ORNL-5263

Ac 719

CUMEX—A Cumulative Hazard Index for Assessing Limiting Exposures to Environmental Pollutants

P. J. Walsh
G. G. Killough
D. C. Parzyck
P. S. Rohwer
E. M. Rupp
B. L. Whitfield
R. S. Booth
R. J. Raridon

MASTER

ENVIRONMENTAL SCIENCES DIVISION
Publication No. 1007

BLANK PAGE

**Printed in the United States of America. Available from
National Technical Information Service
U.S. Department of Commerce
5285 Port Royal Road, Springfield, Virginia 22161
Price: Printed Copy \$5.00; Microfiche \$3.00**

This report was prepared as an account of work sponsored by the United States Government. Neither the United States nor the Energy Research and Development Administration, United States Nuclear Regulatory Commission, nor any of their employees, nor any of their contractors, subcontractors, or their employees, makes any warranty, express or implied, or assumes any legal liability or responsibility for the accuracy, completeness or usefulness of any information, apparatus, product or process disclosed, or represents that its use would not infringe privately owned rights.

ONNL-5263
Distribution Category UC-41

Contract No. W-7405-eng-26

**CUMEX - A CUMULATIVE HAZARD INDEX FOR ASSESSING LIMITING
EXPOSURES TO ENVIRONMENTAL POLLUTANTS**

P. J. Walsh
G. G. Killough
D. C. Parzyck
P. S. Rohwer
E. M. Rupp
B. L. Whitfield
R. S. Booth
R. J. Raridon

1 Information Division
2 Instrumentation and Controls Division
3 Computer Sciences Division

Date Published: April 1977

ENVIRONMENTAL SCIENCES DIVISION
Publication No. 1007

OAK RIDGE NATIONAL LABORATORY
Oak Ridge, Tennessee 37830
operated by
UNION CARBIDE CORPORATION
for the
ENERGY RESEARCH AND DEVELOPMENT ADMINISTRATION

MASTER

ACKNOWLEDGMENTS

The authors wish to acknowledge the helpful review and comments provided by the following members of the Environmental Sciences Division staff: S. I. Auerbach, E. G. Struxness, S. V. Kaye, R. I. Van Hook, C. W. Miller, C. C. Travis, and C. A. Little. The authors would especially like to thank E. C. Jones for her important contribution toward completion of the final report and all previous versions. Also, thanks is due to A. L. Lawrence for assistance with early drafts of the report.

ABSTRACT

WALSH, P. J., G. G. KILLOUGH, D. C. PARZYCK, P. S. ROHWER, E. M. RUPP, B. L. WHITFIELD, R. S. BOOTH, and R. J. RARIDON.
1977. CUMEX - A Cumulative Hazard Index for Assessing Limiting Exposures to Environmental Pollutants. ORNL-5263. Oak Ridge National Laboratory, Oak Ridge, Tennessee. 63 pp.

A hazard index methodology called CUMEX has been developed for limiting human exposure to environmental pollutants. Hazard index is defined as Q/Q_L where Q is exposure or dose to total-body, organ or tissue from all environmental pathways and Q_L is a limit which should not be exceeded because of health risk to humans.

Mathematical formulations for hazard indices are developed for each sampling medium corresponding to each effluent type. These hazard indices are accumulated into composite indices such that total human intake or dose would not exceed the health risk limit. Mathematical formulation for composite hazard indices or CUMEX indices for multiple pollutants are presented.

An example CUMEX application to cadmium release from a smelter complex in East Helena, Montana demonstrates details of the methodology for a single pollutant where human intake occurs through inhalation and ingestion.

TABLE OF CONTENTS

	PAGE
LIST OF TABLES	ix
LIST OF FIGURES	xi
LIST OF APPENDICES	xiii
INTRODUCTION	1
CHAPTER 1. CUMEX FORMULATION	2
1.1 Hazard Index	2
1.2 CUMEX Index, Single Pollutant, Multiple Effluent Types, Multiple Pathways	5
1.3 Formulation of a Composite CUMEX Index for Multiple Pollutants	7
1.4 Multiple Pollutants, Different Biological Effects	10
1.5 Conclusions	11
CHAPTER 2. TRANSPORT AND PHYSIOLOGICAL MODELS	11
2.1 Atmospheric Transport Model	12
2.1.1 Input Data	12
2.1.2 Examples	13
2.2 Terrestrial Transport	13
2.2.1 Input Data	15
2.2.2 Output Data	16
2.3 Aquatic Transport	16
2.4 Physiological Models	16
2.5 Selection of Limits	17
CHAPTER 3. JUSTIFICATIONS FOR EXAMPLE CUMEX APPLICATION	18
3.1 Selection of Cadmium	18
3.2 Selection of Endpoint	18

BLANK PAGE

TABLE OF CONTENTS (continued)

	PAGE
3.3 Selection of Helena Smelter Complex	19
3.4 Environmental Concentrations of Cadmium	19
3.5 Standards	21
CHAPTER 4. CUMEX APPLICATION	21
4.1 Atmospheric Transpo. Model	21
4.1.1 Input Data	21
4.1.2 Output Data	22
4.1.3 Comparison with Measurements	23
4.2 Terrestrial Transport Model	31
4.2.1 Input Data	32
4.2.2 Output Data	33
4.2.3 Comparison with Measurements	34
4.3 Calculation of CUMEX Index	34
CONCLUSION	42
APPENDIX I. MODIFICATIONS OF TERMOD	43
APPENDIX II. CADMIUM REVIEW	47
REFERENCES	59

LIST OF TABLES

TABLE		PAGE
3.4.1	Average cadmium concentrations	19
3.4.2	The average daily intake of cadmium as a function of diet for the U.S. and the study site in Helena, Montana	20
4.1.1.1	Source data for East Helena smelter area	22
4.1.2.1	Deposition of cadmium around Helena smelter area	24
4.1.2.2	Air concentrations of cadmium around Helena smelter area	25
4.1.2.3	Deposition of cadmium around Helena smelter area	30
4.1.3.1	Cadmium concentrations in area around East Helena	31
4.2.2.1	Equilibrium cadmium concentrations in various compartments	33
4.2.2.2	Equilibrium intakes by reference man	33

LIST OF FIGURES

FIGURE	PAGE
2.2.1 Block diagram of the terrestrial food pathways	14
4.1.2.1 Average deposition of cadmium (5 _L) around East Helena smelter (mg/m ² /month)	26
4.1.2.2 Average air concentration of cadmium (5 _L) around East Helena smelter (nG/m ³)	27
4.1.2.3 Air concentration of cadmium around East Helena smelter area as a function of particle size and distance	28
4.1.2.4 Deposition of cadmium around East Helena smelter as a function of particle size and distance	29
4.2.3.1 Intake rate of cadmium vs time after an impulse, ground deposition of 15 µg/m ² . Intake is by reference man ⁴³ whose diet consists of 250 dry grams of vegetables/day, 1.0 l milk/day, 300 wet grams of beef/day and a total, solid food intake (less fish, beef, and vegetables) of 2500 wet grams/day. The half-time for loss of cadmium from the subsurface soil pool and pasture soil was 10 years	35
4.2.3.2 Intake rate of cadmium vs time after an impulse, ground deposition of 15 µg/m ² . Intake is by reference man. ⁴³ The half-time for loss of cadmium from the subsurface soil pool and pasture soil was 50 years	36
4.2.3.3 Percent of daily cadmium intake from each pathway as a function of time after an impulse, ground deposition of 15 µg/m ² . Intake is for reference man. ⁴³ The half-time for loss of cadmium from the subsurface soil pool and pasture soil was 10 years. See Fig. 4.2.3.1 for intake rates in µg/day	37
4.2.3.4 Percent of daily cadmium intake from each pathway as a function of time after an impulse, ground deposition of 15 µg/m ² . Intake is for reference man. ⁴³ The half-time for loss of cadmium from the subsurface soil pool and pasture soil was 50 years. See Fig. 4.2.3.2 for intake rates in µg/day	38

BLANK PAGE

LIST OF FIGURES (continued)

FIGURE	PAGE
4.2.3.5 Percent of the total, 50-year intake of cadmium through each pathway up to time t after an impulse, ground deposition of $15 \mu\text{g}/\text{m}^2$. Also, multiplication by 1.05 converts these plots to the intake rate ($\mu\text{g}/\text{day}$) of cadmium as a function of time after a constant deposition source of $15 \mu\text{g}/\text{m}^2\text{-day}$ first appears. Inputs are for reference man. ⁴³ The half-time for loss of cadmium from the subsurface soil pool and pasture soil was 10 years	39
4.2.3.6 Percent of the total, 50-year intake of cadmium through each pathway up to time t after an impulse, ground deposition of $15 \mu\text{g}/\text{m}^2$. Also, multiplication by 2.12 converts these to the intake rate ($\mu\text{g}/\text{day}$) of cadmium as a function of time after a constant deposition source of $15 \mu\text{g}/\text{m}^2\text{-day}$ first appears. Inputs are for reference man. ⁴³ The half-time for loss of cadmium from the subsurface soil pool and pasture soil was 50 years	40

LIST OF APPENDICES

APPENDIX		PAGE
I	Modifications of TERMOD	43
II	Cadmium Review	47

INTRODUCTION

This report describes a hazard assessment methodology for limiting human exposures to environmental pollutants such that exposure or dose will not exceed some preselected value. An estimation of total pollutant intake by humans and resulting health effects must include contributions from all possible routes of exposure. Models must be used for estimation of potential health impacts of pollutant releases that have not yet occurred. They are also useful when measurements can be made. Therefore, an assessment approach has been developed using environmental transport models to predict transport from point of pollutant release or measurement to eventual intake and absorption by humans.

This assessment methodology called CUMEX (cumulative hazard index) is based on consideration of interrelationships between environmental compartments. A measurement in a particular sampling medium, for example, is sufficient to calculate total human intake via all routes of exposure from that sampling medium, within limits imposed by model input data. The CUMEX assessment methodology in part extends an existing radiological assessment technology (CUEX) to the nonradioactive area.^{1,2} Although the assessment problems are similar, some features of radioactive releases have facilitated assessment of their environmental impact. Radioactive releases have been relatively small and subject to strict controls. Possible health hazards of radiation were recognized early, thus research on biological effects and transport of radionuclides through the food chain has been underway for many years. The pharmacological action of radionuclides has not been considered. If only radiation effects are considered, a common unit, the rem, is used to sum effects of several different radionuclides. No comparable index of effect exists for nonradioactive pollutants. This latter point is particularly critical in nonradioactive pollution assessment because simultaneous exposure to several pollutants may result in synergistic or antagonistic effects rather than simple additive effects. Experimental data are essential to assess these effects and are not available for most combinations of pollutants. Therefore, at present, the CUMEX methodology is strictly applicable to single pollutants only. The CUMEX

BLANK PAGE

methodology provides a way to organize and use data obtained from experimental and theoretical studies. In its initial stages, the methodology is purposefully kept as simple as possible while still considering multiple pollutants and multiple environmental pathways.

Transport models used in CUMEX application estimate pollutant dispersion through air and water, deposition on soil and plant surfaces, uptake by plants, concentrations in air and water, intake by animals and human exposure through inhalation and ingestion. Physiological models estimate pollutant uptake and subsequent distribution among organs. Models used at the Oak Ridge National Laboratory for CUMEX application are described in Chapter 2.

The overall CUMEX concept, which ties together pollutant emission, environmental transport, and a human health effect endpoint (for example a statutory limit or standard, organ level, total-body burden, or specific disease state), provides a useful method to assess compliance with existing standards; to suggest standards where none exist; to estimate health effects of a given pollutant release; or to estimate the amount of pollutant released based on concentrations found in a sampling medium.

The major feature of CUMEX is a calculated CUMEX index which relates the concentration of a pollutant in air or other sampling medium to a preselected endpoint such as an organ concentration by consideration of all pathways from point of measurement to endpoint. Derivation of the formulation required for this calculation is presented in Chapter 1 and Chapter 3 discusses selection of cadmium as an important environmental pollutant.

Chapter 4 illustrates application of the CUMEX methodology to cadmium release from the smelter complex in East Helena, Montana.

