Cardiac Mortality Is Higher Around Christmas and New Year’s Than at Any Other Time

The Holidays as a Risk Factor for Death

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Background—Research published in Circulation has shown that cardiac mortality is highest during December and January. We investigated whether some of this spike could be ascribed to the Christmas/New Year’s holidays rather than to climatic factors.

Methods and Results—We fitted a locally weighted polynomial regression line to daily mortality to estimate the number of deaths expected during the holiday period, using the null hypothesis that natural-cause mortality is unaffected by the Christmas/New Year’s holidays. We then compared the number of deaths expected during the holiday period, given the null hypothesis, with the number of deaths observed. For cardiac and noncardiac diseases, a spike in daily mortality occurs during the Christmas/New Year’s holiday period. This spike persists after adjusting for trends and seasons and is particularly large for individuals who are dead on arrival at a hospital, die in the emergency department, or die as outpatients. For this group during the holiday period, 4.65% (95% CI, 4.06% to 5.24%) more cardiac and 4.99% (95% CI, 4.17% to 5.81%) more noncardiac deaths occur than would be expected if the holidays did not affect mortality. Cardiac mortality for individuals who are dead on arrival, die in the emergency department, or die as outpatients peaks at Christmas and again at New Year’s. These twin holiday spikes also are conspicuous for noncardiac mortality. The excess in holiday mortality is growing proportionately larger over time, both for cardiac and noncardiac mortality.

Conclusions—Our findings suggest that the Christmas/New Year’s holidays are a risk factor for cardiac and noncardiac mortality. There are multiple explanations for this association, including the possibility that holiday-induced delays in seeking treatment play a role in producing the twin holiday spikes.

Key Words: epidemiology ■ mortality ■ heart diseases ■ patients ■ holidays

Every year, during the Christmas/New Year’s holiday season, millions of Americans abruptly change their patterns of traveling, eating, drinking, exercising, working, and vacationing.1,2 These large-scale behavioral changes may affect cardiac mortality. Some patients might inappropriately delay seeking necessary medical treatment until after the holidays3–7; others who are traveling might take longer than usual to find competent medical help.8 Despite these considerations, we found no previous studies that determined whether the Christmas/New Year’s holiday season affects cardiac (or noncardiac) mortality.

See p 3744

Two bodies of literature are linked to this topic, but only indirectly: (1) Some studies indicate that suicides, homicides, and automobile fatalities increase during the winter holidays.9–11 but these studies do not attempt to determine whether natural-cause mortality also increases during the holiday season. (2) The Christmas/New Year’s holiday season occurs during winter, and the effects of winter on natural-cause mortality have been studied extensively.12–17 These investigations do not attempt to determine whether the winter holiday period has an effect on natural-cause mortality separate from the effects of winter itself.

In the present article, we seek to provide separate measures of these effects. We build on earlier work published in Circulation by Kloner et al,17 who studied daily coronary heart disease (CHD) deaths in Los Angeles, Calif. These authors found that CHD mortality reached an absolute peak “around the winter holiday period,”17 but they did not find separate peaks at Christmas and New Year’s. Kloner et al hypothesized that some of the CHD mortality peak occurred because of behavioral changes, but their study design did not allow them to test this hypothesis. In addition, they made no formal attempt to measure the separate effects on CHD mortality of winter itself versus the winter holiday season.
We investigated questions raised but not answered by Kloner et al in this journal: Is there a peak, not only in CHD mortality but also in mortality from other types of heart disease and nonheart diseases? Is there a peak, not only in Los Angeles but also across the entire United States, and is this peak larger in the states with colder climates? How big is the mortality effect of the winter holidays, separate from the effect of winter weather? If there is a holiday peak in cardiac and noncardiac mortality, is the peak growing over time? If there is a holiday peak, can this peak be linked to behavioral changes?

To investigate these questions, we used death certificates to examine daily nationwide mortality. Following Kloner et al, we focused on cardiac deaths. In contrast to Kloner et al, we examined both CHD and non-CHD, both heart and nonheart diseases, a larger dataset (n=53 million versus their n=220 000), a longer time period (1973 to 2001 versus their 1985 to 1996), and the United States rather than only Los Angeles.

