**Chapter One: Introduction** 

# 1. INTRODUCTION

Prevalence is a measurement of the proportion of population actually having the disease at a specific period of time; in other word the prevalence tells us of the number of people with the disease divided by the number of population at a specific time (Webb et al., 2005).

The prevalence provides an estimation of probability that an individual will have oral disease at a specific period of time and also identifying risk groups within the population studied (Heinekens and Buring. 1987).

Globally, studies providing a wide spectrum of oral lesion includes a study reporting on the prevalence of oral lesions among 20,000 adult Swedish population by Axell (1976) and another study carried out in the United States of American (USA) which reported the common oral lesions among the USA population (Bouquot., 1986). In Asia the survey provided the prevalence of oral lesions was found to be in Indian population (Smith et al., 1975; Metha et al., 1972).

Tobacco smoking and alcohol consumption has long been associated and indicated worldwide as the major factors in the development of cancer and other systemic diseases in developed countries (Peto et al., 1992; Jaber et al., 1999). Smoking habit has a great impact on oral carcinogenesis prior to malignant transformation and the alcohol drinking in high level has also been shown to have strong association with oral cancer in the American Population (Morse et al., 2007).

In Asian countries particularly in India, oral cancer and precancer has been associated with betel quid chewing (Nair et al., 1999). Betel quid chewing is a common habit in many Asian countries and this habit spread to other regions of the world through emigration (Reichart et al., 1987).

Similar studies have been also conducted Malaysia had been able to identify the high risk groups for oral precancerous lesions where strong causal relationship with quid chewing was reported (Zain and Ghazali 2001). In Malaysia approximately 25% of all causes of death in Malaysia are due to tobacco usage (Ministry of Health Malaysia, 1997). A nationwide survey on oral mucosal lesions in Malaysia in 1993/1994 (Zain et al., 1997) showed high prevalence of oral precancerous lesions among the Indians and Indigenous people of Sabah and Sarawak who practiced betel quid chewing. In Yemen Scheifele et al (2007) reported a significant association between oral leukoplakia and shammah usage (tobacco quid form of quid). The prevalence of oral cancer was 1% among Shammah users. Among the gat chewer's there are different of oral keratotic white lesions with different degrees of underlying pathology depending on the frequency and duration of qat chewing (Aiman et al., 2004). Histopathologic alteration in the oral mucosa such as acanthosis, orthokeratosis, epithelial hyperplasia with irregular rete redges have been described in gat chewers. Qat chewing with cigarette or water-pipe smoking may increase the risk of developing such pathologic changes in the oral mucosa (Aiman., 2007). Although prevalence of oral mucosal lesions has been reported in many countries, these prevalence data are usually restricted to very few lesions in each study. There is thus a need to obtain data from different countries with a large random sample which can be tedious and thus appropriate information on oral mucosal lesion prevalence can still be obtained from small low budget studies on selected population (Axell et al., 1990).

**Chapter Two: Literature Review** 

## 2.1. Prevalence of oral mucosal lesions

Reports on prevalence of oral mucosal lesions showed variations in the prevalence rate which may be related to methodology, difference in diagnostic criteria used, selection of participants and the risk habits practiced among the population.

## 2.1.1. Worldwide distribution

A study by Axell (1976) where he conducted oral examination on 22033 partciptants, reported the prevalence of oral mucosal lesions in adult Swedish population. In this study; the prevalence of about 60 oral mucosal lesions were recorded and compared with previous findings. Prevalence of lesions detected were of focal epithelial hyperplasia (0.11%), leukoedema (49.07%), geographic tongue (8.45%) and lichen planus (1.85%). Some lesions which was found in this study are directly or indirectly related to local etiologic factors such as denture status and tobacco habits.

Bouqout in 1986 reported on the prevalence of common oral lesions during mass screening of American population. The most oral mucosal lesions were white lesions which accounted for 37.6% however, the most common clinical appearance of oral lesions was that of a single, exophytic mass which accounted for 37.4% of all recorded lesions. In this study the leukoplakia reached to more than 26% and the prevalence of other lesions were listed as traumatic ulcer, aphthus ulcer, leukoedema, glossitis, ranula and candidiasis.

In Thailand the prevalence of oral mucosal lesions such as chewers mucosa was 13.1%. Leukoedema was12.4% and slightly more common among women, preleukoplakia was 1.8%, more among men and the leukoplakia was 1.1% which more frequent among men. High prevalence of smoking cigarrette was obseved among the middle age however, betel chewing was more prevalent among the old age. There was a postive correlation between some oral mucosal lesion and the risk habits (smoking ,quid chewing) (Reichart et al.,1987).

In a study conducted on adults Southern Chinese; the prevalence of oral mucosal lesion was found to be 13% in urban men, 6% in urban women, 15% in rural men, and 4% in rural women. Tongue lesions and white lesions were relatively common, this study showed there is a positive relationship between risk habits (smoking and alcohol consumption) and prevalence of oral mucosal lesions (some white lesions and tongue lesions) (Lin et al., 2001).

From a study conducted in 2004 in London among alcohol misusers, the prevalence of oral mucosal lesion was found to be 28.1% (n=227). The high prevalence of oral mucosal lesion was frictional lesions (8.8%), scar tissue of lip 4.8%, candidosis 3.8% and angular cheiltis 3.0%. The alcohol related lesion was white patch similar to the diagnosis of leukoplakia. The study also that 56% were alcohol users and 46% were alcohol and substance abuse users. The prevalence of tobacco smoking was 85% among only alcohol users and 95% among the other group (alcohol with substance abuse). There was no significant relationship between the prevalence of oral mucosal lesion and smokers (Harris et al., 2004).

The prevalence of oral mucosal lesions in South India was 4.1%. The prevalence of leukoplakia, oral submucous fibrosis and oral lichen planus was 0.59%, 0.55%, and

0.15% respectively. The prevalence of smoking, alcohol drinking and quid chewing was 15.02%, 8.78% and 6.99% respectively. Smoking and quid chewing were significant predictors of leukoplakia in this population (Saraswathi et al., 2006).

In Taiwan the prevalence of leukoplakia, erythroplakia, oral lichen planus, oral submucous fibrosis and verrucous lesions were 7.44%, 1.95%, 2.98%, 1.58% and 0.84% respectively. The prevalence of smoking habit was 20.4%; areca nut chewing was 7.16% while high prevalence of alcohol consumption which was 18.14%. There is a statistically significant association between leukoplakia, oral submucous fibrosis, verrucous lesions and the risk habit, areca quid chewing (Chung et al., 2005).

#### 2.1.2. Malaysian prevalence of oral mucosal lesions

The first epidemiology study in Malaysia was a dental survey conducted in 1962 where the interdepartmental committee on National defence (ICCND) comprising a joint United States – Malaysia team conducted in a Federation of Malaysia Nutrition Survey, quoted from Zain et al (1997). The next population–based dental survey was conducted by the Ministry of Health Malaysia in 1974/75. This study was confined to peninsular Malaysia where the precancerous lesion was found (1.3%). However, the other lesions was (0.4%) where put under smoker keratosis.

From the early study was that study done by Ramanathan et al (1973(a)) and reported the prevalence of Oral cancer and precancer was found (1.5%) where the prevalence of oral cancer was (0.5%). However, the precancerous lesions included smoker's keratosis was found (0.12%). Other study by Ramanathan et al (1973(b)) which conducted on 407 medical attendants and health workers and reported 55 (13.55%) subjects had oral precancerous lesions. Also the smoker's keratosis included in this study of which 6 subjects (12.0%) had oral precancerous lesions.

The other study in 1978 carried out by Dental Division by Ministry of Health on total of 9073 Malaysian subjects and reported the prevalence of leukoplakia was (1.3%), erthroplakia (0.2%) and oral cancer as (0.01%).

Many studies in Malaysia reported that the quid chewing is a risk factor like other countries in the spread of oral mucosal lesion particularly oral precancerous and cancerous lesions, the Indian and Indigenous people were high risk group especially the women in both group due to using the tobacco in their quid (Gupta et al., 1997).

The positive association of oral mucosal lesion and cigarette smoking such as leukoedema as well as denture stomatitis. There was no relationship between the cigarette smoking and prevalence of aphthous ulcer and coated tongue. There was no statistically significant differences between the cigarettes smokers and non smokers in prevalence of pre-leukoplakia (Zain and Razak.,1989).

The prevalence of oral soft tissue lesions in Malaysia was recorded from examination of dental outpatients in Thailand and Malaysia where three cases of leukoplakia (1.3%), one case of betel quid related lesion and one case squamous cell carcinoma (0.4%) was detected in Malaysians. The was a high prevalence of lichen planus (2.1%) in Malaysian oupatients. The prevalence of tobacco in some form was 27.5% where the cigerrate smoking was the predominant habit and the prevalence of quid chewing among the Malaysian out patients was 2.6% (n=6). Three tobacco associated leukoplakia were found and also three betel quid lesions (Axell et al., 1990).

The prevalence of oral mucosal lesions among elderly Malaysians was found to be 22.8% (n=111). A total of 145 oral lesions were detected. The prevalence of oral mucosal lesions was highest among Indians and least among the Chinese. The most common finding was tongue lesions which was found to be 10.7%, followed by oral pigmentation (4.9%) and white lesions (4.3%). Denture related lesions were comparatively low at 2.5%. Two cases of oral cancer was detected giving a relatively high prevalence of 0.4 % (Taiyeb et al., 1995).

A nationwide Malaysian dental survey showed the prevalence of oral mucosal was 9.7% with no predictable difference between males (9.1%) and females (10.1%). The most common lesion was denture stomatitis; leukoplakia, an oral precancerous lesion was the most common oral lesion where the males and females ratio for leukoplakia was 3:1. The smokers palate was more among male while betel chewers mucosa was more among female. Five cases of oral cancer was reported in three male and two in female.

One humdred sixty five (165) subjects had oral lesions which includes precancerous lesions and 187 (1.6%) had betel chewers mucosa. The prevalence of oral precancer lesions in decreasing order was firstly the Indians (4.0%) followed by the in Other Bumiputras who are mainly the indigenous people of Sabah & Sarawak (2.5%). The lowest prevalence was among the Chinese(0.05%). The prevalence risk habits among Malaysian was found to be 19.2% smokers, 4.87% betel–quid chewers and 1.7% were alcohol consumers (Zain et al., 1997).

In a study by reviewing different types of studies that proved the importance in making comparisons between studies such as the incidence of data for oral cancer in Malaysia was reported by Hirayama in 1966, 35 years ago which estimated that 3.1 new cases per 100,000 population was diagnosed for the year 1963 (Zain and Ghazali., 2001).

#### 2.1.3. Yemeni prevalence of oral mucosal lesions

From earliest epidemiology studies in Yemen which deal about with the oral lesion was that study which was carried out in 1987 by Hill and Gibson. This study reported that keratosis of buccal mucosa was related to gat chewing.

The other study conducted in 2004 showed oral white lesions (oral kerstosis ) in 342 (22.4%) Yemeni subjects with a mean age of 27 years old with 87.4% being. The white lesion was graded from mild whitening in appearance to homogenous-like lesions. The prevalence of qat chewing in this study was 61.12% while the pevalence of smoking habit was found to be 26.36%. There was a significant relationship between risk habits (qat chewing, smoking, and shammah usage) and the prevalence of oral white lesions. (Aiman et al., 2004).

In a study carried out among the Yemeni shammah users, the prevalence of oral squamous carcinoma (OSCC) among the shammah users was 1% (n=2). The prevalence of mucosal burn (MB) was 31%, oral leukoplakia was 27%. No shammah users was diagnosed with either mucosal burn and or leukoplakia. When shammah associated lesiosn was combined, the prevalence of shammah-associated lesions was found to be 58%. The prevalence of lichen planus was 0.5% and oral lichenoid reaction was 4.0% while the prevalence of other lesions such as frictional lesion was 4.0%, pseudomemebranous candidosis was 2.5%, mosrsicatio buccarum was 0.5% and white sponge nevous was 0.5%. All the participtants in this study were shammah users. There was a significant association between of the prevalence of oral leukoplakia and the daily duration of the contact of shammah with the oral mucosa (Scheifele et al., 2007).

In another study the possible synergistic effect of qat in the development of OSCC of the floor of the mouth was reported (Kennedy et al., 1983). For another case report, it was shown that of plasma cell gingivitis can be induced by qat, where the lesion disappeared after discontinuation of qat chewing (El-Shoura et al., 1995).

#### 2.2. Characterstics of oral mucosal lesions

## 2.2. 1. Normal Oral mucosa

Oral mucosa is the lining of the oral cavity which has a variety of functions, such as protection, sensation and secretion, and histologically adapted to the unique environment inside the mouth. Oral mucosa lacks the appendages seen in skin, but sebaceous glands can be found in the upper lip and buccal mucosa. The mobile part of oral mucosa which lined the vestibule and floor of mouth joins the tightly adherent gingiva of the dental alveolus and is easily visible in normal mucosa. Gingiva appears paler pink secondary to decreased visibility of underlying blood vessels through the relatively opaque keratin layer. The gingival margin should be is usually well defined with slightly rolled margin. The interdentally papillae is pointed and the texture of the attached gingiva exhibits stippling, representing collagen fibres attaching the gingiva to the underlying periosteum (Bruch and Treister., 2009).

## 2.2.2. Definition of oral mucosal lesions

Oral mucosal lesion is defined as any change in oral mucosal surface and these changes may present as red, white, ulcerative and pigmented or as any swelling or as variants of developmental defects (Epinoza et al., 2003). The oral mucosal lesions have many causes which include infection from bacteria, viruses, fungi, parasites; other influences such as physical and thermal causes; changes in immune system; the systemic diseases; neoplasia; trauma and other factors including aging and chronic habits such as the use of tobacco and alcohol (Reichart., 2000).

#### 2.2.3. Types of oral mucosal lesions

The oral mucosal lesion can be classified into broad categories namely: oral malignant lesions, oral potentially malignant disorder and the other oral mucosal lesions which are not malignant and not potentially malignant disorders.

## 2.2.3.1. Oral malignant lesions (OML)

Malignant epithelial lesions include squamous cell carcinoma, verrucous carcinoma, basaloid squamous cell carcinoma, papillary squamous cell carcinoma, spindle cell carcinoma, acantholytic squamous cell carcinoma, adenosquamous carcinoma, carcinoma cuniculatum and lymphoepithelial carcinoma (Barnes et al., 2005).

The most prevalent of oral malignant lesions in the world is oral squamous cell carcinoma which is one of the 10 common causes of death (Baum, 2007; Bruch and Treister., 2009) :

Squamous cell carcinoma (SCC):

This lesion may appear a flat raised exophytic growing or ulcerated (showing surface erosion). The surface texture can range from smooth to irregular with induration, firmness or hardness and fixation immobility or palpable adherence to underlying structures indicating infiltration of cancer cells into deeper tissue

a. Verrucous carcinoma

It is a low-grade variant of SCC with a distinctive exophytic and papillary, or warty, appearance atypically whitish or gray color and common sites are the buccal mucosa, gingiva, and vestibule.

#### **2.2.3.2.** Oral potentially malignant disorders (OPMD)

Malignant transformations have been discussed in a World Health Organization workshop held in 2005, the potentially malignant disorders were recommended in reference to precancerous lesions as not all disorders described under this term may transform to cancerous lesions (Warnakulasuriya et al., 2007). Leukoplakia and erythroplakia are the most common ones potentially premalignant disorders. The diagnosis of these lesions with exclusion of the other red and white lesions in addition to the lichen planus seemed to be accepted in the literature as being a potentially malignant disorder. However, the risk of malignant transformation for the other red and white lesions is lower than leukoplakia (Van Der Waal., 2009).

## a. Leukoplakia

Leukoplakia was defined in 1877 by Schwimmer as a white lesion in the tongue that was probably syphilitic glossitis for a long time leukoplakia has been used to describe white plaque or patches.

WHO in 1978 defined the leukoplakia as a white patch or plaque that cannot be characterized clinically or histopathologically as any other disease which is based on the exclusion of other conditions to get the diagnosis of leukoplakia and described it as a protective reaction against a chronic irritation. In 1980 WHO described the leukoplakia as white patches which vary from quite small to an extensive lesion involving large area of oral mucosa and the surface of this lesion maybe smooth, wrinkled with shallow small crack.

From the international seminar hold in 1983 recommended that the use of the term leukoplakia should be avoided if the cause is known except in those cases where it was believed that the cause was tobacco (Axell et al., 1984). Leukoplakia was then, defined as a predominantly white lesions of oral mucosa which cannot be characterized as any other definable disease (Axell et al., 1996). Recently, Warnakulasuriya et al (2007) recommended that the term leukoplakia should be used to recognize white plaques of questionable risk having excluded other known disease or disorders that carry no increased risk for oral cancer.

# b. Erythroplakia

Erythroplakia is a fiery red patch of the oral mucosa that cannot be characterized clinically or microscopically as any other definable entity, which would exclude all the inflammatory condition which may cause red appearance of oral mucosa. Erythroplakia is precancerous lesion and some cases of erythroplakia showed different degrees of dysplasia histologically (Shafer and Waldron., 1975). The common sites in the oral cavity affected by erythroplakia are soft palate, floor of the mouth and buccal mucosa (Scully, 2004).

# c. Oral Lichen planus (LP)

The oral lichen planus (OLP) presents as reticular, erythematous and erosive lesions with distinct white mucosal changes called Wickham's striae. Women are more affected

than men, with most patients diagnosed at ages of 40-50 years old (Bruch and Treister., 2009). Lichen planus may contain both red and white appearance with different texture such as reticular, papules, plaque; bullous, erythematous and ulcerative forms (Greenberg and Glick, 2009).

The oral lichen planus affects from 1- 4% of the adult population (Bougout and Gorlin, 1986, Axell, 1987; Axell and Rundquist, 1987; Axell et al., 1990; Salonen et al., 1990; Banoczy and Rigo, 1991; Albrecht et al' 1992). Oral lichen planus may affect the middle aged, elderly and also affects the childern and young adults (Silverman and Griffith, 1972).

There are two types of OLP according to the site of the lesion namely the extra-oral and intra-oral type. Typically 90% intra oral lesion affects the posterior buccal mucosa 30% the tongue, 13% the alveolar ridge /gingiva and rarely on the lip vermillion or palate (Axell and Rundquist, 1987).

## d. Oral submucous fibrosis(OSF)

Oral submucous fibrosis has been conservatively diagnosed only on the basis of palpable fibrous bands. The palpable fibrous bands are not always present, in several instances a tough leathery mucosa with all the associated symptomatic, clinical and histopathological characteristics of OSF is seen (Pindborg et al., 1980; Seedat et al., 1988). Areca nut is the principle aetiological agent, also the gentic traits play rule in occurrence of this type of disease in some cases (Pindborg et al., 1997).

OSF can be diagnosed on the basis of the presence of one or more of the following characteristics:

1. Palpable fibrous bands

2. The mucosal texture feels tough and leathery

3. Blanching of the mucosa Blanching is further defined as a persistent, white, marblelike appearance. This blanching needs to be distinguished from the pale appearance of the mucosa due to vascular or haematological disorders, or from the loss of normal pigmentation (Zain et al., 1999).

## 2.2.3.3. Other lesions (not OML /OPMD)

Clinically the oral mucosal lesions may be seen as according to the disorder of oral mucosa to red and white and it may appear white or red appearance and white red in the same time (Greenberg and Glick, 2008) :- these lesions can be discuss as white, red, white and red, ulcerated and swelling /pigmented lesion.

# a. White lesions

#### i. Fordyce's granules

Clinical features of Fordyce's granules are yellow spots beneath the oral mucosa as a result of ectopic sebaceous glands which are more common in the buccal mucosa and also in retro molar area. The spots may be seen in the lips and in vermillion border (scully, 2004).

## ii. Lina Alba

Lina Alba is a common oral finding that appears as a raise wavy line located in the occlusal line of buccal mucosa bilaterally extends from the canine area to retromolar area which cannot be rubbed off (Langlais et al., 2009).

## iii. Leukoedema

Leukoedema is a common mucosal alteration which represents the variation of normal condition in the buccal mucosa bilaterally and it may be seen rarely on the labial mucosa, soft palate, and floor of the mouth. It usually has a faint, white, diffuse, and filmy appearance, with numerous surface folds resulting in wrinkling of the mucosa. It cannot be scraped off, and it disappears or fades upon stretching the mucosa most common in black adult (Greenberg and Glick. 2008).

