



DEMOGRAPHIC RESEARCH

A peer-reviewed, open-access journal of population sciences

DEMOGRAPHIC RESEARCH

**VOLUME 37, ARTICLE 45, PAGES 1445–1476
PUBLISHED 16 NOVEMBER 2017**

<http://www.demographic-research.org/Volumes/Vol37/45/>

DOI: 10.4054/DemRes.2017.37.45

Research Article

**Summertime, and the livin' is easy: Winter
and summer pseudoseasonal life expectancy in
the United States**

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Summertime, and the livin' is easy: Winter and summer pseudoseasonal life expectancy in the United States

Tina Ho¹

Andrew Noymer²

Abstract

BACKGROUND

In temperate climates, mortality is seasonal with a winter-dominant pattern, due in part to specific causes of death, including pneumonia, influenza, and cold-induced thrombosis. Cardiac causes, which are the leading cause of death in the United States, are winter-seasonal, although the pathways are incompletely understood. Interactions between circulating respiratory viruses (e.g., influenza) and cardiac conditions have been suggested as a cause of winter-dominant mortality patterns.

OBJECTIVE

In this paper we aim to quantify the total mortality burden of winter in the United States.

METHODS

We calculate 'pseudoseasonal' life expectancy, dividing the year into two six-month spans, one encompassing winter, the other summer.

RESULTS

During the summer when cold weather is absent and the circulation of respiratory viruses is significantly reduced, life expectancy is about one year longer. We also quantify the seasonal mortality difference in terms of seasonal 'equivalent ages' (defined herein) and proportional hazards.

CONTRIBUTION

We quantify the effects of winter mortality. The population-level mortality reduction of a perfect influenza vaccine (which can only reduce a portion of winter-attributable mortality) would be much more modest than is often recognized.

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*Summertime,
And the livin' is easy
Fish are jumpin'
And the cotton is high*
– George Gershwin
“Summertime”

1. Introduction

The primary goal of this paper is to quantify the mortality impact of winter in the United States, in terms of life expectancy. To accomplish this, we analyze life expectancy in the United States from a seasonal perspective. We calculate two life expectancies per 12-month period (‘pseudowinter’ and ‘pseudosummer’), using methods described below. The point is to estimate life expectancy in the absence of respiratory viruses (most notably, influenza) and the effects of cold, using pseudosummer as an approximation. Pseudowinter, on the other hand, estimates life expectancy in the presence of these viruses and cold-induced conditions. The difference between life expectancy in pseudowinter and pseudosummer gives the total mortality impact of winter. The pseudoseasonal approach also illuminates within-year mortality fluctuations.

Mortality in temperate climates is highly seasonal, with winter peaks and summer troughs (Rosenberg 1966; Land and Cantor 1983; Kalkstein and Davis 1989; Mackenbach, Kunst, and Looman 1992; Rau 2006; Deschênes and Moretti 2009; Deschênes and Greenstone 2011). Respiratory and cardiovascular causes, including stroke (Sheth et al. 1999; Yang et al. 2016), dominate the seasonal effects, with cancer being negligibly cyclical (Crombie et al. 1995). Heat wave mortality peaks are ephemeral interruptions of this overall pattern (e.g., Basu and Samet 2002; Klinenberg 2002; Valleron and Boumendil 2004; Kaiser et al. 2007; Toulemon and Barbieri 2008; Rocklöv, Ebi, and Forsberg 2011; Robine, Michel, and Herrmann 2012; Åström et al. 2013). Heat-wave-associated deaths have a different composition by cause compared to summer mortality (Huynen et al. 2001; Basagaña et al. 2011), although Rey et al. (2007) report increases in most causes at older ages. Heat wave mortality interacts with air pollution (Rooney et al. 1998; Bhaskaran et al. 2009). Both severity and duration of heat waves are important for mortality (Anderson and Bell 2009).

Temperature is thought to play a role in the seasonality of mortality (Mackenbach, Looman, and Kunst 1993; Braga, Zanobetti, and Schwartz 2001, 2002; Curriero et al. 2002; Mercer 2003). However, temperature-associated deaths in a literal sense (e.g., hypothermia or heat stroke) are relatively unimportant, with cold-related deaths slightly exceeding heat-related deaths, at least in the United States (Berko et al. 2014). Nonetheless, the expansion over time of adequate winter heating in the United States has been

suggested as a possibly overlooked factor in the long-term decline of heart disease (Sere-takis et al. 1997). Congruently, Kunst, Looman, and Mackenbach (1991) report declines in winter excess mortality in the Netherlands between the 1950s and the 1980s. On the other hand, van Rossum et al. (2001) do not find changes in seasonal mortality over 25 years in English civil servants. Insufficient winter heating among the poor may not play a significant role in mortality in Britain (Wilkinson et al. 2004) or Denmark (Rau 2004). Healy (2003) demonstrates that the coefficient of seasonal variation in mortality (CSVM) is correlated with mean winter temperature (warmer temperature, higher CSVM) at the country level in Europe; see also Keatinge et al. (1997, 2000), Díaz et al. (2005), Gaspar-rini et al. (2015), and Carmona et al. (2016). Analitis et al. (2008) also find an association between cold weather and mortality in European cities, and similarly note greater cold effect in warmer climates. Yang et al. (2012) and Zhao, Zhu, and Tu (2015) find similar results in subtropical Asia. Kysely et al. (2009) find increased cardiovascular mortality in all ages above 25 during cold spells in the Czech Republic. Mortality in nursing homes appears to be sensitive to both hot and cold temperature extremes (Stafoggia et al. 2006; Hajat, Kovats, and Lachowycz 2007).

