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PROFILES OF OCCUPATIONAL EXPOSURE IN PATIENTS WITH WOOD DUST-INDUCED NASAL CARCINOMA

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Abstract
Objectives: Regulations to certify nasal carcinoma as occupational disease induced by wood dust vary in the European countries. In Germany, it must be adenocarcinoma and the dust originating from beech or oak trees. In other countries, the disease is reconized as induced by occupation independent of the kind of wood dust or the type of carcinoma. With regard to the harmonization of regulations necessary in Europe we investigate whether these differences are acceptable from the scientific viewpoint. **Materials and Methods:** In a retrospective analysis of a group of 28 patients with wood dust-induced nasal carcinoma in North Rhine-Westphalia, Germany, we studied whether significant differences in exposure, personal risks (smoking) and histological tumor type could justify the current German, Austrian and Luxembourg pattern of occupational disease certification. To quantify cumulative time of exposure we developed a new semiquantitative formula for the calculation of wood dust years (WDY). **Results:** Our results indicate that there are no significant differences between the subgroups, i.e., exposure to hard wood (n = 13) or other woods (n = 15), WDY (13.1 ± 11.3), smoking (n = 17) or nonsmoking, type of work and the diagnosis of nasal squamous cell carcinoma (n = 8) versus nasal adenocarcinoma (n = 20). The review of literature suggests systematic errors in the studies that led to the current German regulations. **Conclusions:** We recommend to amend the German, Austrian and Luxembourg regulations and to recognise nasal carcinomas as occupational disease, regardless of the type of wood dust exposure, and regardless of the tumor histology as also recommended by the International Agency for Research on Cancer (IARC).

Key words:
Nasal carcinoma, Wood dust, Occupational disease

INTRODUCTION

An association between wood dust and malignant nasal tumors has been suspected for centuries [1], but the first research into the etiology was conducted in England and Czechoslovakia as late as the 1960s [2–5]. In 1981, the International Agency for Research on Cancer (IARC)

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recommended that tumors should be recognized as occupational disease [6]. In Germany, special procedures are usually involved before any illness is certified as occupation-induced disease. First of all, a scientific board works out an expertise with a specific recommendation. It includes a scientific discussion. Based on the available data, the board recommended exclusively nasal adenocarcinoma as wood dust-induced malignancies in 1988 [7], although the IARC has long recommended the recognition of other types of wood as well.

Today the requirements for recognizing nasal tumor as an occupational disease vary throughout Europe. The more restrictive German criteria for recognizing wood species and histological cancer type are used in Germany, Luxembourg and Austria [8,9]. Denmark, England, Sweden, Belgium, France, Italy and Switzerland require a proven long-term exposure to any kind of wood dust and recognize nasal carcinoma of any histological type. These different requirements for recognizing an occupational disease are firstly due to a lack of understanding of the precise etiology of the tumor, and secondly due to a lack of data indicating what kind of exposure leads to which type of tumor [10].

Consequently, the same tumor can be recognized as related to occupational exposure in one European country, whilst a few kilometers further across the border it would be refused recognition. The implications for the patient can be significant, including lost support, compensation, and so forth. With data from North Rhine-Westphalia, the main industrial region of Germany, we investigated the differences between the patients suffering from nasal adenocarcinoma (AC) versus squamous cell carcinoma (SCC) and their exposure to hard wood (HW; e.g., oak or beech) versus other wood (OW; e.g., pine or fir tree).

MATERIALS AND METHODS

The analysis included all fully documented cases of nasal carcinoma registered over a 7-year period (1988–1995) in the archive of the National Institute for Occupational Health and Safety of North Rhine-Westphalia (n = 28). In this archive, all cases suspected of any occupational disease are stored. This is independent of the final outcome whether this disease will be certified as occupationally induced or not. We analyzed the group of patients for differences in the following variables: age, gender, occupational history, duration and kind of work, wood dust exposure (type, degree, duration), symptoms at the time of diagnosis, smoking, tumor localization, and histological type.

