

The effect of wood dust on the nasal cavity and paranasal sinuses

E. Güney, Y. Tanyeri, B. Kandemir and Ş. Yalçın, Samsun, Turkey

SUMMARY

The effect of wood dust on the mucous lining of the nose and paranasal sinuses is investigated experimentally in 20 rats. Our short-term study revealed that wood dust is a chronic irritative agent for these tissues.

The relationships between cancers, especially adenocarcinoma of the nose and paranasal sinuses, and occupational exposure to wood dust were first mentioned by Acheson et al. (1968). Since then this association has been confirmed by many reports from various countries (Andersen et al., 1977; Black et al., 1974; Brinton et al., 1975). Some of these investigations have indicated that the nasal mucociliary function is impaired and mucostasis develops in woodworkers (Andersen et al., 1977; Black et al., 1974). It was suggested that the "mucostatic factor" in wood dust is of importance in the development of nasal adenocarcinoma in furniture workers because of the persistent irritation by wood dust in the nasal cavity. In this study we wanted to investigate the effect of wood dust on the mucosal lining of the nasal cavities and paranasal sinuses of rats.

MATERIAL AND METHOD

20 rats (240-300 g) of the Swiss Albino species were used in this experiment. Four of them were excluded from the research for various reasons and the experiment was carried out on 16 rats. We made use of the dusty environment of the chairmaker industry. All animals were exposed to 7-10 µm sized wood dust particles for 12 hours daily. This procedure was performed for four months and for the same period of time. A control group of 10 rats of the same size was followed up in their normal living conditions. After the four months, all animals were sacrificed by intravenous high dose penthotal and total autopsy was performed. Tissue samples were taken from the mucous membranes of the nose and paranasal sinuses, nasopharynx, oropharynx, larynx, trachea and lung and fixed with 10% buffered neutral formalin. After the routine paraffine processing 7 µm sections were done with a microtome and stained with H+E and alcian blue. All sections

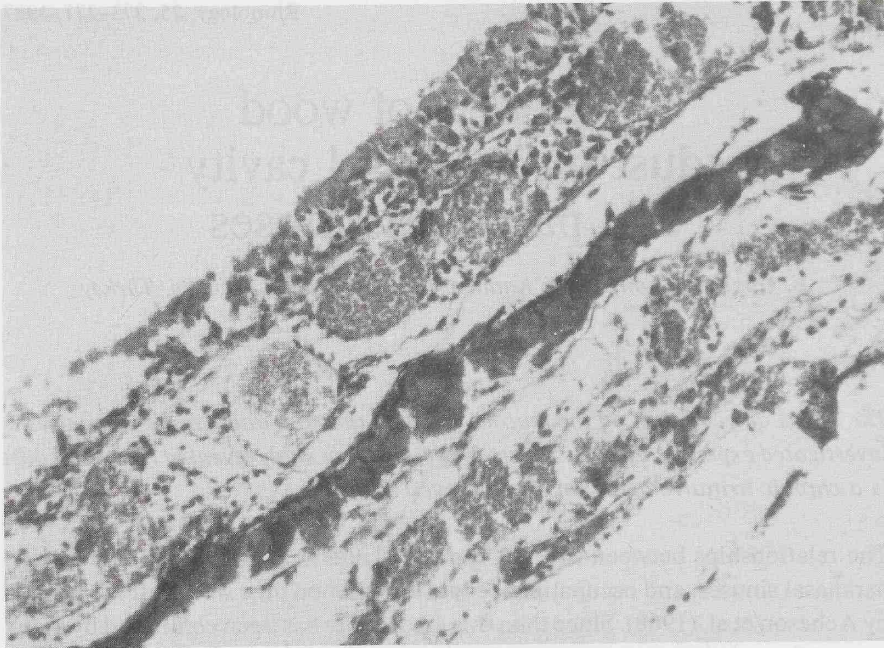


Figure 1. Ciliary loss, submucosal congestion and edema (H + E x 125).

were examined under the light microscope and the positive findings were photographed.

RESULTS

Macroscopic and microscopic examination of the tissue samples taken from the control animals revealed totally normal mucous membranes.

Macroscopically, we found no abnormality other than congestions and edema in all the experimental animals. Microscopically we found in 40% of the specimens that the surface epithelium of the nasal cavity and paranasal sinuses were occasionally desquamated and approximately 50% of the animals lost their cilia. Most of the superficial epithelium contained pinocytic vesicles in their cytoplasm. Mucosal and submucosal congestion were seen in 87.5% of the animals and 75% of the specimens showed diffuse edema separating the mucosal glands (Figure 1). The mucinous component of the seromucous glands of the lamina propria was increased in relation to the serous component and 50% of the glands were totally transformed into mucinous glands (Figure 2). Most of the specimens were infiltrated with inflammatory cells composed of plasmocytes, lymphocytes and macrophages (Figure 3). In one of the specimens there was diffuse submucosal eosinophilic homogenous material deposition.

