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Epidemiological Study of Pneumoconiosis in the Italian Poly(vinyl chloride) Industry

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Among 1216 workers employed in a poly(vinyl chloride) production factory, 20 cases of pneumoconiosis were found. None of these workers had had previous exposure to organic or inorganic dusts; 731 had been exposed to PVC dust (employed in drying, sacking and blending of polymer) and 485 had been exposed to monomer alone. Chest x-ray films were read by two independent physicians utilizing the ILO/UC Pneumoconiosis Classification, 1971. X-ray abnormalities were characterized by limited profusion, irregular type and low gravity; in a small percentage of cases these were associated with slight restrictive respiratory function impairments. All 20 workers with PVC-induced pneumoconiosis had been exposed to high PVC dust pollution for at least five years. Mild nonspecific alterations (profusion of 0/1 class) were found both in the group exposed to PVC dust and in the group exposed to VCM alone. Such changes (observed in 388 cases, 31.9% of the whole population), are related mainly to age and smoking habits, and the role of exposure is minor.

We examined the working population of plants producing poly(vinyl chloride) (PVC) in Porto Marghera, Italy; 1216 workers had no previous dust exposure. Of these 731 were exposed to PVC dust polymer alone while 485 were exposed to vinyl chloride monomer (VCM). In the drying, sacking and blending departments, PVC dust concentrations were over 10 mg/m^3 of total dust in about 60% of the samples, whereas in the polymerization departments no concentration over 10 mg/m^3 was found. In the samples taken, particles with diameters of 1 μm to 6 μm constituted 4.5 to 30.9% of total dust weight.

All the workers had chest x-rays according to ILO standards and a spirographic examination. Chest x-ray films were read by two independent physicians utilizing the ILO/UC pneumoconiosis classification. For statistical analysis, a consensus reading was used.

Table 1 shows that there are no significant differences in age and smoking habits, but the exposure duration is higher in the workers not exposed to dust.

Table 1.

Group	No. of subjects	% of smokers	Age, yr (mean ± SD)	Exposure, yr (mean ± SD)
Exposed to PVC dust	731	73.9	37.7 ± 8.8	6.1 ± 4.0
Not exposed to PVC dust	485	68.7	35.7 ± 8.5	8.6 ± 4.5

Table 2.

PVC dust exposure, yr	Age distribution of cases[a]			
	≤ 30 yr	31–40 yr	41–50 yr	> 50 yr
< 5	–	–	–	–
5–10	–	2(2.4)	5(8.3)	1(5.9)
> 10	–	2(3.8)	8(9.0)	2(9.5)

[a]Values in parentheses are percentages of subjects with PVC pneumoconiosis in each class of age and exposure.

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In 20 subjects we found chest x-ray changes of at least class 1 according to the ILO/UC classification. The mean age of this group was 44.9 ± 5.2 years and the mean length of exposure was 11.6 ± 5.4 years. Sixteen subjects (80%) were either smokers or ex-smokers.

Table 2 summarizes the distribution of cases in relation to age and length of exposure. In all age groups there is an increase of disease prevalence associated with the increasing length of exposure.

In the case of the x-ray changes, 16 subjects had class 1/0 profusion, 2 cases class 1/1, 1 case class 2/1, and 1 case class 2/2. Irregular opacities were prevalent: 10 were type s, three were type t, six were type p and one was type r. They were diffused, mainly over median lobe areas.

A chest x-ray of a worker exposed to PVC dust (Fig. 1) for 15 years shows a gross reticular pattern: profusion is class 2/2, type t. In the right hemithorax of the same subject (Fig. 2) there is a mottled reticular pattern. Figure 3 is an enhanced view of the right hemithorax showing the mottled reticular pattern more clearly.

Another case of PVC induced pneumoconiosis can be seen in Figure 4. The worker was exposed to PVC dust for 20 years. A fine, dense reticular micronodulation is evidenced. Profusion is class 2/1, type p-s. Figure 5 shows the right hemithorax of the same subject; pin-point opacities can be seen.

In spite of age and the considerable exposure, the majority of cases were in a low profusion category, indicating the slow evolution of the disease. All had worked in high air-borne dust level environments (mostly drying and sacking) for at least five years. None of the 20 subjects was in a group unexposed to dust and none had experienced previous occupational exposure to organic or inorganic dusts. We considered the alterations to be PVC pneumoconiosis.

In 388 subjects (31.9%) we found slight chest x-ray alterations consisting of linear or irregular vanishing opacities or both, classified as class 0/1; the remaining 808 subjects were class 0/0.

