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The Global Burden of Non-Malignant Respiratory Disease Due to Occupational Airborne Exposures

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Background *Occupational non-malignant respiratory disease arises from exposure of workers to airborne agents, mostly particulate or dusts. We describe the worldwide mortality and morbidity from asthma, chronic obstructive pulmonary disease (COPD), and pneumoconioses arising from these occupational exposure and focus on cases reported in the year 2000.*

Methods *The proportions of workers exposed to the agents, and their levels of exposure, were estimated using workforce data and the CAREX (CARcinogen EXposure) database. These were combined with relative risk measures (for asthma and COPD) or absolute risk measures (for the pneumoconioses) to develop estimates of deaths, disability-adjusted life years (DALYs) and attributable fraction (for asthma and COPD).*

Results *There were an estimated 386,000 deaths (asthma: 38,000; COPD: 318,000; pneumoconioses: 30,000) and nearly 6.6 million DALYS (asthma: 1,621,000; COPD: 3,733,000, pneumoconioses: 1,288,000) due to exposure to occupational airborne particulates.*

Conclusions *Occupational airborne particulates are an important cause of death and disability worldwide.* Am. J. Ind. Med. 48:432–445, 2005. © *2005 Wiley-Liss, Inc.*

KEY WORDS: asthma; COPD; pneumoconiosis; occupation; work; deaths; DALYs; global burden

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INTRODUCTION

Occupational non-malignant respiratory disease arises as a result of the exposure of workers to airborne agents, mostly in the form of particulates or dusts.[1] The primary route of exposure is inhalation, whereby these agents gain access to the respiratory system and are either deposited

[1] Dusts are technically defined as dry particle aerosols produced by mechanical processes such as breaking, grinding and pulverizing (Johnson and Swift, 1997). Particle sizes range from less than 1 μm to over 100 μm. The smaller particles present a greater hazard, as they remain airborne longer and are more likely to enter the respiratory tract. Dusts may be organic (e.g., grain dust) or inorganic (e.g., silica, asbestos, and coal mine dust).

(in the case of dusts) or enter the circulatory system. For some exposures, there is a very clear connection between the exposure and the disease (e.g., silicosis is only caused by exposure to silica). Some exposures cause more than one type of disease, and even more than one type of respiratory disease. For example, asbestos can result in malignant conditions of the lung and the pleura (the inside lining of the chest), malignant conditions of the peritoneum (the inside lining of the abdomen) and non-malignant conditions of the lungs (asbestosis and, probably, chronic obstructive pulmonary disease (COPD) [Churg, 1988; Ohar et al., 2004]). Other exposures have not been well characterized, but the condition they cause is well defined (such as some forms of occupational asthma).

The worldwide impact of work in causing non-malignant respiratory disease, in terms of mortality and morbidity (disability), focusing on the year 2000 and resulting from relevant past and current exposures is described. There are a vast number of respiratory conditions that can arise directly, or indirectly, from work. However, estimating exposures, risks, and attributable proportions is not possible for many of these on an international (or even national) scale, because of lack of appropriate data sources. Therefore, only the more important of the work-related respiratory conditions, in terms of the total number of cases or the risks arising from exposure, are included. These are asthma, COPD and the three main pneumoconioses (asbestosis, silicosis, and coal workers' pneumoconiosis). Malignant respiratory disease is not included here as it is described elsewhere [Driscoll et al., 2005]. Infectious respiratory conditions related to work, hypersensitivity pneumonitis and beryllium disease are also not included.

MATERIALS AND METHODS

The general methodology, Comparative Risk Assessment (CRA) used in this study is described in detail in Nelson et al. [2005]. In summary, the methods used to determine appropriate exposure-risk relationships varied depending on the condition in question. Asthma and COPD can be caused by occupational and non-occupational factors. For these two diseases, the approach was to estimate the proportion of the population exposed (and thus at risk), and determine the relative risks of developing the condition of interest in response to a certain level of exposure (or a surrogate of exposure—occupation for asthma, and economic sub-sector for COPD). From this information, the population attributable fraction (AF) (the fraction of disease or death due to occupation) was calculated, which in turn, allowed the determination of population burden. To calculate the attributable fraction of deaths or DALYs due to exposure to a specific health risk factor, the estimates of the proportion of a population (f_i) exposed to the risk factor at k levels of exposure,[2] and the relative risks of morbidity and/or mortality from a specific adverse health effect due to that exposure (RR_i), were combined in the following equation:

$$AF = \left(\sum_{i=0}^{k} f_i RR_i - 1\right) \Big/ \left(\sum_{i=0}^{k} f_i RR_i\right)$$

The population attributable fractions were determined in two ways. The mortality attributable fraction (the fraction of deaths due to work) was determined on the basis of mortality. The overall attributable fraction was based on DALYs (disability-adjusted life years), which take into account both mortality and morbidity.

A different approach was taken for the pneumoconioses, which are fibrotic lung diseases caused only by occupational exposure to specific mineral dusts. In the case of specific pneumoconioses, the burden is best estimated by using absolute risk rather than relative risk, since there is virtually no background risk for the non-exposed. The burden (in terms of deaths and DALYs) of pneumoconiosis arising from exposure to silica, asbestos, or coal was determined by applying absolute risk estimates directly to the estimated population at risk.

Estimating Risk Factor Levels

Causative agents of asthma

Asthma, which is a narrowing of the upper respiratory passages resulting in difficult breathing and wheezing, has both non-occupational and occupational causes. Many hundreds of occupational agents, including some inorganic and organic dusts, have been associated with occupational asthma [Chan-Yeung and Malo, 1994; Venables and Chang-Yeung, 1997]. Biological agents include grains, flours, plants and gums, fur, feathers and other animal parts, insects and fungi, drugs and enzymes, and various types of wood. Chemical agents include chlorofluorocarbons, alcohols, metals and their salts, and welding fumes [CCOHS, 1997]. These agents are found in a variety of workplaces, including food and natural products processing, animal handling facilities, manufacturing, and construction.

It would not be possible to conduct exposure assessments and to obtain relative risk data for all the factors contributing to this important occupational disease, especially since they often occur in combination. Therefore, occupation was used as a proxy for exposure to agents that are associated with occupational asthma. The basis for this approach was the work of Karjalainen et al. [2001, 2002], who conducted extensive epidemiological studies of the

[2] Although a CRA analysis can be conducted with only two levels of exposure (e.g., "nonexposed" and "exposed"), smaller values of k may compromise accuracy of the analysis.

entire Finnish workforce and developed relative risk estimates for specific occupations. A similar but less extensive study based in 12 industrialized countries was also used for agricultural occupations [Kogevinas et al., 1999].

