LEAD IN MAN AND THE ENVIRONMENT

LEAD IN MAN AND THE ENVIRONMENT

J. M. RATCLIFFE, Visiting Scientist, National Institute for Occupational Safety and Health, Cincinnati, Ohio

This book is unique in its provision of an integrated up-to-date review of the health effects, environmental pathways and control strategies for lead, both in man and his environment. We know of no other text providing such a broad and comprehensive overview at this level, that is also unbiased, detailed, accurate and readable.

The book offers discussion of the available literature, with extensive references and, unlike existing works, is a critical and independent review. It informs on government policy, legislative measures, and the toxicological bases for current biological standards. Here is a • bringing-together of the various and topical problems of lead to illustrate the principles, the measurements and the evaluation of environmental hazards, the derivation of biological standards and the ways in which sources of lead intake and acceptable limits of exposure are integrated into control strategies.

Features include: full discussion of the available epidemiological data on the effects of lead on intellectual functions and behaviour in children; differing viewpoints and their significance; discussion of control strategies, e.g. air quality standards as promulgated in the U.S.A. and proposed by the E.E.C.; technological and economic feasibility of the removal of lead from petroleum; the relationship between airborne Pb and blood Pb; current biological guidelines in relation to population blood lead distributions and implications for control; airborne lead and the controversy of dustborne lead. The use of animal models to elucidate mechanisms of neurotoxicity is discussed, a variety of tables help annotate the text.

Readership: For all concerned with the toxicology, epidemiology and control of lead, e.g. those in industrial and research laboratories, public authorities concerned with environmental health and pollution control; those in community medicine, occupational health and safety, public health engineering, as well as conservationists and environmentalists. And for graduate courses in all the above studies, polytechnic courses for environmental health officers, and for those studying medicine, the biological sciences, and in particular toxicology, epidemology and environmental sciences.

LEAD IN MAN AND THE ENVIRONMENT

J. M. RATCLIFFE, B.Sc., M.Sc., Ph.D.
Laboratory of Toxicology
University College Medical School
London

ELLIS HORWOOD LIMITED
Publishers · Chichester

Halsted Press: a division of
JOHN WILEY & SONS
New York · Chichester · Brisbane · Toronto

First published in 1981 by

ELLIS HORWOOD LIMITED
Market Cross House, Cooper Street, Chichester, West Sussex, PO19 1EB, England

The publisher's colophon is reproduced from James Gillison's drawing of the ancient Market Cross, Chichester.

Distributors:

Australia, New Zealand, South-east Asia:
Jacaranda-Wiley Ltd., Jacaranda Press,
JOHN WILEY & SONS INC.,
G.P.O. Box 859, Brisbane, Queensland 40001, Australia

Canada:
JOHN WILEY & SONS CANADA LIMITED
22 Worcester Road, Rexdale, Ontario, Canada.

Europe, Africa:
JOHN WILEY & SONS LIMITED
Baffins Lane, Chichester, West Sussex, England.

North and South America and the rest of the world:
Halsted Press: a division of
JOHN WILEY & SONS
605 Third Avenue, New York, N.Y. 10016, U.S.A.

British Library Cataloguing in Publication Data
Ratcliffe, J. M.
Lead in man and the environment. –
(Ellis Horwood series in environmental science)
1. Lead – Environmental aspects
2. Lead-poisoning
I. Title
363.7'38 RA1231.L4

ISBN 0-85312-257-1 (Ellis Horwood Ltd., Publishers)
ISBN 0-470-27184-1 (Halsted Press)

Typeset in Press Roman by Ellis Horwood Ltd.
Printed in the U.S.A. by Eastern Graphics Inc., Old Saybrook, Connecticut

