

CHEMICAL AGENTS AND RELATED OCCUPATIONS

VOLUME 100 F
A REVIEW OF HUMAN CARCINOGENS

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Evaluation of Carcinogenic Risks to Humans,
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OCCUPATIONAL EXPOSURES DURING IRON AND STEEL FOUNDING

Iron and steel founding was considered by previous IARC Working Groups in 1983 and 1987 ([IARC, 1984, 1987](#)). Since that time, new data have become available, which have been incorporated in this *Monograph*, and taken into consideration in the present evaluation.

1. Exposure Data

1.1 Manufacturing process

Foundries produce shaped castings from re-melted metal ingots and scrap. Although foundry work is assumed to start with re-melting of ingots and scrap and to end with the fettling of castings, the industry is often so integrated that the distinction is not obvious. Machine shops are not normally part of the work environment where castings are produced; however, simple and accessory machining may be carried out, and these activities may be part of small foundry operations. The processes in iron and steel founding generally comprise pattern-making, moulding and core-making, melting, pouring and shake-out, and fettling. A detailed description of these production steps can be found in [IARC \(1984\)](#).

1.2 Human exposures

The iron and steel industry is very diverse in materials and processes, resulting in occupational exposures to a wide variety of substances. Substantial exposures to silica and carbon

monoxide continue to occur in many foundries. Occupational exposures to airborne polycyclic aromatic hydrocarbons (PAHs) are also present, resulting mainly from the thermal decomposition of carbonaceous ingredients commonly added to foundry sand. In addition, some steel-foundry workers (e.g. fettlers) are exposed to airborne chromium and nickel compounds. The introduction of organic binder materials in the late 1950s has resulted in exposures of foundry workers to other chemicals, including phenol, formaldehyde, isocyanates and various amines.

Earlier exposure studies have been reviewed previously ([IARC, 1984](#)). More recent studies are presented here and summarized in [Table 1.1](#).

1.2.1 Respirable dust and respirable quartz

Nearly all production workers in iron and steel foundries are exposed to silica dust and other mineral constituents of foundry sand ([IARC, 1984](#)). In a study from Sweden, the ‘furnace and ladle repair workers’ and fettlers were the highest exposed individuals. For the furnace workers ($n = 33$), geometric mean levels of respirable dust and respirable quartz were 1.2 mg/m^3 (range, 0.25–9.3) and 0.052 mg/m^3

Table 1.1 Exposures of workers in the iron and steel founding industry

Reference, Country	Year of study	Operation/Job/Task	No. of subjects	No. of samples	No. of smokers	Exposure	Air levels ($\mu\text{g}/\text{m}^3$)		Urine levels ($\mu\text{mol}/\text{mol}$ creatinine)	
							Geometric Mean	Range	Mean	Range or SD
Respirable dust and respirable quartz										
Andersson <i>et al.</i> (2009) Sweden	2005–2006	11 iron foundries		436		Respirable dust	580	76–31000		
				435		Respirable quartz	28	3–2100		
Chen <i>et al.</i> (2006) Taiwan, China	1990		1234	277	645	Respirable dust				
		Iron making								
		Sinter		112	27			90–5080		
		Coal preparation		40	9			20–430		
		Blast furnace		311	72			90–830		
		Steel making								
		Steel materials plant		192	30			130–7590		
		Basic oxygen furnace		174	47			170–310		
		Continuous steel casting		333	75			320–790		
		Steel slab rectifying		72	17			160–1140		
Binder compounds – methyl isocyanate, isocyanic acid and formaldehyde										
Westberg <i>et al.</i> (2005) Sweden	2001	Four foundries using the Hot Box core-binder system								
						Short-term samples:				
		Total		298		methyl isocyanate	4.9	< 4–68		
				297		isocyanic acid	24	< 4–280		
		Core maker		179		methyl isocyanate	4	< 4–60		
				178		isocyanic acid	22	< 4–130		
		Die caster		71		methyl isocyanate	10	< 4–68		
				71		isocyanic acid	48	< 4–280		
		Others		48		methyl isocyanate	3.4	< 4–12		
				48		isocyanic acid	12	< 4–66		