To assess the degree of wood dust-exposure, which has never been quantified in the case file, we established a semiquantitative model in analogy to those used for the estimation of asbestos exposure or the working level months (WLM) in uranium mining: One wood dust year (WDY) represents one year full-time mechanical work at "natural" ventilation (without special devices). Mechanical processing went into the calculation with a factor of 1.0, as opposed to manual work with a factor of 0.5 (because of less production of dust). Moderate ventilation, e.g., working outdoors, or with very good natural ventilation, was given a factor of 0.5, when optimal devices for ventilation were given a factor of 0.25. The cumulative WDY were calculated by a formula devised by us in accordance with the validated formula for asbestos fibre years (formula 1) [11].

$$WDY_{total} = \sum ((T \bullet A \bullet B \bullet C \bullet D) + (t \bullet a \bullet b \bullet c \bullet d))$$

T, t – years exposed to wood dust (mechanical or manual work).

A, a – type of wood (%100 of T/t of every type of wood).

B, b – kind of work (%100 of T/t, mechanical or manual work).

C, c – ventilation (%100 of T/t of any ventiltion at mechanical or manual work).

D, d – suction (%100 of T/t of any suction at mechanical or manual work).

The time of exposure was defined as the number of years during which processing of wooden material was documented. Latency was defined as the time between first exposure and the time of diagnosis. Pack years equal to the number of years during which 20 cigarettes per day were smoked.

Statistical analysis

Statistical analysis included nonparametric tests (Mann-Withney U test, x^2-test) and was performed with

SPSS software. Differences with $p < 0.05$ were considered significant.

In addition to the statistical analysis of our group of patients, we tried to establish any morphological differences between selected wood species. This was performed as a single-blind pilot study. Wood dust from pinetree, poplar tree, maple, cherry, two species of spruce, fir, birch, beech, and oak tree was manually produced using abrasive paper with a grain density of 120 DIN (German Industrial Norm). The specimens were mounted and coated with gold to be studied with a scanning electron microscope (SEM). From each specimen several photographs were taken at a magnification of 4000x and 20 000x. The size was measured with a graticule. The photographs were analyzed visually, then given to trained investigators, familiar with the correctly identified pictures, to identify the type of wood by looking at unlabeled photographs.

RESULTS

In a cohort of 28 patients, we found 14 different occupations (Table 1). Of the 28 patients, 20 developed a nasal adenocarcinoma (AC) at a mean age of 59.3 (±5.4) years; 5 developed squamous cell carcinoma (SCC) at a mean age of 54.0 (±5.6) years; and 3 other cases, recognized as occupational disease, had other nasal neoplasms (1 rhabdomyosarcoma, 1 transitional cell carcinoma and 1 Wegener granulomatosis).

These 3 cases were not included in our analysis. The detailed histology showed that of the 20 adenocarcinomas, 14 were of intestinal or mucinous type, 4 of papillary and 2 of tubular types. Of the 5 SCC, 3 were keratinizing, and 2 others were non-keratinizing. The mean latency was 39.5 (±10.5) years, with a latency of 42.9 ± 6.3 years for AC and 36.2 ± 6.2 years for SCC. The total mean WDY were 13.1 (±11.3) years. The proportion of time of exposure to hard wood was higher than that to other types of wood in both groups. The time-dependent data for the AC and SCC groups and their relation to the exposure to various types of wood is shown in Table 2. The group suffering from AC had processed a higher percentage of hard wood (57.3%) as compared to the group with SCC (40.1%), the SCC group had more exposure to other types of wood (52.2%). The combination of the wood subgroups and tumor histology is shown in Table 3. Of the 28 persons, 17 smoked and consumed 31.5 (±30.0) pack years of cigarettes on average. The percentage of smokers was equal in the AC and SCC groups. At the time of diagnosis, syptoms were similar in both groups, with obstruction of nasal breathing, followed by epistaxis and headache (Fig. 1). The distribution of tumor localizations is shown in Fig. 2. The differentiation between AC and SCC showed no tumor-correlated localization.

