Chapter 21

Selected occupational risk factors

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Summary

Many of the 2.9 billion workers across the globe are exposed to hazardous risks at their workplaces. This chapter examines the disease and injury burden produced by selected occupational risk factors: occupational carcinogens, airborne particulates, noise, ergonomic stressors and risk factors for injuries. Owing primarily to lack of data in developing countries, we were unable to include important occupational risks for some cancers, reproductive disorders, dermatitis, infectious diseases, ischaemic heart disease, musculoskeletal disorders (MSDs) of the upper extremities, and other conditions such as workplace stress. Mesothelioma and asbestosis due to asbestos exposure, silicosis and coal workers' pneumoconiosis are almost exclusively due to workplace exposure, but limitations in global data precluded a full analysis of these outcomes.

The economically active population (EAP) aged ≥15 years, which includes people in paid employment, the self-employed, and those who work to produce goods and services for their own household consumption, were considered the group at risk of exposure to occupational hazards. Both formal and informal sectors of employment are included in the EAP, but child labour was excluded. Exposure was quantified based on the economic sector (where people do the work) and on occupation (what people do). Our sources of data to delineate categories of exposed workers included economic databases and publications of the International Labour Organization (ILO) and the World Bank and the published scientific literature. For most risk factors the workers were grouped into high- and low-exposure categories, and the exposed population was distributed by age, sex and subregion.[1] Risk estimates for the occupational hazards were obtained from the published epidemiological literature, particularly from studies of large populations, reviews and meta-analyses when available.

The occupational risk factors in our study accounted for an estimated 37% of back pain, 16% of hearing loss, 13% of chronic obstructive

pulmonary disease (COPD), 11% of asthma, 8% of injuries, 9% of lung cancer and 2% of leukaemia. These work-related risks caused 775 000 deaths worldwide in 2000. There were five times as many deaths in males as in females (647 000 vs 128 000). The leading occupational cause of death among the six risk factors was unintentional injuries (41%) followed by COPD (40%) and cancer of the trachea, bronchus or lung (13%). Workers who developed outcomes related to the occupational risk factors lost about 22 million years of healthy life. By far the main cause of years of healthy life lost (measured in disability-adjusted life years [DALYs]), within occupational diseases, was unintentional injuries with 48% of the burden. This was followed by hearing loss due to occupational noise (19%) and COPD due to occupational agents (17%). Males experienced almost five times greater loss of healthy years (DALYs) than females. Low back pain and hearing loss have in common the fact that they do not directly produce premature mortality, but they cause substantial disability and have multiple consequences for the individual and society, particularly for workers suffering the outcomes at an early age.

The major source of uncertainty in our analysis was characterizing exposure, which was based solely on economic subsectors and/or occupations and involved a large number of extrapolations and assumptions. High-quality exposure data are lacking, especially in developing countries, and European and American exposure estimates were thus applied in many instances in developing regions. This extrapolation could have substantial impact on the accuracy of analysis for the developing regions if exposures, as usually occur, vary from place to place and over time. Diseases with long latency (e.g. cancers) are more susceptible to the assumptions and extrapolations. In addition to problems produced by the length of the latency period, the magnitude of the excess risk may vary depending on the age of the person when exposure began, the duration and strength of exposure and other concomitant exposures. The turnover of workers is another issue that affects both exposure and risk assessment. Sources of uncertainty in hazard estimates (relative risk and mortality rates) include variations determined from the literature (once again caused by the use of different exposure proxies), extrapolations to regions with different working conditions, the application to females of risk measures from male cohorts, and the application of the same relative risk values to all age groups (e.g. carcinogens). Restricting the analysis to persons aged ≥15 years excludes the quantification of child labour. The exclusion of children in the estimation was due to the wide variation in the youngest age group for which countries reported economic activity rates (EARs). In addition to inconsistent data on EARs for children, there were virtually no data available on their exposure to occupational risk factors or the relative risks of such exposures. Specific, focused research on children is needed to quantify the global burden of disease due to child labour and the resulting implications.

1. Introduction

Throughout the world, most adults—and many children—spend much of their waking hours at work. Work provides a number of economic and other benefits. At the same time, people at work face a variety of hazards owing to chemicals, biological agents, physical factors, adverse ergonomic conditions, allergens, a complex network of safety risks, and many and varied psychosocial factors. In addition to injuries, more than 100 occupational diseases have been classified according to the tenth revision of the International Classification of Diseases and Related Health Problems (ICD-10). Broadly, these include respiratory, musculoskeletal, cardiovascular, reproductive, neurotoxic, skin and psychological disorders, hearing loss and cancers.

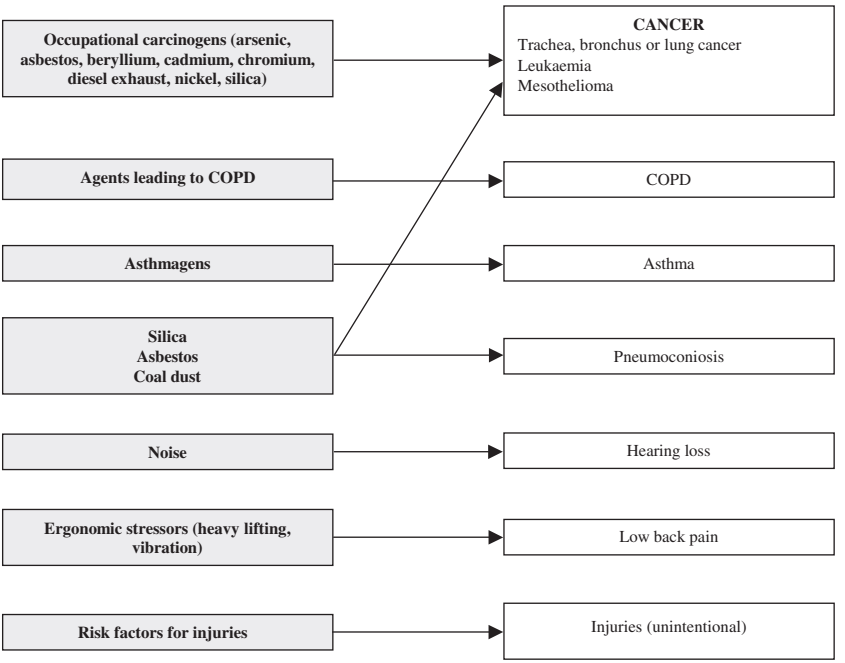
Of the wide variety of work-related exposures, only the most widespread are evaluated here. Other criteria for selection of risk factors include adequacy of exposure information and the applicability of health outcome data to all regions of the globe, and the inclusion of the relevant health outcomes in the global burden of disease (GBD) database of diseases and injuries.

Exposure to occupational hazards can adversely affect the human body. Adverse effects range from asymptomatic physiological and biochemical changes to symptoms of illness, to diagnosed diseases and, finally, to death. For some risk factors there is a very clear connection between the exposure and the disease. For example, the primary route of exposure to airborne particulates, gases and vapours is inhalation, whereby these agents gain access to the respiratory system and are either deposited (in the case of particulates) or enter the circulatory system (gases and vapours). Many risk factors cause more than one type of outcome of interest. For example, exposure to asbestos can result in malignant conditions of the lung and the pleura, malignant conditions of the peritoneum, and nonmalignant conditions of the lung (asbestosis). Some exposures, such as occupational noise, are well characterized. Others have not been well characterized or are multi-faceted, but the condition they cause is clear (such as occupational injuries).

Following a general description of methods and data sources, individual sections provide details of specific aspects of methodology and results for each of the selected occupational health risk factors that were analysed: occupational carcinogens, occupational airborne particulates, occupational noise, occupational ergonomic stressors and occupational risk factors for injuries.

In this study, the term "occupational risk factor" is defined as a chemical, physical, biological or other agent that may cause harm to an exposed person in the workplace and is potentially modifiable. Figure 21.1 shows the selected risk factors along with related health outcomes. Owing to complex etiology and lack of data, a different approach was developed for some conditions such as asthma and low back pain, using

Figure 21.1 Relationship between occupational risk factors and outcomes[a]



[a] Only selected relationships were quantified.

occupation as a proxy for exposure to the causative agents. The utility of this work as a risk-based framework has thus been limited.

1.1 Excluded exposures and outcomes

No effects specific to the hazards associated with child labour are addressed in this report owing to a lack of data. Other excluded risks or outcomes include respiratory diseases other than COPD and asthma; some infectious diseases; less widespread cancers and carcinogens (e.g. bladder cancer and cancer of the liver); MSDs such as carpal tunnel syndrome; intentional injuries in the workplace; organ and systemic diseases resulting from occupational exposure to solvents, pesticides and heavy metals such as lead or mercury; maternal and perinatal conditions resulting from occupational exposures; skin disorders, including dermatitis, dermatosis and melanoma; ischaemic heart disease and other outcomes associated with work-related stress.

Malignant mesothelioma of the pleura and peritoneum is virtually uniquely due to asbestos exposure. Occupational dusts can also result in

nonmalignant respiratory diseases other than 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 dust, respectively. While evidence for a causal relationship is strong, lack of data on accumulated exposure, especially in developing countries, restricted the ability to provide a detailed assessment of attributable mortality and disease burden for these outcomes. Preliminary estimates are provided in the note under Table 21.62 in Section 7.

Because of lack of available data and difficulties in quantification, it was not possible to conduct a global quantitative analysis for the health consequences of stress at work. Overall, the evidence indicates that incidence of stress-related cardiovascular disease is likely to be higher in the blue-collar occupations when the following factors are present: restricted discretion, shiftwork (particularly nightshift), effort-reward imbalance, high demands, poor psychosocial work environment, social isolation, physical inactivity or occupational violence. These risk factors may be interactive. Nurminen and Karjalainen (2001) estimated for Finland an attributable fraction of 16.9% (18.9% for men and 9.1% for women) for ischaemic heart disease due to the combined occupational risk factors of shiftwork, noise, and exposure to engine exhaust and environmental tobacco smoke. For ischaemic heart disease, Steenland et al. (2003) used an attributable fraction of 6–18% for individuals in the United States of America aged 24–64 years, based on the combined effects of noise, job strain (stress), shiftwork and environmental tobacco smoke. Occupational dermatitis accounts for about 10% of all occupational disease in the United States (Emmett 2002) but exposure data are lacking at global level.

Although there were adequate global data to analyse the risks to health care workers from contaminated sharps (e.g. syringe needles and scalpels), the full analysis has been omitted from this chapter. Since health care workers make up only 0.6% of the global population, the contribution to hepatitis B, hepatitis C and HIV/AIDS infections on a global level was close to zero. However, health care workers are at high risk of preventable infection from bloodborne pathogens, owing to occupational exposure to infected blood and body fluids.

1.2 Choice of theoretical-minimum-risk exposure distributions

For some occupational hazards, a theoretical minimum exposure of zero is not possible, as there is some low-level environmental exposure. Two occupational risk factors (carcinogens and airborne particulates) involve workplace exposure at concentrations higher than the environmental or background levels of these substances. For noise, the theoretical minimum was defined as less than 80 dBA, a level found not to have an increased risk of causing hearing loss (NIOSH 1998). For the other risk factors (ergonomic stressors and work-related risk factors for injuries), a category of workers with the lowest risks was identified as the com-

parison group for occupational categories of workers with higher risks. Thus, the theoretical minimum risk corresponds to "no occupational exposure above levels found in the defined comparison group". Selection of a defined comparison group provides a realistic basis for a theoretical minimum, but it does not establish the lowest rate of adverse outcome that could ever be experienced. While it is not expected that occupational exposures will be eliminated in the foreseeable future, it is possible to control exposures through recognized industrial hygiene practices. Engineering controls (including prevention, substitution of materials, process automation, enclosure, process elimination, isolation of workers and process change) constitute effective methods of minimizing exposures (Burton 1997). Administrative controls (such as education and training, work practice controls, worker rotation, maintenance and housekeeping) provide another means of risk reduction.

1.3 Data sources

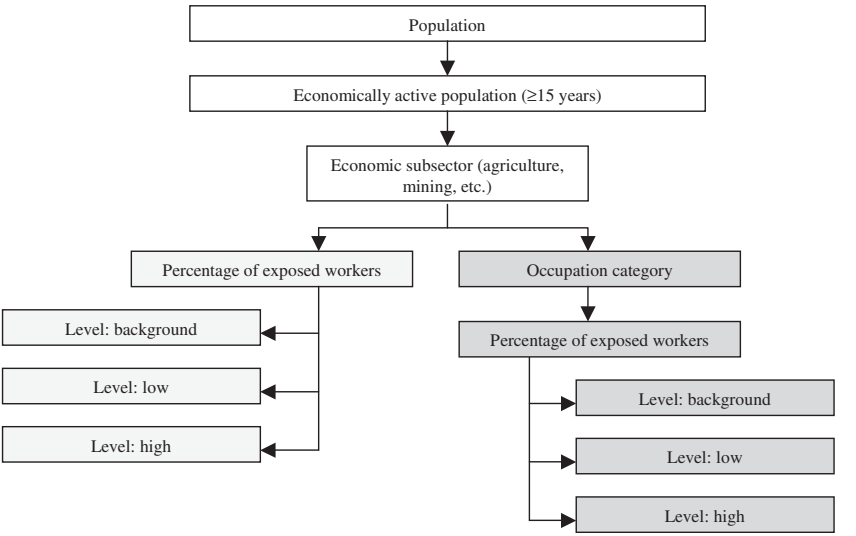
A systematic assessment of the literature was carried out to identify studies on occupational exposures and health outcomes. This included searching Medline, occupational health and safety databases such as OSHROM and NIOSHTIC and databases of various organizations; reviewing relevant references cited in publications identified through the initial literature search and of references cited in these secondary references; communicating with relevant experts; and seeking other information recommended by referees following the initial review of the draft manuscript. PubMed was searched using keywords for exposures and health outcomes, including (separately and in combination, with no limit on year of publication): exposure, occupational, cancer, carcinogen, silica, silicosis, benzene, asbestos, asbestosis, pneumoconiosis and developing country. Names of regions (e.g. Africa, Asia) and specific countries were also used as keywords. A systematic search was conducted using Ovid Healthstar and the former HealthSTAR databases, covering the period 1975–2001. Keywords included: asbestos; asthmagens; chronic obstructive lung disease; cancer and diesel exhaust; arsenic; benzene and leukaemia; ionizing radiation and leukaemia; back (for low back pain); injury; accidents; ergonomics; and hearing impairment and noise.

Studies of large populations, reviews and meta-analyses were specifically sought. Reports and publications were critically assessed to determine their methodology, validity and the characteristics of the population studied.

1.4 Estimating risk factor levels

In general, since the types of risk factor to which workers are exposed are primarily influenced by where the work is performed (economic sector) and the type of work they do (occupation), the assessment of proportion of population exposed in each subregion was based on (Figure 21.2):

Figure 21.2 Exposure assessment overview



- economic sector distribution (total nine sectors), used for carcinogens and agents leading to COPD) (Equation 1);
- occupational distribution (occupation within economic sector) (total seven occupations), used for asthmagens, noise and ergonomic stressors (Equation 2); and
- exposure could not be estimated for injury risk factors, and thus estimates of disease burden were made based on the reported rates of the outcome (injury mortality) rather than on exposure.

$$\mathrm{PEP}(r,g,a) = \mathrm{EAR}(r,g,a) \times \mathrm{OT}(r) \times \mathrm{EPF}(r) \sum_{i=1}^{9} (\mathrm{PW}(\mathrm{es}(r,g)i) \times \mathrm{PEW}(\mathrm{es}(r,g)i) \quad (1)$$

$$\mathrm{PEP}(r,g,a) = \mathrm{EAR}(r,g,a) \times \mathrm{OT}(r) \times \mathrm{EPF}(r) \sum_{i=1}^{7} (\mathrm{PW}(\mathrm{oc}(r,g)i) \times \mathrm{PEW}(\mathrm{oc}(r,g)i) \quad (2)$$

where

PEP(r,g,a) = proportion of the population occupationally exposed to a specific risk factor in that subregion, by sex and age, at low or high level

EAR(r,g,a) = economic activity rate, by subregion, sex and age

OT(r) = occupational turnover, if applicable, to account for workers exposed in the past, by subregion

EPF(r) = exposure partitioning factor, by subregion, to delineate proportion exposed at low or at high levels

PW(es(r,g)i) = proportion of the population working in economic subsector (i), by subregion and sex

PEW(es(r,g)i) = proportion of workers in economic subsector (i) with exposure to the specific risk factor, by subregion and sex

PW(oc(r,g)i) = proportion of the population working in occupational category (i), by subregion and sex

PEW(oc(r,g)i) = proportion of workers in occupational category (i) with exposure to the specific risk factor, by subregion and sex

The differences between the two equations are the term PW(es(r,g)i) in Equation 1, which is used when exposure data are available by economic sector, and the term PW(oc(r,g)i) in Equation 2, which is used when exposure data are available by occupational category. Occupational turnover (OT), defined as "the rate of replacement of workers due to departures from the workplace", was utilized for carcinogens because health effects due to these risk factors occur many years after exposure (latency) and it was therefore necessary to know how many persons had been exposed in the past to these risk factors. The effects of noise, ergonomic stressors and risk factors for injuries are relatively immediate; latent effects were therefore not a consideration for these risk factors. Additional detail on each term is provided below in the text, and is also summarized in Table 21.1.

The primary data sources used for the exposure assessment and the determination of some of the risk measures (see Table 21.2) included: the World Bank, ILO, the European Union carcinogen exposure (CAREX) database, published literature on prevalence and level of exposure to occupational risk factors, and published literature on epidemiology of health outcomes linked to occupational risk factors, as cited in the relevant sections for each risk factor.

Economic activity rate

EAR is defined as the proportion of the economically active population (EAP) among the overall population. EAR was calculated for each region and sex in persons aged ≥15 years, and used to estimate the proportion of the population potentially exposed to occupational risks. EAR provides the most comprehensive accounting of persons who may be exposed to occupational risks, as it includes people in paid employment, the self-employed, and people who work to produce goods and services

Table 21.1 Summary of determinants of population exposure to occupational risk factors

Term	*Comments*	*Application*	*Primary data sources*
EAP	Economically active population is calculated by application of the EAR to the national population	Used for injuries	ILO (2002a)
EAR	Economic activity rate, calculated as the EAP in each age group compared to the number of people in that age group, males and females ≥15 years	Used in exposure assessments of all risk factors	ILO (2002a)
PW(es)	Proportion working, i.e. fraction of EAP in economic sector. Data on distribution of EAP into three economic sectors (agriculture, industry, service) or nine economic subsectors. Country-level data were weighted by working-age population to develop subregional averages	Used for carcinogens, selected airborne particulates (agents leading to COPD)	World Bank (2001)
PW(oc)	Proportion working, i.e. fraction of EAP in occupational category. Country-level data for about 31 countries were weighted by working-age population to develop subregional averages. Owing to lack of country-level data, EMR-B was based on EMR-D data, EUR-C was based on EUR-B data and WPR-A was based on AMR-A data	Used for asthmagens, noise, ergonomic stressors	ILO (1995a); World Bank (2001)
PEW	Proportion exposed working, i.e. fraction of population working in economic sector (or in an occupational category) with exposure to risk factor. Owing to data limitations, data from developed countries were usually applied to developing countries, verified where possible by data on specific risk factors from specific countries	PEW(es): carcinogens PEW(es): selected airborne particulates (agents leading to COPD) PEW(oc): selected airborne particulates (asthmagens) PEW(oc): noise PEW(oc): ergonomic stressors	FIOH (1999); Kauppinen et al. (2000) FIOH (1999); Kauppinen et al. (2000); Korn et al. (1987); USEIA (2001) Karjalainen et al. (2002); Kogevinas et al. (1999) NIOSH (1998) Leigh and Sheetz (1989)
EPF	Exposure partitioning factor, i.e. proportion of PEW with low- or high-level exposure to risk factor	Carcinogens, selected airborne particulates, noise	NIOSH (1998, 1999, 2000a); Pearce et al. (1994); Yin et al. (1987)
OT	Occupational turnover factor. Used only for risk factors for which latent effects must be considered (carcinogens, selected airborne particulates). A factor of 4 was estimated on the basis of published data on labour turnover rates, published cohort data and modelling of cohorts with various mean lengths of exposure. Higher value used for specific regions for coal mining	Carcinogens	K. Steenland, personal communication, 2002

Table 21.2 Key sources, data supplied and special characteristics of the sources used to estimate exposure

Source	*Data supplied*	*Comments*
ILO (1995a, 2000, 2002b)	Employment in economic sectors and subsectors, and in occupations within economic sectors; EARs by age and sex for selected countries	Collected by national EAP surveys. Differences among and within countries (e.g. applicable ages, time period covered) limit international comparability
World Bank (2001)	Distribution of EAP (males and females) in agriculture, industry and services; participation of females in the EAP	Based on ILO data
FIOH (1999); Kauppinen et al. (2000)	Proportion of the working population with occupational exposure to carcinogens in the European Union, by economic sector and subsector, at the 3-digit classification level	Applicable to A subregions, extrapolated to B, C, D and E subregions
EIA (2001)	Country-level data on coal production	
ILO (1995b)	Country-level data on number of coal miners	
NIOSH (1991, 1998); USDHHS (1986)	Data on noise exposure of American workers	Applicable to A subregions, extrapolated to B, C, D and E subregions

for their own household consumption. According to ILO (2002b), the majority of those who work in the informal sector are included in the "employed" category, and the remainder are in the "unemployed" category; thus, the informal sector workers are included in this analysis. At the same time, persons in precarious or contingent employment often face an increased risk of occupational health and safety hazards, which are not quantified here (Quinlan 2002). The use of EAR for persons aged ≥15 years excludes children under 15 who work.

Estimates and projections of EAP were developed by ILO by applying estimates and projections of activity rates, by sex and age group, to the population estimates and projections assessed by the United Nations (ILO 1996). ILO estimates and projections of economic activity are taken primarily from population censuses and/or sample surveys carried out between 1975 and 1994. ILO also takes data from specific publications by national, interregional and/or international institutions.

Country-level data from the ILO electronic database were used to develop subregion-specific EARs for ages 15 years and above, for males and females (Table 21.3). EARs were estimated for 60–69-year olds by using data for 60–64-year olds. Data for people aged ≥65 years were

Table 21.3 Economic activity rates by subregion, sex and age group

		Age group (years)						
Subregion[a]	*Sex*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*	*Total ≥15*
AFR-D	Male	0.77	0.97	0.95	0.85	0.65	0.33	0.85
	Female	0.50	0.61	0.62	0.48	0.28	0.14	0.53
AFR-E	Male	0.78	0.97	0.95	0.86	0.66	0.33	0.86
	Female	0.64	0.72	0.69	0.54	0.36	0.18	0.65
AMR-A (95%)	Male	0.70	0.93	0.87	0.50	0.13	0.07	0.73
	Female	0.64	0.81	0.71	0.32	0.07	0.04	0.59
AMR-B	Male	0.78	0.97	0.89	0.66	0.33	0.17	0.82
	Female	0.46	0.53	0.39	0.20	0.07	0.04	0.42
AMR-D	Male	0.71	0.98	0.96	0.86	0.61	0.31	0.82
	Female	0.38	0.48	0.39	0.29	0.17	0.09	0.39
EMR-B (90%)	Male	0.66	0.97	0.92	0.74	0.45	0.23	0.79
	Female	0.33	0.37	0.26	0.18	0.09	0.05	0.31
EMR-D (40%)	Male	0.73	0.97	0.94	0.76	0.44	0.22	0.82
	Female	0.37	0.43	0.37	0.25	0.12	0.06	0.37
EUR-A	Male	0.66	0.96	0.84	0.35	0.05	0.03	0.68
	Female	0.59	0.74	0.56	0.14	0.02	0.01	0.47
EUR-B	Male	0.72	0.96	0.80	0.41	0.22	0.11	0.74
	Female	0.56	0.77	0.59	0.23	0.12	0.06	0.54
EUR-C	Male	0.72	0.97	0.89	0.30	0.11	0.06	0.74
	Female	0.61	0.94	0.74	0.17	0.05	0.03	0.58
SEAR-B	Male	0.74	0.98	0.94	0.73	0.44	0.22	0.83
	Female	0.55	0.70	0.65	0.44	0.21	0.11	0.58
SEAR-D (95%)	Male	0.77	0.98	0.95	0.72	0.53	0.27	0.85
	Female	0.45	0.57	0.50	0.32	0.16	0.08	0.47
WPR-A	Male	0.67	0.97	0.95	0.69	0.30	0.15	0.76
	Female	0.57	0.70	0.67	0.36	0.13	0.07	0.52
WPR-B (90%)	Male	0.81	0.98	0.92	0.61	0.29	0.15	0.84
	Female	0.77	0.89	0.67	0.29	0.09	0.05	0.71

[a] When data were not available for all countries, the percentage of the regional working age population (≥15 years) represented by data is indicated. Some very small countries, e.g. Grenada, were not included in these calculations.

Source: ILO (2002a).

applied to the 70–79 age group. The ≥80 age group was estimated at one half of the rate for the ≥65 age group (by comparison with country-level data, which is reported by some countries for elderly workers) (ILO 2001).

PROPORTION OF THE POPULATION WORKING IN AN ECONOMIC SECTOR OR OCCUPATIONAL CATEGORY

The distinction between "where people work", i.e. economic sector and "what they do", i.e. occupation, is important in exposure characterization. For example, within the economic subsector of manufacturing there

Table 21.4 Illustration of the ISIC classification system used in exposure assessment

Economic sector	*Economic subsectors*	*Occupational categories*						
		Professional	*Administration*	*Clerical*	*Sales*	*Service*	*Agriculture*	*Production*
Agriculture								
Industry	Mining							
	Manufacturing							
	Electrical							
	Construction							
Services	Trade							
	Transport							
	Finance							
	Services							

Source: ILO 1987.

are people who work as production workers, but also people who work as clerical or sales people (Table 21.4). EAP was used for injuries. EAP by economic sector and subsector was used for carcinogens and agents leading to COPD, because available data do not distinguish exposures by occupational category within economic sectors. For asthmagens, noise and ergonomic stressors, the analyses were conducted on the basis of exposure by occupational category within economic sectors.

The approach used here is based on the International Standard Industrial Classification of All Economic Activities (ISIC), an economic classification system of the United Nations, which organizes all economic activities by economic sectors and relevant subgroupings (ILO 1987; UN 2000). The ISIC system is used almost universally by national and international statistical services to categorize economic activity, and therefore allowed us to make global comparisons. Table 21.4 illustrates the ISIC classification scheme of economic sectors, economic subsectors and occupational categories that were used to estimate exposures to workers in this project. We did not subdivide agriculture into economic subsectors.

Economic sector

For each subregion, a weighted proportion of working men and women (EAP) in each of the three economic sectors was constructed (Table 21.5) (World Bank 2001, data from 1990 and 1996–1998). Economic sector employment data were used to subdivide the number of workers in industry into the economic subsectors of mining, manufacturing, electricity (and other utilities) and construction. In a similar manner, the data for the service sector were subdivided into the economic subsectors of trade, transport, finance and services. The agriculture sector was not subdivided.

Table 21.5 EAP distribution in economic sectors and subsectors, by subregion and sex

			Industry				*Services*				
Subregion[a]	*Sex*	*Agriculture*	*Mining*	*Manufacturing*	*Electricity*	*Construction*	*Trade*	*Transport*	*Finance*	*Services*	*Sum*
AFR-D (20%)	Male	0.55	0.01	0.09	0.01	0.04	0.06	0.04	0.03	0.16	1.00
	Female	0.68	0.00	0.05	0.00	0.01	0.06	0.03	0.02	0.16	1.00
AFR-E (20%)	Male	0.55	0.01	0.09	0.01	0.04	0.05	0.03	0.03	0.18	1.00
	Female	0.65	0.00	0.03	0.00	0.00	0.05	0.02	0.02	0.22	1.00
AMR-A (95%)	Male	0.05	0.01	0.21	0.01	0.09	0.21	0.06	0.10	0.26	1.00
	Female	0.02	0.00	0.09	0.00	0.01	0.23	0.03	0.14	0.47	1.00
AMR-B	Male	0.20	0.01	0.15	0.04	0.08	0.10	0.05	0.25	0.12	1.00
	Female	0.12	0.00	0.12	0.00	0.01	0.14	0.01	0.05	0.55	1.00
AMR-D (70%)	Male	0.07	0.01	0.16	0.01	0.11	0.18	0.07	0.09	0.30	1.00
	Female	0.03	0.00	0.12	0.00	0.00	0.31	0.01	0.06	0.46	1.00
EMR-B (5%)	Male	0.15	0.01	0.16	0.03	0.11	0.15	0.08	0.05	0.27	1.00
	Female	0.09	0.00	0.09	0.00	0.01	0.20	0.05	0.09	0.47	1.00
EMR-D (20%)	Male	0.45	0.00	0.11	0.01	0.07	0.30	0.00	0.03	0.02	1.00
	Female	0.68	0.00	0.10	0.01	0.01	0.20	0.00	0.00	0.01	1.00
EUR-A	Male	0.06	0.01	0.27	0.01	0.11	0.01	0.00	0.05	0.48	1.00
	Female	0.05	0.00	0.15	0.00	0.02	0.01	0.01	0.12	0.64	1.00

continued

Table 21.5 EAP distribution in economic sectors and subsectors, by subregion and sex (*continued*)

			Industry				*Services*				
Subregion[a]	*Sex*	*Agriculture*	*Mining*	*Manufacturing*	*Electricity*	*Construction*	*Trade*	*Transport*	*Finance*	*Services*	*Sum*
EUR-B (70%)	Male	0.29	0.02	0.20	0.02	0.08	0.07	0.04	0.07	0.20	1.00
	Female	0.44	0.00	0.16	0.01	0.01	0.03	0.01	0.04	0.30	1.00
EUR-C (35%)	Male	0.21	0.04	0.14	0.03	0.15	0.05	0.20	0.12	0.06	1.00
	Female	0.16	0.03	0.12	0.02	0.04	0.12	0.24	0.12	0.15	1.00
SEAR-B (30%)	Male	0.46	0.01	0.12	0.01	0.07	0.14	0.06	0.01	0.13	1.00
	Female	0.45	0.00	0.15	0.00	0.01	0.22	0.01	0.01	0.15	1.00
SEAR-D (80%)	Male	0.53	0.02	0.13	0.02	0.03	0.01	0.06	0.04	0.17	1.00
	Female	0.80	0.01	0.10	0.00	0.01	0.00	0.00	0.02	0.06	1.00
WPR-A	Male	0.05	0.00	0.24	0.01	0.13	0.17	0.10	0.09	0.21	1.00
	Female	0.06	0.00	0.17	0.00	0.04	0.27	0.04	0.11	0.31	1.00
WPR-B (95%)	Male	0.44	0.03	0.14	0.01	0.05	0.09	0.06	0.02	0.16	1.00
	Female	0.40	0.01	0.12	0.01	0.01	0.17	0.06	0.06	0.17	1.00

[a] When data were not available for all countries, the percentage of the regional working age population (≥15 years) represented by data is indicated. Some very small countries, e.g. Grenada, were not included in these calculations.

Note: AFR-D and AFR-E (combined), EMR-B, EMR-D and SEAR-D data are based on 1990 employment data from the World Bank world development indicators, as EAP data were very limited. All others are taken from 1996–1998 World Bank EAP data. Subregional averages were calculated using country values weighted by the working-age population.

Occupational category

Regional tables of occupation within economic sector distributions were constructed using the number of employed people by occupation and economic sector. For comparison purposes, data were obtained from one source (ILO 1995a). For a subregion with only one country represented, the distribution of occupation within economic sector was assumed to represent the regional employment patterns. Where more than one country was represented, a weighted average was constructed. Where there were no data for the subregion, patterns for the most similar subregion were applied (EMR-B based on EMR-D, EUR-C based on EUR-B and WPR-A based on AMR-A). Because of limited data on occupational distribution by sex within an economic sector, the same distribution (i.e. proportional division) was applied within a subregion to ages 15 and above, and to males and females. The A subregions had higher proportions of EAP in the professional, managerial and administrative categories, while the B, C, D and E subregions had proportionally more workers in the production categories.

Proportion of workers in an economic sector or occupational category with exposure

Worldwide data on worker exposure are limited. Therefore, several assumptions were made, validated where possible, to establish the proportion of workers exposed to a specific risk factor within an economic sector (PEW). More detail is presented in the sections on specific risk factors.

Exposure partitioning factor (EPF)

In order to partition into high and low exposure groups those workers exposed to carcinogens, we chose the United States Occupational Health and Safety Administration (OSHA) Permissible Exposure Levels (PELs). For most carcinogens we were then able to estimate the risks for the low and high exposure groups from the literature.

The OSHA PELs state a level of the agent that can never be exceeded in the workplace (usually based on eight-hour time-weighted average exposures), and these have had the force of law in the United States as maximum limits of exposure since the creation of OSHA in 1971. Similar occupational exposure limits (OELs) have been promulgated as law by many countries, particularly in the A subregions, and as recommendations by professional expert groups. It is generally considered that a long-term mean exposure in a "minimally controlled" work environment will be in the range 0.3–0.5 times the PEL (Hewett 1996). For example, the American Industrial Hygiene Association suggests that a typical long-term average exposure may be one third of an eight-hour PEL (Roach 1992).

A different approach was used for asthmagens and agents leading to COPD. The actual disease-causing exposures themselves, within these occupations, are either generic (e.g. dust) or too numerous to be useful (e.g. there are over 200 known asthmagens). In both instances there were no international data on the number of workers exposed, which dictated the approach of using occupations or economic subsectors. For asthma, different relative risks were available for eight large occupational groups, while for COPD we partitioned the overall relative risk for the exposed population into high and low relative risks, and assigned these to different economic subsectors according to Korn et al. (1987).

Occupational turnover (OT)

Cancers and lung diseases have long latency periods and once the disease process has begun the worker continues to be at risk, even after exposure ceases. This means that persons who were exposed in the past must be considered as ever-exposed, even if they are currently working in non-exposed jobs or have retired. Furthermore, OT increases the number of persons ever exposed to an occupational risk. This approach was consistent with cohorts represented in the epidemiological studies from which relative risks were taken. The OT factor was not utilized in estimating the numbers of workers exposed to noise, ergonomic stressors or risk factors leading to occupational injuries, as these risk factors do not have latent effects. No turnover was estimated for asthma and COPD owing to a lack of sufficient information on the applicability to studies in which relative risk was measured. Table 21.6 presents data from the literature on annual OT rates in various countries and industries throughout the world, organized by subregion. These reports did not indicate if employees were new to the job or to the industry, although several studies were at the company level, indicating that the worker was new to the company. Therefore, to account for previously unexposed workers entering jobs with carcinogen or dust exposures, an annual turnover rate (ATR) of 10% was selected for all subregions.

An adjustment factor (noted as OT) to account for annual turnover in jobs with exposure to occupational carcinogens was determined as follows:

Computation of adjustment factor to correct for occupational turnover (OT)

$$\begin{aligned}\text{Adjustment factor, } OT &= P_t/P_0 \\ &= [\text{original workers} + \text{new workers} - \text{deaths}]/\text{original workers} \\ &= \{P_0 + [P_0 \times ATR \times t] - [(\text{mortality rate})(P_0 + (P_0 \times ATR \times t)]\}/P_0\end{aligned} \quad (3)$$

where

P_t = the proportion who have ever been occupationally exposed, during a period of 40 years, still living

Table 21.6 Turnover rates in various industries and countries

Country or area	*Basis of measurement*	*Annual turnover rate (ATR)*	*Comments*	*Source*
A subregions				
Italy	Metal—mechanical engineering industry	13.4%	Industry level, based on 2729 observations	Lucifora (1998)
Italy		26%	Total worker turnover rate, including accession and separation	Lucifora (1998)
Spain				
Basque	Industrial production cooperative (manufacturing)	3%	65 firms—cited as low rate	Johnson and Whyte (1977)
United States	Restaurant industry	500%	8 southern restaurants	Butler and Twaddle (1979)
United States	Garment manufacturing	140%	153 female workers at a single plant in the south-west	Koch and Rhodes (1981)
United States	One interstate trucking firm	40%	Expected rate for 1997—truck drivers	EIU (1997a)
United States				
California	Silicon Valley, one financial firm	25%	Software services group	EIU (1997f)
United States				
New Mexico	State-wide survey by New Mexico Department of Labor, January–March 2001	25% per quarter, ranging from 29% in agriculture to 15% in public administration	Agriculture rates show greatest seasonal variation	Moffett (2002)
B subregions				
Brazil	Brazilian labour market	47%	Cited as higher than most markets for which data are available	EIU (1997b)

continued

Table 21.6 Turnover rates in various industries and countries (*continued*)

Country or area	*Basis of measurement*	*Annual turnover rate (ATR)*	*Comments*	*Source*
China	Foreign Investment Enterprise, key managers and technicians	Support staff median rate, 7%, up to >50%; middle management median rate 4%, up to >35%		EIU (1996a)
China	One manufacturer of spun polyester	40% (1992–1993) 21% (1994) 17% (1995) 7% (expected for 1996)	Turnover reductions due to establishment of housing programme	EIU (1996b)
China	Foreign Investment Enterprise	11.8% (1999) 13.4% (2001)	Staff turnover varies widely by subregion, industry and type of enterprise	EIU (2001a, 2002)
China Hong Kong Special Administrative Region	Expatriate teachers, 2000	25%		EIU (2001c)
Eastern Caribbean	Informatics firms	2%	Cited as low rate	EIU (1997d)
Eastern Europe	Security personnel	160%		EIU (1998)
Lao People's Democratic Republic	Vientiane University College	Half of staff have 2-year tenure, average elsewhere is 3 months	Improved owing to staff training	EIU (1997c)
Republic of Korea	National level	3%	Cited as low rate	EIU (1997e)
D subregions				
India	Managers	Minimum 10% estimated	Mostly foreign-invested ventures	EIU (1995)
India	One computer manufacturer	9% (1994)	200 employees	EIU (1995)
E subregions				
Uganda	Uganda Railway Corporation (mid-1990s)	15%	Annual employee turnover rate	EIU (2001b)

P_0 = the proportion who are occupationally exposed at time t=0

ATR = turnover/year, taken as 0.10

t = time, taken as 40 years, a typical working lifetime

mortality = 20% of total cohort, based on published death rates of about 5 deaths per thousand over a period of 40 years (Minino and Smith 2001).

Equation 3 results in an adjustment factor of OT = 4 to correct for occupational turnover over a 40-year period with a median exposure duration of 10 years.

In addition to knowing the numbers of workers exposed to agents with latent health effects, in some cases it was also useful to know the duration of exposure to agents with latent effects for outcomes for which the risks were based on cumulative exposure. Cohort modelling was conducted to determine the typical duration of exposure (K. Steenland, personal communication, 2002). This modelling assumed that people worked for a maximum of 40 years, that 10% of the workers were replaced each year, and that 20% died over the 40-year period. Exposure durations were randomly selected from a log-normal distribution. Persons were also randomly assigned a starting age at entry between 20 and 45 years, and were assumed to retire at age 65 years if they had not already left the cohort by that age. A steady-state working population was produced by using a log-normal distribution for exposure with a geometric mean of 9 years. Using this, the mean length of exposure (in years) at the end of 40 years could be estimated (by age) for all persons ever exposed in the cohort. The average estimated length of exposure, as shown in Table 21.7, was 9.8 years, which is consistent with data on a wide range of cohorts presented in the published literature (Steenland et al. 1991a, 1991b, 2001b).

1.5 Risk factor–disease relationship

Risk measures (relative risks or mortality rates) for the health outcomes resulting from exposure to the risk factors considered in this study were determined primarily from peer-reviewed, published studies. Adjustments were made, as appropriate, to account for differences in levels of exposure, exposure duration and/or age, sex and subregion.

- For carcinogens leading to cancer of the lung, trachea or bronchus, and for leukaemogens, composite values were taken from the literature and adapted to exposure patterns in the various subregions.
- For asthma, the relative risks for different occupations were taken from Karjalainen et al. (2002), with the exception of work in agriculture, for which the relative risk was taken from Kogevinas et al. (1999).

Table 21.7 Exposure duration after 40 years in model cohort

Age group (years)	*Number*	*Total exposure (years)*	*Average exposure (years)*
15–29	12	50	4.2
30–44	86	575	6.7
45–59	117	1182	10.1
60–69	105	1195	11.4
70–79	53	618	11.7
≥80	19	234	12.3
All ages	392	3854	9.8

- For COPD, the relative risks for different economic subsectors were taken from Korn et al. (1987).
- For noise, relative risks of noise-induced hearing loss were calculated from data on hearing loss in workers with different levels of noise exposure in the United States (NIOSH 1998).
- The relative risks of low back pain, given employment in different occupational categories, were taken from Leigh and Sheetz (1989).
- Owing to heterogeneity of factors leading to occupational injuries, relative risks could not be extrapolated from one setting to another. As a result, the mortality rates for workers exposed to risk factors leading to injuries were estimated for different subregions from various sources, including Laborsta (ILO 2001).

