MASTEROPPGAVE

Prevalence of Oral Mucosal Lesions

at external dental clinics of the University of Tromsø and a comparison with other studies

Gro Eirin Olsen Holde og Ida Birgitte Hammervold

Veileder: Maud Bergdahl

UNIVERSITETET I TROMSØ
Det helsevitenskapelige fakultet
Institutt for Klinisk Odontologi

Juni 2010

Abstract

Introduction

The aim of this study was to find the prevalence of oral mucosal lesions in the adult and child populations, examined or treated at external university dental clinics of the University of Tromsø, and then compare these prevalences with prevalences found in previous studies.

Methods

A questionnaire was sent to the external University dental clinics (n=14), inviting all dental practitioners and hygienists to participate. During eight weeks the examiners reported all the oral mucosal lesions found during examination or treatment of their patients.

Results

Twelve out of 14 clinics participated in the study (86%), and a total of 8088 (3122 adults and 4966 children) patients were examined by 39 examiners.

Oral mucosal lesions were found in 7.59% of adult patients and in 2.68% of patients under the age of 18. The prevalence's of oral mucosal lesions were generally lower compared to previous studies.

Conclusion

Some of our results may indicate underregistration of oral mucosal lesions compared to previous studies. However, the previous studies have been conducted in other countries, and may not produce valid estimators of prevalences in Norway. To find the prevalence of oral mucosal lesions in Norway, a study on a general population that is stratified and randomly selected should be carried out.

Index

1.0 Introduction

Dental practitioners and hygienists have an essential role in detecting diseases in the oral cavity. Diagnosis and treatment of oral mucosal lesions should be just as important as treating caries and other dental diseases in a dental practice.
Previous studies on the prevalence of oral mucosal lesions are relatively sparse. Norwegian studies mostly consider specific oral lesions, case-reports and special non-representative populations (1,2,3,4). Studies of a general adult population have been conducted in Sweden (5), the U.S. (6) and Germany (7). Studies of a general child and youth population have been done in the U.S. (8,9) and of a general child population in Brazil (10).

With many patients and not enough time it is possible that the dental practitioner or dental hygienist gets too focused on the teeth, and forgets the important role he or she has in detecting and diagnosing oral mucosal lesions. We have experienced a difference in how thorough the oral mucosa is examined during our training in the University dental clinic, and during work in the summer at public dental clinics. When time is short during an examination, the oral mucosa is sometimes not checked properly. We therefore hypothesized that oral mucosal lesions are underregistered by general dental practitioners and hygienists.

The purpose of this study was to find the prevalence of oral mucosal lesions in the adult and child populations, examined or treated by dentists and hygienists at external university dental clinics of the University of Tromsø, and then compare the prevalences of our study with prevalences found in previous studies.

2.0 Oral mucosal lesions

2.1 Leukoplakia

Leukoplakia is an asymptomatic white patch on the oral mucosa, including the tongue, which can not be wiped off or characterized clinically as any other lesion. Leukoplakia can be divided into homogenous lesions with a smooth surface and non-homogenous with a verrucous, papilla-like, nodular or spotted surface, or as a mixture of white and red lesions.

The cause is unknown. It could be related to use of tobacco, use of alcohol, trauma, infection or nutritional factors. Approximately 5% of leukoplakias are malignant at the time of excision, and 5% undergo malignant transformation (11).

2.2 Erythroplakia

Erythroplakia appears as an asymptomatic well-defined, velvety red patch that can not be diagnosed clinically as any other disease or lesion. It is usually found in the floor of the mouth, the tongue and behind the molars. Focal white areas can also be seen in some lesions. Because most of the lesions are in situ or invasive squamous cell carcinoma, the causes are believed to be the same as the reason for oral cancer: tobacco, alcohol, nutritional defects and other factors. About 40 % of the lesions show severe dysplasia, and about 50 % are invasive (11).

2.3 Oral lichen planus and lichenoid reactions

Oral lichen planus can appear in different forms. The most common form is the reticular form, which appear as a white keratotic pattern of threads and webs. The lesion is most often found on the buccal mucosa, bilaterally, and is asymptomatic. The erythematous form appears as thin white striae with red patches and commonly affects the attached gingiva. Patients may feel discomfort. The erosive form has an ulcerated center covered by a fibrinous plaque or pseudomembran, with keratotic striae peripherally.

The cause of the lesion is unknown, but generally it is considered to be an immunologically mediated process. The risk of malignant transformation is higher with

the erosive form, approximately 0.4–2.5%. Oral manifestations of drug reactions and dental materials, may look like lichen planus. They are called lichenoid reactions (11).

2.4 Exophytic neoplasia

Oral fibroma

Oral fibroma is an asymptomatic, firm, broad-based nodule caused by a reaction to trauma or consistent irritation to the oral mucosal membranes. The trauma or irritation is repaired by the formation of fibrous connective tissue and results in a sub-mucosal scar. The lesion is found in areas subjected to trauma, like the buccal mucosa, lateral border of the tongue and lower lip. The fibroma is pale and usually does not become larger than 1-2 cm. If it is secondarily traumatized, the lesion can be ulcerated. No malignant transformation is seen (11).

Squamous papilloma/Oral Wart

The term oral squamous papilloma/oral wart includes papillary and verrucous growths of epithelium origin that contains minor amounts of supporting connective tissue.
Squamous papilloma is painless, and appears as a white to pink, soft, cauliflower-like lesion. It is usually solitary and located on the tongue, floor of the mouth, palate, uvula, and lips. The cause is mostly *Human papilloma virus* or of unknown origin. The lesion has no malignant transformation.
An oral wart can be considered a type of papilloma. Keratin overgrowth causes the projective growth above the surface. The overgrowth is caused by *Human papilloma virus* (11).

