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WINTER MORTALITY AND ITS CAUSES

W.R.Keatinge PhD FRCP

In the 1970s scientific research focussed for the first time on dramatic rises in mortality every winter, and on smaller rises in unusually hot weather. Following the recent decline in influenza epidemics, approximately half of excess winter deaths are due to coronary thrombosis. These peak about two days after the peak of a cold spell. Approximately half the remaining winter deaths are caused by respiratory disease, and these peak about 12 days after peak cold. The rapid coronary deaths are due mainly to haemoconcentration resulting from fluid shifts during cold exposure; some later coronary deaths are secondary to respiratory disease. Heat related deaths often result from haemoconcentration resulting from loss of salt and water in sweat.

With the possible exception of some tropical countries, global warming can be expected to reduce cold related deaths more than it increases the rarer heat related deaths, but statistics on populations in different climates suggest that, given time, people will adjust to global warming with little change in either mortality. Some measures may be needed to control insect borne diseases during global warming, but current indications are that cold will remain the main environmental cause of illness and death. Air pollution in cities may also still be causing some deaths, but these are hard to differentiate from the more numerous deaths due to associated cold weather, and clear identification of pollution deaths may need more extensive data than is currently available.

Key words: Cold. Heat. Mortality. Coronary thrombosis. Respiratory. Global warming.

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Important ill-effects of heat and cold on human health were recognised as early as the fifth century BC, in the first book written on the continent of Europe, but the nature of these problems has only become clear in the last 15 years. Daily mortality in Britain is lowest when outdoor temperature is around 18°C. As temperature falls from that level mortality rises progressively (1, 12, 6), to cause at present about 40,000 more deaths per year than would have occurred if mortality had remained at the minimum level. This gives a measure of cold related deaths. If temperature rises from 18°C, mortality again increases, to cause around 1,000 extra deaths per year. That gives a measure of heat related deaths.

Most governmental statistics for winter mortalities are based on a comparison of mortality in December-March (in the northern hemisphere) either with mortality in the rest of the year or in summer months, or with average mortality in the whole year. Methods of that sort have to be used if only monthly, rather than daily, mortalities are available, but they greatly underestimate the size of the problem. This is the case particularly in regions where there is substantial heat related mortality in summer. The effect of that mortality is then simply to reduce the apparent size of the cold related (winter) mortality. The daily mortality data now available for most developed countries is needed for any assessment of heat related mortality, as well as for accurate assessment of cold related mortality.

DEATHS FROM CORONARY THROMBOSIS AND RESPIRATORY DISEASES

A simple picture of the time relationships between short spells of cold weather and mortality can be built up by calculating repeated single regression coefficients, first of daily temperature and then of daily mortality, at successive days leads and lags on daily temperature (4). This shows that deaths from coronary thrombosis rise sharply in cold weather, and peak about two days after peak cold. Respiratory deaths rise more slowly, and only peak at about 12 days after peak cold.

An explanation for the rapid deaths from coronary thrombosis was provided by experiments in which volunteers were exposed to mild degrees of cold, just sufficient to cause slight shivering, and changes in their blood composition were measured (9, 16). Such cold exposure causes shut-down of most of the blood supply to the skin.

This reduces transfer of body heat to the skin and so reduces body heat loss, but it shifts a litre or more of blood from the skin. That blood overloads the central organs of the body with blood, and the excess volume is then disposed of by removal of salt and water from the blood, partly by the kidneys as urine, and partly into the general intercellular space of the body.

This leaves most of the other components of the blood more concentrated. Red cells, white cells, platelets, cholesterol, and fibrinogen all increase in concentration by around 10%, and the blood viscosity increases by around 20%. All of these make the blood more prone to clot. Blood also contains an anticoagulant, protein C. If that increased by a similar amount it would counter much of the increased clotting tendency, but protein C is a small molecule which can move through the walls of the capillaries and redistribute into the general extracellular space of the body. The result is that while the many factors that promote clotting increase in concentration during cold exposure, the protective protein C does not. This causes little trouble to young adults, but it greatly increases the likelihood of a thrombus forming in the arteries of elderly people, which are frequently roughened by atheroma.

