Chemical exposures and respiratory cancer among Finnish woodworkers

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Abstract

A case-control study of respiratory cancer, nested within a cohort of male woodworkers, was updated in Finland. The update extended the initial follow up of 3805 workers from 19 plants to 7307 workers from 35 plants. Each case of respiratory cancer (n = 136) diagnosed between 1957 and 1982 within the cohort was matched by year of birth with three controls (n = 408) from the cohort. Chemical exposures were assessed for the cases and the controls by a plant and period specific job exposure matrix. An excess of respiratory cancer was associated with phenol. Concomitant exposures to several other agents occurred as well, however, and no exposure-response relation for phenol was seen. An excess risk and an increasing exposure-response relation were found for engine exhaust from petrol and diesel driven factory trucks. The excess risk associated with pesticides was lower than in our previous study, an indication of qualitative and quantitative differences in exposure between the initial and augmented cohorts. Slightly increased risks were found for terpenes and mould spores, which may be due to chance although the contribution of occupational exposure cannot be ruled out. Exposure to wood dust, mainly from pine, spruce and birch, at a level of about 1 mg/m^3, was not associated with lung cancer, upper respiratory cancer, or adenocarcinoma of the lung.

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Furniture workers have been shown to have an excess risk of nasal cancer in many studies from different countries.[1] The relative risks have been 10-fold or even higher, especially for nasal adenocarcinomas.[2] Slightly lower and less consistent risks have been found among carpenters and other woodworkers. The probable cause of these excesses is exposure to wood dust,[1 3] but neither the causative agents in the dust nor the hazardous species of wood have been identified conclusively. Although mainly oak, beech, mahogany, and other hardwood species have been processed by the workers studied,[4] exposure to species of softwood cannot be ruled out as a risk factor.[5] Because a significant proportion of the inhaled wood dust is transported to the lungs, woodworkers may also have an increased risk of lung cancer. Epidemiological publications are inconsistent, however. Some studies showed an excess of lung cancer among woodworkers[6–12] whereas others did not.[13–20] There are indications that the increased lung cancer may, parallel to nasal cancer, concern mainly adenocarcinomatous tumours. In a United States case-control study of lung cancer, the odds ratios for adenocarcinomas among carpenters and cabinet and furniture makers were higher than for other histological types of lung cancer.[8]

Lung cancer is potentially an important occupational disease among woodworkers. Should there be even a small work related excess among woodworkers, the absolute number of lung cancer cases caused by occupational exposure easily overshadows that of nasal cancers. So far the associations between cancer and specific exposures encountered in the wood industry have rarely been scrutinised. As well as wood dust, many other agents related to gluing (formaldehyde, phenol, melamine, etc), wood preservation (chlorophenols, arsenic compounds, chromium compounds, other pesticides), surface treatment (solvents, pigments) or other activities (engine exhaust, terpenes, etc) are present in the wood processing facilities.

The objective of the present study was to investigate associations between chemical agents in the wood industry and respiratory cancer. The associations were studied conditionally on potential confounding factors (smoking, vital status), grade of exposure, and the induction period of the disease. This paper reports the results of chemical exposures other than formaldehyde. Formaldehyde has been

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dealt with in a separate paper.[21] The results of an earlier case-control analysis based on the initial cohort have been published in two earlier papers.[22 23]

Material and methods

A nested case-control design was applied in a cohort of Finnish male woodworkers. A total of 7307 workers from 35 plants were followed up for the development of respiratory cancer. The cohort comprised sawmill workers (n = 2531), plywood workers (n = 1775), furniture workers (n = 1483), workers in construction carpentry factories (n = 876), particle board workers (n = 630), and workers producing glues for the wood industry (n = 12). Employment lasting for one year or more between 1 January 1944 and 31 December 1965 was required for admission to the cohort.

All cases of respiratory cancer (n = 136) diagnosed between 1957 and 1982 among the members of the cohort were identified through the Finnish Cancer Registry, the coverage of which is practically complete across the nation. The respiratory cancers were, by definition, primary malignant neoplasms at sites with a possibility of direct epithelial contact with inhaled agents—namely, lungs, trachea (ICD–7 code 162·0–1; n = 117 cases); larynx, epiglottis (161; n = 12); tongue (141; n = 3); pharynx (145–8; n = 2); mouth, other (143–4; n = 1); and nose, sinuses (169; n = 1). Four diagnoses of lung cancer could not be confirmed by the pathologist in our team (JN) on the basis of histological, cytological, necropsy, and hospital data available, and they were omitted from the material. Seven of the lung cancers were adenocarcinomas by histological type.

