表2 中皮腫の将来予測，増加傾向を扱った最近の論文

PMID	年	著者	掲載雑誌	国	論文タイトル	備考
16362942	2006	Murayama T, et al	Am J Ind Med	日本	日本における年齢コホートモデルに基づく胸膜中皮腫死亡の将来予測	
15668716	2005	Hodgson JT, et al	Br J Cancer	英国	英国における中皮腫死亡の予測	
16032742	2005	Dave SK & Beckett WS	Am J Ind Med	インド	インドにおける職業性石綿ばく露と予測できる石綿関連疾患	インドの石綿使用実態から定性的に将来増加を予測
15757980	2005	McElvenny DM, et al	Occup Med（Lond）	英国	英国の1968～2001年における中皮腫死亡	最近の死亡傾向を分析
15668716	2005	Hodgson JT, et al	Br J Cancer	英国	英国の2002～2050年の中皮腫死亡予測	
15570336	2004	Smartt P	N Z Med J	ニュージーランド	ニュージーランドの死亡・疾患・石綿肺症：石綿ばく露の遺産	最近の増加傾向がテーマ
15090665	2004	Weill H, et al	Occup Environ Med	米国	米国中皮腫死のトレンドの変化	
15031405	2004	Berry G, et al	Occup Environ Med	西オーストラリア	西豪 Wittenoom のクロシドライト鉱夫や精製所労働者における悪性胸膜腹膜中皮腫	累積数を報告
12499457	2003	Segura O, et al	Occup Environ Med	オランダ	オランダにおける胸膜中皮腫死亡予測のアップデート	
14674744	2003	Montanaro F, et al	Cancer Causes Control	ヨーロッパ	ヨーロッパの胸膜中皮腫罹患：増加傾向の減速の証拠	
11198540	2000	Kjellstrom T & Smartt P	N Z Med J	ニュージーランド	ニュージーランドの中皮腫罹患の増加：石綿がんの流行が開始	
10854503	2000	Banaei A, et al	Occup Environ Med	フランス	フランス男性における中皮腫死亡の将来動向	
10817376	2000	Kjaergaard J & Andersson M	Scand J Work Environ Health	デンマーク	デンマーク男性における悪性中皮腫の罹患率および将来予測人数	
10448315	1999	Jarvholm B, et al	Occup Environ Med	スウェーデン	スウェーデンの胸膜中皮腫：石綿使用量に応じた罹患の分析	最近の増加傾向がテーマ
9924453	1998	Ilg AG, et al	Occup Environ Med	フランス	フランスにおける中皮腫死亡の過去と将来の人数の推定	
9012593	1997	Price B	Am J Epidemiol	米国	米国中皮腫死亡の現在の傾向の分析	現在の傾向の分析がテーマ
9380136	1997	Burdorf A, et al	Ned Tijdschr Geneeskd	オランダ	過去の石綿ばく露による中皮腫罹患の増加	［オランダ語］
7776771	1995	Peto J, et al	Lancet	英国	英国における中皮腫死亡の増加は続く	
1663385	1991	Berry G	Br J Ind Med	オーストラリア	西豪 Wittenoom の元石綿労働者における中皮腫・肺がん・石綿肺症の予測	

かなり高くなると予測している．ただし，米国ではピークを越えたとする意見が強い．

中皮腫の将来予測に採用された統計モデルはほとんどが出生年コホート法を基本とし，暦年・年齢（階級）別の死亡を計数する2要因モデルを採用している．これとは別に将来の中皮腫発生を引き起こす要因として，すなわち独立のパラメーターとして，ばく露指標である石綿使用量を取り込んだ Banaei らによる分析手法もある．前述のように，集団単位で石綿使用と中皮腫の発生（死亡）の間には相関が見込まれる．そこから Banaei はフランス社会全体に着目し，そこで発生する中皮腫の将来予測を行ったわけである．我が国で石綿消費量との関連において将来予測を実施する場合や，白石綿と青石綿の影響を分けて解析する場合などは，類似の方法論を検討する必要がある．

最後に

本稿では，国段階の統計を概観し，マクロレベルで石綿および石綿関連疾患の実態描写を試みた．いわば国際比較疫学的な手法と言えるが，方法論的に確立されたものではない．国を解析単位とした生態学的研究は従来から適用例があるが，我々の研究室では「石綿関連疾患と歴史的石綿使用量の生態学的関連：グローバルな解析」と題する論文を報告した[11]．国段階の統計を使って 1960 年代の歴史的石綿使用量と直近の石綿関連疾患の死亡率の間の生態学的関連を評価し，強い相関があることを示した．さらに，回帰式に基づいて単位石綿使用量当たりのリスクの大きさを定量的に評価した．既存の知見に比べて国別データや石綿関連疾患の範囲を広げるとともにデータ精度も向上し，統計学的に妥当性の高い解析手法を適用している．その結果，男性中皮腫での歴史的石綿使用量の説明率は 74%（$p<0.001$）および単位石綿使用量当たり 2.4 倍のリスク上昇，男性石綿肺症での説明率 79%（$p<0.001$）および単位石綿使用量当たり 2.7 倍のリスク上昇，などの知見を得た．

近年，国際労働機関（ILO）と世界保健機関（WHO）は，石綿関連疾患の撲滅を両機関の共同事業の優先事項として位置づけている．現在 ILO は，石綿関連疾患の流行が顕在化し，かつ増大しつつあるとの認識から警戒感を表明している．その際，石綿関連疾患に特有な長期の潜伏期間に配慮する必要があることを，"iron grip of latency"（潜伏期間は鉄のように硬いグリップで握られている）と表現している．また 2006 年 6 月の国際労働総会は，世界規模で石綿の使用禁止を促進するための決議文を採択している．さらに，WHO は石綿関連疾患の撲滅を目指した政策文書を草稿中であることに付言して稿を終えたい．

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International Comparative Epidemiology of Asbestos Exposure and Asbestos Related Disorders

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Occupational Lung Diseases and the Mining Industry in Mongolia

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Mining production has accounted for around 50% of the gross industrial product in Mongolia since 1998. Dust-induced chronic bronchitis and pneumoconiosis currently account for the largest relative share (67.8%) of occupational diseases in Mongolia, and cases are increasing annually. In 1967–2004, medically diagnosed cases of occupational diseases in Mongolia numbered 7,600. Of these, 5,154 were confirmed cases of dust-induced chronic bronchitis and pneumoconiosis. Lung diseases and other mining-sector health risks pose major challenges for Mongolia. Gold and coal mines, both formal and informal, contribute significantly to economic growth, but the prevalence of occupational lung diseases is high and access to health care is limited. Rapid implementation of an effective national program of silicosis elimination and pneumoconiosis reduction is critical to ensure the health and safety of workers in this important sector of the Mongolian economy. *Key words*: Mongolia; coal mining; gold mining; informal sector; pneumoconiosis; dust-induced chronic bronchitis.

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Mongolia is a large, landlocked country located between Russia and China in the northern part of Central Asia. The population of Mongolia is approximately 2.6 million, of which there are some 930,000 employed workers, with 60,000 in the mining sector and 3,500 working in power plants.[1] Mongolia is a developing country and had a per capita gross domestic product (GDP) of approximately USD 2,056 in 2005.[2,3] According to Mongolian government statistics, mining production has contributed around 50% of the gross industrial product since 1998.[4] Foreign investment accounts for approximately 30% of the total investment in the mining sector. Since 2000, the mining sector has been a major driver of economic growth in Mongolia, accounting for 32.6% of GDP and 65.6% of exports.[5–7] Mongolia is rich in mineral resources, including coal, gold, copper, molybdenum, fluorspar, iron, limestone, zinc, salt, silver, tin, tungsten, uranium, and clays. Currently, the largest mining sectors in terms of number of mines are gold (63.5%), coal (14%), fluorspar (22%), and copper (0.5%) (Table 1).