Table 1.1 (continued)

Reference, Country	Year of study	Operation/Job/Task	No. of subjects	No. of samples	No. of smokers	Exposure	Air levels ($\mu\text{g}/\text{m}^3$)		Urine levels ($\mu\text{mol}/\text{mol}$ creatinine)	
							Geometric Mean	Range	Mean	Range or SD
Westberg <i>et al.</i> (2005) (contd)						8-hour TWA samples				
		Total		64		methyl isocyanate	5.3	< 4–31		
				64		isocyanic acid	27	< 4–190		
				64		formaldehyde	120	14–1600		
		Core maker		39		methyl isocyanate	4.2	< 4–20		
				39		isocyanic acid	24	5.3–84		
				39		formaldehyde	200	50–1600		
		Die caster		15		methyl isocyanate	12	< 4–31		
				15		isocyanic acid	55	6–190		
				15		formaldehyde	63	21–220		
		Others		10		methyl isocyanate	3.5	< 4–8.2		
				10		isocyanic acid	14	< 4–32		
				10		formaldehyde	32	14–170		

Table 1.1 (continued)

Reference, Country	Year of study	Operation/Job/Task	No. of subjects	No. of samples	No. of smokers	Exposure	Air levels ($\mu\text{g}/\text{m}^3$)		Urine levels ($\mu\text{mol}/\text{mol}$ creatinine)	
							Geometric Mean	Range	Mean	Range or SD
Polycyclic aromatic hydrocarbons										
Hansen <i>et al.</i> (1994)	1988–89	Iron foundry workers		24						
Denmark		- melters		5		Pyrene		0.02		
		- transporters		2				0.0		
		- casters		9				0.22		
		- machine moulders		1				0.12		
		- hand moulders		2				0.22		
		- shake workers		3				0.01		
		- finishing workers		2				0.0		
						Benzo[a]pyrene				
		- melters						0.01		
		- transporters						0.0		
		- casters						0.02		
		- machine moulders						0.04		
		- hand moulders						0.03		
		- shake workers						0.0		
		- finishing workers						0.0		
						Sum of 15 PAHs				
		- melters						6.66		
		- transporters						5.78		
		- casters						10.91		
		- machine moulders						9.64		
		- hand moulders						5.63		
		- shake workers						11.17		
		- finishing workers						0.58		

Table 1.1 (continued)

Reference, Country	Year of study	Operation/Job/Task	No. of subjects	No. of samples	No. of smokers	Exposure	Air levels ($\mu\text{g}/\text{m}^3$)		Urine levels ($\mu\text{mol}/\text{mol}$ creatinine)	
							Geometric Mean	Range	Mean	Range or SD
Hansen <i>et al.</i> (1994) (contd)		B[a]P exposed workers:								
		- Foundry 1 – low		19						0.023
		- Foundry 2 – low		13						0.065
		- Foundry 1 – medium		14						0.030
		- Foundry 2 – medium		24						0.046
		- Foundry 1 – high		1						–
		- Foundry 2 – high		18						0.033
		Pyrene exposed workers:								
		- Foundry 1 – low		19						0.023
		- Foundry 2 – low		5						0.062
		- Foundry 1 – medium		5						0.013
		- Foundry 2 – medium		32						0.044
		- Foundry 1 – high		10						0.036
		- Foundry 2 – high		18						0.046
		PAH exposed workers^b:								
		- Foundry 1 – low		0						–
		- Foundry 2 – low		0						–
		- Foundry 1 – medium		29						0.022
		- Foundry 2 – medium		29						0.041
		- Foundry 1 – high		5						0.043
		- Foundry 2 – high		26						0.053

