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A prevalence study of

ORAL MUCOSAL LESIONS

in an adult Swedish population

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INTRODUCTION

Epidemiologic prevalence studies primarily "describe the amount of disorder existing in a population at a particular time" (Susser 1973). Basic information of this kind can "provide guidance in the administration of health services", it may be utilized to "explain local disease occurrence", and eventually contribute to the understanding of "the natural history of a disease" (MacMahon and Pugh 1970).

To what extent these positive values can be approached in any particular prevalence study, depends on a wide range of given, or else controllable, factors. For instance, diseases having a short duration are difficult to account for. The occurrence of such diseases is often better elucidated in incidence studies, which describe the frequency of diseases met with during a defined period of time. The majority of oral mucosal lesions are of chronic nature, however, and a prevalence study can therefore be expected to yield satisfactory information. Uncommon diseases are often difficult to survey properly, and this is especially true if prevalences in a general population are looked for. As many oral mucosal lesions are relatively uncommon, an epidemiologic prevalence study including such lesions should preferably comprise a large population. Practical difficulties may thus be an explanation why such studies on large general populations are uncommonly reported. Besides, those available are usually restricted to but a few types of lesions.

SOME ASPECTS ON PREVALENCE STUDIES OF ORAL MUCOSAL LESIONS

The present investigation was performed to assess prevalences of oral mucosal lesions in a Swedish population. Although matching studies in Sweden or in other countries are infrequent, there are many reports from where information on prevalences of oral mucosal lesions can be obtained. It should be noted that these reports describe investigations which differ in important respects from each other. In addition, many investigations are incomplete or inadequately delineated, which makes a coherent literature review and detailed comparisons hazardous. Alternatively therefore, some of the factors that should be observed when appraising the results of, as well as planning prevalence studies of oral mucosal lesions will be briefly reviewed and discussed.

On populations

Two different types of populations, general or special, can be *selected* for investigation. In this report a *general population* is defined as a population, that consists of all individuals in a chosen geographic region or of complete age strata within that region. A *special population* is defined as a group of individuals which in some way is selfselected.

Information on prevalences obtained from investigations of special populations

can not be generalized to the same extent as that obtained from general populations. However, some special populations offer better possibilities in this respect than others.

Schoolchildren often represent rather complete age strata within a region and investigations on this kind of population can thus give prevalence figures that are nearly in agreement with findings in a general population. More limited possibilities in this respect are presented by studies performed among *college students* who usually are highly selected.

Among *outpatients* at different clinics, lesions associated with subjective symptoms tend to be overrepresented. Patients suffering from a lesion may themselves seek out a clinic where an investigation on that particular type of lesion is performed. A clinic can also be a place of referral for certain diseases. It is thus important that the type of clinic, where an investigation is performed, is considered when evaluating prevalence figures. All investigations of outpatients have, in addition, one drawback in common. Individuals who do not seek medical care are not investigated. These individuals might be more healthy than those attending the clinic. They might also be less healthy and not bother.

The value of prevalence information from investigations made on *institutionalized individuals* varies. Populations at homes for the aged may, in certain communities, be largely representative for a general population of comparable age composition. Individuals who are cared for at hospitals and similar institutions are supposedly less healthy than individuals in a general population.

Investigations of various *occupational groups* may be expected to give results which differ from those found in general populations. For instance, these groups supposedly include only relatively healthy individuals of employment age, and besides, these individuals may be exposed to a potentially noxious environment. The influence of environmental factors on oral mucosal lesions is, at present, barely understood.

All individuals in a selected population are seldom examined. Instead, portions are *sampled*, and the sampling method used should then be accurately reported. The individuals finally chosen should be *well described* giving appropriate demographic data.

Both the size of, as well as the reasons for any *non-participation* should be reported. Also, the significance of non-participation as it affects the results of the investigation should be analysed.

On investigatory procedures

Many reports state that the investigations from which prevalences were obtained, had a primary aim other than that of estimating the occurrence of oral mucosal lesions. Such studies with a low *registration priority*, i.e. where there is a risk that lesions were registered in passing, should be interpreted with great caution.

A large number of oral mucosal lesions have a subtle clinical appearance and therefore demand particularly good *investigatory conditions* for their registration. The conditions under which the clinical investigation took place should, therefore, be described.

Generally recognized clinical *criteria* are lacking for most oral mucosal lesions. It is thus extremely important that the criteria

used in any particular investigation are clearly stated.

The *investigator's background* in terms of clinical experience in recognizing oral mucosal lesions is important. Any special training or calibration measures undertaken should preferably be accounted for.

On validity and reliability

Evaluation of *validity* is facilitated if the methods of registration are tested, for instance, through comparison with results obtained with other methods of established validity. A comparison between registrations made by different investigators (inter-examiner variability) may also give a comprehension of the validity of the results. Uncertainty concerning the *reliability* of the results stem partly from random errors and partly from systematic errors. Random errors can be evaluated, for example, by repeated registrations. Systematic errors are usually more difficult to determine in clinical investigations, since they demand a cognizance of the real disease situation.

SOME PREVIOUS STUDIES ON THE OCCURRENCE OF ORAL MUCOSAL LESIONS

Studies in Sweden

Prevalence figures on the occurrence of oral mucosal lesions in Sweden are found in only a few reports (Table 1). Only one of these relates to a *general population.* Markén and Hedegård (1970) thus investigated a random sample from the population, aged 60 years and above, of Stockholm. They found that 54.2 % had a "non healthy oral mucosa", but reported no precise diagnoses. Among Swedish studies in *special populations,* Nyquist (1952, 1962), Bergman et al. (1964, 1971) and Olsson and Bergman (1971) investigated patients attending prosthetic departments of dental schools. Their primary aim was to study various functional aspects of dentures, but they also noted the occurrence of some oral mucosal lesions. Prevalences were given for denture-related lesions, such as denture stomatitis, denture hyperplasia and flabby ridge. Kristerson and Kvint (1969) and Chrigström et al. (1970) investigated the oral status and treatment needs among institutionalized individuals, and also registered a few, mostly denture-related lesions. Carlson et al. (1971) studied four populations with respect to dark, localized discolorations in the oral mucosa. It seems that this investigation is the sole study performed in Sweden, where the primary aim was to register prevalences of any oral mucosal lesions.

Studies in other countries

Some studies have been performed on *general populations* and it appears that two types of lesions, leukoplakia and focal epithelial hyperplasia, have been most often investigated.

Leukoplakia was one of the lesions studied in India by Mehta et al. (1971), and this investigation appears up to now to be the most extensive study performed on a general population. 50,915 villagers over the age of 15 years in five districts were investigated concerning the prevalence of oral cancer and precancerous conditions and the relationship of these lesions to different chewing and smoking habits. The prevalence of leukoplakia in general populations has also been reported

Table 1. *Swedish investigations containing prevalence information on oral mucosal lesions. Figures in italics have been calculated from reported data.*

Author(s)	Year	Number of individuals examined	Type of population	Age years	Lesion	Prevalence %
Nyquist	1952	1,090	Full denture wearers at a dental school	>20	Denture stomatitis	*22.8*
					Flabby ridge	*7.2*
Nyquist	1962	1,093	Full denture wearers at a dental school	>20	Angular cheilitis	30.0
Bergman et al.	1964	91	Full denture wearers at a dental school	>29	Denture stomatitis	*47.3* max.
						12.1 mand.
					Resilient alveolar ridge[1]	*80.2*[1] max.
						61.5[1] mand.
Kristerson and Kvint	1969	166	Patients at clinics for chronically ill. 125 denture wearers among 172 patients	$\tilde{x}$=74 19—94	Denture hyperplasia	*11.4* max.
						25.9 mand.
					Denture stomatitis	*28.9* max.
						16.3 mand.
Chrigström et al.	1970	201	Individuals with dentures at old age home and retirement home	60—96	Angular cheilitis	*6.5*
					Atrophy of tongue papillae	*10.4*
					Coated tongue	*5.5*
					Denture hyperplasia[2]	*1.5*
					Denture hyperplasia[3]	*1.0*
					Herpes labialis	*0.5*
					Leukoplakia	*3.0*
					Plicated tongue	*2.0*
					Traumatic ulcer	*2.5*
		160	Full denture wearers		Denture stomatitis	11 max.
						8 mand.
Markén and Hedegård	1970	168	City inhabitants 111 denture wearers	60—84	Non-healthy oral mucosa	54.2
Bergman et al.	1971	54	Patients with 1 year old full dentures at a dental school	$\tilde{x}$=58.6	Denture stomatitis	*35.2* max.
						22.2 mand.
					Resilient alveolar	*68.5*[1]

					Leukoplakia	*3.0*
					Plicated tongue	*2.0*
					Traumatic ulcer	*2.5*
		160	Full denture wearers		Denture stomatitis	11 max. *8 mand.*
Markén and Hedegård	1970	168	City inhabitants 111 denture wearers	60—84	Non-healthy oral mucosa	54.2
Bergman et al.	1971	54	Patients with 1 year old full dentures at a dental school	$\bar{x}$=58.6	Denture stomatitis	*35.2* max. *22.2* mand.
					Resilient alveolar ridge	*68.5*[1]
Carlson et al.	1971	505	Child patients at Eastman Institute, patients at a dental school and an ENT clinic, draftees	all	Localized dark discolorations	6.0
Olsson and Bergman	1971	118	Patients with full upper dentures at a dental school	30	Denture stomatitis[4]	*25.4*
		29	Patients with denture stomatitis		Resilient alveolar ridge	*72.4*[1]

[1] Includes a minor portion of cases with complete loss of the alveolar process
[2] Due to pressure
[3] Due to suction
[4] Papillomatous type excluded

from Hungary (Bruszt 1962), New Guinea (Atkinson 1964, Forlen et al. 1965, Pindborg et al. 1968a), Salomon Islands (Bailit 1968), Denmark (Grabowski 1974) and Argentina (Borghelli et al. 1975).

Focal epithelial hyperplasia was first named by Archard et al. (1965) and case reports have thereafter been published from several countries throughout the world. Some prevalence studies have been reported, but these are almost entirely confined to American Indians and Eskimos (for review see Praetorius-Clausen 1973).

Some other lesions investigated in general populations are: *preleukoplakia* (Pindborg et al. 1968a, Mehta et al. 1971), *leukokeratosis nicotina palati* (Metha et al. 1971, Reddy et al. 1973), *leukoedema* (Pindborg et al. 1968a, Mehta et al. 1971, Roed-Petersen and Pindborg 1973b), *excessive melanin pigmentation NOS* (Jakobsen 1968), *plicated tongue and geographic tongue* (van Wyk et al. 1974), *denture stomatitis* (Swallow and Adams 1967, Grabowski 1974), *lichen planus* (Metha et al. 1971, Pindborg et al. 1972a) and *submucous fibrosis* (Metha et al. 1971).

There are numerous studies performed among *special populations* in which prevalences of oral mucosal lesions are reported. Populations of outpatients appear to have been investigated most frequently. The next most commonly studied populations seem to be shoolchildren and college students.

A more detailed account of pertinent literature concerning prevalences of different oral mucosal lesions will be given below in connection with the presentation of each specific lesion (see Results and Discussion).

AIM OF STUDY

The aim of the present study was to register prevalences of oral mucosal lesions in a general Swedish population and in addition to record some lesions on the prolabium and in the circumoral regions.

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MATERIAL AND METHODS

The large general population considered necessary for the present study was selected in a way similar to that previously used in Sweden by Hellgren (1967). He investigated prevalences of skin diseases in a population of 39,571 individuals and thereby collaborated with a comprehensive, national chest x-ray service. That national service was no longer in operation at the start of this study, but a similar service has been carried on since 1964 by a public health screening organization in the county of Uppsala (Hillerdal and Irnell 1969). The present study was performed in collaboration with this organization.

GENERAL CHARACTERISTICS OF THE AREA SELECTED FOR THE STUDY*

Uppsala County is situated in the middle of Sweden and has approximately 225,000 inhabitants. From a demographic standpoint, the most well-suited area within this county for an investigation of the present type was the southern part consisting of the municipalities of Enköping and Håbo (Fig. 1). This area comprises, by Swedish standards, one medium-sized city (Enköping), a suburb of Stockholm (Bålsta) and a rural region. The population of this southern part of the county is approximately 40,000 and the population density 31 persons/km^2.

The *city of Enköping* has approximately 19,000 inhabitants and is predominantly an industrial city with engineering plants, foundries and textile companies. *Bålsta* with about 6,000 inhabitants is a rapidly growing town. About 20 % of the population are commuters, the majority of which work in the greater Stockholm area. The local industry mainly consists of factories producing various kinds of cement and construction products. *The countryside* is one of the largest grain districts in Sweden. Six small, built-up areas form the neighbourhood service centers.

Around 32 % of the gainfully employed population in the selected area are engaged in industry, 21 % in farming and 36 % in service trades.

Division of the investigated area

The investigated area contains two administrative entities, the municipalities of Enköping and Håbo. It is further divided

* Information has been obtained from:

Björkman, B. and Nordström, Å.: Utvecklingsplan för Enköpings kommunblock, 1970.

Swedish Society for Anthropology and Geography. National Atlas of Sweden. Stockholm 1953—1971.

National Central Bureau of Statistics, Population Dec. 31, 1973, according to the subdivisions of Jan. 1, 1974 (SOS). Part 1. Communes and parishes. Stockholm 1974.

National Central Bureau of Statistics, Statistical Abstract of Sweden. Volume 61. Stockholm 1974.

The Swedish Meteorological and Hydrological Institute. Personal communication, 1976.

Fig. 1. *Area of investigation indicated on a map of Scandinavia and detailed to the right.*

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into parishes, 36 of which are located in the municipality of Enköping and 5 in the municipality of Håbo.

A division of the area based on demographic data is methodologically desirable. Portions or regions should in themselves be homogeneous, but between themselves heterogeneous. In this respect, division according to municipalities is unsatisfactory and division into parishes somewhat inconvenient. Against this background a division of the selected area was made into the following regions:

Region I (Urban)	City of Enköping
Region II (Suburban)	Densely populated parishes in the municipality of Håbo, mainly corresponding to the town of Bålsta
Region III (Rural)	The remaining portion of the area, consisting mainly of the rural countryside.

Fig. 1. *Area of investigation indicated on a map of Scandinavia and detailed to the right.*

Geography, geology, vegetation and climate

Geography. The selected area is bounded 59°23′—59°53′ N and 16°44′—17°39′ E. Its greatest extension North to South as well as East to West is about 50 km. The total land area is 1,320 km². In the southern portion, there is a shoreline to Lake Mälaren of approximately 430 km. The highest point above sea level is 96 m.

Geology. The bedrock mainly comprises granites, syenites and sedimentary gneisses. The soil consists of moraine, clay, silt and very fine sand. In the southern portion, glacifluvial sediments are also found.

Vegetation. In general, the area is fertile farming land with open plains. In the southern and the middle portions, small areas of coniferous and deciduous forests with mosses and dwarf shrubs are found along with an abundance of grasses and herbs. In the north and northwestern parts, there are larger areas covered by pine and spruce forests.

Climate. The yearly mean temperature in the area is +6°C. The coldest month is January, and the warmest month July, with mean temperature of −4°C and +18°C, respectively. The annual amount of sunshine averages 1,650 hours, and the annual precipitation 500—550 mm. A permanent covering of snow is usually formed by January and disappears during the first half of April.

THE PUBLIC HEALTH SCREENING ORGANIZATION

This organization in Uppsala County comprises two health screening groups. One of these is permanently attached to the county capital Uppsala. The other group is mobile for the remaining area of the county and returns to each place visited about every third year. Each screening group includes an x-ray technician and two nurses. In addition, the mobile group is assisted by two persons provided by the local authorities at each respective place of examination. The examination, which is state-subsidized, consists of chest x-ray, measurements of weight, height, blood pressure and haematocrit, and testing for the presence of albumin and glucose in the urine. Depending on circumstances, ECG is also included.

Individuals summoned to the health screening are traced from the national civil register, which is kept as current as possible. All individuals who, during the year of examination, reaches the age of 15

years or above are selected. The health screening unit receives a personal card on these individuals, listing their complete name, date of birth, social security number, address and name of their local parish. The card is placed in a box which is kept at the examination locale. An additional card containing the above information, as well as an appointment time, is sent to each individual two weeks before examination. The card is mailed as part of a parcel, which also contains a questionnaire concerning the present state of health, and a container for a urine sample, which the individual is requested to bring to the examination locale.

One week before the mobile group moves to a new locale, the place and date of the examinations are made known in the local newspapers. To facilitate the attendance of handicapped and elderly the local authorities assist with transportation. The examination locales are also provided by these authorities, and usually consist of schools, regional farmhouses or administrative buildings.

Approximately 200 persons are summoned for each day of examination. In the ages 30—60 years the frequency of participation is about 70 % in the cities and 80 % in the rural areas. In the age groups below 30 and above 60 years, the corresponding figures are 50 % for both areas (Hillerdal and Irnell 1969).

The examinations are performed for about five hours each day with afternoon and evening sessions. Men and women are examined on alternate days. Persons appearing for the examination deliver their questionnaires and urine samples. The personal card is removed from the card box and taken by the individual to the different examinations, where the results are recorded. This information forms the basis for subsequent electronic data processing. Using this procedure, persons who do not appear, will be represented by the cards remaining in the box. No additional measures, such as contact by telephone, are taken to ensure participation in the examination. Any information concerning reasons for non-appearance is noted on the cards.

BASIC ORGANIZATION FOR THE ORAL SCREENING

It was, in some respects, necessary to supplement the facilities of the public health screening organization to accomplish the present oral screening investigation.

Each *examination locale* was arranged so that it was possible to control that all individuals who participated in the health screening also appeared for the subsequent oral examination. At least two rooms were available at every locale. One was used for the filling in of special questionnaires, while the other served as the clinical examination room. The *basic equipment* was a portable dental chair (from Den-Tal-Ez field module CM 185), a light-weight hydraulic model that allowed the investigator to work in a sit-down-position. The working illumination was provided by a low-voltage halogen bulb (Da-Ray).

For the primary investigation (see p. 15), the *personnel* consisted of the investigator, one dental nurse and one aid. The two first mentioned persons were the same during the entire investigatory period.

Individuals, who were summoned to the health screening and thus to the primary oral investigation, received a parcel. Added to each parcel was a *questionnaire* concerning the con
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In addition to advertising in local newspapers, some special *precautions to ensure maximal participation* were undertaken. Pamphlets with information concerning times and locales were mailed to all households in the area of investigation. Information was also broadcasted on local radio programs.

POPULATION

The total population of the present study was identical with that summoned to the public health screening in the municipalities of Enköping and Håbo. In order to obtain current data, and to make allowance for the time-schedule of the mobile health screening group, the population was drawn from the civil register in three groups at approximately six month intervals. The first group was drawn in July 1972 and comprised individuals born in 1957 or before. The other two groups were drawn in January 1973 and August 1973, respectively, and included individuals born in 1958 or before.

The population comprised 30,118 individuals, aged 15 years or above. Age was defined as the difference between the year of selection, 1972 or 1973, and the year of birth, the latter being at the latest 1957 or 1958.

The distribution of the population with respect to age and sex is shown in Table 2 and illustrated in Figure 2. The mean age was 43.0 years (males 42.6, females 43.4). The mean ages in regions I, II and III were 42.1, 36.9 and 46.7 years, respectively.

Foreign citizens represented 6.8 % of the population and the corresponding figure for regions I, II and III was 9.5 %, 9.3 % and 1.6 %, respectively. The largest foreign group, 5.2 % of the population, consisted of Finns. The next largest group, 0.5 %, were Greeks. A total of 39 nationalities were represented.

Primary investigation

The first part of the study, hereafter called the *primary investigation,* was undertaken in direct collaboration with the mobile health screening group. This group moved from place to place according to a predetermined schedule. In the selected area, seven places were visited (Fig. 1), and these are listed below together with the respective examination time periods.

Place of examination	*Date of examination*
Bålsta	Jan. 12—March 1, 1973
Lillkyrka	March 2—March 14
Grillby	March 15—April 5
Örsundsbro	April 6—May 3
Enköping	May 4—June 20 Aug. 6—Nov. 25
Hummelsta	Nov. 26—Dec. 16
Fjärdhundra	Dec. 17—Dec. 20 Jan. 7—Jan. 23, 1974

Of the 30,118 individuals summoned to the primary investigation 18,659 (9,173 males; 9,486 females)—or 62 %—attended. The proportional distribution of these participants in relation to the total population is shown in Table 3 and illustrated in the left hand diagram in Figure 2.

