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Short-term variation during asbestos abatement activities

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SHORT-TERM VARIATION DURING ASBESTOS ABATEMENT ACTIVITIES

by

Erle Baxter Jones

A Thesis Submitted to the Faculty of the

DEPARTMENT OF PHARMACOLOGY AND TOXICOLOGY

In Partial Fulfillment of the Requirements
For the Degree of

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THE UNIVERSITY OF ARIZONA

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This thesis has been approved on the date shown below:

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Wesley Clayton PhD.
Professor of Toxicology

July 28, 1987
Date

To

Bill and Lori

whose open house provided

the ideal environment to

pursue this endeavor

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ABSTRACT

The standard method (NIOSH 7400) employed to assess asbestos fiber levels in an abatement environment is based on full period sampling (2-4 hours) using a membrane filter attached to a constant flow pump. Optical analysis with a phase-contrast light microscope is then used to count fibers. Many employers find this method slow and tedious, and are turning to particle counters which offer a direct assessment of the abatement environment.

The fibrous aerosol monitor (FAM) was designed to count fibrous particulates as accurately as the standard method, P & CAM 239, now NIOSH 7400. The Hi-Vol (HV) method a modification of the standard method, was developed to collect fibers over shorter time intervals (i.e., 10-minutes) with the same accuracy as the standard method. The current study sought to compare the HV method with the FAM to assess its validity in measuring airborne asbestos levels and to assess the importance of short-term variation in asbestos levels in determining appropriate respiratory protection.

Correlation between the FAM and HV showed a poor linear relationship in all comparison studies. However,

there was good correlation between the average of short-term HV samples and long-term time-weighted average (TWA) samples at reported low levels of airborne asbestos. All membrane filter techniques showed strong correlation with each other, but were weakly correlated with the FAM. These observations suggest that the FAM needs further refinement to improve its accuracy vs. the membrane filter methods. Based upon the low fiber levels seen in this study, more stringent types of respiratory protection for use by the abatement worker to restrict airborne asbestos fiber exposures, are not justified.

INTRODUCTION

Asbestos containing materials (ACM) release fibers during product manufacture, use, and subsequent handling during repair, renovation or removal activities. These fibers have been implicated in various asbestos-related diseases that include asbestosis, lung cancer, mesothelioma, gastro-intestinal cancer, and thickening of the pleural lining. Two divergent viewpoints have evolved concerning allowable exposure limits for asbestos. One states that there is a threshold for exposure to asbestos below which the great majority of exposed workers will not develop occupational illness, disease, or death. The American Conference of Governmental Industrial Hygienists (ACGIH) exemplifies this approach with their threshold limit value (TLV), which is based on an 8-hour time-weighted average (TWA) concentration of airborne asbestos fibers. The other viewpoint implies there is no safe level of exposure (NIOSH, 1976) based on limited findings which suggest that very short exposures to asbestos fibers have resulted in cancer many years later (Federal Register, 1986).

The Present Controversy over Asbestos

Large-scale usage of asbestos by industry began around 1880. The first report of an association between asbestosis and lung cancer was presented by Lynch and Smith (1935) in the U.S., and by Gloyne (1935) in the U.K. Nearly ten years later, case reports of pleural and peritoneal tumors associated associated with asbestos appeared (Wedler, 1943, Wyers, 1946). Epidemiologic evidence from Doll (1955) showed a ten-fold excess risk of lung cancers in U.K. asbestos textile workers who were employed before 1930. The next year the Factory Inspectorate mandated the regulations which subsequently improved dust conditions in factories. Mesotheliomas were also detected, but these findings were not reported until later by Mancuso and Coulter (1963), and Selikoff et al. (1964).

In 1968, the British Occupational Health Society (BOHS) published an epidemiological study of 290 asbestos workers relating their exposure to health status. After a review of this data, the BOHS proposed a 5 f/cc standard for airborne asbestos levels in the workplace which was adopted with minor modifications by the British government in 1969, and implemented as law in May 1970. Similarly in the U.S., this initial standard was adopted as a guideline by the ACGIH (1970), and implemented as law by OSHA in 1972

as its permissible exposure limit (PEL) for asbestos (Federal Register, 1972).

Since 1970, the ACGIH has annually published a TLV booklet listing recommended maximum airborne concentrations for substances. These threshold limit values represent best estimates of workplace concentrations to which nearly all workers can be repeatedly exposed over a working lifetime without adverse effect.

OSHA is a government administrative agency within the Department of Labor. Its purpose is to promulgate and enforce standards (PELs) that are designed to protect nearly all workers from workplace hazards. The PEL for asbestos initially promulgated by OSHA in 1971 was the prevailing ACGIH TLV of 5 fibers (>5um in length) per cubic centimeter of air (5 f/cc). In 1975, the BOHS recommended reducing the standard to 2 f/cc. Both the ACGIH TLV and OSHA PEL were modified to reflect this change. Concurrently in 1975, OSHA announced its intention to consider lowering the PEL to 0.5 f/cc. In October 1983 OSHA published an Emergency Temporary Standard of 0.5 f/cc. Their intent was to change the PEL without benefit of the normal rule-making procedures which include public hearings. This attempt to circumvent established rule-making procedures was challenged in Federal Court by the Asbestos Information Association (AIA). The court

agreed to set aside the ETS pending further review and decision (Federal Register, 1986).

However, in a criteria document for asbestos (NIOSH 1976), NIOSH recommended a standard of 0.1 f/cc for all forms of asbestos fibers which has remained unchanged since its initial publication. The justification for this recommendation was a NIOSH goal to provide a standard as close to zero as possible, and 0.1 f/cc represented the lowest level detectable by the available analytical technique (P & CAM 239, in NIOSH, 1976). It is noteworthy that the process of criteria development often involves preparation by contract personnel without formal peer review. As a consequence, there is no guarantee of peer review of NIOSH recommended standards by qualified and experienced industrial hygienists in the field. NIOSH recommendations, therefore, do not necessarily represent a consensus opinion regarding safety and health. The Building and Construction Trades Department (BCTD) has adopted the 0.1 f/cc limit, and claims that even at this recommended PEL a significant risk of mortality from cancer still exists.

On June 13, 1986, OSHA promulgated a new PEL for asbestos of 0.2 f/cc (Federal Register, 1986). The new standard is based on epidemiological data which indicates a significant health risk existed at the old PEL of 2 f/cc.

The new standard was implemented to reduce or eliminate that risk (see Appendix 1). OSHA agreed with the BCTD that some risk of asbestos-related disease would exist even under a PEL of 0.1 f/cc, but explained that its decision to promulgate a PEL of 0.2 f/cc was based more on reduction than elimination of risk. Finally, OSHA determined that the 0.2 f/cc PEL was the lowest level that could feasibly be attained in operations and workplaces in both general industry and construction.

During the rulemaking period in April 1986, several opponents who wanted a higher new standard than the 0.2 f/cc level argued that the actual risk to workers exposed to asbestos was approximately one-sixteenth that predicted by OSHA (0.5% incidence of asbestosis), because average exposures over an average working life would be one-fourth the level of OSHA's lifetime (45 year) exposure predictions. Led by the AIA of North America, the opponents to the proposed new lower standard claimed that significant risk would be eliminated at a new PEL of 0.5 f/cc. The present controversy over asbestos continues.

Sources of Asbestos Exposures

Occupational and environmental exposures to asbestos containing materials (ACM) occur primarily during the construction, occupation, and maintenance or renovation of buildings. ACM is normally found in three forms: (1)

sprayed or troweled on ceilings and walls (surfacing material); (2) in insulation around hot or cold pipes, ducts, boilers and tanks (pipe and boiler insulation); and (3) in a variety of other products such as ceiling and floor tiles and wall boards (miscellaneous materials). In general, ACM in the first two categories is of greatest concern, especially if it is friable. ACM is defined as friable if it can can be crumbled, pulverized, or reduced to powder by hand pressure.

Both the Occupational Safety and Health Administration (OSHA) and the Environmental Protection Agency (EPA) have published regulations and guidelines to reduce asbestos exposure (Federal Register, 1986; EPA, 1985). EPA guidelines focus on application and removal of ACM in new or remodeled buildings, and identification of friable asbestos in schools. EPA also regulates the industrial emission of asbestos fibers and the disposal of asbestos waste. The agency is considering banning all nonessential uses of asbestos and asbestos products. The prohibitions being considered would be phased in over a period of time and would exempt certain essential uses for which reasonable substitutes do not exist and which do not pose an unreasonable health risk. One example of an exemption from the ban would be fire protection suits for firemen. If the asbestos is totally enclosed, sealed, or

bonded into the suit, it is presumed that no asbestos fibers would be released into the air.

Once ACMs are discovered in a building regulated by EPA, a maintenance program must be established to control asbestos exposure. The maintenance program is designed to: (1) clean-up asbestos fibers previously released (2) repair or remove damaged or deteriorated ACM, (3) prevent future release of asbestos fibers by minimizing disturbance or damage to ACM in the buildings, and (4) periodically monitor the condition of ACM.

Activities associated with the regulations and programs implemented to control asbestos fiber release can pose a significant potential for exposure to many abatement workers. This study focuses on short-term exposure variation experienced by abatement workers, and examines methodologies for their determination.

Asbestos Related Diseases

Diseases associated with asbestos exposure include asbestosis, malignant mesothelioma, carcinoma of the lung, and parietal pleural plaques. Gastrointestinal cancer has recently been linked to asbestos exposure, but more studies are needed before a direct causal link can be made.

Commonly used forms of asbestos may be divided into two general groups: serpentine and amphiboles. Chrysotile asbestos makes up the serpentine group, and its fibers tend

to be longer and more curved than the amphiboles. Chrysotile has an alternating sheet structure of magnesium hydroxide and silica, rolled in a 1:1 layer. The curvature of the layer and the tubular morphology of the mineral result from bidimensional differences between the unit cell dimensions of the two sheets (Bonneau et al., 1986). Rupture along the long axis of a single fiber can result in several smaller diameter fibrils, which has been confirmed histologically in rat lung pathogenesis studies (Davis et al., 1986).

The amphibole group consists of amosite and crocidolite, which are of commercial value, and actinolite, tremolite, and anthophyllite, which are commonly found as contaminants of chrysotile. The amphiboles are fibers containing double chains or ribbons of silica, cross-linked to cations (Fe^{2+}, Fe^{3+}, Mg^{2+}, Na^{+}), which form straight fibers with longitudinal grooves and diameters somewhat greater than chrysotile. Rupture of the chain structure does not produce fibrils. All types of asbestos can produce pulmonary fibrosis and no evidence suggests that the morphologic features of the lesions differ. In addition, many commercial asbestos products contain mixtures of more than one type of fiber. Although fibers of the amphibole series are better asbestos-body (AB)

formers, exposure to chrysotile asbestos via inhalation is well correlated with the development of fibrosis.

A paper by Gylseth et al. (1983) noted that studies of lung dust content in humans have shown that while chrysotile has usually formed the major part of most asbestos dust exposures, amphibole fibers predominate in the lung at autopsy. Since chrysotile asbestos comprises 95% of all forms of asbestos used in the U.S., one would expect it to be more causal in fiber pathogenesis than the amphiboles; however in studies done by Wagner et al. (1960), and Weill et al. (1979), it was noted:

> The separation of chrysotile fibrils in tissue could explain the major anomaly that has been found between animal experimental studies and human epidemiology. While experimental studies almost without exception have reported chrysotile to be the most dangerous fiber tested, studies of asbestos workers have indicated that amphibole dusts, particularly crocidolite, are the most harmful to humans.

Recently, other researchers (Davis et al., 1986) have obtained similar results when studying the anomaly in asbestos type pathogenesis between rats and humans. It would appear very likely that while chrysotile can persist long enough in a short-lived species like the rat to provoke a neoplastic response or pulmonary fibrosis, it [chrysotile] may be removed from human lungs before the disease can develop. The separation of chrysotile bundles

into individual fibrils is likely to be important in this process, since these long but extremely thin structures might be expected to be more susceptible to dissolution.