GOLD MINING

Since the Mongolian government implemented the "Gold Program" in 1992, annual gold extraction has increased dramatically, hitting a peak of 12.0 tons in 2001. Two types of mining currently exist in Mongolia: formal (with a government licence to use mining land) and informal (without a government license). In 2004, 127 formal gold mines employing approximately 20,000 workers were registered.[4–5] Informal placer gold mining operations have grown in recent decades, although there are no statistics currently available on their number. It is estimated that 100,000 workers are currently engaged in informal gold mining, 73% of whom work in placer mining operations (Figure 1). Placer mining is an open-pit or open-cast form of mining by which certain valuable minerals are extracted from the surface of the earth without tunnelling.[8] Placer gold mining results in many small holes and tunnels dug in riverbanks.[9] The number of informal mines has increased rapidly since 2000 due to rising urban and rural unemployment/underemployment, lack of a legal framework, a decline in agriculture, and the loss of livestock during natural disasters.[10]

UNICEF estimated that 36.6% of children in Mongolia aged 5 to 14 years were working in 2000.[11] Some of them work in informal coal mining, either in the mines or scavenging for coal outside as well as in informal gold mining.* It is estimated that 10–12% of informal gold miners are children between the ages of 12 and 16 years.[9,12] During the school summer vacation

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*Most mines in Nalaikh were closed almost a decade ago, but since many of the openings still exist coal mining continues. For a discussion of the conditions children face working in the sector, see the Mongolian Women's Federation Study, commissioned by ILO-IPEC in 2000, as cited in references 22 and 23.

TABLE 1 Output of Mining Products and Number of Mining Industries in Mongolia, 2004

	Production Volume in Metric Tons	Number in Licensed Mining Industries	Percentage of Mining Industries
Gold (chemically pure)	0.0186×10^3	127	63.5
Coal	$6,800 \times 10^3$	28	14.0
Fluorspar		44	22.0
Fluor flotation concentrate	104.8×10^3		
Fluor metallurgic concentrate	206×10^3		
Copper		1*	0.5
Copper in concentrate	130×10^3		
Cathode copper	2.37×10^3		
Molybdenum in concentrate	1.14×10^3	1*	

*The same industry produces copper and molybdenum.

(June–August), this is thought to rise to 35%.[9,12] Some children drop out of school after their primary years to stay with working parents who have migrated from other provinces, or to engage in full-time work. Child miners in Mongolia are not formally registered with the relevant local administration and thus do not have access to health care and education.

In 1999, the government developed a list of hazardous employment activities prohibited for minors,† Article 16 of the Constitution of Mongolia prohibits forced labor.[13] The Labor Inspection Department under the State Professional Inspection Agency, which is responsible for enforcing child labor, now collects data on children engaged in hazardous work. However, there are only a small number of inspectors, and labor inspectors rarely inspect medium-sized and small enterprises and mines.[14]

Informal gold miners, including children, are exposed to mercury through amalgamation, burning, and storage at home. In 2003, the Public Health Institute of Mongolia evaluated the health status of children exposed to mercury in informal mines and concluded that 24.2% of these children showed signs of chronic mercury intoxication, indicated by respiratory problems, tremor, and depression.[12] Other health risks faced by informal gold miners, both children and adults, include unsafe working conditions and silica dust exposure. Most informal mines are simple hand-dug holes (1 m × 2 m) (Figure 2). The collected soil is carried to a nearby river for washing (Figures 1 and 2) and is later extracted with mercury. Workers are thus exposed to mercury vapor. Additionally, in the fall (September–November) and spring (March–May), most of these rivers are frozen and thus present the additional risks of frostbite and/or hypothermia during processing. Currently, there are no data on the prevalence of occupational lung diseases in the informal gold mining sector, nor are work-related illnesses or industrial accidents officially reported to local or health authorities.

A survey of small-scale mining in Mongolia was financed by Danish trust funds and carried out by a consultant from the Geological Survey of Denmark and Greenland (GEUS) in 2004. The survey identified a lack of knowledge of recycling and safe mercury handling practices, as well as a lack of knowledge of how to diagnose mercury poisoning among local medical doctors. In the scale of the survey, one training course was held in the Bornuur informal and formal mining areas of Mongolia. This course was highly successful, and several mercury-recycling devices (retorts) were given to the participants.[16] There is pending legislation in Mongolia dealing with small-scale mining. There are currently several versions of a draft mining law in circulation.[9]

Figure 1—Informal gold miners in quest of gold washing soil in a puddle near a frozen river, December 2004. Zaamar, Selenge Province, Mongolia

†Prohibited types of employment include underground work, mining, exploration and mapping, mineral processing, and energy, ceramic, and glass production. See list of Prohibited Jobs for Minors/people.No.A/204, (August 13, 1999).

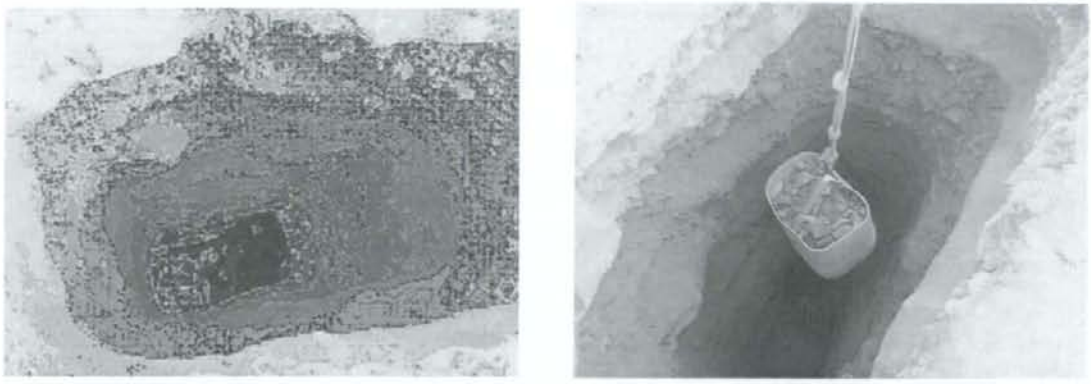

Figure 2—Hand-dug hole in an informal gold mining area, November 2006, Uvurkhangai Province, Mongolia.

Occupational health and safety risks at some of the formal mines may be fewer than at the informal mines. Some large gold mines use automated excavating machinery, while some medium- and small-scale formal mines utilize bulldozers, excavators, and water-gun methods for gold refining. Formal gold mines are required to implement occupational health and safety measures, including conducting annual worker health examinations, retaining a full-time occupational safety officer or hygienist, and providing personal protective equipment (PPE) that conforms to occupational safety and health standards.

COAL MINING SECTOR

In 2004, 82 coal deposits were identified in Mongolia, with mining operations at 28 of them.[4] Coal is the oldest mining sector in the country, with the first coal mine ("Nalaikh") established in 1924.[15] This state-owned mine produced 800,000 metric tons of coal annually during the 1980s, but was closed in 1992 because of outdated technology and poor working conditions.[6] Despite the closure, a number of small-scale and informal mines are currently operating at the Nalaikh site (Figures 3–9).

Most Mongolia-mined coal is used in central and local electric power plants and in household stoves for heating and cooking. Coal is the principal fuel in cities.[16] In the capital city of Ulaanbaatar, there were three central power plants and more than 250 local electric power plants (coal-burning chimneys) operating in 2005. In the same year, 5 million tons of coal were burned in the power stations, 400,000 tons in the chimneys, and 200,000 tons in the 80,000 gers (traditional nomadic homes) located in Ulaanbaatar.[16]

There are three types of coal mines in Mongolia: company-owned mines (Figure 3), formal (licensed) self-employed mines (Figure 4), and informal (unlicensed or illegal) mines (Figure 5). Approximately 10,000 workers were engaged in company-owned coal

Figure 3—Small-scale company-owned coal mine, December, 2004, Nalaikh district, Ulaanbaatar, Mongolia.

