8 Basis for the Recommended Standard

8.1 Background

In the Occupational Safety and Health Act of 1970 (Public Law 91–96), Congress mandated that NIOSH develop and recommend criteria for identifying and controlling workplace hazards that may result in occupational illness or injury. In fulfilling this mission, NIOSH continues to investigate the potential health effects of exposure to naturally occurring and synthetic airborne fibers. This interest stems from the results of research studies confirming asbestos fibers as human carcinogens. Significant increases in the production of RCFs during the 1970s and concerns about potential health effects led to experimental and epidemiological studies as well as worker exposure monitoring. Chronic animal inhalation studies demonstrated the carcinogenic potential of RCFs, with a statistically significant increase in the incidence of lung cancer or mesothelioma in two laboratory species—rats and hamsters [Bunn et al. 1993; Mast et al. 1995a; McConnell et al. 1995]. Evidence of pleural plaques observed in persons with occupational exposures to airborne RCFs is clinically similar to that observed in asbestos-exposed persons after the initial years of their occupational exposures to asbestos [Hourihane et al. 1966; Becklake et al. 1970; Dement et al. 1986]. NIOSH considers the discovery of pleural plaques in U.S. studies of RCF manufacturing workers to be a significant finding because the plaques were correlated with RCF exposure [Lemasters et al. 1994; Lockey et al. 1996]. In addition, NIOSH considers the respiratory symptoms and conditions (including dyspnea, wheezing, coughing, and pleurisy) observed in RCF workers to be adverse health effects associated with exposure to airborne RCFs [Lemasters et al. 1998; Lockey et al. 1993; Trethowan et al. 1995; Burge et al. 1995; Cowie et al. 1999].

An association between inhaling RCFs and fibrotic or carcinogenic effects has been documented in animals, but no evidence of such effects has been found in workers in the RCF manufacturing industry. The lack of such an association could be influenced by the small population of workers in this industry, the long latency period between initial exposure and development of measurable effects, the limited number of persons with extended exposure to elevated concentrations of airborne fibers, and declining occupational exposure concentrations. However, the evidence from animal studies suggests that RCFs should be considered a potential occupational carcinogen. This classification is consistent with the conclusions of ACGIH, EPA, DECOS, and IARC. (See discussion in Chapter 7.)

Given these considerations, the NIOSH objective in developing an REL for RCFs is to reduce the possible risk of lung cancer and mesothelioma. In addition, maintaining exposures below the REL will also help to prevent other adverse effects, including irritation of the skin, eyes, and respiratory tract in exposed workers. To establish an REL for RCFs, NIOSH took into account not only the animal and human health data but also exposure

information describing the extent to which RCF exposures can be controlled at different workplaces. On the basis of this evaluation, NIOSH considers an REL of 0.5 f/cm^3 (as a TWA for up to 10 hr/day during a 40-hr workweek) to be achievable for most workplaces where RCFs or RCF products are manufactured, used, or handled. Maintaining exposures at the REL will minimize the risk for lung cancer and reduce the risk of irritation of the eyes and upper respiratory system. The residual risks of lung cancer at the REL are estimated to be 0.073 to 1.2 per 1,000 based on extrapolations of risk models from Moolgavkar et al. [1999] and Yu and Oberdörster [2000].

The risk for mesothelioma at the REL of 0.5 f/cm^3 is not known but cannot be discounted. Evidence from epidemiologic studies showed that higher exposures in the past resulted in pleural plaques in workers, indicating that RCFs do reach pleural tissue. Both implantation studies in rats and inhalation studies in hamsters have shown that RCF fibers can cause mesothelioma. Because of limitations in the hamster data, the risk of mesothelioma cannot be quantified. However, the fact that no mesothelioma has been found in workers and that pleural plaques appear to be less likely to occur in workers with lower exposures suggests a lower risk for mesothelioma at the REL.

Because residual risks of cancer (lung cancer and pleural mesothelioma) and irritation may exist at the REL, NIOSH further recommends that all reasonable efforts be made to work toward reducing exposures to less than 0.2 f/cm^3. At this concentration, the risks of lung cancer are estimated to be 0.03 to 0.47 per 1,000 based on extrapolations of risk models from Sciences International [1998], Moolgavkar et al. [1999], and Yu and Oberdörster [2000].