2.5 Herpes and aphthous lesions

Herpes lesions are caused by *Herpes simplex virus*, most commonly by the type 1 virus. The primary infection, usually occurring during childhood, first appears as multiple vesicles followed by small oral ulcers. It is painful, and usually accompanied by fever and gingivitis. The primary lesions can appear on any mucosal site. The secondary infection is caused by reactivation of the latent virus, and starts with a burning or tingling sensation, followed by vesicles and then multiple small ulcers. The reactivation is related

to a local “breakdown” of the immune system, e.g. UV-light, stress, or a change in local inflammatory mediators. The secondary lesion appear on the vermilion and the surrounding skin, and intra orally on the hard palate or gingiva.
Both the primary and secondary lesion heals in approximately 2 weeks. Herpes simplex 2 is associated with carcinoma of the cervix, but the association of Herpes simplex 1 and oral cancer is unclear (11).

Aphthous lesions are the most common non-traumatic ulceration that affects the oral mucosa. They appear as round, oval, superficial yellow-white blisters or lesions with a red halo. They can be located on the tongue, palate, floor of the mouth or the mucosa of the cheeks and lips. There are three forms of aphthous ulcers: Minor, major and herpetiform aphthae. Minor aphthae appears as 1-5 ovoid ulcers, less than 0.5 cm in size, located on non-keratinized mucosa. Major aphthae are bigger than 0.5 cm, with a more ragged, oval shape. Herpetiform aphthae consists of multiple small ulcers and can be located on any intraoral site. The cause of aphthous ulcers is unknown, but there is evidence that they are related to a focal immune dysfunction (11).

2.6 Tobacco related lesions

White lesions associated with smokeless tobacco

White lesions associated with smokeless tobacco are an inflammation and keratosis of the oral mucosa as a consequence of using moist snus. Dysplastic changes may appear after excessive or long term use. The risk of malignant transformation is low. The alterations in soft mucosal tissue are thought to be a response to constituents in tobacco and possibly other agents added in addition to tobacco. The alterations are seen in the area where the tobacco is placed. The clinical appearance is a granular to wrinkled mucosa, with heavy folded appearance in advanced cases. In some cases an erythroplakic or red area is present together with the white keratosis. The lesions are generally asymptomatic (11).

Nicotine stomatitis

Keratosis related to the use of tobacco, typically pipe and cigar smoking is called nicotine stomatitis. The severity of the condition aggravates with increased intensity of smoking.

The smoke has a direct topical effect on the palatal mucosa, as a result of tobacco carcinogens and heat. Initially the palatal mucosa is erythematous, followed by keratinization. Red dots surrounded by white keratotic rings then appear in the palate, as a result of inflammation of the salivary gland excretory ducts. Nicotine stomatitis rarely transforms to malignancy, but should be viewed as a marker of heavy tobacco use and a potential indicator of epithelial dysplasia in the oral cavity other than the hard palate (11).

2.7 Non-specific ulcerations

Ulceration is defined as the loss of epithelium.

This oral mucosal lesion does not include aphthous ulcers, herpetic ulcers or ulcers caused by trauma (11).

2.8 Candidiasis

Candidiasis is caused by *Candida albicans* and other *Candida* species in the oral microbial flora. These are commensal organisms found in the oral cavity in a majority of the population. Opportunistic overgrowth is related to local and systemic factors e.g. immunodeficiency, systemic antibiotic therapy, xerostomia and poor oral hygiene.

Classification of Candidiasis:

Acute

Acute pseudomembranous candidiasis is the most common form. Clinical features are white, soft plaques that leave a painful erythematous, eroded or ulcerated surface when removed. The lesions may develop at any location, but is mostly seen in the buccal mucosa and mucobuccal folds, oropharynx and the lateral aspects of the tongue. Young infants, elderly people and immunodeficient patients are commonly affected.

The erythematous form succeeds persistent pseudo membranous candidiasis. The clinical feature is a generalized red lesion. Depapillation and dekeratinization along the dorsum of the tongue may be seen.

Chronic

Chronic erythematous candidiasis is commonly seen in individuals who wear complete maxillary dentures. Clinical feature is a bright red, velvety to pebbly surface, with little keratinization.

The hyperplastic form may occur in the retrocommissural area and is known in some classifications as *candidal leukoplakia*. The lesion resembles speckled leukoplakia and may represent a premalignant lesion. Hyperplastic candidiasis may also occur on the dorsum of the tongue and is seen as a red, lobular elevation anterior to the circumvallate papillae in the midline. It is usually asymptomatic and is known as *median rhomboid glossitis* (11).

2.9 Traumatic ulcers

Traumatic ulcer is the most common lesion in oral soft tissue. Mechanical trauma and accidental trauma are the main causes for these lesions, where a cause-and-effect relationship often is clear. They can also be iatrogenic (accidentally caused by health care personnel, a medical treatment or diagnostic procedures) and in unusual circumstances self-induced because of an abnormal habit. Traumatic ulcers can be divided into acute or chronic ulcers. Acute ulcers can often be related to a trauma. They are often painful and heal in 7 to 10 days if the cause is removed. The ulcers are covered by a yellow-white fibrin network and surrounded by an erythematous halo. Chronic ulcers may cause little or no pain, and they can have delayed healing if irritation is continuous. The base is covered by a yellow membrane, with elevated margins that sometimes shows sign of hyperkeratosis. Indurations, because of scar formation and chronic inflammatory cell infiltration, are often seen with these lesions. Chronic traumatic ulcers may often resemble carcinoma and infectious ulcers, and it is important to consider differential diagnosis (11).

2.10 Geographic tongue

Geographical tongue is a completely benign condition of unknown cause. The clinical features are red atrophic patches, surrounded by elevated white keratotic margins. It usually affects dorsum and lateral surfaces of the tongue, more rarely other mucosal sites. The pattern changes over a period of time (days to weeks) and appears to move across the tongue. It periodically disappears and recurs. There is a strong association between geographic tongue and fissured (plicated) tongue. Lesions are usually asymptomatic, but

the red desquamated areas may be slightly painful. Symptoms are more common when fissured tongue is present, probably because of secondary fungal infection (11).

