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The relation between snoring and smoking, body mass index, age, alcohol consumption and respiratory symptoms

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ABSTRACT: Potential risk factors for snoring were studied in a population of 457 middle-aged men. Eversnoring was reported by 60% of the men and snoring with an age of onset before or equal to 20 years by 13%. Eversnoring was significantly related to older age, higher body mass index and smoking habits. Alcohol consumption, estimated by questionnaire and gamma-glutamyl transpeptidase was unrelated to a history of snoring. Logistic regression showed that snoring was independently associated with age, body mass index and smoking habits. An exposure-effect relationship clearly appeared between tobacco consumption and snoring. After adjustment for smoking habits, none of the upper or lower respiratory symptoms was significantly related to snoring.

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There is increasing evidence that snoring might be a risk factor for various diseases, particularly cardiovascular [1–4]. Snoring is the major (and may be the only) symptom of the obstructive sleep apnoea syndrome, probably a marker of its preapnoeic stage or it may also be considered as an entity in itself. Very few reports have considered the risk factors for snoring, but recent epidemiological data obtained in a general population have shown that obesity and cigarette smoking were important risk factors for snoring [5]. In that population, total alcohol consumption was unrelated to the prevalence of snoring. The present report describes the associations of age, body mass index, smoking and drinking habits as well as respiratory symptoms with snoring in a population of 457 middle-aged men.

Subjects and methods

The 457 men of the present study conducted in 1985/86 came from a larger population of 912 men surveyed five years earlier. At the first survey, they were aged 22 to 54 years and working in a large Parisian administration, mainly as policemen. For a second survey, 599 men were selected to participate among those who performed good spirometric tracings in 1980. The primary aim of that second survey was to study factors related to hyperresponsiveness, men included were all those with a history of asthma or wheezing, any perceived hyperresponsiveness symptoms, eczema, urticaria, bronchopneumonia before 2 years of age or the PiMZ phenotype, and a similar number of control subjects chosen at random in the remaining original population [6]. At the time of the second survey, 9 were dead, 133 refused to participate or were not traced and 457 participated in the survey (76.3%). Among the latter, 390 were interviewed and 67 only answered a mail questionnaire. Subjects who answered a mail questionnaire were more often retired and older, but did not differ in other respects using data collected at the first survey. Except eczema which was less prevalent among snorers than nonsnorers (p=0.04), no other selection criteria was significantly related to snoring.

Subjects were interviewed using a modified British Medical Research Council/European Coal and Steel Community (BMRC/ECSC) questionnaire on respiratory symptoms; in 1985 additional questions were asked on loud snoring. The basis for that questionnaire was that already used and validated in a large sample of French men [7]. Snoring was assessed by the question: "Have you been told that you snore loudly?" For 88% of the men, the age of onset of snoring was recorded. Those who answered "always" were arbitrarily coded with an age of onset of 10 years. Early snorers were those with an age of onset of snoring of 20 years or less. Incidence of snoring between the two surveys and for each decade was estimated taking into account both the age of the subject and the age at onset of snoring.

The daily consumption of tobacco was calculated considering 1 cigarette equal to 1 g, a cigarillo equal to 2 g, and a cigar equal to 5 g. Subjects were then classified into neversmokers, ex-smokers (for one month at least), moderate smokers (less than 20 g of tobacco a day) and heavy smokers (20 g or more). Data on parental smoking were also recorded.