CHAPTER 1. CUMEX FORMULATION

1.1 Hazard Index

A hazard index (H.I.) is a quotient of exposure or dose of a given environmental pollutant with some "accepted" or "selected" limit. Its application is implied in any case where actual or estimated exposure or

dose to total-body, organ or tissue is compared to a recommended limit for a particular agent. The President's Science Advisory Committee on Chemicals and Health³ recommends that some index of probability of human exposure be used to rank chemicals in the environment. It is suggested that physical, chemical and economic information along with information on biological effects might be combined into an aggregate index of hazard. However, estimation of hazard index when a straightforward measurement or calculation does not apply is neither simple nor widely accomplished. When exposure of humans to multiple environmental pollutants via multiple environmental pathways is of concern, complexities and lack of information sometimes render determination of hazard indices very crude at best. Still the concept is very useful in a general way because it provides a convenient and perhaps meaningful comparison between exposure or dose levels on the one hand and limits based on biological effects on the other.

The hazard index may be defined mathematically as,

$$H.I. = Q/Q_L , \quad (1.1.1)$$

where Q is exposure, or dose to total-body, organ or tissue from all environmental pathways and Q_L is a selected or established limit which should not be exceeded because of health risk to man. The determination of Q may include metabolic or physiological models to connect total-body, organ or tissue burden to environmental levels if Q_L is available in these terms.

Fortunately, exposure modes resulting in intake by man are limited. They are inhalation, ingestion, transpiration or entry through a break or wound. Thus with Q defined as organ burden, we may write

$$Q = Q_H + Q_G + Q_T + Q_W , \quad (1.1.2)$$

where Q_H , Q_G , Q_T , and Q_W represent components due to inhalation, ingestion, transpiration, and entry through a wound, respectively. We may

further express these quantities in terms of environmental concentrations affecting them as follows:

$$Q_H = C_A V_A f_a \tilde{R} \text{ (}\mu\text{g)} , \quad (1.1.3)$$

$$\tilde{R} = \int_0^T R(t) dt ,$$

where

C_A = pollutant concentration in air ($\mu\text{g}/\text{cm}^3$),

V_A = breathing rate of reference individual (cm^3/day),

f_a = fraction of inhaled pollutant deposited in reference organ,

$\tilde{R}$ = cumulative retention to time T of pollutant in reference organ (days), and

$R(t)$ = time dependence of fractional retention of pollutant in reference organ.

$$Q_G = (C_F M_F + C_W V_W) f_w \tilde{R} \text{ (}\mu\text{g)} , \quad (1.1.4)$$

where

C_F = average concentration of pollutant in food ($\mu\text{g}/\text{g}$),

M_F = mass consumption rate of food (g/day),

C_W = average concentration of pollutant in drinking water ($\mu\text{g}/\text{ml}$),

V_W = volume consumption rate of drinking water (ml/day), and

f_w = fraction of ingested pollutant reaching reference organ.

$$Q_T = C_A K f_b \tilde{R} \text{ (}\mu\text{g)} , \quad (1.1.5)$$

where

C_A = concentration of pollutant in air ($\mu\text{g}/\text{cm}^3$),

K = partition coefficient for pollutant between air and blood ($\mu\text{g}/\text{ml}$ per $\mu\text{g}/\text{cm}^3$), and

f_b = fraction of pollutant in blood that is deposited in the reference organ.

Intake due to entry through break or wound is not generally quantifiable and must be analyzed for each specific instance. Fortunately, this intake route is not usually significant compared to the major routes of inhalation and ingestion.

Additional factors or fewer factors could be included in the above equations depending on whether Q applies to tissue, organ or total-body concentrations.

1.2 CUMEX Index, Single Pollutant, Multiple Effluent Types, Multiple Pathways

Determination of C_A , C_F and C_W defined in Section 1.1 requires an environmental assessment or human exposure assessment. If the relationships among the various environmental compartments are understood, then measurements in a particular sampling medium (air, water, food) can suffice, along with transport models, to assess human intake. Measurements in at least two sampling media, along with transport models, will be necessary to assess total human intake if there is more than one effluent type. For example, suppose we wish to determine an air concentration, C_A , such that an organ burden Q from all pathways and all routes of entry is equal to an accepted organ burden limit Q_L . In such a case we set $Q = Q_L$ and solve for C_A , thus:

$$C_A = \frac{Q_L}{\left[V_A f_a + (C_F/C_A) M_F f_w + (C_W/C_A) V_W f_w + K f_b \right] \bar{R}} \quad (1.2.1)$$

Equation (1.2.1) obviously requires relationships among C_A , C_F , and C_W . The ratio (C_F/C_A) is not simply the transfer coefficient from air to food since the concentration in food results from other pathways as well. Similarly (C_W/C_A) is not simply the transfer coefficient from air to water. Calculation of C_A by Eq. (1.2.1) requires that C_A , C_F and C_W

be determined separately. However, there is a simpler approach which yields to more direct analysis.

A hazard index may be determined for each sampling medium corresponding to each effluent type. Hazard indices may then be accumulated into a composite hazard index. From these hazard indices one may determine an exposure such that total human intake or dose is equal to some preselected value. We call such a cumulative exposure index a CUMEX index. If only one sampling medium contributes to human intake then the CUMEX index is determined such that intake via all environmental pathways from that sampling medium is equal to some preselected value. Thus the CUMEX index, C_A^* , for an airborne effluent with air as the sampling medium is,

$$C_A^* = \frac{Q_L}{(V_A f_a + f_{AF} M_F f_w + f_{AW} V_W f_w + k f_b) \tilde{R}} , \quad (1.2.2)$$

where

f_{AF} = transfer coefficient from air to food, and

f_{AW} = transfer coefficient from air to water.

Both f_{AF} and f_{AW} are determined by environmental transport models.

The CUMEX index for a liquid effluent with water as the sampling medium is,

$$C_W^* = \frac{Q_L}{(f_{WF} M_F f_w + V_W f_w) \tilde{R}} , \quad (1.2.3)$$

where f_{WF} = transfer coefficient from water to food. When both effluent types are present, joint limits on C_A and C_W are set by the relation,

$$\frac{C_A}{C_A^*} + \frac{C_W}{C_W^*} \leq 1 . \quad (1.2.4)$$

The inequalities, Eq. (1.2.4), and $C_A \geq 0$, $C_W \geq 0$ correspond to the shaded region in the diagram below. The analysis could, of course, be extended to higher dimensions.

The above discussion illustrates the major features of the CUMEX concept for a single pollutant. As environmental transport models evolve for determination of f_{AF} , f_{AW} , f_{WF} , etc., so will the CUMEX concept. As more complicated kinetic processes are incorporated into the models, such as photochemical, chemical or biological alterations of pollutants, determination of CUMEX indices will become more precise.

1.3 Formulation of a Composite CUMEX Index for Multiple Pollutants

We consider measures of exposure Q_1 , Q_2 to two pollutants, such as burdens in an organ. Associated with the presence of either or both is a discernible effect which can be used to quantify limits.

We conceive of a series of experiments in which various combinations of the two toxicants are introduced into the tissues at risk. For a given level of Q_1 , we imagine an increase in Q_2 until the limiting

effect is observed, and the process is repeated for other levels of Q_1 . The results might be visualized as in the diagram below:

where the plotted x's represent the attainment of the limiting effect. A model for the limiting curve on which x's lie might be hypothesized as

$$F(Q_1, Q_2) = A_1 Q_1 + A_2 Q_2 + K_{12} Q_1 Q_2 = 1 \text{ (conic section) .} \quad (1.3.1)$$

Our problem may be viewed as that of estimating the parameters A_1 , A_2 , and K_{12} in order that we might control the biological availability of the pollutants in such a way as to keep

$$F(Q_1, Q_2) < 1 . \quad (1.3.2)$$

The coefficients have the following interpretations:

$$\begin{aligned} A_1^{-1} &= \text{limiting burden of } Q_1 \text{ in the absence of } Q_2, \\ A_2^{-1} &= \text{limiting burden of } Q_2 \text{ in the absence of } Q_1, \end{aligned}$$

K_{12} = interaction term which characterizes the synergistic effect of both agents:

$K_{12} > 0$ implies reinforcement,

$K_{12} < 0$ implies interference, and

$K_{12} = 0$ implies no interaction.

These interpretations may be visualized as follows:

The inequalities $F(Q_1, Q_2) < 1$, $Q_1 \geq 0$, $Q_2 \geq 0$ correspond to the shaded region under the curve limited below and to the left by the coordinate axes:

Generalization of the formulation to higher dimensions is straightforward:

$$F(Q_1, \dots, Q_N) = \sum_{i=1}^N A_i Q_i + \sum_{j=1}^N \sum_{i=1}^{j-1} K_{ij} Q_i Q_j \quad (1.3.3)$$

+ higher-order interaction terms.

In general, however, it is doubtful that sufficient experimental information could be generated for estimation of higher-order interaction coefficients.

To illustrate the application, consider environmental levels of pollutants 1 and 2, with air concentrations $C_{A,1}$, $C_{A,2}$. The equation for one pollutant is Eq. (1.2.2). The limit for two pollutants is reached whenever

$$F(Q_1, Q_2) = A_1 E_1 C_{A,1} + A_2 E_2 C_{A,2} + K_{12} E_1 E_2 C_{A,1} C_{A,2} = 1, \quad (1.3.4)$$

where

$$E_j = (V_A f_{a,j} + f_{AF,j} M_F f_{w,j} + f_{AW,j} V_W f_{w,j} + K_j f_{b,j}) \tilde{R}_j. \quad (1.3.5)$$

Thus a critical air concentration pair $(C_{A,1}, C_{A,2})$ lies on the curve whose equation is Eq. (1.3.4). This curve takes synergism into account in the third term.

The above analysis applies for multiple pollutants in one sampling medium for one effluent type. The application may be extended to incorporate multiple effluent types and multiple sampling media in a manner similar to that given in Section 1.2.

1.4 Multiple Pollutants, Different Biological Effects

The discussion of multiple pollutant effects given in Section 1.3 applies strictly only if the multiple pollutants affect the same target

organ and influence the same biological effects. If the pollutants affect different organs or produce different biological effects in the same or different organs, then other considerations will have to be included in the model. For example, each pollutant may have to be weighted according to its influence on the total well being of the organism. Research along such lines should eventually lead to a comprehensive approach for determination of cumulative hazard indices or CUMEX indices.

1.5 Conclusions

It is conceptually feasible to determine multimedia, multipathway and multipollutant cumulative hazard indices such that control of pollutant concentrations in one or two sampling media corresponding to whether there is one or more effluent types will assure that total human intake will not exceed some preselected value. Such cumulative hazard indices determined as limiting exposures are called CUMEX indices. A CUMEX index is site specific in terms of emission sources and sampling media but there is no conceptual barrier to determination of CUMEX indices for multiple sources as well as multiple pathways to the sampling medium.

CUMEX indices conceptually provide an interface between human exposure assessments and human health risk assessment. Their practical determination, however, requires detailed knowledge of source emission characteristics, environmental transport processes and biological effects. Thus the CUMEX concept provides a framework to assimilate such knowledge and to point out where more research is needed. Its applications will be more definitive as information develops; however, it is certain that such concepts will have to be applied conservatively before all desired information is available.

CHAPTER 2. TRANSPORT AND PHYSIOLOGICAL MODELS

A brief description of models used in present application of CUMEX methodology at Oak Ridge National Laboratory (ORNL) follows.

2.1 Atmospheric Transport Model

The Atmospheric Transport Model (ATM) is based on a Gaussian plume model and is capable of calculating wet and dry deposition and air concentration of gaseous and particulate pollutants at various receptor points located within 50 km of a source or sources.^{4,5} The ATM can treat point, area, and line sources, including wind blown sources. As the plume from each source travels downwind (x-direction) it spreads out (y- and z-directions). The user can specify computation of the amount of such spreading from either Pasquill-Gifford or Briggs-Smith dispersion parameters. The plume is depleted by both dry and wet removal. Dry removal involves fallout and deposition. The dry fall deposition mechanism employs a deposition and terminal velocity concept such that the net flux of material is from plume to landscape surface at a rate proportional to the atmospheric concentration in the layer adjacent to the ground surface. Gases such as SO_2 will deposit on a surface even though they have no terminal velocity. For particulate emissions, the plume is assumed to "tilt" downward at an angle determined by the terminal velocity of the particle in question. For gases, the plume does not tilt.

