

# Chapter 4

## Health Disparities in Occupational Exposures

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### Key Points

- Respiratory occupational health disparities have occurred from the overrepresentation of minorities in hazardous industries and job titles with respiratory toxins.
- Known respiratory occupational health disparities have been identified from medical studies and high profile events as there is no ongoing nationwide surveillance system that is tracking work-related health disparities.
- Minorities continue to be overrepresented in particular industries and occupations despite the cessation of overt discriminatory hiring practices.
- The addition of occupation and industry to health surveys and race/ethnicity to occupational injury and illness surveillance systems is needed to provide ongoing evaluation.

*“When the white man had a job, his job wasn’t molding and shaking out. He had a job like setting cores. You couldn’t hardly find a one that shake out [foundry department with the highest silica dust levels].”* (Quote from a black retired Michigan foundry worker describing work conditions from the mid-1950s to the mid-1970s) (see Fig. 4.1).

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**Fig. 4.1** African-American foundry worker preparing to pour molten metal at a foundry in Minnesota. Photo by David Parker, MD

## Introduction

Federal and state governmental programs to address health disparities generally have not included addressing occupational health disparities as part of their mission [1]. However, for the first time in 2013, the Centers for Disease Control and Prevention (CDC) report on health disparities contained two chapters related to occupational health which focused on disparities in work-related nonfatal injuries and illnesses and on work-related fatal injuries [2]. The long-standing omission in addressing occupational health disparities has occurred despite the many examples in the medical literature of a disproportionate occurrence of work-related injuries and illnesses in minority and immigrant populations [3–7], the estimated economic costs of occupational injuries and illnesses in low-wage workers of \$15 billion for medical care and another \$24 billion for lost productivity [8], and a history of tragedies among minority and immigrant workers. An example of a well-known occupational tragedy in labor history was the 1911 Triangle shirtwaist factory fire in New York City when 146 predominantly female immigrant textile workers died [3]. This chapter will focus on health disparities in the USA as they relate to occupational respiratory exposures and illnesses.

Most respiratory-related occupational health disparities are related to the over-representation of minorities in hazardous industries and job titles/assignments that involve increased exposure to respiratory toxins [9–14]. Additionally, there has

been incomplete penetration of occupational health and safety interventions to certain worker populations due to barriers created by social, cultural, and economic issues including language, literacy, and marginal economic status [15–17]. Examples of the latter would be migrant farm workers or construction day laborers, who are predominately Hispanic. The aggregation of lower socioeconomic and immigrant populations in certain industries and occupations is an ongoing issue that suggests that historically well-documented occupational respiratory health disparities have the potential to continue in the future. The uneven distribution of race and ethnicity by occupations in Michigan is described later in the chapter as an illustration of job placement throughout the USA.

Although work-related health disparities have been recognized through high profile events and research studies, there is no ongoing nationwide surveillance system to track work-related health disparities [18]. Race and ethnicity data from the Bureau of Labor Statistics annual Survey of Occupational Injuries and Illnesses (SOII) are very limited and not summarized in official publications because employer reporting of these data elements is voluntary and is missing in 37 % of submitted records [19]. Additionally, most workers compensation data systems are not useful for tracking disparities by race and ethnicity because the national standard for recording worker compensation claims that is used by 80 % of state systems does not have data elements that cover race or ethnicity [20].

A limited number of states (23) have their own occupational health surveillance system and even fewer (California, Massachusetts, Michigan, New Jersey, and New York) conduct any surveillance for occupational respiratory disease, which is for the most part limited to work-related asthma. At this time, Michigan is the only state that conducts surveillance for all occupational lung diseases. Although limited in their scope, these state programs are capable of collecting data about race and ethnicity through access to demographic information in hospital discharge records, health care provider reports, and death certificates.

This chapter will review the overrepresentation of racial and ethnic minority workers in the most hazardous occupations and industries, providing specific examples of increased respiratory morbidity and mortality in minority workers due to disparities in exposure to chromates, coke oven emissions, cotton dust, radiation, and silica using information from epidemiologic studies and public health surveillance data [9–14].

## Chromates

Beginning in the 1930s, cases of lung cancer among chromate production workers were reported in the German medical literature. In the 1950s, the United States Public Health Service (USPHS) studied the workers at all seven US production facilities (four in New Jersey, one in New York, one in Ohio, and one in Maryland) where chromates and bichromates were made from chromite ore [9]. USPHS found that black males constituted 37 % of the total workforce, and most black workers