The mean daily alcohol consumption expressed in grams of pure alcohol (1 litre of 10° wine contains 80 g of pure alcohol) was assessed from the quantities of the various drinks (wine, beer, cider, liqueurs,..) and their respective alcohol content. To decrease a possible underestimation of alcohol consumption, questions were included in a larger questionnaire on any beverage. No questions on alcohol were included in the mail questionnaire used for 65 men, but all subjects answered questions on alcohol during the first survey in 1980. Data on alcohol were validated by internal consistency (alcohol consumption in 1980 and 1985 were highly correlated: r=0.65, p<0.001) and biological measurements: mean corpuscular volume (MCV) (estimated in 1980 and 1985) and gamma glutamyl transpeptidase (GGT), (assessed only in 1980). Significant correlations were observed between alcohol consumption and age-adjusted MCV at both surveys (r=0.28 and r=0.25 in 1980 and 1985 respectively; p<0.001). In 1980, significant correlations were also observed between alcohol consumption and Log_{10} GGT in 1980 (r=0.38; p<0.001). Geometric means of GGT according to alcohol consumption of 0, 1–14, 15–29, 30–44, 45–59, 60–74, and 75 g or more daily were respectively 21, 22, 24, 26, 32, 37, and 47 UI·ml^{-1} (p<0.001). Heavy drinking was defined as a daily consumption of alcohol equal to or greater than 60 g per day.

Weight and height were recorded and subjects interviewed about their weight at 20 years of age. Body mass index (BMI = weight/height^2) and BMI quintals were used in the analysis.

Statistical analysis used χ^2, Mantel-Haenszel test, analysis of variance and logistic and linear regressions [8, 9]. A value of p≤0.05 was considered significant; associations of borderline statistical significance (0.05<p≤0.10) are indicated.

Table 1. – Snoring and upper and lower airways symptoms

		Eversnoring No	Eversnoring Yes
No.		184	273
Rhinitis	(%)	19.6	27.6*
Chronic rhinitis	(%)	9.8	15.8†
Cough	(%)	8.2	13.6†
Chronic cough	(%)	3.2	6.6
Phlegm	(%)	7.7	11.8
Chronic phlegm	(%)	3.3	5.9
Wheezing	(%)	10.9	16.6
Dyspnoea grade 1	(%)	14.8	17.4
Hay fever	(%)	20.1	27.0†
Asthma	(%)	7.1	6.3
Tonsillectomy	(%)	37.1	33.9
Adenoidectomy	(%)	34.4	33.2

* p≤0.05, † p≤0.10

Results

Eversnoring (regular snoring) was reported by 60% of the men and 13% reported early snoring. Snoring was significantly related to the upper airways symptom of stuffy or runny nose (rhinitis) (table 1). For most of the other upper or airways symptoms, the prevalence was also higher among snorers than among non snorers, but never significantly (table 1).

Eversnorers were older and had higher BMI than nonsnorers (table 2). Ten-year incidence of snoring did not increase with age, with figures of 18%, 16% and 12% in the third, fourth and fifth decades respectively. The older age of snorers compared to non snorers therefore reflects that the prevalence of eversnoring was a cumulative prevalence. The association between snoring and body mass index was not restricted to an association between obesity and snoring: there was a continuum, as shown in fig. 1. Early snorers did not have higher body mass index in 1985 than those who were not, but exhibited higher body mass index at 20 years of age (p=0.08). BMI increase between 1980 and 1985 was slightly steeper among those who became snorers between 1980 and 1985 than among the neversnorers, but the difference was not statistically significant.

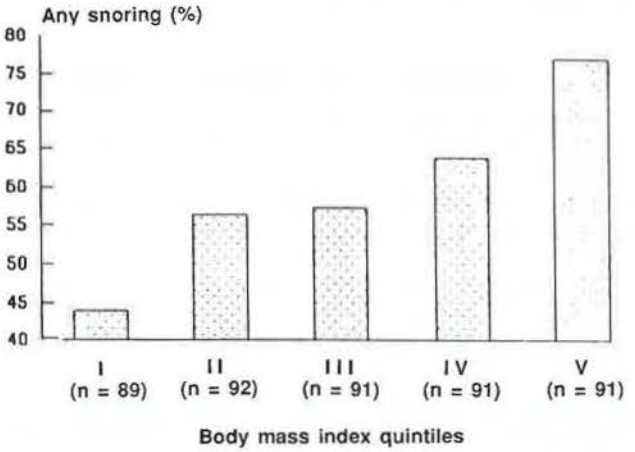

Fig. 1. Prevalence of snoring according to body mass index. P≤0.001 The quintals of body mass index corresponds to 19.5≤BMI≤23.7, BMI≤25.4, BMI≤26.6, BMI≤28.4 and BMI≤37.8. kg·m^2

