Risk factors for atherosclerosis

- Smoking
- Blood pressure
- Diet
- Diabetes
- Blood cholesterol concentration

The risk of heart attack is trebled if the patient smokes, has a serum cholesterol concentration of 6 mmol/l, *and* has a diastolic blood pressure of 85 mm Hg

Treatment of arterial disease

- Operation
- Control of hypertension, blood sugar, and lipids
- Thrombolytic drugs
- **STOP SMOKING**

Smoking

The third important risk factor is smoking—especially cigarettes but probably also pipes and cigars if the smoke is inhaled. Smoking roughly doubles the risk of heart attack and (in hypertensive patients) of stroke, and it massively aggravates the progression of ischaemic complications of peripheral arterial disease. The number of cigarettes smoked seems less important than the mere fact of smoking, and the type of cigarette is largely irrelevant (for the vascular system there is no "safer cigarette"). Unfortunately we still do not know the guilty component(s) of tobacco smoke or their mode of action, but this probably includes thrombogenesis, and stopping smoking brings swift benefit.

Combinations of risk factors

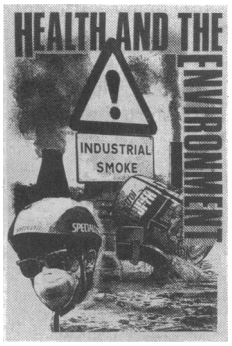
Combinations of risk factors are the main trouble makers. The risk of coronary disease is roughly trebled by a serum cholesterol concentration of 9 mmol/l or by a diastolic blood pressure of 115 mm Hg, and when seen these will not be overlooked because they are most unusual. The risk is also trebled (compared with a low risk subject) by cigarette smoking, plus a cholesterol concentration of 6 mmol/l, plus a diastolic pressure of 85 mm Hg—a combination so common that the doctor may say "My patient has no risk factors." Most vascular disease is caused by such combinations because the arteries stand up reasonably well to one single factor in isolation. The therapeutic corollary of this is that management must also take account of all aspects. Surgery, acute care of patients with myocardial infarction, control of blood sugar concentrations in diabetes, or control of blood pressure in hypertension—each is just one component in the management of a chronic disease with many causes.

The data from the Basle study were presented by Schering. The pictures of self mutilation by smoking and the fatty streaks are reproduced by kind permission of Professor Sir Geoffrey Slaney, KBE, FRCS, and Professor Neville Woolf, PHD, FRCPATH, respectively. We acknowledge with thanks the assistance of the audiovisual department, St Mary's Hospital, London, in the preparation of the illustrations.

Professor Geoffrey Rose was formerly consultant in medicine and epidemiology, St Mary's Hospital, London.

The ABC of Vascular Diseases has been edited by Mr John H N Wolfe, FRCS.

Health and the Environment



Air pollution: II—road traffic and modern industry

Fiona Godlee

Summer smog is a cocktail of volatile hydrocarbons, oxides of nitrogen, sulphur dioxide, and carbon monoxide emitted from road vehicles, industry, and power stations. When acted on by sunlight it produces ozone, which is a potent respiratory irritant. At best photochemical smog is unpleasant, at worst it is harmful to health (table).

Ozone

Ozone at ground level builds up on sunny, still days when temperature inversion—a cold layer of air at ground level covered by a zone of warmer air—prevents the air from circulating. Concentrations reach a peak in the early afternoon and are often highest in rural areas. This is because anticyclones spread photochemical smog from cities, and rural areas lack nitric oxide, a constituent of urban pollution that scavenges ozone to form nitrogen dioxide and oxygen.