Three controls (n = 408) from the cohort who had not contracted respiratory cancer and who were alive at the time of the diagnosis of the case were matched to each case on year of birth but not on vital status.

The initial, smaller cohort comprised 3805 workers (52% of the extended cohort, to which the data of this study refer). The follow up (1957–80) of the initial cohort resulted in 57 respiratory cancers (42% of the 136 respiratory cancers of the extended cohort). The follow up of the extended cohort was discontinued in 1982 for practical reasons.

Plant and time specific job exposure matrices (JEMs, see[24]) were constructed for 12 major agents present in the wood industry. The workers were assigned to homogenous exposure zones on the basis of their work area and job, and a computer programme transformed their work histories into exposure histories by using the JEMs. The work histories were based on plant records. Questionnaires on former employments were mailed to the cases and controls or to their next of kin, and interviews of supervisors and coworkers were conducted during the plant visits to improve the assessment of exposure. The questionnaires also inquired into former smoking habits of the cases and controls. The JEMs were based on industrial hygiene measurements, walk throughs in the plants, interviews of persons with long experience on circumstances of exposure in the plants, and documents provided by the plants. The collection of the work histories and the construction of the JEMs were carried out independently of each other, and the assignment of cases and controls to the exposure categories was "blinded" as to the case or control state.

Several indicators of exposure were derived for the agents under study. Exposure to wood dust was treated both as a qualitative indicator (yes *v* no; yes referring to exposure to at least 0·1 mg/m^3 in the workroom air during at least one month), and as several quantitative indicators, such as the mean level of exposure and the cumulative exposure (product of the mean level and the duration of exposure). Less data were available on phenol, pesticides, terpenes, engine exhaust, chlorophenols, solvents, casein-albumin glues, melamine glues, mould spores, and bis(chloromethyl)ether. For them, a qualitative (yes *v* no) and a simple quantitative (the duration of exposure) indicator were used. Vital status and smoking were considered potential confounders, and the analyses were adjusted for them to control their effect. An induction period of 10 years was allowed for in some analyses to obtain additional information on the time dependence of the possible risks.

In most statistical analyses, all respiratory cancers were pooled because the number of exposed cases was small for most exposures. Exposure to wood dust could be analysed by cancer site, however, (upper respiratory cancer *v* lung cancer), and partially by histological type (adenocarcinoma of the lung).

Estimates for odds ratios (ORs) were calculated whenever possible by conditional logistic regression and the 90% confidence intervals (90% CIs) under the assumption of Gaussian distribution of the estimates of the coefficients in the logit model. The results of the conditional logistic regression were checked against unconditional logistic regression analyses, based on cross stratification of the unmatched data and the estimation method of Gart[25] and Cornfield.[26] Indicators of vital status (alive *v* deceased), smoking years (<35 *v* $\geqslant 35$ years), and the induction period (0 *v* $\geqslant 10$ years) were included in both analyses. Cases and controls with missing smoking data (35% of subjects) were discarded from the analyses in which the indicator of the smoking history was involved. Because the results from conditional and unconditional analyses in most cases agreed well, the results of unconditional analyses are reported only when the conditional analysis was not applicable.

Table 1 Odds ratios (ORs) and 90% confidence intervals (90% CIs) for exposure to wood dust and respiratory cancers (pooled), upper respiratory cancer, lung cancer, and adenocarcinoma of the lung

