HEALTH IN THE HOME

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CONTENTS

THERMAL COMFORT AND HYPOTHERMIA

K.J. Collins

(Medical Research Council Environmental Physiology Unit,
London School of Hygiene & Tropical Medicine)

INTRODUCTION

Hypothermia, defined as a state in which deep body temperature falls below 35°C (95°F), became recognised during the 1950's and 60's as an urban problem particularly affecting the very young and the very old. The condition can occur in any age group if, for any reason, the body's temperature-regulating mechanism is impaired in a cold environment or if the cold stress is overwhelming. The physiological disturbance is not particularly serious when hypothermia is of mild degree, say down to body temperatures of 32 to 33°C when the reactions against cold, vasoconstriction in the skin and shivering, can still be evoked. At lower body temperatures, however, consciousness is progressively lost, tissue metabolism depressed, and urgent treatment is required in order to restore body functions by raising the deep body temperature.

In infants, hypothermia is no longer the problem it was 20 years ago mainly because of a better public awareness of the risks of exposure to cold during the first few weeks of life and also to the trend for babies to be born in hospital. Hypothermia may also occur in young adults who, by accident or design, have taken an overdose of drugs such as sedatives, tranquillizing agents or anti-depressants. One of the effects of excessive quantities of these drugs, made especially potent when combined with alcohol, is to depress the normal nervous mechanisms of thermoregulatory control and artificially to induce a state of hypothermia.

In the old-age group, hypothermia is regarded as an important social problem in Britain for it is associated with a vulnerable sector of the population, with elderly people living alone in poorly insulated or inadequately heated dwellings and adverse socio-economic circumstances. Chronic hypothermia in the elderly may develop during long periods of cold winter weather and again it is the body's failing thermoregulatory capacity which precipitates the condition. Most cases of profound hypothermia are transferred to hospital where, even with careful management, mortality is unacceptably high. In the old-age group it should be said that the condition is often associated with serious concurrent illness and should then be considered as secondary hypothermia. But there remains a significantly large number of old people, the old and cold, who are on the borderline of hypothermia in cold conditions and whose pre-hypothermic state puts them at risk. The magnitude of this problem is difficult to forecast accurately but the combination of greater heating costs, increasing numbers of old people in the population and the promise by climatologists of longer and more severe winters, highlights the importance of maintaining adequate preventive measures.

POPULATION STUDIES OF URBAN HYPOTHERMIA

Although a number of temperature surveys have been conducted in Great Britain since 1960 results have often been based on mouth temperatures which can be misleading and inadequate sampling techniques. Surveys unfortunately have usually not coincided with very cold winter

conditions such as were experienced in the winter of 1962-63.

The Nuffield Foundation supported the first large-scale domiciliary survey of environmental and deep body temperatures of old people during the first three months of 1972 (Fox et al., 1973). The sample included 1000 people over the age of 65 in a National Survey and a similar number of elderly living in Camden, London. The winter temperatures in 1972 were slightly higher than average, but 75% of the sample had living-room temperatures in the morning below 18.3°C, the minimum recommended by the Parker Morris Report on Council Housing (1977), and in 10% the room temperatures were very cold, at or below 12°C.

One finding of the National Survey was that 0.5% of the elderly sample were hypothermic with a urine temperature of less than 35°C but within the range 34.2 to 34.9°C. If translated into terms of the total elderly population in Great Britain (i.e., those over 65 yr but not necessarily living alone) this suggests that hypothermia might occur in 35,000 elderly people even in a mild winter. It does not mean, however, as is sometimes expressed in the press and media, that this number of elderly people are dying of hypothermia. The National and Camden surveys in fact showed that urine temperatures in the hypothermic elderly did not remain below 35°C when body temperatures were taken in the afternoon or evening. The risk is clearly greater at night time when elderly people get out of bed and fall and become immobilized in cold bedrooms, and in the early morning when body temperature tends to be low.

In addition to those in the hypothermic range, a further 10% of the elderly sample were in a low-temperature group with urine temperature less than 35.5°C. These were thought to have signs of thermoregulatory failure as shown by the inability to maintain an adequate core-skin temperature gradient. In this group who might be considered at risk in cold environments, low body temperatures were significantly correlated with advancing age and the receipt of supplementary benefits. The absence of a relationship between room temperature and age seems to exclude one hypothesis for the decline of deep body temperature with age, namely that it is simply due to the older people living in colder accommodation.

CHANGES IN THERMOREGULATION WITH AGE

Research into the biology of ageing has shown that there is a decline in the efficiency of thermoregulation in old age. There is obviously a wide range of individual variability in susceptibility to cold, but a proportion of elderly people show impairment of the ability to control body temperature in cold conditions and clearly it is important to be able to identify this group. For example, elderly people appear to shiver less readily and some have poor control of their skin circulation. From the standpoint of the general physician certain key factors serve to alert attention to those at risk, e.g. (1) low temperatures in the living room and bedroom and lack of potential heating sources, (2) poverty combined with social isolation and (3) deterioration in total body functions. A single definitive test of thermal lability is not appropriate at present but a number of basic physiological observations point to the condition, e.g., generally low body temperature (urine or rectal temperature), an unusually small temperature gradient difference between deep body and skin

temperature, poor circulatory responses particularly the presence of postural hypotension (Collins et al., 1977), and a blunted sense of thermal perception compared to normal. There is evidence that the elderly are more insensitive to warm and cold stimulation at the skin surface and tests of digital temperature perception performed under controlled conditions appear to show that discrimination deteriorates markedly in people over 60 years of age (Figure 1). A decrease in blood supply to the skin tissues and in the density of functional nerve cells may both contribute to these changes in peripheral perception in old age.

THERMAL COMFORT AND HYPOTHERMIA

What has thermal comfort in the elderly to do with the prevention of hypothermia? The answer to this might be threefold. Firstly, it has been said that urban hypothermia would virtually be eliminated if all old people lived in comfortably warm surroundings. This, in fact, is not quite true because there have been recorded cases of episodic hypothermia in people living in adequately heated houses. Secondly, thermal discomfort may be regarded as a warning signal which operates to condition our behaviour. We might even say that thermal discomfort, e.g. feeling too cold, has a certain survival value in that it stimulates us to increase activity, increase our use of insulative clothing and switch on heating appliances in very cold conditions. The indications are that some elderly people lack both the ability to sense the need for more warmth and the capability of obtaining more warmth if they feel it is required. Thirdly, it is perhaps useful to remember that physical and intellectual performance is generally regarded as being at its peak when a person is in thermal comfort and that therefore the behavioural mechanisms of thermoregulation operate at their optimum.

The expectation is that in old age, people will require higher ambient living temperatures than young adults (a) because of a generally lower metabolic heat production and (b) because of the relative inactivity compared with the young. However, in laboratory experiments in the United States and Denmark (Fanger, 1972), the neutral ambient temperature for thermal comfort was found to be the same for both elderly and young adults. To be more specific, during tests lasting 3 hours the average preferred temperature in resting elderly and young adults wearing light-weight cotton clothing of 0.6 clo., was 25.6°C. In winter conditions in Great Britain, however, living and bedroom temperatures particularly during the morning are generally much lower than this and more clothing is worn indoors. Investigations made in controlled environments over the range of temperature expected in homes in Britain confirm that most healthy elderly people have the same thermal comfort preference as young adults, and that for a clothing insulation value of 1.0 clo the comfortable temperature during rest was 21.1°C (Collins, 1979, Figure 2).

Another approach used to assess preferred temperatures, employs the technique of remote control of room temperature in which each subject can select his own preferred ambient temperature (McIntyre, 1975). Although the preferred temperature of young and elderly subjects with a clothing value of 0.8 clo was again found on average to be the same in both groups, i.e. 22.4°C, elderly people on the whole demonstrated a much less precise control of the environment and allowed much larger swings in temperature to occur (Figure 3). About

20% of the elderly subjects studied in these experiments have displayed a poor peripheral temperature discrimination response (i.e. their threshold of temperature discrimination is 3°C or greater). It is of considerable interest that this particular group also tend to report that they are more comfortable in cold environments than other elderly subjects (Collins & Exton-Smith, 1980).

SOCIAL CARE AND PREVENTION OF HYPOTHERMIA

Public attention inevitably focusses on the more dramatic aspects of hypothermia especially in elderly people. It is important, however, not to lose sight of the much larger general problem of the cold elderly living in uncomfortably cold surroundings. There is often an association between low deep body temperature in old people and adverse social circumstances, the 'elderly poor' being recognized as those over 75 years of age, living alone and eligible for supplementary benefits (Wicks, 1978).

The principles of prevention of hypothermia are now generally recognized (Maclean & Emslie-Smith, 1977). Of primary importance is early detection by regular surveillance; the diagnosis of hypothermia is much more readily made now that low-reading clinical thermometers (the lowest reading being 25°C rather than 35°C on the conventional thermometer) are in more general use.

A proportion of elderly people are thought to enter a state of 'voluntary hypothermia' in an effort to conserve resources by reducing expenditure on increasingly expensive fuel. Many, unfortunately, do not take full advantage of existing benefits. The 1972 National Survey suggested that 3 out of 4 elderly people were unaware of the extent of supplementary benefits and only 11% were actually receiving extra heating allowances. Recent estimates indicate that now more than 60% of supplementary pensioners are receiving extra heating allowances. It should not be forgotten, however, that 77% of those over pensionable age are not entitled to supplementary benefits or help with heating, though many may be only marginally more affluent than supplementary pensioners. Difficulties experienced by old people in meeting heating costs have been minimized in some areas of the country by the introduction of flat-rate payment schemes. Regional variations are also apparent in efforts to supply heating appliances and materials for improving insulation in the homes of the elderly.

Adequate clothing is obviously an important defence against cold and it provides a simple and relatively cheap method of control of the individual micro-climate for most mobile elderly people. The physics of clothing, however, militates against it as a means of efficiently preventing hypothermia in old people. Clothing in cold conditions becomes efficient in controlling heat loss when metabolic heat production is high and in the hypothermic elderly patient internal heat production is low so that the thickness of clothing has to be increased out of all proportion to provide efficient insulation. Furthermore, there are instances of hypothermia in elderly persons found in bed apparently well-covered with bedclothes and with good external insulation. In the daytime, excessive amounts of clothing may also restrict mobility and encourage a static state. 'Space suit' housecoats designed to combat hypothermia are commercially available but are expensive, while restriction of heat loss from the head which may be as

much as 20% in the clothed individual gives reason as well as fashion to the habit in earlier days of wearing a smoking cap or nightcap. During the night too, the use of a low-voltage electric over-blanket is a most economic insurance against hypothermia.

In conclusion, the important requirement is to ensure that homes for elderly people conform to standards of insulation and heating capable of achieving adequate warmth for thermal comfort. As a guideline this may be 21°C for the sedentary person wearing 1.0 clo of insulation, but it should be recognized that more active elderly people may feel uncomfortably warm at this temperature and will prefer lower ambient temperatures.

