some miners, and that miners experience COPD at a significantly higher rate than the general population (Becklake 1989, 1992; Oxman 1993; NIOSH 1995). This would appear to place affected miners in a subpopulation specifically identified as susceptible to the adverse health effects of respirable particle pollution (EPA, 1996). The Mine Act requires that standards ''* * * most adequately assure on the basis of the best available evidence that *no miner* suffer material impairment of health or functional capacity * * *'' (Section 101(a)(6), emphasis added).

In sum, MSHA believes it would be a serious omission to ignore the body of evidence from air pollution studies and the Agency is, therefore, taking that evidence into account. The Agency would, however, welcome additional scientific information and analysis on ways of applying this body of evidence to miners experiencing acute and/or chronic dpm exposures. MSHA is especially interested in receiving information on whether the elevated prevalence of COPD among miners makes them, as a group, highly susceptible to the harmful effects of fine particulate air pollution, including dpm.

III.2.b. Acute Health Effects

Information relating to the acute health effects of dpm includes anecdotal reports of symptoms experienced by exposed miners, studies based on exposures to diesel emissions, and studies based on exposures to particulate matter in the ambient air. These will be discussed in turn.

III.2.b.i. Symptoms Reported by Exposed Miners

Miners working in mines with diesel equipment have long reported adverse effects after exposure to diesel exhaust. For example, at the workshops on dpm conducted in 1995, a miner reported headaches and nausea among several operators after short periods of exposure (dpm Workshop; Mt. Vernon, IL, 1995). Another miner reported that the smoke from equipment using improper fuel or not well maintained is an irritant to nose and throat and impairs vision. ''We've had people sick time and time again * * * at times we've had to use oxygen for people to get them to come back around to where they can feel normal again.'' (dpm Workshop; Beckley, WV, 1995). Other miners (dpm Workshops; Beckley, WV, 1995; Salt Lake City, UT, 1995), reported similar symptoms in the various mines where they worked.

Kahn *et al.* (1988) conducted a study of the prevalence and seriousness of such complaints, based on United Mine Workers of America records and subsequent interviews with the miners involved. The review involved reports at five underground coal mines in Utah and Colorado between 1974 and 1985. Of the 13 miners reporting symptoms: 12 reported mucous membrane irritation, headache and light-headiness; eight reported nausea; four reported heartburn; three reported vomiting and weakness, numbness, and tingling in extremities; two reported chest tightness; and two reported wheezing (although one of these complained of recurrent wheezing without exposure). All of these incidents were severe enough to result in lost work time due to the symptoms (which subsided within 24 to 48 hours).

MSHA welcomes additional information about such effects including information from medical personnel who have treated miners and information on work time lost, together with information about the exposures of miners for whom such effects have been observed. The Agency would be especially interested in comparisons of effects observed in workers subjected to filtered exhaust as compared to those subjected to unfiltered exhaust.

III.2.b.ii. Studies Based on Exposures to Diesel Emissions

Several scientific studies have been conducted to investigate acute effects of exposure to diesel emissions.

In a clinical study (Battigelli, 1965), volunteers were exposed to different levels of diesel exhaust and then the degree of eye irritation was measured. Exposure for ten minutes to diesel exhaust produced ''intolerable'' irritation in some subjects while the average irritation score was midway between ''some'' irritation and a ''conspicuous but tolerable'' irritation level. Cutting the exposure by 50% significantly reduced the irritation.

In a study of underground iron ore miners exposed to diesel emissions, Jörgensen and Svensson (1970), found no difference in spirometry measurements taken before and after a work shift. Similarly, Ames *et al.* (1982), in a study of coal miners exposed to diesel emissions, detected no statistically significant relationship between exposure and pulmonary function. However, the authors noted that the lack of a positive result might be due to the low concentrations of diesel emissions involved.

Gamble *et al.* (1978) did observe decreases in pulmonary function over a single shift in salt miners exposed to diesel emissions. Pulmonary function appeared to deteriorate in relation to the concentration of diesel exhaust, as indicated by NO_2; but this effect was confounded by the presence of NO_2 due to the use of explosives.

Gamble *et al.* (1987a) assessed response to diesel exposure among 232 bus garage workers by means of a questionnaire and before- and after-shift spirometry. No significant relationship was detected between diesel exposure and change in pulmonary function. However, after adjusting for age and smoking status, a significantly elevated prevalence of reported symptoms was found in the high-exposure group. The strongest associations with exposure were found for eye irritation, labored breathing, chest tightness, and wheeze. The questionnaire was also used to compare various acute symptoms reported by the garage workers and a similar population of workers at a lead acid battery plant who were not exposed to diesel fumes. The prevalence of work-related eye irritations, headaches, difficult or labored breathing, nausea, and wheeze was significantly higher in the diesel bus garage workers, but the prevalence of work-related sneezing was significantly lower.

Ulfvarson *et al.* (1987) studied effects over a single shift on 47 stevedores exposed to dpm at particle concentrations ranging from 130 $\mu g/m^3$ to 1000 $\mu g/m^3$. A statistically significant loss of pulmonary function was observed, with recovery after 3 days of no occupational exposure.

To investigate whether removal of the particles from diesel exhaust might reduce the ''acute irritative effect on the lungs'' observed in their earlier study, Ulfvarson and Alexandersson (1990) compared pulmonary effects in a group of 24 stevedores exposed to unfiltered diesel exhaust to a group of 18 stevedores exposed to filtered exhaust, and to a control group of 17 occupationally unexposed workers. Workers in all three groups were nonsmokers and had normal spirometry values, adjusted for sex, age, and height, prior to the experimental workshift.

In addition to confirming the earlier observation of significantly reduced pulmonary function after a single shift of occupational exposure, the study found that the stevedores in the group exposed only to filtered exhaust had 50–60% less of a decline in forced vital capacity (FVC) than did those stevedores who worked with unfiltered equipment. Similar results were observed for a subgroup of six stevedores who were exposed to filtered exhaust on one shift and unfiltered exhaust on another. No loss of pulmonary function was observed for the unexposed control group. The authors suggested that these results ‘‘support the idea that the irritative effects of diesel exhausts to the lungs [sic] is the result of an interaction between particles and gaseous components and not of the gaseous components alone.’’ They concluded that ‘‘* * * it should be a useful practice to filter off particles from diesel exhausts in work places even if potentially irritant gases remain in the emissions.’’

Rudell *et al.*, (1996) carried out a series of double-blind experiments on 12 healthy, non-smoking subjects to investigate whether a particle trap on the tailpipe of an idling diesel engine would reduce acute effects of diesel exhaust, compared with exposure to unfiltered exhaust. Symptoms associated with exposure included headache, dizziness, nausea, tiredness, tightness of chest, coughing, and difficulty in breathing, but the most prominent were found to be irritation of the eyes and nose, and a sensation of unpleasant smell. Among the various pulmonary function tests performed, exposure was found to result in significant changes only as measured by increased airway resistance and specific airway resistance. The ceramic wall flow particle trap reduced the number of particles by 46 percent, but resulted in no significant attenuation of symptoms or lung function effects. The authors concluded that diluted diesel exhaust caused increased symptoms of the eyes and nose, unpleasant smell, and bronchoconstriction, but that the 46 percent reduction in median particle number concentration observed was not sufficient to protect against these effects in the populations studied.

Wade and Newman (1993) documented three cases in which railroad workers developed persistent asthma following exposure to diesel emissions while riding immediately behind the lead engines of trains having no caboose. None of these workers were smokers or had any prior history of asthma or other respiratory disease. Although this is the only published report MSHA knows of directly relating exposure to diesel emissions with the development of asthma, there have been a number of recent studies indicating that dpm exposure can induce bronchial inflammation and respiratory immunological allergic responses in humans. These are reviewed in Peterson and Saxon (1996) and Diaz-Sanchez (1997).

III.2.b.iii. Studies Based on Exposures to Particulate Matter in Ambient Air

As early as the 1930’s, as a result of an incident in Belgium’s industrial Meuse Valley, it was known that large increases in particulate air pollution, created by winter weather inversions, could be associated with large simultaneous increases in mortality and morbidity. More than 60 persons died from this incident, and several hundred suffered respiratory problems. The mortality rate during the episode was more than ten times higher than normal, and it was estimated that over 3,000 sudden deaths would occur if a similar incident occurred in London. Although no measurements of pollutants in the ambient air during the episode are available, high PM levels were obviously present (EPA, 1996).

A significant elevation in particulate matter (along with SO_2 and its oxidation products) was measured during a 1948 incident in Donora, PA. Of the Donora population, 42.7 percent experienced some adverse health effect, mainly due to irritation of the respiratory tract. Twelve percent of the population reported difficulty in breathing, with a steep rise in frequency as age progressed to 55 years (Schrenk, 1949).

Approximately as projected by Firket (1931), an estimated 4,000 deaths occurred in response to a 1952 episode of extreme air pollution in London. The nature of these deaths is unknown, but there is clear evidence that bronchial irritation, dyspnea, bronchospasm, and, in some cases, cyanosis occurred with unusual prevalence (Martin, 1964).

These three episodes ‘‘left little doubt about causality in regard to the induction of serious health effects by very high concentrations of particle-laden air pollutant mixtures’’ and stimulated additional research to characterize exposure-response relationships (EPA, 1996). Based on several analyses of the 1952 London data, along with several additional acute exposure mortality analyses of London data covering later time periods, the U.S. Environmental Protection Agency (EPA) concluded that increased risk of mortality is associated with exposure to particulate and SO_2 levels in the range of 500–1000 $\mu g/m^3$. The EPA also concluded that relatively small, but statistically significant increases in mortality risk exist at particulate levels below 500 $\mu g/m^3$, with no indications of any specific threshold level yet indicated at lower concentrations (EPA, 1986).

Subsequently, between 1986 and 1996, increasingly sophisticated particulate measurements and statistical techniques have enabled investigators to address these questions more quantitatively. The studies on acute effects carried out since 1986 are reviewed in the 1996 EPA Air Quality Criteria for Particulate Matter, which forms the basis for the discussion below (EPA, 1996).

At least 21 studies have been conducted that evaluate associations between acute mortality and morbidity effects and various measures of fine particulate levels in the ambient air. These studies are identified in Tables III–2 and III–3. Table III–2 lists 11 studies that measured primarily fine particulate matter using filter-based optical techniques and, therefore, provide mainly qualitative support for associating observed effects with fine particles. Table III–3 lists quantitative results from 10 studies that reported gravimetric measurements of either the fine particulate fraction or of components, such as sulfates, that serve as indicators.

A total of 38 studies examining relationships between short-term particulate levels and increased mortality, including nine with fine particulate measurements, were published between 1988 and 1996 (EPA, 1996). Most of these found statistically significant positive associations. Daily or several-day elevations of particulate concentrations, at average levels as low as 18–58 $\mu g/m^3$, were associated with increased mortality, with stronger relationships observed in those with preexisting respiratory and cardiovascular disease. Overall, these studies suggest that an increase of 50 $\mu g/m^3$ in the 24-hour average of PM_{10} is associated with a 2.5 to 5-percent increase in the risk of mortality in the general population. Based on Schwartz et al. (1996), the relative risk of mortality in the general population increases by about 2.6 to 5.5 percent per 25 $\mu g/m^3$ of fine particulate ($PM_{2.5}$) (EPA, 1996).

A total of 22 studies were published on associations between short-term particulate levels and hospital admissions, outpatient visits, and emergency room visits for respiratory disease, Chronic Obstructive Pulmonary Disease (COPD), pneumonia, and heart disease (EPA, 1996). Fifteen of these studies were focussed on the elderly. Of the seven that dealt with all ages (or in one case, persons less than 65 years old), all showed positive results. All of the five studies relating fine particulate measurements to increased hospitalization, listed in Tables III–2 and III–3, dealt with general age populations and showed statistically significant associations. The estimated increase in risk ranges from 3 to 16 percent per 25 $\mu g/m^3$ of fine particulate. Overall, these studies are indicative of acute morbidity effects being related to fine particulate matter and support the mortality findings.

Most of the 14 published quantitative studies on ambient particulate exposures and acute respiratory symptoms were restricted to children (EPA, 1996). Although they generally showed positive associations, and may be of considerable biological relevance, evidence of toxicity in children is not necessarily applicable to adults. The few studies on adults have not produced statistically significant evidence of a relationship.

Fourteen studies since 1982 have investigated associations between ambient particulate levels and loss of pulmonary function (EPA, 1996). In general, these studies suggest a short term effect, especially in symptomatic groups such as asthmatics, but most were carried out on children only. In a study of adults with mild COPD, Pope and Kanner (1993) found a 29±10 ml decrease in 1-second Forced Expiratory Volume (FEV_1) per 50 μg/m^3 increase in PM_{10}, which is similar in magnitude to the change generally observed in the studies on children. In another study of adults, with PM_{10} ranging from 4 to 137 μg/m^3, Dusseldorp et al. (1995) found 45 and 77 ml/sec decreases, respectively, for evening and morning Peak Expiratory Flow Rate (PEFR) per 50 μg/m^3 increase in PM_{10} (EPA, 1996). In the only study carried out on adults that specifically measured fine particulate ($PM_{2.5}$), Perry et al. (1983) did not detect any association of exposure with loss of pulmonary function. This study, however, was conducted on only 24 adults (all asthmatics) exposed at relatively low concentrations of $PM_{2.5}$ and, therefore, had very little power to detect any such association.

III.2.c. Chronic Health Effects

During the 1995 dpm workshops, miners reported observable adverse health effects among those who have worked a long time in dieselized mines. For example, a miner (dpm Workshop; Salt Lake City, UT, 1995), stated that miners who work with diesel ‘‘have spit up black stuff every night, big black—what they call black (expletive) * * * [they] have the congestion every night * * * the 60-year-old man working there 40 years.’’ Scientific investigation of the chronic health effects of dpm exposure includes studies based specifically on exposures to diesel emissions and studies based more generally on exposures to fine particulate matter in the ambient air. Only the evidence from human studies will be addressed in this section. Data from genotoxicology studies and studies on laboratory animals will be discussed later, in the section on potential mechanisms of toxicity.

III.2.c.i. Studies Based on Exposures to Diesel Emissions

The discussion will summarize the epidemiological literature on chronic effects other than cancer, and then concentrate on the epidemiology of cancer in workers exposed to dpm.

III.2.c.i.A. Chronic Effects Other Than Cancer

There have been a number of epidemiological studies that investigated relationships between diesel exposure and the risk of developing persistent respiratory symptoms (i.e., chronic cough, chronic phlegm, and breathlessness) or measurable loss in lung function. Three studies involved coal miners (Reger *et al.*, 1982; Ames *et al.*, 1984; Jacobson *et al.*, 1988); four studies involved metal and nonmetal miners (Jörgenson & Svensson, 1970; Attfield, 1979; Attfield *et al.*, 1982; Gamble *et al.*, 1983). Three studies involved other groups of workers—railroad workers (Battigelli *et al.*, 1964), bus garage workers (Gamble *et al.*, 1987), and stevedores (Purdham *et al.*, 1987).

Reger *et al.* (1982) examined the prevalence of respiratory symptoms and the level of pulmonary function among more than 1,600 underground and surface coal miners, comparing results for workers (matched for smoking status, age, height, and years worked underground) at diesel and non-diesel mines. Those working at underground dieselized mines showed some increased respiratory symptoms and reduced lung function, but a similar pattern was found in surface miners who presumably would have experienced less diesel exposure. Miners in the dieselized mines, however, had worked underground for less than 5 years on average.

In a study of 1,118 coal miners, Ames *et al.* (1984) did not detect any pattern of chronic respiratory effects associated with exposure to diesel emissions. The analysis, however, took no account of baseline differences in lung function or symptom prevalence, and the authors noted a low level of exposure to diesel-exhaust contaminants in the exposed population.

In a cohort of 19,901 coal miners investigated over a 5-year period, Jacobsen *et al.* (1988) found increased work absence due to self-reported chest illness in underground workers exposed to diesel exhaust, as compared to surface workers, but found no correlation with their estimated level of exposure.

Jörgenson & Svensson (1970) found higher rates of chronic productive bronchitis, for both smokers and nonsmokers, among underground iron ore miners exposed to diesel exhaust as compared to surface workers at the same mine. No significant difference was found in spirometry results.

Using questionnaires collected from 4,924 miners at 21 metal and nonmetal mines, Attfield (1979) evaluated the effects of exposure to silica dust and diesel exhaust and obtained inconclusive results with respect to diesel exposure. For both smokers and non-smokers, miners occupationally exposed to diesel for five or more years showed an elevated prevalence of persistent cough, persistent phlegm, and shortness of breath, as compared to miners exposed for less than five years, but the differences were not statistically significant. Four quantitative indicators of diesel use failed to show consistent trends with symptoms and lung function.

Attfield *et al.* (1982) reported on a medical surveillance study of 630 white male miners at 6 potash mines. No relationships were found between measures of diesel use or exposure and various health indices, based on self-reported respiratory symptoms, chest radiographs, and spirometry.

In a study of salt miners, Gamble and Jones (1983) observed some elevation in cough, phlegm, and dyspnea associated with mines ranked according to level of diesel exhaust exposure. No association between respiratory symptoms and estimated cumulative diesel exposure was found after adjusting for differences among mines. However, since the mines varied widely with respect to diesel exposure levels, this adjustment may have masked a relationship.

Battigelli *et al.* (1964) compared pulmonary function and complaints of respiratory symptoms in 210 railroad repair shop employees, exposed to diesel for an average of 10 years, to a control group of 154 unexposed railroad workers. Respiratory symptoms were less prevalent in the exposed group, and there was no difference in pulmonary function; but no adjustment was made for differences in smoking habits.

In a study of workers at four diesel bus garages in two cities, Gamble *et al.* (1987b) investigated relationships between tenure (as a surrogate for cumulative exposure) and respiratory symptoms, chest radiographs, and pulmonary function. The study population was also compared to an unexposed control group of workers with similar socioeconomic background. After indirect adjustment for age, race, and smoking, the exposed workers showed an increased prevalence of cough, phlegm, and wheezing, but no association was found with tenure. Age- and height-adjusted pulmonary function was found to decline with duration of exposure, but was elevated on average, as compared to the control group. The number of positive radiographs was too small to support any conclusions. The authors concluded that the exposed workers may have experienced some chronic respiratory effects.

Purdham *et al.* (1987) compared baseline pulmonary function and respiratory symptoms in 17 exposed stevedores to a control group of 11 port office workers. After adjustment for smoking, there was no statistically significant difference in self-reported respiratory symptoms between the two groups. However, after adjustment for smoking, age, and height, exposed workers showed lower baseline pulmonary function, consistent with an obstructive ventilatory defect, as compared to both the control group and the general metropolitan population.

In a recent review of these studies, Cohen and Higgins (1995) concluded that they did not provide strong or consistent evidence for chronic, nonmalignant respiratory effects associated with occupational exposure to diesel exhaust. These reviewers stated, however, that ''several studies are suggestive of such effects * * * particularly when viewed in the context of possible biases in study design and analysis.'' MSHA agrees that the studies are inconclusive but suggestive of possible effects.

III.2.c.i.B. Cancer

Because diesel exhaust has long been known to contain carcinogenic compounds (e.g., benzene in the gaseous fraction and benzopyrene and nitropyrene in the dpm fraction), a great deal of research has been conducted to determine if occupational exposure to diesel exhaust actually results in an increased risk of cancer. Evidence that exposure to dpm increases the risk of developing cancer comes from three kinds of studies: human studies, genotoxicity studies, and animal studies. MSHA places the most weight on evidence from the human epidemiological studies and views the genotoxicological and animal studies as lending support to the epidemiological evidence.

In the epidemiological studies, it is generally impossible to disassociate exposure to dpm from exposure to the gasses and vapors that form the remainder of whole diesel exhaust. However, the animal evidence shows no significant increase in the risk of lung cancer from exposure to the gaseous fraction alone (Heinrich *et al.*, 1986; Iwai *et al.*, 1986; Brightwell *et al.*, 1986). Therefore, dpm, rather than the gaseous fraction of diesel exhaust, is assumed be the agent associated with an excess risk of lung cancer.

III.2.c.i.B.i. Lung Cancer

Beginning in 1957, at least 43 epidemiological studies have been published examining relationships between diesel exhaust exposure and the prevalence of lung cancer. The most recent published reviews of these studies are by Mauderly (1992), Cohen and Higgins (1995), Stöber and Abel (1996), Morgan et al. (1997), and Dawson et al. (1998). In addition, in response to the ANPRM, several commenters provided MSHA with their own reviews. Two comprehensive statistical ''meta-analyses'' of the epidemiological literature are also available: Lipsett and Alexeeff (1998) and Bhatia et al. (1998). These meta-analyses, which analyze and combine results from the various epidemiological studies, both suggest a statistically significant increase of 30 to 40 percent in the risk of lung cancer, attributable to occupational dpm exposure. The studies themselves, along with MSHA's comments on each study, are summarized in Tables III–4 (24 cohort studies) and III–5 (19 case-control studies).[11] Presence or absence of an adjustment for smoking habits is highlighted, and adjustments for other potentially confounding factors are indicated when applicable.

Some degree of association between occupational dpm exposure and an excess risk of lung cancer was observed in 38 of the 43 studies reviewed by MSHA: 18 of the 19 case-control studies and 20 of the 24 cohort studies. However, the 38 studies reporting a positive association vary considerably in the strength of evidence they present. As shown in Tables III–4 and III–5, *statistically significant* results were reported in 24 of the 43 studies: 10 of the 18 positive case-control studies and 14 of the 20 positive cohort studies.[12] In six of the 20 cohort studies and nine of the 18 case-control studies showing a positive association, the association observed was not statistically significant.

Because workers tend to be healthier than non-workers, the incidence of disease found among workers exposed to a toxic substance may be lower than the rate prevailing in the general population, but higher than the rate occurring in an unexposed population of workers. This phenomenon, called the ''healthy worker effect,'' also applies when the rate observed among exposed workers is greater than that found in the general population. In this case, assuming a study is unbiased with respect to other factors such as smoking, comparison with the general population will tend to *underestimate* the excess risk of disease attributable to the substance being investigated. Several studies drew comparisons against the general population, including both workers and nonworkers, with no compensating adjustment for the healthy worker effect. Therefore, in these studies, the excess risk of lung cancer attributable to dpm exposure is likely to have been underestimated, thereby making it more difficult to obtain a statistically significant result.

Five of the 43 studies listed in Tables III–4 and III–5 are negative—i.e., a lower rate of lung cancer was found among exposed workers than in the control population used for comparison. None of these five results, however, were statistically significant. Four of the five were cohort studies that drew comparisons against the general population and did not take the healthy worker effect into account. The remaining negative study was a case-control study in which vehicle drivers and locomotive engineers were compared to clerical workers.

Two cohort studies (Waxweiler et al., 1973; Ahlman et al., 1991) were performed specifically on groups of miners, and one (Boffetta et al., 1988) addressed miners as a subgroup of a larger population. Although an elevated prevalence of lung cancer was found among miners in both the 1973 and 1991 studies, the results were not statistically significant. The 1988 study found, after adjusting for smoking patterns and other occupational exposures, an 18-percent increase in the lung cancer rate among all workers occupationally exposed to diesel exhaust and a 167-percent increase

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[11] For simplicity, the epidemiological studies considered here are placed into two broad categories. A *cohort study* compares the health of persons having different exposures, diets, etc. A *case-control study* starts with two defined groups that differ in terms of their health and compares their exposure characteristics.

[12] A statistically significant result is a result unlikely to have arisen by chance in the group, or statistical *sample*, of persons being studied. An association arising by chance would have no predictive value for workers outside the sample. Failure to achieve statistical significance in an individual study can arise because of inherent limitations in the study, such as a small number of subjects in the sample or a short period of observation. Therefore, the lack of statistical significance in an individual study does not demonstrate that the results of that study were due merely to chance—only that the study (viewed in isolation) is inconclusive.

among miners (relative risk = 2.67). The latter result is statistically significant.

In addition, four case-control studies, all of which adjusted for smoking, found elevated rates of lung cancer associated with mining. The results for miners in three of these studies (Benhamou et al., 1988; Morabia et al., 1992; Siemiatycki et al., 1988) are given little weight because of potential confounding by occupational exposures to other carcinogens. The other study (Lerchen et al., 1987) showed a marginally significant result for underground non-uranium miners, but this was based on very few cases and the extent of diesel exposure among these miners was not reported. Although they do not pertain specifically to mining environments, other studies showing statistically significant results (most notably those by Garshick et al., 1987 and 1988) are based on far more data, contain better diesel exposure information, and are less susceptible to confounding by extraneous risk factors.

Since none of the existing human studies is perfect and many contain major deficiencies, it is not surprising that reported results differ in magnitude and statistical significance. Shortcomings identified in both positive and negative studies include: possible misclassification with respect to exposure; incomplete or questionable characterization of the exposed population; unknown or uncertain quantification of diesel exhaust exposure; incomplete, uncertain, or unavailable history of exposure to tobacco smoke and other carcinogens; and insufficient sample size, dpm exposure, or latency period (i.e., time since exposure) to detect a carcinogenic effect if one exists. Indeed, in their review of these studies, Stöber and Abel (1996) conclude that ''In this field * * * epidemiology faces its limits (Taubes, 1995) * * * Many of these studies were doomed to failure from the very beginning.''

Such problems, however, are not unique to epidemiological studies involving diesel exhaust but are common sources of uncertainty in virtually all epidemiological research involving cancer. Indeed, deficiencies such as exposure misclassification, small sample size, and short latency make it difficult to detect a relationship even when one exists. Therefore, the fact that 38 out of 43 studies showed any excess risk of lung cancer associated with dpm exposure may itself be a significant result, even if the evidence in most of those 38 studies is relatively weak.[13] The sheer number of studies showing such an association readily distinguishes this body of evidence from those criticized by Taubes (1995), where weak evidence is available from only a single study.

At the same time, MSHA recognizes that simply tabulating outcomes can sometimes be misleading, since there are generally a variety of outcomes that could render a study positive or negative and some studies use related data sets. Therefore, rather than limiting its assessment to such a tabulation, MSHA is basing its evaluation with respect to lung cancer largely on the two comprehensive meta-analyses (Lipsett and Alexeeff, 1998; Bhatia et al., 1998) described later, in the ''material impairments'' section of this risk assessment. In addition to restricting themselves to independent studies meeting certain minimal requirements, both meta-analyses investigated and rejected publication bias as an explanation for the generally positive results reported.

All of the studies showing negative or statistically insignificant positive associations were either based on relatively short observation or follow-up periods, lacked good information about dpm exposure, involved low duration or intensity of dpm exposure, or, because of inadequate sample size, lacked the statistical power to detect effects of the magnitude found in the ''positive'' studies. As stated by Boffetta et al. (1988, p. 404), studies failing to show a statistically significant association—

> * * * often had low power to detect any association, had insufficient latency periods, or compared incidence or mortality rates among workers to national rates only, resulting in possible biases caused by the ''healthy worker effect.''