Table of Contents

Part II: SOURCES OF LEAD IN MAN AND THEIR CONTROL

Acknowledgements

I would like to thank the following authors and holders of copyright for permission to reproduce previously published figures: Dr. A. C. Chamberlain and the U.K. Atomic Energy Research Establishment Harwell (Figs. 1.2, 1.3 and 6.1): Professor H. A. Roels of the University of Brussels, Louvain, and Heldref Publications, Washington, D.C. (Figs. 2.2, 2.4, 2.5, 2.6); Drs. S. Selander and K. Cramer and the Editor of the British Medical Journal (Fig. 2.3); The Royal Society of Health, London (Fig. 5.2); The Controller of Her Majesty's Stationery Office (Fig. 5.3); Professor E. R. Reiter of Colorado State University (Fig. 5.4); Dr. A. C. Turner of Warren Spring Laboratory, Stevenage, U.K. (Fig. 5.7) and Pergamon Press Ltd. Oxfordshire, U.K. (Fig. 5.8). I am most grateful to Dr. R. A. Goyer of the National Institute of Environmental Health Sciences, U.S. for the photographs reproduced in Plates I and II, and to Professor P. J. Lawther of the M.R.C. Toxicology Unit Clinical Section, London, for the photographs reproduced in Plates III–VI.

The generous assistance of the Royal Society and the Sir Samuel Scott of Yews Trust in financing a study visit to government and research institutions in the U.S.A. is gratefully acknowledged; thanks are also extended to the many people both there and in the U.K. who gave information and with whom many stimulating discussions took place.

Finally, I would like to thank Professor R. S. Scorer of Imperial College, London and Dr. A. E. M. McLean of University College Medical School, London for their help, advice and encouragement in the preparation of this volume. And not least, my parents and friends for non-professional but vital support throughout. To them this book is dedicated.

Abbreviations and Symbols

ALA	δ-aminolaevulinic acid
ALAD	δ-aminolaevulinic acid dehydratase
ALAS	δ-aminolaevulinic acid synthetase
(Ca)EDTA)	calcium disodium ethylenediamine tetra-acetic acid.
CEC	Commission of the European Communities
DoE	Department of the Environment (U.K.)
DHSS	Department of Health and Social Security (U.K.)
DMED	diffusional mean equivalent diameter
DoT	Department of Transport (U.K.)
EPA	Environmental Protection Agency (U.S.)
(F.)E.P.	(free) erythrocyte protoporphyrin
FAO	Food and Agriculture Organisation (United Nations)
FDA	Food and Drugs Administration (U.S.)
ICRP	International Commission on Radiological Protection
kg	kilogram
MMED	mass median equivalent diameter
NAS	National Academy of Sciences (U.S.)
Pb[B/U/A/W]	lead in blood, urine, air, water.
μg/g*	microgram/gram (=p.p.m.)
$\mu g/m^3$	microgram/cubic metre
μg/l	microgram/litre
μg/dl*	microgram/decilitre
μmol/l*	micromol/litre
μmol/g*	micromol/gram
WHO	World Health Organisation
ZPP	zinc protoporphyrin

*Blood lead values are commonly expressed in units of μg/dl (sometimes written as μg/100 ml) which are taken to be equivalent to μg/100 g. These units are used in the text for clarity and familiarity.
For conversion to newer SI units: 1 μmol/l = 20.7 μg/dl.
Similarly, 1 μmol/g = 207 μg/g (p.p.m.)

Introduction

Lead is the most abundant of the heavy metals in the earth's crust, occurring principally as the sulphide ore, galena. There is a certain natural background concentration of lead in the physical and biological environment due to its mobilization and dissemination from these deposits. There have been various attempts to estimate these concentrations, by both theoretical extrapolation and the measurement of lead in remote parts of the earth and in the remains of ancient man. Patterson (1965) for example, has estimated the natural lead concentration in air to be some 3-5 orders of magnitude less than current levels, at about 0.0005 $\mu g/m^3$, and in water, 2-3 orders of magnitude less, at about 0.5 $\mu g/l$. Grandjean *et al.* (1979) measured the lead in the teeth and bones of ancient Nubians buried in the Sudanese desert, with some samples originating from the period 3300-2800 BC, and found concentrations of approximately 0.6 $\mu g/g$, some 10-100 times less than in some modern specimens. While it is still possible to measure environmental levels as low as those attributable to 'natural' contamination in certain remote areas today (Chow *et al.* 1972), it is clear that man-made mobilization has increasingly contaminated all phases of the physical and biological environment. The bulk of this arises from the use of lead primarily in the northern hemisphere (Murozumi *et al.* 1969), and now runs at more than 4.5 million metric tons worldwide.

