C.L. SHEFFLER

PLAINTIFF'S EXHIBIT
JM 954

CONFIDENTIAL FINAL REPORT

THE BIOTOXICITY OF PERLITE

to

The Perlite Institute
10 East 40th Street, New York 16, New York

by

The Saranac Laboratory
7 Church Street, Saranac Lake, New York

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Submitted by

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Director

CONTENTS

I. INTRODUCTION

This final report from The Saranac Laboratory to the Perlite Institute covers all observations made during the recently completed experiment which was designed as a study of the effect of inhalation of Perlite dust upon the course of tuberculosis in the experimental animal.

By means of a previous experiment it had been shown that the inhalation of Perlite dust caused no significant pulmonary fibrosis in normal animals, but that the presence of the Perlite in the lung tissue stimulated a tuberculous infection. In view of the rarity of such a combination of findings, a further study of the relationship between Perlite inhalation and tuberculosis was indicated. The experiments which were designed were intended primarily to reveal whether the tuberculogenic effect of the dust deposited in the lung tissues would continue to be exerted during the months after cessation of exposure. This point was considered to be of practical importance in industry, because, although any dust-exposed laborer who might develop tuberculosis would be removed from exposure for treatment, the outlook for successful treatment would depend in large measure upon whether or not the stimulatory effect of dust on a pulmonary tuberculous complication would persist. In human silicosis complicated by tuberculosis, for instance, the prognosis is usually grave. This fact has been demonstrated also in animals, the stimulatory effect of quartz dust on tuberculosis in experimental animals persisting long after dust exposure has stopped.

The present study has provided observations which permitted submission to the Perlite Institute on December 10, 1954, of the following summarized statement of the effects of Perlite on animals:

A. Perlite does not provoke any detectable primary lung disease.

B. The continuous inhalation of Perlite dust stimulates the development of tuberculosis in animals, which therefore develop progressive lesions.

C. Interruption of Perlite dust exposure after six months and after tuberculosis has become established does not modify materially the rate of progression of the tuberculosis during the succeeding 8 months.

D. About 8 to 12 months after the withdrawal of the Perlite dust exposure the tuberculous process begins to wane in the sense that a previously progressive disease commences to show signs of healing through fibrosis. Ultimately the disease is arrested.

E. Healing of a tuberculous infective process has never before in the experience of The Saranac Laboratory occurred under comparable experimental circumstances in which animals were exposed to quartz dust, and survival for as long as 12 months is exceedingly rare.

F. The healing process is incomplete in the sense that while the cellular components of the tuberculous lesions disappear, marked fibrous scar formation takes its place. The healing sequence of tuberculosis in animals is normally complete without the formation of such extensive cicatrization. In this respect, therefore, the lung fibrosis must be interpreted as a significant pathological residuum.

The evidence upon which this summary was based will be presented.

II. MATERIAL

The product used for dusting in the present study was the same as that used in the original experiment concerning which a final report was issued on March 12, 1954. This product is identified by The Saranac Laboratory No. MD 201, and its physical and chemical composition as determined by us are presented in Table 1.

III. METHODS

A. Dust Room: The procedure with respect to the dusting of the animals was identical to that in the earlier study. The experiment was conducted in a specially-constructed cubical dust room 8 feet in each dimension. The dust was generated mechanically by a paddle wheel agitator rotating in a dust hopper. The dusting apparatus was kept in operation for 8 hours a day, 5-1/2 days a week. The animals lived continuously in the dust room but the room was ventilated by means of fresh outside air during periods when the dusting apparatus was not functioning. The dust concentration was controlled at regular intervals by counting the particles collected by means of the midget impinger.

B. Infection of Animals: As in the previous study, the infecting tubercle bacilli were of the attenuated human R1 strain which, as has been repeatedly emphasized, is capable of producing in guinea pigs a tuberculous infection which develops and progresses for about 2 months and subsequently, over a period of many months, regresses and heals. The infection was accomplished by preparing a dilute suspension of the organisms in saline solution and the animals were caused to inhale for a fixed period of time an atomized mist of this suspension. Control animals were infected with the same dose of organisms at the same time as were the animals to be exposed to dust.

C. Experimental Animal Groups: As the purpose of the present study was to ascertain whether or not inhaled Perlite dust, once deposited in the lung, could stimulate tuberculosis during a subsequent period of non-exposure to the dust, the following protocol of dust exposure and sacrificing intervals of animal groups was designed:

Group A. Infection by inhalation at the beginning of a six-month exposure to Perlite dust. Sacrificings during and after exposure in order to follow the course of the tuberculosis. Sacrificing periods at 2, 4 and 6 months of exposure, and at 3, 6, 9 and 12 months after the end of exposure making a total observation period of 18 months following infection.

Group B. Infection at the end of a six-month exposure to Perlite dust. Sacrificings scheduled after the infection at 2, 4, 6, 8 and 12 months.. The total duration of the study in this group was also 18 months.

Group C. Exposure to Perlite dust for six months with sacrificings during exposure at 2, 4 and 6 months, and after exposure at 3, 6, 9 and 12 months. These are non-infected control animals for Groups A and B.

Group D. Infection without dust exposure. These are infected control animals for Group A and were sacrificed at intervals corresponding to those in Group A.

Group E. Infection without dust exposure. These are infected control animals for Group B, and were sacrificed at intervals corresponding to those in Group B. Two groups of infected control animals were required because the infecting procedure was done separately, Groups A and D being infected at the start of the study while Groups B and E were infected six months later. This was to ensure that results did not vary much because of differences in the culture batches.

D. Chemical Analysis of Animal Lungs for Silica: In the Progress Report dated March 12, 1953, it was pointed out (Page 6, Paragraph B) that in certain respects the progressive tuberculosis seen in infected animals exposed to Perlite dust was different from that in animals exposed to quartz dust. These peculiarities suggested that there might be excessive accumulation of Perlite in the infected lungs as compared with the normal lungs. Accordingly, the lungs of all animals involved in the present study have been analyzed for their silica content. The technique used permits reporting of the silica in terms of total weight in the lungs, rather than in terms of its concentration in the lung ash. It is of interest also to record the ash weight of the total lung.

IV. RESULTS

A. Dust Concentration: Samples of dust were collected for determination of atmospheric concentration at approximately weekly intervals throughout the six-month period of exposure of the animal to the Perlite dust. Usually the collecting apparatus was the Smith-

Greenburg Midget Impinger and the dust concentration was ascertained by counting the particles using a light-field technique. The average of all counts was 552.2 million particles per cubic foot. At monthly intervals the concentration of dust was determined on a weight basis, using the electrostatic precipitator. The average of all such determinations was 3.03 mgms. per cubic foot of air. The range of variation in the levels of dustiness is summarized in Table 2 and in Figures 1 to 3. It is to be particularly noted that the absolute dispersion in particle counts was, except for occasional unexpected peaks, kept within as satisfactory control as is generally possible under experimental conditions, the coefficient of variation not exceeding 39.86 per cent average. The size frequency distribution of the airborne dust is given in Figure 4. About 50 per cent of the particles were seen to be less than 2 microns and about 80 per cent were less than 5 microns. These were the respirable constituents of the aerosol of Perlite.

B. Course of Infection: The course of the infection in the experimental animals has been presented in progress reports dated October 19, 1953, and April 5, 1954. In this final report all observations are presented, whether or not they have appeared in earlier reports. The detailed observations in each individual animal are presented in Tables 3 and 4 and are graphically portrayed in Figures 12 and 13.

The points to be particularly noted are: (a) the progressive character of the disease became retarded from about the 8 months after cessation of dust exposure; (b) after 12 months had elapsed all animals showed healing lesions without any suggestion of further progression but with considerable scar formation. Thus the numbers in the tables, indicating the relative extent of disease, reveal that even though healing is well advanced it is manifested by replacement of the inflammatory process by scar tissue rather than by restoration of the tissue to normal.

C. Ash and Silica Content of Lungs: In the original experiment showing progression of tuberculosis in animals exposed to Perlite dust, the impression was gained that there might be excessive accumulation of dust in the lungs of infected animals. This impression was derived from the fact that less dust was transferred from the lung to the hilar lymph nodes of infected animals than was transferred in uninfected animals. Since this phenomenon might account for the progression of the disease in the infected animals an attempt was made to demonstrate such an excessive accumulation of dust by chemical analysis of the lung tissues of the animals in this experiment for silica. At the same time the weight of the ash of the animal lungs was determined.

The results of the chemical analyses of the pulmonary tissue are presented in Figures 9, 10 and 11. In each of these figures, the sloping straight lines indicate trends and the vertical lines connecting dots above and below the trend lines present the actual observed values. As the figures reveal, the animals were exposed to Perlite dust for six months and were followed for 12 months thereafter.

Attention is called particularly to the fact that the presence of infection does not alter the rate of accumulation or elimination of silica as compared with uninfected controls, Figures 9 and 11. Thus the lesser quantity of dust seen in the lymph nodes of infected animals merely indicates that dust is mainly eliminated from the lungs via the respiratory passages, rather than through the lymphatic system.

A further point of interest recorded in Figures 10 and 11 is the line representing the amount of lung ash which remains after subtraction of the silica. Note that this quantity increased after cessation of exposure in infected animals and decreased in non-infected animals.

D. Rates of Accumulation and Excretion of Silica in Animals Exposed to Perlite

(Supplementing Final Report of 1953)

At the time of issuing the final report concerning the action of perlite dust upon the lungs of normal, non-infected animals it had not been possible to complete all chemical analyses of the lung tissues for silica content. Since these data are now available they are presented herewith in Figures 5, 6, 7 and 8. In each figure there are two pairs of lines, one pair revealing silica in terms of weight and the other pair representing a percentage of the lung ash. The lungs from two animals were analyzed at each time interval and the upper and lower lines of each pair represent maximum and minimum values.

In Figure 7 it is shown that the percentage of silica rapidly reached about 60 at 8 months of exposure and during the succeeding 16 months rose at a retarded rate to a maximum of only 66 per cent. In contrast the weight of silica increased at a uniform rate up to 24 months except for one pair of high values at 18 months.

In Figure 5 the analytical results obtained in animals killed at various intervals after the 24 month exposure period are presented. There is considerable variation in the data obtained but in general there appears to be little tendency for rapid removal of the deposited dust.

Figure 6 reveals the findings in a brief experiment involving exposure for a period of 9 days followed by 9 days in normal air. Again very little if any of the accumulated dust was removed during 9 of the days in normal air.

In Figure 8 data are assembled showing the rates of silica accumulation in the lungs of rats exposed to perlite dust over an 18 month period. The results with rats are more variable than for guinea pigs concurrently exposed (Fig. 7) but general trends are similar.