2.1.1 Input Data

Input data required to apply ATM to a given area are as follows:

- (1) Source characteristics. Sources of pollutants may be points such as smoke stacks; lines, such as roads; or areas, such as cities or industrial complexes. There is also provision for including a wind blown source such as a pile of mine tailings. For each source location it is necessary to specify rate of pollutant output, height at which the pollutant is being emitted, and for stack release, plume rise parameters, which are related to effluent velocity and temperature.
- (2) Pollutant characteristics. For gases the boundary layer thickness and diffusion constant for washout are required. For particulates the density and diameter or particle size distribution must be specified. Chemical properties for both gases and particulates should be specified.

(3) Receptor characteristics. Location of receptor points relative to the sources must be given. Also, type of cover (grassland or forest) and surface roughness must be specified.

(4) Meteorological conditions. The wind rose for the source region is required as a function of time period which may be an hour, day, month, season or year. The windrose is built into the model as a frequency table, which describes as a fraction of time the occurrence of a particular combination of stability class type, wind direction, and wind speed class. Frequency and intensity of rainfall is also required and in some cases it may be necessary to differentiate between daylight and night conditions.

2.1.2 Examples

The ATM has been applied to the area around Oak Ridge, Tennessee, to estimate the impact of effluents from coal-fired steam plants on the environment. Correlation of sulfur dioxide measurements at several gages with ATM predictions was good ($r=0.80$).⁶ Application of the ATM on Walker Branch Watershed illustrated close agreement between measured and computed depositions of particulates.⁷ For example, for Cr, Mn, and Ni, maximum computed depositions were 7, 30, and 10 g/ha-month, respectively, while corresponding ranges of measured input were: Cr (0.5-11), Mn (4-58), and Ni (1-20).

If source strength and input data are known, deposition and air concentration at a given location can be computed. Conversely, if a maximum limit of deposition or air concentration is specified, ATM can be used to compute maximum allowable pollutant output from a given source.

2.2 Terrestrial Transport

Output from the ATM in terms of $\mu\text{g}/\text{m}^2\text{-day}$ of the pollutant deposited on the ground at a given distance from the pollutant source can serve as input to TERMOD.^{1,2} Terrestrial transport pathways considered by TERMOD are shown in Fig. 2.2.1.

ORNL-DWG 70-11733AR3

Fig. 2.2.1. Block diagram of the terrestrial food pathways.

In general, build-up of pollutants in food chains and their flow and distribution in the environment can be divided into a series of compartments. Distribution in soil will depend upon air concentrations and hydrologic redistribution as well as tilling of soil and action of earthworms, erosion, resuspension, rainfall, types of vegetation present, soil type, pH, and wind action.^{8,9,10}

Since a smelter or power plant may be considered a point source, there is an inverse relationship between concentration at the source and distance from the source. In plants, as in soil, there is a correlation between distance from the pollutant source and pollutant concentrations in vegetables and grasses.⁷ There is also a positive correlation between soil concentration and levels in plants.^{11,12,13}

2.2.1 Input Data

Application of TERMOD is illustrated in Fig. 2.2.1. Pollutant deposition is the only source compartment and man is the only receptor compartment of interest. Detailed discussions of TERMOD parameters are given in Refs. 2 and 21.

Three transfer coefficients are usually considered a function of the element being modeled. These are:

- (1) $\tau_{g,b}$, transfer coefficient from grass to beef,
- (2) $\tau_{g,c}$, transfer coefficient from grass to cow (milk),
- (3) $\tau_{p,m}$, transfer coefficient from subsurface soil pool for the food crop to man.

A single value or set of values are usually selected for the remaining transfer coefficients. They are selected with the objectives that the model be realistically conservative, the dependent variables simulate a realistic dynamic behavior and the biomass and pollutant mass be conserved.² Application of TERMOD in CUMEX application to cadmium is described in Chapter 4. Modifications of TERMOD necessary for CUMEX application to cadmium are given in Appendix I.

2.2.2 Output Data

Outputs of TERMOD, as illustrated in Fig. 2.2.1, are intakes of pollutants by man through consumption of milk, beef and plant parts contaminated directly by deposited pollutants as well as by uptake from soil.

Pollutant concentrations in soil, plants and animals as calculated by the transport model as well as model predictions of dietary intake by humans can be compared to experimentally determined values. Predictions can be made of environmental responses to changes in pollutant emission levels.

2.3 Aquatic Transport

Aquatic transport models can be used to estimate pollutant flow through the aquatic food chain. In the Environmental Sciences Division at ORNL AQUAMOD¹⁴ is the aquatic transport model. AQUAMOD is an aquatic ecosystem model and was originally intended for predicting dose to man from a radioactively contaminated aquatic environment. Modifications to AQUAMOD are being made in order to have the capability of predicting aquatic food chain transport of nonradioactive pollutants. A specific activity approach is used for both radioactive and nonradioactive pollutants. In the near future AQUAMOD will have the capability of calculating time dependent bioaccumulation factors. These will be useful in assessment work when empirically determined time dependent bioaccumulation factors are unavailable for accidental releases of pollutants into an aquatic environment.

2.4 Physiological Models

Physiological models used in CUMEX application vary with properties of the pollutant being considered. For the assessment of cadmium release presented in Chapter 4 the model discussed by Friberg et al. was

used.^{9,13} The model computes total-body burden $B(t)$ (μg) at time t as follows:

$$B(t) = \frac{If(1 - e^{-\lambda t})}{\lambda}, \quad (2.4.1)$$

where I = intake rate ($\mu\text{g/day}$) for the particular exposure mode (ingestion or inhalation),

f = fractional absorption for the particular exposure mode, and

λ = fractional daily excretion rate.

2.5 Selection of Limits

Selection of the exposure, dose or concentration limit Q_L depends upon knowledge of biological effects of the particular environmental pollutants of concern. Sometimes such information is readily available as exposure or dose limits but in other cases Q_L will require a fully documented derivation. It is imperative that the method of determination of Q_L be clearly specified in each application.

Selection of kidney cortex as the critical tissue for cadmium is discussed in Chapter 3; however, to enable the physiological model to calculate cadmium concentration in kidney cortex as the critical tissue the following formula must be included:

$$C(t) = \frac{1}{3} (1.5) \frac{B(t)}{m}, \quad (2.5.1)$$

where

$C(t)$ = concentration in kidney cortex ($\mu\text{g/g}$),

$\frac{1}{3}$ = fraction of total-body burden $B(t)$ found in kidneys,

m = mass of kidneys (300 g), and

1.5 = increase in cadmium concentration of kidney cortex over that of kidney as a whole.

CHAPTER 3. JUSTIFICATIONS FOR EXAMPLE CUMEX APPLICATION

3.1 Selection of Cadmium

Cadmium is a toxic heavy metal widely used in a variety of consumer products. It produces acute health effects when inhaled or ingested at high concentration and chronic effects at lower concentrations. As an ubiquitous element in the environment, it is found in normal diet and in air in most urban areas. It is excreted from the body extremely slowly, accumulating to potentially toxic levels when intake is sufficient. As an environmental pollutant of increasing importance due to increasing commercial use and fossil fuel combustion and as a pollutant about which health effects data are available, cadmium is well suited for testing the CUMEX methodology. (See Refs. 7 and 9 and Appendix II for more detailed discussion.)

3.2 Selection of Endpoint

The endpoint chosen for calculation of a CUMEX index for cadmium release is 200 $\mu\text{g Cd/g wet weight of kidney cortex}$ (i.e. $Q_L = 200 \mu\text{g/g}$). From epidemiological and experimental studies kidneys are the long-term target organ for low-level exposure to cadmium.⁹ A concentration of 200 $\mu\text{g Cd/g wet weight of kidney cortex}$ is generally accepted as a level above which kidney damage occurs.⁹ At constant exposure levels the increase in kidney cadmium concentration is almost linear through age 50 years. The CUMEX index calculations in this report are intended to prevent accumulation above 200 $\mu\text{g Cd/g wet weight kidney cortex}$ in 50 years, assuming a total kidney weight of 300 g and a fractional absorption of 0.05 for ingestion and 0.25 for inhalation.^{7,9} Excretion is assumed to be 0.005% of total body burden per day. (See Refs. 7 and 9 and Appendix II for more detail.)

3.3 Selection of Helena Smelter Complex

CUMEX is a site specific assessment methodology; therefore, a site of cadmium release was selected where source data and other input data for transport models were available. A study by the U.S. Environmental Protection Agency (EPA), conducted between June 1969 and June 1970 provided the required data for an area around East Helena, Montana, site of a lead smelter and slag processing facility.⁸ Cadmium is one of the major pollutants released from this smelter complex.

3.4 Environmental Concentrations of Cadmium

Average cadmium concentrations in air and water for the United States as a whole and for the Helena Valley and East Helena are given in Table 3.4.1.

Table 3.4.1. Average cadmium concentrations

Sample Medium	U.S. ⁹	Helena Valley ⁸	East Helena
Air ($\mu\text{g Cd/m}^3$)	0.02 urban 0.003 nonurban	0.03	0.06-0.29
Water ($\mu\text{g Cd/l}$)	<10	<10	<10

Average Cd concentrations in foods are quite variable but usually less than $0.05 \mu\text{g Cd/g}$ wet weight.⁷

Table 3.4.2 shows cadmium concentrations in items of selected food categories given on a wet weight ($\mu\text{g/g}$) basis. The cadmium concentration values have been taken from a number of different source documents and these references are given in Table 3.4.2. Table 3.4.2 also gives the daily intake of cadmium for each of the major food categories with the daily cadmium intake value for all categories given at the bottom of the table.

Daily intake values given in Table 3.4.2 are based on a daily food intake of 1.75 kg as compiled by the U.S. Department of Agriculture¹⁵

Table 3.4.2. The average daily intake of cadmium as a function of diet for the U.S. and the study site in Helena, Montana

Food category	Consumption g/day - edible portions	Cadmium concentration	Average intake - µg/day			References
		Range - µg/g wet wt ^a	U.S.	Helena ^b Valley ^b	East Helena ^b	
Meat, fish, poultry	245	0.03-0.054	11.9	12.3	15.5	7,40,27
Vegetables, potatoes	434	0.038-0.090	31.5	69.2	109.4	28,40,7,9,54
Fruits, juices	179	0.040-0.070	12.0	11.4	13.0	9,54
Dairy products, eggs	486	0.021-0.028	13.3	13.3	13.3	16,55
Grain products	177	0.026-0.211	21.8	21.8	21.8	28
Fats, oils (coffee, tea)	81	0.01-0.04	2.7	2.7	2.7	9
Sugars, sweeteners	149	0.04	6.0	6.0	6.0	9
Water and beverages	1270	0.01	12.7	12.7	12.7	
Total from food	1751		99.2	136.7	181.7	
Total including water and beverages			111.9	149.4	194.4	

^aBecause of differences in the amount of consumption of various foods within a category, an average of the range of concentrations of cadmium in foods cannot be used. This becomes obvious when food categories are further broken down into individual items; and fresh vs. processed foods.

^bEast Helena and Helena Valley figures are based on the assumption that only 12% of the garden vegetables consumed there are grown locally and that the other 88% of the diet is shipped in from other areas.⁸

rather than 2.0-2.5 kg per day as usually reported for the United States.^{7,9,16} Extrapolation from intake values in Table 3.4 2 to those arising from intake of 2.0-2.5 kg per day gives the following daily intake of cadmium:

Average U.S.	128-160 μ g
Helena Valley	170-213 μ g
East Helena	222-278 μ g

3.5 Standards

There are no standards set for cadmium in food products or ambient air; however, a World Health Organization Report on food additives proposed an intake limit of 400-500 μ g Cd per week.¹⁷ A threshold limit value (TLV) of 100 μ g/m³ of air is established for occupational exposure to cadmium.^{18,19} The maximum permissible concentration of cadmium in drinking water is 0.01 ppm (10 μ g/l).²⁰

CHAPTER 4. CUMEX APPLICATION

4.1 Atmospheric Transport Model

A brief description of the atmospheric transport model used at ORNL was given in Section 2.1.

4.1.1 Input Data

Input data necessary to apply CUMEX methodology to cadmium release from the smelter complex in East Helena, Montana, were obtained from the open literature. Source data were taken from Ref. 8. The two sources considered, the American Smelting and Refining Company (ASARCO) lead smelter and the Anaconda slag processing facility, are located side-by-side in East Helena. Table 4.1.1.1 lists the source data.

