Occurrence of leukoplakia and some other oral white lesions among 20 333 adult Swedish people

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Abstract – Among 20 333 people aged 15 yr or above, the prevalences of oral white lesions were calculated based on a partly new classification. The total prevalences were: cheek and lip biting 5.1%, smoker's palate 1.1%, frictional white lesion 5.5%, snuff dipper's lesion 8.0%, preleukoplakia 6.4% and leukoplakia 3.6%. If all these lesions were pooled, the prevalence was 24.8% and if only the entities cheek and lip biting and smoker's palate were excluded it became 20.1%. If weak "preleukoplakic" lesions were excluded from the latter figure the prevalence for marked whitish lesions was 13.8%. Etiologic and clinical subgroups of leukoplakia showed the following prevalences: using the etiologic subgroups, idiopathic leukoplakia 0.7% and tobacco-associated leukoplakia 2.9%; using the clinical subgroups, homogeneous leukoplakia 3.5% and non-homogeneous leukoplakia 0.3%. The intraoral location pattern of leukoplakias was preponderant in the commissural and buccal areas. However, the idiopathic leukoplakias showed a somewhat more even distribution and thus a more similar distribution to that of oral cancer.

Key words: cheek and lip biting; epidemiology, oral; frictional white lesions; leukoplakia; preleukoplakia; snuff dipper's lesion; smoking; smoker's palate; tobacco

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Leukoplakia is the most prevalent precancerous lesion of the oral mucosa. The label "leukoplakia" was coined by SCHWIMMER in 1877 (1) and was for a long time thereafter an ambiguous concept. Thus, various authors over the years have used it as a histologic as well as a clinical concept, leading to misunderstandings and misinterpretation of results (2). However, during the last decades it has been widely accepted that leukoplakia is a term assigned to clinical lesions and the concept bears no histologic connotations (3). This opinion has also been adopted by WHO Collaborating Centre for Oral Precancerous Lesions, which in 1978 proposed that leukoplakia be "defined as a white patch or plaque that cannot be characterized clinically or pathologically as any other disease" (4). However, this delimitation of the concept leukoplakia could also lead to misunderstandings. According to the WHO publication ICD-DA (5) "smoker's palate" and "cheek and lip biting" are clinical entities and should then accordingly not be considered leukoplakias, while similar white lesions, caused by friction or other trauma, are included in the leukoplakic group of lesions. This is unfortunate, since such lesions have hardly any premalignant potential. Further, the minimum degree of whiteness required to define a leukoplakia is a matter of arbitrary decision. These circumstances have been considered in some previous prevalence studies (6–8), and thoroughly discussed at an international seminar on oral leukoplakia and its classification (9). At that meeting it was demonstrated how the prevalence of oral leukoplakia could vary between 0.7 and 24.8% in the same population just by using varying clinical criteria.

The aim of the present publication was to compile data on the occurrence of leukoplakia and some other oral white lesions collected in an investigation reported on by AXÉLL (6). The guidelines on definitions and classification from the above-mentioned international seminar will be used.

Material

A total of 20 333 people were clinically examined in a survey undertaken in 1973–74 in the central part of Sweden. The chosen area comprised, by Swedish standards, a medium-sized city, a suburb (of Stockholm) and a rural area. Those examined were people aged 15 yrs or above representing the total population of the area, 30 118 individuals, all of whom were summoned to a primary and later to a secondary investigation. The primary investigation was undertaken in cooperation with a public health screening organization. At this primary investigation 18 659, or 62%, attended. The non-participants were the subject of a secondary investigation for which every fifth individual, i.e. 2292 individuals, was sampled and to which 1674 attended. Thus, in total, 20 333 persons were examined, representing, from the standpoint of frequency of participation, 89.7% of the total adult population in the investigated area. Table 1 gives the distributions of the total and the examined populations with respect to age and sex and the frequency of participation, taking into account the fact that the numbers of individuals examined in the secondary inves-