V. DISCUSSION

The most important and practical point demonstrated by this study is that, although pulmonary deposition of Perlite dust stimulates pulmonary tuberculosis, the stimulatory action persists for only a relatively short period of about 8 months after interruption of dust exposure. When this fact is contrasted to the indefinite persistence of the stimulatory effect of quartz dust in the lung under comparable circumstances it becomes clear that Perlite dust, though dangerous, is distinctly less potent than quartz in this respect.

This less pronounced tuberculogenic propensity of Perlite should not be underestimated. While the experimental evidence suggests that tuberculosis in the presence of Perlite dust does not have the high mortality rate of tuberculosis in the presence of quartz, there is yet a rather prolonged illness and in the animals the extensive residual fibrosis may well be associated with some loss of lung function.

It is granted that the course of events in R_1 infected guinea pigs does not necessarily indicate precisely what is to occur in industrial workers exposed to Perlite dust who may develop tuberculosis. Thus it is possible that in Man there may be no demonstrable effect of the dust upon infection. On the other hand, the effect may be even more pronounced and prolonged in Man than it is in the guinea pig. However, the good correlation found in previous studies between the effect of various dust upon R_1 tuberculosis in guinea pigs and on tuberculosis in Man indicates that the experimental findings with inhaled Perlite dust should be considered as a warning that some degree of hazard exists but that the dust is by no means as noxious as quartz. An attempt has been made to present graphically in Figure 14 the correlation we believe may exist between the guinea pig results and the possible effects to be expected in Man.

With respect to the application of the experimental results to the estimation of allowable dust concentrations in industry, the observations in these experiments with tuberculous guinea pigs seem to require that atmospheric suspensions of Perlite products should be regarded as somewhat more serious than more nuisance dusts. Since the evidence is that at least one of the Perlite dusts can stimulate tuberculous infection, it seems wise to suggest a somewhat lower limit of concentration than the 50 million particles per cubic foot mentioned above. Since the influence of Perlite dust upon tuberculous infection is not as pronounced as the influence of quartz dust, it would appear that concentrations higher than those allowed for quartz dust would be permissible. In the case of atmospheric suspensions of relatively pure quartz dust, the generally accepted maximum allowable concentration is 5 million particles per cubic foot of air. On the basis of the considerations just mentioned, it would seem that a maximum allowable concentration for Perlite dust may be greater than this though appreciably lower than the standard permitted for mere nuisance dusts (50 million particles per cubic foot of air).

The attainment of such a maximum level of dust concentration in many plants may indeed require strenuous precautions, but such efforts will surely not be as intense or costly as they would be were it necessary to recommend a maximum concentration of the order of 5 million particles per cubic foot.

Great attention will also have to be given to careful pre-employment selection of personnel who do not have tuberculosis or scars of past tuberculosis. All persons exposed to Perlite should be subjected to annual radiographic examination and any showing suggestions of tuberculosis forthwith eliminated from further exposure.

No persons already suffering from tuberculosis or whose x-rays show signs of past parenchymal tuberculosis should be employed under circumstances where they may be caused to inhale Perlite dust. Personnel exposed to Perlite dust should undergo periodic x-ray examinations for tuberculosis. When tuberculosis is suspected in any worker exposed to Perlite dust, such a person should be immediately removed from such an environment and carefully studied.

The reason for the stimulatory effect of Perlite dust upon tuberculosis remains obscure. The identification of the mechanism of such action would constitute an important step towards its ultimate prevention. It was suggested in the progress report of March 12, 1953, that the mechanism might be related to excessive pulmonary accumulation of Perlite dust. The present study, Figures 9, 10 and 11, rules out this possibility. Other possibilities exist and are being examined.

The nature of the healing process in these animals is also of interest, in that it is accomplished chiefly by scarring of the lesions rather than by resolution or disappearance. The healing of tuberculosis in experimental animals is usually by resolution, and involves much less scarring than is customary in healing human tuberculosis. The course of these phases of the disease is summarized in Figures 12 to 14.

The rather extensive scarring noted in this study may provide a tool with which the effects of the residual fibrosis could be investigated. However, such investigation must await the availability of funds for basic research.

A final point of academic interest is apparent in the results of chemical analysis of the lungs. This point pertains to the rising trend of the line representing the pure lung ash, minus silica, in the infected animals after interruption of dust exposure. In uninfected animals this trend is downwards. The difference between the weight of pure ash in each group of infected animals and the weight in uninfected animals is statistically significant and may parallel the degree of tissue reaction present.

VI. FURTHER RESEARCH

In view of the tremendous industrial importance of Perlite and the likelihood of its future use on an extensive scale, further research on its unique tuberculogenic propensity is to be highly recommended, particularly with reference to the questions:

A. Whether animals nearer in the evolutionary scale to Man are likely to react in the same manner as do guinea pigs. We recommend a study in which Monkeys are to be used.

B. Whether the progressive tuberculosis observed under experimental conditions is amenable to satisfactory treatment by means of modern bio- and chemotherapy.

C. Whether the disabling effects of the ultimate fibrosis may be prevented or minimized through the administration of substances, such as cortisone, which are known to be effective against a variety of fibrotic phenomena in Man.

D. Whether the admixture of other commercial substances with Perlite is likely to enhance or reduce its harmful effects.

E. Whether Perlite in a state of finer subdivision would have effects materially different than those of the product we studied.

Throughout this report we have so far assumed that what happens to the guinea pig is likely also to happen to Man. This is not necessarily the case. In the case of quartz dust the lesions seen in guinea pigs are identical with those seen in human victims of silicosis. However, in the experience of The Saranac Laboratory with an extensive series of substances other than quartz a marked species variability has often been observed. Though we have thus far optimistically extrapolated our results directly from guinea pigs to Man (by analogy with quartz) it may yet prove to be the case that Perlite will prove to be more dangerous to Man than the guinea pig results would seem to indicate. The tuberculous complication may, on the contrary, also prove to be either less or more serious in Man. To test these possibilities we would like to recommend a study of the effects of Perlite on a group of about 10 monkeys.

The tuberculous complication was shown to heal by means of scarring. This is the result without chemotherapy. It is deemed to be a matter of great practical importance to study the influence of the modern chemo- and biotherapeutic agents which have proved so successful in human tuberculosis. These drugs have, however, consistently been a failure in cases of tuberculosis complicating or combined with silicosis. At the most they hold the disease temporarily in check and relapse usually follows soon after the drug therapy is withdrawn.

The Saranac Laboratory has had experience with one other substance (aluminum) in which tuberculosis was stimulated by the metallic dust though the latter alone had no primary fibrosing effect on the lung tissue. In this case the tuberculosis responded rapidly and completely to chemotherapy. It is possible that the tuberculosis provoked by Perlite may show a similarly favorable effect provided therapy be introduced sufficiently early. It is therefore strongly recommended that experiments be sponsored in which the effect of drug therapy in Perlite induced tuberculosis can be determined. If the disease responds readily and permanently the outlook for Perlite will be considerably improved and it would be perhaps permissible to relax somewhat in respect of dust precautions. On the other hand, if the tuberculosis were in spite of therapy to follow the same inexorable course as does the tuberculosis provoked by quartz dust, even greater precautions will have to be observed. The residual fibrosis of the lungs may in itself be a severely crippling terminal result, judging by the extensive nature of the lesions in animals. The disability to be anticipated from such fibrosis may be compared with that found in association with advanced silicosis. It has been shown that a number of the fibrotic complications of pulmonary disease respond well to substances such as cortisone and ACTH. It would be of considerable medical interest to determine whether these same results could be expected in the case of the residual fibrosis likely to be induced by tuberculosis supervening on Perlite exposure. If ACTH or cortisone are likely to prove beneficial the gravity of the tuberculogenic propensity of Perlite would once more be minimized to some extent.

It seems highly probable that workmen will seldom be exposed to Perlite alone. It is even possible that Perlite will be commercially mixed with other substances such as cement, gypsum, talc, asbestos, etc. The effect of double exposures of this kind may be either worse or less harmful than the individual substances used in isolation. Cement, for instance, is, except for its influence in causing bronchitis, harmless to the lung tissue itself in spite of its high free silica content. Most of the silicates are similarly relatively innocuous. Quartz combined with talc or with asbestos, however, have an enhanced deleterious effect on lung tissue. Gypsum, on the contrary, tends to neutralize the effects of quartz to a certain extent. It cannot be predicted yet how any particular combination of substances will behave when introduced into human lungs as dust. If it is already the practice to combine Perlite with other substances it is recommended that their combined pathogenicity be experimentally explored.

It is of further importance to examine the particle size range under which exposures to Perlite are likely to take place. Recent studies in The Saranac Laboratory have revealed that substances which are inert in the larger particle size range may nevertheless become pathogenic in smaller and especially ultra microscopic particle size range. As shown in Table 1 the median size of the 1.64 per cent of particles in the Perlite

we studied was less than 5 microns. However, in the dust clouds raised in the Perlite chamber we maintained an average particle size of 82 per cent of the microns. Quartz of a particle size larger than 5 microns is virtually inert and the particle size range exerting maximum pathogenic effects lies below 0.5 microns. In the Perlite experiment fewer than 50 per cent of the particles were less than 2 microns and less than 15 per cent were under 1 micron in size. It should be pointed out, therefore, that the favorable result thus far obtained with Perlite only applies for products comparable to that represented by Substance MD 201. Should Perlite products be marketed or used in such a way as to yield dust clouds with a particle size range considerably smaller than the median diameter of 7 micron diameters we have studied thus far it will be necessary to re-examine their separate effects individually. The smaller the particle sizes to be encountered the more necessary will a renewed experimentation become. It must also be pointed out that the present product contained 1 per cent quartz only in the fraction with a particle size smaller than 200 mesh. Any other products with a quartz content substantially higher than 1 per cent will require renewed experimentation. It is particularly to be emphasized also that when quartz is heated to temperatures in the range of 3000° F. to 2300° F. it may undergo a change with its isomers tridymite and cristobalite. These substances are appreciably more harmful to human and animal lungs than is quartz itself. Should Perlite be treated commercially to such temperature ranges it will be necessary to determine the presence or absence of cristobalite and tridymite, and if these are found the whole issue will have to be reviewed.

VII. SUMMARY

1. An experiment, designed to reveal the influence of inhaled Perlite dust upon the R_1 tuberculosis in guinea pigs during a period after removal of the animals from dust exposure to normal air, has been completed.