Some respondents to the ANPRM argued that such methodological weaknesses may explain why not all of the studies showed a statistically significant association between dpm exposure and an increased prevalence of lung cancer. According to these commenters, if an epidemiological study shows a statistically significant result, this often occurs *in spite of* methodological weaknesses rather than because of them. Limitations such as potential exposure misclassification, inadequate latency, inadequate sample size, and insufficient duration of exposure all make it more difficult to obtain a statistically significant result when a real relationship exists.

On the other hand, Stöber and Abel (1996) argue, along with Morgan et al. (1997) and some commenters, that even in those epidemiological studies showing a statistically significant association, the magnitude of relative or excess risk observed is too small to demonstrate any causal link between dpm exposure and cancer. Their reasoning is that in these studies, errors in the collection or interpretation of smoking data can create a bias in the results larger than any potential contribution attributable to diesel particulate. They propose that studies failing to account for smoking habits should be disqualified from consideration, and that evidence of an association from the remaining studies should be discounted because of potential confounding due to erroneous, incomplete, or otherwise inadequate characterization of smoking histories.

MSHA concurs with Cohen and Higgins (1995), Lipsett and Alexeeff (1998), and Bhatia et al. (1998) in not accepting this view. MSHA does recognize that unknown exposures to tobacco smoke or other human carcinogens, such as asbestos, can distort the results of some lung cancer studies. MSHA also agrees that significant differences in the distribution of confounding factors, such as smoking history, between study and control groups can lead to misleading results. MSHA also recognizes, however, that it is not possible to design a human epidemiological study that perfectly controls for all potentially confounding factors. Some degree of informed subjective judgement is always required in evaluating the potential significance of unknown or uncontrolled factors.

Sixteen of the published epidemiological studies involving lung cancer did, in fact, control or adjust for exposure to tobacco smoke, and some of these also controlled or adjusted for exposure to asbestos and other carcinogenic substances (e.g., Garshick *et al.*, 1987; Steenland *et al.*, 1990; Boffetta *et al.*, 1988). All but one of these 16 epidemiological studies reported some degree of excess risk associated with exposure to diesel particulate, with statistically significant results reported in seven. These results are less likely to be confounded than results from studies with no adjustment. In addition, several of the other studies drew comparisons against internal control groups or control groups likely

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[13] The high proportion of positive studies is statistically significant according to the 2-tailed sign test, which rejects, at a high confidence level, the null hypothesis that each study is equally likely to be positive or negative. Assuming that the studies are independent, and that there is no systematic bias in one direction or the other, the probability of 38 or more out of 43 studies being either positive or negative is less than one per million under the null hypothesis.

to have similar smoking habits as the exposed groups (e.g., Garshick *et al.*, 1988; Gustavsson *et al.*, 1990; and Hansen, 1993). MSHA places more weight on these studies than on studies drawing comparisons against dissimilar groups with no controls or adjustments.

According to Stöber and Abel, the potential confounding effects of smoking are so strong that they could explain even statistically significant results observed in studies where smoking was explicitly taken into account. MSHA agrees that variable exposures to non-diesel lung carcinogens, including relatively small errors in smoking classification, could bias individual studies. However, the potential confounding effect of tobacco smoke and other carcinogens can cut in either direction. Spurious positive associations of dpm exposure with lung cancer would arise only if the group exposed to dpm had a greater exposure to these confounders than the unexposed control group used for comparison. If, on the contrary, the control group happened to be more exposed to confounders, then this would tend to make the association between dpm exposure and lung cancer appear negative. Therefore, although smoking effects could potentially distort the results of any single study, this effect could reasonably be expected to make only about half the studies that were explicitly adjusted for smoking come out positive. Smoking is unlikely to have been responsible for finding an excess prevalence of lung cancer in 15 out of 16 studies in which a smoking adjustment was applied. Based on a 2-tailed sign test, this possibility can be rejected at a confidence level greater than 99.9 percent.

Even in the 27 studies involving lung cancer for which no smoking adjustment was made, tobacco smoke and other carcinogens are important confounders only to the extent that the populations exposed and unexposed to diesel exhaust differed systematically with respect to these other exposures. Twenty-three of these studies, however, reported some degree of excess lung cancer risk associated with diesel exposure. This result could be attributed to non-diesel exposures only in the unlikely event that, in nearly all of these studies, diesel-exposed workers happened to be more highly exposed to these other carcinogens than the control groups of workers unexposed to diesel. All five studies not showing any association (Kaplan, 1959; DeCoufle, 1977; Waller, 1981; Edling, 1987; and Bender, 1989) may have failed to detect such a relationship because of too small a study group, lack of accurate exposure information, low duration or intensity of exposure, and/or insufficient latency or follow-up time.

It is also significant that the two most comprehensive, complete, and well-controlled studies available (Garshick *et al.*, 1987 and 1988) both point in the direction of an association between dpm exposure and an excess risk of lung cancer. These studies took care to address potential confounding by tobacco smoke and asbestos exposures. In response to the ANPRM, a consultant to the National Coal Association who was critical of all other available studies acknowledged that these two:

> * * * have successfully controlled for severally [sic] potentially important confounding factors * * * Smoking represents so strong a potential confounding variable that its control must be nearly perfect if an observed association between cancer and diesel exhaust is * * * [inferred to be causal]. In this regard, two observations are relevant. First, both case-control [Garshick *et al.*, 1987] and cohort [Garshick *et al.*, 1988] study designs revealed consistent results. Second, an examination of smoking related causes of death other than lung cancer seemed to account for only a fraction of the association observed between diesel exposure and lung cancer. A high degree of success was apparently achieved in controlling for smoking as a potentially confounding variable. [Submission 87–0–10, Robert A. Michaels, RAM TRAC Corporation, prepared for National Coal Association].

Potential biases due to extraneous risk factors are unlikely to account for a significant part of the excess risk in all studies showing an association. Excess rates of lung cancer were associated with dpm exposure in all epidemiologic studies of sufficient size and scope to detect such an excess. Although it is possible, in any individual study, that the potentially confounding effects of differential exposure to tobacco smoke or other carcinogens could account for the observed elevation in risk otherwise attributable to diesel exposure, it is unlikely that such effects would give rise to positive associations in 38 out of 43 studies. As stated by Cohen and Higgins (1995):

> * * * elevations [of lung cancer] do not appear to be fully explicable by confounding due to cigarette smoking or other sources of bias. Therefore, at present, exposure to diesel exhaust provides the most reasonable explanation for these elevations. The association is most apparent in studies of occupational cohorts, in which assessment of exposure is better and more detailed analyses have been performed. The largest relative risks are often seen in the categories of most probable, most intense, or longest duration of exposure. In general population studies, in which exposure prevalence is low and misclassification of exposure poses a particularly serious potential bias in the direction of observing no effect of exposure, most studies indicate increased risk, albeit with considerable imprecision. [Cohen and Higgins (1995), p. 269].

MSHA solicits comment on the issue of the potential for biases in these studies.

III.2.c.i.B.ii. Bladder Cancer

With respect to cancers other than lung cancer, MSHA's review of the literature identified only bladder cancer as a possible candidate for a causal link to dpm. Cohen and Higgins (1995) identified and reviewed 14 epidemiological case-control studies containing information related to dpm exposure and bladder cancer. All but one of these studies found elevated risks of bladder cancer among workers in jobs frequently associated with dpm exposure. Findings were statistically significant in at least four of the studies (statistical significance was not evaluated in three).

These studies point quite consistently toward an excess risk of bladder cancer among truck or bus drivers, railroad workers, and vehicle mechanics. However, the four available cohort studies do not support a conclusion that exposure to dpm is responsible for the excess risk of bladder cancer associated with these occupations. Furthermore, most of the case-control studies did not distinguish between exposure to diesel-powered equipment and exposure to gasoline-powered equipment for workers having the same occupation. When such a distinction was drawn, there was no evidence that the prevalence of bladder cancer was higher for workers exposed to the diesel-powered equipment.

This, along with the lack of corroboration from existing cohort studies, suggests that the excessive rates of bladder cancer observed may be a consequence of factors other than dpm exposure that are also associated with these occupations. For example, truck and bus drivers are subjected to vibrations while driving and may tend to have different dietary and sleeping habits than the general population. For these reasons, MSHA does not find that convincing evidence currently exists for a causal relationship between dpm exposure and bladder cancer.

III.2.c.ii. Studies Based on Exposures to Fine Particulate in Ambient Air

Longitudinal studies examine responses at given locations to changes in conditions over time, whereas *cross-sectional studies* compare results from locations with different conditions at a given point in time. Prior to 1990, cross sectional studies were generally used to evaluate the relationship between mortality and long-term exposure to particulate matter, but unaddressed spatial confounders and other methodological problems inherent in such studies limited their usefulness (EPA, 1996).

Two recent prospective cohort studies provide better evidence of a link between excess mortality rates and exposure to fine particulate, although the uncertainties here are greater than with the short-term exposure studies conducted in single communities. The two studies are known as the Six Cities study (Dockery *et al.,* 1993), and the American Cancer Society (ACS) study (Pope *et al.,* 1995).[14] The first study followed about 8,000 adults in six U.S. cities over 14 years; the second looked at survival data for half a million adults in 151 U.S. cities for 7 years. After adjusting for potential confounders, including smoking habits, the studies considered differences in mortality rates between the most polluted and least polluted cities.

Both the Six Cities Study and the ACS study found a significant association between increased concentration of $PM_{2.5}$ and total mortality.[15] The authors of the Six Cities Study concluded that the results suggest that exposures to fine particulate air pollution ''contributes to excess mortality in certain U.S. cities.'' The ACS study, which not only controlled for smoking habits and various occupational exposures, but also, to some extent, for passive exposure to tobacco smoke, found results qualitatively consistent with those of the Six Cities Study.[16] In the ACS study, however, the estimated increase in mortality associated with a given increase in fine particulate exposure was lower, though still statistically significant. In both studies, the largest increase observed was for cardiopulmonary mortality. Both studies also showed an increased risk of lung cancer associated with increased exposure to fine particulate, but these results were not statistically significant.

The few studies on associations between chronic $PM_{2.5}$ exposure and morbidity in adults show effects that are difficult to separate from measures of PM_{10} and measures of acid aerosols. The available studies, however, do show positive associations between particulate air pollution and adverse health effects for those with pre-existing respiratory or cardiovascular disease; and as mentioned earlier, there is a large body of evidence showing that respiratory diseases classified as COPD are significantly more prevalent among miners than in the general population. It also appears that PM exposure may exacerbate existing respiratory infections and asthma, increasing the risk of severe outcomes in individuals who have such conditions (EPA, 1996).

III.2.d. Mechanisms of Toxicity

As described in Part II, the particulate fraction of diesel exhaust is made up of aggregated soot particles. Each soot particle consists of an insoluble, elemental carbon core and an adsorbed, surface coating of relatively soluble organic compounds, such as polycyclic aromatic hydrocarbons (PAH's). When released into an atmosphere, the soot particles formed during combustion tend to aggregate into larger particles.

The literature on deposition of fine particles in the respiratory tract is reviewed in Green and Watson (1995) and U.S. EPA (1996). The mechanisms responsible for the broad range of potential particle-related health effects will vary depending on the site of deposition. Once deposited, the particles may be cleared from the lung, translocated into the interstitium, sequestered in the lymph nodes, metabolized, or be otherwise transformed by various mechanisms.

As suggested by Figure II–1 of this preamble, most of the aggregated particles making up dpm never get any larger than one micrometer in diameter. Particles this small are able to penetrate into the deepest regions of the lungs, called *alveoli.* In the alveoli, the particles can mix with and be dispersed by a substance called *surfactant,* which is secreted by cells lining the alveolar surfaces.

MSHA would welcome any additional information, not already covered cited above, on fine particle deposition in the respiratory tract, especially as it might pertain to lung loading in miners exposed to a combination of diesel particulate and other dusts. Any such additional information will be placed into the public record and considered by MSHA before a final rule is adopted.

III.2.d.i. Effects Other than Cancer

A number of controlled animal studies have been undertaken to ascertain the toxic effects of exposure to diesel exhaust and its components. Watson and Green (1995) reviewed approximately 50 reports describing noncancerous effects in animals resulting from the inhalation of diesel exhaust. While most of the studies were conducted with rats or hamsters, some information was also available from studies conducted using cats, guinea pigs, and monkeys. The authors also correlated reported effects with different descriptors of dose. From their review of these studies, Watson and Green concluded that:

> (a) Animals exposed to diesel exhaust exhibit a number of noncancerous pulmonary effects, including chronic inflammation, epithelial cell hyperplasia, metaplasia, alterations in connective tissue, pulmonary fibrosis, and compromised pulmonary function.
>
> (b) Cumulative weekly exposure to diesel exhaust of 70 to 80 $mg \bullet hr/m^3$ or greater are associated with the presence of chronic inflammation, epithelial cell proliferation, and depressed alveolar clearance in chronically exposed rats.
>
> (c) The extrapolation of responses in animals to noncancer endpoints in humans is uncertain. Rats were the most sensitive animal species studied.

Subsequent to the review by Watson and Green, there have been a number of animal studies on allergic immune responses to dpm. Takano et al. (1997) investigated the effects of dpm injected into mice through an intratracheal tube and found manifestations of allergic asthma, including enhanced antigen-induced airway inflammation, increased local expression of cytokine proteins, and increased production of antigen-specific immunoglobulins. The authors concluded that the study demonstrated dpm's enhancing effects on allergic asthma and that the results suggest that dpm is ''implicated in the increasing prevalence of allergic asthma in recent years.'' Similarly, Ichinose et al. (1997) found that five different strains of mice injected intratracheally with dpm exhibited manifestations of allergic asthma, as expressed by enhanced airway inflammation, which were correlated with an increased production of antigen-specific immunoglobulin due to the dpm. The authors concluded that dpm enhances manifestations of allergic airway inflammation and that ''* * * the cause of individual differences in humans at the onset of allergic asthma may be related to differences in antigen-induced immune responses * * *.''

Several laboratory animal studies have been performed to ascertain

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[14] A third such study only looked at TSP, rather than fine particulate. It did not find a significant association between total mortality and TSP. It is known as the California Seventh Day Adventist study (Abbey et al., 1991).

[15] The Six Cities study also found such relationships at elevated levels of $PM_{15/10}$ and sulfates. The ACS study was designed to follow up on the fine particle result of the Six Cities Study, but also looked at sulfates.

[16] The Six Cities study did not find a statistically significant increase in risk among non-smokers, suggesting that this group might not be as sensitive to adverse health effects from exposure to fine particulate; however, the ACS study, with more statistical power, did find an association even for non-smokers.

whether the effects of diesel exhaust are attributable specifically to the particulate fraction. (Heinrich *et al.*, 1986; Iwai *et al.*, 1986; Brightwell et al., 1986). These studies compare the effects of chronic exposure to whole diesel exhaust with the effects of filtered exhaust containing no particles.

The studies demonstrate that when the exhaust is sufficiently diluted to nullify the effects of gaseous irritants (NO_2 and SO_2), irritant vapors (aldehydes), CO, and other systemic toxicants, diesel particles are the prime etiologic agents of noncancer health effects. Exposure to dpm produced changes in the lung that were much more prominent than those evoked by the gaseous fraction alone. Marked differences in the effects of whole and filtered diesel exhaust were also evident from general toxicological indices, such as body weight, lung weight, and pulmonary histopathology. This provides strong evidence that the toxic component in diesel emissions producing the effects noted in other animal studies is due to the particulate fraction.

The mechanisms that may lead to adverse health effects in humans from inhaling fine particulates are not fully understood, but potential mechanisms that have been hypothesized for non-cancerous outcomes are summarized in Table III–6. A comprehensive review of the toxicity literature is provided in U.S. EPA (1996).

Deposition of particulates in the human respiratory tract could initiate events leading to increased airflow obstruction, impaired clearance, impaired host defenses, or increased epithelial permeability. Airflow obstruction could result from laryngeal constriction or bronchoconstriction secondary to stimulation of receptors in extrathoracic or intrathoracic airways. In addition to reflex airway narrowing, reflex or local stimulation of mucus secretion could lead to mucus hypersecretion and could eventually lead to mucus plugging in small airways.

Pulmonary changes that contribute to cardiovascular responses include a variety of mechanisms that can lead to hypoxemia, including bronchoconstriction, apnea, impaired diffusion, and production of inflammatory mediators. Hypoxia can lead to cardiac arrhythmias and other cardiac electrophysiologic responses that, in turn, may lead to ventricular fibrillation and ultimately cardiac arrest. Furthermore, many respiratory receptors have direct cardiovascular effects. For example, stimulation of C-fibers leads to bradycardia and hypertension, and stimulation of laryngeal receptors can result in hypertension, cardiac arrhythmia, bradycardia, apnea, and even cardiac arrest. Nasal receptor or pulmonary J-receptor stimulation can lead to vagally mediated bradycardia and hypertension (Widdicombe, 1988).

In addition to possible acute toxicity of particles in the respiratory tract, chronic exposure to particles that deposit in the lung may induce inflammation. Inflammatory responses can lead to increased permeability and possibly diffusion abnormality. Furthermore, mediators released during an inflammatory response could cause release of factors in the clotting cascade that may lead to an increased risk of thrombus formation in the vascular system (Seaton, 1995). Persistent inflammation, or repeated cycles of acute lung injury and healing, can induce chronic lung injury. Retention of the particles may be associated with the initiation and/or progression of COPD.

III.2.d.ii. Lung Cancer

III.2.d.ii.A. Genotoxicological Evidence

Many studies have shown that diesel soot, or its organic component, can increase the likelihood of genetic mutations during the biological process of cell division and replication. A survey of the applicable scientific literature is provided in Shirnamé-Moré (1995). What makes this body of research relevant to the risk of cancer is that mutations in critical genes can sometimes initiate, promote, or advance a process of carcinogenesis.

The determination of genotoxicity has frequently been made by treating diesel soot with organic solvents such as dichloromethane and dimethyl sulfoxide. The solvent removes the organic compounds from the carbon core. After the solvent evaporates, the mutagenic potential of the extracted organic material is tested by applying it to bacterial, mammalian, or human cells propagated in a laboratory culture. In general, the results of these studies have shown that various components of the organic material can induce mutations and chromosomal aberrations.

A critical issue is whether whole diesel particulate is mutagenic when dispersed by substances present in the lung. Since the laboratory procedure for extracting organic material with solvents bears little resemblance to the physiological environment of the lung, it is important to establish whether dpm as a whole is genotoxic, without solvent extraction. Early research indicated that this was not the case and, therefore, that the active genotoxic materials adhering to the carbon core of diesel particles might not be biologically damaging or even available to cells in the lung (Brooks et al., 1980; King et al., 1981; Siak et al., 1981). A number of more recent research papers, however, have shown that dpm, without solvent extraction, can cause DNA damage when the soot is dispersed in the pulmonary surfactant that coats the surface of the alveoli (Wallace et al., 1987; Keane et al., 1991; Gu et al., 1991; Gu et al., 1992). From these studies, NIOSH has concluded:

> * * * the solvent extract of diesel soot and the surfactant dispersion of diesel soot particles were found to be active in procaryotic cell and eukaryotic cell *in vitro* genotoxicity assays. The cited data indicate that respired diesel soot particles on the surface of the lung alveoli and respiratory bronchioles can be dispersed in the surfactant-rich aqueous phase lining the surfaces, and that genotoxic material associated with such dispersed soot particles is biologically available and genotoxically active. Therefore, this research demonstrates the biological availability of active genotoxic materials without organic solvent interaction. [Cover letter to NIOSH response to ANPRM].

From this conclusion, it follows that dpm itself, and not only its organic extract, can cause genetic mutations when dispersed by a substance present in the lung.

The biological availability of the genotoxic components is also supported directly by studies showing genotoxic effects of exposure to whole dpm. The formation of DNA adducts is an important indicator of genotoxicity and potential carcinogenicity. If DNA adducts are not repaired, then a mutation or chromosomal aberration can occur during normal mitosis (i.e., cell replication). Hemminki et al. (1994) found that DNA adducts were significantly elevated in nonsmoking bus maintenance and truck terminal workers, as compared to a control group of hospital mechanics, with the highest adduct levels found among garage and forklift workers. Similarly, Nielsen et al. (1996) found that DNA adducts were significantly increased in bus garage workers and mechanics exposed to dpm as compared to a control group.

III.2.d.ii.B. Evidence From Animal Studies

Bond et al. (1990) investigated differences in peripheral lung DNA adduct formation among rats, hamsters, mice, and monkeys exposed to dpm at a concentration of 8100 λg/m^{3} for 12 weeks. Mice and hamsters showed no increase of DNA adducts in their peripheral lung tissue, whereas rats and monkeys showed a 60 to 80% increase. The increased prevalence of lung DNA adducts in monkeys suggests that, with respect to DNA adduct formation, the human lungs' response to dpm inhalation may more closely resemble that of the rat than that of the hamster or mouse.

Mauderly (1992) and Busby and Newberne (1995) provide reviews of the scientific literature relating to excess lung cancers observed among laboratory animals chronically exposed to filtered and unfiltered diesel exhaust. The experimental data demonstrate that chronic exposure to whole diesel exhaust increases the risk of lung cancer in rats and that dpm is the causative agent. This carcinogenic effect has been confirmed in two strains of rats and in at least five laboratories. Experimental results for animal species other than the rat, however, are either inconclusive or, in the case of Syrian hamsters, suggestive of no carcinogenic effect. This is consistent with the observation, mentioned above, that lung DNA adduct formation is increased among exposed rats but not among exposed hamsters or mice.

The conflicting results for rats and hamsters indicate that the carcinogenic effects of dpm exposure may be species-dependent. Indeed, monkey lungs have been reported to respond quite differently than rat lungs to both diesel exhaust and coal dust (Nikula, 1997). Therefore, the results from rat experiments do not, by themselves, establish that there is any excess risk due to dpm exposure for humans. The human epidemiological data, however, indicate that humans comprise a species that, like rats and unlike hamsters, do suffer a carcinogenic response to dpm exposure. Therefore, MSHA considers the rat studies at least relevant to an evaluation of the risk for humans.

When dpm is inhaled, a number of adverse effects that may contribute to carcinogenesis are discernable by microscopic and biochemical analysis. For a comprehensive review of these effects, see Watson and Green (1995). In brief, these effects begin with phagocytosis, which is essentially an attack on the diesel particles by cells called alveolar macrophages. The macrophages engulf and ingest the diesel particles, subjecting them to detoxifying enzymes. Although this is a normal physiological response to the inhalation of foreign substances, the process can produce various chemical byproducts injurious to normal cells. In attacking the diesel particles, the activated macrophages release chemical agents that attract neutrophils (a type of white blood cell that destroys microorganisms) and additional alveolar macrophages. As the lung burden of diesel particles increases, aggregations of particle-laden macrophages form in alveoli adjacent to terminal bronchioles, the number of Type II cells lining particle-laden alveoli increases, and particles lodge within alveolar and peribronchial tissues and associated lymph nodes. The neutrophils and macrophages release mediators of inflammation and oxygen radicals, which have been implicated in causing various forms of chromosomal damage, genetic mutations, and malignant transformation of cells (Weitzman and Gordon, 1990). Eventually, the particle-laden macrophages are functionally altered, resulting in decreased viability and impaired phagocytosis and clearance of particles. This series of events may result in pulmonary inflammatory, fibrotic, or emphysematous lesions that can ultimately develop into cancerous tumors.

Such reactions have also been observed in rats exposed to high concentrations of fine particles with no organic component (Mauderly et al., 1994; Heinrich et al., 1994 and 1995; Nikula et al., 1995). Rats exposed to titanium dioxide or pure carbon (''carbon-black'') particles, which are not considered to be genotoxic, developed lung cancers at about the same rate as rats exposed to whole diesel exhaust. Therefore, it appears that the toxicity of dpm, at least in some species, may result largely from a biochemical response to the particle itself rather than from specific effects of the adsorbed organic compounds.

Some researchers have interpreted the carbon-black and titanium dioxide studies as also suggesting that (1) the carcinogenic mechanism in rats depends on massive overloading of the lung and (2) that this may provide a mechanism of carcinogenesis specific to rats which does not occur in other rodents or in humans (Oberdörster, 1994; Watson and Valberg, 1996). Some commenters on the ANPRM cited the lack of any link between lung cancer and coal dust or carbon black exposure as evidence that carbon particles, by themselves, are not carcinogenic in humans. Coal mine dust, however, consists almost entirely of particles larger than those forming the carbon core of dpm or used in the carbon-black and titanium dioxide rat studies. Furthermore, although there have been nine studies reporting no excess risk of lung cancer among coal miners (Liddell, 1973; Costello *et al.*, 1974; Armstrong *et al.*, 1979; Rooke *et al.*, 1979; Ames *et al.*, 1983; Atuhaire *et al.*, 1985; Miller and Jacobsen, 1985; Kuempel *et al.*, 1995; Christie *et al.*, 1995), five studies have reported an elevated risk of lung cancer for those exposed to coal dust (Enterline, 1972; Rockette, 1977; Correa *et al.*, 1984; Levin *et al.*, 1988; Morfeld *et al.*, 1997). The positive results in two of these studies (Enterline, 1972; Rockette, 1977) were statistically significant. Furthermore, excess lung cancers have been reported among carbon black production workers (Hodgson and Jones, 1985; Siemiatycki, 1991; Parent *et al.*, 1996). MSHA is not aware of any evidence that a mechanism of carcinogenesis due to fine particle overload is inapplicable to humans. Studies carried out on rodents certainly do not provide such evidence.

The carbon-black and titanium dioxide studies indicate that lung cancers in rats exposed to dpm may be induced by a mechanism that does not require the bioavailability of genotoxic organic compounds adsorbed on the elemental carbon particles. These studies do not, however, prove that the only significant agent of carcinogenesis in rats exposed to diesel particulate is the non-soluble carbon core. Nor do the carbon-black studies prove that the only significant mechanism of carcinogenesis due to diesel particulate is lung overload. Due to the relatively high doses administered in the rat studies, it is conceivable that an overload phenomenon masks or parallels other potential routes to cancer. It may be that effects of the genotoxic organic compounds are merely masked or displaced by overloading in the rat studies. Gallagher et al. (1994) exposed different groups of rats to diesel exhaust, carbon black, or titanium dioxide and detected species of lung DNA adducts in the rats exposed to dpm that were not found in the controls or rats exposed to carbon black or titanium dioxide.

Particle overload may provide the dominant route to lung cancer at very high concentrations of fine particulate, while genotoxic mechanisms may provide the primary route under lower-level exposure conditions. In humans exposed over a working lifetime to doses insufficient to cause overload, carcinogenic mechanisms unrelated to overload may dominate, as indicated by the human epidemiological studies and the data on human DNA adducts cited above. Therefore, the carbon black results observed in the rat studies do not preclude the possibility that the organic component of dpm has important genotoxic effects in humans (Nauss et al., 1995).

