Seasonal variation in mortality in Scotland

Islay Gemmell, Philip McLoone, FA Boddy, Gordon J Dickinson and GCM Watt

Background
Seasonal patterns in mortality have been recognized for many years. This study assesses seasonal variation in mortality in Scotland between 1981 and 1993 and considers its association with socioeconomic status and outdoor temperature.

Methods
Lagged Poisson regression analysis of numbers of deaths and average weekly temperature with adjustment for serial autocorrelation and influenza epidemics.

Results
There was significant seasonal variation in weekly death rates with a difference of about 30% between a summer trough and a winter peak. This variation was principally attributable to respiratory disease, cerebrovascular disease and coronary artery disease. Seasonal variation in mortality fell from around 38% in 1981–1983 to around 26% in 1991–1993. There was no clear evidence of a relationship between socioeconomic status and seasonal mortality, however the extent of the fall in seasonal variation was greater in deprived areas than in affluent areas. Overall, a 1°C decrease in mean temperature was associated with a 1% increase in deaths one week later. The lag in this relationship varied by cause of death and underlying temperature.

Conclusions
Seasonal variations in mortality and the relationship between temperature and mortality are a significant public health problem in Scotland. It is likely that the strength of this relationship is a result of the population being unable to protect themselves adequately from the effects of temperature rather than the effects of temperature itself.

Keywords
Seasonal, mortality, temperature, deprivation

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Weekly variation in mortality was examined for all causes of death and separately for diseases of the respiratory system (ICD-9 460–519), ischaemic heart disease (IHD, ICD-9 410–414), cerebrovascular disease (CVD, ICD-9 430–438) and all cancers (ICD-9 140–239). These groupings were preferred in order to reduce the effects of possible confounding between individual causes of death (for example, between influenza-like illnesses and other respiratory causes). Seasonal variation for deaths from cancers is small; these deaths were included to provide a comparison with groups of causes known to exhibit seasonal variation.

Commonly, seasonal patterns of mortality are described by comparing monthly numbers of deaths. This can be as the ratio of deaths in the first quarter of the year to the third quarter, a comparison with groups of causes known to exhibit seasonal variation.

In this analysis the seasonal pattern was estimated using Poisson regression analysis on weekly data. Firstly the trend was fitted using

$$y_t = a + bt$$

where $y_t$ is the number of deaths in week $t$, $t = 1, \ldots, 676$

this gave an expected number of deaths per week assuming no seasonal variation was present. Then a generalized linear model with a Poisson error and a log link of the form

$$\ln(y_t) = \alpha \cos(\omega t) + \beta \sin(\omega t),$$

where $\omega = \frac{2\pi}{t}$

was fitted using the expected values as an offset term.

The inclusion of the offset term meant that when $\lambda = \sqrt{\alpha^2 + \beta^2}$ was computed $\lambda$ represents the amplitude of the seasonal curve expressed as a ratio of the mean number of weekly deaths. This can then be expressed as the percentage increase in mortality from summer to winter.

The relationship between patterns of mortality and temperature was assessed using a time series regression model. Zeger described an approach that enables time series methodology to be applied within the framework of a generalized linear model. This method involves modelling the weekly number of deaths in a Poisson regression analysis and assessing the significance of short-term changes in weekly average temperature. The model controlled for long-term and seasonal trends in mortality, serial auto-correlation (AR(1) model), over-dispersion and influenza epidemics. The temperature terms in the model measure the effects of short-term changes in temperature over and above average annual variation. The parameter estimates obtained from the model were expressed as the percentage change in deaths following a fall in temperature of 1°C. This change in mortality was assessed for up to 5 weeks after the fall in temperature. The modelling was carried out using Splus.

### Results

The results of the sinusoidal analysis of weekly mortality are shown in Table 1. For all causes, the percentage summer to winter difference in weekly mortality rates is about 30% which, in Scotland for these years, translates into 350 extra deaths per week during the winter peak compared to the summer trough. The increase is principally attributable to respiratory disease (with summer to winter variation of 89% for males and 114% for females); IHD (with seasonal increases of 34% and 36%) and CVD (36% for both males and females). In contrast, seasonal variations in cancer deaths were only about 5%. As contributions to overall mortality, these changes need to be seen in the context of the pattern of Scottish deaths: respiratory deaths comprise 11% of the total and IHD deaths almost 30%

Although seasonal variation in respiratory deaths was greater than that for IHD, each cause of death contributes an extra 100 deaths per week in the winter peak compared to the summer trough. Seasonal variation in weekly death rates was related to the extremes of age and especially marked for respiratory deaths in children aged 0–9 years when the summer to winter difference was in excess of 200%. Even at ages 10–59, however, seasonal differences remain substantial with increases in IHD rates of 20% and 26% and an excess of CVD rates of 28% and 26% for

