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ЧТО, ЕСЛИ ДЕМОГРАФИЧЕСКИЙ КРИЗИС В РОССИИ ИМЕЕТ СКРЫТУЮ ЭКОЛОГИЧЕСКУЮ ПРИЧИНУ?

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WHAT IF THE RUSSIAN DEMOGRAPHIC CRISIS HAD A HIDDEN ECOLOGIC CAUSE?

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Abstract. Russia death rates decline during the 1950s, rose from the mid-1960s until the early 2000s before declining slightly again after 2004. The trends have been attributed to economic trends and prospects, and alcohol consumption. Not-disregarding a contribution of social factors, a new idea is proposed here: that the observed trends in mortality (and fertility) could have resulted from an ecologic interplay between two populations: humans and influenza A viruses. It is proposed that the immune-inflammatory phenotype emerging from the interaction between early priming and re-infection by influenza A subtypes may be protective (if same subtype) or enhancer (if different subtypes) of morbidity upon challenges by other environmental exposures. Conclusions: The use of 1-year intervals to describe APC mortality trends both increases the amount of information available, thus enhancing the opportunities for patterns' recognition, and increases our capability of interpreting those patterns by describing trends across smaller intervals of time (period or birth-cohort). A preliminary analysis of the Russia mortality experience having this influenza hypothesis in mind is shown here, but it needs refinement based on better knowledge of demographics and influenza in Russia. Some patterns described here are similar to ones found in the US. One example is the increase in AIDS mortality among those born from 1947–1968, in periods dominated by H3 strains. Comparative analysis of mortality landscapes across countries may help us to straighten our record of past circulation of Influenza viruses and document associations between influenza recycling and mortality (and fertility) changes.

Keywords: Russia, Age-period-cohort trends, epidemic constitutions, fertility, influenza, mortality.

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Аннотация. В России уровень смертности снижался в течение 1950-х гг., увеличивался с середины 1960-х до начала 2000-х гг., прежде чем снова началось небольшое снижение после 2004 г. Эти тренды объяснялись влиянием тенденций и перспектив экономического развития, а также уровнем потребления алкоголя. Не отрицая влияния социальных факторов, в статье высказывается новая идея о том, что наблюдаемые тенденции в изменении уровня смертности (и рождаемости) могли быть вызваны экологическим взаимодействием двух популяций: человеческой и вируса гриппа типа А. Делается предположение, что иммуновоспалительный фенотип, возникающий в результате взаимодействия между первичным и повторным заражением подтипами гриппа А может снижать (если

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подтип один и тот же) или усиливать (если подтипы разные) заболеваемость в результате воздействия других внешних факторов. Выводы: Использование одногодичных интервалов для описания тенденций смертности из-за осложнений аденовирусной инфекции позволяет в равной мере увеличить количество доступной информации (тем самым повышая возможность признания паттерна) и увеличить наши возможности для интерпретации этих паттернов, описывая тренды для меньших временных интервалов (по возрастным группам). В статье показаны результаты предварительного анализа изменений уровня смертности в России (с учетом высказанной гипотезы о влиянии вируса гриппа), но они нуждаются в уточнении на основе более глубокого анализа демографических показателей и статистики, связанной с эпидемиями гриппа в России. Некоторые из описанных паттернов схожи с теми, которые были обнаружены в США. Один из примеров – увеличение смертности из-за ВИЧ-инфекции среди родившихся в 1947–1968 гг. в периоды, отмеченные преобладанием штаммов H3. Сравнительный анализ уровня смертности в разных странах может помочь нам уточнить наши данные о прошлых эпидемиях гриппа и зафиксировать связь между рециркуляцией гриппа и изменениями уровня смертности (и рождаемости).

Ключевые слова: Россия, Возрастно-периодические когортные тренды, эпидемии, рождаемость, грипп, смертность.

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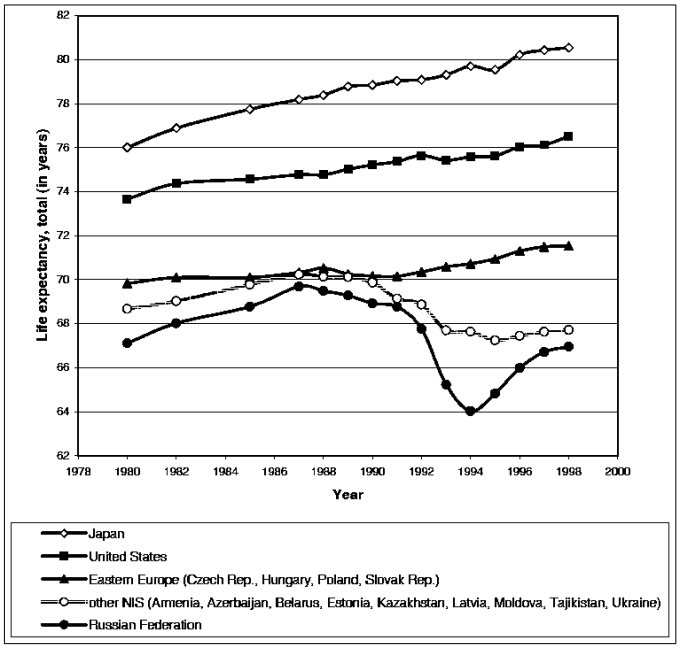
Introduction. During the last decades of the 20th century, most developed countries had significant increases in longevity, mostly attributed to declines in CHD mortality at middle and old ages (Azambuja 2009, Murphy 2009). Russia and other eastern European countries did not share the same pattern of change (fig. 1).

Figure 1



Longevity showed marked fluctuations, particularly among men (Fig. 2). The steepest fall occurred from 1990–1995, and it was accompanied by a significant fall in fertility. The period followed the end of the Soviet Union in 1989. In the late 1990s, several papers were published internationally about what was called a “Russia demographic crisis”. Epidemiologic studies identified violent deaths and cardiovascular diseases as the main underlying causes of the high mortality

rates recorded in 1992–1995, and suggested increased alcohol consumption[1] and disruption of health and social services as possible intermediary variables to a causal link between the political transition and the demographic changes.

