PLAINTIFF'S EXHIBIT
JM-1383

(A)

Government Inspection & Sampling

Station	Date	Location	Rec	T.L.V
#1	11/8/71	A-Bldg Stocker-No. Card Room	10.5 F/cc	60 F
#2	11/8/71	A-Bldg Front Tender-At Card Rm. #30-31-40 & #41	4.5 F/cc	60 F
#3	11/8/71	A Bldg Breaker Operator-No. Card Rm. #52 #62 #55 & #63	1.2 F/cc	60 F
#4	11/8/71	A Bldg Waste & Service Operator North & South Card Room	10.5 F/cc	60 F
#5	11/8/71	A Bldg. Wick Former-Twisting	5.4 F/cc	60
#6	11/8/71	A Bldg #6 Tape Loom-Weave Room	4.3 F/cc	60 F
#7	11/8/71	A Bldg #36 Listing Loom-Weave Room	12.5 F/cc	60 F
#8	11/8/71	A Bldg Listing Winder-Weave Room	3.2 F/cc	60
#9	11/8/71	A Bldg #31 Heavy Duty Brake Lamming Loom-Weave Room	5.1 F/cc	60 F
#10	11/8/71	C Bldg. Sewing Mach Operator	0.4 F/cc	60 F
#11	11/8/71	B-Bldg Operator for #1-2-3-4 5-6-7 & 8 Braiders	0.5 F/cc	60
#12	11/8/71	B Bldg. Operator for #9-12-13-16-17-18-19-20 & #21 Braiders	0.5 F/cc	60 F
#13	11/8/71	B Bldg Operator for Large Pipe Loom	0.4 F/cc	60
#14	11/8/71	B Bldg Winding Room Mezz (Yarn Issuing For Danbury	0.2 F/cc	60 F
#15	11/8/71	B Bldg. Danbury Operator-Plexar	0.2 F/cc	60
#16	11/8/71	B Bldg #27 Mini Shuttle Operator	1.3 F/cc	60 F
#17	11/8/71	B Bldg Finishing & Packing Room Floor	0.1 F/cc	60 F

to error.

One additional important possibility related to increased awareness of mesothelioma should be kept in mind when considering neighborhood exposures. This factor has been evidenced in several studies. For example, the following information has been taken from Table II in the McDonald and McDonald study (1973):

Distribution of reported cases of mesothelioma by provinces (1960-1970)

	Ontario	Quebec	other Provinces
No of reported cases	69	102	65
Reviewed by pathology Panel	57	80	47
accepted as mesothelioma	71%	42%	57%

The study by McDonald and McDonald (1973) was based on a formal national survey of all pathologists in Canada. The differences in percentages accepted as mesotheliomas (42%, 71%, 57%) were statistically significant ($P < .01$). The lower percentage accepted in Quebec (42%) supports the falsely higher reporting with increased awareness in that geographic area.

It is unreasonable that OSHA has failed to cite the only report of a formal national survey of all pathologists, conducted in Canada and published by McDonald and McDonald (1973). On community exposure, quoting from the summary of that paper: "No case other than those occupationally or domestically exposed had lived within 20 miles of asbestos mines or mills." Neighborhood exposures (asbestos factories and shipyards) were reported for 9 of 214 mesotheliomas regarded as "definite" in the article by Greenburg and Lloyd Davies (1974). However, possible past exposures, local awareness and lack of reliable baseline rates render a formal evaluation impossible.

The discussion by Webster (1973) points out the difficulties in interpreting the South Africa mesothelioma experience. Another complete pathological survey was reported for Scotland for the period from 1950 to 1967 (88 cases) period. The study was designed and carried out as a retrospective matched case-control (two sets of controls were selected). Of the 51 cases who had residential exposure, all but one also had some evidence of occupational exposure to asbestos. (There were 5 females with

residential exposure without occupational exposure.)

Thus, considering the number of studies that have been completed, there is little evidence that past residential exposures have appreciably increased the risk. Some cases would be expected near asbestos operations due to the small but accepted natural rate, and the increased awareness of the disease near asbestos operations. Mesotheliomas deaths in occupationally exposed cohorts have been well documented. This documentation was available before 1972. Long latent periods for the disease are evidenced -- meaning that current cases were exposed long ago at a time when the levels were undoubtedly higher. In addition, high wartime exposures due to factors such as shipbuilding and long work days undoubtedly contribute to recent cases

It should be reiterated that none of the mesotheliomas studies thus far referred to have been accompanied by numerical estimates of actual exposures, and hence are not of practical help in arriving at a numerical standard.

