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List of participants

Dr A. Adelstein	Office of Population Censuses & Surveys, London
Dr Eva Alberman	Paediatric Research Unit, Guy's Hospital, London
Dr E. Ammundsen	National Health Service of Denmark, Copenhagen
Professor Gavin Arneil	Royal Hospital for Sick Children, Yorkhill, Glasgow
Dr Philip Banister	Environmental Health Directorate, Ottawa, Canada
Dr Donald Barltrop	St Mary's Hospital Medical School, Paddington, London
Mr Kenneth Bayes	Design Research Unit, 32 Aybrook Street, London
Dr Alistair Beattie	Dept. of Materia Medica, University of Glasgow
Sir Cyril Clarke	Royal College of Physicians, London
Professor B. Clayton	The Hospital for Sick Children, London
Dr John R. T. Colley	London School of Hygiene & Tropical Medicine, London
Professor S. D. M. Court	British Paediatric Association, London
Sir Richard Doll	Regius Professor of Medicine, University of Oxford
Dr P. C. Elwood	Medical Research Centre, Epidemiology Unit, Cardiff
Dr D. B. Gammack	Unigate Chief Scientist, Central Laboratory, London.
Dr Roger Glass	Dept. of Regius Professor of Medicine, University of Oxford
Sir George Godber	Unattached
Professor R. J. Haggerty	University of Rochester School of Medicine, Rochester, New York
Dr Clark W. Heath	Dept. of Health, Education and Welfare, Atlanta, Georgia
Mr R. A. Hendey	Unigate Foods Limited, Guidford, Surrey
Sir James Howie	Consultant Bacteriologist to Unigate
Professor D. Hull	Dept. of Child Health, City Hospital, Nottingham
Professor P. J. Lawther	Medical Unit, St Bartholomew's Hospital, London
Dr K. Newell	World Health Organisation, Geneva
Professor T. E. Oppé	St Mary's Hospital Medical School, Paddington, London
Dr A. Parry Jones	County Medical Officer of Health, Somerset
Professor J. Pepys	Cardio-Thoracic Unit, Brompton Hospital, London
Professor Michael Rutter	Dept. of Child Psychiatry, Institute of Psychiatry, London
Mr J. A. Silk	Unigate Foods Limited, Guildford, Surrey
Dr Esther Simpson	Dept. of Health & Social Security, London

Dr Stig Sjölin	Dept. of Paediatrics, University Hospital, Uppsala
Professor R. W. Smithells	Dept. of Paediatrics & Child Health, University of Leeds
Professor Eric Stroud	King's College Hospital Medical School, Denmark Hill, London
Professor Jack Tizard	Thomas Coram Research Unit, University of London
Professor H. K. A. Visser	Sophia Children's Hospital and Neonatal Unit, Rotterdam
Dr J. A. C. Weatherall	Office of Population Censuses & Surveys, London

Editorial

Increasingly, the term 'environment' has become associated with the problem of pollution, and this has tended to obscure its correct usage. In a biological sense, environment has been defined as 'the sum of external influences acting upon the organism', and it was this broader concept in relation to human development that stimulated the meeting reported in this volume.

Perhaps the greatest value of the conference lay in the confrontation of disparate viewpoints and disciplines and the mutual recognition of the complexity of the factors which determine the optimal development of children. Thus it is noteworthy that the psychosocial aspects of the child's environment attracted as much attention as the chemical and physical hazards more commonly recognized in this context.

Unlike clinical problems affecting individual children, environmental factors may involve whole communities so that their perception and control require a collaborative approach which, in the past, has not always been achieved. It is hoped that this record of the proceedings has captured some of the flavour of a truly multi-disciplinary meeting and that it will provoke discussion among its readers.

My thanks are due to Mrs D. Challis and Miss Susan Madge for devoted secretarial assistance. Mrs A. Moorhouse and her staff gave invaluable help in the transcript of the tapes and the preparation of the manuscript.

Paediatric Unit,
St Mary's Hospital Medical School,
London, W2 1PG.

DONALD BARLTROP.

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This Workshop, sponsored by Unigate Foods Ltd, London, is the second of a series designed to encourage specialists in different fields pertaining to paediatrics and to stimulate further research in the subject.

