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The seasonality in heart failure deaths and total cardiovascular deaths in Australia

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Abstract

Objectives: To examine the seasonal pattern in heart failure (HF) and cardiovascular disease (CVD) by climate and cause of death in Australia.

Methods: A retrospective analysis of a national database of deaths in the eight Australian state and territory capitals between January 1997 and November 2004. We examined the seasonal pattern in HF and CVD deaths, we identified variations in the pattern by: sex, age, time, climate and cause of death (for total CVD using seven groups determined by ICD-10 code).

Results: Deaths in all seven groups of CVD significantly increased in winter. The largest increase in mortality rates was 23.5% observed for HF. The magnitude of this increase varied greatly between cities, with the highest winter mortality in the most temperature cities. The pattern in CVD deaths showed a clearer correlation with climate than HF deaths.

Conclusion and implications: Winters in Australia are mild but winter increases in HF and CVD are a significant problem. Increased blood pressure and lack of vitamin D in winter are the most likely causes of the increase. Reducing exposure to cold, particularly in the elderly, should reduce the number of winter CVD deaths in Australia.

Key words: Cardiovascular disease; seasonality; heart failure; hypertensive diseases; vitamin D
1 Introduction

Cardiovascular disease (CVD) shows a strong seasonal pattern in many parts of the world, with greatly increased rates in winter compared to summer.\(^1\) Similar strong seasonal peaks in admissions to hospital have also been observed in relation to heart failure.\(^2\)–\(^5\) Counter-intuitively the seasonal pattern is strongest in regions with warmer climates.\(^6\),\(^7\) For example, a strong association between relatively cold temperatures and cardiovascular disease was found in Newcastle, Australia, but no association was found in three populations in Finland.\(^1\)

Winter increases in heart failure (HF) admissions have been found in France,\(^2\) Spain,\(^3\) Scotland,\(^5\) Japan,\(^4\) and Argentina.\(^8\) In France HF deaths were 20% above average in January and 15% below average in August.\(^2\) In Madrid HF hospitalisations were 25% above average in January and 33% below average in August.\(^3\) In Scotland HF hospitalisations were 8% to 12% above average in December and 7% to 8% below average in July, and the seasonal increase was 23% to 35% larger in the elderly.\(^5\) In Japan HF admissions were highest in Spring and lowest in Summer and Autumn.\(^4\)

The cause of the winter increase in HF and CVD remains in doubt. Colder temperatures are not the only possible cause, as a number of other CVD risk factors change over the seasons.\(^9\) A summary of the putative pathways between winter and cardiovascular disease is given in Figure 1 (adapted from Scragg\(^10\)). There are accepted strong associations (in any season) between
CVD and the five risk factors of body mass index (BMI), cholesterol, blood pressure, inflammation (e.g., fibrinogen) and heart rate. However, it is not known which risk factor (or factors) are the cause of the seasonal change in CVD, or what seasonal exposures are driving the seasonal change in risk. A number of exposures have been proposed, those shown in Figure 1 are physical activity,\(^{11}\) sunlight and vitamin D,\(^{12}\) temperature,\(^{11,13}\) influenza\(^{14}\) and air pollution\(^{15}\) (which may also interact with temperature\(^{16}\)).

The number of possible pathways between winter and increased CVD has made it difficult to identify the cause of the seasonal change in CVD. In this paper we aim to add to the evidence by looking at seasonal patterns in CVD by cause of death and climate. Although previous studies have examined the effect of climate on seasonal changes very few have examined cause of death. We also examine the seasonal pattern in HF, and compare the results to CVD.

2 Methods

2.1 Cardiovascular death data

We examined the monthly counts of cardiovascular and heart failure deaths in the eight Australian states and territory capital cities for the years 1997 to 2004 (8 years). The eight cities studied are shown in Table 1.

Because of a delay in registration of deaths we excluded the last month of the study period (December 2004). The monthly counts of deaths were
stratified by: month and year of death, sex, age (18–64 years, 65+ years) and the International Classification of Diseases (ICD-10) code of underlying cause of death.\textsuperscript{17} We classified the cause of death into seven groups using the subsections of the ICD classification.

