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MAJOR ARTICLE

A Missed Summer Wave of the 1918–1919 Influenza Pandemic: Evidence From Household Surveys in the United States and Norway

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Background. Reanalysis of influenza survey data from 1918 to 1919 was done to obtain new insights into the geographic and host factors responsible for the various waves.

Methods. We analyzed the age- and sex-specific influenza morbidity, fatality, and mortality for the city of Baltimore and smaller towns and rural areas of Maryland and the city of Bergen (Norway), using survey data. The Maryland surveys captured the 1918 fall wave, whereas the Bergen survey captured 3 waves during 1918–1919.

Results. Morbidity in rural areas of Maryland was higher than in the city of Baltimore during the fall of 1918, that was almost equal to that in Bergen during the summer of 1918. In Bergen, the morbidity in the fall was only half of that in the summer, with more females than males just above the age of 20 falling ill, as seen in both regions of Maryland. In contrast, more males than females fell ill during the summer wave in Bergen. Individuals <40 years had the highest morbidity, whereas school-aged children had the lowest fatality and mortality.

Conclusion. A previously unrecognized pandemic summer wave may have hit the 2 regions of Maryland in 1918.

Keywords. fatality; gender; morbidity; Spanish influenza; vitamin D.

The so-called "Spanish Influenza" pandemic appeared in several waves in 1918 through 1920. Not all societies experienced all of them nor at the same time. In the Northern hemisphere, recent literature has documented prepandemic herald waves in both the United States and Norway during the spring of 1918 (February–April). This wave was mild with low mortality; it often started in military populations and did not always spread to the civil populations [1–6].

The first distinct and recognized pandemic bout in both military and civilian populations occurred during the summer of 1918 (May–September). At that time, morbidity was often very high but with relatively low fatality. Morbidity also occurred in mostly urban areas in countries well connected to international transport, whereas rural areas and isolated areas often escaped the pandemic. The second distinct influenza wave occurred during the fall of 1918 (October–December) and affected urban, rural, and isolated communities with higher fatality rates. In many communities, a third but less severe bout appeared during the winter of 1919 (January–March 1919). Some isolated areas that were not affected in 1918 or 1919 experience their first encounter with the pandemic in a fourth bout during the winter of 1920, often with devastating consequences [7]. Although it is established that the H1N1 pandemic virus was responsible for both the summer and fall waves in the United States in 1918 [8], it is still unclear whether the herald wave during the spring of 1918 was caused by the pandemic virus or whether it was caused by a seasonal influenza or another endemic virus [6, 9].

In recent studies of the 1918–1919 pandemic, mortality data were used to document the age-specific severity, transmission, and wave-like behavior of this disease [10–14]. Although historical survey data from Maryland [15] were recently used to determine the overall transmission rates [16–18], they have not been reanalyzed to study the details of age- and sex-specific morbidity nor fatality and mortality related to influenza-like illness (ILI).

In this study, we analyze the age- and sex-specific pattern of morbidity and mortality at various communities in the state of Maryland [15] and the city of Bergen (Norway) [19] using household survey data. The survey from Bergen is less well known, and the data have not been analyzed in detail. One advantage of the Bergen survey is that data are available for the summer, fall, and winter outbreaks, respectively, whereas the data from Maryland are only available for the fall outbreak. However, none of the surveys captures possible herald waves

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in the spring of 1918 or recurrences of the outbreak in the winter of 1920. Our reanalysis of survey data from the 2 countries in concert provides insight into the geographic and host factors responsible for the age- and sex-specific pattern of morbidity, fatality, and mortality by wave.

METHODS

The age- and sex-specific morbidity, fatality, and mortality rates from ILI per 100 population (in %) were calculated using the aggregate-level household survey data from 2 of 18 canvassed areas in the United States: the city of Baltimore and some towns and rural communities in Maryland [15] and similar survey data from the city of Bergen, Norway [19]. A statistically significant difference in the outcomes by subgroups were estimated using a one-sided z-test with alpha = 5%, ie, $z \geq 1.65$ (when alpha is 10%, 1%, or 0.1%, then the z-values are $\geq$1.29, $\geq$2.33, and $\geq$3.12, respectively).