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SECTION Ia

Introduction

SIR GEORGE GODBER

It is my privilege to welcome you all here for two days that I am sure will be a valuable experience for each of us. It will lead to a publication of interest and value to a great many people who are going to be able to share that experience vicariously in our report. This is the second workshop sponsored by Unigate and I hope you have all seen, if you have not all read, the admirable publication which followed the first workshop on Clinical Immunology in Paediatric Medicine a year ago. This is an enlightened initiative by Unigate which we in Britain very much welcome. We in medicine are dependent on Unigate for many things but they are also dependent on us. As a former medical bureaucrat I very much welcome this recognition of the interdependence of interests with those of this particular aspect of the health service.

My occupancy of this chair is the outcome of my long association with Professor Oppé, first as Secretary of the British Paediatric Association and then as Consultant Adviser to the Department of Health on Paediatrics.

I should like to offer a special welcome to those participants from Sweden, Denmark, The Netherlands, Geneva, Canada, and the United States. It is very good of them to make the long journeys that they have accepted and to prepare the contributions to be made.

Although the title we have chosen suggests the specialty of Paediatrics, we are really going to discuss the effect of the environment on children. Except for the problem of hazards from oral contraceptives I doubt if there is a medical question on which more nonsense appears in print than the hazards of the environment. After all, no child can do without an environment and at least the major hazards from a lot of bacteria and viruses in that environment have been greatly reduced if not eliminated.

If this meeting had been taking place 40 years ago there would have been, even in the summer months more children in hospital suffering from communicable diseases than there are for all conditions today. In 1933 about 840 children out of every million living under the age of 15 in Britain died from the five commonest communicable diseases of childhood. In 1973 the rate will have been about 1/200th of that.

Although the environment has been cleaned up a great deal and its microbiological hazards enormously reduced, many respiratory virus infections and more insidious hazards to health of an inanimate kind remain. The gross pollution of the atmosphere has been much reduced at least as far as suspended solids are concerned, but babies are more commonly exposed to the atmospheric pollution provided by their own mothers' cigarette smoke.

There are many other physical and chemical hazards that are certainly more common, even if this is a result of other triumphs of medicine which bring more drugs into the child's environment because the adults who share it with him are using them. Whether the psycho-social hazards are greater or just better recognized may emerge from the next session. We are seeking a better understanding of the effects in the very long term of relatively small contaminations or malfunctions.

The major contamination that can occur from the ingestion of lead-based paint leads to clinically recognized poisoning, and something can be done about it, though seldom fast enough. The small increase in lead intake that can give a child a blood lead level as much as twice what might be called the baseline, may even be associated with demonstrable

change in enzyme activity but not with any clinical evidence of harm. That can send some moderately well-informed journalist off on a witch hunt which can cause great alarm and cannot yet be answered with unequivocal re-assurance.

The experts here are going to talk about this and I will not trample over their territory further. I am simply making the point that we can do more here by trying to probe into the longer term effects of environmental factors which may do harm from chronic exposure, perhaps to a minority of those exposed, than by talking about gross exposure and frank poisoning. There was a very sad example as a result of a manufacturing accident which led to manifest poisoning from hexachlorophene, but the longer term possibilities of much lower level exposure through the regular use of hexachlorophene in baby toilet preparations by large numbers of hospitals and families may have much more significance for us. I suggest that we are looking for many small things rather than for guides to a few large ones.

Physical hazards

P. J. LAWTHER *and* R. E. WALLER

*MRC Air Pollution Unit, St Bartholomew's Hospital, London, E.C.*1

The most dramatic effects of air pollution on health have been seen in terms of sudden increases in deaths during major 'fog' episodes. In the London fog of December 1952 it was estimated that the number of deaths in the Greater London area during and just after the fog was about 4000 more than would otherwise have been expected (Ministry of Health, 1954). The main impact was on the elderly chronic sick, but an analysis by age at death (Table 1, available for the inner area of London only) showed an appreciable increase in deaths, on a proportionate basis, for children under one as well as for adults over the age of 45. In absolute terms, however, the number of infant deaths in the week of the fog was only 26 more than in the previous week, and children in the age range 1 to 14 appeared to escape lethal effects almost completely.

Details of the age distribution of deaths in other London 'fogs' in the 1950's and early 60's are not available, but in a study of general practitioner consultations during the winter of 1962–63 (Carne, 1964) there was a small increase in onsets of respiratory disease at the time of the major 'fog' in December 1962 among the under fives, but again the main impact was on adults over the age of 45.