**Methods**

The National Center for Health Statistics maintains a computerized administrative database of death certificates. Using this database, which covers all US deaths, we examined daily mortality throughout the year and focused on the “holiday period,” predefined as the 2 weeks from December 25 to January 7. We adopted January 7 as the end point of our study period rather than January 1 because any effects of the holidays on health may take some time to appear. Our study period begins in 1973, the first year for which exact day of death was recorded on all computerized death certificates, and ends in 2001, the last year for which computerized death certificates were available.

We examined all holiday periods between July 1, 1973, and June 30, 2001, with 2 exceptions. The disease classification scheme shifted from ICD-8 to ICD-9 (on January 1, 1979) and from ICD-9 to ICD-10 (on January 1, 1999). These shifts in International Classification of Disease measures produced marked coding changes for some diseases. Hence, we did not examine mortality during the Christmas/New Year’s holidays for the transitional periods between classification schemes: July 1, 1978, to June 30, 1979, and July 1, 1998, to June 30, 1999. This procedure abridges the dataset but eliminates a confounding factor, and the dataset remains large at 53 million deaths.

We focused on heart disease but also examined all natural causes of death. In some analyses, we examined death certificates that list both heart disease and additional, secondary causes of death such as influenza; these analyses begin in 1983, the first year in which computerized death certificates listed secondary causes of death.

We fitted a locally weighted polynomial regression (LOESS) line to daily deaths from January 1, 1973, through December 31, 2001. Use of this standard nonparametric smoothing procedure has several benefits: The procedure makes minimal distributional assumptions about the data, it corrects for the influence of trends and seasonal factors on mortality, and it enables us to estimate the number of deaths that would be expected during the holiday period, given the null hypothesis that natural-cause mortality is unaffected by the Christmas/New Year’s holidays.

The LOESS procedure requires the choice of a bandwidth (roughly speaking, the span of data over which the local averaging takes place). Choosing a bandwidth that is too wide (ie, oversmoothing) would flatten the regression curve near a peak, and would consequently magnify the apparent size of any holiday spike. We proceeded conservatively, choosing a narrow bandwidth of 6 weeks, and thus undersmoothed according to conventional guidelines.

This undersmoothing mitigates any bias in the estimate of excess holiday mortality. As an additional check, we reanalyzed our key findings with an exceptionally conservative bandwidth of 1 week to ensure that our findings remained statistically significant at 0.05 or better. We compared the number of deaths observed during the 2-week holiday period with the number expected under the null hypothesis: 100 × [observed number of deaths − expected number of deaths] / expected number of deaths.

For convenience, we call this statistic “the holiday effect.” A holiday effect of, for example, 5% would indicate that 5% more deaths occurred during the holiday period than would be expected if the holidays had no effect on mortality.

Following official recommendations and our earlier practice, we calculated standard errors and significance levels, even though we examined complete counts, not samples. As in our previous work, the research design allowed examination of numbers of deaths rather than death rates.

**Results**

In Figure 1, the solid line indicates the observed number of cardiac deaths for each day of the year, and the dotted line indicates the number of cardiac deaths predicted by the null
Size of the Holiday Effect for Cardiac and Noncardiac Mortality, by Year

