrimental consequences in later life; secondly, some will die as a consequence, either in utero or early in life. The former effect is in line with the fetal origins hypothesis, famously proposed by Barker (1990). The latter is often referred to as culling. Culling can induce a selection bias in estimates of later life outcomes, also referred to as survivorship bias or survivor bias. The implications of culling bias depend on the affected sub-population's unobserved determinants of human capital. If those affected by culling have relatively bad unobserved determinants of human capital — i.e. they are relatively 'weak' in terms of potential outcomes — the estimated effect of the fetal health shock is biased towards zero. This view is expressed in many studies on fetal health shocks, e.g. in the review of Almond and Currie (2011): "[...] estimates of the effects of fetal health shocks are generally conservative when the shock also increases mortality." The assumption that those affected by culling would have had relatively bad (or at least similar) potential outcomes is crucial for the internal validity of many studies investigating the long-term impact of a fetal health shock. While this assumption is plausible in many settings, it is fundamentally untestable, often difficult to assess, and hard to justify in certain cases.

We focus our analysis on the perhaps most prominent fetal health shock in the economics literature: the 1918 flu pandemic. The work of Almond (2006) exploits the 1918 flu pandemic as an exogenous fetal health shock, a methodological improvement that sparked a lively literature on the impact of fetal health shocks on later life outcomes. Almond (2006) finds strong negative effects on later life outcomes and argues that culling potentially biases his estimates towards zero. It might seem natural to assume that those affected by culling are negatively selected, but the medical literature indicates that the nature of culling in the 1918 flu pandemic is more complex. Recent medical evidence suggests that age groups were affected differently because of childhood exposure to related flu strains (Worobey *et al.*, 2014). Moreover, "vigorous immune responses directed against the virus in healthy young persons could have caused severe disease in 1918" (Morens *et al.*, 2010). While the role of the immune system in influenza infection is highly complex and incompletely understood (Iwasaki and Peiris, 2013), it is plausible that women with a suppressed immune system were partly protected against the most severe consequences of the virus (Morens et al., 2010). If this holds true, and children of mothers with a suppressed immune system have relatively bad potential outcomes in later life, the estimates of Almond (2006) and similar studies of the 1918 flu pandemic might be inflated by 'survival of the weakest'.

It is well documented that culling was quantitatively important in the 1918 flu pandemic as "influenza [...] acted like a veritable plague, carrying off pregnant women as it did no other class of people" (Titus and Jamison, 1919). Mortality rates among infected women were documented at 27%, while pregnancy termination was observed in over 50% of infected pregnant patients (Harris, 1919; Bland, 1919). Pregnant women were affected so severely, that the medical profession discussed whether abortions should be recommended (Titus and Jamison, 1919). More recent evidence shows substantial decreases in birth rates (Bloom-Feshbach *et al.*, 2011) and increased risks of stillbirth (Nishiura, 2009) in the months after the 1918 flu pandemic.

We create a new data set from historical birth records in the maternity hospital of Bern, Switzerland, to shed light on culling during the 1918 flu pandemic. These data provide us with detailed information on all women delivering babies in the maternity hospital in Bern between 1913 and 1922. The deliveries in our data represent a subsample of the population, and exclude home deliveries and women delivering babies in other hospitals. Using stillbirth as a direct measure of culling, our empirical results demonstrate that stillbirths were substantially increased by the 1918 flu pandemic: we find a 4.4 percentage points higher stillbirth probability at peak exposure in trimester 1 and slightly smaller effects in trimesters 2 and 3.

Assessing the nature of culling is challenging because the potential later life outcomes of those affected by culling are naturally unobservable. As a second-best option, we propose an observed variable that is plausibly related to later life outcomes: marital status. In the early 20th century, single mothers in Switzerland were discriminated against at all levels—economically, socially, and legally (Kraft, 1908). Their children grew up in generally unfavorable living conditions, including a lack of breastfeeding, poor nutrition, and low education levels (Kraft, 1908). We find that culling during the 1918 flu pandemic was almost exclusively driven by married mothers. This finding provides suggestive evidence for 'survival of the weakest' in the 1918 flu pandemic.

We conclude that careful consideration of the nature of culling is paramount for the evaluation of any fetal health shock; whether a health shock leads to culling or survival of the weakest in a given context must be thoroughly evaluated. To illustrate the broader implications of our paper, we discuss two well-studied fetal health shocks: the Dutch potato famine of 1846–1847 and the French phylloxera crisis of 1863–1890. Survival of the weakest appears to be unlikely in the former case, but is potentially more concerning in the latter. We conclude that the direction of culling bias in a given context must be thoroughly assessed.

The remainder of this paper is structured as follows. Section 2 discusses culling in the context of the fetal origins literature, Section 3 describes potential culling mechanisms for

the special case of the 1918 flu pandemic, and Section 4 discusses the historical background. Section 5 explains the empirical research design, including data, descriptive statistics, and the estimation strategy. Section 6 describes the empirical results, Section 7 discusses the implications of our paper for the literature on fetal origins, and Section 8 concludes.

# 2 Culling in the fetal origins literature

The fetal origins hypothesis postulates that fetal conditions may have persistent (and possibly latent) health effects that reflect a biological mechanism (Almond and Currie, 2011). While David Barker was not the first to hypothesize latent health effects of early life conditions (e.g. Forsdahl, 1977), his name became almost synonymous with the fetal origins hypothesis (Barker, 1990). Since then, this hypothesis has received much attention in the fields of epidemiology, epigenetics, and economics. Rigorous testing of the fetal origins hypothesis, however, is a difficult empirical task.

The seminal work of Almond (2006) broke new ground in the study of fetal origins. He uses the 1918 flu pandemic as a natural experiment to test the fetal origins hypothesis and extends the discussion to non-health endpoints. Almond (2006) finds strong negative effects of exposure to the 1918 flu pandemic on socioeconomic status, education, and labor market outcomes. These findings proved highly influential and triggered a comprehensive economics literature on the effects of the 1918 flu pandemic and other fetal health shocks.

To date, the effects of fetal exposure to the 1918 flu pandemic on various outcomes were evaluated in different countries. Examples include the US (Almond and Mazumder, 2005; Brown and Thomas, 2018; Beach *et al.*, 2018; Garthwaite, 2008; Mazumder *et al.*, 2010), Brazil (Nelson, 2010), Taiwan (Lin and Liu, 2014), Switzerland (Neelsen and Straatman, 2012), and Sweden (Richter and Robling, 2016; Bengtsson and Hertz, 2015). Moreover, researchers used similar methods to study a range of other fetal health shocks. These include the Asian flu (Kelly, 2011), hunger crises (Almond *et al.*, 2010; Lindeboom *et al.*, 2010), Ramadan fasting (Almond and Mazumder, 2011; van Ewijk, 2011; Almond *et al.*, 2015; Majid, 2015), agricultural shocks (Banerjee *et al.*, 2010), stressful events (Camacho, 2008; Currie and Rossin-Slater, 2013), infections (Barreca, 2010; Schwandt, 2018), environmental pollution (Currie and Walker, 2011; Currie and Schwandt, 2017; Isen *et al.*, 2017), and various other fetal health shocks. Almond *et al.* (2017) provide a comprehensive summary of this literature.

This large and growing body of literature uses adverse shocks to test the fetal origins hypothesis. However, such a shock may not only scar survivors, but also affect whether a fetus survives to eventually end up in the data set (Preston *et al.*, 1998). There are many ways in which such a culling effect may manifest: the adverse shock may prevent conception biologically or as a parental choice, cause a miscarriage or a stillbirth, or lead to early mortality. In all these cases, survivors constitute a potentially selected sample. Whenever culling is correlated with potential outcomes,<sup>1</sup> estimated effects will be biased if the researcher cannot effectively control for selection. This can be interpreted as an omitted variable problem. As an illustration, we consider a simple linear model where the later life outcome y is regressed on exposure to the fetal health shock e, and a set of control variables X. The researcher aims to obtain a causal estimate of  $\beta$ , in order to identify the impact of the fetal health shock:

$$y = \alpha + \beta e + \gamma X + \varepsilon(c)$$

Some unobserved determinant of human capital, c, positively influences the outcome variable, i.e. Corr(y,c) > 0. In the sample of survivors, exposure to the adverse shock emight be correlated with this unobserved variable c. Clearly, the sign of bias in  $\beta$  depends on the correlation between these two variables. The literature usually assumes  $Corr(e,c) \ge 0$ , implying that survivors either have comparable or favorable unobserved determinants of human capital. In this case, the true scarring effect might be even larger and  $\hat{\beta}$  would be a conservative estimate of  $\beta$ .

Assuming survivors to be positively selected might be plausible and convenient, but it might not be appropriate in certain settings. More specifically, this assumption can be violated in two ways—even if the health shock occurs unexpectedly. First, exposure to the adverse shock might *coincide with another event* that affects subpopulations of parents differently. Second, *the adverse health shock itself* might induce culling among a positively selected subpopulation.

Brown and Thomas (2018) provide an example of the first threat during the 1918 flu pandemic. Their results show that the US draft policy deterred parents of high socioeconomic status from conceiving during the peak of the pandemic. Brown and Thomas (2018) find that the effects of the flu pandemic become smaller and statistically insignificant once socioeconomic variables are controlled for.<sup>2</sup>

In contrast to Brown and Thomas (2018), we argue that a fetal health shock *itself* can induce culling among a positively selected subpopulation. This concern is difficult to address with improved identification strategies. In particular, positively selected survivors would inflate the estimated effects of a fetal health shock even if exposure was experimentally assigned—'survival of the weakest' would imply differential attrition in this hypothetical experiment.

In the next section, we turn to the case of the 1918 flu pandemic.