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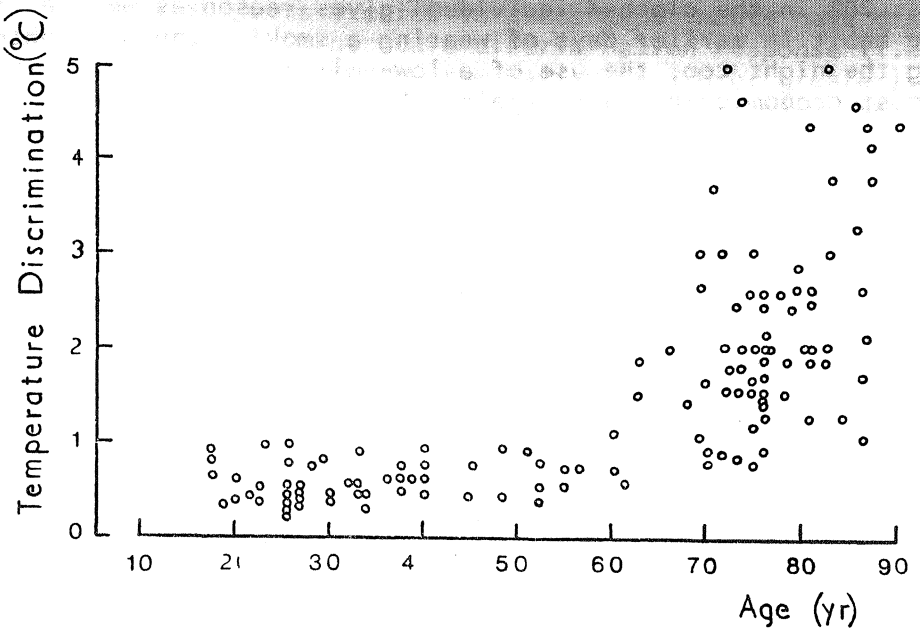

Figure 1 Peripheral temperature discrimination (°C) in relation to age.

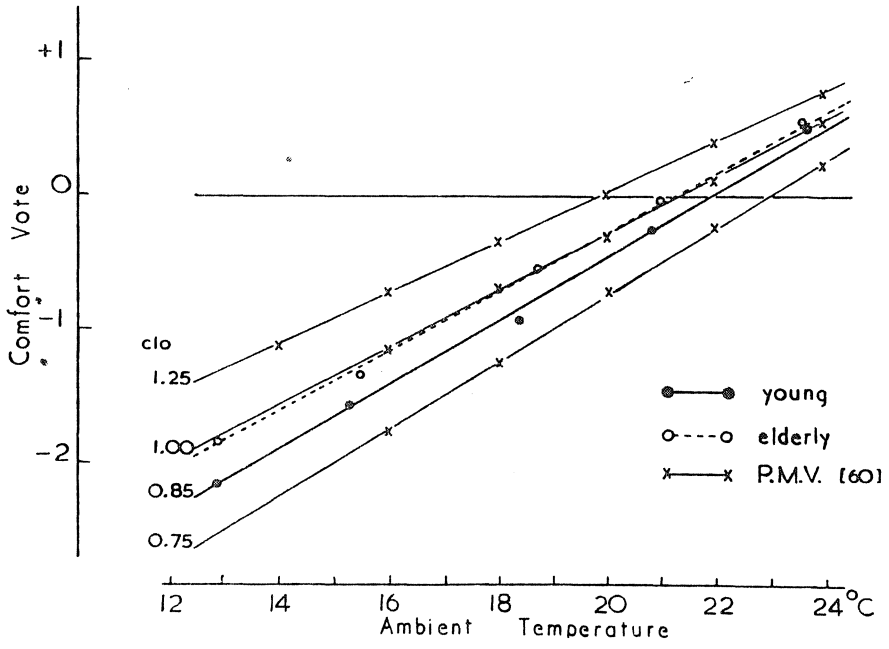

Figure 2 Subjective thermal comfort votes (+1 comfortably warm, 0 comfortable, -1 comfortably cool, -2 too cool) in young adult and elderly subjects at rest. Regression lines correspond to different clothing ensembles (0.75 to 1.25 clo). PMV (60) represents the Predicted Mean Vote for young adults at rest (from Fanger 1972).

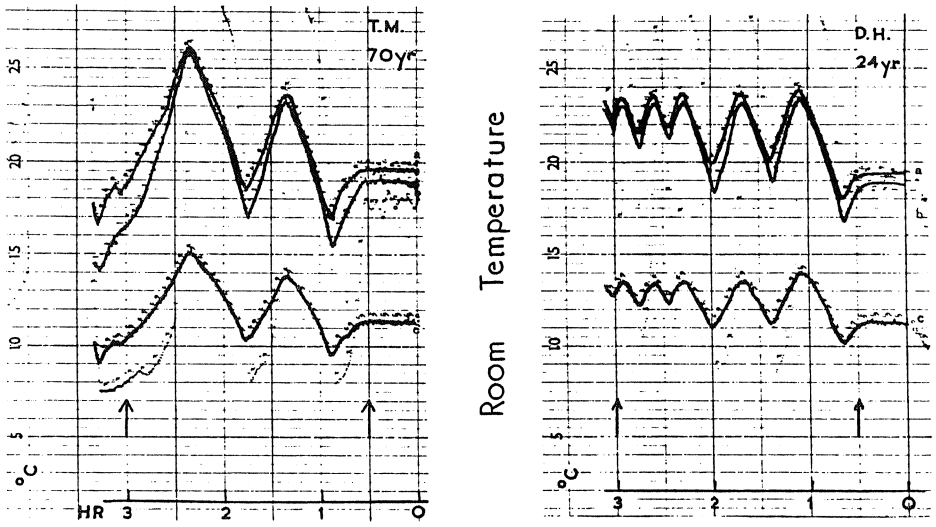

Figure 3 Room temperature control operated by a young adult (D.H.) and an elderly subject (T.M.) at rest wearing 0.8 clo of clothing. Room temperature was kept at 19°C for 30 min before the remote control period which is indicated by arrows (time scale from R to L). Air temperature measured at table height (a) and at a height of 2 m (b); wet bulb temperature (c).

HUMIDIFIER FEVER

J.H. Edwards

MRC Pneumoconiosis Unit, Llandough Hospital, Penarth,
S. Glamorgan, U.K.

Humidification and temperature controls are necessary in environments where individuals have to work for any length of time. The minimum acceptable indoor temperature is around 18°C and ventilation is required to reduce carbon-dioxide, body odour and cigarette smoke to a tolerable level (Brundrett, 1977). Also for certain industrial processes, e.g. manufacturing non-woven fabrics, printing, etc., controls on temperature and humidity are required for the maximal efficiency in processing.

Temperature, air flow and humidity controls can be achieved by a range of systems, the choice depending on many factors such as room volume, number of workers, type of occupation, work process, temperature/humidity requirements, access to outside air, outside temperature, etc., however, in terms of installation and running costs a simple radiator system is far more economical than air conditioning with complete temperature and humidity controls (Ratcliffe, 1977).

Humidity control usually requires the introduction of water into a moving current of air, either as steam or as a fine spray and the latter is achieved by fixed or rotating sprays or by a spinning disc type spray. In some systems (as described by Pickering et al., 1976) baffle plates may be used to eliminate large droplets from moving air, also any unused water is usually recirculated to the spray. Organic dust and microbes taken into the humidification system via the air or from the water supply can be deposited on the baffle plates and within the air/water mixing chamber such that a build-up of organic material and a biomass can build up (Hughes,1977). Microbial products, allergens, cysts, spores, etc., may be liberated into the atmosphere by air passing over the surface of the biomass (Hughes, 1977) as well as from the microbes developing in the recirculation water.

Under appropriate exposure conditions, susceptible individuals may succumb to humidifier fever - an influenza-like illness with pyrexia (high temperature) and malaise (feeling unwell) (Banaszak et al., 1970), as the main symptoms but also cough, dyspnoea (difficulty in breathing) and weight loss may be seen (Fink et al., 1976). Industrially related humidifier fever tends to start towards the end of a shift or at night, usually on a Monday (Friend et al., 1977), i.e., the first day back to work after a rest period (Pickering et al., 1976; Friend et al., 1977). Individuals usually recover by the next day (Friend et al., 1977) and no further episodes may take place until after the next break from work. The disease is usually of the winter months probably due to the large amount (up to 98%) of fresh air drawn into the humidifier during the summer (Pickering et al., 1976) also the tendency to open windows and doors in the summer minimizes disease incidence. Fink et al., (1976) have described acute and insidious forms of the disease and Miller et al. (1976) a chronic case of humidifier fever. The acute phase occurs as described with a fever up to 40°C; bibasilar and endinspiratory rales (chest sounds) were prominent usually resolving

within 12-18 hours. A leucocytosis (increase in white blood cells) was seen and elevated levels of immunoglobulins (antibodies) observed. Lung function testing showed a reduced vital capacity (lung volume) and decreased diffusing capacity (ability to transfer oxygen to tissues). X-rays also showed changes and lung biopsies confirmed these.

The insidious form is characterized by a progressive respiratory disability of the affected individual and acute episodes are rare. The pulmonary function findings and chest X-rays were similar to the acute form as were the lung biopsies.

In one case of chronic hypersensitivity lung disease due to a contaminated humidifier, Miller et al. (1976) described one individual with a recurrent lung inflammation, dry cough, occasional wheezing and chest sounds. The X-ray showed recurrent pattern of changes and lung function testing showed a small airways obstruction. In all forms of humidifier fever relief was obtained by removing the offending humidifier from the environment or avoiding the place of work where the humidifier was in operation (Fink et al., 1976; Burke et al., 1977). Therapeutic relief could be obtained from corticosteroids (Fink et al. 1976; Sweet et al., 1971).

Evidence of sensitization to humidifier material is provided by the presence of precipitating antibody (positive blood test) against humidifier material, e.g. an extract of baffle plate sludge (Pickering et al., 1976), although the presence of antibody is not necessarily indicative of disease (Longbottom, 1977; Edwards, 1977). Skin (prick) testing using humidifier water extracts may produce a positive, immediate response and inhalation challenge with the same extracts can reproduce the illness (Friend et al., 1977).

The source of allergen in humidifiers has proven difficult to isolate (Keller et al., 1972). Several workers have commented on the inability of extracts of isolated organisms to reproduce the precipitin, skin test or inhalation challenge responses of the humidifier extracts (Pickering et al., 1976; Edwards, 1977). Banaszak et al. (1976) considered Thermoactinomyces vulgaris to be the causative organism and the same group in other publications (Fink et al., 1971; 1976) incriminated this and other thermophilic actinomycetes (heat loving bacteria). This is due to isolation of these organisms from the humidifiers, the association of thermophilic actinomycetes with other lung diseases, e.g. farmer's lung (Pepys and Jenkins, 1965), mushroom worker's lung (Sakula, 1967), the positive gel diffusion reaction with sera, i.e. positive blood test, from cases and the reproduction of the disease using nebulized (aerosol) extracts of T. vulgaris (Banaszak et al., 1970). Other workers have considered thermotolerant bacteria to be a more likely source (Kohler et al., 1976). However, it must be pointed out that T. vulgaris is ubiquitous (Waksman, 1970), that extracts of this organism produce non-specific gel diffusion reactions with about 50% of normal sera and contain pyrogenic material capable of inducing pyrexial episodes in rabbits (Edwards, in press). Also farmers exposed to high numbers of T. vulgaris spores (Lacey and Lacey, 1964) rarely respond to this organism by producing a positive blood test. Our studies have revealed that protozoal allergens are present in humidifier sludge (Edwards et al., 1976). Of 18 workers in an office affected by humidifier fever, 16 had a positive blood test to extracts

of humidifier sludge (Edwards, in press). Only 2/18 non-affected but exposed individuals had a positive test ($P < 0.001$). Reactions to extracts of the amoeba Naegleria gruberi correlated absolutely with these findings and amoebae were isolated from the humidifiers (Edwards et al., 1976).