<table>
<thead>
<tr>
<th>Year</th>
<th>Heart*</th>
<th>Nonheart</th>
<th>All Natural</th>
</tr>
</thead>
<tbody>
<tr>
<td>1973</td>
<td>-0.44</td>
<td>0.26</td>
<td>-0.04</td>
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<tr>
<td>1974</td>
<td>1.36</td>
<td>1.15</td>
<td>1.24</td>
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<tr>
<td>1975</td>
<td>1.94</td>
<td>0.07</td>
<td>0.86</td>
</tr>
<tr>
<td>1976</td>
<td>1.90</td>
<td>1.18</td>
<td>1.48</td>
</tr>
<tr>
<td>1977</td>
<td>2.09</td>
<td>1.51</td>
<td>1.76</td>
</tr>
<tr>
<td>1979</td>
<td>0.89</td>
<td>-0.23</td>
<td>0.24</td>
</tr>
<tr>
<td>1980</td>
<td>5.19</td>
<td>3.13</td>
<td>4.00</td>
</tr>
<tr>
<td>1981</td>
<td>-0.90</td>
<td>-0.59</td>
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</tr>
<tr>
<td>1982</td>
<td>2.09</td>
<td>-0.07</td>
<td>0.83</td>
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<tr>
<td>1983</td>
<td>4.29</td>
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<td>3.64</td>
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<td>1984</td>
<td>1.01</td>
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<td>1985</td>
<td>2.79</td>
<td>0.18</td>
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<tr>
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<tr>
<td>1987</td>
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<td>3.05</td>
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</table>

The holiday effect is calculated for the 2-week holiday period, December 25–January 7, and equals 100 × [(observed number of deaths — expected number of deaths) ÷ expected number of deaths]. The expected number of deaths is determined from the LOESS regression procedure described in the text.


hypothesis. Aside from the 2-week holiday period, the solid and dotted lines agree closely. For the 351 days outside the holiday period, observed and expected daily mortality levels correlate =0.995 (P<0.000001). Thus, our regression procedure corrects well for trends and seasonal influences and accurately predicts cardiac mortality outside the holiday period.

Figure 1 shows 2 distinct spikes in cardiac mortality: one around Christmas and one around New Year’s. Because so many deaths are examined (≈55 000/day), the standard error for each daily count is small (≈235, or 0.4% of the daily count). Consequently, the observed number of deaths during the holidays is many standard errors above the number expected under the null hypothesis.

Excess holiday mortality is evident not only when the data-years are summed, as in Figure 1, but also when each data-year is examined separately. We studied holiday mortality in 26 separate years; for 24 of the 26 years, the observed number of deaths during the holidays exceeded the number expected under the null hypothesis (P=0.00001, binomial test). Full details on each year’s holiday effect are provided in the Table.

The percentage excess in holiday mortality has gradually increased during these 26 years. The size of the holiday effect correlates with year of death (Spearman r =0.492; P<0.02, 2-tailed test). In the latest 3 years, observed holiday mortality was 4.4% above the number expected; in the earliest 3 years, holiday mortality was only 0.95% above the number expected.

The excess in cardiac holiday mortality remains statistically significant (both on a year-by-year basis [P<0.05] and for the summed yearly data [P<0.05]) even when an exceptionally conservative bandwidth of 1 week is substituted for the 6-week bandwidth we have used. When a 1-week bandwidth is used, the LOESS regression line exhibits “wiggles,” a classic indication of a bandwidth that is too narrow (details available on request). Thus, the holiday peak, first found by Kloner et al for CHD in Los Angeles,17 is evident nationwide, not only for CHD (2.46% more deaths than expected [SE ±0.12%; 95% CI, 2.22% to 2.69%]) but also for non-CHD deaths (2.81% [SE ±0.25%; 95% CI, 2.32% to 3.30%]).

The double spike on Christmas and New Year’s is particularly striking for cardiac deaths that occur rapidly after presentation of the medical problem (ie, individuals who are dead on arrival [DOA] or die in the emergency department [ED] or as outpatients; Figure 2). For this DOA/ED/outpatient group, more cardiac deaths occur on December 25 than on any other day of the year; the second largest number of cardiac deaths occurs on December 26, and the third largest number occurs on January 1. (A more detailed examination of this double spike is provided later in Figure 6.) For inpatients, no obvious double spike occurs on Christmas and New Year’s, although a dispersed spike takes place during the holiday period and just afterward (Figure 3).

For DOA/ED/outpatients, 4.65% more cardiac deaths (±0.30%; 95% CI, 4.06% to 5.24%) occur during the holiday period than would be expected from the dotted regression line. For inpatients, this cardiac holiday peak is 1.60% (±0.21%; 95% CI, 1.19% to 2.01%). Information on nursing facility residents is available only for 1989 to 2001 (versus 1979 to 2001 for inpatients and DOA/ED/outpatients). Nursing facility residents also produce a cardiac holiday spike (3.72±0.38%; 95% CI, 2.97% to 4.46%).

