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Dying for Work: The Magnitude of US Mortality From Selected Causes of Death Associated With Occupation

Kyle Steenland,[1,2]* Carol Burnett,[1] Nina Lalich,[1] Elizabeth Ward,[1,3] and Joseph Hurrell[1]

Background *Deaths due to occupational disease and injury place a heavy burden on society in terms of economic costs and human suffering.*
Methods *We estimate the annual deaths due to selected diseases for which an occupational association is reasonably well established and quantifiable, by calculation of attributable fractions (AFs), with full documentation; the deaths due to occupational injury are then added to derive an estimated number of annual deaths due to occupation.*
Results *Using 1997 US mortality data, the estimated annual burden of occupational disease mortality resulting from selected respiratory diseases, cancers, cardiovascular disease, chronic renal failure, and hepatitis is 49,000, with a range from 26,000 to 72,000. The Bureau of Labor Statistics estimates there are about 6,200 work-related injury deaths annually. Adding disease and injury data, we estimate that there are a total of 55,200 US deaths annually resulting from occupational disease or injury (range 32,200–78,200).*
Conclusions *Our estimate is in the range reported by previous investigators, although we have restricted ourselves more than others to only those diseases with well-established occupational etiology, biasing our estimates conservatively. The underlying assumptions and data used to generate the estimates are well documented, so our estimates may be updated as new data emerges on occupational risks and exposed populations, providing an advantage over previous studies. We estimate that occupational deaths are the 8th leading cause of death in the US, after diabetes (64,751) but ahead of suicide (30,575), and greater than the annual number of motor vehicle deaths per year (43,501).*
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KEY WORDS: occupation; attributable fraction; mortality

INTRODUCTION

Deaths from occupational disease and injury place a heavy burden on society, both in terms of economic costs and human suffering. Several investigators have recently attempted to estimate the magnitude and cost of occupationally-related mortality in the United States and elsewhere [Landrigan and Markowitz, 1989; Olsen and Kristensen, 1991; Kraut, 1994; Kerr et al., 1996; Leigh et al., 1997; Nurminen and Karjalainen, 2001]. For example, Leigh et al. [1997] estimated 65,800 deaths each year can be attributed to occupational injury (6,500) or illness (60,300), with a total cost to society of over $23 billion.

No national occupational disease mortality surveillance system exists in the US. Many life-threatening diseases have been associated with hazardous exposures at work, however, only a handful of these diseases (e.g., the pneumoconioses) are caused exclusively by work-related exposures [Rutstein et al., 1983; Mullan and Murthy, 1991]. Therefore, any

[1]National Institute for Occupational Safety and Health, Cincinnati, Ohio
[2]School of Public Health, Emory University, Atlanta, Georgia
[3]American Cancer Society, Atlanta, Georgia
*Correspondence to: Kyle Steenland, Rollins School of Public Health, Emory University, 1518 Clifton Road, Atlanta, GA 30322. E-mail: nsteenl@sph.emory.edu

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estimates of the magnitude of occupational disease must be generated from a number of different data sources and published epidemiologic studies.

Table I summarizes the existing estimates of the magnitude of US occupational mortality from the early and more recent studies. While the early studies by Bridbord et al. [1978] and Doll and Peto [1981] focused on cancer, more recent studies have incorporated estimates for fatal injuries and other chronic diseases that contribute to mortality. Occupational risk factors have been identified and the strength of the associations quantified for several types of cancer, cardiovascular disease, asthma, chronic obstructive pulmonary disease, and chronic renal disease [Rosenstock and Cullen, 1994]. Both the Landrigan and Markowitz [1989] and the Leigh et al. [1997] studies included all of these causes of death, as well as fatal injuries and pneumoconioses, in their estimates. Leigh et al. [1997] also included cerebrovascular disease, and both studies included nervous system disorders. Both these studies used a combination of quantitative methods and expert opinions to arrive at their estimates.

We have attempted to improve on recent efforts in estimating the magnitude of mortality from occupational disease. We have restricted ourselves to only those diseases with the most well-established occupational mortality, so the estimates are likely to be conservative. Estimation of occupational disease morbidity is also obviously important, but beyond the scope of this document. In contrast to occupational disease deaths, occupational injury deaths are relatively reliably enumerated because they are well-defined outcomes that do not require a many assumptions about occupational causality. We have added the deaths from occupational injuries to our estimated deaths from occupational disease to come up with a total number of annual deaths attributable to occupation.

METHODS

General Methods

Attributable fractions

We have estimated the percentages of deaths attributable to occupation among causes we consider as known to have an occupational component. This percentage is known as the attributable fraction (AF). Other frequently used terms are the population attributable risk (PAR) and the etiologic fraction (EF), although more strictly the EF may be considered a different measure than the AF [Greenland and Robins, 1988; Greenland, 1999].

TABLE I. Existing US Estimates of the Magnitude of Occupationally-Related Mortality

Study	Cause of death categories included	Exposures &/or occupations included	Estimated number or percent of deaths attributed to occupation
Bridbord et al., 1978; US	Respiratory cancer, mesothelioma, GI cancer, leukemia	Asbestos, arsenic, benzene, chromium, nickel, petroleum	20% of total cancer mortality, 58,000–75,000 annual asbestos-related deaths. 33,000 due to other exposures
Doll and Peto, 1981; US	Cancers "definitely" or "possibly" produced by occupational hazards	Known carcinogens	4% or 17,000 total cancer deaths annually
Landrigan and Markowitz, 1989; New York state extrapolated to US	Cancer	Not specified	10% of cancer deaths
	Pneumoconioses		100% of pneumoconioses
	Chronic respiratory		1–3% of chronic respiratory deaths
	Cardiovascular disease		1–3% of cardiovascular deaths, renal disease, and neurologic disorders
	Renal disease		Extrapolated US estimate of 50,000–70,000 annual disease deaths
	Neurologic disorders		
Leigh et al., 1997; US	Fatal injuries	Not specified	6,529 injury deaths
	Cancer		6–10% of cancer deaths (n = 31,025–51,708)
	Cardiovascular disease, cerebrovascular disease		5–10% of cardiovascular and cerebrovascular deaths ages 25–64 years (n = 5,092–10,185)
	Chronic respiratory disease		10% of chronic respiratory disease deaths (n = 9,154)
	Pneumoconioses		1,136 pneumoconioses deaths
	Nervous system disorders		1–3% of nervous system disorders (n = 269–808)
	Renal disorders		1–3% of renal disorders (n = 223–689) 53, 428–80,209 total deaths

The AF may be defined as ''the proportion of cases that are related to a given exposure'' or ''the fraction of disease in a population that might be avoided by reducing or eliminating exposure to an etiologic agent, provided that it is causative'' [Coughlin et al., 1994]. The formula for the AF is:

$$AF = \frac{P(E)(RR - 1)}{1 + P(E)(RR - 1)}$$

where P(E) is the proportion of the general population exposed to a particular agent, and the RR is the relative risk (or rate ratio, or odds ratio (OR)) of disease or death for the exposed vs. nonexposed. Population-based case-control studies can provide estimates both P(E) and RR. Cohort studies provide an estimate of RR; ancillary exposure information must be obtained to estimate P(E).

To combine AF estimates where there were multiple occupational risk factors for a single cause of death, an overall AF, assuming independent among exposures, is calculated. The formula for the combined AF for n independent exposures [Miettenin, 1974] is:

$$AF_{combined} = 1 - [1 - AF_1][1 - AF_2] \ldots [1 - AF_n]$$

The AF includes deaths (or cases) which would not have occurred at all except for an occupational exposure, or would have occurred at a later date. From a public health standpoint it would be useful to calculate a measure such as ''years of life lost'' which would quantify the prematurity of death or disease due to occupational exposure, but that requires more detailed data than we have on the age distribution of death (or disease) among the exposed and nonexposed [Greenland, 1999]. This broader approach has been used recently for some occupational injuries and disease in the WHO's Global Burden of Disease project [Majid et al., 2002].

For some diseases the AF can be derived from a study of diseased subjects only. In the case of asthma, for example, researchers have designed several studies to estimate the proportion of cases that can be attributed to occupational factors [e.g., Milton et al., 1998; Blanc and Kjell, 1999]. Among all cases of the disease, a subset of cases are identified that meet specific criteria that define them as occupationally-related. The criteria vary from study to study, but are usually based on individual patient information about symptoms, clinical data, and exposure history. Similarly, for hepatitis the proportion of infections due to occupation can be estimated from surveillance data, and using an estimate of case fatality, the number of occupational deaths can be estimated directly, without recourse to estimates of RR for the exposed versus non-exposed.

The principal limitations of AFs are that they do not include a measure of the impact of premature mortality, they have usually been used to estimate the number or proportion of occupational deaths at a single point in time [an exception is the asbestos literature, e.g., Enterline, 1981; Nicholson et al., 1982; Peto et al., 1995], and they are generally based on a dichotomous exposure/nonexposure classification, and do not take into account duration or level of exposure among the exposed. Despite these limitations, AFs offer the advantage of summarizing in a succinct way the burden of mortality due to occupational disease.