Figure 2

The end of the Soviet Union was a good hypothesis to explain the 1990–1995 trends, but how to explain 1) the partial recovery, both of longevity and fertility, recorded from 1996–1999? 2) the new wave of rising rates of deaths from 2000–2005, particularly among working-age man?[2] 3) the fast recovery that has happened during the last decade? and 4) how to account for the earlier wave of demographic change that occurred in the 1980s?

According to VasiliVlasov (2015), contributor to a Lancet Paper on comparative global trends in longevity, "serious scientists don't have a solid explanation for it. It is a mystery to some extent"[3].

Since the first time that I heard about the "Russia demographic crisis" I was curious to look at the data to see if the theory that I had developed upon studying the 20th Century Coronary Heart Disease (CHD) mortality would apply to the Russia's case. Finally I had the opportunity. As a medical doctor and epidemiologist, during the last 30 years I have been interested in variations in diseases occurrences. My initial objective was, still in the 1980s, to explain the decline in CHD mortality. I believe that I found an explanation but, first I had to learn that 1) to get a different answer (because the existing ones were not satisfactory) you need to change your question; and 2) to change your question you need to "unlearn" what was conceived under particular circumstances and taken for granted ever since. And I have to convince people that I may be right. After a hypothesis finds its way to the mainstream – especially if it resonates with the general common sense, to dispute it may be very challenging. The CHD mortality case has similarities with the Russia's case that deserve to be considered.

The CHD mortality trend. Time. One of the first things that I learned and I believe that occurs with the Russia's case was that *how we interpret temporal trends depends on when we look at them.* And here I see one thing in common between attempts of interpreting the rise in CHD

[1] Leon, D, Chenet, L., Shkolnikov, V.M. et al. (1997). Huge Variation in Russian Mortality Rates 1984–94: Artefact, Alcohol, or What? In *The Lancet*. Vol. 350, Iss. 9075, pp. 383–388.

[2] Malysheva, Ye. Live longer! Putin Calls for Improving Life Expectancy. In *Russia Beyond the Headlines.* Available at: URL: https://www.rbth.com/politics_and_society/2017/03/24/live-longer-putin-calls-for-improving-life-expectancy_726283 (date of access: 10.06.2023).

[3] «The Moscow Times». Available at: URL: https://themoscowtimes.com/news/why-is-russias-growth-in-life-expectancy-slowing-49224 (18+ настоящий материал (информация) произведен и (или) распространен иностранным агентом интернет-издание «The Moscow Times», либо касается деятельности иностранного агента интернет-издание «The Moscow Times»).

mortality and the decline in Russia's longevity and fertility…: hypotheses were developed before the complete cycle had presented itself.

CHD mortality became to be acknowledged as a public health problem in developed countries after the WW2. In the US, it grew steeply from the 1940s to the 1960s, being, in the mid-1960s, responsible for 30 % of the total CHD mortality. Similar trends were taking place in many other developed countries. In the US, the first epidemiologic study commissioned to find the determinants of this *"chronic, degenerative, non-communicable disease"* was the Framingham Heart Study. The research group was constituted by cardiologists with no relevant knowledge of epidemiology, in fact, a discipline that had lost its relevance after the 1930s, as the causal studies on epidemic diseases became centered on infectious agents and the quest for new medicines and vaccines, all done in laboratories. This new generation of cardiovascular diseases epidemiologists, most of them originally medical doctors, naturally assumed that individuals and whatever they founded that would differ among them and be associated with developing CHD, like high serum cholesterol and high blood pressure, would be the answers they needed about causality. Common sense informed all that those were times of economic abundance and rise of a modern urban way of life, which favored smoking and high-fat diets in an environment more stressful and less demanding in terms of physical exercise. But that's not enough. After 1968, and more intensively after 1972, a decline as steep as the previous rise took place in the US, followed by other countries. From 1970 to 2000, CHD mortality had fallen 60 % in the US!

I entered the field in the early 1980s, when several international conferences were being held to explain the decline. I was looking at the CHD mortality trend for the first time then, and it seemed evident to me that the narrative that had been developed to explain the rise could not account for the decline. So, we needed a new way of looking at the problem. But the point that I wanted to stress here is that my consideration would only be possible after the beginning of the decline! The same would be the case with Russia's demographic trend.

Different answers require different questions. Looking at the CHD mortality trend for the first time at the 1980s, I did not see a rise and than a decline, I saw a true epidemic! That change open a window to a new question. What caused the epidemic wave? Epidemics depend on changes on an environmental exposure but also on changes on the population vulnerability to the exposure. A measles epidemics decay because the number of vulnerable individuals is too low to sustain the circulation of the agent. Our dominant causal model does not contemplate vulnerability, because 1- biologically, regarding chronic diseases, we still emphasize genetic inheritance when dealing with differences in vulnerability, and genetics cannot account for short-term changes in trends; and 2- because if it is not genetic, we still don't know how to profile it in individuals. This is why we need population studies.

According to Stephen Jay Good and Richard Lewontin[4] we can look at population occurrences changing over time with Platonic or Darwinian eyes. The Platonic way assumes that the population remains unchanged, and that variations in mortality and fertility would depend on effects of concurrent exposures. The Darwinian way admits that the population also changes over time. And that changes in trends may mean, not that the exposure or its effect is changing, but that the relative composition of the population is varying while the effects of the exposure remain the same in each sub-population. This is what my work is about.

