Pneumoconiosis in Carbon Black Workers

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Abstract : Pneumoconiosis is a chronic inflammatory and fibrotic lung disease caused by the inhalation of many substances in various forms, of which carbon black is one of them. Workers exposure may occur during production, collection and handling of the substance as well as in many other ways. Most reports describe the effect of carbon black on the lung of human's certain changes which are typical for pneumoconiosis and changes that are suspected to become pneumoconiosis in the future. There were many studies describing carbon black pneumoconiosis from 1951 to 1994. This disease, therefore, has been considered as an occupational disease of workers exposed to carbon black in its production and usage.

Key words : carbon black, pneumoconiosis, suspected pneumoconiosis.

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Pneumoconiosis is a chronic inflammatory and fibrotic lung disease caused by the inhalation of many substances in various forms. This overview will present a general hypothesis drawn from available clinical and laboratory evidence to explain the pathogenetic mechanism that produces pneumoconiosis [1].

A worker's exposure to carbon black may occur during production, collection and handling of the substance, particularly during pelletisation, screening, packaging, stacking, loading and unloading. Exposure may also occur when cleaning equipment, when leaks develop in the conveyor system and from spills [2].

Most reports describing the effects of carbon black on humans have dealt with pulmonary effects. The environmental concentrations at which people are exposed and those concentrations that are available for total dust rather than for carbon black alone have seldom been reported. Carbon blacks are not soluble in water or in the fluid lining the respiratory tract, thus inhaled particles could damage the lung causing pneumoconiosis in a manner analogous to certain mineral dusts such as free crystalline forms of silica [3]. Pneumo-

coniosis may also be caused by pure graphite without any admixture of silica or silicates [2]. On the other hand, this disease can be caused in workers by dust in which silica constitutes only a trace [4]. Coal and carbon themselves are not cytotoxic and they behave as inert dusts causing only mild reticulin proliferation in the lungs of experimental animals. A small quantity of quartz, though muted in its effects by coal and other dusts, may influence the pathogenesis of coal pneumoconiosis [5].

Apart from certain changes which are typical for pneumoconiosis, the radiological picture of the lung dose frequently shows some irregular small opacities and deformation and reinforcement of the pulmonary outline and macroreticular fibrosis. People with these changes are likely to contract pneumoconiosis in the future [6].

As mentioned above, many health studies have been conducted in work forces occupationally exposed to carbon black. It is the ability of the fine particles of carbon black to penetrate to the deeper regions of the lung and perhaps to exert a pathogenic effect on the pulmonary function through diseases such as pneumoconiosis.

The first paper describing carbon black pneumoconiosis published in 1951 by Gartner and Braus, investigated carbon black workers and coal miners in the Ruhr area and in South Wales and identified 31 cases of radiographic changes analogous to pneumoconiosis [7]. The association between exposure to carbon black and pneumoconiosis was confirmed in 1952 by Töpner [8] and in 1956 and 1957 by Meiklejohn [9, 4]. They investigated a few cases of simple pneumoconiosis. In 1961 Miller and Ramsden reported a case of pneumoconiosis in a man exposed to carbon black in a rubber factory [10]. Publications in 1960 [11], 1961 [12], 1968 [13], 1969 [14, 15], 1970 [16], 1973 [17], 1975 [18–20], 1976 [21], and 1980 [22], also reported many cases of pneumoconiosis and suspected pneumoconiosis in carbon black workers in many countries.

The most recent publication in 1985, by Beck and his coworkers presented lung findings among 13 workers employed in a carbon black plant. X-rays showed disseminated small irregular and large shadows with slow progress. In conclusion, the authors wrote that according to the X-rays and histological findings, the lung disease could be estimated as pneumoconiosis [23]. In the same year, Thumer and Beck summarised the relationship between carbon black dust in the workplace and findings of pneumoconiosis and suspected pneumoconiosis in employees [24]. Also Kandt reported cases of this disease in workers exposed to carbon black dust for more than 20 years [25]. In 1986 Crosbie reported on the respiratory health of 3027 carbon black workers employed in 19 plants (18 in Western Europe and 1 in the United States). There were 6 cases of simple type pneumoconiosis all of whom had more than 10 years of dust exposure [26].