Table 4.1.1.1. Source data for East Helena smelter area

	ASARCO	Anaconda
Stack height	122 m	36 m
Stack radius	2.44 m	1.38 m
Stack gas velocity	8.2 m/sec	2.1 m/sec
Stack gas temperature	127°C	121°C
Particulate emissions	56 g/sec	112 g/sec
Particle size	unknown ^a	unknown ^a
Particle density	unknown ^b	unknown ^b
Cd concentration in particulate emissions	200-1000 ppm ^c	200-1000 ppm ^c
Plume rise parameters		
PKAPPA ^d	877	155
QKAPPA ^d	235	100

^aSimulations made for several particle diameters (0.1, 1.0, 5.0, 10.0, 20.0 μ).

^bAssumed to be 7 g/cm³ (average density of Cd and Cd compounds).

^c1000 ppm used in calculations.

^dValues determined from consideration of stack gas velocity, stack radius, stack gas temperature, wind speed and other factors and used to calculate increase in effective stack height (plume rise) due to buoyancy of the plume (Culkowski and Patterson).⁵

Meteorological data given in Ref. 8 included monthly wind rose data for six velocity groups ranging from 0-12.5 m/sec, percent occurrence of stability classes A through F, average monthly and yearly rainfall and average yearly rainfall intensity.

Terrain surrounding the smelter complex was assumed to be grassland with a surface roughness factor of 0.15 m.⁴ Receptor points were uniformly spaced, 8 each at distances of 1 mile and 2 miles, and 16 each at distances of 4 miles and 8 miles, for a total of 48.

4.1.2 Output Data

Output from ATM includes wet and dry deposition values and air concentrations of cadmium at each of the 48 receptor points for each

quarter of the year. Wet and dry deposition values are nearly equal. Tables 4.1.2.1 and 4.1.2.2 list total depositions and air concentrations of cadmium (5μ particle) for each quarter, plus a yearly average, as a function of distance and direction from the smelter. In general, seasonal deviation is less than 10% of the average. Yearly averages in Tables 4.1.2.1 and 4.1.2.2 are plotted in Figs. 4.1.2.1 and 4.1.2.2 to show spatial distribution. Values are highest toward the east since the wind blows 45% of the time from the WNW to WSW sectors. At a distance of 8 miles, values to the east are five to eight times higher than values to the west. Figures 4.1.2.3 and 4.1.2.4 show how air concentrations and depositions of cadmium change as a function of particle size and distance on a straight line east from the smelter complex. The curves for 0.1μ particles are not shown because they are close to the ones for 1μ particles. There is a decrease of one to two orders of magnitude as the distance increases from 1 to 8 miles. Table 4.1.2.3 gives yearly deposition averages for all five particle sizes assumed. It is apparent that the simulated values for cadmium deposition and air concentration are quite dependent on the assumed particle size. At a distance of 1 mile east, deposition of 20μ particles is about 20 times as great as for 1μ particles. At 8 miles, there is less than a factor of 4 difference since larger particles are depleted from the plume. At a distance of 1 mile east, there are more 20μ particles per unit volume of air than smaller sizes while at 8 miles 5μ particles are most abundant.

4.1.3 Comparison with Measurements

Partial validation of ATM simulations for the smelter area is possible through comparison of the results with data in the EPA report⁸ (see Table 4.1.3.1). Within a radius of 1 mile of the smelter measured cadmium depositions ranged from 1 to $4 \text{ mg/m}^2/\text{month}$. Simulated results at 1 mile east of the smelter ranged from $0.7 \text{ mg/m}^2/\text{month}$ for a 1μ particle to $15 \text{ mg/m}^2/\text{month}$ for a 20μ particle. Thus, simulated results for $1\text{--}10 \mu$ particles compare favorably with experimental data. Within

Table 4.1.2.1. Deposition of cadmium around Helena smelter area

PARTICLE DIAMETER IS 5.0 MICRONS AND DENSITY IS 7.0 GM/CM³

DIR.	MILES	TOTAL DEPOSITION (MG/M ² /PERIOD)				AVERAGE
		JAN-MAR	APR-JUN	JUL-SEP	OCT-DEC	
E	1	1.50E+00	1.41E+00	1.24E+00	1.45E+00	1.42E+00
E	2	8.30E-01	7.04E-01	6.34E-01	7.77E-01	7.36E-01
E	4	4.35E-01	3.35E-01	3.17E-01	4.32E-01	3.81E-01
E	8	2.12E-01	1.53E-01	1.54E-01	2.36E-01	1.89E-01
FNE	4	3.25E-01	2.69E-01	3.31E-01	3.67E-01	3.23E-01
FNE	8	1.43E-01	1.27E-01	1.62E-01	2.09E-01	1.64E-01
NF	1	6.08E-01	5.75E-01	6.42E-01	6.28E-01	6.13E-01
NF	2	3.39E-01	3.00E-01	3.42E-01	3.50E-01	3.33E-01
NF	4	1.56E-01	1.51E-01	1.76E-01	2.06E-01	1.83E-01
NE	8	1.07E-01	7.01E-02	9.03E-02	1.20E-01	9.56E-02
NNF	4	8.83E-02	6.36E-02	6.76E-02	1.01E-01	8.01E-02
NNF	8	4.57E-02	3.03E-02	3.56E-02	5.75E-02	4.23E-02
N	1	4.35E-01	2.52E-01	3.06E-01	5.02E-01	3.54E-01
N	2	2.41E-01	1.57E-01	1.70E-01	2.77E-01	2.11E-01
N	4	1.39E-01	8.33E-02	9.17E-02	1.66E-01	1.20E-01
N	8	7.26E-02	3.58E-02	4.79E-02	9.78E-02	6.46E-02
NNW	4	8.05E-02	4.60E-02	7.43E-02	7.63E-02	6.57E-02
NNW	8	4.21E-02	2.12E-02	3.87E-02	4.60E-02	3.70E-02
NW	1	2.39E-01	2.45E-01	2.74E-01	2.92E-01	2.62E-01
NW	2	1.36E-01	1.35E-01	1.48E-01	1.67E-01	1.46E-01
NW	4	7.56E-02	7.37E-02	7.82E-02	1.01E-01	8.32E-02
NW	8	4.18E-02	3.56E-02	4.04E-02	6.08E-02	4.46E-02
NNW	4	7.71E-02	7.09E-02	6.28E-02	6.76E-02	6.56E-02
NNW	8	4.07E-02	3.45E-02	3.17E-02	4.18E-02	3.71E-02
W	1	1.71E-01	3.11E-01	2.73E-01	1.84E-01	2.35E-01
W	2	9.58E-02	1.65E-01	1.48E-01	1.06E-01	1.30E-01
W	4	6.28E-02	8.72E-02	7.82E-02	6.64E-02	7.27E-02
W	8	3.45E-02	4.15E-02	4.04E-02	4.12E-02	3.94E-02
WSW	4	4.18E-02	7.85E-02	8.21E-02	6.62E-02	6.71E-02
WSW	8	2.30E-02	3.76E-02	4.21E-02	4.12E-02	3.60E-02
SW	1	2.54E-01	4.41E-01	5.57E-01	2.61E-01	3.59E-01
SW	2	1.72E-01	2.42E-01	3.20E-01	1.50E-01	2.21E-01
SW	4	1.07E-01	1.30E-01	1.68E-01	9.31E-02	1.25E-01
SW	8	5.83E-02	6.25E-02	8.58E-02	5.72E-02	6.60E-02
SSW	4	5.66E-02	8.32E-02	6.52E-02	6.62E-02	7.28E-02
SSW	8	3.17E-02	3.58E-02	4.35E-02	4.12E-02	3.50E-02
S	1	5.05E-01	6.70E-01	6.22E-01	3.95E-01	5.48E-01
S	2	2.86E-01	3.45E-01	3.31E-01	2.25E-01	2.97E-01
S	4	1.71E-01	1.76E-01	1.73E-01	1.0E-01	1.65E-01
S	8	9.08E-02	8.24E-02	6.80E-02	8.58E-02	8.68E-02
SSF	4	1.18E-01	1.20E-01	1.04E-01	1.35E-01	1.19E-01
SSF	8	6.08E-02	5.55E-02	5.16E-02	7.99E-02	6.20E-02
SF	1	9.34E-01	5.24E-01	6.64E-01	8.13E-01	7.34E-01
SF	2	5.16E-01	2.66E-01	3.45E-01	4.54E-01	3.95E-01
SF	4	2.54E-01	1.31E-01	1.76E-01	2.73E-01	2.19E-01
SF	8	1.52E-01	6.03E-02	8.75E-02	1.61E-01	1.15E-01
ESF	4	3.25E-01	2.38E-01	2.62E-01	3.59E-01	2.96E-01
ESF	8	1.61E-01	1.06E-01	1.26E-01	1.95E-01	1.48E-01

Table 4.1.2.2. Air concentrations of cadmium around Helena smelter area

PARTICLE DIAMETER IS 5.0 MICRONS AND DENSITY IS 7.0 CM/CM³

DIR.	MILES	AIR CONCENTRATION (MICROGRAMS/1000)				AVERAGE
		JAN-MAR	APR-JUN	JUL-SEP	OCT-DEC	
E	1	3.32E-02	3.37E-02	2.59E-02	2.62E-02	2.57E-02
E	2	2.64E-02	1.82E-02	1.54E-02	1.78E-02	1.80E-02
E	4	1.76E-02	5.7CF-03	8.89E-03	1.24E-02	1.09E-02
E	8	6.87E-03	4.75E-03	4.88E-03	7.85E-03	6.09E-03
ENE	4	9.46E-03	7.45E-03	9.27E-03	1.06E-02	9.26E-03
ENE	8	5.47E-03	3.81E-03	5.19E-03	7.14E-03	5.36E-03
NE	1	8.53E-03	1.14E-02	1.09E-02	8.12E-03	9.72E-03
NE	2	7.21E-03	7.28E-03	7.71E-03	7.26E-03	7.39E-03
NE	4	5.74E-03	4.33E-03	5.05E-03	5.95E-03	5.28E-03
NE	8	3.48E-03	2.26E-03	3.06E-03	4.16E-03	3.22E-03
ENE	4	2.58E-03	1.83E-03	1.95E-03	2.92E-03	2.32E-03
ENE	8	1.55E-03	9.54E-04	1.23E-03	1.95E-03	1.42E-03
N	1	6.20E-03	4.86E-03	4.17E-03	6.45E-03	5.47E-03
N	2	5.23E-03	3.55E-03	3.81E-03	5.74E-03	4.52E-03
N	4	4.09E-03	2.46E-03	2.63E-03	4.86E-03	3.5CE-03
N	8	2.47E-03	1.32E-03	1.65E-03	3.39E-03	2.21E-03
NNW	4	2.34E-03	1.31E-03	2.13E-03	2.24E-03	2.01E-03
NNW	8	1.44E-03	6.84E-04	1.31E-03	1.61E-03	1.26E-03
NW	1	2.55E-03	3.45E-03	4.23E-03	3.06E-03	3.41E-03
NW	2	2.84E-03	2.93E-03	3.25E-03	3.27E-03	3.08E-03
NW	4	2.33E-03	2.13E-03	2.24E-03	2.96E-03	2.42E-03
NW	8	1.44E-03	1.2CE-03	1.38E-03	2.14E-03	1.54E-03
NNW	4	2.26E-03	2.06E-03	1.78E-03	1.99E-03	2.02E-03
NNW	8	1.40E-03	1.17E-03	1.06E-03	1.48E-03	1.28E-03
W	1	1.26E-03	5.42E-03	4.23E-03	1.45E-03	3.05E-03
W	2	1.82E-03	3.75E-03	3.25E-03	1.97E-03	2.72E-03
W	4	1.87E-03	2.51E-03	2.24E-03	1.97E-03	2.15E-03
W	8	1.22E-03	1.36E-03	1.35E-03	1.47E-03	1.35E-03
WSW	4	1.24E-03	2.24E-03	2.35E-03	1.96E-03	1.55E-03
WSW	8	8.17E-04	1.26E-03	1.41E-03	1.46E-03	1.24E-03
SW	1	2.37E-03	6.75E-03	9.64E-03	2.28E-03	5.26E-03
SW	2	3.27E-03	5.24E-03	7.14E-03	2.86E-03	4.65E-03
SW	4	3.18E-03	3.75E-03	4.79E-03	2.74E-03	3.61E-03
SW	8	2.04E-03	2.1CE-03	2.87E-03	2.02E-03	2.26E-03
SSW	4	1.65E-03	2.4CE-03	2.42E-03	1.96E-03	2.12E-03
SSW	8	1.13E-03	1.32E-03	1.45E-03	1.46E-03	1.34E-03
S	1	5.59E-03	1.35E-02	1.26E-02	3.68E-03	8.45E-03
S	2	5.84E-03	8.36E-03	7.48E-03	4.33E-03	6.50E-03
S	4	5.04E-03	5.07E-03	4.92E-03	4.14E-03	4.79E-03
S	8	3.14E-03	2.67E-03	2.72E-03	3.04E-03	2.94E-03
SSF	4	3.44E-03	3.45E-03	2.96E-03	3.96E-03	3.45E-03
SSF	8	2.04E-03	1.77E-03	1.68E-03	2.78E-03	2.07E-03
SF	1	1.41E-02	1.15E-02	1.27E-02	9.81E-03	1.20E-02
SF	2	1.13E-02	6.66E-03	8.07E-03	9.27E-03	8.83E-03
SF	4	8.61E-03	3.76E-03	4.98E-03	7.96E-03	6.33E-03
SF	8	5.15E-03	1.51E-03	2.85E-03	5.62E-03	3.88E-03
PSF	4	9.43E-03	6.75E-03	7.36E-03	1.04E-02	8.48E-03
PSF	8	5.30E-03	3.22E-03	3.93E-03	6.67E-03	4.78E-03

Fig. 4.1.2.1. Average deposition of cadmium (5 μ) around East Helena smelter (mg/m²/month).