Statistical analysis of the data showed no difference between the AC and SCC groups in exposure (neither amount nor type), age at the time of diagnosis, smoking, or non-smoking and pack years. The only difference found was a longer latency of 42.9 ± 6.3 years in the AC group compared to 36.2 ± 6.2 years in the SCC group ($p < 0.05$).

Table 1. Occupations found in the cohort (multiple voting possible)

Construction joiner	13	Barrel joiner	1
Cabinet-maker	5	Cartwright	1
Furniture joiner	3	Cooper	2
Coffin maker	3	Forestry worker	1
Parquet layer	2	Wood worker	1
Stairway joiner	2	Farmer	1
Box joiner	1	Construction worker	1

Table 2. Time-dependent data on the AC and SCC groups

		Adenocarcinoma (n = 20)	Squamous cell carcinoma (n = 8)
Age at the time of diagnosis		59.3 ± 5.4 y	54.0 ± 5.6 y
WDY	total	16.6 ± 15.0	11.9 ± 12.3
	HW	8.7 ± 9.9	2.4 ± 2.7
	OW	4.1 ± 7.3	1.0 ± 1.1
Latency		42.9 ± 6.3 y	36.2 ± 15.3 y
Pack years		28.7 ± 31.5	27.6 ± 28.1

WDY – wood dust years.
HW – hard wood
OW – other wood

Table 3. Tumor histology depending on wood types processed

Types of wood	Adenocarcinoma (n = 20)	Squamous cell carcinoma (n = 8)
Other wood	9	4
Hard wood	12	3
Not exactly specified	3	1

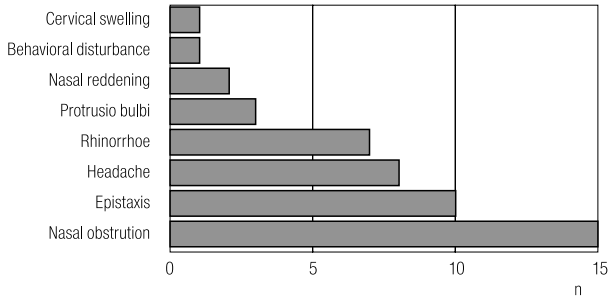

Fig. 1. Symptoms at the time of diagnosis (multiple voting possible).

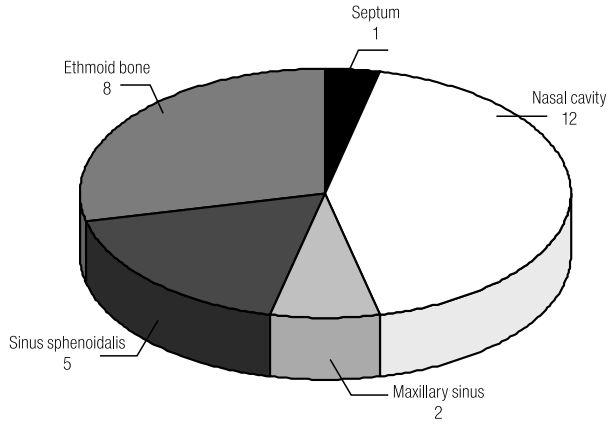

Fig. 2. Localisation of the tumor at the time of diagnosis.

If a cohort study had been used to verify or falsify the correlation between wood dust or special types of wood dust and histological types of tumors, the estimation of the sample size with usual statistical methods [12] would result in numbers which are so large that a realization seems to be impossible due to ethical (duration more than 20 years), practical (more than 1 500 patients necessary) and financial reasons (see discussion).