The proportion of the total population with occupational exposure to asthmagens was estimated by determining the proportion of the population working in occupations that matched as closely as possible those identified by Karjalainen et al. [2001, 2002] and for which relative risk values were provided. These calculations were done separately for men and women for each subregion (termed ''region'' henceforth for simplicity) of the world (see [Nelson et al., 2005]). Population workforce participation [ILO, 2002] and occupation [ILO, 1995] data came from the International Labour Organisation (ILO). The best estimate of the proportion of persons considered at risk is difficult to determine. Not all workers in each occupation would have been exposed to asthmagens, and persons who were exposed in the past but not currently are assumed to remain at risk, at least for a number of years. Therefore, as for COPD (described below) it was assumed that the number of persons currently employed in specific occupations corresponds roughly to the population at risk (the number ever occupationally exposed to asthmagens).

Causative agents of COPD

The causative agents of COPD are non-specific dust and fumes, with dusts showing a more consistent relationship than fumes [Becklake, 1989]. Unfortunately, there are no data to estimate the proportion of the world's workers exposed to dust and/or gas/fumes. A study by Korn et al. [1987] provides a link between self-reported exposure to dust (current and past exposure) and some categories of economic activity[3] among the currently employed. Categories of economic activity among the currently employed are available worldwide, and can provide a broad approximation to the proportion of the world's population with current or past exposure to dust and/or gas/fumes. The estimates of exposed populations used here are based on data on employment in the economic sectors of agriculture, industry and service [World Bank, 2001], supplemented by data on employment [ILO, 2001] in economic activities (or sub-sectors), and data on the Economically Active Population [ILO, 2002].

Korn et al. [1987] defined as low-exposed those in finance, as medium-exposed those in the manufacture of non-durable goods, transport, utilities, and the wholesale and retail trades, and as highly exposed those in the manufacture of durable goods, agriculture, mining and construction. Exposure was to ''dusts'' and to ''gases,'' without these being further defined. Similar categories were adopted for this study, except that the three categories were termed ''non-exposed,'' ''low,'' and ''high.'' There was also some modification to account for factors such as lack of data on the type of manufacturing industry and the fact that agriculture in developed and developing countries probably involves different types of exposure to respirable dust. Lacking data that would have permitted manufacturing to be divided into low and high potential for dust exposure, it was classified as having low dust exposure, although noting that in much of the world manufacturing involves more dust exposure than is typical in the United States where the Korn study [Korn et al., 1987] was done [Gomes et al., 2001; Chien et al., 2002]. Those not in the workforce and those in utility, trade, finance, and services were defined as not exposed. Those in agriculture, manufacturing and transportation were defined as having low exposure, while those in mining and construction were defined as having high exposure. In the Korn et al. study [1987], the proportion of workers currently employed in the medium- and high-exposure industries listed above corresponded approximately to the proportion of those reporting ever having been occupationally exposed to dust in that study. We similarly assumed that the number of currently employed in specific industries corresponds roughly to the number ever occupationally exposed to dusts, and therefore roughly to the true proportion of the population at risk, since past exposure as well as current exposure increases risk of COPD**.** The proportion exposed in different economic activities in each region was adjusted to account for an average labor force participation among the currently exposed in that region, which was applied across all ages.

Causative agents of pneumoconiosis: silica, asbestos, and coal

Occupational dusts can result in other non-malignant respiratory diseases apart from asthma and COPD. The most important of these are silicosis, asbestosis, and coal workers' pneumoconiosis, which are caused by exposure to silica, asbestos, and coal mine dust, respectively. Assessment of the proportion of workers exposed to silica and asbestos was based on the distribution of the economically active population by economic sub-sector and the percentage of workers exposed to these substances by economic sub-sector. An adjustment factor of four was used to account for turnover in jobs with exposure to occupational particulates; that is, the number of currently exposed workers was multiplied by four to estimate the number of ever-exposed workers, again relevant because both past exposure and current exposure increase risk. The factor of four was derived assuming a steady state exposed population with 10% turnover per year and a follow-up period of 40 years [Nelson et al., 2005].

The primary data source on the proportion of workers exposed to silica and asbestos for each economic sub-sector was the CAREX (CARcinogen EXposure) database [FIOH,

[3] Economic activities comprised agriculture, mining, manufacturing, utilities, construction, trade, transport, finance, and services.

TABLE I. Mean Proportions of Workers Exposed to Silica and Asbestos, by Economic Sub-Sector, in the European Union[a]

Carcinogen	Agriculture	Mining	Manufacturing	Electrical	Construction	Trade	Transport	Finance	Services
Silica	0.00372	0.23049	0.02327	0.01415	0.18860	0.00017	0.00476	0.00002	0.00061
Asbestos	0.01248	0.10248	0.00590	0.01702	0.05203	0.00292	0.00684	0.00016	0.00284

[a]From CAREX [FIOH, 1998].

1998], which presents data on the proportion of workers in the European Union exposed to 139 carcinogens (IARC Group 1, 2A and selected 2B agents) at levels above background in 1990–1993. These estimates were based on national workforce data and exposure prevalence estimates from Finland and the United States, adjusted for the economic structure of each country, then refined by national experts (Table I).

It was assumed that the proportion of workers exposed to silica and asbestos in a specific economic sub-sector was constant throughout the world; and that within a given economic sub-sector, both male and female workers and younger and older workers had the same probability of exposure. (This probability of exposure was taken from CAREX, which gave a single combined probability based on both genders and all ages). Data were available on the distribution of males and female in the different economic sectors, and these were taken into account in the analysis.

The US Occupational Safety and Health Administration (OSHA) permissible exposure levels (PELs) were used to classify exposures to silica and asbestos as Low or High Level. Due to the greater prevalence of occupational health and safety regulatory programs and infrastructure in the A regions (highly developed countries), it was estimated that a larger proportion of workers was exposed at the lower levels in these regions than in the BCDE regions (less developed countries)[4] [Roach, 1992; Hewett, 1996]. In addition, the absolute exposure levels in the A regions were estimated to be lower than in the BCDE regions (i.e., ''Low'' exposure in A regions was defined as a lower absolute level of exposure than ''Low'' exposure in BCDE regions).

