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Influenza as a proportion of pneumonia mortality: United States, 1959–2009

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Abstract

As causes of death, influenza and pneumonia are typically analyzed together. We quantify influenza's contribution to the combined pneumonia and influenza mortality time series for the United States, 1959–2009. A key statistic is I/(P+I), the proportion of pneumonia and influenza mortality accounted for by influenza. Year-to-year, I/(P+I) is highly variable and shows long-term decline. Extreme values of I/(P+I) are associated with extreme P+I death rates and vice-versa, but I/(P+I) is a weak predictor of P+I mortality overall. Prominence of influenza in the medical news is associated with high I/(P+I). Influenza and pneumonia should be analyzed as a combined cause.

Keywords

cause of death classification; influenza; methodology; mortality; pneumonia; time series

Introduction and overview

Influenza is an acute infection of the respiratory tract, caused by the influenza virus. Most fatal cases of influenza involve pneumonia (Wright and Webster 2001). Deaths due to influenza-caused pneumonia may be recorded as influenza or as pneumonia, depending on (among other things) whether the medical professional filling out the death certificate has laboratory confirmation of influenza. Pneumonia deaths are seasonal, peaking in the winter, but occur at some level year-round. This indicates that not all pneumonia mortality is due to influenza virus, which has negligible or zero circulation during the summer (Glezen et al. 1987). Other causes of fatal pneumonia include the bacterium *Streptococcus pneumoniae*, commonly called pneumococcus (Bogaert et al. 2004).

Combined, influenza and pneumonia killed 56,284 people in the United States in 2008 (Miniño et al. 2011), making it the eighth leading cause of death, and accounting for 2.3 percent of all mortality. Moreover, influenza has been implicated as playing a causal role in the winter increase in cardiovascular disease mortality (Reichert et al. 2004). Influenza mortality is usually studied as an amalgam of influenza and pneumonia (Thompson et al. 2009*a*; Noymer 2008). The goals of this analysis are (i) to quantify influenza's contribution to the combined mortality from pneumonia and influenza for the United States, 1959–2009, (ii) to determine if influenza mortality data can be meaningfully interpreted separately from

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pneumonia, (iii) to identify medico-social correlates of the changing proportion of combined influenza and pneumonia mortality that is coded as influenza.

We characterize the relationship between influenza mortality and pneumonia mortality, as coded on the death certificate. Thus, hereinafter, when we speak of influenza, we mean mortality explicitly coded as influenza, and for pneumonia we mean that coded as pneumonia without mention of influenza.

We analyzed age- and sex-specific mortality for influenza and pneumonia for the United States, by month, from 1959 to 2009. The overall trend of combined pneumonia and influenza mortality is steady, but punctuated by events such as the 1968–69 “Hong Kong” H3N2 pandemic. Yet, influenza as a proportion of combined pneumonia and influenza mortality is highly variable over time and exhibits a long-term decline.

Taken at face value, there is high year-to-year variability of influenza mortality. Nonetheless, combined pneumonia and influenza mortality does not show the same level of variation. This suggests that other causes of fatal pneumonia become more prominent in years when there is less influenza. That is to say, in years when there is little influenza mortality, the other causes of pneumonia pick up the slack, to fill-out the total pneumonia and influenza (P+I) mortality. Conversely, these causes, such as pneumococcus or respiratory syncytial virus, must become less prominent when influenza mortality is ascendant. Biologically, this is implausible. A more parsimonious explanation is that the cause of death classification for influenza changes from year-to-year.

It is important to understand thoroughly the relative trends in influenza and pneumonia mortality. For example, Serfling regression (Dauer and Serfling 1961; Eickhoff et al. 1961; Serfling 1963) is a widely-used technique to estimate excess mortality from seasonal diseases, especially influenza. This technique calculates a cyclical baseline from summer-only data, since little or no influenza virus is in circulation. This baseline is then subtracted from the observed winter mortality; the result, which can be negative, is excess mortality. Some modern approaches to excess-mortality estimation differ in the details from Serfling regression but adopt similar overall logic (e.g., Choi and Thacker 1981*a*; Schanzer et al. 2007; Thompson et al. 2009*b*; Newall et al. 2010; Nunes et al. 2011).

Serfling regression takes mortality data on pneumonia and influenza (usually combined) as its input. Doshi (2008), however, considered influenza mortality, solely — i.e., without pneumonia, and without calculating excess. His data have subsequently been used by other investigators (Juzeniene et al. 2010). The analysis of influenza mortality without including pneumonia is unusual, and the present paper seeks to clarify best practice.

