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By
Svenn-Erik Mamelund

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Department of Economics
University of Oslo

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University of Oslo
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P. O.Box 1095 Blindern
N-0317 OSLO Norway
Telephone: + 47 22855127
Fax: + 47 22855035
Internet: http://www.oekonomi.uio.no/
e-mail: econdep@econ.uio.no

In co-operation with
The Frisch Centre for Economic Research

Gaustadalleén 21
N-0371 OSLO Norway
Telephone: +47 22 95 88 20
Fax: +47 22 95 88 25
Internet: http://www.frisch.uio.no/
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Can the Spanish Influenza pandemic of 1918 explain the baby-boom of 1920 in neutral Norway?

SVENN-ERIK MAMELUND

Department of Economics, University of Oslo, P.O. Box 1095 Blindern, 0317 Oslo, Norway
(Tel: +47 22 85 51 61; Fax: +47 22 85 50 35; E-mail: sem@econ.uio.no)

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Abstract. The main purpose of this paper is to test the hypothesis that Spanish Influenza is the explanation of the dramatic fertility decline in Norway, from 1918 to 1919, and the subsequent baby-boom in 1920. The European country analyzed was not randomly picked; a neutral haven was chosen to possibly rule out the other, and probably more obvious candidate explaining the baby-boom of 1920; the First World War. The data used in the analysis are, in a European context, of superior quality as registration of population data including vital statistics continued as normal in Norway because the First World War did not disturb it. The paper also draws attention to the importance of including – in epidemic crisis models – not only mortality but also fear of contracting a coming or present epidemic – as well present epidemic disease experience – to explain why conception rates may fall in connection with an epidemic.

1. Introduction[1]

A number of European countries experienced a relatively sharp decline in fertility during the years 1914-1919, especially from 1918 to 1919, and a subsequent baby-boom in 1920 (Festy, 1979). Surprisingly, there are few studies that in any depth have analyzed the baby-boom after the First World War as opposed to the more famous Second World War baby-boom. The commonly accepted explanation of the Second World War baby-boom, a compensation of the low level of marriages and births during the war, is probably not invalid for the First World War baby-boom either, especially not for the belligerent countries. This paper, however, will give attention to a possibly less obvious candidate, the Spanish Influenza pandemic of 1918 (Höijer, 1959; Pool,

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1973; Rice, 1983; Underwood, 1983; Mills, 1986; Johnson, 2000). At the time of armistice 11 November 1918, Spanish Influenza spread through Europe on its second out of three waves in 1918. Globally, and during all three waves, it affected at least 500 million persons or over one fourth of the population at that time (Laidlaw, 1935). The global death toll was between 50 and 100 million (Johnson and Mueller, 2002), which is five to ten times the death toll of the First World War. The highest incidences and mortality were observed among the most fertile and productive population in the age span 15-40 years. A reasonable hypothesis is therefore that the large cohort size of 1920 is a result of a compensating rebound of conceptions in 1919 due to postponed conceptions during Spanish Influenza in 1918 as well as a "demand" to replace the dead. The paper tests this hypothesis by looking at neutral Norway where it can be assumed that Spanish Influenza was the dominating factor shaping the extraordinarily fertility pattern of 1918-1920. There was no separation of people planning to marry or to have children in Norway during the war. Consequently, people had the chance to have the children they wanted and those who wanted to marry probably did so. Although front pages of most Norwegian newspapers brought the latest news from the war literary every day, it can further be assumed that the fear of being dragged into the war was not enough to consider postponing births and marriages 1914-1918. The war did not, following these assumptions, create any potential for a compensating rebound of births and marriages after the war. For the belligerent countries, however, the war certainly created a potential for a compensating rebound due to postponed conceptions as well as new pregnancies to replace the dead. For Britain, Johnson (2000:230) believes "quantifying these, particularly the portion due to influenza and that due to the effects of war, is virtually impossible". Johnson explains this by "the fact that both events fell so heavily on the same segments of the population, the young adults. Further, the disruption of population by war rendered the basic population data unreliable" (p. 231). The second point of this argument is invalid for Norway as registration of population data including vital statistics continued as usual and was not disturbed by the war. The first point of Johnson's argument might also be partly incorrect, at least if Britain is analyzed in a wider European perspective. By analyzing European countries with sufficiently detailed data, it should be possible, in regression models, to estimate the effect of Spanish Influenza mortality (morbidity data do not exist for most countries) on the fertility fluctuations in 1918 and 1919 by including a control for military status during WW1 (belligerent or neutral). To do a comparative analysis goes beyond the goal of this paper, but is definitely a task worth pursuing in a later study.

The present paper is structured as follows. In section 2, a framework, which describes the demographic development during a single and combined mortality-fertility crisis, is presented.

Section 3 shows the data and methods used. In section 4.1, I describe the effects on fertility assumed to be associated with Spanish Influenza 1918-1920. The following specific questions will be addressed. First, did coital frequency and conceptions fall when morbidity and mortality peaked in 1918? Special emphasis will be given to the causes of a possible fall in the coital frequency in 1918. Is it due to couples being sick, couples dissolved by death or both? Also, what is the effect of fear of an inevitably coming epidemic on conceptions? (The epidemic was reported in the United States and other countries in Europe some months before it came to Norway). To my knowledge, no previous study has tried to decompose the effect of fear of Spanish Influenza coming as well as actual morbidity and mortality experience on conceptions postponed in 1918. Second, did high morbidity among pregnant women lead to higher maternal mortality, more abortions and stillbirths, and thus fewer live births in 1918? Third, are the postponed conceptions and pregnancies in 1918 caught up in 1919 and materialized as births in 1920? What are the reasons? To compensate postponed conceptions? Replace the dead? In section 4.2, least square regression is applied on aggregate data for 37 rural and urban parts of 20 Norwegian counties to estimate the independent effect of morbidity and mortality on the downturn in conceptions during the second half of 1918 and the rebound in conceptions in 1919. The paper is closed by the conclusion in section 5.

2. A descriptive framework for analysis of a combined mortality-fertility crisis

Palloni (1988) has developed a general framework that describes the demographic changes during and after a combined mortality-fertility crisis. Palloni distinguishes the following four phases in connection with a single crisis (which I here have labeled model A):

Model A:

1. A gradual and then sudden increase in mortality
2. A drop in fertility following excess mortality
3. A drop in mortality either at or below pre-crisis level
4. A rebound of fertility a year or two after the crisis to levels higher than the pre-crisis level

Palloni (1988) also distinguishes between intensity and duration as the two most important features of a single crisis. There are three factors that determine the *intensity* of a crisis: First, excess mortality during the peak of the crisis, second, the size of the drop in fertility, and third, the size of the rebound in fertility in the post-crisis period. The *duration* of a crisis is defined as the interval where excess mortality is observed. Palloni also emphasis that mortality in phases 1-3

can have different age and sex characteristics, and that the downturn (Phase 2) and the rebound (Phase 4) in fertility can vary by age.

Palloni omits three important factors in his model (Model A). First, levels and characteristics of morbidity in phases 1-3 can also have an impact on the downturn (Phase 2) and rebound (Phase 4) of fertility (to be discussed below). Second, in phases 1 and 2, it is not fertility rates, but *coital frequency*, one of the proximate determinants affecting exposure to intercourse (Bongaarts, 1983), and consequently *conception* rates, that decline immediately with increasing levels of morbidity and mortality and reaches bottom level when morbidity and mortality peak. Fertility rates can, however, also decline during peak morbidity and mortality due to increased occurrence of three other proximate determinants that affect pregnancy outcome, namely spontaneous abortions, stillbirths, and maternal deaths. Third, in Phase 3, when morbidity and mortality have reached pre-crisis level or normal level, the postponed conceptions are gradually being caught up (at a level higher than normal to compensate for the low levels in Phase 2) and the dead (children) are "replaced". In the rebound stage (Phase 4), *fertility* rates rise to levels higher than the pre-crisis level as the postponement and replacement conceptions and pregnancies are materialized as births. The model (labeled here as Model B) I apply to describe the demographic development during Spanish Influenza in this paper can thus be summarized as follows:

Model B:

1. A gradual and then sudden increase in morbidity and mortality goes parallel with a drop in coital frequency and conceptions below normal level.
2. Coital frequency and conceptions drop further and reach bottom levels when morbidity and mortality peak. Increase in marriages dissolved by death lead to a further decline in coital frequency and conceptions.
3. A drop in morbidity and mortality either at or below pre-crisis level is followed by excess in conceptions compared to normal levels as couples are compensating (for the deficit in Phase 1 and Phase 2) and "replacing" the dead (particularly of children that succumbed).
4. A rebound of fertility a year or two after the crisis to levels higher than the normal pre-crisis level (due to compensation and replacement).