Although BMI and alcohol consumption were related (r=0.14, p<0.01), eversnorers were not significantly different from non snorers for daily alcohol consumption (table 3) and heavy drinking (prevalences of snoring were 67% among the 55 heavy drinkers *vs* 60% among the 329 others, p=0.28). However, increases of MCV were observed for snorers compared to non snorers, but no association was found with GGT (table 3).

Snoring was significantly related to a higher tobacco consumption (fig. 2). Out of the 128 never or ex-smokers in 1980, 7 were current smokers in 1985. The incidence of snoring between 1980 and 1985 was higher among those who took up smoking than for the others (43% *vs* 15%, Fisher exact test p=0.09). Early snoring was unrelated to smoking habits, or to parental smoking.

Table 2. – Snoring, age and body mass index

	Eversnoring		Early snoring	
	No	Yes	No	Yes
No.	184	273	367	57
Age (years)	42.0±0.61	43.9±0.45**	43.3±0.42	42.6±0.95
Body mass index ($kg \cdot m^2$)	25.5±0.20	26.8±0.20***	26.3±0.16	27.0±0.52
Body mass index at 20 years of age ($kg \cdot m^2$)	22.6±0.15	22.8±0.15	22.7±0.12	23.3±0.35†

**p≤0.01 *** p≤0.001 † p≤0.10, mean±SD

Table 3. – Snoring and alcohol consumption

	Eversnoring		Early snoring	
	No	Yes	No	Yes
1981 data				
No.	181	271	363	57
Alcohol (g per day)	33.6±30.8	35.9±29.1	35.0±29.4	36.6±32.3
$Log_{10}GGT$ ($IU \cdot l^{-1}$)	1.421±0.305	1.446±0.303	1.426±0.288	1.485±0.381
Age-adjusted MCV (μ^3)	89.8±4.7	90.0±4.1	89.7±4.3	91.0±4.6*
1986 data				
No.	151	233	315	46*
Alcohol (g per day)	28.0±26.2	32.1±30.5	30.0±27.7	36.0±33.8
Age-adjusted MCV (μ^3)	93.9±4.0	94.7±4.2†	94.2±4.1	95.4±4.5†

MCV: mean corpuscular volume, GGT: gamma glutamyl transpeptidase, * p≤0.05, † p≤0.10, mean±SD

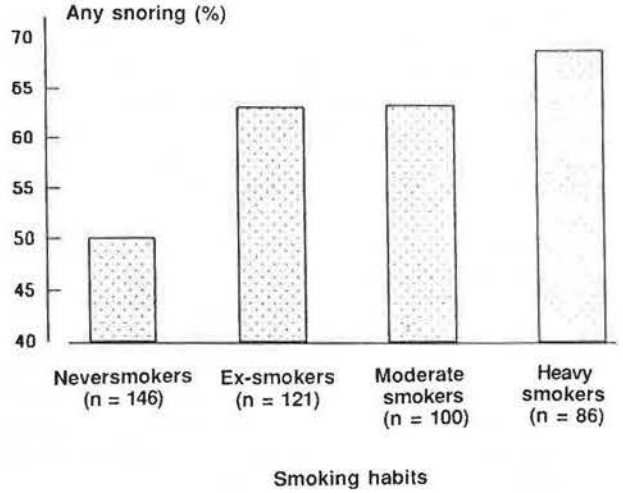

Fig. 2. Prevalence of snoring according to smoking habits. χ^2, p=0.02; χ^2 for trend p=0.005.