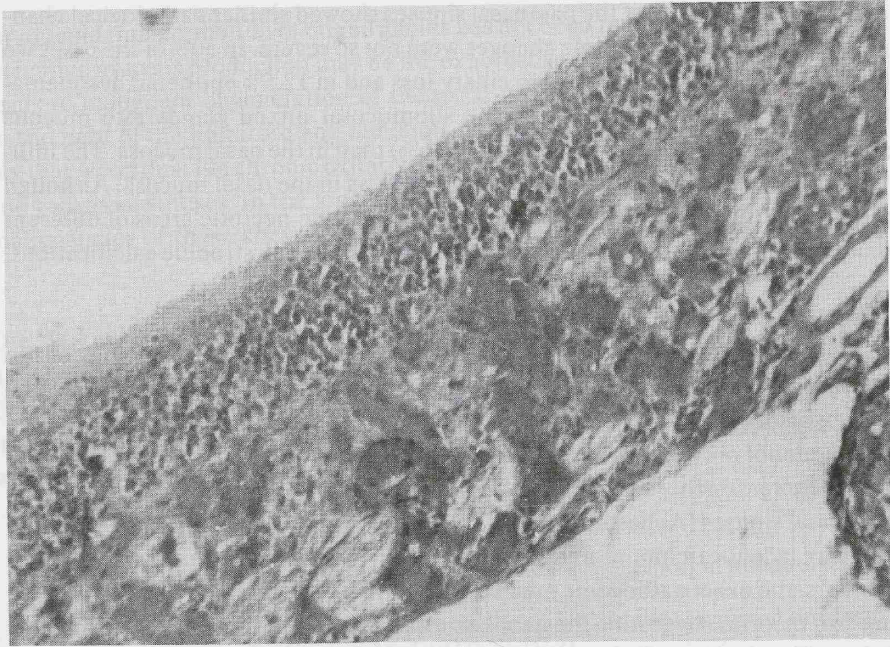


Figure 2. Increased mucinous component against the serous component (H + E x 250).

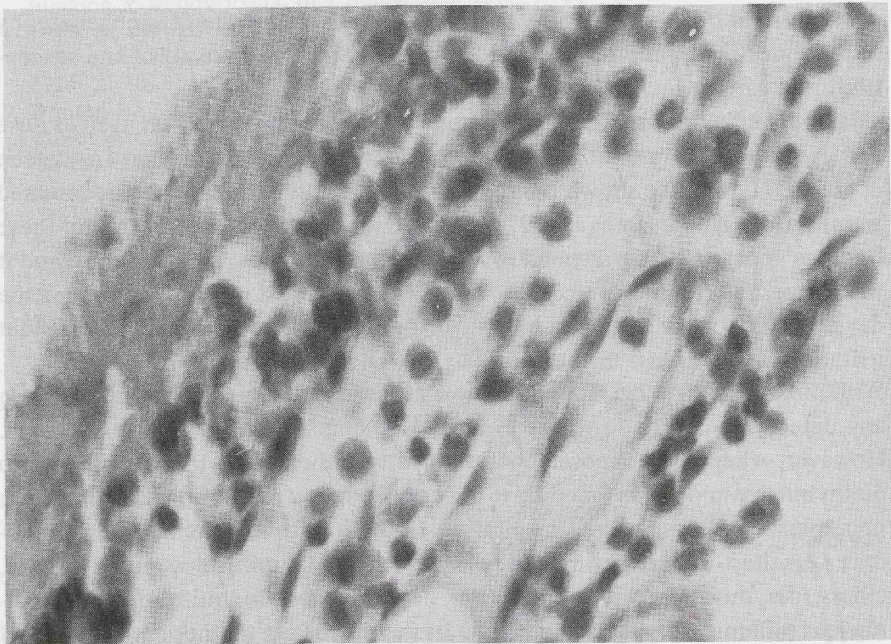


Figure 3. Mixed inflammatory infiltration of lamina propria (H + E x 500).

Although the mucosa of the paranasal sinuses showed similar pathological changes as the nasal mucosa, these changes were not so severe. In 50% of the cases we found submucosal edema, in 50% ciliary loss and in 12.5% epithelial desquamation. However, transformation of the submucosal mixed glands into mucous glands was found at a much higher ratio (56%) than in the nasal mucosa. The infiltration of inflammatory cells was not so severe as in the nasal mucosa. Although pinocytotic vesicles were not seen, there were multiple necrotic areas of different diameters in the mucosa and one of these areas showed dystrophic calcifications.