Materials and methods

We obtained data on number of deaths, by cause, from the mortality detail files of the National Center for Health Statistics (NCHS 2012). Deaths were stratified by age, sex, month, and underlying cause as coded on the death certificate. We extracted data on deaths from influenza, and from pneumonia without mention of influenza, from January 1959 to December 2009. This period spans four revisions of the International Classification of Diseases (ICD 7–10); the specific ICD codes used for each revision are given in Appendix 1. To ensure comparability, all data were converted to ICD-10 using the published crossover tables (Klebba and Dolman 1975; Klebba 1980; Anderson et al. 2001).

One advantage of working with data on death counts is that deaths are well-documented. The US has complete mortality registration, so every death results in a death certificate with a cause. On the other hand, rate data also require population counts from the census, which

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are subject to higher error rates. Censuses are generally regarded as having small undercounts, and the data are interpolated between decennial censuses, which compounds uncertainties. So while rates are subject to error in both numerator and denominator (Brillinger 1986), the data we use are mostly numerator data, where the count error rates are minimal. Conveniently, the ratio of counts and the ratio of death rates are equal, since the population denominators of the rates cancel out. For example, I/(P+I), the ratio of influenza to combined pneumonia and influenza, is the same whether "I" and "P+I" denote counts or rates. The quantity I/(P+I) plays an important role in our analysis. This is analogous to the use of (P+I)/(all causes) (Choi and Thacker 1981*b*), but at a different level of specificity.

To examine age-group-specific relationships, however, we also analyze some rate data. Rates were calculated using the above-described death counts in the numerator, and exposure data (i.e., person-years at risk) from the Human Mortality Database (accessed July 2012) in the denominator. Analyses were conducted using AWK (Aho et al. 1988), Stata v10.1 (College Station, TX, USA), and IDL v8.1 (Boulder, CO, USA).

Results and discussion

Time series of influenza and pneumonia deaths

Figure 1 plots two mortality time series for females: influenza, and pneumonia excluding influenza. Figure 2 plots the same data for males. These figures display two noteworthy patterns. Over the 51-year span, it is strikingly regular how much the two causes of death follow each other. Both peak in the winter and are in seemingly perfect synchrony, except for 2009. Pneumonia kills far more than influenza: the left axes (pneumonia) range from 800 to 8,000 deaths per month, while the right axes (influenza) range from 1 to 2,500 deaths per month (and the data rarely exceed 1,000).

The second thing to note in figures 1 and 2 is the long-run change in the two causes of death. Pneumonia deaths have moved upward with population growth, with the summer troughs going from about 1,000 deaths per month per sex in the 1960s, to approximately 2,000 deaths per month per sex in the 2000s. Influenza deaths, on the other hand, and despite population growth, have become rarer, with the summer troughs going from about 10 or more deaths per month per sex in the 1960s to under 5 deaths per month per sex in the 2000s. Starting in the 1990s, some months did not experience a single influenza death for either sex.

Advances in influenza surveillance have lead to the knowledge that summertime outbreaks of influenza-like illness (ILI) are only rarely caused by the influenza virus (Kohn et al. 1995). Evidently, this knowledge has influenced death recording practices. Even in the winter, influenza is becoming a less-used cause of death. Another feature of figures 1 and 2 is the decline over time in the relative peak-to-trough amplitude of pneumonia mortality; the reason for this is unknown.

Influenza as a proportion of influenza and pneumonia, I/(P+I)

The changes documented in figures 1 and 2 are seen more starkly in figure 3, which plots I/(P+I), influenza as a proportion of all pneumonia and influenza mortality, over time. The use of influenza as a cause of death has diminished in the long term. In the 1960s and 1970s it was not unusual, in the peak month of the flu season, to see at least one-quarter of all P+I deaths attributed to influenza. There has been a steady decline in this pattern, starting in the 1980s. More recently, influenza typically accounts for less than 10 percent of all P+I deaths. The 1980-81 flu season was the last in which influenza deaths exceeded 25 percent of all P+I deaths in a given month. Females are almost always slightly higher than males during the

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flu seasons of figure 3; the only exception is the fall wave of the 2009 H1N1 influenza pandemic.

Certain years are noteworthy in figure 3. The 1968–69 flu season — the "Hong Kong" flu pandemic of H3N2 (Cockburn et al. 1969) — recorded the highest influenza proportion of the 1960s. Over the 51-year span, the highest flu season on record for I/(P+I) was 1975–76, coincident with the "swine flu" scare (Stuart-Harris 1976). Specifically, from January to March 1976, the age-standardized death rate (ASDR, per 100,000) for P+I for males increased 112 percent, from 39.8 to 84.3, while I/(P+I) increased a whopping 966 percent, from 3.8 percent to 40.9 percent. Females showed a parallel trend: the ASDR increased 150 percent and I/(P+I) increased 896 percent.