In the rest of this section, I discuss the underlying causes why we should expect cyclical movements in fertility due to Spanish Influenza morbidity and mortality 1918-1920.

i) *Change in coital frequency due to Spanish Influenza morbidity*

There are two reasons to assume that a couple would reduce their coital frequency during the peak of the pandemic (Phase 2). First, spouses struck by influenza lost their libido and did not want to have sex. The duration of the bed rest and the time to recover from influenza and pneumonia affect the period of abstinence. Second, in order to reduce the chances of infecting each other, couples not previously infected restrained from having sex (Phases 1-3) (Mills, 1986). I further assume that couples where both spouses survived decided to become pregnant in 1919 (Phase 3) if they postponed conceptions in 1918. The bulk of the baby-boom would then be materialized in 1920 (Phase 4).

ii) *Marriages dissolved by Spanish Influenza*

According to the Norwegian marriage law, a widow was not allowed to remarry before she had had a year of mourning. The law, however, opened for earlier remarriage if the widow and her children experienced severe poverty and distress. Although the law did not apply for men, it was also customary for widowers to have a year of mourning. If we assume that the widowed did not remarry in 1918 or 1919, that there were no sex without remarrying, and that the widows did not become pregnant just before their husbands died, neither widows nor widowers were able to realize a conception in 1918 or in 1919 to compensate for the postponed conceptions in 1918. This mechanism would actually depress the potential for a compensating rebound of fertility in 1920.

iii) *Death and diseases in connection with pregnancy and birth*

Women in pregnancy, especially those in the last trimester, were extremely vulnerable to spontaneous abortion, stillbirth and maternal death if Spanish Influenza was followed by bacterial complications, for example pneumonia (Harris, 1919; Bourne, 1922). Two contemporary investigations report that a fifth to half of pregnant women struck by fatal influenza died (Bland, 1919; Harris, 1919). The increased risk of spontaneous abortions, stillbirths and death of the mother and child during birth, significantly reduced the chances of a pregnant woman delivering a live born baby in 1918, thus reducing the fertility rates.

iv) *The effect of the pandemic on fecundity*

The pandemic can also have had an indirect effect on conception rates during Spanish Influenza by affecting female and male fecundity. As reported in iii) an unknown number of expected live-births never happened during the peak of Spanish Influenza due to higher rates of spontaneous

abortion and stillbirths (Phase 2). It is possible that this phenomena gave both more or less "power" to the process of catch up of postponed conceptions from Phase 2 to Phase 3. One the one hand, women who failed to conceive in Phase 2 was not pregnant in Phase 3. They were therefore at risk of another pregnancy after a short while, even during the peak of the crisis (Juhasz, 1971). In addition, women who failed to conceive did not breast-feed their babies. Lack of lactation shortens the postpartum infecundable period or the time it takes before ovulation and regular menstruation resumes after an abortion, stillbirth or live birth (Watkins and Menken, 1985; Lee, 1990). This is expected to give extra power to the rebound in conceptions. On the other hand however, the average postpartum infecundable period is still 2 months without breastfeeding, and in some societies it has been registered intervals of infecundity up to two years (Bongaarts, 1983). This would actually impede the catch up process of conceptions. Spanish Influenza may also have affected male fecundity negatively, additionally inhibiting the catch up process. Temporary sterility in connection with epidemic influenza has been reported, but only for men (Biraben, 1973). The net effect of Spanish Influenza on female and male fecundity seems thus to be negative. Therefore, it is reasonable to assume that high (male) morbidity rates (which is assumed to give relatively high temporary male sterility) and high spontaneous abortion- and stillbirth rates in Phase 1 and 2 have a negative effect on the rebound in conceptions in Phase 3.

3. Data and methods

To descriptively analyze the effect of Spanish Influenza on fertility, in section 4.1, I have calculated the mere differences between observed seasonal fluctuation in total mortality, influenza and pneumonia incidence and mortality, spontaneous abortions, stillbirths, maternity deaths, live-births, and marriages dissolved by death 1918-1920 from the "normal" seasonal levels of the same demographic events 1915-1917. Data on morbidity and mortality are taken from the annual health reports *Sundhetstilstanden og medisinalforholdene* published by Det civile medisinalvesen (DCM) and the data on live- and still births are from the annual population report *Folkemengdens bevegelse* published by Statistics Norway (SSB). In section 4.2, I have applied least square estimations to look at the relationship, in 1918, between excess morbidity (influenza and pneumonia) and excess mortality (all causes of death) on the one hand, and excess conceptions on the other hand (models 1-4). I also analyze whether the rebound in conceptions in 1919 can be explained by the downturn in conceptions in 1918, and the level of excess influenza and pneumonia morbidity and excess all-cause mortality in the second half of 1918 (models 5-7). Descriptive statistics for the dependent and independent variables included in the seven models applied are listed in Table 1. The dependent and independent variables are calculated for 37 units

of analysis, which are the administrative rural and urban parts of 20 counties. The dependent variable, crude conception rate (CCR), is calculated for the summer wave (July-September) and for the fall wave (October-December) of 1918 respectively, and is defined as conceptions that ended in a stillbirth (7-9 months later) or a live birth (9 months later) pr 1 000 population pr month. From 1919 and onwards the monthly data for regional stillbirths and live births were compiled according to parents permanent residence, while the practice in previous years was to compile the data for present population of parents. The denominator in the CCR from April 1918 and onwards (corresponds to births in January 1919 and onwards) is therefore de jure population. To control for the normal seasonal fluctuations, I subtracted the observed monthly conception rates in 1918 and 1919 by the average monthly conception rates in 1915-1917. As we will see in section 4.1 (Figure 1), excess morbidity and mortality during each of the two three month periods analyzed appeared only for a short time, usually a month. The effect of morbidity and of mortality on conceptions is therefore believed to be strongest in these months. The dependent variable in the final analysis is therefore computed for:

1. The month with the largest downturn or negative excess in CCR (LD = largest downturn)
2. The downturn in CCR in the month with the lowest observed level of CCR (DB = downturn bottom)

[Table 1 approximately here]

The independent variables for each of the two three-month periods analyzed were calculated according to the month when LD and DB in CCR occurred. The influenza and pneumonia incidence (IPI) is the number of *new reported cases* of present (de facto) population to district physicians or to doctors at hospitals pr 1 000 of present population pr month. As in the case with CCR, the IPI included in the final models are corrected for normal seasonal fluctuations, that is a subtraction of the observed monthly incidence rates in 1918 by the average monthly incidence rates in 1915-1917. IPI can be higher than 1 000 as a person can have visited a physician due to influenza or pneumonia more than one time a month (see Table 1). To take account of spatial differences in the average distance a doctor has to travel to reach his patients, doctor and hospital coverage as well as public sickness insurance coverage, which are factors believed to influence the number of reported influenza or pneumonia cases and thus affecting the level of IPI, weights are applied to all observations used in the analysis; The weights are a product of doctors pr km^2, share of population with public sickness insurance, and respectively physicians and hospitals pr 1

000 of present population. The reported IPI for fall of 1918 is probably closer to actual IPI (and is therefore the most reliable) than IPI during the summer-months. This is explained by the increase of virulence or lethality of Spanish Influenza from the summer to the fall, making reporting of a case more likely in the latter period. Unfortunately, data needed to estimate regional *duration* of the disease do not exist. Obviously, *intensity* (here IPI) alone cannot satisfactorily explain the assumed decline in the conception rates since duration of the average sick leave will clearly have a crucial effect on the conception rates.

The crude death rate in 1918 (CDR) is the number of deaths in present population pr month pr 1 000 present population pr month, while in 1919 the CDR is the number of deaths pr month among those with permanent residence pr 1 000 population with permanent residence pr month. The calculation of excess CDR's was conducted the same way as was done for the CCR's and the IPI's. The crude stillbirth rate (CSR) for the second half of 1918 is the number of deaths of fetuses 7-9 month old pr 1 000 population. The calculation of excess CSR's was conducted the same way as was done above for the CCR's, the IPI's, and the CDR's. The denominators in the CCR's, IPI's, CDR's, and CSR's pr month are adjusted for length of month (see Cassel, 2001). Age standardization of the dependent and independent variables was not conducted as part of the data needed to calculate standardized rates (age- and sex distribution) only exist for the census of 1920 (and not for the years 1915-1919). I believe, however, that controlling for regional differences in the age-distribution among those 20-40 years play a minor role since high morbidity and mortality primarily affect the same age-group.