Even if the genotoxic organic compounds in dpm were biologically unavailable and played no role in human carcinogenesis, this would not rule out the possibility of a genotoxic route to lung cancer (even for rats) due to the presence of dpm particles themselves. For example, as a byproduct of the biochemical response to the presence of dpm in the alveoli, free oxidant radicals may be released as macrophages attempt to digest the particles. There is evidence that dpm can both induce production of active oxygen agents and also depress the activity of naturally occurring antioxidant enzymes (Mori, 1996; Sagai, 1993). Oxidants can induce carcinogenesis either by reacting directly with DNA, or by stimulating cell replication, or both (Weitzman and Gordon, 1990). This would provide a mutagenic route to lung cancer with no threshold. Therefore, the carbon black and titanium dioxide studies cited above do not prove that dpm exposure has no incremental, genotoxic effects or that there is a threshold below which dpm exposure poses no risk of causing lung cancer.

It is noteworthy, however, that dpm exposure levels recorded in some mines have been almost as high as laboratory exposures administered to rats showing a clearly positive response. Intermittent, occupational exposure levels greater than about 500 μg/m^3 dpm may overwhelm the human lung clearance mechanism (Nauss et al., 1 995). Therefore, concentrations at levels currently observed in some mines could be expected to cause overload in some humans, possibly inducing lung cancer by a mechanism similar to what occurs in rats. MSHA would like to receive additional scientific information on this issue, especially as it relates to lung loading in miners exposed to a combination of diesel particulate and other dusts.

As suggested above, such a mechanism would not necessarily be the only route to carcinogenesis in humans and, therefore, would not imply that dpm concentrations too low to cause overload are safe for humans. Furthermore, a proportion of exposed individuals can always be expected to be more susceptible than normal. Therefore, at lower dpm concentrations, particle overload may still provide a route to lung cancer in susceptible humans. At even lower concentrations, other routes to carcinogenesis in humans may predominate, possibly involving genotoxic effects.

III.3. Characterization of Risk.

Having reviewed the evidence of health effects associated with exposure to dpm, MSHA has evaluated that evidence to ascertain whether exposure levels currently existing in mines warrant regulatory action pursuant to the Mine Act. The criteria for this evaluation are established by the Mine Act and related court decisions. Section 101(a)(6)(A) provides that:

> The Secretary, in promulgating mandatory standards dealing with toxic materials or harmful physical agents under this subsection, shall set standards which most adequately assure on the basis of the best available evidence that no miner will suffer material impairment of health or functional capacity even if such miner has regular exposure to the hazards dealt with by such standard for the period of his working life.

Based on court interpretations of similar language under the Occupational Safety and Health Act, there are three questions that need to be addressed: (1) Whether health effects associated with dpm exposure constitute a ''material impairment'' to miner health or functional capacity; (2) whether exposed miners are at significant excess risk of incurring any of these material impairments; and (3) whether the proposed rule will substantially reduce such risks.

The criteria for evaluating the health effects evidence do not require scientific certainty. As noted by Justice Stevens in an important case on risk involving the Occupational Safety and Health Administration, the need to evaluate risk does not mean an agency is placed into a ''mathematical straightjacket.'' [*Industrial Union Department, AFL-CIO* v. *American Petroleum Institute*, 448 U.S. 607, 100 S.Ct. 2844 (1980), hereinafter designated the ''Benzene'' case]. When regulating on the edge of scientific knowledge, certainty may not be possible; and—

> so long as they are supported by a body of reputable scientific thought, the Agency is free to use conservative assumptions in interpreting the data * * * risking error on the side of overprotection rather than underprotection. [Id. at 656].

The statutory criteria for evaluating the health evidence do not require MSHA to wait for absolute precision. In fact, MSHA is required to use the ''best *available* evidence.'' (Emphasis added).

III.3.a. Material Impairments to Miner Health or Functional Capacity

From its review of the literature cited in Part III.2, MSHA has tentatively concluded that underground miners exposed to current levels of dpm are at excess risk of incurring the following three kinds of material impairment: (i) sensory irritations and respiratory symptoms; (ii) death from cardiovascular, cardiopulmonary, or respiratory causes; and (iii) lung cancer. The basis for linking these with dpm exposure is summarized in the following three subsections.

III.3.a.i. Sensory Irritations and Respiratory Symptoms

Kahn et al. (1988), Battigelli (1965), Gamble et al. (1987a) and Rudell et al. (1996) identified a number of debilitating acute responses to diesel exhaust exposure: irritation of the eyes, nose and throat; headaches, nausea, and vomiting; chest tightness and wheeze. These symptoms were also reported by miners at the 1995 workshops. In addition, Ulfvarson et al. (1987, 1990) found evidence of reduced lung function in workers exposed to dpm for a single shift.

Although there is evidence that such symptoms subside within one to three days of no occupational exposure, a miner who must be exposed to dpm day after day in order to earn a living may not have time to recover from such effects. Hence, the opportunity for a so-called ''reversible'' health effect to reverse itself may not be present for many miners. Furthermore, effects such as stinging, itching and burning of the eyes, tearing, wheezing, and other types of sensory irritation can cause severe discomfort and can, in some cases, be seriously disabling. Also, workers experiencing sufficiently severe sensory irritations can be distracted as a result of their symptoms, thereby endangering other workers and increasing the risk of accidents. For these reasons, MSHA considers such irritations to constitute ''material impairments'' of health or functional capacity within the meaning of the Act, regardless of whether or not they are reversible. Further discussion of why MSHA believes reversible effects can constitute material impairments can be found earlier in this risk assessment, in the section entitled ''Relevance of Health Effects that are Reversible.''

The best available evidence also points to more severe respiratory consequences of exposure to dpm. Significant associations have been detected between acute environmental exposures to fine particulates and debilitating respiratory impairments in adults, as measured by lost work days, hospital admissions, and emergency room visits. Short-term exposures to fine particulates, or particulate air pollution in general, have been associated with significant increases in the risk of hospitalization for both pneumonia and COPD (EPA, 1996).

The risk of severe respiratory effects is exemplified by specific cases of persistent asthma linked to diesel exposure (Wade and Newman, 1993). There is considerable evidence for a causal connection between dpm exposure and increased manifestations of allergic asthma and other allergic respiratory diseases, coming from recent experiments on animals and human cells (Peterson and Saxon, 1996; Diaz-Sanchez, 1997; Takano et al., 1997; Ichinose et al., 1997). Such health outcomes are clearly ''material impairments'' of health or functional capacity within the meaning of the Act.

III.3.a.ii. Excess Risk of Death from Cardiovascular, Cardiopulmonary, or Respiratory Causes

The evidence from air pollution studies identifies death, largely from cardiovascular or respiratory causes, as an endpoint significantly associated with acute exposures to fine particulates. The weight of epidemiological evidence indicates that short-term ambient exposure to particulate air pollution contributes to an increased risk of daily mortality. Time-series analyses strongly suggest a positive effect on daily mortality across the entire range of ambient particulate pollution levels. Relative risk estimates for daily mortality in relation to daily ambient particulate concentration are consistently positive and statistically significant across a variety of statistical modeling approaches and methods of adjustment for effects of relevant covariates such as season, weather, and co-pollutants. After thoroughly reviewing this body of evidence, the U.S. Environmental Protection Agency (EPA) concluded:

> It is extremely unlikely that study designs not yet employed, covariates not yet identified, or statistical techniques not yet developed could wholly negate the large and consistent body of epidemiological evidence * * *.

There is also substantial evidence of a relationship between chronic exposure to fine particulates and an excess (age-adjusted) risk of mortality, especially from cardiopulmonary diseases. The Six Cities and ACS studies of ambient air particulates both found a significant association between chronic exposure to fine particles and excess mortality. In both studies, after adjusting for smoking habits, a statistically significant excess risk of cardiopulmonary mortality was found in the city with the highest average concentration of fine particulate (i.e., $PM_{2.5}$) as compared to the city with the lowest. Both studies also found excess deaths due to lung cancer in the cities with the higher average level of $PM_{2.5}$, but these results were not statistically significant (EPA, 1996). The EPA concluded that—

> * * * the chronic exposure studies, taken together, suggest there may be increases in mortality in disease categories that are consistent with long-term exposure to airborne particles and that at least some fraction of these deaths reflect cumulative PM impacts above and beyond those exerted by acute exposure events * * * There tends to be an increasing correlation of long-term mortality with PM indicators as they become more reflective of fine particle levels (EPA, 1996).

Whether associated with acute or chronic exposures, the excess risk of death that has been linked to pollution of the air with fine particles like dpm is clearly a ''material impairment'' of health or functional capacity within the meaning of the Act.

III.3.a.iii. Lung Cancer

It is clear that lung cancer constitutes a ''material impairment'' of health or functional capacity within the meaning of the Act. Questions have been raised however, as to whether the evidence linking dpm exposure with an excess risk of lung cancer demonstrates a causal connection (Stöber and Abel, 1996; Watson and Valberg, 1996; Cox, 1997; Morgan et al., 1997; Silverman, 1998).

MSHA recognizes that no single one of the existing epidemiological studies, viewed in isolation, provides conclusive evidence of a causal connection between dpm exposure and an elevated risk of lung cancer in humans. Consistency and coherency of results, however, do provide such evidence. Although no epidemiological study is flawless, studies of both cohort and case-control design have quite consistently shown that chronic exposure to diesel exhaust, in a variety of occupational circumstances, is associated with an increased risk of lung cancer. With only rare exceptions, involving too few workers and/or observation periods too short to have a good chance of detecting excess cancer risk, the human studies have shown a greater risk of lung cancer among exposed workers than among comparable unexposed workers.

Lipsett and Alexeeff (1998) performed a comprehensive statistical meta-analysis of the epidemiological literature on lung cancer and dpm exposure. This analysis systematically combined the results of the studies summarized in Tables III–4 and III–5. Some studies were eliminated because they did not allow for a period of at least 10 years for the development of clinically detectable lung cancer. Others were eliminated because of bias resulting from incomplete ascertainment of lung cancer cases in cohort studies or because they examined the same cohort population as another study. One study was excluded because standard errors could not be calculated from the data presented. The remaining 30 studies were analyzed using both a fixed-effects and a random-effect analysis of variance (ANOVA) model. Sources of heterogeneity in results were investigated by subset analysis; using categorical variables to characterize each study's design; target population (general or industry-specific); occupational group; source of control or reference population; latency; duration of exposure; method of ascertaining occupation; location (North America or Europe); covariate adjustments (age, smoking, and/or asbestos exposure); and absence or presence of a clear healthy worker effect (as manifested by lower than expected all-cause mortality in the occupational population under study).

Sensitivity analyses were conducted to evaluate the sensitivity of results to inclusion criteria and to various assumptions used in the analysis. This included substitution of excluded ''redundant'' studies of same cohort population for the included studies and exclusion of studies involving questionable exposure to dpm. An influence analysis was also conducted to examine the effect of dropping one study at a time, to determine if any individual study had a disproportionate effect on the ANOVA. Potential effects of publication bias were also investigated. The authors concluded:

> The results of this meta-analysis indicate a consistent positive association between occupations involving diesel exhaust exposure and the development of lung cancer. Although substantial heterogeneity existed in the initial pooled analysis, stratification on several factors identified a relationship that persisted throughout various influence and sensitivity analyses* * *.
>
> This meta-analysis provides evidence consistent with the hypothesis that exposure to diesel exhaust is associated with an increased risk of lung cancer. The pooled estimates clearly reflect the existence of a positive relationship between diesel exhaust and lung cancer in a variety of diesel-exposed occupations, which is supported when the most important confounder, cigarette smoking, is measured and controlled. There is suggestive evidence of an exposure-response relationship in the smoking adjusted studies as well. Many of the subset analyses indicated the presence of substantial heterogeneity among the pooled estimates. Much of the heterogeneity observed, however, is due to the presence or absence of adjustment for smoking in the individual study risk estimates, to occupation-specific influences on exposure, to potential selection biases, and other aspects of study design.

A second, independent meta-analysis of epidemiological studies published in peer-reviewed journals was conducted by Bhatia et al. (1998).[17] In this analysis, studies were excluded if actual work with diesel equipment ''could not be confirmed or reliably inferred'' or if an inadequate latency period was allowed for cancer to develop, as indicated by less than 10 years from time of first exposure to end of follow-up. Studies of miners were also excluded, because of potential exposure to radon and silica. Likewise, studies were excluded if they exhibited selection bias or examined the same cohort population as a study published later. A total of 29 independent studies from 23 published sources were identified as meeting the inclusion criteria. After assigning each of these 29 studies a weight proportional to its estimated precision, pooled relative risks were calculated based on the following groups of studies: all 29 studies; all case-control studies; all cohort studies; cohort studies using internal reference populations; cohort studies making external comparisons; studies adjusted for smoking; studies not adjusted for smoking; and studies grouped by occupation (railroad workers, equipment operators, truck drivers, and bus workers). Elevated risks were shown for exposed workers overall and within every individual group of studies analyzed. A positive duration-response relationship was observed in those studies presenting results according to employment duration. The weighted, pooled estimates of relative risk were identical for case-control and cohort studies and nearly identical for studies with or without smoking adjustments. Based on their stratified analysis, the authors argued that—

> the heterogeneity in observed relative risk estimates may be explained by differences between studies in methods, in populations studied and comparison groups used, in latency intervals, in intensity and duration of exposure, and in the chemical and physical characteristics of diesel exhaust.

They concluded that the elevated risk of lung cancer observed among exposed workers was unlikely to be due to chance, that confounding from smoking is unlikely to explain all of the excess risk, and that ''this meta-analysis supports a causal association between increased risks for lung cancer and exposure to diesel exhaust.''

As discussed earlier in the section entitled ''Mechanisms of Toxicity,'' animal studies have confirmed that diesel exhaust can increase the risk of lung cancer in some species and shown that dpm (rather than the gaseous fraction of diesel exhaust) is the causal agent. MSHA, however, views results from animal studies as subordinate to the results obtained from human studies. Since the human studies show increased risk of lung cancer at dpm levels lower than what might be expected to cause overload, they provide evidence that overload may not be the only mechanism at work among humans. The fact that dpm has been proven to cause lung cancer in laboratory rats is of interest primarily in supporting the plausibility of a causal interpretation for relationships observed in the human studies.

Similarly, the genotoxicological evidence provides additional support for a causal interpretation of associations observed in the epidemiological studies. This evidence shows that dpm dispersed by alveolar surfactant can have mutagenic effects, thereby providing a genotoxic route to carcinogenesis independent of overloading the lung with particles. Chemical byproducts of phagocytosis may provide another genotoxic route. Inhalation of diesel emissions has been shown to cause DNA adduct formation in peripheral lung cells of rats and monkeys, and increased levels of human DNA adducts have been found in association with occupational exposures. Therefore, there is little basis for postulating that a threshold exists, demarcating overload, below which dpm would not be expected to induce lung cancers in humans.

Results from the epidemiological studies, the animal studies, and the genotoxicological studies are coherent and mutually reinforcing. After considering all these results, MSHA has concluded that the epidemiological studies, supported by the experimental data establishing the plausibility of a causal connection, provide strong evidence that chronic occupational dpm exposure increases the risk of lung cancer in humans.

III.3.b. Significance of the Risk of Material Impairment to Miners

The fact that there is substantial evidence that dpm exposure can materially impair miner health in several ways does not imply that miners will necessarily suffer such impairments at a significant rate. This section will consider the significance of the risk faced by miners exposed to dpm.

III.3.b.i. Definition of a Significant Risk

The benzene case, referred to earlier in this section, provides the starting point for MSHA's analysis of this issue. Soon after its enactment in 1970, OSHA adopted a ''consensus'' standard on exposure to benzene, as required and authorized by the OSH Act. The basic part of the standard was an average exposure limit of 10 parts per million over an 8-hour workday. The consensus standard had been established over time to deal with concerns about poisoning from this substance (448 U.S. 607, 617). Several years later, NIOSH recommended that OSHA alter the standard to take into account evidence suggesting that benzene was also a carcinogen. (*Id.* at 619 *et seq.*). Although the ''evidence in the administrative record of adverse effects of benzene exposure at 10 ppm is sketchy at best,'' OSHA was operating under a policy that there was no safe exposure level to a carcinogen. (*Id.*, at 631). Once the evidence was adequate to reach a conclusion that a substance was a carcinogen, the policy required the agency to set the limit at the lowest level feasible for the industry. (*Id.* at 613). Accordingly, the Agency proposed lowering the permissible exposure limit to 1 ppm.

The Supreme Court rejected this approach. Noting that the OSH Act requires ''safe or healthful employment,'' the court stated that—

> * * *'safe' is not the equivalent of 'risk-free'* * *a workplace can hardly be considered 'unsafe' unless it threatens the workers with a significant risk of harm. Therefore, before he can promulgate any permanent health or safety standard, the Secretary is required to make a threshold finding that a place of employment is unsafe—in the sense that significant risks are present and can be eliminated or lessened by a change in practices. [Id., at 642, italics in original].

The court went on to explain that it is the Agency that determines how to make such a threshold finding:

> First, the requirement that a 'significant' risk be identified is not a mathematical straitjacket. It is the Agency's responsibility to determine, in the first instance, what it considered to be a 'significant' risk. Some risks are plainly acceptable and others are plainly unacceptable. If, for example, the odds are one in a billion that a person will die from cancer by taking a drink of chlorinated water, the risk clearly could not be considered significant. On the other hand, if the odds are one in a thousand that regular inhalation of gasoline vapors that are 2% benzene will be fatal, a reasonable person might well consider the risk significant and take appropriate steps to decrease or eliminate it. Although the Agency has no duty to calculate the exact probability of

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[17] To address potential publication bias, the authors identified several unpublished studies on truck drivers and noted that elevated risks for exposed workers observed in these studies were similar to those in the published studies utilized. Based on this and a ''funnel plot'' for the included studies, the authors concluded that there was no indication of publication bias.

harm, it does have an obligation to find that a significant risk is present before it can characterize a place of employment as 'unsafe.' [*Id.*, at 655].

The court noted that the Agency's "*** determination that a particular level of risk is 'significant' will be based largely on policy considerations." (Id., note 62).

III.3.b.ii. *Evidence of Significant Risk at Current Exposure Levels.* In evaluating the significance of the risks to miners, a key factor is the very high concentrations of diesel particulate to which a number of those miners are currently exposed—compared to ambient atmospheric levels in even the most polluted urban environments, and to workers in diesel-related occupations for which positive epidemiological results have been observed. Figure III–4 compared the range of median dpm exposures measured for mine workers at various mines to the range of geometric means (i.e., estimated medians) reported for other occupations, as well as to ambient environmental levels. Figure III–5 presents a similar comparison, based on the highest mean dpm level observed at any individual mine, the highest mean level reported for any occupational group other than mining, and the highest monthly mean concentration of dpm estimated for ambient air at any site in the Los Angeles basin.[18] As shown in Figure III–5, underground miners are currently exposed at mean levels up to 10 times higher than the highest mean exposure reported for other occupations, and up to 100 times higher than comparable environmental levels of diesel particulate.

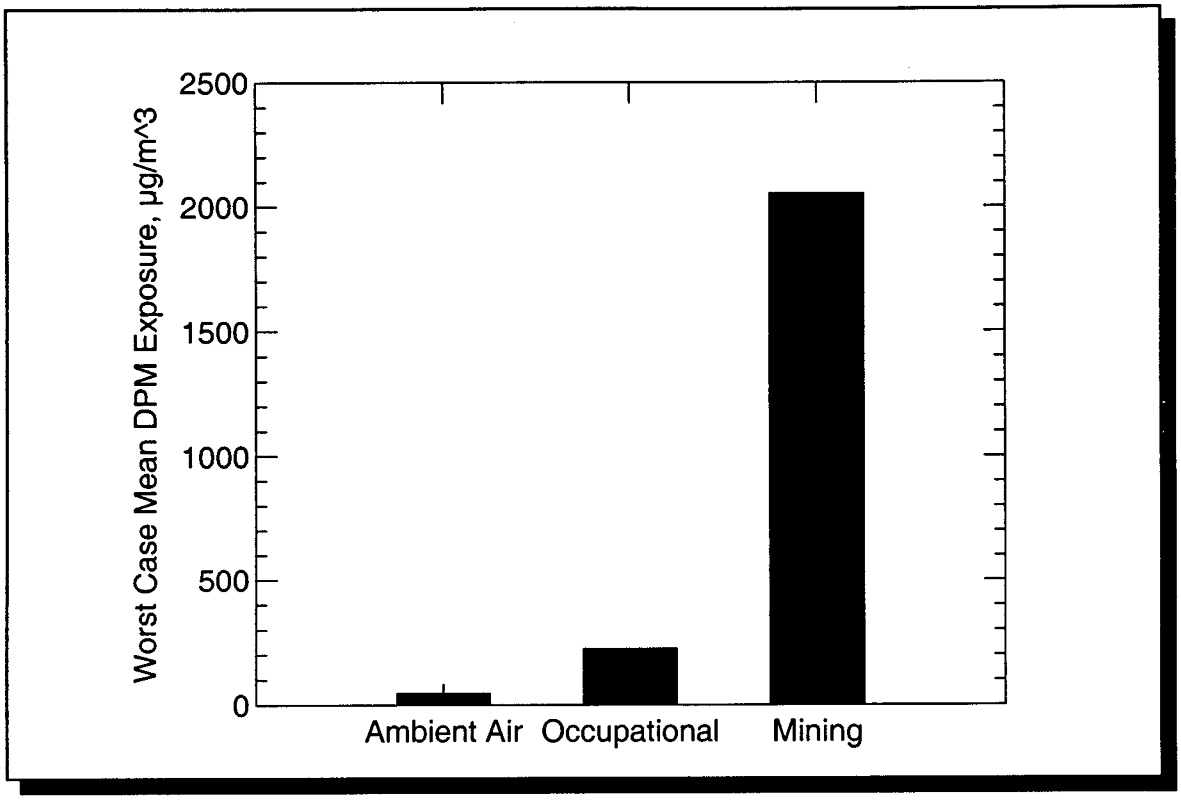

Figure III-5.--Worst case observed or reported mean diesel particulate exposure concentrations for urban ambient air, occupations other than mining, and mining. Worst case for mining is mean dpm measured within an underground mine. Worst case for occupations other than mining is mean respirable particulate matter, other than cigarette smoke, reported for railroad workers classified as hostlers (Woskie et al., 1988). Worst case for ambient air is mean estimated for peak months at most heavily polluted site in Los Angeles area (Cass and Gray, 1995), multiplied by 4.7 to adjust for comparability with occupational lifetime exposure levels. For additional information on means and ranges see section III.1.d.

Given the significantly increased mortality and other acute, adverse health effects associated with increments of 25 µg/m³ in fine particulate concentration (Table III–3), the relative risk for some miners, especially those already suffering respiratory problems, appears to be extremely high. Acute responses to dpm

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[18] For comparability with occupational lifetime exposure levels, the environmental ambient air concentration has been multiplied by a factor of approximately 4.7. This factor reflects a 45-year occupational lifetime with 240 working days per year, as opposed to a 70-year environmental lifetime with 365-days per year, and assumes that air inhaled during a work shift comprises half the total air inhaled during a 24-hour day.

exposures have been detected in studies of stevedores, whose exposure was likely to have been less than one tenth the exposure of some miners on the job.

Both existing meta-analyses of human studies relating dpm exposure and lung cancer suggest that, on average, occupational exposure is responsible for a 30 to 40-percent increase in lung cancer risk across all industries studied (Lipsett and Alexeeff, 1998; Bhatia et al., 1998). Moreover, the epidemiological studies providing the evidence of this increased risk involved average exposure levels estimated to be far below levels to which some underground miners are currently exposed. Specifically, the elevated risk of lung cancer observed in the two most extensively studied industries—trucking (including dock workers) and railroads—was associated with average exposure levels estimated to be far below levels observed in underground mines. The highest average concentration of dpm reported for dock workers—the most highly exposed occupational group within the trucking industry—is about 55 $\mu g/m^3$ total elemental carbon at an individual dock (NIOSH, 1990). This translates, on average, to no more than about 110 $\mu g/m^3$ of dpm. Published measurements of dpm for railworkers have generally been less than 140 $\mu g/m^3$ (measured as respirable particulate matter other than cigarette smoke). The reported mean of 224 $\mu g/m^3$ for hostlers displayed in Figure III–5 represents only the worst case occupational subgroup (Woskie et al., 1988). Indeed, although MSHA views extrapolations from animal studies as subordinate to results obtained from human studies, it is noteworthy that dpm exposure levels recorded in some underground mines (Figures III–1 and III–2) have been well within the exposure range that produced tumors in rats (Nauss et al., 1995).

The significance of the lung cancer risk to exposed underground miners is also supported by a recent NIOSH report (Stayner et al., 1998), which summarizes a number of published quantitative risk assessments. These assessments are broadly divided into those based on human studies and those based on animal studies. Depending on the particular studies, assumptions, and methods of assessment used, estimates of the exact degree of risk vary widely even within each broad category. MSHA recognizes that a conclusive assessment of the quantitative relationship between lung cancer risk and specific exposure levels is not possible at this time, given the limitations in currently available epidemiological data and questions about the applicability to humans of responses observed in rats. However, all of the very different approaches and methods published so far, as described in Stayner et al. 1998, have produced results indicating that levels of dpm exposure measured at some underground mines present an unacceptably high risk of lung cancer for miners—a risk significantly greater than the risk they would experience without the dpm exposure.

Quantitative risk estimates based on the human studies were generally higher than those based on analyses of the rat inhalation studies. As indicated by Tables 3 and 4 of Stayner et al. 1998, a working lifetime of exposure to dpm at 500 $\mu g/m^3$ yields estimates of excess lung cancer risk ranging from about 1 to 200 excess cases of lung cancer per thousand workers based on the rat inhalation studies and from about 50 to 800 per 1000 based on the epidemiological assessments. Even the lowest of these estimates indicates a risk that is clearly significant under the quantitative rule of thumb established in the benzene case. [*Industrial Union* v. *American Petroleum*; 448 U.S. 607, 100 S.Ct. 2844 (1980)].

Stayner et al. 1998 concluded their report by stating:

> The risk estimates derived from these different models vary by approximately three orders of magnitude, and there are substantial uncertainties surrounding each of these approaches. Nonetheless, the results from applying these methods are consistent in predicting relatively large risks of lung cancer for miners who have long-term exposures to high concentrations of DEP [i.e., dpm]. This is not surprising given the fact that miners may be exposed to DEP [dpm] concentrations that are similar to those that induced lung cancer in rats and mice, and substantially higher than the exposure concentrations in the positive epidemiologic studies of other worker populations.

The Agency is also aware that a number of other governmental and nongovernmental bodies have concluded that the risks of dpm are of sufficient significance that exposure should be limited:

(1) In 1988, after a thorough review of the literature, the National Institute for Occupational Safety and Health (NIOSH) recommended that whole diesel exhaust be regarded as a potential occupational carcinogen and controlled to the lowest feasible exposure level. The document did not contain a recommended exposure limit.