The 1977–78 flu season, in which there was reemergence of "Russian" H1N1 influenza A virus (Nakajima et al. 1978), shows much higher I/(P+I) than either of the surrounding seasons. More recently, influenza was more seldom used. After 2000, the two highest peaks are the 2003–04 and fall 2009 flu seasons. In 2003–04, there was a fall vaccine shortage (Nelson 2003), and the emergence of the Fujian strain of influenza A/H3N2 (Centers for Disease Control and Prevention 2010). Throughout the early 2000s, there was also concern about the possibility of human transmission of H5N1 "bird flu" (Oxford 2005). The 2009 flu season corresponds to the H1N1 pandemic, which affected up to one-fifth of the US population (Shrestha et al. 2011; Cox et al. 2011). Thus, the use of influenza on death certificates seems to reflect its presence in the medical news.

Intra-season timing of I/(P+I)

The curve in figure 3 follows a half-wave rectified sinusoidal pattern. The use of influenza as a cause of death builds along with the number of deaths from P+I, as well as declines with it. It makes sense that the proportion of P+I deaths attributed to influenza is very low in the summer (e.g., Glezen et al. 1987). However, this graph could in theory follow more of a square wave pattern: during the flu season, some constant proportion is influenza, and during the summer a much lower (or zero) proportion is influenza.

To examine the idea that I/(P+I) builds during the flu season, table 1 compares the intra-season timing of the peak of I/(P+I), and the peak of P+I age-standardized death rate. The cross-tabulation shows that the two quantities either peak concurrently (i.e., on the diagonal), or with I/(P+I) lagging the P+I ASDR. Only three times in 51 flu seasons does I/(P+I) lead the P+I ASDR — twice for males and once in the female series — each time by one month. On the other hand, I/(P+I) lags the ASDR in 13 seasons for males and 18 seasons for females. For two seasons for males and one for females, I/(P+I) peaks in April, lagging the ASDR by three months. The only occurrence of December in table 1 is 2003, evidently an unusual flu season. Not only was influenza prominent in the medical news due to the vaccine shortage, but P+I death rates peaked unusually early. The only occurrence of November is in 2009, the first influenza pandemic since 1968–69.

Relationship between I/(P+I) and P+I death rates

Figure 4 shows the variation between the intensity of the flu season (measured by the P+I death rate), and the propensity for influenza to be used explicitly as the cause of death (measured by I/(P+I) mortality). The graphs plot monthly data for each sex separately. Three age groups are shown: 0–19, 20–64, and 65 and older. Over time, crude death rates have increased through population aging. Thus, we disaggregate by age to provide a better comparison (Cohen 1986). For these graphs, we use two six-month pseudoseasons: winter (November through April), and summer (May through October). These approximate the circulation of influenza virus better than any other half-year periods (Thompson et al.

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2009*b*). Summer and winter pseudoseasons are plotted in gold and purple, respectively, along with their corresponding regression lines. The plots are log-log, or scale-invariant (Keeling 1999; Rhodes and Anderson 1996). Appendix 2 provides a regression table showing that this age-, sex-, and pseudoseason-disaggregated analysis is justified.

The one-way variation in figure 4 is worth noting. Specifically, as age increases, the P+I death rate shows less total variation: the 0–19 year-old data span about 2 logs on the horizontal axis, the 20–64 year-old data span about one log, and the 65 and older data are nested within one log. On the other hand, for I/(P+I), the oldest age group shows the most variation, spanning about three logs on the vertical axis, while the other two age groups span about two logs.

The hard boundary with a striped appearance, seen on the lower left of each panel, is an artifact of integer constraints on the number of deaths per month. The 0–19 age group has many months with 1, 2, or 3 influenza deaths, which makes the stripes particularly apparent. The time span is 612 months, but months in which I/(P+I)=0 cannot be plotted on log scale. Hence, the number of points plotted in each figure is less than 612 (the numbers are given in the panel captions).

The bivariate analysis in figure 4 reveals important relationships. The 0–19 age group has the fewest deaths, with overlapping summer and winter data, making it diffecult to discern a clear differentiation between the two pseudoseasons. The regression lines for winter and summer are negative. In other words, as the P+I death rate increases, I/(P+I) decreases, or viceversa, as causality could run the other way.

The 20–64 age group, on the other hand, has the opposite bivariate relationship, with positive slopes for both the summer and winter regression lines. As the P+I death rate increases, so does I/(P+I). The winter slope is steeper than that of the summer. While a clear distinction between the summer and winter data is lacking here as well, the differentiation is more apparent than in the younger group.