Causes of death included

We considered the following major categories of cause of death to have an occupational component: selected non-malignant respiratory diseases, selected cancers, heart disease, chronic renal disease, and cirrhosis/liver cancer. Within these categories, for the sake of brevity we included only specific causes of death which had at least 50 deaths attributable to occupation (resulting in the exclusion of rare causes such as deaths due to toxic fumes, hypersensitivity pneumonitis, and scrotal cancer, or some rare agents (carbon disulfide) where the number of workers exposed was too small to result in many attributable deaths). Other causes of death were excluded, either because little evidence or agreement about the existence of an occupational component was found, or because inadequate data existed upon which to base an estimate. The causes included comprise about 18% of all deaths.

Derivation of AFs

A variety of strategies was employed to determine the AFs. In general, data to calculate the AFs were derived from epidemiologic studies. In some cases previously published AFs were adopted directly from the literature, usually based on case-control studies or sometimes from review articles (e.g., asthma). In others we calculated the AFs ourselves as described previously, based on published RRs from cohort studies and ancillary population exposure data. One exception to this general procedure is pneumoconiosis, where we assumed all deaths were due to occupation (AR = 100%).

An exhaustive literature review for every cause of death was not conducted. For causes that have been well-studied, recent review articles were used. For other causes, we reviewed a representative sample of studies from the 1980s and 1990s (earlier studies may not accurately reflect the mortality risks faced by workers in the 1990s). RRs were primarily, but not exclusively, taken from US studies. Data on exposure prevalence were restricted to US data.

Exposure data for the US population

When using cohort studies to calculate AFs, we used estimates of the proportion of the population exposed from

two surveys conducted by NIOSH in the early 1970s (the National Occupational Hazard Survey (NOHS)) and early 1980s (the National Occupational Exposure Survey (NOES)) [NIOSH, 1974, 1977, 1978, 1988, 1989, 1990]. The NOHS and NOES surveys collected information from a representative sample of approximately 4,500 establishments representative of all nonagricultural and nonpublic businesses covered under the OSH Act of 1970, and employing eight or more persons. In addition, to excluding agriculture and the public sector, the NOES excluded the finance, insurance, and real estate industries. In general we relied on the more recent NOES rather than the NOHS survey. The NOES covered about 33 million, or one-third of the 102 million employed civilians and military personnel in the US in 1982 [U.S. Bureau of the Census, 1984]. Nonetheless, estimates of the proportion of the population exposed taken from NOES data were considered to be broadly representative of the whole US population.

NOES estimates of the proportion of the population exposed refer to only one point in time (the early 1980s). In order to estimate the number of individuals at excess risk of mortality in 1997 due to prior occupational exposure, the NOES estimate was multiplied by a factor of 4. This factor accounted for an approximate 10% occupational turnover, which would increase the size of the ever-exposed population about 5 times over a 40-year period, and an approximate 20% loss due to mortality prior to 1997. Using the NOES data requires the assumption that exposures during the relevant time period for disease causation can be adequately represented by the NOES survey conducted in the 1980s.

To calculate the proportion of the total population exposed, the number of exposed workers (sex-specific) estimated from NOES (multiplied by 4) was divided by the 1980 population (sex-specific) in the 18–64 age range as reported by the Bureau of the Census Current Population Reports [National Center for Health Statistics, 1999]. The same proportion was assumed to be applicable to persons over the age of 64 years.

Range of AFs

A range of AFs is generally reported for each cause. It could come from the range of AFs reported in well-designed case-control studies, or alternatively, from a range of RRs from cohort studies (resulting in a range of AFs). In three cases when using cohort studies (heart disease deaths related to job control, shift work, and passive smoking at work), adequate information was available to calculate an inverse-variance weighted average of RRs across studies, i.e., to conduct a meta-analysis [DerSimonian and Laird, 1986]. In these cases, the endpoints of the confidence interval for the weighted average were used as the range for the resulting AFs. This type of quantitative method was not possible for most other causes of death due to the lack of consistency in the methods and information available through published studies. Finally, in one instance a single well designed cohort study was used as the basis for the AF, and the range for the corresponding AF was estimated based on the confidence interval for the RR from that study. Hence, the ranges reported are by no means confidence intervals for the AR, but merely our educated estimate of the likely range for the true AR.

Attributable deaths

Annual deaths attributable to occupational exposures were calculated by multiplying the AFs by the total number of deaths from each specific cause in 1997 [National Center for Health Statistics, 1999]. Exceptions were diseases where deaths were estimated based on incidence data and case-fatality assumptions (mesothelioma, TB, and hepatitis). We used 1997 deaths occurring among US residents for the following age groups: ages 20 and over (asthma); ages 30 and over (non-malignant pulmonary disease, cancer, chronic renal failure); and ages 20–69 years (heart disease). We included both male and female deaths whenever possible, i.e., when there was evidence that significant number of females had experienced exposure.

Specific Methods

Pneumoconioses

Major types of pneumoconioses include: asbestosis, coal workers' pneumoconiosis, and silicosis. While byssinosis is not considered as a true pneumoconiosis by all occupational lung disease experts [NIOSH, 1996], byssinosis is included as a pneumoconiosis in this report. NIOSH currently tracks pneumoconiosis mortality based on multiple cause of death counts [NIOSH, 1996, 1999]. However, for consistency only deaths with pneumoconiosis as the underlying cause are considered here. All pneumoconiosis deaths are considered occupational.

Occupational asthma

Occupational asthma is defined as reversible, generalized narrowing of the airways caused by exposure to airborne dusts, gases, vapors, or fumes in the work environment. Work-related asthma includes preexisting asthma that is exacerbated by occupational exposure [CDC, 1994; Wagner and Wegman, 1998], as well as classic occupational asthma (i.e., asthma caused de novo by workplace exposure). Hundreds of occupational agents, including various dusts, gases, vapors, and fumes, are known to cause asthma [Chan-Yeung and Malo, 1995], and numerous other agents can aggravate preexisting asthma. Work-related asthma affects workers in most, if not all, occupations and industries.

There are a large number of studies of asthma morbidity in which the AF of asthma cases due to occupation has been estimated. Generally attribution of asthma to occupation was based on reported occupational exposures and/or work-related symptoms. The method of estimation has either been via an estimation of work-related asthma from a case series of asthma cases, or via the standard formula for AF applying a RR and a proportion exposed within a general population. Important recent studies include the review by Blanc and Kjell [1999], a large European-wide study by Kogevinas et al. [1999], and a large Finnish study by Karjalainen et al. [2002]. Most recently the American Thoracic Society reviewed 21 asthma studies and concluded that the occupational AF for asthma morbidity was approximately 15% (the median). Excluding one outlier study (estimated AF 76%), the interquartile range of these data was 11–21%. We have assumed that these estimates for asthma morbidity can also be applied to asthma mortality.

Chronic obstructive pulmonary disease (COPD)

The term COPD encompasses several conditions, including chronic bronchitis, emphysema, and chronic airway obstruction. Innumerable workplace- and industry-specific studies have clearly documented reduced airways function due to occupational exposure [Becklake, 1989; Oxman et al., 1993; Kennedy, 1997]. Community-based studies have also presented strong evidence attributing COPD to occupational exposures [Becklake, 1989; Kennedy, 1997]. A recent review of ten studies (some workplace based and some community based) by the American Thoracic Society [Balmes et al., 2002] concluded that approximately 15% of COPD was attributable to occupational exposures.

An overall AF for COPD due to occupation is best derived from community-based studies of the general population rather than from workplace- and industry-specific studies.

In the United States, two community-based studies provide the major sources of information for estimation of the occupation-related AF for COPD. The study by Lebowitz [1977] included a high proportion of mild disease unlikely to cause death.

A more relevant community-based study defined COPD on the basis more severe disease, measured objectively by lung function testing as $FEV_1/FVC < 60\%$ [Korn et al., 1987]. This study was also much larger, involving a random sample of over 8,500 white adults (both males and females, ages 25–74 years) in six cities in the United States. Thirty-one percent (45% of men and 19% of women) were considered to have had occupational dust exposure. An overall OR (adjusted for age, sex, smoking, and city of residence) of 1.5 (95% CI = 1.2–2.0) was calculated for those occupationally exposed to dust versus no exposure. The overall occupational AF computed from this study is 14% (5–24%), men and women combined. We have chosen to use this estimate as the most representative for well-defined COPD in the US, noting that it is consistent with the recent estimate of the American Thoracic Society [Balmes et al., 2002].

Tuberculosis

An increased risk of TB infection and/or active disease has been recognized among: occupations associated with silica exposure and silicosis, which increases susceptibility to TB (e.g., sandblasters and foundry workers); occupations associated with transmission of TB through exposure to active cases (e.g., health care workers, correctional facility workers, funeral directors) [Markowitz, 1994; CDC, 1995; McKenna et al., 1996; Boudreau et al., 1997; Steenland et al., 1997b; Miller et al., 2002], and occupations attracting individuals from segments of the general population at high risk of TB (e.g., migrant farm workers) [Bowden and McDiarmid, 1994].