The data from Maryland were collected from November 20, 1918 to December 15, 1918, covering only the fall wave from September 1, 1918 to November 30, 1918, whereas the Bergen data were collected from the end of 1918 to the end of 1919, covering all 3 waves of the pandemic there (July–September 1918, October–December 1918, and January–March 1919). To our knowledge, the Bergen survey may be the only one with information on morbidity and mortality before and after the main pandemic wave in the fall of 1918. Because the 1918 spring wave was not anticipated and produced relatively mild disease, it often went unnoticed and was not included in any retrospective survey.

There are 2 reasons we only considered the US survey data for the city of Baltimore and the data for 4 smaller towns and 3 rural communities in Maryland ([15]; see footnote to Table 1). First, the raw event and population data needed to recalculate the age- and sex-specific morbidity and mortality rates for the fall wave were only published for the 2 Maryland communities. Second, the Maryland data also gave us an opportunity to study urban-rural differences.

The canvassed areas were selected at random in both countries to make the samples representative for the population with respect to demographic variables and the impact of the pandemic upon morbidity and mortality. One of the most important factors related to both morbidity and mortality, which are observable for both the samples and the populations, is the age distributions; the sample age-distributions did not deviate from the age distributions in the population of the 3 canvassed areas [15, 19]. The overall sample mortality rate was equal to the overall population mortality rate in Bergen [19], whereas the overall sample mortality rate was slightly lower than the population mortality rate in Maryland [20, 21].

A potential weakness of using survey data to analyze the epidemiology of the 1918–1919 pandemic is when small subgroups are studied, for example, mortality or fatality rates by age, sex, and wave. In our analysis, none of the differences in fatality and mortality rates for any subgroup were statistically significant because of a low number of sample deaths. Because the chief feature of pandemics is much higher morbidity than during seasonal influenza epidemics, finding statistically significant differences in morbidity rates by subgroups, using sample survey data, are not a problem. Although official population records are a preferable source for documenting variation in 1918–1919 pandemic mortality rates (percentage of a population dying from influenza and pneumonia), survey data are needed to estimate reliable morbidity and fatality rates. Fewer people might see a doctor in mild compared with severe disease outbreaks. Therefore, survey data may better capture the actual magnitude of mild outbreaks than routine notification data. A comparison of survey data and routine notification data for Bergen confirms this hypothesis. Although the number of persons seeing a doctor for an ILI during the mild summer wave (as measured from routine notification data) was only one third of new ILI cases as reported in the survey, there were no differences in the number of ILI cases in the 2 sources of data during the more lethal 1918 fall wave [19].

The ILI cases and deaths in Maryland and Bergen were self-reported to the data collectors, usually by a homemaker, and were of course not laboratory-confirmed at the time. The age-specific data for the Maryland communities were published for

Table 1. Demographic and Survey Information for the City of Baltimore and Some Smaller Towns and Rural Areas of Maryland and for the City of Bergen, Norway

Community	Wave	Population Size	Sample Size (% of Population)	ILI Cases (% of Sample)	Fatalities (% of Cases)	Mortality (% of Sample)
Maryland, City of Baltimore	Wave II	594 639	33 776 (5.68%)	7868 (23.29%)	156 (1.98%)	156 (0.46%)
Towns/rural areas[a]	Wave II	46 154	12 759 (27.64%)	5169 (40.51%)	87 (1.68%)	87 (0.68%)
City of Bergen, Norway	Waves I, II and III	90 275	10 633 (11.77%)	4818 (45.31%)	72 (1.49%)	72 (0.68%)
	Wave I			2739 (25.76%)	27 (0.99%)	27 (0.25%)
	Wave II			1408 (13.24%)	31 (2.20%)	31 (0.29%)
	Wave III			671 (6.31%)	14 (2.09%)	14 (0.13%)

Abbreviation: ILI, influenza-like illness.

[a] This sample includes inhabitants surveyed in the towns of Cumberland (n = 5234), Frederick (n = 2420), Salisbury (n = 1735), Lonaconing (n = 1840), and 3 rural communities in Frederick, Washington, and Wicomico Counties (n = 1530).

5-year age intervals (<1, 1–4, 5–9, . . . 70–74, 75+), and similar data for Bergen were published for 10-year age intervals (0–9, . . . 60–69, 70+). Other demographic and survey information are listed in Table 1.