As expected, BMI increased markedly with age, current smoking was related to lower BMI and ex-smoking to older age and higher BMI. The real effect of smoking might be therefore underestimated by univariate analysis. Furthermore, rhinitis and other respiratory symptoms were related to smoking habits. Therefore, a multiple logistic regression was performed, with snoring as the dependent variable, to assess the respective effects of age, body mass index, symptoms and smoking habits (table 4). Adjusted for age and smoking, the odds-ratio of snoring for those in the upper quintal of BMI *vs* those in the first was almost 5, but also almost 2 for those in the second and third quintals. Independent of age and BMI, the effect of smoking is clearly shown, with increasing odds-ratio when tobacco consumption increased. The prevalence of snoring was significantly higher among ex-smokers than among neversmokers. After adjustment for smoking, none of the upper or lower respiratory symptoms was significantly related to snoring. The relation of

smoking habits with snoring was similar among obese (men in the upper quintal of BMI) than in non obese subjects. A multiple logistic regression (with age, body mass index quintals and duration since quitting smoking) conducted among ex-smokers showed an odds-ratio of 0.53 for those who stop smoking for more than 4 years compared to the other ex-smokers, but it was not significantly different from 1.

Table 4. – Factors related to eversnoring. Logistic regression among 450 men with 269 snorers

	Odds-ratio	p value
Age (years)	1.33†	*
Body mass index		
Second quintial *vs.* first	1.89	*
Third quintal *vs.* first	1.94	*
Fourth quintal *vs.* first	2.59	**
Fifth quintal *vs.* first	4.82	***
Smoking habits		
Ex-smokers *vs.* neversmokers	1.67	*
Moderate smokers *vs.* neversmokers	2.10	**
Heavy smokers *vs.* neversmokers	2.89	***

Odds-ratios were estimated from the exponential of the logistic regression coefficients. † per 10 years; OR=exp (10·0.0288)=1.33; * $p \leq 0.05$, ** $p \leq 0.01$; *** $p < 0.001$. None of the upper or lower respiratory symptoms included in the model was related ($p > 0.05$) to snoring.

Discussion

A history of snoring appeared to be very prevalent (60% in this population of middle-aged men) and significantly related to smoking, but not to drinking habits. Besides a relation between obesity and snoring, the prevalence of snoring increased with body mass index.

The prevalence of snoring is consistent with other reports [1, 3, 4], when considering the differences in the questionnaires used. Standardization of the questions on snoring is therefore warranted for epidemiological studies, as it has been previously for respiratory symptoms. Standardization would be particularly useful for defining a degree of snoring, based on chronicity, intensity, and so on. Early snoring prevalence (13%) retrospectively assessed appeared similar to that observed among French draftees aged 17–22 years (14%) [7]. This gives some validation to the retrospective assessment of ten-year incidence. Most studies also observed increasing prevalence in the age range of our population [5, 9]. The incidence of snoring was roughly the same in the third, fourth or fifth decade. It appears that the older age of snorers compared to non snorers reflects that the prevalence of snoring recorded at the time of the survey is a cumulative prevalence, and not a point-prevalence (which could be assessed by a question such as; last night (week),...).

The proportion of obstructive sleep apnoea cannot be adequately assessed in our survey. However, various estimations of obstructive sleep apnoea give prevalences between 1 and 10% [10]. In our study, all snorers were invited to answer the question on sleep apnoea, which gave a prevalence in the whole group of 4%. Although this type of question has not been validated yet, the prevalence is coherent with other studies including polysomnographic recordings. It is therefore likely that the great majority of the eversnorers studied here did not have obstructive sleep apnoea.