2. Occupational carcinogens

The International Agency for Research on Cancer (IARC 2002) has classified 150 chemical or biological agents or exposure situations as known or probable human carcinogens. IARC has classified 87 agents, mixtures or exposure circumstances as Group 1 (carcinogenic to humans), including various chemical compounds, pharmaceuticals and bacterial and viral infections. Many are encountered in occupational settings, e.g. asbestos and cadmium. An additional 63 agents, mixtures or exposure circumstances have been classified as Group 2A (probably carcinogenic to humans). Those with occupational significance include diesel fumes and benzidine-based dyes (IARC 2001). Although IARC classifies agents according to their overall carcinogenicity, specific sites are also considered.

Work-related malignant conditions can arise from a large variety of occupational exposures. However, the main groups of conditions are relatively few—lung cancer and leukaemia. The exposures selected for assessment in this study were based on how common they may be, the

risk arising from exposure, the strength of evidence and the availability of data. Table 21.8 shows the definition of each of the chemical and physical agents, along with the related cancer.

The analysis included relevant Group 1 and 2A carcinogens, with the following exceptions.

- Tetrachloroethylene and trichloroethylene, both classified in Group 2A, were not included as carcinogens because the evidence for cancer is weak.
- The aromatic amines and dyes, including 2-naphthylamine, benzidine-based dyes, benzidine and 4,4'-methylenebis(2-chloroaniline) (also known as MOCA) were excluded owing to lack of data for developing countries.
- Occupational carcinogens with extremely limited exposures (e.g. bis-chloromethyl ether, also known as BCME) were not included.
- Compounds for which exposure estimates were not available from the CAREX database (e.g. soot, xenylamine, 4-nitrobiphenyl and polycyclic aromatic hydrocarbons) were not included.
- Although radon is an IARC Group I carcinogen with large estimated exposures, it was excluded from consideration owing (i) to worldwide differences in naturally occurring radon emissions, (ii) to wide variations in climate and construction methods, which substantially affect the concentration of radon retained in buildings, and (iii) to difficulties in separating occupational and nonoccupational radon exposures.

Other conditions have insufficient relevant exposure data, insufficient risk data or insufficient number of cases worldwide to allow them to be usefully included. These conditions include:

- bladder cancer (aromatic amines, benzidine dyes, MOCA);
- liver (vinyl chloride);
- nasal cavity and middle ear (hardwood dust, chromium VI compounds, nickel compounds);
- bone and articular cartilage (ionizing radiation);

Table 21.8 Occupational carcinogens and health outcomes

Occupational carcinogen	*Outcome*
Arsenic, asbestos, beryllium, cadmium, chromium, diesel exhaust, nickel, silica	Cancer of the trachea, bronchus or lung
Benzene, ethylene oxide, ionizing radiation	Leukaemia

- skin (arsenic, by-products of distillation, ionizing radiation); and
- lung cancer due to passive smoking in the workplace.

2.1 Exposure variable and theoretical-minimum-risk exposure

Exposure was divided into three categories: background, low and high. The occupational risk factors for cancer involve workplace exposure, at concentrations higher than background level, to various chemical and physical agents that are known to cause malignant neoplasms. Thus, the theoretical minimum risk corresponds to "no occupational exposure to physical, chemical or biological agents or other factors above background levels".

2.2 Estimating risk factor levels

The general exposure assessment methodology was described earlier. This assessment was based on the distribution of the EAP by economic subsector, because the primary exposure data sources used in this analysis organized carcinogen exposure data by economic subsector (Equation 1). The regional distributions of workers into economic subsectors were adjusted by data on the carcinogens to which people in the various economic subsectors were exposed. As described earlier, an adjustment factor of 4 was used to account for turnover in jobs with exposure to occupational carcinogens.

The primary data source on work-related exposure to carcinogens for each economic subsector (PEW(es(r,g)i) in Equation 1) is the CAREX database (FIOH 1999), which presents data on the number 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. Table 21.9 lists the CAREX data for the carcinogens in our study. 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.

It was assumed that the proportion of workers exposed to a particular carcinogen in a specific economic subsector was constant throughout the world. To check the validity of this assumption, the literature was searched for estimates of the number of workers exposed to silica. Silica was chosen as an indicator because there are more data on silica available for developing countries than on other carcinogens. This search yielded a range of study types, from rough estimates (Zou Changqi et al. 1997) to studies in which air concentrations were measured in workplaces (Yin et al. 1987). Estimates of the number of workers exposed to silica in China, Thailand and Viet Nam, and to benzene in China, were compared to the number of persons employed in that country, either in a specific economic sector or overall. The results obtained were compared with CAREX data. With a few exceptions, the estimated fraction

Table 21.9 Mean proportions of workers exposed to selected carcinogens, by economic sector and subsector, in the European Union

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
Cadmium	0.00000	0.00000	0.00487	0.00287	0.00291	0.00002	0.00065	0.00000	0.00047
Nickel	0.00000	0.02025	0.01680	0.00352	0.00047	0.00007	0.00003	0.00000	0.00043
Arsenic	0.00054	0.00072	0.00400	0.00148	0.00134	0.00006	0.00000	0.00002	0.00011
Chromium	0.00000	0.00346	0.02079	0.00409	0.00237	0.00017	0.00370	0.00000	0.00225
Diesel fumes	0.00646	0.21970	0.01110	0.03358	0.05816	0.00485	0.13438	0.00000	0.00914
Beryllium	0.00000	0.00055	0.00207	0.00070	0.00004	0.00002	0.00011	0.00000	0.00003
Asbestos	0.01248	0.10248	0.00590	0.01702	0.05203	0.00292	0.00684	0.00016	0.00284
Benzene	0.00100	0.00200	0.00300	0.00100	0.00100	0.01000	0.00500	0.00000	0.02000
Ionizing radiation	0.00000	0.01100	0.00000	0.03400	0.00000	0.00000	0.00400	0.00000	0.00000
Ethylene oxide	0.00012	0.00137	0.00060	0.00006	0.00027	0.00000	0.00002	0.00000	0.00057

Source: Calculated from CAREX (FIOH 1999).

of workers exposed to silica was equal to or higher in these countries than indicated by CAREX (Juengprasert 1997; T. Nguyen, personal communication, 2001; NIEHS 1999; Phan Hong Son et al. 1999; Yin et al. 1987; Zou Changqi et al. 1997). For example, the proportion of workers exposed to silica in manufacturing in Viet Nam is 3.7%, compared to the CAREX estimate of 2.3%.

It was assumed that, within a given economic subsector, both male and female workers and younger and older workers had the same probability of exposure. For example, if 2.3% of people working in manufacturing were exposed to silica, it was assumed that 2.3% of males and 2.3% of females working in manufacturing were exposed to silica, young and old alike. There were, however, fewer females working in manufacturing, so that at the population level the proportion of females with exposure to silica was lower than that of males.

There are few data on the distribution of exposure monitoring values, which are needed to accurately estimate the proportion of workers exposed to above or below a specific value (EPF(r) in Equation 1). Therefore, the demarcation between low and high exposure was established as the PELs enforced by OSHA. Some reasons for selecting the PELs as partitioning values include the following.

- Exposure data for the United States are often reported based on "compliance with" or "exceeding" the PELs.
- The risks corresponding to low or high exposure have been linked to the PELs.
- As cancers have long latency periods, the exposures of concern have occurred several decades in the past. The OSHA PELs for many carcinogens have not changed since their adoption in 1971, allowing a stable benchmark for comparison (Table 21.10).

The peer-reviewed literature was searched for studies that included proportions of workers exposed above and below particular levels. There are many reports of exposures to contaminants in the literature, and even on the distribution of exposures at low and high levels in developed countries. However, there are few data on distribution of exposure values for developing countries. A summary of the major sources used to decide how to partition exposure values for carcinogens for the B, C, D and E subregions is presented in Tables 21.11 and 21.12 for benzene and metals, respectively.

The following data were used to partition exposure for A subregions:

- Finnish data (Partanen et al. 1995), indicating 11–94% exposed above 0.2 mg/m^3 respirable silica in a range of industries;
- NIOSH (1999) estimates of proportions of workers exposed above the PELs of 4% (asbestos) and 13.6% (silica); and

Table 21.10 OSHA permissible exposure levels (PELs) for carcinogens

Chemical/ physical agent	*PEL*	*Source*	*Comment*
Arsenic	Inorganic: 10 μg/m^3 Organic: 0.5 mg/m^3	OSHA, 29 CFR 1910.1018 OSHA, 29 CFR 1910.1000, Table Z-1	Effective 1978 Effective 1971
Asbestos	Varied[a]		
Benzene	10 ppm	OSHA, 29 CFR 1910.1000, Table Z-2	Effective 1971
	1 ppm	OSHA, 29 CFR 1910.1028	Effective 1987
Beryllium	2 μg/m^3	OSHA, 29 CFR 1910.1000, Table Z-2	Effective 1971
Cadmium	Fume: 0.1 mg/m^3	OSHA, 29 CFR 1910.1000, Table Z-2	Effective 1971
	Dust: 0.2 mg/m^3	OSHA, 29 CFR 1910.1000, Table Z-2	Effective 1971
	5 μg/m^3	29 CFR 1910.1027	Effective 1992
Chromium	Chromic acid and chromates: 0.1 mg/m^3	OSHA, 29 CFR 1910.1000, Table Z-2 (ceiling)	Effective 1971
	Chromium metal: 1 mg/m^3	OSHA, 29 CFR 1910.1000, Table Z-1	Effective 1971
Diesel exhaust	NA		
Ethylene oxide	1 ppm	OSHA, 29 CFR 1910.1047	Effective 1984
Ionizing radiation	Rems/calendar quarter: whole body, 1.25; hands, forearms, feet, ankles, 18.75; skin, 7.5	OSHA, 29 CFR 1910.1096, Table G-18	Effective 1974
Nickel	Metal, insoluble and soluble compounds: 1 mg/m^3	OSHA, 29 CFR 1910.1000, Table Z-1	Effective 1971
Silica	Respirable quartz: (10 mg/m^3)/ (per cent SiO_2 + 2)	OSHA, 29 CFR 1910.1000, Table Z-3	Effective 1971. For 100% silica dust, this is equivalent to 0.1 mg/m^3. Halve this value for cristobalite and tridymite

NA Not applicable.

[a] As shown in this table, most of the PELs have not changed since they were put in place. However, there were considerable changes in the United States PEL for asbestos during the years of interest to the current analysis, with a level before 1972 of 12 fibres/ml before the first OSHA-issued PEL in 1972 decreasing, through several steps, to 0.1 fibres/ml in 1994 (Martonik et al. 2001; Nelson 1997).

Source: USDOL OSHA (2002a).

Table 21.11 Occupational exposure to benzene in developing countries

Country	*Industry*	*Concentration*	*Year (or year reported)*
Egypt	Rubber coating	0–74 mg/m^3	(1986)
Turkey	Shoemaking	48–96 mg/m^3 672 mg/m^3 (maximum level)	1970
India	Petrol pump	4.5 mg/m^3 (mean)	1991
China	Various: paint, chemical, varnish works, shoemaking	0.06–850 mg/m^3	(1987)
Brazil	Steel workers Petrochemical	960–3200 mg/m^3 per day 140 mg/m^3, maximum of personal samples	(1993) (1993)

Source: Pearce et al. (1994).

Table 21.12 Occupational exposures to metals in developing countries

Country	*Industry*	*Concentration*	*Year (or year reported)*
China	Tin mine	Arsenic: 0.42 mg/m^3, mean Arsenic: 0.01 mg/m^3	1952 1980s
China	Cadmium refining	Cadmium: 0.04–0.074 mg/m^3	1970s
Singapore	Storage battery factory	Cadmium: 0.13–58.3 mg/m^3, geometric means of three sets of samples	1980
China	Chromate production	0.02–21.3 mg/m^3 0.55 mg/m^3, mean	1960s–1980 (1989)

Source: Pearce et al. (1994).

- NIOSH (2000b) data on miners, indicating silica exposures above the PEL for 8% of coal mine samples, 16% of metal mine samples, 9% of stone mine samples and 8% of sand and gravel facility samples.

For the B, C, D and E subregions, important evidence includes:

- Chinese data (Dosemeci et al. 1995), indicating roughly three quarters of samples above 0.1 mg/m^3 respirable silica;
- a study of a South African brickworks (Myers et al. 1989), in which 45% of presented sample values were above 0.1 mg/m^3 and roughly two thirds and four fifths of samples in medium and dusty areas, respectively, were above 0.1 mg/m^3 respirable silica;
- a study of a South African pottery (Rees et al. 1992), where roughly three quarters of samples that included silica analysis were above the Threshold Limit Value (TLV); and

Table 21.13 Exposure partition factors for carcinogens for the A and for the B, C, D and E subregions

Subregion	*Proportion of exposed workers with low exposures (at or below the PEL)*	*Proportion of exposed workers with high exposures (above the PEL)*
A	0.90	0.10
B, C, D and E	0.50	0.50

- the Chinese benzene study (Yin et al. 1987), in which 35% of over 50 000 workplaces had concentrations at or above 40 mg/m^3, in comparison to the current OSHA PEL of 3.2 mg/m^3 for benzene, and in which the benzene concentration in 86% of 141 shoe factories was above 25 mg/m^3.

Based on these data, partition factors for carcinogen exposures were determined for the A and for the B, C, D and E subregions, as shown in Table 21.13.

Lung carcinogens

The proportions of the population exposed to the occupational lung carcinogens included in the study (Table 21.8) are shown in Tables 21.14 and 21.16 by subregion, age, sex and level of exposure.

Leukaemogens

The proportions of the population exposed to occupational leukaemogens (Table 21.8) are presented in Table 21.15 by subregion, age, sex and level of exposure.

2.3 Risk factor–disease relationships

Relative risk estimates were used for lung carcinogens and leukaemogens. Table 21.17 summarizes the chemical or physical agent, the specific cancer and the key data sources that provided evidence of the link between the two. These review studies assessed risk measures for the main sites of occupational cancer, including the lung (which, for the purposes of this study, includes the trachea, bronchus and lung), the haematopoietic system (represented in this study by leukaemia) and malignant mesothelioma.

Relative risks for lung cancer and leukaemia were taken from studies of cohorts of workers with variable exposure durations and intensities, variable periods from the last exposure and variable lengths of follow-up. They therefore compare exposed with unexposed groups. In preparing relative risk estimates for exposure outcomes of interest, several assumptions were made:

Table 21.14 Proportions of the population exposed to lung carcinogens by subregion, age, sex and level of exposure

			Age group (years)					
Subregion	*Sex*	*Exposure level*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	Background	0.837	0.837	0.837	0.837	0.837	0.837
		Low	0.082	0.082	0.082	0.082	0.082	0.082
		High	0.082	0.082	0.082	0.082	0.082	0.082
	Female	Background	0.934	0.934	0.934	0.934	0.934	0.934
		Low	0.033	0.033	0.033	0.033	0.033	0.033
		High	0.033	0.033	0.033	0.033	0.033	0.033
AFR-E	Male	Background	0.839	0.839	0.839	0.839	0.839	0.839
		Low	0.080	0.080	0.080	0.080	0.080	0.080
		High	0.080	0.080	0.080	0.080	0.080	0.080
	Female	Background	0.929	0.929	0.929	0.929	0.929	0.929
		Low	0.035	0.035	0.035	0.035	0.035	0.035
		High	0.035	0.035	0.035	0.035	0.035	0.035
AMR-A	Male	Background	0.802	0.802	0.802	0.802	0.802	0.802
		Low	0.178	0.178	0.178	0.178	0.178	0.178
		High	0.020	0.020	0.020	0.020	0.020	0.020
	Female	Background	0.936	0.936	0.936	0.936	0.936	0.936
		Low	0.058	0.058	0.058	0.058	0.058	0.058
		High	0.006	0.006	0.006	0.006	0.006	0.006
AMR-B	Male	Background	0.793	0.793	0.793	0.793	0.793	0.793
		Low	0.103	0.103	0.103	0.103	0.103	0.103
		High	0.103	0.103	0.103	0.103	0.103	0.103
	Female	Background	0.951	0.951	0.951	0.951	0.951	0.951
		Low	0.024	0.024	0.024	0.024	0.024	0.024
		High	0.024	0.024	0.024	0.024	0.024	0.024
AMR-D	Male	Background	0.761	0.761	0.761	0.761	0.761	0.761
		Low	0.119	0.119	0.119	0.119	0.119	0.119
		High	0.119	0.119	0.119	0.119	0.119	0.119
	Female	Background	0.961	0.961	0.961	0.961	0.961	0.961
		Low	0.019	0.019	0.019	0.019	0.019	0.019
		High	0.019	0.019	0.019	0.019	0.019	0.019
EMR-B	Male	Background	0.760	0.760	0.760	0.760	0.760	0.760
		Low	0.120	0.120	0.120	0.120	0.120	0.120
		High	0.120	0.120	0.120	0.120	0.120	0.120
	Female	Background	0.963	0.963	0.963	0.963	0.963	0.963
		Low	0.019	0.019	0.019	0.019	0.019	0.019
		High	0.019	0.019	0.019	0.019	0.019	0.019
EMR-D	Male	Background	0.840	0.840	0.840	0.840	0.840	0.840
		Low	0.080	0.080	0.080	0.080	0.080	0.080
		High	0.080	0.080	0.080	0.080	0.080	0.080
	Female	Background	0.955	0.955	0.955	0.955	0.955	0.955
		Low	0.023	0.023	0.023	0.023	0.023	0.023
		High	0.023	0.023	0.023	0.023	0.023	0.023

Table 21.14 Proportions of the population exposed to lung carcinogens by subregion, age, sex and level of exposure (*continued*)

			Age group (years)					
Subregion	*Sex*	*Exposure level*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
EUR-A	Male	Background	0.802	0.802	0.802	0.802	0.802	0.802
		Low	0.179	0.179	0.179	0.179	0.179	0.179
		High	0.020	0.020	0.020	0.020	0.020	0.020
	Female	Background	0.937	0.937	0.937	0.937	0.937	0.937
		Low	0.057	0.057	0.057	0.057	0.057	0.057
		High	0.006	0.006	0.006	0.006	0.006	0.006
EUR-B	Male	Background	0.779	0.779	0.779	0.779	0.779	0.779
		Low	0.111	0.111	0.111	0.111	0.111	0.111
		High	0.111	0.111	0.111	0.111	0.111	0.111
	Female	Background	0.920	0.920	0.920	0.920	0.920	0.920
		Low	0.040	0.040	0.040	0.040	0.040	0.040
		High	0.040	0.040	0.040	0.040	0.040	0.040
EUR-C	Male	Background	0.654	0.654	0.654	0.654	0.654	0.654
		Low	0.173	0.173	0.173	0.173	0.173	0.173
		High	0.173	0.173	0.173	0.173	0.173	0.173
	Female	Background	0.801	0.801	0.801	0.801	0.801	0.801
		Low	0.099	0.099	0.099	0.099	0.099	0.099
		High	0.099	0.099	0.099	0.099	0.099	0.099
SEAR-B	Male	Background	0.798	0.798	0.798	0.798	0.798	0.798
		Low	0.101	0.101	0.101	0.101	0.101	0.101
		High	0.101	0.101	0.101	0.101	0.101	0.101
	Female	Background	0.922	0.922	0.922	0.922	0.922	0.922
		Low	0.039	0.039	0.039	0.039	0.039	0.039
		High	0.039	0.039	0.039	0.039	0.039	0.039
SEAR-D	Male	Background	0.805	0.805	0.805	0.805	0.805	0.805
		Low	0.098	0.098	0.098	0.098	0.098	0.098
		High	0.098	0.098	0.098	0.098	0.098	0.098
	Female	Background	0.934	0.934	0.934	0.934	0.934	0.934
		Low	0.033	0.033	0.033	0.033	0.033	0.033
		High	0.033	0.033	0.033	0.033	0.033	0.033
WPR-A	Male	Background	0.745	0.745	0.745	0.745	0.745	0.745
		Low	0.230	0.230	0.230	0.230	0.230	0.230
		High	0.026	0.026	0.026	0.026	0.026	0.026
	Female	Background	0.914	0.914	0.914	0.914	0.914	0.914
		Low	0.078	0.078	0.078	0.078	0.078	0.078
		High	0.009	0.009	0.009	0.009	0.009	0.009
WPR-B	Male	Background	0.769	0.769	0.769	0.769	0.769	0.769
		Low	0.115	0.115	0.115	0.115	0.115	0.115
		High	0.115	0.115	0.115	0.115	0.115	0.115
	Female	Background	0.875	0.875	0.875	0.875	0.875	0.875
		Low	0.063	0.063	0.063	0.063	0.063	0.063
		High	0.063	0.063	0.063	0.063	0.063	0.063

Table 21.15 Proportions of the population exposed to leukaemogens by subregion, age, sex and level of exposure

			Age group (years)					
Subregion	*Sex*	*Exposure level*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	Background	0.980	0.980	0.980	0.980	0.980	0.980
		Low	0.010	0.010	0.010	0.010	0.010	0.010
		High	0.010	0.010	0.010	0.010	0.010	0.010
	Female	Background	0.989	0.989	0.989	0.989	0.989	0.989
		Low	0.005	0.005	0.005	0.005	0.005	0.005
		High	0.005	0.005	0.005	0.005	0.005	0.005
AFR-E	Male	Background	0.979	0.979	0.979	0.979	0.979	0.979
		Low	0.010	0.010	0.010	0.010	0.010	0.010
		High	0.010	0.010	0.010	0.010	0.010	0.010
	Female	Background	0.984	0.984	0.984	0.984	0.984	0.984
		Low	0.008	0.008	0.008	0.008	0.008	0.008
		High	0.008	0.008	0.008	0.008	0.008	0.008
AMR-A	Male	Background	0.973	0.973	0.973	0.973	0.973	0.973
		Low	0.025	0.025	0.025	0.025	0.025	0.025
		High	0.003	0.003	0.003	0.003	0.003	0.003
	Female	Background	0.970	0.970	0.970	0.970	0.970	0.970
		Low	0.027	0.027	0.027	0.027	0.027	0.027
		High	0.003	0.003	0.003	0.003	0.003	0.003
AMR-B	Male	Background	0.980	0.980	0.980	0.980	0.980	0.980
		Low	0.010	0.010	0.010	0.010	0.010	0.010
		High	0.010	0.010	0.010	0.010	0.010	0.010
	Female	Background	0.977	0.977	0.977	0.977	0.977	0.977
		Low	0.011	0.011	0.011	0.011	0.011	0.011
		High	0.011	0.011	0.011	0.011	0.011	0.011
AMR-D	Male	Background	0.967	0.967	0.967	0.967	0.967	0.967
		Low	0.016	0.016	0.016	0.016	0.016	0.016
		High	0.016	0.016	0.016	0.016	0.016	0.016
	Female	Background	0.979	0.979	0.979	0.979	0.979	0.979
		Low	0.010	0.010	0.010	0.010	0.010	0.010
		High	0.010	0.010	0.010	0.010	0.010	0.010
EMR-B	Male	Background	0.969	0.969	0.969	0.969	0.969	0.969
		Low	0.015	0.015	0.015	0.015	0.015	0.015
		High	0.015	0.015	0.015	0.015	0.015	0.015
	Female	Background	0.984	0.984	0.984	0.984	0.984	0.984
		Low	0.008	0.008	0.008	0.008	0.008	0.008
		High	0.008	0.008	0.008	0.008	0.008	0.008
EMR-D	Male	Background	0.984	0.984	0.984	0.984	0.984	0.984
		Low	0.008	0.008	0.008	0.008	0.008	0.008
		High	0.008	0.008	0.008	0.008	0.008	0.008
	Female	Background	0.995	0.995	0.995	0.995	0.995	0.995
		Low	0.003	0.003	0.003	0.003	0.003	0.003
		High	0.003	0.003	0.003	0.003	0.003	0.003

Table 21.15 Proportions of the population exposed to leukaemogens by subregion, age, sex and level of exposure (*continued*)

			Age group (years)					
Subregion	*Sex*	*Exposure level*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
EUR-A	Male	Background	0.968	0.968	0.968	0.968	0.968	0.968
		Low	0.029	0.029	0.029	0.029	0.029	0.029
		High	0.003	0.003	0.003	0.003	0.003	0.003
	Female	Background	0.973	0.973	0.973	0.973	0.973	0.973
		Low	0.024	0.024	0.024	0.024	0.024	0.024
		High	0.003	0.003	0.003	0.003	0.003	0.003
EUR-B	Male	Background	0.978	0.978	0.978	0.978	0.978	0.978
		Low	0.011	0.011	0.011	0.011	0.011	0.011
		High	0.011	0.011	0.011	0.011	0.011	0.011
	Female	Background	0.983	0.983	0.983	0.983	0.983	0.983
		Low	0.009	0.009	0.009	0.009	0.009	0.009
		High	0.009	0.009	0.009	0.009	0.009	0.009
EUR-C	Male	Background	0.982	0.982	0.982	0.982	0.982	0.982
		Low	0.009	0.009	0.009	0.009	0.009	0.009
		High	0.009	0.009	0.009	0.009	0.009	0.009
	Female	Background	0.981	0.981	0.981	0.981	0.981	0.981
		Low	0.009	0.009	0.009	0.009	0.009	0.009
		High	0.009	0.009	0.009	0.009	0.009	0.009
SEAR-B	Male	Background	0.980	0.980	0.980	0.980	0.980	0.980
		Low	0.010	0.010	0.010	0.010	0.010	0.010
		High	0.010	0.010	0.010	0.010	0.010	0.010
	Female	Background	0.985	0.985	0.985	0.985	0.985	0.985
		Low	0.008	0.008	0.008	0.008	0.008	0.008
		High	0.008	0.008	0.008	0.008	0.008	0.008
SEAR-D	Male	Background	0.979	0.979	0.979	0.979	0.979	0.979
		Low	0.011	0.011	0.011	0.011	0.011	0.011
		High	0.011	0.011	0.011	0.011	0.011	0.011
	Female	Background	0.995	0.995	0.995	0.995	0.995	0.995
		Low	0.003	0.003	0.003	0.003	0.003	0.003
		High	0.003	0.003	0.003	0.003	0.003	0.003
WPR-A	Male	Background	0.975	0.975	0.975	0.975	0.975	0.975
		Low	0.023	0.023	0.023	0.023	0.023	0.023
		High	0.003	0.003	0.003	0.003	0.003	0.003
	Female	Background	0.979	0.979	0.979	0.979	0.979	0.979
		Low	0.019	0.019	0.019	0.019	0.019	0.019
		High	0.002	0.002	0.002	0.002	0.002	0.002
WPR-B	Male	Background	0.978	0.978	0.978	0.978	0.978	0.978
		Low	0.011	0.011	0.011	0.011	0.011	0.011
		High	0.011	0.011	0.011	0.011	0.011	0.011
	Female	Background	0.980	0.980	0.980	0.980	0.980	0.980
		Low	0.010	0.010	0.010	0.010	0.010	0.010
		High	0.010	0.010	0.010	0.010	0.010	0.010

Table 21.16 Proportions of the population exposed to asbestos by subregion, age, sex and level of exposure

			Age group (years)					
Subregion	*Sex*	*Exposure level*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	Background	0.961	0.961	0.961	0.961	0.961	0.961
		Low	0.019	0.019	0.019	0.019	0.019	0.019
		High	0.019	0.019	0.019	0.019	0.019	0.019
	Female	Background	0.978	0.978	0.978	0.978	0.978	0.978
		Low	0.011	0.011	0.011	0.011	0.011	0.011
		High	0.011	0.011	0.011	0.011	0.011	0.011
AFR-E	Male	Background	0.961	0.961	0.961	0.961	0.961	0.961
		Low	0.020	0.020	0.020	0.020	0.020	0.020
		High	0.020	0.020	0.020	0.020	0.020	0.020
	Female	Background	0.975	0.975	0.975	0.975	0.975	0.975
		Low	0.012	0.012	0.012	0.012	0.012	0.012
		High	0.012	0.012	0.012	0.012	0.012	0.012
AMR-A	Male	Background	0.973	0.973	0.973	0.973	0.973	0.973
		Low	0.025	0.025	0.025	0.025	0.025	0.025
		High	0.003	0.003	0.003	0.003	0.003	0.003
	Female	Background	0.991	0.991	0.991	0.991	0.991	0.991
		Low	0.008	0.008	0.008	0.008	0.008	0.008
		High	0.001	0.001	0.001	0.001	0.001	0.001
AMR-B	Male	Background	0.966	0.966	0.966	0.966	0.966	0.966
		Low	0.017	0.017	0.017	0.017	0.017	0.017
		High	0.017	0.017	0.017	0.017	0.017	0.017
	Female	Background	0.992	0.992	0.992	0.992	0.992	0.992
		Low	0.004	0.004	0.004	0.004	0.004	0.004
		High	0.004	0.004	0.004	0.004	0.004	0.004
AMR-D	Male	Background	0.965	0.965	0.965	0.965	0.965	0.965
		Low	0.017	0.017	0.017	0.017	0.017	0.017
		High	0.017	0.017	0.017	0.017	0.017	0.017
	Female	Background	0.994	0.994	0.994	0.994	0.994	0.994
		Low	0.003	0.003	0.003	0.003	0.003	0.003
		High	0.003	0.003	0.003	0.003	0.003	0.003
EMR-B	Male	Background	0.963	0.963	0.963	0.963	0.963	0.963
		Low	0.018	0.018	0.018	0.018	0.018	0.018
		High	0.018	0.018	0.018	0.018	0.018	0.018
	Female	Background	0.994	0.994	0.994	0.994	0.994	0.994
		Low	0.003	0.003	0.003	0.003	0.003	0.003
		High	0.003	0.003	0.003	0.003	0.003	0.003
EMR-D	Male	Background	0.962	0.962	0.962	0.962	0.962	0.962
		Low	0.019	0.019	0.019	0.019	0.019	0.019
		High	0.019	0.019	0.019	0.019	0.019	0.019
	Female	Background	0.985	0.985	0.985	0.985	0.985	0.985
		Low	0.008	0.008	0.008	0.008	0.008	0.008
		High	0.008	0.008	0.008	0.008	0.008	0.008

Table 21.16 Proportions of the population exposed to asbestos by subregion, age, sex and level of exposure (*continued*)

			Age group (years)					
Subregion	*Sex*	*Exposure level*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
EUR-A	Male	Background	0.971	0.971	0.971	0.971	0.971	0.971
		Low	0.026	0.026	0.026	0.026	0.026	0.026
		High	0.003	0.003	0.003	0.003	0.003	0.003
	Female	Background	0.991	0.991	0.991	0.991	0.991	0.991
		Low	0.008	0.008	0.008	0.008	0.008	0.008
		High	0.001	0.001	0.001	0.001	0.001	0.001
EUR-B	Male	Background	0.962	0.962	0.962	0.962	0.962	0.962
		Low	0.019	0.019	0.019	0.019	0.019	0.019
		High	0.019	0.019	0.019	0.019	0.019	0.019
	Female	Background	0.982	0.982	0.982	0.982	0.982	0.982
		Low	0.009	0.009	0.009	0.009	0.009	0.009
		High	0.009	0.009	0.009	0.009	0.009	0.009
EUR-C	Male	Background	0.949	0.949	0.949	0.949	0.949	0.949
		Low	0.025	0.025	0.025	0.025	0.025	0.025
		High	0.025	0.025	0.025	0.025	0.025	0.025
	Female	Background	0.975	0.975	0.975	0.975	0.975	0.975
		Low	0.013	0.013	0.013	0.013	0.013	0.013
		High	0.013	0.013	0.013	0.013	0.013	0.013
SEAR-B	Male	Background	0.959	0.959	0.959	0.959	0.959	0.959
		Low	0.020	0.020	0.020	0.020	0.020	0.020
		High	0.020	0.020	0.020	0.020	0.020	0.020
	Female	Background	0.981	0.981	0.981	0.981	0.981	0.981
		Low	0.010	0.010	0.010	0.010	0.010	0.010
		High	0.010	0.010	0.010	0.010	0.010	0.010
SEAR-D	Male	Background	0.959	0.959	0.959	0.959	0.959	0.959
		Low	0.021	0.021	0.021	0.021	0.021	0.021
		High	0.021	0.021	0.021	0.021	0.021	0.021
	Female	Background	0.978	0.978	0.978	0.978	0.978	0.978
		Low	0.011	0.011	0.011	0.011	0.011	0.011
		High	0.011	0.011	0.011	0.011	0.011	0.011
WPR-A	Male	Background	0.967	0.967	0.967	0.967	0.967	0.967
		Low	0.030	0.030	0.030	0.030	0.030	0.030
		High	0.003	0.003	0.003	0.003	0.003	0.003
	Female	Background	0.988	0.988	0.988	0.988	0.988	0.988
		Low	0.011	0.011	0.011	0.011	0.011	0.011
		High	0.001	0.001	0.001	0.001	0.001	0.001
WPR-B	Male	Background	0.955	0.955	0.955	0.955	0.955	0.955
		Low	0.022	0.022	0.022	0.022	0.022	0.022
		High	0.022	0.022	0.022	0.022	0.022	0.022
	Female	Background	0.974	0.974	0.974	0.974	0.974	0.974
		Low	0.013	0.013	0.013	0.013	0.013	0.013
		High	0.013	0.013	0.013	0.013	0.013	0.013

Table 21.17 Examples of sources used to assess the risk factor–disease relationship for selected occupational carcinogens

Selected risk factor	*Health outcome*	*Examples of key sources of evidence of causality*
Lung carcinogens	Cancer of the trachea, bronchus or lung	Nurminen and Karjalainen (2001); Steenland et al. (1996, 2003)
Leukaemogens	Leukaemia	Lynge et al. (1997); BEIR V (1990); IARC (1997)

- that relative risks are the same for men and women;
- that relative risk values are constant with age; and
- that the relative risks apply equally to the risk of developing the malignant condition (incident cases) and to the risk of dying from the condition (fatal cases); where relative risk values were based on disease incidence studies, the incidence ratio was comparable to the corresponding mortality risk ratio.

Steenland et al. (1996) estimated for the United States the relative risk of exposure to nine lung carcinogens (arsenic, asbestos, beryllium, cadmium, chromium, diesel fumes, nickel, silica and radon). They did not consider agents to which relatively few workers were exposed (BCME, coke oven and coal gasification fumes and soot) and they did not consider smoking, beyond the selection where possible of relative risk factors that had been adjusted for smoking. Combined relative risk values (ranging from 1.31 to 3.69) were calculated for all but radon, using inverse variance and a random-effects model and relying on major cohort studies of the specific agents. The authors estimated that 9000–10 000 men and 900–1900 women develop lung cancer annually in the United States owing to past exposure to occupational carcinogens (except radon). This would account for approximately 9% of lung cancer deaths in males and 2% in females, or 0.5% of all deaths annually in the United States.

Steenland et al. (2003) examined the population-attributable risk (PAR) from several studies (including Steenland et al. 1996). They applied the PAR to deaths occurring in 1997 in the United States to determine occupational deaths from lung cancer, among other outcomes. The authors determined a PAR for lung cancer in the range 6.1–17.3% for men and 2% for women. For overall cancer they determined a PAR of 7–19% for men and 11% for women. For leukaemia, a combined PAR for men and women of 0.8–2.8% was calculated.

Nurminen and Karjalainen (2001) estimated the proportion of fatalities related to occupational factors in Finland. The average number of exposed workers in Finland was estimated from census data by sex, age, occupation and industry, and the FINJEM national job-exposure matrix.

Relative risks were obtained from a review of epidemiological studies, focusing on risk estimates that were most valid for the Finnish exposure circumstances. The attributable fraction methodology was used to determine the proportion of deaths in the population attributable to occupational factors. The authors reported that 30% of deaths due to occupational disease in Finland in 1996 were caused by cancer. Occupational lung cancer accounted for 0.9% of all deaths. They attributed 24% of cancer of the bronchus and lung (29% for men and 5.3% for women) to occupational exposure to combined risk factors. The attributable fractions for urinary cancer were 10.3% overall—14.2% for men and 0.7% for women. Combined occupational risk factors resulted in 10.9% (18.5% for males, 2.5% for females) of leukaemia deaths being attributed to occupational exposures, the majority (17.8% and 2.3%, respectively) from electrical occupations, in contrast to 0.7% and 0.2%, respectively, from benzene. An average of 71.3% (90% for males, 25% for females) of malignant mesothelioma was attributed to occupation.

The three review papers described above (Nurminen and Karjalainen 2001; Steenland et al. 1996, 2003) provided summary measures, or information that can be used to determine summary measures, of relative risk for one or more of the main agents and outcomes of interest. The study by Nurminen and Karjalainen (2001) focused on Finland, and preferentially used studies based in Scandinavia or thought to be most relevant to Finland. Most of its relative risk estimates relate to lung cancer, with attributable fractions presented for leukaemia. The paper by Steenland et al. (1996), although focused on the United States, was more inclusive of studies of suitable quality. The other paper by Steenland et al. (2003) provided information on relative mortality risks similar to the first (1996) paper. All papers provided similar summary measures of relative risk for lung cancers, but the Steenland et al. (1996) results were used preferentially because they are generally based on a broader range of studies. However, the Steenland paper provided information only on lung cancer. Table 21.18 gives a summary of the risk measures for each of the carcinogens and the relevant outcomes. The basis for these risk estimations is described in more detail below.

Lung cancer

The evidence for substance-specific relative risk values, which were used to calculate the overall relative risk for the eight lung carcinogens, is briefly discussed below, relying heavily on the review paper by Steenland et al. (1996). The data in the paper provide a summary relative risk of 1.6 for occupational exposure to the set of lung carcinogens considered here.

Smoking is the main important potential confounder of lung cancer, and potentiates the effect of some exposures (notably with asbestos and lung cancer). In this analysis, where possible, studies were used that produced risk estimates for lung cancer after controlling for smoking.

Table 21.18 Summary of risk measures (relative risk and mortality rates) for occupational carcinogens

Health outcome	*Risk measure*	*Estimate and 95% confidence interval (CI)*	*Comments*	*Primary data sources*
Cancer of the trachea, bronchus and lung	Relative risk	Low exposure: 1.22 (1.09–1.35) to 1.32 (1.17–1.48) High exposure: 1.79 (1.59–1.97) to 1.93 (1.71–2.16)	Composite relative risk based on individual relative risk of arsenic, asbestos, beryllium, cadmium, chromium, diesel exhaust, nickel and silica	Steenland et al. (1996)
Leukaemia	Relative risk	Low exposure: 1.67 (1.51–2.00) to 1.93 (1.76–2.17) High exposure: 3.06 (2.64–3.80) to 3.86 (3.48–4.32)	Composite relative risk based on individual relative risk of benzene, ionizing radiation and ethylene oxide	BEIR V (1990); IARC (2000); Lynge et al. (1997); Steenland et al. (2003)

Arsenic

Arsenic is accepted as a Group 1 carcinogen (IARC 1980, 1987a). The six principal epidemiological studies (covering nearly 18 000 workers) reviewed by Steenland et al. (1996) indicated a combined relative risk of 3.69, with a range of 1.31–15.2 reported for individual studies and a clear dose–response relationship. The lowest relative risk arose from a study in which exposures mostly ranged from 7 to 13 $\mu g/m^3$, compared to the OSHA level of 10 $\mu g/m^3$ (Enterline et al. 1987). Excess cancers in other studies were probably due to high exposures that occurred largely in the past. A combined relative risk of 3.69 (95% CI 3.06–4.46) was determined by the Steenland et al. (1996) review, whereas 3.2 was used by Nurminen and Karjalainen (2001).

Asbestos

Both serpentine and amphibole asbestos have been shown to cause lung cancer in humans, with a clear dose–response relationship and a synergy between asbestos and tobacco (Lee 2001). Over 100 cohort studies and many case-referent studies, plus animal and cellular studies, provide ample evidence for causation. In six cohort studies of nearly 6000 asbestosis patients, the standardized mortality rate ranged from 3.5 to 9.1, with a combined relative risk of 5.9. In 20 studies of over 100 000 asbestos workers, the standardized mortality rate ranged from 1.04 for chrysotile workers to 4.97 for amosite workers, with a combined relative risk of 2.00. It is difficult to determine the exposures involved because few of the studies reported measurements, and because it is a problem to convert historical asbestos measurements in millions of dust particles per cubic foot to gravimetric units. Nevertheless, little excess lung cancer is expected from low exposure levels. These studies have been the subject of several reviews (IARC 1977; IPCS 1998; Nurminen and Karjalainen 2001; Steenland et al. 1996). The main papers provided a range of relative risks (1.04–7.4), with summary relative risks of 2.0 (Steenland et al. 1996) and 2.3 (Nurminen and Karjalainen 2001) cited in the two most recent reviews. The lower value (2.0, 95% CI 1.90–2.11), which is based on a wider range of studies, is accepted for this analysis.

