III. HEALTH AND SAFETY HAZARDS

A. Introduction

Foundry workers may be exposed to many potential health and safety hazards [4,8]. These potential hazards along with their health effects and exposure limits are summarized in Appendix B. Sand-handling, sand preparation, shakeout, and other operations create dusty conditions exposing the worker to free silica. Chipping and grinding operations to remove molding sand which adheres to the casting may create a dust hazard in the foundry cleaning room area [7]. Mechanical sand removal aids, such as abrasive blasting machines, that operate on the principles of impact or percussion create high noise levels [13,26]. Foundry workers may be affected by the heat produced during melting and pouring operations [26]. In addition, the handling of molten metal and manual handling of heavy materials contribute to the burns and musculoskeletal illnesses and injuries suffered by foundry workers.

Respiratory disorders, particularly silicosis, are among the most commonly reported occupational health effects in foundry workers. As early as 1923, Macklin and Middleton [27] found that 22.8% of the 201 steel-casting dressers examined had pulmonary fibrosis. In 1936, Merewether [28] reported that after 10 years of employment, seven sandblasters of metal castings had died from silicosis at an average age of 40.7 years. After 8 years of employment, 16 sandblasters had died from silicosis complicated by tuberculosis. The average age at the time of death was 44.2 years. Unless sandblasting of castings was conducted in an enclosed chamber that allowed the operator to remain outside, the worker could not work at the trade for more than 1-2 years without serious lung disease. In the United States, Trasko [29] (using state records) identified 12,763 reported cases of silicosis during 1950-56. Of all the industries having a silicosis hazard, 16% of the total identified cases occurred in the foundries as compared to 66% in the mining industries and 18% in the pottery, brick, stone, talc, clay, and glass industries combined. Although foundry operations and conditions have changed considerably for the better since these early historical studies, there are a number of more recent studies [30,31,32,33,34] which indicate that silicosis still occurs. Recent comprehensive epidemiologic studies on the prevalence of fibrotic lung disease in foundry workers are lacking; however, data from NIOSH Health Hazard Evaluations (HHE's) [35,36,37,38] and recent Occupational Safety and Health Administration (OSHA) consultation visits [39] show that silica levels exceeding the NIOSH recommended exposure limit (REL) and the OSHA permissible exposure limit (PEL) do occur in both ferrous and nonferrous foundries, creating a potential increased risk of silicosis for foundry workers.

An increased risk of lung cancer among foundry workers has been shown in a number of studies [30,31,40,41,42,43,44,45]. Based on 1931 census data, the relationship between occupation and cancer deaths in the Sheffield, England foundries was studied in a population of approximately 178,600 male workers over 14 years of age and retired workers. Of all occupations, the furnace

and foundry workers had the highest mortality rate from lung cancer; the lung cancer deaths were 133% above the expected rate (126 observed vs. 54 expected) [45].

The potential for lung cancer is not merely of historical interest. In the recent reports of Egan et al. [31,41], an increased risk of death from cancer of the trachea, bronchus, and lung was reported among foundry workers with a proportional mortality ratio (PMR) of 176 for black and of 144 for white workers, ($p<0.01$ for both). Statistically significant increases in deaths for respiratory tuberculosis with a PMR of 232 for white workers ($p<0.05$) and in deaths for nonmalignant respiratory disease with a PMR of 138 for white workers ($p<0.01$) and of 151 for black workers ($p<0.05$), were also reported. These findings were based on an analysis of the death certificates of 2,990 foundry workers who had died between 1971-75 and who had paid monthly union dues from at least 1961 until the time of death or until receiving a 45-year life membership card. Histories of smoking and occupational exposure to carcinogenic agents, which are important causative factors in lung cancer, were lacking. Processes and materials which have been introduced into foundry technology over the past 20-30 years complicate the problem of identifying potential etiologic carcinogenic agents [31,41,46].

In a recent comparison of the relative risk of death from lung cancer among a cohort of 1,103 nonfoundry and 439 foundry workers, the overall death rate and incidence of neoplasms were not significantly increased, but risk of death from lung cancer was five times higher in the foundry workers with a standardized mortality ratio (SMR) of 250 and $p<0.01$ [30]. A 1981 review of the lung cancer data in foundry workers showed that particularly the molders, metal pourers, and cleaners have a two- to threefold increased risk of death from lung cancer [47].

The working conditions in foundries are further complicated by the safety hazards which may be confronted on a daily basis by foundry workers. These conditions have resulted in minor, as well as major, traumatic injuries and deaths. The incidence rate of lost workday cases of disabling injuries and illnesses in California foundries in 1975 per 100 workers was almost three times that in manufacturing as a whole [48]. National Safety Council (NSC) [49] data also indicate that foundries have higher injury and illness rates than other industries (Table III-1). In 1980, similar high accident rates were reported for the Ohio foundries in 1980 [50]. Statistical studies of foundry injuries show that foundry workers have a wide range of on-the-job injuries such as loss of limbs, burns, strain and overexertion, and foreign particles in the eyes [48,50,51,52]. Based on these data, foundries were designated by OSHA as a high hazard industry and selected as a first project under the National Emphasis Program (NEP) [53].

Studies of health effects presented later in this chapter show that in addition to being at risk for developing certain chronic respiratory diseases such as silicosis and lung cancer, foundry workers may be exposed to health hazards which could result in carbon monoxide poisoning, metal fume fever, respiratory tract irritation, dermatitis, and other illnesses.

TABLE III-1. Recordable occupational injury and illness incidence rates by industry, reported to the National Safety Council, 1981-83*

Industry	SIC[a] code	Incidence rates per 100 full-time workers: Total recordable cases	Total lost workday cases	Cases involving days away from work and death	Nonfatal cases without lost workdays	Total lost workdays
Iron and steel foundries	332	12.51	5.11	2.86	7.40	83
Nonferrous foundries	336	9.41	4.09	2.65	5.32	74
Construction	15-17	10.51	4.01	3.54	6.48	71
Mining	10-14	6.13	1.99	1.73	4.13	58
Manufacturing	20-39	6.82	2.70	1.93	4.11	51
All Industries	--	6.75	2.98	2.22	3.77	54

[a]2- and 3-digit Standard Industry Classification (SIC)

*Adapted from reference [49]

B. Health Hazards in Foundries

The potential health hazards present in the working environment of foundries are dependent upon a number of factors. Among these are the types of processes employed and the materials used in each process, including the type of metal cast, size of castings produced, sand-to-metal ratio, molding material bonding agents used, engineering controls, ventilation, building design, etc.

Health hazards in foundries include: (1) chemical hazards such as silica and other nonmetallic dust, metal dusts and fumes, carbon monoxide, and other chemical compounds including thermal decomposition products; and, (2) physical hazards associated with various foundry processes such as noise, vibration, and heat.

1. Chemical Hazards

a. Silica and Other Nonmetallic Substances

Crystalline silica dusts present the greatest and most widespread hazard to the health of foundry workers. Silica is silicon dioxide (SiO_2) that occurs both in a crystalline form, in which molecules are arranged in a fixed pattern, and in an amorphous form where

molecules are randomly arranged. The fine silica dust in foundries and other industries is produced by rubbing, abrading, or mechanical action on quartz sand, which is composed primarily of crystalline silica. Quartz sand is the main molding material in iron and nonferrous foundries and in many steel foundries. Silica refractories are used to line many foundry furnaces and ladles. When quartz is heated, the crystalline structure slowly changes to produce tridymite (above 860°C) and cristobalite (above 1470°C), which are considered even more fibrogenic to the lungs than quartz [54]. In 1983, OSHA established PEL's for cristobalite and tridymite which are one-half that for quartz [55].

The major foundry operations that produce fine particle-size silica dusts are sand-mold preparation, removing the castings from the mold, and cleaning the castings. A large quantity of dust arises from cleaning with pneumatic chisels and portable grinders and during abrasive blasting and tumbling. Molding and coremaking operations are less dusty, especially when damp or chemically-bonded sand is used. Preparing and reclaiming sand and repair and maintenance of process equipment are also potentially hazardous in terms of crystalline silica dust producing diseases [7,19]. An increased hazard has been created in the past by the coating of molds, patterns, and cores with finely divided high silica-containing dry powders and washes [56]. The extent to which crystalline silica exposure creates a significant hazard in a given foundry depends upon the size and type of the foundry, the arrangement of processing within it, the adequacy of dust controls, and the standards for housekeeping and other work practices [57].

The fibrotic reaction of the lung tissue to the accumulation of respirable crystalline silica is a pneumoconiosis known as silicosis [58,59,60,61,62]. The onset of this disease is slowly progressive. Usually after several years of exposure to silica dust of respirable size (<10 micrometers in diameter), the worker may develop fibrotic changes in the lungs and may become progressively more breathless, often developing a persistent cough. As the fibrosis progresses, it produces abnormalities which on the x-ray film appear as nodules that ultimately may coalesce. The silicotic lung is more susceptible to infections, particularly to tuberculosis, and may lead to cardiopulmonary impairment and cardiac failure [59]. Other dust-related lung disorders, such as benign siderosis, may be confused on the x ray with the diagnosis of silicosis [63].

Other refractory materials are also used in foundry operations. In some cases, usually in steel foundries, asbestos has had a limited application in riser sleeves and in the lining of furnaces and ladles [31]. Talc (of unspecified composition) is a silicate sometimes used as a parting agent in many foundries. Talc appears to be less fibrogenic than crystalline silica and is generally regarded as a safer substitute for the fibrogenic silica flour [19] unless the talc is contaminated with asbestiform fibers. Other

refractories, such as silicates, alumina, mullite, sillimanite, magnesia, and spinel, are considered unlikely to constitute a serious hazard to foundry workers [19], but little research has been done on these compounds.

Other sands are used with silica sand for special casting purposes. For example, steel foundries use zircon or chromite sands to prevent metal penetration at the mold-metal interface. Zircon and olivine sands have not been studied to determine their fibrogenic effects in humans.

b. Metal Dusts and Fumes

Metal dusts may be released into the foundry environment during the charging of the furnaces and the cleaning of castings. Metal fumes are emitted during melting and pouring processes, sometimes in large quantities, when one component metal has a lower boiling temperature than the melt temperature.

Lead (Pb) is a hazard in those foundries where it is used in the melt or is present in contaminated scrap, but the hazards from Pb or Pb contaminated dust and fume exist principally in nonferrous foundries producing leaded bronzes. Early symptoms of Pb poisoning are nonspecific and may include fatigue, pallor, disturbance of sleep, and digestive problems. Individuals may also develop anemia and severe abdominal pain from Pb colic. Central nervous system (CNS) damage, peripheral neuropathy, or kidney damage may occur [58,59,64].

Inhalation of freshly formed oxides of some metals may give rise to metal fume fever, otherwise known as brassfounders' ague, Monday fever, or foundry fever. Although metal fume fever is most commonly associated with the inhalation of zinc oxide fumes, other metals or their oxides, including copper and magnesium [58,65,66], may cause this condition. The syndrome usually begins with a metallic-like taste in the mouth followed by a dry throat, fever, and chills accompanied by sweating, generalized aches and pains, and fatigue, all of which usually disappear within 24-48 hours. This tolerance to metal fumes tends to be lost quickly, and the symptoms commonly reappear when the individual returns to work after a weekend or after a holiday [19,58].

Some metals to which the foundry worker may be exposed are either known or suspected carcinogens. Certain forms of chromium VI [61,67] (used as a trace alloying element) have been found to increase respiratory cancer mortality among workers; nickel and beryllium (used as a nonferrous alloy) are potential human carcinogens [61,68] (see Appendix B).

c. Carbon Monoxide

Carbon monoxide (CO), produced by the decomposition of sand binder systems and carbonaceous substances when contacted by the molten

metal, is a common and potentially serious health hazard in foundries. It may be produced in significant quantities during preheating of the furnace charge, melting or pouring, ladle or core curing, or from any other source of combustion, including space heating equipment or internal combustion engines; it may also evolve from indoor settling ponds for cupola or scrubbers [69,70,71].

Carbon monoxide quickly combines with blood hemoglobin to form carboxyhemoglobin which interferes with the oxygen carrying capacity of the blood, resulting in tissue anoxia. Symptoms of CO poisoning may include headache, dizziness, drowsiness, nausea, and vomiting [70,71].

d. Other Chemical Hazards

Other chemicals which are present in the foundry environment can have adverse health effects. Numerous chemical compounds or their decomposition products may result from binding agents, resins, and catalysts used in sand molds and cores. Additional emissions may be generated by paints, oils, greases, and other contaminants present in scrap metal and other materials introduced into the melting furnace [13,15,69,72,73]. Data on the potential health hazards of some chemicals, chemical binding systems and their emissions, and foundry processes are listed in Table III-2 for a simulated mold pouring. These data do not represent actual breathing zone samples collected from workers. Data are listed in Tables III-3 and III-4 and Appendix B for coremaking [72,74].