Although effects of exposure to major episodes of high pollution are less evident among children than among the elderly, there is by now much evidence of an association between the general prevalence of respiratory illnesses in children and the amounts of pollution in the areas where they live. Some years ago the opportunity arose to incorporate studies on air pollution and respiratory illness into the National Survey of Health and Development (Douglas and Waller, 1966). In this, a sample of children all born in a single week in 1946 had been followed from birth, and the average exposure to pollution of each child, up to the age of 15, was assessed in terms of the coal consumption in the areas where he (or she) had lived. The results of this study were remarkably consistent: children who had lived in areas having 'very low' pollution had the lowest incidence of chest illnesses, with the frequency increasing through 'low' and 'moderate' to a maximum in 'high' pollution areas. Many health indices were examined, covering the whole period from birth to age 15, and examples of results are shown in Table 2.

In contrast, there was no significant relationship between indices of upper respiratory tract illnesses and air pollution levels. Other factors associated with area of residence could have influenced the results, but the association of lower respiratory tract illnesses with air pollution persisted even when the social class of the family was taken into account.

Lunn *et al.* (1967, 1970) were able to find a similar association between respiratory illnesses and exposure

TABLE 1. Deaths registered in London Administrative County classified by age. December, 1952—the week of the fog, compared with the previous week

	Age (years)					
Week ending	<1	1–14	15–44	45–64	65–74	75+
6th December	28	10	61	237	254	355
13th December	54	13	99	652	717	949
Ratio of 13th/6th	1·9	1·3	1·6	2·8	2·8	2·7

TABLE 2. 1946 cohort. Percent of subjects reporting lower respiratory tract illnesses (bronchitis, pneumonia or broncho-pneumonia), by air pollution category (Douglas & Waller, 1966)

	Air pollution category			
Illnesses reported	A Very low	B Low	C Moderate	D High
First attack in first 9 mths	7·2	11·4	16·5	17·1
One or more attacks in first 2 yrs	19·4	24·2	30·0	34·1
Two or more attacks in first 2 yrs	4·3	7·9	11·2	12·9
Hospital admission in first 5 yrs	1·1	2·3	2·6	3·1

to pollution among school-children in Sheffield, and the contrasts found in their original study in 1963–65 had diminished by the time of their follow-up in 1967–69, in parallel with improvements in air pollution conditions. Colley and Reid (1970) have also studied respiratory disease among school-children, in towns with relatively high levels of pollution (Newcastle and Bolton), others with lower levels (Bristol and Reading) and in rural areas. They found a marked difference in the prevalence of chronic cough between social classes, but there was also an association with pollution, particularly within Social Classes IV and V (Table 3). Similar results were obtained for histories of bronchitis.

TABLE 3. Morbidity ratios (SMR %) for chronic cough among primary schoolchildren, classified by Social Class and area of residence (Colley and Reid, 1970)

Social Class	I and II	III	IV and V
Newcastle and Bolton	56	105	149
Bristol and Reading	69	104	126
English rural	48	89	96

A further large study among school-children was undertaken by Holland *et al.* (1969) in four areas of Kent. One of the advantages in confining attention to children when examining relationships between the prevalence of respiratory disease and air pollution is that the effects of other factors, such as smoking and occupational exposures to dusts, can be avoided to a certain extent. Even so, in the study in Kent, which included children up to the age of 16, smoking was found to be a relevant factor in the prevalence of respiratory symptoms. Histories of smoking were not long enough however to affect objective measurements of lung function, and it was found that peak flow values were more closely associated with area of residence than with other factors, being lowest in the area with highest pollution levels.

The general conclusion reached from all these studies on children was that environmental factors, and particularly the exposure to pollutants such as smoke and sulphur dioxide, had an important bearing on the incidence of chest illnesses from a few months of age onwards, and on the gradual development of respiratory symptoms. We decided, therefore, to make a special study of children exposed to an exceptionally high level of pollution in infancy—those born in London just before the fog of December 1952. In 1970 it was possible to take advantage of the electoral registers, which then included 18-year-olds for the first time, to trace people born in 1952. Three small areas of London were selected, and within these all the residents reaching age 18 in 1970 were contacted, and visited in their own homes. Approximately 800 subjects were seen, and then a further sample was taken in the same areas, from the 1971 registers, so that all subjects were very close to age 18 when seen, and the second group differed only in having missed the 1952 fog.