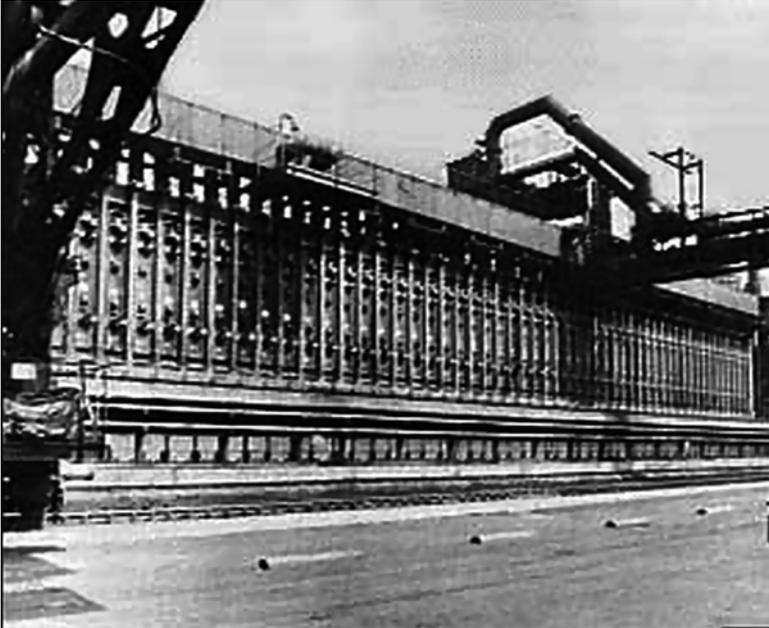
came from the South. For example, less than 4 % of the black workers in the four New Jersey facilities were born in that same state. The vast majority (86 %) of black workers in the chromate industry were employed at the two largest facilities, where they represented 46 % and 37 % of the workforce, respectively. The proportion of white workers was much lower in the production areas (mill room 67 %, kiln room 53 %, primary leach and residue drying 29 %, liquor room 71 %, and special processes 68 % white) compared to areas of lower exposure, such as maintenance (93 % white) and office, laboratory, and outside yard workers (79 % white).

The USPHS study found that respiratory morbidity and mortality were high in all chromate workers, but notably, black workers had greater risk than white workers in these facilities [9]. For example, the prevalence of nasal perforation, which has been associated with chromate work since the early 1800s, was higher in black workers (76.6 %) compared to white workers (49.3 %). Moreover, chromate workers overall demonstrated a 29-fold increased risk of death from respiratory cancer, but separated by race, the risk of lung cancer was increased 80-fold in black workers compared to only 15-fold in white workers. This is due to the production of hexavalent chromium compounds, which are known human carcinogens, during conversion of chromite ore to chromates. The increased risk of respiratory cancer death in blacks compared to whites was particularly notable because the prevalence of smoking among white and black workers was similar (81 % vs. 84 %), the prevalence of heavy smoking was higher among whites (32 % vs. 12 %), and a higher proportion of white workers had a longer duration of exposure (proportion of white workers with  $\geq 20$  years employment was 21.8 % compared to 10.2 % among black workers). However, hexavalent chromium exposures were highest in those areas with a higher percentage of black workers (kiln, leach, and liquid areas). There is no discussion in the USPHS report describing how workers were assigned jobs in these facilities. In the early 1990s, a follow-up study was conducted examining the vital status of the New Jersey portion of the cohort. For workers with more than 20 years since first exposure and work duration of at least 20 years, the proportionate cancer mortality ratio for lung cancer was 3.08 (95 % CI 1.13–6.71) in black workers compared with 1.94 (95 % CI 1.15–3.06) in white workers [21]. Follow-up of these former workers is still ongoing although the facilities in New Jersey have closed.

Chromite ore continues to be imported into the USA, and the USA continues to be a major producer of chromium products with the largest facilities in Indiana, Louisiana, Massachusetts, Nebraska, Ohio, Pennsylvania, South Carolina, Texas, and Wisconsin.

## Coke Oven Emissions

Metallurgical coke is used in the process of making iron. Iron ore, limestone, and coke are added to the blast furnace to produce iron, which can be further processed into steel. By the 1930s, most coke was made in “by-product” ovens from coal (Fig. 4.2). Coke is the residue of coal after the volatile components of coal are removed during



**Fig. 4.2** Pusher side of a by-product coke oven battery Courtesy of “The Making, Shaping and Treating of Steel.” Courtesy association of iron and steel Engineers

6–20 h of heating at temperatures from 700 to 1200°C. The tars, oils, and chemicals removed from the coal during the heating process are recovered and sold. Coal tar pitch volatiles released during the heating process are known human carcinogens [22] and contain polycyclic aromatic hydrocarbons such as anthracene and benzo(a) pyrene, aromatic compounds such as benzene and  $\beta$ -naphthylamine, and metals such as arsenic and chromium. Coke oven emissions also include gases such as nitric oxides and sulfur dioxide, and emissions are highest in the work areas on the top of the coke ovens.

In 1970, an important study was published that examined mortality by specific work area in 58,828 steel workers employed at seven facilities, including two with coke manufacturing, in Allegheny County, Pennsylvania [23]. Race was identified from personnel files as white and non-white. All-cause mortality among white coke workers was not increased compared to the general population, but there was a 22 % increase in the risk of death among non-white coke workers. Similarly, there was no increase in risk of overall cancer death among white coke workers, but among non-white coke workers, the risk of cancer death was double that which was observed in the general population.

Coke plant workers worked in three distinct areas: (1) handling coal; (2) loading and unloading the coke ovens; and (3) processing the by-products removed from the coal. Of the total workforce, 89 % of non-white workers versus 32 % of white

workers participated in loading and unloading the coke ovens. Non-white workers made up 61 % of the coke oven workforce but only 8 % of the non-oven workforce. Across the entire coke plant workforce, there was a 75 % increase in the risk of respiratory cancer compared to the general population, but this was due to increased risk among non-white workers. White coke plant workers had less respiratory cancer than expected, while non-white coke plant workers had an increased risk of respiratory cancer death [10]. These findings reflect the work area location of non-white coke plant workers in the coke oven area. Further characterizing the coke oven workers as “topside” versus “never worked topside” found that non-white workers made up 73.5 % of the topside workforce and had 10.8-fold increased risk of respiratory cancer. Although there was a fivefold increased risk of respiratory cancer among the fewer number of white topside workers, with one death compared to 0.2 expected, no statistical tests were performed in this study where there were less than five deaths.