The age group 65 and older has the most deaths and the most interesting bivariate relationship. The summer and winter data show a salient differentiation, clearly occupying different regions. What is more, the slopes of the summer and winter regression lines have different signs. In the winter, as the P+I death rate increases, I/(P+I) does too. In the summer, there is a negative relationship between P+I death rate and I/(P+I). In the end of the pseudosummer (e.g., October), P+I death rates begin to increase but I/(P+I) stays low, which creates a negative relationship overall. For males, in the summer there is large spread of I/(P+I) over a narrow range of rates, and hence no strong relationship.

The most important point from the scatterplots, especially for the oldest age group, is that the highest P+I death rates are predictive of the highest I/(P+I) proportion (or vice-versa); this only applies to a handful of months, however. Beyond that, there is a poor relationship between I/(P+I) and the P+I death rate, despite the fact that, as seen in figures 1 and 2, the cycles follow each other so well. Indeed, the goodness of fit for the age group 65 and older of figure 4, as measured by the R^2, is quite poor in the winter for females ($R^2 = 0.051$) and higher for males but hardly overwhelming ($R^2 = 0.33$). This is seen not only in the scatterplots but by reconsideration of figure 3, where there are huge year-to-year swings in I/(P+I). These drastic changes may be compared to figures 1 and 2; over short time spans, these are good approximations to the rate changes, since the populations at risk in the rate denominators change relatively slowly.

Influenza mortality, the numerator of the key quantity of this study, drives most of the month-to-month variation in I/(P+I). Over all ages, the R^2 for I predicting I/(P+I) in an OLS

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regression (N = 612 months) is 0.9031 for males, and 0.9062 for females. This is hardly surprising, given the way I/(P+I) is set-up, and the fact that influenza experiences more dramatic relative peak-to-trough seasonal swings compared to pneumonia (figures 1 and 2). One could essentially replace the data on the vertical axis of figure 4 with influenza alone without altering the pattern. This only reinforces our point, that expressly-coded influenza mortality is not a good proxy for influenza-attributable mortality. It does not predict the pneumonia and influenza death rate very well, and it is known to have spurious peaks, such as the during the spring 1976 swine flu scare.

Our results suggest that the variation over time in influenza-only mortality is just as affected, if not more so, by seemingly-random year-to-year reporting changes as by actual changes in influenza-associated mortality. These results strongly endorse the standard practice of combined analysis of pneumonia and influenza mortality.

Conclusion

As a cause of death, influenza is highly variable from year-to-year. Influenza and pneumonia are typically combined in mortality analysis, although this has been challenged by Doshi (2008). We analyzed disaggregated influenza and pneumonia data to quantify their relationship and to determine best practice. To produce estimates of excess mortality, Serfling regression (and similar techniques), takes as its inputs the data considered herein (Eickhoff et al. 1961). Detailed knowledge of the inputs can help interpretation of the outputs and models (Nishiura 2011).

Over the 51-year span, influenza has seen a decline in use on the death certificate. Years in which influenza is in the medical news are exceptions to this trend, with the 1975–76 “swine flu” scare on record as the highest proportion I/(P+I). Despite the decline, during each flu season, the proportion I/(P+I) builds during the winter. Of course, this could only go to show that as influenza viral circulation grows each winter, so does its impact on mortality. This is probably part of what is happening, but it does not explain the tremendous year-to-year variation in the proportional use of influenza, in the face of more-or-less similar overall P+I mortality.

Increased influenza vaccination, especially since the 1980s, could play a role in the secular decline of I/(P+I). Higher vaccination rates may reduce the propensity to code a pneumonia death as specifically attributable to influenza. It is possible that influenza vaccination impacts morbidity (Nichol et al. 2007) more than mortality. The actual role of increased influenza vaccination in the reduction of influenza mortality has been debated (Simonsen et al. 2005*a*; Thompson et al. 2005; Simonsen et al. 2005*b*).

As the flu season builds, so does short-term medical awareness of influenza, and this is reflected by the patterns of I/(P+I) reported herein. It may be that explicitly-coded influenza deaths are the result of greater laboratory confirmation, but that just begs the question of whether there is increased testing in years when influenza is making medical news. For example, 1975–76 was the swine flu scare but not an actual outbreak. The 2009 mortality data further support this hypothesis, with the 2009 I/(P+I) being the highest since the 1980–81 flu season.