In a similar vein, Morgan and Holmes (1986) studied the solubility of asbestos fibers in vivo and found that the magnesium in chrysotile dissolves at a rate specific to its surface area, while the silicate structure of the amphiboles remains insoluble in lung tissue. This, they feel, explains the persistence of amphibole fibers in human lung many years after the cessation of occupational exposure. It was also surmised that chrysotile fibers are preferentially removed from the lung with respect to the amphiboles due to the disintegration of the leached chrysotile fibers into particles that are then removed mechanically by alveolar macrophages, or by other means. However, more studies are needed to resolve this question.

Asbestosis is defined histologically as the presence in tissue sections of both asbestos bodies (ABs) and peribronchiolar fibrosis with or without fibrosis of the alveolar septa, and with or without honeycombing (Roggli et al., 1986). Four different gradings of the disease have been used to determine mineral fiber content of the lungs and its associations with lung cancer and mesothelioma. Minimal asbestosis is present when minimal collagen is seen in the interalveolar septa or around

respiratory bronchioles and alveolar ducts in the presence of ABs. Slight asbestosis shows as a definite thickening of the walls of the respiratory bronchioles extending into the alveolar duct, again with the presence of ABs. Moderate asbestosis is indicated when the lesions are more profuse and show some linkage between the lesions. Marked/severe asbestosis is diagnosed when complete distortion of the normal architecture occurs with large areas of fibrosis, ABs, and fibers being seen in the distorted air spaces (Wagner et al., 1986).

Clinical signs of asbestosis include dyspnea, coughing, shortness of breath, rales in both lung bases, clubbing of the fingers, and X-ray abnormalities. Amphibole fiber amounts are seen to increase with the severity of asbestosis and occupational exposure, but chrysotile fiber amounts remain constant over the grades. In addition, mesotheliomas occur mostly with a minimal or slight grading and low exposures, while lung carcinomas are seen to occur with the moderate to severe grades with heavier exposures (Wagner et al., 1986).

Malignant mesothelioma is defined as a cancerous tumor derived from cells which line the abdominal, chest, and heart cavities. Carcinoma of lung and gastro-intestinal tract (G.I.) are cancers derived from the lining cells of these organs. They are classified

histologically according to the criteria proposed by the World Health Organization (1982). Parietal pleural plaques are described as ivory colored, circumscribed foci of pleural thickening, with or without calcification. They most often affect the posterolateral chest wall and domes of the diaphragm and exhibit microscopic features of layers of almost cellular hyalinised collagen (Roggli et al., 1986). The generally accepted latency period between asbestos exposure and onset of disease is: 10-20 years for asbestosis, 16-35 years for mesothelioma, and 20-40 years for lung cancer.

Asbestos bodies (ABs), the hallmark of exposure to asbestos, are formed by the coating of partially phagocytized asbestos fibers with an iron/protein mucopolysaccharide complex. The typical beaded configuration (dumbbell, javelin, or segmented morphology) and thin transparent central core are virtually always nucleated on asbestos fibers, with the vast majority of these fibers being the commercial amphiboles - crocidolite and amosite (Churg and Warnock, 1981).

Roggli et al. (1986) also found reasonably reliable correlation between AB content in lung tissue, and lung concentrations of long (> 5um) commercial amphibole fibers. On the other hand, the correlation between AB counts and concentrations of chrysotile or non-commercial amphibole

fibers was poor, the vast majority of these fibers being $\leq$ 5um in length. The investigators concluded that while some ABs are nucleated on chrysotile fibers, they give little or no indication of chrysotile fiber content in the lung.

However, in a study of ABs in 25 chrysotile miners, Holden and Churg (1986) reported that the usual histologic criteria for the diagnosis of asbestosis (diffuse interstitial fibrosis plus ABs) were easily found in all cases. Mineralogic analysis of four cases showed that 46 of 72 ABs (64%) isolated and examined contained chrysotile cores, and 21 of 72 ABs (29%) contained cores of the amphiboles - tremolite and actinolite. Mean fiber lengths for the fiber types were 35um and 36um, respectively. To conclude at this time that only fibers of a certain type and size play a significant role in the genesis of asbestos-related disease does not seem to be justified.

Fiber size has also been graded for risk of asbestos-related disease with evidence pointing to longer fibers as the problem species, but there are possible effects related to shorter fibers. A fiber is defined by OSHA and the ACGIH as having a length greater than or equal to 5um with an aspect ratio, length-to-apparent diameter, of 3. The fiber length basis for their standards was not due to any specific toxicological properties of asbestos at

5um, but rather based on the ready availability of optical analysis techniques.

Particle size or relative fiber size is related to penetration and deposition in the lung. Langer and Wolff (1978) reported that fiber lengths > 8um are deposited in the trachea and tracheo-bronchial tree, while fibers of shorter length are deposited by diffusion and inertial impaction deeper into the lung. Aalto and Heppleston (1984) presented the concept that short fibers are important in the early stages of asbestos-related diseases, while longer lengths become important in later disease states. In addition, small fibers have been seen to migrate more efficiently (translocate), possibly indicating increased risk (Langer and Wolff, 1978). Platek et al. (1985) have suggested that inhalation of fibers < 5um long will not induce a fibrogenic response.

Another factor increasing the risk of asbestos-related disease in the exposed worker is smoking. In an animal study (McFadden et al., 1986), the lungs of smoke-exposed guinea pigs were seen to retain more shorter fibers than the lungs of the control (non-smoked) animals. It has been shown (Holmes and Morgan, 1980) that, ordinarily, short fibers are cleared more effectively from the lung than are long fibers, and hence, retained fiber length appears to increase with time. In the study, this

phenomenon was seen in the non-smoked animals for both tissue fibers and lavaged fibers. In both groups of smoking animals, however, the length and aspect ratio of retained tissue fibers decreased from one week to one month, and a similar process was seen in the lavaged fibers. Presumably the difference in fiber sizes between smoked and non-smoked animals reflect smoke-induced interference with the mucociliary transport and/or macrophage clearance of fibers.

In a follow-up study, McFadden et al. (1986) looked at penetration of asbestos fibers into airway walls between smoking vs. non-smoking animals and concluded that continued transport of fibers into interstitial tissues may be the reason that asbestosis can progress after cessation of exposure. Cigarette smoke increases the penetration of fibers into airway walls. This effect may play a role in the increased incidence of disease seen in smoking versus non-smoking asbestos workers.

Mechanisms of Toxicity

Pulmonary macrophages have been proposed as potential mediators of fibrotic lung disease. In asbestos-exposed humans, rats, sheep, and mice, a progressive interstitial fibrosis results, and macrophages are associated with this response (Warheit et al., 1986). The mechanism(s) through which the macrophages could

mediate the interstitial response remains undefined. If macrophages do indeed have the potential to initiate, modify, or even ameliorate the disease process, it is essential to understand the factors that control the macrophage response to the etiologic agent.

Pulmonary macrophages are largely responsible for the normal sterility of the lung and for protecting the respiratory tract against a wide variety of foreign materials. They act by ingesting and killing pathogens with lysosomal enzymes (phagocytosis), which digest bacteria. They also phagocytize non-living, insoluble dust particles and debris. Macrophages have other roles besides phagocytosis, involving both host defense and host damaging reactions. They secrete a variety of substances which interact with multi-enzyme cascades and other cells such as lymphocytes, fibroblasts, and other macrophages. Other biologically active materials secreted by macrophages include angiogenesis factor, plasminogen activator, prostaglandins, nucleosides, cyclic nucleotides, pyrogens, granulopoietins, and other factors influencing fibroblast proliferation and tumor growth.

Alveolar macrophages are large, mononuclear, phagocytic cells found on the alveolar surface. They do not form part of the continuous epithelial layer; rather, they rest on this lining made up of Type I epithelial and

Type II alveolar cells. Because the macrophages are actively phagocytic, inhaled toxic, radioactive, or carcinogenic particles become concentrated within pulmonary macrophages. What begins as a diffuse and relatively even exposure, becomes highly localized and non-uniform. "Hot spots" of high dosage are formed which may exceed the thresholds for certain effects and cause damage. Similarly, in the airways, adherence of alveolar macrophages to the epithelium may increase epithelial exposure to inhaled toxic materials. More importantly, perhaps this close association with the bronchial epithelium can lead to transbronchial transport of inhaled particles and subsequent reingestion by sub-epithelial connective tissue macrophages (Brain, 1980). These cells, like their relatives, also segregate, retain, and perhaps metabolize carcinogenic and other toxic particles.

Asbestos, glass, and other fibrous dusts all have been shown to stimulate collagen synthesis. This is thought to occur as a two-step process whereby the fiber interacts with the macrophage which release factors that stimulate local production of collagen by fibroblasts (Allison, 1974; Ross et al., 1970). Silica and asbestos present an added hazard of being cytotoxic to alveolar macrophages. Within a few minutes, they can lyse cells by direct interaction with the plasma membrane, or if

successfully ingested, in several hours cause rupture of secondary lysosomes, releasing lysosomal hydrolases into the cytoplasm (Allison et al., 1966). Release of lysosomal enzymes, particularly proteases from activated macrophages and leukocytes, promote the development of emphysema. The resulting dead macrophages can become focal points for further fibrogenesis. In addition, the particles are released anew on the alveolar surface to cause more irritation.

Inflammatory Response

Hamilton (1980) injected chrysotile fibers into the peritoneal cavity of mice to study macrophage stimulation and the inflammatory response to asbestos. The asbestos-induced macrophages appeared to have descended from recently divided precursors which indicated increased synthesis. These same macrophages also produced high levels of the neutral protease, plasminogen activator, when compared with the resident peritoneal macrophage population. It is proposed that a part of the granulomatous response to asbestos fibers might be the interaction of asbestos with monocyte-macrophages resulting in the plasmin-mediated destruction of neighboring tissue and in the liberation of inflammatory mediators. In support of the proposal that the macrophage plasminogen activator-plasmin system might be relevant for the

processes of chronic inflammation (particularly those associated with asbestosis), it has been demonstrated that low concentrations of anti-inflammatory steroids can inhibit macrophage plasminogen activator production. No such influence was found on lysozyme synthesis or on lysosomal enzyme levels (Hamilton et al., 1976). Hamilton (1976) also observed that different asbestos types induce hyperemia in skin, shorten the partial thromboplastin time of plasma and generate the release of kinins. These observations could be interrelated and are suggested as representing some aspects of the inflammatory response of the host to asbestos exposure.

Factors Found In Vitro and In Vivo

Brody (1986) explored the macrophage response to inhaled asbestos and proposed several different mechanisms of how pulmonary macrophages could participate in the disease process, including: 1) production of tissue-damaging oxygen radicals; 2) release of arachidonic acid metabolites; 3) secretion of neutral proteases such as elastase and collagenase; and 4) elaboration of growth factors that control the proliferation and/or collagen production of interstitial fibroblasts. However, he noted that while all of these secretions and factors have been shown in vitro, direct evidence of the role these macrophage products play in causing interstitial disease is

lacking. Similarly, using a brief asbestos exposure in rats of one hour to investigate basic pathogenetic mechanisms of the disease, Warheit et al. (1986), have shown that inhaled asbestos fibers activate a complement-dependent chemoattractant (chemotactic factor) that induces macrophage accumulation at sites of fiber deposition (i.e., alveolar duct bifurcations). The data supports the concept that generation of a chemotactic factor precedes the macrophage migratory response. The significance of this notion is that these cells, upon arrival at the duct bifurcations, have accumulated at anatomic locations where macrophage secretory products, such as oxygen radicals, growth factors, enzymes and arachidonic acid metabolites could mediate the pathogenesis of asbestos-induced lung disease.

Fiber Length-Dependent Cellular Effects

In an effort to understand the mechanism of fiber length-dependent cellular effects, Hesterberg et al. (1986) examined the phagocytosis and intracellular distribution of glass fibers of differing lengths in Syrian hamster embryo (SHE) cells at various times post-treatment. Glass fibers rather than asbestos were used in this study because it has been shown that milling glass fibers decreases the average length with little or no effect on the average diameter, while asbestos milling results in changes in both

dimensions (Assucaó and Corn, 1975). Both asbestos (crocidolite) and glass fibers are phagocytized by SHE cells in culture and accumulate in the perinuclear region of the cytoplasm. Milling of the glass fibers resulted in a nearly 7-fold decrease in length, and reduced phagocytosis. Phagocytized fibers were more than 2-fold longer than surface fibers in both the unmilled and milled glass fiber-treated cells which suggested that cells selectively internalized the longer fibers. Fiber length, however, did not appear to affect the migration of intracellular fibers to the perinuclear region of the cytoplasm. Even though cells treated with milled glass fibers contained a number of fibers similar to those treated with unmilled glass fibers, the resulting cytotoxicity, transformation frequency, and frequency of micronucleus induction were much less in the milled glass fiber-treated cells. Thus, fiber length appeared to affect not only the ability of fibers to be phagocytized, but also the ability of intracellular fibers to induce cytogenetic damage and cell transformation.