Beryllium

Beryllium is an IARC Group 1 carcinogen (IARC 1993), although epidemiological evidence is rather limited. A standardized mortality rate for lung cancer of 2.0 was determined from a registry cohort of 689 women and men (Steenland and Ward 1991), and an overall standardized mortality rate of 1.24 was found in a study of 9225 male workers from seven beryllium plants (1.49 at plants with higher exposure) (Ward et al. 1992). Steenland et al. (1996) utilized a smoking-adjusted relative risk of 1.49 (no 95% CI reported), based on a beryllium plant with high exposures.

Cadmium

Cadmium is an IARC Group 1 carcinogen (IARC 1993). The best epidemiological evidence of its relationship to lung cancer comes from a cohort study by Stayner et al. (1992), although the evidence for carcinogenicity is stronger in animals and has recently been questioned in humans (Jarup and Nordberg 1998). The most recent follow-up study suggests a relative risk of 1.49 (95% CI 0.96–2.22) (Steenland et al. 1996). Nurminen and Karjalainen (2001) used 1.2, based on a Scandinavian study.

Chromium

Chromium is an IARC Group 1 carcinogen (IARC 1990a). There is ample epidemiological evidence of its causal association with lung cancer, with many cohort studies showing a dose–response relationship. Based on the largest and best designed studies of chromium production workers, producers of chromate paints and chromate plating workers, the overall relative risk is 2.78 (95% CI 2.47–3.52) (Steenland et al. 1996). Nurminen and Karjalainen (2001) used a lower relative risk of 1.4 from a hospital-based case-referent study.

Diesel exhaust

Polycyclic aromatic hydrocarbons comprise the main components of diesel exhaust, which contains a mixture of substances. Diesel exhaust has been accepted as a Group 2A carcinogen (IARC 1989) and was scheduled for further review in 2001. Owing to limitations in exposure assessment to diesel exhaust, human epidemiology has been difficult to conduct. However, cohort studies and meta-analyses confirm a relationship between diesel exhaust exposure and lung cancer, with summary relative risks in the range 1.3–1.5 (Bhatia et al. 1998; Lipsett and Campleman 1999). Based on six relatively consistent recent studies with good documentation of exposure to diesel exhaust, in which the number of cases ranged from 50 to 1256, Steenland et al. (1996) determined a combined relative risk of 1.31 (95% CI 1.13–1.44), and Nurminen and Karjalainen (2001) used the same estimate.

Nickel

Nickel is an IARC Group 1 carcinogen (IARC 1990a). Based on data from the 1990 report of the International Committee on Nickel Carcinogenesis in Man (ICNCM 1990), Steenland et al. (1996) calculated a combined relative risk of 1.56 (95% CI 1.41–1.73). Nurminen and Karjalainen (2001) used an estimate of 1.4 based on a Finnish study.

Silica

On the basis of detailed reviews, silica has been classified as an IARC Group 1 carcinogen (IARC 1987b, 1997). Several cohort studies in silica-

exposed and silicosis patients showed a dose–response relationship between silica exposure and lung cancer relative risk, and this was confirmed by meta-analyses and a pooled study (Steenland and Sanderson 2001). Animal and cellular studies provided supporting evidence. Controversy remains as to whether silicosis is a necessary precursor for the development of lung cancer, but this does not affect the underlying status of silica as a carcinogen (Checkoway 2000; Hnizdo and Sluis-Cremer 1991; Soutar et al. 2000). Steenland et al. (1996) based their combined relative risk of 1.33 (95% CI 1.21–1.45) on 13 large cohort and case–control studies of silica-exposed workers. These studies included granite workers, stone workers, pottery workers, brick workers, gold miners and diatomaceous earth miners, and covered a range of workers generally numbering from almost 1000 to over 5000. Half of the studies controlled for smoking. Nurminen and Karjalainen (2001) used a slightly higher estimate of 1.4.

Combined estimates

A common methodology, similar to that used by Steenland et al. (1996) and Nurminen and Karjalainen (2001), was used in this analysis for all lung carcinogens, in that occupational exposure to carcinogens was estimated and applied to relative risk estimates to enable the determination of attributable fractions. A mean relative risk of 1.63 was determined for eight lung carcinogens (not including radon), using data reported by Steenland et al. (1996). This was done by calculating a weighted average of the substance-specific relative risks, and weighting the substance-specific relative risks by the proportion of workers exposed to each substance to determine a mean relative risk for workers exposed to the eight lung carcinogens. This was done separately for each subregion, using the proportion of workers in each subregion exposed to specific agents to weight the relative risk for each of the agents. However, the resulting average relative risks were not clearly different from each other (all were close to 1.6).

In addition, to estimate an uncertainty range for the initial mean relative risk, a weighted average was calculated of the lower and upper 95% CI values of the relative risk reported for each substance (except beryllium, for which there were no estimated CI). These values (not to be confused with the partitioned relative risk values for low- and high-level exposure) were within 15% of the mean relative risk values. This is demonstrated for the AMR-A subregion (Table 21.19).

To produce relative risk estimates for low and high exposure, it was necessary to partition the mean relative risks into values that correspond to low- and high-level exposure. A mean relative risk (of 1.6) was determined for the United States. Based on the estimates of 90% of American workers exposed at or below about one fifth of the PEL values and 10% exposed at or above the PEL values, and an estimate of the American population-attributable fraction of lung cancer due to occupation

Table 21.19 Lung cancer relative risk, substance-specific and weighted average, for the AMR-A subregion

Carcinogen	*Combined relative risk*[a] *(95% CI)*	*Proportion of workers exposed*
Silica	1.33 (1.21–1.45)	0.0248
Cadmium	1.49 (0.96–2.22)	0.0015
Nickel	1.56 (1.41–1.73)	0.0039
Arsenic	3.69 (3.06–4.46)	0.0011
Chromium	2.78 (2.47–3.52)	0.0055
Diesel fumes	1.31 (1.13–1.44)	0.0217
Beryllium	1.49	0.0005
Asbestos	2.00 (1.90–2.11)	0.0094
Total[b]	1.59 (1.41–1.77)	

[a] Derived from major epidemiological studies.

[b] Weighted summary relative risk, weighted by the proportion of workers exposed to each contributing carcinogen.

Source: Steenland et al. (1996).

of 9% (Steenland et al. 1996), the mean relative risk of 1.6 was partitioned into a relative risk of 1.3 for low-level exposure to lung carcinogens, and 1.9 for high-level exposure. The United States ratios of the lower (1.3/1.6) and the higher (1.9/1.6) relative risks to the average relative risk were then applied to the average relative risks estimated for each subregion to produce estimated relative risks at low and high exposures for each subregion. In the same manner, upper and lower 95% CI were produced for these relative risks, based on the limits estimated for the average relative risks. The results of this process are shown in Table 21.20.

Leukaemia

Leukaemia has been linked to exposure to benzene, ionizing radiation and ethylene oxide, all of which are IARC Group 1 carcinogens (IARC 2001; WHO 1999). There is also some evidence that exposure to low-frequency electric fields may be leukaemogenic (Nurminen and Karjalainen 2001; WHO 2001). However, as this physical agent has not been included in CAREX, it has been excluded from this study.

Benzene

The causal relationship between leukaemia and benzene is well recognized, including data from cohort studies in China and the United States covering workers in chemical plants, refineries, machine production, and textile and cloth factories. Excesses of nonlymphocytic, myelogenous and acute myeloid leukaemias occurred. There is also limited evidence in

Table 21.20 Weighted summary relative risks for lung cancer for all subregions

Subregion	Summary relative risk[a]	Low exposure: Combined relative risk[a] (95% CI)	High exposure: Combined relative risk[a] (95% CI)
AFR-D	1.61	1.31 (1.17–1.45)	1.91 (1.72–2.11)
AFR-E	1.62	1.32 (1.18–1.45)	1.92 (1.72–2.12)
AMR-A	1.59	1.29 (1.14–1.44)	1.88 (1.67–2.11)
AMR-B	1.58	1.28 (1.14–1.42)	1.87 (1.67–2.08)
AMR-D	1.56	1.26 (1.13–1.41)	1.85 (1.64–2.05)
EMR-B	1.56	1.26 (1.13–1.40)	1.85 (1.64–2.05)
EMR-D	1.61	1.31 (1.18–1.45)	1.92 (1.72–2.10)
EUR-A	1.62	1.32 (1.17–1.48)	1.93 (1.71–2.16)
EUR-B	1.59	1.29 (1.15–1.44)	1.89 (1.69–2.10)
EUR-C	1.50	1.22 (1.09–1.35)	1.79 (1.59–1.97)
SEAR-B	1.58	1.28 (1.15–1.42)	1.88 (1.68–2.07)
SEAR-D	1.61	1.31 (1.17–1.45)	1.91 (1.70–2.09)
WPR-A	1.57	1.27 (1.13–1.42)	1.86 (1.65–2.08)
WPR-B	1.58	1.28 (1.14–1.42)	1.87 (1.67–2.07)

[a] Weighted summary relative risk, weighted by the proportion of workers exposed to each contributing carcinogen in each subregion.

mammals (Hayes et al. 1997; IARC 1990b). A recent review (Lynge et al. 1997) provides a low-exposure relative risk of 2.0 (95% CI 1.8–2.2) and a high-exposure relative risk of 4.0 (3.6–4.4).

Ionizing radiation

The causal relationship between ionizing radiation and leukaemia is well recognized. There is consistency across numerous studies, strong association between exposure and outcome, and evidence of a dose–response gradient. Excess leukaemia has been observed in survivors of the atomic explosions at Hiroshima and Nagasaki, and also among patients medically treated with X-rays or γ-rays. The risk of leukaemia increases over fivefold at sufficiently high doses (BEIR V 1990; IARC 2000; ICRP 1991). Models describing risk have been proposed as: Linear RR model $1+5.5$ _ dose in Sv, quadratic RR $= 1 + 0.24$ dose $+ 0.27$ dose^2 (dose in Sv) (BEIR V 1990). Relative risks of 1.22 (1.07–1.70) for low exposure and 1.57 (1.18–2.88) for high exposure are accepted as the best available estimates (BEIR V 1990; IARC 2000).

Ethylene oxide

Workers have exposure to ethylene oxide either as a sterilant or as a chemical intermediary or final product. In a study in the United States,

ethylene oxide used as a sterilant was associated with lymphatic leukaemia and non-Hodgkin's lymphoma, with a rate ratio of 1.2 estimated for 45-year exposure to 1 ppm. Other studies in Sweden and the United Kingdom of Great Britain and Northern Ireland showed nonsignificant excesses of these cancers. Of six studies of chemical plant workers (two in Sweden and one each in Germany, Italy, the United Kingdom and the United States), two found significant excesses, two found nonsignificant excesses and two found expected rates (IARC 1997). Relative risk was found to range from 1.1 to 3.5 (Steenland et al. 2003).

An approach similar to that used for lung carcinogens was applied to the leukaemogens. The separate relative risks for the development of leukaemia arising from exposures to the main relevant occupational carcinogens were combined into single summary relative risks, one for low exposure and one for high exposure. This was done separately for each subregion, using the exposure prevalence of the workforce in each subregion to weight the exposure-specific risks. However, the resulting average relative risks were not clearly different from each other. CI were estimated in the same manner, weighting the estimated CI for benzene and ionizing radiation (there were no estimated CI for ethylene oxide). Unlike lung cancer, the low- and high-exposure relative risks were available for each exposure, and these were directly incorporated into low- and high-exposure summary measures through the weighting process. An example of this approach is shown in Table 21.21, using the WPR-B subregion. The results of this approach for each subregion are shown in Table 21.22.

Table 21.21 Leukaemia relative risk, substance-specific and weighted average, for the WPR-B subregion

	Low exposure	*High exposure*	
Carcinogen	*Combined relative risk*[a] *(95% CI)*	*Combined relative risk*[a] *(95% CI)*	*Proportion of workers exposed*
Benzene	2.0 (1.8–2.2)	4.0 (3.6–4.4)	0.0051
Ionizing radiation	1.22 (1.07–1.7)	1.57 (1.18–2.88)	0.0010
Ethylene oxide	1.1	3.5	0.0003
Total[b]	1.84 (1.68–2.12)	3.60 (3.20–4.16)	

[a] Derived from major epidemiological studies.

[b] Weighted summary relative risk, weighted by the proportion of workers exposed to each contributing carcinogen.

Table 21.22 Weighted summary relative risks for leukaemia for all subregions

	Low exposure	*High exposure*
Subregion	*Combined relative risk*[a] *(95% CI)*	*Combined relative risk*[a] *(95% CI)*
AFR-D	1.88 (1.72–2.15)	3.73 (3.34–4.24)
AFR-E	1.89 (1.73–2.15)	3.75 (3.37–4.25)
AMR-A	1.91 (1.74–2.16)	3.80 (3.41–4.28)
AMR-B	1.77 (1.61–2.07)	3.38 (2.98–4.01)
AMR-D	1.91 (1.74–2.16)	3.78 (3.39–4.27)
EMR-B	1.87 (1.70–2.13)	3.66 (3.26–4.19)
EMR-D	1.89 (1.72–2.15)	3.73 (3.33–4.23)
EUR-A	1.93 (1.76–2.17)	3.86 (3.48–4.32)
EUR-B	1.83 (1.67–2.11)	3.57 (3.18–4.13)
EUR-C	1.67 (1.51–2.00)	3.06 (2.64–3.80)
SEAR-B	1.89 (1.73–2.15)	3.76 (3.37–4.26)
SEAR-D	1.81 (1.65–2.10)	3.51 (3.11–4.09)
WPR-A	1.90 (1.73–2.15)	3.77 (3.38–4.26)
WPR-B	1.84 (1.68–2.12)	3.60 (3.21–4.16)

[a] Weighted summary relative risk, weighted by the proportion of workers exposed to each contributing carcinogen in each subregion.

Estimates of risk reversibility

There are limited data on risk reversibility from occupational exposure to carcinogens. The studies from which the estimated risks arise are based on cohorts of people exposed for different periods of time, followed up for various periods of time and with various periods of time between exposure cessation and follow-up, with follow-up periods varying between zero (still exposed) and many decades. Therefore, most of the absolute and relative risks produced by the studies already depend on whatever change in risk might occur once exposure ceases. However, some indication of the extent of risk reduction that might occur is given by a recent paper by Peto et al. (2000), which examined changes in the risk of developing lung cancer as a result of stopping smoking. The study estimated that, compared to the risk in persons who continued to smoke, the risk of lung cancer in males declined to about 0.66 within 10 years, to 0.44 between 10 and 20 years, to 0.2 between 20 and 30 years, and to 0.1 after 30 years.

3. Occupational airborne particulates

There are a vast number of respiratory conditions that can arise directly or indirectly from work. However, estimating exposures, risks and attrib-

utable 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 here. All of these arise from exposure to particulates. Malignant respiratory disease is not included here because it is described in section 2.

Nonmalignant respiratory disease arises as a result of the exposure of workers to airborne agents, mostly in the form of particulates or dusts.[2] The primary route of exposure is inhalation, whereby these agents gain access to the respiratory system and are either deposited (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 (for example, 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 nonmalignant conditions of the lungs (asbestosis and COPD). Other exposures have not been well characterized, but are believed to result in certain conditions (such as some forms of occupational asthma).

3.1 Exposure variable

Causative agents of asthma

Asthma, which is a narrowing of the upper respiratory passages resulting in difficult breathing and wheezing, has both nonoccupational and occupational causes. Many hundreds of occupational agents, including some inorganic and organic dusts, have been associated with occupational asthma (Balmes et al. 2003; Chan-Yeung and Malo 1994; Venables and Chan-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. We therefore used occupation 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 entire Finnish workforce and developed relative risks for specific occupations. A similar but less extensive study based in 12 industrialized countries was also used (Kogevinas et al.

1999). Relative risks were applied to these occupational data to produce estimates of the number of deaths due to work-related asthma.

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). Because of a lack of worldwide data on the prevalence of occupational exposure to dusts and their combinations, work in specific economic subsectors was used as a surrogate for dust exposure. Relative risks were applied to these workforce data to produce estimates of the number of deaths from COPD arising from work-related exposures.

3.2 Estimating risk factor levels

The general exposure assessment methodology was described earlier. Occupation was used for asthma (Equation 2) and economic sector for COPD (Equation 1). The theoretical minimum risk corresponds to no occupational exposure above background levels to airborne particulates or other agents that cause nonmalignant respiratory disease.

Agents causing asthma

The proportion of the total population with occupational exposure to asthmagens was estimated using Equation 2. Estimates were made for each occupational category by determining the proportion of the population working in occupations that matched as closely as possible to those identified by Karjalainen et al. (2001, 2002) and for which relative risk values were provided (Table 21.23). Those not working and those employed in administration were together considered to be the nonexposed reference category (relative risk = 1). These calculations were done separately for men and women for each subregion of the world. Relative risks and the proportions exposed by occupational category were applied across all age groups from age 15 to ≥80 years.

Table 21.24 summarizes the age-adjusted distribution of the labour force into occupations matching the categories for which relative risks were identified by Karjalainen et al. (2002).

Agents causing COPD

It is not possible to estimate the proportion of the world's population exposed to the large number of agents identified in occupation-specific and agent-specific studies. Community-based studies have therefore been preferred. The most common exposure in these studies is exposure to dust and/or gas/fumes (e.g. Korn et al. 1987; Kryzanowski et al. 1986; Xu et al. 1992). Unfortunately, there are also no data to estimate the proportion of the world's workers exposed to dust and/or gas/fumes. The 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

Table 21.23 Comparison of Finnish occupational categories with 1968 ISIC codes

Finnish classification[a]	*Description*	*Examples*[a,b]	*1968 ISIC*[c]	*Description*	*Examples*	*Comments*
1	Administrative, managerial and clerical workers	No examples given	2	Administrative and managerial workers	Government officials, managers (upper level)	Combine Categories 2 and 3 (ISIC) for all economic subsectors
			3	Clerical and related workers	Office managers, government workers, secretaries, bookkeepers, stock clerks	
0	Technical, physical science, social science, humanistic and artistic workers	Engineers, medical personnel, child care givers, religious and social workers	0/1	Professional, technical and related workers	Scientists, technicians, engineers, medical and related workers, mathematicians, teachers, religious workers, artists	Use Category 0/1 (ISIC) for all economic subsectors
2	Sales workers	Wholesale and retail dealers, other sales workers	4	Sales workers	Working proprietors, sales managers, sales workers, insurance agents	Use Category 4 (ISIC) for all economic subsectors
3	Agriculture, forestry, commercial fishing	Farmers and managerial workers in agriculture, forestry and horticulture, agricultural and horticultural workers, animal husbandry workers	6	Agricultural, animal husbandry and forestry workers, fishermen, hunters	Farm managers and supervisors, agriculture and animal husbandry workers, forestry workers, fishermen	Use Category 6 (ISIC) for all economic subsectors
4	Mine and quarry workers	Miners, quarrymen				Use Category 7/8/9 for mining economic subsector

5	Transport and communications workers	Road transport workers and supervisors, transport service workers, postal services and couriers, engine room crews, motor vehicle and tram drivers, railway and station personnel, telephone switchboard operators, newspaper delivery workers, office receptionists, messengers				Use Categories 7/8/9 in ISIC in transport economic subsector (see below); does not include communications workers
6/7	Manufacturing and related workers	Workers in the following industries: textiles; smelting, metallurgical and foundry; iron and metalware; electrical; wood; painting and lacquering; other construction; food and beverage; chemical processing; packing and wrapping; stationary engine and machine; dock and warehouse; other manual	7/8/9	Production and related workers, transport equipment operators and labourers	Production supervisors, miners, quarrymen; workers in metal, wood, chemicals, textiles, food and beverages, tobacco, leather, stone, rubber, paper and construction industries, labourers, machine operators, electricians, material handlers	Use Categories 7/8/9 in all economic subsectors except mining and transport
			7/8/9	Specific transport equipment operators: motor vehicle drivers, bus, truck and tram drivers		

continued

Table 21.23 Comparison of Finnish occupational categories with 1968 ISIC codes (*continued*)

Finnish classification[a]	*Description*	*Examples*[a,b]	*1968 ISIC*[c]	*Description*	*Examples*	*Comments*
8	Service work	Firefighters and police, watch and security guards, cooks, housekeepers, domestic workers, building caretakers and cleaners, hygiene and beauty operators, launderers, dry cleaners and pressers	5	Service workers	Hotel managers, cooks, waiters, housekeepers, caretakers, beauty operators, firefighters, police	Use Category 5 (ISIC) for all economic subsectors

[a] Source: Karjalainen et al. (2001).

[b] Source: Karjalainen et al. (2002).

[c] Source: UN (2000).

Table 21.24 Proportion of the population in occupational categories based on exposure to agents causing asthma, by subregion and sex

		Proportion exposed by occupation								
Subregion	*Sex*	*Background*	*Administration*	*Technical*	*Sales*	*Agricultural*	*Mining*	*Transport*	*Manufacturing*	*Services*
AFR-D	Male	0.1595	0.0498	0.0562	0.0513	0.4612	0.0081	0.0289	0.1342	0.0510
	Female	0.4645	0.0249	0.0303	0.0324	0.3551	0.0012	0.0145	0.0482	0.0288
AFR-E	Male	0.1510	0.0524	0.0618	0.0447	0.4662	0.0082	0.0219	0.1377	0.0563
	Female	0.3498	0.0360	0.0497	0.0328	0.4171	0.0010	0.0114	0.0547	0.0475
AMR-A	Male	0.2746	0.1971	0.1080	0.0875	0.0327	0.0035	0.0196	0.1940	0.0830
	Female	0.4079	0.1772	0.1223	0.0789	0.0134	0.0005	0.0080	0.0928	0.0991
AMR-B	Male	0.1896	0.1124	0.0794	0.0665	0.1592	0.0077	0.0310	0.2225	0.1317
	Female	0.5823	0.0662	0.0671	0.0410	0.0531	0.0013	0.0032	0.0878	0.0979
AMR-D	Male	0.1785	0.1894	0.0346	0.0912	0.0521	0.0020	0.0386	0.3115	0.1021
	Female	0.6110	0.1033	0.0198	0.0466	0.0119	0.0001	0.0026	0.1328	0.0719
EMR-B	Male	0.2134	0.1042	0.1499	0.0854	0.1194	0.0046	0.0409	0.2072	0.0749
	Female	0.6903	0.0523	0.0866	0.0443	0.0293	0.0001	0.0101	0.0463	0.0407
EMR-D	Male	0.1805	0.0419	0.0490	0.1742	0.3584	0.0014	0.0000	0.1465	0.0480
	Female	0.6303	0.0110	0.0105	0.0509	0.2427	0.0002	0.0000	0.0401	0.0142

continued

Table 21.24 Proportion of the population in occupational categories based on exposure to agents causing asthma, by subregion and sex (*continued*)

		Proportion exposed by occupation								
Subregion	*Sex*	*Background*	*Administration*	*Technical*	*Sales*	*Agricultural*	*Mining*	*Transport*	*Manufacturing*	*Services*
EUR-A	Male	0.3227	0.1176	0.2145	0.0252	0.0420	0.0040	0.0000	0.1934	0.0807
	Female	0.5296	0.0942	0.1889	0.0131	0.0254	0.0008	0.0033	0.0733	0.0713
EUR-B	Male	0.2593	0.0747	0.0680	0.0400	0.2133	0.0136	0.0226	0.2490	0.0595
	Female	0.4624	0.0453	0.0552	0.0131	0.2352	0.0020	0.0041	0.1379	0.0449
EUR-C	Male	0.2700	0.0946	0.0532	0.0317	0.1536	0.0212	0.1097	0.2239	0.0421
	Female	0.4269	0.0818	0.0542	0.0512	0.0919	0.0138	0.1041	0.1239	0.0522
SEAR-B	Male	0.1756	0.0599	0.0482	0.0729	0.3664	0.0051	0.0336	0.1930	0.0452
	Female	0.4240	0.0397	0.0368	0.0812	0.2487	0.0013	0.0032	0.1244	0.0407
SEAR-D	Male	0.1502	0.0645	0.0550	0.0150	0.4634	0.0149	0.0392	0.1535	0.0444
	Female	0.5298	0.0134	0.0125	0.0028	0.3781	0.0026	0.0000	0.0509	0.0098
WPR-A	Male	0.2447	0.2058	0.1023	0.0787	0.0336	0.0014	0.0340	0.2270	0.0723
	Female	0.4795	0.1410	0.0832	0.0755	0.0281	0.0002	0.0094	0.1118	0.0713
WPR-B	Male	0.1600	0.1023	0.0655	0.0454	0.3659	0.0194	0.0370	0.1399	0.0645
	Female	0.2901	0.0928	0.0588	0.0753	0.2812	0.0066	0.0290	0.0925	0.0738

Note: See also Table 21.26.

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. We based our estimates of exposed populations on data on employment in economic sectors of agriculture, industry and service from the World Bank (2001), supplemented by data from ILO (2000) on employment in economic activities. The proportions of the population with occupational exposure at medium and high levels to agents causing COPD were estimated using Equation 1.

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. We adopted these categories with some modification to account for our lack of data on the type of manufacturing industry and for the fact that agriculture in developed and developing countries probably involves different types of exposure to respirable dust. Lacking data that would have permitted us to divide manufacturing into medium and high potential for dust exposure, we have classified it as having potentially high dust exposure, given that in much of the world manufacturing involves more dust exposure than is typical in the United States where the Korn et al. study (1987) was done (Chien et al. 2002; Gomes et al. 2001). We have defined as nonexposed those not in the workforce and those in utility trade, finance and services. Those in agriculture, manufacturing and transportation were defined as having low exposure, while those in mining and construction were defined as having high exposure. Many workers in the medium and highly exposed economic activities are in fact not exposed to dusts, but on the whole the proportions in these industries are taken to represent the approximate proportion of those ever exposed to low and high levels of dusts in the general population. In Korn et al. (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. This approach was followed in our study, in which it was assumed that the number of currently employed in specific industries corresponds roughly to the number ever occupationally exposed to dusts. The proportion exposed in different economic activities in each subregion was adjusted to account for an average labour force participation among the currently exposed in that subregion, which was applied across all ages. The results are presented in Table 21.25.

Table 21.25 Proportion of the population exposed to agents causing COPD, by subregion, sex and level of exposure

		Proportion ever exposed	
Subregion	*Exposure level*	*Male*	*Female*
	Background	0.3722	0.5920
AFR-D	Low	0.5086	0.3776
	High	0.1192	0.0305
	Background	0.3744	0.5386
AFR-E	Low	0.5051	0.4365
	High	0.1204	0.0249
	Background	0.6879	0.9056
AMR-A	Low	0.0879	0.0314
	High	0.2242	0.0630
	Background	0.5653	0.8908
AMR-B	Low	0.2336	0.0553
	High	0.2011	0.0539
	Background	0.6465	0.9337
AMR-D	Low	0.1253	0.0169
	High	0.2281	0.0494
	Background	0.5829	0.9256
EMR-B	Low	0.2007	0.0441
	High	0.2164	0.0303
	Background	0.5818	0.7780
EMR-D	Low	0.2204	0.1776
	High	0.1978	0.0444
	Background	0.6819	0.8965
EUR-A	Low	0.0565	0.0253
	High	0.2616	0.0781
	Background	0.5096	0.6598
EUR-B	Low	0.2636	0.2469
	High	0.2268	0.0933
	Background	0.4312	0.6463
EUR-C	Low	0.3273	0.2409
	High	0.2415	0.1128
	Background	0.4190	0.6694
SEAR-B	Low	0.4112	0.2384
	High	0.1698	0.0922
	Background	0.3965	0.5723
SEAR-D	Low	0.4822	0.3869
	High	0.1213	0.0408
	Background	0.5994	0.8387
WPR-A	Low	0.1200	0.0531
	High	0.2806	0.1082
	Background	0.3700	0.5244
WPR-B	Low	0.4474	0.3807
	High	0.1826	0.0949

3.3 Risk factor–disease relationships

Asthma

Occupational asthma is a condition characterized by variable airflow limitation or bronchial hyper-responsiveness related to workplace exposure. However, the precise definition of occupational asthma has been widely debated. The most controversial issue concerns whether only immunologically-mediated asthma should be considered to be occupational asthma or whether asthma arising as result of workplace exposure to irritants, or exacerbation of pre-existing asthma by workplace irritants, should also be considered in the definition (Lombardo and Balmes 2000; Malo and Chan-Yeung 2001; Wagner and Wegman 1998). Recently, consensus seems to have been reached in favour of a broad definition (American Thoracic Society review: Balmes et al. 2003). A broader approach has been supported by others (Blanc and Toren 1999; Karjalainen et al. 2001; Kogevinas et al. 1999; Milton et al. 1998; Toren et al. 1999), and recent studies of occupational asthma have tended to use a more inclusive approach (Karjalainen et al. 2001, 2002; Milton et al. 1998).

Occupational asthma is probably the most common work-related respiratory disorder in industrialized countries (Kogevinas et al. 1999), and is either stable (Singh and Davis 2002) or increasing in incidence (Sears 1997). Many hundreds of occupational agents, including some inorganic and organic dusts, have been associated with occupational asthma (Balmes et al. 2003; Chan-Yeung and Malo 1994; Venables and Chan-Yeung 1997).

Until recently, there has been limited information on the total risk of developing asthma from workplace exposure. The United States magnitude of mortality study (Steenland et al. 2003) estimated that about 5% of mortality from nonmalignant work-related respiratory disease was due to asthma. Studies of substance-specific risks have helped to identify or implicate particular substances as likely causative agents (e.g. Monso et al. 1998), but these studies have generally focused on agents thought to be sensitizers, and usually on only a limited number of these. They are therefore not useful for determining the true extent of asthma occurring as a result of work-related exposure. Several population-based studies have partially rectified this problem (Karjalainen et al. 2001, 2002; Kogevinas et al. 1996, 1999; Ng et al. 1994; Toren 1996; Toren et al. 1999), focusing on occupation-specific rather than substance-specific risks because of the plethora of potential causative exposures and the difficulty in characterizing them. These studies provided measures of relative risk and/or population-attributable fractions. Recent studies in Finland have estimated population-attributable fractions for occupational asthma of 18% (Nurminen and Karjalainen 2001) and of 17% (for women) and 29% (for men) (Karjalainen et al. 2002). A comprehensive review undertaken before these two Finnish studies found a

median value for population-attributable fraction of 9% for all relevant studies, and a median value of 15% for the highest-quality studies (Blanc and Toren 1999). The American Thoracic Society (Balmes et al. 2003) has recently reviewed the literature and estimated that approximately 15% of asthma is attributable to occupational exposure, based largely on studies in developed countries.

Of these studies, only that by Karjalainen et al. (2001, 2002) provides useable risk information to cover the whole workforce, while that by Kogevinas et al. provides useful information for agriculture. The study by Karjalainen et al. (2001, 2002) was a longitudinal study over 13 years covering the entire Finnish population, and provided relative risks for a large number of broad occupational categories. In that study, asthma was defined by the occurrence of clinically diagnosed asthma (n=49 575) during the follow-up period; national medical records were linked to census data on an individual's occupation. The study population was composed of all those currently employed, aged 25–59 years at baseline, without prior history of asthma. Relative risks were calculated by comparing the occupation-specific incidence to the incidence of occupational asthma in administrative, managerial and clerical workers, whose risk was assumed to be similar to the background population risk. The relative risks were adjusted for age, and 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 a cross-sectional study of asthma involving 15 000 people in 12 European countries. In both studies, relative risks of asthma morbidity were assumed to apply for asthma mortality. This assumption is likely to be reasonable in most circumstances, but may lead to some underestimation or overestimation of asthma mortality, depending on whether exposure results in asthma incidence or exacerbation.

The approach used here was based on the work of Karjalainen et al. (2001, 2002). The work by Kogevinas et al. (1999) was also used for the relative risk of asthma due to occupational exposure in agriculture. While the Finnish study was large, prospective and covered all occupations, there was concern that Finnish exposures within specific occupations might be atypical of the rest of the world. In particular, this was considered likely to be true for agriculture, since Finnish agriculture might involve more indoor work where the relative risks for asthma were relatively high. Therefore the Kogevinas et al. results were used for agriculture since they were believed to be more generalizable to agriculture in the rest of the world, especially the developing world.

We assumed that the relative risk of asthma morbidity owing to employment in occupational categories was approximately equal to the relative risks of asthma mortality. Those not working and those employed in administration were together considered to be the nonexposed reference category (relative risk=1). These calculations were done separately for men and women for each subregion of the world. Rela-

Table 21.26 Relative risks for occupational asthma by original occupation and economic subsector, 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
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)

tive risks and the proportions exposed by occupational category were applied across all age groups from age 15 to ≥80 years. The relative risks by occupation are shown in Table 21.26.

Chronic obstructive pulmonary disease (COPD)

Tobacco smoking is clearly the most important risk factor, but many work-related exposures have been demonstrated to cause COPD (Hendrick 1996). A recent United States study estimating the magnitude of mortality due to occupational exposure (Steenland et al. 2003) used an estimate of 14% for the population-attributable fraction for COPD due to occupational dust exposure (based on a community study of severe COPD) (Korn et al. 1987), and found that COPD represented 87% of all fatal work-related nonmalignant respiratory disease, although some of the other types of respiratory disease may have been underestimated. A review of Finnish data also used a population-attributable fraction of 14% for men (and 5% for women) (Nurminen and Karjalainen 2001), and a similar figure (15%) was recently used in a review by the American Thoracic Society (Balmes et al. 2003).

As for asthma, difficulties arise from the vast array of definite, probable and possible causes of work-related COPD. The role of smoking, particularly in causing possible confounding effects, makes interpretation of studies difficult. Apparently significant individual differences in susceptibility, and uncertainty about pathological mechanisms, also cause problems. This area has been the subject of several reviews (Attfield and Wagner 1998; Balmes et al. 2003; Becklake 1989, 1994; Hendrick 1996; NIOSH 1996; Oxman et al. 1993), some covering all exposures and some concentrating on mineral dusts.

As a result of difficulties in characterizing all the likely causative occupational exposures, few published papers provide information that com-

prehensively describes the risk of developing COPD as a result of work. The paper by Korn et al. (1987) has been used in this analysis, as it provides relative risk information covering all workplace exposures. This study (the methods of which were described in more detail in an earlier study by Ferris et al. 1979) used data from a random sample of white adults aged 25–74 years from six United States cities and their surrounding areas (8515 people were included in the final sample). The definition of COPD was $FEV_1/FVC < 0.6$, representing reasonably severe disease. Logistical regression analyses were undertaken, determining the odds ratios for various respiratory conditions and controlling for age, sex, current and lifetime smoking history and city of residence. These odds ratios for COPD morbidity from Korn et al. (1987) were assumed to apply to COPD mortality.

The study by Korn et al. (1987) provides a strict definition of COPD and relative risks for both men and women, and was based on a large number of participants. This study was therefore used as the basis of the relative risk and attributable fraction estimates presented here. Relative risks for COPD prevalence were used as an approximation of the relative risks for COPD mortality.

Korn et al. (1987) found relative risks of COPD of 1.62 for men and 1.24 for women for a history of exposure to dusts. We partitioned these relative risks into high- and low-exposure categories, and also used slightly different relative risks for low exposure in developed and developing countries. In developing countries the great majority of low-exposure employment is in agriculture, where much dust is nonrespirable. 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. The estimated relative risks are shown in Table 21.27.

Risk reversibility

As for carcinogens, there are limited data on risk reversibility. The studies from which the estimated risks arise are based on cohorts of people

Table 21.27 Annual risks of COPD mortality

	Developing countries		*Developed countries (AMR-A, EUR-A, WPR-A)*	
Relative risk	*Men*	*Women*	*Men*	*Women*
Nonexposed	1.0	1.0	1.0	1.0
Low	1.2	1.1	1.4	1.2
High	1.8	1.4	1.8	1.4

Source: Korn et al. (1987).

exposed for different periods of time, followed up for various periods of time and with various periods of time between exposure cessation and follow-up, with follow-up periods varying between zero (still exposed) and many decades. Therefore, most of the absolute and relative risks produced by the studies are already dependent on whatever change in risk might occur once exposure ceases. Indications of risk reversibility for COPD may be obtained from the literature on smoking.

4. Occupational noise

Noise is a common occupational hazard. The unit for sound (noise) level, whether measuring noise exposure or hearing loss, is the decibel (dB). Noise exposure levels as used in this document have the unit dBA.[4] Noise-induced hearing loss is reported in dBHL. There is variability in the literature in the use of the terms to describe hearing ability. As used here, hearing loss refers to a decline in an individual's hearing ability. Hearing impairment refers to the effect of hearing loss on the individual's ability to function. The U.S. National Institute for Occupational Safety and Health (NIOSH) uses the term "material hearing impairment"[5] to describe a hearing loss greater than 25 dB, and most occupational studies refer to 25 dBHL. The WHO definition used in this study is hearing loss greater than or equal to 41 dBHL. Therefore, extrapolations were made from the occupational studies to fit the requirements of the WHO study.

4.1 Exposure variable

The exposure variable used in this analysis is a direct measure of the risk factor, i.e. occupational exposure to noise, which is the causative agent of noise-induced hearing loss. As global data on the frequency of occurrence, duration and intensity of noise exposure do not exist, it was necessary to model this exposure for workers employed in various occupational categories. The theoretical minimum is based on expected background levels of noise, and consistency with national and international standards. Most experts agree that levels below 80 dBA would result in minimal risk of developing hearing loss.

For workers in various occupational categories, three levels of exposure were estimated:

- minimum exposure, less than 85 dBA;
- moderately high noise, ≥85–90 dBA; and
- high noise, >90 dBA.

The choice of these levels was based on the recommended exposure limits (RELs) for occupational noise exposure around the world. In most developed countries the REL is 85 dBA as an eight-hour time-weighted average without hearing protection. In the United States the PEL is

90 dBA for an eight-hour day, although a hearing conservation programme is required for all employees exposed above 85 dBA for an eight-hour day. In developing countries, the REL is usually 90 dBA (Ahmed et al. 2001; Alidrisi et al. 1990; Hernandez-Gaytan et al. 2000; Hessel and Sluis-Cremer 1987; Osibogun at al 2000; Shaikh 1996; Sriwattanatamma and Breysse 2000).

Although the theoretical minimum exposure to noise was determined to be 80 dBA, it was not possible to estimate frequency of exposure by occupational category to occupational noise between 80 and 85 dBA. Therefore, persons with occupational exposure <85 dBA were included with the background population.

4.2 Estimating risk factor levels

Data sources

Potentially useful studies were identified using the various approaches described in section 1. The key terms used were "occupational noise" and "occupational hearing impairment". Relevant studies were identified by critically appraising the references obtained. This included consideration of the approaches to selection, measurement, analysis and control of confounding. Potential confounders of noise-induced hearing loss include nonoccupational exposure to noise, undocumented occupational noise levels, use of personal protective equipment, use of some medicines, and outer- and middle-ear pathology. Recent review articles were used where available, and the main articles were obtained and appraised.

The main reason for excluding studies was that they did not contain data appropriate for determining risk of noise-induced hearing loss. Problems included an inappropriate (for this purpose) exposure measurement (such as reporting for only one or a few occupational groups or tasks); inappropriate (for this purpose) outcome measurement (such as dB per year loss with age or no data as to the number of cases vs total population); poorly characterized exposure or self-reported hearing loss; and inadequate control of confounding.

In the United States, about 9 million workers are exposed to time-weighted average sound levels of 85 dBA and above (Simpson and Bruce 1981, quoted in Suter 2000), and about 10 million have noise-induced hearing loss >25 dB (USDOL OSHA 2002b). About 17% of American "production workers" are exposed to average noise levels at or above 85 dBA (NIOSH 1998). In the European Union, 28% of workers surveyed reported that for at least 25% of the time they were occupationally exposed to noise loud enough to cause them to raise their voices during conversation (corresponding to approximately 85–90 dBA) (EASHW 2000). The highest percentages of exposed workers were reported for mining, quarrying, manufacturing and construction. Australia compensates about 10 000 people each year for noise-induced

hearing loss; evidence indicates that only one third of workers with noise-induced hearing loss file compensation claims (NOHSC 1993). Summary statistics on noise exposure are not available for most industrializing and nonindustrialized countries. However, most published reports indicate that average noise exposure levels are well above the recommended occupational level in many industrialized countries, which is generally established at 85–90 dB for an eight-hour work day (Suter 2000; WHO/FIOSH 2001).

