# Epidemiology of occupational nasal cancer and other occupational nasal problems caused by wood dust

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An association between adenocarcinoma of the nose and paranasal sinuses and occupational exposure to dust from hard woods has been recognized since the middle of the nineteen-sixties when it was found that this disease was about 500 times more common among wood furniture workers than among the general male population in the same area (Acheson et al., 1968; Macbeth, 1965). Epidemiological studies in many other countries, including Sweden, have since revealed the similar relationship (Andersen, et al., 1977; Brinton et al., 1977; Debois, 1969; Engzell et al., 1978; Gignoux and Bernard, 1969; Loebe and Ehrhardt, 1976). The risk of developing cancer seems to be highest for turners, machinists and sanders (Acheson, 1976). On the other hand, carpenters and joiners do not show an increased incidence. Smoking and snuff-taking have been discussed as contributory factors, but most studies have not revealed any correlation between these habits and the incidence of nasal adenocarcinoma. The incidence of other nasal cancers is also significantly increased among furniture industry workers but not at all to the same degree as that of adenocarcinoma (Acheson, 1976). Acheson (1976), who has carried out most of the epidemiological research concerning wood dust and adenocarcinoma of the nose, found that adenocarcinoma comprised 82% of all malignant nasal tumours in wood workers, compared with 8% in persons who had not been exposed to wood dust. Until quite recently it was considered that hard types of wood, especially beech, and probably oak, were the types particularly associated with the disease, and Acheson (1976) found that fine wood dust was the only common factor underlying the adenocarcinoma cases among the wood workers whom he investigated. He considered that the effects of polishes, varnishes, preservatices, and so on, could be essentially excluded. In a recently published Scandinavian multicentre study performed by Hernberg et al. (1983), it was found that exposure to hard wood dust was associated with adenocarcinoma and that exposure to soft wood was related to epidermoid and anaplastic carcinoma, but the latter findings did not reach statistical significance in that study.

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#### Nasal cancer and problems by wood dust

In a recent Norwegian epidemiological study, not yet published, performed by Voss et al. (1985), a strong association between cancer of the sino-nasal cavities and exposure to soft wood was found. Squamous cell carcinoma was the pre-dominant type of cancer in joiners, carpenters and loggers while non Hodgkin lymphomas appeared to be associated with employment in saw- and planning mill industries. A Norwegian case control study is under preparation.

In England, there has not so far been any significant indication of a declining frequency of wood dust induced adenocarcinoma of the nose and paranasal sinuses according to Acheson (1982).

The average latency period from the commencement of employment to the diagnosis has been about 45 years, ranging from 20 to 70 years (Acheson, 1976; Engzell, 1978; Klintenberg, 1984). In England patients with nasal adenocarcinoma associated with wood dust exposure were 10 years younger, on an average, than those with other kinds of nasal cancers (Acheson, 1976).

Wood dust exposure can not only give rise to nasal cancer but also cause less severe problems in the nose. It has long been recognized that nasal discomfort such as obstruction and discharge is of frequent occurrence among wood furniture workers. An investigation in Swedish wood furniture factories was performed enclosing studies of the environment in industries and of hypersensitivity reactions among the workers (Wilhelmsson et al., 1984).

## CONCLUSION

Nasal discomfort as obstruction and hypersecretion was significantly more common and the incidence of common colds was significantly higher among furniture workers exposed to wood dust than among controls. The air conditioning capacity of the nose was normal in the furniture workers. One half of them had pathologically slow nasal mucociliary clearence. In those workers with a history of nasal occupational hypersensitivity, rhinomanometry indicated a more congested mucosa than in workers without symptoms. Spirometry revealed lower forced vital capacity than expected.

High concentration of moulds, up to  $645\ 000\ \text{spores/m}^2$  air was found in some of the investigated factories. The endotoxin concentration in the air was high in a few occasions, up to  $0.35\ \mu\text{g/m}^2$ . The wood dust concentration was most often below the Swedish threshold limit value which today is  $4\ \text{mg/m}^2$  air. 16% of the 268 wood workers who answered the questionnaire had symptoms compatible with nasal hypersensitivity with connection to there work. 3% of the workers were shown to have a nasal allergy to moulds and 2% to wood dust. Most of them were atopic persons with other allergies and high IgE-values. That means that the other 11% had a nasal hyperreactivity of unknown cause.

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