The role of temperature in mortality is an important topic in historical demography, too large to survey completely here. Much of this work focuses on summer mortality, especially diarrhea among infants and children (e.g., Galloway 1985; Breschi and Livi-Bacci 1986a,b,c; Woods, Watterson, and Woodward 1989). There is a smaller body of work on winter peaks in infant mortality before the 20th century. In particular, the hypothermia hypothesis suggests that neonatal mortality increased in cold periods (Dalla-Zuanna and Rosina 2009, 2011; see also Derosas 2009, 2010 and Dalla-Zuanna and Rosina 2010). Analyzing historical data from a cold-winter climate, Åström et al. (2016) find that warmer spells are associated with lower mortality. Ekamper et al. (2009) find a strong social class influence on temperature–mortality relationships in historical data from the Netherlands, and review some of the literature on cold and mortality in the past.

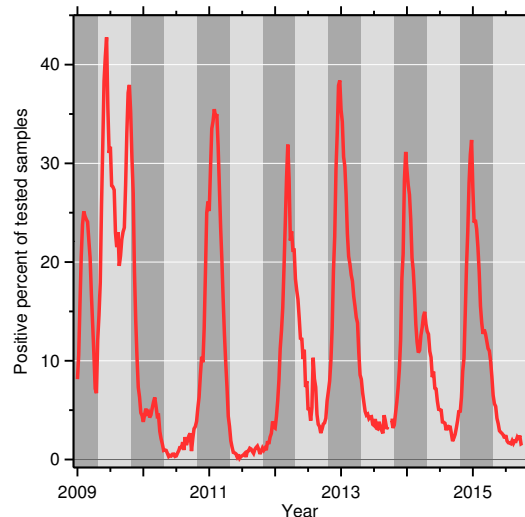
Cold temperature affects susceptibility to viruses in mice (Foxman et al. 2015) and in human cells in vitro (Foxman et al. 2016), although the evidence in humans is mixed (Dowling et al. 1958; Douglas, Jr., Lindgren, and Couch 1968; Graham 1990; Mourtzoukou and Falagas 2007; Footitt and Johnston 2009; Eccles and Wilkinson 2015; Ikäheimo et al. 2016). There may be synergistic effects of temperature and humidity (Lowen et al. 2007; Mäkinen et al. 2009; Shaman and Kohn 2009; te Beest et al. 2013). In the United States, mortality peaks coincide with the Christmas and New Year’s holidays, which occur during the northern hemisphere winter (Phillips et al. 2004; Phillips, Barker, and Brewer 2010). However, Christmas effects on cardiovascular mortality also occur in New Zealand, where the holiday falls in the summertime (Knight et al. 2016). Hypovitaminosis D, which is seasonal with winter peaks (Kasahara, Singh, and Noymer 2013), is also thought to play a role in fatal diseases (Holick 2007).

Mortality seasonality at temperate latitudes is influenced by more than just viral circulation. Cold can affect thrombosis, with consequences for stroke and myocardial infarction (Keatinge et al. 1984, 1986). Pan, Li, and Tsai (1995) write: “Poor thermoregulation in older people may precipitate cardiovascular disease events.” Zöller et al. (2013) note winter peaks in venous thromboembolism in Sweden. The causal connection between winter cold and thromboembolism may be coagulation factors (Dentali, Manfredini, and Ageno 2009). On the other hand, there is some evidence that extreme cold (below -30°C) may reduce mortality (Otrachshenko, Popova, and Solomin 2017), although the mechanism may be as prosaic as people staying indoors when it is that cold outside.

Air pollution is another contributor to mortality seasonality in temperate climates. All else equal, air pollution is positively associated with increased mortality (Schwartz and Dockery 1992; Chay, Dobkin, and Greenstone 2003; Chay and Greenstone 2003, 2005; Currie, Neidell, and Schmieder 2009). This relationship can affect mortality in winter and summer. In the summertime, daytime UV radiation plays a role in air pollution (Cleveland and Graedel 1979). In the wintertime, solar UV incidence is lower, but some air pollution, including particulates, is often higher due to home heating emissions (Currie and Neidell 2005; Janhäll et al. 2006; Olofson et al. 2009; Johnston et al. 2013; Tang et al. 2017). Pollution is another example of multidimensionality of the seasonality of mortality.

