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Cadmium Contamination of the Environment: An Assessment of Nationwide Risk

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16. ABSTRACT

The incidence of cadmium induced harm to human health and aquatic life was evaluated and linked to pathways of exposure and means of disposal. The population's median exposure appears to be 12% of the kidney toxicity threshold. Statistical extrapolations suggest that 1-2 persons/million might exceed a toxic threshold among nonsmokers, and somewhat more among smokers. For the potential of lung cancer, the median ambient inhalation exposure could be projected to yield a lifetime upper-bound risk of 4×10^{-6}.

Data indicate that tobacco smoking and food contribute most of the population's total cadmium burden, and that ambient air inhalation and drinking water contribute much less. The cadmium content of food and tobacco is believed to be related to the cadmium content of topsoil. Most of the cadmium handled by man is likely to be disposed of by landfill burial. Nevertheless, there are some pathways for the addition of cadmium to cropland topsoil. These include phosphate fertilizer, sewage sludge landspreading, emissions deposition, and irrigation water. Modeling suggests a very gradual increase in population exposure due to these pathways.

The extent of any cadmium induced impairment of aquatic life remains uncertain. Point source discharges of cadmium are estimated to be decreasing, however.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS		b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Cadmium	Sludge disposal	Pollutant pathways	13J
Exposure	Phosphates	Soil contamination	06T
Risk	Food contamination		06F
Water pollution	Toxic diseases		
Air pollution	Urologic diseases		
Effluents	Carcinogens		
Waste disposal	Population (statistics)		

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CADMIUM CONTAMINATION OF THE ENVIRONMENT:

AN ASSESSMENT OF NATIONWIDE RISKS

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FOREWORD

Effective regulatory action for toxic chemicals requires an understanding of the human and ecological risks associated with the manufacture, use, and disposal of such chemicals. Assessment of risk requires scientific judgement about the probability of harm to the environment resulting from measured or predicted environmental concentrations. The risk assessment process integrates data on harmful effects and information on exposure. The components of an exposure assessment include evaluations of the environmental sources, exposure pathways, ambient levels, and exposed populations.

This assessment was initiated as part of a program to determine the environmental risks associated with current use and disposal patterns for 65 chemicals or classes of chemicals (expanded to 129 individual "priority pollutants") named in the 1977 Clean Water Act. It includes an assessment of risks to humans and aquatic life, and is intended to serve as a technical basis for identifying unacceptable risks and developing the most appropiate strategy for their mitigation.

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TABLE OF CONTENTS

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Substantial help was provided by individuals who reviewed the first draft of this document (December 1983): James Ryan, Steven Weil, Paul Brown, Thomas Gleason, Ray Morrison, Robert Kellam, Sandra Lee, Charles Spooner, Charles Stephan, Nelson Thomas, and members of the Cancer Assessment Group and Exposure Assessment Group. By reviewing the second draft (November 1984), considerable additional help was provided by James Ryan and Donald MacGregor. Such review does not necessarily imply concurrence. The shortcomings of this assessment, errors in fact or in judgement, are the sole responsibility of the author.

EXECUTIVE SUMMARY

Introduction

- Cadmium is a metal used primarily for corrosion resistant plating, for plastic and pigment formulation, and for batteries. In addition, substantial quantities reach the environment as impurities in other materials, particularly fossil fuels and phosphate fertilizers.

- While cadmium has been studied extensively for several years, and numerous regulatory actions have already been taken, there are some remaining questions and regulatory issues. This assessment has focussed on human, non-occupational exposure, and aquatic life exposure. The results suggest that in the long term, the most potentially bothersome problem may be the possibility of gradually increasing dietary exposure due to accumulating contamination of cropland topsoil.

Human Health Concerns

Exposure and Effects

- The human health effects of concern are kidney dysfunction and (for inhaled cadmium) probable carcinogenicity. Preventing kidney dysfunction has been the basis for existing EPA criteria and regulations.

- A detectable (but not necessarily clinically significant) effect on kidney function is expected to occur when cumulative exposure has raised cadmium concentrations in the renal cortex above approximately 200 µg/g.

- Current median exposure of the population appears to be 12% of the kidney effect threshold. Extrapolations suggest that possibly 1-2 persons per million may exceed this threshold among nonsmokers and somewhat more among smokers. The accuracy of such extrapolations is not certain, however.

- Some evidence suggests that population exposure levels may have been increasing at a rate of more than 1% per year during this century. Other evidence suggests that exposure may not have been increasing. Whether any increase is now occurring is not known.

- Assuming that inhaled cadmium is carcinogenic, and using the EPA Cancer Assessment Group (CAG) evaluation of lung cancer potency, the median inhalation exposure to ambient urban air would be projected to result in an upper bound risk of 4×10^{-6}. Exposure to indoor air contaminated with cadmium from cigarette smoke would pose additional risks.

- The CAG has found no compelling evidence for carcinogenicity of ingested cadmium.

Key Pathways of Human Exposure

- Tobacco smoking and food are the major exposure routes for the population. Ambient air inhalation and drinking water are minor routes. Median exposure via food appears to be 50 fold higher than the combined median exposures via ambient inhalation and drinking water. Vegetables, potatoes, and grains are the largest contributors of cadmium in the average diet.

- Topsoil appears to be the medium potentially most sensitive to contamination because it appears to link directly to the critical food and tobacco exposure routes. As topsoil is also difficult to decontaminate, accumulated contamination of topsoil merits concern.

- Phosphate fertilizer constitutes the largest pathway to topsoil. Emissions deposition and irrigation water are of lesser but somewhat uncertain importance in this respect. In terms of total quantity of cadmium, the landspreading of sewage sludge is also of lesser importance; however, the hazards of such landspreading have been a concern due to a potential for causing very intense contamination of small areas.

- Phosphate minerals mined to produce fertilizer naturally contain elevated but variable levels of cadmium. Commonly used Eastern phosphates are much lower in cadmium than the less commonly used Western phosphates. Any increase in the market share of Western phosphate would increase the size of this large but low intensity pathway.

- RCRA/CWA regulations controlling the landspreading of municipal sewage sludge are rather effective in reducing the risks associated with growing food on such disposal sites. With these regulations in place, the most significant overall effect of pretreatment for important sources (such as metal finishers) may be to reduce the sludge disposal costs for those municipalities that want to landspread their sludge but are constrained by the cadmium limits imposed by the regulation. For municipalities where sludge is landfilled (or possibly ocean dumped or incinerated) rather than landspread, the benefits of pretreatment are less.

- The sale and give-away of sludge is not controlled under current regulations, but is of concern particularly for home gardeners. Contamination of equivalent areas of commercial agricultural plots and home garden plots have different results. The former results in low level exposure for large numbers of people; the latter results in high level exposure for a few people.

- Although the direct inhalation of cadmium in ambient air contributes little to total exposure, the widespread low-intensity deposition of airborne cadmium onto agricultural soils may contribute some additional exposure. The major sources of atmospheric cadmium appear to be coal and oil combustion and some types of metals smelting. Zinc/cadmium smelting no longer appears to be a major source.

- Irrigation water appears to be only a minor source of topsoil contamination overall in this country, although reliable data on many waters are not available.

- Based on currently estimated rates of cadmium deposition onto soils (from phosphate fertilizer, sewage sludge, emissions deposition, and irrigation water), it has been theoretically estimated that population exposure may increase perhaps 70% over a period of several centuries. Such an increase in exposure could be projected to increase the estimated number of persons at risk for cadmium induced kidney dysfunction from the current 1.5 persons per million to 120 persons per million. These projections involve considerable uncertainty, however. Indeed, it is possible that no significant increase may occur.

Other Pathways of Human Exposure

- Recent data show drinking water to be an insignificant exposure route. In the rare cases where the 10 µg/L standard has been violated, it has generally been attributed to corrosion of the distribution system rather than contamination of the raw water by point or nonpoint sources.

- Exposure to waterborne cadmium also occurs by ingestion of shellfish, many of which greatly bioconcentrate metals. While shellfish are too small a portion of the average diet to significantly affect population exposure as a whole, individuals who eat unusual amounts of shellfish may be exposed to 2-4 fold more cadmium than average.

- Cadmium uses in 1981 were as follows:

Metal Plating	34%
Pigments	27%
Batteries	16%
Plastics	15%
Other	8%

The cadmium incorporated into end products is believed to have relatively little exposure potential. Only if such products are disposed of to the 5-10% of municipal refuse that is incinerated, then the cadmium escaping emissions controls would be released to air.

- Nearly all cadmium handled by human society is mined or extracted from rather stable geological formations incidently with other materials (zinc and lead, fossil fuels, and phosphate ores). The bulk of cadmium handled by man is thought to be disposed of by landfill burial. While this matrix is probably not as isolated from the biosphere as that from which the metal was originally extracted, it appears to be substantially safer than the other major modes of release (i.e., to air, water, or topsoil). Nevertheless, while significant exposure to cadmium (and several other toxic metals) via groundwater appears to be rare, the long term behavior of landfilled cadmium is not known with confidence.

Ecological Concerns

- The effects of cadmium on aquatic life have been a concern for EPA's water program. Nevertheless, it is difficult to ascertain confidently how widespread are problems of cadmium induced ecological impairment. The newly revised criteria for protection of aquatic life have the same general magnitude as the human health criteria. The freshwater criterion appears to be exceeded at most a few percent of the time.

- Best Available Technology (BAT) and Pretreatment Standards for Existing Sources (PSES) seem likely to substantially reduce the point source discharge of cadmium from levels thought to be discharged in the late 1970's.

- Effects of cadmium on terrestrial ecosystems have not been a factor in regulatory efforts. The overall concern has been low.

PROGRAMMATIC CONSIDERATIONS

Current Regulatory Status

Several EPA programs have regulations affecting cadmium. The most significant of these include (Kayser et al. 1982):

RCRA/CWA (Resource Conservation and Recovery Act; Clean Water Act) sludge landspreading rules for disposal sites.

RCRA standards for designating certain wastes as hazardous, thereby requiring special precautions for landfilling.

RCRA regulations for generators and transporters of hazardous wastes; standards for storage and disposal.

CWA (Clean Water Act) pretreatment and effluent standards for a number of industries, including metal finishing.

CWA water quality criteria for protection of aquatic life and human health (advisory).

CWA ocean dumping regulations.

CWA control of discharged pollutants through NPDES permits.

CAA (Clean Air Act) New Source Performance Standards and State Implementation Plans controlling particulate emissions (thereby indirectly affecting cadmium).

SDWA (Safe Drinking Water Act) maximum contaminant level for drinking water: 10 ug/L.

SDWA underground injection control requirements.

FIFRA (Federal Insecticide, Fungicide, and Rodenticide Act) rebuttable presumption against registration (RPAR) of cadmium pesticides.

Issues on Problem Assessment and Regulatory Strategy

The single most important goal of a regulatory strategy for cadmium would appear to be to prevent any substantial increase in the accumulation of cadmium in topsoil. While other goals are also apparent, the agency's regulatory actions to date have been compatible with each other. Generally the safest means of disposal appears to be landfill burial. Fulfilling the need to use sewage sludge as a soil enhancing resource is thus contingent on continuing actions to prevent serious contamination of sewage sludge.

Although a number of possible new regulatory initiatives could be identified, the most important regulatory questions appear to be the following:

1) Should EPA regulate the sale and give-away of severely contaminated sewage sludge? The existing RCRA/CWA landspreading regulations apply to disposal sites, not to marketing and distribution programs. Developing additional CWA 405 regulations intended to control this potentially hazardous pathway continues to be the first priority of the EPA Sludge Task Force.

2) Should EPA regulate cadmium under CAA Section 112? This decision, called for by Section 122(a), has not yet been made. Some factors affecting the decision include: (a) Cadmium has been judged to be a lung carcinogen. (b) Widespread, low-level deposition of emissions onto cropland soils can be expected to increase dietary exposure slightly; nevertheless, imminent hazards may not be possible to identify. (c) Many formerly important cadmium emission sources are now tightly controlled through limitations on particulate emissions; the potential for substantial additional reductions has become more limited.

3) Should EPA take any action to encourage the substitution of low cadmium Eastern phosphate in place of the small volume of high cadmium Western phosphate used for fertilizer? A significant reduction in amount of cadmium reaching topsoil would result from such an action; however, demonstrating an immediate hazard by this pathway would probably be dubitable. Rather, the concern is long term, and may merit further scrutiny in future decades.

4) Should EPA take any action to ban uses of cadmium in pigments, plastics batteries, or metal plating (under TSCA), or in pesticides (under FIFRA)? While such actions have been considered for some time, an unambiguous need for them seems yet to be established.

A counterpoint to the above possibilities is to undertake no new initiatives to regulate cadmium at this time. Many controls are either already in place or else under development. Consequently, while on-going work affecting decisions on the first two issues (1 and 2 above) is proceeding, the Agency is not undertaking regulatory work involving the latter two issues (3 and 4).

In addressing the above regulatory issues, the following analytical issues seem most important:

In estimating the risks of kidney dysfunction, only the long term total exposure by all exposure routes is of interest. Assesments of single exposure routes, unless they provide information on the general magnitude of other exposure routes, tend to occlude the perspective needed to reach an appropriate risk management decision. On the other hand, for the risks of cadmium induced lung cancer, inhalation exposure alone is relevant.

For consistency, an interoffice consensus is needed on some issues: (a) Cadmium's renal toxicity threshold (in terms of kidney concentration and in terms of daily uptake) is not completely resolved. (b) For

cross-media comparisons, the means of comparing exposure by different routes (i.e., absorption efficiencies) is not uniformly established, and (c) the means of assessing plant uptake (and resulting human exposure) of cadmium reaching topsoil by different pathways is uncertain.

Based on the work described herein, it appears that cadmium contamination of the environment may remain under scrutiny over a long period of time.

SECTION 1

INTRODUCTION

The environmental hazards of cadmium, a metal being dispersed into the biosphere through several pathways, have been a concern for many years, particulary after it was linked with the occurrence of Itai-Itai disease in post-war Japan. In the U.S. it appears that the margin of safety between the current population exposure and the kidney effect threshold is not particularly large. Furthermore, a possibility of carcinogenicity has also been a concern.

The Office of Water Regulations and Standards of the Environmental Protection Agency began a multi-media assessment of cadmium in 1978 as part of a program to evaluate the exposure and risk of the 129 priority pollutants. Through this effort several reports (referenced herein) on selected aspects of cadmium use and environmental dispersal were produced by consultants. An assessment of exposure and risk, performed in-house in 1979, resulted in recommendations for an agency course of action.

In autumn of 1983 the Deputy Administrator requested that the Office of Water Regulations and Standards assess the problems of environmental cadmium, and determine the needs for regulatory action and inter-office coordination. This assignment provided an opportunity to update and expand the previous assessment and prepare this technical report of findings.

The purpose of this document is to summarize the key information on cadmium use, dispersal, exposure, and risk. The intent is to quantify, to the extent possible, the effect of human activities on the incidence of cadmium-induced human disease and ecological impairment. The assessment is focussed primarily on contamination of food, ambient outdoor air, and drinking water (as well as ambient surface waters); contamination of the workplace is not considered here. The presentation has been kept as brief as possible; no attempt has been made to reference all relevant literature.

The organization of the material presented was selected in order to expedite relating exposure to health risks, and to focus sharply on the important exposure pathways. It may be noted that the critical analysis of data is concentrated on cadmium releases to and dispersal through the environment, the resulting exposure, and (given a particular toxic potency) the resulting incidence of harm. By contrast, the information an cadmium's toxicity (and carcinogenicity) is rather uncritically accepted from other sources.

Section 2 describes the human health effects of concern, assesses the current exposure of the population, and predicts the resulting disease incidence.

Section 3 presents cadmium uses, estimates release to all environmental media, and assesses the key pathways contributing to human exposure.

Section 4 discusses concerns about cadmium effects on aquatic ecosystems.

Although cadmium has been one of the most intensely studied toxic pollutants, uncertainties in its assessment remain. Many of the uncertainties in the levels of exposure and in the quantities discharged, emitted, or disposed of are caused by the difficulty of detecting cadmium at its usual trace concentrations. It is common for cadmium to be undetected in a substantial portion of samples. Other uncertainties arise in extrapolating from the limited data available. Still other uncertainties apply to cadmium's toxicity and especially its carcinogenicity. Thus, while this analysis has attempted to be quantitatively definitive, the results and conclusions are not indisputable. Indeed, substantial differences in opinion exist on many important issues.

SECTION 2

HUMAN EXPOSURE AND EFFECTS

2.1 Human Health Effects

Damage to the kidney's ability to reabsorb blood protein is the known (non-carcinogenic) effect having the lowest exposure threshold. Increasing degrees of cadmium induced renal tubular dysfunction are manifested in β_2-microglobulin proteinuria, general proteinuria, aminoaciduria, and glycosuria, in order of increasing severity. Effects on bone and mineral metabolism have accompanied kidney damage in severe cases as found in the Itai-Itai or "Ouch-Ouch" disease in Japan (EPA 1979, 1980).

Elevated β_2-microglobulin excretion is not equivalent to clinically significant proteinuria. Without continued high exposure to cadmium, there is little evidence of either a progession of severity of kidney dysfunction, or a significant shortening of life expectancy. Nevertheless, while some elevation of β_2-microglobulin excretion appears to be a relatively benign condition, it is usually taken as the threshold health effect in setting ambient cadmium criteria (EPA 1979, 1980).

Emphysema is another clearly documented health effect, having resulted from chronic occupational (inhalation) exposure to cadmium. However, as its threshold is higher than for kidney dysfunction, it is not used as a basis for environmental standards.

Inhaled cadmium also appears to be carcinogenic. EPA's Carcinogen Assessment Group (CAG) has suggested that cadmium be considered "probably carcinogenic in humans," based on lung cancer rates observed in smelter workers and based also on animal studies (EPA 1984c). Carcinogenicity is generally assumed to be a nonthreshold effect, with probability of occurrence proportional to the total cumulative dose. For lung cancer, inhalation may be deemed the only relevant exposure route.

While several other health effects, for example, immunosuppressive and other carcinogenic effects, have sometimes been ascribed to cadmium, the data indicating such effects have been considered too weak or conflicting to support regulatory standard setting. Thus, the effects considered in this document are kidney dysfunction and (for inhaled cadmium) lung cancer.

Whether the effect to be prevented involves a threshold (kidney dysfunction) or no threshold (cancer), the cumulative exposure to cadmium is of primary interest. Body burdens of cadmium (about one-half of which are concentrated in the liver and kidneys) steadily increase from very low levels at birth to maximum levels at age 40-50 years. Thus, cadmium depuration is very slow; once absorbed into the body, its half-life is estimated to be 18-38 years (EPA 1979, 1980).

Of the total quantity of cadmium brought into the lungs or digestive tract, only a portion actually crosses membranes into the blood stream. This percentage, the absorption efficiency, varies with the type of exposure typically as follows:

Inhalation	25%
Ingestion	5-6%

Such efficiencies may vary substantially from individual to individual and from time to time within the same individual. Some studies have found absorption of cadmium from cigarette smoke to be substantially higher than 25%.