To estimate exposure to silica (the only cause of silicosis) and asbestos (the only cause of asbestosis), values above and below the silica and asbestos PELs were selected to characterize the Low- and High-exposure groups. These values were used to estimate cumulative exposure, which was needed because the epidemiological data provided estimates of silicosis and asbestosis risk in terms of cumulative asbestos exposure. For A regions, exposure was assumed to be at (for High exposure) or 20% of (for Low exposure) the prevailing PEL in the United States during the relevant years of exposure for various ages. For B, C, D, and E regions, exposure was assumed to be 1.5 times (for High exposure) or 0.5 times (Low exposure) the prevailing PEL in the United States during the relevant years of exposure for various ages. Cumulative exposure estimates for workers of different ages were determined by modeling of an artificial cohort to estimate the average cumulative exposure for such a representative cohort in steady state. A steady state working population was produced by randomly assigning durations of exposure to exposed workers based on a log normal distribution with a geometric mean of 10 years (the average duration of exposure across a large number of industrial cohorts studied by NIOSH (personal communication, Kyle Steenland, 2000)). Considering 40 years follow-up, a 10% annual turnover, a mean age at first employment of 20 years, and geometric mean duration of exposure of 10 years, we then estimated the age-specific duration of exposure for all workers in the artificial cohort at the end of 40 years. Cumulative exposures for silica and asbestos for exposed workers of specific ages in the year 2000, assumed to have been exposed over the previous 40 years, were then estimated by applying these exposure durations to the relevant exposure intensities for Low and High exposures, as described earlier. The relevant PEL for silica was 0.1 mg/m^3. Cumulative exposures for asbestos took into account changes in the United States PEL for asbestos during the years of interest to the current analysis, with the level before 1972 of 12 fibers/ml decreasing, through several steps, to 0.1 fibers/ml in 1994 [Nelson, 1997; Martonik et al., 2001]. Both males and females were assumed to be at risk if employed in the relevant industry and occupation.

Because the CAREX database does not include data on exposure to coal mine dust, it was necessary to use a different methodology for this exposure than that used for silica and asbestos. The estimates of the proportion of the population exposed to coal mine dust were based on global coal production, but the approach to partitioning of exposure into Low/High categories was the same as described for silica and asbestos. We estimated that 10 million workers worldwide were exposed to coal in 1990 [ILO, 1995; Energy Information Administration, 2001]. The data for 1990 were used in order to allow for an average 10-year latency period for development of CWP. It has been assumed that a negligible proportion of coal miners are female. Therefore, all

[4] Regions: AFR, Africa; AMR, Americas; EMR, Eastern Mediterranean; EUR, Europe; SEAR, South-East Asia; WPR, Western Pacific; A, Very low child, very low adult mortality; B, Low child, low adult mortality; C, Low child, high adult mortality; D, High child, high adult mortality; E, High child, very high adult mortality.

TABLE II. Relative Risks for Occupational Asthma by Original Occupation and Economic Sub-Sector, and Sex, Age-Adjusted

Occupation	Relative risk (males)	Relative risk (females)	Source
Background	1.00	1.00	Non-working population, used as reference
Administration	1.00	1.00	Karjalainen et al. [2002], also used as reference level
Technical	1.05	1.06	Karjalainen et al. [2002]
Sales	1.14	1.13	Karjalainen et al. [2002]
Agricultural	1.41	1.41	Kogevinas et al. [1999]
Mining	1.95	1.00	Karjalainen et al. [2002]
Transport	1.31	1.22	Karjalainen et al. [2002]
Manufacturing	1.56	1.33	Karjalainen et al. [2002]
Services	1.53	1.41	Karjalainen et al. [2002]

employment figures were attributed to the male labor force. As for silica and asbestos, a turnover factor of four was applied to account for persons not currently working on coal mining but who had been exposed previously. The ''PEL'' used for coal was the United States Mine Safety and Health Administration (MSHA) value of 2 mg/m^3 [NIOSH, 1995].

Risk Factor—Disease Relationships

Asthma

Estimates of relative risk primarily came from the work of Karjalainen et al. [2001, 2002], in which asthma was defined by the occurrence of clinically diagnosed asthma. Relative risks were calculated in that study by comparing the occupation-specific incidence to the incidence of occupational asthma in administrative jobs, for which the risk was assumed to be similar to the background population risk. Separate risks were available for males and females, although these were very close to each other and certainly within the limits of random variation. The study by Kogevinas et al. [1999] was used for the relative risk of asthma due to occupational exposure in agriculture, since the results were believed to be more generalizable to agriculture in the rest of the world, especially the developing world. This was because, while the Finnish study was large, prospective and covered all occupations, there was concern that Finnish exposures in agriculture might be atypical of the rest of the world.

The relative risk of asthma morbidity owing to employment in occupational categories was assumed to be approximately equal to the relative risks of asthma mortality, although there is no specific evidence to support or refute this assumption. Those not working and those employed in administration were together considered to be the non-exposed reference category (relative risk = 1). Relative risks and the proportions exposed by occupational category were applied across all age groups from age 15 to 80 years (Table II). These relative risks were used together with estimates of the numbers employed in different occupational groups worldwide to produce the estimates of the attributable fraction and number of deaths due to work-related asthma.

Chronic obstructive pulmonary disease

The paper by Korn et al. [1987] was used as the basis of the relative risk and attributable fraction estimates in this analysis, because it was population-based, used a strict definition of COPD, provides relative risks (estimated using odds ratios) for both men and women covering all workplace exposures, and was based on a large number of participants. The definition of COPD was $FEV_1/FVC < 0.6$, representing reasonably severe disease. Odds ratios were determined for COPD controlled for age, sex, current and lifetime smoking

TABLE III. Annual Risks of COPD Mortality

	Developing countries		Developed countries (AMR-A, EUR-A, WPR-A)		
Relative risk	Men	Women	Men	Women	Industry sub-sector
Non-exposed	1.0	1.0	1.0	1.0	Trade, finance, services, not in workforce
Low	1.2	1.1	1.4	1.2	Agriculture, utilities, transport
High	1.8	1.4	1.8	1.4	Mining, construction, manufacturing

Source: [Korn et al., 1987].