The root causes of infectious disease mortality seasonality remain poorly understood (Dowell 2001; Cheng 2005). There seems to be a nexus between viral activity and adverse cardiovascular events (Bainton, Jones, and Hole 1978; Kunst, Looman, and Mackenbach 1993; Madjid et al. 2004; Huy et al. 2012; Udell et al. 2013). However, the extent to which respiratory virus transmission during the winter (Glezen et al. 1987) causes increased mortality from other causes is debated (Reichert et al. 2004; Warren-Gash et al. 2012; Foster et al. 2013). The role of the astronomical season (*viz.*, through associated weather changes) in the cyclicity of infectious disease is also debated (Fisman 2012; Treanor 2016), with the school calendar (Grenfell and Anderson 1989), and dynamic resonance (Dushoff et al. 2004) among alternate hypotheses. The plurality of explanations suggests to us that “the mechanisms underlying seasonality [of viral transmission] still remain essentially unexplained” (Yorke et al. 1979: 104–105; see also Lofgren et al. 2007). Determining the causes of seasonality of respiratory virus transmission is beyond our scope. Rather, we are concerned with estimating the mortality consequences of such seasonality.

Figure 1: Percentage of respiratory specimens testing positive for influenza virus, 2009–2015



Note: Weekly data from CDC (2015). Pseudowinters are shaded dark.

2. Data and methods

We present a simple approach to estimate the overall impact of winter on mortality. We divide the year into two six-month ‘pseudoseasons’ and calculate life expectancy for these periods. The seasonal binning approach using all-cause mortality avoids potential classification pitfalls of counterfactual approaches such as cause-deleted life tables (in which winter-dominant causes such as respiratory viruses and thrombosis are deleted) or other approaches which rely on cause-of-death reporting (e.g., Stewart 2011). Cause-deleted life expectancy is calculated with specific causes statistically removed (Manton, Stallard, and Vaupel 1986), while our approach studies all-cause mortality, but truly in the absence of winter-related mortality, i.e., in the summertime. The major strength of our approach is that our mortality estimates are not hypothetical ‘as if’ constructs but reflect observed conditions when no (or very little) flu virus circulates and in which there is no cold-related thrombosis mortality, and so on. Among the problems that this approach avoids are classification errors regarding what is an influenza (or thrombosis, etc.) death (cf. Noymer and Nguyen 2013).

Regarding the influenza component of winter mortality, the noncirculation of flu viruses in the summertime is not absolute, as Figure 1 shows. This is a time series plot of respiratory specimens (e.g., nasal swabs) that are positive for any strain of the influenza virus (as a percentage, so peaks are not reflective of more samples during the winter). Note that even in the peaks, most samples test negative; there are many causes of upper respiratory illness other than influenza virus. Most peaks of influenza occur in the shaded pseudowinters, but the 2009 swine-origin influenza pandemic is a major exception. During influenza pandemics, which involve emergence of new strains, viral circulation in the summer is more likely (Webster et al. 1992).

From the mortality detail files of the National Center for Health Statistics (NCHS 2015), we extracted monthly data on every death in the United States, January 1959 to December 2014.³ The data was then aggregated by sex and 22 age groups (0, 1–4, 5–9, . . . , 95–99, ≥ 100), and binned into six-month pseudoseasons. Pseudowinter is November through April, and pseudosummer is May through October; pseudoseasons do not nest into calendar years. In long-run averages, these six-month periods best capture influenza virus circulation or lack thereof.⁴ The data begins with pseudosummer 1959 and ends with pseudosummer 2014 (56 pseudosummers). There are 55 pseudowinters (1960–1961 to 2013–2014). Data for January through April 1959 was discarded since using this data for pseudowinter 1958–1959 would be biased due to the omission of November and December 1958. Similarly, November and December 2014 were discarded. We constructed denominators using age- and sex-specific calendar-year exposure data from the Human Mortality Database. We graduated this person-years-at-risk data to months, adjusting for days per month and leap years, and then reaggregated to make pseudoseasonal exposures. The specifics of the denominator construction are described in the appendix. We then calculated sex- and age-specific death rates (deaths divided by person-years lived) for each pseudoseason, from which we calculated sex-specific period life tables in the standard way (Keyfitz 1970; Preston, Heuveline, and Guillot 2001).

Using pseudoseasons, as defined, is the closest approximation to respiratory virus transmission patterns. Some early flu seasons start in November, and some late flu seasons can persist into April. An alternative would be to omit shoulder periods, by using

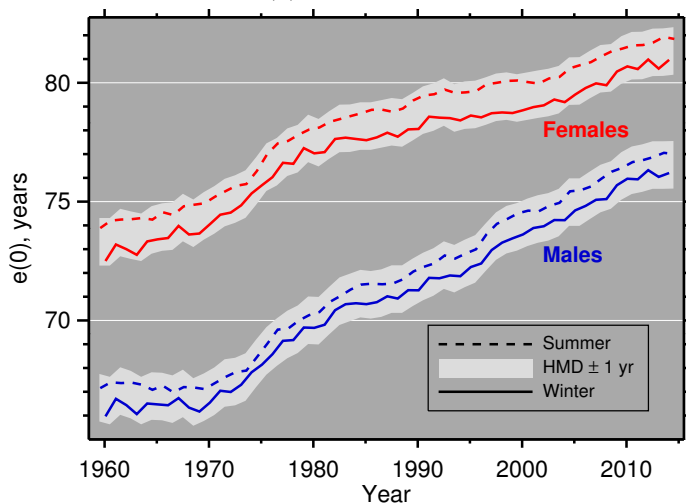
³ Prior to 1959, digitized mortality data is not available for the United States that are simultaneously disaggregatable by age, sex, and month.