Figure 4—Formal, self-employed coal mine, December 2004), Nalaikh district, Ulaanbaatar, Mongolia.

TABLE 2 Dust-Induced Occupational Diseases in Mongolia, 1968–2004

	1968–1979	1980s	1990s	2000–04	Total
Pneumoconiosis	636	204	72	8	920
Silicosis	286	112	38	5	441
Welder's pneumoconiosis	9	11	2	1	23
Anthraco-silicosis	330	76	30	2	438
Other types*	11	5	2	0	18
Dust-induced chronic bronchitis	210	952	2,090	982	4,234
Total	846	1,156	2,162	990	5,154

*Mostly pneumoconiosis caused by organic dust.

mines, and 2,000 in informal coal mines, in 2004.[9,17] Lkhasuren and Takahashi (2004) have observed that some company-owned coal mines have implemented modest occupational safety and health precautionary measures, such as providing wooden ceilings and pillars to prevent collapse, local exhaust ducts, electricity-powered trolleys, and lighting. However, the use of PPE is limited, with few workers wearing helmets and none wearing respirators. Self-employed formal and illegal informal mines employ few or no physical barriers to collapse. Since these mines lack electricity, workers typically use bulldozers to pull up trolleys. Further, informal mines typically have only enough space to work in a crawling or squatting position. Typically, PPE (helmets or respirators) is not used in informal mines.

OCCUPATIONAL LUNG DISEASES

Until the 1990s, most Mongolian industries (including the mining industry) were owned by the State, and occupational health and safety measures were the responsibility of the Mongolian Trade Union. The Trade Union was founded by the government and is financed by membership tax. Since 1990, occupational health and safety have been the responsibility of the Ministry of Labor and Social Welfare. As the number of privately-owned companies grew in the 1990s,[18] the labor law stated that the company owners have responsibility for financing and implementing occupational health and safety measures for their workers.[19]

The Center for Occupational Disease (COD) was established within the Department of Social Insurance of the Ministry of Labor and Social Welfare in 1967. At that time, silicosis was the only officially recognized occupational lung disease. Since then, the COD has been the center of the country's efforts in the identification, notification, and treatment of occupational diseases, and has organized pre-employment and periodic health examinations of employees nationwide. To date, COD physicians have focused their efforts on annual visits to the large formal mines (i.e., those with more than 500 workers) to conduct periodic occupational health examinations.

The COD estimates that the cumulative number of cases of occupational diseases during 1967–2004 was 7,600.[21,22] Of these, 5,154 (67.8%) were diagnosed as dust-induced chronic bronchitis and pneumoconiosis, and their incidences continue to increase (Table 2). During the COD's 2003 annual health examinations, 521 new occupational cases were registered, of which

Figure 5. Informal coal mine, December 2004, Nalaikh district, Ulaanbaatar, Mongolia.

Figure 6—Inside a small-scale coal mine, December 2005. Nalaikh district, Ulaanbaatar, Mongolia.

Figure 7—Shaft of a formal, self-employed coal mine, December 2004, Nalaikh district, Ulaanbaatar, Mongolia.

Figure 8—Informal miners pulling out loaded coal with a tractor, December 2005, Nalaikh district, Ulaanbaatar, Mongolia.

251 were dust-related occupational diseases, including dust-induced chronic bronchitis and pneumoconiosis. Of the nearly 1,000 cases of pneumoconiosis in Mongolia in 2004, most were anthraco-silicosis (47.6%) and silicosis (47.9%). The diagnosis of these occupational lung diseases in Mongolia depends on evidence of occupational history, radiographic findings, and results of pulmonary function tests. The radiographic diagnostic criteria for pneumoconiosis and dust-induced chronic bronchitis[22] were based on the Russian system, vis-à-vis International Classification of Radiographs of Pneumoconiosis by the International Labor Organization (ILO), and since 2002 the COD has used standard ILO films. However, the quality of chest x-rays is still poor. Most patients with pneumoconiosis are treated by rehabilitation, palliative care, and symptomatic treatment in the outpatient departments of their home provinces and industries.

Since 2000, 82% of the diagnosed cases of occupational lung disease in Mongolia have come from coal mines and power plants; a further 11% came from construction and industries producing construction materials, 3% from manufacturing, 0.7% from social services, and 0.8% from agriculture.[21] Moreover, it is likely that many cases go unreported, especially in the small-scale, informal mining sector not included in the official reporting system.

In 2004, the School of Public Health of the Health Sciences University of Mongolia initiated activities as part of the program on global elimination of silicosis.[23] The National Program on Silicosis Elimination and Pneumoconiosis Reduction is currently under development. In the framework of the national and global programs, there is a need to improve the COD's capacity to provide confirmatory diagnoses of pneumoconiosis and dust-related diseases. In particular, training is needed to improve the quality of chest x-ray films, the reading of films, and statistical and epidemiologic data analysis. The COD adopted ILO standard films for diagnosis and a revised classification of occupational diseases in Mongolia in 2004.[24]

A national database on pneumoconiosis and dust-related diseases and dust measurements is needed so that effective interventions can be designed and implemented. Such a database would facilitate prioritization of industry sectors, job types, and levels of dust exposure, in addition to providing baseline data for evaluating the adequacy of control measures. Within the National Program on Silicosis Elimination and Pneumoconiosis Reduction framework, morbidity and mortality data related to pneumoconiosis (silicosis, coal-worker's pneumoconiosis, asbestosis, etc.), as well as other dust-related disorders, including cancers (lung cancer and mesothelioma), will be collected at all levels (factory, provincial "aimag," and national). Acknowledgement of successful models through official awards or certifications could facilitate recognition of the program.

Figure 9—Loading coal for commercial use, December 2005, Nalaikh district, Ulaanbaatar, Mongolia.

TABLE 3 Total Dust Concentrations in Coal and Gold Mines and Power Plants in Mongolia

	Geometric Mean Concentration (mg/m³)*					
	1980s	1990s	2000	2001	2002	2003
Coal mines						
Company-owned	68.3	54.8	50.2	44.2	42.4	34.6
Formal but self-employed	75.8	60.8	57.2	49.9	45.3	42.5
Informal	n.a.†	n.a.	—†	40.3	48.7	44.2
Gold mines	n.a.	—	—	15.7	10.9	10.6
Power plants						
II (built 1924)	102.0	93.4	89.3	80.4	52.0	34.4
III (built 1968)	75.2	52.3	55.1	40.2	25.3	22.1
IV (built 1978)	72.3	56.1	53.7	41.3	21.8	22.2
Mongolian MALs:						
Coal dust (silica content 10% or less)					6–10 mg/m³	
Gold mine dust (silica content 10% or less)					10 mg/m³	
Total dust (silica content 10-70%)					2 mg/m³	
Total dust (silica content more that 70%)					1 mg/m³	

*135 samples collected from the mines and power plants.
†Not applicable because informal coal mines did not exist at that time.
‡No data available.

DUST MEASUREMENTS

Airborne total dust, respirable dust, and silica dust concentrations are critical indicators of the safety of the work environment in Mongolian mining industries. Mongolian governmental inspectors and researchers of the School of Public Health took samples from 15 coal mines and three power plants and analyzed these for total dust concentration in 2001–2003. Table 3 compares these recent measurements with total dust concentrations during 1980s and 1990s computed using data from the Report of Occupational Health and Safety, the State Professional Inspection Agency.[20] Geometric mean total dust concentrations ranged from 68.3 to 102.0 mg/m³ in the 1980s and from 54.8 to 93.2 mg/m³ in the 1990s in 135 samples.[27–28] Table 3 shows that total dust concentrations were highest (102 mg/m³) in coal mines and power plants during the 1980s and 1990s. Although dust concentrations in the 2001–2003 samples still exceeded the Mongolian maximum allowable limits (MALs), it is likely that the decline in total dust levels in recent years reflects improvements in technology and exposure control.