RESULTS

Almost 1 in 4 samples in Baltimore reported ILI during the second wave of the disease, whereas 4 in 10 in the smaller towns and rural areas of Maryland had ILI during that same period (Table 1), ie, significantly higher morbidity than in the city of Baltimore (at 0.1% level, z = 16.57). On the other hand, in the city of Bergen, approximately half of the samples reportedly had ILI at one time from July 1918 to March 1919, covering all 3 waves of the disease (Table 1). The ILI rate in Bergen was highest during the first wave, followed by successively lower rates during the second and third waves, and was significantly lower at a level of 0.1% during both the second (z = −9.13) and third wave (z = −14.18). Morbidity in the winter of 1919 was also significantly lower at a level of 0.1% compared with morbidity in the fall of 1918 (z = −5.05). Moreover, the ILI rate during the fall wave of 1918 in Bergen was significantly lower (at a level of 0.1%) than the fall waves in both the city of Baltimore (z = −9.04) and in the smaller town and rural areas of Maryland (z = −11.24).

An inverted U-shaped pattern of morbidity was apparent when related to age, irrespective of country/continent, urban-rural dimension, and gender (Figure 1A and 1B). This shape of the curve was marked for smaller towns and rural areas of Maryland during the second wave, showing that 5- to 34-year-old participants had the highest risk, with values approximately 50% irrespective of gender (Figure 1A). The same age pattern was also evident in Baltimore but at a markedly lower risk of catching the disease, ie, approximately 30% for the 1- to 34-year-olds (Figure 1A). In Bergen, however, this age-specific pattern was only evident during the first wave, showing that individuals between 10 and 39 years of age had the highest risk of falling ill with ILI, with values approximately 30% (Figure 1B). From the age of 30 in both areas of Maryland, as in Bergen during the first wave, the risk of being ill was steadily declining with age to reach values below 20% for those over 70 years.

In Bergen, more males than females aged 10–39 years reported having ILI during the summer wave (but boarder significant sex-difference only for those 20–29 years with z = 1.61), whereas more females than males aged 20–39 years had the illness during the fall wave (but boarder significant sex-difference only for those aged 20–29 with z = 1.58) (Figure 1B). This coincides with the finding that more females than males aged 10–24 years had the illness during the fall wave in both Baltimore and the smaller town and rural areas of Maryland (but boarder significant sex-difference only for those aged 20–24 years with a z = 1.49, in Baltimore, and at 5% level, z = 1.66, in the other areas of Maryland) (Figure 1A). There was a higher rate of

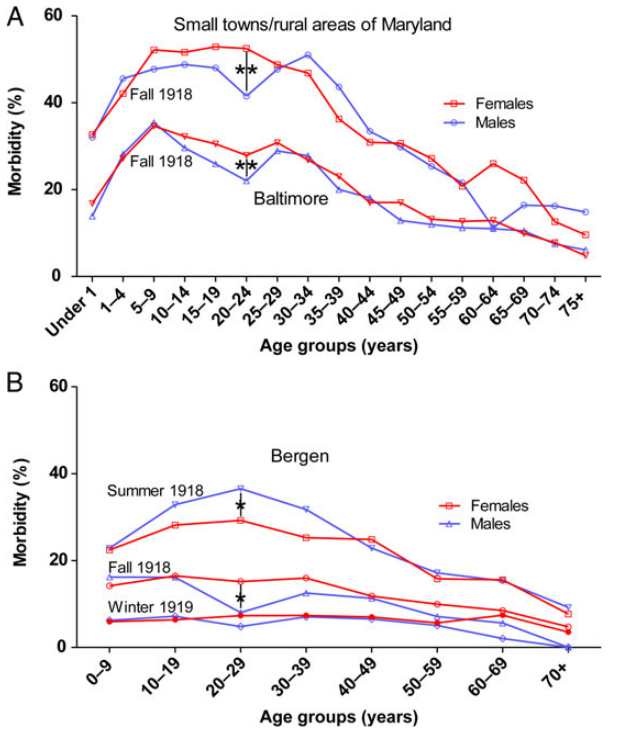

Figure 1. Morbidity, as percentage suffering from an influenza-like illness, among males and females of different ages living in (A) some smaller towns and rural areas of Maryland, and in the city of Baltimore, during the fall of 1918, as well as in (B) the city of Bergen, Norway, during the summer and fall of 1918 and the winter of 1919, as revealed by household surveys in the 2 areas of the United States, and in Bergen, Norway, including, respectively, 12 759, 33 776, and 10 633 individuals. The * and ** indicate significant gender differences at 10% and 5% levels, respectively.