(2) In 1995, the American Conference of Governmental Industrial Hygienists placed on the Notice of Intended Changes in their Threshold Limit Values (TLV's) for Chemical Substances and Physical Agents and Biological Exposure Indices Handbook a recommended TLV of 150 $\mu g/m^3$ for exposure to whole diesel particulate.

(3) The Federal Republic of Germany has determined that diesel exhaust has proven to be carcinogenic in animals and classified it as an A2 in their carcinogenic classification scheme. An A2 classification is assigned to those substances shown to be clearly carcinogenic only in animals but under conditions indicative of carcinogenic potential at the workplace. Based on that classification, technical exposure limits for dpm have been established, as described in part II of this preamble. These are the minimum limits thought to be feasible in Germany with current technology and serve as a guide for providing protective measures at the workplace.

(4) The Canada Centre for Mineral and Energy Technology (CANMET) currently has an interim recommendation of 1000 $\mu g/m^3$ respirable combustible dust. The recommendation was made by an Ad hoc committee made up of mine operators, equipment manufacturers, mining inspectorates and research agencies. As discussed in part II of this preamble, the committee has presently established a goal of 500 $\mu g/m^3$ as the recommended limit.

(5) Already noted in this preamble is the U.S. Environmental Protection Agency's recently enacted regulation of fine particulate matter, in light of the significantly increased health risks associated with environmental exposure to such particulates. In some of the areas studied, fine particulate is composed primarily of dpm; and significant mortality and morbidity effects were also noted in those areas.

(6) The California Environmental Protection Agency (CALEPA) has identified dpm as a toxic air contaminant, as defined in their Health and Safety Code, Section 39655. According to that section, a toxic air contaminant is an air pollutant which may cause or contribute to an increase in mortality or in serious illness, or which may pose a present or potential hazard to human health. This conclusion, unanimously adopted by the California Air Resources Board and its Scientific Review Panel on Toxic Air Contaminants, initiates a process of evaluating strategies for reducing dpm concentrations in California's ambient air.

(7) The International Programme on Chemical Safety (IPCS), which is a joint venture of the World Health Organization, the International Labour Organisation, and the United Nations Environment Programme, has issued a health criteria document on diesel fuel and exhaust emissions (IPCS, 1996). This document states that the data support a conclusion that inhalation of diesel exhaust is of concern with respect to both neoplastic and non-neoplastic diseases. It also states that the particulate phase appears to have the greatest effect on health, and both the particle core and the associated organic materials have biological activity, although the gas-phase components cannot be disregarded.

Based on both the epidemiological and toxicological evidence, the IPCS criteria document concluded that diesel exhaust is "probably carcinogenic to humans" and recommended that "in the occupational environment, good work practices should be encouraged, and adequate ventilation must be provided to prevent excessive exposure.'' Quantitative relationships between human lung cancer risk and dpm exposure were derived using a dosimetric model that accounted for differences between experimental animals and humans, lung deposition efficiency, lung particle clearance rates, lung surface area, ventilation, and elution rates of organic chemicals from the particle surface.

As the Supreme Court pointed out in the benzene case, the appropriate definition of significance also depends on policy considerations of the Agency involved. In the case of MSHA, those policy considerations include special attention to the history of the Mine Act. That history is intertwined with the toll to the mining community due to silicosis and coal miners' pneumoconiosis (''black lung''), along with billions of dollars in Federal expenditures.

At one of the 1995 workshops on diesel particulate co-sponsored by MSHA, a miner noted:

> People, they get complacent with things like this. They begin to believe, well, the government has got so many regulations on so many things. If this stuff was really hurting us, they wouldn't allow it in our coal mines * * * (dpm Workshop; Beckley, WV, 1995).

Referring to some commenters' position that further scientific study was necessary before a limit on dpm exposure could be justified, another miner said:

> * * * if I understand the Mine Act, it requires MSHA to set the rules based on the best set of available evidence, not possible evidence * * * Is it going to take us 10 more years before we kill out, or are we going to do something now * * *? (dpm Workshop; Beckley, WV, 1995).

Concern with the risk of waiting for additional scientific evidence to support regulation of dpm was also expressed by another miner who testified:

> What are the consequences that the threshold limit values are too high and it's loss of human lives, sickness, whatever, compared to what are the consequences that the values are too low? I mean, you don't lose nothing if they're too low, maybe a little money. But *** I got the indication that the diesel studies in rats could no way be compared to humans because their lungs are not the same * * * But * * * if we don't set the limits, if you remember probably last year when these reports come out how the government used human guinea pigs for radiation, shots, and all this, and aren't we doing the same thing by using coal miners as guinea pigs to set the value? (dpm Workshop; Beckley, WV, 1995).

III.3.c. Substantial Reduction of Risk by Proposed Rule

A review of the best available evidence indicates that reducing the very high exposures currently existing in underground mines can substantially reduce health risks to miners—and that greater reductions in exposure would result in even lower levels of risk. Although there are substantial uncertainties involved in converting 24-hour environmental exposures to 8-hour occupational exposures, Table III–3 suggests that reducing occupational dpm concentrations by as little as 75 $\mu g/m^3$ (corresponding to a reduction of 25 $\mu g/m^3$ in 24-hour ambient atmospheric concentration) could lead to significant reductions in the risk of various adverse acute responses, ranging from respiratory irritations to mortality.

Schwartz et al. (1996) found an increase of 1.5 percent in daily mortality associated with each increment of 10 $\mu g/m^3$ in the concentration of fine particulates. Somewhat higher increases were reported specifically for ischemic heart disease (IHD: 2.1 percent) and chronic obstructive pulmonary disease (COPD: 3.3 percent). Within the range of dust concentrations studied, the response appeared to be linear, with no threshold. Nor did Schwartz et al. find an association between increased mortality and the atmospheric concentration of larger particles.

If the 24-hour average concentrations measured by Schwartz et al. are assumed equivalent, in their acute effects, to eight-hour average concentrations that are three times as high, then (assuming the mining and general populations respond in similar ways) each increment of 30 $\mu g/m^3$ would, in an 8-hour shift occupational setting, be associated with a 1.5-percent increase in daily mortality. Since COPD and IHD were the diseases most clearly identified with acute diesel exposures, a conservative approach would be to limit consideration of any reduction in daily mortality risk under the proposed rule to deaths from IHD and COPD. IHD and COPD accounted for about one-third of the overall mortality. Thus, for purposes of estimating potential benefits, each reduction of 30 $\mu g/m^3$ in 8-hour average dpm concentration may be assumed to correspond to a 0.5-percent reduction (i.e., one-third of 1.5 percent) in daily mortality. This estimate is somewhat conservative, insofar as the reported effects on IHD and COPD mortality were both greater than the effects on overall mortality.

There are, however, additional problems in applying this incremental risk factor to underground M/NM miners. First, the levels of fine particulate concentration studied averaged around 20 $\mu g/m^3$, which is only about 10 percent of the final dpm concentration limit proposed and an even smaller fraction of average dpm concentrations measured at some underground M/NM mines. It is unclear whether the same incremental effects on mortality risks would apply at these much higher exposure levels. Second, Schwartz et al. studied fine particulate concentrations, which, though generally related to combustion products, include but are not limited to dpm. It is unclear how closely these results would match the effects of fine particulate dust made up exclusively of dpm. Third, and also discussed elsewhere in MSHA's risk assessment, is the question of whether underground M/NM mine workers comprise a population less, equally, or more susceptible than the general population to acute mortality effects of fine particulates. It is unclear how similar an exposure-response relationship for miners would be to the relationship observed for the general population. For these reasons, benefits of the proposed rule, as it impacts deaths related to IHD and/or COPD among M/NM miners, cannot be quantified with a high degree of confidence. Subject to these caveats, however, applying the findings of Schwartz et al. (adjusted as discussed above) would suggest that, for miners currently exposed to dpm at an average concentration of 830 $\mu g/m^3$ (i.e., the average of measurements made by MSHA at underground M/NM mines), the proposed rule would reduce the acute risk of IHD/COPD mortality by about 10 percent $[(830 - 200)\ \mu g/m^3 \times (0.5\% \div 30\ \mu g/m^3)]$.

Quantitative assessments of the relationship between human dpm exposures and lung cancer, which would show just how many cases of lung cancer a given reduction in exposure could be expected to prevent, have produced varying results and are subject to considerable uncertainty (Stayner *et al.*, 1998; US–EPA, 1998). None of the human-based dose-response relationships has been widely accepted in the scientific community, most likely due to a lack of precisely quantified dpm exposures in the available epidemiological studies. Although future studies may provide a better foundation for quantitative risk assessment, the Agency believes it would not be prudent to postpone protection of miners exposed to extremely high dpm levels until a conclusive dose-response relationship becomes available. In the meantime, the published, human-based quantitative risk assessments reviewed by Stayner et al. (1998) provide the best available means of estimating the reduction in lung cancer risk to underground M/NM miners that may be expected from reducing dpm exposures.

Among the human-based assessments reviewed, even the lowest estimate of unit risk of developing lung cancer is 10^{-4} per each µg/m^{3} of dpm exposure over a 45-year occupational lifetime at 8 hours of exposure per workday. It should be noted that this risk estimate was derived from exposures estimated to be generally below the proposed final limit. As Stayner et al. point out, there are some questions raised by extrapolating estimated risks to exposure levels up to 10 times as high, but doing so is unavoidable in order to estimate benefits based on existing data. On the other hand, the issue of whether a threshold exists is of little or no concern when assessing risk at these higher exposure levels. MSHA specifically requests information regarding any studies on miner mortality at high dpm exposures and the accuracy of the assumption of linearity.

Assuming this dose-response relationship, it is possible to estimate the reduction in lung cancers that could be expected as a result of implementing the proposed rule. To form such an estimate, however, measures of both current and proposed levels of dpm exposure are also required.

Table III–7 presents three estimates of current dpm exposure levels:

TABLE III–7.—MEASURES OF DPM EXPOSURE IN PRODUCTION AREAS AND HAULAGEWAYS OF UNDERGROUND M/NM MINES

	Employment size of mine			
	<20	20 to 500	>500	All Affected Mines
Number of Affected Mines	82	114	7	203
Number of Affected Miners	460	3,770	3,270	7,500
Dpm Concentration Estimated from Diesel Equipment Inventory				
Based on Test Data (µg/m^{3})	2,766	1,880	1,232	1,863
Adjusted for Observed Duty Cycle (µg/m^{3})	1,951	1,331	877	1,319
Mean dpm Concentration Level Observed in Underground M/NM Mines (µg/m^{3})				830

In its inventory of underground M/NM mines, MSHA collected data on diesel powered equipment, ventilation throughput, and the volume of the work areas. MSHA then estimated dpm concentration levels in the mines by combining these data with emissions data for the diesel engines obtained during testing in accordance with MSHA's engine approval process. The estimate of mean dpm concentration obtained by this method is 1,863 µg/m^{3}.

MSHA then compared the duty cycles for the diesel powered equipment used in the tests to the duty cycles observed in the mines. Recalibrating the results for the observed duty cycles lowered the estimated dpm concentrations by approximately 30 percent. The adjusted estimate of mean dpm concentration is 1,319 µg/m^{3}.

The third estimate of current mean dpm concentration shown in Table III–7 is the mean dpm concentration measured during MSHA's field studies, as shown in Table III–1 of this preamble. MSHA's dpm measurements averaged 830 µg/m^{3} at underground M/NM mines.

Applying the 10^{-4} estimate of unit risk to these three dpm concentration levels produces estimates of excess risk, for a 45-year period of exposure, of 186 cancers per 1,000 miners, 132 cancers per 1,000 miners, and 83 cancers per 1,000 miners, respectively. These estimates assume that the 45-year period of occupational exposure begins at age 20 and that the excess risk of dying from lung cancer is accumulated from age 20 through age 85-a span of 65 years.

Approximately 9,400 miners work in underground areas of M/NM mines that use diesel powered equipment, and MSHA estimates that about 80 percent (i.e., 7,500) of these work in production or development areas including haulageways. Therefore, if the 7,500 affected miners were all exposed for a full 45 years, this dose-response relationship would yield, over the 65-year period from time of first occupational exposure, 1,395 excess cancers, 990 excess cancers, or 622 excess cancers, corresponding to the three estimates of current mean exposure. For purposes of projecting benefits of the proposed rule, MSHA is restricting its attention to the lowest of these estimates, since it is based on actual measurements of dpm concentration.

Although many individual miners may work in underground M/NM mines for a full 45 years (and the Mine Act requires MSHA to set standards that protect workers exposed for a full working lifetime), MSHA believes that it may also be appropriate to estimate benefits of the proposed rule based on the mean duration of exposure. If the mean exposure time is actually 20 years, then the estimated excess risk of lung cancer could be reduced by roughly a factor of 20/45, from 83 per thousand miners to about 37 per thousand miners. However, since the total number of miners exposed during a given 45-year period will now be increased by a factor of 45/20, the total number of excess lung cancers expected at current exposure levels remains the same: 622, or an average of 9.6 per year, spread over an initial 65-year period.

After final implementation of the proposed rule, dpm concentrations in underground M/NM mines would be limited to a maximum of approximately 200 µg/m^{3} on each and every shift. Therefore, since concentrations would be expected to generally fall below their maximum value, it would be reasonable to assume that the average concentration would fall below 200 µg/m^{3}. (MSHA's sampling found concentrations under controlled conditions as low as 55 µg/m^{3}). So as not to overstate benefits, MSHA has projected residual risk under the proposed rule assuming the concentration limit of 200 µg/m^{3} is exactly met on all shifts at all mines.

From Table IV of Stayner et al. (1998), the lowest human-based risk estimate among workers occupationally exposed to 200 ➢g/m^{3} for 45 years is 21 excess lung cancers per 1000 exposed miners. For the population of 7,500 underground M/NM mine workers, this would amount to 158 excess lung cancers over an initial 65-year period, or an average of 2.4 excess lung cancers per year. If, as before, a 20-year average is assumed for occupational exposure, this reduces an individual miner's risk to a hypothetical 9.3 excess lung cancers per thousand exposed miners under the proposed rule, but the total number of excess lung cancers expected over the initial 65-year period remains the same. Thus, under the assumptions stated, the benefit of the proposed rule in reducing incidents of lung cancer can be expressed as:

- 622 − 158 = 464 lung cancers avoided over an initial 65-year period;[19] or
- 464 ÷ 65 = approximately 7 lung cancers avoided per year over an initial 65-year period; or
- 83 − 21 = 62 lung cancers avoided per 1,000 miners occupationally exposed for 45 years; or
- 37 − 9.3 = 28 lung cancers avoided per 1,000 miners occupationally exposed for 20 years.

The Agency recognizes that a conclusive, quantitative dose-response relationship has not been established between dpm and lung cancer in humans. However, the epidemiological studies relating dpm exposure to excess lung cancer were conducted on populations whose average exposure is estimated to be less than 200 $\mu g/m^3$ and less than one tenth of average exposures observed in some underground mines. Therefore, the best available evidence indicates that lifetime occupational exposure at levels currently existing in some underground mines presents a significant excess risk of lung cancer.

In the case of underground M/NM mines, the proposed rule limits dpm concentration to 200 $\mu g/m^3$ by limiting the measured concentration of total carbon to 160 $\mu g/m^3$. The Agency recognizes that although health risks would be substantially reduced, the best available evidence indicates a significant risk of adverse health effects would remain at these levels. However, as explained in Part V of this preamble, MSHA has concluded that, because of both technology and cost considerations, the underground M/NM mining sector as a whole cannot feasibly reduce dpm concentrations further at this time.

Conclusions. MSHA has reviewed a considerable body of evidence to ascertain whether and to what level dpm should be controlled. It has evaluated the information in light of the legal requirements governing regulatory action under the Mine Act. Particular attention was paid to issues and questions raised by the mining community in response to the Agency's Advance Notice of Proposed Rulemaking and at workshops on dpm held in 1995. Based on its review of the record as a whole to date, the agency has tentatively determined that the best available evidence warrants the following conclusions:

1. The health effects associated with exposure to dpm can materially impair miner health or functional capacity.

These material impairments include sensory irritations and respiratory symptoms; death from cardiovascular, cardiopulmonary, or respiratory causes; and lung cancer.

2. At exposure levels currently observed in underground M/NM mines, many miners are presently at significant risk of incurring these material impairments over a working lifetime.

3. The proposed rule for underground M/NM mines is justified because the reduction in dpm exposure levels that would result from implementation of the proposed rule would substantially reduce the significant health risks currently faced by underground M/NM miners exposed to dpm.

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[19] In the long run, the average approaches 464 ÷ 45 = 10 lung cancers avoided per year as the number of years considered increases beyond 65.

Table III-2. Studies of acute health effects using filter based optical indicators of fine particles in the ambient air.

City	Study Years	Indicator*	Reference
Acute Mortality			
London	1963-1972, winters 1965-1972, winters	BS	Thurston et al., 1989 Ito et al., 1993
Athens	1975-1987 July, 1987 1984-1988	BS	Katsouyanni et al., 1990 Katsouyanni et al., 1993 Touloumi et al., 1994
Los Angeles	1970-1979 1970-1979	KM	Shumway et al., 1988 Kinney and Ozkaynak, 1991
Santa Clara	1980-1986, winters	COH	Fairley, 1990
Increased Hospitalization			
Barcelona	1985-1989	BS	Sunyer et al., 1993
Acute Change in Pulmonary Function			
Wageningen, Netherlands		BS	Hoek and Brunkreef, 1993
Netherlands		BS	Roemer et al., 1993

*BS (black smoke), KM (carbonaceous material), and COH (coefficient of haze) are optical measurements that are most directly related to elemental carbon concentrations, but only indirectly to mass. Site specific calibrations and/or comparisons of such optical measurements with gravimetric mass measurements in the same time and city are needed to make inferences about particle mass. However, all three of these indicators preferentially measure carbon particles found in the fine fraction of total airborne particulate matter. (EPA, 1996).

TABLE III–3.—STUDIES OF ACUTE HEALTH EFFECTS USING GRAVIMETRIC INDICATORS OF FINE PARTICLES IN THE AMBIENT AIR

	Indicator	RR(± CI)/25µg/m^3 PM increase	Mean PM levels (min/max)†
Acute Mortality			
Six Cities[A]			
Portage, WI	$PM_{2.5}$	1.030 (0.993,1.071)	11.2 (±7.8)
Topeka, KS	$PM_{2.5}$	1.020 (0.951,1.092)	12.2 (±7.4)
Boston, MA	$PM_{2.5}$	1.056 (1.038,1.0711)	15.7 (±9.2)
St. Louis, MO	$PM_{2.5}$	1.028 (1.010,1.043)	18.7 (±10.5)
Kingston/Knoxville, TN	$PM_{2.5}$	1.035 (1.005,1.066)	20.8 (±9.6)
Steubenville, OH	$PM_{2.5}$	1.025 (0.998,1.053)	29.6 (±21.9)
Increased Hospitalization			
Ontario, CAN[B]	$SO_4^=$	1.03 (1.02, 1.04)	Min/Max = 3.1 – 8.2
Ontario, CAN[C]	$SO_4^=$	1.03 (1.02, 1.04)	Min/Max = 2.0 – 7.7
	O_3	1.03 (1.02, 1.05)	
NYC/Buffalo, NY[D]	$SO_4^=$	1.05 (1.01, 1.10)	NR
Toronto, CAN[D]	H+ (Nmo1/m^3)	1.16 (1.03, 1.30) *	28.8 (NR/391)
	$SO_4^=$	1.12 (1.00, 1.24)	7.6 (NR, 48.7)
	$PM_{2.5}$	1.15 (1.02, 1.78)	18.6 (NR, 66.0)
Increased Respiratory Symptoms			
Southern California[F]	$SO_4^=$	1.48 (1.14, 1.91)	R = 2 – 37
Six Cities[G] (Cough)	$PM_{2.5}$	1.19 (1.01, 1.42)**	18.0 (7.2, 37)***
	$PM_{2.5}$ Sulfur	1.23 (0.95, 1.59)**	2.5 (3.1, 61)***
	H+	1.06 (0.87, 1.29)**	18.1 (0.8, 5.9)***
Six Cities[G] (Lower Resp. Symp.)	$PM_{2.5}$	1.44 (1.15 – 1.82)**	18.0 (7.2, 37)***
	$PM_{2.5}$ Sulfur	1.82 (1.28 – 2.59)**	2.5 (0.8, 5.9)***
	H+	1.05 (0.25 – 1.30)**	18.1 (3.1, 61)***
Denver, CO[P] (Cough, adult asthmatics)	$PM_{2.5}$	0.0012 (0.0043)***	0.41 – 73
	$SO_4^=$	0.0042 (0.00035)***	0.12 – 12
	H+	0.0076 (0.0038)***	2.0 – 41
Decreased Lung Function			
Uniontown, PA[E]	$PM_{2.5}$	PEFR 23.1 (– 0.3, 36.9) (per 25 µg/m^3).	25/88 (NR/88)
Seattle, WA[Q] Asthmatics	$b_{ext.}$	FEV1 42 ml (12, 73)	5/45
	calibrated by $PM_{2.5}$	FVC 45 ml (20, 70)	

(EPA, 1996).

[A] Schwartz et al. (1996a).

[B] Burnett et al. (1994).

[C] Burnett et al. (1995) O_3.

[D] Thurston et al. (1992, 1994).

[E] Neas et al. (1995).

[F] Ostro et al. (1993).

[G] Schwartz et al. (1994).

[Q] Koenig et al. (1993).

[P] Ostro et al. (1991).

† Min/Max 24 – h PM indicator level shown in parentheses unless otherwise noted as (±S.D), 10 and 90 percentile (10, 90).

* Change per 100 nmoles/m^3.

** Change per 20 µg/m^3 for $PM_{2.5}$; per 5 µg/m^3 for $PM_{2.5}$; sulfur; per 25 nmoles/m^3 for H+.

*** 50th percentile value (10, 90 percentile).

**** Coefficient and SE in parenthesis.

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Table III-4. Summary of published information from cohort studies on lung cancer and exposure to diesel exhaust.

Authors (Date)	Occupation	No. of Subjects	Follow-up period	Exposure Assessment	Smk Adj.	Findings[a]	Stat. Sig.[b]	Comments
Ahlberg et al. (1981)	Male truck drivers	35,883	1961-73	Occupation only		RR = 1.33 for drivers of "ordinary" trucks.	*	Risk relative to males employed in trades thought to have no exposure to "petroleum products or other chemicals." Comparison controlled for age and province of residence (Sweden). Based on comparison of smoking habits between truck drivers and general Stockholm population, authors concluded that excess rate of lung cancer could not be entirely attributed to smoking.
Ahlman et al. (1991)	Underground sulfide ore miners	597	1968-86	Job histories from personnel records. Measurements of alpha energy concentration from radon daughters at each mine worked.		RR = 1.45 overall. RR = 2.9 for 45-64 age group.		Age-adjusted relative risk compared to males living in same area of Finland. No excess observed among 338 surface workers at same mines, with similar smoking and alcohol consumption, based on questionnaire. Based on calculation of expected lung cancers due to radon, excess risk attributed by author partly to radon exposure and partly to diesel exhaust.
Balarajan & McDowall (1988)	Professional drivers	3,392	1950-84	Occupation only		SMR = 0.86 for taxi drivers. SMR = 1.42 for bus drivers. SMR = 1.59 for truck drivers.	 *	Possibly higher rates of smoking among bus and truck drivers than among taxi drivers.
Bender et al. (1989)	Highway maintenance workers	4,849	1945-84	Occupation only		SMR = 0.69		No adjustment for healthy worker effect.
Boffetta et al. (1988)	Railroad Wrkr. Truck driver Heavy Eq. Op. Miner General Popula.	2,973 16,208 855 2,034 476,648	1982-84	Occupation and diesel exposure by questionnaire	✓	RR = 1.59 for railroad workers. RR = 1.24 for truck drivers. RR = 2.60 for heavy Eq. Op's. RR = 2.67 for miners. RR = 1.18 for subjects reporting diesel exposure compared to subjects reporting no diesel exposure.	 * *	Overall RR adjusted for occupational exposures to asbestos, coal and stone dusts, coal tar & pitch, and gasoline exhaust (in addition to age and smoking). Possible biases due to volunteered participation and relatively high lung cancer rate among 98,026 subjects with unknown dpm exposure.

Dubrow & Wegman (1984)	Truck & tractor drivers	not reported	1971-73	Occupation only		sMOR = 1.73 based on 176 deaths.	*	Excess cancers observed over the entire respiratory system and upper alimentary tract.
Edling et al. (1987)	Bus workers	694	1951-83	Occupation only		SMR = 0.7 for overall cohort		Small size of cohort lacks statistical power to detect excess risk of lung cancer. No adjustment for healthy worker effect.
Garshick et al. (1988)	Railroad workers	55,407	1959-80	Job in 1959 & years of diesel exposure since 1959		RR = 1.20 for 1-4 yr. exposure. RR = 1.24 for 5-9 yr. exposure. RR = 1.32 for 10-14 yr. exposure. RR = 1.72 for ≥15 yr. exposure. Higher RR for each exposure group if shopworkers and hostlers are excluded. RR = 1.45 within highest-exposed age group (40-44).	* * * * *	Exposure groups based on exposure accumulated more than 4 yr prior to observation. Subjects with likely asbestos exposure excluded from cohort. Statistically significant results corroborated if 12,872 shopworkers and hostlers possibly exposed to asbestos are also excluded. Missing 12% of death certificates. Cigarette smoking judged to be uncorrelated with diesel exposure within cohort.
Guberan et al. (1992)	Professional drivers	1,726	1961-86	Occupation only		SMR = 1.50	*	Approx. 1/3 to 1/4 of cohort reported to be long-haul truck drivers. SMR based on regional lung cancer mortality rate.
Gustafsson et al. (1986)	Dock workers	6,071	1961-80	Occupation only		SMR = 1.32 (mortality). SMR = 1.68 (morbidity).	* *	
Gustavsson et al. (1990)	Bus garage workers	708	1952-86	Semi-quantitative based on job history & exposure intensity estimated for each job.		SMR = 1.22 for overall cohort. SMR = 1.27 for highest-exposed subgroup.		Lack of statistical significance may be attributed to small size of cohort.