The correlation between variable number 8 (CDR) and 9 (IPI) in model 3 ($r = 0.72$), and variable number 11 (CDR) and 12 (IPI) in model 4 ($r = 0.76$), are alarmingly high and may cause a multicollinearity problem (Table 2). The problem is that, although the parameter estimates will be correct, they are unstable and have too large standard errors. It may also be impossible to assess the independent effect of each of the two correlated covariates on the dependent variable. However, when I apply model 3 with and without, for instance IPI, but include controls for previous postponement in conceptions and CDR, the explanatory power differs substantially (uncorrected R^2 increases by 30 percentage points to 62 per cent including IPI). In addition, the sign of the coefficients already included in the model did not change and the statistical levels of significance increased (the standard error of the estimated coefficients decreased). Worth to note is that the two estimated coefficients without IPI included in the model were already significant at 1.0 per cent levels. In sum, this indicate, although IPI and CDR explain more than half of the same variance in CCR (52 per cent in model 3, 58 per cent in model 4), that IPI has large explanatory power on CCR independent of CDR. More or less the same story goes for model 4. This overrules the possibility of a multicollinearity problem in

models 3 and 4. The correlations between the independent variables included in the other models, are relatively low, and should therefore cause no multicollinearity problem (Table 2).

[Table 2 approximately here]

4. Results

4.1. The effects of Spanish Influenza on….

4.1.1. Conceptions, pregnancies and births

Before analyzing the assumed causal mechanisms between Spanish Influenza morbidity and mortality on the one hand, and conceptions on the other hand, I briefly describe the fertility pattern before and during WW1, to see if there was a decline in fertility connected to the war. There was no dramatic change in total fertility rate (TFR) in Norway 1912-1918. Indeed, TFR fell by 7 per cent from 1914 to 1915, which is possibly due to a decline in conceptions in the months after the outbreak of war July-August 1914, but in the years 1916-1918 TFR rose and nearly reached the pre-war level (Table 3). The decline in TFR from 1914 to 1915 may have created a potential for a compensating rebound in fertility, at first glance (on the data) seemingly not realized in the years 1916-1918. The relatively high and stable TFR in the years 1916-1918 is, however, realized in a period with more or less constant fertility decline from the 1890s to the mid 1930s (Brunborg and Mamelund, 1994). It is therefore likely that a large part of the assumed postponed conceptions in 1914 was caught up in the years 1915-1918. One cannot rule out the possibility that a "rest" of the assumed "catch-up potential" was realized after the war ended in November 1918. Also, more people than normal may have realized a pregnancy in pure joy of the war being over. Hence, WW1 is then a competitor (to take seriously) to Spanish Influenza to explain the large cohort-size in 1920. As I will discuss next in this section, only Spanish Influenza can have caused the relatively small birth cohort of 1919.

[Table 3 approximately here]

Approximately 6 000 people where off from work with influenza symptoms in the first week of April 1918, but these cases were not reported in the official statistics as influenza (Mamelund, 1998, see Figure 1). A suspicious number of influenza cases were also reported in military camps, but the disease did not spread to the general population. At this "pre-seeding" stage of the pandemic, mortality was low and deaths were only reported among those who normally die of influenza, the very young and the oldest old. The CDR from January to June 1918 was also lower

than the "normal" seasonal level. Nevertheless, the increased influenza activity in April may explain why CCR is declining by 0.7 conceptions pr 1 000 from March to April instead of increasing by 1.9 conceptions pr 1 000 as it on average did 1915-1917 (Figure 1). The fall in CCR by 1.0 conception pr 1 000 from April to May is fully explained by normal seasonal fluctuations, but it occurs at a level 2.5 conceptions pr 1 000 lower than normal. On 28 May 1918, most Norwegian newspapers printed a wire from Reuters news agency revealing that a pandemic of influenza was spreading in Europe. Only two weeks later, on 15 June, the very first scattered cases of influenza, which later proved to be the moldering of a pandemic wave, occurred in the capital of Kristiania (renamed Oslo in 1924) (Mamelund, 1998). It was not before the first week of July, however, that the number of reported cases skyrocketed and took the dimensions of a pandemic wave (see Figure 1). One out of four conceptions pr 1 000 that was postponed in total from April-December 1918 was postponed in the second quarter from April to June. The postponed conceptions in this period must basically have been due to *fear* of contracting influenza, and cannot be explained by high morbidity and mortality among couples considering to have a baby (Figure 1).

CCR normally rises and reaches the highest level of the year during the three summer months ending with August. This was not true in 1918. Instead of a normal increase in CCR of 1.4 conceptions pr 1 000 from June to August, CCR decreased by 1.0 conception pr 1 000. Of the further decline by 1.7 conceptions pr 1 000 from August to September, 40 percent is explained by normal seasonal fluctuations. The other 60 per cent is probably explained by a decline in coital frequency in connection with the increase in IPI and CDR during the first pandemic wave of Spanish Influenza (Phase 1). 44 per cent of the conceptions postponed pr 1 000 in total from April to December 1918 was postponed during the third quarter from July to September. The third quarter of 1918 was not the quarter with the highest excess mortality in 1918, but, according to the only influenza-census in Norway, carried out for the city of Bergen, the *actual* incidence was clearly highest in this quarter, especially for those in the most reproductive ages (Hanssen, 1923). When we look at the IPI-curve in Figure 1, however, it seems as if the *reported* incidence was higher during the fall of 1918 than during the summer of 1918. Many cases were probably not reported during the summer wave, however, due to the mild character of the virus at this stage.

[Figure 1 approximately here]

The local, and stable average minimum of 21.6 conceptions pr 1 000 September-November, is nearly 4.0 conceptions pr 1 000 lower than normal and is probably due to the high IPI and CDR

during the second wave of Spanish Influenza (Phase 2). Of the increase of 4.8 conceptions pr 1 000 in CCR from November (21.7 pr 1 000) to December (26.5 pr 1 000) 1918, close to 60 per cent (2.8 pr 1 000) is explained by normal seasonal fluctuations (Figure 1). The other 40 per cent of the increase in CCR is probably explained by a strong decline in both IPI and CDR. It is also possible that the end of First World War 11 November 1918 stimulated coital frequency. If this effect had any immediate significance, however, CCR should have increased from October to November, but this did not happen. Alternatively, the end of WW1 may have stimulated coital frequency in November, but as the discourage of sexual intercourse, IPI, and the loss of sexual partners (CDR as proxy), was peaking in the very same month, any assumed effect of the end of the war on CCR in this month is likely to have been overshadowed or put on hold due to the peak in IPI and CDR.

During the fourth quarter from October to December 31 percent of the conceptions postponed in total from April to December 1918 was postponed. The reason why this quarter, with the highest excess mortality did not coincide with the highest percentage of conceptions postponed must be the fact that the *actual* incidence, especially for men and women in their most reproductive ages 20-39, was much lower in the third compared to the fourth quarter of 1918 (due to gain of relative immunity) (Hanssen, 1923). The IPI-curve in Figure 1, however, was highest in the last quarter of 1918, but this is again probably due to the virus becoming more virulent (with more persons *reporting* themselves sick). When I use data from the Norwegian Sickness Insurance Fund (Riksforsikringsanstalten, 1920), I find excess in sick days of respectively 10.7 and 6.9 (which I interpret as caused by Spanish Influenza) for an average rural and urban member in the last quarter of 1918, and no departure from the normal (here; 1920-1921) during the three first quarters of 1918. The above explanation is probably also the reason why I only find excess in sick days in the last quarter of 1918.

The negative excess in CCR April-December 1918 was more than compensated by positive excess in CCR in 1919 and the first three months in 1920 (Phase 3) (Figure 1). Although CCR increased from November 1918 to March 1919 (positive excess rates compared to normal seasonal fluctuations in January-March 1919 only), births declined by 4 000 (0.26 births pr woman) to 60 000 from 1918 to 1919 (Table 3, Figure 1). Obviously, conceptions that were supposed to have occurred during the last nine months of 1918 were postponed to 1919 due to Spanish Influenza. Nearly 70 000 births were recorded in 1920 (Table 3). This was about 10 000 more births or 0.44 more births pr woman compared to 1919. The Spanish Influenza baby-boom cohort of 1920 is the second largest birth cohort ever registered in Norway, only beaten by the Second World War baby-boom cohort of 1946 with 70 747 births. The noticeable positive excess

in CCR during the first quarter of 1920 (see Figure 1), also explain the relatively large birth cohort of 1921 (see Table 3).