⁴ The exact onset and cessation of the flu season – by which we mean epidemiologically significant influenza virus transmission – varies from year to year. Thompson et al. (2009: 38) use October to April, but we prefer, for symmetry, to have six-month pseudoseasons. There is not much flu transmission in October in the United States (Simonsen et al. 2005: 268). Furthermore, Nunes, Natário, and Carvalho note: “In the absence of any reliable external source of information to define the E_a [i.e., transmission] periods one should definitely choose the fixed period approach” (2011: 343). Since most of our data precedes influenza viral monitoring, we definitely lack such external source data, so our approach is best-practice. Moreover, evaluations have shown that fixed versus variable winter-season approaches produce very similar results for flu-specific disease burden estimates (Newall, Viboud, and Wood 2010).

December–March for pseudowinter and June–September for pseudosummer. This would provide greater winter–summer contrast but has the disadvantage of leaving some data unused. More importantly, enhancing contrast is not our goal. We are trying to account for the overall influence of winter on mortality, whether there is contrast or not. What is more, cold weather occurs well before and after the winter astronomical season (i.e., between the winter solstice and vernal equinox), so for cold-related causes of death like thrombosis, a similar logic applies.

3. Results

Figure 2: Life expectancy ($e(0)$) time series by sex and by pseudoseason



Notes: The band enveloping the series is two years in height, centered on the calendar-year $e(0)$ estimates from the Human Mortality Database; it is not an uncertainty interval.

Figure 2 presents four $e(0)$ (life expectancy) time series: pseudowinter (solid) and pseudosummer (dashed), for both males (lower series, blue) and females (upper series, red). The gray tubes enveloping each sex are two-year-wide bands centered on calendar-year life expectancy from the Human Mortality Database (HMD); these are not uncertainty intervals. The top of the gray band represents the calendar year $e(0)+1$; thus, in our data, the summer pseudoseasonal life expectancy is never greater than one year above the neighboring calendar year life expectancy. Similarly, since the bottom of the gray band is the calendar year $e(0)-1$, it shows that, in this data set, winter pseudoseasonal life expectancy is always within one year of the neighboring calendar year $e(0)$. Using

the HMD $e(0)$ data as the center of the band also provides an external check of our life expectancy calculations because our pseudoseasonal data should fairly neatly sandwich the calendar-year series.

Figure 3 shows the difference between $e(0)$ in summers and their preceding winters (from the summer of 1960 minus the winter of 1959–1960, to the summer of 2014 minus the winter of 2013–2014); all comparisons are summer-to-previous-winter, although summer-to-following-winter analyses give similar results. There are three important features. First, no secular time trend is evident. Second, the data is strongly negatively autocorrelated: Declines are followed by increases, and vice versa. Third, in addition to higher life expectancy, women have a higher summer–winter difference, 0.99 ± 0.22 years, versus 0.64 ± 0.23 years for males (mean \pm SD).

Figure 3: Summer advantage over winter in pseudoseasonal life expectancy, females (upper series) and males (lower series)

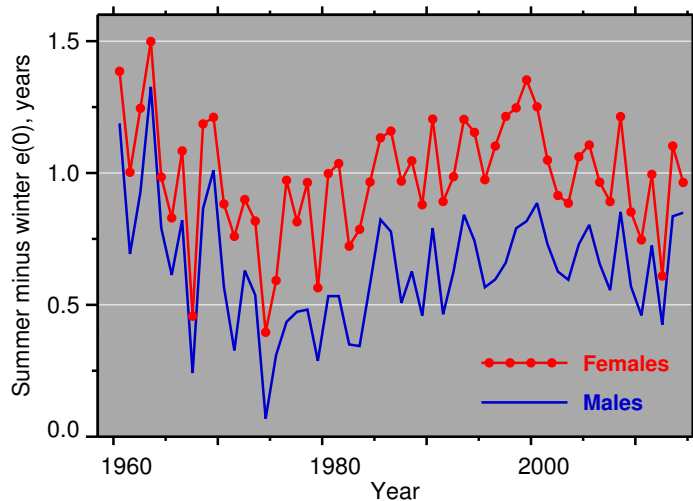


Figure 4 is a heat map of the winter–summer ratio of the mortality rate by age (M_x), over time. Several features of Figure 4 are especially relevant to seasonal differences. First, summer advantage in mortality is an age-related phenomenon. At younger ages (approximately 5–35), summers are more deadly. The summer excess is more pronounced for males, and is declining over time. It is particularly noticeable in the so-called accident bump (Pampel 2001). Indeed, summer mortality at younger ages is associated with motor vehicle fatalities (Farmer and Williams 2005) and external causes generally (Feinstein

2002). Winter overtakes summer above age 45, where death rates are (much) higher in absolute terms.