DANL-DWG 76-1417

Fig. 4.1.2.2. Average air concentration of cadmium (5μ) around East Helena smelter (nG/m³).

JHNL-CWC 76-14174

Fig. 4.1.2.3. Air concentration of cadmium around East Helena smelter area as a function of particle size and distance.

Fig. 4.1.2.4. Deposition of cadmium around East Helena smelter as a function of particle size and distance.

Table 4.1.2.3. Deposition of cadmium around Helena smelter area

AVERAGE YEARLY DEPOSITION (MG/M**2/PCNT)						
DIR.	MILES	C.I	PARTICLE SIZE (MICRONS)			
			1.0	5.0	10.0	20.0
F	1	6.60E-01	6.88E-01	1.42E+00	3.53E+00	1.48E+01
F	2	3.78E-01	3.53E-01	7.36E-01	2.10E+00	5.75E+00
F	4	2.06E-01	2.14E-01	3.61E-01	8.14E-01	1.23E+00
F	8	9.83E-02	1.03E-01	1.89E-01	2.68E-01	3.49E-01
ENE	4	1.68E-01	1.76E-01	3.23E-01	6.73E-01	8.80E-01
ENE	8	8.16E-02	8.58E-02	1.64E-01	2.14E-01	2.51E-01
NE	1	2.13E-01	2.28E-01	6.13E-01	1.46E+00	7.63E+00
NE	2	1.47E-01	1.55E-01	3.33E-01	1.04E+00	2.56E+00
NE	4	9.10E-02	9.56E-02	1.83E-01	3.67E-01	3.85E-01
NE	8	4.51E-02	4.77E-02	9.56E-02	1.13E-01	1.16E-01
NNE	4	3.98E-02	4.18E-02	8.01E-02	1.60E-01	1.67E-01
NNE	8	1.98E-02	2.10E-02	4.23E-02	4.93E-02	5.17E-02
N	1	1.18E-01	1.29E-01	3.64E-01	8.95E-01	5.04E+00
N	2	8.78E-02	9.35E-02	2.11E-01	6.91E-01	1.60E+00
N	4	5.85E-02	6.15E-02	1.20E-01	2.36E-01	2.29E-01
N	8	2.94E-02	3.12E-02	6.46E-02	7.29E-02	7.54E-02
NNW	4	3.36E-02	3.54E-02	6.92E-02	1.36E-01	1.23E-01
NNW	8	1.68E-02	1.79E-02	3.70E-02	4.08E-02	3.74E-02
NW	1	7.35E-02	8.10E-02	2.62E-01	6.11E-01	3.59E+00
NW	2	5.89E-02	6.30E-02	1.46E-01	4.79E-01	1.10E+00
NW	4	3.99E-02	4.21E-02	8.32E-02	1.62E-01	1.35E-01
NW	8	2.02E-02	2.14E-02	4.46E-02	4.82E-02	4.30E-02
NNW	4	3.36E-02	3.53E-02	6.96E-02	1.35E-01	1.12E-01
NNW	8	1.69E-02	1.79E-02	3.71E-02	4.01E-02	3.84E-02
W	1	6.69E-02	7.36E-02	2.35E-01	5.42E-01	3.21E+00
W	2	5.21E-02	5.57E-02	1.30E-01	4.25E-01	9.49E-01
W	4	3.54E-02	3.74E-02	7.37E-02	1.42E-01	1.16E-01
W	8	1.78E-02	1.89E-02	3.94E-02	4.21E-02	4.06E-02
WSW	4	3.21E-02	3.39E-02	6.71E-02	1.29E-01	1.04E-01
WSW	8	1.61E-02	1.71E-02	3.60E-02	3.83E-02	3.64E-02
SW	1	1.14E-01	1.25E-01	3.99E-01	9.24E-01	5.42E+00
SW	2	8.93E-02	9.55E-02	2.21E-01	7.16E-01	1.62E+00
SW	4	5.99E-02	6.34E-02	1.25E-01	2.40E-01	1.99E-01
SW	8	3.01E-02	3.19E-02	6.60E-02	7.09E-02	6.55E-02
SSW	4	3.49E-02	3.68E-02	7.28E-02	1.40E-01	1.12E-01
SSW	8	1.75E-02	1.86E-02	3.90E-02	4.15E-02	3.91E-02
S	1	1.86E-01	2.00E-01	5.48E-01	1.25E+00	6.98E+00
S	2	1.28E-01	1.35E-01	2.97E-01	9.46E-01	2.19E+00
S	4	8.17E-02	8.60E-02	1.65E-01	3.25E-01	3.30E-01
S	8	4.04E-02	4.28E-02	8.68E-02	9.99E-02	1.09E-01
SSF	4	6.05E-02	6.34E-02	1.19E-01	2.42E-01	2.85E-01
SSF	8	2.97E-02	3.13E-02	6.20E-02	7.67E-02	8.87E-02
SE	1	2.64E-01	2.82E-01	7.34E-01	1.75E+00	8.99E+00
SE	2	1.76E-01	1.86E-01	3.95E-01	1.25E+00	3.07E+00
SE	4	1.09E-01	1.15E-01	2.19E-01	4.41E-01	4.92E-01
SE	8	5.42E-02	5.73E-02	1.15E-01	1.38E-01	1.52E-01
ESF	4	1.59E-01	1.66E-01	2.95E-01	6.35E-01	9.48E-01
ESF	8	7.63E-02	7.97E-02	1.45E-01	2.00E-01	2.69E-01

Table 4.1.3.i. Cadmium concentrations in area around East Helena

Cadmium concentration		
	<u>Measured</u>	<u>Calculated</u>
Deposition (mg/m ² /month)	1-4 (1 mile radius of smelter)	0.7 (1.0 μ particle - 1 mile east of smelter)
	0.1-1 (within 60 square miles around East Helena)	0.1-0.3 (1-10 μ particles 8 miles east of smelter)
Air concentration (μ g/100 m ³)	6-29 (within East Helena)	3-7 (1-20 μ particle 1 mile east of smelter)

60 square miles around the smelter, levels of 0.1 to 1 mg/m²/month were measured. At a distance of 8 miles east, the simulated results were 0.1 to 0.3 mg/m²/month for 1-10 μ particles. Measured 24-hour average air concentrations of cadmium at various locations within East Helena ranged from 6.0-29.0 μ g/100 m³, while simulated results at a distance of 1 mile east ranged from 3.0-7.0 μ g/100 m³ for 1-20 μ particles.

Thus, the ATM appears to be giving reasonable results for cadmium concentrations in the area surrounding the East Helena Smelter. These data may be used to estimate annual additions to the soil or depositions on crops at specified locations within an 8 mile radius.

4.2 Terrestrial Transport Model

Starting with given cadmium ground deposition rates, obtained from monitoring data for the U.S. and from the ATM output for East Helena, TERMOD simulated cadmium intake by the average U.S. adult and by an East Helena adult. Pathways considered were air inhalation, milk, beef, and solid-food consumption, and direct deposition on truck crops (uptake of truck crops is after washing which is assumed to remove 75% of the

cadmium).³⁸ The model applies only for soil concentrations of cadmium ≤ 10 ppm. Crop production is reduced for higher soil concentrations (Appendix II).

4.2.1 Input Data

The input to the model is a constant ground deposition rate of $15 \mu\text{g Cd/m}^2\text{-day}$ which corresponds to an average air concentration of $0.025 \mu\text{g/m}^3$ and a deposition velocity of 600 m/day. The air concentration is a typical value for areas near large cities in the U.S.⁹ Therefore, the model predictions are for current, equilibrium (background) concentrations in soil, grass, milk, and beef and current uptakes of cadmium via the pathways considered. A ground deposition of $86 \mu\text{g Cd/m}^2\text{-day}$ at 1 mile east of the smelter was obtained from the ATM output for the East Helena simulation. This ground deposition corresponds to an air concentration of $0.14 \mu\text{g/m}^3$ for a deposition velocity of 600 m/day.

The model was run for two values (10 years and 50 years) for the half-time of cadmium in subsurface soil pool and pasture soil (Appendix II).

A literature survey was made to find reasonable values for all TERMOD input parameters and to insure that the calculated transfer coefficients were reasonable. The only parameter not specifically calculated for cadmium or verified to apply to cadmium was $\tau_{s,p}$, the coefficient associated with transfer from soil surface to subsurface soil pool. A value of 0.0693 days^{-1} was assigned to this coefficient. A sensitivity study was conducted to determine the importance of $\tau_{s,p}$ to model calculations. It was found that model calculations are relatively insensitive to this parameter.

The model simulation began with cadmium present only in the air. Build-up of cadmium in other compartments (from initial values of zero) was calculated as a function of time after this constant cadmium concentration appeared in air. Cadmium concentrations in these compartments were also calculated as a function of time after an impulse source of cadmium was in the air.

4.2.2 Output Data

Equilibrium concentrations in various compartments resulting from ground deposition rates of $15 \mu\text{g Cd/m}^2\text{-day}$ and $86 \mu\text{g Cd/m}^2\text{-day}$ are shown in Table 4.2.2.1.

Table 4.2.2.1. Equilibrium cadmium concentrations in various compartments

Compartment	Equilibrium concentration (ppm)			
	10-year half-time in soil		50-year half-time in soil	
	15 ^a	86 ^a	15 ^a	86 ^a
Surface food	0.07	0.40	0.07	0.40
Subsurface soil pool	0.27	1.55	1.30	7.45
Grass	0.46	2.64	0.51	2.92
Pasture soil	0.24	1.38	1.16	6.65
Milk	0.002	0.011	0.003	0.017
Beef	0.06	0.34	0.07	0.40

^aDeposition rates in $\mu\text{g/m}^2\text{-day}$.

Equilibrium intakes by reference man⁴³ for constant ground deposition rates of $15 \mu\text{g Cd/m}^2\text{-day}$ and $86 \mu\text{g Cd/m}^2\text{-day}$ are shown in Table 4.2.2.2.

Table 4.2.2.2. Equilibrium intakes by reference man

Pathway or Compartment	Intake ($\mu\text{g/day}$)			
	10-year half-time in soil		50-year half-time in soil	
	15 ^a	86 ^a	15 ^a	86 ^a
Subsurface soil pool	67	384	326	1870
Surface food	18	103.1	18	103
Beef	20	114.6	22	126
Milk	2.3	13.2	2.6	15
Air inhalation	0.5 ^b	3 ^c	0.5 ^b	3 ^c
Total	108	618	369	2115.0

^aDeposition rates in $\mu\text{g/m}^2\text{-day}$.

^bAir concentration $0.025 \mu\text{g/m}^3$.

^cAir concentration $0.14 \mu\text{g/m}^3$.