Fiber Deposition

The initial deposition and subsequent translocation of chrysotile asbestos were studied in the lungs of rats exposed for 1 hour in inhalation chambers. Using scanning and transmission electron microscopy of tissue fixed by

perfusion, Brody et al. (1981) determined that the majority of fibers that pass through the conducting airways deposits at the bifurcations of alveolar ducts. The farther an alveolar duct bifurcation was from its terminal bronchiole, the less asbestos was observed. The amount of asbestos present on the alveolar duct surfaces was significantly decreased 5 hours after cessation of the 1 hour exposure. Some fibers were taken up by Type I epithelial cells during the first hour of dusting, and this process continued through the 8-day period in which the animals were studied. As early as 24 hours after exposure, there was an accumulation of macrophages at the sites of initial asbestos deposition, indicating a significant cellular response in the early pathogenesis of asbestosis.

Immunoregulatory Response

The alveolar macrophage has been shown to be a potent immunoregulatory cell, as well as the primary line of defense against inhaled particulate and infectious agents. Individuals chronically exposed to asbestos fibers develop a fibrotic lung disease and are at increased risk for developing pulmonary neoplasms (Selikoff et al., 1972). In addition, these patients often present with evidence of B-lymphocyte hyperactivity manifested by hypergammaglobulinemia and autoantibody formation, and depression of cellular immune function. These

abnormalities suggest that the immune system may be involved in the pathogenesis of asbestosis. Using murine alveolar macrophages from the lavaged lungs of mice, Bozelka et al. (1986) studied the cytostatic activity of these macrophages after addition of either amosite or chrysotile fibers. After 1 hour of incubation, the cytostatic activity of chrysotile-supplemented alveolar macrophages was diminished. Extension of the culture times (4-24 hours) led to a progressive loss in the regulatory capacity of chrysotile-treated macrophages. A similar, although less pronounced pattern was also noted for the amosite-supplemented alveolar macrophages. Both fiber types were capable, in vitro, of significantly suppressing the ability of these cells to interfere with lymphocyte mitogenesis. The importance of these in vitro observations to disease pathogenesis remains to be determined by future investigations. However, humans with asbestosis manifest alterations in the peripheral blood suggestive of B-cell hyperactivity (Lange, 1980; Doll et al., 1983). It is possible, therefore, that macrophages exposed in vivo to asbestos may lose their inherent capacity to control T and B-lymphocyte responses - a finding which may be important in the pathogenesis of asbestos-related disease.

Early Response

The early response of the lung to a single exposure of amosite asbestos was examined by Davis and Dodson (1985) using SEM and correlated light microscopy in the guinea pig model. *In vivo* studies involving amphibole asbestos are particularly relevant considering that amphibole fibers have a longer retention time in the human lung than do chrysotile fibers. Moreover, a positive correlation has been shown between the number of amphibole fibers present and the grade of asbestosis. At 2 hours post-exposure, lesions consisted of discrete areas of atelectasis with an influx of neutrophils and macrophages. Free asbestos fibers were evident in affected areas. By 4 hours post-exposure, affected regions were more extensive, with phagocytic cell numbers increased both in reactive sites and in adjacent tissue. By 1-day post-exposure, the inflammatory response was well developed and encompassed wide areas of the lung. Activated phagocytes were congregated in atelectatic regions and on blood vessel walls. Numerous macrophages were present even in alveoli distant to reactive loci. The 6-day and 12-day time frames marked a subsidence of the inflammatory response. There was a notable decrease both in marginated leucocytes and in accumulations of phagocytes in tissue adjacent to affected regions.

Risk Assessment

The determination that asbestos workers faced a significant risk of asbestos-related disease prior to implementation of the new standard (0.2 f/cc) was primarily based on results of a quantitative risk assessment study performed by OSHA in 1986 (Federal Register, 1986).

Significance of Risk

OSHA is guided in its interpretation of significance of risk by the Supreme Court which recognized that any Agency determination of a particular exposure level as significant would be based largely on policy considerations (Industrial Union Department, AFL-CIO vs. American Petroleum Institute, 448 U.S. 601, 65L. Ed. 2d 1010, 100 S. Ct. 2844, 1980). The Court also indicated that the significant risk determination required of OSHA was not a mathematical straightjacket, and that OSHA was not required to support the finding that a significant risk exists with anything approaching scientific certainty. Furthermore, OSHA was free to use conservative assumptions in interpreting the data with respect to carcinogens, risking error on the side of overprotection rather than underprotection.

Risk Assessment Model

OSHA chose several well-designed and -conducted epidemiological studies of human cohorts in the workplace as its basis for quantitative risk assessment. In testimony for OSHA, Dr. Hans Weill noted: "The greatest public confidence in decision-making to reduce an environmental or occupational risk results when the data used are the product of...relevant human populations."

OSHA chose not to use animal studies to predict quantitative estimates of risk from asbestos exposure since many high quality human studies were available. The studies cited below were conducted in actual workplace situations. However, OSHA does believe that the animal studies provide valuable qualitative information on asbestos-related disease. For example, animal studies show that all commercial asbestos types can cause cancer and pulmonary fibrosis (Reeves et al., 1974). Animal studies also indicate that longer, thinner fibers may have greater carcinogenic potency than short, coarse fibers. A study by Brody (1986) noted that, "shorter fibers were cleared earlier leaving the longer, thinner fibers that appear to remain entrained in the lung interstitium. Thus, it seems likely that these are the fibers that will interact with the various pulmonary cells and dictate future pathogenic events."

For lung cancer, OSHA chose a linear model to describe the relationship between the excess relative risk of lung cancer and asbestos exposure (dose). Relative risk is defined as the ratio of the mortality rate of exposed persons to the mortality rate of equivalent unexposed persons. Relative risk is frequently approximated by the standard mortality ratio (SMR), which is the observed number of deaths in the exposed population divided by the number of deaths that would be expected in the exposed population. Expected deaths are usually derived from the specific age, sex, and calendar year mortality rates in the comparison population.

Data for the calculation of the dose-response relationship for lung cancer and asbestos exposure was derived from eight studies (Selikoff et al., 1979; Seidman, 1984; Henderson and Enterline, 1979; Weill et al., 1979; Finkelstein, 1983; Peto, 1980; Dement et al., 1982; Berry and Newhouse, 1983). Similarly, a linear, dose-response model was used to describe the relationship between the absolute risk of mesothelioma and exposure. Absolute risk is calculated as observed deaths divided by the number of person-years at risk. It was believed that use of SMR's or relative risk was not appropriate for mesothelioma because the expected number of deaths in a cohort would be close to zero due to the rarity of the disease. Six studies were

used to develop the dose-response relationship of the linear model (Selikoff et al., 1979; Seidman et al., 1984; Finkelstein, 1983; Peto, 1980; Weill et al., 1979; Dement et al., 1982). For gastro-intestinal cancer, OSHA considered a simple risk model in which GI cancer risk was assumed to be equal to 10% of the lung cancer excess risk. Other cancers were grouped under the same excess risk category as GI cancer (i.e. laryngeal, buccal cavity, kidney, pharyngeal) due to the difficulty in obtaining good data to quantify risk. Finally, for asbestosis, OSHA again employed a linear model in the prediction of risk from asbestosis. According to the agency, exposure over a working lifetime (45 years) to the 2 f/cc level will result in approximately a 5% incidence of asbestosis. Reducing the exposure to 0.2 f/cc will result in a lifetime asbestosis incidence of 0.5%. Three studies (Berry et al., 1979; Berry and Lewinsohn, 1979; Finkelstein, 1982) were used to support this determination.

Current Control Techniques

Based on an analysis of the industries affected by their revised PEL, OSHA has concluded that compliance with the 0.2 f/cc limit is feasible most of the time through the use of wet methods, engineering controls, and good housekeeping practices. Affected industries include primary manufacturing, secondary manufacturing, automotive

brake and clutch repair, shipbuilding and ship repair, and construction. Environmental monitoring is required to determine the extent to which current control techniques are able to limit exposures to 0.2 f/cc, thereby eliminating the need to use respiratory protection in affected work areas. For those operations where current control techniques cannot be effectively applied, (e.g. textiles, nuclear rip-out, cutting asbestos-cement pipe, sanding asbestos-cement sheet, etc...), respiratory protection must be used.

Statement of the Problem

When current techniques are inadequate to control asbestos exposures below 0.2 f/cc during operations, proper respiratory protection must be provided to and used by affected workers. In order to adequately assess the inhalation hazard, the Industrial Hygienist (IH) must have information on exposure variation during asbestos abatement operations. With this information the IH can better select appropriate respiratory protection for abatement workers. Exposure variation cannot be determined by the current standard method (NIOSH 7400) approved by OSHA to measure average airborne asbestos concentrations. However, with a few modifications to the 7400 method, exposure variation can be measured. The Hi-Vol (HV) method was developed to provide information on short-term exposure variation of

airborne asbestos fibers. A method not approved by OSHA, which purportedly can measure airborne asbestos fibers from a real-time fibrous aerosol monitor (FAM), was also available to study exposure variation alongside the HV method.

In keeping with their zero threshold concept for asbestos exposure, NIOSH together with EPA recommended that only supplied-air respirators be used for asbestos abatement work (OSH Newsletter, Maine Labor Group On Health, 1986). In addition, they recommended that the first four respirators on the OSHA list (see Appendix 2) be restricted to use only for glove bag removal situations where the air is free of any measurable concentration of asbestos. To date, OSHA has resisted proponents both for underprotection (no respiratory protection needed) and overprotection (supplied-air only), but more information is needed to better characterize exposure levels during typical abatement activities so that reasonable choices can be made in the selection of respiratory protection. This study is necessary to provide the Industrial Hygienist with a clearer understanding of exposure levels in typical abatement environments.

Objectives

Three specific research objectives of this study were:

(1) To develop a modified Hi-Vol method capable of examining short-term variation in asbestos exposure during removal and related activities.

(2) To examine the correlation between the short-term results obtained from the HV method and the direct-readout data from the FAM.

(3) To relate the findings of short-term variation in asbestos levels to criteria for specification of industrial respiratory protection.

This research should provide the practicing I.H. with clearer information on variation in asbestos levels over a shift, thereby aiding in the selection of proper respiratory protection for abatement workers. In addition, the appropriateness of employing the FAM in an abatement environment to determine airborne asbestos levels will be carefully studied.

METHODS

Occupational asbestos exposures are normally determined by personal sampling techniques which involve outfitting workers with belt-mounted portable sampling pumps and filter cassettes located in the workers' breathing zone. Maximum personal sampling flowrates are typically less than 4 LPM. Consequently, sample collection periods of >100 minutes are required to collect the minimum sample volume of 400 L recommended by the NIOSH 7400 method.

Sample results using the standard (NIOSH 7400) method represent integrated fiber concentrations over the sample period, and yield little information on actual variation in fiber concentrations during the sample period. Maximum fiber concentrations are an important IH consideration when selecting respiratory protection for asbestos workers. Therefore, a new high volume (Hi-Vol) method was developed for this study to examine the range of short-term variation in fiber concentrations during selected abatement activities. Specifically, the Hi-Vol method involved modification of the NIOSH 7400 method. Activities selected for this study involved scraping asbestos insulation from ceilings and removing asbestos-backed tiles from floors.

Development of the Hi-Vol method involved modification of the NIOSH 7400 method.