Maintaining airborne RCF concentrations at or below the REL requires the implementation of a comprehensive safety and health program that includes routine monitoring of worker exposures, installation and routine maintenance of engineering controls, and worker training in good work practices. To ensure that worker exposures are routinely maintained below the REL, NIOSH recommends that an AL of 0.25 f/cm^3 be part of the workplace exposure monitoring strategy to ensure that all exposure control efforts (e.g., engineering controls and work practices) are in place and working properly. The purpose of the AL is to indicate when worker exposures to RCFs may be approaching the REL. Exposure measurements at or above the AL indicate a high degree of certainty that concentrations of RCFs exceed the REL. The AL is a statistically derived concept permitting the employer to have confidence (e.g., 95%) that if exposure measurements are below the AL, only a small probability exists that the exposure concentrations are above the REL. When exposures exceed the AL, employers should take immediate action (e.g., determine the source of exposure, identify measures for controlling exposure) to ensure that exposures are maintained below the exposure limit. NIOSH has concluded that an AL allows for the periodic monitoring of worker exposures in the workplace so that resources do not need to be devoted to conducting daily exposure measurements. The AL concept has been an integral element of recommended occupational standards in NIOSH criteria documents and in comprehensive standards promulgated by OSHA and MSHA.

8.2 Rationale for the REL

The recommendation to limit occupational exposures to airborne RCFs to a TWA of 0.5 f/cm^3 is based on data from animal and human studies, risk assessments, and the

availability of methods to control RCF exposures at the REL in many workplaces. Establishing the REL for RCFs is consistent with the mission of NIOSH mandated in the Occupational Safety and Health Act of 1970. This Act states that NIOSH is obligated to "develop criteria dealing with toxic materials and harmful physical agents and substances which will describe exposure levels that are safe for various periods of employment, including but not limited to the exposure levels at which no employee will suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience." The carcinogenicity findings from the chronic nose-only inhalation assays of RCF1 in rats and hamsters [Mast et al. 1995a,b; McConnell et al. 1995] warrant concern about possible health effects in workers exposed to RCFs. Although no increase in lung cancer or mesothelioma mortality has been observed in worker populations exposed to RCFs, radiographic analyses indicate an association between pleural changes (including pleural plaques) and RCF exposure [Lemasters et al. 1994; Lockey et al. 1996; Cowie et al. 1999, 2001]. Both the U.S. [Lockey et al. 1993; Lemasters et al. 1998] and the European [Trethowan et al. 1995; Burge et al. 1995; Cowie et al. 1999, 2001] studies have found RCF-associated respiratory symptoms, pulmonary function reductions, and pleural abnormalities among RCF production workers.

Several independent evaluations have quantitatively estimated the risk of lung cancer for workers exposed to RCFs at various concentrations [DECOS 1995; Fayerweather et al. 1997; Moolgavkar et al. 1999]. NIOSH evaluated these studies to determine whether an appropriate qualitative or quantitative assessment of lung cancer risk could be achieved. In addition, exposure information was collected during the 5-year monitoring period covered under the consent agreement between RCFC and EPA [Maxim et al. 1994, 1997, 1998]. NIOSH used the exposure information to evaluate the feasibility of controlling workplace exposures at manufacturing and end-use facilities where RCFs and RCF products are handled.

8.2.1 Carcinogenesis in Animal Studies

Chronic inhalation studies with RCFs demonstrate significant increases in the incidence of mesothelioma in hamsters and lung cancer in rats. Tables 8–1 through 8–4 present a synopsis of the major findings of these studies [Mast et al. 1995a,b; McConnell et al. 1995]. Results from chronic animal inhalation studies with chrysotile and amosite are also presented (i.e., results for the positive control groups); these data provide a reference point for determining the relative toxicity of RCFs [Mast et al. 1995a; McConnell et al. 1999].

Chronic inhalation exposure to RCF1 at 30 mg/m^3 (187 WHO f/cm^3) induced a 13% (16/123) incidence of lung tumors in F344 rats [Mast et al. 1995a]. The incidence of lung cancer at lower doses did not show a statistically significant difference from the unexposed control group. Lung fiber burdens in the multi-dose chronic rat study revealed a dose-response relationship [Mast et al. 1995b]. In the rat, 16 mg/m^3 (120 WHO f/cm^3) appeared to be the NOAEL for lung cancer and 3 mg/m^3 (26 WHO f/cm^3) appeared to be the NOAEL for fibrosis. Although it has been suggested that fibrosis in animals is a precursor to carcinogenesis, a definite link has not been shown for RCFs or other fibers. No lung cancers were found in hamsters exposed to RCF1 [McConnell et al. 1995].

Table 8–1. Doses and dimensions of RCFs* in chronic inhalation studies with F344 rats

Reference	Fiber type	Dose (mg/m³)	WHO f/cm³	WHO SD	Total f/cm³	Total SD	% Fibers >20 µm long	Mean fiber diameter† µm	Mean fiber diameter† SD	Mean fiber length† µm	Mean fiber length† SD
Mast et al. 1995a	RCF1	30	187	53	234	35	43	0.98	0.61	22.3	17.0
Mast et al. 1995b	RCF1	6	120	35	162	37	43	0.98	0.61	22.3	17.0
		9	75	35	91	34	—	—	—	—	—
		3	26	12	36	17	—	—	—	—	—
		0	0	—	0	—	—	—	—	—	—
Mast et al. 1995a	Chrysotile asbestos	10	1.06	$+1.14\times10^4$	1×10^5		NR	0.10	0.15	2.2	3.0

*Abbreviations: NR=not reported; RCFs=refractory ceramic fibers; SD=standard deviation; WHO=World Health Organization.
†Arithmetic mean.