Hansen (1993)	Truck drivers	14,225	1970-80	Occupation only		SMR = 1.60 for overall cohort. Some indication of increasing SMR with age (i.e., greater cumulative exposure).	*	Compared to unexposed control group of 38,301 laborers considered to "resemble the group of truck drivers in terms of work-related demands on physical strength and fitness, educational background, social class, and life style." Correction for estimated differences in smoking habits between cohort and control group reduces SMR from 1.60 to 1.52. Results judged "unlikely *** [to] have been seriously confounded by smoking habit differences."
Howe et al. (1983)	Railroad workers	43,826	1965-77	Jobs classified by diesel exposure		Rr = 1.20 for "possibly exposed." RR = 1.35 for "probably exposed."	* *	Risk is relative to unexposed subgroup of cohort. Similar results obtained for coal dust exposure. Possible confounding with asbestos and coal dust.
Kaplan (1959)	Railroad workers	32000 (Approx.)	1953-58	Jobs classified by diesel exposure		SMR=0.88 for operationally exposed. SMR = 0.72 for somewhat exposed. SMR = 0.80 for rarely exposed.		No adjustment for healthy worker effect. Clerks (in rarely exposed group) found more likely to have had urban residence than occupationally exposed workers. No attempt to distinguish between diesel and coal-fired locomotives. Results may be attributable to short duration of exposure and/or inadequate follow-up time.
Leupker & Smith (1978)	Truck drivers	183,791	May-July, 1976	Occupation only		SMR = 1.21		Lack of statistical significance may be due to inadequate follow-up period.
Lindsay et al. (1993)	Truck drivers	not reported	1965-79	Occupation only		SMR = 1.15	*	
Menck & Henderson (1976)	Truck drivers	34,800 estimated	1968-73	Occupation only		SMR = 1.65	*	Number of subjects in cohort estimated from census data.
Raffle (1957)	Transport engineers	2,666 Est. from man-years at risk	1950-55	Occupation only		SMR = 1.42		SMR calculated by combining data presented for four quadrants of London.

Rafnsson & Gunnarsdottir (1991)	Truck drivers	868	1951-88	Occupation only		SMR = 2.14	*	No trend of increasing risk with increased duration of employment or increased follow-up time. Based on survey of smoking habits in cohort compared to general male population, and fact that there were fewer than expected deaths from respiratory disease, authors concluded that differences in smoking habits were unlikely to be enough to explain excess rate of lung cancer. However, not all trucks were diesel prior to 1951, and there is possible confounding by asbestos exposure.
Rushton et al. (1983)	Bus maintenance workers	8,480	5.9 yrs (mean)	Occupation only		SMR = 1.01 for overall cohort. SMR = 1.33 for "general hand" subgroup.	*	Short follow-up period. SMR based on comparison to national rates, with no adjustment for regional or socioeconomic differences, which could account for excess lung cancers observed among general hands.
Schenker et al. (1984)	Railroad workers	2,519	1967-79	Job histories, with exposure classified as unexposed, high, low, or undefined.		RR = 1.50 for low exposure subgroup. RR = 2.77 for high exposure subgroup.		Risk relative to unexposed subgroup. Jobs considered to have similar socioeconomic status. Differences in smoking calculated to be insufficient to explain findings. Possible confounding by asbestos exposure.
Waller (1981)	Bus workers	16,828 Est. from manyears at risk	1950-74	Occupation only		SMR = 0.79 for overall cohort.		Lung cancers occurring after retirement or resignation from London Transport Authority were not counted. No adjustment for healthy worker effect.
Waxweiler et al. (1973)	Potash miners	3,886	1941-67	Miners classified as underground or surface		SMR = 1.12 for surface miners. SMR = 1.08 for underground miners.		No adjustment for healthy worker effect. SMR based on national lung cancer mortality, which is about 1/3 higher than lung cancer mortality rate in New Mexico, where miners resided. A substantial percentage of the underground subgroup may have had little or no occupational exposure to diesel exhaust.
Wong et al. (1985)	Heavy equipment operators	34,156	1964-78	Job histories, latency, & years of union membership		SMR = 0.99 for overall cohort. SMR = 1.07 for ≥20 yr member. SMR = 1.12 for ≥20 yr. latency. SMR = 1.30 for 4,075 "normal" retirees.	*	Increasing trend in SMR with latency and (up to 15 yr) with duration of union membership. Statistically significant excess lung cancers for dozer operators with 15-19 yr union membership and ≥20 yr latency. No adjustment for healthy worker effect.

a RR = Relative Risk; SMR = Standardized Mortality Ratio. Values greater than 1.0 indicate excess prevalence of lung cancer associated with diesel exposure.

b An asterisk (*) indicates statistical significance based on 2-tailed test at confidence level of at least 95%.

Table III-5 – Summary of published information from case-control studies on lung cancer and exposure to diesel exhaust.

Authors (Date)	Cases	Controls	No. of Cases	No. of Controls	Exposure Assessment	Matching		Findings[a]	Stat. Sig.[b]	Comments
						Smk.	Additional			
Benhamou et al. (1988)	Histologically confirmed lung cancers	Non-tobacco related diseases	1,625	3,091	Occupational history by questionnaire.	✓	Sex, age at diagnosis, hospital, interviewer.	RR = 2.14 for miners RR = 1.42 for professional drivers.	* *	Mine type not reported. No evidence of an increase in risk with duration of exposure
Boffetta et al. (1990)	Hospitalized males with lung cancer	Hospitalized males with no tobacco related disease	2,584	5,099	Occupation classified by probability of diesel exposure	✓	Sex, age, hospital, year of interview.	OR = 0.88 for truck drivers. OR = 0.95 for probable exposure.		Adjusted for race, asbestos exposure, education.
			477	846	Occupational history & duration of diesel exposure by interview			OR = 1.21 for any self-reported diesel exposure. OR = 2.39 for than 30 yr of self-reported diesel exposure.		
Buiatti et al. (1985)	Histologically confirmed lung cancers	Patients at same hospital	376	892	Occupational history from interview	✓	Sex, age, admission date.	OR = 1.8 for taxi drivers.		
Coggon et al. (1984)	Lung cancer deaths of males under 40	Deaths from other causes in males under 40	598	1,180	Occupation from death certificate, classified as high, low, or no diesel exposure		Sex, death year, region, and birth year (approx.)	RR = 1.3 for all jobs with diesel exposure. RR=1.1 for jobs classified as high exposure.	*	Only most recent full-time occupation recorded on death certificate.
Damber & Larsson (1985)	Male patients with lung cancer	One living and one deceased without lung cancer	604	1,071	Job, with tenure, from mailed questionnaire	✓	Sex, death year, age, municipality	RR = 1.9 for non-smoking truck drivers aged <70 yr. RR = 4.5 for non-smoking truck drivers aged ≥70 yr.	 *	Ex-smokers who did not smoke for at least last 10 years included with non-smokers.

DeCoufle et al. (1977)	Male patients with lung cancer	Non-neoplastic disease patients	Not reported	Not reported	Occupation only, from questionnaire	✔	Unmatched	RR = 0.92 for bus, taxi, and truck drivers. RR = 0.94 for locomotive engineers.		Selected occupation compared to clerical workers. Positive associations found before smoking adj.
Emmelin et al. (1993)	Deaths from primary lung cancer among dock workers	Dock workers without lung cancer	50	154	Semi-quantitative from work history & records of diesel fuel usage	✔	Date of birth, port, and survival to within 2 years of case's diagnosis of lung cancer	RR = 1.6 for "medium" duration of exposure. RR = 2.9 for "high" duration of exposure.		Increasing relative risk also observed using exposure estimates based on machine usage & diesel fuel consumption. Confounding from asbestos may be significant.
Garshick et al. (1987)	Deaths with primary lung cancer among railroad workers	Deaths from other than cancer, suicide, accidents, or unknown causes	1,256	2,385	Job history and tenure combined with current exposure levels measured for each job	✔	Date of birth and death	RR = 1.41 for 20+diesel-years in workers aged≤64 yr. RR = 0.91 for workers aged ≥65 yr.	*	Adjusted for asbestos exposure. Older workers had relatively short diesel exposure, or none.
Gustavsson et al. (1990)	Deaths from lung cancer among bus garage workers	Non-cases within cohort mortality study	20	120	Semi-quantitative based on job, tenure, & exposure class for each job		Born within two years of case.	RR = 1.34, 1.81, and 2.43 for increasing cumulative diesel exposure categories, relative to lowest exposure category.	*	Authors judged smoking habits to be similar for different exposure categories. RR did not increase with increasing asbestos exposure
Hall & Wynder (1984)	Hospitalized males with lung cancer	Hospitalized males with no tobacco-related diseases	502	502	Usual occupation by interview	✔	Age, race, hospital, and hospital room status	RR = 1.4 for jobs with diesel exposure.		Confounding with other occupational exposures possible.

Hayes et al. (1989)	Lung cancer deaths pooled from 3 studies	Various -- lung disease excluded	2,291	2,570	Occupational history by interview	✓	Sex, age, and either race or area of residence	OR = 1.5 for ≥10 yr truck driving. OR = 2.1 for ≥10 yr operating heavy equipment. OR = 1.7 for ≥10 yr bus driving.	*	OR adjusted for birth-year cohort and state of residence (FL, NJ, or LA), in addition to average cigarette use. Smaller OR for <10 yr in these jobs.
Lerchen et al. (1987)	New Mexico residents with lung cancer	Medicare recipients	506	771	Occupational history, industry, & self-reported exposure, by interview	✓	Sex, age, ethnicity	OR = 0.6 for ≥1 yr occupational exposure to diesel exhaust. OR = 2.1 for underground non-uranium mining.		Small number of cases and controls in diesel-exposed jobs. Possibly insufficient exposure duration. Not matched on date of birth or death.
Milne et al. (1983)	Lung cancer deaths	Deaths from any other cancer	925	6,565	Occupation from death certificate		None	OR = 3.5 for bus drivers. OR = 1.6 for truck drivers.	*	
Morabia et al. (1992)	Male lung cancer patients	Patients without lung cancer or other tobacco-related condition	1,793	3,228	Job, with coal and asbestos exposure durations, by interview	✓	Race, age, hospital, and smoking history	OR = 2.3 for miners. OR = 1.1 for bus drivers. OR = 1.0 for truck or tractor drivers.		Lung cancer reported to be associated with increasing duration of exposure to coal.
Pfluger and Minder (1994)	Professional drivers	Workers in occupational categories with no known excess lung cancer risk.	284	1,301	Occupation only, from death certificate.		None.	OR = 1.48 for professional drivers.	*	Stratified by age. Indirectly adjusted for smoking, based on smoking-rate for occupation.

Siemiatycki et al. (1988)	Squamous cell lung cancer patients by type of lung cancer	Other cancer patients	359	1,523	Semi-quantitative, from Occupa. history by interview, & exposure class for each job	✓	None	OR = 1.2 for diesel exposure; OR = 2.8 for mining.		Stratified by age, socioeconomic status, ethnicity, and blue- vs. white-collar job history. Examination of files indicated that most miners "were exposed to diesel exhaust for short periods of time."
Steenland et al. (1990)	Deaths from lung CA among Teamsters	Deaths excluding LC, bladder cancer, and motor vehicle accidents	996	1,085	Occupational history and tenure from next-of-kin, supplemented by IH data	✓	None	OR = 1.27 for diesel truck drivers with 1-24 yr. tenure. OR = 1.26 for diesel truck drivers with 25-34 yr. tenure. OR = 1.89 for diesel truck drivers with ≥35 yr. tenure.	*	Years of tenure not necessarily all at main job (i.e., diesel truck driver). OR adjusted for asbestos exposure.

Swanson et al. (1993) See also Burns & Swanson (1991)	Detroit lung cancers	Colon or rectal cancer cases	5,935	3,956	Occupational history from interview	✔	None	OR = 1.4 for heavy truck drivers with 1-9 yr tenure. OR = 1.6 for heavy truck drivers with 10-19 yr tenure. OR = 2.4 for heavy truck drivers with ≥20 yr tenure. ------------------ OR = 1.2 for railroad workers with 1-9 yr tenure. OR = 2.5 for railroad workers with ≥10 yr tenure. ------------------ OR = 5.03 for mining machine operators.	* * *	OR for truck drivers & RR workers is for white males, relative to corresponding group with <1 yr tenure, adjusted for age at diagnosis. Pattern of increasing risk with duration of employment also reported for black male railroad workers, based on fewer cases. OR for mining machine operators is for all males, adj. for race and age at diagnosis.
Williams et al. (1977)	Male lung cancer patients	Other male cancer patients	432	2,817	Main lifetime occupation from interview	✔	Sex	OR = 1.52 for male truck drivers.		Controlled for age, race, alcohol use, and socioeconomic status. Unexplained discrepancies in reported number of controls.

[a] RR = Relative Risk; OR = Odds Ratio. Values greater than 1.0 indicate excess prevalence of lung cancer associated with diesel exposure.

[b] An asterisk (*) indicates statistical significance based on 2-tailed test at confidence level of at least 95%.

Table III-6. — Hypothesized Mechanisms of Particulate Toxicity[a]

Response	Description
Increased Airflow Obstruction	PM exposure may aggravate existing respiratory symptoms which feature airway obstruction. PM-induced airway narrowing or airway obstruction from increased mucous secretion may increase abnormal ventilation/perfusion ratios in the lung and create hypoxia. Hypoxia may lead to cardiac arrhythmias and other cardiac electrophysiologic responses that in turn may lead to ventricular fibrillation and ultimately cardiac arrest. For those experiencing airflow obstruction, increased airflow into non-obstructed areas of the lung may lead to increased particle deposition and subsequent deleterious effects on remaining lung tissue, further exacerbating existing disease processes. More frequent and severe symptoms may be present or more rapid loss of function.
Impaired Clearance	PM exposure may impair clearance by promoting hypersecretion of mucus which in turn results in plugging of airways. Alterations in clearance may also extend the time that particles or potentially harmful biogenic aerosols reside in the tracheobronchial region of the lung. Consequently alterations in clearance from either disturbance of the mucociliary escalator or of macrophage function may increase susceptibility to infection, produce an inflammatory response, or amplify the response to increased burdens of PM. Acid aerosols impair mucociliary clearance.
Altered Host Defense	Responses to an immunological challenge (e.g., infection), may enhance the subsequent response to inhalation of nonspecific material (e.g., PM). PM exposure may also act directly on macrophage function which may not only affect clearance of particles but also increase susceptibility and severity of infection by altering their immunological function. Therefore, depression or over-activation of the immune system, caused by exposure to PM, may be involved in the pathogenesis of lung disease. Decreased respiratory defense may result in increased risk of mortality from pneumonia and increased morbidity (e.g., infection).
Cardiovascular Perturbation	Pulmonary responses to PM exposure may include hypoxia, bronchoconstriction, apnea, impaired diffusion, and production of inflammatory mediators that can contribute to cardiovascular perturbation. Inhaled particles could act at the level of the pulmonary vasculature by increasing pulmonary vascular resistance and further increase ventilation/perfusion abnormalities and hypoxia. Generalized hypoxia could result in pulmonary hypertension and interstitial edema that would impose further workload on the heart. In addition, mediators released during an inflammatory response could cause release of factors in the clotting cascade that may lead to increased risk of thrombus formation in the vascular system. Finally, direct stimulation by PM of respiratory receptors found throughout the respiratory tract may have direct cardiovascular effects (e.g., bradycardia, hypertension, arrhythmia, apnea and cardiac arrest).
Epithelial Lining Changes	PM or its pathophysiological reaction products may act at the alveolar capillary membrane by increasing the diffusion distances across the respiratory membrane (by increasing its thickness) and causing abnormal ventilation/perfusion ratios. Inflammation caused by PM may increase "leakiness" in pulmonary capillaries leading eventually to increased fluid transudation and possibly to interstitial edema in susceptible individuals. PM induced changes in the surfactant layer leading to increased surface tension would have the same effect.
Inflammatory Response	Diseases which increase susceptibility to PM toxicity involve inflammatory response (e.g., asthma, COPD, and infection). PM may induce or enhance inflammatory responses in the lung which may lead to increased permeability, diffusion abnormality, or increased risk of thrombus formation in vascular system. Inflammation from PM exposure may also decrease phagocytosis by alveolar macrophages and therefore reduce particle clearance. (See discussions above for other inflammatory effects from PM exposure.)

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[a] This table reproduces Table V-2 of the EPA staff paper. The citation in the staff paper indicates the table is derived from information in the EPA criteria document on particulate matter (p. 13-67 to 72; p. 11-179 to 185) and information in Appendix D of EPA staff paper.

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IV. Discussion of Proposed Rule

This part of the preamble explains, section-by-section, the provisions of the proposed rule. As appropriate, this part references discussions in other parts of this preamble: in particular, the background discussions on measurement methods and controls in Part II, and the feasibility discussions in Part V.

The proposed rule would add nine new sections to 30 CFR Part 57 immediately following § 57.5015. It would not amend any existing sections of that part.

Section 57.5060 Limit on Concentration of Diesel Particulate Matter

This section of the proposed rule limits the concentration of dpm in underground metal and nonmetal mines. It has four subsections.

Paragraph (a) of § 57.5060 provides that 18 months after the date of promulgation, dpm concentrations to which miners are exposed would be limited by restricting total carbon to 400 micrograms per cubic meter of air. As proposed by the rule, this limit would apply only for a period of 36 months; accordingly, it is sometimes referred to in this preamble as the ''interim'' concentration limit.

Paragraph (b) of § 57.5060 provides that after five years the proposed concentration limit would be reduced, restricting total carbon to 160 micrograms per cubic meter of air. This is sometimes referred to in this preamble as the ''final'' concentration limit.

Paragraph (c) of § 57.5060 provides for a special extension of up to two additional years in order for a mine to comply with the final concentration limit. This special extension is only available when the mine operator can establish that the final concentration limit cannot be met within the five years allotted due to technological constraints. The proposed rule establishes the details that must be provided in the application process, and conditions that must be observed during the special extension period. Paragraph (c) of the proposed rule refers to this extension as ''special'' because the proposed rule would also provide all mines in this sector with up to five years to meet the final concentration limit.

Paragraph (d) of § 57.5060 provides that an operator shall not utilize personal protective equipment to comply with either the interim or final concentration limit. Moreover, it provides that an operator shall not utilize administrative controls to comply with either the interim or final concentration limit. These restrictions do not explicitly apply to an operator who has been provided with a special extension of time to comply with the final concentration limit pursuant to paragraph (c).

Choice of Controls. With the exceptions specified in paragraph (d), the proposed rule contemplates that an operator of an underground metal or nonmetal mine have complete discretion over the controls utilized to meet the interim and final concentration limits. No specific controls would be required for any type of diesel engine, for any type of diesel equipment, or for any type of mine in this sector. An operator could filter the emissions from diesel-powered equipment, install cleaner-burning engines, increase ventilation, improve fleet management, or use a variety of other available controls.

Because information on available controls has been described in Part II of this preamble, including the ''Toolbox'' (appended to the end of this document is a copy of an MSHA publication, ''Practical Ways to Reduce Exposure to Diesel Exhaust in Mining—A Toolbox''), further discussion is not provided here. Reviewers are also referred to the extensive discussion of available controls in Part V of this preamble concerning the technological and economic feasibility of this rule for the underground metal and nonmetal mining sector.

To help mine operators decide among various alternative combinations of engineering and ventilation controls, MSHA has developed a model that it believes will assist an operator to determine, for a production area of a mine, the effect of any combination of controls on existing dpm concentrations in that area. This model, known as the ''Estimator'', is in the form of a spreadsheet template; this permits instant display of outcomes as inputs are altered. The model is described in detail in Part V of this preamble, and some examples illustrating its potential utility are described there. MSHA welcomes comments from the mining community concerning this model, and encourages mine operators to submit their results as part of their comments.

Expression of Limits. The interim and final concentration limits on diesel particulate matter are expressed in terms of a restriction on the amount of total carbon present. The purpose of the interim and final concentration limits is to limit the amount of diesel particulate matter to which miners are exposed; but the limit is being expressed in terms of the measurement method that MSHA intends to utilize to determine the concentration of dpm. The idea is to enable miners, mine operators and inspectors to directly compare a measurement result with the applicable limit.

As discussed in connection with proposed § 57.5061(a), MSHA intends to use a sampling and analytical method developed by NIOSH (NIOSH Analytical Method 5040) to measure dpm concentrations for compliance purposes. NIOSH's Analytical Method 5040 accurately determines the amount of total carbon (TC) contained in a dpm sample from any underground metal and nonmetal mine.

As explained in detail in Part II of this preamble, whole diesel particulate matter can be measured in a variety of ways. But to date, a method that measures whole dpm directly has not been validated as providing accurate measurements at lower concentration levels with the consistency desirable for compliance purposes. However, MSHA believes that for underground metal and nonmetal mines, there is a surrogate method with the requisite accuracy. The surrogate is a method that determines the amount of certain component parts of whole dpm. Whole dpm basically consists of: the elemental carbon (EC) making up the core of the dpm particle; the organic carbon (OC) contained in adsorbed hydrocarbons; and some sulfates. (See Figure II–3 for a graphic representation of a dpm particle). The total carbon (TC) consists of the EC and the OC. NIOSH Method 5040 has been shown to measure TC with adequate accuracy. As discussed in Part II, MSHA is not aware at this time of any interferents that would in practice preclude MSHA from using this method to obtain consistent results in underground metal and nonmetal mines; hence, the Agency is proposing to use this method for compliance.

TC represents approximately 80–85 percent of the total mass of dpm emitted in the exhaust of a diesel engine (the remaining 15–20 percent consists of sulfates and the various elements bound up with the organic carbon to form the adsorbed hydrocarbons). Using the lower boundary of this range, limiting the concentration of total carbon to 400 micrograms per cubic meter (400_{TC} $\mu g/m^3$) limits the concentration of whole diesel particulate to about 500_{DPM} $\mu g/m^3$. Similarly, limiting the concentration of total carbon to 160_{TC} $\mu g/m^3$ limits the concentration of whole diesel particulate to about 200_{DPM} $\mu g/m^3$.

By way of comparison, MSHA has measured dpm average concentrations in underground metal and nonmetal mines from about 68_{DPM} $\mu g/m^3$ to $1{,}835_{DPM}$ $\mu g/m^3$. MSHA has recorded some concentrations as high as $5{,}570_{DPM}\ \mu g/m^3$. Complete information about these measurements, and the methods used in measuring them, are discussed in Part III of this preamble.

Where the Concentration Limit Applies. The concentration limits—both interim and final—would apply only in areas where miners normally work or travel. The purpose of this restriction is to ensure that mine operators do not have to monitor particulate concentrations in areas where miners do not normally work or travel — e.g., abandoned areas of a mine. However, the appropriate concentration limit would need to be maintained in any area of a mine where miners normally work or travel even if miners might not be present at any particular time. (For a discussion of MSHA's proposed sampling strategy, see the discussion of proposed § 57.5061(a)).

Full-shift, 8-hour Equivalent. The proposed interim and final concentration limits are expressed in terms of the average airborne concentration during each full shift expressed as an 8-hour equivalent. Measuring over a full shift ensures that average exposure is monitored over the same period to which the limit applies. Using an 8-hour equivalent dose ensures that a miner who works extended shifts—and many do—would not be exposed to more dpm than a miner who works a normal shift. The Agency welcomes comment on whether a more explicit definition is required in this regard.

Concentration Limit: Time to Meet. As noted, the dpm limitation being proposed would require metal and nonmetal mines to reduce dpm concentrations in areas where miners normally work or travel to about 200 micrograms per cubic meter of air (specifically, total carbon would have to be restricted to 160 micrograms per cubic meter of air). Proposed § 57.5060 provides an extension of time for underground metal and nonmetal mines to meet the concentration limit. Mines would not have to meet any limit within 18 months of the rule's promulgation. This period would be used to provide compliance assistance to the metal and nonmetal mining community to ensure it understands how to measure and control diesel particulate matter concentrations in individual operations. Moreover, the proposed rule would provide all mines in this sector three and a half additional years to meet the final concentration limit established by proposed § 57.5060(b). During this time, however, all mines would have to bring dpm concentrations down to 500 micrograms per cubic meter by complying with a restriction on the concentration of submicrometer total carbon of 400 micrograms per cubic meter.

MSHA established these requirements after carefully reviewing questions presented by the mining community regarding economic and technological feasibility of requiring all mines in this sector to meet the proposed concentration limit with available controls. This review is presented in Part V of this preamble. MSHA has studied a number of metal and nonmetal mines in which it believed dpm might be particularly difficult to control. The Agency has tentatively concluded that in combination with the ''best practices'' required under other provisions of the proposed rule (§§ 57.5065, 57.5066 and 57.5067), engineering and work practice controls are available that can bring dpm concentrations in all underground metal and nonmetal mines down to or below $400_{TC}\ \mu g/m^3$ within 18 months. Moreover, based on the mines it has examined to date, the Agency has tentatively concluded that controls are available to bring dpm concentrations in underground metal and nonmetal mines down to or below $160_{TC}\ \mu g/m^3$ within 5 years.

The Agency has tentatively concluded that it may not be feasible to require this sector, as a whole, to lower dpm concentrations further, or to implement the required controls more swiftly. Nevertheless, as noted in Part V, the Agency is seeking information, examples and comment that will assist it in making a final determination on these points.

Special Extension. An operator may request more than five years to comply with the final concentration limit only in the case of technological constraints that preclude compliance. MSHA has determined that it is economically feasible for the mining industry as a whole to comply with the proposed concentration limit within five years. In light of the risks to miners posed by dpm, the Agency does not believe the economic constraints of a particular operator should provide an adequate basis for a further extension of time for that operator, and the proposal would not provide for any extension grounded on economic concerns. Moreover, if it is technologically feasible for an operator to reduce dpm concentrations to the final limit in time through any approach, no extension would be permitted even if a more cost effective solution might be available in the future for that operator.

However, the Agency believes that if an operator can actually demonstrate that there is no technological solution that could reduce the concentration of dpm within five years, a special extension would be warranted. As a practical matter, MSHA believes that very few, if any, underground metal and nonmetal mining operations should need a special extension. MSHA bases this belief on information discussed in Part V of this preamble with respect to the feasibility of the proposed standard, and comments on that information are specifically solicited. Despite this information, and just in case a few mines experience technical problems that cannot be foreseen at this time, the proposed rule would make provision for a special extension to allow up to an additional two years to comply with the final concentration limit.

Extension Application. Proposed § 57.5060(c)(1) provides that if an operator of an underground metal or nonmetal mine can demonstrate that there is no combination of controls that can, due to technological constraints, be implemented within five years to reduce the concentration of dpm to the limit, MSHA may approve an application for an additional extension of time to comply with the dpm concentration limit. Under the proposal, such a special extension is available only once, and is limited to 2 years. To obtain a special extension, an operator must show that diesel powered equipment was used in the mine prior to publication of the rule, demonstrate that there is no off-the-shelf technology available to reduce dpm to the limit specified in § 57.5060, and establish the lowest achievable concentration of dpm attainable. The proposed rule further requires that to establish the lowest achievable concentration, the operator is to provide sampling data obtained using NIOSH Method 5040 (the method MSHA will use when determining concentrations for compliance purposes). The sampling method is further discussed in connection with proposed § 57.5061(a).

The application would also require the mine operator to specify the actions that are to be taken to ''maintain the lowest concentration of diesel particulate achievable'' (such as strict adherence to an established control plan) and to minimize miner exposure to dpm (e.g., provide suitable respirators). MSHA's intent is to ensure that personal protective equipment and administrative controls are permitted only as a last and temporary resort to bridge the gap between what can be accomplished with engineering and work practice controls and the concentration limit. It is not the Agency's intent that personal protective equipment or administrative controls be permitted during the extension period as a substitute for engineering and work practice controls that can be implemented immediately. The Agency would welcome comments on whether more explicit clarification of this point in the proposed rule is required.