^a Calculated from short-term samples

^b Sum of 15 PAHs

(range, 0.0098–0.83), respectively. For the fettlers ($n = 115$), geometric mean levels of respirable dust and respirable quartz were 0.69 mg/m^3 (range, 0.076–31) and 0.041 mg/m^3 (range, 0.004–2.1), respectively. Major improvements with respect to occupational hygiene in the foundries showed maximum average concentrations of respirable dust being almost equal to the minimum average concentrations for various jobs in the past ([Andersson *et al.*, 2009](#)). A study from Taiwan, China in a continuous steel-casting plant showed similar respirable dust levels ([Chen *et al.*, 2006](#)).

1.2.2 Carbon monoxide

Carbon monoxide (CO) is formed whenever there is incomplete combustion of carbonaceous material. In the foundry environment, CO is mainly produced by cupola melting and casting operations. Other sources of CO exposure are oil burners used for charge and ladle preheating, furnaces for annealing and carburizing, transport equipment powered by internal combustion engines and fettling operations such as welding and flame cutting. Workers in the furnace area may be exposed to gases leaking from the charging and bottom levels of the cupolas ([IARC, 1984](#)).

Results of recent measurements of exposure levels to CO in iron and steel foundries were not available to the Working Group.

1.2.3 Binder compounds

Organic binder materials for cores and moulds include furan, phenol-formaldehyde, urea-formaldehyde and urethane resins as well as oleo-resinous oils. These ingredients may volatilize into the workplace air during mixing, blowing, ramming, drying or baking operations. Curing reactions and thermal decomposition give rise to formation of additional compounds, which are released during pouring and shake-out. When organic binders are subjected to

high temperatures, pyrolysis may produce gases and smoke aerosols. Only a few components of these emissions have been identified: aliphatic components include methane, ethane, ethylene, acetylene, and smaller amounts of high molecular-weight compounds; aromatic substances include benzene, toluene, xylenes, naphthalenes and a variety of PAHs in lower concentrations. Nitrogen compounds such as ammonia, cyanides and amines may be formed from the nitrogen-containing urea, ammonium salts and hexamethylenetetramine that are used as binder chemicals. Urethane resins may emit free isocyanates under moulding and pouring conditions. No-bake catalysts, based on arylsulphonic acids, may produce sulfur dioxide and hydrogen sulphide by thermal processes. If phosphoric acid is used as a catalyst, phosphine can be formed in the strongly reducing atmosphere of the hot emissions. In air, phosphine rapidly oxidizes to phosphorus oxide. Furan binders contain free furfuryl alcohol, which can volatilize during mixing, moulding or core-making. Similarly, furan and phenolic resins may emit formaldehyde, phenol and other derivatives by volatilization or thermal decomposition. Core oils and alkyd-isocyanate resins are partly composed of natural drying oils, and heating of these materials gives rise to acrolein, various aldehydes, ketones, acids and esters as well as aliphatic hydrocarbons. When organic solvents are used in sand binders, the vapours may add to the exposure of workers ([Toeniskoetter & Schafer, 1977](#); [IARC, 1984](#)).

A study from Sweden by [Westberg *et al.* \(2005\)](#) showed that core-makers had higher average exposure to formaldehyde than casters who were more exposed to methyl-isocyanate and isocyanic acid. All four Swedish foundries using the Hot Box core-binder system were included in this study.

1.2.4 Polycyclic aromatic hydrocarbons

PAHs result from thermal decomposition of carbonaceous ingredients in foundry sand. During casting, PAHs are formed and partly vapourised under the extremely hot and reducing conditions at the mould-metal interface. They are then adsorbed onto soot, fume or sand particles and spread throughout the workplace during shake-out and other dusty operations. Although the mechanism of PAH formation is complex and variable, the reactions proceed via pathways that involve free radicals. Various radical species containing carbon atoms combine in rapid fashion at the temperature range of 500–800 °C. This pyro-synthesis is influenced by many variables, such as the composition of the gaseous atmosphere and the chemical structure of the carbonaceous material. Organic binders, coal powder and other carbonaceous additives are the predominant sources of PAHs in iron and steel foundries. In some cases, exhaust gases from engines, furnaces and ovens may increase the exposure of workers to these compounds ([IARC, 1984](#)).

