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EDITORIAL

DELETERIOUS EFFECTS OF LOW LEVELS OF MISINFORMATION

The environment is finally one of the top global issues. There is virtually no political party that does not include environmental protection in its platform, and there are political parties solely or predominantly devoted to environmental protection. In certain countries political elections are won and lost on environmental issues and, due to their popularity, many politicians consider themselves to be environmentalists. It is not surprising that environmental issues are greatly mixed with political goals, and thus the interest of the environment is not always served by those who advocate environmental protection.

Inherent in proper societal decision making is the availability of reliable information. Environmental protection, like any other important topic, requires the availability of information based on science that has been subjected to scrutiny by those who are qualified to express their views. Those with political goals will inevitably try to use information that promotes their point of view and disregard that which enhances the opposing views. Given the nature of the political system in a pluralistic society, it is inevitable that the public is exposed to the misinformation. However, repeated and somewhat erroneous misinformation cannot be readily discarded by the public.

The example of the toxic effects of environmental pollutants may be used to illustrate the point. Exposure to large quantities of a chemical can lead to a quick death of the exposed individuals. This acute effect can be relatively easily traced to the causative agent. However, exposure to low levels of certain toxicants can lead to chronic effects which are often not easily traceable to the exposure. The environmental scientific community has recognized deleterious effects of exposure to low levels of chemicals, and risk assessment is extensively used to quantify these effects.

Deleterious effects of exposure to misinformation are analogous to adverse effects of chemicals. Gross misinformation may cause immediate harm. A great deal of suffering has been caused by rumors that proved to be incorrect. Large-scale panick shopping for food and for other consumer products has repeatedly caused shortages and the associated hardships. There is no dispute that these are harmful and governments spend their best efforts to combat these kinds of misinformation. In contrast to these, there appears little effort to combat low levels of misinformation. For example, the popular news media, both print and electronic, have repeated news items on cancer-causing agents and their effects. Accordingly, these indicate there is an epidemic of cancer caused by food additives, pesticides, and industrial effluents. Not surprisingly, a large segment of the public believes that, if these sources were eliminated, the number of cancer cases would be substantially reduced. In fact, their contribution to the cancer burden is small and probably negligible as compared to the effects of smoking and dietary intake of fat and other natural food constituents. Consequently, societal efforts and funds are concentrated on testing and controlling chemicals.

It is obvious that there are significant adverse effects caused by misinformation. A society that spends its resources on controlling the wrong sources of pollutants will have the same results as a physician who misdiagnoses a disease. The fate of the public will be comparable to the fate of the patient.

A. Alan Moghissi

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HUMAN PERFORMANCE DURING EXPERIMENTAL FORMALDEHYDE EXPOSURE

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Sixty-one subjects were exposed in a climate chamber for 5.5 hours to a controlled atmospheric environment. Formaldehyde vapours were added in concentrations of 0, 0.15, 0.40, or 1.20 mg/m³. The exposures were arranged in a 4 x 4, balanced latin square design, involving four days in each of four weeks. The subjects were all males. Of these, 32 had occupational exposure to formaldehyde in industrial productions for more than five years. Twenty-nine were randomly selected, matched controls from the normal population. The hypothesis tested was that significant, but different dose-response relations exist in a number of performance tests for these two groups of subjects. The results indicate such differences in reactions to tests of short term memory and ability to concentrate (digit span tests, digit symbol test, graphic continuous performance test) and an addition test. Whether these results indicate chronic or acute CNS effects or they are caused by distractive sensory irritation due to formaldehyde exposure is discussed.

INTRODUCTION

Sensory irritation due to formaldehyde is well-known in industry (Arbeta and Hälsa 1978; Gibson 1983; NIOSH 1977). Furthermore, within the last ten years formaldehyde has been recognized as an indoor air pollutant in nonindustrial environments. The main effects of formaldehyde are irritation of mucous membranes in eyes, nose, and throat (Andersen 1978; Andersen et al. 1979; Andersen and Mølhave 1983). Formaldehyde is a known allergen (Cronin 1980) and a potential carcinogen (NIOSH 1989; Olsen et al. 1984). The occupational threshold limit in Danish industries is 1.20 and 0.4 mg/m^3 in production lines established before and after 1982, respectively (Danish Labour Inspection 1986). For the indoor nonindustrial environment a limit of 0.15 mg/m^3 is recommended.

The aim of this experiment was to test whether humans react acutely to experimental exposure to these three concentrations of formaldehyde (0.15, 0.40, 1.20 mg/m^3), using clean air as the control condition. A second aim was to determine if chronic effects occur after several years of occupational exposure to the chemical. For the latter purpose, reactions of randomly selected subjects were compared with the reactions of subjects who for more than five years had been occupationally exposed to formaldehyde.

Among the most frequent complaints following formaldehyde exposure are headache, dizziness, and nausea (Arbeta and Hälsa 1978). These reactions indicate possible effects on the central nervous system (CNS). A number of performance tests, therefore, were included in the test battery of the experiment. This publication deals with these performance tests only. Other parts of the experiment have been published elsewhere (Bach et al. 1987).

MATERIALS AND METHODS

The exposure experiment was performed in the climate chamber at the Institute of Environmental and Occupational Medicine, University of Aarhus, Denmark (Bach et al. 1987). Four subjects were exposed simultaneously for 5.5 hours to one of four concentrations of formaldehyde (0.0, 0.15, 0.40, or 1.20 mg/m^3). These concentrations represented the average background concentration in the chamber and the three existing threshold limit values for non-industrial environments, new, and old production lines, respectively (Table 1).

Table 1. Controlled indoor climate parameters and exposure variables.

Variable:	Unit	intended	obtained ± SD	
Formaldehyde concentration	mg/m³	0.0	0.04	0.03
		0.15	0.21	0.12
		0.40	0.48	0.11
		1.20	1.10	0.16
Parameters:				
Air temperature	°C	23.0	22.8	0.2
Globe temperature	°C	23.0	22.8	0.2
Air humidity (relative)	%	45	45.5	0.8
Ventilation	ACH	5.0	5.4	0.4
Air movements	cm/s.	2.5	2.5	2.0
Light level	Lux	>400	419	13
Sound level	dB(A)	<65	62	1
Activity	Watt	0.75	-	-
CO_2	ml/l	<0.04	-	-
Dress	CLO	0.75	-	-

Note: - = not measured.

ACH = Air changes per hour.

Measurements during the experiment showed that the actual mean concentrations (mean ± SD) during the exposure periods were: 0.04; ± 0.03; 0.21 ± 0.12; 0.48 ± 0.11; and 1.10 ± 0.16 mg/m^3. The exposure periods of 5.5 hours were preceded by a 30 minute preexposure period in the chamber to establish baselines for the subjects. The exposure concentration in the chamber was established gradually within the first half hour of the exposure period and with the subjects in the chamber. The exposure was constant during the last five hours.

The experiment was designed to evaluate only the subacute effects due to 5.5 hours of exposure. The experimental situation simulated that of an occupant adapted for 30 minutes to formaldehyde, and not that of a visitor entering a room with an already established concentration. Therefore no effect occurring at a time scale less than 30 minutes would be registered in this experiment.

Indoor climate factors other than the formaldehyde concentration were set to preselected values as shown in Table 1. These factors represent what is generally considered to be a comfortable, healthy indoor climate.

During the experimental session, two workers, two control subjects, and two staff members were in the chamber at the same time. Each subject was exposed only for one day, and the exposure was arranged in a balanced 4 × 4, latin square design including four concentrations for four week days in each of four weeks. A total of 16 exposure days was used. The design is shown in Table 2. The exposure was arranged in a double blind way, and a strong odorant (Furfurylmercaptan, a coffee aroma constituent) was used to mask the formaldehyde odor in the chamber during exposure.

The subjects were all males aged 18 to 64 years. Thirty-two subjects were selected among 108 workers with more than five years of occupational exposure to formaldehyde. They were selected from local factories, where formaldehyde was regularly used.

From a group of 546 randomly selected males found via the population registry, 29 male subjects were matched to the workers to give the same average age, education, and smoking habits as for the exposure groups. Control subjects who had previously been occupationally exposed to formaldehyde were excluded, as well as subjects with illnesses that might disturb the experiment. The matching of the workers and controls was not completely successful; some subjects failed to appear on the assigned day and had to be replaced with short notice. Generally, the group of workers, especially the group exposed to 1.20 mg/m^3, contained more smokers and had a lower average school education. The group exposed to 1.20 mg/m^3 had an average age 10 years below that of the three other groups and contained fewer smokers. No significant differences were found in relation to medication, alcohol consumption, or occupational education between the two exposure groups.

During the experiment, general comfort was measured with a standardized questionnaire. Only questions which may be related to neurotoxic effects will be mentioned here. These concerned headache, physical, and mental tiredness ("heavy head"). Questions with no relation to the CNS have been described elsewhere (Bach et al. 1987). The questions were answered both on a linear analog scale between no effect and extreme effect and with "Yes " and "No" for acceptable conditions.

Four performance tests with different scores were administered in two runs during each exposure period.

The digit span test is a subtest from the WAIS-test battery (Wechsler's adult intelligence scale)(Wechsler 1955). It was used both as a measure of ability to concentrate (a measure of distraction stability) and as a test of short term memory. The test is known to be sensitive to memory impairments in persons exposed to neurotoxins (Motarazzo 1972).

Both "Digit forwards", "Digit backwards", and the sum of these scores ("Digit sum"), were used as scores of the test. The subjects were tested twice during the exposure period, each test supplying all three measures. Thus six measures were available from each subject.

The digit symbol test is another subtest from the WAIS-test battery (Wechsler 1955). The test indicates the subjects' distraction stability and capability to understand and perform a task. It also indicates change in psychomotor functions. The test is based on a printed test formula with symbols for digits one to nine. These symbols are used as substitutes for numbers in the next line on the formula and are written into empty boxes. The number of correct substitutions within 90 seconds gives a score number (max. 90). The test was used twice during the exposure.

The graphic continuous performance test is a pencil and paper test (Andersen 1978). It measures the subjects' capability to concentrate. It may be used as a psychomotor test as well. During the test, three different graphic patterns of lines were shown one by one to the subjects. After learning the patterns, the subjects were asked to draw five continuous lines of each pattern with a pencil without lead on a paper with 0.5 × 0.5 cm squares. The drawings were recorded by use of carbon black beneath the paper. Thus the subjects were unable to check the results. Both the total number of errors and the total time used to cover five lines with a 30 cm pattern each were recorded. The test was administered twice during the exposure period.

The computerized addition test focused on distraction stability and on short term memory (Bach et al. 1987). The test is run on a one digit display, showing digits between 0 and 9, one by one, and in a random sequence. Two push buttons are used to indicate whether the sum of the present and the former number on the display is less than ten or more. The display changes after each answer, and the total test time is ten minutes. The test was used once during the preexposure period and twice during the exposure period. The test scores were the total number of additions in ten minutes test time, the total number of errors, and the average reaction time.

The statistical analyses differ among the tests according to the approximation adapted for the distribution and the homogeneity of the variance. The analyses will be specified in relation to the results of each test. Generally continuous variables like linear analog responses in the questionnaire, the graphic performance test, digit symbol test, and the addition test were analysed in an analysis of variance if homogeneity of variance could be demonstrated. F-tests were applied. Non-parametric tests were used

Table 2. The experimental design.

Week

Day	W1	W2	W3	W4
D1	A	B	C	D
D2	B	A	D	C
D3	C	D	A	B
D4	D	C	B	A

A = 0.15 mg/m³
B = 1.20 -
C = 0.40 -
D = 0.0 - (background)

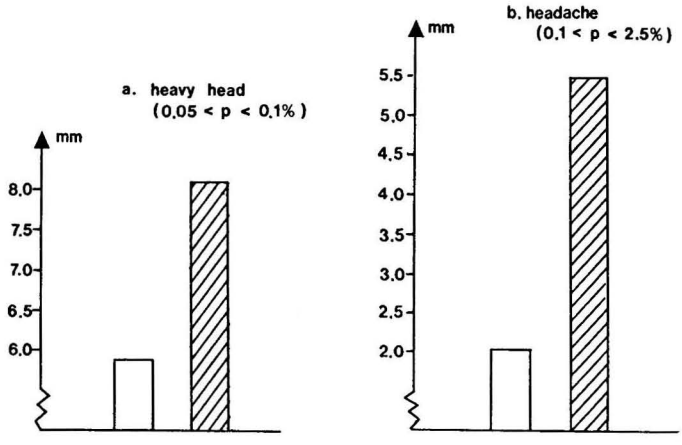

Fig. 1. Answers from the questionnaire about "heavy head" (a) and headache (b) for workers (white) and controls (grey). The ordinate shows differences in mm on a 60 mm linear rating scale between exposure and pre-exposure. The higher the score, the worse the condition.

when homogeneity was missing. The analyses were done using SPSS (1975).

Discrete variables like yes/no answers in the questionnaire or the digit span test were analysed by a χ-square test or Fishers exact test, taking into account covariables like age, education, and smoking habits.

Differences were considered significant for $p < 0.05$. Levels of $0.05 < p < 0.10$ were considered indicative of tendencies. Some results were considered "interactions", e.g., when the workers were found to react differently from their controls. The interaction term is also used for significant but nonlinear dose response reactions, e.g., when the maximum reactions were found at the exposure level 0.4 mg/m^3, and not for 1.2 mg/m^3.

RESULTS

The questionnaire

Significant effects of exposure were found for questions indicating neurotoxic effects, e.g., headache and tiredness. Headache was indicated late in the exposure period by the control subjects, but not by the formaldehyde workers (Fig. 1). Such differences in reactions between the workers and their controls were also seen in questions related to heavy head and physical tiredness.

Performance tests

The correlation between the different performance tests and the reproducibility of the test results was examined in order to demonstrate to what extent they measure independent effects. Some of the correlations are shown in Table 3. It appeared that the correlation among the performance tests was acceptable, both with regard to the in-between runs correlation of the same test and the correlations between different tests in the same run.

Only one of six measures in the digit span test was significantly related to the experimental exposure, as shown in Table 4. The statistical analysis compared the mean scores for each exposure group with mean scores for the workers and the control subjects. The possible effect of variables like age, educations, intake of alcohol, and medication was examined in χ-square tests of contingency tables. At the higher formaldehyde concentrations the total digit sum score decreased in the last of the two runs during the exposure. In digit forward a significant difference between the previously unexposed control persons and the previously occupationally exposed subjects was found.

The results of the digit symbol test were analysed, using both one-sided and two-sided multivariate analyses. The difference between the exposure groups was compared for the formaldehyde workers and the control groups. A significant effect was found when the results from exposure to formaldehyde level 0, 0.15, and 0.40 mg/m^3 were pooled and compared with results from exposure to the level 1.20 mg/m^3 (Fig. 2). The scores of the test decreased for formaldehyde workers, but increased for control persons as the exposure level increased. This indicates that formaldehyde ex-

Table 3. Coefficients of correlations (R) between scores in different performance tests. Only correlations significantly different from zero are shown.

	Run	Digit symbol run 1	Digit symbol run 2	Continuous line:time run 1	Continous line:time run 2
Digit symbol:	2	++	0	0	0
Continuous line:					
time	1	++	++	0	0
time	2	++	++	++	0
errors	1	++	++	++	++
errors	2	++	++	++	++
Addition test:					
Distraction stability	1	-	-	-	-
Distraction stability	2	-	-	-	-
Number of errors	1	++	++	(+)	++
Number of errors	2	0	0	0	0
number	1	++	++	++	++
number	2	++	++	++	++
Digit span test:					
Digit forward	1-2	++	++	++	++
Digit backward	1-2	++	++	++	++
Digit sum	1-2	++	++	++	++

++	:	0.1% < p < 2.5%
+	:	2.5% < p < 5%
(+)	:	5% < p < 10%
-	:	p > 10%
0	:	not analysed

posure increases performance in controls, but decreases it for the occupationally exposed subjects (interaction). The significance pattern of the digit symbol test is found in Table 4.

Data from the graphic continuous performance test were also analysed, using multivariate analysis. It showed a significant difference between groups exposed to different concentrations (exposure groups), as seen in Table 4. The total time used for the test was longer for the exposure level 0.40 mg/m^3. A one-sided analysis of variance showed that formaldehyde workers, but not control persons, reacted to the exposure by using a significantly longer time for the test at 0.40 mg/m^3.

No other differences, and especially no difference in the number of errors at the four exposure levels, were found.

The computerized addition test was analysed by comparison of the baseline value found before the exposure, both to individual scores of the four exposure groups and to the scores from each of the two runs during the exposure period. The baseline values did not differ significantly among the groups.

Age and education were used as explaining variables in the statistical analysis. Both the total number of errors and a total number of additions during the ten minute period showed significant exposure effect (Table 4). Increasing concentration of formaldehyde is related to decreasing number of additions and increasing reaction time. A dose-response relationship was found in both runs, both for the number of additions and the reaction time (Figs. 3 and 4, respectively).

Table 4. Significant responses from four performance tests.

	Run	Exposure effect	Interaction	Occupational effect	Explaining variables
	a)	b)	c)	d)	
Digit span test					
Digit forward	1	-	0	(+)	(+) education
- -	2	-	0	+	-
Digit backward	1	-	0	-	++ education
- -	2	-	0	-	-
Digit sum	1	-	0	-	(+) education
- -	2	++	0	-	+ education
Graphic continuous line test					
Total time	1	+ ***)	++	-	-
- -	2	+ ***)	++	-	-
Number of errors	1	-	-	-	-
- - -	2	-	-	-	-
Addition test					
Number of additions	1	++	0	-	+ age
- -	2	++	0	-	++ age
Number of errors	1	+ ***)	0	-	-
- -	2	-	0	-	-
Distraction	1	-	0	-	-
- -	2	(+)	0	-	-
Digit symbol test					
Score	1	++ **)	(+)	-	++ education
-	2	++ **) ***)	++	-	++ education

++ : 0.1% < p < 2.5%
+ : 2.5% < p < 5%
(+) : 5% < p < 10%
- : p > 10%
0 : no analysis needed. No interaction.
*) Exposure groups 0.0, 0.15, and 0.40 pooled against exposure group 1.2 mg/m^3.
**) One-sided analysis only for occupational formaldehyde exposed.
***) One-sided analysis only for controls.
a) Each test was run two times.
b) Exposure effect means effect of the present experimental exposure.
c) Interaction refers to different reactions among the eight subject groups (occupational — non-occupational exposure subjects) x (four experimental exposure levels).
d) Occupational effect means effect of previous occupational exposure.

DISCUSSION

Effects of acute exposures

The two major aims for this study were to test if exposure to low levels of formaldehyde can provoke acute human reactions and to examine the effect of chronic occupational exposures on such reactions.

Under the present experimental conditions, subjective ratings (Bach et al. 1987) generally did not confirm the significant dose-response relationships typical of irritation caused by exposure to formaldehyde (Andersen 1978; Andersen et al. 1979; Andersen and Mølhave 1983). It is especially surprising that irritation of the mucous membranes in eyes,

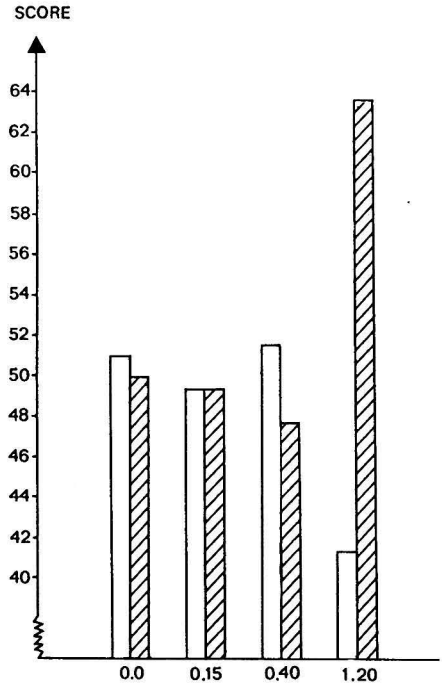

Fig. 2. The digit symbol test. Scores in run 2 for workers (white) and controls (grey). The abscissa refers to exposure concentrations in mg/m³.

nose, and throat did not correlate significantly to the exposure. This may be due to the fact that the subjects were adapted to the experimental formaldehyde exposure for 30 minutes before any tests were performed.

Several other factors may explain the low levels of acute irritation observed. Among these are that comparison of the results obtained in this experiment with previously published epidemiological studies must take into account that this experiment deals with formaldehyde as the only variable factor in the experiment. Under field conditions, the exposure inevitably consists of formaldehyde plus a number of other physical and chemical factors with which it may interact.

To the extent that such interactions occur, a number of extra effects may be expected under field conditions. The effects seen in this experiment, therefore, should be considered more specific for formaldehyde than effects found in field investigations.

Asthmatic or allergic persons were excluded from this study. A general population, which includes asthmatic and allergic persons, may be more sensitive than our present subjects. In field studies of occupationally exposed workers, the "healthy workers effect" may cause underestimation of measured effects because subjects who feel especially affected by formaldehyde or co-existing exposures would leave the work environment.

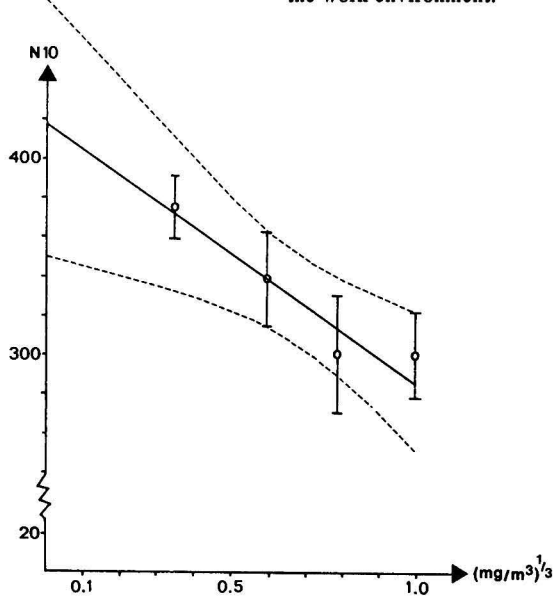

Fig. 3. Addition test (run 2): Number of additions made in 10 minutes. The abscissa is the concentration of formaldehyde in $(mg/m^3)^{1/3}$. Confidence interval (95%) of a linear regression analysis and ± one standard error of the averages are indicated.

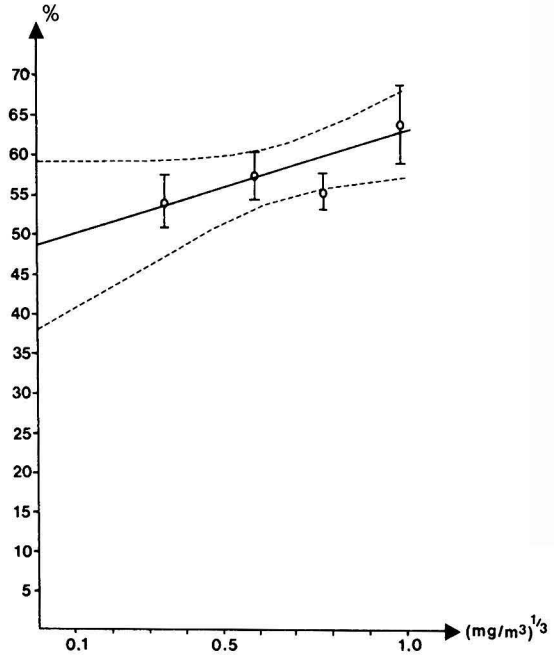

Fig. 4. Addition test (run 2): Standard deveation of the average response time used as a measure of level of distraction. Confidencre interval (95%) of a linear regression analysis and ± one standard error of the averages are indicated.

Exposure to formaldehyde has not yet been related in the literature to chronic or acute CNS effects. One reason for this may be lack of experimental evidence and the belief that no such effects should be expected because of the high chemical and biological reactivity of formaldehyde. This would cause formaldehyde in the air to react at the body surface and mucous membranes before reaching vital organs. The compound therefore is expected to be absorbed to these surfaces. Only small quantities are expected to be distributed into the body, and they would be bound to other compounds or chemically changed, e.g., by metabolism. Consideration of the effects of such mechanisms is, however, beyond the scope of this paper.

One of the conclusions of this experiment is, as shown in Table 4, that CNS effects may be possible even at low and short-term experimental exposure levels. The effects may be attributed to either a direct CNS effect or to distraction due to irritation caused by the exposure. Some of the results of subjective ratings may be related to CNS function. However, although none of these show any effect of the experimental exposure, it is possible that subjective effects indicative of neurotoxic effects are too weak to be registered subjectively after only 5.5 hours of exposure.

The single factor most affecting the conclusiveness of the experiment is the inhomogeneity of the dose-effects. The graphic continuous performance test and the digit span test showed the poorest performance at a formaldehyde exposure level of 0.4 mg/m^3. This exposure level was found to cause the strongest reactions in many of the tests. This highest sensitivity at 0.4 mg/m^3 may be a true effect only in case of nonlinearity of the dose-response relations, e.g., caused by different thresholds for initiation of biological protective mechanisms. Alternative explanations such as non-comparable exposure groups, especially for the exposure group at 1.2 mg/m^3, are more likely in this experiment.

The 1.2 mg/m^3 exposure group turned out to have lower average age and fewer smokers than the other three groups. This imperfect matching of controls to workers, which is due to last minute substitution of subjects who failed to appear, reduces the power of

the experiment somewhat, as each cell in the experimental design contains only eight persons.

Effects of previous chronic exposure

One of the conclusions of Table 4 is that the workers may react differently from the controls to the exposure, as differences between exposure and worker effects are indicated in the results. Formaldehyde workers had the lowest score and the highest count of errors in the tests, e.g., see the results of the digit span test when compared to the control persons. This may indicate possible CNS effects of chronic, prolonged exposure to formaldehyde.

The number of performance tests (4) and the number of different scores (18, see Table 4) used in this experiment, and the consistency of the reactions found are limited. In the digit span test, e.g., only one out of six measures showed significant effect of the exposure. One positive and five inconclusive results do not allow any final positive conclusion regarding effects being due to CNS changes or even being secondary effects caused by distraction from the exposure. However, the workers who were most familiar with formaldehyde and, therefore, supposedly less distracted decreased their performance in the digit symbol test. The controls did not, and all dose-effects showed a decreasing performance with increasing exposure.

The experimental exposure chamber is a very unfamiliar surrounding for the subjects. The industrial workers may, however, be more familiar with such a technical environment than the controls. The two groups may react differently in this environment. This may cause an uncontrolled experimental difference between the two groups.

Subjects suffering from CNS diseases or psychiatric diseases were excluded from our investigation, but possible previous exposure to organic solvents could not be taken into account. Such exposure might lead to the same type of differences in the performance test as seen here. A long-term effect of solvents, therefore, cannot be excluded as the reason for any poorer performance observed among the occupationally exposed subjects. Any conclusion, however, cannot be definitively made as this experimental design did not include objective measures of the previous occupational exposures and because performance is dependent on education; the educational level was lower among the occupationally exposed subjects.

CONCLUSIONS

Our investigations indicate that exposure to formaldehyde at levels of 0.40 and 1.2 mg/m^3 under controlled conditions may cause acute CNS effects. Furthermore, it cannot be excluded that effects of a long-term occupational formaldehyde exposure may result in poorer mental performance. Further investigation, however, will be needed to verify our hypothesis of possible acute or chronic effects on the CNS due to formaldehyde exposure.

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DESIGN AND EVALUATION OF A HIGH-FLOW RATE DICHOTOMOUS AEROSOL SAMPLER

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Two prototype high-flow dichotomous samplers, each simultaneously collecting fine (0.0-2.5 μm), coarse (2.5-10.0 μm), and total (0.0-10.0 μm) particle samples from a single air stream, have been designed and evaluated. A series of tests were conducted to estimate the prototype sampler collection efficiencies over a range of particle concentrations, to compare prototype sampler collection efficiencies with standard particle monitors, and to evaluate relationships between particle size and sample chemistry. No statistical differences in particle collection efficiencies were observed between the two prototype samplers or between the prototype samplers and standard PM_{10} and $PM_{2.5}$ high volume samplers. The study's analytical results indicate that the particle-bound organic compounds are primarily associated with the fine-particle sample and that the organics associated with the coarse-particle fraction did not significantly contribute to the total sample organic composition. The prototype sampler can be constructed locally from routine sampler and machine shop components. The design minimizes the extensive resource requirements associated with low-flow dichotomous sampling and is an excellent alternative for organizations not having access to commercial low flow dichotomous instrumentation.

INTRODUCTION

The results of past environmental investigations have documented increases in fine-particle (<2.5 μm, aerodynamic diameter) concentrations that are strongly correlated with the increased use of fossil fuels. Detailed chemical analyses of the ambient air, indoor air, and combustion source samples collected in airsheds significantly influenced by combustion emissions reveal that the associated particle and organic pollutants are rich in products of incomplete combustion and polycyclic organic matter. Both compound classes have been associated with increased risks for acute and chronic human health effects. The mechanisms for fine-particle deposition through impaction, encapsulation, or interception in the human lung have been well characterized. Fine particles, because of their physical and aerodynamic characteristics, are more likely to reach and remain in the lower levels of the human lung, a region where highly contaminated particles are provided the opportunity to directly interact with the sensitive human tissue. The documented relationship between combustion source emission exposures and human health mandates the development of improved scientific methods for the collection and characterization of fine particles.

The standard method for fine-particle measurement within the U.S. Environmental Protection Agency (EPA) utilizes a low-flow PM_{10} dichotomous sampler to collect particles less than 10 μm, aerodynamic diameter. This low flow rate sampler (0.0167 m^3/min total flow) employs a virtual impactor to separate and simultaneously collect fine particles (< 2.5 μm) and coarse particles (2.5-10.0 μm). PM_{10} particle concentrations reported across the United States typically range between 10.0 and 150.0 μg/m^3 depending on particle size fraction and sampling location. Although conventional dichotomous sampler technology

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is well designed and characterized, several factors influence its international use. First, commercially available dichotomous samplers are not readily available in many developing countries. Second, required day-to-day operational and support resources (filter media, maintenance and operation costs, and laboratory costs) are approximately five times the costs associated with more conventional high volume sampling. Equally important, economical as well as political considerations may prevent the use of commercially available dichotomous instrumentation to support environmental monitoring programs. These limitations on the collection and characterization of size-fractionated particle data minimize and in some cases prevent direct comparisons between internationally collected data sets.

The Integrated Air Cancer Project (IACP) is a long-term EPA research project designed to identify principal airborne carcinogens, determine the major emission sources for these carcinogens, and improve the estimate of comparative human cancer risks from specific air pollution emission sources (Lewtas and Cupitt 1987). Preliminary IACP studies (Highsmith et al. 1987) focused primarily on fine-particle sample collection in airsheds impacted by residential wood combustion and automotive emissions. These sources were selected for the early IACP studies on the basis of abundance of particles and organic pollutant emissions. Extensive, detailed chemical analyses, including standard Ames bioassay (Ames et al. 1975), were subsequently conducted on these fine-particle samples to characterize the aerosol, estimate mutagenicity, and fully evaluate the impact of these combustion sources on the local airsheds. Although extensive research has been conducted on fine-particle samples, the relationship between particle sizes and sample chemistry as a function of emission source has been of special interest, but remains poorly defined.

Even in airsheds significantly influenced by local and regional sources, such as residential woodsmoke and automotive emissions, the quantity of particle mass collected with standard dichotomous samplers (0.0167 m^3/min) is insufficient to support both detailed chemical and mutagenic analyses. Standard Ames bioassay determination for sample mutagenicity (Ames et al. 1975) requires near-milligram quantities of sample mass to support the analytical scheme. This shortfall in sample collected versus sample required to support the design analytical scheme becomes dramatically amplified when attempting to characterize airsheds influenced by more efficient sources.

Earlier IACP studies offset this large sample requirement by employing multiple high volume samplers (1.13 m^3/min) equipped with $PM_{2.5}$ impactors to collect fine-particle samples for bioassay. Several inherent problems associated with impactor plate samplers make these monitoring methods impractical for routine, large-scale environmental programs. Although many impactor systems collect large samples of particle mass, they are highly resource-dependent (space, electrical, manpower, and material). Secondly, the existing technology does not allow the investigator to easily estimate the coarse-particle contribution to the total PM_{10} sample mass or the influence of the coarse-particle mutagenicity on the total PM_{10} sample mutagenicity. The intermediate collection stages are either contaminated by the oil or grease used to spray the foil or biased by particle bounce and re-entrainment. The relationships among fine, coarse, and total PM_{10} sample particle fractions must be accurately evaluated to fully characterize and interpret the relationship between particle size and sample chemistry. Once defined, the relationships can be used to extrapolate the extensive IACP study findings with existing particle sampler data bases as well as to future data collected in accordance with the new federal PM_{10} standard (U. S. Environmental Protection Agency 1987).

A high flow rate dichotomous (HFD) sampling system has been designed and evaluated. The prototype sampler, based on conventional dichotomous sampler technology and a novel high flow rate virtual impactor (Solomon et al. 1983), simultaneously collects fine, coarse, and total particle size fraction data compatible with the new federal PM_{10} standard. The system can be readily fabricated in a local machine shop using standard high-volume and medium-volume sampler components. The design does not require the extensive resources associated with standard dichotomous sampler operation. A series of tests was conducted with two prototype HFD units to evaluate sampler precision and accuracy and to directly compare the prototype sampling systems against more standard EPA sampling systems.

DESIGN AND FABRICATION

The high-flow virtual impactor (Solomon et al. 1983) incorporated in the prototype sampler design has been reported to efficiently partition and simultaneously collect fine and coarse particle samples at a total flow rate of 0.565 m^3/min (20 ft^3/min). Their evaluation was conducted with a single high-volume motor and a series of connections and critical orifices. Similar to that of existing dichotomous sam-

pler designs, the coarse channel flow rate represented 10% of the sample flow with the remaining flow directed to the fine-particle collection media. Sampler design and construction considerations included (1) simultaneous collection of three independent size fractions (total, coarse, and fine) using a single PM_{10} size selective inlet, (2) nominal flow rate of 0.565 m^3/min for the fine and total channels to collect sufficient mass for detailed analysis, (3) minimal particle loss or re-entrainment from internal surfaces, (4) field reliability, (5) operating procedures similar to existing samplers, (6) filter face velocity (ca. 40 cm/s), equal to or less than standard particulate samplers, (7) use of standard high volume (200 mm × 250 mm) or medium volume (102 mm diameter) filters weighable on four-place analytical balances, (8) use of readily available system components, and (9) simple flow monitoring and auditing procedures.

The initial HFD sampler design (Fig. 1) focused on those dimensions directly affecting particle transport or loss, such as flow separation angles, joints and construction materials. Ambient air is sampled at 1.13 m^3/min (40 ft^3/min) through a standard PM_{10} size-selective inlet (Andersen 321A, Andersen Samplers, Atlanta, GA). A sharp edge located directly below the inlet divides the airstream into two equal components and directs the sample stream through a "Y" transition. The small separation angle (20°, < 10° from the perpendicular on each side) and the minimized inlet extension length (< 55 cm) were empirically determined to minimize particle losses prior to sample deposition on the filter media. Fabricated component transitions and bends were smoothed and gradual, minimizing internal losses. An adjustable, transformer-controlled, standard high-volume motor pulls one-half (0.565 m^3/min) of the PM_{10} inlet stream directly through a 200 mm × 250 mm high-volume filter for a total sample collection. Standard PM_{10} high-volume sampler mass flow controllers were initially used but proved unstable at this low flow rate. The balance of the inlet stream is pulled into the dichotomous separator which further divides the sample stream into the two distinct (fine and coarse) components. Two independent pump systems control the separator flow rates. A high volume motor operating at maximum voltage draws the fine particles initially 90° through a horizontal tube and then down

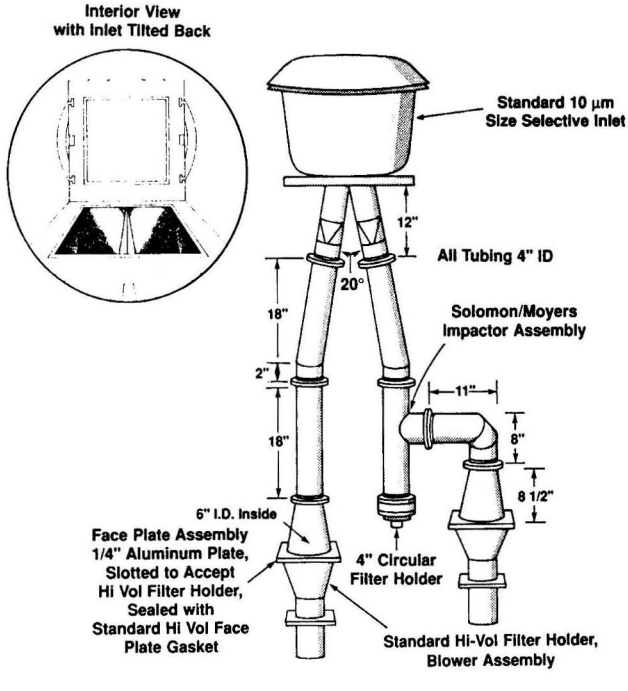

Figure 1. Schematic of high-flow rate dichotomous sampler.

onto a 200 mm × 250 mm filter. The separator critical orifice maintained constant fine-particle flow, eliminating the need for an additional voltage transformer to adjust the fine flow. A rotary vane pump (0.057 m^3/min) pulls the coarse particles directly down through the separator onto a 102 mm diameter filter. Filter cassettes are used both during sampling and in sample transport to minimize handling losses.

Modifications to the motor housing orifices to further restrict the exit flow were made to increase the readings of the standard Dickson (Dickson Company, Addison, Illinois) pressure recorders (manufacturer set for 1.13 m^3/min) which were originally too low for accurate interpretation. A pneumatic flow controller/needle valve assembly controls the coarse channel flow rate. The coarse flow was monitored by an in-line flow orifice connected to a Dwyer (Dwyer Instruments, Michigan City, Indiana) Magnehelic gauge.

EXPERIMENTAL

The sampling site was located in a Research Triangle Park, NC, residential neighborhood impacted by nighttime residential wood combustion emissions. Samples were collected only during nighttime hours, when meteorological conditions were most stable and maximum particle concentrations were expected. Two IACP $PM_{2.5}$ high volume samplers, one Andersen 321A PM_{10} high-volume sampler, one Andersen PM_{10} dichotomous sampler and two prototype HFD samplers were installed on a one-story building roof in accordance with standard Inhalable Particulate Network siting criteria (Environmental Monitoring Systems Laboratory, 1983). The $PM_{2.5}$ samplers used three-stage Andersen cascade impactors with a calculated final stage D_{50} of 2.5 μm. The sample stream passed through the impactors where particles larger than $PM_{2.5}$ were impacted on silicon oil-coated foils. Particles less than 2.5 μm were then collected on backup 200 mm × 250 mm filter media.