Table 1. Number of people in total population and number and proportion of examined individuals. Proportion figures from secondary investigation are weighed by a factor of five, the sampling proportion. Distribution according to age and sex

Age, yr	No. of total population			No. of examined people			Proportion examined, percent		
	Men	Women	Total	Men	Women	Total	Men	Women	Total
15–24	3009	3021	6030	1757	1907	3664	86	86	86
25–34	3441	3120	6561	2230	2297	4527	86	90	88
35–44	2253	2078	4331	1594	1600	3194	89	95	92
45–54	2137	2106	4243	1565	1599	3164	98	96	97
55–64	2042	1923	3965	1476	1375	2851	92	94	93
65–74	1325	1386	2711	881	938	1819	86	91	88
75–	1022	1255	2277	553	581	1114	80	85	83
Total	15229	14889	30118	10036	10297	20333	89	91	90

tigation are weighed by a factor of five, i.e. a factor corresponding to the sampling proportion. The most frequent reasons for final non-participation were "change of residence" (5.1%), "no contact" (1.6%), "recently dead" (1.4%) and "refusal" (1.4%). The clinical examination was undertaken in artificial light using mouth mirrors and tongue depressors. Lesions were recorded on pretyped forms and color photographs were taken. From 327 oral white lesions (about 8% of the total number) biopsies were taken at random for validity evaluation and when there was doubt as to the diagnosis. Information on tobacco habits was collected on questionnaires, which were filled in with the assistance of trained personnel. Clinical criteria for the lesions discussed in this study are given below.

Definition of lesions

Smoker's palate

The palatal mucosa is whitish and shows multiple nodules with red, centric dots and/or fissuring. The patient is a habitual smoker.

Cheek and lip biting

A rough, macerated surface with irregular, flaky desquamation. The lesion is diffusely outlined and situated where self-infliction by chewing is possible.

Frictional white lesion

A whitish or grayish patch, the location of which corresponds to the site for a readily recognizable physical trauma.

Snuff dipper's lesion

On the site for the regular placing of snuff, there is a wrinkling of the mucosa and/or a whitish or yellowish brown discoloration. The lesion is subdivided into four clinical stages (degrees 1–4).

Leukoplakia

A whitish patch or plaque that cannot be characterized as any other disease and which is not associated with any physical or chemical causative agent except the use of tobacco. Leukoplakia is subdivided according to etiological and clinical factors as follows.

Idiopathic (cryptogenic) leukoplakia – No etiology for the patch can be found.

Tobacco-associated leukoplakia – The patch is associated with and thought to be the result of the use of tobacco. "Smoker's palate" and "snuff dipper's lesion" are listed separately.

Homogeneous leukoplakia – A uniformly whitish lesion with a smooth or corrugated surface.

Non-homogeneous leukoplakia – The lesion includes red areas (erythroleukoplakia).

Preleukoplakia – A grayish area with indistinct boundaries blending into a normal mucosa. The lesion cannot be diagnosed as any other disease.

Statistical methods

The prevalence for one demographic group was calculated according to the formula

$$p=\frac{1}{N}(x_1+\frac{M_2}{m_2}\cdot x_2),$$

where p = prevalence, N = total number of individuals, x_1 = number of individuals with a lesion in the primary investigation, x_2 = number of individuals with a lesion in the secondary investigation, M_2 = number of non-participants in the primary investigation, m_2 = number of examined individuals among primary nonparticipants (M_2).

The prevalences from 14 various demographic groups were then weighed to give prevalence figures for age and sex strata as well as for the total population. Standard errors for the various groups were calculated by a similar procedure.

When testing prevalence differences between various demographic groups, two methods were used. When the prevalences equaled or exceeded 2% the following formula was used.

$$z=\frac{p_M-p_F}{\sqrt{SE(p_M)^2+SE(p_F)^2}}$$

When the prevalences were lower than 2% the differences were tested by means of chi-square tests or, if necessary, Fisher's exact test.