NIOSH Method: 7400

On February 15, 1984, NIOSH issued a revised standard analytical method for sampling and analyzing airborne asbestos fibers (NIOSH, 1984). The 7400 method revised the Physical & Chemical Analysis Method (P & CAM) 239 adopted in 1975. The 7400 method was officially adopted by OSHA in 1986 as the approved method for sampling and analyzing airborne asbestos fibers. The 7400 method is intended to increase sensitivity and minimize "within-lab" and "lab-to-lab" variations in fiber counts. It incorporates the use of a 25 mm diameter membrane filter instead of the 37 mm filter used in the P & CAM 239 method. This smaller diameter filter improves method sensitivity (from 0.1 to 0.02 f/cc) by concentrating collected fibers in a smaller total filter area. The estimated limit of detection (LOD) for the 7400 method is 7 fibers/mm^2 of filter area. The working range is 0.02 f/cc (for a minimum loading of 100 f/mm^2) to 1.25 f/cc (for a maximum loading of 1300 f/mm^2). This working range reflects the recommended maximum and minimum air volumes collected during sampling. A maximum volume of 1920 liters (L) is recommended for minimum loading of 100 f/mm^2 of filter area, and a minimum volume of 400 L is recommended for a maximum

loading of 1300 f/mm^2 of filter area. Because of past inaccuracies associated with low fiber counts, the minimum loading was increased to 100 f/mm^2 of filter area in order to achieve a total count of 80 fibers per 100 fields. This level yields a total coefficient of variation (CV_T) of 0.13 (see Fig. 1), which is the minimum acceptable overall precision using the A-counting rules set forth in the analytical method.

Other features of the 7400 method are listed below. Actual materials and equipment used in the study are shown in parentheses.

(1) The use of an optical graticule (Walton-Beckett G-22, Graticules Ltd., Kent, England), rather than a reticule, to standardize the field area observed through the eyepiece.

(2) The use of a standard phase shift test slide (HSE/NPL) to calibrate the phase-contrast microscope (Nikon Labophot Pol) and ensure that all fibers down to a set diameter (0.25 um) are visible.

(3) The replacement of the dimethyl phthalate/diethyl oxalate clearing technique with an acetone

vapor-triacetin method (Mallinckrodt Inc., Paris, Kentucky). This method is faster, yields a more permanent analytical specimen, and avoids using the potentially carcinogenic phthalate ester.

(4) An expanded flowrate range of 0.5-16 LPM.

(5) Substitution of a 25 mm filter and cassette assembly with improved geometry and decreased electrostatic properties (Millipore 1.2 um cellulose ester with 50 mm conductive extension cowl, Molsheim, France).

Hi-Vol Method

A normal sampling strategy for collecting asbestos fibers in the abatement area involves a calibrated high volume pump connected by vacuum tubing to a three-piece 25 mm cellulose ester filter cassette. Sample time normally ranges from 1-3 hours, depending upon airborne particulate concentrations. Typical Hi-Vol flowrates range from 4-16 LPM.

The Hi-Vol method used for sampling asbestos fibers in this study is a modified version of the 7400 method. The Hi-Vol method uses the same material and equipment as the 7400 method, and differs only in minimum collection time, air volume sampled, and total number of filter fields counted. A collection time of 10 minutes was chosen to

allow examination of short-term exposure variation to asbestos fibers. A flowrate of 10 LPM was used, which yielded a total volume of 100 L for each sample.

The minimum recommended total volume in the NIOSH 7400 method is 400 L. That total volume was reduced to 100 L in the Hi-Vol method, and the total number of filter fields counted was doubled from 100 to 200. Using these parameters and the collection area of a 25 mm diameter filter, expected fiber loading was determined by the equation:

$$E = \frac{(t)\ (Q)\ (L)\ 10^3}{Ac} = 52\ f/mm^2$$

where E = expected fiber loading

t = time = 10 minutes

Q = flowrate = 10 LPM

L = fiber concentration = 0.2 f/cc

A_c = collection area = 385 mm^2

The total fiber count was determined with the equation:

$$F = (E)(n)(A_f) = 82 \text{ fibers}$$

where F = total fiber count

E = expected fiber loading = 52 f/mm^2

n = number of fields counted = 200

A_f = graticule field area = 0.00785mm^2

The total fiber count of 82 fibers per 200 fields, which would be achieved at the OSHA PEL of 0.2 f/cc, yields a

total coefficient of variation of 0.13. This meets the minimum precision requirement of the 7400 method using the A-counting rules.

Sample Collection and Analysis

A Gilian Air Con 520 air sampling pump (Gilian Inst. Corp., Wayne, N.J.) with a variable flowrate range of 2 - 25 LPM was used in this study. Three constant flow pumps from BGI Inc. (Waltham, MA) were also used. All pumps were used at a 10 LPM flowrate. A Gilian-HFS Pump Calibrator (model 1 HCP HL 300) was used to calibrate all pumps, and a Mini-Master Flowmeter (Dwyer Inst. Inc, Michigan City, IN) was used to periodically check flowrates in the field.

After collection, filters were mounted whole on labeled glass slides (Gold Seal Micro Slides 3" x 1") using the acetone-triacetin mounting technique. A 125 ml Erlenmeyer flask (Pyrex) was filled to the 75 ml mark with acetone (AR Lot # 2440) and stoppered with a single-holed rubber stopper. A hollow piece of glass tubing was inserted through the stopper and bent outside the flask at an angle of 45° to the horizontal. The flask was then placed on a hotplate (VWR Dylatherm) inside the lab hood, and heated until a steady acetone vapor stream was generated. Glass slides with the filter samples were then

exposed to the vapor stream for a few seconds until cleared. The cleared filter was then treated with 2-3 drops of triacetin from a 22 gauge needle (Becton-Dickinson, N.J.) and covered immediately with a 25 x 25 mm Micro cover glass (VWR Scientific Inc., San Francisco, CA). The edges of the cover slip were then glued to the slide with a generic brand of clear lacquer nail polish. All samples were analyzed with a phase-contrast microscope (Nikon Labophot - POL, Allen & Associates, Scottsdale, AZ) using a 40X phase objective and 10X eyepiece for a 400X magnification.

A study was designed to assess any variation attributable to sampler co-location in an abatement environment. It was important to ascertain whether co-located samples in an abatement environment would exhibit significant variation in asbestos fiber concentrations as a function of their location. Four pumps were run simultaneously at 10 LPM and spaced approximately 1 ft apart. This spacing was found to be more than sufficient to preclude disruption of normal flow patterns into the filter cassettes attached to each pump. The effect of capture velocity vs. distance from a cassette face can be examined with an equation by Dalla Valle (1946).

$$V = \frac{Q}{10X^2 + A}$$

where V = capture velocity

Q = pump flowrate

X = capture distance

A = orifice area

For a constant Q = 10 LPM and A = 5.4 x 10^{-3} ft^2 (25 mm diameter cassette orifice), the capture velocity (V) is seen to decrease inversely as a function of the square of the capture distance (X).

Capture Distance (X) inches	Capture Velocity (V) fpm
0.5	15.4
1.0	4.7
1.5	2.2

This indicates that for a filter cassette spacing of 12", the capture velocity from the four pumps would have no effect on normal flow patterns into co-located sample cassettes.

Fibrous Aerosol Monitor (FAM)

The process of sampling and analyzing airborne asbestos is currently based on integrated techniques which yield no information on short-term variation, and involve a delay in obtaining sample results. While this delay is

acceptable in most cases, it would be useful to have a direct read-out monitor that would reduce the delay in obtaining results and also provide a means of characterizing variations in fiber concentrations with time.

The fibrous aerosol monitor (FAM) was developed under a contract jointly supported by NIOSH, EPA, and the Bureau of Mines in 1977 (Lilienfeld and Elterman, 1977). To date, OSHA has not approved the FAM as a recognized method for counting asbestos fibers. A portable instrument about the size of a medium suitcase, the FAM weighs 12.5 kg and can be operated continuously from an electrical outlet or for up to 4 hours on its rechargeable battery. The operation of the FAM is based on detection of light-scattering particles passed through a rotating electric field. The ferruginous asbestos fibers rotate while aligning with an alternating field and scatter light from a continuous wave helium-neon laser beam. The light pulses are detected by a photomultiplier tube and digitally displayed as fiber counts. To prevent the FAM from counting spurious pulses, a count is registered only if pulses are synchronous with field oscillations and persist for the time required for a fiber to travel through the detection region. The minimum length of fiber counted is set with a `ratio' control, which sets a minimum threshold

for the ratio between the peak amplitude and average area of the pulses associated with a fiber. The ratio control is calibrated so that for straight fibers, the setting corresponds to the nominal fiber length in micrometers (um). In this study, the FAM's ratio control was set at 5.0 for all fiber types.

The amplitude of the pulse is also analyzed. The pulse is not counted as a fiber if its threshold is below a set amplitude. This prevents random noise from being erroneously interpreted as fibers. The threshold is set with the `amplitude' control on the FAM. A setting of 0.5 is recommended by Monitoring Instruments for the Environment (MIE), and was used for all fiber types.

The FAM is calibrated by MIE against a light microscope count of amosite asbestos collected on a membrane filter. The FAM is set with the ratio control at 5.0 and the amplitude control at 0.5 for this calibration. The FAM also has a control which adjusts the photomultiplier tube gain; this was set to the `normal' position for the study.

An indicator light on the FAM shows whether the photomultiplier tube is overloading because of too much scattered light. A second indicator light shows when the count rate is so high that coincidence counting within the detection volume is likely to cause a significant error.

The FAM displays the measured fiber concentration on a digital display at the end of the sampling period, which may be selected as 1, 10, 100, or 1000 minutes. During the sampling period, the FAM displays the number of fibers as they are counted. In this study, the sampling period was selected as 10 minutes.

A 37 mm cellulose ester filter cassette attached to the FAM collects fibers and other non-fibrous particles after they have passed through the detection region. This filter was analyzed and used as an additional source of integrated exposure data.

Statistical Evaluation

Statistical analysis on paired data was computed manually on a 10-digit LCD Scientific Calculator (Radio Shack EC-499), and verified later using a statistical program (SYSTAT). A paired samples t-test was computed both on the Hi-Vol (HV) vs. FAM results, and the co-located Hi-Vol results (i.e., HV1 vs. HV2). Correlation coefficients were computed on the HV vs. FAM data to examine the relationship between the two sampling methods. Significance of results was determined by the ANOVA technique (one-way analysis of variance, $p \leq 0.05$)

Fifty-nine paired short-term samples were collected during selected abatement activities using the FAM and modified Hi-Vol method. Seven blank filters were analyzed

from the filter batch to determine possible contamination and background levels of asbestos. Forty-eight samples including four blanks were collected from concurrent sample periods using four co-located pumps and analyzed in an abatement environment characterization study.

Asbestos concentrations determined during abatement activities covering a 16 month period were obtained from two companies (A & B). Monthly average concentrations were calculated and compared against results from selected abatement activities monitored during the study. Lettered companies were used in deference to requests made by the companies' management not to use their names.

RESULTS

Short-term variation of asbestos fiber levels during selected abatement activities was assessed using the modified Hi-Vol (HV) method at Good Samaritan Hospital in Phoenix, Arizona. Correlation of the data collected by the HV and FAM methods over identical periods was evaluated by regression analysis. A paired-samples t-test was used to determine whether equivalence of concurrent sampling results existed between the two methods.

A paired-samples t-test was also used to compare asbestos fiber levels determined with four high volume pumps sampling concurrently at closely proximated locations in the same abatement area.

Correlation Results

The extent of the relation between results obtained from the FAM and HV methods was quantified by the correlation coefficient $|r|$. For the abatement activity involving scraping sprayed-on insulation from ceilings (Fig. 2,3), correlation is graphically displayed as a scatter plot of the paired data. Numerically, the closer $|r|$ is to 1, the more closely related the variables. If $|r|$ is 0, the variables are unrelated.

A poor correlation ($r = 0.447$) for HV and FAM results was seen for the data collected on July 12 (Fig. 2), and a weaker correlation ($|r| = 0.121$) on July 15 data (Fig. 3). A weak correlation was also seen in the data collected on August 1 & 6 when the workers switched to removing asbestos tile from the floor (Fig. 4,5). Although not as divergent as the average ceiling data, the floor data was consistent in exhibiting a weak correlation between the HV and FAM results.

Paired-Samples t-Test

Examination of HV vs. FAM paired-data (Appendix 3) collected from concurrent samples was accomplished using the paired-samples t-test (Table 1). The mean difference was both large and negative for each sample day, which indicated that higher fiber levels were measured by the FAM than by the HV throughout the study. A significance level of 0.05 was used to determine acceptance or rejection of the stated hypothesis - Ho: mean HV fiber levels from were no different than mean FAM fiber levels during selected abatement activities. Table 1 illustrates the p-values computed for each sample day. The highest p-value (0.032) was reported on August 5 when only (3) concurrent samples were collected. On August 1 and 6, when the highest number (10) of concurrent samples was collected, the highest p-value reported was 0.002.