Analytical methods for measuring respirable dust and silica dust, as well as occupational dust standards, were published in Mongolia in 2004.[30] However, enforcement of these standards has been limited because of a shortage of resources for sampling and laboratory equipment. A single published study reported measured silica contents in coal mine dust in 2004.[25–26] In this study, the silica in coal mine dust was 15.3–17.5 mg/m³ and that in respirable dust was 7.1mg/m³.[25]

Air-monitoring data conducted by provincial (aimag) or industrial hygienists are collected by the Department of Professional Inspection Agency at the provincial level and by the State Professional Inspection Agency at the national level. At present, there is a need to better coordinate data sharing among the responsible agencies, as well as to ensure the dissemination of results to consumers and decision makers.

CONCLUSION

Occupational lung diseases and other mining-sector health risks pose major challenges for Mongolia. On the one hand, the growing number of gold and coal mines, both formal and informal, contributes significantly to economic growth and development. On the other hand, occupational health and safety problems continue to plague the mining industries. The prevalence of occupational lung diseases remains high, and access to health care, especially for workers in informal mines, is limited. Rapid implementation of an effective National Silicosis Elimination and Pneumoconiosis Reduction Program is critical to ensure the health and safety of workers in this important sector of the Mongolian economy.

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Mortality of Iron–Steel Workers in Anshan, China:

A Retrospective Cohort Study

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Foundry workers have increased mortality and morbidity risks from numerous causes, including various cancers. A retrospective Chinese iron–steel cohort study was conducted to examine the mortality effects of exposure to foundry work. Standardized mortality ratios (SMRs) and standardized rate ratios (SRRs) were calculated to evaluate mortality risks among male workers with exposure to 15 hazardous factors, adjusting for confounders. During 14 years of follow-up, 13,363 of 121,846 male workers died. SMR analysis showed a healthy-worker effect in comparison with the general population. SRR analysis showed increased risks for all causes, all neoplasms, and others among the exposed workers compared with non-exposed blue-collar workers. Combined exposure to polycyclic aromatic hydrocarbons and two or more dusts increased the risks of lung cancer (SRR = 654; 95% CI: 113–3,780) and other malignancies. Foundry work has adverse health effects, including carcinogenic risks. *Key words*: iron and steel founding; cohort study; carcinogenic risk; job exposure matrix; lifetime job.

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Epidemiologic studies of iron–steel workers in various countries have shown a range of adverse health effects, such as respiratory cancer, non-malignant diseases, and cardiovascular diseases.[1–7] According to the monograph by the International Agency for Research on Cancer (IARC), occupational exposure to iron–steel founding was upgraded in 1987 to "sufficiently carcinogenic to humans (Group 1)" from "probably carcinogenic (Group 2A)."[8]

Of the numerous iron–steel plants in China, the Anshan Iron and Steel Company ("Angang") is one of the largest. It is capable of annually turning out 10 million tons of pig iron, 10 million tons of steel, and 9.5 million tons of rolled steel products.[9] Previous epidemiologic studies conducted at Angang showed excess risks of accidents, total cancers, lung cancer, stomach cancer, and colon cancer.[10] Dose–response relationships were shown between exposures to dust and benzo(a)pyrene and mortality of lung cancer, stomach cancer, and esophageal cancer.[11,12] Thus, such adverse health effects among the workers in Angang needed to be investigated by an epidemiologic study, ideally a cohort study.

This study evaluated the mortality risk of male workers using standardized mortality ratios (SMRs) and standardized rate ratios (SRRs), adjusting for confounding factors.

MATERIALS AND METHODS

Setting

Angang is located in the city of Anshan in northeastern China. The company covers a land area of 176 km^2, including 129.19 km^2 for industrial use, and still has 130,960 active workers and staff members.[9]

Definition of Cohort

Of the workers who had entered Angang before January 1, 1980, those who were alive, regardless of whether

TABLE 1 Vital Status among Chinese Iron–Steel Workers (1980–1993, Males Only)*

	No. (%)
Total	121,846 (100)
Living	108,483 (89.0)
Active employees	67,993 (55.8)
Retired	40,054 (32.9)
Left	436 (0.4)
Deceased	13,363 (11.0)
Before retirement	3,145 (2.6)†
After retirement	10,218 (8.4)†

*Vital status of the workers as of December 31, 1993.
†All but one worker who left the company in 1982 and died in 1988 were actively employed when they died.
†Of these, 5,074 had retired before January 1, 1980.

they were currently actively employed or not, on January 1, 1980, with at least six months of employment were included in the cohort. The follow-up period was from January 1, 1980, to December 31, 1993, and was stopped when a member died or left the company. A computerized personnel data file was established in 1993 by Angang for all the employees for personnel and salary management. For active employees, information regarding date of hire, current factory, current workshop/department, current job, and time spent at current job was collected. For inactive employees, retirement date and the longest job title of his/her experiences were recorded. For all employees who had left the company during 1980–1993, retirement date and information about the longest held job were obtained from the personnel department.

Definition of Reference Group

The ambient pollution levels of total suspended particles (TSP) in Angang and in the residential area of Anshan in 1991 were 0.68 and 0.32, respectively, i.e., 4.5 and 2.1 times higher than the national standard of 0.15 mg/m^3.[13]

Since the situation with a high level of background air pollution may mask the effect of occupational exposure, we designated the general population of Anshan as the external reference group.

We also chose the nonexposed blue-collar workers in Angang as the internal reference group. That was because various substances ubiquitously existed in the work environment in Angang, so that the nonexposed blue-collar workers were more comparable to the exposed blue-collar workers than to the white-collar workers.

Confirmation of Vital Status

All deaths during 1980–1993 were collected from the death registry of the company, which was established in 1980. The causes of death listed on individual death certificates were reported by the five hospitals attached to the company, where medical services were provided to all employees almost free of charge. The municipal death registry, established in Anshan City in 1972, collected information on death certificates received from medical practitioners. All causes of death were classified according to the ninth revision of the International Classification of Diseases (ICD-9) in both registries by medical doctors responsible. When the causes of death recorded in the two registries differed, the cause of death was verified by consulting the municipal registry and the company registry to confirm accuracy.

Exposure Assessment and Procedure of Job Exposure Matrix Establishment

A workshop, job title, and exposure matrix method (job exposure matrix, JEM) was used for occupational exposure assessment. The subjects in the cohort worked in a total of 836 workshops of about 90 factories or facilities at Angang. Up to 1,583 job titles were identified according to the coding manual of the personnel data file issued by industrial hygienists.[14] Occupational exposures to 15 agents were determined: silica dust, iron dust, cement dust, welding dust, asbestos, coal dust, wood dust, grinding dust, other dusts, high temperature (heat), carbon monoxide (CO), polycyclic aromatic hydrocarbons (PAHs), oil mist, acid mist, and benzene.

Exposure to each of these 15 factors was evaluated as a dichotomous variable (yes or no) by linking the JEM with *only one job* for each worker: the longest job title was used for the retired, the deceased, and the employees who had left the company, and the current job title was used for the active employees.

Statistical Analyses

First, SMRs and the corresponding 95% confidence intervals (CIs)[15] were calculated for the total cohort. Person-years at risk were accumulated for the cohort in the follow-up period. Expected numbers of deaths were calculated by multiplying the person-years with gender-, age-, calendar-year–, and cause-specific death rates of the general population of Anshan City, 1980–1993. SMRs were also calculated by job types and exposure status, i.e., white-collar, nonexposed blue-collar, and exposed blue-collar, to adjust for the healthy-worker effect (HWE).[16,17] A test for linear trend of SMR was conducted to evaluate dose–response relationships.[15] Although some white-collar workers (n = 2,260, 7% of males followed up) were secondarily exposed to some factors, they were analyzed as nonexposed white-collar workers.