Table 3 reports the total population distribution excluding 20 subjects with PVC pneumoconiosis. Results are presented in a two-way table: each entry reports the number of observations. Samples are classified according to age and PVC dust exposure: PVC + represents presence, and PVC – absence of PVC; x-ray + indicates the group with

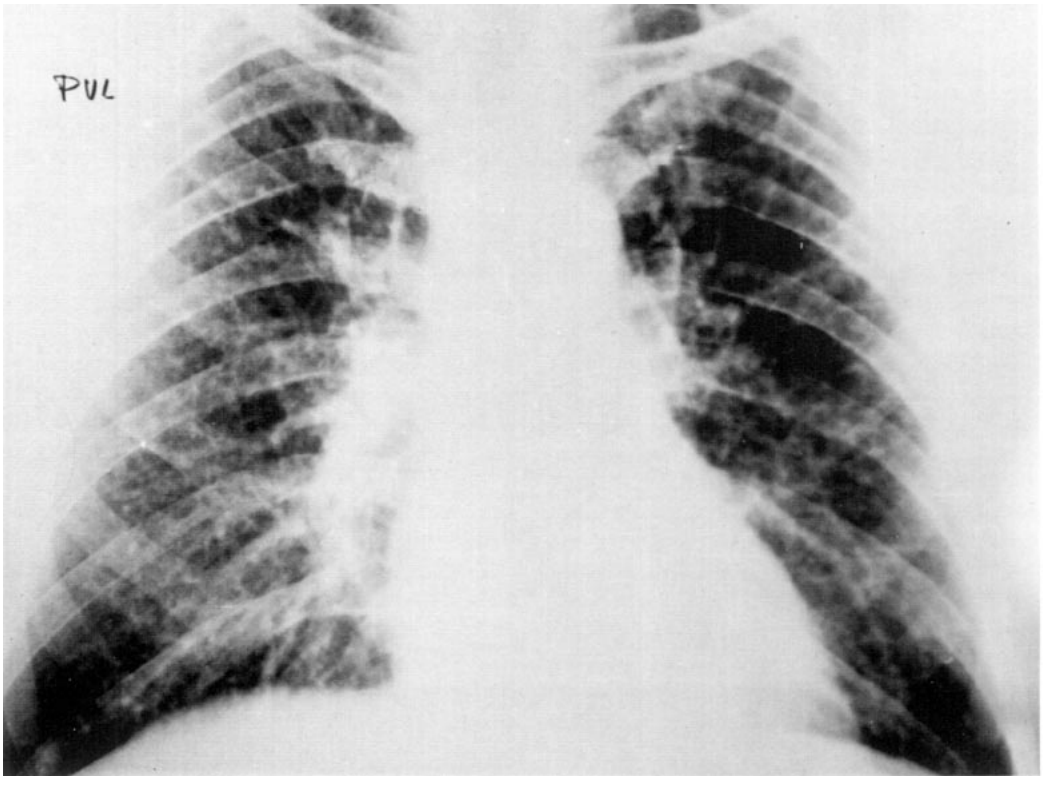

FIGURE 1. Chest x-ray of a worker exposed to PVC dust for 15 years. Profusion class 2/2; type t.

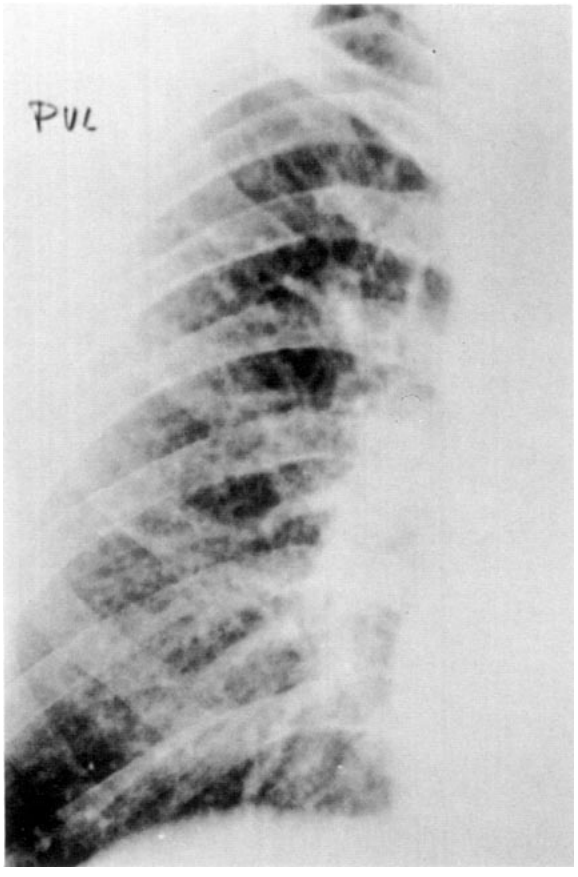

FIGURE 2. Right hemithorax of the same subject as in Fig. 1.