Results from four (4) co-located samplers (Table 2; Appendix 4) were also evaluated using the paired-samples t-test. Four high volume pumps with membrane filters spaced 1 ft. apart were operated concurrently at 10 LPM for 10 minute intervals in an abatement area where amosite asbestos bricks were being removed. Mean differences between all sampler data were small. P-values were large, which indicated acceptance of the hypothesis - Ho: no difference in mean results between the four samplers. This data demonstrates both the uniform distribution of fibers in the abatement environment, as well as the consistency of the sampling method.

Other Statistical Parameters

Paired data from the FAM and HV methods was quantified by five parameters - minimum value, maximum value, mean, median, and standard deviation (Table 3,4). Sample means determined using the FAM and HV data were compared to integrated sample results collected at the same time using the standard (NIOSH 7400) method (Appendix 5), which was also employed as a reference sampling technique during the study.

Close agreement in mean fiber levels was observed between the HV and standard methods (Appendix 5). FAM results showed consistently higher mean fiber levels,

differing in most cases by an order of magnitude from the membrane filter techniques.

A 37 mm membrane filter cassette attached downstream of the FAM's detection region was employed as a quality control check on the real-time readings. On two selected days the FAM filter (FAMF) was used to collect fibers from the abatement area during the entire sampling period. The filters were analyzed and compared to mean fiber levels determined with the other methods (Appendix 6). The (3) membrane filter techniques (FAMF, HV, and 7400) were seen to correlate well with each other, whereas the FAM differed by an order of magnitude.

Distribution of asbestos fiber concentrations (Appendix 7) for all Hi-Vol sample data revealed no levels > 0.10 f/cc. Graphically, this is shown (Fig. 6) for both selected abatement activities. The ordinate axis was scaled from zero to 0.2 f/cc, the current standard (PEL) for exposure to airborne asbestos fibers. In contrast, when the FAM was used to measure the same environment (Appendix 7), seventy-two per cent of its reported fiber levels were above the action level (0.1 f/cc) of the current standard. The action level signals employers to institute increased surveillance, monitoring and control of the environment. The ordinate axis for the FAM data (Fig. 7) was scaled from zero to 5 f/cc to reflect the higher

fiber levels reported. The variation in levels between the FAM and HV methods is shown in Appendix 8, where variation is seen to range from 10X greater (FAM vs. HV) to 105X greater. Variations > 1000X seen on August 5 were discarded because of the low number (3) of paired-samples collected that day.

Finally results of asbestos fiber levels from two different companies engaged in similar abatement activities (Appendix 9) are displayed to show similar discrepancies in reported fiber levels between the FAM and HV method. Again, all membrane filter techniques employed during similar abatement activities appear to yield equivalent results.

TABLE 1. - PAIRED-SAMPLES T-TEST ON HV vs. FAM

Paired-Sample t-test	Mean* Difference	SD* Difference	PROB p-value
HV vs. FAM			
July 12	-0.491	0.240	0.001
July 14	-0.950	0.762	0.010
July 15	-0.251	0.239	0.014
July 16	-0.063	0.050	0.029
July 18	-0.636	0.241	0.004
August 1	-2.551	1.236	0.000
August 5	-2.293	0.723	0.032
August 6	-0.719	0.538	0.002

*Values reflect units in concentrations of f/cc.
Mean Difference = HV - FAM (mean values)

TABLE 2. - PAIRED-SAMPLES T-TEST ON CO-LOCATED SAMPLERS

Paired-Sample t-test	Mean* Difference	SD* Difference	PROB p-value
HV1 vs. HV2	0.001	0.004	0.653
HV1 vs. HV3	0.000	0.004	0.789
HV1 vs. HV4	0.003	0.010	0.350
HV2 vs. HV3	-0.001	0.004	0.510
HV2 vs. HV4	0.002	0.009	0.440
HV3 vs. HV4	0.003	0.006	0.127

*Values reflect units of concentration in f/cc.

TABLE 3. - HI-VOL DATA ANALYSIS (f/cc)

Date	Cases	Min	Max	Mean	Median	SD
7/12	8	0.030	0.100	0.061	0.058	0.020
7/14	8	0.010	0.036	0.019	0.018	0.009
7/15	9	0.002	0.030	0.012	0.010	0.009
7/16	6	0.002	0.022	0.009	0.004	0.009
7/18	5	0.025	0.030	0.028	0.030	0.002
8/1	10	0.015	0.083	0.040	0.040	0.026
8/5	3	0.000	0.001	0.000	0.000	0.001
8/6	10	0.016	0.028	0.022	0.020	0.004

TABLE 4. - FAM DATA ANALYSIS (f/cc)

Date	Cases	Min	Max	Mean	Median	SD
7/12	8	0.12	0.8	0.553	0.58	0.231
7/14	8	0.15	2.4	0.969	0.8	0.767
7/15	9	0.05	0.78	0.262	0.16	0.237
7/16	6	0.02	0.15	0.072	0.09	0.048
7/18	5	0.42	1.06	0.664	0.64	0.24
8/1	10	0.83	4.56	2.591	2.53	1.225
8/5	3	1.61	3.05	2.293	2.0	0.723
8/6	10	0.3	2.0	0.74	0.58	0.536

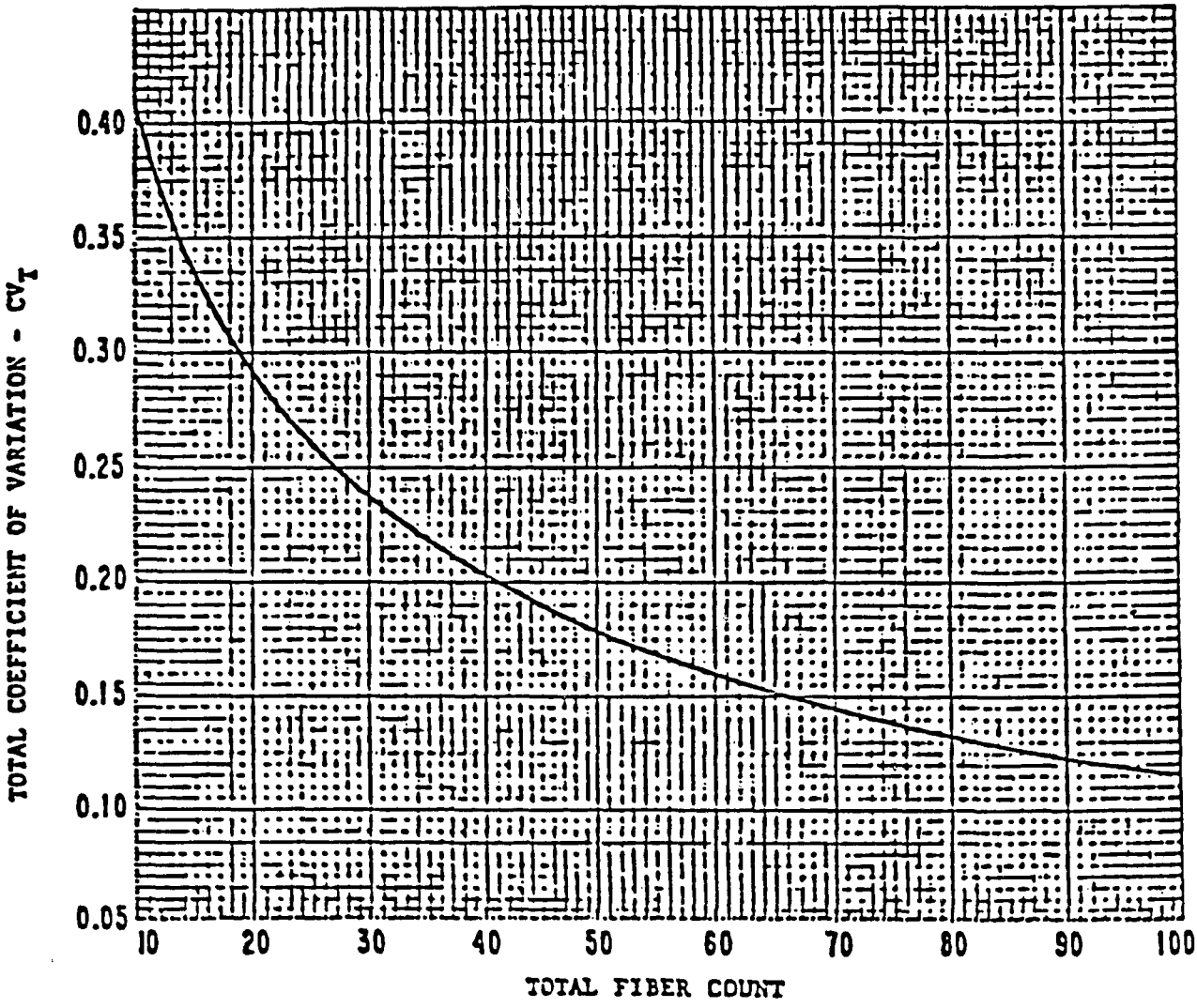

Figure 1. Total coefficient of variation as a function of total fiber count (including pump error)

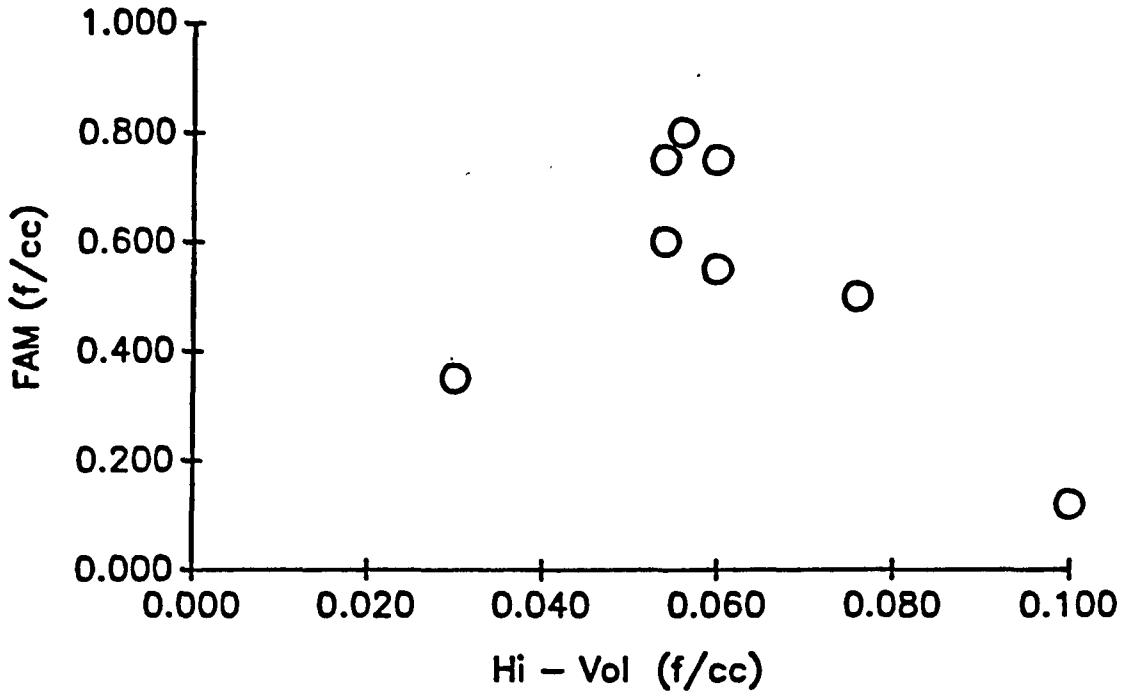

Fig. 2. FAM - HV paired-data collected July 12, 1986 at Good Samaritan Hospital during an abatement activity that involved scraping asbestos insulation from ceilings.