At low concentrations ozone causes coughing; nausea; irritation of the eyes, nose, and throat; and headaches. At higher concentrations, 150-200 parts per billion, it damages lung function. Laboratory studies have shown reversible reduction in forced vital capacity, forced expiratory volume, and peak expiratory flow rate in people with asthma and in healthy volunteers.[1] The effects of ozone are worsened by exercise and prolonged exposure, although tolerance seems to occur after a few days. Ozone may also increase the susceptibility of people with asthma to common allergens.[2]

The link between ambient ozone concentrations and impaired lung function has been shown by studies on children in summer camps in the United States. Children had reduced forced vital capacity, forced expiratory volume, and peak expiratory flow rate when ozone concentrations rose during hazy weather. Lung function in children is affected to the same extent as in adults but children develop fewer symptoms and so are less aware of respiratory irritation.[3]

Chronic exposure to ozone may cause structural damage to the lungs. A pathologist in Los Angeles found that 29 out of 107 healthy teenagers who died in road accidents or of other non-respiratory causes had severe respiratory bronchiolitis of the type found in young smokers and in monkeys chronically exposed

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to high concentrations of ozone. (R P Sherwin, V Richters. Centriacinar region disease in the lungs of young adults. Meeting of the AWMA, Los Angeles, March 1990.) A further 51 teenagers had moderate changes.

No legislation controlling ozone concentrations in air exists in the United Kingdom or European Community, and guidelines from the World Health Organisation for peak ozone concentrations leave little or no safety margin. The guidelines are regularly exceeded in Britain during the summer, with ozone reaching concentrations known to cause acute symptoms and long term structural damage.

Asthma and allergy

Between 1971 and 1981 the number of people attending general practitioners' surgeries with asthma and hay fever doubled despite a fall in concentrations of grass pollen in the air. Read has summarised the evidence linking asthma and allergic disease with air pollution and concludes that "pollutants derived from traffic exhaust may exacerbate, and in some cases even initiate, both conditions."[2] But air pollution is not the only possible explanation for the rising toll from asthma; more inclusive diagnostic criteria, inadequate management, and side effects of drug treatments are alternative explanations.

Several constituents of air pollution cause bronchospasm in people with mild asthma and, at higher doses, in non-asthmatic people. Sulphur dioxide, for example, provokes asthma in susceptible people at 200 parts per billion,[4] a concentration which exceeds the one hour average concentration recommended in the WHO guidelines and which is regularly found in Britain. People with asthma also seem particularly susceptible to nitrogen oxides and acid aerosols.[5] In contrast, ozone impairs lung function equally in people with and without asthma, though it also sensitises people with asthma to other pollutants such as sulphur dioxide as well as to common allergens.[6]

Air pollution has increased over the past 40 years and so have admissions to hospital and deaths associated with asthma. Asthma was rare in Britain until after the first world war but the prevalence rose dramatically after the second world war. Rates of hospital admission for children with asthma trebled between 1959 and 1973,[7] and over the past 10 years Australia, France, England and Wales, Canada, and the United States have recorded an increase in deaths from asthma in the 5-34 year age group of 30% to 60%. (M Sears, world conference of lung health, Boston, Masachussetts, 1990.) Whether this is because of an increase in severity or in prevalence of asthma remains debatable.

Epidemiologists studying asthma face several difficulties. Potential confounding factors include idiosyncratic responses to allergens, smoking, and infectious agents and the effects of the weather on both the disease and levels of pollution. Added to these are the difficulty of relating levels of pollutants recorded at monitoring stations with actual levels experienced by individuals and the lack of universal criteria for diagnosing asthma.

But studies of major episodes of air pollution have shown an increase in new cases of asthma and in attack rates among people known to have asthma. Examples are studies of American army staff in Tokyo in the 1940s,[8] and of episodes during the middle of this century in the Meuse Valley, Belgium, Donora in Pennsylvania, and London. Since then several studies have found associations between local levels of pollutants, mainly sulphur dioxide and photochemical smog, and rates of asthma attacks and hospital admissions.[2]

Concentrations of pollutants in the environment rarely reach those required in laboratory studies to induce asthma, but a Spanish study suggests that pollutants act synergistically with allergens so that even low levels can cause bronchospasm.[9] Japanese studies have linked the prevalence of allergic rhinitis with particulate pollution from diesel engines, and in Britain air pollution has been shown to add to the effect of pollen in causing symptoms of hay fever.[10]

Cancer and air pollution

There is continuing controversy over the extent to which urban air pollution causes cancer. "In murder mysteries," says Dr Simon Wolff, a toxicologist at University College, London, "one often finds a corpse but no smoking gun. But when looking for specific evidence of the health impact of transport . . . there are a lot of smoking guns but apparently no corpses." Much research has been done, he says, on the risks of lung cancer from passive smoking and domestic radon, but there are few studies on the relation between traffic pollution and cancer. These studies, he says, show that exhaust fumes represent a risk for many different cancers far outweighing the small effects linked with passive smoking, radon, or diet.