<sup>&</sup>lt;sup>1</sup>We refer to potential outcomes as the outcome that would have been observed in the absence of the adverse shock. Depending on the context, subjects affected by culling may be positively or negatively selected.

<sup>&</sup>lt;sup>2</sup>Beach *et al.* (2018) use World War II enlistment records to exploit both time and spatial variation in influenza exposure, as well as household fixed effects. Their results are consistent with the original findings of Almond (2006).

# 3 Case of the 1918 flu pandemic

The 1918 flu pandemic was the most devastating of all known pandemics.<sup>3</sup> The virus infected approximately one-third of the world's population in three waves in 1918 and 1919, with total deaths being estimated between 50 and 100 million (Taubenberger and Morens, 2006). The pandemic was caused by a virulent influenza A virus of subtype H1N1. Infected persons frequently developed pneumonia and related pulmonary infections. It was hypothesized that the influenza infection itself did not directly cause mortality in the majority of cases, but rather led to highly lethal secondary bacterial pneumonia (Brundage and Shanks, 2008). Morens *et al.* (2008) examined lung tissue from 1918 influenza victims and find strong evidence for this hypothesis. They conclude that the majority of deaths during the 1918 flu pandemic were likely due to bacterial pneumonia, which likely affected many pregnant women (over 50% in the sample of Harris, 1919, suffered from pneumonia).

How socioeconomic status mediated the effect of the 1918 flu pandemic is subject to ongoing debate. The view that the 1918 flu pandemic was 'socially neutral' was investigated by e.g. Mamelund (2006), who finds no significant effects of social class or marital status on influenza mortality in 1918. Living in a small apartment and a poor parish, however, increases the mortality hazard in his Norwegian data (Mamelund, 2006). More recent evidence suggests that the first wave of the pandemic disproportionately hit the poor, while the second wave hit the rich more strongly (Mamelund, 2018). Grantz *et al.* (2018) find particularly high mortality rates among underprivileged households in Chicago. Bengtsson *et al.* (2018) use Swedish data and find particularly high excess mortality among low-skilled manual workers. They see no perfect social class gradient, and class differences during the 1918 flu pandemic were less pronounced among women (Bengtsson *et al.*, 2018). We are not aware of evidence regarding the impact of socioeconomic status on the effect of the 1918 flu pandemic specifically among pregnant mothers.

Culling in the context of the 1918 flu pandemic most probably occurred in various forms: maternal mortality, miscarriages, stillbirths, and abortions. In the wake of the pandemic, mortality rates were documented at 27% among infected pregnant women (Harris, 1919) and 51% among infected pregnant women with suspected pneumonia (Woolston and Conley, 1918). In terms of miscarriages and stillbirths, Harris (1919) and Bland (1919) documented pregnancy termination in more than 50% of infected pregnant patients. Given that pregnancy was such an important risk factor for influenza mortality, the scientific community discussed whether pregnant women should be advised to have an abortion (Titus and Jamison, 1919). More recently, Bloom-Feshbach *et al.* (2011) show that birth rates in the US, Copenhagen, Norway, and Sweden declined substantially in the aftermath of the 1918 flu pandemic. Their results suggest that roughly 1 in 10 pregnant women were affected,

 $<sup>^{3}</sup>$ See the overviews in Taubenberger and Morens (2006) and Barry (2009).

mainly by miscarriages in trimester 1.<sup>4</sup> Considering data from Japan, Nishiura (2009) find a 10% to 30% increased risk of stillbirths because of the 1918 flu pandemic. Dahal *et al.* (2018) find substantially increased stillbirth risk during the 1918 flu pandemic in Arizona.<sup>5</sup> Reid (2005, p. 34) summarizes the literature on the effects of the 1918 flu pandemic on pregnant women: "The 'Spanish flu' [...] hit pregnant women hard, with the consequent effects of increased stillbirths [...]"

In contrast to other flu pandemics, the mortality pattern of the 1918 flu pandemic was highly unusual. While influenza mortality is normally concentrated among the very young and the very old, many young and otherwise healthy individuals were affected in 1918 (Oxford and Gill, 2018; Taubenberger and Morens, 2006).

Worobey *et al.* (2014) study the origins of the 1918 virus and argue that certain cohorts were protected by childhood exposure to genetically similar influenza viruses.<sup>6</sup> The very old were likely exposed to a H1N1 virus in their childhood, offering them a high degree of protection to the 1918 H1N1 virus. The next-oldest cohort was likely primed by a virus of type H1N8, matching only one antigen of the 1918 virus. The cohorts around 29 years old in 1918 were likely exposed to a H3N8 virus in childhood. Mismatching the 1918 virus on both antigens, they would have had little protection in 1918. Those even younger experienced another H1N8 virus, offering intermediate protection in 1918, and the very young were likely unexposed to influenza at all. Worobey *et al.* (2014) show that this explanation matches the actual data well, including the high mortality rates among individuals of prime age.

Morens *et al.* (2010) provide a competing explanation for the excess mortality among young and healthy individuals: excessive immune response. Cytokines play an important role in the human body's innate immune response to influenza A infection.<sup>7</sup> While these proteins are crucial for the human body's protection against influenza A, they can cause serious harm if the immune reaction is unregulated (Fukuyama and Kawaoka, 2011). Socalled cytokine storms<sup>8</sup> are particularly observed in H5N1 and virulent influenza strains, and a cytokine storm might have had deleterious effects on the young and healthy who were so strongly affected during the 1918 flu pandemic (Taubenberger and Morens, 2010). Mounting evidence shows that the 1918 virus triggered a vigorous and pathogenic immune

<sup>&</sup>lt;sup>4</sup>Mamelund (2012) discusses how behavioral changes induced by World War I, spousal separations, and bereavement relate to these results. Bloom-Feshbach *et al.* (2012) argue that these explanations are unlikely to explain the temporal pattern in their data.

<sup>&</sup>lt;sup>5</sup>Recent evidence suggests that the 1918 flu pandemic decreased conception rates and increased miscarriages (Chandra and Yu, 2015; Chandra and Lu, 2015). We focus on stillbirth effects, because we can directly observe this outcome in our data.

<sup>&</sup>lt;sup>6</sup>Influenza protection by childhood exposure to related influenza strains in childhood is also discussed in van Wijhe *et al.* (2018), Gagnon *et al.* (2018), Gagnon *et al.* (2015), and Gagnon *et al.* (2013). See Baumgarth *et al.* (2013) for a comprehensive summary of antibody-mediated immunity in the context of influenza infections.

<sup>&</sup>lt;sup>7</sup>Hayden *et al.* (1998) experimentally infected human subjects with an influenza A virus of subtype H1N1 to investigate their cytokine response. See Iwasaki and Peiris (2013) for a comprehensive summary on innate immunity in the context of influenza infections.

<sup>&</sup>lt;sup>8</sup>A review of cytokine storms is provided by Tisoncik *et al.* (2012).

response.<sup>9</sup> Given these findings, it is plausible that those with the most vigorous immune system suffered the most. Women with a suppressed immune system, however, might have been protected from culling, potentially inflating the results in the literature by 'survival of the weakest'. Put differently, a suppressed immune system during a pregnancy characterized by stress, malnutrition, or other environmental factors might have been lifesaving in 1918.<sup>10</sup>

# 4 Historical background

#### 4.1 Bern during World War I

Despite neutrality, Switzerland suffered from the outbreak of World War I (WWI) because of its dependency on imports of grain and raw materials. The specialization of the agricultural sector in producing mainly milk and meat products was seen as a problem at an early stage (e.g. Jöhr, 1912).

In addition to the deteriorating trade conditions, military mobilization in August 1914 withdrew approximately 200,000 men from the labor market because military service was compulsory (one-third of all men between 20 and 48 years of age were liable for military service) (Senn, 2013). Throughout the war, the number of soldiers on duty varied between 26,742 (November 1918) and 103,226 (April 1917) (General Ulrich Wille, 1919). Without compensation for income losses, one-third of soldiers' families had to rely on a relief measure ("*Militärnotunterstützung*", Degen, 2013). The peak of the intervention was 1915, with an amount of 543,492 Fr. distributed to 1,875 families, or 290 Fr. per family (Figure A.1). To put this into perspective: the annual income of an unskilled worker's family in 1919 was 5,326 Fr. (Eidgenössisches Arbeitsamt, 1923, Table 8, p. 6).

The general effect on the labor market was not catastrophic. At the beginning of the war, the labor bureau of the city pointed out that it was similar to other difficult years (Stadt Bern, 1915, p. 157), and that the main issue was coordination (Stadt Bern, 1916, p. 148). For male workers and employees, the ratio of positions to job seekers fluctuated around 1 during the time of the war, but it increased from January 1918 onward. Notwithstanding this sign of improved labor market conditions, food and supply conditions remained poor until summer, and social tensions increased. Female job seekers always outnumbered the available open positions: the ratio of open positions to job seekers increased from about 0.5

<sup>&</sup>lt;sup>9</sup>Liu *et al.* (2016) review the literature on cytokine storms in severe influenza infection and discuss potential therapeutic strategies. Oldstone and Rosen (2014) discuss the role of cytokine storms in influenzarelated morbidity and mortality. Kash *et al.* (2006) experimentally infected mice with the reconstructed 1918 virus. The infected mice showed enhanced inflammatory and cell-death immune responses associated with severe pulmonary pathology.

<sup>&</sup>lt;sup>10</sup>A similar pattern was observed in HIV patients who suffered severe inflammatory responses to virus infections after their immune function was restored (Mayer *et al.*, 2004). It must be noted that pathogenicity of a virus infection is determined in complex ways by both virus and host factors (Fukuyama and Kawaoka, 2011). Moreover, if a suppressed immune system is protective against cytokine storms, it might still be detrimental in other ways, e.g. through increased susceptibility to bacterial infection.

at the beginning of the war to a peak of close to 2 at the beginning of 1920. After that, it began to fall again, even below the prewar level (Figure 1).