r = 0.447

p-value = 0.266

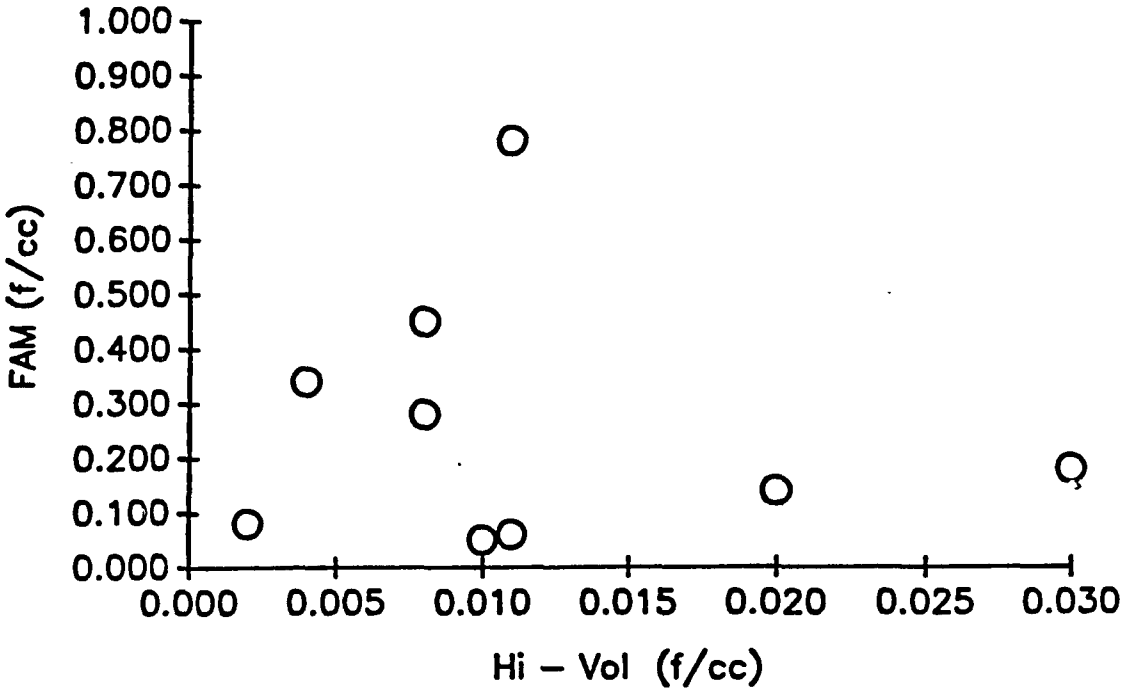

Fig. 3. FAM - HV paired-data collected July 15, 1986 at Good Samaritan Hospital during an abatement activity that involved scraping asbestos insulation from ceilings.

r = 0.121

p-value = 0.756

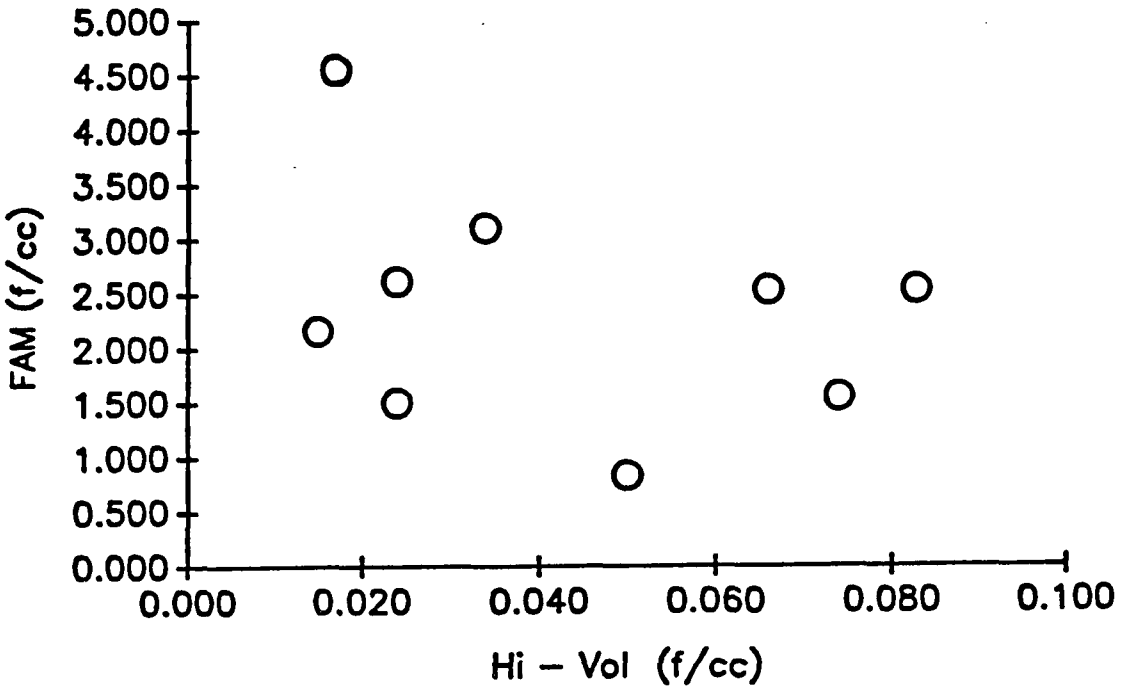

Fig. 4. FAM - HV paired-data collected August 1, 1986 at Good Samaritan Hospital during an abatement activity that involved machine- and hand-stripping of asbestos-backed vinyl floor tile.

r = 0.430

p-value = 0.215

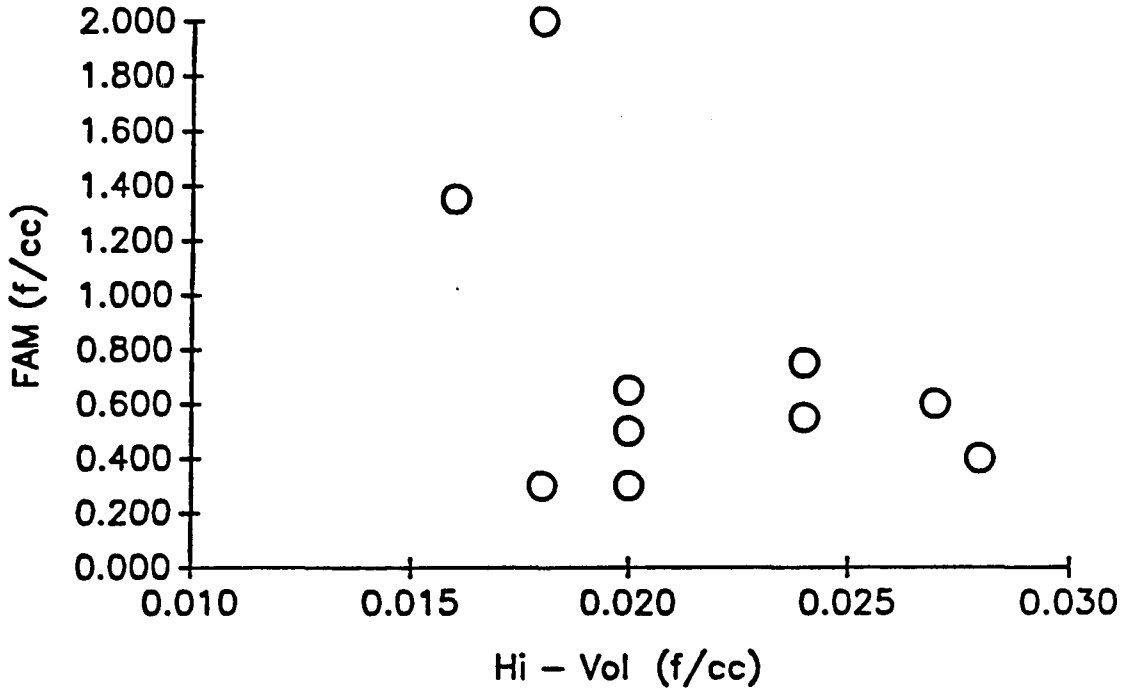

Fig. 5. FAM - HV paired-data collected August 6, 1986 at Good Samaritan Hospital during an abatement activity that involved machine- and hand-stripping of asbestos-backed vinyl floor tile.

r = 0.437

p-value = 0.207

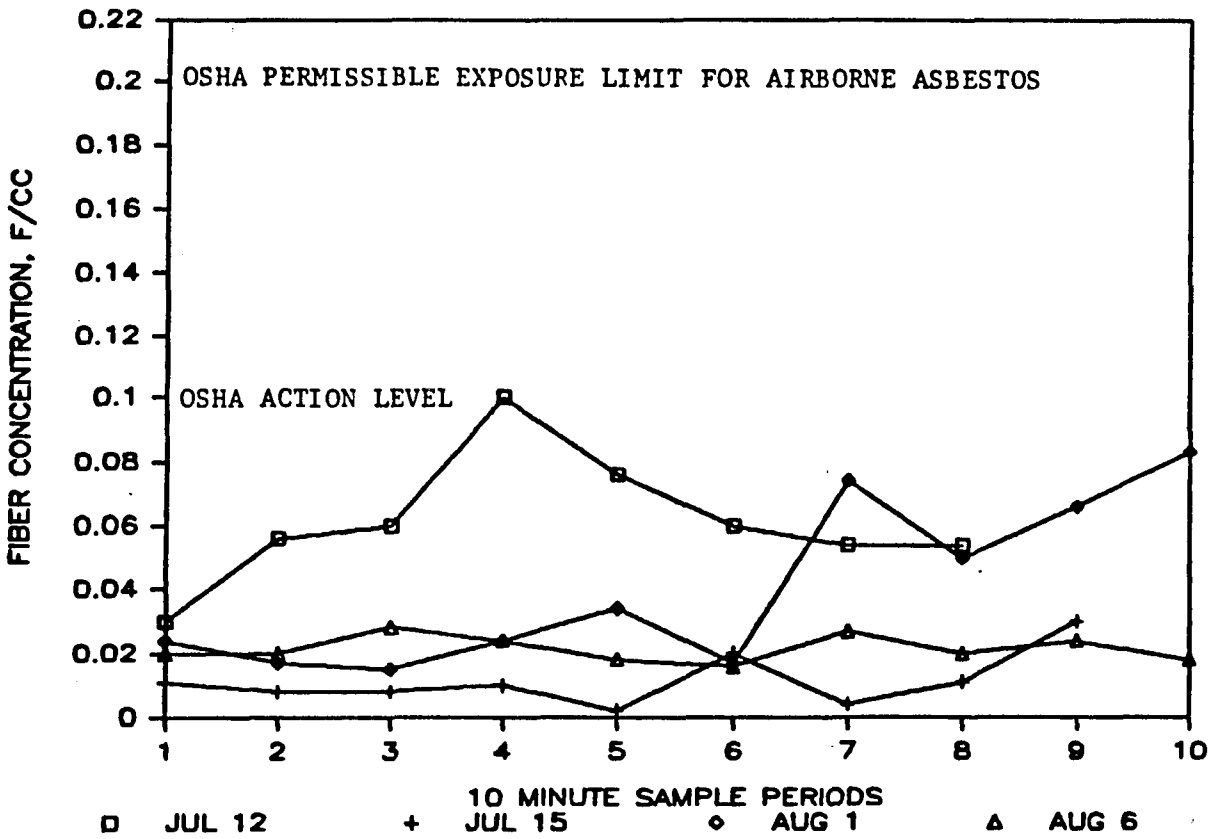

Fig. 6. Airborne asbestos fiber levels determined with the HV method during Ceiling Scraping (Jul 12 & 15)/Tile Removal (Aug 1 & 6) abatement activities. The ordinate axis reflects the current OSHA PEL (0.2 f/cc) and Action Level (0.1 f/cc) for airborne asbestos.