(1) Amines

Triethylamine (TEA) and dimethylethylamine (DMEA) are used as catalysts in a cold-box system. These amine catalysts are volatile and flammable, and vapors may present a safety hazard. TEA exposure in industry can result in eye and lung irritation as well as halo vision at high TEA concentrations [15,69].

(2) Ammonia

Ammonia (NH_3) is produced during core curing of nitrogen-containing organic binders and during thermal decomposition of hexamethylenetetramine catalyst. It is also formed when pouring molten metal into cores of nitrogen-containing organic binders [13,15,69,75,76]. Ammonia is extremely irritating to the eyes and respiratory tract and in high concentrations may result in chronic lung disease and eye damage [58,61,69,77]. Continued worker exposure to a high concentration is intolerable.

(3) Benzene, Toluene, and Xylene

Decomposition of organic materials used during metal-pouring operations may produce a wide variety of aromatic compounds including benzene [72]. Chronic benzene exposures may cause

TABLE III-2. Hazard evaluation of potential chemical emissions during simulated foundry molding

Chemicals	Green sand	Dry sand	Sodium silicate ester	Core oil	Alkyd isocyanate	Phenolic urethane	Phenolic no-bake	Low N2 furan H3P04	Med N2 furan TSA	Furan hot box	Phenolic hot box	Shell
Carbon monoxide (30 min)	A	A	A	A	A	A	A	A	A	A	A	A
Carbon dioxide (30 min)	B	B	B	B	B	B	B	B	B	B	B	B
Sulfur dioxide	B	B	C	C	C	C	A	B	B	C	C	B
Hydrogen sulfide	B	B	C	C	C	C	B	B	B	C	C	C
Phenols	B	C	C	C	C	B	B	C	C	C	C	C
Benzene	B	B	B	B	B	B	B	C	B	B	B	B
Toluene	B	C	C	C	B	B	B	C	B	C	C	B
Meta-Xylene	C	C	C	C	B	B	C	B	B	C	C	C
O-Xylene	C	C	C	C	B	B	C	B	C	C	C	C
Naphthalene	C	C	C	C	C	C	C	C	C	C	C	C
Formaldehyde	C	C	C	C	C	C	C	C	C	C	C	C
Acrolein	C	C	C	C	C	C	C	C	C	C	C	C
Total aldehydes (Acetaldehyde)	C	C	C	C	B	C	B	C	B	C	C	C
Nitrogen oxides	B	C	C	C	B	C	C	C	B	B	B	C
Hydrogen cyanide	C	C	C	C	B	B	C	B	B	B	B	B
Ammonia	C	B	C	C	C	C	C	C	B	A	A	B
Total amines (as Aniline)	C	C	C	C	C	B	C	C	B	B	B	B

A = Chemical agent present in sufficient quantities to be considered a definite health hazard. Periodic monitoring of concentration levels in workplace recommended.

B = Chemical agent present in measurable quantities, considered to be a possible health hazard. Evaluation of hazard should be determined for given operation.

C = Chemical agent found in minute quantities--not considered a health hazard under conditions of use.

Adapted from reference [72]

TABLE III-3. Airborne emissions from chemically-bonded thermosetting systems during mixing, molding, and coremaking*

Chemicals	Core oil	Furan hot box	Phenolic hot box	Shell	Carbohydrate
Carbon monoxide	?	?	?	?	?
Formaldehyde	0	?	X	X	?
Hydrogen cyanide	0	0	0	?	0
Ammonia	0	0	0	X	0
MDI	0	0	0	0	0
Sulfur dioxide	0	0	0	0	0
Hydrogen sulfide	0	0	0	0	0
Phenols	?	0	?	?	0
Benzene	?	?	?	?	0
Toluene	?	?	?	?	0
Furfuryl alcohol	0	?	?	0	0
Methanol	0	?	?	0	?

*NOTE: Assuming normal ventilation, optimized binder usage, and proper handling of binder system.

? = Possibly present in working environment--depending upon specific formulation and sand quality.

0 = Not expected to be present in sufficient quantities to be considered a health hazard.

X = Present in sufficient quantities to be considered a possible health hazard.

Adapted from reference [74]

TABLE III-4. Airborne emissions from chemically-bonded "no-bake" systems during mixing, molding, and coremaking*

Chemicals	Furan no-bake H3P04(PA)	Furan no-bake (TSA)	Phenolic no-bake (TSA)	Alkyd urethane	Phenolic urethane	Sodium Silicate (carbon dioxide or ester)
Carbon monoxide	0	0	0	0	0	0
Formaldehyde	?	?	X	0	?	0
Hydrogen cyanide	0	0	0	0	0	0
Ammonia	0	0	0	0	0	0
Aromatic amines	0	0	0	?	?	0
MDI-Isocyanates	0	0	0	0	0	0
Sulfur dioxide	0	0	?	0	0	0
Hydrogen sulfide	0	?	?	0	0	0
Phenols	?	?	X	0	?	0
Benzene	0	?	?	0	0	0
Toluene	0	?	?	0	?	0
Furfuryl alcohol	?	?	0	0	0	0
Methanol	0	?	?	0	0	0

*NOTE: Assuming normal ventilation, optimized binder usage, and proper handling of binder system.
0 = Not expected to be present in sufficient quantities to be considered a health hazard.
? = Possibly present in working environment--depending upon specific formulation and sand quality.
X = Present in sufficient quantities to be considered a possible health hazard.
TSA = Toluene sulfonic acid.
PA = Phosphoric acid.

Adapted from reference [74]

blood dyscrasias, convulsions, ventricular fibrillation, chromosomal aberrations, aplastic anemia, and leukemia. Acute exposure may result in CNS depression and skin irritation [58,78]. Because benzene may induce progressive malignant disease of blood-forming organs (leukemogenic), NIOSH has designated benzene as a carcinogen [78]. Xylene and toluene may be used as solvents in core wash materials [69]. Exposure to high concentrations of toluene may result in impaired muscular coordination and reaction time, mental confusion, irritation of the eyes and mucous membranes, and transient liver injury [58,61,79]. Exposures to high concentrations of xylene may produce CNS depression, minor reversible liver and kidney damage, corneal vacuolization, and pulmonary edema [58,80].

(4) Chlorine

Chlorine (Cl_2) used as a degassing agent with nonferrous alloys, mostly aluminum, is extremely irritating to the eyes and respiratory tract. In acute, high concentration exposures, Cl_2 acts as an asphyxiant by causing cramps of the laryngeal muscles; pulmonary edema and pneumonia may develop later [58,61,81].

(5) Diphenylmethane Diisocyanate (MDI)

Polymeric polyisocyanates (of the MDI type) are used in urethane cold-box and no-bake binder systems. Inhalation exposure is most likely to occur during pouring, cooling, and shakeout. MDI is irritating to the eyes, respiratory tract, and skin and may produce bronchitis or pulmonary edema, nausea, vomiting, and abdominal pain. Sensitization may occur under high exposures and may cause an asthmatic reaction [58,82].

(6) Formaldehyde and Other Aldehydes

Formaldehyde may be combined with urea, phenol, or furfuryl alcohol to form resinous binders used in shell, hot-box, and no-bake coremaking and no-bake molding [15,83]. Formaldehyde is also a constituent of resinous binders used for phenolic urethane and furan-sulfur dioxide (SO_2) cold-box processes.

Formaldehyde and other volatile aldehydes are strong irritants and potential sensitizers to the skin, eyes, and respiratory tract. Short-term exposure to high concentrations may produce pulmonary edema and bronchitis. Contact dermatitis and allergic sensitization may also develop [58,84]. Formaldehyde is designated a potential human carcinogen by NIOSH [85].

(7) Furfuryl Alcohol

Furfuryl alcohol is added to urea-formaldehyde resins to form a modified furan resin in hot box coremaking and is the feedstock

for formulating furan no-bake binders [13]. Exposure to furfuryl alcohol may result in lacrimation, bronchitis, mild sore throat, and allergic contact dermatitis [86].

(8) Hexamethylenetetramine

Hexamethylenetetramine (Hexa) is used as a curing agent and catalyst in shell molding [13,15,69]. It is a mild skin irritant. Side effects from ingestion include urinary tract irritation, digestive disturbances, and skin rash [58].

(9) Polycyclic Aromatic Hydrocarbons (PAH's)

Polycyclic Aromatic Hydrocarbons (PAH's) such as benzo(a)pyrene, naphthalene, and perylene are produced by low-temperature, destructive distillation during pouring of iron into green-sand molds [13,22]. Coal-tar fractions containing mixed PAH's have been shown to be carcinogenic when applied to the skin of experimental animals [87], and benzo(a)pyrene is considered to be a human carcinogen [88]. High naphthalene exposure may result in erythema, dermatitis, eye irritation, cataracts, headache, confusion, nausea, abdominal pain, bladder irritation, and hemolysis [58].

(10) Sulfur Oxides and Hydrogen Sulfide

Sulfur dioxide (SO_2) and other sulfur oxides may be formed when high sulfur content charge materials are added to furnaces, usually cupolas [22,69]. Sulfur dioxide is found as an emission during magnesium casting [89], in some core-curing operations, and in the sulfur dioxide-furan cold-box processes [15,69]. Gaseous sulfur dioxide has a strong suffocating odor. Long-term chronic exposure may result in chronic bronchitis and severe acute over-exposure may result in death from asphyxiation. Less severe exposures have produced eye and upper respiratory tract irritation and reflex bronchoconstriction [58,61,69,90].

Hydrogen sulfide (H_2S) can be formed from water quenching of sulfurous slag material. Catalysts based on arylsulfonic acids used in phenol-formaldehyde and furan binders also produce SO_2 and H_2S emissions during pouring [15,75]. Hydrogen sulfide exposures can produce eye, respiratory tract, and lung irritation; headache; dizziness; sensory impairment; sleep disturbance; loss of appetite; and death from paralysis of the respiratory centers in the brain [58,61,91].

2. Physical Hazards

a. Noise

The NIOSH criteria document on noise [92] states that exposure to daily noise levels above 90 dBA for an 8-hour time-weighted average

(TWA) may cause hearing loss and, may trigger changes in cardiovascular, endocrinologic, neurologic, and other physiologic functions. Hazardous noise levels are produced in many foundry operations, with levels over 105 dBA's being recorded near tumbling mills, arc furnaces, molding machines, sand shakeout, grinding, and combustion flame areas [7]. In addition to the possibility of temporary or permanent hearing loss, high noise levels may cause difficulty in verbal communications and in hearing warning signals or emergency commands.

b. Vibration

Foundry workers may be subject to whole-body vibration from shakeout, sand-slinging, forklift trucks, conveyors, overhead cranes, pneumatic ramming tools, and jolt-squeeze machines. Hand-arm, or segmental, vibration occurs from using hand-held power grinders, chippers, and other pneumatic tools [7,93,94].

Whole-body vibration may induce increased pulmonary ventilation and oxygen consumption, increased gastric secretions and intestinal motility, and marked changes in skeletal structure [58]. Hand-arm vibration is a more localized stress that may result in Raynaud's phenomenon otherwise known as Vibration White Finger. Symptoms include blanching and numbness in the fingers; decreased sensitivity to touch, temperature, and pain; and loss of muscular control. Chronic exposure may result in gangrenous and necrotic changes in the finger [58,95].

c. Heat

Both radiant and convective heat generated in the foundry during the melting and pouring of metal creates a hot environment for these and other foundry operations. The heating of molds and cores and the preheating of ladles are additional heat sources. Workers engaged in furnace or ladle slagging and those working closest to molten metal, including furnace tenders, pourers, and crane operators, experience the most severe exposures [7,48]. Molten metal and hot surfaces that exist in foundry operations create a potential hazard to workers who may accidentally come in contact with hot objects. Besides the direct burn hazard caused by hot objects, environmental heat appears to increase the frequency of accidents in general [96].

During the first week or two of heat exposure, most, but not all, healthy workers can become acclimatized to working in the heat. However, acclimatization can also be lost rather rapidly; a significant reduction in heat acclimatization can occur during a short vacation or a few days in a cool environment [58,97]. The health effects of acute heat exposure range in severity from heat rash and cramping of the abdominal and extremity muscles, to heat exhaustion, heat stroke, and death. Chronic exposure to excessive heat may also result in behavioral symptoms such as irritability, increased anxiety, and lack of ability to concentrate [58,97].

d. Nonionizing Radiation

Both ultraviolet (UV) and infrared (IR) radiation pose potential health hazards, especially to the skin and eyes. Radiation from molten metal around cupolas and pouring areas and the arc in electric furnaces can produce inflammation of the cornea and conjunctiva, cataracts, and general skin burns [2,58,98]. Other problems associated with exposure to UV radiation can include synergistic interactions with phototoxic chemicals and increased susceptibility to certain skin disorders including possible skin cancer [58,98]. UV radiation is also present in other foundry operations such as welding and arc-air gouging.