3.0 Material and methods

3.1 Selection of study population

The study population consists of patients examined or treated at external university dental clinics of the University of Tromsø. These patients have either contacted the clinic for an appointment or been called in for routine check-ups. Patients of all age groups have been included. There has been no selection based on gender, health condition or ethnicity.
The examination of the oral cavity was part of the standard treatment given to the patients. Therefore, the primary aim of the examiners was not to estimate the occurrence of oral mucosal lesions.

3.2 Selection of examiners

We decided to ask the dental practitioners and dental hygienists at the external University dental clinics to participate in the study. We presented our study to the external supervisors during a seminar at the University of Tromsø, and asked them to participate in the study along with their colleagues at the respective external University clinics. The examiners were not calibrated other than a guideline paper describing the selected oral mucosal lesion with a short text and picture (Appendix 1).

3.3 Selection of scientific literature

The following databases were searched for studies of the prevalence of oral mucosal lesions: MEDLINE and Pubmed. We used the following keywords in the search: prevalence; oral mucosal lesions; oral mucosal alterations. We extracted the papers with studies on general populations of adults and children/youths.
In the guideline paper we used Oral Pathology – Clinical pathologic Correlations as a reference (11).

3.4 Selection of oral mucosal lesions

The oral mucosal lesions included in the study were selected based on the most common lesions found in previous studies. The selection was made to simplify the comparison of our results with the existing material.

The following oral mucosal lesions were selected for our study:

For adults: Leukoplakia, erythroplakia, lichenoid reactions, connective tissue lesion, tobacco-related lesions, herpes and aphthous lesions, ulcerations and candidiasis. For children 0–18 years old: Traumatic lesions, herpes and aphthous lesions, candidiasis and geographic tongue.

3.5 The questionnaire

Ahead of the study we presented the aim and the methods of the study to the external supervisors and explained how to fill in the questionnaire.

The questionnaire (Appendix 2) had one table for adults and one table for children from 0-18 years old, where the examiner could make a note every time he or she observed an oral mucosal lesion when treating or examining patients. If the lesion did not fit the selected categories they could note it under a category called “other” and specify the lesion.

The tables were divided into four columns where each column represented two weeks – which gives a total duration of eight weeks. At the end of each two weeks the examiners were supposed to note the total sum of adult patients and child patients treated or examined during this period. The questionnaire also contained a short explanatory text.

A pilot questionnaire was shown to fellow dental students at the *Institute of Clinical Dentistry* (IKO) at the University of Tromsø.

The questionnaire was then sent to the 14 external University dental clinics along with a letter and the guideline paper. The guideline paper contained a text that described each oral lesion and clinical photos of the lesions. This was meant to assist the examiners in diagnosing the lesions.

We invited all the dentists and dental hygienists at the clinics to participate in the study.

3.6 Statistic analysis

The statistic calculations are based on the books “Statiskikk for helse- og sosialfagene” and “Practical Statistics for Medical Research” (12,13).

We calculated the 95% confidence interval (CI) by using the formula: $CI = p \pm (1.96 * se_{andel})$ where $se_{andel} = \sqrt{((p(100-p)/n)}$, where n = study population [12]. The 95%-confidence interval is the range of values which we can be confident includes the true value. Many statistical tests operate with a level of significance of 5 %, and therefore it is common to use 95% confidence intervals. A value that lies outside the 95 % confidence interval can be said to diverge significantly from the expectation.

The standard deviation (SD) between prevalences of oral mucosal lesions found at the different clinics from the total prevalences of oral mucosal lesions, was calculated by the formula: $SD = \sqrt{((\sum(x- \bar{x})^2)/n-1)}$, where n (number of observed values) = 12 (the number of clinics participating in the study), x = each single observation and $\bar{x}$ = the mean value (12). The standard deviation indicates the average, or standard deviation of scores away from the mean. Ergo how much the observations from the different clinics deviate from the mean observation (13).

4.0 Results

4.1 Participation

A total of 14 clinics were asked to participate in the study. Twelve (86%) clinics returned the questionnaires. One of the two non-participating clinics did not want to take part in the study. The second clinic was not able to participate because the external supervisor was not present at the clinic when the study was carried out.

Forty-two dentists and dental hygienists completed the questionnaires, but one of the questionnaires was rejected because the total amount of patients examined or treated was not stated.

The number of participating dentists and dental hygienists varied from clinic to clinic, with 8 at the most and in some clinics only one participant.

4.2 Results of the Study

Results I

The dentists and dental hygienists that took part in the study examined or treated a total of 8088 patients; 3122 adults and 4966 children (0-18 years).

The results of the study are presented in tables showing the actual number of oral mucosal lesions found, the prevalence of the lesions, interclinical variations, standard deviation (SD) and 95 percent confidence intervals (CI) for adults (Table 1) and children (Table 2).

The oral mucosal lesions reported in this study are diagnosed without the benefit of laboratory or histological tests. Therefore, for proper and definite diagnosis further tests should be taken of the reported oral mucosal lesions.

<u>Prevalence of oral mucosal lesions in adults</u>

A total of 3122 adult patients were examined, where 237 had oral mucosal lesions (7.59%). The most common oral mucosal lesion was tobacco-related lesions, seen in 68 patients (2.18%), followed by herpes and aphthous lesions found in 32 patients (1.03%)

and lichen planus found in 24 (0.77%). Erythroplakia was the least common lesion, found in 6 patients (0.19%).

Other lesions found, which were not included in the questionnaire, were traumatic ulcer, Fordyce's granules, gingival hypertrophy (medically induced), denture related ulcer, geographic tongue, amalgam tattoo, mucocele and exostoses.

All oral mucosal lesions are listed in Table 1.

Table 1. Prevalence of oral mucosal lesions in adults >18 years.