### Table 1: Seasonal percentage increase in mortality from summer to winter (with 95% CI)

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Age group</th>
<th>0–9</th>
<th>10–59</th>
<th>60–69</th>
<th>70–79</th>
<th>80+</th>
<th>All ages</th>
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<td>16</td>
<td>20</td>
<td>27</td>
<td>42</td>
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<td></td>
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<td>26</td>
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<td>36</td>
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<td>35</td>
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<td>685</td>
<td>28</td>
<td>37</td>
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<td>34</td>
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<td>84</td>
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<td>86</td>
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<td>104</td>
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<td>5</td>
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the two sexes at these ages. At ages over 60, respiratory disease death rates almost double in winter, and in those aged 10–59 increase by about 75%.

All these seasonal increases are significant as the 95% CI demonstrate. For deaths from all causes seasonal variation in mortality increased significantly with age. The CI for the very young age group are large mainly because of the small numbers involved.

The pattern of these changes is illustrated in Figure 1 which represents weekly deaths in circular form. In this Figure, the broken line represents the number expected if there was no seasonal variation and the solid line the average weekly number of actual deaths. The dark shaded area represents 25th and 75th percentiles and the light shaded area represents the maximum and minimum in a given week over this 13-year period. On average, the weekly number of deaths was greater than that expected from late October to mid-May with a pronounced steepening of this difference from November to January and a fairly slow return to the expected number during the later winter and early spring months. A second feature, however, is that there is much greater weekly variation (the shaded areas) in winter than in summer when the maximum and minimum range is much closer to the weekly average. The large peak in the weekly maximum for December reflects the 1989 influenza epidemic. Other year-to-year variation in weekly winter deaths is similarly attributable to influenza epidemics.

Figure 2 describes this pattern of weekly deaths throughout an ‘average’ year but divides the seasonal effect between three groupings of the Registrar General’s social classes. The social class analysis was restricted to male deaths because of uncertainties about the use of this classification for female deaths. For social classes I and II, weekly death rates at the peak of the winter cycle were 17% greater than those of the annual average but 24% greater for social classes IV and V with social class III deaths occupying an intermediate position. This difference in the ratio of weekly deaths to annual average almost reached statistical significance during the 4-week period covering late December/early January ($P = 0.055$). However, this was the only time in the year where clear social class differences were identified.

No similar pattern was observed when the data were grouped according to area-based Carstairs deprivation categories. However the decline in death rates between the 1981 and 1991 Censuses has been shown to be greater in affluent localities than in those categorized as deprived. A decline in seasonal variation in mortality was also observed between the two censuses. In 1981–1983, the seasonal increase in mortality was 35% for male deaths and 42% for female deaths: in 1991–1993, the seasonal variation was 23% for males and 29% for females although the CI of these proportions overlapped. In a further analysis comparing the change in seasonal patterns between 1981–1983 and 1991–1993 the change in the autumn/winter differential was greatest in the deprived category.

Figure 3 shows the percentage fall in death rates by season in the three main Carstairs categories between 1981–1983 and 1991–1993. The percentage fall in death rates was lower in the deprived category compared to the affluent category in every season except winter. The reduction in the winter rate in the deprived category was almost three times that for the autumn rate, whereas in the affluent category, the winter reduction was less than twice that for the autumn weeks. Thus, between the two censuses, the seasonal pattern in the deprived category reduced at a greater rate than in the affluent category although this differential in level of reduction failed to reach statistical significance.

The lagged effect of reductions in temperature across the year (modelled using Zeger’s method and including influenza
deaths) is summarized in Figure 4. A 1°C reduction in weekly average temperatures resulted in an increase in mortality of the order of 1% although with a lag that persisted for several weeks. For CVD, the associated increase in deaths was weakest, but with a significant increase in mortality that lasted for 3 weeks. The IHD deaths exhibited a more immediate response with a statistically significant increase of around 1% in the week of the fall in temperature (0.9%, 95% CI: 0.66, 1.13) and one week later (1.04%, 95% CI: 0.78, 1.29) but little relationship thereafter. The effect on deaths attributed to respiratory disease was less immediate and had a much longer lag period—a 1°C fall in temperature in week 0 was associated with significantly increased respiratory deaths 4 weeks later. A residual analysis showed that the residuals were almost free of auto-correlation and were normally distributed.

No difference was found in the direct effects of temperature on mortality when the data were analysed separately according to social class and area-based deprivation categories. Similarly there was no difference between the three cities, Glasgow, Edinburgh and Aberdeen when city-based mortality rates were associated with weekly temperature in each city.