It is, of course, probable that more than one type of organism can produce humidifier fever; workers in a sewage processing plant produced episodes of pyrexia in response to the inhalation of Gram negative organisms (Rylander, 1977). Longbottom (1977) investigating three outbreaks of humidifier fever in Britain, found extracts of the humidifier material to contain similar minor allergens but with no reactions of identity between the major allergens. In order to check this, three sources of material from three outbreaks of humidifier fever in Wales, Spain and Sweden were tested against sera from each outbreak.

We found that two of the extracts produced multiple precipitin lines with three sera from these outbreaks. There was a join in precipitin lines indicating identical allergens and antibodies in the three geographically distinct outbreaks.

Microbial analysis revealed amoebas to be present in these humidifier materials. Thus, common antigens are present in these extracts but some allergens appear to be specific to an extract. Amoebae extracts are in general less reactive than humidifier sludge, but there is a great deal of difficulty in concentrating amoebae extracts due to extraneous materials, e.g. gel material from amoebae plates, K. aerogenes with amoebae cysts.

Therefore it is possible that one allergen source may be used in a blood test in other outbreaks. However, other workers (Longbottom, 1977) have not found this to be so, and certainly other microbial complexes can produce allergens giving the same disease syndrome, e.g., Sewage Sludge Disease (Rylander, 1977) and the findings of the USA workers (Banaszak et al., 1970; Sweet et al., 1971) although capable of an alternative explanation cannot be ignored.

The presence of antibody may be used as a monitor of successful remedial action, i.e., by quantitation or by investigating the serology of individuals newly exposed to a previously offending environment. It is perhaps one of the more fortunate aspects of humidifier fever that removal from the offending environment leads to cessation of symptoms, indeed, this may be a diagnostic procedure in certain cases (Fink et al., 1976). Obviously, discontinued use of humidification is effective (Arnow et al., 1976). Other remedial measures have included changing from water to steam humidification (Pickering et al., 1976) or running water from the systems to waste. The addition of biocides to these systems is not always effective (Pickering et al., 1976). In our cases the disease was eliminated by cleaning the humidifier and installing a prefilter which removed more than 99.9% airborne particles such that a biomass did not build up in the humidifier (Edwards, in press). In one outbreak air from vacuum and compressor pumps produced disease due to contamination of the water used in the pumps. This was eliminated by blowing the air out of the factory (Friend et al., 1977).

The nature of the induction of the pyrexial episode is not certain. The offending agents are unlikely to affect the thermoregulatory centres

of the brain directly but rather indirectly through the release of pyrogen (heat producing) granules from polymorphonuclear leucocytes (white blood cells). It is known that this can occur in response to allergen and antibody combination (Cranston, 1977) by endotoxin activity (Elin et al., 1976) or by lymphocyte lymphokine induced pyrogen release from polymorphonuclear leucocytes (Chao et al., 1977).

The presence of antibodies in the sera of exposed but non-affected workers (Edwards, 1977) argues that if allergen/antibody induced pyrogen release causes the pyrexial episode then either the amount of pyrogen granules released by polymorphonuclear leucocytes differs from individual to individual or that there is a difference in the response to released pyrogen granules between individuals. Certainly, the variation in response to drugs is a universal phenomenon and certain groups of responders may emerge, viz., Fish et al., (1976). However, it may be that the form of allergen presentation might be an important feature in this disease.

Summary

Current findings in humidifier fever are reviewed. Whereas previous work has suggested thermophilic actinomycetes to play a causative role, our findings implicate protozoa, e.g., amoebae, as sources of antigen.

We have found common antigens in three outbreaks of humidifier fever from different parts of the world. Since the nutritional cycle of protozoa is complex it may be that breaking one part of the food chain by biocides or other procedures could result in the elimination of the antigen biomass. Thus, the universal elimination of humidifier fever may be possible.

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HUMIDIFIER FEVER

J.H. Edwards

Practical Considerations

Humidifiers likely to produce humidifier fever allergens are those where water is recirculated. Steam injection humidifiers do not lead to antigen development.

The method of testing for allergens is to concentrate the water about one thousand times and use serological tests with known positive sera.

If the humidifier is positive then it has to be cleaned and steps taken to ensure no allergen build-up occurs.

Since there is opposition by the Health and Safety people to adding biocides, indeed most general acceptable biocides are ineffective, then prevention of allergen build-up must rely on other procedures. It has been found that both

i. running water to waste

ii. running out (draining) the recirculating water reservoir every day

are effective in preventing build-up. It may be that a monitored amount left to run out continuously will be the best answer, otherwise convert to steam.

If you are concerned about a humidifier send about 1 litre of water from it to:

Dr. John Edwards,
MRC Pneumoconiosis Unit,
Llandough Hospital,
Penarth,
South Glamorgan.

and it will be tested for humidifier fever allergens.

POLLEN PROBLEMS

J. Mullins

Asthma Research Unit, Sully Hospital, Penarth, S. Glamorgan

The air we breathe varies at different times and different places and may be considered variously to be relaxing or invigorating or plain disagreeable. Historically the wind has been thought to carry disease and the night air to be most 'unhealthy'. Now the air contains chemical and radioactive pollution introduced by man but it has long contained a form of 'biological pollution', introduced in particular by plants and fungi, which use the air as a means of dispersing seeds, spores and pollens. These airborne particles are referred to collectively as the airspora. The average pollen and spore concentration recorded for the 7th May in Cardiff (over a 10 year period) is 1479 spores and 59 pollen grains per cubic metre of air.

These airborne pollens and spores can cause allergies to individuals. Hayfever is an illness caused by pollen; in Great Britain it is most often due to grass pollen. It affects some 0.5 per cent of the population and of these some 30 to 40 per cent also get summer asthma due to the same cause, each year around a quarter of a million people are obliged to go sick from hayfever. Asthma affects around 1-2 per cent of the population and can cause much incapacity with approximately 35 per cent of asthmatics being occasionally, and 15 per cent frequently incapacitated. Each year between 150 and 200 patients are admitted to the Cardiff hospitals with severe asthma and 4 to 5 of these die. Nationally the figures for deaths from asthma have varied from year to year but reached a peak in the 1960's when as many as 440 asthmatics died in England and Wales, in one year alone.

Airborne spores originate from mosses, ferns, algae and fungi and pollen comes from wind pollinated or anemophilous plants.

The census of airborne pollens and spores is carried out by means of the Hirst spore trap (Figure 1) in which air is drawn through an orifice onto a sticky glass slide moving up past the orifice. Pollens and spores are impacted onto the surface of the slide and are subsequently identified and counted by microscopy. Air is drawn through the trap at a constant rate of 10l/min so that counts can be expressed as the number of spores in a cubic metre of air.

The sampling rate of the Hirst spore trap (10l/min) is roughly equivalent to the ventilation rate of a small adult when sleeping but in a large adult doing strenuous work the figure would be closer to 120l/min, so that an individual will increase his own exposure to pollen and spores by strenuous exercise or work, as a sedentary person will take as much as 1½ hours to breathe a cubic metre of air in contrast to one doing hard physical work who will take 10 minutes.

Spore concentrations vary considerably from day to day and from week to week. When there is a ground cover of snow the air can be expected to be clear of pollen and spores. Spore concentrations reach their maximum in summer and we have recorded a spore concentration of 161,000/m^3 and the grass pollen concentrations once reached 2088/m^3 in the centre of Cardiff.

Pollens and spore concentrations diminish with distance from the source, the spore concentration has been suggested to vary with the inverse of the first power of the distance from source, the inverse of the square of the distance from source and the inverse of the cube of the distance from source. In fact the relationship has proved to be more complex depending on topographic and meteorological conditions. It is possible for pollen and spores to be transported over large distances and this is of great significance in the spread of plant diseases, however highest concentrations of pollens occur nearest to the source. It has been estimated that a square yard of relatively poor grassland near Cardiff was producing 280 million pollen grains per day at the height of flowering or over 2,000 million during the entire hayfever season, which corresponds to the production by an acre of meadow of about one hundredweight of grass pollen during the season.

It is found that the time at which pollens of different plants appear in the air each year follows a fairly constant pattern so that when data has been accumulated for several years it is possible to use this in the production of a calendar (Figure 2) which is used as a guide to which particular pollens may be causing a patient's annually recurring symptoms and then as a guide to when medication will be required.

We find that certain of the pollens are more likely to cause recurrent symptoms year after year than others. A comparison of total catches of pollen of different plants over several years indicates that there is a great deal more variation from year to year in some plants than others, so that in some years although the pollen will be recorded at the appropriate time each year, it will not reach clinically significant concentrations (Figure 3). Except, that is for the people who have a local source, such as a particular tree in their garden or who live alongside a wood.

In addition to distinct seasonal variations pollens and spores are subject to variations during the course of the day, and these diurnal or circadian rhythms follow various patterns, with morning, noon and evening peaks being found, or the incidence of the spore being associated with a particular meteorological event, such as a rain storm. In recent studies at Cardiff we have found that grass pollen shows a very distinct circadian rhythm with highest concentrations occurring during the evening (Figure 4).

A comparison of meteorological conditions during the day indicates a similar tendency and serves to explain the variation of grass pollen. The anthers in grass are hygroscopic and respond to changes in relative humidity, opening as the humidity falls and closing as the humidity increases; as a consequence, grass pollen is only available for release into the air at certain times of day.

The direction of the wind also plays a major role in the incidence of grass pollen in Cardiff, concentrations being lower when the wind is from a generally Southerly direction, which is accounted for by the presence in that direction of the Bristol Channel.

Regional differences in pollen concentration do occur, and these do not remain constant from year to year. So that in one year whereas one part of the country is experiencing a season of high pollen counts another part of the country might be experiencing low

pollen counts with few patients complaining of symptoms. Just such an event occurred in 1976 when South Wales was experiencing drought conditions and we were recording atypically low grass pollen counts during the hayfever season. Meanwhile our colleagues in Derby were recording record high concentrations.

In the production of every pollen season two factors have to be taken into consideration, firstly the weather conditions when the plants are growing and the flowers are produced, on these conditions will depend the number of pollen grains potentially available for release, secondly the weather conditions pertaining when the anthers are mature, for on this will depend whether the pollen produced is released. The conditions favouring a high grass pollen season are a mild spring followed by periods of sunny weather in early summer.

In addition to regional differences there are national differences, such as the tendency for the grass pollen season to occur earlier in Southern Europe than in Britain, in addition the climate of other countries may favour growth of different plants which may in turn be a major source of allergenic pollen, in this respect the presence of so many Birch trees in Scandinavia results in a higher incidence of hayfever due to Birch pollen than found in Britain. In the U.S.A. grass pollen is a minor allergen in comparison with Ragweed, and in southern Europe the pollen from citrus trees and Olive trees are known to cause hayfever.

It is apparent that the dominant source of pollen and spores for an allergic patient is essentially extramural, our studies indicate that provided the doors and windows of a room are kept closed, the pollen concentration within that room will be negligible, and our advice to hayfever sufferers who come home from work with a runny nose and itching eyes and throat, is to keep a room in their house closed up in this way with the curtains drawn during the day to prevent the room from getting too hot, and when they get home to sit in this room and their symptoms will disappear within half an hour. What we recommend them not to do is to go outside in the evening (when the pollen count is high) and run around playing tennis or cutting grass or digging the garden (and so increasing the ventilation rate of the lungs and hence the number of pollen grains which will be inhaled).