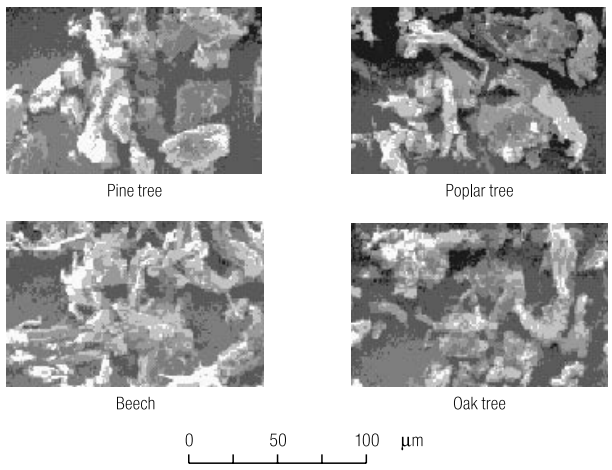

Fig. 3. Wood dust of some different species (SEM, 4 000x).

Visual analysis of several types of wood dust did not reveal any morphological differences. None of the 5 investigators was able to identify the different types of wood dust on electron microscopy pictures (Fig. 3). All the wood fibres measured were >10 μm in size.

DISCUSSION

In Germany, about 200 000 people are working in the wood industry with declining numbers over the last decades (1978: 237 600; 1986: 185 600) [13]. Annually, about 15–20 persons develop nasal tumors, which are recognized as occupational disease according to the current German regulations [14]. This makes nasal carcinoma induced by wood dust a very rare disease although there may be further unidentified cases, including those not considered to be of occupational origin according to current German legislation (e.g., SCC) and thus missing from the statistics. The incidence is low throughout Europe, but in the European Community there are no uniform data on this disease allowing for comparisons.

A possible way to a new appraisal or adaptation of the procedure of recognizing wood dust-induced carcinoma as occupational disease would be a precise description of the etiological factors causing the disease. But to date, the precise mechanism and triggers inducing nasal tumors are not known. Substances of the wood itself, e.g., phenoxyl radicals, chlorophenole, tannine, alcohols are thought to play a role in carcinogenesis [15–18]. This theory is supported by the documented carcinogenic effect of beech extract on skin that can lead to SCC [19]. Other authors view chemicals used during processing, e.g., potassium chromate, sodium chromate, nitrosamines, polycyclic hydrocarbons, formaldehyde or Lindan, pyrolytic substances produced during processing with high-speed devices, or metabolites of fungi which grow on the material, as being responsible for tumorigenesis [20–29].

We additionally investigated the morphology of wood dust. We considered a fiber hypothesis, like in asbestos-induced tumors, where fibers of a certain size (5 μm) are deemed to be more carcinogenic. Examining dust fibers from different types of wood to assess differences between

them, we could not document any differences in size (all >10 μm) or morphology (Fig. 3). One needs to bear in mind, however, that fiber size may vary with different types of processing. In addition, wood dust does degrade and get cleared from tissues in contrast to asbestos fibres which persist a very long time or even lifelong. We conclude therefore that wood fiber size and morphology are probably not related to carcinogenicity.

There is a following argument against the procedure in Germany, Austria, and Luxembourg from a pathological viewpoint. Taking into account the minimal knowledge about the factors of wood dust, which cause the nasal malignancies on one hand, and the remarkable potential of epithelial cells for transformation into different tumor types on the other, any relation of wood dust to a special epithelial tumor type must be discussed very carefully. For example, epithelial cells can develop SCC in tissues, which are normally not containing squamous cells [30]. Adenocarcinoma does not arise uniformly either. In view of this potential of epithelial cells and the resulting variability, we think that nasal SCC cannot be excluded from the list of wood dust-induced nasal neoplasms [31,32].