Figure 5 describes these relationships at different underlying temperatures. In week 0, the week that the fall in temperature occurred, mortality increased by 1.06% (95% CI: 0.10, 2.03) when the underlying temperature was less than 2°C and by around 0.8% when the underlying temperature was between 2°C and 10°C. When the temperature was milder, between 10°C and 14°C the increase in mortality related to a fall in temperature was negligible. A significant fall in mortality however occurred when the temperature was hot, over 14°C. At this underlying temperature a fall of 1°C was associated with a 1% (95% CI: 0.32, 1.64) fall in mortality. Lag effects were observed for all these lower temperatures but with an enhanced short-term effect when the underlying baseline temperature was less than 2°C: at these temperatures, the effect of a further 1°C fall was a 1.15% (95% CI: 0.54, 1.77) increase in mortality in the succeeding week. These increases are offset by reduced mortality at these lower temperatures in week 2, the effect in later weeks is less clear. The pattern is one of a direct link at low temperatures up to one week after the fall in temperature, with a reverse effect at high temperatures during the week of the fall in temperature. A more complex pattern of association occurs at longer lag periods for temperatures when the influence of temperature per se may be mediated by other environmental variables.
Discussion

This study has shown that seasonal variation in mortality in Scotland is still a significant public health problem. The annual average seasonal increase in mortality between 1981 and 1993 was 29% for females and 34% for males. During the study period influenza epidemics occurred every 3–4 years. For each recorded death from influenza, there was an increase in deaths from all causes of 1.4%. However during an influenza epidemic the increase in all-cause mortality associated with the epidemic was considerably greater.

One hypothesis to explain these seasonal variations in mortality is that they simply represent shifts in the timing of events that lead to death. The effects of season or temperature could be seen as a kind of ‘harvesting’ of vulnerable members of the population. This view might be supported by the age gradients and causes of death, which demonstrate the greatest seasonal variation. However, it would require a detailed prospective study involving information about the prior health of those dying in different seasons to investigate this hypothesis further.

The fact that seasonal variation in mortality fell during the study period, that the degree of change was greater in deprived areas compared to affluent areas and that social class differences were only evident in the winter weeks would also lend support to this hypothesis. Although no significant differences were found these observations indicate that while socioeconomic status has some effect on seasonal variation the strength of this association has declined. Indeed an analysis of seasonal variation according to deprivation category failed to display a similar pattern to that for social class. Similar results were found recently for London.27

Improvements in environmental characteristics such as the quality of housing, increased use of central heating and other factors closely related to socioeconomic progress could explain this apparent fall in seasonal variation in mortality. The lack of substantial evidence of a relationship between socioeconomic status and seasonal mortality may be due to the fact that area-based measures of deprivation and individual-based social class data are not adequate proxy measures of housing conditions and fuel poverty.

The observed patterns of seasonal variation are ‘driven’ by temperature. On average a fall in temperature of 1°C was associated with an increase in mortality of 1%. Many studies28–30 have examined the methods by which falling temperatures can affect the physiological mechanisms of the human body. Seasonal variations have been demonstrated in several risk factors for vascular disease, high blood pressure,31 cholesterol levels32,33 and fibrinogen concentration in the blood.34–36 The U-shaped relationship between temperature and mortality, with very hot temperatures also having an adverse effect on health, has also been described elsewhere.18,20,21

Clearly the relationship between temperature and mortality influences seasonal patterns. However it is the vulnerability of a population and the ability of the population to protect themselves from this relationship which may determine the degree of seasonal variation in mortality experienced. Britain is generally considered to have a mild climate and, perhaps because of the perception of climate, has less strict building legislation than countries such as Finland.37 The fact that Scotland still experiences seasonal increases in mortality of the order of 30% while other European countries experience lower rates may be to do with the ‘susceptibility’ of the population to changes in temperature rather than the changes in temperature themselves. This study has also shown that while slight differences in seasonal patterns in mortality can be related to socioeconomic status the ‘susceptibility’ of the population is not identified by these measures. It would be difficult experimentally to isolate and modify specific aspects of lifestyle or environment as a way of demonstrating cause and effect. However specific housing characteristics such as fuel poverty,27,38 or the presence of dampness and condensation in homes may be related to the seasonal patterns in mortality experienced in the UK. This type of information is available from sample-based house condition surveys but relating sample-based explanatory variables to population-based mortality measures could lead to spurious results. A further study using housing measures available from the census such as tenure and amenities might provide more reliable insights into the relationship. Housing improvement schemes of various kinds are now fairly widespread in Scotland. With specifically collected data it is possible to envisage more focused ‘case-control’ studies of housing improvements in terms of the health outcomes they achieve.

Acknowledgement

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References