Reference high volume and dichotomous samplers were calibrated to operate at the manufacture-recommended flow rates (1.13 m^3/min and 0.0167 m^3/min, respectively). The two prototype HFD samplers were calibrated for 1.13 m^3/min total PM_{10} inlet flow, with each side of the transition (total and virtual impactor side) calibrated for 0.565 m^3/min. The virtual impactor coarse channel was initially calibrated for 10% of the total impactor flow, 0.057 m^3/min. However, the experimental flow rate through the virtual impactor equaled only about 0.460 m^3/min resulting from the increased pressure drop across the virtual impactor orifice. The coarse flow was therefore corrected to represent 10% of the actual total flow from the experimental dichotomous separator.

Calibration curves and interpolated tables were generated in accordance with routine Inhalable Particulate Network procedures. Both fine and total flow systems were independently calibrated with a standard high-volume calibration orifice. The calibration orifice inlet size was reduced in a fashion similar to that used to modify the blower housings. A mass flow meter was used to calibrate the coarse channel flow rate. The systems were frequently flow- and leak-checked at four locations: (1) directly below the PM_{10} inlet, (2) at the total pump, (3) at the inlet to the separator, and (4) at the coarse channel pump.

A series of 10 sample sets were collected to compare the samplers for size fraction collection efficiencies. Quartz 200 mm × 250 mm, quartz 102 mm and Teflon 37 mm filters were conditioned in a climate-controlled chamber (40% relative humidity and 20°C) for 24 h prior to tare weighing. Tared filters were loaded in cassettes, hand-carried to the sampling site, and loaded into the appropriate sampler. The samplers were simultaneously turned on and operated overnight for 12-16 h. The prototype HFD sampler seals and flows were checked 30 min into each sample run to ensure proper operation. At the completion of each sampling session, all samplers were simultaneously turned off, and the samples were immediately removed and returned for 24 h conditioning before final weighings.

A second series of sampling sessions was conducted to compare and evaluate the chemical composition of the sampler size collections. PM_{10} high-volume samples were collected on nontared quartz 200 mm × 250 mm filter media. Nontared Pallflex (Pallflex Products Corporation, Putnam, CT) Teflon impregnated glass fiber (TIGF) 200 × 250 mm and 102 mm filter media were employed for all other high volume and prototype HFD sample collections. PM_{10} dichotomous sample size fractions were collected on tared 37 mm filters identical to the earlier study filters. Sampling followed the procedures described above with one exception. Immediately following sample collection, the nontared filters were stored on dry ice (-80°C) pending analysis. The dichotomous filters were conditioned for 24 h, gravimetrically weighed, and then analyzed for metals by x-ray fluorescence. A portion of the PM_{10} quartz filters was extracted and analyzed for volatile and elemental carbon concentrations. For each sampling period, composites of each high volume and prototype HFD size fraction were prepared. Each compos-

ite and the remaining PM_{10} quartz filter were independently extracted with methylene chloride. Extractable organics were determined from an aliquot of the extract according to standard EPA procedures (Lentzen,1978). The remaining extracts were stored at -80°C for later analysis.

RESULTS AND DISCUSSION

Sampler particle collection data are summarized by size fraction in Table 1. Fine-particle concentrations for the initial 10 sampling periods ranged from 15 to 60 $\mu g/m^3$ with a coarse/fine ratio of 0.34. These values compare well with the particle size distributions reported previously for the background airshed of the Raleigh, North Carolina, region which includes Research Triangle Park (Highsmith et al. 1987). Correlation coefficients (r) calculated from regressing paired prototype HFD size fractions, as well as correlations between averaged prototype HFD size fractions and corresponding dichotomous, $PM_{2.5}$, and PM_{10} sampler size fractions, indicated excellent agreement (r=0.94) for each comparison (Table 2). Differences in the number of paired observations resulted from either torn filter media, operator-voided samples, or a non-operational dichotomous sampler during one sampling period. A paired T-test was used to test for significant (5% level) differences in sampler means. Linear regression analyses provided estimates of fixed (intercept) and proportional (slope) differences or relative bias between any two samplers. Tests for the ideal linear relationship (slope = 1, intercept = 0) were also conducted at the 5% significance level. Test statistics for the prototype HFD fine- and coarse-particle collections indicate that the two virtual impactor systems are indistinguishable. A marginally significant difference (mean difference = 3.1 $\mu g/m^3$) was observed between the two prototype HFD total particle collections (see Table 1) and is most probably associated with experimental error. Simultaneous testing of the prototype HFD total regression slope and intercept also resulted in rejection of the test hypothesis at the 0.022 significance level. This bias could not be independently attributed to either the regression slope or intercept. A physical examination of the two prototype HFD systems revealed that the two units employed manufacturer-reported equivalent, but slightly different model PM_{10} size selective inlets. Extensive research subsequently conducted (Rodes et al. 1985; Burton and Hayward 1988) has characterized and quantified differences in particle collection efficiencies between commercially available high-volume PM_{10} inlets. The statistically marginal differences noted above for the prototype HFD total collections may be an artifact of the two different model inlets employed in this evaluation.

Averaged values were determined for each of the three prototype HFD size fractions for testing against the other EPA samplers. Comparisons between averaged prototype HFD and dichotomous fine-particle and total-particle collection efficiencies yielded marginally significant differences between sampler means (4.4 $\mu g/m^3$ and 4.3 $\mu g/m^3$, respectively). These comparisons are significantly impacted by one highly questionable sampling period when the two prototype HFD collection efficiencies were highly reproducible (fine concentrations = 19.0, 19.7 $\mu g/m^3$; total concentrations = 38.2, 38.1 $\mu g/m^3$), but represented only 50% and 80%, respectively, of the fine-and total-particle fractions collected by the collocated

Table 1. Sampler particle concentrations ($\mu g/m^3$).

	Fine		Coarse		Total	
	$\bar{x}$	σ	$\bar{x}$	σ	$\bar{x}$	σ
Prototype [a]HFD_1	30.8	16.3	10.8	3.9	43.1	17.0
Prototype HFD_2	31.5	17.6	10.5	4.3	46.2	17.8
PM_{10} Dichotomous	33.4	14.4	10.8	5.8	44.3	18.8
$PM_{2.5}$ High Volume	31.2	16.8	-	-	-	-
PM_{10} High Volume	-	-	-	-	46.8	20.5

[a] HFD = High-Flow Rate Dichotomous Sampler

dichotomous sampler. Eliminating this sampling period reduces the difference in mean fine-particle collection differences to less than 3.0 $\mu g/m^3$. For both the prototype HFD-versus-dichotomous fine and total comparisons, the direction of the relative bias consistently suggests a 6-8% mass under-collection by the prototype HFD samplers during this test period. A visual inspection of the separator cones revealed depositions prior to particle separation, suggesting that particle loss in the separator was the principal cause for this bias. This relative bias is most probably associated with sampler construction and can be minimized through sampler design and operational improvements. No differences were observed between the averaged prototype HFD and dichotomous coarse-particle collections. Hypothesis testing of the coarse regression components suggested that the slope does not, however, follow the theoretical 45° line. Each prototype HFD unit was independently tested against corresponding dichotomous size fraction collections. The statistical results revealed one prototype HFD total channel to be statistically different from the dichotomous sampler at the 5% significance level. The remaining five paired prototype HFD and dichotomous parameter tests revealed no statistical differences between mean particle collections or biases estimated by the regression analysis.

The averaged prototype HFD size fractions were statistically compared against standard EPA high-volume sampler collections. Correlations between the two prototype HFD and conventional high-volume sampler size fractions were again excellent ($r > 0.95$), with no statistical differences in sampler means observed. The averaged prototype HFD total versus PM_{10} regression slope was marginally rejected at the $p = 0.015$ level. Testing the EPA high-volume samplers against corresponding dichotomous samplers again yielded only marginal differences between sampler means (2.9 $\mu g/m^3$) and a rejection of the slope hypothesis ($p = 0.025$) for the PM_{10} size fraction comparisons.

Comparisons between PM_{10}, $PM_{2.5}$, and prototype HFD high-volume collections with corresponding dichotomous particle collections revealed consistent, negative relative biases (ranging from -1.3 to -3.1 $\mu g/m^3$). Similar particle or substrate losses resulting from sampling and shipment of high volume sampler quartz fiber filters have been previously reported (Highsmith et al. 1986). Other potential contributors for this under-collection of particles are the observed difficulties in sampler flow calibrations and measurement, acceptable errors in filter weighings, leaks, particle loss, etc.

Samples collected during the second period were evaluated for both particle concentration as well as sample chemistry. Fine-particle concentrations during this period averaged 36.1 $\mu g/m^3$ (ranging from 19.1 to 78.7 $\mu g/m^3$) with a coarse/fine ratio of 0.20. Fine-particle potassium and volatile carbon concentrations (mean = 0.124 g K/m^3 and 16.2 $\mu g\ C_v/m^3$) correlated well with fine-particle concentrations ($r = 0.949$ and 0.975, respectively). Fine-particle lead (mean = 0.034 $\mu g\ Pb/m^3$) and elemental carbon (mean = 1.86 $\mu g\ C_e/m^3$), inorganic constituents typically associated with automotive activity, did not vary with fine-particle concentrations, suggesting minimal contributions from automotive emissions. The lead-to-bromine ratio averaged slightly higher than 3, the value associated with automotive emissions. The increase in total particle concentrations, shift in particle size distribution, and elemental composition of the samples collected indicates that woodsmoke emissions heavily impacted the airshed during this second sampling period. These results approximate the values observed in the earlier IACP pilot study results collected from a woodsmoke impacted neighborhood.

Prototype HFD, $PM_{2.5}$, and PM_{10} samples from eight of the nighttime sampling periods were analyzed for extractable organic matter (EOM). Paired-sampler size fraction data was statistically analyzed, as previously discussed, and summarized in Table 2. Results for the prototype HFD and EPA high volume sampler fine- and total-fraction extractable organic matter agreed well ($r > 0.99$), with no statistically significant differences observed between paired data. Tests for proportional (slope # 1) or fixed (intercept # 0) relative bias yielded no statistical differences between prototype HFD sampler and corresponding $PM_{2.5}$ and PM_{10} high volume organic collection efficiencies. Prototype HFD coarse-particle concentrations were relatively small and accounted for slightly more than 15% of the total sample organics. The coarse sample extractable organic matter represented 33% of the coarse particle concentration (mean = 2.2 $\mu g/m^3$ EOM), a value approximating the experimental error typically associated with the sampling and analytical procedures. Correlation between coarse-particle concentration and extractable organics ($r = 0.759$) is poor and suggests that, in this experiment, coarse particles did not significantly contribute to or interfere with the total sample organic compounds. Samples collected in airsheds with a larger coarse-to-fine ratio or in airsheds where the

Table 2. Statistical analysis by sampler particle size fraction.

Sampler Comparison	Comparison	N	Slope	Intercept	Correlation Coefficient	Paired Difference Student t-test	Hypothesis M=1, I=0
[a]HFD_1 vs HFD_2	Fine	10	1.049	-0.827	0.970	No	OK
	Coarse	8	1.061	-0.976	0.975	No	OK
	Total	9	1.037	1.470	0.990	Yes	Reject
Average HFD vs Dichot	Fine	9	1.085	-7.164	0.950	Yes	OK
	Coarse	9	0.820	1.225	0.981	No	Reject, slope
	Total	9	0.924	-0.762	0.979	Yes	Reject
Average HFD vs Hi Vol	Fine	10	0.956	-2.201	0.944	No	OK
	Total	9	0.839	5.408	0.989	No	Reject, slope
Hi Vol vs Dichot	Fine	9	1.068	-3.292	0.996	No	OK
	Total	9	1.095	-7.092	0.992	Yes	Reject, intercept
$PM_{2.5}$ vs PM_{10} Hi Vol	-	10	0.767	1.165	0.982	Yes	Reject, slope
Average HFD vs Hi Vol	Fine [b]EOM	8	0.979	0.964	0.991	No	OK
	Total EOM	8	0.925	1.739	0.988	No	OK
Average HFD Coarse vs Total	EOM	7	0.154	1.392	0.421	Yes	Reject, slope
$PM_{2.5}$ Hi Vol vs PM_{10} Hi Vol	EOM	10	0.907	0.719	0.990	No	Marginally Reject

[a] HFD = High-Flow Rate Dichotomous Sampler

[b] EOM = Extractable Organic Matter

coarse particles are more heavily influenced by local emissions may yield slightly different results.

Overall, these statistical findings suggest that the two prototype HFD samplers are indistinguishable. The results demonstrate that the prototype samplers collect fine, coarse, and total size fractions directly relatable to standard PM_{10} dichotomous or EPA high-volume sampling instrumentation for the ambient conditions observed during this study. Although the prototype samplers may require validation prior to implementation in airsheds having high coarse concentrations, these statistical results should encourage the immediate use of locally fabricated prototype units by organizations lacking access to commercially available dichotomous instrumentation. The results also support the use of commercially

available PM_{10} sampling instrumentation in lieu of the resource-demanding $PM_{2.5}$ impactor high volume samplers for the collection of particulate samples when detailed organic or bioassay analyses are planned. When large quantities of the three size fractions are required, the prototype HFD samplers can be employed to collect sufficient quantities to support the extensive analytical schemes.

CONCLUSIONS

The results demonstrate excellent reproducibility between the two prototype samplers. Comparisons between the prototype samplers and standard EPA high volume samplers revealed no statistically significant differences in mass collection or chemical composition. The data demonstrates that the prototype HFD sampler collections are comparable to standard PM_{10} dichotomous sampler collections without the additional filter media and analytical resource expenses. This sampler can be readily assembled through a machine shop by using standard high-volume and medium-volume sampler components. Difficulties in flow measurement and particle losses can be minimized through additional sampler design modification. The major benefits of the prototype sampler are that it: (1) is comprised of standard high-volume sampler components and locally procured components, (2) is supported with a laboratory four-place analytical balance, and (3) collects sufficient mass for bioassay analysis. This sampler could be readily built from drawings available through EPA and operated to characterize airsheds where current PM_{10} dichotomous samplers are unavailable or resource-prohibitive.

The study results also demonstrate the comparability between standard PM_{10} and $PM_{2.5}$ methodology in airsheds dominated by fine-particle emission sources. The results suggest that when the coarse-particle fraction represents less than 5% of the PM_{10} sample, the coarse size fraction does not significantly contribute or interfere with the PM_{10} sample chemistry. These results must be validated prior to the use of the prototype samplers in airsheds where the coarse-to-fine ratio exceeds 0.5 or where the coarse-particle concentrations result from local emission sources.

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DISCLAIMER

The research described in this paper has been reviewed by the Atmospheric Research and Exposure Assessment Laboratory, EPA, and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the Agency nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

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ASSESSMENT OF LEAD IN ROADSIDE VEGETATION IN BAHRAIN

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Leaves from five species of trees were sampled at 18 locations in Bahrain and were analyzed for lead. Lead levels in leaves from areas with high traffic density were generally higher than lead levels in leaves from areas with low traffic density. The lead levels found in this study are in the range of 9μg/g to 420 μg/g, which is low compared to values reported for leaves of trees along busy roads in developed countries. However, the concentration of lead is increasing because of the extreme meteorological situation in Bahrain and the heavy traffic coming from other Gulf countries across King Fahad Causeway between Saudi Arabia and Bahrain.

INTRODUCTION

Environmental studies have shown that vegetation is an important sink for atmospheric pollutants (Clyde 1971). Results from the developed countries of Europe and the United States correlate metallic air pollutants with actual levels present in vegetation (Barness et al. 1976). Studies from various developing countries, which monitored atmospheric pollution through vegetation, have shown lead uptake in plants from automobile exhaust (Osibanjo and Ajayi 1980; Fatoki 1987; Krishnayya and Bedi 1986).

Due to the rapid establishment of new industries, burning fossil fuel for various purposes to fulfill energy requirements, and the tremendous increase in the number of automobiles, environmental pollution might reach an alarming level in the Arabian Gulf countries. Therefore, there is a need for supporting data from these countries.

It is reported that the quantity of lead in air in some urban and rural areas, which ranged from 0.29 to 1.04 $\mu g/m^3$ according to a study carried out by the Bahrain Centre for Studies and Research (BCSR), is mainly from automobile exhaust emissions, as there are no other sources around these areas. A 366% increase in traffic density caused a lead increase in air of 182%, which consequently led to an increase of 29% lead in blood among adults and children (BCSR 1985).

The level of lead in Bahrain's super grade and normal grade gasoline is 0.84 g/L, which is higher than the permissible levels allowed in Europe and the United States, and ranges from 0.15 to 0.4 g/L. Since August 1989, the level of lead in Bahrain's super grade and normal grade gasoline has been cut down to 0.4 g/L. Most of the industries are located in the southeastern region of Bahrain which is relatively away from residential areas. Therefore, a 182% increase of lead in air is mainly from automobile exhaust emissions. No measurements of the accumulated uptake of lead in vegetation as an indicator of air pollution is reported in Bahrain. This paper provides results of a study carried out in low, medium, and high traffic density areas in Bahrain on the level of lead uptake in roadside vegetation from automobile exhaust.

Table 1. Description of tree species, location, and concentration of lead in leaves.

Botanical Name	Leaf Feature	Tree Location, and distance from road (m)	Site Description, and traffic density*		Concentration (ug Pb/g sample) dry weight
Ficus (Singaporans)	Slightly smooth, thick, large surface area	S_1, 5	Al-Naim Hospital	H	420
Terminalia Catappa	Rough, brittle, large surface area	S_2, 5	International Hospital	H	410
Nerium Oleander	Smooth, thick, large surface area	S_3, 5	Al-Naim Hospital	H	210
Acasia	Soft, slightly smooth, small surface area	S_4, 5	International Hospital	H	180
Eucalyptus Camaldulensis	Slightly smooth, porous thick	S_5, 50	International Hospital	M	100
Nerium Oleander	Smooth, thick, large surface area	S_6, 50	International Hospital	M	58
Ficus (Singaporans)	Slightly smooth, thick large surface area	S_7, 50	Ibn-Sina Health Centre	M	90
Nerium Oleander	Smooth, thick, large surface area	S_8, 50	Ibn-Sina Health Centre	M	52
Acasia	Soft, slightly smooth, small surface area	S_9, 50	Jalal Gardens, Budaiya	M	80
Nerium Oleander	Smooth, thick, large surface area	S_{10},50	Jalal Gardens, Budaiya	M	52
Terminalia Catappa	Rough, brittle, large surface area	S_{11},50	Budaiya Health Centre	M	77
Nerium Oleander	Smooth, thick, large surface area	S_{12},50	Budaiya Health Centre	M	50
Eucalyptus Camaldulensis	Slightly smooth, porous thick	S_{13},50	International Hospital	M	20
Eucalyptus Camaldulensis	Slightly smooth, porous thick	S_{14},50	Barbar Village (Budaiya)	L	10
Terminalia Catappa	Rough, brittle, large surface area	S_{15},50	Barbar Village (Budaiya)	L	10
Ficus (Singaporans)	Slightly smooth, thick large surface area	S_{16},50	Barbar Village (Budaiya)	L	10
Nerium Oleander	Smooth, thick, large surface area	S_{17},50	Barbar Village (Budaiya)	L	9
Acasia	Soft, slightly smooth, small surface area	S_{18},50	Barbar Village (Budaiya)	L	9

* H, M, and L = high, medium, and low traffic density, respectively.

EXPERIMENTAL

Sampling and sample description

Five species of trees with different leaf features (degree of roughness and surface area), as shown in Table 1, were sampled from 18 locations in Bahrain to represent areas of different traffic density. The sampling points, designated S_1 to S_{18}, included high traffic density areas approximately 5 m away from the highway (S_1 to S_4), medium traffic density areas (S_5 to S_{12}), and low density residential areas (S_{13} to S_{18}) about 50 m away from the highway. Trees of about the same age, situated close to the highway, were selected. The leaves were carefully removed at an average height of about 2 m above the ground, following a similar procedure by Barness et al. (1976) and Ostibanjo and Ajayi (1980).

The leaf samples were kept in polyethylene bags. The samples were dried at an oven temperature of 110°C for about 2 h. Then they were pulverized to uniform size with a mill. After each grinding, the mill was thoroughly cleaned and dried to avoid contaminating the next sample.

METHOD

About 1 g of each sample was accurately weighed into a properly cleaned beaker. Ten mL of concentrated HNO_3 were added and allowed to stand for 24 h. The mixture was carefully heated on a hot plate until the production of NO_2 fumes had ceased. After cooling the mixture, 3 mL of 70% $HCLO_4$ were added. The mixture was heated again and allowed to evaporate to a small volume. The sample was transferred to a 100 mL flask and diluted to the proper volume with distilled water. All the reagents used were of research grade. Lead standard solutions (1.0 mg/L) were certified and obtained from Fischer Scientific Company (United States). These solutions were diluted carefully to the required concentrations with doubly deionized water. The analysis of each sample was done in triplicate, using a SP-800 series spectrophotometer (Atomic Absorption Spectrophotometer by PYE -- Unicam). A computerized PU 9095 data videographic furnace with autosampler, a PM 8251 chart recorder, and a PU 9090 data computer interfaced with the spectrophotometer were used for analysis. For quality assurance purposes, these analyses were done at two laboratories: Environmental Protection Committee (EPC) (Bahrain) and Bahrain University.

The instrument was set at 283.3 nm with a slit width of 0.7 nm, using a background correction. A 1% nitric acid solution was used as a blank and analyzed before each analysis to avoid matrix interference. Both a graphite furnace and a flame were used to check the reproducibility and accuracy of the data. After each analytical run, the calibration curve was displayed on the screen and a visual check was made for linearity and good replication.

RESULTS AND DISCUSSION

The results obtained from our investigation are reported in Table 1. These results give an indication that lead uptake in plants varies in accordance with the amount of lead emitted from automobile exhaust. For example, in Table 1, tree leaves in areas of relatively high traffic volume (S_1 to S_4) show high concentrations of lead, as these spots (S_1 to S_4) are close to road junctions and are also not far from gasoline stations. The lead concentration in samples S_5 to S_{12} are lower compared to S_1 to S_4, even though the tree leaves are from the same species. These samples, S_5 to S_{12}, were taken about 50 m from the highway.

These results show that the lead uptake is high in plants growing near the highway (about 5 m), where automobile exhaust emissions from leaded gasoline are high. This uptake decreases with increasing distance of the plants growing away from the highway (Cannon and Bowles 1962; Quinche et al. 1969; Page et al. 1971; Barness et al. 1976; Harve and Underdal 1976; Rodriguez-Flores and Rodriguez-Castellon 1982; Brunekreef 1983; Krishnayya and Bedi 1986). Samples S_{13} to S_{18} also show a similar trend. These samples were taken approximately 50 m from the highway where traffic density is low, which means less automobile exhaust emissions and, therefore, less uptake.

The types of tree leaves also appear to affect the lead concentration in or on leaves. For example, lead concentration from two different samples (S_1 and S_3; S_2 and S_4; S_5 and S_6; S_7 and S_8; S_9 and S_{10}; S_{11} and S_{12}) from the same location are different. This observation can be related to the nature of the leaves, since a rough-surface leaf is known to accumulate particles more than a smooth-surface leaf (Fatoki 1987).

From our study, the concentration range of lead in tree leaves from high and low traffic density areas in Bahrain is 9 μg/g to 420 μg/g. Compare this to values reported elsewhere, e.g. Canada — 100-3000 μg/g (Cannon and Bowles 1962), France — 50-400 μg/g (Quinche et al. 1969), and Britain — 100-700 μg/g (Barness et al. 1976).

A potential new source of lead is an estimated 20 to 30% (Madany and Danish 1988) increase in traffic coming into Bahrain because of the King Fahad

Causeway between Saudi Arabia and Bahrain (Statistical Abstracts 1986). Super grade gasoline in Saudi Arabia also contains 0.84 g/L lead. There is an obvious need for monitoring and control of lead.

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APPLICATION OF ENVIRONMENTAL DOSE-RESPONSE MODELS TO EPIDEMIOLOGY AND ANIMAL DATA FOR THE EFFECTS OF IONIZING RADIATION*

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Previous extrapolations of risk from exposure to radiation at low levels (such as environmental exposures) have focused on various empirical models that have some axiomatic base, sometimes called state-vector models and usually involving linear and quadratic functions. Such models are based on representations of the physical processes occurring in irradiated cells, but do not include consideration of biological factors that could cause variability in biological response. Some mathematical models employed in environmental risk assessments (such statistically based models as the multistage, logit, probit, and Weibull) reverse this problem, showing little axiomatic support but incorporating variability, although this is not biological variability at doses of interest here. This paper presents the results of the predictions of these latter models as they apply to environmental levels of exposure to ionizing radiation compared to the estimates made by the more commonly used models. The study involves analysis of data from hard rock miner exposure to radon, exposure of rats to radon, the ingestion of radium by watch dial painters and beagle dogs, and the exposure of Japanese citizens to atomic bomb explosions. These results then are compared with inferences obtained using the more conventional axiomatic models, such as the linear/quadratic models. It is demonstrated that the results are similar, providing partial evidence that current ranges placed on risk estimates are not altered much by the selection of a particular class of models for use in quantitative risk assessment and uncertainty analysis. (Ranges are dependent on how far the data is to be extrapolated.)

INTRODUCTION

During the past decade, the discipline of risk assessment has become increasingly important in the formulation of regulatory policy, particularly as the assessment relates to quantitative predictions of health effects. In its ideal form, a risk assessment should clearly present the line of reasoning underlying an estimate of risk and should include recognition of the roles of assumptions, approximations, data, theories, models, deductions used in arriving at an inference, and a discussion of the uncertainties involved.

Consider, for example, the case in which a regulatory agency attempts to regulate environmental exposures to ionizing radiation. A step in the regulatory process is the generation of an estimate of the lifetime risk imposed upon members of the regulated population, assuming a constant rate of exposure over the entire lifetime. Three separate forms of evidence might then be offered in support of any infer-

*The thoughts and ideas expressed in this paper are those of the authors and do not necessarily reflect the policies of the U.S. Environmental Protection Agency or any other governmental agency.

ence of the lifetime risk. The first form, known here as "direct empirical evidence", can be offered whenever there is some direct experience with health effects. In this case, the only assumptions required for an inference of risk is that a non-changing estimate of the risk coefficient has been obtained (a problem involving the statistical features of sampling) and that any increased incidence due to other causes has been isolated. There may have been several populations exposed to the radiation in the past, resulting in ambiguity as to which past population "best" represents the characteristics of the entire U.S. population. An alternative is to determine how and to what degree each population resembles the entire U.S. population and to look for different subpopulations. Once a past population, in which both risk and exposure have been defined, is chosen as the primary reference population, a defined link should be sought between the risk to that population and the inference of risk to the regulated population.

This simple case of direct empirical evidence can apply to the setting of limits on occupational exposures. The experience used to regulate environmental exposures is from past experiences which involve levels of radiation which often are many orders of magnitude above those of environmental interest. As a result, the risk at environmental levels is usually extrapolated from higher levels of exposure through use of a model detailing the mathematical relationship between exposures and risk. Such evidence might be termed "semiempirical", since there are data available on the relationship between exposures and risk, but these data are restricted to a domain of exposure that is much higher than the range of environmental exposures. In order to perfectly and completely justify inferences drawn from such data, the risk assessor must demonstrate that the models chosen to extrapolate the risk also explain the data at high exposures (i.e. explains the past experience) and that the same model should apply without modifications at lower levels of environmental exposure. Since data on several sets of past exposures at high levels might be available to the risk assessor, the semiempirical approach shares all of the problems of the direct empirical approach, as well as the additional problem of justifying the adoption of a particular mathematical model for extrapolation.

An important feature of semiempirical evidence is the existence of data in human populations which differ from the regulated population in regard to the magnitude of the exposure variable (environmental concentration, dose, etc.). Risk assessors making use of semiempirical evidence must make competing choices between (1) the form of the dose-response equation and (2) the index to employ as the measure of dose. In other cases, however, there may be no available data at any exposure level concerning the radionuclide of regulatory interest. An additional theory concerning the manner in which results are to be scaled between radionuclides or species then is introduced into the logic of inferences of risk. These theoretical inferences can approximate semiempirical evidence whenever the exposures of regulatory interest can be placed onto a commonly accepted conceptual foundation with other radionuclides and/or species for which direct or semiempirical evidence is available. For example, the risk of developing bone sarcomas from exposures to uranium or plutonium might be inferred from data obtained from exposures to radium, assuming that both exposures can be placed on a common unit of measure for risk (such as the dose to endosteal cells). The consistency of these inferred consequences with direct experience needs to be checked also. Similarly, human risks might be inferred from data on radium exposures in experimental animals, again assuming a common index of risk is available and supportable (such as understanding the relative sensitivity to induction of sarcomas among mammalian species).

The theoretical inference of risk for uranium or plutonium then can involve uncertainties due to: (1) the choice of data concerning radium exposures, if several sets of data are available; (2) the mathematical extrapolation model used to relate risks at high radium exposures to the risks at lower levels; (3) the choice of exposure variable (or "risk surrogate") which should be employed in relating exposures to radium, uranium and plutonium; and (4) where applicable, the choice of theory concerning how results are to be scaled between species. The third form of uncertainty can be appreciable whenever the biological mechanism (such as cancer induction) is not understood. For example, the exposure variable for lung cancer induction could be taken to be the absorbed dose to lung cells, the hit probabilities for these cells, or the fraction of nuclei with a prescribed number of traversals by alpha particles (Crawford-Brown 1985). Each of these choices differs from the others in the form and magnitude of the relationship between exposure (environmental concentration) and the measure of risk (dose, hit probability or nucleus traversals). As a result, they yield different inferences of risk following exposure to uranium or plutonium, even if the same extrapolation model is applied to the set of data concerning exposures to radium.

The present paper examines the effect of choice of model for extrapolation on inferences of environmental risk following exposure to ionizing radiation at environmental levels. The models employed are typically referred to as statistical or environmental models and consist of the multistage, logit, probit, and Weibull models. These models are the ones commonly used to extrapolate the effects of chemical carcinogens to environmental levels. The range of values that they predict are roughly the entire range possible using different mathematical expressions, including supra-linear possibilities. As demonstrated elsewhere for volatile organic compounds and pesticides, the estimates of risk at environmental levels (corresponding to a lifetime risk of 10^{-10} to 10^{-4}) using different environmental models can span a range of four to six orders of magnitude (Cothern 1985, 1986). This situation arises even when only a single set of data forms the basis for semiempirical evidence, indicating that the uncertainty underlying risk estimates for environmental exposures is dominated by the choice of model for extrapolation from high to low doses. In some cases, however, the uncertainty in the exposure estimate can be comparable. Because of this feature, the uncertainty introduced by a choice of model for extrapolation can be taken as a measure of the overall uncertainty associated with quantitative risk estimates. The contribution to overall uncertainty from known biological processes needs to be considered as it can be directly checked. However, its contribution is usually small by comparison.

The following discussion is divided into several parts. The first describes the mathematical models used, including the extent to which the various environmental models can be given support by fundamental scientific evidence. The second part describes the fits of these models to data concerning (a) exposure of miners and rats to radon, (b) exposure of watch dial painters and beagles to radium, and (c) exposure of the atomic bomb survivors to external radiation (primarily gamma). The third part focuses on complexities and problems associated with specific sets of data, particularly as these problems might influence inferences of risk. Finally, the discussion closes with an examination of some generic problems which arise in attempting to draw inferences of lifetime risk from data concerning past exposures.

THE MODELS

A wide range of mathematical models has been used in the past to describe the risks associated with exposures to ionizing radiations. The most commonly adopted models have been the linear, quadratic, and linear-quadratic models employed by the National Academy of Sciences which are predicated upon the induction of one or two cellular lesions (NAS 1980). Other analytical functions, such as the multistage, probit, Weibull, and logit models, have been prevalent in developing predictions of risk for environmental chemicals but have been less widely used for describing radiation effects (Cothern 1985, 1986, 1987). These latter models might, therefore, also form the basis for modeling efforts in radiation exposures. Accordingly, it is of great interest to compare the predictions of these models to those obtained using the more widely employed models.

As will be shown in the next section, these environmental models lead to large differences in the inference of risk at low levels of exposure, even when a single set of data is employed. These various inferences, however, do not possess equal logical support, since they are derived from models with differing levels of validity (or evidential support).

MODEL FITS TO DATA

As mentioned earlier, one of the measures of support for an extrapolation model or equation (aside from the existence of supportable axioms) is the ability of the model to correctly predict available data. Due to the extreme variability in available epidemiological and animal dose-response data, however, this criterion provides only a weak test of competing models. Most models can be made to fit the data to within comparable measures of "goodness-of-fit". For this reason, dose-response data usually are used only to obtain parameter estimates for an equation, assuming that the equation is correct.

In this section, the environmental or statistical models are fit to a variety of sets of epidemiological data and the determined model parameters. These equations then are used to estimate risks at environmental levels of exposure. In each case, the upper 95% confidence level for the curves at several levels of dose (or its surrogate) also is displayed.

Exposure of hard rock miners to radon

Figure 1 displays the dose-response data for four mining groups exposed to radon (data given by the solid symbols). The surrogate measure of dose has been taken to be cumulative exposure in units of Jm^{-3} s (historically working level months). These data include studies of the Colorado Plateau uranium miners, the Swedish metal miners, the Czechoslovakia uranium miners, and the Newfoundland fluorspar miners (Thomas et al. 1985). In all cases the surrogate dose has a large uncertainty associated with it since

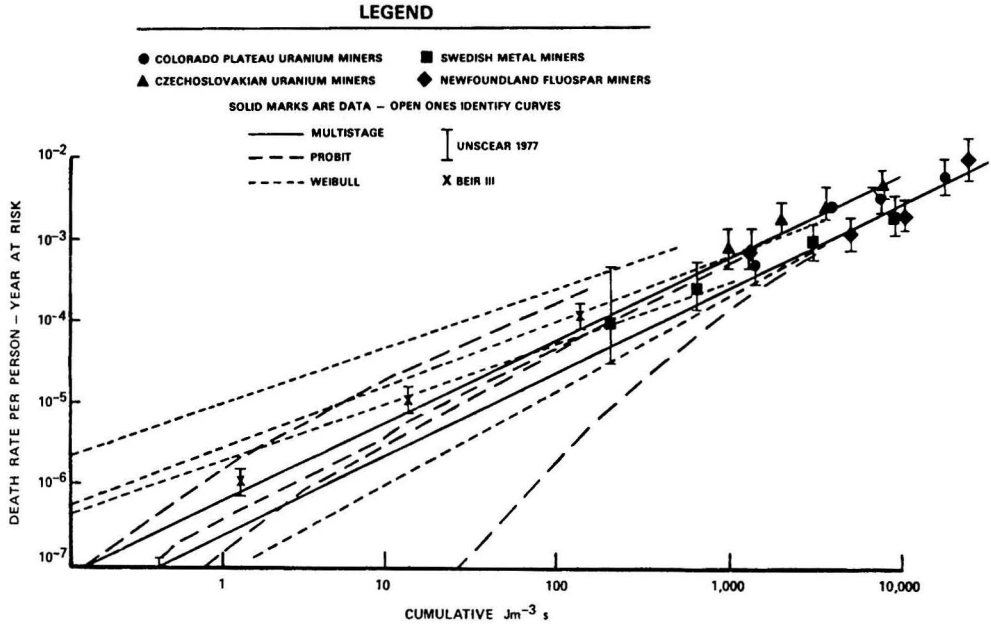

Fig. 1. Dose-response data for radon exposure in four miner studies. The solid symbols are the data and the open symbols are the model fits for the multistage, probit and Weibull models. In general the logit model fits are close to those of the Weibull model. The original data were in units of cummulative working level months (WLM) where 1 WLM = 12.97 Jm^{-3} s.

personnel monitors were not used for any of these data. In each case, the measure of risk is the excess incidence rate, computed using the difference between the observed and expected cases over the course of follow-up. For purposes of the present analysis, confounding factors such as cigarette smoking and exposure of the miners to other contaminants in the mines are not included. The logit and Weibull models yield essentially the same curves for each of the four sets of epidemiological data. Figures 2 to 5 display the data and curve fits separately for each of the study populations. As can be seen from these curves, no model is consistently highest or lowest at the lower levels of cumulative exposure.

The area of environmental interest lies roughly in the range of 10 to 100 Jm^{-3} s. Choosing 10 Jm^{-3} s as the exposure level of interest, the range of risk estimates can be determined directly from Fig. 1. This range then includes the uncertainty introduced by both the choice of data and the model. There is no absolute guarantee that the actual risk is in the ranges given. The actual value could be outside the ranges given. At 10 Jm^{-3} s the range of risk estimates may be between about 10^{-8} and 5×10^{-6} deaths per Jm^{-3} s per person-year. A more reasonable method to assign a range of estimates would be to take the central two-thirds of these estimates. Using this procedure, the range would be 0.3 to 2 deaths per million person-years per Jm^{-3} s.

Assuming 50 years of expected life after exposure and a latent period of 20 years, the above range of risk estimates indicates a lifetime risk of between 1.5×10^{-5} and 10^{-4}. By contrast, Evans et al. (1981) have deduced that a value of approximately 0.8×10^{-5} 10^{-4} excess lung cancer cases per Jm^{-3} s per person is an upper limit to the lifetime risk. A summary of various ranges of risk estimates for radon exposures can be found in Table 1, showing good agreement between the risk factor derived from environmental model analysis with that derived from more conventional analysis.