Table 8–2. Results of RCF* chronic inhalation studies with F344 rats

Reference	Fiber type	Dose (mg/m³)	WHO f/cm³	WHO SD	Time of first occurrence (months) Interstitial fibrosis	Time of first occurrence (months) Pleural fibrosis	Lung neoplasms Number	Lung neoplasms %	Pleural mesotheliomas Number	Pleural mesotheliomas %
Mast et al. 1995a	RCF1	30	187	53	6	9	16/123	13	2/123	1.6
Mast et al. 1995b	RCF1	16	120	35	12	12	2/124	1.6	0	—
		9	75	35	12	18	5/127	3.9	1/127	0.8
		3	26	12	None	None	2/123	1.6	0	—
		0	0	—	None	None	1/129	0.8	0	—
Mast et al. 1995a	Chrysotile asbestos	10	1.06	$+1.14\times10^4$	3	9	13/69	18.8	1/69	1.4

*Abbreviations: RCF=refractory ceramic fiber; SD=standard deviation; WHO=World Health Organization.

Table 8–3. Doses and dimensions of RCF* in chronic inhalation studies with Syrian golden hamsters

Reference	Fiber type	Dose (mg/m³)	WHO f/cm³	WHO SD	Total f/cm³	Total SD	% Fibers >20 µm long %	% Fibers >20 µm long f/cm³	% Fibers >20 µm long SD	Mean fiber diameter† µm	Mean fiber diameter† SD	Mean fiber length† µm	Mean fiber length† SD
McConnell et al. 1995	RCF1	30	215	56	256	58	43	—	—	0.94	0.63	22.1	16.7
McConnell et al. 1995	Chrysotile asbestos	10	3.0×10^3	1.4×10^3	8.4×10^4	9.0×10^4	NR	—	—	0.09	0.06	1.68	2.71
McConnell et al. 1999	Amosite asbestos	7.1	263	90	NR	—	~26	69	24	0.60	0.25	13.4	16.7
		3.7	165	61	—	—	~23	38	14	—	—	—	—
		0.8	36	23	—	—	~28	10	6	—	—	—	—

*Abbreviations: NR=not reported; RCFs=refractory ceramic fibers; SD=standard deviation; WHO=World Health Organization.
†Arithmetic mean.

Table 8–4. Results of RCF* chronic inhalation studies with Syrian golden hamsters

Reference	Fiber type	Dose (mg/m³)	WHO f/cm³	WHO SD	Time of first occurrence: Interstitial fibrosis	Time of first occurrence: Pleural fibrosis	Hamsters with pleural mesotheliomas† Number	Hamsters with pleural mesotheliomas† %
McConnell et al. 1995	RCF1	30	215	56	6 months	6 months	42/123	41.6
McConnell et al. 1995	Chrysotile asbestos	10	3.0×10^3	1.4×10^3	3 months	6 months	0	—
McConnell et al. 1995	Amosite asbestos	7.1	263	90	13 weeks	13 weeks	17/87	19.5
		3.7	165	61	13 weeks	13 weeks	22/85	25.9
		0.8	36	23	13 weeks	13 weeks	3/83	3.6

*Abbreviations: RCF=refractory ceramic fiber; SD=standard deviation; WHO=World Health Organization.
†No lung neoplasms were detected.

Chronic inhalation exposure to RCF1 at 30 mg/m^3 induced a 41% (42/102) incidence of mesotheliomas in Syrian golden hamsters [McConnell et al. 1995]. Determining a dose-response relationship for inducing mesothelioma is not possible based on currently available data because of the single exposure dose tested in the hamster and because of the low, sporadic occurrence of mesothelioma in the exposed rats [Mast et al. 1995a]. Yet the occurrence of mesotheliomas in the rat and the high incidence in the hamster are biologically significant because the spontaneous incidence of mesotheliomas in rats and hamsters has historically been very low [Analytical Sciences Incorporated 1999].

To assess the significance of the mesothelioma incidence observed in RCF-exposed hamsters, these results were compared with those obtained from hamsters that were exposed to chrysotile asbestos and were used as positive controls for the study [McConnell et al. 1995] (see Tables 8–3 and 8–4). However, the chrysotile-exposed hamsters failed to develop any tumors and therefore could not be considered true positive controls. Based on these results, a potency ranking could not be assigned for RCFs relative to chrysotile, since the carcinogenic response rate for the latter was zero in this study. The two fibers tested also differed with regard to their dose and fiber dimension.