β_2-Microglobulin proteinuria may occur when the concentration in kidney cortex reaches approximately 200-400 µg/g (wet weight), although individual susceptibilities may fall outside this range. At the present time a kidney concentration of 200 µg/g is the most widely accepted estimate of the critical threshold (Ryan et al. 1982). This concentration is estimated to result from a daily retention (absorption) rate of 10-15 µg/day over a 25-50 year period (EPA 1979, 1980). If 12 µg/day is taken as the absorbed dose that will produce a kidney cadmium level of 200 µg/g over a 25-50 year period, then at a 6% absorption efficiency this corresponds to a gross ingestion of 200 µg/day, a value sometimes cited as a threshold (Commission of the European Communities 1978). Thus, the WHO (1976) recommended tolerable gross intake from food, 57-71 µg/day, apparently incorporates an additional margin of safety; the appropriateness of this value has been questioned (Page et al. 1984). Ryan et al. (1982) and Logan and Chaney (1984) review some of the difficulties in establishing the dose-response relationship. Individual variations in proteinuria susceptability and cadmium absorption and depuration confound the establishment of a firm threshold.

The Cancer Assessment Group (EPA 1984c) considers the carcinogenicity of cadmium to vary depending upon exposure route. While the available data do not indicate that ingested cadmium is carcinogenic, there is some evidence, provided primarily by studies of smelter workers and of rats, to support classifying inhaled cadmium as being "probably carcinogenic in humans." EPA (1984c) has estimated the probable upper limit for the cancer potency of inhaled cadmium as follows: lifetime (70 year) exposure to 1 $\mu g/m^3$ in ambient air may give rise to risks as high as 2.3×10^{-3} for lung cancer. This estimate involves considerable uncertainty; other data, other assumptions, or other extrapolation procedures can yield different results. The Cancer Assessment Group considers its procedures to provide the best estimate for the upper limit on cancer potency; they do not consider it feasible to estimate either the most probable potency or a lower limit for potency.

2.2 Cumulative Exposure of Population

Long term total exposure can be indicated by the cadmium concentration in kidney cortex. Autopsy studies in Dallas, involving 93 men of greater than 30 years age, revealed an approximately log-normal distribution of exposures,

with a geometric mean of 24 ug/g, as shown in Table 1 (raw data from Johnson et al. 1978). These men had no known occupational exposure to cadmium.

Table 1: Statistics for Cadmium in Kidney Cortex (Dallas autopsy study, wet weight, males aged at least 30 years).

Category	Number	Arithmetic Mean (ug/g)	Geometric Mean (ug/g) [95% Confidence Range]	Geometric Dispersion [95% Confidence Range]
Nonsmokers	21	17.2	15.0 [11.7-19.3]	1.74 [1.53-2.19]
Smokers	72	30.9	27.9 [25.0-31.2]	1.60 [1.50-1.75]
Combined	93	27.8	24.2 [21.6-27.1]	1.73 [1.61-1.89]

The age groups 30-39, 40-49, 50-59, and greater than 60 years, did not differ significantly and consequently were combined together here. It is apparent, however, that the smoking and nonsmoking groups differ substantially: the arithmetic mean concentration for nonsmokers was only 56% of that for smokers. (The tabulated geometric dispersion, a geometric or multiplicative standard deviation, is through its definition the ratio of approximately the 84-th percentile concentration to the 50-th percentile concentration in a log-normal distribution.)

2.2.1 Estimating the Incidence of Exceeding a Kidney Effects Threshold

Figure 1 displays the Dallas autopsy data. The distribution for nonsmokers appears to be log-normal (the number of runs of consecutive data points above and below the straight line falls within the "runs test" 95% confidence range (Remington and Schork 1970)). In a log-normal distribution the fraction, Q, of the population exceeding a particular concentration, x, is given by:

$$Q(Z) = (1/\sqrt{2\pi}) \int_Z^\infty \exp(-z^2/2)\, dz \qquad (2\text{-}1)$$

where Z is the log-normal deviate, or relative distance between the particular concentration, x, and the geometric mean concentration, $\bar{x}_g$:

$$Z = \ln(x/\bar{x}_g)/\ln s_g \qquad (2\text{-}2)$$

where s_g is the geometric dispersion (i.e., geometric standard deviation). For this work Equation 2-1 was evaluated with the numerical integration function of a pocket calculator (Hewlett-Packard 1984). Alternatively, however, the relationship between Q and Z can be obtained from extended tables of the normal distribution, such as provided by Meyer (1975).

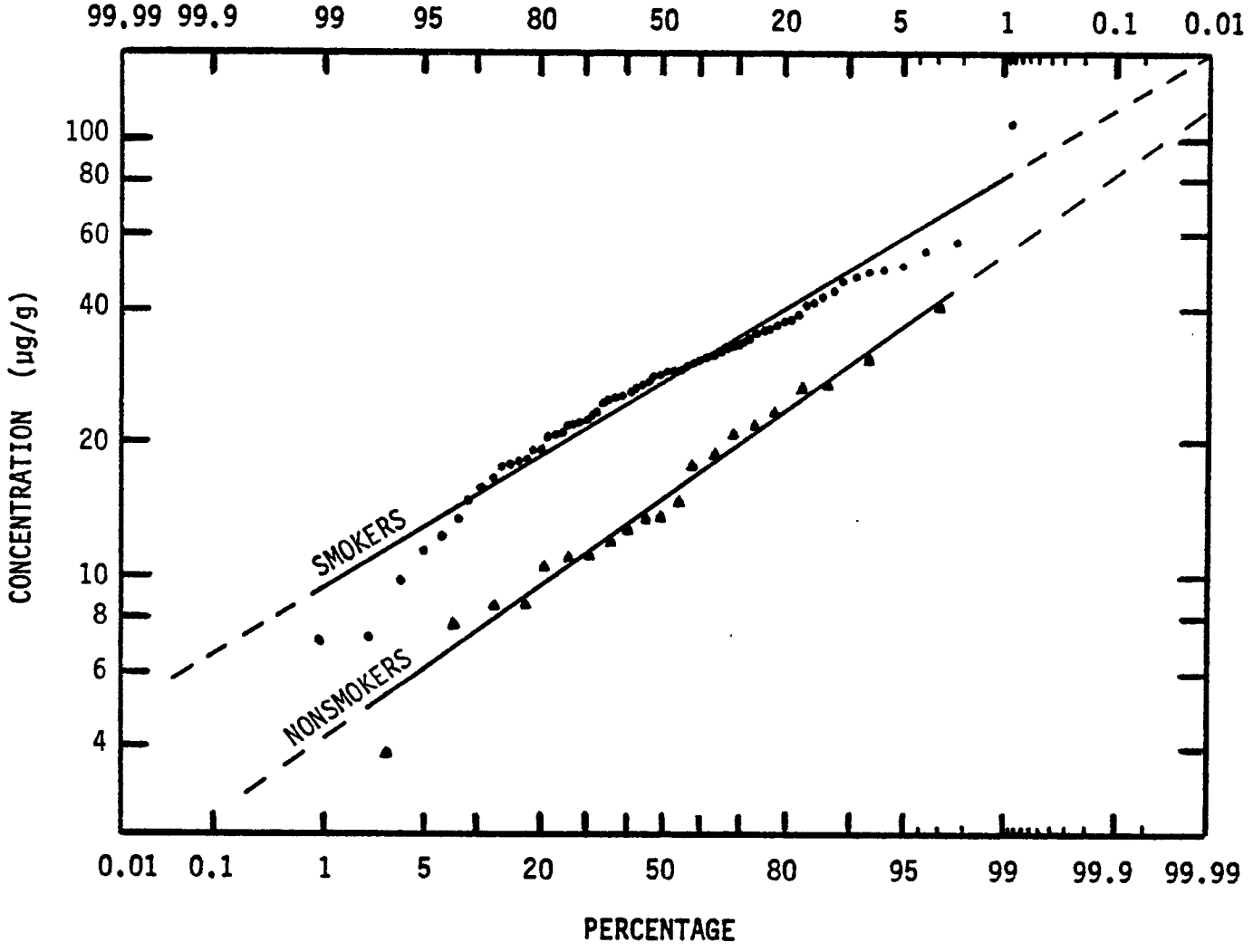

FIGURE 1: CADMIUM CONCENTRATION IN KIDNEY CORTEX: FREQUENCY DISTRIBUTION FOR MALES AGED 30 YEARS OR MORE.

Mathematically extrapolating the log-normal nonsmoker distribution upward in this manner, it would be estimated that 1.5 persons per million (in this age group) exceed a 200 ug/g kidney effects threshold. The validity of such an extrapolation of course hinges on the assumption that log-normality holds in the extreme upper tail of the distribution. Thus, it involves considerable uncertainty, like cancer risk extrapolation. Unlike cancer extrapolations, however, it was not intended here to increase safety by overestimating the risk.

Unlike the distribution for nonsmokers, the Figure 1 distribution for smokers does not appear to be truly log-normal. The lack of log-normality may be an artifact of the way Johnson et al (1978) defined smoking/nonsmoking status in the autopsy study. Perhaps more likely, however, is an inherent lack of log-normality in the distribution of exposure among the smoking population. If it were assumed that the smoker distribution were also log-normal, extrapolation would indicate that 14 persons per million (in this group) would exceed the threshold.

(Assuming that cadmium were also carcinogenic, the potential cancer risks cannot be estimated solely from data on kidney concentrations. Additional consideration of cadmium intake is required and will be taken up later.)

2.2.2 Predictive Uncertainty

The results of the Dallas autopsy study appear consistent with other studies (presented by Ryan et al. 1982). While the samples were not randomly selected from the nation's population, a clear justification for expecting it to differ significantly from the rest of the nation was not apparent.

The 95% confidence limits for the geometric mean and geometric dispersion were presented in Table 1. The confidence interval for the geometric dispersion of the nonsmoker distribution, because it is based on only 21 samples, is rather large and may overstate the true uncertainty in this parameter. An independent study of kidney cadmium, by Indraprasit et al. (1974), also appears to indicate values for the geometric dispersion the range 1.7-1.8. To produce these values the arithmetic mean, $\bar{x}_a$, and standard deviation, s_a, reported by Indraprasit et al. (1974), were converted to a geometric dispersion, s_g, by the expression:

$$s_g = \exp \sqrt{\ln(1 + (s_a/\bar{x}_a)^2)} \qquad (2\text{-}3)$$

Ellis et al. (1979), measuring the total quantity of cadmium in kidney, found a geometric dispersion of 1.7 for smokers and 2.0 for nonsmokers. The variability they found might be expected to be somewhat higher since it involves the variabilities of both kidney size and cadmium concentration. Overall, the results of these other studies, and the weight of evidence from the additional 72 samples of smokers taken by Johnson et al. (1978), suggests that the confidence interval tabulated for the combined smokers and nonsmokers, approximately 1.6-1.9, might better represent the uncertainty in the geometric dispersion.

The uncertainty in estimating the number of persons exceeding a 200 µg/g threshold is quite sensitive to the uncertainty in the geometric dispersion. If the confidence interval for this parameter were taken to be 1.6-1.9, and the mean taken to be 15.0 µg/g, then the corresponding range for the number of persons exceeding the threshold would be 0.018-27.2 persons per million.

The predictions are less sensitive to the uncertainty in the geometric mean. The confidence interval for the nonsmoker mean, 11.7-19.3 µg/g, when coupled with the geometric dispersion of 1.74, corresponds to a range of 0.15-12.1 persons per million exceeding the threshold.

The incidence of kidney dysfunction is quite sensitive to the concentration threshold assumed. If the threshold were increased from 200 µg/g to 300 µg/g, the number of persons exceeding the threshold would drop from 1.5 persons per million to 0.032 persons per million, nearly a 50 fold decrease.

In any case it must be emphasized that the projected incidence assumes a log-normal distribution of kidney cadmium levels. If log-normality does not hold in the extreme upper tail of the distribution, the true incidence may be quite different. The validity of applying log-normality to the extreme upper tail has not been established.

2.2.3 Exposure Trends

Relative to other toxic metals, relatively little margin of safety exists between typical exposures and the threshold effect level. On the other hand, based on the extrapolation from the Dallas autopsy study, cadmium induced β_2-microglobulin proteinuria appears to be very uncommon in the general population.

Perhaps a more appropriate concern is whether exposure may be gradually increasing. Elinder and Kjellstrom (1977) (and Kjellstrom 1979) report that cadmium levels in preserved kidney specimens collected between 1880-1899 are nearly four fold lower than levels for nonsmokers who died in 1974. This represents an annual compound rate of increase of 1.6 %/yr. Interestingly, Kjellstrom et al. (1975) report a corresponding rate of increase, 1.4 %/yr, in cadmium concentrations in a series of fall wheat specimens collected between 1920-1970. The rate of increase estimated for cadmium in spring wheat specimens was less than that for fall wheat and was not statistically significant. Kjellstrom et al. (1975) and Purves (1977) discuss reported cadmium increases in natural vegetation over many years, apparently due to emissions deposition. MacGregor (1981) has reviewed trends in ambient levels; the general tendencies appeared to be increases in some cases and plateaus or stable levels in other cases.

Despite the apparent agreement between the rate of increase in cadmium levels for fall wheat (Kjellstrom et al. 1975) and for human kidney cortex (Elinder and Kjellstom 1977), the existence of and magnitude of any long term increase in exposure is uncertain. Drasch (1983) measured the cadmium content of preserved liver and kidney specimens collected between 1897-1939 and compared

them to autopsy samples taken in 1980. He found a 47 fold increase in kidney cortex cadmium but a negligible increase in liver cadmium. Such results are difficult to reconcile; while the levels in both kidney and liver are considered to be good indicators of cumulative exposure, levels in liver may be the better indicator (Cherry 1981). Overall, it might be concluded that there is some evidence for a significant long term increase in cadmium exposure; however, such evidence may result from artifacts of sampling. Consequently, it is also possible that no significant increase in exposure has been occurring. In any case, it is not known whether any such increase is now occurring.

In linear nonthreshold cancer projections, small percentage increases in (arithmetic) mean exposure produce equally small percentage increases in population risks. For threshold effects, on the other hand, small percentage increases in mean exposure may more sharply increase the population risks. To illustrate, a 1 %/yr increase in mean exposure to cadmium would yield a 22% increase in mean kidney concentrations, when compounded over 20 years. Projecting from the nonsmoker distribution in Figure 1, a 22% increase in geometric mean (with no change in geometric dispersion) would result in more than a 5 fold increase in the number of persons exceeding the 200 ug/g threshold (i.e., raising it from 1.5 persons per million to about 8 persons per million).

Increasing the variability of exposure would even more sharply increase the risks. For example, increasing the geometric dispersion of the nonsmoker distribution from 1.74 to 2.0 (with no change in the geometric mean) would increase the incidence from 1.5 persons per million to 93 persons/million, assuming a log-normal distribution.

2.3 Human Exposure Routes

The concentrations of cadmium associated with airborne particulates have been measured at numerous urban and rural stations belonging to National Air Surveillance Networks. Detectable concentrations occur far more frequently at urban than at rural sites. Figure 2a illustrates the overall distribution of urban concentration statistics provided by Rhodes et al. (1979) for all quarterly composite samples collected in the period 1970-1976. Assuming a log-normal distribution, the geometric mean might be estimated to be perhaps 1.6 ng/m^3.

The variability (slope) shown in Figure 2a represents the variability among quarterly composite samples. Consequently, it has components of both temporal and spatial variability. Since only the spatial variability is of interest in the assessment of long term exposure, the distribution may somewhat overestimate the true variability of long term ambient inhalation exposure.

The arithmetic mean, $\bar{x}_a$, of a log-normal distribution can be estimated from the geometric mean, $\bar{x}_g$, and geometric dispersion, s_g, as follows (Meyer 1975):

$$\bar{x}_a = \exp\left[(\ln \bar{x}_g) + 0.5(\ln s_g)^2\right] \qquad (2\text{-}3)$$

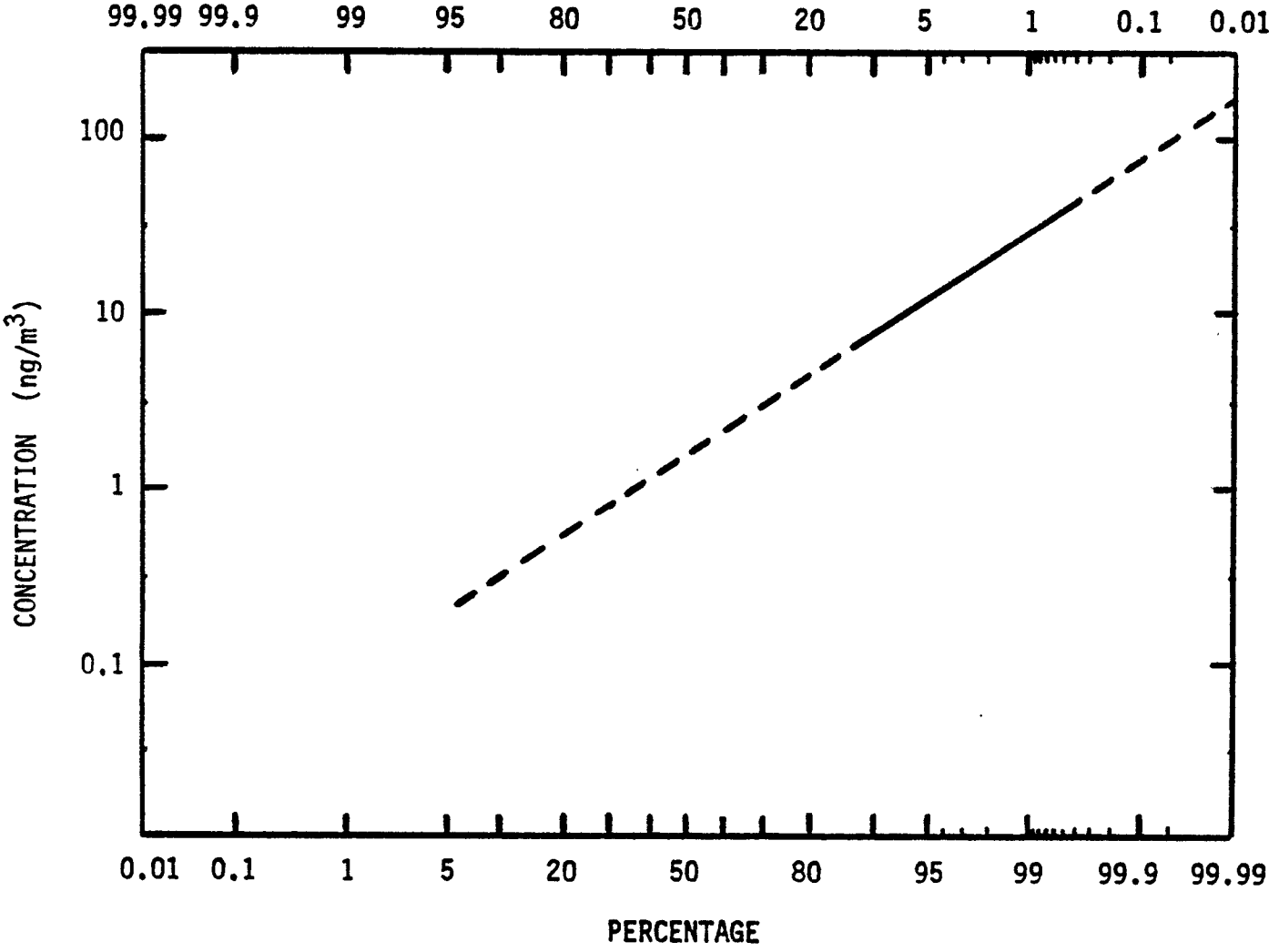

FIGURE 2a: AIR QUALITY DATA FOR CADMIUM: FREQUENCY DISTRIBUTION FOR QUARTERLY COMPOSITE SAMPLES AT URBAN SITES, 1970-1976.