### 2.2 Estimating seasonality

We estimated the seasonality in HF and CVD using a sinusoidal model.\textsuperscript{18} An example of a sinusoidal fit to the total CVD deaths is shown in Figure 2. The amplitude in total deaths is the difference from the mean to the peak value, and is 2700 deaths in this example (or 17\% of the mean). The peak in seasonal deaths in this example is 7.5 months (mid-July). Our main goal was to estimate if the amplitude or peak of the seasonal pattern varied by: sex, age, climate (i.e., city), ICD-10 code and time.

To correctly estimate the seasonality in HF and CVD adjustment for long-term trends was required. This was achieved by using a hierarchical Bayesian model with separate hierarchies for trend and season.\textsuperscript{19} The model was applied using the Bayesian WinBUGS software version 1.4.3.\textsuperscript{20} We used 18,000 Markov chain Monte Carlo iterations with a burn-in of 6,000 thinned by 3.

The dependent variable in the model was the monthly number of deaths. These counts were standardised to account for the uneven number of days between months.\textsuperscript{18} We used a generalised linear model assuming a Poisson distribution for the monthly counts.\textsuperscript{21} We adjusted for changes in the pop-
ulation over time by including annual city population as an offset (using population estimates from the Australian Bureau of Statistics).

The seasonal patterns in HF and CVD may vary by city, sex, age group, ICD code and time, and by interactions between these variables. As an example, previous studies have shown larger seasonal changes in warm compared to cold climates.\textsuperscript{1,7} In order to find the optimal models we examined a number of different models using combinations of these variables and their interactions. Each model was based on evidence from the literature concerning seasonal patterns in CVD. We selected the optimal models based on the deviance information criterion (DIC).\textsuperscript{22} This is an estimate of model fit that compromises between a close fit to the data and over-fitting from including unnecessary parameters.

We checked the residuals of the final model for any remaining seasonal pattern using the cumulative periodogram test.\textsuperscript{23} We performed this check for each city, age group and sex.

We looked for climatic patterns in seasonality by plotting the estimated amplitudes for each city against four indicators of climate: latitude, average January temperature, average July temperature, difference between January and July temperature. As the patterns appeared non-linear we fitted a generalised additive model to the estimates using a penalised spline with 3 degrees of freedom.\textsuperscript{24} The model weighted the estimates by the inverse of the standard error, which gave greater weight to those cities with a more accurate amplitudes.
3 Results

The amplitudes and peaks of the seasonal change in cardiovascular disease by ICD group are shown in Table 2. These results are averaged across cities, age groups and sex. The largest amplitude was for heart failure (23.5%) and this peak occurred in early August (8.0 months).

Although Table 2 shows a variation in the seasonal amplitude by ICD group, the amplitude in every group was statistically significant as the lower limit of the 95% posterior interval was always greater than zero. The range in mean amplitudes was from 8.7% to 23.5%, and such figures also represent a clinically significant increase in deaths.

The peak months were broadly similar across the seven ICD groups, and all occurred in July to August (Table 2). The earliest peak was for ischemic heart disease (7.5 months), and the latest for the electrical conduction system (8.2 months).

The differences in the seasonal pattern by age, sex and time are shown in Table 3. The table shows the baseline (intercept) results and the changes due to age, sex and time.

The winter increase in CVD deaths was 8.5% larger in the 65+ year age group compared to the 18–64 age group. A similar sized increase was observed in HF (7.7%) but with a much larger posterior interval. The seasonal increases in CVD and HF deaths did not change greatly by sex. For both CVD and HF there was a steady decrease in the seasonal amplitude over
time. The peak time for both CVD and HF was 7.7 months. For HF deaths this peak was slightly later in women.

There was a large variation in the seasonal amplitude of CVD and HF by city. In an attempt to explain this difference the mean CVD amplitude is plotted against average January and July temperature in Figure 3, and the mean HF amplitude in Figure 4. These results are averaged across age and sex.