Studies from the late 1980s in Denmark showed low levels of exposure to PAHs in the foundry workplace atmosphere ([Hansen et al., 1994](#); [Table 1.1](#)).

1.2.5 Metals

Metal fumes are formed by evaporation, condensation and oxidation of metals in air. Furnace tenders, melters, casters, ladle-men, pourers and crane drivers are exposed to fumes from molten metal; fitters are exposed to metal fumes and dusts from grinding, welding and flame-cutting operations ([IARC, 1984](#)).

[Apostoli et al. \(1988\)](#) measured concentrations of thallium in urine of workers in two cast-iron foundries (with 9 and 12 subjects, respectively) employed in cupola furnace operating and iron casting. These workers were potentially exposed

to coke dust. The mean urinary thallium concentrations were higher for the cast-iron foundry workers ($n = 21$; mean, 0.33 µg/l; range, 0.06–1.04) than for non-exposed individuals ($n = 72$; mean, 0.22 µg/l; range, 0.06–0.61), but lower than those of cement workers ($n = 30$; mean, 0.38 µg/l; range, 0.08–1.22).

In a study from the United Kingdom, concentrations of 14 elements in the blood of workers with low-alloy steel and stainless steel, and of workers exposed to lead- and cadmium-containing dusts were measured and compared with values of similar measurements in an unexposed control group. Concentrations of 20 elements in dust samples were also measured. The only elements that showed significantly elevated levels in whole blood were cadmium in workers with non-ferrous metals and lead in all workers. The values for lead were 97.2 ± 39.9 ppb in 19 workers with low-alloy steel, 74.9 ± 25.1 ppb in 20 workers with stainless steel, and 54.0 ± 18.2 ppb in 42 controls. The high concentrations cadmium and lead in the dusts confirmed the exposure of the workers ([Triger et al., 1989](#)).

In a Danish study conducted over a 16-month period in 1996–1997, manganese concentrations were measured in air, and in blood from 24 furnace-men employed in three small-size foundries and from 21 scrap-recycling workers at another plant. Furnace-men who work in poorly ventilated smelting departments were found to have higher levels of manganese in their blood (approx. 2.5 ± 5 µg/L above reference values) despite low concentrations of manganese in fumes (0.002 ± 0.064 mg/m³) ([Lander et al., 1999](#)).

In a Taiwan, China study among 63 steel-production workers, [Horng et al. \(2003\)](#) measured urinary concentrations of cadmium, cobalt, nickel and lead by use of differential-pulse stripping voltammetry. Mean concentrations of the metals were elevated: cadmium: 9.52 µg/L, range, 3.19–22.07; cobalt: 8.18 µg/L, range, 3.06–23.30; nickel: 33.10 µg/L, range, 13.90–78.90; lead:

53.50 µg/L, range, 28.90–85.60. Age and duration of employment were correlated with the urinary concentrations of these metals. [The limited information provided on the actual job content of the workers did not give insight whether these workers were employed in primary steel production or in the steel foundry.]

1.2.6 Refractory ceramic fibres

In a study on refractory ceramic fibres (RCF) from Belgium, ferruginous bodies mimicking asbestos were detected in nine of 1800 bronchoalveolar lavage samples collected during 1992–1997 ([Dumortier et al., 2001](#)). The nine individuals from whom these samples originated had been employed either as foundry worker, steel worker, or welder and the majority had worked on furnace insulation. Mean airborne fibre concentrations measured during installation or removal of RCF furnace insulation are close to 1 fibre/mL air ([Maxim et al., 1997](#)), but concentrations as high as 23 fibres/mL have been detected ([Strübel & Faul, 1994](#)).