Type of cancer and exposure to wood dust	*Minimum induction period*	*Adjustment for smoking*	*No of exposed cases*	*OR (90% CI)†*
Respiratory cancers (pooled):				
Any exposure	No	No	82	1·05 (0·74–1·50)
(>1 month)		Yes	48	0·98 (0·60–1·61)
	10 y	No	74	0·91 (0·65–1·28)
		Yes	43	0·97 (0·60–1·56)
Level 0·1–1 mg/m³	No	No	53	1·19 (0·74–1·89)
		Yes	30	0·86 (0·46–1·59)
	10 y	No	49	0·99 (0·68–1·44)
		Yes	27	0·98 (0·57–1·68)
Level >1 mg/m³	No	No	29	0·64u (0·30–1·37)
		Yes	18	0·74u (0·22–2·56)
	10 y	No	25	0·87 (0·53–1·43)
		Yes	16	0·83 (0·44–1·57)
Cumulative	No	No	29	1·09 (0·69–1·74)
0·01–5 mg/m³—y		Yes	14	0·96 (0·48–1·92)
	10 y	No	33	0·84 (0·56–1·26)
		Yes	16	0·94 (0·52–1·69)
Cumulative	No	No	53	1·12 (0·75–1·68)
>5 mg/m³—y		Yes	34	1·00 (0·58–1·71)
	10 y	No	41	1·05 (0·69–1·60)
		Yes	27	1·09 (0·60–1·99)
Upper respiratory cancer:				
Any exposure	No	No	11	1·18 (0·39–3·53)
(>1 month)		Yes	7	1·56 (0·26–9·33)
	10 y	No	10	1·14 (0·38–3·37)
		Yes	6	0·60 (0·04–8·27)
Lung cancer:				
Any exposure	No	No	71	0·68 (0·39–1·18)
(>1 month)		Yes	41	1·31u (0·82–2·10)
	10 y	No	64	0·59 (0·35–0·99)
		Yes	37	0·44 (0·18–1·08)
Adenocarcinoma of the lung:				
Any exposure	No	No	3	*
(>1 month)		Yes	0	*
	10 y	No	3	*
		Yes	0	*

*Small numbers (No of exposed cases <4).
†Values by conditional logistic regression; adjusted for vital status.
u Unconditional logistic regression because conditional regression was not applicable.

Results

No indications of raised risk or exposure-response relation concerning exposure to wood dust were found (table 1). Three of the seven cases of adenocarcinoma (43%) were exposed to wood dust, which was fewer than among the rest of the cases of lung cancer (64%), or among all controls (60%). The estimated mean level of exposure was about 1 mg/m³. The subjects were mainly exposed to softwood dust, such as from pine and spruce, but many of them were also exposed to hardwood dust, such as from birch. The estimated proportions of workers exposed to individual wood species were: pine 90–100%, spruce 60–70%, birch 50–60%, oak 20–30%, teak 10–20%, ash 10–20%, elm 10–20%, obeche <10%, mahogany <10%, and other species <10% each. Almost all workers had been exposed to several types of wood dust.

Significantly raised ORs were found for phenol and engine exhaust (table 2). Exposure to phenol was mainly due to the use of phenol-formaldehyde resins in the gluing of plywood. Engine exhaust was produced mainly by the forklifts used, for example, in plywood mills and sawmills. The fuel used by the forklifts was predominantly petrol or diesel. Pesticides refer to impregnation agents of wood used in the manufacture of plywood, doors, and windows (containing, for example, inorganic arsenic, copper, and chromium compounds), and insecticides mixed to certain plywood glues (containing for example lindane, aldrin, and tributyltin oxide). Terpene vapours were released from fresh coniferous wood during sawing or drying. In plywood plants the occurrence of terpenes was often characterised by a bluish haze containing a complex mixture of compounds generated in dimerisation, oxidation, and other chemical reactions of terpenes. The main chlorophenol preparation used in sawmills as fungicide was the sodium salt of 2,3,4,6-tetrachlorophenol that also contained the 2,4,6-tri- and penta-isomers. Solvent vapours originated mainly from painting or varnishing of furniture, involving the common use of mixtures of aliphatic and aromatic hydrocarbons, ketones, esters, and alcohols as solvents. Casein-albumin glues based on milk and blood were commonly used in the gluing of plywood in the 1940s and

Table 2 Odds ratios (ORs) and 90% confidence intervals (90% CIs) for respiratory cancers and exposure to agents other than wood dust