Exposure of rats to radon

Chemelevsky et al. (1982) and Chameaud et al. (1985) have published dose-response data for exposure of rats to inhaled radon. Their data are reproduced in Fig. 6, accompanied by the various model predictions and upper 95% confidence limits for the risk. The estimated fit by Chemelevsky of various exposure levels is displayed by the curved brackets

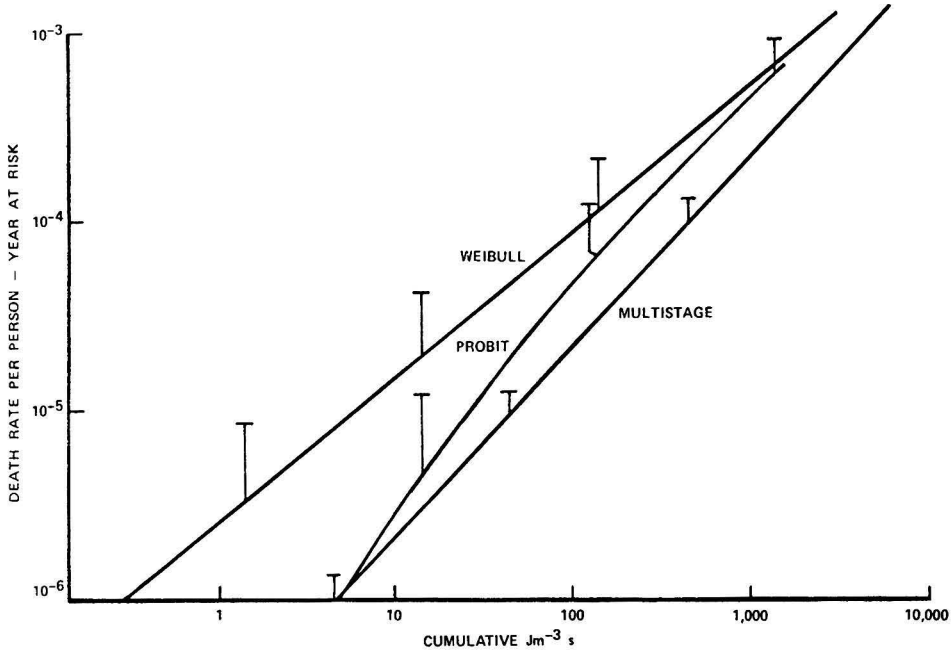

Fig. 2. Fits of the multistage, Weibull and probit models to the radon exposure data for the Colorado Plateau uranium miners. The error bars are the upper 95% confidence limits. The lower 95% confidence limits are the horizontal axis at zero risk. The logit model fits are essentially the same as those for the Weibull model. The original data were in units ofcummulative working level months (WLM) where 1 WLM = 12.97 Jm^{-3} s.

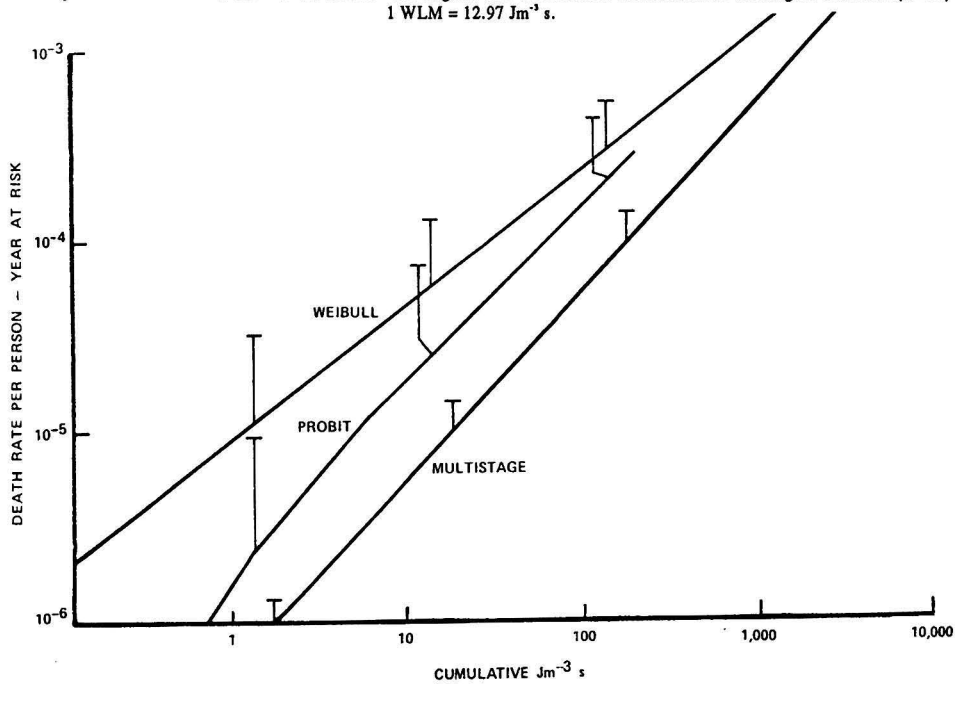

Fig. 3. Fits of the multistage, Weibull and probit models to the radon exposure data for the Czechoslovakian uranium miners. The error bars are the upper 95% confidence limits. The lower 95% confidence limits are the horizontal axis at zero risk. The logit model fits are essentially the same as those for the Weibull model. The original data were in units of cummulative working level months (WLM) where 1 WLM = 12.97 Jm^{-3} s.

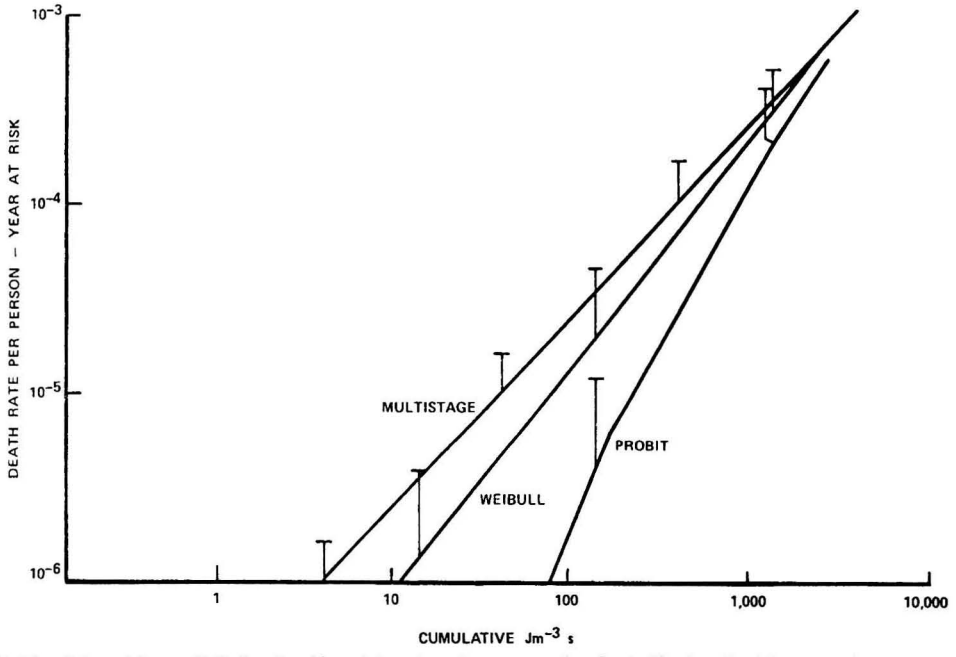

Fig. 4. Fits of the multistage, Weibull and probit models to the radon exposure data for the Newfoundland fluorspar miners. The error bars are the upper 95% confidence limits. Thelower 95% confidence limits are the horizontal axis at zero risk. The logit model fits are essentially the same as those for the Weibull model. The original data were in units of cummulative working level months (WLM) where 1 WLM = 12.97 Jm^{-3} s.

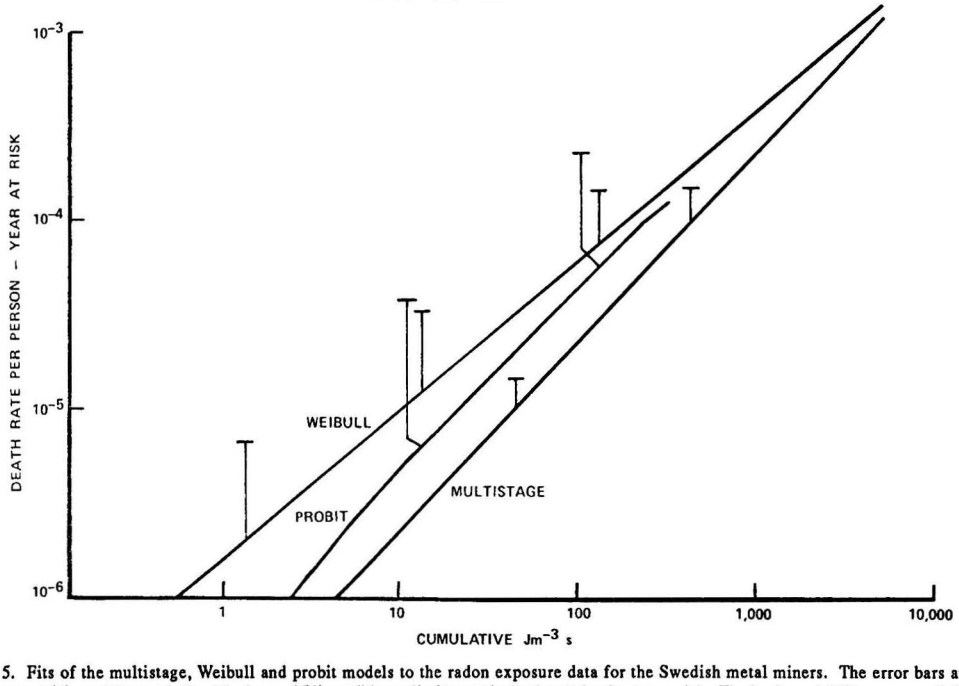

Fig. 5. Fits of the multistage, Weibull and probit models to the radon exposure data for the Swedish metal miners. The error bars are the upper 95% confidence limits. The lower 95% confidence limits are the horizontal axis at zero risk. The logit model fits are essentially the same as those for the Weibull model. The original data were in units of cummulative working level months (WLM) where 1 WLM = 12.97 Jm^{-3} s.

Table 1. Comparison of risk factors derived from human epidemiology studies of exposure to radon (Miller and Buster 1985).

Average Injected ^{226}Ra μCi/kg	Number of Dogs Injected	Number of Dogs With More Than One Bone Tumor
10	10	9
3.2	12	12
1.1	12	11
0.34	12	5
0.17	12	2
0.062	23	2
0.022	25	1
0.0074	10	0
0	44	0

in the figure. Risks are computed as the excess incidence over the complete lifetime of the rats, although the clinical observation was restricted to nonlethal tumors. Competing effects were accounted for in the analysis. The cumulative exposures to the rats were converted to units of human equivalent dose through the assumption that the dose levels related to the surface area of the species (Cothern and Van Ryzin 1985).

Examining the curves at a cumulative exposure of 10 Jm^{-3} s, the range of lifetime incidence is about 8×10^{-6} (for the probit) to 1.1×10^{-3}. The confidence level for this range might be of the order of 95%. If the probit model is rejected, the range narrows to

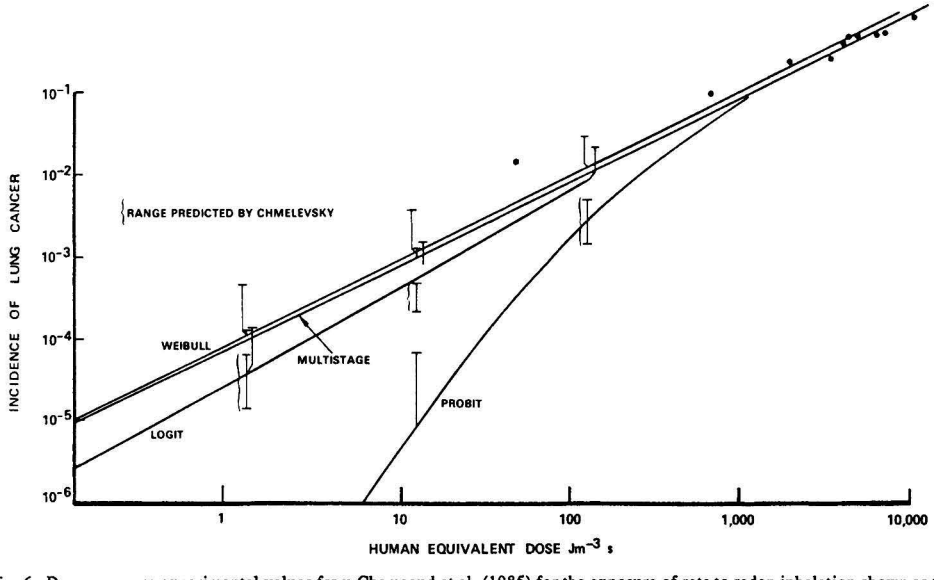

Fig. 6. Dose-response experimental values from Chameaud et al. (1985) for the exposure of rats to radon inhalation shown as the solid dots. Shown also are the fits to these points for the multistage, probit, Weibull and logit models. The error bars are the upper 95% confidence limits. The lower 95% confidence limit is the zero incidence axis. The three error bar ranges with curly brackets are the range predicted by Chemelevsky et al (1982). The original data were in units of working level months (WLM) where 1 WLM = 12.97 Jm^{-3} s.

6×10^{-4} to 10^{-3} for which a two-thirds confidence level might be implied. This is slightly above the range of 0.8×10^{-5} to 3×10^{-5} excess cases per Jm^{-3} s reported by Cross et al. (1985). These results also agree with the data on human populations detailed in the previous subsection.

Radium watch dial painters

The most complete set of data concerning the cancer incidence in the radium watch dial painter population has been assembled and analyzed at Argonne National Laboratory (Rowland et al. 1978, 1983). Those data are displayed in Fig. 7, which also shows the curve fits for the various models and the upper 95% confidence limits. The figure also shows the curve determined by Rowland et al. (1983) using a quadratic model for bone sarcoma induction (including cell killing).

If the concentration of environmental radium is taken to be 0.2 Bq L^{-1} in drinking water (the average concentration of ^{226}Ra in public drinking water supplies is in the range of 0.01 to 0.07 Bq L^{-1}), and ingestion is at a rate of 2 liters per day, the systemic intake, I, to blood over the course of 50 years would be 1.4 Bq. In this calculation, 0.2 is used as the fraction of the radium reaching the bloodstream (Mays et al. 1985). Only the systemic intake is important, since this was the unit of exposure utilized in the epidemiological studies as summarized in Fig. 7.

If the probit model is rejected as an outlier, the range of risks from ingestion of 1.4 Bq of radium would be about 1×10^{-7} to 5×10^{-7} bone sarcomas per person-year. Assuming a normal life span of 70 years and a 20-year latency period, the lifetime risk would be 5×10^{-6} to 2.5×10^{-5} per person. This range is slightly below the values of 3.5×10^{-5} given by Rowland (1978, 1983) and 4.4×10^{-5} given by the International Commission on Radiological Protection (ICRP 1979) dosimetric model as calculated by Cothern et al. (1983).

Radium in beagles

The above values for humans can be contrasted with the two principal studies concerning bone sarcomas induced in beagles reported by Wrenn et al. (1986) (see Table 2) and by Raabe et al. (1981). The data extrapolation curves and upper 95% confidence limits for these two sets of data are given in Figs. 8 and 9. In each case, the doses are in units of human equivalent-injected radium, scaled according to relative body masses. Using the assumed intake of 1.4 Bq computed in the previous subsection, the corresponding human-equivalent injected radium for a 70 kg-human would be approximately 200 Bq kg^{-1}. The range of lifetime risks for the data from both Raabe et al. (1981) and Wrenn et al. (1986) then would be

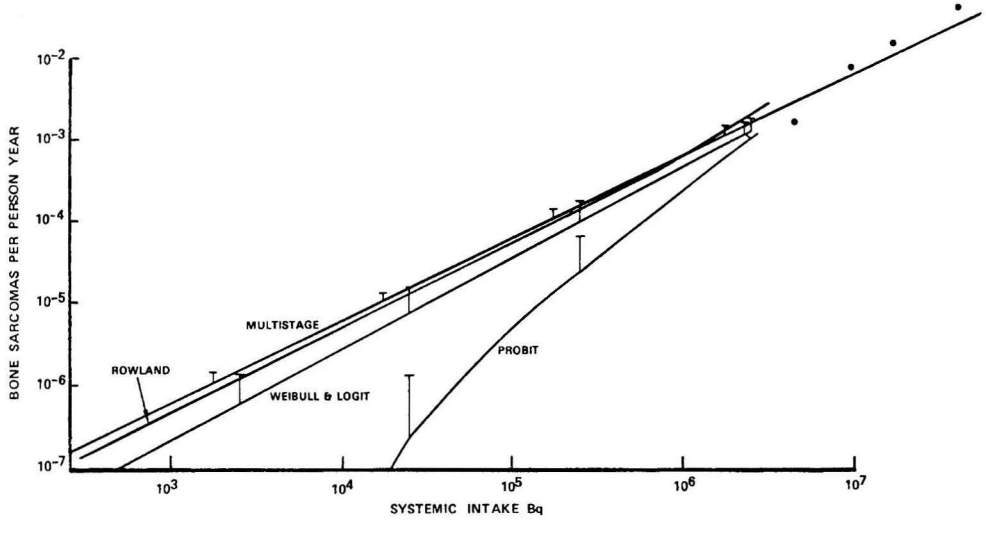

Fig. 7. The solid points are the dose-response data from Rowland et al. (1978) for the radium watch dial painters who ingested radium. The curves are the fits to that data for the multistage, probit, logit and Weibull models. Also shown is the curve suggested by Rowland et al. The original data were in units of microcuries.

about 10^{-6} to 10^{-3} bone cancers per person per lifetime. This range overlays that discussed above for the radium watch dial painters, but based on this analysis the dog appears to be more likely radiosensitive to bone sarcoma induction than man (Wrenn 1986).

The atomic bomb survivors

A major effort has been expended since World War II to collect and analyze the data describing the human health effects from the atomic bombs dropped on Hiroshima and Nagasaki (Ishikawa et al. 1981). Although the data covers all cancer endpoints, only that due to leukemia will be analyzed here, since it is the one set of data which shows a distinct correlation with exposure.

The data and the fits for the various models (along with 95% confidence intervals) are shown in Fig. 10. Also displayed are the risk estimates from Land (1980) for the linear model ($I = 2.5 \times 10^{-4}$ D, where D is in grays) and quadratic model ($I = 0.016 \times 10^{-2}$ D^2, where D is in grays) (Land 1980). At an environmental level of 0.01 Gy, the range of model estimates is 2.5×10^{-10} to 2.5×10^{-6} leukemias per person-year or a range of 10^4.

PROBLEMS AND COMPLEXITIES

The extrapolation of lifetime risk estimates from the study populations to the general population will contain areas of logical uncertainty unrelated to the choice of doseresponse model. The first to be discussed here concerns selected bias, introduced by the feature that epidemiological populations often enter the study only because they possess some characteristics which differ from the average characteristics of the total population. A typical example is the healthy worker effect. The standard procedure for reducing confounding variables is to employ internal reference groups as the control, or unexposed population, rather than using the U.S. population as a base. Use of such an internal reference group does not alleviate the problem completely, however, if the study population differs systematically from the U.S. population with respect to radiosensitivity.

Fortunately, in each of the studies employed in this paper, the reference group was the internal group with lowest exposures. It must be noted, however, that for the hard rock miners and watch dial painters all participants in the study were part of the general work force and, hence, not a representative sample from the U.S. population either in health status or age. In addition, only males form the data base for the hard rock miners, and mostly females form the data base for the watch dial painters. In the case of the radium dial workers, only those women who agreed to participate were used in the study, raising the possibility of selective bias if cases were more (or less) likely to agree to participate.

Table 2. Summary of results for beagles injected with ^{226}Ra.

Information Source	Death Rate From Lung Cancers Per Million Person-Year Per WLM*	Death Rate From Lung Cancers Per Million Person-Year Per $Jm^{-3}s$
Thomas et al. (1985)	3.4-17.8	0.26-1.4
United Nations Scientific Committee on the Effects of Atomic Radiation (1977)	6.7-15	0.52-1.2
BEIR III (NAS 1980)	10	0.77
BEIR IV (NAS 1988)	5	0.39
Cross et al. (1985)	1.5-50	0.12-3.9
NCRP Report 78 (1984)	8-16	0.62-1.2
Present Work	3-25 (at the 1 WLM level)	0.2-2 (at the 10 $Jm^{-3}s$ level)

*The estimates in terms of WLM are provided for historical reference (1 WLM = 12.97 $Jm^{-3}s$).

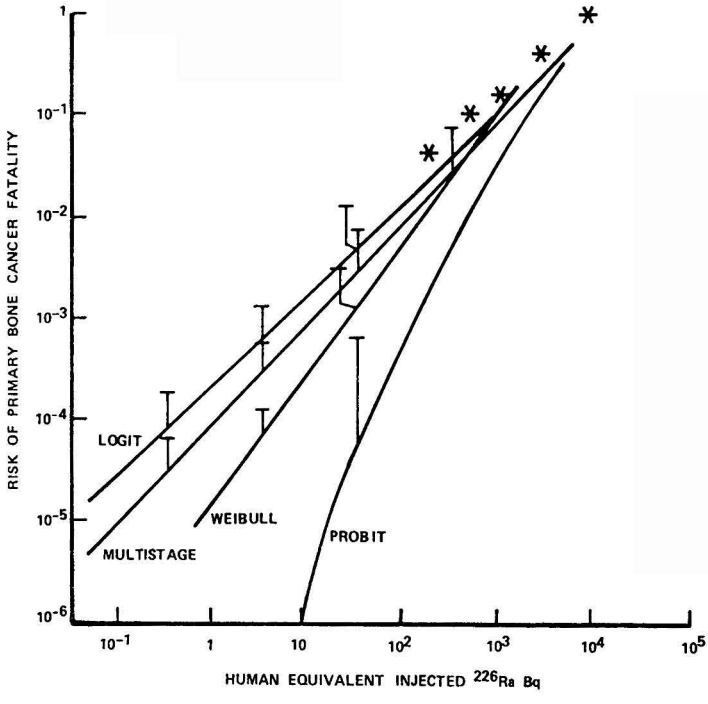

Fig. 8. Dose-response data from Table 3 for ingestion of ^{226}Ra by beagle dogs. The original data were in units of microcuries.

A second potentially significant problem with epidemiological studies is the possible presence of unaccounted past exposures in a study population or exposure to confounding substances and prior exposure to ionizing radiation. A typical confounding factor present in essentially all human epidemiological groups is smoking, with only very limited information available on smoking histories for the groups used in epidemiological studies reported earlier in this paper. Some unknown fraction of the health effects in each dose group might be due to the confounding factors (such as smoking) with the fraction perhaps changing between the various groups. The problem of confounding exposures is relatively unimportant for the studies using rats and beagles, since the exposures were controlled and included only the radon or radium. For the radium dial painters, the primary exposures are likely to have been only to the radium, although the base for the radium paint may have contributed minor chemical exposures. The atomic bomb survivors were exposed primarily to the radiation from the bomb blasts, although the stress of the bombing may have played an important role in promoting tumor expression. Finally, the studies of the uranium miners appear to be beset by the greatest potential for confounding exposures, especially to cigarette smoke, dust, diesel fumes, and particulates containing uranium and other heavy metals. There is still a great deal of controversy over the issue of the contribution of these other exposures to the induction of lung cancer in the mining groups.

The problem of previous exposures to substances associated with lung cancer, leukemia, or bone sarcomas also is of importance only in the human groups. Previous exposures should not be important for the atomic bomb survivors, since the exposures should be distributed equally between the study group and the general population of Japan. The same should roughly be true for the watch dial painters, although the potential for previous exposures which differ from the U.S. population cannot be ignored. To date, however, no evidence has been offered to suggest that these women worked in industries which involved exposures to hazardous chemicals prior to working with the radium. The miners, on the other hand, hold the greatest potential for previous exposures to hazardous materials, since their working lives generally are spent in mines. As in the case of the "healthy worker effect", this problem was alleviated partially in epidemiological studies by using

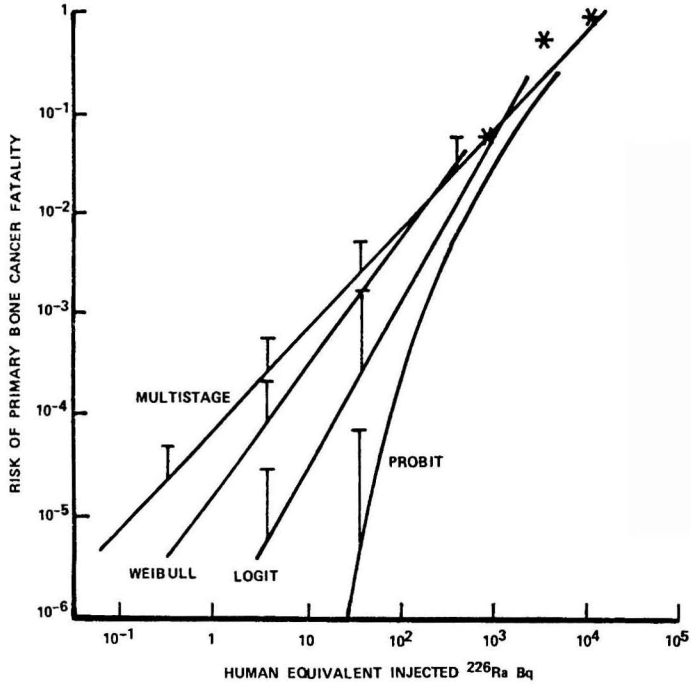

Fig. 9. Dose-response data from reference Raabe (1981) for the ingestion of ^{226}Ra by beagle dogs. The original data were in units of microcuries.

inferred reference groups chosen from the industry rather than from the U.S. population. Unfortunately, there is no information available on previous occupational exposures to radon or other substances in any dose group for the miner studies.

Both doses and disease classifications can be incorrectly assigned in an epidemiological study, resulting in misclassification. The effect on the epidemiological study can be a significant biasing or dampening of the dose-response curve. This classification of disease should not be large in the study populations used in this report, but misclassification of doses is a potentially large problem for the human studies. For example, the burdens of radium in the dial painters were obtained only retrospectively in light of existing bone burdens at the time of the study. The scarcity of good information on metabolic and dosimetric properties of radium can introduce large errors into the dose estimates (probably on the order of a factor of 4 to 5). Misclassification of doses probably is of the same order as for the watch dial painters, while random misclassification would be small for the nonhuman (rats and beagle) studies due to direct measurements of the dose delivered to and retained by the animals. Since the degree of misclassification is unknown for the various studies, all that can be stated at present is that it is likely that the direction of the bias in the dose-response curves may be dampened to varying degrees: strongest for the miners, less so for the atomic bomb survivors and watch dial painters, and insignificant for the rat and beagle studies. The risk coefficients for the rat and beagle studies, however, may be systematically too low or too high, depending upon the validity of the conversion factor used to scale doses and responses to human equivalent doses and responses.

Another problem is the potential for loss of workers to follow-up. This occurs either from the inability to locate a worker after some point in time or due to an incomplete determination of the entire exposed population. The problem is particularly acute whenever cases are more (or less) likely than controls to remain in the study. For example, cases might remain in contact out of a desire to retain perceived health care benefits resulting from the study. This would cause the inferred dose-response curve to be biased towards overestimates of response per unit exposure.

Finally, there is the consideration of the length of follow-up for an epidemiological study. For regulatory purposes, it is necessary to determine lifetime

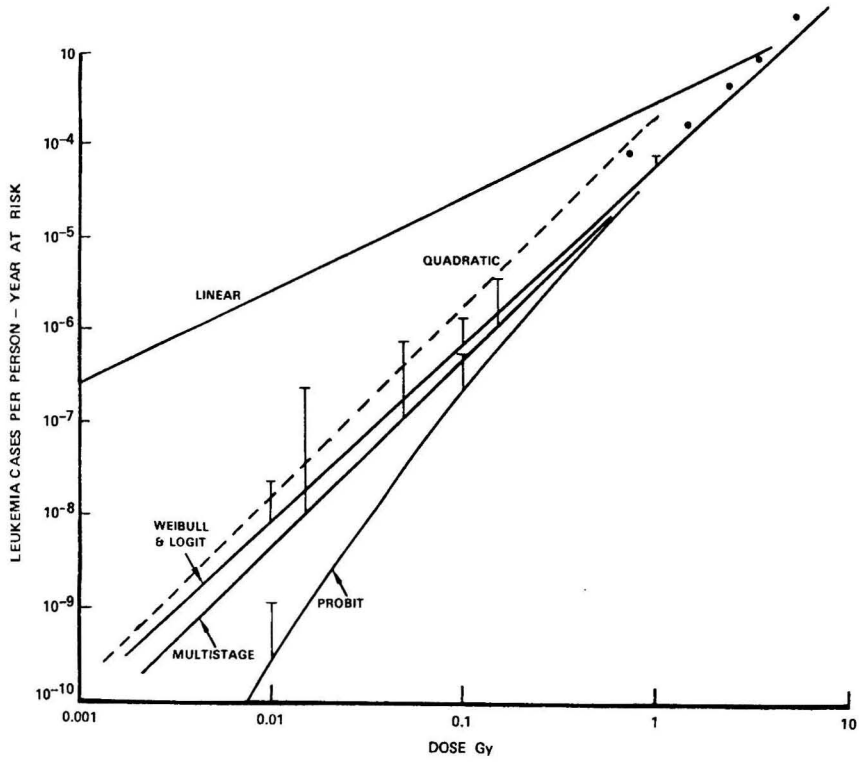

Fig. 10. Dose-response data for the incidence of leukemia (observed-expected) among the atomic bomb survivors from Hiroshima and Nagasaki. The data are combined males and females of all ages for the time period 1950-1978 (NAS 1981). The solid dots are the data taken from estimates by Land (1980). The error bars are the upper 95% confidence estimates. The lower 95% confidence limit is the zero risk level. The original data were in units of rad.

risk, rather than simply the risk during the period of follow-up. If the population has not been followed until the end of life, additional cancers may appear in the future which would result in an increased estimate of the lifetime risk coefficient. The problem becomes less important if the excess incidence rate has already dropped to its preexposure level by the end of the study, or if the population has been followed for a significant fraction of life. Since many "naturally occurring" cancers occur rapidly above age 50 this effect may be non-trivial.

For both the rat and beagle studies, most of the animals have reached the end of life. The lifetime risk coefficient, therefore, should be stable for these populations. The leukemias in the atomic bomb survivor studies have already returned to the preexposure rates, so increased follow-up is unimportant. For both the radium dial worker and human radon studies, however, the population has not reached the end of life. The estimate of the risk coefficients may, therefore, continue to change. The change probably will not be important for the radium dial workers, since the incidence rate has slowed appreciably as of the 1980s (NAS 1980). It is important to bear in mind that the lifetime risk can only increase with increasing length of follow-up, but the average annual incidence may increase or decrease depending upon whether the incidence rate is increasing or decreasing with time.

GENERAL REMARKS AND CONCLUSIONS

This paper has examined the impact of the choice of dose-response function on the extrapolation of risk to environmental levels of ionizing radiation. From this analysis, it appears that the range of risk estimates obtained using the environmental models discussed here is not appreciably different from the range obtained using axiomatic models more common to radiation risk assessments. As mentioned ear-

lier, the axiomatic models strictly apply to the induction of cells into a precancerous state, but do not formally recognize intersubject variability in the promotion of these cells. The environmental models give more formal recognition to this variability, but are not founded on physical axioms. Since the "correct" dose-response model may contain aspects of both approaches, it is encouraging to find that the two approaches approximately coincide in their range of risk inferences. The statistical approach used here is admittedly imprecise, and the agreement between approaches appears to be at least as good as the uncertainties involved.

All of the studies reported here have used absorbed dose, or some surrogate such as working level months (WLM), or microcuries of ingested radium as the predictive index. No attempt has been made to reanalyze the data in terms of other predictive indices such as hit probability.

Several other issues have remained unexamined in this paper. There is some indication that the latency period for cancers may change significantly with dose and dose-rate, suggesting that the lifetime risk may be overestimated at low doses and dose-rates if high doses and dose-rates form the epidemiological base (NAS 1980). At least in the case of alpha radiation, however, this potential overestimation is offset by the findings which suggest an increase in the risk factor (risk per unit dose) at lower dose-rates (NAS 1980) both for humans (Mays et al. 1978) and for mice (Muller et al. 1978). It is not clear, therefore, whether the net effect of lowered dose rates will be an increase or decrease in the lifetime risk coefficients.

Essentially all of the human studies (excepting the atomic bomb survivors) have examined only adult exposures. Environmental exposures, however, will occur in all age groups. From the atomic bomb survivor studies, both the relative risk and absolute risk coefficients for leukemia appear to increase at very young ages (<10 years) relative to midlife, increasing again after age 40. From these same studies it appears that the latency period decreases at younger ages (Leggett et al. 1983). The increasing risk factors, therefore, may be offset by a shorter period of expression for the leukemias. Similar information is not available for the other tumors in any detail, although the data on lung cancers in the atomic bomb survivor population should provide insight as the very young population (<15 years old at the time of the bombing) reaches the age of natural expression. It might also be noted that Spiess and Mays have found little evidence of a significant effect of age or sex in the incidence of bone sarcomas induced by ^{224}Ra (Spiess and Mays 1970, 1973).

Much more is known about the carcinogenic effects of ionizing radiation than about other environmental contaminants. Still, the major source of uncertainty in estimating risk from exposure to environmental levels of radiation lies in the choice of dose-response models. Due to the number of samples required to obtain a measure of risk at environmental levels in the form of direct empirical evidence, it appears unlikely that the range of uncertainty will be narrowed significantly by further human studies. Even if all of the problems associated with the epidemiological analysis of the data were solved, this would have little impact on the level of uncertainty in risk estimates. Only an improved understanding of the mechanism of carcinogenesis by radiation will significantly lower the uncertainty, since this will help to narrow the range of reasonable models (or at least cause some models to gain in weight over others). This points up the need to focus attention on axiomatic models which can incorporate findings from fundamental radiobiological studies.

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NATURAL RADIOACTIVITY OF FRESH WATERS IN SLOVENIA, YUGOSLAVIA

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The radioactivity of surface and ground waters in Slovenia, Yugoslavia, was assayed. About 700 samples of surface and 500 samples of ground waters were analyzed for ^{222}Rn, ^{226}Ra, natural uranium, and gross beta activity. In surface waters the highest concentrations found were 5365 Bq m^{-3}, 92 Bq m^{-3}, 215 Bq m^{-3}, and 0.4 mg m^{-3} for radon, radium, gross beta, and uranium, respectively. In surface waters the radioactivity is higher in the region of uranium ore deposits, in the vicinity of a phosphate processing plant, and in the region rich in thermal and mineral waters.

INTRODUCTION

Measurements of radioactivity of the main surface and ground waters in Slovenia, the NW Republic of Yugoslavia, started a long time ago. They were initiated by the advent of the nuclear age in Yugoslavia; namely, the construction of the first nuclear power plant at Krško and the first uranium mine and mill at Žirovski Vrh. The main purpose of this study was to screen the radioactive background before operation of any nuclear technology installation in the country in order to be able to estimate its later radiological impact on the environment. Nevertheless, there are a few non-nuclear plants (coal-fired thermal plants, phosphate mill) supposed to increase environmental radioactivity.

The main Slovene surface waters are the rivers Sava, Soča, and Drava with their tributaries (Fig. 1). The largest water catchment area belongs to the Sava. It originates under the highest Yugoslav mountain, Triglav, from two springs: one as a waterfall, Slap Savica, flowing into the glacier lake of Bohinj, and the other near Kranjska Gora skiing resort. The Sava flows diagonally through the country and joins the river Danube near Beograd, the Yugoslav capital, thus being the longest Yugoslav river. Its main tributaries are the following rivers: the Sora (composed of the Poljanska Sora and Selška Sora), Krka and Kolpa on its right bank, and the Kokra, Savinja, and Sotla on its left bank. Along its course through Slovenia, the following industries and plants are situated: an iron smelter and steel plant at Jesenice, rubber and electronic factories at Kranj, a TRIGA Mark II 250 kW research reactor near Ljubljana, the cement industry, coal mines and a coal-fired 300 MW thermal plant with fly-ash deposits at Trbovlje, a phosphate mill at Hrastnik, and a paper and pulp plant as well as a Westinghouse 635 MW nuclear power plant at Krško (started 1980).

Among the Sava tributaries, the river Sora is expected to be contaminated by waters from the uranium mine area (Kobal et al. 1978). The mine is situated at Žirovski Vrh, about 30 km upstream of the confluence of the Sora with the Sava. Waters originating from the uranium mine, the mill, deposits of barren material, and tailings of dry technological wastes flow into a small stream (Brebovščica) which after a few kilometers flows into the Polijanska Sora,

Fig. 1. Map of Slovenia with coded sampling sites for surface waters.

one of the two branches of the Sora, which later flow into the Sava.

The Ljubljanica starts as a karst river, the Pivka, which after a few km sinks underground into the Postojna Cave, emerges from the Planina Cave as the river Unica which after a few km of surface flow sinks again and finally comes out as the Ljubljanica. On its banks is Ljubljana, the capital of Slovenia with about 300 000 inhabitants Ljubljana, the capital of Slovenia is situated with its numerous small industries and a 150 MW coal-fired power plant.

The Savinja river collects waters from an industrial basin comprising coal mines, coal-fired power plants (with a joint capacity of 800 MW), factories for household appliances and leather products, production of sulphuric acid and titania, and springs of thermal waters with increased radioactivity (Kobal et al. 1979).

The other tributaries of the Sava are not considered to have enhanced radioactivity.

The Drava river comes from Austria; the Dravinja and the Mura are its main tributaries, and it runs into the Danube near Osijek. Along its course there is a series of dams for hydroelectric plants and the city of Maribor (120 000 inhabitants) with its household appliance and truck factories. The Drava collects water from the mountainous region of Pohorje known for its elevated radioactive background due to granite rocks.

The Mura river comes from Austria, too, and has only a short flow through Slovenia. It runs through a region abundant both on the Austrian and Yugoslav sides in thermal and mineral waters with increased levels of radioactivity (Kobal et al. 1979; Friedmann and Hernegger 1978).

The rivers Sava and Drava flow into the Danube and thence into the Black Sea.

On the other hand, the Soča (Isonzo) river, with its spring under Triglav, flows into the northern Adriatic Sea. Along its course there are three dams for

hydroelectric plants and a cement mill at Anhovo. Its tributary, the river Idrijca, flows through a mercury mining and smelting plant at Idrija and is thus heavily polluted by mercury. It might also be contaminated by natural radionuclides because it collects streams from nearby hills known for their uranium and thorium deposits. Another tributary of the Soča is the river Vipava originating from an underground karst lake and flowing through an agricultural area. Another karst river, the Reka, collects waters from a large area of the Trieste Karst, sinks into the Škocjan Caves, and comes from several springs as the river Timava at the Adriatic Coast near Trieste. There are also two short individual rivers, the Rižana and the Dragonja, with their springs near the coast which run into the northern Adriatic Sea.