TABLE IV. Annual Silicosis Mortality Risk by Cumulative Exposure to Silica[a]

	A regions				BCDE regions			
	Low exposure[b]		High exposure[c]		Low exposure[d]		High exposure[e]	
Age	Cumulative exposure[f]	Annual risk[g]	Cumulative exposure	Annual risk	Cumulative exposure	Annual risk	Cumulative exposure	Annual risk
20–29	0.08	0.000004	0.42	0.000020	0.21	0.000010	0.63	0.000030
30–44	0.13	0.000006	0.67	0.000032	0.33	0.000016	1.00	0.000048
45–59	0.20	0.000010	1.01	0.000049	0.51	0.000024	1.52	0.000107
60–69	0.23	0.000011	1.14	0.000064	0.57	0.000027	1.71	0.000128
70–79	0.23	0.000011	1.17	0.000067	0.58	0.000028	1.75	0.000132
80+	0.25	0.000012	1.23	0.000074	0.62	0.000029	1.85	0.000143

By region, exposure level and age.
[a]Based on Mannetje et al. [2002].
[b]At 0.2* the United States' PEL (of 0.1 mg/m^3).
[c]At 1.0* the United States' PEL.
[d]At 0.5* the United States' PEL.
[e]At 1.5* the United States' PEL.
[f]Average exposure in mg/m^3·year.
[g]Annual risk of death from silicosis.

history, and city of residence. These odds ratios for COPD morbidity were assumed to apply to COPD mortality.

Korn and co-workers found relative risks of COPD of 1.62 for men and 1.24 for women for a history of exposure to dusts. These relative risks were used as a basis to estimate RRs for high- and low-exposure categories, and slightly different relative risks were used for Low exposure in developed and developing countries (Table III). In developing countries, the great majority of low-exposure employment is in agriculture, where much dust is non-respirable. In developed countries, much of the exposure in the low categories is in industries other than agriculture, where a higher percentage of dust exposure may be respirable and toxic. It was assumed that the relative risks applied across all age categories. Relative risks were applied to the worldwide workforce data to produce estimates of the attributable fraction and number of deaths from COPD arising from work-related exposures.

TABLE V. Annual Asbestosis Mortality Risk by Cumulative Exposure to Asbestos[a]

	A regions				BCDE regions			
	Low exposure[b]		High exposure[c]		Low exposure[d]		High exposure[e]	
Age	Cumulative exposure[f]	Annual risk[g]	Cumulative exposure	Annual risk	Cumulative exposure	Annual risk	Cumulative exposure	Annual risk
20–29	0.1	0.00000	0.5	0.00001	0.3	0.00001	0.8	0.00002
30–44	1.1	0.00002	5.7	0.00011	2.8	0.00006	8.5	0.00017
45–59	8.4	0.00017	42.0	0.00106	21.0	0.00043	63.1	0.00167
60–69	12.2	0.00024	60.9	0.00161	30.5	0.00071	91.4	0.00253
70–79	17.4	0.00035	86.8	0.00238	43.4	0.00110	130.1	0.00405
80+	23.3	0.00050	116.3	0.00345	58.1	0.00153	174.4	0.00617

By region, exposure level and age.
[a]Based on Stayner et al. [1997].
[b]At 0.2* the United States' PEL.
[c]At 1.0* the United States' PEL.
[d]At 0.5* the United States' PEL.
[e]At 1.5* the United States' PEL.
[f]Average exposure in fibers/ml·year.
[g]Annual risk of death from asbestosis.

TABLE VI. Annual Coal Workers' Pneumoconiosis Mortality Risk by Cumulative Exposure to Coal[a]

	A regions				BCDE regions			
	Low exposure[b]		High exposure[c]		Low exposure[d]		High exposure[e]	
Age	Cumulative exposure[f]	Annual risk[g]	Cumulative exposure	Annual risk	Cumulative exposure	Annual risk	Cumulative exposure	Annual risk
20–29	1.7	0.00001	8.3	0.00004	4.2	0.00002	12.5	0.00007
30–44	2.7	0.00001	13.4	0.00007	6.7	0.00004	20.1	0.00011
45–59	4.0	0.00002	20.2	0.00011	10.1	0.00005	30.3	0.00023
60–69	4.6	0.00002	22.8	0.00012	11.4	0.00006	34.1	0.00029
70–79	4.7	0.00002	23.3	0.00012	11.7	0.00006	35.0	0.00030
80+	4.9	0.00003	24.6	0.00013	12.3	0.00006	36.9	0.00034

By region, exposure level and age.
[a]Based on Kuempel et al. [1995].
[b]At 0.2* the United States' MSHA PEL (of 2 mg/m^3).
[c]At 1.0* the United States' MSHA PEL.
[d]At 0.5* the United States' MSHA PEL.
[e]At 1.5* the United States' MSHA PEL.
[f]Average exposure in mg/m^3·year.
[g]Annual risk of death from coal workers pneumoconiosis.

Pneumoconioses

For silicosis mortality, a paper by Mannetje et al. [2002] was used. The information in the paper was used to produce estimates of risk at various cumulative exposures. This exposure-cumulative risk information was converted to average annual risks within different age ranges, for Low and High exposures (as defined previously). There was no information available on gender-specific risks; the Mannetje et al. paper included only males. We have assumed the same risks for males and females (Table IV).

For asbestosis mortality, the observed exposure-response relationship for mortality from asbestos from Stayner et al. [1997] was used. The hazard rates presented in the paper can be interpreted as an incidence rate determined by cumulative exposure. The hazard rates (and thus the annual incidences) for different cumulative exposures (and thus ages) were read directly from Figure 2 in the Stayner paper. Average annual risks were estimated for different age ranges, with risks for cumulative exposures above 150 fibers/ml·year (the highest shown in the paper) extrapolated linearly. Although separate measures of risks were available in the paper for males and females, the cumulative risks provided in the paper, and the annual risk estimates for the broad age-groups used in this analysis, were similar for males and females (Table V).

For mortality from coalworkers' pneumoconiosis, cumulative risk information was available from a study by Kuempel et al. [1995]. The paper considers coal workers' pneumoconiosis mortality at various exposure levels, and Table III of the paper allows risks to be calculated using presented information on the observed number of deaths and the person-years of exposure in various exposure ranges. Using this information, annual risks at various cumulative exposures were determined (Table VI).

TABLE VII. Attributable Fractions (%) for Mortality in 2000 From Asthma and COPD Caused by Workplace Exposure

	Asthma			COPD		
Region[a]	Males	Females	Total	Males	Females	Total
AFR-D	21	15	18	16	5	11
AFR-E	23	18	20	16	5	11
AMR-A	15	9	11	18	3	11
AMR-B	20	8	13	17	3	11
AMR-D	19	7	13	15	2	9
EMR-B	18	5	12	17	2	11
EMR-D	20	10	16	17	3	11
EUR-A	16	7	11	19	4	13
EUR-B	22	14	18	19	6	14
EUR-C	21	12	18	21	6	16
SEAR-B	23	14	18	18	6	13
SEAR-D	23	14	18	16	5	11
WPR-A	17	9	13	21	5	16
WPR-B	22	16	19	19	7	12
World	21	13	17	18	6	12

[a]Regions: AFR, Africa; AMR, Americas; EMR, Eastern Mediterranean; EUR, Europe; SEAR, South-East Asia; WPR, Western Pacific; A, Very low child, very low adult mortality; B, Low child, low adult mortality; C, Low child, high adult mortality; D, High child, high adult mortality; E, High child, very high adult mortality.