Our results extend the previous observations of NORTON *et al.* [3] of an increasing proportion of smokers with snoring grade as well as of the study of BLOOM *et al.* [5]. After adjustment for age and body mass index, the present study clearly shows an exposure-effect relationship. Ex-smokers were in an intermediate position between neversmokers and moderate smokers. The prevalence of snoring among ex-smokers was significantly higher than among neversmokers. Our results extend those of Bloom *et al.* who did not observe a difference between ex-smokers and neversmokers. However, they observed an association of borderline significance between duration since quitting smoking and snoring, with a prevalence of heavy snoring among ex-smokers for 4 years at least similar to that of neversmokers. In our study, no definite relation with time since quitting was shown, although some trend was observed. However, whether snoring is reversible when a smoker quits needs follow-up studies. Smoking habits, but not rhinitis, remained significantly associated with snoring, when including both in a logistic regression. Therefore, rhinitis does not appear to be the explanation of the relation between smoking and snoring, partial nocturnal upper airway obstruction. This result is coherent with observations of the pharynx as the predominant site of the obstruction leading to snoring [11]. Therefore, it is likely that smoking exerts deleterious effects on all the lower and upper airways, including the pharynx. Smoking might induce snoring by increasing upper airway resistance at that level [12]. At variance with BLOOM *et al.* [5], we did not observe significant associations between respiratory symptoms and snoring, after adjustment for smoking. Discrepancies might be due either to the smaller size of our sample or to difference in the definition of snoring.

The lack of association with drinking habits supports the observations of BLOOM *et al.* [5] in their population-based study in which total alcohol consumption was not related to snoring. Special attention was given to validate our assessment of alcohol consumption with biological markers, and we observed the expected correlations of alcohol with GGT and MCV [13]. Among the various variables considered, only an association of snoring with MCV was observed. We observed an unexpected increase in MCV between surveys, unexplained by variation in age, smoking or alcohol, which might be related to technical problems, as both measurements were not done in the laboratory. However, this increase in MCV between surveys would be unlikely to bias associations of MCV with alcohol or snoring. Despite the lack of association of total alcohol consumption with snoring, BLOOM *et al* [5] observed that snoring was more prevalent among those who used alcohol or medications as aids to sleep (considered

together) than among those who did not, which is consistent with a possible role of bedtime alcohol. Acute effects of alcohol on snoring on the following night have been clearly demonstrated [14]. However, both epidemiological studies suggest that the total usual consumption of alcoholic beverages does not seem to be a risk factor for snoring. Further epidemiological studies should consider the hour of alcohol intake.

We confirmed the well-known associations of smoking with lower weight [15]. The association of snoring with obesity is also established [3] and we confirmed here that the odds-ratio associated with the upper quintal versus the lowest of body mass index (which corresponds here at a BMI of>28.4 $kg \cdot m^2$) was nearly 5. However, men in the second quintal exhibited an odds-ratio of nearly 2 compared to those in the lowest quintal. As both blood pressure and snoring are continuously related to BMI, studies looking at the potential effect of snoring on blood pressure should adjust on BMI on a quantitative basis and not only on obesity [16].

The consistency of our observations is supported by the association of early snoring with BMI at 20 years. Furthermore, it is coherent that we did not observe an association with active smoking, as it usually began later, especially in the group of men studied. No association was observed between early snoring and parental smoking. However, large tonsils or adenoids cause snoring in childhood [17,18] and parental smoking was related to tonsillectomy and/or adenoidectomy in a study conducted in children [19]. The lack of association of early snoring with parental smoking in this study might be due to the low prevalence of maternal smoking in our group (5%) and the retrospective assessment of the data. Studies in children would better assess this potential hazard of smoking.

From these results the potential deleterious cardiopulmonary effects of snoring cannot be assessed, but it appears clearly that snoring is another consequence of smoking habits. The benefits of diet among overweight subjects and quitting smoking are clearly demonstrated for a number of diseases. The potential usefulness of such advice for the (at least) socially annoying symptom of snoring might be another argument to be used in daily practice.

