Bull Yamaguchi Med Sch 30 : 55-63, 1983

Chest X-ray Findings of Woodworkers, Epidemiological and Experimental Study

Isao Okita, Takashi Nakanishi* and Genkichi Asaeda***

*Department of Radiology, **Department of Hygiene, Yamaguchi University School of Medicine, Ube, Yamaguchi 755, Japan

(Received March 1, revised November 28, 1983)

Abstract Mass examination of the laborers in wood and products industries was conducted. The workers showing abnormal chest x-ray findings were over 40 years of age and had worked longer than 20 years in these industries. The abnormal x-ray findings were linear and small opacities. From these observations, it is likely that the abnormal findings of chest x-ray films were associated with wooden dust. The experimental studies by infusing paulownia dust into the lungs of rats showed the foreign body granuloma. These epidemiological and experimental studies suggest that wooden dust, namely paulownia, causes pneumoconiosis in the wood workers.

Key Words: Woodworkers, saw dust, pneumoconiosis, chest x-ray findings

Introduction

Woodworkers often suffer from coryza-like disorders and bronchial asthma. Various reports describe the disorders to be a reaction to allergen. Ito[1] has clinically reported bronchial asthma caused by the minute saw dust of Thuja Standishi. Sano[2] and Abe[3] reported an animal experiment using Japanese cedar dust. A clinical case of saw dust does not seem to have been reported so far. Recently, a man who had manufactured furniture and fixtures from lumber as well as other wood products consulted the out-clinic.

He had been working with these products for 45 years. His chest x-ray showed linear opacities and small opacities suspected to be pneumoconiosis. It was believed that abnormal findings were fibrosis as a result of being exposed to saw dust for a long period of time. Consequently, an epidemiological survey and an animal experiment were conducted.

Case Report

Case: 60-year-old-male.

Chief Complaints: cough, sputum, dyspnea and general fatigue.

Past History: not remarkable. no history of allergies.

Family History: not remarkable.

Present Illness: Up until this illness, the patient was healthy. In June of 1967, an abnormal shadow was discovered in his chest x-ray film following a complete physical examination. He consulted our out-patient clinic in June of 1967 with the chief com-

plaints listed above.

Occupational History: He had been employed as a furniture and fixture maker for 32 years; since he was 15 years old. He then manufactured lumber and other wood products for 13 years. All together, he had been employed for 45 years in the wood processing industry. He had handled paulownia, cryptomeria, lauan, etc., and there had been a considerable amount of saw dust in his area of work. He had never used polishing powder.

Laboratory Findings: Skin reaction to cryptomeria: negative. B.S.R.: 1 h, 9 mm, 2 h, 18 mm, B.P. 150/80 mmHg. CRP (−). The white blood cell count was 4900 with the following differential: 66 per cent segmental cells, 27 per cent lymphocytes, 1 per cent basophil, and 6 per cent monocytes. The red blood cell count was 4.89 million with 19.5 g of hemoglobin. Systemic blood chemistry was not unusual.

Pulmonary Function Test: FVC 3785 ml, $FEV_{1.0}$ 2421 ml, %VC 110%, $FEV_{1.0\%}$ 63.9% (obstructive impairment), MBC 43.7 1/min/m^2 (slight impairment), EI 58.3% (high impairment).

Chest X-ray: The routine chest x-rays showed the crossing of linear and strand opacities, and the scattering of small opacities in both lungs from middle fields to lower fields. In the left lung these abnormilities had spread gradually to the upper fields. There were bullae at the outer side of the left upper field (Fig. 1). The direct two-fold magnification radiogram showed deformities of blood vessels such as bending, beading, meandering and partial fragmentation (Fig. 2). Scintigram of lung using ^{131}I-MAA did not show impaired pulmonary blood flow. As mentioned above, this patient had worked around saw dust for 45 years and had never used polishing powder. His chest x-rays showed linear and nodular opacities similar