EXPERIMENTAL

Along a river, several sampling points were selected, according to suspected sources of contamination and at places with good access to the bank. Altogether about 700 samples of surface waters were analyzed. All the samples along a river were taken in plastic bottles on the same day, and then the samples were transported to the laboratory, filtered through a 0.45 μm Millipore filter, acidified to a pH of less than 2, and analyzed.

Ground waters were sampled and analyzed according to a water prospecting plan of the Geological Survey of Ljubljana. Boreholes were made around Krško and Žirovski Vrh for hydrogeological studies connected with the nuclear power plant and the uranium mine, respectively. In addition, a number of boreholes were made in aquifers at different places throughout Slovenia while prospecting for water resources. Altogether in the period from 1972 to 1987 about 500 ground water samples were analyzed.

Though ^{226}Ra was of primary concern, also ^{222}Rn, gross beta activity, and natural uranium were measured. For ^{222}Rn analyses, water samples were filled into 0.6 dm^3 glass flasks, the flask connected to a gas transfer apparatus (Kobal and Kristan 1972), radon transferred into an alpha scintillation cell (Kristan and Kobal 1973), and alpha activity measured after three hours. The detection limit was 50 Bq m^{-3}, the experimental errors about 30% for concentrations of 50 to 200 Bq m^{-3}, and less than 10% for concentrations above 200 Bq m^{-3}.

For ^{226}Ra determination, 20 to 30 dm^3 of water were acidified by concentrated nitric acid to a pH of less than 2 and passed down a cationic exchange resin, contained in a 0.2 dm^3 glass column. The resin was purged with radon-free nitrogen gas and left for 10 to 20 days to allow radon to grown out of the adsorbed radium. After that time the ingrown radon was determined (Kobal et al. 1974). The detection limit was 0.5 Bq m^{-3} and the experimental errors about 30% for concentrations below 10 Bq m^{-3} and about 10% above that value.

For gross beta measurements, 1 dm^3 of a water sample was evaporated, dried, and ignited at 450°C. The ignited residue was mounted on a planchette and counted on a low background anticoincidence Phillips G.M. counter. The detection limit was about 10 Bq m^{-3}, with an experimental error of 30% below 100 Bq m^{-3} and 10% above that concentration. The dry residue in beta measurements was weighed and reported.

Uranium was determined by the fluorimetric method with a K-Na-carbonate-fluoride flux following TBP extraction from 1 dm^3. The detection limit was 0.1 mg m^{-3} and experimental errors were about 25% for concentrations less than 1 mg m^{-3} and about 15% for higher concentrations.

RESULTS AND DISCUSSION

Flags with the appropriate map codes in Fig. 1 indicate places where surface waters were sampled and analyzed while the results obtained are collected in Table 1. The results for ground waters are collected in Table 2. In both tables lower limits of detection were calculated as $LLD = 4.66 \times \sqrt{n_b}$, with n_b as background count (HASL 1983).

Surface waters

Radon concentrations in Slovene surface waters range from 95 to 5370 Bq m^{-3} with an average of 777 ± 958 Bq m^{-3}. Plotted on a log-normal diagram (Aitchison and Brown 1976) in Fig. 2, they show with only a few exceptions only one distribution. These data are comparable to the values obtained for the river Vistula in Poland ranging from 390 to 3210 Bq m^{-3} (Grzybowska et al. 1983), or to surface waters in northern Italy ranging from 65 to 1944 Bq m^{-3} (D'Alessio et al. 1985) or for the Volga River in the Soviet Union ranging from 200 to 2800 Bq m^{-3} (Hristianov and Korchuganov 1971), but they are much higher than concentrations found in rivers around Calcutta, India ranging from 20 to 60 Bq m^{-3} (Mallik et al. 1979).

Radium concentrations in surface waters in Slovenia are very low, usually below 10 Bq m^{-3} and often hardly exceeding the detection limit. Concentrations are distinctly higher (between 10 and 30 Bq m^{-3}) only as a result of phosphate processing (sample site No. 11), or uranium mining and milling (sample site No. 17).

Table 1. Natural radioactivity of Slovene surface waters.

sampling site	*code*	^{222}Rn $Bq\ m^{-3}$	^{226}Ra $Bq\ m^{-3}$	*gross* β $Bq\ m^{-3}$	*nat.U* $mg\ m^{-3}$	*residue* $g\ m^{-3}$
The Sava catchment						
The Sava on Sept. 6, 1975, at :						
Slap Savica (one spring)	SV–1	110	1.1	70	LLD=0.5	80
Outlet of Bohinj Lake	SV–2	100			LLD=0.5	85
Lesce/Bled	SV–3	190	LLD=0.5	45	0.2	140
Zelenci	SV–4	5400		59	0.2	140
Hrušica/Jesenice	SV–5	740	LLD=0.5	60	0.3	145
Posavec/Otoče	SV–6	230	LLD=0.5	65	0.3	150
Breg/Kranj	SV–7	290	LLD=0.5	55	–	150
Medvode, before inflow of the Sora	SV–8	740	LLD=0.5	58	0.3	145
Šentjakob/Ljubljana	SV–9	480	LLD=0.5	41	0.4	155
Sava after inflow of the Ljubljanica	SV–10	790	1.2	215	0.5	420
Zidani most	SV–11	1400	25	96	0.5	240
Krško	SV–12	740	4.8	56	0.6	165
Krško, nuclear power plant	SV–13	530	4.0	16	0.6	240
Brežice	SV–14	300	2.2	52	0.6	170
Jesenice/na Dolenjskem	SV–15	580	2.5	55	0.6	170
The Poljanska Sora on Dec. 8, 1977, at :						
Gorenja vas, above uranium mine	SV/D-1	740	1.9	51	LLD=0.5	135
Poljane, below uranium mine	SV/D-2	850	10	42	0.8	110
The Ljubljanica on Oct. 18, 1974, at:						
Pivka River/Pivka	SV/D-3	3400	1.5			
Pivka, before inflow in Postojna Cave	SV/D-4	870	7.1			
Unica River/Planina	SV/D-5	710	2.6			
Ljubljanica River/Vrhnika	SV/D-6	480	1.9			
Ljubljanica River, above Ljubljana	SV/D-7	1300	LLD=0.5			
Ljubljanica River, before inflow into Sava	SV/D-8	370	LLD=0.5			
The Krka on Sept. 23, 1975, at:						
Spring	SV/D-9	3700	LLD=0.5	52	0.4	195
Krka village	SV/D-10	1600	3.1	81		220
Žužemberk	SV/D-11	590	1.9	78		220
Straža	SV/D-12	520				
Muhaber pod Turško goro	SV/D-13	890	LLD=0.5	85		195
before inflow into Sava	SV/D-14	590	1.2	63	LLD=0.5	210

Table 1 – continued

sampling site	code	^{222}Rn Bq m^{-3}	^{226}Ra Bq m^{-3}	gross β Bq m^{-3}	nat.U mg m^{-3}	residue g m^{-3}
The Kolpa on Sept. 29, 1974, at:						
Sečje/Vinica	SV/D-16	480	3.3			
Radoviči/Rosalnica	SV/D-17	910	4.5			
The Kokra on Nov. 22, 1974, at:						
Jezersko	SV/L-1	410	LLD=0.5			
Preddvor	SV/L-2	1300	1.1			
above Oljarica/Britof	SV/L-3	340	1.7			
below Oljarica/Britof	SV/L-4	300	1.5			
before inflow into Sava	SV/L-5	95	LLD=0.5			
The Savinja on Oct. 3, 1974, at:						
above inflow of Paka River	SV/L-6	150	1.1			
Polzela	SV/L-7	280	1.5			
Megojnica	SV/L-8	200	1.5			
below Celje	SV/L-9	730	LLD=0.5			
below Laško	SV/L-10	440	1.5			
Zidani most, before inflow into Sava	SV/L-11	370	1.5			
The Sotla on Sept. 5, 1974, at:						
Rogatec	SV/L-12	2200	1.9			
Podčetrtek ("Atomic Spa")	SV/L-13	1500	4.1			
The Drava catchment						
The Drava on Sept. 5, 1975, at:						
Dravograd	DR–1	1200	1.5			
Maribor Island	DR–2	300	2.6			
Zgornji Duplek	DR–3	1700	1.9			
Borl	DR–4	3800	2.2			
Središče	DR–5	450	2.2			
The Dravinja on Sept. 3, 1974, at:						
Zreče/above	DR/D-1	450	1.9			
Zreče/below	DR/D-2	340	1.5			
Slovenske Konjice	DR/D-3	200	2.2			
Majšperk	DR/D-4	340	1.5			
Stagovice	DR/D-5	360	1.5			

Table 1 – continued

sampling site	code	^{222}Rn Bq m^{-3}	^{226}Ra Bq m^{-3}	gross β Bq m^{-3}	nat.U mg m^{-3}	residue g m^{-3}
The Mura on Jan. 20, 1986, at:						
Gornja Radgona	DR/L-1		28			
Radenci	DR/L-2		23			
Veržej	DR/L-3		32			
Razkrižje	DR/L-4		19			
The Soča catchment						
The Soča on Nov. 12, 1974, at:						
Spring	SC–1	250	LLD=0.5			
Žaga/Bovec	SC–2	290	1.9			
Idrsko/Kobarid	SC–3	350	1.2			
Tolmin	SC–4	340	1.5			
Plave	SC–5	280	1.1			
Solkan	SC–6	240	1.5			
The Idrijca on Oct. 26, 1974, at:						
Spodnja Idrijca, below mercury mine	SC/L-1	1300	2.3			
Bača/Modrej, above inflow into Soča	SC/L-2	820	1.5			
The Vipava on Oct. 2, 1986, at:						
Spring	SC/L-3		5.9			
Miren	SC/L-4	150	2.3	33	195	
The Hubelj on Oct. 2, 1986, at:						
Spring	SC/L-5		7.1			
Individual rivers						
The Rižana on March 20, 1986, at:						
Spring	RZ–1		8.5			
before inflow into the sea	RZ–2		8.4			
The Dragonja on March 20, 1986, at:						
Spring	DG–1		17			
The Reka on Jan. 26, 1978, at:						
above Škocjan Caves	RK–1	70	LLD=0.5	LLD=0.5		205
Trieste (as Timava River)	RK–2		6.5	92		225

Table 2. Natural radioactivity of Slovene ground waters.

borehole code number	date	^{222}Rn Bq m^{-3}	^{226}Ra Bq m^{-3}	gross β Bq m^{-3}	nat.U mg m^{-3}	residue g m^{-3}
CTZ/VR-1	May 13, 1972	1500	93			
CTZ/VR-2	May 13, 1972	3600	33			
TZH-D/KOV	June 12, 1973	11000	41			
PTUJ-GV	Aug. 9, 1973	7500	49			
PTUJ-V	Aug. 9, 1973		47			
GZL-BC-1	Nov. 2, 1973	11000	8.5			
GZL-BC-2	Nov. 2, 1973		7.4			
GZL-BC-3	Nov. 20, 1973	12000	1.9			
GZL-VC-1	Nov. 2, 1973	8900	17			
GZL-VC-2	Nov. 20, 1973	9600	1.1			
GZL-VC-3	Nov. 20, 1973	9800	1.9			
GZL-B-12	Nov. 20, 1973	11000	7.4			
GZL-B-14	Nov. 2, 1973	12000	11			
GZL-B-15	Nov. 2, 1973		8.2			
GZL-B-18	Nov. 2, 1973		19			
GZL-B-21	Nov. 2, 1973		7.4			
GZL-B-22	Nov. 2, 1973		15			
GZL-V-5	Nov. 20, 1973	23000	3.3			
GZL-V-6	Nov. 20, 1973	8900	2.2			
GZL-V-11	Nov. 20, 1973	7800	13			
GZL-OV-19	Nov. 2, 1973		49			
IJS/NEK	Nov. 2, 1973	9300	3.0			
SM-TPL-V6/73	Nov. 15, 1973	9100	6.3			
CTZ/BREG	Nov. 15, 1973	12000	8.9			
CTZ-V-12	Nov. 15, 1973	9600	30			
LAS-SV	Nov. 23, 1973	12000				
G-3	July 18, 1973	8000	20			
V-3-66	July 18, 1973	370	36			
SC-1-67	July 18, 1973	1300	160			
V-6-67	July 18, 1973	350	420			
V-M	April 25, 1973	450	290			
V-K	April 25, 1973	1900	500			
V-B	April 25, 1973	190	510			
V-A	April 25, 1973	560	190			
VE-1	July 6, 1973	3100	75			
VE-2	July 6, 1973	1900	450			
V-49	July 6, 1973	370	300			
V-1	July 6, 1973	700	210			
V-R	July 6, 1973	12000	170			
V-R	July 6, 1973	810	95			
Z/GZ-1	Feb. 28, 1975	4600	LLD=0.5	82	0.2	165
Z/GZ-2	March 21, 1975	10000	7.4			
Z/GZ-3	March 21, 1975	70000	1.5			
Z/GZ-4	March 21, 1975	14000	15			
Z/GZ-5	March 21, 1975	16000	1.1	26	0.5	180
Z/GZ-6	March 27, 1975	8500	LLD=0.5	37	0.4	195
Z/GZ-7	April 21, 1975	7000	LLD=0.5	52	0.3	200
VRB-6	April 29, 1975		LLD=0.5	81	0.3	210
JA-3	April 29, 1975	9100	1.9	19	0.4	195
JA-4	June 10, 1975	10000	LLD=0.5	44	0.4	205
U-10/75	July 9, 1975	8500	1.5	74	0.5	210

Table 2 – continued

borehole code number	date	^{222}Rn Bq m^{-3}	^{226}Ra Bq m^{-3}	gross β Bq m^{-3}	nat.U mg m^{-3}	residue g m^{-3}
TOPL	Aug. 18, 1975	4400	10	190	0.3	235
ZV	Sept. 19, 1975		2.2			
C-TOP	Sept. 23, 1975	29000		140	0.3	230
VRBINA-3	Nov. 11, 1976	14000	2.2	170	1.5	405
SP-STRGR	Nov. 11, 1976	18000	1.5	70	1.8	250
VX	Nov. 12, 1976	520	69	2000	1.2	3500
VP-2	Nov. 12, 1976	4800	228	2200	0.4	2900
VI	Nov. 12, 1976	LLD=0.05	26	5100	0.4	4000
ZELEZNI	Nov. 12, 1976	27000	30	1700	0.4	1800
RAD-K-1	Nov. 12, 1976	6700	93	3300	0.2	2500
RAD-K-2	Nov. 12, 1976	8700	73	1300	LLD=0.5	850
RAD-K-3	Nov. 12, 1976	21000	58	1300	LLD=0.5	840
CTZ-CR-TPLV	Dec. 8, 1976	14000	43	120	LLD=0.5	215
VP-1	Dec. 10, 1976	9900	140	1100	0.9	1500
V-67	Jan. 21, 1977	6800	74	89	LLD=0.5	100
V-49	Jan. 21, 1977	1100	150	4400	LLD=0.5	5600
V-R	Jan. 21, 1977	2000	48	7900	0.2	5500
VX-2	Jan. 21, 1977	850	96	3200	LLD=0.5	3800
VP-2/2	Jan. 21, 1977	16000	270	2300	0.6	2500
V-O	Jan. 21, 1977	830	93	4900	LLD=0.5	5700
STP-B-1	March 24, 1977	71000	3.7	590	0.3	1200
VP-2/3	April 1, 1977	75000	300	2400	0.2	3500
VX-3	April 1, 1977	750	110	2900	LLD=0.5	3500
VX-4	April 1, 1977	870	100	2800	LLD=0.5	3700
V-72	April 1, 1977	1400	120	3900	0.2	4100
VRBINA-3/2	June 28, 1977	15000	2.5	210		395
LIBNA-STD	Oct. 28, 1977	26000	8.7	96		250
IJS/NEK-2	Oct. 28, 1977		6.9	63		260
SP-STRG-V	Oct. 28, 1977	7200	1.7	56		440
BREST/ZLEB	Sept. 15, 1977	220	23	260	4.2	525
BREST-B-2	Dec. 15, 1977	27000	19	48		355
BREST-B-5	Dec. 15, 1977	12000	10	120		315
BREST-B-7	Dec. 12, 1977	6800	17	84		235
BREST-B-4	Dec. 2, 1977	8700	19	220		865
BREST-B-8	Dec. 12, 1977	6200	2.4	120		365
STRIT-G/STD	Dec. 15, 1977	15000	1.9	120		265
IJS/NEK-3	Dec. 15, 1977	8100	2.4	140		420
STRG-ZAJ	Dec. 15, 1977	13000	2.3	67		280
LIBNA-STD-2	Dec. 15, 1977	38000	26	89		230
KRSK-SRD	Dec. 15, 1977		2.7	27		360
RAD-V-Z2	Aug. 23, 1978	2400	130			
RAD-VX	Aug. 23, 1978	1200	83			
RAD-V-35/MEL	Aug. 23, 1978	23000	43			
PET-V-59	Sept. 7, 1978	1300	155			
LIBNA-STD-3	Oct. 27, 1978	43000	26			
NEK-VR-11	Nov. 22, 1978	5100	2.2			
NEK-VR-2	Dec. 18, 1978	5900	LLD=0.5			
NEK-VR-3	Dec. 18, 1978	9100	2.1			
NEK-VR-10	Dec. 18, 1978	7200	1.3			
VRBINA-3/3	Feb. 7, 1979	9800	1.3			

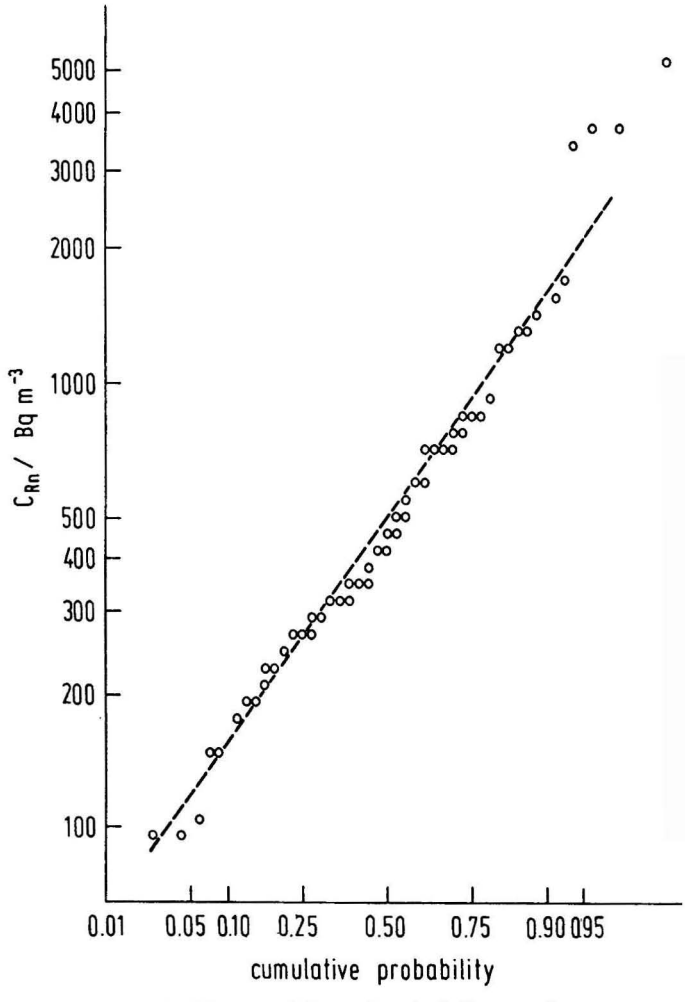

Fig. 2. Log-normal diagram for radon in Slovene surface waters.

Higher values are also found for the Mura River (sample site No. 56 to 59) flowing through a territory in Austria and Yugoslavia abundant in thermal and mineral waters. No increase of radium concentration due to effluents from a coal-fired plant was observed. If the results are plotted on a log-normal diagram as shown in Fig. 3, two different population groups could be distinguished, one for the lower and another for the higher concentration range; the first is 1.66 ± 0.37 Bq m^{-3} ranging from 0.5 to 1.2 Bq m^{-3} representing a natural occurrence of radium, and the second one of 6.2 ± 3.7 Bq m^{-3} ranging from 5 to 30 Bq m^{-3} is elevated by additional sources of radium. Our values are of the same order of magnitude as those obtained in Poland (Grzybowska et al. 1983), Taiwan (Tsai and Weng 1973), Federal Republic of Germany (Wicke and Porstendörfer 1980), and the Soviet Union (Hristanov and Korchuganov 1971), namely 1 to 30, about 20, 0.5 to 20, and 2 to 30 Bq m^{-3}, respectively. But they are lower than those in India -- 20 to 70 Bq m^{-3} (Chatterjee and Banerji 1978), Brazil -- 7 to 220 Bq m^{-3}, and Australia -- 3 to 315 Bq m^{-3} (Iyengar 1984).

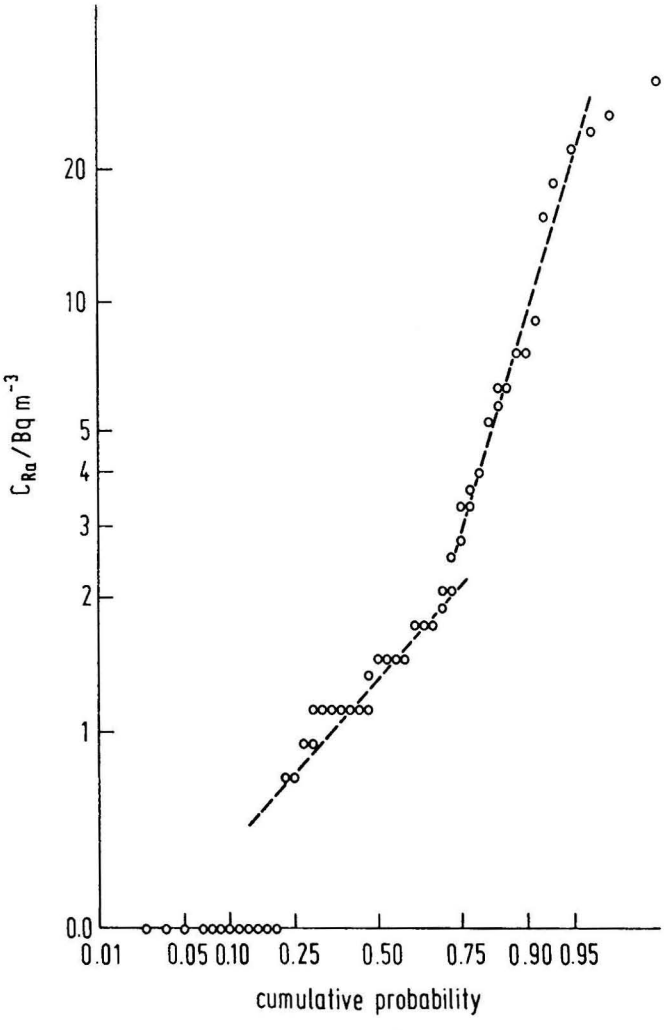

Fig. 3. Log-normal diagram for radium in Slovene surface waters.

The highest value of gross beta activity, 215 Bq m^{-3}, was found in the Sava after the inflow of the Ljubljanica (sampling site No. 10). This is downstream from the inflow of effluents from the TRIGA Mark II 250 kW research reactor and effluents from hospitals in Ljubljana using radioactive isotopes. All values lay between 10 and 210 Bq m^{-3} with an average value of 64 ± 38 Bq m^{-3}.

Concentrations of natural uranium in Slovene surface waters range from 0.1 to 0.8 mg m^{-3}, with an average value of 0.37 ± 0.22 mg m^{-3}. The highest value of 0.8 mg m^{-3} was found in the Poljanska Sora (sampling site No. 17) after the inflow of uranium mine effluents.

The number of gross beta measurements and uranium analyses is too low to justify a statistical elaboration.

Ground waters

As expected, radon concentrations are much higher in ground waters than in surface waters (Table 2). They range from 50 to 75 000 Bq m^{-3}. If plotted on a log-normal paper as in Fig. 4, they could be distin-

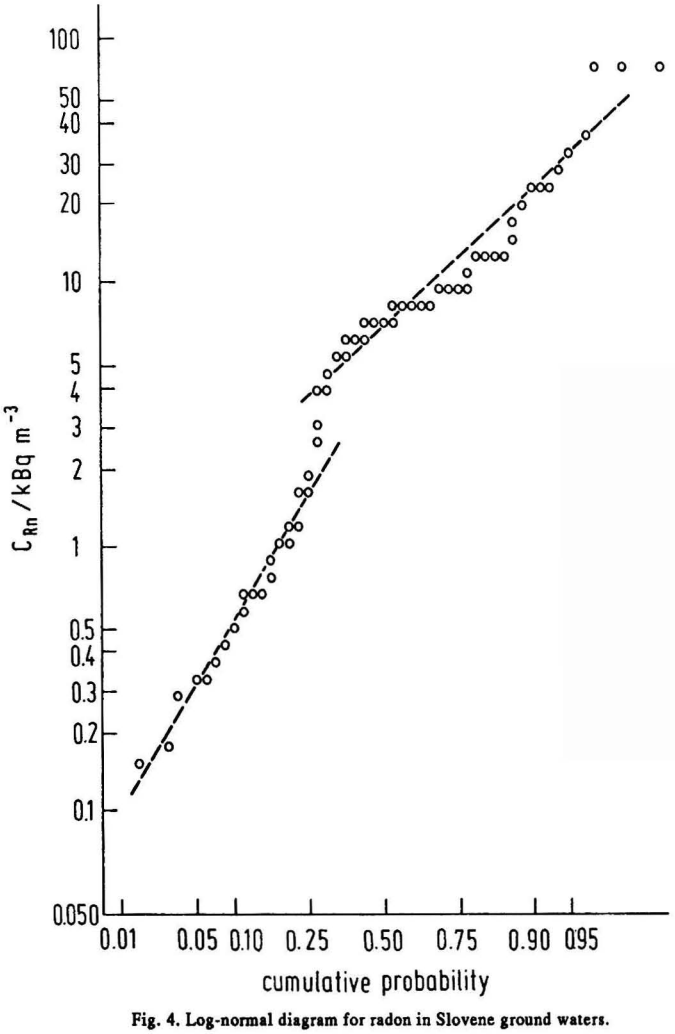

Fig. 4. Log-normal diagram for radon in Slovene ground waters.

guished into two separate groups; one at an average of 12 ± 7 and another at 995 ± 615 Bq m^{-3}.

Around Beograd, the capital of Yugoslavia, an extensive study of radon in surface and well waters was carried out (Kirić 1970). In the majority of these samples radon concentrations range from 800 to 12 000 Bq m^{-3}, while in some samples concentrations between 12 and 120 kBq m^{-3} were found. The values of radon concentrations in Yugoslav ground waters can be compared to those obtained in Greece -- 1 to 15 kBq m^{-3} (Kritidis and Angelou 1984), Japan -- 14.5 ± 12 kBq m^{-3} (Fukui and Katsurayama 1983), and in Poland -- 1.2 to 57.7 kBq m^{-3} (Grzybowska et al 1983), but are much lower than in France -- 5.6 to 712 kBq m^{-3} (Bizollon et al. 1971) or 8 to 3600 kBq m^{-3} (Saumande et al. 1973), Bulgaria -- 12 to 1200 kBq m^{-3} (Karamihajlova and Zhelev 1975 and 1976), New Zeland -- 30 ± 7.6 kBq m^{-3} (Gregory 1976), and Finland with values up to 4.8 MBq m^{-3} for drilled wells and up to 1.7 MBq m^{-3} for dug wells and springs (Asikainen and Kahlos 1979 and 1980).

Depth for the ground waters analysed are not available, and thus we could not look for a correlation

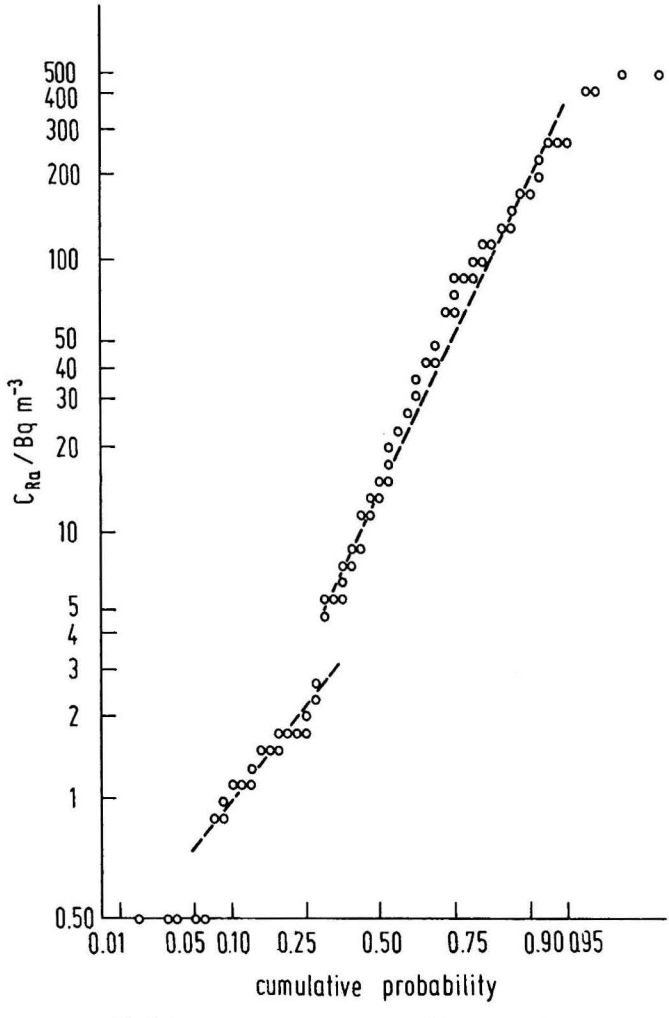

Fig. 5. Log-normal diagram for radium in Slovene ground waters.

between radon concentration and depth (Cech et al. 1987; Fukui 1985).

Radium concentrations in ground waters are plotted in Fig. 5. A slight tendency for the results to fall into two groups could be seen. The values range between 0.5 and 510 Bq m^{-3} with an average value for the first group at 2.1 ± 0.7 and for the second one at 83 ± 98 Bq m^{-3}. The highest values are usually related to mineral and thermal waters in the north-eastern part of Slovenia (Kobal et al. 1979). These waters are extensively exploited not only as tap water but are bottled on a large commercial scale. Radium concentrations in ground waters are thus of major concern to public health inspectors and water installation operators because they can easily exceed the limits allowed for drinking water, and additional measures are needed to lower them.

Our results are comparable to radium concentrations in some ground waters in France -- 4 to 560 Bq m^{-3} (Saumande et al. 1973), and in deep waters in Poland -- 104 ±114 Bq m^{-3} (Grzybowska et al. 1983). They are higher than values in wells in the Federal Republic of Germany -- 2 to 36 Bq m^{-3} (Wicke and Porstendörfer 1980), and in Poland -- 12 to 8 Bq m^{-3} (Grzybowska et

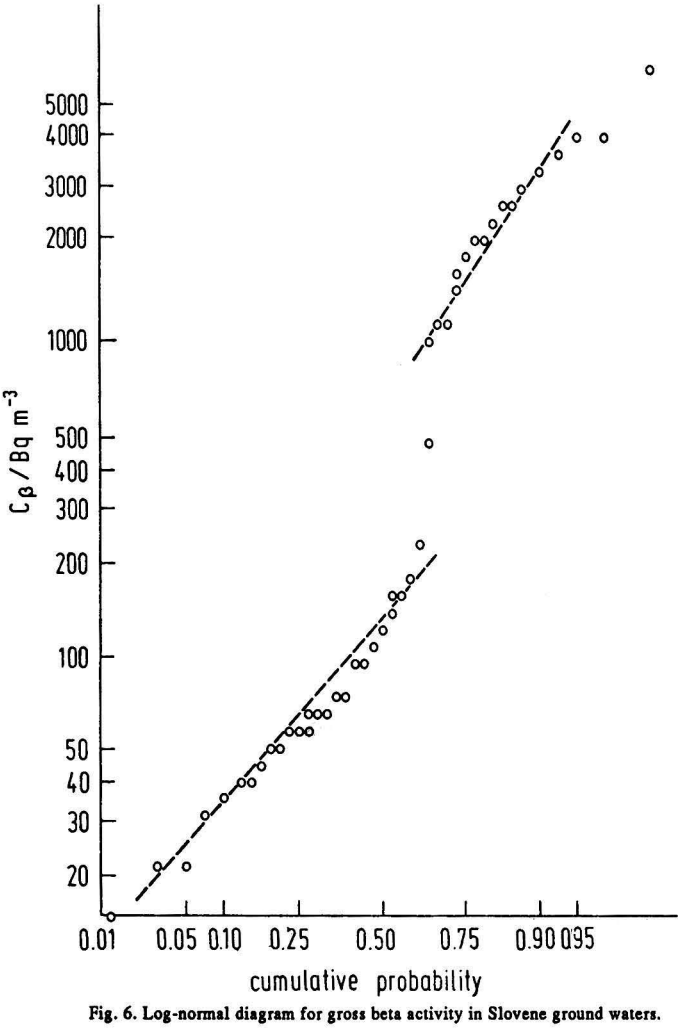

Fig. 6. Log-normal diagram for gross beta activity in Slovene ground waters.

al. 1983), and in drilled wells in Finland -- mean value 16, maximum value 240 Bq m^{-3} (Asikainen and Kahlos 1979). But they are much lower than values in the United States (Iyengar 1984) -- 2 to 1960 Bq m^{-3} (Grants Region), 2 to 55 000 Bq m^{-3} (Geothermal, Western USA), 0 to 2800 Bq m^{-3} (Florida), 55 to 903 Bq m^{-3} (Serasota, Florida), 11 to 6290 Bq m^{-3} (Texas), Brazil -- 0.5 to 3480 Bq m^{-3} (Iyengar 1984), Australia -- 19 to 33 374 Bq m^{-3} (Iyengar 1984), France -- 80 to 4000 Bq m^{-3} (Bizollon et al. 1971) Soviet Union -- 1.4 to 25 kBq m^{-3} (Shchepak 1964) and 200 to 16 000 Bq m^{-3} (Shchepak 1970), and in Finland -- in drilled wells, mean value 320 and maximum value 10 240 Bq m^{-3} (Asikainen and Kahlos 1979).

A log-normal plot of gross beta activities in Slovene ground waters (Fig. 6) shows two distinguishable distribution groups, the lower values (105 ± 61 Bq m^{-3}, ranging from 40 to 800 Bq m^{-3}) originating from ordinary waters, while the higher ones (2800 ± 1265 Bq m^{-3}, ranging from 2 to 5 kBq m^{-3}) arise from thermal and mineral waters. The lower values are close to results obtained for Finnish wells

and springs with a mean value of 96 and a maximum value of 1.2 kBq m^{-3} (Asikainen and Kahlos 1980).

Values for uranium in Slovene ground waters range from 0.1 to 4.2 mg m^{-3} with an average of 0.51 ± 0.75 mg m^{-3}. These values are similar to those in Poland -- 0.22 ±0.17 mg m^{-3} (Grzybowska et al. 1983), but lower than in France -- 0.8 to 24 mg m^{-3} (Saumande et al. 1973), and much lower than in the Soviet Union -- 480 to 970 mg m^{-3} (Shchepak 1970), and in Finland with a mean value of 19.8 and a maximum value of 2100 mg m^{-3} (Asikainen and Kahlos 1980).

CONCLUSIONS

Surface waters in Slovenia were found to contain low radioactivity in comparison with data in the literature for some other countries. Radium concentrations are slightly higher in the close vicinity of the uranium mine and mill, and of the phosphate processing plant, and in the region abundant with thermal and mineral springs and wells.

In ground waters radon and uranium concentrations are low compared to values in other countries, while radium and gross beta activity in numerous wells and boreholes (usually in the region with thermal and mineral water reservoirs) can easily reach or exceed radiological limits for drinking waters.

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CONCENTRATION OF TCDD IN FISH AND THE POTENTIAL FOR HUMAN EXPOSURE

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Levels of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) measured in fish from U.S. lakes and rivers confirm that TCDD is bioaccumulating in fish, and that low-level contamination of fish is widespread. As a result, many state and federal agencies have issued guidelines restricting or banning the consumption of TCDD-contaminated fish. This paper evaluates and interprets data on TCDD levels in fish collected as part of the EPA's National Dioxin Study and estimates the extent of human exposure due to ingestion of TCDD-contaminated fish. Results indicate that mean TCDD concentrations in whole fish and fish fillets from urban areas are low (0.4 and 0.3 pg/g, respectively) and that consumption of TCDD-contaminated fish is not a major pathway of human exposure, accounting for only 5% to 12.5% of total daily intake of TCDD. Assuming that the mean concentration of TCDD in whole fish is 0.4 pg/g, the steady-state bioconcentration factor for TCDD in fish is estimated to be approximately 140 000. The maximum concentration in fish fillets that does not pose an unacceptable health risk to average U.S. fish consumers is 81 pg/g.

INTRODUCTION

Because of its extreme toxicity, much concern and debate has arisen about human exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Levels measured in fish taken from lakes and rivers in the U.S. confirm that TCDD is bioaccumulating in fish and that low-level contamination of fish is widespread (USEPA 1987). Many state and federal agencies have issued guidelines restricting or banning the consumption of TCDD-contaminated fish. The U.S. Food and Drug Administration (FDA), for example, suggests that a concentration of more than 25 picograms TCDD per gram of fish fillet (pg/g) poses an unacceptable risk to humans (increased lifetime cancer risk equals 3×10^{-6}) (cited in USEPA 1987). Additionally, New York and Michigan recommend avoiding fish fillets with a TCDD concentration greater than 10 pg/g, while Canada uses 20 pg/g as the level that poses an unacceptable risk to humans (Ryan et al. 1983).

Despite the fact that dioxin contamination of fish is widespread, the extent and level of dioxin contamination in commercial and sport fish have not been fully evaluated. This paper evaluates and interprets data on TCDD levels in fish collected as part of the U.S. Environmental Protection Agency's (EPA's) National Dioxin Study (USEPA 1987) and estimates the extent of human exposure resulting from ingesting TCDD-contaminated fish.

CONCENTRATION OF TCDD IN FISH

As part of their National Dioxin Study, the EPA (1987) analyzed TCDD concentrations in fish sampled at 304 urban sites located near population centers or areas with known commercial fishing activity, including sites from the Great Lakes region. None of the urban or Great Lakes sites tested had a previously known source of TCDD contamination.