The highest seasonal change in CVD mortality was in Perth (15.2%) and the lowest in Darwin (7.6%). The highest seasonal change in HF was in Sydney (40.5%) and the lowest in Hobart (15.4%). The seasonal amplitudes for total CVD showed a quadratic pattern when plotted against summer temperature (Figure 3). The amplitudes were largest in the temperature cities, and smaller in the warmer and colder cities. The quadratic pattern was less clear when the amplitudes were plotted against winter temperature. For heart failure there was also a quadratic pattern between season and climate, but the pattern was much noisier (Figure 4).

The residual checks for any remaining seasonal pattern were statistically significant for total CVD in older women in both Sydney and Brisbane, and for HF in older women in Sydney and young women in Hobart (4 statistically significant results out of 64 tests). Further investigation revealed that the sinusoidal model did not fully capture the sharp winter peak in deaths for the older age groups. In Hobart there were no HF deaths in young women.
4 Discussion

Australia has a large variation in climates across its capital cities (Table 1) but a similar health system. This makes it an ideal place to study seasonal patterns in cardiovascular disease and heart failure. Despite Australia’s predominantly mild climate we found a large seasonal change in CVD-related deaths. All categories of cardiovascular disease were significantly seasonal, but by far the most seasonal was heart failure (Table 2). The average winter increase in heart failure of 23.5% in Australia is comparable to other increases found in France, 20%;2 Madrid, 25%;3 and Scotland 12%.

Congestive heart failure (CHF) is the end stage of many cardiac disease process, with hypertension and ischaemic heart disease being the two largest causes of CHF. The major cause of conduction system abnormality in Australasia is ischaemic heart disease (IHD), and this may be seen as a subset of the larger primary IHD category. The main cause of pulmonary circulation is pulmonary hypertension, and whilst this has a number of specific disease processes associated with abnormally reactive vasculature and plexiform lesions, the major cause of pulmonary hypertension in the adult populations remains left ventricular failure.

As the end stage of these many conditions, the patient with CHF has little physiological reserve to deal with an increase in cardiac workload. With a temperature drop comes a compensatory vasoconstrictive response, particularly to the skin. This is associated with an increased after-load for the
failing heart, and is achieved by up-regulation of the neuro-humoral cascade, and increased levels of vasoconstrictors. Hence cardiac work increases to overcome the rise in after-load, and at the end-stage the failing heart is unable to cope with this increased demand. There may be associated increases in exacerbations in minor respiratory tract infections, which will compound workload for the failing heart.

Magnitude of temperature change may correlate with degree of change in systemic vascular resistance (via release of circulating catecholamines), and hence cardiac workload. A heart that is already sick would cope poorly with this change and may fail. Blood pressure changes may also directly correlate with catecholamine levels, and previous studies have shown that blood pressure changes are greatest with large temperature changes.\textsuperscript{11,13}

There is strong evidence to show that vitamin D insufficiency can precipitate heart failure.\textsuperscript{12} Australia has a very sunny climate, but vitamin D levels in adults have been shown to be very low in winter.\textsuperscript{25} People with pre-existing heart failure often have a sedentary lifestyle, which may result in few hours outdoors with minimal exposure to sunlight. For these people a vitamin D supplementation may reduce their event risk, although a specific trial of this intervention needs to be carried out before supplementation can be recommended.
4.1 Differences in the elderly and over time

The seasonal change in total CVD was strongly dependent on age (Table 3). In the elderly the seasonal increase was 8.5% higher. A similar, but non-statistically significant, increase in deaths was found for HF. A study in Scotland found that the winter increase in HF was 15–16% in those aged over 85, and 7–8% in those aged 65–74. The elderly are more frail and less well physiologically equipped to deal with the winter drop in temperature. Many may also have pre-existing cardiovascular disease, and so an increase in blood pressure (due to exposure to cold) may have more serious consequences. The elderly may also be more susceptible to infection.