Secondary investigation

This investigation comprised a sample taken from the individuals who did not

Table 2. *Distribution of total population with respect to age, sex and region investigated.*

Age years	Region investigated									All regions			
	I (urban)			II (suburban)			III (rural)						
	Total	Males	Females	Total	Males	Females	Total	Males	Females	Total	Males	Females	%
15—24	3422	1699	1723	857	399	458	1751	911	840	6030	3009	3021	20.0
25—34	3394	1795	1599	1487	764	723	1680	882	798	6561	3441	3120	21.8
35—44	2243	1155	1088	746	407	339	1342	691	651	4331	2253	2078	14.4
45—54	2237	1095	1142	423	230	193	1583	812	771	4243	2137	2106	14.1
55—64	2072	1026	1046	293	148	145	1600	868	732	3965	2042	1923	13.2
65—74	1343	630	713	191	87	104	1177	608	569	2711	1325	1386	9.0
$\geq$75	1042	422	620	157	80	77	1078	520	558	2277	1022	1255	7.6
Total	15753	7822	7931	4154	2115	2039	10211	5292	4919	30118	15229	14889	100

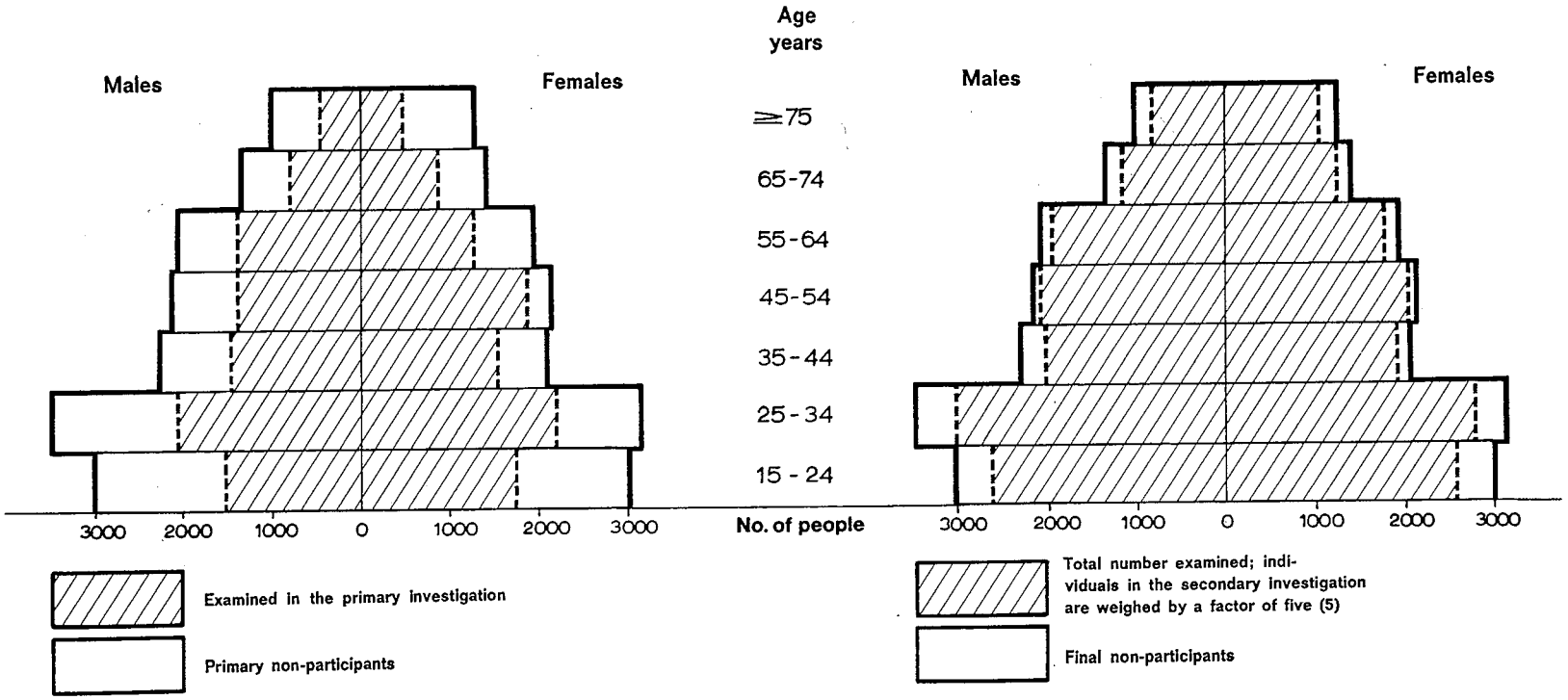

Fig. 2. *Distribution with respect to age and sex of populations described in the investigation.*

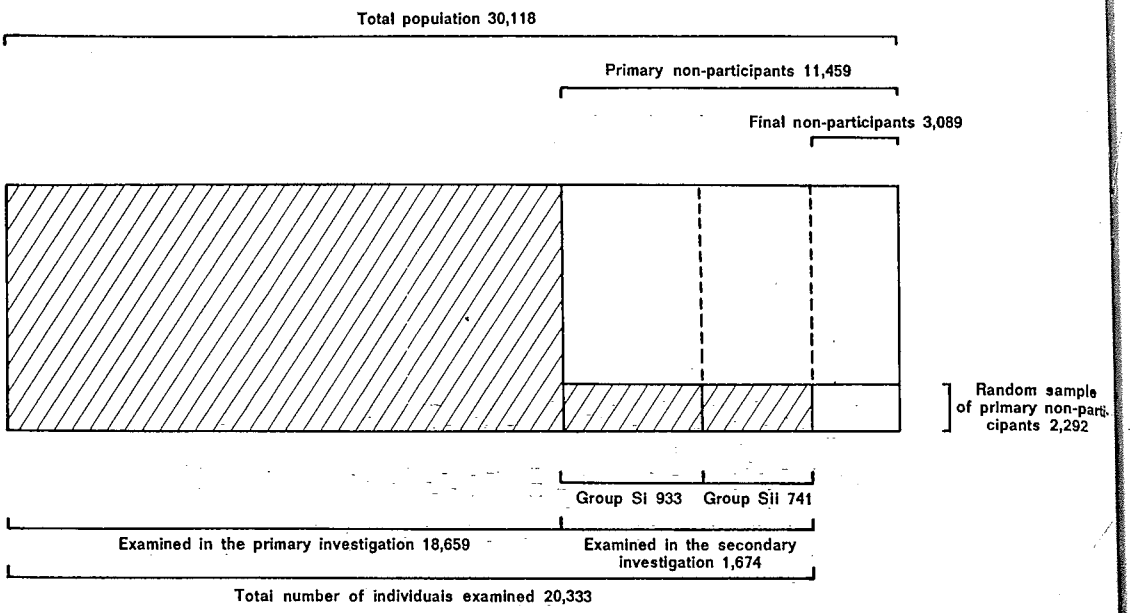

Fig. 3. *Number of individuals in the total population and in various sub-populations. Dashed area indicates individuals examined.*

attend after the first summoning, i.e. at the primary investigation. From these 11,459 non-participants every fifth individual, e.g. 2,292 persons, was systematically sampled from the cards remaining in the card box after the completion of the primary investigation. They were individually summoned through a letter containing information on locale and appointment time. If the person did not appear, telephone contact was made and a new appointment arranged. Following these procedures 933 persons (487 males; 446 females), hereafter referred to as *Group SI*, attended (see Fig. 3). The conditions for examining Group SI were nearly identical with those of the primary investigation, the main difference being that the health screening group was not present. The time period for investigating Group SI and the places of examination are shown below.

Place of examination	*Date of examination*
Enköping	Feb. 4—April 16, 1974
Örsundsbro	April 17—April 24
Lillkyrka	April 25—May 3
Grillby	May 4—May 14
Hummelsta	May 15—May 27
Fjärdhundra	May 28—June 7
Bålsta	June 8—June 28

Individuals who still did not attend were contacted in person. In this way 741 additional individuals (376 males; 365 females), hereafter referred to as *Group SII* (see Fig. 3), were examined. These examinations were carried out in private homes, places of work, hospitals, retirement homes and other institutions. In a few instances, examinations had to be performed outside the area of investigation, and these concerned:

1) persons attending the University of Uppsala who could not be reached for examination at their home residence.

Table 3. *Frequency of participation (%) in the primary investigation with respect to age, sex and region investigated.*

Region investigated	I (urban)	II (suburban)	III (rural)	All regions

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7—April 24
5—May 3
—May 14
5—May 27
3—June 7
—June 28

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Random sample of primary non-participants 2,292

3,089

Table 3. *Frequency of participation (%) in the primary investigation with respect to age, sex and region investigated.*

Age years	Region investigated: I (urban) Total	I (urban) Males	I (urban) Females	II (suburban) Total	II (suburban) Males	II (suburban) Females	III (rural) Total	III (rural) Males	III (rural) Females	All regions Total	All regions Males	All regions Females
15—24	51	47	55	53	46	58	61	61	62	54	51	57
25—34	61	57	66	65	59	71	70	65	75	64	59	69
35—44	65	64	67	66	60	72	78	73	82	69	66	73
45—54	65	63	68	63	57	70	76	76	76	69	67	71
55—64	64	66	62	65	65	66	71	70	72	67	67	66
65—74	59	60	59	58	57	59	65	64	66	62	62	62
≥75	40	43	38	39	45	34	41	47	36	40	45	37
Total	59	57	61	61	57	66	67	66	68	62	60	64

Table 4. *Frequency of participation (%) in the total investigation with respect to age, sex and region investigated. Individuals from the secondary investigation weighed by a factor of five (5).*

Age years	Region investigated: I (urban) Total	I (urban) Males	I (urban) Females	II (suburban) Total	II (suburban) Males	II (suburban) Females	III (rural) Total	III (rural) Males	III (rural) Females	All regions Total	All regions Males	All regions Females
15—24	83	82	84	85	87	83	92	93	91	86	86	86
25—34	85	84	87	90	86	94	92	91	94	88	86	90
35—44	92	90	93	92	89	97	92	89	97	92	89	95
45—54	96	99	93	96	93	98	99	97	100	97	98	96
55—64	93	92	94	91	85	97	93	93	93	93	92	94
65—74	84	84	85	90	86	92	93	88	98	88	86	91
≥75	84	79	87	81	83	79	82	80	84	83	80	85
Total	88	87	89	90	87	92	92	91	94	90	89	91

2) persons at the Ulleråker Mental Hospital, Uppsala.
3) developmentally retarded children at Rickomberga Hospital, Uppsala.

Approximately one third of Group SII was examined during the same time period as Group SI, Feb. 4—June 28, 1974, and the remaining two thirds during the time July 6—Oct. 31, 1974. Examination of individuals in Group SI and the first third of Group SII was undertaken by the investigator and the dental nurse, while examination of the last two thirds of Group SII was performed solely by the investigator. All equipment used in the examination of Group SII was contained in a portable bag. Illumination was provided by a low-voltage lamp (Accu-Lux).

In summary a total of 1,674 persons, or 73.2 % of the sampled population from the primary non-participants, were examined in the secondary investigation (Group SI + Group SII).

Frequency of participation

Of the total population of 30,118 individuals 67,5 % or 20,333 (10,036 males; 10,297 females) were finally examined; 18,659 in the primary and 1,674 in the secondary investigation. The last-mentioned individuals had been collected by a random sample of every fifth person among the primary non-participants. Those examined in the secondary investigation thus represented $5 \times 1{,}674$, or 8,370, individuals. From the standpoint of frequency of participation, all those examined consequently represented 27,029 persons or 89.7 % of the total population.

Table 4 shows the frequency of participation as calculated above and the right hand diagram of Figure 2 illustrates this frequency graphically.

Reasons for non-participation

Some details as to the reasons for not appearing was obtained in connection with the primary investigation, but the major information in this context was provided during the secondary investigation and through other means, such as examining the files in the local parish offices. The various reasons for non-participation are defined below. Some definitions differ between the primary (P) and secondary (S) investigations or apply only to the primary investigation.

Refusal. Persons where information was obtained revealing that they did not want to be examined. Upon inquiry, no reason was given as to why the examination was not permitted (P, S).

Change of residence. Persons who had moved to a permanent address outside the area of investigation. The date of transfer could be controlled by checking the files of the local parish. The registration of these persons could thus be assigned to either the primary or secondary investigation (P, S).

Temporarily away. Persons having a permanent address in the area of the investigation, but who were temporarily away because of, for instance, work, study, vacation or military service (P, S).

Hospitalized. Persons hospitalized at the time of investigation. These persons were questioned as to the reasons for hospitalization (P, S).

Illness at home. Persons who were ill and remained at home, or persons remaining at home and caring for sick children or other acquaintances. Those who were ill were questioned as to the nature of their illness (P, S).

Old age. Persons who because of advanced age found it difficult to attend the examination and did not receive help with transportation (P).

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Pregnancy. P participate bec foetal injuries examination (

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Recently dead. Persons who had died after the selection of the population was made. Information as to the date of death was obtained from the files of the local parish. Thus, registration could be assigned to either the primary or secondary investigation (P, S).

Work. Persons who could not leave their work at the time of the investigation (P, S).

Pregnancy. Pregnant women who would not participate because of, for example, concern for foetal injuries as a result of the chest x-ray examination (P).

Recently participated in another health examination. Many companies offer their employees regular health examinations. Besides, Swedish law requires annual health examinations for those working in the food industry. A large number of participants in such examinations deemed a further health survey as superfluous (P, S).

Fear of physicians and/or dentists. Persons who stated that they avoid all types of professional contact with physicians and/or dentists (P, S).

Dislike of mass investigations. Persons who dislike being examined together with others. These individuals often experience such examinations as an infringement on their personal integrity. Persons with fear of crowds (agoraphobia) were also included in this category (P, S).

Long distance to examination locale. Persons who, because of transportation difficulties, found it impossible to appear. The longest distance between residence and examination locale was approximately 20 km (P).

Forgot to come. Persons who remember receiving a parcel but forgot to come (P).

Did not receive parcel. Persons who denied receiving a parcel or who did not observe that it had arrived (P).

No remembrance of reason. Persons not remembering the reason for non-appearance (P).

No contact. No information was available as to the reason for non-appearance (P). Persons who were not present at the given address at any of at least four visits by the investigation team. The addresses were verified from the local parishes or by neighbours (S).

Other reasons. This category includes, for instance, persons on whom the available information was insufficient (P, S).

The frequencies of the different reasons for non-participation in the primary investigation (primary non-participation) and in the secondary investigation (final non-participation) are shown in Tables 5 and 6. Calculation of the frequencies is entirely based on information from the sampled individuals of the secondary investigation. Since every fifth person was sampled, the information from each individual was weighed by a factor of five when estimating the frequencies in the total population.

Large differences were found between the groups of primary and final non-participants when analysing the reasons for not attending. The frequencies for the majority of reasons for non-participation were considerably reduced through personal contact being made with the individuals, and thereby the significance of other reasons showed a relative increase. This latter increase was particularly noted for individuals who had changed their residence. These persons constituted nearly half of the final non-participants. To a large extent they were Finns who had returned to their home country. For this category of individuals, as well as for those recently dead, the increase was also one of absolute numbers.

Concerning those individuals who were not examined because of illness there was no instance where an obvious connection between the actual illness and any specific oral mucosal lesion could be suspected.

Table 5. *Frequency of reasons for primary non-participation after the primary investigation. Reasons listed according to decreasing frequencies.*

	Number of individuals	Weighed % of total population	% of primary non-participants
Recently participated in another health examination	371	6.2	16.2
Work	328	5.4	14.3
Temporarily away	281	4.7	12.3
No contact	231	3.8	10.1
Change of residence	224	3.7	9.8
Illness at home	152	2.5	6.6
No remembrance of reason	149	2.5	6.5
Refusal	149	2.5	6.5
Hospitalized	115	1.9	5.0
Did not receive parcel	64	1.1	2.8
Forgot to come	48	0.8	2.1
Old age	35	0.6	1.5
Recently dead	28	0.5	1.2
Long distance to examination locale	24	0.4	1.0
Fear of physicians and/or dentists	20	0.3	0.9
Dislike of mass investigations	18	0.3	0.8
Pregnancy	10	0.2	0.4
Other reasons	45	0.7	2.0
Total	2292	38.1	100

Table 6. *Frequency of reasons for final non-participation after the secondary investigation. Reasons listed according to decreasing frequencies.*

	Number of individuals	Weighed % of total population	% of final non-participants
Change of residence	308	5.1	49.8
No contact	97	1.6	15.7
Recently dead	84	1.4	13.6
Refusal	84	1.4	13.6
Temporarily away	11	0.2	1.8
Hospitalized	10	0.2	1.6
Recently participated in another health examination	5	<0.1	0.8
Illness at home	4	<0.1	0.6
Dislike of mass investigations	3	<0.1 —0.2	0.5
Fear of physicians and/or dentists	1	<0.1	0.2
Work	1	<0.1	0.2
Other reasons	10	0.2	1.6
Total	618	10.3	100

INVESTIGATOR

Pretyped forms

Six forms were
data (see Figs.
main purpose of
the following info

Form 1: demograph
Form 2: medicines
Form 3: tobacco a
Form 4: oral hygie
Form 5: lesions of
Form 6: histologic
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INVESTIGATORY PROCEDURES

Pretyped forms

Six forms were used for registration of data (see Figs. 5—10, Appendix). The main purpose of each form was to collect the following information.

Form 1: demographic data
Form 2: medicines taken
Form 3: tobacco and alcohol consumption
Form 4: oral hygiene and prosthetic status
Form 5: lesions of the oral mucosa
Form 6: histologic evaluation of tissue specimens.

Forms 1 and 3 were also printed in Finnish and Greek. The lay-out of the forms was adapted to the special routines devised for the subsequent data handling.

Clinical examination

The circumoral area, prolabium and oral mucosa were systematically examined using a mouth mirror and tongue depressor. When present, dentures were removed from the mouth. The extent of some lesions or structures was measured with a specially constructed sliding caliper or a probe, graded in millimeters. The time for each examination averaged 3 minutes.

Localizations were recorded using the topographical division of the oral mucosa reported by Roed-Peterson and Renstrup (1969). For circumoral regions, a similar division was designed for the present study. The lesions and localizations were recorded in code form by the dental nurse.

After the clinical examination, all individuals were questioned concerning previous recurrent rashes or sores on the lips or in the oral cavity. At the same time they were shown color pictures of herpes labialis and aphthous ulcers.

Photographic documentation

Color photographs were taken of all lesions diagnosed as leukoplakia or lichen planus. All lesions where doubt as to the diagnosis existed were also photographed, as well as all lesions from which biopsies were to be taken. A total of 7,300 photographs were taken on 1,800 patients.

The camera equipment consisted of an Asahi Pentax Spotmatic II with a 135 mm lense and 3 extension rings. The electronic flash attachment was a Braun F 800. The color film, Agfachrome 50 S 135/36, was of the same emulsion batch and stored in a refrigerator.

Tissue specimens

Biopsies were obtained from lesions on which there was doubt as to the diagnosis. In addition, biopsies were procured for validation of various clinical diagnoses. The majority of biopsies were taken with a punch instrument (diameter 5.5 mm) under local anesthesia using 3 % Citanest-Adrenaline®. They were immediately fixed in 10 % neutral formalin. The wounds were closed with resorbable suture material.

Smears were taken from all lesions with a preliminary diagnosis of leukoplakia as well as from several lesions where an infection by Candida albicans was suspected. Smears were also taken from a number of vesicular lesions where a virus etiology was thought to exist. All smears were fixed in 95 % alcohol.

All tissue specimens were sent to the Department of Oral Pathology, School of Dentistry, Malmö for evaluation.

Smear specimens, as a rule, were taken in connection with the initial examination. Biopsies, however, were taken a few days

later after making a special appointment. When an absolute indication for taking a biopsy existed, i.e. when there was doubt about the diagnosis and the patient was unwilling to appear, a notation of refusal was made on the patient's form. It was also noted if the patient was unable to return for the biopsy procedure.

A total of 715 biopsies and 410 smears were taken on 639 patients. Twenty patients refused biopsy and 18 could not return for the biopsy procedure due to lack of time.

Hospital referrals

A total of 141 patients were referred to the University Hospital in Uppsala. All patients with skin diseases revealing manifestations on the skin as well as in the oral mucosa, were referred to the Dermatology Clinic. Patients with lesions that were suspected to be malignant in nature, or with lesions which for other reasons required further investigations or treatment, were referred to the Oral Surgery Clinic. The number of patients referred to these two clinics were 122 and 19, respectively.

Diagnostic procedure

The diagnoses were based on specific clinical criteria set forth for this investigation. Definite diagnoses were settled either directly or following supplementary diagnostic procedures (see Fig. 4).

If a definite diagnosis could be made directly *without biopsy*, this was noted as C2 in column C2/CH2 on Form 5 at the time of the examination. If doubtfulness as to the diagnosis existed, C1 was noted on the form, and notations were made in column C1/C2. In addition, the patient's

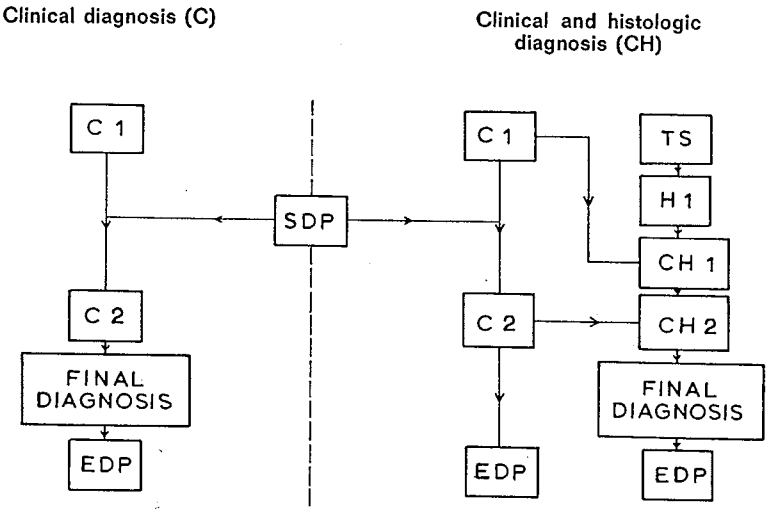

Fig. 4. *Diagnostic procedure shown schematically. Left-hand side followed without, and right-hand side with tissue specimen.*

forms were placed into a particular C1 group. After obtaining information from supplementary diagnostic procedures, e. g. consultations, C1 was either confirmed or revised to C2 and noted in column C2/CH2. *C2,* irrespective of the way it was obtained was registered as the *final diagnosis* and stored in the computer.

When *biopsies* had been obtained, the specimens were sent together with Form 6 for microscopic evaluation. A primary clinical-histologic diagnosis, CH1, was thus obtained. C2 was settled either directly or after supplementary diagnostic procedures, without guidance of CH1 and noted in column C1/C2 on Form 5. A consideration of C2 and CH1 then resulted in a definite clinical-histologic diagnosis, *CH2,* which became the *final diagnosis.* This was noted in column C2/CH2 on Form 5. Information from all cases where biopsies had been obtained was stored in the computer as C2 and CH2.

A final diagnosis could be made at the time of the initial examination for most of the lesions. The majority of the remaining lesions were given a final diagnosis with the aid of histopathologic information. The main principle in arriving at the definite clinical-histologic diagnosis CH2 was, that C2 was confirmed to CH2 if the microscopic evaluation either supported or did not contradict the proposed C2. On the other hand, if the histologic appearance was specific for a lesion other than C2, this information dictated a diagnosis, CH2, divergent from C2. In cases where the histologic information contradicted the diagnosis C2, but still was not specific for any other lesion, the final diagnosis was made after subjective weighing of all available clinical and histologic information. Such weighing occurred in relatively few instances.

Handling of data

Information on Forms 1—6 was transferred to special sheets constructed for optical read-out (Studiagnos®, Studentlitteratur AB, Lund). This procedure was performed by five persons, all of whom were given written instructions as well as practical training before receiving the forms. The transferred data were controlled against the original information by a sixth person, who was one and the same during the entire period of the investigation.

The optical read-out was done with a Datasaab OMR-5900, connected to a punch-tape machine. The information on the tapes was then entered in a computer, Univac 1108. All stored data were tested for logical errors and corrections made.

DIAGNOSTIC CRITERIA

The final diagnoses in this study were based on defined criteria, which had been formulated with due consideration to the special conditions that might be expected to exist in a mass investigation. Consequently, the criteria describe the minimum of clinical observations considered necessary to make a specific diagnosis. For some lesions, the criteria have been grouped to describe basic features. These features could, in defined combinations, either determine the main diagnosis of the lesion or be used to permit subtyping. The criteria may to some extent be evaluated with aid of the references attached to each diagnosis.

Diagnostic labels have been chosen in accordance with WHO's Application of the International Classification of Diseases to Dentistry and Stomatology, ICD-DA, Geneva, 1973. Its 5-digit numbers, or for some tumors 3-digit numbers, are applied.

In those cases where a diagnostic label was not found in the WHO publication, a working name, used in the present study, is given. The sequence in listing the lesions deviates somewhat from that of ICD-DA.

Herpes zoster 053.X0

Clustered vesicles and/or erosions distributed segmentally in an area, which apparently corresponds to ramifications of a nerve in the oral cavity. The rash is unilateral and accompanied by pain or intense itching.

Cooke 1960, Nally and Roos 1971.

Herpes labialis 054.X0

Cluster(s) of vesicles and/or crusts on or adjacent to the vermilion border of the lip, not involving the moist mucosa. There is a past history of recurrence and healing time in previous lesions has not exceeded three weeks.

Graykowski et al. 1966, Ship et al. 1967.

History of herpes labialis 054.X0

The lesion of herpes labialis, as described by words and pictures, is well recognized by the patient, who has suffered from this lesion at least once during the last two years. There is a past history of recurrence and the healing time of the lesion has not exceeded three weeks. There is no present lesion of herpes labialis.

Ship et al. 1967.

Intraoral herpetiform lesion 054.X9

Cluster(s) of vesicles and/or ulcerations on the oral mucosa. There is no red inflammatory halo around the lesion.

Griffin 1965, Lehner 1967a.

Herpangina 074.00

Minute vesicles and/or ulcers on an erythematous mucosa in the posterior part of the oral cavity. The patient has angina and symptoms such as elevated temperature, headache, pain in the back, neck or extremities.

Zahorsky 1924, Kramer 1967.

Acute necrotizing ulcerative gingivitis 101.X0

At the top of the interdental papillae there is a necrotic area covered by a grayish-yellow pseudomembrane, which can readily be wiped off leaving a red, bleeding and tender surface.

Pindborg 1951.

Acute pseudomembranous candidosis 112.X0

Creamy white or grayish patches or nodules, which can easily be rubbed off leaving a bright erythematous or bleeding surface.

Storgård-Jensen and Holst 1969.