Source: Stadt Bern (1914, p. 137, 138; 1915, p. 160, 161, 1916, p. 152, 153; 1917, p. 198, 199; 1918, p. 184, 185; 1919, p. 167, 168; 1920, p. 171, 172; 1921, p. 146, 147; 1922, p. 158, 159; 1923, p. 144, 145).

**Figure 1** – Labor market, 1910–1922

From March 1917 onward, the City of Bern implemented food rationing, starting with sugar and rice. Bread followed in October, and milk was rationed from January 1918 to March 1920, when rationing stopped (Figure A.2). To support families in need, milk and bread where distributed at reduced prices. In 1917, 26,883 persons were eligible for this measure, which was 26% of Bern's population (Stadt Bern, 1918, p. 100). This number decreased to 10,057 in 1920 (10% of the population, Stadt Bern 1921, p. 92).

#### 4.2 The 1918 flu pandemic in Switzerland

The 1918/19 influenza pandemic is estimated to have caused 50–100 million deaths worldwide (Johnson and Mueller, 2002).<sup>11</sup> The term 'Spanish Flu' is misleading because the pandemic did not originate in Spain or hit Spain especially hard: because Spain was a neutral country in World War I, its press remained largely free of censorship. Hence, detailed information on the Spanish outbreak was available.

The first recorded outbreak took place on March 5, 1918, in a US Army training camp in Kansas.<sup>12</sup> With the US troops, the influenza arrived in Europe in May and June 1918,

<sup>&</sup>lt;sup>11</sup>For historical overviews, see e.g. Kilbourne (1987) and Barry (2009).

 $<sup>^{12}</sup>$ The exact geographical origin of the disease is subject to competing theories (e.g. Oxford *et al.*, 1999; Shortridge, 1999).

affecting first the allied forces and then the armies of the central powers, before it hit the civilian population. This initial wave was followed by a second outbreak in fall 1918 and a third wave in early 1919.



Source: Stadt Bern (1919, p. 104-106); Schweizerisches Gesundheitsamt (1919), p. 5,
p. 13, p. 20, p. 28, p. 36, p. 46, p. 52, p. 61, p. 68, p. 84, p. 101, p. 108, p. 116, p. 132,
p. 140, p. 157, p. 172, p. 180, p. 196, p. 205, p. 220, p. 228, p. 236, p. 244, p. 252,
p. 269.

Figure 2 – Weekly influenza cases in Bern, July 1918 to June 1919

In Switzerland, about two million people were affected. The death toll between July 1918 and June 1919 was 24,449 (0.62% of the total population in 1918, Sonderegger 2007). In Bern, 19,429 infections were reported to the city in 1918, the highest number since the influenza pandemic of 1889/90 (Stadt Bern, 1919, p. 104). Based on a ruling from the Federal Council (July 18, 1918), the city banned public events and closed schools. The organization of the health service turned out to be a challenge because of the sheer number of patients. Doctors being away for military service compounded the problem (Stadt Bern, 1919, p. 103).

#### 4.3 Maternity hospital

The maternity hospital of Bern, (*Frauenspital*, until 1892: *Kantonale Entbindungs- und Frauenkrankenanstalt*) was founded in 1874/76. Besides providing medical care especially for women in need,<sup>13</sup> it also served as a training hospital for the university and as a midwifery

<sup>&</sup>lt;sup>13</sup>The fact that 27% of the mothers in our data set are not married, compared with 8.6% of mothers giving birth in the city of Bern in the same period (Eidg. statistisches Bureau, 1924), shows that the maternity hospital of Bern attracted a specific socioeconomic group.

school (Guggisberg 1931, p. 6–8, Dübi and Berger 1976, p. 12–19). The increasing demand for the services of the hospital led to unacceptable conditions, triggering an extension of the existing building in 1919. The total number of births in the city of Bern is displayed in Figure 3, together with the births at the maternity hospital and the home births assisted by maternity hospital staff.



Hospital births: maternity hospital; Home births: births assisted by maternity hospital; Total births: city of Bern (including still-births); Source: Dübi and Berger 1976, p. 73–74

Figure 3 – Births in the city of Bern, 1910–1922



Source: Report of the Cantonal Administration (1910), Annual Reports of the Hospital (1911–1922)

Figure 4 – Patients at the maternity hospital, 1910–1922: Origin

## 5 Research design

In this section, we describe the data, provide descriptive statistics, and explain our estimation strategy.

#### 5.1 Data

The data were transcribed from the original birth records of the maternity hospital in Bern. These records include the following individual information for all deliveries that took place at the hospital: admission date, birth date, infant sex, singleton/multiple birth, mother's age, parity, marital status, date of last menstruation (used to calculate influenza exposure), and stillbirth/live-birth. For the purpose of this study, we use deliveries admitted in the years 1913 to 1922. This yields a total of 7,769 deliveries.

Exposure to the influenza pandemic is measured by the weekly reported numbers of new influenza cases in the city of Bern (see Figure 2). The existing legal obligation for medics to report infectious diseases to the city authorities was extended to influenza no later than July 16, 1918, in the second week of the pandemic (Simonin *et al.*, 1918). It is known that such morbidity data might have been subject to reporting bias (unreported cases and/or misdiagnosis) (Tscherrig, 2016), but we do not have evidence that underreporting was stronger in the first wave than in second wave, as has been shown for Bergen, Norway (Mamelund *et al.*, 2016).

Daily values are interpolated from this source. Starting from the day of last menstruation, exposure variables for each trimester are calculated as the sum of all infections during the respective trimesters' time period.<sup>14</sup> Finally, these three measures are normalized such that the highest possible exposure is 1. The resulting three variables are lagged moving averages of the original exposure variable that are normalized to 1, as illustrated in Figure 5. Current exposure is depicted in solid blue and measured on the left axis, with a maximum value of 2,201 weekly infections in the city of Bern. The two influenza waves in spring and autumn 1918 fade away only in late spring 1919. The trimester exposure variables are smoothed and lagged versions of current exposure, depicted as dashed red (third trimester), dash-dotted green (second trimester), and dotted orange (first trimester) and measured on the right axis.

#### 5.2 Descriptive statistics

Simple summary statistics in Table 1 show that girls are slightly underrepresented in our data: 3% are multiple deliveries, the average mother gives birth to her third child at 28 years, and 73% of our sample are married, while 6% of the sample were stillborn.

 $<sup>^{14}</sup>$  The respective time periods are: weeks 0 to 13 for trimester 1, weeks 14 to 26 for trimester 2, and weeks 27 to 40 for trimester 3.



Figure 5 – Exposure variables

	Mean	SD	Min	Max
Girl	0.482	0.500	0.000	1.000
Multiples	0.031	0.172	0.000	1.000
Age	27.731	6.387	13.000	50.000
Parity	2.817	2.611	1.000	20.000
Married	0.734	0.442	0.000	1.000
Stillbirth	0.063	0.244	0.000	1.000
Weeks 0-13	0.045	0.160	0.000	1.000
Weeks $14-26$	0.045	0.164	0.000	1.000
Weeks $27-40$	0.043	0.156	0.000	1.000
Observations	7769			

Table 1 – Summary statistics

Figure 6 reveals that stillbirth incidence (depicted in solid blue) increased sharply in 1919, immediately after influenza infections (depicted in dashed red) peaked in 1918. In particular, the stillbirth rate increased from around 6% to almost 9% in 1919.

Next, we split the sample by marital status. Figure 7 shows that the increase in stillbirths following the influenza pandemic is particularly strong for married mothers. Single mothers—in Figure 8—do not exhibit a similar pattern.

Figures 7 and 8 are also informative about the selection of married and single mothers into the maternity hospital in Bern. The official records of the city of Bern report stillbirth rates of 3.01% among married women and 5.26% among single women (Eidg. statistisches Bureau, 1924). Comparing the number for married mothers to Figure 7 reveals that married mothers in our sample have an unusually high stillbirth rate, i.e. they are negatively selected in terms of stillbirth risk. The number for single mothers in relation to Figure 8 suggests that, in terms of stillbirth risk, single mothers in our sample are quite representative of single mothers in the city of Bern.



Figure 6 – Stillbirth



 ${\bf Figure}~{\bf 7}-{\rm Stillbirth:}~{\rm Married}$ 



Figure 8 – Stillbirth: Single

Figure 7 and Figure 8 indicate that the 1918 flu pandemic affected married mothers more strongly than single mothers. This finding is consistent with Morens *et al.* (2010), but it could be driven by changes in our sample. To investigate whether the share of married mothers in our sample changes systematically with the 1918 flu pandemic, we plot this variable over time in Figure 9. Reassuringly, while the share of married mothers seems to increase from 1913 to 1916, we see no trend during the 1918 flu pandemic. Next, we investigate the impact of the 1918 flu pandemic in a multivariate regression framework and describe our empirical identification more formally.



Figure 9 – Share of married mothers over time

#### 5.3 Estimation strategy

We estimate logistic regression models of the following form and present marginal effects in Section 6.

$$stillbirth = \alpha + \beta e^{trim} + \gamma_1 qirl + \gamma_2 multiples + \delta^{age} + \delta^{parity} + \delta^{month} + \delta^{neighborhood} + \epsilon$$

Our outcome of interest is *stillbirth*, a dummy variable equal to 1 for stillbirths and 0 for live births. Depending on the model,  $e^{trim}$  stands for one exposure variable out of  $e^{first}$ ,  $e^{second}$ ,  $e^{third}$ , or the full set of these three variables. The variable girl indicates the infant's sex and *multiples* indicates multiple pregnancies. Finally,  $\delta^{age}$ ,  $\delta^{parity}$ ,  $\delta^{neighborhood}$ , and  $\delta^{month}$  are fixed effects for age categories, parity categories,<sup>15</sup> neighborhood,<sup>16</sup> and month of the year.