Note: Solid line indicates range of detected concentrations.

Using this relationship an arithmetic mean of 3.5 ng/m^3 was estimated for the air data.

Concentrations of cadmium in drinking water almost never exceed the standard of 10 ug/l. A 1969 nationwide survey showed 63% exceeding 1 ug/l and 0.15% exceeding 10 ug/l (Battelle 1977). While this frequency distribution is plotted in Figure 2b, it is important to note that the measurements were generated using crude flame atomic absorption spectroscopy procedures, now considered to be unreliable due to their strong overestimating bias when applied to cadmium at its usual ambient levels.

A nationwide survey using a sensitive graphite furnace AAS method has apparently not been done in this country; however, such work has been done in Canada. As the complete data sets could not be obtained, rudimentary summaries of these data (Meranger et al. 1981) were used to construct the estimated distribution. As shown in Figure 2b, the constructed distribution has a geometric mean of 0.04 ug/L, a geometric dispersion of 2, and an arithmetic mean of 0.05 ug/L. To compensate for the uncertainty in portraying this distribution, a high bias was intentionally incorporated. It is estimated that the mean of this distribution over-estimates the true Canadian mean by a factor of two, based on the discussion of HWC and EC (1983). In the absence of reliable data for the U.S., the distribution constructed from the Canadian data will be assumed to provide the best estimate for U.S. nationwide exposure via drinking water. The variability shown applies to grab samples.

Dietary intake of cadmium may be estimated from either food or fecal analyses. Estimates of cadmium gross intake based on measurements of concentrations in foodstuffs coupled with diet information (FDA data, as analyzed and reported by Yost et al. 1978) result in a log-normal distribution of cadmium intake with geometric mean of 12 ug/day and geometric dispersion of 4.6. Estimates of cadmium gross intake based on measurements of fecal concentrations in 477 persons in Chicago and Dallas (as reported by Kowal et al. 1979) indicate an approximately log-normal distribution having a geometric mean of 11.4 ug/day and geometric dispersion of approximately 1.9-2.1. These are plotted in Figure 2c. Both studies provide a population distribution of cadmium intake on a single day. Yost's analysis, however, applies to a teenage male diet. Kowal's fecal data includes both sexes and all age groups from 0-70 years, but its variability applies primarily to the cadmium concentration, and only partially to the fecal volume.

Although the geometric mean intakes obtained from both the food/diet and fecal methods are virtually identical, the much greater variance obtained using food data results in a significant difference in arithmetic means between the two. Yost's analysis of food data indicated an arithmetic mean of 39 ug/day (which is identical to the mean reported by Pahren et al. (1979) for 7 years of FDA data). The fecal data has an arithmetic mean of approximately 14 ug/day.

As indicated by the previously mentioned difference in kidney cadmium levels between smokers and nonsmokers, tobacco is an important source of cadmium exposure. Cigarettes are estimated to result in the inhalation of 3 ug/pack coupled with a 25% absorption efficiency (EPA 1980). Smokers, of which there

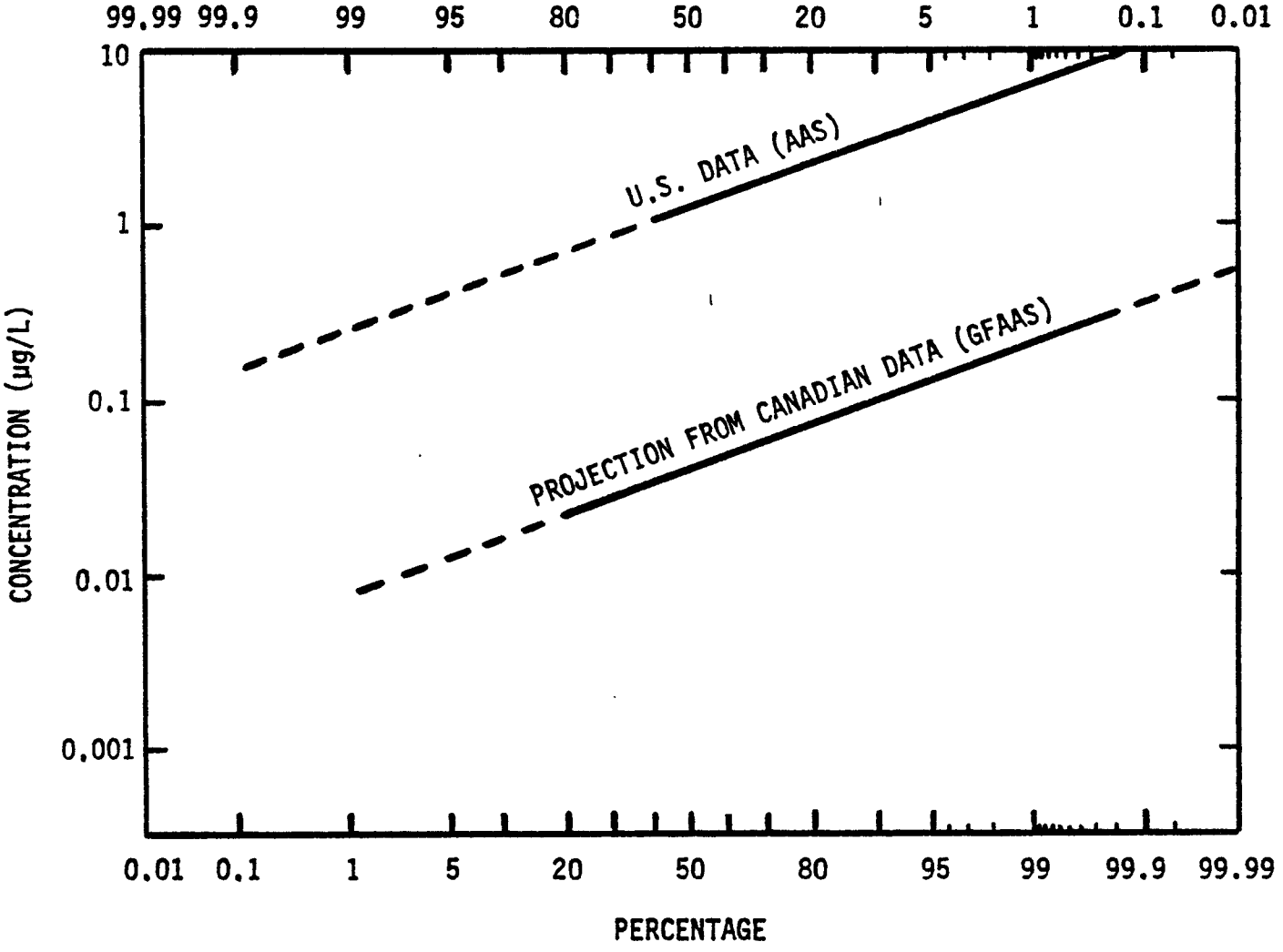

FIGURE 2b: DRINKING WATER DATA FOR CADMIUM: FREQUENCY DISTRIBUTION OF GRAB SAMPLES AT TAP OR WITHIN DISTRIBUTION SYSTEM.

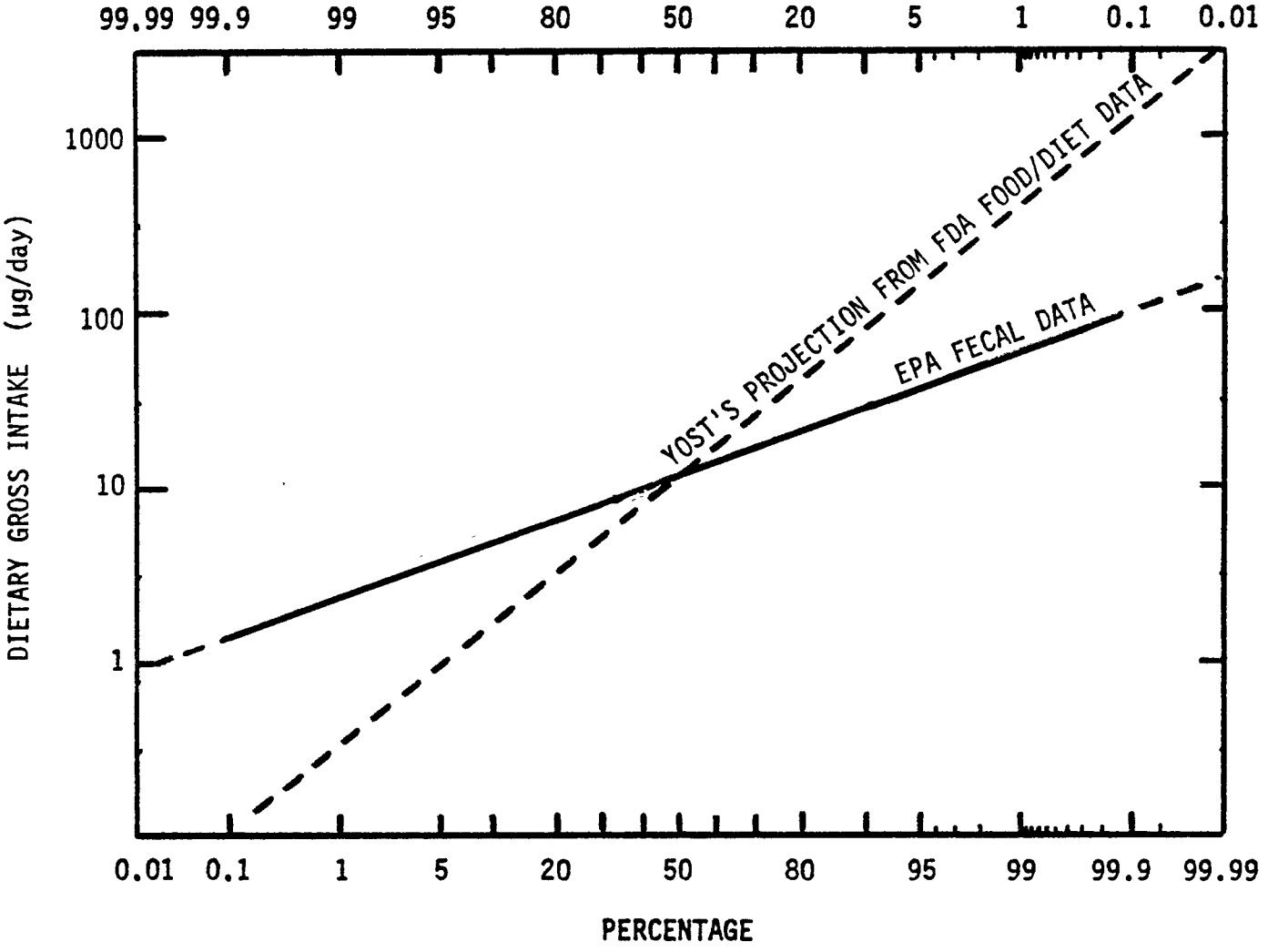

FIGURE 2c: DIETARY INTAKE OF CADMIUM: FREQUENCY DISTRIBUTION FOR GROSS QUANTITY INGESTED ON A SINGLE DAY.

were 54 million in 1978, each consume an average of 1.56 packs/day (Richmond 1981, as reported by Gilbert 1982). As smoking is not done over an entire lifetime, 1 pack/day might approximate an effective average consumption of cigarettes over the first 50 years of a lifetime for the smoking subpopulation.

It should be noted that cadmium has been detected in air contaminated by tobacco smoke at levels as high as 100 ng/m^3 (Brodie and Matousek 1974, as reported by MacGregor 1981). Such levels are higher than normally found in outdoor urban air.

Comparisons between the contributions of air (with outdoor quality), water, and food to human uptake of cadmium are shown in Figure 3. These results have been summarized and compared with cadmium exposure via tobacco smoking in Table 2. Both Figure 3 and Table 2 were generated from the following data and assumptions:

Air: Figure 2a concentration distribution, assuming 20 m^3/day inhalation rate, 25% absorption efficiency.

Water: Figure 2b Canadian data, assuming 2 L/day ingested, 6% absorption efficiency.

Food: Figure 2c fecal data, assuming 6% absorption efficiency.

Tobacco: 3 ug/pack cigarettes, 1-3 packs/day, assuming 25% absorption efficiency.

It is apparent from Figure 3 that population exposure via food greatly surpasses the exposures via drinking water and ambient air inhalation. With a long term exposure threshold (for kidney dysfunction) of 10-15 ug/day, it is also apparent that hazardous ambient exposure is almost not possible unless dietary exposure is elevated.

It should be noted that the variabilities (slopes) in Figure 3 may not be strictly comparable due to the different time frames for sampling. Furthermore, in comparing to a long term exposure threshold (10-15 ug/day), it must be noted that the short term exposure variability illustrated is inherently larger than the long term exposure variability, which was discussed in the previous section and illustrated in Figure 1. It should also be noted that air, water, and food exposures may vary independently of each other.

The Table 2 exposure route estimates seem consistent with the Table 1 kidney cadmium levels. In Table 1 the nonsmoker arithmetic mean is 56% of the smoker arithmetic mean, while in Table 2 it is 54%. In Table 1 the nonsmoker arithmetic mean is 8.6% of the 200 ug/g kidney cadmium threshold, while in Table 2 it is 5.8-8.7% of the 10-15 ug/day absorbed dose threshold. As Table 2 is based on dietary intakes predicted from fecal measurements, it appears that these produce reasonable exposure estimates. The FDA food data, on the other hand, would seem to overestimate the current exposure when compared to the threshold. (Nevertheless, the FDA food data can produce equally consistent results if the ug/day cadmium level producing the 200 ug/g kidney

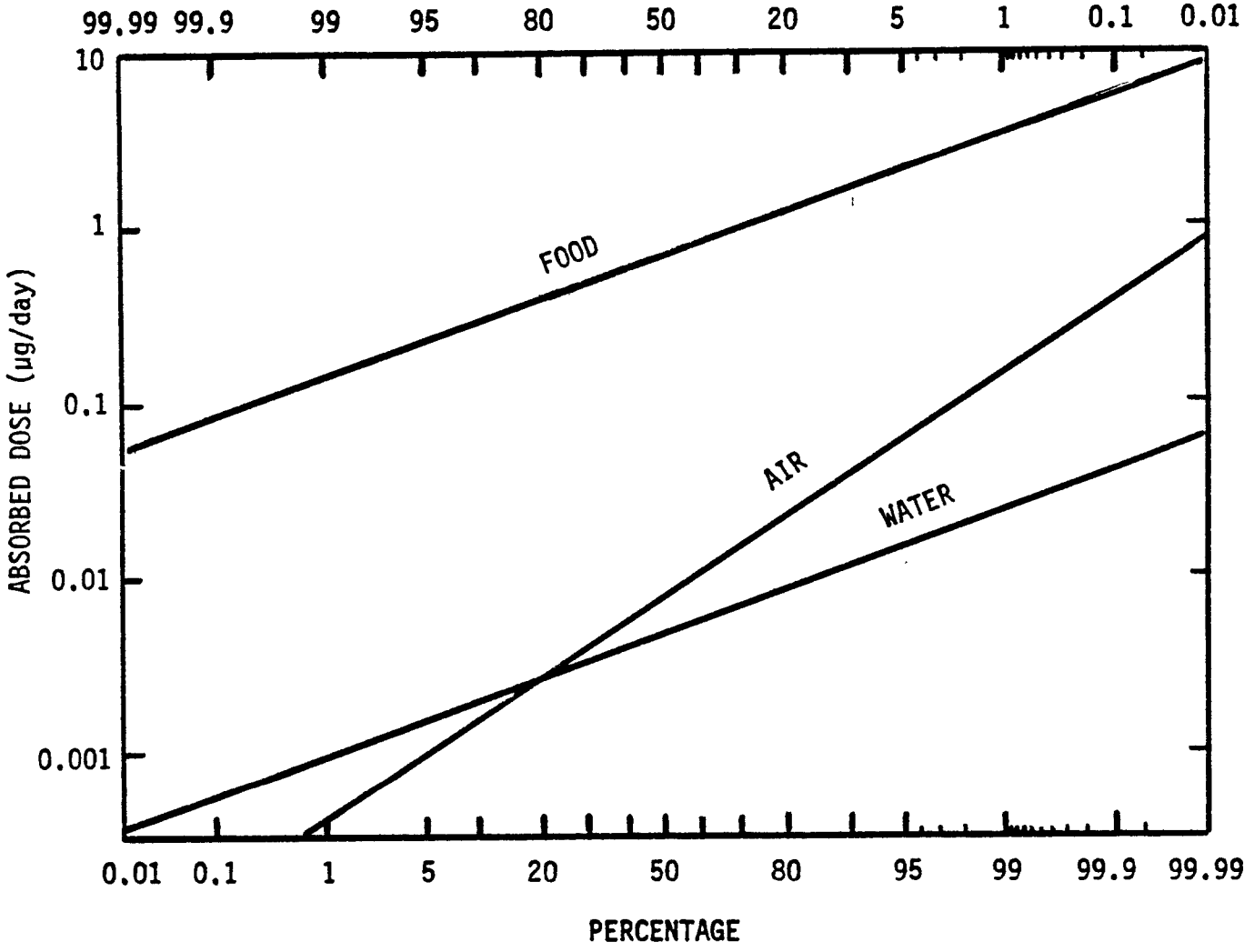

FIGURE 3: COMPARISON OF DISTRIBUTIONS OF ABSORBED DOSE VIA INHALATION AND VIA INGESTION OF FOOD AND WATER.

Table 2: Comparison of cadmium exposure routes.