In a subsequent publication, the study was expanded to include coke plant workers from ten other facilities in the USA and Canada. Non-whites made up 70.4 % of topside coke oven workers in this larger cohort. The highest risk for cancer of the lung, bronchus, and trachea continued to be among non-white topside workers with a 7.68-fold significantly increased risk compared to non-white, non-coke plant workers [24]. Follow-up of the vital status of this cohort through 1982 found risk of respiratory cancer was 1.59-fold higher (95 % CI 1.19–2.12) for white workers and 2.12-fold higher (95 % CI 1.70–2.84) for non-white workers among all coke plant workers compared to non-coke plant workers [25]. Topside workers continued to have the highest risk of death from respiratory cancer, demonstrating 4.25-fold higher risk (95 % CI 2.91–7.14) after working 10–14 years topside, 4.45-fold higher risk (95 % CI 2.79–7.56) after working 14–19 years topside, and 4.34-fold higher risk (95 % CI 2.89–6.97) after working 20 or more years topside compared to non-coke plant workers [25].

In 1966, coke oven exposure assessments were performed that measured coal tar pitch volatiles, thereby allowing for dose–response studies [26, 27]. Reinforcing the notion that topside jobs are associated with the greatest exposure, the average concentration of coal tar pitch volatiles for topside jobs was 3.15 mg/m<sup>3</sup>, which was 3.6-fold higher than the average level for side oven jobs and dramatically higher than the Occupational Safety and Health Administration’s current permissible exposure limit of 0.2 mg/m<sup>3</sup>. In further support of this, measurements of polycyclic aromatic hydrocarbons in the air and in worker urine samples from multiple coke ovens in different countries in the 1980s and 1990s demonstrated consistently higher exposure levels to topside workers [22] (IARC, 2005). Estimates of cumulative exposure to coal tar pitch volatiles among non-white workers, consistent with their more frequent assignment to topside jobs, were higher than white workers [26] (see Table 4.1). The risk for lung cancer mortality between non-white and white workers has been reported to be similar when duration of employment and job location topside was taken into account [28]. The disparity in lung cancer mortality observed is thus attributed to the overrepresentation of non-whites in topside jobs. A 1973 affirmative action ruling at a steel plant in Maryland documented ongoing

**Table 4.1** Cumulative exposure to coal tar pitch volatiles among non-white and white coke oven workers based on sampling performed in 1966

Cumulative exposure (mg/m <sup>3</sup> -months)	Non-white workers		White workers	
	#	%	#	%
<200	865	(32.3)	970	(49.0)
200–499	1042	(38.9)	807	(40.8)
500–699	422	(15.7)	140	(7.1)
700+	353	(13.2)	62	(3.1)
Total	2682		1979	

Data obtained from [26]

discriminatory job placement in this industry with blacks making up 75–100 % of the workforce of some departments including coke ovens and 0 % of other departments such as pipefitting or machining [29].

From 1950 to 1968, the lung cancer mortality rate for non-white males in Allegheny County was 28 % higher than that in white males. This is in contrast to the fact that non-white males had a slightly lower mortality rate for lung cancer than white males during the same time period at the national level [29]. The increased risk from a given work area in a given industry is presumably one factor explaining this difference between Allegheny County and the national rates. It is example of how the increased risk of cancer from an occupational exposure may affect local geographic rates when that industry workforce makes up a large enough proportion of the population in a geographic area [30].

## Cotton Dust

Exposure to cotton in textile mills is associated with acute respiratory symptoms and the development of chronic bronchitis and COPD, particularly with long-term work in high dust areas such as opening bales, “picking” (a process that is no longer necessary in modern textile mills that used to prepare the cotton lint for carding), and carding (separation and alignment of fibers). In 1973, a survey that measured respiratory symptoms and pulmonary function was conducted in 6432 workers at 14 US textile plants [12]. Black men had a higher prevalence of acute symptoms consistent with byssinosis (5.5 % vs. 3.6 % in white men). Among the 165 cases of byssinosis identified, 64 % of the cases worked in the high dust work areas, where only 12 % of the overall textile mill workforce was located. Blacks made up 64.5 % of the workforce in the high dust areas, which was much greater than their overall workforce prevalence of 34.4 %. Despite the fact that blacks had a shorter duration of work, chronic bronchitis prevalence was similar between blacks and whites. Moreover, blacks had lower pulmonary function results than whites, with workers in the dustier area having the lowest results. Data on cigarette smoking was not provided by race, but it was noted, that there were less heavy smokers in the dustier

work areas, where the workforce was 64.5 % black. Much of this industry has now moved overseas with reports of respiratory disease among cotton textile workers now being reported from China, India, Pakistan, and Turkey.