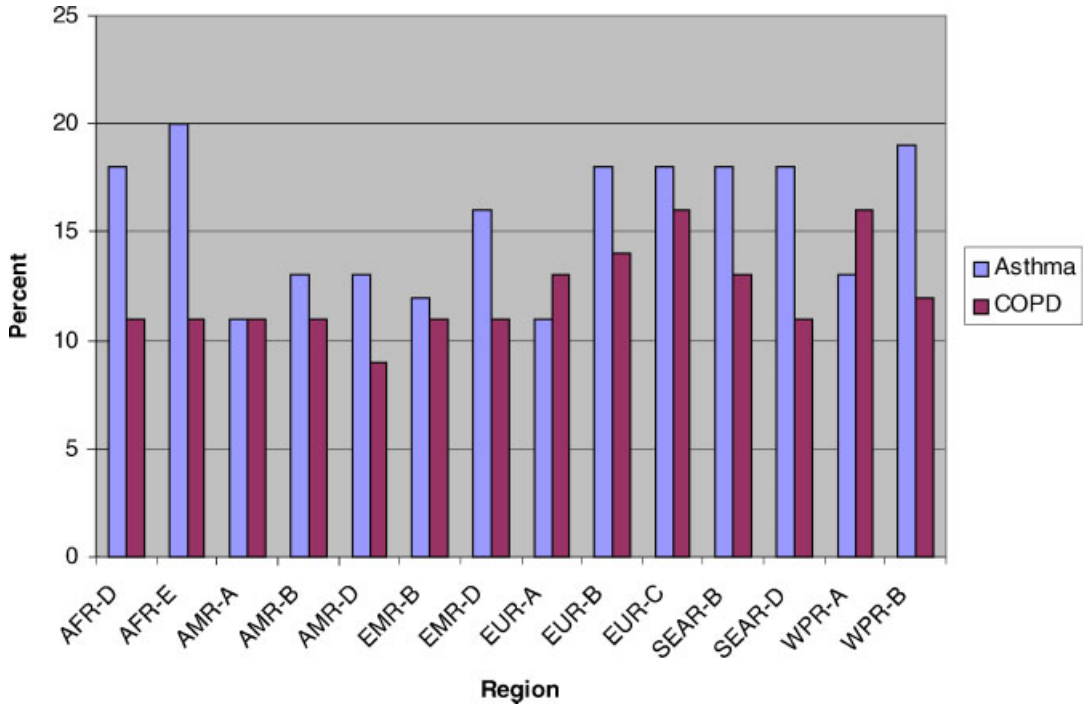

FIGURE 1. Attributable fractions (%) for mortality in 2000 from asthma and COPD caused by workplace exposure. By region.

TABLE VIII. Attributable Fractions (%) for Burden of Disease (DALYs) in 2000 for Asthma and COPD Caused by Workplace Exposure

	Asthma			COPD		
Region[a]	Males	Females	Total	Males	Females	Total
AFR-D	11	7	10	16	5	11
AFR-E	13	9	11	16	5	12
AMR-A	9	4	7	18	3	11
AMR-B	12	4	8	17	3	10
AMR-D	11	3	7	13	1	7
EMR-B	11	2	7	17	2	12
EMR-D	14	6	10	17	3	11
EUR-A	11	4	8	19	4	12
EUR-B	15	8	12	19	6	13
EUR-C	18	8	14	21	6	14
SEAR-B	16	9	13	18	6	13
SEAR-D	17	10	13	16	5	11
WPR-A	12	5	9	21	5	14
WPR-B	15	9	12	19	7	14
World	14	7	11	18	6	13

[a]Regions: AFR, Africa; AMR, Americas; EMR, Eastern Mediterranean; EUR, Europe; SEAR, South-East Asia; WPR, Western Pacific; A, Very low child, very low adult mortality; B, Low child, low adult mortality; C, Low child, high adult mortality; D, High child, high adult mortality; E, High child, very high adult mortality.

Large changes in exposure to these risk factors in different regions (e.g., owing to changes in the coal industry), and the use of absolute hazards, made detailed quantification by age and sex very difficult. For this reason, only overall results in terms of deaths and DALYs are presented for the pneumoconioses.

Attributable Fractions and DALYs

DALYs are ''disability-adjusted life years,'' a weighted estimate of the number of years lived with disability. The weighting refers to the severity of the disability. DALYs require an estimate of the age at which a disease occurs, an estimate of the duration of the disease, and often an estimate of the life expectancy of the person who is ill. Calculation of DALYs also requires a severity weighting that is based on expert judgment of the relative importance of the disability. In the case of premature death due to the disease, the weighting is 1.0 and DALYs are in effect an estimate of the years of life lost due to premature death. (For this project, disability weights were developed in collaboration with Member States by methods including general and specific population surveys.) The DALY thus represents the gap between the current situation and an ideal situation where everyone achieves an agreed standard life expectancy in perfect health. More detail is provided in the introductory paper to this series [Nelson et al., 2005] and elsewhere [Murray and Acharya, 1997; Ezzati et al., 2004].

For asthma and COPD, two attributable fraction measurements were estimated. The mortality attributable fraction measurements represent the percentage of all deaths due to these diseases that arise as a result of workplace exposures. In addition, the number of DALYs due to these conditions was estimated using standard WHO approaches (as described above). By dividing this number by the number of DALYs arising from all causes for a particular disease (asthma or COPD in this case), an estimate was made of the attributable fraction due to both mortality and morbidity arising from occupational exposure. Both attributable fraction estimates are presented here.

For the pneumoconioses, the attributable fraction was assumed to be 100%, since virtually all exposure occurs in an occupational setting. DALYs were estimated using the standard WHO approach based on the estimated number of deaths (estimated as described above). More detail is provided in reference Nelson et al. [2005].

RESULTS

For asthma and COPD, two attributable fraction measurements were estimated. The mortality attributable fraction measurements, which represent the percentage of all deaths due to these diseases that arise as a result of workplace exposures, are presented in Tables VII, XI, and XII and Figure 1. The attributable fraction due to both mortality and morbidity arising from occupational exposure is presented in Table VIII.