	Cadmium Absorbed into the Body, µg/day (a)		
	Typical Exposure		
Route	Median	Arithmetic Mean	Elevated Exposure (b)
Air	0.008	0.018	0.4
Water alone (c)	(0.005)	(0.006)	(0.04)
Food + Water	0.66	0.85	5.6
Total Ambient	0.67	0.87	6.0
Cigarettes		0.75	2.3

(a) Origins of data are described in text.
(b) For air, water, and food individually, elevated implies 99.9%.
(c) The Kowel et al. (1979) fecal data provides estimate of combined food plus drinking water exposure. Consequently, "water alone" is not added into the "total ambient" exposure.

level is approximately doubled and if the absorption efficiency for tobacco smoke is doubled.) Thus, the data and assumptions on exposure and toxic threshold used in this assessment seem to fit together in a consistent manner (although other data and assumptions are also capable of such consistency).

2.4 Potential Cancer Risks

The incidence of cadmium induced kidney dysfunction has already been discussed in terms of cumulative body burden in Section 2.2. As cancer risks are estimated from cadmium intakes rather than body burdens, such risks can be dealt with here. EPA (1984c) has estimated that lifetime exposure to an air concentration of 1 ug/m^3 would yield (as an upper bound) a lifetime incremental risk of 2.3×10^{-3} for lung cancer. The upper-bound risk corresponding to the 0.0016 ug/m^3 estimated median concentration in urban air would thus be 3.7×10^{-6}. The highest 0.1% of the quarterly composite samples shown in Figure 2a exceeded the median by nearly 50 fold; however, it is not known by what factor the highest 0.1% of individual lifetime exposures exceed the median.

For the arithmetic mean concentration, estimated to be 0.0035 ug/m^3 in urban air, the upper-bound risk would be 8.1×10^{-6}. This would correspond to an upper-bound incidence of 25 cases/year nationwide (assuming that indoor air is similar to outdoor air, and overlooking the fact that the urban mean concentration is higher than the rural mean). It must be recognized that this prediction involves many uncertainties and is intended to be an upper bound. The actual incidence of cadmium induced lung cancer could be much different.

The contamination of indoor air with cadmium from tobacco smoke (or other sources) could further increase the risks; however, the amount of this increase, which depends on concentration and duration of exposure, has not been estimated.

For cigarette smokers, the risks from direct inhalation of tobacco cadmium can be estimated by noting that smoking of 1 pack of cigarettes containing 3 ug/pack is equivalent to inhaling air having concentration 0.15 ug/m^3 for 1 day. The upper-bound cancer risk (due to cadmium alone) associated with averaging 1 pack/day over a 70 year lifetime would thus be 3.5×10^{-4}. The upper-bound cancer incidence associated with 54 million smokers averaging 1.56 packs/day would be 415 cases/year. As the actual nationwide incidence of lung cancer is around 90,000 cases/year (based on U.S. rates tabulated by Schottenfeld 1975), it appears that inhalation of cadmium is not a major contributor to the nationwide incidence of lung cancer.

Cancer risks (if any) associated with exposure via food and drinking water cannot be expressed quantitatively but may be considered to be low, since EPA (1984c) found no evidence for carcinogenicity of ingested cadmium.

SECTION 3

ENVIRONMENTAL SOURCES AND PATHWAYS

3.1 Use and Environmental Release

Cadmium is obtained as a by-product primarily during the smelting of zinc but also during the smelting of lead and copper. Total domestic use of imported and domestically produced cadmium has fluctuated between around 3000 and 6000 mt/yr (metric tons per year) in the period 1970-1982, depending in part on the overall level of of economic activity (U.S. Bureau of Mines 1982). Table 3a shows the total domestic use for recent years (U.S. Bureau of Mines 1982). Table 3b identifies the distribution of total use for the year 1979 (U.S. Bureau of Mines 1980, as reported by Kayser et al. 1982) and the year 1981 (U.S. Bureau of Mines 1981).

Table 3a: Total Domestic Use of Cadmium.

Year	Use (mt/yr)
1978	4510
1979	5099
1980	3534
1981	4378
1982	3707

Table 3b: Distribution of Domestic Use in 1979 and 1981.

	Percentage of Total	
Uses of Cadmium	1979	1981
Metal Plating	51	34
Batteries	22	16
Pigments	13	27
Plastics	11	15
Other	3	8

The largest users of the cadmium-bearing materials from the above catagories are the transportation equipment and defense industries.

In addition to the commodity volumes shown, a significant amount of cadmium is handled and released as an impurity in other materials. Of particular importance are cadmium impurities in phosphate fertilizer, in fossil fuels, and in other metals, especially zinc.

An integrated multimedia materials balance for cadmium release to the environment is presented in Table 4, derived from data from several sources

Table 4: Materials Balance for Cadmium (mt/yr)

	POTW	MUNICIPAL REFUSE	AIR	WATER	LANDFILL	LANDSPREAD
Zn/Pb Mining & Beneficiation	-	-	- (V)	8 (W) a	250 (V)	-
Zn/Cd Smelting	- (W) w	-	7 (G)	1 (W) w	- (V)	-
Electroplating	82 (U) v	817 d,e	-	4 (U)	370 y	-
Batteries	5 (U)	672 d,f	1 (V)	13 (U)	9 (V)	-
Pigments & Plastics	7 (V)	1801 d,n	13 (V)	1 (V)	17 (V)	-
Pesticide	-	-	-	-	-	9 (S) h
Other Cd Products	N	341 d	N	N	N	N
Impurity in Zn Products	N	N b	N	N	N b	N
Iron & Steel Industry	10 x	-	14 (G)	10 (U)	400 (Y)	-
Primary Non-Ferrous/Non-Zinc	- (W) w	-	218 (G)	1 (W) w	-	-
Secondary Non-Ferrous	1 (W)	-	2 (V)	- (V)	20 (V)	-
Printing/Photography	11 (V)	N	-	- (V)	N	-
Other Manufacturing Activity	45 (U) u	N	N	12 (U) u	N	-
Coal Mining	- (W)	-	-	45 (A) i	N	-
Coal Combustion	- (W)	-	202 (G)	28 (W)	429 (V)	-
Oil Combustion	-	-	363 (G)	-	-	-
Gasoline Combustion	-	-	13 (E)	-	-	-
Lubricating Oil	-	N	1 (V)	N	N	-
Tire Wear	-	N	5 (E,V)	-	-	-
Phosphate Detergent	10 (V)	-	-	-	-	-
Phosphate Fertilizer	-	-	-	-	-	400 (X) s
Urban Runoff	2 c	-	-	19 c	-	-
Culturally Hastened Erosion	-	-	N	182 q	-	-
Natural Weathering	-	-	N	170 r	-	-
Potable Water Supply	2 p	-	-	-	-	-
POTW Effluent		-	-	76 j	-	-
POTW Sludge		-	14 (G)	- m	211 j,k	123 j,k
Municipal Refuse	-		38 (G)	-	3593 g	-
Totals	175/485 j,t	3631 t	891	570	5299	532

Symbol "N" signifies "no data"; symbol "-" signifies "insignificant."
See following page for notes a-y and references (A)-(Y).

Table 4 (Continued): Explanatory notes.

The nominal reference year for the table is 1981, although many values are taken from studies applicable to other recent years. The table recognizes four ultimate dispositions, to air, water, landfill, and landspread. The totals contributed to POTWs and municipal refuse are ultimately redistributed (near the bottom of the table) to air, water, landfill, and landspread.

a. Alternative estimate for Zn/Pb mine discharges: 114 mt/yr (active mines) plus 90 mt/yr (inactive mines (Yost 1978).
b. The Cd impurity in zinc products has been alternatively estimated as 173 mt/yr (Yost 1978) or as 2371 mt/yr (JRB 1980). Disposition of this is not known but may be mostly toward landfill.
c. Urban runoff Cd: concentration roughly 1 ug/L (EPA 1983) for 21 trillion liters/yr (Sullivan 1977), flowing either to POTWs or directly to surface waters. This quantity may include material corroded from Cd and Zn plating as well as Cd emissions washout.
d. The quantity ultimately disposed of in municipal refuse is estimated to be the entire amount of virgin Cd used in the industrial catagory in 1981, minus the quantities identified to be released to the other media shown. Material quantities have been balanced assuming that there is no change in the accumulation within the technosphere (socio-economic system); that is, quantities entering the technosphere are balanced by quantities disposed of or released to the environment. Where population numbers and wealth are increasing, such a simplifying assumption is not strictly accurate; nevertheless, it is used here to facilitate understanding of cadmium movement.
e. Quantity in spent electroplated products is estimated as in Note d, minus an additional 216 mt/yr cycled as scrap to the iron and steel industry (Yost 1978).
f. Recycle of battery Cd is estimated to be 89 mt/yr (Yost 1978 and Versar 1980). The demand for virgin Cd in 1981 is assumed to be required to replace battery Cd which is not recycled. That Cd which is not recycled is assumed to be disposed of or released to the environment.
g. Refuse Cd entering landfill is assumed to be the total municipal refuse Cd minus the municipal incinerator emissions.
h. Alternative estimate for landspread pesticide quantity: Versar (1980) notes an additional 280 mt/yr of imported Cd fungicide/nemotocide. Such Cd is used on non-agricultural turf.
i. From active coal mines Versar (1984) estimates 15 mt/yr to water.

Table 4 (Continued):

j. Two independent estimates of POTW influent Cd are as follows: 175 mt/yr is sum of identified contributions; 485 mt/yr is estimated from a flow-weighted mean concentration of 13.4 µg/L for 39 of 40 POTWs sampled by EPA (1982) (rejecting one outlier), multiplied by a total nationwide POTW flow of 26.2 bgd (EPA 1979b). The 485 mt/yr influent Cd estimate almost balances the 76 mt/yr total effluent Cd estimate (from the same EPA (1982) data, mean effluent concentration 2.1 µg/L), added to 425 mt/yr total sludge Cd quantity independently estimated from a production-weighted mean concentration of 85 ppm for 353 POTWs listed by EPA (1979a) multiplied by 5 million mt/yr sludge production (EPA 1979a).

k. The disposition of 5 million mt/yr sludge is taken as follows (EPA 1979a):
   Air (incineration): 21%, less quantity captured by emission controls.
   Ocean dump: 18%. This quantity does not appear on the table.
   Landfill: 32%, plus captured emissions.
   Landspread: 29%. (More recent data (EPA 1980) indicate landspreading of 31% of all sludge.)
   These quantities do not take into account RCRA/CWA regulations limiting the rate of landspreading sludge cadmium on disposal sites.

m. No sludge is expected to be released to fresh or estuarine waters. The table does not include 77 mt/yr sludge Cd ocean dumped.

n. The pigment and plastic refuse Cd quantity compares favorably with the combustable refuse Cd quantity independently estimated from the data of Campbell (1976): 100 million mt/yr combustable refuse having Cd concentration 14 ppm.

p. Background concentration of drinking water (measured within the distribution system) is estimated from Meranger (1981), and assumed to apply to 26.2 mgd (EPA 1979b). It might also be noted that since fecal measurements indicate excretion of 10-20 ug/person-day (Kowel et al. 1979), the 150 million people served by POTWs would generate only about 1 mt/yr of the POTW influent Cd.

q. The culturally accelerated soil loss is 70% of the roughly 1 billion mt/yr soil carried to major streams (Wischmeir 1976). Cd concentration of the eroded soil is estimated to be 0.26 ppm (Carey 1979). Much of this release may be carried as a stream's particulate load.

r. Natural background concentration is highly uncertain but is assumed here to be 0.1 µg/L; continental U.S. streamflow 1200 bgd (Miller et al. 1963).

Table 4 (Continued):

s. Versar (1979b) projection for 1980.
t. Totals in POTW influent and municipal refuse are redistributed to the four basic media (air, water, landfill, and landspread). The total quantity passing through the technosphere is the sum of the totals for the four basic media.
u. Other manufacturing applies to industries evaluated by Versar (1984): Aluminum and Non-Ferrous Metals Forming, Coil Coating, Copper Forming, Electrical and Electronics, Foundries, Inorganic Chemicals, Leather Tanning, Pesticides Manufacturing, Petroleum Refining, Pharmaceuticals, Plastics Molding, Porcelain Enameling, Pulp and Paper, and Textiles.
v. The electroplating (metal finishing) contribution to POTW, 82 mt/yr, assumes that 18.8% of the industry discharges raw wastes, and 81.2% has installed pretreatment meeting EPA's PSES standards. As the raw waste before pretreatment is estimated to be 313 mt/yr, the recent installation of such pretreatment has greatly reduced this formerly large source.
w. Alternate estimate for the entire non-ferrous metals smelting industry: 10 mt/yr to POTW and 10 mt/yr to surface water (Versar 1984).
x. The Iron and Steel contribution to POTW is considered to be somewhere between the Versar (1984) raw waste estimate of 37 mt/yr and pretreatment estimate of 1 mt/yr.
y. The quantity landfilled is assumed to be the quantity removed from wastewater prior to discharge: 456 mt/yr raw waste (Versar 1984) less 86 mt/yr discharged to water or POTW.

REFERENCES:

(A) Arthur D. Little (1979)
(E) EEA (Coleman et al. 1978)
(G) GCA (1981)
(S) SRI (Casey 1979)
(U) Versar (1984)
(V) Versar (1980)
(W) Versar (Alchowiak and Maestri 1980)
(X) Versar (1979b)
(Y) Yost (1978)

identified therein. The table identifies four basic media (air, water, subsoil or landfill, and topsoil) and two submedia (municipal wastewaters and refuse) which in turn feed into the four basic media. The table represents a closed balance for commodity cadmium, assuming no change in the accumulation within the socio-economic system.

Publicly owned treatment works (POTW) receive wastes from households and industries discharging to sewers. It may be noted that the quantity estimated to reach POTWs (based on POTW sampling) exceeds the contributions estimated from industries and households. It is not known whether the tabled values underestimate some sources (such as electroplating wastes, which are projected to be undergoing a substantial reduction), or whether the POTW influent data are no longer representative.

Municipal refuse is loosely defined to include junked end products, after completion of their useful life. For example, the entire volume of plated cadmium, less plating wastes (liquid and solid) and metal recycle, is assumed to be disposed of in the manner of municipal refuse. Thus, most of the values in this catagory are not based on waste sampling.

The major atmospheric releases have been estimated from U.S. EPA Air Program information. Such releases exclude those associated with windblown soil or other natural emissions. Fossil fuel combustion and nonferrous metals (excluding zinc) industries appear to constitute the largest sources to air; zinc smelters, formerly a large source, now appear to be tightly controlled.

The releases directly to water involve discharges and runoff to surface waters, including estuarine and coastal waters; they exclude dumping in open ocean waters. With implementation of national BAT (Best Available Technology) standards, it appears that the formerly large industrial sources, such as electroplaters, are being substantially reduced. Consequently, it appears that nonpoint sources, containing essentially background levels of cadmium, may become the dominant sources when aggregated nationwide. The total estimated discharge of cadmium expected under BAT requirements for various industries is presented later in Table 12; the values presented in Table 4 may or may not represent BAT.

The landfill catagory is loosely defined as any solid waste that is disposed of on land but not spread thinly over the surface. Most of these values are highly uncertain, and no data are available for some potentially large waste volumes. Under the assumption that junked end products are disposed of in manner resembling that of municipal refuse, most of the commodity cadmium would end up in landfill.

The landspread catagory, on the other hand, involves material spread thinly into the the topsoil, generally with the intention of increasing vegetative productivity. This catagory will be more fully discussed below.

3.2 Pathways for Contamination of Food via Topsoil

The most critical environmental pathway for cadmium exposure involves the contamination of topsoil. Terrestrial plants uptake and concentrate cadmium from the root zone. The largest component of human exposure results from consuming the edible portions of terrestrial plants (and for tobacco, inhaling the combustion emissions); the other major component involves consumption of animals (or animal products) fed with terrestrial vegetation (diet data from Yost et al. 1978 and Pahren et al. 1979). The consumption of fish and other products of aquatic rather than terrestrial origin is too small a component in the average American diet to account for a significant part of the overall population exposure.

Major pathways to agricultural topsoil are summarized in Figure 4. The potentially important pathways appear to be phosphate fertilizer, emissions deposition, irrigation water, and sewage sludge landspreading.

3.2.1 Phosphate Fertilizer

Phosphate fertilizer, projected by Versar (1979b) to be 400 mt/yr for 1980, appears to be the largest pathway. It is believed to affect nearly all of the 130-150 million hectares cropland. This contamination stems from cadmium's natural association with phosphate minerals. The cadmium content of phosphate rock is variable and depends on the origin of the phosphate. Western phosphates carry high concentrations of cadmium and thus result in more intense contamination than the more commonly used Eastern phosphates (Versar 1979b). The cadmium concentration in phosphates has been estimated as follows (Versar 1979b):

Region	Cd Concentration (µg/g)
Florida	10
North Carolina	20
Tennessee	3
Western	100

The production and use of phosphate ore is summarized in Figure 5. Western fertilizer has only about 6% of the market; however, its high cadmium content makes makes it responsible for around 40% of the total quantity of cadmium in fertilizer. Significant increases in the Western market share would result in significant increases in cadmium application to cropland. Although Versar did not anticipate large changes in the relative proportions of Eastern and Western market shares before year 2000, reserves in many Eastern mines are dwindling. While some shifts in supply will have to occur, ample world-wide reserves make it difficult to predict which sources will be tapped (Stowasser 1975, Emigh 1972).