Filing, Posting and Approval of Extension Application. The proposed rule would require that an application for an extension be filed (after being posted for 30 days at the mine site) no later than 6 months (180 days) in advance of the date of the final concentration limit (160tc $\mu g/m^3$). The proposed rule would also require that a copy of the approved extension be posted at the mine site for the duration of the extension period. In addition, a copy of the application would also have to be provided to the authorized representative of the miners.

The application would be required to be approved by MSHA before it becomes effective. While pre-approval of plans is not the norm in this sector, an exception to the final concentration limit cannot be provided without careful scrutiny. Moreover, in some cases, the examination of the application may enable MSHA to point out to the operator the availability of solutions not considered to date.

While the proposed rule is not explicit on the point, it is MSHA's intent that primary responsibility for approval of the operator's application for an extension will rest with MSHA's district managers. This ensures familiarity with the mine conditions, and provides an opportunity to consult with miners as well. At the same time, MSHA recognizes that district managers may not have the expertise required to keep fully abreast of the latest technologies and of solutions being used in similar mines elsewhere in the country. Accordingly, the Agency intends to establish, within its Technical Support directorate in Washington, D.C., a special panel to consult on these issues and to provide assistance to its district managers. MSHA would welcome comments on this matter, and as to whether it should incorporate further specifics in this regard into the final rule.

Personal Protective Equipment and Administrative Controls. Paragraph (d) provides that an operator shall not utilize personal protective equipment (e.g., respirators) or administrative controls (e.g., rotation of miners) to comply with either the interim or final concentration limit. Moreover, it provides that an operator shall not utilize administrative controls (e.g., the rotation of miners) to comply with either the interim or final concentration limit.

Limiting individual miner exposure through rotation or through the use of respirators would not reduce the airborne concentrations of particulate matter. It is accepted industrial hygiene practice to eliminate or minimize hazards at the source by using engineering or work practices, before resorting to alternative controls. Moreover, administrative controls are not considered acceptable in the case of potential carcinogens, since they result in placing more workers at risk.

MSHA intends that the normal meaning be given to the terms personal protective equipment and administrative controls, and welcomes comments as to whether more specificity would be useful. For example, the Agency assumes the mining community understands that an environmentally controlled cab for a piece of equipment is not a piece of personal protective equipment; indeed, the cost estimates for the proposed rule assume that such cabs will be a commonly used control to meet the proposed limits in those situations in which the only miners present in an area are equipment operators (see Part V of this preamble and the Agency's PREA).

Section 57.5061 Compliance Determinations

Under the proposed rule, compliance sampling would be performed by MSHA directly, and a single sample would be adequate to establish a violation.

The proposed rule further provides that MSHA will collect and analyze dpm samples for total carbon (TC) content using NIOSH Method 5040 (or by using any method subsequently determined by NIOSH to provide equal or improved accuracy in mines subject to this part). NIOSH Method 5040 provides for sample collection using a dust sampler pump and an open face filter. The filters are analyzed for elemental carbon (EC) and organic carbon (OC) content using the thermo-optical technique; the EC and OC concentration determinations are then added together to obtain the TC concentration of the sample.

Measurement Method for Compliance. Section 3 of Part II of this preamble discusses alternative methods for measuring dpm concentrations. As noted in that discussion, after considering the comments received in response to MSHA's ANPRM, reviewing the available technical information submitted in response to the ANPRM and reviewing the status of current technology, MSHA believes that NIOSH Method 5040 provides an accurate method of determining the total carbon content of a sample collected in any underground metal or nonmetal mine when using the sampling procedures specified in Method 5040. At the present time, Method 5040 is the only method that meets NIOSH's accuracy criterion for determinations of both EC and OC down to concentrations as low as those that will need to be measured to determine compliance with the final concentration limit being proposed. Accordingly, MSHA proposes to use this method for determining TC concentrations for compliance purposes.

Margin of Error. Before issuing a citation, MSHA intends to take into consideration uncertainty associated with the sampling and analytical process, as it does in other cases. While the measurement uncertainty has not been established for samples collected in mines, NIOSH has established the variability associated with Method 5040 to be approximately 6% (one relative standard deviation). If MSHA used the variability value established by NIOSH and allowed for a confidence level of 95%, MSHA would not issue a citation until the measured value was greater than 1.10 times the levels established in § 57.5060. For example, if the variability established by NIOSH is used, during the interim period when the limit is 400_{TC} $\mu g/m^3$ a noncompliance determination would not be made unless the TC measurement exceeded 440 $\mu g/m^3$.

MSHA recognizes that the measurement uncertainty may be higher for samples collected in mines, and intends to establish as the ''margin of error'' required to achieve a 95% confidence level for all noncompliance determinations based on samples collected in mines. The Agency anticipates that the margin of error will end up being somewhere between 10% and 20%, but will be governed by the actual data on this point.

Sampling Strategy. Proposed § 57.5060 would establish a concentration limit for areas of a mine where miners normally work or travel to limit miner exposure to dpm. In using this language, MSHA intends that the limits on the concentration of dpm would apply to persons, occupations or areas, as with coal dust. Accordingly, MSHA intends that inspectors have the flexibility to determine, on a mine by mine basis, the most appropriate method to assess the level of hazard that exists. The Agency may sample by attaching a sampler to an individual miner, or by locating the sampler on a piece of equipment where a miner may work, or at a fixed site where miners normally work or travel.

Sampling strategy was discussed by commenters who responded to the ANPRM. Several commenters indicated that the sampling strategy should ensure that samples taken are representative of actual exposure. Other commenters stated that the sampling strategy would be dictated by the measurement method, and that several strategies could be used to determine the hazard. They stated that the strategy should not be defined so narrowly as to exclude development of new sampling methods.

A related issue addressed by the commenters was whether personal or area sampling would be more appropriate. Most commenters indicated that personal sampling was the most reliable indicator of worker exposure. Some noted that in underground mines which use mobile diesel equipment, the positions of diesel-powered vehicles with respect to intake and return air streams vary from hour to hour. Therefore, it is virtually impossible to obtain meaningful information from stationary instruments. Several commenters stated that area sampling was appropriate to define action levels that may trigger personal sampling or to evaluate effectiveness of controls. Some additional concerns were raised concerning the accuracy of the sampling device when worn by a miner.

MSHA agrees that there may be circumstances when either area or personal sampling may be appropriate. Considering the mobility of the equipment it may not always be feasible to sample individual workers; for example, if work practice would include rotation of workers into an area. In this case, area sampling would be more appropriate to establish a hazard. MSHA does recognize that the diesel particulate is ultimately transported to return entries or exhaust openings of a mine.

The purpose of these entries is to provide a means to transport contaminated air away from the active workings. MSHA does not intend to conduct area sampling in these areas; however, personal sampling of workers who enter these areas could be conducted. These circumstances would be evaluated on a mine-by-mine basis during mine inspections. Accordingly, MSHA will utilize either area or personal (within 36'' of a miners breathing zone) sampling to determine whether corrective actions must be taken by a mine operator. In return entries, measurements made in the immediate area where diesel equipment is being operated will be collected at locations that are no closer than five feet from any piece of operating diesel equipment.

Section 57.5062 Diesel Particulate Matter Control Plan

A determination of noncompliance with either the interim or final concentration limit prescribed by § 57.5060 would trigger a requirement that: first, the operator establish a diesel particulate matter control plan (dpm control plan)— or modify the plan if one is already in effect; and second, the operator demonstrate that the new or modified plan is effective in controlling the concentration of dpm to the applicable concentration limit.

No Advance Approval Required. The agency proposes to continue to observe the metal and nonmetal mine plan tradition by not requiring a formal plan approval process. That is, the plan would not require advance approval of the MSHA District Manager. A dpm control plan would, however, have to meet certain requirements set forth in the proposed rule, and it would be a violation of § 57.5062 if MSHA determines the operator has failed to include the necessary particulars.

Elements of Plan. Under proposed § 57.5062(b), a dpm control plan must describe the controls the operator will utilize to maintain the concentration of diesel particulate matter to the applicable limit specified by § 57.5060. The plan must also include a list of diesel-powered units used by the mine operator, together with information about any unit's emission control device, and the parameters of any other methods used to control the concentration of diesel particulate matter.

Relationship to Ventilation Plan. At the discretion of the operator, the dpm control plan may be consolidated with the ventilation plan required by § 57.8520.

Demonstration of Plan Effectiveness. The proposed rule would require monitoring to verify that the dpm control plans are actually effective in reducing dpm concentrations in the mine to the applicable concentration limit. Because the dpm control plan was initiated as a result of a compliance action, the proposed rule would require the use of the same measurement method used by MSHA in compliance determinations—total carbon using NIOSH Method 5040—to conduct verification sampling.

Effectiveness must be demonstrated by ''sufficient'' monitoring to confirm that the plan or amended plan will control the concentration of diesel particulate to the applicable limit under conditions that can be ''reasonably anticipated'' in the mine. The proposed rule does not specify that any defined number of samples must be taken—the intent is that the sampling provide a fair picture of whether the plan or amended plan is working. MSHA will determine compliance with this obligation based on a review of the situation involved. While an MSHA compliance sample may be an indicator that the operator has not fulfilled their obligation under this section to undertake monitoring ''sufficient'' to verify plan effectiveness, it would be inconclusive on that point. The Agency welcomes comment on this point.

Similarly, the Agency welcomes comment on whether, and how, it should define the term ''reasonably anticipated.'' With respect to coal dust, the Dust Advisory Committee recommended that ''MSHA should define the range of production values which must be maintained during sampling to verify the plan. This value should be sufficiently close to maximum anticipated production'' (MSHA, 1996). For dpm, the equivalent approach might be based on worst-case operating conditions of the diesel equipment—e.g., all equipment is being operated simultaneously with the least ventilation.

Recordkeeping Retention and Access. Pursuant to § 57.5062(b), a copy of the current dpm control plan is to be maintained at the mine site during the duration of the plan and for one year thereafter. Proposed § 57.5062(c) would require that verification sample results be retained for 5 years. Proposed § 57.5062(d) provides that both the control plan and sampling records verifying effectiveness be made available for review, upon request, by the authorized representative of the Secretary, the Secretary of Health and Human Services, and/or the authorized representative of miners. Upon request of the District Manager or the authorized representative of miners, a copy of these records is to be provided by the operator.

Duration. The proposal would require the dpm control plan to remain in effect for three years from the date of the violation resulting in the establishment/modification of the plan. As discussed in Part I of this preamble (Question and Answer 18), MSHA believes operators have sufficient time under the proposed rule to come into compliance with the concentration limits. If a problem exists, maintaining a plan in effect long enough to ensure that daily mine practices really change, is an important safeguard.

Modification During Plan Lifetime. A violation of § 57.5060 would require the mine operator to modify the dpm control plan to reflect changes in mining equipment and/or the mine environment and the operator would be required to demonstrate to MSHA the effectiveness of the modified plan.

Also, proposed § 57.5062(e)(2) would require the mine operator to modify the dpm control plan to reflect changes in mining equipment and/or the mine environment and the operator would be required to demonstrate to MSHA the effectiveness of the modified plan.

Compliance with Plan Requirements. Once an underground metal or nonmetal mine operator adopts a dpm control plan, it will be considered regulation for the mine. Proposed 57.5062(f) specifically provides that MSHA would not need to establish (by sampling) that an operator is currently in violation of the applicable concentration limit under § 57.5060 in order to determine by observation that an operator has failed to comply with any requirement of the mine's dpm control plan.

Section 57.5065 Fueling and idling practices

Fueling Practices. Part II of this preamble contains some background information on fueling practices, together with information about the rules currently applicable in underground coal mines.

Proposed § 57.5065(a) would require underground metal and nonmetal mine operators to use only low-sulfur fuel having a sulfur content of no greater than 0.05 percent. This requirement is identical to that currently required for diesel equipment used in underground coal mines [30 CFR 75.1901(a)]. Both number 1 and number 2 diesel fuel meet the requirement of this proposal.

Sulfur content can have a significant effect on diesel emissions. Use of low sulfur diesel fuel reduces the sulfate fraction of dpm emissions, reduces objectionable odors associated with diesel exhaust, and allows oxidation catalysts to perform properly. A major benefit of using low sulfur fuel is that the reduction of sulfur allows for the use of some aftertreatment devices such as catalytic converters and catalyzed particulate traps which were prohibited with fuels of high sulfur content (greater than 0.05 percent sulfur). MSHA believes the use of these aftertreatment devices is important to the mining industry because they will be necessary to meet the levels specified. The requirement to use low sulfur fuel will allow these devices to be used without additional adverse effects caused by the high sulfur fuel. As noted in Part IV of the PREA, MSHA does not believe such a requirement will add additional cost.

Proposed paragraph (b) of this section would require mine operators to use only diesel fuel additives that have been registered by the Environmental Protection Agency (40 CFR Part 79). Again, this proposed rule is consistent with that currently required for diesel equipment used in underground coal mines [30 CFR 75.1901(c)]. The restricted use of additives would ensure that diesel particulate concentrations would not be inadvertently increased, while also protecting miners against the emission of other toxic contaminants. MSHA issued Program Information Bulletin No. P97–10, on May 5, 1997, that discusses the fuel additives list. The requirements of this paragraph do not place an undue burden on mine operators because operators need only verify with their fuel suppliers or distributors that the additive purchased is included on the EPA registration list.

Idling Practices. Proposed § 57.5065(c) would prohibit idling of mobile-powered diesel equipment, except as required for normal mining operations. The idling requirements being proposed for underground metal and nonmetal mines are consistent with the idling requirements currently required for underground coal mines (§ 75.1916(d)).

MSHA believes that keeping idling to a minimum is very important to reduce pollution in mine atmospheres. Engines operating without a load during idling can produce significant levels of both gaseous and particulate emissions. Even though the concentration emitted from a single idling engine might have little effect on the overall mine environment, a localized, increased exposure of the gaseous and particulate concentrations would occur. In underground operations, an engine idling in an area of minimal ventilation or a "dead air" space could cause an excess exposure to the gaseous emissions, especially carbon monoxide, as well as to dpm. Eliminating unnecessary idling would reduce localized exposure to high particulate concentrations.

While the proposed rule is intended to prevent idling except as required for normal mining operations, it does not define normal mining operations. MSHA envisions "normal mining operations" to be activities such as idling while waiting for a load to be unhooked, or waiting in line to pick up a load. These types of activities would be permitted. Idling while eating lunch is normally not part of the job and operators would be in violation of the standard. Idling necessary due to very cold weather conditions would be permitted. On the other hand, idling in other weather conditions just to keep balky, older engines running would not be permitted; in such cases, the correct approach is better maintenance. MSHA welcomes comments on whether a more specific definition is necessary, particularly in light of any experience to date under the parallel rule for diesel equipment in underground coal mines.

Section 57.5066 Maintenance Standards

Proposed § 57.5066(a) would place emphasis on the fact that diesel engine emissions are lower from an engine that is properly maintained than from an engine that is not. Part II of the preamble provides more information on this point.

Approved Engines. Proposed § 57.5066(a)(1) would require that mine operators maintain any approved diesel engine in "approved" condition. Under MSHA's approval requirements, engine approval is tied to the use of certain parts and engine specifications. When these parts or specifications are changed (i.e., an incorrect part is used, or the engine timing is incorrectly set), the engine is no longer considered by MSHA to be in approved condition.

Often, engine exhaust emissions will deteriorate when this occurs. Maintaining approved engines in their approved condition will ensure near-original performance of an engine, and maximize vehicle productivity and engine life, while keeping exhaust emissions at approved levels. The proposed maintenance requirements for approved engines in this rule are already applicable to underground coal mines, where only approved engines may be utilized (30 CFR 75.1914).

Thus in practice, with respect to approved engines, mine maintenance personnel will have to maintain the following engine systems in near original condition: air intake, cooling, lubrication, fuel injection and exhaust. These systems must be maintained on a regularly scheduled basis to keep the system in its "approved" condition and thus, operating at its expected efficiency.

One of the best ways to ensure these standards are observed is to implement a proper maintenance program in the mine—but the proposed rule would not require operators to do this. A good program should include compliance with manufacturers' recommended maintenance schedules, maintenance of accurate records and the use of proper maintenance procedures. MSHA's diesel toolbox provides more information about the practices that should be followed in maintaining diesel engines in mines.

Non-approved Engines. For any non-approved diesel engine, proposed paragraph (a)(2) would require mine operators to maintain the emissions related components to manufacturer specifications.

The term ''emission related components,'' refers to the parts of the engine that directly affect the emission characteristics of the raw exhaust. These are basically the same components which MSHA examines for ''approved'' engines. They are the piston, intake and exhaust valves, cylinder head, injector, fuel injection pump, governor, turbocharger, after cooler, injection timing, and fuel pump calibrator.

It is not MSHA's intent that engines be torn down and the engine components be compared against the specifications in manufacturer maintenance manuals. Primarily, the Agency is interested in ensuring that engines are maintained in accordance with the schedule recommended by the manufacturer. However, if it becomes evident that the engines are not being maintained to the correct specifications or are being rebuilt in a configuration not in line with manufacturers' specifications or approval requirements, an inspector may ask to see the manuals to confirm that the right manuals are being used, or call in MSHA experts to examine an engine to confirm whether basic specifications are being properly observed. MSHA welcomes comment on alternative ways to phrase this requirement so Agency has a basis for ensuring compliance while minimizing the opportunity for over-prescriptiveness.

Emission or Particulate Control Device. Proposed paragraph (a)(3) would require that any emission or particulate control device installed on diesel-powered equipment be maintained in effective operating condition. Depending on the type of devices installed on an engine, this would involve having trained personnel perform such basic tasks as regularly cleaning aftertreatment filters, using methods recommended by the manufacturer for that purpose, or inserting appropriate replacement filters when required, checking for and repairing any exhaust system leaks, and other appropriate actions.

Tagging of Equipment for Noncompliance. Proposed § 57.5066(b)(1) would require underground metal and nonmetal mine operators to authorize and require miners operating diesel powered equipment to affix a visible and dated tag to the equipment at any time the equipment operator detects an emission-related problem.

MSHA believes tagging will provide an effective and efficient method of alerting all mine personnel that a piece of equipment needs to be checked by qualified service personnel. The tag may be affixed because the equipment operator detects a problem through a visual exam conducted before the equipment is started, or because of a problem that comes to the attention of the equipment operator during mining operations, (i.e., black smoke while the equipment is under normal load, rough idling, unusual noises, backfiring, etc.)

MSHA is not proposing that equipment tagged for potential emission problems be automatically taken out of service. The proposal is not, therefore, directly comparable to a ''tag-out'' requirement like OSHA's requirement for automatic powered machinery, nor is it as stringent as MSHA's requirement to remove from service certain equipment ''when defects make continued operation hazardous to persons'' (see 30 CFR 57.14100). The proposed rule is not as stringent as these requirements because, although exposure to dpm emissions does pose a serious health hazard for miners, the existence or scope of an equipment problem cannot be determined until the equipment is examined or tested by a person competent to assess the situation. Moreover, the danger is not as immediate as, for example, an explosive hazard.

Proposed § 57.5066(b)(2) would require that the equipment be ''promptly'' examined by a person authorized by the mine operator to maintain diesel equipment. (The qualifications for those who maintain and service diesel engines are discussed below). The Agency has not tried to define the term ''promptly,'' but welcomes comment on whether it should do so—in terms, for example, of a limited number of shifts. The presence of a tag serves as a caution sign to miners working on or near the equipment, as well as a reminder to mine management, as the equipment moves from task to task throughout the mine. While the equipment is not barred from service, operators would be expected to use common sense and not use it in locations in which diesel particulate concentrations are known to be high.

Proposed paragraph (b)(2) would permit a tag to be removed after the defective equipment has been examined.

The design of the tag is left to the discretion of the mine operator, with the exception that the tag must be able to be marked with a date. Comments are welcome on whether some or all elements of the tag should be standardized to ensure its purpose is met.

Tagged Equipment Log. Proposed § 57.5066(b)(3) would require a log to be retained of all equipment tagged. Moreover, the log must include the date the equipment is tagged, the date the tagged equipment is examined, the name of the person making the examination, and the action taken as a result of the examination. Records in the log about a particular incident must be retained for at least a year after the equipment is tagged.

MSHA does not expect the log to be burdensome to the mine operator or mechanic examining or testing the engine. Based on MSHA's experience, it is common practice to maintain a log when equipment is serviced or repaired, consistent with any good maintenance program. The records of the tagging and servicing, although basic, provide mine operators, miners and MSHA with a history that will help in determining whether a maintenance program is being effectively implemented.

Qualified Person. Proposed paragraph (c) would require that persons who maintain diesel equipment in underground metal and nonmetal mines be ''qualified,'' by virtue of training and experience, to ensure the maintenance standards of proposed § 57.5066(a) are observed. Paragraph (c) also requires that an operator retain appropriate evidence of ''the competence of any person to perform specific maintenance tasks'' in compliance with the requirement's maintenance standards for one year.

The ANPRM requested information concerning specialized training for those persons working on equipment that uses particulate reduction technology and the costs associated with the training. Commenters stated that any equipment modifications will require additional training. The extent and costs would vary widely depending on the type of devices used. MSHA agrees that training should be given when new devices or modifications to machines are made. The training cost will be dependent on the complexity of the control device.

Operators of underground coal mines where diesel-powered equipment is used are required, as of November 25, 1997, to establish programs to ensure that persons who perform maintenance, tests, examinations and repairs on diesel-powered equipment are qualified (30 CFR 75.1915). The unique conditions in underground coal mines require the use of specialized equipment. Accordingly, the qualifications of the persons who maintain this equipment generally must be appropriately sophisticated.

If repairs and adjustments to diesel engines used in underground metal and nonmetal mines are to be done properly, personnel performing such tasks must be properly trained. MSHA does not believe, however, that the qualifications required to perform this work in underground metal and nonmetal mines necessarily require the same level of training as for similar work in underground coal mines. Under the proposed rule, the training required would be that which is commensurate with the maintenance task involved. If examining and, if necessary, changing a filter or air cleaner is all that is required, a miner who has been shown how to do these tasks would be qualified by virtue of training or experience to do those tasks. For more detailed work, specialized training or additional experience would be required. Training by a manufacturer's representative, completion of a general diesel engine maintenance course, or practical experience performing such repairs could also serve as evidence of having the qualifications to perform the service.

In practice, the results will soon be revealed by performance. If MSHA finds a situation where maintenance appears to be shoddy, where the log indicates an engine has been in for repair with more frequency than should be required, or where repairs have damaged engine approval status or emission control effectiveness, MSHA would ask the operator to provide evidence that the person(s) who worked on the equipment was properly qualified by virtue of training or experience.

It is MSHA's intent that equipment sent off-site for maintenance and repair is also subject to the requirement that the personnel performing the repair be qualified by virtue of training or experience for the task involved. It is not MSHA's intent that a mine operator have to examine the training and experience record of off-site mechanics, but a mine operator will be expected to observe the same kind of caution as one would observe with a personal vehicle—e.g., selecting the proper kind of shop for the nature of the work involved, and considering prior direct experience with the quality of the shop's work.

Section 57.5067 Engines

The proposed rule would require that, with the exception of diesel engines used in ambulances and fire-fighting equipment, any diesel engines added to the fleet of an underground metal or nonmetal mine in the future must be an engine approved by MSHA under Part 7 or Part 36. This requirement would take effect 60 days after the date the rule is promulgated.

The composition of the existing fleet would not be impacted by this part of the proposed rule. However, after the rule's effective date, an operator would not be permitted to bring into underground areas of a mine an unapproved engine from the surface area of the same mine, an area of another mine, or from a non-mining operation. Promoting a gradual turnover of the existing fleet to better engines is an appropriate response to the health risk presented by dpm.

Approval is not something that has to be done by individual mine operators. Approved engines carry an approval plate so they are easy to distinguish. Approval is a process that is handled by engine manufacturers, involving tests by independent laboratories.

MSHA is assuming in the PREA accompanying this proposed rule that this additional requirement will require manufacturers to obtain approval on one additional diesel engine model per year. Some engines currently used in metal and nonmetal mines may have no approval criteria; in such cases, MSHA will work with the manufacturers to develop approval criteria consistent with those MSHA uses for other diesel engines. Based upon preliminary analysis, MSHA has tentatively concluded that any diesel engine meeting current on-highway and non-road EPA emission requirements would meet MSHA's engine approval standards of Part 7, subpart E, category B type engine. (See section 4 of Part II of this preamble for further information about these engines.)

Currently, the EPA non-road test cycle and MSHA's test cycle are the same for determining the gaseous and particulate emissions. MSHA envisions being able to use the EPA test data for engines run on the non-road test cycle for determining the gaseous ventilation rate and particulate index. The engine manufacturer would continue to submit the proper paper work for a specific model diesel engine to receive the MSHA approval. However, engine data run on the EPA on-highway transient test cycle would not as easily be usable to determine the gaseous ventilation and particulate index. Comments on how MSHA can facilitate review of engines not currently approved would be welcome.

Engines in diesel-powered ambulances and fire-fighting equipment would be exempted from these requirements. This exemption is identical with that in the rule for diesel-powered equipment in underground coal mines.

Section 57.5070 Miner Training

Proposed § 57.5070 would require any miner ''who can reasonably be expected to be exposed to diesel emissions'' be trained annually in: (a) The health risks associated with dpm exposure; (b) the methods used in the mine to control dpm concentrations; (c) identification of the personnel responsible for maintaining those controls; and (d) actions miners must take to ensure the controls operate as intended.

The purpose of the proposed requirement is to promote miner awareness. Exposure to diesel particulate is associated with a number of harmful effects as discussed in Part III of this preamble, and the safe level is unknown. Miners who work in mines where they are exposed to this risk ought to be reminded of the hazard often enough to make them active and committed partners in implementing actions that will reduce that risk.

The training need only be provided to miners who can reasonably be expected to be exposed at the mine. The training is to be provided by operators; hence, it is to be without fee to the miner.

The rule places no constraints on the operator as to how to accomplish this training. MSHA believes that the required training can be provided at minimal cost and minimal disruption. The proposal would not require any special qualifications for instructors, nor would it specify the hours of instruction.

Instruction could take place at safety meetings before the shift begins. Devoting one of those meetings to the topic of dpm would be a very easy way to convey the necessary information. Simply providing miners with a copy of MSHA's ''Toolbox'' and, a copy of the plan, if a control plan is in effect for the mine, and reviewing these documents, can cover several of the training requirements. One-on-one discussions that cover the required topics are another approach that can be used.

Operators could also choose to include a discussion on diesel emissions in their Part 48 training, provided the plan is approved by MSHA. There is no existing requirement that Part 48 training include a discussion of the hazards and control of diesel emissions. While mine operators are free to cover additional topics during the Part 48 training sessions, the topics that must be covered during the required time frame may make it impracticable to cover other matters within the prescribed time limits.