The McConnell et al. [1999] study of hamsters exposed to amosite asbestos provides dose-response data for comparison with the RCF1 data of McConnell et al. [1995] (See Tables 8–3 and 8–4.). These separate studies examined the effects of RCF1 or amosite asbestos in hamsters using relatively similar exposure conditions, experimental conditions, and fiber dimensions [McConnell et al. 1995, 1999]. Exposure to 263 WHO f/cm^3 and 165 WHO f/cm^3 of amosite asbestos induced pleural mesotheliomas in 20% and 26% of the hamsters, respectively [McConnell et al. 1999]. A concentration of 215 RCF1 WHO f/cm^3 induced mesotheliomas in 41% of hamsters [McConnell et al. 1995]. Interstitial and pleural fibrosis were first observed at 13 weeks following amosite asbestos exposure and at 6 months following RCF1 exposure. Although average fiber dimensions for the RCF1 and amosite asbestos samples were similar, the RCF1 sample contained a higher percentage of fibers longer than 20 μm [McConnell et al. 1995, 1999]. Longer fibers have been associated with increased toxicity in experimental animal studies [Davis et al. 1986; Pott et al. 1987; Davis and Jones 1988; Warheit 1994; Blake et al. 1998].

Results from a dose-response analysis using the mesothelioma data from the RCF and amosite asbestos hamster studies [McConnell et al. 1995, 1999] indicated that the carcinogenic potency estimates for RCFs ranged from about half to two times the carcinogenic potency estimates for amosite asbestos [Dankovic 2001] (see Section 5.1.2). This analysis may not predict the mesothelioma risk in humans, since RCF1 contained a greater percentage of fibers longer than 20 μm and because of differences in fiber durability. Amosite asbestos is a more durable fiber with a longer in vivo half-life than RCF1 [Maxim et al. 1999b; Hesterberg et al. 1993] (see Table 8–5). Yet RCFs are more durable and less soluble than many other types of SVFs that have not demonstrated carcinogenicity in experimental studies. This characteristic is significant, as the durability of asbestos and SVFs (including RCFs) may be linked to the risk of lung cancer in animals chronically exposed to these fibers [Bignon et al. 1994; Bender and Hadley 1994; Hammad et al. 1988; Luoto et al. 1995]. Because of the long latency period for the development of mesotheliomas in humans, Berry [1999] hypothesized that fibers of sufficient durability are needed to cause this disease in humans. Extrapolation of the RCF dose-response data for lung cancer and mesothelioma in exposed rodents should take into

Table 8–5. Dissolution constant (K_{dis}) and weighted in vivo half-life ($t_{0.5}$) of amosite asbestos and RCF1

Fiber type	K_{dis} (ng/cm² per hr)	$t_{0.5}$(days)
RCF1	7.6	89.6
Amosite asbestos	1.3	418.0

Source: Adapted from Maxim et al. [1999].
*Abbreviation: RCF=refractory ceramic fiber.

account the durability of RCFs in humans. Some evidence indicates that rats are less sensitive than humans to the development of lung cancer and mesothelioma from exposure to asbestos and may therefore represent an inappropriate model for human risk assessment. Pott et al. [1994] hypothesized that in chronic inhalation studies, rats may have a lower sensitivity to inorganic fiber toxicity than humans. The lung cancer risk from inhaling asbestos may be two orders of magnitude lower in rats than in humans, and the mesothelioma risk from inhaling asbestos may be three orders of magnitude lower for rats. Rödelsperger and Woitowitz [1995] measured amphibole fiber concentration in the lung tissues of humans with mesothelioma and compared the results with fiber burdens reported in rats. A significantly increased OR (OR=4.8, 95%; CI=1.05–21.7) for mesothelioma was seen in humans with amphibole concentrations between 0.1 and 0.2 fiber/μg of dried lung tissue. The lowest tissue concentration reported to produce a significant carcinogenic response in rat inhalation studies of amphiboles (specifically crocidolite) was 1,250 fibers/μg of dried lung tissue. By comparing these results, Rödelsperger and Woitowitz [1995] estimated that humans are at least 6,000 times more sensitive than rats to a given tissue concentration of amphibole fibers.

This work is refuted by other scientists who favor the rat as an appropriate model for evaluating the risk evaluation of lung cancer in humans [Maxim and McConnell 2001]. Limitations of the Rödelsperger and Woitowitz [1995] and Pott [1994] analyses (discussed earlier) include the lack of a dose-response analysis, analysis of only one epidemiologic study, inadequate comparisons of exposure duration, lack of accounting for the potentially multiplicative effect of smoking and asbestos exposure, lack of consideration of latency and clearance, and different fiber measurement techniques.

In summary, multiple factors affecting the comparability of different fiber types and animal models reported in the literature make it difficult to determine whether the carcinogenic potency of RCFs in animals is similar to that in humans. Extrapolation of the animal data to humans is further complicated by a limited understanding of the mechanisms of fiber toxicity. Consequently, any extrapolation of the cancer risk found in animals exposed to RCFs must account for differences between humans and rodents with regard to fiber deposition and clearance patterns, uncertainty about the role of RCF durability for potentiating an adverse effect, and possible species differences in sensitivity to fibers.