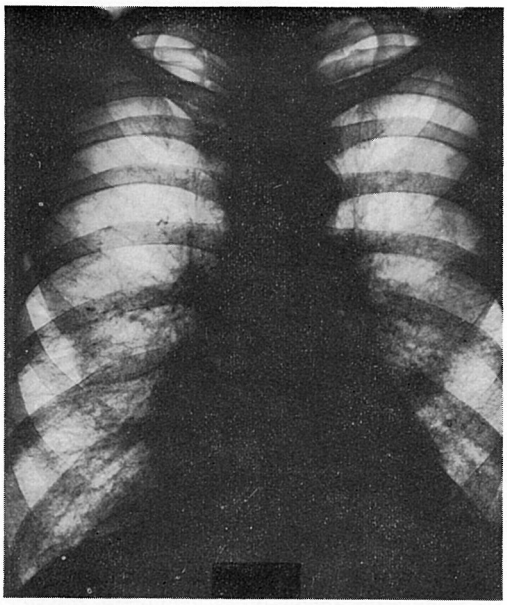

Fig. 1 60-year-old-male
Linear opacities and puncti-form opacities are seen in middle to lower fields bilaterally.

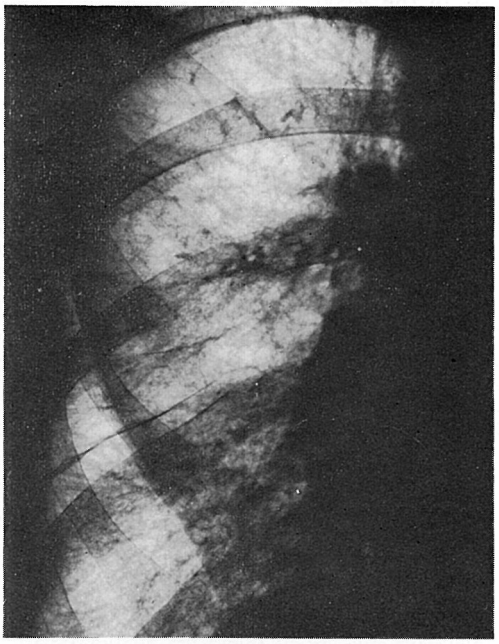

Fig. 2 Part of direct two fold magnification radiogram of Fig. 1.

to pneumoconiosis. These abnormal findings were assumed to be pulmonary fibrosis which had occurred as a result of inhaling saw dust over a long period of time.

Epidermiological Investigation.

To study the chest x-ray findings of workers who had been employed in lumber and wood products, and furniture and fixture manufacturing companies. individual workshops were randomly selected in Yamaguchi Prefecture. Workers employed at these workshops were given chest x-rays and a medical examination. The concentration of dust of these workshops was measured, and was within the legal limit. See Table 1. Those

Table 1 Pollution by wooden dust in workshops (mg/m^3)

		Furniture and fixtures	Lumber and wood products
Needle leaved trees	with local ventilation	0.40	0.12
		0.44	0.16
			0.23
			0.20
	(mean)	(0.42)	(0.18)
	without local ventilation	0.27	0.26
		0.86	0.25
		0.49	0.37
		1.16	0.33
			0.40
			0.42
			0.31
	(mean)	(0.70)	(0.33)
Lauan	without local ventilation	1.66	
		2.86	

measured with digital dust meter P-3 Type.

who took direct chest x-ray films totalled 121 persons. Table 2 and Table 3 show the age and occupational periods of these workers. Age is relative to the duration of employment for these workers ($r=0.60$, 0.01 sig.).

Table 2 Ages of subjects

Age	Number of subjects
under 29	26
30-39	31
40-49	28
over 50	36
Total	121

Table 3 The duration of dust exposure in subjects

Duration of dust exposure	Number of subjects
under 9 years	33
10-19 years	40
over 20 years	48
Total	121

1. X-ray findings.

In these x-rays 11 workers showed linear opacities and 5 workers showed small opacities (Tab. 4, Tab. 5). L_1 totalled 9 workers; L_2, 2. P_1 totalled 4 workers; P_2, 1. These workers whose chest x-rays showed abnormalities were over 40 years of age and had occupational periods greater than 20 years, and one worker was between 30-39 years of age. In comparing those over 40 with those under 40, there was a significant difference of the rate of findings in their chest x-rays. Chi-square test was done to determine frequency of these linear opacities in relation to age. Over 40 and under 40 years of age showed (16%), (5%) respectively significant ($\chi_S^2=5.44$). Over 50 and under 50 years of age showed (22%), (1%) respectively significant ($\chi_S^2=8.55$). A significant difference is recognized beyond 40 years of age and it is extremely evident beyond 50 years of age.