2. The previously observed stimulatory action of Perlite dust upon R_1 tuberculosis was a gain demonstrated and was shown to persist for about 8 months after interruption of dust exposure. Beyond 8 months, however, the stimulatory action diminished and the disease healed.

3. Healing occurred by means of scar formation, rather than by disappearance (resolution) of the tissue reaction and restoration of the lung to normal.

4. An overall evaluation of the present study and the earlier one previously reported, when compared with similar studies with quartz dust, warrants the suggestion that the tentative allowable dust concentrations for Perlite of the type studied at The Saranac Laboratory might be somewhat higher than that for quartz. On the other hand, if Perlite products are to be marketed with either a higher free silica content or with a greater proportion of fine particles less than 5 microns in diameter the whole situation will require to be reviewed.

Table 1

PHYSICAL AND CHEMICAL CHARACTERISTICS OF PERLITE - PRODUCT MD 201

I. Screening Test

Percentage of Bulk Sample within Each Range of Sieve Sizes

Greater than 20 mesh	-	32.0%
20 to 40 mesh	-	38.2%
40 to 100 mesh	-	20.8%
100 to 200 mesh	-	7.0%
Smaller than 200 mesh	-	1.64%

II. Median Size Range of Portion Smaller than 200 Mesh: 7 microns.

III. Quartz Content of Portion Smaller than 200 Mesh: 1.0%

IV. Chemical Composition of Bulk Sample

SiO_2	75.45
Fe_2O_3	0.55
TiO_2	0.05
Al_2O_3	12.92
CaO	0.85
MnO	0.07
MgO	0.10
Na_2O	3.42
K_2O	4.81
Loss on ignition	1.51
	99.73

Table 2

VARIATION IN LEVELS OF DUSTINESS

Perlite Experimental Chambers:

September 19, 1951 - October 29, 1953

CURRENT NUMBER	NUMBER OF SAMPLES	AVERAGE in x 10^6/ft.^{3}	R-A-N-G-E			ABSOLUTE DISPERSION %	STANDARD DEVIATION in x 10^6/ft.^{3}	STANDARD ERROR in x 10^6/ft.^{3}	COEFFICIENT of VARIATION %	YEAR
			Maximum	Mass Median	Minimum					
I	10	564.4	690.0	587.1	335.5	81.8	146.6	46.6	26.4	1951
II	40	604.4	738.35	600.85	507.0	110.7	223.0	35.7	37.0	1952
III	30	561.0	636.56	572.35	502.55	136.8	255.9	46.9	45.6	1953
TOTAL AVERAGE IV	80	582.2	694.4	588.4	483.9	116.9	225.8	41.1	38.86	9/19/51 10/29/53
a	b	c	d	e	f	g	h	i	j	k

Table 1

COURSE OF R_1 TUBERCULOSIS IN GUINEA PIGS
EXPOSED TO PERLITE DUST AND IN UNEXPOSED CONTROLS

Group A — INFECTION PLUS PERLITE — Infection at Start of Dust Exposure					Group D — INFECTION WITHOUT DUST — Controls to Group A			
Animal Number	Duration in Months		Status of Disease		Animal Number	Duration in Months		Status of Disease
	Perlite Dust	Normal Air				Perlite Dust	Normal Air	
3 K	2	0	S-1	F-0	21 K	0	2	S-1
4 K	2	0	S-2	F-0	22 K	0	2	S-1
7 K	4	0	S-1	F-0	23 K	0	4	F-0
8 K	4	0	P-1	F-0	24 K	0	4	F-0
9 K	6	0	P-3	F-1	25 K	0	6	F-0
10 K	6	0	P-2	F-0	26 K	0	6	F-0
11 K	6	3	P-4	F-1	27 K	0	9	F-0
12 K	6	3	P-3	F-0	28 K	0	9	F-0
13 K	6	6	P-4	F-3	29 K	0	12	F-0
14 K	6	6	P-3	F-3	30 K	0	12	F-0
15 K	6	9	P-1	F-3	31 K	0	15	F-0
16 K	6	9	P-2	F-3	32 K	0	15	F-0
17	6	12	P-0	F-3	34 K	0	18	F-0
18	6	12	P-0	F-4	35 K	0	18	F-0

Key: K = Killed
D = Died
S = Stabilizing
P = Progressing
F = Fibrosis (Collagen deposition)
Numbers indicate relative extent of involvement.

Table 4

COURSE OF TUBERCULOSIS IN ANIMALS
EXPOSED TO PERLITE DUST AND IN INFECTED CONTROLS

Group B — INFECTION PLUS PERLITE — Infection at End of Dust Exposure					Group E — INFECTION WITHOUT DUST — Controls to Group B			
Animal Number	Duration in Months		Status of Disease		Animal Number	Duration in Months		Status of Disease
	Perlite Dust	Normal Air				Perlite Dust	Normal Air	
57 K	6	2	P-2	F-0	76	0	2	S-1
58 K	6	2	P-2	F-0	77	0	2	S-1
60 K	6	4	P-2	F-1	78	0	4	F-0
61 K	6	4	P-2	F-1	79	0	4	F-0
63 K	6	6	P-2	F-2	80	0	6	F-0
64 K	6	6	P-1	F-0	81	0	6	F-0
67 D	6	6	P-4	F-1				
68 K	6	8	P-3	F-1	82	0	8	S-1
69 K	6	8	P-2	F-1	83	0	8	F-0
74 D	6	11	P-1	F-3				
72 K	6	12	P-1	F-2	85	0	12	F-0
73 K	6	12	P-0	F-2	86	0	12	S-1
75 K	6	12	P-0	F-2	88	0	12	F-0
					89	0	12	F-0
					90	0	12	F-0

Key: K = Killed
D = Died
S = Stabilizing
P = Progressing
F = Fibrosis (Collagen deposition)
Numbers indicate relative extent of involvement.