It was estimated that 38,000 deaths (23,000 men and 15,000 women) and 1.6 million DALYs result from occupational asthma each year. One quarter to one third of the asthma deaths and DALYs occurred in SEAR-D, and about one fifth occurred in WPR-B. The attributable fraction for mortality from asthma varied between regions from 11% in AMR-A and EUR-A to 20% in AFR-E, with worldwide attributable fractions estimated to be 21% for men and 13% for women (17% overall). The overall attributable fraction for asthma morbidity plus mortality was about two thirds of that for mortality, reflecting the fact that globally a great deal of asthma occurs at younger ages and is non-fatal and non-occupational in origin (Tables VII–X and Figs. 2 and 3).

For COPD mortality, the attributable fraction varied from 9% in AMR-D to 16% in EUR-C and WPR-A. Worldwide mortality attributable fractions for COPD were estimated to be 18% for men and 6% for women (12%

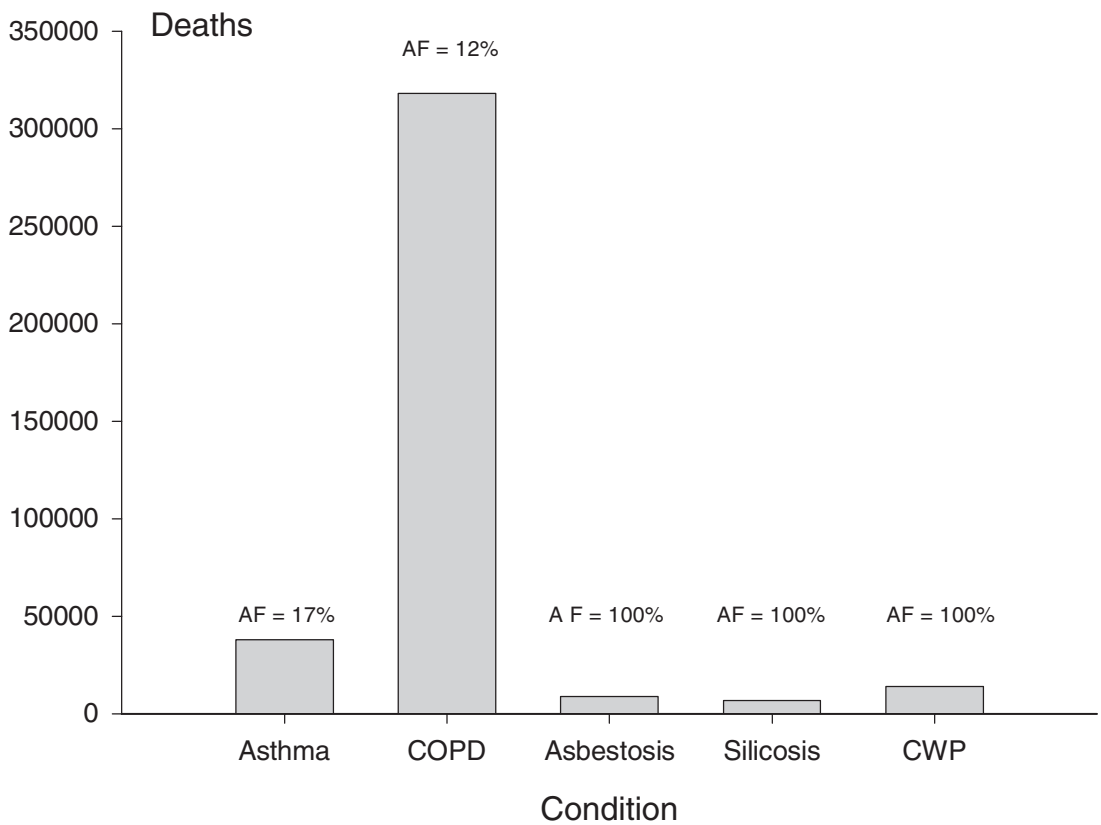

FIGURE 2. Global deaths from asthma, COPD, silicosis, asbestosis, and coal workers' pneumoconiosis (CWP) in the year 2000. AF, attributable fraction.

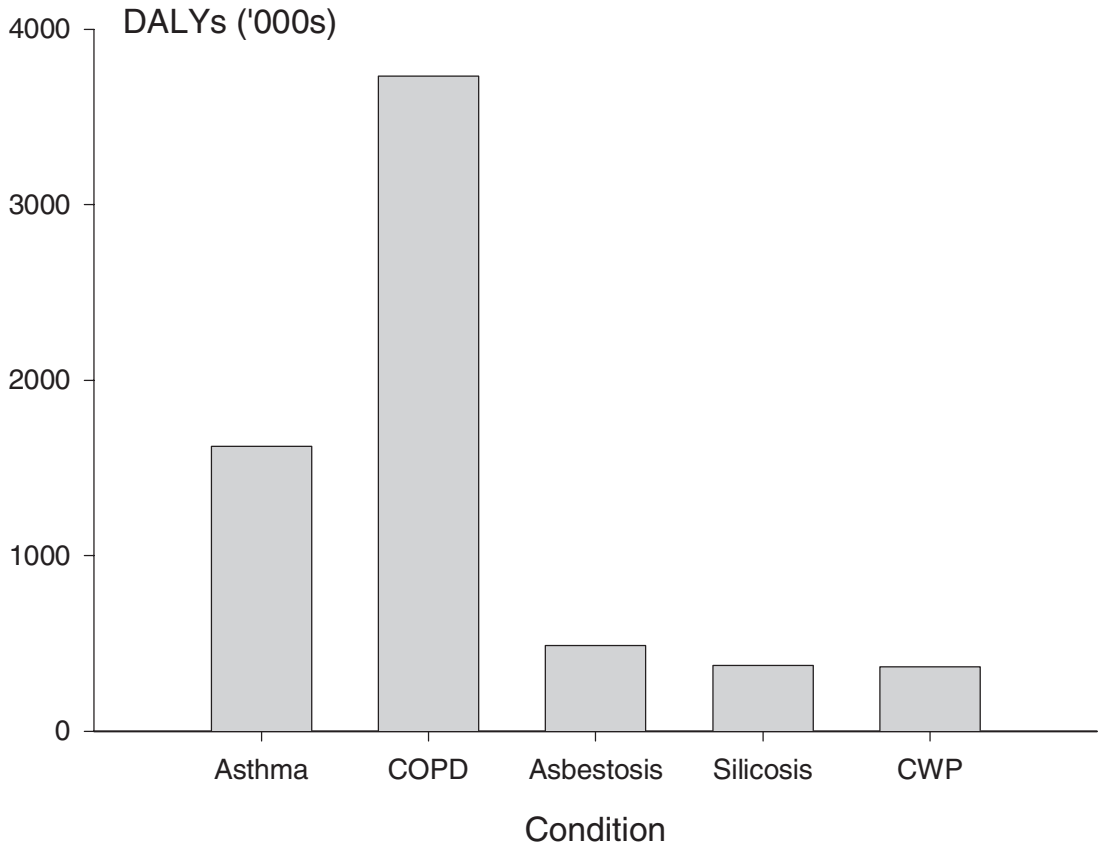

FIGURE 3. DALYs due to asthma, COPD, silicosis, asbestosis, and coal workers' pneumoconiosis (CWP) in the year 2000.