A large variety of possible carcinogenic substances in the wood and the tissue potential for degeneration into different tumors may explain the multitude of results obtained by numerous authors in various studies [22,33–42]. Most of these are cohort studies or pooled meta-analyses, and are unsuitable for multivariate analysis of the data to assess the effect of other possible influences. The results of our study are in accordance with those reported in the literature [10], but a multivariate analysis to assess additional effects (ethnic predisposition or other carcinogenic organic solvents, aromatic hydrocarbons, chrome, nickel and many others that can induce nasal tumors) [43] was not possible in our study because of a small size of the group. When comparing the results of the studies performed to date, there are no clear trends, and in addition some studies have systematic and statistical deficits [44]. A meta-analysis of the existing data might be beneficial in the future.

The results of the existing case-control studies are similar to ours. For example, Acheson [33] found a higher risk for nasal carcinoma after exposure to hard wood as well as to other wood; Elwood [35] reported a 2.5-time higher risk for a group exposed to other wood compared to a population without wood dust exposure. The data reported by Hernberg [37] and also Vaughan [41] suggest a higher incidence of AC when workers are exposed to hard wood and a higher incidence of SCC after exposure to other types of wood. Demers et al. [34] however, could not show any uniform tendency. The studies, on which current German restrictions in recognising occupational nasal carcinoma are based, were conducted by Grimm and Wolf [45–48]. These case-studies included only data on patients with AC but not on patients with SCC. They documented a relatively higher risk with hard wood exposure and this led to the current German regulations. These studies deserve some criticism because of their systematic errors; a conclusion that has been reached by previous authours as well [10,44].

We set out to detect any differences between the groups of patients, their cancer types and exposure, which would justify the restrictions for recognising nasal carcinoma as an occupational disease as currently applied in Germany, Austria and Luxembourg. We were not able to find any significant differences. However, to increase the statistical significance of any study in this field, a large scale investigation would have to be launched.

If a significance of $p < 0.05$ is to be realized in a cohort study, some 1500 cases will have to be used if the sample size is calculated using our data. Based on the incidence of nasal carcinoma 1:10 000, this would need several decades. Therefore, we suggest a case-control study, a design which is useful for rare diseases, as an alternative.

Taking into account that (i) the IARC recommended the recognition of nasal carcinoma as occupational disease independent of the type of wood dust and the histological type of tumor for more than 20 years, and (ii) that the majority of countries in Europe, except Germany, Austria, and Luxembourg followed this recommendation, our analysis supports the demand for a uniform procedure in whole Europe.

CONCLUSIONS

We propose that in cases of proved high exposure to wood dust any nasal carcinoma should be recognised as an occupational disease independent of the type of wood exposure and the histology of the tumor.