Sir Richard Doll, professor of epidemiology in Oxford, disagrees. "In 1948 when we started working

Effects of pollutants, World Health Organisation standards and when they're exceeded

	Airborne particulates	Sulphur dioxide	Nitrogen oxides	Carbon monoxide	Ozone	Benzene
Source	Diesel exhaust (90% in towns), coal burning	Fossil fuels, power stations (73%), diesel exhaust	Motor vehicles (45%), power stations (35%)	Incomplete combustion fossil fuel, tobacco smoke	Photochemical reaction between nitrogen oxides and hydrocarbons	Emissions and evaporation from petrol engine. Highest at petrol stations and in cars
Health effects	Carry acidic gases and volatile hydrocarbons into lungs. May be carcinogenic	Bronchitis, bronchospasm (especially in asthmatic people)	Respiratory irritation	Reduces oxygen carry capacity of blood. Causes headaches, impairs concentration, exacerbates angina and can precipitate arrhythmias and cardiac arrest. Can retard fetal growth	Coughing; impaired lung function; eye, nose, and throat irritation; headaches. Aggravates asthma and bronchitis	Causes leukaemia
Environmental effects	Soiling of buildings. Reduce visibility. Odour	Main constituent of acid rain. Damages plants and aquatic life	One third of acidity of rainfall	Oxidises to carbon dioxide, contributing to greenhouse effect	Greenhouse gas. Damages crops, trees, plastics, rubber, and paints	
WHO air quality standard:						
1 h average		350 μg/m³	400 μg/m³	30 mg/m³	76-100 ppb (150-200 μg/m³)	No safe level as carcinogenic
8 h average				10 mg/m³	50-60 ppb (100-120 μg/m³)	
24 h average	120 μg/m³	125 μg/m³	150 μg/m³			
1 year average		50 μg/m³				
When exceeded	Regularly	1 h average regularly in London and throughout Britain	Busy roadside locations	8 h guideline exceeded 24 days in winter 1988 at one London site	Several times during summer 1989. One site in Devon reached 135 ppb	

Source: *Air Quality Briefing Sheet.* Friends of the Earth, 1991.

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on trying to explain the increased incidence of cancer, air pollution was the favoured explanation," he said. "Most of us thought the rise in cancer was due to motor traffic. But the more work that has been done, the less connection is found." He acknowledges the small increase in risk of cancer that has been shown in some occupational groups such as engine and truck drivers but has not seen this reflected in population based data. "Its not a very popular thing to say these days, because nobody believes it," he said. "But there is no demonstrable relationship between air pollution and cancer." He believes that this does not rule out the possibility of an environmental risk too small to be detected.

In 1988 a working party for the International Agency for Research on Cancer concluded that diesel engine exhaust is "probably" and petrol engine exhaust "possibly" carcinogenic to humans.[11] This conclusion is based mainly on occupational studies, which use a person's job as a surrogate measure for their individual exposure to pollutants. Jobs producing high exposure to diesel exhaust include work on the railway, in bus garages, and as truck drivers, while petrol exhaust exposure is high among traffic control workers and professional drivers. Some studies have shown no excess risk of cancer, or a non-significant increase in risk, in these occupational groups.[12,13] Other studies have, however, found a small but significant excess risk of cancer, especially of the lung and bladder.

Garshick *et al* examined 19 396 deaths among a cohort of middle aged railway workers,[14] and found more deaths from lung cancer among workers regularly exposed to diesel exhaust than among those with no exposure. The excess risk was small but statistically significant and increased with duration of exposure. In another American study 1256 railroad workers who died of primary lung cancer were matched with two controls by age and date of death.[15] The study found a significantly increased risk of lung cancer (odds ratio 1·4; 95% confidence interval 1·1 to 1·9) in those aged 64 or less who had been exposed to diesel exhaust for 20 years. No such effect was found in the older age group, but many of these had retired before the large scale introduction of diesel engines to the railways.