Two Finnish steel plants, three foundries and a repair shop were studied for the presence of airborne ceramic fibres under normal production and maintenance conditions (i.e. during the replacement of oven insulation). While ceramic fibres were found in most production phases (range < 0.01–0.29 fibres/cm³), considerably higher fibre counts were obtained during maintenance work (range < 0.01–14.2 fibres/cm³). Results from nasal sampling were found to correlate with the airborne fibre concentrations at the group level. The mean fibre concentrations varied from 34 to 930 fibres/cm³ of lavage fluid ([Linnainmaa et al., 2007](#)).

2. Cancer in Humans

2.1 Cohort studies

There are 13 cohort studies available on iron and steel founding workers in various parts of the world. Nearly all of these show a significantly increased risk for lung cancer, either in the entire cohort or in high-exposed subgroups ([Koskela et al., 1976](#); [Gibson et al., 1977](#); [Tola et al., 1979](#); [Sitas et al., 1989](#); [Andjelkovich et al., 1990, 1992, 1995](#); [Moulin et al., 1993](#); [Sorahan et al., 1994](#); [Adzersen et al., 2003](#); [Hoshuyama et al., 2006](#); see Table 2.1, available at <http://monographs.iarc.fr/ENG/Monographs/vol100F/100F-29-Table2.1.pdf>). There was a non-significantly increased lung-cancer risk in one study ([Decoufle & Wood, 1979](#)). In the cohort study from the United Kingdom ([Sorahan et al., 1994](#)) an internal dose–response in terms of years of employment was found. A study from the USA showed a significantly increased lung-cancer risk after adjustment for smoking ([Andjelkovich et al., 1994](#)). There are two additional cohorts, based on proportional mortality, that provide supporting evidence for an excess of lung cancer in foundry workers ([Egan-Baum et al., 1981](#); [Silverstein et al., 1986](#)).

2.2 Case–control studies

Two population-based case–control studies, one from the USA ([Blot et al., 1983](#)) and one from Poland ([Becher et al., 1989](#)), showed a statistically significant excess of lung cancer in association with foundry work, with adjustment for smoking (see Table 2.2, available at <http://monographs.iarc.fr/ENG/Monographs/vol100F/100F-29-Table2.2.pdf>).

2.3 Synthesis

Considering the consistent findings in the available cohort studies and the positive evidence from two case-control studies, the epidemiological data clearly support the notion that work in iron and steel foundries is associated with an increased risk for lung cancer. Chance, bias and confounding are not likely to explain the excess risk.

3. Cancer in Experimental Animals

No data were available to the Working Group.

4. Other Relevant Data

4.1 Mechanistic evidence relevant to the carcinogenic hazards from occupational exposures during iron and steel founding

4.1.1 Experimental systems

In several studies extracts of particulates from samples collected at a steel foundry were mutagenic in *Salmonella typhimurium* strain TA98 in the presence or absence of an exogenous metabolic activation system. In another study filter extracts were mutagenic in *Salmonella typhimurium* strains TA98 and TA100 in the presence or absence of an exogenous metabolic activation system ([IARC, 1984](#)).

Exposures in the iron and steel founding industry are complex and include a wide variety of known genotoxic and carcinogenic substances including PAHs, metals (e.g. nickel, chromium) and formaldehyde. These agents have been previously reviewed by [IARC \(1983, 1990, 1995, 2010\)](#).

4.1.2 Humans

Workers in a Finnish iron foundry with occupational exposure to PAHs and control subjects without occupational exposure were

examined for the presence in peripheral white blood cells of aromatic DNA adducts by use of ³²P-postlabelling. There was a significant correlation between the estimated exposures and DNA-adduct levels ([Phillips et al., 1988](#); [Reddy et al., 1991](#)). The competitive ELISA assay was used to study another population of foundry workers with respect to the presence of aromatic DNA-adducts, which showed a positive trend with exposure ([Santella et al., 1993](#)). Foundry workers studied over a period of five years were evaluated for aromatic DNA adducts in their leukocytes. During this time exposure to PAHs decreased and the level of DNA adducts decreased also. In the total group exposure was related to the level of DNA adducts ([Perera et al., 1994](#); [Hemminki et al., 1997](#); [Perera et al., 2000](#)).