Mucosal lesion adults	Number (n)	Prevalence (p) %	Interclinical variations	Standard deviation (SD)	95% Confidence interval (CI)
Leukoplakia	14	0.45	0.00–2.30	0.82	0.10–0.80
Erythroplakia	6	0.19	0.00–3.70	1.09	0.04–0.34
Lichen planus	24	0.77	0.00–1.57	1.05	0.46–1.08
Exophytic neoplasia (fibroma, papilloma)	18	0.58	0.00–1.52	0.55	0.31–0.85
Tobacco-related lesions	68	2.18	0.00–7.41	2.20	1.67–2.69
Herpes and aphthous lesions	32	1.03	0.00–2.78	0.98	0.68–1.38
Ulcerations	20	0.64	0.00–1.47	0.57	0.36–0.92
Candidiasis	21	0.67	0.00–10.00	2.90	0.38–0.96
Other	34	1.09	0.00–6.33	1.88	0.73–1.45
Total	237	7.59	–	–	6.66–8.52
Total amount examined/treated	**3122**				

Prevalence of oral mucosal lesions in children and youths

A total of 4966 children and youths (0–18 years old) were examined, and oral mucosal lesions were found in 133 (2.68%).

The most common lesions were herpes and aphthous lesions, found in 62 patients (1.62%), followed by traumatic ulcer found in 37 patients (0.75%). Candidiasis was the least common lesion, found in only one of the patients. The low prevalence of candidiasis gave a negative lower limit of the 95 percent CI and was truncated to zero*.

Other lesions found, listed in the "other" category of the questionnaire, were fibroma, tobacco-related lesions, angular chelitis, lichenoid reactions, erythroplakia and scar tissue. All oral mucosal lesions are listed in table 2.

Table 2. Prevalence of oral mucosal lesions in children and youths 0–18 years.

Mucosal lesions children and youths	**Number (n)**	**Prevalence (p) %**	**Interclinical variations**	**Standard error (SE)**	**95 % Confidence interval (CI)**
Traumatic ulcer	37	0.75	0.00–2.56	0.76	0.51–0.99
Herpes and aphthous lesions	62	1.25	0.00–5.13	1.57	0.94–1.56
Candidiasis	1	0.02	0.00–0.10	0.03	0.00*–0.06
Geographic tongue	10	0.20	0.00–1.82	0.52	0,08–0,32
Other	23	0.46	0.00–2.13	0.75	0.27–0.65
Total	133	2.68	–	–	2.23–3.13
Total amount examined/treated	**4966**				

* Negative lower bound rounded to zero

Results II

Oral mucosal lesions in adults

A comparison of the prevalences of oral mucosal lesions found in adults in the present study with Splieth et al. (2007), Axéll (1976) and Shulman et al. (2004) showed that we found a higher prevalence of erythroplakia. Compared to Axéll (1976) the prevalence of tobacco-related lesions was much lower, but it coincided with the prevalence found in Shulman et al. (2004). The prevalence of candidiasis in the present study was much lower than the prevalence found in the other studies. Over all, the prevalences found in the present study were lower then those found by Axéll (1976), but more in range with the prevalences found in the two other studies. See table 3. In our study the total prevalence of oral mucosal lesions in adults were 7.59%, which is lower than the total prevalences in the previous studies ranging from 28.24% to 11.75%.

Table 3. Comparison of studies of the prevalences of selected oral mucosal lesions in adults.

Table 3 Adults	**Shulman et al. 2004 (%)**	**Splieth et al. 2007 (%)**	**Axéll 1976 (%)**	**Hammervold & Holde 2009 (%)**	**Hammervold & Holde 2009 95% CI**
Leukoplakia	0.42	2.92	3.60	0.45	0.10–0.80
Erythroplakia	0.00	0.02	–	0.19	0.04–0.34
Lichen planus	0.10	0.48	1.89	0.77	0.46–1.08
Exophytic neoplasia (fibroma, papilloma)	0.73	3.00	3.46	0.58	0.31–0.85
Tobacco-related lesions	2.13	–	9.16	2.18	1.67–2.69
Herpes and aphthous lesions	2.50	1.66	5.40	1.03	0.68–1.38
Ulcerations	0.12	0.76	1.22	0.64	0.36–0.92
Candidiasis	6.21	–	16.64	0.67	0.38–0.96
Total	28.24	11.75	–	7.59	6.66–8.52

Oral mucosal lesions in children and youths

When comparing the prevalences found in children and youths in the present study with prevalences from Shulman (2005) and Bessa et al. (2004), we found that the prevalences in our study were lower in all categories. Compared to Kleinman (1994) our prevalences of Candidiasis and traumatic ulcers were some what higher. The total prevalence of oral mucosal lesions was 2.68% in the present study, compared to 4.08% found in Kleinman (1994), 10.26% found in Shulman (2005) and 27.00% found in Bessa et al. (2004) (Table 4).

Table 4. Comparison of studies of the prevalences of selected oral mucosal lesions in children and youths

Table 4 Children and youths	**Shulman 2005**	**Bessa et al. 2004**	**Kleinman 1994**	**Hammervold & Holde 2009**	**Hammervold & Holde 2009 95% CI**
Traumatic ulcer	2.30	8.34	0.09-0.29†	0.75	0.51–0.99
Herpes and aphthous lesions	3.09	2.72	2.01	1.25	0.94–1.56
Candidiasis	0.08	1.82	0.01	0.02	0.00*–0.06
Geographic tongue	1.05	9.08	0.60	0.20	0.08–0.32
Total	10.26	27.00	4.08	2.68	2.23–3.13

† In Kleinman's study the traumatic ulcers were divided in two categories (Non-specific ulcers (0.09%) and "other"(0.29%)) so the prevalence is somewhere between these numbers.