Grass pollen grains are in the region of 20 μm in diameter and tend to penetrate far into the respiratory tract, hence their major effect in causing hayfever rather than asthma, their deposition rate in still air is satisfactorily predicted by Stoke's law.

$$v_s = \frac{2}{9} \frac{\sigma - \rho}{\mu} \cdot g r^2$$

where at ordinary temperature and pressure:

v_s = terminal velocity in cm/s

σ = density of sphere in g/cm^3

ρ = density of medium

g = acceleration of gravity

μ = viscosity of medium

r = radius of sphere in cm

The terminal velocity of grass pollen is in the region of 3 cm/s, but no pollen has a terminal velocity much below 1.5 cm/s. So that the net effect of keeping a room closed up is to allow the pollen to settle out of the air in a matter of a few hours. The terminal velocity of fungal spores may be less than 0.03 cm/s, so that getting a room spore-free by allowing sedimentation may be more difficult.

Air conditioning units of different kinds have been marketed for hayfever sufferers, portable units are available to sit on a table within a room, the idea being that the patient sits directly in the air stream produced by the machine, but the only really effective form of air conditioning unit should replace the window as a source of fresh air and should filter incoming air to remove pollen and spores as far as possible. A filter capable of trapping 10 micron particles is adequate. With respect to air conditioning units, some thought should also be given to the siting of the filters and their replacement, as we found when studying the air in an operating theatre. Occasional, unexplained, sudden increases of spore concentration resulted in the theatre being closed until the concentration fell. Attention was paid to the filters to see that they were not choked, but we were assured that the filters were regularly replaced, as indeed they were, but the siting of the filter changing area in the scrub room of the theatre caused a cloud of carefully filtered spores to be released into the air of the operating theatre whenever the filter was changed.

Overall the home is a favourable environment for patients allergic to pollens, if care is taken to ensure that windows remain closed. If windows are open then the airspora inside the house closely mirrors that outside and the occupant will experience similar concentrations to those outside. The housewife who makes up floral decorations of catkins may succeed in producing higher concentrations inside than outside for a short period. In their ability to grow within the house the moulds are more likely to influence the spore content of inside air independent of outside spore concentrations. Similarly if dry rot is present in a house the production of a fruit body will result in high concentrations of spores throughout the house.

Overall, a building which is effectively sealed off from the outside environment provides a potential haven for hayfever sufferers. Often the greatest problem is persuading other occupants of the building to manage without 'fresh air' and its pollen load.

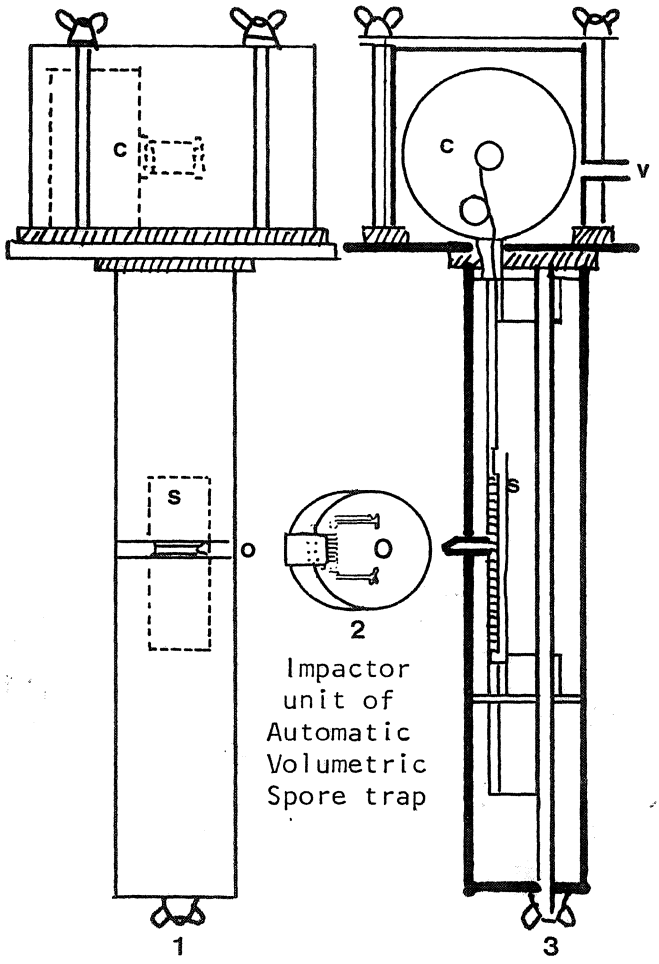
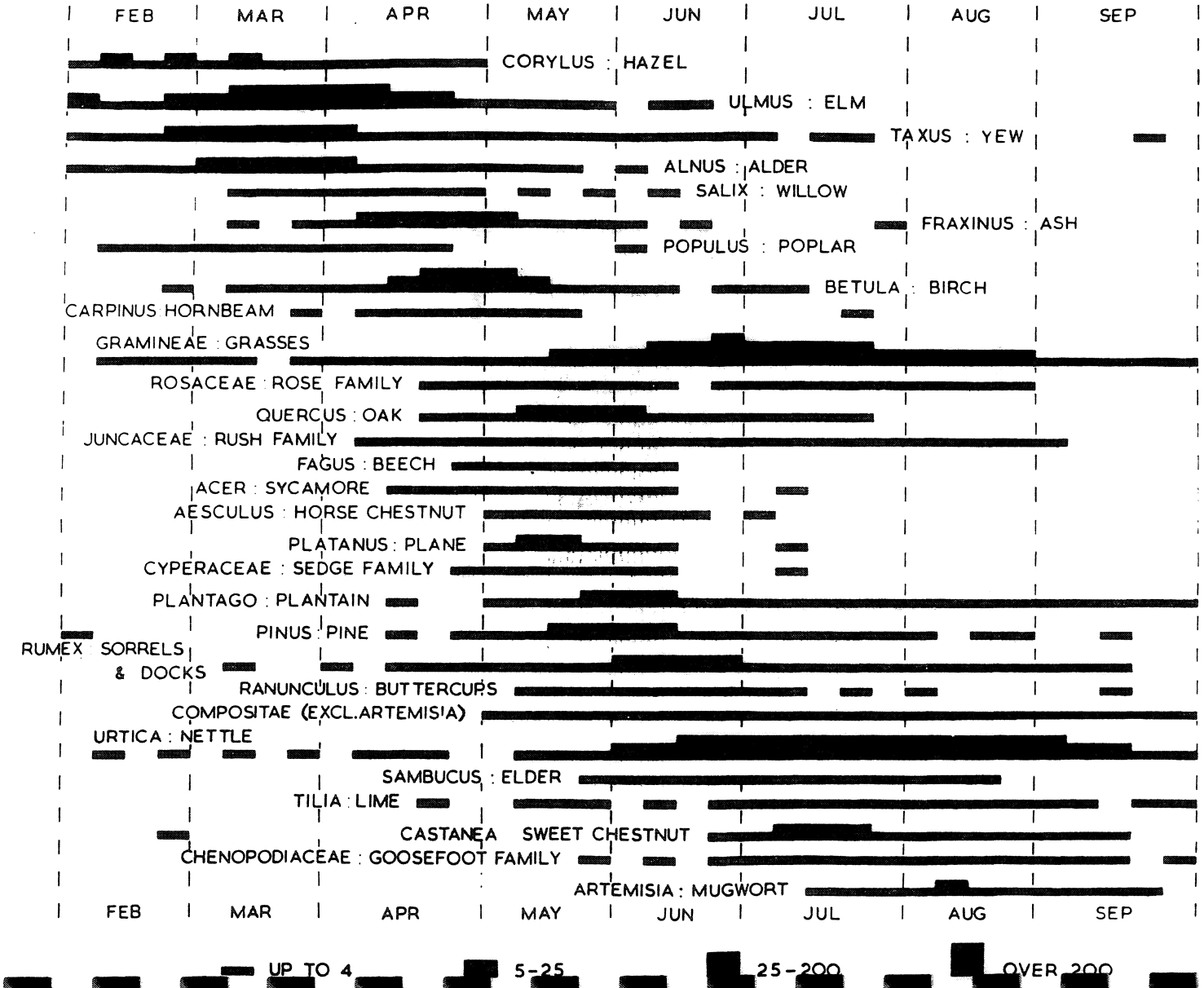


Figure 1 Diagram of the Hirst volumetric suction trap: 1 = elevation facing wind; 2 = plan of section through orifice; 3 = elevation side view; **s** = sticky microscope slide; **c** = clockwork drive for raising slide; **o** = orifice facing wind, with slide in holder moving up past orifice; **v** = connection to vacuum pump. (Wind-vane and cap to protect orifice from rain are not shown.)

FIGURE 2 - POLLEN GRAINS IN THE AIR AT CARDIFF 1962 — 1971

MEAN 24 HOUR COUNTS AVERAGED WEEKLY IN GRAINS PER CUBIC METRE OF AIR



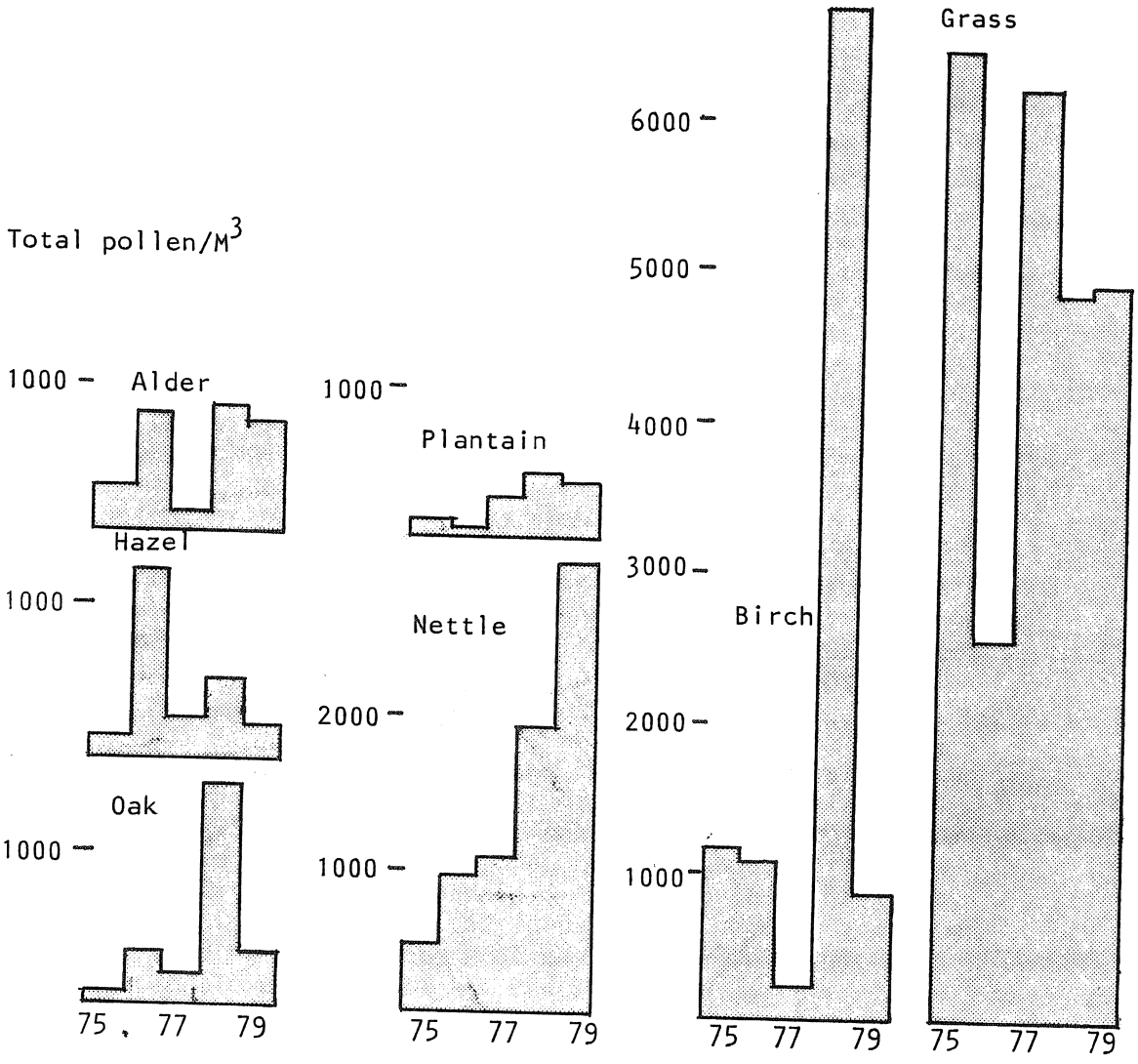


Figure 3 The incidence of pollen over several years showing the marked annual variations.