IR radiation from molten metal may produce skin burns and contribute to hyperthermia. Although there is no evidence that IR alone will cause cancer, it may be implicated in carcinogenesis induced by some other agents [99].

C. Epidemiologic and Other Foundry Studies of Adverse Health Effects

1. Respiratory Disease in Foundry Workers

The most commonly reported respiratory disorder among foundry workers who are exposed to crystalline silica and mixed dust exposures is pneumoconiosis. Also, the incidence of bronchiogenic lung cancer is believed to be higher among foundry workers.

a. Pneumoconiosis

The term, pneumoconiosis, literally means dust in the lungs. However, because not all dusts deposited in the lungs will result in recognized lung diseases, pneumoconiosis has been given medically significant definitions which have differed somewhat with time. In the 1965 24th edition of Dorland's Illustrated Medical Dictionary pneumoconiosis is defined as, "a chronic fibrous reaction in the lungs to the inhalation of dust." In the 1981 26th edition, the definition was expanded to, "a condition characterized by permanent deposition of substantial amounts of particulate matter in the lungs, usually of occupational or environmental origin, and by the tissue reaction to its presence." The 1981 revision better defined the deposition of particulate matter (dust), in which not all types of dust lead to significant fibrotic lung tissue reactions. Based on the type of deposited particulate matter, the nononcogenic lung tissue response can be divided into fibrotic, nonfibrotic, or mixed tissue reactions [58]. The general category of pneumoconiosis is also divided according to the dust involved, e.g., silicosis (silica), siderosis (iron), asbestosis (asbestos), coal workers pneumoconiosis (coal), berylliosis (beryllium), and byssinosis (cotton dust).

The clinical diagnosis of pneumoconiosis is based mainly on: (1) the history of exposure; (2) the symptomatology; (3) the lung x-ray findings; and, (4) pulmonary function tests [58]. None of

these approaches provide sufficient information for diagnosing a specific type of pneumoconiosis; therefore, radiographic evidence and the history of exposure are fundamental for a diagnosis [58,100].

For the chest x rays to be useful, it is necessary that a standard classification of radiographic changes be adopted and utilized in all clinical studies. This was not the case in the past. Not until the International Labor Organization (ILO) U/C classification was adopted in 1972 has a simple reproducible system for recording radiographic changes in the lungs been available [58,60,101]. The most recent ILO U/C classification system is the 1980 ILO version [102].

The lack of a standard system for describing radiographic changes in lung structure has made it difficult to compare data presented in early studies of pneumoconiosis in foundry workers with the data presented in recent studies.

Silicosis is the most prevalent and the most serious of the fibrogenic pneumoconioses seen in foundry workers. Its pathogenesis and pathology are not different from the silicosis found in any other group of workers exposed to excessive levels of respirable free silica. The primary causative agent is crystalline silica dust deposited in the lungs [54,56,58,61]. The severity of the fibrotic response in silicosis is generally proportional to the level of fine respirable silica dust exposure and the number of years of exposure [54,56,100,103].

Early studies of pneumoconioses in foundry workers provided the basis for worker compensation for pneumoconiosis in the industry both in the United States and abroad. In 1923, Macklin and Middleton [27] reported the first large-scale investigation in England of chest disorders in foundry workers. Based on clinical examinations of 201 steel casting dressers surveyed, 22.8% had pulmonary fibrosis. At that time, fettling or cleaning was done mainly with hand tools rather than with pneumatic tools, and the authors emphasized that even then fettlers were exposed to large amounts of dust. Later, the use of pneumatic tools created more dust, increasing the potential for silicosis among fettlers [56].

Because of an expanding awareness of the problem of silicosis among foundry workers in the United States, several studies on pulmonary fibrosis in foundry workers were carried out (Table III-5). In addition to these compensation studies, other studies were done in the United States and abroad to evaluate workers' health in individual foundries.

In other studies [104,297], only workers with the longest periods of exposure were selected. These early studies varied greatly not only in the numbers of workers examined but also in the types of foundries observed. In some investigations, only workers employed in a specific foundry occupation, such as cleaning of castings, were

TABLE III-5. Prevalence rates for pulmonary fibrosis as reported in early studies of foundry workers

Year of report	Investigators	Location of study	Type of foundry	Numbers of workers examined	Percent of workers with pulmonary fibrosis diffuse	nodular	Ref.
1931	Komissaruk	Austria	Iron	40	N/A	30	[104]
1933	Landau	Germany	Iron, steel	126	N/A	69	[105]
1934	McConnell and Fehnel	Wisconsin	Iron, steel	210	13.3	31.4	[297]
1934	Pope and Zacks	Massachusetts	Not stated	Not stated	11.4	N/A	[103]
1935	Warfield	Wisconsin	Iron	691	N/A	17.5	[106]
1936	Kuroda	Japan	Steel	314	26	8	[107]
1937	Osmond	Pennsylvania	Iron, steel	686	15.1	10.1	[108]
1937	Kelley and Hall	New York	Not stated	N/A	7.5	1.2	[100]
1938	Sander	Wisconsin	Iron, steel	4,035	N/A	7.0	[109]
1938	Trice and Easons	North Carolina	Iron, steel, nonferrous	546	8.2	1.0	[110]
1938	Greenburg, Siegal, and Smith	New York	Iron	1,960	4.7	3.7	[111]
1939	Sander	Wisconsin	N/A	8,377	21.0	5.8	[112]
1942	Brown and Klein	U.S.	Steel nonferrous	454	3.7	2.4	[113]
1945	Keatinge and Potter	Britain	Iron	60	22	N/A	[114]

N/A - Not available

Adapted from references [56,115]

examined [27,105,116]. Some studies did not include chest x rays; for example, Macklin's findings [27] were based on clinical examination alone. In other studies, x rays were not taken of the entire study population; Kuroda [107] x rayed only 314 of the 715 workers examined. In some cases, the population studied was small; Komissaruk [104] examined 40 workers in one foundry.

In evaluating the data and comparing the findings of these early studies, variations in x-ray techniques and classifications must be considered, especially in the borderline cases. The reported prevalence in foundry workers of what was variously labeled as fibrosis, a term used for different lung structure appearances, varied from 1.5 to 24%; the reported prevalence of stage I silicosis varied from 1 to 40%; and the presence of stage II and III silicosis varied from 1 to 65%. These and other studies carried out in 13 different countries reported the presence of silicosis in foundry workers [56]. It must be concluded from these early studies that foundry workers throughout the world suffered from dust diseases of the lungs and that certain foundry jobs (e.g., shakeout and cleaning castings) were more hazardous as judged by the prevalence, severity, and complication of lung diseases. The highest incidence of silicosis, ranging from mild to severe and disabling was found among casting cleaners [27,56,105,106,108,109,111,115,117].

In 1950, two major studies of pneumonconiosis in foundry workers were published, one in Great Britain by McLaughlin [56] and the other by Renes et al. [115] in the United States. McLaughlin's report included the results of clinical, spirometric, and radiographic examinations of 3,059 workers (2,815 men and 244 women) in 19 foundries (iron, steel, and mixed iron and steel). Each x ray was viewed at least four times by each of three observers. By majority vote, the films were categorized as (I) normal, (II) early reticulation, (III) marked reticulation, or (IV) nodulation and opaque or massive shadows. A complete occupational history was taken, and a family history and previous health record were noted with particular reference to tuberculosis. The physical examination included measurement of the chest girth and expansion, exercise tolerance tests, and in one foundry, measurements of tidal air and vital capacity. In order to attribute prevalence rates to different environments, and thereby assess the risk of one occupational group against another, the data were standardized for age and length of exposure. Of the 244 women, 242 had normal chest x rays and for this reason they were omitted from the statistical analyses. When the data from all occupational groups for all foundries were combined, 71% showed no abnormal x-ray changes, 17% showed category II changes, 10% showed category III changes, and 2% showed category IV changes. However, when the data for the three categories of foundries, iron, steel, and mixed, were examined separately, steel workers showed a statistically higher prevalence ($p<0.001$) of category III changes (16%) than did the iron and mixed iron and steel workers (6%). The difference was calculated to be significant

even when corrections were made for the differences in age and length of exposure in the three groups. For category II changes, the incidence data were similar for all three types of foundries.

When the workers were subdivided into the broad occupational groups of (1) molding shop workers, (2) fettling shop workers, and (3) other workers, the molding shop workers had a prevalence of severe x-ray abnormalities (x-ray categories III and IV) of 13% in steel foundry workers vs. 7% in workers in both iron and nonferrous foundries. For fettlers, the prevalence of severe x-ray abnormalities was 34% in steel foundries compared with 12% in iron and 13% in mixed foundries. The higher prevalence of the more severe x-ray abnormalities (categories III and IV) among steel workers for all occupations combined was essentially a feature of work in the molding and fettling shops, and of the two, the fettling shop operations were the most hazardous. Steel melt pouring temperature is higher than iron melt temperature and results in more sand fracturing and silica dust production.

The overall conclusions of the McLaughlin [56] study indicate that foundry workers are at a substantial risk of developing silicosis and lesser forms of pneumoconioses and that steel foundry workers are at higher risk than iron foundry workers. The most marked radiographic changes were most frequently seen in all workers in the fettling shops, mainly among fettlers, and shot blasters.

In 1950, Renes et al. [115] reported on a 1948 and 1949 survey conducted in 16 ferrous foundries which were considered representative of the 185 Illinois foundries surveyed. Occupational and medical histories were taken from 1,937 of the 2,000 workers employed in these foundries. Chest x rays of 1,824 workers were classified by the classification recommended at that time by the U.S. Public Health Service (PHS). Significant pulmonary fibrosis of occupational origin was identified in 9.2%; 7.7% were ground glass 2 stage (classification D) and 1.5% nodular (classifications E, F, and G). Nodular pulmonary fibrosis occurred with about equal frequency in the steel and gray iron foundrymen. The classification 0, and E, F, and G are roughly comparable to the "ground glass" and "nodular" classes (See Table III-6). In general, it required 14 or more years of exposure to develop nodular silicosis in the foundry industry. The prevalence of nodular silicosis was 0.1% under 10 years of exposure, 1% for 10-19 years, and 5% for 20 or more years of exposure. The only diagnosis greater than nodular stage 1 was in the group with 20 years or more exposure. In this long-exposure group, an additional 20.9% showed ground glass 2 changes. Symptoms were considered to be of minor significance in the instances of pulmonary fibrosis observed in this study. Nodular pulmonary fibrosis occurred predominantly among the molders in the gray iron foundrymen and among the cleaning and finishing workers in the steel foundrymen.

The Renes et al. and the McLaughlin studies [56,115] had certain similarities in numbers and types of foundries surveyed. These two studies remain among the best in regard to the interpretation of radiographic findings. They both have used two or more x-ray readers and have recognized the problem of intra- and inter-observer variation.

The "categories" of the British survey [56] and "classifications" of the U.S. survey [115] are not strictly comparable as shown in Table III-6. Age and length of exposure would have to be taken into account for a more meaningful comparison of incidence of x-ray abnormalities.

Most of the early studies on the hazards of foundry work have related mainly to ferrous foundries; some of the larger studies do not specify the foundry types. Greenburg [111] x rayed 347 workers in 17 nonferrous foundries and found that 2.2% had fibrosis and 2.8% had silicosis vs. 4.7% and 2.7% in iron foundry workers and 5.5% and 3.7% in steel foundry workers. Of the 215 foundry workers x rayed by McConnell and Fehnel [297], only five were employed in nonferrous foundries; one x ray showed nodulation and one showed fibrosis. In both cases, the worker was employed in the molding department.

In 1959, Higgins et al. [118] described the results of a random sample of 776 men in Staveley, England, including 189 foundry workers or former foundry workers. The workers were divided into two age groups: 25-34 and 55-64 years of age. No reason was given by the investigators for selecting only these two age categories. Based on radiographic evidence, 23% of the foundry workers 55-64 years of age had pneumoconiosis, while none of the workers in the 25-34 age group had pneumoconiosis.

In 1970, Gregory [119] reported an analysis of chest film surveys conducted from 1950 to 1960 of about 5,000 workers employed in steelworks in Sheffield, England, of which 877 were employed in one large steel foundry. Medical surveillance was conducted during the last 6 years of the 10-year study. Pneumoconiosis was diagnosed based on chest x rays and occupational histories. During the 6 years from 1954 to 1960, the prevalence rate of silicotic nodulation in all steel foundry workers was 6.4%. A higher prevalence rate for pneumoconiosis was found in workers in the fettling shop (14.7%) than in workers in the main foundry area (2.0%). The average time of exposure to crystalline silica before the development of nodulation was about 31 years for workers in the fettling and grinding shops and 36 years for workers employed in the main foundry. Workers exposed to crystalline silica before the age of 25 averaged a longer period at work before showing nodulation (36 years at work) than did workers who were first exposed after 25 years of age (23 years at work). The author was unable to relate the observed development of pneumoconiosis to specific exposure levels.