4.2 Synthesis

There is moderate evidence that extracts of particles collected from a steel foundry act via a genotoxic mechanism, based on bacterial mutation studies. There is weak evidence for a genotoxic mechanism of action for exposures during iron and steel founding, based on DNA-adduct studies.

5. Evaluation

There is *sufficient evidence* in humans for the carcinogenicity of occupational exposures during iron and steel founding. Occupational exposures during iron and steel founding cause cancer of the lung.

No data on the carcinogenicity to experimental animals of mixtures present in iron and steel founding were available to the Working Group.

Occupational exposures during iron and steel founding are *carcinogenic to humans (Group 1)*.

References

- Adzersen KH, Becker N, Steindorf K, Frentzel-Beyme R (2003). Cancer mortality in a cohort of male German iron foundry workers. *Am J Ind Med*, 43: 295–305. doi:10.1002/ajim.10187 PMID:12594777
- Andersson L, Bryngelsson I-L, Ohlson C-G *et al.* (2009). Quartz and dust exposure in Swedish iron foundries. *J Occup Environ Hyg*, 6: 9–18. doi:10.1080/15459620802523943 PMID:18982534
- Andjelkovich DA, Janszen DB, Brown MH *et al.* (1995). Mortality of iron foundry workers: IV. Analysis of a subcohort exposed to formaldehyde. *J Occup Environ Med*, 37: 826–837. doi:10.1097/00043764-199507000-00012 PMID:7552467
- Andjelkovich DA, Mathew RM, Richardson RB, Levine RJ (1990). Mortality of iron foundry workers: I. Overall findings. *J Occup Med*, 32: 529–540. doi:10.1097/00043764-199006000-00010 PMID:2166148
- Andjelkovich DA, Mathew RM, Yu RC *et al.* (1992). Mortality of iron foundry workers. II. Analysis by work area. *J Occup Med*, 34: 391–401. PMID:1564577
- Andjelkovich DA, Shy CM, Brown MH *et al.* (1994). Mortality of iron foundry workers. III. Lung cancer case-control study. *J Occup Med*, 36: 1301–1309. doi:10.1097/00043764-199412000-00011 PMID:7884571
- Apostoli P, Maranelli G, Minoia C *et al.* (1988). Urinary thallium: critical problems, reference values and preliminary results of an investigation in workers with suspected industrial exposure. *Sci Total Environ*, 71: 513–518. doi:10.1016/0048-9697(88)90226-4 PMID:3406716
- Becher H, Jedrychowski W, Flak E *et al.* (1989). Lung cancer, smoking, and employment in foundries. *Scand J Work Environ Health*, 15: 38–42. doi:10.5271/sjweh.1884 PMID:2922587
- Blot WJ, Brown LM, Pottern LM *et al.* (1983). Lung cancer among long-term steel workers. *Am J Epidemiol*, 117: 706–716. PMID:6859026
- Chen P-C, Doyle PE, Wang J-D (2006). Respirable dust exposure and respiratory health in male Taiwanese steelworkers. *Ind Health*, 44: 190–199. doi:10.2486/indhealth.44.190 PMID:16610559
- Decoufle P & Wood DJ (1979). Mortality patterns among workers in a gray iron foundry. *Am J Epidemiol*, 109: 667–675. PMID:453187