## iv. White sponge naevous

White sponge nevus presents as bilateral symmetric white, soft, "spongy," or velvety thick plaques in the buccal mucosa and may be the ventral tongue, floor of the mouth, labial mucosa, soft palate, and alveolar mucosa (Greenberg and Glick, 2008).

### v. Frictional white lesion

Frictional white lesions can be caused by a variety of physical and chemical irritants such as frictional trauma, heat, prolonged aspirin contact and excessive use of mouthwash or other caustic liquids. Frictional trauma is often noted on the attached gingiva. It is cause by excessive tooth brushing, movement of oral prostheses and chewing on the edentulous ridge. With time the mucosa becomes thickened with a roughened white surface (Langlais et al., 2009).

Any friction in oral mucosa may result in hyperkeratosis that means a thickening of the keratin on the surface which has an opaque white appearance of the tissue. There are main lead to frictional keratosis is trauma and the diagnosis will be identified by know

the trauma causing the lesion and it will be recovery after elimination of the cause. (Ibsen,and Phelasn., 2009).

## b. Red lesions

# i. Erythematous candidosis (EC)

EC is present in three forms (acute EC, chronic EC and chronic nodular / hyperplasic form as (Greenberg and Glick, 2008).

# 1. Acute form of EC

This type of EC presents as red painful areas of oral mucosa sometime may be seen as circumscribed multifocal erythematous patches.

#### 2. Chronic EC

The chronic EC appears as erythematous area of mucosa with or without irregular white patches in the centre of the lesion.

## 3. Chronic nodular/hyperplasic form

This form of candidosis is presents as an erythematous area with white pinhead –sized nodules surrounded by whitish margin and cannot be rubbed off.

## ii. Median rhomboid glossitis (MRG)

This lesion appears as a red smooth and sometimes slightly elevated and lobulated of tongue mucosa anterior to the foramen caecum which mostly appears in adults. Candida albicans plays a role in its aetiology (Pinborg et al., 1997).

## **C. Ulcerated lesion**

#### (i). Aphthous ulcer

Aphthous ulcers (aphthae or canker sores) are painful solitary or multiple erosions of the oral mucous membrane. Aphthous ulcer is the most common condition of the oral mucosa in developed countries, affecting around 20% of the general population, mostly young adults. Diagnosis is based on history and examination .Recurrence of aphthous ulcerations is idiopathic in most patients. However, in a minority of patients, recurrent aphthae can be an oral manifestation of systemic diseases or vitamin deficiencies.

Minor aphthae which comprises of 80-85% of cases often cause minimal symptoms will heal spontaneously without scarring within one to two weeks and recur at intervals of one to four months. However Major aphthae <10% of cases are often more painful and usually heal within one to two months with scarring and recur frequently.

The other ulcer which are called herpiform aphthous ulcers comprise of 5% of the cases and is very painful and can be recovered from within one month (Bischoff et al., 2009).

#### (ii). Traumatic ulcer

This type ulcer may be burns from chemicals of various kinds of heat (cold, or ionizing radiation or factitious ulceration, especially of the maxillary gingivae or palate. At any age, trauma, hard foods, appliances may also cause ulceration. The lingual fraenum may be traumatized by repeated rubbing over the lower incisor teeth in cunnilingus, in recurrent coughing as in whooping cough, or in self-mutilating conditions. Most ulcers of local cause have an obvious aetiology, are acute, usually single ulcers and last less

than three weeks and heal spontaneously. Chronic trauma may produce an ulcer with a keratotic margin (Scully and Felix, 2005).

## d. Swelling and pigmented lesions

## i. Pyogenic granuloma

Pyogenic granuloma is a pedunculated hemorrhagic nodule that occurs most frequently on the gingiva and has a strong tendency to recur after simple excision. Chronic irritation is a causative factor for these lesions may sometimes be hard to identify, but the fact that they are usually located close to the gingival margin suggests that calculus (Greenberg and Click, 2008). It is rapidly growing lesion that develops as a response to local irritation, poor hygiene, overhanging dental fillings, trauma, or increased hormone levels in pregnancy (Demir et al., 2004).

## ii. Fibro epithelial polyp

Fibro epithelial polyps tend to form smooth nodules or swellings that may be soft or firm and usually covered by normal, pink mucosa unless ulcerated. The polypoid swellings may be sessile or pedunculated (Cardesa and Slootweg., 2006).

## E. Quid /Shammah/Qat related lesions (Not.ML and not PML)

## 1. Quid related lesion

Oral mucosal lesions may result in mechanical or chemical trauma of quid chewing which are categorized according to the affected area by betel quid such as overleaf.

#### i. Betel Chewer's mucosa (BCM)

Betel Chewer's mucosa is related to betel quid use and this condition is induced by either direct chemical effect of quid substance or due to action of chewing as a traumatic effect.

The clinical finding of Betel chewer's mucosa is a red-brownsih discolouration of the affected oral mucosa in the buccal mucosa where the habitual chewing of betel quid. The coloured material stems from the betel quid which is composed of calcium hydroxide and poly phenols that make the teeth black in colour due to polymerisation (Reichart et al, 1985).

Some cases of (BCM) showed desquamate or peel where loose detached white tags of tissues which can be seen and felt,wrinkled appearance of oral mucosa with evidence of incoorportion of the quid ingredients in the form of yelowish or reddishbrown peel (Gupta, 1980).

#### ii. Areca nut related lesion

The oral mucosa of areca nut chewers appears healthy mucosa from the clinical appearance with no textural and color changes.but Buccal mucosa, both sides and one side of oral cavity may show an ill-defined whitish gray discoloration that cannot be rubbed off. The mucosa also may show rough linen-like texture .

Rarely, typical localized leukoplakia, erythroplakia, erythroplakialike lesions (possibly due to chronic trauma) and frank malignancies may be seen among areca nut chewers so these lesions need to be identified from other lesions induced from other habits (Seedat ,1985).

## iii. Betel Quid lichenoid lesion

This lesion resembles oral lichen planus as a result of using the quid however, there are specific differences. It is characterized by the presence of fine, white, wavy, parallel lines that do not overlap or criss-cross, are non-elevated, and in some instances radiate from a central erythematous area. The lesion generally occurs at the site of placement of the quid. This lesion was described as a lichen planus-like lesion but it is now termed a ''betel-quid lichenoid lesion''. This lesion may regress with decrease in the frequency or duration of quid use or a change in the site of placement of the quid. There may be complete regression if the quid habit is given up (Zain et al., 1999).

## 2. Shammah related lesions

A study carried out in Saudi Arabia by Salem et al, 1984 showed the prevalence of oral lesion especially the pre malignant lesion and malignant lesion and relatively high incidence of oral cancer between shammah users and The oral leukoplakia associated with shammah chewing. The white lesion which look like oral leukoplakia caused by shammah was extended from the labial frenum in the mandible to the canine region (Zhang et al., 2001).

Cases with oral leukoplakia (OL) or mucosal burns (MB) were compared with users without any lesion. MB was detected in 31%, of which 46.8% were located on the tongue or floor of the mouth, and OL in 27%,Oral mucosal burns (MB) were defined as: Clinically; white or white-yellow lesions that could not or only partly be wiped of a history of burning sensation during 48 h before examination, and (3) an individual experience where is comparable lesions normally quickly disappeared, when *shammah* had been placed elsewhere or the use had been temporarily stopped (Scheifele et al., 2007).

#### 3. Qat related lesions

Keratotic white lesions associated with qat chewing reported by Airman et al. (2004) as a result of the mechanical friction during chewing, the chemical constituents or additives to qat or both of these mechanisms vary in their clinical features and graded as the following:

Grade I: mild whitening at the site of qat chewing that is similar to leukoedema as defined by (Lynch et al., 2003).

Grade III: a very clear oral keratotic white lesion at the site of chewing that is similar to homogenous leukoplakia as defined in Malmo<sup>...</sup> International Seminar for oral white lesions (Axell et al .,1984).

Grade II: oral white lesions at the site of chewing which are defined more than grade I and less than grade III.

White lesions on the oral mucosa were most common on the lower buccal attached gingival mucosa, the alveolar mucosa and the lower mucobuccal fold at the and non-homogenous were noted at the chewing site of qat chewers (Gorsky et.al.,2004).

#### 2.3. Risk habits

#### 2.3. 1.Tobacco smoking

# 2.3. 1.1. Types of tobacco smoking

Tobacco smoking is the most popular smoking particularly in the developed countries; this habit is increasing rapidly in the developing countries. The cigarette consists of very small pieces of tobacco wrapped in paper with different grades or blends of tobacco. There are many brands of cigarettes with many changes in cigarette design during the last decades due to a demand of cigarettes in many countries (WHO, 1985).

Cigarettes are shreds of tobacco wrapped in paper as compared to cigars, where the shredded tobacco is wrapped in tobacco leaf also, there are variation of cigars and cigarettes which exist such as; bidis (tobacco hand –rolled in dried leaf of various plants) chuttas (small cigars smoked with the burning end held in the mouth) all of these often have high nictotine and tar substance. Other form of tobacco smoking is pipe smoking (Kupper et al., 2002).

The smoking of tobacco in different forms such as cigars or cheroots, loose tobacco in pipe and loose tobacco rolled into hand-made cigarettes is familiar in many countries.

There is wide Variation in the tar, nicotine and nitrosamine contents according to spices, curing, additives and the way of combustion (Jonshon, 2001).

The smoking device (cigarettes, cigars. pipe, etc) determine the intensity of exposure to tobacco in addition to the method, may be determined by the depth inhalation.

Filtered cigarettes have low risk for the most tobacco-related oral lesions than unfiltered and high tar cigarettes (WHO, 2003).

#### **2.3. 1.2.** Prevalence of tobacco smoking and relation to oral disease

Globally, there are currently 1.3 billon smokers with 900 of smokers in the developing countries as reported by the World Health Organization (WHO) and Federation Dental international (FDI). The prevalence of smoking in the world is 29% (57% of males and 10.3% of females) from the age above 15 years old of age (WHO, 2004b). In India the prevalence of smoking tobacco was 16.2% and was high among men (Neufeld et al., 2005).

In Malaysia as reported by Abu Bakar (2006) the smokers over 15 years old are five million. According to the second national health morbidity survey which reported that one in every four Malaysians was smokers. Smoking habits indicated 27.9% in Malays, 19.2% in Chinese and 16.2% in Indians (Haniza et al., 1999).

Yemen ranks the second country from the Arabic countries in the number of smokers after Tunisia. Yemeni smoke 604 billion cigarettes per year according to a study conducted by World Health Organization (Alaya'a., 2009)

There are 11 compounds find in cigarettes such as 2-napthylamine, vinyl chloride, arsenic and chromium are group1human carcinogenic (WHO, 2004(b)).

Among smokers were found benign oral mucosa alterations in the palate, tongue and any part of oral cavity such as:

a- Nicotine stomatitis

This oral mucosal lesion appears in those who smoke pipe and cigar due to the trauma of heat or chemical irritants in tobacco. Clinically presents as multiple discrete keratotic papules with depressed red centres which represent dilute and inflammation of minor salivary glands (Langlais et al., 1998).

b- Reverse smoking and palatal mucosal change

The reverse smoking is a habitual practice in many parts of the world such as India and Philippines in this habit the lit end is held inside the mouth, this habit leads to palatal change including leukoplakia, fissuring, mucosal thickening, pigmentation, erythema and ulceration (Silverman and Shillitoe, 1990).

c. Hairy tongue

This lesion present as hyper trophy of the filiform papilla of dorsal surface of tongue produce hairy like appearance(hairy tongue, black hairy tongue )these lesions have been associated with heavy smokers (Regezi et al., 2008).

## 2.3.2. Quid chewing

## 2.3.2.1. Types of quids

Quid is defined as a substance or mixture of components placed in the oral cavity and chewed thus, remaining in contact to oral mucosa which varies in composition. Usually composing from one or both of two substances tobacco or areca nut in raw form or any manufactured or processed form (proceeding workshop, 1997). The composition of quid mixture can be divided into three categories namely: areca nut quid (quid without tobacco), tobacco quid (quid without areca nut) quid with areca nut and tobacco called areca nut quid (Zain et al.1999) the further termed "betel quid that means tobacco quid included betel leaf therefore any form of quid mixture when using betel leaf should be "betel quid'. This habit comes as tobacco chewing (tobacco quid) and snuff (ground or powdered tobacco, either moist or dry which inhaled or placed in the oral cavity. Tobacco quid chewing habit is common in South and South East Asia where the tobacco is usually chewed together with another substance such as areca nut, betel leaf, ash, lime and cotton or sesame oil as termed 'betel–quid. The average of consumption of betel quid in these regions is 10-15 g/day in regular users and kept in oral cavity in contact with oral mucosa for several hours per day (Kupper et al., 2002).

Betel quid chewing is a practice in many countries in Asia like India, Thailand, Sri Lanka, Malaysia, Myanmar, Taiwan and China .betel quid is a combination of betel leaf, areca nut and slaked lime (Gupta et al., 2004).

The dried ripe areca nut with slaked lime is used In India, Sir Lanka and Malaysia (Reichert, 2006). The Taiwanese quid chewers used unripe areca nut with slaked lime and betel inflorescence which is wrapped in betel without tobacco (WHO, 2004 (a)).

In Thailand, Cambodia and Myanmar the quid contains cloves, cinnamon and roots of certain local plant to their quid however, the Cambodian betel quid includes tobacco where it used to rub their gum after chewing (Gupta et al., 2004).

There are various types of tobacco quid in many forms which can be chewed, sucked or applied to the teeth and gum in India (Gupta and Ray, 2003).

There is another form of tobacco quid snuff type called *shammah* which is a native name for a mixture of powdered tobacco leaves, carbonate, lime, ash and other substance (Yousif and Hashash.., 1983).

Some species of *shammah* including black pepper and flavouring agents the different of additives result in characteristics of colour and /or different brands of *shammah* which is greenish yellow powder or paste that is placed in the lower buccal sulcus or sometimes in upper labial vestibule. *Shammah* is practiced in Yemen, south Saudi Arabia, Algeria (Salem et.al.1984; Stirling et al., 1981; Amer et.al., 1985; El-Alkkad et al., 1986).

## 2.3.2.2. Prevalence of quid chewing

Around 600 million betel chewers in the world with commonly practice quid chewing in Asia –pacific region (Gupta et al., 2002). In Northern Mariana Island were found 64.3% betel quid chewers from 309 school children (Oakley et al., 2005).

According to population based survey carried out in India, Nepal and Pakistan by Gupta et al (2004). It was found that 20%-40% of age people 15years old were quid chewers. The World Health Organization in 2004 reported that many people in the Asian regions chewed areca nut with a higher percentage among women who also added tobacco to the quid.

In Taiwan 2million from 20 million were betel chewers or ex-chewers (Lin et al., 2006).

In Malaysia betel chewing is a dying habit particularly in the younger generation and urban communities, and this habit is widely practiced among the Indian and Malay communities (Reichart., 2006). A nationwide survey conducted in 1993/1994 reported the overall betel quid prevalence as 6.9%, the betel quid habit was found more among Indians, Malays and other Bumiputras (Zain et al., 1997).

## 2.3.3.Al cohol drinking habit

#### 2.3.3.1Type of alcohol beverages

Alcholol beverages containing alcohol (common name for ethanol ) and can be termed as beers (typically containing 5% of alchohol), wine (containing 12% of alchohol) spirits (40% alcohol),other less common beverages include cider, fortifed wine and flavoured wine which are limited in particular regions. The distribution between each type of beverage is different from region to region where there is decrease in alcohol consumption in developed countries and a corresponding increase in cosumption in less developed countries.(WHO, 2003).

On global scale the consumption of alcohol beverage by an adult is 9g/per day against roughly 3% of calories (WHO, 1999).

In Malaysia beer and stout are the most common types of alcohol drinking habit by many ethnic groups. However, homemade are also widely used in some ethnic groups such as rice alcohol 'tuak which is popular in the indigenous people of Sabah and Sarawak (WHO, 2004) but " toddy" (alcohol that is tapped from conconut palm with varying degrees of fermentation fall within proof spirit range from 3.8%-15.1%)among the Indians, another type is consumed called "samsu "(locally brewed Chinese alcohol that can reach up 169.1% proof spirit (Ramanathan et al., 1976).

#### 2.3.3.2. Prevalence of alcohol drinking and relation to oral disease

The United Nation Food & Agriculture Organization (FAO) reported that Thailand ranked fifth worldwide in alcohol consumption with 15.3 million drinkers in 2001. The prevalence of alcohol consumption among the Korean population was 51.5% for moderate, 12.5% for excessive and 8.0% for heavy consumers. The prevalence of alcohol consumption in Malaysia was 4.2% with high prevalence among Indians (13%) followed by indigenous Sabah and Sarawak (10%) followed by Chinese (7.8%) (Zain et al., 1995). In 1999 the World Health Organization reported that the recorded adult per capita consumption for Malaysia was 1.06 litres of pure alcohol.

Approximately 75% of all cancer arise in association with alcohol and tobacco use (La vecchie et al .,2004 ; Llewellyn et al .,2004). For almost half a century alcohol has been recognized as an important risk factor for oral cancer (Wynder and Bross .1957).

Oral cancer rate in United Kingdom was more than double in 20 years where 7% of population are dependent alcohol with an increase of oral cancer incidence Europe and United States (la vecchia et al., 2004 Schantz and Yu, 2002).

There was strong evidence that high alcohol intake is related to carcinogenesis especially cancer of oral cavity pharynx, larynx and liver (Gerhauser, 2005). Alcohol beverage is causally related to cancers of oral cavity and other parts of the human body (Baan et al., 2007).

#### 2.3.4. Qat chewing

Qat is a green-leaved plant that has been chewed for its stimulant effect for centuries the most active ingredients of qat are alkaloids such as cathinone and cathine. Cathinone is the main psychoactive constituent of qat, and has a similar action to amphetamine, inducing the release of dopamine, a neurotransmitter, from pre-synaptic storage (Kalix,

1992; Patel, 2000). This type of plant is known by different names in different countries: chat in Ethiopia, qat in Yemen, mirra in Kenya and qaad or jaad in Somalia, but in most of the literature it is known as qat. In qat-growing countries, the chewing of qat leaves for social and psychological reasons has been practised for many centuries. Its use has gradually expanded to neighbouring countries and beyond through commercial routes, recently, increasing numbers of immigrants have spread the practice to Europe and the United States( Nencini et al., 1988)

The leave of this plant elevate and produce stimulant effect unlike the chewing of tobacco. The first qat was found in 1237 and the production of this plant is more especially in Yemen. The other countries producing this type of plant is Ethiopia and Kenya (McKee, 1987). This is a destructive habit and has effects similar to those of amphetamines with mild euphoria, energy. The active substance in the fresh qat leave is cathinone which causes sympato-mimetic effects and induces symptoms such as euphoria and hyperactivity. Cathinone has analogous mechanisms of action with pharmacological properties (Valterio & Kalix , 1982). Purified cathinone is a Class C drug, and thus controlled by the Misuse of Drugs Act 1971, but when present in the form of qat, it has no legal implications in the UK, whereas certain European countries and the United States consider qat to be a controlled substance (El-Wajeh, Thornhill., 2009).

#### **2.3.4.1.** Prevalence qat chewing and relation to oral disease

In study carried out in 2500 Yemeni subjects showed 1528 of them (61.12%) were qat chewers; 342 cases (22.4%) had oral keratotic white lesions at the site of qat chewing, while only 6 (0.6%) non-chewer cases had white lesions in their oral caviy and the

relation between qat chewer and oral white lesion was significant (p-value=0.00) (Aiman et al., 2004).

In other study done by Gorsky et.al (2004) which conducted on 1500 Yemenite Israeli Jews qat chewers. This study presented white lesions on the oral mucosa which was most common on the lower buccal attached gingival mucosa, the alveolar mucosa and the lower mucobuccal fold at the second premolar and molar areas. White lesions were identified in 39 subjects (83%) of the Qat chewers compared to only 9 individuals (16.3%) of the control group (p < 0.001). White lesions were identified in 48 individuals, and in 41 (85.4%) subjects were completely homogenous. Five of the seven non-homogenous lesions (71.4%) in qat chewers. These findings indicate an approximately threefold higher risk of developing non-homogenous white changes in Qat chewers compared to non-chewers. A significantly higher occurrence of white lesions was seen on the chewing side (37 subjects (100%) versus 3 lesions (7.7%) on the non-chewing side. Although 3 subjects (8.1%), were smokers, had white lesions on the non-chewing side. White lesions were noted at the chewing site of all chewers. Two patients chewed on both sides, and white lesions were identified in these patients on both sides of their oral cavities.