TABLE III-6. Interpretation of x-ray abnormalities

McLaughlin		Renes	
Categories	**Results (%)**	**Results (%)**	**U.S.P.H.S classification**
I (Normal)	71	68.2	A, B (normal, linear exaggeration 1 and 2)
II (Early reticulation)	17	21.6	C (ground glass one)
III (Reticulation)	10	7.7	D (ground glass two)
IV (Nodulation and/or massive shadows)	2	1.5	E, F, G (nodulation, conglomerate masses)

A,B = No definite sign of dust exposure
C = First degree ground glass appearance
D = Second degree ground glass appearance
E,F,G = Disseminated coalescence nodules and increasing size of conglomerate shadows

Adapted from references [56,115]

However, it was noted that the average time of exposure before the development of silicotic lesions seemed to be increasing, suggesting some gradual improvement in foundry environmental conditions.

In 1971, Davies [33] reported on respiratory disease among British foundrymen, including the prevalence of pneumoconiosis. The 2,427 foundries were divided into four categories: those estimated to employ 1-9, 10-49, 50-249, or >250 workers. A sample of 1 in 40 of each foundry size group was selected using tables of random sample numbers. The 66 selected foundries employed a total of 1,997 foundry workers, of whom 1,780 (93%) were included in the study; they were matched for age, height, and weight with 1,730 other factory workers used as controls. However, chest roentgenograms were obtained on only 1,308 foundry floormen, 352 fettlers, and 321 from the nonfoundry group. Of the foundry workers, 1,015 foundry floormen and 179 fettlers were accepted as not having been exposed to dust except in a foundry, and they provide the roentgenogram data base. Chest x rays were taken on the nonfoundry workers only when clinical reasons warranted it. The roentgenograms were read by three experienced readers using the National Coal Board version of the ILO (Geneva 1958) Classification. Category I was accepted as evidence of the existence of obvious pneumoconiotic changes. Category I pneumoconiosis was found in 12% of the foundry floormen and 23% of the fettlers. In Category II, 1.3% of the floormen and 11% of the fettlers developed the disease. In Category III and above, the rates were 0.3% for foundry floormen and 0.6% for fettlers. The degree of pneumoconiosis was related to years of foundry work and to job classification. Although this study primarily investigated chronic bronchitis, the quality of its design and execution provides a good estimate of the prevalence of silicosis in foundry workers and it confirms the greater risk of silicosis for workers who clean castings.

In 1972, Clarke [32] reported on the examination of 1,058 retired male workers from a large iron foundry. There were 76 workers with x-ray signs of pulmonary silicosis (26 in grade 1 and 50 in grade 2). Of these 76 workers, nine had decreased physical ability and a forced vital capacity (FVC) that was less than 48% of the predicted values; three had lung cancer. No data were provided on the total population from which the 1,058 retirees were selected.

The earlier studies of pneumoconiosis in foundry workers were essentially prevalence surveys of radiographic abnormalities in the workers examined rather than in the entire population at risk. Several authors have commented on the practice of transferring workers who showed x-ray evidence of pneumoconiosis to less dusty work areas, thereby excluding them from later surveys and artificially reducing the observed prevalence [56,111,115,119].

Data on the progression in the severity of pneumoconiosis in individual foundry workers are sparse. Sander reported no visible progression over a period of 4 years [112]; Keatinge reported

progression in only three cases over a 9-year period [114]; Gregory [119] suggested that even removal from exposure to crystalline silica dust did not necessarily prevent the development of silicosis. However, in general, survey data indicated a trend toward more severe x-ray abnormalities with increasing age, age at first starting foundry work, and the number of years of exposure [34,56,115,120]. Dust exposure data with which to correlate the trends were generally lacking.

The question of the progression of pneumoconiosis as expressed by lung x-ray abnormalities, with continued exposure in foundry workers, was the thrust of the study by the Subcommittee on Dust and Fumes of the British Joint Standing Committee on Health, Safety, and Welfare in Foundries [34,120]. In 1958, a chest x-ray survey of iron foundry workers was conducted [120]. In 1968 the foundry workers from the same group who showed evidence of pneumoconiosis in 1958 were again given chest x rays.

Among the iron foundry workers who had chest x rays in 1958, 238 showed evidence of pneumoconiosis Category 1 (early reticulation) or above (11.5%). In the 1968 survey, the 1958 films were reexamined and all those showing Category 1 pneumoconiosis or above were selected for further study. The 176 selected cases were given a chest x ray and each pair of 1958 and 1968 films was compared to assess progression, if any, of pneumoconiosis during the 10 years of foundry work. Radiologic readings found that 48 of the 176 cases had progressed during the 10 years. The authors caution that the data "may provide a guide to the foundry population in general but it is unreliable in providing representative material when broken down into the (work category) groups used for this study" [34].

The amount of progression of pneumoconiosis was, in the above study, estimated "as the amount that a man's radiological pneumoconiosis would increase if he works for 10 years in the job." Progression was expressed as a "fraction of the width of Category 1." The rate of progression cannot be used as an index of the severity of the pneumoconiosis. The rate of change differed between foundries and between jobs within the foundry. In general, the rate of progression was highest among the knockout and fettling workers who, on the average, progressed about one-third to one-half of an x-ray category in ten years. This translates into a progression of radiological reading of one category in 20-30 years (e.g., from category 1-0 to 2-0, or from 2-0 to 3-0 in 30 years).

Pulmonary function data, corrected for age and height on the workers studied in 1968, provided no evidence that early radiologic pneumoconiosis is associated with reduced ventilatory capacity. On the other hand, reduction in ventilatory capacity was associated with smoking history--being greatest in workers who smoked more than 15 cigarettes a day [34]. These studies support the observations that the prevalence of pneumoconiosis is associated with the foundry job category, the number of years of exposure, the age of the

worker, and the age of the worker when starting foundry work. The rate of progression of radiologic pneumoconiosis is also probably associated with the same set of factors. Smoking cigarettes increases the risk of incurring pulmonary function impairment.

b. Chronic Bronchitis

The comparative assessment of the prevalence of chronic bronchitis among foundry workers in different countries and foundries is confounded by the varying diagnostic criteria and definitions used by investigators. In the past, the term "chronic bronchitis" usually meant any chronic respiratory or pulmonary condition associated with a cough and not ascribable to other recognized causes [33]. Some authors have been more specific in their definition by including sputum and breathlessness lasting over most of the year [119] or chest illness causing absence from work during the past three years [118]. The most recently accepted criterion for chronic bronchitis includes cough with phlegm which occurs on most days for at least three months a year for three consecutive years [121].

British national statistics indicate that foundry workers and miners have suffered an excess mortality and morbidity rate caused by bronchitis as compared with other workers. SMR's for bronchitis were also high for foundry workers' wives which suggests etiologic factors besides occupation [44,118].

In 1959 and 1960, Higgins et al. [118,122] published the results of a prevalence study of chronic bronchitis and respiratory disability in a 776-man (92% response rate) random sample of the 18,000 population of an English coal-mining and industrial town. An occupational and residential history and a respiratory symptom questionnaire were completed for each worker. Pulmonary function tests such as the forced expiratory volume in 0.5 second (FEV 0.5), maximum breathing capacity (MBC), forced vital capacity (FVC), and a chest x ray were obtained. The population studied was divided into two age groups, 25-34 and 55-64, for comparison of data. In age group 25-34, workers with no occupational exposure to dust had persistent cough and sputum in 16%, while in foundrymen the prevalence was 19%. For symptoms of chronic bronchitis, the prevalence was 2% and 6%, respectively. In age group 55-64, persistent cough and sputum were present in 32% of nondusty workers, 30% of foundrymen without pneumoconiosis, and 36% of foundrymen with pneumoconiosis. Mean MBC was 143 and 140 liters per minute (L/min) in the 25-34 year age group for nondusty trade workers and foundrymen, respectively. For the 55-64 year age group, MBC was 90 L/min for nondusty trade workers, 85 L/min for foundrymen without pneumoconiosis and 82 L/min for foundrymen with pneumoconiosis. The small numbers of subjects in some of the cells made statistical comparisons unreliable.

Although the results of the study are essentially negative, the care taken in the selection of the study population, the stratification of the random selection into age and occupation groups, and the comparisons made between the groups demonstrated the difficulties in the etiology of bronchitis.

Some other British investigators failed to demonstrate that foundry workers suffer a greater prevalence of chronic bronchitis than the general population in an industrial area [119,123].

In 1965, Zenz et al. [124] analyzed pulmonary function in three occupational groups employed in a diversified manufacturing company. Of the workers studied, 64 worked in the iron foundry, 61 were clerks, and 81 worked in the machine shop. All of the workers had a minimum of 20 years of service. Included in the pulmonary function analysis were tests for FVC, FEV_1, Maximal Expiratory Flow Rate (MEFR), and Maximal Mid-Expiratory Flow (MMF). No statistically significant differences in pulmonary functions were found between groups nor between smokers and nonsmokers in the three occupational groups.

Higgins et al. [118] reported a significant increase in the prevalence of persistent cough and sputum and decrease in MBC as related to the cigarette smoking experience in the group studied. For nonsmokers, light smokers, heavy smokers, and exsmokers, the prevalence of cough and sputum was 9, 22, 44, and 13%, respectively; grade 2 or over chronic bronchitis 4, 7, 8, and 13%, respectively; and MBC 145, 140, 133, and 143 L/min, respectively, in the 25-34 year age group. For the 55-64 year age group the prevalence was 3, 39, 52, and 21%, respectively, for cough and sputum and 3, 20, 22, and 13%, respectively, for chronic bronchitis. The MBC was 101, 87, 80, and 89 L/min, respectively.

In 1976, Koskela et al. [125] compared the prevalence of health problems in current and past employed foundry workers. A questionnaire was completed by 1,576 current foundrymen, 493 workers whose foundry employment terminated after they had worked for at least 5 years, and 424 workers who had worked in foundries for less than 1 year. The frequency of chronic bronchitis was similar among both current and former foundrymen: 16 and 14%, respectively, in nonsmokers; 29 and 23%, respectively, in smokers with slight or medium dust exposure; and 28 and 31%, respectively in smokers with high dust exposure. The authors concluded that chronic bronchitis was associated with exposure to dust among the current foundrymen and that chronic bronchitis may be a reason why older (55-64 years) workers leave foundry work. Results from the pulmonary function tests indicated that smoking was a major factor in the reduction in lung function.

In the Davies study [33], the "sputum-breathlessness" syndrome was found to be significantly more prevalent in foundry workers than in the control group of engineering factory workers (25% of foundry

floormen, 31% of fettlers, and 20% of control workers). However, when the prevalence is standardized for smoking history, the prevalence was 20% for foundry floormen, 22% for fettlers and 22% for the control engineering factory workers. The prevalence ratio of the "sputum-chest illness" syndrome among nonsmoking foundry workers was 2.5 times that in the nonsmoking control workers. However, when the heavy smokers are compared, the ratio falls to 1.2. The prevalence ratio of "sputum-chest illness" syndrome increased with the number of years of foundry employment to approximately 1.58 after 15 years of foundry work as compared to the control group. Prevalence of this syndrome increased with smoking history in all the groups studied, and the combination of foundry work and smoking gave the highest prevalence rate.

In 1974, Mikov [126] reported the results of a retrospective investigation of the prevalence of respiratory symptoms, including chronic bronchitis, among the workers in five nonmechanized foundries in the Province of Vojvodina, Yugoslavia. The definitions and criteria of the Commission for the Aetiology of Chronic Bronchitis of the MRC were used. A completed questionnaire on respiratory symptoms, complete clinical examinations, and chest x rays were obtained. The data from the 535 workers studied (95% response rate) were matched with those from a control group consisting of 244 workers who worked at other jobs in the workshop but who did not experience unusual exposure to airborne pollutants in the working environment. The two groups were carefully matched for social and economic status (but not for smoking history). The prevalence of chronic bronchitis among the foundry workers was 31.03%, while the control group had only 10.26% ($p<0.001$).

The epidemiologic data do not prove a clear relationship between chronic bronchitis and foundry exposure. In 1971, at the ILO International Conference on Pneumoconiosis--IV in Bucharest, Rumania a working group concluded that "occupational exposures to dust may also be one factor among several more important ones in the aetiology of chronic bronchitis. In the present state of our knowledge there is insufficient evidence that chronic bronchitis may be considered an occupational respiratory disease of workers exposed to dust" [101].