The blinding strength of paradigms. Which hypothesis could be sought to explain a CHD epidemic in the 1980s? The 1960s – 1980s CHD epidemiology established the pattern to our way of thinking about causation. So, it is important to retrospectively review how many things were (and continue to be) just "assumed": 1- that CHD was a non-infectious, degenerative condition; 2) that the right place to look for answers were the individuals; 3) that differences on environmental exposures (smoking, high-fat diets) and on constitutional traits (high cholesterol levels, high blood pressure) between individuals who developed and did not develop CHD would imply causation ("risk factors" amenable to treatment (like high serum cholesterol treated with cholesterol lowering

[4] Azambuja, M.I., Levins, R. (2007). Coronary Heart Disease (CHC) – One or Several Diseases? Changes in the prevalence and features of CHD. In *Perspectives in Biology and Medicine.* Vol. 50, No. 2, pp. 228–242.

drugs, for example), instead of “factors of risk” or indicatives of vulnerability, as originally conceived[5]; 4) that whatever were identified as cause of the cases, based on differences between cases and non-cases, could be extrapolated to explain mortality trends – notwithstanding the fact that a few (Reul Stallones, Geoffrey Rose, Henry Blackburn), in the 1980s, had defended that causes of cases and causes of occurrences might not be the same.

In the late 1980s I discovered that paradigms may completely blind us. Almost 30 years after the first results of the Framingham studies identifying high serum cholesterol, smoking and hypertension as the main “ risk factors” to CHD, we lived so immerse within the degenerative idea and the diet-heart paradigm that we did not even perceived them as a created narrative anymore. They had become incontestable. So, I spent some years seeking for causes of variations in population vulnerability that could explain an epidemic presentation of CHD within the realm of the degenerative paradigm, like, for example, a negative selection of the fittest by the world wars. Until I read, in 1990, a 1988 Circulation’s Editorial called “The potential role of viruses in the pathogenesis of atherosclerosis”[6]. I was shocked! How could I have never thought of it, even knowing that I was dealing with an epidemic?

The hypothesis. My approach consisted of using the best method developed to investigate epidemics (not cases!): the Epidemiologic Inquiry: a descriptive study of the event according to time, person and place plus external knowledge to be explored upon inductive reasoning: from the data to a hypothesis, biologic reasoning to evaluate consistence, and if possible, reproduction in other settings.

Which infectious event preceded the beginning of the CHD mortality rise, was as huge and worldwide, and affected mostly men and whites born around the turn to the 20th Century? In 1994 I presented, for the first time, the hypothesis of a *birth-cohort* association between 1918 influenza mortality and the 20th Century. CHD epidemic.

Since the late 1990s I have been proposing that we humans have co-evolved with Influenza A viruses (ubiquitous agents). Both, the human and the viral populations, select each other. The expression of this selection upon the influenza A population is the recycling of Influenza subtypes (H1N1, H2N2, H3N2)[7] . And I propose that the expression of this selection upon us is the secular variation in prevailing diseases, mortality and fertility. A phenomenon identified in 1953 and described in 1969 – *the original antigenic sin*[8] – would explain heterosubtypic immune responses that would induce inflammation and disease[9]. Biologically, our immune-inflammatory phenotypes are continuously modulated by successive interactions between early priming and re-infections by influenza A subtypes. This successive Cohort X Period interactions would result in phenotypes more protective (if same subtype) or enhancer (if different subtypes) of immune-pathologic responses upon challenges by other contextual factors.

I have explored this hypothesis with graphic displays of age-period-cohort variations in mortality by one-year intervals (Azambuja, BAJ 2009). This methodology has uncovered very interesting period and cohort (or both) effects on countries’ mortality landscapes, frequently coinciding with years of occurrence of influenza epidemics.

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[5] Oppenheimer, G.M. (2006). Profiling Risk: The Emergence of Coronary Heart Disease Epidemiology in the United States (1947–70). In *International Journal of Epidemiology*. 2006. Vol. 35, Iss. 3, pp. 720–730.

[6] Cunningan, M.J., Parternack, R.C. (1988). The Potential Role of Viruses in the Pathogenesis of Atherosclerosis. In *Circulation.* Vol. 77, pp. 964–966.

[7] Influenza recycling is the re-introduction, in the population, of subtypes of the influenza A virus antigenically similar to viruses that circulated in the past. The antibody spectrum of the human population would pressure for change, and a limited number of viable variations would favor the return of old strains.

[8] “The major antigens of the influenza strains of first infection of childhood permanently orient the antibody-forming mechanisms so that, on subsequent exposures, the cohort of the population would respond with marked reinforcement of the primary antibody” Davenport et al, 1969.

[9] Chen, H.D. et al. (2003). Specific History of Heterologous Virus Infections Determines Anti-Viral Immunity and Immunopathology in the Lung. In *The American Journal of Pathology*. Vol. 163. No. 4, pp. 1341–1355; Thomas, P.G. et al. (2007). Hidden Epitopes Emerge in Secondary Influenza Virus-Specific CD8+ T Cell Reponses. In *The Journal of Immunology.* Vol. 178. No. 5, pp. 3091–3098.