Where the time is available in mines using diesel-powered equipment, operators would be free to include the dpm instruction in their Part 48 training plans. The Agency does not believe special language in the proposed rule is required to permit this action under Part 48, but welcomes comment in this regard.

The proposal does not require the mine operator to separately certify the completion of the dpm training, but some evidence that the training took place would have to be produced upon request. A serial log with the employee's signature is an acceptable practice.

To assist mine operators with the proposed training requirement, it is MSHA's intent to develop an instruction outline that mine operators can use as a guide for training personnel. Instruction materials will be provided with the outline.

Section 57.5071 Environmental Monitoring

Operator's Monitoring Responsibility. Proposed § 57.5071(a) would require that mine operators sample their mine environments to evaluate environmental conditions to which miners are exposed. It is proposed that sampling be performed as often as necessary to ''effectively evaluate''—under conditions that can be reasonably anticipated in the mine—(1) Whether the dpm concentration in any area of the mine where miners normally work or travel exceeds the applicable limit; and (2) the average full shift airborne concentration at any position or on any person designated by the Secretary.

There are two important aspects of this proposed operator monitoring requirement. First, it would clarify that it is the responsibility of mine operators to be aware of the concentrations of dpm in all areas of the mine where miners normally work or travel, so as to know whether action is needed to ensure that the concentration is kept below the applicable limit. Secondly, this requirement would ensure special attention to locations or persons known to MSHA to have a significant potential for overexposure to dpm.

The obligation of operators to ''effectively evaluate'' concentrations in a mine is a separate obligation from that to keep dpm levels below the established limit, and can be the basis of a separate citation from MSHA. The proposed rule is performance-oriented in that the regularity and methodology used to make this evaluation are not specified. However, MSHA expects mine operators to sample with such frequency that they and the miners working at the mine site are aware of dpm levels in their work environment. In this regard, MSHA's own measurements will assist the Agency in verifying the effectiveness of an operator's monitoring program. If an operator is ''effectively evaluating'' the concentration of dpm at designated positions, for example, MSHA would not expect to regularly record concentrations above the limit when it samples at that location. If MSHA does find such a problem, it will investigate to determine how frequently an operator is sampling, where the operator is sampling, and what methodology is being used, so as to determine whether the obligation in this section is being fulfilled.

MSHA proposed a performance-oriented operator sampling requirement in its recent proposed rule on noise, and is seeking some consistency of approach in this regard for uniform health standards.

Operator Monitoring Methods. The proposed rule requires that full-shift diesel particulate concentrations be determined during periods of normal production or normal work activity, in areas where miners work or travel. The proposed rule does not specify a particular monitoring method or frequency; rather, the proposal is performance-oriented. Operators may, at their discretion, conduct their monitoring using the same sampling and analytical method as MSHA, or they may use any other method that enables that mine to ''effectively evaluate'' the concentrations of dpm. Monitoring performed to verify the effectiveness of a diesel particulate control plan would probably meet the obligation under proposed § 57.5071 if it is done with enough sufficiency to meet the obligation under proposed § 7.5062(c).

As discussed in connection with proposed § 57.5061, MSHA intends to use NIOSH Method 5040, the sampling and analytical method that NIOSH has developed for accurately determining the concentration of total carbon. Operators are also required to use the TC method for verifying the effectiveness of dpm control plans, as discussed in connection with proposed § 57.5062. But the method may not be necessary to effectively evaluate dpm in some mines. For example, dpm measurements in limestone, potash and salt mines could be determined using the RCD method, since there are no large carbonaceous particles present that would interfere with the analysis. Such estimates can be useful in determining the effectiveness of controls and where more refined measurements may be required.

Of course, mine operators using the RCD, or size-selective methods, to monitor their diesel particulate concentrations would have to convert the results to a TC equivalent to ascertain their exact compliance status. At the present time, MSHA has no conversion tables for this purpose. In most cases, the other methods will provide a good indication of whether controls are working and whether further action is required.

Part II of this preamble provides information on monitoring methods and their constraints, and on laboratory and sampler availability.

Observation of Monitoring. Section 103(c) of the Mine Act requires that:

> The Secretary, in cooperation with the Secretary of Health, Education, and Welfare, shall issue regulations requiring operators to maintain accurate records of employee exposures to potentially toxic materials or harmful physical agents which are required to be monitored or measured under any applicable mandatory health or safety standard promulgated under this Act. Such regulations shall provide miners or their representatives with an opportunity to observe such monitoring or measuring, and to have access to the records thereof.

In accordance with this legal requirement, proposed § 57.5071(b) requires a mining operator to provide affected miners and their representatives with an opportunity to observe exposure monitoring required by this section. Mine operators must give prior notice to affected miners and their representatives of the date and time of intended monitoring.

MSHA has proposed identical language in a supplement to its proposed rule on noise (62 FR 68468).

Corrective Action if Concentration is Exceeded. Proposed § 57.5071(c) provides that if any monitoring performed under this section indicates that the applicable dpm concentration limit has been exceeded, an operator shall initiate corrective action by the next work shift, promptly post a notice of the corrective action being taken and promptly complete such corrective action.

MSHA welcomes comments as to what guidance to provide with respect to the obligations in this regard where an operator is not using the total carbon method. MSHA also welcomes comment as to whether personal notice of corrective action would be more appropriate than posting, given the health risks involved.

The Agency wishes to emphasize that operator monitoring of dpm concentrations would not take the place of MSHA sampling for compliance purposes; rather, this requirement is designed to ensure the operator checks dpm concentrations on a more regular basis than it is possible for MSHA to do.

Proposed paragraph (c) provides that if sampling results indicate the concentration limit has been exceeded in an area of a mine, an operator would initiate corrective action by the next work shift and promptly complete such action.

In certain types of cases (e.g., 30 CFR 75.323), MSHA has required that when monitoring detects a hazardous level of a substance, miners must be immediately withdrawn from an area until abatement action has been completed. Although MSHA has not proposed such action in this case, MSHA would like advice from the mining community on whether such a practice should be required in light of the evidence presented on the various risks posed by exposure to diesel particulate. There is good evidence, for example, that acute short-term increases in exposure can pose significant risks to miner health.

The Agency welcomes comment on whether clarification of this proposed requirement is necessary in light of the fact that operators using more complex analytical procedures (e.g., the total carbon method) may not receive the results for some time period after the sampling has taken place.

Posting of Sample Results. Proposed § 57.5071(d)(1) would require that monitoring results be posted on the mine bulletin board within 15 days of receipt, and remain posted for 30 days. A copy of the results would be provided to the authorized miners' representative. Posting of the results would ensure that miners are kept aware of the hazard so they can actively participate in efforts to control dpm.

Retention of Sample Results. Proposed § 57.5071(d)(2) would require that records of the sampling method and the sample results themselves be retained by operators for five years. This is because the results from a monitoring program can provide insight as to the effectiveness of controls over time and provide a history of occupational exposures at the mine. MSHA would welcome comment on the sample retention period appropriate for the risks involved.

Section 57.5075 Diesel Particulate Records

Various recordkeeping requirements are set forth in provisions of the proposed rule. For the convenience of the mining community, these requirements are also listed in a table entitled ''Diesel Particulate Recordkeeping Requirements,'' which can be found in proposed § 57.5075(a). Each row involves a record that must be kept. The section requiring the record be kept is noted, along with the retention time. MSHA would welcome input from the mining community as to whether it likes this approach or finds it duplicative or confusing.

Location of Records. Proposed § 57.5075(b)(1) would provide that any record which is required to be retained at the mine site may be retained elsewhere if it is immediately accessible from the mine site by electronic transmission. Compliance records need to be where an inspector can view them during the course of an inspection, as the information in the records may determine how the inspection proceeds. If the mine site has a fax machine or computer terminal, there is no reason why the records cannot be maintained elsewhere. MSHA's approach in this regard is consistent with Office of Management and Budget Circular A–130.

MSHA encourages mine operators who store records electronically to provide a mechanism which will allow the continued storage and retrieval of records in the year 2000.

Records Access. Proposed § 57.5075(b) also covers records access. Consistent with the statute, upon request from an authorized representative of the Secretary of Labor, the Secretary of Health and Human Services, or from the authorized representative of miners, mine operators are to promptly provide access to any record listed in the table in this section. A miner, former miner, or, with the miner's or former miner's written consent, a personal representative of a miner, is to have access to any exposure record required to be maintained pursuant to § 57.5071 to the extent the information pertains to the miner or former miner. Upon request, the operator must provide the first copy of such record at no cost. Whenever an operator ceases to do business, that operator would be required to transfer all records required to be maintained by this part to any successor operator.

General Effective Date. The proposed rule provides that unless otherwise specified, its provisions take effect 60 days after the date of promulgation of the final rule. Thus, for example, the requirements to implement certain work practice controls (e.g., fuel type) would go into effect 60 days after the final rule is published.

A number of provisions of the proposed rules contain separate effective dates that provide more time for technical support. For example, the initial concentration limit for underground metal and nonmetal mines would be delayed for 18 months.

A general outline of effective dates is contained in Question and Answer 10 in Part I of this preamble.

V. Adequacy of Protection and Feasibility of Proposed Rule

The Mine Act requires that in promulgating a standard, the Secretary, based on the best available evidence, shall attain the highest degree of health and safety protection for the miner with feasibility a consideration.

Overview

This part begins with a summary of the pertinent legal requirements, followed by a general profile of the economic health and prospects of the metal and nonmetal mining industry.

The discussion then turns to the proposed rule for underground metal and nonmetal mines. MSHA is proposing to establish a concentration limit for dpm, supplemented by monitoring and training requirements. An operator in the metal and nonmetal sector would have the flexibility to choose any type or combination of engineering controls to keep dpm levels at or below the concentration limit. In addition, the proposed rule would require this sector to implement certain work practices that help reduce dpm concentrations—practices similar to those already required in the underground coal mining industry. Miner hazard awareness training would also be required.

This part evaluates the proposed rule for underground metal and nonmetal mines to ascertain if, as required by the statute, it achieves the highest degree of protection for underground metal and nonmetal miners that is feasible, both technologically and economically, for underground metal and nonmetal mine operators to provide. Some significant alternatives to the proposed rule were also reviewed in this regard—for example, reducing the concentration limit or the time permitted to come into compliance with the limit. Based on the best evidence available to MSHA at this time, the Agency has tentatively concluded that the proposed rule for the underground metal and nonmetal sector meets the statutory requirements. The Agency has also tentatively concluded that the alternatives considered are not feasible for underground metal and nonmetal mine operators as a whole—for technological reasons, economic reasons, or both.

An Appendix to this part provides additional information about an approach to simulating the dpm reduction in mines that can be achieved with various types of controls. Some simulations using this model were among the facts considered by MSHA in reaching its tentative conclusions about the feasible concentration limit in underground metal and nonmetal mines.

Pertinent Legal Requirements

Section 101(a)(6)(A) of the Federal Mine Safety and Health Act of 1977 (Mine Act) states that MSHA's promulgation of health standards must:

> * * * [A]dequately assure, on the basis of the best available evidence, that no miner will suffer material impairment of health or functional capacity even if such miner has regular exposure to the hazards dealt with by such standard for the period of his working life.

The Mine Act also specifies that the Secretary of Labor (Secretary), in promulgating mandatory standards pertaining to toxic materials or harmful physical agents, base such standards upon:

> * * * [R]esearch, demonstrations, experiments, and such other information as may be appropriate. In addition to the attainment of the highest degree of health and safety protection for the miner, other considerations shall be the latest available scientific data in the field, the feasibility of the standards, and experience gained under this and other health and safety laws. Whenever practicable, the mandatory health or safety standard promulgated shall be expressed in terms of objective criteria and of the performance desired. [Section 101(a)(6)(A)].

Thus, the Mine Act requires that the Secretary, in promulgating a standard, based on the best available evidence, attain the highest degree of health and safety protection for the miner with feasibility a consideration.

In relation to feasibility, the legislative history of the Mine Act states that:

> * * * This section further provides that ''other considerations'' in the setting of health standards are ''the latest available scientific data in the field, the feasibility of the standards, and experience gained under this and other health and safety laws.'' While feasibility of the standard may be taken into consideration with respect to engineering controls, this factor should have a substantially less significant role. Thus, the Secretary may appropriately consider the state of the engineering art in industry at the time the standard is promulgated. However, as the circuit courts of appeal have recognized, occupational safety and health statutes should be viewed as ''technology-forcing'' legislation, and a proposed health standard should not be rejected as infeasible when the necessary technology looms in today's horizon. *AFL-CIO* v. *Brennan,* 530 F.2d 109 (1975); *Society of the Plastics Industry* v. *OSHA,* 509 F.2d 1301, *cert. denied,* 427 U.S. 992 (1975).
>
> Similarly, information on the economic impact of a health standard which is provided to the Secretary of Labor at a hearing or during the public comment period, may be given weight by the Secretary. In adopting the language of [this section], the Committee wishes to emphasize that it rejects the view that cost benefit ratios alone may be the basis for depriving miners of the health protection which the law was intended to insure. S. Rep. No. 95–181, 95th Cong., 1st Sess. 21 (1977).

Court decisions have clarified the meaning of feasibility. The Supreme Court, in *American Textile Manufacturers' Institute* v. *Donovan* (OSHA Cotton Dust), 452 U.S. 490, 101 S. Ct. 2478 (1981), defined the word ''feasible'' as ''capable of being done, executed, or effected.'' The Court stated that a standard would not be considered economically feasible if an entire industry's competitive structure was threatened. According to the Court, the appropriate inquiry into a standard's economic feasibility is whether the standard is capable of being achieved.

Courts do not expect hard and precise predictions from agencies regarding feasibility. Congress intended for the ''arbitrary and capricious standard'' to be applied in judicial review of MSHA rulemaking (S.Rep. No. 95–181, at 21.) Under this standard, MSHA need only base its predictions on reasonable inferences drawn from the existing facts. MSHA is required to produce reasonable assessment of the likely range of costs that a new standard will have on an industry. The agency must also show that a reasonable probability exists that the typical firm in an industry will be able to develop and install controls that will meet the standard. See, *Citizens to Preserve Overton Park* v. *Volpe,* 401 U.S. 402, 91 S. Ct. 814 (1971); *Baltimore Gas & Electric Co.* v. *NRDC,* 462 U.S. 87 103 S. Ct. 2246, (1983); *Motor Vehicle Manufacturers Assn.* v. *State Farm Mutual Automobile Insurance Co.,* 463 U.S. 29, 103 S. Ct. 2856 (1983); *International Ladies' Garment Workers' Union* v. *Donovan,* 722 F.2d 795, 232 U.S. App. D.C. 309 (1983), *cert. denied,* 469 U.S. 820 (1984); *Bowen* v. *American Hospital Assn.,* 476 U.S. 610, 106 S. Ct. 2101 (1986).

In developing a health standard, MSHA must show that modern technology has at least conceived some industrial strategies or devices that are likely to be capable of meeting the standard, and which industry is generally capable of adopting. *United Steelworkers of America* v. *Marshall,* 647 F.2d 1189, (D.C. Cir. 1980) at 1272. If only the most technologically advanced companies in an industry are capable of meeting the standard, then that would be sufficient demonstration of feasibility (this would be true even if only some of the operations met the standard for some of the time). *American Iron and Steel Institute* v. *OSHA,* 577 F. 2d 825, (3d Cir. 1978); see also, *Industrial Union Department, AFL-CIO* v. *Hodgson,* 499 F. 2d 467 (1974).

Industry profile. The industry profile provides background information describing the structure and economic characteristics of the metal and nonmetal mining industry. This information was considered by MSHA as appropriate in reaching tentative conclusions about the economic feasibility of various regulatory alternatives. MSHA welcomes the submission of additional economic information about the metal and nonmetal mining industry, and about underground mining in particular, that will help it make final determinations about the economic feasibility of the proposed rule.

This profile provides data on the number of mines, their size, the number of employees in each segment, as well as selected market characteristics. It does not provide information about the use of diesel engines in the industry; information in that regard was provided in the first section of part II of this preamble.

Overall mining industry. MSHA divides the mining industry into two major segments based on commodity: The coal industry and the metal and nonmetal (M/NM) mining industry. These major industry segments are further divided based on type of operations (underground mines, surface mines, and independent mills, plants, shops, and yards). MSHA maintains its own data on mine type, size, and employment. MSHA also collects data on the number of contractors and contractor employees.

MSHA categorizes mines as to size based on employment. Over the past 20 years, for rulemaking purposes, MSHA has consistently defined small mines to be those having fewer than 20 employees and large mines to be those having at least 20 employees. For this Preliminary Regulatory Economic Analysis and Initial Regulatory Flexibility Analysis, MSHA will continue to use this small mine definition. However, for the purposes of the Small Business Regulatory Enforcement Fairness Act (SBREFA) amendments to the Regulatory Flexibility Act (RFA), MSHA has also included SBA's definition of small (500 or fewer employees) in the evaluation of impacts.

Table V–1 presents the number of small and large M/NM mines and the corresponding number of miners, excluding contractors, by major industry segment and mine type. Table V–1 uses three size classes: Less than 20 employees (MSHA's definition of small), 20 to 500 employees (also small by SBA's definition, but not by MSHA's), and over 500 employees. Table V–2 presents similar MSHA data on the numbers of independent contractors and the corresponding numbers of employees by the size of the operation, based on employment. Table V–3 shows numbers of M/NM mines and workers by class of commodity produced.

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Table V-1: Distribution of Operations and Employment (excluding contractors) by Mine Type and Size

Mine Type	Size of M/NM Mine						All M/NM Mines	
	Less than 20 Employees		20 to 500* Employees		Over 500* Employees			
	Mines	Miners	Mines	Miners	Mines	Miners	Mines	Miners
Under-ground	130	1,103	124	10,152	7	6,531	261	17,786
Surface	8,781	48,924	1,175	63,753	18	16,723	9,974	129,400
Shop/Yd/ Mill/Plt	284	2,195	212	15,792	4	2,584	500	20,571
Office Workers	-	8,422	-	16,244	-	2,389	-	27,055
Total M/NM	9,195	60,644	1,511	105,941	29	28,227	10,735	194,812

(*) Based on MSHA's traditional definition, large mines include all mines with employees of 20 or greater.

Source: U.S. Department of Labor, Mine Safety and Health Administration, Office of Standards, Regulations, and Variances, based on preliminary 1996 MIS data (quarter 1 - quarter 4, 1996).

Table V-2: Distribution of Contractors and Contractor Employment by Size of Operation

Contractors	Size of Contractor						All Contractors	
	Less than 20 Employees		20 to 500* Employees		Over 500* Employees			
	Mines	Miners	Mines	Miners	Mines	Miners	Mines	Miners
Firms	2,621	13,058	340	18,810	1	897	2,962	32,765
Office Workers	-	691	-	902	-	140	-	1,733
Total Contractors	2,621	13,749	340	19,712	1	1,037	2,962	34,498

(*) Based on MSHA's traditional definition, large contractors include all contractors with employees of 20 or greater.

Source: U.S. Department of Labor, Mine Safety and Health Administration, Office of Standards, Regulations, and Variances, based on preliminary 1996 MIS data (quarter 1 - quarter 4, 1996).

Table V-3: Estimated Distribution of Metal and Nonmetal Mines and Miners by Commodity and Size Category

Commodity	Size of M/NM Mine						All M/NM Mines	
	Less than 20 Employees		20 to 500* Employees		Over 500* Employees			
	Mines	Workers	Mines	Workers	Mines	Workers	Mines	Workers
Metal	175	1,191	167	21,944	25	24,417	367	47,552
Non-Metal	542	3,471	225	21,685	4	3,810	771	28,966
Stone	2,619	22,838	889	53,413	0	0	3,508	76,251
Sand/ Gravel	5,859	33,144	230	8,899	0	0	6,089	42,043
Total	9,195	60,644	1,511	105,941	29	28,227	10,735	194,812

(*) Based on MSHA's traditional definition, large mines include all mines with employees of 20 or greater.

Source: MSHA's Office of Standards, Regulations, and Variances. Employment figures includes office workers.

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Underground M/NM Mines That Use Diesel Powered Equipment

Impacted Mines by Size. A January 1998 count of diesel powered equipment performed by MSHA's Metal and Nonmetal inspectors shows that 203 of the 261 underground M/NM mines (about 78 percent) regularly use diesel powered equipment. Table V–4 shows the 203 underground M/NM mines that use diesel powered equipment, by size and subsector.

Based on MSHA's traditional definition of a small mine (fewer than 20 employees), Table V–4 shows that of the 203 underground M/NM mines, 82 mines (40 percent) are small mines and 121 mines (60 percent) are large mines. Small mines employ about 4 percent of the workforce (849 employees), while large mines employ about 96 percent of the workforce (18,073 employees).

Based on SBA's definition of a small mine (500 or fewer employees), 196 mines (97 percent) are considered small and 7 mines (3 percent) are large. Under this definition, small mines employ 65 percent of the workforce (12,391 employees), while large mines employ 35 percent of the workforce (6,531 employees).

Impacted Mines by Commodity. The M/NM mining industry consists of about 70 different commodities that can be classified into four commodity categories: Metals, nonmetals, stone, and sand and gravel. Some examples of metals mines are gold, silver, and copper, while some examples of nonmetals mines are potash, salt, and trona. Examples of stone mines are limestone, marble, and granite. Table V–4 also presents the numbers of underground mines operators by these four categories.

Table V-4: Number of Underground Metal and Nonmetal Mines and Miners that Use Diesel Powered Equipment by Commodity and Size Category

Commodity	Size of Underground M/NM Mine						Underground M/NM Mines That Use Diesel Equip.	
	Less than 20 Employees		20 to 500* Employees		Over 500* Employees			
	Mines	Workers	Mines	Workers	Mines	Workers	Mines	Workers
Metal	15	103	44	4,691	4	2,517	63	7,311
Non-Metal	15	100	29	4,645	3	4,014	47	8,759
Stone	52	646	41	2,206	0	0	93	2,852
Sand/Gravel	0	0	0	0	0	0	0	0
Total	82	849	114	11,542	7	6,531	203	18,922

(*) Based on MSHA's traditional definition, large mines include all mines with employees of 20 or greater.

Source: MSHA's Metal and Nonmetal inspectors count of underground Metal and Nonmetal mines that use diesel powered equipment. Includes office workers.

There are no underground mine operators using diesel powered equipment that are classified as sand or gravel. A substantial portion of such small underground mine operators, however, are classified as stone, using either MSHA's definition or SBA's definition of a small mine. Large underground mine operators that use diesel powered equipment are predominantly classified as metal or nonmetal. By MSHA's definition of a large mine (those that employ 20 or more), two thirds (66 percent) of large mines are classified as metal or nonmetal. With respect to SBA's definition of a large mine (those that employ over 500), all large underground mine operators that use diesel powered equipment are classified as either metal or nonmetal.

Structure of Underground M/NM Mining Subsectors

Metal mining. Metal mining in the U.S. consists of about 25 different commodities. Most metal commodities include only one or two mining operations. As is shown in Table V–3, metal mining operations represent 3 percent of the M/NM mines; employ 24 percent of the M/NM miners; and account for 33 percent of the value of M/NM mineral produced in the U.S. (U.S. Geological Survey, 1997, p. 6). By MSHA's definition, 48 percent of the metal mining operations are small. Among underground M/NM mines using diesel powered equipment, Table V–4 shows that metal mining operations represent 31 percent of mines and 39 percent of miners, and (by MSHA's definition) 24 percent are small.

Underground metal mining uses a few basic mining methods, such as stope, room and pillar, and block caving. Larger underground metal mines use more hydraulic drills and track-mounted haulage, whereas smaller underground metal mines use more hand-held pneumatic drills.

Nonmetal Mining (Excluding Stone, Sand and Gravel). For enforcement and statistical purposes, MSHA separates stone mining and sand and gravel mining from other nonmetal mining. There are about 35 different nonmetal commodities, not including stone or sand and gravel. Overall (Table V–3), nonmetal mining operations represent 7 percent of the M/NM mines; employ 15 percent of the M/NM miners; and account for 35 percent of the value of M/NM mineral produced in the U.S. (Ibid., p. 160, 162). By MSHA's definition, 70 percent of the nonmetal mining operations are small. Among underground M/NM mines using diesel powered equipment, Table V–4 shows that nonmetal mining operations represent 23 percent of mines and 46 percent of miners, and (by MSHA's definition) 32 percent are small.

Nonmetal mining uses a wide variety of underground mining methods. For example, potash mines use continuous miners similar to coal mining; oil shale uses in-situ retorting; and gilsonite uses hand-held pneumatic chippers. Some nonmetal commodities use kilns and dryers in ore processing. Others use crushers and mills similar to metal mining. Underground nonmetal mining operations generally use more block caving, room and pillar, and retreat mining methods; less hand-held equipment; and more electrical equipment than metal mining operations.

Stone Mining. There are basically only 8 different stone commodities, of which 7 are further classified as either dimension stone or crushed and broken stone. Overall, stone mining operations represent 33 percent of all M/NM mines; employ 39 percent of the M/NM miners; and account for 19 percent of the value of M/NM mineral produced in the U.S. By MSHA's definition, 75 percent of the stone mining operations are small. Among underground M/NM mines using diesel powered equipment, stone mining operations represent 46 percent of mines and 15 percent of miners, and (by MSHA's definition) 56 percent are small.

Sand and Gravel Mining. Although 57 percent of all M/NM mines are sand and gravel operations, these are all surface mines. No sand and gravel mines will be affected by this regulation.

Economic Characteristics of the M/NM Mining Industry

Overview. The 1996 value of all M/NM mining output was $38 billion (Ibid., p. 6). Metal mining, which includes metals such as aluminum, copper, gold, and iron, contributed $12.5 billion to this total. Nonmetal mining, which includes commodities such as clay, phosphate rock, salt, and soda ash, was valued at $13.3 million. Stone mining contributed $7.4 billion, and sand and gravel contributed $4.8 billion to this total.

The entire M/NM mining industry is markedly diverse, not only in terms of the breadth of minerals but also in terms of each commodity's usage. For example, metals such as iron and aluminum are used to produce vehicles and other heavy duty equipment, as well as consumer goods such as household equipment and beverage cans. Other metals, such as uranium and titanium, have limited uses. Nonmetals like cement are used in construction, while salt is used in a variety of ways, including as a food additive and highway deicing. Soda ash, phosphate rock, and potash also have various commercial uses. Stone and sand and gravel are used in numerous industries including the construction of roads and buildings.

A detailed financial picture of the M/NM mining industry is difficult to develop because most mines either are privately held corporations or sole proprietorships or they are subsidiaries of publicly owned companies. Privately held corporations and sole proprietorships do not make their financial data available to the public; parent companies are not required to separate financial data for subsidiaries in their reports to the Securities and Exchange Commission. As a result, financial data are available for only a few M/NM companies, and these data are not representative of the entire industry. Each commodity has a unique market demand structure. The following discussion focuses on market forces on a few specific commodities of the M/NM industry.