A modified form of the MRC Questionnaire on Respiratory Symptoms (1966) was used in this study and measurements of forced expiratory volume in 1 second (FEV_1) forced vital capacity (FVC) and peak expiratory flow (PEF) were made. Over 40 questions on respiratory symptoms, previous illnesses, smoking habits and environmental conditions were asked of each subject (with help from parents when necessary), and results for some of the main questions, and for the ventilatory capacity measurements are shown in Table 4.

There were no differences in these results between subjects born before the fog and those born after it (taking the day on which the fog cleared as the dividing line), and for none of the questions asked was there any difference between the two groups that was statistically significant and consistent between the two sexes. There were differences, however, with respect to other factors that had been considered.

TABLE 4. 1952–53 cohort. Prevalence of respiratory symptoms and ventilatory capacity among subjects born before and after the 1952 fog

	Males		Females	
	Pre-fog	Post-fog	Pre-fog	Post-fog
Symptom, % positive				
Persistent cough and phlegm	4·5	4·7	2·7	2·9
Breathlessness	11·7	13·1	26·8	27·1
Chest illnesses (past 3 yrs)	12·7	10·0	12·8	14·5
Ventilatory capacity, mean				
FEV_1 litres	4·34	4·39	3·20	3·26
FVC litres	5·16	5·12	3·71	3·76
PEF l/min	549	546	431	425
No. of subjects	291	428	298	339

TABLE 5. 1952–53 cohort. Prevalence of respiratory symptoms and ventilatory capacity among smokers and non-smokers

	Males			Females		
	Non-smokers		Smokers	Non-smokers		Smokers
Symptom, % positive						
Persistent cough and phlegm	3·8		6·6	0·6	*	7·5
Breathlessness	7·9	*	16·3	18·7	*	45·1
Chest illnesses (past 3 yrs)	10·2		12·9	8·7	*	27·6
Ventilatory capacity, mean						
FEV_1 litres	4·42	*	4·32	3·26	*	3·18
FVC litres	5·15		5·12	3·74		3·74
PEF l/min	550		545	432		422
No. of subjects	453		380	497		255

* Significant difference ($P < 0.05$).

TABLE 6. 1952–53 cohort. Prevalence of respiratory symptoms and ventilatory capacity in relation to history of respiratory illnesses

	Males			Females		
	Pneumonia, pleurisy		No illnesses	Pneumonia, pleurisy		No illnesses
Symptom, % positive						
Persistent cough and phlegm	7·4		3·6	5·7		2·3
Breathlessness	14·7		9·5	37·7	*	23·0
Chest illnesses (past 3 yrs)	23·5	*	5·4	24·5	*	7·7
Ventilatory capacity, mean						
FEV_1 litres	4·21	*	4·42	3·17		3·25
FVC litres	5·05		5·14	3·74		3·74
PEF l/min	525	*	553	417		430
No. of subjects	68		608	53		570

* Significant difference ($P < 0.05$).

Smoking was associated with a substantial increase in the prevalence of respiratory symptoms, and a slight reduction in some of the ventilatory capacity measurements (Table 5), even though smoking histories were short.

The main factor associated with reduced ventilatory capacity was a history of respiratory illness in childhood. Results for the small proportion of subjects who said that they had had pneumonia or pleurisy are shown in Table 6. These probably underestimate the true differences however, since, as Lunn *et al.* (1970) found in their study in Sheffield, there is

a tendency for the illnesses of early childhood to be forgotten as time goes on. Reduced ventilatory capacity was also found among subjects who reported histories of bronchitis or asthma.

None of the other environmental factors considered had any substantial and consistent effect on the prevalence of respiratory symptoms, nor on ventilatory capacity, although there was a slight tendency for results to be poorer for subjects who had lived as infants in old, damp houses, and parental smoking habits appeared to be of some relevance, but this was difficult to separate from the effects of the subjects' own smoking habits.

Whilst the results showed clearly that exposure to the London fog of December 1952 when only a few months old had no dominant effect on the development of respiratory symptoms, the whole of this '1952–53 cohort' was exposed during childhood to the high levels of pollution that prevailed in London through the 1950's and into the 1960's. The prevalence of respiratory symptoms was higher than expected from some other small-scale studies of adolescents, and further studies are planned to compare the results with those for later cohorts of Londoners, who will have lived through less polluted conditions and for cohorts born and brought up in rural surroundings.