A possible explanation for the apparent divergence of findings between different investigators may be their failure to clearly state whether they were discussing chronic simple bronchitis (chronic mucus hypersecretion) or chronic obstructive bronchitis (chronic airway obstruction). Parkes concluded that there is evidence that chronic simple bronchitis is related to the inhalation of dust and some toxic gases, but there is no evidence that chronic obstructive bronchitis is directly or consistently attributable to such exposures in foundries [121].

c. Lung Cancer

Evidence for an increased risk of lung cancer among foundry workers has been derived mainly from mortality data. These data may contain several serious problems such as (1) death certificates and autopsy reports that may contain only the record of occupation at the time of death and may not reflect previous occupations and their associated exposure to potential cancer producing substances and (2) smoking histories are usually lacking. The potential bias introduced in epidemiologic studies by different smoking behavior may be substantial since it has been estimated that the incidence of lung cancer in men would be significantly reduced in the absence of cigarette smoking [47]. In evaluating the lung cancer risk studies, the positive and negative biases inherent in such studies must be kept in mind.

The Registrar General's study from 1930-32 summarized by Doll in 1959 [43] reported that, in England and Wales, "metal molders and coremakers" (SMR=155, observed 158), and "iron foundry furnacemen and laborers" (SMR=142, observed 17; SMR=131, observed 136, respectively) ranked fourth and fifth in the list of occupations with the highest mortality rates from lung cancer. The highest death rates for lung cancer among the workers in Sheffield, England were reported to occur among foundry workers, smiths, and metal grinders [45]. It was suggested that iron in certain forms might promote the development of cancer [127,128].

The results of two series of autopsy studies reported by McLaughlin [56] and McLaughlin and Harding [42] showed a higher-than-expected frequency of lung cancer among ferrous foundry workers, many of whom also had accompanying siderosis. An overall prevalence at death of 10.8% of carcinoma of the bronchi was much higher than would be expected from the prevalence in the general population. The authors speculated that mineral oil, soot, crystalline silica, and fumes resulting from the pyrolysis of organic oils and binders in the foundry environment may have contributed to the increased incidence of lung cancer in the workers studied.

With respect to crystalline silica, very little has been established regarding the role of quartz containing dusts in the induction of lung cancer in foundry workers, primarily because exposure to such dusts is frequently concomitant with exposure to low concentrations of volatile carcinogens such as polyaromatic hydrocarbons (PAH) or other suspect carcinogens, e.g. chromium and nickel, that are found in foundry atmospheres. While data presently available from human exposures indicates that exposure to crystalline silica dusts alone does not lead to an increased incidence of lung cancer. Thus, until adequate human studies show otherwise, it is prudent to recommend avoidance of exposure by foundry workers to combinations of crystalline silica dusts and any concentration of airborne carcinogens, known or suspect [129,130].

Estimates of lung cancer prevalence rates based on selected cases among workers employed in several industries were published in 1971. A prevalence of lung tumors among foundry workers of 9.6 tumors/1,000 workers vs. 4.7 tumors/1,000 in a nonindustrial population was based on seven such tumors in an unspecified population of foundry workers. The foundries from which the populations at risk were drawn included iron, steel, and brass. However, the author stated that no specific carcinogens or other contributing variables had been identified that could be associated with this cancer prevalence rate. Only the lung cancer incidence rates in the asbestos and chemical manufacturing industries and in asbestos and anthracite coal mining exceeded the incidence rate in the foundries [131].

In 1976, Koskela et al. [40] studied the mortality experience of 3,876 men from a total of 15,401 workers who had at least 3 months of exposure in 20 iron, steel, and nonferrous foundries randomly selected for the Finnish Foundry Project. The age-adjusted mortality rate of foundry workers approached the expected level, with an SMR of 90 for all foundry workers and 95 for workers in typical foundry occupations, these slight deficits may in part be explained by the healthy worker effect. However, the lung cancer mortality for the entire group was higher than expected with an SMR of 175 (21 observed vs. 12 expected, $p<0.05$). The excess lung cancer deaths were confined to iron foundry workers, especially those with more than five years of exposure (SMR 270 $p<0.05$). Of 21 lung cancer cases, only one had never smoked; but the questionnaire suggested that the smoking habits of foundry workers were similar to those of the general population. The authors concluded that perhaps the foundry environment contained carcinogenic agents which require smoking as a cocarcinogen.

In 1977, Gibson et al. [30] described the results of a retrospective mortality study in which a group of 439 foundry workers employed in the foundry division of a Canadian steel mill was compared with 1,103 nonfoundry workers over a 10-year period beginning in 1967. Death certificates were obtained for all deaths in both groups, and each death was classified according to the International Classification of Diseases Adapted (ICDA). Total expected deaths in both groups was calculated from 1971 vital statistics for nearby metropolitan Toronto. Relative risk of lung cancer was significantly higher for foundry workers. The overall lung cancer SMR for foundry workers was 250 (8.4 expected vs. 21 observed). During this 10-year period, 21 of the foundry workers, or 4.8%, died of lung cancer, while 11 of the nonfoundry workers, or 1%, died of lung cancer. After age 45 a foundry worker was 5 times more likely to die of lung cancer than was a nonfoundry worker. Although the relative risk of dying from lung cancer was greater for foundry workers after the age of 45, the relative risk for total neoplasms and total deaths was not increased for foundry workers when compared with that for nonfoundry workers. In addition, there was a statistically significant ($p<0.005$) increase in lung cancer among foundry workers with more than 20 years of exposure to the foundry

environment as compared with foundry workers with fewer years of work exposure. SMR's from lung cancer were calculated by work categories as follows: crane operators, 714 (4 observed vs. 0.5 expected); finishers, 314 (4 observed vs. 1.6 expected); molders, 255 (4 observed vs. 1.6 expected); coremakers, 208 (2.6 observed vs. 0.96 expected); and electric furnace/open hearth operators, 114 (3 observed vs. 2.7 expected). Environmental samples showed airborne particulate concentrations to be highest for the finishing jobs. The benzene-soluble fraction of total suspended particulates varied among job categories and could not readily be related to increased lung cancer. The authors stressed that the absence of smoking histories on the entire population was a serious deficiency. The smoking histories sampled in 1976 showed that 58% of the foundry workers smoked cigarettes compared with 53% of the nonfoundry workers. Of the 24 individuals in the lung cancer group on whom smoking histories were obtained, 22 (93%) were smokers.

Egan et al. [31] and Egan-Baum et al. [41] reported on mortality patterns from the death benefit records of the International Molders and Allied Workers Unions (IMAWU). To be eligible for death benefits a worker had to be a union member prior to 1961 and must have paid monthly union dues until death or until a life membership card was obtained. The death records included both active foundry workers and retired foundry workers. For each of the 2,990 death records for the years 1971-75 used in the study (99.2% of total), the underlying cause of death was classified according to the 8th International Classification of Disease Adapted (ICDA) classification. Smoking histories were not available for this decedent population. The age- and race-specific cause distributions of all deaths among males in the United States for 1973 were used as the standards from which expected deaths were calculated. Each comparison between observed and expected numbers of deaths was summarized as a PMR. The statistical significance of differences between observed and expected numbers of deaths was determined by a Chi-square test. Of the total number of deaths, 2,651 were white males and 339 were black males. The distribution of deaths by age in foundry workers, in contrast to the distribution of all deaths in the United States for males above age 30, showed a slight over-representation for above age 75 (45% vs. 38%) and an under-representation for under age 45 (7% vs. 15%). Death due to malignant neoplasms was associated with a PMR of 110 (545 observed vs. 497.65 expected) for white males and a PMR of 124 (86 observed vs. 69.29 expected) for black males, both of which were statistically significant increases ($p<0.05$). Cancer of the trachea and bronchus, and the lung, more than accounted for this by contributing 86 excess cases; a PMR of 144 (224 observed vs. 155.17 expected) was reported in white males and a PMR of 176 (39 observed vs. 22.10 expected) was reported in black males, both PMRs were significant increases ($p<0.01$). Also, nonmalignant respiratory diseases were significantly increased with a PMR of 138 (277 observed vs. 200.40 expected) in white males ($p<0.01$) and a PMR of 151 (30 observed vs. 19.81 expected) in black males ($p<0.05$). This latter observation was in large part attributable to a sixfold

increase in pneumoconiosis with a PMR of 576 (30 observed vs. 5.21 expected) in white males ($p<0.01$) and a PMR of 1154 (3 observed vs. 0.26 expected) in black males (significance level not indicated due to small numbers). Additionally, in white males, while a decreased PMR of 73 was reported for the pneumonia and influenza death category, the remaining nonmalignant respiratory disease categories were associated with the following increased PMRs: "bronchitis" 140 (not significant), "emphysema" 159 ($p<0.01$) and "other respiratory diseases" 190 ($p<0.01$). These three categories for black males represented few deaths and therefore were not evaluated. Across all age groups, the PMR for heart disease was close to the expected, and mortality from nonmalignant respiratory diseases was higher than predicted for especially those over 65 years of age (PMR=144) with a moderate excess in persons 55-64 years of age (PMR=122). Excess lung cancer peaked at ages 60-64 (PMR=179).

In the most recent review of the epidemiologic literature on lung cancer in ferrous foundry workers, Palmer and Scott [47] concluded that there was a two- to threefold increased incidence of lung cancer associated with ferrous foundry work. The increased incidence was higher among the molders, casters, and cleaning room workers. The authors emphasized that these data reflect exposures that occurred years ago and that the cancer risk reflecting today's exposure may be quite different. The introduction of new foundry practices and molding materials could substantially change a specific foundry environment for better or for worse [47].

An apparent excess of lung cancer among foundry workers has been noted from a review of vital statistics [43,44,45], mortality studies [30,31,40,46], and other investigations [42,56,131]. The complexity and variety of foundry exposures, changing work forces, changes in work practices and molding materials, and inadequacy of occupational, medical, and smoking history documentation all hinder a definitive answer to the cause-effect relationship which the overall data on lung cancer in foundry workers strongly suggest.

Three recent review papers and one epidemiologic study support the earlier conclusions that the risk of lung cancer is increased in foundry workers [132,133,134,135]. In a 1983 review of the mortality experience of foundry workers, SMR's of between 147 to 250 were reported in nine different studies included in the review. In the four cohort studies included in the review, SMR's of approximately 200 were reported with one of the studies having an SMR of 250 [134].

In 1984 Fletcher and Ades [135] published the results of a study in which they followed the health experience of a cohort of male workers from England who had started foundry work between 1946 and 1965 and had worked in a foundry at least one year. The cohort was followed prospectively until 1978. Of the cohort group, 7,988 were traced and alive, 1,858 were traced and dead, 173 had left England, and 231 could not be traced. Of the 1,858 deaths, details of cause of death were available on all except 14. Observed and expected

deaths were calculated and grouped by foundry, occupational category, and 5-year entry. No data on smoking habits of the cohort were collected. Mortality from lung cancer was increased among the foundry and fettling shop area workers (SMR's of 142 and 173, respectively, $p<0.001$). The authors commented that "the narrowness of the range of most of the risk estimates, approximately 1.5 to 2.5, is striking, as is the fact that of 12 investigations from which relative risk from lung cancer might be estimated for foundry workers, none of the risk estimates were close to or below unity."

2. Nonrespiratory Effects in Foundry Workers

a. Zinc Oxide

In 1969, Hamdi [136] observed 12 brass foundry furnace operators who had been subjected to chronic exposure to zinc oxide fumes. Ten unexposed subjects were also studied. Determinations of zinc (Zn) concentrations in the plasma, red blood cells, whole blood, and urine were made for each worker and control subject. Zinc concentrations were also determined in the gastric juices of eight workers and seven controls. No environmental data were reported. The author found a significant increase in Zn concentration in the red blood cells, whole blood, and fasting gastric juices of the exposed foundry workers as compared to the control group. The absorbed Zn appeared to be rapidly eliminated through the gastrointestinal and urinary tracts, with excess Zn being stored in the red blood cells. The author speculated that elevated Zn concentrations in gastric fluids in the exposed workers might account for the high incidence of gastric complaints reported [136]. However, there are no sufficient data to link Zn levels in body fluids to any specific system disorder [58,61].

b. Inorganic Lead

Although many epidemiologic studies on the health status of workers exposed to lead (Pb) workers have been made, few have included foundry workers. On the basis of blood analysis, Stalker found that 79% of 98 brass foundry workers examined showed excessive Pb absorption. For this study, a high concentration of Pb in the blood was defined as one greater than 70 micrograms per deciliter (μg/dl) whole blood [137]. By comparison, NIOSH in 1978 [138] determined that unacceptable absorption of Pb and a risk of Pb poisoning are demonstrated at levels >80 μg/dl of whole blood. Stalker analyzed the blood of 24 of the workers who had had urinary Pb values above 150 μg lead/liter of urine or stippled erythrocyte counts above 1,000 per million red blood cells. These workers had a blood Pb level of 120 μg/dl. Followup physical examinations of 75 of the foundry workers revealed that 50% exhibited symptoms indicative of a mild "alimentary type of lead poisoning." However, the kind and incidence of symptoms in a group of 25 workers with high urinary leads did not differ significantly from the group as a

whole [137]. The most frequently occurring symptoms included excessive urination at night (nocturia), gingivitis, headache, constipation, vertigo, and weight loss.