The Occupational Safety and Health Administration (OSHA) has published a comprehensive risk assessment for TB infection, disease, and death among US workers [OSHA, 1997], using a variety of epidemiologic evidence. Workers with potential occupational exposure in the mid-1990s were considered to be those in hospitals, nursing homes, homeless shelters, and correctional facilities (estimated total 5.3 million). For hospitals OSHA estimated lifetime risk of TB infection due to occupational exposure for workers in hospitals with good exposure controls [Aitken et al., 1987], some controls [Price and Rutala, 1987], and poor controls [Boudreau et al., 1997]. In each case a range of excess risks was given which corresponded to the range of background rates in different parts of the country, since excess risk depends on the level of background risk. Lifetime excess risks of infection ranged from 0.004 to 0.072 for good controls, 0.034 to 0.472 for average controls, and 0.067 to 0.723 for poor controls.

OSHA further assumed that approximately 10% of all TB infections progress to active TB disease and that a fraction (7.8%) of these active cases are not successfully treated and develop further complications leading to death. OSHA then estimated the number of workers exposed in different sectors, the number likely to be infected per year, the number likely to develop active TB, and the number of annual expected deaths due to TB due to occupational exposures (115–136). Subsequently, the Institute of Medicine [IOM, 2001] issued a report on occupational risk of TB which presented data suggesting that the rate of progression from infection to active TB was more likely to be 5% rather than 10%. OSHA is currently revising its 1997 risk assessment, and is considering the IOM concerns. We have used the 5% estimate of progression from IOM, and reduced OSHA's original estimates by 50%.

Cancer

We focused on specific cancers identified as occupationally-related by the International Agency for Research on Cancer (IARC) [Vainio et al., 1994], and in agents classified by IARC as carcinogens in either Group 1 (sufficient evidence) or Group 2A (probable evidence). We based our lung and bladder cancer estimates on AF estimates from several US studies that included a wide range of occupations and industries, including most of the exposures related to lung and bladder cancer on the IARC 1 and 2A lists. For other cancer sites, we used the IARC Group 1 list, supplemented by recent literature, to identify well-documented associations and RR estimates [Vainio et al., 1994; IARC at www.iarc.fr, 2002], and then used ancillary exposure data to estimation the proportion exposed. Epidemiologic data for most Group 2A carcinogens are insufficient to calculate AFs.

Lung cancer. Arsenic, asbestos, beryllium, cadmium, chromium, diesel fumes, nickel, silica, environmental tobacco smoke (ETS), and radon are the principal occupational exposures which have been strongly linked to lung cancer [Steenland et al., 1996].

Estimates in the literature for AFs due to occupational lung carcinogens from US population-based case-control studies range from 6.1% to 17.3% for men [Hinds et al., 1985; Lerchen et al., 1987; Vineis et al., 1988; Morabia et al., 1992]. Steenland et al. [1996] used RRs from cohort data and estimates of the exposed populations from NIOSH NOES data to estimate an AF for men and women of 9% and 2%, respectively. Using the low and high AF estimates for men from the above studies, and the single estimate for women from the Steenland study, the AF for lung cancer deaths attributable to occupational exposures was estimated to be between 6.1% and 17.3% among men, and 2% for women.

The RR for lung cancer from ETS exposure among never smokers is approximately 1.3 [Hackshaw et al., 1997]. Assuming that 20% of never smokers exposed to ETS in the workplace, and 10% of lung cancer deaths per year occur among never smokers, and there are 153,000 lung cancer deaths per year, so there should be about 15,300 never-smoker lung cancer deaths per year, the AF is approximately $0.2*(1.3-1.0)/0.2*(1.3-1.0)+1=5.7\%$.

Indoor radon exposures at work may also cause some lung cancers, based on the assumption of no threshold, analogous to the situation for indoor radon exposures at home [Lubin and Boice, 1997]. A person spending 70 years in a residence and 45 years working lifetime in buildings, both with the US average radon concentration of 1.5 pCi/L [Samet, 1989] would accumulate approximately 20 WLM (working level months) of exposure to radon progeny from the home and approximately another 5 WLM from buildings [by comparison, the median exposure to a uranium miner in the US has been about 430 WLM, see Roscoe et al., 1989]. Estimates of the number of lung cancers due to residential exposure to radon in homes are about 10,000 per year [Samet, 1989]. Based on relative dose at home and at work (approximately 5:1), occupational exposure to radon in buildings may account for another 2,000 annual cancers, presumably equally divided between males and females (AF 1.3%).

Bladder cancer. A number of occupational exposures have been associated with bladder cancer, e.g., 2-naphthylamine, magenta manufacturing, benzidine, auramine manufacturing, and 4-aminobyphenyl. For men we used the AFs calculated by Vineis and Simonato [1991] assuming a broad definition of exposure from 18 bladder cancer studies, six of which were US population-based studies (AF range 7–19%). For women we used the AF of 11% (95% CI 3–19%) estimated by Silverman et al. [1990] in a large population based case-control study.

Mesothelioma. Mesothelioma is a rare cancer that is usually related to asbestos exposure. The incidence rate in 1991 was 1.7/100,000 for white males and 0.4/100,000 for white females [Ries et al., 1996b]. Mesothelioma is greatly underestimated in the mortality files as there is no ICD code in the 9th revision that is specific for mesothelioma. Since the cancer is nearly always fatal within a year, the best estimate of the number of mesothelioma deaths is from the incidence data from SEER.

Mesothelioma is usually work-related. One study estimated that 88% of pleural and 58% of peritoneal mesotheliomas (overall 85%) in men were attributable to work-related asbestos exposure [Spirtas et al., 1994]. For women the overall attributable risk was 23%. If the deaths due to ''take-home'' asbestos exposure were considered, the attributable risks may be around 90%.

We have used these figures as our upper and lower bounds for the AFs for men and women.

Leukemia. Occupational exposure to external ionizing radiation is known to be associated with leukemia incidence and mortality [Ulrich, 1946; Seltser and Sartwell, 1965; Caldwell et al., 1980, 1983; Smith and Douglas, 1986; Koshurnikova et al., 1996; Cragle et al., 1999]. A point estimate for the RR from the BEIR V report for an exposure greater than 100 mSv (millisievert) is 1.57, with a 95% CI of 1.18–2.88. For those exposed to between 50 and 100 mSv, a RR point estimate of 1.22 was used, with a 95% CI of 1.07–1.70 [NAS/NRC, 1990].

Wing et al. [2000] estimate that approximately 5% of workers in five combined DOE cohorts had a cumulative external dose of at least 100 mSv, and an additional 5% exposed to between 50 and 100 mSv. It is estimated that there have been approximately 550,000 persons employed at DOE facilities since the 1940s [US DOE, 1999, pp 8, with

additional information from NIOSH databases]. Another large group of radiation-exposed workers includes civilian nuclear power employees, numbering approximately 500,000. A 1992 study estimated that 6% of these workers have been exposed cumulatively to 100 mSv or more, and 7% have been exposed to between 50 and 100 mSv [Shore et al., 1992]. A study of 143,000 radiologic technologists estimated that about 3% were exposed to greater than 100 mSv, and about 6% were exposed to between 50 and 100 mSv [Boice et al., 1992].

From the numbers and percentages exposed given above, we estimate that a total of 61,700 US non-military employees have been exposed to cumulative external ionizing radiation doses of 100 mSv or greater, as of the 1990s. A total of 70,900 US non-military employees have been exposed to cumulative external ionizing radiation doses of 50–100 mSv. An AF of 0.02% was calculated for workers exposed to 100 mSv or greater and an AF of 0.01% for those with exposures between 50 and 100 mSv.

IARC has classified four work-related agents or exposure circumstances as carcinogens causing leukemia [Vainio et al., 1994]; benzene, ethylene oxide, the rubber industry, and boot and shoe manufacture and repair. A recent review by Lynge et al. [1997] estimated a range of RRs for benzene and leukemia of 2–4. A similar review by Blair and Kazerouni [1997] for ethylene oxide reported a range of RRs of 1.1–3.5 for leukemia. We have used the two ranges of RRs for the respective AF calculations. A recent review by Ward et al. [1997] found no evidence of excess leukemia in three recent US studies of the boot and shoe manufacture and repair industries, nor in studies of the rubber industry since 1981. Separate AFs for these two industries were therefore not estimated; any deaths in these industries would be assumed to be due to benzene exposure and would therefore be included in the AF for benzene.

Laryngeal cancer. Sulfuric acid is recognized by IARC as a laryngeal carcinogen [Vainio et al., 1994], while mineral oils, and asbestos have been reported as possible occupational exposures associated with laryngeal cancer. A review by Blair and Kazerouni [1997] of 16 studies of sulfuric acid and laryngeal cancer was used to determine the range of RRs. The RRs in the positive studies ranged from 1.1 to 5. A review by Tolbert [1997] of nine studies reporting 18 RRs of workers exposed to mineral oils found RRs ranging from 0.5 to 2.5 for laryngeal cancer. Thirteen of 18 RRs were elevated, ranging from 1.1 to 2; five were statistically significant. We have calculated AFs for laryngeal cancer for both sulfuric acid and mineral oils using the above ranges of RRs, and exposure estimates from NOES [Ruder, 1996]. A combined AF for the two exposures was estimated to be from 1.0% to 20.0%.