The importance of respiratory illnesses in childhood in relation to the subsequent development of respiratory symptoms, which was apparent in this study, has also been shown in a further follow-up of the 1946 cohort. Colley, Douglas and Reid (1973) have found that although the prevalence of respiratory symptoms at age 20 was mainly affected by smoking, the past history of respiratory illnesses, which had been fully documented in this group, was also of some importance. Exposure to current levels of pollution appeared to have little effect, but in view of the earlier results in this cohort, the main role of pollution may be in enhancing the incidence of acute lower respiratory tract infections at an early age, with this in turn contributing to the gradual development of chronic respiratory symptoms. A possible mechanism for the enhancement of acute infections is suggested by the results of some laboratory experiments, in which it has been found that particulate air pollutants are capable of stimulating the growth of *Haemophilus influenzae* (Lawther *et al.*, 1969).

Some further indication of the susceptibility of children at various ages to the effects of environmental influences can be extracted from mortality statistics. Seasonal variations in deaths, involving the effects of temperature extremes and of the greater prevalence of respiratory infections in the winter months as well as any effects of pollution, are greater among the very old and the very young than among school-age children or young adults, and the 'urban excess' in mortality from respiratory diseases is also most marked in the very old and the very young. When planning the 1952–53 cohort study reported above, the contrasts in death rates from respiratory diseases between London and the surrounding areas of S.E. England were examined, and found to be greatest in the age range 3–5 months (Table 7).

TABLE 7. Bronchitis + pneumonia death-rates for children, classified by age. Five-year periods, Greater London and 'remainder' of S.E. Region

	1951–55		1966–70	
Age	Greater London	Rem. S.E.	Greater London	Rem. S.E.
0–2 mths	200	188	172	118
3–5 mths	93	68	98	63
6–8 mths	35	40	34	27
9–11 mths	20	23	18	14
1–4 yrs	18	20	10	9
5–14 yrs	3	3	2	2

All rates quoted on an annual basis, deaths per 100,000 live births at ages under 1, and per 100,000 living otherwise. Based on data from the Registrar General's Statistical Reviews, Tables 19 and 28 (26 from 1968, and with a change in cause of death classification then, the absolute values for the second period are not strictly comparable with those for the first).

This lent some support to the findings from the 1946 cohort study that differences in the incidence of chest illnesses, apparently related to environmental factors, were already evident by the age of 9 months, and it was considered that the greatest chance of finding any long-term effects of the 1952 fog might be by examining people who were only a few months old at the time. This may remain true, but it is interesting to note, in the second part of Table 7, that despite major reductions in pollution during the past 15 years, there is still a London 'excess' of respiratory deaths in the age-range 3–5 months, and this extends to ages under 3 months also now. The interpretation of these figures is no doubt complicated by the extensive movement of population in and out of Greater London during the past 15 years, but they may indicate a continuing influence of urban environmental factors on the occurrence of respiratory disease among the very young. It may be that more attention should be given now to the 'micro-environment' of the child in the home. Formerly, when concentrations of smoke and sulphur dioxide in the outdoor air were high in London, and open coal fires induced such large draughts that indoor conditions often differed little from those outdoors, the general concentration of these pollutants in an area provided an adequate guide to the exposure of any members of the population. Now, with lower

outdoor concentrations and reduced ventilation rates in homes, exposures may need to be assessed on a more individual basis. In general, concentrations of of smoke and sulphur dioxide should be relatively low indoors, but in some circumstances other pollutants from inadequately flued heating or cooking appliances, or from cigarette smoking, could be of some importance. An extreme example of the ill-effects of indoor pollutants has been reported by Sofoluwe (1968), who found very high concentrations of carbon monoxide, oxides of nitrogen and other pollutants from cooking sources in primitive dwellings in Nigeria where cases of broncho-pneumonia in infants had occurred. Whilst problems of this magnitude are outside the range of anything likely to be found in urban areas of this country, they indicate very forcibly the vulnerability of the young child, who cannot escape from his immediate environment, and suggest that even in our own conditions still more attention should be given to the exposure of infants to locally high concentrations of a variety of pollutants.

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