ILI in 20- to 29-year-old females than in males during the third pandemic wave in Bergen, although this was not significant (z = 0.56).

The overall fatality and mortality rates were similar for the 3 survey areas in Maryland and Bergen, although they were not calculated for the same time-periods (Table 1). However, during the fall wave, fatality was not significantly higher in Bergen than in Baltimore (z = 0.08) and the smaller town and rural areas of Maryland (z = 0.17). It is worth noting that the fatality rates within each pandemic wave in Bergen varied: it was barely <1.0% during the first wave, whereas it was more than twice that number during both the second and the third waves. Mortality rates (deaths in percentages of the sample) in Bergen increased marginally from the first to the second wave, but the mortality rate in the third wave was only half of that in the 2 first waves. Due to the relatively low number of deaths, the differences over time in fatality and mortality in Bergen were far from being statistically significant. Finally, the mortality rate in Bergen during the fall wave were actually lower than the corresponding figures for both communities of Maryland, but they were likewise not significant (z = 0.15 for Baltimore and z = 0.30 for smaller towns and rural areas).

Because the number of deaths in Bergen were too few for reliable estimates of age- and sex-specific fatality and mortality rates by wave, these rates are therefore presented for the 3 waves combined from July 1918 to March 1919. Accordingly, the case fatality and mortality in Bergen for the complete pandemic period was lowest for individuals between 10 and 19 years of age, with slightly lower values for males relative to females (Figures 2C and 3C). For males between 20 and 29, however, this difference was reversed. This same tendency for age- and sex-difference in fatality and mortality rates was also seen in Baltimore during the second pandemic wave, ie, lower rates in males than in females aged 10–14 years and higher rates in males compared with females aged 25–29 (Figures 2B and 3B). In the smaller towns and rural survey areas of Maryland, however, marked and equally low fatality and mortality rates were observed in 5- to 9-year-old girls and boys, whereas these rates were higher in 25- to 44-year-old males than females (Figures 2A and 3A).

Results for all study settings, when looking at males and females combined, showed that the youngest children, as well as young adults and the oldest individuals, had the highest fatality and mortality rates, whereas children above 4 years as well as the middle-aged had lower rates (Figures 2A–C and 3A–C). Thus, it appeared that both fatality and mortality curves were roughly W-shaped.

DISCUSSION

Approximately half of the respondents reported to have had an ILI in Bergen in 1918–1919, covering all 3 waves of the pandemic, with most cases occurring during the first wave, followed in numbers by the second and third wave. This result suggests that infection was once protected against further attacks. The demonstration of higher morbidity in smaller towns and rural areas of Maryland during the pandemic fall wave, compared with Baltimore, is in line with previous assumptions that people living in less densely populated areas had been less exposed to influenza during the first wave than had those living in cities, leaving them more susceptible to ILI during the second wave of the disease [10, 19, 22]. Even though a summer wave of influenza is not mentioned in previous reports on the surveys conducted in the United States [15, 20, 21], the survey data from Bergen, together with the recognition of higher morbidity in