Possible Explanations for the Cardiac Holiday Peak

Kloner et al17 proposed that colder temperatures cannot explain the holiday peak because daily CHD mortality correlated only weakly with daily temperatures during December and January in Los Angeles. Our data support their proposition for the following reasons: (1) The climatic hypothesis cannot easily explain the twin mortality spikes on Christmas and New Year’s. (2) The cardiac mortality peak exists after correction for seasonal fluctuations. (3) The cardiac mortality peak is slightly smaller in the northern border states (states that bordered Canada) than in the southern border states (states that bordered Mexico or the Gulf of Mexico) (2.22%...
versus 3.10%). The cardiac holiday effect pervades the United States, and the size of this effect varies insignificantly from region to region: northeast (2.32%; 95% CI, 1.88% to 2.75%), south (2.66%; 95% CI, 2.29% to 3.03%), midwest (2.29%; 95% CI, 1.87% to 2.72%), and west (2.86%; 95% CI, 2.32% to 3.39%).

Kloner et al proposed but did not test 4 additional explanations, which we assess below.

- **Respiratory diseases.** Respiratory diseases increase during winter, and patients weakened by respiratory diseases can die from cardiac diseases. The respiratory hypothesis is undermined by 2 considerations: (1) People dying from cardiac diseases with respiratory disease listed as a secondary cause of death produce a smaller holiday peak than do people dying from cardiac diseases alone: 3.51% versus 3.77%. (2) Interaction between cardiac and respiratory diseases cannot easily explain the twin mortality spikes on Christmas and New Year’s.

- **Emotional stresses associated with holidays.** It seems plausible that people with Alzheimer’s disease are less aware of holidays than are people without Alzheimer’s disease. Thus, given the hypothesis that emotional stress is associated with holidays, the holiday peak should be relatively smaller for people dying from cardiac diseases with Alzheimer’s disease listed as a secondary cause of death. The cardiac peak, however, is slightly larger when Alzheimer’s disease is listed as a secondary cause than it is for people dying from cardiac diseases alone: 3.97% versus 3.77%.

- **Changes in diet and alcohol consumption.** This explanation is undermined by the following findings: (1) Inpatients, whose diet and alcohol consumption are strictly regulated, produce a holiday peak (Figure 3). (2) People dying from

Figure 2. Daily US cardiac deaths, 1979–2001, for DOA/ED/outpatients. Solid line indicates the observed number of deaths for each day of the year, summed over the study period. Dotted regression line indicates the expected number of deaths for each day, given seasonal fluctuations and the null hypothesis that mortality is unaffected by the holidays.

Figure 3. Daily US cardiac deaths, 1979–2001, for inpatients. Solid line indicates the observed number of deaths for each day of the year, summed over the study period. Dotted regression line indicates the expected number of deaths for each day, given seasonal fluctuations and the null hypothesis that mortality is unaffected by the holidays.
cardiac diseases with substance abuse listed as a secondary cause of death produce a smaller holiday peak than do people dying from cardiac diseases alone: 3.46% versus 3.77%.

- **Increased particulate pollution.** The increase in particulate pollution during the winter might be consistent with a general increase in winter mortality, but this hypothesis cannot easily explain the twin mortality spikes on Christmas and New Year’s.

We considered 5 additional explanations not proposed by Kloner et al, as follows.

- **Month boundary effect.** Deaths generally peak at the beginning and dip at the end of each month. If a “month boundary effect” could explain the holiday peak, then the equivalent of a holiday peak should occur at every month boundary, not only at the December/January boundary. To test this hypothesis, we applied our regression procedure to 11 dummy holiday periods, each centered on a different month boundary (February 1, March 1, etc). For example, instead of using December 25 to January 7 as the holiday period, we substituted January 25 to February 7 as the dummy holiday period, and then re-ran the regression procedure. On average, for the 11 dummy holidays periods, no mortality peak occurred; the observed mortality almost exactly equals the level expected (observed/expected = 0.999; SD = 0.0023). The mortality peak observed for the real Christmas/New Year’s holiday period is far larger than the peak at any of the other month boundaries. Thus, the month boundary effect cannot account for our findings.