Chronic candidosis 112.X8

1. An erythematous area of the mucosa with or without irregular white patches in the center of the lesion.
2. An erythematous area with white, pinhead-sized nodules.
3. If the lesion is surrounded by a whitish borderline this does not exceed 5 mm in width. The white structures cannot be rubbed off and cannot be attributed to any other diagnosable disease.

Diagnosis: 1+3 atrophic type
2+3 hyperplastic type

Cernéa et al. 1965, Cohen 1965, Jepsen and Winther 1965, Lehner 1967, Roed-Petersen et al. 1970.

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Papilloma 210

includes verruca vulgaris 079.10

Pedunculated or sessile tumor with a papillomatous surface. The color is white or slightly pink. When located at the vermilion border of the lips the lesion may be grayish-brownish.

Rose 1965, Praetorius-Clausen 1972.

Focal epithelial hyperplasia 528.73

includes 529.73

1. Circumscribed, sessile, soft, rounded and nodular elevation(s) with a color like the adjoining mucosa.
2. Circumscribed, from the adjoining mucosa well demarcated, soft, slightly elevated papule(s) with a flat, whitish surface or with a color like the adjoining mucosa.
3. The boundary between affected and unaffected mucosa is partly irregular.

Diagnosis: 1+3 or 2+3.

Praetorius-Clausen et al. 1970.

Lipoma 214.X0

A well-defined, sessile or partly submerged round or ovoid, soft tumor with a smooth surface and a yellowish appearance. Small subepithelial blood vessels are often clearly visible against the yellow tumor background.

Hatziotis 1971.

Haemangioma 227.X0

1. Well demarcated, somewhat raised, isolated, multilobulated projections. The color is red-purple to pink.
2. On the tongue there is a raised, smooth tumor blending out in the normal mucosa without demarcation. The pattern of the tongue papillae appears normal or somewhat thinned out. The color is slightly bluish.
3. The lesion blanches upon pressure.

Diagnosis: 1+3 or 2+3.

Shklar and Meyer 1965.

Lymphangioma 227.X0

Multiple, small, painless, firm papillary projections or vesicles which are grayish, yellowish or purplish. The lesion has been recognized at least since youth.

Lichtenstein et al. 1965, Shklar and Meyer 1965.

Gingival fibromatosis and localized enlargement e.g. tuberosity 523.90

1. General gingival enlargement.
2. Symmetric enlargement of the lingual or palatal gingiva in the molar region. The enlargement increases in width posteriorly.
3. The mucosa is smooth or stippled and of normal or almost normal color.
4. At least on the anterior teeth, the anatomic crown is covered up to one-half by gingival tissue.
5. There is a family history of the lesion.

Diagnosis: 1+3+4, 1+3+5 or 2+3

Rushton 1957, Srsen and Mocik 1973.

Excessive melanin pigmentation NOS 528.96

includes melanoplakia

Macules or diffusely outlined areas of brown to brownish-blue color. The lesion cannot be classified as due to exogenous pigmentation.

Dummet 1962, Eleutério 1969.

Gingival cyst 523.92

Local, painless, rounded swelling on the attached mucosa of the alveolar process. The lesion is of normal mucosal or yellowish color and firm or fluctuant at palpation. Probing releases a serous or mucous fluid.

Moskow et al. 1970.

Chronic desquamative gingivitis 523.13

Intensely erythematous surface of the marginal and attached gingiva, which looks glistening. The epithelial covering of the gingival mucosa can be peeled off leaving a raw, painful surface. The lesion is restricted to the gingiva. There is no concommitant oral diagnosis of lichen planus, pemphigoid, pemphigus or any other disease in which the gingivitis could be a symptom.

McCarthy et al. 1960.

Recurrent aphthae 528.20

1. Well defined, rounded ulcer(s) covered by a grey-white or yellowish fibrinous exudate and surrounded by a red, inflammatory halo.
2. A red macule or a slightly raised papule.
3. The lesion is located to non-keratinized mucosa or to the specialized mucosa of the tongue. There is intense or moderate pain, a past history of recurrence and the healing time in previous lesions has not exceeded three weeks.

Diagnosis: 1+3 or 2+3.

Graykowski et al. 1966, Stanley 1972.

History of recurrent aphthae 528.20

The lesion of recurrent aphthae, as described by words and pictures, is well recognized by the patient, who has suffered from this lesion at least once during the last two years. There is a past history of recurrence and the healing time of the lesion has not exceeded three weeks. There is no present lesion of recurrent aphthae.

Ship 1965, Ship et al. 1967.

Periadenitis mucosa necrotica recurrens 528.22

1. Ulceration(s) covered with a gray-white or yellowish fibrinous exudate.
2. The lesion is deep and indurated and there is severe pain.
3. Scarring after previous episodes.
4. Healing time exceeds three weeks.

Diagnosis: 1+2+4 or 1+3+4.

Hjørting-Hansen and Siemssen 1961, Graykowski et al. 1966.

Preleukoplakia 528.78

includes 529.78

Grayish-white area with indistinct boundaries blending into the adjacent normal mucosa. The lesion cannot be rubbed off and cannot be attributed to any other diagnosable lesion.

Mehta et al. 1971.

Leukoplakia 528.6X

includes 529.72

1. A whitish patch of the oral mucosa with demarcated margins or with indistinct boundaries blending into the adjacent normal mucosa.
2. A whitish patch of the oral mucosa. In parts of the lesion, there is one or more erythematous areas of atrophic or erosive nature. There is a homogeneous white marginal area exceeding 5 mm in width. This is either well demarcated or blend
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Pindborg
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or blends into the adjacent normal mucosa.

3. A whitish patch of the oral mucosa. In parts of the lesion there are white, pinhead-sized nodules, which may be present on an erythematous base. There is a homogeneous white marginal area exceeding 5 mm in width. This is either well demarcated or blends into the adjacent normal mucosa.
4. The lesion cannot be rubbed off and cannot be attributed to any other diagnosable lesion.

Diagnosis: 1+4 homogeneous type
2+4 atrophic type
3+4 nodular type

Silverman et al. 1963, Jepsen and Winther 1965, Storgård-Jensen and Holst 1969, Roed-Petersen et al. 1970.

Leukokeratosis nicotina palati 528.72

1. The mucosa of the palate shows fissuring and is grayish-white with loss of translucence.
2. In the palate there are multiple nodules with red, centric dots.
3. The patient is or has recently been a habitual smoker.

Diagnosis: 1+3 or 2+3.

Cooke 1956, Chapman and Redish 1960, Saietz 1975.

Snuff dipper's lesion 528.78

There is a slight or heavy wrinkling of the mucosa with or without whitish-yellowish to brown discoloration and with or without an obvious thickening. The localization of the lesion is the site for regular placing of snuff.

Pindborg and Renstrup 1963, van Wyk 1965, Axéll et al. 1976.

Leukoedema 528.71

A diffuse, grayish-white, velvet-like edematous film covers a smooth mucosa. In a buccal location delicate folding can be seen. The folds are more or less vertical and do not disappear at maximal opening of the mouth. The surface of the lesion can be scraped off or displaced and is reestablished within a short time. The lesion is not restricted to the parotid duct papilla or to the area corresponding to the occlusal plane.

Sandstead and Lowe 1953, Hamner et al. 1971.

Frictional keratosis 528.78

includes 529.78

A whitish or grayish patch of the mucosa. The patch cannot be rubbed off and cannot be attributed to any other specific lesion. The localization of the lesion corresponds to the site for a readily recognizable mechanical trauma.

Cooke 1956, Cawson 1969.

Cheek and lip biting 528.93

includes 529.98

The oral mucosa shows a rough, macerated surface with irregular, flaky desquamation. The lesion, which is diffusely outlined, is situated where self-infliction by chewing is possible.

Hjørting-Hansen and Holst 1970, Sewerin 1971.

Traumatic ulcer 528.92

includes 529.98

Mildly or moderately symptomatic ulceration of the oral mucosa or the lips. Trauma can be related to the site of the lesion either at examination or from anamnestic data.

Ulcus mucosae oris NOS 528.99

includes 529.99

Mildly or moderately symptomatic ulceration of the oral mucosa or the lips. The etiology of the lesion is unknown and the lesion cannot be assigned any specific diagnosis.

Mucocele 527.69

1. Pink, yellow or bluish, painless and firm or fluctuant swelling. Probing releases a serous or mucous fluid.
2. A deep seated, firm or fluctuant lesion of normal mucosal color which varies in size from time to time.

Diagnosis: 1 or 2.

Cataldo and Mosadomi 1970.

Pyogenic granuloma 528.94

includes 210.43, 523.98, 529.98 and 634.91

Elevated, sessile or pedunculated tumor with a smooth, granular or lobulated surface. The lesion is markedly red or reddish-brown as compared to the surrounding mucosa and bleeds freely when manipulated.

Shklar and Meyer 1965, Angelopoulos 1971.

Fibroepithelial polyp 528.90

includes fibroma 210

Circumscribed, rounded, sessile or pedunculated, slowly developing overgrowth of the oral mucosa covered with a smooth surface similar to the adjacent mucosa or with a somewhat whitish surface.

Barker and Lucas 1967.

Denture hyperplasia 528.90

1. Redundancies of the oral mucosa in areas beneath the periphery of a denture. The color is like the surrounding mucosa or slightly reddened.
2. Hyperplasia of the mucosa in a localization corresponding to a suction chamber of an upper denture.

Diagnosis: 1 or 2.

Bethmann 1962, Ralph and Stenhouse 1972.

Flabby ridge 528.90

Part of or the whole of the alveolar process is resilient. The top of the ridge can be displaced at least 2 mm.

Bergman et al. 1971.

Denture stomatitis 528.91

1. The denture-covered area is diffusely red and contrasts markedly from the adjacent mucosa.
2. In the denture-covered area there are multiple, small, papillomatous, reddened hyperplasias.
3. The lesion includes less than two thirds of the denture-covered area.
4. The lesion includes two thirds or more of the denture-covered area.

Diagnosis: 1+3 localized type
1+4 generalized type
2 papillomatous type

Guernsey 1965, Budtz-Jørgensen 1974.

Papilliform hyperplasia of palate 528.98

Multiple, sessile, spherical projections in the palate. The mucosa shows normal color or is somewhat reddened. There is no denture in contact with the affected area.

Schmitz 1964, Guernsey 1965, O'Driscoll 1965.

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Diagnosis: 1+

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Denture-related mucosal petechiae 528.98

In the denture-covered palatal mucosa there are distinct, multiple, often coalescing red to bluish-purple subepithelial spots, which do not blanch when pressure is applied.

Angular cheilitis 528.50

1. Fissuring or ulceration of the skin and/or oral mucosa in the labial commissure(s).
2. Discontinuity of the commissural mucosa or skin can be provoked by a slight stretching.
3. Healing time exceeds two weeks.

Diagnosis: 1+3 or 2+3.

Nyquist 1962, Cawson 1963, Mäkilä 1969.

Median rhomboid glossitis 529.2X

In the area near foramen caecum of the tongue there is an ovoid-shaped lesion with a smooth or lobular surface completely devoid of papillae. The color may be somewhat reddened. The lesion is demarcated from the surrounding mucosa by a furrow.

Martin and Howe 1938, Sammet 1939, Delemarre and van der Waal 1973.

Atrophy of tongue papillae, unspecified 529.49

1. The tongue shows one or more areas with complete loss of filiform papillae.
2. The lesion is located to the area of, or immediately in front of, the terminal sulcus. It is rounded and may be depressed, elevated or in level with the surrounding mucosa.
3. The lesion is located to the margins in the posterior part of the tongue. It may extend on the dorsum of the tongue with a bow-shaped delineation to the normal mucosa. There may be a subtle whitish, dry surface.
4. The lesion is located to the central parts of the tongue, well separated from the terminal sulcus. There may be a subtle whitish, dry surface.
5. The lesion comprises almost the total area of the dorsal surface of the tongue.
6. The localization and distribution does not correspond to criterion 2, 3, 4 or 5.
7. There is no obvious reddening of the tongue mucosa and there are no symptoms of burning or itching.
8. There is no diagnosis of lichen planus determined in any other location of the oral mucosa.

Diagnosis: 1+2 median type
1+3+8 or 1+4+8 lichenoid type
1+5+7 generalized type
1+6+7 miscellaneous type

Cernéa et al. 1965, Kuffer 1973, Cooke 1975.

Glossitis, unspecified 529.09

1. The tongue shows partial or total depapillation.
2. The tongue is markedly reddened.
3. There are symptoms like itching or burning.
4. The lesion cannot be classified as any other tongue lesion.

Diagnosis: 1+2+4, 1+3+4 or 2+4.

Geographic tongue 529.1X

1. Well demarcated areas of the tongue with absence of filiform papillae and showing peripheral reddening.

2. White or yellowish, serpiginous lines are partly surrounding red, depapillated areas or are seen isolated.

Diagnosis: 1 or 2.

Hume 1975.

Geographic stomatitis 528.78

Bright red, demarcated areas which are partly surrounded by delicate, serpiginous white or yellowish lines. The lesion is not located to the tongue.

Hume 1975.

Plicated tongue 529.5X

The dorsum or margins of the tongue are crossed by one or several grooves, which are estimated to be at least 2 mm deep in a minimum total length of 15 mm.

Witkop and Barros 1963, Redman 1970, Halperin et al. 1953.

Coated tongue 529.30

1. The dorsal surface of the tongue shows a coating which conspiciously contrasts to a normal condition.
2. The dorsal surface of the tongue shows a pigmentation of exogenous nature.
3. The length of the filiform papillae does not exceed 3 mm.

Diagnosis: 1+3 or 2+3.

Celis and Little 1966.

Hairy tongue 529.31

The length of the filiform papillae of the tongue exceeds 3 mm.

Celis and Little 1966.

Discoid lupus erythematosus 695.40

Well-defined, erythematous, atrophic and, when present on the vermilion border of the lips, scaly areas. The areas show 2—4 mm broad, slightly raised whitish margins from which numerous delicate lines are radiating. In the periphery of the lesions dilated blood capillaries can be recognized.

Monash 1931a, Andreasen 1964.

Lichen planus 697.00-697.08

1. White, pinhead-sized papules.
2. White, distinct striae forming linear, reticular or annular patterns.
3. White, plaquelike lesions with striae at the margins.
4. Red, erythematous areas with striae at the margins.
5. Atrophy of tongue papillae. The atrophic area has a whitish, dry surface and white papules or striae are present in other localizations of the mouth.
6. Areas of erosions or ulcerations with striae at the margins.
7. Vesicles or bullae with content of clear or slightly red fluid in areas with lesions compatible with criterion 1, 2, 3, 4 or 5.
8. The white structures cannot be rubbed off.

Diagnosis: 1+8 papular type
2+8 reticular type
3+8 plaque type
4+8 or 5+8 atrophic type
6+8 erosive type
7+8 bullous type

Cooke 1954, Andreasen 1968, Kuffer 1971, Shklar 1972.

Fordyce's condition 750.85

Small, sometimes slightly elevated circular or rosetteshaped chamois-colored spots which are distinctly demarcated from the surrounding mucosa.

Miles 1958, Sewerin 1975.

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Diagnosis: 1

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White sponge naevus 750.86

1. White or grayish, folded or corrugated mucosa which appears soft and spongy. The lesion has a symmetrical distribution.
2. The patient can remember the lesion being present since at least youth.
3. A family history can be obtained.

Diagnosis: 1+2 or 1+3.

Cooke 1956, 1967.

Gingival hyperplasia, adverse effect of hydantoin derivatives N 966.10

The marginal gingiva shows hyperplastic changes around at least the anterior teeth, and the interdental papillae have assumed a rounded, blunt form. The anatomic crown of at least one tooth is covered up to one-third by gingival tissue, which at least in minor forms of the lesion is smooth or stippled and of normal or almost normal color. The patient receives daily doses of hydantoin derivatives.

Aas 1963, Angelopoulos and Goaz 1972.

Amalgam tattoo N 998.40

A blue to gray or black lesion with a poorly defined periphery. The lesion is covered by a normal mucosal surface.

Weathers and Fine 1974.

PILOT STUDY OF PROCEDURES

Questionnaires and details concerning equipment were tested on a number of small groups of individuals. To test the entire oral screening organization, an integrated trial investigation was performed in collaboration with the mobile health screening group. This investigation was undertaken among 500 individuals (264 males; 236 females) who were summoned to the health screening in Tensta parish during 14 days in October, 1972.

During the first days questionnaires and forms, clinical examination procedures, photographing of the lesions and taking of biopsies were separately tested and evaluated. During the last five days, all procedures were co-ordinated. The information obtained was then used to test all the routines in connection with data handling.

Improvements were found necessary concerning lighting conditions, assistance with answering the questionnaires, the capacity for transferring data from the forms to data sheets and transportation of the collected material. Adjustments of these factors were made before the main investigation was initiated.

STATISTICAL METHODS

Calculation of prevalences

Prevalences are first calculated in the various demographic groups and thereafter weighed together, yielding prevalences for males, females and total population. For the i:th demographic group let

N_i = total number of individuals

p_i = prevalence

M_{i1} = number of people in the primary investigation (all of whom were examined)

$M_{i2} = N_i - M_{i1}$ = number of primary non-participants

m_{i2} = number of people examined among the primary non-participants

x_{i1} = number of individuals in the primary investigation with a lesion

x_{i2} = number of individuals in the secondary investigation with a lesion

p_{i1} = prevalence in the primary investigation

p_{i2} = prevalence in the secondary investigation

Assuming that the m_{i2} individuals examined in the secondary investigation constitute a simple random sample from the primary non-participants, the prevalence in the i:th demographic group is

$$p_i = \frac{1}{N_i}\left(x_{i1} + \frac{M_{i2}}{m_{i2}} x_{i2}\right)$$

yielding the prevalence for a combined demographic group

$$p = \frac{N_1 p_1 + N_2 p_2 + \ldots + N_k p_k}{N_1 + N_2 + \ldots + N_k} =$$

$$= \frac{1}{\Sigma N_i}\left(\Sigma x_{i1} + \Sigma \frac{M_{i2}}{m_{i2}} x_{i2}\right). \qquad (1)$$

In order to make statistical comparisons with other similar investigations meaningful, the interrogated population is regarded as a random sample from an infinite population. Then x_{i1} and x_{i2} are binomially distributed given the sample sizes M_{i1}, M_{i2} and m_{i2}. Hence the standard error for p_i is

$$SE\ (p_i) = SE_i = \frac{1}{N_i}\sqrt{M_{i1} p_{i1}(1-p_{i1}) + \left(\frac{M_{i2}}{m_{i2}}\right)^2 p_{i2}\,(1-p_{i2})}$$

and the standard error for the prevalence p of a combined demographic group

$$SE\ (p) = \frac{1}{N_1 + N_2 + \ldots N_k} \cdot \sqrt{N_1^2 (SE_1)^2 + N_2^2 (SE_2)^2 + \ldots + N_k^2 (SE_k)^2}.$$

Note. If interest is focused only on the finite population in the investigated area, the primary investigation is a total one. Hence random errors arise only from the secondary investigation making

$$SE\ (p_i) = \sqrt{\frac{M_{i2} - m_{i2}}{M_{i2} - 1} \cdot \frac{p_{i2}\,(1-p_{i2})}{m_{i2}}} \cdot \frac{M_{i2}}{N_i}$$

Assuming $p_{i1} = p_{i2}$ and $m_{i2}/M_{i2} = 1/6$ in all groups, it can be shown that the standard error for a combined prevalence is about 18 % smaller than when assuming infinite populations.

Prevalence differences between sexes

Two different tests of significance depending on the numerical value of the prevalences were applied.

(i) The total prevalence exceeds or equals 2 %. Then the total number of individuals with a lesion exceeds about 15 both among males and females in the secondary investigation, and so the crucial second term in formula (1), and thus the prevalences of males (p_M) and females (p_F) are approximately normally distributed.

The null hypothesis, no difference in prevalence between males and females, is tested by means of the quantity

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

which is approximately N (0,1)-distributed if the null hypothesis is true.

(ii) The total prevalence is lower than 2 %. Then it cannot be assumed, that z is normally distributed. However, by assuming that the prevalence in the primary and secondary investigations are equal, sex differences can be tested by simple homogeneity tests.

n_M = total number (= 10,036)

n_F = total number (= 10,297)

y_M = total number

y_F = total number

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No lesion	n_M

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Dem. group	Numb: No. of people	Pr in
1	N_1	
2	N_2	
14	N_{14}	
	Total N	

n_M = total number of males examined (= 10,036)

n_F = total number of females examined (= 10,297)

y_M = total number of males with lesion

y_F = total number of females with lesion.

The findings can be summarized in the following two-by-two table:

	Males	Females
Lesion	y_M	y_F
No lesion	$n_M - y_M$	$n_F - y_F$

and the hypothesis $p_M = p_F$ is then tested by means of the ordinary chi-square-test or if necessary Fisher's exact test.

Note. The test of prevalence differences between sexes according to (ii) is less powerful than the test according to (i). Because of this and the extra assumption in (ii), the significance test based on prevalences smaller than 2 % should be evaluated with caution.

Prevalence differences between the primary and secondary investigations

In all, 14 different demographic groups with the following characteristics can be recognized

	Number of examined people			
Dem. group	No. of people	Primary invest.	Secondary invest. Group SI	Group SII
1	N_1	M_{11}	m_{1I}	m_{1II}
2	N_2	M_{21}	m_{21}	m_{2II}
|	|	|	|	|
14	N_{14}	M_{141}	m_{14I}	m_{14II}
	Total N			

	Prevalences		
Dem. group	Primary invest.	Secondary invest. Group SI	Group SII
1	p_{1p}	p_{1I}	p_{1II}
2	p_{2p}	p_{2I}	p_{2II}
|	|	|	|
14	p_{14p}	p_{14I}	p_{14II}

The comparison between the primary investigation and the two groups of non-participants are made after matching as to age and sex against the total population. Hence, the mean differences are:

$$d_I = \frac{1}{N}\Sigma N_i\,(p_{iI} - p_{ip}),$$

$$d_{II} = \frac{1}{N}\Sigma N_i\,(p_{iII} - p_{ip}).$$

The standard error of d_I is

$$SE\,(d_I) = \frac{1}{N}\sqrt{\Sigma N_i^2\left(\frac{p_{iI}\,(1-p_{iI})}{m_{iI}} + \frac{p_{ip}\,(1-p_{ip})}{M_{iI}}\right)}$$

SE (d_{II}) is calculated analogously.

If the total prevalence exceeds or equals 2 % the hypotheses

$d_I = 0$ and $d_{II} = 0$ are tested by normal tests assuming that d_I and d_{II} are approximately normally distributed

If the prevalence is lower than 2 %, all demographic groups are combined and the differences are tested by means of chi-square-tests or if necessary Fisher's exact test.

Reproducibility of registrations

Lesions were registered at two separate examinations (I and II) of the same individuals. It cannot be assumed, that I and II were performed under identical

conditions, nor that either of the examinations was completely correct. These circumstances make an exact determination of the reproducibility impossible. Hence, the conditional probability is preferably used, stating that the same lesion was registered in both I and II if it was noted at any of the two examinations.

This probability and its standard error are calculated below. The possible outcome of the registrations can be summarized in the following two-by-two table, where − indicates negative finding, + positive finding and n_{ij} the number of cases.