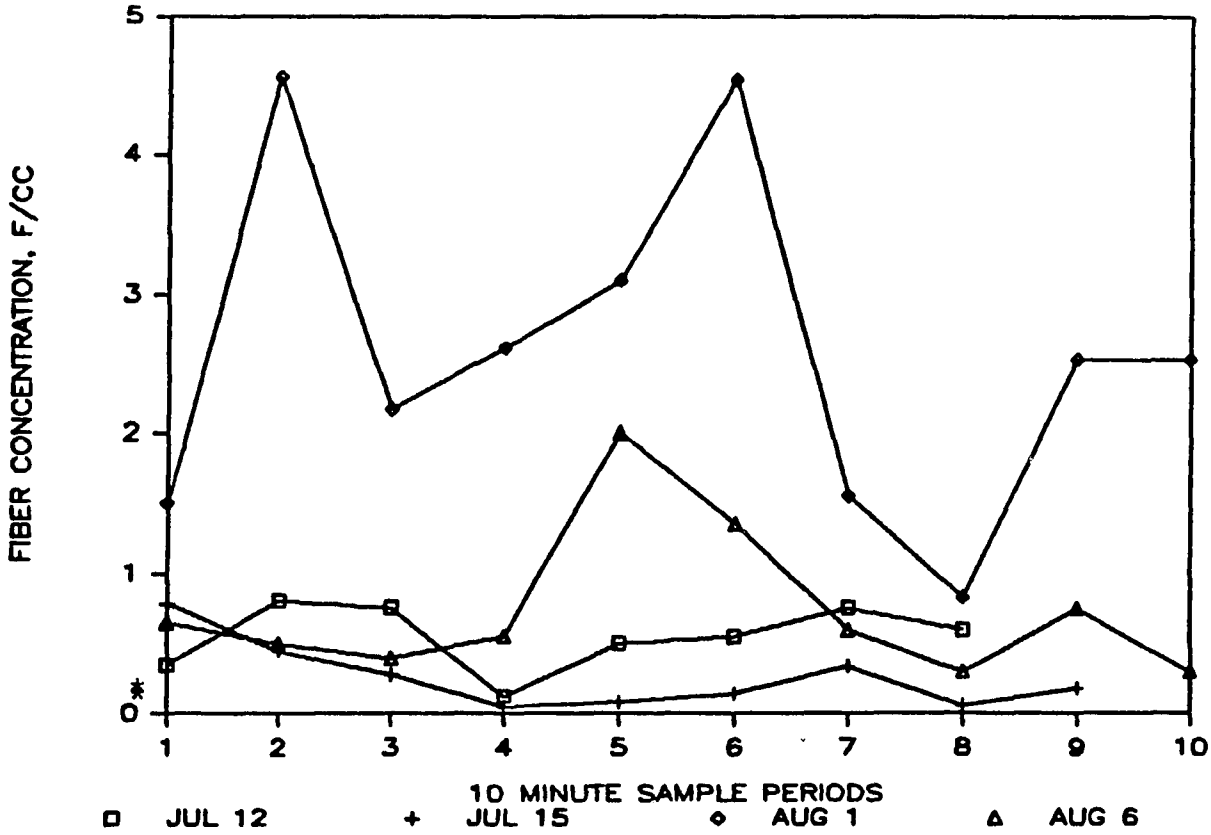

Fig. 7. Airborne asbestos fiber levels determined with the FAM. * denotes the positions of the OSHA PEL and Action level, respectively. The top portion of the character represents 0.2 f/cc while the bottom part reflects the Action Level (0.1 f/cc).

DISCUSSION

Paired-data reported by this study was collected during the summer of 1986 from an asbestos abatement job at Good Samaritan Hospital in Phoenix Arizona. The individual and mean values of airborne asbestos fiber levels collected with a fibrous aerosol monitor (FAM) and high volume (HV) method did not show good correlation. FAM data was consistently higher than HV data for both individual and mean values. Interference from non-fibrous dust particulate was examined as a possible contributing factor to the higher FAM detected levels. HV data was low relative to both FAM levels and the current OSHA standard (0.2 f/cc) for airborne asbestos fibers. Correlation among the mean values of the membrane filter methods (HV, standard, and FAM filter) compiled during the study was strong. Comparison data compiled by a power company and an asbestos abatement contracting company also exhibited good correlation with HV data for means, medians, and standard deviations.

Short-term variation seen in the HV data was small in comparison to time-weighted average (TWA) data collected over the same sample period. Based on the results of this study, options for selection of appropriate respiratory

protection are more extensive than are currently suggested by NIOSH and the EPA.

Correlation of FAM vs. HV Methods

Eight short-term (10 min) paired samples were collected on July 12, 1986 (Appendix 3) using the FAM and HV methods. Field data was collected during wet removal of asbestos insulation from ceilings by scraping. It displayed a poor correlation between the FAM and HV methods (Fig. 2, r = 0.447). Similar results were observed for all other field data collected during this study. The weakest correlation occurred on July 15 (9 pts.) which also involved removal of asbestos insulation from ceilings (Fig. 3, r = 0.121). Additional sampling during asbestos ceiling insulation removal occurred on July 14 (8 pts.) and July 16 (6 pts.). Correlations for these two days were r = 0.341 and r = 0.161, respectively.

Data collected during the following four days reflected results from an abatement activity involving removal of asbestos backed floor tile by machine stripping and hand scraping. Poor correlation was again observed among ten paired-data points collected with the FAM and HV methods on both August 1 and 6 (Figs. 4,5), with coefficients r = 0.430 and r = 0.437, respectively. July 18 (9 pts.) and August 5 (3 pts.) had measured correlation coefficients of r = 0.484 and r = 0.819, respectively. The

strong correlation indicated by the August 5 data was deceptive because only three paired-data points were collected and reported, and two of them showed HV fiber levels of zero. The third HV data point had a value of 0.001 f/cc. In contrast, all three FAM data points had values above 1.5 f/cc. The data spread in this case indicated an anomalous association between the two variables.

Other researchers have assessed correlation between the FAM and membrane filter methods using controlled and actual workplace conditions. During a controlled study in the laboratory, Page (1980) generated both fibrous (anthophyllite asbestos) and non-fibrous (Arizona road dust - ARD) particulate in a dust chamber to assess the FAM's fiber response and particulate response relative to corresponding results from membrane filter analysis of fiber counts. He found excellent FAM-membrane filter count correlation from two FAM units when only anthophyllite asbestos fibers were generated in the dust chamber ($r = 0.94$ and $r = 0.93$). However, when only non-fibrous ARD particulate was generated in the dust chamber, there was poor FAM-membrane filter count correlation due to the spurious counting of non-fibrous ARD by the FAM.

Iles and Shenton-Taylor (1986) carried out both laboratory and factory tests of the FAM using chrysotile

asbestos fibers. They found good correlation between the FAM-membrane filter measurements under laboratory conditions when a chrysotile aerosol was generated in a pyramid-shaped dust box by a Timbrell generator. However, factory tests covering three different manufacturing processes (asbestos cement, textiles, and friction materials) showed poor correlation between FAM-membrane filter data.

Bulk sample analysis at Good Samaritan Hospital revealed 30 - 40% chrysotile asbestos for both abatement activities studied. The type of asbestos seems to be important when using the FAM to count fibers insofar as fiber shape may contribute to erroneous counts in situations where other airborne dusts are present in varying amounts (Iles and Shenton-Taylor, 1986). The fundamental assumption concerning fiber shape is that it is approximately cylindrical. In general, fibrous aerosols such as fibrous glass and the amphibole fibers (amosite, crocidolite, and anthophyllite) meet this criterion. Chrysotile fibers do not, being neither straight nor cylindrical. Light-scattering from straight, cylindrical fibers in the FAM can be theoretically approximated (GCA, 1983) and counted as pulses much better than light scatter from particles such as chrysotile, which show substantial departure from ideal cylindrical geometry. The poor FAM-HV data correlation could be partly

attributable to the fiber type (chrysotile) sampled in the study.

Sample Collection Variation

An abatement area study was conducted to assess paired-sample location variability, and to determine HV method variation. The study was conducted at a site where abatement workers were removing bricks containing 80 - 85% amosite asbestos as determined by bulk sample analysis. Four high volume pumps collected concurrent samples at different locations in the abatement area and showed very strong correlation between individual short-term data (Appendix 4). A paired-samples t-test calculated for each combination of paired pumps (Table 2) revealed an insignificant difference in mean fiber levels, suggesting uniformity over co-located sample distances in the abatement environment, and low variability in the HV method.

Comparison of FAM vs. HV Method

FAM data was consistently higher than corresponding HV data (Tables 3,4) for both individual short-term values and mean values. FAM individual short-term data ranged from 1.1X to 268X HV data (Appendix 8). In that same appendix, average variation of FAM levels can be seen to range from 10X to 105X higher than corresponding HV levels. August 5 data was not included due to the extremely low levels

reported by the HV method. In factory tests conducted by Iles and Shenton-Taylor (1986), FAM mean levels was reported to vary from 4X to 65X the corresponding membrane filter levels.

Fiber levels are displayed in Figures 6 & 7 to show variation above OSHA compliance limits for data collected with the FAM and HV. The highest individual short-term fiber level observed with the HV method coincided with the OSHA action level (0.1 f/cc), which indicates to the employer a need for increased surveillance of the workplace. In contrast, 78% of the individual short-term fiber levels detected by the FAM exceeded the OSHA PEL (0.2 f/cc), which indicates occupational overexposure to asbestos fibers and the need for implementation of control measures to reduce or eliminate the exposure.

Significance of variation between FAM and HV data was determined by a paired-samples t-test (Table 1). The mean difference (HV - FAM) between FAM and HV values is large and negative, which indicates higher FAM data relative to HV data. These differences are recorded as being either significant (p-value ranging from 0.01 - 0.05), or highly significant (p-value ranging from 0.001 - 0.01).

Another illustration of variation between FAM and HV levels is described by a distribution tally of concentration ranges (Appendix 7). Sixty-seven asbestos fiber concentra-

tions determined by each method (FAM & HV) were distributed across nine ascending concentration ranges from < 0.05 to > 2.0 f/cc. All HV fiber levels were ≤ 0.10 f/cc with 82% being < 0.05 f/cc. Seventy-two per cent of all FAM fiber levels were > 0.1 f/cc with 88% of them exceeding 0.2 f/cc.

Comparison of Short-term vs. TWA Data

Individual short-term data collected every 10 minutes by the HV method showed little variation above the time-weighted average (TWA) data (Table 3 & Appendix 5). Concurrent short-term data collected with the FAM showed as much as two orders of magnitude variation above the TWA value (Table 4 & Appendix 5).

Correlation of Membrane Filter Techniques

While FAM-membrane filter data exhibit poor correlation, a strong correlation is seen among the membrane filter methods (HV and standard, Appendix 5) employed in the study. Results obtained with the two methods are nearly identical. In order to be compared with the time-weighted average (TWA) data from the standard and FAM filter (FAMF) techniques, short-term data collected with the HV and FAM methods had to be averaged over each full sample period.

The membrane filter of the FAM (FAMF) was analyzed to assess correlation among fiber levels observed by the FAM and collected by the FAM filter, as shown in Appendix 6.

Poor correlation exists between the value derived by integrating short-term FAM samples from a total sample period, and the corresponding FAMF sample collected over the same sample period.

However, strong correlation is seen between the FAMF results and the other membrane filter techniques utilized in this study. The results from four techniques are tabulated (Appendix 6). This is a further indication that the FAM was counting particulate that do not meet the NIOSH 7400 definition for fibers.

Integrated data obtained from two companies performing similar abatement activities and analyzed by the standard method correlates better with the study's membrane filter data than with its FAM data (Appendix 9).

Interference from Non-Fibrous Particulate

The paired-data presented in Appendix 3 reflects the fact that not one HV value equals or exceeds its FAM counterpart. Non-fibrous dust particulate have been found to affect the response of the FAM in controlled and actual workplace environments. Other researchers have reported higher values for the FAM than corresponding data collected with a reference membrane filter method.

Page (1980) used Arizona road dust (ARD), a standard test dust, to gravimetrically determine the maximum dust concentration that would not produce FAM counts. The FAM

produced counts as soon as the dust feeder was turned on. Gravimetrically, Page found that as particle size became smaller, so did the mass concentration necessary to produce a fiber-count. He also found that a smaller mass concentration consisting of smaller particles would produce a FAM fiber-count saturation. This was evident in the value for mass concentrations corresponding to the number concentration (30 particles/ml) sufficient to produce fiber-count saturations in the FAM; 5.1 mg/m3 for 5 um ARD, 0.33 mg/m^3 for 2 um ARD, and 0.04 mg/m^3 for 1 um ARD.

Page also found that ARD produced light-scattering signals that were similar to fiber-produced signals, and that this similarity might be attributed to the irregular shape of the particulate. The smaller aspect ratio of ARD vs. fibers was shown to synchronously follow the rotating electric field more easily than particles with larger aspect ratios, and therefore have a higher probability of detection by the FAM. This research on ARD lends credence to the speculation that higher FAM readings may be attributed in part to the presence of non-fibrous particulate.