PERLITE - TRUDEAU

Room 7
Period 9/19/51 - 12/14/51

The Saranac Laboratory
Perlite Institute
Final Report 3-19-55

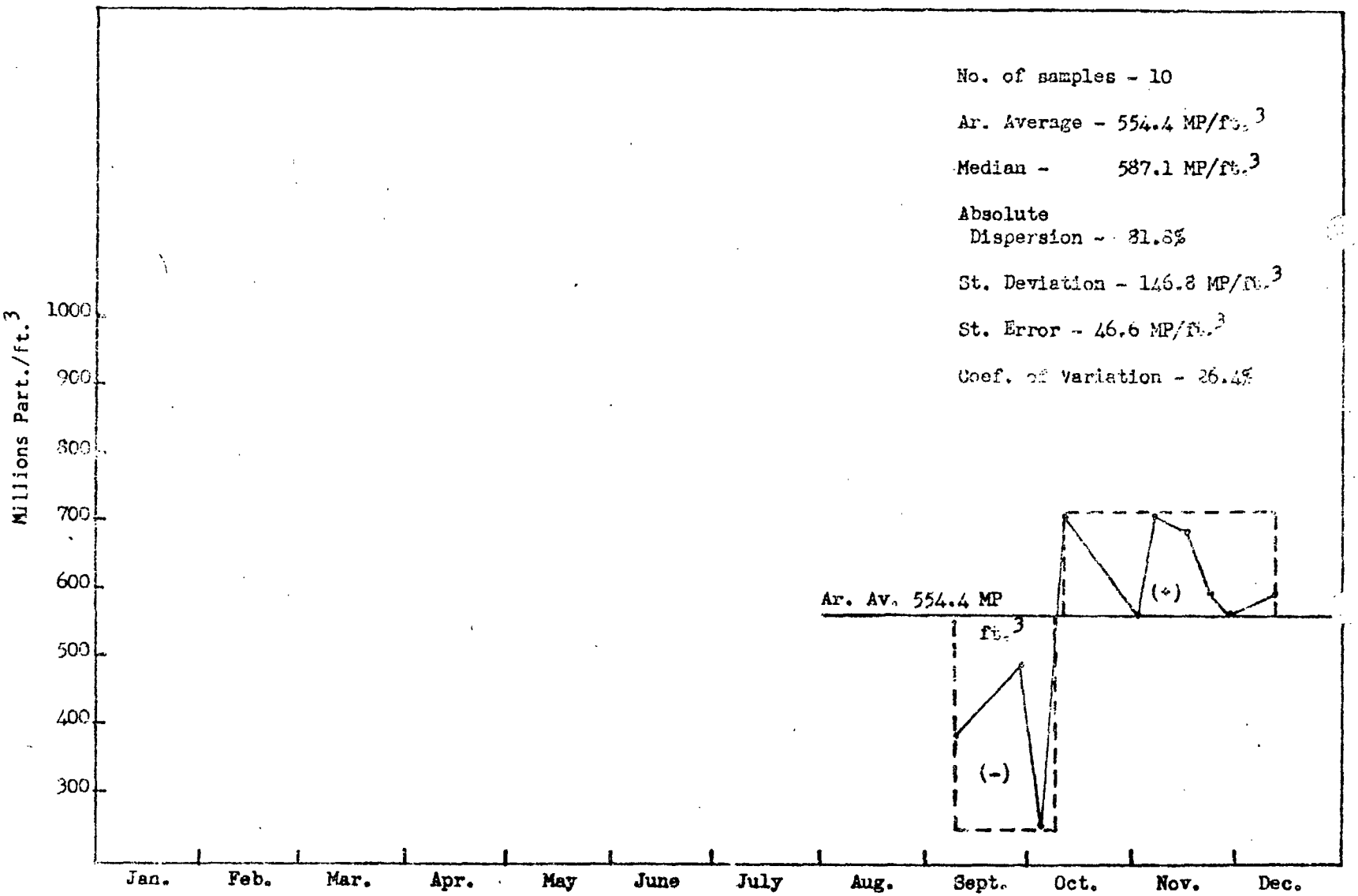

Figure 1

MTC 013957

PERLITE - TRUDEAU

Room 7
Period 1/9/52 - 12/17/52

The Saranac Laboratory
Perlite Institute
Final Report 3-19-55

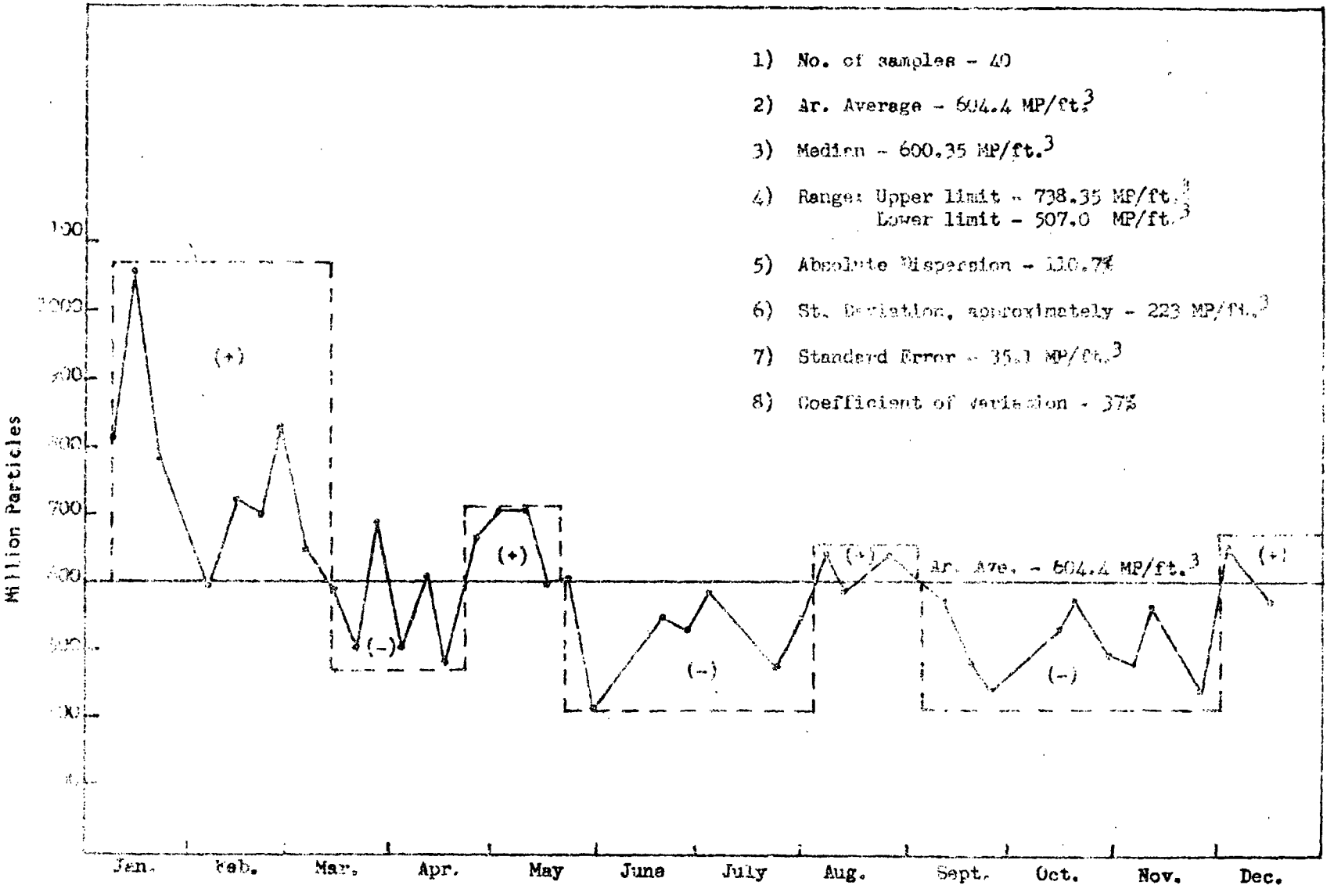

Figure 2

MTC 013958

Trudeau - Room 7
Until 3/4/53

Saranac Laboratory - Room 2
3/4/53 - 10/15/53

PERLITE

The Saranac Laboratory