During the study period (1997–2004) the seasonal change in CVD and HF deaths got gradually smaller. For all CVD deaths there was a decrease of 0.9% per year, and for HF deaths a decrease of 1.2% per year. This suggests that the seasonal pattern in deaths may disappear in time. Although in Sydney where the seasonal increase in HF deaths is 40.5% this would take roughly 34 years. The reduction in seasonal deaths over time may be due to: gradual improvements in housing and treatment, an increased uptake of flu vaccinations, or possibly even higher ambient temperatures associated with the process of global warming.
4.2 Seasonality and climate

We found large differences in the seasonal pattern of CVD dependent on climate. Darwin is in the tropics with distinct wet and dry seasons (May to September). It showed the smallest average seasonal change in CVD deaths. Hobart is the coldest city on average, and it had the next smallest seasonal change (Figure 3). The largest seasonal changes in CVD deaths were in the temperate cities (Figure 1). This pattern of increasing seasonal change in CVD with milder climates has been previously found in Europe\textsuperscript{7} and using worldwide data.\textsuperscript{1} The results shown here suggest that it is summer temperature, rather than winter temperature, that most strongly dictates the degree of seasonality in CVD in Australia. This could be because there is no perceived need to trap heat in warm Australian cities, and building design is geared towards cooling. For example, many homes in Brisbane are raised on stumps to increase ventilation.

The pattern between climate and HF (Figure 4) was not as clear as the pattern between climate and CVD. This was a surprise as the strongest candidates of the winter increase in HF are a lack of vitamin D,\textsuperscript{12} or increase in blood pressure due to lower skin temperature.\textsuperscript{13,26} It is possible that these factors are still important but that they are modified by local factors (e.g., housing type). Another possibility is that multiple factors are involved (Figure 1). Without detailed individual level data it is difficult to separate the many different seasonal exposures. Future studies would ideally collect information from new heart failure patients on recent physical activity, housing
type, and their blood pressure and vitamin D levels.

4.3 Preventing seasonal deaths

It seems likely that reducing exposure to cold temperatures could prevent many CVD and HF deaths in Australia. Strategies for reducing exposure to cold need not be costly. One option it to encourage people (particularly the elderly) to wear warmer clothes on relatively cold days. A more expensive alternative is to insulate homes. Only 58% of Australian homes had roof insulation in 2002.27

The financial burden of heart failure in Australia is high, and is estimated to cost over $1 billion each year and affect more than 500,000 Australians.28 The cost per admission with a primary diagnosis of CHF is approximately $10,500 (Australian) per admission (at our institution), and many patients require several admissions. Given this high financial cost, it would appear that further studies delineating methods of minimisation of temperature-related morbidity and mortality are called for. Such studies may reduce health care costs, as the age of the population and hence incidence of cardiac disease increases exponentially.
References


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ship to geography, climate, behaviour and cold-related mortality in Europe. *Int J Biometeorol* 2001, **45**:45–51.


20. Spiegelhalter D, Thomas A, Best N, Lunn D: WinBUGS Ver-


Figure 1: The putative pathways between winter and cardiovascular disease. Adapted from Scragg.\textsuperscript{10}

Notes:
Risk factors are shown in boxes with dotted lines; exposures are in boxes with solid lines.
Figure 2: Total cardiovascular disease deaths in Australian capital cities (1997–2003) and fitted seasonal sinusoid

Notes:
Note, the y-axis starts at 13,000 deaths

Figure 3: Cardiovascular disease seasonal amplitudes by city against January and July temperatures (degrees C). Smooth line and shaded area are the mean and 95% confidence interval from a non-linear model

Figure 4: Heart failure seasonal amplitudes by city against January and July temperatures (degrees C). Smooth line and shaded area are the mean and 95% confidence interval from a non-linear model
Table 1: Basic characteristics of the eight Australian state and territory capital cities ordered by population size