Second, SRRs and the corresponding 95% CIs[18] were calculated by exposure status for the 15 factors individually with the nonexposed blue-collar workers as the internal reference group. When SRRs were calcu-

TABLE 2 SMRs for Mortality of Diseases by Job Types and Exposure Status among Chinese Iron-steel Workers (Male, 1980–1993)

	All Job Types (n = 121,846)		White Collar (n = 32,664)		Non-exposed Blue Collar (n = 39,048)		Exposed Blue Collar (n = 50,134)		
Cause of Death (ICD-9)	Obs. (%)	SMR (95% CI)	Obs. (%)	SMR (95% CI)	Obs. (%)	SMR (95% CI)	Obs. (%)	SMR (95% CI)	p for Trend¶
Infectious (1–139)	377 (2.8)	69 (62–76)	58	45 (34–58)	128	71 (59–84)	191	80 (69–92)	<.001
Tuberculosis (11–12)	271 (71.9)[a]	73 (64–82)	36	43 (30–58)	85	69 (54–84)	150	91 (77–106)	<.001
Hepatitis (70)	52 (13.8)[a]	55 (40–71)	17	65 (38–100)	19	63 (38–94)	16	41 (23–63)	NS
Neoplasms (140–239)	4,141 (31.0)	75 (72–77)	743	56 (52–60)	1,395	78 (73–82)	2,003	82 (78–86)	<.001
Stomach cancer (151)	628 (15.2)[b]	75 (69–81)	82	44 (34–54)	225	81 (72–92)	321	86 (77–96)	<.001
Liver cancer (155)	795 (19.2)[b]	77 (72–82)	154	58 (49–68)	265	81 (72–92)	376	85 (76–94)	<.001
Lung cancer (162)	1,522 (36.9)[b]	85 (81–89)	265	62 (54–70)	507	88 (80–96)	750	96 (88–102)	<.001
Cerebrovascular (430–439)	3,577 (26.8)	68 (66–70)	500	45 (41–49)	1,259	70 (66–74)	1,818	77 (73–81)	<.001
Intracranial hemorrhage (431–432)	1,696 (47.4)[c]	68 (64–72)	265	47 (41–53)	592	72 (66–78)	839	77 (71–82)	<.001
Cerebral infarction (434)	1480 (41.4)[c]	93 (88–99)	176	59 (50–68)	525	93 (85–101)	779	108 (100–116)	<.001
Circulatory system (390–429, 440–459)	1,588 (11.9)	62 (59–65)	304	54 (48–60)	542	62 (57–68)	742	66 (61–71)	<.01
AMI† (410)	651 (41.0)[d]	60 (55–65)	154	60 (51–70)	205	57 (49–65)	292	62 (54–69)	NS
Ischemic heart disease other than AMI (411–412)	397 (25.0)[d]	54 (49–60)	74	50 (39–62)	134	52 (43–62)	189	58 (50–67)	NS
Respiratory system (460–519)	1,615 (12.1)	56 (53–59)	180	33 (28–39)	557	54 (49–58)	878	67 (63–72)	<.001
COPD‡ (491–492)	1,126 (69.7)[e]	49 (46–53)	133	31 (26–37)	422	52 (46–57)	571	55 (51–60)	<.001
Pneumoconiosis (502–509)	310 (19.2)[e]	157 (139–175)	18	43 (25–65)	66	100 (77–125)	226	252 (220–286)	<.001
Injuries (800–959)	676 (5.1)	76 (71–82)	82	37 (29–45)	234	81 (71–92)	360	97 (87–107)	<.001
Other	1,389 (10.4)	66 (62–69)	227	46 (40–53)	516	72 (65–78)	646	71 (65–76)	<.001
All causes	13,363 (100)	67 (66–68)	2,094	48 (45–50)	4,631	69 (67–72)	6,638	76 (74–78)	<.001

[a]Against total number of infectious diseases.
[b]Against total number of neolasms.
[c]Against total number of cerebrovascular diseases.
[d]Against total number of circulatory system diseases.
[e]Against total number of respiratory system diseases.

†Acute myocardial infarction.
‡Chronic obstructive pulmonary diseases including chronic bronchits (ICD-9: 491) and pulmnonary emphysema (ICD-9: 492).
¶Test for trend was done only for disease categories with highlited background. NS: not significant.

TABLE 3 SRRs for Mortality of Diseases by Exposure Factors among Chinese Iron-Steel Workers (Male, 1980–1993)

	Silica Exposure (n = 9,542)		Iron Exposure (n = 21,175)		Welding (n = 2,818)	
Cause of Death (ICD-9)	Obs.	SRR (95% CI)	Obs.	SRR (95% CI)	Obs.	SRR (95% CI)
Infectious (1–139)	3	37 (11–121)	36	162 (130–201)	8	193 (97–379)
Tuberculosis (11–12)	3	55 (23–129)	29	195 (137–278)	6	226 (80–629)
Hepatitis (70)	0	—	4	33 (0–18,278)	0	226 (80–629)
Neoplasms (140–239)	27	36 (28–45)	352	147 (139–155)	68	145 (130–161)
Esophagus (150)	1	27 (1–450)	24	228 (139–155)	3	118 (91–151)
Stomach cancer (151)	7	75 (59–94)	56	153 (132–177)	10	129 (104–159)
Colon, sigmoid, and rectum (153–154)	3	69 (34–138)	15	151 (114–200)	3	69 (34–480)
Liver cancer (155)	6	33 (18–61)	64	130 (120–141)	11	92 (86–97)
Lung cancer (162)	8	26 (15–43)	133	154 (139–169)	26	169 (131–218)
Bladder (188)	0	—	4	103 (97–110)	1	161 (61–1,599)
Brain (191–192)	0	—	6	100 (99–101)	0	—
Leukemia (204–208)	0	—	14	156 (112–217)	3	178 (28–1,117)
Cerebrovascular (430–439)	24	36 (28–45)	304	147 (139–155)	52	128 (119–140)
Intracranial (431–432)	11	33 (22–48)	136	135 (127–144)	31	124 (113–137)
Cerebral infarction (434)	11	32 (21–48)	128	155 (140–171)	19	159 (122–205)
Circulatory system (390–429, 440–459)	15	49 (37–64)	145	158 (143–175	20	131 (114–150)
AMI† (410)	4	31 (11–79)	39	108 (105–111)	12	204 (114–363)
Ischemic heart disease other than AMI (411–412)	2	37 (7–177)	48	218 (157–301)	3	95 (86–103)
Respiratory system (460–519)	27	74 (66–82)	145	167 (149–187)	18	115 (106–125)
COPD‡ (491–492)	6	22 (11–44)	97	148 (133–163)	15	122 (107–139)
Pneumoconiosis (502–509)	20	444 (123–1,600)	35	335 (169–661)	2	104 (96–111)
Injuries (800–959)	4	41 (18–91)	61	146 (129–166)	15	150 (117–193)
Others	10	49 (35–69)	115	128 (121–135)	20	112 (105–119)
All causes	110	42 (38–47)	1,158	149 (144–154)	201	133 (127–140)

†Acutemyocardial infarction.
‡Chronic obstructive pulmonary diseases including chronic bronchitis (ICD-9: 491) and pulmonary emphysema (ICD-9:492).

lated, age on January 1, 1980 (39 or younger, 40–49, 50–59, and 60 or older), and period of employment (1950 or before, 1951–1960, 1961–1970, and 1971 or later) were adjusted as potential confounders. Furthermore, to investigate the combined effects of simultaneous exposures to PAHs and dusts, SRRs for all causes, all neoplasms, and selected cancers (lung, liver, stomach, and others) were calculated according to exposure status to PAHs and dusts. Exposure status to dusts was expressed by status (yes/no) and the numbers of exposures to various dusts, i.e., silica, iron dust, welding dust, coal dust, cement dust, grinding dust, asbestos, wood dust, and other dusts. Statistical analyses were performed with SAS version 8. In this paper, analyses of mortality risk cover only male workers.