A maximum of four samples was collected per site. Bottom feeders were analyzed first because they were suspected to contain higher levels of TCDD than predator (or game) fish (USEPA 1987). If TCDD contamination was detected in whole (homogenized) bottom feeders, whole predators and fillets from predator and bottom feeding fish were subsequently analyzed (USEPA 1987). Table 1 summarizes these data.

Table 1. Background concentration of TCDD in fish.

Sample	Number of fish	Range (pg/g WW)[b]	Detection limit (pg/g WW)	Geometric mean[a] (pg/g WW)
Fish sampled from background urban sites				
Whole predators	75	ND[c] (59%)[d] - 85	0.3 - 3.0	0.51
Whole bottom feeders	194	ND (68%) - 29	0.2 - 2.7	0.44
Fillets from predators	40	ND (80%) - 12	0.17	
Fillets from bottom feeding fish	51	ND (65%) - 64	0.2 - 2.9	0.27
Fish sampled from the Great Lakes Region				
Whole predators	20	ND (20%) - 23	1.8 - 3.1	3.61
Whole bottom feeders	20	ND (20%) - 24	0.9 - 1.5	4.50
Fillets from predators	19	ND (42%) - 41	0.2 - 1.2	1.58
Fillets from bottom feeding fish	14	ND (23%) - 13	0.2 - 1.3	2.43

[a] Takes into account both detectable and nondetectable concentrations as determined from a log probit analysis.

[b] Picograms of TCDD per gram of fish wet weight.

[c] Nondetectable.

[d] Numbers in parentheses represent the percentage of fish whose TCDD levels were below current detection limits.

Source: US EPA (1987)

Table 1 shows that 59% to 80% of the fish sampled from background urban sites had TCDD concentrations below detection limits. Traditionally, to estimate the mean concentration from such data, investigators assumed that all nondetectable values were equal to zero, equal to half the detection limit, or at the detection limit. Such assumptions result in either an under- or an over-estimate of the true mean of the data set. We propose an alternative approach of estimating the mean by accounting for both detectable and nondetectable values.

We suggest evaluating the data using a log probit analysis (Figs. 1 - 3), which has been shown to be a robust method for estimating the geometric mean of lognormally-distributed samples with values in the nondetectable range (Gilliom and Helsel 1984). In a probit analysis, all measurements (both nondetectable and detectable values) are assumed to be sampled from the same lognormal probability distribution. When samples from a lognormal distribution are plotted on a probit scale, they lie on a straight line. The difficulty of dealing with nondetectable values is determining where to plot them. In a probit analysis, nondetectable values are treated as unknowns, but their percentile values are accounted for. Thus, if there were 100 samples, 30 of which were nondetectable, the first detectable data point would be plotted at the 31st percentile. If sufficient detectable values exist, they can be used to establish the line using linear regression that characterizes the entire data set. The geometric mean concentration of the entire data set (both detectable and nondetectable

values) is determined from the 50th percentile of the distribution. Thus, a probit analysis allows the geometric mean to be extrapolated from detectable values even if it is below detection limits.

Bottom feeders, especially carp and catfish, were expected to accumulate TCDD at higher concentrations than predators due to their high fat content and routine contact with bottom sediments. A t-test of the EPA data (Table 1), however, indicated no significant difference in mean TCDD levels between bottom feeders and predators (P = 0.4). Although variations in sampling protocols as well as fish size and age could account for the lack of statistical significance between TCDD levels in predator and bottom feeding fish, we took the existing data at face value and combined the bottom feeder and predator data for our subsequent statistical analyses.

RESULTS

Urban sites

Fig. 1 presents the cumulative probability distributions of TCDD contamination (pg/g wet weight, WW) in whole fish (n = 269) and fish fillets (n = 91) sampled from urban sites. Concentrations of TCDD in whole fish from urban sites ranged from nondetectable to 85 pg/g (USEPA 1987), with a geometric mean (extrapolated from detectable concentrations using the probit analysis approach) of 0.4 pg/g (Fig. 1). Only about 5% of the whole fish had TCDD concentrations greater than 10 pg/g, while 80% of the whole fish contained less than 2 pg/g TCDD (Fig. 1). Only three (1%) of the whole fish had TCDD levels that exceeded the FDA advisory limit of 25 pg/g.

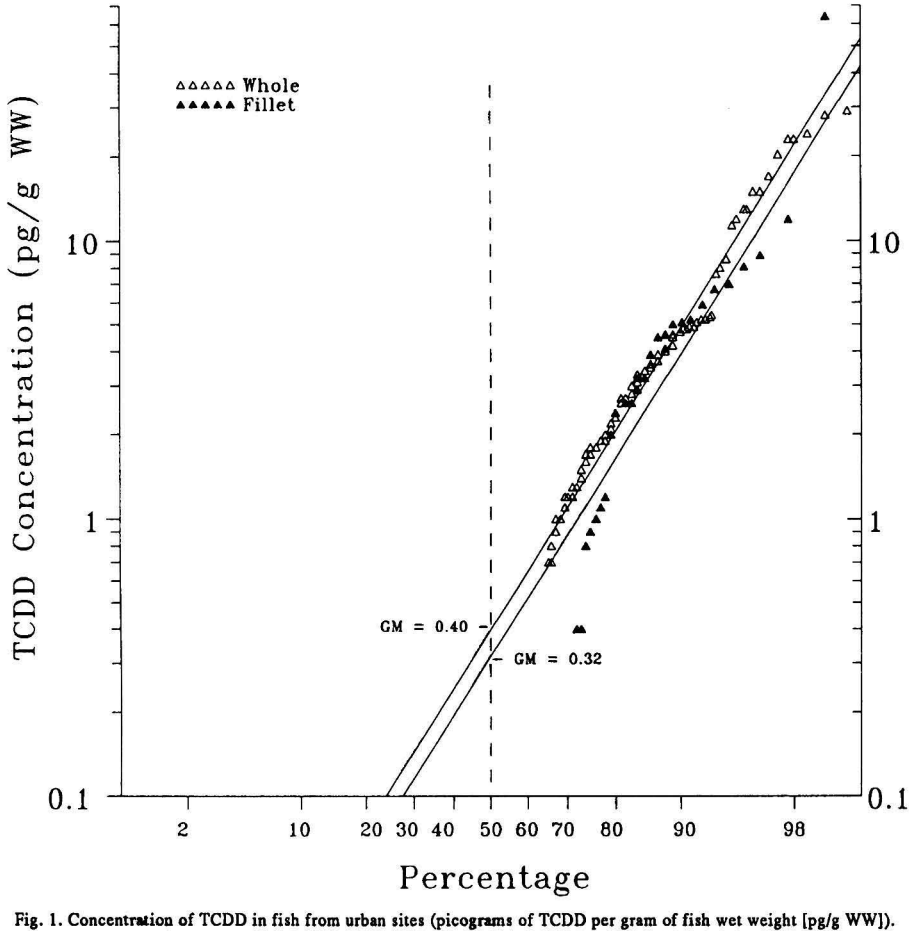

Fig. 1. Concentration of TCDD in fish from urban sites (picograms of TCDD per gram of fish wet weight [pg/g WW]).

Only 29% of the fillets from urban sites had detectable levels of TCDD, with a geometric mean concentration of 0.3 pg/g. Two fish fillets had TCDD concentrations greater than 10 pg/g, and about 80% had TCDD concentrations of ≤1 pg/g.

Ryan et al. (1983) reported that whole fish samples are expected to have 30% to 50% higher TCDD levels than fish fillets (edible portions). The data presented in Table 1 indicate that whole fish (bottom feeders *and* predators) from urban areas contained about 25% more TCDD on the average than fish fillets.

Great Lakes region sites

Fig. 2 shows the cumulative probability distributions of TCDD concentration (pg/g WW) in whole fish (n = 40) and fish fillets (n = 33) from the Great Lakes region. Fish sampled from the Great Lakes region (Fig. 2) had higher TCDD levels than fish from urban areas (Fig. 1). For example, 80% of the whole fish sampled from the Great Lakes region had detectable levels of TCDD, while only 35% of the whole fish from background urban sites had detectable levels of TCDD. TCDD levels in whole fish from the Great Lakes region ranged from nondetectable to 24 pg/g (USEPA 1987), with a geometric mean concentration of 3.8 pg/g (Fig. 1). Hence, TCDD levels in whole fish from the Great Lakes region are about 10 times higher than the concentration in whole fish from urban areas. About 25% of whole Great Lakes fish exceeded the New York and Michigan advisory

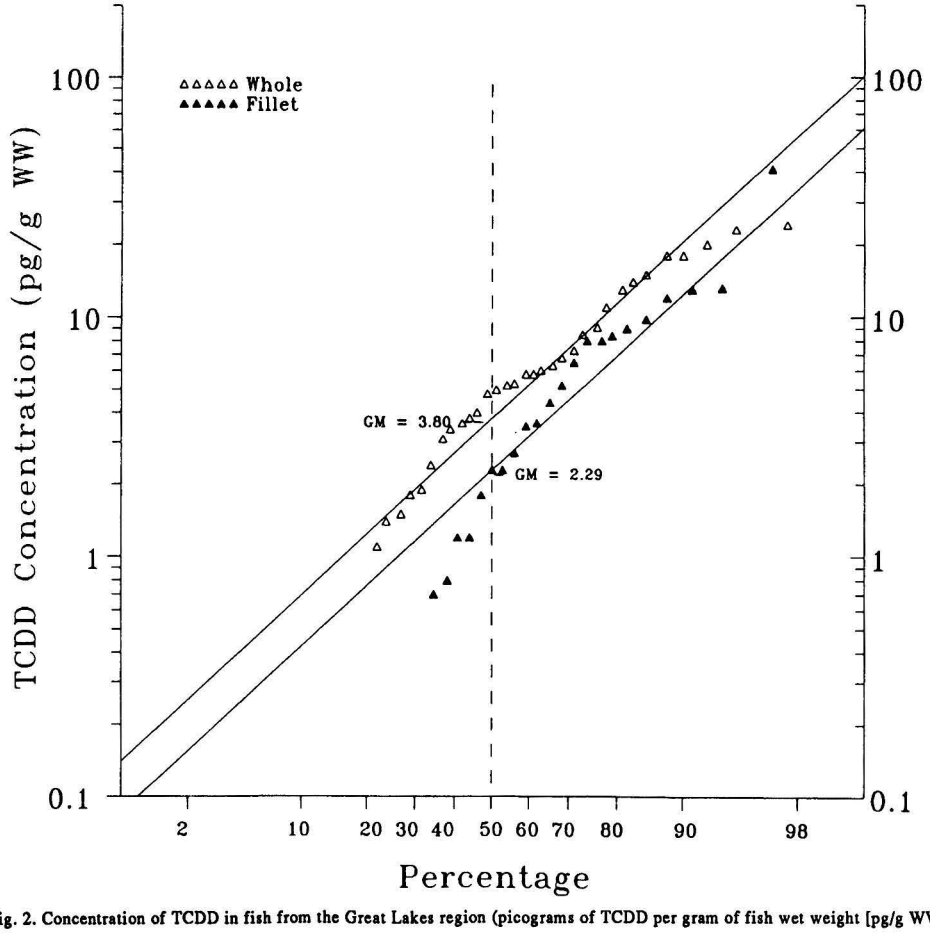

Fig. 2. Concentration of TCDD in fish from the Great Lakes region (picograms of TCDD per gram of fish wet weight [pg/g WW]).

levels of 10 pg/g, and about 8% exceeded the FDA advisory limit.

The mean concentration of TCDD in fish fillets from the Great Lakes region (2.3 pg/g) is about seven times higher than the mean concentration in fillets from urban areas (0.3 pg/g). About 67% of the fish fillets from the Great Lakes region had detectable levels of TCDD, while only 29% of the fillets from background urban sites had detectable levels of TCDD.

Whole bottom feeders and predators from the Great Lakes region accumulated comparable levels of TCDD (Table 1), and whole fish had about 65% more TCDD on the average than fish fillets.

Sites near pulp and paper mills

A previously unsuspected source of TCDD contamination is discharge from pulp and paper mills. Tests conducted at mills using a chlorine bleaching process, however, showed that: (1) TCDD is formed in trace amounts during the bleaching of hard- and soft-wood pulps with chlorine and chlorine derivatives, and (2) the relative amounts of TCDD found in mill discharges varied dramatically from mill to mill (Amendola et al. 1989). The fact that the two whole fish samples with the highest levels of TCDD (29 and 85 pg/g) were taken from sites near two pulp and paper mills (USEPA 1987) contributed to the EPA's initiative to perform a nationwide study of the level of TCDD contamination in fish near these facilities.

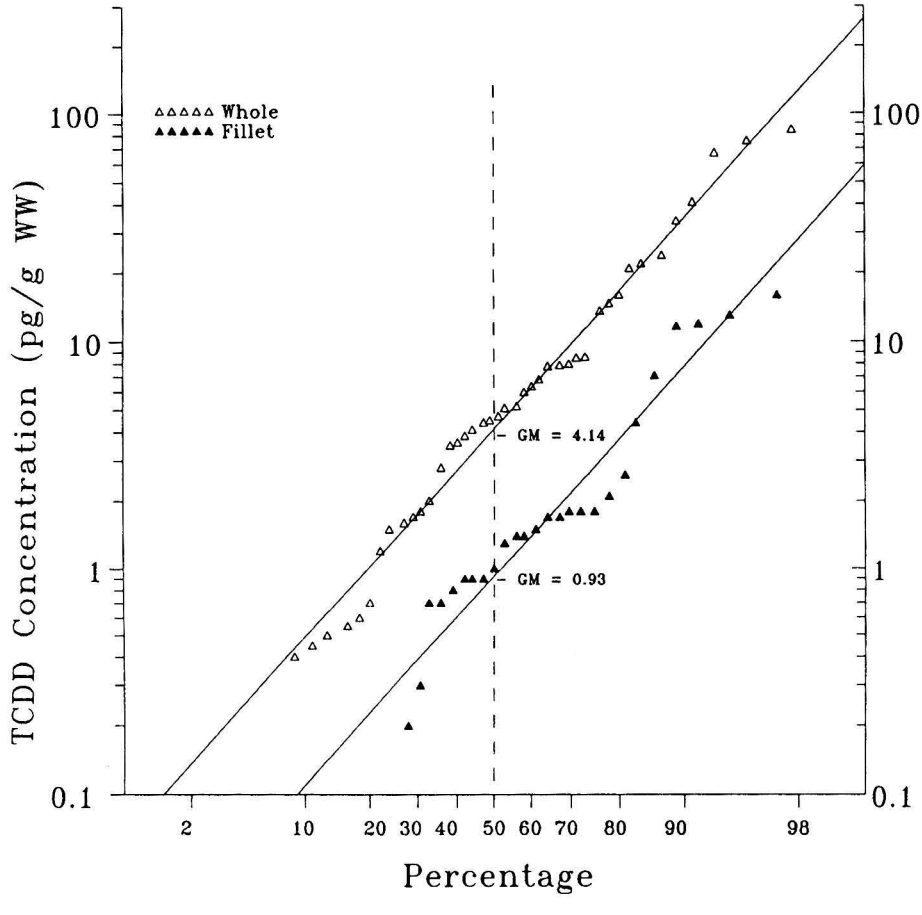

Fig. 3. Concentration of TCDD in fish from sites near pulp and paper mills (picograms of TCDD per gram of fish wet weight [pg/g ww]).

Fig. 3 depicts the cumulative probability distributions of TCDD concentration (pg/g WW) in whole fish (n = 44) and fish fillets (n = 35) sampled from sites near pulp and paper mills (USEPA 1988). TCDD concentrations in whole fish ranged from nondetectable (7%) to 85 pg/g (USEPA 1988). The geometric mean concentration in whole fish (4.1 pg/g) is 10 times higher than the mean concentration of TCDD in whole fish from urban areas, but is comparable to mean levels observed in whole Great Lakes fish.

Concentrations in fish fillets ranged from nondetectable (26%) to 16.1 pg/g, with a geometric mean concentration of 0.9 pg/g, which is three times higher than urban fish fillet concentrations, but about two times lower than the TCDD concentration of fish fillets from the Great Lakes region. The fact that TCDD levels in fish fillets from the Great Lakes region were two times higher than levels in fish fillets taken from sites near pulp and paper mills when TCDD levels in whole fish were comparable is perplexing. Such an inconsistency could be due to physiological factors (e.g., variations in clearance and/or uptake rates) or to discrepancies in study design and implementation (i.e., fish of similar age and species were not sampled at all locations).

Whole fish near pulp and paper mills appear to contain about 400% more TCDD than fish fillets.

BIOACCUMULATION OF TCDD IN FISH

Despite their low water solubility, fish can bioaccumulate chemicals directly from contaminated water and from ingesting contaminated food items (Connolly and Pedersen 1983; Isensee and Jones 1975). When direct analytical measurements are not available, fish contamination levels are typically estimated by multiplying the concentration of a chemical in water times a bioconcentration factor (BCF) for that chemical in fish.

BCFs for TCDD in fish measured in laboratory experiments range from 45 to 63 000 (Adams et al. 1986; Branson et al. 1985; Isensee and Jones 1975; Matsumura and Benezet 1973; Yockim et al. 1978). These BCFs, however, were obtained from short-term studies in which concentrations in fish may not have reached steady-state.

Assuming that the mean background concentration of TCDD in whole fish is 0.4 pg/g (Fig. 1) and that the background concentration of TCDD in surface water is 2.8×10^{-6} pg/g (Travis and Hattemer-Frey 1989), the steady-state BCF for TCDD in fish is estimated to be 140 000. Thus, previous estimates based on short-term laboratory experiments may have underestimated the bioaccumulation potential of TCDD in fish.

CONTRIBUTION OF FISH INGESTION TO TOTAL DAILY INTAKE OF TCDD

Assuming a mean urban concentration of TCDD in fish fillets of 0.3 pg/g (Fig. 1) and an average U.S. fish consumption rate of 6.5 g of freshwater fish per day (USEPA 1987), the general population of the U.S. is exposed to about 2.1 pg of TCDD per day via fish consumption. This intake level corresponds to a lifetime cancer risk of 5×10^{-6} using a cancer potency factor of 1.56×10^{5} for TCDD (USEPA 1985).

Travis and Hattemer-Frey (1989) estimated that the average, long-term daily intake of TCDD by the general population of the U.S. is 40 pg/day and that the food chain, especially milk and meat products, accounts for 99% of human exposure to TCDD. Consumption of contaminated fish and shellfish was estimated to contribute only 7% of the total background human exposure to TCDD (Travis and Hattemer-Frey 1989). This present study, based on actual measurements of TCDD concentrations in fish, indicates that fish ingestion probably accounts for 5% (for average consumers) to 12.5% (for heavy fish eaters) of total daily intake for most Americans.

Table 2 shows the percent of background daily intake resulting from ingestion of fish fillets contaminated at various levels. Average fish consumers who eat fish contaminated at the FDA limit are taking in four times more TCDD than the mean background daily intake, while heavy fish eaters are taking in almost 10 times the background intake level. Average fish consumers who eat fish fillets from the Great Lakes region are taking in about one-third of the background daily intake of TCDD from ingesting contaminated fish, while heavy fish eaters are taking in slightly less than the average background daily intake.

ACCEPTABLE CONCENTRATION OF TCDD IN FISH

An important objective in evaluating TCDD levels in fish is estimating the concentration in fish that will not pose an unacceptable risk to humans. Although U.S. regulatory agencies assume that there is some finite risk associated with human exposure to all carcinogens, several other governments, including Canada (OME 1985), Germany (FHO 1985), and Holland (van der Heijden et al. 1982), assume that some chemical carcinogens (including TCDD) may not cause deleterious health effects until a specific, critical

Table 2. Contribution of fish fillet ingestion to total daily intake of TCDD.

	Average TCDD intake[a] (pg/day)[b]	Percent of background exposure to TCDD[d]	Maximum daily intake[c] (pg/day)	Percent of background exposure to TCDD
Background urban	2.1	5%	5.0	12.5%
Great Lakes	14.9	37%	36.0	90%
Pulp and paper mills	6.0	15%	14.6	36.5%
EPA advisory level (0.7 pg/g)	4.6	11.5%	11.0	27.5%
FDA advisory level (25 pg/g)	162.5	406%	392.5	981%

[a] Assumes a U.S. mean fish consumption rate of 6.5 g of freshwater fish fillets per day (US EPA, 1987).

[b] Picograms per day.

[c] Assumes an upper 90% percentile consumption rate of 15.7 g of freshwater fish fillets per day for consumers in the Great Lakes Region (US EPA, 1987).

[d] Assumes a background total daily intake of 40 picograms of TCDD per day (Travis and Hattemer-Frey, 1989).

dose (the threshold dose) is surpassed. Thus, there are two approaches for solving this problem: (1) base maximum limits on a nonthreshold cancer potency model (i.e., the EPA and FDA approach), or (2) assume that TCDD has a biological threshold for effects (i.e., the Canadian and European approach). The FDA/EPA approach was discussed previously. We shall briefly discuss the Canadian/European approach. We also note that current knowledge concerning the mechanisms of TCDD action is insufficient to select between these two approaches. World opinion, however, seems to favor the threshold approach.

The threshold approach suggests that human exposure to TCDD should not exceed 10 pg/kg-day. This exposure level is based on the No Observed Adverse Effect Level (NOAEL) observed in animal populations with a safety factor of 100 applied for extrapolation to humans. Thus, a daily dose of 700 pg/d (for individuals weighing 70 kg) is the highest value estimated to be compatible with human health. This intake level is based on the assumption that TCDD has a threshold of action below which adverse human health effects will not be observed.

The Canadian government (Federal-Ontario 1988) suggests that human exposure to TCDD should not exceed 10 pg/kg-day, of which 80% (560 pg/day) comes from ingesting contaminated food items. Travis and Hattemer-Frey (1989) estimated that the long-term, average background daily intake of TCDD by the general population of the U.S. from all exposure pathways other than fish ingestion is 35 pg/day. Hence, individuals consuming TCDD-contaminated fish could ingest 525 pg of TCDD per day (the maximum allowable intake from food [560 pg/d] minus the estimated intake from all food items except fish [35 pg/d]) without exceeding the threshold limit.

To experience this level of exposure, average fish eaters (i.e., individuals who consume 6.5 grams of freshwater fish fillet per day) would have to continuously consume fish fillets with a TCDD concentration greater than 81 pg/g (525 pg/d ÷ 6.5 g/day), which is virtually impossible, since this concentration exceeds the 99th percentile of TCDD concentra-

tion in fish fillets (Figs. 1-3). Heavy fish eaters (i.e., individuals who consume 15.7 g of freshwater fish fillet per day) would have to continuously consume fish fillets with a TCDD concentration greater than 33 pg/g; again, this is extremely unlikely, since this concentration exceeds the 95th percentile concentration (Figs. 1-3).

The above analysis indicates that the consumption of fish contaminated with TCDD at background levels does not currently pose a health risk problem in the United States. Although fish taken from locations known to have been previously contaminated may contain much higher levels of TCDD, current advisory levels that suggest limiting the daily consumption of fish contaminated with TCDD at levels greater than 25 pg/g seem reasonable and protective of human health.

CONCLUSIONS

Although the data on TCDD levels in fish are limited, some preliminary conclusions concerning TCDD contamination of fish can be drawn:

- Predators and bottom feeders appear to accumulate comparable levels of TCDD (Table 1).
- Mean levels of TCDD in whole fish and fish fillets from urban areas are low (0.4 and 0.3 pg/g WW, respectively) (Fig. 1).
- Although mean TCDD levels in whole fish from urban areas are only slightly higher than concentrations in fish fillets, whole fish from the Great Lakes region and near pulp and paper mills have two to four times higher TCDD levels, respectively, than fish fillets from those areas.
- Mean TCDD concentrations in whole fish near pulp and paper mills (4.1 pg/g) are about 10 times higher than levels found in fish from urban areas but are comparable to TCDD levels in whole fish from the Great Lakes region.
- The steady-state BCF for TCDD in fish appears to be on the order of 140 000.
- Fish consumption is not a major pathway of TCDD exposure for most Americans, accounting for only 5% to 12.5% of background total daily intake (Table 2).
- Assuming a 10 pg/kg-day threshold level for TCDD, the maximum concentration in fish that does not pose an unacceptable risk to average fish consumers is 81 pg/g.

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CESIUM-137 ACCUMULATION IN HIGHER PLANTS BEFORE AND AFTER CHERNOBYL

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Cesium-137 concentrations in plant species of three biotopes of northern Greece, differing in location as well as in vegetation, are reported following the Chernobyl reactor accident. The cesium uptake by plants was due to the foliar deposition rather than the root uptake. The highest level of cesium in plants was found in *Ranunculus sardous*, a pubescent plant. The ^{137}Cs concentration was about 22 kBq kg^{-1}d.w. A high level of cesium was also found in *Salix alba* (^{137}Cs: 19.6 kBq kg^{-1}d.w.), a deciduous tree showing that hairy leaves or leaves having rough and large surfaces can absorb greater amounts of radioactivity (surface effect). A comparison is also made between the results of measurements of the present study and the results of measurements of some herbarium plants collected one year before the accident as well as the results of measurements of some new plants grown and collected one year after the accident resulting in a natural removal rate of ^{137}Cs in plants varying from 14 to 130 days.

INTRODUCTION

Of the fission product radionuclides emitted into the biosphere during the nuclear accident occurring at Chernobyl, USSR, on 26 April 1986, ^{137}Cs is a major contributor to environmental contamination after the short half-life radionuclides have decayed.

Heavy rain in Greece during May 1986 led to high level concentrations of the fission product radionuclides in the environment, e.g., ^{137}Cs inventories on the ground of approximately 24 kBq m^{-2} (Papastefanou et al. 1988).

So, radioactive particles were deposited by wet deposition rather than by dry deposition onto the plants and ground. Following deposition on the ground, the radionuclide uptake by plants took place directly into above ground parts, particularly by the leaves (Sawidis 1988). Coughtrey and Thorne (1983) described mechanisms for the active translocation and the absorption of radionuclides through the leaves. However, plants also absorb radionuclides from soil through the root system. Under field conditions, it is difficult to illustrate differences between these mechanisms of foliar or root uptake (Russell 1965).

After the decay of the short-lived nuclides, ecologists turned their attention to the contamination of ecosystems by long-lived nuclides, such as ^{137}Cs, which is one of the critical radionuclides studied in the environment because of its long half-life (30.14 y) and its ability to be assimilated into plants (Devell et al. 1986; Bondietti and Brantley 1986; Andrási 1987; Ballestra et al. 1987; Gopalakrishnan and Mishra 1987; Aarkrog 1988; Jaworowski and Kownacka 1988; Joshi 1988; Steinhäusler et al. 1988).

The present study deals with the distribution of ^{137}Cs in various plant species collected from three biotopes of northern Greece differing in location as well as in vegetation. Furthermore, a comparison is made between the measurements of the present study and the measurements of some herbarium plants collected one year before the accident as well as also the measurements of some new plants grown and collected one year after the accident.

MATERIALS AND METHODS

Samples of various plant species were collected immediately after the Chernobyl accident, from 4 May to 25 July 1986. Three main sampling locations of a total area ca 5 km^2 were selected in northern Greece (Fig. 1) in order to compare the radioactivity of various plants under similar contamination conditions. Some other samples of plant species were collected from two of the three above-mentioned locations in summer 1987, one year after the accident. Flora Europaeae (Tutin et al. 1980) was used for the nomenclature of the specific plant names.

All the plant samples were counted using a LKB gamma counter in the Preclinic of the University of Graz, Austria. Each sample consisted of a few grams of air-dried material in a small plastic test tube. Measurements were also made in July 1987 after the almost complete decay of the short-lived nuclides, such as ^{131}I, ^{132}Te, ^{132}I, ^{140}Ba, ^{140}La, etc., as only the long-lived nuclide ^{137}Cs was considered in our study. We could not reconstruct the initial radioactivity on the basis of the physical half-life because there are significant differences between the biological (T_b = 14 d) (Hoffman and Baes 1979; Devell et al. 1986) and the physical half-life of ^{137}Cs (T_d = 10.14 y), and transport phenomena could have resulted in an increase or decrease of radioactivity.

SAMPLING SITES

Mt. Paikon (biotope 1, Fig. 1) has an altitude of 1650 m, many summits, and an even shape. The dominant vegetation consists of the deciduous forests of the Class *Querco-Fagetea* and the pasture communities of the Class *Daphno-Festucetea* (Horvat el al. 1974; Oberdorfer 1979).

The economic importance of the vegetation of Mt. Paikon is primarily related to stock breeding, and most of the species collected from this location are eaten by sheep, goats, and cows. It also has importance for other types of agriculture, notably for the Mt. Paikon potato (*Solanum tuberosum*) which is cultivated in big areas.

The altitude of biotope 2 at Chalkidiki Peninsula, Fig. 1, is less than 10 m. The main vegetation is the

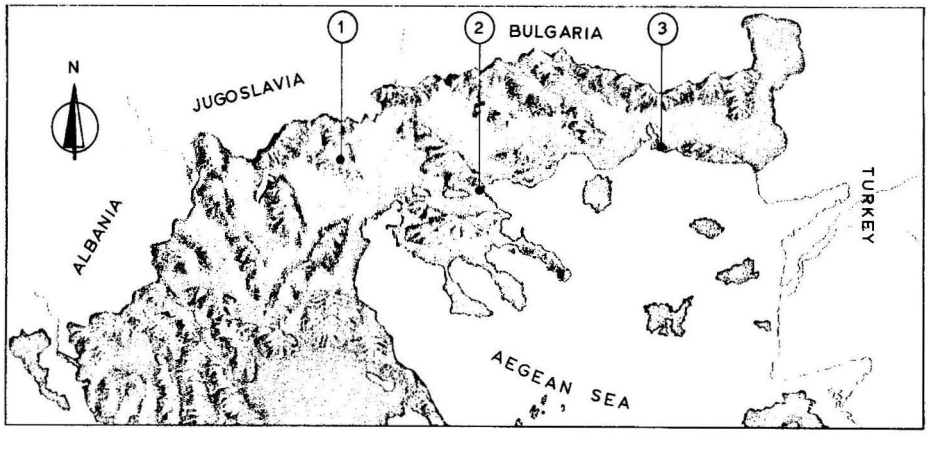

Fig. 1. A map of northern Greece showing the locations of the three biotopes: 1) Mt. Paikon (biotope 1); 2) Chalkidiki peninsula (biotope 2); and 3) coast of Komotini (biotope 3).

evergreen sclerophyl, named Macchie, characteristic of Mediterranean areas, and is interspersed with meadows. This biotope is also of importance for stock breeding and tourism.

The altitude of biotope 3 at the coast of Komotini, Fig. 1, ranges between 0 to 10 m and seems to be almost flat. The most dominant plant communities are the aquatic and helobiae (*Lamnetea, Potametea,* and *Phragmitetea*), the halophilous (*Arthrocnemetea*), and the ammonitrophilous (*Cakiletea* and *Ammopholetea*) (Horvat et al. 1974, Oberdorfer 1979). Trees are completely absent. This biotope is of importance at the international as well at the national level, because it includes a large range of wet biotopes of fresh, saline, and brackish water, with many animal and plant species. About 76 hydrobic species of birds occur in the area, which is under international protection according to the Ramsar agreement. This agreement was signed at the convention on wetlands of international importance, especially waterfowl habitat,at Ramsar, Iran, on 2 February 1971 by the United Nations Educational, Scientific, and Cultural Organization, Office of International Standards and Legal Affairs.

RESULTS AND DISCUSSION

The ^{137}Cs concentrations of the plant species collected from biotope 1 on 24 to 25 July 1986 are shown in Table 1a. For comparison, pre-Chernobyl (6 and 13 July) data for the same biotope are presented in Table 1b.

The plant specimens of biotope 2 were collected on 4 May 1986 just after the nuclear accident and their ^{137}Cs concentrations are shown in Table 2a, while the ^{137}Cs concentrations of plants of the same biotope collected on 10 August 1987 are shown in Table 2b.

The ^{137}Cs concentrations of the plant species of biotope 3 collected between 9 May and 21 July 1986 are shown in Table 3a, while the radioactivity of plants of the same biotope collected on 17 August 1987 is shown in Table 3b.

In biotope 3, a flat coastal area, in the ^{137}Cs concentration in 29 items collected on 9 to 10 May, the radioactivity of the plant species varied between 2816 Bq kg^{-1}d.w. and 22.8 kBq kg^{-1}d.w. (average 9462 Bq kg^{-1}d.w.). The high cesium content of plants must have resulted from foliar deposition of this nuclide. In this case, the structure and the external morphology of the leaves of plants play a very important role in the interception of the radioactive elements (Ludwieg 1962; Heinrich 1987). Hairy leaves or leaves having rough and large surfaces can absorb

Table 1a. Cesium-137 concentrations in plants collected from Mt. Paikon (Biotope 1) after the Chernobyl accident (24-25 July 1986).

Species	^{137}Cs Bq kg^{-1}
Achillea grandifolia Friv.	118.4
Achillea millefolium group	Un
Alisma plantago-aquatica L.	536.0
Anthemis tinctoria L.	1147.0
Atropa belladonna L.	325.6
Brachypodium sylvaticum (Hudson) Beauv.	Un
Calystegia sylvatica (Kit.) Griseb.	121.1
Campanula trachelium L.	525.4
Epilobium angustifolium L.	1073.0
Eryngium creticum Lam.	647.5
Euphorbia myrsinites L.	2012.3
Filipendula ulmaria (L.) Maxim.	747.4
Gentiana cruciata L.	Un
Hieracium murorum group	2068.3
Hippocrepis glauca Ten.	1550.3
Hypericum olympicum L.	518.0
Inula britannica L.	166.5
Juncus effusus L.	503.2
Lamiastrum galeobdolon (L.) Ehrend. et Polatschek	536.5
Leontodon crispus Vill.	1006.4
Lathyrus niger (L.) Bernh.	3185.7
Medicago falcata L.	344.1
Mentha longifolia (L.) Hudson	395.9
Origanum vulgare L.	555.0
Salix cinerea L.	536.0
Sideritis scardica Griseb.	1339.4
Smyrnium perfoliatum L.	825.1
Sonchus palustris L.	Un
Stachys germanica group	954.6
Stachys plumosa Griseb.	144.3
Teucrium montanum L.	3740.7
Teucrium polium L.	2749.1
Thymus praecox Opiz	3278.2
Trifolium alpestre L.	684.5
Trifolium medium L.	2237.7
Verbascum phlomoides L.	4500.0
Veronica anagallis-aquatica L.	377.4
Veronica beccabunga L.	917.2
Vicia cassubica L.	584.6
Vicia cracca L.	266.4
Viola reichenbachiana Jordan ex Boreau	196.1
Viola tricolor L.	580.9
Vitis sylvestris C.C. Gmellin	162.8

Un = undetectable

S = ± 0.5 %

Table 1b. Cesium-137 concentrations in plants collected from Mt. Paikon (Biotope 1) before the Chernobyl accident (6 and 13 July 1985).

Species	^{137}Cs Bq kg^{-1}
Acer platanoides L.	Un
Amelanchier ovalis Medicus	51.8
Angelica sylvestris L.	103.6
Astragalus glycyphyllos L.	Un
Atropa belladonna L.	214.6
Calamagrostis epigeios (L.) Roth	Un
Calamintha grandiflora (L.) Moench	Un
Cotoneaster nebrodensis (Guss.) C.Koch	51.8
Digitalis viridiflora Lindley	144.0
Dorycnium pentaphlyllum Scop.	Un
Epilobium angustifolium L.	Un
Gentiana cruciata L.	Un
Gnaphalium sylvaticum L.	70.3
Hieracium pannosum group	Un
Hypericum perforatum L.	Un
Inula oculus-christi L.	51.8
Lysimachia punctata L.	59.2
Mentha longifolia (L.) Hudson	214.6
Origanum vulgare L.	Un
Ostrya carpinifolia Scop.	62.9
Physospermum cornubiense (L.) DC.	Un
Populus tremula L.	Un
Potentilla argentea L.	Un
Prenanthes purpurea L.	Un
Rhamus alaternus L.	51.8
Salix caprea L.	70.3
Scrophularia nodosa L.	Un
Sideritis scardica Griseb.	133.2
Silene vulgaris (Moench) Garcke	Un
Trifolium medium L.	Un
Trifolium pratense L.	Un
Vaccinium myrtillus L.	151.7

Un = undetectable
S = ± 0.5 %

Table 2a. Cesium-137 concentrations in plants collected early from Chaldidiki Peninsula (Biotope 2) after the Chernobyl accident (4 May 1986).

Species	^{137}Cs Bq kg^{-1}
Anthemis cotula L.	525.4
Chrysanthemum segetum L.	1232.1
Cistus incanus L.	725.2
Cistus salvifolius L.	728.9
Euphorbia cyparissias L.	3126.6
Hypericum montbretii Spach	1924.0
Silene dichotoma Ehrh.	1228.4
Silene italica (L.) Pers.	1243.2
Trifolium hirtum All.	913.9
Vicia sativa L.	1935.1

S = ± 0.5 %

Table 2b. Cesium-137 concentrations in plants collected from Chalkidiki Peninsula (Biotope 2) one year after the Chernobyl accident (10 August 1987).

Species	^{137}Cs Bq kg^{-1}
Hordeum vulgare L.	18.5
Lavandula stoechas L.	373.7
Lotus uliginosus Schkuhr	Un
Minuartia verna (L.) Hiern	93.5
Olea europaea L.	85.1
Pinus halepensis Miller	318.2
Piptatherum miliaceum (L.) Cosson	Un
Silene dichotoma Ehrh.	75.8
Silene italica (L.) Pers.	80.5
Tragopogon porrifolius L.	Un
Trifolium pratense L.	2.1
Triticum aestivum L.	55.5
Vicia cracca L.	Un

Un = undetectable
S = ± 0.5 %

greater amounts of radioactivity (Sawidis 1988). For this reason, the pubescent plant *Ranunculus sardous* shows the highest level of ^{137}Cs (22.8 kBq kg^{-1}d.w.) as well as the deciduous tree *Salix alba* (^{137}Cs: 19.6 kBq kg^{-1}d.w.)) because of the long appraised silky hairs existing on both surfaces of the leaves. The high value of ^{137}Cs (18 kBq kg^{-1}d.w.) in *Trifolium michelianum* is probably the result of the large surface area caused by many deflexed flowers and pubescent seeds. Later, on 21 June 1986 and 12 July 1986, separate samplings of plant species (6 to 10 items each) in the same biotope showed that the ^{137}Cs concentration in plants significantly declined averaging 1887 Bq kg^{-1}d.w. and 1482 Bq kg^{-1}d.w., respectively. The cesium content of plants in that case was due to the root uptake rather than the foliar deposition. Besides, a large part of the amount of cesium had been released by weather effects, particularly by rain washout, resulting in a decrease of foliar deposition (Haunold et al. 1988).