<table>
<thead>
<tr>
<th>Capital, State/Territory</th>
<th>Population(^a)</th>
<th>Total CVD deaths(^b)</th>
<th>Total HF deaths(^b)</th>
<th>Latitude (degrees south)</th>
<th>Average temperature(^c) (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sydney, NSW</td>
<td>3,007,162</td>
<td>74,306</td>
<td>4063</td>
<td>33.9</td>
<td>22.1, 11.8</td>
</tr>
<tr>
<td>Melbourne, Vic</td>
<td>2,570,364</td>
<td>57,878</td>
<td>3894</td>
<td>37.8</td>
<td>19.5, 9.0</td>
</tr>
<tr>
<td>Brisbane, Qld</td>
<td>1,171,367</td>
<td>28,639</td>
<td>1102</td>
<td>27.4</td>
<td>25.0, 14.8</td>
</tr>
<tr>
<td>Perth, WA</td>
<td>1,000,273</td>
<td>20,511</td>
<td>1075</td>
<td>32.0</td>
<td>24.1, 12.8</td>
</tr>
<tr>
<td>Adelaide, SA</td>
<td>817,375</td>
<td>24,472</td>
<td>1414</td>
<td>34.9</td>
<td>22.6, 11.1</td>
</tr>
<tr>
<td>Hobart, Tas</td>
<td>139,946</td>
<td>4224</td>
<td>213</td>
<td>42.9</td>
<td>16.7, 7.9</td>
</tr>
<tr>
<td>Canberra, ACT</td>
<td>222,274</td>
<td>3424</td>
<td>240</td>
<td>35.3</td>
<td>20.2, 5.3</td>
</tr>
<tr>
<td>Darwin, NT</td>
<td>62,260</td>
<td>631</td>
<td>27</td>
<td>12.5</td>
<td>28.5, 24.9</td>
</tr>
<tr>
<td>Total</td>
<td>8,991,021</td>
<td>214,085</td>
<td>12,028</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes:
(a) In 2000 (midpoint of years studied)
(b) January 1997 to November 2004

Table 2: Seasonality in cardiovascular disease deaths by ICD group ordered by amplitude in Australian capital cities, 1997–2004\(^a\)

<table>
<thead>
<tr>
<th>ICD group (ICD-10 codes)</th>
<th>Amplitude (%)</th>
<th>Peak (months(^b))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>95% PI</td>
</tr>
<tr>
<td>Heart failure (I50)</td>
<td>23.5</td>
<td>15.9, 29.9</td>
</tr>
<tr>
<td>Hypertensive diseases (I10–I13)</td>
<td>16.0</td>
<td>8.3, 22.8</td>
</tr>
<tr>
<td>Ischemic heart diseases (I20–I22, I24–I25)</td>
<td>12.3</td>
<td>6.1, 17.2</td>
</tr>
<tr>
<td>Electrical conduction system (I44–I49)</td>
<td>10.7</td>
<td>3.5, 17.1</td>
</tr>
<tr>
<td>Pulmonary &amp; pulmonary circulation (I26–I28)</td>
<td>10.7</td>
<td>2.3, 18.4</td>
</tr>
<tr>
<td>Cerebrovascular diseases (I60–I64, I67, I69)</td>
<td>9.2</td>
<td>3.0, 14.1</td>
</tr>
<tr>
<td>Myocardium/cardiomyopathy (I40, I42)</td>
<td>8.7</td>
<td>1.4, 15.8</td>
</tr>
</tbody>
</table>

Notes:
(a) December 2004 excluded due to registration delays
(b) 1=January, 12=December
ICD = International Classification of Diseases; PI = posterior interval
Table 3: Differences in the seasonal pattern of heart failure and cardiovascular disease deaths by age, sex and time. Intercept rows show the baseline effect, and other rows show the positive and negative changes.

<table>
<thead>
<tr>
<th></th>
<th>All CVD</th>
<th>HF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>95% PI</td>
</tr>
<tr>
<td><strong>Amplitude, percent</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept (Men aged 18–64)</td>
<td>7.1</td>
<td>1.2, 11.5</td>
</tr>
<tr>
<td>Age 65+</td>
<td>+8.5</td>
<td>+0.9, +14.8</td>
</tr>
<tr>
<td>Women</td>
<td>+1.1</td>
<td>-2.5, +5.1</td>
</tr>
<tr>
<td>Time (per year)</td>
<td>-0.9</td>
<td>-1.7, -0.2</td>
</tr>
<tr>
<td><strong>Peak time, months</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept (Men)</td>
<td>7.7</td>
<td>7.3, 8.1</td>
</tr>
<tr>
<td>Women</td>
<td>+0.1</td>
<td>-0.1, +0.2</td>
</tr>
</tbody>
</table>

CVD=cardiovascular disease; HF=heart failure; PI = posterior interval