From the descriptive analysis presented in this section, it is tempting to claim that IPI seem to be more important than CDR explaining the fall in CCR in the second half of 1918. This is due to the finding that the largest percentage of the decline in CCR occurred during the summer wave (Phase 1), which had the highest increase in *actua*l IPI and the lowest increase in CDR. On the other hand, it is likely that CDR was more important than IPI explaining the downturn in CCR during the fall wave of 1918. However, this type of questions obviously has to be dealt with by using multivariate analysis. For example, the independent effect of IPI on CCR can only be estimated by controlling for CDR and vice versa (see section 4.2). Also, there seem to be a "fear-factor" to consider in the pre-seeding period of the pandemic; a considerable decline below normal level in CCR occurred during the spring of 1918, with little or no visible increase in IPI or CDR above normal levels. The third quarter of 1918 is thus the only quarter where we can separate the assumed effect of fear on the one hand, and the assumed effect of morbidity and mortality on the other hand, on postponement of conceptions during the pandemic. Finally, a "joy-factor", represented by the end of the war, probably stimulated coital frequency at the end of fourth quarter of 1918 (Phase 2), boosting the catch up process of conceptions postponed due to Spanish Influenza (Phase 3).

4.1.2. Age-specific pattern of fertility

As discussed in section 2, Palloni (1988) has stressed that the assumed downturn (Phase 2) and the rebound (Phase 4) in fertility can vary by age in connection with a mortality crisis (Model A). This was not the case during Spanish Influenza.

[Figure 2 approximately here]

Figure 2 show that women that experienced the largest downturn in fertility from 1918 to 1919 (following cohorts) also basically experienced the highest rebound in fertility from 1919 to 1920 ($r = 0.66$). The same figure also shows that the youngest and the oldest of the reproductive women experienced the lowest fluctuations in fertility 1918-1920. The most pronounced downturn and rebound in fertility occurred to women in the age group 25-29 years (two third of both the downturn of 0.26 children pr woman and the rebound of 0.44 children pr woman). No fewer than seven birth cohorts, those born between 1889 and 1895, had their modal birth age in

1920; the birth cohorts of 1889 and 1895 gave birth to most children at age 31 and 25 respectively (Brunborg and Mamelund, 1994).

4.1.3. Spontaneous abortions and stillbirths

In sections 4.1.1 and 4.1.2, we have seen that Spanish Influenza seems to have had a negative effect on fertility in 1919. Rising rates of spontaneous abortion and stillbirth due to Spanish Influenza probably also had a negative effect on fertility in 1918. There was actually a significant increase from 1917 to 1918 in both the spontaneous abortion rate, here defined as spontaneous abortions pr 1 000 women 5-6 months pregnant, and the stillbirth rate, defined as stillbirths pr 1 000 women 7-9 months pregnant (Table 4 and Table 5) (note the different definition of the stillbirth rate included in the multivariate models (CSR), see section 3 and model 7, Table 10).

[Table 4 approximately here]

[Table 5 approximately here]

Approximately 45 excess spontaneous abortions or a spontaneous abortion rate of 0.7 pr 1 000 pregnant were due to Spanish Influenza if the calendar year of 1918 is compared to the average normal of 1915-1917. The increase in the stillbirth rate in 1918 is mainly due to excess stillbirth rates from August to December 1918 (Figure 3). The highest excess stillbirth rates occurred in October and November, the same months with the highest excess crude death rates (compare Figure 1 and Figure 3). Approximately 335 excess stillbirths or a stillbirth rate of 22.1 are probably due to Spanish Influenza if the 5 months period from August to December 1918 is compared to the average monthly normal of 1915-1917. What is possibly more striking than the increase in both number and rates of spontaneous abortions and stillbirths from 1917 to 1918 is the marked fall from 1918 to 1919. This is probably explained by a selection effect as Spanish Influenza may have eliminated "weak" fetuses in 1918 and left fetuses with higher risk of surviving to a live birth in 1919 and partly 1920. In the next section, a third factor that must have affected fertility in 1918 negatively is discussed.

[Figure 3 approximately here]

4.1.4. Maternity mortality

Rising rates of maternity mortality due to Spanish Influenza is also thought to be negatively affecting fertility levels in 1918. At the peak of the pandemic in November 1918, the maternity mortality rate was five times higher compared to the average November month in the three previous years (Figure 4). Based on the data used to create the curves in Figure 4, I have estimated that 67 excess (all causes) maternal deaths (1915-1917 normal) or 18.3 deaths pr 1 000 pregnant women was caused by Spanish Influenza from August 1918 to January 1919. Calculated on the basis on data from Figure 2, the excess death rate for all causes (1915-1917 normal) of women 15-49 years irrespective pregnancy status, was 5.3 pr 1 000 in 1918. The lower death rates for women irrespective pregnancy status compared to the death rate of pregnant women, is what I expected to find. There exists no data that allows the calculation of excess death rates for women 15-49 years by month and by pregnancy status in 1918 and 1919. This is unfortunate for two reasons. First, it is highly likely that the excess death rates of women 15-49 who where not pregnant in 1918 (1915-1917 as normal) would be lower than the same death rate calculated irrespective pregnancy status. Second, the death rates for non-pregnant women 15-49 would probably be much higher if the excess was calculated for August 1918 to January 1919 compared to the excess rates calculated by comparing the annual figures of 1918 to that of the three normal years of 1915-1917. I do not believe, however, that calculating the excess mortality rates for the non-pregnant women 15-49 this way would exceed the corresponding rates for pregnant women. Based on the findings of Harris (1919) and Bourne (1922) reported in section 2 iii), I would also expect to find higher maternity mortality rates for pregnant in the last trimester. Unfortunately, the official data on maternity mortality are not broken down by month of pregnancy, and as such, this question reminds open to speculation.

[Figure 4 approximately here]

4.1.5. Marriages dissolved by death

Spanish Influenza had a tremendous effect on family life. An excess of 3 528 marriages or 7.7 marriages pr 1 000 existing marriages (including separated couples) was dissolved by death in 1918 using the annual average in the period 1915-1917 as normal period (see gross figures in Table 6). As we saw in section 4.1.1, CDR (as a proxy of marriage dissolution) seems to have had a negative effect on CCR in 1918.

[Table 6 approximately here]

The statistics presented in Table 6 do not allow us to give estimates on how many of the marriages that were dissolved by the death of each spouse. Statistics Norway (SSB, 1926), however, have estimated that there were approximately 2 000 more influenza and pneumonia deaths among married men above the age of 20 and also that there were 1 950 more influenza and pneumonia deaths among bachelor's in 1918 compared to 1917. The excess influenza and pneumonia death rates of bachelor's and married men respectively, assuming that the risk population of bachelor's above 15 years and married men from the 1920 census apply for 1918, are then 5.2 and 4.7 pr 1 000 for 1918. SSB (1926) did not give corresponding estimates for excess deaths of spinsters and married women. By using the same method as SSB to calculate excess in 1918, which was the difference between number of deaths in 1918 and 1917, the excess of marriages dissolved by death, disregarding sex, is 3 573. By subtracting the estimate of SSB (1926) on excess deaths of married men (2 000) from the above estimate on excess of marriages dissolved by death, disregarding sex (3 573), 1 573 of the marriages were dissolved by the death of the wife in 1918. In this estimate I assume that an insignificant share of marriages were dissolved by the simultaneous death of both spouses during 1918. By subtracting the 1 573 estimated excess deaths of married women 1918 from an estimate of excess of influenza and pneumonia deaths of women 20 years and above irrespective marital status in 1918 (1918 compared to 1917), which is 3 237, 1 664 excess deaths occurred to non-married women in 1918. The excess influenza and pneumonia death rates of spinsters and married women respectively, assuming that the risk population of spinsters above 15 years and married women from the 1920 census apply for 1918, is then 4.1 and 3.7 pr. 1 000. From the above analysis, it seems as being married lowered the risk of dying of Spanish Influenza by 10 per cent for both men and women compared to the non-married.

I expect to find that the largest share of the marriages dissolved by death in 1918 occurred among relatively newly wed and was caused by the death of the husband as the highest death rates were observed for men. This hypothesis is based on the fact that both men and women experienced the highest excess death rates (Figure 2) at or a couple of years above the average age of first marriage, which was 27.6 for men and 25.4 for women. To test this hypothesis, I have applied a method, first suggested by Pool (1973), and later used by others (Rice, 1983; Mills, 1986), on Norwegian mortality data from DCM 1922. I calculated excess age-sex specific influenza and pneumonia death shares irrespective of marital status for 1918 using 1915-1917 as normal years. The share of all marriages that was dissolved by Spanish Influenza is

$$(q_{m,x} * q_{f,y}) + (p_{m,x} * q_{f,y}) + (q_{m,x} * p_{f,y}),$$

where p is the share of the spouses surviving the pandemic, q is the share of spouses dying, m is male, f is female, x are the twelve 10-year male age groups 15-24,…, 70-79, and y are the twelve 5-year female age groups 15-19,…, 70-74. The first product of the equation describes a situation where both spouses die but not simultaneously, in the next product the husband becomes a widower, and in the third product the wife is being widowed. The calculation is done under the following three assumptions. First, women only marry men 0-5 years older than themselves. Second, the percentage married according to the 1920 census apply to 1918. Third, irrespective age and based on the finding above, the share dying is 10 per cent lower among the married than among the non-married. The results are presented in Table 7. The highest share of marriages dissolved by Spanish Influenza death occurred to women 25-29 years married to men 25-34 years (1 per cent). As expected, relatively newly wed women in their most reproductive ages had the highest risk of loosing their husbands and to become a widow due to Spanish Influenza. This result gives support to the finding (in section 4.1.2) that the largest decline in fertility in 1919 occurred to women in their most reproductive ages. In this interpretation I assume that wives not pregnant when they lost their husbands in the latter half of 1918 would not give birth to a baby in 1919.