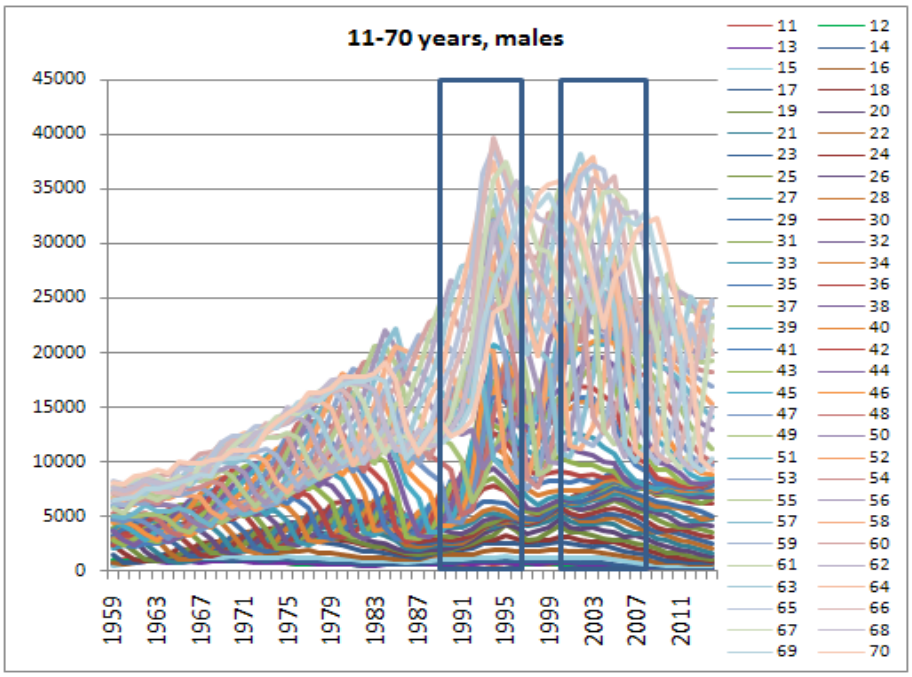
I had never worked with Russia's data, and I wanted to see if I my ideas would apply in this case.

Methods. Data used here (numbers of deaths, population, live births, period 1959–2014) were retrieved from the Human Mortality Database. Descriptive analysis will be based on graphic plotting of mortality by one-year intervals of age, period or cohort. Influenza information was obtained from the literature.

Results. Figures 1 and 2 show the evolution of the numbers of male deaths in Russia during the period 1959–2014, according to calendar years (1) and respective birth-cohorts (2), by 1-year intervals.

Figure 1

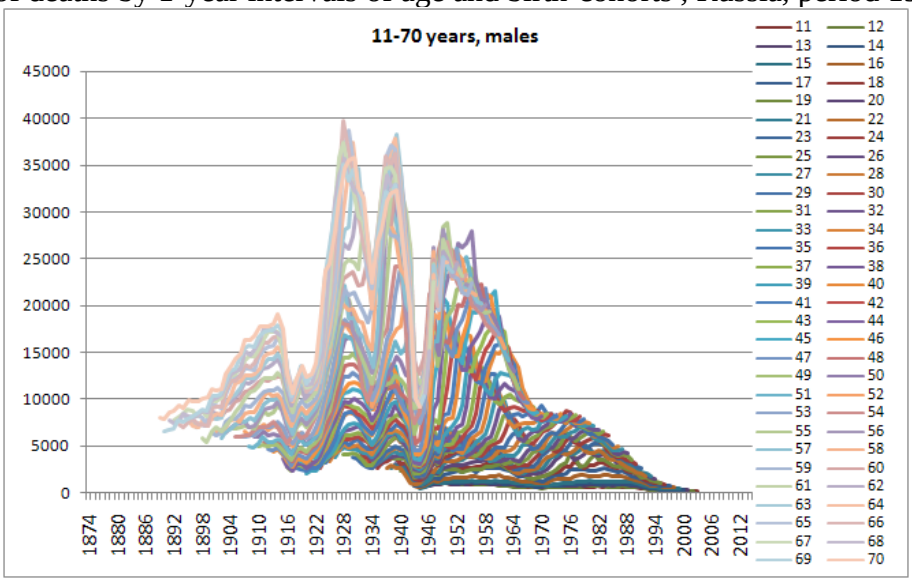
Number of deaths by 1-year intervals of age and calendar years, Russia, Period 1959, 2014



Source of data: Human mortality database (HMD)

Figure 2

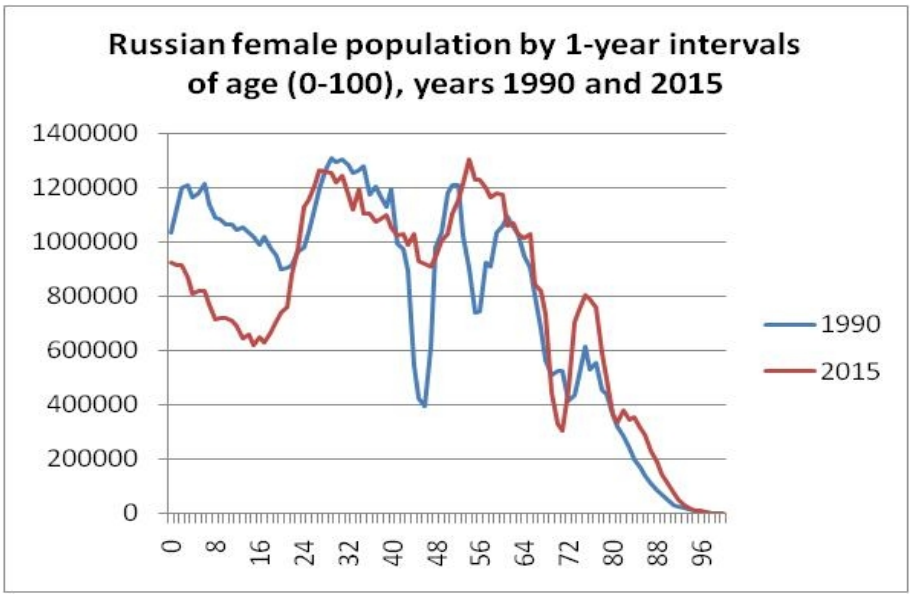
Number of deaths by 1-year intervals of age and birth-cohorts , Russia, period 1959, 2014



The display of trends of the yearly number of deaths by 1-year of age (fig. 1) is messy, and it is easy to understand why when we look at the age x cohort figure (2). The numbers of deaths