Figure 4: Heat map of winter–summer ratio of M_x , 1960–2014

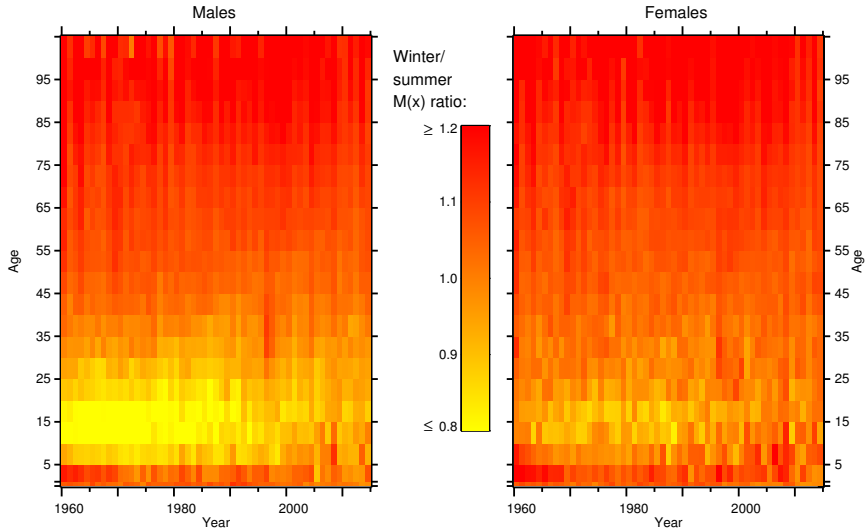


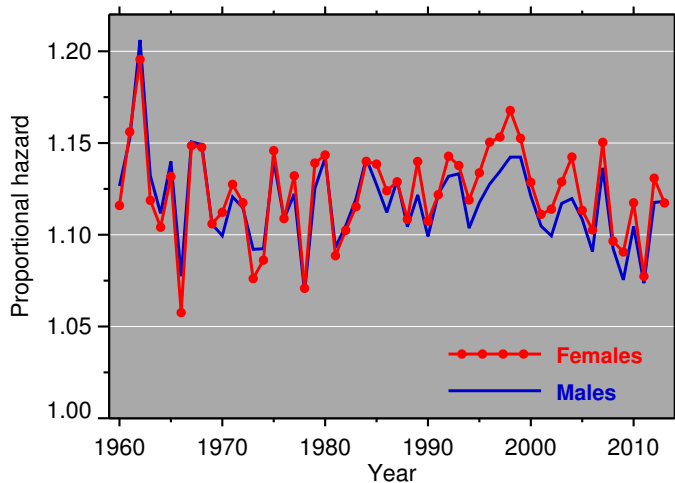
Figure 5 helps quantify the pseudoseasonal differences seen in Figure 4. Here we present, on a year-by-year basis, the proportional hazard (P^Y) of pseudoseasonal mortality for age ≥ 45 , separately by sex. Thus, we model $\mathbf{W} = P^Y \mathbf{S}$, where \mathbf{W} is the N -element (age-wise) vector of winter death rates for a given year, P^Y is the year-specific proportional hazard (scalar), and \mathbf{S} is the vector of summer death rates. The proportional hazard is estimated as:

$$P^Y = \exp \left(\frac{\sum_{x=45}^{\omega} [\log (M_x^W) - \log (M_x^S)]}{N} \right), \quad (1)$$

where the superscripts (W, S) refer to winter and summer, and M_x is the age-specific death rate. The proportional hazard is the same as the winter-to-summer ratio of the geometric mean death rate (see Schoen 1970).⁵ Figure 6 gives an example of how P^Y works, showing winter and summer pseudoseasonal M_x as well as ‘simulated’ winter: $M_{x,\text{simu}}^W = M_{x,\text{data}}^S P^Y$.

⁵ Note that this is not a Cox proportional hazard model, nor is there any statistical modeling in the calculation of P^Y beyond that given in equation 1.

Figure 5: Proportional hazard analysis

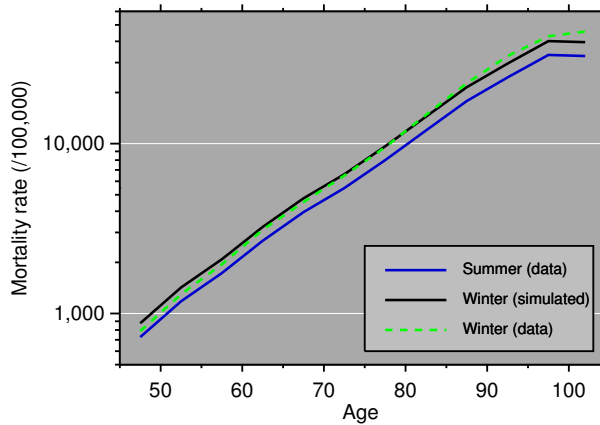


Note: The proportional hazard is winter death rates above age 45 as a multiple of summer death rates.