[Table 7 approximately here]

4.2. Multivariate analysis

To estimate the effect of, for example Spanish Influenza morbidity on conceptions, net of the effect of mortality and vice versa, the descriptive analysis presented in section 4.1 needs to be supplemented by multivariate analysis. In this section, I therefore test the two-folded hypothesis that high Spanish Influenza morbidity and mortality go hand in hand with low conception rates (postponement hypothesis) in the second half of 1918 and high conception rates in 1919 (compensating and replacing hypothesis) by using least square estimations in seven multivariate models. The results are presented in Table 8, Table 9, and Table 10.

I believe that it is necessary to include an assumption that previously postponed conceptions (in 1918) affects further postponement negatively (later in 1918). When I control for conceptions already postponed in the so-called pre-seeding period of the pandemic from April to June (believed to be explained by fear for a pandemic that sooner or later would come to Norway

too), both IPI and CDR have a negative effect on CCR during the summer-wave (Table 8, models 1 and 2).

[Table 8 approximately here]

However, only IPI contribute to explain the downturn in CCR significantly, although in a very weak manner: An increase by one influenza and pneumonia case pr 1 000 population leads to a decline of only 0.006-0.007 conceptions pr 1 000 population. This result seems to confirm the finding in the descriptive analysis in section 4.1.1, that IPI is more important than CDR to explain the downturn in CCR during the period July-September 1918. The negative effect of conceptions already postponed April-June is strongly significant and rather large on the downturn July-September: When one conception pr 1 000 is postponed April-June, only 0.51-0.60 conceptions pr 1 000 are postponed during the summer wave. Since the estimated coefficient is negative and smaller than one, the result implies that people in areas with large postponements April-June postpone less July-September and vice versa.

The results of the regression models for the effect of IPI, CDR and conceptions postponed during the summer on the downturn in conceptions during the fall-wave (October-December) are somewhat striking and surprising (Table 9, models 3 and 4). First, the size of the downturn in the conception rate during the summer-wave (July-September) has a negative effect on the downturn in the conception rate during the fall-wave (October-December). However, the effect is weaker than the corresponding relationship found in model 1 and 2, 0.19-0.22 conceptions pr 1 000 compared to 0.51-0.60 conceptions pr 1 000, implying that the size of further postponement in conceptions, given previous postponement, is decreasing. Second, but maybe not so surprising, the effect of CDR is highly significant and strongly negative (as expected) on the downturn in CCR during the most lethal (fall) wave. An increase in CDR by one death pr 1 000 during the fall-wave gives a reduction in CCR of 0.46-0.49 conceptions pr 1 000 all other factors the same. Third, and most surprisingly, the effect of IPI goes in the opposite direction of what I expected. According to models 3 and 4, an increase in IPI of one influenza and pneumonia case pr 1 000 gives an increase in CCR of 0.0095-0.0158 conceptions pr 1 000. The effect is not large however, as was also found to be the case during the summer-wave (models 1 and 2), but the effect is highly significant. The result, however unexpected, is nevertheless possible to explain. First, some couples might have had sex although they were sick. The reason might be that fear of dying or grief after death of near relatives or friends made husbands and wives more intimate. Second, although people might have reported themselves sick, happiness

and relief that WWI ended 11. November 1918 may have resulted in more people wishing to have sex. Third, many people reporting themselves sick during the fall-wave might not have been very ill, but the increase in lethality of Spanish Influenza probably gave high incentives to report sickness, and to blame all diseases with flu-like symptoms for being influenza.

[Table 9 approximately here]

When I do not control for the increase in IPI, CDR, and CSR July-December 1918, a downturn in CCR by one conception pr 1 000 April-December 1918 leads to a rebound in CCR January-December 1919 of two conception pr 1 000 (model 5, Table 10).

[Table 10 approximately here]

In model 6, however, I add two possible confounding factors. First, IPI to control for the possibility that the disease affected fecundity of the survivors negatively (see section 2 iv)), and second, CDR to control for the assumption that the chances that the widowed in 1918 are realizing a pregnancy in 1919 was low due to a year of mourning (see section 2, part ii). In this case, the independent effect of a downturn in CCR by one conception pr 1 000 April-December 1918 leads to a decrease in the rebound in CCR January-December 1919 by 0.3 conceptions pr 1 000 to 1.6 conception pr 1 000, and the explanatory power of the model (R^2) increases by 16 percentage points to 73 per cent. Surprisingly, the two new independent variables did not affect the size of the rebound negatively as expected. This does not mean that the assumed effects were wrong. Rather, I suspect that other mechanisms have worked simultaneously, positively affecting the dependent variable, and thus canceling out the expected negative effect on the rebound-process of mortality and morbidity. The reason why the effect of CDR 1918 (as a proxy of marital dissolution due to Spanish Influenza deaths) was positive on the rebound in conceptions 1919 can be that a larger proportion of the conceptions than usual were carried out of wedlock since opportunity of remarriage in 1919 was limited due to the year of mourning imposed by the marriage law. The data support this hypothesis: illegitimate births pr 1 000 population increased by 20 per cent in 1920 (1915-1917 as normal period) compared to a more moderate increase of 7.5 per cent in legitimate births pr 1 000 population in 1920 (1915-1917 as normal period) (see Table 3), and the remarriage rates declined and increased as expected for both widowers and widows from 1918 to 1919 and 1919 to 1920 respectively (Table 11).

[Table 11 approximately here]

An alternative, and probably equally important explanation, is that married couples that did not loose their spouse to the flu living in areas with high CDR and dissolution rates of marriages due to the flu, caught up relatively more conceptions than married couples that did not loose their spouse to the flu living in areas with low CDR and dissolution rates of marriages due to the flu. The "underlying" explanation might be twofold; first, loss of a child due to the flu may have increased the whish to have another child (to replace the dead), and therefore also a contribution to maintain the population. Second, high community mortality in general may have given people intended or unintended wish to "replace" the dead. The finding that IPI 1918 positively affected conceptions in 1919 indicates that the "male temporary infertility hypothesis" may not be valid for Spanish Influenza. In model 7, which is an extension of model 6, I include stillbirth rates in the second half of 1918 as a new independent variable to control for the assumed negative effect of high stillbirth rates in 1918 on the rebound of the conceptions rates in 1919 (see section 2 iv)). The effect of CSR 1918 on the rebound in CCR 1919 is negative and significant as expected; an increase by one stillbirth pr 1 000 population in 1918 reduces the rebound potential in 1919 by 3.9 conceptions pr 1 000 population. The independent effect of a downturn in CCR in 1918 by one conception pr 1 000, all other factors the same, now (model 7) leads to a rebound of 1.5 conceptions pr 1 000 in 1919. In total, the best model (model 7), explain nearly 80 percent of the variance in the rebound stage of the pandemic. The reason why one conception postponed in 1918 leads to more than one conception caught up in 1919 may be due to four factors: First, and not necessary in the order of importance, joy that Spanish Influenza is over. Second, a wish, intended or unintended, to replace the dead, especially small children. Third, joy that WW1 is over, and fourth, realization of what is left to catch up from the assumed catch up potential created by the start of WW1 in August 1914, not being caught up before Spanish Influenza arrived in 1918.