From study in Kenya carried out by Fasanmade et al (2007) was found saquamous cell carcinoma in A 42-year-old African woman who chewed qat for along time and preferred placing chewed residues under the tongue on the same side as the subsequent lesion.

Qat chewing is a widely practised in Southern Arabia and Eastern Africa as sociocultural habit (El-Wajeh1 and Thornhill., 2009). Adverse effects of qat chewing have been studied by (Halbach,1972; Luqman and Danowski,1976), and provied that chronic qat chewing caused stomatitis followed by secondary infections. These effects were due to the mechanical action on the oral tissues in additional to the chemical irritation on the oral mucosal surfaces.

Other study showed the occurance of oral cavity tumor among the qat chewers (Kennedy et al., 1983). A study carried out Kenyain population indicated the association between oral leukoplakia and cigarettes smoking, alcohol consumption and qat chewing. There is no significant association between the qat chewing and leukoplakia compared to tobacco and alcohol consumption (Macigo et al., 1995).

**Chapter Three: Purpose of Study** 

## **3.1.** Rationale of this study

The prevalence of oral mucosal lesions and related risk habits differs from region to region in the world. The prevalence of oral mucosal lesions in Malaysia and Yemen has been reported. However, there are few data on the prevalence of oral mucosal lesions in Yemen and data on association of oral mucosal lesions and related risk habits in Yemen is also still not well established. Thus, the need to compare the prevalence data and their related habits of the Yemen population with a well established oral mucosal lesions prevalence and related risk habits of the Malaysian population.

### 3.2. Aim of study

To determine the prevalence of oral mucosal lesions and their related risk habits (smoking, quid chewing, alcohol consumption, and qat chewing) in out-patients of two dental clinics in Malaysia and Yemen. It is also the aim of this study to investigate the influence of these risk habits on the occurrence of oral mucosal lesions.

## **3.3. Specific objectives**

1- To determine and compare the prevalence of oral mucosal lesions in outpatients attending dental clinics in Malaysia and Yemen.

2- To determine and compare the prevalence of risk habits (smoking, quid/qat chewing and alcohol consumption) in outpatients attending dental clinics in Malaysia and Yemen.

3- To determine the relationship between risk habits and the prevalence of oral mucosal lesions in both countries.

**Chapter Four: Methodology** 

# 4.1 .Study design and study population

This is a cross-sectional study conducted from May 2009 to October 2009. It was carried out on adult outpatients aged 18 years and above who were attending dental clinics at the Faculty of Dentistry, University of Malaya (UM) in Kuala Lumpur, Malaysia and the Al-Thawra Modern General Hospital, Sana'a in Yemen.

# 4.2. Sample size estimation

This study was a comparative study between Malaysia and Yemen. Using the PS -Power and Sample Size Calculation Software (Dupont and Plummer, 1998), the sample size was then estimated for each objective of the study. The highest number of sample was yielded for the objective to compare the prevalence of oral mucosal lesion between Malaysia and Yemen, thus the number was used as estimation of sample size needed in this study. It would be able to detect a difference of 8% in the prevalence of oral mucosa lesion between these 2 countries if the different exists. Other parameters used are as below:

Level of significance ( $\alpha$ ) = 5 %.

Power of study = 95 %.

Prevalence of oral white lesions in Malaysia = 14 %. (Axell et al., 1990) Prevalence of white lesion in Yemen = 22.4 %. (Aiman et al., 2004)) Ratio = 1:1

Sample size = 546 for each group.

# 4.3. Inclusion and exclusion criteria

#### **Inclusion criteria**

New patients seeking dental treatment at the Faculty of Dentistry, University of Malaya in Kuala Lumpur, Malaysia from May to July 2009 and Althawra Modern General Hospital in Sana'a, Yemen from August to October 2009 were included in this study.

# **Exclusion criteria**

Patients who have had treatment for oral mucosal lesions were excluded from the study.

# 4.4 Training and calibration

The author of this thesis was trained by Professor Dr. Rosnah Binti Zain (RB Zain) who has been the trainer and consultant for oral cancer screening programme for the Oral Health Division, Ministry of Health, Malaysia since 1993. The sequences of the process of training and calibration were as follows:

Pictorial manual of oral mucosal lesion (Zain et al., 2002) was presented to the trainee prior to the training. The pictorial manual included written clinical criteria for oral mucosal lesions.

- A series of lectures were given on the definition, aetiology and clinical appearance of oral cancer and potentially malignant disorders; biological aspects of oral cancer; oral cancer related risk habits; criteria and differential diagnoses of oral mucosal lesions.
- The trainee was subjected to lesion identification/recognition via 3 spot diagnoses sessions using digital images where the final percentage of accuracy of lesion diagnoses achieved by the trainee against the trainer was 100% pre-survey, 95.2% post-survey.

- 3. The trainee was also given a clinical hands-on demonstration/examination of 10 patients having oral cancer, potentially malignant disorder and no oral lesions. This clinical hands-on demonstration/examination was conducted at the Hospital Tengku Ampuan Rahimah (HTAR), Ministry of Health Malaysia (MOH) in the district of Klang, Selangor.
- 4. The intra-examination accuracy of diagnosing a lesion was 81.0%.
- 5. The post-survey sensitivity of trainee vs trainer was 95.2% and the specificity was 81.2%

# 4.5. Conduct of study

# 4.5.1. Ethical approval and permission

Ethical approval to conduct the study was obtained from the Medical Ethics Committee, Faculty of Dentistry, University of Malaya and the administration of Al-Thawra Modern General Hospital in Sana'a, Yemen.

# 4.5.2. Structured questionnaire

The questionnaire in this study was prepared to obtain information on sociodemographic characteristics of the participants (age, gender, and ethnicity) and questions on oral risk habits commonly practiced in Malaysia and Yemen (smoking, quid chewing, consumption of alcoholic beverages and qat chewing).

With regards to Yemen, questions on alcohol consumed were excluded because the populations of Yemen are Muslims where the Islamic religion prohibited the consumption of alcohol beverages. The qat chewing habit which is a common practice in Yemen was included in the Yemen questionnaire.

# 4.5.2.1. Validation of the questionnaire

The questionnaire was translated from English into two languages (Bahasa Melayu and Arabic language). These were then back-translated to English from the two languages (retranslated back to original language) to detect any anomalies in the first translation.

# 4.5.2.2. Pre-testing the questionnaire (Appendix 9-12).

A pre-test of the questionnaire was conducted prior to actual data collection .It was conducted on 20 participants who are outpatients in the waiting area of the registration counter of the Faculty of Dentistry, UM, including some general staff of the faculty.

The questionnaire was pretested for ambiguity, clarity, sequencing and understanding of the instructional questions. The pre-test interviews were carried out on over 2 days. The time required for each participant to be interviewed was 3-5 minutes.

# 4.5.3 Data collection

# 4.5.3.1 Interview questionnaire

Informed consent was taken from the patients prior to the interview and mouth examination. The participants were subjected to interviews in the waiting area of the dental clinics.

# 4.5. 3.2. Identification of oral mucosal lesions

# Criteria of oral mucosal lesion

The clinical criteria for oral mucosal lesions are as in table 4.1 which was based on Zain et al (2002).For cases not in Zain et al (2002). The criteria was based on Seedat et al (1985) for areca quid, Aiman et al (2004) for qat related lesions, Scheifele et al (2007) for *shammah* related lesions, Bruch and Triester (2009) for hairy tongue, Laskaris (2006) for hyperplastic gingivitis,Field and longman (2003) for coated tongue.

# 4.5.3.3. Clinical examination and recording the oral mucosal lesions

The mouth examination was done systemically using a dental mirror with the participant seated in the dental chair. The mouth examination was carried out under standard dental illumination. The systematic mouth examination procedures were as shown in Zain et al (2002).

All oral mucosal lesions were recorded in the clinical case sheet which contained a topographic mouth map to register the location of oral mucosal lesions in the oral cavity.

# 4.6 Data Entry and Statistical Analysis of Data

All the data collected from this study was entered into the SPSS (statistical software) version 17.0. The chi-square statistical test was used to compare the prevalence of oral mucosal lesions and related risk habits between Malaysian and Yemeni dental outpatients. The same statistical test was used to evaluate the relationship between oral risk habits and oral mucosal lesions. The alpha value was set at p=0.05.

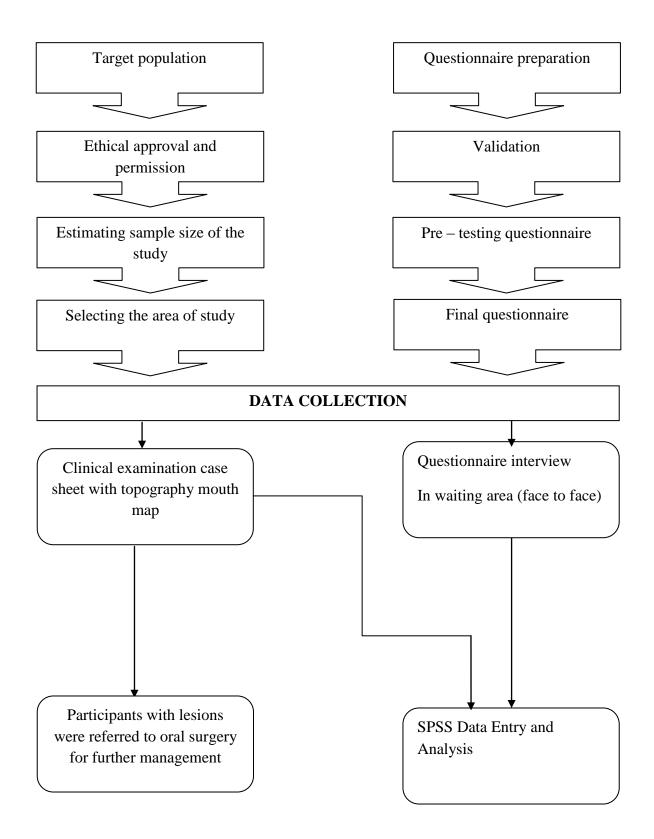


Fig.4.1. Flow Chart showing the methodology of study

Chapter five: Result

# 5.1. Sociodemographic characteristics of the study population

At the dental clinics, the Faculty of Dentistry University of Malaya (UM) Kuala Lumpur (KL); Malaysia and the Al-Thawra Modern General Hospital, Sana'a (SAH); Yemen, a total of 554 and 520 out-patients were interviewed and the oral mucosa examined respectively during the period from May to October 2009. The mean age of the Malaysian dental outpatients was  $41.97\pm17.04$  years with an age range of 18 - 89 years. The mean age of the Yemeni dental outpatients was  $36\pm15.62$  years old with an age range of 18-95 years.

Table 5.1 shows the age and gender distribution of the Malaysian and Yemeni populations. There were 43.9 % (n=243) males and 56.1 % (n=311) females in Malaysia while in the Yemen sample, the male population was 45.0 % (n=234) and females was 55.0 % (n=286).

<b>A</b> 50	Malaysian dental outpatients			Yemeni dental outpatients				
Age Group	Male n=243 (%)	Female n=311 (%)	Total n=554 (%)	Male n=234 (%)	Female n=286 (%)	Total n=520 (%)		
18-34	86 (15.5)	150 (27.1)	236 (42.6)	105 ( 20.2)	173 (33.3)	278 (53.5)		
35-54	81(14.6)	93 (16.8)	174 (31.4)	73 (14.0)	86 (16.5)	159(30.6)		
≥55	76 (13.7)	68 (12.3)	144 (26.0)	56 (10.8)	27 (5.2)	83(16.0)		
Total	243(43.9)	311 (56.1)	554 (100.0)	234(45.0)	286 (55.0)	520(100.0)		

 Table 5.1: Distribution of age and gender in Malaysians and Yemeni sample population (N=1074)

Majority of the outpatients from both countries were from the 18-34 years age groups with 42.6 % Malaysians and 53.5% for Yemenis.

Table 5.2 showed the gender and ethnic distribution of the Malaysian and Yemeni population. There are 4 major groups for the Malaysian sample population consisting of 34.5% (n=191) Malays, 42.6% (n=236) Chinese, 20.0% (n=111) Indians and 2.9% (n=16) others. The majority of the Yemen sample populations were Yemenis comprising of 97.9% and the other group comprising of 2.1%.

 Table 5.2: Distribution of Malaysian and Yemeni dental outpatients according to gender and ethnicity (N=1074)

	Ma	alaysia					Yemen	
	Malays	Chinese	Indian	Others	Total	Yemeni	Others	Total
gender	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Male	79 (14.3)	93 (16.8)	66 (11.9)	5 (0.9)	243 (43.9)	226 (43.5)	8 (1.5)	234 (45.0)
Female	112 (20.2)	143 (25.8)	45 (8.1)	111 (2.0)	311 (56.1)	283 (54.4)	3 (0.6)	286 (55.0)
Total	191 (34.5)	236 (42.6)	111 (20.0)	16 (2.9)	554 (100)	509 (97.9)	11 (2.2)	520 (100)

# 5.2. The prevalence of oral mucosal lesions in Malaysian and Yemeni dental outpatients

The prevalence of oral mucosal lesions in the Malaysian and Yemeni population was 23.3% (n=129) and 22.3% (n=116) respectively (Table 5.3).

Table 5.3 showed the similarity of the oral mucosal lesions prevalence between Malaysian and Yemeni dental outpatients and there is no significant difference between the prevalence of both countries (p=0.703).

Table 5.3: Prevalence of oral mucosal lesions in Malaysian and Yemeni dental outpatients (N=1074)

Ethnic group	Lesion	no lesion	<i>p</i> -value*
	n (%)	n (%)	_
Malaysian (n=554) Yemeni (n=520)	129 (23.3) 116 (22.3)	425 (76.3) 404 (77.7)	0.703

\* Chi square test was used.

Level of significant was set at 0.05.

Table 5.4 showed that among those aged 18-34 years old there were more Yemeni with oral mucosal lesions as compared to Malaysians. This relationship is statistically significant (p=0.049). However, there were no statistically significant difference between the oral mucosal lesions prevalence of Malaysian and Yemenis aged 35-54 years and those aged  $\geq$  55 years (p=0.173 and p=0.950 respectively).

Age group	Lesion	No. lesion	<i>p</i> -value *
	n (%)	n (%)	
Age group: 18-34 years:			
Malaysian (n=236)	29 (12.3)	207 (87.7)	0.040
Yemeni (n=279)	52 (18.6)	227 (81.4)	0.049
Age group: 35-54 years:			
Malaysian (n=174)	52 (29.9)	122 (70.1)	0 172
Yemeni (n=159)	37 (23.3)	122 (76.7)	0.173
Age group - $\geq$ 55 years			
Malaysian (n=144)	48 (33.3)	96 (66.7)	0.950
Yemeni (n=82)	27 (32.9)	55 (67.1)	0.750

 Table 5.4: Comparison of Prevalence of oral mucosal lesion in Malaysian and

 Yemeni outpatient according to age

\* Chi square test was used.

Level of significant was set at 0.05.

Table 5.5 showed that among the gender there were more Malaysian males with oral mucosal lesions as compared to Yemeni males while there were more Yemeni females with oral mucosal lesions compared with Malaysian females. However, there is no statistically significant difference between the Malaysian and Yemeni males and females in prevalence of oral mucosal lesions.

Table 5.5: Comparison	of Prevalence	of oral	mucosal	lesion	in	Malaysian	and
Yemeni outpatient acco	rding to gender						

Gender	Lesion	No. lesion	<i>p</i> -value *	
	n (%)	n (%)		
Male				
Malaysian	84 (34.3)	161 (65.7)	0.001	
Yemeni	63 (26.9)	171 (73.1)	0.081	
Females				
Malaysian	45 (14.6)	264 (85.4)	0.102	
Yemeni	53 (18.5)	233 (81.5)	0.192	

\*chi square test was used.

Level of significant was set at 0.05.

Table 5.6 showed the distribution of oral mucosal lesions in Malaysian and Yemenis dental outpatients. From the Malaysian dental outpatients sample, only one subject (2%) had oral cancer which was in an 86 years old Indian lady (Fig. 5.1) while nine Yemeni subjects (1.7%) had oral cancer with all aged above 65 years where 6 cases were in males and 3 cases were females (Fig. 5.2). The prevalence of potentially malignant disorders (i.e. leukoplakia, and lichen planus) in the Malaysian outpatients was found to be 0.8% (n= 4) as compared to 0.2% (n=1) in the Yemeni dental outpatients. Biopsies for all the oral cancers confirmed to be oral squamous cell carcinoma.

The highest prevalence of oral mucosal lesions among Malaysian dental outpatients was cheek biting (7.2%) while among the Yemenis; the highest prevalence was frictional lesions (5.58%) (Fig.5.30). Among Malaysian patients, the prevalence of Fordyce's spots was 3.4% while this condition was not identified in the Yemeni patients. The prevalence of traumatic ulcers was similar in both countries (1.4% among Yemenis and 1.3% among Malaysians).

			Mal	aysian				<u> </u>	Ye	<u>meni</u>		
Oral mucosal lesions	-	ales		nales	Tota			ales		nales		otal
	(n=	=243)	(n=	=311)	( n=	554)	(n=2	234)	(n=	=286)	(n=520)	
	n	%	n	%	n	%	n	%	n	%	n	%
1. <u>Malignant lesions(ML</u> )												
Oral cancer	0	0.0	1	0.32	1	0.2	6	2.6	3	1.1	9	1.7
2. Potentially malignant disor	ders(	(PMD)										
Leukoplakia	2	0.8	0	0.0	2	0.4	1	0.4	0	0.0	1	0.2
Lichen planus	0	0.0	2	0.6	2	0.4	0	0.0	0	0.0	0	0.0
3. Other lesions (Non-ML & N	lon-P	<u>'MD)</u>										
white lesions												
Cheek biting	23	9.5	17	5.5	40	7.2	11	4.7	13	4.6	24	4.6
Frictional lesions	13	5.4	5	1.6	18	3.2	16	6.8	13	4.6	27	5.
Fordyce's spots	17	7.0	2	0.6	19	3.4	0	0.0	0	0.0	0	0.0
Geographic tongue	2	0.8	1	0.3	3	0.5	2	0.9	3	1.1	5	1.
Lina alba	2	0.8	3	1.0	5	0.9	1	0.4	0	0.0	1	0.2
Leukoedema	0	0.0	0	0.0	0	0.0	1	0.4	0	0.0	1	0.
Coated tongue	0	0.0	0	0.0	0	0.0	5	2.1	4	1.4	9	1.
Red lesions												
Acute erythematous	2	0.8	2	0.6	4	0.7	4	1.7	5	1.8	9	1.7
candidiasis												
Denture stomatitis	5	2.0	6	1.9	11	2.0	0	0.0	0	0.0	0	0.0
Hyperplastic gingivitis	1	0.4	0	0.0	1	0.2	1	0.4	1	0.3	2	0.4
Ulcerated lesions												
Angular cheilitis	0	0.0	1	0.3	1	0.2	0	0.0	0	0.0	0	0.0
Minor aphthous ulcer	2	0.8	6	1.9	8	1.4	2	0.9	1	0.3	3	0.6
Traumatic ulcer	5	2.1	2	0.6	7	1.3	3	1.3	4	1.4	7	1.4
Quid related lesion												
Čhewer's mucosa	0	0.0	2	0.6	2	0.4	0	0.0	0	0.0	0	0.0
shammah related lesion	0	0.0	0	0.0	0	0.0	1	0.4	0	0.0	1	0.2
Pigmented / swelling lesion												
Pyogenic granuloma	1	0.4	0	0.0	1	0.2	0	0.0	1	0.3	1	0.2
Excessive melanin	0	0.0	0	0.0	0	0.0	0	0.0	1	0.3	1	0.2
pigmentation												
Fibroepithelial polyp	2	0.8	1	0.3	3	0.5	0	0.0	3	1.1	3	0.6
Pericoronitis	1	0.4	0	0.0	1	0.2	0	0.0	5	1.8	5	1.0
		0.0		0.0		0.0	3			0.3		0.8

# Table 5.6: Prevalence of oral mucosal lesions in outpatients from 2 dental clinics in Malaysia and Yemen



**Fig.5.1** Oral carcinoma in the mouth of an 86 years old Indian lady who chewed quid without tobacco for long time.



(a)



(c)



(b)



Fig 5.2: (a), (b), (c), (d) Oral carcinomas in Yemeni patients who had shammah (tobacco quid) chewing habits





Fig.5.3. (a)

Fig. 5.3. (b)

Fig.5.3. (a) The frictional lesion on left side of qat chewers (qat related lesion) (b) shammah related lesions in the ventral surface of the tongue and the floor of the mouth due to shammah being in contact with this area.