4.2.3 Comparison with Measurements

A ground deposition rate for cadmium of 1-4 mg/m^2 -month or 30-133 $\mu\text{g}/\text{m}^2$ -day occurred on a 1.5 sq-mile area around the East Helena industrial complex. Equilibrium uptake rates and plots given here apply for a deposition rate of 15 $\mu\text{g}/\text{m}^2$ -day. Thus, TERMOD predicts an equilibrium uptake of cadmium by a "fencepost" reference man⁴³ in East Helena of $[(30-133)/15] \times 107.8 = 216-956 \mu\text{g}/\text{day}$ for a 10-year half-time in soil and $[(30-133)/15] \times 369 = 738-3272$ for a 50-year half-time in soil. The range calculated from food concentrations was 197-811 $\mu\text{g}/\text{day}$ for an average East Helena adult with 876 as the reasonable, maximum daily uptake. The corresponding equilibrium subsurface soil pool concentration would be $[(30-133)/15] \times 0.27 = 0.54-2.4 \text{ ppm}$ for a 10-year half-time in soil and $[(30-133)/15] \times 1.30 = 2.6-11.5 \text{ ppm}$ for a 50-year half-time in soil. Note that the model will not apply for cadmium concentrations in soil $>10 \text{ ppm}$ (Appendix II). The ground deposition rate of 86 $\mu\text{g Cd}/\text{m}^2$ -day calculated by the ATM falls about midway in the range determined by actual measurements.

Figures 4.2.3.1 through 4.2.3.6 show calculated cadmium uptake by reference man⁴³ under a variety of conditions as described in each figure.

4.3 Calculation of CUMEX Index

A CUMEX index for the maximally exposed individual in the area surrounding the smelter complex in East Helena, Montana, is calculated based on the following data and assumptions (see Appendix II):

1. particle size = 10 μ ,
2. deposition velocity = 600 m/day,
3. 100% of diet obtained locally,
4. 2112 $\mu\text{g Cd}/\text{day}$ in diet from 0.14 $\mu\text{g Cd}/\text{m}^3$ in air = 15,000 $\frac{\mu\text{g Cd}}{\text{day}}$ in diet from 1 $\mu\text{g}/\text{m}^3$ in air (50-year cadmium half-time in soil),

Fig. 4.2.3.1. Intake rate of cadmium vs time after an impulse, ground deposition of 15 $\mu\text{g}/\text{m}^2$. Intake is by reference man⁴³ whose diet consists of 250 dry grams of vegetables/day, 1.0 l milk/day, 300 wet grams of beef/day and a total, solid food intake (less fish, beef, and vegetables) of 2500 wet grams/day. The half-time for loss of cadmium from the subsurface soil pool and pasture soil was 10 years.

Fig. 4.2.3.2. Intake rate of cadmium vs time after an impulse, ground deposition of $15 \mu\text{g}/\text{m}^2$. Intake is by reference man.⁴³ The half-time for loss of cadmium from the subsurface soil pool and pasture pool was 50 years.

Fig. 4.2.3.3. Percent of the daily cadmium intake from each pathway as a function of time after an impulse, ground deposition of $15 \mu\text{g}/\text{m}^2$. Intake is for reference man.⁴³ The half-time for loss of cadmium from the subsurface soil pool and pasture soil was 10 years. See Fig. 4.2.3.1 for intake rates in $\mu\text{g}/\text{day}$.

Fig. 4.2.3.4. Percent of the daily cadmium intake from each pathway as a function of time after an impulse, ground deposition of $15 \mu\text{g}/\text{m}^2$. Intake is for reference man.⁴³ The half-time for loss of cadmium from the subsurface soil pool and pasture soil was 50 years. See Fig. 4.2.3.2 for intake rates in $\mu\text{g}/\text{day}$.

Fig. 4.2.3.5. Percent of the total, 50-year intake of cadmium through each pathway up to time t after an impulse, ground deposition of $15 \mu\text{g}/\text{m}^2$. Also, multiplication by 1.05 converts these plots to the intake rate ($\mu\text{g}/\text{day}$) of cadmium as a function of time after a constant deposition source of $15 \mu\text{g}/\text{m}^2\text{-day}$ first appears. Inputs are for reference man.⁴³ The half-time for loss of cadmium from the subsurface soil pool and pasture soil was 10 years.

Fig. 4.2.3.6. Percent of the total, 50-year intake of cadmium through each pathway up to time t after an impulse, ground deposition of $15 \mu\text{g}/\text{m}^2$. Also, multiplication by 2.12 converts these to the intake rate ($\mu\text{g}/\text{day}$) of cadmium as a function of time after a constant deposition source of $15 \mu\text{g}/\text{m}^2\text{-day}$ first appears. Inputs are for reference man.⁴³ The half-time for loss of cadmium from the subsurface soil pool and pasture soil was 50 years.

5. $1 \mu\text{g Cd/m}^3$ inhaled = $100 \mu\text{g Cd/g}$ in kidney cortex after 50 years,
6. $1 \mu\text{g Cd/day}$ in diet = $1 \mu\text{g}$ in kidney cortex after 50 years,
7. selected limit = $200 \mu\text{g Cd/g}$ wet weight of kidney cortex in 50 years,
8. total weight of kidneys = 300 g, and
9. half-time of cadmium in body = 30 years.

Inhalation and ingestion are the routes of exposure considered, therefore Eq. (1.2.2) was used with $f_{AW}V_Wf_w = kf_2 = 0$.

$$C_A^* = \frac{Q_L}{(V_A f_a + f_{AF} M_F f_w) \tilde{R}}, \quad (4.3.1)$$

where

$$\begin{aligned} Q_L &= 200 \mu\text{g/g wet weight kidney cortex} = 4 \times 10^4 \mu\text{g in total kidneys,} \\ V_A &= 20 \text{ m}^3/\text{day,} \\ f_a &= 0.25 \times 1/3 = 0.083, \\ f_w &= 0.05 \times 1/3 = 0.017, \\ f_{AF} M_F &= 15,086 \frac{\mu\text{g}}{\text{day}} \frac{\text{m}^3}{\mu\text{g}}, \text{ and} \\ \tilde{R} &= 1.2 \times 10^4 \text{ days based upon simple exponential retention with } \bar{t} = 50 \text{ years.} \end{aligned}$$

Thus,

$$C_A^* = 1.3 \times 10^{-2} \frac{\mu\text{g}}{\text{m}^3}.$$

The CUMEX index calculated above represents a cadmium concentration in air low enough to prevent a cadmium related health effect to persons receiving exposure in the study area for a 50-year period. It is a conservative estimate when applied such that if the maximally exposed individual is protected, all others will also be protected. The calculated

CUMEX value of $0.013 \mu\text{g}/\text{m}^3$ is near the average urban cadmium concentration of $0.02 \mu\text{g}/\text{m}^3$ in air in the U.S. (Table 3.4.1) and about 5–20 times lower than levels actually measured in East Helena (0.06 to $0.29 \mu\text{g Cd}/\text{m}^3$). Realistically, no individual is likely, over a 50-year period, to breathe only air containing the maximum concentration of cadmium or eat food produced only at the site of maximum cadmium deposition. Less conservative CUMEX indexes can be calculated if justified by site characteristics. For example, if the area of interest is divided into a grid network and air concentrations are predicted by ATM for each section, individual CUMEX values can be determined for each. An average air concentration could be determined for the area and another CUMEX value calculated. The proportion of total diet produced outside the study area can be considered. Obviously any change in the parameters of environmental or physiological models will affect CUMEX values. The degree of conservatism of resulting values will depend on the degree to which model parameters can be quantified.

CONCLUSION

Application of the CUMEX index to cadmium release from the smelter complex in East Helena, Montana, has illustrated the potential usefulness of this approach; however, humans are rarely if ever exposed to only one pollutant from one source. The ultimate usefulness of CUMEX will be realized when information allows multi-source, multi-pollutant models to be tested. The most sophisticated and developed modeling system must agree with experiment, but in addition, must suggest better ways to obtain data. The ability to predict the health impact of multi-pollutant exposure will improve as data accumulate.

APPENDIX I

MODIFICATIONS OF TERMOD

APPENDIX I. MODIFICATIONS OF TERMOD

TERMOD has been described previously in Refs. 1 and 2. Modifications of the model necessary for CUMEX application follow:

The expression for intake by man of elements from the subsurface soil pool, $\tau_{p,m}$, was rederived so that it would not depend on the biological half-time of the element in man. The new expression for $\tau_{p,m}$ is

$$\tau_{p,m} = 10^3 \frac{T_D C_V}{P_{eq}} \quad (\text{days}^{-1}), \quad (1)$$

where the factor 10^3 is required for conversion of units ($1g = 10^{-3} \text{ kg}$),

T_D = total daily intake of solid food excluding fish and meat (2.5 kg/day);

C_V = average background concentration (wet weight) of the element in harvested, solid food excluding fish and meat (0.05 $\mu\text{g/g}$ for cadmium), and

P_{eq} = average background content of element in subsurface soil pool per man. For cadmium the value is,

$$\begin{aligned} P_{eq} &= 0.5 \mu\text{g/g} \times 1.4 \text{ g/cm}^3 \times 20 \text{ cm} \times 10^7 \text{ cm}^2 \\ &= 1.4 \times 10^8 \mu\text{g} \end{aligned}$$

where 0.5 $\mu\text{g/g}$ = average background cadmium concentration in subsurface soil,

1.4 g/cm^3 = soil density,

20 cm = plow layer depth, and

$1 \times 10^7 \text{ cm}^2$ = area of soil required to supply food for one man.

An expression was derived for the transfer of cadmium from pasture soil to pasture grass, $\tau_{r,g}$.

$$\tau_{r,g} = 0.1 \frac{V_c C_p}{A_g C_s \rho d} \quad (\text{days}^{-1}), \quad (2)$$

BLANK PAGE

where the factor 0.1 is required for conversion of units ($10^3 \text{ g/kg}/10^4 \text{ cm}^2/\text{m}^2$),

V_C = dry weight grass consumption per day by a cow (10 kg/day),

C_p = average, background concentration (wet weight) of cadmium in grass ($1.0 \text{ } \mu\text{g/g}$),

A_g = pasture area per cow (10^4 m^2),

C_s = average, background concentration of cadmium in soil ($0.5 \text{ } \mu\text{g/g}$),

ρ = density of soil (1.4 g/cm^3), and

d = plow layer depth (20 cm).

APPENDIX II. CADMIUM REVIEW

Industrial Sources

Total release of cadmium to the environment from all sources for 1968 was estimated to be about 2.50×10^6 kg to 3.58×10^6 kg. The actual release may have been greater because an additional 4.99×10^6 kg was either considered as waste or put into permanent use with final disposal unknown.⁷

As expected, cadmium is released to some degree from industries involved in its production or use.^{7,9} It enters the environment as a by-product in the refining of zinc and other metals. Combustion of fossil fuel adds to the burden since cadmium is present as a trace contaminant, as does use of cadmium in various industrial products such as alkaline batteries, copper alloys, paints, and plastics.⁵² Both airborne and waterborne cadmium can increase concentrations of cadmium in soil and vegetation, in animal products and humans.

Accumulation of cadmium in the environment may be expected to continue since projections show an 86% increase in zinc consumption, a 127% increase in cadmium use and an increase in coal use in steam plants for power production.^{7,23} With increased use of coal, large increases in emissions of trace toxic metals will occur in spite of improved emissions control technology. Electrostatic precipitators or scrubbers, which efficiently remove large fly ash particles, are less efficient for vapors and fine particles. A relationship has been shown between concentration and particle size for some trace elements in fly ash. Lead, antimony, cadmium, selenium, arsenic, chromium, and zinc show clear increases in concentrations as the fly ash particle size decreases.^{24,25} Also because of a slower deposition velocity, fine particles are more widely distributed in the environment. The highest concentrations of these trace elements are found in particles of a size which deposit in pulmonary regions of the respiratory system where absorption is greatest.⁷

Agricultural Sources

Widespread human cadmium exposure may result from certain farming practices in food production. Food is, in general, the major source of cadmium intake by man since plants may absorb toxic heavy metals along with mineral elements required for growth. Some grains and vegetables produced with heavy applications of commercial fertilizers contain relatively high concentrations of cadmium. Schroeder and Balassa found 9 to 36 ppm cadmium in phosphate fractions of five fertilizers.²⁶ Superphosphate contained 7.25 ppm cadmium. Countrywide, 39,902,000 tons of commercial fertilizers were applied in 1970, increasing to 41,582,000 tons in 1972.²⁷ Grains, which are often fertilized with phosphate fertilizers, may accumulate an appreciable amount of cadmium (Table 3.4.2), a situation particularly important because of high wheat consumption by humans. Schroeder et al.,²⁸ however, found no significant difference in cadmium content of fertilized and unfertilized plants and, based on a survey of cadmium in wheat from rural areas, Huffman and Hodgson²⁸ concluded that superphosphate fertilizers did not affect cadmium levels in plants.