Skin cancer. A review by English et al. [1997] found that in 20 occupational studies of solar radiation and melanoma, five were positive, 10 were null, and five were negative; studies of occupation and non-melanoma skin cancer were also contradictory. Because of the conflicting evidence regarding occupational exposure to solar radiation and skin cancer, solar radiation was not included in calculations for skin cancer.

According to IARC, several other occupational exposures are related to non-melanoma skin cancer. They are coal-tar pitches, coal tar, shale oils, soots, coke production, arsenic, and mineral oils. The polycyclic aromatic hydrocarbon (PAH)-exposed populations (coal-tar pitches, coal tar, shale oils, soots, and coke production) probably overlap and are combined and treated as a single group. RR estimates taken from a review by Boffetta et al. [1997] range from 1.1 to 1.5, a range we have used in calculating AFs.

For non-melanoma skin cancer, the RRs for PAH from the Boffetta review and an exposure estimate derived from NOES were used [Pedersen and Young, 1997]. A RR of 2 for arsenic with an exposure estimate derived from NOES were also used [Ruder, 1996]. Exposures were assumed to occur primarily among men. The estimated combined AF for these two exposures ranges from 1.2% to 6.0%.

Sinonasal and nasopharyngeal cancer. Furniture and cabinet making, wood dust, nickel compounds, hexavalent chromium, boot and shoe manufacturing and repair, and isopropanol manufacturing using the strong-acid process have all been associated with cancer of the nasal passages. A number of occupations have potential wood dust exposure, including carpenters, loggers, precision woodworkers (including furniture and cabinet makers), and woodworking machine operators.

A recent pooled re-analysis of five cohorts of wood furniture workers, plywood workers, and wood model makers [Demers et al., 1995] found elevated risks for both nasopharyngeal cancer (RR = 2.4) and sinonasal cancer (RR = 3.1). In contrast, a study of US union carpenters found no excess for either site [Robinson et al., 1996].

A review of metals and cancer [Hayes, 1997] found a wide range of RRs for nickel and nasal cancer, ranging from 1 to 173, depending on the type and level of exposure. In the review, two US studies of nickel mining, smelting, and refining found RRs ranging from 1 to 2.2. Two recent studies of hexavalent chromium found RRs of 10.8 [Davies et al., 1991] and 5.2 [Rosenman and Stanbury, 1996].

Since the industries are not likely to overlap, the estimated deaths for the selected exposures were added to get an overall estimate for nasal cancer. It was also assumed that exposures occurred primarily among men. AFs were calculated using the above RRs and NOES exposure data (30–46%).

Kidney cancer. The only occupation that has been related to cancer of the kidney is coke production [Vainio et al.,

1994]. A recent update of a study of 15,818 coke oven workers [Constantino et al., 1995] found a RR of 2.03 (95% CI 0.89–4.03) for kidney cancer. Using this RR and an exposure estimate from the NOES [Ruder, 1996] gives an estimated AF of 0.8% (range 0–2.3%)(males only).

Liver cancer. Vinyl chloride has been identified as a work-related liver carcinogen. A review of early vinyl chloride studies (1974–1981) by Alderson [1986] found a combined observed/expected of 4.68. The risk in later studies is lower. Combining the four studies from 1988 to 1995 from a review by Blair and Kazerouni [1997] resulted in an SMR of 2.50 (95% CI 1.9–3.3). Because the studies were based on males, we calculated AFs only for males, using the above data and ancillary NOES data (AR 0.4–1.1%).

Coronary heart disease (CHD). CHD, unlike cancer, is in many cases thought to result from recent occupational exposures rather than ones long ago (analogous to cigarette smoke, for which the risk of CHD drops rapidly once exposure ceases). AFs were applied to CHD deaths in the 20–69 year age range, under the assumption that most CHD effects are short-term effects which end not long after exposure ceases. If this assumption is not correct, the estimates in this report may be conservative, as the number of CHD deaths increase sharply after 69 years of age.

We restricted our estimates to the outcome of CHD (ICD9 410–414). Other authors considering occupation [e.g., see Leigh and Schnall, 2000] have included hypertensive disease (ICD9 401–404), peripheral atherosclerotic artery disease (ICD9 440), and stroke (ICD9 430–438). While these are plausible outcomes to include assuming a blood pressure mechanism for increased cardiovascular disease, the occupational epidemiology for cardiovascular disease is virtually all based on CHD (ICD9 410–414), and we have restricted ourselves to this outcome only, rather than assuming the epidemiology might apply to other outcomes. An exception to this general rule was made for noise, where a blood pressure mechanism is well accepted and where we included stroke with CHD. CHD represents the majority of all cardiovascular disease. For example, for ages 20–69 years in 1997, using all possible categories mentioned above, approximately 72% of cardiovascular deaths were from CHD, 8% from hypertensive disease, 19% from stroke, and 1% from peripheral atherosclerotic disease.

Exposures not included

Specific agents linked to heart disease in the literature include carbon disulfide, nitroglycerin, and lead [Olsen and Kristensen, 1991; Fine, 2000]. There is good evidence for carbon disulfide and nitroglycerin (at past levels) but the number of workers exposed is small and the number of attributable deaths are less than 50, and they are not included here. While lead has been shown to be related to hypertension, paradoxically it has not been associated with increased cardiovascular disease in heavily-exposed cohorts [Fine, 1992, 2000], and is also not included.

Carbon monoxide exposure at or above the current permitted occupational level of 50 parts per million time-weighted average has been associated with a 35% increase in CHD mortality in a NIOSH study of bridge and tunnel workers [Stern et al., 1988], but a reduction in risk was seen after 1970 when ventilation systems were improved and exposures lowered. Studies of workers in other occupations with lower and perhaps intermittent exposures (iron foundry workers, steelworkers, and fire fighters) have found no increase in risk [Fine, 2000]. Steenland [1996] reported that the NOES survey estimates that there are more than 3.5 million workers exposed to carbon monoxide, mostly through exposure to motor exhaust, with low and/or intermittent exposures. Since workers exposed to low or intermittent levels of carbon monoxide do not appear to have an increase in risk, carbon monoxide exposure was not included.

For nonchemical risk factors, while there is evidence that physical inactivity at work is a risk factor [Olsen and Kristensen, 1991; Fine, 2000], it is difficult to measure quantitatively in a standardized fashion and needs to be considered in conjunction with the level of physical activity outside of work.

Exposures included

Noise. Noise above 90 dBA induces an acute elevation of blood pressure in humans, returning to normal after noise ceases [Tarter and Robins, 1990]. The more recent and better designed epidemiologic studies, albeit largely cross-sectional, suggest that exposure above 85 dBA is consistent with a 2 mm increase in diastolic blood pressure [Tarter and Robins, 1990; Green et al., 1991; Tomei et al., 1991, 2000; Zhao et al., 1991; Lang et al., 1992; Hessel and Sluis-Cremer, 1994; Kristal-Boneh et al., 1995; Sokas et al., 1995; Hirai et al., 1999; Talbot et al., 1999], although this literature is not entirely consistent. A 2 mm increase in diastolic blood pressure would lead to a estimated RR of 1.11 for CHD for US men, and we here assume that same RR for women [van Hoogen et al., 2000]. There are approximately 9 million workers in the US with exposure at or above 85 dBA as an 8 hr average [NIOSH, 1998], representing about 6% of the US population aged 20–65 years. This suggests an AF of 0.6% for CHD, which might be consider an upper limit, with a lower range of 0 under the assumption of no chronic effect of noise.

Elevated blood pressure also increases risk of stroke. Data from 17 randomized trials of antihypertensive drugs suggests that a 6 mm increase in diastolic blood pressure causes a RR of 1.38 for fatal stroke [MacMahon and Rodgers,

1994]. Assuming a linear relationship, we would expect a 2 mm increase would cause a RR of 1.11, the same as the RR for CHD, leading again to an estimated AF of 0.6%. There were 10,014 deaths from stroke in 1997 among persons 20–65 years old. Again we assumed a lower and upper bound for the AF of 0 and 0.6%.

Job strain and job control. Job strain has been defined by Karasek et al. [1981] as low decision latitude and high psychological demand associated with the job. There is a large body of research in Europe on the effect of job strain on CHD [Johnson and Hall, 1988; Haan, 1988; Johnson et al., 1989, 1996; Hammar et al., 1994, 1998; Bosma et al., 1998; Theorell et al., 1998]. In most of these studies, high job strain was often defined by a combination of the low decision latitude (job control) and the high psychological demand (e.g., using quartile cutpoints), either by job title or self-report, for the most recent or current job. Most of these studies show that increased job strain increases CHD risk.

US studies of job strain and CHD incidence or mortality have failed to find the same effects for job strain as European studies [Reed et al., 1989; Alterman et al., 1994; Steenland et al., 1997a]. Kasl [1996] has suggested that US workers may not attribute as much of their stress to work as do European workers, but rather more of it to family conflict.

In recent years, it has become clear that job control, one component of job strain, is a variable which is more consistently linked to CHD across studies than is job strain. Most studies show an increased risk with decreased job control. High job demands, on the other hand, by themselves do not appear to have a consistent effect on heart disease risk. For this reason we base our estimates here on findings for job control (also known as decision latitude), rather than job strain.