For detailed information concerning population, investigatory procedures, and statistical methods, see AXELL (6) pp. 15–44. Also taken into account in that report are validity and reliability pro-

Table 2. Prevalence (percent) of oral white lesions. Step-by-step exclusion of some clinical entities

Entity			Men	Women
All oral white lesions discussed	24.8		36.5	13.2
	Included	Excluded	Men	Women
Cheek and lip biting, smoker's palate	24.8	20.1	32.2	8.1
Frictional white lesion	20.1	15.7	27.8	3.6
Snuff dipper's lesion	15.7	8.5	13.6	3.4
Preleukoplakia	8.5	3.6	6.1	1.2
Tobacco-associated leukoplakia	3.6	0.7	1.0	0.4
Idiopathic leukoplakia	0.7	–	–	–

Table 3. Prevalence (percent) of cheek and lip biting, ICD-DA code 528.93, and smoker's palate, ICD-DA code 528.72. Distribution according to age and sex

Age, yr	Cheek and lip biting			Smoker's palate		
	Men	Women	Total	Men	Women	Total
15–24	8.3	14.7	1.4	1.2	–	0.6
25–34	6.9	5.5	6.2	2.2	–	1.1
35–44	3.7	4.9	4.3	2.8	0.6	1.8
45–54	3.4	2.5	3.0	3.9	0.1	2.0
55–64	3.3	1.2	2.2	2.5	–	1.3
65–74	2.1	0.4	1.2	1.1	–	0.5
75–	0.3	1.3	0.9	0.2	–	0.1
Total	4.9	5.5	5.1	2.1	0.1	1.1

Table 4. Prevalence (percent) of frictional white lesion and snuff dipper's lesion. Distribution according to age and sex

Age, yr	Frictional white lesion			Snuff dipper's lesion		
	Men	Women	Total	Men	Women	Total
15–24	4.6	3.4	4.0	14.8	–	7.5
25–34	6.8	3.9	5.4	15.8	0.1	8.3
35–44	7.8	4.6	6.2	10.1	–	5.1
45–54	6.1	4.4	5.0	11.9	0.3	6.2
55–64	7.0	6.9	7.0	10.6	–	9.9
65–74	7.4	5.7	6.5	21.4	0.6	10.3
75–	3.7	5.7	4.9	26.6	1.0	11.8
Total	6.0	4.7	5.5	15.9	0.2	8.0

cedures such as inter- and intraexaminer variability, comparison between clinical and histologic data, and errors in data handling. For estimating reliability of information on tobacco habits subjects were requestioned. The results are published in an article on leukoedema by AXÉLL & HENRICSSON (10).

Results

The total prevalence of all whitish lesions for which clinical criteria are given in this study was 24.8%; 36.5 and 13.2% for men and women, respectively, a difference that is statistically highly significant ($P<0.001$). Table 2 describes how the prevalence figures are gradually reduced when the different clinical entities or diagnostic labels are excluded one by one. The lowest prevalence figure, 0.7%, is valid for idiopathic leukoplakia.

For the entities "cheek and lip biting" and "smoker's palate", classified with specific code numbers in ICD-DA (5), prevalence figures are given in Table 3. In the present study two other lesions are handled analogously, i.e. considered separate clinical entities. They are "frictional white lesion" and "snuff dipper's lesion". The prevalence are 5.5% and 8.0%, respectively. The distributions according to age and sex are shown in Table 4. Frictional white lesions are more prevalent among men than among women ($P<0.01$) and the prevalence of snuff dipper's lesion shows a highly significant difference between the sexes ($P<0.001$).