overall) (Table VII). Overall attributable fractions (based on DALYs and reflecting mortality and morbidity) were very similar to the mortality-based fractions (see Tables VII and VIII). The estimated number of deaths was almost an order of magnitude higher for COPD than for asthma, with an estimated 318,000 deaths (240,000 men and 78,000 women) and 3.7 million DALYs resulting from occupational COPD each year. Half of the COPD deaths and half of the DALYs occurred in WPR-B, due in part to the large population of the sub-region and the relatively high employment in mining (Tables VII, IX, and X and Figs. 1 and 2).

For both asthma and COPD, males predominated. Compared to females, males had nearly 50% higher attributable fraction for asthma mortality and three times that for COPD mortality. Similar ratios were seen for the estimated numbers of deaths and DALYs due to these conditions. Asthma deaths were fairly evenly spread among all age groups from 30 to 79 years of age, whereas DALYs predominantly involved persons aged 30–59 years. For COPD, the majority of deaths occurred in persons aged 60 years, whereas DALYs were more evenly spread among all age groups from 30 to 79 years of age (Tables XI and XII).

In addition to asthma and COPD, the estimates for pneumoconioses as a result of exposure to silica, asbestos, and coal mine dust indicate that there may have been approximately 9,000 deaths and 490,000 DALYs from silicosis, 7,000 deaths and 380,000 DALYs from asbestosis, and 14,000 deaths and 370,000 DALYs from coal workers' pneumoconiosis in the year 2000. WPR-A and WPR-B were responsible for about half the deaths and DALYs arising from silicosis and asbestosis, and WPR-B alone was responsible for about half the deaths and DALYs for coal workers' pneumoconiosis (Table XIII and Figs. 1 and 2). More detailed quantification is not presented as it was considered too imprecise.

DISCUSSION

The estimated mortality attributable fractions for asthma of 21% for men and 13% for women (17% overall) are similar to those from two recent reviews, both of which found an occupational attributable fraction of 15% for both sexes combined [Blanc and Toren, 1999; Balmes et al., 2003]. The Finnish study on which most of the occupational relative risk

TABLE IX. Numbers of Deaths (000s) in 2000 From Asthma and COPD Caused by Workplace Exposure

	Asthma			COPD		
Region[a]	Males	Females	Total	Males	Females	Total
AFR-D	0.9	0.7	1.6	4.4	1.2	5.5
AFR-E	1.8	1.4	3.1	5.4	1.4	6.8
AMR-A	0.3	0.3	0.6	11.9	2.0	13.9
AMR-B	0.8	0.5	1.3	8.3	0.9	9.2
AMR-D	0.2	0.1	0.2	0.4	0.0	0.5
EMR-B	0.3	0.1	0.4	1.0	0.1	1.1
EMR-D	1.6	0.7	2.2	7.0	1.2	8.2
EUR-A	0.9	0.5	1.4	16.5	1.8	18.3
EUR-B	1.3	0.7	2.0	5.4	1.1	6.5
EUR-C	2.2	0.6	2.8	12.4	2.1	14.5
SEAR-B	2.2	1.7	3.8	7.5	1.4	8.9
SEAR-D	7.1	4.7	11.8	47.3	12.5	59.8
WPR-A	0.5	0.3	0.8	3.2	0.4	3.6
WPR-B	3.5	2.7	6.2	109.4	51.5	160.9
World	23.4	14.8	38.2	240.3	77.6	317.9

[a]Regions: AFR, Africa; AMR, Americas; EMR, Eastern Mediterranean; EUR, Europe; SEAR, South-East Asia; WPR, Western Pacific; A: Very low child, very low adult mortality; B: Low child, low adult mortality; C: Low child, high adult mortality; D: High child, high adult mortality; E: High child, very high adult mortality.

TABLE X. DALYs (000s) in 2000 From Asthma and COPD Caused by Workplace Exposure

	Asthma			COPD		
Region[a]	Males	Females	Total	Males	Females	Total
AFR-D	63	27	90	43	10	53
AFR-E	84	56	141	57	12	69
AMR-A	37	15	51	147	21	168
AMR-B	98	27	125	115	17	132
AMR-D	16	4	19	6	0	6
EMR-B	18	3	21	20	1	20
EMR-D	74	27	100	75	13	87
EUR-A	41	14	55	176	29	205
EUR-B	30	13	43	75	19	94
EUR-C	32	9	41	135	34	169
SEAR-B	44	26	70	90	21	111
SEAR-D	310	166	476	552	149	701
WPR-A	23	9	33	44	9	53
WPR-B	241	115	356	1485	378	1862
World	1110	511	1621	3020	713	3733

[a]Regions: AFR, Africa; AMR, Americas; EMR, Eastern Mediterranean; EUR, Europe; SEAR, South-East Asia; WPR, Western Pacific; A: Very low child, very low adult mortality; B: Low child, low adult mortality; C: Low child, high adult mortality; D: High child, high adult mortality; E: High child, very high adult mortality.

estimates used in this study were based had higher estimates (29% for men and 17% for women [Karjalainen et al., 2002]), but these estimates were based on Finnish workforce patterns, which are likely to differ from those in most other countries.

Our overall estimates of COPD mortality attributable fraction was 12%, and varied between regions from 9 to 16%. The overall value of 12% is very close to the few published estimates of occupational attributable fraction for COPD of 14% in the United States ([Steenland et al., 2003], based on [Korn et al., 1987]), 14% for men and 5% for women in Finland [Nurminen and Karjalainen, 2001], and 15% in a recent review by the American Thoracic Society [Balmes et al., 2003]. The restrictions of the Korn definition probably results in an underestimate of the true morbidity of COPD attributable to work. A more recent United States study that used a less restrictive definition of COPD and adjusted estimates for several factors, including tobacco use, suggested work resulted in about 22% of COPD cases in Caucasians and 23% in African Americans [Hnizdo et al., 2004].