Perlite Institute
Final Report 3-19-55

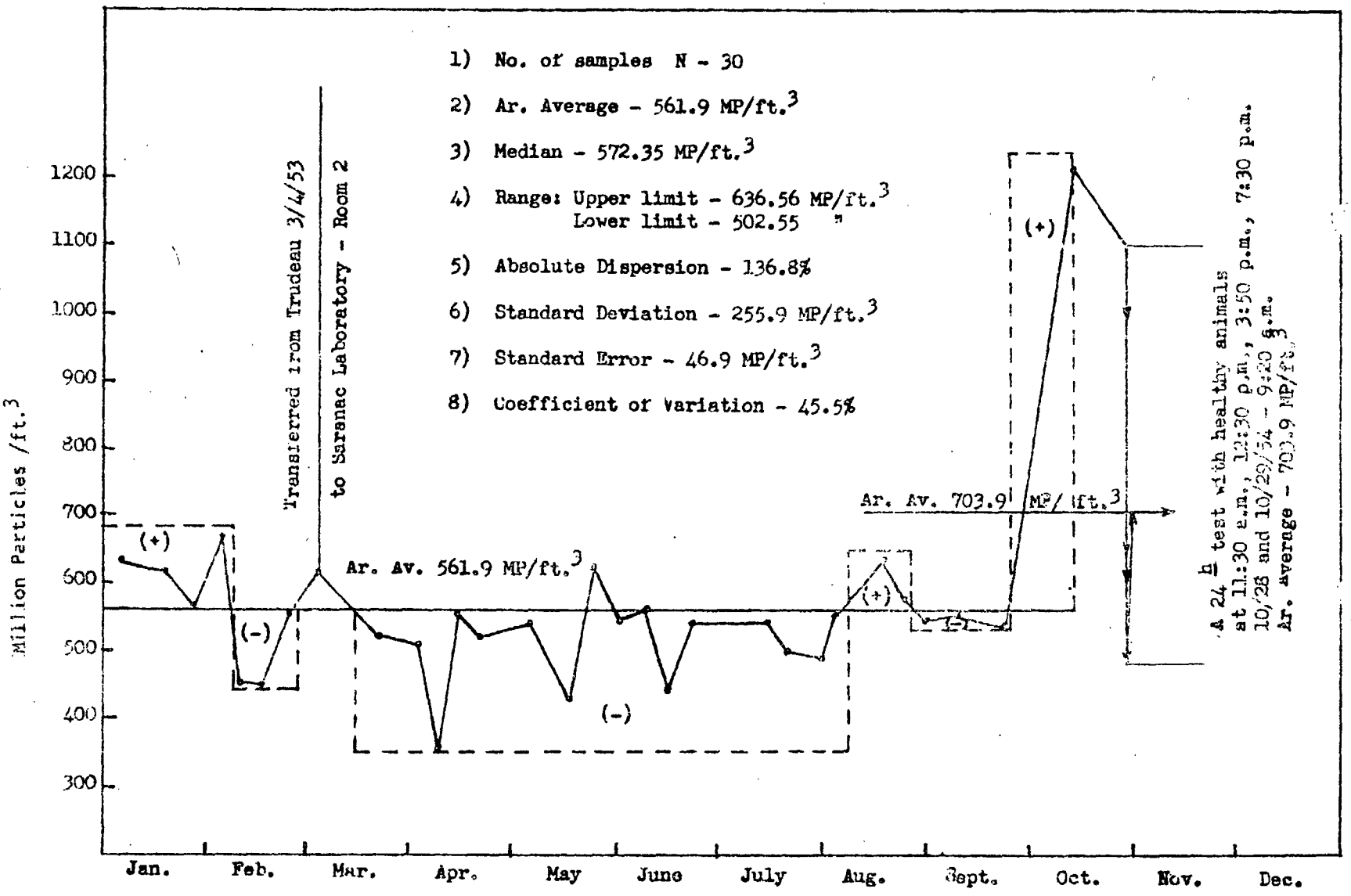

Figure 3

MTC 013959

SIZE FREQUENCY DISTRIBUTION
of
AIRBORNE DUST

1. Average for the experimental period - _____

2. Absolute dispersion around the averages -

(+) above
(-) below

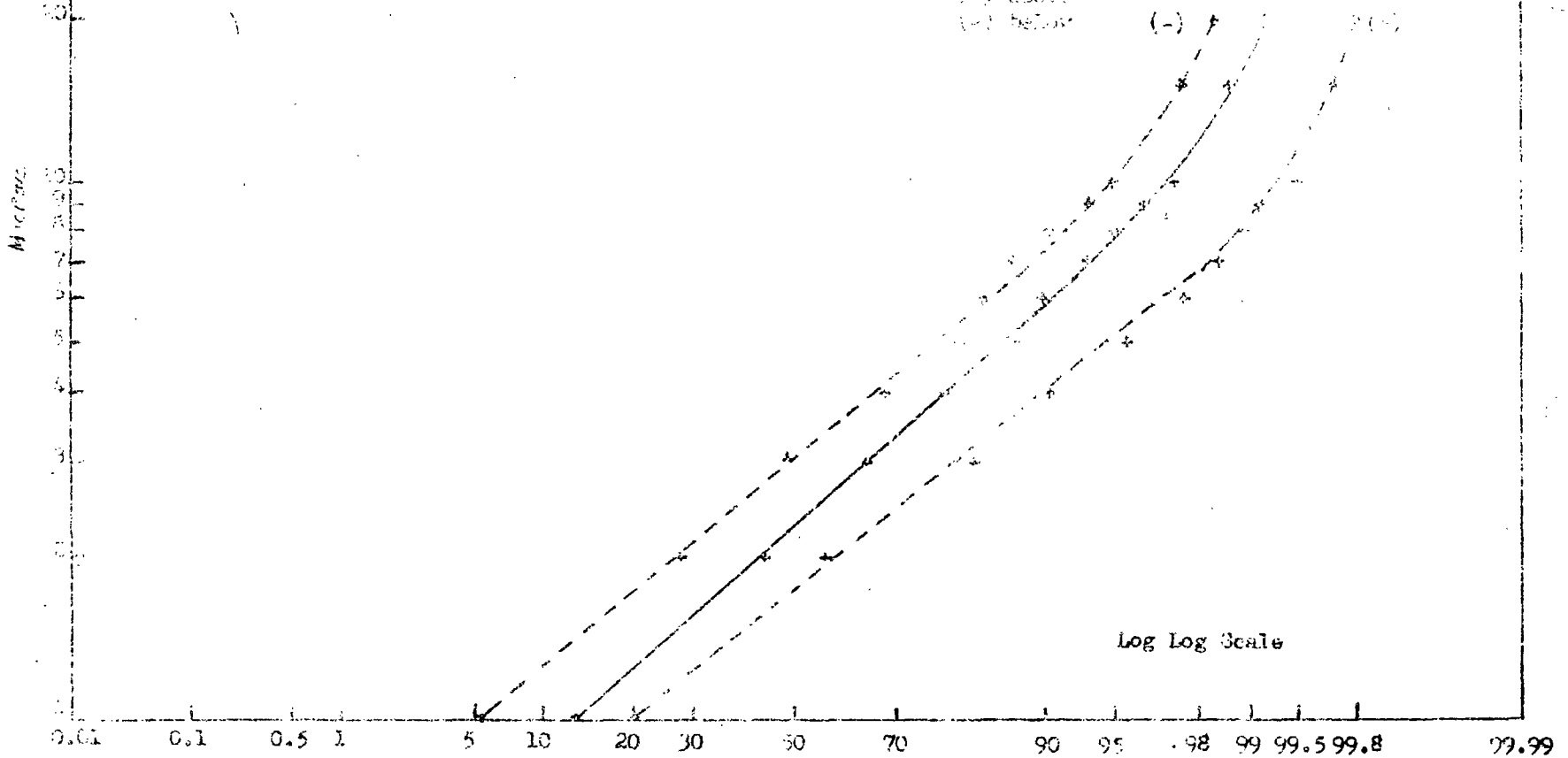

Figure 4

Saranac Laboratory
Perlite Institute
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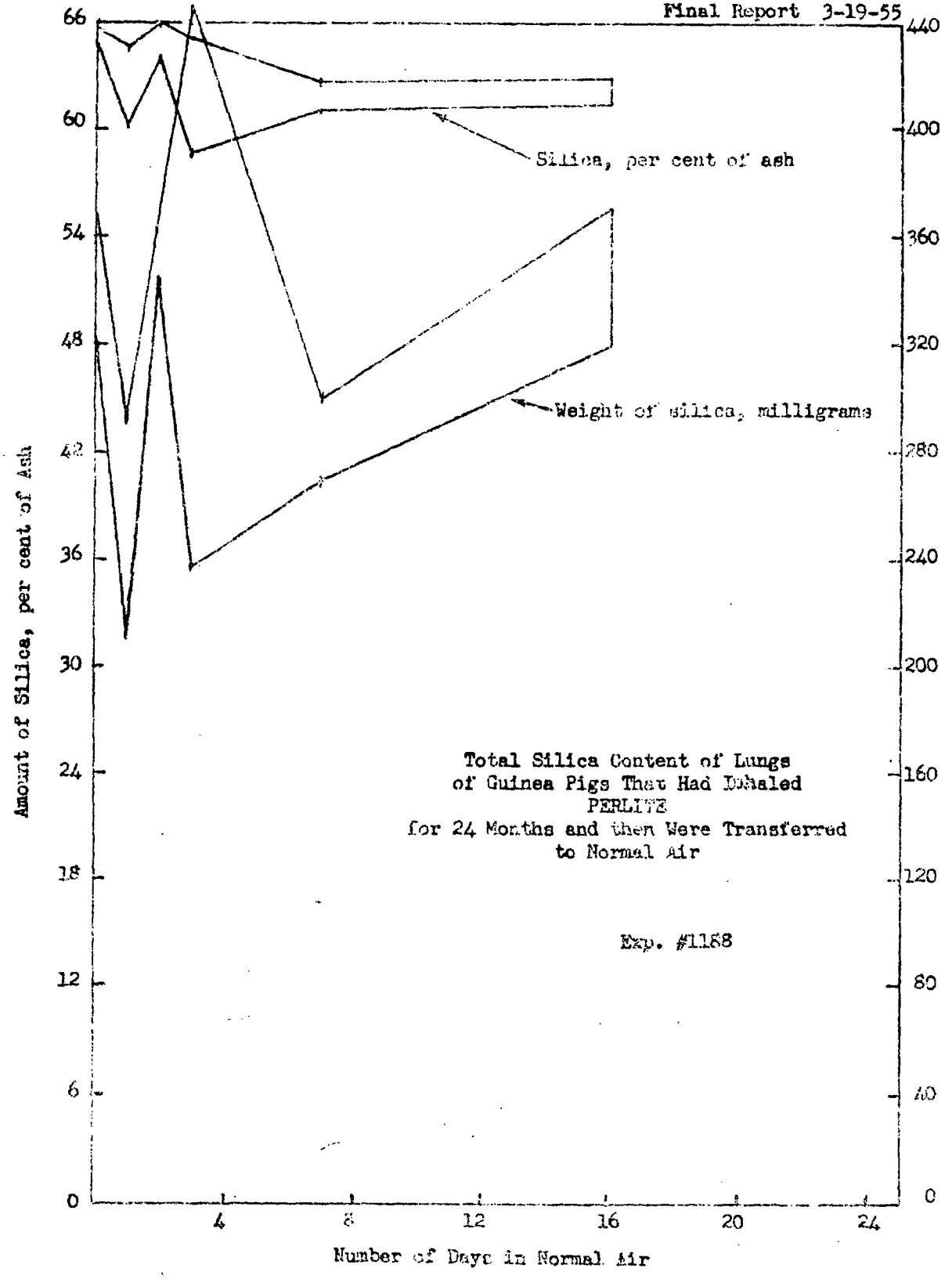