- **Reporting artifact.** The holiday peak does not result from misreporting of death dates because the peak is evident for inpatients, whose death dates are particularly likely to be recorded accurately.

- **Postponement of death.** Perhaps the holiday peak occurs because some patients postpone death to reach an important occasion. Given this explanation for the peak, mortality levels should dip immediately before the holiday period, and the preholiday dip should be about the same size as the holiday peak. These expectations are not supported by the evidence shown in Figures 1 through 3. The postponement hypothesis may also be undermined by other data. As noted above, it seems plausible that people with Alzheimer’s disease are generally less likely than others to be aware of the holidays and thus should be less likely than people without Alzheimer disease to try to postpone death to reach these holidays. Thus, given the postponement hypothesis, the holiday peak should be relatively smaller for cardiac patients with Alzheimer’s disease. As noted above, however, this hypothesis is faulty. The cardiac peak is larger when Alzheimer’s disease is listed as a secondary cause than it is for people who die from cardiac diseases alone: 3.97% versus 3.77%.

- **Precipitation of death.** Perhaps the holidays merely precipitate some deaths that would have occurred soon anyway. Such precipitation should produce a dip in deaths immediately after the holidays. A dip of this sort is evident but is much smaller than the holiday peak.

- **Inappropriate delay in seeking medical care.** Previous studies show that admissions to urgent care facilities drop on holidays and spike immediately thereafter. This phenomenon may occur because some patients inappropriately delay seeking medical services to avoid disrupting their holidays. Any holiday-induced delays in seeking medical care should affect not only cardiac deaths but also other deaths. Thus, given the delay-in-seeking-care hypothesis, natural noncardiac deaths also should display a holiday peak. Such a peak is indeed evident, both for DOA/ED/outpatients (Figure 4; 4.99 ± 0.42%; 95% CI, 4.17% to 5.81%) and for inpatients (Figure 5; 1.30 ± 0.14%; 95% CI, 1.03% to 1.57%). The noncardiac holiday peak constitutes an independent replication of the cardiac holiday peak because the death certificates that we used to generate Figures 4 and 5 are entirely different from the death certificates that we used to generate Figures 1 through 3.

For both cardiac and noncardiac diseases, the holiday peak is most evident for DOA/ED/outpatients. Figure 6 examines
DOA/ED/outpatients and provides a magnified view of cardiac and noncardiac mortality during the period immediately surrounding the winter holidays. Both types of mortality display twin holiday peaks, with the peak for Christmas being slightly larger than that for New Year’s. The number of cardiac deaths is higher on December 25 than on any other day of the year, second highest on December 26, and third highest on January 1. The pattern is similar for noncardiac deaths. The number of noncardiac deaths is highest on December 26 than on any other day of the year, the next highest occurs on December 25, and the third highest occurs on January 1.

We note additional similarities between cardiac and noncardiac mortality during the holiday period. The Table indicates for each year examined the size of the holiday peak for cardiac deaths, noncardiac deaths, and all natural deaths combined. As with cardiac mortality, the percentage excess in noncardiac holiday mortality is gradually increasing during the years under study. The size of the noncardiac holiday peak correlates with the year of death (Spearman $r=0.395; P<0.05, 2$-tailed test). In the latest 3 years, observed holiday mortality was 2.8% above the number expected; in the earliest 3 years, holiday mortality was only 0.50% above the number expected. For each year, the size of the holiday peak for cardiac mortality is strongly correlated with the size of the holiday peak for noncardiac mortality ($r=0.874, t=8.83, P<0.00001$). This strong correlation is also evident from the detailed data in Figure 7.