## Radiation

Significant uranium mining began in the USA during World War II. The mines were predominately located in the southwest on the Colorado Plateau where the four states of Arizona, Colorado, New Mexico, and Utah abut. In this region, major deposits of uranium were identified on the Navajo Reservation. Four centers of mining and milling were established there, and there are an estimated 1000 abandoned shafts on the reservation [31]. Mining was the primary industry offering job opportunities for most Navajo individuals, and the peak years of mining occurred from 1948 to 1969. Although the risk of lung cancer among uranium miners was first recognized in Europe as early as 1879 and was made compensable in Europe in the 1930s, there were no regulatory standards for limiting radiation exposure during the peak years of mining on the Navajo reservation [32]. Navajo miners were paid minimum wage, and they worked underground blasting, building wooden supports, digging blasted rock (muckers), transporting ore, and milling ore. Foremen were white and thus did not spend as much time underground.

Mining dust contains radon progeny which produce alpha particles responsible for the increased lung cancer risk. The nomenclature for expressing radon progeny exposure is based on working levels. A working level (WL) is the amount of radon progeny in a liter of air that releases a specified amount of alpha radiation. Exposure to a WL for 170 h (the average number of work hours in a month) equals a working level month (WLM). Background levels of radon progeny from radon contamination in homes cause the average person in the general population to have a lifetime exposure of 10–20 WLM. In contrast, WLM exposures in miners with lung cancer ranged from 465 to 16,467 [33]. Current workplace regulations allow up to 4 WLM/year or 160 WLM over a 40 year working lifetime.

From 1969 to 1982, 72 % of the lung cancer cases among Navajo men in the New Mexico Cancer Registry had been employed by a uranium mine versus 0 % of controls in a matched case–control study [34]. This corresponds to an infinite odds ratio with a 95 % CI lower limit of 14.4. Thirty-eight percent of the uranium miners with lung cancer in this study were nonsmokers, while the other 62 % of Navajos with lung cancer averaged only one to three cigarettes per day. Consistent with the low smoking burden in the case control study of Navajo miners with lung cancer, a survey of Navajo miners showed low smoking prevalence (41 % ever smokers) and low cigarette consumption among active smokers [31]. An extension of the New Mexico cancer registry case–control study until 1993 continued to find a high percentage (67 %) of lung cancer in Navajo men to be attributable to past work in the uranium mines. This later study was able to calculate an odds ratio since some controls in this later study had worked in the uranium mines; the investigators found an odds

ratio of 28.6 (95 % CI 13.2–61.7) compared to matched Navajo men with non-respiratory cancer [32]. Surveys of white miners have found appreciably higher cigarette smoking rates, with only 18 % of white miners having never smoked versus 59 % in the Navajo miners [35]. The joint effect of cigarette smoking and radon progeny exposure is synergistic [36]. Despite the lower smoking rates in Navajo miners, lung cancer risk between white and Navajo miners were similar and are indicative of higher radon progeny exposure in Navajo miners [14, 35].

A cross-sectional study of miners who participated in the New Mexico Miners Outreach Program found that Navajos who had worked in the uranium mines demonstrated an increased risk of chronic obstructive pulmonary disease (COPD), low lung function (FEV1), and radiologic evidence of pneumoconiosis in relation to years worked [37]. However, this was not true of white miners who had worked in uranium mines. In this study, 73 % of Navajo miners had never smoked cigarettes, and those who had smoked cigarettes averaged 6.4 cigarettes per day. In contrast, only 22 % of white uranium miners had never smoked cigarettes, and among those who had the average were 20.3 cigarettes per day. Thus, the increased risk of respiratory disease in relation to work only occurred among Navajo miners, despite decreased cigarette smoking among Navajo compared to white uranium miners.

## Rubber Workers

Mortality of workers from a large rubber tire manufacturing plant in Akron, Ohio, was examined between 1964 and 1972. Study investigators identified a greater than twofold difference in the proportion of whites and blacks in different occupations with blacks being more likely to have worked in high exposure occupations such as compounding and mixing (27 % of blacks vs. 3 % of whites had worked in this occupation). There was an increased age-standardized risk ratio of lung cancer of 1.4 (99.9 % CI 1.1–2.0) in the compounding and mixing area compared to local county or national rates [11], but this risk was only significantly increased for black workers in the area. This latter difference was “clearly influenced by the racial composition of the workforce in that work area” [11]. Potential exposures to carcinogens in this work area would have included asbestos and silica, which are also known to cause chronic lung disease, as well as multiple solvents. Disparities in respiratory health related to this industry are likely ongoing today, as there are approximately 40 rubber tire manufacturing facilities in the USA and appreciably more worldwide.

