Aims: To explore the interaction of smoking and occupational exposure to noise as risk factors for hearing difficulty in the general population.

Methods: A questionnaire was mailed to 21,201 adults of working age, selected at random from the age-sex registers of 34 British general practices, and to 993 members of the armed services, randomly selected from pay records. Questions were asked about smoking habits, years spent in a noisy occupation, difficulty in hearing conversation, and wearing of a hearing aid. Associations of hearing difficulty with smoking habit were examined by logistic regression and compared across strata of noise exposure, with adjustment for potential confounders.

Results: Among half of the respondents had ever smoked, and half of these still smoked. Among 10,418 who provided details on hearing, 348 were classed as having moderate and 311 as having severe hearing difficulty. Risk of hearing difficulty was 3–5-fold higher in those employed for ≥5 years in noisy work compared with those never employed in a noisy job. Within strata of noise exposure (including those who had never worked in a noisy job), ex- and current smokers had a higher risk of hearing difficulty than lifetime non-smokers. The additional risks were small compared with those of long term noise exposure, and the combination of effects was more consistent with an additive than a multiplicative interaction.

Conclusions: Smoking may adversely affect hearing, and workers should be encouraged to refrain from both smoking and exposure to noise. However, the extra risk to hearing incurred by smoking in high ambient noise levels is small relative to that from the noise itself, which should be the main target for preventive measures.
Hearing, in which the response category “moderate difficulty” corresponded to a measured mean hearing impairment of about 45 dB.20 We classed subjects as severely affected if they wore a hearing aid or reported severe hearing difficulty in the worse affected ear; as being moderately affected if they reported moderate hearing difficulty in the worse ear; and as normal if they reported only slight hearing difficulty in the worse ear or no difficulty at all. (In those who only reported hearing in one ear, the category was that for the ear on which information was given.) Tinnitus was identified by asking: “During the past 12 months have you had noises in your head or ears (such as ringing, buzzing or whistling) which lasted longer than five minutes?” It was defined as “persistent” if it was reported to occur most or all of the time. A smoker was defined as someone who had smoked at least once a day for a month or longer, and classed according to whether he or she still smoked regularly (current smoker) or had stopped (former smoker).

Information was also collected on age and sex as potential confounding factors, and on complaints of frequently feeling tired or stressed or suffering frequent headaches, as possible markers of lowered threshold for reporting symptoms in general. All statistical analyses were carried out in STATA, release 5.21 Associations of hearing impairment and tinnitus with smoking were examined by logistic regression, with adjustment for potential confounding. Separate analyses were conducted according to duration of employment in a noisy job. The findings were expressed as prevalence ratios (PRs) with associated 95% confidence intervals (CIs). These were derived from the corresponding odds ratios using a formula proposed by Zocchetti and colleagues.22

RESULTS
Questionnaires were returned by 12 907 subjects (58% of those selected for study), with a higher response rate in older subjects and women. The occupational distribution of responders was generally similar to that in the preceding national census.23 Further details on the response pattern have been published elsewhere.27

The respondents included 3184 current smokers, 3329 former smokers, and 6394 lifelong non-smokers. Non-smokers tended to be younger and reported working fewer years in a noisy occupation than ex- and current smokers.

Among 10 418 respondents who provided details on hearing, 9759 were classed as having slight or no hearing difficulty, 348 as having moderate hearing difficulty, and 311 as having severe hearing difficulty (including 165 who wore a hearing aid). Persistent tinnitus was reported by 527 subjects. As reported previously,1 the prevalence of tinnitus was higher in those with hearing difficulties (about twice as common in age standardised comparisons), and both symptoms were more common at older ages. Subjects who felt frequently tired or stressed, or suffered frequent headaches, also reported such problems more often.

Table 1 shows the association of hearing difficulties and tinnitus with smoking habit and years in noisy work, after adjustment for report of tiredness, stress, and headaches, and for age and sex. The baseline for comparison was subjects who were free of auditory problems, and all risks were estimated relative to those of non-smokers who had never worked in a noisy job.

The risks of moderate-to-severe hearing difficulty, severe hearing difficulty, and persistent tinnitus increased with years spent in noisy work for all categories of smoking habit, being some three to five fold higher in those employed for more than five years than in those never employed in noisy work.