The only recent publication giving some 'new insight' cited by OSHA was a paper presented, but not yet in print, by Newhouse and Berry. While it is not believed that the projections of mesotheliomas deaths in a workforce exposed before 1960, using the Weibull distribution, help in any way to establish a TLV, one basic assumption of that paper is: "Both the degree and length of exposure are of importance." Evidence was presented to support a dose-response relationship. Even though some mesotheliomas have been reported with short exposures, the intensities and past levels 20 to 40 years ago can only be conjectured. The authors note: "It should be stressed that the population we are considering were all first exposed to asbestos prior to 1964, and most of them before 1951. Therefore, the conditions responsible are not those which should be achieved today. The evidence of a dose response relationship shows that improved factory environments at the present and in the future should markedly reduce risks...." Thus, the new evidence cited by OSHA comes from a manuscript supporting a dose-response for mesotheliomas and reporting "markedly reduced risks" at the present time. In addition, this article is refreshing in its criticism of its own data -- statements apparently ignored by OSHA.

In the Proposal, OSHA states: "mesotheliomas deaths have occurred among the specific group of 298 workers whose experience prior to 1516 had led to the development of the current standard" (this refers to the Enos cohort reported by the BOHS [1968]), suggesting that this would invalidate the standard. As discussed in detail earlier, the cohort of 298 includes a spectrum of past exposures (some extremely high lifetime exposures even as late as 1968), many far in excess of the current OSHA standard. Based on the knowledge from other cohorts, mesotheliomas deaths would be expected in the Enos cohort.

When efforts to relate dose to the risk of excess mesotheliomas are

considered, these are more likely to be successful in an occupational setting since the chance for developing unusual exposure data is virtually nonexistent in "household and community" cases and in these environments there is a large chance of indeterminable spontaneous occurrence of mesotheliomas.

The "New" Evidence Gastrointestinal Cancer

The available information on gastrointestinal cancer in the OSHA references are summarized in the following table.

Summary Table of Information on Gastrointestinal* Cancer Deaths in Cohorts in Cited Studies:

Reference No.	No in cohort	Mortality Data from 1972	Mortality Data other	O.	E
1	1495	1963		18	8.07
2,5	632	1964(68)		37	11.2
16			1972**	4**	1.5**
4	21,755	1967		83	78.6
13,30	870	-		16***	16.0***
26	165	1971		15	<5.16
16	933		1972	26	12.81
16	17800		1972	90	23.42
17	1464		1972	59	45.9
19	609		1971(?)	13	9.8

*Generally ICD's 150-159 or subsets, varying with the study.

**Updated information for preceeding cohort -- obtained by subtradition.

***Obtained by personal communication.

Reviewing the data available on gastrointestinal cancer presents a mixed picture. There is a two- to three-fold increase in risk shown in some studies, while other studies have shown little or no increase in observed over expected. The consistency of an excess in observed over expected, as is shown in lung cancer, is not

present with respect to gastrointestinal. In any event, there does not appear to be any "new" evidence since 1972 which clarifies the pre 1972 picture. Increased regional risks reflected in increased regional rates may account for some of the increases seen in some of the studies. All of the cited data taken together suggest that any increase in risk is somewhat less than the two to three times mentioned in the proposal and also indicates that the excess risk occurs only in the most heavily exposed category (McDonald et al). No evidence to suggest that a 2 f/cc standard is unsafe for gastrointestinal cancer has been presented by OSHA.

The "new" Evidence for Other Neoplasms

Following a brief discussion of possible increased risk of cancer of the larynx, oropharynx and esophagus, the OSHA proposal states: "However, data concerning these neoplasms are less extensive than for lung cancer, mesotheliomas and gastrointestinal cancer and further experiences are awaited. In any case, they are not very common tumors in general and any increase does not weigh heavily on the overall cancer risk of asbestos workers." It may be conjectured that it is unlikely that any possible increased risk of these tumors will ever be fully documented. If the large cohort-mortality studies on the heavily exposed workers of the past have been unable to document an increased risk, these other neoplasms certainly cannot support the contention that the far lower level of a 2 f/cc standard will have any adverse impact on mortality from these tumors.