REFERENCES

1. Schroeder H. *Adenokarzinome der inneren Nase und Holzstaubexposition. Schriftenreihe des Hauptverbandes der gewerblichen Berufsgenossenschaften e.V.* St. Augustin: Hauptverband der Gewerblichen Berufsgenossenschaften; 1989.
2. Hanslian L, Kadlec K. *The products of heat-disintegrated wood.* Pracovni Lekarstvi 1964; 16: 276–82.
3. Macbeth R. *Malignant disease of the paranasal sinuses.* J Laryngol Otol 1965; 79: 592–612.
4. Acheson E, Hadfield E, Macbeth R. *Carcinoma of the nasal cavity and accessory sinuses in woodworkers.* Lancet 1967: 311–2.
5. Hadfield E. *A study of adenocarcinoma of the paranasal sinuses in woodworkers in the furniture industry.* Ann Coll Surg England 1969; 46: 301–19.
6. *Wood, leather and some associated industries.* IARC Monographs Research of Cancer. Vol. 25. Lyon: International Agency for Research on Cancer; 1981.
7. Bundesgesetzblatt 1. 1988: p. 400.
8. Merler E, Alhique D, Bracci C. *The new list of occupational diseases in Italy*. Epid Prev 1994; 18: 180–3.
9. *Arbeitsmedizinische Vorsorge*, 2nd ed. St. Augustin: Hauptverband der Gewerblichen Berufsgenossenschaften; 1998. p. 601–6.
10. Chanda R. *Untersuchung zur Äthiologie von epithelialen Tumoren bei Holzstaubeinwirkung unter dem Aspekt der gegenwärtigen Anerkennungspraxis bei beruflich verursachten Erkrankungen in der Bundesrepublik Deutschland* [Thesis]. Düsseldorf: Heinrich-Heine-University; 1999.
11. Bauer H. Faserjahre: *Berufsgenossenschaftliche Hinweise zur Ermittlung der kumulativen Asbestfaserstaub-Dosis am Arbeitsplatz (Faserjahre) und Bearbeitungshinweise zur Berufskrankheit Nr. 4104 (Lungenkrebs)* [Bk-Report 1/97]. Sankt Augustin: Hauptverband der Gewerblichen Berufsgenossenschaften; 1997.
12. Laatz W. *Empirische Methoden.* Thun: Verlag Harri Deutsch; 1993.
13. *Beiträge zur Statistik des Landes Nordrhein-Westfalen. Die Holzverarbeitung in Nordrhein-Westfalen 1978–1986.* Mitteilungen des Landesamtes für Datenverarbeitung und Statistik NRW; 1988. p. 598.
14. Florian H, Franz J, Zerlett G. *Handbuch Betriebsärztlicher Dienst*. Landsberg: Ecomed Verlag; 1993.
15. Hardell L, Johansson B, Axelson O. *Epidemiological study of nasal and nasopharyngeal cancer and their relation to phenoxy acids or chlorophenol exposure.* Am J Ind Med 1982; 3: 247–57.
16. Hausen B. *Woods injurious to human health. A manual.* Berlin: de Gruyter Verlag; 1991.
17. Kirby K. *Induction of tumors by tannin extracts*. Br J Cancer 1960; 14: 147–52.
18. Weissmann G. *Untersuchungen zur Cancerogenität von Holzstaub.* Holz Roh Werkstoff 1992; 50: 421–8.
19. Mohtashamipur F. *The mouse-skin carcinogenity of a mutagenic fraction from beech wood dusts.* Carcinogenesis 1989; 10(3): 483–7.
20. Blair A. *Mortality among industrial workers exposed to formaldehyde.* JNCI 1986; 76(6): 1071–84.
21. Blair A, Stewart P, Hoover R. *Mortality from lung cancer among workers employed in formaldehyde industries.* Am J Ind Med 1990; 17(6): 683–99.
22. Bolm-Audorff U, Albracht G. *Holzstäube, Nitrosamine und polyzyklische aromatische Kohlenwasserstoffe.* Sicher Sicher 1989; 40(2): 79–84.
23. Flechsig R, Nedo G. *Hazardous health effects of occupational exposure to wood dust.* Ind Health 1990; 28(3): 107–19.
24. *Wood dust and Formaldehyde.* IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol. 62. Lyon: International Agency for Research in Cancer; 1994. p. 11–8.
25. Nylander L, Dement J. *Carcinogenic effects of wood dust: review and discussion.* Am J Ind Med 1993; 24(5): 619–47.
26. Ruetze M, Noak D, Schumacher C. *Mögliche Beurteilung von krebserzeugenden Arbeitsstoffen an der Entstehung von Nasenkrebs bei Beschäftigten im holzverarbeitenden Gewerbe. Chemische Beizen.* Holz Roh Werkstoff 1990; 48: 229–35.
27. Henschler D. Gesundheitsschädliche Arbeitsstoffe. Toxikologisch-arbeitsmedizinische Begründung von MAK-Werten. Weilheim: Chemie; 1983.
28. Holmström M, Wilhelmsson B. *Respiratory symptoms and pathophysiological effects of occupational exposure to formaldehyde and wood dust.* Scand J Work Environ Health 1988; 14(5): 306–11.
29. Wilhelmsson B, Lundh B. *Nasal epithelium in woodworkers in the furniture industry. A histological and cytological study.* Acta Otolaryngol Stockh 1984; 98(3–4): 321–34.
30. Schubert G, Bethke B. *Lehrbuch der Pathologie*. 2nd ed. Berlin: de Gruyter; 1987.