Information on noise exposures and noise-induced hearing loss in developing countries is given in Table 21.28. These studies are characterized by high occupational noise exposure levels, and many report hearing losses in exposed workers. The authors generally recommended engineering controls and hearing conservation programmes, including hearing protection, indicating that hearing protection is not widely used. Seventeen studies conducted in 12 countries in South America, Africa and Asia reported noise levels in a wide range of workplaces, including mining and the manufacture of food, fabrics, printed materials, metal products, drugs and watches. Most studies provided ranges of sound levels, with the lowest reported noise levels often below 80 dBA and the upper levels always above 90 dBA. All the studies that examined the hearing ability of workers revealed increased rates of hearing impairment in noise-exposed workers compared to nonexposed controls.

Exposure estimation

Occupational exposure to elevated noise levels depends on a variety of factors, including (i) occupation and industry and (ii) workplace-specific factors such as type of facility and process, raw materials, machinery, tools, the existence of engineering and work practice controls, and the existence, condition and use of personal protective devices. Thus exposure assessment was conducted using the occupational category approach (Equation 2), modified to reflect different noise exposures in occupations in different economic subsectors.

Our estimation of the proportion of workers in each occupational category with exposure to noise at or above 85 dBA (PEW(oc(r,g)i)) was based on United States data on the prevalence of noise exposure at or above 85 dBA among production workers in nine economic subsectors (NIOSH 1998; USDHHS 1986) (see Table 21.29).

The prevalence values among production workers were calculated from the US National Occupational Exposure Survey conducted during 1981–1983 (NIOSH 1998), which estimated the number of production workers exposed to noise at or above 85 dBA, by economic subsector. All other prevalence values were estimated by us, based on the NIOSH values for production workers. The value of 0.20 calculated for production workers in agriculture was extrapolated to all agricultural workers in all economic subsectors. Similarly, the value of 0.12 for

Table 21.28 Studies of noise exposures and hearing impairment in selected developing countries

Country or area	*Facility/job*	*Sound levels*	*Hearing loss*	*Notes*	*Source*
Brazil	Rotogravure printing workers	Continuous noise levels from 71 to 93 dBA	Some 49% of 124 workers exposed to noise and organic solvents had hearing loss (>25 dB) in the high frequencies, significantly associated with age		Morata et al. (1997)
Egypt	Road traffic policemen in Cairo	Average 97 dBA with horns, 85 without; 97 at railway crossings	About 20-dB loss at all frequencies compared to office policemen		Kamal et al. (1989)
Egypt	Textile factory	78–91 dBA in wool sorting and combing units	Compared to nonexposed controls, workers exposed to <85 dBA had only 1% increase in hearing impairment after 12 years. In workers exposed to >85 dBA the increased risk was 9.6%	Hearing impairment was defined as average of left and right ear thresholds at 0.5, 1 and 2 kHz, >25 dB	Moselhi et al. (1979)
Hong Kong SAR	Five industries: weaving, bottling, metal working, spinning, airport	L_{eq} (8-hour time-weighted average, dBA): weaving 102; bottling 94; metal working 96; spinning 97; airport 80–90	Compared to controls, noise-exposed workers had significantly higher thresholds in most age groups and in all five industries, closely matching predicted values	No evidence was found for any ethnic differences between western groups and Cantonese Chinese, either in general hearing ability or in response to long-term noise exposure	Evans and Ming (1982)
India	Heavy engineering industry: machine shop and press divisions	Ranged from 83–116 dBA. At selected work sites: press 94–110; machine shop 83–92; foundry 86–116	Mean hearing threshold: 40 controls 4–24 dB; 53 machine shop employees 14–40 dB; 60 press employees 19–70 dB	Hearing impairment was progressive with age for all groups. Use of hearing protection was recommended	Raja and Ganguly (1983)

India	Textile mill weavers	102–104 dBA	120 weavers, exposed 1–15 years. In the age range 30–34 years, median threshold of audibility in the right/left ear was 55/55 compared to 15/15 for controls; for 35–39-year olds the threshold was 60/55 compared to 15/15 for controls		Bhattacharya et al. (1981)
India	Drug and pharmaceutical company	Noise levels in dBA: fermentation 100–105; air compressor 95–102; ammonia compressor 93–97; primary air filter 104–106. Night shift levels were 1–3 dBA higher	—	Authors recommended engineering controls and hearing conservation programme, including use of hearing protection	Bhattacharya et al. (1990)
India	Watch factory in Bangalore	Maximum noise levels ranged from 74 in assembly to 99 dBA in the diesel generator room	—		Mukherjee et al. (1995)
Nigeria	Car assembly	94–108 dB	Hearing thresholds of 165 workers were significantly higher than nonexposed controls, and correlated significantly with employment duration		Oleru et al. (1990)

continued

Table 21.28 Studies of noise exposures and hearing impairment in selected developing countries (*continued*)

Country or area	*Facility/job*	*Sound levels*	*Hearing loss*	*Notes*	*Source*
Nigeria	Textile workers in five factories in Lagos	Continuous noise levels of 95–115 dBA	Hearing thresholds of 61 noise-exposed workers were significantly higher than 90 nonexposed controls. After 7 years of employment, exposed workers lost 2–12 dB per year, compared to 0.6–1.8 dB per year in controls	No hearing protection worn. Exposed workers did not display 4000-Hz notch, and the shape of the audiograms[a] was convex upwards, indicating lower losses at the middle frequencies. (Typical audiograms with noise-induced hearing loss display a convex downwards shape, indicating higher losses at the middle frequencies)	Oleru (1980)
Pakistan	Polyester fibre plant	Average noise levels: filament take-up unit 93.2 dBA; texturizing unit 94.8 dBA; compressor house 99.5 dBA	—	Typical exposure is 48 hours per week in these areas. Author recommended engineering controls and hearing conservation, including use of hearing protection	Shaikh (1996)
Saudi Arabia	78 factories producing food, chemicals, plastics, metals, paper and other products	86% exceeded 85 dBA, at least in part of the factory. In 12% all of the factory exceeded 85 dBA	—	None of the factories practised noise protection	Alidrisi et al. (1990)
Singapore	Audiometric testing of noise-exposed workers is mandatory in Singapore. Most cases of noise-induced	Noise dosimetry on 46 of these cases showed a mean time-weighted exposure of 90 dBA	127 cases of NID identified from 1985–1994. On average, after 24 years of exposure, the mean hearing threshold at 1, 2 and 3 kHz was 62 dB	Author stated that NID is the leading occupational disease in Singapore, with >500 new cases per year	Tay (1996)

	deafness (NID) are in those employed in shipping and metal manufacturing, the remainder in transport, quarrying and other manufacturing				
South Africa	Gold mining (cross-sectional survey of 2667 workers in Johannesburg)	Authors quoted a noise survey in which the majority of underground and surface gold mining occupations were exposed above 85 dBA	Hearing impairment was defined as average hearing loss of >25 dB for 500, 1000 and 2000 Hz, with 5 times weighting of better ear. None of the miners <22 years had hearing impairment, rising progressively to 22% of those ≥58 years old	Use of hearing protection increased from 13% in 1979 to 17% in 1982	Hessel and Sluis-Cremer (1987)
Sudan	Cotton ginning	99–107 dB	—	Newly mechanized facility	Khogali (1970)
United Arab Republic	Textile industry (El-Mehalla El-Kobra)	Average of 98 dB in 1200–4800 Hz range; up to 103 dBA	92% (60/73) of workers exposed to noise for ≥10 years in weaving departments had mean hearing impairment of 60 dB compared to 20 dB for control group	Audiometric test methods not described; hearing impairment not defined	Noweir et al. (1968)
Zambia	Copper mines (based on author's experiences as ear, nose and throat specialist in Zambian copper belt in 1975–1977)	"Continuous noise"	100 miners tested audiometrically. Of those with over 20 years, 23% were completely deaf	No hearing protection worn by miners	Obiako (1979)

— No data.

[a] Measurement of hearing loss using an audiometer that produces sounds at specific frequencies and sound pressure levels. The hearing threshold level is a function of frequency, indicating how a person hears at a given time.

Table 21.29 Prevalence of noise exposure ≥85 dBA

Economic subsector	Occupational category						
	Professional	*Administrative*	*Clerical*	*Sales*	*Service*	*Agriculture*	*Production*[a]
Agriculture	0.05	0.05	0.05	0.12	0.12	0.20	0.20
Mining	0.05	0.05	0.05	0.12	0.12	0.20	0.85
Manufacturing	0.05	0.05	0.05	0.12	0.12	0.20	0.22
Electricity	0.05	0.05	0.05	0.12	0.12	0.20	0.15
Construction	0.05	0.05	0.05	0.12	0.12	0.20	0.18
Trade	0.02	0.02	0.02	0.12	0.12	0.20	0.13
Transport	0.02	0.02	0.02	0.12	0.12	0.20	0.12
Finance[b]	0.02	0.02	0.02	0.12	0.12	0.20	0.02
Services	0.02	0.02	0.02	0.12	0.12	0.20	0.03

[a] Source: NIOSH (1998).

[b] Based on 1.5% of workers exposed to noise in "business services".

production workers in transportation was extrapolated to all sales and service workers. The value for professional, administrative and clerical workers was extrapolated from 0.02 indicated for production workers in business services. The remaining value, 0.05 for professional, administrative and clerical workers in agriculture, mining, manufacturing, electricity and construction, was based on expert judgement.

The prevalence values were then partitioned into moderately high and high noise exposures, i.e. ≥85–90 dBA and >90 dBA, to estimate the proportions of workers exposed to moderately high and high levels of noise (EPF(r), exposure partitioning factor). Data from the United States (USDHHS 1986), taken from the 1981 Occupational Safety and Health Administration Final Regulatory Analysis for the Hearing Conservation Amendment, provide the distribution of noise exposure of over nine million American production workers (see Table 21.30). Of the 6 063 000 production workers with exposure at or above 85 dBA, slightly over half (3 407 000 or 56%) were exposed above 90 dBA. The distribution of noise exposure levels among workers exposed over 90 dBA was also used to determine that 95 dBA is a reasonable level of noise to estimate risks among the workers in the high-exposure group (>90 dBA).

The partitioning of workers by occupational category and noise level was assigned as follows, based on data in Table 21.30. Among production workers exposed at or above 85 dBA, half were considered to be exposed at ≥85–90 dBA and half exposed at >90 dBA. (Note that these partitioning values do not consider the use of personal protective equipment.) Of the agricultural workers and sales and service workers exposed at or above 85 dBA it was assumed, based on expert judgement, that approximately 70% are exposed at ≥85–90 dBA and 30% at >90 dBA.

Table 21.30 Distribution of 9 368 000 United States production workers who had noise exposure levels of 80 dBA or greater

Noise-exposure level (dBA)	*Number of workers*
80–85	3 305 000
86–90	2 656 000
91–95	1 936 000
96–100	965 000
>100	506 000
Total >85	6 063 000
Total >90	3 407 000

Source: USDOL OSHA 1981, cited in NIOSH (1991).

Table 21.31 Prevalence of noise exposure 85–90 dBA in A subregions

Economic subsector	*Occupational category*						
	Professional	*Administrative*	*Clerical*	*Sales*	*Service*	*Agriculture*	*Production*
Agriculture	0.05	0.05	0.05	0.09	0.09	0.14	0.10
Mining	0.05	0.05	0.05	0.09	0.09	0.14	0.43
Manufacturing	0.05	0.05	0.05	0.09	0.09	0.14	0.11
Electricity	0.05	0.05	0.05	0.09	0.09	0.14	0.08
Construction	0.05	0.05	0.05	0.09	0.09	0.14	0.09
Trade	0.02	0.02	0.02	0.09	0.09	0.14	0.07
Transport	0.02	0.02	0.02	0.09	0.09	0.14	0.06
Finance	0.02	0.02	0.02	0.09	0.09	0.14	0.01
Services	0.02	0.02	0.02	0.09	0.09	0.14	0.02

All professional, administrative and clerical workers with noise exposure at or above 85 dBA were assumed to be at the ≥85–90-dBA level. Tables 21.31 and 21.32 present the distribution of noise exposure levels among workers in the A subregions by occupational category within economic sectors.

In the absence of global data, it was assumed that the same proportion of workers in these occupational categories in the developing countries would be exposed to noise levels at or above 85 dBA (B, C, D, and E subregions). Given the rarity of hearing conservation programmes in the developing subregions, it was assumed that 5% of production workers would be exposed in the ≥85–90 dBA category and 95% in the >90 dBA category (as opposed to 50/50 for the A subregions). Additionally, because mechanization is not widespread for D and E subregions, the majority (95%) of the agricultural workers exposed at or above 85 dBA were assigned to the ≥85–90-dBA level. Assignment of all

Table 21.32 Prevalence of noise exposure >90 dBA in A subregions

Economic subsector	Occupational category						
	Professional	*Administrative*	*Clerical*	*Sales*	*Service*	*Agriculture*	*Production*
Agriculture	0	0	0	0.03	0.03	0.06	0.10
Mining	0	0	0	0.03	0.03	0.06	0.43
Manufacturing	0	0	0	0.03	0.03	0.06	0.11
Electricity	0	0	0	0.03	0.03	0.06	0.08
Construction	0	0	0	0.03	0.03	0.06	0.09
Trade	0	0	0	0.03	0.03	0.06	0.07
Transport	0	0	0	0.03	0.03	0.06	0.06
Finance	0	0	0	0.03	0.03	0.06	0.01
Services	0	0	0	0.03	0.03	0.06	0.02

Table 21.33 Prevalence of noise exposure 85–90 dBA in B and C subregions

Economic subsector	Occupational category						
	Professional	*Administrative*	*Clerical*	*Sales*	*Service*	*Agriculture*	*Production*
Agriculture	0.05	0.05	0.05	0.09	0.09	0.14	0.01
Mining	0.05	0.05	0.05	0.09	0.09	0.14	0.04
Manufacturing	0.05	0.05	0.05	0.09	0.09	0.14	0.01
Electricity	0.05	0.05	0.05	0.09	0.09	0.14	0.04
Construction	0.05	0.05	0.05	0.09	0.09	0.14	0.01
Trade	0.02	0.02	0.02	0.09	0.09	0.14	0.01
Transport	0.02	0.02	0.02	0.09	0.09	0.14	0.01
Finance	0.02	0.02	0.02	0.09	0.09	0.14	0.00
Services	0.02	0.02	0.02	0.09	0.09	0.14	0.00

other occupational categories was the same as for the A subregions. Tables 21.33–21.36 reflect the different partitioning for the B + C and D + E subregions.

Table 21.37 presents the proportions of workers exposed to moderately high and to high noise levels by subregion, age and sex. The proportions of males exposed to these noise levels were consistently higher than those of females, owing both to higher rates of participation in the labour force and to higher rates of females working in the services sector.

4.3 Risk factor–disease relationships

High noise levels in the workplace may cause elevated blood pressure, sleeping difficulties, annoyance and stress. Excessive noise can interfere

Table 21.34 Prevalence of noise exposure >90 dBA in B and C subregions

Economic subsector	*Occupational category*						
	Professional	*Administrative*	*Clerical*	*Sales*	*Service*	*Agriculture*	*Production*
Agriculture	0.00	0.00	0.00	0.03	0.03	0.06	0.19
Mining	0.00	0.00	0.00	0.03	0.03	0.06	0.81
Manufacturing	0.00	0.00	0.00	0.03	0.03	0.06	0.21
Electricity	0.00	0.00	0.00	0.03	0.03	0.06	0.14
Construction	0.00	0.00	0.00	0.03	0.03	0.06	0.17
Trade	0.00	0.00	0.00	0.03	0.03	0.06	0.12
Transport	0.00	0.00	0.00	0.03	0.03	0.06	0.11
Finance	0.00	0.00	0.00	0.03	0.03	0.06	0.02
Services	0.00	0.00	0.00	0.03	0.03	0.06	0.03

Table 21.35 Prevalence of noise exposure 85–90 dBA in D and E subregions

Economic subsector	*Occupational category*						
	Professional	*Administrative*	*Clerical*	*Sales*	*Service*	*Agriculture*	*Production*
Agriculture	0.05	0.05	0.05	0.09	0.09	0.19	0.01
Mining	0.05	0.05	0.05	0.09	0.09	0.19	0.04
Manufacturing	0.05	0.05	0.05	0.09	0.09	0.19	0.01
Electricity	0.05	0.05	0.05	0.09	0.09	0.19	0.01
Construction	0.05	0.05	0.05	0.09	0.09	0.19	0.01
Trade	*0.02*	*0.02*	*0.02*	0.09	0.09	0.19	0.01
Transport	*0.02*	*0.02*	*0.02*	0.09	0.09	0.19	0.01
Finance	*0.02*	*0.02*	*0.02*	0.09	0.09	0.19	0.00
Services	*0.02*	*0.02*	*0.02*	0.09	0.09	0.19	0.00

with communications in the workplace, resulting in property damage or personal injury. Tinnitus[6] and temporary threshold shift[7] may also occur. However, the most serious effect is irreversible hearing impairment, resulting from damage to the delicate hearing mechanisms of the inner ear. Noise-induced hearing loss typically begins in the frequency range (pitch) of human voices and thus interferes with spoken communication.

Noise-induced hearing loss is caused by exposure to loud noises, such as those produced by woodworking equipment, chain saws, heavy machinery, gunfire, aircraft or amplified music. Permanent hearing loss from exposure to noise may happen quite early and an audiometric notch, or initial loss at or around 4000 Hz, may be noticeable within six

Table 21.36 Prevalence of noise exposure >90 dBA in D and E subregions

Economic subsector	Occupational category						
	Professional	*Administrative*	*Clerical*	*Sales*	*Service*	*Agriculture*	*Production*
Agriculture	0.00	0.00	0.00	0.03	0.03	0.01	0.19
Mining	0.00	0.00	0.00	0.03	0.03	0.01	0.81
Manufacturing	0.00	0.00	0.00	0.03	0.03	0.01	0.21
Electricity	0.00	0.00	0.00	0.03	0.03	0.01	0.14
Construction	0.00	0.00	0.00	0.03	0.03	0.01	0.17
Trade	0.00	0.00	0.00	0.03	0.03	0.01	0.12
Transport	0.00	0.00	0.00	0.03	0.03	0.01	0.11
Finance	0.00	0.00	0.00	0.03	0.03	0.01	0.02
Services	0.00	0.00	0.00	0.03	0.03	0.01	0.03

months to one year from starting a job with a hazardous noise exposure. There is significant variation in the susceptibility to noise damage, so that two workers with the same exposure may not experience the same hearing impairment. With prolonged exposure to the same noise, hearing loss continues to worsen. For a given noise environment, most of the hearing loss occurs in the first few years, although there is a slower continuing progression as long as the noise exposure continues.

When a person is removed from the noise, hearing loss does not worsen but does remain permanent. Any additional hearing loss after termination of work in a noisy environment is due to other causes, most often presbycusis (age-related hearing loss). Most people are subject to presbycusis, which is the most common form of sensorineural hearing impairment. Data show that from as early as 30 years of age, and gradually increasing in later years, some hearing loss occurs in the general population. Individual variation is great, with around 50% of the population maintaining good hearing into old age. Other factors, such as ear infection secondary to airborne contaminants, mechanical injury or chemical substances can lead to or aggravate hearing impairment in the workplace.

Description of studies

In the literature review, only three studies were found that indicated the frequency of hearing impairment at different thresholds of hearing (Davis 1989; Malchaire 2000; Waitzman and Smith 1999). Malchaire compared the expected percentage of subjects who, at age 55, presented with hearing impairment at 25 and 50 dBHL with the personal level of exposure (Lpe) to noise in dBA, in the absence of noises >140 dB. The exposure time frame was 35 years (Table 21.38).

Table 21.37 Proportions of the working-age population occupationally exposed to different noise levels, by sex and subregion

Subregion	Sex	Exposure level	Age group (years)					
			15–29	30–44	45–59	60–69	70–79	≥80
AFR-D	Male	<85 dBA	0.87	0.84	0.84	0.86	0.89	0.95
		85–90 dBA	0.09	0.12	0.11	0.10	0.08	0.04
		>90 dBA	0.04	0.04	0.04	0.04	0.03	0.01
	Female	<85 dBA	0.92	0.90	0.90	0.92	0.96	0.98
		85–90 dBA	0.07	0.09	0.09	0.07	0.04	0.02
		>90 dBA	0.01	0.01	0.01	0.01	0.01	0.00
AFR-E	Male	<85 dBA	0.87	0.84	0.84	0.86	0.89	0.95
		85–90 dBA	0.09	0.12	0.11	0.10	0.08	0.04
		>90 dBA	0.04	0.04	0.04	0.04	0.03	0.02
	Female	<85 dBA	0.92	0.90	0.90	0.92	0.96	0.98
		85–90 dBA	0.07	0.08	0.08	0.07	0.04	0.02
		>90 dBA	0.01	0.01	0.01	0.01	0.01	0.00
AMR-A	Male	<85 dBA	0.92	0.90	0.90	0.91	0.93	0.97
		85–90 dBA	0.05	0.06	0.06	0.06	0.04	0.02
		>90 dBA	0.03	0.04	0.04	0.03	0.02	0.01
	Female	<85 dBA	0.96	0.95	0.95	0.96	0.98	0.99
		85–90 dBA	0.03	0.03	0.03	0.03	0.01	0.01
		>90 dBA	0.01	0.01	0.01	0.01	0.01	0.00
AMR-B	Male	<85 dBA	0.90	0.87	0.88	0.89	0.91	0.96
		85–90 dBA	0.05	0.06	0.06	0.05	0.04	0.02
		>90 dBA	0.06	0.07	0.07	0.06	0.05	0.02
	Female	<85 dBA	0.95	0.94	0.94	0.95	0.97	0.99
		85–90 dBA	0.03	0.03	0.04	0.03	0.02	0.01
		>90 dBA	0.02	0.03	0.03	0.02	0.01	0.01
AMR-D	Male	<85 dBA	0.91	0.89	0.89	0.90	0.93	0.96
		85–90 dBA	0.03	0.04	0.04	0.04	0.03	0.01
		>90 dBA	0.05	0.07	0.07	0.06	0.05	0.02
	Female	<85 dBA	0.95	0.94	0.94	0.96	0.97	0.99
		85–90 dBA	0.02	0.03	0.03	0.02	0.01	0.01
		>90 dBA	0.03	0.03	0.03	0.02	0.01	0.01
EMR-B	Male	<85 dBA	0.91	0.88	0.89	0.90	0.92	0.96
		85–90 dBA	0.04	0.05	0.05	0.04	0.03	0.02
		>90 dBA	0.05	0.07	0.07	0.06	0.05	0.02
	Female	<85 dBA	0.96	0.95	0.95	0.96	0.98	0.99
		85–90 dBA	0.02	0.03	0.03	0.02	0.01	0.01
		>90 dBA	0.02	0.02	0.02	0.02	0.01	0.01
EMR-D	Male	<85 dBA	0.88	0.85	0.85	0.86	0.90	0.95
		85–90 dBA	0.09	0.11	0.11	0.10	0.07	0.04
		>90 dBA	0.04	0.04	0.04	0.04	0.03	0.02
	Female	<85 dBA	0.91	0.89	0.89	0.92	0.95	0.98
		85–90 dBA	0.07	0.09	0.09	0.07	0.04	0.02
		>90 dBA	0.02	0.02	0.02	0.02	0.01	0.00
EUR-A	Male	<85 dBA	0.92	0.90	0.90	0.91	0.93	0.97
		85–90 dBA	0.05	0.06	0.06	0.06	0.04	0.02
		>90 dBA	0.03	0.04	0.04	0.03	0.02	0.01

continued

Table 21.37 Proportions of the working-age population occupationally exposed to different noise levels, by sex and subregion (*continued*)

Subregion	Sex	Exposure level	Age group (years)					
			15–29	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
	Female	<85 dBA	0.96	0.96	0.95	0.97	0.98	0.99
		85–90 dBA	0.03	0.03	0.03	0.02	0.01	0.01
		>90 dBA	0.01	0.01	0.01	0.01	0.01	0.00
EUR-B	Male	<85 dBA	0.88	0.85	0.85	0.87	0.90	0.95
		85–90 dBA	0.05	0.06	0.06	0.05	0.04	0.02
		>90 dBA	0.07	0.09	0.09	0.08	0.06	0.03
	Female	<85 dBA	0.93	0.91	0.91	0.93	0.96	0.98
		85–90 dBA	0.04	0.05	0.05	0.04	0.02	0.01
		>90 dBA	0.03	0.04	0.04	0.03	0.02	0.01
EUR-C	Male	<85 dBA	0.88	0.85	0.85	0.87	0.90	0.95
		85–90 dBA	0.04	0.05	0.05	0.04	0.03	0.02
		>90 dBA	0.08	0.10	0.10	0.09	0.07	0.04
	Female	<85 dBA	0.93	0.92	0.92	0.94	0.96	0.98
		85–90 dBA	0.02	0.03	0.03	0.02	0.01	0.01
		>90 dBA	0.04	0.05	0.05	0.04	0.02	0.01
SEAR-B	Male	<85 dBA	0.88	0.84	0.84	0.87	0.91	0.95
		85–90 dBA	0.06	0.08	0.08	0.06	0.04	0.02
		>90 dBA	0.06	0.08	0.08	0.07	0.05	0.03
	Female	<85 dBA	0.92	0.88	0.88	0.91	0.96	0.98
		85–90 dBA	0.05	0.08	0.08	0.05	0.02	0.01
		>90 dBA	0.04	0.04	0.04	0.03	0.02	0.01
SEAR-D	Male	<85 dBA	0.87	0.79	0.80	0.84	0.88	0.94
		85–90 dBA	0.09	0.15	0.15	0.11	0.08	0.04
		>90 dBA	0.04	0.05	0.05	0.05	0.04	0.02
	Female	<85 dBA	0.91	0.94	0.95	0.96	0.98	0.99
		85–90 dBA	0.07	0.04	0.03	0.02	0.01	0.01
		>90 dBA	0.02	0.02	0.02	0.02	0.01	0.00
WPR-A	Male	<85 dBA	0.92	0.90	0.90	0.92	0.96	0.98
		85–90 dBA	0.04	0.06	0.06	0.04	0.02	0.01
		>90 dBA	0.03	0.04	0.04	0.03	0.03	0.01
	Female	<85 dBA	0.95	0.94	0.94	0.96	0.97	0.99
		85–90 dBA	0.03	0.04	0.04	0.03	0.02	0.01
		>90 dBA	0.02	0.02	0.02	0.01	0.01	0.00
WPR-B	Male	<85 dBA	0.87	0.84	0.84	0.86	0.89	0.95
		85–90 dBA	0.06	0.08	0.08	0.07	0.05	0.03
		>90 dBA	0.07	0.08	0.08	0.07	0.06	0.03
	Female	<85 dBA	0.93	0.91	0.91	0.93	0.96	0.98
		85–90 dBA	0.04	0.05	0.05	0.04	0.02	0.01
		>90 dBA	0.03	0.04	0.04	0.03	0.02	0.01

Table 21.38 Expected percentages of workers with hearing loss of >25 dB or >50 dB after 35 years of exposure, by personal level of exposure

	Personal exposure (dBA)							
Hearing loss	*85*	*90*	*92*	*94*	*97*	*98*	*99*	*100*
50 dB	4	5	7	9	15	18	21	26
25 dB	29	36	40	46	59	65	70	75

Source: Malchaire (2000).

Waitzman and Smith (1999) analysed data from the United States Health Examination Survey (1960–1961) and the National Health and Nutrition Examination Survey (NHANES I, 1971–1975). Hearing loss was rated by a scheme developed by Klockhoff et al. (1974). The Klockhoff analysis used four hearing loss levels (Slight, Moderate, Severe1, Severe2), which were related in this analysis to the standard hearing impairment scales by using typical noise-induced hearing loss curves. The categories developed by Klockhoff et al. were based on the frequencies used in the National Health Examination Survey (500, 1000, 2000, 3000, 4000 and 6000 Hz), and the NHANES I study (500, 1000, 2000 and 4000 Hz). As an example of this conversion procedure, the criteria of Klockhoff et al. for slight hearing loss were based on a 30-dB loss at 4000 Hz. Using audiometric data presented by Klockhoff et al. (1974), we estimated the equivalent hearing losses for each category as follows:

- Slight: >21 dBHL
- Moderate: >38 dBHL
- Severe1: >41 dBHL
- Severe2: >50 dBHL

Waitzman and Smith reported odds ratios calculated by multivariate regressions for two age groups for workers in construction, manufacturing/mining and other subsectors (see Table 21.39). Blue-collar construction workers experienced between 2 and >3.5 times the risk experienced by white-collar workers in "other industries". The pattern of their hearing loss at normal speech frequencies significantly disrupted their ability to communicate.

Davis (1989) reported on the prevalence of hearing loss as a function of age in the adult population of Great Britain. Audiometric analyses on adults ranging in age from 17 to ≥80 years were conducted in four cities. Hearing impairment was reported for >25, >45 and >65 dBHL. Davis found a "significant" level of hearing loss (>25 dBHL) in 16% of the adult population (17–≥80 years).

Table 21.39 Odds ratios from logistic multivariate regressions on audiometric measures of hearing loss, by age group

	Ages 25–44 years				*Ages 45–65 years*			
Type of worker/industry	*Slight >21 dB*	*Moderate >38 dB*	*Severe 1 >41 dB*	*Severe 2 >50 dB*	*Slight >21 dB*	*Moderate >38 dB*	*Severe 1 >41 dB*	*Severe 2 >50 dB*
White collar								
Construction	0.85	0.00	0.00	0.00	2.50	2.17	1.68	2.18
Manufacturing/ mining	0.91	1.45	1.17	1.14	1.02	1.43	0.98	1.25
Other industry groups	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Blue collar								
Construction	2.79	3.50	2.34	2.65	3.08	3.54	1.98	2.38
Manufacturing/ mining	2.01	3.03	1.94	2.40	2.33	1.86	1.88	1.90
Other industry groups	1.38	2.42	1.92	1.95	1.84	1.94	1.40	1.69

Source: Waitzman and Smith (1999).

Several studies were found that presented relative risk estimates by specific occupation. The risk estimates were higher than those determined in this analysis for exposed workers, as they focused on occupations with generally high noise exposures. These studies were based in Canada (Hessel 2000), Germany (Arndt et al. 1996) and Great Britain (Palmer et al. 2001) (see Table 21.40).

In Germany, a prevalence ratio for hearing loss of 1.5 was found in construction workers vs white-collar employees. Hearing impairment was defined as the sum of thresholds at 2000, 3000 and 4000 Hz greater than 105 dB at least in one ear. Hessel (2000), in a similar study in Canada, found that construction workers (with the exception of boilermakers) had a lower prevalence of hearing loss than the Germans. Also, prevalence of hearing impairment in the comparison group in Canada was lower than in Germany. According to the author, the differences found may be due to the year of the study. The Canadian study was carried out 7–9 years later than the German study, and there may have been lower occupational noise levels and/or use of personal hearing protection. These potentially confounding factors were not described in the German study.

In contrast to the Canadian and German studies, prevalence ratios determined in Great Britain were based on self-reported hearing impairment. Prevalence of "ever employed in a noisy job" was compared against "never exposed in a noisy job". A noisy place was defined as one "where there was a need to shout to be heard". The questions used to define hearing impairment were modelled on those used in the Institute

Table 21.40 Prevalence ratio of occupational noise-induced hearing impairment in available studies

Country	*Reference*	*Definition of hearing impairment*	*Occupation*	*Prevalence ratio*	*95% CI*
Canada (Edmonton)	Hessel (2000)	Greater than 105 dB hearing loss at 2, 3, 4 kHz (corresponds to >35 dBHL)	Plumbers	2.91	—
			Boilermakers	3.88	—
			Electricians	1.46	—
Germany	Arndt et al. (1996)	Greater than 105 dB healing loss at 2, 3, 4 kHz (corresponds to >35 dBHL)	Carpenters	1.77	1.48–2.12
			Unskilled workers	1.75	1.47–2.09
			Plumbers	1.49	1.19–1.85
			Painters	1.2	0.96–1.49
			Plasterers	1.29	1.05–1.59
			Overall	1.5	1.29–1.82
Great Britain	Palmer et al. (2001)	Severe: wearing a hearing aid or having great difficulty in both ears on hearing conversation in a quiet room >45 dBHL	Male	2.9	—
			Female	1.8	—
		Moderate and worse: reported moderate difficulty in hearing conversation in a quiet room equivalent to 45 dBHL	Male	3.6	—
			Female	2.9	—

— No data.

of Hearing Research national survey of hearing, in which those who reported moderate or worse hearing impairment were found to have a mean hearing loss of 45 dBHL. The mean hearing loss of 45 dBHL is similar to the cut-off of 41 dBHL used by WHO and the Global Burden of Disease study, whereas the cut-off used in the German and Canadian studies corresponded to slight hearing impairment (>35 dBHL).

NIOSH, in a re-analysis of the data from its Occupational Noise and Hearing Survey (Prince et al. 1997) derived excess risk[8] estimates with a model that used the average of 1000, 2000, 3000 and 4000 Hz and a hearing loss >25 dBHL. We used this information to develop excess risk estimates for workers exposed at 85–90 dBA (defined by us as moderately high exposure) and >90 dBA (defined by us as high noise exposure, equivalent to 95 dBA). To estimate excess risks for workers exposed to moderately high noise, we used the observed exposure–response relationships developed by NIOSH (1998) for workers of different ages who were exposed at 80, 85 and 90 dBA for various numbers of years. The data show that at any noise level, hearing impairment increases with age and/or length of exposure. Also, the highest risk is found at the highest levels of exposure. Prince et al. (1997) found a small increase in excess risk in workers exposed to 80–84 dBA vs a <80 dBA control group; however, these risk estimates are imprecise owing to the low numbers of workers in the study exposed to noise at these levels.

NIOSH (1998) also provides two data points of excess risk for workers exposed at 95 dBA for prolonged periods. Table 21.41 illustrates our estimation of excess risk of material hearing impairment at >25 dBHL for workers exposed at 95 dBA, based on these two data points. The excess risk value of 38.3% at 95 dBA for a 65-year-old worker after 10 or more years of exposure was taken from NIOSH (1998), Appendix Table IV. In addition, an excess risk value of 19.5% was taken from the table for 30-year-old workers exposed to noise at 95 dBA and for a duration of exposure of 5–10 years. The values for 30-, 40- and 50-year olds with >10 years of exposure were interpolated using the ratios of change of hearing loss with age at 90 dBA between each age group in Table 21.41. All calculations of NIOSH exposure–response relationships were based on material hearing impairment at >25 dBHL and were adjusted by us to reflect noise-induced hearing loss at ≥41 dBHL.

The International Organization for Standardization (ISO) has also developed procedures for estimating hearing loss due to noise exposure. Their most recent model (referred to as the "1990-ISO model") and the 1997 NIOSH model (NIOSH 1998) are reasonably similar. Table 21.42 summarizes the excess risk estimates developed separately by NIOSH and ISO for material hearing impairment >25 dBHL caused by occupational noise exposure.

Table 21.41 Excess risk estimates for material hearing impairment >25 dBHL, by age and duration of exposure

	Excess risk (%)			
Average daily exposure (dBA)	*Age 30*	*Age 40*	*Age 50*	*Age 60*
5–10 years of exposure				
95[a] (estimated)	19.5			
90	5.4	9.7	14.3	15.9
85	1.4	2.6	4	4.9
80	0.2	0.4	0.6	0.8
>10 years of exposure				
95[a] (estimated)	24	31	38	38.3
90	10.3	17.5	24.1	24.7
85	2.3	4.3	6.7	7.9
80	0.3	0.6	1	1.3

[a] Estimates for 95 dBA were developed from NIOSH 1998 using methods described in the text.

Source: NIOSH (1998).

Table 21.42 NIOSH and ISO estimated excess risk of incurring material hearing impairment (>25 dBHL at 1, 2, 3 and 4 kHz) over a 40-year working lifetime and at various average noise exposures

	Excess risk (%)	
Average daily noise exposure (dBA)	*ISO*	*NIOSH*
90	17	25
85	6	8
80	1	1

Source: NIOSH (1998).

EXTRAPOLATING FROM RISKS AT >25 dBHL TO RISKS AT ≥41 dBHL

Occupational hearing loss is usually denoted as >25 dBHL but WHO uses ≥41 dBHL as a cut-off point to estimate prevalence of hearing loss. Therefore, extrapolations were made from studies of occupational risks at >25 dBHL to estimate risk to workers of hearing loss at 41 dBHL or greater. Data from the USDOL OSHA 1981 Final Regulatory Analysis for the Hearing Conservation Amendment (NIOSH 1991) provided a means of adjusting the various reports based on material hearing impairment >25 dBHL to >40 dBHL, a level of hearing loss assumed for this project to be equivalent to the WHO definition of ≥41 dBHL. As presented in Table 21.43, OSHA estimated the number of workers with various levels of hearing loss or impairment. The number of expected

Table 21.43 Hearing levels (dBHL) of 9368000 United States production workers with noise exposure levels of ≥80 dBA

Hearing threshold level (1, 2 and 3 kHz)	*Cumulative cases*	*Expected cases*	*Excess cases*
>15 dB (mild hearing loss)	3735000 (40%)	2111000 (23%)	1624000 (17%)
>25 dB (material hearing impairment)	2025000 (22%)	965000 (10%)	1060000 (11%)
>40 dB (moderate to severe hearing impairment)	718000 (8%)	245000 (3%)	473000 (5%)

Source: USDOL OSHA, 1981, cited in NIOSH (1991).

Table 21.44 Estimated excess risk of incurring hearing impairment at 41 dBHL or greater over a 40-year working lifetime and at various average noise exposures

	Excess risk (%)	
Average daily noise exposure (dBA)	*ISO*	*NIOSH*
90	7.6	11.2
85	2.7	3.6
80	0.4	0.4

cases (based on hearing levels of a nationwide sample of adults in U.S. Public Health Service hearing surveys) was subtracted to derive the number of excess cases at various levels of hearing loss or impairment in United States production workers (OSHA 1981, reported in NIOSH 1991).

Using the data in Table 21.43, a factor to correct excess risk data at >25 dBHL to WHO's excess risk at ≥41 dBHL was determined as a ratio of the number of excess cases at >40 dB divided by the number of excess cases at >25 dBHL; i.e. 473000 divided by 1060000 yields a ratio of 0.446. This correction factor of 0.446 was used to correct excess risk at >25 dBHL from the reported excess risk in Table 21.42 to the excess risk at ≥41 dBHL as presented in Table 21.44. As no additional data were available, the hearing impairment of production workers at >40 dBHL was assumed to be equivalent to the WHO definition of hearing loss of 41 dBHL or greater used in this project.

Excess risk estimates for hearing impairment at ≥41 dBHL are presented in Table 21.45. They were generated by applying the same correction factor of 0.446 to Table 21.41.

Relative risk estimation for noise induced hearing loss at ≥41 dBHL

The relative risk values were extrapolated using the following formula:

Table 21.45 Estimated excess risk for hearing impairment at ≥41 dBHL, by age and duration of exposure

	Excess risk (%)			
Average daily exposure (dBA)	*Age 30*	*Age 40*	*Age 50*	*Age 60*
5–10 years of exposure				
95	8.7			
90	2.4	4.3	6.4	7.1
85	0.6	1.2	1.8	2.2
80	0.1	0.2	0.3	0.4
>10 years of exposure				
95	10.7	13.8	16.9	17.0
90	4.6	7.8	10.7	11.0
85	1.0	1.9	3.0	3.5
80	0.1	0.3	0.4	0.6

$$\text{Relative risk} = 1 + (\text{excess risk} / \text{expected risk})$$

Excess risk is defined in this study as "the percentage of workers with a hearing impairment in an occupationally noise-exposed population, minus the percentage who would normally incur such impairment from aging in an unexposed population". The expected risk is the prevalence for the general unexposed population. While the NIOSH document provides the excess risk of the exposed population, the expected risk is not reported by NIOSH. Data from Davis (1989) estimates prevalence as a function of age in the adult population of Great Britain. The average prevalence for both ears for a noise-induced hearing loss of >45 dBHL was calculated by us for the general population, using the data from Davis (1989) and the methods described above to adjust NIOSH data for noise-induced hearing loss at >25 dBHL to generate Table 21.45. The results are reported in Table 21.46.