Neurobehavioral effects of Pb exposure have recently been reported for 103 foundry workers. Sixty-one non-lead exposed assembly plant workers were used as the control group. The blood Pb levels in the foundry workers averaged 33.4 μg/dl (range 8-80) and 18.6 μg/dl (range 8.3-36) for the non-lead exposed group. Lead concentrations in the foundry and assembly plant air were not given. Neurobehavioral changes as measured by the Profile of Mood (POM) states were strikingly increased in the workers with blood Pb levels above 60 μg/dl. The foundry workers' POM scores, using the control group score as 100, were for those with blood Pb levels above 60 μg/dl, tension 155, anger 200, fatigue 135, depression 180, confusion 175 [139]. Blood Pb levels of over 60 μg/dl are considered to indicate exposure to unacceptably high levels of inorganic Pb. Normal blood lead levels range from 10-60 μg/dl [140]. The OSHA safety and health standard 29 CFR 1910, establishes an 8-hour TWA PEL for Pb of 0.05 mg/m^3 [141] and requires monitoring of blood Pb levels at least every 2 months for workers whose last blood sample indicated a blood Pb level at or above 40 μg/dl of whole blood.

c. Carbon Monoxide

Carbon monoxide (CO) is one of the most frequent contaminants in foundry air. In 1976, Hernberg et al., [142] surveyed 931 workers from a sample of 20 foundries for angina pectoris, electrocardiographic (ECG) changes, and blood pressure levels. Occupational and morbidity histories and smoking habits were obtained (53% were smokers). The prevalence of angina pectoris among the factory workers was increased over background for all workers, but was highest among smokers. The prevalence of angina for nonsmokers was 2% in workers without occupational CO exposure and 13% for those with CO exposure. For smokers the prevalence of angina was 15% for those without occupational CO exposure and 19% for those with CO exposure. Rate ratios failed to demonstrate a statistically significant increase in angina rate among nonsmokers due to CO exposure. The ECG showed no systematic increase in abnormality as a function of smoking and/or CO exposure. This may have resulted from the ECG's being taken while at rest and not under maximum CO exposure or levels of physical work; whereas the occurrence of angina pectoris was considered positive irrespective of whether symptoms had occurred under maximum work or conditions of CO exposure. Casters and furnacemen with CO exposure had higher systolic ($p<0.05$) and diastolic ($p<0.01$) blood pressures when compared to other occupational groups. When blood pressures of nonsmokers without occupational CO exposure were compared to blood pressures of smokers with occupational CO exposure, diastolic blood pressures were significantly higher ($p<0.05$) in those occupationally

exposed to CO. The study did not include a nonfoundry control population; ECG's were taken only when workers were at rest; and heat, as a confounding variable, was not analyzed.

d. Beryllium

Beryllium (Be) and its compounds can be highly toxic [58,69,143]. The acute effects are mainly on the respiratory tract with cough, shortness of breath, and substernal pain. Chronic effects may become progressively more severely disabling with pulmonary insufficiency and right heart failure [58,143].

Although beryllium may be present in some foundries, its use is relatively limited. Air concentrations of Be were measured over a 7-year period in a modern copper-beryllium alloy foundry [144]. The general air and breathing zone concentrations of Be exceeded the NIOSH REL of 0.5 micrograms per cubic meter ($\mu g/m^3$) [98] and the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value (TLV®) of 2 $\mu g/m^3$ [88] in more than 50% of the air samples. However, no cases of chronic beryllium-induced disease were found [144]. Evidence for linking Be exposure to the development of a chronic respiratory disease (berylliosis) was reviewed by NIOSH with the conclusion that berylliosis would not occur at Be exposure levels at or below 2 $\mu g/m^3$ [143].

e. Chemical Binders

As a result of the strong evidence that foundry workers are at an increased risk from lung cancer, a search for carcinogenic or potentially carcinogenic substances in the foundry environment has recently been conducted. In particular, the polyaromatic hydrocarbons (PAH's) have been suspected.

Schimberg reported finding approximately 50 PAH compounds in the foundry air dust [145]. The benzo(a)pyrene (BaP) concentration in the air was much higher (mean 4.9, range 0.01-57.5 $\mu g/m^3$) in those foundries where a coal-tar sand-molding material was used than in those where a coal dust/sand mixture was used (mean 0.08, range 0.01-0.82 $\mu g/m^3$). The concentration of BaP also varied with the dust-particle size ranging from 0.3-5.0 $\mu g/m^3$ for dust >7.0 micrometers in diameter to 9.7-16.5 $\mu g/m^3$ for dust <0.5 micrometer.

Mutagenicity studies on material extracted from larger sized dust (>7.0 micrometers) showed relatively large direct acting mutagens with more of the indirect acting mutagen on the smaller sized dust (<1.1 micrometer). The authors concluded that the direct acting mutagens are other than PAH compounds and that BaP level is not a "reasonable marker for mutagenic activity" [145].

The emissions from four types of mold binders (furan, urethane, shell, and green sand) have been analyzed for the presence of carcinogens. They were analyzed for 16 metals, eight PAH's, and

five phenols. The PAH substances present in the highest concentrations in the water insoluble fraction were phenanthrene, benz(a)anthracene, and benzo(a)pyrene, with the concentrations lowest in the furan and urethane binders (0.6-230 $\mu g/g$) and highest with the shell and green-sand binders (200-7200 $\mu g/g$) [146].

In 1982, NIOSH [147,148] reported the levels of several airborne contaminants present in the core- and mold-making and metal-pouring areas of a steel-casting foundry. The diphenylmethane diisocyanate (MDI) concentrations ranged from below 0.042 to 0.173 ppb (0.43 to 1.77 $\mu g/m^3$) (average 0.082 ppb), all of which were far below the NIOSH REL of 50 $\mu g/m^3$; the formaldehyde concentration averaged 0.29 ppm (0.36 mg/m^3) with a highest value of 0.41 ppm (0.50 mg/m^3); dimethylethylamine (DMEA) concentrations ranged from 1.18 to 7.45 ppm (4.2 to 26.5 mg/m^3); trace metals were not present in significant amounts (ranging from none detected to 0.35 mg/m^3 for iron and 0.136 for manganese); CO averaged 82 ppm (94 mg/m^3) for metal skimmers, 50.6 ppm (58 mg/m^3) for pourers, and 9.6 ppm (11 mg/m^3) in the general pouring area--exceeding the NIOSH REL and the OSHA PEL for the skimmers and pourers; ammonia concentrations averaged 5.6 ppm (4 mg/m^3) in the coremaking area, hydrogen cyanide less than 0.9 ppm (1 mg/m^3), and aromatic amines below 1 mg/m^3; crystalline silica concentrations of 120 to 140 $\mu g/m^3$ were found in breathing-zone samples in the shakeout operations--exceeding the NIOSH REL of 50 $\mu g/m^3$ [147].

Concentrations of some contaminants in breathing-zone samples of air in the coremaking (shell, phenolic urethane, and bench processes) area of a foundry were included in a 1984 NIOSH Health Hazard Evaluation report [149]. The mean concentrations found were as follows: ammonia, not detectable; DMEA, 0.34 to 0.65 ppm (1.2 to 2.3 mg/m^3); formaldehyde, 0.24 to 0.73 ppm (0.3 to 0.9 mg/m^3); and acrolein, furfuryl alcohol, Hexa and MDI, none. Formaldehyde was the only one of the contaminants measured whose concentrations were considered potentially hazardous. Crystalline silica was not measured.

Crystalline silica content in dust was found in 116 Japanese foundries to average 16% of the 0.67 mg/m^3 of respirable dust. These levels were considered unacceptably high. Control measures would be required to reduce levels 140 $\mu g/m^3$ of respirable dust with not more than 13.6% crystalline silica to meet the Japanese acceptable environmental levels [150].

Ermolenko et al. [151] reported on the health of coremakers in the foundry of an automobile manufacturing plant in the U.S.S.R. Environmental data were also taken in two-binder system operations in which the coremakers used furfuryl-alcohol-modified carbamide-formaldehyde (KF-90) and phenol carbamide-formaldehyde (FPR-24) resins. Seven air contaminants were found within the breathing zones of those coreroom workers who operated single- and two-stage coremaking machines, who mixed sand for the process, or

who finished the core. These contaminants were formaldehyde, methanol, furfural, ammonia, furfuryl alcohol, CO, and phosphoric acid. Concentrations of formaldehyde reached 1.2 ppm (1.5 mg/m^3) and methanol concentrations reached 3.97 ppm (5.2 mg/m^3) in areas where mixing of materials took place. Table III-7 shows mean concentrations of these compounds (ppm) at the breathing zones of workers who operated coremolding machines.

TABLE III-7. Mean air concentrations (ppm) for coremolding machine operators

	KF-90 binder		FPR-24 binder	
	Single-stage machine	Two-stage machine	Single-stage machine	Two-stage machine
Formaldehyde	5.0	1.8	3.7	2.7
Methanol	16.4	2.5	4.0	3.7
Furfural	0.13	0.008	0.05	0.01
Ammonia	8.9	2.4	1.4	0.43
Furfuryl alcohol	2.3	1.8	0.84	Not detectable
Carbon monoxide	Trace	10.9	Trace	10.9
Phosphoric anhydride (oxide)	0.02	0.01	N/A	N/A

N/A - Not available

Adapted from reference [151]

Except for formaldehyde, the breathing zone concentrations of the substances did not exceed any exposure standard or recommended guideline. Higher concentrations of emissions were present with the single-stage electrically heated core machines than with the two-stage gas-heated machines. The thin-walled single-stage cores probably underwent thermal decomposition and volatilization throughout rather than just on the external surface layer as with the two-staged cores. The gas flames may have helped burn the decomposition products as they evolved. The KF-90 binder may have produced higher concentrations of decomposition products because it has lower thermal stability and the formaldehyde used in its synthesis contained 5-11% methanol. Other sources for air contaminants included containers holding core rejects and inspection tables on which cores lay for cooling. Breathing zone levels of

formaldehyde at those places averaged 3.7 to 2.7 ppm (4.5 and 3.3 mg/m^3) for the single- and two-stage machines, respectively, for binder FPR-24 and 6.2 and 2.2 mg/m^3, respectively, for the KF-90 binder [151].

Formaldehyde concentrations in the breathing zone of coremakers in many samples exceeded the OSHA PEL (8-hour TWA limit) of 3 ppm (3.7 mg/m^3), some exceeded the acceptable ceiling limit of 5 ppm (6.1 mg/m^3), and none exceeded the maximum 30-minute ceiling limit of 10 ppm (12.3 mg/m^3). Total daily exposure time was not given; consequently 8-hour TWA's could not be calculated [151].

To determine the effect of job-related factors on the Russian coremaker's health, 138 workers (125 women and 13 men) were examined and questioned for health effects (no control groups used for comparisons). Of these 138 workers, about half were under 30 years old and most had worked at their jobs from 1 to 5 years. Complaints included frequent throat inflammation (68%), nasal congestion (25%), dryness of nose and throat (20.4%), hoarseness (20.4%), and acute irritation of the upper respiratory tract (63%). Chronic rhinitis was present in 47%, chronic tonsillitis in 31.8%, and chronic pharyngitis in 18%. These studies illustrate some of the respiratory problems that may be associated with the use of chemical binders in the foundry industry. The breathing zone air concentrations of formaldehyde to which these workers were exposed ranged from 0.49 to 8.15 ppm (0.6 to 10 mg/m^3) [151].

Formaldehyde has been reported in sufficient quantities to be considered a health hazard in the phenolic hot-box chemically-bonded thermosetting core system and in the chemically-bonded phenolic no-bake process [152]. Since formaldehyde is considered to be a potential human carcinogen, engineering controls and work practices should be utilized to reduce exposure to its lowest feasible level [85].

Formaldehyde air concentrations at coremaking operations were reported for several NIOSH Health Hazard Evaluations conducted since 1972 [147,148]. Formaldehyde concentrations exceeding 1.0 ppm were found in 3 of 14 samples in one of the foundries (4.4, 10.6 and 18.3 ppm); in the other 11 samples the concentrations were less than 1 ppm (<0.02-0.57 ppm). In the other foundries, formaldehyde concentrations ranged from <0.02-0.73 ppm.

The phenolic, furan, epoxy, and other resins (and their thermal decomposition products) used as binders in hot-box and no-bake mold and other coremaking can cause contact dermatitis and allergic dermatosis [15,58,69]. Although a dermatitis or dermatosis can result from contact with a single substance, several factors are generally involved [153].