Within strata of noise exposure, ex- and current smokers had a moderately higher risk of hearing difficulty than lifetime non-smokers in most comparisons. For example, the PR for moderate-to-severe hearing difficulty was 1.5 (95% CI 1.1 to 2.1) in current versus never-smokers without exposure to noise at work. By contrast, after allowance for age and duration of noisy employment, smoking status was only weakly related to tinnitus if at all.

The combined effect on hearing difficulty of current smoking and long term noise exposure was generally less than multiplicative, and more consistent with an additive effect. Thus, among subjects with moderate-to-severe hearing difficulty, the risk difference between smokers and non-smokers was similar in every stratum of years employed in noise; and the same was true among subjects with severe hearing difficulty who had never worked in a noisy job or had been employed in it for up to five years. In those with severe hearing difficulty who had been employed for more than five years in a noisy job, the risk difference between smokers and non-smokers was greater, but the interaction was still less than multiplicative.

DISCUSSION
This population based survey tends to confirm that smoking is associated with hearing difficulty, and that this relation exists even in those who have never been exposed to occupational sources of noise. However, it indicates that any such effect is modest in comparison with long term employment in a noisy job, and that the combined effects of smoking and noise are additive rather than multiplicative.

A strength of our survey was the large, geographically dispersed, and well defined sampling base, which included over 22 000 randomly selected subjects across England, Wales, and Scotland. The distribution of occupations in respondents was nationally representative, being similar to that at the preceding national census.

Participation was incomplete (response rate 58%), but response bias would only explain the association of smoking with hearing difficulty if smokers with symptoms participated more readily than smokers without. This seems unlikely, and similar associations were found in those who responded to the questionnaire at the first invitation and those who required a reminder (data not presented), providing evidence against this pattern of bias.
### Table 1: Associations between self reported hearing difficulties, tinnitus, smoking, and working in a noisy occupation

| Hearing problem/smoking habits | Years worked in noisy job* | None | <1–5 | >5 | None | <1–5 | >5 | None | <1–5 | >5 |
|-------------------------------|---------------------------|------|------|----|------|------|----|------|------|----|------|------|
| **Severe hearing difficulty†** |                           |      |      |    |      |      |    |      |      |    |      |      |
| Never smoked                  | 2698                       | 2698 | 45   | 1.7| 2698 | 551  | 18 | 3.3  | 2.2 | 1.3 to 3.7| 293  | 31 | 10.6 | 4.6 | 2.9 to 7.1 |
| Formerly smoked               | 1109                       | 1109 | 37   | 3.3| 1109 | 362  | 15 | 4.1  | 2.0 | 1.1 to 3.5| 256  | 47 | 18.4 | 5.9 | 3.9 to 8.7 |
| Currently smoking             | 915                        | 915  | 23   | 2.5| 915  | 338  | 15 | 4.4  | 2.4 | 1.4 to 4.2| 208  | 30 | 14.4 | 5.8 | 3.7 to 8.9 |
| **Moderate/severe hearing difficulty†** |                       |      |      |    |      |      |    |      |      |    |      |      |
| Never smoked                  | 2736                       | 2736 | 83   | 3.0| 2736 | 573  | 40 | 7.0  | 2.4 | 1.7 to 3.3| 341  | 79 | 23.2 | 5.0 | 3.8 to 6.3 |
| Formerly smoked               | 1140                       | 1140 | 68   | 6.0| 1140 | 384  | 37 | 9.6  | 2.4 | 1.7 to 3.4| 319  | 110| 34.5 | 5.9 | 4.7 to 7.4 |
| Currently smoking             | 943                        | 943  | 51   | 5.4| 943  | 367  | 44 | 12.0 | 3.3 | 2.4 to 4.9| 248  | 70 | 28.2 | 5.7 | 4.4 to 7.1 |
| **Persistent tinnitus‡**      |                           |      |      |    |      |      |    |      |      |    |      |      |
| Never smoked                  | 2752                       | 2752 | 99   | 3.6| 2752 | 574  | 41 | 7.1  | 1.9 | 1.4 to 2.7| 312  | 50 | 16.0 | 3.0 | 2.2 to 4.1 |
| Formerly smoked               | 1131                       | 1131 | 59   | 5.2| 1131 | 394  | 47 | 11.9 | 2.4 | 1.7 to 3.3| 277  | 68 | 24.5 | 3.8 | 2.8 to 5.0 |
| Currently smoking             | 929                        | 929  | 37   | 4.0| 929  | 355  | 32 | 9.0  | 2.1 | 1.4 to 3.0| 223  | 45 | 20.2 | 3.6 | 2.5 to 4.9 |

*Years in a job where there was a need to shout to be heard.
†Severe = severe or can’t hear at all in either ear, or wearing of a hearing aid; moderate = moderate difficulty in the worse affected ear (and no use of a hearing aid).
‡Persistent = most or all of the time in the past 12 months.