The useful physical properties of elemental lead, those of comparative ease of extraction from the ore, malleability, ductility, corrosion resistance and poor conductance, have resulted in a multiplicity of uses of the metal for over 3000 years. The ancient civilizations of Phoenicia, Egypt, Greece, India and China are known to have smelted and used lead for vessels, roofs, water ducts, utensils, ornaments and weights, and the Romans used lead extensively in the transport of water and the storage of wine and food. The coloured oxides were also used as pigments in cosmetics and glazes. Until the Industrial Revolution, these uses were mainly confined to products such as sheeting, tubing, vessels, glazes, pigments, and alloys. The advent of mass industrialization and, in particular, the motor vehicle, brought about two new and dramatic increases in lead usage: as a component of the lead-acid storage battery and, from about 1923, as the organic lead alkyl 'anti-knock' additive in petroleum.

The organic lead alkyls, such as tetraethyl and tetramethyl lead, are insoluble in water but soluble in organic solvents such as petrol. When used as an additive in petrol, the effect is to increase the resistance to knocking, that is, auto-ignition of the remaining unburnt hydrocarbon mixture in the combustion chamber, which can damage the engine. The octane rating is a measure of the 'anti-knock' resistance of the petrol; adding lead effectively increases this rating, otherwise achievable by greater refining. During combustion, a high proportion of the alkyl is 'scavenged' by halogenated hydrocarbons also present in the petrol, and lead is emitted in the exhaust as complex inorganic halides either in vapour or particulate form. The proportion of unchanged alkyl lead emitted has been considered to be minimal, although quantitative measurements of organolead compounds have been hampered till recently by a lack of suitable monitoring devices. Work by Harrison & Laxen (1977) suggests that this component may be about 1-10% of the total lead concentration in air.

The toxicological properties of organic lead are substantially different from, and greater than, those of the inorganic form. While the exposure to organolead ments serious concern in an occupational setting, little is known at present of its possible significance at environmental levels. In the text that follows, therefore, 'lead' refers solely to the element or its inorganic compounds.

Table 1.
Uses of Lead

Use	U.S. (1975)†	% total	U.K. (1977)†	% total
Storage batteries	634	54	68	23
Petrol anti-knock additives	189	16	55	19
Other (including pipes, cables, compounds, ammunition, pigments, alloys)	353	30	167	58
Total	1176		290	

U.S. data: Pedco (1976)
U.K. data: *World Metal Statistics* 1961-1978
† 1000s of metric tons per annum

The storage battery industry and lead alkyl manufacturing industry are now the largest and second largest single users of lead in both the U.S. and the U.K. (Table 1). It should be noted, however, that a proportion of the annual consumption is from recovered sources; in the U.K. battery industry, for example, approximately 80% of the lead used is recycled. Consumption figures do not, *per se,* give any indication of the pollution and toxicity potential of the various uses of lead. Some uses of lead result in contamination of the environment and may be described as 'unrecoverable' or 'disseminated' sources of lead, while others do not ('fixed' or 'concentrated' sources) and some uses of lead are potentially toxic to humans while others are, generally, not: the degree of potential toxicity is not *necessarily* a function either of the proportional amount used or of the degree to which it is disseminated. For example, as will be discussed in Part 2, lead in household paint has undoubtedly been responsible for more cases of overt childhood poisoning than any other single source of lead although it has always been a comparatively minor proportion of total lead consumption. Much of the high-lead containing paint is found in older pre-war housing and represents a persistent hazard today, although the lead content of indoor paints is now severely restricted and the problem well-recognized.