To evaluate the accuracy of estimates of pneumoconiosis deaths in this study, deaths in AMR A were compared to findings published by others. The estimated number of deaths for silicosis (100) and coal workers' pneumoconiosis (500) for AMR A were about half the annual number of deaths from these conditions reported in the United States for 1999 (silicosis, 187; coal workers' pneumoconiosis, 998)[NIOSH, 2003]. In contrast, predicted asbestosis deaths in AMR A (50) were a significant underestimate (approximately one twentieth) of the annual number of asbestosis deaths reported in the United States in 1999 (1,265)[NIOSH, 2003]. However, it should be noted that the United Surveillance systems probably considerably underestimate the number of work-related diseases cases (by a factor of two or three in one recent study of silicosis) [Rosenman et al., 2003].

These observations suggest that the estimated number of deaths for silica and coal workers' pneumoconiosis are probably in reasonable agreement with available national data, but that the amount of asbestosis is probably considerably underestimated on a global scale. This reflects the difficulty in accurately estimating the prevalence and level of workforce exposures, even in developed countries. It is also possible that while our estimate for asbestosis was too low in AMR A, it may be more accurate in other regions.

There are a number of limitations to our approach. One possible limitation is that we did not attempt to take into account to what extent the risk of developing occupational respiratory diseases diminishes as a result of exposure ceasing. Unfortunately, there is little published data that addresses this issue. Most studies that provide information on risk consider people who have highly variable periods between end of exposure and end of follow-up, with periods varying between zero (still exposed) and many decades.

TABLE XI. Age-Specific Mortality Attributable Fractions, Deaths, and DALYs in 2000 for Asthma and COPD, Males

	Age group (years)						
	15–29	30–44	45–59	60–69	70–79	80–89	All ages Total males
Attributable fractions (%)							
Asthma	23	23	23	22	22	21	21
COPD	17	18	18	18	18	19	18
Deaths (000s)							
Asthma	2.6	4.2	6.3	4.3	3.9	2.1	23.4
COPD	0.3	2.6	28.6	56.3	90.8	61.7	240.3
DALYs (000s)							
Asthma	670	228	144	43	20	5	1 110
COPD	88	564	992	710	517	149	3 020

Another limitation is that the same risks were applied to each region. Many of the risk estimates come from studies based in single countries and may not provide a good representation of other regions, or may not even necessarily be representative of the single countries where they were conducted. We did not attempt to calculate any kinds of bounds of uncertainty, or reasonable ranges, for our estimates, despite the many sources of uncertainty in our approach. In addition to the usual issues of precision, some differences in risk or the biological effect due to exposure might be expected in different ethnic groups, or as a result of different co-exposures in different cultures. Direct risk data are not available for the exposures of interest in most regions, and there is virtually no information available to allow the precise extent of any possible differences to be determined for any of the exposure-disease relationships of interest.

Perhaps the most important limitation to our approach is that we did not have good data on the percentage of the population exposed to the agents of interest. In the case of asthma and COPD the actual agents of interest are not completely known. We used occupation or economic subsector as a very rough surrogate for exposure to occupational asthmagens or dust. In the case of silicosis and asbestosis we used estimates from Europe and applied them to workforce numbers in the rest of the world. These approaches were probably the best available to us, but nonetheless involve considerable imprecision. Uncertainty also arises from changing exposure patterns (probably reducing in developed countries but possibly increasing with increased mechanization in developing countries).

Additional uncertainties were involved in our estimates of pneumoconioses deaths. For these we relied on estimates of the likely cumulative exposure of the exposed population, and estimates of the absolute risk per unit of cumulative exposure. It is not entirely clear if the additional uncertainties and complexity resulting from our attempt to estimate

TABLE XII. Age-Specific Mortality Attributable Fractions, Deaths and DALYs in 2000 for Asthma and COPD, Females

	Age group (years)						
	15–29	30–44	45–59	60–69	70–79	80–89	All ages total female
Attributable fraction (%)							
Asthma	13	14	14	13	13	12	13
COPD	6	5	5	6	6	6	6
Deaths (000s)							
Asthma	1.8	2.8	3.8	2.2	2.3	1.9	14.8
COPD	0.1	0.7	6.5	12.8	27.7	29.9	77.6
DALYs (000s)							
Asthma	228	95	81	28	15	5	511
COPD	45	133	149	152	166	69	713

TABLE XIII. Deaths (000s) and DALYs (000s) in 2000 From Silicosis, Asbestosis, and Coal Workers' Pneumoconiosis

	Deaths			DALYs		
Region	**Silicosis**	**Asbestosis**	**CWP[a]**	**Silicosis**	**Asbestosis**	**CWP[a]**
AFR-D	0.2	0.2	0.0	8	9	0
AFR-E	0.2	0.2	0.1	10	12	3
AMR-A	0.1	0.0	0.5	6	3	12
AMR-B	0.5	0.2	0.0	20	11	1
AMR-D	0.1	0.0	0.0	3	1	0
EMR-B	0.2	0.1	0.0	11	5	0
EMR-D	0.3	0.2	0.0	18	14	0
EUR-A	0.2	0.1	2.1	11	5	48
EUR-B	0.3	0.2	0.9	18	11	24
EUR-C	0.7	0.3	2.4	47	22	75
SEAR-B	0.3	0.2	0.1	10	8	1
SEAR-D	1.1	1.0	0.8	78	78	27
WPR-A	2.4	2.3	0.1	97	95	3
WPR-B	2.3	1.6	6.7	149	104	172
World	8.8	6.7	13.8	486	376	366

Regions: AFR, Africa; AMR, Americas; EMR, Eastern Mediterranean; EUR, Europe; SEAR, South-East Asia; WPR, Western Pacific; A: Very low child, very low adult mortality; B: Low child, low adult mortality; C: Low child, high adult mortality; D: High child, high adult mortality; E: High child, very high adult mortality.

cumulative exposures for exposed populations resulted in better estimates than those that might have been obtained by a somewhat simpler approach.

CONCLUSIONS

The aim of this study was to estimate the occupational attributable fractions for asthma and COPD, along with the number of associated deaths and DALYs. In addition, the number of deaths and DALYs from the three main pneumoconioses were estimated. The results indicate that non-malignant respiratory diseases arising from occupational exposures are an important cause of death and disability worldwide. Most of these cases should be preventable by adopting better health and safety approaches, particularly through improved engineering and working conditions.

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