The "New" Evidence on Pleural Plaques

The study by Fletcher (1972), reported on a 18 year follow-up of 498 men (selected from 15,524 available for study) with pleural plaques and 484 controls (without plaques selected from the same workforce). The controls were matched for age (within 5 years), but not for onset of exposure or smoking habits. A matched-pair analysis was not possible, as the authors recognized that the controls were definitely younger than the cases. The increase in risk, within a 5 year age group, of some diseases, notably lung cancer, is well known. The data were analyzed as two retrospective-prospective mortality studies (cases and controls separately), using local rates for comparison. Because of factors such as the small percentage of the workforce followed, lack of exposure times or times from onset of exposure, or actual numerical estimates of exposure to asbestos fiber it is unclear how the results of that study support the contention that a 2 f/cc standard will have any adverse impact on morbidity or mortality.

The draft of the paper by Edge (1975) describes another follow-up period for a cohort with pleural plaques and a matched control study for part of the cohort. As in the discussion of the study by Fletcher (1972), no information is available to support the

contention that the 2 f/cc standard will have any adverse impact on morbidity or mortality.

The "new" Evidence on Underground Metal Miners

We have elected to comment on this paper in great detail because of the obvious importance attached to it by OSHA and because of the major implications inherent in uncritically accepting the substance of this study and its conclusions.

As previously indicated, the OSHA proposal is premised on "new information" about the toxic effects of asbestos. The study by Gillam et.al (1979), OSHA reference No. 41, appears to be the one reference most relied upon by OSHA.

Before discussing this NIOSH study in depth, a discussion of the problems inherent in small samples, mentioned several times previously, is appropriate. To clarify some of the problems associated with analyzing the results of small samples, consider the following:

Testing a 1-tailed alternation of interest, the observed level of significance (so-called P-value, or just P) represents the chance of observing a value as large or larger if in fact the workforce happened to be a random sample (representative of or similar in health characteristics) from the general population. For example, for an expected (E) of 2, the chance of observing exactly 5 deaths from a sample of the general population is approximated by the Poisson distribution as .036. The chance of observing 5 or more (5, 6, 7, 8, etc.) is approximated to be 0.051 and is the P-value for a one-sided alternative of interest (that is, if the study population differs from the standard population, we are only interested if there are more deaths). While opinion differs in the "seriousness" of a P of .05 or .01, etc., it is true in any event that, the smaller P is, the more it casts doubt on the possibility that the "study" population is similar to the "standard" population. (In other words, if the two populations are truly the same, the differences observed were "unlikely.")

Observed	Expected	SMR	P
5	2	2.5	.053
5	2.2	2.3	.072
5	2.4	2.1	.096
5	2.6	1.9	.123
10	10.0	1.0	.014
10	11.0	1.6	.032
10	13.0	1.4	.110

For many possible outcomes, a relatively small change in the "expected" can result in a relatively large change in the 'P. For example, an observed of 5 with expected of 2 gives a P of 0.053. Increasing the expected 10 percent to 2.6, increases P 132 percent to 0.123. The other example in the above table is even more "dramatic" -- an E=10.0 and an observed (O) of 10 results in P=.014. Increasing E 30 percent to 13, increases P 686 percent to 0.110. It should be noted that in the two examples cited above, the observed was still larger than the expected after a 10 percent increase. In the absolute sense, the modest increase in E did not "explain" the fact that the observed was in 'excess' of the expected. However, the data are generally not investigated by simply asking: Is O larger than E? The observed will be larger than the expected approximately 50 percent of the time if the null hypothesis is true (i.e. the two populations are the same). The data are analyzed statistically to see if the findings are "consistent" with the null hypothesis ("larger P-values). As can be seen from these examples, modest increases in E can (not necessarily will) result in a dramatic increase in P.

The type of analysis discussed above is based on a number of assumptions. For example, it is known that the risk of death depends upon age, sex and many other factors. Some factors are adjusted in the analysis, others may not be. Larger sample sizes alleviate concern for some unaccounted for factors. To illustrate the possible problems, suppose a standard population had 50 percent smokers and 50 percent nonsmokers, while a study population had 65 percent smokers and 35 percent nonsmokers. Suppose further that nonsmokers and smokers had 'risks' of 1 and 9 respectively, in both populations. On the average then, the standard population risk is (.5) 1+(.5)9=5.0, while the study population is (.4)1+(.6)9=5.8. Although the populations differ only in proportion of smokers, the average risk of the study population is 16 percent higher, and therefore the expected rate would be 16 percent higher if smoking is correctly accounted for.