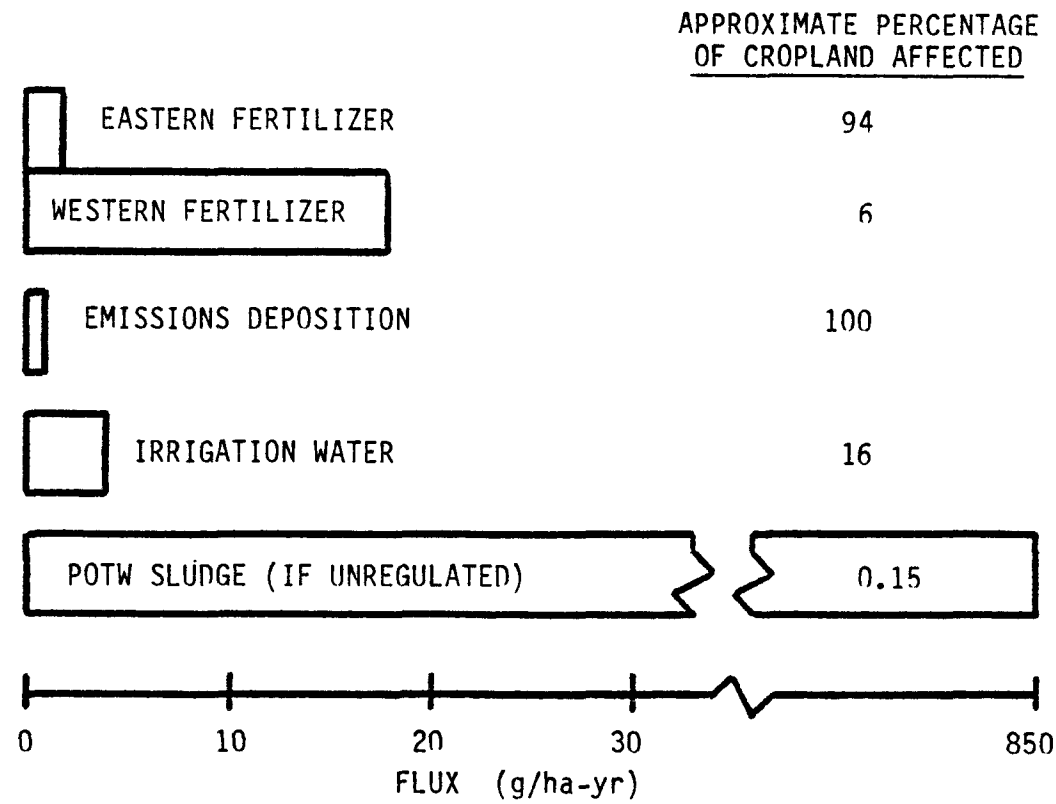

FIGURE 4a: ESTIMATED MEAN FLUX (APPLICATION OR DEPOSITION RATE) OF CADMIUM TO CROPLAND TOPSOIL

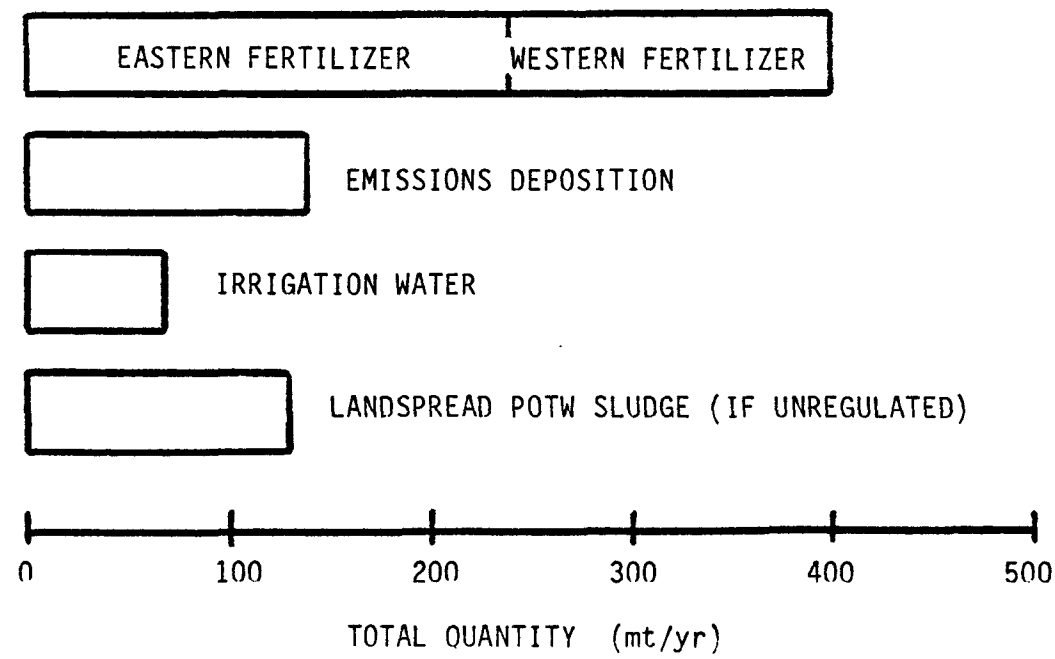

FIGURE 4b: NATIONWIDE QUANTITY OF CADMIUM REACHING CROPLAND TOPSOIL

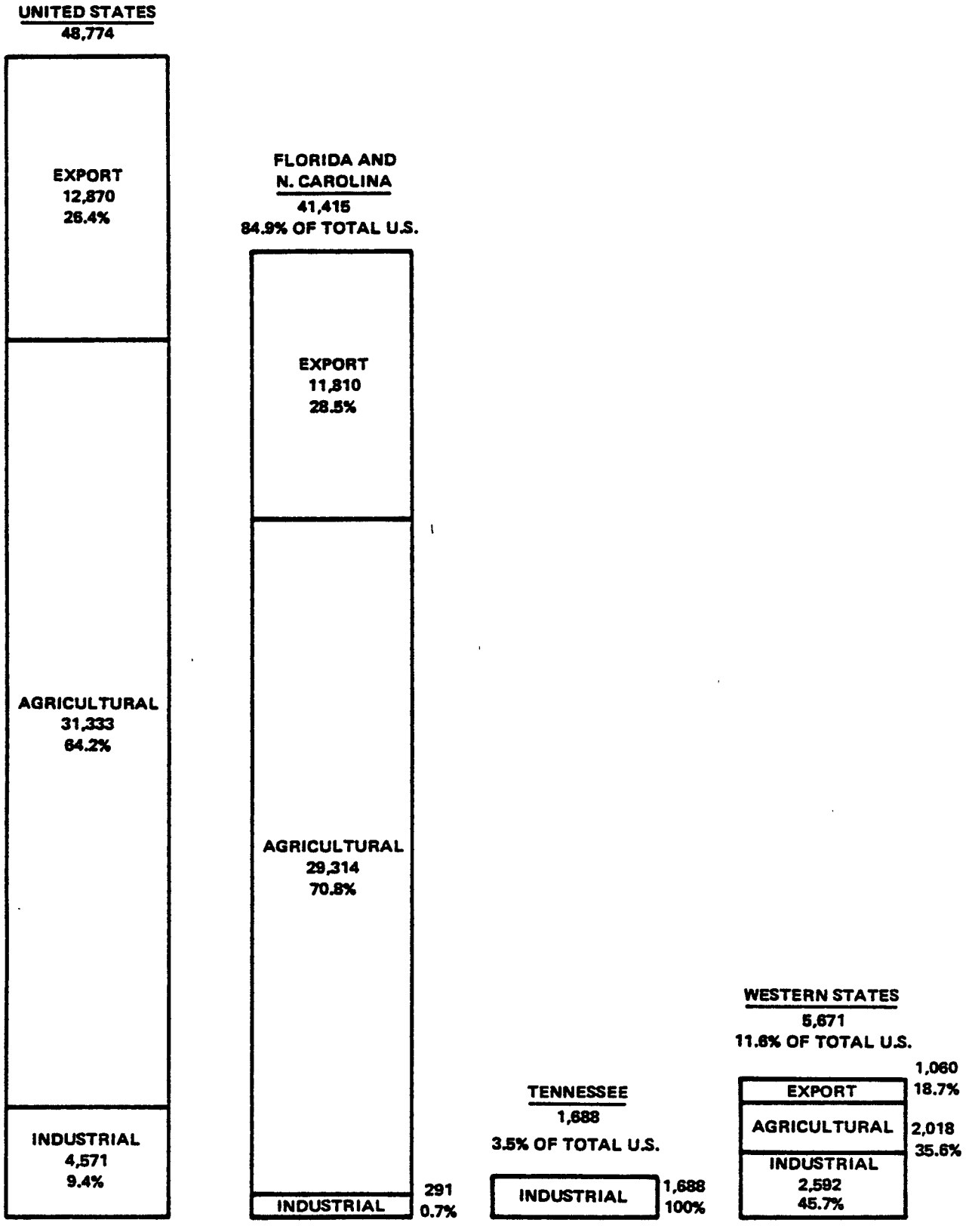

FIGURE 5 PHOSPHATE ROCK SOLD OR USED BY PRODUCERS – 1978
(THOUSANDS OF METRIC TONS)
(VERSAR 1979b)

3.2.2 Emissions Deposition

To varying degrees emissions deposition also affects essentially all of the 130-150 million hectares of cropland. Deposition fluxes are not accurately known, however, and have been roughly estimated here to average 1 g/ha-yr in rural areas. Deposition fluxes appear to be much higher in urban areas than in rural areas, with measurements often in the range 3-30 g/ha-yr (Nriagu 1980). Urban soils are also known to have higher cadmium concentrations than suburban soils (Carey et al. 1980). Near smelters deposition rates have been measured as high as 100-500 g/ha-yr in the 1970s (Nriagu 1980), although the more recent installation of pollution control technologies is believed to have greatly reduced such high rates.

It might be noted that the 1 g/ha-yr estimate is consistent with what would be calculated conservatively assuming that all U.S. emissions were deposited on the area of the continental U.S. Despite such a conservative assumption, however, measured cadmium deposition rates in rural areas are often several fold higher than the above estimate (Nriagu 1980). It is not clear whether such measurements simply reflect the wind blown movement of soil (with its associated cadmium) from one area to another, or whether they indicate actual enrichment of soil with new cadmium. Only the latter process is of concern.

The major emissions sources contributing to this pathway are believed to be coal and oil combustion and primary nonferrous metal industries (excluding zinc/cadmium smelting, formerly an important source, now apparently tightly controlled) as shown in Table 4. Incineration of municipal refuse is apparently a minor emissions source (as shown in Table 4), although refuse cadmium can be volatilized and potentially emitted at the relatively low temperature of 765°C. However, as only 5-10% of all municipal refuse is incinerated, the disposal of products with cadmium pigments, plastics, and batteries appears to have little potential for causing human exposure. (It should be noted, however, that Yost et al. (1980) postulate that a considerable amount of uncontrolled open burning of trash is occurring; under this assumption they identify such products as having significant exposure potential. The basis for expecting much uncontrolled trash burning is not clear, however.)

Although emissions deposition appears to be a potentially sizable route of exposure, its intensity is generally so low that it is not obvious exactly how it can cause specific individuals to exceed the kidney effects threshold. Even in the worst case situation of homegardening on soil contaminated with poorly controlled smelter emissions, exposure does not appear sufficient to induce kidney dysfunction in otherwise typical people.

3.2.3 Irrigation Water

While contaminated irrigation water was the cause of the Itai-Itai problem in Japan, irrigation water does not appear to be a major factor for cadmium contamination in the U.S. Unreliable data generated by the insensitive analytical methods commonly employed make estimations difficult; for Figure 4 the

99 billion gallons/day irrigation water consumed on 23 million hectares irrigated cropland (Versar 1979a) was assumed to have an average concentration of 0.5 ug/l. It was also assumed that the cadmium content of the consumed water was captured in the soil. (Water consumption equals water use minus return flow. It was thus assumed that the cadmium concentration in the return flow was the same as that originally in the irrigation water.) JRB (1980) has identified a few areas where a possibility for irrigation contamination exists: (a) the Snake River Valley in Idaho, (b) the Coeur d'Alene Valley in Washington, (c) Central Florida, and (d) Central California.

3.2.4 Sewage Sludge Landspreading

Landspreading of sewage sludge differs from the above pathways in that it can cause extremely intense contamination of very small areas. Of the 5 million mt/yr sludge produced (EPA 1979a), 31% is estimated to be landspread, 16% on food chain crops, 3% on non-food chain products, and 12% by distribution and marketing, (EPA 1980a).

Concentrations of cadmium in sewage sludge range from less than 1 ug/g to greater than 1000 ug/g. POTWs serving small non-industrial communities tend to have much lower sludge cadmium concentrations than do POTWs serving large industrial cities. Most POTWs are small and have relatively low cadmium concentrations. In the EPA (1979a) listing of 353 facilities, one-half of the facilities had concentrations not greater than 17 ug/g; in a more recent listing of 511 facilities (Booz-Allen 1982) one-half had concentrations not greater than 13 ug/g. (The latter more recent data set is considered more reliable; the former is thus considered to have a slightly high bias.) Neither data set may be representative of the sludge quality that might occur after implementation of pretreatment standards in some important industries.

The above medians are strongly influenced by many small POTWs that together produce relatively little sludge. Most of the sludge is produced by medium or large sized facilities and is somewhat more contaminated. For various cadmium concentrations Table 5 shows the associated cumulative (a) percentage volume of sludge, and (b) the percentage quantity of sludge cadmium. (Table 5 does not show the percentage of POTWs.) The production weighted mean concentration is 85 ug/g. These statistics are based on an analysis previously performed on the EPA (1979a) data set. Such statistics are not available for the Booz-Allen (1982) data set.

RCRA/CWA regulations will limit the rate of cadmium application on sludge disposal sites to 500 g/ha-yr. Sludge spreading rates for food chain crops are often in the neighborhood of 5-20 mt/ha-yr or more (CAST 1976, Yost et al. 1979, LaConde et al. 1978, EPA 1979a). Economics often may not favor sludge spreading at rates much lower than the bottom of this range. At a 5 mt/ha-yr sludge spreading rate, the sludge cadmium concentration could not exceed 100 ug/g without exceeding the RCRA/CWA 500 g/ha-yr cadmium application limit. Table 5 indicates that 73% of the sludge in the EPA (1979a) data set has concentration less than 100 ug/g and could thus be spread at a rate of 5 mt/ha-yr or greater. It also indicates that only 38% of the total quantity of cadmium

Table 5: Cadmium concentrations for cumulative nationwide percentages of (a) the sludge production volume and (b) the quantity of Cd in sludge. (From analysis of data for 353 POTWs listed by EPA 1979a.)

Cadmium Concentration (ug/g)	% of Sludge Volume with Concentration Less than	% of Sludge Cadmium at Concentration Less than
10	8	0.5
25	28	4
50	42	10
75	55	20
100	73	38
125	75	39
150	79	46
175	87	61
200	90	67
250	98	90
300	99	92
500	99	93
1200	100	100

Note: The above statistics are generated tabulating the concentration, c_i, and sludge annual production volume, S_i, for each of 353 facilities. The facilities were then rank ordered by increasing c_i, while adding up the cumulative sludge volume, ΣS_i, and cadmium quantity, $\Sigma S_i c_i$.

represented by this data set is contained in such sludges. Likewise, only 42% of the sludge volume and 10% of the sludge cadmium is contained in sludges having concentrations of less than 50 µg/g, the concentration limit for spreading sludge at the rate of 10 mt/ha-yr or greater.

The overall effectiveness of pretreatment regulations in reducing the concentration of cadmium in sewage sludge has not been estimated; however, pretreatment is believed to be bringing about substantial reductions in important industrial segments such as electroplaters. The general effect of such pretreatment on sludge landspreading can be illustrated as follows. Using the Table 5 statistics, it could be assumed for illustration that (a) 5-10 mt/ha-yr were the mimimum economically feasible application rate, and (b) pretreatment were to bring about a 60% reduction in sludge concentrations across-the-board. In this case 98% of the sludge volume (and 36% of the current cadmium quantity) in the data set could be spread at more than 5 mt/ha-yr; likewise, 75% of the sludge volume (and 16% of the current cadmium) could be spread at more than 10 mt/ha-yr. This hypothetical illustration indicates the general effects of pretreatment when coupled with RCRA/CWA landspreading regulations: pretreatment allows more communities to choose food chain landspreading as an economical disposal alternative. In so doing it is unlikely to bring about a substantial reduction in the total amount of cadmium thereby landspread.

The above described sludge landspreading regulations apply to disposal sites only. They do not apply to sludge which is either sold or given away to individual farmers or gardeners. This potentially critical exposure pathway is currently not regulated. It should be noted that the contamination of equivalent areas of commercial agricultural plots and home garden plots have inherently different results. As the contaminated commercial produce would be widely dispersed to many individuals, the result would be widespread low-level exposure. However, as the contaminated home garden produce could be a substantial portion of the diet of a particular household, the result would be high level exposure confined to a few individuals.

Ryan et al. (1982), using (a) crop uptake factors for sludge amended soils, and (b) Loma Linda University and adjusted FDA average diet data, evaluated a scenario of home gardeners growing the entire vegetable, grain, and fruit component of their diet on sludge amended soil. For example, on neutral pH soils they estimated that a cadmium addition of 8,000-12,000 g/ha would increase the gross ingestion of cadmium to 200 µg/day, a level often considered to be a hazard threshold. For acidic soils, plant uptake of cadmium is more pronounced, and the hazardous application rate somewhat lower.

EPA (1979d) coupled similar reasoning with slightly different data and assumptions. They considered home gardeners growing some portion of the vegetable component of their diet on sludge amended soils. The diet component included legume, leafy, and root vegetables, garden fruits, and potatoes; it excluded grains and regular fruits. Based on this analysis, EPA (1979e) promulgated cumulative application limits of 5,000-20,000 g/ha depending on pH and cation exchange capacity. EPA (1980a) calculated the sludge concentrations corresponding to a cadmium addition of 5,000 g/ha. For example, at a sludge spreading rate of 4 mt/ha-yr, application of sludge containing 54 µg/g

cadmium would result in a cumulative addition of 5,000 g/ha after 25 years. As shown in Table 5, much of the sewage sludge exceeds this concentration.

In conclusion, the total amount of sewage sludge cadmium expected to be landspread can be summarized as follows. Of the estimated 425 mt/yr sewage sludge cadmium, 51 mt/yr (12%) is believed to be disposed of by unregulated distribution and marketing, and will here be assumed to be landspread on crops. If unregulated, another 68 mt/yr (16%) could be landspread onto food chain crops at disposal sites; however, under RCRA/CWA sludge disposal regulations only about 7-26 mt/yr of this seems likely to be landspread. Industrial pretreatment is expected to reduce sludge cadmium concentrations; however, this change might be partially offset by increasing quantities of sludge landspread.

3.3 Long Term Implications of Contaminating Topsoil

The total quantity of cadmium that could reach cropland topsoil may thus be summarized roughly as follows: phosphate fertilizer 400 mt/yr, emissions deposition possibly as much as 140 mt/yr, irrigation water possibly as much as 70 mt/yr, and sewage sludge (under current landspreading regulations) possibly as much as 70 mt/yr. The annual additions would thus appear to total as much as 680 mt/yr.

3.3.1 Current Concentrations in Topsoil

Figure 6 illustrates the measured distribution of cadmium in soils of three states, Kansas, Montana, and Texas (data from Carey 1979). The median is 0.20 ug/g, the arithmetic mean 0.26 ug/g. The total amount of cropland is around 130-150 million hectares (CAST 1976, USDA 1981); they can be assumed to contain 2.2 million kg topsoil per hectare (EPA 1979d). The quantity of cadmium in cropland topsoil would thus be around 74,000-86,000 mt. The estimated annual addition of 680 mt/yr thus represents the addition of nearly 1% of the accumulated amount per year.

Other studies have found soil cadmium levels in this same general range. For example, Pierce et al. (1982) also happened to obtain an arithmetic mean of 0.26 ug/g for biodiagenetically available cadmium in soils of Minnesota. Of particular significance is their finding that the parent subsoils averaged only 0.13 ug/g, indicating cadmium enrichment of the topsoil by some means. It is not known whether such enrichment involves a natural concentrating process or whether it involves the deposition of cadmium onto topsoil.