Our coefficient of interest is  $\beta$ . The necessary assumption for causal inference in our setting is that no unobserved variables correlate both with our outcome variable *stillbirth* 

 $<sup>^{15}\</sup>mathrm{Age}$  categories are split at ages 20, 25, and 30, parity categories at ages 2, 4, and 6.

 $<sup>^{16}{\</sup>rm There}$  are no recorded still births in two small neighborhoods, reducing our effective sample size from 7,769 to 7,711 observations.

and exposure to the 1918 flu pandemic. This assumption could be violated if the population of mothers delivered in the maternity hospital in Bern or other important determinants of birth outcomes (e.g. nutrition) change systematically with exposure to the 1918 flu pandemic. As discussed in Section 4, there are no strong concerns with regard to the local labor market conditions (Figure 1) and the number of births (Figure 3) in the city of Bern. Furthermore, the origin (Figure 4) and marital status (Figure 9) of the patients as well as the food supply (Figure A.4) at the maternity hospital seem innocuous. Because food was rationed at the time when the 1918 flu pandemic was at its peak, we control for this using a *rationing* indicator in a robustness check. In an additional robustness check, we restrict our sample to conceptions before the 1918 flu pandemic. This robustness check allows us to investigate whether the longer run effects of the 1918 flu pandemic or selective conception impact our main estimates.

While these robustness checks are reassuring, our main analysis does not rely on strictly causal estimates. To assess the nature of culling, we are ultimately interested in the heterogeneous effects of the 1918 flu pandemic, i.e. how different subgroups were affected differently. To do so, we perform sample splits with regard to mothers' birth cohort and marital status. We then run our initial analysis on the subsamples that may see differential effects according to the hypotheses of Worobey *et al.* (2014) and Morens *et al.* (2010). If the hypothesis of Worobey *et al.* (2014) applies to stillbirths, we expect a larger effect among mothers in birth cohorts 1885 to 1893 who were around 28 years of age in 1918. However, if the hypothesis of Morens *et al.* (2010) applies to stillbirths, we expect a larger impact among married mothers who may be less likely to be immune suppressed.

#### Marital status as a proxy for long-term child outcomes

We would ideally observe the potential long-term outcomes of the fetuses in our sample directly. As this variable is naturally unobservable (we will never know what would have happened to those affected by culling), we use marital status as a second best option. Marital status plausibly predicts future outcomes. In the city of Zurich for example, the majority of single mothers were maidservants by profession and the majority of single mothers came from the lower class (Joris and Witzig, 2001, p. 313). Between 1911 and 1923, almost half of all mothers of illegitimate live births in the city of Bern were employed as service personnel (hotel and private) and about a third were factory workers or industrial workers (Lauener, 1926, p. 84). Social reform circles in the early 20th century strongly denounced that single mothers and their children were discriminated against at all levels—economically, socially, and legally (Kraft, 1908).

In order to presumably escape the social stigma, many pregnant single women moved to the anonymity of the city (Schreiber, 1993, p. 126–150). Because these women could not give birth at home, it is not surprising that they were increasingly delivering in cantonal maternal clinics by the end of the 19th century (Schreiber, 1993; Lauener, 1926, p. 126–150, p. 85). Since the turn of that century, more and more young women had been looking for employment in the cities, but their employment opportunities were limited and most of them had unattractive, insecure, and underpaid jobs (Alt and Sutter, 1985, p. 123). Most women with an illegitimate child were dependent on their own employment (Schreiber, 1993, p. 126–150). They often continued to work until childbirth, resumed their jobs shortly afterwards, and had to give their children to external childcare at a very early age (Schreiber, 1993, p. 126–150).

Early 20th century social reform circles also indicated that single motherhood was strongly correlated with poor child health (Kraft, 1908). Particular reference was made to the higher stillbirth rate, higher infant mortality rate, lower physical fitness, and poorer occupational education (Kraft, 1908). Between 1913 and 1922, the proportion of stillbirths in live births in the city of Bern was on average five times higher among illegitimate children than among legitimate children (Statistische Bureau des schweiz. Finanzdepartementes, 1916-1924). Moreover, the mortality rate of illegitimate infants in the first year of life in the city of Bern was almost twice as high as that of legitimate children (Lauener, 1926, p. 85). The causes cited were harmful influences during pregnancy, lack of breastfeeding, and generally unfavorable nutritional, living, and educational conditions (Kraft, 1908; Lauener, 1926).

In summary, the contemporary and historical literature suggests that marital status was a strong predictor for later life outcomes of a mother's child.

# 6 Results

This section presents our empirical results. Subsection 6.1 shows the effect of the 1918 flu pandemic on stillbirths. In Subsection 6.2, we examine whether the effects on stillbirths vary by mother's cohort and marital status. Subsection 6.3 shows the results from our robustness checks.<sup>17</sup>

#### 6.1 Culling evidence from stillbirths

Table 2 presents estimates from three different models that include one of the three trimester exposure variables (columns 1 to 3), and the model with all three exposure variables (column 4). We find substantial effects on the probability of a stillbirth. Mothers at peak exposure in trimester 1 carry a 4.4 percentage points higher risk of stillbirth, as compared with unaffected mothers. The effects are comparable for exposure during the second trimester (4.1 percentage points), while the effect is somewhat smaller in the third trimester (3.4 percentage points).

 $<sup>^{17}</sup>$  One might speculate whether exposure to the 1918 flu pandemic changed the sex ratio in our sample. We find no such effect. Results are available upon request.

The effect sizes are large and statistically significant at the 99, 99, and 95 % levels, respectively. The mean stillbirth probability in our sample is 6.3%. An increase of 4.4 percentage points corresponds to a 70% increase in stillbirth probability.

In the model with all trimester exposures, only a trimester 1 exposure is individually statistically significant; however, the exposure variables are jointly significant with an F-test p-value of 0.009.

	First Trimester	Second Trimester	Third Trimester	All Trimesters
Weeks 0-13	0.044***			0.040*
	(0.015)			(0.021)
Weeks 14-26		$0.041^{***}$		-0.000
		(0.015)		(0.028)
Weeks 27-40			$0.034^{**}$	0.027
			(0.015)	(0.022)
Girl	-0.007	-0.007	-0.007	-0.007
	(0.006)	(0.006)	(0.006)	(0.006)
Multiples	0.079***	0.079***	0.080***	0.080***
	(0.010)	(0.010)	(0.010)	(0.010)
Age 20 to $24$	$0.056^{***}$	$0.056^{***}$	0.056***	$0.056^{***}$
	(0.022)	(0.022)	(0.022)	(0.022)
Age $25$ to $29$	0.070***	$0.069^{***}$	0.070***	$0.069^{***}$
	(0.022)	(0.022)	(0.022)	(0.022)
Age 30 +	$0.087^{***}$	0.087***	$0.086^{***}$	$0.087^{***}$
	(0.022)	(0.022)	(0.022)	(0.022)
Parity 2 and 3	-0.015 **	$-0.015^{**}$	$-0.015^{**}$	-0.015**
	(0.007)	(0.007)	(0.007)	(0.007)
Parity 4 and 5	0.009	0.009	0.009	0.009
	(0.009)	(0.009)	(0.009)	(0.009)
Parity 6 +	0.030***	0.030***	0.030***	0.030***
	(0.009)	(0.009)	(0.009)	(0.009)
Neighborhood	Yes	Yes	Yes	Yes
Seasonality	Yes	Yes	Yes	Yes
Number of Observations	7711	7711	7711	7711
Mean of Dependent Variable	0.065	0.065	0.065	0.065
p-value F-test Trimesters				0.009

Table 2 – Effects on stillbirth

#### 6.2 Nature of culling

The results described above provide evidence of substantial culling. As described in Section 2, it seems natural to assume that culling disproportionally affects the weakest among the population of mothers (or fetuses). However, Section 3 suggests that culling in the context of the 1918 flu pandemic might be more complex. Two main hypotheses emerge from our discussion of the medical literature: protective effects of childhood exposure to similar influenza strains (Worobey *et al.*, 2014), and protective effects of a suppressed immune system (Morens *et al.*, 2010). We consider both hypotheses below.

#### Protection by childhood exposure?

(Worobey *et al.*, 2014) argue that the 1889 cohort (and surrounding cohorts) might have had least protective immunity from childhood exposure to the H3N8 virus. We investigate whether this mechanism is reflected in the stillbirths we observe in our data. In particular, we split our sample in two groups: cohorts 1885 to 1893 (with potentially little immunity to the 1918 virus) and all other cohorts (with potentially better immunity). We run our baseline regression on these two subsamples, but exclude age dummies. Table 3 shows the results for cohorts 1885 to 1893, while Table 4 shows the results for the other cohorts.

	First Trimester	Second Trimester	Third Trimester	All Trimesters
Weeks 0-13	0.047**			$0.074^{***}$
	(0.020)			(0.028)
Weeks 14-26		0.019		-0.058
		(0.024)		(0.046)
Weeks 27-40		· · · ·	0.013	0.034
			(0.026)	(0.036)
Girl	-0.006	-0.006	-0.006	-0.006
	(0.008)	(0.008)	(0.008)	(0.008)
Multiples	0.077***	$0.077^{***}$	$0.077^{***}$	$0.076^{***}$
	(0.014)	(0.015)	(0.015)	(0.014)
Parity 2 and 3	$-0.018^{*}$	$-0.017^{*}$	$-0.017^{*}$	$-0.018^{*}$
•	(0.010)	(0.010)	(0.010)	(0.010)
Parity 4 and 5	0.020*	0.021*	0.021*	0.019*
	(0.011)	(0.011)	(0.011)	(0.011)
Parity 6 +	0.011	0.012	0.012	0.010
•	(0.014)	(0.014)	(0.014)	(0.014)
Neighborhood	Yes	Yes	Yes	Yes
Seasonality	Yes	Yes	Yes	Yes
Number of Observations	3442	3442	3442	3442
Mean of Dependent Variable	0.057	0.057	0.057	0.057
p-value F-test Trimesters				0.048

Table 3 – Effects on stillbirths: Cohorts 1885 to 1893

If the explanation of (Worobey *et al.*, 2014) holds for stillbirths in our sample, we would suspect a stronger effect of influenza exposure on the cohorts 1885 to 1893. However, this is not what we find. If anything, we see larger effects of exposure in trimesters 2 and 3 among the cohorts *excluding* 1885 to 1893.