What gets recorded on the death certificate and why has long been a subject of interest for historical demographers (Alter and Carmichael 1996, 1997, 1999). This study shows that, influenza versus pneumonia death classification is, in part, influenced by medical-social factors.

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Figures 1 and 2 show that influenza and pneumonia mortality co-move, but figures 3 and 4 show that, overall, influenza-only mortality is a poor predictor of P+I mortality. Epidemic phenomena are often assumed to be power-law processes, but our results show that the influenza and pneumonia relationship is a poor fit to scale-invariance, reinforcing the notion that influenza alone should not be used as a stand-in for P+I mortality. The simplest interpretation of our results is that influenza is not a cause-of-death classification to be trusted. Barring an especial reason, influenza mortality should never be analyzed as a stand-alone cause, but instead should be combined with pneumonia.

Our analysis both supports and contradicts a recent finding, that "recorded influenza" mortality is in decline (Doshi 2008). It is supportive in the sense that it's replicative: we show that, despite population growth, influenza deaths have indeed declined in the period 1959–2009. However, this is overwhelmingly driven by a reduction in the propensity for influenza (as opposed to pneumonia) to be used as the underlying cause of death. Using vital statistics data alone is not sufficient to address definitively the question of influenza's relative importance in P+I mortality. Autopsies may, in theory, provide more information, but those conducted as a matter of course are performed when the cause of death is unknown. For pneumonia deaths, an autopsy is not normally performed to determine the etiologic agent (Liu et al. 2012). Large-scale autopsy studies specifically designed to address the question of I/(P+I), although prohibitively expensive, would be the gold standard (Kircher et al. 1985).

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Appendix 1: ICD codes for influenza and pneumonia

	Cause of death	
Years	**Influenza**	**Pneumonia**†
1959–1967 (ICD 7)	480–483	490–493
1968–1978 (ICD 8)	470–474	480–486
1979–1998 (ICD 9)	487	480–486
1999–2009 (ICD 10)	J10–J11	J12–J18

†excluding influenza

Appendix 2: Figure 4 regression table

log(I/(P+I))	Coefficient	SE	*t*	*P*> |*t*|	[95% Conf. Interval]	
Winter, female, 0–19	−0.256	0.7747	−0.33	0.741	−1.775	1.263
Winter, male, 20–64	19.036	1.2512	15.21	0.000	16.582	21.489
Winter, female, 20–64	20.506	1.3530	15.16	0.000	17.853	23.159

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log(I/(P+I))	Coefficient	SE	*t*	*P*> |*t*|	[95% Conf. Interval]	
Winter, male, ≥65	21.086	1.4656	14.39	0.000	18.212	23.960
Winter, female, ≥65	7.775	1.2221	6.36	0.000	5.379	10.171
Summer, male, 0–19	−3.773	0.8545	−4.42	0.000	−5.448	−2.097
Summer, female, 0–19	−5.141	0.9112	−5.64	0.000	−6.928	−3.354
Summer, male, 20–64	12.059	1.9099	6.31	0.000	8.315	15.804
Summer, female, 20–64	17.786	2.1834	8.15	0.000	13.504	22.067
Summer, male, ≥65	−6.312	3.3454	−1.89	0.059	−12.871	0.248
Summer, female, ≥65	−29.480	1.9032	−15.49	0.000	−33.212	−25.748
log(P+I death rate)	−0.208	0.0513	−4.05	0.000	−0.308	−0.107
log(P+I death rate) × {Winter, female, 0–19}	−0.027	0.0738	−0.37	0.715	−0.172	0.118
log(P+I death rate) × {Winter, male, 20–64}	2.051	0.1288	15.92	0.000	1.798	2.303
log(P+I death rate) × {Winter, female, 20–64}	2.066	0.1336	15.47	0.000	1.804	2.328
log(P+I death rate) × {Winter, male, ≥65}	3.333	0.2225	14.98	0.000	2.897	3.770
log(P+I death rate) × {Winter, female, ≥65}	1.138	0.1758	6.47	0.000	0.793	1.483
log(P+I death rate) × {Summer, male, 0–19}	−0.287	0.0806	−3.57	0.000	−0.445	−0.129
log(P+I death rate) × {Summer, female, 0–19}	−0.412	0.0858	−4.8	0.000	−0.580	−0.244
log(P+I death rate) × {Summer, male, 20–64}	1.390	0.1940	7.17	0.000	1.010	1.770
log(P+I death rate) × {Summer, female, 20–64}	1.834	0.2091	8.77	0.000	1.424	2.244
log(P+I death rate) × {Summer, male, ≥65}	−0.707	0.4972	−1.42	0.155	−1.682	0.267
log(P+I death rate) × {Summer, female, ≥65}	−4.007	0.2661	−15.06	0.000	−4.529	−3.485
intercept	−5.015	0.5348	−9.38	0.000	−6.064	−3.967