3.3.2 Forecasting Changes in Soil Concentrations

Cadmium, being a stable element, has no half-life for destruction. Nevertheless, contaminated topsoil can eventually be removed from cropland by erosion. Based on the average U.S. agricultural soil loss rate of 18.7 mt/ha-yr (Center for Environmental Reporting 1979), the half-life of a six inch layer

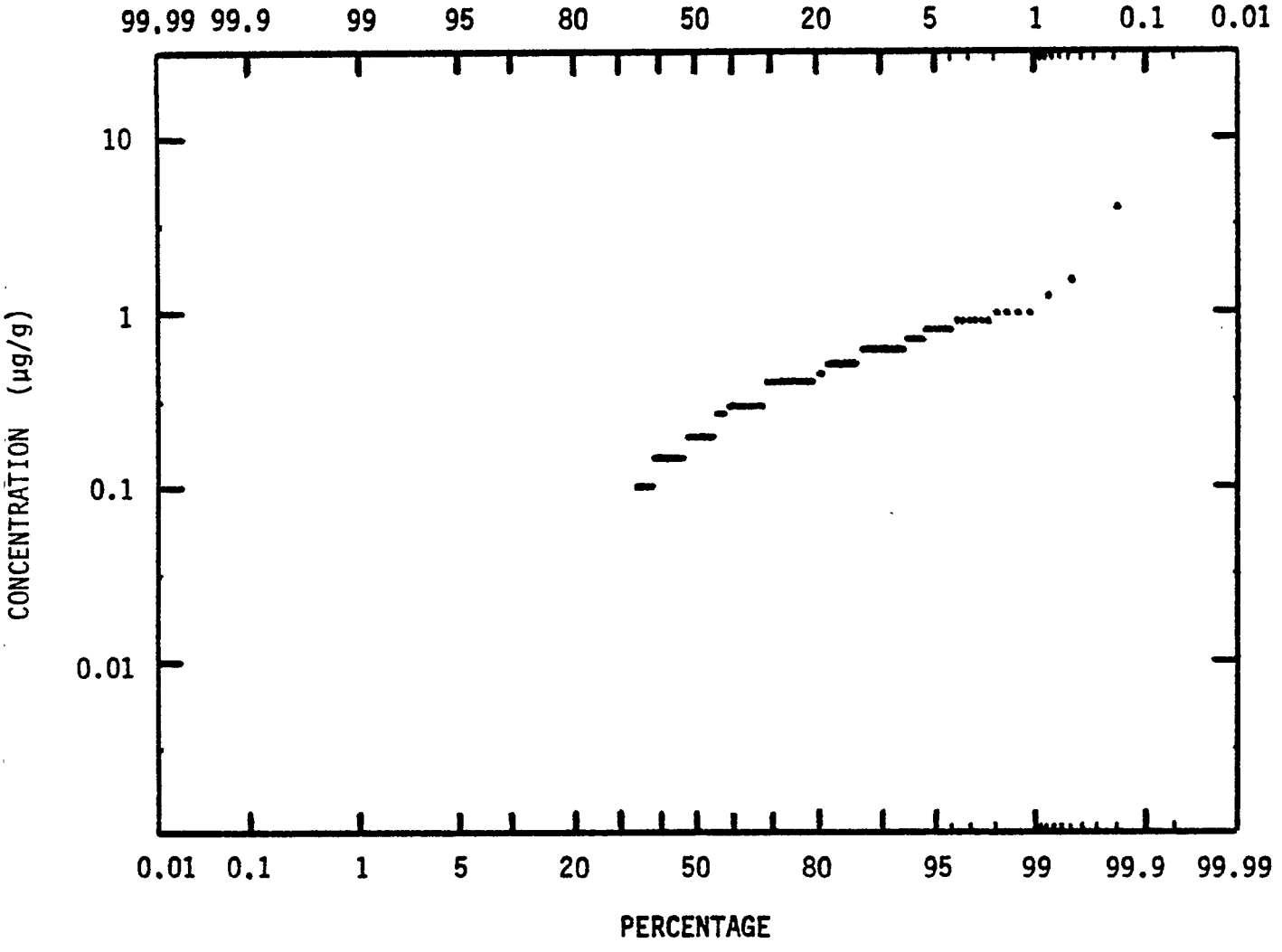

FIGURE 6: CADMIUM CONCENTRATION IN AGRICULTURAL SOILS: FREQUENCY DISTRIBUTION FOR 337 SAMPLES FROM THREE STATES.

of topsoil completely mixed by tillage would be estimated to be 80 years. Soil loss rates of different regions of the country differ considerably, however. Regional average half-lives may range from 29 years in the Lower Mississippi area to 1270 years in California. Site-to-site variability would be even greater.

As erosion may act to remove the cadmium applied or deposited on topsoil, it is apparent that the cadmium concentration will not build up indefinitely. Given a steady cadmium application flux, F (g/ha-yr), and soil erosion rate, E (mt/ha-yr), the cadmium concentration will eventually attain a steady-state concentration, c_{ss} (ug/g), readily calculable by recognizing that a well tilled 6 inch layer of topsoil is analogous to a completely mixed flow reactor:

$$c_{ss} = c_b + F/E \qquad (3\text{-}1)$$

where c_b is the cadmium concentration in the parent subsoil (beneath the topsoil). Table 6 shows the results of such calculations assuming that c_b=0.13 ug/g (as found by Pierce et al. 1982), and assuming various cadmium application rates and soil erosion rates.

It can be seen that long term build-up of cadmium levels can be quite pronounced at low erosion rates. At high erosion rates, on the other hand, the potential for accumulation of cadmium is limited. The table also presents calculations for the nationwide arithmetic and harmonic mean rates of soil erosion. Because the plateau concentration c_{ss} is related to the reciprocal of the erosion rate E, the nationwide arithmetic mean of c_{ss} is related to the harmonic (not arithmetic) mean of E (as indicated by Meyer 1975).

It must be noted that a substantial period of time may be needed to approach a steady state concentration. The time behavior of this type of system is given by O'Connor (1979):

$$c(t) = [c_b + F/E][1 - \exp(-tE/M)] + [c_o \exp(-tE/M)] \qquad (3\text{-}2)$$

where c_o is the initial concentration in topsoil, M is the quantity of soil (2200 mt/ha), and t is time period in years. Symbols F, E, and c_b are as previously defined. For a system where the topsoil concentration begins at the subsoil background concentration, given a value of 0.13 ug/g, and where E is 11.8 mt/ha-yr, and F is 5 g/ha-yr, a period of over 300 years would be needed to attain 90% of the steady-state concentration. (Attaining 100% of steady does not occur until t becomes infinite.) The lower concentration plateaus associated with rapid erosion rates can be approached more quickly, in as little as a few decades. The high concentration plateaus associated with slow erosion may require a few millennia to approach.

Finally, it is worth questioning whether the available data lend any support to the validity of analytical framework applied above (i.e., Equations 3-1 and 3-2). For Minnesota soils Pierce et al. (1982) found 0.26 ug/g to be the current average concentration in topsoil, and 0.13 ug/g to be the average concentration in subsoil. Assuming that the topsoil initial concentration, c_o, was equal to the subsoil background concentration, c_b, a prediction of the

Table 6: Steady-state Plateau Concentrations Predicted for Various Cadmium Application Rates and Soil Erosion Rates.

Regional Soil Loss Catagory	Soil Loss Rate, E (mt/ha-yr)	Calculated Soil Half-life (yr)	Steady-state Concentration, C_{ss} (ug/g), for Various Cd Application Fluxes, F		
			2 g/ha-yr	5 g/ha-yr	20 g/ha-yr
Low	1.2	1270	1.8	4.3	16.8
Harmonic mean	11.8	129	0.30	0.55	1.8
Arithmetic mean	18.7	81	0.24	0.40	1.2
High	52	29	0.17	0.23	0.51

Notes on Derivation of Tabled Values:

Soil erosion rates taken from data presented by Center for Environmental Reporting (1979). The harmonic mean is given by $n/ \Sigma 1/x$.

Half-life $t_{1/2} = 0.693/k$ where $k = -\ln(1 - E/M)$. E is the soil erosion rate in mt/ha-yr and M = 2200 mt/ha (soil quantity in 6 inch layer).

Steady state concentration $c_{ss} = c_b + F/E$ where $c_b = 0.13$ ug/g (the assumed concentration in the parent material), and F is the cadmium application flux in g/ha-yr. (If the total flux of deposited material (mt/ha-yr) (of which cadmium is a part) is significant compared to the soil erosion rate, then E is the sum of soil erosion rate plus total material deposition rate. For example, in sludge application this accounts for dilution of the cadmium in the sludge itself.)

F = 2 g/ha-yr is an application rate typically expected for Eastern phosphate fertilizer by itself.

F = 5 g/ha-yr is the estimated nationwide average application rate from fertilizer, emissions deposition, irrigation water, and sludge (assuming 680 mt/yr Cd on 140 million hectares cropland). This value may be biased high.

F = 20 g/ha-yr is an application rate typically expected for Western phosphate fertilizer by itself. It is also in the range of air deposition rates found in large cities. It is substantially less than often occurs during sludge application, however. Sludge application is not considered in this table because it would not usually be done for a sufficient period of time to attain a steady state.

current value of c(t) can be attempted using Equation 3-2. Lacking historical values for topsoil concentrations, cadmium deposition or application rates, and soil erosion rates, a true verification of the analytical framework is not possible. Nevertheless, it can be noted that the observed topsoil concentration of 0.26 ug/g is consistent with what would be calculated for parameter values that seem reasonable for Minnesota: an application rate of 4-5 g/ha-yr, an erosion rate of 5-15 mt/ha-yr, and a time period of 50-100 years. It must be cautioned, however, that other unrelated processes could account for the observed differences between topsoil and subsoil concentrations.

3.3.3 Influence of Topsoil Contamination on Human Exposure

The intent of the following analysis is to estimate the dietary intake corresponding to growing food on soils of various cadmium concentrations. Ryan et al. (1982), for a similar purpose, coupled crop uptake factors (ug/g in crop versus ug/g or g/ha in soil) with FDA data on diet composition. They discerned between acidic and neutral soils, and average and vegetarian subgroup diets. Due to lack of data on many crops, the needed uptake factors are somewhat uncertain.

To be consistent with the current study's preference for using EPA fecal data rather than FDA food data to estimate cadmium exposure (as discussed in Section 2.3), this analysis will approach the problem by a somewhat different route. It rests on the following basic premises:

(a) Crop uptake of cadmium is taken to be directly proportional to the topsoil cadmium concentration (although the constant of proportionality may differ from crop to crop and from soil to soil).

(b) The nationwide arithmetic mean dietary exposure can be estimated from fecal cadmium measurements (Kowal et al. 1979); its value is taken to be 14 ug/day gross quantity ingested or 0.85 ug/day absorbed dose.

(c) The nationwide arithmetic mean soil concentration can be estimated from available data (Carey 1979); its value is taken to be 0.26 ug/day.

(d) An arithmetic mean exposure would result from consuming an average diet grown on soils having average cadmium concentration (and average other properties). That is, the population's sampled dietary exposure is the result of consuming foods grown on soils having the sampled concentrations.

The above premise (a) is technically sensitive. One problem is that the assumed linearity seems unlikely to extend over a wide range of concentrations. This may not be critical, however, since this analysis seeks only to apply it over a fairly narrow range, with mean topsoil concentrations increasing perhaps 2-3 fold over present values. Another problem is that it implies that cadmium's bioavailability does not depend on the pathway by which it entered topsoil (for example, via emissions deposition, sewage sludge, or parent subsoil). While it has been found that initial bioavailability varies with the chemical

form being added (as discussed by Logan and Chaney 1984), it is not known whether such differences persist through the long time frames being considered here (i.e., after a long period of equilibration). Finally, the above premise implies that it is the cadmium content of the topsoil, not the subsoil, that effectively determines the cadmium content of the crop, even though the root zone of many crops may extend somewhat below the topsoil.

This approach does not assume that all combinations of crops and soils have the same cadmium uptake factor (µg/g in crop verses ug/g in soil), but only that such a proportionality would exist for each crop and soil type. This approach is used here only for approximating average relationships between soil contamination and dietary exposure. It is not believed to be suitable for discerning the effect of certain characteristics, such as site-specific pH, differing markedly from the average.

It is desirable, in any case, to consider how various portions of the average diet are affected by cadmium enriched soils. For example, in evaluating the risks of home gardening scenarios, it is customary to assume that only certain classes of food are garden grown. Consequently, it is necessary to discern the importance of each food class in the average diet. Such information is provided by Pahren et al. (1979) and Ryan et al. (1982). Table 7 summarizes the results in terms of relative contribution of each food class to total dietary exposure to cadmium in the average diet. It can be seen that the responsibility for cadmium exposure is spread broadly across food classes.

Three cases have been considered in this analysis. In Case 1 all food classes have been assumed to be affected. This may be most relevant in evaluating the effects of raising nationwide average soil concentrations. In this case total dietary exposure is estimated to increase in proportion with the increase in soil concentration. There is some question, however, about whether the cadmium contents of all foods, for example, those of animal origin, depend on soil cadmium levels (Logan and Chaney 1984).

Consequently, in Case 2 only leafy, legume, and root vegetables, garden fruits, potatoes, grains, and fruits have been assumed to respond to changes in soil cadmium. As shown in Table 7, these food classes currently contribute 67% of the average dietary exposure. Exposure through these food classes is estimated to increase in proportion to the increase in soil cadmium. Exposure through the other food classes is assumed to remain at current levels.

In Case 3 only leafy, legume, and root vegetables, garden fruits, and potatoes have been assumed to be affected. This is intended to correspond to a scenario of serious gardeners home growing their entire consumption of these food classes. As shown in Table 7, these food classes currently contribute 38% of the average dietary exposure. The other food classes are assumed to remain at current levels.

The total dietary absorbed dose, D (µg/day), is calculated as follows:

$$D = (f_C D_C / c_C)c + (1-f_C)D_C \qquad (3\text{-}3)$$

Table 7: Relative Contributions of Food Classes to Cadmium Ingestion in the Average Diet (adapted from Pahren et al. 1979, and Ryan et al. 1982).

Food Class	% of Total Dietary Exposure
Dairy products	11
Meat, fish, poultry	11
Grain and cereal products	27
Potatoes	24
Leafy vegetables	6
Legume vegetables	1
Root vegetables	3
Garden fruits	4
Fruits	2
Oils, fats, shortenings	3
Sugars and adjuncts	2
Beverages	6
Total diet	100

Table 8: Dietary Absorbed Dose Corresponding to Consuming Food Grown on Soils of Varying Cadmium Concentration.

Topsoil Cadmium (ug/g)	Total Dietary Absorbed Dose (ug/day)		
	Case 1	Case 2	Case 3
0.26	0.85	0.85	0.85
0.55	1.8	1.5	1.2
2.0	6.5	4.7	3.0
5.0	16.3	11.2	6.7
10.0	32.7	22.2	13.0

Case 1 assumes that all food classes respond to changes in soil cadmium; total dietary exposure is thus proportional to soil concentration.

Case 2 assumes that leafy, legume, and root vegetables, garden fruits, potatoes, grains, and fruits respond to changes in soil cadmium. Exposure through other food classes remains at current levels.

Case 3 assumes that leafy, legume, and root vegetables, garden fruits, and potatoes are grown on the affected soil. Exposure through other food classes remains at current levels.

where f_c is the fraction of cadmium currently coming from the affected food classes (from Table 7), D_c is the current average absorbed dose via diet (taken to be 0.85 ug/day), and c_c is the current average concentration of cadmium in soil (taken to be 0.26 ug/g), and c is the concentration of cadmium in the soil on which the affected food classes are grown.

Table 8 shows the calculated exposure level corresponding to various soil concentrations. If the hazardous exposure level for kidney effects were taken to be 12 ug/day absorbed dose, the corresponding hazardous soil concentration would be calculated to be 3.7 ug/g for Case 1 (all foods affected), 5.4 ug/g for Case 2 (leafy, legume, and root vegetables, garden fruits, potatoes, grains, and fruits affected), and 11.5 ug/g for Case 3 (leafy, legume, and root vegetables, garden fruits, and potatoes affected).

It is of interest to compare the results of this analysis with the results of Ryan et al. (1982), who evaluated the equivalent of Case 2 by a different method: an "integrated response curve" crop uptake model, coupled with FDA diet data. In order to make such a comparison, the units of Table 8 were converted to express ug/g gross ingestion per g/ha increase in soil cadmium (over current levels). To account for different assumptions about current exposure, the Table 8 and Ryan results were then normalized to yield the ug/day increase in gross ingestion per g/ha increase in soil cadmium. The comparison indicated that the results of the two methods differed by not more than 20%. That the results were this close must be viewed as somewhat fortuitous, since the two methods incorporate different sets of errors and uncertanties which must be expected to exceed 20% percent. Nevertheless, that the two methods produce nearly the same result lends confidence to the validity of both.

These results were also compared with the results of EPA (1979d), which evaluated the equivalent of Case 3 by a method somewhat related to that of Ryan et al. (1982). Compared to the nationwide average increase in exposure predicted by the present approach, EPA (1979d) predicted lesser increases for neutral soils and greater increases for acid soils. The differences were often roughly two fold.

In coupling the results of Tables 6 and 8, in order to estimate the dietary exposure resulting from various cadmium application rates, it should be noted that Cases 1 and 2 of Table 8 are probably appropriate for coupling with average application and erosion conditions of Table 6. Case 3, on the other hand, may be appropriate for a home gardening scenario, which might under worst case conditions have high application rates and low erosion rates. It must be noted, in any case, that the time period needed to approach the steady-state concentration plateau is perhaps 2-3 fold greater than the erosional half-life of the soil. For typical erosion rates this time period is thus on the order of centuries. Such a planning horizon is greater than ordinarily applied to pollution control problems. However, as soil contamination by metals is often considered to be irreversible within ordinary planning horizons (Purves 1977), such a long range perspective may be appropriate.

In order to predict the effect of soil contamination on kidney cadmium levels and the incidence of toxicity, some assumptions in addition to the

previously stated premises (a)-(d) must be invoked:

(e) The nationwide distribution of kidney cadmium concentrations can be estimated from available data. For nonsmokers aged greater than 30 years, the distribution is taken to be log-normal, with geometric mean 15.0 ug/g (Johnson et al. 1978). The geometric dispersion of 1.74 is assumed to be unchanging.

(f) The kidney cadmium concentrations measured in the population can be taken to be the linear result of the measured exposures via air, food, and drinking water.

Considering premises (a)-(f) together, it can be seen that this method projects nationwide statistics from the statistics of the measured samples. The kidney cadmium levels are assumed to be a function of the exposure via air, food, and drinking water. The exposure via food is assumed to be a function of the soil cadmium concentrations. Exposure via air is assumed not to change with changes in soil cadmium levels. Exposure via drinking water is negligible.

The method thus does not explicitly employ measured soil-to-crop uptake factors. However, an overall uptake factor is implicit in linking the Carey (1978) soil data with the Kowel et al. (1979) dietary exposure data. Likewise, the method does not explicitly state a hazardous threshold for absorbed dose. Nevertheless, by linking the Kowel dietary exposure data (with minor additional exposure via air) with the Johnson et al. (1978) kidney cadmium data, a long-term absorbed dose of approximately 10 ug/day is implicitly made to correspond with with a kidney cadmium level of 200 ug/g.