8.2.2 Health Effects Studies of Workers Exposed to RCFs

Two major research efforts evaluated the morbidity of workers exposed to airborne fibers in the RCF manufacturing industry. One study was conducted in the United States and the other in Europe. The objective of each was to evaluate the relationship between occupational exposure to RCFs and potential adverse health effects. These studies contained multiple components including standardized respiratory and occupational history questionnaires, chest radiographs, pulmonary function tests of workers, and air sampling to estimate worker exposures. The first studies of European plants were conducted in 1986 and included current workers at seven RCF manufacturing plants [Rossiter et al. 1994; Trethowan et al. 1995; Burge et al. 1995]. A followup cross-sectional study conducted in 1996 evaluated the same medical endpoints in workers from six of these seven European manufacturing plants (one plant had ceased operation) [Cowie et al. 1999, 2001]. Current as well as former workers were included as study subjects in the followup study. The studies of U.S. plants began in 1987 and involved evaluations of current workers at five RCF manufacturing plants and former workers at two of the plants [Lemasters et al. 1994, 1998, 2003; Lockey et al. 1993, 1996, 1998, 2002]. In the United States, the earliest commercial production of RCFs and RCF products began in 1953. In Europe, RCF production began in 1968. The demographics of the U.S. and European populations were similar at the time they were studied, but the average age and duration of employment for the U.S. workers were slightly higher than for the workforce in the 1986 European studies because of the earlier development of this industry in the United States.

8.2.2.1 Pleural changes in humans

The radiographic analyses of the U.S. and 1996 European populations in RCF manufacturing detected an association between pleural changes and RCF exposure [Lemasters et al. 1994; Lockey et al. 1996; Cowie et al. 1999, 2001]. In the initial European studies, Trethowan et al. [1995] found pleural abnormalities in a small number of RCF workers who had other confounding exposures that did not include asbestos. Differences observed in pleural abnormalities between the U.S. and European worker populations may be related to the latency of exposure required to cause specific pleural changes [Hillerdal 1994; Begin et al. 1996], especially the formation of pleural plaques, which were first observed in studies of the U.S. RCF manufacturing industry, with its longer latency period. Historical air sampling data also indicate that airborne fiber concentrations were much higher in early U.S. RCF manufacturing. Therefore, in addition to their longer overall latency, RCF manufacturing workers in the United States probably had generally higher exposures in the early years of the industry than did their European counterparts. These factors might explain the appearance of RCF-associated pleural plaques in the U.S. studies before their detection in the European studies. The U.S. and 1986 European studies yielded little evidence of an association between radiographic parenchymal opacities and RCF exposure. In the U.S. study, small opacities were rare, with only three cases noted in one report [Lockey et al. 1996] and only one case (with small rounded opacities of profusion category 3/2 attributable to prior kaolin mine work) noted in the other [Lemasters et al. 1994]. Small opacities of profusion category 1/0 or greater were more frequent in the European study by Trethowan et al. [1995], but confounding exposures were believed to account for many of these cases. The results of statistical analyses indicated either no

association with RCF exposure [Trethowan et al. 1995] or an association slightly suggestive of an RCF exposure effect [Rossiter et al. 1994]. In a more comprehensive evaluation of the European study population, small opacities of category 1/0 or greater were positively associated with RCF exposures that occurred before 1971 [Cowie et al. 1999].

8.2.2.2 Respiratory symptoms, irritation, and other conditions in humans

In both the U.S. [Lockey et al. 1993; Lemasters et al. 1998] and the European [Trethowan et al. 1995; Burge et al. 1995; Cowie et al. 1999, 2001] studies, occupational exposure to RCFs was associated with various reported respiratory conditions or irritation symptoms after adjusting for the effects of smoking. Worker exposure to RCFs at concentrations of 0.2 to 0.6 f/cm^3 was associated with statistically significant increases in eye irritation (OR=2.16, 95% CI=1.32–3.54), stuffy nose (OR=2.06, 95% CI=1.25–3.39), and dry cough (OR=2.53, 95% CI=1.25–5.11) compared with exposure to concentrations lower than 0.2 f/cm^3 [Trethowan et al. 1995]. Between the 0.2 to 0.6 f/cm^3 and >0.6 f/cm^3 RCF exposure groups, a statistically significant increase occurred in ORs for wheezing ($P<0.0001$), grade 2 dyspnea ($P<0.05$), eye irritation ($P<0.0001$), and skin irritation ($P<0.0001$)—but not for stuffy nose [Trethowan et al. 1995]. Lockey et al. [1993] found that dyspnea was significantly associated with cumulative exposure to >15 fiber-months/cm^3 (i.e., >1.25 fiber-year/cm^3) relative to cumulative exposure to ≤15 fiber-months/cm^3 (dyspnea grade 1–OR=2.1, 95% CI 1.3–3.3; dyspnea grade 2–OR=3.8, 95% CI 1.6–9.4) after adjusting for smoking and other potential confounders. Lockey et al. [1993] also found a statistically significant association between cumulative RCF exposure and pleurisy (OR=5.4, 95% CI=1.4–20.2), and an elevated but nonsignificant association between cumulative RCF exposure and chronic cough (OR=2.0, 95% CI=1.0–4.0). Lemasters et al. [1998] also noted associations ($P<0.05$) between employment in an RCF production job and increased prevalence of dyspnea and the presence of at least one respiratory symptom after adjusting the data for confounders. Recurrent chest illness in the European study population was associated with the estimated cumulative exposure to thoracic-sized fibers but was more strongly associated with estimated cumulative exposure to thoracic-sized dust [Cowie et al. 1999, 2001].