A problem with much of work in this area is that effect measures in many studies were not controlled for social class. As lower socioeconomic status (SES) is associated with lower job control, and lower SES is a strong risk factor heart disease in industrialized countries, it is not clear if the effect of job control on heart disease in a number of studies is due to SES or job control. Marmot et al. [1997] has argued that job control is more important than SES in multivariate models, and may play a key role in the observed association of SES with heart disease. It is quite difficult to disentangle measures of job control and SES, and it is possible that different results in multivariate models reflect the precision with which these two variables are measured rather than any inherent independence of one versus another [Wamala et al., 1999].

To estimate the AF of CHD deaths due low job control, the rate ratios from the three US prospective studies were used [Reed et al., 1989; Alterman et al., 1994; Steenland et al., 1997a], as well as four European studies that controlled for social class [Bobak et al., 1997; Bosma et al., 1998; Theorell et al., 1998; Wamala et al., 2000]. These studies all controlled for the conventional risk factors (e.g., age, blood pressure, obesity, cigarette smoking, excess alcohol consumption, and cholesterol), and all controlled for social class with the exception of Reed et al. (who studied a population where SES may have been less disparate than in other studies). Most studies used a job control measure inferred from the occupational title. When results based on both self-report and inferred job were available, we used results based on inference from job title. Some studies included only men, other only women, and some both. Here we have assumed that job control effects for men and women are similar and both have estimated an AF for both sexes combined.

The combined rate ratio via meta-analysis was 1.38 (1.20–1.58). Most studies used quartiles of job control for calculating rate ratios [Hammar et al., 1998 was excluded because only high and low job control was used]. There was no evidence of heterogeneity ($P = 0.74$). Assuming 25% of working men and women exposed to low job control, reduced to 20% of the total population to account for those of working age but not working, the resulting AF is estimated to be 7.1% (range 3.8–10.4%).

It should be noted that some work in this area has been based on another model, the effort and reward model [Bosma et al., 1998]. Data are still somewhat sparse to estimate AFs based on this model.

Shift work. A review by Steenland [2000] includes eight studies of shift work and CVD judged to be of high quality. The results of the studies are not consistent. The RRs range from 0.9 to 1.4 and Steenland states that the epidemiologic data suggest a modest association. Taking an inverse-variance weighted average of the eight studies results in a combined RR of 1.1 (95% CI = 1.0–1.2). An estimate of the percent of workers involved in shift work from the May 1991 Current Population Survey was available [BLS, 1992] for full-time wage and salary workers. Based on the survey, an estimated 20.0% of male workers (or 17.8% of all males ages 20–64 years) and 14.6% of female workers (10.7% of all females ages 20–64 years) are employed in shift work. Using the endpoints of the RR confidence interval from Steenland (1.0–1.2), results in an AF range from 0% to 3.4% for males and 0% to 2.1% for females.

Environmental Tobacco Smoke. Olsen and Kristensen [1991] and Law et al. [1997] estimated a RR of 1.3 for CHD due to ETS. Wells [1998], using data from seven studies of non-smokers with workplace exposure to ETS, estimated an RR of 1.35. Steenland [1999], using data from the five of the same studies (with eight RRs, males and females) that are described as without apparent major design or exposure categorization problems, calculated a combined RR of 1.21

(95% CI 1.04–1.41). The population considered at risk for ETS was non-smoking workers.

An estimated 21% of 81 million workers who were never or former smokers were exposed to ETS in the workplace according to the 1988 Occupational Health Supplement to the National Health Interview Survey [CDC, 1992]. Using a range of RR of 1.21–1.35, and 21% exposed, the AF was estimated to range from 4.2% to 6.8% of nonsmoking workers. Using demographic data, an estimated 53,200,000 nonsmoking workers are aged 35–64 years and an additional 5,000,000 are aged 65–69 years, for a total population of 58,200,000 [Steenland, 1999]. An annual CHD mortality rate of approximately 75/100,000 in non-smoking workers aged 35–69 years is estimated. Applying this death rate to 58,200,000 resulted in an annual number of 43,340 CHD deaths among non-smoking workers aged 30–69 years.

Non-malignant renal disease. Approximately 1/3 of new ESRD cases are due to glomerulonephritis or hypertensive disease [U.S. Renal Data System, 1999] categories which may have an occupational etiology. Other categories, such as diabetic or congenital disease, are unlikely to be related to occupation. Several occupational exposures have been associated with renal disease. The best evidence of an association is for silica, lead, cadmium, and organic solvents [Wedeen, 1992, 1997; De Broe et al., 1996].

At least four case-control studies [De Broe et al., 1996] have found significant associations between incident renal disease and silica exposure, with ORs from 1.4 to 6.5. Calvert et al. [1997] and Steenland et al. [2001] have studied silica-exposed cohorts and a RR of ESRD of 1.4 and 2.0, respectively, concentrated in glomerulonephritis. Mortality data from Steenland et al. [2002] found a RR of underlying cause mortality of 1.4 (1.1–1.9) for three cohorts of silica-exposed workers. The AF was calculated assuming a mortality RR of 1.4, and an estimated 2.0 million workers exposed to silica (1.7 outside mining, an estimated 300,000 in mining) [NIOSH, 1991]. This resulted in an estimated AF of 4%. Virtually all exposed workers are male. The AF was applied to 1/3 of the male 1997 deaths for renal disease, under the assumption that approximately 1/3 of such deaths were due to unknown etiology.

Several large case-control studies have investigated the relationship of organic solvents and incident renal disease [Steenland et al., 1990; Nuyts et al., 1995; Stengel et al., 1995]. ORs ranged from 1.2 to 1.5. We have assumed incidence RRs from these studies apply to mortality, and we have used a range of RR from 1.2 to 1.5. The estimated number exposed is from the NOES [Lynge et al., 1997]. This provides an AF estimate ranging from 1.4% to 17.3%.

Heavy metals have been hypothesized as risk factors for renal disease, primarily due to interstitial nephritis. Wedeen [1992] states that only lead and cadmium are accepted causes of chronic renal disease. Nuyts et al. [1995] states that the epidemiological evidence for the association between lead and renal disease is still unclear and that the current levels are probably less hazardous than earlier ones. Two case-control studies found ORs of 1.73 [Steenland et al., 1990] and 2.11 [Nuyts et al., 1995] for lead exposure. Using an OR of 2 and an estimated 1,650,000 exposed workers [Ruder, 1996] results in an AF of 1.5% for deaths related to lead exposure. Cadmium exposure is known to lead to sub-clinical renal damage but the evidence for frank renal disease is more uncertain. Nuyts reported a nonsignificant excess of about two. Using a RR of 2 and an estimated 500,000 exposed workers [Ruder, 1996] results in an AF of 0.4% for cadmium.

Hepatitis B and C. Hepatitis B virus (HBV) and hepatitis C virus (HCV) infections among health care workers can result in deaths from acute hepatitis (rare, and virtually all due to fulminant Hepatitis B) or from chronic infections which can progress to cirrhosis and/or hepatocellular carcinoma (HPC). Chronic infection can occur with or without acute hepatitis, and it accounts for the great majority of deaths due to hepatitis.

With regard to HCV, Armstrong et al. [2000] have estimated infections in the US from 1950–1990 based on NHANESIII prevalence data. The authors assumed infection always results in sero-conversion, and assumed 2.5% sero-reversion rate per year. Their data indicate an approximately constant infection rate of 20/100,000 from 1950–1959, increasing linearly from 20 to 120/100,000 in 1980, and then increasingly linearly from 120 to 140/100,000 through 1990 [Figure 3 in Armstrong et al., 2000]. We have assumed that approximately 3% of these infections are occupational in origin, based on very sparse data from the CDC's Sentinel Counties Study [Alter, 1997; and personal communication, Ian Williams, CDC]. We assume that 1–5% of infected people will die of HCV related disease (cirrhosis, HPC) in a period 20–40 years later [Liang et al., 2000; personal communication, Miriam Alter, 2001]. This assumption is open to question, as the natural history of HCV infection is not well understood, and for some cases there may be a shorter latency period. We also assume a normal distribution with a mean year of death 30 years after infection and a standard deviation (SD) of 3.3, which ensures virtually all deaths will occur in the 20–40 year period. To derive expected deaths in 1997, we restricted ourselves to occupational infections beginning in 1957 (last year of deaths will be in 1997) and ending in 1977 (first year of deaths will occur in 1997). The total expected HCV deaths due to occupation in 1997 ranges from 37 to 186 under these assumptions, with a midpoint estimate of 111.

An alternative approach to estimating deaths subsequent to HCV infection is to assume that 3% of deaths from chronic HCV infection are due to occupational exposures, in line with the estimated fraction for HCV infection. CDC has estimated [CDC, 1998] that there are approximately 8,000–10,000

deaths per year from cirrhosis/HCC subsequent to chronic HCV infection, which would imply 240–300 deaths per year attributable to occupation. These estimates are higher but broadly consistent with our estimates above. Combining the two approaches, we estimate from 37 to 300 deaths a year in 1997 due to past HCV infection, with a midpoint of 169.

