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Health and the ozone layer

Skin cancers may increase dramatically

It has been recognised for over a decade that chlorine from chlorofluorocarbons may deplete the stratospheric ozone layer. This matters because this layer absorbs all ultraviolet C and a proportion of ultraviolet B from sunlight before it reaches the earth's surface. More recently scientists have observed an appreciable hole in the springtime ozone layer over Antarctica.[1] How is ozone produced and lost, and what might be the consequences for health of the depleted ozone layer?

Ultraviolet radiation with a wavelength of under 240 nm splits an oxygen molecule into two atoms, one of which combines with a molecule of oxygen to form ozone (O_3).[2] Ozone molecules are found in the stratosphere in a concentration of one in one million. This ozone mantle absorbs solar ultraviolet of a longer wavelength and is warmed; the ozone is eventually naturally lost by reacting with an oxygen atom to reform two oxygen molecules. The total amount of ozone is measured in units of milliatmosphere centimetres (or Dobson units), which are hundredths of a millimetre. The amount of ozone absorbing solar ultraviolet is normally about 300 Dobson units; if all the stratospheric ozone were brought to sea level the thickness of the ozone layer would be 3 mm.

Chlorofluorocarbons are man made compounds found as propellants in aerosol spray cans and as refrigerants in refrigerators, freezers, and air conditioners. The loss of ozone is accelerated by chlorine released by the breakdown of these chlorofluorocarbons by solar ultraviolet radiation: one chlorine atom may destroy 10 000 ozone molecules.[3] Chlorofluorocarbons have a half life in the stratosphere of 75 years or more. Man has caused a 10% reduction in the ozone layer over Antarctica in a decade, and the fact that chlorofluorocarbons already present will react with ozone molecules for the next 50-100 years is clearly a serious worry. The dynamics of the stratosphere could change, the radiative balance of the atmosphere could be affected, and the climate could be altered.

The ozone hole occurs over Antarctica because of a complex relation between a large, extremely cold land mass and the formation of an isolated cold stratospheric air mass in winter; the air inside the polar vortex spins rapidly eastwards and is rich in small ice particles.[4,5] The concentration of chlorine monoxide inside this "cold cauldron" is now known to be 100 times greater than that outside, accounting for the fact that the ozone layer over Antarctica was reduced to the equivalent of only 1·3 mm in October 1987. That this observation was made in the Antarctic springtime, when the sun's rays pass through the atmosphere at a lower angle, makes these observations a little less alarming than if they were made at midsummer. Although a reduction of the ozone over the northern hemisphere has not yet been unambiguously shown, there are some indications that there may be transient ozone holes over the Arctic.[6]

A 1% reduction in the ozone shield has been calculated to increase by 2% the amount of ultraviolet B in the 290-330 nm part of the spectrum reaching the earth's surface. Thus a 10% reduction in ozone over a decade could result in a 20% increase in ultraviolet B reaching the surface of Antarctica.

Since 1974 scientists at eight centres in the United States have used Robinson-Berger meters to measure ultraviolet B. They have weighted two meters for the wavelength of 297 nm, which causes maximum skin erythema on white skin.[7] Altitudes at which measurements were taken ranged from sea level to 1619 m. Between 1974 and 1985 an unexpected reduction in ultraviolet B by 11%, significant at the 99% level, was seen in El Paso, Texas; smaller reductions significant at the 95% level were evident in Florida, New Mexico, California, and Minnesota, and there was no change in Fort Worth, Texas, Philadelphia, and North Dakota. Climatic and other environmental factors may affect the absorption of solar ultraviolet radiation.

A further variable is the 11 year solar cycle and associated sunspots. At solar maximum, most recently around 1980, the sun emits more ultraviolet; in theory, more will reach the earth's surface. Whereas current instruments detect ultraviolet B, ultraviolet C may now be penetrating through a depleted ozone layer with grave biological consequences. Evidence of this must be sought.

The clinical condition most closely linked to increased exposure to ultraviolet B is non-melanoma skin cancer.[8] Work from areas (such as Queensland) of long daily exposure of white skin to intense natural sunlight shows that these cancers are already an extremely common problem.[9] The rising figures for incidences of these cancers are unlikely, however, to have been caused by depletion of ozone; a more likely cause is changing habits of sun bathing. The depletion of ozone does, however, make it likely that in future excessive exposure to natural sunlight will be even more hazardous: the hypothesis that the risk of non-melanoma skin cancer rises steeply once a critical number of hours of exposure to ultraviolet B is reached combined with the fact that a higher total dose will be accumulated for the same number of hours out of doors may mean that the incidence of non-melanoma skin cancer will jump dramatically.