The method can be applied to values derived in Table 6. For a cadmium deposition flux of 5 g/ha-yr, considered here to be a slightly high biased estimate of the nationwide cropland average, coupled with the erosion rate of 11.8 mt/ha-yr, the estimated nationwide harmonic mean, the average cadmium concentration in the topsoil is predicted to attain a steady-state plateau of 0.55 ug/g (Table 6), 2.1 fold higher than the current mean. The dietary absorbed dose corresponding to this concentration, shown in Table 8, is 1.5 ug/day for Case 2. After adding the exposure through air, contributing only 0.02 ug/day, the factor increase in arithmetic mean exposure by all routes (excluding tobacco) would be 1.74. Assuming no change in the geometric dispersion, this same factor increase must apply to the geometric mean. As long term exposure is reflected by kidney cadmium concentrations, the geometric mean kidney concentration for a nonsmoking population aged greater than 30 years would be increased to 26 ug/g under Case 2 assumptions.

For any particular geometric mean concentration of cadmium in kidney, the fraction of the population exceeding the 200 ug/g kidney cadmium threshold can be calculated from Equations 2-2 and 2-1 (in Section 2.2.1) for a log-normal distribution. Assuming no change in the geometric standard deviation, the above increase in geometric mean would be predicted to increase the incidence from the currently estimated 1.5 persons per million up to 120 persons per million.

Figure 7 shows the general relationship between topsoil cadmium concentration and the incidence of exceeeding a kidney dysfunction threshold of 200 ug/g in the nonsmoking population aged greater than 30 years. Table 9 shows the mathematical formulation of the model. Incidence in the smoking population should exceed that in the nonsmoking population; however, quantitative predictions have not been attempted, primarily because the distribution of kidney cadmium does not appear to be log-normal in the smoking population.

Finally, to complete the analysis of the risks of soil contamination, it should be noted that there is always some possibility that cadmium may at some future time be judged to be carcinogenic via ingestion. Predicting the potential increase in cancer risk resulting from increases in soil cadmium is straight forward, given the results presented above. Under linear nonthreshold assumptions (or multi-stage assumptions approximating linear nonthreshold), the increase in cancer incidence is directly proportional to the previously shown increase in mean exposure.

3.3.4 Predictive Uncertainties

In summary, the above analysis predicts the ultimate result of practices pursued over a long period of time. For a cadmium deposition rate and soil erosion rate estimated to correspond to the nationwide means, the population mean exposure by all exposure routes is projected to increase by nearly two fold before attaining a plateau. Such an increase in mean exposure is projected to increase the number of persons at risk for cadmium induced β_2-microglobulin proteinuria by 80 fold.

The importance of the parameters used to predict changes in the mean accumulated exposure among nonsmokers was determined by sensitivity analysis. In such an analysis the value of each parameter is varied while the other parameters are held constant. The long-term factor increase in the mean concentration of kidney cadmium (K/K_C) was most sensitive to potential errors in estimating the current mean concentration of cadmium in topsoil (c_C), the flux of cadmium onto cropland (F), and the erosion rate (E). It was less sensitive to potential errors in the fraction of current intake from affected food classes (f_C) and the mean concentration in subsoil (c_b). It was insensitive to the current air and dietary intakes (A_C and D_C).

The propagation of the combined uncertainties of the important parameters, c_C, F, E, f_C, and c_b, was evaluated by first-order error analysis (a procedure described by Reckhow and Chapra 1983, and Meyer 1975). In such an analysis the variance, s_y^2, in the predicted values of a dependent variable, y, is related to the variance in reasonable estimates for the each parameter or independent variable. For this application the dependent variable is the factor increase in mean kidney cadmium levels ($y=K/K_C$). Assuming that estimates for c_C and c_b are correlated, and estimates for the other parameters are not correlated, the uncertainty in y is approximated by the expression:

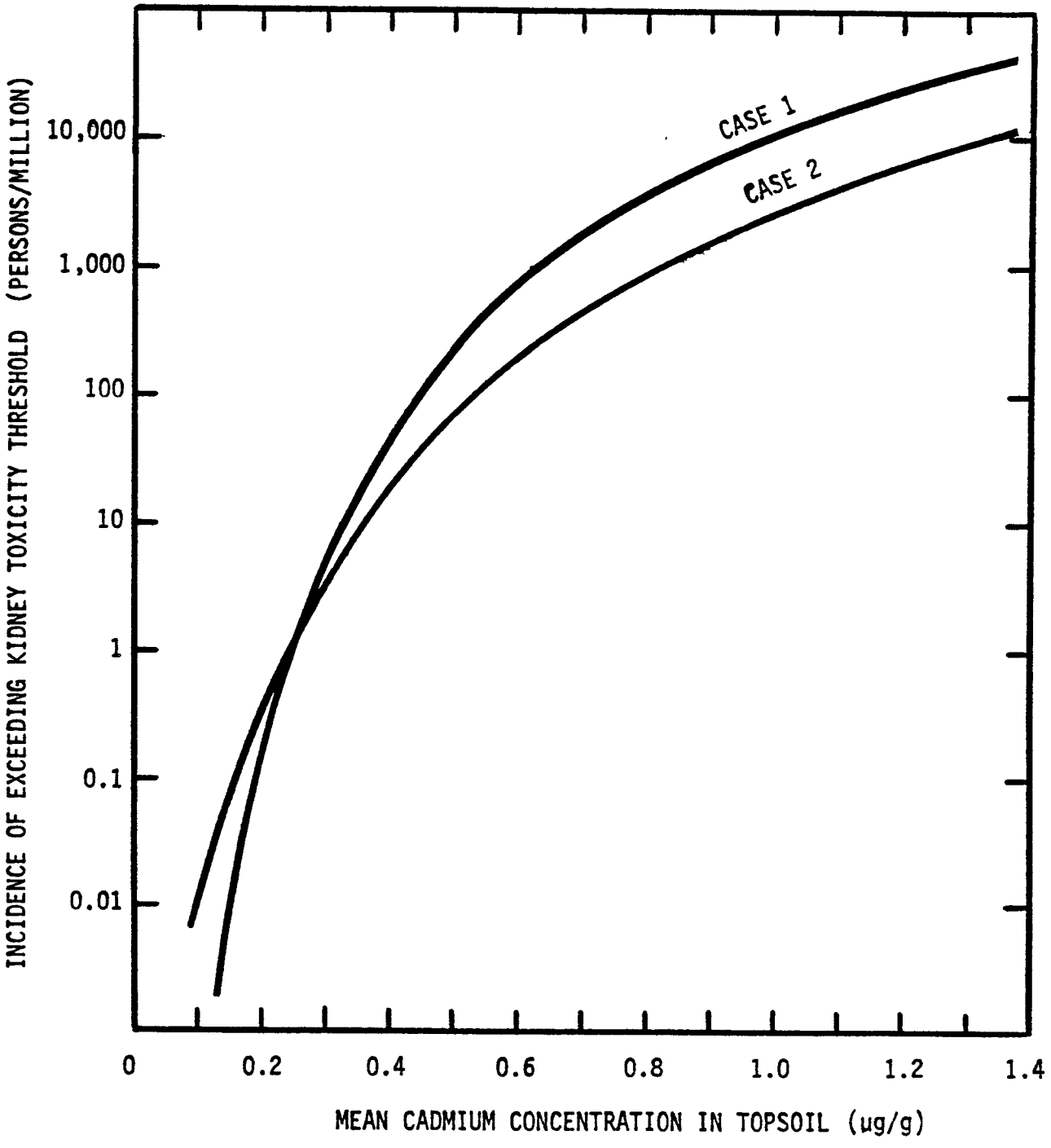

FIGURE 7: PROJECTED RELATIONSHIP BETWEEN NATIONWIDE MEAN CONCENTRATION OF CADMIUM IN CROPLAND TOPSOIL AND INCIDENCE OF EXCEEDING A KIDNEY CADMIUM CONCENTRATION OF 200 µg/g.

Note: The incidence is for a nonsmoking population aged greater than 30 years. The method of calculation is presented in Table 9.

Table 9: Formulations Setting forth a Relationship between Soil Cadmium Levels and the Incidence of Exceeding a Kidney Dysfunction Threshold.

$$D = (f_c\ D_c/c_c)\ c\ +\ (1 - f_c)\ D_c$$

$$K = K_c\ (D + A_c)/(D_c + A_c)$$

$$Z = \ln(K_t/K)/\ln s_g$$

$$Q = (1/\sqrt{2\pi})\ \int_Z^\infty \exp(-z^2/2)\ dz$$

Where:

c = Future arithmetic mean concentration (µg/g) of cadmium in cropland topsoil. This is the independent variable in Figure 7. Its value could also be predicted as shown in Table 6.

c_c = Current arithmetic mean concentration of cadmium in cropland topsoil (estimated to be 0.26 µg/g from Section 3.3.1).

f_c = Fraction of current cadmium dietary intake from affected food classes (f_c=1.0 for Case 1, and f_c=0.67 for Case 2, as noted in Section 3.3.3).

D = Projected arithmetic mean dietary absorbed dose (µg/day) resulting from consuming food grown on soils having mean concentration c.

D_c = Current arithmetic mean dietary absorbed dose; i.e, mean dietary absorbed dose resulting from consuming food grown on soils having mean concentration c_c. (D_c is estimated to be 0.85 µg/day from Table 2.)

A_c = Current arithmetic mean absorbed dose via air (estimated to be 0.02 µg/day from Table 2.) Exposure via drinking water is assumed negligible.

K = Projected geometric mean concentration (µg/g) of cadmium in kidney for a nonsmoking population aged greater than 30 years. This population would have an arithmetic mean absorbed dose of D via food, and A_c via air.

K_c = Current geometric mean concentration of cadmium in kidney for nonsmoking population aged greater than 30 years; i.e., geometric mean kidney concentration occurring in a population having an arithmetic mean absorbed dose of D_c via food, and A_c via air. (K_c is estimated to be 15.0 µg/g from Table 1.)

K_t = Toxicity threshold for cadmium in kidney (taken to be 200 µg/g).

s_g = Geometric standard deviation kidney cadmium concentrations (estimated to be 1.74 from Table 1).

Z = log-normal deviate.

Q = Projected incidence of exceeding the threshold K_t in a population aged greater than 30 years, with kidney cadmium levels log-normally distributed with geometric mean K and geometric standard deviation s_g. Q is the dependent variable in Figure 7.

$$s_y^2 = (dy/dF)^2 s_F^2 + (dy/dE)^2 s_E^2 + (dy/dc_b)^2 s_{c_b}^2 + (dy/dc_c)^2 s_{c_c}^2 + (dy/df_c)^2 s_{f_c}^2$$
$$+ 2(dy/dc_b)(dy/dc_c)s_{c_b} s_{c_c} r_{c_b c_c} \quad (3\text{-}4)$$

where dy/dx is the partial derivative of y with respect to each parameter "x" (i.e., the sensitivity of y to potential errors in that parameter), s_x^2 is the variance in a set of reasonable estimates for the value of each parameter, and r is the correlation coefficient between two parameters.

A rigorous determination of the variance in parameter estimates seems infeasible for this application, in part because the data used to derive the estimates of c_c, F, E, f_c, and c_b are not based on a random sampling. In place of a rigorous determination, the level of uncertainty was illustrated using parameter variance estimates that seem subjectively reasonable (as suggested by EPA 1984b). For estimates of c_c, c_b, and F, the coefficient of variation ($s/\bar{x}$) was taken to be 0.5; for estimates of f_c (Case 2) and E the coefficient of variation was taken to be 0.2. Parameters c_c and c_b were assumed to have a correlation coefficient of 0.75.

It was previously noted that the use of the "best estimates" for F, E, c_b, c_c, and f_c (for Case 2) indicated that, over a long period of time, kidney cadmium would plateau at levels 1.74 fold higher than at present. For the subjectively selected variances, the results of the analysis suggest that the 95% confidence interval for this predicted factor increase, y, might range from less than 1 to almost 4, assuming that the predictions of y are roughly log-normally distributed. The probability of no long-term increase in mean levels of kidney cadmium (i.e., $y \leq 1$) was estimated to be 17%.

First-order error analysis does not include the uncertainty in the validity of the model framework itself. In this respect one important question is the validity of assuming that the effective depth of the roots' nutrient uptake zone is not substantially greater than the depth of cultivation. If a substantial portion of a crop's cadmium burden is due to uptake a background cadmium in the subsoil, then the crop burden would not be proportional to the quantity of cadmium in the topsoil. In that case the above analysis would overestimate the increases in dietary cadmium.

In addition, this first-order analysis has not been extended to the uncertainty in the incidence of exceeding a toxic threshold (or the uncertainty in the factor increase in the incidence). As shown in Section 2.2.2, the predicted incidence is very sensitive to uncertainty in the geometric mean and geometric dispersion of the kidney cadmium distribution.

Finally, it should also be reemphasized that the Figure 7 relationship between mean topsoil cadmium and kidney toxicity incidence assumes that the kidney cadmium distribution is log-normal and that its geometric dispersion (or arithmetic coefficient of variation) is unchanging. If, however, the relative variability of soil cadmium were also to increase in response to contamination, then some type of increase in population variability of kidney cadmium concentrations might also be hypothesized to occur. Figure 8 presents

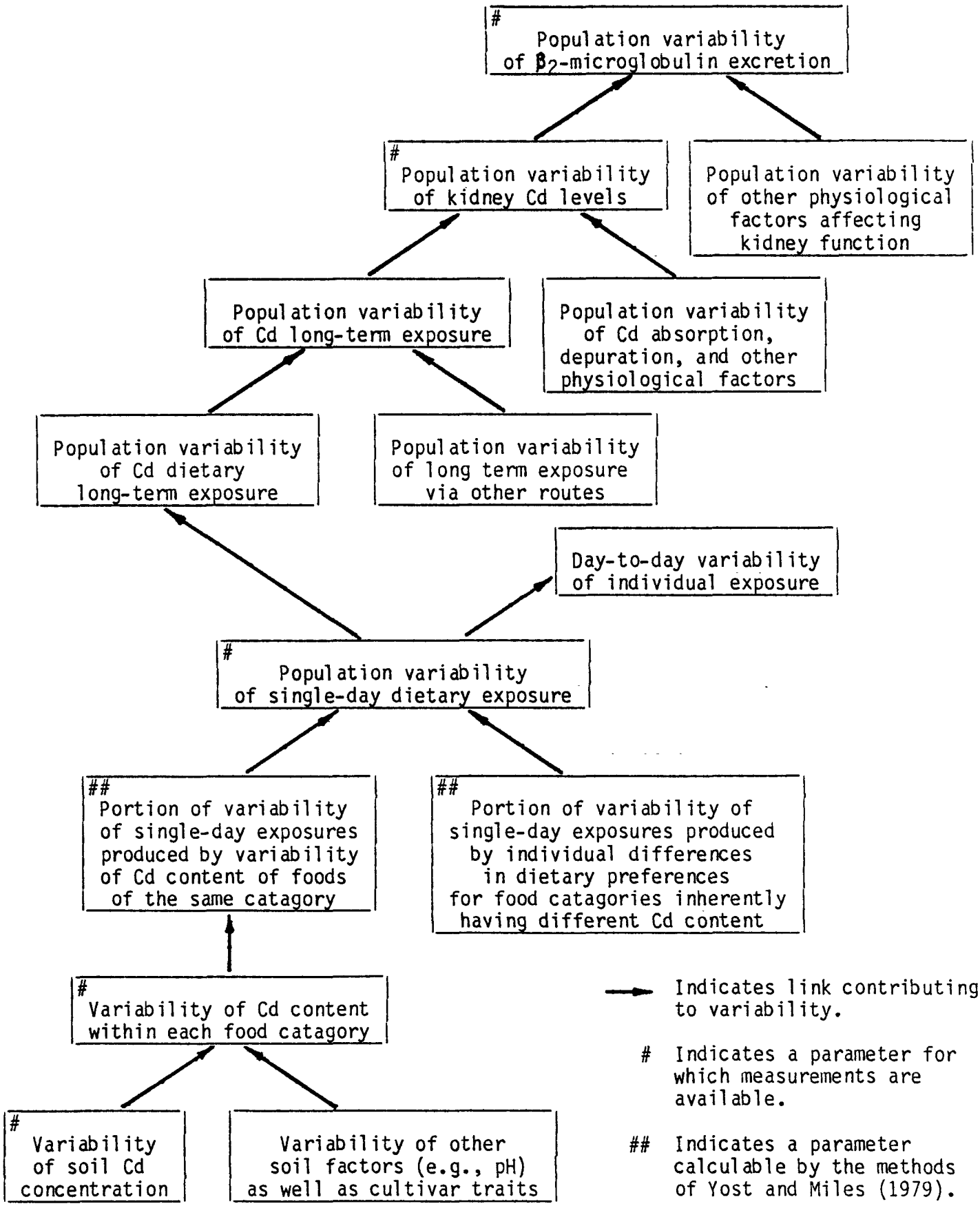

Figure 8: Hypothetical Relationships between Variability in Kidney Function, Long and Short Term Exposure, Food Concentrations, and Soil Concentrations.

a hypothetical framework relating the variabilities of cadmium concentrations and exposures. Such a framework can be used in a qualitative sense only, however, because some of the relationships between parameters cannot be described quantitatively by existing data.

3.4 Other Pathways of Exposure

In addition to the above mentioned critical exposure pathways involving topsoil there are some minor pathways. Direct inhalation of airborne cadmium is of minor significance, as discussed in a previous section. Emissions deposition may constitute a larger portion of topsoil (and thus food) contamination than emissions inhalation contributes to total exposure, as illustrated by comparing the importance of air in Figures 3 and 4b.

Direct ingestion of waterborne cadmium likewise was shown to be generally insignificant (Figure 3). The major releases to surface water would thus appear to have little potential for bringing about hazardous exposure through drinking water.

3.4.1 Bioconcentration in Shellfish

Of somewhat greater concern, however, is that waterborne cadmium may enter the diet after bioconcentration by shellfish. A seafood survey by Zook (1976), as cited by Drury and Hammons (1979), found the cadmium concentration of commercially caught Atlantic coast shellfish to average 0.575 ug/g, and Pacific and Gulf coast shellfish to average 0.077 ug/g. Data from a subsequent survey indicated an average of 0.55 ug/g in 11 types of shellfish (Meaburn et al. 1981).

While shellfish consumption is too small a portion of the average diet for this pathway to have much influence on average exposure, the pathway can be important for individuals if a significant portion of their diet consists of shellfish. Selected data on U.S. per capita fish consumption is shown in Table 10 (Javitz 1980).

Table 10: Selected Data on U.S. Per Capita Consumption of Fish and Shellfish.