Table 3a. Cesium-137 concentrations in plants collected early and late from the coast of Komotini (Biotope 3) (9 May to 12 July 1986).

Species	Sampling Date	^{137}Cs Bq kg^{-1}
Alyssum minus (L.) Rothm.	9.5.1986	10722.6
Astragalus hamosus L.	"	10903.9
Dactylis glomerata L.	"	9039.1
Gladiolus italicus Miller	"	8062.3
Hordeum bulbosum L.	"	8972.5
Jasminum fruticans L.	"	6093.9
Lolium perenne L.	"	5694.3
Medicago litoralis Rohde ex Loisel.	"	4739.7
Medicago marina L.	"	8698.7
Pyrus amygdaliformis Vill.	"	9679.2
Rhazya orientalis (Decne) A.DC. in DC.	"	14781.5
Scirpus maritimus L.	"	6626.7
Sinapis arvensis L.	"	13934.2
Trifolium michelianum Savi	"	18059.7
Vicia sativa L.	"	6652.6
Anthoxanthum odoratum L.	10.5.1986	10012.2
Avena sterilis L.	"	5849.7
Convolvulus cantabrica L.	"	9087.2
Holcus lanatus L.	"	8495.2
Iris pseudacorus L.	"	5291.0
Lupinus graecus Boiss. et Spruner	"	10652.3
Nymphaea alba L.	"	2815.7
Orobanche crenata Forskal	"	4070.0
Ranunculus sardous Grantz	"	22800.0
Salix alba L.	"	19587.8
Sideritis romana L.	"	11962.1
Silene conica L.	"	11662.4
Thymus comptus Friv.	"	5668.4
Typha angustifolia L.	"	3840.6
Alnus glutinosa (L.) Gaertner	21.6.1986	1283.9
Hyoscyamus niger L.	"	558.7
Pistacia terebinthus L.	"	906.5
Salix fragilis L.	21.6.1986	1409.7
Scrophularia umbrosa Dumort.	"	832.5
Verbascum phlomoides L.	"	6334.4
Agrostis stolonifera L.	12.7.1986	Un
Althaea officinalis L.	"	325.6
Aristolochia pallida Willd.	"	Un
Epilobium hirsutum L.	"	55.5
Lythrum salicaria L.	"	118.4
Marrubium peregrinum L.	"	199.8
Potamogeton nodosus Poiret in Lam.	"	370.0
Stachys germanica L.	"	3059.9
Thalictrum lucidum L.	"	207.2
Verbascum haussknechtii Heldr. ex Haussk.	"	8806.0

Un = undetectable
S = ± 0.5 %

Table 3b. Cesium-137 concentrations in plants collected from the coast of Komotini (Biotope 3) one year after the Chernobyl accident (17 August 1987).

Species	^{137}Cs Bq kg^{-1}
Calycotome villosa (Poiret) Link	122.1
Cistus incanus L.	Un
Convolvulus arvensis L.	Un
Delphinium consolida L.	Un
Elymus sabulosus Bieb.	Un
Epilobium hirsutum L.	Un
Frankenia pulverulenta L.	Un
Galilea mucronata (L.) Parl	Un
Inula britannica L.	Un
Lotus aegaeus (Griseb.) Boiss.	Un
Medicago litoralis Rohde ex Loisel.	Un
Medicago marina L.	62.9
Onobrychis caput-galli (L.) Lam.	Un
Ononis pubescens L.	262.7
Salvia pratensis L.	Un
Silene conica L.	Un
Stachys palustris L.	Un
Teucrium polium L.	Un
Thymus comptus Friv.	66.6
Trifolium spumosum L.	Un

Un = undetectable
S = ± 0.5 %

A year after, we repeated the collection and measurement of the radioactivity of plant species at biotope 3. Out of 20 items collected, the ^{137}Cs was undetectable in 16 items; that means the low detection limit was about 1 Bq kg^{-1} while in the remaining 4 items the ^{137}Cs ranged from 63 Bq kg^{-1}d.w. to 263 Bq kg^{-1}d.w. (average 129 Bq kg^{-1}d.w.). The average cesium concentration of the plants collected at biotope 3 versus time passed since the time of the accident is illustrated in Fig. 2. This figure shows an exponential decrease of the cesium content of plants with time. The curve of Fig. 2 consists of three components. The first component results in a natural removal rate of ^{137}Cs in plants during May 1986 of about 14 days and is in agreement with the literature (Devell et al. 1986). The second component for the next two-month period results in a removal rate of ^{137}Cs of about 60 days. The third component corresponding to the period of August 1986 to August 1987 results in a removal rate of about 130 days. The physical half-life of ^{137}Cs is 30.14 y.

In biotope 2, a hilly area, the ^{137}Cs concentration in 10 items of the early collected (4 May 1986) plant

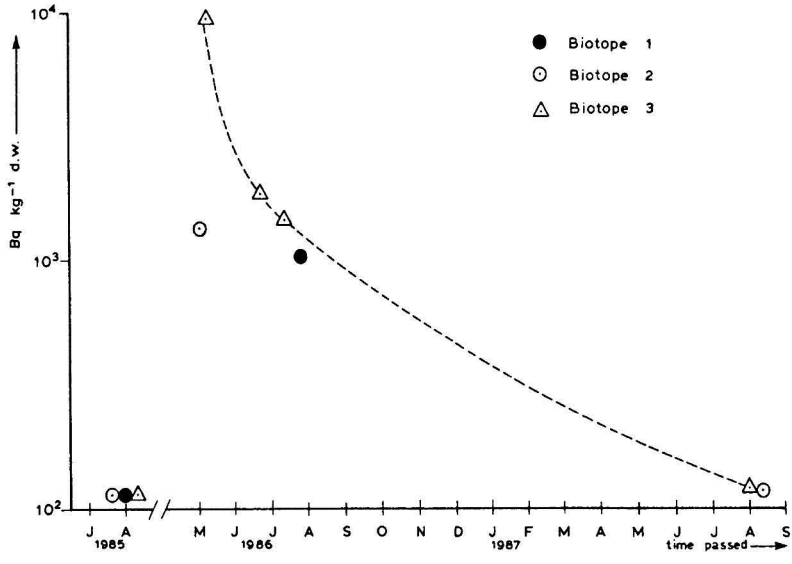

Fig. 2. Average ^{137}Cs concentrations in higher plants vs. time.

species varied from 525 Bq kg^{-1}d.w. to 3127 Bq kg^{-1}d.w. (average 1358 Bq kg^{-1}d.w.). The cesium content of plants in this biotope was lower than that of plants of biotope 3. This was due to the low rates of dry and wet deposition of fallout radionuclides occurring at Chalkidiki Peninsula as indicated by the measurements carried out across Greece (GAEC 1986).

A year after, a repetition of the collection and measurement of the radioactivity of plant species (13 items) at biotope 2 showed that the cesium content of plants significantly declined and averaged 123 Bq kg^{-1}d.w., illustrating the level of ^{137}Cs in plants one year after the Chernobyl accident.

At the highland (Mt. Paikon) of biotope 1, we collected 43 items of plant species after the accident (24 to 25 July 1986). The ^{137}Cs concentration varied between 118 Bq kg^{-1}d.w. and 4500 Bq kg^{-1}d.w. (average 1110 Bq kg^{-1}d.w.) (see Table 1a). Comparison with the ^{137}Cs concentration of the herbarium plants collected from the same biotope in August 1985 (average ^{137}Cs content 102 Bq kg^{-1}d.w.) shows an additional load of cesium in plants as a consequence of the Chernobyl accident (see Table 1a and 1b). Similar measurements were also performed in samples collected from biotope 2 and 3 before the Chernobyl accident. They gave similar results (see the plot of ^{137}Cs concentration versus time as is shown in Fig. 2). From the data of Tables 1a and 3a, the average ^{137}Cs content of plants varied from 1110 Bq kg^{-1}d.w. to 1482 Bq kg^{-1}d.w., illustrating the level of ^{137}Cs in plants about three months after the Chernobyl accident. The largest values of ^{137}Cs concentration in plants of biotope 1 have been observed again in hairy plants, such as *Verbascum phlomoides* (^{137}Cs: 4500 Bq kg^{-1}d.w.), *Teucrium montanum* (^{137}Cs: 3750 Bq kg^{-1}d.w.), *Thymus praecox* (^{137}Cs: 3278 Bq kg^{-1}d.w.), *Lathyrus niger* (^{137}Cs:3185 Bq kg^{-1}d.w.), *Teucrium polium* (^{137}Cs: 2749 Bq kg^{-1}) etc. Besides, the leaching of radionuclides by rainwater (washout) is hindered in plants with hairy leaves or stems and results in radionuclides remaining in the foliage.

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RADON CONCENTRATIONS IN THE AIR OF SLOVENE (YUGOSLAVIA) UNDERGROUND MINES

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Radon-222 was measured in the air of all active underground mines in Slovenia, Yugoslavia, comprising one mercury mine, one lead mine, and six coal mines. Scintillation cells were used for measurement of radon. The highest concentration of 77 kBqm^{-3} was found in the Mežica lead mine, while the concentration in the Idrija mercury mine was usually below 1 kBq m^{-3}; concentrations in coal mines seldom exceeded 0.5 kBq m^{-3}.

INTRODUCTION

Radon in underground uranium mines has been extensively measured and evaluated. Effective ventilation networks have been installed in order to expel radon gas from underground working places and to maintain a satisfactorily low radon concentration, thus minimizing the occurrence of lung cancer in miners.

In the last two decades much concern has been focused on the concentration of radon not only in uranium mines but in all mines, especially metal and coal mines (Duggan et al. 1968; Wetherill 1968; Duggan et al. 1970; Domanski et al. 1970; Strong et al. 1975; Snihs and Ehdwall 1976; Lebecka et al. 1984; Weissbuch et al. 1989; Mair et al. 1985; Tomza and Lebecka 1985. Radon concentrations vary substantially from mine to mine, depending on the geology, geometry, and ventilation of a mine, but they are often found to exceed those in uranium mines, thus justifying the concern.

In Slovenia, the most northwesterly of the six republics in Yugoslavia, there are the following active mines: the Idrija mercury mine, the Mežica lead mine, and six coal mines. In all cases, mining is carried out underground. The Mežica mine is ventilated naturally, but the others have forced ventilation.

This work reports radon measurements made in all active underground mines in Slovenia.

Air was sampled at representative places such as the inlet and exhaust of the mine, in some isolated regions, in some stopes, galleries, haulage ways, shafts, lowest points, etc.

RADON ANALYSIS

Mine air was collected in a 180 cm^3 Pyrex glass α-scintillation cell using a simple laboratory rubber hand pump (Kristan and Kobal 1973). Cells were transported to the laboratory and after three hours α-activity was measured. Scintillation cells were calibrated twice a year according to the Rushing procedure (Rushing 1964) using a radium chloride standard reference solution 4953 D (National Bureau of Standards, now National Institute of Standards and Technology, Washington, D.C.). Before each series of measurements, the counting system was checked by counting a scintillation cell filled with a known activity of radon from a Rushing type bubbler. The background of the cells ranged from 0.5 to 1.0 min^{-1}; the counting efficiency was about 2 (sBq)$^{-1}$

(calibration for radon in equilibrium with its short-lived decay products) and the counting time ranged from 30 to 60 minutes. The lower limit of detection (HASL 1983) was 30 to 85 Bq m^{-3}, depending on counting times and background of scintillation cells used.

DESCRIPTION OF SLOVENE UNDERGROUND MINES

Mežica - Graben lead mine

The yearly capacity is around 250 Gg of lead-zinc ore. Galleries and stopes are left empty after excavation. The volume of the entire empty space underground in 300 years of operation is estimated to be around 20 000 m^3. The length of active haulage ways is around 750 km. There is only natural ventilation with substantial seasonal variations, but no data about the quantity of fresh air circulated are available.

Idrija mercury mine

About 28 Gg of ore is dug per year and 70 Mg of mercury is recovered out of it. The ore deposit lies between 170 and 380 m underground. The total length of the galleries amounts to 150 km. Recently, part of the galleries were refilled by wastes from a smelter plant. There are two fresh air inlets with a total capacity of 3 500 m^3 min^{-1}.

Coal mines

Velenje - Preloge. Lignite is mined at a depth of 100 to 500 m. The method of long wall mining is applied where coal is dug mechanically along a zone of 100 m. The yearly production is about 5 Tg. The length of the galleries is about 90 km. There are four fresh air inlets with a joint fan capacity of 25 000 m^3 min^{-1}. The exhaust air leaves at four outlets.

Trbovlje. About 500 Gg of coal are excavated yearly from a depth of 30 to 170 m. After the excavation the stope advances and the ceiling collapses, thus refilling the empty room. The total length of the galleries is around 35 km. There are two inlet and five outlet fans with a total capacity of 2 000 m^3 fresh air per minute.

Zagorje. The yearly production amounts to 150 Gg of coal from a depth of 430 to 500 m. The empty space after excavation is refilled by soil cover. The total length of the galleries is about 10 km. The joint capacity of the fans is about 900 m^3 min^{-1}.

Hrastnik. Coal is mined at 100 to 200 m underground. Yearly production is 540 Gg. The total length of the galleries is about 12 km. There are three inlet and three outlet fans with a joint capacity of 1 200 m^3 fresh air per minute.

Laško. Approximately 28 Gg of coal is excavated from a depth of 70 to 300 m. The length of the galleries does not exceed 8 km. There is only one inlet and one outlet fan with a total amount of fresh air of 540 m^3 min^{-1}.

Senovo. The yearly production is 28 Gg of coal. The depth goes down to 350 m. The length of the galleries is about 6 km. There is one inlet shaft providing only about 1000 m^3 fresh air per hour.

RESULTS AND DISCUSSION

Results are shown in Table 1. The highest concentrations were found in the lead mine. At working places and in haulage ways concentrations often exceed 1 kBq m^{-3}, and at one place they were about 77 kBq m^{-3} (this value is not included in calculating the mean). Fortunately, this place was in a closed remote area not occupied by miners.

The concentrations were lower in the mercury mine and seldom reached a value of 1 kBq m^{-3}. They were higher than in outdoor air (Kobal et al. 1980), but lower than in nearby underground Karst caves (Kobal et al. 1986).

Elevated radon concentrations were observed in Zagorje, Laško, and Senovo coal mines at all locations where samples were collected. In the others, measured concentrations varied substantially from place to place, depending on the position of the sample location with respect to the main ventilation air stream.

The highest coal mine value of 655 Bq m^{-3} was found in one of the exhaust air streams in the Velenje Preloge mine, a mine where the majority of radon air concentrations hardly exceeded the detection limit. On the other hand, Senovo was uniformly contaminated throughout the galleries. It was not the aim of our work to correlate radon air concentrations to radium content in the coal, nor to the shape and size of the mines, nor to the capacity and distribution of the ventilation networks. The primary goal of this preliminary study was to get an overview of radon concentration levels in the air of all Slovene underground mines. These data are not suitable for making a satisfactorily reliable estimate of radiation doses received by miners. We did not record the time spent by a miner at the working place. Only a rough exposure assessment may be made. The International Commission on Radiological Protection (1986) derived air concentration (DAC) for ^{222}Rn is 1500 Bq m^{-3}

Table 1. Radon-222 concentrations in the air of the Slovene underground mines.

Mine	Date	Time	No. Samples	No. Samples below 100 Bq m^{-3}	Mean + SD (Bq m^{-3})	Range (Bq m^{-3})
Mezica-Graben	3 Mar 1978	9.40-12.10	16	0	1419 ± 498*	590 - 76900
Idrija	11 Sep 1978	9.00-10.52	10	0	658 ± 254	185 - 1050
Velenje-Preloge	4 Oct 1985	8.40-11.25	16	7	171 ± 160	70 - 655
Trbovlje	17 Apr 1986	8.40-11.59	9	5	129 ± 126	30 - 400
Zagorje	22 Apr 1986	8.23-10.40	8	0	264 ± 151	100 - 445
Hrastnik	8 May 1986	9.40-11.35	8	3	102 ± 35	50 - 135
Laško	20 May 1086	9.27-11.50	7	1	281 ± 118	95 - 455
Senovo	4 Jun 1986	10.30-12.30	5	0	334 ± 94	250 - 465

*a single value of 76 900 Bq m^{-3} not included.

for a miner working 2000 h a year and breathing at a rate of 1.2 m^3 h^{-1}. Using this guideline it can easily be seen that even at the most exposed working places (for instance: a stope in the Senovo mine, a region in the Laško mine, a haulage way in the Zagorje mine, a separate exhaust in the Trbovlje mine, or a main exhaust in the Velenje-Preloge mine) miners are not expected to receive more than 15 to 25% of the annual limit for radiation workers. In the lead mine the annual limit can easily be exceeded at almost all work places.

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LETTERS TO THE EDITOR

AN ESTIMATE OF ADULT MORTALITY IN THE UNITED STATES FROM PASSIVE SMOKING; A RESPONSE

Dear Editor:

The health implications of environmental tobacco smoke (ETS) remain controversial. Neither the published reports nor statements from public health officials and agencies have resolved the question of ETS health effects, nor are they likely to in the near future.

A. Judson Wells' paper, "Estimate of Adult Mortality in the United States from Passive Smoking" (1988) is yet another effort to draw scientific verity from a reassessment of published epidemiological data. But this new look does not change the quality or meaning of the existing evidence, which remains equivocal. Neither does it substantively support the author's statement that exposure to ETS "can have adverse long term health effects that are more serious than previously thought".

The conclusions of nonsmokers' increased risk of lung cancer from ETS exposure found in the reports of the National Academy of Sciences (NRC 1986) and of the Surgeon General (USSG 1986) were based on epidemiological studies that produced a wide range of findings. The relative risk (RR) values summarized in Table 12.4 of the NAS report ranged from 0.50 to 3.25, with 17 out of 20 risk estimates (for subgroups by sex) lacking statistical significance. In seven additional reports since the NAS document was published, relative risk values ranged from "<1.00" to 1.65, with only the latter being statistically significant. The RR values from all 29 subgroups in the 20 studies included in the NAS report plus those published later are summarized in Table 1 herein.

All of the epidemiological studies that comprise the data base for estimating nonsmokers' risk of lung cancer in relation to ETS are actually estimates of association based on spousal smoking. In not a single study was either exposure to ETS or retained dosage determined. A few studies have attempted to estimate the degree of exposure to spousal smoking in terms of hours per day or total years of exposure, but none of the studies measured ETS exposure in objective and quantitative terms or even estimated ETS exposure with any degree of reliability. Proximity to a smoker sitting across the dining table does not permit an estimate of the nonsmoker's exposure to ETS, which will vary according to room volume, ventilation rates, the physical and chemical changes in ETS as it ages, and other factors that influence the concentrations and duration of ETS exposure. A spouse's smoking in another room or in another building can have even less or no significance at all in assessing the possible role of passive smoking on a subject's health.

It should be recognized, also, that association can never establish causality. At best, association can only suggest the possibility of causality. Feinstein (1988), discussing public alarms based on epidemiological studies, recently pointed out that "a causal suspicion is supported if an impressive statistical association appears in the 2 by 2 tabulation for subgroups of people reported as being exposed or nonexposed, diseased or nondiseased".

There are many ways to look at data and try to draw meaning from the aggregation of values. After deciding that the 13 studies which survived critical assessment did not, individually or collectively, support a definitive conclusion on the risk of lung cancer in relation to spousal smoking, the NAS Committee performed a meta analysis on the aggregated data, leading to an estimated risk increase of about 34% for nonsmokers married to smokers. This estimate has been questioned on a variety of grounds by a number of investigators (Letzel et al. 1988).

It can be argued that even if a first order relationship does not exist between disease and passive smoking in the epidemiological studies, the data used by Wells are the best evidence available. And it can be argued that even the array of values shown in Table 1 is not impressive in the sense that Feinstein specifies, there are other ways of testing the data, as has been done by Wells.

Table 1. Statistical significance of risk values for lung cancer in relation to spousal smoking.

Investigator	Not Statistically Significant		Statistically Significant	
	Male	Female	Male	Female
*Chan and Fung (1982)		0.75		
*Buffler et al. (1984)	0.50	0.78		
Dalager et al. (1986)	<1.00			
*Kabat and Wynder (1984)	1.00	0.79		
Gao et al. (1987)		0.9 [a]		
*Gillis et al. (1984)		1.00		
*Lee et al. (1986)		1.00		
Gao et al. (1987)		1.1 [b]		
Shimizu et al. (1988)		1.1		
*Garfinkel (1981)		1.17[c]		
*Pershagen et al. (1987)		1.20		
Wu et al. (1985)		1.20		
*Lee et al. (1986)	1.30			
*Garfinkel et al. (1985)		1.31[c]		
*Akiba et al. (1986)	1.80	1.50		
*Koo et al. (1984)		1.64		
Brownson et al. (1987)		1.68		
Humble et al. (1987)	>1.80	1.80		
*Correa et al. (1983)	2.00	2.07[c]		
*Hirayama (1981)			2.25	1.63
Lam et al. (1987)				1.65
*Trichopoulos et al. (1981)				2.11
*Gillis et al. (1984)		3.25		

* Risk values from Table 12.4, National Academy of Sciences Report (1986)
a Exposure in adult life.
b Exposure in childhood.
c Statistically significant trends in one or more data subsets within the study.

There remains, however, the fundamental question of the quality of the individual underlying studies whose data are under consideration. Many of the epidemiological studies assessing the risk of lung cancer from spousal smoking have been criticized for a variety of methodological flaws and weaknesses, especially with regard to the potential for misclassification (Überla 1987; Balter et al. 1986; Lebowitz 1986; OTA 1986).

Misclassification of subjects is a source of error where patients claiming to be never smokers are in fact current or exsmokers. Wells conceded the likelihood of 5% misclassification. But misclassification of smoker status has been found at levels from 10% to 40% (Schwartz et al. 1988; Weiss 1988). NAS noted the likelihood of misclassification and lowered its estimate of the elevated risk to 25% from 34%, but it failed to indicate whether the lower value was statistically significant. (NAS found the combined risk from American studies a 14% increase, which was not statistically significant.)

Misclassification of disease can also be a source of error. There was a marked potential for misclassified disease in the studies having statistically significant risk ratios in the NAS and Surgeon General's reports. In Hirayama's study of Japanese women, his 1984 report suggests that only 21 of the 200 lung cancer cases (10.5%) were histologically confirmed, while the Surgeon General's report states that "none" were verified. Akiba et al. (1986) studying survivors of the Hiroshima and Nagasaki atom bombings, noted 43% of the lung cancer cases had not been histologically confirmed. Weiss (1988) notes that "thirteen percent of the cases [in Garfinkel's study] proved on review not to involve lung cancer".

Misclassification of exposure can be a source of uncertainty in studies that attempt to find exposure-response relationships. There is little basis for considering estimates of spouses' smoking to be reliable. Pron et al. (1988) concluded that "test-retest estimates of reliability [over a six-month time span] would suggest that misclassification of such exposures may be extensive". Vogt (1977) found "twenty-two percent of persons gave different answers on the two questionnaires [on the number of cigarettes smoked per day] given about an hour apart".

Among the variety of flaws and weaknesses found in the various epidemiological studies on ETS and lung cancer, it is worth noting the age bias found by Ahlborn and Überla (1988) in Hirayama's study and their conclusion that "the risk increase ... disappears completely when one removes selection bias by age". Überla (1987), highlighting the weaknesses of the epidemiological studies comprising the NAS data base, had earlier concluded, "False plus false does not equal true."

In addition, most of the epidemiological studies have failed to take into account significant confounding factors in assessing lung cancer risk in relation to ETS. Many risk factors for lung cancer have been identified, including exposure to heavy metals, organic chemicals, combustion by-products, natural and man-made radiation, diet, and nutritional status, personal health history, emotional, and psychological factors. Holst et al. (1988) recently reported significantly increased risk in relation to keeping pet birds and to reduced vitamin C intake. Gao et al. (1987) found no significant increased risk for Chinese women in relation to passive smoking or type of employment but did find significantly increased risk in relation to previous lung disease, cooking practices, and shorter menstrual cycles, reflecting hormonal factors. Some of these factors may act independently, but many may interact. Any attempt to assess the role of one factor must take into account all other relevant factors.

None of the epidemiological studies on spousal smoking took into account confounding factors other than attempting to match cases with controls by age, residence, and general socio-economic status. Of the 20 epidemiological studies, those by Hirayama and by Lam et al. (1987) have the two largest number of lung cancer cases, with the increased risk in both being statistically significant. Both studies are of Oriental populations, which suggests that many factors like cooking practices and fuels for cooking and heating should have been controlled.

All of the studies included in Wells' Table 4, on which he based his estimate of heart disease deaths related to passive smoking, similarly fail to consider the confounding effect of the many cardiovascular disease risk factors that have already been established for that disease.

Some observers have commented that increased risk of lung cancer from ETS exposure seems implausible because the ETS components are so dilute in ambient air compared to the concentrations of these substances in mainstream smoke. In addition, it has been found that nonsmokers retain far less of inhaled ETS than active smokers retain of mainstream smoke. Wells noted that "smoke retention by a passive smoker is only about 1/400 that retained by a direct smoker in a 16 hour day". This is more than one order of magnitude greater than Rickert's calculation (1988) that nonsmokers exposed to ETS retain about 1/8000 the amount of particulate matter retained by the active smoker. Lee (1988) cited estimates of the same range: 1/5000 for males, 1/10 000 for females. All of these estimates are probably on the high side, since none of the studies appears to have considered the chemical and physical changes that occur as ETS ages and the losses of ETS through evaporation, fallout, and deposition over time.

Other observers have commented on the implausibility that lung cancer in nonsmokers might be caused by ETS. Aviado (1988) noted that none of 17 constituents of ETS "designated as suspect carcinogens ... [has] been adequately shown to cause pulmonary cancer via inhalation in animals". Crawford (1988) noted that "no atypical cellular changes have been found in the lungs of nonsmokers". Lee (1987) concluded "that exposure to smoke constituents of nonsmokers is too low to explain the moderate increase in risk of lung cancer seen in epidemiological studies in self-reported never smokers married to smokers. This increase in risk is much more plausibly explained by misclassification of smokers as nonsmokers than by a direct effect of passive smoking".

Wells has attempted to make his calculation of annual deaths from exposure to ETS appear more reasonable by comparing it to the larger estimate of Repace and Lowrey, but their estimate has been severely criticized because the controls were Seventh Day Adventists (SDA) whose life style is so radically different from that of the non-SDAs married to smokers that the comparison is considered inappropriate (OTA 1985; Balter et al. 1986; Überla 1987).

Taking these and other factors into account, Gostomzyk (1986) concluded, following the International Experimental Toxicology Symposium on Passive Smoking in Essen, FRG, that "even toxicology has not been able to ascertain with any greater degree

of probability than did epidemiology that there exists a link between damage to health and passive smoking".

Perhaps it is the weight of these facts, interpretations, and opinions that caused no less an authority than the American Cancer Society to assert last year that "the currently available evidence is not sufficient to conclude that passive or involuntary smoking causes lung cancer in nonsmokers..." (ACS 1988).

A final comment: both the title and the content of the editorial that accompanied the Wells paper suggests that the paper provides stronger evidence of risk of cardiovascular disease (CVD) for nonsmokers married to smokers than the paper in fact offers. In 1986, both the NAS and USSG reports noted the lack of convincing evidence of significant CVD risk from ETS exposure. More recently, Fielding and Phenow (1988) commented on papers reporting an association between ETS exposure and CVD risk, concluding that "no firm conclusion that a causal relation exists is yet warranted".

Wells' calculations with respect to CVD are based on data from epidemiological studies that have the same weaknesses as the lung cancer studies. There is, thus, no basis for greater confidence in his estimate of heart disease deaths in relation to ETS than his estimate of lung cancer deaths.

It is commendable that those who are not satisfied continue to seek more meaning from the data. But in an issue as serious as this, it is important to note when the data fail to meet the standards for scientific inference.

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AN ESTIMATE OF ADULT MORTALITY IN THE UNITED STATES FROM PASSIVE SMOKING; A RESPONSE

Dear Editor:

Wells (1988) estimates that exposure to environmental tobacco smoke (ETS) causes 46 000 deaths per year in the U.S.; 3000 from lung cancer, 11 000 from other cancers, and 32 000 from heart disease. These estimates are scientifically unjustified. Far too much faith is placed on results from often fragile epidemiological studies, with major sources of bias ignored or totally underestimated. In contrast, far too little faith is placed on evidence that nonsmokers have very much lower exposure to tobacco smoke constituents than do smokers, and that smokers are much more exposed to ETS than nonsmokers.

The evidence that exposure to ETS increases the risk of developing heart disease is extremely unconvincing. Of the studies cited by Wells, some are based on unacceptably small numbers of cases, e.g., Garland et al. (1985) where only two deaths occurred in women married to never-smoking husbands, while the only two studies with substantial numbers of deaths are both open to question.

When referencing the Japanese prospective study, Wells uses Hirayama's 1984 report of a statistically significant positive trend in wife's age-adjusted risk according to husband's smoking, but does not comment on the fact that, in 1981, Hirayama reported no association whatsoever. As shown in Table 1, the

Table 1. Female relative risks for heart disease from passive smoking in Japanese study.

Follow-up period	Total cases	Husband's smoking habit: Non-smoker	Ex or <19/day	20+/day
1966-79	406	1	0.97	1.03
1980-82[1]	88	1	2.85	5.07
1966-82	494	1	1.10	1.30

[1] Estimated from 1966-79 data (Hirayama 1981) and from 1966-82 data (Hirayama 1984). The 1984 paper provided relative numbers of deaths as 118, 240, and 136.

1966-79 and 1980-82 data are totally inconsistent and statistical tests confirm the highly significant ($p<0.001$) interaction between relative risk and period of follow-up. A possible explanation might be that the 1981 data, but not the 1984 data, were standardised additionally for occupation, but if this was important, why did Hirayama not standardise for occupation in 1984?

The Maryland prospective study (Helsing et al. 1988), which reported a 24% increase in heart disease risk in women, based on 988 deaths, and a 31% increase in men, based on 370 deaths, in relation to living with a smoker, has a number of features that should be considered when interpreting the data. No attempt was made to follow-up people moving outside Washington County, thus presumably missing large numbers of deaths. No dose-response relationship was reported. Adjustment for age, marital status, years of school and quality of housing had an enormous effect on relative risk, changing estimates from 1.17 to 1.31 in men and from 0.66 to 1.24 in women. No direct adjustment was made for household size, despite the fact that the larger the household, the more likely it is to contain a smoker. Furthermore, no direct adjustment was made for the possible correlation of household size with various coronary risk factors. Also, data were unavailable on a whole range of factors, such as diet and exercise, which might differ in families with and without smokers. In short, several potential confounders were apparently not controlled for.

Wells does not consider the problem of publication bias. This may be particularly acute for heart disease. After all, it is a vastly more common disease than lung cancer in nonsmokers, but the numbers of deaths in Wells' tables are only slightly greater. The possibility can surely not be excluded that other researchers, perhaps with much larger and better data bases, have looked at the relationship and found nothing.

The data for cancer other than the lung are even less convincing than for heart disease. In view of the much greater passive smoke exposure of smokers than nonsmokers, observations that nonsmokers exposed to passive smoking have increases in cancers at sites not increased in smokers seem to me to suggest that something is wrong with the epidemiological studies. And, indeed, the paper showing the strongest association (Sandler et al. 1985) is open to a number of serious criticisms (Lee 1985). Wells, however, remains content to include all epidemiological studies in his meta-analyses, regardless of quality, and attempts to explain obviously spurious relationships by an unsupported, and implausible hypothesis, involving an especially susceptible group of individuals who all die early if they smoke but die later by passive smoking if they do not. Mortality patterns for lung cancer in terms of age, dose, and duration of smoking are in fact well described by models involving no component for variation in susceptibility at all.

Wells' estimate of 3000 lung cancer deaths per year based on the epidemiological data contrasts with that of 12 by Arundel et al. (1987) based on extrapolation using relative amounts of particulate matter retained in the lung by nonsmokers and smokers. As I argue at length elsewhere (Lee 1987; 1988a; 1988b; 1989a; 1989b), it is far more plausible to conclude that the association observed between lung cancer and exposure to ETS arises predominantly because of bias than it arises because of a carcinogenic effect of such low doses of ETS.

Misclassification of smokers as nonsmokers is likely to be a major source of bias in most studies and is one for which Wells' correction is totally inadequate. He does not allow at all for the possibility of misclassified current typical regular smokers, whereas a recent summary of data from large studies shows an average rate of about 4% (Lee 1989a). Nor do his calculations take into account recent data (USSG 1989) showing much higher relative risks in active smokers than in older studies. Preliminary calculations based on these data suggest that the total number of lung cancers occurring in self-reported never smokers in the U.S. may have been substantially overestimated. Rather than 12 000 the figure may be nearer 8000. If reasonable corrections are made for misclassification, the figure of lung cancer deaths among actual never smokers may be less than 6000.

Wells considers his overall estimate of 46 000 deaths conservative. I disagree. When better data are available, it may prove to be about 46 000 too high.

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ISCHEMIC HEART DISEASE; RESPONSE TO LEE

Dear Editor:

Dr. P. Lee questioned the reasons for a discrepancy of my reports in 1981 and in 1984 on husbands' smoking and ischemic heart disease risk in nonsmoking wives.

The 1981 report was based on a 14 year follow-up (n=400) and the 1984 report was based on a 16 year follow-up (n=494) of nonsmoking wives. The relative risks of ischemic heart disease when husbands were nonsmokers, exsmokers, or daily smokers of 1-19 cigarettes and 20 or more cigarettes were 1.00, 1.06, and 1.18 (trend p : 0.061 not significant) in the 14 year follow-up; and 1.00, 1.10, and 1.31 (trend p : 0.019 significant) in the 1984 report.

Table 1. Ischemic heart disease mortality in women by age group, by occupation, and by husbands' smoking habit (patient herself a nonsmoker).

Husband's occupation	Husband's age group	Husband's smoking habit: Nonsmoker		Exsmoker 1-19/day		20+/day		Total	
Agricultural worker	40-49	8	2,502	25	5,941	17	3,636	50	12,079
	50-59	15	3,497	27	6,812	27	3,514	69	13,823
	60-69	36	4,084	79	6,845	27	2,152	142	13,081
	70-	5	323	11	446	2	89	18	858
	Total	64	10,406	142	20,044	73	9,391	279	39,841
Other	40-49	5	3,727	15	9,093	15	7,128	35	19,948
	50-59	11	4,294	29	8,830	23	6,306	63	19,430
	60-69	29	3,036	46	5,598	20	2,499	95	11,133
	70-	9	432	8	619	5	137	22	1,188
	Total	54	11,489	98	24,140	63	16,070	215	51,699
The weighted point eatimate of rate ratio and test-based 90% confidence limits		1.00		1.11 (1.33 / 0.92)		1.36 (1.68 / 1.09)		Mantel extension chi 2.539	
Mantel-Haenszel chi		-		0.882		2.331		One tail p value 0.00916	
One-tail p value		-		0.18889		0.00988			

Possible reasons would be (1) a longer follow-up period and more cases in the 1984 report than in the 1981 report, or (2) husbands' age and occupation were standardized for data in 1981, while data reported in 1984 was standardized by age only. However, the latter is definitely not the reason responsible for the discrepancy, as age-occupation standardized data in 1984 showed almost similar results, corresponding relative risks (r.rs) being 1.00, 1.11, and 1.36 (trend p : 0.009), respectively (Table 1). The results were also similar when standardized by wives' age, corresponding r.rs being 1.00, 1.09, and 1.34 (trend p : 0.019). Therefore, it should be concluded that the more cigarettes the husbands smoke, the higher the ischemic heart disease risk in nonsmoking wives.

In 1980-1981, r.rs of ischemic heart disease in nonsmoking wives were 1.00, 1.29, and 1.87 (trend p : 0.041) when husbands were nonsmokers, exsmokers/10-19 daily, and 20+ daily respectively. One may further consider as the possible reasons for this discrepancy the influence of the changing quality of side-stream smoke coming out of the ignited end of cigarettes in recent years due to the intensive chemical manipulation of the products (e.g., inclusion of tobacco additives) in order to lower tar and nicotine, to improve the flavor, etc. Also, the recent increase in fat consumption in Japan may interact on the risk of ischemic heart disease when exposed to passive smoking.

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REBUTTAL TO LEE/KATZENSTEIN COMMENTARY ON PASSIVE SMOKING RISK

Dear Editor:

Lee (1989) and Katzenstein (1989), in their commentary on Wells'(1988) paper, take issue not only with Wells' estimates of the magnitude of the mortality effect of passive smoking on nonsmokers, but question whether mortality occurs at all. Their arguments are based upon the alleged fragility of the epidemiological studies of passive smoking and disease; the potential for misclassification of subjects, disease, or exposure; possible confounding factors; and the lower doses of smoke to which nonsmokers are exposed relative to smokers.

Let us examine these issues one by one. Are nonsmokers exposed to such low doses of environmental tobacco smoke (ETS) that Wells' estimates of 46 000 nonsmokers' deaths per year from passive smoking are about "46 000 too high", as Lee asserts? Perhaps the most salient point to be considered: active smoking is a cause of more than one out of every six deaths in the U.S.A. every year (USSG 1989). Intentional exposure to tobacco smoke has been judged to cause coronary heart disease, atherosclerotic peripheral vascular disease, lung and laryngeal cancer, oral cancer, esophageal cancer, chronic obstructive pulmonary disease, chronic bronchitis, intrauterine growth retardation, and low birthweight babies. In addition, probable causality has also been established for unsuccessful pregnancies, increased infant mortality, and peptic ulcer disease, as well as cancers of the bladder, pancreas, and kidney, and associations have been reported for cancer of the stomach (USSG 1989). Hardly an organ system of the human body remains undiseased upon exposure to tobacco smoke. To argue, as do Lee and Katzenstein, that the diseases of smoking are not even plausible in nonsmokers does not give us confidence in their deductive abilities. To be sure, it is possible that thresholds for effect may exist for one or more of the diseases of smoking, but neither Lee nor Katzenstein present any evidence whatsoever that such low dose thresholds exist, let alone that all nonsmokers have exposures and susceptibilities which place them within an adequate margin of safety below such thresholds.