Jensen *et al* found a significant excess risk for bladder cancer among land transport workers and bus, taxi, and truck drivers in Denmark.[16] The excess risk increased with exposure for all except land transport workers. Steenland *et al* found a significant increase in the incidence of bladder cancer in men from Ohio with more than 20 years' employment as truck drivers (odds ratio 12·0; 95% confidence interval 2·3 to 62·9) and railroad workers (2·2; 1·2 to 4·0).[17]

The children of adults exposed to engine exhaust may have increased risks of cancer. Possible mechanisms are mutation of germ cells, intrauterine exposure, or early postnatal exposure. One study found that children of car mechanics and service station attendants had a non-significant increase in leukaemia, lymphoma, and neurological cancer and a small but significant increase in cancer of the urinary tract (odds ratio 2·9; 1·0 to 8·1). No increase in any forms of cancer was found in children of motor vehicle drivers.[18] In contrast, a Finnish study showed a significant increase in cancer among the children of professional drivers (odds ratio 1·9; 1·1 to 3·7).[19]

There are problems with all of these studies. The most important one is that engine exhaust is ubiquitous in urban areas: there is therefore no such thing as an unexposed control group. There is also the "healthy worker effect"—people in regular employment tend to have lower than average mortality. Both of these problems will tend to cause underestimation of the risks. Factors likely to exaggerate or simply misrepresent the risks are smoking, passive smoking, and exposure to asbestos, and the use of job histories as surrogate measures of exposure.

The difficulty of showing any excess risk in people exposed to high levels of pollution through their work makes it unlikely that studies of the general population exposed to much lower levels of pollution would show any excess risk of cancer. Studies apparently showing this have, however, been published. An American case control study of 328 children with cancer found that children living in areas of high traffic density (500 or more vehicles a day) had an excess risk of developing cancer in general (odds ratio 1·7; 1·0 to 2·8) and leukaemia in particular (2·1; 1·1 to 4·0).[20] The study has, however, been criticised because of inappropriate controls, and is, according to Richard Doll, "uninterpretable." Another study showed an increase in the overall incidence of cancer in a Swiss mountain valley after a motorway was built and correlated the local increase in cancer and levels of polyaromatic hydrocarbons in the soil.[21] It is, however, confounded by the increase in cancer throughout Switzerland.

In conclusion, people heavily exposed to diesel and petrol exhaust are at increased risk of developing cancer, especially of the lung and bladder, but the risks are small. Whether the general population is at risk of cancer from air pollution remains questionable, but any such risk is likely to be extremely small. Whether petrol or diesel is the most environmentally friendly fuel remains open to debate (box).

Diesel or petrol—which is more environmentally friendly?

Diesel engines burn fuel more efficiently than conventional spark ignition petrol engines and emit fewer hydrocarbons and less carbon monoxide. The lower ignition temperatures required also result in fewer oxides of nitrogen. Higher fuel efficiency means diesel engines emit about 18% less carbon dioxide per kilometre.[22] Energy savings also occur at the refinery since diesel requires less processing than petrol.

The main disadvantage of diesel engines is their emission of sulphur dioxide and particulate pollution—the major components of winter pollution. The popularity of diesel vehicles is adding to sulphurous pollution from other sources in the developing world and threatens to reintroduce sooty smogs to urban areas in the West, where diesel fumes are now responsible for 90% of airborne particulate matter.[23] Diesel engines emit about 10 times more particulate pollution than conventional petrol engines and 30-70 times more than petrol engines fitted with catalytic converters.[24] Diesel vehicles are also noisier.

Benzene

One particular component of petrol, benzene, deserves special comment. Benzene is a volatile hydrocarbon also present in cigarette smoke and some domestic solvents and causes leukaemia in man.[25] The main source of benzene in air is emission from petrol engines and evaporative loss during handling, distribution, and storage of petrol. Levels of airborne benzene are closely related to traffic density and range from one to 50 parts per billion. But benzene also builds up inside cars, reaching concentrations close to the limit for occupational exposure in America.[26] The World Health Organisation insists that there is no safe level of benzene because of its known ability to cause cancer.