2. Investigation of chest x-ray findings of industrial classification.

We classified the workers into either furniture and fixture products, or lumber and

Table 4 Relationship between incidence of wood pneumoconiosis and age

Age	Linear opacities					Small opacities					
	O	L_1	L_2	L_3	L_4	O	p_1	p_2	p_3	m	n
-29	26	0	0	0	0	26	0	0	0	0	0
30-39	30	1	0	0	0	31	0	0	0	0	0
40-49	26	2	0	0	0	27	1	0	0	0	0
50-	28	6	2	0	0	32	3	1	0	0	0
Total	110	9	2	0	0	116	4	1	0	0	0

Table 5 Relationship between incidence of wood pneumoconiosis and duration of dust exposure

Duration of dust	Linear opacities					Small opacities					
exposure	0	L_1	L_2	L_3	L_4	0	p_1	p_2	p_3	m	n
under 9 years	31	2	0	0	0	33	0	0	0	0	0
10-19 years	39	0	1	0	0	39	0	1	0	0	0
over 20 years	40	7	1	0	0	44	4	0	0	0	0
Total	110	9	2	0	0	116	4	1	0	0	0

Table 6 Incidence of linear opacities in the miscellaneous workers through their ages

Occupations	Furniture and fixture		Lumber and wood products	
X-ray findings / Age	normal	abnormal	normal	abnormal
under 29 years	22	0	4	0
30-39 years	15	0	15	1
40-49 years	10	0	16	2
over 50 years	5	7	23	1
Total	52	7	58	4

wood products, and classified linear opacities according to age. See Table 6. Chi-square test was done to determine frequency of occurrence of linear opacities. See Table 7. Frequency of linear opacities in the area of furniture and fixture was greater than that in the area of lumber and wood products.

3. Measurement of concentration of saw dust of industrial classification.

The concentration of saw dust in the work area was measured using a digital dust meter P-3 type according to the methods outlined by the Japan Journal of Industrial Health. The average of this measurement

Table 7 Incidence of linear opacities in the miscellaneous cases with pneumoconiosis using Chi-Square Test

Occupations Age	Furniture and fixture		Lumber and wood products
over 30	18%		7%
over 40	32%	*	7%
over 50	42%	**	4%

* $\chi_S^2=4.93>3.84$ (0.05) 5% significant
** $\chi_S^2=10.63>6.64$ (0.01) 1% significant

was compared between furniture and fixture, and lumber and wood products employers.

Needle-leaved trees were only included in this measurement. The average of furniture and fixture makers was 0.60 mg/m³, N=6. The average of lumber and wood products makers was 0.28 mg/m³, N=11. The break up ratio is $F_S=11.95>F_{10}^5=3.33$ (1% sig.). Comparing both averages, using the method of computing approximate value comes to d=2.313>2.242. The value for furniture and fixture is recognized as significantly greater than that for lumber and wood products.

4. The influence of volume of saw dust using local ventilation.

The comparison of dust value of lumber and wood products was done in area with and without local ventilation.

Average values: With local ventilation 0.18 mg/m³.

Without local ventilation 0.33 mg/m³.

5. Health investigation.

Individual questions regarding subjective symptoms were answered by 107 persons. These questions were; shortness of breath, cough, sputum, chest pain and asthma. Shortness of breath was found in 4 persons, cough in 11, sputum in 12 and chest pain in 7. Only one person who worked in the area of furniture and fixtures complained of asthma. The volume of saw dust in the work area with local ventilation using an impinger was 1.2 mg/m³. The size of particles is as follows:

under 1μm	31.25%
1-5μm	55.13%
5-10μm	12.61%
more than 10μm	2.01%

As mentioned above, laborers who have worked with wood and wood products were observed with abnormal findings in chest x-rays. Moreover, workers who were over 40 and who had been working longer than 20 years had more frequent abnormal chest x-ray findings than those who have been engaged in the lumber and wood products business. The furniture and fixture work area had a higher saw dust volume than the lumber and wood products area.