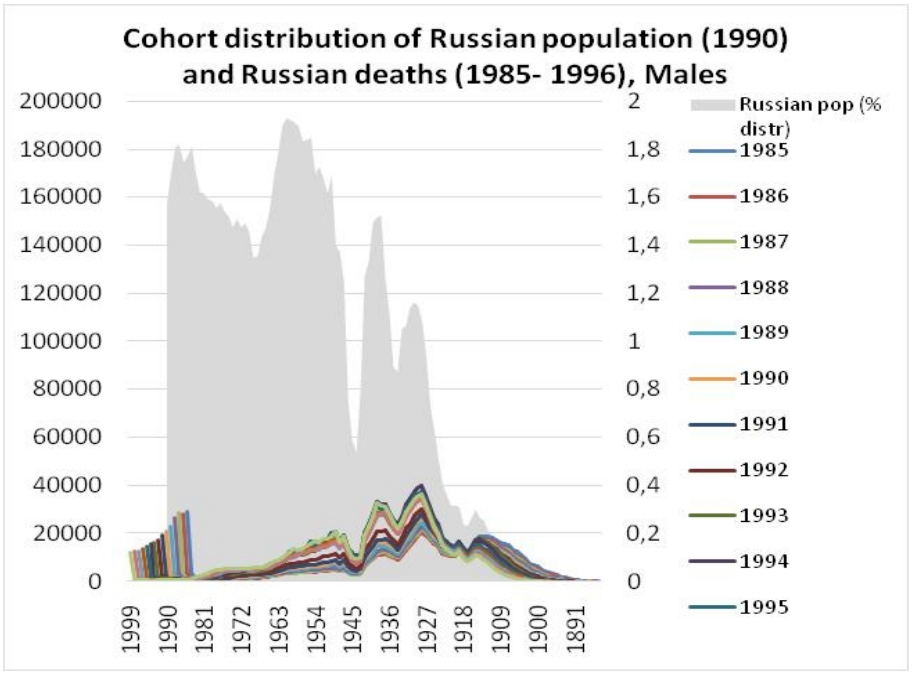
by age strongly depend on population sizes by age (Fig. 4), and Russia, comparatively to the United States for example, suffered huge losses of population (particularly of males) during the world wars. These losses had a wavelength effect on sizes of future generations that continues to date, amplified during the period 1990–1995 (Fig. 3).

Figure 3



Source: HMD

Figure 4



Source: HMD

Using population rates to describe trends by 1-year intervals would eliminate the variations due to the birth-cohort sizes, assuming good estimates of population sizes by 1-year intervals of age and calendar years/ (birth-cohorts) – a challenge, especially in cases like Russia's, with huge variations in cohort sizes – Fig. 5). Less than very good estimates may not completely remove cohort variations or may distort them, and blur localized period and cohort changes perceived with crude numbers of deaths. Death-rates produced with population estimates available at the HMD (Fig. 5) seem to represent very well the variations in numbers of deaths (Fig. 6 and 7). Localized increases in numbers of deaths in a sequence of ages, in 1985, 1988, 1993–94 and 2004, persist when data is presented as death-rates. As expected, when the size of the population at risk is considered, the

comparative size of the mortality across the age-cohort ranges change, like we see at ages 32 and 33 in 1979. The peak observed in 1979 would require more investigation.Considering the good quality of the HMD population estimates and the advantage of removing the spurious effect of different birth-cohort sizes by the use of death-rates, Fig. 8 presents temporal trends of mortality (death-rates) in Russia in the period 1959–2014, displayed as Age x Period and Age X Birth-Cohorts plots.