Table 5. The distribution of adult and child/youth patients examined or treated at the twelve participating clinics and the total amount of patients

Clinic:	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**All clinics**
Adults	156 (74%)	304 (44%)	10 (20%)	87 (49%)	92 (22%)	158 (38%)	27 (40%)	339 (46%)	357 (34%)	765 (39%)	396 (29%)	431 (47%)	3122 (39%)
Children	55 (26%)	393 (56%)	39 (80%)	89 (51%)	328 (78%)	257 (62%)	40 (60%)	399 (54%)	706 (66%)	1187 (61%)	993 (71%)	480 (53%)	4966 (61%)
Total	211	697	49	176	420	415	67	738	1063	1952	1389	911	**8088**

5.0 Discussion

This survey was a cross sectional study designed to register the prevalence of oral mucosal lesions in an adult and child population in selected regions in Norway. Because the study is cross sectional, we can only explore associations and not causal relationships. The study had a high participation rate (86%), and thanks to the cooperation from the external University clinics we received a material with a study population size which is comparable to other studies.

The study design was chosen because we had a short period of time to conduct the survey, and because a part of our aim was to compare the number of registered oral mucosal lesions at external University dental clinics with studies done on a general population. We also chose to work with the external University dental clinics because we thought we were more likely to get a high participation rate.

Prior to the study we presented our report to the external supervisors, so they could ask questions regarding the selected oral mucosal lesions, the questionnaire itself and how to organize it. There was no calibration of the examiners and we did not use any standardized criteria regarding the conditions examined, except the guideline paper. Laboratory or histological tests were not used in the identification of the oral mucosal lesions, although oral mucosal lesions require this for a proper and definite diagnosis. Because of the lack of standardizations regarding lesion diagnostics it is probable that there are inter-examiner variations. The diagnostics of the lesions are therefore mostly based on the previous knowledge and experience of the examiner. Although there were no standardized examination settings we do not think this had a big impact on the reporting of lesions, because all patients were examined in a dental chair and with similar light conditions.

The selection of patients is not representative for a general population. Some of the patients have sought help, and are in a way self-selected. Individuals who do not seek dental care might be healthier than those who attend the clinic, or on the contrary less healthy but does not bother or is not aware of their condition. The study may overstate the prevalence of oral mucosal lesions that present as acute problems because patients may be more inclined to seek treatment for these lesions.

Also because of the study design, the same patient could have had several dental appointments during the study, so it is possible that the same lesion has been registered more than once.

The study period was only eight weeks. This might have caused overrepresentation and underrepresentation of some age groups of children, due to different times of recall for the different age groups. However, there is now a more individual recall in the public dental health system than previously, when children were called at regular time intervals according to age instead of dental health risk. Indicator classes (5, 12 and 18 years of age) are still examined on a regular basis. As mentioned above, this could have caused over- or underrepresentation of these age groups depending on if they were called or not during the study period. One of the clinics reported that the amount of tobacco related lesions in children/youths were most likely too low, because the age groups most prone to have tobacco related lesions were not examined at the time of the study.

The examiners in the study participated on a voluntary basis and this could have led to an unrepresentative selection of examiners, which could represent a possible bias regarding our hypothesis of under registration of oral mucosal lesions. Also, the registration of the oral mucosal lesions was only a part of the examination/treatment given to the patient, not the sole purpose. This represents a weakness in our study because there is a risk that the examiners had a low registration priority. On the other hand it gives a picture of how many oral mucosal lesions that are discovered in a regular day at a dental office.

Comparing with other studies

The articles we selected were based on studies of a representative population. We had difficulties finding studies based on this criterion, therefore only three articles for adults and three articles for children were selected. Most studies of oral mucosal lesions looked at specific oral manifestations of mucosal lesions or cause-related factors like tobacco, amalgam etc., or they looked at special populations. Further more, to compare findings in previous studies is difficult because they often do not use standardized methods of measuring oral mucosal lesions. There are standardized WHO criteria regarding the oral

mucosal lesions examined, but few studies used these. Variations are also seen in examination settings (e.g. lighting conditions, position of examiner and subject), different methods in calibrating examiners and different diagnostic aids (e.g. serology, saliva sampling).

The oral mucosal lesions included in our study were those commonly found in the selected studies. By only including the most common oral mucosal lesions for our study, we were more likely to get enough cases of the different oral mucosal lesions to get reliable prevalences. No similar study has been done in Norway, as we know of. Therefore we had to base the selection of oral mucosal lesions on studies of representative populations in other countries. However, we do not know if the most common oral mucosal lesions found in other countries are the most common lesions in our region.

The selected studies often had subcategories of the oral mucosal lesions. To compare our findings with the other studies, we sometimes had to add up the prevalences for those subcategories. An example is the category "Herpes and aphtheous lesions" in adults in our study. This category includes the prevalences for subcategories like recurrent aphthae, herpes labialis, intraoral herpetiform lesions and herpetic gingivostomatitis found in the other studies. In addition, the different studies had not divided the oral mucosal lesions into similar subcategories. Adding up the prevalences of different subcategories of oral mucosal lesions may lead to inaccuracies. To get the most accurate numbers for comparing, we decided to include the subcategories that fit the description of the oral mucosal lesions given in the guideline paper.

In adults the total prevalence of oral mucosal lesions (7.59%) was lower than the total prevalences found in the other studies (11.75-28.24%). The prevalence of the specific oral mucosal lesions also varied compared to the other studies.

Erythroplakia had a significantly higher prevalence in our study (0.19%) compared to the other studies (0-0.02%), lying outside the 95% CI (0.04-0.34). This can be a random accumulation of the lesion in the study, or it can be incorrect diagnostics. No laboratory or histological tests were taken as a part of the study, and may result in an unreliable diagnosis. Prevalences for rare conditions should be interpreted with caution, especially

when the study population is small. Erythroplakia has a high malign transformation risk (11), but because of the study design we have no means of assuring that patients in the study diagnosed with the lesion are followed up. We can not trace the patients or examiners because the questionnaires were anonymous. We are only able to identify the clinics. Therefore all the clinics will be contacted and recieve information about our results.