In our study, relative risks for the age groups 0–4 and 5–14 years were not estimated, as occupational risks are not present and/or data are unavailable on levels or length of exposure. In the calculation of excess risk, the age group 15–29 years was assigned the lowest excess risk value of 8.7 in Table 21.45 for age 30 with 5–10 years of exposure. For the category of workers with moderately high noise exposure (85–90 dBA), the excess risk estimate is the geometric mean of the excess risk estimates for 85 dBA and 90 dBA for each age group (see Table 21.45).

The older age groups (30–44 years, 45–59 years, etc.) did not neatly fit the age categories in Table 21.45, so worker-population-weighted averages were constructed for excess risk values at the required ages. For example, the excess risk estimate for the age group 30–44 years, at 85–90 dBA, was calculated by first taking the geometric mean of the excess risk

Table 21.46 Prevalence of hearing loss at >45 dBHL for the general population of Great Britain

Age group (years)	*Prevalence*
17–30	1.25
31–40	1.90
41–50	4.75
51–61	6.40
61–70	9.35
71–80	16.55
≥81	25.35

Source: Davis (1989).

Table 21.47 Relative risks by age group and level of exposure

	Age group (years)					
Level	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
<85 dBA	1.00	1.00	1.00	1.00	1.00	1.00
85–90 dBA	1.96	2.24	1.91	1.66	1.12	1.00
>90 dBA	7.96	5.62	3.83	2.82	1.62	1.00

estimates at 85 and 90 dBA, for people with >10 years of exposure in the 30- and 40-year-old categories. These values were then weighted by the worker population in the age groups 30–39 and 40–44 years. In a similar procedure, the prevalence values in Table 21.46 for the general population were adjusted to the same age groups. Relative risks for the age groups 70–79 years and ≥80 years were calculated from figures in Prince et al. (1997) and the prevalence data from Davis (1989). Table 21.47 presents the relative risks by age group and level of exposure.

Risk reversibility

It was assumed that risk reversibility following exposure removal was immediate. In other words, for those previously exposed who have not developed noise-induced hearing loss yet, the risk is removed after exposure removal (no new cases). Those with the condition, however, will continue to be affected by it.

5. Occupational ergonomic stressors

The physical ergonomic features of work that are most frequently cited as risk factors for MSDs include a rapid pace of work and repetitive motion patterns; insufficient recovery time; heavy lifting and other forceful manual exertions; non-neutral body postures (either dynamic or

static); mechanical pressure concentrations; vibration (both segmental and whole-body); and low temperature. For the present analysis, the risk factor is exposure to the combination of occupational exposures that are implicated in the etiology of low back pain, including physical stressors and possibly psychosocial or work organization features as well.

5.1 Exposure variable

Assessing the fraction of back pain disorders that can be attributed to occupation requires that an indicator be identified that can be measured on a global scale and that can also be matched with data on known exposure–risk relationship(s). The various reviews of low back pain epidemiology have implicated an overlapping set of occupational exposures, such as lifting, forceful movements, awkward postures, whole-body vibration and perhaps psychosocial stressors. However, such exposures are rarely assessed in surveillance activities on a large scale, and thus data are not available for risk assessment calculations at the global level.

In contrast, low back pain (and other MSD morbidity) is commonly reported by broad industrial or occupational groupings. Thus, even though occupation is a less precise indicator of risk than a specific exposure, its widespread availability in administrative data sets and some epidemiological studies makes it useful in this context. Some epidemiological studies have also provided sufficient data to relate back pain to the same occupational categories. Occupation was therefore considered as a proxy for specific risk factors. The exposure variable is an occupational category with its assigned level of risk (low, medium or high rate of low back pain). This method thus required the assumption that the distribution of the combined individual risk factors is similar within each occupational group across geographical regions. This argument applies to psychosocial as well as physical exposures.

Given that differences can occur within occupations, the assumed homogeneity of occupational groups in their total exposure to ergonomic risk factors implies that differences in exposure among occupations are substantially larger than differences among workers within the occupation. Although this assertion appears self-evident, only a few investigators to date have examined it explicitly. Burdorf (1992) evaluated the homogeneity of the exposure to postural load on the back within and among four occupational groups, and reported that the exposure variability within occupational groups was small compared with differences among groups. The estimated contribution of the variance for postural load between occupational groups was the largest source of variance. Hollman et al. (1999) and Paquet et al. (1999) have similarly shown that, even within one subsector (health care and construction, respectively), differences in ergonomic exposures among jobs can be large relative to the variability within jobs. These studies provide strong evidence in support of the approach taken in this analysis. Although the literature is less conclusive regarding their effects, to the extent that any specific

ergonomic factor is etiologically important it is assumed to be internalized in the relative risks by occupation. At the same time, specific analyses at the national or local level could increase the precision by assessing specific physical risk factors at the workplace.

5.2 Theoretical minimum risk

For low back pain, "theoretical minimum risk" is considered to represent the level of disease that would occur in the population if all excessive physical workload were abated by effective implementation of ergonomic control measures. While interventions to reduce ergonomic stressors have not yet been widely implemented on a global scale, studies are available from selected settings demonstrating the great potential of exposure (and disease) reduction in this area. Certain interventions have shown that removal of ergonomic stressors can practically lead to the removal of back pain (or its reduction to negligible levels), which justifies the choice of theoretical minimum. For example, in jobs where the entire work activity consists of manually handling materials, lifting equipment can successfully reduce both the biomechanical exposure to the lower back and the risk of low back disorders (Marras et al. 2000).

Westgaard and Winkel (1997) reviewed 89 studies on ergonomic intervention studies, of which 20 were classified as mechanical exposure interventions and 32 as production system interventions. Most mechanical exposure interventions target the ergonomic exposure level directly, through redesign of the work station and work methods. The reviewers concluded that in work situations whose mechanical exposure level is initially high, a reduction in the level of mechanical exposure may be beneficial for musculoskeletal health. For comparison, several other risk factors considered in this chapter have not yet shown that interventions can have such highly effective results when applied to selected population groups.

Since occupation represents a proxy for the composite of etiological exposures, rather than being the exposure *per se*, it is not necessary that persons leave one occupation for another in order to achieve the theoretical minimum risk. Reduction in relative risk would occur through improved job design to reduce exposures within each occupational category. The number of individuals working in each category could remain constant, even though the nature of the risk in each category would change.

5.3 Estimating risk factor levels

Since data were not available worldwide on the prevalence of specific ergonomic exposures, occupations were grouped here by their risk of low back pain. Thus, for this outcome, estimation of risk factor levels is not independent of the levels of disease assigned to them in the next step (see section 5.4). The exposure assessment by occupation was utilized, as described in the Introduction. Using managers and professionals as a

baseline for comparison, epidemiological studies have indicated that clerical and sales workers have a slightly elevated risk, operators and service workers have a moderate risk, and farmers have the highest risk of low back pain (see section 5.4 for details).

The basic approach was to determine economic activity by subregion, age and sex, and then to determine the distribution of the working population in the various occupational categories. As each occupational category was assigned a single relative risk factor (based on methodology described below), it was not necessary to partition exposures into "high" or "low" levels.

The estimates of occupations with risk of low back pain were based on the published literature. The 1968 International Standard Classification Codes for Occupations utilizes the term "production workers", whereas the epidemiological studies refer to "operators". Based on the literature, we made the following assignments.

- Background exposure: professional and administrative workers
- Low exposure: clerical and sales workers
- Moderate exposure: operators (production workers) and service workers
- High exposure: farmers

The non-working population is not considered in this analysis, and is attributed the same relative risk as the background exposure category. It is likely that younger workers are represented more often in the production occupations, and that older workers have more opportunities to move into management and administration positions.

As seen from Table 21.48, the majority of the working-age population is employed in occupations with exposure to factors linked to low back pain. Males have higher exposure in general, owing to higher rates of participation in the labour force. Exposures are higher for men in the less developed subregions, owing to a higher proportion of workers in agriculture than in the more developed subregions.

5.4 Risk factor–disease relationships

Pain in the soft tissues of the back is extremely common among adults. In the United States, the National Arthritis Data Workgroup reviewed national survey data showing that each year some 15% of adults report frequent back pain or pain lasting more than two weeks (Lawrence et al. 1998). Back pain is widespread in many countries, and is associated with substantial financial costs and loss of quality of life. In Canada, Finland and the United States, more people are disabled from working as a result of MSDs—especially back pain—than from any other group of diseases (Badley et al. 1994; Pope et al. 1991; Riihimäki 1995a). MSDs also constitute a major proportion of all registered and/or

Table 21.48 Proportions of the working-age population occupationally exposed to different levels of ergonomic stressor, by sex and subregion

Subregion	*Sex*	*Exposure category*	*Age group (years)*					
			15–29	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	Background	0.29	0.11	0.13	0.22	0.40	0.70
		Low	0.09	0.11	0.10	0.09	0.07	0.04
		Moderate	0.20	0.25	0.25	0.22	0.17	0.09
		High	0.42	0.53	0.52	0.46	0.35	0.18
	Female	Background	0.53	0.42	0.41	0.55	0.73	0.87
		Low	0.05	0.06	0.06	0.05	0.03	0.01
		Moderate	0.09	0.11	0.11	0.08	0.05	0.02
		High	0.34	0.41	0.42	0.32	0.19	0.09
AFR-E	Male	Background	0.29	0.12	0.14	0.22	0.40	0.70
		Low	0.08	0.10	0.10	0.09	0.07	0.03
		Moderate	0.20	0.25	0.25	0.22	0.17	0.09
		High	0.42	0.53	0.52	0.47	0.36	0.18
	Female	Background	0.41	0.34	0.37	0.50	0.67	0.83
		Low	0.06	0.07	0.07	0.05	0.04	0.02
		Moderate	0.11	0.13	0.12	0.10	0.06	0.03
		High	0.41	0.46	0.44	0.35	0.23	0.12
AMR-A	Male	Background	0.49	0.33	0.37	0.64	0.91	0.95
		Low	0.18	0.24	0.23	0.13	0.03	0.02
		Moderate	0.29	0.39	0.36	0.21	0.05	0.03
		High	0.03	0.04	0.04	0.02	0.01	0.00
	Female	Background	0.58	0.47	0.53	0.79	0.95	0.98
		Low	0.19	0.24	0.21	0.09	0.02	0.01
		Moderate	0.22	0.28	0.24	0.11	0.02	0.01
		High	0.01	0.02	0.02	0.01	0.00	0.00
AMR-B	Male	Background	0.32	0.16	0.23	0.43	0.71	0.86
		Low	0.15	0.19	0.17	0.13	0.06	0.03
		Moderate	0.37	0.46	0.43	0.32	0.16	0.08
		High	0.15	0.19	0.17	0.13	0.06	0.03
	Female	Background	0.63	0.57	0.69	0.84	0.94	0.97
		Low	0.10	0.12	0.09	0.04	0.02	0.01
		Moderate	0.21	0.24	0.18	0.09	0.03	0.02
		High	0.06	0.07	0.05	0.03	0.01	0.00
AMR-D	Male	Background	0.41	0.19	0.20	0.29	0.49	0.75
		Low	0.15	0.21	0.20	0.18	0.13	0.06
		Moderate	0.39	0.55	0.53	0.48	0.34	0.17
		High	0.04	0.06	0.06	0.05	0.04	0.02
	Female	Background	0.70	0.62	0.69	0.77	0.86	0.93
		Low	0.09	0.11	0.09	0.07	0.04	0.02
		Moderate	0.20	0.26	0.21	0.16	0.09	0.05
		High	0.01	0.01	0.01	0.01	0.01	0.00
EMR-B	Male	Background	0.48	0.23	0.27	0.41	0.64	0.82
		Low	0.15	0.22	0.21	0.17	0.10	0.05
		Moderate	0.27	0.40	0.38	0.31	0.19	0.09
		High	0.10	0.15	0.14	0.11	0.07	0.03

Table 21.48 Proportions of the working-age population occupationally exposed to different levels of ergonomic stressor, by sex and subregion (*continued*)

Subregion	Sex	Exposure category	Age group (years)					
			15–29	30–44	45–59	60–69	70–79	≥80
	Female	Background	0.77	0.74	0.82	0.87	0.94	0.97
		Low	0.10	0.11	0.08	0.05	0.03	0.01
		Moderate	0.10	0.12	0.08	0.06	0.03	0.01
		High	0.03	0.04	0.02	0.02	0.01	0.00
EMR-D	Male	Background	0.32	0.10	0.13	0.29	0.59	0.80
		Low	0.19	0.25	0.24	0.20	0.11	0.06
		Moderate	0.17	0.23	0.22	0.18	0.10	0.05
		High	0.32	0.43	0.41	0.33	0.19	0.10
	Female	Background	0.64	0.59	0.64	0.76	0.88	0.94
		Low	0.06	0.07	0.06	0.04	0.02	0.01
		Moderate	0.05	0.06	0.05	0.04	0.02	0.01
		High	0.24	0.28	0.24	0.16	0.08	0.04
EUR-A	Male	Background	0.58	0.38	0.46	0.78	0.97	0.98
		Low	0.11	0.16	0.14	0.06	0.01	0.00
		Moderate	0.27	0.40	0.35	0.14	0.02	0.01
		High	0.04	0.06	0.05	0.02	0.00	0.00
	Female	Background	0.67	0.59	0.69	0.92	0.99	0.99
		Low	0.11	0.14	0.11	0.03	0.00	0.00
		Moderate	0.19	0.23	0.18	0.04	0.01	0.00
		High	0.03	0.04	0.03	0.01	0.00	0.00
EUR-B	Male	Background	0.36	0.15	0.29	0.64	0.80	0.90
		Low	0.10	0.13	0.11	0.06	0.03	0.01
		Moderate	0.33	0.45	0.37	0.19	0.10	0.05
		High	0.21	0.28	0.23	0.12	0.06	0.03
	Female	Background	0.51	0.32	0.48	0.80	0.89	0.95
		Low	0.05	0.07	0.05	0.02	0.01	0.01
		Moderate	0.20	0.27	0.21	0.08	0.04	0.02
		High	0.25	0.34	0.26	0.10	0.05	0.03
EUR-C	Male	Background	0.35	0.13	0.20	0.73	0.90	0.95
		Low	0.11	0.14	0.13	0.04	0.02	0.01
		Moderate	0.39	0.52	0.48	0.16	0.06	0.03
		High	0.15	0.20	0.19	0.06	0.02	0.01
	Female	Background	0.47	0.18	0.35	0.85	0.96	0.98
		Low	0.13	0.20	0.15	0.04	0.01	0.01
		Moderate	0.31	0.48	0.38	0.09	0.03	0.01
		High	0.10	0.15	0.12	0.03	0.01	0.00
SEAR-B	Male	Background	0.32	0.09	0.13	0.33	0.59	0.80
		Low	0.11	0.14	0.13	0.10	0.06	0.03
		Moderate	0.25	0.33	0.32	0.25	0.15	0.07
		High	0.33	0.44	0.42	0.32	0.20	0.10
	Female	Background	0.49	0.36	0.40	0.60	0.81	0.90
		Low	0.11	0.13	0.13	0.08	0.04	0.02
		Moderate	0.16	0.21	0.19	0.13	0.06	0.03
		High	0.24	0.30	0.28	0.19	0.09	0.05

continued

Table 21.48 Proportions of the working-age population occupationally exposed to different levels of ergonomic stressor, by sex and subregion (*continued*)

Subregion	Sex	Exposure category	Age group (years)					
			15–29	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
SEAR-D	Male	Background	0.29	0.10	0.13	0.34	0.51	0.76
		Low	0.06	0.08	0.08	0.06	0.04	0.02
		Moderate	0.23	0.29	0.28	0.21	0.16	0.08
		High	0.42	0.53	0.52	0.39	0.29	0.14
	Female	Background	0.57	0.45	0.52	0.69	0.85	0.92
		Low	0.01	0.02	0.01	0.01	0.00	0.00
		Moderate	0.06	0.08	0.07	0.04	0.02	0.01
		High	0.36	0.46	0.40	0.26	0.13	0.06
WPR-A	Male	Background	0.51	0.29	0.30	0.49	0.78	0.89
		Low	0.16	0.24	0.23	0.17	0.07	0.04
		Moderate	0.30	0.43	0.42	0.31	0.13	0.07
		High	0.03	0.04	0.04	0.03	0.01	0.01
	Female	Background	0.59	0.50	0.52	0.74	0.91	0.95
		Low	0.16	0.20	0.19	0.10	0.04	0.02
		Moderate	0.21	0.26	0.25	0.13	0.05	0.02
		High	0.03	0.04	0.04	0.02	0.01	0.00
WPR-B	Male	Background	0.27	0.11	0.17	0.45	0.74	0.87
		Low	0.13	0.16	0.15	0.10	0.05	0.02
		Moderate	0.25	0.30	0.28	0.19	0.09	0.04
		High	0.35	0.43	0.40	0.27	0.13	0.06
	Female	Background	0.30	0.20	0.39	0.74	0.92	0.96
		Low	0.17	0.20	0.15	0.07	0.02	0.01
		Moderate	0.22	0.25	0.19	0.08	0.03	0.01
		High	0.30	0.35	0.27	0.11	0.04	0.02

compensable work-related diseases in many countries, representing a third or more of all registered occupational diseases in the United States, the Nordic countries and Japan (Bernard 1997; Pope et al. 1991; Vaaranen et al. 1994).

Guo et al. (1995) estimated that 65% of cases of low back pain in the United States are attributable to occupational activities. To date, there have been no other published estimates of the fraction of back pain (specifically) in the total population that is occupationally induced. However, low back pain was identified by the Pan American Health Organization as one of the top three occupational health problems to be targeted by surveillance within the WHO Region of the Americas (Choi et al. 2001).

Among MSDs caused by occupational ergonomic stressors, only low back pain is currently a separate category in the GBD database and could be assessed. For purposes of this chapter, low back pain is defined as all pain in the back that is not secondary to another disease or injury cause (such as cancer or a motor vehicle accident). This includes disk prob-

lems (displacement, rupture), sciatica and other sources of back pain. Cervical spine problems, such as neck pain or neck torsion problems, are excluded. This category of conditions is considered equivalent to what others have termed non-traumatic MSDs affecting the lower back.

In the epidemiological literature, MSDs of the back are often defined on the basis of pain reported on interview, usually with standardized study criteria referring to time of onset, frequency and/or severity of pain. Physical examinations have sometimes been used to supplement questionnaires, particularly to help localize the symptoms reported on interview and to rule out other causes of those symptoms. However, an important proportion of epidemiologically relevant (exposure-related) back disorders are negative on physical examination (e.g. Punnett et al. 1991) as well as on X-ray (e.g. Riihimäki et al. 1990). Most cases of low back pain cannot be diagnosed by objective criteria and are typically designated idiopathic or non-specific (Frank et al. 1995; Riihimäki 1991, 1995b), even if there are findings on examination or severe symptoms and loss of function.

It is difficult to measure directly the validity of questionnaire responses, since no consensus exists regarding a single "gold standard" against which all other measurements could be compared. The sensitivity and reliability of physical examination manoeuvres for identifying low back pain range from good to poor; not all pain results from known mechanisms for which there is a corresponding objective test (Deyo et al. 1992; Viikari-Juntura and Riihimäki 1999). Deyo et al. (1992) suggested that as many as 85% of cases of low back pain cannot be diagnosed because of the poor performance of examination and imaging tests.

A recent review by NIOSH (Bernard 1997) (see also below) emphasized that health outcomes defined subjectively should be included in any consideration of work-related back disorders. In 24 of the 42 epidemiological studies on low back pain reviewed, the health outcome was defined by reported symptoms on questionnaires or interview, ranging from any back pain to specific symptoms such as those consistent with sciatica. In several studies, MSD cases defined by symptoms alone and those defined by findings on physical examination have shown very similar associations with the ergonomic characteristics of subjects' jobs. Symptom-based case definitions generally appear to be both unbiased and more sensitive than those that require documented abnormalities on physical examination (e.g. Bernard et al. 1993; Punnett 1998; Punnett et al. 1991; Silverstein et al. 1986, 1987).

Other case definitions sometimes used epidemiologically include low back impairment or disability, typically indicated by reduced ability to perform activities of daily living or occupational tasks, work absenteeism and the seeking of medical care for back pain. Such behavioural measures, however, are less desirable than low back pain *per se*, because they are more distal from the direct morbidity and more likely to be affected by interpersonal variability (e.g. tolerance of pain before seeking medical

attention) or by differences in job demands (e.g. pain of the same severity may cause more low back disability in persons whose jobs are more demanding).

At the same time, there is a strong correlation between the frequency of musculoskeletal symptoms by occupation and the frequency of workers' compensation claims and recorded work-related repetitive trauma disorders in those same occupations (Fine et al. 1986; Silverstein et al. 1997). Outcomes such as days of restricted activity, long-term disability, health care utilization and use of medication are very common among people with back pain, indicating the public health importance and cost of these disorders even when self-reported pain is not confirmed objectively (Badley et al. 1994, 1995; Guo et al. 1999; Miedema et al. 1998; Punnett 1999; Westgaard and Jansen 1992).

Back pain has been defined operationally in various ways in epidemiological studies, including both prevalent and incident conditions. Variations in the definition are related to the recall period (e.g. pain now, or within the last week or the past year), the frequency or duration (e.g. at least three times in the past year, or lasting at least one week) and the severity (e.g. at least a "4" on a 7-point pain intensity scale), among others. Even among studies that use similar definitions, prevalence estimates can vary substantially (Loney and Stratford 1999). However, for the purpose of evaluating the exposure–response relationship, as long as comparisons are made within a study population that has been evaluated with a consistent case definition, estimates of relative risk do not appear to be greatly affected (Ozguler et al. 2000).

Evidence of causality

The evidence on low MSDs, including back pain, in relation to workplace factors has been thoroughly reviewed by NIOSH (Bernard 1997). The National Research Council, with the Institute of Medicine, has also published a comprehensive review of the evidence on MSDs in the workplace (National Research Council 2001). Strong or sufficient evidence was found for a number of risk factors at the workplace to be associated with back pain (Table 21.49). The National Research Council report (2001) summarized ranges of risk estimates for specific occupational stressors (Table 21.50).

In addition to these two comprehensive reviews from the United States, numerous other authors from Europe, Asia and Canada reviewed the same epidemiological literature or variously defined subsets, and most reached similar conclusions (e.g. Burdorf and Sorock 1997; Frank et al. 1996; Garg 1992; Hagberg et al. 1995; Hales and Bernard 1996; Hoogendoorn et al. 1999; Hulshof et al. 1987; Jensen 1988; Jin et al. 2000; Johanning et al. 1991; Lagerström et al. 1998; Nachemson and Jonsson 2000; Riihimäki 1991, 1995a; Viikari-Juntura 1997; Wikstrom et al. 1994). For example, in a systematic literature review that focused on 28 cohort and three case–control studies of highest methodological

Table 21.49 Rating of evidence for causal relationships between specific occupational stressors and back disorders according to the NIOSH review

Strength of evidence	*Specific stressor*
Strong evidence[a]	Lifting and forceful movements Whole-body vibration
Evidence[b]	Awkward postures Heavy physical work
Insufficient evidence[c]	Static work postures
Evidence of no effect[d]	Other stressors

[a] *Strong evidence.* A causal relationship is shown to be very likely between intense or long-duration exposure to the specific risk factor(s) and MSD of the back when the epidemiological criteria of causality are used. A positive relationship has been observed between exposure to the specific risk factor and MSD of the back where chance, bias and confounding factors could be ruled out with reasonable confidence in at least several studies.

[b] *Evidence.* Some convincing epidemiological evidence shows a causal relationship when the epidemiological criteria for causality for intense or long-duration exposure to the specific risk factor(s) and MSD of the back are used. A positive relationship has been observed between exposure to the specific risk factor and MSD of the back in studies in which chance, bias and confounding factors are not the likely explanation.

[c] *Insufficient evidence.* The available studies are of insufficient number, quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association.

[d] *Evidence of no effect.* Adequate studies consistently show that the specific workplace risk factor is not related to development of MSD of the back.

Note: In this review, 42 epidemiological studies were selected on the basis of the following criteria: (i) exposed and reference populations were well defined; (ii) MSDs of the back were measured by well defined, explicit criteria determined before the study; (iii) exposure was evaluated so that some inference could be drawn regarding repetition, force, extreme joint position, static loading or vibration and lifting tasks; (iv) study participation of more than 70%. Thirty studies used a cross-sectional design and five a prospective cohort, four were case–control studies and two were retrospective cohorts. Full descriptions of these studies appear in Table 6-6 of the NIOSH review. Criteria for causality were based on strength of association, consistency, specificity of effect or association, temporality, exposure–response relationship and coherence of evidence.

Source: Bernard (1997).

quality, Hoogendoorn et al. (1999) found strong evidence for manual material handling, bending and twisting and whole-body vibration as risk factors for back pain. They found moderate evidence for patient handling and heavy physical work, and no evidence for standing or walking, sitting, sporting activities and total leisure-time physical activity. Specific psychological stressors, supported by weaker evidence, were reviewed by Burdorf and Sorock (1997), and are shown in Table 21.51.

Some of the results of these reviews on specific stressors were obtained from studies that were conducted within various occupational groups, such as operators, mine workers, dentists, office workers and nurses. The evidence presented above implies that preventive interventions reducing the exposure to these risk factors would decrease the occurrence of back disorders considerably, even within an occupation.

Table 21.50 Summary of epidemiological studies with risk estimates of null and positive associations of work-related risk factors and the occurrence of back disorders

	Risk estimate expressed in relative risk or odds ratio				*Attributable fraction (%)*	
	Null association[a]		*Positive association*			
Work-related risk factor	*N*[b]	*Range of odds ratio*	*N*[b]	*Range of odds ratio*	*N*[b]	*Range*
Manual material handling	4	0.90–1.45	24	1.12–3.54	17	11–66
Frequent bending and twisting	2	1.08–1.30	15	1.29–8.09	8	19–57
Heavy physical load	0	NA	8	1.54–3.71	5	31–58
Static work posture	3	0.80–0.97	3	1.30–3.29	3	14–32
Repetitive movements	2	0.98–1.20	1	1.97	1	41
Whole-body vibration	1	1.10	16	1.26–9.00	11	18–80

NA Not applicable.

[a] Confidence intervals of the risk estimates included the null estimate (1.0). In only 12 of 16 null associations was the magnitude of risk estimate presented.

[b] Number of associations presented in the epidemiological studies.

Source: National Research Council (2001).

Table 21.51 Summary of epidemiological studies on associations between specific occupational psychological risk factors and the occurrence of back disorders

	Risk estimate		*Attributable fraction*	
Specific risk factor	*N*[a]	*Range*	*N*[a]	*Range (%)*
Mental stress	4	1.30–2.08	4	23–44
Job dissatisfaction	5	1.39–2.40	4	21–41
Work pace	1	1.12	NA	NA
Monotonous work	5	1.25–2.34	4	20–44

NA Not applicable.

[a] Number of associations presented in the epidemiological studies.

Source: Burdorf and Sorrock (1997).

Furthermore, there is sufficient evidence on biomechanical risk factors to conclude that many cases of low back pain could be prevented by workplace changes. For example, Marras et al. (2000) showed that lifting equipment and other engineering controls had demonstrable effects on lowering both biomechanical exposure to the lower back (compressive force, torque, etc.) and reported rates of low back disorders in 36 repetitive manual material handling jobs at 16 different companies.

The reviews by the National Research Council (2001), Westgaard and Winkel (1997) and Frank et al. (1996) each cited a number of well designed studies that identified opportunities to prevent risk of low back pain by reducing exposure to biophysical and psychosocial factors. To illustrate the improvements that can be obtained by reducing ergonomic stressors at work, selected interventions and their impact are shown in Table 21.52. Certain interventions have practically completely removed ergonomic stressors from the workplace.

Despite this extensive literature, some still dispute the importance of these factors, especially in relation to nonoccupational causes (e.g. Battie and Bigos 1991; Nachemson 1999; Waddell 1991). There are probably several interrelated reasons for the continuing controversy, many of which have been discussed by others (Frank et al. 1995, 1996; Viikari-Juntura and Riihimäki 1999). The available epidemiological evidence still consists largely of cross-sectional and retrospective case–control investigations. With regard to assessment of morbidity, the use of self-reported symptoms for end-points has also generated discussion, as described above. Sparse longitudinal data leave important gaps in knowledge concerning latency of effect, natural history, prognosis and potential for selection bias in employed populations (e.g. the "healthy worker effect"). Few studies have attempted systematically to describe these features of back disorders in populations with defined levels of exposure to ergonomic stressors. Specific, quantitative comparisons of conclusions based on prevalence and incidence data within the same population are rare, and knowledge is still sparse as to the factors that predict recovery or persistence among workers who continue in their jobs after the onset of a disorder. No study has been identified that compares current and former workers with reference to both prior MSD morbidity and exposure status.

In addition, there are many known or suspected nonoccupational risk factors; some study populations have not provided enough statistical power to address potential confounding and effect modification of exposure–response relationships. Exposure assessment has often been limited, with too few exposures characterized to rule out confounding, or the use of crude exposure indicators leading to potential misclassification and unreliable conclusions. Attempts to partition risk between physical and psychosocial domains have obscured the overlap between the two and the distinction between preventable and nonpreventable risk factors (Bongers et al. 1993; MacDonald et al. 2001; Volinn and Punnett 2001; Volinn et al. 2001).

In the light of these issues, it is important to restate that the issue is not whether all back disorders are caused by work; there is a clear consensus that this is not the case. Nevertheless, most investigators and reviewers have concluded, equally clearly, that a large proportion of back disorders *among persons with high exposure to ergonomic stressors at work* could be prevented by reducing those exposures.

Table 21.52 Selected intervention studies on occupational ergonomic stressors and reduction of occurrence of low back pain

Job title/activity	*Intervention: engineering/administrative control*	*Outcome*	*Study*[a]
Nursing assistants (in two nursing home units)	Introduction of walking belts, mechanical hoists and shower chairs	Back injury incidence reduced from 83 to 42 per 100 full-time equivalents	Garg and Owen (1994) [Ex. 26-1415]
Poultry processing employees	Introduction of workstation analysis and redesign, including altering heights of products, providing workstands, and installing tank tilters to reduce manual handling	Back injury rates declined from 4.4 to 3.0 per 200000 hours	Stuart-Buttle (1994) [Ex. 26-1045]
Office furniture manufacturing assembly workers	Scissor lifts installed to aid in packaging file cabinets of different sizes; small-assembly workstations altered to eliminate twisting and bending during lifting	Back injuries cut by 50%	LaBar (1991) [Ex. 26-1078]
Metal castings, unpacking operation	Crates modified by adding gates at each end and installing scissors lift to lift crates; changes made in the way castings were stacked in the crates to permit workers' arms to remain close to the body while unpacking	Back injuries associated with this operation eliminated	Oxenburgh (1994) [Ex. 26-1041]
Palletizing operation	Scissors lift table with turntable tops installed alongside each packing station	Five out of six back injuries eliminated	Benson (1987) [Ex. 26-1062]
Lamp manufacturing	Addition of vacuum hoist; reduced equipment height; reduced box size and weight; introduction of back awareness programme for employees	Back and upper extremity disorders eliminated in the last four years	Carreau and Bessett (1991) [Ex. 26-1071]
Unpacking car parts	Plywood sheets modified to reduce weight and permit them to slide more easily in the grooves	Back injuries associated with this operation eliminated	Oxenburgh (1991) [Ex. 26-1041]

Manual handling of bulk paper	Manual lifting eliminated by installation of scissors lift; trolley runners replaced by roller bearings that enable the paper to be loaded onto the scissors lift without manual lifting	No back injuries in the three years since modifications made	Oxenburgh (1991) [Ex. 26-1041]
Railway repairmen	Storage of tools and materials off the ground between knee and shoulder height; winches to lift and handle heavy equipment; redesigned work tables, trolleys and carts to more easily handle train car parts	Low back injuries and lost work days eliminated	McMahan (1991) [Ex. 26-1083]
Nursing, hospital	Changes in procedures for moving patients, manoeuvring carts and equipment, using gall bladder boards, walking on wet floors, accessing power outlets	Back injury rates reduced by 25% in 18 months since programme implemented	Garg and Dockery (1995) [Ex. 26-1095]
Video display terminal operators	Reduction of keying to no more than five hours per day and evaluation of new chairs; new chairs installed in February 1991	Low back pain reduced from 8.3% to zero between November 1990 and February 1993	[Ex. 500-41-115]
Forestry workers	Forest tractor seats: spring tension and inclination adjustment; accessory lumbar support provided	Reduction of low back pain among forest tractor drivers: 24 of 50 drivers found the lumbar support beneficial	Perkio-Makela and Riihimäki (1997)

[a] All exhibit numbers refer to materials in USDOL OSHA (2000).

Source: Lahiri et al. (2002).

Other outcomes

Although the present analysis was limited to low back pain, the evidence on MSDs caused by occupational ergonomic stressors is broader. MSDs affecting the neck and the upper and lower limbs result from the same risk factors as are implicated in low back pain. For example, in a study of over 10 000 manufacturing employees, the effect of "greater physical demands" of the job on low-back musculoskeletal injuries (relative risk of 1.6, 95% CI 1.2–2.1) was only slightly higher than that for all other musculoskeletal injuries combined (relative risk of 1.4, 95% CI 1.1–1.7) (Tsai et al. 1992). De Zwart et al. (1997), studying over 7300 men in the Netherlands, found higher prevalences of shoulder disorders among employees in heavy physical work (e.g. heavy lifting and frequent stooping) and steeper increases over four years than among employees in less physically demanding jobs. The magnitude of these effects was very similar to those for low back injuries. The work-relatedness of upper and lower extremity MSDs has been discussed extensively, again by European as well as North American reviewers (e.g. Armstrong et al. 1993; Bernard 1997; Bongers et al. 1993; Buckle and Devereaux 1999; Hagberg et al. 1995; National Research Council 2001; Sluiter et al. 2000).

Also excluded here are other types of health effects related to ergonomic stressors, such as acute workplace injuries, cardiovascular disease, mental health and adverse reproductive effects (Punnett 2002). Thus, the total impact of excessively strenuous work activities on morbidity and related quality of life is greater than that estimated in this risk assessment.

Hazard estimates

Data sources

For the purposes of this analysis, studies were sought that compared the risk of low back pain among broad occupational groups (defined similarly to the economic subsectors explained above) and comprehensively enough to cover the range of paid occupations. Smaller, more specific studies limited to relatively narrow occupational groups (e.g. nurses, dock workers, drivers) were checked for consistency with the larger data sets. Studies where the reference groups were also engaged in substantial physical activity (e.g. house painters) were excluded. The most recent literature (1997–2001) was searched for exposure data and exposure–risk relationships, and published statistics of national occupational health and safety institutes were consulted.

In addition to this systematic search, a number of reviews and studies were identified to provide evidence supporting the selected approach. Search strategies were described in the Introduction. Medline was searched for articles more recent than 1985, using any of the keywords back pain, back disorder, back or musculoskeletal combined with any of

the following: occupation, occupational, workplace, work, workers, risk factors, risk.

Description of literature

The studies specifically referred to in this section are summarized in Table 21.53.

The report of the National Research Council (2001) stated that the *occupations* with the highest risk among men were construction labourers, carpenters and industrial truck and tractor equipment operators, while among women they were nursing aides/orderlies/attendants, licensed practical nurses, maids and janitor/cleaners. Other high-risk occupations were hairdressers and automobile mechanics, often employed in small businesses or self-employed. No rates were listed against occupations in the report. The report stated that the highest-risk *industries* for men were lumber and building material retailing, crude petroleum and natural gas extraction and sawmills/millwork. Among women, the highest-risk industries were nursing and personal care facilities, beauty shops and motor vehicle equipment and manufacturing. No rates were listed for industries in the report.

Leigh and Sheetz (1989) measured low back pain on the basis of a national survey and a self-reported statement regarding "trouble with back or pain during the last year". They estimated relative risks by comparing the outcome frequency among occupational groups, using managers as a reference group (Table 21.54). This chapter places great weight on this study, because it was relatively large (1404 participants) compared to many others, it covered a comprehensive sample of occupations, and the results were adjusted for potential confounding variables. One important limitation, however, is that the multivariate analyses simultaneously included two ratings of physical exposure, socioeconomic status and occupational title. Since physical exposure is hypothesized to be the primary pathway through which occupational differences are manifested, these analyses would certainly lead to an underestimation of the effect of occupation on MSDs.

Although operators and service workers have very similar relative risks, it is common that intervention strategies differ among these occupational settings. For that reason, the relative risks and exposure assessments for those two occupational groups are presented separately throughout this analysis.