Number of observations = 3029

$F(23, 3005) = 147.75$

Prob > $F = 0.0000$

$R^2 = 0.5307$

RMS Error = .92988

The analysis of figure 4 is disaggregated into 12 groups: two regression lines (winter, summer) per panel, with six panels (three age groups, two sexes). As noted in the text, this is done because of the important differences between age groups. This regression table shows a three-way interaction (pseudoseason × sex × age group). The 24 coefficients recapitulate the 12 regression lines of figure 4 (one slope and intercept per line). The omitted category is winter males, 0–19. Most age/sex/pseudoseason combinations are statistically different (p <0.0005), justifying the disaggregation. Winter females, 0–19, are not distinguishable from the omitted category. Summer males age 65 and older have a slope that is not statistically distinguishable from the omitted category and an intercept that is borderline (p =0.059).

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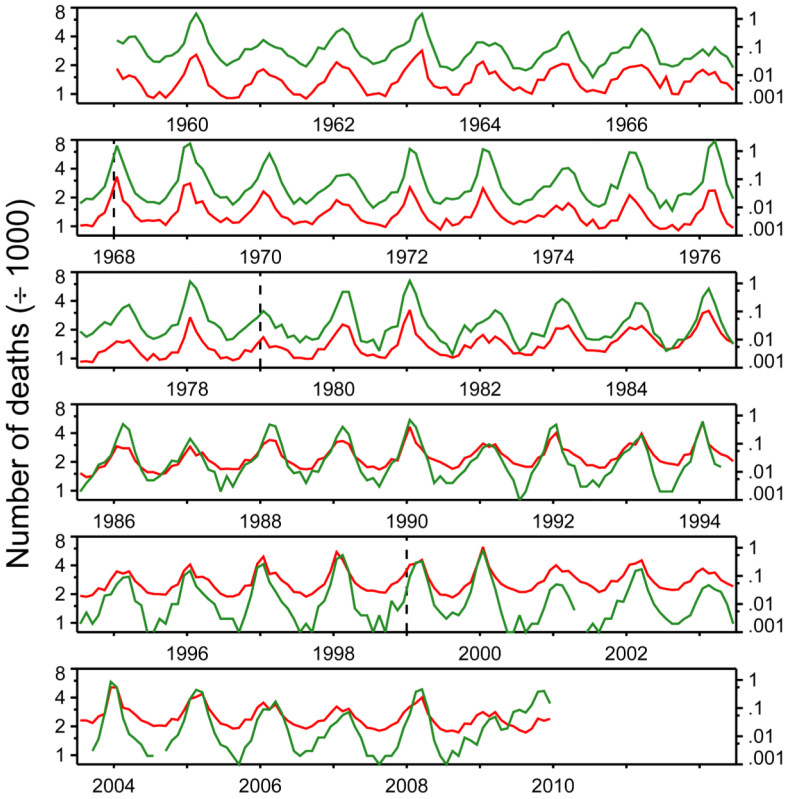

Figure 1.

Females. Time series graph of pneumonia deaths (red), left scale, and influenza deaths (green), right scale. Vertical axes are logarithmic. Vertical dashed lines denote changes in ICD revisions, although the data are ICD-adjusted. Gaps in the green data series correspond to months with no influenza deaths.

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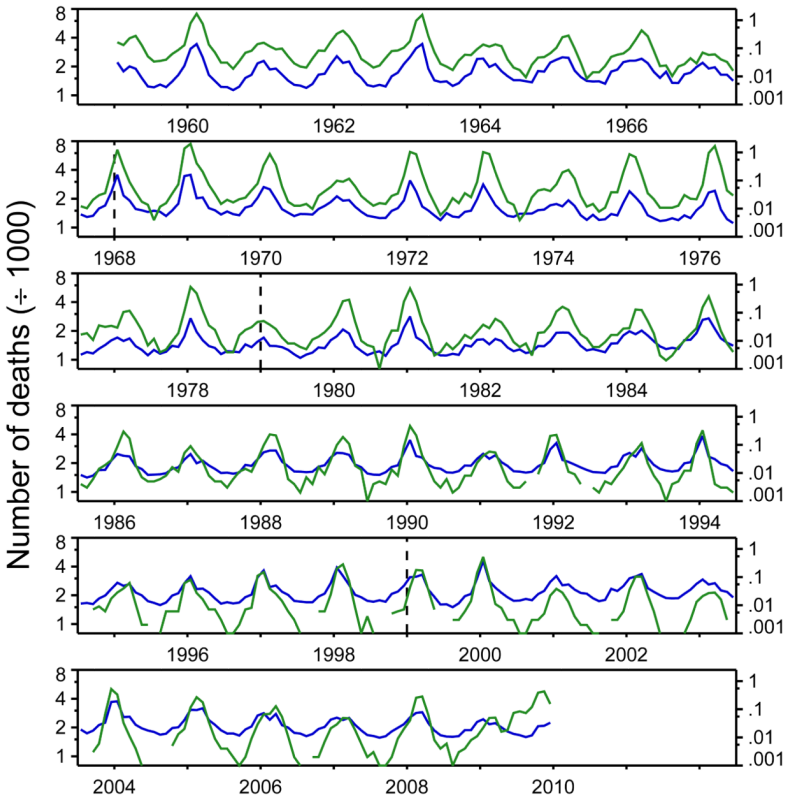