Catagory	Consumption (g/day) Mean	Upper 95th Percentile
Total U.S.	14.3	41.7
Oriental racial identity	21.0	67.3
Large central city residents	19.0	55.6
New England residents	16.3	46.5
Mid-Atlantic residents	16.2	47.8
Higher income family members	16.7	49.0

It can be seen that individuals in the upper 95th percentile of the Oriental subgroup consume 67 g/day of fish and seafood. In the unlikely situation that all of this were in the form of Atlantic coast shellfish, averaging 0.575 ug/g cadmium, this exposure pathway would increase absorbed dose by 2.2 ug/day (assuming 6% absorption and subtracting the cadmium content of the diet component replaced by such seafood). Such exposure is somewhat above the median absorbed dose of 0.67 ug/day but well below the probable kidney hazard level of 10-15 ug/day.

3.4.2 Landfilled Cadmium

The bulk of the cadmium handled by man (both as a commodity and as an impurity) is believed to end up in municipal and industrial landfill. A survey of eight municipal landfills, however, suggests that such landfills do not usually result in groundwater contaminated with hazardous levels of cadmium (Fielding et al. 1981). Concern about industrial solid waste disposal has been greater, although a survey of 50 such sites did not implicate cadmium as one of the metals of concern (U.S. EPA 1977). Although cadmium may be mobile in leachate through some soils (Fuller et al. 1979), such that precautions could be appropriate for some solid wastes, it may be noted that exposure through drinking water in general appears to be very minor (as noted in Table 2). Consequently, as cadmium cannot be destroyed, an appropriate overall strategy might thus aim to steer cadmium away from air, surface water, and topsoil, but rather toward the deeper soil strata, where its potential for return to the biosphere is more restricted.

SECTION 4

ECOLOGICAL CONSIDERATIONS

The toxic effects of cadmium on terrestrial ecosystems have not been widely studied, apparently because the level of concern has not been high. Such ecosystems will not be covered here. Cadmium toxicity to aquatic ecosystems, on the other hand, has received considerable attention. Some aspects are discussed below.

4.1 Aquatic Life Exposure and Effects

The criteria for protection of aquatic life are being revised; the proposed freshwater values depend on water hardness and apply to both acute and chronic exposures (EPA 1984):

Hardness (mg/L $CaCO_3$)	Acute-Chronic Criterion (ug/L)
10	0.31
50	2.0
100	4.5
200	10.
400	22.

The proposed criterion appears to be fully protective of sensitive fish species, despite being about 200 fold higher than the 1980 chronic criterion. The saltwater criteria are 38 ug/L for acute exposure and 12 ug/L for chronic exposure. Both fresh and saltwater criteria are now expressed in terms of "active" cadmium, signifying dissolved plus readily desorbed metal. Recent ambient data, however, are often only available for "total" cadmium, of which "active" cadmium is only a portion.

The levels of cadmium (and several other metals) in natural waters tend to be low relative to the sensitivity of the analytical approaches commonly used and relative to the level of laboratory contamination possible if meticulous procedures are not used. When sensitive methods are applied with care, typical levels of cadmium found in fresh waters appear to fall in the range 0.01 - 0.1 ug/L (Martin et al. 1981).

Frequency distributions of STORET cadmium data are shown in Figure 9. The distribution of all STORET data (available before 1979) suggests a geometric mean of perhaps 0.1 ug/L (by extrapolation), and a large geometric dispersion of around 17. It further suggests that the freshwater criterion may be exceeded around 10% of the time. These data are not considered to be reliable, however, since the level of noise in the analytical methods may exceed the typical

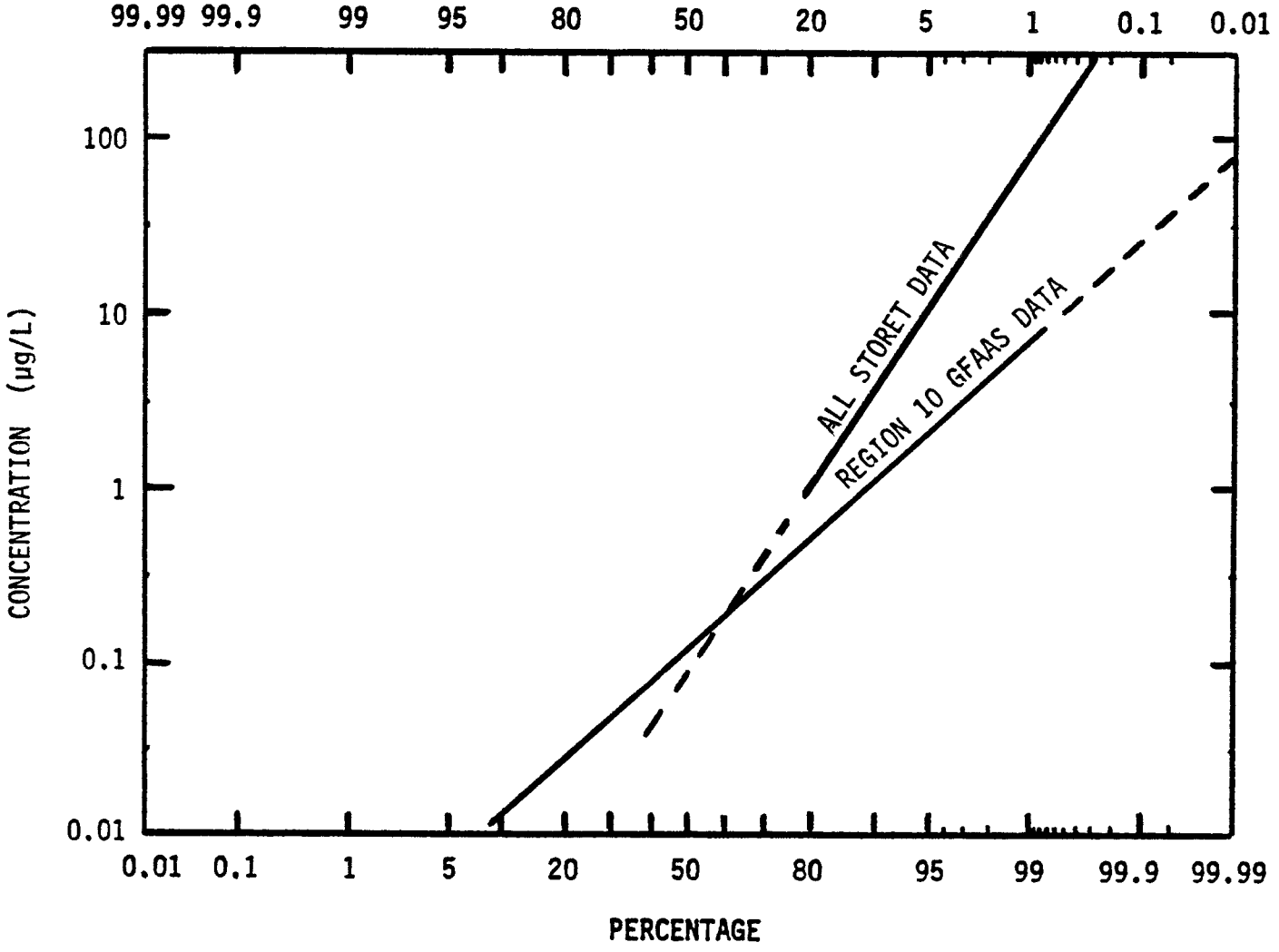

FIGURE 9: CADMIUM CONCENTRATIONS IN AMBIENT SURFACE WATERS: FREQUENCY DISTRIBUTIONS OF STORET DATA.

ambient levels by one to two orders of magnitude. Data checking by the Office of Toxic Substances and the Office of Water Regulations and Standards has revealed systematic errors that produce a large positive bias in major portions of the STORET cadmium data base. In addition, methods analyses indicate a substantial potential for positive bias when measuring cadmium below or near the detection limit with the older procedures. The frequent reporting of cadmium in the ug/L range may thus have little significance (Delos 1981).

In an attempt to circumvent the bias stemming from high detection limits, EPA Regional Office data obtained using the sensitive graphite furnace (GFAAS) method were extracted from STORET. Such data were associated with Region 10 from May 1978 to the time of the study (March 1981). The Region 10 GFAAS data show less variability than the nationwide data, even though the geometric means appear to be almost identical. Furthermore, despite representation of the Coeur D'Alene cadmium mining and smelting area in the data set, none of the 73 values exceeded 10 ug/L. At the hardness levels most commonly found in the U.S. as a whole (50-300 mg/L) such a distribution might exceed the freshwater criterion about 2% of the time. At the low hardness levels prevalent in Region 10, however, perhaps 7% of the samples might exceed the criterion. Either case, however, involves the worst case (and questionable) assumption that all cadmium was in the active form.

Based on single-species laboratory tests (used to derive the criterion), moderate violations of the criterion would affect several species of trout and salmon as well as some other fish and invertebrates. Without ancillary field data, the field applicability of laboratory tests may always be questioned, particularly with regard to the toxic form of the pollutant, physiological acclimation, genetic adaption, and community interactions (NRC 1981). While field validations of the cadmium criteria have not been done, the weight of evidence supporting these criteria seems persuasive, particularly in light of the number of species affected at levels near the criteria, the severity and speed of the observed effects, and the applicability of the criteria only to readily available forms of the metal. Thus, persistent violations of the criteria seem likely to produce noticable changes in many biological communities.

In an attempt to further identify the magnitude of the aquatic cadmium problem, the EPA fish kill files were searched. For the period 1970-1978 only one small fish kill was attributed to cadmium. Nevertheless, cadmium's contribution to some fish kills could be easily overlooked by field investigators due to its presence at low levels relative to other metals such as copper and zinc. It must also be noted, however, that in the year 1976 only 1.3% of all fish kills (and 0.1% of all killed fish) were attributed to metals released during industrial operations (EPA 1979c). While the lack of fish kills strongly suggests the lack of widespread cadmium problems, an argument could possibly be put forth that the effect of continuous discharges might be manifested in a reduction of species diversity or an impairment of ecological function rather than in discrete fish kill incidents.

4.2 Controlling Key Sources

Nationwide mapping of STORET data has not been found useful discerning associations between elevated cadmium levels and geographical areas having heavy concentrations of metal handling industries or high population densities (Delos 1981). Because STORET data are contributed by many different agencies, differences in analytical methods are believed to mask true geographical differences in cadmium levels. Likewise, while State agencies have identified river basins that they consider to have cadmium contamination problems (as shown in Table 11), they apply differing criteria for selecting cadmium as a pollutant worthy of mention. There is no certainty that cadmium is actually impairing the integrity of aquatic ecosystems in the waters identified in Table 11. Likewise, of course, it cannot be said that cadmium is not a problem in waters not identified in Table 11.

Nevertheless, when data from a single laboratory using GFAAS were examined, a relationship between cadmium levels and point source discharges could be discerned. A study of 60 sites in the Pacific Northwest indicated that stations downstream of point sources (of any type) had a geometric mean concentration two fold higher than stations unaffected by point sources (Delos 1981). It must be noted, however, that important industrial catagories are in the process of reducing their cadmium discharges. It might also be noted that some of the most severe metals pollution problems have been associated with mine drainage (Perwak et al. 1980).

Regulatory controls on cadmium and other metal releases to ambient waters have often been directed toward mitigating the effects of point sources on aquatic life. Such controls have been instituted on the basis of technology (for example, industry-wide Best Available Technology, BAT) or on the basis of water quality (site-specific waste load allocations). Table 12 summarizes the total discharge of cadmium estimated to be allowed under BAT for industrial catagories. The table also shows the estimated discharge allowed under PSES (Pretreatment Standards for Existing Sources) for industries connecting to the sewer systems of POTWs (Versar 1984). Based on these data, BAT/PSES controls could be expected to substantially reduce the total point source discharge of cadmium from levels thought to be discharged in the late 1970's. A dramatic example is the electroplating industry, where the estimated BAT/PSES discharge (Versar 1984) is more than an order of magnitude lower than the previous discharge (Alchowiak and Maestri 1980).

Although the overall importance of cadmium in impairing aquatic life is not known with any certainty, it is one of several metals for which aquatic life concerns have been high. In many situations, however, concerns about cadmium cannot be readily isolated from concerns about other metals. Wastewater discharges, as well as the affected water bodies, are often contaminated with numerous metals. Even after ecological impairment is demonstrated by field surveys of fish and macroinvertebrates, and after contamination is documented by chemical analysis of water and sediment, identifying which of the pollutants in a complex mixture are the culprits is a formidable problem. There are two general approaches most likely to find use: (a) pollutant-by-pollutant analyses and (b) whole effluent toxicity analyses.

Table 11: Summary of River Basins Having Cadmium Contamination Problems, as Identified in 1980 State 305(b) Reports.

Basin No.	Basin	Sources Implicated*
0109	MA-RI Coastal	VARIOUS
0202	U. Hudson	NAT, MUN, IND
020301	L. Hudson	VARIOUS
020302	Long Island	VARIOUS
020401	U. Delaware	MIN, MUN, IND
0207	Potomac	MUN, IND
030702	St. Mary's	IND
030801	St. Johns	UR, MUN, IND
030802	E. Fla. Coastal	UR, MUN
030901	Kissimmee	AG, UR
030902	S. Fla.	AG, UR
031001	Peace	IND, AG, UR
031002	Tampa Bay	AG, MIN, IND, UR
031101	Aucilla-Waccasassa	AG, SILV
031102	Suwannee	VARIOUS
031200	Ochlockonee	-
031300	Apalachicola	MUN, IND, AG, SILV
031401	Fla. Panhandle	VARIOUS
031402	Choctawatchee	MUN, AG, SILV
031403	Escambia	MUN, AG, SILV
0410	W.L. Erie	UR, MUN, IND
0411	S.L. Erie	VARIOUS
041202	L. Erie	VARIOUS
0502	Monongahela	UR, MUN, IND
0504	Muskingum	CON, IND, MUN
0505	Kanawha	MUN, AG, CON
0506	Scioto	IND, UR, MUN, CON
050702	Big Sandy	VARIOUS
0508	Great Miami	VARIOUS
051002	Kentucky	VARIOUS
0511	Green	MUN, UR, AG, IND
051302	L. Cumberland	MUN, UR
051401	L. Ohio-Salt	AG, MUN, IND
051402	L. Ohio	VARIOUS
070801	U. Miss-Skunk Wapsipinican	-
0710	Des Moines	NAT, UR, MUN
080202	St. Frances	MUN, AG
100902	Powder	MUN
1018	North Platte	IND, LS, UR, MUN
1019	South Platte	IND, LS, UR, MUN
102001	Mid. Platte	MUN, LS

Table 11 (Continued):

Basin No.	Basin	Sources Implicated
1025	Republican	AG, LS
1102	U. Arkansas	IND, AG, LS
1105	L. Cimarron	UR, AG, MUN
1106	Arkansas, Keyston	AG
110701	Verdigris	AG, MUN
111003	L.N. Canadian	AG, UR
111203	North Ford Red	AG
120301	U. Trinity	MUN, IND, UR
120302	L. Trinity	MUN, IND, AG
120401	San Jacinto	MUN, IND, AG, VES
120402	Galveston Bay-Sabine Lake	VARIOUS
1205	Brazos H.W.	-
120701	L. Brazos	MUN
121004	Central Texas Coastal	MUN, AG
121102	S.W. Texas Coastal	MUN
1301	Rio Grande H.W.	MUN, IND
130201	U. Rio Grande	MUN, AG, UR
130202	Rio Grande-Elephant Butte	MUN, UR
130301	Rio Grande-Caballo	MUN, UR
1401	Colorado H.W.	-
1402	Gunmison	IND
1403	U. Colorado-Delores	-
140401	U. Green	IND, MUN, LS
140801	U. San Juan	MUN, AG
1502	Little Colorado	-
150501	M. Gila	-
150502	San Pedro-Wilcox	-
150602	Verde	-
150701	Aqua Fria L. Gila	-
2101	Puerto Rico	CON, MUN, UR
2102	Virgin Island	-

*Sources are those named by the State for metals in general, not just cadmium. The order of appearance has no significance.

AG = Agriculture
CON = Construction
IND = Industrial
LS = Livestock
NAT = Natural
MIN = Mining
MUN = Municipal
SILV = Silviculture
SW = Solid waste
UR = Urban runoff
VARIOUS = Several categories
VES = Vessels

Table 12: Waterborne Discharges of Cadmium Allowable from Regulated Industrial Catagories under Proposed Best Available Technology (BAT) and Pre-treatment Standards for Existing Sources (PSES) (Versar 1984).

	Direct Dischargers		Indirect Dischargers	
Industrial Catagory	Number of Plants	Catagory Total lb/day (a)	Number of Plants	Catagory Total lb/day (a)
Aluminum Forming	42	0	64	0
Battery Manufacturing	15	0	134	0
Coal Mining	10375	91	0	0
Coil Coating (I)	29	0	39	0
Coil Coating (II)	3	0	80	0
Copper Forming	37	1	45	1
Electrical/Electronic Mfg	83	0	244	1
Foundries	287	0	327	0
Inorganic Chemicals (I)	114	40	21	0
Inorganic Chemicals (II)	35	0	18	0
Iron & Steel	738	15	160	6
Leather Tanning	17	0	141	0
Metal Finishing/Plating	2800	24	10200	53
Non Ferrous Metals	79	3	85	0
Non Ferrous Metals Forming	49	0	147	0
Ore Mining	515	40	0	0
Organic Chemicals	1082	NA (b)	535	NA (b)
Pesticides Mfg	42	0	39	0
Petroleum Refining	164	1	47	0
Pharmaceuticals	80	0	392	0
Plastics Molding/Forming	565	0	1006	0 (c)
Porcelain Enameling	28	1	50	2
Pulp & Paper	355	0	261	0
Textiles	229	5	1047	9

(a) Values are approximate.
(b) Not available.
(c) Excluded from PSES regulation due to minimal importance.

In the pollutant-by-pollutant approach each wastewater constituent is evaluated separately. The traditional method is to compare water column concentrations with effect levels determined by single-species laboratory tests of pure substances. Other methods, for example, utilizing sediment concentrations or utilizing concentration-effect correlations obtained from field data, could be developed in the future.

In the general toxicity approach, on the other hand, control alternatives may be tested at the bench, directly using toxicity tests as the measure of effectiveness. For some types of control alternatives the identity of the culprit pollutant(s) might remain unknown using this approach.

The pollutant-by-pollutant approach indicates the allowable concentrations of pollutants in the effluent. The general toxicity approach indicates an allowable dilution of the whole effluent. Neither approach, however, indicates the allowable frequency of exceeding the target level (criteria). Data indicating the likelihood of observing ecological changes at various exceedance frequencies are not generally available for any pollutant. In conventional practice, the allowable exceedance frequency is set arbitrarily, usually at a low frequency.

Reductions in wastewater concentrations of cadmium can often be readily accomplished by sedimentation with or without chemical addition, by biological treatment, and by some other methods (EPA 1980b). These same processes are used for conventional pollutants and other toxic metals. It would thus appear that reductions in cadmium discharges are being brought about intentionally by limitations on cadmium loads and coincidently by limitations on other pollutant loads.

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