# 5.3. The prevalence of risk habits in outpatients attending dental clinics in

# Malaysia and Yemen.

Table 5.7 showed that the most common habit practised by Malaysian dental outpatients was smoking. Qat chewing was the most common habit among Yemeni dental outpatients.

# Table 5.7: Distribution of risk habits in Malaysian and Yemeni dental outpatients (N=1074)

Malaysian study sample					
Risk habits	No of subjects	Percent (%)			
1-Main habits					
Smoking	108	19.5			
Quid chewing	10	1.8			
Alcohol drinking	24	4.3			
2.Combination Habit					
Smoking with quid chewing	4	0.7			
Smoking with alcohol drinking	15	2.8			
Quid chewing and alcohol drinking	2	0.4			
All three habits	1	0.2			
Total no. of subjects with risk habits	123	22.2			
Total no. of subjects without risk habits	431	77.8			
Total subjects examined	554	100			
Yemen stu	ıdy sample				
1.Main habits					
Smoking	99	19			
Shammah chewing	23	4.4			
Qat chewing	212	40.8			
2.Combination Habit					
Smoking with shammah chewing	9	1.7			
Smoking with Qat chewing	89	17.1			
Shammah chewing and Qat chewing	17	3.3			
All three habits	9	1.7			
Total no of subjects with risk habits	268	51.4			
Total no subjects without risk habits	252	48.6			
Total subjects examined	520	100			

There is difference of prevalence of risk habits in Malaysian and Yemeni dental outpatients the relationship was statistically significant with p < 0.001 (Table 5.8).

 Table 5.8: Prevalence of all risk habits among Malaysian and Yemeni dental outpatients (N=1074)

Population	Habit	No habit	
	n (%)	n (%)	<i>p</i> -value*
Malaysian (n=554)	123 (22.2)	431 (77.8)	< 0.001
Yemeni (n=520)	268 (51.4)	252(48.6)	< 0.001

\* Chi square test was used.

Level of significant was set at 0.05.

Table 5.9 showed that for all age groups, there were more Yemeni dental outpatients with risk habit as compared to the Malaysian dental outpatients. These relationships were statistically significant (p < 0.001 in all age groups).

Age group	habit	No habit	P-value *	
	n (%)	n (%)		
Age group: 18-34 yea	ars:			
Malaysian	45 (19.1)	191 (80.9)	< 0.001	
Yemeni	136 (48.7)	143 (51.3)	< 0.001	
Age group: 35-54 yea	ars:			
Malaysian	38 (21.9)	136 (78.2)	.0.001	
Yemeni	80 (50.3)	79 (49.7)	< 0.001	
Age group $- \ge 55$ yea	rs			
Malaysian	40 (27.8)	104 (72.2)	0.001	
Yemeni	52 (40.7)	30 (36.6)	< 0.001	

Table 5.9: Prevalence of risk habits in Malaysian and Yemeni outpatient according to age (N=1074)

\* Chi square test was used.

Level of significant was set at 0.05.

Table 5.10 showed that; there were more risk habits among Yemeni males and females as compared to Malaysian males and females. These relationships were statistically significant.

Age group	habit	No habit	<i>P</i> -value *	
	n (%)	n (%)		
Males				
Malaysian (n=245)	105 (42.9)	140 (57.1)	< 0.001	
Yemeni (n=234)	140 (59.8)	94 (40.2)	< 0.001	
Females				
Malaysian (n=309)	18 (5.8)	291 (94.2)	.0.001	
Yemeni (n=286)	128 (44.8)	158 (55.2)	< 0.001	

Table 5.10: Prevalence of risk habits in Malaysian and Yemeni outpatient according to gender (N=1074)

\* Chi square test was used.

Level of significant was set at 0.05.

# 5.3.1. Smoking habits

Table 5.11.showed that the prevalence of Malaysian and Yemeni smokers was similar and the relationship was not statistically significant (p = 0.850).

Table 5.11: Prevalence of smoking habits in Malaysian and Yemeni dental outpatients (N=1074).

Population group	Smoking	No smoking	<i>p</i> -value*		
	n (%)	n (%)			
Malaysian (n=554)	108 (19.5)	446 (80.5)	0.850		
Yemeni (n=520)	99 (19.0)	421(81.0)			

\* Chi square test was used.

Level of significant was set at 0.05.

Most of smoking habit was found among Malaysian males in the 18-34 years age group while in the Yemeni outpatients. There were equally high numbers of the males with smoking habit in 18-34 and 35-54 age groups (Table.5.12).

Age		Malaysian dental outpatients			Yemeni dental outpatients							
Group	N	Iale	]	Female	T	otal		Male	F	emale	]	<b>fotal</b>
18-34	36	(33.3)	4	(3.7)	40	(37.0)	28	(28.3)	8	(8.1)	36	(36.4)
35-54	30	(27.8)	5	(4.6)	35	(32.4)	28	(28.3)	10	(10.1)	38	(38.4)
≥55	31	(28.7)	2	(1.9)	33	(30.6)	22	(22.2)	3	(3.0)	25	(25.3)
Total	97	(89.8)	11	(10.2)	108	(100.0)	78	(78.8)	21	(21.2)	99	(100.0)

 Table 5.12 Distribution of smoking habit according to age and gender in Malaysian

 and Yemeni dental outpatients

Table 5.13 showed that, the mean number of cigarettes smoked by Malaysian dental outpatients was higher than Yemenis while there is no difference between them with regards the mean duration and mean age of starting smoking.

 Table 5.13: Number of sticks/per day; duration of smoking and age of starting to smoke among Malaysian and Yemeni dental outpatients

Population group	Mean number of sticks perday	Mean Duration of smoking in years (SD)	Mean age of starting smoking in years (SD)
Malaysian	14 sticks/per day	21.10 (13.31)	19 (3.35)
Yemen	3 sticks/per day	22. 35 (14.31)	20 ( 4.64)

# 5.3.2. Quid / Shammah (tobacco quid) chewing

Among the Yemeni dental outpatients, the prevalence of *shammah* users (tobacco quid) was more than Malaysian quid chewers. This relationship was found to be statistically significant (Table 5.14).

Table 5.14: Prevalence of quid chewing in Malaysian and Yemeni dental outpatients (N= 1074).

Population group	Quid chewing	No.quid chewing	p-value *
	n (%)	n (%)	_
Malaysian (n=554)	10 (1.8)	544 (98.2)	_
			0.013
Yemeni (n=520)	23 (4.4)	497 (95.6)	

\* Chi square test was used.

Level of significant was set at 0.05.

Similarity of prevalence quid chewing without tobacco between males and females in Malaysian outpatients while most of quid chewing with tobacco among Yemeni males particularly in aged group  $\geq 55$  (Table 5.15).

Age	Malay	Malaysian dental outpatients			Yemeni dental outpatients			
Group	Male	Female	Total	Male	Female	Total		
18-34	1 (10.0)	1 (10.0)	2 (20.0)	4 (17.4)	0 (0.00)	4 (17.4)		
35-54	0 (0.00)	0 (0.00)	0 (0.00)	2 (8.7)	1 (4.3)	3 (13.0)		
≥55	4 (40.0)	4 (40.0)	8 (80.0)	12 (52.2)	4 (17.4)	16 (69.6)		
Total	5 (50).	5 (50.0)	10 (100.0)	18 (78.3)	5 (21.7)	23 (100.0)		

 Table5.15.Distribution of quid chewing according to age and gender in Malaysian

 and Yemeni dental outpatients

Table 5.16 showed that Malaysian quid chewers started chewing at an earlier age than Yemenis. However, the Yemenis had longer duration of chewing and a higher frequency than the Malaysian quid chewers.

Table 5.16: Frequency/per day, duration of quid chewing and age of starting to chew (N=33)

Population	Mean Frequency/per day	Mean duration of quid chewing in years (SD)	Mean Age of staring to chew in years (SD)
Malaysian	2.2 times /per day	38.49 (23.02)	21.6 (18.2)
Yemeni	7.9 time/per day	54.81 (18.57)	28 (14.05)

Most of the Malaysian quid chewers kept the quid in the sulcus while the Yemeni quid (*shammah*) chewers kept the quid under the tongue. The other site for Yemeni was the lower sulcus (Table 5.17).

Site of placement of quid/shammah	Malaysian n = 10	Yemeni n=23
	no (%)	no (%)
<b>Part of mouth kept the mixture was kept</b> : Left upper sulcus	2 (20)	0 (0.0)
Left lower sulcus	3 (30)	3 (13.04)
Right lower sulcus	3 (30)	1 (4.35)
Anterior lower sulcus	0 (0.0)	6 (2608)
Underneath the tongue	0 (0.0)	12 (53.6)
Others	2 (20)	1 (4.35)

# Table 5.17: Distribution of placement sites for quid /shammah (tobacco quid) chewing among the Malaysian and Yemeni quid chewers

# 5.3.3. Qat chewing

Table 5.18 showed a very high difference of qat chewing in the males as compared to females.

A	Yer	neni dental outpat	tients
Age Group	Male n= (%)	Female n= (%)	Total n= (%)
18-34	58 (69.1)	26 (31.1)	84 (100)
35-54	53 (61.6)	33 (38.4)	86 (100)
≥55	37 (88.1)	5 (11.9)	42 (100)
Total	148 (69.8)	64 ( 30.2)	212 (100)

Table 5.18: Prevalence of qat chewing in relation to age among individuals Yemeni dental patients (N=212)

Table 5.19 showed among the Yemeni qat chewers there is no big difference between males and female of mean hours of qat chewing. The males qat chewers had longer duration more than females.

Gender	Mean hours of chewing (SD)	Mean duration of qat chewing in years (SD)	Mean Age of staring to chew in years (SD)
Male	4.75 (1.93)	22.25 (14.7)	19.5 (4.2)
Female	3.92 (0.74)	14.99 (10.38)	21.44 (4.3)

Table 5.19: Mean hours, duration of qat chewing and age of starting to chew

The highest frequency of qat chewing was every day for both Yemeni males and females (Table 5.20).

# Table 5.20: Frequency of qat chewing and sites of placement qat among the Yemeni outpatients

Frequency/ site placement of qat	Male (n=148)	Female (n=64)	Total (n=212)
chewing	n (%)	n (%)	n (%)
<i>Frequency of qat chewing</i> Every day	109 (73.65)	31 (48.44)	140 (66.04)
two -three times a week	19 (12.83)	8 (12.5)	27 (12.74)
one time a week	20 (13.51)	25 (39.06)	45 (21.23)
Sites of placement qat			
Left	102 (68.92)	39 (60.94)	141 (66.51)
Right	34 (22.97)	21 (32.81)	55 (25.95)
Both	7 (4.73)	3 (4.69)	10 (4.72)

# 5.3.4 Alcohol drinking

From the whole Malaysian dental outpatients was found 4.3 % (n=24) were alcohol drinkers with the mean age of starting drinking being  $19.5\pm6.78$  years.

Table 5.21 showed that the Malaysian males had a higher prevalence alcohol drinking habit as compared to Malaysian females with high prevalence in the Chinese and Indian males.

Gender	Malaya	Chinese	Indian	Total
	n (%)	n (%)	n (%)	n (%)
Male (20)	2 (10.0)	9 (45.0)	9 (45.0)	20 (100)
Female (4)	1 (25.0)	3 (75.0)	0 (0.00)	4 (100)
Total	3 (12.5)	12 (50.0)	9 (37.5)	24 (100)

Table 5.21: Prevalence of alcohol drinking relation to ethnic, gender in Malaysian dental outpatients (N=24)

Most of the alcohol drinkers consumed alcoholic beverage only once/twice per week while the most common drink was beer with high finding among Chinese follow by Indian (table 5.22).

Alcohol consumption	Malaya	Chinese	Indian	Total
	n (%)	n (%)	n (%)	n (%)
<u>Frequency</u>				
Almost daily	1(50.0)	0 (0.0)	1(50)	2(100)
Three to five a week	1(16.7)	3 (50)	2(33.3)	6 (100)
Once or twice a week	1(6.3)	9 (56.3)	6 (37.5)	16 (100)
<u>Type of alcohol</u>				
beer	2 (9.5)	11(52.4)	8 (38.1)	21 (100)
wine	1(33.3)	1(33.3)	1(33.3	3 (100)
others	0 (0.00)	1(100)	0 (0.00)	1 (100)

Table 5.22: Frequency and types of alcohol consumption by Malaysian dental outpatients (N=24).

# 5.4. The relationship between risk habits and the prevalence of oral mucosal lesions in Malaysia and Yemen

Table 5.25 showed that there is a higher prevalence of oral mucosal lesions among the patients with risk habits as compared to those without habits among Malaysian and Yemeni dental outpatients. The relationship was statistically significant.

Risk habits	Lesion	No lesion	<i>p</i> -value *	
	n (%)	n (%)		
Malaysian				
Habit	60 (48.8)	63 (51.3)	0.001	
Without habit	69(16.0)	362 (84.4)	< 0.001	
Yemeni				
Habit	74 (27.6)	194 (72.4)	0.002	
Without habit	42 (16.7)	210 (83.3)	0.003	

Table 5.23: Relationship between the risk habits and prevalence of oral mucosal lesions in Malaysian and Yemeni dental outpatients (N=1074)

\* Chi square test was used.

Level of significant was set at 0.05.

There were more smokers with oral mucosal lesions among Malaysian and Yemeni

dental outpatients. The relationship was found to be statistically significant (Table.5.24).

Table 5.24: Relationship between smoking habit and prevalence of oral mucosal
lesions in Malaysian and Yemeni dental outpatients (N=1074)

Risk habit	Lesion	No lesion	<i>p</i> -value *	
	n (%)	n (%)		
<u>Malaysian</u>				
Smoking	54(50)	54(50)		
Not smoking	75(16.8)	371(83.2)	< 0.001	
<u>Yemeni</u>				
Smoking	46 (46.5)	53 (53.5)	< 0.001	
Not smoking	70 (16.6)	351(83.4)		

\* Chi square test was used.

Level of significant was set at 0.05.

Table 5.25 showed that for the Malaysian and Yemeni dental outpatients, most of the quid chewers and *shammah* users had oral mucosal lesions and the relationship is statistically significant.

Risk habit	Lesion	No lesion	<i>p</i> -value *
	n (%)	n (%)	
<u>Malaysian</u>			
Quid chewers (n=10)	6 (60)	4 (40)	
Non. quid chewers (n=544)	123(22.6)	421(77.4)	0.006
<u>Yemeni</u>			
Sammah users (n=23)	14(60.9)	9 (39.1)	. 0. 001
Non. Shammah users (n=497)	102(20.5)	395 (79.5)	< 0.001

Table 5.25: Relationship between the quid/ tobacco quid (*shammah*) chewing and prevalence of oral mucosal lesions among Malaysian and Yemeni dental outpatients (N=1074)

\* Chi square test was used.

Level of significant was set at 0.05.

The relationship for alcohol drinking and qat chewing was unique to the Malaysian and Yemeni population respectively and thus the analysis for the relationship between the respective habit and the prevalence of oral mucosal lesions was done only for the respective population.

Table 5.26 showed that there are more oral mucosal lesions among alcohol drinkers as compared to non-drinkers and the relationship was statistically significant.

 Table 5.26: Relationship between alcohol drinking and prevalence of oral mucosal lesions among Malaysian dental outpatients (N=554)

Alcohol drinking	Lesion	No.lesion	<i>p</i> -value*
	n (%)	n (%)	
Alcohol drinking (n=24)	10 (41.7)	14 (58.3)	0.020
Not. Alcohol drinking (n=530)	119(22.5)	425(77.5)	0.029

\* Chi square test was used.

Level of significant was set at 0.05.

There is higher prevalence of oral mucosal lesions among the qat chewers as compared to non qat chewers showing a statistically significant relationship (Table5.27).

 
 Table 5.27 Relation between qat chewing and prevalence of oral mucosal lesions
 among Yemeni dental outpatients (N=520)

Qat chewing	Lesion	No.lesion	<i>p</i> -value *
	n (%)	n (%)	
Qat chewing (n=212)	65 (30.7)	147(69.3)	. 0.001
Not. Qat chewing (n=308)	51 (16.6)	257(83.4)	< 0.001

\* Chi square test was used. Level of significant was set at 0.05.

**Chapter Six: Discussion** 

# 6. DISCUSSION

# 6.1. Limitations of the study

The sample size estimation done for this study took into consideration the anticipated difference to be detected in the outcome variable between the two populations groups. Thus, estimation of the anticipated difference was obtained from the available literature related to the study (Axell et al ., 1990 Aiman et al., 2004; Campbell,M and Machin, D., 2005). The main limitation, there was not adequate information on the prevalence of each oral mucosal lesions comparing Yemen and Malaysia. Therefore, among all the oral mucosal lesions, a difference of 8% in the prevalence of oral white lesions in Yemen (22.4%) and Malaysia (14%) was chosen as estimation. Furthermore, a difference of 8% has yielded us the largest affordable sample size which was 546 patients.

Not all outpatients attending to the primary care unit at the Faculty of Dentistry, University of Malaya, Malaysia, agreed to participate in this study as some of them had intended to only seek dental treatment. They instead viewed this study as a waste of their time. Due to this limitation, when we recruited the samples we have employed convenient sampling method which may introduce sampling bias. In general, sampling bias is important when the sample of cases is unrepresentative with respect to the risk factor being studied where some of the outpatients did not agree to participate and considered as a responsive bias (Hulley et al., 2007). However, the response rate in this study was > 70% which was good and acceptable. In addition to that, it also indicates small number of non respondent (<30%) whereby the characteristics of the non respondent were also noted as not much different from the selected samples.

For those who had participated in the study, an explanation was given as to the importance of the oral health screening. The participants were required to answer the questionnaire while waiting to be called into the surgery room for a systematic clinical examination

Due to the above-mentioned problem, there was difficulty in taking photographs for some lesions. In dental clinics of Althawra Modern General Hospital in Sana'a, the registration department referred some outpatients suspected of having oral lesions to nearby specialist clinics. Thus, although there were limitations in the present research study due to its small sample size in the Yemen population, this has not compromised the power of the study which stipulates a power of 94% through a software application.

# 6.2. Prevalence of oral mucosal lesions and comparison between Malaysia and Yemen

Oral mucosal conditions may be caused by local factors (bacterial or viral), systemic diseases, drug-related reactions or lifestyle factors such as the consumption of tobacco, betel-quid and alcohol (Harris et al., 2004). The method to determine the prevalence of oral mucosal lesions may vary. The majority of previous research studies correlate oral mucosal disease with oral cancer and precancerous conditions, while some authors have recorded overall oral mucosal lesions. Axell (1976) for instance, has reported 60 different oral mucosal lesions in Swedish populations. Although the prevalence of oral mucosal lesions has been reported in many countries, these prevalence data are usually restricted to very few lesions in each study. Thus, there is a need to obtain data from different countries with large random samples though the process can be tedious. However, appropriate information on oral mucosal lesion prevalence can still be obtained from small low budget studies on selected population (Axell et al., 1990). Overall, the prevalence of oral mucosal lesions and related risk habits may differ from

region to region in the world. The prevalence of oral mucosal lesions in countries such as Malaysia and Yemen has been reported. However, there are few data on the prevalence of oral mucosal lesions in Yemen. Further, more data on the association of oral mucosal lesions and related risk habits in Yemen is still not well-established. Thus, there is a need to compare the prevalence of oral mucosal lesions and the related habits of the Yemen population with a well-established data among the Malaysian population. The current study is a hospital-based one where it showed that the prevalence of oral mucosal lesions is similar in both Malaysia and Yemen at 23.3 % (n=129) and 22.3 % (n=116) respectively. However, when comparing within the Malaysian population, the prevalence was high as compared to 9.7 % (n=1131) reported in a previous study (Zain et al., 1997). This may be due to the fact that the current study is a hospital-based study where patients tend to prefer visiting the place. The prevalence of oral mucosal lesions in the current study is similar to the finding in a previous study by Taiyeb et al., (1995) where it was found that 22.8% (n=111) may be due to the participants in both studies practising the same habits. The prevalence for Yemenis in this study was similar to an earlier study in Yemen conducted by Aiman et al., (2004) where the prevalence was found to be 22.4% where both the studies are also hospital-based. However, it is very low compared to that found among the Yemeni subjects (slightly lower than 50%) in a study conducted by Hill and Gibson (1987). This could be due to the latter study being carried out only among the Yemeni qat-chewers. In comparing the finding of the present study with that of worldwide studies such as a study in the USA carried out by Shulman (2005), it was reported that the prevalence of oral mucosal lesions in children and youths was 9.1%, which was very low compared to the finding of the present study. This could relate to low prevalence risk habits among children and youths. In an Italian study carried out by Campisi and Margiotta (2000), they reported the prevalence of oral lesions at 81.3% which is very high compared to the present study. This may be the

result of several factors with the most common habit in the latter study being alcohol drinking. The age of the participants was  $\geq 40$  years old and there was also another risk habit of exposure to actinic radiation.