Candidiasis had a significantly lower prevalence (0.67%) compared to the other studies (Shulman 2004, Splieth 2007, Axéll 1976) ranging from 6.21-16.64% lying outside the 95% CI of candidiasis from our study (0.38-0.96). The low prevalence in our study could be a result of the guideline paper missing a specific category for denture stomatitis under the candidiasis section. Also, denture stomatitis is a lesion associated with specific risk factors (e.g. use of removable dentures) where the overall prevalence depends on the underlying distribution of the risk factors. Therefore, one should be careful to compare prevalences for such lesions without knowing the underlying distribution of their risk factors.

Tobacco-related lesions (2.18%) are in range with Shulman (2004) (2.13%), but much lower than Axéll (1976) who found a prevalence of 9.16% of which 8.04% were snuff dipper's lesions. Snuff is commonly used in Scandinavia (14), and the use of snuff is increasing in Norway. The prevalence of daily snuff-users in Norway in 2008 was 6 % (15), and in 50-60 % of daily users white changes of the oral mucosa are seen (16). This indicates that the prevalence of tobacco-related lesions found in our study is too low and should be close to 3%. Preliminarily we presumed that the prevalences of oral mucosal lesions found in Uppsala County in Sweden by Axéll (1976) would coincide most with our findings, because of the similarities between the Swedish and Norwegian population and customs. However, all the prevalences gathered in our study differed greatly from those found in Axéll (1976).

Herpes and aphthous lesions had a significantly lower prevalence (1.03%, 95% CI 0.68-1.38) in our study compared to the three other studies (1.66-5.40%) (Shulman 2004, Splieth 2007, Axéll 1976). We cannot se any apparent reasons for this low prevalence of herpes and aphthous lesions, and it may therefore be an indication of underregistration.

Ulcerations had a prevalence of 0.64 % (95% CI 0.36-0.92) which was lower than the prevalence found in Axéll (1976) (1.22%) but higher than the prevalence found in Shulman (0.12%) (2004). The category we compared ulcerations to was in Shulman (2004) "non-specific ulcers" and in Axéll (1976) "Ulcus mucosea oris NOS" which included all ulcers that were not caused by trauma. It is difficult to compare the different studies because they have all used different categories for ulcerations.
Both **leukoplakia** (0.45%, 95% CI 0.10-0.80) and **exophytic neoplasia** (0.58%, 95% CI 0.31-0.85) had a significantly lower prevalence than Spliet (2007) and Axéll (1976), but coincided with the results from Shulman (2004).
We believe that the large inter-clinical variations in our study are mainly a result of the difference in the amount of patients examined in the different clinics. The greatest deviation from the mean values is seen in clinics with few patients where registrations of oral mucosal lesions can give a heightened prevalence. In one of the clinics one case of candidiasis had been registered, but only ten patients had been examined, giving a prevalence of 10.0% (Table 5).

In children/youths the prevalences in our study were generally lower compared to previous studies, and the total amount of lesions found was much lower (2.68% compared to 4.08-27.00%).
The prevalence of **herpes and aphthous lesions** (1.25%, 95% CI 0.94-1.56) and **geographic tongue** (0.20%, 95% CI 0.08-0.32) was significantly lower than the prevalences found in all of the three studies, respectively ranging from 2.01-3.09% and 0.60-9.08% (Kleinman (1994), Shulman (2005), Bessa et al. (2004)). **Traumatic ulcerations** and **candidiasis** had a lower prevalence than two of the studies (Shulman (2005), Bessa et al. (2004)). This coincides with our hypothesis of underregistration. On the other hand, it may also be an effect of the study design. We included fewer oral mucosal lesions than the other studies, which will have an effect on the total prevalence, although there was a category in our questionnaire called "other" for oral mucosal lesions not mentioned in the guideline paper. The detection of oral mucosal lesions was not the sole purpose of the examination and there is a risk that lesions were overlooked or registered in passing. Another problem in the comparison of our study with the studies

we selected is possible differences in demographic characteristics of the study populations. Our study population is not randomly sampled and is not stratified for sex, socioeconomic status, race, geographic region etc.

The only stratification of patient groups was children 0–18 years and adults older than 18 years. In one of the other studies of child populations the children were divided in two age groups, 0–4 and 5–12 years (Bessa al. (2004)). In the two other studies children between the age of 2 and 17, and 5 and 17 were included (Shulman (2005), Kleinman 1994)). We chose to include children from 0 to 18 years, because the Norwegian dental health system regards them as children. This made the comparison of lesions found in the different age groups more uncertain. However, we feel that the comparisons are more or less reliable because the age span in our study is covered by the other studies, although no single study uses the same age categories. We have no means of ensuring that the study population is representative for the population of interest. But on the other hand, the population in Norway is rather homogenous and we do not think the lack of randomization will have a large effect on the outcome of the study.

6.0 Conclusion

In adults the registered prevalences of candidiasis, tobacco-related lesions and herpes and aphthous lesions were lower compared to previous studies (Shulman (2004), Splieth (2007), Axéll (1976)) and may indicate an underregistration of oral mucosal lesions. The same results were seen for geographic tongue and herpes and aphthous lesions in children and youths, where the prevalences found were lower compared to all three studies (Kleinman (1994), Shulman (2005), Bessa et al. (2004)). For both adults and children and youths the total prevalence of oral mucosal lesions registered was lower than in all of the previous studies (Shulman (2004), Splieth (2007), Axéll (1976), Kleinman (1994), Shulman (2005), Bessa et al. (2004)). This could indicate that the amount of oral mucosal lesions registered by general dental practitioners and dental hygienists in our study are lower than the actual numbers, and that the oral mucosa might have a lower examination priority than the dental hard tissues. Still it is difficult to compare our findings with previous studies, since they have been conducted in other countries, and may not produce valid estimator of prevalences in Norway. To find the prevalence of oral mucosal lesions in Norway, a study on a general population that is stratified and randomly selected should be carried out.

7.0 Acknowledgements

We would like to thank all the participating clinics and their staff for their kind cooperation and interest in this study. It would not have been possible without their help.