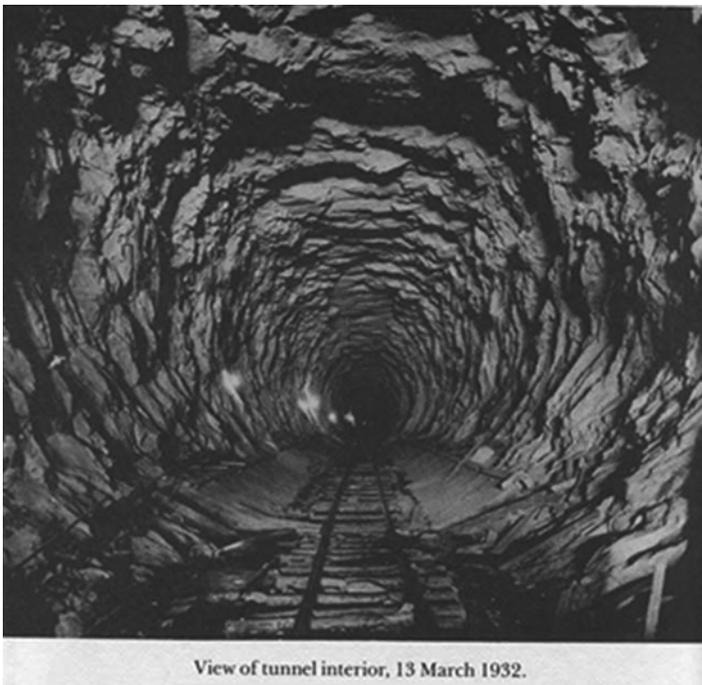
## Silica

Silicon in combination with oxygen, silica (silicon dioxide) is one of the most common constituents of the earth’s crust. Mining for any mineral or tunneling will cause generation of respirable dust that will contain varying percentages of silica

depending on the underlying geology of the particular rock formation. Silica is widely used in foundries in the production of metal products and the production of ceramic products such as sinks and toilet bowls, and there is also risk of silica exposure during abrasive blasting, and more recently in the process of fracking as part of oil and gas drilling.

### **Example #1: Hawk's Nest, West Virginia**

In March 1930, dam construction on the New River began at Hawk's Nest, West Virginia, and this included boring a three mile water tunnel through Gauley Mountain to power a new power plant at Gauley Bridge (see Fig. 4.3). Tunnel construction required drilling through sandstone that was over 99 % silica. Despite the dustiness and high silica content of the rock being tunneled, water suppression to keep dust levels down was not used because of the rapid speed of drilling that was required to meet the 17.5 month contract deadline for completion of the tunnel. It is estimated that the total number of workers at the job site was about 5000, of whom 2500 worked underground in the tunnel. Turnover was high with an average length



**Fig. 4.3** Interior of Hawk Mountain tunnel in 1932. Courtesy West Virginia archives and history

of employment of 15 weeks. There were 600 tunnel workers at any one time. Silica exposure was highest for underground workers, which included 75 % of African-American workers versus only 44 % of white workers. White workers who did work underground were more likely to be foreman or heavy equipment operators, whereas African Americans were drillers, muckers (who hauled away rock and debris), and driller assistants. There were two 10-h work shifts each day with ongoing work 6 days a week. During each shift, drilling was performed to set the dynamite (600–800 lb per charge), the charge was set, and then the rock and debris were removed after the explosion. Eighteen deaths (5 whites and 13 blacks) occurred from acute traumatic injuries, and a much larger number of workers died from respiratory disease. Sixty-five percent of the overall workforce was composed of African-American men who came from southern states to work at the site. Their migrant status and racism are factors that may have delayed the initial recognition and response to addressing the epidemic of work-related respiratory disease that occurred. Other factors that have been identified that contributed to the delay in response include: (1) high worker turnover with workers returning to their home states when they became sick, (2) slow reporting on the part of local newspapers with respect to worker deaths, (3) fear and intimidation on the worksite that limited reporting, and (4) attribution of deaths to poor nutrition and inability of African Americans from southern states to tolerate cold weather.

Within a few years after the tunnel was completed, the related respiratory epidemic was recognized as silicosis that occurred in response to silica dust exposure during the tunneling operation. Unfortunately, no accurate count of the number of workers who died or developed silicosis is available. During congressional hearings that were held 5 years after completion of the tunnel in 1936, a senator stated there were 2000 deaths. There was also testimony from one funeral home director indicating he was paid to dig 169 unmarked graves. In support of this, highway construction records from a 1972 West Virginia Department of Transportation contract documented removal of 63 unmarked graves during construction of a nearby road. For the entire tunneling project, the official death toll from the company was 109, including the traumatic deaths. However, by 1933, worker compensation suits were filed by 336 individuals.

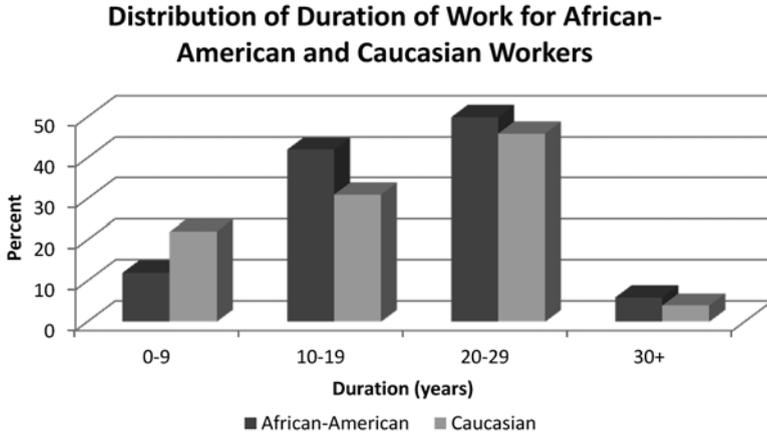
The best estimate of deaths from silicosis was derived in 1986 [13]. This estimate used county death records and determined the excess respiratory deaths from 1931 to 1937 that occurred in Fayette County, where Hawk's Nest was located, in comparison to adjoining West Virginia counties and extrapolated the number of migrant worker deaths that were not reported in the county. Cherniak's calculation was based on 1213 workers, 291 whites and 922 blacks, who worked at least 2 months underground and were considered to be at the highest risk of developing silicosis. The total number of workers estimated to have died from silicosis was 764, including 581 (63 %) of the 922 African-American workers and 183 (62.8 %) of the 291 white workers who worked 2 or more months underground. Typically, silicosis related to work exposure in foundries or mines develops after 20 or more years of exposure, but for the limited number of identified workers with available