However, the statistical results in the present study is higher than that found in another hospital based in Saudi Arabia which reported 15% (n=383) (Mobeeriek and AlDosari, 2009). This may be due to the age of the subjects in the latter study which was 15 years old and above, which could indicate the difference of risk habit practice among the population. Higher frequency of oral mucosal lesions is also found among Yemeni dental outpatients aged 18-34. However, among Malaysian outpatients, most of the oral mucosal lesions were found among those aged  $\geq$ 55 years compared with recent findings among patients ranging between the ages of 65 to 74 (Zain et al., 1997).

Among Malaysian dental outpatients, the prevalence of oral mucosal lesions in males and females was 84 (34.3%) and 45 (14.6%) respectively. This could reflect that the most risk habit was among Malaysian males. However, there is not much difference of the prevalence of oral mucosal lesions in males and females among Yemeni dental outpatients which is 63 (26.9%) to 53 (18.5%). Aiman et al., (2004) reported that the prevalence of oral mucosal lesion among Yemeni females to males was 32.8% (20.8%) That perhaps resulted to the latter study including many females with risk habit (qat chewing).

The most common lesions among Malaysian outpatients found in the present study were cheek biting which represented 7.2%, followed by Fordyce's spots 3.4%, frictional lesions 3.2% and denture stomatitis 2.0%. However, the most common oral mucosal lesion among Malaysian subjects in a study by Taiyeb et al., (1995) was tongue lesions, 10.7%, followed by oral pigmentation (4.9%) and white lesions (4.3%) in a the study conducted on elderly Malaysians. The most common lesion among Yemenis was frictional lesion, similar to that found among Yemeni subjects by Aiman et al., (2004).

Both studies showed that qat chewing was the most common practising habit among them. The prevalence of oral mucosal lesions reported by Harris et al., (2004) was 28.1% (n=195). This is slightly higher than the findings in the present study. The sample size of the study is close to the present study. However, all participants in the study carried out in the UK were alcohol drinkers.

# 6.2.1. Oral malignant lesions and oral potentially malignant disorders

The prevalence of oral malignant lesions among Yemeni outpatients (1.7%) is found to be higher than that of their Malaysian counterparts. This could be due to the fact that more Yemeni dental outpatients chewed quid containing tobacco called shammah. In addition, it is very high compared to the finding by Zain et al., (1997) that may be associated with the latter being a nationwide study and the participants could have also practised different habits. In the present study however, the prevalence of oral malignant in the Malaysian outpatients is similar to the finding among Malaysian outpatients by Axell et al (1990) since both studies are hospital based and the dental outpatients in both studies had practised same habits (quid chewing). However, in the present study, the prevalence of oral malignant lesions among Malaysian outpatients is low as compared to that found in elderly Malaysian subjects (Ali et al., 1996) where 12.7% of subjects had practised quid chewing. Another study among the Yemeni population carried out by Sheilfe et al (2007) reported that the oral cancer rate among *shammah* users is low compared to the finding of the Yemeni dental outpatients in the present study. This may relate to the study being a case control one and the sample was lower than the present study.

In a recent study in India carried out by Mehrota et al (2010), the prevalence of oral squamous cell carcinoma was reported to be 0.5% (n=2) which is slightly higher than the findings among Malaysian outpatients, even though the participants practised the

same habits in both studies. That may be due to the majority of the Indian subjects indulging in risk habits. However in comparison to the Yemeni dental outpatients, it was found to be very low and this maybe the result of the Yemeni dental outpatients practicing different habits. Comparing the Yemeni dental outpatients in the present study and in a recent study in India carried out by Mathew et al., (2008), the prevalence of oral malignant lesions was 1.7% with a high prevalence among males as in the present study. The latter study is a comparable study with the present study because both studies are hospital-based.

The prevalence of potentially-malignant disorders (leukoplakia and lichen planus) among Malaysian outpatients was 0.8% considerably very low compared to the findings among elderly Malaysian subjects (3.3%) where the leukoplakias was the most common potentially malignant disorders (Ali et al., 1996). All the participants in the latter study were aged 60 and above, and most of them were in the risk group: Indians who had chewed quid. The prevalence of potentially malignant disorder among Yemeni outpatients was 0.2% (leukoplakia) very low as compared to (28%) (Leuokplakia, erythroplakia and lichen planus) among Yemeni subjects (Sheilfe et al., 2007). The prevalence of potentially malignant disorders (leukoplakia and lichen planus) in the current study is low as compared to Malaysian subjects reported by Zain et al., (1997) where the study was a population-based and the sampling also came from the indigenous group. In the present study, lichen planus is figured 0.4 % similar to that found among the Malaysian subjects with 0.38% (Zain et al., 1997). However, the lichen planus in the present study was found among the Chinese group only, compared to that found in the latter study in the Malay and Indian groups respectively.

In a study carried out by Chung et al., (2005) in Taiwan, it has been reported that the prevalence of potential malignant disorders is 12.7%, which is high when compared to the findings of the present study (0.89%). This could be attributed to the fact that the

study in Taiwan was a cross-sectional community survey (house to house) in a suburban population where the people practised areca nut chewing in addition to smoking and alcohol drinking. Another study in Thailand carried out by Reichart et al., (1987) showed the prevalence of leukoplakia which was 1.1% high as compared to 0.4% in the current study. This could be referred to the differences in habits practised by the population group (betel and miang chewing). Among the American population, the prevalence of malignant disorders (leukoplakia) was reported around 3% where the leukoplakia was put under oral keratosis (Bouquot and Gorlin ,1986) higher than the finding in the present study. The population in the USA study had practised tobacco in snuff and they were aged 35 and above. The prevalence of leukoplakia among Indian subjects reported by Saraswathi et al., (2006) was 0.6%, close to that found among Malaysian dental outpatients in the present study. This could due to the same habits practiced.

# **6.2.2.** Other lesions (non malignant and non potentially malignant disorder)

#### i. White lesions

In the present study, the prevalence for cheek biting in Malaysian dental outpatients was 7.2% which is higher than the Yemeni outpatients (4.6%). Axell et al., (1990) reported that 5.6% in Malaysian outpatients was presented with white lesions in comparison with 5.1% in the population study conducted among adult Swedish population (Axell, 1976). The present finding for frictional lesions is 5.2% among Yemeni outpatients which is slightly higher than in the Malaysian outpatients 3.2%. However, this finding is similar to that found among Malaysian outpatients and adult Swedish population (Axell et al., 1990; Axell., 1976). In the current study; the finding for leukoedema in the Yemeni outpatients is 0.2% compared to 3.7% found in the Indian outpatients (Methew et al.,

2008). This percentage is also very low compared to 48% that was found in adult Swedish population (Axell, 1976). The finding for geographic tongue in the Malaysian outpatients was 0.5% while 1.0% was found in the Yemeni population, in comparison with the findings reported among Thai and Malaysian population prevalence 5.1% and 6.4% respectively (Axell et al., 1990) and 8.5% among the Swedish population (Axell.1976). Fordyce's spots were observed at 3.1% in the Malaysian outpatients which is more prevalent among males (7%) compared to 0.6% among females. However, Axell et al., (1990) had reported the prevalence of Fordyce's spots in Malaysian outpatients which was 61.8% and 82.8% among the Swedish population (Axell et atl, 1976) which was very different from the finding in the present study.

The frequency of coated tongue in Yemeni dental outpatients was (1.7%) (1.0% in males, 0.8% in female). This finding is lower than that found in the Italian population (47.45%) (Campisi and Margiotta, 2000). The occurrence of hairy tongue in the present study was 0.8% in Yemen outpatients with more frequency in males where a similar percentage of prevalence of 0.9% was reported in Malaysian outpatients (Axell et al., 1990).

# ii. Red oral lesions

The findings of denture stomatitis in Malaysian outpatients was (0.2%) without predilection difference between males and females. This finding was lower than the prevalence of 3.4% found in Malaysian subjects which is more frequent among females (Zain 1995). However, it is close to that found among Indian population (0.84 %) with more frequency among females than males (Mathewa et al., 2008). The prevalence of denture stomatitis was very low compared to the finding in Slovenia population (14.7%) with high prevalence among males (Marji, 2000).

#### iii. Ulcerated lesions

In the current study, the frequency of aphthous ulcer in Malaysian outpatients was 1.4% with a high prevalence among females compared to 0.6% found among Yemen outpatients with more frequency among males. Zain (2000) reported a prevalence of 0.5% among Malaysian subjects. The finding of the present study among Malaysian outpatients was similar to that found among found in adult Chinese (Lin et al., 2001) but lower than that reported by Mathewa et al., (2008) among Indian population with a prevalence of 2.1%.

#### iv. Pigmented / swelling lesion

Fibro epithelial polyp in the present study figured 0.5% and 0. 6% in both the Malaysian and Yemeni participants respectively. The prevalence is low compared to the one found in 3.9% of the Malaysian outpatients (Axell et al., 1990). In the present study, the findings of hairy tongue among Yemeni outpatients was 0.8% similar to that found among the Malaysian outpatients (0.9%) (Axell et al., 1990). Similar observation for pyogenic granuloma was noted in the present study among Malaysian and Yemeni outpatients which was 0.2%. However, this percentage is low compared to that found among Thai and Malaysian outpatients (Axell et al., 1990) but similar to that found in adult Swedish population (Axell., 1976).

# v. Quid related lesion

In the present study, the prevalence of Betel Chewer's Mucosa was 0.4% which was predominant in Indian women compared to 4.9% that was found among the Malaysian subjects with the most frequent being among the Indians and more among women (Zain et al., 1997). Zain et al., (1995) also reported that 1.6% of the Malaysian subjects were presented with Betel Chewer's Mucosa and a majority are subjects of high risk groups such as Indians and other Bumiputras. The prevalence for *Shammah* related lesion in Yemeni outpatients was 0.2%. This percentage is very low compared to the finding in Yemeni subjects with 31% prevalence of shammah relate lesion (Scheifele et al., 2007). However, the latter study was conducted among the *shammah* users.

# 6.3. Prevalence of risk habits and comparison between Malaysia and Yemen

The common risk habits creating oral mucosal lesions are tobacco smoking, quid chewing and alcohol drinking, In addition to the environmental and genetic factors (Johnson, 2003b ) on the other hand, many studies showed the association between some oral mucosal lesion and qat chewing (Alsharabi, 2002; Aiman et al., 2004; Gorsky et al., 2004; Fasanmade, 2007). The prevalence for risk habits (predominately smoking habit) in the Malaysian outpatients is 22.2%, close to that found in in Malaysian outpatients ((Predominately smoking habit) (Axell et al., 1990). Among Yemeni outpatients, the high prevalence of risk habit of 51.4% was qat chewing. This is slightly lower compared to a report by Aiman et al., (2004). In the present study, the most risk habits in Malaysia are among men (85.4%). However, there was no big difference of risk habits between men and women among the Yemeni outpatients.

#### 6.3.1 Smoking habits

In the present study, the similarity of prevalence for smoking habits in Malaysian and Yemeni outpatients of 19.5 and 19.0% respectively was recorded. The prevalence is lower than what was reported among Yemeni subjects (Aiman et al., 2004). The smoking habit in the present study was the predominant habit among Malaysian outpatients similar to that found by Axell, et al (1990). The prevalence of smoking habits in American Indians/ Alaskan natives in a study carried out by Park et al (1997) and other study conducted in rural China by Yang et al (2008) is very high compared to the finding in present study. The current study showed that the smoking habits are more prevalent in Yemeni in the age groups ranging from 18-34. However, the smoking habit in the Malaysian dental outpatients, the frequency of smoking habits among men was 17.3% and this is similar to the other study by WHO (2004 (b)) which showed that the percentage of women smokers was smaller than that of male smokers (women 7.2% and men 49.8%). However, the smoking habits in Yemeni subjects were more pronounced among males than in females at 32.8% and 9.4% respectively (Aiman et al., 2004).

#### 6.3.2. Quid chewing

In a recent study, the prevalence of quid chewing without tobacco in Malaysia was 1.8 %, slightly lower compared to 2.6% reported by Axell et al., (1990). However, this is very low compared to that found in the Asian regional study (WHO,2004(a) (Gupta and Warnakulasuriya, 2002). The prevalence of tobacco quid (*shammah*) in this study among the Yemeni participants was 4.4% which is higher than the that of the Malaysian outpatients. The composition of *shammah* in this study was tobacco, lime and ash. However the composition of the *shammah* in Algerians was tobacco, carbonate lime and other substance (Zhang et al., 2001).

#### 6.3.3 Alcohol drinking

The prevalence of alcohol consumption among Malaysian dental outpatients was found to be at 4.3% which is higher compared to the findings among Singaporean Malays at 1.1%. However, the percentage is very low with that found among Thai population at 31.4% (WHO, 1999). The previous study carried out by Zain et al (1995) had found the alcohol consumption to be 35.4% and more prevalent among the Chinese groups for both studies.

#### 6.3.4 .Qat chewing

In the present study, the prevalence of qat chewing in Yemeni outpatients was figured at 40.8% which is low in comparison with previous studies carried out among Yemeni subjects (Aiman et al., 2004, Alsharabi. 2002). Most of the qat chewers in the present study were males similar to that found in a study by Aiman et al., (2004). In the present study, the males who practised qat chewing were more than the females at 69% (n=148) and 32 .2% (n=64) respectively as compared to qat chewers among Yemeni subjects reported by Aiman et al., (2004) (87.04%) and (12.9%) respectively. In comparison to the frequency of qat chewing, the present study showed high frequency of qat chewing everyday and most of the qat chewers chewed qat leaves in the left side of the mouth similar to a previous study by Aiman et al., 2004.

# 6.4. The relationship between risk habit and prevalence of oral mucosal lesions in Malaysia and Yemen

The findings in the present study showed that there is a higher prevalence of oral mucosal lesions among patients with risk habits as compared to those without habits among Malaysians and Yemenis dental outpatients The relationship was statistically significant supporting the positive association between cigarette smoking and leukoedema, as well as denture stomatitis (p<0.001) (Zain and Razak, 1989). The current study shows more Malaysian and Yemeni smokers with oral lesions. The relationship was found to be statistically significant (p < 0.001) as found in a Spanish population which indicated that there is a significant association between smoking and prevalence of oral mucosal lesion (García-Pola et al., 2002). This study also showed that most of the quid chewers among Malaysian and Yemeni outpatients presented with oral mucosal lesions and the relationship was statistically significant in comparison with a study by Axell et al., (1990) which reported that the oral malignant and the most of potentially malignant disorders has an association with betel quid chewing. In another study by William et al., (1999) in Saudi Arabia, it was reported that oral cancer had appeared where *shammah* (tobacco quid) was commonly used. Among the Malaysian outpatients, there were high oral mucosal lesions among alcohol drinkers similar to another study in an adult Spanish population by García-Pola et al., (2002).

In the current study, there were found more oral mucosal lesions among the qat chewers. There is a high statistically significant relationship between the prevalence of oral mucosal lesions and qat chewing with a p-value <0.001 similar to that found in a study by Aiman et al (2004) which reported that the oral white lesions were more common among the qat chewers than the non- qat chewers. In the present study there is similarity in the prevalence of oral mucosal lesions between Malaysia and Yemen even though there is a big difference of the prevalence of risk habit between Malaysian and

Yemen where the risk habit among Yemeni outpatients was higher than the Malaysians. These may be due to the most common habit among Yemeni was qat chewing. Qat is a green leave. The most active ingredients of qat are alkaloids such as cathinone and cathine. Cathinone is the main psychoactive constituent of qat, and has a similar action to amphetamine, inducing the release of dopamine, a neurotransmitter, from presynaptic storage (Kalix 1992, Patel. ,2000). There are also small amounts of ethereal oil, sterols and triterpenes, together with 5% protein which has insignificant nutritional value. Ascorbic acid is also present in the leaves (Raman, 1983). Khat leaves also contains tannin (7–14% by weight in dried leaves) and minute amount of thiamin, niacin, riboflavin, iron and amino acids (Lugman and Danowski, 1976). Apart from tannin, these substances are unlikely to contribute to the biological effect of khat (Kalix, 1992). The above composition of the qat leaves may be responsible to lesser effect in oral mucosa

**Chapter Seven: Conclusion** 

#### 7.1. Conclusion

This study found that there is no difference in the prevalence of oral mucosal lesions among Malaysian and Yemeni dental outpatients. However, there is a difference in the prevalence of risk habits in Malaysian and Yemeni dental outpatients. The relationship was statistically significant at p <0.001. The prevalence of smoking habits was similarat both centres, and statistically significant difference was found in the prevalence of quid chewing habits between Malaysian and Yemeni dental outpatients (p=0.013). There is a low prevalence of alcohol drinking habits among Malaysians with no such habits found among Yemenis; while there is a high prevalence of qat chewing habits among Yemenis and the habits do not exist among Malaysians. There is a higher prevalence of oral mucosal lesions among the dental outpatients who have risk habits as compared to those without habits and this relationship was found to be statistically significant.

#### 7.2. Recommendation

It is hoped that the results from this study will lead to similar works on a more nationwide scale in Malaysia. In the context of Yemen, it is hoped that the findings will act as a database for a nationwide survey with larger samples to better understand the influence of oral risk habits in the prevalence of oral mucosal lesion especially the precancerous and cancerous lesions. It is recommended that more hospital dental clinics participate in such a research. In addition, this study will help to identify the risk groups and aid programmes that can improve the oral health on a regular basis to promote oral health care and awareness among the population. , Further studies need be conducted to look into the outpatients of variable clinics and Yemen aid nationwide study to determine the prevalence of oral mucosal lesions and related risk habits.

#### 8. REFERENCES

Abu Bakar, S. (2006). Smoking Cessation Programme in Malaysia. Proceeding Oral Cancer in Asia Pacific–A Regional Updated & Networking, Kuala Lumpur, Malaysia p. 49-50.

Aiman, A. A. (2007). Histopathologic changes in oral mucosa of Yemenis addicted to water-pipe and cigarette smoking in addition to *takhzeen al-qat*. Oral Surg Oral Med Oral Pathol Oral Radiol Endod; 103:e55-e59.

Aiman, A. A., Al-Sharabi, A. K. and Aguirre, J. M. (2006). Histopathological changes in oral mucosa due to takhzeen al-qat: a study of 70 biopsies. J Oral Pathol Med; 35:81–5.

Aiman, A. A., Al-Sharabi, A. K., Aguirre, J. M. and Nahas, R. (2004). A study of 342 oral keratotic white lesions induced by *takhzeen* al-qat among 2500 Yemeni. J Oral Pathol Med; 3: 368–72.

Albrecht, M., Bánóczy, J., Dinya, E., Tamás, G. Jr. (1992). Occurrence of oral leukoplakia and lichen planus in diabetes mellitus. J Oral Pathol Med; 21:364.

Al-Mobeeriek, A. and AlDosari, A. M. (2009). Prevalence of oral lesions among Saudi dental patients. Ann Saudi Med; 29: 365-8.

Alaya'a, Z. (2009). Yemen ranks second in the number of smokers in Arab World Observer (ye); issued by World Health Organization.

Ali, T. B., Jalalludin R. L., Abdul Razak, I, I. A., Zain, R. B. (1996). Prevalence of oral precancerous and cancerous lesions in the elderly Malaysians. Asia pacific Academic Consortium for Public Health (ACPACPH) J; 9:24-27.

AlSharabi, A. K. (2002). Oral and para-oral lesions caused by *takhzeen* (chewing) al-Qat. Thesis Doctoral, University of Khartoum: Al-Khartoum (Sudan). p. 122.

Amer, M., Bull, C. A., Daouk, M., McArthur, P. D., Lundmark, G. J. and EL-Sensoussi, M. (1985). 'Shamma ' usage and oral cancer in Saudi Arabia Ann.Saudi Med 5:135-41.

Axell, T. (1976). A Prevalence study of oral mucosal lesions in an adult Swedish population .Odont Revy; 1-100.

Axell, T. (1987). Occurrence of leukoplakia and some other white lesions among 20333 adult Swedish people. Community Dent Oral Epidemiol; 15: 46-51.