Figure 5

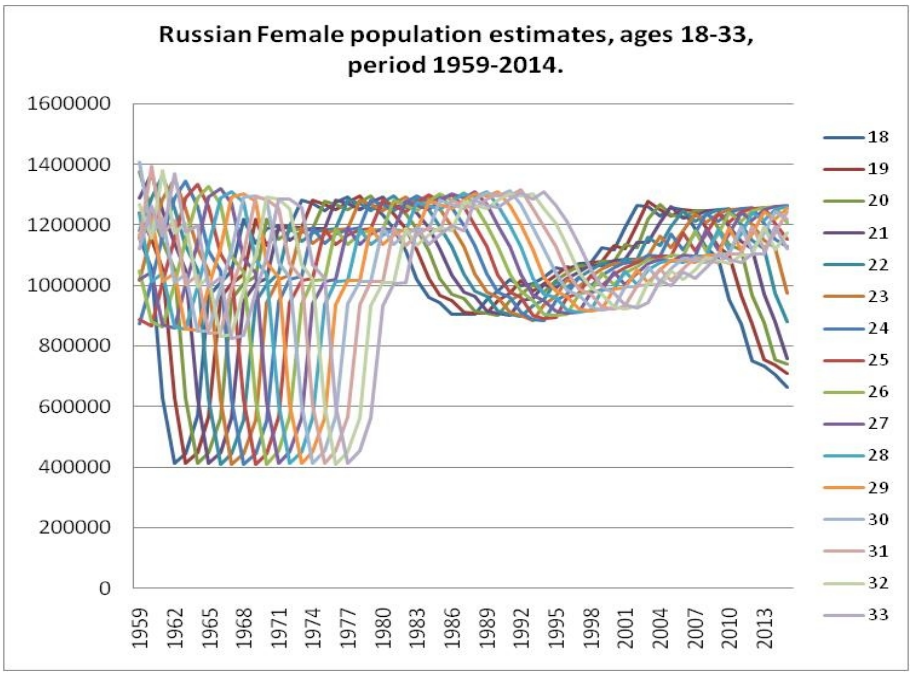


Figure 6

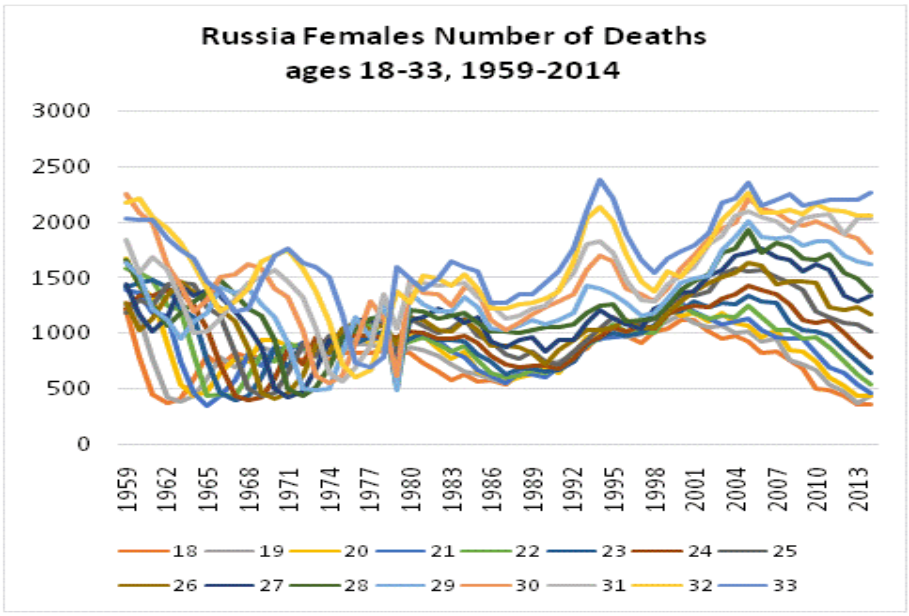


Fig. 8 shows Russia's mortality (death-rates) by 1-year intervals of age and calendar time, ages 5–80, years 1959–2014. It gives additional information.

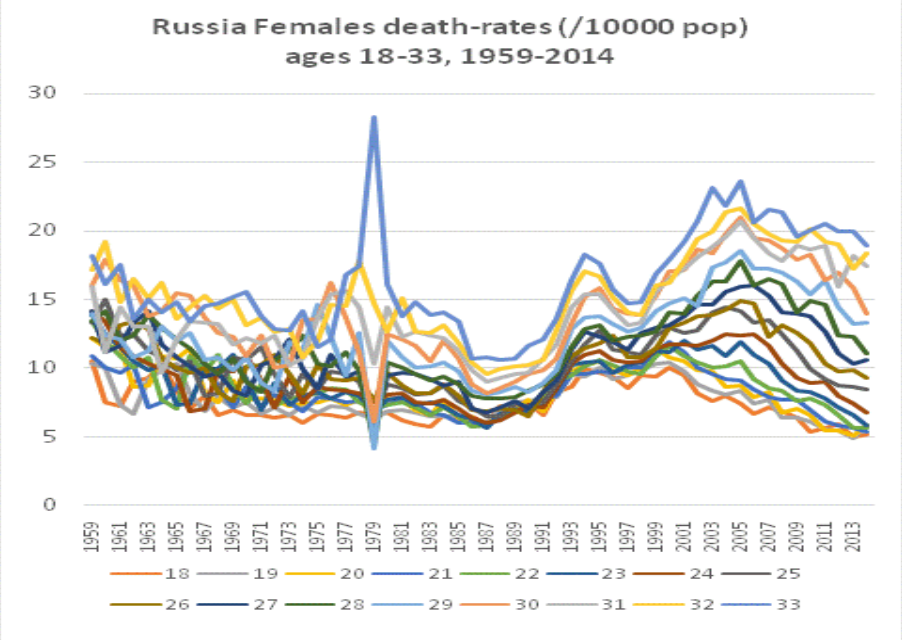
Color bands indicate the influenza A subtypes circulating by the time of the individuals'birth and at the upper area of the grafic indicates the subtypes circulating by the time of the individuals' deaths.

The yellow band encompasses the first documented period of circulation of the H1N1 subtype, suposedly initiated by the 1918 Influenza Pandemic and ended in 1957. There are two visible tranverse marks within the period, one corresponding to the 1935–1936 birth-cohorts (period of an H1N1 influenza epidemic), and the other corresponding to the cohorts born around 1942–

1948, which includes the WWII years and the immediate post-war, plus the 1947 H1N1 Influenza epidemic.

The pink band corresponds to birth cohorts initially exposed to the H2N2 subtype, from 1958 to 1968, and the light green band corresponds to the cohorts originally primed by H3N2 virus, exclusively – the ones born from 1969 to 1977 – or either by H3 or by H1 viruses (after that).

Figure 7



The red (H3) and blue (H1) letters are to show the association suggested in this paper: that rises in mortality were associated with years of preponderant H3 circulation among cohorts non-H3. And that declines occurred in years of H1 circulation (1986–88, 1996–98) in H1 primed birth cohorts. Co-circulation, as may have happened at various periods after 1979, may have resulted in highest mortalities among H2 birth-cohorts, but probably affected them all: H3 re-infected /H1 Primed and H1 reinfected/ H3 primed. Further analysis would be required.