		II: −	II: +
I	−	n_{11}	n_{12}
	+	n_{21}	n_{22}

The conditional probability that I and II both are positive if one of them is positive is estimated by

$$P_c = \frac{n_{22}}{n_{12} + n_{21} + n_{22}}$$

which has the standard error

$$SE\ (P_c) = \sqrt{\frac{P_c\,(1 - P_c)}{n_{12} + n_{21} + n_{22}}}$$

VALIDITY AND RELIABILITY

The general organization of the present study in principle concerns two fundamental aspects. The study as such should be practically feasible, and the results obtained should be as valid and reliable as possible. Any statement as to the objective (or true) presence or absence of any oral mucosal lesion is based on a prior appreciation of what is to be considered as normal. Uncertainty on this level will give rise to false negative and false positive registrations. True, as well as false positive registrations will subsequently be labelled with diagnoses which in themselves can be correct or not. This gives four alternatives, among which only one is true. It is clear, that the likelihood to reach this final objective result is influenced by many factors, and that the outcome will be strongly dependant on what particular lesion is registered and diagnosed.

To create as good conditions as possible for making valid and reliable registrations, specific preparatory measures were undertaken. Some of these, regarding equipment and criteria, have been detailed already. However, some additional measures were also undertaken.

Some months before the beginning of the investigation, a study period was spent at the Department of Oral Pathology, The Royal Dental College, Copenhagen. Then, a general adaptation of the author's way of diagnosing a number of oral mucosal lesions was obtained by having several dentists simultanously evaluate lesions in attending patients. This adaptation became detailed as regards:

a) minimal degree of "whiteness" necessary to establish a diagnosis of leukoplakia (see Fig. 11, Appendix)
b) appearance of different types of leukoplakia, lichen planus and chronic candidosis.

Color photographs of typical lesions, taken with identical photographic equipment and film as later used in the investigation, were procured so that an accurate comparison with subsequent findings could be made.

To what degree the precautions undertaken contributed to ensure high validity and reliability of the results was evaluated as follows.

Table 7. *Compl... concerning some...*

Lesion
Acute pseudome...
Chronic candido...
Papilloma
Preleukoplakia
Leukoplakia
Snuff dipper's l...
Leukoedema
Frictional kerat...
Ulcus mucosae
Mucocele
Fibroepithelial ...
Geographic sto...
Lichen planus

[1] All specimens ...
[2] Smears from ... most cases.

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Table 7. *Comparison between clinical diagnosis (C2) and final clinical histologic diagnosis (CH2) concerning some lesions from which tissue specimens were obtained.*

Lesion	Lesions with diagnosis C2	Lesions where C2 was confirmed to CH2		Lesions with diagnosis CH2 without corresponding initial diagnosis C2
	No.	No.	%	No.
Acute pseudomembranous candidosis[1]	14	14	100	0
Chronic candidosis[2]	103	100	97	6
Papilloma	11	11	100	0
Preleukoplakia	18	18	100	0
Leukoplakia	179	166	93	8
Snuff dipper's lesion	118	118	100	1
Leukoedema	3	3	100	0
Frictional keratosis	12	11	92	2
Ulcus mucosae oris NOS	12	11	92	0
Mucocele	6	3	50	0
Fibroepithelial polyp	29	27	93	3
Geographic stomatitis	4	4	100	0
Lichen planus	170	165	97	9

[1] All specimens consisted of smears.

[2] Smears from atrophic and nodular leukoplakias included. Both smears and biopsies were taken in most cases.

Inter-examiner variability

For evaluation of the criteria, and the diagnoses founded on these criteria, a measurement of inter-examiner variability concerning all lesions encountered in the investigation would have been desirable. This arrangement was not feasible.

Concerning two lesions, denture stomatitis and focal epithelial hyperplasia, the differences in registration and labelling between the author and two control examiners were estimated.

As regards denture stomatitis a clinical blind test was performed together with a colleague who previously had taken part in epidemiologic studies of this lesion. Thirty individuals with full upper dentures, appearing during one regular day of examination, were evaluated. Only one divergent registration was noted; the author registered 20 and the control examiner 19 cases of denture stomatitis.

The cases of focal epithelial hyperplasia were examined by an oral pathologist, who was especially familiar with this lesion. This examination took place after the completion of the field work and was performed on the basis of color photographs and biopsies. There was agreement between the author and the pathologist in 17 of 18 lesions examined.

Comparison between clinical diagnoses and histologic descriptions

In those lesions from which tissue specimens were procured, a definite diagnosis, CH2, was obtained after evaluation of the histologic findings. Irrespective of the histologic information a clinical diagnosis, C2, had also been obtained. By comparing CH2 and C2 an assessment of the validity of the diagnosis settled without complementary histologic information could be made. Table 7 shows the result of such a comparison for some of the

Table 8. *Number of lesions registered in primary examination (I) and at re-examination (II) in 256 individuals. + = positive finding, — = negative finding. P = probability for a lesion being registered in both I and II if it was registered in any of the examinations. SE = standard error of the mean.*

Lesion	Number of lesions registered						P	SE
	I+	II+	I+/II+	I—/II—	I+/II—	I—/II+		
Herpes labialis	9	9	9	247	0	0	1.00	0
History of herpes labialis	43	42	38	209	5	4	0.81	0.035
Intraoral herpetiform lesion	1	1	1	255	0	0	1.00	0
Chronic candidosis	52	52	52	204	0	0	1.00	0
Papilloma	7	7	7	249	0	0	1.00	0
Focal epithelial hyperplasia	9	9	9	247	0	0	1.00	0
Haemangioma	1	1	1	255	0	0	1.00	0
Gingival fibromatosis	1	1	1	255	0	0	1.00	0
Excessive melanin pigmentation NOS	29	35	28	220	1	7	0.78	0.043
Gingival cyst	1	2	1	254	0	1	0.50	0.250
Recurrent aphthae	3	2	2	253	1	0	0.67	0.178
History of recurrent aphthae	25	25	21	227	4	4	0.72	0.053
Preleukoplakia	38	44	33	207	5	11	0.67	0.044
Leukoplakia	123	122	122	133	1	0	0.99	0.005
Leukokeratosis nicotina palati	14	14	13	241	1	1	0.87	0.053
Snuff dipper's lesion	42	42	42	214	0	0	1.00	0
Leukoedema	100	112	94	138	6	18	0.80	0.023
Frictional keratosis	25	22	20	229	5	2	0.74	0.054
Cheek and lip biting	10	10	8	244	2	2	0.67	0.089
Traumatic ulcer	10	10	9	245	1	1	0.82	0.071
Ulcus mucosae oris NOS	6	6	4	248	2	2	0.50	0.125
Mucocele	4	4	3	251	1	1	0.60	0.148
Fibroepithelial polyp	23	24	21	230	2	3	0.81	0.048
Denture hyperplasia	19	20	19	236	0	1	0.95	0.029
Flabby ridge	26	26	25	229	1	1	0.93	0.029
Denture stomatitis	83	87	79	165	4	8	0.87	0.021
	17	21	16	234	1	5	0.73	0.060

Cheek and lip biting	10	10	8	244	2	2	0.67	0.089
Traumatic ulcer	10	10	9	245	1	1	0.82	0.071
Ulcus mucosae oris NOS	6	6	4	248	2	2	0.50	0.125
Mucocele	4	4	3	251	1	1	0.60	0.148
Fibroepithelial polyp	23	24	21	230	2	3	*0.81*	*0.048*
Denture hyperplasia	19	20	19	236	0	1	0.95	0.029
Flabby ridge	26	26	25	229	1	1	0.93	0.029
Denture stomatitis	83	87	79	165	4	8	0.87	0.021
Angular cheilitis	17	21	16	234	1	5	0.73	0.060
Atrophy of tongue papillae, unspecified	22	23	22	233	0	1	0.96	0.024
Glossitis, unspecified	2	2	2	254	0	0	1.00	0
Geographic tongue	25	26	25	230	0	1	0.96	0.022
Geographic stomatitis	1	1	1	255	0	0	1.00	0
Plicated tongue	24	24	23	231	1	1	0.95	0.026
Coated tongue	11	11	10	244	1	1	0.83	0.066
Hairy tongue	2	2	2	254	0	0	1.00	0
Lichen planus	84	84	84	172	0	0	1.00	0
Fordyce's condition	216	231	213	22	3	18	0.91	0.019
Amalgam tattoo	33	35	28	216	5	7	0.70	0.047

lesions included in the investigation. In addition, the number of cases in which a definite diagnosis, CH2, was made without a comparable clinical diagnosis, C2, initially being assigned to the lesion, is also shown.

In general, the agreement between the clinical diagnosis, C2, and the diagnosis based on both clinical and histologic observations, CH2, was good. The main exception was the lesion mucocele. Of the three lesions, that could not be verified histologically, two were found to be gingival cysts and the third lesion could not be classified.

Evaluation of the results in Table 7 must be made against the background of the criteria applied when collecting the tissue specimens. One of these was uncertainty as to the diagnosis. The values shown for agreement, therefore, are thought to be minimal values for most of the lesions.

Intra-examiner variability

To obtain an understanding of the size of the random errors in the registration of the lesions, re-examinations were performed. It was regarded as unpractical to recall a large number of individuals to the examination locales for the sole aim of performing these re-examinations. Hence, they were undertaken on patients who had obtained appointments for biopsies and returned for this a few days later.

During the period August 8, 1973—January 22, 1974, 256 individuals with patient consecutive numbers between 8,934 and 18,486, i.e. persons in the latter part of the primary investigation, were re-examined. Lesions registered in the primary investigation and at re-examination are summarized in Table 8. The probability of a lesion found at one of the occasions also being detected on both occasions is shown with the corresponding P value. The greater the P value, the greater the probability that the lesion was noted on both occasions. It should be noted that the P values for some lesions are followed by large standard errors. These P values should be evaluated with caution. This also applies to lesions with a value of 1.00 and which were registered in only a few instances.

For the more frequently encountered diagnoses, the differences between the two examinations were largest for preleukoplakia, cheek and lip biting, amalgam tattoo, history of recurrent aphthae, angular cheilitis, frictional keratosis and excessive melanin pigmentation NOS. These diagnoses thus were the ones for which clinical criteria were difficult to apply accurately or which were relatively difficult to detect. The differences reflect random errors in the diagnostic procedure and should not noticeably influence the prevalence figures, since over- and under-registration may be supposed to cancel out each other.

The results shown in Table 8 reveal, that in cases of discrepancy the largest number of lesions were most often registered at the re-examination. This could result from the fact that, unintentionally, somewhat longer time was alloted to the clinical inspection at the re-examination. The differences in the findings between the examinations can, for lesions such as ulcerations, also result from actual changes.

The way to select material for testing the intra-examiner variability may be thought to contain a certain bias, since the examiner was aware that the individuals had some lesion(s). The risk for bias, however

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bias, however, can be considered as minimal since 97 % of all individuals examined had at least one lesion and each person, on an average, had three lesions. Hence, it was impossible to memorize in detail each individual's oral mucosal status.

False negative or false positive registrations

A prerequisite for the exact estimation of systematic over- or underregistrations of lesions is that their objective or true presence is known. This knowledge is hardly attainable. However, for one of the lesions, a reasonably accurate prediction can be made as to which individuals should have the lesion. Thus, a characteristic lesion usually appears upon snuff dipping. Axéll et al. (1976) reported that when an average of 11 g/day of snuff is used, a clearly detectable, whitish lesion developed. A daily consumption of 11 g or more of snuff was stated by 551 individuals in the present study. A snuff dipper's lesion was not registered in 32 or 5.5 % of these 551 individuals. On the other hand, among 187 individuals or 1 % of the 18,701 individuals who denied using snuff, a lesion was noted. An explanation for the false negative diagnosis is conceivably that some snuff dipper's lesions were slight in nature. Also, the most common localization for this lesion, the labial groove, is relatively easy to overlook. The false positive diagnoses presumably reflect false information in the questionnaires.

Comparison between findings in primary and secondary investigation

Matched prevalences of some of the oral mucosal lesions found in the primary (P) and secondary (S) investigations are shown in Table 9. The findings from the secondary investigation are shown separately for Group SI and SII. Of 61 lesions investigated only those lesions are listed, where a statistically significant difference between the findings existed. The prevalences were obtained after matching as to age and sex against the total population.

The differences shown in Table 9 can represent:

1) a change in the examiner's sensitivity
2) real differences in the occurrence of the respective lesions
3) a combination of 1) and 2).

If 1) is applicable the results in the two groups of the secondary investigation should each diverge from those in the primary investigation. The information in Table 9 shows that significantly higher prevalences in this respect existed for preleukoplakia, leukoedema, frictional keratosis, traumatic ulcer, fibroepithelial polyp and denture-related mucosal petechiae.

Group SI consisted of individuals who, like those of the primary investigation, attended the examination locale upon a written invitation and who were examined under conditions identical to those existing in the primary investigation. This underlines that the differences between Group SI and the primary investigation as regards the lesions mentioned represent a change in the investigator's sensitivity and not a real difference in occurrence.

Some lesions were found to be more prevalent only in Group SI. Among these were excessive melanin pigmentation NOS and Fordyce's condition. These two lesions

Table 9. *Prevalences of oral mucosal lesions for which statistically significant differences existed between individuals in the primary investigation (P) and those in Group SI and/or Group SII of the secondary investigation. Prevalences obtained after matching as to age and sex against the total population.*

Lesion	P	Group SI	Group SII
Acute pseudomembranous candidosis	0.10	0.53**	0.23
Excessive melanin pigmentation NOS	9.41	13.33**	8.26
History of recurrent aphthae	16.72	15.21	12.27***
Preleukoplakia	5.54	7.49*	8.10*
Leukoplakia	3.33	4.91*	3.37
Leukokeratosis nicotina palati	0.77	1.72**	1.61
Snuff dipper's lesion	7.32	8.33	11.20**
Leukoedema	45.67	54.64***	54.93***
Frictional keratosis	3.98	8.25***	7.86***
Cheek and lip biting	4.51	5.09	7.58**
Traumatic ulcer	2.28	7.56**	7.66***
Ulcus mucosae oris NOS	1.05	1.95**	0.99
Fibroepithelial polyp	2.12	4.94***	5.29***
Denture hyperplasia	2.89	4.37*	3.93
Flabby ridge	8.30	10.55*	8.32
Denture stomatitis	15.17	16.65	18.67*
Denture-related mucosal petechiae	0.09	1.19***	0.47**
Atrophy of tongue papillae, median type	1.18	1.35	2.56*
Hairy tongue	0.34	0.65	1.42***
Fordyce's condition	81.21	88.32***	82.53

* = $p<0.05$ ** = $p<0.01$ *** = $p<0.001$

depend on good illumination for detection. That they were not found in correspondingly high prevalences in Group SII can depend on the fact that the latter group was examined under poorer lighting condition. If so, the higher prevalences for these two lesions should also be related to 1).

As regards differences in prevalences between either Group SI or SII as compared to the primary investigation (P), these may largely be explained in relation to differences in denture status and/or tobacco habits (see Table 10), i.e. as due to real differences. Real differences probably also explain the divergent prevalences found for cheek and lip biting and ulcus mucosae oris NOS.

The significantly lower prevalence of history of recurrent aphthae found in Group SII may be explained as follows. Group SII included institutionalized and sick individuals, many of which were difficult to interview, and this probably gave misleadingly low prevalences. Comparable differences were not found for history of herpes labialis, but this should, in view of the extraoral localization of herpes labialis, not be held as contradicting the consideration above.

In conclusion, this comparison indicates that real differences were present for the majority of the lesions listed in Table 9. However, the comparison also indicates that for nine lesions the examiner's sensitivity in registering was higher in the later

Table 10. *Percentage of denture wearers and habitual tobacco users among the individuals examined. The frequencies are also given separately for individuals examined in the primary investigation (P) and for Group SI and Group SII in the secondary investigation. Frequencies concerning P, Groups SI and SII obtained after matching as to age and sex against the total population.*

	Total population examined	P	Group SI	Group SII
Denture wearers				
Total	29.8	30.3	31.8	30.4
Males	27.1			
Females	32.4			
Habitual tobacco users				
Smokers				
Total	35.2	34.3	40.9	44.2
Males	42.2			
Females	28.3			
Snuff dippers				
Total	7.1	7.2	7.7	10.6
Males	14.2			
Females	<0.1			
Chewers				
Total	0.2	0.2	0.1	0.1
Males	0.4			
Females	<0.1			

part of the study. When considering the total prevalences of these lesions it should, therefore, be kept in mind that a certain underregistration may have been present.

Errors due to final non-participation

In the statistical analysis of the results, individuals who did not participate, representing 10.3 % of the total population, were considered, from a prevalence point of view, as if they did not differ from those examined in the secondary investigation. The errors arising from this way of calculation are difficult to determine. By analysing the reasons for non-participation in Group SI and SII as well as for the final non-participation some estimate can, however, be obtained.

Group SI, comprising 55.7 % of those examined in the secondary investigation, consisted of individuals who appeared for examination after a second summons had been sent. These persons, consequently, had a generally positive attitude to participation and also practical possibilities to attend. Comparable characteristics for the final non-participants can to a large extent be supposed to exist among those, who had changed residence, were temporarily away or for "other reasons" could not attend. Individuals offering these reasons for non-participation represented 53.2 % of the final non-participants (Table 6 p. 22). Considering this, and the fact that the final non-participants were relatively

few in number, any errors that might have arisen from the statistical treatment of the final non-participants are judged as only marginally influencing the results.

Errors in data handling

The routines for data handling involved many separate procedures. In order to obtain an estimate of errors arising in this context, a comparison was made between data noted on the forms and corresponding data from the computer out-put. All stored data registered for a total of 100 randomly chosen individuals were taken out of the computer and manually compared with the information on the forms. In coded form, the latter corresponded to 8,026 data. The comparison revealed 3 divergent recordings representing a frequency of 0.04 %.

This test did not include those errors that could arise at the verbal transferral of information between the examiner and the dental nurse. Errors in this regard have, however, partly been expressed when testing intra-examiner variability.

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RESULTS AND DISCUSSION

The results of the study are presented below under headings which equal diagnostic labels. These are followed by prevalence figures pertaining to the total population (T), to males (M) and females (F). Dashes indicate that no lesions were found. The appearance of the lesions is briefly commented on and the prevalence figures are discussed in relation to previously reported findings. When deemed appropriate prevalences within defined age strata are also presented and discussed. All figures in *italics* have been calculated from information in the studies mentioned.

The results are also presented in Table 11, Appendix. There, standard errors of the mean, differences in prevalences between sexes, and results of significance tests based on these differences are also shown.

Prevalences are not presently correlated with specific intraoral factors such as, for instance, tobacco habits and denture wearing. Such analyses involve multifactorial calculations, prior to which additional qualitative and quantitative aspects —as e.g. oral hygiene, denture status and overall presence of Candida albicans— have to be further considered.

Herpes zoster

T < 0.01 % M 0.01 %; F —

One case of herpes zoster was found. The lesion occurred in the right cheek of a 26 year old male and was accompanied by moderate pain.

No previous reports concerning the prevalence of intraoral herpes zoster were found in the literature. The incidence on the skin has been estimated to 1—34 per 10,000 (for review see Hudson and Vickers 1971). Among 191 patients with herpes zoster at a dermatology clinic in Bologna, Italy, Bonelli (1969) found that about 7.5 % had lesions in the buccal or pharyngeal region.

Herpes labialis and History of herpes labialis

T 17.38 % M 16.04 %; F 18.71 %

The total prevalence is lower than in previous investigations. Ship et al. (1960, 1967) reported prevalences of 38.2 % and 31.5 % among students, and of 44.6 % among hospitalized patients. Embil et al. (1975) performed a world-wide interview and registered, among 635 armed-forces recruits and 9,897 health-profession students, a prevalence of 33.2 % in males and 28.0 % in females. In Europe, the figures were 31.1 % and 30.8 %, respectively. Young et al. (1976) reported 16.3 % among students and a prevalence of 30.7 % in faculty and staff members at a dental school.

Several explanations can be offered for these differences. One can be that previous investigations mostly refer to relatively young patient groups. However, the pre-

sent prevalences varied only slightly between the various age groups. The lowest prevalence was seen in the age group 15—24 years (15.03 %) and the highest in the group 35—44 years (20.22 %). Alternatively, the methods of selection for the previously investigated populations may provide an explanation. Student populations were thus mainly investigated, and it is possible that such individuals are more susceptable to herpes labialis. A third explanation for the higher prevalences noted in previous investigations may be that "lifetime prevalences" were reported, while in the present study only those individuals were registered who reported themselves as having had lesions during the previous two years. The purpose of this way of registration was to obtain an awareness of which individuals still had the disease, i.e. now and then developed rashes. This is considered to be a better measure of the prevalence than "lifetime prevalence", which mainly represents a cumulative incidence.

Intraoral herpetiform lesion

T 0.33 % M 0.28 %; F 0.37 %

Recurrent herpetiform eruptions (Cooke 1960) or herpetiform ulcers (Lehner and Sagebiel 1966) consist of small clustered sores, or sometimes vesicles, which may occur anywhere in the oral mucosa. A viral etiology is probable for this type of lesion, but it is still doubtful whether the virus belongs to the herpes simplex group. In contrast to recurrent aphthae, intraoral herpes simplex lesions are usually found on the keratinized mucous membrane. All 55 lesions in the present investigation were localized to the palate or the attached gingiva. In four patients, all of whom now and then having eruptions of herpes labialis, smears were taken from vesicles. Three of these revealed cellular changes similar to those found in herpes labialis. In this respect, agreement exists between these lesions and four cases reported by Griffin (1965). As recurrent intraoral herpetiform eruptions and recurrent intraoral herpes simplex lesions appear to be clinically very similar, the working name intraoral herpetiform lesion was adopted for this investigation.

Herpangina

T 0.03 % M 0.01 %; F 0.05 %

Herpangina is an infectious disease, which usually affects children (Zahorsky 1924, Kramer 1967), but which may also be seen in adults (Pindborg 1973). It does not in general invite diagnostic difficulties.

Acute necrotizing ulcerative gingivitis

T 0.05 % M 0.05 %; F 0.04 %

Pindborg (1951) divided ulcero-membranous gingivitis into four clinical types, i.e. incipient, acute, subacute and chronic. The present criteria correspond to the incipient and acute types, and the three cases found in this study belonged to the former type. As to the present prevalence, it appears somewhat lower than what can be estimated from the study by Skach et al. (1970) in Prague, Czechoslovakia. The latter authors found an incidence during 10 years of 8.3, 4.8 and 2.1 per 10,000 inhabitants in the age groups of 15—19, 20—24 and 25—29, respectively. In other age groups, the incidence was insignificant. Pindborg (1951) found a prevalence of 3.5 % for incipient and acute ulcero-membranous gingivitis among 6,960 Danish navy recruits, and underlined that, among other factors, oral hygiene and tobacco habits of the lesion. sidered, as the susceptibility necrotizing ul the age of 20

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tobacco habits bear heavily in the etiology of the lesion. Age should also be considered, as there seems to be an increased susceptibility for development of acute necrotizing ulcerative stomatitis around the age of 20 years (Skach et al. 1970).

Acute pseudomembranous candidosis

T 0.21 % M 0.13 %; F 0.28 %

Acute pseudomembranous candidosis, thrush, is most common among children, i.e. in a population that was not included in this study. However, the lesion is also found among older individuals, for instance, being treated with medicines such as antibiotics and corticosteroids (Pindborg 1973).

All lesions registered in this study were found among denture wearers. The general health status of these individuals was not analysed. Most of them, however, showed poor oral hygiene.

Smears were taken in 14 cases and all showed, although usually few in number, hyphae of Candida albicans. It is doubtful if the lesions were representative for the classic entity of acute pseudomembranous candidosis. Possibly, other microorganisms can give rise to plaque-like formations that are difficult to differentiate clinically from those predominantly inhabited by Candida albicans.