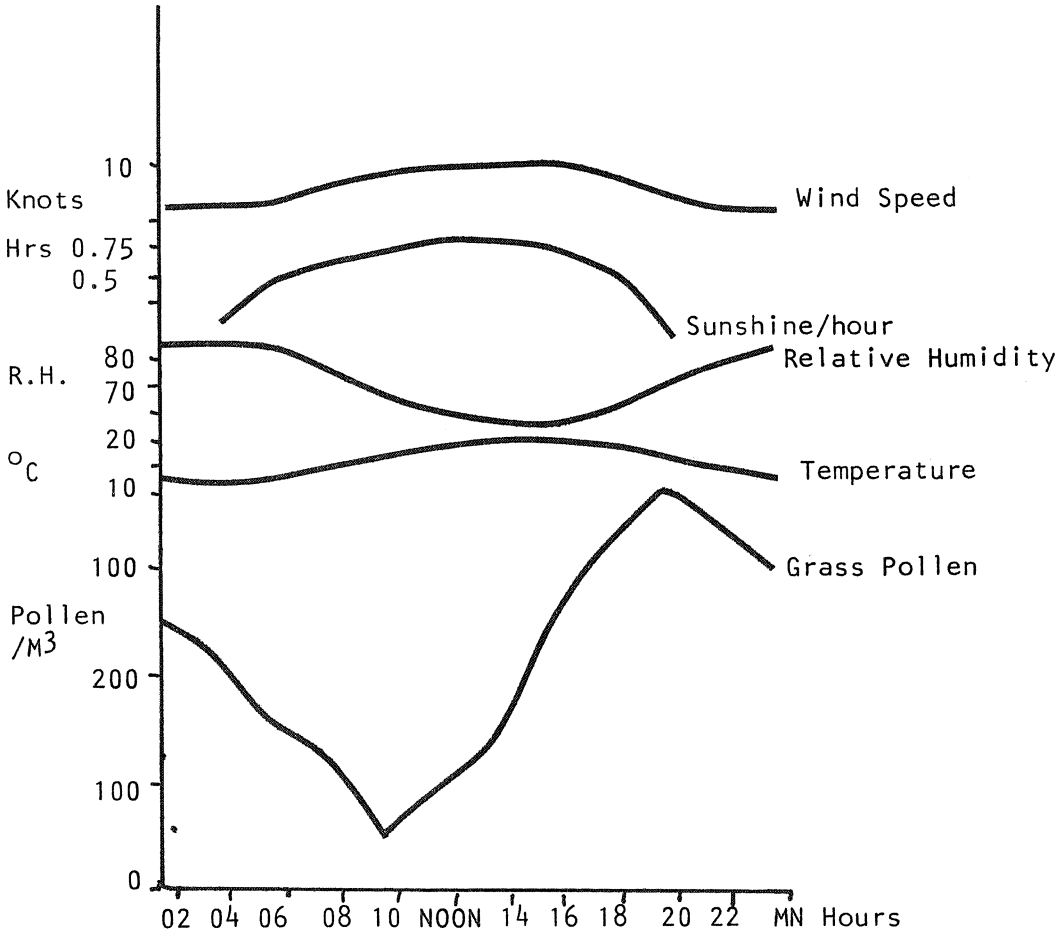


Figure 4 Circadian rhythms of grass pollen, wind speed, sunshine, relative humidity and temperature. These variations are remarkably constant from year to year.

ALLERGY TO MITES IN HOUSE DUST AND THE DOMESTIC ENVIRONMENT

A.M. Cunnington

(lately Slough Laboratory, Ministry of Agriculture, Fisheries and Food, Slough, Berkshire)

In the human environment mites play as important a role as insects but because of their small size are often overlooked and their significance and importance unrealised. Yet in terms of species and numbers they form one of the largest groups in the animal kingdom. Zoologically speaking, they belong to the class Arachnida, that is to say they are more closely related to spiders and scorpions than they are to insects with which they are often confused. They are distinguished from insects by their small size - many are barely visible to the naked eye - by their lack of body segmentation and by the possession of eight legs in the adult stage. They are cosmopolitan in distribution and occupy many varied and diverse habitats. Some are plant feeders, some scavengers living on decayed animal or plant remains, while others are either predators feeding on other small arthropods or mites, or parasites living either internally or externally on their hosts. Many are of considerable economic importance and are serious pests of orchard and field crops, and of stored foodstuffs, causing serious damage and economic loss. Some parasitic species have considerable medical and veterinary importance as vectors of disease organisms, or by virtue of their direct effect on their hosts.

Many different species occur in houses but most domestic infestations are usually temporary and the result of some special circumstance or action, e.g. the intrusion of plant mites from the field or garden, the occurrence of parasitic species brought about by the presence of or contact with infested animals or birds or their habitats, or the purchase and storage of infested foodstuffs. People are sometimes alarmed at finding large numbers of small, dark coloured mites with a hard shiny cuticle clustering on the walls and windows of their houses, or more particularly, especially during the spring and autumn months, of similarly large numbers of small reddish mites. These are (i) Oribatid mites, often called beetle or moss mites and (ii) species belonging to the *Bryobia praetiosa* complex commonly called clover mites. These species are quite harmless and their occurrence is usually the result of disturbance of their natural habitat by building or other operations. A cause of more serious discomfort are the occasional intrusions of parasitic species. The Mesostigmatid mite *Dermanyssus gallinae* is normally a parasite of domestic and wild birds but in the absence of avian hosts will attack man, sometimes causing an acute inflammatory dermatitis. This usually happens when nests are deserted, either because of disturbance or migration of the birds, and the mites then invade the house seeking alternative hosts on which to feed. A similar itching pruritic dermatitis may result after handling pet dogs or cats infested by the parasitic Cheyletid species *Cheyletiella yosguri*. Mention may also be made here of the scabies mite *Sarcoptes scabiei* but the special circumstances relating to the incidence of this disease in human beings are not relevant to this discussion.

Of perhaps more serious concern here are those species which may be considered to be residual or endemic in houses. These are the house dust mite Dermatatophagoides pteronyssinus, the house mite Glycyphagus domesticus and various storage species. House dust is a common and serious cause of allergic asthma and rhinitis but for many years, despite much research, the nature and source of its allergenicity remained unknown. Recent research, however, has demonstrated that house dust supports a considerable mite fauna among which pyroglyphid mites, usually D. pteronyssinus, predominate. These species have been shown to be the major sources of the house dust allergen and causal agents of most house dust allergy and asthma.

Dermatophagoides pteronyssinus is present in virtually every home where it feeds on the shed human skin scales present in house dust. For this reason it is most abundant in the surface dust of mattresses and bedding and sometimes in the dust from old upholstered furniture. Laboratory studies have shown that the mite cannot survive long in dry conditions and requires a moist environment in which to live. Field studies have confirmed these results and revealed that it is far more numerous in dust from old damp houses than in that from modern, centrally heated dwellings. A recent study in which house dust from the homes of a number of patients with house dust allergy and asthma were examined for mites showed that the numbers of D. pteronyssinus found correlated well with the degree of dampness present in the houses. It is clear that dampness in houses favours the growth and development of house dust mite populations while dry conditions inhibit their increase (see Table 1). Clinical studies have shown that the greater the numbers of mites present in the dust the greater the potency of the dust to which occupants of the house are exposed.

Table 1. Relationship between mite numbers and dampness in houses

Category	No. of houses	Mean no. mites per 100mg dust	Range
Very damp	12	600	400-1000
Damp	15	280	100-400
Fairly dry	14	64.3	0-100

The house mite Glycyphagus domesticus is, as its name implies, a common inhabitant of houses and other dwellings where it is particularly associated with damp conditions, the mites feeding on the moulds growing on damp wallpaper and other furnishings. Such conditions are less favourable for the growth of P. pteronyssinus populations but provide optimum conditions for the rapid development and increase of G. domesticus, enormous numbers of which have been found on furniture covered with rush or sea green fibre which has become mouldy. Until recently, apart from the natural distaste of householders to the presence of myriads of these tiny creatures crawling over walls or furniture, they were considered to be harmless and to cause no damage to the fabric or furnishings of the house or to present hazards to the

health of its occupants. Recent research, however, has shown that G. domesticus like D. pteronyssinus is a potent allergen and is responsible for the allergic respiratory disorders of many people. It is also sometimes a pest of stored foodstuffs although of less importance than other storage mites. These species are also important allergens and although the people most at risk are those likely to encounter infested produce during the course of their work, e.g. farmers, flour millers, grain merchants, etc., exposure to storage mites may also occur in the home. For example, animals such as hamsters, guinea pigs and rabbits are popular children's pets. Straw and hay are widely used as bedding materials for these animals and cereal grains and seeds for food. These products are often infested or become infested during storage. The storage of relatively large amounts of flour or other cereal products, a practice common along some immigrant families, may also act as a source of infestation and exposure to these mites.

These species, however, cannot long survive exposure to dry, warm conditions. Glycyphagus domesticus and other storage mites are unable to develop at or below 60% relative humidity, conditions also unfavourable for the growth of D. pteronyssinus. The most effective control measures, therefore, are (1) those designed to prevent or remedy the causes of dampness in houses and (2) those carried out by the householder, such as the regular vacuum cleaning of mattresses and bedding, dust elimination and other environmental control measures, which aim at preventing or reducing the build-up of house-dust mite populations.

House dust and storage mites are important agents of allergic respiratory disease. An awareness of their allergenicity and of the circumstances under which they can occur should help towards the avoidance and prevention of this source of allergy.

INDOOR AIR POLLUTION AND ITS EFFECTS ON HEALTH

BY

R.J.W. Melia, C. du V. Florey, S. Chinn, B.D. Goldstein[1], A.G.F. Brooks[2], H.H. John[3], D. Clark[4], I.B. Craighead[3], X. Webster[3]

Department of Community Medicine, St. Thomas's Hospital Medical School, London, SE1 7EH.