Figure 5

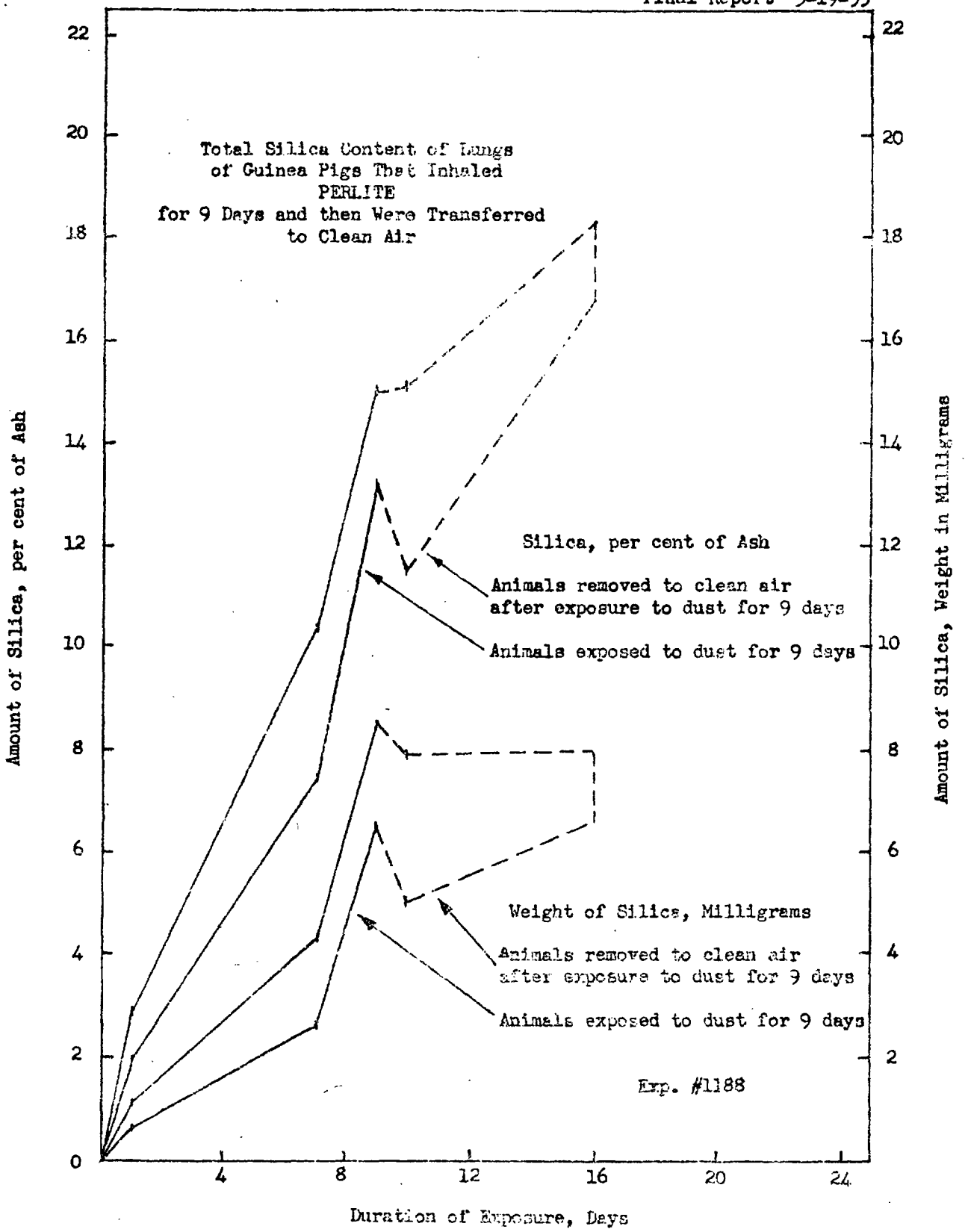


Figure 6

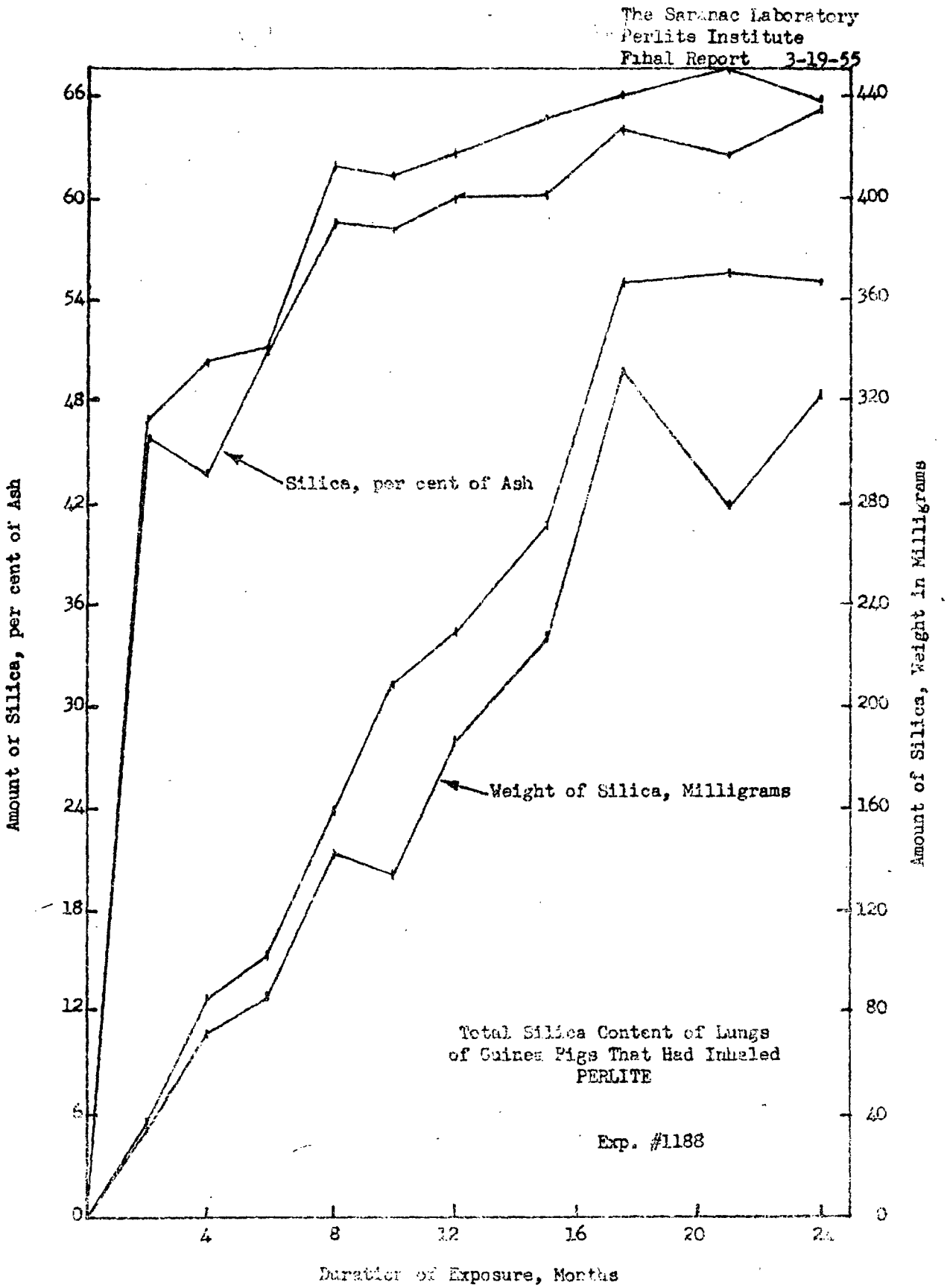


Figure 7

MTC 013963

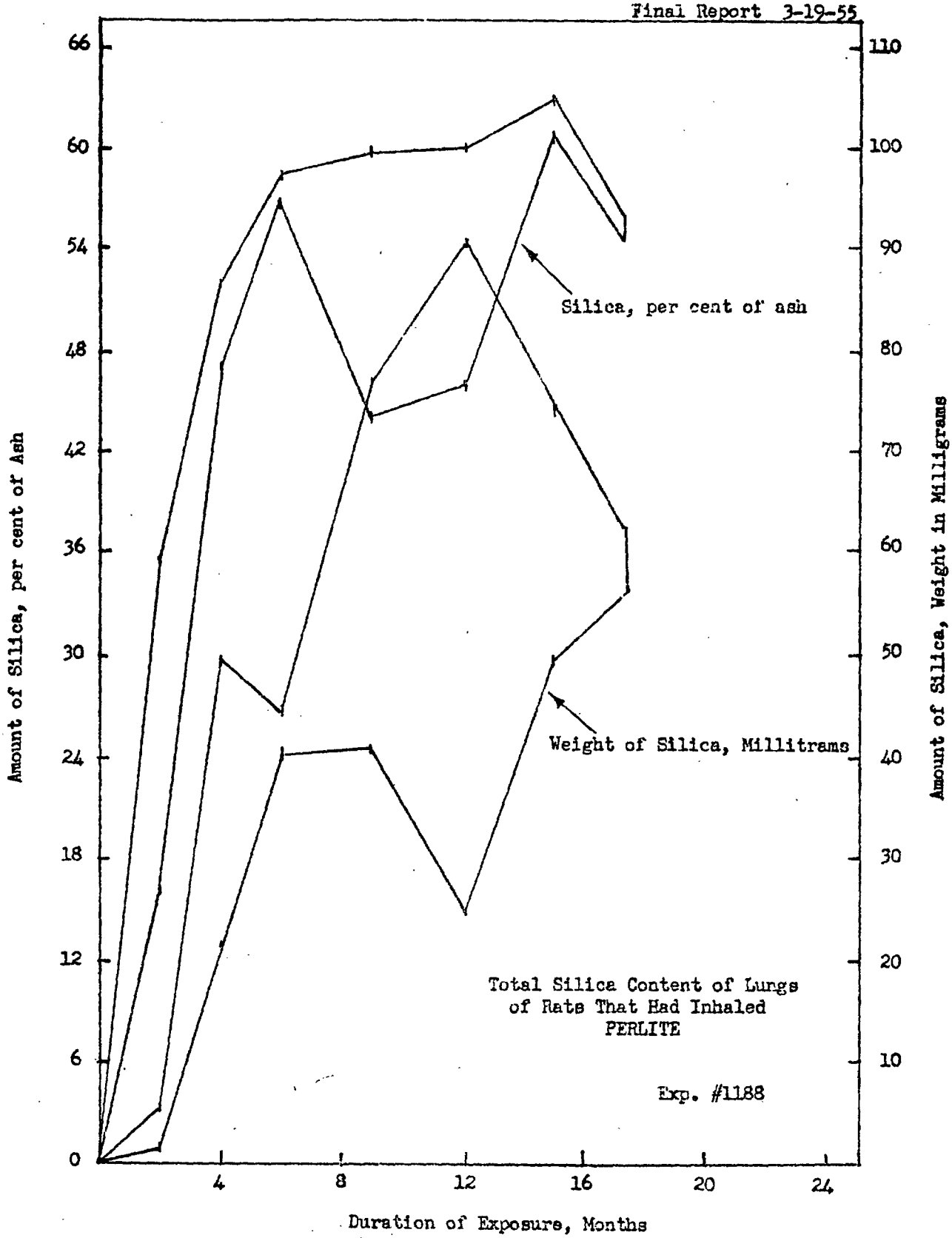


Figure 8

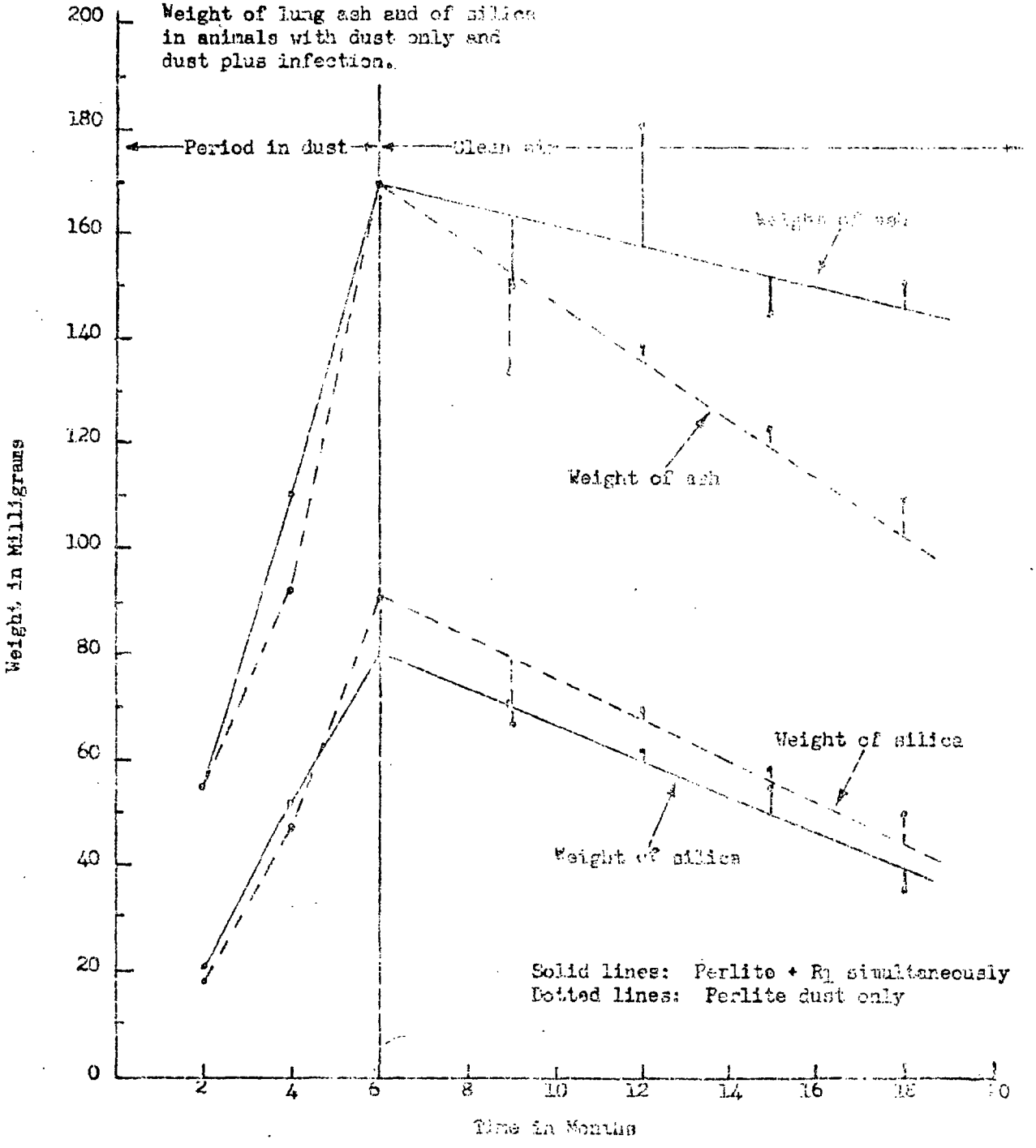