Figure 6 shows good agreement between simulated and true winter, especially between ages 60 and 90. These are the ages (among $x \geq 50$) at which most deaths occur. (Below age 60 there are fewer deaths because rates are lower, and above age 90 because exposures are lower.) Figure 5 shows that most winters have a mortality pattern that is between 110% and 115% of the previous summer's mortality. The proportional hazard model is an excellent fit, with all the year-specific $R^2 > 0.99$, which is not especially surprising since it is based on pairwise comparisons of adjacent pseudoseasons. The year-by-year goodness of fit (R^2) statistics are given in Figure 7. This shows that the proportional hazard approach is an excellent fit for both sexes and over time. Females are a better fit in almost every year. There are no meaningful sex differences in the proportional hazard.⁶ If we drop the Y superscript and model a single proportional hazard for the entire data set, then \mathbf{W} and \mathbf{S} become year \times age matrices, and P is 1.119 for males and 1.124 for females. Naturally, when modeling the whole time span with a single P , the goodness of fit declines, but it is still not poor: $R^2 = 0.89$ for males and $R^2 = 0.88$ for females.

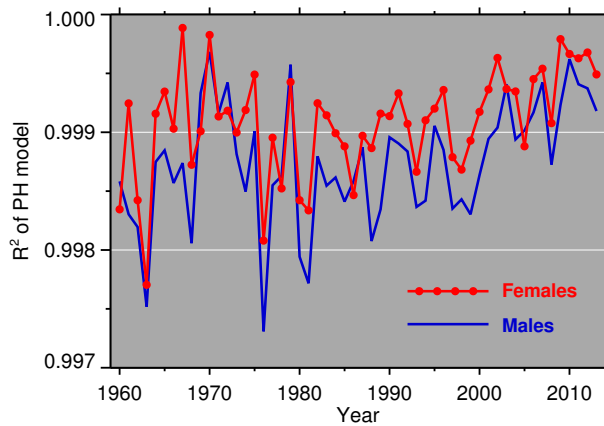
⁶ This is in contrast to Denmark, where Rau and Doblhammer (2003) find greater seasonal fluctuations for males.

Figure 6: Proportional hazard analysis



Note: Example graph showing M_x by age (males, ≥ 45), for summer 1962 and winter 1962–1963, as well as 'simulated' winter data, which is the summer data multiplied by the constant proportional hazard, P^Y . This year was chosen because it has the largest P^Y value (cf. Figure 5).

Figure 7: Proportional hazard analysis, goodness of fit



Note: This gives R^2 of the comparison between real and simulated winter data, by year (see Figure 6 for an example).

4. Discussion

Taking only mortality into account, how much happier should an adult be during the summer? Death rates will be, typically, 10% to 15% higher in the winter (Figure 5). However, by the time summer arrives, up to half a year will have passed, and death rates will be higher due to aging, even half a year's worth. How does the winter–summer mortality difference compare to age-related changes? We propose calculating ‘equivalent ages,’ as follows. In Table 1, the M_x columns give death rates by age, sex, and pseudoseason. The ‘w.e.a.’ columns give the winter equivalent age, or the age at which one would have to be in the winter to experience the same (summer) death rate. Similarly, the ‘s.e.a.’ columns give the summer equivalent age, or the summer age that experiences the same (winter) death rate. The M_x , w.e.a. and s.e.a. are calculated from a Gompertz mortality model estimated by Poisson regression, the coefficients of which are given at the top of the table.⁷ Symbolically:

$$M_x^S = \exp(\hat{\alpha}^S + \hat{\beta}^S \cdot x^S) \quad (2)$$

$$M_x^W = \exp(\hat{\alpha}^W + \hat{\beta}^W \cdot x^W) \quad (3)$$

$$\text{w.e.a.}(x^S) = (\hat{\alpha}^S - \hat{\alpha}^W + \hat{\beta}^S \cdot x^S) / \hat{\beta}^W, \quad (4)$$

where S, W superscripts are for summer and winter, x is age, and $\hat{\alpha}, \hat{\beta}$, are estimated coefficients. The solution for w.e.a. (x^S) in (4) comes from setting mortality rates (i.e., [2] and [3]) equal, and solving for x^W in terms of x^S and the estimated coefficients. Thus, if w.e.a. (x^S) is plugged into 3 for x^W , it will produce a death rate equivalent to the desired M_x^S . The same formula holds, mutatis mutandis, for s.e.a. (x^W). The (winter–summer) equivalent age is a function of the estimated Gompertz coefficients for both pseudoseasons and of the age for which an equivalency is being calculated.

Table 1 gives specific examples, using 2010 data. An 80-year-old woman in the winter experiences death rates of an 81-year-old woman in the summer. Death rates are higher in the winter, so the equivalent age in the summer is older. The difference is one year of age. On the other hand, a 70-year-old man living in the summer has death rates equivalent to a 69.1-year-old man in the winter. Summer mortality is more lenient, and therefore it's as if he is a younger man, compared to winter. The absolute value of the difference between biological age and w.e.a. or s.e.a. becomes larger as biological age increases, since M_x increases exponentially with age.