Chronic candidosis

T 0.36 % M 0.41 %; F 0.31 %

Several lesions of the oral mucosa are infected by Candida albicans. It is well documented, that denture stomatitis (Budtz-Jørgensen and Bertram 1970), leukoplakia (Cawson 1966), angular cheilitis (Cawson 1963), median rhomboid glossitis (Cooke 1975), and discoid lupus erythematosus (Schiødt et al. 1975) in varying degrees show the presence of a chronic candidal infection. Many such lesions may occur in one and the same individual and this condition has been called "les Candidoses à foyers multiples de la cavité buccale" by Cernéa et al. (1965) and chronic multifocal oral candidosis by Storgård-Jensen and Holst (1969). In the present study the above mentioned lesions have, as regards prevalences, been grouped under the respective basic diagnoses, and an estimate of the total prevalence for chronic oral candidal infection should thus encompass a large number of lesions.

The specific heading of chronic candidosis, as applied in this study, describes alterations in which Candida infection has been highly suspected, but where no basic lesion could be identified. Among these alterations are, for instance, commissural lesions with clinical features similar to those found in central areas of atrophic or nodular leukoplakias (*vide infra*), and where basic characteristics fullfilling the criteria set for the diagnosis of leukoplakia were absent. The labels *chronic atrophic candidosis* and *chronic hyperplastic candidosis* thus describe alterations similar to the nonhomogenous features of atrophic and nodular leukoplakias, respectively.

Smears as well as biopsies were taken from 36 lesions compatible with the used criteria for chronic candidosis. All lesions, except three, proved positive for Candida albicans in at least one specimen.

Carcinoma

T < 0.01 % M —; F 0.01 %

Malignant oral lesions may appear in many various forms. Although carcino-

matous ulcers are often characterized as painless, indurated lesions with elevated margins, it has not been considered appropriate in this study to establish a set of clinical criteria for a diagnosis of carcinoma.

One case of adenoid cystic carcinoma of the palate was encountered. An 80 year old woman presented with a small ulcer under her upper full denture. The ulcer occurred together with a slight elevation of the palate, and the patient was immediately referred for further examination.

Most epidemiologic studies on oral cancer have been performed in India, and prevalences between 0.05 and 0.24 % have been reported (Pindborg et al. 1966, Zachariah et al. 1966, Mehrotra 1969, Mehta et al. 1971, Smith et al. 1975). Ross and Gross (1971) examined 14,749 individuals in Brooklyn, New York, and found one case of oral carcinoma.

The number of cases in the above mentioned studies is small. It is, nevertheless, possible to state that the findings are roughly in agreement with available cancer statistics in the respective countries (Pindborg 1963, Pindborg 1965, Smith 1973, Söder 1973).

Papilloma

T 0.10 % M 0.12 %; F 0.08 %

The common wart, verruca vulgaris, has been included under the diagnosis of papilloma as these lesions sometimes are impossible to differentiate clinically or even are identical. One lesion at the border between the skin and the prolabium was included in the material. The other lesions were found on the mucosa.

The prevalence of 0.10 % should be compared with that of *0.4 %* found by McCarthy (1941) among 927 referred patients at a dermatology clinic. A prevalence of *2.9 %* for papilloma and verruca vulgaris was found by Hakemer et al. (1971) among 307 schoolchildren treated at a dermatology clinic because of oral mucosal lesions. Bhaskar (1968) investigated 785 old age people and found a very high prevalence of 6.2 % for papilloma.

Focal epithelial hyperplasia

T 0.11 % M 0.09 %; F 0.12 %

The lesion focal epithelial hyperplasia is probably of a viral origin (Praetorius-Clausen and Willis 1971, Hanks et al. 1972). Praetorius-Clausen et al. (1970) mentioned two different clinical types, and this has been accounted for in the present criteria (p. 27). Criterion 2 thus covers a relatively typical lesion, whereas criterion 1 is somewhat insufficient in order to distinguish this lesion from, for instance, fibroepithelial polyps. The diagnosis of focal epithelial hyperplasia was accepted on clinical grounds only if, in any particular individual, at least one lesion fullfilled criterion 2 or else the border between the lesion and the surrounding mucosa showed irregularities.

Focal epithelial hyperplasia appears to be most prevalent among American Indians and Eskimos (for review see Praetorius-Clausen 1973). The highest prevalences have been reported by Soneira and Fonseca (1964), who found 33.8 % among 160 Indians aged 4—18 years in Tokuko in Venezuela, and by Praetorius-Clausen (1972), who found 35.8 % among 246 Greenlandic Eskimos of all ages in Angmagssalik.

Only a few cases have so far been reported among Caucasians. Prevalence studies were undertaken by Praetorius-

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Haemangioma

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Clausen (1972) who, among 3,000 conscript soldiers, did not find a single case and among 322 Caucasian Danes on Greenland only one case, corresponding to a prevalence of 0.3 %.

Seventeen cases or 0.11 % were registered in the present study. The highest prevalence occurred in the age groups 45—54 and ≥ 75 years, 0.22 % and 0.40 %, respectively. This indicates a considerably less frequent occurrence than in South America and Greenland. This is also true if age distributions in the investigated populations are considered. A genetic and racial predisposition has been discussed in relation to focal epithelial hyperplasia (Witkop and Niswander 1965).

Lipoma

T 0.06 % M 0.06 %; F 0.05 %

Lipomas are, as a rule, easy to diagnose clinically but in exceptional cases difficulties may arise. Mucocele thus sometimes have a yellowish color and clearly visible blood capillaries in the covering mucosa. However, they usually have a tougher consistency than lipomas.

Haemangioma

T 0.08 % M 0.06 %; F 0.11 %

Superficial or small haemangiomas may sometimes be difficult to differentiate from varicose veins and pyogenic granulomas. Varicose veins are, however, usually multiple and show a deep blue color, and pyogenic granulomas are easily bleeding when manipulated.

McCarthy (1941) and Hakemer et al. (1971) reported prevalences of *1.0* % and *1.3* %, respectively. These high prevalences should probably be related to the fact, that they were found among referred patients. Pape et al. (1970), in their investigation among individuals at a home for the aged, found a prevalence of *1.2* %, i.e. more than ten times that found in the present study. This difference can hardly be explained as due to differences in age composition of the populations investigated. Haemangiomas are to a large extent hamartomas, most of which regress during the first years of life. Those persisting should be expected to be found in similar prevalences in all age groups and this was also noted in the present study. It is thus likely that the diagnostic difficulties mentioned above may explain the differing prevalences reported.

Lymphangioma

T 0.02 % M —; F 0.05 %

Only one case of lymphangioma was found, and this was verified histologically. The prevalence of lymphangioma was considerably less than that for haemangioma, and this is in accordance with the findings by McCarthy (1941). He reported prevalences of *0.2* % and *1.0* %, respectively, among patients referred to a dermatology clinic.

Gingival fibromatosis and localized enlargement e.g. tuberosity

T 0.17 % M 0.18 %; F 0.15 %

Among the 50 individuals registered under this diagnosis, a general gingival fibromatosis was found only in a few cases. The majority of the cases presented symmetrical, localized, gingival enlargements, similar to those sometimes described as "symmetrical fibromas". Rushton (1957) suggested that these enlargements

might better be described as partial, idiopathic, fibrous hyperplasias.

Although special efforts to reveal heredity were not undertaken, two cases of gingival fibromatosis were found in a father and his son. None of the 50 individuals presented clinical signs of the presence of a syndrome involving, for instance, hypertrichosis and mental retardation and this is in accordance with the findings of Srsen and Mocik (1973).

Excessive melanin pigmentation NOS

T 9.94 % M 10.49 %; F 9.40 %

Melanin pigmentation of the oral mucosa occurs in several morphologic variants. Apart from those described by Eleutério (1969), this study also included diffuse, extended pigmentations. No attempt was made to investigate whether the recorded pigmentations were related to any general disease (for review see Dummet and Barens 1971). An application of the DOPI-system when registering local pigmentation (Dummet and Gupta 1966) was considered too time-consuming.

Oral pigmentations of melanin show a racial variation. The highest prevalences have been documented among aborigines in Australia, 77.6—100 % (Heithersay 1959, Reade 1962, Brown 1964), bantus in South Africa, *98.4* % (van Wyk 1970) and Greenlandic Eskimos, 98.1 % (Jakobsen 1968). Among Negroes in the United States, a prevalence of around *95* % has been reported by Monash (1932), and, as regards gingival localization, a prevalence of 67 % by Dummet (1946). In a predominantly Negro population, 6—18 years of age, in the Virgin Islands, 83.48 % of gingival pigmentation has been reported by Marshall-Day and Shourie (1950). The prevalence among Japanese has been reported to be 5 % by Fujibayashi (1940) and *36.2* % by Ando et al. (1956). Steigmann (1965) found a prevalence of 68.65 % among Jewish Yemenite children.

Investigations among Caucasians are infrequent. Fry and Almeyda (1968) found the prevalence of buccal pigmentation to be 38 % among colored people in London and the corresponding figure among white people to be 5 %. No gingival pigmentation was registered among the latter people. This is somewhat surprising as the gingiva is held to be the most common localization for oral pigmentation (Monash 1932, Eleutério 1969). Eleutério (1969) investigated 991 individuals aged 5—14 years, and found 12.5 % among 490 white, 70.4 % among 108 yellow and 93.2 % among 393 black people. A corresponding figure in the present study is the prevalence of 9.28 % in the youngest age group, 15—24 years.

One case of intraoral pigmented naevus was found, and this was verified microscopically. The prevalence of naevi has previously been investigated by King et al. (1967), who did not find any case among 1,279 white people in the United States, but 3 cases among 2,912 Negroes.

The present sex distribution, with a slight predominance among males, is in agreement with the findings by Eleutério (1969). Also Dummet and Gupta (1966) found higher DOPI-values among male Negroes.

Gingival cyst

T 0.03 % M 0.02 %; F 0.05 %

It is often impossible, in a clinical setting, to accurately differentiate gingival cysts from mucoceles and lateral parodontal cysts. Sometimes even inflammatory cysts

may simulate true gingival cysts (Moscow et al. 1970). Among the six cases registered, one may eventually, due to its size, be suspected to have been of inflammatory nature. This case was, however, recorded as a gingival cyst, as it fullfilled the clinical criteria for this type of lesion.

Chronic desquamative gingivitis

T 0.01 % M —; F 0.02 %

This lesion has been related to a number of diseases. McCarthy et al. (1960) suggested a classification comprising five different groups: dermatoses, hormonal influences, abnormal response to irritation, idiopathic, and chronic infections. Three cases were registered in the present study, all of them in women. If these lesions should be grouped according to McCarthy et al. (1960), they should in all likelihood be classified as connected with hormonal disturbances or as being idiopathic. None of the women showed symptoms of dermatoses or infections, e.g. by Candida albicans.

Recurrent aphthae and History of recurrent aphthae

T 17.70 % M 16.81 %; F 18.56 %

Recurrent aphthae appear clinically in several stages (Stanley 1972). In addition to the classical clinical lesion, this study also included early and late stages presenting as macules or papules.

The prevalence of recurrent aphthae was 2.00 %. Grabowski (1974) reported a prevalence of 1.3 % in a randomized population over 65 years of age in Denmark. This figure is in very good agreement with the present result, which for the age group of 65—74 years gave a prevalence of 1.95 %.

The present choice to use the diagnosis of history of recurrent aphthae together with that of recurrent aphthae was made in order to give a better comprehension of the number of individuals who can be regarded as suffering from aphthae. This delineation is also considered more valid than cumulative figures relating to "lifetime prevalence", which have sometimes been used. The latter choice, together with the fact that most previous studies have concerned special populations consisting of narrow age strata, probably explains why many of these studies have yielded considerably higher frequencies of history of recurrent aphthae. Thus, Ship et al. (1960, 1967) reported prevalences of *54.4* % and 66.2 % among medical and dental students, respectively. Among dental students, Shapiro et al. (1970) and Donatsky (1973) reported prevalences of *37.6* % and 56 %. Embil et al. (1975) performed a world-wide comprehensive interview among health profession students and reported prevalences of 36.0 % among males and 36.7 % among females in Europe.

The prevalence of recurrent aphthae and history of recurrent aphthae varied considerably between different age groups, being 26.7 % in the group 15—24 years and 7.5 % in the group 65—74 years.

The necessity to consider type of population as well as age is illustrated in the study by Ship et al. (1967), who, among students, registered a prevalence of 66.2 % and among hospitalized individuals aged "36.5 ± 14.6" years, 13.2 %.

Lower prevalences than those referred to above have been reported by Sircus et al. (1957) among hospitalized and ambulatory patients of all ages, 19.3 %, and by Herrmann (1971) among dental school patients, 11.1 %. Fahmy (1976) reported a 5-year incidence of 27 % among 20,000

Arabs of various nationalities living in Kuweit. A very low incidence, 5 %, was reported among the subgroup of Bedouins.

The higher prevalence among females found in the present study is in accordance with most other studies (e.g. Ship et al. 1960, Spouge and Diamond 1963). Herrmann (1971) did not, however, find any sex difference. Discrepancies in this context might partly be explained on the basis of smoking habits. Shapiro et al. (1970) thus found a negative correlation between smoking and the presence of oral aphthae.

Periadenitis mucosa necrotica recurrens

T 0.01 % M 0.01 %; F 0.01 %

Recurrent aphthous ulcers in the oral cavity may occur together with more generalized diseases, as for example Behçet's syndrome (Francis 1970). Also oral aphthous ulcers may be manifest in severe forms, often referred to as periadenitis mucosa necrotica recurrens. This specific form has previously been considered diagnosable on purely clinical criteria (Hjørting-Hansen and Siemssen 1961, Graykowski et al. 1966).

Three cases were found in the present study. Two individuals showed acute, ulcerating lesions accompanied by severe pain. The third individual only showed macules and had no obvious subjective symptoms. The macules were found together with pronounced scarring and the lesion was thus considered as being in a stage of healing.

There are no previous prevalence figures available in the literature. In a study of 62 patients referred to a dental clinic because of recurrent oral ulcers, Graykowski et al. (1966) held 13 patients to suffer from periadenitis mucosa necrotica recurrens.

Preleukoplakia

T 6.35 % M 10.01 %; F 2.69 %

Leukoplakia

T 3.60 % M 6.08 %; F 1.15 %

The diagnostic criteria for leukoplakia were based on the definition used by Silverman et al. (1963), i.e. "any white patches or plaques on the oral mucous membranes that: cannot be removed by scraping; cannot be reversed by removing obvious irritants; and cannot be classified clinically or microscopically as another diagnosable disease". Although the present study did not offer practical possibilities to follow up the eventual reversal of any lesions, it was considered essential to exclude white lesions, which had an obvious relation to well defined local irritation. White lesions seen in connection with, for instance, sharp teeth or prosthetic appliances were thus registered as frictional keratoses (*vide infra*). Similarly, the specific lesion caused by snuff was registered as snuff dipper's lesion (*vide infra*).

Most definitions of leukoplakia emphasize a "color", i.e. white. Clinically, however, there is a wide variation within a spectrum from a slightly diminished translucence of the mucosa, over a subtle greyish appearance to a thick, undisputably white plaque. This fact has been taken into account in some previous epidemiologic studies, and the diagnostic label preleukoplakia has thus been introduced (Pindborg et al. 1968a, Mehta et al. 1971, Tyldesley 1971, Ramanathan et al. 1973). Precise criteria to enable the distinction between preleukoplakia and leukoplakia have usually not been stated. In the present study this was made subjectively as described above (p 36).

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cancerous condition, i.e. some of the lesions ultimately display the development of a carcinoma. The frequency of this outcome has been estimated at 4 % by Einhorn and Wersäll (1967) and at 4.4 % by Pindborg et al. (1968b). Endeavours to distinguish suspected lesions have often yielded subdivisions of the leukoplakias.

Aside from color variations in the greyish-white spectrum, leukoplakias thus often show reddish areas of atrophy or erosion, and surface irregularities recognized as nodules. A lesion displaying only atrophy or erosion has been designated here as an atrophic leukoplakia, a term comparable with the label "leukoplakia erosiva" used by Sugár and Bánóczy (1969) and, partly, with the "ulcerated leukoplakia" described by Mehta et al. (1971). If nodules were also present the lesion was designated a nodular leukoplakia, this being in accordance with Roed-Petersen et al. (1970). The last mentioned study also made use of the term "speckled leukoplakia" to describe lesions showing a sharply demarcated and elevated area with small white spots or nodules on an erythematous basis. No such lesion was found in the present study.

The nonhomogeneous areas within the atrophic and nodular leukoplakias show, clinically as well as histopathologically, striking similarities with the lesions of chronic oral candidosis. These types of leukoplakias have also been incorporated as entities in the diagnosis of chronic multifocal oral candidosis by Storgård-Jensen and Holst (1969).

The present total prevalence of leukoplakia, 3.53 %, is largely in accordance with previous findings in general populations. For instance, Bruszt (1962) found 3.6 % among 5,613 Hungarian villagers, Pindborg et al. (1968a) 4.6 % among 1,226 individuals over 20 years of age in New Guinea, Wahi et al. (1970) 5.2 % among 7,286 individuals aged 35 years or above in India and Mehta et al. (1971) 1.7 % among 50,915 Indian villagers above the age of 15 years.

Relatively large populations have been investigated at dental schools in India. Pindborg et al. (1965a, 1965b, 1966) found between 1.55 and 3.28 % in three investigations, each comprising 10,000 individuals, Zachariah et al. (1966) 2.36 % among 5,000 individuals and Srivastava (1973) 2.8 % among 8,428 individuals aged 18 years and above. Smith et al. (1975) found 11.7 % among 57,518 industrial workers over the age of 35 years, comprising 95 % males, in Gujarat, India.

Information on the prevalence of leukoplakia in Scandinavian populations is available in only few studies. Thus, Grabowski (1974) reported a prevalence of 1.3 % among 557 Danish individuals 65 years of age or above, and Chrigström et al. (1970) 3.0 % among 201 denture wearers living at home for old age people in Stockholm, Sweden. As seen in relation to age, leukoplakia in the present study was found in 2.1, 6.2 and 4.0 % in the age groups 25—34, 55—64 and 65—74 years, respectively.

There was a significant difference between prevalences of leukoplakia for males and females, around 6 % and 1 %, respectively. This is in accordance with previous findings (Bruszt 1962, Mangi et al. 1965, Pindborg et al. 1968a, Srivastava 1973). The difference has usually been related to a higher tobacco consumption among males. The importance of smoking is well underlined in some reports, where leukoplakia is most prevalent among females. This is so in studies from

Andra Pradesh in India, where especially women practice the habit of reversed smoking (Mehta et al. 1971, Pindborg et al. 1971). In contrast to other localizations in the oral cavity, leukoplakias in the floor of the mouth have been found to be more frequent among females than males in Denmark (Pindborg et al. 1972b). Cheroot smoking is a widespread habit among Danish women.

Very high prevalences of leukoplakia, between 12.0 % and 54.1 %, have been reported from Eastern Europe by Anastassow and Welikow (1969), Bieda et al. (1968), Ciechanowicz et al. (1968), Welikow (1968) and Smoljar and Granin (1971). These studies comprised special populations of industrial workers. It is difficult to evaluate those prevalences because diagnostic criteria and/or habits, as e.g. smoking, were only exceptionally accounted for. The high prevalence figures may, however, eventually be explained in relation to specific, initiating or predisposing, factors.

Preleukoplakia was found in around 6 % of the present population. This is higher than the figure for leukoplakia. The finding of a higher prevalence for preleukoplakia than for leukoplakia is in accordance with previous studies (Pindborg et al. 1968a, Mehta et al. 1969, Ramanathan et al. 1973). The distribution as to age for preleukoplakia corresponded to that for leukoplakia, with the highest prevalence in the age group 55—64 years. The total prevalence for preleukoplakia and leukoplakia was 8.49 % (males 13.58 %; females 3.43 %).

Leukokeratosis nicotina palati

T 1.12 % M 2.13 %; F 0.10 %

This lesion is directly related to smoking habits and then especially to pipe smoking (Kerr 1958, Chapman and Redish 1960, Forsey and Sullivan 1961, Saietz 1975). It is noteworthy, however, that similar lesions, although subtle, also have been registered among non-smokers (Cummer 1946, Sewerin 1973).

A slight reduction of translucence or a diffuse increase in whiteness of the palatal mucosa was not considered sufficient for a diagnosis of leukokeratosis nicotina palati. This should, in accordance with Pindborg et al. (1965a, 1965b, 1966), Mehta et al. (1971) include either fissuring or the presence of nodules with central, red dots. Previous investigations using similar criteria have shown prevalences largely in accordance with the present study. Pindborg et al. (1965a, 1965b, 1966) thus reported 0.15—0.71 % in three Indian populations, each consisting of 10,000 patients at dental schools, Zachariah et al. (1966) 0.20 % among 5,000 patients at a dental school in India and Ramanathan et al. (1973) *1.5 %* among 407 individuals aged 19—54 years in Malaysia.

Saietz (1975) investigated 3,819 Danes aged 15 years or above. She divided the lesions into two grades, and the criteria for her degree 2 are largely compatible with those used in this study. She found a total prevalence of 6.0 %, but in grade 2 only *1.4 %*. A similar subdivision was used by Cummer (1946) who, among 587 individuals at a dermatology clinic in the United States, found 39 cases corresponding to a prevalence of *6.6 %*. Six cases, or *1.0 %*, were of the "papillary" form, which is largely compatible with the criteria used in the present study.

Considerably higher prevalences have been reported among populations practicing "reversed smoking". This habit causes severe palatal lesions which should

be regarded as precancerous (Pindborg et al. 1971. Reddy et al. 1973). Reddy et al. (1973) found a prevalence of 17.83 % among 9,400 villagers of all ages in Visakhapatnam, India and Pindborg et al. (1971) 9.5 % among 10,169 villagers above 15 years of age in Andra Pradesh, India. The latter population is identical with one of the five populations investigated by Mehta et al. (1971). The other four were from districts, where the habit of "reversed smoking" is not generally practiced. In these districts the prevalence of leukokeratosis nicotina palati was only 0—0.3 %

The present sex difference is in accordance with previous knowledge and should be related to differences in smoking habits.

Snuff dipper's lesion

T 8.04 % M 15.94 %; F 0.19 %

Almost without exception snuff dipping gives rise to characteristic lesions of the mucosa. These lesions are most often whitish, but there may also be more subtle changes without color changes and with only slight wrinkling (Axéll et al. 1976). The recognition of a well defined irritation has excluded this lesion from the diagnosis of leukoplakia (*vide supra*), a differentiation which also seems warranted in the context of risk for impending malignancy (Roed-Petersen and Pindborg 1973 a, Smith 1975).

Prevalence figures for snuff dipper's lesion are almost totally lacking in the literature. Sandstead and Lowe (1953) reported 19 cases, or *3.4 %*, of "tobacco pouch type of keratosis" among 646 ambulatory patients at a mental hospital in Tennessee, the United States.

The present lesions were almost totally confined to males. This is in contrast to what has been found in, for instance, South Africa and the United States, where females are relatively frequent among snuff dippers (van Wyk 1965, Smith et al. 1970).

Leukoedema

T 48.89 % M 61.92 %; F 35.89 %

In previous investigations, leukoedema has been described in terms of a diffuse veil and/or a marked wrinkling of the mucosa (Pindborg et al. 1968a, Hamner et al. 1971, Borghelli et al. 1975). Sandstead and Lowe (1953) graded the lesions as slight, moderate or severe on criteria partly related to whether the lesion disappeared or not upon "stretching".

In this study, the diagnosis of leukoedema was applied when the mucosa showed a surface structure of fine folds, which upon opening of the mouth often are vertically orientated. These folds are easy to uncrease or to move around. The adequate registration is, as pointed out by Sandstead and Lowe (1953), very much dependent on good illumination. Differences in both criteria and investigatory conditions may to a large measure explain the highly variable prevalences of leukoedema reported in the literature.