[1]Institute of Environmental Medicine, New York University Medical Centre, New York, NY 10016, USA.

[2]MRC Toxicology Unit (Clinical Section), St. Bartholomew's Hospital Medical College, Charterhouse Square, London, EC1M 6BQ.

[3]Cleveland Area Health Authority, Marton House, Borough Road, Middlesbrough, Cleveland, TS4 2EJ.

[4]Middlesbrough Borough Council, Environmental Health Department, Vancouver House, Central Mews, Gurney Street, Middlesbrough, Cleveland, TS1 1QS.

INTRODUCTION

In the past, research into the effects on health from air pollution has concentrated on outdoor pollutants. In the U.K. attention was drawn to the problem of high levels of atmospheric smoke and sulphur dioxide (SO_2) by the rapid increases in mortality and morbidity rates which occurred during episodes of smog (Ministry of Health, 1954). Major disasters of this kind have not arisen from pollution inside the home and the indoor environment was thought to provide protection from pollution outside. SO2 for example is absorbed by fabrics and soft furnishings so unless there is an indoor source the concentrations of SO_2 tend to be lower inside than out (Benarie et al. 1974; Biersteker et al. 1965). However, it is now realised that indoor sources of pollution can cause higher levels of certain pollutants such as oxides of nitrogen and carbon monoxide inside than outside the home (Goldstein et al. 1979; Wade et al. 1975).

The levels of atmospheric smoke and SO_2 in the U.K. have been declining over several decades (Weatherley et al. 1976) and their effects on respiratory illness are no longer easily detected by epidemiological methods (Lawther et al. 1970; Lunn et al. 1970). It is therefore possible that effects from indoor pollution may become more apparent than in the past. Among the pollutants which could be associated with respiratory disease are those which arise from tobacco smoking, and the combustion of gas and oil (Table 1). Coal fires may have been at one time a source of indoor pollution but changes in the type of fires and coal have helped to reduce the levels of pollution from this source in the home.

Pollution from other sources indoors are also potentially harmful to health (Table 1). Asbestos used for thermal insulation and radon

emitted from building material may be hazardous in the long term because of their carcinogenic properties. Carbon monoxide coming from the combustion of gas and present in tobacco smoke interferes with the transport of oxygen in the blood and may exacerbate the condition of cardiorespiratory patients. Formaldehyde released from building materials such as particle boards and foamed insulation can cause irritation to the eyes and respiratory tract. There is also a variety of other pollutants too numerous to mention here which are released from household goods such as sprays. Pollutants known to be harmful at work can also be present at home because the same materials are used for hobbies.

Concern about the indoor environment is increasing as ventilation rates are being reduced to conserve energy and the levels of pollution may become higher than they were in the past. Guidelines for the control of certain pollutants have been proposed by bodies such as the World Health Organisation. Recommendations have been made for the maximum levels above which people should not be exposed but there is much criticism of these guidelines. Some of the guidelines have been based mainly on the results of animal experiments which are not necessarily applicable to man. Further information has come from studies of occupational health but the effects on health and levels of exposure may differ for the general population. There have been many epidemiological investigations into the ill effects of outdoor air pollution but these have been mainly concerned with pollutants such as suspended particulates and SO_2. At a workshop on indoor air pollution organised by WHO (1979) it was recognised that there was a need for research into the indoor environment and its effects on health.

The effects on health of some pollutants are difficult to study epidemiologically because we do not have suitable measuring instruments for use in large surveys. However preliminary investigations may be conducted by using sources of pollution to identify groups of the population exposed to different levels of pollution. Both tobacco smoking (Colley et al. 1974; Leeder et al. 1976) and fuels used for cooking in the home (Melia et al. 1977; Melia et al. 1979) have been employed in this way. The main disadvantage of this method of research is that several types of pollutants may arise from one source so further work is required to identify which of them is causing ill effects on health. Other pollutants cannot be studied by their source because the population cannot easily be divided into exposed and non-exposed groups.

The results of investigations into the association between respiratory illness in primary schoolchildren and use of gas for cooking in the home illustrate the type of epidemiological studies which may be conducted.

INVESTIGATIONS INTO THE EFFECTS OF GAS COOKING

The National Study

An association between respiratory illness and use of gas for cooking in the home was first observed in a National Study of Air Pollution. Six to 11 year olds from selected primary schools in a random sample of 22 English and six Scottish areas were studied annually from 1973 to 1977. Information on the presence of cough, wheeze, colds going to the chest and

history of asthma and bronchitis was collected in a questionnaire completed by the mothers. Questions were also asked about socio-economic characteristics of the home such as the father's social class, and indoor sources of pollution such as the types of fuel used for cooking and heating, and, in 1977 only, the number of smokers in the home. As new children entered the study each year when they reached the age of six and older children left when they moved on to secondary school, the population examined in 1977 differed from that examined in 1973. The association between gas cooking and respiratory illness was studied in both groups of children to see if it was repeatable in two different populations (Melia et al. 1977; Melia et al. 1979).

The results from both years were compared by calculating the risk of having one or more respiratory conditions in homes where only gas was used for cooking relative to the risk in homes where only electricity was used (Table 2). In each group of children the risk was greater in homes with a gas cooker than homes with an electric cooker. After allowing for differences in age and social class between gas and electric cooking homes this relative risk in boys was found to be similar in both years but the relative risk in girls was greater in 1973 than 1977. In further analyses in 1977 the assocation was observed to be independent of the effects of age, social class, the number of smokers in the home and latitude of the areas but it was only statistically significant in urban areas (for boys $p < 0.005$; for girls $p \simeq 0.08$).

As the gas cooker is an unflued appliance we suggested that indoor air pollution might be the cause of the association. Among the many pollutants which arise in the emissions of gas combustion NO_2 was suspect. NO_2 has been found to cause increased susceptibility to respiratory infection in animals exposed to levels of about 500 ppb for three months (Ehrlich and Henry, 1968) and pulmonary oedema in man exposed to much higher levels in industrial and agricultural accidents (Becklake et al. 1957; Grayson, 1956). Furthermore, weekly average concentrations of NO_2 in kitchens with a gas cooker have been shown to be above the maximum annual mean level of 50 ppb recommended by the United States Environmental Protection Agency (Wade et al. 1975).

At the time of writing our first report (Melia et al. 1977) a small personal sampler for NO_2 became available which was highly suitable for use in surveys (Palmes et al. 1976). Before we embarked on a large study of the relation between NO_2 and respiratory illness we tested the reliability of the sampler.

Sampler experiment

The sampler consists of an acrylic tube about 3 in. long with an internal diameter of $\frac{3}{8}$ in. which contains an absorbant specific to NO_2. The sampler depends on the molecular diffusion for the collection of gas so no pump or electrical supply is required. The total amount of NO_2 which has been absorbed over a measured period of time is determined by spectrophotometry to obtain an average measure of the concentration in the atmosphere.

In a designed experiment conducted in two gas and two electric kitchens we found that the reliability of the sampler was highly satisfactory: the measurement error being 1.2 ppb (Melia et al. 1978). When the measurement from the sampler was compared with the measurement taken by the chemiluminescent method it was concluded that the accuracy of

the sampler was better than ±10 per cent when used in domestic kitchens (Apling et al. 1979).

The Cleveland Respiratory Study

The main aim of the study was to investigate whether lung function was related to levels of NO_2 in the home (Florey et al. 1979). We chose to study young primary schoolchildren coming mainly from the manual social classes and living in an urban area of northern England who had a high risk of developing respiratory disease. 808 six to seven year olds who both lived and attended school within a defined area of Local Authority housing in Middlesbrough were included in the study in February, 1978. Outdoor air pollution might have interfered with our results but levels of smoke and SO_2 which were already being measured at two sites in the area were low (annual means ≤ 30 $\mu g/m^3$). Outdoor levels of NO_2 which were measured at 75 sites set up in the study area by staff of the Middlesbrough Borough Council for one week in February were found to be low and similar across the area (range: 14 to 24 ppb).

Height, weight and lung function were measured on each child at school by trained fieldworkers from St. Thomas's Hospital. Lung function was measured using the McDermott Dry Spirometer supplied by the MRC Toxicology Unit. School nurses from the Cleveland Area Health Authority were responsible for the distributions of NO_2 samplers and questionnaires. Measurements of NO_2 were taken by two samplers placed for one week in the kitchen of each home and in a random 25 per cent of homes a third sampler was placed in the child's bedroom. Information on respiratory symptoms and diseases experienced by the child and characteristics of the home was collected in a questionnaire completed by the child's mother.

Data were obtained for 66 per cent of the sample. The levels of NO_2 in the kitchens were considerably higher where gas was used for cooking (range 5-317 ppb; mean 112.2 ppb) than where electricity was used (range 6-188 ppb; mean 18 ppb). Levels in the bedroom were also higher in homes with a gas cooker (range 4-169 ppb; mean 31 ppb) than in homes with an electric cooker (range 3-37 ppb; mean 14 ppb).

The relation between respiratory illness and type of fuel used for cooking was similar to that found in the National Study. The risk of having one or more respiratory conditions was higher in homes with a gas cooker than homes with an electric one ($p = 0.06$) independent of effects from age, sex, social class and smoking in the home. However, no relation was found between the frequency of respiratory conditions and levels of NO_2 in the kitchen or between lung function and levels of NO_2 in the kitchen or bedroom. Only one finding indicated that levels of NO_2 might be related to respiratory illness. Within gas cooking homes the frequency of respiratory conditions was higher in homes with high levels of NO_2 in the bedroom than homes with low levels (Table 3). After allowing for the effects of age, sex, social class and number of smokers in the home the relation between respiratory illness and levels of NO_2 in the bedrooms of homes with gas cookers was statistically significant at the 10 per cent level. No relation was found between respiratory illness in the parents and the type of fuels used for cooking in the home or levels of NO_2.

DISCUSSION

An association between gas cooking and respiratory illness has now been found in three different groups of children in the U.K. Although no association has been found in some studies from the U.S.A. (Lutz et al. 1974; United States Environmental Protection Agency, 1976), Speizer and colleagues (1979) have reported a relation in schoolchildren between use of gas for cooking and both a history of respiratory illness before the age of two and poor lung function.

The suggestion that indoor pollution is the cause of the association needs to be investigated further for the results from the Cleveland Respiratory study show some inconsistencies. An association was found between respiratory illness and levels of NO_2 in the bedroom but not the kitchen. Possibly the levels in the kitchen did not provide an appropriate measure of levels to which the children were being exposed in their homes or peak levels of NO_2 may relate more closely to the frequency of respiratory illness than average levels. Alternatively another environmental agent or social conditions associated with the use of gas may be the cause. We are currently studying the possibility that gas cooking may be associated with high humidity in the home which may lead to condensation, increased ventilation and low temperatures.

The fact that no relation was found between respiratory illness in the parents and gas cooking or levels of NO_2 is not surprising since children tend to suffer from more illness and may be more susceptible to the effects of indoor pollution than their parents. Indoor pollution from tobacco smoking by members of the family did not appear to be related to respiratory illness in the children from Middlesbrough. This may indicate that the effect from gas cooking is more important than that from smoking on primary schoolchildren. In a study of children followed-up from birth to age five (Colley et al. 1974) an association between parents' smoking habits and the incidence of bronchitis and pneumonia was found only during the first year of life and disappeared as the children grew older. The effect of gas cooking was not investigated in this study.