Figure 9

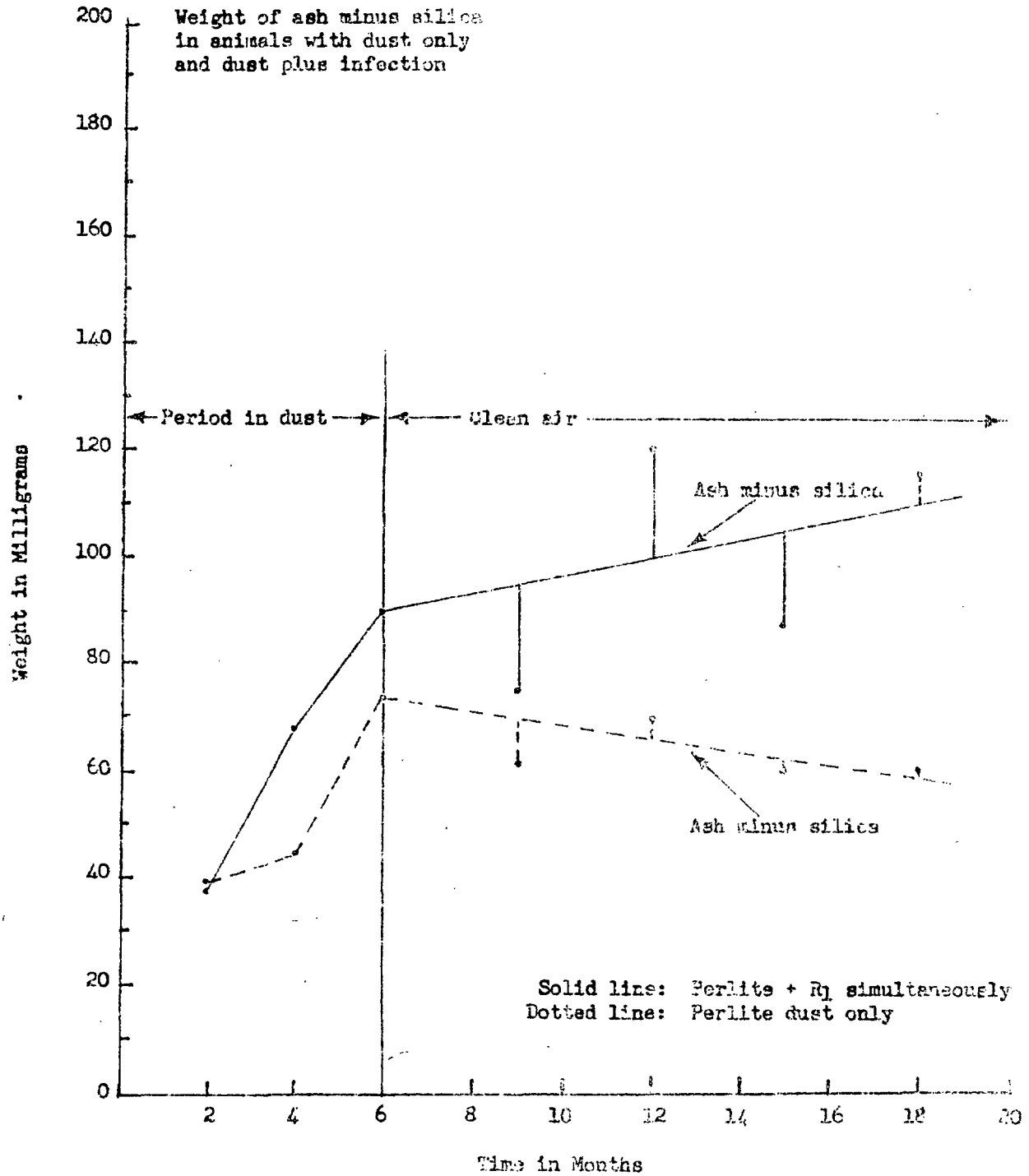
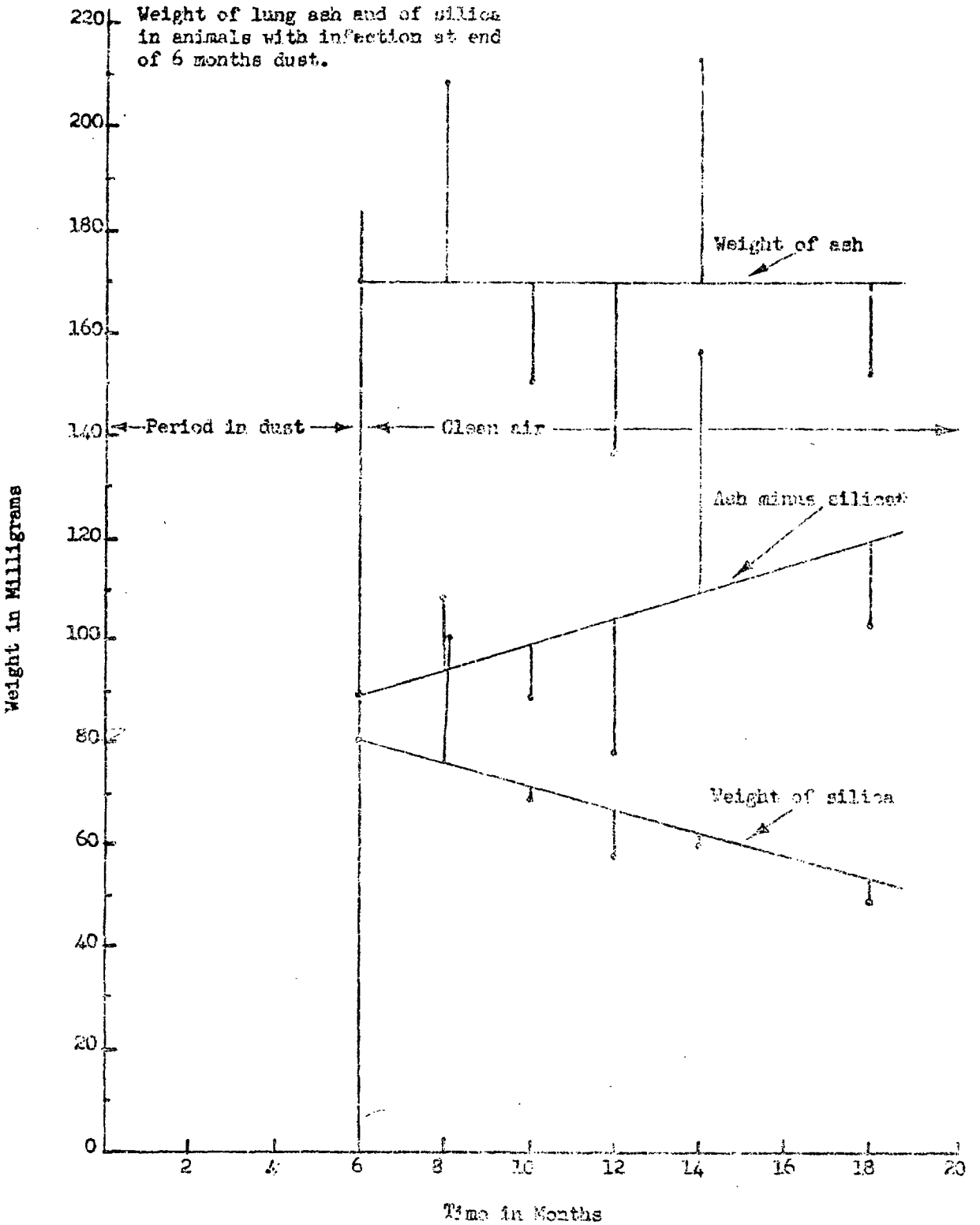


Figure 10



*Compare this line for ash-minus-silica with the line in Chart B for ash-minus-silica in non-infected animals.