5. Conclusion

In this paper the effects of Spanish Influenza morbidity and mortality on fertility in Norway 1918-1920 have been analyzed. The paper gives new insight into the demographic impact of Spanish Influenza beyond mere mortality. The empirical findings suggest that Spanish Influenza, which infected 1.2 million (45 per cent) and killed 15 000 (5.7 deaths pr 1 000 population) Norwegians, and with most victims in the fertile and productive ages of 15-40, is the explanation of the decline of more than 4 000 births or 0.26 births per woman from 1918 to 1919. The baby-boom in 1920, which created the second largest birth cohort ever registered in Norway (nearly 70

000 births), is also explained by Spanish Influenza; First, fear that the new mysterious disease reported elsewhere in Europe and USA would come to Norway (April-June 1918), fear of infection after the first pandemic wave arrived (July 1918), and personal disease experience restrained people from having sex. Second, those that were bereaved their spouses due to Spanish Influenza were legally (no remarriage before a year of mourning) and morally (no marriage, no sex) restrained from becoming pregnant in 1918. In sum, this lead to a massive postponement of conceptions in the last nine months of 1918, caught up in 1919, after Spanish Influenza vanished. The size of the rebound in conceptions, however, surpassed the compensating rebound potential (created by the postponement of conception in 1918) by 50 per cent; one conception postponed pr 1 000 during the last nine months of 1918 resulted in 1.5 conceptions pr 1 000 caught up in 1919. The reason is probably that, in addition to catching up the postponed conceptions, that additional conceptions were realized to replace the dead, especially of children. The possible effect of peace in November 1918 on the (spontaneous) wish to become pregnant (or have sex) is also appreciated, but in general, the conclusion from the analysis is that WW1 did not create a significant potential for a compensating rebound in marriages and fertility as there was no separation of people planning to marry or to have children in Norway during the more than four year long war. Consequently, there was no dramatic decline in fertility in Norway that can be linked to WW1. Also, as the number of military accidents was insignificant in neutral Norway (Mamelund, 2002), the war did not cause a "lost generation" and thus a "demand" to replace the dead and to sustain the population by giving birth to a baby.

The importance of – and implications of the present paper can be summarized as follows; First, it is among few papers that in depth have analyzed the baby-boom after the First World War as opposed to the more famous Second World War baby-boom. Second, the explanation of the baby-boom in 1920 is, by using detailed and reliable historical demographic data available for Norway that has not been analyzed before, explained by a likewise hitherto little appreciated factor; the Spanish Influenza pandemic of 1918. This is mostly surprising when we know that Spanish Influenza claimed five to ten times more deaths in a period less than a year compared to more than four years of war. Third, if the question of interest is the short-term and independent effects of Spanish Influenza on fertility, neutral countries seems to have an advantage as unit of analysis as it is assumed that it is relatively easy to distinguish the effects of the pandemic from the effects of war in these countries. Another advantage of using data from neutral countries is that registration of population data including vital statistics should go relatively undisturbed by the war. Fourth, the paper has shown the importance of including – in epidemic crisis models – not only mortality but also fear of contracting a coming or present epidemic – as well present

epidemic disease experience – to explain why conception rates may fall in connection with an epidemic.

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Table 1. Descriptive statistics of dependent and independent variables

Model	Variable	Dependent variables	Min	Max	Mean	St. dev.
1	1	LD in CCR Summer 1918	0.26	- 28.95	- 7.95	5.41
2	2	DB in CCR Summer 1918	1.05	- 28.95	- 6.62	5.85
3	3	LD in CCR Fall 1918	- 2.04	- 29.63	- 8.43	5.12
4	4	DB in CCR Fall 1918	- 0.70	- 29.63	- 7.46	4.99
5	5	Rebound in CCR January-December 1919 (all twelve months)	- 50.81	72.90	23.51	25.64
Model	**Variable**	**Independent variables**	**Min**	**Max**	**Mean**	**St. dev.**
1	1	Downturn in CCR April-June 1918 (all three months)	19.54	-21.03	- 7.84	8.03
	2	Increase in CDR given LD in CCR, summer 1918	- 7.58	11.73	1.54	4.16
	3	Increase in IPI given LD in CCR, summer 1918	22.49	980.99	239.90	247.13
2	4	Downturn in CCR April-June 1918 (all three months)	19.54	-21.03	- 7.84	8.03
	5	Increase in CDR given DB in CCR, summer 1918	- 7.58	34.33	3.75	8.33
	6	Increase in IPI given DB in CCR, summer 1918	18.72	1145.96	265.01	284.27
3	7	Downturn in CCR July-September 1918 (all three months)	10.13	-34.44	- 14.74	9.70
	8	Increase in CDR given LD in CCR, fall 1918	- 3.81	87.85	18.14	18.06
	9	Increase in IPI given LD in CCR, fall 1918	65.01	1980.69	403.59	407.02
4	10	Downturn in CCR July-September 1918 (all three months)	10.13	-34.44	- 14.74	9.70
	11	Increase in CDR given DB in CCR, fall 1918	- 3.55	87.85	15.95	17.24
	12	Increase in IPI given DB in CCR, fall 1918	52.36	1504.74	351.97	330.52
5	13	Downturn in CCR April-December 1918 (all nine months)	- 2.52	- 73.93	-39.34	14.50
	14	Increase in CDR July-December 1918	33.49	126.32	58.62	18.04
	15	Increase in IPI July-December 1918	898.83	4571.00	2120.95	1027.48
	16	Increase in CSR, second half of 1918	- 5.53	3.95	0.43	1.59

Abbrevations: LD = Largest downturn; DB = Difference bottom; CCR = Crude conception rate (pr 1 000); CDR = Crude death rate (pr 1 000), IPI = Influenza and pneumonia incidence (pr 1 000); CSR = Crude stillbirth rate (pr 1 000).

Table 2. Correlations of independent variables[1]

No	1	2	3	No	4	5	6	No	7	8	9	No	10	11	12	No	13	14	15	16
1	1.00			4	1.00			7	1.00				1.00			13	1.00			
2	-0.21	1.00		5	-0.07	1.00		8	-0.41	1.00			-0.28	1.00		14	-0.32	1.00		
3	-0.28	0.31	1.00	6	-0.12	0.44	1.00	9	-0.25	0.72	1.00		-0.15	0.76	1.00	15	-0.17	0.38	1.00	
																16	-0.03	0.04	0.12	1.00

[1] Full information on each variable is given in Table 1.

Table 3. Live births, crude birth rate (CBR) and total fertility rate (TFR) in Norway 1912-1923

	1912	*1913*	*1914*	*1915*	*1916*	*1917*	*1918*	*1919*	*1920*	*1921*	*1922*	*1923*
Live births	61 409	61 294	62 111	58 975	61 120	63 969	63 468	59 486	69 326	64 610	62 908	61 731
Legitimate	57 207	56 881	57 608	54 881	56 753	59 516	59 256	55 600	64 065	59 725	58 575	57 658
Illegitimate	4 202	4 413	4 503	4 094	4 367	4 453	4 212	3 886	5 261	4 885	4 333	4 073
CBR (‰)	25.3	25.0	25.1	23.6	24.2	25.0	24.6	22.9	26.3	24.2	23.3	22.8
Legitimate	23.6	23.2	23.3	22.0	22.5	23.3	23.0	21.4	24.3	22.4	21.7	21.3
Illegitimate	1.7	1.8	1.8	1.6	1.7	1.7	1.6	1.5	2.0	1.8	1.6	1.5
TFR	3.7	3.6	3.6	3.4	3.4	3.5	3.4	3.2	3.6	3.3	3.2	3.1

Source: Brunborg and Mamelund, 1994; SSB, 1995

Table 4. Spontaneous abortions (death of fetus 5-6 months old) and spontaneous abortions pr 1 000 women 5-6 months pregnant in Norway 1915-1922 [1]

	1915	*1916*	*1917*	*1918*	*1919*	*1920*	*1921*	*1922*
Spontaneous abortions	438	455	491	505	422	443	470	433
Spontaneous abortion rate	6.6	6.6	6.9	7.4	5.9	6.0	6.5	6.3

[1] The pregnant population at risk (women 5-6 months pregnant) in 1915 is the sum of births, stillbirths, and abortions (that occurred to women 7-9 months pregnant) in April 1915 to April 1916, i.e. women who conceived July 1914 to July 1915. The pregnant population at risk in 1916 (women 5-6 months pregnant) is the sum of births, stillbirths, and abortions (that occurred to women 7-9 months pregnant) in April 1916 to April 1917, i.e. women who conceived July 1915 to July 1916, etc.
Source: DCM, 1918-1929

Table 5. Stillbirths (death of fetus 7-9 months old) and stillbirth rate (stillbirths pr 1 000 women 7-9 months pregnant) in Norway 1915-1923 [1]

	1915	*1916*	*1917*	*1918*	*1919*	*1920*	*1921*	*1922*	*1923*
Stillbirths	1 386	1 368	1 447	1 511	1 387	1 427	1 171	1 311	1 283
Stillbirth rate	19.7	18.7	19.0	20.2	18.9	17.5	15.2	17.5	17.6

[1] The pregnant population at risk (women 7-9 months pregnant) in 1915 is estimated as the sum of live and births January 1915 (women in their 9th month of pregnancy January 1915) to February 1916 (women in their 7th month of pregnancy December 1915), i.e. women who conceived April 1914 to May 1915. The pregnant population at risk in 1916 is estimated as the sum of live and stillbirths in January 1916 (women in their 9th month of pregnancy January 1916) to February 1917 (women in their 7th month of pregnancy December 1916), i.e. women who conceived April 1915 to May 1916, etc.
Source: SSB, 1920b, 1921a, 1921b, 1923a, 1935.