Use of sewage sludges as soil conditioners can contribute to cadmium soil burdens. Linnman et al. determined cadmium uptake by wheat grown in soil treated with amounts of sewage sludge (cadmium content 10 ppm) varying from 0 to 175 tons/hectare.¹¹ Cadmium uptake increased with increasing sludge addition at all but the highest level used, reaching a maximum of 257 ± 9 ppb. Strenström and Lönsjö obtained similar results in an experiment using sewage sludge containing 5.2 ppm radiolabeled cadmium.¹² Cadmium uptake by wheat increased with increasing sludge addition with 0.0033% of cadmium originally in sludges appearing in mature crops, indicating that even a limited use of sludge may eventually produce concentrations of cadmium in agricultural products which are intolerable in light of the World Health Organization recommendations for human intake of 70 $\mu\text{g/day}$.¹⁷ Strenström and Vohter made similar observations on cadmium accumulating in arable land from phosphatic fertilizers, disagreeing with Schroeder et al.³⁰

Munshower found only a weak correlation between plant and soil cadmium concentrations, possibly because cadmium soil reservoirs are not the only source of cadmium for plants.¹⁰ Another source is particulate contamination of leaf surfaces from atmospheric pollution. Cadmium-containing insecticide or fungicide sprays also contribute.³¹ In controlled experiments, cadmium concentration of aboveground plant parts was a function of cadmium concentration of the root medium where no atmospheric source was available.³² Uptake of cadmium by plants from soil in the natural environment is affected by plant species, soil type, pH, presence of other metals and organic matter, moisture, farming practices, and location in relation to pollutant sources.^{7,9,33,34}

Cadmium is a constituent of compounds routinely used as fungicides on turf and occasionally as helminth suppressants in cattle.^{35,36} Use of CdCl_2 has been suggested to counteract tobacco mosaic virus.³⁷ Tobacco is a plant which accumulates cadmium. Little and Martin have shown that only 28% of airborne cadmium deposited on elm trees leaves can be removed by washing, indicating penetration and uptake of cadmium by the leaf.³⁸ A study by Ross and Stewart indicated a translocation of cadmium to apples from trees which had received a cover spray of cadmium chloride each year for three years.³¹ Foliage residues slowly declined throughout the season and cadmium accumulated in apples as they matured.

It has been found recently that tobacco smoking contributes to daily intake of cadmium. Smoking one to two packs of cigarettes per day will increase daily intake of cadmium by 2 to 4 μg .^{7,41} Cadmium in tobacco is derived from soil, fertilizers, and agricultural sprays. For this reason, agricultural practices in the tobacco industry, as well as in the food growing industry, must be carefully evaluated in terms of their ultimate impact on humans.

Environmental Concentration of Cadmium

Concentrations in air

Air in urban areas contained cadmium at an average level of 0.02 $\mu\text{g}/\text{m}^3$. The average nonurban air concentration is 0.003 $\mu\text{g}/\text{m}^3$.⁹ Con-

centrations of cadmium near cadmium emitting industrial facilities have been found to reach levels of $0.2 \mu\text{g}/\text{m}^3$.⁴¹ Assuming an inhalation rate of $20 \text{ m}^3/\text{day}$ ⁵³ and an absorption rate of 25%, daily cadmium uptake from inhalation would approach a maximum of $1 \mu\text{g}$. This $1 \mu\text{g}$ amount is a relatively small fraction (<1%) of the amount calculated as uptake from ingestion of food items. Industrial exposure is likely to be much greater in some occupations because the Threshold Limit Value (TLV) for industrial exposure in the United States is $100 \mu\text{g}/\text{m}^3$ for cadmium fumes.^{18,19} If we assume an inhalation rate of $10 \text{ m}^3/8 \text{ hr}$ working day and that 25% of the inhaled cadmium is absorbed, we can arrive at a daily uptake value of $250 \mu\text{g}$. An occupational exposure of this magnitude would be of extreme significance and the TLV itself much too high. Assuming an excretion rate of 0.005%/day and 224 workdays/year along with the above assumptions, the necessary cadmium concentration in industrial air for exposed workers to accumulate a critical cadmium concentration in renal cortex of $200 \mu\text{g}/\text{g}$ wet weight at age 50 through inhalation would be $6.5 \mu\text{g}/\text{m}^3$. For a 50% pulmonary absorption it would be $3.3 \mu\text{g}/\text{m}^3$. Recently the American Conference of Governmental and Industrial Hygienists (ACGIH) has recommended a reduction in the TLV for cadmium to $50 \mu\text{g}/\text{m}^3$.¹⁹ The ACGIH recommendation was set to avoid pulmonary effects and did not consider long term cadmium damage to kidney tissue. An examination of daily intake values which will result in renal damage over a period of 50 years indicates that the $50 \mu\text{g}/\text{m}^3$ occupational exposure level could produce critical levels of cadmium in kidney tissue even without consideration of other exogenous sources of cadmium.

Concentrations in water

Surface waters can be contaminated by cadmium from mine wastes, effluents from electroplating facilities, airborne particulate deposition, fertilizers and agricultural wastes. In addition, cadmium content of tap water is often increased by contact with galvanized pipes. The permissible level of 0.01 ppm cadmium established by the Public Health Service is used as the limiting value for most considerations.²⁰ A study conducted by McCabe et al. of 969 community water systems showed

three systems to be in excess of the limiting value.⁴² The maximum concentration was 0.11 ppm. At an average daily water consumption of 1.2 liters,⁴³ intake of cadmium from water could range up to about 132 µg/day. If we assume that cadmium concentration in drinking water is at the permissible level of 0.01 ppm, daily intake from water will be 12 µg.

Concentrations in food

Normal background levels for cadmium in foods are usually 0.05 µg/g wet weight or less.⁷ However, some plants and animals accumulate more cadmium than others. In calculating daily per capita intake of cadmium via foods, it is, therefore, more realistic to place foods into a number of different categories. Within a given food category, there can be substantial differences in cadmium concentration among various food items. Leafy vegetables such as spinach accumulate more cadmium than root vegetables such as carrots. Carrots, in turn, accumulate more cadmium than above-ground vegetables such as string beans.³⁹ Fulkerson and Goeller reported that cadmium concentrations may vary from 0.009 to 0.128 µg/g for vegetables which are most often eaten.⁷ The average cadmium concentration of Great Lakes fish is 0.044 µg/g wet weight while the average cadmium concentration of fish from 49 other bodies of fresh water is 0.02 µg/g. Bioaccumulation factors for cadmium in shellfish are 20 times greater than those for fresh water fish.^{7,40} Cadmium concentrations vary from 0.1 to 7.8 µg/g wet weight in oysters from the East Coast of the U.S. and from 0.2 to 2.1 µg/g in oysters from the West Coast.⁹

Cadmium Metabolism in Humans

Intake and absorption

The two main human exposure modes to cadmium are through ingestion and inhalation.^{7,9} Absorption from the gut is influenced by several factors. In general, absorption of cadmium from the G.I. tract has been

reported to be about 5 to 6% with the remainder eliminated primarily in the feces. However, an uptake two or more times greater may occur in individuals with a dietary protein deficiency or with a diet low in calcium.

Except for inhalation of cadmium fumes by certain industrial workers, the general population is exposed to airborne cadmium in the form of aerosols. Deposition in the lung will be related to airborne particle size, varying from 10 to 50% for particles with diameters from 0.01 to 5 μ .⁴⁴ Absorption via the respiratory system is generally considered to be approximately 25%.^{7,9} For nonoccupationally exposed persons, cigarette smoking may be the chief source of inhaled cadmium.⁹ There appears to be little danger to the general population from exposure to inhaled cadmium.

Transport and distribution

Whatever the cadmium exposure modes, it will first appear in the bloodstream. Within a few hours there will be a decrease in plasma cadmium levels and an increase in cadmium associated with red cells.⁹ Blood concentrations decrease after exposure has ended so that elevated levels of cadmium in blood generally reflect recent exposure. Nordberg et al. found that erythrocytic cadmium was in a fraction corresponding to that of the metal-binding protein metallothionein.⁴⁵ Metallothionein (the protein thionine plus a metal) binds cadmium, zinc, mercury, and small amounts of copper and iron due to a high cysteine content.⁹

As blood cadmium levels decrease, there is an initial increase in liver concentration. With increasing time after exposure, kidney levels increase and eventually exceed liver concentrations. Even at low levels of continuous exposure cadmium accumulates in the renal cortex, because excretion of the absorbed metal is on an average very low (about 0.005% of body burden per day).¹⁹ Cadmium also accumulates to some extent in pancreas and testes.^{7,9}

Other metals and compounds can influence metabolism of cadmium. Zinc and cadmium are mutually antagonistic.^{9,28} Presence of calcium will decrease absorption of ingested cadmium.⁴⁶ Selenium dioxide,

although increasing uptake of cadmium in testes, protected them from damage.⁴⁷ It also caused lower levels of cadmium in liver and pancreas while not affecting kidney levels. Various chelating agents can affect cadmium distribution and toxicity.⁹ Metallothionein synthesis is apparently induced by cadmium and accumulation by kidneys may be due to reabsorption of metallothionein in renal tubules.⁹

When exposure levels remain constant, increase in renal cadmium concentrations is almost linear from ages 5 to 50 years.⁹ Urinary excretion of cadmium is very low until cadmium reaches a critical level in kidneys causing renal dysfunction and increased cadmium excretion. The amount of cadmium excreted in feces, amounting to 90 to 95% of the total excreted, represents ingested cadmium which was not absorbed from the G.I. tract.

Reference man⁴³ contains on an average about 30 mg of cadmium.⁹ About 50% of the total body burden is in liver and kidney with about 33% in kidney alone. The kidney weight of reference man is 300 g. It has been estimated that renal cortex levels are 30% higher in smokers who smoke a pack of cigarettes a day for 35 years than in nonsmokers.^{9,13}

Human Health Effects

Biological response to a pollutant burden may fall into one of five categories: The pollutant burden (1) may not be associated with a distinct physiological change, (2) may be associated with a change of uncertain significance, (3) may be associated with physiologic changes which are indicators of specific disease states, (4) may be associated with morbidity, or (5) may be associated directly with mortality.⁴⁸

In discussing health effects of cadmium exposure, we usually describe gross, easily recognizable responses of the main target organ in relation to dose. Subtle responses at lower levels are not yet fully understood, although there is increasing evidence that such responses occur. Moreover, each toxic pollutant cannot be treated as an isolated insult since exposure to a number of different pollutants simultaneously

may produce a synergistic effect. This fact should be taken into account whenever possible in evaluating each particular pollutant.