One of the most complete studies on the population of carbon black workers was done by a team from the Institute of Occupational Health, University of Birmingham [27, 28]. Gardiner and his coworkers found prevalences of small opacities (they didn't call them pneumoconiosis). They suggested that the effect of carbon black is associated with some increased prevalence of respiratory symptoms, small decrements of lung function and the accumulation of dust in the lung. The radiographic changes probably indicate parenchymal deposition of carbon black dust rather than pathological change. However, this study will be continued in the near future.

At the end of 1993 I found one case of pneumoconiosis (2p) in a female employed for 26 years in a factory with mixed ventilation disturbance [29]. In 1995 I found another case (1q) in a male employed for 37 years in a workplace with obstructive (obturative) ventilation disturbance. From 1994 to 1995 I found 6 cases of suspected pneumoconiosis. The data of my studies are shown in Table 1. The study of carbon black workers in Poland also will be continued.

The data from epidemiological studies (1951–1995) are shown in Table 2.

Table 1. Pneumoconiosis and suspected pneumoconiosis in Poland (1995)

No.	Diagnosis	Chest x-ray (*)	Sex	Duration (years)	% of the normal FVC	range FEV ₁	FEV ₁ %VC	Smoking history
1	P	2p	F	25	78	68	70	NS
2	P	1q	M	37	86	75	63	S
3	SP	2t	M	41	81	96	83	S
4	SP	1t	F	23	84	90	81	S
5	SP	1t	M	8	88	101	85	NS
6	SP	2s	F	19	61	70	84	NS
7	SP	1t	F	29	95	111	85	NS
8	SP	1t	F	25	73	83	77	NS

P-pneumoconiosis, SP-suspected pneumocniosis

M-male, F-female

S-smoker, NS-nonsnoker

(*)-International Classification of radiographs for pneumoconiosis

Conclusions

For proper assessment of the toxicity of carbon black and its potential hazard to the working population, further animal and human studies are needed.

Pneumoconiosis has to be considered as an occupational disease of workers exposed to carbon black in its production and usage.

Table 2. Pneumoconiosis in carbon black workers

Ref.	Year	Workers (all)			Exposure	Effect	No. of Cases	
No.		No.	Age	Sex	Concentration (mg/m ³)	Duration (years)	Pneumoconiosis	Suspected pneumoconiosis
9	1951						31	
26	1952					> 10	17	
17	1956	44				30–50		
18	1957	42					8	
12	1960				9.3–972.0			
19	1961					> 21	1	
11	1961	89				> 5	4	15
14	1968	52				> 10	7	
6	1969	72					10	5
22	1969					> 21	2	
23	1970	52			> 10.0	> 10	9	
13	1973	643			> 10.0		7	18
27	1975	357				> 16	17	
28	1975	35			10.0– 15.0		6	
29	1975	397				> 6	38	
				*				
3	1976	143				19.1 [†]	29	
2	1980	32			> 10.0	> 19	7	
1	1985				5– 50.0		13	
25	1985				1.1–108.2		7	13
10	1985	129				> 20	3	
4	1986	3027		*		> 10	6	
7	1992				0.01–35.44**			
8	1993	1084	16–65	*			***	***
24	1994	118	26–65	*	< 81.26	26	1	
	1995	152	26–65	*	< 81.26	> 8	2	4

*Male and female

**Total inhalable dust

***Prevalences of small opacities of categories : 1/0(56), 1/1(48), 2/1(2), 2/2(1)

[†]Mean

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カーボンブラック労働者における塵肺症

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要 旨： 種々の物質を吸入することによって起こる塵肺症は肺の慢性炎症と線維化を伴い，カーボンブラックもその原因の一つとなる．労働者への曝露は製造過程の種々の段階で起り得る．ヒトの肺に対するカーボンブラックの影響については，多くの報告が1951年から1994年にかけてもなされており，典型的な塵肺症あるいは将来そうなるであろう病変について記述しており，カーボンブラックの製造や利用にかかわり曝露を受けた労働者の塵肺症は当然職業性疾患とみなすべきである．

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