Other possible factors that may influence the accuracy of the analysis are age distributions (although age is taken into account, differences in age distributions within quinquennia may account for errors of 5 percent or 10 percent), appropriateness of the standard population and differences in regional and/or level of urbanization commonly considered. Of course, not all factors render the estimate of E too low. It is generally accepted that the general health of a cohort, defined from onset of employment, will be better than the general population, at least for a few years. Uncertainty of the standard population estimate may also be a factor. (For example, two National Center for Health Statistics publications, Series 2, Nos. 20 and 30, consider aspects of this problem.) Considerations of factors such as dynamic population rates and natural variation may be magnified when regional rates are used.

The main point to be made from this discussion is that variation and bias unaccounted for in the Poisson analysis should be an important consideration in analyzing small samples.

The above discussion of the problems inherent in studying a small cohort is essential to appreciate the fatal flaws in the Gillan et al (NIOSH) paper. Keeping the above discussion in mind, the critique of the study of Gillam et al (1979) prepared by Paul Kotin, M.D. and Gerald R. Chase, Ph.D., of Johns-Manville is appended. Their critique concludes that the claim of excessive rates in the asbestos-related malignant and nonmalignant disease categories is clearly based on poor and incomplete data analyses. Even if the claim were based on valid analyses, data to incriminate asbestos rather than one or a combination of other coexisting materials in the causation of cancer are entirely lacking. To ascribe the excess of nonmalignant respiratory disease to asbestos and ignore the known exposure to high levels of free crystalline silica in the past, confirmed by the frequent diagnosis of silicosis on the death certificates, borders on irresponsibility. To ignore the potential for carcinogenic and cocarcinogenic effects resulting from the mixed exposure to silica dust, arsenic fumes and particles, blasting powder fumes and possibly radon daughters and to arbitrarily ascribe all of their excess of cancer to asbestos particles is manifestly irresponsible and has no justification in the methodology of science.

Summary

The claim by OSHA that "since the promulgation of the U.S. permanent asbestos standard, considerable new information has been forthcoming on the toxic effects of asbestos" is clearly based on an incomplete and unscientific review and assessment of available data. In its 42 references OSHA has not presented valid evidence to support a need for further reduction of the airborne asbestos standard from 2.0 f/cc longer than 5 microns to 0.5 f/cc longer than 5 microns.

Replication of the findings and discussions of risk already known in 1972 does not constitute "considerable new information." As was true in 1972, it is known that heavily exposed cohorts now dating back 30 to 70 years ago have experienced excess morbidity and mortality, but that is not "considerable new information."

The "new evidence" cited by OSHA from British studies is a prime example of uninformed selective reporting. While ignoring a BOHS subcommittee report (1973) specifically recommending no change, OSHA has misrepresented the available information on the TBA workforce. The available information has been detailed in this report. When the facts are reviewed, it is obvious that there is no evidence available on the TBA workforce that supports a need for further reduction in the asbestos standard now scheduled for July 1976. Indeed, the documented high exposure levels extending into the recent past, plus the fact that such exposures were derived from static, not personal, sampling, when coupled with the recently published health studies, demonstrate that the lessened adverse health effects noted were related to lower but still substantial exposures and provides confidence for maintaining the 2.0 f/cc airborne asbestos standard.

Selective use of preliminary, unpublished and nonpeer-reviewed manuscripts and studies have been used by OSHA as important segments of the "new information." The most notable example is the morbidity and mortality study by NIOSH (Gillam et al [1975]). Since the OSHA citation, the drafts of the study have undergone substantive revisions, including the removal of the entire morbidity portion. The conclusions of that paper are of such questionable scientific quality that it cannot be considered "new information" of the scientific competency and accuracy required for use in decision making.

In reference after reference, detailed review has shown that OSHA has cited information that was already known in 1972, none of which supports a reduction in the standard.

Omission of references, such as a national mesothelioma survey in Canada, is inexcusable. Selective reference to parts of other reports is just as inexcusable. The very report that was cited (Newhouse and Berry [1975]) as providing evidence for a continued increase in mesotheliomas among known heavily asbestos exposed workers was based on a dose-response relationship for mesotheliomas. Further, the limitations of that study, so nicely given by the authors, have been ignored by OSHA.

We have the right and the obligation to insist that all regulatory agencies adhere to the highest requirements of scientific competency and accuracy in reviewing available information for any standard. This was not done for the October 9, 1975 Notice of Proposed Rulemaking on Occupational Exposure to Asbestos.