For HBV, Coleman et al. [1998] have estimated the number of annual infections in the US during 1976–1994 based on prevalence data from NHANESII and NHANESIII. The estimated annual number of new infections during these years was approximately constant at 330,000. Modifications have been made to the figures in Coleman et al. by the CDC staff to better account for age at infection and temporal trends, and we have used these numbers for the years 1980–1998 (personal communication, Ian Williams, 2001) The estimates begin at 207,000 in 1980, peak at 287,000 in 1987, and tail down to 110,000 by 1997. We assumed that the linear decreasing trend from 1987 to 1980 continued back until 1967 and that approximately 4% of HBV infections are due to occupational exposures [personal communication, Ian Williams, 2001]. We further assume that 0.2% of infection lead to deaths from fulminant hepatitis B, that 2–6% of infections become chronic, that 1/3 of these chronic infections result in cirrhosis and/or HPC, and that 3/4 of these serious illnesses result in death [personal communication, Miriam Alter, 2001]. These assumptions are broadly consistent with estimates in a standard pathology text [Cotran et al., 1999], and result in a case fatality rate of 0.7–1.7% for those infected. Finally, we assume that an average of 20 years pass between infection and death, with a range from 10 to 30 years [personal communication, Miriam Alter, 2001], and again we assume a normal distribution for the time until occurrence with a mean of 20 years and a SD of 3.3. Under these assumptions, we estimate that there were 20–49 deaths from prior occupational infection with HBV in 1997 (midpoint 30).

Injuries. For occupational fatalities, we have relied on 1997 estimates from the Census of Occupational Fatalities (CFOI) [CFOI, 1999]. Estimates of occupational fatalities from the most reliable sources tend to agree fairly well; for example, in 1992 the CFOI estimate and the estimate from NIOSH's National Traumatic Occupational Injury data base estimated 6,063 and 5,714 deaths, respectively. Both CFOI and NTOF are largely based on death certificates, however, and are therefore likely to somewhat underestimate worker injury deaths. For a discussion, see Leigh et al. [1997].

RESULTS

Table II shows the estimated number of occupationally-related disease deaths by cause of death. For each cause, the table includes the total number of US deaths, the estimated AF or proportion due to occupational exposures, and the estimated number of occupational deaths. For AF's calculated from RRs and exposure estimates, those numbers are also included in the table. A summary of results follows:

Pneumoconioses

All pneumoconiosis are assumed occupational in origin. Based on underlying cause of death (ICD 9th revision 500–505), pneumoconiosis mortality has declined gradually over the last two decades, from a maximum of 2,150 deaths in 1972 to 1,087 deaths in 1997. By type, asbestosis is exceptional in having increased mortality over this same time period, from fewer than 100 deaths per year to over 300 annually [NIOSH, 2000].

Asthma

Assuming an AF of 15%, there were an estimated 784 deaths due to occupational asthma in 1997 (range 575–1,099).

COPD

Assuming an AF of 14%, there were an estimated 14,257 COPD deaths in 1997 attributable to occupational dust exposure, with a range of 5,092 and 24,440.

Tuberculosis

We estimate that in 1997 there were 55–68 deaths from TB attributable to occupation, out of a total of 1,100 deaths that year (an AF of 5%).

All Cancer Combined

We estimate 12,682–26,244 occupationally-attributable deaths per year (2.4–4.8% of all cancer deaths, 0.8–1.0% among females, 3.3–7.3% among males).

Lung Cancer

Classic industrial lung carcinogens, ETS at work, and radon exposure at work are estimate to cause 9,677–19,901 lung cancer deaths per year (AF 8.0–19.2% for males, 2% for females).

Bladder Cancer

We estimate 534–1,451 deaths for males annually attributable to occupational exposures, and 116–740 deaths for females, for a total estimate of 651–2,191 bladder cancer deaths per year attributable to occupational exposures (AF 5.6–19.0%).

TABLE II. Estimated Number of Occupationally-Related Disease Deaths, by Cause of Death, 1997

Cause of death (ICD) & exposure	No. of US deaths[a] male (M); female (F)	Estimated no. of exposed	Estimated % exposed	RR	Estimated proportion due to occupational exposures (%) (AF)	Estimated no. of occupational deaths
Selected respiratory diseases						
Pneumoconiosis (500–505)	1,087 (M + F)	n.a.[b]		n.a.[b]	100	1,087
Coalworkers' pneumoconiosis (500)	486				100	486
Asbestosis (501)	405				100	405
Silicosis (502)	98				100	98
Unspecified/other [aluminosis, berylliosis, stannosis] (503, 505)	92				100	92
Asthma (493)	5,199 (M + F)	n.a.[c]		n.a.[c]	11–21	571–1,091
COPD (491, 492, & 496)	101,834 (M + F)	n.a.[c]		n.a.[c]	5–24	5,092–24,440
Pulmonary tuberculosis (011)	1,162 (M + F)	n.a.[c]		n.a.[c]	5–6	55–68
Total non-malignant respiratory disease deaths						6,805–26,686
Selected cancers						
Lung cancer (162)	91,289 (M); 61,877 (F)	n.a.[c]		n.a.[c]	6.3–13.0 (combined)	9,677–19,901
Chemical exposures					6.1–17.3 (M); 2 (F)	6,807–17,031
ETS (never smokers only, 10% of all lung cancer deaths)					0.6 (M + F)	870
Indoor radon at work					1.3 (M + F)	2,000
Bladder cancer (188)	7,638 (M); 3,897 (F)	n.a.[c]		n.a.[c]	7–19 (M); 3–19 (F)	651–2,191
Mesothelioma (n.a.)	2,081 (M); 548 (F)	n.a.[c]		n.a.[c]	85–90 (M); 23–90 (F)	1,895–2,366
Leukemia (204–208)	19,038 (M + F)				0.8–2.8 (combined)	152–533
Benzene		1,000,000	0.72	2–4	0.8–2.0	
Ethylene oxide		1,000,000	0.72	1.1–3.5	0–1.6	
Ionizing radiation (100 + mSv)		61,700	0.04	1.3–2.1	< 0.05	
Ionizing radiation (50–100 mSv)		70,900	0.05	1.1–1.4	< 0.05	
Laryngeal cancer (161)	3,016 (M)				1.0–20.0 (combined)	30–603
Sulfuric acid		3,000,000	4.4	1.1–5.0	0.4–15.0	
Mineral oils		4,400,000	6.4	1.1–2.0	0.6–6.0	
Skin cancer (173)	1,407 (M)				1.5–6.0 (combined)	21–84
PAHs		8,222,800	12.0	1.1–1.5	1.2–5.7	
Arsenic		240,000	0.36	2	0.1	
Sinonasal (SN) (160) & nasopharynx (NP) (147)	303 (SN) (M) & 436 (NP) (M)				33.0–46.0(SN) & 30.0–42.0(NP)	231–322 (SN + NP)
Wood dust		4,515,200	6.8	3.1 (SN); 2.4 (NP)	12.5 (SN); 8.7 (NP)	
Nickel compounds		4,000,000	6.0	2.2	6.7 (SN & NP)	
Hexavalent chromium		3,400,000	5.2	5.2–10.8	18.0–33.8 (SN & NP)	
Kidney cancer (189)	7,131 (M)					
Coke production		520,000	0.76	2	0.0–2.3%	0–164
Liver cancer (155)	7,283 (M)					
Vinyl chloride		320,000	0.48	2.5	0.4–1.1	29–80
Total occupationally-related cancer deaths						12,086–26,244
Coronary heart disease (410–414)	76,756 (M); 31,872 (F)					

Noise	9,000,000 (M + F)	5.8 (M + F)	1.0–1.1	0.0–0.6 (M + F)	0–651 for CHD; 0–80 for stroke
Job control	n.a.	20 (M + F)	1.20–1.58	3.8–10.4 (M + F)	4,217–11,297
Shift work	n.a.	17.8 (M); 10.7 (F)	1.0–1.2	0–3.4 (M); 0–2.1 (F)	0–2,609; 0–669
ETS at work	43,340 (nonsmokers aged 35–69)	n.a.	21% (M + F) (nonsmokers aged 35–69)	1.21–1.35	4.2–6.8 (M + F) (among nonsmokers aged 35–69)	1,820–2,947 (nonsmokers aged 35–69)
Total occupationally-related CHD deaths						6,037–18,253
Renal disease (one-third of 580–587[d])	4,000 (M)				8.2–14.5 (combined)	328–580
Silica		2,000,000	2.9	1.4	4.0	
Organic solvents		8,125,000	11.9	1.2–1.5	2.3–8.6	
Lead		1,650,000	1.5	2	1.5	
Cadmium		500,000	0.4	2	0.4	
Liver cancer, non-alcoholic cirrhosis, chronic hepatitis 155, 517.4, 571.5, 571.9	24,288					
Due to hepatitis B and C infection		n.a.	n.a.	n.a.	n.a.	50–350
Total occupationally-related disease deaths						25,910–72,121

[a]Numbers of US deaths are for 1997 for the following age groups: ages 20 years and over (asthma); ages 30 years and over (non-malignant respiratory diseases, cancer, and chronic renal failure); and ages 20–69 years (CHD).
[b]All pneumoconioses assumed to be due to occupational exposures (AF = 100%).
[c]AFs taken from the literature, see text.
[d]See text for which categories of renal disease are considered potentially caused by occupation.