Very low figures have been reported in studies from India, where Hamner et al. (1971) found 0.11 % among 50,915 villagers over the age of 15 years, Pindborg et al. (1965a, 1965b, 1966) 0.96—1.66 % in three populations, each consisting of 10,000 patients and examined at dental schools and Zachariah et al. (1966) 0.96 % among 5,000 patients over the age of 35 years at a dental school. Somewhat higher prevalences have been reported by Pindborg et al. (1968a) among 1,226

Papuans and New Guineans over 20 years of age, 10.0 %, Tyldesley (1971) among 402 English coal miners, *8.5 %*, Roed-Petersen and Pindborg (1973b) among 1,399 urban and rural individuals of both Asian and African descent in Uganda, 14.2 %, and Borghelli et al. (1974) among 2,174 20 year old males in Argentina, *5.1 %*.

Considerably higher prevalences have been reported from the United States. Martin et al. (1970) found *67.7 %* among 300 hospitalized Negroes, Martin and Crump (1972) 50.8 % among 1,000 Negro children and teen-aged patients and Martin (1973) *56.6 %* among 4,230 ambulatory and hospitalized Negroes of all ages. Sandstead and Lowe (1953) investigated outpatients at a mental hospital and found leukoedema in *42.6 %* of 437 white people and as high a prevalence as *90.0 %* among 209 Negroes.

The present prevalence of 48.89 % is in best agreement with the findings among whites in the American investigations. A similar figure, 40 %, has been found by Praetorius-Clausen (1974) in Denmark among marine conscripts. The prevalence among males in the age group 15—24 years was 65 % in the present study.

There was a significant difference between the prevalences for males, 61.92 %, and females, 35.89 %. This probably reflects differences in various habits. It is thus well known, that leukoedema is prevalent among users of tobacco (Pindborg et al. 1968a, Tyldesley 1971, Smith et al. 1975) and cannabis (Ebling et al. 1968/69).

Frictional keratosis

T 5.47 % M 6.29 %; F 4.66 %

Traumatic insults to the oral mucosa sometimes give rise to whitish lesions, which cannot be rubbed off. Cooke (1956) and Cawson (1969) designated such lesions as frictional keratosis and underlined that they sooner or later disappear after removal of the irritant. The present investigation did not, for practical reasons, offer any possibility of therapeutical interventions or follow-ups. It has nevertheless seemed important to apply, according to the set criteria, the diagnostic label frictional keratosis.

Cheek and lip biting

T 5.14 % M 4.86 %; F 5.46 %

This characteristic lesion can sometimes also be seen on the margins of the tongue. It appears upon repeated chewing of the mucosa and should probably better be called "habitual chewing of the oral mucosa". The variant "suctio mucosae oris" pointed out by Hjørting-Hansen and Holst (1970) has not been registered as a separate entity. In some cases this type of lesion has shown marginal areas of flaky desquamation and then accordingly been grouped as cheek and lip biting. Other cases have been grouped under frictional keratosis. Simple bite wounds have been categorized as traumatic ulcers.

The highest prevalence of cheek and lip biting, 11.39 %, was found in the youngest age group, 15—24 years, and in this group there was also a significantly higher prevalence among females than among males, 14.70 % and 8.25 %, respectively. The total prevalence diminished with increasing age, being 6.89 % in the age group 35—44 years and 0.88 % in the group $\geq$ 75 years. This decrease is in accordance with the results reported by Sewerin (1971) who investigated 8,589 persons of all ages attending The Royal Dental College in Copenhagen.

He found 42 cases, corresponding to a total prevalence of *0.49 %*. The highest prevalences were found in the age groups 15—19 and 20—24 years, 1.77 % and 1.20 %, respectively, without any sex difference. The large discrepancies in prevalence figures reported may primarily be related to dissimilar criteria. Subtle lesions without erythema or ulceration were thus included in the present study.

Traumatic ulcer

T 4.27 % M 4.26 %; F 4.27 %

Ulcus mucosae oris NOS

T 1.22 % M 1.84 %; F 0.61 %

Traumatic ulcers should heal rapidly after infliction. This criterion for a simple ulcer could, for practical reasons, not be applied in the present study where instead obvious anamnestic or examination data had to be relied upon. If there was any hesitation as to involvement of trauma, the lesion was put in the category of ulcus mucosae oris NOS. Traumatic ulcers thus included lesions caused by, for instance, biting and brushing, hot food or drink and overextended dentures. A relatively common type of ulcus mucosae oris NOS was a fissuring of the lip, which could not be related to angular cheilitis. Any ulcer, which aroused the slightest suspicion of malignancy, caused an immediate referral of the patient.

Prevalences of traumatic dental ulcer have been reported by Chrigström et al. (1970), who found *2.5 %* among 201 denture wearers aged 60 years or above and by Ritchie (1973), who registered 2 % among 300 geriatric patients. The present prevalence of ulcers caused by any trauma was 4.27 % and in the age group 65—74 years there was an even higher prevalence of 5.5 %.

Mucocele

T 0.11 % M 0.13 %; F 0.08 %

Superficially situated mucocele most often show a typical clinical appearance. They are usually described as whitish, pinkish or bluish (McCarthy and Shklar 1964, Cataldo and Mosadomi 1970, Pindborg 1973). Sometimes, especially in the floor of the mouth, mucocele may be yellowish and then mimic lipoma. However, the mucocele regularly is more firm at palpation. Deeply located lesions are clinically difficult to diagnose and non-biopsied lesions were thus accepted as mucoceles only if they varied in size from time to time, indicating an intermittent release of fluid material.

The prevalence figure is somewhat lower than that of 0.4 % reported by Bhaskar (1968) among 785 old age people. However, in the age group $\geq$ 75 years, 3 cases, corresponding to a prevalence of 0.4 %, were found in the present study.

Pyogenic granuloma

T 0.08 % M 0.03 %; F 0.13 %

The present diagnosis of pyogenic granuloma includes the clinically identical lesion which may be seen during puberty or pregnancy. It has further not been considered possible to distinguish clinically the peripheral giant cell granuloma from pyogenic granuloma.

Pyogenic granuloma is a well circumscribed, deeply red and freely bleeding tumor. No such typical lesion was found. All 18 lesions could rather be characterized as intermediate forms between fibroepithelial polyp and pyogenic granuloma.

The latter diagnosis was applied, as the lesions were more red than the surrounding normal mucosa and freely bleeding when manipulated.

Previous studies have most often concerned the pregnancy granuloma. The incidence has been reported to be between 0 % and 5 % (Hilming 1950, Monash 1931b). Shklar and Meyer (1965) describe the pyogenic granuloma as "an extremely common oral mucosal lesion" without giving or referring to any prevalence figures. McCarthy (1941) reported a prevalence of 0.2 % among referred patients at a dermatology clinic.

In this study the pyogenic granuloma was somewhat more common among females than among males. This is in accordance with the report by Angelopoulos (1971) on 46 new and 789 previously published cases.

Fibroepithelial polyp

T 3.25 % M 3.02 %; F 3.46 %

Fibroepithelial polyps are clinically very often called "fibromas". For most lesions, this is a misnomer. Barker and Lucas (1967) thus scrutinized 650 biopsies of localized, fibrous overgrowths from the palate, tongue, cheeks and lips, and found only two cases which could be labelled benign neoplasms, i.e. fibromas. Considering this low incidence of fibromas, and the fact that fibromas presumably are clinically indistinguishable from fibroepithelial polyps, the latter term has been used.

McCarthy (1941) reported the presence of 30 "fibromas" among 927 referred patients at a dermatology clinic in Boston, USA, i.e. a prevalence of *3.2 %* and Bhaskar (1968) 9 "fibromas", or 1.1 %, among 785 individuals at the US Old Soldier's Home, Washington, D.C., USA. These prevalences are roughly in agreement with that found for fibroepithelial polyp in this study.

Denture hyperplasia

T 3.41 % M 2.33 %; F 4.43 %

Two types of hyperplasias due to irritation from dentures have been recognized, one type associated with overextended flanges and another with suction chambers. Within the total prevalence, there was an obvious increase with advancing age. Thus, 6.31 % was found in the age group 55—64 years, 10.23 % in the group 65—74 years, and 11.45 % in the group $\geq$ 75 years.

Previous studies in Sweden have revealed highly variable results. Chrigström et al. (1970) found only *1.5 %* of vestibular hyperplasia and 1.0 % suction hyperplasia among 201 denture wearers of old age. Kristerson and Kvint (1969) reported prevalences of *11.4 %* and *25.9 %* in the upper and lower jaw, respectively, among 172 patients including 125 denture wearers at a hospital for the chronically ill. A population, similar to the last one, was investigated by Manderson and Ettinger (1975) in Scotland. They found only 4.5 % and 6.6 % hyperplasias in the upper and lower jaw, respectively, among 442 individuals with a mean age of 79 years, including 407 denture wearers. Among 260 patients at a dental school in Finland Mäkilä (1974) reported 8 % and 7 % of denture hyperplasia in the upper and lower jaw, respectively, among denture wearers.

Very low prevalences were reported by Pape et al. (1970), who at a German home for the aged found *2.5 %* among 1,502 individuals (60 % denture wearers), of

mean age 77.2 years, and 1.1 % among 468 alcoholics (29 % denture wearers), of mean age 43.9 years. Low prevalences were also registered by Ritchie (1973) who, among 300 geriatric patients of mean age 74 years, found 3.0 % denture hyperplasias.

A very high prevalence, *26.6 %*, was found by Grabowski (1974) among 557 Danes aged 65 years or above, 464 of whom were denture wearers.

Flabby ridge

T 8.57 % M 5.52 %; F 11.54 %

This lesion is sometimes included among denture hyperplasias (Nordenram and Landt 1969), but it may be of interest to consider it separately. Flabby ridge may thus primarily be looked upon as a sequela to alveolar bone resorption rather than as a hyperplasia of the oral mucosa.

Swedish prevalence studies of flabby ridge all concern highly selected populations of denture wearers attending prosthetic departments at dental schools. Nyquist (1952) found a prevalence of *7.2 %* among 1,090 patients aged 20 years or above. This prevalence is numerically similar to the present one, but it should be observed, that Nyquist examined denture wearers only. Higher prevalences are indicated in reports by Bergman et al. (1964, 1971) and Olsson and Bergman (1971) who investigated populations of 29—91 individuals.

Mäkilä (1974) found 19 % of flabby ridge in the upper jaw and 13 % in the lower jaw among denture wearers at a dental school in Finland.

Denture stomatitis

T 16.02 % M 12.27 %; F 19.65 %

In previous investigations, denture stomatitis has been classified in somewhat different ways. Bergman et al. (1964) only separated "healthy" and "inflamed" mucosa. Newton (1962) mentioned three different types, i.e. "pinpointed hyperemia", "diffuse erythema" and "granular inflammation", and Budtz-Jørgensen (1974) used a rather similar subdivision in terms of "localized simple", "generalized simple" and "papillomatous" stomatitis. The papillomatous type has often been registered separately under the name "(inflammatory) papillary hyperplasia" (Yrastorza 1963, Schmitz 1964, Guernsey 1965, O'Driscoll 1965, Lambson and Anderson 1967, Bhaskar 1968, Bhaskar et al. 1970, Ettinger 1975, Manderson and Ettinger 1975). Separate cases have also shown this type of change to be present outside the denture-covered mucosa (O'Driscoll 1965). The present choice has primarily been to recognize the three variants dealt with by Newton (1962) and Budtz-Jørgensen (1974). However, in view of the fact, that a "granular" or "papillomatous" appearance often occurs as an additive feature in both the localized and the generalized types of denture stomatitis, this appearance has always been registered in parallel with the two other types.

Comparisons with previous investigations are difficult to perform as these usually have been carried out on special populations consisting of, for instance, patients at prosthetic departments or old, institutionalized individuals (for review see Budtz-Jørgensen 1974, Grabowski 1974). There are, however, some studies on general populations. Swallow and Adams (1967), in collaboration with the Epidemiological Research Unit in the Rhondda Fawr-district in Wales, investigated 418 individuals aged 15—74 years and found denture stomatitis among

36.3 % of 171 full denture wearers. Grabowski (1974) investigated 560 individuals including 467 denture wearers over the age of 65 years in Denmark and found a prevalence of 53.6 %. The corresponding figure in the age group 65—74 years was around 36 % in the present study.

The papillomatous type of denture stomatitis represented around 12 % of all the present lesions which is lower than previously reported (Budtz-Jørgensen 1974, Grabowski 1974). This may largely be due to the somewhat dissimilar ways of typing the lesions. In studies, where the papillomatous type has been separately studied (as "inflammatory papillary hyperplasia"), prevalences between *1.7 %* and 2.9 % have been reported (Yrastorza 1963, Guernsey 1965, Bhaskar 1968, Manderson and Ettinger 1975). This is largely in accordance with the present prevalence of 2.1 % for papillomatous denture stomatitis. Ettinger (1975) reported a prevalence of 13.9 % among 700 hospitalized patients of mean age 61.9 years and belonging to lower socio-economic groups in Iowa, the United States. This high figure should probably be related to poor oral hygiene. The etiology of denture stomatitis is thus linked with both trauma (Nyquist 1952, Love et al. 1967, Budtz-Jørgensen 1970, Bergman et al. 1971) and candidal infection (Cawson 1965, Lehner 1965, Davenport 1970), and the latter is especially seen in connection with the generalized and papillomatous types of the lesion (Budtz-Jørgensen 1974).

The higher prevalence noted among females in this study is in accordance with some previous studies (Nyquist 1952, Olsson and Bergman 1971, Mäkilä 1974), but not with others (Pape et al. 1970, Grabowski 1974).

Papilliform hyperplasia of palate

T 0.03 % M 0.06 %; F —

In this study the term papilliform hyperplasia of palate designates a lesion which is not in contact with a denture. Such lesions have been described by e.g. Schmitz (1964) and Guernsey (1965). The latter author reported a prevalence of 0.2 % among 5,892 patients and this is around ten times the figure in the present study and in a study by Yrastorza (1963), who found one case among 5,059 patients.

Denture-related mucosal petechiae

T 0.39 % M 0.38 %; F 0.41 %

Thirty two individuals showed palatal changes of a petechial nature without any clinically obvious inflammation. Because of this, the lesion was not grouped together with denture stomatitis but registered separately.

Angular cheilitis

T 3.76 % M 2.95 %; F 4.59 %

This lesion has previously been subdivided on the basis of concommitant folding by Cawson (1963) and angulation from the commissure by Nyquist (1962). Such divisions were not applied in this study. The basic criterion for the registration of angular cheilitis was, in accordance with Mäkilä (1969), a discontinuity of the epithelium. In a few cases, this discontinuity was present only on the mucosa and not on the skin.

The etiology of angular cheilitis is most probably multifactorial, and the importance of vitamin and/or iron deficiencies and of denture-related factors, such as decreased vertical intermaxillary dimension, denture stomatitis and infection by Candida albicans, are usually stressed (for reviews see Turrell 1967 and Mäkilä 1969).

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These circumstances presumably underlie the fact that most previous studies have been performed on special populations. Nyquist (1962) found a prevalence of 30.0 % among 1,093 dental school patients aged 20 years or above having full dentures. Chrigström et al. (1970) found 7 % among 201 denture wearers at homes for the aged, and a similar figure 7.9 %, was found by Budtz-Jørgensen (1972) among 303 denture wearing patients with a mean age of 60.6 years at a dental school. Mäkilä (1974) found 18.0 % of angular cheilitis among 339 dental school patients with median age 54 years (range 18—83) and edentulous in at least one jaw.

Prevalence figures in populations not restricted to denture wearers have been reported by Ritchie (1973), 10 % among 300 geriatric patients, and by Manderson and Ettinger (1975), 5 % among 442 institutionalized individuals aged above 60 years and including 369 denture wearers. Grabowski (1974) investigated a randomized sample from a general population in Denmark, $\geq$ 65 years of age, and found a prevalence of 19.2 % among 557 individuals, 467 of whom were denture wearers. The present total prevalence of around 4 %, comprising a 10 % figure in the age group 65—74 years, has not so far been related to denture wearing.

The prevalence was higher among females than males and this is in accordance with previous findings (Nyquist 1962, Mäkilä 1969).

Median rhomboid glossitis

T 0.05 % M 0.06 %; F 0.04 %

Atrophy of tongue papillae, median type

T 1.39 % M 1.75 %; F 1.02 %

The lesion of median rhomboid glossitis is most often thought of as a developmental anomaly, i.e. the persistence of the tuberculum impar in a surface position of the tongue (Martin and Howe 1938). This nature of the lesion has been questionned by Baughman (1971), and the etiology has instead been connected with the presence of Candida albicans (Cooke 1975). Most of these lesions in the present study have been registered as atrophy of tongue papillae, median type. The diagnosis of median rhomboid glossits has, however, been applied in 11 cases, where the lesions were clearly demarcated from the surrounding mucosa by a sharp furrow (Martin and Howe 1938).

Some previous studies of median rhomboid glossitis have recorded prevalences between 0.01 % and 0.42 % (McCarthy 1941, Halperin et al. 1953, Witkop and Barros 1963, Richardson 1968, Redman 1970, Schaumann et al. 1970, Sedano 1975). These figures should be compared with the present total prevalence of median rhomboid glossitis and atrophy of tongue papillae, median type, which was 1.44 %.

Higher prevalences have been reported by Luigi (1968), 3.35 % among 3,274 individuals aged 7—75 years, and by Budtz-Jørgensen (1972), 7.3 % among 303 denture wearers of mean age 60.6 years. The lesion illustrated by Luigi is closely compatible with the present criteria for atrophy of tongue papillae, median type. Budtz-Jørgensen (1972) reported that 21 of his 22 lesions were found in patients with a "generalized simple" or "granular" type of denture stomatitis. As these types often showed the presence of Candida albicans (Budtz-Jørgensen 1970), it is probable that his diagnosis of median rhomboid glossitis corresponds to the present category of atrophy of tongue papil-

lae, median type. A similar lesion has been reported by Mehta et al. (1971) under the name "localized papillary atrophy of the tongue". They found the lesion in *0.28 %* among 50,915 Indian villagers over the age of 15 years and stated that it "is caused by the habit of cleaning the surface of the tongue with fingers, steel blades, or sticks".

Atrophy of tongue papillae, unspecified

T 2.50 % M 2.52 %; F 2.48 %

Atrophy of tongue papillae, with or without inflammatory changes, occurs both together with other illnesses and as a feature in other specific lesions such as e.g. median rhomboid glossitis, geographic tongue and lichen planus. It is thus evident that this atrophy invites diagnostic difficulties and in some cases a clear-cut assignment is impossible. Although these cases have been grouped here under an unspecified heading, subtyping has nevertheless seemed motivated.

The *median type* has been discussed above together with median rhomboid glossitis. As observed by Cooke (1975) this type is seen immediately in front of the terminal sulcus, usually in a central but sometimes in a somewhat lateral position. It may occasionally present a white surface.

The *lichenoid type* presents two patterns. One of these shows bilateral or sometimes unilateral areas of atrophy in the dorso-lateral part of the tongue. The other pattern is characterized by a diffuse atrophy, often showing a subtle whitish dry surface and seen somewhat excentric on the dorsum of the tongue. Similar patterns of atrophy of the tongue papillae have been described in connection with lichen planus by Kuffer (1973), who designated the first pattern as "forme depapillante marginale symmétrique". Cases presenting this lichenoid pattern only, have not been included under the diagnosis of lichen planus in this study.

A *generalized type*, involving a total atrophy of almost all of the papillae, is often seen among old individuals. Depapillation imparts such a red color to the tongue that it may simulate inflammation. It is only when a deep red color or subjective symptoms have been present that glossitis, unspecified (*vide infra*) has been registered.

Cases which did not comply with the above types were labelled *miscellaneous type*.

Glossitis, unspecified

T 0.33 % M 0.11 %; F 0.55 %

Tongue lesions of inflammatory nature have been grouped under several different headings (see e.g. median rhomboid glossitis, atrophy of tongue papillae, unspecified, and geographic tongue). Lesions, not compatible with the criteria set forth for these headings have been registered as glossitis, unspecified, when there was an obvious inflammatory reddening and/or atrophy of tongue papillae with symptoms like itching or burning. Local irritation as well as general disease can give rise to glossitis (Hine 1956, Burket 1971). In the present study there has so far been no possibilities to analyse these circumstances.

Prevalences for glossitis have previously been reported by e.g. McCarthy (1941) who found *5.8 %* among 927 referred patients at a dermatology clinic and Budtz-Jørgensen (1972) who found *10.9 %* among 303 denture wearers of mean age 60.6 years at a dental school. The present figure is considerably lower, and this is

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certainly due to differing diagnostic criteria. Differences in demographic data are also probably of importance, as the prevalence for glossitis seems to rise markedly with age. The highest prevalence in the present study, 2.03 %, was thus found in the age group ≥ 75 years. Denture wearing may also partly explain this rising prevalence. A high incidence of glossitis has been reported to occur together with denture stomatitis and angular cheilitis (Storgård-Jensen and Holst 1969, Budtz-Jørgensen 1972).

Geographic tongue

T 8.45 % M 8.61 %; F 8.24 %

Geographic stomatitis

T 0.05 % M 0.06 %; F 0.03 %

Geographic tongue shows a varying clinical picture. Two basic features seem to be present; on the one hand depapillated areas of the tongue with peripheral reddening, and on the other, a yellowish, serpiginous structure which often partly surrounds the atrophic area. In most cases both features occur simultaneously, but each feature may alternatively be the dominating one. They can, however, be seen separately. The present diagnosis of geographic tongue encompasses all these aspects.

Hume (1975) reported on geographic stomatitis and divided this entity into four different types on the basis of both appearance and localization. When, in the present study, lesions were seen both on the tongue and in other localizations, they were always registered in parallel i.e. as both geographic tongue and as geographic stomatitis. Lesions localized only outside the tongue have been reported (Rood 1974), but no such case was found in this study.

The prevalence for geographic tongue is clearly higher than figures reported in most previous studies. Prevalences between 0.29 % and 3.6 % have thus been reported by Eisenlaub (1941), McCarthy (1941), Halperin et al. (1953), Kocsard et al. (1957/58), Ship et al. (1960), Meskin et al. (1963), Witkop and Barros (1963), Redman et al. (1966), Bhaskar (1968), Luigi (1968), Richardson (1968), Redman (1970), Schaumann et al (1970), Hakemer et al. (1971), Chosack et al. (1974), van Wyk et al. (1974) and Sedano (1975).

Prevalences of the same order as found in this study have been reported by Redman et al. (1966), who found 7.17 % among 530 patients over the age of 17 years at a psychiatry clinic in Minnesota, the United States. As they found prevalences of only 1.15 % and 0.29 % among students and mentally deficient individuals, respectively, they suggested that the lesion geographic tongue might be of a psychosomatic nature.

Togo (1961) found 8.33 % among 18,503 Japanese children and Rahamimoff and Muhsam (1957) 15.0 % among 8,305 Israelian children. The latter study was an incidence study in which 53.8 % of the lesions were encountered at the first examination, corresponding to a prevelence of around *8* %. This high prevalence has been interpreted as reflecting an ethnological predisposition (Hume 1975). However, Chosack et al. (1974) reported a prevalence of only 1.14 % among 70,359 Israelian schoolchildren.

In the present study no sex difference was found, and this is in accordance with most previous reports (Togo 1961, Meskin et al. 1963, Witkop and Barros 1963, Red-

man 1970). However, Rahamimoff and Muhsam (1957) and Chosack et al. (1974) found a slight predominance among males and Halperin et al. (1953) a higher prevalence among females.