DISCUSSION

In 1968 Acheson et al. reported that the incidence of adenocarcinomas of the nasal cavity and paranasal sinuses was 1000 times more frequent among the woodworkers than among the normal population. It was also proved that wood dust does not only increase the incidence of the adenocarcinoma but also significantly increases the occurrence of other types of carcinomas of the nose and paranasal sinuses (Acheson et al., 1972). Though wood dust is very well known to increase adenocarcinoma and other types of malignancies among the woodworkers, the exact pathogenic mechanisms have not been understood yet. There are two theories suggesting the neoplastic mechanism of wood dust. The first one is based on the idea of inhalation of carcinogenic substances such as tannins and aldehydes during wood processing. The second one introduces the idea that wood dust particles bigger than 5 μm could be responsible for the carcinogenesis by interference of the normal mucociliary action and the increase of the susceptibility to carcinogens (Elwood, 1981; Newbill, 1983).

The initial effect of wood dust on the mucous membrane manifests itself as nose and eye irritation, nasal obstruction, prolonged colds and headaches (Andersen et al., 1977). It is also shown that ciliary activity of the lining mucosa decreased significantly (Andersen et al., 1977; Black et al., 1974). The occurrence of these findings appeared to correspond to the amount of the dust concentration and it was suggested that the "mucostatic factor" in wood dust is of importance in the development of nasal carcinomas in furniture workers because of persistent irritation of wood dust in the nasal cavity (Andersen et al., 1977).

Wilhelmsson et al. (1985) found that low doses of wood dust alone did not cause any detectable nasal and tracheal mucosal change in Syrian golden hamsters. However, when they exposed the animals to either diethylnitrosamine or to diethylnitrosamine plus wood dust they observed several lesions ranging from hyperplasia and dysplasia of respiratory epithelium to papillomas and carcinomas. Our short-term experimental research revealed diminishing ciliary activity, ciliary loss, mucoid gland proliferation, heavy congestion, infiltration of mononuclear inflammatory cells and edema in the mucosa of the nose and paranasal sinuses which are identical to the initial findings mentioned in the literature. So

far mucoid transformation of mixed glands has not been reported. Ciliary loss and excessive mucous production may be the exact cause of mucostasis which further leads to malignant degeneration of the nasal mucosa.

In the light of the literature and our short-term experimental research we could say that wood dust is a chronic irritative agent for mucous membranes of the nose and paranasal sinuses. In the long run this chronic irritation may be the cause of malignancies of these sites.

ZUSAMMENFASSUNG

Die Wirkungen vom Holzpulver an die Nasen- und Nasennebenhöhlenschleimhaut wurden experimentell bei 20 Mäusen untersucht. Nach der histologischen Untersuchung der Schleimhäuten wurde gezeigt, dass Holzpulver ein chronisch-irritatives Agent für diese Gewebe ist. Die zusammenhängende Literature wurde weitgehend auseinandergesetzt.

REFERENCES

1. Acheson ED, Cowdell RH, Hadfield E, Macbeth RG. Nasal cancer in woodworkers in the furniture industry. *Br Med J* 1968; 2:587-596.
2. Acheson ED, Cowdell RH, Rang E. Adenocarcinoma of the nasal cavity and sinuses in England and Wales. *Br J Indust Med* 1972; 29:21-31.
3. Andersen HC, Andersen I, Solgaard J. Nasal cancers, symptoms and upper airway function in woodworkers. *Br J Indust Med* 1977; 34:201-207.
4. Black A, Evans JC, Hadfield E, Macbeth RG, Morgan A, Walsh M. Impairment of nasal mucociliary clearance in woodworkers in the furniture industry. *Br J Indust Med* 1974; 31:10-17.
5. Brinton LA, Blot WJ, Stone BJ, Fraumeni JF. A death certificate analysis of nasal cancer among furniture workers in North Carolina. *Cancer Res* 1977; 37:3473-3474.
6. Cecchi F, Buiatti E, Kriebel D, Nastasi L, Santucci M. Adenocarcinoma of the nose and paranasal sinuses in shoemakers and woodworkers in the province of Florence, Italy, 1963. *Br J Indust Med* 1963; 37:222-225.
7. Elwood JM. Wood exposure and smoking: Association with cancer of the nasal cavity and paranasal sinuses in British Columbia. *CMA J* 1981; 124:1573-1577.
8. Ironside P, Matthews J. Adenocarcinoma of the nose and paranasal sinuses in woodworkers in the state of Victoria, Australia. *Cancer* 1975; 36:1115-1121.
9. Newbill ET. Resident's page: Pathologic quiz Case 2. *Arch Otolaryngol* 1983; 109:134-135.
10. Wilhelmsson B, Lundh B, Drettner B. Effects of wood dust exposure and diethylnitrosamine in an animal experimental system. *Rhinology* 1985; 23:114-117.

Dr. Yücel Tanyeri
Dept. of Otorhinolaryngology
Medical School of 19 Mayıs University
Samsun
Turkey