The fact that respiratory diseases increase in winter has been known for centuries. The cause of this is still not fully known, but at least two factors are almost certainly involved. One is that people tend to congregate together in confined and poorly ventilated spaces in cold weather, increasing the opportunities for cross infection. The other is that cooling of the mucosa of the upper respiratory tract reduces its ability to counter infection. Early evidence had suggested that cold exposure at the time that common cold virus is introduced into the nose does not increase the likelihood of infection, but it was found that inhaling warm moist air at a later stage, when the infection was starting to cause symptoms, greatly alleviated the entire subsequent course of the illness (17,21). Respiratory infections also increase fibrinogen levels in the blood, and through this cause some delayed deaths from arterial thrombosis after spells of cold weather (23). There is also evidence that one respiratory pathogen, *Chlamidia pneumoniae*, may directly invade atheromatous plaques in arteries and lead to their breakdown (see 19). It may also promote thrombosis by increasing plasma triglycerides and cholesterol (14).

Some of the heat related deaths are also due to arterial thrombosis. This is due to loss of salt and water in sweat, which directly produces increased concentration of other components of the blood (10). Like exposure to cold, exposure to heat for long enough to cause sweating produces increases in the concentration of red cells, white cells, and platelets, and in the viscosity of the blood, all of which make the blood more liable to clot. Again, this does little harm to healthy young adults, but increases the risk of a coronary or cerebral thrombosis in elderly people with atheromatous arteries.

In developed countries, very few of the cold related deaths are due to hypothermia, simple cooling of the body (22), and deaths due to hyperthermia, simple overheating of the body, are also rare. Less information is available from developing countries, and it is possible that simple hyperthermia is responsible for substantial numbers of deaths in some of those in the tropics.

In recent years, interest in heat and cold related deaths has focussed increasingly on how they will change as a result of the global warming that is already under way in many parts of the world. Temperatures are predicted to increase by around 2°C over the next 50 years in Britain (6). A simple assessment of the effect of such a rise in temperature on mortality could be made if it is assumed that mortality at particular daily temperatures remains the same as it is now. If that happened, the 2°C rise in temperature would decrease cold related mortality in Britain by around 20,000 and increase heat related mortality by around 2,000.

GEOGRAPHICAL DIFFERENCES IN WINTER MORTALITY

In practice, populations can be expected to adjust to the warmer climate with relatively small changes in either of these mortalities. The Eurowinter study (12), with active surveys of house temperatures, clothing, and outdoor cold exposures, showed that people in cold regions such as the north of Finland protected themselves so well from cold that they experienced no more winter mortality than people of London or Athens did during much milder winters. People in north Finland also had almost as much heat related mortality in summer as people in Athens, where the summers were much hotter (13). In that case, the main reason was that heat related mortality started at much higher temperatures in Athens than in Finland. Similar differences in mortality/temperature relationships have also been reported between hot and cold regions of the United States (2).

Populations are therefore likely to adjust successfully to climate change, but it is uncertain how rapidly this will take place. Physical changes such as altered design of houses and installation of air conditioning systems cannot be expected to happen very rapidly, and there is clearly scope for governmental action to promote these as global warming proceeds. The general conclusions are that with the possible exception of some tropical countries global warming can be expected to bring net health benefits to most regions, that cold will remain a much larger cause of illness and mortality than heat, and that governmental action is needed to promote protection against summer heat and maintain protection against winter cold. There is also a need to promote assembly and publication of daily mortality figures in underdeveloped countries to allow assessment of heat related mortalities there.

HOW TO MINIMIZE THE RISK OF COLD WEATHER

Since cold is likely to remain the most important environmental cause of mortality in most parts of the world, the main priority is to reduce it. There has been uncertainty whether indoor or outdoor exposure to cold is the principal cause of this mortality. It has for example been suggested that the cold shock on going outside from a warm house on a cold day could be the main problem. If so, warmer housing could even have been harmful. Analysis of the Eurowinter data in fact showed that both warm housing and warm outdoor clothing had independent protective action against winter mortality.