Extrapolation of the risks of environmental exposure to benzene from data on high dose is speculative, but WHO guidelines estimate that the lifetime risk of leukaemia at benzene concentrations of 0·3 parts per

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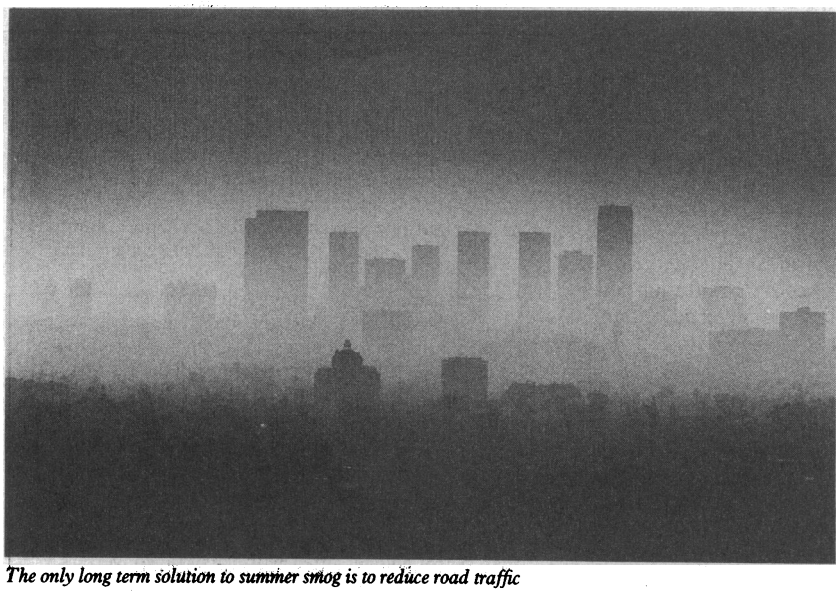


MIKE ABRAHAMS/NETWORK

The only long term solution to summer smog is to reduce road traffic

billion is four cases per million people. The California Air Resource Board has estimated that the added lifetime risk of developing leukaemia from exposure to benzene in Los Angeles is 101 to 780 cases per million people.[27]

Pollution control

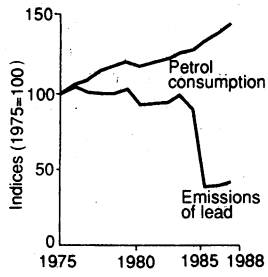
Strategies to reduce photochemical smog must reduce both stationary and mobile sources of pollution. Substantial progress has been made in reducing emissions from power stations in the past 20 years, but many obstacles still remain. Electrostatic precipitators and filters can reduce particulate emissions by 99·5% and are now mandatory in almost all countries in the Organisation for Economic Cooperation and Development. But they have no effect on particulates of sulphates and nitrates formed from gaseous emissions outside the chimney stack. Though not as visible in the air as particles of soot, these cause damage to the lungs.[28]

Emissions of sulphates can be reduced by scrubbers, which reduce emissions of sulphur dioxide from individual power stations by as much as 95%. They are fitted only to newer power stations. Reducing emissions of nitrates is more difficult. Selective catalysts can reduce emissions of oxides of nitrogen by 80-90%, but they are expensive and are in widespread use only in Japan. Elsewhere in the world, various forms of modified combustion are used which reduce emissions by only 30-50%.

In the West new power stations must now conform to strict regulations for pollution control. But controlling emissions from existing plants means fitting the devices retroactively, and only Britain, the Netherlands, Scandinavia, and Germany have undertaken this on any scale.

Like the tall stack policy of the 1950s and 1960s, which cured the winter fogs but caused far flung problems with acid rain, these strategies create problems of their own. For example, desulphurising emissions from power stations produces large amounts of hazardous ash. The strategies also have no effect on emissions of carbon dioxide—the main greenhouse gas.