medical records from Hawk's Nest, it appears the high silica exposure in the tunnel resulted in a short time to development of respiratory disease, with workers dying within months and a few years of exposure due to acute silicosis (pathology similar to alveolar proteinosis) and accelerated silicosis (same pathology as chronic silicosis but rapid onset). No long-term follow-up was conducted on this workforce, and there is no data on the incidence of chronic silicosis or the development of tuberculosis. Presumably, this was already a population with an increased prevalence of tuberculosis, and active tuberculosis is more common and virulent in individuals with a history of silica exposure [38].

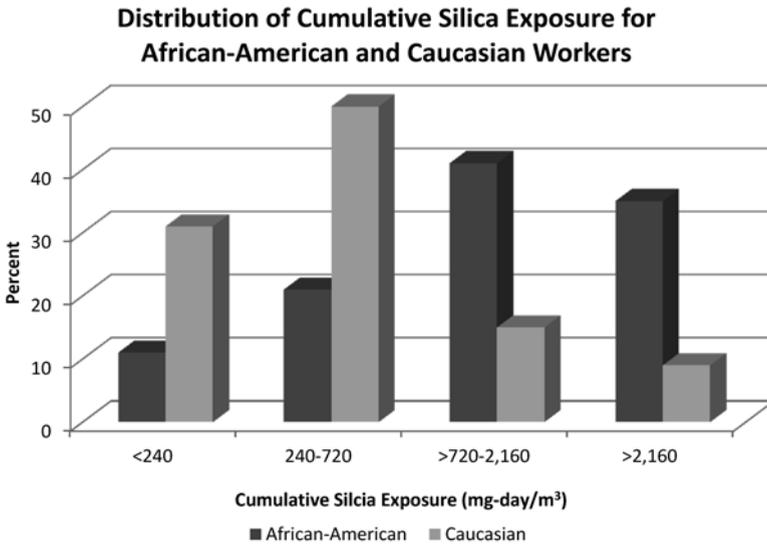
## Example #2: Foundries

Ford Motor Company was the largest employer of black auto workers prior to World War II, as they started to hire a large number of African-Americans in 1918. Other auto manufacturers did not begin to hire blacks in appreciable numbers until the labor shortages of World War II. For example, the largest grey iron foundry in the USA located in Muskegon, Michigan, sent personnel managers to the south during World War II and paid black workers to move north to work in their foundry. Ford paid black and white workers the same wage but placed blacks in the undesirable, hot, and more dangerous foundry jobs where the quit rates of white workers had been high [39]. Given the lack of alternatives for black workers and the relatively good pay, quit rates from foundry jobs by blacks were low, and foundries became known as the "black departments." The consequence of the concentration of blacks in foundries is reflected in the current statistics on the incidence of silicosis. In Michigan, 40 % of individuals with silicosis are African-American, although African-Americans only make up 14.3 % of the Michigan population [40]. The annual average incidence rate of silicosis among African-American males (8.8 cases per 100,000) is 5.5 times higher than that of white males (1.6 cases per 100,000). The rates within specific Michigan counties range between 2 and 366 times higher for African-American males than the rates for white males [40].

Significant racial disparity in silica exposure in foundries is documented from a study of a foundry in another Midwestern state [41]. In a cross-sectional study of current and retired workers, the prevalence of silicosis was 8.3 % among blacks and 4.0 % among whites. This higher prevalence of silicosis among African-Americans was found despite the fact that white and black workers had a similar distribution of duration of work, which is a common surrogate measure often used to estimate workplace exposures (see Fig. 4.4). The cause of the higher silicosis prevalence in black workers was their higher levels of silica exposure despite working the same number of years as white workers (see Fig. 4.5). This later determination was possible because of a detailed job exposure matrix that was developed during a review of industrial hygiene sampling results over many years, which documented that African-Americans had jobs with higher silica exposure [42]. In analyses control-



**Fig. 4.4** Duration of work for African-American and Caucasian workers at a gray iron foundry. Reprinted with permission Am J Epid 1996; 144: 890–900



**Fig. 4.5** Mean silica exposure for African-American and white workers at a gray iron foundry. Reprinted with permission Am J Epid 1996; 144: 890–900

ling for silica exposure, blacks and whites had a similar prevalence of silicosis [41]. The discriminatory practices leading to placement of black workers in foundries has been well documented [39]. In a collection of interviews conducted by a historian at Michigan State University, retired black foundry workers with confirmed silicosis who had moved from the South for work describe how being black affected job

placement within the foundries [20]. Please see the quote from one of the individuals who were interviewed at the beginning of this chapter.