Mesothelioma

Using SEER rates for mesothelioma and the 1997 US population estimate of 130,046,000 males and 136,984,000 females, an estimate 2,081 male deaths and 548 female deaths were due to mesothelioma. Applying the attributable risk estimates of 85% for males and 23% for females as a low estimate, and 90% as the high, results in a range of 1,895–2,366 work-related mesothelioma deaths per year.

Leukemia

Using the range of RRs reported for benzene (2–4) and ethylene oxide (1.1–3.5), and exposure estimates for these chemicals from the NOES [Ruder, 1996], plus the 95% confidence limits for the RRs reported for the two levels of ionizing radiation with the estimated number of exposed workers, a combined AF of 0.8–2.8% was calculated. This results in an estimated 152–533 leukemia deaths per year attributable to occupational exposures.

Laryngeal Cancer

We estimate an AF of 1–20% for this cancer (males only), resulting in an estimated 30–603 laryngeal cancer deaths per year attributable to occupational exposures.

Non-Melanoma Skin Cancer

Using an AF of 1.2–6%, we estimate 24–92 deaths among males due to PAH or arsenic exposure.

Nasal Cancer

A total of 231–322 annual male deaths from sinonasal and nasopharyngeal cancer are estimated to be attributable to occupational exposures (31–43% AF).

Kidney Cancer

We estimate 57 kidney cancer deaths per year attributable to occupational exposures (range 0–164)(AF 0–2.3%).

Liver Cancer

We estimate 29–80 deaths per year among males due to occupational exposures to vinyl chloride (AF 0.4–0.11%).

Coronary Heart Disease (CHD)

Combining noise, job control, shift work, and ETS results in an overall estimate of 4,500–12,900 CHD deaths attributable to occupation in 1997 (6.3–18.0% of all CHD deaths among persons ages 20–69 years). Of these noise

was responsible for an estimated 0–462 deaths (AF 0–0.6%), job control for an estimated 2,707–7,331 deaths (AF 3.8–10.4%), shift work for an estimated 0–2152 deaths (AF 0–3.4%), and occupational ETS (nonsmokers only) for an estimated nonsmokers of 1,820–2,947 deaths (AF 4.2–6.8%).

Non-Malignant Renal Disease

We estimate an AF for kidney disease due silica, solvents, lead, and cadmium combined of 8.2–14.5%, and an estimate of attributable deaths ranging from 328 to 580.

Liver Cancer, Non-Alcoholic Cirrhosis, and Chronic Hepatitis Due to Hepatitis B and C

Combining the HCV and HBV estimates, we estimate approximately 200 deaths in 1997 (range 50–350) from cirrhosis/HPC subsequent to occupational infection. This represents an AF of approximately 2% (0.2–1.4%).

All Occupational Disease Mortality Combined

Summing the estimates across all disease categories, the estimate of the total annual burden of occupational disease mortality is between 26,000 and 72,000. The midpoint of 49,000 is our best overall estimate.

All Occupational Mortality Combined

The Census of Fatal Occupational Injuries (CFOI) estimated that there were 6,238 US 1997 deaths due to occupational injury in the US [CFOI, 1999]. Combining disease deaths with injury deaths leads to an overall annual estimate of 55,200 occupationally caused deaths per year (range 32,200–78,200), using 1997 data.

DISCUSSION

Comparison With Previous Studies

This review provides an overall range of estimates for occupationally-related disease deaths that is broadly consistent with previous reports for the US by Landrigan and Markowitz [1989] and Leigh et al. [1997]. In contrast to prior estimates, we have restricted ourselves to well-established occupation-disease associations which are quantifiable, and calculated all AFs ourselves, with full documentation.

Differences in results between our study and prior ones stem from a variety of reasons, which we will discuss for the most important disease categories. We will also comment on another recent report on occupational mortality from Finland [Nurminen and Karjalainen, 2001]. While this report is not directly relevant to the US (exposure prevalences were taken from a Finnish job-exposure matrix, and AFs depend on these exposure prevalences as well as assumed RRs), and while the authors tended in our view to include exposures for which the epidemiologic evidence remains doubtful in our view [a view shared by others, see Coggon, 2000], this effort represents one of the more thorough efforts to estimate occupational AFs to date. Another important effort for a single country (Denmark) can be found in Olsen and Kristensen [1991].

Cancer

We estimated a range of total cancer deaths between 13,000 and 26,000, which is between 2.4% and 4.8% of all cancer deaths and 5–10% of the cancer deaths for cancer sites for which estimates could be generated. Most of these deaths were due to lung cancer, bladder cancer, and mesothelioma. Our overall range of 2.4–4.8% is similar to the estimate made earlier by Doll and Peto [1981], who attributed 4% of all US cancer deaths to occupational exposures. Landrigan and Markowitz [1989] estimated 10% of all cancer deaths were caused by occupation, although details for this estimate were not provided. Leigh et al. [1997] estimated a range of 31,000–52,000 cancer deaths, or about 6–10% of all cancer deaths. Leigh et al. used higher AF estimates for both lung and bladder cancer than were used in this report. These two causes alone accounted for 15,000–20,000 cancer deaths in the Leigh et al. study. Estimates by Landrigan et al. and Leigh et al. were also higher than the estimates derived here in part because they applied cancer risk data from primarily male industrial populations to the female population, which in most instances had little or no exposure to industrial carcinogens. Gender-specific estimates made by Doll and Peto [1981] were 1.2% for females and 7% per males; our estimates are 0.8–1.0% among females, 3.3–7.3% among males. Leigh et al. also adjusted their estimate upward based on continuing recognition of new and suspected carcinogens in the workplace, and many unexplained cancer excesses in a variety of occupational groups. We believe that this reasoning was correct, and that the true proportion of cancer deaths due to occupation is higher than that estimated based on well-documented epidemiologic associations, although this underestimation cannot be quantified (see Discussion below, in ''sources of underestimation'').

Nurminen and Karjalainen [2001] estimated that 8% of cancers in Finland were due to occupation, including 24% of lung cancers. They derived AFs largely on the basis of estimates of the number exposed in Finland and RRs from cohort studies. Their estimates are higher than most reported in the literature, partly due to their inclusion of some agents for which the epidemiologic evidence is not universally

accepted (e.g., lead exposure and work as a hairdresser are considered lung carcinogens), and possibly due partly to their estimates of the exposed population (numbers not provided). Their estimate of the AF due to asbestos exposure, the main contributor to the lung cancer AF, is 14% among men (RR used, 2.3), somewhat higher than a corresponding estimate for the US in the 1990s (6%, based on Nicholson, 1982), or the 8% which can be inferred from Morabia et al. [1992].

Coronary heart disease (CHD)

Estimation of CHD deaths due to occupation is difficult because there are a large number of established nonoccupational risk factors which make it difficult to detect a possibly weaker occupational effect; most of the suspected occupational agents (e.g., job control) are difficult to measure and the epidemiology is not definitive; and a working population is generally healthier than the general population. We estimate 6,000–18,300 CHD deaths are attributable to occupational exposures (6.3–18.0% of all CHD deaths among persons ages 20–69 years, midpoint 12.2%) in 1997. This estimate is not that different from the estimate by Leigh et al. [1997] of 5,100–10,200 or 5–10% of all cardiovascular (no ICD codes given) and cerebrovascular deaths of persons aged 25–64 years in 1992. Leigh and Schnall [2000] use an AF of 5–20% for all occupational cardiovascular risk factors, similar to our estimate, but provide no detailed justification for these numbers. Landrigan and Markowitz [1989] do not present an AF for cardiovascular disease specifically.

Nurminen and Karjalainen [2001] estimate an AF of 17% for CHD, higher than our midpoint estimate, based on shift work/strain (11% for men, 6% for women), noise (6% for men, 2% for women), carbon monoxide (2% for men, 0% for women), and ETS (2% for men and women). These numbers are higher than ours for shift work and noise, due to both higher assumed RRs and higher estimated exposure prevalences. We did not include carbon monoxide. Nurminen and Karjalainen included stroke (AF 11%) along with CHD in assessing cardiovascular disease; we excluded stroke because in our view there is a lack of well-established epidemiologic evidence (see below).

Non-malignant respiratory disease

Our combined estimate for occupationally caused deaths from non-malignant respiratory disease (TB, COPD, pneumoconoses, and asthma) ranged from 7,000 to 27,000 (midpoint 17,000), with an AF of 6–25%. The wide range in our estimate is due to the wide confidence interval for the OR in the single study used for estimating COPD deaths. Our estimate exceeds both the Landrigan and Markowitz [1989] estimate of 1–3% of chronic respiratory deaths (i.e., 1,000–3,000 deaths), and the Leigh et al. [1997] estimate of 11% (9,000). Both previous estimates were based on applying a single AF to the overall category of non-malignant respiratory disease.