Experimental Study.

Material and Methods.

Seventy five rats were used for this experiment in groups of five. Male-4-week-old rats were given suitable solid food and drinking water. Paulownia dust was used for injections. 0.5 g of dust was crushed for 5 min with a ring mill, then sterilized and 10 ml of physiological solution of salt was added.

This mixture was then crushed and agitated for two minutes with a homogenizer and 200,000 units of penicillin was added to this solution. The suspension was administered after sufficient agitation. The diameter of dust is as follows:

under 1μm	61.68%
1-5μm	33.37%
5-10μm	3.46%
more than 10mμ	1.49%

In the constant-amount experiments, the dust suspensions were injected into the lungs of the rats intratracheally[4]. The rats were anesthesized lightly with ether, and were fixed in the supine position for skin incisions. The trachea was exposed by blunt dissection.

0.3 ml of the sterile dust suspension was administered. After infusion, about 1 ml of

air was injected in order to diffuse the suspending dust in the lung. Infusion was done only once for each rat. The wound was closed by a single suture. The rats were killed at weekly intervals up to 3 weeks, then at monthly intervals up to one year. The lungs of the killed rats were gently distended by injecting about 10 ml of 10% formal-saline through the trachea, exposed at the neck. Most of the air in the lungs was expressed by gentle pressure on the side of the thorax before the injection was made. The trachea was tied off, the thoracic cavity opened, and the lungs with the tied off position of the trachea, were removed and placed in the fixative. After preliminary fixation for four days, blocks were selected in the long axes of both the lungs at the level of the hilum to obtain maximum representative areas from each lung, and to include the hilar lymph nodes. After complete fixation, the blocks were embedded and cut in paraffin at 5 μm, then stained with haematoxyline and eosin.

Histological findings.

1 week post injection. The alveolar spaces where paulownia dust had existed were filled with many mononuclear phagocytes. The infiltrate did not invade the adjacent alveoli in which foreigh bodies were absent (Fig. 3). The affected alveoli were scattered in a patchy fashion throughout the lung.

2 weeks post injection. In the alveolar spaces numerous multi-nuclear giant cells appeared surrounding the foreign bodies with above mentioned phagocytes. The bordering alveolar walls were slightly thickened (Fig. 4).

1 month post injection. The number of phagocytic giant cells increased greatly since the 2 weeks observation. The proliferation of fibroblasts was seldom seen in this stage (Fig. 5).

3 months post injection. Granulomatous change as shown in Fig. 6. The alveolar

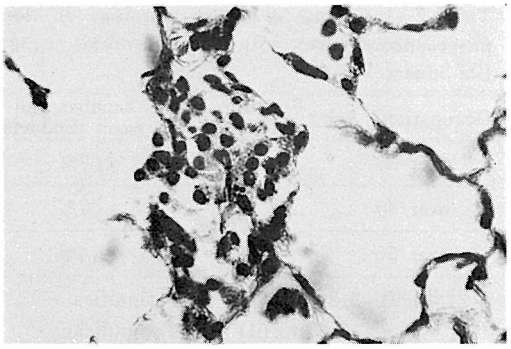

Fig. 3 Seven days post injection
Many phagocytes are migrated in alveolar spaces.

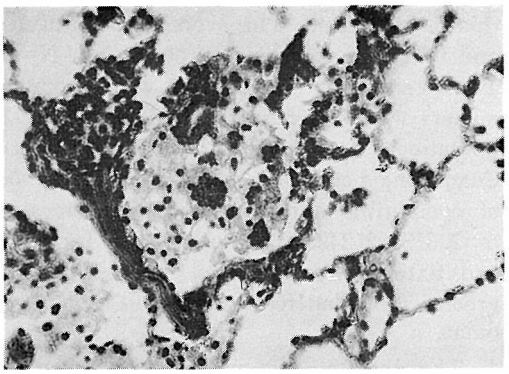

Fig. 4 Fourteen days post injection
Multinuclear giant cells are seen in the migrated areas of phagocytes.