Figure 8

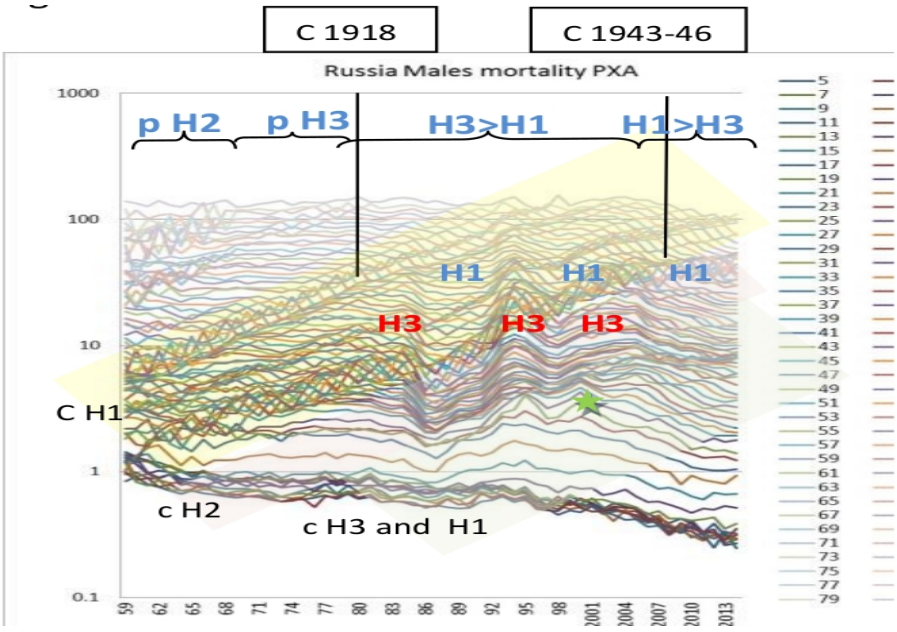
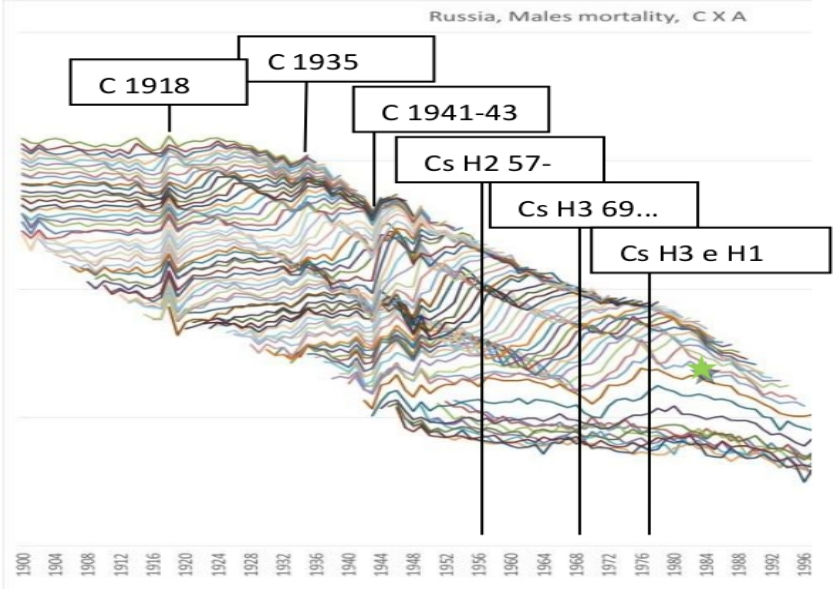


Figure 9



Fertility. As already mentioned, Russia suffered a huge loss of population during the world wars and its effect propagated in waves over the century.

Fertility directly depends on the number of women at reproductive ages. Of course there are several social and economic factors capable of changing fertility rates.

But maybe we do not consider enough infection and its potential effect upon the fetal viability. Fertylity rates vary with seasons of the year. Would this be just the result of opportunities varying with the seasons? Or Influenza might be implied? Increase in the number of premature deliveries and abortions was documented in 1918 (Azambuja, BAJ 2009). And after 2009 we all became more aware of the effects of influenza infection upon the mother and the pregancy. The Zika also increased general awarness regarding fetal infections.

Figure 10

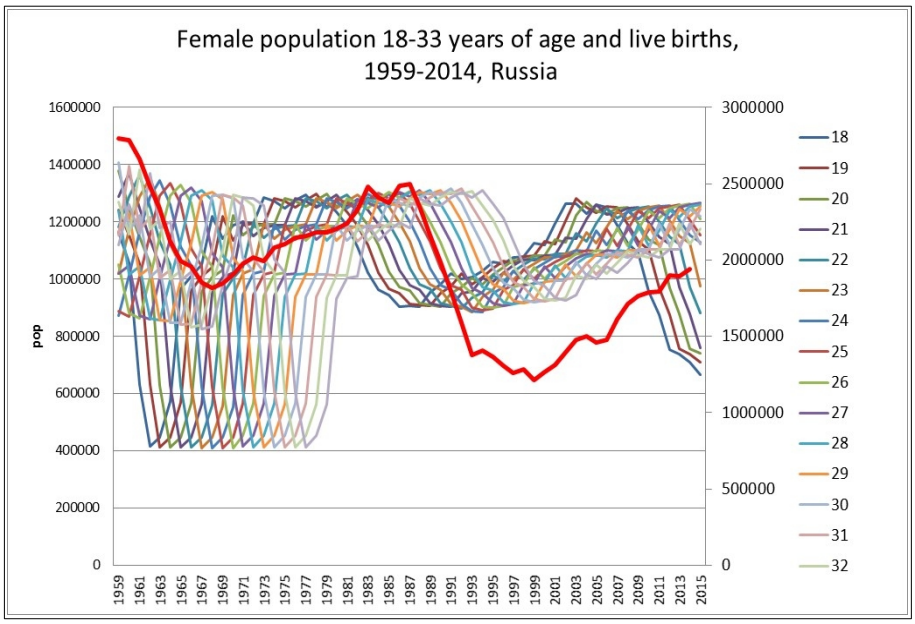


Figure 10 displays the female populations of Russia at ages 18-33, and the number of live births in the same period. A good correlation exists, as expected, but the cohort variation in the number of potential mothers does not seem enough to explain the decline in fertility seen in the period 1990–1995.