There is a micro–macro disconnect here: For populations, mortality is clearly lower in the summer, holding age constant, and holding the pair of pseudoseasonal life tables

⁷ That is to say, we estimated a Poisson regression like: $\log(D_x) = \alpha + \beta x + \log(K_x)$ where D_x are deaths in the age group centered on x , K_x is the exposure (person-years) for that age group, and α, β are the coefficients to be estimated; cf. Abdullatif and Noymer 2016: 207. This is a Poisson rate regression, with exposure as an offset (see Agresti 2002: 385).

constant. From the point of view of an individual, holding age constant is meaningless; one cannot go from winter to summer without aging approximately half a year. Thus, the winter-into-summer mortality changes experienced by an individual in the above thought experiment are less than the ceteris paribus analysis represented by the proportional hazards (and the summer-into-winter changes, more). Moreover, this ignores long-term trends; it only considers seasonal and age-related changes.

Table 1: Equivalent age analysis for 2010, as explained in the main text

Women					Men			
Age (x)	M_x	w.e.a.	M_x	s.e.a.	M_x	w.e.a.	M_x	s.e.a.
50	231.1	49.40	245.1	50.60	428.6	49.53	446.8	50.48
60	612.6	59.27	658.7	60.74	1,021.9	59.32	1,085.5	60.70
70	1,623.7	69.13	1,770.0	70.89	2,436.0	69.11	2,637.0	70.91
80	4,303.9	78.99	4,756.4	81.03	5,807.4	78.89	6,406.2	81.13
90	11,408.1	88.85	12,781.3	91.17	13,844.4	88.68	15,563.1	91.35

Notes: M_x is modeled death rate per 100,000; w.e.a. is 'winter equivalent age,' or the age at which an individual would experience the same death rate, living in the winter, and s.e.a. is the same, mutatis mutandis, for living in the summer. Coefficients from a Poisson regression; the prediction equation is $M_x = \exp(\alpha + \beta x)$.

In terms of life expectancy, the effect of the winter increase in mortality is similarly modest: on average, just over one year of life expectancy for women and just under one year for men. If we could wave a magic wand, making the winter pattern of mortality look like the summer pattern, this would be equivalent to about seven years' worth (in terms of time) of recent mortality progress (i.e., based on the slopes of Figure 2).

The reason for this modest difference is easy to see, at least in retrospect. If we could eradicate winter-related mortality, then it would be like living in the summer. To put it another way, people would experience their 'summer equivalent age,' as in the example above. Although reducing death rates by about 12% seems like a great thing, it only makes a small difference in equivalent age and, therefore, has a modest effect on $e(0)$. Mindel Sheps's (1958) observation that changes in death rates usually are smaller when viewed through the lens of concomitant changes in survival rates, is highly relevant. The appreciable pseudoseasonal difference in M_x results in a rather modest difference in $e(0)$ because life expectancy is the integral of the life table l_x , or survivor, column, not the M_x column. The Gompertzian relationship that holds above age 45, where by far the majority of deaths occur, guarantees that age-associated increases in mortality would swamp the hypothetical change generated by eradication of winter excess mortality.

The insight from our approach lies not in the idea that elimination of seemingly major causes (in this case, approximated by pseudosummer) will have a small impact on $e(0)$. This is well understood; for example, Keyfitz (1985: 62–72) considers it in relation to the Shannon entropy, H , of the life table ℓ_x column. Because of competing risks of, say, heart disease, even eradicating cancer does not cause huge changes in $e(0)$ (ibid.), so it is clear that removing winter deaths also won't have a big effect. Vaupel's study (1986) of the relation between $e(0)$ and M_x is also relevant (see also Pollard 1982). The greater mortality seasonality of women as measured by life expectancy differences (Figure 3) than as measured by the winter:summer proportional hazard (Figure 5), is consistent with this; the effect of a constant multiple of M_x affects $e(0)$ differently at different levels of M_x . What the present study shows, however, is that the total mortality impact of winter (viz., including knock-on effects on heart disease) is not very large in the grand scheme of things.

Another approach to estimating the role of viruses in all-cause mortality would be to use direct measures of viral circulation (as in Figure 1) instead of summer and winter as instrumental indicators. This would have the disadvantage of not being applicable to historical data. (Viral surveillance like that shown in Figure 1 begins in the late 1990s.) On the other hand, an advantage is that it could be applied to the tropics, where influenza circulation is more haphazard (see, e.g., Aungkulanon et al. 2015), and therefore the colinearity of flu season and 'winter' is neither an appropriate identification strategy nor a lurking problem. Using nominal influenza mortality as an instrument instead of the seasons is another possibility, but it is not without its problems (Noymer and Nguyen 2013). Our results are broadly consistent with other seasonal analyses of mortality in the United States, going back to Rosenwaike (1966), as well as more recent work (Feinstein 2002).