Table 6. Marriages dissolved by death (MDD) and marriages dissolved by death pr 1 000 existing marriages (including separated couples) in Norway 1913-1923

	1913	*1914*	*1915*	*1916*	*1917*	*1918*	*1919*	*1920*	*1921*	*1922*	*1923*
MDD	10 552	10 541	10 970	11 072	10 953	14 526	11 663	10 783	10 182	10 968	10 734
MDD pr 1 000 [1]	26.8	26.5	27.2	27.0	26.3	34.5	27.4	25.2	23.6	25.0	24.2

[1] The annual population at risks in non-census years are estimated adding contracted marriages and subtracting the number of divorces and marriages (including separated) dissolved by death between the censuses of 1910, 1920 and 1930. Net migration of married and separated couples is assumed to be zero.
Source: Mamelund, Brunborg and Noack, 1997

Table 7. Share of marriages dissolved by Spanish Influenza death of the wife or by the husband in Norway 1918

Age of			*Marriages dissolved by the death of*	
Wife	*Husband*	*Total (%)*[1]	*Husband (%)*	*Wife (%)*
15-19	**15-24**	0.94	0.69	0.25
20-24	**20-29**	0.95	0.56	0.39
25-29	**25-34**	0.98	0.52	0.45
30-34	**30-39**	0.70	0.43	0.28
35-39	**35-44**	0.64	0.33	0.31
40-44	**40-49**	0.35	0.22	0.14
45-49	**45-54**	0.36	0.20	0.16
50-54	**50-59**	0.28	0.16	0.12
55-59	**55-64**	0.29	0.16	0.14
60-64	**60-69**	0.35	0.15	0.20
65-69	**65-74**	0.43	0.19	0.24
70-74	**70-79**	0.49	0.26	0.23

[1]The total also includes very low shares caused by the death of both spouses, which are not assumed to have occurred simultaneously during Spanish Influenza from July to December 1918.
Source: The shares are calculated by the author using data from DCM 1922, and on the basis of several assumptions, see text.

Table 8. Results of linear regression analyses for the month with the largest downturn in CCR (LD) July-September 1918 and the downturn in CCR in the month with the lowest observed level of CCR (DB) July-September 1918

No	**Independent variables**	**Model 1** LD July-September 1918		**Model 2** DB July-September 1918	
		Coefficient	t-stat	Coefficient	t-stat
1 or 4	Downturn in CCR April-June 1918 (all three months)	-0.5978	-4.40***	-0.5119	-4.10***
2 or 5	Increase in CDR given LD (model 1) or DB (model 2) in CCR	-0.2200	-0.99	-0.0275	-0.17
3 or 6	Increase in IPI given LD (model 1) or DB (model 2) in CCR	-0.0063	-1.79*	-0.0071	-2.07**
	Constant	-10.7598	-6.09***	-10.7332	-5.95***
	N (rural and urban parts of counties)	37		37	
	Adjusted R^2	0.3761		0.3507	

* p < 0.10, ** p < 0.05, *** p < 0.01

Table 9. Results of linear regression analyses for the month with the largest downturn in CCR (LD) October-December 1918 and the downturn in CCR in the month with the lowest observed level of CCR (DB) October-December 1918

No	Independent variables	Model 3 LD October-December 1918		Model 4 DB October-December 1918	
		Coefficient	t-stat	Coefficient	t-stat
7 or 10	Downturn in CCR July-September 1918 (all three months)	-0.2238	-3.70***	-0.1941	-4.27***
8 or 11	Increase in CDR given LD (model 3) or DB (model 4) in CCR	-0.4581	-7.13***	-0.4918	-9.54***
9 or 12	Increase in IPI given LD (model 3) or DB (model 4) in CCR	0.0095	5.11***	0.0158	5.66***
	Constant	-8.0227	-5.80 ***	-8.9587	-8.29***
	N (rural and urban parts of counties)	37		37	
	Adjusted R^2	0.5860		0.7272	

* p < 0.10, ** p < 0.05, *** p < 0.01

Table 10. Results of linear regression analyses for rebound in CCR January-December 1919

No	Independent variables	Model 5		Model 6		Model 7	
		Coefficient	t-stat	Coefficient	t-stat	Coefficient	t-stat
13	Downturn in CCR April-December 1918	1.9937	6.98***	1.6863	6.07***	1.4613	5.59***
14	Increase in CDR July-December 1918			0.6868	2.12**	0.3749	1.21
15	Increase in IPI July-December 1918			0.0085	1.75*	0.0139	2.94***
16	Increase in CSR July-December 1918					-3.9104	-2.97***
	Constant	104.35	7.87***	35.1778	1.98*	32.3270	2.02*
	N (rural and urban parts of counties)	37		37		37	
	Adjusted R^2	0.5699		0.7323		0.7834	

p < 0.10, ** p < 0.05, *** p < 0.01

Table 11. Remarriages and remarriage rates of widowers and widows 1914-1924

Period	*Remarriages of widowers*	*Remarriage rates of widowers*	*Remarriages of widows*	*Remarriage rates of widows*
1914	1 267	28.1	567	5.7
1915	1 236	27.0	549	5.5
1916	1 323	28.4	598	5.9
1917	1 389	29.4	655	6.4
1918	1 436	29.9	653	6.3
1919	1 164	23.9	504	4.8
1920	1 562	31.6	721	6.8
1921	1 449	29.2	682	6.4
1922	1 332	26.8	670	6.3
1923	1 286	25.8	646	6.0
1924	1 280	25.6	581	5.4

1 The widowed and widower population at risk between the censuses of 1910, 1920 and 1930 is linearly interpolated assuming that net-migration of widowers/widows is zero.
Source: Regional population database (RD) of Statistics Norway, SSB, 1995.

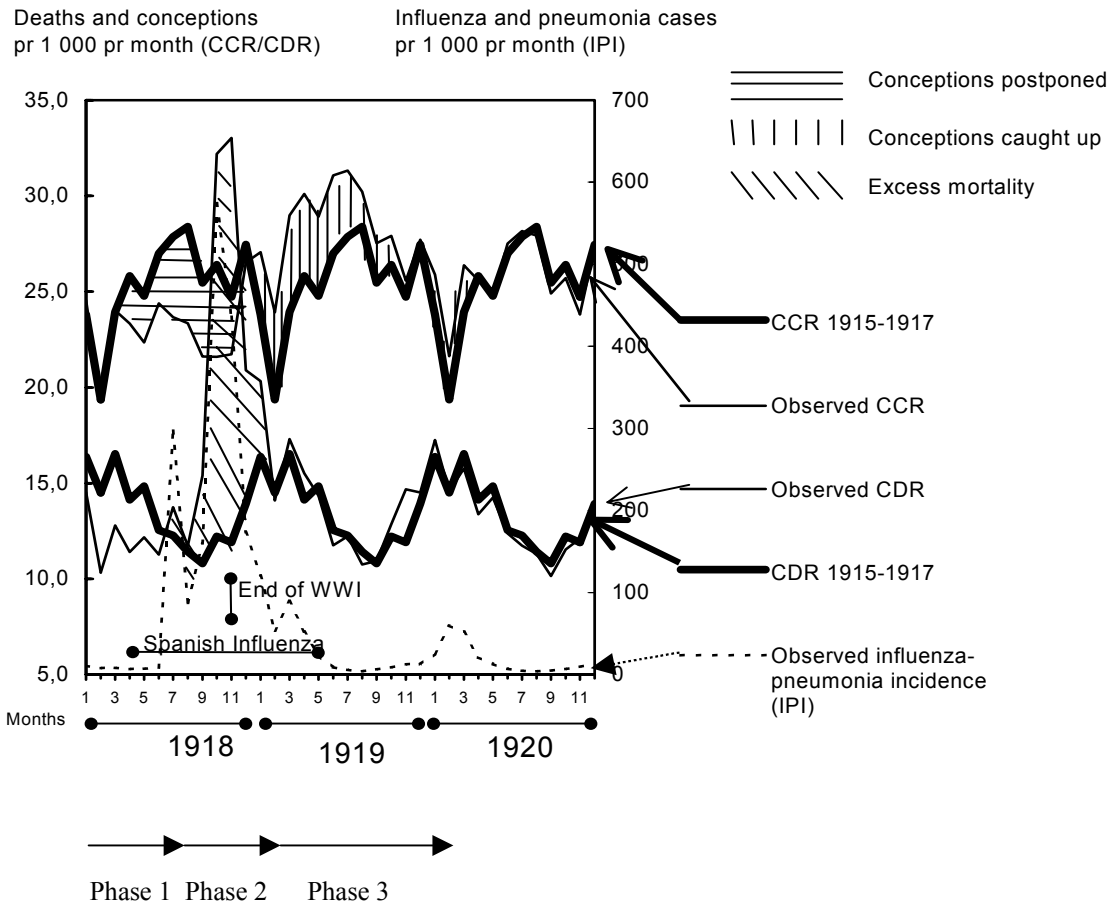

Figure 1. Monthly influenza and pneumonia incidence (IPI), crude death rate (CDR) and crude conception rate (CCR) in Norway 1915-1917 (average) and 1918-1920 (observed)

Sources: DCM, 1922, 1923, 1924; and SSB, 1920a, 1920b, 1921a, 1921b, 1923a, 1923b.