In cross-sectional analyses, both the U.S. [Lockey et al. 1998; Lemasters et al. 1998] and the 1986 European [Trethowan et al. 1995; Burge et al. 1995] studies found that cumulative RCF exposure is associated with pulmonary function decrements among current and former smokers. Lemasters et al. [1998] also found statistically significant deficits in pulmonary function measures for nonsmoking female workers. The decreased pulmonary function in the European study population remained significantly associated with cumulative RCF exposure, even after controlling for cumulative dust exposure [Burge et al. 1995]. The 1996 European study found pulmonary function decrements only in current smokers [Cowie et al. 1999, 2001]. In a longitudinal analysis of data from multiple serial pulmonary function tests, Lockey et al. [1998] concluded that the more recent RCF concentrations occurring after 1987 were not associated with decreased pulmonary function; rather, decreases in pulmonary function were more closely related to typically higher concentrations that occurred before this time period. The U.S. and European studies suggest that decrements in pulmonary function observed primarily in current and former smokers are evidence of an interactive effect between smoking and RCF exposure.

8.2.3 Carcinogenic Risk in Humans

Moolgavkar et al. [1999] derived risk estimates for lung cancer in humans on the basis of the

results from the two chronic bioassays of RCFs in male Fischer 344 rats [Mast et al. 1995a,b]. Several models (linear, quadratic, exponential) were used to estimate and compare risks using reference populations comprised of either a nonsmoking ACS cohort or a cohort of steel workers not exposed to coke oven emissions (see Table 5–10 for risk estimates). The exponential model provided the best statistical fit of the data. The linear model provided the highest estimates of human lung cancer risks from exposure to RCFs when used with the referent steel workers cohort (considered to be most representative of workers exposed to RCFs because it includes blue collar workers who smoke). Lung cancer risk estimates were calculated using each model at exposure concentrations of 0.25 f/cm^3, 0.5 f/cm^3, 0.75 f/cm^3, and 1.0 f/cm^3. The RCF-related lung cancer risk determined from the linear model for the lowest concentration (0.25 f/cm^3) was 0.27/1,000 for the cohort of steel workers compared with 0.036/1,000 using the exponential model and 0.00088/1,000 for the quadratic model when using the same referent population.

The risk estimates incorporated multiple assumptions, including a human breathing rate of 13.5 L/min (considered light work) and multiple criteria for defining the length of time a worker could be exposed to RCFs over a working lifetime. Higher risk estimates could be expected if the assumptions more closely represented those used by NIOSH and OSHA: (1) a human breathing rate of 20 L/min and (2) a worker exposure duration of 8 hr/day, 5 days/wk, 50 wk/yr, from age 20 to 65 with the risk calculated beyond age 70 (e.g., to age 85). Furthermore, the calculated risk estimates could be an underestimation of the lung cancer risk to humans because the models assumed that the tissue sensitivity to RCFs in the rat is equal to that in humans. Although the lung cancer risk estimates derived from the rat data are reason for concern, estimates of human risk for mesothelioma from the high incidence (41%) of mesothelioma in hamsters cannot be appropriately modeled since only a single exposure was administered in the study. Primarily on the basis of chronic animal inhalation studies [Mast et al. 1995a,b; McConnell et al. 1995], NIOSH concludes that RCFs are a potential occupational carcinogen. Furthermore, the evidence of pleural plaques [Lemasters et al. 1994; Lockey et al. 1996] observed in persons with occupational exposures to airborne RCFs is clinically similar to that observed in asbestos-exposed persons after the initial years of their occupational asbestos exposures [Hourihane et al. 1966; Becklake et al. 1970; Dement et al. 1986]. NIOSH considers the discovery of pleural plaques in U.S. studies of RCF manufacturing workers to be a significant finding because the plaques were correlated with RCF exposure [Lemasters et al. 1994; Lockey et al. 1996]. In addition, NIOSH considers the respiratory symptoms and conditions (including dyspnea, wheeze, cough, and pleurisy) [Lemasters et al. 1998; Lockey et al. 1993; Trethowan et al. 1995; Burge et al. 1995; Cowie et al. 1999] in RCF workers to be adverse health effects that have been associated with exposure to airborne fibers of RCFs.