Alta tannklinikk

Ankenes tannklinikk

Finnsnes tannklinikk

Hamar tannklinikk

Hammerfest tannklinikk

Lyngseidet tannklinikk

Målselv tannklinikk

Namsos tannklinikk

Selfors tannklinikk

Skien tannklinikk

Storslett tannklinikk

Sør-Tromsøya tannklinikk

A special thanks to our supervisor Maud Bergdahl, Professor of Clinical Dentistry, for her help and guidance with the master paper.

8.0 References

1) Holsen DS, Johannessen AC. Sykdommer som affiserer hud og munnslimhinne Tidsskr Nor Lægeforen 2006; 126: 1214–7.

2) Steinsvoll S, Herlofson BB. Orale manifestasjoner ved HIV-infeksjon. Tidsskr Nor Lægeforen 2006; 126: 1218–21.

3) Morken T & Gjerdet NR. Kontaktallergi i munnhulen. Nor Tannlegeforen Tid 2006; 116: 404–7.

4) Løkken P, Skoglund LA. Legemiddelbivirkninger i munnhulen. Tidsskr Nor Lægeforen 2006; 126: 1345-48.

5) Axéll T. A prevalence study of oral mucosal lesions in an adult Swedish population. Odontol Revy Suppl. 1976;36:1-103.

6) Shulman JD, Beach MM, Rivera-Hidalgo F. The prevalence of oral mucosal lesions in US adults: data from the Third National Health and Nutrition Examination Survey, 1988-1994. J Am Dent Assoc 2004;135(9):1279-86.

7) Splieth CH, Sümnig W, Bessel F, John U, Kocher T. Prevalence of oral mucosal lesions in a representative population. Quintessence Int. 2007 Jan;38(1):23-9.

8) Shulman JD. Prevalence of oral mucosal lesions in children and youths in the USA. Int J Paediatr Dent. 2005 Mar;15(2):89-97.

9) Kleinman DV, Swango PA, Pindborg 11. Epidemiology of oral mucosal lesions in United States school children: 1986-87. Community Dent Oral Epidemiol 1994; 22: 243-253.

10) Bessa CFN, Santos PJB, Aguiar MCF, do Carmo MA. Prevalence of oral mucosal alterations in children from 0 to 12 years olds. J Oral Pat Med. 2004 Jan;33(1):17-22 (6).

11) Regezi J. A. Sciubba J. J. Jordan R. C. K. Oral pathology: Clinical Pathologic Correlations. 5th ed. London: Saunders; 2008.

12) Bjørndal A & Hofoss D. Statistikk for helse- og sosialfagene. Rev. 1. Oslo: Gyldendahl; 2004. p. 47-48, 64-65.

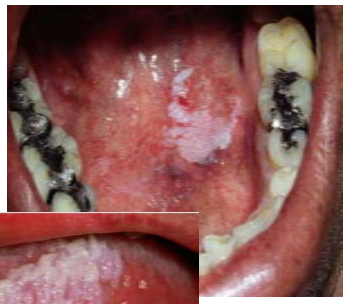
13) Altman DG. Practical Statistics for Medical Research. London: Chapman & Hall; 1993. p. 348.

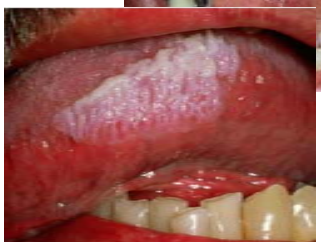
14) Johnson N, Tobacco use and oral cancer: A global perspective. J Dent Educ 2001; 65: 328-39.

15) http://www.helsedirektoratet.no/tobakk/statistikk/bruk_av_snus_/ read Feb. 1st. 2010.
16) Åstrøm AN, Bui L, Læknes H, Johannessen AC, Gjerdet NR. Snusbruk og holdninger til snus. Nor Tannlegeforen Tid 2007; 117: 146 – 52.
17) Rioboo-Crespo Mdel R, Planells-del Pozo P, Rioboo-García R. Epidemiology of the most common oral mucosal diseases in children. Med Oral Patol Oral Cir Bucal. 2005 Nov-Dec;10(5):376-87.
18) Furlanetto DL, Crighton A, Topping GV. Differences in methodologies of measuring the prevalence of oral mucosal lesions in children and adolescents. Int J Paediatr Dent. 2006 Jan;16(1):31-9.
19) Erguna S, Özelb S, Koraya M, Kürklüa E, Ak G, Tanyeri H. Dentists' knowledge and opinions about oral mucosal lesions. J Oral Maxillofac Surg. 2009 Dec;38(12):1283-1288.
20) Zatterstrom UK, Svensson M, Sand L, Nordgren H, Hirsch JM. Oral cancer after using Swedish snus (smokeless tobacco) for 70 years – a case report. Oral Diseases. 2003 Dec;10(1):50-53.
21) Gordis L. Epidemiology. 3rd ed. Philadelphia: Elsevier Saunders; 2004.

Appendix 1

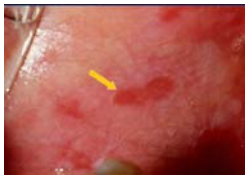
Oversikt over orale slimhinneforandringer





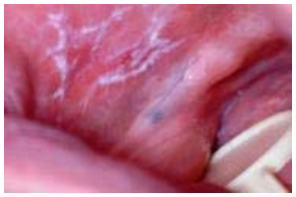
Leukoplaki
Ikke-avskrapbar hvit flekk i munnslimhinnen, inkludert tungen. Leukoplaki er en utelukkingsdiagnose som gis dersom forandringen ikke kan identifiseres som noen annen hvitaktig sykdom eller forandring. Leukoplakier deles inn i homogene (glatt overflate) og non-homogene (verrukøse – papilomatøse/vortelignende, nodulære – spettede,/lett hevede, og erytro-leukoplakier (blanding av hvite og røde slimhinneforandringer)