Other non-fibrous dusts have been found to interfere with the FAM's response. Iles and Shenton-Taylor (1986) used talc and silicon carbide particles in a gravimetric study similar to that of Page. They found that while only 0.3% of silicon carbide particles fit the usual fiber size

definition (3:1 aspect ratio; > 5 um length, and < 0.3 um diameter) 15% of the particles passing through the FAM detection volume were counted. Similarly, for talc, the majority of particles passing through the FAM were counted. Very similar response to chrysotile and talc was observed at the 0.5 amplitude setting recommended by the manufacturer. This finding did not support the claim that the FAM's amplitude setting could be used to discriminate talc from chrysotile fibers.

Potential interference of non-fibrous particulate with FAM performance was also briefly assessed in the present study. Two HV slides corresponding to the highest reported FAM data (August 1, 4.56 & 4.54 f/cc), were observed to have greater total particulate deposition than two other HV slides corresponding to the lowest reported FAM data (July 16, 0.03 & 0.02 f/cc). This rough assessment coupled with evidence from previous studies, suggest that higher FAM counts may be attributable to non-fibrous dust particulate in the abatement environment.

Short-Term Variation and Respiratory Protection

The HV method has proven, empirically, to be a reliable method for measuring short-term asbestos fiber levels in an abatement environment. Actual maximum and minimum levels reported by the HV (Table 3) of 0.1 and 0.002 f/cc, respectively, illustrate the low fiber levels typical-

ly encountered by abatement workers. These levels do not demonstrate a regulatory need to wear respiratory protection since they did not exceed the action level. Still, all abatement workers observed in this study were outfitted with and used respiratory protection (3M 8710 disposable dust respirator).

Selection of proper respiratory protection is of great concern to the practicing Industrial Hygienist. Variation of fiber levels in the abatement environment is one factor that must be considered when selecting respiratory protective equipment. An estimated mortality risk from asbestos-related cancer of 3.4 per 1000 (Appendix 1) occurs at the 0.1 f/cc level in those persons exposed without a respirator. Even the 8710 disposable respirator, which is no longer approved by OSHA could lower the already low risk of mortality if worn. Currently, asbestos abatement workers use air-purifying respirators with HEPA filters. Appendix 2 shows the range of protection afforded by these respirators (10X - 100X). Use of this type of respirator would virtually eliminate the risk of mortality from asbestos-related cancer if worn and properly maintained.

Conclusions

A method has been developed for determining short-term environmental asbestos concentrations at or above the

existing OSHA PEL of 0.2 f/cc. The HV method modified the standard method by increasing flowrate and number of optical fields analyzed to compensate for lower total volume collected. The method was observed to correlate well with standard methods at fiber levels below the PEL.

The fibrous aerosol monitor showed poor correlation with the HV and standard membrane filter methods used in this study. The apparent cause of the poor correlations was due to the FAM's detection of other non-fibrous aerosols in the abatement environment.

Short-term asbestos concentrations measured in this study, and integrated averages observed in similar operations indicate that adequate respiratory protection can be provided to abatement workers without resort to the supplied-air respirators currently advocated by NIOSH and the EPA.

Implications for Future Studies

The HV method has given results that are comparable to those of the approved method. Future research on the HV method should focus on defining appropriate sampling intervals which will more accurately assess airborne asbestos fibers at any concentration.

A more rigorous field study of the FAM in those environments where airborne levels of chrysotile asbestos

predominate should be conducted to more accurately assess variation with the membrane filter methods.

APPENDIX 1

ESTIMATED ASBESTOS RELATED CANCER MORTALITY PER 100,000 BY NUMBER OF YERS EXPOSED AND EXPOSSURE LEVEL[1]

Asbestos fiber concentration (f/cc)	Cancer mortality / 100,000 exposed			
	Lung	Meso	G.I.[2]	Total
	1 year exposure			
0.1	7.2	6.9	0.7	14.8
0.2	14.4	13.8	1.4	29.6
0.5	36.1	34.6	3.6	74.3
2.0	144	138	14.4	296.4
4.0	288	275	28.8	591.8
5.0	360	344	36	740
10.0	715	684	71.5	1470.5
	20 years exposure			
0.1	139	73	13.9	225.9
0.2	278	146	27.8	451.8
0.5	692	362	69.2	1123.2
2.0	2713	1408	271.3	4392.3
4.0	5278	2706	527.8	8511.8
5.0	6509	3317	650.9	10476.9
10.0	12177	6024	1217.7	13996.7
	45 years exposure			
0.1	231	82	23.1	336.1
0.2	460	164	46	670
0.5	1143	407	114.3	1664.3
2.0	4416	1554	441.6	6411.6
4.0	8441	2924	844.1	12209.1
5.0	10318	3547	1031.8	14896.8
10.0	18515	6141	1851.5	26507.5

[1] Assumes exposure begins at age 25. Risks are calculated using U.S. male lung cancer background rates for 1977.

[2] Estimated as 10% of lung cancer risk rather than calculated using dose-response information.

APPENDIX 2

RESPIRATORY PROTECTION FOR ASBESTOR

Asbestos airborne concentration	Required Respirator
Not in excess of 2 f/cc (10 X PEL)	1. Half mask air-purifying respirator with HEPA.[1]
Not in excess of 10 f/cc (50 X PEL)	2. Full facepiece APR [2] with HEPA filters.
Not in excess of 20 f/cc (100 X PEL)	3. Any powered APR with HEPA filters, or any supplied-air respirator in continuous flow mode.
Not in excess of 200 f/cc (1000 X PEL)	4. Full facepiece supplied air respirator operated in pressure demand mode.
Greater than 200 f/cc (1000 X PEL) or	5. Full facepiece supplied air respirator operated unknown concentration in pressure demand mode equipped with auxiliary positive pressure SCBA.[3]

[1] Air-purifying respirator
[2] High-efficiency particulate air filter
[3] Self-contained breathing apparatus

APPENDIX 3

FIBER LEVEL RESULTS FROM HI-VOL AND FAM (F/CC) TAKEN CONCURRENTLY AT 10 MINUTE INTERVALS

July 12		July 14	
Hi-Vol	FAM	Hi-Vol	FAM
0.030	0.35	0.026	0.15
0.056	0.80	0.011	0.20
0.060	0.75	0.010	0.80
0.100	0.12	0.016	0.80
0.076	0.50	0.014	0.80
0.060	0.55	0.018	0.80
0.054	0.75	0.018	1.80
0.054	0.60	0.036	2.40
July 15		July 16	
0.011	0.78	0.022	0.09
0.008	0.45	0.004	0.15
0.008	0.28	0.005	0.09
0.010	0.05	0.004	0.03
0.002	0.08	0.002	0.05
0.020	0.14	0.018	0.02
0.004	0.34		
0.011	0.06		
0.030	0.18		

APPENDIX 3-(continued)

July 18		August 1	
0.030	0.65	0.024	1.50
0.025	0.64	0.017	4.56
0.030	0.42	0.015	2.16
0.030	0.55	0.024	2.61
0.027	1.06	0.034	3.10
		0.017	4.54
		0.074	1.55
		0.050	0.83
		0.066	2.53
		0.083	2.53

August 5		August 6	
0.000	3.05	0.020	0.65
0.000	2.22	0.020	0.50
0.001	1.61	0.028	0.40
		0.024	0.55
		0.018	2.00
		0.016	1.35
		0.027	0.60
		0.020	0.30
		0.024	0.75
		0.018	0.30

APPENDIX 4

CO-LOCATED SAMPLE RESULTS FROM (4) HIGH VOLUME PUMPS

HV1	HV2	HV3	HV4
0.015	0.012	0.012	0.015
0.015	0.007	0.012	0.010
0.015	0.010	0.017	0.010
0.005	0.012	0.010	0.010
0.002	0.002	0.002	0.005
0.002	0.002	0.005	0.000
0.002	0.002	0.002	0.002
0.002	0.002	0.005	0.002
0.005	0.005	0.007	0.005
0.002	0.005	0.007	0.005
0.090	0.090	0.080	0.060

All sample results reflect airborne amosite levels in f/cc

APPENDIX 5

COMPARISON OF CONCURRENT MEAN SAMPLING DATA FROM FAM, HV, & STANDARD (NIOSH 7400) METHOD

Date	FAM	Hi-Vol	Standard
July 12	0.55	0.061	0.065
July 14	0.95	0.019	0.010
July 15	0.24	0.012	0.030
July 16	0.07	0.009	0.005
July 18	0.66	0.028	0.010
August 1	2.59	0.040	0.015
August 5	2.29	<0.001	0.050
August 6	0.74	0.022	0.050

All values reflect mean airborne asbestos levels in f/cc

APPENDIX 6

COMPARISON OF MEAN SAMPLING DATA COLLECTED WITH THE FAM FILTER, FAM, HV & STANDARD METHOD

July 12, 1986

FAMF	FAM	Hi-Vol	Standard
0.05	0.55	0.06	0.06

August 6, 1986

FAMF	FAM	Hi-Vol	Standard
0.03	0.74	0.02	0.05

All values reflect mean data in f/cc

APPENDIX 7

DISTRIBUTION OF FIBER LEVEL RESULTS FOR HI-VOL VS. FAM

NUMBER OF SAMPLES BY FIBER CONCENTRATION (F/CC)

<0.05	0.05-0.10	0.11-0.20	0.21-0.30	0.31-0.40	0.41-0.50	0.51-1.0	1.1-2.0	>2.0	Total
1	7	-	-	-	-	-	-	-	8
-	-	1	-	1	1	5	-	-	8
9	-	-	-	-	-	-	-	-	9
1	-	2	-	-	-	5	1	1	10
9	-	-	-	-	-	-	-	-	9
-	4	2	1	1	1	1	-	-	10
6	-	-	-	-	-	-	-	-	6
2	3	1	-	-	-	1	-	-	7
5	-	-	-	-	-	-	-	-	5
-	-	-	-	-	1	3	1	-	5
12	5	-	-	-	-	-	-	-	17
-	2	-	-	-	-	-	3	8	13
3	-	-	-	-	-	-	-	-	3
-	-	-	-	-	-	-	2	2	4
10	-	-	-	-	-	-	-	-	10
3	4	-	-	-	-	1	2	-	10
61	25	6	1	2	3	16	9	11	134
55	12	0	0	0	0	0	0	0	67[1]
6	13	6	1	2	3	16	9	11	67[2]

All values are expressed chronologically from July 12 thru August 6, with Hi-Vol results in top rows and FAM results in the bottom rows. Total number of samples recorded by fiber concentration is broken down in appropriate columns.

[1] Number of Hi-Vol samples at their reported fiber level.
[2] Number of FAM samples at their reported fiber level.

APPENDIX 8

AVERAGE VARIATION OF FAM RESULTS ABOVE HV RESULTS

July 12	July 14	July 15	July 16
12	6	71	4
14	18	56	37.5
13	80	35	18
1.2	50	5	7.5
7	57	40	25
9	44	7	1.1
14	100	85	-
11	67	5.5	-
-	-	6	-
10X	53X	35X	16X

July 18	August 1	August 5	August 6
22	62.5	*	32.5
26	268	*	25
14	144	*	14
18	109		23
39	91		111
-	267		84
-	21		22
-	17		15
-	38		31
-	30		17
24X	105X	* = all > 1000X	37X

APPENDIX 9

REPRESENTATIVE FIBER LEVEL RESULTS OF ASBESTOS REMOVAL ACTIVITIES FROM 2 COMPANIES

	Jan-Dec'86	Mar-Aug'86
	Company A	Company B
	0.070	0.080
	0.040	0.050
	0.040	0.010
	0.710	0.020
	0.160	0.010
	0.070	0.340
	0.020	0.030
	0.020	0.089
	0.030	0.140
	0.050	0.010
	0.080	0.150
	0.080	0.220
Mean	0.114	0.096
Median	0.080	0.065
SD	0.191	0.102
	Jan-Apr'87	Feb-Mar'87
	0.370	0.078
	0.350	0.020
	0.550	0.070
	0.230	0.120
Mean	0.375	0.072
Median	0.360	0.074
SD	0.132	0.041

All values reflect airborne asbestos levels in f/cc

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