Figure 11

SARANAC LABORATORY
"PERLITE" EXPERIMENT

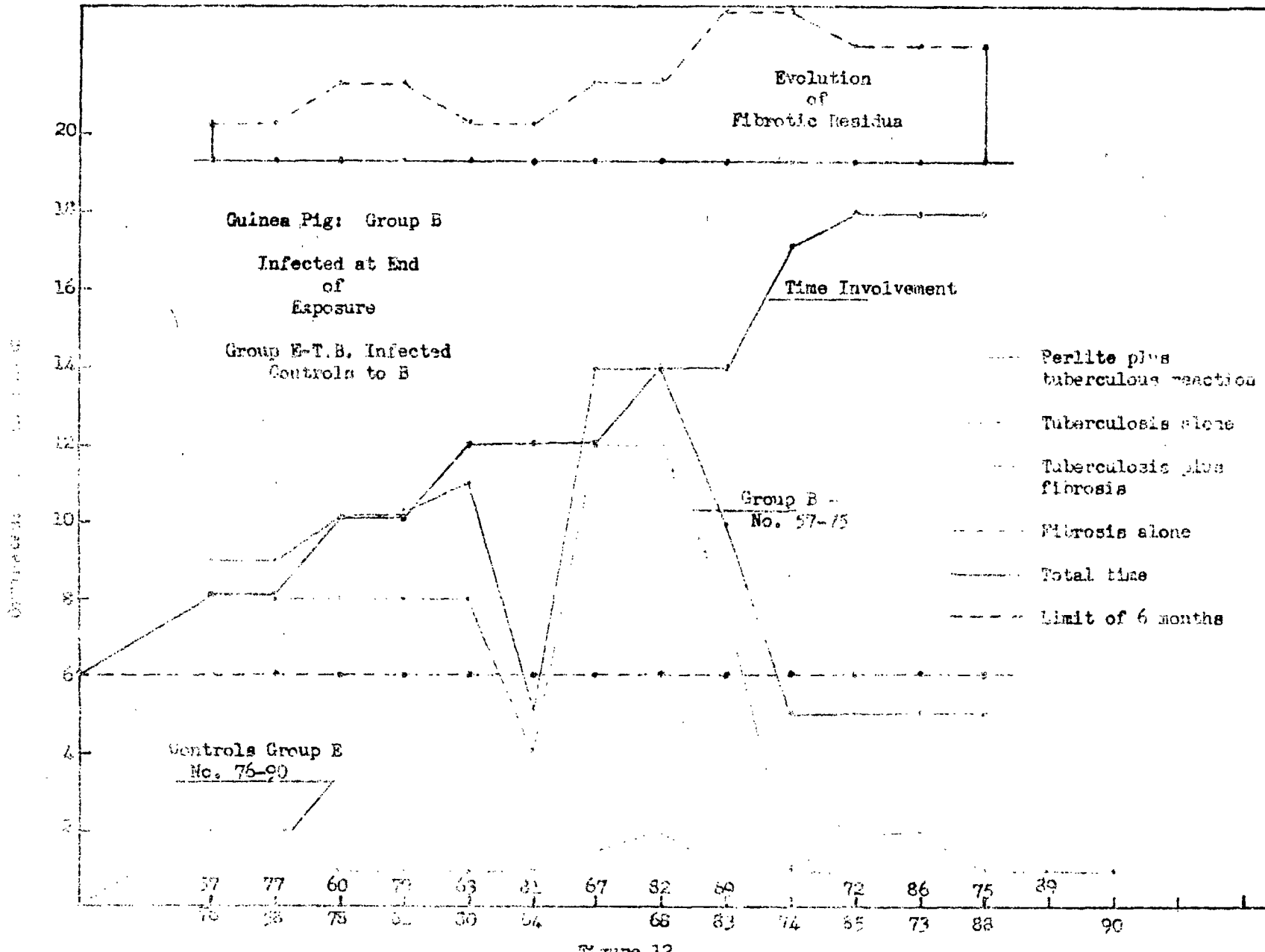


Figure 12

MTC 013968

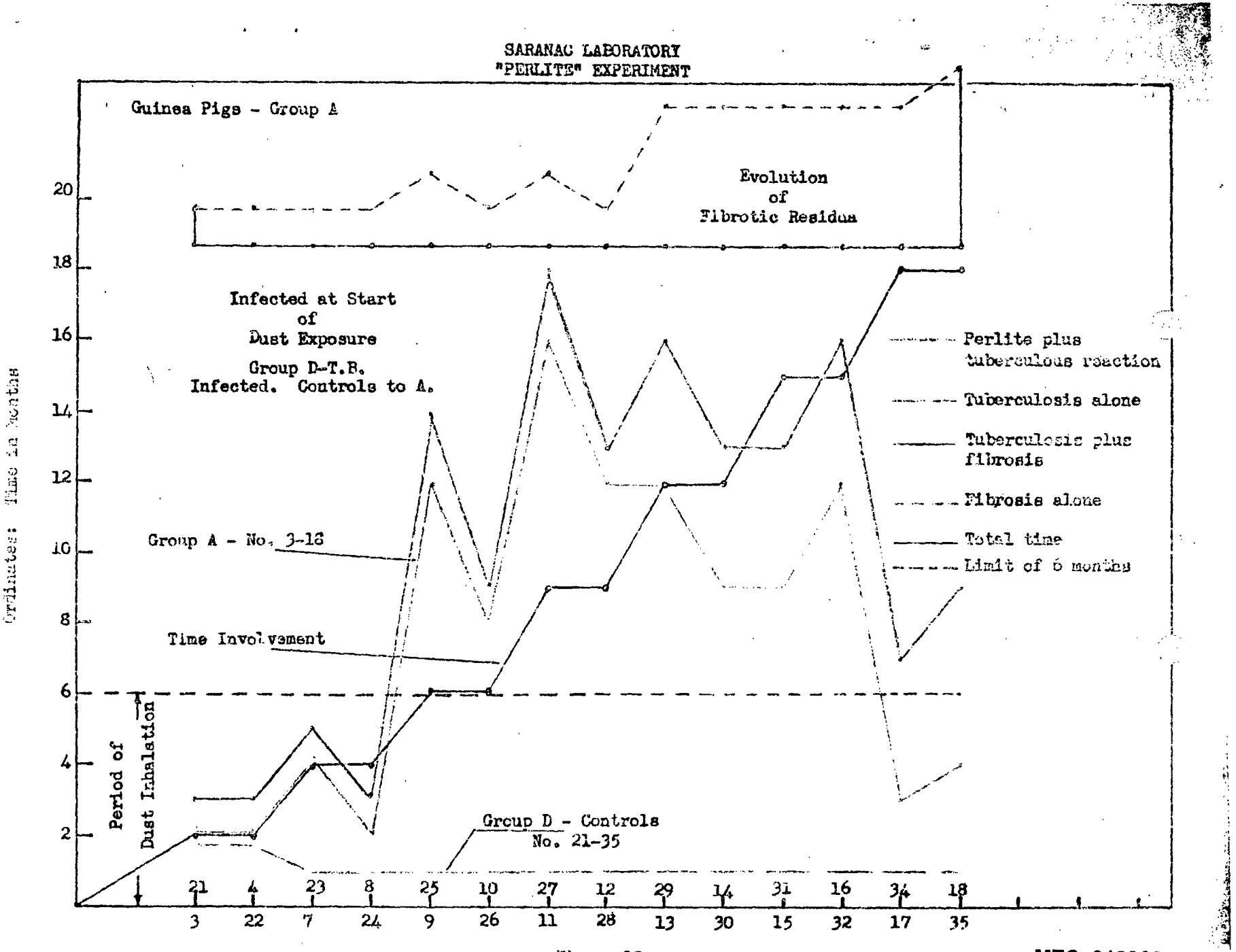
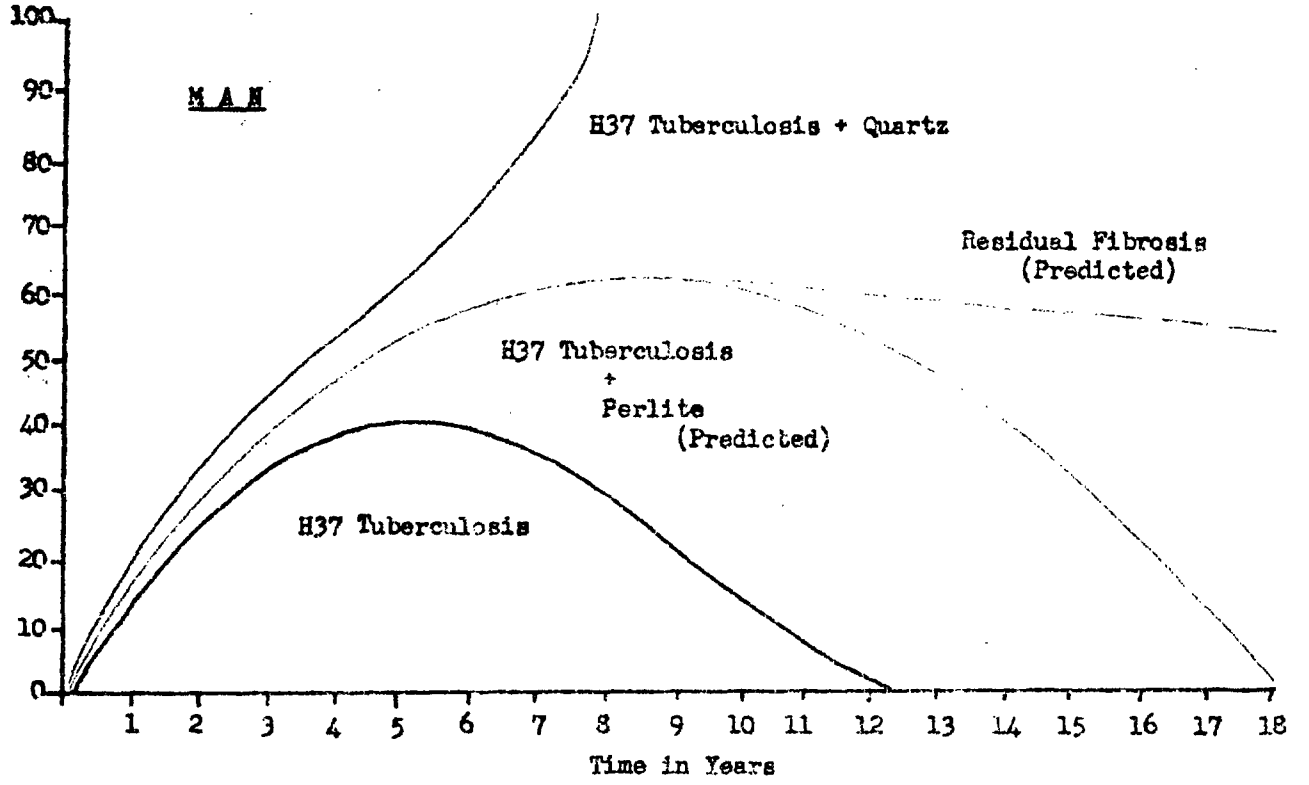


Figure 13

MTC 013969

RE[illegible]IVE INFLUENCE OF PERLITE AND QU[illegible]TZ ON THE COURSE OF A TUBERCULOUS INFECTION



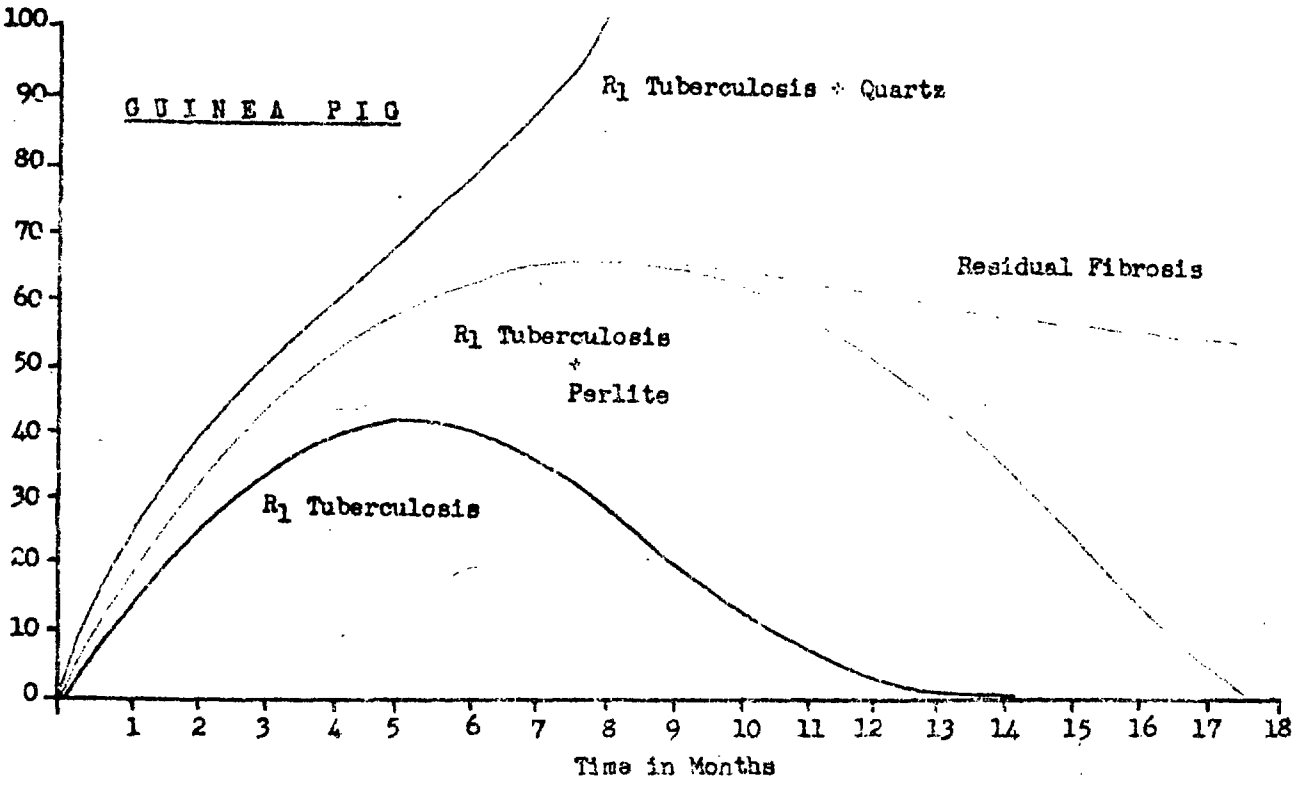


Figure 14

MTC 013970