RESULTS

A total of 149,887 workers were initially identified from the personnel files. Of them, 3,351 were inactive workers, of whom 526 were followed up with complete information. Vital status and complete information were confirmed for 147,062 subjects, 98.1% of the total cohort. The remaining 2,825 workers, or 1.9%, had left the company or had incomplete information. Therefore, analyses were restricted to 147,062 subjects, 121,846 males and 25,216 females. During the follow-up period of 1980–1993, 13,363 male workers died (Table 1). The observed person-years are 1,619,005 for males.

Of the male workers, 21,175 (17.4%) were exposed to iron dust, 20,729 (17.0%) to heat, 9,542 (7.8%) to silica, and 5,245 (4.3%) to PAHs. Some workers had exposures to three or more of the factors.

Table 2 shows the SMRs for the male mortality of all causes, the six main disease categories, i.e., infectious diseases, neoplasms, cerebrovascular diseases, circulatory system diseases, nonmalignant respiratory diseases, and injuries, and selected subcategories of diseases. The most frequent deaths were from neoplasms, 4,141 (31%), followed by cerebrovascular diseases, 3,577 (27%). Despite significantly lower SMRs for all causes, including the six disease categories, compared with the general population, significantly higher SMR of 157 (95% CI: 139–175) was shown for pneumoconiosis.

Table 2 also shows an increasing trend of SMRs for all causes, all six disease categories, and almost all selected subcategories by job types and exposure status with statistical significance, when the comparison was between exposed and nonexposed workers. For all

TABLE 3 (continued from preceding page)

Coal Exposure (n = 3,536)		Cement Exposure (n = 3,635)		Grinding (n = 401)		Asbestos Exposure (n = 4,269)	
Obs.	SRR (95% CI)	Obs.	SRR (95% CI)	Obs.	SRR (95% CI)	Obs.	SRR (95% CI)
9	127 (102–158)	26	198 (135–288)	0	—	21	164 (123–218)
7	137 (92–202)	20	226 (133–386)	0	—	17	196 (124–309)
1	118 (60–229)	1	50 (0–4,852)	0	—	3	173 (53–560)
68	99 (98–100)	183	132 (126–140)	3	19 (5–60)	204	140 (132–148)
5	151 (83–271)	19	252 (129–490)	0	—	14	211 (119–374)
11	93 (88–97)	19	100 (100–101)	0	—	43	192 (146–252)
2	71 (39–128)	6	72 (51–101)	0	—	11	163 (106–502)
12	101 (100–101)	36	151 (128–179)	2	42 (7–228)	38	133 (119–149)
21	86 (81–92)	71	146 (131–162)	1	30 (1–669)	79	147 (132–164)
1	74 (27–195)	3	111 (94–131)	0	—	4	166 (59–467)
4	272 (32–2,275)	4	228 (16–3,197)	0	—	1	27 (1–659)
0	—	5	103 (98–107)	0	—	2	37 (3–358)
75	117 (112–122)	203	153 (142–166)	7	40 (25–61)	180	140 (131–149)
37	119 (111–127)	82	142 (129–155)	2	17 (3–80)	92	152 (136–170)
29	112 (106–116)	97	163 (143–185)	5	76 (63–92)	69	128 (119–137)
25	87 (82–92)	63	113 (109–118)	1	9 (0–154)	81	146 (131–163)
10	101 (100–102)	28	131 (116–148)	0	—	42	203 (149–276)
7	87 (76–100)	14	108 (103–113)	1	38 (4–303)	20	137 (113–165)
25	86 (81–92)	110	188 (160–222)	2	20 (3–104)	86	155 (137–135)
17	76 (67–86)	67	149 (132–168)	2	27 (5–124)	62	148 (130–167)
1	32 (1–654)	37	553 (180–1,700)	0	—	15	228 (122–426)
12	104 (101–107)	30	149 (124–179)	0	—	33	131 (117–148)
22	78 (70–86)	57	124 (116–132)	2	21 (1–228)	69	134 (123–306)
236	100 (99–100)	672	144 (139–150)	15	22 15–33)	674	142 (137–147)

continued on next page

causes, the SMRs (95% CI) were 48 (45–50) in white-collar workers, 69 (67–72) in nonexposed blue-collar workers, and 76 (74–78) in exposed blue-collar workers. For all neoplasms, they were 56 (52–60), 78 (73–82), and 82 (78–86), respectively. For pneumoconiosis, a clear trend was seen, i.e., 43 (25–65), significantly lower than expected, in white-collar workers, 100 (77–125) in nonexposed blue-collar workers, and 252 (220–282), significantly higher than expected, in exposed blue-collar workers.

Table 3 shows SRRs of the mortality for all causes, the six main disease categories, and selected disease subcategories by exposure to the respective hazardous factors. Compared with the nonexposed blue-collar workers (internal reference), SRRs for all causes were significantly higher in those with exposure to iron dust (SRR = 149; 95% CI: 144–154), welding dust (133; 127–140), cement dust (144; 139–150), asbestos (142; 137–147), heat (126; 124–128), PAHs (176; 166–186), acid mist (199; 171–232), and benzene (112; 109–115). Among those with exposures to these factors, the SRRs for a number of the main disease categories and subcategories were also significantly elevated. Those with exposure to silica showed increased SRR only for pneumoconiosis, 444 (95% CI: 123–1,600). Among those with exposures to iron dust, cement dust, and heat, SRRs exceeded 300 with statistical significance.

In Table 3, the SRRs for several selected sites of malignant neoplasms are shown. Significantly elevated SRRs for sites including lung, liver, stomach, and esophagus were found for exposures to many factors such as iron dust, cement dust, asbestos, heat, PAHs, and acid mist.

Since many of the workers had been exposed simultaneously to several factors, some factors may have affected each other to generate additive or synergistic action. Therefore, to distinguish the effects of single and multiple exposures, directions of the effects were summarized according to exposure factors in Table 4. For all factors, mortality was increased for all causes of death and all neoplasms when exposure was multiple. For PAHs, significant increases of mortality were found for many of the disease categories irrespective of single or multiple exposure. For iron dust, welding dust, asbestos, heat, and benzene, single exposures were associated with decreased mortality risks for all neoplasms and lung cancer, although the multiple exposures to the factors showed significantly increased mortality risks.

TABLE 3 (continued from preceding page)

Cause of Death (ICD-9)	Wood Exposure (n = 1,892) Obs.	Wood Exposure (n = 1,892) SRR (95% CI)	Heat Exposure (n = 20,729) Obs.	Heat Exposure (n = 20,729) SRR (95% CI)	CO Exposure (n = 5,083) Obs.	CO Exposure (n = 5,083) SRR (95% CI)
Infectious (1–139)	9	114 (109–191)	39	107 (104–110)	8	71 (56–90)
Tuberculosis (11–12)	7	187 (106–329)	30	124 (112–136)	7	88 (79–98)
Hepatitis (70)	0	—	4	70 (41–118)	1	79 (34–179)
Neoplasms (140–239)	61	72 (66–78)	452	113 (111–114)	79	58 (52–65)
Esophagus (150)	3	57 (29–111)	35	189 (139–257)	3	54 (27–104)
Stomach cancer (151)	11	57 (41–80)	64	100 (99–100)	7	31 (17–55)
Colon, sigmoid, and rectum (153–154)	1	16 (0–1,108)	22	129 (112–148)	1	11 (0–265)
Liver cancer (155)	9	62 (47–82)	88	116 (111–120)	20	81 (75–88)
Lung cancer (162)	26	87 (83–92)	169	115 (112–118)	34	65 (57–74)
Bladder (188)	2	257 (20–3,188)	3	50 (22–114)	1	53 (8–335)
Brain (191–192)	1	158 (6–4,088)	9	116 (102–131)	2	74 (46–117)
Leukemia (204–208)	0	—	10	66 (50–87)	4	96 (92–100)
Cerebrovascular (430–439)	63	81 (77–86)	446	124 (121–128)	67	56 (50–63)
Intracranial (431–432)	28	79 (73–86)	229	136 (129–144)	31	50 (41–61)
Cerebral infarction (434)	27	80 (74–86)	174	116 (113–120)	29	64 (56–73)
Circulatory system (390–429, 440–459)	21	72 (63–82)	181	123 (119–128)	31	58 (49–68)
AMI† (410)	8	52 (31–92)	77	132 (121–142)	11	58 (44–76)
Ischemic heart disease other than AMI (411–412)	3	54 (31–92)	44	117 (110–124)	5	42 (21–82)
Respiratory system (460–519)	20	53 (42–66)	251	167 (153–183)	46	94 (92–96)
COPD† (491–492)	17	54 (42–68)	130	115 (111–118)	31	84 (78–89)
Pneumoconiosis (502–509)	2	87 (67–114)	99	549 (291–1,035)	12	199 (110–356)
Injuries (800–959)	8	77 (63–94)	103	150 (135–167)	10	57 (40–79)
Others	14	42 (28–60)	170	118 (114–122)	31	71 (65–76)
All causes	196	71 (68–75)	1,642	126 (124–128)	272	63 (59–66)

With single exposure to silica, significantly increased mortality risk was seen only for pneumoconiosis.