#### Protection by a suppressed immune system?

As Section 3 indicates that 'survival of the weakest' is not implausible in the context of the 1918 flu pandemic, we investigate this hypothesis by using marital status as a proxy variable for the mother's immune system and her offspring's potential future outcomes. Tables 5 and 6 show the estimation results for married and single mothers, respectively.

We clearly see that—in terms of stillbirth—married mothers are affected more strongly by the 1918 flu pandemic. The effects on married mothers are larger than the effects on the overall sample in all three trimesters and statistically significant at the 95% level or higher.

	First Trimester	Second Trimester	Third Trimester	All Trimesters
Weeks 0-13	0.041*			0.004
	(0.023)			(0.034)
Weeks 14-26		$0.062^{***}$		0.048
		(0.020)		(0.038)
Weeks 27-40			$0.051^{**}$	0.019
			(0.021)	(0.031)
Girl	-0.006	-0.006	-0.005	-0.006
	(0.008)	(0.008)	(0.008)	(0.008)
Multiples	0.102***	0.101***	$0.103^{***}$	0.102***
	(0.016)	(0.016)	(0.016)	(0.016)
Parity 2 and 3	0.012	0.013	0.013	0.014
	(0.011)	(0.011)	(0.011)	(0.011)
Parity 4 and 5	$0.035^{**}$	$0.035^{**}$	$0.035^{**}$	$0.035^{**}$
	(0.014)	(0.014)	(0.014)	(0.014)
Parity 6 +	$0.079^{***}$	$0.079^{***}$	$0.078^{***}$	$0.079^{***}$
	(0.010)	(0.010)	(0.010)	(0.010)
Neighborhood	Yes	Yes	Yes	Yes
Seasonality	Yes	Yes	Yes	Yes
Number of Observations	4070	4070	4070	4070
Mean of Dependent Variable	0.071	0.071	0.071	0.071
p-value F-test Trimesters				0.022

Table 4 – Effects on still births: Cohorts other than 1885 to 1893

On the contrary, the effects for single mothers are close to zero and statistically insignificant at conventional levels. We interpret these results as suggestive evidence for 'survival of the weakest'.

#### 6.3 Robustness checks

As described in Section 4, the city started to ration various food items between 1917 and 1920. As this period overlaps with the 1918 flu pandemic, our results in Table 2 could pick up the independent effects of the rationing period. In our first robustness check, we control for the main rationing period by including a *rationing* indicator variable for the period from October 1917 to March 1920. This variable is supposed to capture a possible independent effect of the main rationing measures. The results, depicted in Table A.1, are reassuringly similar to the results of our baseline specification in Table 2.

The 1918 flu pandemic could also affect those who are not directly affected, but born in the years after the pandemic. Potential long-run consequences include influenza mortality of a supporting family member or effects on older siblings. If these effects were important, our control group would receive partial treatment. Moreover, Chandra *et al.* (2018) find a steep drop in birth numbers 9 to 10 months after peak influenza mortality in their US data. While Figure 3 indicates only a slight drop in the number of births at the maternity hospital in 1919, it is plausible that those who conceived during the 1918 flu pandemic constitute an unusually selected sample. To demonstrate that our results are not driven by these two concerns, we exclude all births that were conceived during or after the 1918 flu pandemic. In particular, we restrict our sample to those mothers whose last menstruation occurred

	First Trimester	Second Trimester	Third Trimester	All Trimesters
Weeks 0-13	0.051***			$0.059^{**}$
	(0.017)			(0.025)
Weeks 14-26		$0.045^{**}$		-0.026
		(0.018)		(0.035)
Weeks 27-40			$0.046^{**}$	$0.053^{**}$
			(0.018)	(0.026)
Girl	-0.004	-0.004	-0.004	-0.004
	(0.007)	(0.007)	(0.007)	(0.007)
Multiples	$0.074^{***}$	0.074***	$0.074^{***}$	0.074***
	(0.013)	(0.013)	(0.013)	(0.013)
Age 20 to $24$	0.060	0.059	0.059	0.060
-	(0.044)	(0.044)	(0.044)	(0.044)
Age $25$ to $29$	0.080*	$0.079^{*}$	0.080*	$0.080^{*}$
	(0.044)	(0.044)	(0.044)	(0.044)
Age 30 +	0.098**	0.096**	$0.097^{**}$	$0.097^{**}$
	(0.044)	(0.044)	(0.044)	(0.044)
Parity 2 and 3	$-0.026^{***}$	$-0.025^{***}$	$-0.026^{***}$	$-0.026^{***}$
	(0.009)	(0.009)	(0.009)	(0.009)
Parity 4 and 5	0.003	0.003	0.003	0.003
	(0.011)	(0.011)	(0.011)	(0.011)
Parity 6 +	0.028***	0.028***	0.028***	0.028***
	(0.010)	(0.010)	(0.010)	(0.010)
Neighborhood	Yes	Yes	Yes	Yes
Seasonality	Yes	Yes	Yes	Yes
Number of Observations	5613	5613	5613	5613
Mean of Dependent Variable	0.069	0.069	0.069	0.069
p-value F-test Trimesters				0.003

Table 5 – Effects on stillMarried

on June 15, 1918, or earlier. The results of this robustness check, depicted in Table A.2, suggest that our main results in Table 2 are not driven by longer run effects of the 1918 flu pandemic or selective conception.

# 7 Discussion

We want to raise awareness of the possibility of 'survival of the weakest' in the context of the 1918 flu pandemic, but also in other contexts. Culling does not necessarily imply a bias toward zero, and future research in the fetal origins literature should take this concern more seriously. In particular, future research may discerningly address the following question:

"Which mechanisms could plausibly lead to 'survival of the weakest' in the fetal health shock under study?"

'Survival of the weakest' may result from various mechanisms. Our results might not generalize to other influenza outbreaks (e.g. Kelly, 2011; Schwandt, 2018)<sup>18</sup> and the im-

<sup>&</sup>lt;sup>18</sup>The case of seasonal influenza in recent Danish data (Schwandt, 2018) illustrates this point. Compared with the 1918 flu pandemic, seasonal influenza does not feature sizable effects on fetal or maternal mortality. Moreover, in the data of Schwandt (2018), disadvantaged mothers are particularly likely to be hospitalized

	First Trimester	Second Trimester	Third Trimester	All Trimesters
Weeks 0-13	0.024			-0.019
	(0.030)			(0.046)
Weeks 14-26		0.036		0.075
		(0.028)		(0.051)
Weeks 27-40			0.001	-0.056
			(0.037)	(0.055)
Girl	$-0.017^{*}$	$-0.017^{*}$	-0.017	-0.017
	(0.010)	(0.010)	(0.010)	(0.010)
Multiples	0.100***	0.099***	0.099***	0.097***
	(0.020)	(0.020)	(0.020)	(0.020)
Age 20 to $24$	0.053**	0.053**	0.054**	0.053**
	(0.023)	(0.023)	(0.023)	(0.023)
Age $25$ to $29$	$0.056^{**}$	$0.055^{**}$	$0.056^{**}$	$0.057^{**}$
	(0.024)	(0.024)	(0.024)	(0.024)
Age $30 +$	$0.075^{***}$	$0.074^{***}$	$0.075^{***}$	$0.075^{***}$
	(0.025)	(0.025)	(0.025)	(0.025)
Parity 2 and 3	0.013	0.014	0.013	0.013
	(0.012)	(0.012)	(0.012)	(0.012)
Parity 4 and 5	0.033	0.034	0.034	0.033
	(0.024)	(0.024)	(0.024)	(0.024)
Parity 6 +	0.013	0.014	0.012	0.012
	(0.031)	(0.030)	(0.031)	(0.030)
Neighborhood	Yes	Yes	Yes	Yes
Seasonality	Yes	Yes	Yes	Yes
Number of Observations	1984	1984	1984	1984
Mean of Dependent Variable	0.055	0.055	0.055	0.055
p-value F-test Trimesters				0.396

Table 6 – Effects on stillbirths:Single

plications for altogether different fetal health shocks are unknown. Hence, 'survival of the weakest' needs to be discussed on a case-by-case basis, and we encourage researchers in the field to lead this discussion constructively and creatively. In this section, we provide sample discussions of selected health shocks to facilitate these discussions. We focus our discussion on two distinct agricultural shocks: the Dutch potato famine of 1846–1847 and the French phylloxera crisis of 1863–1890.

The Dutch potato famine was caused by a combination of plant diseases and weather conditions that severely impacted potato and rye crops in 1845 and 1846. These conditions led to large increases in food prices. "Potatoes and rye bread were the staple food of the poorest" (Lindeboom et al., 2010), so it is unsurprising that "lower social classes suffered the most from the famine" (Lindeboom et al., 2010). Given this background, culling was likely concentrated among the fetuses of the poorest, who would likely have had relatively bad adult outcomes. Thus, 'survival of the weakest' is unlikely to be important in the context of the Dutch potato famine of 1846–1847, and the results of Lindeboom et al. (2010) would be—if at all—biased toward zero.

with severe influenza. Both facts suggest that 'survival of the weakest' is not a major concern in this analysis.