Figure 11

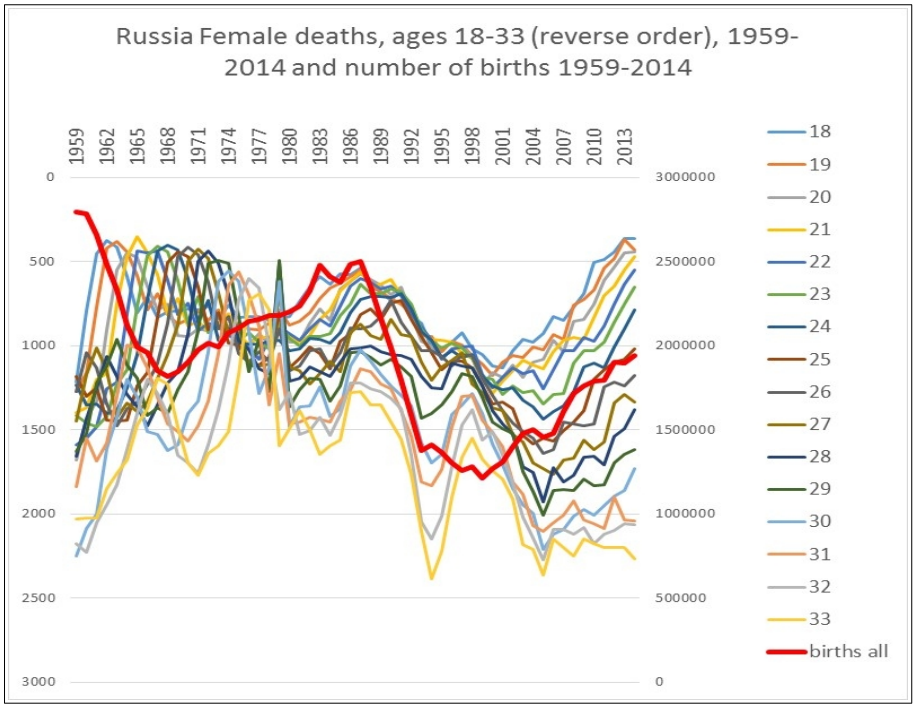


Fig. 11 shows the number of deaths of women 18-33 (presented as its inverse) and the number of live births for the period 1959–2014, in Russia. The rational is that mortality would be indicative of the expected number of severe diseases , by its turn expected to be related with the discordant subtypes met at the year of birth and the year of death.

The figure shows that when the number of deaths of women at reproductive ages increases, the number of births decreases (Number of deaths are presented in reverse order to facilitate the visualization). It seems that the decline in fertility correlates well with the increase in mortality during the period 1990–95, especialy of women born during the H2 era. (1960s).

More investigation is required.

Discussion. The analysis presented here suggests that there is a role for influenza in the demographic variations seen in Russia during the last 50 years.

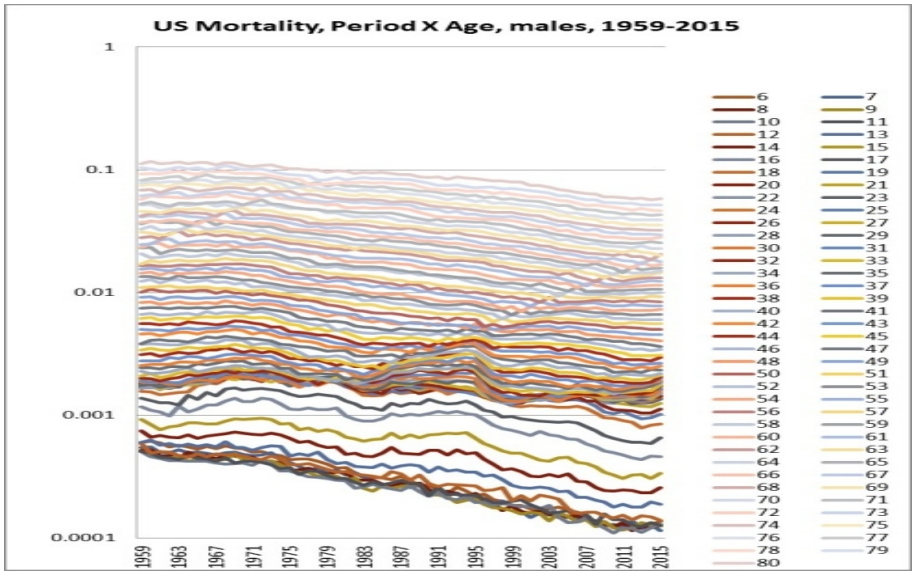
To reinforce the argument, Fig 11 shows the evolution of the death-rates of the US population, during the same period. There are similarities and differences.

During the period 1990–1995 period, like in Russia, mortality increased in US cohorts, particularly among those born after 1947 (H1 until 1957 and H2 until 1968), and it also fell in 1996. In both countries, the H1 virus made a reapperance in 1996, after some years of apparent absence[10]. In the US, the rise in mortality was not as great and it does not seem to have affect as much the cohorts born during the period 1918–1947. Like in Russia, after a decline in 1996–1997 mortality increased again towars 2003-4, declined, and unlike Russia, it seems to be increasing once more. The figure also shows a rise in mortality during the period H2 (1960s) that affected cohorts born after and before 1918.

Differences in the size of the effect, with much higher rates in Russia, possibly resulted from the contextual situation of relative social disruption. But it seems that a background of increased vulnerabulity to CVD (Congestive Heart Failure) and Mental disorders possibly due to immunopathologic effects of influenza, might be operating.

[10] Ivanova, E.T. et al. (2000). Variability and Prevalence. Characteristics of Influenza A Virus (H1N1) in Period 1990–1998. In *Voprosy Virusologii.* Vol. 45, No. 5, pp. 18–22.
Brammer, T.L. et al. (2000). Surveillance for Influenza – United States 1994–95, 1995-96 and 1996-97 Seasons. In *MMWR.* Vol. 49, No. 3, pp. 13–28.

Figure 11



This is still a provisional work and I would like very much to hear criticisms to it.

If this hypothesis is correct, the change that we are seeing in patterns of diseases occurrence, particularly among those with less than 55 years of age, suggests changes in the relative constitution of the population, as H1 primed individuals born at the beginning of the 20th century are dying and being substituted by subpopulations primed by H2 and H3 subtypes. It may be possible to predict a new epidemiologic transition whose direction will depend on the influenza viruses that will predominate during the next years.

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