It is the proportion of lead released into the environment which is the major source of concern and controversy at present, however, owing to a combination of its ubiquity and at least partial unavoidability by the 'receptor'. Most of this disseminated lead is emitted initially into the atmosphere, from two principal sources: motor vehicles and a variety of stationary source processes such as metallurgical smelting, coal and oil combustion, iron and steel production, waste oil combustion and the burning of demolition waste, cables, battery cases (Table 2). It can be seen that mobile sources account for about 90% of all atmospheric emissions of lead.

Table 2.
Atmospheric emissions of lead.

Type	Metric tons × 10		% total Pb consumption/year	
	U.S.	U.K.	U.S.	U.K.
Mobile emissions	142	9	11.9	2.5
Stationary source emissions	19.2	0.3	1.6	0.08

U.S. data: Pedco (1976)
U.K. data: author's estimates from available data

A simplified diagram of the principal pathways of lead through the environment is shown in Fig. 1. To these sources and routes must be added lead from paint, pipes, food processing and cooking vessels and a variety of miscellaneous sources which may be available to certain sections of the population.

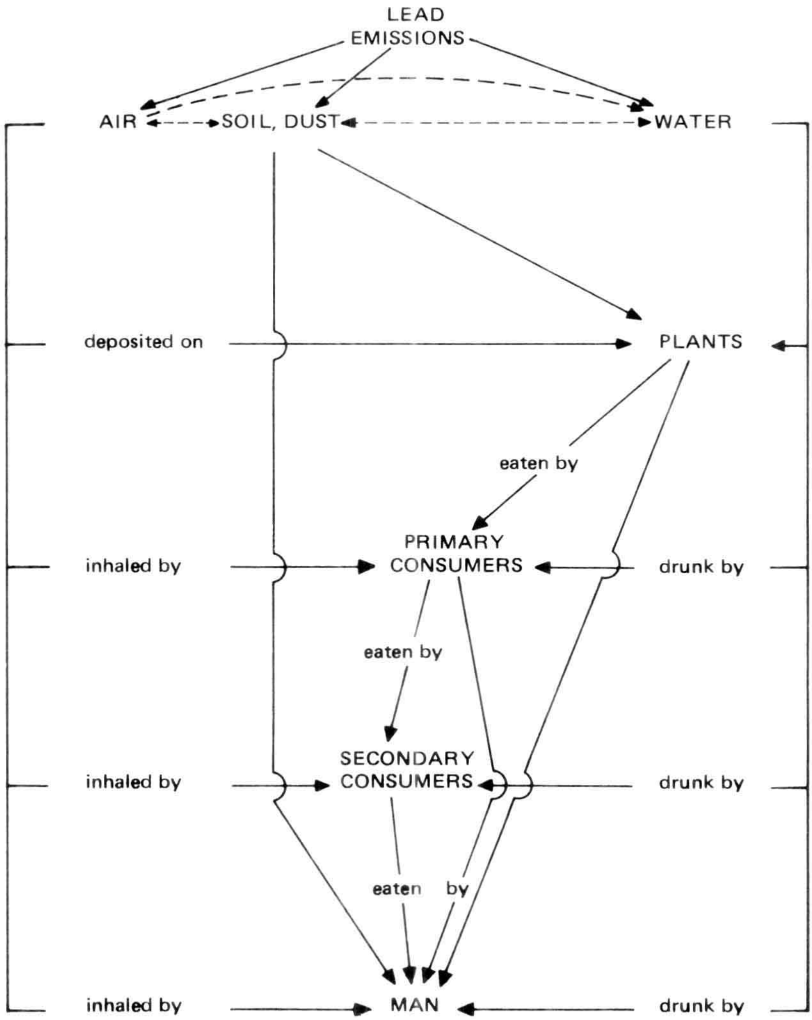

Fig. 1 Principal pathways of lead to man.