Plicated tongue

T 6.48 % M 5.86 %; F 7.03 %

Fissuring of the dorsum of the tongue can appear in different forms and patterns. Fitzwilliams (1927) offered a division into a foliaceous, a cerebriform and a transverse form. The criteria for these forms are difficult to apply, however, as there are many intermediate varieties. Criteria similar to those used by Redman (1970) were thus applied in this study. In contrast to the studies by Witkop and Barros (1963) and Fischman (1974), no distinction was made between scrotal and fissured tongue.

The present total prevalence is in rather good agreement with several previous investigations in populations of broad age composition. Thus, 5.14—8.28 % was reported by Halperin et al. (1953), Luigi (1968), Schaumann et al. (1970), Fischman (1974), and van Wyk et al. (1974).

Lower prevalences have previously been reported by McCarthy (1941), *1.5 %*, among referred patients at a dermatology clinic in the United States, Chrigström et al. (1970), *2.0 %* among denture wearers aged 60—96 years in Sweden and by Aboyans and Ghaemmaghami (1973) 2.56 % among Persians of all ages.

A higher prevalence of 12.07 % was reported by Witkop and Barros (1963) among 1,906 Chileans of all ages. This figure may perhaps be explained by their inclusion of "minor forms", i.e. scrotal tongue. High prevalence figures were also reported by Mäkilä (1974) among 260 patients at a Finnish dental school. In the age groups 20—64 and 65—83 years fissuring of the tongue was found in 9 and 19 %, respectively.

Previous findings have shown an increasing prevalence with age (for review see Hume 1975). This was also found in the present study as there was 1.65 % in the age group 15—24 years and 3.35, 6.15, 8.41, 11.18, 11.22 and 11.49 %, respectively, in the successive 10-year strata.

Highly differing occurrences of plicated tongue have been reported among children. The prevalences given by Rolleri (1939), Redman (1970) and Chosack et al. (1974), 1.08—3.28 %, are largely in agreement with the present findings in the age group 15—24 years. Higher prevalences were reported by Hanhart (1934) and Seiler (1936), 6.7 % and 9.9 %, respectively. Remarkably high prevalences were found among mentally retarded children by Rolleri (1939) with 12.12 % and by Hanhart (1934) with 46.8 %.

Plicated tongue was somewhat more frequent among females than among males in this study. This is in contrast to the results of Halperin et al. (1953) and van Wyk et al. (1974), who did not find any sex difference and to those of Aboyans and Ghaemmaghami (1973) and Chosack et al. (1974), who found it more common in males.

Racial differences as regards the occurrence of plicated tongue have been reported by Turpin and Caratzali (1933), who found a significantly higher prevalence among Polish Israelians than among several other ethnic groups. However, the above mentioned study by Chosack et al. (1974) was performed among Israelian schoolchildren and showed prevalences which were not higher than those found among other, non-Israelian children.

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Coated tongue

T 1.88 % M 2.39 %; F 1.40 %

Hairy tongue

T 0.58 % M 0.83 %; F 0.32 %

The present diagnosis of coated tongue includes lesions showing pigmentations of exogenous origin. The slight discoloration and/or coating often seen among smokers, as well as the causal type of discoloration associated with the consumption of certain types of food or candy, have not been registered.

Prevalence figures for coated tongue have previously only been recorded among special populations. Celis and Little (1966) found around 5 % among 700 hospitalized patients. They stressed that the lesion should probably be less frequent in a general population. Chrigström et al. (1970) found *5.5* % among 201 denture wearers aged 60—96 years. This is similar to the present maximal prevalence figure of around 5 %, recorded in the age group ≥ 75 years. Mäkilä (1974) reported 4 % among 52 dental school patients in the age group ≥ 65 years.

The present prevalence for hairy tongue is largely in agreement with previous figures, 0.2—1.16 %, reported by Eisenlaub (1941), McCarthy (1941), Luigi (1968), Redman (1970) and Schaumann et al. (1970). Celis and Little (1966) reported 5 %, but questioned whether their figure was representative. A considerably higher prevalence, 13 %, was reported by Kocsard et al. (1957/58) among 232 individuals, 73 % of whom were above 50 years of age, at a hospital and a home for the aged in Lidcombe, Australia. Whether coated tongue was included in this latter figure was not stated. The authors pointed to senility, alcohol and tobacco as possible etiologic factors. That smoking may play a role in this respect is also suggested from the report by Celis and Little (1966), and this may partly explain the sex difference found in the present study.

Discoid lupus erythematosus

T 0.01 % M 0.01 %; F 0.01 %

Two persons were diagnosed as suffering from discoid lupus erythematosus. One of them was a woman, 50 years of age, with bilateral, intraoral lesions in the dorsal part of the cheek and the retromolar area. There were no skin manifestations, but the lesions were clinically typical and microscopically compatible with the diagnosis. The other person was a man, 51 years of age, with lesions on the vermilion border of the lower lip. He stated that skin eruptions over the nose and malar eminencies appeared when he was exposed to sunlight. The two localizations, cheek and lip, have been put forth as the most common sites for oral discoid lupus erythematosus (Monash 1931a, Sugár 1954, Andreasen 1964).

No prevalence figures for oral discoid lupus erythematosus in general populations are available as yet. Schiødt et al. (1975) have estimated the number of patients with discoid lupus erythematosus in Copenhagen to be around 250. As apparently every fifth patient should have oral manifestations (Andreasen 1964), an estimate of the prevalence in Copenhagen would be in good agreement with the present finding. In a special population of 927 referred patients at a dermatology clinic in Boston, the United States, McCarthy (1941) found a prevalence of *1.0* % of oral lesions.

Lichen planus

T 1.89 % M 1.59 %; F 2.19 %

Lichen planus appears in many different forms in the oral cavity. The many varieties described by Kuffer (1971) have in this study been grouped in six main types, largely in accordance with Andreasen (1968). The reticular type thus includes linear and annular forms, the atrophic type erythematous forms, and the plaque type hyperkeratotic and verrucous forms. Pigmented lichen planus lesions, described by Kuffer (1971), have been registered both as lichen planus and excessive melanin pigmentation NOS.

Often several types of lichen planus occur simultanously in the same individual, and these types have been registered separately. Each type should show an extension of at least 5 mm. Exceptions from the last requirement have been erosions and vesicles or bullae, which have been registered regardless of size.

The total prevalence for lichen planus was 1.89 %. If the diagnosis athrophy of tongue papillae, lichenoid type, is included, the prevalence becomes 2.20 %. Both these figures are higher than those in previous reports, most of which stem from India. Mehta et al. (1971) reported *0.3* % among 50,915 villagers over the age of 15 years, Pindborg et al. (1965a, 1965b, 1966) 0.02—0.22 % in three populations, each consisting of 10,000 patients at dental schools, Zachariah et al. (1966) 0.40 % among 5,000 patients at a dental school, and Smith et al. (1975) 0.6 % among 57,518 textile workers over the age of 35 years. A higher prevalence, approaching the present one, was reported by Pindborg et al. (1972a), who found 1.5 % in a door-to-door investigation among 7,639 villagers in Kerala.

Low prevalences have also been reported from Switzerland, where Rufenacht (1968) found *0.14* % among 32,879 patients of all ages at a dermatology clinic and from Argentina, where Borghelli et al. (1974) found *0.28* % among 2,174 males, 20 years of age.

Somewhat higher prevalences have been reported from the United States by Bhaskar (1968), who found 1.1 % among 785 individuals, aged 45—97 years at US Old Soldier's Home in Washington, D.C., and by McCarthy (1941), who found *1.7* % among 927 referred patients at a dermatology clinic in Boston.

The relative frequency of the different types of lichen planus in this study is somewhat different from that reported by Andreasen (1968). The plaque type was thus relatively more common and this may depend on differences in criteria. Erosive lichen planus was less common and this may be explained by the fact, that Andreasen investigated referred patients. Erosive lesions are thus often accompanied by subjective symptoms.

There was a significantly higher prevalence of lichen planus among females. This is in accordance with most reports on referred patients (for review see Andreasen 1968). Pindborg et al. (1972a), however, did not find any sex difference.

Fordyce's condition

T 82.80 % M 85.93 %; F 79.55 %

Sebaceous glands have been registered both intraorally and on the prolabium. The total prevalence is higher than in most previous reports (for review see Sewerin 1975) but in relative agreement with Halperin et al. (1953) who found *85.33* % in corresponding age groups.

Thorough studies by Miles (1958) and Sewerin (1975) showed prevalences of around *72 %* and *87 %*, respectively, intraorally and *69 %* and *76 %*, respectively, on the prolabium among individuals corresponding in age to those of the present study.

A higher prevalence was found among males, and this is in accordance with previous studies (for review see Sewerin 1975). The opposite sex distribution has occasionally been reported (see for instance White 1905). Miles (1958) and Sewerin (1975) found no sex difference. These discrepancies may be due to registration difficulties. Sewerin (1975) thus found that sebaceous glands were more easily recognized in males.

Mani et al. (1976) suggested that the use of tobacco could influence the registration of sebaceous glands. These authors, in a population of 43,654 industrial workers over the age of 35 years in Gujarat in India, found a lower prevalence among individuals using tobacco. The reason for this should be that smoking and chewing "makes the normal epithelium become increasingly tense and opaque, thereby making the sebaceous glands invisible clinically through an otherwise translucent epithelium". This hypothesis seems to be contradicted by the present findings, as sebaceous glands as well as tobacco habits were more prevalent among males.

White sponge naevus

T 0.02 % M 0.05 %; F —

"Developmental keratotic lesions" or "oral epithelial naevi" are recognized in two forms, which can be differentiated clinically as well as microscopically (Cooke 1967). In the present study, these lesions have been collectively labelled white sponge naevus.

One lesion was found. An 87 year old man thus presented with a symmetrical, white, thick and somewhat elevated alteration on the margins and the ventral surface of the tongue. The lesion had been present as long as he could remember. The diagnosis was microscopically verified. A son of the patient was also examined, but no similar lesion was found. These findings would group the present case as the type of white sponge naevus which is congenital but not necessarily hereditary in nature (Cooke 1956).

There is very little information as to the prevalence of white sponge naevus. Borghelli et al. (1974) in an investigation in Argentina of 2,174 males, aged 20 years, found 202 individuals with white lesions. Of the latter, 1 % were diagnosed as white sponge naevus, which corresponds to a total prevalence of *0.1 %* in the population investigated.

Gingival hyperplasia, adverse effect of hydantoin derivatives

T 0.07 % M 0.07 %; F 0.08 %

Gingival hyperplasia is a well known complication to diphenylhydantoin therapy. The lesion has often been subdivided in different clinical degrees (see for instance Angelopoulos and Goaz 1972), but this was not done in the present study. Difficulties exist to differentiate minor degrees of hyperplasia from gingivitis. Aas (1963) reported, that inflammatory symptoms were far less evident among minor hydantoin hyperplasias, and it was thus considered mandatory that the present diagnostic criteria for these

cases included a normal color and surface structure of the gingiva.

Previous studies have recorded the incidence of hyperplasia among patients on hydantoin medication (for review see Angelopoulos and Goaz 1972). In recent studies Angelopoulos and Goaz (1972) and Klar (1973) reported incidences of 53.2 % and 63.8 %, respectively. No sex differences were revealed and this in accordance with the present findings.

Edentulous individuals taking hydantoin derivatives occasionally develop fibrous hyperplasia. Thus, a 60 year old man in the present study showed multiple nodular hyperplasias over the entire palate under a full upper denture. There were no signs of denture stomatitis. This case appeared very similar to one illustrated by McCarthy and Shklar (1964).

Amalgam tattoo

T 8.22 % M 5.41 %; F 11.02 %

Dark, localized discolorations of the oral mucosa may reflect, for instance, amalgam tattoos, naevi, melanosis or haemangiomas (Carlson et al. 1971). However, amalgam tattoo, which is caused by traumatic implantation of amalgam fragments, does not usually invite diagnostic difficulties (Weathers and Fine 1974). The presence of a malignant melanoma should anyhow, because of its fatal nature, always be kept in mind. In the present study biopsies were taken from two doubtful cases of amalgam tattoo. For both, the preliminary diagnosis was verified.

The present prevalence is largely in agreement with previous reports (Bhaskar 1968, Carlson et al. 1971, Weathers and Fine 1974).

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GENERAL DISCUSSION

In studies, similar to the present one, where an effort is made to survey the general health status of the oral mucosa, some lesions of short duration will of necessity be incorporated. As noted in the introduction it may be suspected that the occurrence of such lesions in the population would have been better understood from an incidence study. A relationship usually prevails, however, between incidence and prevalence. If the mean duration of a lesion is known, and if incidence and duration are relatively stable, prevalence information can give a good idea as to the incidence (MacMahon and Pugh 1970). For most lesions of short duration included in the present study, the mean duration is relatively well known. Also, for most such lesions, incidence and duration can be considered as relatively stable.

Concerning some lesions, seasonal variations have been reported. This is for instance true for acute necrotizing ulcerative gingivitis, which is reported to occur more frequently during autumn and winter (Pindborg 1951, Skach et al. 1970). The possibility to estimate the real prevalence of such lesions is to a large extent dependant upon how long time is spent for the investigation. If it is carried out during a long time period as, for instance, a whole year, lesions showing seasonal variations can be expected to be well represented.

The population selected for the present study was summoned according to routines developed for a public health screening organization in Uppsala County, Sweden. The frequency of participation in the primary investigation did not differ from that previously reported in connection with this health screening (Hillerdal and Irnell 1969). The addition of an oral examination thus did not appear to have influenced the attitude to participation.

In comparison with the total population, the population attending the primary investigation was skewed as regards demographic factors. There was a marked underrepresentation of the young and the old, as well as of males in comparison to females. The frequency of participation was clearly lower in regions I and II (densely populated) than in region III (thinly populated). This skewness from the primary investigation was greatly reduced when the secondary investigation had been performed. There was, however, still a small underrepresentation within the youngest and oldest age groups. The reason for this persisting inequity probably was that individuals, who had changed residence or had recently died, were most frequent in these age groups.

The undertaking of the secondary investigation resulted in an improved final frequency of participation, which was in parity with previously performed mass investigations including oral aspects (Cochrane et al. 1952, Wash. Publ. Hlth. Serv.

1965). The standard errors connected to the prevalences were thereby diminished.

The carrying out of the secondary investigation, i.e. a deliberate effort to examine primary non-participants, was advantageous in several respects. When performing prevalence studies, the non-participants should thus preferably be investigated. However, the effort associated with such a retrieval should be considered.

The use of well-defined criteria and/or diagnoses should be a prerequisite for carrying out prevalence investigations. If comparisons with other studies are aimed at, the criteria should advantageously specify commonly used diagnostic labels. Criteria and/or diagnoses should also be adequately reported. The significance of all this can be illustrated by a comparison between the prevalence of leukokeratosis nicotina palati found in the present study, 1.12 %, and that found by Saietz (1975), 6.0 %. Saietz categorized this lesion in two groups and reported criteria for both. The criteria for one of the groups agree well with those of the present study, and for this group Saietz found a prevalence of 1.4 %. The marked difference in total prevalences can thus be better understood.

The importance of accounting for criteria can further be illustrated in relation to the diagnosis of leukoplakia, which as applied here gave a prevalence for this lesion of 3.53 % in the present population. If the lesion preleukoplakia is included under the diagnosis of leukoplakia, the prevalence becomes 8.32 %. If frictional keratosis and snuff dipper's lesion (often termed snuff leukoplakia) also are included, the prevalence becomes 15.53 %.

Several attempts were made in the present study to estimate the validity and reliability of the registrations. Tests were carried through for the majority of the lesions, and it can be concluded that the registrations were in general satisfactory. It must be kept in mind, however, that each type of lesion presents unique characteristics, and that any generalized statement as to validity and reliability concerning all diagnoses taken together is somewhat inappropriate.

As the investigation proceeded, a tendency towards a systematic increase in the examiner's sensitivity in registration seemed to have occurred for some of the lesions. This was so for excessive melanin pigmentation NOS, leukoedema, frictional keratosis, traumatic ulcer, fibroepithelial polyp, denture-related mucosal petechiae and Fordyce's condition. A certain underregistration of these lesions could thus have occurred in the earlier part of the investigation. It should again be emphasized, however, that this conclusion is derived from comparisons between groups of individuals wihch were matched only as to age and sex. An accurate analysis of such a gradual change requires detailed calculations involving a number of other factors such as denture status, tobacco habits, occupational and environmental conditions and socio-economic status.

In the present study prevalences are reported for about 60 oral mucosal lesions. The prevalence information is primarily valid only for the area investigated, i.e. the municipalities of Enköping and Håbo. Although no detailed comparison has been made between the population investigated and that of Sweden at large, it seems probable, that the findings adequately

describe conditions existing in most of the country. This presumption is based on the fact that, among else, the distribution of the population investigated as regards age and sex as well as the percentage of denture wearers and smokers is close to that of the whole population of Sweden (Table 12, Appendix).

To allow a comparison of the results of this study with those of other prevalence studies on finite populations, the standard errors given for the prevalence figures have been calculated as if the population investigated was a sample from an infinite population. If standard errors would have been given for the finite population, they would have been around 18 % lower. That this change is of such a low magnitude is mainly due to the fact, that a high proportion of the total population was examined.

The prevalences found in the study agree, for the most part, with findings in previous reports. However, some noteworthy exceptions were observed.

Focal epithelial hyperplasia was found in a definitely lower prevalence than what has previously been found among American Indians and Eskimos. Among Caucasians, only separate cases have so far been reported, and the impression has been gained that among these, focal epithelial hyperplasia is a rare lesion. Against this background, the present prevalence of 0.11 % seems surprisingly high. In fact, if one does not label fibroepithelial polyps as fibromas, focal epithelial hyperplasia was the most common tumor of the oral mucosa in the population investigated. That this lesion previously has been noted in Sweden only occasionally, may depend on the lesion being relatively unknown and on the fact that it often has a clinical appearance easily confused with other tumors or tumorlike conditions such as papillomas and fibroepithelial polyps.

Leukoedema, with a prevalence of 49.07 %, seems to be a relatively common lesion in the population investigated. It can be suspected that this partly relates to the use of tobacco. However, very low prevalences of leukoedema have been found in other countries where tobacco habits are widespread as, for instance, in India. It should then be born in mind that differences in tissue reaction patterns among different populations may exist and influence the development of, for instance, leukoedema.

Geographic tongue has previously been reported to be considerably less prevalent than plicated tongue. In the present study the prevalences for these lesions are notably similar, 8.45 % and 6.48 %, respectively. This finding is especially interesting in view of the fact, that both lesions often have been suggested to be genetically determined and, as regards prevalence figures, to covariate (Hume 1975). The relatively high prevalence of geographic tongue found in this study may be explained in relation to the use of criteria which include several different clinical manifestations. Thus, the diagnosis of geographic tongue was also applied when only characteristic papillary atrophy or white serpiginous structures were present, as well as when no migration was documented.

Lichen planus. The prevalence of 1.89 %, and if the lichenoid type of atrophy of tongue papillae is included, 2.20 %, seems to be the highest one so far reported. It is also a notably higher prevalence than what has been recorded for lichen ruber planus on the skin. In five regions

in Sweden, Hellgren (1970) found skin lesions in 0.13—0.39 % among 39,418 individuals. At the present, one can only speculate as to the cause for the high prevalence of oral lichen planus. The lesion is often regarded to be of a psychosomatic nature (Shafer et al. 1974), and it may be, that lichen planus is more frequent in highly industrialized countries, where stress situations probably are relatively more common. Some authors have pointed to a possible connection between oral lichen planus and different types of trauma as, for instance, galvanism and use of tobacco (Frykholm et al. 1969, Pindborg et al. 1972a) and such factors may perhaps influence the occurrence.

Comparisons of the present prevalence figures with previously reported data are difficult to undertake for a number of lesions without a detailed knowledge as to the presence of various intraoral, etiologic factors. Also, any specific, etiologic factor may influence the prevalence of several separately registered lesions. This applies, for instance, to lesions connected with denture wearing and tobacco habits.

Lesions related to denture status are primarily denture hyperplasia, flabby ridge, denture stomatitis and denture-related mucosal petechiae. A number of other lesions can also be regarded as more or less related to denture wearing. These include traumatic ulcer and frictional keratosis. Further, lesions that are infected by Candida albicans are thought, to a certain extent, to be associated with denture status. To this category belong acute pseudomembranous candidosis and lesions included in chronic multifocal oral candidosis, e.g. chronic candidosis, angular cheilitis, atrophic and nodular leukoplakia, median type of atrophy of tongue papillae and glossitis, unspecified.

Lesions related to tobacco habits are primarily leukokeratosis nicotina palati and snuff dipper's lesion. The prevalence of a number of other lesions, however, are thought also to be influenced by tobacco habits. Seen in the light of the findings in previous studies, these lesions may be acute necrotizing ulcerative gingivitis, recurrent aphthae, preleukoplakia, leukoplakia, leukoedema, coated tongue, hairy tongue and lichen planus.

The information collected in this study will be used for further analyses of these lesions related to denture status and tobacco habits. It will also serve as a basis for follow-up investigations. Special interest will then be focused on leukoplakia, lichen planus and snuff dipper's lesion, since these lesions have previously been reported to be of a precancerous nature.

GENERAL SUMMARY

The prevalence of oral mucosal lesions in Sweden is virtually unkown. Available prevalence figures concern but a few types of lesions and relate to highly selected, special populations. Investigations on general populations are sparse also in other countries.

The aim of the present study was to survey the occurrence of oral mucosal lesions in a relatively large general population in Sweden.

The study was performed in collaboration with a health screening organization in the middle of Sweden. The populations of two municipalities in the County of Uppsala were investigated. A mobile health screening group visited several places in the selected area and during the time of the present investigation a total of 30,118 persons, aged 15 years or above, were summoned. Of these, 62 % or 18.659 individuals attended and were examined.

The non-participants became the subject of a special investigation for which every fifth individual, or 2.292 persons, was randomly sampled. After a second summons, 933 persons presented for examination. Through personal contact, an additional 741 individuals were examined in private homes, places of work, hospitals, old age homes and other institutions. The final non-participation, representing 10.3 % of the total population, was considered to have but a marginal influence on the results of the study.

The diagnostic procedure was based on clinical criteria set forth especially for the investigation. The validity of the final diagnoses was evaluated by, for instance, inter-examiner tests and comparisons between clinical diagnoses and histologic descriptions and was found to be acceptable. Tests of the reliability were, among else, undertaken through re-examinations and indicated, that underregistration apparently occurred for a few of the lesions investigated.

The prevalences of about 60 oral mucosal lesions were recorded and compared with previous findings. Notably high prevalences were found for focal epithelial hyperplasia (0.11 %), leukoedema (49.07 %), geographic tongue (8.45 %) and lichen planus (1.85 %).

For some lesions prevalence figures are difficult to compare with findings from previous studies. This includes lesions which are directly or indirectly related to local etiologic factors such as denture status and tobacco habits. As regards these factors basic information has been collected and will be used for further analyses. The collected total material has also been designed to form the basis for longitudinal studies of, for example, precancerous lesions.

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This monograph is signed Tony Axéll. The efforts and sacrifices behind it belong equally to Elisabeth, Anna, Karin and Göran.