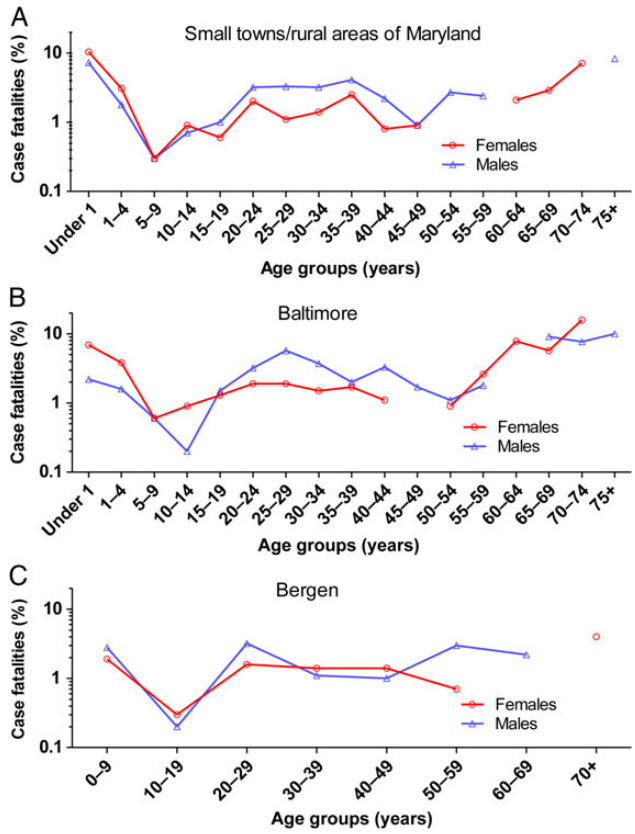

Figure 2. Case fatalities, as percentage of those with an influenza-like illness who is dying, among males and females of different ages living in (A) some smaller towns and rural areas of Maryland, and (B) the city of Baltimore during the fall of 1918, and in (C) the city of Bergen, Norway, during July 1918 through March 1919. None of the gender differences were statistically significant.

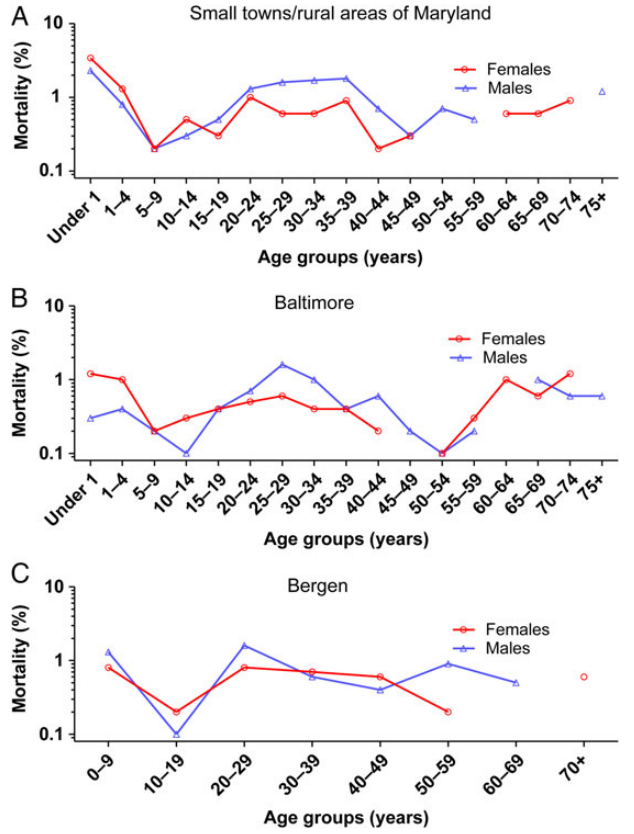

Figure 3. Mortality, as percentage of the population dying with an influenza-like illness, among male and females of different ages living in (A) some smaller towns and rural areas of Maryland, and (B) the city of Baltimore during the fall of 1918, and in (C) the city of Bergen, Norway, during July 1918 through March 1919. None of the gender differences were statistically significant.

smaller towns compared with more urban areas in Maryland, gives evidence supporting that a pandemic wave before the autumn of 1918 may actually have hit the city of Baltimore as well. This assumption is also is supported by several others showing that a first wave of influenza was present in the northeastern United States and the maritime region of Canada in April to August 1918 [3–5, 8, 23]. It is consistent with the distinct outbreaks in several Scandinavian cities during the summer of 1918 [24].

Furthermore, in support of this finding, more females than males just over the age of 20 fell ill in all areas of Maryland, similarly to in Bergen during the second wave of the disease, whereas more males than females of that age in Bergen reported to have been ill during the first wave in the summer. The author of the report from the Bergen survey believed an explanation for this marked gender difference could be that young male workers were more likely to be exposed to influenza during the first pandemic wave than women, who were the mostly home-based, leading to men being better protected during following waves [19]. On the other hand, authors of reports from the US surveys, claim that there were no marked difference in the ILI rates of the 2 sexes in this age group during the fall wave, and they argue that a higher ILI rate in females compared with males (as seen in Figure 1A) were due to the fact that the homemakers remember their own cases best [20, 21]. However, this argument does not explain the higher male-to-female ILI-rates at age 20–29 years in the Bergen during the summer.