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References

1. Lugaresi E, Cirignotta F, Coccagna G, Montagna P, Zucconi M, Mondini S. – Sleep related respiratory impairment. *Bull Europ Physiopathol Respir*, 1982, 18, 187–198.
2. Block AJ. – Is snoring a risk factor? *Chest*, 1981, 80, 525–256.
3. Norton PG, Dunn EV. – Snoring as a risk factor for disease: an epidemiological survey. *Br Med J*, 1985, 291, 630–632
4. Partinen M, Palomäki H. – Snoring and cerebral infartion. *Lancet*, 1985, ii, 1325–1326.
5. Bloom JW, Kaltenborn WT, Quan SF. – Risk factors in a general population for snoring. Importance of cigarette smoking and obesity. *Chest*, 1988, 93, 678–683.
6. Kauffmann F, Neukirch F, Annesi I, Korobaeff M, Doré MF, Lellouch J. – Relation of perceived nasal and bronchial hyperresponsiveness to FEV_1, basophil counts, and methacholine response. *Thorax*, 1988, 43, 456–461.
7. Billiard M, Alpérovitch A, Perot C, Jammes A. – Excessive daytime somnolence in young men : prevalence and contributing factors. *Sleep*, 1987, 10, 297–305.
8. Snedecor GW, Cochran WG. – Statistical methods. Ames: The Iowa State University Press 1967, pp 381–418, 484.
9. Cox DR. – The analysis of binary data. Methuen &Co, London, 1969.
10. Krieger J. Les syndrômes d'apnées du sommeil de l'adulte. *Bull Eur Physiopathol Respir*, 1986, 22, 147–189.
11. Bradley TD, Brown IG, Grossman RF *et al.* – Pharyngeal size in snorers, nonsnorers, and patients with obstructive sleep apnoea. *N Engl J Med*, 1986, 315, 1327–1331.
12. Issa FG, Sullivan CE. – Upper airway closing pressures in snorers. *J Appl Physiol*, 1984, 57, 528–535.
13. Papoz L, Warnet JM, Péquignot G, Eschwege E, Claude JR, Schwartz D. – Alcohol consumption in a healthy population. Relationship of glutamyl transferase activity and mean corpuscular volume. *JAMA*, 1981, 245, 1748–1751.
14. Issa FG, Sullivan CE. – Alcohol, snoring and sleep apnoea. *J Neurol Neurosurg Psychiatry*, 1982, 45, 353–359.
15. Higgins MW, Kjelsberg M. – Characteristics of smokers and nonsmokers in Tecumseh, Michigan. II The distribution of selected physical measurements and physiologic variables and the prevalence of certain diseases in smokers and nonsmokers. *Am J Epidemiol*, 1967, 86, 60–77.
16. Gislason T, Aberg H, Taube A. – Snoring and systemic hypertension - An epidemiological study. *Acta Med Scand* 1987, 222, 415–421.
17. Robin IG. – Snoring. *Proc Roy Soc Med*, 1968, 61, 13–20.
18. Mauer KM, Staats BA, Olsen KD. – Upper airway obstruction and disordered nocturnal breathing in children. *Mayo Clin Proc*, 1983, 58, 349–353.
19. Said G, Zalokar J, Lellouch J, Patois E. – Parental smoking related to adenoidectomy and tonsillectomy in children. *J Epidemiol Commun Health*, 1978, 32, 97–101.

Relation entre ronflement, tabagisme, index de masse corporelle age, consommation d'alcool et symptômes respiratoires. F. Kauffmann, I. Annesi, F. Neukirch, M. P. Oryszczyn, A. Alpérovitch.

RÉSUMÉ: Des facteurs de risque du ronflement ont été recherchés dans une population de 457 hommes. Plus de 60% des hommes ont rapporté avoir ronflé toute leur vie et 13% dès l'âge de 20 ans. Le ronflement était significativement lié à un âge plus avancé, un index d'obésité plus élevé et à la consommation de tabac. La consommation d'alcool, estimée par questionnaire et par le taux de gamma glutamyl transpeptidase, n'était pas liée au ronflement. Une régression logistique a montré que le ronflement était associé indépendamment à l'âge, à l'index d'obésité et au tabagisme. Une relation dose effet a été mise en évidence entre la consommation de tabac et la prévalence du ronflement. Après ajustement sur le tabagisme, aucun des symptômes des voies respiratoires supérieures ou inférieures n'était significativement lié au ronflement.

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