ASSESSMENT OF RISKS POSED BY EXPOSURE TO LOW LEVELS OF ASBESTOS IN THE GENERAL ENVIRONMENT

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I. EXECUTIVE SUMMARY

Estimates of probable excess risks of lung cancer and mesothelioma are made for low levels of exposure to airborne asbestos under environmental conditions. This is done by extending to low doses the estimated risks observed for industrial workers who were exposed at substantially higher doses. An extensive review of the dose-response data for industrially exposed workers (extending an earlier review by Nicholson) leads to the conclusion that at an exposure of 1 fiber x years/ml the expected lifetime excess risk of lung cancer would lie in the following ranges:

	Men	Women
Smokers	10^{-3} to 10^{-2}	3×10^{-4} to 3×10^{-3}
Nonsmokers	10^{-4} to 10^{-3}	4×10^{-5} to 4×10^{-4}

There is some empirical evidence that these risks are not strongly dependent on the age when exposure starts.

For pleural and peritoneal mesotheliomas, the corresponding excess risk after half a lifetime's exposure (36-37 years) is estimated to be between 1 and 6×10^{-4}. This risk appears to be independent of smoking habits (and not clearly dependent on sex). However, it is strongly dependent on the elapsed time since first exposure.

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These estimates are derived from five independent studies which yielded reasonably consistent results. Two studies which yielded lower estimates of risk per unit exposure are discounted because of methodological deficiencies. Two other studies of workers exposed during mining and milling operations yielded much lower estimates of risk per unit exposure, perhaps indicating lower biological activity of asbestos at early stages in processing.

These risk estimates are extended to environmental exposure levels, using a number of assumptions about relationships between response, dose, age, and timing of exposure. There is considerable empirical and theoretical evidence to support the assumption of linear, nonthreshold relationships between dose and response, for both lung cancer and mesotheliomas. Data for mesotheliomas fit reasonably well to a "quadratic residence time" model, in which cumulative risks depend approximately on the third power of elapsed time since exposure. Response-time relationships for lung cancers are less consistent, so that environmental risk estimates are more uncertain.

Lifetime risk estimates for the general population are presented in Table 11 in Section V. For all categories except male cigarette smokers, lifetime risks for mesothelioma are predicted to be higher than those for lung cancer. For mesotheliomas, the most important period of exposure is expected to be the first 20 years of life, but most effects are expected after age 55. "Virtually safe" concentrations (i.e., exposure

levels corresponding to excess lifetime risks of 10^{-6}) are predicted to be in the range 10^{-5} to 10^{-4} fibers/ml for outdoor exposure, and 10^{-6} to 10^{-5} fibers/ml for indoor exposure. These concentrations are well below current detection limits. These estimates cannot be expressed reliably in units of ng/ml, because of wide variability in measured conversion factors.

These estimates are subject to considerable uncertainties. The 10-fold range in the estimates that are presented reflects the variability in the results of occupational studies, and probably reflects the variability in the properties of asbestos. However, the estimates of risk may be much too high to apply to asbestos at an early stage in processing. They would also be much too high if the assumption of a linear, nonthreshold dose-response relationship proves incorrect.

Risks of gastrointestinal and other cancers resulting from exposure to airborne asbestos have not been considered in detail in this report, but probably would add a small fraction (20-30%) to the estimates of excess risk that are presented.

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II. INTRODUCTION

This report has been prepared for the Institut für Wasser,- Boden- und Lufthygiene des Bundesgesundheitsantes. It presents estimates of health risks, specifically risks of cancer, posed to the general population by low concentrations of asbestos present in the general environment. This report is part of the development of health criteria for airborne asbestos, on which ambient standards may be based. Accordingly, we have developed estimates of "virtually safe" airborne concentrations of asbestos. These are the concentrations of asbestos which, if inhaled throughout life, would be expected to give rise to an excess risk of cancer of about 10^{-6} (1 in a million). Separate estimates are provided for indoor and outdoor exposure, the sole difference being the difference in the length of time which the average person spends indoors and outdoors.

The basis for the risk assessments presented in this report is the extensive information on excess frequency of cancers (primarily lung cancers and pleural and peritoneal mesotheliomas) observed in workers exposed to relatively high levels of asbestos in the workplace. These data have recently been reviewed by Nicholson (1981), who derived a number of estimates of dose-response relationships for lung cancer. In this report, we have extended Nicholson's review, primarily by deriving estimates of dose-response relationships for mesotheliomas, and by making

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a preliminary analysis of relationships between response, age, and elapsed time.

The results of these analyses are then used to derive estimates of risks likely to be experienced by the general population, exposed to much lower levels of asbestos from birth onwards. The extrapolation involves a number of assumptions about dose-time-response relationships. These assumptions are based on substantial empirical and theoretical evidence, but nevertheless introduce a number of uncertainties into the final estimates of general population risks. Uncertainties are also introduced by variability in the results of the epidemiological studies, which reflect variability in the properties of asbestos. The final estimates are presented to order of magnitude only.

In preparing this report, we have greatly benefited from the pioneering efforts of Dr. William Nicholson in constructing dose-response relationships. We also acknowledge helpful advice from Dr. Nicholson and from Dr. Charles C. Brown of the National Cancer Institute.

We emphasize that our analysis of dose-time-response relationships is preliminary, and that we have probably not extracted all the information that is available in published and unpublished studies. We recommend that a more formal and complete mathematical analysis of the available data should be conducted, to serve as the basis for improved estimates of general population risk.

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