Axell, T and Rundquist, L. (1987). Oral lichen planus – a demographic study. Community Dent Oral Epidemiol; 15: 52.-56.

Axell, T., Holmstrup, P., Kramer, I. R. H., Pindborg, J. J.and Shear, M. (1984). International seminar on leukoplakia and associated lesions related to tobacco habits.Community Dent Oral Epidemiol; 12:145-154.

Axell, T., Pindborg., J. J., Smith, C. J., van der Waal, I. (1996). International collaborative group on oral white lesions with special reference to precancerous and tobacco related lesion. Conclusions of an international symposium held in Uppsala, Sweden. J Oral Pathol Med; 25:49-54.

Axell, T., Zain, R. B., Siwamogstham, T. D and Thampipt, J. (1990). Prevalence of oral soft tissue lesion in out-patients at two Malaysain and Thai dental schools. Community Dent Oral Epidemiol; 18:95-9.

Baan, R., Straif, K., Grosse, Y., Secretan, B., EL Ghissassi, F., Bouvard, V. Altieri, A., Cogliano, V. (2007). Carcinogenicity of alcohol beverages. Lancet Oncol; 8: 292-293.

Banoczy, J. and Rigo, O. (1991). Prevalence study of oral precancerous lesions with a complex screening system in Hungary. Community Dent Oral Epidemiol; 19: 265-267.

Barnes L,Eveson J.W., Reichart P., Sidransky D.(2005). World Health Organization classification of tumors.Pathology and Gentics of Head and Neck Tumors. IARC Press. Lyon p.166

Baum, B. J. (2007). Inadequate training in biological sciences and medicine for dental students impending crisis for Dentistry. J Am Dent Assoc; 138:16-25.

Bischoff, E.W., Uijen, A., Van der wel, M. (2009). Aphthous ulcers. BMJ Publishing Group Ltd .339, 25, p b2382.

Bouquot, J. E. Gorlin, R.J. (1986). Leukoplakia, lichen planus and other oral keratoses in 23,616 white Americans over 35 years of age. Oral Surg Oral Med Oral Pathol; 61:373-381.

Bougout, J. E. (1986). Common oral lesions found during a mass screening examination. J Am Dent Assoc; 112:50-57.

Bruch, J. M and Treister, N. S. (2009). Clinical oral medicine and pathology. Humana Press. London. P: 11-23.

Cawson, R. A., Langdon, J. D. and Eveson, J. W. (1996). Erythroplasia ('erythroplakia').In: Surgical pathology of the mouth and jaws. Wright—an imprint of Butterworth–Heinemann Ltd., Oxford, London, Boston. p.180.

Chung C. H., Yang Y.H., Wang T. Y., Shieh T.Y., Warnakulasuriya S. (2005). Oral precancerous disorders associated with areca quid chewing, smoking, and alcohol drinking in southern Taiwan. J Oral Pathol Med 34: 460–6.

Demir, Y., Demir, S. and Aktepe, F. (2004). Cutaneous lobular capillary hemangioma induced by pregnancy. J Cutan Pathol; 31: 77-80.

Dental Division. Ministry of Health. Malaysia (1978). Dental epidemiological survey of adults in peninsular Malaysia 1974-1975. Dental Division. Ministry of Health. Malaysia; 36-37: 98-9.

Dupont, W. D., Pulmmer, W.D.Jr. (1998). Power and sample size calculation for studies involving linear regression. Contal Clin Trail; 19(6):589-601.

El- Akkad, S. M., Amer, M. H., Lin, G. S., Sabbah, R. S. and Godwin, J. T. (1986). Pattern of cancer in Arabians referred to King Faisal Specialist Hospital Cancer. 58:1172-8.

El-Shoura, S. M., Abdel Aziz, M. and Ali, M. E. (1995). Deleterious effects of khat addiction on sperm parameters and sperm ultra-structure. Hum Reprod; 10: 2295-2300. El-Wajeh, Y. A. M. and Thornhill, M. H. (2009). Qat and its health effects; Br Dent J; 206:17–21.

Epinoza, I., Rojas, R., Aranda, W. and Gamonal, J. (2003). Prevalence of oral mucosal lesion in elderly people in Santiago Chisle. J Oral Pathol Med; 32:571-5.

Fasanmade, A., Kwok, E. and Newman, L. (2007). Oral squamous cell carcinoma associated with khat chewing. Oral Surg Oral Med Oral Pathol Oral Radio Endo; 104(1):e53-e55.

Field, A., Longman, l. (2003). Tyldesley's oral medicine fifth edition Oxford University Press. UK. Chapter 6.p. 69.

Campisi, G. and Margiotta, V. (2001). Oral mucosal lesions and risk habits among men in an Italian study population. J Oral Pathol Med; 30:22-8.

Cardesa A., Slootweg. P. J. (2006). Pathology of head and neck. Springer-Verlag Berlin Heidelberg. p.108

Gerhauser, C. (2005). Beer constituents as potential cancer chemopreventive agents. Eur.J Cancer; 41(13): 1941-54.

García-Pola Vallejo, M. J., Martínez Díaz-Canel A. I, García MartínJ, .M., González García M. (2002). Risk factors for oral soft tissue lesions in an adult Spanish population Community Dent Oral Epidemiol; 30: 277–85.

Greenberg, M. S. and Michael Glick, M. (2008). Burket'sOral MedicineDiagnosis & Treatment 11<sup>th</sup> edition Hamilton. Spain. p. 10, 77.

Gorsky, M., Epstein, J. B., Levi, H., Yarom, N. (2004). Oral white lesions associated with chewing khat. Tobac induced dis; 2: 145–150.

Gupta, P. C., Mehta, F. S., Daftary, D. K., Pindborg, J. J., Bhonsle, R. B., Jalnawalla, P. N.Sinor P.N,Pitkar V.K,Murti P. R., Irani R.R ,Shanh H.T., Kadam, P.M., Iyer, K.S, Iyer H.M., Hegde, A. K, Chandrashekar, G.K, Shiroff B.C., Sahiar, B.E. and Meha, M.N (1980). Incidence of oral cancer and natural history of oral precancerous lesions in a 10-year follow-up study of Indian villagers. Community Dent Oral Epidemiol; 8(6):283–333.

Gupta, P. C. and Ray, C. S. (2003). Smokeless tobacco and health in India and South Asia. Respirology; 8:419–31.

Gupa, P. C., Zain, R. B., Ikeda, N., Yaacob, M. and Metha, H. (1997). Prevalence of tobacco use behavior in Malaysia-result of sample survey .Book of abstract for the 10<sup>th</sup> World Conference on tobacco or health in Beijing ,China –August 24-28: p 60,abstract #OS 110.

Gupta, P. C and Warnakulasuriya, S. (2002). Global epidemiology of areca nut usage addict. Biol.7:77-83.

Halbach, H. (1972). Medical aspects of the chewing of khat leaves. Bull WHO; 47:21-29.

Haniza, M. A., Maimunah, A. H., Rusilwati, J., Latipah, S. and Surya, A. (1999). National Health and Mobility Survey: Smoking among adult. Institute of public health, Ministry of Health Malaysia. Kaula Lumpur. 15:23.

Harris, C. K., Warnakulasuriya, K. A., Cooper D.J., Gelbier, S. (2004). Prevalence of oral mucosal lesions in alcohol misusers in south London. J Oral Pathol Med.; 33(5):253-9.

Heinekens, C. H. and Buring, J. E. (1987). Epidiomlogy in Medicine Boston /Tornoto .Little Brown and Company; PP.64-65.

Hill, C. M and Gibson, A. (1987). The oral and dental effect of the qat chewing. Oral Surg Oral Pathol; 63:433-6.

Hulley, S, B, Cummings, S,R. Browner, W, S; Grady, D, G. Newman, T, B. (2007) Designing Clinical Research, 3rd Edition Lippincott Williams & Wilkins. USA. P.116

Ibsen, O. Phelan, J, (2009). Oral Pathology for Dental Hygienist. , 5th edition. Philadelphia,USA Elsevier Health Sciences. p.

Jaber, M. A., Porter, S. R., Gilthorpe, M. S., Bedi, R. and Scully, C. (1999). Risk factors for oral epithelial dysplasia--the role of smoking and alcohol Oral Oncol 35 :2; 151-6.

Johnson, N. (2001). Tobacco use and oral cancer: a global perspective. J Dent Educ.65:328-339.

Johnson., N.W. Aetiology and risk factors for oral cancer. In: Shah JP, Johnson NW, Batsakis JG, eds. (2003) Oral Cancer. London: Martin Dunitz, an imprint of the Taylor & Francis Group. Pp.33–75.

Kalix, P. (1992). Cathinone: a natural amphetamine. J Pharmacol Toxicol; 70:77-86.

Kennedy, J., Teague, J., Rokaw, W. and Cooney, E. (1983). A medical evaluation of qat use in North Yemen .Social Science and Med; 11:783-793.

Kupper, H., Boffetta, P., Adami, H. O. (2002). Tobacco use and cancer causation: association by tumor type. J Int Med.252:206-224.

Langlais, R. P., Miller, C. S., Nield-Gehrig, J.S. (2009). Color atlas of common oral disease. Lippincott Williams & Wilkins. Philadelphia . p. 55–7.

Laskari, G. (2006). Pocket Atlas of oral diseases. Georg Thieme Verlag. Germany. 2<sup>nd</sup> edition. P. 224

Llewellyn, C. D., Johnson, N. W. and Warnakulasuriya, K. A. A. S. (2004). Risk factors for oral cancer in newly diagnosed patients aged 45 years and younger: a case–control study in Southern England J Oral Patho.Med. 33; 9: 525-532.

Lin, C. F., Wang, J. D., Chen, P. H., Chang, S. J., Yang, H.Y. and Ko, Y. C. (2006). Predictors of betel quid chewing behaviour and Cessation patterns in Singapore pharmacogeneti; 5: 332-334.

Lin H. C., Corbet E.F., Lo E. C. (2001). Oral mucosal lesions in adult Chinese. J Dent. Res.; 80(5): 1486-90

Lu, C. T., Yen, Y. Y., Ho, C., Ko, Y. C., Tsai, C. C., Hsieh, C. C. and Lan, S. J. (1996). A case control study of oral cancer in Changhua County Taiwan. J Oral Pathol Med; 25:245-248.

Luqman, W. and Danowski, T. S. (1976). The use of qat in Yemen: Social and medical observations .Annals of Internal Medicine 85:246-249.

Lynch, M. A., Brightman, V. J., Greenberg, M. S. (2003). Burkets OralMedicine, 9th edn. New York: Lippincott-Raven.

La Vecchia, C., Lucchini, F., Negri, E., and Levi, F. (2004). Trends in oral cancer mortality in Europe. Oral Oncol 40, 433–439

Macigo, F. G., Mwaniki, D. L. Gutha, S. W. (1995). The association between oral leukoplakia and use tobacco, alcohol and khat .Euro J Oral Sci;103(5): 268-273.

Machin, D Campbell,M,J, (2005). Design of Studies for Medical Researchd John Wiley & Sons Ltd, Englan. P.44,45 Mehrota., R, Thomas., S, Nair., P, Pandya., S, Singh.,M, Nigam., N.S., Shulka P.(2010). Prevalence of oral soft tissue lesions in Vidisha. BMC Res Notes. 2010 Jan

25; 3:23.

Metha, F. S., Gupta, P. C., Daftary, D. K., Pindborg, J. J. Choski.k.S. (1972). An Epidemiologic Study Of Oral Cancer And Precancerous Conditopn Among 101761 Villagers In Matharashtra, India. Int Jcancer;10:134-41.

Methew, A. L., Pai, K. M., Sholapurkar, A. A. Vengal, M. (2008). The prevalence of oral mucosal lesions in patients visiting a dental school in southern india .Indian J Dent Res; 9:99-103.

Marija Kovac-Kavcic Uros Skaleric (2000). The prevalence of oral mucosal lesions in a population in Ljubljana, Slovenia J Oral Pathol Med; 29: 331–5.

McKee, C. M. (1987). Medical and social aspects of qat in Yemen: a review. Journal of the Royal Society of Medicine; 80 (12): 762-5.

Morse, D. E., Psoter, W.J., Cleveland, D., Cohen D, Mohit-Tabatabai, M.,Kosis, D.L., Eisenberg, E. (2007). Smoking and drinking in relation to oral cancer and oral epithelial dysplasia cancer causes control 18(9):919-929

Nair, U. J., Nair, J., Mathew, B. and Bartsh, H. (1999). Gluthathione S-tranferase MI and T1null genotype as risk factorsfor oral leukoplakia in ethnic Indian betel/quid chewers. Carcinogensis;20:743-748.

Nencini, P., Grasssi, M. C., Botan, A. A., Asseyr, A. F. and Paoli, E. (1988). Khat chewing spread to the Somali community in Rome. Drug Alcohol Depend; 23: 255-258.

Neufeld, K. J., Peters, D. H., Rani, M., Bonu, S., Brooner, R. K. (2005). Regular use of alcohol and tobacco in India and its association with age, gender and poverty. Drug Alcohol Depend; 77(3):283-91.

Park, J.K., Muscat, J. E., Ren, Q., Schantz, S. P., Harwwhick, R. D., Stern, J. C., Pike, V., Richie, J. P. Jr., and Lazarus, P. (1997). CYPIAI and CSTMI polymorphysims and oral cancer risk. Cancer Epidemiol Biomakers PREV.6:791-797.

Patel, N. B. (2000). Mechanism of action of cathinone: the active ingredient of qat (Catha edulis). East Afr Med J; 77:329–332.

Peto R, Lopez AD, Boreham J, Thun M, Heath C Jr (1992) Mortality from tobacco in developed countries. The Lancet 339: 1268–1278.

Pindborg, J. J., Reichart, P. A., Smith, C. J. and Van der Waal, I. (1997). A denoid squamous cell carcinoma. World Health Organization: Histology typing of cancer and precancer of oral mucosal. 2. Springer, Berlin Heidelberg New York; p. 15.

Proceeding of workshop on lesions associated with betel-quid & tobacco chewing habits with special consideration on betel chewer's mucosa. Dent J Malaysia, 1997; 18:6-8.

Pindborg, J. J., Bhonsle, R. B., Murti, P. R., Gupta, P. C., Daftary, D. K. and Mehta, F. S. (1980). Incidence rate and early forms of oral submucous fibrosis. Oral Surg Oral Med Oral Pathol; 50: 40–4

Oakley, E., Demaine, L. and Warnakulasuriya, S. (2005). Areca (betel) nut chewing habit among high-school children in the Commonwealth of the Northern Mariana Islands (Micronesia). Bull World Health Organ 83:656–660.

Ramanthan K and Lakshami S. (1976). Oral carcinoma in peninsula Malaysia: racial variation in the Indian, Malays, Chinese and Caucasian .G.A.N.N Monograph on Cancer Res. 18:27-36.

Ramanthan, K., Keat, T. C., Retnanesan, A. and Canaganayagam, A. (1973a). Oral precancerous condition frequency in 1648 Malaysians with correlation to oral habits. Dent J Malaysia-Singapore; 13:11-12.

Ramanathan, K., Canayanayagam, A., Tan, C. K. and Ratnanesan, A. (1973b). Freguency of oral precancerous conditions to oral habits Med J Malays; 27: 173-81

Raman R (1983). Catha Edulis Forskal. Geographical dispersal, botanical, ecological and agronomical aspects with special reference to Yemen Arab Republic.Gottingen: University of Gottingenpublications, 30–119.

Reichart, P., Lenz, H., Beck-Er, J. and Mohr, U. (1985). The black layer on the teeth of betel chewers:a light microscopic, microdiographic and electron-microscope study. J Oral Pathology; 14: 466-75.

Reichart, P A. (2000). Oral mucosal lesions in a representative cross-sectional study of aging Germans. Community Dent Oral Epidemiol; 28:390-8.

Reichart, P. A, Mohr, U., Srisuwan, S., Geerling, H., Theetranont, C. kangwanpong, T. (1987). Precancerous and other oral mucosal lesions related to chewing, smoking ,and drinking habit in Thailand, community Dent Oral Epidemiol .15:152-160.

Reichart, P. A. (2006). Risk habits and oral cancer in Asia-Pacific Region .oral cancer in the Asia pacific – A Regional Update & Networking .Kuala Lumpur, Malaysia (Pg19-23).

Regezi J.A., Sciubba, J.J., Jordan, C.K. (2008). Oral Pathology: Clinical Pathologic Correlations. Philadelphia: WB Saunders Co. p.84

Salonen, L., Axell, T., Hellden, L. (1990). Occurance of oral mucosal lesion s, the influence of tobacco habits and estimate of treatment time in an adult population. J Oral Pathol. Med.; 19: 170-6.

Salem, G., Juhl, R. and Schiodt, T. (1984). Oral malignant and premalignant change in shammah'-users from Gizan region, Saudi Arabia. Acta OdontolScand; 42 (1): 41-5.

Saraswathi, T. R., Ranganathan k., Sharmugam S., Sowmya R., Narasimhan P.D., Gunaseelan R. (2006). Prevalence of oral lesions in relation to habits:cross-sectional study in South India. Indian J Dent.Res.17 (3):121-5

Schantz, S. P. and Yu, G. P. (2002). Head and neck cancer incidence trends young Americans, 1973-1997, with a special analysis for tonguecancer. Arch Otolaryngol Head Neck Surg. 128: 268–274.

Scheifele, C., Nassar, A. Reichart, P.A. (2007). Prevalence of oral cancer and potentially malignant lesions among shammah users in Yemen. Oral Ocol; 43 (1): 42-50.

Schwimmer, E. (1877). Some rare clinical pictures of oral and lingual mucosa .Orv Enyesult Evkonyve ; No48.

Scully, C. and Felix, D. H. (2005). Oral medicine—Update for the dental practitionerAphthous and other common ulcers. British Dental Journal ; 199: 259 –264.

Scully, C. (2004). Oral and maxillofacial medicine. The basis of diagnosis and treatment. Edinburgh, London, New York: Wright. Elsevier Science Ltd.; p. 289–90.

Seedat, H. (1985). Oral submucous fibrosis in Durban, Natal: a study of its epidemiology, aetiology and morphological features. PhD Thesis.University of Stellenbosch.

Seedat, H. A. and Van Wyk, C. W. (1988). The oral features of betel nut chewers without submucous fibrosis. J Biol Buccale; 16:123–8.

Shafer, W. G. and Waldron, C. A. (1975). Erythroplakia of the oral cavity. Cancer;36:1021-1028.

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

Silverman, S.J and Shillitoe, E. J. (1990). Oral cancer. Atlanta: American Cancer Society; 2:12–15.

Silverman, Jr. S. and Griffith, M. (1972). Smoking characteristics of patients with oral carcinoma and risk for second oral primary carcinoma. J Am Dent Assoc; 85: 637–640.

Smith, L.W., Bhargava, K., Mani, N. J., Malaowallla, A.M. and Silverman, S. (1975). Oral cancer and precancerous lesions in 57518 industrial workers of Gujera. India. Indian J Cancer ;12:118-23.

Stewart, B. W. and Kleihues, P. (edu) (2003). World Cancer Report. IRAC Press. ILyon. p22,29.

Stirling G., Zaharan, F., Jamjoon, A and Eed, D. (1981). Cancer of the mouth in the western region of saudia Arabia . A histopathological and experimental study. King Abdulaziz Med J.1: 10-16.

Taiyeb Ali, T. B., Razak, I. A., Raja Latifah, R. J, Zain, R. B. (1995). An epidemiology survey of oral mucosal conditions among eldely Malaysians. Gerodontology; 12:37-40.

Valterio, C. and Kalix, P. (1982). The effect of the alkaloid cathinone on motor activity of mice. Arch Int Pharmacodyn; 255: 196-203.

Van der waal, I. (2009). Potentially malignant disorders of the oral and oropharyngeal mucosa; terminology, classification and present concepts of management. Oral Oncology 45 4-5:317-323.

Warnakulasuriya, S., Mak, V., Moller. H. (2007). Oral cancer survival in young people in South East England. Oral Oncol; 43:982–986.

Wynder, E. L., Bross, I.J. (1957). Aetiological Factors in Mouth Cancer; An Approach to its Prevention, British med. J; 5028:1137-43.

Webb, P., Bain, C. and Pirozzo, S. (2005). Essential of epidemiology: an introduction for students and health professionals, University Press Cambridge. UK. P: 30-31.

William, F., Allard, Edward, B., DeVol and Ofelia, B. Te. (1999). Smokless tobacco (shamma) and oral cancer in Saudi Arabia Community Dent. Oral.Epidemiology; 27: 398-405.