CONCLUSIONS

There is a variety of pollutants in the home which are potentially harmful to health. If ventilation rates are reduced to conserve energy the risk to health may increase as the levels of pollution are raised. Changes in the indoor environment and its effects on health should be carefully monitored. Some pollutants are difficult to study because their effects on health will take some time to develop or because suitable monitoring instruments are not available for use in surveys of health. In preliminary investigations the effects of pollution may be studied indirectly using their sources to identify groups of the population who may be exposed to different levels of pollution. Pollution arising from gas cooking has been studied in this way.

If pollution from gas combustion is indeed harmful to health it might be simply dispelled by improving ventilation in the kitchen. This could be done by opening the windows while cooking or an aid to ventilation such as an air vent, cooker hood or even a flue could be employed.

ACKNOWLEDGEMENTS

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Table 1 Summary of some indoor air pollutants, their sources and potential effects on health

Pollutant	Source	Potential effect on health
Asbestos	Building materials	Cancer
Carbon Monoxide	Gas appliances Tobacco smoke	Hypoxia
Formaldehyde	Building materials	Irritation to eyes and respiratory tract
Nitrogen oxides	Gas appliances Oil fires Tobacco smoke	Respiratory illness
Radon	Building materials	Cancer
Suspended particulates	Coal fires Tobacco smoke	Respiratory illness

Table 2 THE NATIONAL STUDY

The risk of having one or more respiratory conditions in primary schoolchildren from homes where gas was used for cooking relative to the risk in children from homes where electricity was used after allowing for differences in age and social class given by sex and year of examination.

	YEAR OF EXAMINATION	
	1973	1977
BOYS	1.29 ($p < 0.05$)	1.25 ($p < 0.05$)
GIRLS	1.40 ($p < 0.001$)	1.19 ($p \simeq 0.07$)

Table 3 THE CLEVELAND RESPIRATORY STUDY

The frequency (%) of one or more respiratory conditions in boys and girls from homes where only electricity was used for cooking and from homes where only gas was used grouped according to the weekly average level of NO_2 in the children's bedrooms (ppb).
(Total number of children given in brackets)

	Electric cooking homes	Gas cooking homes: level of NO_2 (ppb)		
		4-19	20-39	40-169
BOYS	40 (42)	43 (23)	58 (19)	69 (13)
GIRLS	47 (42)	44 (25)	60 (15)	75 (8)

RADON IN THE HOME

B.L. Davies

National Radiological Protection Board,
Harwell, Didcot, Oxon.

Introduction

The naturally radioactive element uranium is widely distributed in the earth's crust. It is present in most rocks, soils and common building materials at concentrations of a few parts per million (1). Uranium is the parent of a whole series of radionuclides which continue to decay until they reach the stable lead isotope lead-206. The fifth member of this chain is radium-226 which is present at concentrations of a few parts in 10^{12}. The immediate product of the decay of radium-226 is radon-222 which is a radioactive noble gas having a half-life of 3.8 days. As a noble gas radon does not react with other elements and its half-life is long enough for it to diffuse from building materials and from the subsoil into buildings and into the atmosphere. The daughters of radon are radioactive particulates which are isotopes of polonium, bismuth and lead. The first four of these are polonium-218 (an α-emitter of half-life 3.05 minutes), lead-214 (a β-emitter of half-life 26.8 minutes), bismuth-214 (a β-emitter of half-life 19.7 minutes) and polonium-214 (an α-emitter of half-life 164 μS). In view of its short half-life polonium-214 is always in equilibrium with bismuth-214 and for practical purposes bismuth-214 can be regarded as a combined α,β-emitter. These radionuclides are generally referred to as the short-lived daughters of radon and are often known by their historic names of radium A, radium B, radium C and radium C^1 respectively. The radon daughters are inhaled, either as free atoms or attached to dust particles and become deposited in the lungs. As their half-lives are short, virtually all of these deposited radionuclides decay within the lung and give rise to a radiation dose to the cells of the bronchial epithelium, mainly due to the alpha-emissions from polonium-218 and polonium-214.

Exposure, units and effects

In radiological protection work the absorbed dose is measured in grays (1 Gy = 1 joule per kilogram = 100 rads) and the dose-equivalent in sieverts (1 Sv = 100 rem) but in the dosimetry of radon daughters very great difficulties are encountered in applying this concept. Most of the studies of people exposed to high levels of radon and its daughters have taken place in underground uranium and other mines and here the unit of exposure commonly used is the Working Level Month (WLM). One Working Level (WL) is a unit of concentration and is defined as any combination of the nuclides polonium-218, lead-214 and polonium-214 in one cubic metre of air which will result in the ultimate release of 1.3×10^8 MeV of alpha particle energy. The WLM corresponds to a continuous exposure to air containing 1 WL for one working month, assumed to be of 170 hours duration.

Exposure to air containing radon and its daughters results in irradiation of the bronchial epithelium as described above and it would be expected that this would give rise to an increased incidence of lung cancer. For exposures at very high concentrations this is, in fact,

the case, and deleterious effects have been known for a long time (2), even if the true nature of the disease has only been recognised recently. Epidemiological studies were carried out among uranium miners in Czechoslovakia (3) and in the United States (4) and in fluorspar miners in Newfoundland (5) where the radon daughter concentrations were so high that lifetime exposures of several hundreds of WLM were recorded. It was clearly demonstrated that there was a correlation between an excess incidence of lung cancer and high exposures to radon daughters. The risk estimate for miners derived from these figures is about 200 lung cancers per million people exposed per WLM (6).

Measurement of population exposure

In the light of the known hazard from high concentrations of radon daughters in mines various workers have investigated the concentrations in buildings. In general, however, these investigations were confined to those cases where the concentrations were expected to be high, for example in poorly ventilated basements or in buildings with high radium concentrations in the structural materials. It was felt however that the exposure of the population at large should also be measured so the Board carried out a survey of ordinary dwellings chosen to be as representative as possible of the housing stock in the United Kingdom (7,8). The method is fully described in the references and will not be repeated in detail here. The procedure was to determine the rate at which radon diffused into the room air, which was assumed to be invariant for any particular room, and to combine this with estimated or measured ventilation rates to calculate the radon daughter concentrations. By making allowances for the time spent out of doors in the, usually lower, atmospheric concentrations of radon daughters, it was possible to calculate overall exposure.

The rate at which radon diffused into the rooms was found to spread over three orders of magnitude, Figure 1. It did not show any consistent relationship with type of building or constructional materials or with geographical location. The rate tended to be higher, however, in rooms with exposed brick, stone or plaster. Where it was possible to estimate the radium content of the structural materials and hence the radon emission it was usually the case that this was insufficient to account for the radon actually measured. It appears that a considerable fraction of the radon in dwellings comes from the sub-soil and enters through cracks and openings in the floor. This conclusion has been supported by other workers.

The conditions necessary to enable the diffusion rate to be measured required the room to be closed for long periods, so the actual concentrations in WL as found in ordinary circumstances were also measured and showed a very similar distribution, Figure 2.

Despite the very wide range of production rates found it was felt that a representative value should be determined and the arithmetic mean rate was found to be 20 Bq m^{-3} h^{-1}. The concentrations of radon daughters, and hence the exposure in WLM are very markedly dependent on the ventilation rate and for this study an annual average of 1 air change per hour was used. This gives rise to a mean concentration of 0.0035 WL and hence to an average exposure of 0.15 WLM in a year to the population of the U.K.

Possible consequences of the exposure

It is assumed in radiological protection that the dose-effect relationship is linear down to zero dose with no threshold, the effects at low doses being estimated from the known effects at high doses. However, in this case the opinion of the Medical Research Council was that the circumstances of exposure for the miners were so different from those in normal dwellings that the risk estimate derived from the miners was inappropriate and in its stead a figure of 100 lung cancers per million per WLM was suggested. This figure and the average exposure rate to radon daughters derived above suggests an incidence of lung cancer of 15 per million per year attributable to radon.

Because of the ever increasing cost of energy many people are reducing the ventilation rates in their homes to conserve heat. For the same reason buildings are being designed for use at very low ventilation rates, in some cases down to 0.2 air changes per hour. These reduced ventilation rates will cause increased concentrations of radon daughters and will increase the exposure of the population to this source of radiation. It is possible to calculate the increased incidence of lung cancer that might arise from this cause for different mean ventilation rates. The table (9) shows the predicted incidence of lung cancer for a series of reduced ventilation rates for the winter, which is taken to be 7 months long. It has been assumed that the summer ventilation rate is 2 air changes per hour for the remaining 5 months of the year but because of the non-linear relationship between ventilation rate and radon daughter concentration the calculation is not very sensitive to the actual figure chosen for the summer rate. The current incidence of lung cancer in the United Kingdom is 650 per million year; the prevailing exposure of 0.15 WLM in a year would therefore appear to make a negligible contribution to the prevailing incidence.

If this is a real effect, and it must be strongly emphasised that there is no epidemiological evidence for effects at these low rates of exposure, what steps can be taken to reduce radon daughter concentrations when ventilation rates are reduced if this were deemed desirable? There are two approaches: in the first, action is taken to prevent radon entering the living spaces, and in the second the radon daughters are removed from the room by suitable air treatment.

Methods of preventing the entry of radon

As mentioned above, the one constructional factor which did seem to give rise to greater output rates of radon was the presence of bare surfaces in the room. It would seem then that the use of the large areas of rough hewn masonry, bare brick, or unpainted or unpapered plaster might be discouraged. Also since emanation rates from all materials rise for moderate degrees of humidity, the internal structure of the building should be kept as dry as possible.

The most significant route of entry for radon however is through cracks and openings in the floor. In certain parts of Canada and the United States where towns are built on uraniferous outcrops or soils this problem has been particularly acute. It is aggravated by the common practice there of building houses with full or semi-basement. Remedial action by sealing with epoxy resins has cost up to £10,000 and

now, even though the methods have been improved and simplified, averages about £2,000 per dwelling (10). The incorporation of impermeable barriers and other preventive techniques at the construction stage costs much less and for new buildings may well make little difference to the price. Settlement, shrinkage, and other movements of the building may well nullify this approach after a few years.

Trapping the radon in the walls or below the floor means that the radon will decay in situ and therefore the gamma ray dose from the walls and floor will increase. However, the increase represents a much smaller increase in dose than the reduction achieved by preventing radon entering the room.

Methods of removing radon daughters from the room air

With a view to reducing the concentration of radon daughters in rooms having very low ventilation rates, some measurements have been made of the effects of various air treatment devices (11). This work was carried out by the Board under a contract from the Electricity Council Research Centre. The devices used were a humidifier, a dehumidifier, and an electrostatic precipitator. Of these three devices only the precipitator had any effect but in this case the results were dramatic.

The measurements were carried out in a sealed environmental chamber of 33m^3 volume with a ventilation rate of 0.05 air changes per hour Radon was released into the chamber and measurements made over a period of 4 hours of WL value and condensation nucleus concentration. The air was continuously mixed using domestic fans. It was found that in the absence of any air treatment devices the condensation nucleus concentration decreased with time due to sedimentation and attachment to the walls. The radon daughter concentration also reduced slightly with time probably due to plate-out on the chamber surfaces, Figure 3. This effect was enhanced and supplemented by plate-out on the blades of the fans which is a mechanism which has only recently been reported (12). In the subsequent experiments the condensation nucleus concentration was periodically increased by allowing a cigarette to smoulder. When the electrostatic precipitator was switched on it soon reduced both the condensation nucleus concentration and the radon daughter concentration by over an order of magnitude, Figure 4. In the case of the radon daughter concentration it appeared to be a two-stage process, direct removal by the precipitator being supplemented by plate-out on room surfaces as there were far fewer dust particles to which the radon daughters could attach themselves. From this it appears that reducing the dust level in a room will also reduce radon daughter concentrations by promoting more rapid plate-out.