Figure 2.

Males. Time series graph of pneumonia deaths (blue), left scale, and influenza deaths (green), right scale. Vertical axes are logarithmic. Vertical dashed lines denote changes in ICD revisions, although the data are ICD-adjusted. Gaps in the green data series correspond to months with no influenza deaths.

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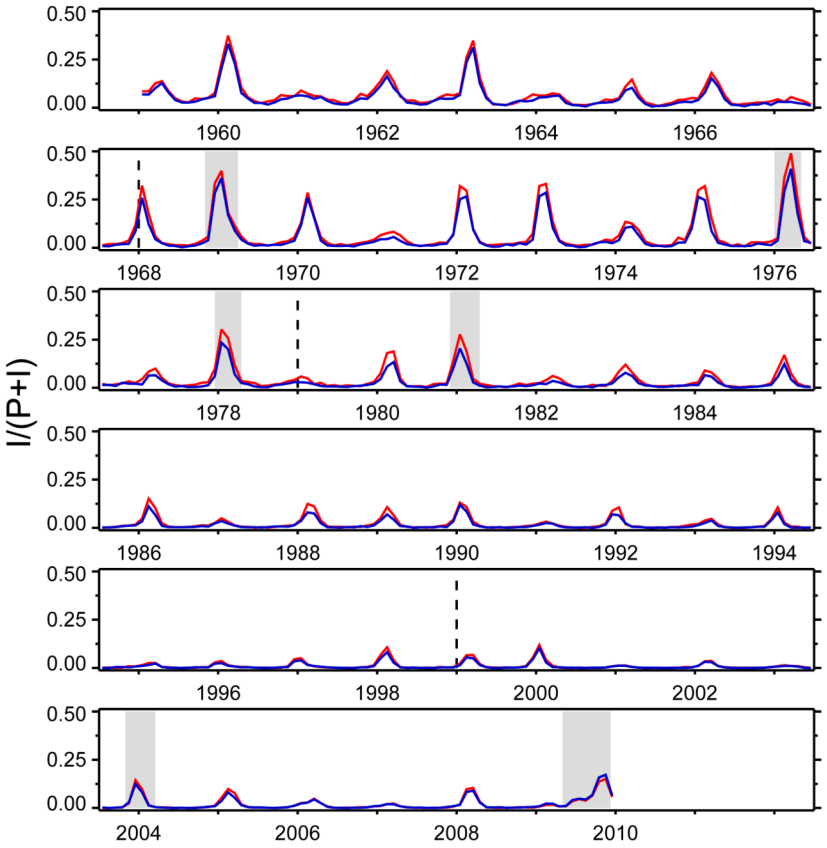

Figure 3.

Time series graph of influenza as a proportion of total pneumonia and influenza mortality, all ages. Red, females; blue, males. Vertical dashed lines denote changes in ICD revisions, although the data are ICD-adjusted. Shaded regions are discussed in detail in the text.