Are the epidemiological studies of passive smoking and lung cancer really all to be explained by misclassification of smokers as nonsmokers as Lee has proposed? Nonsmokers who report no passive smoking nevertheless possess levels of nicotine and cotinine in body fluids which are significant fractions of those who report a lot of exposure. For

example, of 100 nonsmokers studied by Jarvis (1987), the 46% who reported "no" exposure had measured urinary cotinine levels which were nearly a third of the levels of those 27% of nonsmokers who reported "some" or "a lot" of passive smoking exposure. This suggests that there is major misclassification of nonsmoking controls as "unexposed". The result of this kind of misclassification of nonsmokers is to cause epidemiological studies to lack statistical significance or to find no effect. Nevertheless, despite such misclassification of controls, fully two-thirds of the studies shown by Katzenstein in his Table 1 showed a positive result.

Are confounding factors such as higher exposure to carcinogenic organic chemicals from non-ETS sources in the spouses of smokers, as Katzenstein asserts, really responsible for the consistent reports linking lung cancer to passive smoking from 15 different researchers in six different countries? To the contrary: Wallace (1989), in making measurements of personal exposure to benzene, a known human carcinogen and a prominent constituent of tobacco smoke, found that benzene exposures were 50% higher in the nonsmoking children and spouses of smokers than for nonsmokers in nonsmoking households.

Finally, what of the magnitude of Wells' (1988) estimates which Lee asserts are 46 000 too high? Let us take lung cancer, which Wells has estimated at 3000 U.S. lung cancer deaths (LCDs) per year. Lee selectively contrasts the estimate of 12 LCDs/yr from passive smoking by Arundel et al. (1987), but omits the mention of eight other risk assessments with which Wells' assessment agrees, all eight of which taken together average 5000 ± 2400 LCDs/yr. (Repace and Lowrey 1990). It is Arundel et al. who are out of step with the rest, not Wells. This lends credence to Wells' risk assessment methodology.

As far as heart disease mortality is concerned, this is primarily a disease of those aged ≥ 35 years. In 1985 there were roughly 105 million Americans in this age bracket, roughly 72 million nonsmokers, and 33 million smokers. Among the 33 million smokers, there were 120 000 active smoking-attributable heart disease deaths (HDDs) in 1985, or 3.6×10^{-3} HDD/smoker. By comparison, Wells' estimates 32 000 passive smoking-attributable nonsmokers' HDDs per year in a population of 72 million, or 4.4×10^{-4} HDD/nonsmoker. Thus, the ratio of ETS-induced heart disease deaths per nonsmoker to smoking-induced heart disease deaths per smoker is only 12%, which does not seem excessive considering that tobacco smoke is known to be one of three major risk factors for HDD, and synergistic (USSG 1989) with the other two factors (hypertension and elevated serum cholesterol) which are also common in nonsmokers.

A final note on Katzenstein's attack on the risks of passive smoking-induced lung cancer death (LCD) estimated by Repace and Lowrey (1985, 1986, 1987). The radical difference in lifestyle between never-smoking Seventh Day Adventist (SDA) controls and never-smoking non-SDAs is the avoidance of passive smoking in the SDA lifestyle, which we believe convincingly accounts for their lower lung cancer rate. As Katzenstein selectively notes, we were criticised by OTA (1985) and by tobacco industry consultants for attributing the entire LCD rate difference to passive smoking, but what our critics have conveniently ignored is that, since 60% of the SDA control group were potentially exposed to passive smoking, this was in fact a conservative estimate. Moreover, Katzenstein selectively omits mention of the analysis of our work by Weiss (1986), who found our figures to be "the best current estimates of lung cancer deaths from passive smoking".

In sum, contrary to the assertions of Lee and Katzenstein, we find Wells' predictions of 46 000 deaths per year from passive smoking to be credible, and to indicate, as Wells concluded, that exposure to ETS can have adverse long-term health effects that are more serious than previously thought.

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AN ESTIMATE OF ADULT MORTALITY IN THE UNITED STATES FROM PASSIVE SMOKING; A RESPONSE

Dear Editor:

An article in Inside EPA (January 13, 1989) is headlined: "EPA weighs Impact of Study Linking Passive Smoke Exposure to Heart Deaths..." It leads with the statement: "EPA is giving serious attention to a recently published study that pinpoints passive smoking . . . as a significant cause of heart disease and cancer-related deaths". The article states: "Passive smoking causes 46 000 deaths a year, according to a study by A. Judson Wells published last month in *Environment International*". An EPA source is quoted: "The 46 000 mortality was surprising because such a large component was from heart disease . . ." This statement is similar to one made by EPA's James Repace on national television when the report was first released.

What is surprising is that anyone from the EPA can consider this recent review surprising. Dr. Wells has not completed an epidemiological study, new or otherwise, and has in no way contributed to pinpointing passive smoking as a significant cause of heart disease, lung cancer, or other cancer deaths. What he did was publish the results of a series of calculations based on the results of existing epidemiological studies and a number of assumptions (Wells 1988). Dr. Wells presented a similar analysis at the 1986 Air Pollution Control Association meeting, which was published in the meeting proceedings (Wells 1986). There should have been no sudden surprise at EPA; an EPA official chaired the 1986 session in which this paper was presented. Dr. Wells encourages the view that he had done something new by failing to even acknowledge his previous presentation.

Wells used the data of previously published (and in some cases, unpublished) studies as a basis for calculating annual mortality statistically associated with ETS exposure. These calculations do not in any way establish that ETS does, in fact, cause death in exposed individuals. Rather, such calculations rely on an independent conclusion, based on a review of the available data, that ETS causes lung cancer, other cancers, and cardiovascular diseases. If such a conclusion cannot be supported, then the estimate of ETS-associated mortality rests on the assumption that ETS causes these diseases, and it is incumbent upon the author to state this underlying assumption when reporting the results of his calculations.

The issue of causation is never addressed by Wells. The studies cited in Wells' Tables 1-4 are discussed below with particular attention to whether they establish a causal relationship between ETS and disease in non- or never-smokers. The vast majority of the studies were included in reviews published by the National Academy of Sciences (NAS 1986) and the Surgeon General (USSG 1986). Therefore, these reports are used as a starting point for addressing the question of causality.

Lung Cancer: Almost all of the epidemiological studies listed in Wells' Tables 1 and 2 were considered in the NAS and Surgeon General's reports, as well as other reviews appearing at about the same time (Blot and Fraumeni 1986; Überla 1987). The Surgeon General's Report was alone in concluding that ETS causes lung cancer in nonsmokers; the other reviews generally concluded that although a statistical association appeared to exist between marriage to a smoker and the risk of lung cancer, the lack of adequate exposure information, and the potential influence of differential misclassification of smoking status precluded a conclusion of causality. The lung cancer studies published since these reviews have the same limitations as the previous studies. Little has been published since 1986 that adequately addresses the issues of exposure and misclassification.

All of the studies attempting to link cancer to ETS have been epidemiological. An epidemiology study attempts to relate the frequency of a certain health effect or disease with the frequency of specific envi-

ronmental exposures within a study group. Because of the nature of this type of study, all it can conclude is that the exposure and health effect do occur together with a measurable frequency. They do not prove a cause and effect relationship.

Koo et al. (1988) performed a detailed investigation of potential confounding factors in the lifestyle of nonsmoking women married to either a nonsmoking spouse or a smoking spouse. Overall, women married to ever smokers had a less healthy lifestyle, ate less vitamin A vegetables, ate more cured foods, ate more spicy foods, and drank more alcohol than women married to nonsmokers. Their analyses show that caution should be exercised when interpreting data on ETS without considering other factors.

Feinstein (1988) described some of the problems or failings that have come to characterize many epidemiological studies. Several examples are given where commonly used substances were accused of being a menace to daily life after epidemiologists reported a relatively weak association between use of the substance and adverse health effects. Some of these accusations have subsequently been refuted or withdrawn. Feinstein states that "[d]espite peer-review approval, the current methods need substantial improvement to produce trustworthy scientific evidence".

Other Cancers: With the exception of the Reynolds et al. study (which is unpublished and, therefore, inappropriately included in the analysis), all of the studies cited in Wells' Table 3 were included in the NAS and Surgeon General's reports. The NAS concludes that there is no consistent evidence, based on these studies, of any increased risk of ETS exposure for "cancers other than lung cancer". The Surgeon General's report similarly suggests that further investigation will be needed before any conclusion can be made.

Cardiovascular Disease: Wells suggests that a considerable body of new epidemiological data on ETS and cardiovascular disease has become available, which significantly impacts the analysis of data for this disease endpoint. This assertion is emphasized in the Inside EPA report. In fact, with the exception of Helsing et al. (1988), all of these data were available to the NAS and Surgeon General's review panels. The study of Martin et al. was available at the time but was unpublished, and for good reason, it thus was not cited in these reviews. The study remains unpublished, and the data should not be included in the present analysis.

Both the Surgeon General's and the NAS reports find the data on ETS and cardiovascular disease, available at the time of their reviews, to be inconclusive. The inconclusiveness of the studies reflects not only small sample sizes but also a number of significant deficiencies in their design, as detailed in both the NAS and Surgeon General's reports. The questionable mathematical combination of the findings of these studies, as done by Wells, overcomes the problem of small sample size but in no way addresses the methodologic issues that have been raised.

The prospective study of Helsing et al. (1988) reports a statistically significant increased risk of death from cardiovascular disease in nonsmokers exposed to tobacco smoke in the home compared to those not so exposed. The authors of the study conclude that "[i]t seems reasonable to suppose that tobacco smoke is a risk factor in the increased risk". That rather weak conclusion reflects, in part, some aspects of the Helsing study that are inconsistent with such a conclusion. For example, the relative risk (RR) of death from heart disease associated with household exposure to ETS is reported as highest in the youngest age group studied (25-44 years old), even though the individuals in the older age groups presumably were exposed to ETS for much longer periods. Given the same estimate of household exposure, individuals in the older age groups would be expected to have had a higher risk of cardiovascular death than those in the younger group.

Both the Surgeon General's and NAS reports are cautious in their discussions of the quantitative risk associated with ETS exposure. Appendix D of the NAS report, which Wells cites in support of his risk models, emphasizes the underlying assumptions on which the calculations for lung cancer are based. The results are summarized in a section entitled, "Summary of Main Results Under the Assumption That the Summary Rate Ratio of 1.3 is Causal". The Surgeon General's report states (p. 96): "The quantification of the risk associated with involuntary smoking for the U.S. population is dependent on a number of factors for which only a limited amount of data are currently available". These factors include a better understanding of the magnitude of ETS exposure, its distribution among different segments of the U.S. population, and changes in the patterns of ETS exposure that have occurred over the last century. There is no better understanding of these factors now than there was in 1986. Wells bases his exposure estimates on data published by Friedman et al. (1983) — data that apparently were considered to be insufficient by the authors of the Surgeon General's report.

As Wells depended to a large extent on the Helsing (1988) report, it is important to review carefully the methodology used in that report. A general census

was taken in Washington County, MD, in 1963 that included, among other factors, smoking histories of families and number of rooms in the house. Twelve years later, Helsing and colleagues reviewed death certificates to determine cause of death over the 12 years. They noted those deaths that were coded as arteriosclerotic heart disease and other myocardial degeneration. They then calculated a relative risk of death due to arteriosclerotic heart disease of nonsmokers married to smokers versus nonsmokers married to nonsmokers. The relative risks were 1.31 for men and 1.24 for women after adjusting for age, marital status, years of schooling, and quality of housing.

It is very important to note that the authors reported that there was a small difference in RR if heart disease was listed as the underlying cause of the death or just listed on the certificate as one of several reasons for death. The actual cause of death as listed on death certificates could in itself be a confounding factor in this study. In addition, overall relative risks were adjusted for age, marital status, etc. There is no description of how the quality of housing is calculated or adjusted for, nor is there any attempt to look at other possibly related health factors in the subpopulations to determine if these factors could have influenced arteriosclerotic heart disease. In addition, no attempt was made to measure smoking status misclassification.

Wells concludes his report by suggesting that exposure to ETS actually may cause more than 46 000 additional deaths per year. He quotes Repace and Lowrey (1985) and their estimate of 4665 additional lung cancer deaths as support for that suggestion. The Repace and Lowrey estimate scares a lot of people who have not taken the opportunity to review their underlying assumptions. What is overlooked in the emotionalism is what the Repace and Lowrey report really says.

Repace and Lowrey start with the assumption that direct smoking and ETS both cause cancer. They do nothing to prove this. They then use a long series of estimates of exposure concentrations and exposure durations to compare ETS exposure to direct smoking. Finally, they calculate the death rate from lung cancer using these assumptions and estimates. What they generate is a calculated guess, not a prediction based on facts.

Most of the research done since the Repace and Lowrey study has not supported its findings. One of the better studies has calculated that a person exposed to ETS actually retains 0.02 percent (or 1/5000) of the particulates of a direct smoker (Arundel et al. 1988).

Repace and Lowrey calculate a nonsmoker to be exposed to an average of 1.43 mg/day of particulates from ETS. Arundel et al. calculated the amount to be 0.07 mg/day for male nonsmokers and 0.03 mg/day for female nonsmokers. These two estimates of ETS exposure differ by a factor of between 20 and 45. Thus, estimates based on exposure assumptions and models are simply estimates. One needs only to change a few of the basic premises to arrive at a completely different set of conclusions. Wells' reliance on assumptions derived from the exposure assumptions of Repace and Lowrey leave his own conclusions highly questionable.

It is apparent from this brief overview that Wells' computations rely on risk ratios derived from epidemiological studies that do not establish a causal link between ETS exposure and the risk of disease. What part, if any, of the association between marriage to a smoker and lung cancer or cardiovascular disease is due to ETS is a matter of debate. Resolution of that debate depends on further research to address the exposure and misclassification issues. Pending resolution of these questions, Wells is obligated to state and fully discuss the assumptions that underlie his calculations.

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AN ESTIMATE OF ADULT MORTALITY IN THE UNITED STATES FROM PASSIVE SMOKING; A RESPONSE TO CRITICISM

Dear Editor:

Lee (1990), Katzenstein (1990), and Holcomb (1990) have commented negatively on my paper (Wells 1988a) in *Environment International*, in which it was suggested that the U.S. death toll from passive smoking may be 46 000 per year. Space does not allow me to deal with all of the points raised, but the more important ones are covered below.

Lee, as tobacco consultants usually do, attacks the underlying studies that I used, particularly the heart studies. I cannot speak for these authors. Dr. Hirayama has written a reply of his own. Dr. Sandler (private communication) has told me that they (Helsing et al. 1988) did look at family size and found no effect. The Johns Hopkins School of Public Health (Helsing et al. 1988) and the University of California, San Diego (Garland et al. 1985) are respected schools of epidemiology, whose researchers presumably know how to adjust for confounding variables. They attempted, within the limits of the data available, to account for known heart risk factors as noted in my paper. What is striking about the heart data in my Table 4 (Wells 1988a) is the consistency of the various results. It is interesting that Lee et al. (1986) made no attempt to adjust for any of the known heart risk factors except age.

Publication bias in smoking studies is an issue often raised by tobacco industry consultants, but so far no one has found a live passive smoking case that is negative. I have dealt with that issue vis-à-vis passive smoking and male lung cancer in my comment (Wells 1988b) on Vandenbroucke (1988). There, it was pointed out that the only available unpublished data were on the high side of the most probable relative risk, not low or negative. In that letter, I asked investigators to send me any data on passive smoking that had not been published or that they had not been able to get published. So far I have received none. As Lee says, the possibility of a large, unpublished data set that found nothing cannot be excluded; it is just extremely unlikely.

For cancers other than lung that are passive smoking related, all except nasal sinus cancer and lung cancer are non-contact sites, as is heart disease. For these sites to be activated, the disease-producing entities must, in most cases, be metabolized and then circulate in the blood and lymphatic systems. Earlier work (Eatough et al. 1986) has shown that 90% of the nicotine in environmental tobacco smoke (ETS) is in the vapor phase. Now Pritchard et al. (1988) have shown that 70% of the tar in ETS is also in the vapor phase. The nicotine and the tar in direct smoking is in the particulate phase. It is true, as Lee says, that smokers are also passive smokers, but for the non-contact sites there is growing evidence that smokers have a higher risk if they are exposed to ETS other than their own than if they are not so exposed. For example, Palmer et al. (1988) found a relative risk for heart disease of 1.34 for spouse exposure of light smoking women and 1.32 for heavy smoking women, and Sandler et al. (1985) found overall cancer risks increasing from unity to 2.4 as active smokers were exposed to an increasing number of household members who smoked. This means that smokers may also be at considerable risk from passive smoking of their own smoke. In other words, for the non-contact sites, the vapor phase tar and nicotine may be the primary culprits, with the particulate phase having less effect. The particulate phase, at least most of it, is relatively quickly cleared. It probably contributes heavily to the contact sites (central lung, mouth, esophagus, and stomach) but then may be eliminated in the feces. All this means that Lee's model for passive smoking, which is based on direct smoking and particulate phase deposition and retention, is likely to predict relative risks for passive smoking that are far too low for the non-contact sites and probably for peripheral lung cancer as well.

There are other factors that make the prediction of passive smoking health effects by rationing down from the particulate dose of direct smoking chancy. One factor is the possible protective effects in direct smoking. Smoking is known to depress estrogen levels which can protect against breast cancer. Such a protective effect is unlikely from passive smoking. Remmer (1987) postulates that direct smoking activates protective enzymes. Lassila et al. (1988), in their interesting work with monozygotic twins, have shown that direct smoking results in higher levels of prostacyclin, a reactive vasodilator, which, they note, could compensate for the vasoconstrictive effects of cigarette smoking. The dose from passive smoking is probably too low to promote this protective effect. Sinzinger et al. (1982), later confirmed by Burghuber et al. (1986) and Davis et al. (1989), found that platelet sensitivity, a known risk factor for heart attacks, is depressed about 30% in passive smokers, almost to the level found in active smokers. There is no way that the relative retained particulate dose could account for this phenomenon.

Direct smoking and passive smoking are both complex phenomena, with both disease promoting and disease protective components that differ between direct and passive smoking, and where the balance between them differs among individuals. Lee denigrates my suggestion that individual susceptibility could explain, in part, the higher than expected adverse health effects of passive smoking. The science of identifying highly susceptible people is progressing. See for example the work that Caparosa et al. (1989) are doing at the National Cancer Institute on "fast metabolizers" of potential carcinogenic materials. Jones (1986) has shown a substantial difference in sensitivity of different individuals to nicotine and its effect on pulse rate. Khoury et al. (1989) have developed equations for estimating the proportion of persons who are susceptible to a risk factor. They estimate that 13% of smokers are susceptible to lung cancer, whereas only 0.9% of smokers are susceptible to esophageal cancer. My calculations, using their formulae, indicate that only about 0.4% of nonsmokers are susceptible to death by lung cancer from passive smoking.

Lee says that I am "content to include all epidemiological studies" in my meta-analyses, regardless of quality. Actually, I discarded four lung cancer studies because they did not meet stated criteria. The admission criteria are admittedly broad because I did not wish to be accused of biased selection. Originally, I had intended to use only statistically significant data, but the meta-analysis technique allowed the inclusion of smaller studies when properly weighted. A certain amount of scatter is to be expected and is observed in the relative risks from these smaller, low power studies.

Lee (1990) argues that the association between lung cancer and exposure to ETS arises predominately because of bias caused by misclassifying smokers as nonsmokers. In his analysis he seems to have gone out of his way to stretch the data to fit his hypothesis. For example, he states that current typical regular smokers are misclassified to the extent of about 4%. In his workup (Lee 1986, 1987), he has confused smokers who say they are current non-users of tobacco with smokers who say they are never smokers. Yet the epidemiology of passive smoking deals almost exclusively with people who say that they are never smokers. Lee also averages male and female data in order to get higher misclassification factors. Normally in misclassification calculations, one uses sensitivity, which is defined as stated positives divided by stated positives plus false negatives, or in other words, the percent correctly classified as exposed, or in this case, the percent of ever smokers that are correctly classified as ever smokers. By basing his calculations on the number misclassified relative to never smokers instead of relative to ever smokers as he should have, he claims to be able to average male misclassifieds (who are mostly exsmokers) as 18% of self-reported never smokers with female misclassifieds as 6% of never smokers to yield a 10% misclassification factor. The misclassified males as 18% of never smokers are equivalent to only 6% of ever smokers (18% × 25/75) which is essentially the same as the female result (6% × 50/50). Of course the safe thing to do when estimating the bias in female passive smoking relative risks is to use only female data. In a paper in preparation for which I am a co-author, we found, when averaging data from five cotinine studies, including Lee's, that only 1% of female ever smokers said they were never smokers when they were actually current regular smokers, not 4% as Lee contends. Lee uses 10 as the observed relative risk for the regular current smokers that are misclassified as never smokers. The proper procedure is to use smoker relative risks that are consistent with the time frame and locale of the epidemiological studies for which a bias calculation is being made. Fortunately many of the passive smoking epidemiological studies on lung cancer have concurrent estimates of the relative risk of current or ever smokers, and values for the other studies can be estimated from available data. In fact, many of these values are shown on page 72 of Lee's book (1988). A

log weighted average of the current smoker relative risk for the studies shown in Table 1 of my paper (Wells 1988a) is 4.56 (it was assumed that current smoker relative risk was 30% higher than ever smoker relative risk if those were the only data available) which is less than half the value of 10 used by Lee. Lee's book (1988) has whole sections devoted to misclassification factors for people who say they are recent exsmokers. These data appear to be introduced simply to confuse the reader since they have no bearing on passive smoking epidemiology which deals essentially entirely in self-reported never smokers. If proper factors are used for the extent of smoker misclassification and smoker relative risk, the bias that one calculates agrees with the values previously estimated by Wells (1986a, 1988a) and Wald et al. (1986), and not with those of Lee.

Lee suggests that my estimate of passive smoking deaths may be high. My heart relative risk of 1.23 is supported by two new studies and an update on a third. Palmer et al. (1988) report a female heart relative risk for passive smoking of 1.2, and Humble et al. (1990) report 1.6. Hole et al. (1989), in an update on the study of Gillis et al. (1984), report a female heart relative risk of 2.1 for low exposure passive smokers and 4.1 for high exposure. Sandler et al. (1989) found no increase in risk for total cancer in women, but Miller (1989) in his new study found that non-smoking, non-employed wives of nonsmokers accounted for only 3% of cancer deaths but a much higher percentage of total deaths. These two new results will offset each other. Sandler et al. (1989) also show a statistically significant female all cause relative risk of 1.15 for passive smoking, essentially identical to the 1.165 value I had derived in Appendix B (Wells 1988a) from earlier data. This tends to validate my estimate of 34 000 female all cause deaths from passive smoking. Sandler et al. (1989) also report a statistically significant all cause relative risk for men of 1.17 (the first such data available) that would result in 29 000 deaths per year for a total for both sexes of 63 000, higher than, but not too far distant from the 46 000 deaths that I estimated from the three-disease approach.

In our Western, non-traditional societies, it is very difficult to carry out these low-risk epidemiological studies because of the difficulty of finding a truly nonexposed reference category. Cummings et al. (1990) point out that 91% of the nonsmokers they interviewed had measurable cotinine in their urine while only 76% reported they had been exposed to tobacco smoke in the previous four days. Eighty-four percent of those not living with a smoker had measurable cotinine. If these people are getting nicotine, known to be in the vapor phase of ETS, they must also be getting tar, now known also to be in the ETS vapor phase (Pritchard 1988). Miller (1989) has probably done the best job of ferreting out a nonexposed reference group with the result that he is finding very high relative risks for total cancer from passive smoking.

As Goldstein (1986) has said, "Chemicals shown to be carcinogenic are considered by regulators as 'guilty until proven innocent' of having no threshold. This conservative approach essentially puts the burden on the producer or user of providing the scientific evidence justifying a threshold in regulating a carcinogen." The purpose of my paper was to provide regulators with an estimate of the most probable death toll from passive smoking given the existing epidemiological evidence, and also data from which to calculate an upper bound estimate, as they usually wish to do. Nothing in Lee's comments, with his botched bias analysis and his flimsy dose model, does anything to "justify a threshold" for this known human carcinogen.

Katzenstein (1990) also appears to be very selective in the data that he reports in Table 1 of his letter and he does not appear to have done his homework in finding all the reports on passive smoking and lung cancer that have issued since the 1986 reports of the National Academy of Sciences (NRC 1986) and the Surgeon General (USSG 1986). Commenting first on the reports that he lists, Chan and Fung (1982) is simply a restatement of the more detailed data in Chan et al. (1979). I had rejected Chan et al. (1979) and Dalager et al. (1986) for reasons stated in my paper. Dalager's crude relative risk of 1.00 that Katzenstein reports is for both sexes. The only female all exposure relative risk in that paper is 1.96 for spouse exposure, not statistically significant. However among older women, 63 plus years of age, with high intensity exposure, the odds ratio was 5.14 with 95% confidence limits of 1.4 to 18.95. A dose response trend was also observed. Kabat et al. (1984) found a statistically significant odds ratio of 3.3 for male exposure at work and also found a statistically significant Mantel test for linear trend in the frequency of exposure (four levels) for males ($p < 0.005$). Garfinkel et al. (1985) had a statistically significant odds ratio of 2.0 at the highest exposure. The results that Katzenstein quotes from Gao et al. (1987) are for never smoking women who ever lived with a smoker. For spouse exposure they report a rising relative risk from 1.0 for less than twenty years exposure to a statistically significant 1.7 for forty

plus years exposure. Shimizu et al. (1988), besides reporting the 1.1 nonsignificant risk for nonsmoking wives exposed to a husband's smoke also report a 4.0 significant risk for exposure to a mother's smoking and 3.2 for exposure to the husband's father's smoking. The latter is not unusual since wives in Japan, after they leave their mother's home, often live with the husband's family and the husband's father is often retired. Wu et al. (1985), Brownson et al. (1987), Humble et al. (1987), and Lam et al. (1987) were covered in my paper (Wells 1988a). The male relative risk in Humble et al. (private communication) is a statistically significant 4.2. New reports that Katzenstein evidently is not aware of are (1) the Hong Kong thesis of W. K. Lam (1985) with 60 female cases and a statistically significant relative risk of 2.01 and a risk for peripheral tumors of 2.64 ($p < 0.05$); (2) Geng et al. (1988) with 54 cases and a statistically significant odds ratio of 2.16 for all levels of exposure, and 2.76 with 95% confidence limits of 1.85 to 4.10 for exposure to 20 plus cigarettes per day. They also report a relative risk from ETS for smoking wives of 1.88; (3) Inoue and Hirayama (1988) with 22 cases report a nonsignificant odds ratio of 2.25 for all exposure levels, but for exposure to 20 plus cigarettes a day the odds ratio is a statistically significant 3.35 (they also report a statistically significant positive trend); (4) Svensson (1988), in a thesis from Sweden, with 34 female nonsmoking lung cancer cases, found a relative risk of 1.2 for exposure at home or at work and a relative risk of 2.1 for exposure at home and at work. He also found a relative risk of 1.4 for exposure as a child or as an adult and 1.9 for exposure both as a child and as an adult. None of Svensson's relative risks is statistically significant; and (5) Varela (1987) also in a thesis, this one from Yale University, reports on 218 female cases and 221 male cases which included both never smokers and long-term exsmokers. He found no increase in risk for spouse exposure or workplace exposure but found a statistically significant relative risk of 1.87 multiple exposures at home.

Katzenstein's attack on the underlying studies is a typical tobacco industry approach. As we know, all epidemiological studies are flawed to one extent or another. However the National Academy and the Surgeon General, looking at the totality of the studies then available, concluded that passive smoking can cause lung cancer, and inclusion of the studies new since 1986 would not change that conclusion.

Katzenstein is wrong when he says that the heart studies failed to consider cardiovascular risk factors. Garland et al. (1985) and Helsing et al. (1988) adjusted for several of them. The Svendsen study (1987) considered ten of the most frequently studied heart risk factors, comparing 286 nonsmoking men married to smokers and 959 married to nonsmokers. The differences were small, and adjusting for them did not decrease the observed risk. Katzenstein quotes an American Cancer Society 1988 release saying that currently available evidence is not sufficient to conclude that passive or involuntary smoking causes lung cancer in nonsmokers. He must have found this in the rare book store since neither the Delaware office nor the national office of the American Cancer Society could find this reference. On the contrary the ACS "Cancer Facts and Figures for 1989" states that involuntary smoking increased the risk of lung cancer, and their "The Smoke Around You" pamphlet issued in 1987 quotes the 35% increase in lung cancer risk for passive smoking that is found in the National Academy report (NRC 1986).

In Katzenstein's "final comment" where he quotes the NAS and USSG reports on passive smoking and heart disease, he fails to note that the best heart evidence is in papers issued since those reports came out. It is interesting that the newest reports (Palmer 1989; Hole 1989; Humble 1990) all support a positive relative risk.

Holcomb (1990) states that I had encouraged the view that the results in Wells (1988a) were new. Actually that paper has a long history. The original version was presented at a seminar at the Harvard School of Public Health in December, 1984. An update was presented to the National Research Council in January, 1986. The version Holcomb refers to was presented at the June, 1986, meeting of the Air Pollution Control Association, and in September, 1986, before the Natural Resources, Agriculture Research and Environment Subcommittee of the Committee on Science and Technology of the U.S. House of Representatives. It is published in the proceedings of those meetings (Wells 1986b, 1987). After extensive revision, a shortened version was presented at the 6th World Conference on Smoking and Health in Tokyo in November of 1987. A summary is published in the proceedings of that meeting (Wells 1988c). The first draft of the current version (Wells 1988a) contained a summary of this history, but the editors of *Environment International* decided that since none of the earlier versions had been adequately peer reviewed, reference to them could be omitted. It should be noted that the current paper profited by the many comments received over the years from many experts in the field who either commented gratuitously or whose advice was solicited. James Repace was sur-

prised in 1986 at the large number of heart deaths and is probably still surprised, as are many others, but that is the way the numbers come out.

Holcomb states that I did not address the issue of causation. Perhaps this should have been done more explicitly in the paper. It was pointed out on the first page of the paper that the Surgeon General's report (USSG 1986) and the National Academy report (NRC 1986) both stated that passive smoking can cause lung cancer. I thought that was adequate coverage for that issue. (Incidentally Holcomb states that "the Surgeon General's report was alone in concluding that ETS causes lung cancer in nonsmokers," but on page 10 of the National Academy report it is stated, "Considering the evidence as a whole, exposure to ETS increases the incidence of lung cancer in nonsmokers.") Then I went on to show that the heart data, including the new data, had most of the same characteristics as the lung cancer data in terms of number of cases, statistical significance, dose response, and biological plausibility. Hence one could infer causation.

Holcomb references a paper by Koo et al. (1988) that allegedly shows that nonsmoking women married to ever smokers had a less healthy life style than nonsmoking women married to nonsmokers. Careful analysis of their voluminous data indicates eight life style factors where the test p and the p for trend were both reasonably small. Five indicated a healthier life style for the women married to the never smokers and three for those married to the smokers. About all this paper shows is that nonsmoking women in Hong Kong who lived in rural areas are more likely to be married to nonsmokers and to have a more rural life style. Humble et al. (1990), in their soon-to-be-published paper on passive smoking among never smoking women in Georgia, found that higher social status white women had a higher relative risk of heart disease from ETS than lower social status white women, quite the reverse from what Koo et al. concluded. Humble et al. also adjusted for age, diastolic blood pressure, total serum cholesterol, and body mass. The tobacco people have used misclassification as their principal smoke screen to discredit lung cancer risk from passive smoking. They know that misclassification can't possibly explain the heart effects of passive smoking so they have embraced "life style". This also is proving to be ephemeral.

Holcomb complains that I included unpublished studies in the analysis, but Katzenstein complains that publication bias is likely to omit pertinent data. I chose to include all the data I knew about, favorable or unfavorable. Omission of the unpublished studies would not change the conclusions.

Holcomb states that I based my exposure estimates on data published by Freidman et al. (1983). Actually, the exposure of never smokers living with ever smokers was obtained from the exposure of controls reported in the various U.S. passive smoking studies. This represents the major factor in female exposure. Only the exposure of nonsmokers living with nonsmokers was estimated using Friedman et al.

There is no question that my conclusions on heart disease and cancers other than lung go further than the cautious statements in the Surgeon General and National Academy of Science reports. So far, however, the new data support my position. Whether causation has been "proved" or not, public health officials need to know the mortality stakes involved. They can then make their own judgments as to the likelihood of causality.

Holcomb has not read the paper of Repace and Lowrey (1985) carefully. Their estimate of 4665 lung cancer deaths from passive smoking is based on a comparison of lung cancer mortality rates of Seventh Day Adventists who never smoked with those of non-Seventh Day Adventists who never smoked, not on exposure estimates as Holcomb claims. My estimates in no way rely on the exposure estimates of Repace and Lowrey. There are some nine studies in the literature that estimate lung cancer deaths from passive smoking. Except for Arundel et al. (1987) estimate, they range from 600 to 5600. The Arundel estimate is based on extrapolation from smokers to nonsmokers of retained particulate dose, an idea discredited earlier in this letter.

As Katzenstein says, death from passive smoking is a serious issue, serious to the health of the tobacco industry, and serious to the public health. We can expect vigorous (but misleading) attacks from the tobacco side, as these three letters show, but it is still best to lean toward safety when the health of the public is at stake.

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BOOK REVIEWS

The Risk Professionals, by Thomas M. Dietz and Robert W. Rycroft, Russell Sage Foundation, New York, NY, 1988; (161 pp., US$8.95).

The complex science and number of actors involved in today's environmental risk problems confound and mystify the process of environmental policy making for the majority of concerned observers. The authors of *The Risk Professionals* attempt to demystify the process of how policies for managing environmental risks are formulated by characterizing and critically dissecting the principal actors involved in the process.

The authors conducted face-to-face and telephone interviews with over 200 environmental risk professionals in the Washington, D.C. area. The interviews were built around a carefully selected combination of questions replicated from prior studies and new questions and themes . The resulting interview guide and the interviewing methodology (which are presented in the book) facilitated the development of a "natural history" of risk professionals which contained information with which hypotheses about their perspectives and values, and how they impact the policy making process, could be tested. The risk professionals were asked questions which explored why they thought that environmental policy debates are often so heated "How important are public mistrust of industry, and misunderstanding and fear on the part of the public as sources of this controversy?"; "Would arranging mediation or other conflict resolution procedures outside of standard legal or regulatory procedures be effective in reducing the controversy?". Other questions focused on the factors involved in disputes between experts "How often do these disputes involve ambiguities which result in different assumptions and judgments?"; "How frequently are they due to polarization resulting from difference in politics, paradigms, or position?"; "How useful would it be to have technical debates resolved through a peer-review process?". The risk professionals also were asked about their views on the methodologies and techniques used in risk policy making "Do you favor or oppose the use of formal risk analysis or benefit-cost analysis in environmental policy making?"; "How do you feel about the use of short-term, high-dose animal studies as a basis for environmental regulation?". Their values and opinions were characterized by noting their responses to statements such as: "Society has only perceived the tip of the iceberg with regard to the risks associated with modern technology"; "A high level of public involvement often leads to bad policy decisions"; and "Scientific information is often used to justify decisions made on political grounds". The affiliations, academic training, age, and philosophies of the risk professionals also were surveyed.

Using the answers to these questions, the authors systematically tested various hypotheses about the distinguishing characteristics of different types of risk professionals (e.g., regulators, industry, academics) with the goal of better explaining the environmental policy making process. The authors first examined the viewpoint that the risk professionals function as a network of elitists, sharing similar educational backgrounds, gender, race, age, generation, employment, and affiliations. Although exhibiting a few of the characteristics suggested by the elite perspective (e.g., similar race, gender, level of education), the risk professionals were found to not share the common backgrounds and organizational links that have been shown to create solidarity in other, more classic elite structures. A great deal of diversity among the risk professionals was observed in many key areas. Secondly, the viewpoint that the risk professionals constitute a "new class" of environmentalists was critically examined. Initially, the authors postulated and refined various definitions of the new environmentalist, finally arriving at a working definition. The basic philosophies of the respondents toward various components of environmental policy — the economy, politics, the resilience of nature, and our state of knowledge — were then analyzed in the context of this definition. The risk professionals showed considerable support for the new environmental paradigm described by the authors, which was based on employment, class, ideology, and social movement. Place of employment was highlighted as the strongest determinant of the risk professional's world view. The authors also explored the characterization of risk professionals as an "establishment". Based on tests of this perspective, the authors uncover and describe

a complex mixture of agreement and disagreement on the ways risk policy should be formulated, on the reasons for problems in the process, and on the kinds of techniques and evidence that should be used in developing and implementing policy. Finally, the perspective that the risk professionals constitute a "policy community" in which professional contacts and cross employment are central forces was examined. The networked structure of the risk policy community was nicely described, in which intra-group channels of communication were highlighted and the perceived roles of one group in the policy process by the others were examined on the basis of frequency and type of contact.

Readers with an interest in the development and methodology of the study will find this series of examinations of great interest. The difficulties in framing complex generalized hypotheses and interpreting tests in which clear-cut categorizations and distinctions rarely exist are in evidence. The authors' efforts in this regard are noteworthy. Their style of presentation of results seems at points to be very "shotgun", covering a number of elements with at times insufficient attention paid to keeping a reader with a different background focused on the main issue. However, the initial chapters of this book can in one view be taken as a collection of the authors' findings which the reader can interpret and integrate on his or her own.

Those seeking only the authors' "bottom line" and its implications to environmental policy making may opt to focus more heavily on the conclusions in Chapter 6. General themes supported by the findings and their implications for environmental policy making are presented. The risk policy system was found to be very open, with few institutional or ideological barriers to entry. The hybrid character of the risk policy system, where issues are based on science but must be solved in a context of sharp ideological or political differences, was found to create inherent contradictions which complicated the work of individual risk professionals and the workings of the overall system. While science is generally central to these issues, many risk policy problems are "transscientific". Application of scientific tools and techniques to these transscientific problems was indicated to sometimes create additional conflict and misunderstandings. The chapter presents a fairly tightly written summary of the study which remains focused on the hypotheses tested, without degenerating into a general philosophical discussion. Readers may find it useful to read the "Conclusions" chapter before the series of chapters addressing the various tested perspectives, and should take care not to overlook the collection of sources cited in the "Notes", as they may be enticed to do a little follow-up reading on certain topics.

As the authors depict, environmental risk policy making is inherently interdisciplinary and hybrid in nature. This leads to contradictions on many aspects associated with risk management. Similarly, one can not expect a definitive classification and description of a "risk professional". There is no such thing. The book, however, goes a long way to present the commonalities among risk professionals that were supported by the authors' survey and discount some commonly held generalizations that were not found to be robust. A reader of this book will develop a better understanding of the risk policy system and the risk professional who works in that system to engineer environmental policy.