- Dumortier P, Broucke I, De Vuyst P (2001). Pseudoasbestos bodies and fibers in bronchoalveolar lavage of refractory ceramic fiber users. *Am J Respir Crit Care Med*, 164: 499–503. PMID:11500357
- Egan-Baum E, Miller BA, Waxweiler RJ (1981). Lung cancer and other mortality patterns among foundrymen. *Scand J Work Environ Health*, 7: Suppl 4147–155. PMID:7330626
- Gibson ES, Martin RH, Lockington JN (1977). Lung cancer mortality in a steel foundry. *J Occup Med*, 19: 807–812. doi:10.1097/00043764-197712000-00006 PMID:592002
- Hansen AM, Omland O, Poulsen OM *et al.* (1994). Correlation between work process-related exposure to polycyclic aromatic hydrocarbons and urinary levels of alpha-naphthol, beta-naphthylamine and 1-hydroxypyrene in iron foundry workers. *Int Arch Occup Environ Health*, 65: 385–394. doi:10.1007/BF00383249 PMID:8034363
- Hemminki K, Dickey C, Karlsson S *et al.* (1997). Aromatic DNA adducts in foundry workers in relation to exposure, life style and CYP1A1 and glutathione transferase M1 genotype. *Carcinogenesis*, 18: 345–350. doi:10.1093/carcin/18.2.345 PMID:9054627
- Horng C-J, Horng P-H, Hsu JW, Tsai J-L (2003). Simultaneous determination of urinary cadmium, cobalt, lead, and nickel concentrations in steel production workers by differential pulse stripping voltammetry. *Arch Environ Health*, 58: 104–110. doi:10.3200/AEOH.58.2.104-110 PMID:12899211
- Hoshuyama T, Pan G, Tanaka C *et al.* (2006). Mortality of iron-steel workers in Anshan, China: a retrospective cohort study. *Int J Occup Environ Health*, 12: 193–202. PMID:16967824
- IARC (1983). Polynuclear aromatic compounds, Part 1, chemical, environmental and experimental data. *IARC Monogr Eval Carcinog Risk Chem Hum*, 32: 1–453. PMID:6586639
- IARC (1984). Polynuclear aromatic compounds, Part 3, industrial exposures in aluminium production, coal gasification, coke production, and iron and steel founding. *IARC Monogr Eval Carcinog Risk Chem Hum*, 34: 1–219.
- IARC (1990). Chromium, nickel and welding. *IARC Monogr Eval Carcinog Risks Hum*, 49: 1–648. PMID:2232124
- IARC (1995). Wood dust and formaldehyde. *IARC Monogr Eval Carcinog Risks Hum*, 62: 1–405.
- IARC (1987). Overall evaluations of carcinogenicity: an updating of IARC Monographs volumes 1 to 42. *IARC Monogr Eval Carcinog Risks Hum Suppl*, 7: 1–440. PMID:3482203
- IARC (2010). Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures. *IARC Monogr Eval Carcinog Risks Hum*, 92: 1–853. PMID:21141735 PMID:18756632
- Koskela RS, Hernberg S, Kärävä R *et al.* (1976). A mortality study of foundry workers. *Scand J Work Environ Health*, 2: Suppl 173–89. doi:10.5271/sjweh.2829 PMID:968468
- Lander F, Kristiansen J, Lauritsen JM (1999). Manganese exposure in foundry furnacemen and scrap recycling workers. *Int Arch Occup Environ Health*, 72: 546–550. doi:10.1007/s004200050414 PMID:10592008