Emissions of carbon dioxide from domestic and industrial combustion in Britain have now stabilised because of energy efficiency measures, and in the next 10 years further reductions in emissions of nitrogen oxides and sulphur dioxide will be required to conform with European Community directives on large combustion plants. But these improvements will have little impact on overall emissions of pollutants, a growing proportion of which come from road transport.



Consumption of petrol and emissions of lead from road vehicles with petrol engines, United Kingdom[30]

Technical fixes for road traffic emissions

Road traffic emits a fifth of carbon dioxide, a third of airborne particulates and volatile organic compounds, half of oxides of nitrogen, and almost all carbon monoxide in the air. Attempts to reduce this pollution involve various technical fixes such as catalytic converters and filter traps. A European Community directive will enforce the fitting of catalytic converters to all new cars by 1993 and of particulate traps to all new trucks and buses by 1996. Environmental groups are now calling for the compulsory fitting of similar devices to existing vehicles.

Oxidation catalysts convert hydrocarbons and carbon monoxide into carbon dioxide and water. But they have no effect on oxides of nitrogen and as a result have been largely superceded by three way catalytic converters. These reduce emissions of carbon monoxide by 80%, hydrocarbons by 90%, and oxides of nitrogen by 95%.[28]

One of the unforeseen advantages of catalytic converters is that they can work only with lead free fuel. Their widespread introduction in America and Japan, prompted by the need to reduce photochemical smog, has therefore dramatically reduced the amount of lead in air (figure). In America, lead in air fell by 96% between 1970 and 1987, and average blood levels fell by more than one third between 1976 and 1980.[31]

But catalytic converters also have disadvantages,[22] and they have no effect on emissions of carbon dioxide from cars. Because of their adverse effect on fuel economy—reducing it by 1-10%[32]—they may even increase emissions of carbon dioxide. They begin to act only when the engine has warmed up, and most journeys are short and do not allow the engine to warm up sufficiently.

Neither oxidation nor three way catalytic converters are compatible with diesel engines. Pollution from diesel vehicles is being tackled by improved engine design—such as turbo charging which makes use of the exhaust fumes—and particulate traps, which may reduce particulate emissions by up to 85%.

The main source of benzene is evaporative emission, and carbon cannisters to reduce evaporative emissions at refueling sites are being considered by the British government. Vapour retrieval systems are already compulsory in California. Reducing the amount of benzene in petrol would, however, be impractical, according to the Department of Transport and would increase the amount of carbon dioxide produced.

Technical fixes have their limits. They can reduce emissions from each vehicle and so reduce the amount of pollution produced per kilometre travelled. In the short term this will reduce overall emissions, but the effect will soon be swamped by the growth in the number of vehicles. The Department of Transport estimates that by 2025 vehicle miles travelled in the United Kingdom will double. Catalytic converters will cause a fall in emissions of oxides of nitrogen from their current level in Britain of 1·3 million tonnes, but by 2020 emissions will be back up to 1·38 million tonnes and rising.[33]

Conclusion

Photochemical smog is dangerous to health. The contribution from stationary sources of pollution—power stations and industry—is shrinking. Further reductions could result from investing in energy efficiency programmes and transferring to alternative energy sources like wind, wave, and solar power. But attempts to reduce mobile sources of pollution through

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technical fixes will be effective only in the short term. The only effective long term solution to modern air pollution is to reduce road traffic.

I am grateful to Fiona Weir of Friends of the Earth, Dr Simon Wolff, and Dr Peter Burney, for help in preparing this article.