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Environmental Management Handbook: Toxic Chemical Materials and Wastes. L.C. Kokoszka and J.W. Flood, 1989. (656 pp., bound, illustrated. $125 for U.S. and Canada, $150 for all other countries).

Toxic chemicals, as the preface of the book claims, will continue to be a major concern for some time to come. This handbook treats the subject of toxic regulation and actual practices in handling, treatment, and disposal. It comes at a time when regulatory controls have advanced, and several new ways of managing toxic chemicals have also been attempted.

Several references are provided at the end of each of the 13 chapters. A list of other references available for most chapters may be consulted for an even broader level of knowledge about the subject concerned. A list of U.S. Environmental Protection Agency (EPA) documents in solid waste management and Superfund programs is provided as one of the 16 appendices at the end of the handbook.

The goals of laws enacted in the U.S., particularly the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA), and existing regulatory programs are concisely explained. Toxic chemicals listed in the tables are identified by chemical abstract registry number. Useful addresses of vendors of toxic waste technologies and regional regulatory offices in the U.S. are also provided. Despite the abundance of tables and figures, a listing of

these is not provided, but the index may be used instead.

The chapter on sampling and analysis test methods stands out with relevant information concerning air, water, and solid waste, and a separate list of standard procedures for the analysis of PCBs (which appears to be author Kokoszka's area of interest). An idea about industrial compliance can be gleaned from a table that gives a description of how waste minimization was achieved, as well as a few cost data. Treatment and disposal techniques presently practiced and under development are also compiled.

The handbook, in general, gives the user a summarized update about the state-of-the-art and leads him to sources on topics of direct concern. Future trends are also briefly discussed; one of the trends is the development of in situ methods and movable units that were initiated in response to the NIMBY (or "not in my backyard") syndrome.

The handbook should be very useful to environmental consultants and program managers in both private and government sectors. Environmental engineering students may also find this a good reference to consult on present practices and information sources in the U.S.

Antonio L. Fernandez
Osaka University
Osaka, Japan

Quality Assurance for Diagnostic Imaging Equipment. NCRP Report No. 99. National Council on Radiation Protection and Measurements, Bethesda, MD; 1988. 250 pp., $18.00.

This report addresses factors that influence production of an image. It contains the necessary information to enable the imaging physician to report the diagnostic findings to the referring physician. Some of these factors are human, others are inherent to technique and equipment. The subject of the report is quality assurance, i.e., all of the management practices instituted by the imaging physician, to assure highest quality medical care, but an essential element of quality assurance is quality control. The first three sections are concerned with quality assurance in general and the remainder with quality control in specific circumstances.

This report provides a systematic approach to procedures which can ensure that the physician and the imaging facility consistently achieve their optimal performance. This in turn, ensures that there is optimal use of radiation and that radiation exposures to patient and staff are maintained at a level consistent with the principles of optimization of radiation protection espoused by the NCRP.

Living with Radiation: The Risk, the Promise. Henry N. Wagner, Jr. and Linda E. Ketchum. The Johns Hopkins University Press, Baltimore, MD; 1989. 193 pp., $17.95

The authors present an illuminating account of the hopes and fears associated with ionizing radiation, extending from nuclear energy and medical radiation to nuclear weapons. They make it clear that a justified fear of nuclear weapons has led to a widespread, unjustified, and unreasoning fear of the beneficial applications of radiation. Although these two aspects of atomic energy are tied together — they both involve the nucleus of the atom and its radioactive rays — a deep misunderstanding of this relationship by the general public has evolved since the time of the atomic bombing of Hiroshima and Nagasaki. The authors' aim is to place the beneficial applications of nuclear radiation in perspective.

Early in the book the groundwork is laid in understandable language of the scientific principles involved, including the basic discoveries of radioactivity and nuclear science. This aids the reader in understanding the discussion of nuclear radiation, whose role is so pervasive but at the same time so generally misunderstood and so unreasonably feared. The inability to understand the value of a risk-benefit analysis leads to a failure to take sufficient advantage of the beneficial applications of radioactive tracers and other forms of radiation.

The Facts on File Dictionary of Physics. Revised and expanded edition. Facts On File Publications, New York; 1988. 235 pp., $19.95 hardbound.

This book is an important tool for students and those whose daily work involves physics. It includes definitions, 50 line drawings, extensive cross-references and tables listing symbols for physical quantities, conversion factors and other frequently consulted information.

BOOKS RECEIVED

Exploited Earth: Britain's Aid and the Environment. Teresa Hayter. Earthscan Publications, London, UK, 1989. 276 pp., £7.95 paper.

No Timber Without Trees, Sustainable Management in the Tropical Forest. Duncan Poore. Earthscan Publications, London, UK, 1989. 252 pp., £9.95 paper.

Poverty and the Environment: Reversing the Downward Spiral. Alan B. Durning. Worldwatch Paper 92. Worldwatch Institute, Washington, D.C., 1989. $4.00.

Water for Agriculture: Facing the Limits. Sandra Postel. Worldwatch Institute, Washington, D.C., 1989. $4.00.

Cypermethrin. Environmental Health Criteria 82. International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, 1989. SwFr. 16.-.

DDT and its Derivatives — Environmental Aspects. Environmental Health Criteria 83. International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, 1989. SwFr. 13.-.

2,4-Dichlorophenoxyacetic acid (2,4-D) — Environmental Aspects. Environmental Health Criteria 84. International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, 1989. SwFr. 12.-.

Lead — Environmental Aspects. Environmental Health Criteria 85. International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, 1989. SwFr. 13.-.

Mercury — Environmental Aspects. Environmental Health Criteria 86. International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, 1989. SwFr. 14.-.

Dangerous Premises. An Insider's View of OSHA Enforcement. Don J. Lofgren. Cornell University ILR Press, Ithaca, NY, 1989. 256 pp., cloth $32.00, paper $14.95.

Pollution in Lake Titicaca, Peru: Training, Research and Management. T.G. Northcote, P. Morales S., D.A. Levy and M.S. Greavan. Westwater Research Centre, University of British Columbia, Vancouver, Canada, and Instituto de Aguas Alto Andinas, Universidad Nacional del Altiplano, Puno, Peru, 1989. 259 pp., Can$20.00, paper.

For the Common Good. Redirecting the Economy Toward Community, the Environment, and a Sustainable Future. Herman E. Daly and John B. Cobb, Jr. Beacon Press, Boston, MA, 1990. 482 pages, $24.95, hardcover.

Environmental Science: A Framework for Decision Making. Daniel Chiras. Addison-Wesley Publishers, Ltd., Wokingham, UK, 1988. 530 pp., £34.95.

Mathematical Submodels in Water Quality Systems. S.E. Jørgensen and M.J. Gromiec, eds. Elsevier, Amsterdam, or in the USA/Canada, Elsevier Science Publishing Co., Inc. New York, NY, 1989. 408 pp., US$ 117.00/Dfl. 240.00.

Environmental Data Report. Second Edition. United Nations Environment Programme. Basil Blackwell, Oxford, UK, 1989. 550 pp., £45.

This Section contains abstracts and, where appropriate, illustrations of recently issued United States patents and published patent applications filed from over 30 countries under the Patent Cooperation Treaty. This information was obtained from recent additions to the Pergamon PATSEARCH" online database in accordance with interest profiles developed by the Editors. Further information about Pergamon PATSEARCH" can be obtained from Pergamon Orbit InfoLine Inc., 8000 Westpark Drive, McLean, Virginia 22102 U.S.A.

Copies of complete patents announced in this Section are available from Pergamon Orbit InfoLine Inc. for $8 per copy. Payment with order is required. Orders outside North America add $2 for air postage. Order by patent number for Pergamon Orbit InfoLine only.

4783991

IGNITION AND COMBUSTION ENGINE PERFORMANCE MONITOR

Glenn H Wixon

A semiautomated diagnostic apparatus primarily used with ignition systems on internal combustion engines is novel in that every firing is examined for peak breakdown voltage level in a synchronized sequential manner to create a display of abnormalities and sequence number identification. Normal conditions can also be displayed in sequence when desired. A variable input permits examining relative peak voltages for further user analysis. An acoustic alert is provided as is an RPM indication and novel realtime indication of spark advance angle. The system operates at any engine speed.

4785573

PORTABLE ELECTRONIC INSECT CONTROLLER

Lance L Millard

A portable camping lantern includes an electrified grid including two half sections embracing the light unit and having indexing structure cooperating with a valve control on the lantern which functions to limit how the grid is placed on the lantern such that a safety interconnect extending from the grid to the battery below the capped refuel opening extends thereacross preventing the refueling of the lantern without disconnecting the power supply from the grid. Insect-attracting odor is sprayed across the grid upwardly from a manifold extending around the lantern and is carried by a carbon dioxide propellant. An insect-attracting sound-emitting unit is positioned closely adjacent to the grid for attracting insects to the grid. An appropriately colored glass enclosure extends around the lantern light to attract insects to the grid.

4785748

METHOD SUDDEN EXPANSION (SUE) INCINERATOR FOR DESTROYING HAZARDOUS MATERIALS & WASTES

Mark L Sujata, Thomas D Burnette, H Clyde Long, Raymond E Wieveg assigned to The Marquardt Company

The method of incinerating hazardous materials and fluidizable wastes, such as liquids, gases, entrained solid particles, fumes and slurries, utilizes an incinerator in the form of a sudden expansion burner. The incinerator has a relatively small diameter cylindrical inlet pipe connected by a circular plate to a relatively larger diameter elongated cylindrical combustion chamber. A waste injection line passes into the incinerator adjacent the inlet pipe for transfer of incineratable waste therethrough and into the upstream end of the combustion chamber. Air inlets connected to a blower also terminate adjacent the inlet pipe for supplying air at a high flow rate to the combustion chamber. One or more fuel nozzles extend through the plate into the combustion chamber to provide an overstoichiometric concentration of fuel adjacent the plate. The fuel is ignited through an electrically powered, fuel supplied ignitor extending into the combustion chamber through the inlet pipe. The supply of fuel, waste, air and igniter fuel are monitored. Total combustion of hazardous waste materials is carried very rapidly out by utilizing the present method and incinerator.

4788936

METHODS FOR BEDDING LIVESTOCK AND TREATING SLUDGE

Lanny D Billings

A method of bedding livestock includes placing an Aspen bark-based pellet product on the floor of a livestock confinement area, arranging the pellets in a layer of a generally uniform depth and housing livestock in the area on the layer of pellets. A sludge treatment method includes mixing the Aspen bark-based pellet product with sludge in a ratio of 1 part pellet to up to 40 parts sludge by weight in composting the mixed pellets and sludge to form a composted product. The Aspen bark-based pellets include between 5 and 45% Aspen bark, the balance including Aspen sawdust, ground wood chips and ground lumber.

4789387

DUST COLLECTOR

Stephen A Nemesi, William Klimczak assigned to Venturedyne Ltd

The invention provides a filter apparatus for use in air pollution control which includes at least one hollow filter assembly disposed in a hopper and having an open upper end registering with a wall opening. Contaminated air is introduced into the hopper through an inlet and is caused to flow into an exhaust chamber through the filter and the opening. The filter apparatus includes an end plate supporting the filter assembly and sealing the lower end of the filter assembly. The filter apparatus also includes a nozzle spaced above the filter assembly for selectively directing a reverse pulse of air into the open upper end of the filter such that the reverse air pulse enters the filter assembly and flows through the filter medium in the reverse of the direction of contaminated airflow to thereby remove accumulated contaminants from the filter medium. The nozzle is constructed such that the air pulses emitted from the nozzle propagate toward the wall opening in a cone-shaped pattern with the base of the cone engaging the wall at the wall opening and surrounding the open end of the filter, and such that the air pulses propagate downwardly into the filter assembly and impinge directly upon the end plate and do not impinge upon the filter medium.

4791688

JET PUMP MACERATOR PUMP SEWAGE HANDLING SYSTEM

C K Krishnakumar, P Saigh assigned to Chamberlain Manufacturing Corporation

A jet pump macerator pump sewage handling system which provides a unique waste control system for use in mobile units such as passenger trains and ships in which multiple toilets are connected to a transfer manifold pipe which communicates with a non-clogging jet pump which is formed with a venturi and through which liquid material is driven by a macerator pump. Waste material from the toilets is drawn by the jet pump through the transfer manifold pipe and discharged into the retention tank. The macerator pump withdraws material from the retention tank, grinds it and discharges it back into the retention tank. The macerator pump has a self-cleaning feature in that it reverses and cleans itself whenever a loss in discharge pressure due to excessive solids loading is sensed. The transfer manifold pipe is virtually free from clogging due to the action of an air induction valve which keeps waste material in the manifold pipe moving without allowing it to accumulate.

4795607

HIGH-TEMPERATURE REACTOR

Herbert Reutler, Gunter Lohnert, Johannes Lukaszewicz, Kfederal Republic Of Germana assigned to GHT Gesellschaft fur Hochtemperaturreaktor-Technik mbH

Gas-cooled high-temperature nuclear reactor having a reactor core comprising individual fuel elements provided with means for forming a barrier against the release of fission products producible therein during reactor operation, the fuel elements being received in a cylindrical barrel formed of an inner graphite layer functioning as a reflector, an outer layer of insulating material surrounding the inner layer, and a metallic receptacle, the inner and outer layers and the receptacle being formed of respective side, bottom and cover portions, the side and cover portions of the inner layer being formed with first channels into which means for controlling the reactors are insertable, the bottom, side and cover portions of the inner layer being further formed with second channels wherein, during reactor operation, cooling gas is circulated under pressure from the bottom to the top of the recep-

tacle, the bottom portion of the inner layer having first openings for introducing cooling gas into the second channels during reactor operation and second openings for withdrawing during reactor operation cooling gas heated by passage through the reactor core; the inner and outer layers and the cylindrical core barrel having a heat conductivity and a thermal capacity and the reactor core having such a size, shape, power density and moderation ratio that a first temperature at which the core becomes subcritical for all possible accident conditions is below a second temperature at which the barrier means are destroyed, and, when loss of pressure of the cooling gas is experienced, after-heat generated in the core being removable by heat conduction and radiation through the inner and outer layers and the core barrel to a heat sink located outside the receptacle, in such a way that the fuel elements remain at a temperature below the second temperature.

4796466

SYSTEM FOR MONITORING PIPELINES

Ed Farmer

A system for monitoring pipelines through which fluids, be they gases or liquids flow using conventional readily available monitoring equipment, that determines the probability of a leak as opposed to the actuality of a leak using a moving average of statistical information gained from a plurality of monitoring stations that monitor either pressure or flow.

4797089

SYSTEM CONTROL MEANS TO PREHEAT WASTE OIL FOR COMBUSTION

Gary Schubach, Frank Schubach

An apparatus and method for a system control means to control the temperature and preheat of waste oil in a waste oil feed system and burner. The apparatus and method include the combination of a heat transfer assembly with a helical passageway through a preheat means, a system control means, an anticipatory rate-proportional band temperature control means, and an expansion pressure relief means.

4797156

METAL TREATED BY A SOLUTION CONTAINING FERRIC FERROUS SALT

Shoji Yamashita, Aichi, Japan assigned to I B E Co Ltd

A compound containing ferric ferrous salt and a producing method thereof are provided in the present invention. Said compound comprises ferric ferrous salt and a salt of alkaline metals or a compound containing a metal which belongs to zinc family, and said producing method of said compound comprises adding ferric ferrous salt into an aqueous solution of strong acid and then adding a salt of alkaline metals or a compound containing a metal which belongs to zinc family. Said compound containing ferric ferrous salt may be very useful in a wide variety of fields, such as water cleaning, keeping freshness of vegetation, antisepsis, antifungi, antibacteria, rust preventing, effluent treatment, soil improvement, ionization control, feed enriching, petroleum improvement, antistatic technique, and the like.

4797212

BIOLOGICAL PURIFICATION OF WASTE WATERS

Reinhart von Nordenskjold, 8011 Solalinden, Federal Republic Of Germany

In the particular embodiments of the waste water treatment system described in the specification, a waste water treatment basin has spaced-apart, moving aerator chains extending across the basin in a direction transverse to the flow of waste water through the basin. When an aerator chain is provided with air, bottom aerators suspended from the chain adjacent to the bottom of the basin move with respect to the basin to mix sludge collected on the basin with the waste water and to supply air to the waste water. A control system supplies air selectively to the aerator chains to provide zones of full aeration and intermediate zones with little or no aeration and the selection of the aerator chains is changed periodically in a progressive manner along the basin so that the zones move progressively through the basin. As a result, sufficient air is supplied to the waste water in the basin for purification of the waste water and to maintain sludge in suspension as required in the waste water at minimum expense of energy. In addi-

tion, the oxygen potential of the waste water in the zones is controlled so that successive reactions relating to the purification process are accomplished in successive zones, for example, including nitrification of the waste water in zones having a relatively high oxygen potential and denitrification in adjacent zones having relatively low oxygen potential. Phosphorus removal is accomplished in a similar manner.

4797367

AEROBIC COMPOSTING APPARATUS

John A Pinder, Scarborough, Ontario, Canada

The invention concerns an apparatus including an air diffuser device and process useful in the domestic scale aerobic composting of organic wastes. The air diffuser has within its air admitting opening means to exclude insects, small animal pests and to also exclude waste from entering the air diffuser. Containers may be employed which have only a single opening admitting access to the contents of the container and the side walls of the container need not have apertures. The air diffuser device may be used with a variety of types of containers and particularly with plastic bags. By using containers which do not have openings to the exterior, except for the opening in which the air diffuser is located and possible small liquid drainage openings, composting may be undertaken with control of pests and flies in regions near residences and without excessive drying of the edges of the compost mass, in a way not previously known.

4797804

HIGH DENSITY, HIGH PERFORMANCE, SINGLE EVENT UPSET IMMUNE DATA STORAGE CELL

Leonard R Rockett assigned to International Business Machines Corporation

The data cell invention disclosed herein is a CMOS latch having a first CMOS inverter and a second CMOS inverter which have their respective storage nodes interconnected by cross-coupling connections which each include a gated polysilicon resistor. The respective storage nodes of the cell are connected through word line transfer gates to bit lines which serve to both write in and read out the state of the cell. The control gate of the word line transistors is also connected to the control gates of the respective gated polysilicon resistors in the cross-coupled connections for the cell. In normal operation, when the word line transfer gates are not conductive, the gated polysilicon resistors are also not conductive. When it is desired to write information into the cell during normal conditions, when the word line transfer gates are made conductive, the gated polysilicon resistors are also made conductive, thereby offering a minimum resistance to charge which is to be transferred between the respective storage nodes of the cell. However, during a single event upset condition, when one or more of the sensitive regions of the cell undergo an abrupt charge transfer due to the presence of cosmic rays or other ionizing radiation phenomena, since the gated polysilicon resistors are in their high resistance state, they impede the flow of any charges between the respective storage nodes of the cell. In this manner, after the single event upset has dissipated, the cell will have retained its original stored binary state. Thus, an enhanced resistance to single event upset conditions is provided by the invention while not imposing significant reductions in the speed of operation during normal conditions.

4797840

INFRARED ELECTRONIC THERMOMETER AND METHOD FOR MEASURING TEMPERATURE

Jaco Fraden assigned to Thermoscan Inc

An electronic infrared thermometer is disclosed comprising a housing forming an interior chamber, a pyroelectric sensor mounted within the chamber for sensing temperature change and generating an indicative electrical signal, means for directing infrared radiation from the object to be measured to the pyroelectric sensor, a shutter assembly for controlling the passing of infrared radiation to the pyroelectric sensor, an ambient temperature sensor for sensing ambient temperature within the interior chamber and generating an electrical signal indicative thereof, an electrical circuit for processing the electrical signals to calculate the temperature of the object to be measured, and an indicator for indicating the calculated temperature. The process for measuring the temperature of an object is also disclosed comprising shielding the pyroelectric sensor from infrared radiation from exterior to

the thermometer housing, selectively exposing the pyroelectric sensor to infrared radiation substantially solely from the object to be measured to generate a first electrical signal related to the absolute temperature of the object to be measured, sensing the ambient temperature of the pyroelectric sensor and generating a second electrical signal proportional thereto, and electrically processing the first and second electrical signals to calculate the temperature of the object to be measured.

4799243

DIRECTIONAL PEOPLE COUNTING ARRANGEMENT

Bruce E Zepke assigned to Otis Elevator Company

An arrangement for detecting the passage of living beings through a surveillance region into and out of a controlled-access space includes at least one pyroelectric detector device that includes two detector elements which have active areas that are directly exposed to thermal radiation from the surveillance region and convert the thermal radiation energy received thereby into electrical signals with opposite polaritites. The detector elements are situated in succession in the direction of passage into the controlled-access space. The surveillance region is optically subdivided into two surveillance zones arranged in succession as considered in the passage direction, for instance by a mask that masks an intervening zone situated between the surveillance zones to prevent thermal radiation from the intervening zone from reaching either one of the detector elements. Thus, the thermal radiation energy from a living being passing through the surveillance region reaches initially only one and subsequently only the other of the detector elements when such living being is entering, and initially only the other and subsequently only the one detector element when such living being is leaving, the controlled-access space. The opposite polarity electrical signals are then evaluated on the basis of their then existing lead/lag relationship.

4799461

WASTE HEAT RECOVERY BOILER

Toshinori Shigenaka, Iwao Kusaka, Kure, Japan assigned to Babcock Hitachi Kabushiki Kaisha

A waste heat recovery boiler comprising heat exchange rate switching means for controlling the rate of heat exchange between an exhaust gas and feedwater in a heat exchanger by changing the state of the feedwater in accordance with the concentration of sulfur oxides in the exhaust gas, thereby maintaining the temperature at which low temperature corrosion due to the exhaust gas is prevented in a downstream portion of the heat exchanger in the direction in which the exhaust gas flows. Accordingly, it is possible to achieve the maximum efficiency of heat recovery since the phenomenon of steaming in the heat exchanger is eliminated irrespective of whether the kind of exhaust gas is a dirty gas or a clean gas.

4800886

SENSOR FOR MEASURING THE CONCENTRATION OF A GASEOUS COMPONENT IN A FLUID BY ABSORPTION

James R Nestor assigned to C R Bard Inc

Apparatus and methods are provided for determining the concentration of a gaseous component in a fluid. A solid body of a natural or synthetic high polymer, which is permeable to the gaseous component, is exposed to the fluid, the polymer is exposed to infrared radiation, and the infrared absorption by the gas in the polymer is measured. In the preferred embodiment, a sensor is provided for making in vivo measurements of the concentration of CO2 in the blood. The sensor includes an optical fiber which is non-permeable to CO2 and substantially transparent at the CO2 absorption wavelength range, and a solid body of polymeric material at the distal end of the fiber which is substantially transparent to the absorption wavelength range and permeable to CO2. An incident infrared signal is transmitted down the fiber, passes through the body, is reflected off the distal end of the body, and the intensity of the return signal is measured by a detector. The return signal is diminished in proportion to the concentration of the CO2 in the polymeric body. The sensor is disposed within a catheter and is positionable within the narrow blood vessels of the body for continuous real time monitoring of the carbon dioxide concentration of the blood.

4801312

LAMINAR AIR FLOW HAZARDOUS MATERIALS ABATEMENT METHOD AND SYSTEM

Mark E Mateson

A work space for removing hazardous materials within an occupied building is made safe both for the workers working within the work space and others outside the work space by defining a decontamination space having a plurality of rooms and air locks between the rooms and a work space opening on the decontamination space, supplying fresh air to the work space through a duct in the wall of the work space, supplying fresh air to each of the rooms and air locks and evacuating air from each of the rooms and the air locks and forcing the evacuated air through the same work space wall to provide a substantially laminar air flow from the wall. A row of air filtration devices normal to the air flow filters the air flow and expels the air in the same direction away from the wall. A second row of air filtration devices receives the previously filtered air, further filters it, and expels it into a duct leading to the outside of the building. The air pressure within the work space and the decontamination space may be independently controlled.

4801440

PARTIAL OXIDATION OF SULFUR-CONTAINING SOLID CARBONACEOUS FUEL

Mitri S Najjar, Roger J Corbeels assigned to Texaco Inc

A process for the simultaneous partial oxidation and desulfurization of sulfur and silicate-containing solid carbonaceous fuel for the production of gaseous mixtures comprising H2 and CO and containing less than about 0.05 volume % of H2S and COS. In the process, the solid carbonaceous fuel and a calcium-containing compound are reacted by partial oxidation in the reaction zone of a free-flow unobstructed gas generator with a controlled amount of free-oxygen containing gas and a temperature moderator so that an equilibrium oxygen concentration is provided in the gas phase in the reaction zone having a partial pressure which is less than about 10-12 atmospheres. The total moles of calcium in the reaction zone is at least equal to about 1.0 times the moles of sulfur in the solid carbonaceous fuel. The partial oxidation and desulfurization reactions take place simultaneously at a temperature which is about 10 degrees to 200 degrees F. below the softening temperature of the ash in the solid carbonaceous fuel and below about 2000 degrees F. at an increased gasification efficiency. The sulfur in the solid carbonaceous fuel in the reaction zone is converted into calcium sulfide particulate matter which leaves the reaction zone along with the fly-ash entrained in the hot raw effluent gas stream.

4801551

RUGGED DISSOLVED CARBON DIOXIDE MONITOR FOR HIGH PURITY WATER

William A Byers, Gerald L Carlson, James C Bellows assigned to Westinghouse Electric Corp

A continuous on-line monitor of carbon dioxide dissolved in high purity water includes a cation conductivity cell and associated bridge for measuring first and second cation conductivities at first and second temperature and a heater for heating the sample from the first to the second temperature. A microcomputer calculates first and second concentrations of a fully dissociated acid alone and carbon dioxide alone, respectively, which will produce the first measured cation conductivity at the first temperature and then determines the actual carbon dioxide concentration from the first and second concentrations and the second measured cation conductivity.

4802899

PRESSURE SWING ADSORPTION APPARATUS

Edward E Vrana, Ravinder Bansal assigned to AirSep Corporation

An air separator for separating at least one constituent element from air delivered from an air source by a pair of adsorption beds utilizes a control box and assembly of working components arranged in such a relationship to one another within the control box so as to facilitate service and maintenance of the air separator. The working components of the air separator include filtering componentry, a pressure regulator, a pair of feed valves, a pair of waste valves, a pair of product and equalization valves and a product

flow controller operatively connected in flow communication with one another for routing the source air through the separator and which are arranged in a substantially vertical plane within the control box. Because the working components are arranged in a generally planar arrangement, each of the working components is readily and easily accessible.

4804845

LUMINESCENCE DETECTING DEVICES

Kenichi Takeuchi, Tokyo, Japan assigned to Sony Corporation

A luminescence detecting device including a luminescence sensitive member made of a transparent resin or the like and containing at least two different kinds of organic phosphors dispersed therein. The first organic phosphor is operative to absorb fluorescence from incident radiation such as the index phosphor stripes from a reflex color cathode ray tube, and has a response spectrum which overlaps with the wavelength range of the incident fluorescence. The first phosphor is designed to emit fluorescence in a higher wavelength range upon excitation by the incident fluorescent radiation. The second phosphor is responsive to the fluorescence emitted by the first phosphor since the spectra of the two phosphors overlap. The second type of organic phosphor emits its own range of fluorescence in a particular wave band and can be detected by a luminescence sensing portion of the device to provide a signal which can be used to control the operation of the cathode ray tube.

4805445

PROCESS FOR MONITORING THE LEAKTIGHTNESS OF A STORAGE FACILITY

Jacques Grappe, Paris, France assigned to Societe Francaise de Stockage Geologique-Geostock

A storage device has an inner enclosure (6) and a leakproof and a rigid outer enclosure (5), which are mounted one inside the other and separated by an intermediate space (9) containing a fluid, continuous in phase at pressures other than that of the material stored, and pressure measurement equipment (16, 15) enabling the differences in the pressure of the fluid to be monitored. Advantageously, the device additionally features sampling equipment (18, 17) enabling the composition of the fluid to be monitored. The device may be buried, the outer enclosure being then placed against the cement walls (4) of a cavity, which may be a drilled well. The inner enclosure may feature a leakproof skin which is made rigid by the presence in the intermediate space of a porous and rigid filling material in which the fluid can circulate.

4805630

PRESSURE MONITORING DEVICES

Philip A Storey, Hertfordshire, United Kingdom assigned to The BOC Group Inc

A probe for monitoring the blood pressure of a patient includes a light reflective diaphragm 4 deformable in response to the blood pressure. A single emitter light guide 10 is arranged off-set from the central axis of the diaphragm 4 and directs light at an off-axis region of the diaphragm. A pair of receiver light guides 8, 12 are arranged one on each side of the emitter light guide 10 so that the axes of the light guides lie in or adjacent the same plane as the center of the diaphragm 4. Each receiver light guide 8, 12 receives light reflected from the diaphragm 4 in accordance with movement of the diaphragm 4.

4806320

PROCESS FOR NOX CONTROL

Sidney G Nelson assigned to Sanitech Inc

A process is described for reducing the levels of nitrogen oxides present in flue gases from stationary sources. The process consists of mixing ammonia or methane with the flue gas and then passing the mixtures through a bed of expanded vermiculite, the latter serving as a catalyst for reactions between the nitorgen oxides and ammonia or methane. The reactions result in the formation of nitrogen and water and/or carbon dioxide.

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EDITORIAL:
SOFTWARE SURVEY SECTION

The purpose of the Software Survey Section in ENVIRONMENT INTERNATIONAL is to encourage the open exchange of information on software programs unique to our professional field. With the rapid penetration of computers into academic and industrial institutions has come a parallel increase in the number of scientists and researchers designing their own software. The existence of much of this software remains unknown to even those of us who could most benefit from its use. We believe that it is of vital importance to our readers that such information be made available. We believe also that a professional journal is the best place to share such information. Your contribution would be most welcome.

The questionnaire on the following pages is designed to assist you in reporting on software that you may have developed or be in the process of developing. By completing this form, your information will reach thousands of your colleagues who may benefit from your work and may possibly offer suggestions for further enhancements to your software. Please complete the enclosed form and return it to:

Barbara Moghissi
Environment International
P.O. Box 7166
Alexandria, VA 22307

We do not intend to review or comment on the contents of the questionnaire. It will be published as is in order to expedite the information cycle process. I would welcome any comments you may have.

JOURNAL NAME ENVIRONMENT INTERNATIONAL

PERGAMON PRESS
SOFTWARE DESCRIPTION FORM

Title of software program: _____

_____

Type of program: [ ] Application [ ] Utility [ ] Other_____

Category: _____ (ie. Psychological assessment, statistics, thermodynamics, etc.)

Developed for (name of computer/s): _____

in (language/s):_____

to run under (operating system): _____

available on: [ ] Floppy disk/diskette. Specify:

Size _____ Density _____ [ ] Single-sided [ ] Dual-sided

[ ] Magnetic tape. Specify:

Size _____ Density _____ Character set _____

Hardware required: _____

Memory required: _____ User training required: [ ] Yes [ ] No

Documentation: [ ] None [ ] Minimal [ ] Self-documenting
[ ]Extensive external documentation

Source code available: [ ] Yes [ ] No

Stage of development: [ ] Design complete [ ] Coding complete
[ ] Fully operational [ ] Collaboration welcomed

Is program in use? [ ] Yes How long? _____ How many sites? _____
[ ] No

Is the contributor available for user inquiries: [ ] Yes [ ] No

Distributed by: _____

Cost of program: _____

Demonstration disk available? [ ] Yes Cost: _____
[ ] No

(continued)

RETURN COMPLETED FORM TO:

Ms. Barbara Moghissi
Environment International
P.O. Box 7166
Alexandria, VA 22307

Description of what software does [maximum: 200 words]:

Potential users: _____

Field/s of interest: _____

# # # # # # #

Name of contributor: _____

Institution: _____

Address: _____

_____

Telephone number:_____

# # # # # # #

Reference No. [Assigned by Journal Editor] _____

- -

[The information below is not for publication.]

Would you like to have your program:

Reviewed? [ ] Yes [ ] No [ ] Not at this time

Marketed and distributed? [ ] Yes [ ] No [ ] Not at this time

REMOTE SENSING OF ATMOSPHERE AND OCEANS

Proceedings of Symposium 1 and of the Topical Meeting of the COSPAR Interdisciplinary Scientific Commission A (Meetings A1, A2) of the COSPAR Twenty-seventh Plenary Meeting held in Espoo, Finland, 18-29 July 1988

Edited by **E RASCHKE,** *Universität zu Köln, D-5000 Köln 41, FRG;*
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P MCCORMICK, *NASA Headquarters, Washington DC 20546, USA* and
A GRUBER, *NOAA/NESDIS, E/RAII, Washington DC 20233, USA* et al.

The 63 papers presented highlight the status of space-based measurements techniques for the Global Change Programme. Observations from space of atmospheric, oceanic and continental surface properties now form the basis of any global research programme which is concerned with changes in our environment. Particular reference is made to the use of satellite data in both meteorology and oceanography and also the studies of mesoscale phenomena in the atmosphere.

For oceanographers, atmospheric physicists and meteorologists.

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ENVIRONMENT INTERNATIONAL

Aims and Scope

Environment International, published bimonthly, is a multidisciplinary forum for the publication of original environmental literature. Vital data, causes of pollution, and methods for protection are all featured. Covering the entire field of environmental protection, *Environment International* includes contributions from the following areas:

- Concentration of elements and compounds, notably pollutants
- Release rates of pollutants from various sources
- Transport of pollutants in the environmental media
- Health and ecological effects of pollutants
- Control technologies
- Description and interpretation of laws, regulations, and standards
- Information which will contribute to the understanding of environmental behavior of pollutants or will promote environmental protection
- Public policy alternatives including legislation
- National and international recommendations and practices to help bring about a lasting improvement in environmental protection

From time to time *Environment International* will publish issues devoted exclusively to special topics, e.g., specific pollutants. These issues will discuss such problems as environmental concentrations, health and ecological effects, environmental kinetics, production and release rates, legislative and regulatory aspects, control technologies, and other pertinent data. Other special issues will be devoted to pollution problems in selected geographical areas. Authors and readers are encouraged to send in suggestions to the Editor-in-Chief recommending topics for these topical issues.

Information for Contributors

Since the journal will serve a multidisciplinary audience, authors are urged to write for nonspecialists. In particular, they are discouraged from using expressions which are understandable only to a select audience. Clarity should be the guide when preparing manuscripts. All the contributions will be subjected to peer reviews. Owing to the broad coverage of *Environment International*, no single format can accommodate all contributions. Manuscripts must be submitted in English. Mail manuscripts to the Editor-in-Chief. *Environment International*, P.O. Box 7166, Alexandria, VA 22307, U.S.A. The following categories of contributions will be considered for publication in the journal:

Articles — Previously unpublished information including data from routine monitoring operations will be published in this category. Papers dealing with new analytical techniques should contain data indicating their usefulness in environmental measurements. Similarly, basic biological effects data should be related to environmental pollutants.

Discussions — This section is intended as a forum for the readers to express their views on environmental problems on a global or national basis. Letters to the editors, editorials, contributions containing personal opinions and comments on articles published in the journal or elsewhere will be included in this section.

Book Reviews — Solicited and unsolicited reviews on relevant books will be considered for inclusion in this section.

News — This section will contain newsworthy items in the scientific, technological, governmental and political areas relating to the environment. Readers are encouraged to suggest subjects for inclusion in this section.

Preparation of Manuscripts — The Journal is produced using a computer-based desktop publishing system. Accordingly, the processing time of manuscripts submitted in a computer-compatible form is greatly reduced.

Manuscripts should be typed double-spaced on one side of bond paper with wide margins. Each paper should include a title page with the title of the paper, name(s) of author(s), affiliation(s) and mailing address. In the case of co-authors, it should be clearly indicated which is to receive correspondence and proofs for correction.

All tables and figures must be camera-ready. Use a new ribbon for typing tables. Photocopies, blue ink or pencil are not acceptable. Use blue ink for lettering figures. The lettering should be large enough to be legible after the diagram has been reduced in size for printing. In the interest of conserving space, it is desirable that figures are drawn so they will reduce to the single column width of 8.5 cm (3.35 in). Type figure legends double-spaced on a separate sheet of paper. Write article title and figure number lightly in pencil on the back of each piece of artwork.

Submissions in a computer-compatible form require a disk, one original copy on paper containing the original figures and tables, and a second paper copy. The system requires compatibility with IBM computers and can accept 13.5 cm (5.25 in) and 9 cm (3.5 in) disks. The WordPerfect word processing system prepared with an IBM-compatible (MS-DOS or PC-DOS) computer is the preferred submission. If WordPerfect is unavailable, WordStar, MultiMate, Revisable-Form-Text (IBM DCA Format) can be used. If none of these is available, use ASCII (American Standard Code for Information Interchange) or DOS text file. Indicate which program was used. In the absence of a computer, one original and two copies are required.

References: The Journal generally follows the referencing style of the *Council of Biological Editors*. In the text, refer to the author(s) and the year. Examples for one author, two authors and more than two authors are: Wolf (1984); Repace and Lowrey (1985); and Smith et al. (1979). If there is more than one listing of the same author and year, use a, b, c, etc. Prepare a list in alphabetical order containing all literature cited. In this list of references, include initials and surnames of the authors, title of the paper, name of the journal using the *World List of Scientific Periodicals* abbreviations, volume number and page number(s). Note that et al. is not acceptable in the reference list unless the publication has more than six authors. The following examples should be used as a guide.

***Article*:** Repace, J. L.; Lowrey, A. H. A quantitative estimate of nonsmokers' lung cancer from passive smoking. Environ. Int. 11:3–22; 1985.
***Report*:** NIH (National Institutes of Health). Report of the National Institutes of Health ad hoc working group to develop radioepidemiological tables. NIH 85-2748. Washington, D.C.: U.S. Department of Health and Human Services; 1985.
***Book*:** Henderson, P. Inorganic geochemistry. New York, NY: Pergamon Press; 1982.
***Regulation*:** USEPA (U.S. Environmental Protection Agency). National primary drinking water regulations: fluoride. 40 CFR Parts 141, 142 and 143. Fed. Reg. 50:47142-48933; 1985.
***Proceedings*:** Swedjemark, G. A.; Mjönes, L. Exposure of the Swedish population to radon daughters. Berglund, B.; Lindvall, T.; Sundell, J., eds. Proc. 3rd international conference on indoor air quality and climate. Vol. 2. Stockholm: Swedish Council for Building Research; 1984:37–43.

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