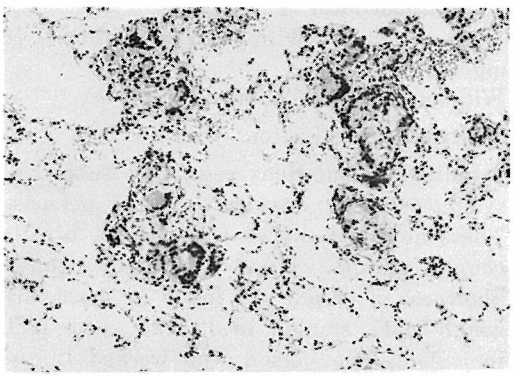

Fig. 5 One month post injection
Many multinucleated giant cells are migrated with mononuclear cells.

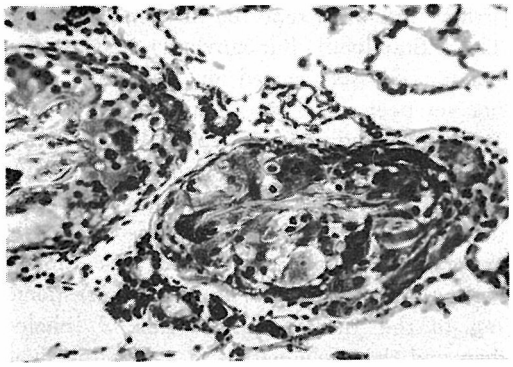

Fig. 6 Three months post injection
Granulomatous changes are begun in this stage.

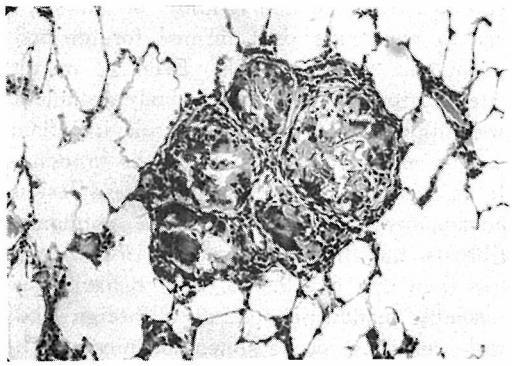

Fig. 7 Three months post injection
Several affected alveoli are aggregated and formed a foreigh body nodule.

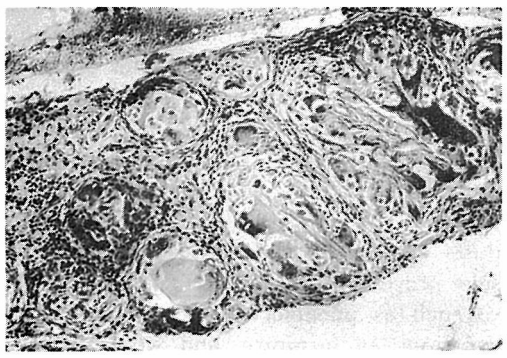

Fig. 8 Six months post injection
The dense fibros is seen in the periphery of the foreign body nodule.

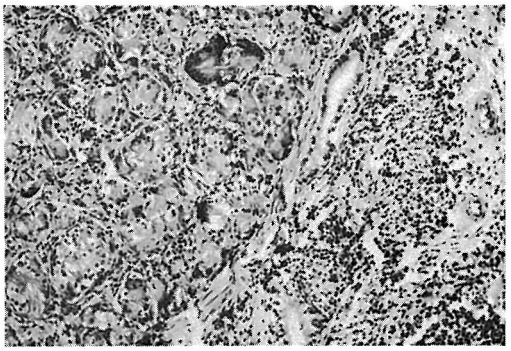

Fig. 9 Twelve months post injection
Some of the nodules are surrounded with infiltrated lymphocytes and histiocyte.

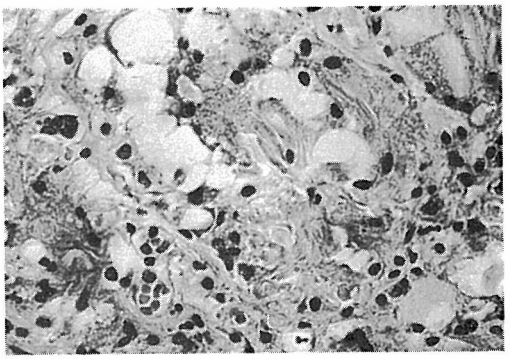

Fig. 10 Twelve months post injection
Vacuolar degeneration and necrosis of multinucleated giant cells.

spaces were replaced by increased foreign body giant cells and proliferated fibroblasts. The several affected alveoli were aggregated and formed a foreign body nodule (Fig. 7). Occasionally, atelectatic changes were found around nodules with inflammatory infiltrate.