1 World Health Organisation. *Air quality guidelines*. Copenhagen: WHO, 1987.
2 Read C. *Air pollution and child health*. London: Greenpeace, 1991.
3 Avol EL, Linn SW, Shamoo DA, Valeria LM, Anzar WT, Venet TG, *et al*. Respiratory effects of photochemical oxidant air pollution in exercising adolescents. *Am Rev Respir Dis* 1985;**132**:619-22.
4 Linn WS. Respiratory effects of sulphur dioxide in freely breathing, heavily exercising asthmatics: a dose response study. *Am Rev Respir Dis* 1983;**127**:234-9.
5 Mohsenin V. Airway response to nitrogen dioxide in asthmatic subjects. *J Toxicol Environ Health* 1987;**22**:371-80.
6 Koenig JQ. Prior exposure to ozone potentiates subsequent response to sulphur dioxide in adolescent subjects. *Am Rev Respir Dis* 1990;**141**:377-80.
7 Anderson HR. Increase in hospitalisation for childhood asthma. *Arch Dis Child* 1987;**53**:295-300.
8 Huber TE. New environmental respiratory disease (Yokohama asthma). *Archives of Industrial Hygiene and Occupational Medicine* 1954;**10**:399-408.
9 Berciano FA, Dominguez J, Alvarez FV. Influence of air pollution on intrinsic childhood asthma. *Ann Allergy* 1989;**62**:135-41.
10 Gunner G, Riley GA, Fitzharns P, Ashmore MR. Effect of air pollutants on hay fevery symptom severity [abstract]. *J Allerg Clin Immunol* 1991;**87**:185.
11 International Agency for Research on Cancer. *Diesel and gasoline engine exhausts and some nitroarenes*. Lyons: IARC, 1988. (IARC monographs on the evaluation of carcinogenic risk to humans. Vol 46.)
12 Kaplan I. Relationship of noxious gases to carcinoma of the lung in railroad workers. *JAMA* 1959;**171**:97-101.
13 Williams RR, Stegens NL, Goldsmith JR. Associations of cancer site and type with occupation and industry from the third national cancer survey interview. *J Natl Cancer Inst* 1977;**59**:1147-85.
14 Garshick E, Schenker MB, Munoz A, Segal M, Smith TJ, Woskie SR, *et al*. A retrospective cohort study of lung cancer and diesel exhaust exposure in railroad workers. *Am Rev Respir Dis* 1988;**137**:820-5.
15 Garshick E, Schenker MB, Mũnoz A, Segal M, Smith TJ, Woskie SR, *et al*. A case-control study of lung cancer and diesel exhaust exposure in railroad workers. *Am Rev Respir Dis* 1987;**135**:1242-8.
16 Jensen OM, Wahrendorf J, Knudsen JB, Sorensen BL. The Copenhagen case reference study on bladder cancer. Risks among drivers, painters, and certain other occupations. *Scand J Work Environ Health* 1987;**13**:129-43.
17 Steenland K, Burnett C, Osorio AM. A case-control study of bladder cancer using city directories as a source of occupational data. *Am J Epidemiol* 1987;**126**:247-57.
18 Kwa SL, Fine LJ. The association between parental occupation and childhood malignancy. *J Occup Med* 1980;**22**:792-4.
19 Hemminki K, Saloniemi I, Salonen T, Partanen T, Vainio H. Childhood cancer and parental occupation in Finland. *J Epidemiol Community Health* 1981;**35**:11-5.
20 Savitz D, Feingold L. Association of childhood cancer with residential traffic density. *Scand J Work Environ Health* 1989;**15**:360-3.
21 Blumer M, Blumer W, Reich T. Polyaromatic hydrocarbons in soils of a mountain valley: correlation with highway traffic and cancer incidence. *Environmental Science and Technology* 1977;**11**:1082-6.
22 Howard D. *Energy, transport, and the environment*. London: Transnet, 1990.
23 Van den Hout KD, Rukeboer RC. *Diesel exhaust and air pollution*. Delft: Research Institute for Road Vehicles, 1986.
24 Organisation for Economic Cooperation and Development. *Transport and the environment*. Paris: OECD, 1988.
25 International Agency for Research on Cancer. Benzene and annex. In: *Some industrial chemicals and dyestuffs*. Lyons: IARC, 1982. (IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Vol 29.)
26 Gilks JML, Boelsma van Houte E, Eyres AR, Rousseaux G, Roythorne C, Stanton DW, *et al*. *Health risk of exposure to non occupational sources of benzene*. Netherlands: The Hague, 1989. (CONCAWE report No 8/89.)
27 California Air Resource Board. *Report to the scientific review panel on benzene*. Los Angeles: Department of Health Services, 1984.
28 Ostro BD, Rothschild S. Air pollution and acute respiratory morbidity: an observational study of multiple pollutants. *Environ Res* 1989;**50**:238-47.
29 Holman C, Fergusson M, Robertson T. *The route ahead: vehicle pollution—causes, effects, answers?* London: World Wildlife Fund, 1990.
30 Central Statistical Office. Transport and the environment. *Social Trends* 1991;**20**:147.
31 Brown LR. *State of the world 1990*. New York: W W Norton, 1990:103.
32 Organisation for Economic Cooperation and Development. *Energy and cleaner air: costs of reducing emissions*. Paris: OECD, 1987.
33 Fergusson M, Holman C, Barrett M. *Atmospheric emissions from the use of transport in the UK*. Vol 1. *The estimation of current and future emissions*. London: World Wildlife Fund/World Resources Research, 1989.