The phylloxera crisis in nineteenth-century France, analyzed by Banerjee *et al.* (2010), was an agricultural shock that did not directly affect food availability. As the pest affected wine production, its main effect was income losses for landowners and workers in winegrowing regions. However, "the area planted with vines did not decline during the crisis, both because many parcels of land that had been planted with vines would have been ill suited to all other crops and also because most growers were expecting a recovery" (Banerjee et al., 2010). Hence, it seems likely that employment among wine workers was not strongly affected, while landowners most probably suffered severe income losses. If these income losses induced culling primarily among the supposedly better-off landowners, the estimates of Banerjee *et al.* (2010) might be inflated by 'survival of the weakest'. Indeed, the authors find a decline in birth numbers in the affected regions. They argue, however, that culling "would introduce positive selection (if unborn children would have been weaker)" (Banerjee et al., 2010). Given the discussion above, we argue that the nature of culling in this setting might not be innocuous.

These two agricultural health shocks were chosen somewhat arbitrarily, but they illustrate that 'survival of the weakest' needs to be taken seriously and evaluated on a case-bycase basis. Because health shocks often have heterogeneous effects, it is crucial to critically assess the typical assumption that culling affects the weakest.

### 8 Conclusions

The problem of culling bias (selective fetal mortality) plays a relatively minor role in the fetal origins literature. Many studies assume positively selected survivors, but we argue that this seemingly natural assumption does not necessarily hold in any setting. To make our case more tangible, we investigated the perhaps most prominent fetal health shock in the literature: the 1918 flu pandemic. Our review of the medical literature suggests that culling was of major quantitative importance, as the pandemic caused maternal mortality, miscarriages, stillbirths, and potentially also abortions. Assessing the nature of culling is complex, but two medical hypotheses cast doubt on the standard assumption of positively selected survivors. In particular, women may have seen some degree of protection against the virus if they were exposed to a similar virus in childhood; and if their immune system was suppressed. It is unclear how a woman's birth cohort relates to her offspring's future outcomes, but a suppressed immune system may well be associated with worse prospects. Although these hypotheses are not based on rigorous evidence, they certainly demonstrate how intricate the evaluation of culling bias can be. Moreover, these hypotheses suggest that 'survival of the weakest' is not implausible in the context of the 1918 flu pandemic.

We collected data from the maternity hospital in Bern, Switzerland, and found that culling as measured by stillbirths was indeed quantitatively important. This effect of the 1918 flu pandemic is driven by married mothers in our sample. We interpret this result as suggestive evidence of 'survival of the weakest', but data limitations preclude stronger conclusions. In particular, we cannot speak to the size of the resulting bias, as such a calculation requires estimates of the correlations of the unobserved determinant of human capital with the outcome, and with exposure. Our analysis suggested that the latter correlation is plausibly negative, but a quantitative estimate of the resulting bias is beyond the scope of our study.

Future work should use richer data to evaluate whether our results hold in full-population samples. Important open questions for future research also include whether our results generalize to the determinants of a child's potential outcomes other than marital status; and whether 'survival of the weakest' is a serious concern for other fetal health shocks.

To summarize, this study aims to raise awareness of the intricacy and importance of culling. The credibility of the fetal origins literature will be further enhanced if researchers discerningly assess culling in the light of the medical literature, historical background, and empirical evidence.

# Acknowledgments

For helpful discussions and comments, we thank the participants at the Zurich Workshop on Economics in Murg, the ATHEA Conference in Vienna, the Congress of the International Health Economics Association in Boston, the Annual Congress of the Swiss Society for Economics and Statistics in Sankt Gallen, the World Economic History Congress in Boston, and the Annual Conference of the Verein für Socialpolitik in Freiburg im Breisgau. Christian Rohr and Barbara Studer Immenhauser supported us in finding and accessing the data source for this project. Martin Rölli, Michael Schumacher, Daniel Gammenthaler, Nadine Duss, Fiona Freiburghaus, Nina Tabord, Jelka Calabretti, Dominik Tedja, and Philipp Handler transcribed the data for this project. The usual disclaimer applies. Funding: This work was supported by the Swiss National Science Foundation, Project Number 156683; the Mäxi Foundation, Zurich, Switzerland; and Frank Rühli, Zurich, Switzerland. No funding source had an active role in this study. Declarations of interest: none.

# References

- Almond, D. (2006), "Is the 1918 influenza pandemic over? Long-term effects of in utero influenza exposure in the post 1940 U.S. population." *Journal of Political Economy* 114, 672–712.
- Almond, D. and Currie, J. (2011), "Killing me softly: The fetal origins hypothesis." Journal of Economic Perspectives 25, 153–172.
- Almond, D., Currie, J., and Duque, V. (2017), "Childhood circumstances and adult outcomes: Act II." NBER Working Paper 23017.
- Almond, D., Edlund, L., Li, H., and Zhang, J. (2010), The economic consequences of demographic change in East Asia, NBER EASE, Vol. 19, chap. Long-term effects of the 1959-61 China famine: ;ainland China and Hong Kong, 321–350. Chicago: University of Chicago Press.
- Almond, D. and Mazumder, B. (2005), "The 1918 influenza pandemic and subsequent health outcomes: An analysis of SIPP data." *American Economic Review* 95, 258–262.
- Almond, D. and Mazumder, B. (2011), "Health capital and the prenatal environment: The effect of Ramadan observance during pregnancy." *American Economic Journal: Applied Economics* 3, 56–85.
- Almond, D., Mazumder, B., and van Ewijk, R. (2015), "In utero Ramadan exposure and children's Aacademic performance." *Economic Journal* 125, 1501–1533.

- Alt, M. and Sutter, E. (1985), "Bethoert, verfuehrt, gefallen ...": Zur Situation der unverheirateten Mütter in der Stadt Zürich um die Wende zum 20. Jahrhundert." Itinera 2-3, 120–148.
- 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." *Review of Economics* and Statistics **92**, 714–728.
- Barker, D. J. (1990), "The fetal and infant origins of adult disease." *British Medical Journal* .
- Barreca, A. I. (2010), "The long-term economic impact of in utero and postnatal exposure to malaria." *Journal of Human Resources* **45**, 865–892.
- Barry, J. M. (2009), The great influenza. London: Penguin.
- Baumgarth, N., Carroll, M. C., and Gonzalez, S. (2013), Textbook of influenza, chap. Antibody-mediated immunity, 283–297. John Wiley & Sons, Ltd.
- Beach, B., Ferrie, J. P., and Saavedra, M. H. (2018), "Fetal shock or selection? The 1918 influenza pandemic and human capital development." NBER Working Paper 24725.
- Bengtsson, T., Dribe, M., and Eriksson, B. (2018), "Social class and excess mortality in Sweden during the 1918 influenza pandemic." American Journal of Epidemiology.
- Bengtsson, T. and Hertz, J. (2015), "The long lasting influenza: The impact of fetal stress during the 1918 influenza pandemic on socioeconomic attainment and health in Sweden 1968-2012." *IZA Working Paper 9327*.
- Bland, P. B. (1919), "Influenza in its relation to pregnancy and labor." The American Journal of Obstetrics and Diseases of Women and Children.
- Bloom-Feshbach, K., Simonsen, L., Viboud, C., Mølbak, K., Miller, M. A., Gottfredsson, M., and Andreasen, V. (2011), "Natality decline and miscarriages associated with the 1918 influenza pandemic: The Scandinavian and United States experience." *The Journal* of Infectious Diseases 204, 1157–1164.
- Bloom-Feshbach, K., Simonsen, L., Viboud, C., Mølbak, K., Miller, M. A., Gottfredsson, M., and Andreasen, V. (2012), "Reply to Mamelund." *The Journal of Infectious Diseases* 206, 141–143.
- Brown, R. and Thomas, D. (2018), "On the long term effects of the 1918 influenza pandemic." *Working paper*.
- Brundage, J. F. and Shanks, G. D. (2008), "Deaths from bacterial pneumonia during 1918-19 influenza pandemic." *Emerging Infectious Diseases* 18, 1193–1199.

- Camacho, A. (2008), "Stress and birth weight: Evidence from terrorist attacks." American Economic Review: Papers and Proceedings 98, 511–515.
- Chandra, S., Christensen, J., Mamelund, S., and Paneth, N. (2018), "Short-term birth sequelae of the 1918-20 influenza pandemic in the United States: State-level analysis." *American Journal of Epidemiology*.
- Chandra, S. and Lu, Y.-L. (2015), "Fertility decline and the 1918 influenza pandemic in Taiwan." *Biodemography and Social Biology* **61**, 266–272.
- Chandra, S. and Yu, Y.-L. (2015), "The 1918 influenza pandemic and subsequent birth deficit in Japan." *Demographic Research* **33**, 313–326.
- Currie, J. and Rossin-Slater, M. (2013), "Weathering the storm: Hurricanes and birth outcomes." *Journal of Health Economics* **32**, 487–503.
- Currie, J. and Schwandt, H. (2017), "The 9/11 dust cloud and pregnancy outcomes: A reconsideration." *Journal of Human Resources* **51**, 805–831.
- Currie, J. and Walker, R. (2011), "Traffic congestion and infant health: Evidence from E-ZPass." American Economic Journal: Applied Economics 3, 65–90.
- Dahal, S., Mizumoto, K., Bolin, B., Viboud, C., and Chowell, G. (2018), "Stillbirth risk and spatial variation in excess death rates during the 1918-1920 influenza pandemic in Arizona, USA." American Journal of Epidemiology.
- Degen, B. (2013), "Erwerbsersatzordnung." Historisches Lexikon der Schweiz (HLS), 20.07.2013, www.hls-dhs-dss.ch/textes/d/D16610.php.
- Dübi, P. and Berger, M. (1976), 100 Jahre kantonales Frauenspital Bern. Bern: Haupt.
- Eidg. statistisches Bureau (Ed.) (1924), Die Bewegung der Bevölkerung in der Schweiz im Jahre 1922. Schweizerische statistische Mitteilungen, Bern: A. Francke, (annual publication; considered from 1913 to 1922).
- Eidgenössisches Arbeitsamt (Ed.) (1923), Haushaltsrechnungen schweizerischer Familien aus dem Jahre 1921, Vol. 1 of Sozialstatistische Mitteilungen. Bern: Eidgenössisches Arbeitsamt.
- Forsdahl, A. (1977), "Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic disease?" British Journal of Preventive and Social Medicine 31, 91–95.
- Fukuyama, S. and Kawaoka, Y. (2011), "The pathogenesis of influenza virus infections: The contributions of virus and host factors." *Current Opinion in Immunology* 23, 481–486.