Within the limits of the available literature, the results of the Leigh and Sheetz analyses appear to be generally consistent with other reported relative risk values (Table 21.55). Since many other studies used office workers or other sedentary occupations as the reference group, it is appropriate to adjust the Leigh and Sheetz findings for comparative purposes. This can be done by dividing the relative risks for categories 3, 4, and 5 by 1.38 (the relative risk for clerical or sales work), in order to estimate a relative risk with clerical jobs as the reference group. The new

Table 21.53 Key studies and reviews on work-related back pain

Study population (source) or literature reviewed	*Population size*	*Outcome measured/reported*	*Magnitude or relative risk*	*Comments*	*Reference*
Reviews					
Back pain in relation to heavy physical work, lifting and forceful movements, non-neutral postures, and/or whole-body vibration: Belgium, Finland, Italy, Japan, Netherlands, Russian Federation, Sweden, USA	42 studies, evaluated according to 4 criteria for methodological quality	Back pain (multiple definitions among the studies)	Heavy physical work: [a] 1.0–12.1 Lifting and forceful movements: [a] 0.9–10.7 Non-neutral postures: 1.2–10.7 Static work postures: 0.8–24.6 Whole-body vibration: 1.0–39.5 Conclusions: "strong evidence" for causal relationship with lifting and whole-body vibration; "evidence" for causal relationship with awkward posture and heavy physical work	Literature review: etiological studies	Bernard (1997)
Important risk factors for work-related back disorders, and strength of the association between the consistent risk factors and back disorder were identified	35 epidemiological studies published from 1980 to 1996	Back disorders (multiple definitions among the studies)	Manual materials handling: 1.12–3.07 Frequent bending and twisting: 1.29–8.09 Heavy physical load: 1.54–3.71 Static work movement: 1.30–3.29 Repetitive movement: 1.97 Whole-body vibration: 1.47–9.00 Mental stress: 1.30–2.08 Job satisfaction: 1.39–2.40 Pace of work: 1.21 Job decision latitude or monotonous work: 1.25–2.34 Conclusion: lifting or carrying loads, whole-body vibration and frequent bending and twisting consistently associated with work-related back disorder. Job dissatisfaction and low job decision latitude also important	Illustration on individual risk factors, such as age, smoking and education	Burdorf and Sorock (1997)

Intensive and systematic review on physical load during work or leisure time as risk factors for back pain. Literature sources: Medline (1966–1997), Embase (1988–1997), Psyclit (1974–1997), NIOSHTIC, CISDOC and HSELINE (1977–1997) and Sportdiscus (1949–1997)	31 publications in Dutch, English, French and German	Inclusion criteria (health outcome): back pain based on symptoms or signs of non-specific back pain, self-reported or measured (via sick leave, medical consultation, treatment and disability due to back pain, etc.)	Strong evidence for manual material handling, bending and twisting, and whole-body vibration as risk factors. Moderate evidence for patient handling and heavy physical work. No evidence for standing or walking, sitting, sports and total leisure-time physical activity	Cross-sectional studies were excluded	Hoogendoorn et al. (1999)
Review of studies of work-related low back pain in China among literature issued from 1983 to 1997	16 epidemiological studies selected; quality inclusion/ exclusion criteria were applied; all studies included were cross-sectional	Occurrence of work-related low back pain (multiple definitions among the studies)	*Prevalence ratios:* Bending and twisting: 2.0–8.5 Static posture: 1.5–14.3 Whole-body vibration: 1.9–5.5	Suggestion of potentially greater exposure in China's work environment and potential systematic bias	Jin et al. (2000)
		Back pain (multiple definitions among the studies), muscle activity, tissue load and tissue damage	"linkages … between external loads and biomechanical loading of the spine, biomechanical loading and internal tolerances of the spine, and internal tolerances and outcomes (from pain through disability) are well established…The literature relating to causal factors in work-related low back disorders is coherent and provides ample evidence on how adverse work situations can lead to them." (pp. 357–358)	Literature review: etiological, experimental and intervention studies	National Research Council (2001)
Systematic literature review of articles published in English, 1980–1995, 25 most populous countries	NA	Back pain (multiple definitions among the studies); point prevalence of low back pain/ annual or lifetime incidence	2–4 times higher rates of back pain in high-income than in low-income countries. Within low-income countries, rates are higher among urban than among rural populations	Comparison of low-income and high-income countries	Volinn (1997)

continued

Table 21.53 Key studies and reviews on work-related back pain (*continued*)

Study population (source) or literature reviewed	*Population size*	*Outcome measured/reported*	*Magnitude or relative risk*	*Comments*	*Reference*
Original studies					
Current full-time employees at one site of large chemical manufacturing company, USA, 1987–1989	5 903 employees	Back or joint pain; back pain >30 days; visit to physician for back pain	35.4% reported back or joint pain during the past year, 5.3% back pain lasting >30 days; managers and technicians had highest prevalences of back pain >30 days	Adjusted for age, sex and ethnicity	Burchfiel et al. (1992)
Random sample of retired workers living in the Paris area, members of a supplementary interprofessional retirement pension fund	993 people in total (626 in first survey in 1982–1983, 464 in second survey in 1987–1988)	Osteoarticular disorders: presence of pain with/ without restricted joint movement for at least 6 months before interview	Increased frequency of osteoarticular pain during the 5 years between the two interviews; from 52% to 65% in men and 72% to 82% in women A significance increase in frequency of osteoarticular pain for those exposed to heavy weights and awkward postures (from 68% to 77% and 56% to 76%, respectively)	Significantly higher frequencies of osteoarticular pain for women than men in both interviews	Derriennic et al. (1993)
Probability sample of total working population in USA, 1988	30 074 workers	Back pain every day for a week or more during 12 months before interview	National estimate of back pain: 22.4 million cases and a prevalence of 17.6% in 1988 65% of cases were attributable to occupational activities The risk was highest for construction labourers among males (prevalence 22.6%) and nursing aides among females (18.8%)		Guo et al. (1995, 1999)
Random sample: all small and medium-sized factories listed with Labour Department in Delhi, India	60 of 6 076 factories; 631 workers selected randomly from 60 factories	Lumbar pain diagnosed by medical practitioner	Buffing workers, operators and assembly workers had highest pain prevalences (30.4%, 28.7% and 27.2%, respectively)		Joshi et al. (2001)

Workers' compensation claims for back injuries in 26 states (among 1 705 674 workers' compensation claims), all workers employed in 26 states, USA, 1979	329 474 claims for back injuries	Compensation claims filed for back injury or strains/sprains	Back injury constitutes 19.3% of compensation claims in 26 states Construction and mining industries have largest incidence: 1.6 and 1.5 claims/100 workers, respectively Occupations with the largest incidence compared to total workers: miscellaneous labourers 12.3 claims/100 workers, garbage collectors 11.1 claims/100 workers	Percentage of workers employed in 26 states in 1979, obtained from 1970 census	Klein et al. (1984)
Diagnosed low back pain cases, among all employed persons in Japan, 1986 and 1988	13 166 low back pain cases	Accidental low back pain caused by the action of a sudden force; required absence of ≥4 days	Transport occupations: 8 cases per 10 000 workers Construction: 4 cases per 10 000 workers Sales/service: 0.5 cases per 10 000 workers Mining and cargo handling: 13.9 and 12.3 cases per 10 000 workers	National survey data	Kuwashima et al. (1997)
Probability sample of United States workers employed ≥20 hours a week, 1972–1973	959 working males and 455 working females	Low back pain: experience of trouble with back or spine in past year	1-year low back pain past prevalence: 19.4% males, 20.7% females Relative risk: farmers 5.17, operators 2.39, service 2.67, clerical 1.38 compared to managers and professionals Relative risk for high job demands: 1.68	Health outcome did not distinguish between upper and lower back pain Over-adjustment by including socioeconomic status, occupation and physical risk factors in same multivariate analyses	Leigh and Sheetz (1989)
Three random samples of the Finnish population, 20–64 years of age, 1988–1990	7 544 people	Any back pain in past 30 days/back pain in past 12 months	Odds ratios of back pain: 2.1 in farmers, 1.8 in manual workers, 1.7 in entrepreneurs, 1.4 in lower white-collar workers	Results adjusted for age, height, marital status, smoking, body mass index, physical activity, mental distress	Leino-Arjas et al. (1998)
Workers at 8 aluminium plants in Norway, 1998	5 654 people	One-year prevalence of musculoskeletal symptoms by Standardized Nordic Questionnaire	Odds ratio for low back symptoms among operator vs office workers: 1.8 (range 1.5–2.1)	Adjusted for sex, weight, height, smoking, and nonoccupational physical activity	Morken et al. (2000)

continued

Table 21.53 Key studies and reviews on work-related back pain (*continued*)

Study population (source) or literature reviewed	*Population size*	*Outcome measured/reported*	*Magnitude or relative risk*	*Comments*	*Reference*
Active workers from 4 occupational sectors (office, hospital, warehouse, airport registration), France, 1991	725 people	Six definitions of low back pain (pain or discomfort in a lumbar area in the previous 6 months/pain at least one day/pain >30 days/intensity of pain above 3/physician visit/ treatment for low back pain)	Prevalence of low back pain varied from 8% to 45% according to the case definition Carrying heavy loads and bending postures showed consistently high odds ratios (1.88–2.14) for most low back pain definitions	Over-adjustment by including both occupation and physical risk factors in multivariate analyses; also adjusted for sex, age and body mass index	Ozguler et al. (2000)
All back disorders reported to clinic in one automobile assembly plant (referents from same production departments)	95 assembly workers with back disorders (cases) and 124 without (referents)	Back cases: workers who sought medical attention for "new" back disorders during a 10-month period	Exposed workers: 84% Bending and twisting (100% vs 0%): odds ratio 8.09 (range 1.5–44.0) Lifting (>44.5 Newtons/min): odds ratio 2.2 (range 1.0–4.7)	Adjusted for age, sex and sports activity	Punnett et al. (1991)
Employed persons in Washington State, USA, 1990–1998	Approximately 1.23 million full-time equivalent workers per year	Workers' compensation claims for nontraumatic soft tissue disorders of the back	Rates by industry sector ranged from 43.5 to 280.0 per 10 000 full-time equivalents	Surveillance data	Silverstein et al. (2002)
Population-based survey of approx 9.9 million adults (15 years or older), Belgium, 1991	3 829 people	Reported symptoms: low back pain, history of low back pain, first low back pain and daily low back pain	Current low back pain: 33% of population Work dissatisfaction associated with low back pain history (odds ratio >2.4)	Adjusted for age, sex, language, residence, social class and job satisfaction	Skovron et al. (1994)
Full-time regular workers in Shell Oil Company's manufacturing facilities between 1987 and 1989, USA	10 350 people	Low-back injury (ICD-9 CM, 722, 724)	Physically demanding jobs have relative risk of 1.57 for low back injury and 1.35 for non-low back musculoskeletal injury Smoking and overweight showed high relative risks (1.54 and 1.42, respectively)	Job title used to identify potential for increased physical demand at work	Tsai et al. (1992)

NA Not applicable.

[a] Range of effects for studies that met at least one criterion for epidemiological quality.

Table 21.54 Relative risks of low back pain for occupational groups, with managers and professionals as the reference group

Occupational activity	*Relative risk (95% CI)*
Managers and professionals	1.0 (NA)
Clerical or sales worker	1.38 (0.85–2.25)
Operators	2.39 (1.09–5.25)
Service workers	2.67 (1.26–5.69)
Farmers	5.17 (1.57–17.0)

NA Not applicable.

Source: Based on data from Leigh and Sheetz (1989).

values would be 1.73, 1.93 and 3.75, respectively. Keeping in mind that these estimates represent the average values for the entire occupational category, it can be seen that the other studies cited fall within the CIs, with very few exceptions, and in fact generally have similar point estimates (Table 21.55).

The only study that can directly and numerically be compared to that of Leigh and Sheetz (1989) is that by Leino-Arjas et al. (1998). However, the only value corresponding directly to one of the categories of Leigh and Sheetz is that for farmers. The relative risk is lower (2.13) than the one put forward by Leigh and Sheetz (5.17), which may reflect better working conditions for farmers in Finland. For this analysis we therefore used the average of these two results (see below).

Also available are administrative statistics from several countries on the number of cases of back conditions. These are generally compiled from employers' surveys or compensation statistics and typically report lower rates than those assessed by population surveys. Rates for certain occupations, as compared to managers and professionals, can be estimated on the basis of these statistics. Table 21.56 summarizes administrative workplace statistics on conditions involving the back, based on reports by employers in the United States of work-related injuries (Bureau of Labor Statistics 2001) and compensation statistics of the Australian workforce (National Occupational Health and Safety Commission 2001) and of the German national workforce (Bundesverband der Betriebskrankenkassen 2001).

All three of these data sets show higher risks for occupations other than managers and professionals, although the point estimates vary somewhat. None of these frequency estimates is adjusted for potential confounding variables. The incidents assessed in the first two data sets are limited to cases that have been recognized as work-related cases and involve behaviour such as absence from work or filing a claim against the employer. In contrast, the German study sought to assess the health status of the population more comprehensively and these data are

Table 21.55 Relative risks of occupational groups by exposure level

	Source							
Occupation (exposure category)	*Leigh and Sheetz (1989)*[a]	*Astrand (1987)*[b]	*Bongers et al. (1990)*[b]	*Bovenzi and Betta (1994)*[b]	*Burdorf et al. (1993)*[b]	*Hildebrandt (1995)*[b]	*Johanning et al. (1991)*[b]	*Magnusson et al. (1996)*[b]
Managers and professionals	1.00/NA							
Professionals								
Managers								
Teachers								
Clerical or sales workers	1.38/1.00							
Office workers (sedentary)				1.00	1.00	1.00	1.00	1.00
Clerks		1.00						
Air force officers			1.00					
Civil servants								
Sales								
Operators	2.39/1.73						3.90	
Construction labourers								
Manual workers		2.28						
Pilots and aircrew			9.00					
Drivers (bus, truck, tractor)				1.83–5.49	2.51	1.32		1.55–2.10
Crane operators					3.29			
Dock workers								
Plumbers						1.32		
Carpenters								
Technicians								
Assembly, packing, food processing								
Automobile mechanics								
Maintenance								
Service workers	2.67/1.93							
Airport registration workers								
Hospital workers								
Warehouse workers								
Stock handlers, baggers								
Janitors, cleaners								
Waitresses								
Nurses								
Farmers	5.17/3.75							

NA Not applicable.

[a] Relative risks used in estimation of global burden of disease. The second set of relative risk values was estimated using clerical/sales jobs as the reference group, for the purpose of comparison with other studies.

Source									
Partridge and Duthie (1968)[b]	*Riihimäki et al. (1989)*[b]	*Riihimäki et al. (1994)*[b]	*Videman et al. (1990)*[b]	*Burchfiel et al. (1992)*	*Ozguler et al. (2000)*	*Joshi et al. (2001)*	*Guo et al. (1995)*[c] *[female]*	*Morken et al. (2000)*	*Leino-Arjas et al. (1998) (male)*
									1.00
				1.00					
				1.80					
							[1.2]		
	1.00	1.00	1.00	0.89	1.00	1.00		1.00	1.35
1.00									
				1.10					
	1.0–1.5	1.40		1.10		1.83		1.80	
							2.10		
			3.60	1.49					1.84
			2.90				2.00		
1.27									
							1.70		
		1.50					2.10		
				1.20		1.59			
						1.73			
							1.80		
						1.59	1.70		
				1.03					
					0.86				
					1.13				
					0.54				
							1.70		
							[2.0]		
							[1.6]		
							[1.5]		
							1.80		2.13

[b] Cited in Bernard (1997).

[c] Compared to all male or female workers.

Table 21.56 Relative risks of occupational conditions involving the back by occupational title, compared to managers and professionals, from national surveillance data

	Relative risk for back conditions		
Occupational activity[a]	*USA*[b]	*Australia*[c]	*Germany*[d]
Managers and professionals	1.0	1.0	1.0
Tradespeople	—	5.5	—
Clerks	—	1.1	1.5
Technical, sales and administrative support	2.2	—	—
Sales and service workers	—	2.2	2.9
Service workers	7.4	—	—
Operators	9.1	—	2.4
Farmers, fishermen and forestry workers	4.3	—	3.6
Operators and farmers	—	8.8	—

— No data.

[a] Owing to the different reporting schemes, some rows (occupational activities) represent the sum of several other rows.

[b] Bureau of Labor Statistics (2001), nonfatal occupational injuries and illnesses involving days away from work, for injuries involving the back.

[c] National Occupational Health and Safety Commission (2001), conditions affecting the upper and lower back.

[d] Bundesverband der Betriebskrankenkassen (2001), musculoskeletal illnesses of the lower back.

Table 21.57 Exposure categories and relative risks of low back pain for occupational groups selected for this analysis, with managers and professionals as the reference group

Exposure category	*Relative risk*	*95% CI*	*Occupational activity*
Background	1.0	NA	Managers and professionals
Low	1.38	0.85–2.25	Clerical and sales workers
Moderate	2.53	1.09–5.69	Operators and service workers
High	3.65	1.57–17.0	Farmers

NA Not applicable.

Source: Exposure level adapted from Leigh and Sheetz (1989).

therefore likely to be more comparable to those reported by Leigh and Sheetz. The values are, in fact, relatively close except for agricultural workers.

Given that the study by Leigh and Sheetz (1989) best fits the format required for this analysis, and the supporting evidence displaying very similar quantitative values, the proposed exposure categories and attributed relative risks are displayed in Table 21.57. The value for farmers is provided by an average of the relative risks for farmers in the Leigh and

Sheetz (5.17) and Leino-Arjas et al. (2.13) studies, resulting in a relative risk of 3.65. The CI, however, remains the same, because the CI from the Leigh and Sheetz study (1.57–17.0) includes the CI provided by Leino-Arjas et al. (1.6–2.9) and is wider, which is probably a truer representation of the statistical uncertainty of this estimate.

Methodological quality of the literature

Many of the reviews cited above used systematic criteria to evaluate the potential for selection bias, information bias and confounding in the individual investigations. Several of them identified the methodologically stronger studies and relied primarily or exclusively on those to draw conclusions about the strength of the evidence.

Potential confounding by nonoccupational factors such as sex, age, anthropometry, smoking, heredity and general medical history was extensively investigated in the great majority of studies cited above. All of the studies on which NIOSH relied most heavily, as being rigorous and methodologically sound, controlled for multiple potential confounding variables, permitting the conclusion that physical job factors cause low back pain independently of other factors. Burdorf and Sorock's (1997) review also summarized the associations between low back pain and specific occupational exposures, and relied more heavily on data with adjustment for important covariates. For example, Smedley et al. (1995) adjusted for age, height and nonmusculoskeletal symptoms (the only nonoccupational factors associated with low back pain—see below) in their analysis of low back pain and patient handling demands among female nurses. Tsai et al. (1992) examined the effect of greater vs less physical demands in the job, adjusting for six nonoccupational covariates.

Leigh and Sheetz (1989) adjusted for sex, race, education, height and smoking. In addition, they included terms for occupation *and* for physical effort and repetitive work; this means that the effect of occupation is likely to be underestimated, since the primary intermediate variable (physical effort) was also included. There is also a great deal of discussion in the epidemiological literature about the mechanisms of the effect of socioeconomic status (see below). It could easily be argued that the inclusion of terms for education also results in overadjustment, since a lower level of education is strongly associated with employment in "unskilled" jobs with higher physical exposures and is likely to act at least in part through such limited job opportunities.

Ozguler et al. (2000) analysed multiple low back pain case definitions. The same set of covariates was examined for each one, and all those nonoccupational factors (sex, age, obesity, psychosomatic "well-being") that were associated with low back pain were kept in the model. Like that of Leigh and Sheetz, this study overadjusted the estimates for occupation, because exposure variables such as carrying heavy loads and bending posture were entered in addition to the occupation indicators.

Many investigators have treated socioeconomic status and sex as potential confounding variables that require adjustment in statistical analysis of MSD etiology. However, to the extent that these factors act through or are surrogates for working conditions, both physical and psychosocial, such analyses may in fact serve to obscure the role of those exposures. Both the incidence and the severity of low back pain show an inverse gradient with socioeconomic status (blue collar vs white collar jobs, income, education level) in both men and women (Bergenudd and Nilsson 1988; Broersen et al. 1996; Heistaro et al. 1998).

It seems highly plausible that a large part of the gradient of socioeconomic status in MSDs is due to differences in the work performed, since jobs with lower socioeconomic status consistently involve more physically strenuous and repetitive work (Behrens et al. 1994; Hollman et al. 1999). In a large study of metal working employees, psychosocial conditions at work and physical load were generally correlated with each other and were worse for blue-collar than for white-collar employees, as well as for women compared with men (Leino and Hänninen 1995). In each of these subgroups, adverse working conditions predicted the development or worsening of MSDs over a 10-year follow-up period. The effect of social class was not explained by "lifestyle" factors such as smoking, leisure-time physical activity, body mass index, alcohol consumption or marital status (Leino-Arjas et al. 1998).

Sex differences

Sex is also often described as a "risk factor" for MSDs. In the great majority of studies relied on here, either the population was restricted to one sex or relative risks were adjusted for sex. However, in most studies of low back pain the prevalence was the same or only slightly higher in men than in women (e.g. Behrens et al. 1994; Guo et al. 1995; Morken et al. 2000; Tsai et al. 1992). Skovron et al. (1994) found a higher prevalence among men than among women aged 20–49 years, whereas from 50 years of age the prevalence in women gradually increased relative to that in men. Thus sex is not a strong risk factor for low back pain in any case, and confounding of these effect estimates is not of concern.

Women and men typically experience qualitatively and quantitatively different working conditions (Punnett and Herbert 2000). Women are overrepresented in "light", monotonous jobs that require precise, repetitive hand motions with less latitude for decision-making. Men are more often found in jobs with heavy whole-body workload, such as manual materials handling. In general, once job assignments and the consequent occupational exposures are taken into account, sex differences become negligible (Punnett and Herbert 2000).

Table 21.58 Prevalence and attributable risk of joint pain at 6 and 11 years after retirement, among workers with prior exposure to "heavy physical work"

	Interview 1 (after 6 years)	*Interview 2 (after 11 years)*
Men		
Exposed	58%	75%
Unexposed	49%	57%
Attributable risk	*15.5%*	*24.0%*
Women		
Exposed	87%	91%
Unexposed	68%	79%
Attributable risk	*21.8%*	*13.2%*

Source: Derriennic et al. (1993).

Estimates of risk reversibility

Although no explicit studies have been carried out on low back pain attributed to occupational factors before retirement, it has been assumed that leaving the job would reduce the risk of back pain. The burden of work-related back pain would diminish gradually once the theoretical minimum exposure was reached. Since the theoretical minimum is zero, no new cases would arise. However, morbidity from past exposure might persist or worsen after retirement (Derriennic et al. 1993; Holte et al. 2000; Sobti et al. 1997).

Derriennic et al. (1993) defined a closed cohort of retirees from mixed occupations in France, with an average elapsed period of six years from retirement (at age 63) to the baseline survey. A follow-up survey was conducted after five years. Joint pain was reported by 29% of men and 42% of women at baseline. At 11 years post-retirement, the attributable risk was even higher among men, although it decreased in women because of the high prevalence of joint pain among unexposed persons (Table 21.58).

In summary, there are few or no epidemiological data on whether or not new back disorders develop after leaving work that can be attributed to ergonomic stressors in previously high-exposed (vs low-exposed) workers. Thus, we have assumed the work-related incidence to be zero after retirement from paid employment. However, we do have the high impact of interventions on exposed workers, which supports a reversibility of 100%. This means that the incidence of low back pain is zero for chronic and acute cases of low back pain after exposure ceases. However, chronic low back pain will continue (i.e. the incidence will be zero, but those who have already developed it will continue to experience it).

Extrapolation of risk factor–disease relationships from one subregion to another

Because occupational group was used as an indicator of the average level of combined risk factors for low back pain within each occupation, differences in distribution of risk factors that might exist within occupations or between countries are an important consideration. Risk ratios among occupations vary somewhat from one country to another. This could be due to differences in distributions of risk factors for low back pain, or regional or cultural divergences in symptom reporting. These discrepancies become even more difficult to interpret when the comparisons are made between developing and developed countries. Unfortunately, the data are sparse regarding cross-national differences, both in exposure distributions (within similar types of job) and in reporting of low back pain.

One important element is the extent to which ergonomic interventions have been implemented in the various countries or regions. Although there are insufficient data to quantify the extent of effective ergonomic programmes in each region, it is generally true that occupational health and safety legislation, enforcement and adaptation of engineering controls (ergonomic changes) tend to be more widespread in developed countries, especially in northern Europe followed by North America. If this is correct, then application of occupation-specific relative risks from developed countries (e.g. Leigh and Sheetz 1989) to developing countries would produce conservative estimates.

Similarly, it would be easy to assume that, because of mechanization and other changes in production technology, more developed countries would typically have fewer ergonomic stressors in the same type of work than developing countries, even without intention to reduce ergonomic stressors. For example, Bao et al. (1997) compared shoulder-neck ergonomic exposures in a Chinese and a Swedish assembly line workplace. The Swedish workplace had a better ergonomic workstation design and was better balanced, as well as less sensitive to production irregularities, than the Chinese workplace. The Swedish operators were less exposed to awkward postures during work.

However, in contrast to the general assumption that low back pain rates should be higher in low-income than in high-income countries, a systematic review by Volinn (1997) showed 2–4 times higher rates among Belgian, German and Swedish general populations than among southern Chinese, Philippine, Indonesian and Nigerian farmers. Mentioning that the prevalence of low back pain is higher in the urban populations of low-income countries, and sharply higher in enclosed workshops in low-income countries compared to low-income rural populations, Volinn suggested that low back pain might be associated with urbanization and rapid industrialization, which imply more repetitive movements and loss of control over work pace and scheduling. The author noted that interpretation of the findings requires consideration of

Table 21.59 Comparison of ranges of effect estimates for selected risk factors for low back pain in some working populations of China, India and the Russian Federation

	China, India, Russian Federation		*Developed countries*[a]	
Risk factor	*Studies (n)*	*POR range*	*Studies (n)*	*POR range*
Bending and twisting[b]	4	3.1–16.5	9	1.3–8.1
Static posture[c]	5	2.0–19.9	3	1.3–3.3
Whole-body vibration[a]	4	2.5–14.2	14	1.5–9.0
Heavy manual lifting	2[c]	1.4 ~ 3.5	9	1.5 ~ 3.1

[a] Data taken from National Research Council (2001).

[b] Data taken from Jin et al. (2000) (China).

[c] Summary of data from Ory et al. (1997) (India) and Toroptsova et al. (1995) (Russian Federation).

the methodological quality of population surveys, such as sampling procedure, formulation of questions, procedures for administration of the survey, and nonresponse bias.

Little information on risk of low back pain by occupation is available from developing countries, in particular studies that would pass the quality criteria of the NIOSH or National Research Council reviews. One summary of the literature from China (Jin et al. 2000) reported risk factors for back pain similar to those reported in developed countries. However, in a comparison of the effect estimates for specific risk factors, the authors found slightly higher prevalence odds ratios (POR) in Chinese low back pain studies than other studies (Table 21.59). Alternative explanations would include unmeasured confounding or effect modification. Studies on occupational back pain performed in developing countries do generally report prevalences of back pain within specified occupations, but without comparing them to a reference group (Chiou and Wong 1992; Chiou et al. 1994; Joshi et al. 2001; Kumar et al. 1999; Muruka 1998; Omokhodion et al. 2000; Toroptsova et al. 1995; Yip 2001). Prevalences were generally high for the studied groups, but the lack of comparison to reference groups did not allow conversion into relative risk information, which was necessary for this analysis.

In summary, plausible arguments can be and have been advanced in favour of low back pain rates in specific occupational groups (farmers, factory workers, etc.) being both higher and lower in developing countries compared with developed countries, but the available data permit neither confirmation of this nor quantification of the differences in risk.

6. OCCUPATIONAL RISK FACTORS FOR INJURIES

Workplace injuries are a common hazard for workers. Deaths due to occupational injuries are defined as any potentially avoidable death due

to an external cause resulting from an exposure related to the person's work. The definition excludes death during commuting to or from the workplace. Workers travelling for work purposes are included.

Data in developed countries indicate that differential risks for injury exist by sector, being highest in agriculture and production, less in sales and service, and lowest in professional, administrative and clerical sectors. But similar data are unavailable for developing countries. At the same time, occupational registries provide some indication of injury outcomes—vs risk factor exposure—which can be used to assess the mortality associated with occupational factors. Applying the fatality rates due to occupational injuries per 100 000 insured workers (Table 21.60) to the number of persons in the EAP, as defined earlier in the chapter, gives an indication of total deaths from injuries among workers. The rates reported here for fatal injuries were reported in most countries only for insured populations. Thus, we made the assumption that the same rates applied to all in the EAP, whether or not they were insured, despite some evidence that fatality rates are higher in uninsured populations (Dror 2001; Forastieri 1999; Loewenson 1998). Unfortunately, there is a lack of adequate data on work-related injuries in developing countries to make it possible to generate plausible rates for economic sectors by age, sex and subregion. Mortality outcomes were distributed in the same age pattern as reported in the United States for unintentional injuries.

Because no risk factor exposure is defined in this approach, the counterfactual risk (e.g. theoretical minimum risk level) was defined based on the outcome rather than risk factor exposure. To approximate the safest working conditions observed where all avoidable injury hazards are controlled by effective preventive measures, we chose the injury fatality rate of 0.1 (per 100 000 workers) in the age group 16–17 years and in the occupation category "service" from the National Traumatic Occupational Fatalities surveillance system for the United States for the period 1980–1995 (Marsh and Layne 2001).

6.1 Outcomes considered

The outcomes considered were unintentional injuries, which include motor vehicle accidents, poisonings, falls, fires, drownings and the category "other unintentional injuries". Other unintentional injuries comprise exposure to inanimate mechanical forces, exposure to mechanical forces, other accidental threats to breathing, exposure to electric current, radiation and extreme ambient air temperature and pressure, contact with venomous animals and plants, exposure to forces of nature and accidental exposure to other and unspecified factors. Homicide at the workplace was not assessed owing to a complete lack of data from developing countries. To estimate the impact of the disability produced by nonfatal injuries, years lived with disability (YLD) were estimated using

Table 21.60 Fatality rates due to occupational injuries (per 100000 insured workers) by country and year

Country	*Year(s)*	*Fatality rate per 100000*	*Source*
Australia	1982–1984	8.06	Harrison et al. (1989)
	1998–1999	4.0	Worker's Compensation Cases (NOSHC 2002)
Austria	1998	5.3	ILO (2000)
Bolivia	1995	3.7	PAHO/WHO (1998)
Brazil	1995	13.3	PAHO/WHO (1998)
Canada	1970–1997	8.79	Human Resources Development Canada (2000)
China	1997	11.1	Kam Lam (2000)
	1991–1997	9.1 (1991–1997); 11.5 (1997)	Xia et al. (2000)
Costa Rica	1996	10.5	PAHO/WHO (1998)
Cuba	1996	4.2	PAHO/WHO (1998)
Czech Republic	1999	4.2	ILO (2000)
Denmark	1999	2	ILO (2000)
Dominican Republic	1996	6.3	PAHO/WHO (1998)
El Salvador	1996	4.7	PAHO/WHO (1998)
European Union	1998	5.03	Dupre (2001)
Finland	1997	3.1	ILO (2000)
Ireland	1999	4.21	ILO (2000)
Jamaica	1996	11.8	PAHO/WHO (1998)
Jordan	1980–1993	25.5	Atallah et al. (1998)
Mexico	1996	10.4	PAHO/WHO (1998)
Namibia	1998/1999	25	Amweelo (2000)
New Zealand	1985–1994	5.03	Feyer et al. (2001)
Panama	1996	14.5	PAHO/WHO (1998)
Peru	1996	190	PAHO/WHO (1998)
Philippines	1999	11	National Statistics Office Philippines (2000)
Poland	1999	4.5	ILO (2000)
Slovenia	1998	4	ILO (2000)
Singapore	2000	10.82	Singapore Government (2000)
Spain	2000	9.2	Ministerio de Trabajo y Asuntos Sociales (2002)
Sweden	1998	1.7	ILO (2000)
Thailand	1999	11.48	ILO (2000)
United Kingdom	1998	0.8	ILO (2000)
United States	1980–1995	4.25	NIOSH (2000a)
Venezuela	1997	0.58	ILO (2000)

the same attributable fractions as for mortality (age and sex) (i.e. it was assumed that an occupational injury had the same likelihood of being fatal as injuries caused by other factors).

6.2 Underreporting

Conventional sources of data on fatal injuries at work are compensation registries, insurance companies, death certificates and autopsy reports based on mortuary records. Data from compensation registries and insurance companies underestimate the magnitude of fatal injuries, either because they do not cover some sectors of the workforce or because they refer only to successful claims. To improve the accuracy of the reporting of fatal injuries at work, many countries gather data from different systems and data sources (death certificates, insurance companies, labour inspectorates, coroners' files, medical examiners' files) or develop specific projects. Wide disparities exist regarding the accuracy of these sources in identifying fatal injuries at work.

Despite the usefulness of the death records, data from the United States reveal that such records identify only between 67% and 90% of fatal injuries at work. A similar underreporting (72.3%) has been found in the Mortality Registry of Tuscany in Italy (Chellini et al. 2002). The only study in a developing country that analysed underreporting showed that 28% of occupational fatalities in Cape Town, South Africa had not been reported in terms of statutory regulations (Lerer and Myers 1994). The level of underreporting increases to between 78% and 85% in rural areas (Schierhout et al. 1997). On the other hand, special registries also underreport; the National Fund for Occupational Diseases in Italy, for example, has a reporting rate of only 56.4% (Chellini et al. 2002).

To our knowledge, the most accurate system currently in place that uses multiple data sources to identify and classify work-related injuries is in the United States. Data are gathered from death certificates in two surveillance systems: the National Traumatic Occupational Fatality System (NTOF) of NIOSH and in the Bureau of Labor Statistics CFOI system. Thus it would appear that the United States has fairly complete records of occupational deaths due to injury (CDC 2001). Although, owing to paucity of data, we did not use the estimates of underreporting to calculate the rates of fatal injuries due to risks at work, this does indicate likely underestimation.

7. Results

Tables 21.61–21.63 present the overall attributable fractions, mortality and burden of disease for the selected occupational risk factors considered here.

In total, occupational risk factors considered here were responsible for 775 000 deaths worldwide in 2000. There were five times as many deaths in males as in females: 647 000 vs 128 000. The leading occupational

Table 21.61 Attributable fractions (%) for the disease burden due to occupational exposure

Risk factor	*Outcome*	*Males*	*Females*	*Total*
Ergonomic stressors	Low back pain	41	32	37
Noise	Hearing Loss	22	11	16
Agents leading to COPD	COPD	18	6	13
Asthmagens	Asthma	14	7	11
Risk factors for injuries	Unintentional injuries	12	2	8
Beryllium, cadmium, chromium, diesel exhaust, nickel, arsenic, asbestos, silica	Trachea, bronchus or lung cancer	10	5	9
Benzene, ethylene oxide, ionizing radiation	Leukaemia	2	2	2

Table 21.62 Deaths (000s) due to occupational exposure[a]

				Total	
Risk factor	*Outcome*	*Males*	*Females*	*Deaths*	*% total from occupational risk factors*
Agents leading to COPD	COPD	240	78	318	41
Risk factors for injuries	Unintentional injuries	291	19	310	40
Beryllium, cadmium, chromium, diesel exhaust, nickel, arsenic, asbestos, silica	Trachea, bronchus or lung cancer	88	14	102	13
Asthmagens	Asthma	23	15	38	5
Benzene, ethylene oxide, ionizing radiation	Leukaemia	4	3	7	1
Total		647	128	775	100

[a] Asbestos exposure is the most important cause of mortality from mesothelioma. Cause-of-death statistics coded in ICD-10 allow direct estimation of the total number of mesothelioma deaths. Using this method, recent studies suggest that each year there are about 700 malignant mesothelioma deaths in Australia (Leigh and Driscoll 2003), 700 in Japan (Furuya et al. 2003), 2600 in the United States (Price and Ware 2004), and 4000 in Europe (Peto et al. 1999). A large proportion of these deaths are undoubtedly caused by asbestos exposure, primarily work-related. Combining estimates of asbestos exposure in all 14 subregions with hazards obtained from these studies would result in an estimate of more than 40 000 mesothelioma deaths caused by asbestos exposure in the world. Of these preliminary estimates, about 9000 occur in developed countries (AMR-A, EUR and WPR-A), 9 000 in SEAR-D, and 16 000 in WPR-B. These estimates are subject to uncertainty, especially in developing countries where ICD-10 cause-of-death data and detailed data on history of asbestos exposure are not available. These preliminary estimates are currently undergoing further refinement by authors. Preliminary estimates also indicate that there may have been approximately 9000 deaths from silicosis, 7000 deaths from asbestosis and 14 000 deaths from coal workers' pneumoconiosis as a result of exposure to occupational dusts (silica, asbestos and coal dust) in 2000.

Table 21.63 DALYs (000s) due to occupational exposure

				Total	
Risk factor	*Outcome*	*Males*	*Females*	*DALYs*	*% total from occupational risk factors*
Risk factors for injuries	Unintentional injuries	9779	718	10496	48
Noise	Hearing Loss	2788	1362	4150	19
Agents leading to COPD	COPD	3020	713	3733	17
Asthmagens	Asthma	1110	511	1621	7
Beryllium, cadmium, chromium, diesel exhaust, nickel, arsenic, asbestos, silica	Trachea, bronchus or lung cancer	825	144	969	4
Ergonomic stressors	Low back pain	485	333	818	4
Benzene, ethylene oxide, ionizing radiation	Leukaemia	66	35	101	0
Total		18073	3816	21889	100.0

cause of death was COPD (41%) followed by unintentional injuries (40%) and trachea, bronchus or lung cancer (13%). Workers who developed outcomes related to occupational risk factors lost about 22 million years of healthy life. By far the main cause of years of healthy life lost, within occupational diseases, was unintentional injuries (with 48% of the burden). This was followed by hearing loss due to occupational noise (19%) and COPD due to occupational agents (17%). Among the occupational factors analysed in this study, these three conditions accounted for 84% of years of healthy life lost. DALYs were almost five times greater in males than in females. Low back pain and hearing loss have in common the fact that they do not directly produce premature mortality, but substantial disability. This feature differentiates these conditions from the others analysed in the study. Results for specific risk factors are provided below.

7.1 Carcinogens

Tables 21.64–21.68 summarize the attributable fractions, mortality and burden of disease for the occupational carcinogens considered here.

For lung cancer, the attributable fraction varied from 5% in AMR-A to 14% in EUR-C, with overall attributable fractions for lung cancer estimated to be 10% for men and 5% for women (9% overall). For leukaemia, estimates of the attributable fraction varied from 1% in EMR-D to 3% in several subregions. There were estimated to be approximately 7000 deaths from leukaemia each year, with a much more even proportion between males and females than was seen for lung cancer, although approximately two thirds of the DALYs are due to male cases.

Table 21.64 Attributable fractions for lung cancer and leukaemia disease burden caused by workplace exposure

	Lung cancer			*Leukaemia*		
Subregion	*Males*	*Females*	*Total*	*Males*	*Females*	*Total*
AFR-D	9	4	7	3	1	2
AFR-E	9	4	7	3	2	3
AMR-A	6	2	5	3	3	3
AMR-B	11	3	8	2	2	2
AMR-D	12	2	8	3	2	3
EMR-B	12	2	9	3	2	2
EMR-D	9	3	7	2	1	1
EUR-A	7	2	6	3	3	3
EUR-B	12	4	10	3	2	3
EUR-C	15	9	14	2	2	2
SEAR-B	10	4	9	2	2	2
SEAR-D	11	4	9	2	0	2
WPR-A	8	3	6	2	2	2
WPR-B	12	7	10	2	2	2
World	10	5	9	2	2	2

Table 21.65 Deaths (000s) from lung cancer and leukaemia caused by workplace exposure

	Lung cancer			*Leukaemia*		
Subregion	*Males*	*Females*	*Total*	*Males*	*Females*	*Total*
AFR-D	1	0	1	0	0	0
AFR-E	1	0	1	0	0	0
AMR-A	7	2	8	0	0	1
AMR-B	4	0	4	0	0	0
AMR-D	0	0	0	0	0	0
EMR-B	1	0	1	0	0	0
EMR-D	1	0	1	0	0	0
EUR-A	11	1	12	1	0	1
EUR-B	6	0	6	0	0	0
EUR-C	12	1	14	0	0	0
SEAR-B	3	0	3	0	0	0
SEAR-D	11	1	12	0	0	1
WPR-A	3	0	4	0	0	0
WPR-B	27	7	34	1	1	2
World	88	14	102	4	3	7

Table 21.66 DALYs (000s) due to lung cancer and leukaemia caused by workplace exposure

	Lung cancer			*Leukaemia*		
Subregion	*Males*	*Females*	*Total*	*Males*	*Females*	*Total*
AFR-D	6	1	7	2	1	3
AFR-E	9	2	11	4	2	6
AMR-A	53	13	65	4	3	7
AMR-B	34	4	38	4	4	8
AMR-D	2	0	2	2	1	2
EMR-B	10	1	11	2	1	3
EMR-D	14	2	16	3	1	4
EUR-A	89	9	99	6	4	10
EUR-B	60	5	65	3	2	5
EUR-C	127	14	140	2	2	4
SEAR-B	32	3	34	3	2	5
SEAR-D	109	11	120	10	1	11
WPR-A	23	3	26	1	1	2
WPR-B	257	76	333	19	11	30
World	825	144	969	66	35	101

Table 21.67 Age-specific attributable fractions, deaths and DALYs for lung cancer and leukaemia, males

	Age group (years)						
	15–29	*30–44*	*45–59*	*60–69*	*70–79*	*80–89*	*All ages*
Attributable fractions (%)							
Lung cancer	11	11	10	10	10	9	10
Leukaemia	3	3	3	3	3	3	2
Deaths (000s)							
Lung cancer	0	3	20	30	26	8	88
Leukaemia	1	1	1	1	1	0	4
DALYs (000s)							
Lung cancer	10	76	306	279	136	18	825
Leukaemia	29	13	11	7	4	1	66

For each condition, deaths were predominantly among older persons up to 79 years, whereas DALYs tended to be highest in the younger age groups.

7.2 Nonmalignant respiratory diseases

Tables 21.69–21.74 summarize the attributable fractions, mortality and disease burden for asthma and COPD risk factors, each estimated as described earlier.

Table 21.68 Age-specific attributable fractions, deaths and DALYs for lung cancer and leukaemia, females

	Age group (years)						
	15–29	*30–44*	*45–59*	*60–69*	*70–79*	*80–89*	*All ages*
Attributable fractions (%)							
Lung cancer	5	5	5	5	4	4	5
Leukaemia	2	3	3	3	3	3	2
Deaths (000s)							
Lung cancer	0	1	3	4	4	2	14
Leukaemia	0	0	0	0	1	1	3
DALYs (000s)							
Lung cancer	3	19	52	41	25	4	144
Leukaemia	10	8	8	4	3	1	35

Table 21.69 Attributable fractions (%) for mortality from asthma and COPD caused by workplace exposure

	Asthma			*COPD*		
Subregion	*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

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. The attributable fraction for mortality from asthma varied between subregions 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

Table 21.70 Attributable fractions (%) for burden of disease (DALYs) for asthma and COPD caused by workplace exposure

	Asthma			*COPD*		
Subregion	*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

Table 21.71 Numbers of deaths (000s) from asthma and COPD caused by workplace exposure

	Asthma			*COPD*		
Subregion	*Males*	*Females*	*Total*	*Males*	*Females*	*Total*
AFR-D	1	1	2	4	1	6
AFR-E	2	1	3	5	1	7
AMR-A	0	0	1	12	2	14
AMR-B	1	0	1	8	1	9
AMR-D	0	0	0	0	0	0
EMR-B	0	0	0	1	0	1
EMR-D	2	1	2	7	1	8
EUR-A	1	1	1	16	2	18
EUR-B	1	1	2	5	1	7
EUR-C	2	1	3	12	2	15
SEAR-B	2	2	4	8	1	9
SEAR-D	7	5	12	47	13	60
WPR-A	1	0	1	3	0	4
WPR-B	3	3	6	109	52	161
World	23	15	38	240	78	318

Table 21.72 DALYs (000s) from asthma and COPD caused by workplace exposure

	Asthma			*COPD*		
Subregion	*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

Table 21.73 Age-specific mortality attributable fractions, deaths and DALYs for asthma and COPD, males

	Age group (years)						
	15–29	*30–44*	*45–59*	*60–69*	*70–79*	*80–89*	*All ages*
Attributable fractions (%)							
Asthma	23	23	23	22	22	21	21
COPD	17	18	18	18	18	19	18
Deaths (000s)							
Asthma	3	4	6	4	4	2	23
COPD	0	3	29	56	91	62	240
DALYs (000s)							
Asthma	670	228	144	43	20	5	1110
COPD	88	564	992	710	517	149	3020

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 nonfatal and nonoccupational in origin.