Other methods of reducing radon daughter concentration

One other possible method of reducing the radon daughter concentration relies on the development of very efficient heat exchangers. By using a very high ventilation rate the residence time of the radon could be made so short that very few daughters would be produced in the room and the heat loss could be overcome by using the heat exchanger to heat the incoming outdoor air which would have a very much lower radon daughter concentration. These devices however are only in the very early stages of development.

Conclusions

The reduction of ventilation rates in the quest for economy in energy use will result in increases in radon daughter concentrations in dwellings and hence in radiation dose to the lung tissues of occupants. Risk estimates derived from occupational exposure to high concentrations suggest that there might be an increase in the incidence of lung cancer as a result. This requires us to consider whether and to what degree the exposure of persons to radon daughters in houses might be limited. If it should be deemed necessary to do so, then methods are available to reduce radon daughter concentrations.

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Table

Predicted lung cancer incidence in the UK due to environmental ^{222}Rn daughter concentrations as the mean winter (7 months) ventilation rate is reduced: the summer (5 months) mean ventilation rate is assumed to be constant at 2 air changes per hour.

Winter Ventilation Rate h^{-1}	Mean population exposure WLM y^{-1}	Lung cancer incidence predicted per 10^6 population per year	Number of cigarettes smoked per week to give the same lung cancer incidence. (Derived from Ref.13)
0.8	0.15	15	1.5
0.5	0.22	22	2.2
0.4	0.28	28	2.8
0.3	0.38	38	3.8
0.2	0.58	58	5.8
0.1	1.15	115	11.5

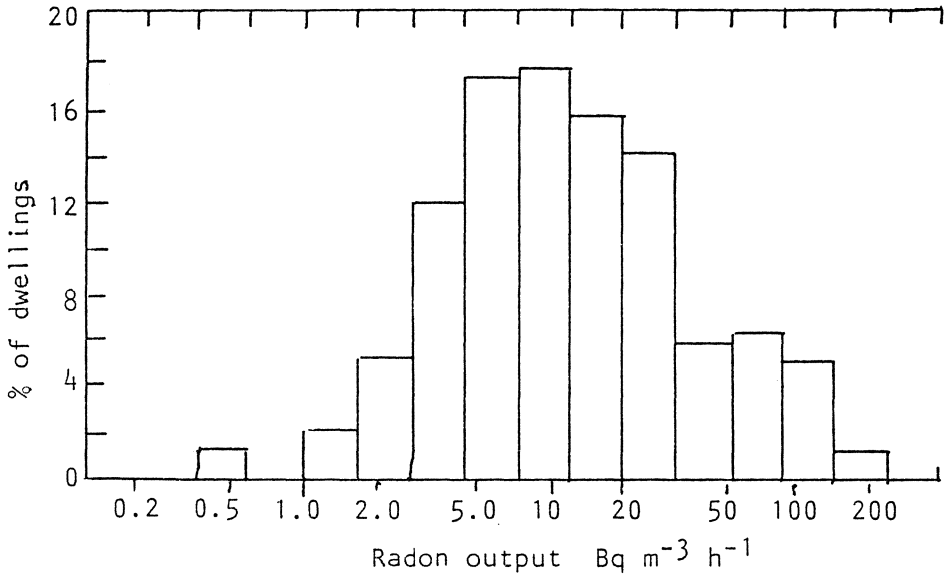

Figure 1 Distribution of ^{222}Rn production rates in living rooms of dwellings in the UK.

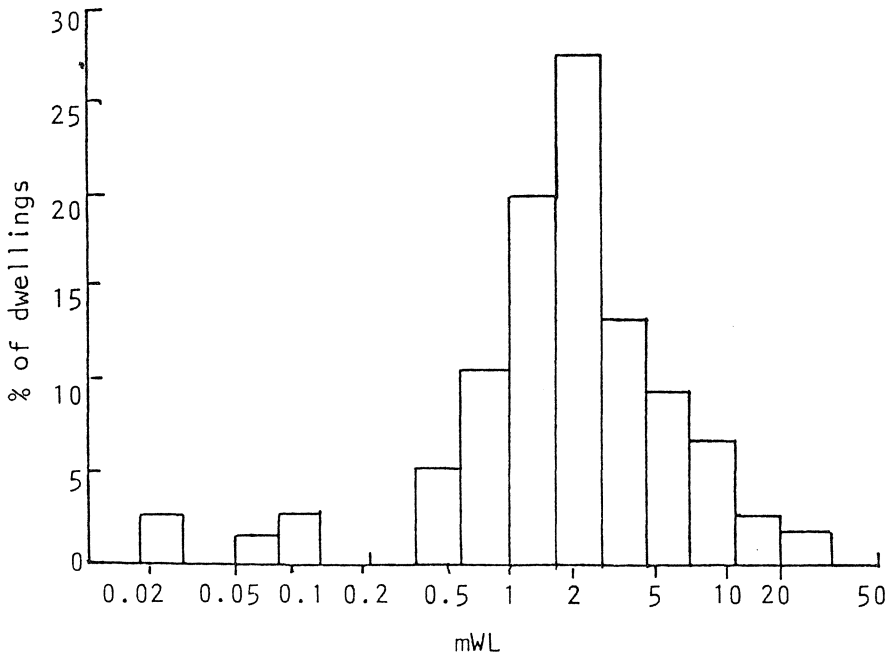

Figure 2 Distribution of WL values recorded in living rooms of dwellings in the UK under normal occupational conditions between 0830 and 1030 hours.

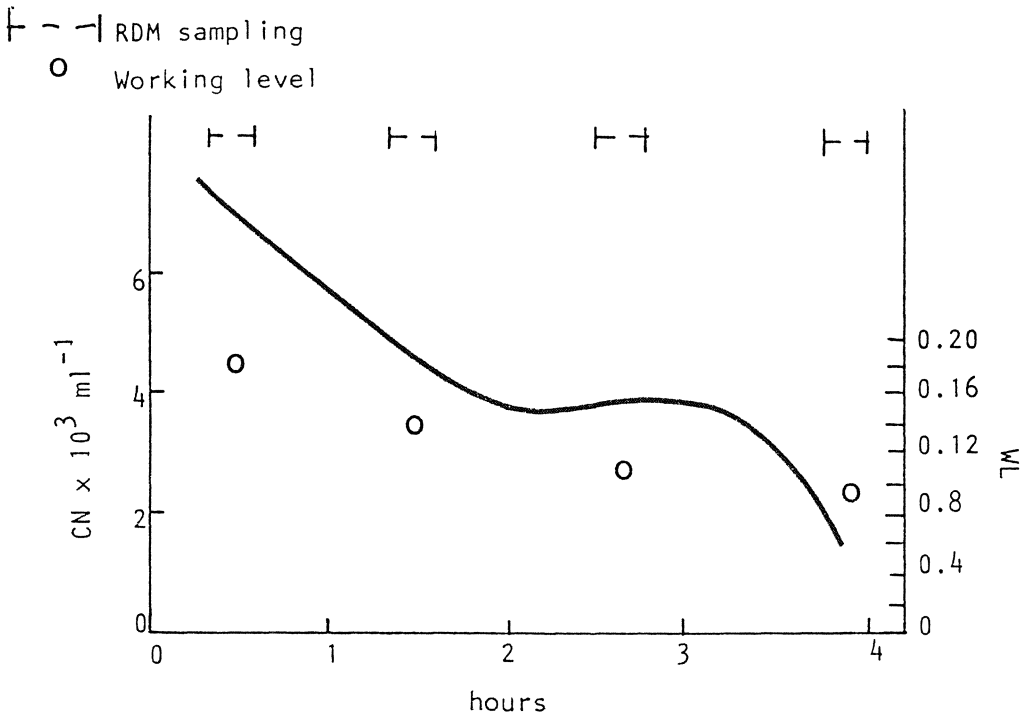

Figure 3 Variation of WL and condensation nucleus concentrations in a sealed room without the operation of the electrostatic precipitator.

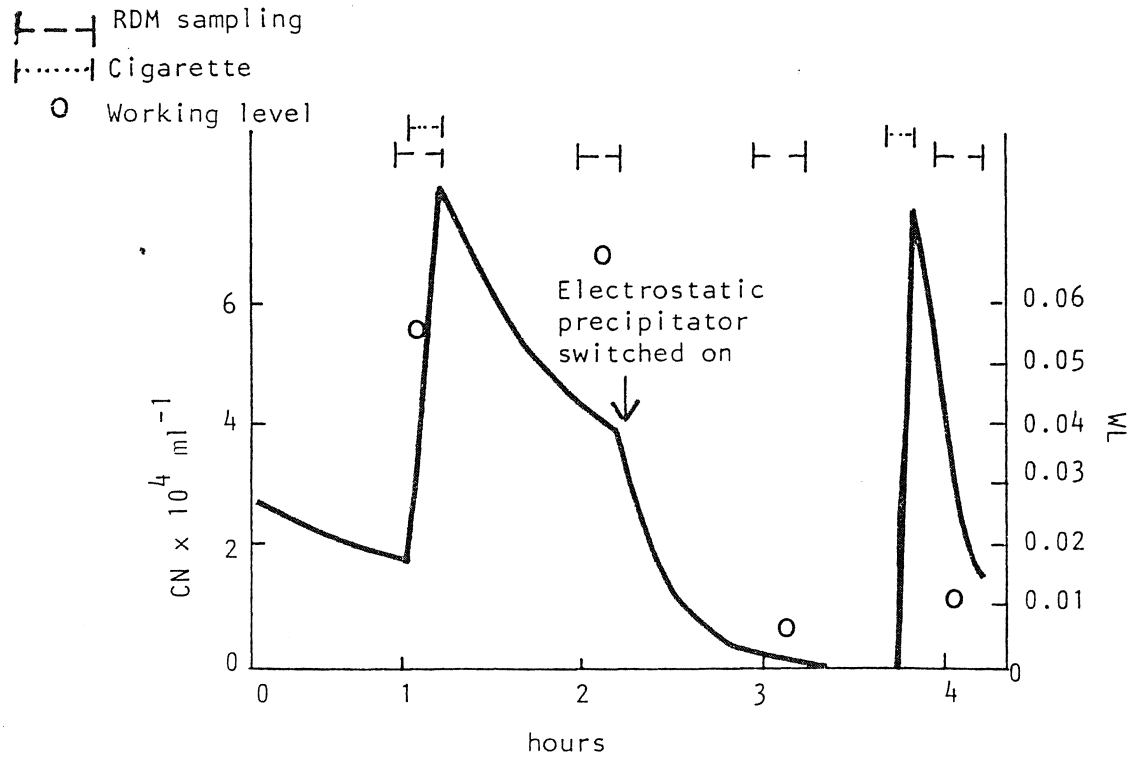

Figure 4 Variation of WL and condensation nucleus concentration in a sealed room demonstrating the effect of enhanced condensation nucleus concentrations by means of cigarette smoke and the effects of the electrostatic precipitator.