- Gagnon, A., Acosta, E., Hallman, S., Bourbeau, R., Dillon, L. Y., Ouellette, N., Earn, D. J. D., Herring, D. A., Inwood, K., Madrenas, J., and Miller, M. S. (2018), "Pandemic paradox: Early life H2N2 pandemic influenza infection enhanced susceptibility to death during the 2009 H1N1 pandemic." mBio 9.
- Gagnon, A., Acosta, J. E., Madrenas, J., and Miller, M. S. (2015), "Is antigenic sin always "original?" Reexamining the evidence regarding circulation of a human H1 influenza virus immediately prior to the 1918 Spanish flu." *PLOS Pathogens* 11.
- Gagnon, A., Miller, M. S., Hallman, S. A., Bourbeau, R., Herring, D. A., Earn, D. J. D., and Madrenas, J. (2013), "Age-specific mortality during the 1918 influenza pandemic: Unravelling the mystery of high young adult mortality." *PLOS One* 8.
- Garthwaite, C. (2008), "The effect of in-utero conditions on long term health: Evidence from the 1918 flu pandemic." *Working paper*.
- General Ulrich Wille (1919), Bericht an die Bundesversammlung über den Aktivdienst 1914/18. Zürich: Buchdruckerei Arnold Bopp & Cie.
- Grantz, K. H., Rane, M. S., Salje, H., Glass, G. E., Schachterle, S. E., and Cummings, D. A. T. (2018), "Disparities in influenza mortality and transmission related to sociodemographic factors within Chicago in the pandemic of 1918." *PNAS* 13, 13839–13844.
- Guggisberg, H. (1931), Das kantonale Frauenspital Bern. Küssnacht am Rigi: Fritz Lindner Verlag.
- Harris, J. W. (1919), "Influenza occuing in pregnant women: A statistical study of thirteen hundred and fifty cases." JAMA 72, 978–980.
- Hayden, F. G., Fritz, R. S., Lobo, M. C., Alvord, W. G., Strober, W., and Straus, E. (1998), "Local and systemic cytokine responses during experimental human influenza A virus infection." *The Journal of Clinical Investigation* **101**, 643–649.
- Isen, A., Rossin-Slater, M., and Walker, W. R. (2017), "Every breath you take every dollar you'll make: The long-term consequences of the clean air act of 1970." *Journal of Political Economy* 125, 848–902.
- Iwasaki, A. and Peiris, M. (2013), Textbook of influenza, chap. Innate immunity, 269–282. John Wiley & Sons, Ltd.
- Johnson, N. P. A. S. and Mueller, J. (2002), "Updating the accounts: Global mortality of the 1918-1920 'Spanish' influenza pandemic." Bulletin of the History of Medicine 76, 105–115.
- Jöhr, A. (1912), Die Volkswirtschaft der Schweiz im Kriegsfall. Zürich: NN.

- Joris, E. and Witzig, H. (Eds.) (2001), Frauengeschichte(n). Dokumente aus zwei Jahrhunderten zur Situation der Frauen in der Schweiz. Fourth ed., Zürich: Limmat Verlag.
- Kash, J. C., Tumpey, T. M., Proll, S. C., Carter, V., Perwitasari, O., Thomas, M. J., Basler, C. F., Palese, P., Taubenberger, J. K., García-Sastre, A., Swayne, D. E., and Katze, M. G. (2006), "Genomic analysis of increased host immune and cell death responses induced by 1918 influenza virus." *Nature* 443, 578–581.
- Kelly, E. (2011), "The scourge of Asian flu: In utero exposure to pandemic influenza and the development of a cohort of British children." *Journal of Human Resources* **46**, 669–694.
- Kilbourne, E. D. (1987), Influenza. New York: Plenum Medical Book Comp.
- Kraft, A. (1908), "Die sozialen Verhältnisse der unehelichen Kinder in ihren Ursachen und Wirkungen." Jahrbuch der Schweizerischen Gesellschaft für Schulgesundheitspflege 9, 291–308.
- Lauener, P. (1926), "IV. Geburtenrückgang und Säuglingssterblichkeit in der Stadt Bern."
  In: S. A. der Stadt Bern (Ed.), *Beiträge zur Statistik der Stadt Bern*, Vol. 7, 71–109, Bern: Statistisches Amt der Stadt Bern.
- Lin, M. J. and Liu, E. M. (2014), "Does in utero exposure to illness matter? The 1918 influenza epidemic in Taiwan as a natural experiment." *Journal of Health Economics* 37, 152–163.
- Lindeboom, M., Portrait, F., and van den Berg, G. J. (2010), "Long-run effects on longevity of a nutritional shock early in life: The Dutch potato famine of 1846–1847." *Journal of Health Economics* 29, 617–629.
- Liu, Q., Zhou, Y., and Yang, Z. (2016), "The cytokine strom of severe influenza and development of immunomodulatory therapy." *Cellular and Molecular Immunology* **13**, 3–10.
- Majid, M. (2015), "The persistent effects of in utero nutrition shocks over the life cycle: Evidence from Ramadan fasting." *Journal of Development Economics* **117**, 48–57.
- Mamelund, S.-E. (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, 923–940.
- Mamelund, S.-E. (2012), "Fertility fluctuations in times of war and pandemic influenza." The Journal of Infectious Diseases 206, 140–141.
- Mamelund, S.-E. (2018), "1918 pandemic morbidity: The first wave hits the poor, the second wave hits the rich." *Influenza and Other Respiratory Viruses* **12**, 307–313.

- Mamelund, S.-E., Haneberg, B., and Mjaaland, S. (2016), "A missed summer wave of the 1918-1919 influenza pandemic: Evidence from household surveys in the USA and Norway." Open Forum Infectious Diseases 3.
- Mayer, K. H., Hirsch, H. H., Kaufmann, G., Sendi, P., and Battegay, M. (2004), "Immune reconstitution in HIV-infected patients." *Clinical Infectious Diseases* **38**, 1159–1166.
- Mazumder, B., Almond, D., Park, K., Crimmins, E., and Finch, C. (2010), "Lingering prenatal effects of the 1918 influenza pandemic on cardiovascular disease." *Journal of Developmental Origins of Health and Disease* 1, 26–34.
- Morens, D. M., Taubenberger, J. K., and Fauci, A. S. (2008), "Predominant role of bacterial pneumonia as a cause of death in pandemic influenza: Implications for pandemic influenza preparedness." *The Journal of Infectious Diseases* **198**, 962–970.
- Morens, D. M., Taubenberger, J. K., Harvey, H. A., and Memoli, M. J. (2010), "The 1918 influenza pandemic: Lessons for 2009 and the future." *Critical Care Medicine* **38**, e10–e20.
- Neelsen, S. and Straatman, T. (2012), "Long-run effects of fetal influenza exposure: Evidence from Switzerland." Social Science and Medicine 74, 58–66.
- Nelson, R. (2010), "Testing the fetal origins hypothesis in a developing country: Evidence from the 1918 influenza pandemic." *Health Economics* 19, 1181–1192.
- Nishiura, H. (2009), "Excess risk of stillbirth during the 1918-1920 influenza pandemic in Japan." Theoretical Epidemiology 147, 115.
- Oldstone, M. B. A. and Rosen, H. (2014), Sphingosine-1-phosphate signaling in immunology and infectious diseases. Current topics in microbiology and immunology, chap. Cytokine storm plays a direct role in the morbidity and mortality from influenza virus infection and is chemically treatable with a single sphingosine-1-phosphate agonist molecule, 129–147. Springer, Cham.
- Oxford, J. S. and Gill, D. (2018), "Unanswered questions about the 1918 influenza pandemic: Origin, pathology, and the virus itself." *Lancet Infectious Disease*.
- Oxford, J. S., Sefton, A., Jackson, R., Johnson, N. P. A. S., and Daniels, R. S. (1999), "Who's that lady?" *Nature Medicine* 5, 1351–1352.
- Preston, S. H., Hill, M. E., and Drevenstedt, G. L. (1998), "Childhood conditions that predict survival to advanced ages among African-Americans." Social Science & Medicine 47, 1231–1246.
- Reid, A. (2005), "The effects of the 1918-1919 influenza pandemic on infant and child health in Derbyshire." *Medical History* 49, 29–54.