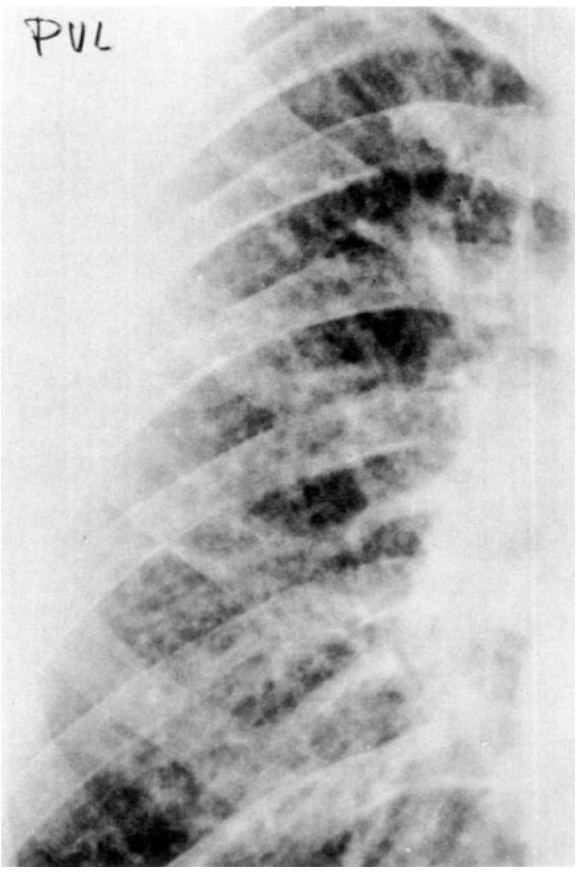

FIGURE 3. Enhanced view of the right hemithorax of Fig. 2, showing the mottled reticular pattern.

class 0/1 profusion. To assess the influence of both risk indicators we performed a two-way analysis of variance for proportions.

Table 4 shows that both age and exposure were significant factors influencing x-ray abnormalities. The square of a multiple partial association coefficient for qualitative data was calculated to measure the degree of association between the dependent variable (x-ray changes) and each of the two predictor variables (age and exposure). Age and exposure are alternately held constant. Age is a most important factor. When exposure is held constant, 33.2% of the x-ray changes are shown to depend upon age, when age is held constant, 6.5% are shown to depend upon exposure.

Table 5 summarizes the distribution of the cases according to smoking habits in workers exposed and not exposed to PVC dust.

Table 6 shows that chest abnormalities are significantly influenced by both risk indicators.

Table 3.

	Age distribution of subjects			
	≤ 30 yr	31–40 yr	41–50 yr	> 50 yr
PVC +				
X-ray +	20	78	123	35
X-ray –	133	197	108	17
PVC –				
X-ray +	17	45	55	15
X-ray –	119	158	67	9

Table 4.

Source of variation	Degrees of freedom	Sum of squares	Mean squares	F
Age	3	30.9713	10.3238	53.81[a]
PVC dust exposure	1	0.7843	0.7843	4.09[b]
Interaction	3	0.9882	0.3294	1.72
Error	1188	227.9403	0.1919	

[a] $p < 0.01$.
[b] $p < 0.05$.