Exposure	Minimum induction period	Adjustment for smoking	No of exposed cases	OR (95% CI)†
Phenol:				
Any exposure (>1 month)	No	No	14	3·16 (1·77–5·62)
		Yes	9	2·47 (1·22–5·01)
	10 y	No	6	3·54 (1·79–7·02)
		Yes	5	4·04 (1·83–8·89)
Duration 1 month–5 y	No	No	7	3·33u (1·01–10·9)
		Yes	3	*
Duration >5 y	No	No	7	1·40u (0·55–3·61)
		Yes	6	3·08u (0·70–13·6)
Engine exhaust:				
Any exposure (>1 month)	No	No	11	2·60 (1·16–5·83)
		Yes	8	1·70 (0·55–5·20)
Duration 1 month–5 y	No	No	4	1·57u (0·35–7·04)
		Yes	3	0·39u (0·05–2·94)
Duration >5 y	No	No	7	2·71u (1·10–6·67)
		Yes	5	2·21u (0·65–7·48)
Pesticides:				
Any exposure (>1 month)	No	No	17	1·34 (0·84–2·13)
		Yes	10	1·16 (0·63–2·16)
	10 y	No	17	1·40 (0·76–2·75)
		Yes	10	1·49 (0·81–2·74)
Duration 1 month–5 y	No	No	8	1·22u (0·52–2·86)
		Yes	4	1·00u (0·20–5·04)
Duration >5 y	No	No	9	1·41u (0·57–3·48)
		Yes	6	1·41u (0·32–6·18)
Terpenes and other heating products of coniferous wood:				
Any exposure (>1 month)	No	No	17	1·48 (0·91–2·41)
		Yes	16	1·33 (0·78–2·27)
	10 y	No	14	1·44 (0·86–2·40)
		Yes	13	1·23 (0·70–2·18)
Duration 1 month–5 y	No	No	5	0·97u (0·34–2·73)
		Yes	5	1·74u (0·42–7·17)
Duration >5 y	No	No	12	1·23u (0·52–2·91)
		Yes	11	0·79u (0·23–2·73)
Chlorophenols:				
Any exposure (>1 month)	No	No	9	0·87 (0·42–1·79)
		Yes	4	0·80 (0·28–2·22)
	10 y	No	8	0·52 (0·22–1·24)
		Yes	2	*
Solvents:				
Any exposure (>1 month)	No	No	3	*
		Yes	2	*
Casein-albumin glues:				
Any exposure (>1 month)	No	No	23	1·33 (0·87–2·04)
		Yes	12	1·15 (0·61–2·15)
Melamine glues;				
Any exposure (>1 month)	No	No	2	*
		Yes	1	*
Mould spores:				
Any potential Exposure (>1 month)	No	No	28	1·14 (0·65–2·02)
		Yes	23	1·50 (0·64–3·51)
Bis(chloromethyl)ether:				
Any potential Exposure (>1 month)	No	No	9	1·20 (0·42–3·46)
		Yes	4	0·23 (0·04–1·53)

*Small numbers (No of exposed cases <4).
†Values by conditional logistic regression; adjusted for vital status.
u Unconditional logistic regression because conditional regression was not applicable.

1950s before the introduction of urea and phenol formaldehyde glues. Melamine formaldehyde glues are newer and have not been used much in Finland. Mould spores growing in wood were present in workroom air especially in sawmills. Bis(chloromethyl)ether is a known lung carcinogen. Its formation is theoretically possible when formaldehyde reacts with ammonium chloride during hot pressing of particle board or plywood glued with urea-formaldehyde resin. The presence or absence of bis(chloromethyl)ether in the workroom air of particle board or plywood plants was not verified by measurements.

Discussion

The present analysis is based on the extension of the

initial cohort. The number of cases increased 2·4-fold, thus improving the power of the study correspondingly. The methods used were nearly the same as in the previous study and they have been discussed elsewhere.[21–24] Vital status, smoking habits, and the induction period were considered potential confounding factors and they were therefore incorporated in the logistic models used in the statistical analyses. The smoking data allowed only an incomplete control for confounding, however, and there may still be residual confounding in the results. Instead of unconditional logistic regression analysis, which was used due to small numbers in the analysis of the initial cohort, conditional logistic regression models could this time be applied in most analyses. The present results may therefore be considered as more precise (more data) and valid (improved statistical methods) than those of the previous study.

Exposure to wood dust was not associated with respiratory cancers. The ORs were close to unity and no trends by level or cumulative exposure indicating an exposure-response relation were found. Adjustment for smoking and induction period did not alter the results. This finding is in accordance with most other studies that have considered the risk of lung cancer among woodworkers. Our study did not support the hypothesis that wood dust would cause specifically adenocarcinoma of the lung as suggested in one previous study.[8] It should be noted, however, that the mean level of exposure was rather low (about 1 mg/m^3), and only about 30% of the workers were exposed to any extent to hardwood dust other than from birch.

Only one case of nasal cancer was found among the cohort. A worker, who had been exposed to birch dust in 1946–50 while making plywood barrels, contracted a nodular lymphoma of the left sinus in 1982. The expected number of nasal cancers in the cohort was also close to one. The lack of an excess may have several explanations. It might be argued that the species of wood in the present study are less carcinogenic than in many other studies showing an excess. It is also possible that the lack of an excess may be due to the comparatively short follow up (18–39 years, mean 27 years) and the fairly low level of exposure to wood dust (around 1 mg/m^3 on average). The latency period for the development of nasal adenocarcinomas in woodwork is usually very long. For example, Acheson *et al*[27] have reported a mean of 44 years for patients, most of whom had been exposed to a level of over 5 mg/m^3 for more than 20 years. Hence, our study should not be considered to indicate absence of risk, but rather as uninformative for nasal cancer.