Lead, in common with other heavy metals, can be thus described; its toxic properties have been recognized for over two centuries, and a well-established toxicology has been developed in connection with the exposure of industrial workers to lead. As the pattern of lead usage and control has changed over the last century, however, so has the pattern of toxicity. Awareness of the effects of acute, 'clinical' poisoning, that is, intoxication sufficient to produce outward signs and symptoms which can be recognized and diagnosed by a doctor and/or the sufferer, has resulted in a gradual decrease in reported adult cases (principally from industry), although the incidence of acute plumbism is still a considerable problem among young urban children, especially in some inner urban communities in the United States.

The increase in dissemination of lead into the environment from stationary and mobile sources has resulted in concern about the toxicological significance of body burdens of lead insufficient to cause overt plumbism but experienced by large numbers of individuals in the general population. In particular, attention has been focused on the significance to health of lead emissions from motor vehicles since, as we have seen, these provide the bulk of the artificial contamination of the environment. This rapid rise in concern over the last decade is not simply related to the increase in the amount of lead in our surroundings and our bodies, but is also a function of a concomitant growth in environmental awareness about pollutants, accompanied by improvements in our ability to monitor lower levels of pollutants in physical or biological media and the earlier, more subtle effects of toxic agents in organisms. Further, greater affluence and economic growth support the demand for a higher quality of life, including better standards of health and safety at work and in the community, while tending to increase the quantity and type of toxic hazards with which we have to cope. These factors have in turn required the establishment of new branches of scientific research, together with the setting up of bodies to evaluate the toxicity of lead and other hazards and to develop and administer control strategies.

The operation of ensuring the safety of the population with respect to lead, or any other pollutant, involves a multiple-step process (Fig. 2). Firstly, the establishment of dose-effect and response relationships (sometimes referred to as 'health criteria') is aimed at so that the effect of the pollutant on various systems and in different population subgroups in terms of the concentration, rate, frequency and duration of lead dose can be evaluated. The evaluation of these data by various bodies would generally result in the establishment of a biological standard or guideline. The essential prerequisite for this guideline is that it should be expressed in terms of a biological index of dose which can be related to levels of effect on the one hand and levels of exposure on the other; it must also be a fairly simple and 'non-invasive' measure of dose for the purposes of monitoring the population by screening procedures. If certain groups within the population are found to exceed the guideline (on the basis of such biological screening), a control strategy must then be adopted to reduce the relevant source of exposure. This implies that the contribution of the source to the body can be established and the corresponding degree of control required derived. This is not particularly easy in the simple case of a single source pollutant with a single pathway to the human receptor, and is enormously complicated in the case of lead by the multiplicity of sources and pathways. For example, initially airborne lead may contribute to the body burden as both an inhaled dose and as an ingested dose, following deposition. Once the lead is absorbed, the source is of little toxicological consequence to the ultimate receptor but is important when considering the most appropriate control strategy.