World Health Organization. (1980). Guide to epidemiology and diagnosis of oral mucosal lesions and conditions. Community Dent Oral epidemiol; 8: 1- 26.

World Health Organization . (1978). Definition of leukoplakia and related lesions; an aid to studies on oral per cancer. Oral Surg Oral Med Oral Pathol 46:518-539.

World Health Organization. (1999). Global status report on alcohol, Geneva. Malaysia. west.htm.

World Health Organization (2003). World Cancer Report. Lyon: International Agency for Research on Cancer, IRAC Press. ILyon . p22, 29.

World Health Organization (2004a) International Agency for research on cancer (Monographs, on the evaluation of carcinogenic risks to Humans: betel quid and areca nut chewing and some areca nut derived Nitrosamines –summary of data reported and evaluation Vol .85.Lyon, France.

World Health Organization (2004b). International Agency for research on cancer (IARC) (Monographs, on the evaluation of carcinogenic risks to Humans: Tobacco in involuntary smoking Vol 83.Lyon, France.

World health organization. (1985). Tobacco habits other than smoking; betel quid and areca nut chewing and some related nitrosamines. IRAC Monogr. Eval. of the Carcinog. Risk of Chem Hum.; 37:141-200.

Yang, T., Li, F., Yang, X., Wu, Z., Feng, X., Wang, Y., Wang, X. and Abudllah, A. S (2008). Smoking patterns and socio-demographic factors associated with tobacco use among Chinese rural males residents: a descriptive analysis .BMC Public health 8:284.

Yang, Y-H., Lien1, Y-C., Ho. P-S., Chen, C-H., Chang, JSF, Cheng, T-C. and Shieh, T-Y. (2005). The effects of chewing areca/betel quid with and without cigarette smoking on oralsubmucous fibrosis and oralmucosal lesionsOral Diseases;11:88–94.

Yousif, A. and Hashash, M. (1983). Common features and surgical interference in prevalent oral cancer in Saudi Arabia (a preliminary report). J Laryngol Otolaryngol; 37(9):837-43.

Zain, R. B. (1995). A preliminary report on the prevalence of oral mucosal lesions in army personnel, in Johor, Malaysia Dental J Malaysia; 16(2):40-43.

Zain, R. B. and Ghazali, M. (2001). A Review of Epidemiological Studies Of Oral Cancer And Precancer In Malaysia. Annals of Dentistry, 8 (1). pp. 50-56. ISSN 0128-7532.

Zain, R. B., Ikeda, N., Gupta, P. C., Warnakulasuriya, K. A. A. S., van Wyk, C. W. and Shrestha, P. (1999). Oral mucosal lesions associated with betel quid, areca nut and tobacco chewing habits: consensus from a workshop held In Kuala Lumpur, Malaysia, November 25–27, 1996. J Oral Pathol Med; 28(1):1-4.

Zain, R. B., Ikeda, N., Razak, I. A., Axell, T., Majid, Z. A., Gupta, P. C. and Yaacob M. (1997). Anational epidemiology survey of oral mucosal lesion in Malaysia Community Dent Oral epidemiol; 25: 377-83.

Zain R.B. (2000). Oral recurrent aphthous ulcers/stomatitis: prevalence in Malaysia and epidemiological update J Oral Sci; (42) (1): 15-9.

Zain, R. B., Ikeda, N., Reichart, P. Axell, T. (2002). Clinical criteria for diagnosis of oral mucosal lesion published by Faculty of Dentistry, University of Malay Kuala Lumpur. Malaysia;10-13.

Zain, R. B., Ikeda, N. and Yaacob, M. (1995). Oral mucosal lesions survey of adults in Malaysia. Kuala Lumpur, Ministry of health, Malaysia University of Malaya, Malaysia and Aichingakuin University. Japan; 1-61.

Zain, R. B and Razak, I. A. (1989). Association between cigarette smoking and prevalence of oral mucosal lesions among Malaysian army personnel. Community Dent Oral Epidemiol; 17: 148-9.

Zhang, X., Schmitz, W., Gelderblom, H. R. and Reichart, P. (2001). Shammah induced oral leukoplakia like – lesions. Oral Oncology; 37: 609-612.

# APPENDIX

# **STUDY POPULATION**

# **APPENDIX 1**











A.1. Fig. Overview of field work (interview questionnaire and clinical examination)

# **CONSENT & APPROVAL**

#### A.2. Patient information sheet (English version)



#### **Patient Information Sheet**

Study title: prevalence of oral mucosal lesion in Malaysia and Yemen

#### Introduction:

Oral mucosal lesions may be defined as any alteration in oral mucosa .This alteration or lesions can be congenital, precancerous or cancerous. Risk factors are invariably associated to the spread of these lesions. These factors range from habits like smoking, tobacco chewing, betel quid, areca nut, to qat chewing etc. Additionally, there are sociodemographic factor which can play a role in the occurrence of these lesion.

#### What is the purpose of this study?

The main purpose of this study is to know the prevalence of oral mucosal lesions and it's relation to risk habits and sociodemographic factors.

#### What is the procedure to be followed?

Collection of information on the personal background, sociodemographic factors, and oral habit from a set of questionnaire will be taken. The participant will be seated on the dental chair for examination the oral condition. Photograph may be taken in some of these cases. Any oral disease requiring treatment will be referred to the relevant specialist.

#### Who is eligible for the study?

Patient who visits primary dental care in the Faculty of Dentistry in University of Malaya and patients who visit dental clinic in althwara hospital in Sana'a Yemen will be involved in the study.

#### Who is not eligible for the study?

Patient who had treatment of recent oral disease.

#### What is the benefit is this study?

Patient has the opportunity to be screened for any oral mucosal lesions and know the risk habit contributing to the prevalence of oral mucosal lesion.

What are the possible of drawbacks?

There is no drawback.

Who should I contact if I have additional questions about the study?

Doctor's name; Rajy Mansoor Saleh

Tel .no.0133061654

Supervisor: Dr. Nor Himazian Mohamed

Prof. Dr. Rosnah Mohd. Zain

#### A.3. Patient information sheet (Bahasa melayu verion)



#### **Borang Maklumat Pesakit**

Tajuk Penyelidikan: Kelaziman Kejadian Lesi Mukosa Mulut di Malaysia dan Yaman

#### Pengenalan:

Lesi mucosa mulut boleh ditafsirkan sebagai apa-apa perubahan yang berlaku pada mukosa mulut. Perubahan atau lesi ini boleh dikategorikan sebagai hadir sejak dilahirkan, peringkat pra-kanser atau peringkat kanser. Faktor risiko berkemungkinan berkait kepada perebakkan lesi ini. Faktor-faktor ini adalah seperti tabiat merokok, mengunyah tembakau, mengambil sireh-pinang, mengunyah pinang dan lain-lain lagi. Disamping itu, faktor sosiodemografik juga memain peranan penting berlakunya lesi ini.

#### Apakah tujuan penyelidikan ini?

Tujuan penyelidikan ini adalah untuk mengetahui kekerapan berlakunya lesi mukosa mulut ini dan kaitannya dengan risiko tabiat dan faktor-faktor sosiodemografik.

#### Apakah urutan yang perlu dipatuhi?

Pengambilan maklumat berkaitan latarbelakang peribadi, faktor sosiodemografik dan tabiat mulut akan diambil semasan sesi soaljawab dari soalan yang telah disediakan. Kemudian pesakit akan dibuat pemeriksaan mulut semasa berada diatas kerusi pergigian. Gambar bahagian mulut yang terlibat akan diambil jika perlu. Jika didapati pemeriksaan lanjut dan rawatan diperlukan, pesakit ini akan dirujuk kepada pakar.

#### Siapakah yang bersesuaian untuk mengikuti penyelidikan ini?

Pesakit-pesakit yang hadir ke Klinik Rawatan Utama, Fakulti Pergigian, Universiti Malaya atau pesakit yang melawat Klinik Pergigian di Hospital Althwara di Sana'a, Yaman akan terlibat didalam penyelidikan ini.

#### Siapakah yang tidak sesuai untuk penyelidikan ini?

Pesaki-pesakit yang sudah/sedang mendapat rawatan bagi penyakit mulut ini.

#### Apakah kelebihan mengikuti penyelidikan ini?

Pesakit-pesakit akan bermanafat jika diberi penyaringan lesi mukosa mulut ini serta mendapat maklumat tentang risiko tabiat yang boleh membawa tkearah kejadian lesi mukosa mulut ini.

Apakah keburukkan jika menarik diri dari penyelidikan ini?

Tidak ada perkara buruk yang akan berlaku.

Siapakah yang patut saya hubungi jika ingin mengemukakan soalan berkaitan penyelidikan ini?

Anda boleh hubungi : Dr. Rajy Mansoor Saleh Tel: 0133061654

Penyelia Projek Penyelidikan: 1) Prof. Dr. Rosnah Mohd. Zain

2) Dr. Nor Himazian Mohamed

.

A.4. Patient information sheet (Arabic version)



#### استمارة مطومات عن المريض

عنوان الدراسة : انتشار مرض الأغشية المخاطبة في ماليزيا واليمن المقدمة: تعرف أعراض الأغشية المخاطبة على أنها تغير في الغشاء المخاطي. إن هذا التغير قد يكون خلقياً أو خطر سرطاني أو قبل سرطان. هذا العوامل تختلف من العسادات مثل التدخين والشمة وتناول بذرة الغوظ والأريقة و مضغ القات إلى أخرد. إضافة إلى ذلك هنـــاك عامل لجتماعي يمكن أن يلعب دور في حدوث مثل هذا المرض مسا هسو غسرض الدرامسة ديمو غراقي. ان الغرض الرئيسي من ا لدراسة هو معرفة انتشار الأمراض المخاطبة الفعويسة وعلاقتهسا بمخاطر العادات والعوامل الاجتماعية الديغراقية. ما هي الإجراءات المتبعة: جمع معلومات شخصية والعولمل الاجتماعية الديموغرافية والعادات من خلال عمل استبيان سوف يتم وضع المشارك في كرسي خاص بالأسنان ومن ثم فحص حالة الفم لديه. قد يتم أخذ صور لبعض الحالات. أي مرض فموي يحتاج إلى معالجة يتم تحويل المريض إلى المختص. من هو الشخص الذي تصلح عليه الدر اسة: هم المرضى الذين يزورون عوادة الأسنان في كلية طب الأسنان في جامعة الملايسا وعيسادة الأسنان في م/الثورة -صنعاء- اليمن. من هم الذين غير مؤهلين لمهذه الدراسة: المرضى الذين قد تعالجوا من أمراض فموية حديثة. ما هي أهمية الدراسة: سوف يستفيد المريض من خلال معرفة أي أمراض في الغشاء المخاطي الفموي ويتعرف عن مخاطر العادات للتي تؤدي إلى الأمراض المخاطية الفعوية. ما هي العوائق المحتملة؟ لا توجد عوائق. مع من أتواصل إذا كان لدى استفسارات عن الدراسة الدكتور/ راجي منصور صالح تلغون: 0133061654 المشرفون: الدکتور / نور هیمازیان محمد

96

يروقسور/روسته محمد زين

#### A.5. Consent form (English version)



CONSENT BY PATIENT FOR CLINICL RESEARCH Faculty of Dentistry, University of Malaya, Kuala Lumpur.

I am	Identify o	card no:	
(Name of patient)			

Of.....

. (Address) Hereby agree to take part in the clinical research (clinical study) specified below: Title of study: Prevalence of oral mucosal lesions in Malaysia and Yemen The nature and the purpose of study have been explained to me by Dr Raji Mansoor Saleh Hajeb – (dentist) and interpreted by ...... to the best of his /her ability

(Name of interpreter) in ......Language / dialect

I have been told the nature of clinical research in terms of methodology, possible adverse effects and complications (as per patient information sheet). After knowing and understanding all the possible advantages and disadvantages of this clinical research, I voluntary consent of my own free will to participate in the clinical research specified above. I understand that I can withdraw from this clinical research at any time without assigning my reason whatsoever and in such a situation shall not be denied the benefit of usual treatment by attending doctors.

Date	Signature or thumbprint

IN THE PRESENCE OF

(Attending doctor)

(Patient)

#### A.6. Consent form (Bahasa melayu verion)



#### KEIZINAH OLEH PESAKIT UNTUK MENYERTAI PENYELIDIKAN KLINIKAL

Dengan ini bersetuju menyertai dalam penyelidikan klinikal (kajian klinikal) seperti berikut:

Tajuk penyelidikan: Kelaziman Lesi Mukosa Mulut di Malaysia dan Yaman

Yang mana sifat dan tujuannya telah diterangkan kepada saya oleh Dr Raji Mansoor Saleh Hajeb (doktor gigi) mengikut terjemahan

(Nama & jawatan penterjemah)

dengan sepenuh kemampuan dan kebolehannya di dalam bahasa/loghat.....

Saya telah diberitahu bahawa sifat dan tujuan penyelidikan klinkal ini dari segi metodologi, risiko dan komplikasi (mengikut kertas maklumat pesakit). Selepas mengetahui dan memahami semua kemungkinan kebaikan dan keburukan penyelidikan klinikal ini, saya merelakan/mengizinkan diri saya untuk menyertai penyeliikan klinikal tersebut di atas.

Saya faham bahawa saya boleh menarik diri daripada penyelidikan klinikal ini pada bila-bila masa tanpa memberi sebarang alasan dan dalam situasi ini tidak akan dikecualikan dari kebaikan rawatan oleh doktor.

Tarikh .....

Tandatangan /cap jari.....

(Pesakit

#### DI HADAPAN

Nama	
No.K/P	Tandatangan
	(Saksi untuk tandatangan pesakit)
Jawatan	······

Saya sahkan bahawa saya telah menerangkan kepada pesakit tentang sifat dan penyelidikan klinikal tersebut di atas.

Tarikh.....

A.7. Consent form (Arabic verion)



جامعة الملايا

أوافق للمشاركة في البحث الطبي الموضح أدناه:-عنوان الدراسة: انتشار الأمراض المخاطية الفموية في ماليزيا واليمن. طبيعة وغرض الدراسة قد وضحت لي من قبل الدكتور / راحي منصور صالح وترجمت مـــن قبل ...... لقد وضح لي طبيعة البحث الطبي على أساس البحث وكذلك الأعراض الجانبية والمضاعفات. يعد معرفة كل المزايا والسلبيات المرافقة للبحث فإنني أوافق طواعية للمشاركة قــي البحـث الطبي المحدد أعلاه كما إنني بإمكاني الانسحاب من البحث الطبيء في أي وقت بدون توضـح السبب وهذا الإجراء لا يعرقل المعالجة المعتادة من قبل الأطباء. التاريخ......البصمة

> بحضور الاسم: أؤكد أننى قد وضحت للمريض طبيعة وغرض البحث الموضح أعلاه. التاريخ ...

# A.8.Ethic approval

UNIVERSITY OF MALAYA KUALA LUMPUR	وزيرسيتهماليا
Producing Leaders Since 1905	
Ruj: UM.D/PD211/09	
Tarikh: 12 <sup>th</sup> May 2009	
Dr. Raji Mansoor Saleh Hajeb	
Through:	
Supervisor Prof. Dr. Rosnah Md. Zain Departmet of Oral Pathology, Oral Medicine & Peri Faculty of Dentistry	iodontology
Dear Sir,	
ETHICS APPROVAL	
It is a pleasure to inform you that your application 'Prevalence of Oral Mucosal Lesion in Malaysia a approval number is <b>DF DP0904/0030(P)</b> .	
Thank You.	
Yours Sincerely,	
hould	
PROF. DATO' DR. ABDUL AZIZ ABDUL RAZAK	
Chairperson Faculty of Dentistry Medical Ethics Committee	
s.k. Dean, Faculty of Dentistry Head of Oral Pathology, Oral Medicine & P	Periodontology Department

# QUESTIONNAIRE

# A.9. Questionnaire (English version)

Department of General Dental Practice and Maxillofacial Imaging Faculty of Dentistry, University of Malaya ©Dr. Raji Mansoor Saleh		
Supervisors: Dr. Nor Himazian Mohamed/ Prof. Dr. Ro	osnah Mohd. Zain	
R/N:         Centre no:         Subject n           Date:         day         month         year	o: 🗆 🗖 🗖	
Please circle your selected answer or fill the space provided	for office use	
A. Personal Background 1. Date of birth: day month year 2. Sex: a. Male b. Female 3. Ethnic group: a. Malay b. Chinese c. Indian d. Others (specify )	1 🗆 🗆 2 🛄 3 🗔	
B. Oral habit         B1.Smoking         4. Have you ever smoked tobacco or cigarette or any form?         a. Yes       b. No (if no go to B3)         5. At what age did you start smoking?         6. Do you currently smoke?         a. Yes       b. No         7. How many months ago did you stop smoking?         B2. Detail of smoking	4 5	
8. Do you smoke cigarette?         a. Yes       b. No         9. How many cigarettes per day do you smoke?         10. What type of cigarette do you smoke?         a. Filtered       b. None filtered         c. Both filter and none filte         11. Do you smoke "kretek"?         a. Yes       b. No         12. How many "kretek" per day do you smoke?         13. What type of "kretek" do you smoke?         a. Filtered       b. None filtered	8    9       10    11    12       13	

14. Do you smoke cigars?		
a. Yes b. No	)	14
15. How many cigars per d	ay do you smoke?	15
16. Do you smoke leaf toba	cco (rokok daun)?	_
a. Yes b. No	)	16
•	co per day do you smoke?	17
18. Do you smoke pipe?		
a. Yes b. No		
	ay do you smoke pipe?	19
20. Do you have other smol	0	a
a. Yes (specify	)	20
b. No	J	21
B3. Smokeless tobacco/Ouio	do you smoke?	21 🗆 🗆
	areca nut or use tobacco in any Smokeless form?	
a. Yes b. No		22
	art chewing areca nut /tobacco?	23
24. Do you currently chew?		
a. Yes b. No		24
25. How many months ago	did you stop chewing?	25
AV E		26
26. Frequency per day		
27. Average duration in mi	o you most commonly keep the mixture?	2/ []
a. left upper sulcus		28
c. right upper sulcus		20
e. anterior upper sulcus	f. anterior lower sulcus	
g. On the tongue	h. underneath the tongue	
i. Others specify (	)	
29. Do you use tobacco in a	iny other way?	
a. Yes (if yes specify	)	29
b. No B4. Oat chewing:		
30. Have you ever chewed of		
a. Yes	nat?	
and the later	-	30
31. How many times per da	b.No (if no go to B5)	30 🗌 31 🗆 [
	b.No (if no go to B5) ay do you chewing?	31
32. What age did you start	b.No (if no go to B5) ay do you chewing? chewing qat?	
	b.No (if no go to B5) ay do you chewing? chewing qat?	31
<ul><li>32. What age did you start</li><li>33. Do you currently chewing</li><li>a. Yes</li></ul>	b.No (if no go to B5) ay do you chewing? chewing qat? ing qat?	31 🗌 🗌 32 🔲 🗌
<ul><li>32. What age did you start</li><li>33. Do you currently chewing</li><li>a. Yes</li></ul>	b.No (if no go to B5) ay do you chewing? chewing qat? ing qat? b. No	31 🗌 🗌 32 🔲 🗌

35. How many months ago did you stop chewing qat?		35
36. Do you us	e any tobacco product (shamma) with qat chewing?	
a. Yes	b. No	36
37. Do you us	e pipe water with qat chewing?	
a. Yes	b. No	37 🗌
38. Do you sm	oke with gat chewing?	
a. Yes	b. No	38
<b>B5.Alcohol hal</b>	pit	
39. How often	do you consume alcoholic beverage?	0.20.02
a. Do not consume (Questions finish here)		39
b. Almost d	aily (6/7time a week)	
c. Three to f	five times a week	
d. Once or t	wice a week	
40. At what a	ge did you start costuming alcohol beverage?	40
41. Do you cu	rrently consume alcohols beverage?	
a. Yes	<b>b.</b> No (Questions finish here)	41
42. What kind	l of alcohol beverage do you drink most often?	
a. Beer	b. Stout c. Wine d. Toddy	42
e. Samsu	f. Others specify ()	

Thank you for your participation and cooperation in this survey. All information provided will strictly be kept confidential and be used for our purposes only.