Nurminen and Karjalainen [2001] estimated COPD AFs of 12%, similar to our own of 14%. For asthma, these authors used an AF of 18% similar to the 15% we used.

Sources of Underestimation

Many diseases not included

The overall estimate presented here considers only selected causes of death (selected cancers, respiratory diseases, CHD before the age of 70 years, renal failure, cirrhosis), for which an association with occupation is most clearly established and which account for at least 50 occupationally-caused deaths per year. Together these causes comprise about 18% of all US deaths. There are a large number of other causes for which there is suggestive evidence of an occupational association, but the association is either not well established or quantitative data are not available to estimate the number of occupation-related deaths. These include gastrointestinal cancer [Cocco et al., 1996; Kang et al., 1997], prostate cancer [Aronson et al., 1996; Krstev et al., 1998a,b], breast cancer [Loomis et al., 1994; Cantor et al., 1995; Petralia et al., 1998], autoimmune diseases associated with silica [Parks et al., 1999], other infectious causes associated with occupation (e.g., AIDS, infections acquired from animal handling), cardiovascular disease subsequent to high blood pressure due to lead exposure [Schwartz, 1995], interstitial fibrosis and occupation [Coultas et al., 1994; Baumgartner et al., 1997; Mullen et al., 1998], pneumonia [Coggon et al., 1994], and neurodegenerative diseases [Schulte et al., 1996]. Deaths due to the late sequelae of disabling occupational injuries may also be appreciable but are difficult to estimate with current data.

Coronary heart deaths after age 69 excluded, and other cardiovascular endpoints excluded

Our estimates for CHD were restricted to CHD deaths occurring before the age of 70 years. Most occupational risk factors for CHD are thought to be short-term and to not persist long after exposure ceases. If this assumption is not true then CHD deaths could be substantially underestimated. Similarly, most cardiovascular epidemiology is based on CHD and we have considered only CHD. Were hypertensive disease, stroke, and peripheral atherosclerotic disease included, our estimates for cardiovascular disease would increase by about 25%. Leigh and Schnall [2000] argue for an even broader definition of cardiovascular disease, and an age limit of 74 years on risk. While we believe this position to be currently unjustified (lacking epidemiologic data to support

it), it is certainly arguable. Nurminen and Karjalainen [2001] used decreasing weights for CHD risk for ages after 60 years, and assumed no risk after the age of 74 years. They included stroke as well as CHD in considering cardiovascular disease.

Contributory causes of death excluded

We included only those deaths in which the occupational disease was listed as the underlying cause of death. Many deaths occur not only as a result of the underlying cause, but also as a result of one or more other causes that contribute to death, but are not directly related to the underlying cause. For example, two-thirds of decedents who die with pneumoconiosis list the disease as a contributory cause of death.

Underascertainment and underreporting

Another important concern, relating especially to pneumoconiosis, asthma, and mesothelioma, is underascertainment and underreporting of cases and/or deaths. The pneumoconioses are diseases of long latency and physicians who are unfamiliar with the disease may misdiagnose and record them as a more familiar non-occupational disorder on the death certificate. The current national count of fewer than 300 silicosis deaths has been estimated to be from one-third to one-fourth of what would be reasonable in light of the Department of Labor estimates that 59,000 new cases of silicosis would develop among the work force exposed to silica in 1980 [Bang et al., 1995]. Weiss and Wagener [1990] have noted that for asthma in general mortality is rare, and may be under-recorded on death certificates.

Limited exposure data

Another factor contributing to underestimation is incomplete knowledge regarding the range of occupational exposures that can lead to fatal illness. For example, there are likely to be a large number of unknown carcinogens in the workplace. There is evidence of excess cancer risk in several groups of workers, where the specific agent or carcinogen has not been identified [e.g., farmers, see Blair et al., 1992; Khuder and Mutgi, 1997].

Many case-control studies of cancer limit their exposure definition to occupational groups without identifying any suspected carcinogens. In other cases, the exposure is not sufficiently well quantified to be included here (e.g., sedentary work and colon cancer). Similarly, for our cancer estimates that were based on RRs from cohort studies and exposure estimates from national surveys, we only included hazards identified by IARC as Group 1 carcinogens, except for lung and bladder cancer, which were more inclusive. Although the inclusion of Group 2A and 2B carcinogens probably would not have added a large number of deaths, there are certainly many suspect carcinogens and chemicals in this group that have yet to be studied in humans [Karstadt, 1998]. There are 298 agents, mixtures and exposures listed by IARC in Group 2A and 2B, most of which have sufficient evidence for carcinogenicity in animals but limited or insufficient evidence in humans.

Sources of Overestimation

Inadequate adjustment for confounding factors

A key element in the validity of the AF is that the risk factor in question must have a causal relationship with the disease. For most diseases considered here there is a well established causal relation between occupational exposure and disease. However, one threat to the validity of the data is confounding, and in a number of studies discussed here, information on confounding factors was not available. However, Simonato et al. [1988] found that adjustment for smoking—one of the principal important potential confounders—did not have much of an impact on RR estimates for lung cancer. Furthermore, most of the estimates for respiratory disease considered here did in fact conduct some adjustment for potential confounding by smoking. Confounding is likely to be less important for other factors less strongly related to disease.

Causality

Evidence for occupational causality is in general, weaker for heart disease than for other diseases considered here. The complex interactions between socioeconomic class, personal risk factors, and job-related factors, have made it difficult to establish causality in studies of heart disease in relation to job control, shift work, noise, and ETS, for example. If the observed associations are not causal, we will have overestimated the CHD deaths due to occupation. However, we have generally included conservative estimates of the effects of these factors, including in some cases a lower range of no effect. It is therefore possible that we have in fact underestimated the fraction of CHD attributable to these exposures.

Assuming independence of multiple occupational exposures

In the case of coronary disease and a few of the cancers, the method for estimating a combined AF for multiple occupational risk factors could have led to an overestimation. For heart disease in particular, there was no single study that could provide information concerning interactions and adjusted RRs for all of the occupational factors (job control,

shift work, noise, etc.), as well as the joint distribution of those factors in the US population. Instead, the assumption was made that the risk factors are independent in order to calculate the combined AF for all factors. Certainly for some of the coronary disease risk factors (shift work, job control), this is not an entirely valid assumption, and it may have resulted in an overestimation of the AFs.

Relative risks too high

Another factor that might lead to overestimation is the lack of recent studies for many causes of death, particularly cancer. Many estimates of RRs relied on older studies that looked at cancer incidence or deaths in the 1980s and 1970s, in some cases involving exposures that occurred decades earlier. In many cases, adequate data were not available to evaluate the changes in exposure over this time period through the present, and to adjust RRs accordingly.

Exposures overestimated

Many of the exposure estimates were from the NOES, which was conducted in the early 1980s. This is generally not a limitation, since the NOES data were used mainly in conjunction with cancer estimates where a long latency period is appropriate. However, the NOES may overestimate some exposures, because it was not based on actual measurements of the level or duration of exposure, only the surveyor's observations that the potential for exposure was present in a particular workplace.

The estimate for exposure to ETS in the workplace (21% of nonsmokers reported discomfort from ETS at work) is from a 1988 survey, and may be overestimated; it is likely that changing practices and laws have resulted in a lower rate of exposure since that time. However, a study based on the 1992–1993 US Current Population Survey found that as many as 35% of workers in indoor workplaces reported that their workplace either had no policy restricting smoking, or allowed smoking in some or all work areas [Gerlach et al., 1997].

Other Limitations

Survival rates

Some of the RRs used here were based on studies of disease incidence or prevalence, rather than mortality studies. While a high percentage of cases of some diseases, such as lung cancer and mesothelioma will result in death, individuals with other diseases such as asthma, COPD, bladder cancer, and coronary disease will experience higher survival rates, possibly biasing some of the RRs used here in unpredictable fashion.

Broad range of estimates

Most of the estimates fell within a broad range of possible values. This was at least partly due to variations in study designs and study populations among the studies contributing to the estimates. For example, the authors of the large reviews of lung and bladder cancer case-control studies noted that the main sources of variation in the AF estimates were study design and geographic variation in exposure prevalence [Vineis and Simonato, 1986; Simonato et al., 1988]. While some studies were designed to represent a cross-section of industries, others were intentionally located in areas with high cancer rates and high densities of suspect industries. Furthermore, studies used different methods for selecting controls, and varied in their use of next-of-kin respondents. The authors speculated that these differences contributed to a very broad range of AFs for lung and bladder cancer.

CONCLUSIONS

We believe our estimate of 49,000 (range 26,000–72,000) annual US deaths due to occupational disease mortality is the most well-documented estimate to date for the US. The Census of Fatal Occupational Injuries (CFOI) estimated that there were 6,238 US 1997 deaths due to occupational injury in the US [CFOI, 1999]. Adding this figure to our own, we estimate that overall there were approximately 55,200 annual US deaths due to occupational causes in 1997. This would make deaths attributable to occupation the 8th leading cause of death in the US, after diabetes (64,751) but ahead of suicide (30,575), and greater than the annual number of motor vehicle deaths per year (43,501) (www.cdc.gov/health/causes.htm).

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