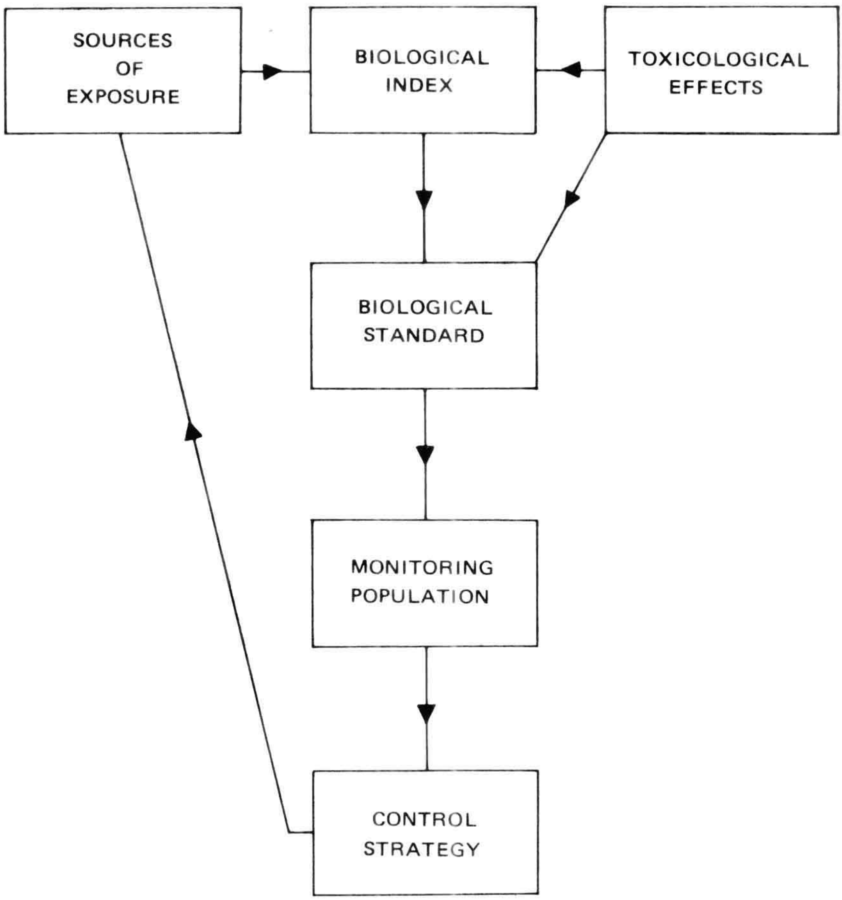

Fig. 2 Steps in the control of environmental lead.

It is the purpose of this book to discuss these processes and their interrelationships. In the first part, the toxicity of lead and the evaluation of data for the establishment of biological guidelines in terms of a suitable biological index will be discussed. Particular attention will be focused on health effects in young children, who may be said to represent a critical subgroup of the population from the point of view of exposure and, arguably, physiological susceptibility. The occupational exposure of adults to lead, since it involves the use of separate criteria for acceptable exposure and somewhat different control strategies, will not be dealt with in this volume.

The second part deals with the relative contribution of different sources of exposure and the development of control strategies in relation to biological guidelines of acceptable dose and exposure. In particular, the problems of assessing the importance of airborne lead, and the appropriateness of air quality standards in its control, will be discussed.

PART 1:
THE HEALTH EFFECTS OF LEAD AND THEIR EVALUATION

Chapter 1

Health effects of lead: I

1.1 DEFINITIONS

There are several terms, such as 'exposure', 'dose', 'effect' and 'response' which are used frequently and sometimes interchangeably in the literature, so that some clarification of terms as they will be used throughout the text is necessary.

The **exposure** of an individual may be considered as consisting of two components: (1) the external exposure, which concerns the concentration of lead in air, food, water and other sources which are available to (that is, can reach) the receptor (for example, the child) and (2) the intake, which is the amount of lead (per unit time) taken into the gut or the lung from these sources. (For example, if a child eats 100 grams of food per day which has a lead concentration of 2 μg/g, its daily intake of lead from food is 200 μg/day).

The concept of **dose** has, *inter alia,* been discussed at some length by members of the Task Group on Metal Toxicity of the Subcommittee on the Toxicology of Metals under the Permanent Commission and International Association on Occupational Health. The consensus report states: 'Ideally, the dose should be defined as the amount or concentration of a given chemical at its site of effect, i.e. where its presence leads to a given effect'. (p. 15. Nordberg, 1976). The concentration of lead in a specific organ or site of effect depends on the kinetics of absorption, distribution and excretion of lead. Since the measurement of dose at the site of effect cannot often be measured directly, reliance must therefore be placed on *estimates* of 'site of effect' dose from the measurement of lead in more accessible biological media such as blood, urine and faeces. (Nordberg & Norseth 1976) (see biological index, below). The term **whole body dose** may also be used to describe the total amount of lead *absorbed* from the gut and lung (per unit time) and available for distribution, storage and excretion. The amount of lead absorbed through the gut or lung is a proportion of the intake and varies according to factors such as the physical and chemical characteristics of the lead species, rate and depth of ventilation in the lung, presence of other nutrients in the gut, and so forth. It is difficult to measure the whole body dose with accuracy except under certain controlled experimental conditions, and again, estimates are used based on averages for intake and expected proportional absorption under different conditions.