Table 5 shows the combined effects of exposures to PAHs and dusts for all causes, all neoplasms, and selected sites of cancer. Compared with the nonexposed blue-collar workers as the internal reference, positive trends of SRRs were found more among those with exposures to PAHs than among those without PAH exposure for all causes and all neoplasms. The SRRs were 164, 208, and 654 in the former group, and 85, 226, and 332 in the latter group, according to the numbers of dust exposures. For stomach and liver cancers, such interaction was not clear between workers exposed to PAHs and those not exposed.

DISCUSSION

To our knowledge, this 1980–1993 cohort study enrolled the largest number (147,062) and observed the greatest number of person-years (1,967,620) among iron–steel foundry cohorts. In a U.S. steelworker cohort, the corresponding numbers were 59,072 and 511,864 during 1953–1961.[19–21] In a British steel foundry cohort, they were 10,250 and 203,070 from 1946–1965 to 1978.[22–24] In a Brazilian steel cohort, they were 21,816 and 197,499 during 1977–1990.[25] Only 1.9% (2,825/149,887) were lost to follow-up, comparable with previous studies.[19,22,25] The exclusion of 2,825 workers is unlikely to have had a large influence on the study findings.

This study evaluated the mortality risks of iron–steel foundry workers in relation to various exposure factors, in contrast to most previous studies, where job titles and/or job categories were used in the exposure assessments.[21,24–28] Our method enabled internal comparison of mortality between those with exposure and those without.

Mortality risks for almost all categories of cause of death were significantly increased among the exposed workers compared with the nonexposed blue-collar workers. SRRs stratified by factors in Table 4 indicated that, in particular, PAH exposure may strongly and broadly increase risk irrespective of single or multiple exposures, and that many dusts (except for cement) may increase risk weakly in single exposures but more strongly in multiple exposures. In Table 5, increasing trends were more apparent for those workers with PAH exposure than those without it, suggesting that the combined effect of PAH and dust exposure may be present for malignant neoplasms, especially for lung cancer.

TABLE 3 (continued from preceding page)

PAH Exposure (n = 5,245)		Oil Mist Exposure (n = 2,955)		Acid Mist Exposure (n = 866)		Benzene Exposure (n = 971)	
Obs.	SRR (95% CI)	Obs.	SRR (95% CI)	Obs.	SRR (95% CI)	Obs.	SRR (95% CI)
14	80 (69–92)	4	97 (92–102)	4	164 (123–218)	2	95 (85–106)
10	109 (101–116)	2	92 (72–118)	4	328 (24–4,302)	1	68 (22–207)
1	33 (0–18,278)	0	—	0	—	0	—
296	208 (184–235)	56	90 (87–93)	53	236 (167–334)	32	120 (111–130)
17	261 (125–543)	5	198 (72–541)	3	246 (34–1,752)	2	187 (36–959)
45	208 (152–284)	9	109 (101–117)	11	329 (97–1,113)	10	242 (103–563)
12	231 (106–502)	2	117 (75–179)	2	163 (106–250)	3	287 (16–5,089)
53	179 (143–223)	10	66 (50–86)	13	278 (102–754)	1	22 (0–1,343)
116	228 (181–285)	21	88 (83–93)	13	167 (110–252)	11	104 (100–108)
3	156 (69–352)	1	148 (7–2,975)	0	—	2	406 (1–99,967)
7	230 (75–701)	0	—	2	337 (4–25,380)	0	—
12	198 (109–360)	1	29 (1–630)	4	482 (10–21,981)	0	—
215	174 (157–193)	48	99 (99–100)	35	173 (136–220)	28	113 (107–120)
93	161 (141–182)	18	70 (47–61)	17	164 (120–223)	11	94 (90–98)
100	196 (161–236)	26	137 (118–159)	14	183 (118–283)	15	144 (114–181)
102	191 (160–228)	23	97 (96–99)	19	213 (130–348)	10	94 (89–99)
35	170 (134–215)	11	120 (106–136)	8	259 (71–941)	4	121 (81–179)
33	263 (155–446)	4	60 (34–102)	4	140 (83–234)	4	118 (91–152)
73	141 (127–156)	16	79 (71–88)	11	151 (110–207)	10	95 (91–98)
58	149 (130–169)	13	85 (78–93)	9	159 (106–238)	9	110 (102–118)
7	114 (101–128)	0	—	0	—	0	—
38	151 (128–178)	11	65 (49–86)	8	192 (86–428)	7	171 (86–337)
77	140 (127–154)	19	91 (87–95)	18	194 (123–306)	9	102 (100–103)
815	176 (166–186)	177	91 (90–93)	148	199 (171–232)	98	112 (109–115)

According to a review of the recent literature, the lung is a major target organ of PAH carcinogenicity.[29] Our study corroborated this, because SRR for lung cancer with single exposure to PAHs was 159 (95% CI: 115–219). Regarding other organs, skin and urinary bladder were reported to be target organs.[29] In this study, the SRRs of single exposures to PAHs were 1,577 for malignant neoplasm of skin (except melanoma) and 333 for urinary bladder, but neither was statistically significant. Further follow-up is necessary to evaluate this question.

The current study involves some limitations in estimating mortality risk. First, only dichotomous exposure assessment was conducted for the risk estimation, so detailed evaluations such as determining dose–response relationships between exposure and disease mortality could not be performed. The exposure assessment was performed without consideration of exposure concentration and using the only-one-job method as a surrogate measure of lifetime job method, although length of employment was adjusted. Therefore, it is possible that misclassification of the exposure status occurred. Misclassification is most likely to occur nondifferentially, leading to underestimation of mortality risks. Second, information about smoking and other lifestyle factors was not available in the study, so the effect of life style of the workers was not completely controlled. It is well known that the interactions between smoking, drinking, and some occupational factors such as asbestos exposure can significantly increase the risk for diseases. There is at least a theoretical possibility that men in different trades may incur these risks differentially. For these reasons, interpretation of the risk estimates should be done cautiously.?

On the other hand, the present study took some unique measures to overcome disadvantages inherent in previous studies. The mortality risks from all causes and main causes were significantly decreased compared with those of the general population, as expected due to the healthy-worker effect. To avert this problem, comparison between the workers who had the same employment process but different exposure status was recommended.[16] Therefore, we first calculated SMRs by job type and exposure status, and found positive trends of SMRs, where the healthy-worker effect could be effectively controlled. Second, we evaluated mortality risks using SRR analysis, which enabled the assessment of exposure–response trends adjusting for confounding factors, i.e., age and employment period.[30] We believe this approach minimized potential confounding by the healthy-worker effect.