Adverse medical symptomatology was elicited from workers in the coreroom of a ferrous foundry as part of a NIOSH Health Hazard Evaluation [154]. The sand cores were produced either by heating

the resin-coated sand or by the cold-box process. Automatic, electric-heated, and gas-fired core blow machines were in operation. At the time of the interviews, no adverse medical symptomatology such as eye and throat irritation was reported. However, symptoms typical of exposure to corebox gases and fumes (burning of the eyes, nose, and throat) were reported as having been experienced in the past.

f. Manganese

Foundry use of manganese (Mn) is mainly in iron and steel alloys and as an agent to reduce oxygen and sulfur content of molten steel [58,69]. Manganese dust and fumes may be a minor irritant to the eyes and respiratory tract. Chronic Mn poisoning can be an extremely disabling disease resembling Parkinsonism [58,155,156].

3. Thermal Stress and Strain

Foundry workers may be exposed to heat stress, particularly during the hot summer months. Thermal stress with Wet Bulb Globe Temperature (WBGT) levels of 30° to 50°C (80° to 122°F) have been measured in several foundry surveys [7,157,158,159,160]. At WBGT levels over 30°C the risk of incurring heat illness progressively increases [58,88,97,161,162], with the level of risk being higher for the heavier physical work. In those foundry studies where the level of physical work was measured, the 8-hour TWA metabolic rate was, for most jobs, 250 kcal/hr or less, which falls within the light to moderate physical work category [97,157,159,160]. This may account for the fact that heart rate, body temperature, sweat production, and fluid balance measurements on foundry workers have not indicated high levels of heat strain even when the environmental stress exposures were very high [157,159]. The amount of dehydration experienced by the foundry workers may approach critical levels [157,159,160]. Heat-related morbidity and mortality data on foundry workers are not available. An epidemiologic study of steel mill and foundry workers has implicated chronic heat exposure as a risk factor for cardiovascular and digestive disorders [163,164]. Even those who have worked at hot jobs with heat exposure below the ACGIH TLV [88] for 15 or more years had an increased incidence of digestive disease (excluding cirrhosis).

Several factors that may be involved in fatal heat stroke include relative obesity, dehydration, high environmental heat load, lack of acclimatization, and inadequate rest periods. Those working at hot jobs should be encouraged to take cooling breaks, drink sufficient liquids (water), and immediately report any feelings of not being well [165].

4. Auditory Effects

Noise levels during many operations in foundries are high and generally fall within the range of 85-120 dBA [7]. With proper engineering control and/or hearing protective devices, the actual exposure levels are usually below 90 dBA [7,166,167,168]. The noise levels at foundry operations without adequate engineering controls were found to be 108 to

433% above the OSHA PEL 8-hour TWA of 90 dBA [92]. The ACGIH TLV® of 85 dBA for an 8-hour TWA [88] was exceeded frequently even when engineering controls were in place [7].

Work stations in an integrated steel plant were monitored and studied by Martin et al. [166] to determine potential hearing loss among the foundry workers who were exposed to noise levels in the 85 - 90 dBA range. A total of 228 noise-exposed workers and 143 controls were tested. The average exposure noise level was 86 dBA for the slinger floor workers and 89 dBA for the electric furnace operators. The audiometers used in the testing were self-recording and manual types that conformed to ANSI Standard S3.6-1969 and were calibrated biologically and acoustically at regular intervals. The audiometer operator was a certified audio technician. The workers were tested at the start of the workshift to minimize temporary threshold shift effects. Workers were excluded for testing if they had worked in another noise area for more than three years, had more than a 40 dB hearing difference between ears at two or more frequencies (in which case only data from the better ear were used), or had been previously diagnosed for bilateral nonneurosensory hearing loss. The workers tested had not worn hearing protectors. The control group consisted of office staff workers having minimal occupational noise exposure. The workers tested were divided into four age groups of 18-29, 30-39, 40-49, and 50-65 years. A hearing level index (HLI) was computed as the average of the audiometric thresholds at 500, 1,000, and 2,000 Hertz (Hz). Hearing impairment was considered to have occurred when the HLI exceeded 25 dB. In general, the HLI increased with age (Table III-8), as also did the percentage of impairment (Table III-9). The "normalized" values showed that for electric furnace workers 50-65 years old, 32.5% had impaired hearing and that in slinger floor workers in this age group, 26.5% had impaired hearing compared with 10% of the controls.

The increased risk (percentage differences between the subject group and the control group) was 22.5% for the oldest electric furnace workers and 16.5% for the oldest workers on the slinger floor. These data indicate that an increase in hearing loss (corrected for age) can occur in some workers with occupational noise exposure in the dBA 85-90 range.

5. Chronic Trauma

In 1973, Mintz and Fraga [169] described eight cases of moderate-to-severe osteoarthritis of the elbow in foundry workers between 41 and 63 years of age who had been foundry workers for 8 to 21 years. Their work required the use of tongs 20 to 34 inches long to lift or twist metal rods, which produced large stresses and forces on the elbow joint. The main complaint was a limitation in the range of joint motion, rather than pain. The x-ray examinations revealed degenerative joint disease of the elbow. Similar changes in the elbow and wrist have been seen following prolonged use of pneumatic tools [170]. The observed changes were thought to be related to general stress and trauma at the joints rather than a specific foundry-related phenomenon.

TABLE III-8. Mean hearing level indices

Area	Age	Sample size	HLI	Statistically significant (p=0.05)
Floor slinger	18-29	25	9.4	S
	30-39	21	8.5	NS
	40-49	11	9.8	NS
	50-65	7	20.0	NS
Electric furnaces	18-29	23	7.8	NS
	30-39	16	8.7	NS
	40-49	12	12.1	NS
	50-65	13	21.4	S
Control Group	18-29	56	6.2	--
	30-39	36	8.4	--
	40-49	30	9.0	--
	50-65	21	10.5	--

S = significant
NS = nonsignificant
Adapted from reference [166]

TABLE III-9. Percentage with impaired hearing*

Area	Age	Sample size	% Impaired raw data	% Impaired "normalized"	Increased risk
Floor slinger	18-29	25	4.0 (1)	4.6	4.1
	30-39	21	0.0 (0)	1.8	0.3
	40-49	11	0.0 (0)	2.5	2.0
	50-65	7	42.8 (3)	26.5	16.5
Electric furnaces	18-29	23	0.0 (0)	4.0	3.5
	30-39	16	6.3 (1)	2.3	0.8
	40-49	12	16.7 (2)	15.5	11.0
	50-65	13	46.2 (6)	32.5	22.5
Control group	18-29	56	0.0 (0)	0.5	--
	30-39	36	0.0 (0)	1.5	--
	40-49	30	3.3 (1)	4.5	--
	50-65	21	4.8 (1)	10.0	--

*HLI >25 dB
Adapted from reference [166]

Partridge et al. [171] interviewed 858 male workers in six iron foundries for rheumatic complaints. Only workers actively involved in the production of metal parts and finished products were included. The observed prevalence of rheumatic complaints which increased with age among the floor molders was 61.5% (104 observed vs. 68 expected). Floor molders were the only group of foundry workers that had a significantly increased Standardized Complaint Ratio (SCR 153, $p<0.001$). Average worker absence due to rheumatic causes was 0.44 weeks/year; this was not different from that in other industries such as brewery, mining, and dock workers. Neither the levels of heat or cold nor psychological factors appeared to be related to the prevalence of rheumatic complaints, absence because of illness, or other complaints.

6. Vibration Syndrome

It has been recognized for some time that foundry workers, especially chippers and grinders, who use hand-held vibrating tools, may incur the clinical condition of "Vibration Syndrome," also known as "Raynaud's Phenomenon of Occupational Origin" or "Vibration White Finger." The symptoms may range from blue, cold fingers to necrosis and gangrene of the finger tissue requiring amputation of the affected parts [95,172,173].

Agate et al. [93] reported on a study of Raynaud's phenomenon among grinding room workers in a British foundry. Based on statements by the workers, 27 of 29 men and 5 of 8 women reported signs of Raynaud's phenomenon. Twenty-three of the 37 developed the phenomenon in both hands. The men affected were from 29 to 50 years old, with a mean age of 37; women were from 24 to 45 years old, with a mean age of 36. The time between starting grinding work and the onset of symptoms ranged from 0 to 7 years, with a mean of 1.75 years. All attacks occurred after exposure to cold conditions. The duration of attacks varied from 10 to 180 minutes, often lasting until the hands became warm. Disability in these workers, which was difficult to assess due to inadequate diagnostic methods, appeared minimal. In a few cases, 1-2 hours of work were lost while the hands were being warmed. When pain occurred, it was most often associated with the return of blood flow to the affected fingers. Of the 12 workers who had stopped grinding, three claimed no improvement after as long as 5.5 years. Nine claimed improvement to some extent, and one even had a cessation of attacks one year after stopping such work.

Cold water immersion test 59°F (15°C) induced pallor or cyanosis of the fingers in 21 cases, while 10 others who allegedly had the phenomenon showed no abnormal responses. The size of the grinding wheel used appeared to be related to the number of finger segments affected. Those workers using small wheels had a mean of 7.7 finger segments affected while those using larger wheels had 13.7 segments affected ($p<0.05$). The duration of employment, compared with the number of segments affected (index of severity), showed a significant degree of association

($r=0.65$, $p=0.05$). The study reported no preventive measures that could be effectively utilized. Some workers used gloves or strips of cloth, but most did not. This study demonstrated the presence of an annoying, and in some cases a mildly disabling, condition resulting from exposure to segmental vibration.

Leonida [94] reported on the occurrence of Raynaud's phenomenon among workers in an Illinois gray iron foundry. Of the 2,030 workers examined over a 16-month period, 107 of 123 who currently used hand-held air hammers for 6.5 to 7 hours per day or had done so within 2 years were symptomatic, having white fingers, numbness, tingling, swollen hands, loss of grip, and painful shoulders and elbows. The remaining workers using air hammers were not affected. Of the 1,904 workers who did not use air hammers, 16 were symptomatic and the remaining 1,888 were not. The study showed that the risk of developing these symptoms was greatest among users of air hammers and less among other workers using other tools, including grinders.

In the same report [94], a study of recently hired chippers and grinders showed that during 76 months of follow-up, 33 of 144 chippers (22.9%) and 7 of 34 grinders (20.6%) became symptomatic. Two chippers had symptoms after 4 months of work, but the first symptomatic grinders did not show up until after 9 months. The author concluded that this demonstrates that a longer latent period exists for grinders, even though the percentage who were symptomatic after 16 months of exposure was the same. The implication was that all chippers used air hammers and that was the cause for the earlier occurrence of Raynaud's phenomenon. However, several other factors that may be related to the occurrence of Raynaud's phenomenon are: (1) physical condition and maintenance of the pneumatic tool; (2) length of chisel used on the chipping tool; and, (3) force used in holding the tool. The recent studies by NIOSH support these findings [173].

D. Injuries to Foundry Workers

While the health hazards (chemicals, dusts, gases, and fumes) to which foundry workers are exposed are of great concern, equally important problems confronting foundry workers are the daily exposure to safety hazards that may result in injury or death. Incidents such as the explosion that killed 4 and hospitalized 17 workers at Burnside Steel Foundry in February 1979 [174] continue to occur because of poor work conditions and practices, inadequate engineering controls, and improper or inadequate training. The OSHA National Emphasis Program (NEP) consultation service program, conducted in 1977-78 [39], estimated that each worker in a foundry is at risk to approximately one and one-half serious "safety hazards" and two other-than-serious "safety hazards." A "serious hazard" is defined as one that could result in severe injury or death. The incidence rate for lost workday injuries was 14.9 cases per 100 full-time iron and steel foundry workers, which averages about three times that of all manufacturing industries [48].