For COPD mortality, the attributable fraction varied between subregions from 9% in AMR-D to 16% in EUR-C and WPR-A (Table 21.69).

Table 21.74 Age-specific mortality attributable fractions, deaths and DALYs for asthma and COPD, females

	Age group (years)						
	15–29	*30–44*	*45–59*	*60–69*	*70–79*	*80–89*	*All ages*
Attributable fractions (%)							
Asthma	13	14	14	13	13	12	13
COPD	6	5	5	6	6	6	6
Deaths (000s)							
Asthma	2	3	4	2	2	2	15
COPD	0	1	6	13	28	30	78
DALYs (000s)							
Asthma	228	95	81	28	15	5	511
COPD	45	133	149	152	166	69	713

Worldwide attributable fractions for COPD were estimated to be 18% for men and 6% for women (12% overall). Overall attributable fractions (based on DALYs and reflecting mortality and morbidity) were very similar to the mortality-based fractions (see Tables 21.69 and 21.70). The estimated number of deaths is 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, owing in part to the large population of the subregion, high background COPD mortality rates and the relatively high employment in mining.

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. The ratio was about two for disease burden. 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 (see Tables 21.73 and 21.74).

7.3 Noise

Occupational noise-induced hearing loss accounted for more than four million DALYs, all of them produced by the disability associated with hearing loss (YLD). Worldwide, the burden of hearing loss attributed to occupational noise is 16%, ranging between 7% in WPR-A and 21% in WPR-B. By sex, the effects of exposure to occupational noise are larger for males than for females in all subregions (Table 21.75). Attributable fractions are related to age group and sex in all subregions. Males usually

Table 21.75 Attributable fractions of occupational noise-induced hearing loss, by sex and subregion

Subregion	*Males*	*Females*	*All*
AFR-D	23	11	17
AFR-E	23	12	18
AMR-A	12	5	9
AMR-B	19	9	15
AMR-D	18	9	14
EMR-B	20	9	15
EMR-D	20	13	16
EUR-A	13	5	9
EUR-B	24	13	19
EUR-C	24	13	18
SEAR-B	23	16	19
SEAR-D	24	9	16
WPR-A	9	6	7
WPR-B	26	15	21
World	22	11	16

Table 21.76 Attributable fractions (%) and DALYs (000s) for occupational noise-induced hearing loss, by age group

	Age group (years)[a]												
	15–29		*30–44*		*45–59*		*60–69*		*70–79*		*Total*		
	Males	*Females*	*Males*	*Females*	*Males*	*Females*	*Males*	*Females*	*Males*	*Females*	*Males*	*Females*	*All*
Attributable fraction	29	16	29	16	21	11	13	6	3	1	22	11	16
DALYs	425	206	1144	530	925	482	271	136	23	9	2788	1362	4151

[a] AF was set to zero for ≥80.

experience greater exposure to noise at work than females, owing to differences in occupational categories, economic sectors of employment and working lifetime. In this study, the attributable fraction decreased with age group after 30–44 years, indicating the heavy impact of occupational noise on the burden of hearing loss at younger ages (Table 21.76). The 30–44-year age group accounted for the highest number of DALYs and the 70–79-year age group for the lowest (1 673 000 vs 32 000).

Table 21.77 provides estimates of the number of DALYs (in thousands) produced by occupational noise-induced hearing loss by subregion in the year 2000. Overall, four million DALYs were lost owing to noise-induced hearing loss. SEAR-D and WPR-B accounted for more

Table 21.77 DALYs (000s) due to occupational noise-induced hearing loss, by sex and subregion

Subregion	*Males*	*Females*	*All*
AFR-D	109	49	157
AFR-E	127	60	186
AMR-A	92	31	123
AMR-B	122	43	165
AMR-D	15	6	20
EMR-B	60	21	81
EMR-D	142	88	230
EUR-A	117	47	164
EUR-B	92	50	142
EUR-C	136	92	228
SEAR-B	219	185	404
SEAR-D	799	303	1101
WPR-A	26	22	48
WPR-B	735	365	1100
World	2788	1362	4151

Table 21.78 Attributable fraction and DALYs of low back pain due to occupational ergonomic stressors, by sex and subregion

	AF (%)			*DALYs (000s)*		
Subregion	*Males*	*Females*	*All*	*Males*	*Females*	*All*
AFR-D	36	29	33	21	16	37
AFR-E	36	31	33	25	20	45
AMR-A	35	25	30	17	10	27
AMR-B	41	23	33	32	15	47
AMR-D	34	18	27	4	2	6
EMR-B	31	12	22	9	3	12
EMR-D	36	25	31	25	16	41
EUR-A	34	22	29	21	11	32
EUR-B	43	37	40	18	12	30
EUR-C	45	36	41	21	14	34
SEAR-B	43	34	39	26	19	46
SEAR-D	43	34	38	111	78	189
WPR-A	38	27	33	9	5	14
WPR-B	44	38	41	146	110	256
World	41	32	37	485	333	818

Table 21.79 Summary results describing the global burden of occupational injuries

Measure	*Males*	*Females*	*Total*
Attributable fraction for disease burden (%)	12	2	8
Deaths (000s)	291	19	310
DALYs (000s)	9779	718	10496

than half of the years of healthy life lost (1.1 million each in SEAR-D and WPR-B). Males lost twice the number lost by females (2 788 000 vs 1 362 000).

7.4 Ergonomic factors

The attributable fractions for low back pain ranged from 22% to 41% among the subregions (Table 21.78). Differences by age group were quite small, and the attributable fractions for the total working population (ages 15–65 years) were rather consistent. In most geographical regions, women have a lower attributable burden of low back pain than men, although the difference is most pronounced in the eastern Mediterranean region and the less developed countries in the Americas.

Occupational ergonomic stressors caused 818 000 DALYs due to low back pain in 2000 (Table 21.78). Globally, 37% of low back pain was attributable to occupational causes. The occupational contribution to the burden of low back pain varied relatively little between subregions, with 22% being the lowest (EMR-B) and 41% the highest (EUR-C and WPR-B). The attributable fraction in men (41%) was slightly higher than that in women (32%), which is mainly due to the type of work men perform, involving more vibration, heavy physical loads or handling of materials.

7.5 Risk factors for injuries

Work-related risk factors for unintentional injuries represent 8% of the burden of unintentional injuries. In all regions, the highest attributable fractions were found in males, reflecting the high number of males exposed to hazardous conditions in the workplace. Overall, the attributable fraction for males was 12% and for women 2%. Occupational injuries were responsible for 310 000 deaths (291 000 males and 19 000 females).

In 2000, there were 10 496 000 years of healthy life (DALYs) lost among exposed workers (Table 21.79). Overall, males lost about 90% of healthy life years owing to unintentional injuries at work.

8. Discussion

We have attempted to estimate the burden of disease due to selected occupational risk factors by considering exposure, rather than the

common actuarial approach. In this study, a methodology based on the EAP, economic sectors and subsectors and occupational categories was developed to quantify the exposure. Assignment of exposure (low/high) within these categories allowed us to make estimations about the amount of exposure to a given risk factor or groups of risk factors causing an outcome. The dominant source of uncertainty in this analysis was characterizing exposure, which was solely based on economic subsectors and/or occupations and involved a large number of extrapolations and assumptions. High-quality exposure data are lacking, especially in developing countries, and European and American exposure estimates were thus applied in many instances in developing regions (B, C, D and E subregions). This extrapolation could have substantial impact on the accuracy of analysis for the developing regions if exposures, as usually occur, vary from place to place and over time. Diseases with long latency (e.g. cancers) are those that are more susceptible to the assumptions and extrapolations. In addition to problems produced by the length of the latency period, the magnitude of the excess risk may vary depending on the age of the person when exposure began, the duration and strength of exposure and other concomitant exposures. The turnover of workers is another problem that affects both exposure and risk assessment.

The accuracy of the exposure data is fairly coarse because exposures vary greatly within an occupation. This indirect estimation of exposure may cause misclassification of the true exposure situation. The proportions of exposed workers with high exposure in the A and in the B, C, D and E subregions were less than the published data would indicate. This may be partly because the published literature often focuses on industries and/or occupations with high exposure, but may also indicate an underestimation of true exposure.

Sources of uncertainty in hazard estimates (relative risk and mortality rates) include variations determined from the literature (once again caused by the use of different exposure proxies), extrapolations to regions with different working conditions, the application to females of risk measures from male cohorts, and the application of the same relative risk values to all age groups (e.g. carcinogens).

Restricting the analysis to persons aged ≥15 years excludes the quantification of child labour. The exclusion of children in the estimation was due to the wide variation in the youngest age group for which countries reported EARs. In addition to inconsistent data on EARs for children, there was virtually no data available on their exposure to occupational risk factors or the relative risks of such exposures. Specific, focused research on children is needed to quantify the global burden of disease due to child labour and the resulting implications.

Owing to lack of global data, we could not analyse occupational contributions to the global burden of infectious diseases, cardiovascular dis-

orders, MSDs of the upper extremities, skin disorders and other conditions with recognized occupational etiologies.

8.1 Occupational carcinogens

For each condition, deaths were predominantly in older persons up to 79 years, whereas DALYs tended to be highest in the younger age groups. The estimated overall attributable fractions for lung cancer of 10% for men and 5% for women (9% overall) are similar to those from a recent United States study, based on a review of relevant studies, in which the attributable fraction for lung cancer was estimated to be between 6% and 17% for men, and to be about 2% for women (Steenland et al. 2003). A similar Finnish study used estimates of 29% (men) and 5% (women) but these included a contribution from environmental tobacco smoke, which the study estimated to be about 2% or 3% (Nurminen and Karjalainen 2001). (The United States study did not include any contribution from environmental tobacco smoke, but separately estimated the contribution of workplace environmental tobacco smoke to be 5.7% [Steenland et al. 2003]).

The estimated 2% attributable fraction for leukaemia compares with 0.8–2.8% for the United States (Steenland et al. 2003) and 18% (men) and 2% (women) for Finland (Nurminen and Karjalainen 2001). The higher Finnish estimate seems to arise from the inclusion of occupational exposure to electromagnetic fields, from the reliance on different studies for relative risk estimates, and from the exposure patterns in the Finnish population.

8.2 Nonmalignant respiratory diseases

Many of the issues relevant to a discussion of the results for particulates are also relevant to carcinogens, and were discussed in detail under that rubric. The estimated attributable fractions for asthma mortality 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% (Balmes et al. 2003; Blanc and Toren 1999). The Finnish study on which most of the occupational relative risk estimates used in this study were based had higher estimates for men (29%) and women (17%) (Karjalainen et al. 2002), but these estimates are based on Finnish workforce patterns, which are likely to differ from those in most other countries.

Estimates of the attributable fraction for COPD mortality varied between subregions 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).

8.3 Noise

Occupational noise-induced hearing loss accounted for more than four million DALYs, all of them produced by the disability associated with hearing loss (YLD). Worldwide, the burden attributed to occupational noise is 16%, ranging between 7% in WPR-A and 21% in WPR-B. By sex, the effects of exposure to occupational noise are larger for males than for females in all subregions. The attributable fraction decreased with age group after 30–44 years, indicating the heavy impact of occupational noise on the burden of hearing loss at younger ages.

In addition to causing irreversible hearing loss, high noise levels in the workplace cause elevated blood pressure, sleeping difficulties, annoyance and stress. Our findings indicate that occupational noise has multiple consequences, both for the individual and for society, and particularly for those suffering hearing loss at young ages. Most occupational noise exposure can be minimized by the use of engineering controls to reduce the generation of noise at its source, within complete hearing loss prevention programmes that include noise assessment, audiometric monitoring of workers' hearing, appropriate use of hearing protectors and worker education.

8.4 Ergonomic factors

Human capacity for work depends on many functions and attributes: body size, muscle strength, aerobic fitness, sensory perception and cognitive capacity. Features of the work environment that do not accommodate these needs may produce physical or psychosocial stressors on the human system. Work features that have received attention because of their adverse health effects include heavy manual handling and other types of strenuous work, and awkward body postures.

For this analysis, the exposure variable was work in an occupational category with its assigned level of risk (low, medium or high rate of low back pain). This exposure variable is the "proxy" for the combination of occupational exposures found in the specified occupation that are implicated in the etiology of low back pain.

Occupational ergonomic stressors caused 818 000 DALYs from low back pain in 2000. The attributable fractions of low back pain ranged from 22% to 41% among subregions, with the global fraction amounting to 37%. The African countries had the highest attributable fraction of low back pain for all age groups analysed. Fractions of 40% or above were reached in EUR-B and EUR-C and in SEAR-B. The attributable fraction in men (41%) was slightly higher than that in women (32%), which is mainly due to the type of work men perform, involving more vibration, heavy physical load or material handling. Subregional variations reflect differences in occupational types and exposure. Over half of the working population in AFR-D and AFR-E was employed in agriculture. In contrast, about one third of the working populations in the AMR

and EUR subregions were employed in production occupations ("operators") and another large fraction (40% or more) in professional, sales and clerical jobs. In general, males are more exposed than females because they constitute a higher proportion of the labour force. In the less developed subregions, males are generally more exposed because of the higher proportions of workers in formal agriculture than in the developed subregions. The proportion of females in the labour force was particularly low in EMR-B and EMR-D.

The available literature demonstrates the feasibility and benefits of workplace ergonomic interventions (training and engineering controls) that have been implemented by employers in numerous economic sectors. Effective abatement measures include redesigning workstations to eliminate the need for bending and twisting; installing material or patient hoists and other lifting devices; a greater variety of work tasks, to avoid repetitively loading the same body tissues; and improving the mechanical isolation of seating to reduce transmission of whole-body vibration. Training programmes are most effective when they address job design, target supervisory and management personnel along with the manual labour force, and take place in a setting where workers are empowered to utilize the knowledge imparted. In general, the coordination of multiple activities—workstation improvements, training, enhanced medical surveillance and management—within an intervention programme appears to be the most effective. This is consistent with the conclusions of Shannon et al. (1996, 1997) that lower injury rates are associated with workplace characteristics such as general workforce empowerment and top management's active leadership, together with delegation of decision-making authority regarding occupational safety.

8.5 Risk factors for injuries

To our knowledge, this is the first study to estimate attributable fractions of work-related risk factors for unintentional injuries within the overall burden of DALYs. Lack of data on exposure did not allow a risk based approach and the estimates were based on occupational injury registries. This will limit the applicability of these estimates to preventive purposes which are based on exposure. Our findings show that the overall attributable fraction of 9% reported in this study is above the upper range of values reported by Chen et al. (2001) in the United States. Chen et al. reported an overall attributable fraction of 3.8%, varying between 1.5% in Arizona and 9.8% in Alaska. The difference in the findings between the two studies is explained by the heavy burden of mortality in the DALY estimation in developing countries, especially when deaths occur in younger populations.

Our findings understate the importance of the impact of occupational risk factors leading to injuries in the overall burden of disease due to injuries. A major factor in the underestimation was our use of data from an insured population from one country. There is some evidence that

mortality can be greater in uninsured populations, but in the absence of consistent evidence, a similar mortality in the insured and uninsured populations was assumed (Dror 2001; Forastieri 1999; Loewenson 1998). Lerer and Myers (1994) found that 28% of occupational fatalities in Cape Town, South Africa, were not reported despite a statutory requirement to do so. Using this fraction, we may have missed about 100 000 occupational injury deaths due to underreporting. Also, we did not estimate the injury mortality due to intentional injuries such as homicides in the workplace, owing to the lack of data from developing countries. However, current evidence shows that intentional injuries must be present in such countries; thus the lack of an estimation of deaths due to this cause increases the degree of underestimation of the number of deaths due to injuries (e.g. by approximately 4% in Australia and New Zealand).

Analysis of the full contribution of injuries at work within the overall burden of injuries requires indicators that measure not only mortality but also morbidity. In some countries and regions, with constant or slightly decreasing mortality patterns, it has been observed that the decline in mortality is balanced by an increase in the severity of injuries and morbidity, especially long-lasting or permanent disabilities (CDC 2001; Guerrero et al. 1999). In these cases, evaluation of the effectiveness of preventive measures is also hampered.

Injuries are largely preventable by improvements to make work safer and healthier. Engineering controls, administrative policies, health and safety information and education to promote safety-conscious attitudes and behaviour are needed. Surveillance data must be developed to provide the basis for targeting preventive measures towards high-risk groups of workers. The distribution of burden by type of external cause of mortality has allowed the developed countries to focus on preventive actions at work, resulting in a reduction in injury rates over time. Similar analysis and preventive actions in other countries could greatly reduce injuries at the workplace.

8.6 Conclusion

The aim of this study was to estimate the attributable fractions of selected occupational exposures. The risk factors were selected according to the availability of data, the strength of evidence linking the occupational exposure and the outcome, and the amount of risk arising from the exposure. An important feature of these risk factors and the resulting disease burden is their concentration among the working population, especially those in high-risk occupations and sectors. Hazards at workplaces and the resulting illness and injury are understood most accurately in the formal sector, and even there much undercounting occurs. The burden in the informal sector in developing countries, where large proportions of the population work, is high and largely lacks description. Neither household and family agricultural work by women nor child

Table 21.80 Expected rate of growth of the economically active population between 2000 and 2010, by sex and subregion

	Growth rate	
Subregion	*Males*	*Females*
AFR-D	0.33	0.38
AFR-E	0.27	0.25
AMR-A	0.08	0.12
AMR-B	0.17	0.27
AMR-D	0.27	0.45
EMR-B	0.32	0.66
EMR-D	0.31	0.53
EUR-A	–0.35	0.03
EUR-B	–0.26	0.16
EUR-C	0.00	0.00
SEAR-B	0.17	0.24
SEAR-D	0.21	0.27
WPR-A	–0.03	0.04
WPR-B	0.11	0.11

Source: ILO (2002a).

labour were addressed in our study. Due primarily to lack of data in developing countries, we were unable to include important occupational risks for infectious diseases, dermatitis, reproductive disorders, some cancers, ischaemic heart disease, musculoskeletal disorders of the upper extremities, and other conditions such as workplace stress.

The estimated burden of occupational risk factors can be diminished by improving working conditions, as many examples from different countries have shown. Work-related diseases are largely preventable. For example, many dusty activities can be made safer by using wet methods, thus reducing workers' exposures to silica. Work surfaces can be adjusted to a worker's height, thereby reducing suffering from low back pain. Substituting safe chemicals for known carcinogens can prevent many cancers. A change of process can reduce noise levels, thus protecting workers' hearing. Attention to electrical safety or machine guarding can eliminate tragic injuries at the workplace.

9. Projections of future exposure

In the next 50 years, the population of the developing regions will steadily rise, whereas that of more developed regions is expected to change little because fertility levels will remain below replacement level (UN 2001). There will also be differences in growth rates between the

sexes. A negative growth rate among economically active males is expected to occur between 2000 and 2010 in developed regions such as Europe, while comparable female rates will continue increasing in most of the regions, including the developed ones (ILO 2002a) (Table 21.80).

The expected changes in the world population will affect the EAP as well as the median age of workers (Fullerton and Toosi 2001). These changes in the characteristics of the working population will be accompanied by a different distribution of employment in the economic sectors (agriculture, industry and services). Currently, the service sector of many economies is growing at a fast rate, while the agricultural sector is rapidly declining in developing countries and remains at a stable low level in developed countries. It is expected that these different patterns of growth within the economic sectors will continue in the coming years. Moreover, the expected changes will affect the distribution of occupations within an economic sector. In developed countries in which a change in the structure of the economy has been observed, there has been a shift in the proportion of workers from the "production" category in favour of professional, managerial, clerical and sales occupations.

9.1 Exposure estimation for the years 2010, 2020 and 2030

As mentioned above, the EAP by economic sector was used to estimate the working population exposed to some risk factors, including car-

Table 21.81 Projected EARs for the year 2010 by sex and subregion

Subregion	*Males*	*Females*
AFR-D	0.84	0.55
AFR-E	0.85	0.64
AMR-A	0.7	0.59
AMR-B	0.8	0.45
AMR-D	0.81	0.44
EMR-B	0.78	0.39
EMR-D	0.81	0.42
EUR-A	0.57	0.47
EUR-B	0.69	0.56
EUR-C	0.75	0.59
SEAR-B	0.82	0.62
SEAR-D	0.84	0.48
WPR-A	0.71	0.52
WPR-B	0.81	0.68

Source: ILO (2002a).

Table 21.82 Projected distribution of EAP by subregion in 2010, 2020 and 2030

	2010		2020		2030	
Subregion	EAP	% total	EAP	% total	EAP	% total
AFR-D	151 300 284	4.6	199 904 691	5.4	260 126 023	6.3
AFR-E	181 011 306	5.5	232 504 879	6.3	300 771 980	7.3
AMR-A	176 129 373	5.4	191 817 362	5.2	201 632 376	4.9
AMR-B	218 574 298	6.7	251 138 963	6.8	278 348 992	6.8
AMR-D	35 233 802	1.1	43 360 108	1.2	51 164 255	1.2
EMR-B	67 730 185	2.1	82 896 189	2.2	98 826 530	2.4
EMR-D	165 776 470	5.1	214 302 617	5.8	269 408 795	6.5
EUR-A	172 528 633	5.3	171 619 225	4.6	165 811 266	4.0
EUR-B	110 565 142	3.4	118 115 432	3.2	123 452 320	3.0
EUR-C	125 923 283	3.8	118 343 637	3.2	112 001 074	2.7
SEAR-B	173 799 078	5.3	196 214 683	5.3	213 861 114	5.2
SEAR-D	663 743 911	20.3	784 531 784	21.1	885 891 293	21.5
WPR-A	77 452 109	2.4	76 367 545	2.1	72 515 258	1.8
WPR-B	952 086 321	29.1	1 030 847 264	27.8	1 086 544 112	26.4
Total	3 271 854 196	100.0	3 711 964 378	100.0	4 120 355 388	100.0

cinogens, while occupational category within a sector was used for others, including noise and ergonomic stressors. Therefore, to project the exposed population for the years 2010, 2020 and 2030 a three-step procedure was followed: (i) the EAP was estimated; (ii) the EAP was distributed among economic sectors; and, where needed, (iii) occupational categories were distributed within the economic sectors.

EAP estimation

To obtain the EAP for the year 2010, we multiplied the overall population (2010) by the EARs by subregion for the year 2010 as estimated by ILO (See Table 21.81). Then, in the absence of other data, the same EAR by subregion was used for the years 2020 and 2030 to generate the EAP (see Equation 4). Calculations were restricted to persons aged ≥15 years by sex and subregion, thus allowing regional patterns to be preserved.

$$EAP_{15+j} = \sum [EAR_{2010}(\text{for each age group} \geq 15 \times \text{Population}_i(\text{for each age group} \geq 15))] \quad (4)$$

where

EAP_{15+j} = economically active population ≥15 years, φ=year (2010, 2020, 2030)

$Population_i$ = population year 2010, 2020, 2030

EAR = economic activity rate, year 2010

The EAP will increase steadily towards 2010, 2020 and 2030, but the amount of the increase and the patterns are somewhat different between developed and developing countries, as well as among countries having a similar degree of development. The percentage distribution of the EAP by subregion reflects the growth of the overall population, with greater growth in developing countries. WPR-B and SEAR-D will contribute 49.4% of global EAP in the year 2010, whereas developed subregions will contribute only 13.1% (Table 21.82).

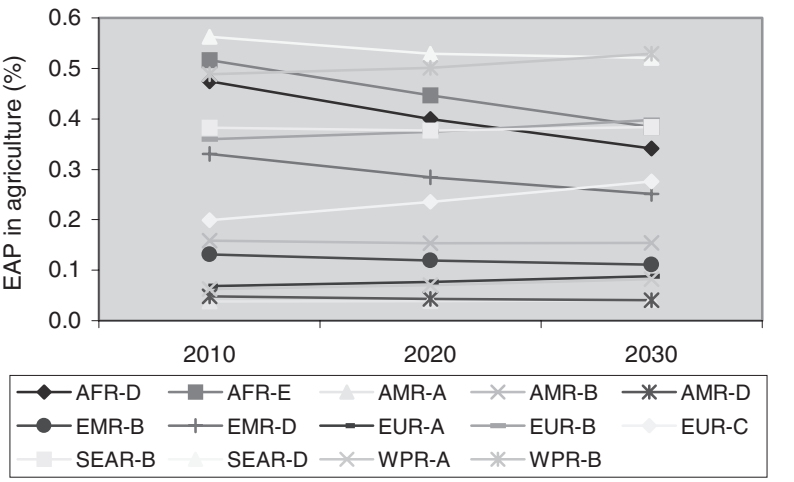
EAP DISTRIBUTION AMONG ECONOMIC SECTORS FOR 2010, 2020 AND 2030

The basic approach to estimating the EAP among economic sectors was to use regression analysis to identify the relationship between the distribution of the economic sectors and the projected years of interest. The dependent variables (proportion of EAP employed in agriculture, industry or services) were separately compared to the independent variable time,[10] using the following model:

$$PEAPA = \ln(a\ Y_T) + \ln b \qquad (5)$$

where

Figure 21.3 Projected distribution of the agricultural sector by year and subregion, 2010, 2020 and 2030



PEAPA = proportion of EAP in agriculture (similarly, PEAPI and PEAPS for industry, or service) in Year T

Y_T = Time (Year T)

The slope factors and intercepts obtained by regression analysis, using the EAP proportion by economic subsector for the years 1990–2000, were then used to estimate the proportion of the EAP for the years 2010, 2020 and 2030, separately for each economic subsector. We did not include economic development (e.g. measured as GDP per capita) as an additional variable in the analysis, assuming that previous trends capture the effects of trends in GDP. Given the economic and social factors that determine occupational distributions, the changes in the EAP in the future are subject to behavioural decisions by individuals, policy decisions in home countries and abroad, and developments in education. The project distribution of EAP among economic sectors showed different patterns among different subregions. As an example, Figure 21.3 presents the distribution of EAP in agriculture.

Occupational categories adjustment

No data were available to develop trends for employment in occupational categories in 2010, 2020 and 2030. Therefore, proportions of exposed workers within occupational categories were adjusted according to the distribution pattern of the year 2000, adjusted only for the new proportions employed within economic sectors in the year of interest.

Notes

1 See preface for an explanation of this term.

2 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 dust).

3 Economic activities comprise agriculture, mining, manufacturing, utilities, construction, trade, transport, finance and services.

4 dBA is the unit of sound pressure level in decibels that has been A-weighted, i.e. measured with an A-weighted sound level meter. Sound levels measured in dBA have been widely used to evaluate occupational and environmental exposures because of the good correlations between the "A" scale and human hearing ability at different frequencies, hearing damage and environmental annoyance.

5 The average of the hearing threshold levels for both ears that exceeds 25 dB at 1000, 2000, 3000 and 4000 Hz.

6 Tinnitus is noise originating in the ear rather than in the environment. The noise may be a buzzing, ringing, roaring, whistling, humming or hissing in the ears. Ringing in the ears is an extremely common phenomenon experienced by up to a third of the adult population at one time or another.

7 A temporary increase in the threshold of hearing for an ear caused by exposure to high-intensity noise.

8 The percentage of workers with a hearing impairment in an occupationally noise-exposed population, after subtracting the percentage in an unexposed population who would normally incur such impairment owing to ageing.

9 Year was the predictor of the data.

References

Ahmed HO, Dennis JH, Badran O et al. (2001) Occupational noise exposure and hearing loss of workers in two plants in eastern Saudi Arabia. *Annals of Occupational Hygiene*, **45**:371–380.

Alidrisi M, Jamil ATM, Jiffry MSA, Jefri MA, Erturk F (1990) Evaluation of noise stresses in Jeddah Industrial State. *Journal of Environment Science and Health*, **A25**:873–896.

Amweelo M (2000) Accident prevention in Namibia. *African Newsletter on Occupational Health and Safety* **10**(1).

Armstrong TJ, Buckle P, Fine LJ et al. (1993) A conceptual model for work-related neck and upper-limb musculoskeletal disorders. *Scandinavian Journal of Work, Environment and Health*, **19**:73–84.

Arndt V, Rothenbacher D, Brenner H et al. (1996) Older workers in the construction industry: results of a routine health examination and a five-year follow-up. *Occupational and Environmental Medicine*, **53**:686–691.

Astrand NE (1987) Medical, psychological, and social factors associated with back abnormalities and self reported back pain: a cross sectional study of male employees in a Swedish pulp and paper industry. *British Journal of Industrial Medicine*, **44**:327–336.

Atallah ZR, Jamous LW, AbuDhaise BA, Alwash RH (1998) Fatal occupational injuries in Jordan during the period 1980 through 1993. *Safety Science*, **28**:177–187.

Attfield M, Wagner G (1998) Respiratory disease in coal miners. In: *Environmental and occupational medicine*, 3rd edn. Rom W, ed. Little, Brown and Co., Boston, MA.

Badley EM, Rasooly I, Webster GK (1994) Relative importance of musculoskeletal disorders as a cause of chronic health problems, disability, and health care utilization: findings from the 1990 Ontario Health Survey. *The Journal of Rheumatology*, **21**:505–514.

Badley EM, Webster GK, Rasooly I (1995) The impact of musculoskeletal disorders in the population: are they just aches and pain? Findings from the 1990 Ontario Health Survey. *The Journal of Rheumatology*, **22**:733–739.

Balmes J, Becklake M, Blanc P et al. (2003) American Thoracic Society statement on occupational contribution to the burden of airway disease. *American Journal of Respiratory and Critical Care Medicine*, **167**:787–797.

Bao S, Winkel J, Mathiassen SE, Shahnavan H (1997) Interactive effect of ergonomics and production engineering on shoulder-neck exposure: a case study of assembly work in China and Sweden. *International Journal of Industrial Ergonomics*, **20**:75–85.

Battie MC, Bigos SJ (1991) Industrial back pain complaints: a broader perspective. *Orthopedics Clinics of North America*, **22**:273–282.

Becklake M (1989) Occupational exposures: evidence for a causal association with chronic obstructive pulmonary disease. *American Review of Respiratory Disease*, **140**:S85–91.

Becklake MR (1994) Symptoms and pulmonary functions as measures of morbidity. *Annals Occupational Hygiene*, **38**:569–580, 418.

Behrens V, Seligman P, Cameron L et al. (1994) The prevalence of back pain, hand discomfort, and dermatitis in the U.S. working population. *American Journal of Public Health*, **84**:1780–1785.

BEIR V (1990) *Health effects of exposure to low levels of ionizing radiation: BEIR V*. Committee on the biological effects of ionizing radiation. National Academy Press, Washington, DC.

Bergenudd H, Nilsson B (1988) Back pain in middle age: occupational work load and psychologic factors: an epidemiologic study. *Spine*, **13**:58–60.

Bernard BP, ed. (1997) *Musculoskeletal disorders and workplace factors: a critical review of epidemiologic evidence for work-related musculoskeletal disorders of the neck, upper extremity, and low back*. CDC/NIOSH, Cincinnati, OH.

Bernard BP, Sauter SL, Petersen M et al. (1993) Health hazard evaluation report: Los Angeles Times. (NIOSH Report No. 90-013-2277). Cincinnati, OH.

Bhatia R, Lopipero P, Smith A (1998) Diesel exhaust exposure and lung cancer. *Epidemiology*, **9**:84–91.

Bhattacharya SK, Saiyed HN, Roy A, Chatterjee SK (1981) Hearing acuity in weavers of a textile mill. *Indian Journal of Medical Research*, **74**:779–785.

Bhattacharya SK, Tripathi SR, Kashyap S (1990) Heat and noise problems in a firm in a drug and pharmaceutical firm in India. *Industrial Health*, **28**:203–207.

Blanc P, Toren K (1999) How much adult asthma can be attributed to occupational factors? *American Journal of Medicine*, **107**:580–587.

Bongers PM, de Winter CR, Kompier MAJ et al. (1993) Psychosocial factors at work and musculoskeletal disease. *Scandinavian Journal of Work, Environment and Health*, **19**:297–312.

Bongers PM, Hulshof CTJ, Dijkstra L, Boshuizen HC, Groenhout HJM, Valken E (1990) Back pain and exposure to whole body vibration in helicopter pilots. *Ergonomics*, **33**:1007–1026.

Bovenzi M, Betta A (1994) Low-back disorders in agricultural tractor drivers exposed to whole-body vibration and postural stress. *Applied Ergonomics*, **25**:231–241.

Broersen JPJ, de Zwart BCH, Dijk F et al. (1996) Health complaints and working conditions experienced in relation to work and age. *Occupational and Environmental Medicine*, **53**:51–57.

Buckle PW, Devereaux J (1999) *Work-related neck and upper limb musculoskeletal disorders*. European Agency for Safety and Health at Work, Luxembourg.

Bundesverband der Betriebskrankenkassen (2001) *Krankheitsartenstatistik 1997*. (Internet communication of 20 September 2001 at: http://de.osha.eu.int/statistics/bkk/index.stm).

Burchfiel CM, Boice JA, Stafford BA, Bond GG (1992) Prevalence of back pain and joint problems in a manufacturing company. *Journal of Occupational Medicine*, **34**:129–134.

Burdorf A (1992) Sources of variance in exposure to postural load on the back in occupational groups. *Scandinavian Journal of Work, Environment and Health*, **18**:361–367.

Burdorf A, Naaktgeboren B, deGroot HC (1993) Occupational risk factors for low backpain among sedentary workers. *Journal Occupational Medicine*, **35**:1213–1220.

Burdorf A, Sorock G (1997) Positive and negative evidence of risk factors for back disorders. *Scandinavian Journal of Work, Environment and Health*, **23**:243–256.

Bureau of Labor Statistics (2001) *Safety and health statistics*. Office of Safety, Health and Working Conditions. U.S. Department of Labor. (Internet Communication of 10 September 2001 at: http://stats.bls.gov/oshhome.htm).

Burton, DJ (1997) Chapter 31. General methods for the control of airborne hazards. In: *The industrial environment—its evaluation and control*. Di Nardi S, ed. AIHA, Fairfax, VA.

Butler S, Twaddle S (1979) Turnover in the restaurant industry: a structural explanation. Mid-South Sociological Association. *Sociological Abstracts*, **27**(4).

CCOHS (1997) *OSH answers*. Canadian Centre for Occupational Health and Safety. Available at http://www.ccohn.ca/oshanswers/diseases.

CDC (2001) Non-fatal occupational injuries and illnesses treated in hospital emergency departments—Unites States 1998. Centers for Disease Control and Prevention. *Journal of the American Medical Association*, **285**:2443–2444.

Chan-Yeung M, Malo J (1994) Aetiological agents in occupational asthma. *European Respiratory Journal*, **7**:346–371.

Checkoway (2000) Epidemiological evidence on the carcinogenicity of silica: factors in scientific judgement: letter. *Annals of Occupational Hygiene*, **44**:483–487.

Chellini E, Baldasseroni A, Giovannetti L, Zoppi O (2002) A survey on fatal work accidents based on mortality registry data: results of the Tuscany study

on INAIL and RMR cases in the period 1992–1996. *Epidemiologia e Prevenzione*, **26**:11–17.

Chen W, Zhuang Z, Attfield MD et al. (2001) Exposure to silica and silicosis among tin miners in China: exposure-response analyses and risk assessment. *Occupational and Environmental Medicine*, **58**:31–37.

Chien V, Chai S, Hai D et al. (2002) Pneumoconiosis among workers in a Vietnamese refractory brick facility. *American Journal of Industrial Medicine*, **42**:397–402.

Chiou, WK, Wong MK (1992) Epidemiology of low back pain in the nurses of Chang Gung Memorial Hospital. *Chang Gung Medical Journal*, **15**: 64–71.

Chiou WK, Wong MK, Lee YH (1994) Epidemiology of low back pain in Chinese nurses. *International Journal of Nursing Studies*, **31**:361–368.

Choi BCK, Tennassee LM, Eijkemans GJM (2001) Developing regional workplace health and hazard surveillance in the Americas. *Pan American Journal of Public Health*, **10**: 376–381.

Davis AC (1989) The prevalence of hearing impairment and reported hearing disability among adults in Great Britain. *International Journal of Epidemiology*, **18**:911–917.

de Zwart BCH, Broersen JP, Frings-Dresen MHW, van Dijk FJH (1997) Repeated survey on changes in musculoskeletal complaints relative to age and work demands. *Occupational and Environmental Medicine*, **54**:793–799.

Derriennic F, Iwatsubo Y, Monfort C et al. (1993) Evolution of osteoarticular disorders as a function of past heavy physical work factors: longitudinal analysis of 627 retired subjects living in the Paris area. *British Journal of Industrial Medicine*, **50**:851–860.

Deyo RA, Rainville J, Kent D (1992) What can the history and physical examination tell us about low back pain? *Journal of the American Medical Association*, **268**:760–765.

Dosemeci M, McLaughlin JF, Chen J-Q et al. (1995) Historical total and respirable silica dust exposure levels in mines and pottery factories in China. *Scandinavian Journal of Work, Environment and Health*, **2**:S39–43.

Dror DM (2001) Reinsurance of health insurance for the informal sector. *Bulletin of the World Health Organization*, **79**:672–678.

Dupre D (2001) *Accidents at work in the EU 1998–1999*. EUROSTAT (Theme 3-16/2001). Statistics in Focus, Population and Social Conditions, Luxembourg.

EASHW (2000) *Monitoring the state of occupational safety and health in the European Union—pilot study*. European Agency for Safety and Health at Work. Luxembourg.

EIA (2001) Energy Information Administration, U.S. Department of Energy, International Energy Database, January 2001, available at www.eia.doe.gov/emeu/iea/coal.html.

EIU (1995) Business operations report, India, 4th quarter, 1995. Shortage of managers. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (1996a) China hand. Ch. 13. Human resources: knowing why staff leave, 1 Sept. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (1996b) China labour: unemployed reluctant to trade welfare for work. Country view, 27 Nov. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (1997a) 28 February. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (1997b) Brazil regulations: rules eased on loans for exporters. Country alert, 6 May. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (1997c) Business operations report, Laos. Importance of training: training breeds staff loyalty, 26 Jun. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (1997d) Caribbean industry: region seeks to lure informatics firms. Country alert, 2 Jul. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (1997e) Country commerce South Korea. Human resources. Overview, 1 Jul. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (1997f) USA industry: Silicon Valley survey—of mice & men. Country view, 1 Apr. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (1998) Business operations report Eastern Europe. Business services: background checks on security personnel essential, 16 March.

EIU (2001a) Country commerce China. Human resources. Overview, 28 Feb. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (2001b) Country forecast Africa. AIDS/Africa: labour costs are set to increase substantially, 14 May. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (2001c) Country profile Hong Kong. Resources and infrastructure: education, 1 Jul. Economist Intelligence Unit. Available at http://www.eiu.com.

EIU (2002) Country commerce China. Human resources. Overview, 28 Feb. Economist Intelligence Unit. Available at http://www.eiu.com.

Emmett EA (2002) Occupational contact dermatitis I: incidence and return to work pressures. *American Journal of Contact Dermatitis*, **13**:30–34.

Enterline P, Marsh G, Esmen N, Henderson V, Callahan C, Paik M (1987) Some effects of cigarette smoking arsenic, and SO_2 on mortality among US copper smelter workers. *Journal of Occupational Medicine*, **29**:831–838.

Evans WA, Ming HY (1982) Industrial noise-induced hearing loss in Hong Kong: a comparative study. *Annals of Occupational Hygiene*, **25**:63–80.

Ezenwa A (2001) A study of fatal injuries in Nigerian factories. *Occupational Medicine*, **51**:485–489.

Ferris BG Jr, Speizer FE, Spengler JD et al. (1979) Effects of sulfur and respirable particles on human health: methodology and demography of populations in study. *American Review of Respiratory Disease*, **120**:767–779.

Feyer AM, Williamson AM, Stout N, Driscoll T, Usher H, Langley JD (2001) Comparison of work related fatal injuries in the United States, Australia, and New Zealand: method and overall findings. *Injury Prevention*, **7**:22–28.

Fine LJ, Silverstein BA, Armstrong TJ, Anderson CA, Sugano DS (1986) Detection of cumulative trauma disorders of upper extremities in the workplace. *Journal of Occupational Medicine*, **28**:674–678.

FIOH (1999) *CAREX database*. Finnish Institute of Occupational Health. Available at http://www.ocuphealth.fi/internet/english.