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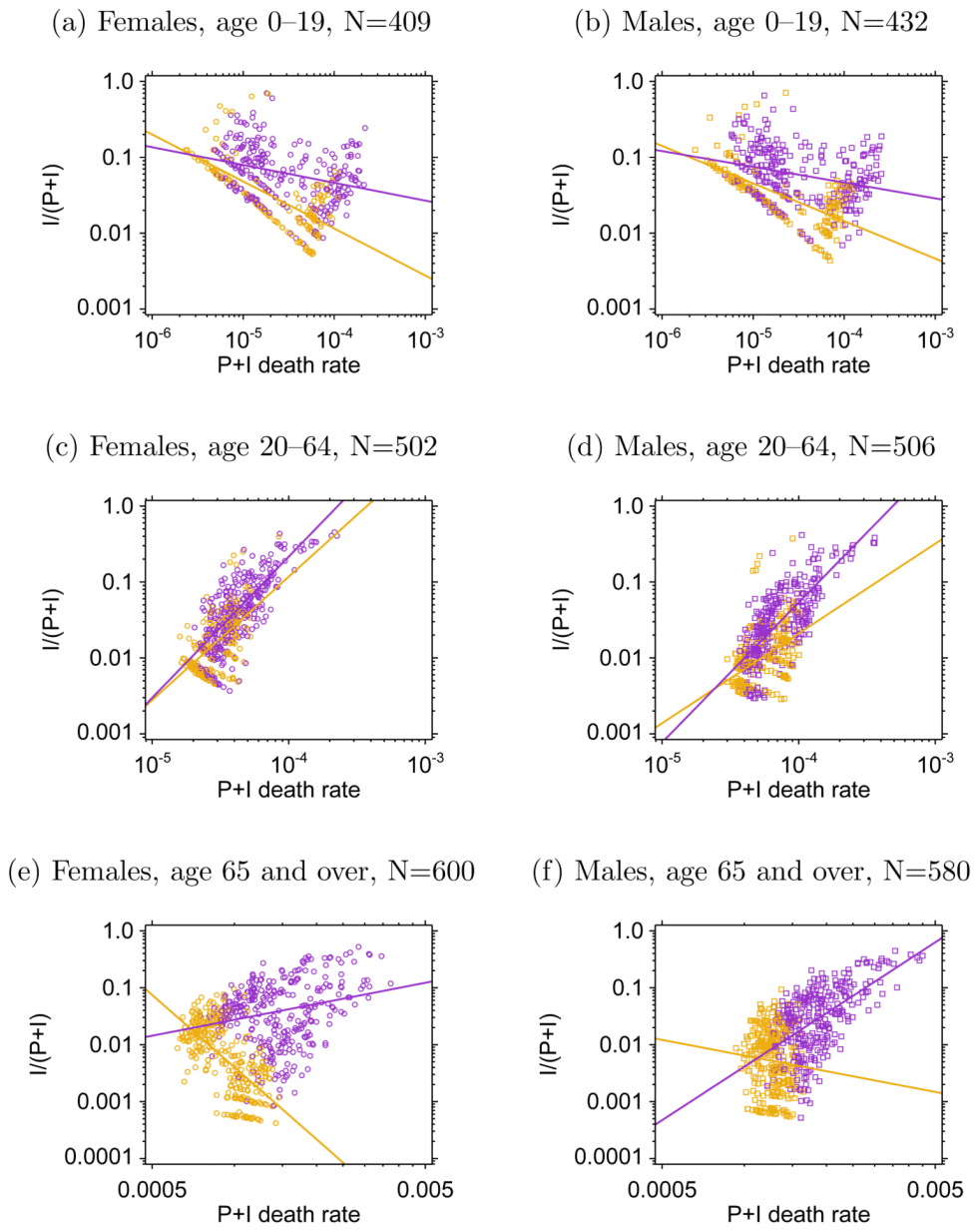

Figure 4.

Scatterplots of I/(P+I) vs. P+I death rate, age 0–19 (a,b), age 20–64 (c,d), and 65 and older (e,f). Females, left panels (circles); males, right panels (squares). Each plotting symbol represents one month, 1959–2009. Summers are gold; winters are purple. Ordinary least-squares regression lines are also shown for each pseudoseason; all slopes differ significantly from zero ($p < 0.0005$) except panel (f), summer ($p = 0.17$), but the two lines in panel (f) are statistically different from each other ($p < 0.0005$). Months plotted in random sequence to avoid systematic summer or winter overlap.

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Table 1

Cross-tabulation of peak months for I/(P+I) and P+I ASDR, 51 winters, 1958–59 to 2008–09, plus November–December 2009. The first win-ter starts in January 1959 due to data availability; all others run November–April, inclusive (except the 2009–10 season). Peaks of I/(P+I) almost always co-occur or lag the peak P+I death rate.

Peak month, I/(P+I)	Peak month, P+I Age-Standardized Death Rate									
	Males					Females				
	Nov	Dec	Jan	Feb	Mar	Nov	Dec	Jan	Feb	Mar
November	1	0	0	0	0	1	0	0	0	0
December	0	1	2	0	0	0	1	0	0	0
January	0	0	14	0	0	0	0	14	0	0
February	0	0	5	13	0	0	0	5	11	1
March	0	0	1	5	8	0	0	4	8	6
April	0	0	2	0	0	0	0	1	0	0