Our finding that the case fatality rate in Bergen during the summer of 1918 (although not significant) was only half the rates during the following 2 waves is in line with observations that pandemic summer waves in the Northern Hemisphere are usually mild, partly due to the protective effect of vitamin D induced by sunlight [25, 26]. Access to vitamin D from ultraviolet-B radiation during the summer months was actually negatively correlated with case fatality rates in a study of 10 of the 18 canvassed areas of the US household surveys conducted in 1918–1919. Furthermore, these rates depended on how far north they were located [27]. The demonstration that induction of previtamin D3 during the summer months was much higher in Boston, at 42°N, than in Bergen, at 60°N [28], could thus possibly explain why a pandemic summer wave in Baltimore, at 39°N, might have been overlooked.

Despite the fact that the effect of sunshine in Bergen is less than in Baltimore, the morbidity during the autumn wave in 1918 was only approximately half that in Baltimore, and with similar case fatality rates, indicating that the population in Bergen was somehow otherwise protected. At that time, ie, approximately 1900, Norway and the other Scandinavian countries had higher life expectancy than any other European country [29]. It is tempting to link this to recent studies showing that the Norwegian population had high levels of vitamin D, probably due to year-round consumption of fish and cod liver oil [25].

The household surveys in both Baltimore and Bergen have demonstrated clear associations between morbidity, economic status, and congestion measures, such as persons per room and apartment size [19, 30]. Other factors may have played a role for the variation in fatality or mortality, but not morbidity, for example, variation in prepandemic immunity, baseline comorbidities and health, indigenous and ethnic background, occupational structure, and adequate nursing and rest [7, 10, 31–33]. Because we do not have access to raw data on all of these variables for our study units, we were unable to assess the role of these factors in explaining the observed variation in fatality and mortality.

Because Norway was a neutral country, whereas the United States participated in the First World War, the data for young adult men in the United States might have been biased due to a large proportion of them being in Europe as soldiers, leaving the more frail men behind [15, 20, 21]. However, the mortality of both males and females in both areas of Maryland follow the same pattern and level as in Bergen (Figure 2A–C and Figure 3A–C).

When relating the reported ILI-rates to age, in both areas of Maryland during the second wave, and in Bergen during the first wave, it appeared that the disease was most frequent in school-age children and young adults, with low morbidity rates in the very young children and steadily declining values in older individuals, creating inverted U-shaped curves. Low exposure due to less contact with nonfamily members might explain the low morbidity in the very young, whereas immunity induced by a previous influenza infection might explain the lower values in the middle-aged and older individuals [10, 34]. The steadily declining morbidity that appeared from 30 years of age may thus be due to some kind of immunity in those who lived during the 1889–1890 influenza pandemic [10, 34].

Our results showing the highest fatality rates in the youngest children, the young adults, and the oldest ones are similar to the age-related W-shaped fatality and mortality curves with data from total populations during the 1918–1919 pandemic [1, 10]. In the literature on the 1918–1919 pandemic, it has also been demonstrated that young adults aged 20–40 years had high mortality rates, with a peak at age 28–29 years, whereas middle-aged and older adults had unexpectedly low mortality rates, even into their sixties [10, 13]. Similar to the morbidity, the apparent protection of those beyond the age of 29 has thus been ascribed to cross-protective T- and B-cell memory responses induced in those who lived through the H3N8 influenza pandemic in 1889–1890. On the other hand, the high mortality of young adults could be due to a lack of prior influenza A infection (ie, immunological-naive individuals), or it could result from infection early in life to a heterologous virus, leading to either an insufficient memory B-cell response incapable of producing cross-protective antibodies, or an overwhelming immunopathology due to either exaggerated cross-reactive T-cell responses, or induction of T-cell responses incapable of controlling infection [34].

There is no clear evidence why school-age children, including youths up to 19 years in Bergen, should be protected from a fatal outcome of the disease, despite the fact that they were among those with the highest morbidity. A remarkably low fatality and mortality at school-age seems to have been registered worldwide and cannot be explained by a massive exposure to influenza virus previously [10, 13, 35]. Reduced death rates in 5- to 14-year-old children, living in England and Wales, was similarly observed in 1918, as well as in 1913 [35]. Moreover, this age group was largely spared from dying from tuberculosis in the United States during all the years from 1900 through 1940 [33]. Thus, it has been suggested that mortality from tuberculosis in 1918 and several years later was in some way interconnected with the 1918–1919 influenza pandemic [36]. From recent studies, however, it appears that children between the ages of 4–13 years, of both sexes, are better fit to cope with infections in general than are young adults [35], a phenomenon referred to as "the honeymoon of infectious diseases". This may be explained by an immune system, both innate and adaptive, at its very best.

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