When the four above-mentioned entities are excluded from the total number of oral white lesions, the remaining lesions are preleukoplakia and leukoplakia lesions, with a prevalence of 6.4% and 3.6%, respectively, and 8.5% together. For both diagnoses there is a highly significant preponderance of men ($P<0.001$). The distributions according to age and sex are shown in Table 5. If a similar dividing up of lesions, based on grade of whiteness, is done for frictional white lesion and snuff dipper's lesion, the prevalences are 1.3% for each "pre"-lesion and 4.3% and 6.7%, respectively, for the more marked lesions. If, for frictional white lesion, snuff dipper's lesion and leukoplakia all "pre"-lesions are pooled, the prevalence is 8.8%, 13.7% for men and 4.1% for women. The pooled prevalence for the more marked lesions is 13.8%, 22.9% for men and 4.8% for women. As shown in Table 2 the prevalence of "pre"-lesions combined with the more marked lesions is 20.1%, 32.2% for men and 8.1% for women.

Leukoplakia has been subdivided in two ways, according to etiologic and to clinical factors. If subdivided etiologically, the prevalence for idiopathic leukoplakia is 0.7% and for tobacco-associated leukoplakia 2.9%. If subdivided clinically, the prevalence for homogeneous leukoplakia is 3.5% and for non-homogeneous leukoplakia 0.3%. The distributions according to age and sex are shown in Table 6. While tobacco-associated and homogeneous leukoplakias are much more prevalent among men than among women ($P<0.001$) there are no statistically significant differences between the sexes regarding the prevalences of idiopathic or non-homogeneous leukoplakias ($P>0.05$).

The distributions according to some intraoral locations for leukoplakia and its subdivisions are shown in Tables 7 and 8. The most frequent location for leukoplakia is the commissure and/or the buccal mucosa. This is especially true for the non-homogeneous leukoplakia. A somewhat different pattern is seen for the idiopathic leukoplakia, which is rela-

Table 5. Prevalence (percent) of preleukoplakia, leukoplakia and of either one of the lesions. Distribution according to age and sex

Age, yr	Preleukoplakia			Leukoplakia			Preleukoplakia or leukoplakia		
	Men	Women	Total	Men	Women	Total	Men	Women	Total
15–24	3.8	0.9	2.4	0.9	0.4	0.6	4.2	1.2	2.7
25–34	7.4	1.6	4.6	3.7	0.3	2.1	8.4	1.8	5.8
35–44	10.5	4.5	7.6	8.1	2.5	5.5	16.0	6.0	11.1
45–54	14.8	3.7	9.3	9.2	1.7	5.5	19.2	5.0	12.2
55–64	14.9	4.1	9.6	10.3	2.0	6.2	21.1	5.3	13.3
65–74	15.4	3.0	8.9	6.7	1.5	4.0	18.8	3.9	11.0
75–	9.3	2.4	5.0	9.4	0.2	4.1	16.4	2.6	8.4
Total	10.0	2.7	6.4	6.1	1.2	3.6	13.6	3.4	8.5

Table 6. Prevalence (percent) of leukoplakia subdivided etiologically and clinically. Distribution according to age and sex

Age, yr	Etiological subdivision						Clinical subdivision					
	Idiopathic leukoplakia			Tobacco-associated leukoplakia			Homogeneous leukoplakia			Non-homogeneous leukoplakia		
	Men	Women	Total	Men	Women	Total	Men	Women	Total	Men	Women	Total
15–24	0.1	-	0.1	0.8	0.4	0.6	0.9	0.4	0.6	-	-	-
25–34	0.1	0.1	0.1	3.5	0.3	2.0	3.7	0.3	2.1	0.1	0.1	0.1
35–44	1.1	0.1	0.6	7.1	2.4	4.8	8.1	2.4	5.3	0.2	0.1	0.1
45–54	1.1	0.5	0.8	8.0	1.2	4.7	8.5	1.7	5.2	1.2	0.1	0.7
55–64	2.1	1.1	1.6	8.2	1.0	4.7	10.2	2.0	6.2	1.1	0.4	0.8
65–74	1.1	1.4	1.3	5.7	0.1	2.8	6.6	1.5	4.0	0.7	–	0.3
75–	3.7	0.2	1.6	5.7	0.1	2.4	9.4	0.2	4.1	0.1	–	0.1
Total	1.0	0.4	0.7	5.1	0.8	2.9	6.0	1.1	3.5	0.4	0.1	0.3