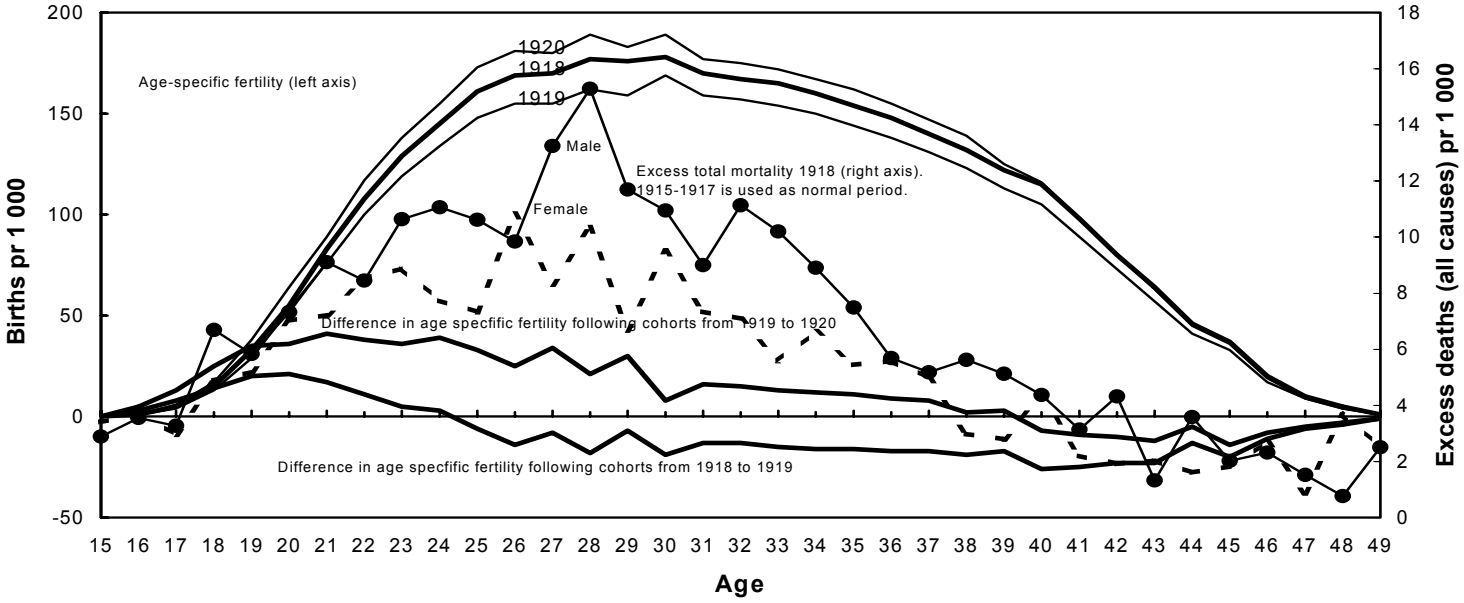

Figure 2. Age-specific period-fertility 1918-1920, the downturn and rebound of fertility following birth cohorts, and excess age-sex-specific period death rates 1918 (all causes) in Norway

Source: Calculated from Brunborg and Mamelund, 1994; Mamelund and Borgan, 1996.

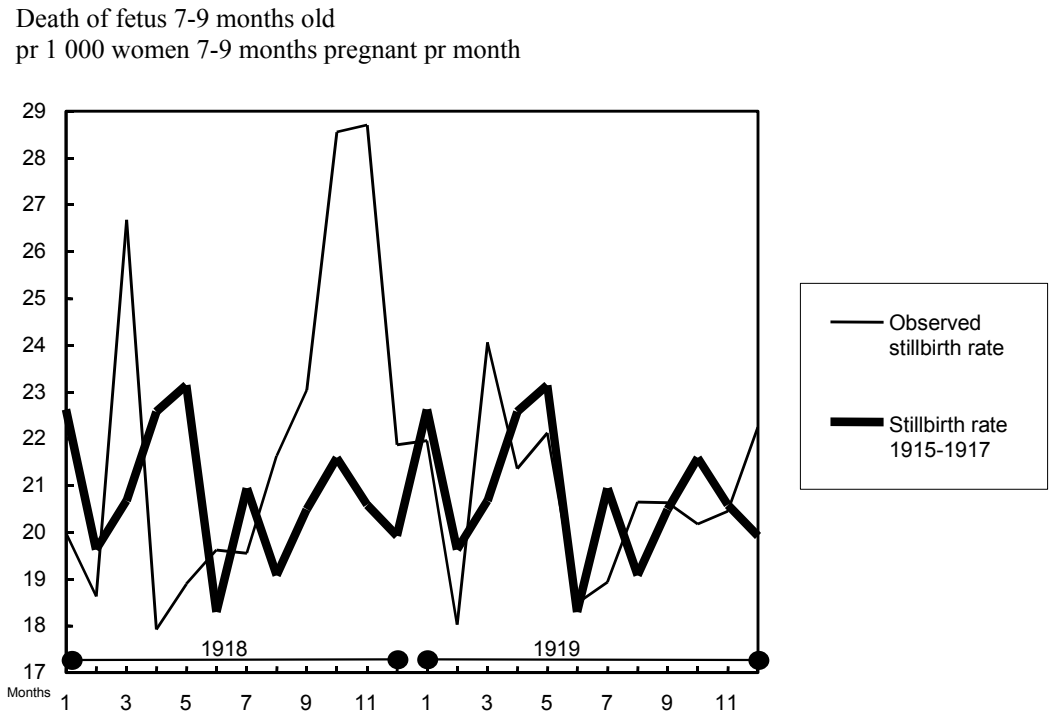

Figure 3. Monthly stillbirth rate in Norway 1918-1919 compared to the monthly average normal of 1915-1917 [1]

Source: SSB, 1920a, 1920b, 1921a, 1921b, 1923a, and 1923b

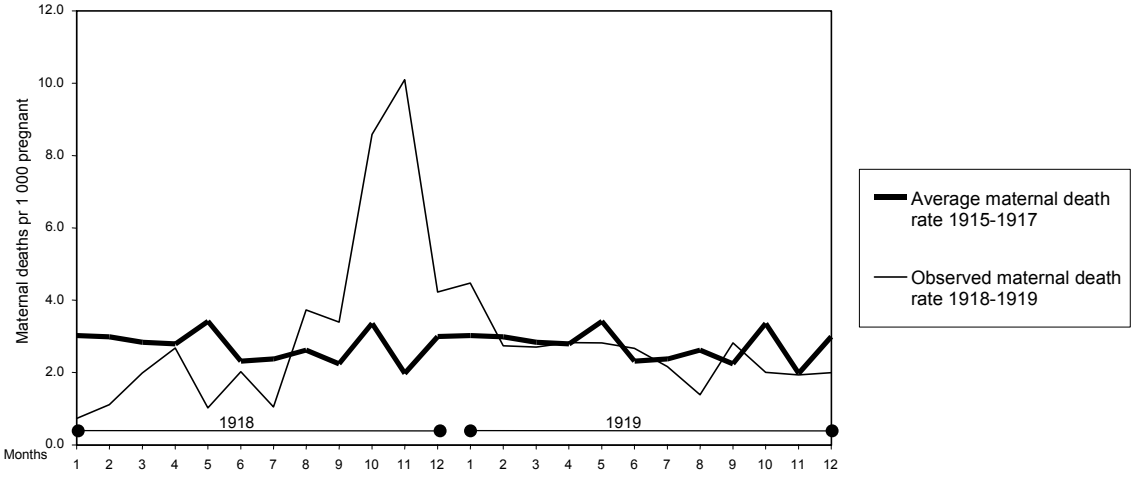

Figure 4. Monthly maternal death rates 1918-1919 compared to monthly average maternal death rate 1915-1917 in Norway [1]

Sources: DCM, 1918, 1920, 1921, 1922, and 1923