### **Example # 3: South African Gold Mines**

Occupational health disparity is not unique to the USA. Respiratory exposure disparity has been described among black South African gold miners. The workforce in the South African mines in the 1990s was 600,000, with 90 % of the workers being black. The black workers were the laborers, while white workers were generally in supervisory jobs. The first study of black gold miners was not published until 1991 because black miners were considered transient rural individuals who worked to obtain sufficient money to return to their farms [43]. This first study of black miners was a cross-sectional study that only included current workers, but in spite of that limitation, study investigators found that 857 (71.6 %) of 1197 black miners had silicosis and 62 % had chronic bronchitis including 45 % of the miners who had never smoked cigarettes. In contrast, a study published 13 years earlier of white south African gold miners found that only 134 of 1973 (6.8 %) had silicosis [44]. While the prevalence of chronic bronchitis among the white miners was similar to the results in black miners (62.7 %), the percentage of nonsmokers among black miners was 29.2 % versus 11.3 % among white miners. Although changes since the end of apartheid have ended legislated differences, such as increased compensation rates for white miners who develop silicosis compared to black miners, “race remains an important determinant of occupation, salary, housing and disease burden” [45].

### **Work-Related Asthma**

American Thoracic Society (ATS) consensus statements have determined that 36.5 % of adult asthma is either caused or aggravated by work. The median estimate is that 15 % of adult asthma is caused by work exposures and that 21.5 % of adult asthma is aggravated by work exposures [46, 47].

The prevalence of work-related asthma among adults was calculated from the Behavioral Risk Factor Surveillance System (BRFSS) asthma call-back survey that was administered to a random sample of the general population in 38 states and the District of Columbia from 2006 to 2009 [48]. Work-related asthma was defined as having current asthma and responding yes to the following question: “Were you ever told by a doctor or other health professional that your asthma was related to any job you ever had?” The overall prevalence of work-related asthma was 9.0 % (95 % CI 8.4–9.6). Black and Hispanic participants reported a greater prevalence of work-related asthma compared to white participants (blacks 12.5 % (95 % CI 9.8–15.2), Hispanics 10.5 % (95 % CI 7.7–13.4), whites 8.2 % (95 % CI 7.6–8.8)) [48].

The BRFSS survey did not collect industry or occupation data as a core variable of the survey.

Incidence data from Michigan demonstrated similar differences in work-related asthma by race. The annual incidence rate of work-related asthma for African Americans was 4.8/100,000, which was nearly twofold greater than that of whites (2.5/100,000) [20]. This incidence data was derived from the state's occupational disease reporting system based on reports received from health care facilities and practitioners.

Cross-sectional surveys of the general population have shown marked differences in the prevalence of asthma by occupation [49, 50]. The National Health Interview Survey (NHIS) collects information on industry and occupation as well as self-report of health care provider diagnosed asthma. The overall prevalence of asthma from the combined surveys from 1997 to 2004 was 9.21 % with Blacks having the highest prevalence of 9.42 %, then Whites with a prevalence of 9.28 % and Hispanics with the lowest prevalence of 6.77 %. By occupation, service occupations had the highest prevalence of asthma at 10.58 %, and farming, forestry, and fishing occupations had the lowest prevalence at 6.83 % [50]. Within a given occupation, the prevalence of asthma by race differed from the overall prevalence of asthma by race. For example, among individuals who reported having a service occupation, Whites had the highest prevalence of asthma at 11.01 %, compared with 9.82 % among Blacks and 6.39 % among Hispanics in the same occupation category. Interpretation of this data is limited because the data collected was related to current occupation, which is not necessarily the same occupation that participants had when their asthma began as they may self-select a new occupation after development of their asthma [51]. Additionally, individuals with childhood asthma may self-select into certain occupations [52].

Industries with exposures that cause occupational lung diseases from mineral exposures have typically had a predominately male workforce, i.e., construction and manufacturing, and consequently most cases of pneumoconiosis have occurred in men. An exception to this generalization in gender differences in occupational lung disease is that more cases of work-related asthma are reported in women than men (60 % vs. 40 %) [53]. The more common occurrence of work-related asthma in women can be partially attributed to exposure to allergens in some industries that are predominately female [54] and differential exposure to men and women who are in the same industry, i.e., health care [55].

In addition to considering the work relatedness of asthma, the 2003 ATS statement on occupational airways disease described that work was a significant contributor to the development of COPD in 15 % of cases [46]. Cross-sectional studies of COPD in the general population, like those for asthma, have clearly shown marked differences in prevalence of obstructive lung disease by occupation [50, 56]. While the overall prevalence of COPD is greatest among whites [50, 56], the estimated proportion of cases associated with work is greatest among Mexican Americans (49.6 %), followed by Blacks (23.4 %) and whites 22.2 % [56]. The industries contributing to the attributable fraction of COPD caused by work also differ by race. Among whites, the most important industries were armed forces, rub-

ber, plastics and leather manufacturing utilities, and textile manufacturing. In contrast, among blacks, the related industries included construction, metal and automobile manufacturing, food product manufacturing, and agriculture. Among Mexican Americans, the important industries included agriculture, construction, and services. The authors concluded the higher percentage of COPD attributable to work among Mexican-Americans was due to the lower cigarette smoking burden in Mexican-Americans.