In sum, both cardiac and noncardiac deaths spike during the 2-week holiday period from December 25 through January 7. In the 26 years under study, 42 039 “excess” deaths occurred during this holiday period (95% CI, 39 098 to 44 980). In other words, our findings indicate that during the Christmas/New Year’s holiday periods from 1973 to 2001, 42 039 more deaths occurred than would be expected if the holidays did not affect mortality.

**Discussion**

For cardiac and noncardiac diseases, a spike in daily mortality occurs during the Christmas/New Year’s holiday season. This spike persists after adjusting for trends and seasonal factors and is particularly large for the DOA/ED/outpatient population. For this group during the holiday period, 4.65% ($\pm 0.30%$; 95% CI, 4.06% to 5.24%) more cardiac deaths and 4.99% ($\pm 0.42%$; 95% CI, 4.17% to 5.81%) more noncardiac deaths occurred than would be expected if the holidays did not affect mortality. DOA/ED/outpatient cardiac mortality is higher on December 25 than on any other day of the year, second highest on December 26, and third highest on January 1. These twin holiday spikes are also conspicuous for noncardiac mortality.

We considered 10 explanations for the holiday spike. Earlier studies3–7 suggested that some patients delay seeking treatment until after the holidays. These studies did not investigate whether such delays produced additional deaths. Our data suggest that holiday-induced delays in seeking treatment may contribute to additional cardiac and noncardiac deaths around the holidays. Thus, our findings extend the earlier literature by raising the possibility that holiday-induced delays in seeking treatment may have fatal consequences.

Our findings also extend and modify 2 other literatures: (1) European researchers12–15 have found an increase in winter mortality, but they have not sought to determine whether some of this increase results from the winter holidays rather than from winter itself. Future research should seek to disaggregate the effects of winter and the winter holidays. (2) Other research9–11 found that suicides, homicides, and accidents increase on Christmas, New Year’s, or both. This research examined 5% of all deaths; our study examined the remaining 95% of deaths and indicates that deaths from natural causes also spike during the holidays.

Delays in seeking treatment could result in sicker patients, some of whom die as inpatients. Thus, the inpatient holiday peak may be consistent with the delay-in-seeking-treatment hypothesis. This hypothesis, however, cannot easily explain the holiday peak in nursing facility residents’ deaths (3.72±0.38%; 95% CI, 2.97% to 4.46%). Some other processes may also play a role—for example, changes in medical staffing during the holidays.32,33 Future research should investigate the potential effect of these staff changes.
The epidemiological data used in this article are appropriate for examining a large (n=53 million), nationwide, multiyear dataset and for demonstrating the existence of a previously unknown double spike in cardiac and noncardiac mortality; however, our data are not appropriate for definitively identifying the detailed causes of this double spike. Future investigations should seek an answer to this question and to additional questions raised by the data in the Table and in Figure 7. For example, cardiac holiday peaks occurred in 24 of the 26 years under study but not in 1973 or 1981. Was this a fluke or are these years unusual in some way? The Organization of Petroleum-Exporting Countries’ embargo on exporting petroleum products to the United States and other countries included the holiday period from December 25, 1973, to January 7, 1974. Travel during the embargo was markedly reduced, and it also was reduced during the recession of 1981. If the holiday effect occurs in part because of delays in seeking medical treatment by travelers, then a reduction in travel may produce a reduction in the holiday effect. Future research should assess this possibility.

Potential explanations for the holiday effect need to be assessed further in follow-up investigations with different types of datasets, which provide more details on patients and their circumstances. In comparison with the large-scale dataset we have used, these follow-up datasets are likely to be richer in detail but more limited in size, geographic area, and time period. Even before these follow-up studies are performed, however, the current evidence seems sufficient to demonstrate that the Christmas/New Year’s holiday season is a risk factor for both cardiac and noncardiac mortality. Because this risk factor is growing with time, it seems particularly important to investigate it and control it.

Acknowledgments

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writing and revising of the article. The authors thank Evelyn and Robert Kleinberg, Christy G. Kwan, Kevin Lewis, and Miranda and Rachel Phillips for useful comments.

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