A.10. Questionnaire (Bahasa melayu verion)

A Start	UNIVERSITI MALAYA KUALA LUMPUR	
	Jabatan Rawatan Pergigian Am dan Pengimejan Fakulti Pergigian, Universiti M	
	©Dr. Raji Mansoor Saleh Supervisors: Dr. Nor Himazian Mohamed/ Prof. I	
R/N: Tarikh: hari Sira bulathan ja	No. Pusat: No. Subjek : bulan awapan pilihan atau isi ruang yang disediatan.	Untuk kegunaan pejabat
A. Latar belakar 1. Tarikh lahir:		
2. Jantina:		
a. Lelaki 3. Kumpulan Et a. Melayu	b. Cina c. India	$2$ $\Box$ $3$ $\Box$
<b>B.</b> Tabiat Oral	(Sila nyatakan)	
B1. Merokok 4. Pernahkah a	nda mengisap tembakau atau rokok atau bentuk la	ain?
a. Ya 5. Umun katika	b. Tidak (Sila pergi B3)	4
6. Anda masih		,
a. Ya 7 Berene bular	b. Tidak n lepas anda berhenti merokok?	
B2 .Maklumat n	nerokok	
8. Anda mengis a. Ya	ap rokok? b. Tidak	8
9. Berapa batar 10. Jenis rokok		_ 9 🗆 🗆
a. Bertapis 11. Anda hisap	b. Tidak bertapis c. Kedua-dua bertap kretek?	is dan tidak 10 ∟
a. Ya	b. Tidak	11
12. Berapa ban 13. Jenis kretek	yak kretek anda hisap sehari? k anda hisap?	
a. Bertapis	b. Tidak bertapis	13 🗌
14. Anda hisap a. Ya	b. Tidak	14
15. Berapa bata	ang curut anda hisap setiap hari?	
16. Anda hisap		16
a. Ya	b. Tidak	

	rokok daun anda hisap setiap ha	uri?	17 🗆 🗆
18. Anda hisap paip			
a. Ya	b. Tidak		
	anda hisap setiap hari? merokok yang lain?		
a. Ya (Sila nyataka b. Tidak	n)		20
B3. Tembakau/Kuny	i batang anda hisap setiap hari? <u>ah sireh pinang</u> i mengunyah pinang atau sentel		21
a. Ya	b. Tidak (Sila pergi B4)	tembukuu.	22
	ula mengunyah pinang/tembaka	u?	23
a. Ya	b. Tidak		24
25. Berapa bulan le	pas anda berhenti mengunyah?		25
	p hari		26
27. Purata tempoh	The second se		27
	mulut anda menyimpan bahan	kunyahan?	_
<ul> <li>a. A tas sulkus kiri</li> <li>c. Atas sulkus kana</li> <li>e. Atas sulkus depar</li> <li>g. Di atas lidah</li> </ul>		n sulcus depan	28
i. Lain-lain. Sila nya	itakan(	)	
29. Adakah anda gu	nakan tembakau cara lain-lain?		_
a. Ya (jika ya sila nya b. Tidak <b>B4. Mengunyah Oat</b>	atakan)		29
30. Adakah anda m			30
a. Ya	b. Tidak (Sila pergi B5)	)	50 L
	a mengunyah?		31
	pa anda mula mengunyah qat? ·		32
a. Ya	b. Tidak n mulut anda menyimpan mengu	unvah gat?	33 🗌
a. kiri mulut	b. kanan mulut	c. nedua-du	a belah 34
35. Berapa bulan ya	ng lepas anda telah berhenti me enggunakan produk tembakau (	engunyah qat?	35 🗆 🗆
a. Y	b. Tidak	snamma) uengan me	
	enggunakan paip air dengan me	ngunyah qat?	
a. Ya	b. Tidak		37
38. Adakah anda m	engisap rokok/curut sambil me	ngunyah qat?	_
a. Ya	b. Tidak		38

# B5.ATabiat minum arak

39. Berapa kei	apan anda meminum arak?		_
a. Tidak mir	num (soalen berhenti disini)		39
b. Hampir se	etiap hari (6/7kali seminggu)		
c. Tiga hing	ga lima kali seminggu		
d. Sekali ser	ninggu		
40. Pada umu	r berapa anda mula minum arak?		40
	nda masih minum arak?		
a. Ya			41
	alan berhenti disini)		41
	yang biasa diminum?		
a. Bir	b. Stout c. Arak. D. Todi		42
e. Samsu	f.Lain-lain sila nyatakan (	)	

Terima kasih kerana penglibatan dan kerjasama anda dalam kajian ini. Semua maklumat adalah sulit dan digunakan untuk tujuan kami sahaja.

## **APPENDIX 11**

## A.11 Questionnaire (Arabic version)

	قسم عواده الاسنان العامه والتصوير القمي والوجه والفكين كلية طب الاسنان جامعة اليو ام الدكتور : راجي منصور صلاح المشرفون: الاستاذ دكتور/ روزنا محد زين _الدكتور/نور همازيان محمد
	رقم التسجيل
	ار المطومات الشخصية ١. تاريخ الميلاد: اليوم 🗌 الشهر 🗌 المنة 🛄 🛄
۲ ای	۲.النوع: المنكر 🗌 ب.انشي 🕤
1 4	۳ الجنسيه: ۱. يمني ] باخرى حدد ()
٤ [	٤ کم محل دخلك الشهري (ریال بمني)؟ ١.٠٠٠ او تقل ک ب.١٠٠٠ ب. ٤٠٠٠٠ ج.٤٠٠٠ ج.٤٠٠٠ او تقل ک
र •	٥ المستوى التطيمي ١. امى . ب. تطيم غير اسلسي . ج. التطيم الابتدائي
	د.التطيم الثانوي معهد و. التطيم الجنعي
۲ ک	٦. اين تسكن؟ المالويف ببضواهي المدينة ج المدينة ج المدينة الي العلامات بمالويف المدينة ج المدينة المدينة المدينة ج المدينة ال المدينة المدينة ا المدينة المدينة الم
	ب ١ التدخين
V [2]	٧. هل دخنت التيغ او المسجلير من قبل ؟ .نعم لا ٢٠ (٤) انتقل الى المسؤال ب ٣
	٨. في اي عر بدأت التدخين ؟
1	٩. هل انت مستمر في التدخين ؟ ١. تعم ب ٢
,.	١٠ منذ كم شهر تركت التدخين ؟
	ب٢ :مطومات عن التدخين:
	۱۱. هل تحخن السجائر ؟ المم ب لا ۲۱. مل تحخن السجائر المم
	١٣. مقوع المديجارة التي تدخنها ؟ ١. المعجلار المفلترة ب. المدجلار الغير مفلترة
11	ع: -وسع الميجار؟ الم المع الم بيان المراجع عن المراجع المراجع الم
10	٥٥ كم عد المديجار التي تدخذها في اليوم ؟
	١٢. هل تدخن اوراق المتبغ؟ المنعم 🗌 ب. لا
	<b>`</b>

1 11	
	١٧ کم ورکه تيغ ندهن في انبوم:
	١٨. هل تدخن تيغ الظيون؟ المعم 🛄 ب ٢٠ س
	۱۹. کم مرد ای انہوم تشخن نیخ الطبون:
	٢٠. هل عندك علدات تشغين الخرى؟ ١. نعم 🛄 حند (
Y1	٢١ كم مرة/قطعة تنخن من هذا النوع ؟
	ب٣: عادات مضغ التيغ
	٢٢. هل سبق لله أن مضغت التبغ بلا دخان في أي شكل (الشمه أو التميل)؟ أ. تعم 🗌 ب. لا 🖾 إذا (لا)
	انتقل الى المنوال ب ٤
	۲۲ کې ای عمر بدات مصلع النبع ا
Y£	٤ ٢ . هل انت مستمر في عادة المضغ؟ ١. نعم ب. لا
Yo	٢٥ .كم شهر مضى منذ تركت مضغ التبغ ؟
	تركيب المواد المستخدمة في المضغ
Y1	۲۲.اریکا البندق : ا.نعم 🗌 ب.لا
YY	٧٧.التيغ : ا,نعم 🗌 ب.لا
TA	۸۷.الکلس : ۱.نمم 🗌 ب.لا
79	
٣.	۲۹.اخرى : ا.نعم ] حدد () ب.لا ]
٣١	٢٠ : مَ مَسَرَوَ الْمَعَوِّ في الوَّذِي . ٣١ . المدة المتوسطة في الدقيقة؟
	٣٢ في اى جزء من اللهم غلباً تحفظ المادة ؟ [الاخدود اليسار الاعلى] بالاخدود اليمين الاعلى]
77 17	ج الاخدود اليسار الاسفل 🗌 د. الاخدود اليمين الاسفل 🤇 هرالاخدود الامامي الاعلى
	و الاخدود الاملمي الاسفل 🗌 زيفوق اللسان
	طراخری. حدد()
77	٣٣. هل تستخدم النبغ باى طريقة اخرى ؟ ابنعم 🗌 حدد () ب.لا
٣٤	عند من المنه الله المن المنه الم المنه المنه الم المنه المنه المنه المنه المنه المنه المنه المنه المنه الم المنه الم المنه الم المنه المنه المنه المنه المنه المنه منه منه المنه منه منه المنه المنه المنه المنه ا منه المنه الم
50	
	٥٣ كم مره تخزن القلت؟ ( يوميا ب مرتين ثلاث مرت في الاسبوع ج. مره في الاسبوع
TT	۲۰ ایم مناعه نظری الملک می الموم. ۳۷ فی ای عمر بدأت تخزن القلت؟
YA	
<b>F</b> 9	۳۸ هل مازلت تخزن القلت ؟ ارتعم بلا بلا
 £•	
	٤٠ في اى جهة من الله غلايا تخزن القلت ؟ افي الجهة اليسرى ب في الجهة اليمنى
	ج.في الجهتين
٤١	٤١ . هل تستخدم اي تدغ (الشمه) مع القات ؟ ١ ينعم 🔤 ب. لا
٤٢	٢ ٤. هل تنخن المداعة مع القلت ؟ ١. نعم 📃 ب. لا
٤٣ ٢	٤ × هل تدخن المدجلير مع القلت ؟ ابنعم بي لا
££	٤ ٤ كم سيجاره تدخن مع القلت؟

Table 4.1 Diagnostic criteria of ora	l mucosal lesions (contd.)
--------------------------------------	----------------------------

Oral mucosal lesions	White lesions	Red lesions	ulcerated lesions	Quid related lesions	Exophytic/swell ing, pigmented and other lesions
		1.Oral malignant lesion	S		1
Oral carcinoma (OC)+	May appears as a white area and indurated. May be fixation of movable part of mucosa, the surface maybe nodular or ulcerated and may appear as a fungating mass.	OC may develop in a red area with induration, and firm and thickened through the lesions or at the ulcerated margin.	This lesion is ulcerated with induration at the margins. The ulcer have raised, rolled border and may develop in a white area.		OC may appear as fumigating exophytic mass, which may be bleeding easily at a later stage.
		2.Oral Potentially maligna	nt disorders		
Leukoplakia+	A predominately white lesion of the oral mucosa that cannot be characterized as any other definable lesion.				
Erythroplakia+		A reddish area with irregular outline and sometimes a granular surface and cannot be diagnosed as any other definable lesion			
Oral Submucous Fibrosis +				There are palpable bands in the oral mucosa which lead to limited mouth opening. The tongue may be small and show a marked loss of papillae in early lesion as well as vertical fibrous bands which can be detected in the cheek. The buccal mucosa may appear atrophy with presence of betel quid stain	

Oral mucosal lesions	White lesions	Red lesions	Ulcerated lesions	Quid related lesions	Exophytic/swel ling, pigmented and other lesions
Lichen planus (LP) +	<ul> <li>(i) The papular form consists of white pinhead sized papules</li> <li>(which cannot be rubbed off) and may form linear, reticular or annular pattern.</li> <li>(ii) The reticular form consists of white distinct striae (which cannot be rubbed off) forming linear, reticular, and annular pattern.</li> <li>(iii) The plaque form consists of white plaque like lesions with striae at the margins (which cannot be rubbed off.</li> </ul>	<ul> <li>(i) The erythematous form consists of red areas with papules, striae at the margin (which cannot rubbed off).</li> <li>(ii) The tongue atrophic form consists of the atrophy of tongue papillae with a whitish, dry surface, white patches or striae (which cannot be rubbed off ) are present in other areas of mouth (iii) The bullous form consists of vesicles/bullae in the area of white and red forms of LP.</li> </ul>	<ul> <li>(i) The erythematous form consists of red areas with papules, striae at the margin (which cannot rubbed off).</li> <li>(ii) The tongue atrophic form consists of the atrophy of tongue papillae with a whitish, dry surface, white patches or striae (which cannot be rubbed off) are present in other areas of mouth</li> <li>(iii) The bullous form consists of vesicles/bullae in the area of white and red forms of LP.</li> </ul>		

#### 3. Other Lesions (Non oral malignant & Non oral potentially malignant Disorders)

Oral mucosal lesions	White lesions	Red lesions	ulcerated lesions	Quid related lesions	Exophytic/swelling, pigmented and other lesions
Betel chewer 's mucosa+		The oral mucosa shows a tendency to desquamate or peel and detached tags of tissue can be seen or felt. The underlying areas assume reddish membranous or wrinkled appearance; the area may show yellowish or reddish-brown surface tag that is the evidence of incorporation of ingredients of quid.		The oral mucosa shows a tendency to desquamate or peel and detached tags of tissue can be seen or felt. The underling areas assume reddish membranous or wrinkled appearance; the area may show yellowish or reddish- brown surface tags that is the evidence of incorporation of ingredients of quid.	

Oral mucosal lesions	White lesions	Red lesions	Ulcerated lesions	Quid related lesions	Exophytic/swelling, pigmented and other lesions
Betel quid lichenoid lesion+	It resembles lichen planus with specific difference such as presence of white linear, wavy, non elevated parallel lines which do not overlap or criss-cross and in some instances radiate from a central erythematous area.			It resembles lichen planus with specific difference such as presence of white linear, wavy, non elevated parallel lines which do not overlap or criss-cross and in some instances radiate from a central erythematous area.	
Areca quid related lesions (Seedat ,1985)	An ill-defined whitish gray discoloration on the buccal mucosa either uni- or bilaterally that cannot be rubbed off. The mucosa may show line like texture.			An ill-defined whitish gray discoloration on the buccal mucosa either uni- or bilaterally that cannot be rubbed off. The mucosa may show line like texture.	
Qat related lesions ( Aiman et al., 2004)	Mild whitening in the buccal mucosa develops till very clear white keratosis at the site of qat chewing. As a result of the mechanical friction during chewing, the chemical constituents or additives to qat.				
Shammah related lesions (Scheifele et al., 2007)	<ul><li>(i) Clinically white or white-yellow lesions that could not or only partly be wiped off, (ii) a history of burning sensation during 48 h before examination.</li></ul>			1) Clinically white or white-yellow Lesions that could not or only partly be wiped off. (2) a history of burning sensation during 48 h before examination	
Frictional lesion+	It is a whitish area on the mucosa which is directly related to a traumatic agent.				

Table 4.1 Diagnostic criteria of oral mucosal lesions (	(contd.)	
Tuble 41 Diagnostic criteria of oral macosar resions	(contra.)	

Oral mucosal lesions	White lesions	Red lesions	Ulcerated lesions	Quid related lesions	Exophytic/swelling, pigmented and other lesions
Lina alba	It appears as raise wavy lines located in the occlusal line of buccal mucosa bilaterally extend from the canine area to retro molar area which cannot be rubbed off.				
Aphthous ulcer +			The recurrent minor ulcers aphthous ulcers are usually confined to non keratinized oral mucosa or tongue with 1- 4 ulcers at one episode which measure up to 1cm and heal within 1-2 weeks without scarring. The ulcer well defined and covered by a grey- white or yellowish fibrinous coating surrounded by an erythematous halo. The recurrent major aphthous ulcer may be present as 1-2 ulcers at each episode usually with firm margin and heals with scarring.		
Traumatic ulcer +			It appears as mild or moderate symptomatic ulcer on the oral mucosa which is related to trauma.		
Angular cheiltis +			There is fissuring or ulceration of skin and/oral mucosa in the labial commissure or discontinuity of the commissural mucosa or the skin which can be provoked by slight stretching.		

Oral mucosal lesions	White lesions	Red lesions	ulcerated lesions	Quid related lesions	Exophytic/swelling, pigmented and other lesions
Check and lip biting+	It is definable and diffusely outlined lesions where there is self-infliction from chewing with a whitish, rough, macerated flaky surface due to surface desquamation and a red underlying desquamative area of the mucosa.				
Acute erythematous candidosis (EC) +	This lesion may appear as red painful areas of the oral mucosa, which may occur during treatment with antibiotics such as the 'antibiotic sore tongue.				
Denture stomatitis +	This lesion may be a form of chronic EC and shows a diffusely red dnture covered mucosa (DCA). May have multiple, small, papillomatous, reddened hyperplasias. It may be present for less than 2third (localized type) or more of DCA (generalized type).				
Geographic tongue +	It is a well defined entity which presents as well demarcated areas of depapillation with reddening or whitish/yellowish, serpinginous lines partly surrounding red depapillated areas.	It is a well defined entity which presents as well demarcated areas of depapillation with reddening or whitish/yellowish, serpinginous lines partly surrounding red depapillated areas.			

Oral mucosal lesions	White lesions	Red lesions	ulcerated lesions	Quid related lesions	Exophytic/swelling, pigmented and other lesions
Coated tongue (Field and Longman., 2003)	It is a coating consisting of a layer of mucus, desquamated epithelial cells, organisms, and debris. This coating may quickly become very much thicker. A lack of mobility of the tongue, which may be caused by the most minor painful lesions, an excess of tobacco or of alcohol, a gastric or respiratory upset, or a febrile condition, may result in a build-up of the tongue coating sufficient to produce a white or coloured plaque. The colour of such a coating depends on a variety of factors, such as tobacco usage and dietary habits				
Hairy tongue (Bruch and Treister., 2009).	It appears as elongation of filiform papillae on the dorsum surface of the tongue as hair like with gagging or sensation irritation. The debris, coffee and tobacco can cause a range of colour variation from black to pink and green.				
Leukoedema+	A diffuse grayish- white, smooth, edematous film bilaterally in the buccal mucosa. there are delicate folds which do not disappear on maximal opening but the lesions surface can be scrapped or displaced and the folds re-established themselves within a short time				

Oral mucosal lesions	White lesions	Red lesions	ulcerated lesions	Quid related lesions	Exophytic/swelling, pigmented and other lesions
Fibroepithelial polyp+					It is an exophytic nodular sessile or pedunculated overgrowth of the mucosa which has a surface colour similar to oral mucosa
Pyogenic granuloma+		It is usually nodular, sessile /pedunculated with a smooth, granular or lobulated surface and it is reddish /brownish in colour and bleeds easily.			It is usually nodular, sessile /pedunculated with a smooth, granular or lobulated surface and it is reddish /brownish in colour and bleeds easily.
Hyperplatic gingivitis (Laskaris, 2006)					

+. Based on Zain et al (2002).clinical criteria

A.12. Clinical examination form

#### **Clinical examination**

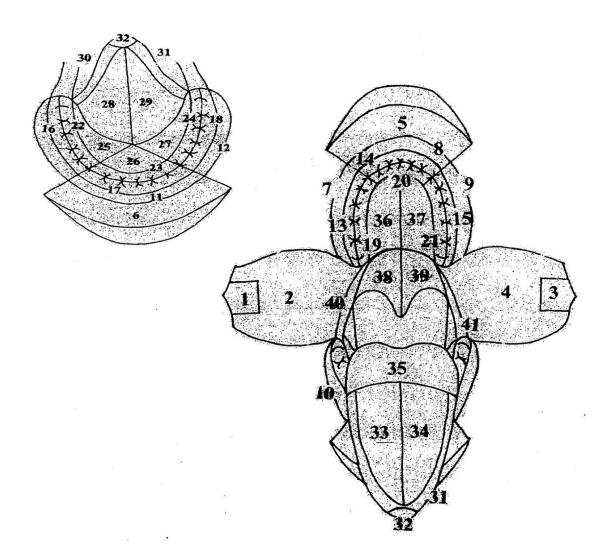
Subject No



A. Type of lesion

1.Suspicious of oral cancer
 2.Leukoplakia
 3.Erthroplakia
 4.Lichen planus
 5.Oral sub mucous fibrosis
 6.Chewer's mucosa
 7.Other lesion specify (.....)

#### **B.SITE OF LESION**



Appendix 14



(a)

(b)



(c)

(d)

A.14. Fig.5.4 (a) Qat chewer appear to chew on the left side (b) Qat chewing on right side and a bunch of qat leaves (c) qat chewing session (common habit) (d) leave of qat