No. (%) = number and % positive for the hearing outcome.

The baseline for all risk estimates was subjects who were free of auditory problems. All risks were estimated relative to those of non-smokers who had never worked in a noisy job. All PRs were adjusted for age, sex, and self report of frequent tiredness or stress and frequent headaches. The analysis excludes 50 subjects with severe hearing difficulty, 77 with moderate to severe hearing difficulty, and 49 with tinnitus who failed to provide information on years worked in a noisy job or frequency of tiredness, stress, and headaches.
In view of the relatively small number of working aged subjects reporting hearing loss, smoking status rather than pack-years of smoking was used as the index of exposure. This precluded examination of a dose-response relation, but subjects are less likely to have been misclassified using this metric of exposure. Major error arising from the question on hearing impairment also seems unlikely, as the validity of this item has previously been established. However, our metric of exposure to noise (years worked in a noisy job where there was a need to shout to be heard) was crude to the extent that it did not fully reflect intensity of noise above a likely threshold of 85–90 dB(A). Among noise exposed workers, differences in intensity of noise exposure or degree of compliance with wearing of ear defenders may have existed by smoking status. This may have caused residual confounding after allowance for years of employment in noise, but cannot explain the increased risk in smokers who had never held a noisy job.

A number of other potential explanations for the association need to be considered. Smokers may have reported hearing difficulty more readily than non-smokers, perhaps as part of a general tendency to report somatic symptoms at a lower threshold; but the association persisted despite adjustment for markers of such a tendency (frequent complaints of headaches, tiredness, and stress).

Alternatively, assuming a genuine effect, smoking may act as a direct ototoxin; may impair blood flow to the cochlea by inducing vasospasm, atherogenic narrowing, and thrombotic occlusion of the nutrient arteries; or may alter blood viscosity, or generate carboxyhaemoglobin, exacerbating any problems of local hypoxia.

There have been only a few population based surveys of smoking and hearing loss previously. When 1662 elderly subjects from the Framingham study underwent audiometric screening, no association was found between hearing thresholds and current smoking status or number of cigarettes smoked. And hearing thresholds in middle age did not deteriorate more rapidly during follow up among smokers than non-smokers from rural Denmark. But in the US Health Interview Survey, men who smoked two or more packs per day more often reported hearing problems than non-smokers; in a population based cross-sectional survey based in Beaver Dam, USA, a 25 dB hearing loss in the worse ear was 1.7 times more common in current smokers than lifetime non-smokers after adjustment for other factors; and in another study, which sampled non-smokers from the community, a moderately increased risk of hearing loss (OR 1.3) was found in association with exposure to environmental tobacco smoke. Surveys set within programmes of health screening and hearing conservation have produced some of the most convincing evidence that smoking is detrimental to hearing. For example, in Japan, where labour law mandates periodic audiometry for all employees irrespective of noise exposure, two large surveys found worse hearing among smokers than non-smokers in non-noisy white collar occupations, and a dose-response effect among current smokers which was much clearer for high frequency hearing loss than for low frequency loss. Mizoue and colleagues have suggested that inner ear cells responsible for high frequency hearing are more vulnerable to ischaemic damage, as they are located at the end of nutrient arteries, and that the inconsistency of findings across studies may reflect failure to distinguish the different potential patterns of hearing loss.

The combined effects of noise and smoking have only occasionally been explored. In keeping with our findings, Mizoue et al reported an additive effect, based on periodic health data from 4624 Japanese steel company workers. But by contrast, Prince et al found that the combined effect of these risk factors was more than that from simple additivity.