Erytroplaki

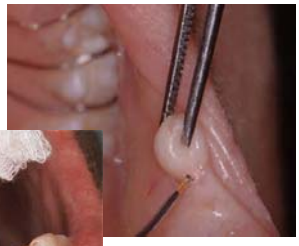


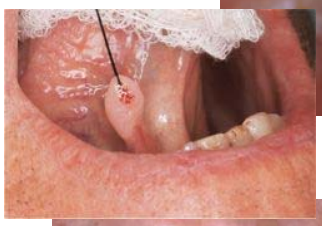
En erytroplaki er en rød flekk som ikke kan diagnostiseres som noen annen sykdom eller lesjon. Den opptrer ofte som en velavgrenset lesjon med en jevn fløyelsaktig rødfarge

Lichenoide forandringer
Et hvitt mønster av tråder og nett (retikulær form), eller det kan være partier som kun er røde (erytematøs form), eller det kan være sårdannelser (erosiv form).



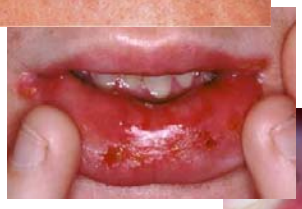
Bindevevsutvekster (fibrom, papillom)
Fibrom, fast konsistens, normal slimhinnedekning, bredbaset. Fibrøst bindevev
Papillom, blomkållignende utvekst, velavgrenset, vanligvis liten og hvit/lys rosa. Epitelutvekst med bindevevsstreng

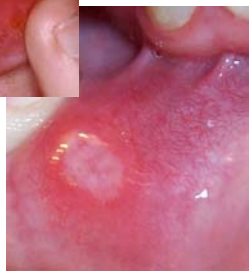




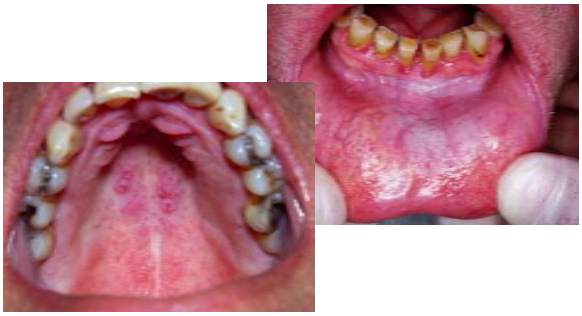
Herpes og after
Herpes: Klaser av blemmer som kan ligne på after

After: runde til ovale, overflatiske, gulhvite blemmer eller sår med rød halo. Kan finnes på tungen, i ganen, i munngulvet, på innsiden av kinnene eller leppene. De er vanligvis 2-8 mm i diameter, men kan bli mye større.



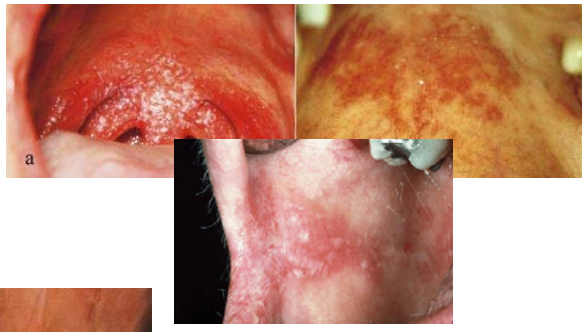


Tobakkrelaterte endringer
Hyperkeratinisering som følge av snusbruk, nikotin stomatitt etc.

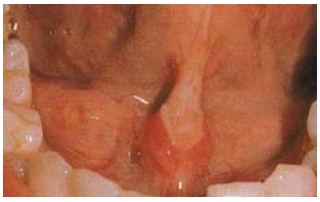


Ulcerasjoner
Defineres som tap av epitel
Ulcerasjoner som ikke er after, herpetiforme eller av traumatisk årsak

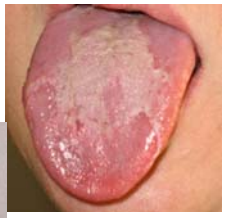
Candida
Pseudomembranøs (hvite avskrapbare kolonier)
Erythematøs (rød slimhinne)
Hyperplastisk (hvitt keratinisert plakk)

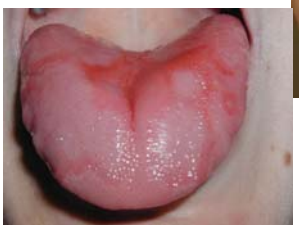


Traumatiske sår
Sår på grunn av mekanisk skade



Geografisk tunge
Karakteriseres av atrofiske flekker omgitt av forhøyede keratiniseringer. De deskvamerte områdene er røde og kan være ømme. Over tid vil mønsteret flytte seg over tungeryggen.





Appendix 2

Spørreskjema – orale slimhinneforandringer

Dette spørreskjemaet gjelder for 4 x 2 uker, der to og to uker er slått sammen i én kolonne. Hver gang man ser en av følgende slimhinneforandringer noteres det ned i ruten ved siden av (en strek). Det er *ett skjema for voksne* og *ett for barn.*

Etter to uker teller man *alle pasientene man har behandlet*, totalt antall voksne pasienter og totalt antall barnepasienter, og fører det inn på nederste rad.

Voksne: (19-…år)

	u. 16-17	u. 18-19	u. 20-21	u. 22-23
Leukoplaki				
Erytroplaki				
Lichenoide forandringer				
Bindevevsutvekster (fibrom, polypp)				
Tobakksrelaterte forandringer				
Herpes og after				
Ulcerasjoner				
Candida				
Annet (……)				
Antall behandlede pasienter totalt (Med og uten slimhinneforandringer)				

Barn (0-18år)

	u. 16-17	u. 18-19	u. 20-21	u. 22-23
Traumatiske sår				
Herpes og after				
Candida				
Geografisk tunge				
Annet (……)				
Antall pasienter totalt uke 1-2 (Med og uten slimhinneforandringer)				