Table 7. Leukoplakia distributed according to some oral locations. Proportions (percent) in each location out of total number of recorded locations with lesions

Diagnosis	Total no. of locations	Commissure	Buccal mucosa	Bucca or commissure	Lip mucosa	Vestibulum	Alveolar ridge	Floor of mouth	Tongue	Palate
Leukoplakia	1607	50.7	38.6	89.3	1.7	0.5	2.2	0.8	1.1	0.5
Idiopathic	217	44.2	29.0	73.2	1.4	2.3	4.1	1.4	5.5	1.8
Tobacco-associated	1390	51.7	40.2	91.9	1.8	0.3	1.9	0.7	0.4	0.3
Homogeneous	1536	49.4	39.6	89.0	1.8	0.6	2.3	0.8	1.1	0.5
Non-homogeneous	71	78.9	18.3	97.2	1.4	-	-	-	1.4	-

Table 8. Leukoplakia distributed according to some oral locations. Proportions (percent) in each location out of total number of recorded people with lesions

Diagnosis	Total no. of people with lesion(s)	Commissure	Buccal mucosa	Bucca or commissure	Lip mucosa	Vestibulum	Alveolar ridge	Floor of mouth	Tongue	Palate
Leukoplakia	705	85.0	64.3	89.5	3.7	1.1	3.0	1.3	1.4	0.9
Idiopathic	122	63.1	41.0	68.9	2.5	3.3	5.7	0.8	6.6	2.5
Tobacco-associated	583	89.5	69.1	93.8	4.0	0.7	2.4	1.4	0.3	0.5
Homogeneous	693	81.5	64.8	89.3	3.6	1.2	3.0	1.3	1.4	0.9
Non-homogeneous	52	90.4	23.1	98.1	1.9	-	-	-	1.4	-

tively more evenly distributed in the oral cavity than other subgroups of leukoplakia.

Discussion

Oral white lesions reflect many different diseases and pathological changes. Some of them relate to diseases such as lichen planus and lupus erythematosus. Others are local changes with a clearly defined or highly probable etiology. This is emphasized in the WHO publication ICD-DA (5) where they are given separate code numbers. Cheek and lip biting and smoker's palate are such examples.

Cheek and lip biting, which also includes similar changes at the margins of the tongue, shows prevalence figures which are about 10 times as high as those found by SEWERIN (11). He reported a prevalence of 0.49% among 8589 patients of all ages seen at the Royal Dental College in Copenhagen. The large discrepancy is probably related to the use of dissimilar clinical criteria. In the present study very subtle lesions without erythema and ulcerations were included.

Smoker's palate is used as an alternative term for leukokeratosis nicotina palati and thought to be more adequate, since the lesion has little to do with nicotine, but rather with other components of smoke or smoking. The present finding of a prevalence of about 1% is in accordance with previous findings in various populations. PINDBORG *et al.* (12–14) reported 0.15–0.71% in three Indian populations examined at dental schools and RAMANATHAN *et al.* (15) 1.5% in Malaysia. SAIETZ (16) investigated 3819 Danes aged 15 yr and above. She used a two grade clinical scale and found 1.4% with smoker's palate when criteria comparable to those presently used were applied.

Neither cheek and lip biting nor smoker's palate are to be considered precancerous lesions. It should be emphasized that the diagnosis of smoker's palate should not be used for those palatal lesions seen in reverse smokers. Such lesions should be regarded as precancerous (17).