- Richter, A. and Robling, P. O. (2016), "Multigenerational effects of the 1918-19 influenza pandemic on educational attainment: Evidence from Sweden." *Working paper*.
- Schreiber, H. (1993), Die Amtsvormundschaft Zürich: Zur Entstehung einer sozialpädagogischen Institution. Zürich: Zentralstelle der Studentenschaft.
- Schwandt, H. (2018), "The lasting legacy of seasonal influenza: In-utero exposure and human capital development." *CEPR Discussion Paper 12653*.
- Schweizerisches Gesundheitsamt (Ed.) (1919), Bulletin des Schweizerischen Gesundheitsamtes. Berne: E. Bühlmann & Co.
- Senn, H. (2013), "Armee." Historisches Lexikon der Schweiz (HLS), 16.07.2013, www.hlsdhs-dss.ch/textes/d/D8683.php.
- Shortridge, K. F. (1999), "The 1918 'Spanish' flu: pearls from swine?" Nature Medicine 5, 384–385.
- Simonin, von Erlach, and Burren (1918), "Verwaltungsbericht der Sanitätsdirektion." Bericht über die Staatsverwaltung des Kantons Bern 225–231.
- Sonderegger, C. (2007), "Grippe." Historisches Lexikon der Schweiz (HLS), 13.2.2007, www.hls-dhs-dss.ch/textes/d/D22714.php.
- Stadt Bern (Ed.) (1911), Bericht des Gemeinderates der Stadt Bern an den Stadtrat betreffend den allgemeinen Gang und die Ergebnisse der Gemeindeverwaltung im Jahre 1910. Berne: Buchdruckerei W. Wälchi.
- Stadt Bern (Ed.) (1912), Bericht des Gemeinderates der Stadt Bern an den Stadtrat betreffend den allgemeinen Gang und die Ergebnisse der Gemeindeverwaltung im Jahre 1911. Berne: Buchdruckerei W. Wälchi.
- Stadt Bern (Ed.) (1913), Bericht des Gemeinderates der Stadt Bern an den Stadtrat betreffend den allgemeinen Gang und die Ergebnisse der Gemeindeverwaltung im Jahre 1912. Berne: Buchdruckerei Blaser & Tschanz.
- Stadt Bern (Ed.) (1914), Verwaltungsbericht. Berne: Buchdruckerei Rösch & Schatzmann.
- Stadt Bern (Ed.) (1915), Verwaltungsbericht pro 1914. Berne: Buchdruckerei Stalder & Sieber.
- Stadt Bern (Ed.) (1916), Verwaltungsbericht pro 1915. Berne: Buchdruckerei Büchler & Co.
- Stadt Bern (Ed.) (1917), Verwaltungsbericht für das Jahr 1916. Berne: Buchdruckerei Emil Sieber.

- Stadt Bern (Ed.) (1918), Verwaltungsbericht für das Jahr 1917. Berne: Buchdruckerei Tschannel & Züttel.
- Stadt Bern (Ed.) (1919), Verwaltungsbericht für das Jahr 1918. Berne: Buchdruckerei Berner Tagblatt.
- Stadt Bern (Ed.) (1920), Verwaltungsbericht für das Jahr 1919. Berne: S.B.V. 1503-600.
- Stadt Bern (Ed.) (1921), Verwaltungsbericht für das Jahr 1920. Berne: S.B.V. 2761-600.
- Stadt Bern (Ed.) (1922), Verwaltungsbericht für das Jahr 1921. Berne: S.B.V. 3995-600.
- Stadt Bern (Ed.) (1923), Verwaltungsbericht für das Jahr 1922. Berne: S.B.V. 5201-630.
- Statistische Bureau des schweiz. Finanzdepartementes (Ed.) (1916-1924), Die Bewegung der Bevölkerung in der Schweiz im Jahre 1913-1922. Bern: Kommissionsverlag A. Francke.
- Taubenberger, J. K. and Morens, D. M. (2006), "1918 influenza: the mother of all pandemics." *Emerging Infectious Diseases* 12, 15–22.
- Taubenberger, J. K. and Morens, D. M. (2010), "Influenza: The once and future pandemic." *Public Health Reports* 125, 16–26.
- Tisoncik, J. R., Korth, M. J., Simmons, C. P., Farrar, J., Martin, T. R., and Katze, M. G. (2012), "Into the eye of the cytokine storm." *Microbiology and Molecular Biology Reviews* 16–32.
- Titus, P. and Jamison, J. M. (1919), "Pregnancy complicated by epidemic influenza." *JAMA* **72**, 1665–1668.
- Tscherrig, A. (2016), Krankenbesuche verboten! Die Spanische Grippe 1918/19 und die kantonalen Sanitätsbehörden in Basel-Landschaft und Basel-Stadt. Verlag Basel-Landschaft, Liestal.
- van Ewijk, R. (2011), "Long-term health effects on the next generation of Ramadan fasting during pregnancy." *Journal of Health Economics* **30**, 1246–1260.
- van Wijhe, M., Mølbak Ingholt, M., Andreasen, V., and Simonsen, L. (2018), "Loose ends in the epidemiology of the 1918 pandemic: Explaining the extreme mortality risk in young adults." *American Journal of Epidemiology*.
- Woolston, W. J. and Conley, D. O. (1918), "Epidemic pneumonia (Spanish influenza) in pregnancy." JAMA 71, 1898–1899.
- Worobey, M., Han, G. Z., and Rambaut, A. (2014), "Genesis and pathogenesis of the 1918 pandemic H1N1 influenza A virus." PNAS 111, 8107–8112.

# Appendix



Source: Stadt Bern (1911, p. 56; 1912, p. 60; 1913, p. 66; 1914, p. 52; 1915, p. 59; 1916, p. 58; 1917, p. 60; 1918, p. 68; 1919, p. 81; 1920, p. 84; 1921, p. 67; 1922, p. 58; 1923, p. 68).





Without additional rations. Source: Stadt Bern (1918, p. 89; 1919, p. 115; 1920, p. 116; 1921, p. 89).

Figure A.2 – Rationing, 1917–1920



Source: Stadt Bern (1918, p. 100; 1919, p. 117; 1920, p. 117/118; 1921, p. 92). Figure A.3 – Price limits, 1917–1920





Figure A.4 – Food supply at the maternity hospital, 1913–1922

	First Trimester	Second Trimester	Third Trimester	All Trimesters
Weeks 0-13	0.039**			0.042*
	(0.017)			(0.024)
Weeks 14-26		$0.035^{**}$		-0.001
		(0.017)		(0.028)
Weeks 27-40			0.025	0.029
			(0.018)	(0.024)
Girl	-0.007	-0.007	-0.007	-0.007
	(0.006)	(0.006)	(0.006)	(0.006)
Multiples	0.079***	0.079***	0.079***	0.080***
	(0.010)	(0.010)	(0.010)	(0.010)
Rationing Period	0.004	0.005	0.007	-0.002
-	(0.007)	(0.007)	(0.007)	(0.008)
Age 20 to 24	0.056***	0.056***	$0.056^{***}$	0.056***
-	(0.022)	(0.022)	(0.022)	(0.022)
Age 25 to 29	0.070***	0.069***	0.069***	0.069***
-	(0.022)	(0.022)	(0.022)	(0.022)
Age 30 +	$0.087^{***}$	0.086***	0.086***	0.087***
-	(0.022)	(0.022)	(0.022)	(0.022)
Parity 2 and 3	$-0.015^{**}$	-0.014*	$-0.015^{**}$	$-0.015^{**}$
	(0.007)	(0.007)	(0.007)	(0.007)
Parity 4 and 5	0.009	0.009	0.009	0.009
-	(0.009)	(0.009)	(0.009)	(0.009)
Parity 6 +	0.030***	0.030***	0.030***	0.030***
·	(0.009)	(0.009)	(0.009)	(0.009)
Neighborhood	Yes	Yes	Yes	Yes
Seasonality	Yes	Yes	Yes	Yes
Number of Observations	7711	7711	7711	7711
Mean of Dependent Variable	0.065	0.065	0.065	0.065
p-value F-test Trimesters				0.061

 ${\bf Table} ~ {\bf A.1}-{\rm Effects} ~ {\rm on} ~ {\rm stillbirths:} ~ {\rm Controlling} ~ {\rm for} ~ {\rm rationing}$ 

	First Trimester	Second Trimester	Third Trimester	All Trimesters
Weeks 0-13	0.058			0.076
	(0.036)			(0.069)
Weeks 14-26		$0.036^{**}$		-0.035
		(0.018)		(0.048)
Weeks 27-40			0.040**	$0.050^{*}$
			(0.016)	(0.026)
Girl	-0.004	-0.004	-0.004	-0.004
	(0.007)	(0.007)	(0.007)	(0.007)
Multiples	0.062***	0.061***	0.062***	0.063***
	(0.014)	(0.014)	(0.014)	(0.014)
Age 20 to $24$	0.047**	$0.047^{*}$	$0.047^{*}$	0.046*
	(0.024)	(0.024)	(0.024)	(0.024)
Age $25$ to $29$	$0.045^{*}$	$0.044^{*}$	$0.043^{*}$	$0.043^{*}$
	(0.024)	(0.024)	(0.024)	(0.024)
Age 30 +	0.072***	0.072***	0.071***	$0.071^{***}$
	(0.024)	(0.024)	(0.024)	(0.024)
Parity 2 and 3	$-0.016^{*}$	-0.016*	-0.016*	$-0.015^{*}$
	(0.009)	(0.009)	(0.009)	(0.009)
Parity 4 and 5	$0.019^{*}$	$0.019^{*}$	$0.019^{*}$	$0.019^{*}$
	(0.011)	(0.011)	(0.011)	(0.011)
Parity 6 +	0.033***	0.033***	0.033***	0.034***
	(0.011)	(0.011)	(0.011)	(0.011)
Neighborhood	Yes	Yes	Yes	Yes
Seasonality	Yes	Yes	Yes	Yes
Number of Observations	4954	4954	4954	4954
Mean of Dependent Variable	0.061	0.061	0.061	0.061
p-value F-test Trimesters				0.048

Table A.2 – Effects on still births: Conceived before the 1918 flu pandemic