Exposure to phenol was significantly associated with respiratory cancers. This finding is in accordance with the result obtained from the initial cohort. The initial and extended materials overlap strongly because only two new cases exposed to phenol occurred in the extension of the study. The pattern of the observed ORs is difficult to interpret. There were no clear trends by duration of exposure or by latency period. Workers exposed to phenol mainly work in plywood mills where they are usually exposed to several other agents such as formaldehyde, engine exhaust, wood dust, and sometimes pesticides. It is conceivable that phenol has a combined effect with some other agents present. For example, when exposure to formaldehyde and phenol was analysed, the combined exposure provided a higher OR (1·6) than exposure to formaldehyde alone (1·0).[21] Exposure to phenol alone could not be analysed because everyone exposed to phenol was also exposed to formaldehyde. Phenolic compounds have been reported to exert a promoter activity in old studies in animals, and the evidence for the carcinogenicity of phenol has been evaluated as being inadequate by an expert group convened by the International Agency for Research on Cancer (IARC).[28]

Engine exhausts from petrol and diesel fuel powered factory trucks were associated with a raised OR that was slightly reduced after adjustment for smoking. There was a positive trend by duration of exposure indicating an exposure-response relation. These results are in keeping with the evaluation made by the IARC according to which diesel engine exhaust is probably carcinogenic and petrol engine exhaust possibly carcinogenic in humans.[29]

The ORs for exposure to pesticides were lower than in the initial analysis. The overall OR was 1·3 based on 17 exposed cases, whereas previously it was 2·9 based on seven exposed cases. The positive trends of the OR by duration of exposure and by latency period could still be seen but they were now more attenuated than in the previous analysis. The reason for this is partly that many of the new cases came from construction carpentry (manufacture of doors, windows, and wooden houses), whereas the initial cases mainly came from the plywood industry. There were therefore both qualitative and quantitative differences in exposure to pesticides between the initial and extended cohorts. Also, conditional regression analysis instead of unconditional analysis caused slight differences in the results.

Exposure to terpenes and other heating products of pine and spruce were only weakly associated with respiratory cancers. The positive trend by duration of exposure, which was strong in the initial material, had disappeared suggesting that the previous finding may have been attributable to chance. We are not aware of any epidemiological or experimental studies on the carcinogenicity of α pinene or δ carene, which are the major terpenes released from pine and spruce.

Potential exposure to mould spores was weakly associated with respiratory cancer both in the previous and present analyses. This association may

well be due to chance, but the contribution of mould spores cannot be ruled out. Even though the species of mould known to occur in wood have not been reported to produce such mould borne carcinogens as aflatoxins, other toxins with unknown carcinogenic potential may be produced and inhaled by woodworkers.

Non-positive or uninformative results were found for exposure to chlorophenols, casein-albumin glues, and bis(chloromethyl)ether. Even though some chlorophenols are carcinogenic in experimental animals, and even if there is limited evidence of their carcinogenicity among exposed workers, the types of cancer associated with exposure to chlorophenols have been mainly soft tissue sarcomas and non-Hodgkin's lymphomas, not respiratory cancers.[30] Bis(chloromethyl)ether is a known lung carcinogen.[31] One interpretation of the absence of risk in the present material is that the formation of bis-(chloromethyl)ether in the studied industries is negligible.

In summary, we found that exposure to wood dust, predominantly from pine, spruce, and birch, at the estimated level of 1 mg/m^3 was not associated with the risk of lung cancer, upper respiratory cancer, or adenocarcinoma of the lung. Exposure to phenol was significantly associated with respiratory cancers, but the role of phenol itself is difficult to assess because simultaneous exposure to several other agents took place. An increased risk and an exposure-response relation by duration of exposure was found for engine exhaust from petrol and diesel driven factory trucks. A lower risk than in our previous study was found for pesticides, suggesting qualitative and quantitative differences in exposure between the initial and extended cohorts. Exposures to terpenes and mould spores were slightly associated with respiratory cancers, which may be due to chance, although the role of occupational exposure cannot be ruled out.

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