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APPENDIX

(Figures 5—11 and
Tables 11—12)

ORAL MUCOSAL LESIONS
T. AXÉLL
Dpt. Oral Surgery
Faculty of Odontology
University of Lund

Consecutive pat.no. _____

F 1

Complete name (preferred name underlined) _____

Residence address _____ zip code _____ city or town _____

Residence telephone (also area code) Work telephone (also area code) _____

Occupation _____

Swedish citizen ☐ Foreign citizen ☐

Nationality

Unmarried ☐ Married ☐ Divorced ☐ Widow/Widower ☐

Marital status

EVERYTHING BELOW FILLED IN BY INVESTIGATION PERSONNEL

Investigator ☐ Investigation ☐☐ Region of investigation ☐

Local parish ☐☐ Date of examination ☐☐☐

Yes-answer no. _____

Current illness(-es) _____

Sex M ☐ F ☐

Population density Dense ☐ Thin ☐ Class

Reason for non-participation _____ Date of examination _____

Time for recall: _____ Date of examination: _____

Time for recall: _____ Date of examination: _____

Referral to Oral Surgery: _____ Referral to Dermatology: _____

Referral to C-lab for Wassermann (date) _____ neg _____ pos _____

Referral to _____ Date _____

Notations: _____

F1	F2	F3	F4	F5	F6

Fig. 5. *Form 1.*

Fig. 6. *Form*

F 1

ORAL MUCOSAL LESIONS
T. AXELL
Dpt. Oral Surgery
Faculty of Odontology
University of Lund

F 2

Consecutive pat. no.

PLEASE FILL IN THIS CARD AND BRING IT WITH YOU TO THE EXAMINATION LOCALE

Complete name (preferred name underlined)

Do you take or have you, within the last week, taken any medicine or pills prescribed by a physician or dentist?

Yes ☐ No ☐

If you answer Yes, please write below the names of the different medicines or pills.

a. _____ Dose[x] _____
b. _____ Dose _____
c. _____ Dose _____
d. _____ Dose _____
e. _____ Dose _____
f. _____ Dose _____
g. _____ Dose _____

x) Dose = number of pills or amount of liquid per day or week. This is noted on the container.

F4	F5	F6

Fig. 6. *Form 2.*

ORAL MUCOSAL LESIONS
T. AXÉLL
Dpt. Oral Surgery
Faculty of Odontology
University of Lund

Consecutive pat. no. _____

F 3

The purpose of this questionnaire is to survey the consumption of tobacco and alcohol. This is part of a research project that will, in part, attempt to estimate the importance of these habits to various oral diseases. The information is treated confidentially and only used for this particular research project. After completion of the project the questionnaire will be destroyed. Your anonymity is completely protected.

NOTE! Fill in the questionnaire yourself. If you have problems ask the investigation personnel for help.

Name _____

TOBACCO HABITS

Place [X] in the square that best suits your situation!

1. Do you use tobacco (smoking, snuff dipping, chewing) ?

Never ☐ Occasionally ☐ Daily ☐

If your answer to question 1 is Daily, please answer questions 2 - 13. Otherwise proceed to question 14.

2. Did you first begin to use tobacco during the last 3 months?

Yes ☐ No ☐

3. Approximately how much tobacco do you use? (Fill in all lines that apply).

a. Cigarettes without filter: _____ no/day
b. Cigarettes with filter: _____ no/day
c. Cigarcigarettes (cigarillos): _____ no/day
d. Cigars: _____ no/day

e. Pipe: One can (box) lasts approx. _____ days
One large package (50 g) lasts approx. _____ days
One small package (25 g) lasts approx. _____ days

f. Snuff: In the mouth approx. _____ hrs/day
One package (250 g) lasts approx. _____ days
One box (50 g) lasts approx. _____ days

g. Chewing tobacco: In the mouth approx. _____ hrs/day

If you are unsure as to the size of the package it may be of help to examine the illustration placed on the wall and showing the sizes of the most common packages.

4. How long have you

a. Smoked _____ years
b. Used snuff _____ years
c. Chewed tobacco _____ years

OVER!

Fig. 7. *Form 3.*

5. If you smoke *cigarettes*, what brand do you commonly use?

_____

6. Do you use a cigarette holder?

Yes ☐ No ☐

7. If you smoke *cigarillos*, what brand do you commonly use?

_____

8. If you smoke *cigars*, what brand do you commonly use?

_____

9. If you smoke pipe, what brand of *tobacco* to you commonly use?

_____

10. If you use *snuff*, what brand do you commonly use?

_____

11. If you are *chewing tobacco*, what brand do you commonly use?

_____

12. If you use *snuff*, do you put the snuff in

Same place in the mouth ☐ Different places in the mouth from time to time ☐

13. If you *smoke*, do you inhale?

Never ☐ Seldom ☐ Often ☐ Always ☐

To be answered by those who answer question 1 that you Never or Occasionally use tobacco:

14. Have you previously used tobacco daily?

Yes ☐ No ☐

If Yes, please answer the following

15. I smoked in _____ years
I used snuff in _____ years
I chewed tobacco in _____ years

16. If you have *completely* stopped using tobacco, when?

a. _____ years ago

b. _____ months ago Answer either a. or b.

ALCOHOL HABITS:

17. How often have you consumed alcoholic beverages during the last 3 months?

	Never	Seldom	Approx. once a week	A few times a week	Daily
Beer	☐	☐	☐	☐	☐
Wine	☐	☐	☐	☐	☐
Hard liquor	☐	☐	☐	☐	☐

ORAL MUCOSAL LESIONS
T. AXÉLL
Dpt. Oral Surgery
Faculty of Odontology
University of Lund

F 4

Consecutive pat. no. ____________

Examination date ____________

Oral hygiene

Denture status

	Max.	Mand.
No denture in totally edentulous jaw	☐	☐
Part. denture	☐	☐
Full denture	☐	☐

Dental work and irritants

- Amalgam ☐
- Clasp ☐
- Gold ☐
- Chrome-cobalt ☐
- Stainless steel ☐
- Sharp prosthetic teeth in relation to a lesion ☐
- Sharp teeth or fillings in relation to a lesion ☐

Date of photo ____________

Preliminary DIAGNOSIS

1. ____________
2. ____________
3. ____________
4. ____________
5. ____________

Fig. 8. *Form 4.*

F 4

ORAL MUCOSAL LESIONS
T. AXÉLL
Dpt. Oral Surgery
Faculty of Odontology
University of Lund

Consecutive pat. no.

F 5

no. | total no.

Biopsy code | Localization code

Lesion number 1. _____
2. _____
3. _____
4. _____
5. _____

Lesion no.	DIAGNOSIS C1/C2	DIAGNOSIS C2/CH2	Localization	Teeth or not	Local. type	Extent			Noted by	months since	years since	Photo
							x	=				

Special information

Lesion number 1. _____
2. _____
3. _____
4. _____
5. _____

Fig. 9. *Form 5.*

ORAL MUCOSAL LESIONS
T. AXÉLL
Dpt. Oral Surgery
Faculty of Odontology
University of Lund

Consecutive pat no.

F 6

Complete name (prefcrred name underlined)

No. (filled in by dpt.)

Address

Civil registration number

Biopsy (local. code) ☐☐ date ___ Lesion nr ☐

Clinical DIAGNOSIS (C 1)

Clinical DIAGNOSIS (C 2)

Code ☐☐☐

Clinical-histologic DIAGNOSIS (CH 2)

Code ☐☐☐

Histologic description

Histologic DIAGNOSIS (H1)

Clinical-histologic DIAGNOSIS (CH 1)	WHO-code

Fig. 10. *Form 6.*

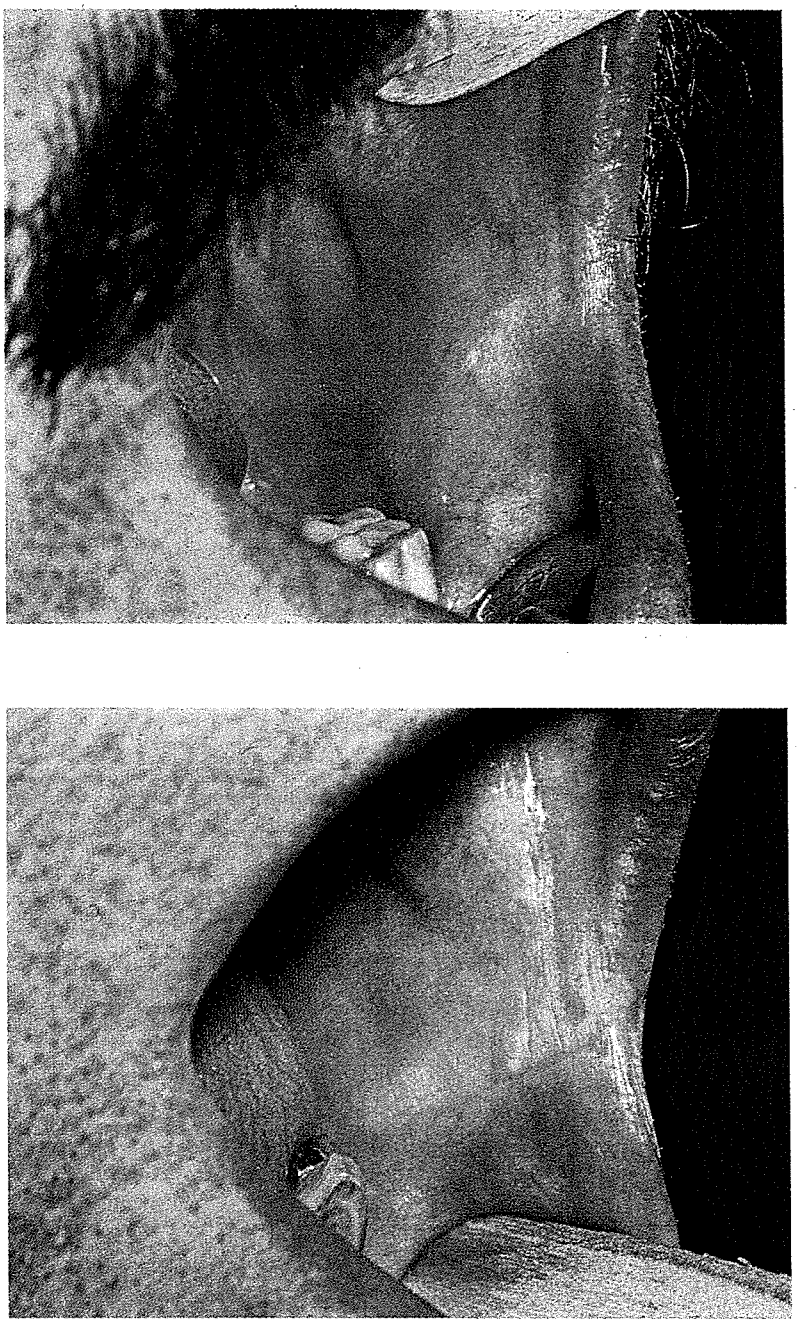

Fig. 11. *Illustrations indicating the minimal degree of "whiteness" demanded for determining a diagnosis of leukoplakia on non-kreatinized mucosa.*

Table 11. *Prevalences of oral mucosal lesions. Number of individuals examined 20,333. x = number of individuals with lesions. p = prevalences in %. SE = standard error of the mean for p. P = probability for sex difference.*

	Total			Males			Females				
	x	p	SE	x_1	p_1	SE_1	x_2	p_2	SE_2	p_1—p_2	P
Herpes zoster	1	*<0.01*	0.003	1	*0.01*	0.007	—	—	—	0.01	n.s.
Herpes labialis	652	*3.07*	0.172	313	*2.92*	0.242	339	3.22	0.245	—0.30	n.s.
History of herpes labialis	2835	*14.32*	0.368	1259	*13.12*	0.515	1576	*15.50*	0.522	—2.38	<0.01
Herpes labialis + history of herpes labialis	3487	*17.38*	0.395	1572	*16.04*	0.554	1915	*18.71*	0.559	—2.67	<0.001
Intraoral herpetiform lesion	54	*0.33*	0.068	16	*0.28*	0.101	38	*0.37*	0.089	—0.09	<0.01
Herpangina	4	*0.03*	0.023	1	*0.01*	0.007	3	*0.05*	0.042	—0.04	n.s.
Acute necrotizing ulcerative gingivitis	3	*0.05*	0.031	1	*0.05*	0.049	2	*0.04*	0.039	0.01	n.s.
Acute pseudomembranous candidosis	23	*0.21*	0.059	7	*0.13*	0.069	16	*0.28*	0.095	—0.15	n.s.
Chronic candidosis	67	*0.36*	0.062	41	*0.41*	0.093	26	*0.31*	0.085	0.10	n.s.
Atrophic	59	*0.33*	0.062	36	*0.38*	0.092	23	*0.29*	0.084	0.09	n.s.
Hyperplastic	11	*0.04*	0.011	7	*0.05*	0.017	4	*0.03*	0.013	0.02	n.s.
Carcinoma	1	*<0.01*	0.003	—	—	—	1	*0.01*	0.007	—0.01	n.s.
Papilloma	24	*0.10*	0.027	12	*0.12*	0.052	12	*0.08*	0.023	0.04	n.s.
Focal epithelial hyperplasia	17	*0.11*	0.038	10	*0.09*	0.040	7	*0.12*	0.061	—0.03	n.s.
Lipoma	7	*0.06*	0.031	4	*0.06*	0.045	3	*0.05*	0.042	0.01	n.s.
Haemangioma	19	*0.08*	0.028	9	*0.06*	0.020	10	*0.11*	0.054	—0.05	n.s.
Lymphangioma	1	*0.02*	0.025	—	—	—	1	*0.05*	0.049	—0.05	n.s.
Gingival fibromatosis and localized enlargement e.g. tuberosity	50	*0.17*	0.023	28	*0.18*	0.035	22	*0.15*	0.031	0.03	n.s.
Excessive melanin pigmentation NOS	1959	*9.94*	0.313	1040	*10.49*	0.458	919	*9.40*	0.428	1.09	n.s.
Gingival cyst	6	*0.03*	0.020	3	*0.02*	0.011	3	*0.05*	0.039	—0.03	n.s.
Chronic desquamative gingivitis	3	*0.01*	0.006	—	—	—	3	*0.02*	*0.012*	—*0.02*	n.s.
Recurrent aphthae	456	*2.00*	0.133	218	*1.89*	0.184	238	*2.08*	0.186	—0.19	n.s.
History of recurrent aphthae	3359	*15.70*	0.359	1536	*14.91*	0.511	1823	*16.48*	0.501	—1.57	<0.05
Recurrent aphthae + history	3815	*17.70*	0.375	1754	*16.81*	0.532	2061	*18.56*	0.525	—1.75	<0.05

Excessive melanin pigmentation NOS	1959	*9.94*	0.313	1040	*10.49*	0.458	919	*9.40*	0.428	1.09	n.s.
Gingival cyst	6	*0.03*	0.020	3	*0.02*	0.011	3	*0.05*	0.039	—0.03	n.s.
Chronic desquamative gingivitis	3	*0.01*	0.006	—	—	—	3	*0.02*	0.012	—0.02	n.s.
Recurrent aphthae	456	*2.00*	0.133	218	*1.89*	0.184	238	*2.08*	0.186	—0.19	n.s.
History of recurrent aphthae	3359	*15.70*	0.359	1536	*14.91*	0.511	1823	*16.48*	0.501	—1.57	<0.05
Recurrent aphthae + history of recurrent aphthae	3815	*17.70*	0.375	1754	*16.81*	0.532	2061	*18.56*	0.525	—1.75	<0.05
Periadenitis mucosa necrotica recurrens	3	*0.01*	0.006	1	*0.01*	0.007	2	*0.01*	0.009	<—0.01	n.s.
Preleukoplakia	1189	*6.35*	0.262	942	*10.01*	0.459	247	*2.69*	0.244	7.32	<0.001
Leukoplakia	705	*3.60*	0.194	602	*6.08*	0.355	103	*1.15*	0.162	4.93	<0.001
Homogenous	661	*3.37*	0.189	563	*5.70*	0.347	98	*1.08*	0.156	4.62	<0.001
Atrophic	48	*0.24*	0.049	42	*0.40*	0.085	6	*0.07*	0.043	0.33	<0.001
Nodular	8	*0.03*	0.009	8	*0.05*	0.019	—	—	—	0.05	<0.01
Leukokeratosis nicotina palati	178	*1.12*	0.124	173	*2.13*	0.239	5	*0.10*	0.059	2.03	<0.001
Snuff dipper's lesion	1466	*8.04*	0.299	1459	*15.94*	0.568	7	*0.19*	0.085	15.75	<0.001
Leukoedema	9463	*48.89*	0.492	5862	*61.92*	0.676	3601	*35.89*	0.661	26.03	<0.001
Frictional keratosis	881	*5.47*	0.265	486	*6.29*	0.410	395	*4.66*	0.334	1.63	<0.01
Cheek and lip biting	933	*5.14*	0.243	449	*4.86*	0.336	484	*5.46*	0.351	—0.60	n.s.
Traumatic ulcer	546	*4.27*	0.257	275	*4.26*	0.363	271	*4.27*	0.355	—0.01	n.s.
Ulcus mucosae oris NOS	218	*1.22*	0.124	153	*1.84*	0.224	65	*0.61*	0.112	1.23	<0.001
Mucocele	22	*0.11*	0.033	15	*0.13*	0.043	7	*0.08*	0.047	0.05	n.s.
Pyogenic granuloma	18	*0.08*	0.027	4	*0.03*	0.013	14	*0.13*	0.050	—0.10	<0.05
Fibroepithelial polyp	485	*3.25*	0.209	235	*3.02*	0.285	250	*3.46*	0.301	—0.44	n.s.
Denture hyperplasia	590	*3.41*	0.199	212	*2.33*	0.236	378	*4.43*	0.310	—2.10	<0.001
Flabby ridge	1654	*8.57*	0.278	553	*5.52*	0.329	1101	*11.54*	0.433	—6.02	<0.001
Denture stomatitis	3113	*16.02*	0.356	1236	*12.27*	0.469	1877	*19.65*	0.518	—7.38	<0.001
Localized	2824	*14.64*	0.346	1121	*11.20*	0.451	1703	*17.98*	0.511	—6.78	<0.001
Generalized	285	*1.35*	0.116	113	*1.03*	0.141	172	*1.65*	0.176	—0.62	<0.001
Papillomatous	377	*2.10*	0.158	191	*2.03*	0.219	186	*2.16*	0.225	—0.13	n.s.
Papilliform hyperplasia of palate	3	*0.03*	0.023	3	*0.06*	0.049	—	—	—	0.06	n.s.
Denture-related mucosal petechiae	32	*0.39*	0.086	13	*0.38*	0.127	19	*0.41*	0.119	—0.03	n.s.
Angular cheilitis	715	*3.76*	0.202	285	*2.95*	0.259	430	*4.59*	0.309	—1.64	<0.001
Median rhomboid glossitis	10	*0.05*	0.021	4	*0.06*	0.037	6	*0.04*	0.016	0.02	n.s.

Table 11 continued.

	Total			Males			Females				
	x	p	SE	x_1	p_1	SE_1	x_2	p_2	SE_2	$p_1—p_2$	P
Atrophy of tongue papillae, unspecified	469	*2.50*	0.169	242	*2.52*	0.240	227	*2.48*	0.232	0.04	n.s.
Median	251	*1.39*	0.128	160	*1.75*	0.206	91	*1.02*	0.152	0.73	<0.001
Lichenoid	50	*0.31*	0.065	22	*0.19*	0.060	28	*0.41*	0.110	—0.22	n.s.
Generalized	47	*0.27*	0.059	9	*0.10*	0.047	38	*0.44*	0.103	—0.34	<0.001
Miscellaneous	118	*0.54*	0.072	49	*0.47*	0.100	69	*0.60*	0.099	—0.13	n.s.
Glossitis, unspecified	53	*0.33*	0.069	10	*0.11*	0.055	43	*0.55*	0.120	—0.44	<0.001
Geographic tongue	1664	*8.45*	0.293	852	*8.61*	0.423	812	*8.24*	0.399	0.37	n.s.
Geographic stomatitis	8	*0.05*	0.026	3	*0.06*	0.050	5	*0.03*	0.015	0.03	n.s.
Plicated tongue	1268	*6.48*	0.255	602	*5.86*	0.343	666	*7.03*	0.368	—1.17	<0.05
Coated tongue	364	*1.88*	0.145	223	*2.39*	0.239	141	*1.40*	0.171	0.99	<0.001
Hairy tongue	78	*0.58*	0.094	62	*0.83*	0.158	16	*0.32*	0.104	0.51	<0.001
Discoid lupus erythematosus	2	*0.01*	0.005	1	*0.01*	0.007	1	*0.01*	0.007	<—0.01	n.s.
Lichen planus	410	*1.89*	0.133	154	*1.59*	0.190	256	*2.19*	0.185	—0.60	<0.001
Papular	39	*0.18*	0.042	19	*0.16*	0.054	20	*0.20*	0.063	—0.04	n.s.
Reticular	317	*1.46*	0.117	112	*1.14*	0.162	205	*1.78*	0.170	—0.64	<0.001
Plaque	177	*0.77*	0.079	67	*0.60*	0.098	110	*0.92*	0.118	—0.32	<0.01
Atrophic	134	*0.56*	0.066	42	*0.40*	0.090	92	*0.73*	0.097	—0.33	<0.001
Erosive	32	*0.16*	0.043	8	*0.14*	0.069	24	*0.20*	0.054	—0.06	<0.01
Bullous	3	*0.01*	0.006	1	*0.01*	0.007	2	*0.01*	0.009	<—0.01	n.s.
Fordyce's condition	16667	*82.80*	0.375	8551	*85.93*	0.502	8126	*79.55*	0.558	6.38	<0.001
White sponge naevus	1	*0.02*	0.023	1	*0.05*	0.051	—	—	—	0.05	n.s.
Gingival hyperplasia, adverse effect of hydantoin derivatives	10	*0.07*	0.035	5	*0.07*	0.048	5	*0.08*	0.052	—0.01	n.s.
Amalgam tattoo	1686	*8.22*	0.279	580	*5.41*	0.319	1106	*11.02*	0.448	—5.61	<0.001

Table 12. *Some charac*

Figures based on in
[a] 15 years or above
[b] 16 years or above
[c] 15—75 years

Information in Tab
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Percentage of habitual smokers
Total
Males
Females

Percentage of denture wearers
Total
Males
Females

No. of physicians per 10,000 inhabitants

No. of dentists per 10,000 inhabitants

Percentage of foreign citiziens

Percentage of people living in densely populated area

Population density
IV 65 years

Percentage of people in age strata
0—14 years
15—64 years

Average age years
Total
Males
Females

Ratio males: females

Table 12. *Some characteristics of the population in the area investigated and in Sweden at large.*

	Area investigated	*Sweden at large*
Ratio males: females	0.97:1[a]	0.98:1[a]
Average age years		
Total	43.0[a]	43.3[a]
Males	42.6[a]	44.1[a]
Females	43.4[a]	42.6[a]
Percentage of people in age strata		
0—14 years	26.1	20.7
15—64 years	61.7	64.6
≥ 65 years	12.2	14.7
Population density	31/km^2	20/km^2
Percentage of people living in densely populated area	62.6	81.4
Percentage of foreign citiziens	6.8[a]	4.2[a]
No. of dentists per 10,000 inhabitants	5.9	8.6
No. of physicians per 10,000 inhabitants	7.4	14.7
Percentage of denture wearers		
Total	29.8[a]	31[b]
Males	27.1[a]	26[b]
Females	32.4[a]	37[b]
Percentage of habitual smokers		
Total	35.2[a]	35.7[c]
Males	42.2[a]	49.2[c]
Females	28.3[a]	22.8[c]

Figures based on individuals aged
[a] 15 years or above
[b] 16 years or above
[c] 15—75 years

Information in Table 12 has been obtained from:

National Central Bureau of Statistics. Population and Housing Census 1970 (SOS). Part 2. Population in localities. Stockholm 1972.

National Central Bureau of Statistics. Population Dec. 31, 1973 according to the subdivisions of Jan. 1, 1974 (SOS). Part 1. Communes and parishes. Stockholm 1972.

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INDEX TO DIAGNOSTIC LABELS

Page numbers in *italics* refer to definitions