1. Potential Sources of Safety Hazards in Foundries

Foundry worker accidents can result in injuries from (1) manual materials handling, (2) machinery, (3) walking and working surfaces, (4) mechanical materials handling, (5) foreign particles in the eye, and (6) contact with hot material. Injuries in all of these operations have resulted in disability, dismemberment, or death to foundry workers.

a. Manual Materials Handling

Manual materials handling in foundries involves the moving by hand of castings, cores, molds, molten metal in ladles or other devices, or any other material. The amount of manual materials handling in a foundry is highly dependent on foundry size, age, and layout [7]. In general, the smaller, older, nonferrous foundries have heavy manual materials handling requirements [175]. Overexertion and poor lifting techniques are the most prevalent causes of injury to foundry workers, especially in coremaking, cleaning, and molding operations [48,50,176]. In addition, workers handling castings or process tools often receive traumatic injuries by being struck by or who come in contact with these objects. Burns are often received by workers while handling hot cores in coremaking processes or from molten metal during pouring, melting, and inoculation operations because of inadequate personal protective equipment and work practices [48].

b. Machinery

In the 282 foundries visited during the OSHA NEP consultation service program [39], an average of four instances was found involving improper machine guarding that could potentially cause worker injury. Molding and coremaking operations, utilizing automatic and semiautomatic machinery, presented hazards from moving machine parts and flying or ejected materials [48]. Improper maintenance, repair, guarding, and use of grinders and abrasive wheels may also result in worker injury.

c. Walking and Working Surfaces

Injuries resulting from falls from elevated work surfaces may result in more severe injuries than most other foundry accidents. These occur in charging areas of cupolas and during maintenance and repair of mixers, mullers, and furnaces. Poor housekeeping and poorly lighted areas may result in slips, trips, and other types of falls on walking and working surfaces [48].

d. Mechanical Materials Handling

Foundry operations require significant movement of both heavy and molten materials. As a necessity and labor-saving convenience, a variety of mechanical handling devices such as cranes, hoists, monorails, conveyors, forklifts, trucks, and electromagnets are used. Stress on crane components is greater under the elevated

temperatures found in a foundry operation than under normal temperatures. In addition, some of these devices are continuously vibrating, resulting in mechanical stress on nuts, bolts, chains, and cables which eventually may result in equipment failure. Such equipment failures may lead to major explosions, fires, spills, and burns [48].

e. Foreign Particles in the Eye

Because of the heavy use of abrasive actions for cleaning foundry castings, eye injuries occur frequently. Metal dust or chips may be propelled at workers during chipping, grinding, abrasive blasting operations, and abrasive wheel use. Ambient dust may also contribute to eye injury, unless adequate safety glass protection is provided [48].

f. Contact with Hot Material

The data pertaining to injuries from contact with hot materials are presented in Section III.D.2.c.

2. Statistical Data and Case Reports of Foundry Injuries

The 1973-80 Bureau of Labor Statistics (BLS) data show that the overall illness and injury rate (lost workday and nonworkday lost cases) in the ferrous foundries was two times that of manufacturing industries as a whole and about three times that of the private sector (Tables III-10 and III-11) [52,177,178,179,180]. These data include both occupational illnesses and injuries; however, occupational injuries account for more than 98% of the total cases [179]. Although, during the past 8 years, there has been some yearly variation in total cases and in incidence rates, there is no consistent trend that would indicate that conditions have become either better or worse.

Other recent studies have analyzed safety conditions in the foundry industry: the OSHA NEP study [181] conducted from 1975-77; the California Department of Industrial Relations, Division of Labor Statistics and Research (California) study of 1974-76 [48]; the AFS-Sponsored American National Standards Institute (ANSI) Safety Committee study [51]; and the 1981 Accidents Statistics (for foundries in Ohio), Industrial Commission of Ohio study [50].

The OSHA Hazard Analysis and Program Evaluation Study (HAPES) [181] was based on information collected by OSHA compliance officers during inspections of 86 foundries in 17 states for the NEP between March and June 1977. Of the 4,194 OSHA recordable case reports received, covering 20 million hours of exposure, 1,638 involved lost workdays, 5 were fatalities, and the remainder were nonfatal cases without lost workdays.

TABLE III-10. Comparative occupational injury and illness rates, 1973-76

Industry	SIC codes	1976 Annual average employment*	Incidence rates per 100 full-time workers: Total cases				Lost workday cases				Nonfatal cases without lost workdays				Lost workdays			
			73	74	75	76	73	74	75	76	73	74	75	76	73	74	75	76
Iron and steel foundries	332	225.1	32.0	30.4	28.5	26.9	10.3	11.6	11.4	10.7	21.7	18.8	17.1	16.1	156.7	163.1	191.4	173.0
Nonferrous foundries	336	82.3	29.0	27.6	22.1	22.8	9.9	11.1	8.1	9.6	19.0	16.5	14.0	13.2	134.9	151.8	141.6	148.8
All manufacturing	--	18,883.1	15.3	14.6	13.0	13.2	4.5	4.7	4.5	4.8	10.8	9.9	8.5	8.3	68.2	72.7	75.4	79.5
Total private sector	--	64,689.8	11.0	10.4	9.1	9.2	3.4	3.5	3.3	3.5	7.5	6.6	5.8	5.7	53.3	54.6	56.1	60.5

*In thousands

Adapted from references [52,177,178]

TABLE III-11. Comparative occupational injury and illness rates, 1977-80

Industry	SIC codes	1980 Annual average employment*	Incidence rates per 100 full-time workers: Total cases				Lost workday cases				Nonfatal cases without lost workdays				Lost workdays			
			80	79	78	77	80	79	78	77	80	79	78	77	80	79	78	77
Iron and steel foundries	332	209.6	23.6	26.0	24.6	24.4	13.0	11.4	11.6	10.9	12.2	13.0	13.0	13.5	183.0	186.0	169.2	164.0
Nonferrous foundries	336	90.7	21.3	23.5	23.0	22.6	12.0	10.9	11.2	10.4	10.4	11.5	11.8	12.2	162.5	177.9	158.7	166.2
All manufacturing	--	20,300.0	12.2	13.3	13.2	13.1	5.4	5.9	5.6	5.1	6.8	7.4	7.6	8.0	86.7	90.2	84.9	82.3
Total private sector	--	74,961.1	8.7	9.5	9.4	9.3	4.0	4.3	4.1	3.8	4.7	5.2	5.3	5.5	65.2	67.7	63.5	61.6

*In thousands

Adapted from references [179,180]

The California study [48] was based on 3,525 workers' compensation records of disabling injuries and illnesses from all foundries within the state during 1973-76. The disabling injuries and illnesses considered were those that resulted in worker absence for at least a full day or a workshift beyond the day when the accident occurred.

The AFS study [51] considered 2,844 OSHA-recordable cases which were submitted voluntarily by 26 sand-casting foundries at the request of the AFS/ANSI Safety Committee. The reports covered only the injuries and illnesses that occurred in 1972. The California and AFS studies each presented the total number of injuries in each job category considered, while the HAPES study presented the data as either lost workday cases or nonfatal cases without lost workdays. All lost workday cases were reviewed, but only a portion of the nonfatal cases without lost workdays were reviewed because of insufficient time. The incidence rates of and total injuries listed in all three studies are presented in Table III-12. Six categories of injury hazards are discussed: (1) strains or overexertion; (2) struck by or contact with objects; (3) contact with hot materials; (4) caught in or between machine parts or struck by ejected objects; (5) falls; and, (6) foreign substances in the eyes.

a. Overexertion

Injuries resulting from strains or overexertion were reported to be the most frequent type involving lost workdays in both the California study (30% of all injuries reported) [48] and the HAPES study (1981) [181]. Both studies showed that most of these injuries occurred in the molding and coremaking departments during manual materials handling such as the lifting and lowering of molds, jackets, and cores. Typical examples of overexertion included: a worker who lost 34 workdays when he strained his back pulling on a stuck box; another worker who sprained his back while lifting pieces of metal labelled "50 kg (110 lbs)" which he mistakenly read as "50 lbs (22.6 kg)"; and a worker who sprained his forearm while pouring molten aluminum from a ladle [176].

b. Struck by or in Contact with Objects

Injuries resulting from being struck by or coming in contact with objects were found to be the second most frequent type involving lost workdays in the California study (15.8%) [48], the second most frequent in the AFS study (17.6%) [51], and the most frequent type in the HAPES study [181]. These injuries occurred most frequently in the cleaning and finishing departments, usually during the handling of castings and hand tools, and in the melting, pouring, molding, and coremaking departments, during the handling of molds, flasks, cores, and hand tools. Workers in the melting and pouring areas commonly experienced injuries when handling scrap metals, castings, and hand tools [48].

TABLE III-12. Injury incidence from the California, HAPES, and AFS studies

Cause of injury by category	California study		HAPES study		AFS study	
	No. of disabling injuries	Percentage	No. of total cases	Percentage	No. of OSHA recordable cases	Percentage
Strains or overexertion	**1,071**	**30.4**	**517**	**19.2**	**247**	**15.8**
Lifting castings, cores, metal scrap	623		117		223	
Pushing or pulling molds, cores, wheelbarrows	81		64		17	
Handling molds, castings, ladles	131		106		N/A	
Using tools or shovels, sledgehammers, molding machines	122		39		N/A	
Other	114		151		N/A	
Chronic	N/A		40		7	
Struck by or in contact with	**558**	**15.8**	**558**	**20.7**	**276**	**17.6**
Dropped while handling castings, molds, scrap metal, containers	148		205		45	
Otherwise handling	209		156		N/A	
Handtools	N/A		75		2	
Rubbed or abraded	N/A		75		229	
Other	201		47		N/A	
Contact with hot materials	**356**	**10.1**	**241**	**8.9**	**23**	**1.5**
Hot or molten metal	245		111		N/A	
Hot objects: molds, castings	37		68		N/A	
Other	74		62		23	

N/A - Not available or applicable due to variations in initial compilation categories of separate studies.

Adapted from references [48,51,181]

TABLE III-12. Injury incidence from the California, HAPES, and AFS studies--Continued

Cause of injury by category	California study		HAPES study		AFS study	
	No. of disabling injuries	Percentage	No. of total cases	Percentage	No. of OSHA recordable cases	Percentage
Caught in or between machine parts or struck by ejected objects	**225**	**6.4**	**251**	**9.3**	**62**	**4.0**
Point of operation	N/A		109		11	
Moving machine parts	N/A		79		51	
Struck by object ejected	N/A		63		N/A	
Falls	**267**	**7.6**	**338**	**12.5**	**85**	**5.4**
Hazardous floor conditions	90		151		54	
From elevation	35		116		31	
Other	142		71		N/A	
Foreign substances in eyes	**383**	**10.9**	**354**	**13.1**	**707**	**45.3**
Metal scrap, chip, or dust	205		187		N/A	
Molten metal, slag	30		9		N/A	
Sand	6		33		N/A	
Other	142		125		707	
Total, categories presented	**2,860**	**81.1**	**2,259**	**83.9**	**1,400**	**90**
Total, including other miscellaneous categories	**3,525**	**100**	**2,694**	**100**	**1,561**	**100**

N/A - Not available or applicable due to variations in initial compilation categories of separate studies.

Adapted from references [48,51,181]

c. Contact with Hot Materials

Burns accounted for approximately 10% of the lost workdays in both the California (10.1%) [48] and HAPES (9.6%) [181] studies. Melting and pouring operations accounted for the majority of the burn injuries. Burns resulted from worker contact with molten metal; the majority were foot burns. The HAPES study [181] observed that in nearly all of the cases in which workers' feet were burned, the injuries might have been reduced in severity or prevented if proper protective footwear, e.g., nonflammable metatarsal guards, had been worn. Spats and gaiter-type boots worn inside the trousers are necessary because serious burns in foundries do occur when molten metal is spilled on the legs or inside the shoes [176].

d. Caught in or Between Machine Parts or Struck by Ejected Objects

Foundry machinery, such as automated or semiautomated molding and coremaking, presents a serious hazard from exposure to both flying or ejected materials and moving parts. The grinding operations in the cleaning and finishing departments account for numerous injuries from flying particles.

The HAPES study [181] listed contact with machine gears, pulleys, belts, and operating machine points as the causes of more than 8% of foundry lost workday injuries. The California study [48] reported that 6.4% of the lost workday cases involved workers being caught in or between moving machine parts.

e. Falls

In the HAPES study, injuries resulting from falls on or from walkways or work surfaces were the second most frequent cause of lost workday cases (13.8%) and ranked second in actual days lost (18.0%). Such falls also accounted for two of the five fatalities reported in the HAPES study [181]. The California study reported 7.6% of the lost workday cases involved falls [48].

Injuries due to falls from elevated work surfaces, ladders, stairs, or platforms are commonly more severe than those due to falls occurring on the same level. The majority of injuries involving slipping on substances or tripping over objects resulted from poor housekeeping practices where floors were wet, slippery, or littered.

f. Foreign Substances in the Eyes

Eye injuries were the most frequent nonfatal injury involving no lost workdays and the third most frequent cause of lost workdays reported in the HAPES study [181]. The California study recorded eye injuries as almost 10% of the lost workday cases [48]. In the AFS study, eye injuries occurred in 45.3% of all the reported injuries [51].

By far, the most frequent form of eye injury is caused by a foreign substance in the eye, either from dust in the air or particles propelled in foundry operations. These flying objects include metal chips; dust and abrasive material from cleaning, finishing, and grinding operations; sand in coremaking and molding operations; and metal particles, molten metal, and molten metal/steam explosions in melting and pouring operations. For the most part, the hazard of flying particles can be effectively reduced by a combination of machine safeguarding, personal protective equipment, and safe work practices.

The founding process generates a considerable amount of particulate matter in almost all operations. Engineering controls can significantly reduce worker exposure to dust hazards but cannot control eye injuries from propelled particles and eliminate dust hazards completely. In cleaning and finishing operations, even the use of air-supplied helmets has not completely prevented foreign substances from entering the eyes.