TABLE 4 Summary of Direction¶ of Mortality Effect by Harmful Agents among Chinese Iron-steel Workers (Male, 1980–1993)

Cause of Death (ICD-9) and Exposure Type	Silica	Iron	Welding	Coal	Cement	Grinding	Asbestos
All causes							
Single†	↓*	↓*	↑*	↓*	↑*	↓*	↓*
Multiple†	↑*	↑*	↑*	↑*	↑*	↑*	↑*
Neoplasms (140–239)							
Single†	↓*	↓	↑*	↓*	↑*	↓	↓*
Multiple†	↑*	↑*	↑*	↑*	↑*	↑	↑*
Lung cancer (162)							
Single†	↓*	↓*	↓*	↓*	↑*	↓	↓*
Multiple†	↑*	↑*	↑*	↑*	↑	↑	↑*
Stomach cancer (151)							
Single†	↓*	↑*	↑*	↓*	↓*	—	↑*
Multiple†	↑*	↑*	↑	↑*	↑	↑	↑*
Liver cancer (155)							
Single†	↓*	↓*	↓	↓	↑*	—	↓*
Multiple†	↑*	↑*	↑*	↑*	↑*	↑*	↑*
Respiratory system (460–519)							
Single†	↓*	↓*	↓*	↓*	↑*	—	↓*
Multiple†	↑*	↑*	↑*	↑*	↑	↑	↑*
Pneumoconiosis (502–509)							
Single†	↑*	↑*	↑	↓	↓	—	↓
Multiple†	↑*	↑*	↑	↑	↑	—	↑

¶↑: SRR more than 100. ↓: SRR less than 100. —: deceased workers not detected. †Without any other exposure. ‡With at least one other exposure. *Statistically significant, $p < 0.05$.

In conclusion, in a Chinese iron–steel industry cohort study with a total of 121,846 male subjects, 13,363 deaths were observed during the 14-year follow-up period. Although the SMRs for all causes and all neoplasms in comparison with the general population were lower than expected, this would have been due to the healthy-worker effect. The SRRs for the causes in comparison with the nonexposed blue-collar workers

TABLE 5 SRRs for All Causes and Malignant Neoplasms by Exposure to PAHs and Number of Dust Exposures among Chinese Iron–Steel Workers (Male, 1980–1993)

			Exposed to PAHs					
	Reference* (n = 39,048)		No Dust Exposure* (n = 1,067)		One Dust Exposure (n = 3,409)		Two or More Dust Exposures (n = 769)	
Cause of Death (ICD-9)	Obs.	SRR	Obs.	SRR (95% CI)	Obs.	SRR (95% CI)	Obs.	SRR (95% CI)
All causes	4,631	100	**180**	**147 (137–158)**	**514**	**166 (156–176)**	**188**	**480 (302–761)**
All neoplasms (140–239)	1,395	100	**68**	**181 (149–219)**	**186**	**195 (170–223)**	**66**	**541 (209–1,395)**
Lip, oral cavity, and pharynx (140–149)	17	100	3	487 (1–>9,999)	4	283 (23–3,417	2	1,278 (0–>9,999)
Esophagus (150)	65	100	2	131 (76–225)	**10**	**222 (104–474)**	6	1,162 (0–>9,999)
Stomach (151)	225	100	**20**	**326 (127–834)**	**24**	**187 (134–261)**	7	374 (43–3,212)
Colon, sigmoid, and rectum (153–154)	60	100	4	282 (29–2,698	8	231 (96–554)	3	544 (2–>9,999)
Liver (155)	265	100	9	119 (99–142)	**37**	**186 (139–247)**	9	286 (68–1,199)
Pancreas (157)	32	100	2	223 (13–3,742	2	126 (78–203)	0	—
Larynx (161)	18	100	1	129 (42–391)	6	428 (25–7,249)	3	2,827 (0–>9,999)
Lung	507	100	**21**	**164 (124–215)**	**74**	**208 (162–266)**	**28**	**654 (113–3,780)**
Bladder (188)	24	100	1	152 (6–3,381)	2	181 (42–772)	0	—
Brain (191–192)	27	100	1	125 (29–530)	5	240 (62–918)	2	768 (0–>9,999)
Leukemia (204–208)	51	100	3	207 (42–1,020)	7	172 (96–307)	2	215 (14–3,236)

*Non-exposed blue-collar workers. Bold-typed characters indicate statistically significant. †Including silica, iron dust, welding dust, coal dust, cement dust, grinding dust, asbestos, wood dust, and other dusts.

TABLE 4 (continued from previous page)

Cause of Death (ICD-9) and Exposure Type	Wood	Heat	CO	PAH	Oil Mist	Acid Mist	Benzene
All causes							
Single†	↓*	↓*	↓*	↑*	↓*	↑*	↓*
Multiple†	↑*	↑*	↑*	↑*	↑*	↑*	↑*
Neoplasms (140–239)							
Single†	↓*	↓*	↓*	↑*	↓*	↑*	↓*
Multiple†	↑	↑*	↑*	↑*	↑*	↑*	↑
Lung cancer (162)							
Single†	↓*	↓*	↓*	↑*	↓	↓	↓
Multiple†	↑	↑*	↑	↑*	↑	↑	↑
Stomach cancer (151)							
Single†	↓*	↓*	—	↑	↓	↑	↑*
Multiple†	↑	↑*	↓*	↑*	↑	↑	↑
Liver cancer (155)							
Single†	↓	↓*	↓	↑	↓	↑	—
Multiple†	↑*	↑*	↑*	↑*	↑*	↑*	↑*
Respiratory system (460–519)							
Single†	↓	↑*	↓	↑*	↓	↓	↓
Multiple†	↑	↑*	↑*	↑*	↑	↑	↑
Pneumoconiosis (502–509)							
Single†	↓	↑*	↑	—	—	—	—
Multiple†	↑	↑*	↑*	↑*	—	—	—

tended to increase according to single or multiple exposure. In particular, the combined exposures of PAHs and two or more dusts increased the risks of lung cancer (SRR = 654; 95% CI: 113–3,780) and other malignancies. The results indicate the adverse health effects, including carcinogenic risks, of foundry working.

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TABLE 5 (continued from previous page)

	Not Exposed to PAHs					
	One Dust Exposure* (*n* = 33,461)		Two Dust Exposures (*n* = 4,930)		Three or More Dust Exposures (*n* = 2,890)	
Cause of Death (ICD-9)	Obs.	SRR (95% CI)	Obs.	SRR (95% CI)	Obs.	SRR (95% CI)
All causes	**4,023**	**89 (88–90)**	**1,001**	**226 (209–243)**	**531**	**294 (253–341)**
All neoplasms (140–239)	**1,193**	**87 (85–88)**	**275**	**205 (181–231)**	**172**	**333 (246–451)**
Lip, oral cavity, and pharynx (140–149)	**19**	**114 (104–125)**	1	67 (20–219)	1	144 (15–1,294)
Esophagus (150)	**84**	**129 (118–139)**	**22**	**350 (136–901)**	**14**	649 (62–6,783)
Stomach (151)	**181**	**80 (77–84)**	**41**	**200 (148–268)**	**35**	**442 (166–1,173)**
Colon, sigmoid, and rectum (153–154)	56	100 (99–101)	**10**	**170 (106–272)**	3	111 (92–134)
Liver (155)	**236**	**93 (91–94)**	**50**	**192 (149–248)**	**29**	**323 (155–630)**
Pancreas (157)	**27**	**82 (73–91)**	9	292 (85–1,006)	5	422 (23–7,621)
Larynx (161)	**20**	**107 (102–111)**	5	259 (62–1,078)	1	130 (32–512)
Lung (162)	**433**	**85 (83–87)**	**112**	**226 (179–284)**	**64**	**332 (198–555)**
Bladder (188)	**12**	**56 (38–83)**	7	323 (38–2,729)	3	348 (6–>9,999)
Brain (191–192)	**25**	**96 (93–98)**	3	82 (51–129)	2	88 (57–134)
Leukemia (204–208)	**32**	**67 (55–80)**	6	126 (95–167)	6	285 (53–1,509)

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