6 months post injection. The nodule components were essentially the same as 3 months post injection. In the periphery of the nodule dense fibrosis was present, therefore the nodule was well demarcated (Fig. 8).

12 months post injection. Occasionally, a collar of lymphocytes and histiocytes surrounded some of the nodules so that the outline of the nodule was indistinct (Fig. 9).

Vacuolar degeneration and karyorrhexis were seen in some of the giant cells of the nodules (Fig. 10).

The fibrosis occurred in the outer zone of other nodules. However, the degree of fibrosis, in contrast with silicosis, was not as severe and the arrangement of fibers was not concentric in pneumoconiosis of saw dust. The foreign bodies were neither resolved nor absorbed even after 12 months. Throughout the experiment, the fusion and marked enlargement of nodules did not occur. In some nodules, necrosis and purulent change were also present. Chronic lymphadenitis was frequently found, but no foreign body reaction was observed in hilar lymph nodes.

The histological findings were summerized as follows:

In the early stage,

1. Emigration of phagocytes to foreigh bodies (saw dust).
2. Transformation of these cells into giant multi-nuclei cells.

In the late stage,

3. Formation of foreign body nodules.
4. Fibrosis with or without inflammatory infiltrate in the outer zone of the nodule.

Discussion

Much experimental research of pneumoconiosis has been reported since Gardner[5]. Experimental methods for dust reaction are inhalation, intratracheal infusion, abdominal infusion, bone marrow infusion, venous injection, subcutaneous injection, etc[6,7]. Most of these reports are concerned with inorganic dust but a few reports are concerned with organic dust. Up to this time inorganic dust has been related to pneumoconiosis, and organic dust to allergy and inflammation instead of pneumoconiosis. Reports on organic dust include Byssinosis[8], Suberosis[9], Farmer's Lung[10], Josstick Lung[11], Straw dust Lung[12], etc. Related to wooden dust, asthma has been reported in connection with Thuja Standishii. But currently, reports that pneumoconiosis caused by saw dust have not yet been reported. In animal experiment-s, Sano[2] and Abe[3] reported that intratracheal infusion of Japanese cedar dust occurred in collagen fibers in the nodules due to saw dust. They[2,3] reported that either organic dust or inorganic dust allowed various pneumoconiosis to occur corresponding to the increase in volume of inhaled dust and the prolongation of stagnating dust.

In this respect, it is believed that saw dust, if it is not dissolved, is responsible for producing pneumoconiosis. In our experimental results mentioned above, lungs of rats exposed to paulownia dust formed foreign body granuloma of giant cells. Even 12 months later, fibrosis around foreign body granuloma was slight. Most instances result in inflammation mainly centering near the bronchus. It is supposed that dust and these lesions accompanying dust may cause pulmonary fibrosis. But this fibrosis found in nodules is less than that of silicosis. It is believed that insoluble paulownia dust as a foreign body will certainly cause pneumoconiosis. The results of this experiment are led by the method that intra-tracheal infusion of solution suspending dust is infused into the lungs of rats. It is difficult to relate this experimental result directly to human pneumoconiosis. But it is supposed that if either inhalation or infusion dust reaches bronchiole or terminal bronchiole, it will become suspended and mixed with secretion. Finally ,it is supposed that the dust will transpose into the same state either by inhalation or infusion, and that both inhaled and infused dusts are similar[13]. These experimental results provide evidence that abnormal shadows such as pneumoconiosis seen in people employed in furniture and wood products businesses have been caused by saw dust[14].

In order to diagnose pneumoconiosis, three points are necessary[15].