Metal Mining. Historically, the value of metals production has exhibited considerable instability. In the early 1980's, excess capacity, large inventories, and weak demand depressed the international market for metals, while the strong dollar placed U.S. producers at a competitive disadvantage with foreign producers. Reacting to this, many metal mining companies reduced work forces, eliminated marginal facilities, sold non-core businesses, and restructured. At the same time, new mining technologies were developed, and wage increases were restrained. As a result, the metal mining firms now operating are more efficient and have lower break-even prices than those that operated in the 1970's.

Variations in the prices for iron and alloying metals, such as nickel, aluminum, molybdenum, vanadium, platinum, and lead, coincide closely with fluctuations in the market for durable goods, such as vehicles and heavy duty equipment. As a result, the market for these metals is cyclical in nature and is impacted directly by changes in aggregate demand and the economy in general. Both nickel and aluminum have experienced strong price fluctuations over the past few years. With the U.S. and world economies improving, however, demand for such alloys is improving, and prices have begun to recover. It must be noted that primary production of aluminum will continue to be impacted by the push to recycle.

The U.S. market for copper and precious metals, such as gold and silver, is uncertain, which makes consistent production growth in such areas difficult. U.S. gold production in 1996 was estimated at slightly above 1995 levels, which maintains the U.S. position as the world's second largest gold producing nation, after South Africa. U.S. silver production in 1996 increased slightly from 1995 levels to equal the highest production since 1992. U.S. copper production in 1996 continued its modest upward trend, rising to 1.9 million metric tons (Ibid, p. 52).

Overall, the 1996 production from all metal mining is estimated to decrease by about 10 percent from 1995 levels; 1996 estimates put capacity utilization at 84 percent (Ibid., p. 6). MSHA expects that the net result for the metal mining industry may be reduced demand but sustained prices.

Nonmetal Mining. Major commodities in the nonmetal category include salt, clay, phosphate rock, and soda ash. Market demand for these products tends not to vary greatly with fluctuations in aggregate demand. Stone is the leading revenue generator. The U.S. is the largest producer of soda ash and salt. In 1996, the U.S. produced 10.1 million metric tons of soda ash, valued at $778 million, and 40.1 million metric tons of salt, valued at $930 million (Ibid., p. 143). Soda ash is used in the production of glass, soap, detergents, paper, and food. Salt is used in highway deicing, food production, feedstock, and the chemical industry. Phosphate rock is used primarily to manufacture fertilizer. Approximately 42.5 million metric tons of phosphate rock, valued at $900 million, was produced in the U.S. in 1996 (Ibid., p. 124). The remaining nonmetal commodities, which include boron fluorspar, oil shale, and other minerals, are typically produced by a small number of mining operations.

Stone production includes granite, limestone, marble, slate, and other forms of crushed and broken or dimension stone. Sand and gravel products and stone products, including cement, have a cyclical demand structure. As a recession intensifies, demand for these products sharply decreases. Demand for stone, particularly cement, is expected to grow by as much as 3.0 percent, and demand for sand and gravel is expected to grow by as much as 1.2 percent (Ibid., p. 145).

Overall, the 1996 production from nonmetal mining was estimated to increase by 4.5 percent from 1995 levels; 1996 estimates put capacity utilization for stone and earth minerals at about 91 percent (Ibid., p. 6). The net result for the nonmetal mining industry may be higher demand for stone and various other commodities, as well as increased prices.

Adequacy of Miner Protection Provided by Proposed Rule in Underground Metal and Nonmetal Mines. In evaluating the proposed rule, it should be remembered that MSHA has measured dpm concentrations in this sector as high as $5{,}570_{\mathrm{DPM}}$ $\mu g/m^3$—a mean of 830_{DPM} $\mu g/m^3$. See Table III–1 and Figure III–2 in part III of the preamble. As discussed in detail in part III of the preamble, these concentrations place underground metal and nonmetal miners at significant risk of material impairment of their health, and it does not appear there is any lower boundary to the risk. Accordingly, in accordance with the statute, the Agency has to set a standard which reduces these concentrations as much as is both technologically and economically feasible for this sector as a whole.

In this sector, the Agency is proposing a concentration limit on dpm. The proposed concentration limit would be expressed in terms of a restriction on the amount of total carbon because of the measurement system which MSHA proposes to utilize. The proposed limit is $160_{\mathrm{TC}}\ \mu g/m^3$—the equivalent of $200_{\mathrm{DPM}}\ \mu g/m^3$. This permits concentrations of diesel particulate matter in this sector above those which MSHA hopes to achieve in the underground coal sector with the use of 95% particulate filter technology, as described earlier in this part.

Accordingly, the Agency has explored some significant alternatives to the proposal to ascertain if additional protection can feasibly be provided in this sector.

(1) *Establish a lower concentration limit for underground metal/nonmetal mines.* Based on the Agency's risk assessment, a lower concentration limit would provide more miner protection. The Agency has tentatively concluded, however, that at this time it may not be feasible for the underground metal and nonmetal sector to reach a concentration limit below that proposed. The evidence on this point is somewhat mixed, and comments and specific examples to illustrate them would be most welcome.

Technological feasibility of lower limit. In evaluating whether a lower concentration limit is feasible for this sector, MSHA has considered some examples of real-world situations. As described in more detail in the Appendix to this part, MSHA has developed a simulator or model to estimate the ambient dpm that would remain in a mine section after the application of a particular combination of control technologies. The model uses a spreadsheet template into which data can be entered; the formulae in the spreadsheet (described in the Appendix) instantly make the calculations and display the results. This model is hereinafter referred to as ''The Estimator''.

The examples presented here are based on data from several underground metal and nonmetal mines. The first three have been written up in detail and placed into MSHA's record, with actual mine identifiers removed; the fourth is based on information supplied by inspectors, and all available data is presented here. MSHA had picked these mines because the Agency originally thought the conditions there were such that these mines would have great difficulty in controlling dpm concentrations, but this turned out to not always be the case.

FIGURE V–1.—WORK PLACE EMISSIONS CONTROL ESTIMATOR

[Mine Name: Underground Nonmetal Mine A]

	Column A
1. MEASURED OR ESTIMATED IN MINE DP EXPOSURE (μg/m^3)	760 μg/m^3
2. VEHICLE EMISSION DATA	
EMISSIONS OUTPUT (gm/hp-hr)	
VEHICLE 1 INDIRECT INJECTION 0.3–0.5 gm/hp-hr FEL	0.3 gm/hp-hr
VEHICLE 2 OLD DIRECT INJECTION 0.5–0.9 gm/hp-hr SCALER	0.3 gm/hp-hr
VEHICLE 3 NEW DIRECT INJECTION 0.1–0.4 gm/hp-hr DRILL	0.3 gm/hp-hr
VEHICLE 4 BOLTER	0.7 gm/hp-hr
VEHICLE OPERATING TIME (hours)	
VEHICLE 1 FEL	6 hours
VEHICLE 2 SCALER	6 hours
VEHICLE 3 DRILL	6 hours
VEHICLE 4 BOLTER	6 hours
VEHICLE HORSEPOWER (hp)	
VEHICLE 1 3 @ 480 FEL	1440 hp
VEHICLE 2 2 @ 250 SCALER	500 hp
VEHICLE 3 2 @ 250 DRILL	500 hp
VEHICLE 4 2 @ 82 BOLTER	164 hp
SHIFT DURATION (hours)	8 hours
AVERAGE TOTAL SHIFT PARTICULATE OUTPUT (gm)	0.13 gm/hp-hr
3. MINE VENTILATION DATA	
FULL SHIFT INTAKE DIESEL PARTICULATE CONCENTRATION	50 μg/m^3
SECTION AIR QUANTITY	209000 cfm
AIRFLOW PER HORSEPOWER	80 cfm/hp
4. CALCULATED SWA DP CONCENTRATION WITHOUT CONTROLS	
5. ADJUSTMENTS FOR EMISSION CONTROL TECHNOLOGY	
ADJUSTED SECTION AIR QUANTITY	330000 cfm
VENTILATION FACTOR (INITIAL CFM/FINAL CFM)	0.63
AIRFLOW PER HORSEPOWER	127 cfm/hp
OXIDATION CATALYTIC CONVERTER REDUCTION (%)	
VEHICLE 1	0%
VEHICLE 2 IF USED ENTER 0–20%	0%
VEHICLE 3	0%
VEHICLE 4	0%
NEW ENGINE EMISSION RATE (gm/hp-hr)	
VEHICLE 1	0.1 gm/hp-hr
VEHICLE 2 ENTER NEW ENGINE EMISSION (gm/hp-hr)	0.1 gm/hp-hr
VEHICLE 3	0.1 gm/hp-hr
VEHICLE 4	0.1 gm/hp-hr
AFTERFILTER OR CAB EFFICIENCY (%)	
VEHICLE 1	0%
VEHICLE 2 USE 65–95% FOR AFTERFILTERS	0%
VEHICLE 3 USE 50–80% FOR CABS	0%
VEHICLE 4	0%

FIGURE V–1.—WORK PLACE EMISSIONS CONTROL ESTIMATOR—Continued

[Mine Name: Underground Nonmetal Mine A]

	Column A
6. ESTIMATED FULL SHIFT DP CONCENTRATION	194 μg/m^3

The mining community is encouraged to obtain a copy of the Estimator from MSHA and run simulations of its own in individual mines. MSHA would welcome having such examples submitted for the record as part of comments submitted on this proposed rulemaking.

The first example, summarized in Figure V–1, involves a section of an underground salt mine. This section has 9 diesel engines, most of them very heavy duty: three front end loaders of 480 hp each, 2 scalers and 2 drills at 250hp each, and an 82 hp bolter.

Entered in section 1 of the figure is the measured level of dpm, 760_{DPM} μg/m^3. This measurement reflects the fact that the equipment was all equipped with oxidation catalytic converters; otherwise, the measurement would have been on the order of 20% higher.

Entered in sections 2 and 3 is information about the engines, operating cycle, horsepower, shift duration, intake dpm concentration, and ventilation currently used in the mine. The entries for engines of a similar type and horsepower were combined. The intake concentration is dpm coming from outside the section, and in the case of these examples has been estimated to be about 50_{DPM} μg/m^3. This information is retained by the Estimator as a baseline against which to compare a particular combination of proposed controls.

Sections 2 and 3 of the Estimator also calculate two ratios — the average total shift particulate output, and the airflow per horsepower—that provide useful insights into what controls might be available. For example, in this case, an airflow of 80 cfm/hp is below recommended levels, suggesting that a ventilation increase should be part of the solution to the high dpm concentrations.

The controls to be modeled are entered into section 5 of the Estimator. In this example, the ventilation is increased enough to increase the airflow per horsepower to 127 cfm/hp. Oxidation catalytic converters are already on the equipment, so nothing can be added in that regard. In the example, all 9 engines (grouped into 4 lines by combining those with similar horsepower, as originally entered) would be replaced by newer engines with lower emission rates. No filters or cabs would be used. The calculated result is an ambient dpm concentration of 194_{DPM} μg/m^3.

This mine section could actually lower its dpm concentrations more using different combinations of controls. For example, using 80% filters on the three front-end loaders instead of new engines would, according to the Estimator, result in an ambient dpm level of 161_{DPM} μg/m^3. If both the 80% filters and new engines were used, the ambient dpm level would be 128_{DPM} μg/m^3. Keep in mind that of the amount that remains, 50_{DPM} μg/m^3 comes from the intake to the section. The next two studies are of an underground limestone mine that operates in two shifts: one for production, and one for support.

Figure V–2.—Work Place Emissions Control Estimator

[Mine Name: Underground Nonmetal Mine B Production Shift]

	Column A
1. MEASURED OR ESTIMATED IN MINE DP EXPOSURE (μg/m^3	330 μg/m^3
2. VEHICLE EMISSION DATA	
EMISSIONS OUTPUT (gm/hp-hr)	
VEHICLE 1 INDIRECT INJECTION 0.3–0.5 gm/hp-hr FEL	0.1 gm/hp-hr
VEHICLE 2 OLD DIRECT INJECTION 0.5–0.9 gm/hp-hr Truck 1	0.2 gm/hp-hr
VEHICLE 3 NEW DIRECT INJECTION 0.1–0.4 gm/hp-hr Truck 2	0.1 gm/hp-hr
VEHICLE 4	0.0 gm/hp-hr
VEHICLE OPERATING TIME (hours)	
VEHICLE 1 FEL	9 hours
VEHICLE 2 Truck 1	9 hours
VEHICLE 3 Truck 2	9 hours
VEHICLE 4	0 hours
VEHICLE HORSEPOWER (hp)	
VEHICLE 1 FEL	315 hp
VEHICLE 2 Truck 1	250 hp
VEHICLE 3 Truck 2	330 hp
VEHICLE 4	0 hp
SHIFT DURATION (hours)	10 hours
AVERAGE TOTAL SHIFT PARTICULATE OUTPUT (gm)	0.09 gm/hp-hr
3. MINE VENTILATION DATA	
FULL SHIFT INTAKE DIESEL PARTICULATE CONCENTRATION	50 μg/m^3
SECTION AIR QUANTITY	155000 cfm
AIRFLOW PER HORSEPOWER	173 cfm/hp
4. CALCULATED SWA DP CONCENTRATION WITHOUT CONTROLS	
5. ADJUSTMENTS FOR EMISSION CONTROL TECHNOLOGY	

Figure V–2.—Work Place Emissions Control Estimator—Continued

[Mine Name: Underground Nonmetal Mine B Production Shift]

	Column A
ADJUSTED SECTION AIR QUANTITY	155000 cfm
VENTILATION FACTOR (INITIAL CFM/FINAL CFM)	1.00
AIRFLOW PER HORSEPOWER	173 cfm/hp
OXIDATION CATALYTIC CONVERTER REDUCTION (%)	
VEHICLE 1	0%
VEHICLE 2 IF USED ENTER 0–20%	0%
VEHICLE 3	0%
VEHICLE 4	0%
NEW ENGINE EMISSION RATE (gm/hp-hr)	
VEHICLE 1	0.1 gm/hp-hr
VEHICLE 2 ENTER NEW ENGINE EMISSION (gm/hp-hr)	0.2 gm/hp-hr
VEHICLE 3	0.1 gm/hp-hr
VEHICLE 4	0.0 gm/hp-hr
AFTERFILTER OR CAB EFFICIENCY (%)	
VEHICLE 1 CABS	70%
VEHICLE 2 USE 65–95% FOR AFTERFILTERS	70%
VEHICLE 3 USE 50–80% FOR CABS	70%
VEHICLE 4	0%
6. ESTIMATED FULL SHIFT DP CONCENTRATION	134 µg/m^3

Figure V–3.—Work Place Emissions Control Estimator

[Mine Name: Underground Nonmetal Mine B Support Shift]

	Column A
1. MEASURED OR ESTIMATED IN MINE DP EXPOSURE (µg/m3)	600 µg/m^3
2. VEHICLE EMISSION DATA	
EMISSIONS OUTPUT (gm/hp-hr)	
VEHICLE 1 INDIRECT INJECTION 0.3–0.5 gm/hp-hr Drill	0.3 gm/hp-hr
VEHICLE 2 OLD DIRECT INJECTION 0.5–0.9 gm/hp-hr Bolter	0.6 gm/hp-hr
VEHICLE 3 NEW DIRECT INJECTION 0.1–0.4 gm/hp-hr Scaler	0.7 gm/hp-hr
VEHICLE 4 Anfo	0.7 gm/hp-hr
VEHICLE OPERATING TIME (hours)	
VEHICLE 1 Drill	8 hours
VEHICLE 2 Bolter	4 hours
VEHICLE 3 Scaler	8 hours
VEHICLE 4 Anfo	4 hours
VEHICLE HORSEPOWER (hp)	
VEHICLE 1 Drill	116 hp
VEHICLE 2 Bolter	193 hp
VEHICLE 3 Scaler	119 hp
VEHICLE 4 Anfo	86 hp
SHIFT DURATION (hours)	8 hours
AVERAGE TOTAL SHIFT PARTICULATE OUTPUT (gm)	0.39 gm/hp-hr
3. MINE VENTILATION DATA	
FULL SHIFT INTAKE DIESEL PARTICULATE CONCENTRATION	50 µg/m^3
SECTION AIR QUANTITY	155000 cfm
AIRFLOW PER HORSEPOWER	302 cfm/hp
4. CALCULATED SWA DP CONCENTRATION WITHOUT CONTROLS	
5. ADJUSTMENTS FOR EMISSION CONTROL TECHNOLOGY	
ADJUSTED SECTION AIR QUANTITY	155000 cfm
VENTILATION FACTOR (INITIAL CFM/FINAL CFM)	1.00
AIRFLOW PER HORSEPOWER	302 cfm/hp
OXIDATION CATALYTIC CONVERTER REDUCTION (%)	
VEHICLE 1	0%
VEHICLE 2 IF USED ENTER 0–20%	0%
VEHICLE 3	0%
VEHICLE 4	0%
NEW ENGINE EMISSION RATE (gm/hp-hr)	
VEHICLE 1	0.3 gm/hp-hr
VEHICLE 2 ENTER NEW ENGINE EMISSION (gm/hp-hr)	0.6 gm/hp-hr
VEHICLE 3	0.7 gm/hp-hr
VEHICLE 4	0.7 gm/hp-hr
AFTERFILTER OR CAB EFFICIENCY (%)	
VEHICLE 1	80%

Figure V–3.—Work Place Emissions Control Estimator—Continued

[Mine Name: Underground Nonmetal Mine B Support Shift]

	Column A
VEHICLE 2 USE 65–95% FOR AFTERFILTERS	80%
VEHICLE 3 USE 50–80% FOR CABS	80%
VEHICLE 4	80%
6. ESTIMATED FULL SHIFT DP CONCENTRATION	160 µg/m3

The two shifts use completely different types of diesel-powered equipment.

Figure V–2 summarizes the study of the production shift, and Figure V–3 summarizes the study of the support shift.

The production shift already has low-emission engines on the three pieces of equipment present—a front-end loader and two trucks, as well as oxidation catalytic converters on each engine.

Its ventilation provides 173 cfm/hp. Accordingly, the measured dpm for this shift is only about 330$_{DPM}$ µg/m^3 With the addition of a cab on each unit providing roughly 70% effectiveness (see part II of this preamble on cab effectiveness), the ambient concentration (to which the equipment operator would be exposed) can be reduced to 134$_{DPM}$ µg/m^3.

In the case of the support shift, the engines do emit particulate at a high rate; but they all are low horsepower engines, and all have oxidation catalytic converters. The ventilation is the same as on the production shift. Hence the measured dpm is on the order of 600$_{DPM}$ µg/m^3. In the example shown, 80% filtration of each piece of equipment would bring the concentration down to 160$_{TC}$ µg/m^3. If 95% filters were used, the Estimator indicates this concentration could be reduced to 77$_{DPM}$ µg/m^3. Since 50$_{DPM}$ µg/m^3 of this is the estimated intake into the section, the filters and controls already in place appear to be capable of eliminating almost all dpm generated within the section itself.

FIGURE V–4.—WORK PLACE EMISSIONS CONTROLS ESTIMATOR

[Mine Name: Underground Gold Mine]

	Column A
1. MEASURED OR ESTIMATED IN MINE DP EXPOSURE (ug/m3)	1000 us/m^3
2. VEHICLE EMISSION DATA	
EMISSIONS OUTPUT (gm/hp-hr)	
VEHICLE 1 INDIRECT INJECTION 0.3–0.5	
gm/hp-hr FEL	0.7 gm/hp-hr
VEHICLE 2 OLD DIRECT INJECTION 0.5–0.9	
gm/hp-hr Scaler	0.7 gm/hp-hr
VEHICLE 3 NEW DIRECT INJECTION	
0.1–0.4 gm/hp-hr Drill	0.7 gm/hp-hr
VEHICLE 4	0.0 gm/hp-hr
VEHICLE OPERATING TIME (hours)	
VEHICLE 1 FEL	6 hours
VEHICLE 2 Scaler	6 hours
VEHICLE 3 Drill	6 hours
VEHICLE 4	0 hours
VEHICLE HORSEPOWER (hp)	
VEHICLE 1 FEL	315 hp
VEHICLE 2 Scaler	250 hp
VEHICLE 3 Drill	330 hp
VEHICLE 4	0 hp
SHIFT DURATION (hours)	8 hours
AVERAGE TOTAL SHIFT PARTICULATE OUTPUT (gm)	0.44 gm/hr-hr
3. MINE VENTILATION DATA	
FULL SHIFT INTAKE DIESEL PARTICULATE CONCENTRATION	50 ug/m^3
SECTION AIR QUALITY	185000 cfm
AIRFLOW PER HORSEPOWER	207 cfm/hp
4. CALCULATED SWA DP CONCENTRATION WITHOUT CONTROLS	
5. ADJUSTMENTS FOR EMISSION CONTROL TECHNOLOGY	
ADJUSTED SECTION AIR QUANTITY	185000 cfm
VENTILATION FACTOR (INITIAL CFM/FINAL CFM)	1.00
AIRFLOW PER HORSEPOWER	207 cfm/hp
OXIDATION CATALYTIC CONVERTER REDUCTION (%)	
VEHICLE 1	20%
VEHICLE 2 IF USED ENTER 0–20%	20%
VEHICLE 3	20%
VEHICLE 4	0%
NEW ENGINE EMISSION RATE (gm/hp-hr)	
VEHICLE 1	0.7 gm/hp-hr
VEHICLE 2 ENTER NEW ENGINE EMISSION (gm/hp-hr)	0.1 gm/hp-hr
VEHICLE 3	0.1 gm/hp-hr
VEHICLE 4	0.0 gm/hp-hr

FIGURE V–4.—WORK PLACE EMISSIONS CONTROLS ESTIMATOR—Continued

[Mine Name: Underground Gold Mine]

	Column A
AFTERFILTER OR CAB EFFICIENCY (%)	
VEHICLE 1 FILTER	95%
VEHICLE 2 USE 65–95% FOR AFTERFILTERS	0%
VEHICLE 3 USE 50–80% FOR CABS	0%
VEHICLE 4	0%
6. ESTIMATED FULL SHIFT DP CONCENTRATION	134 ug/m^3

The final study, summarized in Figure V–4, involves a multi-level underground gold mine. Each level had one production unit on a separate split of ventilation air. The three engines are large and have a high emission rate, and have no oxidation catalytic converters. The ventilation produces over 200 cfm/hp. In this case, no initial measurement was taken; instead, an initial concentration of 1000$_{DPM}$ µg/m^3 was estimated by taking a percentage of the respirable dust concentration (a method discussed in the Appendix).

By replacing all of the current engines with low-emission engines equipped with catalytic converters, the Estimator calculates that the ambient concentration can be reduced to 159$_{DPM}$ µg/m^3, of which 50$_{DPM}$ µg/m^3 again constitutes the estimated intake to the section. Further reductions could be achieved by adding a filter to the front-end loader and/or drill.

These studies seem to suggest that using a combination of available technologies, even mine sections with significant ambient intake and standard ventilation parameters can reduce dpm concentrations well below the proposed concentration limit.

Economic feasibility of lower concentration limit. MSHA's cost estimates for the proposed concentration limit of 200$_{DPM}$ µg/m^3 for underground metal and nonmetal mines comes to about $19.2 million a year. (See Table I–1, in the response to Question 5 in part I of the preamble). For an average underground metal and nonmetal dieselized mine that uses diesel powered equipment, this amounts to about $94,600 per year to comply with the proposed concentration limits.

The assumptions used in preparing the cost estimates are discussed in detail in the Agency's PREA, and are based on a January 1998 count of diesel powered equipment that regularly operates in the underground metal and nonmetal mines. The count was performed by MSHA's metal and nonmetal inspectors. The assumptions can be summarized as follows: engineering controls, such as low emission engines, ceramic filters, oxidation catalytic converters, and cabs would be needed on certain diesel powered equipment. Most of the engineering controls would be needed on diesel powered equipment used for production, while a small amount of diesel powered equipment that is used for support purposes would need engineering controls. In addition to these controls, MSHA assumed that some underground metal and nonmetal mines would need to make ventilation changes in order to meet the proposed concentration limits.

While the four studies presented here suggest it might be economically feasible for some mines in this sector to reduce dpm concentrations below the concentration level proposed, the Agency is reluctant to conclude on the basis of the examples that most underground metal and nonmetal operators would find it economically feasible to reduce concentrations below the proposed limit of 160$_{TC}$ µg/m^3 (200$_{DPM}$ µg/m^3). The Agency welcomes additional examples and information it can use to make a better assessment of the costs operators would incur to reduce dpm to various concentration limits, as well as other considerations relevant to economic feasibility.

(2) *Shorten the phase-in time to reach the final concentration limit in underground metal/nonmetal mines.* Under the proposed rule, there is a phase-in period for a dpm concentration limit (see proposed § 57.5060). Operators would have 18 months to reduce dpm concentrations in areas of the mine where miners work or travel to 400$_{TC}$ µg/m^3 (500$_{DPM}$ µg/m^3), and up to 60 months in all to reduce dpm concentrations in those areas to 160$_{TC}$ µg/m^3 (200$_{DPM}$ µg/m^3). MSHA established this phase-in period because it has tentatively concluded that it would be infeasible for the underground metal and nonmetal mining industry as a whole to implement the requirements sooner.

With respect to technological feasibility, MSHA notes that many of these mines face unique difficulties in using ventilation to lower dpm concentrations; and high efficiency particulate filters may not yet be commercially available for certain types or sizes of engines and equipment used in this sector. The proposed rule includes a provision for a special time extension to deal with unique situations. Shortening the normal time frame available to this sector could create a situation where special exemptions would become the norm.

The costs of the proposed rule would also increase significantly were the final concentration limit to become effective sooner. As explained in the Agency's PREA, a substantial portion of the costs to implement these provisions were calculated using a 5-year discounting process to reflect the phase-in schedule. Speeding implementation would significantly impact costs.

Accordingly, MSHA has tentatively concluded that, for the underground metal and nonmetal sector as a whole, an accelerated approach may not be feasible.

(3) *In lieu of a concentration limit, require high efficiency filters on certain types of equipment.* In the underground coal sector, MSHA has proposed requiring high efficiency filters on all but light-duty equipment. This appears to be a very effective and feasible way of reducing dpm concentrations in that sector. Accordingly, MSHA considered requiring a similar approach in underground metal and nonmetal mines.

MSHA estimates that to require 95% efficient filters on all diesel engines in underground metal and nonmetal mines after 30 months would cost about $41 million a year. On the other hand, to require that only heavy duty equipment use 95% filters after 30 months would cost about $20 million a year. (''Heavy duty'' equipment here means equipment that moves rock or ore; for costing purposes, MSHA assumed this included production equipment and about five percent of support equipment, which is about 46% of the diesel equipment in underground metal and nonmetal mines).

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