If frictional white lesions and snuff dipper's lesions are included among the leukoplakias, the prevalence of leukoplakia, provided subtle "pre"-lesions are excluded, is 13.8%. Compared to prevalence figures in various studies this is rather high. In agreement with the conclusions and recommendations from the international meeting in Malmö (9) frictional white lesions have been handled separately in this study. This is also justified by the opinion that frictional lesions,

as opposed to leukoplakia, are not intrinsically precancerous. In a previous study (6) the label frictional keratosis has been used. This is somewhat inadequate since the term keratosis should preferably be reserved for microscopic findings in histologic specimens.

Snuff dipper's lesions could be classified as tobacco-associated leukoplakia. However, in the present study they have been handled separately. The reason for this is manifold. Snuff dipper's lesion is a well defined clinical entity with a specific etiology. The changes are frequently yellowish or brownish, and in many cases there is a lesion of the mucosa without any obvious change of color, making the clinical description leukoplakia ("white patch") somewhat inappropriate. The precancerous potential of leukoplakia is well documented in several reports. About 4% of leukoplakias in Scandinavia develop a carcinoma within a 10–20 year period (18, 19). The precancerous nature of snuff dipper's lesion is more doubtful. Among 200000 male snuff dippers in Sweden one case a year of oral carcinoma may be associated with snuff dipping (20).

In Sweden snuff dipper's lesion is almost exclusively found among men. Detailed information on snuff dipping habits and related lesions will be published separately. It should be emphasized that the present study was undertaken 1973–74 and that snuff dipping habits have changed considerably during the last decade. Thus, there has been an increase every year of sales figures and the habit seems to have increased among Swedish women.

In this study the label leukoplakia is used as a clinical term for whitish lesions which are not associated with a physical chemical causative agent except tobacco smoking. The minimal degree of "whiteness" demanded to separate it from preleukoplakia is indicated by color illustrations in a previous publication (6). The prevalence of leukoplakia is in good agreement with the finding by BRUSZT from Hungary of 3.6% in the only previous European study in a general population (21). Most other studies are from India, showing prevalences of 0.2–4.9% (2). The finding of a higher prevalence for preleukoplakia than for leukoplakia is in accordance with previous studies (8, 22). The age and sex distributions for preleukoplakia are very similar to those for leukoplakia.

Idiopathic leukoplakias differ in various ways from the tobacco-associated ones. The prevalence difference between the sexes is not so pronounced for the idiopathic leukoplakia and, further, the intraoral distribution is more even for that category. Concerning these parameters, idiopathic leukoplakias are more similar to what can be found from data on the occurrence of oral cancer in Sweden (23). This might support previous reports of a higher frequency of malignant development among idiopathic leukoplakias than tobacco-associated leukoplakias (18). On the other hand, they constitute only about 15% of the total number of leukoplakias and might therefore be related to a smaller number of oral cancers than tobacco-associated leukoplakias.

Non-homogeneous leukoplakias are considered to carry a considerably higher risk for malignant change than homogeneous ones (24–26). In the present study 97% of the lesions were located on the commissure or the buccal mucosa, locations which are unusual for the development of oral cancer in Sweden (23). However, they make up less than 5% of all leukoplakias and thus, even if they relatively frequently develop into oral cancer, might not show up in the total epidemiologic material.

The overall distribution of leukoplakia shows the same pattern as has been shown in many previous studies (27, 28). However, the overrepresentation of lesions in the commissures and the buccal mucosa seems to be a little more pronounced in the present study. Such somewhat varying patterns might be due to

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differences in smoking habits. Thus, commissural and buccal lesions are most frequently seen among tobacco-associated and/or non-homogeneous leukoplakias. In the present study the latter are almost exclusively found among smokers.

In this study an attempt has been made to present epidemiologic data on oral white lesions so that relevant comparisons with other investigations may be accomplished. Further, the lesions have been defined and categorized to fulfil requirements for follow-up investigations, especially on precancerous lesions.

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