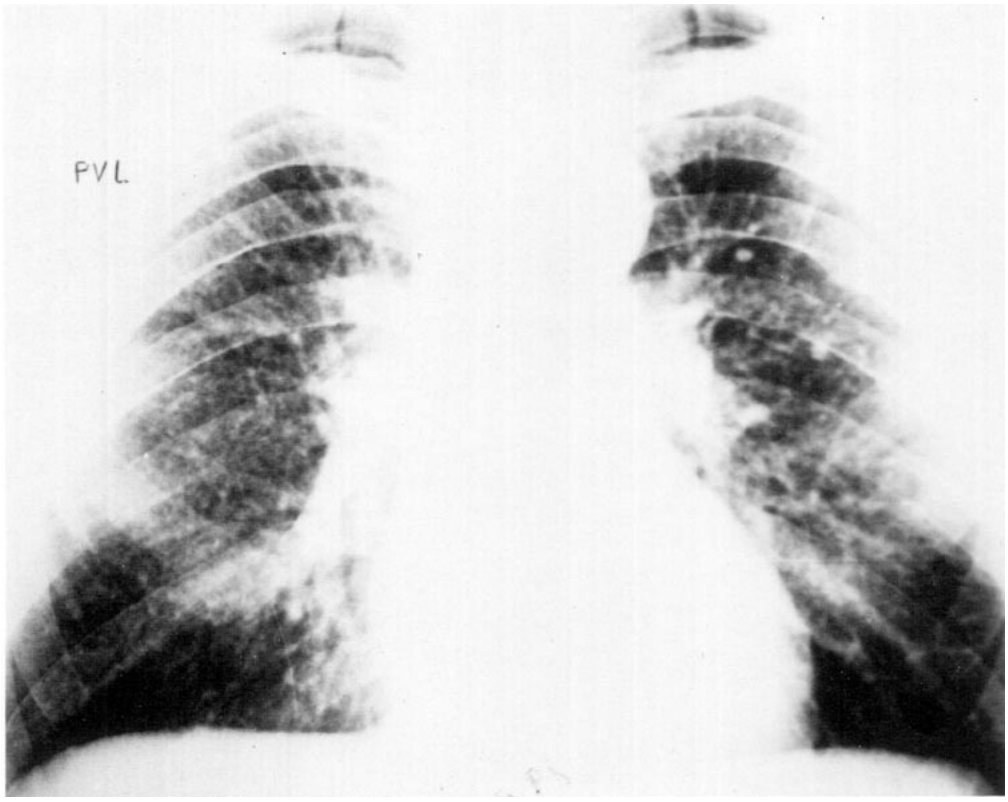

FIGURE 4. Chest x-ray of a worker exposed to PVC dust for 20 years. Profusion class 2/1; type p-s.

Table 5.

	Nonsmokers	Smokers
PVC +		
X-ray +	40	216
X-ray –	147	308
PVC –		
X-ray +	25	107
X-ray –	127	226

Table 6.

Source of Variation	Degrees of freedom	Sum of squares	Mean squares	F
Age	1	7.8850	7.8850	37.31[a]
PVC dust exposure	1	1.7847	1.7847	8.44[a]
Interaction	1	0.1023	0.1023	1.0
Error	1192	251.9125	0.2113	

[a] $p < 0.01$.

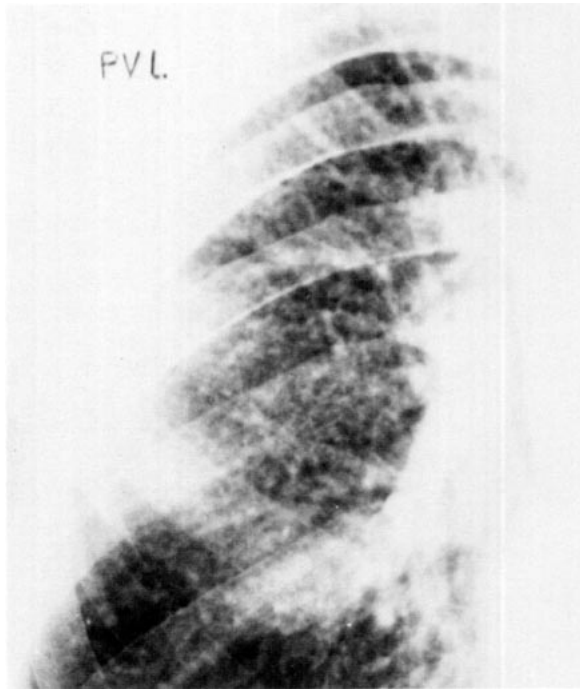

FIGURE 5. Right hemithorax of the same subject as in Fig. 4.

When exposure is held constant, habitual smoking is responsible for 17.1% of the changes. When smoking habits are held constant, exposure to PVC dust is shown to be responsible for 8.9% of the x-ray abnormalities.

Our epidemiological study confirms experimental and pathological data already reported regarding the effects of PVC dust. Lung changes are directly related to PVC dust exposure, whereas VCM exposure alone fails to cause these changes. Therefore, we believe that pulmonary changes are not pathogenically similar to other vinyl chloride induced abnormalities, that is fibrosis of the liver, scleroderma-like skin changes, and peripheral vascular damage.

In our study there was only a 1.6% prevalence of pneumoconiosis in the total population, but in the workers exposed to effective risk of pneumoconiosis (731 subjects) the prevalence rose to 2.7%.

Apart from 20 cases of pneumoconiosis, mild nonspecific alterations (profusion of 0/1 class) were found in the group exposed to PVC dust and in the group exposed to VCM alone. Such changes are related mainly to age and smoking habits, and the role of exposure is minor.