Workers should be encouraged as a matter of good public health policy to avoid the hazards of both noise and smoking. But our results suggest that from the viewpoint of hearing there is no strong imperative to limit the employment of smokers to a greater extent than non-smokers where ambient noise levels are significant. Counselling may be appropriate in these circumstances, but our data indicate only a small additional risk from smoking and underscore the importance of noise as the primary target for preventive measures in hearing conservation programmes.

ACKNOWLEDGEMENTS

This study was supported by a grant from the Health and Safety Executive. We thank the Royal College of General Practitioners, the Primary Care Rheumatology Society, and the general practices for help in assembling the mailing lists, and the MRC staff who were involved in data handling. Professor Adrian Davies kindly recommended the questions on hearing difficulty and tinnitus. Denise Gould prepared the manuscript.

Authors’ affiliations

K T Palmer, H E Syddall, D Coggan, MRC Environmental Epidemiology Unit, Community Clinical Sciences, University of Southampton, UK
M J Griffin, Institute of Sound and Vibration Research, University of Southampton, UK

REFERENCES


Clinical Evidence—Call for contributors

Clinical Evidence is a regularly updated evidence-based journal available worldwide both as a paper version and on the internet. Clinical Evidence needs to recruit a number of new contributors. Contributors are health care professionals or epidemiologists with experience in evidence based medicine and the ability to write in a concise and structured way.

Currently, we are interested in finding contributors with an interest in the following clinical areas:

- Altitude sickness; Autism; Basal cell carcinoma; Breast feeding; Carbon monoxide poisoning; Cervical cancer; Cystic fibrosis; Ectopic pregnancy; Grief/bereavement; Haltitosis; Hodgkins disease; Infectious mononucleosis (glandular fever); Kidney stones; Malignant melanoma (metastatic); Mesothelioma; Myeloma; Ovarian cyst; Pancreatitis (acute); Pancreatitis (chronic); Polymyalgia rheumatica; Post-partum haemorrhage; Pulmonary embolism; Recurrent miscarriage; Repetitive strain injury; Scoliosis; Seasonal affective disorder; Squint; Systemic lupus erythematosus; Testicular cancer; Varicose veins; Viral meningitis; Vitiligo

However, we are always looking for others, so do not let this list discourage you.

Being a contributor involves:

- Appraising the results of literature searches (performed by our Information Specialists) to identify high quality evidence for inclusion in the journal.
- Writing to a highly structured template (about 2000–3000 words), using evidence from selected studies, within 6–8 weeks of receiving the literature search results.
- Working with Clinical Evidence Editors to ensure that the text meets rigorous epidemiological and style standards.
- Updating the text every eight months to incorporate new evidence.
- Expanding the topic to include new questions once every 12–18 months.

If you would like to become a contributor for Clinical Evidence or require more information about what this involves please send your contact details and a copy of your CV, clearly stating the clinical area you are interested in, to Claire Folkes (cfolkes@bmjgroup.com).

Call for peer reviewers

Clinical Evidence also needs to recruit a number of new peer reviewers specifically with an interest in the clinical areas stated above, and also others related to general practice. Peer reviewers are health care professionals or epidemiologists with experience in evidence based medicine. As a peer reviewer you would be asked for your views on the clinical relevance, validity, and accessibility of specific topics within the journal, and their usefulness to the intended audience (international generalists and health care professionals, possibly with limited statistical knowledge). Topics are usually 2000–3000 words in length and we would ask you to review between 2–5 topics per year. The peer review process takes place throughout the year, and our turnaround time for each review is ideally 10–14 days.

If you are interested in becoming a peer reviewer for Clinical Evidence, please complete the peer review questionnaire at www.clinicalevidence.com or contact Claire Folkes (cfolkes@bmjgroup.com).
Cigarette smoking, occupational exposure to noise, and self reported hearing difficulties

K T Palmer, M J Griffin, H E Syddall and D Coggon

*Occup Environ Med* 2004 61: 340-344
doi: 10.1136/oem.2003.009183

Updated information and services can be found at:
http://oem.bmj.com/content/61/4/340

*These include:*

**References**
This article cites 23 articles, 7 of which you can access for free at:
http://oem.bmj.com/content/61/4/340#BIBL

**Email alerting service**
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Topic Collections**
Articles on similar topics can be found in the following collections

Other exposures (1023)

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/