OBITUARY

N Veall

Obituaries of any doctors will be considered for publication. Obituaries must be submitted exclusively to the BMJ and should be up to about 250 words long; they should be sent within six months, and preferably within three months, of death. "Self written" obituaries are welcome. Good quality, recent photographs are encouraged.

N VEALL

DSc

Norman Veall had an outstanding ability to see through a fog of confusion to the underlying simple explanation. Having left school at 15, he worked as a meteorologist during the war and was recruited to the Cavendish laboratories in Cambridge after hitching a lift from Hans van Halban, a joint discoverer of the atomic chain reaction. Norman arrived on the medical scene from the Atomic Energy Authority at a time of turmoil after the discovery that his tutor for his BSc, Nunn May, had been passing secrets to the Russians. At Hammersmith Hospital he quickly acquired a deep understanding of human physiology and enough understanding of medicine to be able to talk on equal terms with doctors. His first paper was on measuring blood volume with phosphorus-32. Thereafter followed a string of firsts in what is now known as nuclear medicine. These included the first thyroid scan (in the teeth of opposition from his director at the Medical Research Council); the first quantitative radiocardiogram (ventriculography); the invention of techniques for measuring the viability of skin flaps and femoral heads; a series of methods based on the use of edetic acid labelled with chromium-51 for measuring glomerular filtration rate; and the first methods for measuring cerebral blood flow non-invasively with xenon-133.

In all this work Norman formed highly effective collaborations with young and not so young medical colleagues. At the Clinical Research Centre hardly any clinically oriented research group could not claim him as one of its own. Here he invented techniques for measuring sodium fluxes across the gut in cholera, lung and gut permeability, the dynamics of oxalate in hyperoxaluria, bone blood flow, and regional uptake of transmitter analogues in the brain.

Norman Veall was widely known around the world partly because of his book with H Vetter, *Radioisotope Techniques in Clinical Research and Diagnosis*, which was translated into several languages; partly because of his lucid lectures, such as those during courses at Harwell; but principally because he was always infinitely patient in helping the novices achieve a solution to their problems. His contributions to medicine were recognised by one of the first honorary MRCPs awarded to a scientist without a medical qualification and by the Hevesy medal.—J REEVE, T SMITH

Norman Veall, head of the radioisotopes division of the Medical Research Council (MRC) Clinical Research Centre at Northwick Park Hospital until 1984, died 30 August aged 72. Born Welwyn, 24 May 1919; gained BSc in physics and chemistry 1943 (London University). Joined MRC Radiotherapeutics Unit at Hammersmith Hospital 1946. Appointed senior research fellow at Guy's Hospital 1953; while there began working for CRC radioisotopes division in 1967, formally appointed 1969. Awarded honorary MRCP 1984.

R BELLAMY

MB, BCHIR

Dick Bellamy joined the practice in Ashburton, Devon, in 1950 and became senior partner in 1970. In 1978 he was joined by his son, Peter.

"Doctor Dick," as he was always known, had wide interests outside medicine. He had played hockey for Caius College and the army in his younger days. But he was a great sportsman all his life, being a skilful fly fisherman on his beloved river Dart and enjoying riding on the moor, shooting, cricket, and, perhaps above all, golf. He was captain and president of Wrangaton Golf Club and vice captain of Thurlestone Golf Club and was much in demand as an after dinner

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