31. Kleinsasser O, Schroeder H. *Adenocarcinomas of the inner nose after exposure to wood dust Morphological findings between histopathology and clinical behavior in 79 cases.* Arch Otorhinolaryngol 1988; 245(1): 1–15.
32. Schroeder H. *Adenocarcinomas of the nose after exposure to wood dust.* Adv Otorhinolaryngol 1991; 46: 107–15.
33. Acheson E, Cowdell R, Rang E. *Adenocarcinoma of the nasal cavity and sinuses in England and Wales*. Br J Ind Med 1972; 29: 21–30.
34. Demers P. *Pooled reanalysis of cancer mortality among five cohorts of workers in wood-related industries.* Scand J Work Environ Health 1995; 21: 179–90.
35. Elwood J. *Wood exposure and smoking: Association with cancer of the nasal cavity and paranasal sinuses in British Columbia.* Can Med Assoc J 1981; 124: 1573–7.
36. Gerhardsson M. *Respiratory cancers in furniture workers*. Br J Ind Med 1985; 42: 403–5.
37. Hernberg S. *Nasal and sinusoidal cancer. Connection with occupational exposures in Denmark, Finland, and Sweden.* Scand J Work Environ Health 1983; 9(4): 315–26.
38. Kauppinen T. *Chemical exposures and respiratory cancer among Finnish woodworkers.* Br J Ind Med 1993; 50(2): 143–8.
39. Leclerc A. *Sinonasal cancer and wood dust exposure: Results from a case-control study.* Am J Epidemiol 1994; 140(4): 340–9.
40. Tola S. *A case-control study of the ethiology of nasal cancer in Finland.* Int Arch Occup Environ Health 1980; 46: 79–85.
41. Vaughan T, Davis S. *Wood dust exposure and squamous cell cancers of the upper respiratory tract.* Am J Epidemiol 1991; 133(6): 560–4.
42. Viren J. *Case-control study of nasal cancer in workers employed in wood-related industries.* J Occup Med 1989; 31(1): 35–40.
43. Arnold W. *Checkliste Hals-Nasen-Ohrenheilkunde*. 2nd ed. Stuttgart: Thieme Verlag; 1997.
44. Brüske-Hohlfeld I. *Das Adenokarzinom der Nase als Berufskrankheit (BK 4203): Ist die Beschränkung auf Eichen- und Buchenholzstaub als verursachendes Prinzip berechtigt?* Zbl Arbeitsmed 1992; 42: 258–64.
45. Grimm H. *Über das Vorkommen von Adenokarzinomen der Nasenhaupt- und Nasennebenhöhlen bei Holzarbeitern. Empirisch-kasuistische Studie.* Arbeitsmed Sozialmed Präventivmed 1984 (Sonderheft 4).
46. Wolf J. *Über das Vorkommen von Adenokarzinomen der Nasenhaupt- und Nasennebenhöhlen bei Holzarbeitern. Weitere Untersuchungen (II).* Arbeitsmed Sozialmed Präventivmed 1986. Sonderheft 7.
47. Wolf J. *Bösartige Tumoren der Nase im Bereich der Holzwirtschaft. Empirisch-kasuistische Studie zur Belastungssituation (III)*. Arbeitsmed Sozialmed Präventivmed 1988. Sonderheft 10.
48. Wolf J. *Berufliche Fremdstoffbelastung in der Holzwirtschaft. Ergebnisse einer empirisch-kasuistischen Studie über das Vorkommen von Nasentumoren.* Staub Reinerhaltung Luft 1990; 50: 29–32.