Insufficient evidence exists to document an association between fibrotic or carcinogenic effects and the inhalation of RCFs by workers in the RCF manufacturing industry though these effects have been demonstrated in animal studies. The lack of an observed association between RCF exposure and these effects among workers could be affected by one or more factors, including several relating to the study population: the relatively small cohort, the proportion of workers with short tenure relative to what might be expected (on the basis of an asbestos analogy) in terms of disease latency, and workers with limited cumulative exposures to RCFs.

8.2.4 Controlling RCF Exposures in the Workplace

Table 8–6 summarizes exposure monitoring data collected by the RCFC under a consent agreement with the EPA [Everest 1998; Maxim et al. 1997]. These data indicate that exposures to RCFs during 1993–1998 had an AM fiber concentration of about 0.3 f/cm^3 for manufacturing and nearly 0.6 f/cm^3 for end users. Maxim et al. [1997, 1999a] reported results for both manufacturing and end-use sectors in which airborne fiber concentrations through 1997 were reduced to an AM <0.3–0.6 f/cm^3.

The exposure monitoring data collected as part of the RCFC/EPA consent agreement provide assurance that when appropriate engineering controls and work practices are used, airborne exposure to RCFs can be maintained for most functional job categories (FJCs) at the REL of 0.5 f/cm^3. For many manufacturing processes, reductions in exposures have resulted from the improved ventilation, engineering or process changes, and product stewardship programs [Rice et al. 1996; Maxim et al. 1999b]. These data provide the basis for the NIOSH determination that a REL of 0.5 f/cm^3 as a TWA can be achieved.

Although many RCF manufacturing and end-user job tasks have exposures to RCFs at concentrations below 0.5 f/cm^3, exposure monitoring data also indicate that not all FJCs may be able to achieve these RCF concentrations consistently. FJCs that currently experience airborne AM fiber concentrations >0.5 f/cm^3 include finishing (manufacturing and end use) and removal (end use). Typical processing during finishing operations (e.g., sawing, drilling, cutting, sanding) often requires high-energy sources that tend to generate larger quantities of airborne dust and fibers. For RCF insulation removal, activities are performed at remote sites where conventional engineering controls and fixed ventilation systems are more difficult to implement. For some operations, such as removal of RCF insulation tiles from furnaces, the release of high airborne fiber concentrations can occur. However, removal of RCF insulation tiles is not routine and is generally accomplished in a short period of time. Workers almost universally wear PPE and respiratory protection during these job tasks [Maxim et al. 1997, 1998].

NIOSH acknowledges that the frequent use of PPE, including respirators, may be required for some workers handling RCFs or RCF products. The frequent use of PPE may be required during job tasks for which (1) routinely high airborne concentrations of RCF (e.g., finishing, insulation removal) exist, (2) the airborne concentration of RCF is unknown or unpredictable, and (3) job tasks are associated with highly variable airborne concentrations because of environmental conditions or the manner in which the job task is performed. In all work environments where RCFs or RCF products are handled, control of exposure through the engineering controls should be the highest priority.

8.3 Summary

The following summarize the relevant information used as the basis for the NIOSH assessment of occupational exposures to RCFs:

- Airborne concentrations of RCFs have been characterized as containing fibers of dimensions in the thoracic and respirable size ranges. RCFs are among the most durable types of SVFs. In tests of solubility, RCFs are nearly as durable as chrysotile asbestos but significantly less durable than amphibole asbestos fibers such as amosite.
- Chronic, nose-only inhalation studies with RCFs in animals show a statistically

Table 8–6. Airborne fiber concentrations in the RCF* industry during 1993–1998, by functional job category and production status† (f/cm³ as TWA)

Functional job category and production status	Minimum value	First quartile	Median	Geometric mean	Arithmetic mean	Third quartile	Maximum value
Total:							
Manufacturing	0.001	0.070	0.186	0.16	0.313	0.407	7.700
End use	0.002	0.052	0.173	0.16	0.560	0.524	30.000
Assembly:							
Manufacturing	0.001	0.110	0.208	0.18	0.281	0.366	2.154
End use	0.002	0.050	0.159	0.14	0.316	0.402	2.837
Auxiliary:							
Manufacturing	0.001	0.019	0.038	0.05	0.112	0.132	1.347
End use	0.002	0.021	0.066	0.07	0.198	0.198	2.678
Fiber:							
Manufacturing	0.004	0.063	0.145	0.14	0.257	0.299	7.700
End use	—	—	—	—	—	—	—
Finishing:							
Manufacturing	0.004	0.316	0.488	0.47	0.663	0.803	4.044
End use	0.006	0.124	0.383	0.35	0.991	0.986	30.000
Installation:							
Manufacturing	—	—	—	—	—	—	—
End use	0.003	0.084	0.236	0.20	0.434	0.559	3.371
Mixing/forming:							
Manufacturing	0.004	0.090	0.184	0.17	0.292	0.364	1.445
End use	0.010	0.074	0.159	0.17	0.319	0.369	4.109
Other:							
Manufacturing	0.007	0.027	0.070	0.07	0.112	0.177	1.900
End use	0.003	0.013	0.030	0.04	0.194	0.102	6.400
Removal:							
Manufacturing	—	—	—	—	—	—	—
End use	0.010	0.373	1.914	0.82	1.816	2.340	16.000