People in the coldest regions of the world go to great trouble to keep warm both indoors and outdoors, and have little increase in mortality as temperature falls. The people of Yakutsk in eastern Siberia, the coldest city in the world, wore massive fur clothing with many layers beneath it, and showed no increase in all-cause mortality as temperature fell to -48°C (3). There are indications that outdoor exposure to cold is now the main cause of Britain's high winter mortality. Residents of Anchor Housing, which was fully heated, but who spent much time outdoors, had as large an increase in mortality in winter as the rest of the elderly population (8). The lesson is that campaigns to reduce winter mortality need to improve protection against outdoor as well as indoor cold.

Cold related mortality is falling steadily in Britain. This is probably due to a variety of factors, some based on advice from research,

and others related to rising prosperity. Warmer houses, greater availability of private cars, and increased tendency to be warmly dressed while waiting outdoors for public transport, have probably all contributed.

THE ROLE OF FLU

Another major factor has been the decline in influenza epidemics over the last few decades. Up to 1976, severe epidemics of influenza caused massive mortality in winter every two years or so, mainly among elderly people. The reason for the recent dramatic decline in these epidemics probably includes more settled conditions in the world, which give less opportunity for human and animal strains of influenza A virus to interact and create lethal new strains. The number of deaths from influenza has traditionally been assessed by comparing mortality in an influenza outbreak with that shortly before, or in previous non-epidemic years. This can give an exaggerated picture, as it takes no account of the fact that influenza outbreaks tend to occur in particularly cold weather, which increase mortality even in the absence of an epidemic (5). If allowance is made for the cold weather by multiple regression analysis, influenza epidemics account for less than 5% of the excess winter mortality in Britain over the last ten years. It must not be forgotten, though, that a new strain of influenza could once again cause devastating mortality, and immunisation programmes that can focus rapidly on new strains remain important.

THE ROLE OF AIR POLLUTION

Much attention has been given to air pollution as a cause of mortality. Specific pollutants such as asbestos or silica are well established causes of long term damage to the lungs. Recent interest has focussed on suggestions that common city pollutants might still be causing short term increases in mortality in many cities of the world, and time series analyses have been used to look for such short term relationships. The massive levels of pollution during the London smogs of the 1950s caused clear increases in short term mortality, but there is general agreement that any such mortality due to combustion products in cities is now small compared to mortality due to cold weather.

Recent studies have generally suggested some increase in mortality on, or up to around three days after, an episode of air pollution by

SO₂, CO, or particulates (eg 7, 18), but the results have been extremely varied in detail. Such studies generally attempted to allow for the effects of cold weather associated with pollution by using daily temperature on, or a few days before, the polluted day, as a confounding variable. This would allow for an average pattern of cold weather centred on that day, but the patterns of cold weather associated with pollution are often not typical and not centred on that day. To allow for such weather patterns effectively it is necessary to use temperature and other weather factors as confounding variables at multiple simultaneous lags, but this requires long runs of continuous data. These runs are available for SO₂ and CO in London, and when analysed showed no clear effect of these pollutants, at their current levels, on mortality (11). In the case of SO₂ this is to be expected, as SO₂ is almost entirely absorbed in the nose and throat and does not reach the lungs in measurable amounts (20).

Airborne PM10 particles do reach the lungs, and so are a plausible cause of short term mortality, but data runs for these are not yet long enough for decisive multiple delay analysis of the relationship. These particles are also a plausible cause of long term damage to the lungs (15), and possibly of cancer, but again there are many confounding factors that are hard to allow for in epidemiological studies. People with long term exposures to PM10 have usually also been exposed to a wide range of other environmental and lifestyle factors that are extremely difficult to estimate and to adjust for.

All forms of air pollution are unpleasant and have adverse effects on the quality of life. SO₂ for example irritates the nose, can trigger asthma, erodes buildings, and kills trees through acid rain. This alone is sufficient reason to control air pollution, even without decisive evidence that current levels of this are causing significant mortality, but prolonged runs of daily values for each pollutant could allow reliable estimates of any mortality that is caused by specific pollutants. Assembling such databases remains important.

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