The relation between intensity of ultraviolet radiation and cutaneous malignancy is complex. Current models suggest, however, that ozone depletion may cause a more rapid increase in melanoma than in non-melanoma skin cancer.[10] This is because short episodes of intense and burning ultraviolet exposure are linked to melanoma, whereas non-melanoma skin cancer is associated with the lifetime build up of exposure to ultraviolet.

To quantify changes in incidence and risk of skin cancer caused by the depletion of ozone is clearly important, but changes in holiday and clothing habits may make it impossible to attribute a percentage of the increased cancers to this depletion.

What action is required? Firstly, more nations should set up monitoring equipment at various locations to record ultraviolet changes through the entire ultraviolet A, ultraviolet B, and ultraviolet C portions of the spectrum. Secondly, those who have recently spent a long time in Antarctica should be observed for cutaneous malignancies. The outdoor protection needed in the Antarctic may well, however, have effectively protected people against ultraviolet.[11] Resident fauna such as the Emperor penguins may show ocular damage induced by ultraviolet and might merit a field study. Plankton in the surface waters around Antarctica may for the first time in their evolutionary history experience ultraviolet B or ultraviolet C. Lastly, the Montreal convention, signed last September, should be strengthened.[12] The convention aims at reducing the production of chlorofluorocarbons by half by the end of the 1990s. This is too little and too late. A drastic reduction of chlorofluorocarbon production is needed as soon as possible to prevent an environmental problem becoming an environmental catastrophe.

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Prophylactic sclerotherapy for varices

Useful only in limited circumstances

Sclerotherapy stops variceal bleeding,[1] reduces rebleeding,[2,3] and improves survival.[4] Yet about a third of patients still die from an episode of variceal bleeding.[2,5,6] Is there a case for prophylactic sclerotherapy? The answer would undoubtedly be yes if three conditions were fulfilled: the procedure was innocuous; all patients with varices were eventually to bleed; and the risk of death during a bleed was substantial and the same in all groups. Unfortunately none of these conditions is fully met.

Sclerotherapy is not innocuous. Non-fatal complications include stricture formation and dysphagia in 10-30% of patients.[5,7,8] Moreover, 1-7% of patients die of bleeding from ulceration after sclerotherapy, oesophageal perforation, and mediastinitis.[7,9-11] Paralysis of the spinal cord has also been recorded.[12]

Not all patients with varices do bleed. Only 10-20% of the controls in large trials of prophylactic shunt operations bled each year during the first two years, and only a fifth died.[13-15] In such a population 1800 patients would be needed to prove that prophylactic sclerotherapy reduced mortality by 25%.[16]

Until such a trial is available is it possible to select subgroups who are more likely to bleed or in whom a bleed is more or less likely to be fatal? Admission mortality is strongly dependent on functional hepatic reserve as measured, for instance, by a modified Child's grade.[17] Only about 6% of those with the most reserve will die after the first bleed. Similarly, only one out of 159 children and young adults with extrahepatic block died after the first bleed.[18,19] Thus the potential benefit of prophylactic sclerotherapy is low among those with the most hepatic reserve.

Those with less reserve, however, have a high risk of death during their first bleed. There might be potential for improving survival among them by prophylactic sclerotherapy, especially if there are other indications that they are likely to bleed soon. Such increased risk is shown by large variceal size,[20,21] prolonged prothrombin time,[22] the presence of red spots or weals over the varices,[21-23] and continuing abuse of alcohol in those with alcoholic cirrhoses.[24]

Paquet included endoscopic signs of impending variceal bleeding to select a group at high risk for a controlled trial of prophylactic sclerotherapy.[22] Mortality was significantly reduced from 14 out of 33 (42%) in controls to 2 out of 32 (6%) in treated patients at two years. Bleeding episodes were also reduced from 22 out of 33 (66%) to 2 out of 32 (6%). In a less selected series Witzel *et al* noted a reduction in mortality from 29 out of 53 (55%) in controls to 12 out of 56 (21%) over 25 months in the group given sclerotherapy.[25] Bleeding occurred in 30 out of 53 (57%) in the controls and five out of 56 (9%) in the treated group. These trials have been criticised not least because the control groups had unusually high mortality and rates of bleeding, at least compared with controls from historical trials of prophylactic portacaval shunts and other studies.[26] The contrary view is that the very purpose of having a control group in a study is to avoid reliance on comparison with patients from other studies, continents, and decades.

Three large randomised trials have been reported recently, and are not optimistic about the benefits of prophylactic sclerotherapy.[27-29] Sauerbruch *et al* performed prophylactic sclerotherapy in 41 out of 103 patients with cirrhosis and large varices.[27] During an average follow up of 17 months mortality was decreased in the group given sclerotherapy (20%) compared with the control group (35%). Yet a similar proportion of each group had variceal bleeding (29% against 35%). Bleeding occurred earlier in the group given sclerotherapy (mean 4·1 months) than in the control group (mean 6·7 months) and may have been precipitated by sclerotherapy.