1. Inhalation of dust in one's occupational history.

2. Evidence of clinical symptoms which have been attributed to inhaling dust.

3. Special x-ray findings which show the inhalation of various dust particles. In this respect, workers who had undergone medical examination have occupational histories of inhaling saw dust over a long period of time. Several workers complained of subjective symptoms. The chest x-rays of several people showed linear opacities and small opacities. Those who showed abnormal findings in chest x-rays were mostly over 40 years of age and had been employed longer than 20 years. Pneumoconiosis caused by wooden dust has neither been reported in clinical cases nor in autopsies. Based on our experimental results, we believe that the clinical case report mentioned above is lung lesions caused by inhaling saw dust over a long period of time. This pulmonary fibrosis was caused by saw dust, namely, pneumoconiosis. Though no histological evidence is available, we believe that the diagnosis of pulmonary fibrosis, that is, pneumoconiosis, is made on account of radiographic evidence and the extended exposure to a dusty atmosphere.

Conclusion.

Those who were older than 40 years and had worked longer than 20 years inhaling saw dust, mainly showed linear opacities and small opacities such as pneumoconiosis in chest x-rays taken during complete physical examinations. Moreover, workers employed in the furniture and fixture business had more abnormal findings in their chest x-rays than workers employed in the lumber and wood products industries, although the latter produces more dust. Intra-tracheal infusion of suspending paulownia dust caused foreign body granuloma in the lungs of rats. These foreign bodies consisted of giant foreign cells with fibrosis. Nodules caused fibrosis relating to the inflammation and other minor reactions. Judging from the epidermiological investigation and animal experiment, we consider that saw dust is likely to cause pneumoconiosis.

References

1) Ito, K.: Allergic disorders of upper respiratory tract by sawdust of Thuja Standishi (in Japanese). *J. Sci. Lab.*, 39 : 27-38, 1963.

2) Sano, T.: Pathology and pathogenesis of organic dust pneumoconiosis (in Japanese). *J. Sci. Lab.*, 43 : 3-18, 1967.

3) Abe, A and Ishikawa, T.: Studies on pneumoconiosis caused by organic dust (in Japanese). *J. Sci. Lab.*, 43 : 19-41, 1967.

4) King, E.J., Mohanty, G.P., Harrison, C.V. and Nagelschmidt, G.: The action of flint of variable size injected at constant weight and constant surface into the lungs of rats. *J. Industr. Med.*, 10 : 76-92, 1953.

5) Gardner, L.U.: Studies on experimental pneumoconiosis Ⅷ. Inhalation of quartz dust. *J. Industr. Hyg.*, 14 : 18-38, 1932.

6) Sugiyama, S.: Experimental studies of tissue reaction produced by dust (in Japanese). *Acta Med.*, 27 : 52-82, 1957.

7) Asako, Y.: An experimental study on pneumoconiosis (in Japanese). *Ochanomizu Med. J.*, 7 : 1890-1912, 1959.

8) Sano, T.: Pulmonary diseases caused by dust (in Japanese). *J. Sci. Lab.*, 29 : 4-14, 1961.

9) Iwai, K.: [Introduction of suberose] (in Japanese). *Jpn. J. Chest Dis.*, 20 : 599-602, 1961.

10) Frank, R.C.: Farmer's lung-a form of pneumoconiosis due to organic dust. *Am. J. Roentg.*, 79 : 189-215, 1960.

11) Sera, Y., Konishiike, J. and Sano, T.: An autopsy case of pneumoconiosis due to organic substance dust (Josstick Lung) (in Japanese). *J. Sci. Lab.*, 40 : 547-555, 1964.

12) Bugyi, B. und Molnár, L.: Über Lungenveränderungen bei der Verarbeitung von Reisstroh. *Beitr. Silikose-forsch.*, 51 : 37-48, 1958.

13) Fujii, T.: Pneumoconiosis caused by Sendo dust among rush-mat workers (in Japanese). *Nipp. Act. Radiol.*, 30 : 266-288, 1970.

14) Okita, I.: Pneumoconiosis caused by wooden dust among workers of lumber and wood products (in Japanese). *Yamaguchi Med. J.*, 24 : 255-268, 1975.

15) Hohrai, Z.: [Diagnosis of pneumoconiosis] (in Japanese). *J. Chest Dis.* 5 : 1432-1441, 1961.