Source: Adapted from Everest [1998].
*Abbreviations: RCF = refractory ceramic fiber; TWA = time-weighted average.
†Fiber concentrations were determined during monitoring performed over a 5-year period (1993–1998) under the Refractory Ceramic Fibers Coalition/Environmental Protection Agency (RCFC/EPA) consent agreement. Concentrations were determined by NIOSH method 7400 "B" counting rules.

significant increased incidence of lung tumors in rats and pleural mesotheliomas in hamsters. These data support the NIOSH determination that RCFs are a potential occupational carcinogen.

- Epidemiologic studies of workers in the RCF manufacturing industry show an increased incidence of pleural plaques, respiratory symptoms (dyspnea and cough), skin and eye irritation, and decreased pulmonary function related to increasing exposures to airborne fibers. Some of these conditions are documented for exposure concentrations in a range as low as 0.2 to 0.6 f/cm^3. Studies of workers exposed to airborne RCFs show no evidence of excess risk for lung cancer or mesothelioma. However, the inability to detect such an association could be because of (1) the low statistical power for detecting an effect, (2) the short latency period for most workers occupationally exposed, and (3) the historically low and decreasing fiber exposures that have occurred in this industry.

- Risk assessment analyses using data from chronic inhalation studies in rats indicate that the excess risk of developing lung cancer when exposed to RCFs at a TWA of 0.5 f/cm^3 for a working lifetime is 0.073 to 1.2/1,000. However, on the basis of the assumptions used in the risk analyses, NIOSH concludes that this risk estimate is more likely to underestimate than to overestimate the risk to RCF-exposed workers. Reduction of the RCF TWA concentration to 0.2 f/cm^3 would reduce the risk for lung cancer to 0.03 to 0.47/1,000. OSHA attempts to set PELs for carcinogens that reflect an estimated risk of 1/1,000 but is limited by considerations of technologic and economic feasibility.

- RCF exposure data gathered under a consent agreement between RCFC and EPA, which included a 5-year comprehensive air monitoring program (1993–1998), indicate that airborne exposure concentrations to RCFs have been decreasing. Monitoring results show that 75% to >95% of TWA exposure concentration measurements in all FJCs (with one exception) were below 1.0 f/cm^3. In all but two of the eight FJCs, >70% of TWA measurements were below the RCFC recommended exposure guideline of 0.5 f/cm^3. On the basis of the review of these data, NIOSH has concluded that the REL of 0.5 f/cm^3 can be achieved in most work places where RCFs or RCF products are manufactured or used.

- The combined effect of mixed exposures to fibers and nonfibrous particulates may contribute to increased irritation of the respiratory tract, skin, and eyes. Engineering controls and appropriate work practices used to keep airborne RCF concentrations below the REL should help to minimize airborne exposures to nonfibrous particulates as well. Because the ratio of fibers to nonfibrous particulate in airborne exposures may vary among job tasks, exposure monitoring should include efforts to characterize particulate composition and to control and minimize airborne fibers and nonfibrous particulate accordingly.

From the assessment described above and throughout this document, NIOSH concludes that on a continuum of fiber toxicity, RCFs relate more closely to asbestos than to fibrous glass and other SVFs and should be handled accordingly. NIOSH considers all asbestos types to be carcinogens and has established a REL of 0.1 f/cm^3 for airborne asbestos fibers. This value was determined on the basis of extensive human and animal health effects data and the recognized limits of analytical methods.

Recognizing that RCFs are carcinogens in animal studies and given the limitations in deriving an exposure value that reflects no excess risk of lung cancer or mesothelioma for humans, NIOSH recommends that every effort be made to keep exposures below the REL of 0.5 f/cm^3 as a TWA for up to 10 hr/day in a 40-hr workweek. These efforts will further reduce the risk for malignant respiratory disease and protect workers from conditions and symptoms deriving from irritation of the respiratory tract, skin, and eyes.

From the analysis of historical exposure data (see Chapter 4) and the exposure data collected as part of the RCFC/EPA consent agreement monitoring program (Table 8–6), NIOSH has determined that compliance with the REL for RCFs is achievable in most manufacturing and end-use job categories. Although routine attainment of TWA exposures below the REL may not currently occur at all job tasks, it does represent a reasonable objective that can be achieved through modification of the job task or the introduction or improvement of ventilation controls.