- Linnainmaa M, Kangas J, Mäkinen M *et al.* (2007). Exposure to refractory ceramic fibres in the metal industry. *Ann Occup Hyg*, 51: 509–516. doi:10.1093/annhyg/mem027 PMID:17607019
- Maxim LD, Allshouse JN, Kelly WP *et al.* (1997). A multi-year workplace-monitoring program for refractory ceramic fibers: findings and conclusions. *Regul Toxicol Pharmacol*, 26: 156–171. doi:10.1006/rtph.1997.1153 PMID:9356279
- Moulin JJ, Wild P, Mantout B *et al.* (1993). Mortality from lung cancer and cardiovascular diseases among stainless-steel producing workers. *Cancer Causes Control*, 4: 75–81. doi:10.1007/BF00053147 PMID:8386949
- Perera F, Tsai WY, Dickey C, Hemminki K (2000). Re: Hemminki, K., Dickey, C., Karlsson, S., Bell, D., Hsu, Y., Tsai, W.-Y., Mooney, L.A., Savelle, K. and Perera, F.P. (1997) Aromatic DNA adducts in foundry workers in relation to exposure, lifestyle and CYP1A1 and glutathione transferase M1 genotype. *Carcinogenesis*, 18, 345–350. *Carcinogenesis*, 21: 849 doi:10.1093/carcin/21.4.849 PMID:10753227
- Perera FP, Dickey C, Santella R *et al.* (1994). Carcinogen-DNA adducts and gene mutation in foundry workers with low-level exposure to polycyclic aromatic hydrocarbons. *Carcinogenesis*, 15: 2905–2910. doi:10.1093/carcin/15.12.2905 PMID:8001254
- Phillips DH, Hemminki K, Alhonen A *et al.* (1988). Monitoring occupational exposure to carcinogens: detection by ³²P-postlabelling of aromatic DNA adducts in white blood cells from iron foundry workers. *Mutat Res*, 204: 531–541. doi:10.1016/0165-1218(88)90047-X PMID:3347220
- Reddy MV, Hemminki K, Randerath K (1991). Postlabeling analysis of polycyclic aromatic hydrocarbon-DNA adducts in white blood cells of foundry workers. *J Toxicol Environ Health*, 34: 177–185. doi:10.1080/15287399109531557 PMID:1920524
- Santella RM, Hemminki K, Tang DL *et al.* (1993). Polycyclic aromatic hydrocarbon-DNA adducts in white blood cells and urinary 1-hydroxypyrene in foundry workers. *Cancer Epidemiol Biomarkers Prev*, 2: 59–62. PMID:8420613
- Silverstein M, Maizlish N, Park R *et al.* (1986). Mortality among ferrous foundry workers. *Am J Ind Med*, 10: 27–43. doi:10.1002/ajim.4700100105 PMID:3740065
- Sitas F, Douglas AJ, Webster EC (1989). Respiratory disease mortality patterns among South African iron moulders. *Br J Ind Med*, 46: 310–315. PMID:2751928
- Sorahan T, Faux AM, Cooke MA (1994). Mortality among a cohort of United Kingdom steel foundry workers with special reference to cancers of the stomach and lung, 1946–90. *Occup Environ Med*, 51: 316–322. doi:10.1136/oem.51.5.316 PMID:8199681
- Strübel G & Faul L (1994). Pollutant load caused by ceramic fibers and new results about their behaviour of recrystallization. *Ann Occup Hyg*, 38: Suppl 1713–722.
- Toeniskoetter RH, Schafer RJ (1977). *Industrial hygiene aspects of the use of sand binders and additives*. In: *The Working Environment in Iron Foundries*. Birmingham, United Kingdom: British Cotton Industry Research Association, pp. 19–1–19–28.
- Tola S, Koskela RS, Hernberg S, Järvinen E (1979). Lung cancer mortality among iron foundry workers. *J Occup Med*, 21: 753–759. doi:10.1097/00043764-197911000-00010 PMID:512720
- Triger DR, Crowe W, Ellis MJ *et al.* (1989). Trace element levels in the blood of workers in two steel works and a non-ferrous plant handling lead and cadmium compared with a non-exposed population. *Sci Total Environ*, 78: 241–261. doi:10.1016/0048-9697(89)90037-5 PMID:2717924
- Westberg H, Löfstedt H, Seldén A *et al.* (2005). Exposure to low molecular weight isocyanates and formaldehyde in foundries using hot box core binders. *Ann Occup Hyg*, 49: 719–725. doi:10.1093/annhyg/mei040 PMID:16126762