The strongest associations between COPD and specific work-related exposures are with chlorine, coal, cotton dust, silica, and welding fumes [57]. There are no data available that assess current exposure to these substances by age, race, or ethnicity. There are limited data on differential exposures between men and women [54, 55]. It is generally acknowledged that lower socioeconomic, labor intensive jobs are likely to have greater safety and health risks, including exposure to respiratory toxins, than higher socioeconomic white collar/office jobs.

## **Current Racial Distribution of the US Workforce**

This chapter contains multiple historical examples where discrimination in hiring practices led to disparities in the development of occupational respiratory disease. Given the typical long latency between onset of exposure and development of pneumoconiosis or lung cancer, some current racial differences in respiratory disease rates may represent a legacy from past discriminatory hiring practice (i.e., lung cancer). This certainly is the case with the occurrence of silicosis in Michigan. Even though discriminatory hiring practices are now illegal, there are other reasons why there continue to be marked differences in the distribution of the races across occupations. These include residential clustering by race with geographic limitations on the availability of jobs and differences in educational attainment. Table 4.2 shows the distribution of the ten most common occupations where individuals in each race/ethnicity group were employed in Michigan in 2011. The top ten occupations among blacks and Hispanics were service or manual labor; in contrast, six of the ten top occupations of whites and only one among Asians were service or manual labor. The CDC has shown using 2010 data that minorities are overrepresented in occupations having the highest occupational injury rates—24.4 % of Hispanics versus 11.6 % of Blacks versus 3.8 % of Whites are employed in high-risk occupations [2]. This determination of high-risk occupations was based on current work-related injury rates. No similar analysis of the current workforce for occupations with increased risk for respiratory exposures has been performed. Establishment of the Occupational Safety and Health Administration in 1970 and the implementation of workplace standards and reductions in exposures for respiratory toxins over time have ideally made the workplace safer for all workers. However, there continues to be documentation of disparities in work-related injuries [4–7]. On the other hand, there have been no dramatic disparities in mortality from work-related lung cancer documented

**Table 4.2** Ten most common occupations for Hispanics, African Americans, Asians and Whites, Michigan 2011<sup>a</sup>

<i>Hispanic (# employed: 161,489)</i>	<i>African Americans (#employed: 435,105)</i>
Agricultural workers (9.8 %)	Nursing/home health aides (4.6 %)
Assemblers and fabricators (4.1 %)	Janitors (3.1 %)
Grounds maintenance workers (3.1 %)	Assemblers and fabricators (3.1 %)
Retail salespersons (3 %)	Personal and home care aides (2.8 %)
Janitors (2.8 %)	Cashiers (2.8 %)
Cooks (2.5 %)	Laborers (2.5 %)
Food preparation workers (2.2 %)	Customers service reps (2.4 %)
Packers and packagers, hand (2.2 %)	Retail salespersons (2.3 %)
Waiters/waitresses (1.9 %)	Cooks (2.2 %)
Secretaries (1.8 %)	Bus drivers (2.1 %)
<i>Asian (# employed: 129,414)</i>	<i>White (# employed: white—3,558,662)</i>
Mechanical engineers (9 %)	Cashiers (2.4 %)
Software developers (7.5 %)	Retail salesperson (2.4 %)
Postsecondary teachers (4.3)	Driver/sales workers and truck drivers (2.8 %)
Computer/information systems managers (3.9 %)	Secretaries (2.3 %)
Physical therapists (3.9 %)	Managers, all other (2.1 %)
Managers (3.4 %)	Nurses (2.1 %)
Nurses (3.1 %)	Elementary/middle school teachers (2.1 %)
Cooks (2.9 %)	Supervisors of retail sales workers (1.9 %)
Accountants (2.9 %)	Waiters/waitresses (1.6 %)
Physicians (2.7 %)	Assemblers/fabricators (1.5 %)

<sup>a</sup>Rankings of most common occupations are from the 2011 Current Population Survey, U.S. Bureau of Census. Occupations are the occupational categories used by the Bureau of Census [<http://www.census.gov/people/io/methodology>]

Percents in the table are the percent of all employed members of that race/ethnicity group who work in that particular occupation (Table adapted from ref [20])

in the medical literature for the last 30 years. The overrepresentation of minorities in certain occupations such as service occupations (see Table 4.2) indicates the potential for ongoing occupational respiratory disparities secondary to differential exposure risks. For example, service workers have an increased prevalence of asthma [50] and are likely to have an increased potential for exposure to cleaning agents, which are associated with the development of work-related asthma [58].

Given the documented deficiencies in the current occupational surveillance system, changes are necessary if we are to accurately assess the occurrence of current and future occupational respiratory health disparities. The modifications needed include adding race to surveillance systems that already collect information on work-related injuries and illnesses and adding information on occupation and industry to surveys/medical records that collect medical and race data. Recommendations include: (1) requiring the reporting of race in the annual Bureau of Labor Statistics employer-based survey on injuries and illnesses; (2) adding race as a core variable

in worker compensation state data systems; (3) adding industry and occupation to the core module of the annual BRFSS survey administered in the 50 states; and (4) routinely collecting information about occupation/employer in medical records and making collection of such information a requirement for future meaningful use incentives as part of the transition to electronic medical health records [59]. Successful implementation of the above changes will not only allow for the generation of valuable data on occupational respiratory health disparities but will also allow for development of targeted interventions.

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