FIOH (2001) CAREX database. Finnish Institute of Occupational Health. Available at http://www.occuphealth.fi/list/data/CAREX.

Forastieri V (1999) *Improvement of working conditions and environment in the informal sector through safety and health measures*. International Labour Organization, Geneva.

Frank JW, Kerr MS, Brooker A-S et al. (1996) Disability resulting from occupational low back pain, Part I: What do we know about primary prevention? A review of the scientific evidence on prevention before disability begins. *Spine*, **21**:2908–2917.

Frank JW, Pulcins IR, Kerr MS et al. (1995) Occupational back pain—an unhelpful polemic. *Scandinavian Journal of Work, Environment and Health*, **21**:3–14.

Fullerton H, Toosi M (2001) Labor force projections to 2010:steady growth and changing composition. *Monthly Labor Review*, **124**:21–38.

Furuya S, Natori Y, Ikeda R (2003) Asbestos in Japan. *International Journal Occupational Environmental Health*, **9**:260–265.

Garg A (1992) Occupational biomechanics and low-back pain. *Occupational Medicine: State of the Art Review*, **7**:609–628.

Gomes J, Lloyd O, Norman N, Pahwa P (2001) Dust exposure and impairment of lung function at a small iron foundry in a rapidly developing country. *Occupational and Environmental Medicine*, **58**:656–662.

Guerrero JL, Sniezek JE, Sehgal (1999) The prevalence of disability from chronic conditions due to injury among adults ages 18–69 years: United States, 1994. *Disability and Rehabilitation*, **21**:187–192.

Guo H-R, Tanaka S, Cameron LL et al (1995) Back pain among workers in the United States: national estimates and workers at high risk. *American Journal of Industrial Medicine,* **28**:591–602.

Guo H-R, Tanaka S, Halperin WE, Cameron LL (1999) Back pain prevalence in U.S. industry and estimates of lost workdays. *American Journal of Public Health*, **89**:1029–1035.

Hagberg M, Silverstein BA, Wells RP et al., eds (1995) *Work-related musculoskeletal disorders (WMSD): a handbook for prevention*. Taylor and Francis. London.

Hales TR, Bernard BP (1996) Epidemiology of work-related musculoskeletal disorders. *Orthopedic Clinics of North America*, **27**:679–709.

Harrison JE, Frommer MS, Ruck EA, Blyth FM (1989) Deaths as a result of work-related injury in Australia, 1982–1984. *Medical Journal of Australia*, **50**:118–125.

Hayes R, Yin S, Dosemeci M et al. (1997) Benzene and the dose-related incidence of hematologic neoplasms in China. Chinese Academy of Preventive Medicine—National Cancer Institute Benzene Study Group. *Journal of the National Cancer Institute*, **89**:1065–1071.

Heistaro S, Vartiainen E, Heliövaara M et al. (1998) Trends of back pain in eastern Finland, 1972–1992, in relation to socioeconomic status and behavioral risk factors. *American Journal of Epidemiology*, **148**:671–682.

Hendrick DJ (1996) Occupational and chronic obstructive pulmonary disease (COPD). *Thorax* **51**:947–955.

Hernandez-Gaytan SI, Santos-Burgo AC, Becker-Meyer JP, Macias-Carrillo C, Lopez-Cervantes M (2000) Prevalence of hearing loss and correlated factors in a cement plant. *Salud Publica de Mexico*, **42**:106–111.

Hessel P (2000) Hearing loss among construction workers in Edmonton, Alberta, Canada. *Journal of Occupational and Environmental Medicine*, **42**:57.

Hessel PA, Sluis-Cremer GK (1987) Hearing loss in white South African gold-miners. *South African Medical Journal*, **71**:364–367.

Hewett P (1996) Interpretation and use of occupational exposure limits for chronic disease agents. *Occupational Medicine: State of the Art Reviews*, **11**:561–590.

Hildebrandt VH (1995) Back pain in the working population: prevalence rates in Dutch trades and professions. *Ergonomics*, **38**:1283–1298.

Hnizdo E, Sluis-Cremer G (1991) Silica exposure, silicosis, and lung cancer: a mortality study of South African gold miners. *British Journal Industrial Medicine*, **48**:53–60.

Hollman S, Klimmer F, Schmidt K-H et al. (1999) Validation of a questionnaire for assessing physical work load. *Scandinavian Journal of Work, Environment and Health*, **25**:105–114.

Holte HH, Tambs K, Bjerkedal T (2000) Manual work as predictor for disability pensioning with osteoarthritis among the employed in Norway 1971–1990. *International Journal of Epidemiology*, **29**:487–494.

Hoogendoorn WE, van Poppel MNM, Bongers PM, Koes BW, Bouter LM (1999) Physical load during work and leisure time as risk factors for back pain. *Scandinavian Journal of Work, Environment and Health*, **25**:387–403.

Hulshof CTJ, Veldhuijzen van Zanten B (1987) Whole-body vibration and low-back pain: a review of epidemiologic studies. *International Archives of Occupational and Environmental Health*, **59**:205–220.

Human Resources Development Canada (2000) *Surveys until 2000*. Available at http://www.hrdc-drhc.gc.ca/hrib/hrp-prh/ssd-des/english/glossary/survey.shtml.

IARC (1977) *Asbestos*. (Monograph 14.) International Association of Cancer Registries, Lyon.

IARC (1980) *Some metals and metallic compounds*. (Monograph 23.) International Association of Cancer Registries, Lyon.

IARC (1987a) *Overall evaluations of carcinogenicity: an updating of IARC*. (Monographs 1–42, Supplement 7.) International Association of Cancer Registries, Lyon.

IARC (1987b) *Silica and some silicates*. (Monograph 42.) International Association of Cancer Registries, Lyon.

IARC (1989) *Diesel and gasoline engine exhausts and some nitroarenes*. (Monograph 46.) International Association of Cancer Registries, Lyon.

IARC (1990a) *Chromium, nickel and welding*. (Monograph 49.) International Association of Cancer Registries, Lyon.

IARC (1990b) *Cancer*. IARC Scientific Publications. International Association of Cancer Registries, Lyon.

IARC (1993) *Beryllium, cadmium, mercury, and exposures in the glass manufacturing industry*. (Monograph 58.) International Association of Cancer Registries, Lyon.

IARC (1997) *Silica, some silicates, coal dust and para-aramid fibrils*. (Monograph 68.) International Association of Cancer Registries, Lyon.

IARC (2000) *Ionizing radiation, part 1: X- and gamma (g)-radiation, and neutrons*. (Monograph 75.) International Association of Cancer Registries, Lyon.

IARC (2001) *Overall evaluation of carcinogenicity to humans*. International Association of Cancer Registries, Lyon. Available at http://193.51.164.11/monoeval/crthall.html.

IARC (2002) *IARC Monographs Programme on the Evaluation of Carcinogenic Risks to Humans*. Available at http://193.51.164.11.

ICNCM (1990) Report of the International Committee on Nickel Carcinogenesis in Man. *Scandinavian Journal of Work, Environment and Health*, **16**:1–84.

ICRP (1991) *Recommendations of the International Commission on Radiological Protection*. (ICRP Publication No. 60. Vol 21:1–3.) International Commission on Radiological Protection, Oxford.

ILO (1987) *Resolutions adopted by the International Conference of Labour Statisticians*. Annex 1. Major, sub-major, minor and unit group titles. International Labour Organization. Available at http://www.ilo.org/public/english/bureau/stat/download/res/isco.pdf.

ILO (1995a) *Yearbook of labour statistics 1995, 54th issue*. International Labour Organization, Geneva.

ILO (1995b) *Sectoral activities programme, recent developments in the coal-mining industry, coal mines committee, 13th session, Report I*. International Labour Organization, Geneva.

ILO (1996) ILO *Yearbook of labour statistics 1996, 55th issue*. International Labour Organization, Geneva.

ILO (2000) *Yearbook of labour statistics, 59th issue*. International Labour Organization, Geneva.

ILO (2001) Laborsta, the labour statistics database. International Labour Organization. Available at http://laborsta.ilo.org/.

ILO (2002a) *Economically active population 1950–2010, 4th edn, rev. 2*. International Labour Organization, Geneva.

ILO (2002b) *The International Labor Conference, Report VI. Decent work and the informal economy*. International Labour Organization. Geneva.

IPCS (1998) *Chrysotile asbestos. Environmental health criteria 203*. International Programme on Chemical Safety, Geneva.

Jarup L, Nordberg G (1998) Cancer. In: *Health effects of cadmium exposure: a review of the literature and a risk estimate*. Jarup L, Berglund M, Elinder CG, Nordberg G, Vahter M, eds. *Scandinavian Journal Work, Environment and Health*, **24**:34–36.

Jensen RC (1988) Epidemiology of work-related back pain. *Topics Acute Care Trauma Rehabilitation*, **23**:1–15.

Jin K, Sorock GS, Courtney T et al (2000) Risk factors for work-related low back pain in the People's Republic of China. *International Journal of Occupational and Environmental Health*, **6**:26–33.

Johanning E (1991) Back disorders and health problems among subway train operators exposed to whole-body vibration. *Scandinavian Journal of Work Environment and Health*, **17**:414–419.

Johanning E, Wilder D, Landrigan P (1991) Whole-body vibration exposure in subway cars and review of adverse health effects. *Journal of Occupational Medicine*, **33**:605–612.

Johnson AG, Whyte WF (1977) The Mondragon system of worker production cooperatives. *Industrial and Labor Relations Review*, **31**:18–30.

Johnson D, Swift D (1997) Sampling and sizing particles. In: *The occupational environment—its evaluation and control*. Di Nardi SR, ed. The American Industrial Hygiene Association, Fairfax, VA.

Joshi TK, Menon KK, Kishore J (2001) Musculoskeletal disorders in industrial workers of Delhi. *International Journal of Occupational and Environmental Health*, **7**:217–222.

Juengprasert W (1997) Towards elimination of silicosis in Thailand. Mineral dusts and prevention of silicosis. *Asian-Pacific Regional Network on Occupational Safety and Health Information (ASIA-OSH)*, **4**(2).

Kam Lam TF (2000) *Occupational safety and health in China*. Asian labour update. Available at http://www.amrc.org.hk/alu/Alu39/013906.htlm.

Kamal AA, Eldamati SE, Faris F (1989) Hearing threshold of Cairo traffic policemen. *International Archives of Occupational and Environmental Health*, **61**:543–545.

Karjalainen A, Kurppa K, Martikanen R, Karjalainen J, Klaukka T (2002) Exploration of asthma risk by occupation -extended analysis of an incidence study of the Finnish population. *Scandinavian Journal of Work, Environment and Health*, **28**:49–57.

Karjalainen A, Kurppa K, Martikanen R, Klaukka T, Karjalainen J (2001) Work is related to a substantial portion of adult-onset asthma incidence in the Finnish population. *American Journal of Respiratory Critical Care Medicine*, **164**:565–568.

Kauppinen T, Toikkanen J, Pedersen D et al. (2000) Occupational exposure to carcinogens in the European Union. *Occupational and Environmental Medicine*, **57**:10–18

Khogali M (1970) Industrial health problems of developing countries. 1.1. Types of risk and their prevention. *Journal of Tropical Medicine and Hygiene*, **73**:269–274.

Klein BP, Jensen RC, Sanderson LM (1984). Assessment of workers' compensation claims for back strains/sprains. *Journal of Occupational Medicine*, **26**:443–448.

Klockhoff I, Drettner B, Svedberg A (1974) Computerized classification of the results of screening audiometry in groups of persons exposed to noise. *Audiology*, **13**:326–334.

Koch JL, Rhodes, SR (1981) Predictors of turnover of female factory workers. *Journal of Vocational Behavior*, **18**:145–161.

Kogevinas M, Antó JM, Soriano JB, Tobias A (1996) The risk of asthma attributable to occupational exposures: a population-based study in Spain. *American Journal of Respiratory Critical Care Medicine*, **154**:137–143.

Kogevinas M, Anto JM, Sunyer J, Tobias A, Kromhout H, Burney P (1999) Occupational asthma in Europe. *The Lancet*, **353**:1750–1754.

Korn RJ, Dockery DW, Speizer FE, Ware JH, Ferris BG Jr (1987) Occupational exposures and chronic respiratory symptoms: a population based study. *American Review of Respiratory Disease*, **136**:298–304.

Krzyzanowski M, Jedrychowski W, Wysocki M (1986) Factors associated with the change in ventilatory function and the development of chronic obstructive pulmonary disease in a 13-years follow-up of the Cracow study. *American Review of Respiratory Disease*, **134**:1011–1019.

Kumar A, Varghese M, Mohan D, Mahajan P, Gulati P, Kale S (1999) Effect of whole-body vibration on the low back. *Spine*, **24**:2506–2515.

Kuwashima A, Aizawa Y, Nakamura K, Taniguchi S, Watanabe M (1997) National survey on accidental low back pain in workplace. *Industrial Health*, **35**:187–193.

Lagerström M, Hansson T, Hagberg M (1998) Work-related low back problems in nursing. *Scandinavian Journal of Work, Environment and Health*, **24**: 449–464.

Lahiri S (2002) *Analysis of cost-effectiveness of interventions to reduce low back pain as a consequence of occupational ergonomic stressors*. World Health Organization, Geneva.

Lawrence RC, Helmick CG, Arnett FC et al. (1998) Estimates of the prevalence of arthritis and selected musculoskeletal disorders in the United States. *Arthritis and Rheumatism*, **41**:778–799.

Lee P (2001) Relation between exposure to asbestos and smoking jointly and the risk of lung cancer. *Occupational and Environmental Medicine*, **58**:145–153.

Leigh J, Driscoll T (2003) Malignant mesothelioma in Australia, 1945–2002. *International Journal Occupational Environmental Health*, **9**:206–217.

Leigh JP, Sheetz RM (1989) Prevalence of back pain among full-time United States workers. *British Journal of Industrial Medicine*, **46**:651–657.

Leino PI, Hänninen V (1995) Psychosocial factors at work in relation to back and limb disorders. *Scandinavian Journal of Work, Environment and Health*, **21**:134–142.

Leino-Arjas P, Hanninen K, Puska P (1998) Socioeconomic variation in back and joint pain in Finland. *European Journal of Epidemiology*, **14**:79–87.

Lerer LB, Myers JE (1994) Application of two secondary documentary sources to identify the underreporting of fatal occupational injuries in Cape Town, South Africa. *American Journal of Industrial Medicine*, **26**:521–527.

Lipsett M, Campleman S (1999) Occupational exposure to diesel exhaust and lung cancer: a meta-analysis. *American Journal of Public Health*, **89**:1009–1017.

Loewenson R (1998) Health impact of occupational risks in the informal sector in Zimbabwe. *International Journal of Occupational and Environmental Health*, **4**:264–274.

Lombardo LJ, Balmes JR (2000) Occupational asthma: a review. *Environmental Health Perspectives*, **8**:S697–704.

Loney P, Stratford P (1999) The prevalence of low back pain in adults: a methodological review of the literature. *Physical Therapy*, **79**:384–396.

Lucifora C (1998) The impact of unions on labour turnover in Italy: evidence from establishment level data. *International Journal of Industrial Organization*, **16**:353–376.

Lynge E, Anttila A, Hemminki K (1997) Organic solvents and cancer. *Cancer Causes and Control*, **8**:406–419.

MacDonald LA, Karasek RA, Punnett L, Scharf T (2001) Covariation between workplace physical and psychosocial stressors: evidence and implications for occupational health research and prevention. *Ergonomics*, **44**:696–718.

Magnusson ML, Pope MH, Wilder DG,Areskoug B (1996) Are occupational drivers at an increased risk for developing musculoskeletal disorders? *Spine*, **6**:710–717.

Malchaire J (2000) Strategy for prevention and control of the risks due to noise. *Occupational and Environmental Medicine*, **57**:361–369.

Malo J, Chan-Yeung (2001) Occupational asthma. *Journal of Allergy and Clinical Immunology*, **108**:317–328.

Marras WS, Allread DL, Burr DL, Fathallah FA (2000). Prospective validation of low-back disorder risk model and assessment of ergonomic interventions associated with manual materials handling tasks. *Ergonomics*, **43**: 1866–1886.

Marsh SM, Layne LA (2001) *Fatal injuries to civilian workers in the United States, 1980–1995 (National profile)*. Department of Health and Human Services. National Institute for Occupational Safety and Health (DHHS/NIOSH) 2001-129. Available at http://www.cdc.gov/niosh/NTOF2000/2001129.htlm.

Martonik JF, Nash E, Grossman E (2001) The history of OSHA's asbestos rule-makings and some distinctive approaches that they introduced for regulating occupational exposure to toxic substances. *American Industrial Hygiene Association Journal*, **62**:208–217.

Miedema HS, Chorus AMJ, Wevers CWJ, van der Linden S (1998) Chronicity of back problems during working life. *Spine*, **23**:2021–2029.

Milton DK, Solomon DM, Roseillo RA, Herrick RF (1998) Risk and incidence of asthma attributable to occupational exposure among HMO members. *American Journal of Industrial Medicine*, **33**:1–10.

Minino AM, Smith BL (2001) Deaths: preliminary data for 2000. *National Vital Statistics Reports*, **49**:1–40.

Ministerio de Trabajo y Asuntos Sociales (2002) [Ministry of Labour and Social Affairs (2002)] Gabinete de Comunicación. Información Estadística. [Communications Bureau. Statistical Information.] Available at http://www.mtas.es/. Informe de Resultados España [Report of results for Spain].

Moffett M (2002) *New Mexico labor market dynamics. Labor market review, special article*. New Mexico Department of Labor, February. Available at http://www3.state.nm.us/dol/dol_lmrsa6.htm.

Monso E, Munoz-Rino F, Izquierdo J et al. (1998) Occupational asthma in the community: risk factors in a western Mediterranean population. *Archives of Environmental Health*, **53**:93–98.

Morata TC, Fiorini AC, Fischer FM et al. (1997) Toluene-induced hearing loss among rotogravure printing workers. *Scandinavian Journal of Work, Environment and Health*, **23**:289–298.

Morken T, Moen B, Riise T et al (2000) Prevalence of musculoskeletal symptoms among aluminum workers. *Occupational Medicine*, **50**:141–421.

Moselhi M, El-Sadik YM, El-Dakhakhny A (1979) A six-year follow up study for evaluation of the 85 dBA safe criterion for noise exposure. *American Industrial Hygiene Association Journal*, **40**:424–426.

Mukherjee A K, Nag DP, Kakde Y, Prakash MN, Rao SR (1995) Noise level monitoring in a watch factory in Bangalore. *Indian Journal of Industrial Medicine*, **41**:42–44.

Muruka AO (1998) Age, height and duration in of service in relation to back pain among tea pickers in Kenya. *African Newsletter for Occupational Health and Safety*, **8**(3). Accessed at http://www.occuphealth.fi/e/info/anl/398/ in May 2002.

Myers JE, Lewis P, Hofmeyr W (1989) Respiratory health of brickworkers in Cape Town, South Africa: background, aims, and dust exposure determinations. *Scandinavian Journal of Work, Environment and Health*, **15**:180–187.

Nachemson A (1999) Back pain: delimiting the problem in the next millennium. *International Journal of Law and Psychiatry*, **22**:473–490.

Nachemson A, Jonsson E, eds (2000) *Neck and back pain: the scientific evidence of causes, diagnosis, and treatment*. Lippincott, Williams and Wilkins, Philadelphia, PA.

National Research Council (2001) *Musculoskeletal disorders and the workplace: low back and upper extremities*. Panel on musculoskeletal disorders and the workplace. Commission on Behavioral and Social Sciences and Education, National Research Council and Institute of Medicine. National Academy Press, Washington, DC. (also accessed at http://www.nap.edu/openbook).

National Statistics Office Philippines (2000*) Proceedings of the seminar on the ILO project on occupational injuries, 6 December*. National Statistics Office, Manila.

Nelson DI (1997) Risk assessment in the workplace. In: *The occupational environment—its evaluation and control*. Di Nardi SR, ed. The American Industrial Hygiene Association, Fairfax, VA.

Ng TP, Hong CY, Goh LG, Wong ML, Koh KT, Ling SL (1994) Risks of asthma associated with occupations in a community-based case-control study. *American Journal of Industrial Medicine*, **25**:709–718.

NIEHS (1999) United States. A world-class program. National Institute on Environmental Health Sciences. *Environmental Health Perspectives*, **107**(7). Available at http://ehpnet1.niehs.nih.gov/docs/1999/107-7/niehsnews.html.

NIOSH (1991) *NIOSH publications on noise and hearing*. Perspectives in disease prevention and health promotion, leading work-related diseases and injuries—United States. *Noise-induced Loss of Hearing*, July. National Institute for Occupational Safety and Health, Washington, DC.

NIOSH (1996) *Violence in the workplace: risk factors and prevention strategies*. National Institute for Occupational Safety and Health (NIOSH-CDC), Cincinnati, OH. Available at http://www.cdc.gov/niosh/violpr.htlm.

NIOSH (1998) *Criteria for a recommended standard: occupational noise exposure. Revised criteria 1998*. National Institute for Occupational Safety and Health, Cincinnati, OH. Available at http://www.cdc.gov/niosh/98-126.html.

NIOSH (1999) *Work-related lung disease surveillance report 1999*. U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health, Washington, DC.

NIOSH (2000a) *Worker health chartbook, 2000*. NIOSH-CDC. US Department of Health and Human Services, National Institute for Occupational Safety and Health, Washington, DC.

NIOSH (2000b) *Injuries, illnesses, and hazardous exposures in the mining industry, 1986–1995: a surveillance report*. (No. 2000-117.) National Institute for Occupational Safety and Health, Washington, DC.

NOHSC (1993) *National code of practice for management and protection of hearing at work* (NOHSC:2009.) National Occupational Health and Safety Commission, Australia. Available at http://www.nohsc.gov.au/OHSInformation/NOHSCPublications/fulltext/docs/h3/19.htm.

NOHSC (2001) National Occupational Health and Safety Commission, Australia. Available at http://www.nohsc.gov.au.

NOHSC (2002) *Statistics*. National Occupational Health and Safety Commission, Australia. Available at http://www.nohsc.gov.au/statistics/default.htm.

Noweir MH, El-Dakhakhny AA, Valic F (1968) Exposure to noise in the textile industry of the U.A.R. *American Industrial Hygiene Association Journal*, **29**:541–546.

Nurminen M, Karjalainen A (2001) Epidemiologic estimate of the proportion of fatalities related to occupational factors in Finland. *Scandinavian Journal Work, Environment and Health*, **27**:161–213.

Obiako MN (1979) Deafness and the mining industry in Zambia. *East African Medical Journal*, **56**:445–449.

Oleru UG (1980) Comparison of the hearing levels of Nigerian textile workers and a control group. *American Industrial Hygiene Association Journal*, **41**:283–287.

Oleru UG, Ijaduola GTA, Sowho EE (1990) Hearing thresholds in an auto assembly plant: prospects for hearing conservation in an [sic] Nigerian factory. *International Archives of Occupational and Environmental Health*, **62**:199–202.

Omokhodion FO, Umar US, Ogunnowo BE (2000) Prevalence of low back pain among staff in a rural hospital in Nigeria. *Occupational Medicine*, **50**:107–110.

Ory FG, Rahman FU, Katagade V, Shukla A, Burdorf A (1997) Respiratory disorders, skin complaints, and low-back trouble among tannery workers in Kanpur, India. *American Industrial Hygiene Association Journal*, **58**: 740–746.

Osibogun A, Igweze IA, Adeniran LO (2000). Noise-induced hearing loss among textile workers in Lagos Metropolis. *The Nigerian Postgraduate Medical Journal*, **7**:104–111.

Oxman A, Muir D, Shannon H, Stock S, Hnizdo E, Lange H (1993) Occupational dust exposure and chronic obstructive pulmonary disease. A systematic overview of the evidence. *American Review of Respiratory Disease*, **148**:38–48.

Ozguler A, Leclerc A, Landre M, Pietri-Taleb F, Niedhammer I (2000) Individual and occupational determinants of low back pain according to various definitions of low back pain. *Journal of Epidemiology and Community Health*, **54**:215–220.

PAHO/WHO (1998) *Informe del proyecto sistematización de datos básicos sobre salud de los trabajadores en países de las Américas—Agosto [Report of the project: systematisation of basic data of health of workers in LAC countries—August]*. División del Ambiente y Salud [Division for Environment and Health]. Pan American Health Organization/World Health Organization.

Palmer K, Pannett B, Griffin M (2001) *Occupational exposure to noise and hearing difficulties in Great Britain*. (Contract Report 361/2001.) University of Southampton for the Health and Safety Executive, Southhampton.

Paquet VL, Punnett L, Buchholz BO (1999) An evaluation of manual materials handling in highway construction work. *International Journal of Industrial Ergonomics*, **24**:431–444.

Partanen T, Jaakkola J, Tossavainen A (1995) Silica, silicosis and cancer in Finland. *Scandinavian Journal of Work, Environment and Health*, **2**:S84–86.

Partridge REH, Duthie JJR (1968) Rheumatism in dockers and civil servants: a comparison of heavy manual and sedentary workers. *Annals of Rheumatic Diseases*, **27**:559–568.

Pearce N, Matos E, Vainio H, Boffetta P, Kogevinas M (1994) *Occupational cancer in developing countries*. (IARC Scientific Publications No. 129) Lyon.

Perkio-Makela M, Riihimäki H (1997) Intervention on seat adjustment among drivers of forest tractors. *International Journal of Industrial Ergonomics*, **19**:231–237.

Peto R, Darby S, Deo H, Silcocks P, Whitley E, Doll R (2000). Smoking, smoking cessation, and lung cancer in the UK since 1950: combination of national statistics with two case-control studies. *British Medical Journal*, **321**: 323–329.

Peto J, Decarli A, La Vecchia C, Levi F, Negri E (1999) The European mesothelioma epidemic. *British Journal of Cancer*, **79**:666–672.

Phan Hong Son, Tran Ngoc Lan, Le Van Trung et al. (1999) *Use geographical information system to identify risk for silicosis and planning strategies for prevention, July, 1999*. Funded by the Fogarty International Center, NIH. Washington, DC.

Pope MH, Andersson GBJ, Frymoyer JW, Chaffin DB, eds (1991) *Occupational low back pain: assessment, treatment and prevention*. Mosby-Year Book, Inc., St. Louis, MO.

Price B, Ware A (2004) Mesothelioma trends in the United States: an update based on surveillance, epidemiology, and End Results program. Data for 1973 through 2003. *American Journal of Epidemiology*, **159**:107–112.

Prince MM, Stayner LT, Smith RJ, Gilbert S J (1997) A re-examination of risk estimates from the NIOSH Occupational Noise and Hearing Survey (ONHS) . *Journal of the Acoustic Society of America*, **101**:950–963.

Punnett L (1998) Ergonomic stressors and upper extremity disorders in vehicle manufacturing: cross-sectional exposure-response trends. *Occupational and Environmental Medicine*, **55**:414–420.

Punnett L (1999) The costs of work-related musculoskeletal disorders in automotive manufacturing. *New Solutions: A Journal of Environmental and Occupational Health Policy*, **9**:403–426.

Punnett L (2002) *Ergonomics and public health. Oxford textbook of public health*. 4th edn. Oxford University Press, Oxford.

Punnett L, Fine LJ, Keyserling WM, Herrin GD, Chaffin DB (1991) Back disorders and non-neutral trunk postures of automobile assembly workers. *Scandinavian Journal of Work, Environment and Health*, **17**:337–346.

Punnett L, Herbert R (2000) Work-related musculoskeletal disorders: is there a gender differential, and if so, what does it mean? In: *Women and health*. Goldman MB, Hatch MC, eds. Academic Press. San Diego, CA.

Quinlan M (2002) *Workplace health and safety effects of precarious employment*. In the Global Occupational Health Network Newsletter, Issue No. 2. World Health Organization, Geneva.

Raja S, Ganguly G (1983) Impact of exposure to noise on the hearing acuity of employees in a heavy engineering industry. *Indian Journal of Medical Research*, **78**:100–113.

Rees D, Cronje R, du Toit RSJ (1992) Dust exposure and pneumoconiosis in a South African pottery. 1. Study objectives and dust exposure. *British Journal of Industrial Medicine*, **49**:459–464.

Riihimäki H (1991) Low-back pain, its origin and risk indicators. *Scandinavian Journal of Work, Environment and Health*, **17**:81–90.

Riihimäki H (1995a) Back and limb disorders. In: *Epidemiology of work related diseases*. McDonald C, ed. BMJ Publishing Group, London.

Riihimäki H (1995b) Hands up or back to work- future challenges in epidemiologic research on musculoskeletal diseases. *Scandinavian Journal of Work, Environment and Health*, **21**:401–403.

Riihimäki H, Mattsson T, Zitting AJ et al. (1990) Radiographically detectable degenerative changes of the lumbar spine among concrete reinforcement workers and house painters. *Spine*, **15**:114–119.

Riihimäki H, Tola S, Videman T, Hänninen K (1989) Low-back pain and occupation: a cross-sectional questionnaire study of men in machine operating, dynamic physical work, and sedentary work. *Spine*, **14**:204–209.

Riihimäki H, Viikari-Juntura E, Moneta G, Kuha J, Videman T, Tola S (1994) Incidence of sciatic pain among men in machine operating, dynamic physical work and sedentary work. *Spine*, **19**:138–142.

Roach S (1992) *Health risks from hazardous substances at work—assessment, evaluation and control*. Pergamon Press, New York.

Schierhout GH, Midgley A, Myers JE (1997) Occupational fatality underreporting in rural areas of the Western Cape Province, South Africa. *Safety Science*, **25**:113–122.

Sears MR (1997) Descriptive epidemiology of asthma. *The Lancet*, **350**:S1–4.

Shaikh GH (1996) Noise problem in a polyester fiber plant in Pakistan. *Industrial Health*, **34**:427–431.

Shannon HS, Mayr J, Haines T (1997) Overview of the relationship between organizational and workplace factors and injury rates. *Safety Science*, **26**:201–17.

Shannon HS, Walters V, Lewchuk W et al. (1996) Workplace organizational correlates of lost-time accident rates in manufacturing. *American Journal of Industrial Medicine*, **29**:258–268.

Silverstein BA, Fine LJ, Armstrong TJ (1986) Hand wrist cumulative trauma disorders in industry. *British Journal of Industrial Medicine*, **43**:779–784.

Silverstein BA, Fine LJ, Stetson DS (1987) Hand-wrist disorders among investment casting plant workers. *The Journal of Hand Surgery*, **12A**:838–844.

Silverstein BA, Stetson DS, Keyserling WM, Fine LJ (1997) Work-related musculoskeletal disorders: comparison of data sources for surveillance. *American Journal of Industrial Medicine*, **31**:600–608.

Silverstein BA, Viikari-Juntura E, Kalat J (2002) Use of a prevention index to identify industries at high risk for work-related musculoskeletal disorders of the neck, back, and upper extremity in Washington state, 1990–1998. *American Journal of Industrial Medicine*, **41**:149–169.

Simpson M, Bruce R (1981) *Noise in America: the extent of the noise problem*. (EPA Report No. 550/9-81-101). Environmental Protection Agency, Washington, DC.

Singapore Government (2000) Statistics Singapore—statistical resources—statistical classifications—Singapore standard classification (SSOC), online portal available at http://www.gov.sg.

Singh N, Davis GS (2002) Review: occupational and environmental lung disease. *Current Opinions on Pulmonary Medicine*, **8**:117–125.

Skovron ML, Szpalski M, Nordin M, Melot C, Cukier D (1994) Sociocultural factors and back pain: A population-based study in Belgian adults. *Spine*, **19**:129–137.

Sluiter JK, Rest KM, Frings-Dresen MHW (2000) *Criteria document for evaluation of the work-relatedness of upper extremity musculoskeletal disorders*. SALTSA Joint Programme for Working Life Research in Europe and Academic Medical Center, University of Amsterdam, Amsterdam.

Smedley J, Egger P, Cooper C, Coggon D (1995) Manual handling activities and risk of low back pain in nurses. *Occupational and Environmental Medicine*, **52**:160–3

Sobti A, Cooper C, Inskip H et al. (1997) Occupational physical activity and long-term risk of musculoskeletal symptoms: a national survey of post office pensioners. *American Journal of Industrial Medicine*, **32**:76–83.

Soutar C, Robertson A, Miller B, Searl A, Bignon J (2000) Epidemiological evidence on the carcinogenicity of silica: factors in scientific judgement. *Annals of Occupational Hygiene*, **44**:3–14.

Sriwattanatamma P, Breysse P (2000) Comparison of NIOSH noise criteria and OSHA hearing conservation criteria. *American Journal of Industrial Medicine*, **37**:334–338.

Stayner L, Smith R, Thun M, Schnorr T, Lemen R (1992) A dose response analysis and quantitative assessment of lung cancer risk and occupational cadmium exposure. *Annals of Epidemiology*, **2**:177–194.

Steenland K, Beaumont J, Elliot L (1991a) Lung cancer in mild steel welders. *American Journal of Epidemiology*, **133**:220–229.

Steenland K, Burnett C, Ward E, Lalich N, Hurrell J (2003) Dying for work: the magnitude of US mortality from selected causes of death associated with occupation. *American Journal of Industrial Medicine*, **43**:461–482.

Steenland K, Loomis D, Shy C, Simonsen N (1996) Review of occupational lung carcinogens. *American Journal of Industrial Medicine*, **29**:474–490.

Steenland K, Sanderson W (2001) Lung cancer among industrial sand workers exposed to crystalline silica. *American Journal of Epidemiology*, **53**:695–703.

Steenland K, Sanderson W, Calvert GM (2001b) Kidney disease and arthritis in a cohort study of workers exposed to silica. *Epidemiology*, **12**:405–412.

Steenland K, Stayner L, Greife A et al. (1991b) Mortality among workers exposed to ethylene oxide. *New England Journal of Medicine*, **324**: 1402–1407.

Steenland K, Ward E (1991) Lung cancer incidence among patients with beryllium disease: A cohort mortality study. *Journal of the National Cancer Institute*, **83**:1380–1385.

Suter A (2000) Standards and regulations. In: *The noise manual, 5th edn*, Berger EH, Royster LH, Rozster JD, Driscoll DP, Layne M, eds. American Industrial Hygiene Association, Fairfax, VA.

Tay P (1996) Severe noise-induced deafness: a 10-year review of cases. *Singapore Medical Journal*, **37**:362–364.

Toren K (1996) Self-reported rate of occupational asthma in Sweden 1990–1992. *Occupational and Environmental Medicine*, **53**:757–761.

Toren K, Balder B, Brisman J et al. (1999) This risk of asthma. *European Respiratory Journal*, **13**:496–501.

Toroptsova NV, Benevolenskaya LI, Karyakin AN, Sergeev IL, Erdesz S (1995) "Cross/sectional" study of low backpain among workers at an industrial enterprise in Russia. *Spine*, **20**:328–332.

Tsai SP, Gilstrap EL, Cowles SR, Waddell LC, Ross CE (1992) Personal and job characteristics of musculoskeletal injuries in an industrial population. *Journal of Occupational Medicine*, **34**:606–612.

UN (2000) *International standard industrial classification of all economic activities (ISIC). Third revision*. United Nations publication (St/ESA/STAT/SER.M/4/Rev.3). United Nations, New York.

UN (2001) *World population prospects. The 2000 revision highlights*. Population Division. Department of Economic and Social Affairs, United Nations, New York.

USDHHS (1986) Perspectives in disease prevention and health promotion, leading work-related diseases and injuries—United States. *Morbidity and Mortality Weekly Report*, **35**(12). (Noise-induced Loss of Hearing. Reprinted in USDHHS 1991, *NIOSH Publications on Noise and Hearing*.)

USDOL OSHA (2000) *Docket for the Federal Register.* (Vol. 65, No. 220.) U.S. Department of Labor, Occupational Safety and Health Administration, Washington, DC.

USDOL OSHA (2002a) *Permissible exposure limits, codified at 29 CFR 1910.1000*. U.S. Department of Labor, Occupational Safety and Health Administration. Available at http://www.osha.gov/SLTC/pel/index.html.

USDOL OSHA (2002b) *Noise and hearing conservation*. U.S. Department of Labor, Occupational Safety and Health Administration. Available at http://www.osha-slc.gov/SLTC/noisehearingconservation/index.html

USEIA (2001) U.S. Energy Information Administration, U.S. Department of Energy, International Energy Database. Accessed January 2001 at www.eia.doe.gov/emeu/iea/coal.html.

Vaaranen V, Vasama M, Toikkanen J et al. (1994) *Occupational diseases in Finland l993*. Institute of Occupational Health, Helsinki.

Venables KM, Chang-Yeung M (1997) Occupational asthma. *The Lancet*, **349**:1465–1469.

Videman T, Nurminen M, Troup JDG (1990) Lumbar spine pathology in cadaveric material in relation to history of back pain, occupation, and physical loading. *Spine*, **15**:728–740.

Viikari-Juntura E (1997) The scientific basis for making guidelines and standards to prevent work-related musculoskeletal disorders. *Ergonomics*, **40**:1097–1117.

Viikari-Juntura E, Riihimäki H (1999) New avenues in research on musculoskeletal disorders. *Scandinavian Journal of Work, Environment and Health*, **25**:564–568.

Volinn E (1997) The epidemiology of low back pain in the rest of the world. A review of surveys in low-and middle-income countries. *Spine*, **22**:1747–1754.

Volinn E, Punnett L (2001) Point of view. *Spine*, **26**:1902–1903.

Volinn E, Spratt KF, Magnusson M, Pope MH (2001) The Boeing prospective study and beyond. *Spine*, **26**:1613–1622.

Waddell G (1991) Low back disability: a syndrome of Western civilization. *Neurosurgery Clinics of North America*, **2**:719–738.

Wagner GR, Wegman DH (1998) Occupational asthma: prevention by definition. *American Journal of Industrial Medicine*, **33**:427–429.

Waitzman N, Smith K (1999) *Unsound conditions: work-related hearing loss in construction, 1960–75*. The Center to Protect Worker's Rights. CPWR Publications, Washington, DC.

Ward E, Okan A, Ruder A, Fingerhut M, Steenland K (1992) A mortality study of workers at seven beryllium processing plants. *American Journal of Industrial Medicine*, **22**:885–904.

Westgaard RH, Jansen T (1992) Individual and work related factors associated with symptoms of musculoskeletal complaints: I. A quantitative registration system. *British Journal of Industrial Medicine*, **49**:147–153.

Westgaard RH, Winkel J (1997) Ergonomic intervention research for improved musculoskeletal health: a critical review. *International Journal of Industrial Ergonomics*, **20**:463–500.

WHO (1999) *International statistical classification of diseases and related health problems (ICD-10) in occupational health*. Karjalainen A, ed. Protection of the Human Environment, Occupational and Environmental Health Series, (WHO/SDE/OEH/99.11). World Health Organization, Geneva.

WHO (2001) *Electromagnetic fields and public health: extremely low frequency fields and cancer*. (Fact Sheet No. 263). World Health Organization, Geneva. Available at http://www.who.int/inf-fs/en/fact263.html.

WHO/FIOSH (2001) *Occupational exposure to noise: evaluation, prevention and control*. Goelzer B, Hansen CH, Sehrndt GA, eds. On behalf of WHO by the Federal Institute for Occupational Safety and Health (FIOSH), Dortmund.

Wikstrom B-O, Kjellberg A, Landstrom U (1994) Health effects of long-term occupational exposure to whole-body vibration: a review. *International Journal of Industrial Ergonomics*, **14**:273–292.

World Bank (2001) *World development indicators 2001*. Available at http://worldbank.com.

Xia Z, Courtney TK, Sorock GS, Zhu J, Fu H, Liang Y, Christiani DC (2000) Fatal occupational injuries in a new development area in the People's Republic of China. *Occupational and Environmental Medicine*, **42**: 917–922.

Xu X, Christiani DC, Dockery DW et al. (1992) Exposure-response relationships between occupational exposures and chronic respiratory illness: a community-based study. *American Review of Respiratory Disease*, **146**:413–418.

Yin SN, Li Q, Liu Y, Tian F, Du C, Jin C (1987) Occupational exposure to benzene in China. *British Journal of Industrial Medicine*, **44**:192–195.

Yip YB (2001) A study of work stress, patient handling activities and the risk of low back pain among nurses in Hong Kong. *Journal of Advanced Nursing*, **36**:794–804.

Zou Changqi, Gao Yun, Ma Qingyan (1997) Pneumoconiosis in China: current situation and countermeasures. *Mineral dusts and prevention of silicosis*, **4**(2) September 1997. Asian-Pacific Regional Network on Occupational Safety and Health Information (ASIA-OSH). Available at http://www.ilo.org/public/english/region/asro/.