This study has a number of strengths and weaknesses. The principal strength is that it uses all-cause mortality and so automatically includes any winter-related deaths that would be missed by studying an ensemble of winter-dominant causes like influenza and thrombosis. One limitation is that we can observe only summers that follow winters, and vice versa. The more lenient mortality of the summer leads to the accumulation of frail individuals who then become more likely to die in the winter. Similarly, the more severe mortality of the winter leaves a more robust residual population, less likely to die in the summer; here we use 'robust' and 'frail' in the usual demographic sense (Keyfitz and Littman 1979; Vaupel, Manton, and Stallard 1979; Vaupel and Yashin 1985; Manton, Stallard, and Vaupel 1986). Thus, diminution of winter mortality from, for example, the invention of a perfect influenza vaccine could precipitate small increases in summer mortality as a result of perturbing the frail/robust cycle. Goldstein et al. (2012: 833) likewise speculate that these effects are limited in magnitude. The negative autocorrelation seen in Figures 3 and 5 may well be driven by similar effects. It is also possible that influenza and other respiratory pathogens are underascertained in the summertime, and

thus that the roots of seasonal mortality are misunderstood, although this seems unlikely given Figure 1 and other work (Hayward et al. 2014).

Using data from the entire United States is both a strength and limitation. If the intent is to draw conclusions regarding the whole United States, then using data from the entire country is clearly appropriate. Given the size and climatic diversity of the United States, idiosyncratic factors such as summer heat waves tend to get averaged out, which can be regarded as a positive given our focus on winter. However, the national approach also averages out the potentially interesting relation between winter temperature and the coefficient of seasonal variation in mortality that is seen in Europe (cf. the literature review).

5. Conclusion

It is reasonably well established that at least part of the reduction in cardiovascular mortality during the summer is due to the absence of influenza virus (Warren-Gash, Smeeth, and Hayward 2009; Warren-Gash et al. 2011). The connection between influenza vaccine use and reduction of wintertime heart mortality is less clear but has been studied (Seo et al. 2014). Influenza vaccine use is correlated with general health-seeking behavior, and so confounding is a problem in direct empirical approaches to this question. In this study, we took an indirect approach, using whole-population data, and summers as a sort of natural experiment. This quantifies the mortality impact of winter, not of influenza per se, which is smaller.

Progress is being made toward a much better flu vaccine (Keitel et al. 1997; Treanor 2004; Pica and Palese 2013). A universal flu vaccine (as such shots are called) is a clearly expressed public health desideratum (Fineberg 2014) but remains on the drawing board. Realistic expectations for mortality changes should be part of the policy analysis in this area. The mortality burden of winter, quantified herein, includes a number of factors of which influenza is only one; universal flu vaccines, even if successful, will have a modest impact on life expectancy.

In conclusion, the effect of winter on life expectancy in the United States is less than 1.25 years for women and less than 1.0 year for men. This estimate is based on summer–winter differences and so implicitly includes the synergies among causes that can be hard to measure using a bundle of cause-specific approaches. The insalubrious effects of winter include not only respiratory viruses but also air pollution as well as the linkage between cold and thrombosis, affecting, in turn, heart attack and stroke. The mortality impact of winter, quantified in terms of life expectancy, is neither negligible nor enormous.

6. Acknowledgments

The idea for this paper is an offshoot of a stimulating conversation with Viggo Andreasen. For helpful suggestions, we thank Bob Schoen and Monica He King, as well as seminar audiences at Ohio State, Université de Montréal, and at UC Irvine (both the Health Policy Research Institute and the Institute for Mathematical Behavioral Sciences). Tim Bruckner, Duncan Thomas, Philip Stark, and Amar Hamoudi provided useful feedback. Ken Wachter provided especially detailed comments. Carter Butts suggested the title. Rahema Haseeb provided research assistance. We thank the anonymous peer reviewers for excellent suggestions.

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Appendix: Denominator construction

The numerators (i.e., the death counts) are recorded monthly, so aggregation of these into pseudoseasons is completely straightforward. Our denominator, or exposure, values (i.e., person-years at risk) come from the Human Mortality Database (HMD), and are for calendar years. To recast these into pseudoseasons, we followed the following graduation/aggregation procedure.

The HMD exposure values for each year are centered at mid-year (i.e., July 1st), and are one year (of time) wide. We interpolated these values between consecutive Julys by fitting an exponential function using the two July 1st values, K_0 and K_1 . That is to say, we estimate the function $K_t = K_0 \exp(rt)$ where K_t is the calendar-year-wide exposure, centered at an arbitrary time, K_0 is the earlier of the two July 1st exposures, t is time since July 1st, and r is the growth rate, which works out to $\log(K_1/K_0)$. We did this repeatedly, always estimating r from the two July 1st values that sandwich the months being interpolated. While it is possible to fit r from three or more Julys, doing it pairwise ensures that the fitted curve passes through both K_0 and K_1 . The goal here is interpolation, not smoothing, so by repeatedly fitting off two points we stick as close as possible to the data. As described so far, the procedure produces K_t values centered on any month desired, but these are still one year wide, each. These values were then adjusted to months by multiplying by 31/365 for January, 28/365 for February, and so on. In leap years, 31/366, 29/366, . . . , were used.

In practice, these values are not far off simply averaging consecutive K values and dividing by 12 (especially post-aggregation into pseudoseasons), but nonetheless, doing the interpolation and using days per month gives more accuracy. The months were then aggregated into November through April for pseudowinter and May through October for pseudosummer. All these calculations were performed with HMD '1×1' exposures, that is to say, values for one year of age and one year of time. Therefore, these were aggregated by age (into 5-year age groups) as well as by pseudoseason.