Some tissue and organ responses to a given burden of cadmium can be described. In lung, cadmium attacks pulmonary epithelium and damages alveolar walls, producing bronchitis and emphysema.^{49,50} In occupational situations, high concentrations of cadmium oxide fumes (1 mg/m^3) inhaled for as little as 8 hours are considered immediately dangerous.^{9,40} Exposure for longer periods of time to lower concentrations, less than 0.1 mg/m^3 of cadmium compounds, can cause chronic pulmonary disorders.⁹ In the G.I. tract, cadmium decreases absorption of such essential elements as calcium, copper, and iron.⁴¹ Concentrations of 13 to 15 mg Cd/l of drinking fluid produces immediate vomiting and gastrointestinal cramps. Cadmium has been shown to affect hematopoiesis probably because of a deficient absorption of iron from food. This effect can result in iron-deficiency anemia.⁴¹ In the liver, cadmium may cause morphological damage as well as changes in activity of certain enzymes. This may occur for long-term exposures at moderate levels and may not show up in clinical diagnostic tests for liver damage.⁴¹ Kidneys accumulate cadmium under conditions of low-level long-term exposure. Renal dysfunction may result if cadmium in the cortex exceeds $200 \text{ } \mu\text{g/g}$ wet weight.⁹ The chief manifestation of renal dysfunction is proteinuria and tubular damage.^{7,9,41,49} It may also be significant that immunoglobulins are excreted during proteinuria. Cadmium accumulates in the interstitial tissue of testicles and testicular necrosis may result.⁴⁷ The activity of testosterone may be decreased during long-term cadmium exposure.⁴¹ High concentrations of cadmium in the pancreas found during autopsy of cadmium workers indicate the possibility of disturbed function in this organ following exposure to cadmium.⁹

The World Health Organization is now conducting a survey on cardiovascular disease and trace elements in tissue and in the environment. Enough evidence has been accumulated to indicate a positive correlation between trace elements and hypertension.⁵¹ This effect has been thought to arise at concentrations of cadmium in kidneys below those producing obvious signs of toxicity.¹¹

Cadmium has also been shown to produce effects at the molecular level at concentrations below which any obvious symptoms would be exhibited. Among these are uncoupling of oxidative phosphorylation and cadmium exchange with zinc in zinc-dependent enzyme systems.^{7,9,49}

REFERENCES

1. R. S. Booth, S. V. Kaye, and P. S. Rohwer, "A Systems Analysis Methodology for Predicting Dose to Man from a Radioactively Contaminated Terrestrial Environment," *Proceedings of Third International Symposium on Radioactivity*, AEC-CONF-710501, (May 10-12, 1971).
2. G. G. Killough and L. R. McKay, *A Methodology for Calculating Radiation Dose from Radioactivity Released to the Environment*, ORNL-4992 (March 1976).
3. National Science Foundation, *Chemicals and Health*, Report of the Panel on Chemicals and Health of the President's Science Advisory Committee, Science and Technology Policy Office, Washington, D.C. (September 1973).
4. M. T. Mills and M. Reeves, *A Multi-Source Atmospheric Transport Model for Prediction of Trace Contaminants*, ORNL/NSF/EATC-2 (October 1973).
5. W. V. Culkowski and M. R. Patterson, *A Comprehensive Atmospheric Transport and Diffusion Model*, ORNL/NSF/EATC-17 (April 1976).
6. S. E. Lindberg, A. W. Andren, R. J. Raridon, and W. Fulkerson, "Mass Balance of Trace Elements in Walker Branch Watershed - The Relation to Coal Fired Steam Plants," *Environ. Health Perspectives* 17: 9-18 (1975).
7. W. Fulkerson and H. E. Goeller (eds.), *Cadmium, the Dissipated Element*, ORNL/NSF-EP-21 (1973).
8. Environmental Protection Agency, U.S., *Billings Valley, Montana, Area Environmental Pollution Study*, Office of Air Programs Publication No. AP-91, Research Triangle Park, North Carolina (1972).
9. L. Friberg, M. Piscator, G. F. Nordberg, and T. Kjellström, *Cadmium Toxicology*, 2nd ed., Chemical Rubber Company, Cleveland, Ohio, 1974.
10. F. Munshower, *Cadmium Concentration and Uptake in a Grassland Ecosystem in the Billings Valley, Montana*, Ph.D. Dissertation, University of Montana, 1972.
11. L. Linnman, A. Andersson, K. O. Nilsson, B. Lind, T. Kjellström, and L. Friberg, "Cadmium Uptake by Wheat from Sewage Sludge Used as a Plant Nutrient Source," *Arch. Environ. Health* 17: 45-47 (1973).

BLANK PAGE

12. T. Strenström and H. Lönsjö, "Cadmium Availability of Wheat: A Study with Radioactive Tracers Under Field Conditions," *Abmio* 3(2): 87-90 (1974).
13. T. A. Kjellström, "A Mathematical Model for the Accumulation of Cadmium in Human Kidney Cortex," *Nord. Hyg. Tidskr.* 53(2): 111-119 (1971).
14. R. S. Booth, "A Systems Analysis Model for Calculating Radionuclide Transport Between Receiving Waters and Bottom Sediments," Chapter 7, pp. 133-164 in *Environmental Toxicity of Aquatic Radionuclides: Models and Mechanisms*, ed. by Morton W. Miller and J. Newell Stannard, Ann Arbor Science Publishers Inc., Ann Arbor, Michigan, 1976.
15. U.S. Department of Agriculture, Agricultural Statistics, *Total and per Capita Civilian Consumption of Foods, 1954-1970*, compiled by the U.S. Dept. of Agriculture. Published by the Government Printing Office, Washington, D.C., 1971, pp. 48, 203, 205, 206, 259, 287, 588.
16. G. K. Murthy, U. Rhea, and J. T. Peeler, "Levels of Antimony, Cadmium, Chromium, Cobalt, Manganese, and Zinc in Institutional Diets," *Environ. Sci. and Tech.* 5: 436-442 (1971).
17. World Health Organization, *Evaluation of Certain Food Additives and the Contaminants Mercury, Lead and Cadmium*, W.H.O. Tech-Rep. Ser. 505 (1972).
18. American Conference of Governmental and Industrial Hygienists, *Documentation of the Threshold Limit Values for Substances in Workroom Air*, 3rd ed., p. 35, American Conference of Government Industrial Hygienists, Cincinnati, Ohio, 1971.
19. American Conference of Governmental and Industrial Hygienists, *TLV's for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1973*, pp. 12, 36, American Conference of Governmental and Industrial Hygienists, Cincinnati, Ohio, 1973.
20. U.S. Department of Health, Education, and Welfare, *Public Health Service Drinking Water Standards*, PHS Pub. No. 956, Washington, D.C., 1962.
21. U.S. Department of Commerce, "Summary of Hourly Observations, Helena, Montana," *Climatology of the United States*, No. 30-24, U.S. Department of Commerce, Washington, D.C., 1956.
22. National Oceanographic and Atmospheric Administration, *Climates of the States*, Vol. 2, p. 755, Water Information Center, National Oceanographic and Atmospheric Administration, Port Washington, New York, 1974.

23. F. Harris, A. Andren, and G. Henderson, "Trace Elements from Coal-Fired Plants," *Oak Ridge National Laboratory Review*, pp. 20-24, Spring, 1975.
24. D. H. Klein et al., "Pathways of 37 Trace Elements through Coal-Fired Power Plants," *Environ. Sci. and Tech.* 9: 973-979 (1975).
25. R. L. Davison, D.F.S. Natusch, and J. R. Wallace, "Trace Elements in Fly Ash. Dependence of Concentration on Particle Size," *Environ. Sci. and Tech.* 8: 1107-1113 (1974).
26. H. A. Schroeder and J. J. Balassa, "Cadmium: Uptake by Vegetable from Superphosphate in Soil," *Science* 140: 819-820 (1973).
27. U.S. Department of Commerce, *Statistical Abstracts of the U.S., National Data Book and Guide to Sources*, U.S. Department of Commerce, Social and Economic Statistics Administration Bureau of the Census, Washington, D.C., 1974.
28. H. A. Schroeder, A. P. Nason, I. H. Tipton, and J. J. Balassa, "Essential Trace Elements in Man: Zinc in Relation to Environmental Cadmium," *J. Chronic Dis.* 20: 179-210 (1967).
29. E.W.D. Huffman, Jr., and J. F. Hodgson, "Distribution of Cadmium and Zinc/Cadmium Ratios in Crops from 19 States East of the Rocky Mountains," *J. Environ. Qual.* 2(2): 289-291 (1973).
30. T. Strenström and M. Vohter, "Cadmium and Lead in Swedish Commercial Fertilizers," *Ambio* 3(2): 91-92 (1974).
31. R. G. Ross and D.K.R. Stewart, "Cadmium Residue in Apple Fruit and Foliage Following a Cover Spray of Cadmium Chloride," *Can. J. Plant Sci.* 49: 49-52 (1969).
32. M. K. John, "Cadmium Uptake by Eight Food Crops as Influenced by Various Soil Levels of Cadmium," *Environ. Pollut.* 4: 7-15 (1973).
33. M. K. John, "Uptake of Soil-Applied Cadmium and its Distribution in Radishes," *Can. J. Plant Sci.* 52: 715-719 (1972a).
34. M. K. John, "Factors Affecting Plant Uptake and Phytotoxicity of Cadmium Added to Soils," *Environ. Sci. and Tech.* 6(12): 1005-1009 (1972b).
35. G. McGrath, "Chemicals for Plant Disease Control," *Agri. Chem.* 10: 18-37 (1964).
36. K. K. Wychoff and J. E. Guthrie, "Methods of Eliminating Helminths with Cadmium Compounds," *U.S. Patent Office Gaz.* 801: 295 (1965).

37. M. Ulrychová-Zelinková, "Cadmium Ions as Inhibitors of Tobacco Mosaic Virus," *Biol. Plantarum* 2(2): 135-141 (1959).
38. P. Little and M. H. Martin, "A Survey of Zinc, Lead, and Cadmium in Soil and Natural Vegetation Around a Smelting Complex," *Environ. Pollut.* 3: 241-254 (1972).
39. D. D. Hemphill, "Availability of Trace Elements to Plants with Respect to Soil-Plant Interaction," *N.Y. Acad. of Sci. Ann.* 199: 46-51 (1972).
40. A. Ishizaki, M. Fukushima, and M. Sakamoto, "Distribution of Cadmium in Biological Materials, Part 2. Cadmium and Zinc Contents of Foodstuffs," *Jap. J. Hyg.* 25: 207-222 (1970).
41. G. F. Nordberg, "Health Hazards of Environmental Cadmium Pollution," *Ambio* 3(2): 55-56 (1974).
42. L. J. McCabe, J. M. Symons, R. D. Lee, and G. G. Roebuck, "Survey of Community Water Supply Systems," *J. Amer. Water Works Assoc.* 62: 670 (1970).
43. International Commission on Radiological Protection, *Report of the Task Group on Reference Man*, ICRP Publication No. 23, Pergamon Press, New York, 1975.
44. Task Group on Lung Dynamics, "Deposition and Retention Models for Internal Dosimetry of the Human Respiratory Tract," *Health Phys.* 10: 173-207 (1966).
45. G. F. Norberg, M. Piscator, and B. Lind, "Distribution of Cadmium among Protein Fractions of Mouse Liver," *Acta Pharmacol. Toxicol.* 22: 456 (1971).
46. H. A. Schroeder, A. P. Nason and J. J. Balassa, "Trace Metals in Rat Tissues as Influenced by Calcium in Water," *J. Nutr.* 93: 331-336 (1967).
47. S. A. Gunn, T. C. Gould, and W.A.D. Anderson, "Mechanisms of Zinc, Systeine, and Selenium Protection Against Cadmium-Induced Vascular Injury to Mouse Testes," *J. Reprod. Fertil.* 15: 65-70 (1968).
48. J. F. Finklea, D. J. Hammer, T. A. Hinners, and C. Pinkerton, "Human Pollutant Burdens," *Proceedings of the A.C.S. Symposium on Determination of Air Quality*, pp. 49-55, Los Angeles, California, April 1-2, 1971.
49. G. Buell, "Some Biochemical Aspects of Cadmium Toxicology," *J. Occupational Med.* 17: 189-195 (1975).

50. G. P. Lewis, H. Lyle, and S. Miller, "Association Between Elevated Hepatic Water-Soluble Protein-Bound Cadmium Levels and Chronic Bronchitis and/or Emphysema," *The Lancet* :: 1339-1333 (1969).
51. H. A. Schroeder, "The Role of Trace Elements in Cardiovascular Diseases," *Medical Clinics of North America* 58(2): 381-396 (1974).
52. A. J. Goldberg, *A Survey of Emissions and Controls for Hazardous and Other Pollutants*, EPA-R-4-73-G21, U.S. Environmental Protection Agency (1973).
53. International Commission on Radiological Protection, *Report of ICRP Committee II on Permissible Dose for Internal Radiation*, ICRP Publication 2, Pergamon Press, New York, 1959.
54. E. J. Underwood, (ed.), *Trace Elements in Human and Animal Nutrition*, 3rd ed., pp. 267-280, Institute of Agriculture, University of Western Australia, Nedlands, Western Australia, Academic Press, New York and London, 1971.
55. R. Ratu and A. Sporn, "Contributions to the Determination of Cadmium Supplied by Foods," *Nahrung* 14: 25-31 (1970).